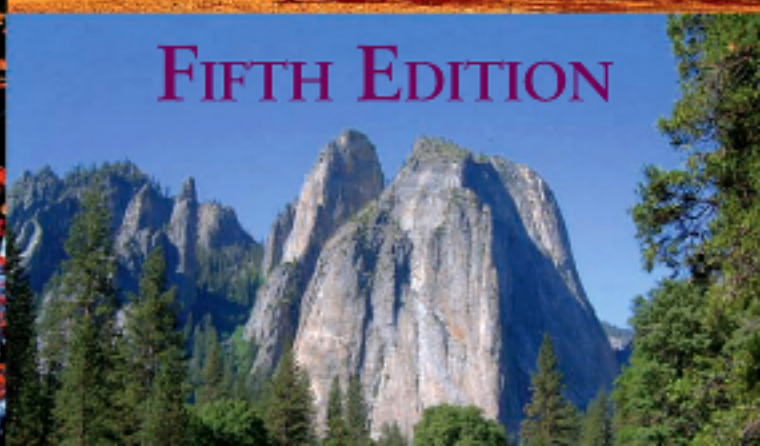
Paul S. Auerbach

Wilderness Medicine

Fifth Edition



MOSBY
ELSEVIER

Conversion Charts

▶ LENGTH

1 inch = 2.54 cm	1 cm = 0.3937 inches
1 foot = 0.3048 m	1 m = 3.2808 feet
1 yard = 0.9144 m	1 m = 1.0936 yards
1 mile = 1.609 km	1 km = 0.6214 miles

▶ VOLUME

1 oz (U.S., liquid) = 29.5735 mL	1 mL = 0.0338 oz (U.S., liquid)
1 oz (U.K.) = 28.4131 mL	1 mL = 0.0352 oz (U.K.)
1 cup = 0.2366 L	1 L = 4.2268 cups
1 pint (U.S., liquid) = 0.4732 L	1 L = 2.1134 pints (U.S., liquid)
1 pint (U.K.) = 0.5683 L	1 L = 1.7598 pints (U.K.)
1 quart (U.S.) = 0.95 L	1 L = 1.06 quarts (U.S.)
1 quart (U.K.) = 1.14 L	1 L = 0.88 quarts (U.K.)
1 gallon (U.S.) = 3.7854 L	1 L = 0.2642 gallons (U.S.)
1 gallon (U.K.) = 4.55 L	1 L = 0.22 gallons (U.K.)

▶ WEIGHT

1 oz = 28.3495 g	1 g = 0.0353 oz
1 lb = 0.4536 kg	1 kg = 2.2046 lbs

▶ ENERGY

1 Joule = 0.000239 Kcal	1 Kcal = 4184 Joules

▶ TEMPERATURE

To convert Fahrenheit to Celcius: degrees F – 32 × 5/9
To convert Celcius to Fahrenheit: degrees C × 9/5 + 32

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Foreword

More than a decade ago, I had the good fortune to be searching for new species of flowering lianas in the remote jungles of central Suriname. A tropical downpour had just ended, and we wandered through a forest still dripping from the rain shower. The air had become cool and buoyant, perfumed with a panoply of enticing floral scents.

Within a few hours of collecting, however, the heat had once again set into the equatorial lowland forest. Members of our small expedition sat down underneath the shade of a large bergibita tree on granitic stones covered with a fine green moss to take a rest in the torpid jungle atmosphere. As I struggled to fish granola out of my waterproof daypack, the botanist accompanying me cursed and jumped to his feet. "Something bit me!" he exclaimed.

We searched the site but never found the offending creature. Only two small pinpoints on the skin of his ankle, separated by no more than a couple of millimeters, could scarcely be discerned, nothing remotely commensurate to the pain experienced by my friend. I asked our Indian guide—a shaman of the Trio tribe and a man of few words—what had happened. He replied, "Mwe," the Trio word for spider.

In short order, the botanist became very ill. He became dizzy and dropped to his knees. Delirium set in as he begged for water and crawled into a nearby shallow stream. Nausea set in, accompanied by dry heaves. This famously stoic scientist was reduced to sobbing from the excruciating pain, which seemed to wrack every joint of his body.

My first aid kit offered no remedy with which we could be hopeful of improvement. My colleague's condition steadily deteriorated. Fighting the urge to panic, I tried to determine the best course of action. Should I attempt to move him? Leave him and seek help? We were dozens of miles of trackless jungle away from any Western-trained health care professional or even the most rudimentary pharmaceuticals or supportive care. I was terrified and powerless to help my friend.

I felt a presence and looked up from my incapacitated colleague onto the stream bank. There sat the medicine man, serenely surveying the scene with calm, knowing eyes. I had hired him to teach us the indigenous names of the plants we were collecting, never anticipating that we might need his healing services as well. Working in the Amazon as an ethnobotanist for 20 years, I have developed an immense respect for the traditional knowledge of these ancient healers. I asked the shaman if my ailing colleague would die.

The Indian grunted as he pointed his chin at the botanist. "Not going to die," he said, in his language. "Going to suffer, but not die."

His words began to lift my weight of concern, but were soon contradicted by the pitiful cries and moans of my colleague. "Well," I urgently asked, "do you know of any therapies that can help him?"

"Yes," he nodded, standing up from his resting place on the ground and brushing coarse sand off his red breechcloth. "Give me your machete."

He took the knife and disappeared into the bush. About three minutes later, he returned with two meters of a dull brown liana stem from the Philodendron family. The medicine man walked into the stream, turned the botanist onto his back, sliced the liana into four pieces and carefully let the liana sap drip onto the bite marks on the victim's ankles.

In less than 10 minutes, our patient felt well enough to sit up. His dizziness and nausea had diminished substantially. Within a half-hour, we were able to help him back to camp, where he spent the rest of the day recuperating in his hammock. He dozed fitfully for most of the afternoon, awoke in time to eat a hearty dinner by the campfire, and then slept through the night. By the next morning, he seemed back to normal.

This episode is likely how most of wilderness medicine was originally practiced and how, in more than a few remote corners of the world, it is still practiced today. For most of human history and prehistory, there was not a significant presence of doctors, validated drugs, or hospitals. There were, of course, healers (e.g., shamans, herbalists, bonesetters) and remedies (usually plants, but also other substances ranging from molds to insects to honey to soils); these evolved from trial and error to our current scientific approach. So it was a supreme irony when Paul Auerbach and his colleagues coalesced the field of Wilderness Medicine two and a half decades ago—clearly a case of back to the future!

In my world of ethnobotany, work and adventure carry me far from medical security. I have the good fortune to work in remote places where much of Western medicine has never been available. This is not thrill seeking—it is my professional calling. For large portions of my life, I live in the wilderness. Thus, I regularly witness the practice of wilderness medicine. It is at once remarkable and frightening—remarkable in the sense that indigenous healers know so many things from which we could derive enormous benefit and frightening because their medicine is laden with empiricism, mysticism, and lack of what we would consider detailed scientific substantiation. Some would call it "primitive," but that shouldn't be taken to mean it is incorrect. In fact, time and time again, as in the case of the spider bite, I have seen it heal.

In an age in which medicine becomes increasingly specialized and enhanced by technology, I like to think of practitioners of wilderness medicine as masters of "Renaissance medicine." They must be willing to abandon urban trappings and tools to get "down and dirty." Furthermore, they must know much more than they learned in medical school. If you are in a mountaineering accident, do you want the first healer on the scene to be a doctor whose expertise is confined to cancer? If you fall out of a tree and break your bones, do you want the first responder to be someone who has never attended a fracture?

When my wife was bitten by a copperhead snake in Virginia several years ago, she was taken by ambulance to a major, well-equipped suburban hospital. The good news was that she had no end of doctors eager to examine her. The bad news was that none of them had ever treated or even seen a snakebite, hence their interest in having a look! I was able to track down Paul Auerbach, who talked the treating physician through the necessary treatment. I had known enough to turn to my friend from the Wilderness Medical Society. Where else in the medical world can you consistently find people adept at treating victims of snakebite, frostbite, lightning strikes, volcanic eruptions, and scorpion envenomation?

This is not to argue that wilderness medicine is restricted solely to treating obscure afflictions or problems of the past. The marriage of traditional wisdom with cutting edge science offers new and important potential for better treatment of everything from high-altitude pulmonary and cerebral edema to heat stroke. These new approaches may incorporate everything from wisdom of local peoples to biodesign of novel medical devices. Remember that doctors now place leeches on healing wounds, which is more effective than drugs designed by modeling proteins with computers.

We live in an interesting epoch in which our societies are increasingly urban and technology oriented. Yet, something in our souls yearns for regular and sustained contact with the wonders of Mother Nature. The demand for ecotourism is expanding at an astounding rate. Doctors, nurses, and other health care providers simply must know what to do when confronted by the inevitable afflictions that result from our species' collisions with the natural world.

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Preface

I am very excited to present this fifth edition of *Wilderness Medicine*, which supports a phase of tremendous growth and excitement in the discipline. As man extends into remote reaches of the globe and large populations encounter environmental changes at an ever-increasing rate, this medicine of exploration, adventure, travel, and disaster response has become indispensable. Although much of the medicine practiced in remote areas or under environmental extremes is emergency in nature, and local conditions and considerations by necessity dominate, the field of wilderness medicine has advanced beyond the exciting rescues of extreme alpinists and survivalists. It has expanded in scope to include the practice of medicine in situations of constrained resources, during times of catastrophe, and often in impoverished countries. In noble responses to events that generate urgent and profound medical needs, practitioners skilled in wilderness medicine have become rescuers and leaders noted for their resourcefulness and rugged practicality. Because humans continually try to dominate the landscape in their disregard for the environment and fellow man, nature is the force with which we must constantly reckon. All major hazards ultimately involve the power and energy of mighty winds, harsh solar radiation, extreme cold, and the like.

I am particularly gratified that wilderness medicine is a discipline on the verge of becoming a specialty, on its own and possessed of profound relevance. Wilderness medicine is advanced in basic science laboratories and by academicians who witness their efforts translated under harsh field conditions within hazardous wilderness areas above, upon, and under every natural surface on Earth, and soon, in space. We might investigate the application of protein kinase inhibition to treat reperfusion injury in the mitochondria of endothelial cells, then find willing volunteers on expeditions to investigate its application for prevention and treatment of frostbite. Curiosity about the stinging mechanism of jellyfish, combined with observation of clownfish protected with a host anemone, leads to clinical trials in which volunteers demonstrate the efficacy of a topical jellyfish sting inhibitor. Drugs designed to treat hypertension and sexual dysfunction are evaluated for potency in the prevention and treatment of high altitude pulmonary edema. Cooling devices designed for elite athletes may save the lives of firefighters. On mountaintops, within jungle canopies, and in arid canyonlands, we seek to understand the forces of pressure, temperature, and weather, so that we may enjoy better health and safer journeys.

The growth of wilderness medicine as a field of study and practice is remarkable only with respect to the brief history of the term. When Ed Geehr, Ken Kizer, and I dreamed up the Wilderness Medical Society, it was not a brainstorm, but an obvious response to pent-up demand. There are innumerable physicians, allied health professionals, rescuers, and laypersons dedicated by profession and avocation to wilderness environments. The current status of wilderness medicine is characterized by increasing interest from the medical community as structure is imposed on persons and institutions possessing expertise. Wilderness medicine is now well established, so training programs are being tailored to correspond to the educational level and certifications of trainees and practitioners. However, as we attempt to apply urban standards of hygiene and health to every environment in which man has a consistent presence, wilderness medicine must never lose sight of its origin in the medical concerns existing in true wilderness territories. Herein lies the essential nature and appeal of the specialty.

Is wilderness medicine important in an age of widespread communicable disease and potential pandemics? How many people are victims of shark attacks versus how many persons suffer from diabetes? How many climbers reach the Seven Summits versus how many elders are stricken with congestive heart failure? How many children fall prey to high altitude pulmonary edema versus how many infants acquire HIV from their mothers? The inquisitive mind seeks not to partition what we learn from our test tubes and urban patients, but to constantly integrate and extrapolate. I am thoroughly convinced that wilderness medicine supports some of the most practical education and skills acquisition in the health care profession. Medicine and its science are intricate, so what is learned in one venue is commonly applicable across a wide spectrum of human disease. We owe it to our hearts and minds to pursue every aspect of medicine that challenges our imaginations and ingenuity. As an additional benefit, it is a joy to observe a healer normally bound to an urban existence lifted in spirit by recognition that medicine applies equally well to adventure, travel, and discovery. Furthermore, while wilderness medicine was not conceived solely to be medicine in the absence of customary resources, it has to a certain extent evolved that way. Wilderness medicine is not practiced in wilderness hospitals, but in the field—not on paved pathways, but in forests, at base camp, and on the beach.

We will advance the science, increase the teaching, and improve the practice of wilderness medicine. Even while wilderness medicine is often practiced solo, it brings people together, bonded by their community of search and rescue, medicine, and love of the outdoors. We will reach out in public health and service related to wilderness medicine. Many global health issues are in part within the purview of wilderness medicine. In response to these, and other, scourges of mankind, practitioners of wilderness medicine will bring their expertise to bear on developing sensible solutions that can actually be implemented. Injury prevention will have its day in a more concerted effort to make the outdoors safe for those who choose or need to enter it. In this new age of medicine, when diagnostics and therapeutics in the hospital have evolved to molecular and nano methods, what is the impact of technology on wilderness medicine? At the true wilderness level, not much. Technology can mitigate risk only to a certain degree. In the field, there is not yet computed tomography or magnetic resonance imaging, and I do not foresee a wilderness intensive care unit, notwithstand-

ing the space station. Perhaps when we have miniaturized everything, we will occasionally interpret ultrasounds and read brain waves on mountains and glaciers, but that will not change the essence of making do when all you have is your senses and courage. So, downstream wilderness medicine is very hands-on and frequently improvisational. One need only consider what was valuable in the wake of recent tidal waves, hurricanes, mudslides, and earthquakes to appreciate our increasing need for people who practice medicine with very little on short notice under extreme environmental stress.

Much in this volume is new and enhanced. In deference to current political conflicts that are perpetrated in rugged environments, it has become appropriate to introduce concepts related to the military, so that medics are familiar with considerations of tactics. Because astronauts seek to expand their boundaries, unique medical concerns of this new wilderness are once again part of the discussion. Competitive athletes take their marathons, races, and survival events farther into the backcountry, so there is new emphasis on adventure sports and the medicine necessary to support them. When the first edition of this book was published, my colleagues and I dove, climbed, soared, and trekked with impunity. These days, our lungs and limbs talk back to us, and we appreciate the opportunities for contemplation as much as challenges. Who among us will not grow older or develop functional limitations? The wilderness is for everyone and should not be limited to only the hardiest and most intrepid. Therefore, I have added chapters on pre-existing conditions, chronic illnesses, and persons with disabilities. More in-depth coverage of "traditional" wilderness medicine topics, such as acclimatization, natural hazards, search and rescue, and poisonous and venomous creatures is ably provided by a very dedicated group of experts. To allow sufficient space for all the upgrades, the references have been moved to an accompanying DVD-ROM. As in previous editions, I am greatly indebted to my remarkable wife Sherry and children, Brian, Lauren, and Dan; to all of the tireless contributors; and to the supportive and gracious Todd Hummel, Jennifer Shreiner, and Michael Goldberg at Elsevier.

I practice and preach wilderness medicine with passion—for medicine, for my patients, for my colleagues, and for the wilderness. I am perpetually amazed by remarkable tales of adversity, endurance, rescue, faith, the will to live, and the indomitable human spirit. That is most fitting for the setting in which these events occur, for at most times in the wilderness, we are able to appreciate everything that is natural and wonderful upon this Earth. As you peruse this book, take a few moments to reflect upon the images of the wilderness that grace the opening of the sections. Whether you stand before a majestic mountain, descend into a vast blue ocean, walk softly through a coniferous forest, or lie in a misty moonlit meadow and count shooting stars, you are at peace. Imagine the soothing sounds of a river, the caress of a snowflake, or the penetrating warmth of a desert rock. Wilderness is our greatest blessing, for it exists to support life, yet bestows uncommon beauty. We may manipulate and seek to conquer our environment, but in the end, it is always we who must adapt to the forces of nature. Wilderness medicine, then, exists on the premise that humans will encounter forces beyond their control. As we learn to keep ourselves healthy and whole in the outdoors, we come to appreciate the essences of healing and renewal. In medicine, there is service to the living. In wilderness medicine, there is service to humankind and our precious habitat.

Paul S. Auerbach, MD, MS

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1 High-Altitude Medicine

Peter H. Hackett and Robert C. Roach

Millions of people visit recreation areas above 2400 m in the American West each year. Hundreds of thousands visit central and south Asia, Africa, and South America, many traveling to altitudes over 4000 m.[311] In addition, millions live in large cities above 3000 m in South America and Asia. The population in the Rocky Mountains of North America has doubled in the past decade. Increasingly, physicians and other health care providers are confronted with questions of prevention and treatment of high-altitude medical problems (Box 1-1) as well as the effects of altitude on preexisting medical conditions. Despite advances in high-altitude medicine, significant morbidity and mortality persist (Table 1-1). Clearly, better education of the population at risk and of those advising them is essential. This chapter reviews the basic physiology of ascent to high altitude, as well as the pathophysiology, recognition, and management of medical problems associated with high altitude. The clinical issues likely to be encountered in lowlanders visiting high-altitude locations are emphasized, and medical problems of people living at high altitude are discussed (see Box 1-1).

DEFINITIONS

High Altitude (1500 to 3500 m*)

The onset of physiologic effects of diminished inspiratory oxygen pressure (P_{IO_2}) includes decreased exercise performance and increased ventilation (lower arterial P_{CO_2}) (Box 1-2). Minor impairment exists in arterial oxygen transport (arterial oxygen saturation [Sa_{O_2}] at least 90%), but arterial P_{O_2} is significantly diminished. Because of the large number of people who ascend rapidly to 2500 to 3500 m, high-altitude illness is common in this range (Table 1-2, and see Table 1-1).

Very High Altitude (3500 to 5500 m)

Maximum Sa_{O_2} falls below 90% as the arterial P_{O_2} falls below 60 mm Hg (Table 1-3 and Fig. 1-1). Extreme hypoxemia may occur during exercise, during sleep, and in the presence of high-altitude pulmonary edema or other acute lung conditions. Severe altitude illness occurs most commonly in this range.

Extreme Altitude (above 5500 m)

Marked hypoxemia, hypocapnia, and alkalosis are characteristic of extreme altitudes. Progressive deterioration of physiologic function eventually outstrips acclimatization. As a result, no permanent human habitation occurs above 5500 m. A period of acclimatization is necessary when ascending to extreme altitude; abrupt ascent without supplemental oxygen for other than brief exposures invites severe altitude illness.

*To convert meters to feet, multiply meters × 3.2808. To convert feet to meters, multiply feet × .3048.

ENVIRONMENT AT HIGH ALTITUDE

Barometric pressure falls with increasing altitude in a logarithmic fashion (see Table 1-2). Therefore, the partial pressure of oxygen (21% of barometric pressure) also decreases, resulting in the primary insult of high altitude: hypoxia. At approximately 5800 m, barometric pressure is one-half that at sea level, and on the summit of Mt. Everest (8848 m), the P_{IO_2} is approximately 28% that at sea level (see Figure 1-1 and Table 1-2).

The relationship of barometric pressure to altitude changes with the distance from the equator. Thus, polar regions afford greater hypoxia at high altitude in addition to extreme cold. West[467] has calculated that the barometric pressure on the summit of Mt. Everest (27° N latitude) would be about 222 mm Hg instead of 253 mm Hg if Everest were located at the latitude of Mt. McKinley (62° N). Such a difference, he claims, would be sufficient to render an ascent without supplemental oxygen impossible.

In addition to the role of latitude, fluctuations related to season, weather, and temperature affect the pressure–altitude relationship. Pressure is lower in winter than in summer. A low-pressure trough can reduce pressure 10 mm Hg in one night on Mt. McKinley, making climbers awaken "physiologically higher" by 200 m. The degree of hypoxia is thus directly related to the barometric pressure, not solely to geographic altitude.[467]

Temperature decreases with altitude (an average of 6.5° C per 1000 m [3.6° F per 1000 ft]), and the effects of cold and hypoxia are generally additive in provoking both cold injuries and high-altitude pulmonary edema.[351,462] Ultraviolet light penetration increases approximately 4% per 300-m gain in altitude, increasing the risk of sunburn, skin cancer, and snowblindness. Reflection of sunlight in glacial cirques and on flat glaciers can cause intense radiation of heat in the absence of wind. The authors have observed temperatures of 40° to 42° C (104° to 108° F) in tents on both Mt. Everest and Mt. McKinley. Heat problems, primarily heat exhaustion, are often unrecognized in this usually cold environment. Physiologists have not yet examined the consequences of heat stress or rapid, extreme changes in environmental temperature combined with the hypoxia of high altitude.

Above the snow line is the "high-altitude desert," where water can be obtained only by melting snow or ice. This factor, combined with increased water loss through the lungs from increased respiration and through the skin, commonly results in dehydration that may be debilitating. Thus, the high-altitude environment imposes multiple stresses, some of which may contribute to or be confused with the effects of hypoxia.

BOX 1-1. Medical Problems of High Altitude

LOWLANDERS ON ASCENT TO HIGH ALTITUDE

- Acute hypoxia
- High-altitude headache
- Acute mountain sickness
- High-altitude cerebral edema
- Cerebrovascular syndromes
- High-altitude pulmonary edema
- High-altitude deterioration
- Organic brain syndrome
- Peripheral edema
- Retinopathy
- Disordered sleep
- Sleep periodic breathing
- High-altitude pharyngitis and bronchitis
- Ultraviolet keratitis (snowblindness)
- Exacerbation of preexisting conditions

LIFE-LONG OR LONG-TERM RESIDENTS OF HIGH ALTITUDE

- Chronic mountain sickness (chronic mountain polycythemia)
- High-altitude pulmonary hypertension, with or without right heart failure
- Problems of pregnancy: preeclampsia, hypertension, and low-birth-weight infants
- Exacerbation of common illnesses, such as lung disease

ACCLIMATIZATION TO HIGH ALTITUDE

Rapid ascent from sea level to the altitude at the summit of Mt. Everest (8848 m) causes loss of consciousness in a few minutes and death shortly thereafter. Yet climbers ascending Mt. Everest over a period of weeks, without supplemental oxygen, have experienced only minor symptoms of illness. The process by which individuals gradually adjust to hypoxia and enhance survival and performance is termed acclimatization. A complex series of physiologic adjustments increases oxygen delivery to cells and also improves their hypoxic tolerance. The severity

BOX 1-2. Glossary of Physiologic Terms

P_B	Barometric pressure*
P_{O_2}	Partial pressure of oxygen
P_{IO_2}	Partial pressure of inspired oxygen ($0.21 \times [P_B - 47\,mm\,Hg]$) ($47\,mm\,Hg$ = vapor pressure of H_2O at 37°C)
PA_{O_2}	P_{O_2} in alveolus
PA_{CO_2}	P_{CO_2} in alveolus
Pa_{O_2}	P_{O_2} in arterial blood
Pa_{CO_2}	P_{CO_2} in arterial blood
Sa_{O_2}%	Arterial oxygen saturation ($HbO_2 \div$ total Hb $\times$ 100)
R	Respiratory quotient (CO_2 produced $\div$ O_2 consumed)
Alveolar gas equation	$PA_{O_2} = P_{IO_2} - (PA_{CO_2}/R)$

*Pressures are expressed as mm Hg (1 mm Hg = 1 torr).

TABLE 1-1. Incidence of Altitude Illness in Various Groups

STUDY GROUP	NUMBER AT RISK PER YEAR	SLEEPING ALTITUDE (m)	MAXIMUM ALTITUDE REACHED (m)	AVERAGE RATE OF ASCENT*	PERCENT WITH AMS	PERCENT WITH HAPE AND/OR HACE	REFERENCES
Western State Visitors	30 million	~2000 ~2500 ~≥3000	3500	1–2	18–20 22 27–42	0.01	183
Mt. Everest Trekkers	15,000	3000–5200	5500	1–2 (fly in) 10–13 (walk in)	47 23 30–50	1.6 0.05	155 — 321
Mt. McKinley Climbers	1200	3000–5300	6194	3–7	30	2–3	147
Mt. Rainier Climbers	10,000	3000	4392	1–2	67	—	250
Mt. Rosa, Swiss Alps	†	2850 4559	2850 4559	1–2 2–3	7 27	— 5	273 83, 273, 401
Indian Soldiers	Unknown	3000–5500	5500	1–2	†	2.3–15.5	420, 421
Aconcagua Climbers	4200	3300–5800	6962	2–8	39 (LLS > 4)	2.2	341

*Days to sleeping altitude from low altitude.
†Reliable estimate unavailable.
AMS, acute mountain sickness; HACE, high-altitude cerebral edema; HAPE, high-altitude pulmonary edema; LLS, Lake Louise score.

TABLE 1-2. Altitude Conversions
Barometric Pressure (P_B), Estimated Partial Pressure Inspired Oxygen (P_{IO_2}), and the Equivalent Oxygen Concentration at Sea Level (F_{IO_2} at SL)*

m	ft	P_B	P_{IO_2}	F_{IO_2} AT SL
Sea level	Sea level	759.6	149.1	0.209
1,000	3,281	678.7	132.2	0.185
1,219	4,000	661.8	128.7	0.180
1,500	4,921	640.8	124.3	0.174
1,524	5,000	639.0	123.9	0.174
1,829	6,000	616.7	119.2	0.167
2,000	6,562	604.5	116.7	0.164
2,134	7,000	595.1	114.7	0.161
2,438	8,000	574.1	110.3	0.155
2,500	8,202	569.9	109.4	0.154
2,743	9,000	553.7	106.0	0.149
3,000	9,843	536.9	102.5	0.144
3,048	10,000	533.8	101.9	0.143
3,353	11,000	514.5	97.9	0.137
3,500	11,483	505.4	95.9	0.135
3,658	12,000	495.8	93.9	0.132
3,962	13,000	477.6	90.1	0.126
4,000	13,123	475.4	89.7	0.126
4,267	14,000	460.0	86.4	0.121
4,500	14,764	446.9	83.7	0.117
4,572	15,000	442.9	82.9	0.116
4,877	16,000	426.3	79.4	0.111
5,000	16,404	419.7	78.0	0.109
5,182	17,000	410.2	76.0	0.107
5,486	18,000	394.6	72.8	0.102
5,500	18,045	393.9	72.6	0.102
5,791	19,000	379.5	69.6	0.098
6,000	19,685	369.4	67.5	0.095
6,096	20,000	364.9	66.5	0.093
6,401	21,000	350.7	63.6	0.089
6,500	21,325	346.2	62.6	0.088
6,706	22,000	337.0	60.7	0.085
7,000	22,966	324.2	58.0	0.081
7,010	23,000	323.8	57.9	0.081
7,315	24,000	310.9	55.2	0.077
7,500	24,606	303.4	53.7	0.075
7,620	25,000	298.6	52.6	0.074
7,925	26,000	286.6	50.1	0.070
8,000	26,247	283.7	49.5	0.069
8,230	27,000	275.0	47.7	0.067
8,500	27,887	265.1	45.6	0.064
8,534	28,000	263.8	45.4	0.064
8,839	29,000	253.0	43.1	0.060
8,848	29,029	252.7	43.1	0.060
9,000	29,528	247.5	42.0	0.059
9,144	30,000	242.6	40.9	0.057
9,500	31,168	230.9	38.5	0.054
10,000	32,808	215.2	35.2	0.049

*Barometric pressure is approximated by the equation $P_B = \text{Exp}(6.6328 - \{0.1112 \times \text{altitude} - [0.00149 \times (\text{altitude}^2)]\})$, where altitude = terrestrial altitude in meters/1000, or km. P_{IO_2} is calculated as (P_B – 47) (where 47 = water vapor pressure at body temperature) × fraction of O_2 in inspired air. The equivalent F_{IO_2} at sea level for a given altitude is calculated as $P_{IO_2} \div (760 - 47)$. Substituting ambient P_B for 760 in the equation allows similar calculations for F_{IO_2} at different altitudes. Exp, Exponent.
Meters = feet × .3048.
Feet = meters × 3.2808.

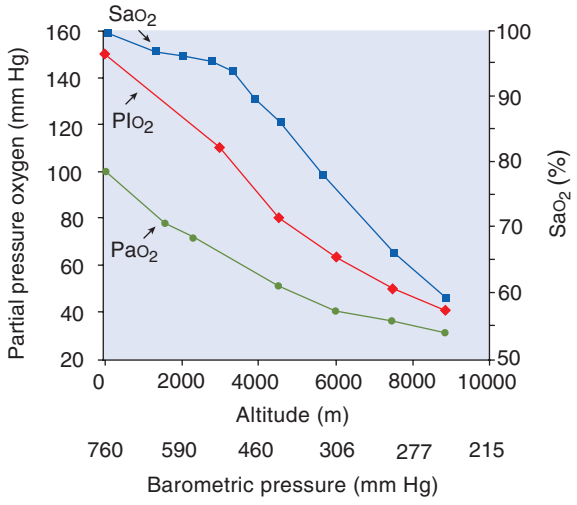

Figure 1-1. Increasing altitude results in a decrease in inspired P_{O_2} (P_{IO_2}), arterial P_{O_2} (Pa_{O_2}), and arterial oxygen saturation (Sa_{O_2}). Note that the difference between P_{IO_2} and Pa_{O_2} narrows at high altitude because of increased ventilation, and that Sa_{O_2} is well maintained while awake until over 3000 m. (Data from Morris A: Clinical pulmonary function tests: A manual of uniform lab procedures. Intermountain Thoracic Society, 1984, and Sutton JR, Reeves JT, Wagner PD, et al: Operation Everest II: Oxygen transport during exercise at extreme simulated altitude. J Appl Physiol 64:1309–1321, 1988, with permission.)

of hypoxic stress, rate of onset, and individual physiology determine whether the body successfully acclimatizes or is overwhelmed.

Individuals vary in their ability to acclimatize, no doubt reflecting certain genetic polymorphisms. Some adjust quickly, without discomfort, whereas acute mountain sickness (AMS) develops in others, who go on to recover. A small percentage of people fail to acclimatize even with gradual exposure over weeks. The tendency to acclimatize well or to become ill is consistent on repeated exposure if rate of ascent and altitude gained are similar, supporting the notion of important genetic factors. Successful initial acclimatization protects against altitude illness and improves sleep. Longer-term acclimatization (over weeks) primarily improves aerobic exercise ability. These adjustments disappear at a similar rate on descent to low altitude. A few days at low altitude may be sufficient to render a person susceptible to altitude illness on re-ascent, especially high-altitude pulmonary edema (HAPE). The improved ability to do physical work at high altitude, however, persists for weeks.[268] People who live at high altitude during growth and development appear to realize the maximum benefit of acclimatization changes; for example, their exercise performance matches that of persons at sea level.[312]

Ventilation

By reducing alveolar carbon dioxide, increased ventilation raises alveolar oxygen, improving oxygen delivery (Fig. 1-2, and see Figure 1-1). This response starts at an altitude as low as 1500 m (P_{IO_2} = 124 mm Hg; see Table 1-2) and within the first few minutes to hours of high-altitude exposure. The carotid body, sensing a decrease in arterial P_{O_2}, signals the central res-

TABLE 1-3. Blood Gases and Altitude

POPULATION	ALTITUDE (m)	ALTITUDE (ft)	P_B (mm Hg)	Pa_{O_2} (mm Hg)	Sa_{O_2} (%)	Pa_{CO_2} (mm Hg)
Altitude residents*	1,646	5,400	630	73.0 (65.0–83.0)	95.1 (93.0–97.0)	35.6 (30.7–41.8)
Acute exposure†	2,810	9,200	543	60.0 (47.4–73.6)	91.0 (86.6–95.2)	33.9 (31.3–36.5)
	3,660	12,020	489	47.6 (42.2–53.0)	84.5 (80.5–89.0)	29.5 (23.5–34.3)
	4,700	15,440	429	44.6 (36.4–47.5)	78.0 (70.8–85.0)	27.1 (22.9–34.0)
	5,340	17,500	401	43.1 (37.6–50.4)	76.2 (65.4–81.6)	25.7 (21.7–29.7)
	6,140	20,140	356	35.0 (26.9–40.1)	65.6 (55.5–73.0)	22.0 (19.2–24.8)
Chronic exposure	6,500	21,325	346	41.1 ± 3.3	75.2 ± 6	20 ± 2.8
during Operation	7,000	22,966	324	—	—	—
Everest II‡	8,000	26,247	284	36.6 ± 2.2	67.8 ± 5	12.5 ± 1.1
	8,848	29,029	253	30.3 ± 2.1	58 ± 4.5	11.2 ± 1.7
Acclimatized subjects studied during acute exposure to the simulated summit of Everest§	8,848	29,029	253	30.6 ± 1.4	—	11.9 ± 1.4

Data are mean values and (range) or ±SD, where available. All values are for subjects of age 20 to 40 years who were acclimatizing well.
*Data from reference 265.
†Data from reference 293.
‡Data from reference 434.
§Data from reference 360.

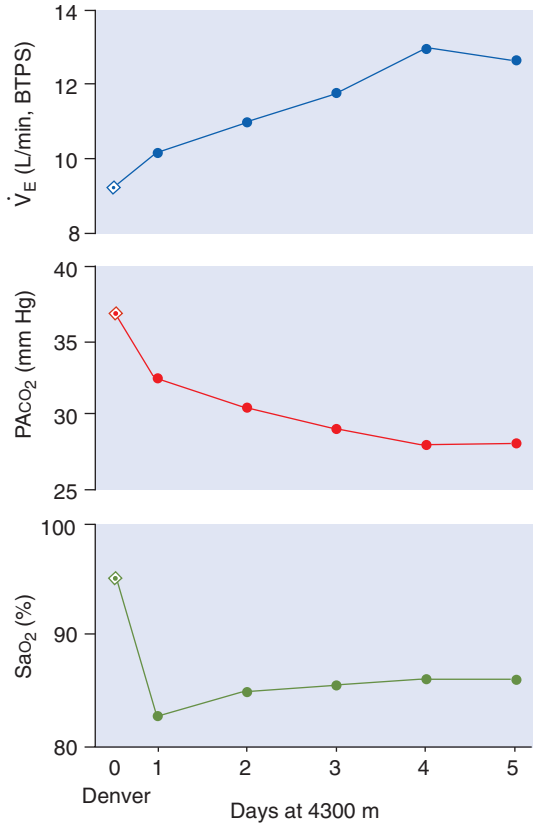

Figure 1-2. Change in minute ventilation ($\dot{V}_E$), alveolar carbon dioxide (PA_{CO_2}), and arterial oxygen saturation (Sa_{O_2}) during 5 days' acclimatization to 4300 m. BTPS, Body temperature, ambient pressure, saturated with water vapor. (Modified from Huang SY, Alexander JK, Grover RF, et al: J Appl Physiol 56:602–606, 1984, with permission.)

piratory center in the medulla to increase ventilation. This carotid body function (hypoxic ventilatory response, or HVR) is genetically determined[464] but influenced by a number of extrinsic factors. Respiratory depressants such as alcohol and soporifics, as well as fragmented sleep, depress the HVR. Agents that increase general metabolism, such as caffeine and coca, as well as specific respiratory stimulants, such as progesterone[242] and almitrine,[159] increase the HVR. (Acetazolamide, a respiratory stimulant, acts on the central respiratory center rather than on the carotid body.) Physical conditioning apparently has no effect on the HVR. Numerous studies have shown that a good ventilatory response enhances acclimatization and performance and that a very low HVR may contribute to illness[363] (see Acute Mountain Sickness, and High-Altitude Pulmonary Edema). However, over a normal range of values, the HVR is not a reliable predictor of susceptibility to altitude illness.

Other factors influence ventilation on ascent to high altitude. As ventilation increases, hypocapnia produces alkalosis, which acts as a braking mechanism on the central respiratory center and limits a further increase in ventilation. To compensate for the alkalosis, within 24 to 48 hours of ascent the kidneys excrete bicarbonate, decreasing the pH toward normal; ventilation increases as the negative effect of the alkalosis is removed. Ventilation continues to increase slowly, reaching a maximum only after 4 to 7 days at the same altitude (see Figure 1-2). The plasma bicarbonate concentration continues to drop and ventilation to increase with each successive increase in altitude. People with lower oxygen saturation at altitude have higher serum bicarbonate values; whether the kidney might be limiting acclimatization or whether this reflects poor respiratory drive is not clear.[86] This process is greatly facilitated by acetazolamide (see Acetazolamide Prophylaxis).

The paramount importance of hyperventilation is readily apparent from the following calculation: the alveolar P_{O_2} on the summit of Mt. Everest (about 33 mm Hg) would be reached at only 5000 m if alveolar P_{CO_2} stayed at 40 mm Hg, limiting an

ascent without supplemental oxygen to near this altitude. Table 1-3 gives the measured arterial blood gases resulting from acclimatization to various altitudes.

Circulation

The circulatory pump is the next step in the transfer of oxygen, moving oxygenated blood from the lungs to the tissues.

Systemic Circulation

Increased sympathetic activity on ascent causes an initial mild increase in blood pressure, a moderate increase in heart rate and cardiac output, and an increase in venous tone. Stroke volume is low because of decreased plasma volume, which drops as much as 12% over the first 24 hours[478] as a result of the bicarbonate diuresis, a fluid shift from the intravascular space, and suppression of aldosterone.[30] Resting heart rate returns to near sea level values with acclimatization, except at extremely high altitude. Maximal heart rate follows the decline in maximal oxygen uptake with increasing altitude. As the limits of hypoxic acclimatization are approached, maximal and resting heart rates converge. During Operation Everest II (OEII), cardiac function was appropriate for the level of work performed and cardiac output was not a limiting factor for performance.[349,431] Interestingly, myocardial ischemia at high altitude has not been reported in healthy persons, despite extreme hypoxemia. This is partly because of the reduction in myocardial oxygen demand from reduced maximal heart rate and cardiac output. Pulmonary capillary wedge pressure is low, and catheter studies have shown no evidence of left ventricular dysfunction or abnormal filling pressures in humans at rest.[140,207] On echocardiography, the left ventricle is smaller than normal because of decreased stroke volume, whereas the right ventricle may become enlarged.[431] The abrupt increase in pulmonary artery pressure can cause a change in left ventricular diastolic function, but because of compensatory increased atrial contraction, no overt diastolic dysfunction results.[5] In trained athletes doing an ultramarathon, the strenuous exercise at high altitude did not result in left ventricular damage; however, wheezing, reversible pulmonary hypertension, and right ventricular dysfunction occurred in a third of those completing the race and resolved within 24 hours.[91]

Pulmonary Circulation

A prompt but variable increase in pulmonary vascular resistance occurs on ascent to high altitude as a result of hypoxic pulmonary vasoconstriction, which increases pulmonary artery pressure. Mild pulmonary hypertension is greatly augmented by exercise, with pulmonary pressure reaching near-systemic values,[140] especially in people with a previous history of high-altitude pulmonary edema.[27,93] During OEII, Groves and colleagues[140] demonstrated that even when associated with a mean pulmonary artery pressure of 60 mm Hg, cardiac output remained appropriate and right atrial pressure did not rise above sea level values. Thus, right ventricular function was intact in spite of extreme hypoxemia and pulmonary hypertension.

Administration of oxygen at high altitude does not completely restore pulmonary artery pressure to sea level values, an indication that increased pulmonary vascular resistance does not result solely from hypoxic vasoconstriction.[274] The explanation is likely to be vascular remodeling with medial hypertrophy. See Stenmark and colleagues for an excellent review of molecular and cellular mechanisms of the pulmonary vascular response to hypoxia, including remodeling.[427] Pulmonary vascular resistance returns to normal within days to weeks after descent to low altitude.

Cerebral Circulation

Cerebral oxygen delivery is the product of arterial oxygen content and cerebral blood flow (CBF) and depends on the net balance between hypoxic vasodilation and hypocapnia-induced vasoconstriction. CBF increases, despite the hypocapnia, when Pa_{O_2} is less than 60 mm Hg (altitude greater than 2800 m). In a classic study, CBF increased 24% on abrupt ascent to 3810 m and then returned to normal over 3 to 5 days.[412] More recent studies have shown considerable individual variation[39,41,218] and also an impairment of cerebral autoregulation.[216,260,450] The individual variation in cerebral blood flow is linked to individual variation in the ventilatory response to hypoxia.[2] Regional brain tissue oxygenation assessed by near-infrared spectroscopy reveals mild tissue hypoxia.[165,212,388] Overall, global cerebral metabolism is well maintained with moderate hypoxia.[87,304]

Blood

Hematopoietic Responses to Altitude

Ever since the observation in 1890 by Viault[453] that hemoglobin concentration was higher than normal in animals living in the Andes, scientists have regarded the hematopoietic response to increasing altitude as an important component of the acclimatization process. On the other hand, hemoglobin concentration apparently has no relationship to susceptibility to high-altitude illness on initial ascent, although this has not been adequately studied.

In response to hypoxemia, erythropoietin is secreted and stimulates bone marrow production of red blood cells.[409] The hormone is detectable within 2 hours of ascent, nucleated immature red blood cells can be found on a peripheral blood smear within days, and new red blood cells are in circulation within 4 to 5 days. Over a period of weeks to months, red blood cell mass increases in proportion to the degree of hypoxemia. Iron supplementation can be important: women who take supplemental iron at high altitude approach the hematocrit values of men at altitude[169] (Fig. 1-3).

The increase in hemoglobin concentration seen 1 to 2 days after ascent is due to hemoconcentration secondary to decreased plasma volume, rather than a true increase in red blood cell mass. This results in a higher hemoglobin concentration at the cost of decreased blood volume, a tradeoff that might impair exercise performance. Longer-term acclimatization leads to an increase in plasma volume as well as in red blood cell mass, thereby increasing total blood volume. Overshoot of the hematopoietic response causes excessive polycythemia, which may actually impair oxygen transport because of increased blood viscosity. Although the "ideal" hematocrit at high altitude is not established, phlebotomy is often recommended when hematocrit values exceed 60% to 65%. During the American Medical Research Expedition to Mt. Everest (AMREE), hematocrit was reduced by hemodilution from 58% ± 1.3% to 50.5% ± 1.5% at 5400 m with no decrement in maximal oxygen uptake and an increase in cerebral functioning.[393]

Oxyhemoglobin Dissociation Curve

The oxyhemoglobin dissociation curve (ODC) plays a crucial role in oxygen transport. Because of the sigmoidal shape of the

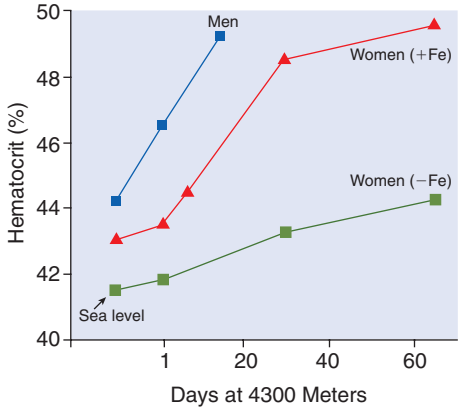

Figure 1-3. Hematocrit changes on ascent to altitude in men and in women, with and without supplemental iron (Fe). (Modified from Hannon JP, Chinn KS, Shields JL: Fed Proc 28:1178–1184, 1969, with permission.)

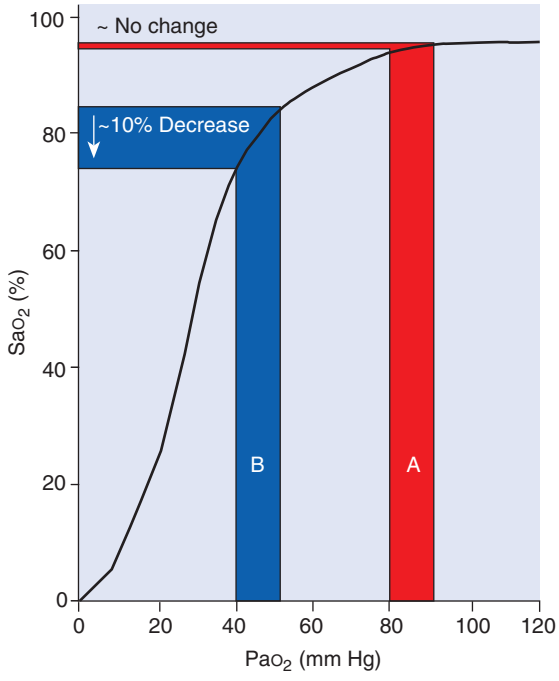

Figure 1-4. Oxyhemoglobin dissociation curve showing effect of 10 mm Hg decrement in Pao_2 on arterial oxygen saturation at sea level **(A)** and near 4400 m **(B)**. Note the much larger drop in Sao_2 at high altitude. (Modified from Severinghaus JW, Chiodi H, Eger EI, et al: Circ Res 19:274–282, 1966, with permission.)

curve, Sao_2% is well maintained up to 3000 m, despite a significant decrease in arterial Po_2 (see Figure 1-1). Above that altitude, small changes in arterial Po_2 result in large changes in arterial oxygen saturation (Fig. 1-4). The oxygen saturation determines arterial oxygen transport, but the Po_2 determines diffusion of oxygen from the capillary to the cell.

In 1936, Ansel Keys and colleagues[230] demonstrated an in vitro right shift in the position of the ODC at high altitude, a shift that favors release of oxygen from blood to the tissues. This change occurs because of the increase in 2,3-diphosphoglycerate (2,3-DPG), which is proportional to the severity of hypoxemia. In vivo, however, this is offset by alkalosis, and at moderate altitude little net change occurs in the position of the ODC. On the other hand, the marked alkalosis of extreme hyperventilation, as measured on the summit and simulated summit of Mt. Everest (Pco_2 8 to 10 mm Hg, pH > 7.6), shifts the ODC to the left, which facilitates oxygen–hemoglobin binding in the lung, raises Sao_2%, and is thought to be advantageous.[391] This concept is further supported by observing that when people with a very left-shifted ODC, caused by an abnormal hemoglobin (Andrew-Minneapolis), were taken to moderate (3100 m) altitude, they had less tachycardia and dyspnea and remarkably had no decrease in exercise performance.[175] High-altitude adapted animals also have a left-shifted ODC.

Tissue Changes

The next link in the oxygen transport chain is tissue oxygen transfer, which depends on capillary perfusion, diffusion distance, and driving pressure of oxygen from the capillary to the cell. The final link, then, is use of oxygen within the cell. Banchero[15] has shown that capillary density in dog skeletal muscle doubled in 3 weeks at a barometric pressure of 435 mm Hg. A recent study in humans noted neither change in capillary density, nor in gene expression thought to enhance muscle vascularity.[267] Ou and Tenney[334] revealed a 40% increase in mitochondrial number but no change in mitochondrial size, whereas the study of Oelz and colleagues[332] showed that high-altitude climbers had normal mitochondrial density. A significant drop in muscle size is often noted after a high-altitude expedition due to net energy deficit,[267,270] and it results in shortening of the diffusion distance for oxygen. This occurs in spite of no de novo synthesis of capillaries or mitochondria, yet results in increased capillary density and ratio of mitochondrial volume to contractile protein fraction, which are primarily a result of the atrophy.[267,270]

Sleep at High Altitude

Disturbed sleep is common at high altitude. Its cause appears to be multifactorial. Reflecting the great interest in sleep in general, more than 140 papers have addressed sleep at altitude in the last 10 years; a complete discussion is outside our purview. The interested reader is referred to recent reviews and online databases (Medline).[454,463] Nearly all subjects complain of disturbed sleep at high altitude, with severity increasing with the altitude. At moderate altitude, sleep architecture is changed, with reduction in stage 3 and 4 sleep, increase in stage 1 time, and little change in stage 2. Overall, there is a shift from deeper sleep to lighter sleep. In addition, more time is spent awake, with significantly increased arousals. Authors have reported either slightly less rapid eye movement (REM) time, or no change in REM compared to low altitude. REM sleep may improve over time at altitude.[232] The subjective complaints of poor sleep are out of proportion to the small reduction (if any) in total sleep time, and appear to be due to sleep fragmentation. With more extreme hypoxia, sleep time was dramatically short-

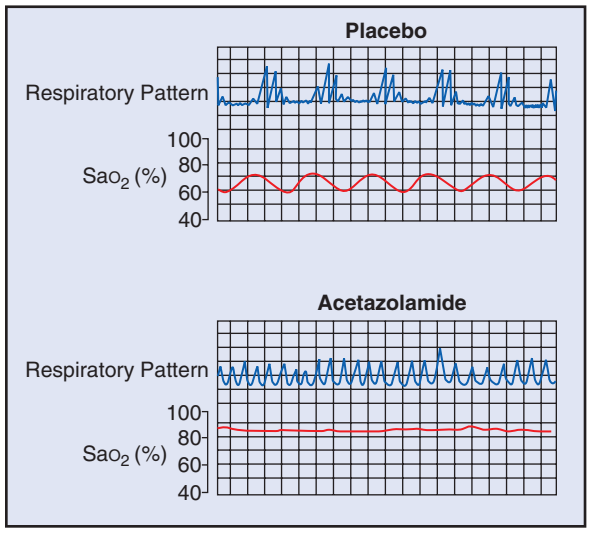

Figure 1-5. Respiratory patterns and arterial oxygen saturation ($Sa0_2$) with placebo and acetazolamide in two sleep studies of a subject at 4200 m. Note pattern of hyperpnea followed by apnea during placebo treatment, which is changed with acetazolamide. (Modified from Hackett PH, Roach RC, Harrison GL, et al: Am Rev Respir Dis 135:896–898, 1987, with permission.)

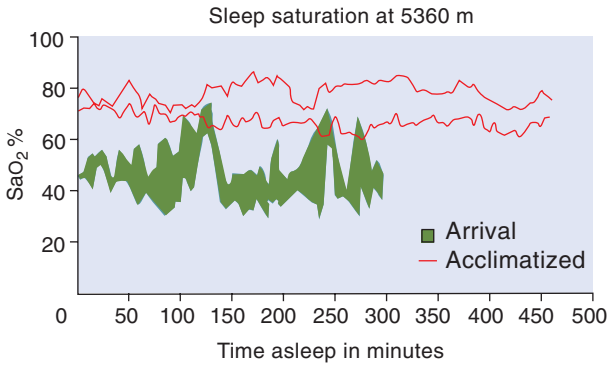

Figure 1-6. Sleep oxygenation improves with acclimatization to same altitude. *Top line* is maximum and *bottom line* is minimum $Sa0_2$ in an acclimatized person. *Shaded area* shows maximum and minimum $Sa0_2$ values for new arrival at 5360 m (17,581 feet). (Modified from Sutton JR, Houston CS, Mansell AL, et al: N Engl J Med 301:1329–1331, 1979, with permission.)

ened and arousals increased, without a change in ratio of sleep stages but with a reduction in REM sleep.[8] The mechanisms of this change in sleep architecture and fragmentation are poorly understood. Periodic breathing appears to play only a minor role in altering sleep architecture at high altitude.[390] The arousals have been linked to periodic breathing in some studies but not others. Other factors might include change in circadian rhythm and perhaps body temperature.[82] Problematic sleep is quite variable, with predisposing factors such as obesity explaining a degree of susceptibility to both deranged sleep and sleep-disordered breathing in some individuals.[125] Recent studies of infants and children[488] and athletes in simulated altitude devices used for training also revealed deranged sleep quality in these groups.[233,234,337] Although deranged sleep is a frequent complaint in high-altitude visitors, it seems to have little relation to susceptibility to altitude illness or other serious problems. Symptomatic treatment that avoids respiratory depression is safe. (See Treatment under Acute Mountain Sickness section.)

Periodic Breathing

Periodic breathing is most common in early and light sleep, may occur during wakefulness when drowsy, and does not occur in REM sleep. The pattern is characterized by hyperpnea followed by apnea (Fig. 1-5), and it is caused by a battle for control of breathing between the peripheral chemoreceptors (carotid body) and the central respiratory center. Respiratory alkalosis during the hyperpnea acts on the central respiratory center, causing apnea. During apnea, Sao_2% decreases, carbon dioxide increases, and the carotid body is stimulated, causing a recurrent hyperpnea and apnea cycle. The apnea is central, not associated with snoring, and with absence of rib cage movement. Persons with a high hypoxic ventilatory response have more periodic breathing, with mild oscillations in Sao_2%,[246] whereas persons with a low hypoxic ventilatory response have more regular breathing overall but may suffer periods of apnea with extreme hypoxemia distinct from periodic breathing.[159] As acclimatization progresses, periodic breathing lessens but does not disappear, especially over 5000 m, and Sao_2% increases (Fig. 1-6).[8,432] Periodic breathing has not been implicated in the etiology of high-altitude illness, but nocturnal oxygen desaturation has been implicated.[111,121] Eichenberger and colleagues have also reported greater periodic breathing in persons with HAPE, secondary to lower Sao_2%.[108] As with fragmented sleep, the intensity of periodic breathing is quite variable. Total sleep time with periodic breathing can vary from 1% to over 90%.[494] Most studies report no association between periodic breathing and AMS. This may relate to the fact that persons with periodic breathing tend to have higher HVR, and greater average ventilation and oxygenation.[463]

Pharmaceutical Aids

Acetazolamide, 125 mg at bedtime, diminishes periodic breathing and awakenings, improves oxygenation and sleep quality, and is a safe and superior agent to use as a sleeping aid (see Figure 1-5). It has the added benefit of diminishing symptoms of AMS. Other agents include diphenhydramine (Benadryl, 50 to 75 mg) or the short-acting benzodiazepines such as triazolam (Halcion, 0.125 to 0.25 mg) and temazepam (Restoril, 15 mg). Although caution is warranted for any agent that might reduce ventilation at high altitude, some studies have suggested that benzodiazepines in low dosages are generally safe in this situation.[104,131,327] Another option is to use both acetazolamide and a benzodiazepine. Bradwell and colleagues[55] showed that acetazolamide (500 mg slow-release orally) given with temazepam (10 mg orally) improved sleep and maintained Sao_2%, counteracting a 20% decrease in Sao_2% when temazepam was given alone. The nonbenzodiazepine hypnotic zolpidem (Ambien, 10 mg) was shown to improve sleep at 4000 m without adversely affecting ventilation.[42]

Exercise

Maximal oxygen consumption drops dramatically on ascent to high altitude (see references 122 and 364 for recent reviews).

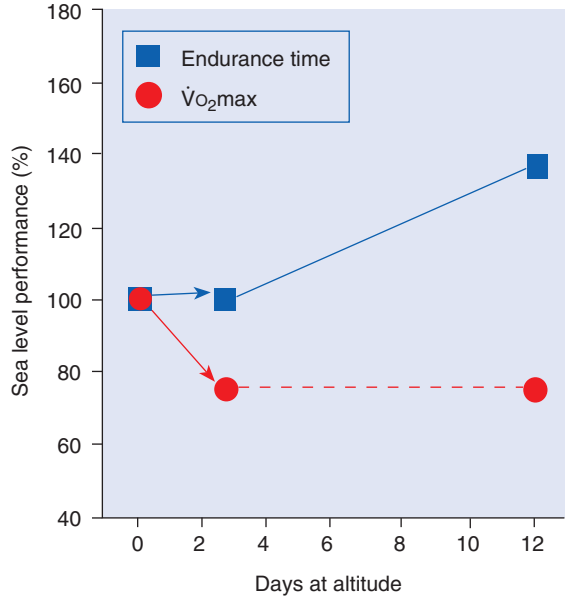

Figure 1-7. On ascent to altitude, $\dot{V}O_2max$ decreases and remains suppressed. In contrast, endurance time (minutes to exhaustion at 75% of altitude-specific $\dot{V}O_2max$) increases with acclimatization. (Modified from Maher JT, Jones LG, Hartley LH: J Appl Physiol 37:895–898, 1974, with permission.)

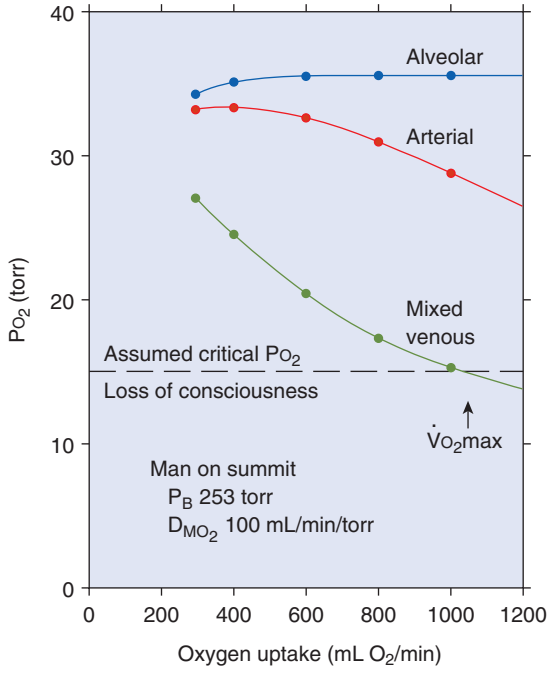

Figure 1-8. Calculated changes in the P_{O_2} of alveolar gas and arterial and mixed venous blood as oxygen uptake (mL O_2/min) is increased for a climber on the summit of Mt. Everest. Unconsciousness develops at a mixed venous P_{O_2} of 15 mm Hg. D_{MO_2}, muscle diffusing capacity for O_2. (Modified from West JB: Respir Physiol 52:265–279, 1983, with permission.)

Maximal oxygen uptake ($\dot{V}O_2max$) falls from sea level by approximately 10% for each 1000 m of altitude gained above 1500 m. Those with the highest sea level $\dot{V}O_2max$ values have the largest decrement in $\dot{V}O_2max$ at high altitude, but overall performance at high altitude is not consistently related to sea level $\dot{V}O_2max$.[332,358,469] In fact, many of the world's elite mountaineers have quite average $\dot{V}O_2max$ values, in contrast to other endurance athletes.[332] Acclimatization at moderate altitudes enhances submaximal endurance time but not $\dot{V}O_2max$ (Fig. 1-7).[122]

Oxygen transport during exercise at high altitude becomes increasingly dependent on the ventilatory pump. The marked rise in ventilation produces a sensation of breathlessness, even at low work levels. The following quotation is from a high-altitude mountaineer:

> *After every few steps, we huddle over our ice axes, mouths agape, struggling for sufficient breath to keep our muscles going. I have the feeling I am about to burst apart. As we get higher, it becomes necessary to lie down to recover our breath.*[297]

In contrast to the increase in ventilation with exercise, at increasing altitudes in OEII, cardiac function and cardiac output were maintained at or near sea level values for a given oxygen consumption (workload).[349] Recent work has attributed the altitude-induced drop in $\dot{V}O_2max$ to (1) the lower P_{IO_2}, (2) impairment of pulmonary gas exchange, and (3) reduction of maximal cardiac output and peak leg blood flow, each explaining about one third of the loss in $\dot{V}O_2max$.[65] However, mechanisms to explain impairment of gas exchange and lower blood flow remain elusive. Wagner[459] proposes that the pressure gradient for diffusion of oxygen from capillaries to the working muscle cells may be inadequate. Another concept is that increased cerebral hypoxia from exercise-induced desaturation is the limiting factor.[75,211] Mountaineers, for example, become lightheaded and their vision dims when they move too quickly at extreme altitude (Fig. 1-8).[466]

Training at High Altitude

Optimal training for increased performance at high altitude depends on the altitude of residence and the athletic event. For aerobic activities (events lasting more than 3 or 4 minutes) at altitudes above 2000 m, acclimatization for 10 to 20 days is necessary to maximize performance.[88] For events occurring above 4000 m, acclimatization at an intermediate altitude is recommended. Highly anaerobic events at intermediate altitudes require only arrival at the time of the event, although mountain sickness may become a problem.

The benefits of training at high altitude for subsequent performance at or near sea level depend on choosing a training altitude that maximizes the benefits and minimizes the "detraining" inevitable when maximal oxygen uptake is limited (altitude greater than 1500 to 2000 m). Hence, data from training above 2400 m have shown no increase in subsequent sea level performance. Also, intermittent exposures to hypoxia seem to have no benefit.[221,445] Runners returning to sea level after 10 days' training at 2000 m had faster running times and an increase in aerobic power, plasma volume, and hemoglobin concentration.[14] More recent work suggests training benefits from training at low altitude while sleeping at high altitude—the "live

high, train low" approach.[258,429] Coaches and endurance athletes from around the world are convinced of the benefits of training and/or sleeping at moderate altitude to improve sea level performance. The benefit appears due to enhanced erythropoietin production and increased red cell mass, which requires adequate iron stores and thus usually iron supplementation.[279,374,428] Some individuals do not respond to "live high, train low," perhaps because of an inability to increase erythropoietin levels sufficient to raise red cell mass and thus increase oxygen carrying capacity.[68,217]

HIGH-ALTITUDE SYNDROMES

High-altitude syndromes are illnesses attributed directly to hypobaric hypoxia, even if exact mechanisms are unclear. Clinically and pathophysiologically, these syndromes overlap. Rapidity of onset of hypoxia is crucial; for example, sudden exposure to extreme altitude may result in death from acute hypoxia (asphyxia), whereas more gradual ascent to the same altitude may result in AMS or no illness at all. Where symptoms of acute hypoxia end and AMS begins is vague, as reflected in the classic experiments of Bert.[49] In terms of altitude illness, the general concept of a spectrum of illness with a common underlying pathogenesis is well accepted and provides a useful framework for discussion. For the neurologic syndromes, the spectrum progresses from high-altitude headache to AMS to high-altitude cerebral edema (HACE). In the lung, the spectrum includes pulmonary hypertension, interstitial edema, and HAPE. These problems all occur within the first few days of ascent to a higher altitude, have many common features, and respond to descent or oxygen. Longer-term problems of altitude exposure include high-altitude deterioration and chronic mountain sickness.

Neurologic Syndromes

Neurologic syndromes at high altitude reflect both the nervous system's sensitivity to hypoxia and the effects of compensatory mechanisms. Although brain ATP production and metabolism remain intact during hypoxia, neurotransmitter synthesis is sensitive to hypoxia, and impaired synthesis of neurotransmitters accounts for symptoms of acute cerebral hypoxia as well as cognitive defects at high altitude. The compensatory cerebral vasodilation at altitude appears to play a role in causing altitude headache, and it also contributes to development of AMS and HACE. However, much of the pathophysiology of AMS and HACE remains a mystery. Focal neurologic deficits without cerebral edema are also difficult to explain; cerebral artery spasm and watershed hypoxia have both been invoked. The understanding of the exact etiology of these neurologic syndromes due to hypoxia will parallel advances in neuroscience; the emphasis in altitude illness is finally and appropriately on the brain.[368]

Acute Cerebral Hypoxia

Acute, profound hypoxia, although of greatest interest in aviation medicine, may also occur on terra firma when ascent is too rapid or when hypoxia abruptly worsens. Carbon monoxide poisoning, pulmonary edema, sudden overexertion, sleep apnea, or a failed oxygen delivery system may rapidly exaggerate hypoxemia. In an unacclimatized person, loss of consciousness from acute hypoxia occurs at an Sa_{O_2} of 40% to 60% or at an arterial P_{O_2} of less than about 30 mm Hg. Tissandier, the sole survivor of the flight of the balloon Zenith in 1875, gave a graphic description of the effects of acute hypoxia:

> *But soon I was keeping absolutely motionless, without suspecting that perhaps I had already lost use of my movements. Towards 7,500 m, the numbness one experiences is extraordinary. The body and the mind weakens little by little, gradually, unconsciously, without one's knowledge. One does not suffer at all; on the contrary. One experiences inner joy, as if it were an effect of the inundating flood of light. One becomes indifferent; one no longer thinks of the perilous situation or of the danger; one rises and is happy to rise. Vertigo of the lofty regions is not a vain word. But as far as I can judge by my personal impression, this vertigo appears at the last moment; it immediately precedes annihilation—sudden, unexpected, irresistible. I wanted to seize the oxygen tube, but could not raise my arm. My mind, however, was still very lucid. I was still looking at the barometer; my eyes were fixed on the needle which soon reached the pressure number of 280, beyond which it passed. I wanted to cry out "We are at 8,000 meters." But my tongue was paralyzed. Suddenly I closed my eyes and fell inert, completely losing consciousness.*[49]

The ascent to over 8000 m took 3 hours, and the descent less than 1 hour. When the balloon landed, Tissandier's two companions were dead.

The prodigious work that Paul Bert conducted in an altitude chamber during the 1870s showed that lack of oxygen, rather than an effect of isolated hypobaria, explained the symptoms experienced during rapid ascent to extreme altitude.

> *There exists a parallelism to the smallest details between two animals, one of which is subjected in normal air to a progressive diminution of pressure to the point of death, while the other breathes, also to the point of death, under normal pressure, an air that grows weaker and weaker in oxygen. Both will die after having presented the same symptoms.*[49]

Bert goes on to describe the symptoms of acute exposure to hypoxia:

> *It is the nervous system which reacts first. The sensation of fatigue, the weakening of the sense perceptions, the cerebral symptoms, vertigo, sleepiness, hallucinations, buzzing in the ears, dizziness, pricklings . . . are the signs of insufficient oxygenation of central and peripheral nervous organs. . . . The symptoms . . . disappear very quickly when the balloon descends from the higher altitudes, very quickly also . . . the normal proportion of oxygen reappears in the blood. There is an unfailing connection here.*[49]

Bert was also able to both prevent and immediately resolve symptoms by breathing oxygen. Modern studies of acute hypoxic exposure in simulated altitude chambers use the measurement of time of useful consciousness; that is, the time until a subject can no longer take corrective measures, such as putting on an oxygen mask. With exposure to 8500 m (28,000 feet), that time is 60 seconds during moderate activity and 90 seconds at rest.

Acute hypoxia can be quickly reversed by immediate administration of oxygen, rapid pressurization or descent, or correc-

tion of an underlying cause, such as relief of apnea, removal of a carbon monoxide source, repair of an oxygen delivery system, or cessation of overexertion. Hyperventilation increases time of useful consciousness during severe hypoxia.

High-Altitude Headache

Headache is generally the first unpleasant symptom consequent to altitude exposure and is sometimes the only symptom.[183] It may or may not be the harbinger of AMS, which is defined as the presence of headache plus at least one of four other symptoms, in the setting of an acute altitude gain.[365] One could even argue that it is the headache itself that causes other symptoms such as anorexia, nausea, lassitude, and insomnia, as is commonly seen in migraine or tension headaches, and that mild AMS is essentially due to headache. The International Headache Society (IHS) has defined high-altitude headache as "a headache that develops within 24 hours after sudden ascent to altitudes above 3,000m" and that is "associated with at least one other symptom typical of high altitude, including (a) Cheyne-Stokes respiration at night, (b) a desire to overbreathe and (c) exertional dyspnea." This definition is problematic in that many experience altitude headache between 2000 and 3000 m, and the other symptoms have no demonstrated association with headache.

Recent studies have attempted to characterize the clinical features and incidence of headache at altitude. Silber and colleagues found that 50 of 60 trekkers (83%) in Nepal up to 5100 m developed at least one headache when over 3000 m.[416] Older persons were less susceptible; women and those with headaches in daily life had more severe headaches but not more headaches than others. Of those with headache, 52% did not have AMS by the Lake Louise criteria; 44% did not have one of the symptoms in the IHS definition, indicating its ineffectiveness. Various medications alleviated the headache 70% of the time, especially when it was mild. The clinical features were widely variable. In general, the headaches were bilateral, generalized, dull, and exacerbated by exertion or movement, and they often occurred at night and resolved within 24 hours. Thus, the headaches had some features of increased intracranial pressure. Persons with history of migraine did not have a higher incidence of headache.

The term *high altitude headache* (HAH) has been used in the literature for decades, and studies directed toward the pathophysiology and treatment of HAH have been reported. Obviously, these are, to an extent, studies of AMS as well. Headache lends itself to investigation better than some other symptoms do, as headache scores have been well validated.[214] In general, the literature suggests that HAH can be prevented by the use of nonsteroidal anti-inflammatory drugs[57,61] and acetaminophen[172] as well as the drugs commonly used for prophylaxis of AMS, acetazolamide and dexamethasone. Some agents appear more effective than others, with ibuprofen and aspirin apparently superior to naproxen.[57,58,62] A serotonin agonist (sumatriptan, a 5-HT_1 [serotonin type 1] receptor agonist) was reported to be effective for HAH prevention or treatment in some studies[59,448] but not in others.[23] Flunarizine, a specific calcium antagonist used for treatment of migraine, was not effective in one study.[40] Interestingly, oxygen is often immediately effective for HAH (within 10 minutes) in subjects with and without AMS, indicating a rapidly reversible mechanism of the headache.[21,158]

The response to many different agents might reflect multiple components of the pathophysiology or merely the nonspecific nature of analgesics. As Sanchez del Rio and Moskowitz have pointed out, different inciting factors for headache may result in a final common pathway, such that the response to different therapies is not necessarily related to the initial cause of the headache.[392] They recently provided a useful multifactorial concept of the pathogenesis of HAH, based on current understanding of headaches in general.[392] They suggest that the trigeminovascular system is activated at altitude by both mechanical and chemical stimuli (vasodilation, nitric oxide, and other noxious agents), and in addition, the threshold for pain is quite likely altered at high altitude (Fig. 1-9).[392] If AMS and especially HACE ensue, altered intracranial dynamics may also play a role, via compression or distention of pain-sensitive

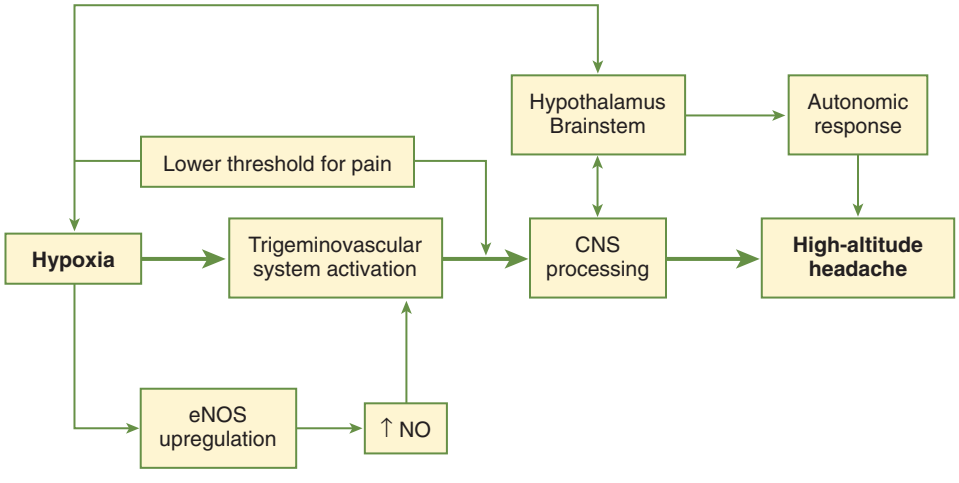

Figure 1-9. Proposed pathophysiology of high-altitude headache. CNS, central nervous system; eNOS, endothelial nitric oxide synthase; NO, nitric oxide. (Modified from Sanchez del Rio M, Moskowitz MA: High altitude headache. In Roach RC, Wagner PD, Hackett PH [eds]: Hypoxia: Into the Next Millennium. New York, Plenum/Kluwer Academic, 1999, pp 145–153, with permission.)

structures. The near future may bring a better understanding of the pathophysiology of these often debilitating headaches and new treatments as well.

Acute Mountain Sickness

Epidemiology and Risk Factors

Although the syndrome of AMS has been recognized for centuries, modern rapid transport and proliferation of participants in mountain sports have increased the number of victims and therefore public awareness (see Table 1-1). The incidence and severity of AMS depend on the rate of ascent and the altitude attained (especially the sleeping altitude), duration of altitude exposure, level of exertion, recent altitude exposure, and inherent physiologic (genetic) susceptibility.[155,370,401] For example, AMS is more common on Mt. Rainier because of the rapid ascent, whereas high-altitude pulmonary or cerebral edema is uncommon because of the short stay (less than 36 hours). People with a demonstrated susceptibility to AMS had twice the incidence of AMS compared to nonsusceptible people, and this was independent of rate of ascent.[401] The basis for inherent susceptibility is still unknown; clearly it depends on genetic factors. Recent altitude exposure can be protective; 5 days or more in the previous 2 months above 3000 m reduced susceptibility to AMS on ascent to 4559 m by one half, which was as effective as slow ascent[401] (Fig. 1-10). Compared with persons living at a lower altitude, residence at 900 m or above reduced the incidence of AMS from 27% to 8% when ascending to between 2000 and 3000 m in Colorado.[183] Age has an influence on incidence,[155] with those older than 50 years somewhat less vulnerable.[369,416] In a large study in Colorado, those older than 60 years had an incidence of AMS that was half that seen in those younger than 60. In contrast, a study of 827 mountaineers in Europe showed no influence of age on susceptibility.[401] Perhaps the different populations and physical activity explain the different results. No study has ever shown the older ages to be more susceptible. Children from 3 months to puberty studied in Colorado had the same incidence as young adults.[440,489] A small study of tourists in Chile, however, found lower oxygen saturation and higher AMS in the 6- to 48-month age group at 4440 m. The largest study of children to date, of Han Chinese after ascent to Tibet, showed essentially the same incidence of AMS in 464 children as in 5335 adults, 34% and 38%, respectively.[481] Women apparently have the same[371,401] or a slightly greater incidence of AMS[155,183,451] but may be less susceptible to pulmonary edema.[83,424] There appears to be no relationship between AMS and the menstrual cycle.[355] Most studies show no relationship between physical fitness and susceptibility to AMS. However, obesity seems to increase the odds of developing AMS.[124,183] Smoking does not increase risk of AMS,[183,225] and neither does use of oral contraceptives.[225,330] In summary, the most important variables related to AMS susceptibility are genetic predisposition, altitude of residence, rate of ascent, and prior recent altitude exposure.

Diagnosis

The diagnosis of AMS is based on the setting, symptoms, physical findings, and exclusion of other illnesses. The setting is generally rapid ascent of unacclimatized persons to 2500 m or

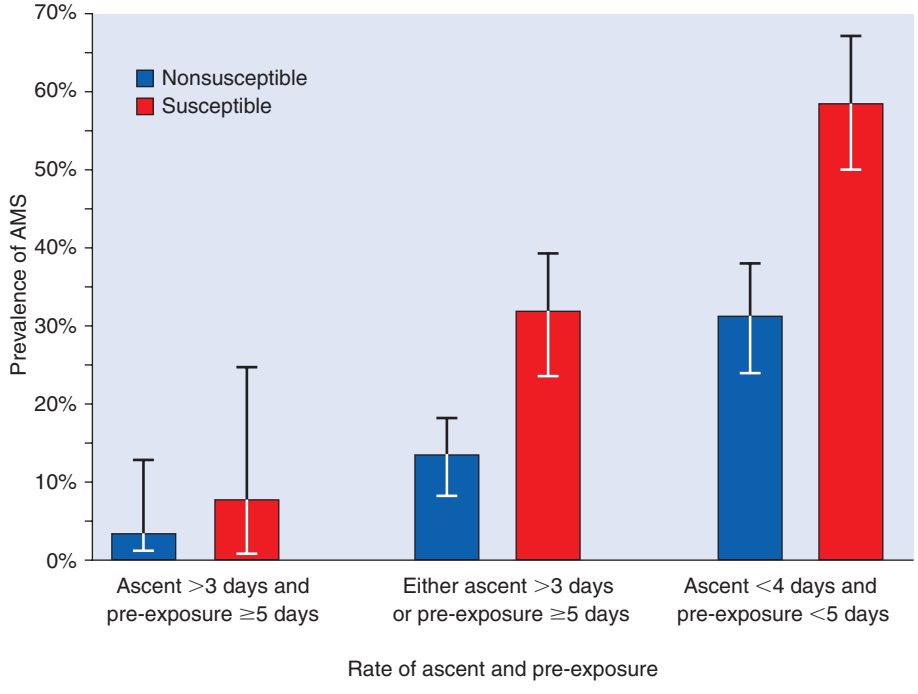

Figure 1-10. The prevalence of acute mountain sickness (AMS) and 95% confidence intervals in nonsusceptible *(blue bars)* and susceptible *(red bars)* mountaineers in relationship to the state of acclimatization defined as slow ascent (more than 3 days), fast ascent (3 days or less), pre-exposed (5 days or more above 3000 m in the preceding 2 months), and not pre-exposed (4 days or less above 3000 m in the preceding 2 months). From Schneider M, Bernasch D, Weymann J, et al: Med Sci Sports Exerc 34:1886–1891, 2002, with permission.)

higher from altitudes below 1500 m. For partially acclimatized persons, abrupt ascent to a higher altitude, overexertion, use of respiratory depressants, and perhaps onset of infectious illness[321] are common contributing factors.

The cardinal symptom of early AMS is headache, followed in incidence by fatigue, dizziness, and anorexia.[155,183,421] The headache is usually throbbing, bitemporal, worse during the night and on awakening, and made worse by Valsalva's maneuver or stooping over (see High-Altitude Headache). A good appetite is distinctly uncommon; nausea is common. These initial symptoms are strikingly similar to an alcohol hangover. Frequent awakening may fragment sleep, and periodic breathing often produces a feeling of suffocation. Although sleep disorder is nearly universal at high altitude, also affecting those without AMS, these symptoms may be exaggerated during AMS. Affected persons commonly complain of a deep inner chill, unlike mere exposure to cold temperature, accompanied by facial pallor. Other symptoms may include vomiting, dyspnea on exertion, and irritability. Lassitude can be disabling, with the victim too apathetic to contribute to his or her own basic needs.

Pulmonary symptoms vary considerably. Everyone experiences dyspnea on exertion at high altitude; it may be difficult to distinguish normal from abnormal. Dyspnea at rest is distinctly abnormal, however, and presages HAPE rather than AMS. Cough is also extremely common at high altitude, and not particularly associated with AMS. Recent work suggests that altitude hypoxia actually lowers the cough threshold, as measured with an inhaled citric acid stimulus.[283] However, any pulmonary symptom mandates careful examination for pulmonary edema.

Specific physical findings are lacking in mild AMS. A higher heart rate has been noted in those with AMS,[29,330] but Singh and associates[421] noted bradycardia (heart rate less than 66 beats/min) in two thirds of 1975 soldiers with AMS. Blood pressure is normal, but postural hypotension may be present. Occasionally, localized rales are present,[273] but this has also been observed in those without AMS.[153] A slightly increased body temperature with AMS may be present but is not diagnostic.[271] Peripheral oxygen saturation as measured by pulse oximetry correlated poorly with presence of AMS during rapid ascent[330,369,380] but was related to AMS during trekking.[34] SaO_2 at altitude on Denali was predictive of developing AMS on further ascent.[366] Overall, pulse oximetry is of limited usefulness in diagnosis of AMS. Funduscopic examination reveals venous tortuosity and dilation. Retinal hemorrhages may or may not be present and are not diagnostic; they are more common in AMS than non-AMS individuals at 4243 m.[153] Absence of the normal altitude diuresis, evidenced by lack of increased urine output and retention of fluid, is an early finding in AMS, although not always present.[30,154,373,421,435]

More obvious physical findings develop if AMS progresses to HACE. Typically, with onset of HACE, the victim wants to be left alone, lassitude progresses to inability to perform perfunctory activities such as eating and dressing, ataxia develops, and, finally, changes in consciousness appear, with confusion, disorientation, and impaired judgment. Coma may ensue within 24 hours of the onset of ataxia. Ataxia and confusion are the most useful signs for recognizing the progression from AMS to HACE; all persons proceeding to high altitudes should be aware of this fact. It is clinically useful to classify AMS as mild or as moderate to severe on the basis of symptoms (Table 1-4). Importantly, AMS can herald the beginning of life-threatening cerebral edema.

TABLE 1-4. Clinical Classifications of Acute Mountain Sickness (AMS)

	HIGH-ALTITUDE HEADACHE (HAH)	MILD AMS	MODERATE TO SEVERE AMS	HIGH-ALTITUDE CEREBRAL EDEMA (HACE)
Symptoms	Headache only	Headache, plus one more symptom (nausea/vomiting; fatigue/lassitude; dizziness or difficulty sleeping) All symptoms of mild intensity	Headache, plus one or more symptoms (nausea/vomiting; fatigue/lassitude; dizziness or difficulty sleeping) Symptoms of moderate to severe intensity	±Headache Worsening of symptoms seen in moderate to severe AMS
LL-AMS score*	1–3, headache only	2–4	5–15	—
Physical signs	None	None	None	Ataxia Altered mental status
Findings	None	None	Antidiuresis Slightly increased body temperature Slight desaturation Widened A-a gradient Elevated ICP White matter edema (CT, MRI)	HAPE common: positive chest film, rales, dyspnea at rest Elevated ICP White matter edema (CT, MRI)
Pathophysiology	Unknown; cerebral vasodilatation, trigeminovascular system†?	Unknown; same as HAH?	Vasogenic edema	Advanced vasogenic cerebral edema

*The self-report Lake Louise AMS score.
†See Figure 1-11.
CT, computed tomography; HAPE, high-altitude pulmonary edema; ICP, intracranial pressure; MRI, magnetic resonance imaging.

Box 1-3. Differential Diagnosis of High-Altitude Illnesses

Acute mountain sickness and high-altitude cerebral edema	Dehydration; exhaustion; viral or bacterial infection; alcohol hangover; hypothermia; carbon monoxide poisoning; migraine; hyponatremia; hypoglycemia; diabetic ketoacidosis; CNS infection; transient ischemic attack; arteriovenous malformation; stroke; seizures; brain tumors; ingestion of toxins, drugs, and alcohol; acute psychosis
High-altitude pulmonary edema	Asthma, bronchitis, pneumonia, mucus plugging (secondary to previous), hyperventilation syndrome, pulmonary embolus, heart failure, myocardial infarction

Differential Diagnosis

Given the nonspecific nature of the symptoms, AMS is commonly confused with other conditions. Box 1-3 lists conditions that the authors have seen confused with AMS and HACE. AMS is most commonly misdiagnosed as a viral flulike illness, hangover, exhaustion, dehydration, or medication or drug effect. Unlike an infectious illness, uncomplicated AMS is not associated with fever and myalgia. Hangover is excluded by the history. Exhaustion may cause lassitude, weakness, irritability, and headache and may therefore be difficult to distinguish from AMS. Dehydration, which causes weakness, decreased urine output, headache, and nausea, is commonly confused with AMS. Response to fluids helps to differentiate the two. AMS is not improved by fluid administration alone, and body hydration does not influence susceptibility to AMS (contrary to conventional wisdom).[9] Hypothermia may manifest as ataxia and mental changes. Sleeping medication can cause ataxia and mental changes, but soporifics may also precipitate high-altitude illness because of increased hypoxemia during sleep. Migraine may be very difficult to distinguish from AMS. A trial of oxygen breathing or descent can be helpful to discriminate these other conditions from AMS.

Carbon Monoxide

Carbon monoxide poisoning is a danger at high altitude, where field shelters are designed to be small and windproof. Cooking inside closed tents and snow shelters during storms is a particular hazard.[116,228,255,441,447] The effects of carbon monoxide and high-altitude hypoxia are additive. A reduction in oxyhemoglobin caused by carbon monoxide increases hypoxic stress, rendering a person at a physiologically higher altitude, which may precipitate AMS. Because of preexisting hypoxemia, smaller amounts of carboxyhemoglobin produce symptoms of carbon monoxide poisoning. These two problems may coexist. Immediate removal of the victim from the source of carbon monoxide and forced hyperventilation, preferably with supplemental oxygen, rapidly reverse carbon monoxide poisoning.

Pathophysiology

The basic cause of AMS is hypoxemia (Fig. 1-11), but the syndrome is different from acute hypoxia. Because symptoms are somewhat worse with hypobaric hypoxia than with normobaric hypoxia, hypobaria apparently also plays a minor role, most likely through its effect on fluid retention.[266,372] Because of a time lag in onset of symptoms after ascent and lack of immediate or complete reversal of all symptoms with oxygen, AMS is thought to be secondary to the body's responses to modest hypoxia. In addition, even though an altitude of 2500 to 2700 m presents only a minor decrement in arterial oxygen transport (Sa_{O_2} is still above 90%), AMS is common and some individuals may become desperately ill. An acceptable explanation of pathophysiology must therefore address lag time, individual susceptibility to even modest hypoxia, and how acclimatization prevents the illness.

Findings documented in mild to moderate AMS include relative hypoventilation,[288,313] impaired gas exchange (interstitial edema),[138,250] fluid retention and redistribution,[30,373,435] and increased sympathetic drive.[26,29] In mild to moderate AMS, limited data show that neither intracranial pressure (ICP) nor cerebrospinal fluid (CSF) pressure is elevated.[13,173,480] In contrast, increased ICP and cerebral edema are documented in moderate to severe AMS, reflecting the continuum from advanced AMS to HACE.[190,241,287,421,474]

Relative hypoventilation may be due primarily to decreased drive to breathe (low hypoxic ventilatory response) or may be secondary to ventilatory depression associated with AMS.[313,363] Persons with quite low hypoxic ventilatory response are more likely to suffer AMS than are those with a high ventilatory drive.[180,288,313] For persons with intermediate hypoxic ventilatory response values (the majority of people), ventilatory drive has no predictive value.[300,363] The protective role of a high hypoxic ventilatory response most likely results from overall increased oxygen transport, especially during sleep and exercise.

Pulmonary dysfunction in AMS includes decreased vital capacity and peak expiratory flow rate,[421] increased alveolar–arterial oxygen difference,[138,181] decreased transthoracic impedance,[213] and occasionally rales.[273] These findings are compatible with interstitial edema—that is, increased extravascular lung water, most likely related to fluid retention and an increased interstitial water compartment. That exercise can contribute to interstitial edema at altitude was recently confirmed.[7] Whether this can be considered a mild form of HAPE is unclear. The fact that nifedipine effectively prevents HAPE but does not prevent AMS or the increased alveolar–arterial oxygen gradient observed in AMS[181] speaks against the increased lung water of AMS being related to HAPE. In addition, in contrast to the results in HAPE, alveolar lavage analysis in persons with AMS was normal.

The mechanism of fluid retention may be multifactorial. Renal responses to hypoxia are variable and depend on plasma arginine vasopressin (AVP) concentration and sympathetic tone.[176,435] Persons with AMS had elevated plasma or urine AVP levels in some studies,[26,421] but cause and effect could not be established. Other studies showed no AVP elevation.[30] The usual decrease in aldosterone on ascent to altitude does not occur in persons with AMS, and this may contribute to the antidiuresis.[30] The renin-angiotensin system, although suppressed compared with its activity at sea level in both AMS and non-AMS groups, was more active in persons with AMS.[29] Atrial natriuretic peptide (ANP) is elevated in AMS. Although this is most

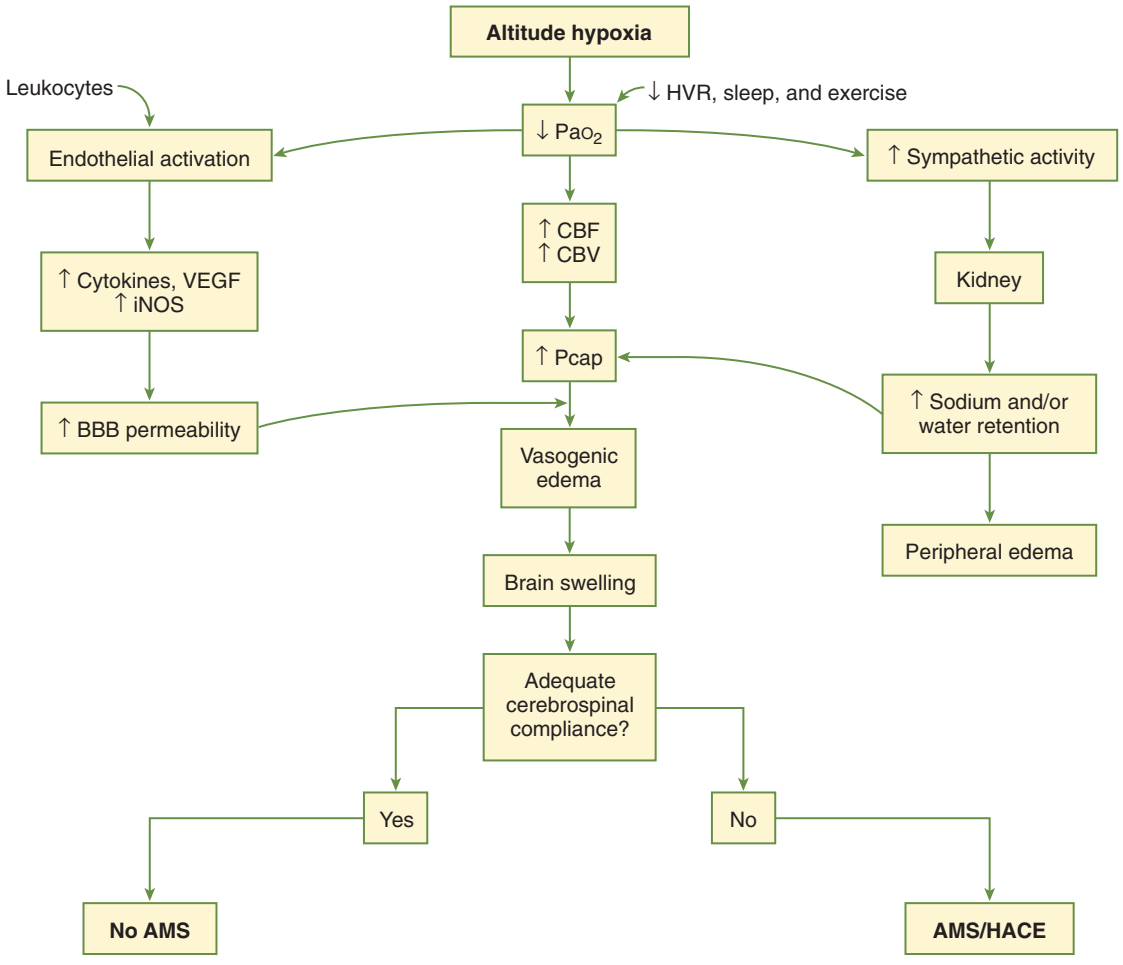

Figure 1-11. Proposed pathophysiology of acute mountain sickness (AMS). BBB, blood–brain barrier; CBF, cerebral blood flow; CBV, cerebral blood volume; HACE, high-altitude cerebral edema; HVR, hypoxic ventilatory response; iNOS, inducible nitric oxide synthase; Pcap, capillary pressure; VEGF, vascular endothelial growth factor.

likely compensatory, elevated plasma ANP levels may contribute to vasodilation and increased microvascular permeability.[29,472] One factor that can explain many of these changes is increased sympathetic activity, which reduces renal blood flow, glomerular filtration rate, and urine output, and suppresses renin.[435] Increased sympathetic nervous system activity is also consistent with the greater rise in norepinephrine noted in subjects with AMS.[26] See Krasney[237] for a discussion of the critical role of central sympathetic activation on the kidney and its role in the pathophysiology of AMS. Whatever the exact mechanism, it seems that renal water handling switches from net loss or no change to net gain of water as persons become ill with AMS. The effectiveness of diuretics in treating AMS also supports a pivotal role for fluid retention and fluid shifts in the pathology of AMS.[138,421]

Persons with moderate to severe AMS or HACE display white matter edema on brain imaging and elevated intracranial pressure, whereas those with mild AMS do not.[13,164,190,241,259,287,421] Possible mechanisms include cytotoxic edema with a shift of fluid into the cells, or vasogenic edema from increased permeability of the blood–brain barrier (BBB), or both. The classic view that hypoxia causes failure of the ATP-dependent sodium pump and subsequent intracellular edema[188] is untenable, given the newer understanding of brain energetics; ATP levels are maintained even in severe hypoxemia.[415] The evidence now favors vasogenic brain edema as the cause of advanced AMS or HACE.[164] The fact that dexamethasone is so effective for AMS also suggests vasogenic edema, as this is the only steroid-responsive brain edema. In addition, a model of AMS in conscious sheep exposed to 10% oxygen for several days supports the vasogenic brain-swelling hypothesis.[239] The pathophysiology may be similar to that of hypertensive encephalopathy, another type of hyperperfusion encephalopathy[407] in which loss of vascular autoregulation results in increased pressures transmitted to the capillaries with resultant white matter edema.[251,252] Supporting this notion, Van Osta and colleagues showed that cerebral autoregulation was impaired the most in those with more severe AMS.[450] Because prolonged cerebral vasodilation

by itself, however, is not sufficient to induce vasogenic edema, Hackett[148] and Krasney[238] have proposed the additional factor of increased BBB permeability in the pathophysiology of AMS. Possible mechanisms of altered BBB permeability in AMS/HACE include vascular endothelial growth factor (VEGF), inflammatory cytokines, products of lipid peroxidation, reactive oxygen species, endothelium-derived products such as nitric oxide, and direct neural and humoral factors known to affect the BBB. The study by Schoch and colleagues provided impressive evidence for a role for VEGF.[402] Mice on hypoxic exposure developed brain edema associated with elevated brain levels of VEGF; when given an antibody to VEGF, mice developed no cerebral edema.[402] A recent study testing the hypothesis of a leaky BBB due to reactive oxygen species found evidence of free radical generation in subjects with AMS, but no evidence of altered BBB permeability despite the presence of mild brain swelling.[13] Although the free radicals were not associated with BBB leak, their role in causing symptoms of AMS by another mechanism could not be excluded.

The question of whether mild AMS, especially headache alone, is due to vasogenic cerebral edema is not yet answered (see High-Altitude Headache). Magnetic resonance imaging (MRI) studies demonstrated brain swelling in all subjects ascending rapidly to moderate altitude, regardless of the presence of AMS.[114,210,322] The change in brain volume was greater than that expected from increased cerebral blood volume alone (due to vasodilation), but the individual components of blood and brain parenchyma could not be determined with MRI. Therefore, whether edema was present was not established. Regardless, the changes in the ill and the well groups were similar. Interestingly, Kilgore and colleagues did show a small but significant increase in T2 signal of the corpus callosum, hinting that vasogenic edema was starting, and the increase in the AMS group was twice that of the non-AMS group, though not quite statistically significant.[231] Although it is still very much an open question, the literature to date does not confirm that mild AMS or headache alone is related to brain edema.

To summarize, moderate to severe AMS and HACE represent a continuum from mild to severe vasogenic cerebral edema. Headache alone, or the earliest stages of AMS, might be related to edema or could be related to other factors, such as cerebral vasodilation or a migraine mechanism; further research is needed to clarify this issue.

Individual Susceptibility and Intracranial Dynamics. What might explain individual susceptibility to AMS? Correlation of AMS with HVR, ventilation, fluid status, lung function, and physical fitness has been weak at best. Ross hypothesized in 1985 that the apparently random nature of susceptibility might be explained by random anatomic differences.[386] Specifically, he suggested that persons with smaller intracranial and intraspinal CSF capacity would be disposed to develop AMS, because they would not tolerate brain swelling as well as those with more "room" in the craniospinal axis. It is the displacement of CSF through the foramen magnum into the spinal canal that is the first compensatory response to increased brain volume, followed by increased CSF absorption and decreased CSF formation. Studies have shown that the increase in ICP for a given increase in brain volume is directly related to the tightness of the brain in the cranium (the brain volume–to–intracranial volume ratio) and to the volume of the spinal canal.[413] Thus, the greater the initial CSF volume, the more accommodation that can take place in response to brain edema. Increases in volume are buffered by CSF dynamics. In light of our present understanding of increased brain volume on ascent to altitude, Ross's hypothesis is very attractive. Preliminary data that showed a relationship of pre-ascent ventricular size or brain volume–to–cranial vault ratios and susceptibility to AMS support this hypothesis, and the idea deserves further study.[148,490] Figure 1-11 incorporates this concept into the pathophysiology.

Natural Course of Acute Mountain Sickness

The natural history of AMS varies with initial altitude, rate of ascent, and clinical severity. Symptoms can start within as little as 2 hours after arrival, and rarely if ever start after 36 hours at a given altitude. AMS usually resolves over the next 24 to 48 hours. The more rapid the ascent, and the higher the altitude, the more likely it is that the symptoms will appear sooner and be worse. Singh and associates[421] followed the illness in soldiers airlifted to altitudes of 3300 to 5500 m. Incapacitating illness lasted 2 to 5 days, but 40% still had symptoms after 1 week and 13% after 1 month. Nine soldiers failed to acclimatize in 6 months and were considered unfit for duty at high altitude.[421] Chinese investigators report that a percentage of lowland Han Chinese stationed on the Tibet Plateau cannot tolerate the altitude because of persistent symptoms and must be relocated to the plains.[483] Persistent anorexia, nausea, and headache may afflict climbers at extreme altitude for weeks and can be considered a form of persistent AMS. The natural history of AMS in tourists who sleep at more moderate altitudes is much more benign. Duration of symptoms at 2700 m was 15 hours, with a range of 6 to 94 hours.[309] Most individuals treat or tolerate their symptoms, as the illness resolves over 1 to 3 days while acclimatization improves, but some people with AMS seek medical treatment or are forced to descend if symptoms persist. A small percentage of those with AMS (8% at 4243 m)[155] go on to develop cerebral edema, especially if ascent continues in spite of illness.

Treatment

The proper management of AMS is based on the severity of illness at presentation, and often depends on logistics, terrain, and experience of the caregiver. Early diagnosis is the key, as treatment in the early stages of illness is easier and more successful (Box 1-4). Proceeding to a higher sleeping altitude in the presence of symptoms is contraindicated. The victim must be carefully monitored for progression of illness. If symptoms worsen despite an extra 24 hours of acclimatization or treatment, descent is indicated. The two indications for immediate descent are neurologic findings (ataxia or change in consciousness) and pulmonary edema.

Mild AMS can be treated by halting the ascent and waiting for acclimatization to improve, which can take from 12 hours to 3 or 4 days. Acetazolamide (250 mg twice a day orally, or as a single dose) speeds acclimatization and thus terminates the illness if given early.[138,276] Symptomatic therapy includes analgesics such as aspirin (500 mg or 650 mg), acetaminophen (650 to 1000 mg), ibuprofen[57] or other nonsteroidal anti-inflammatory drugs, or codeine (30 mg) for headache. Promethazine (Phenergan, 25 to 50 mg by suppository or ingestion) is useful for nausea and vomiting. Persons with AMS should avoid alcohol and other respiratory depressants because of the danger of exaggerated hypoxemia during sleep.

BOX 1-4. Field Treatment of High-Altitude Illness

HIGH-ALTITUDE HEADACHE AND MILD ACUTE MOUNTAIN SICKNESS
- Stop ascent, rest, and acclimatize at same altitude
- Acetazolamide, 125 to 250 mg bid, to speed acclimatization
- Symptomatic treatment as necessary with analgesics and antiemetics
- OR descend 500 m or more

MODERATE TO SEVERE ACUTE MOUNTAIN SICKNESS
- Low-flow oxygen, if available
- Acetazolamide, 125 to 250 mg bid, with or without dexamethasone, 4 mg PO, IM, or IV q6h
- Hyperbaric therapy
- OR immediate descent

HIGH-ALTITUDE CEREBRAL EDEMA
- Immediate descent or evacuation
- Oxygen, 2 to 4 L/min
- Dexamethasone, 4 mg PO, IM, or IV q6h
- Hyperbaric therapy

HIGH-ALTITUDE PULMONARY EDEMA
- Minimize exertion and keep warm
- Oxygen, 4 to 6 L/min until improving, then 2 to 4 L/min
- If oxygen is not available:
  - Nifedipine, 10 mg PO q4h by titration to response, or 10 mg PO once, followed by 30 mg extended release q12 to 24h
  - Inhaled beta-agonist
  - Consider sildenafil 50 mg every 8 hrs
  - Hyperbaric therapy
  - OR immediate descent

PERIODIC BREATHING
- Acetazolamide, 62.5 to 125 mg at bedtime as needed

Descent to an altitude lower than where symptoms began effectively reverses AMS. Although the person should descend as far as necessary for improvement, descending 500 to 1000 m is usually sufficient. Exertion should be minimized. Oxygen, if available, is particularly effective (and supply is conserved) if given in low flow (0.5 to 1 L/min by mask or cannula) during the night. Hyperbaric chambers, which simulate descent, have been used to treat AMS and aid acclimatization. They are effective and require no supplemental oxygen. Lightweight (less than 7 kg) fabric pressure bags inflated by manual air pumps are now being used on mountaineering expeditions and in mountain clinics (Fig. 1-12). An inflation of 2 pounds per square inch (PSI) is roughly equivalent to a drop in altitude of 1600 m; the exact equivalent depends on the initial altitude.[223,361] A few hours of pressurization results in symptomatic improvement and can be an effective temporizing measure while awaiting descent or the benefit of medical therapy.[223,320,336,375,439] Long-term (12 hours or more) use of these portable devices is necessary to resolve AMS completely.

Acetazolamide is of unquestionable prophylactic value and is now commonly and successfully used to treat AMS as well,

Figure 1-12. Gamow bag used to treat a patient with high altitude pulmonary edema at Everest Base Camp Medical Clinic (5350 m). (Photo courtesy Luanne Freer, MD.)

although data supporting this are minimal.[138,276] Singh and colleagues successfully used furosemide (80 mg twice a day for 2 days) to treat 446 soldiers with all degrees of AMS; it has not since been studied for treatment.[421] Furosemide induced brisk diuresis, relieved pulmonary congestion, and improved headache and other neurologic symptoms. Spironolactone, hydrochlorothiazide, and other diuretics have not yet been evaluated for treatment.

The steroid betamethasone was initially reported by Singh and colleagues[421] to improve symptoms of soldiers with severe AMS. Subsequently, studies have found dexamethasone to be effective for treatment of all degrees of AMS.[113,163,227] Hackett and colleagues[163] used 4 mg orally or intramuscularly every 6 hours, and Ferrazinni and associates[113] gave 8 mg initially, followed by 4 mg every 6 hours. Both studies reported marked improvement within 12 hours, with no significant side effects. Symptoms increased when dexamethasone was discontinued after 24 hours.[163] Clinicians debate whether the use of dexamethasone should also require descent. Is it safe to continue on after treatment with dexamethasone, or while taking the medication? In reality, people do, and problems seem to be few. In the authors' opinion, dexamethasone use should be limited to less than 48 hours, to minimize side effects. This is generally sufficient time to descend, or to better acclimatize, with or without acetazolamide. The mechanism of action of dexamethasone is unknown; it does not affect oxygen saturation, fluid balance, or periodic breathing.[259] The drug blocks the action of VEGF,[402] diminishes the interaction of endothelium and leukocytes (thus reducing inflammation),[134] and may also reduce cerebral blood flow.[220] Dexamethasone seems to not improve acclimatization, as symptoms recur when the drug is withdrawn. Therefore, an argument could be made for using dexamethasone to relieve symptoms and acetazolamide to speed acclimatization.[48]

Prevention

Graded ascent is the surest and safest method of prevention, although particularly susceptible individuals may still become ill. Current recommendations for people without altitude

experience are to avoid abrupt ascent to sleeping altitudes greater than 3000 m and to spend 2 to 3 nights at 2500 to 3000 m before going higher, with an extra night for acclimatization every 600 to 900 m if continuing ascent. Abrupt increases of more than 600 m in sleeping altitude should be avoided when over 2500 m. However, a recent study on Aconcagua reinforced the notion that individual susceptibility is a key factor; those who are resistant to AMS can safely proceed much more quickly on the mountain.[341] Day trips to higher altitude, with a return to lower altitude for sleep, aid acclimatization. Alcohol and sedative-hypnotics are best avoided on the first 2 nights at high altitude. Whether a diet high in carbohydrates reduces AMS symptoms is controversial.[80,171,437] Exertion early in altitude exposure contributes to altitude illness,[370] whereas limited exercise seems to aid acclimatization. Because altitude exposure in the previous weeks is protective, a faster rate of ascent may then be possible.

Acetazolamide Prophylaxis. Acetazolamide is the drug of choice for prophylaxis of AMS. A carbonic anhydrase (CA) inhibitor, acetazolamide slows the hydration of carbon dioxide:

$$CO_2 + H_2O \underset{CA}{\longleftrightarrow} H_2CO_3 \underset{CA}{\longleftrightarrow} H^+ + HCO_3^-$$

The effects are protean, involving particularly the red blood cells, brain, lungs, and kidneys. By inhibiting renal CA, acetazolamide reduces reabsorption of bicarbonate and sodium and thus causes bicarbonate diuresis and metabolic acidosis starting within 1 hour after ingestion. This rapidly enhances ventilatory acclimatization, increasing oxygenation. Importantly, the drug maintains oxygenation during sleep and prevents periods of extreme hypoxemia (see Figure 1-5).[159,433,436] Because of acetazolamide's diuretic action, it counteracts the fluid retention of AMS. It also diminishes nocturnal antidiuretic hormone (ADH) secretion[72] and decreases CSF production and volume and possibly CSF pressure.[410] Which of these effects is most important in preventing AMS is unclear. Numerous studies taken together indicate that acetazolamide is approximately 75% effective in preventing AMS in persons rapidly transported to altitudes of 3000 to 4500 m.[110]

Indications for acetazolamide prophylaxis include rapid ascent (in 1 day or less) to altitudes over 3000 m; a rapid gain in sleeping altitude, for example, moving camp from 4000 to 5000 m in a day; and a past history of AMS or HAPE. The ideal dosage of acetazolamide for prevention is debated. Many studies have shown that 250 mg twice a day or three times a day was effective, as well as 500 mg in sustained action capsule taken every 24 hours.[70,126,137,152,250,353,476] To reduce the side effects, especially paresthesias, clinicians have more recently been using smaller dosages (125 mg twice a day)[298]; two studies so far support this,[32,254] whereas one does not.[66] Renal carbonic anhydrase is blocked at a dosage of 5 mg/kg/day. As this seems to be the important effect for preventing AMS, this is probably the ideal dosage, both in children and adults. Thus, adjusting the dosage for body weight might provide the best effect with the fewest side effects. Duration of medication use varies; the standard advice is to start 24 hours before ascent. Most authors recommend continuing for at least the first 2 days at high altitude, and longer if there is a continuing gain in altitude. Acetazolamide can also be taken episodically, to speed acclimatization at any point while gaining altitude, or to treat mild AMS. There is no rebound when discontinued. Although the danger of altitude illness passes after a few days of acclimatization, acetazolamide may still be useful for sleep.

Acetazolamide has side effects, most notably peripheral paresthesias and polyuria, and less commonly nausea, drowsiness, impotence, and myopia. Because it inhibits the instant hydration of carbon dioxide on the tongue, acetazolamide allows carbon dioxide to be tasted and can ruin the flavor of carbonated beverages, including beer. As a nonantibiotic sulfonamide drug, acetazolamide is usually tolerated well by people with a history of sulfa antibiotic allergy; approximately 10% may have an allergic reaction.[430] In those without a history of allergy to sulfa antibiotic, the incidence of hypersensitivity reaction to a sulfonamide nonantibiotic was 1.6%.[430] The same analysis concluded that people who are penicillin allergic are in fact more likely to react to drugs such as acetazolamide than are people who are sulfa allergic. Nonetheless, it is wise to be cautious for those with a history of allergy, especially anaphylaxis to either sulfa or penicillin. Many experts recommend a trial dose of the medication well before the altitude sojourn, to determine if the drug is tolerated well. Although the usual allergic reaction is a rash starting a few days after ingestion, anaphylaxis to acetazolamide does rarely happen.

Dexamethasone Prophylaxis. Dexamethasone effectively prevents AMS. A dosage of 2 mg every 6 hours or 4 mg every 12 hours was sufficient for sedentary subjects,[220] but for exercising subjects at or above 4000 m, this was insufficient,[47,163,377] and 4 mg every 6 hours was necessary to prevent AMS.[378] The initial chamber study in 1984 was with sedentary subjects.[220] The drug reduced the incidence of AMS from 78% to 20%, comparable to previous studies with acetazolamide. Dexamethasone was not as effective in exercising subjects on Pike's Peak,[377] but subsequent work has shown effectiveness comparable to that of acetazolamide.[37,110,259,308,491] The combination of acetazolamide and dexamethasone proved superior to dexamethasone alone.[491] Because of potential serious side effects and the rebound phenomenon, dexamethasone is best reserved for treatment rather than for prevention of AMS, or used for prophylaxis when necessary in persons intolerant of or allergic to acetazolamide.

Other Agents for AMS Prevention. Studies with *Ginkgo biloba* have had inconsistent results. Four studies had positive results, ranging from 100% to 50% reduction in AMS when given either 5 days or 1 day before ascent.[127,269,315,381] Three studies were negative, two of which were follow-up studies by investigators with positive results previously.[70,126,254] Conflicting results can possibly be explained by differences in dosing, duration of pretreatment, and varying rates of ascent, but most likely they result from differences in preparations of ginkgo. *Ginkgo biloba* is a complicated plant extract whose active ingredient in terms of preventing AMS is unknown. Even in the "standardized" preparations (24% flavonoids and 6% terpene ginkolides), the amounts of specific chemicals can vary considerably. Until the active ingredient is discovered and standardized, results with ginkgo will continue to be mixed. Meanwhile, as the product is very safe and inexpensive, does not require a prescription, and can be helpful, it is reasonable to

consider its use. Acetazolamide, however, is probably the superior agent.

Spironolactone,[215,249] naproxen, calcium channel blockers, antacids, and medroxyprogesterone acetate have shown ineffectiveness for AMS prevention. Bailey and Davies tested an antioxidant cocktail for prevention of AMS.[12] They reasoned that free-radical–mediated damage to the BBB might play a role in the pathophysiology. They preloaded nine subjects with daily L-ascorbic acid, DL-α-tocopherol acetate, and α-lipoic acid, and nine subjects with placebo for 3 weeks, and then maintained dosing during a 10-day trek to Everest base camp. Those on antioxidants had a slightly lower AMS score, higher arterial oxygen saturation, and better appetite.[12] Although these results were of marginal clinical value, the use of antioxidants certainly deserves further study. See Bärtsch and colleagues for a detailed discussion of the antioxidant hypothesis.[20]

High-Altitude Cerebral Edema

High-altitude cerebral edema is an uncommon but deadly condition, usually occurring in those with AMS or HAPE. Although HACE is most common over 3000 m, it has been reported as low as 2100 m.[96] Reliable estimates of incidence range from 0.5% to 1% in unselected high-altitude visitors,[28,152] but it was 3.4% in those who had developed AMS.[152] HACE occurs in 13% to 20% of persons with HAPE,[123,179,203] and in up to 50% of those who die from HAPE. In addition, HAPE is very common in those diagnosed with HACE; one series from Colorado reported that 11 of 13 with a primary diagnosis of HACE also had HAPE. Pure cerebral edema, without pulmonary edema, appears to be uncommon. The mean altitude of onset was 4730 m in one survey but was lower (3920 m) when there was an association with HAPE.[204] Data are insufficient to draw any conclusions regarding effects of sex, age, preexisting illness, or genetics on susceptibility to HACE. Clinically and pathophysiologically, advanced AMS and HACE are similar, so a distinction between them is inherently blurred. See a recent review for a more complete discussion.[156]

HACE is an encephalopathy, whereas AMS is not. The hallmarks of HACE are ataxic gait, severe lassitude, and altered consciousness, including confusion, impaired mentation, drowsiness, stupor, and coma. Headache, nausea, and vomiting are frequent but not always present. Hallucinations, cranial nerve palsy, hemiparesis, hemiplegia, seizures, and focal neurologic signs have also been reported.[99,167,190,421] Seizures are distinctly uncommon. Retinal hemorrhages are common but not diagnostic. The progression from mild AMS to unconsciousness may be as fast as 12 hours but usually requires 1 to 3 days; HACE can develop more quickly in those with HAPE. An arterial blood gas study or pulse oximetry often reveals exaggerated hypoxemia. Clinical examination and chest radiography may reveal pulmonary edema. Laboratory studies and lumbar puncture are useful only to rule out other conditions. Computerized tomography (CT) may show compression of sulci and flattening of gyri, and attenuation of signal more in the white matter than gray matter. MRI is more revealing, with a characteristic high T2 signal in the white matter, especially the splenium of the corpus callosum, and most evident on diffusion-weighted images.[164,479]

The following case report from Mt. McKinley illustrates a typical clinical course of HACE in conjunction with HAPE.

Clinical Presentation

H.E. was a 26-year-old German lumberjack with extensive mountaineering experience. He ascended to 5200 m from 2000 m in 4 days and attempted the summit (6194 m) on the fifth day. At 5800 m he turned back because of severe fatigue, headache, and malaise. He returned alone to 5200 m, stumbling on the way because of loss of coordination. He had no appetite and crawled into his sleeping bag too weak, tired, and disoriented to undress. He recalled no pulmonary symptoms. In the morning H.E. was unarousable, slightly cyanotic, and noted to have Cheyne-Stokes respirations. After 10 minutes on high-flow oxygen, H.E. began to regain consciousness, although he was completely disoriented and unable to move. A rescue team lowered him down a steep slope, and on arrival at 4400 m 4 hours later he was conscious but still disoriented, able to move extremities but unable to stand. Respiratory rate was 60 breaths/min and heart rate was 112 beats/min. Papilledema and a few rales were present. Sa_{O_2}% was 54% on room air (normal is 85% to 90%). On a nonrebreathing oxygen mask with 14 L/min oxygen, the Sa_{O_2}% increased to 88% and the respiratory rate decreased to 40 breaths/min. Eight milligrams of dexamethasone were administered intramuscularly at 4:20 PM and continued orally, 4 mg every 6 hours. At 5:20 PM, H.E. began to respond to commands. The next morning he was still ataxic but was able to stand, take fluids, and eat heartily. He was evacuated by air to Anchorage (sea level) at 12:00 PM. On admission to the hospital at 3:30 PM, roughly 36 hours after regaining consciousness, H.E. was somewhat confused and mildly ataxic. Arterial blood gas studies on room air showed a P_{O_2} of 58 mm Hg, pH of 7.5, and P_{CO_2} of 27 mm Hg. Bilateral pulmonary infiltrates were present on the chest radiograph. Magnetic resonance imaging of the brain revealed white matter edema, primarily of the corpus callosum (Fig. 1-13). On discharge the next morning H.E. was oriented, bright, and cheerful and had very minor ataxia and clear lung fields.

Pathophysiology

The pathophysiology of HACE is a progression of the same mechanism seen in advanced AMS (see Acute Mountain Sickness, Pathophysiology; also see Figure 1-11), and appears to be vasogenic edema. In cases similar to this, lumbar punctures have revealed elevated CSF pressures, often more than 300 mm H_2O,[190,474] evidence of cerebral edema on CT scans and MR images,[164,236] and gross cerebral edema on necropsy.[98,99] Small petechial hemorrhages were also consistently found on autopsy, and venous sinus thromboses were occasionally seen.[98,99] Well-documented cases have often included pulmonary edema that was not clinically apparent.

Whereas the brain edema of reversible HACE is most likely vasogenic, as the spectrum shifts to severe, end-stage HACE, then gray matter (presumably cytotoxic) edema develops as well, culminating in brain herniation and death. As Klatzo[235] has pointed out, as vasogenic edema progresses, the distance between brain cells and their capillaries increases, so that nutrients and oxygen eventually fail to diffuse and the cells are rendered ischemic, leading to intracellular (cytotoxic) edema. Raised intracranial pressure produces many of its effects by decreasing cerebral blood flow, and brain tissue becomes

Figure 1-13. Magnetic resonance image of a patient with high-altitude cerebral edema. Increased T2 signal in splenium of corpus callosum *(arrow)* indicates edema.

ischemic on this basis also.[294] Focal neurologic signs caused by brainstem distortion and by extra-axial compression, as in third and sixth cranial nerve palsies, may develop,[382] making cerebral edema difficult to differentiate from primary cerebrovascular events. The most common clinical presentation, however, is change in consciousness associated with ataxia, without focal signs.[487]

Treatment

Given the sporadic nature and generally remote location of this disorder, it is not surprising there are no controlled trials regarding treatment of HACE. All experts agree that successful treatment requires early recognition. At the first sign of ataxia or change in consciousness, descent should be started, dexamethasone (4 to 8 mg intravenously, intramuscularly, or orally initially, followed by 4 mg every 6 hours) administered, and oxygen (2 to 4 L/min by vented mask or nasal cannula) applied if available (see Box 1-4). Oxygen can be titrated to maintain SaO_2 at greater than 90% if oximetry is available. Comatose patients require additional airway management and bladder drainage. Attempting to decrease intracranial pressure by intubation and hyperventilation is a reasonable approach, although these patients are already alkalotic, and over-hyperventilation could result in disastrous cerebral ischemia. Loop diuretics such as furosemide (40 to 80 mg) or bumetanide (1 to 2 mg) may reduce brain hydration, and have been used successfully,[97,421] but an adequate intravascular volume to maintain perfusion pressure is critical. Hypertonic solutions of saline, mannitol, or oral glycerol have been suggested but are rarely used in the field. Controlled studies are lacking, but empirically the response to steroids and oxygen seems excellent if they are given early in the course of the illness and disappointing if they are not started until the victim is unconscious. Coma may persist for days, even after evacuation to low altitude, but other causes of coma must be considered and ruled out by appropriate evaluation.[190] The average duration of hospital stay in one series of patients with severe HACE was 5.6 days, and average time to full recovery was 2.4 weeks, with a range of 1 day to 6 weeks.[164] Two patients of the 44 in the series by Dickinson remained in coma for 3 weeks.[99] Sequelae lasting weeks are common[164,190]; longer-term follow-up has been limited, but presumed permanent impairment has been reported.[156,190] Prevention of HACE is the same as for AMS and HAPE.

Focal Neurologic Conditions without AMS or Cerebral Edema

Various localizing neurologic signs, transient in nature and not necessarily occurring in the setting of AMS, suggest migraine, cerebrovascular spasm, transient ischemic attack, local hypoxia without loss of perfusion (watershed effect), or focal edema. Cortical blindness is one such condition. Hackett and colleagues[161] reported six cases of transient blindness in climbers or trekkers with intact pupillary reflexes, which indicated that the condition was due to a cortical process. Treatment with breathing of either carbon dioxide (a potent cerebral vasodilator) or oxygen resulted in prompt relief, suggesting that the blindness was due to inadequate regional circulation or oxygenation. Descent effected relief more slowly. Other conditions that could be attributed to spasm or transient ischemic attack (TIA) have included transient hemiplegia or hemiparesis, transient global amnesia, unilateral paresthesias, aphasia, and scotoma.[31,36,52,67,262,477,493] The true mechanism of these focal findings is unknown, and it may be multifactorial. Young, healthy altitude sojourners are unlikely to have TIA syndrome from cerebrovascular disease.

The occurrence of stroke in a young, fit person at high altitude is uncommon but tragic. A number of case reports have described climbers with resultant permanent dysfunction.[71,189,423] Indian soldiers at extreme altitude have a high incidence of stroke.[219] Cerebral venous thrombosis presents more insidiously, and diagnosis is often delayed.[143,222,389,423,443,493] Factors contributing to stroke may include polycythemia, dehydration, patent foramen ovale, and increased intracranial pressure if AMS is present, increased cerebrovenous pressure, cerebrovascular spasm, and, in certain episodes, coagulation abnormalities.[33] Stroke may be confused with HACE. Neurologic symptoms, especially focal abnormalities without AMS or HAPE that persist despite treatment with oxygen, steroids, and descent, suggest a cerebrovascular event and mandate careful evaluation with a complete neurologic workup.

Clinical Presentation

E.H., a 42-year-old male climber on a Mt. Everest expedition, awoke at 8000 m with dense paralysis of the right arm and weakness of the right leg. On descent the paresis cleared, but at base camp (5000 m) severe vertigo developed, along with extreme ataxia and weakness. Neurologic consultation on return to the United States resulted in a diagnosis of multiple small cerebral infarcts, but none was visible on CT scan of the brain. The hematocrit value 3

weeks after descent from the mountain was 70%. Over the next 4 years, signs gradually improved, but mild ataxia, nystagmus, and dyslexia persist. The focal and persistent nature of the cerebral symptoms and signs, although multiple, indicates an etiology related to cerebrovascular effects rather than intracranial pressure. The hematocrit value on the mountain was greater than 70%, high enough for increased viscosity and microcirculatory sludging to contribute to ischemia and infarction. No familial thrombophilia was detected.

Treatment of stroke is supportive. Oxygen and steroids may be worthwhile to treat any AMS or HACE component. Immediate evacuation to a hospital is indicated. Persons with transient ischemic attacks at high altitude should probably be started on aspirin therapy and proceed to a lower altitude. Oxygen may quickly abort cerebrovascular spasms and will improve watershed hypoxic events. When oxygen is not available, rebreathing to raise alveolar P_{CO_2} may be helpful through increasing cerebral blood flow.

Cognitive Changes at High Altitude

If cerebral oxygen consumption is constant, what causes the well-documented, albeit mild, cognitive changes at high altitude? The cognitive changes may be related to specific neurotransmitters that are affected by mild hypoxia. For example, tryptophan hydroxylase in the serotonin synthesis pathway has a high requirement for oxygen (K_m = 37 mm Hg).[79,129] Tyrosine hydroxylase, in the dopamine pathway, is also oxygen sensitive. Gibson and Blass suggested that a decrease in acetylcholine activity during hypoxia might explain the lassitude.[129] In a fascinating study, Banderet and Lieberman showed that increased dietary tyrosine reduced mood changes and symptoms of environmental stress in subjects at simulated altitude.[16] Further work with neurotransmitter agonists and antagonists will help shed light on their role in cognitive dysfunction at altitude and could lead to new pharmacologic approaches to improve neurologic function.

High-Altitude Pulmonary Edema

The most common cause of death related to high altitude, HAPE is completely and easily reversed if recognized early and treated properly. Undoubtedly, HAPE was misdiagnosed for centuries, as evidenced by frequent reports of young, vigorous men suddenly dying of "pneumonia" within days of arriving at high altitude. The death of Dr. Jacottet, "a robust, broad-shouldered young man," on Mt. Blanc in 1891 (he refused descent so that he could "observe the acclimatization process" in himself) may have provided the first autopsy of HAPE. Angelo Mosso wrote,

> *From Dr. Wizard's post-mortem examination . . . the more immediate cause of death was therefore probably a suffocative catarrh accompanied by acute edema of the lungs. . . . I have gone into the particulars of this sorrowful incident because a case of inflammation of the lungs also occurred during our expedition, on the summit of Monte Rosa, from which, however, the sufferer fortunately recovered.*[319]

On an expedition to K2 (Karakoram Range, Pakistan) in 1902, Alistair Crowley[85] described a climber "suffering from edema of both lungs and his mind was gone." In the Andes, physicians were familiar with pulmonary edema peculiar to high altitude,[209,264] but it was not until Houston[187] and Hultgren and Spickard[197] that the English-speaking world became aware of high altitude pulmonary edema (see Rennie[354] for a review). Hultgren and colleagues[208] then published hemodynamic measurements in persons with HAPE, demonstrating that it was a noncardiogenic edema. Since that time, many studies and reviews have been published, and HAPE is still the subject of intense investigation. The reader is referred to recent reviews.[19,27,145,403,468]

Individual susceptibility, rate of ascent, altitude reached, degree of cold,[351] physical exertion and certain underlying medical conditions are all factors determining the prevalence of HAPE. The incidence varies from less than 1 in 10,000 skiers at moderate altitude in Colorado to 1 in 50 climbers on Mt. McKinley (6194 m) and up to 6% of mountaineers in the Alps ascending rapidly to 4559 m.[24] Hultgren and colleagues reported 150 cases of HAPE over 39 months at a Colorado ski resort at 2928 m.[206] Some regiments in the Indian Army had a much higher incidence of HAPE (15%) because of very rapid deployment to the extreme altitude of 5500 m[422] (see Table 1-1). Persons who had a previous episode of HAPE had a 60% attack rate when they went to 4559 m in 36 hours; however, with a slower ascent, some of the same individuals were able to climb over 7000 m without developing HAPE.[19,25] Although HAPE occurs in both sexes, it is perhaps less common in women.[83,203,424] Whether all persons are capable of developing HAPE (with a very rapid ascent to a sufficiently high altitude and with heavy exercise) is arguable.[27] With a sudden push to a higher altitude, even well-acclimatized individuals can succumb to HAPE, and some studies suggest that HAPE is a spectrum, with many persons contracting subclinical extravascular lung water.[84] Nonetheless, a population of HAPE-susceptible persons with unique physiologic characteristics has been described (see HAPE Susceptibility). These individuals represent the small percentage of people who develop HAPE when others in the same circumstances do not.

Clinical Presentation

D.L., a 34-year-old man, was in excellent physical condition and had been on numerous high-altitude backpacking trips, occasionally suffering mild symptoms of AMS. He drove from sea level to the trailhead and hiked to a 3050 m sleeping altitude the first night of his trip in the Sierra Nevada. He proceeded to 3700 m the next day, noticing more dyspnea on exertion when walking uphill, a longer time than usual to recover when he rested, and a dry cough. He complained of headache, shivering, dyspnea, and insomnia the second night. The third day the group descended to 3500 m and rested, primarily for D.L.'s benefit. That night D.L. was unable to eat, noted severe dyspnea, and suffered coughing spasms and headache. On the fourth morning, D.L. was too exhausted and weak to get out of his sleeping bag. His companions noted that he was breathless, cyanotic, and ataxic but had clear mental status. A few hours later he was transported by helicopter to a hospital at 1200 m. On admission he was cyanotic, oral temperature was 37.8°C (100°F), blood pressure 130/76 mm Hg, heart rate 96 beats/min, and respiratory frequency 20 breaths/min. Bilateral basilar rales were noted up to the scapulae. Findings of the cardiac examination

TABLE 1-5. Severity Classification of High-Altitude Pulmonary Edema

GRADE	SYMPTOMS	SIGNS	CHEST FILM
1 Mild	Dyspnea on exertion, dry cough, fatigue while moving uphill (if any)	HR (rest) < 90–100; RR (rest) < 20; dusky nail beds; localized rales	Minor exudate involving less than 25% of one lung field
2 Moderate	Dyspnea, weakness, fatigue on level walking; raspy cough; headache; anorexia	HR 90–100; RR 16–30; cyanotic nail beds; rales present; ataxia may be present	Some infiltrates involving 50% of one lung or smaller area of both lungs
3 Severe	Dyspnea at rest, productive cough, orthopnea, extreme weakness	Bilateral rales; HR > 110; RR > 30; facial and nail-bed cyanosis; ataxia; stupor; coma; blood-tinged sputum	Bilateral infiltrates > 50% of each lung

HR, heart rate; RR, respiratory rate.
Modified from Hultgren HN: In Staub NC (ed): New York, Dekker, 1978, pp 437–469.

were reported as normal. Romberg and finger-to-nose tests revealed 1+ ataxia. Arterial blood gas studies on room air revealed P_{O_2} 24 mm Hg, P_{CO_2} 28 mm Hg, and pH 7.45. The chest radiograph showed extensive bilateral patchy infiltrates (Fig. 1-14C). D.L. was treated with bed rest and supplemental oxygen. On discharge to his sea level home 3 days later, his pulmonary infiltrates and rales had cleared, although his blood gas values were still abnormal: P_{O_2} 76 mm Hg, P_{CO_2} 30 mm Hg, and pH 7.45. He had an uneventful, complete recovery at home. D.L. was advised to ascend more slowly in the future, staging his ascent with nights spent at 1500 m and at 2500 m. He was taught the early signs and symptoms of HAPE and was advised about pharmacologic prophylaxis.

This case illustrates a number of typical aspects of HAPE. Victims are frequently young, fit men who ascend rapidly from sea level and may not have previously suffered HAPE even with repeated altitude exposures; this ascent may have been faster than previous ascents. HAPE usually occurs within the first 2 to 4 days of ascent to higher altitudes (above 2500 m), most commonly on the second night.[146] Decreased exercise performance and increased recovery time from exercise are the earliest indications of HAPE. The victim usually notices fatigue, weakness, and dyspnea on exertion, especially when trying to walk uphill; he or she often ascribes these nonspecific symptoms to various other causes. Signs of AMS, such as headache, anorexia, and lassitude, are present about 50% of the time.[203] A persistent dry cough develops. Nail beds and lips become cyanotic. The condition typically worsens at night, and tachycardia and tachypnea develop at rest. Dyspnea at rest and audible congestion in the chest herald the development of a serious condition.

In contrast to the usual 1- to 3-day gradual onset, HAPE may strike abruptly, especially in a sedentary person who may not notice the early stages.[455] Orthopnea is uncommon (7%). Pink or blood-tinged, frothy sputum is a very late finding. Hemoptysis was present in 6% in one series.[206] Severe hypoxemia may produce cerebral edema with mental changes, ataxia, decreased level of consciousness, and coma. Hultgren and colleagues reported an incidence of HACE of 14% in those with HAPE at ski resorts.[206]

On admission to the hospital, the victim does not generally appear as ill as would be expected on the basis of arterial blood gas and radiographic findings. Elevated temperature of up to 38.5°C (101.3°F) is common. Tachycardia correlates with respiratory rate and severity of illness (Table 1-5).[208] Rales may be unilateral or bilateral and usually originate from the right middle lobe. Concomitant respiratory infection is sometimes present.

Patients with pulmonary edema sometimes present with predominantly neurologic manifestations and minimal pulmonary symptoms and findings. Cerebral edema, especially with coma, may obscure the diagnosis of HAPE.[157] Pulse oximetry or chest radiography confirms the diagnosis. The differential diagnosis includes pneumonia, bronchitis, mucus plugging, pulmonary embolism or infarct, heart failure, acute myocardial infarction, and sometimes asthma (see Box 1-3). Complications include infection, cerebral edema, pulmonary embolism or thrombosis, and such injuries as frostbite or trauma secondary to incapacitation.[19,157,204]

Hemodynamics

Hemodynamic measurements show elevated pulmonary artery pressure and pulmonary vascular resistance, low to normal pulmonary wedge pressure, and low to normal cardiac output and systemic arterial blood pressure (Table 1-6).[205,339] Echocardiography demonstrates high pulmonary artery pressure, tricuspid regurgitation, normal left ventricular systolic function, somewhat abnormal diastolic function,[5] and variable right-sided heart findings of increased atrial and ventricular size.[160,333]

The electrocardiogram usually reveals sinus tachycardia. Changes consistent with acute pulmonary hypertension have been described, such as right axis deviation, right bundle branch block, voltage for right ventricular hypertrophy, and P wave abnormalities.[19,203] Atrial flutter has been reported, but ventricular arrhythmias have not.

Laboratory Studies

Kobayashi and colleagues[236] reported clinical laboratory values in 27 patients with HAPE that showed mild elevations of hematocrit and hemoglobin, probably secondary to intravascular volume depletion and perhaps plasma leakage into the lung. Elevation of the peripheral white blood cell count is common, but rarely is it greater than 14,000 cells/mL^3. The serum concentration of creatine phosphokinase (CPK) is increased. Most of the rise in CPK has been attributed to skeletal muscle damage, although in two patients, CPK isoenzymes showed brain fraction levels of 1% of the total, which may have indicated brain

TABLE 1-6. Hemodynamic Measurements during High-Altitude Pulmonary Edema (HAPE) and after Recovery in Two Subjects and in a Group of 31 Control Subjects

MEASUREMENT	HAPE*	RECOVERY*	CONTROLS†
Sa_{O_2}%	58.0	84.0	89.0
Mean pulmonary artery pressure (mm Hg)	63.0	18.0	21.3
Wedge pressure (mm Hg)	1.5	3.5	7.1
Cardiac index (L/m^2)	2.5	4.4	4.1
Pulmonary vascular resistance ($dyne/cm^{-5}$)	1210.0	169.0	169.0
Mean arterial blood pressure (mm Hg)	82.0	—	96.0

*HAPE and recovery values from Penaloza D, Sime F: Am J Cardiol 23:369–378, 1969.
†Mean values from 31 normal subjects studied at 3700 m; from Hultgren HN, Grover RF: Ann Rev Med 19:119–152, 1968.

damage.[236] BNP values in patients with HAPE may be normal or elevated.

Arterial blood gas studies consistently reveal respiratory alkalosis and marked hypoxemia more severe than expected for the patient's clinical condition. Because respiratory or metabolic acidosis has not been reported, arterial blood gas studies are unnecessary if noninvasive pulse oximetry is available to measure arterial oxygenation. At 4200 m on Mt. McKinley, the mean value of arterial P_{O_2} in HAPE was 28 ± 4 mm Hg. Values as low as 24 mm Hg in HAPE are not unusual. Arterial oxygen saturation values in HAPE subjects at 4300 m ranged from 40% to 70%, with a mean of 56% ± 8%,[406] and at 2928 m, mean Sa_{O_2} was 74%.[206] Arterial acid–base values may be misleading in patients taking acetazolamide, because this drug produces significant metabolic acidosis.

Radiographic Findings

The radiographic findings in HAPE have been described in original reports.[208,281,457,458] Findings are consistent with noncardiogenic pulmonary edema, with generally normal heart size and left atrial size and no evidence of pulmonary venous prominence, such as Kerley lines. The pulmonary arteries increase in diameter.[458] Infiltrates are commonly described as fluffy and patchy with areas of aeration between them and in a peripheral location rather than central. Infiltrates may be unilateral or bilateral, with a predilection for the right middle lung field, which corresponds to the usual area of rales. Pleural effusion is rare. The radiographic findings generally correlate with the severity of the illness and degree of hypoxemia. A small right hemithorax, absence of pulmonary vascular markings on the right, and edema confined to the left lung are criteria for diagnosis of unilateral absent pulmonary artery.[151] Radiographic findings of HAPE are presented in Figure 1-14.

Clearing of infiltrates is generally rapid once treatment is initiated. Depending on severity, complete clearing may take from 1 day to several days. Infiltrates are likely to persist longer if the patient remains at high altitude, even if confined to bed and receiving oxygen therapy. Radiographs taken within 24 to 48 hours of return to low altitude may still be able to confirm the diagnosis of HAPE.

Pathologic Findings

More than 20 autopsy reports of persons who died of HAPE have been published.[10,98,324,421,422,474] Of those whose cranium was opened, more than half had cerebral edema. All lungs showed extensive and severe edema, with bloody, foamy fluid in the airways. Lung weights were 2 to 4 times normal. The left side of the heart was normal. The right atrium and main pulmonary artery were often distended. Proteinaceous exudate with hyaline membranes was characteristic. All lungs had areas of inflammation with neutrophil accumulation. The diagnosis of bronchopneumonia was common, although bacteria were not noted. Pulmonary veins, the left ventricle, and the left atrium were generally not dilated, in contrast to the right ventricle and atrium. Most reports mention capillary and arteriolar thrombi and alveolar fibrin deposits, as well as microvascular and gross pulmonary hemorrhage and infarcts. The autopsy findings thus suggest a protein-rich, permeability-type edema, with thrombi or emboli. Confirmation of HAPE as a permeability edema was obtained by analysis of alveolar lavage fluid by Schoene and associates.[404,406] These authors found a 100-fold increase in lavage fluid protein levels in patients with HAPE compared with well control subjects and patients with AMS.[406] The lavage fluid also had a low percentage of neutrophils, in contrast to findings in adult respiratory distress syndrome. Further evidence for a permeability edema was a 1:1 ratio of aspirated edema fluid protein to plasma protein level found by Hackett and colleagues.[150] In addition, the lavage fluid contained vasoactive eicosanoids and complement proteins, indicative of endothelium–leukocyte interactions. More recent research using repeated bronchoalveolar lavage as HAPE was developing found that inflammation was absent early on, suggesting that inflammation is a response to the alveolar damage rather than an initiating event.[438]

Mechanisms of High-Altitude Pulmonary Edema

An acceptable explanation for HAPE must take into account three well-established facts: excessive pulmonary hypertension, high-protein permeability leak, and normal function of the left side of the heart. A mechanism consistent with these facts is failure of capillaries secondary to overperfusion and capillary hypertension (Fig. 1-15).

Role of Pulmonary Hypertension. Excessive pulmonary artery pressure (PAP) is the sine qua non of HAPE; no cases of HAPE have been reported without pulmonary hypertension. All persons ascending to high altitudes or otherwise enduring hypoxia, however, have some elevation of PAP. The hypoxic pulmonary vasoconstrictor response (HPVR) is thought to be useful in humans at sea level because it helps match perfusion with ventilation. When local areas of the lung are poorly ventilated because of infection or atelectasis, for example, the HPVR directs blood away from those areas to well-ventilated regions. In the setting of global hypoxia, as occurs with ascent to high altitude, HPVR is presumably diffuse and all areas of the lung constrict, causing a restricted vascular bed and an increase in PAP, which is of little if any value for ventilation–

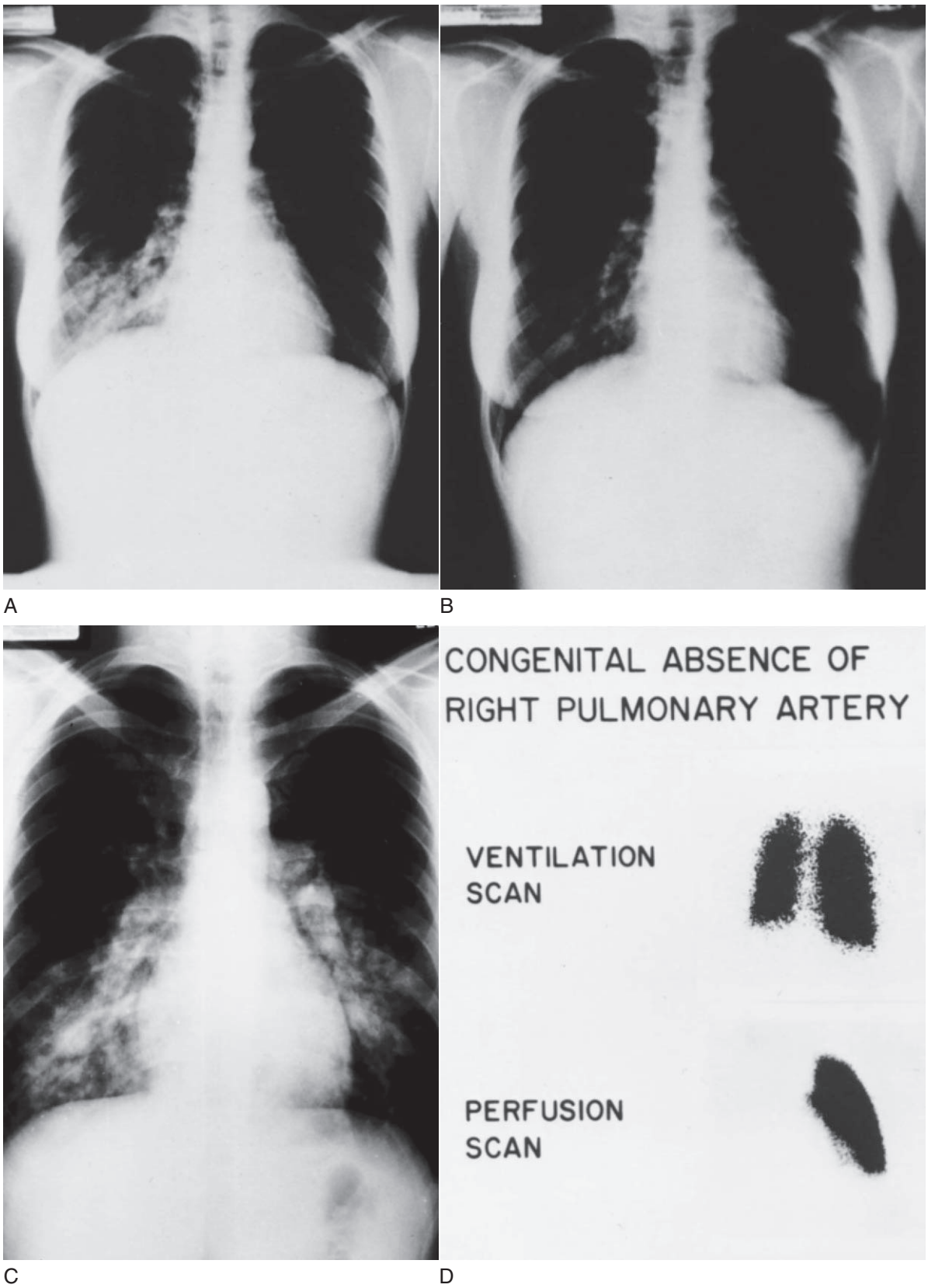

Figure 1-14. **A,** Typical radiograph of high-altitude pulmonary edema (HAPE) in 29-year-old female skier at 2450 m. **B,** Same patient 1 day after descent and oxygen administration, showing rapid clearing. **C,** Bilateral pulmonary infiltrates on radiograph of a patient with severe HAPE after descent (case presented in text). **D,** Ventilation and perfusion scans in person with congenital absence of right pulmonary artery after recovery from HAPE.

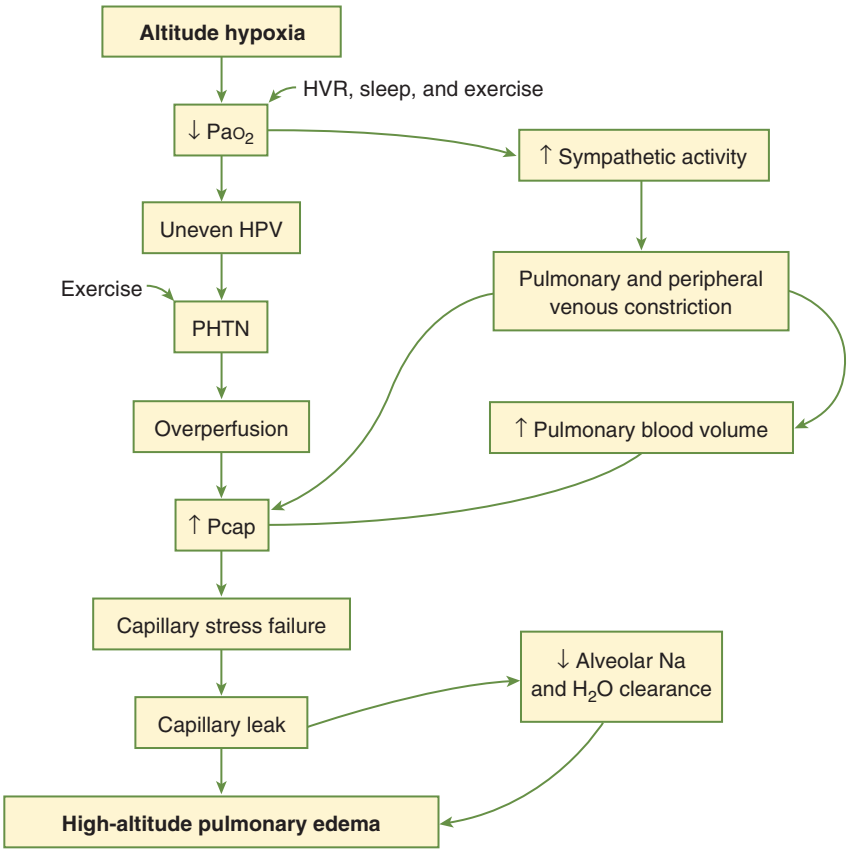

Figure 1-15. Proposed pathophysiology of high-altitude pulmonary edema. HPV, hypoxic pulmonary vasoconstriction; HVR, hypoxic ventilatory response; Pcap, capillary pressure; PHTN, pulmonary hypertension.

perfusion matching at high altitude. The degree of HPVR varies widely among individuals (as well as among species) and is most likely an inherent trait. Persons who are HAPE susceptible have a greater increase in PAP than those who are not (see HAPE Susceptibility). Although other factors, such as the vigor of the ventilatory response and the subsequent alveolar P_{O_2} value, may help determine the ultimate degree of pulmonary hypertension, HPVR appears to be the dominant factor. Because all persons with HAPE have excessive pulmonary hypertension, but not all those with excessive pulmonary hypertension have HAPE, it appears that pulmonary hypertension is a necessary factor but in itself is not the cause of HAPE.

Overperfusion and Capillary Leak. To explain how pulmonary hypertension might lead to edema, Hultgren suggested that in those who develop HAPE, the hypoxic pulmonary vasoconstriction is uneven and the delicate microcirculation in an unconstricted (relatively dilated) area is subjected to high pressure and flow, leading to leakage (edema). The unevenness could be due to anatomic characteristics, such as distribution of muscularized arterioles, or to functional factors, such as loss of HPVR in severely hypoxic regions.[198] Uneven perfusion is suggested clinically by the typical patchy radiographic appearance and is supported by lung scans and MRI during acute hypoxia that show uneven perfusion in persons with a history of HAPE.[184,456] Persons born without a right pulmonary artery are highly susceptible to HAPE (see Figure 1-14D),[151] supporting the concept of overperfusion of a restricted vascular bed as a cause of edema, since the entire cardiac output flows into one lung. Other causes of overperfusion of the pulmonary circulation include left-to-right shunts, such as atrial septal defect (ASD), ventricular septal defect (VSD), and patent ductus arteriosus (PDA). The rapid reversibility of the illness and the response to vasodilators are also consistent with this mechanism. When the hydrostatic pressure is reduced, the alveolar fluid is quickly reabsorbed.

Other factors contributing to increased hydrostatic pressure, such as exercise or a high salt load with subsequent hypervolemia, also may play a role in HAPE. In fact, the authors have several times observed onset of HAPE after a large salt intake. Some studies have also suggested a role for pulmonary venous constriction, which would contribute to increased capillary hydrostatic pressure.[160,275]

The end result of overperfusion and increased capillary pressure[275] is distention, increased filtration of fluid, and even rupture of the capillary–alveolar membrane, termed "stress failure,"[470,471] with subsequent leakage of cells and proteins.

Alveolar Fluid Balance. Fluid filtration into the interstitial and alveolar space, reabsorption back across the epithelial membrane, and clearance of interstitial fluid by lymph result in a dynamic balance that generally prevents alveolar flooding. On ascent to altitude, the hydrostatic pressure gradient for filtration is increased. As might be expected, multiple lines of evidence suggest that extravascular lung water is increased at altitude.[84,136,460] Despite this, very few people develop frank alveolar flooding. This may well be because of differences in hydrostatic pressure in those resistant and susceptible to HAPE. However, persons who contract HAPE appear to also have an impaired ability to clear alveolar fluid. On a constitutive (genetic) basis, HAPE subjects have lower activity of the epithelial sodium channel (ENaC) and therefore reduced ability to transport sodium across the epithelium back into the interstitial space.[280,394,395] (It is the sodium gradient across the epithelium that determines movement of water from the alveolus.) In addition, epithelial sodium transport is diminished by hypoxia per se, so that those with already impaired function become more impaired at altitude. To what extent HAPE may be due to failure of reabsorption of alveolar fluid will require more research[185] (see Figure 1-15).

Control of Ventilation. As in AMS, control of ventilation may play a role in the pathophysiology of HAPE. Those with HAPE had a lower HVR than persons who acclimatized well,[162,285] but not all persons with a low HVR become ill. Thus, low HVR appears to play a permissive, rather than causative, role in the development of HAPE. A brisk HVR, and therefore a large increase in ventilation, appears to be protective. Persons who tend to hypoventilate are more hypoxemic and presumably suffer greater pulmonary hypertension. Possibly more important, a low HVR may permit episodes of extreme hypoxemia during sleep (see Figure 1-6). Supporting this concept is the frequency with which the onset of HAPE occurs during sleep, especially in persons who have ingested sleep medications.[146,162] In addition, a HAPE victim with a low HVR does not mount an adequate ventilatory response to the severe hypoxemia of the illness and may suffer further ventilatory depression through central nervous system (CNS) suppression (hypoxic ventilatory depression). Such persons, when given oxygen, show a paradoxical increase in ventilation.[162] Despite the correlation of HAPE with low HVR, its value as a sea-level predictor is poor.[93]

HAPE Susceptibility

Persons susceptible to HAPE (HAPE-s) show at sea level an abnormal rise of pulmonary artery pressure and pulmonary vascular resistance during a hypoxic challenge at rest and during exercise, and even during exercise in normoxia, suggesting overreactivity of the pulmonary circulation to both hypoxia and exercise.[109,141,224] Part of this reactivity may be related to greater alveolar hypoxemia secondary to lower HVR,[162,182,286] but other factors have been uncovered. Microneurographic recordings from the peroneal nerve during hypoxia established a direct link in HAPE-s between the rise in PAP and greater sympathetic activation,[106] indicating that sympathetic overactivation might contribute to HAPE. Smaller and less distensible lungs have been noted in HAPE-s, which might limit the ability to accommodate increased flow by vascular recruitment and thus result in higher vascular resistance.[109,182,426] Another characteristic of HAPE-s is abnormal endothelial function, as evidenced by reduced nitric oxide (NO) synthesis during hypoxia[46,63,400] and during HAPE,[105] and higher levels of endothelin, a potent pulmonary vasoconstrictor.[397,427] The importance of reduced NO is reinforced by studies showing improvement in pulmonary hemodynamics when either NO or a phosphodiesterase (PDE)-5 inhibitor, sildenafil or tadalafil, was given to HAPE subjects[6,128,272,400] (Fig. 1-16). As mentioned previously, HAPE-s subjects are also characterized by impairment of respiratory transepithelial sodium and water transport, making it harder to reabsorb alveolar fluid[396,399] (see Figure 1-15).

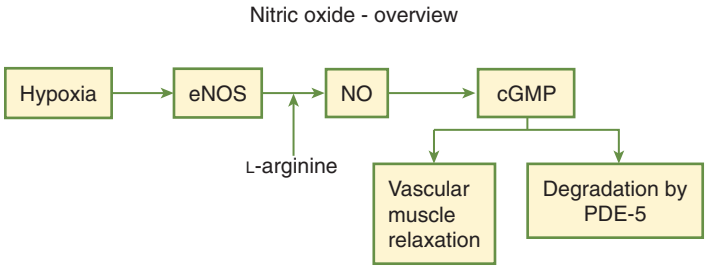

Figure 1-16. The nitric oxide pathway and action of PDE-5 inhibitors. In the presence of a PDE-5 inhibitor, cGMP, the second messenger of NO, is not degraded and vasodilation is therefore enhanced. eNOS, endothelial nitric oxide synthase; NO, nitric oxide; cGMP, cyclic guanosine monophosphate; PDE-5, phosphodiesterase 5.

Genetics of HAPE

The first major genetic studies in lowlanders visiting high altitudes have focused on HAPE because it is the most fully understood pathophysiologic process occurring in lowlanders visiting high altitudes. Although many of the characteristics of HAPE-susceptible persons are apparently genetically determined, actual genetic studies are conflicting and difficult to interpret. Good evidence for a genetic component to HAPE susceptibility comes from study of major human leukocyte antigen (HLA) alleles in 28 male and 2 female subjects with a history of HAPE compared with HLA alleles in 100 healthy volunteers.[168] The HLA-DR6 and HLA-DQ4 antigens were associated with HAPE, and HLA-DR6 with pulmonary hypertension. These findings suggest that immunogenetic susceptibility may underlie the development of HAPE, at least in some cases. Given the importance of endothelial function in HAPE, investigators have looked at gene polymorphisms for endothelial nitric oxide synthase (eNOS). Two recent studies have established a link between HAPE-s and the Glu298Asp alleles of the eNOS gene in Japanese and Indian subjects.[1,103] An additional study of NOS3-related gene polymorphisms that could explain decreased NO synthesis found no relation to HAPE-s.[465] This may be explained by racial differences, or by the specific processes encoded by the different gene fragments. The insertion-deletion polymorphism of the angiotensin-converting enzyme (ACE) gene has also garnered attention, because the renin-aldosterone-angiotensin system (RAAS) is known to be activated in HAPE,[29] and the ACE gene has also been linked to performance at altitude.[310] The I/D polymorphism was not associated with HAPE susceptibility in populations of Indians, Japanese, and whites.[94,186,245] However, one variant of the angiotensin receptor gene was correlated with HAPE susceptibility in Japanese people.[186]

In summary, gene polymorphisms coding for the angiotensin receptor and endothelial nitric oxide distinguish HAPE-sensitive from HAPE-resistant subjects. In the near future, single nucleotide polymorphism scanning techniques, as well as RNA gene expression techniques, will be used to explore genetic contributions to high-altitude physiology and pathophysiology. The search for a genetic basis of HAPE susceptibility continues.[102,318]

Treatment

Treatment choices for HAPE depend on severity of illness and logistics. Early recognition is the key to successful outcome, as with all high-altitude illnesses (see Box 1-4). In the wilderness, where oxygen and medical expertise may be unavailable, persons with HAPE need to be urgently evacuated to lower altitude. However, because of augmented pulmonary hypertension and greater hypoxemia with exercise, exertion must be minimized. If HAPE is diagnosed early, recovery is rapid with a descent of only 500 to 1000 m and the victim may be able to re-ascend slowly 2 or 3 days later. In high-altitude locations with oxygen supplies, bed rest with supplemental oxygen may suffice, but severe HAPE may require high-flow oxygen (4 L/min or greater) for more than 24 hours. Hyperbaric therapy with the fabric pressure bag is equivalent to low-flow oxygen and can help conserve oxygen supplies.[361]

Oxygen immediately increases arterial oxygenation and reduces pulmonary artery pressure, heart rate, respiratory rate, and symptoms. When descent is not possible, oxygen (or a hyperbaric bag) can be lifesaving. Rescue groups should make delivery of oxygen to the victim, by airdrop if necessary, the highest priority if descent is slow or delayed. If oxygen is not available, immediate descent is lifesaving. Waiting for a helicopter or rescue team has too often proved fatal. Since cold stress elevates PAP, the victim should be kept warm.[69] The use of a mask providing pressure (resistance) on expiration (EPAP) was shown to improve gas exchange in HAPE, and this may be useful as a temporizing measure.[405] The same is accomplished with pursed-lip breathing. An unusual case report suggested that a climber may have saved his partner's life by postural drainage to expel airway fluid.[50]

Drugs are of limited necessity in HAPE, because oxygen and descent are so effective. Medications that reduce pulmonary blood volume, PAP, and pulmonary vascular resistance are physiologically rational to use when oxygen is not available or descent is delayed. Singh and associates[421] reported good results with furosemide (80 mg every 12 hours), and greater diuresis and clinical improvement occurred when 15 mg parenteral morphine was given with the first dose of furosemide. Their use, however, has been eclipsed by recent results with vasodilators. The calcium channel blocker nifedipine (30 mg slow release every 12 to 24 hours or 10 mg orally repeated as necessary) was effective in reducing pulmonary vascular resistance and PAP during HAPE, and it slightly improved arterial oxygenation.[25] Clinical improvement, however, was not dramatic. Nifedipine is well tolerated and unlikely to cause significant hypotension in healthy persons, and it avoids the danger of CNS depression from morphine and possible hypovolemia from diuretics. Clinical improvement is much better, however, with oxygen and descent than with any medication. Nitric oxide is a potent pulmonary vasodilator and improves hemodynamics in HAPE, but it is rarely available, and in any event it is usually given with oxygen. The PDE-5 inhibitors, which increase cyclic guanosine monophosphate (cGMP) to produce pulmonary vasodilation during hypoxia[128,356,357] (see Figure 1-16), have shown value for prevention of HAPE[272] but have not yet been studied for treatment. Whether these agents will prove to be more effective than nifedipine for treatment is unknown. A theoretical advantage is that the PDE-5 inhibitors produce less systemic vasodilation. Nifedipine, and perhaps other vasodilators, might be useful as adjunctive therapy but are no substitute for definitive treatment (see Box 1-4).[144]

Figure 1-17. Chest radiograph of severe HAPE in a 4-year-old girl with a small, previously undiagnosed patent ductus arteriosus that predisposed her to HAPE.

After evacuation of the victim to a lower altitude, hospitalization may be warranted for severe cases. Treatment consists of bed rest and oxygen (sufficient to maintain Sa_{O_2}% greater than 90%). Rapid recovery is the rule. A rare instance of progression to adult respiratory distress syndrome has been reported, but it was impossible to exclude other diagnoses completely.[495] Antibiotics are indicated for infection when present. Occasionally, pulmonary artery catheterization or Doppler echocardiography is necessary to differentiate cardiogenic from high-altitude pulmonary edema. Endotracheal intubation and mechanical ventilation are rarely needed. A HAPE victim demonstrating unusual susceptibility, such as onset of HAPE despite adequate acclimatization, or onset below 2750 m, might require further investigation, such as echocardiography, to rule out an intracardiac shunt. In children, undiagnosed congenital heart disease is worth considering (Fig. 1-17). Hospitalization until blood gases are completely normal is not warranted; all persons returning from high altitude are at least partially acclimatized to hypoxemia, and hypocapnic alkalosis persists for days after descent. Distinct clinical improvement, radiographic improvement over 24 to 48 hours, and an arterial P_{O_2} of 60 mm Hg or a Sa_{O_2}% greater than 90% are adequate discharge criteria. Patients are advised to resume normal activities gradually and are warned that they may require up to 2 weeks to recover complete strength. Physicians should recommend preventive measures, including graded ascent with adequate time for acclimatization, and should provide instruction on the use of acetazolamide, nifedipine, or PDE-5 inhibitors for future

ascents. An episode of HAPE is not a contraindication to subsequent high-altitude exposure, but education to ensure proper preventive measures and recognition of early symptoms is critical.

Prevention

The preventive measures previously described for AMS also apply to HAPE: graded ascent, time for acclimatization, low sleeping altitudes, and avoidance of alcohol and sleeping pills (see Box 1-4). Exertion may contribute to onset of HAPE, especially at moderate altitude. Reports from North America at 2500 to 3800 m have included hikers, climbers, and skiers, all of whom were exercising vigorously. Menon[296] clearly showed that sedentary men taken abruptly to higher altitude were just as likely to become victims of HAPE. Prudence dictates not overexerting for the first 2 days at altitude. Considerable clinical experience suggests that acetazolamide prevents HAPE in persons with a history of recurrent episodes. Recent work showing that acetazolamide blocks hypoxic pulmonary hypertension supports this practice.[45,240] Nifedipine (20 mg slow release every 8 hours) prevented HAPE in subjects with a history of repeated episodes.[25] The drug should be carried by such individuals and started at the first signs of HAPE or, for an abrupt ascent, started when leaving low altitude. The PDE-5 inhibitors sildenafil and tadalafil effectively block hypoxic pulmonary hypertension and will also prevent HAPE.[272] The optimal dosage has not been established; regimens for sildenafil have varied from a single dose of 50 or 100 mg just prior to exposure for acute studies,[128,356] to 40 mg three times a day for 2 to 6 days at altitude,[340,357] and for tadalafil, 10 mg every 12 to 24 hours.[115,272] Most recently, and rather surprisingly, dexamethasone was found effective in preventing HAPE in susceptible subjects. Maggiorini and colleagues gave 8 mg dexamethasone every 12 hours, starting 2 days prior to exposure, and found it as effective as tadalafil in reducing PAP and preventing HAPE.[272] Dexamethasone has many actions in the lung, and which action might be responsible for this observed effect is unknown.[27] As evidenced by this list of therapies, any agent that blocks hypoxic pulmonary hypertension will block onset of HAPE, reinforcing the concept of pulmonary hypertension as the sine qua non of high-altitude pulmonary edema.

Reentry Pulmonary Edema

In some persons who have lived for years at high altitude, HAPE develops on re-ascent from a trip to low altitude.[107] Authors have suggested that the incidence of HAPE on re-ascent may be higher than that during initial ascent by flatlanders,[29,200] but data on true incidence are difficult to obtain. Children and adolescents are more susceptible than adults.[107] Hultgren[199] found a prevalence of HAPE in Peruvian natives of 6.4 per 100 exposures in the 1- to 20-year age group, and 0.4 per 100 exposures in persons over 21 years. The phenomenon has been observed most often in Peru, where high-altitude residents can return from sea level to high altitude quite rapidly. Cases have also been reported in Leadville, Colorado,[408] but reports are conspicuously rare from Nepal and Tibet,[482] perhaps because such rapid return back to high altitude is not readily available.[485] Severinghaus[411] has postulated that the increased muscularization of pulmonary arterioles that develops with chronic high-altitude exposure generates an inordinately high pulmonary artery pressure on re-ascent, causing the edema.

OTHER MEDICAL CONCERNS AT HIGH ALTITUDE

High-Altitude Deterioration

The world's highest human habitation is at approximately 5500 m, and above this altitude, deterioration outstrips the ability to acclimatize.[229] The deterioration is more rapid the higher one goes above the maximum point of acclimatization. Above 8000 m, deterioration is so rapid that without supplemental oxygen, death can occur in a matter of days.[461] Life-preserving tasks such as melting snow for water may become too difficult, and death may result from dehydration, starvation, hypothermia, and especially neurologic and psychiatric dysfunction.[387]

Loss of body weight is a prominent feature of high-altitude deterioration. Body weight is progressively lost because of anorexia and malabsorption during expeditions to extreme high altitude. Pugh[345] reported a 14 to 20 kg body weight loss in climbers on the 1953 British Mt. Everest Expedition. Nearly 30 years later, with improvement in food and cooking techniques, climbers on the American Medical Research Expedition to Mt. Everest still lost an average of 6 kg.[54] This was due in part to a 49% decrease in fat absorption and a 24% decrease in carbohydrate absorption. During OEII, in which the "climbers" were allowed to eat foods of their choosing ad libitum, they still suffered large weight losses: 8 kg overall, including 3 kg of fat and 5 kg of lean body weight (muscle).[191,384] At 4300 m, weight loss was attenuated by adjusting caloric intake to match caloric expenditure.[64] Thus, significant weight loss with prolonged exposure to high altitude may be overcome with adequate caloric intake, but decreased appetite is a problem.[226,446] At very high altitudes, an increase in caloric intake may not be sufficient to completely counteract the severe anorexia and weight loss, as other mechanisms may come into play.

At extreme altitude, Ryn[387] reported an incidence of acute organic brain syndrome in 35% of climbers going above 7000 m, in association with high altitude deterioration. This syndrome, with its features of frank psychosis and impaired judgment, could directly threaten survival.

Children at High Altitude

Children born at high altitude in North America appear to have a higher incidence of complications in the neonatal period than do their lower altitude counterparts.[329] In populations better adapted to high altitude over many generations, neonatal transition has not been as well scrutinized, but there does appear to be some morbidity.[473] High-altitude residence does not clearly impact eventual stature, but growth and development are slowed.[92,314] In the developing world, confounding factors such as nutrition and socioeconomic status make these issues difficult to assess.[195] Children residing at high altitude are more likely to develop pulmonary edema on return to their homes from a low-altitude sojourn than are lowland children on induction to high altitude. Some of these children may have pre-existing pulmonary hypertension of various causes.[90]

Lowland children traveling to high altitude are just as likely to suffer AMS as are adults.[344] No data indicate that children are more susceptible to altitude illness, although diagnosis can be more difficult in preverbal children.[489] Despite this somewhat reassuring fact, very conservative recommendations are made regarding taking children to high altitude; it should be made

clear that these opinions are not based on science.[35,343] Durmowicz and colleagues showed that children with respiratory infections were more susceptible to HAPE.[107] Children can be given acetazolamide or dexamethasone as necessary for AMS or HACE. The dosage of acetazolamide for prevention or treatment of AMS in children is 5 mg/kg/day in divided doses. See Pollard and colleagues for an excellent consensus document on children at altitude.[344]

High-Altitude Syncope

Syncope within the first 24 hours of arrival occurs occasionally at moderate altitude[325,326] but is not observed in mountaineers at higher altitudes; it is a problem of acute induction to altitude. The mechanism is an unstable cardiovascular control system, and it is considered a form of neurohumoral (or neurocardiogenic) syncope.[119] An unstable state of cerebral autoregulation may also play a role.[492] These events appear to be random and seldom occur a second time. Preexisting cardiovascular disease is not a factor in most cases. Postprandial state and alcohol ingestion seem to be contributing factors. Altitude syncope has no direct relationship to high-altitude illness.[36]

Alcohol at High Altitude

Two questions regarding alcohol are frequently asked: (1) does alcohol affect acclimatization, and (2) does altitude potentiate the effects of alcohol? Epidemiologic research indicated that 64% of tourists ingested alcohol during the first few days at 2800 m.[183] The effect of alcohol on altitude tolerance and acclimatization might therefore be of considerable relevance. Roeggla and colleagues determined blood gases 1 hour after ingestion of 50 g of alcohol (equivalent to 1 L of beer) at 171 m and again after 4 hours at 3000 m. A placebo-controlled, double-blinded, paired design was used. For the 10 subjects, alcohol had no effect on ventilation at the low altitude, but at the high altitude it depressed ventilation, as gauged by a decreased arterial P_{O_2} (from 69 to 64 mm Hg) and increased P_{CO_2} (from 32.5 to 34 mm Hg).[379] Whether this degree of ventilatory depression would contribute to AMS and whether repeated doses would have greater effect were not tested. Nonetheless, the authors argue that alcohol might impede ventilatory acclimatization and should be used with caution at high altitude.

Conventional wisdom proffers an additive effect of altitude and alcohol on brain function. McFarland, who was concerned about the interaction in aviators, wrote, "The alcohol in two or three cocktails would have the physiological action of four or five drinks at altitudes of approximately 10,000 to 12,000 feet."[291] Also, "Airmen should be informed that the effects of alcohol are similar to those of oxygen want and that the combined effects on the brain and the CNS are significant at altitudes even as low as 8,000 to 10,000 feet."[291] His original observations were made on two subjects in the Andes in 1936. He found that blood alcohol levels rose more rapidly and reached higher values at altitude, but he noted no interactive effect of alcohol and altitudes of 3810 and 5335 m.[292] Most subsequent studies refuted the increased blood alcohol concentration data except at the highest altitudes, over 5450 m. Higgins and colleagues, in a series of chamber studies,[177,178] found blood alcohol levels were similar at 392 m and at 3660 m, and they noted no synergistic effects of alcohol and altitude. Lategola and colleagues[253] found that blood alcohol uptake curves were the same at sea level and at 3660 m, and performance on math tests showed no interaction between alcohol and altitude. In another study of 25 men, performance scores were similar at sea level and at a simulated 3810-m altitude, with a blood alcohol level of 88 mg%.[77] Performance was not affected by hypoxia, only by alcohol, and older subjects were more affected. When more demanding tasks were tested, Collins found that a blood alcohol level of 91 mg% affected performance, as did an altitude of 3660 m during night sessions when the subjects were sleep deprived, but there was no significant interaction between altitude and alcohol.[76] In the one study in which Collins and colleagues were able to discern some altitude effect, there was a simple additive interaction of altitude (hypoxic gas breathing) and alcohol.[78] He concluded that performance decrements due to alcohol might be increased by altitudes of 3660 m (12,000 feet) if subjects are negatively affected by that altitude without alcohol. All of these aviation-oriented studies used acute hypoxia equivalent to no more than 3500 m. Perhaps the highest altitude (without supplemental oxygen) at which alcohol was studied was 4350 m, on the summit of Mt. Evans in Colorado. Freedman and colleagues found that alcohol affected auditory evoked potentials to the same extent as that seen in Denver—that is, no influence of altitude was detectable.[118]

In summary, the limited data on blood gases at altitude after alcohol ingestion support the popular notion that alcohol could slow ventilatory acclimatization and therefore might contribute to AMS. Considerable data at least up to 3660 m, however, refute the belief that altitude potentiates the effect of alcohol. How altitude and alcohol might interact during various stages of acclimatization in individuals at higher altitudes is still unknown.

Thrombosis: Coagulation and Platelet Changes

After deaths due to altitude illness, autopsy findings of widespread thrombi in the brain and lungs, as well as the impression that thrombosis is greater at altitude,[219] have led to many investigations of the clotting mechanism at high altitude. For a review, see Grover and Bärtsch.[139] Although changes in platelets and coagulation have been observed in rabbits, mice, rats, calves, and humans on ascent to high altitude,[174] these generally occur with very rapid ascent. In vivo studies using more realistic ascent profiles up to 4500 m in the mountains, and higher in chambers, have generally not found changes in coagulation and fibrinolysis.[139] Although the increased incidence of thrombosis in soldiers and others at extreme altitude can be attributed to dehydration, polycythemia, and forced inactivity, there is some evidence of enhanced fibrin formation with a stay of a few weeks above 5000 m.[379]

As for thrombosis in HAPE, Singh and colleagues[419] reported increased fibrinogen levels and prolonged clot lysis times during HAPE, attributed to a breakdown of fibrinolysis. These authors also reported thrombotic, occlusive hypertensive pulmonary vascular disease in soldiers who had recently arrived at extreme altitude.[418] A series of experiments by Bärtsch and colleagues, however, carefully examined this issue in well subjects and in those with AMS and HAPE.[22,139] They concluded that HAPE is not preceded by a prothrombotic state and that only in "advanced HAPE" is there fibrin generation, which abates rapidly with oxygen treatment. They considered the coagulation and platelet activation as an epiphenomenon rather than as an inciting pathophysiologic factor, and most likely due to

inflammation from the structural damage to the capillaries or the extreme hypoxemia.

A difficult clinical question is whether ascent to altitude might result in thrombosis in persons with familial thrombophilia, such as factor V Leiden, a common anomaly, or protein C deficiency, antiphospholipid syndrome, or others. Such cases have been reported,[33,53] but cause and effect cannot be established. In addition, persons with a past history of deep vein thrombosis or pulmonary embolism wonder if they are at increased risk at high altitude, as do women on hormonal contraceptives. Unfortunately, the literature does not help provide guidance for these individuals. Some experts empirically recommend an aspirin a day during altitude exposure for such patients, and this seems to be a safe suggestion. Others have raised the possibility that aspirin therapy might cause or exacerbate retinal hemorrhages at high altitude. Although no study has investigated this at altitude, research from patients with diabetic retinopathy has shown no danger of increased hemorrhages from aspirin.[17] In addition, a retinal hemorrhage in a climber was treated with aspirin, and the hemorrhage resolved.[248]

Peripheral Edema

Edema of the face, hands, and ankles at high altitude is common, especially in females. Incidence of edema in at least one area of the body in trekkers at 4200 m was 18% overall, 28% in women, 14% in men, 7% in asymptomatic trekkers, and 27% in those with AMS.[153] Although not a serious clinical problem, edema can be bothersome. The presence of peripheral edema demands an examination for pulmonary and cerebral edema. In the absence of AMS, peripheral edema is effectively treated with a diuretic. Treatment of accompanying AMS by descent or medical therapy also results in diuresis and resolution of peripheral edema. The mechanism is presumably similar to fluid retention in AMS, but it may also be merely due to exercise.[299]

Immunosuppression

Mountaineers have observed that infections are common at high altitude, slow to resolve, and often resistant to antibiotics.[321] On the American Medical Research Expedition to Mt. Everest in 1981, serious skin and soft tissue infections developed. "Nearly every accidental wound, no matter how small, suppurated for a period of time and subsequently healed slowly."[393] A suppurative hand wound and septic olecranon bursitis did not respond to antibiotics but did respond to descent to 4300 m from the 5300-m base camp. Nine of 21 persons had significant infections not related to the respiratory tract. Most high-altitude expeditions report similar problems.

Data from OEII indicated that healthy individuals are more susceptible to infections at high altitude because of impaired T-lymphocyte function; this is consistent with previous Russian studies in humans and animals.[295] In contrast, B cells and active immunity are not impaired. Therefore, resistance to viruses may not be impaired, whereas susceptibility to bacterial infection is increased. The degree of immunosuppression is similar to that seen with trauma, burns, emotional depression, and space flight. The mechanism may be related, at least in part, to release of adrenocorticotropic hormone, cortisone, and beta-endorphins, all of which modulate the immune response. Intense ultraviolet exposure has also been shown to impair immunity. Persons with serious infections at high altitude may need oxygen or descent for effective treatment. Impaired immunity because of altitude should be anticipated in situations where infection could be a complication, such as trauma, burns, and surgical and invasive procedures.

High-Altitude Pharyngitis and Bronchitis

Sore throat, chronic cough, and bronchitis are nearly universal in persons who spend more than 2 weeks at an extreme altitude (over 5500 m).[263,283] All 21 members of the 1981 American Medical Research Expedition to Mt. Everest suffered these problems.[393] Only two of eight subjects in OEII (where the temperature was greater than 21°C [70°F] and relative humidity was greater than 80%) developed cough, and only above 6500 m. Only four had sore throat. Acute hypoxia directly lowers the cough threshold, thus exacerbating high-altitude cough.[283] But other factors are at play. In the field, these problems usually appear without fever or chills, myalgia, lymphadenopathy, exudate, or other signs of infection. The increase in ventilation, especially with exercise, forces obligate mouth breathing at altitude, bypassing the warming and moisturizing action of the nasal mucous membranes and sinuses. Movement of large volumes of dry, cold air across the pharyngeal mucosa can cause marked dehydration, irritation, and pain, similar to pharyngitis. Vasomotor rhinitis, quite common in cold temperatures, aggravates this condition by necessitating mouth breathing during sleep. For this reason, decongestant nasal spray is one of the most coveted items in an expedition medical kit. Other countermeasures include forced hydration, hard candies, lozenges, and steam inhalation.

High-altitude bronchitis can be disabling because of severe coughing spasms. Cough fractures of one or more ribs are not rare.[263] Purulent sputum is common. Response to antibiotics is poor; most victims resign themselves to taking medications such as codeine and do not expect a cure until descent. Bronchitis developed in 13 of 19 climbers above 4300 m on Aconcagua.[347] Mean sputum production was 6 teaspoons per day. All reported that onset was after a period of excessive hyperventilation associated with strenuous activity. Although an infectious etiology is possible, experimental evidence suggests that respiratory heat loss results in purulent sputum and in sufficient airway irritation to cause persistent cough.[290] This is supported by the beneficial effect of steam inhalation and lack of response to antibiotics. Many climbers find that a thin balaclava, porous enough for breathing, traps some moisture and heat and effectively prevents or ameliorates the problem.

Chronic Mountain Sickness

In 1928 Carlos Monge[307] described a syndrome in Andean high-altitude natives that was characterized by headaches, insomnia, lethargy, plethoric appearance, and polycythemia greater than expected for the altitude. Known variously as Monge's disease, chronic mountain polycythemia, and chronic mountain sickness (CMS), the condition has now been recognized in all high-altitude areas of the world.[244,306,338] Both lowlanders who relocate to high altitude and native residents are susceptible. Chinese investigators reported that 13% of lowland Chinese males and 1.6% of females who had relocated to Tibet developed excessive polycythemia (hemoglobin level greater than 20 g/dL blood).[486] The incidence in Leadville, Colorado, is also high in men over age 40 and distinctly low in women.[243] The increased hematopoiesis is apparently related to greater hypoxic stress, which may have a number of causes, such as lung disease, sleep apnea syndromes, and idiopathic hypoventilation. A diag-

nosis of "pure" chronic mountain polycythemia excludes lung disease and is characterized by relative alveolar hypoventilation, excessive nocturnal hypoxemia, and respiratory insensitivity to hypoxia.[257,306] Some studies suggest that even for the degree of hypoxemia, the red blood cell mass is excessive, implying excessive amounts or overactivity of erythropoietin.[475] The new international guidelines propose a hemoglobin value greater than 21 g/dL for men and 19 g/dL for women as essential for the diagnosis, as well as residence above 2500 m and absence of lung disease.[257] The reader is referred to recent reviews for in-depth information.[305,350,352,484] In addition, an international consensus group has recently published their papers on definition and scoring of CMS.[256,257]

Therapy of CMS is routinely successful. Descent to a lower altitude is the definitive treatment. The syndrome reappears after returning to high altitude. Supplemental oxygen during sleep is valuable. Phlebotomy is a common practice and provides subjective improvement, although without significant objective changes.[475] The respiratory stimulants medroxyprogesterone acetate (20 to 60 mg/day)[242] and acetazolamide (250 or 500 mg/day)[359] have also been shown to reduce the hematocrit value by improving oxygenation. Acetazolamide (250 mg) increased nocturnal Sa_{O_2} by 5%, decreased mean nocturnal heart rate by 11% and the number of apnea/hypopnea episodes during sleep by 74%, and decreased hematocrit by 7%.[359] The response to acetazolamide emphasizes the contribution of hypoventilation and nocturnal desaturation to CMS. Another approach was based on the knowledge that ACE inhibitors blunt hypoxia-mediated erythropoietin release. Plata and colleagues showed that 5 mg/day of enalapril for 2 years reduced hemoglobin concentration, packed cell volume, and proteinuria, and reduced the need for phlebotomy.[342] Pulmonary hypertension and right heart failure may also occur in those with CMS.

High-Altitude Pulmonary Hypertension

High-altitude pulmonary hypertension (HAPH) is a syndrome occurring in children and adults living over 2500 m. It is characterized by a mean pulmonary artery pressure greater than 30 mm Hg or a systolic pressure greater than 50 mm Hg, measured at the altitude of residence; it is associated with right ventricular hypertrophy and heart failure, and the absence of CMS.[257] Historical terms for this condition include subacute adult and subacute infantile mountain sickness, and high-altitude heart disease. Symptoms include cough, cyanosis, dyspnea, and signs of right heart failure. Treatment is similar to that for CMS, with relocation to a low altitude the best solution. Other but inferior therapies include supplemental oxygen, and pulmonary vasodilators such as calcium channel blockers, PDE-5 inhibitors, and nitric oxide and prostaglandin inhibitors.[257]

High-Altitude Retinopathy and Ultraviolet Keratitis

See Chapter 25.

COMMON MEDICAL CONDITIONS AND HIGH ALTITUDE

Persons with certain preexisting illnesses might be at risk for adverse effects on ascent to high altitude, either because of exacerbation of their illnesses or because these illnesses might impact acclimatization and susceptibility to altitude illness. Certain populations also require special consideration, such as the very young, the pregnant, and older adults. This section presents an overview of current knowledge regarding these issues. Despite the importance of the interaction of altitude and common medical conditions, research has so far been limited. See the review by Hackett for a more complete discussion.[149] Conditions that can be aggravated by high-altitude exposure are listed in Box 1-5.

Box 1-5. Advisability of Exposure to High and Very High Altitude for Common Conditions (without Supplemental Oxygen)

PROBABLY NO EXTRA RISK
- Young and old
- Fit and unfit
- Mild obesity
- Diabetes
- Previous coronary artery bypass grafting (without angina)
- Mild chronic obstructive pulmonary disease (COPD)
- Asthma
- Low-risk pregnancy
- Controlled hypertension
- Controlled seizure disorder
- Psychiatric disorders
- Neoplastic diseases
- Inflammatory conditions

CAUTION
- Moderate COPD
- Asymptomatic pulmonary hypertension
- Compensated congestive heart failure (CHF)
- Morbid obesity
- Sleep apnea syndromes
- Troublesome arrhythmias
- Stable angina or coronary artery disease
- High-risk pregnancy
- Sickle cell trait
- Cerebrovascular diseases
- Any cause of restricted pulmonary circulation
- Seizure disorder (not on medication)
- Radial keratotomy

CONTRAINDICATED
- Sickle cell anemia (with history of crises)
- Severe COPD
- Symptomatic pulmonary hypertension
- Uncompensated CHF

Respiratory Diseases

Chronic Lung Disease

While oxygen saturation remains above 90% in a normally acclimatizing, healthy, awake person until over 3000 m (see Figure 1-1), persons with hypoxemic lung disease reach this threshold at a lower altitude that depends on the baseline blood oxygen values. As a result, these persons might have altitude-related problems at lower altitudes than would healthy individuals. In terms of their lung disease, improved airflow will

result from decreased air density at high altitude, but hypoxemia, pulmonary hypertension, disordered control of ventilation, and sleep-disordered breathing could all become worse. Unfortunately, few data are available to guide the clinician advising such a person undertaking a trip to altitude.[74] Hypoxic gas breathing at sea level can predict oxygenation at high altitude, but this does not always correlate with symptoms, and it is not convenient. Sea level P_{O_2} values of 68 and 72 mm Hg successfully classified more than 90% of the subjects with a P_{aO_2} greater than 55 mm Hg at simulated altitudes of 1525 m and 2440 m, respectively.[132,133] Such predictions have been further refined with the addition of spirometry.[101] A P_{aO_2} of 55 mm Hg results in a saturation of 90% at high altitude, where there is slight alkalosis. These data suggested that persons with P_{aO_2} values lower than these at sea level might require supplemental oxygen at modest altitudes. However, in the only clinical studies to date, patients with moderate chronic obstructive pulmonary disease (COPD) did quite well at altitude.[135,289] Graham and Houston found that eight subjects with COPD taken to 1920 m had only minor symptoms on ascent, despite the fact that mean P_{aO_2} declined from 66 mm Hg at sea level to 51 mm Hg while at rest, and from 63 to 47 mm Hg with exercise. The patients did acclimatize, with a drop in P_{CO_2} and a corresponding increase in P_{aO_2} over 4 days, the same response as seen in healthy persons. Matthys and colleagues studied 10 patients at a simulated altitude of 2500 m. Mean PAP increased from 21 to 25 mm Hg, P_{aO_2} decreased from 68 to 51 mm Hg at rest, and the only symptom was an increase in fatigue.[289] The authors concluded that travel to this moderate altitude is safe for such patients. They speculated that these persons might have been partially acclimatized because of their hypoxic lung disease, and they were therefore less likely to develop AMS. Unfortunately, no further investigations with sicker patients or at higher altitudes have yet been reported.

Persons with COPD who become uncomfortable at altitude should be treated with oxygen therapy. Oxygen should also be considered for those predicted to become severely hypoxemic.[43] To adjust oxygen therapy at altitude for persons already on supplemental oxygen, the fractional concentration of O_2 in inspired gas (F_{IO_2}) is increased by the ratio of higher to lower barometric pressure (see Table 1-2). Oxygen also improved hemodynamics (lowered blood pressure) and decreased pulsus paradoxus and pulse pressure in patients with COPD at a simulated altitude of 2438 m.[44] With the advent of simple and inexpensive pulse oximetry, patients can be counseled to monitor their oxygen saturation, determine the need for oxygen, and titrate their own oxygen use.

Interestingly, reports of patients with COPD developing altitude illness are absent from the literature. On the other hand, the issue has not been specifically addressed. Any degree of pulmonary hypertension might be expected to increase the likelihood of HAPE, and although this has been clearly demonstrated in other conditions (see High-Altitude Pulmonary Edema), it has not yet been reported with pulmonary hypertension associated with COPD. No research has yet addressed the use of medications such as acetazolamide or medroxyprogesterone in these patients, to determine if respiratory stimulants might improve altitude tolerance.

Cystic Fibrosis

Children with cystic fibrosis have been reported to do poorly at high altitude,[425] and hypoxic testing has also tried to predict the need for supplemental oxygen on ascent in this condition.[331,383] As with COPD, such tests are not particularly useful and tend to underestimate the oxygen requirements, as they are done only during rest and while awake. Supplemental oxygen should be available for these children, and oxygen saturation monitoring might be desirable in certain circumstances. The physician should be liberal with the use of antibiotics and adjunctive therapy for exacerbations at high altitude, given the likely danger of greater hypoxemia and the greater difficulty of treating infections at high altitude. Cystic fibrosis encompasses a wide range of pulmonary impairment; one patient with mild disease was able to hike to 5600 m.[74]

Asthma

The available literature suggests that people with asthma, both residents and sojourners, do well at moderate altitude, primarily because of decreased allergens and pollution.[51,417,452] Indeed, high altitude as a treatment for asthma has been popular in Europe for many decades. The effect is comparable to that seen with high dosages of inhaled steroids.[74] However, because altitude exposure often includes exercise (and cold), people with asthma who have exercise-induced bronchospasm, rather than allergic asthma, might have problems at altitude. Matsuda and colleagues[284] investigated the effect of altitude on 20 asthmatic children with exercise-induced bronchospasm in a hypobaric chamber simulating 1500 m, but with the temperature and humidity held constant. Except for the increased respiratory rate during exercise, as expected, all other physiologic variables were unchanged compared with those at sea level. The authors concluded that the modest altitude of 1500 m does not exacerbate exercise-induced asthma. Golan and colleagues screened travelers and found 147 people with asthma who trekked to high altitude. Risk factors for an asthma attack at altitude were use of inhaled bronchodilators more than three times a week prior to travel, and intensive physical exertion during the trek.[130] A small but careful study was done with 11 people with asthma who were in stable condition with normal respiratory function at sea level. Cogo and her colleagues tested both methacholine challenge and hyperosmolar aerosol at sea level and multiple altitudes up to 5050 m. At no altitude was there an increase in bronchial responsiveness, and at the highest altitude there was a decrease.[73] They concluded that the positive aspects of high altitude prevailed over potential negative factors for people with asthma, and they attributed the benefit to the increase in circulating catecholamines found in their subjects.

In the presence of bronchoconstriction at high altitude, however, hypoxemia is likely to be greater than at low altitude, and for this reason there could be an association between asthma and HAPE or AMS. Reassuringly, no such relationship has yet been reported. Mirrakhimov and colleagues investigated the effect of acetazolamide in 16 asthmatic patients taken to 3200 m. Taking acetazolamide resulted in these patients' having the same benefits as people without asthma, with higher oxygen saturation and fewer AMS symptoms compared with the placebo control group.[303] Seven of the eight asthmatic patients in the control group developed symptoms of AMS, a rather high incidence, but there was no nonasthmatic control group for comparison. Whether this incidence was abnormal is unknown.

Persons with asthma ascending to high altitude should be advised to be at maximal function before ascent, to continue on their usual medications, including steroids, and to have steroids

and bronchodilators with them in the event of an exacerbation. Because airway heat loss can be a trigger for bronchospasm, the use of an airway warming mask might be helpful, but this is unproven.[385] In summary, the available data, although limited, suggest that high altitude does not exacerbate asthma, and that it actually improves allergic asthma. Further work needs to determine if asthma might have any influence on susceptibility to AMS and HAPE; anecdotally, this does not seem to be the case. Although it seems likely that a severe asthma attack at high altitude would be more dangerous than at low altitude, no data are available to answer this question. Although caution and adequate preparation are necessary, asthma is not a contraindication to high-altitude travel.

Pulmonary Vascular Disorders

Because of the danger of HAPE, pulmonary hypertension (of any etiology) is at least a relative contraindication to high-altitude exposure. In addition, hypoxic pulmonary vasoconstriction will most likely exaggerate preexisting pulmonary hypertension and could lead to more significant symptoms in those with congenital cardiac defects, primary pulmonary hypertension (PPH), and related disorders. This caution also applies to unilateral absent pulmonary artery, granulomatous mediastinitis, and restrictive lung diseases, all of which have been associated with HAPE.[151,361,444] As Hultgren has observed, however, some patients with PPH are able to tolerate high altitude, and hypoxic gas breathing can be used to identify an individual's response to hypoxia if clinically indicated. Persons with PPH who must travel to high altitude might benefit from calcium channel blockers, isoproterenol, and/or low flow oxygen.[202] A report by Naeije and colleagues highlighted the increased susceptibility to HAPE in those with pulmonary hypertension: a lowland woman with pulmonary hypertension secondary to fenfluramine developed two episodes of HAPE.[323] The first episode was at 2300 m, and the second one at only 1850 m, with skiing up to 2350 m. Other conditions warranting caution include bronchopulmonary dysplasia, recurrent pulmonary emboli, mitral stenosis, kyphoscoliosis, and scleroderma. Whether pulmonary hypertension is primary or secondary, patients should be made aware of the potential hazards of high altitude, including right heart failure and HAPE. A mean PAP of greater than 30 mm Hg is a useful threshold for caution (or oxygen) on ascent to altitude.[74]

Sleep Apnea, Sleep-Disordered Breathing

Persons with snoring, sleep apnea syndrome, and sleep-disordered breathing (SDB) who become mildly hypoxemic at sea level may become severely hypoxemic at high altitude. This could contribute to high-altitude illness and aggravate attendant problems such as polycythemia, pulmonary hypertension, cardiac arrhythmia, or insomnia. On the other hand, changes in ventilatory control and breathing secondary to altitude hypoxia might conceivably improve certain apnea syndromes. In general, the scant research available has shown that obstructive sleep apnea tends to improve at altitude, whereas central apnea can become worse.[57a] Patients with SDB being treated with continuous positive airway pressure (CPAP) should be aware that the hypobaria of high altitude decreases the delivered pressure of CPAP machines that do not have pressure-compensating features. They therefore might need to adjust their machines. The error is greater at higher altitude and higher initial pressure setting.[120] For those not being treated with CPAP but who exhibit hypoxemia during sleep at low altitude, the physician might want to consider supplemental nocturnal oxygen during an altitude sojourn.

Cardiovascular Conditions

Hypertension

In healthy persons rapidly ascending to high altitude, the change in blood pressure, if any, is variable, depending on the magnitude of hypoxic stress, cold, diet, exercise, and genetic factors. Most studies report a slight increase in blood pressure, associated with increased catecholamine activity and increased sympathetic activity.[348] One well-controlled study showed an increase in blood pressure at 3500 m from a mean of 105/66 mm Hg at sea level to 119/77 mm Hg at 3 days, 111/75 mm Hg at 3 weeks, and back to 102/65 mm Hg on return to sea level.[282] Pugh reported transient increases in blood pressure in athletes at the Olympics in Mexico City.[346] Certain individuals, however, appear to have a pathologic response on induction to high altitude. For example, arterial hypertension develops in 10% of lowland Chinese who move to Tibet.[414] The authors consider this a form of altitude maladaptation and treat the condition by returning the affected individuals to low altitude. After a period of at least 2 months, however, downregulation of adrenergic receptors results in attenuation of the initial blood pressure response. This mechanism is thought to be the reason that long-term residents of high altitude have lower blood pressure than do their sea level counterparts.[201,376] Apparently for the same reason, chronic altitude exposure has also been shown to inhibit progression of hypertension.[302]

As for the effect of short-term altitude exposure on preexisting hypertension, studies have generated mixed results. In general, the response in patients with hypertension is similar to that in those without hypertension—that is, a small increase in blood pressure, with an exaggerated response in some individuals. The greater the hypoxic stress (the higher the altitude), the greater is the change in blood pressure. Altitudes less than 3000 m seem to result in little if any change.[369] Palatini and colleagues studied 12 normotensive patients and 12 untreated mild hypertensive patients with 24-hour ambulatory blood pressure monitoring at sea level, after 12 hours at 1210 m and after 1.5 to 3 hours at 3000 m.[335] The authors concluded that the increase of blood pressure in both normotensive and hypertensive patients was not important at 1200 m but could become so at 3000 m. However, individual variability was great; the maximal change was 17.4 mm Hg for systolic and 16.3 for diastolic blood pressure. Two other studies were able to demonstrate a slightly greater blood pressure response in hypertensive compared with normotensive patients on ascent to 2572 and 3460 m.[89,398] Again, these authors also noted important individual variation, with some subjects increasing their systolic blood pressure by as much as 25 mm Hg at rest and 40 mm Hg during exercise, compared with sea level measurements. The important question of whether the blood pressure would continue to increase over the first 2 weeks at high altitude, as it does in normotensive patients, has not yet been addressed. At a more modest altitude, Halhuber and coworkers claimed a significant reduction in the blood pressure of 593 persons with hypertension after 14 days at 1700 to 2000 m in the Alps.[166] A similar study of hypertensive patients at higher altitude is needed.

Patients receiving antihypertensive treatment should continue their medications while at high altitude. Because some persons

may unpredictably become markedly hypertensive acutely,[202] blood pressure monitoring should be considered, especially in those with labile hypertension or those who become symptomatic at altitude. Hypertension in short-term high-altitude sojourners for the most part should be considered transient and should not be treated, as it rarely reaches dangerously high levels and will resolve on descent. Given the large number of hypertensive patients visiting ski resorts and trekking at high altitude, however, the occasional person with an exaggerated response will require treatment.[202] Because the mechanism appears to be increased alpha-adrenergic activity, an alpha-blocker might be the best choice of therapy for these individuals. A preliminary report also suggested that nifedipine might be useful, and superior to atenolol.[95] The best medication, dosage, and duration still need to be determined. There is no evidence to date to suggest that hypertensive patients are more likely to develop high-altitude illnesses. Although it requires some caution, hypertension does not seem to be a contraindication to high-altitude exposure.

Arteriosclerotic Heart Disease

Life-long residence at high altitude appears to offer some protection from coronary artery disease and the attendant acute coronary artery events,[301] perhaps in part because of increased myocardial vascularity. Other factors that might explain this finding, such as genetics, fitness, and diet, have not been adequately evaluated. The effect on the healthy heart of acute, transient exposure to high altitude also appears to be benign. Various avenues of research have indicated that the healthy heart tolerates even extreme hypoxia quite well, all the way to the summit of Mt. Everest (Pa_{O_2} less than 30 mm Hg). Numerous electrocardiograms (ECGs), echocardiograms, heart catheterizations, and exercise tests have failed to demonstrate any evidence of cardiac ischemia or cardiac dysfunction in healthy persons at high altitudes. This could partly result from the marked reduction in maximal exercise with increasing altitude, which reduces maximal heart rate and myocardial oxygen demand, and also from the increased coronary blood flow. A person with coronary artery disease (CAD), however, may not have the same adaptive capacities. For example, diseased coronary arteries might have limited ability to vasodilate and might actually constrict, because of unopposed sympathetic activation.[261] What, then, are the risks, and what should be the advice to those with CAD considering a visit to high altitude?

Surprisingly little literature is available to help the physician advise such persons. Does high altitude provoke acute coronary events or sudden death? In the United States, no evidence from state or county mortality statistics suggests an increased prevalence of acute coronary events in visitors to high-altitude locations. In Europe, Halhuber and colleagues reported an incidence of only 0.2% for myocardial infarction in 434 patients with CAD taken to altitudes between 1700 and 3200 m for 4 weeks in the Alps.[166] He also reported a very low incidence of sudden death in 151,000 vacationers in the Alps, 69,000 of who were over age 40. In contrast are data from Austria claiming a higher rate of sudden cardiac death in the mountains, compared with the overall risk of sudden cardiac death.[60] However, the altitudes were rather low (1000 to 2100 m), and no increased risk was evident in men who participated regularly in sports. The authors suggested that abrupt onset of exercise in sedentary men combined with altitude stress might induce cardiac sudden death, but whether altitude contributed at all is unclear. In summary, limited data suggest no increased risk for sudden cardiac death or myocardial infarction at altitudes up to 2500 m.

Another important question is whether altitude will exacerbate stable ischemia. The slight increase in heart rate and blood pressure on initial ascent to altitude might exacerbate angina in those with coronary artery disease, as described by Hultgren.[202] One study evaluated nine men with stable exercise-induced angina by exercise treadmill test at 1600 m (Denver), and within the first hour of arrival at 3100 m.[316] Cardiac work was slightly higher for a given workload at high altitude compared with low altitude, and as a result, the onset of angina was at a slightly lower workload. The authors found that a heart rate of 70% to 85% of the rate that produced ischemia at low altitude was associated with angina-free exercise at 3100 m, and they suggested that angina patients at altitude adjust their activity level on the basis of heart rate, at least on the day of arrival.[316] Brammel and colleagues reported similar results and suggested that those with angina need to reduce their activity at high altitude to avoid angina episodes.[56] In a more recent study, Levine and colleagues investigated 20 men who were much older than those in the previous investigations (mean age, 68 ± 3 years), and they performed symptom-limited exercise tests.[261] With acute exposure to 2500 m, the double product (heart rate times systolic blood pressure) required to induce 1 mm ST depression was decreased about 5%, but after 5 days of acclimatization at 2500 m, this value was unchanged from sea level. The degree of ischemia (maximal ST-segment depression) was the same at sea level, with acute altitude exposure, and after 5 days at 2500 m. Also, no new wall motion abnormalities on echocardiography were seen at high altitude. Only one subject exhibited increased angina at altitude, and one person with severe coronary artery disease developed a myocardial infarction after maximal exercise at 2500 m. The authors concluded that patients with CAD who are well compensated at sea level do well at a moderate altitude after a few days of acclimatization, but that acutely, the angina threshold may be lower and activity should be reduced.[261] Finally, a study of 97 older adults visiting 2500 m, many with CAD and abnormal ECGs, found no new ECG changes and no events suggestive of ischemia. In contrast to the Levine study, these subjects did not do exhaustive exercise tests but merely their usual activities, which included walking in the mountains.[369] Taken altogether, these various investigations indicate that those with CAD, including older adults, generally do well at the modest altitude of 2500 m, but that reducing their activities the first few days at altitude is wise.

To address the question of whether altitude might provoke cardiac arrhythmia, Levine and colleagues, in their study mentioned previously,[261] found that premature ventricular contractions (PVCs) increased 63% on acute ascent but returned to baseline after 5 days of acclimatization. A simultaneous rise in urine norepinephrine in these subjects indicated that sympathetic activation was the cause of the increased ectopy. They observed no increase in higher-grade ectopy, however, and no changes in signal-averaged ECG suggestive of a change in fibrillation threshold; in other words, the PVCs appeared benign. Halhuber and coworkers also found increased ectopy in their subjects, and also no serious adverse events.[166] In addition, Alexander described asymptomatic PVCs and ventricular bigeminy in himself while trekking to 5900 m. Subsequent evaluation found no evidence of heart disease, and the event prompted him to thoroughly review the subject of altitude, age,

and arrhythmia.[4] Although no dangerous arrhythmias have ever been reported in high-altitude studies, persons with troublesome or high-grade arrhythmia have not been evaluated on ascent to high altitude. The available evidence suggests that patients whose arrhythmias are well controlled on medication should continue the medication at altitude, whereas those with poorly controlled arrhythmias might do better to avoid visiting high altitude.

In terms of advising persons with CAD or high likelihood of CAD about altitude exposure, the stress of high altitude on the coronary circulation appears to be minimal at rest but significant in conjunction with exercise. Ideally, no one with known CAD or even risk factors for CAD should undertake unaccustomed exercise at any altitude, and especially at high altitude. Therefore, advising an exercise program at sea level prior to exercising at altitude is prudent. The same technique of risk stratification that is commonly used at sea level can be applied for providing advice for high altitude.[196] Using the standard recommendations, asymptomatic men over age 50 with no risk factors require no testing. For asymptomatic men over age 50 with risk factors, an exercise test is recommended to determine risk status prior to exercising at high altitude, and then further evaluation as indicated. Patients with previous myocardial infarction, bypass surgery, or angioplasty are considered at high risk only if they have a strongly positive exercise treadmill test. Patients with multiple-vessel bypass grafts who were asymptomatic, and who had normal exercise test results at sea level, have successfully visited altitudes over 5000 m. High-risk patients may require coronary angiography to establish appropriate management. Alexander has proposed different criteria for those with CAD at high risk at altitude: an ejection fraction less than 35% at rest, a fall in exercise systolic blood pressure, ST-segment depression greater than 2 mm at peak heart rate, and high-grade ventricular ectopy.[3] For these persons, he recommends ascent to no more than 2500 m, and proximity to medical care. Both sets of recommendations, while reasonable, need to be validated with outcome studies.

Heart Failure

Although information on the effect of high altitude on heart failure is scant, physicians in resort areas have noted a tendency toward acute decompensation within 24 hours of arrival in those with a history of heart failure. Those with CAD and low ejection fractions (less than 45%), but without active heart failure, actually did quite well, as gauged by exercise tests during acute exposure to 2500 m.[112] Compared with 23 control subjects, the decrement in exercise performance was similar, and no complications or signs of ischemia developed. Although these results are encouraging for such patients, observations were not made past the first few hours at altitude. One concern is that those with heart failure might be more likely to retain fluid at altitude, especially if AMS were to develop, and that this could aggravate failure. Supporting this notion, Alexander found that ejection fraction declined at altitude during an exercise study in patients with angina, with an increase in end-diastolic and systolic volume as measured by two-dimensional echocardiogram.[3] Ventricular contractility was not depressed, however, and these changes were attributed to fluid overload. Patients with heart failure need to be informed about possible consequences of high-altitude exposure. In particular, they need to avoid AMS (which is associated with fluid retention), continue their regular medications, and be prepared to increase their diuretic should symptoms of failure exacerbate. Acetazolamide prophylaxis may be useful to consider for speeding acclimatization, inducing diuresis, and preventing AMS, but its efficacy in these patients remains untested.

Obesity

The interaction of obesity and altitude has not received much attention. A small study suggested a slight increased susceptibility to AMS in mildly obese men,[124] possibly due to lower nocturnal oxygen saturation that was present despite greater than normal hypoxic and hypercapnic chemosensitivity.[125] Another, larger study examined men with metabolic syndrome (obesity, hypertension, diabetes, and hyperlipidemia) with a 3-week stay at 1700 m and found normal altitude responses and a loss of body fat.[142] In fact, altitude has been suggested as a treatment for obesity. Obese men and women (mean body mass index, 47.1) who were permanent residents at a mean altitude of 2448 m, however, had a 96% incidence of systolic pulmonary artery hypertension (>30 mm Hg), which was related to alveolar hypoventilation.[449]

Sickle Cell Disease

Sickle cell (SC) crisis is a well-recognized complication of high-altitude exposure.[117] Even the modest altitude of a pressurized aircraft (1500 to 2000 m) causes 20% of persons with hemoglobin SC and sickle-thalassemia genetic configuration to have a vaso-occlusive crisis.[278] High-altitude exposure may precipitate the first vaso-occlusive crisis in persons previously unaware of their condition. Persons with sickle cell anemia and a history of vaso-occlusive crises are advised to avoid altitudes over 1800 m unless they are taking supplemental oxygen. Persons with sickle cell disease who live at high altitude in Saudi Arabia have twice the incidence of crises, hospitalizations, and complications as do Saudis at low altitude. Splenic infarction syndrome has been reported more commonly in those with sickle cell trait than in those with sickle cell anemia, probably because sickle cell disease produces autosplenectomy early in life. Frequent reports in the literature emphasize the need to consider splenic syndrome caused by sickle cell trait in any person with left upper quadrant pain, even at an altitude of only 1500 m.[247,278] A number of authors have suggested that non-black persons with the trait may be at greater risk for splenic syndrome at high altitude than are black persons.[247] Treatment of splenic syndrome consists of intravenous hydration, oxygen, and removal to a lower altitude.[442] The overall incidence of problems in persons with the trait is low, however, and no special precaution other than recognition of the splenic syndrome is recommended. The U.S. Army, for example, does not consider soldiers with the trait unfit for duty at high altitude.[100]

Pregnancy

In high-altitude natives, pregnancy-associated hypertension is 4 times more common than in low-altitude pregnancies, preeclampsia is more common, and full-term infants are small for gestational age.[311,314] These problems raise the issue of whether short-term altitude exposure may also pose a risk. So far, there is no evidence that these problems, or others such as spontaneous abortion, abruptio placentae, or placenta previa, can result from a sojourn at high altitude.[328] Unfortunately, however, few data exist on the influence of a high-altitude visit during pregnancy on the mother and the fetus. For moderate

altitude, the research to date has been reassuring.[193,194] Artal and colleagues studied seven sedentary women at 34 weeks gestation. Maximal and submaximal exercise tests were completed at sea level and 6000 feet (1830 m) after 2 to 4 days of acclimatization.[11] They reported the expected decrease in maximal aerobic work but found no difference from sea level in fetal heart rate responses, or in maternal lactate, epinephrine, and norepinephrine levels. In a small number of subjects, the authors considered it safe for third-trimester women to engage in brief bouts of exercise at moderate altitude. A similar conclusion was reached in a study of 12 pregnant women who exercised after ascent to 2225 m. The authors found no abnormal fetal heart rate responses and considered the exercise at altitude benign for both mother and fetus.[38] Huch also concluded that short-term exposure, with exercise, was safe during pregnancy.[193] In summary, the available data, though limited, indicate that short-term exposure to altitudes up to 2500 m, with exercise, is safe for a lowland woman with a normal pregnancy.

Another avenue of research has been alteration of blood gases during pregnancy. Human and animal studies with acute hypoxic challenge, as well as oxygen-breathing studies, have drawn two conclusions: (1) that a compromised placental–fetal circulation could be unmasked at high altitude, and (2) that a fetus with a normal placental–fetal circulation seems to tolerate a level of acute hypoxia far exceeding a moderate altitude exposure.[18,81,362]

On the basis of the available research, it seems prudent to recommend that only women with normal, low risk pregnancy undertake a sojourn at high altitude. For these women, exposure to an altitude at which Sao_2 will remain above 85% most of the time (up to 3000 m altitude) appears to pose no risk of harm, but further study is needed to place these recommendations on a more solid scientific footing. An ultrasound or other assessment may be useful to rule out the more common complications prior to travel. Of course, it is not the altitude per se that determines whether the fetus becomes stressed but rather the maternal (and fetal) arterial oxygen transport. A woman with high-altitude pulmonary edema at 2500 m, for example, is much more hypoxemic than a healthy woman at 5000 m. Therefore, a strategy for preventing altitude illness, especially pulmonary edema, must be explained and implemented. Similarly, elevated carboxyhemoglobin from smoking, lung disease, and other problems of oxygen transport will render the pregnant patient at altitude more hypoxemic, and physiologically comparable to a higher altitude. Consideration of a high-altitude sojourn in the developing world, or in a wilderness setting, raises other issues that may be more important than the modest hypoxia. These include remoteness from medical care should a problem arise, quality of available medical care, use of medications for such important things as malaria and traveler's diarrhea (many of which are contraindicated in pregnancy), and risks for trauma.

The references for this chapter can be found on the accompanying DVD-ROM.

2 Avalanches

Knox Williams, Dale Atkins, and Colin K. Grissom

An avalanche is a mass of snow that slides down a mountainside. In the United States, approximately 100,000 avalanches occur annually, of which about 100 cause injury, death, or destruction of property. Based on a database of reported incidents, about 200 people a year are caught in avalanches (that is, they are bodily involved in the moving snow or its effects). Of these, 84 are partly or wholly buried, 30 sustain injury, and 30 are killed. Average annual property damage varies tremendously depending on the winter. In the last 10 years, damages have ranged from as low as $30,000 to a high of $13.5 million; the median is $298,000. This chapter describes the properties of the mountain snowpack that contribute to avalanche formation and describes avalanche safety techniques.

PROPERTIES OF SNOW

Physical Properties

Although snow cover appears to be nothing more than a thick, homogeneous blanket covering the ground, it is in fact one of the most complex materials found in nature. It is highly variable and goes through significant changes in relatively short periods.

In nature, snow cover is variable on both the broad geographic scale (Antarctic snow is quite different from snow found in the Cascade Mountains of North America) and on the microscale (where snow conditions may vary greatly from one side of a rock or tree to the other). All snow crystals are made

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of the same substance, the water molecule, but local environmental conditions control the type and character of snow found at a given location. At a single site, the snow cover varies from top to bottom, resulting in a complex, layered structure.

Individual layers may be quite thick or very thin. In general, thicker layers represent consistent conditions during one storm, when new snow crystals falling are of the same type, wind speed and direction vary little, and temperature and precipitation are fairly constant. Thinner layers, perhaps only millimeters in thickness, often reflect conditions between storms, such as the formation during fair weather of a melt–freeze crust, a period of strong winds creating a wind crust, or the occurrence of surface hoar, the winter equivalent of dew. Delicate feather-shaped crystals of surface hoar deposited from the moist atmosphere onto the cold snow surface overnight offer a beautiful glistening sight as they reflect the sun of the following day. However, they are very fragile and weak, and once buried by subsequent snowfalls, they may be major contributors to avalanche formation.

One property of snow is strength, or hardness, which is of great importance in terms of avalanche formation. Snow can vary from light and fluffy, easy to shovel, and especially delightful to ski through, to heavy and dense, impossible to penetrate with a shovel, and hard enough to make it very difficult for a skier to carve a turn, even with sharp metal edges. The arrangement of the ice skeleton and the changing density (mass per unit volume) produce this wide range of conditions. In the case of snow, density is determined by the volume mixture of ice crystals and air. The denser the snow layer, the harder and stronger it becomes, as long as it is not melting.

The density of new snow can have a wide range of values. This depends on how closely the new snow crystals pack together, which is controlled by the shape of the crystals. The initial crystals have a variety of shapes, and some pack more closely together than others (Fig. 2-1). For example, needles pack more closely than stellars and as a consequence may possess a density 3 to 4 times that of stellars.

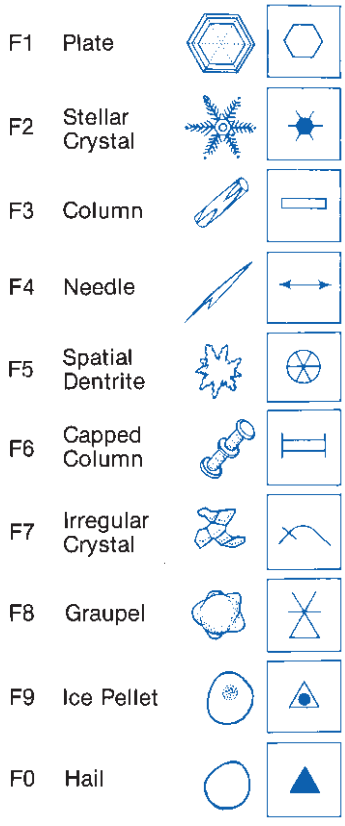


Figure 2-1. International classification of solid precipitation. (From the International Association of Scientific Hydrology, with permission.)

Wind can alter the shape of new snow crystals, breaking them into much smaller pieces that pack very closely together to form *wind slabs*. These in turn may possess a density 5 to 10 times that of new stellars falling in the absence of wind. Because these processes occur at different times and locations at the surface of the snow cover and are buried by subsequent snowfalls, a varied, nonhomogeneous layered structure results. Therefore, what may seem to the casual observer to be minor variations in atmospheric conditions can have an important influence on the properties of snow.

After snow has been deposited on the ground, the density increases as the snow layer settles vertically or shrinks in thickness. Because an increase in density equals an increase in strength, the rate at which this change occurs is important with respect to avalanche potential. Snow can settle simply because of its own weight. It is highly compressible because it is composed mostly of empty air pockets within an ice skeleton of snow crystals. In a typical layer of new snow, 85% to 95% of the volume is empty air pockets. Individual ice crystals can move and slide past each other, and because the force of gravity causes them to move slowly downward, the layer shrinks. The heavier the snow above is and the warmer the temperature, the faster this settlement proceeds.

At the same time, the complex, intricate shapes that characterize the new snow crystals begin to change. They become rounded and suitable for closer packing. Intricate crystals change because they possess a shape that is naturally unstable. New snow crystals have a large surface area-to-volume ratio and are composed of crystalline solid close to its melting point. In this aspect, snow crystals are almost unique among materials found in nature. Surface energy physics dictates that this unstable condition will change; the warmer the temperature is, the faster the change. Under very cold conditions, the original shapes of the snow crystals are recognizable after they have been in the snow cover for several days or even a week or two. As temperatures warm and approach the melting point, such shapes disappear within a few hours to a day. Changes in the shape or texture of snow crystals are examples of initial metamorphism. The geologic term *metamorphism* defines changes that result from the effects of temperature and pressure. As the crystal shapes simplify, they can pack more closely together, enhancing further settlement (Fig. 2-2).

The changes generally occur within hours to a few days. The structure of snow cover changes over a period of weeks to months via other processes. Settlement, which may initially have been rapid, continues at a much slower rate. Other factors begin to exert dominant influences on metamorphism. These factors include the difference in temperature measured upward or downward in the snow layer, called the temperature gradient.

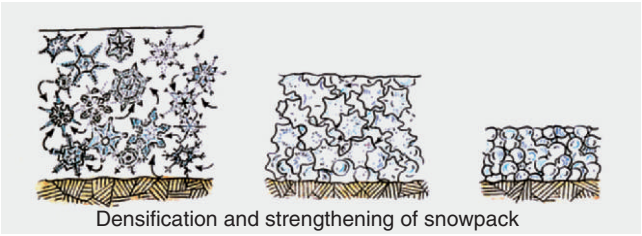


Figure 2-2. Settlement. As the crystal shapes become more rounded, the crystals can pack more closely together, and the layer settles or shrinks in thickness.

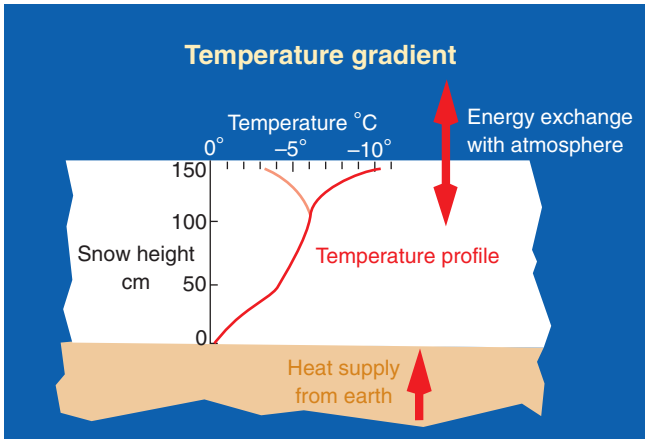


Figure 2-3. When an insulating layer of snow separates the warm ground from the cold air, a temperature gradient develops across the snow layer.

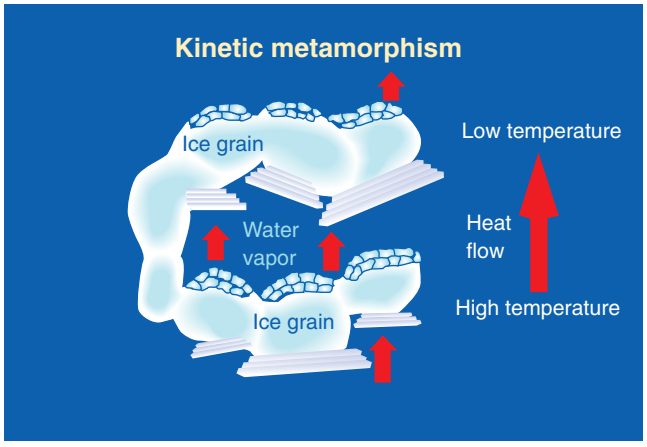


Figure 2-4. In the temperature gradient process, ice sublimates from the top of one grain, moves upward as water vapor, and then is deposited on the bottom surface of the grain above. If conditions allow this process to continue long enough, all of the original grains are lost as the recrystallization produces a layer of new crystals.

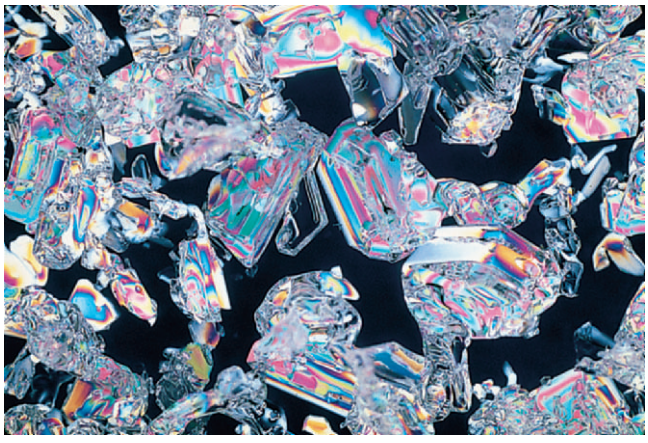
Averaged over 24 hours, snow temperatures generally are coldest near the surface and warmest near the ground at the base of the snow cover, creating a temperature gradient across a snow layer sandwiched between cold winter air and relatively warm ground (Fig. 2-3). The temperature gradient crosses both ice and large void spaces filled with air. Within the ice skeleton, the temperature adjacent to the ground is warmer than that of the snow layer just above, and this pattern continues through the snow cover in the direction of the colder surface.

Warm air contains more water vapor than cold air; this holds true for the air trapped within the snow cover. The greater the amount of water vapor, the greater the pressure. Therefore, both a pressure gradient and a temperature gradient exist through the snow cover. When a pressure difference exists, the difference naturally tends to equalize, just as adjacent high and low atmospheric pressure centers cause movement of air masses. Pressure differences within snow cause vapor to move upward through the snow layers. The air within the layers of the snow cover is saturated with water vapor, with a relative humidity of 100%. When air moves upward to a colder layer, the amount of water vapor that can be supported in the air pocket diminishes. Some vapor changes to ice and is deposited on the surrounding ice grains. We witness a similar process when warm, moist air in a heated room comes in contact with a cold windowpane. The invisible water vapor is cooled to its ice point, and some of the vapor changes state and is deposited as frost on the window.

Figure 2-4 shows how the texture of the snow layer changes during this temperature-gradient process. Water molecules sublimate from the upper surfaces of a grain. The vapor moves upward along the temperature (and vapor) gradient and is deposited as a solid ice molecule on the underside of a colder grain above. If this process continues long enough (it continues as long as a strong temperature gradient exists), all grains in the snow layer are transformed from solid to vapor and back to solid again; that is, they recrystallize. New crystals are completely different in texture from their initial form. They become large, coarse grains with facets and sharp angles and may eventually evolve into a hollow cup form. Examples of these crystals are shown in Figure 2-5. The process is called temperature-gradient metamorphism, or kinetic metamorphism, and well-developed crystals are commonly known as depth hoar.

Depth hoar is of particular importance to avalanche formation. It is very weak because there is little or no cohesion or bonding at the grain contacts. Depth hoar or temperature-gradient snow layers can be compared to dry sand. Each grain may possess significant strength, but a layer composed of grains is very weak and friable because the grains lack connections. Thus depth hoar is commonly called "sugar snow." Depth hoar usually develops whenever the temperature gradient is equal to or greater than about 10°C (18°F) per meter. In the cold, shallow snow covers of a continental climate, such as that of the Rocky Mountains, a gradient of this magnitude is common within the first snow layers of the season. Therefore, a layer of depth hoar is frequently found at the bottom of the snow cover, and the resulting low strength becomes a significant factor for future avalanches.

In the absence of a strong temperature gradient, a totally different type of snow texture develops. When the gradient is less



A

B

Figure 2-5. **A,** Mature depth hoar grains. Facets and angles are visible. Grain size, 3 to 5 mm. **B,** Advanced temperature-gradient grains attain a hollow cup-shaped form. Size, 4 mm. (Polarized-light photos by Doug Driskell.)

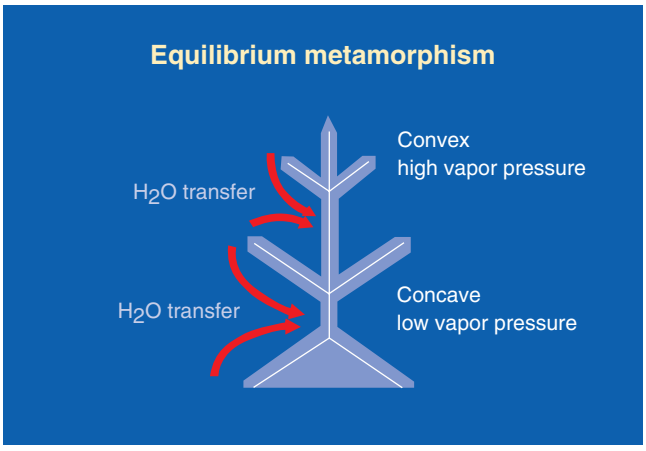


Figure 2-6. In the equilibrium metamorphism process, ice molecules sublimate from crystal points (convexities) and redeposit on flat or concave areas of the crystal.

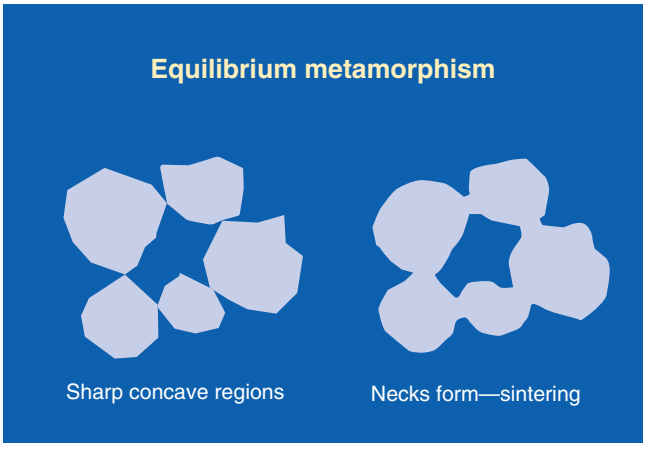


Figure 2-7. Equitemperature grain growth. In the presence of weak temperature gradients, bonds grow at the grain contacts.

than about 10°C (18°F) per meter, there is still a vapor pressure difference, but upward movement of vapor through the snow layers is at a much slower rate. As a result, water vapor deposited on a colder grain tends to cover the total grain in a more homogeneous manner, rather than showing the preferential deposition characteristic of depth hoar. This process produces a grain with a smooth surface of more rounded or oblong shape. Over time, vapor is deposited at the grain contacts (concavities), as well as over the remaining surface of the grain (convexity) (Fig. 2-6). Connecting bonds formed at the grain contacts give the snow layer strength over time (Fig. 2-7). Bond growth, called sintering, yields a cohesive texture, in complete contrast to the cohesionless texture of depth hoar. This type of grain has been referred to by various terms (destructive metamorphism, equitemperature metamorphism, and equilibrium metamorphism) but can generally be described as fine-grained or well-sintered (bonded) snow. Bonded and interconnected grains are shown in Figure 2-8.

The preceding paragraphs describe the big picture in terms of what happens to snow layers after they have been buried by

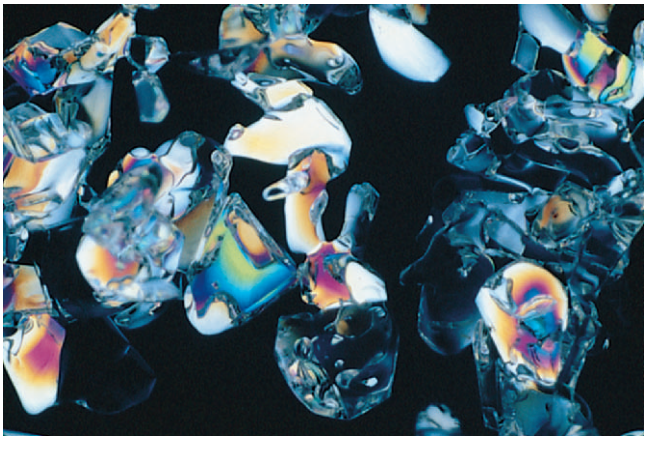


Figure 2-8. Bonded or sintered grains resulting from equitemperature metamorphism. Grain size, 0.5 to 1 mm. (Polarized-light photo by Doug Driskell.)

subsequent snowfalls. If the layer is subfreezing (i.e., if no melt is taking place), one of the two processes described previously is occurring, or perhaps a transition exists between the two. Within the total snow cover, these processes may occur simultaneously, but only one can take place within a given layer at a given time. Both processes accelerate with warmer snow temperature because more water vapor is involved. The temperature gradient across the layer determines whether the process involves the growth of weak depth hoar crystals or the development of a stronger snow layer with a sintered, interconnected texture.

Slab Avalanche Formation

There are two basic types of avalanche release. The first is a point-release, or loose snow, avalanche (Fig. 2-9). A loose snow avalanche involves cohesionless snow and is initiated at a point, spreading out laterally as it moves down the slope to form a characteristic inverted V shape. A single grain or a clump of grains slips out of place and dislodges those below on the slope, which in turn dislodge others. The avalanche continues as long as the snow is cohesionless and the slope is steep enough. This type of avalanche usually involves only small amounts of near-surface snow.

The second type of avalanche, the slab avalanche, requires a cohesive snow layer poorly anchored to the snow below because of the presence of a weak layer. The cohesive blanket of snow breaks away simultaneously over a broad area (Fig. 2-10). A slab release can involve a range of snow thicknesses, from the near-surface layers to the entire snow cover down to the ground. In contrast to a loose snow avalanche, a slab avalanche has the potential to involve very large amounts of snow.

To understand the conditions in snow cover that contribute to slab avalanche formation, it is essential to reemphasize that snow cover develops layer by layer. Although a layered structure can develop by metamorphic processes, distinct layers develop in numerous other ways, most of which have some influence on avalanche formation. The layered structure is directly tied to the two ingredients essential to the formation of slab avalanches: the cohesive layer of snow and the weak layer beneath. If the snow cover is homogeneous from the ground to the surface, there is no danger of slab avalanches, regardless of the snow type. If the entire snow layer is sintered, dense, and strong, stability is very high. Even if the entire snow cover is composed of a very weak layer of depth hoar, there is still no hazard from slab avalanches because the cohesionless character does not allow propagation of the cracks necessary for slab avalanches to form. However, the combination of a basal layer of depth hoar with a cohesive layer above, for example, provides exactly the ingredients for slab avalanche danger. For successful evaluation of slab avalanche potential, information is needed about the entire snowpack, not just the surface. A hard wind slab at the surface may seem strong and safe to the uninitiated, but when it rests on a weaker layer, which may be well below the surface, it may fail under the weight of a skier and be released as a slab avalanche.

Many snow structure combinations can contribute to slab formation. One scenario involves thick layers of weak snow, which result from the development of depth hoar early in the season. The typical combination of climatic factors that produce these layers is early winter snowfalls followed by several weeks of clear, cold weather. Even at higher elevations in the mountains, snow cover on the slopes with a southerly aspect may melt off during a period of fair weather. However, in October and early November, the sun angle is low enough that steep slopes with a northerly aspect receive little or no direct heating from the sun. Snow remains on the ground but not without change. Snow on north-facing slopes experiences optimal conditions for depth hoar formation: a thin, low-density snow cover (maximum opportunity for vapor flow) is sandwiched between

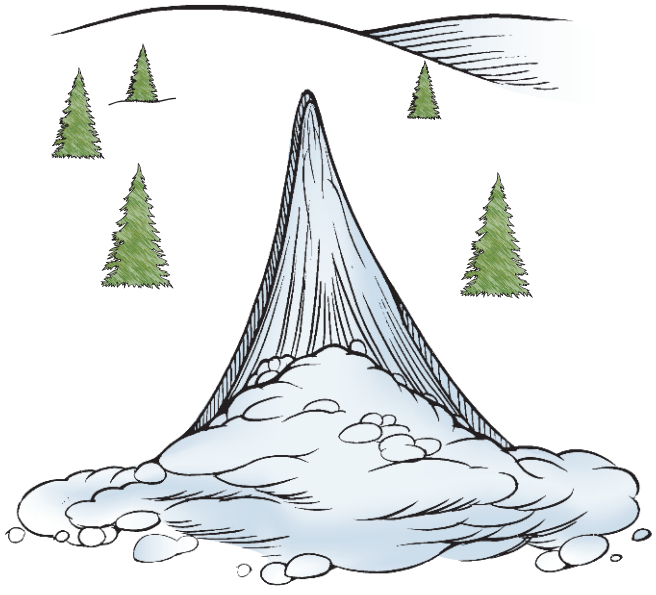


Figure 2-9. Loose snow or point-release avalanche. (Photo courtesy USDA Forest Service.)

Figure 2-10. Slab avalanche. (From USDA Forest Service: Williams K, Armstrong B: The Snowy Torrents. Jackson, WY, Teton Bookshop, 1984, with permission. Photo by Alexis Kelner.)

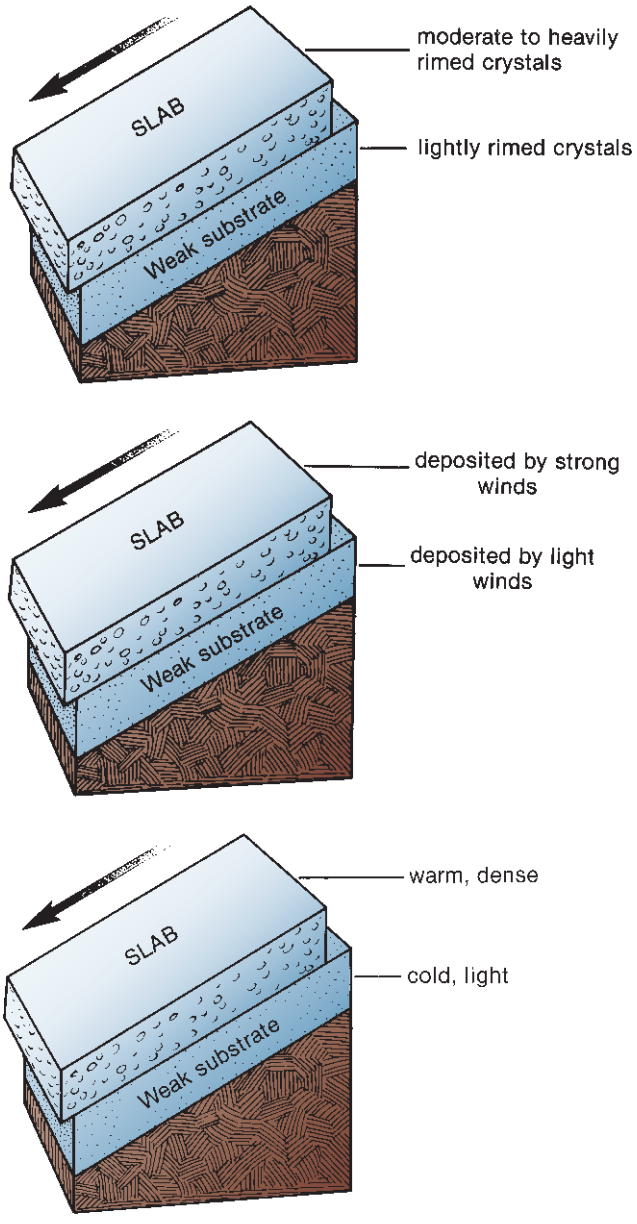


Figure 2-11. Snow layer combinations that often contribute to avalanche formation.

the warm ground, still retaining much of its summer heat, and the cold air above. This snow layer recrystallizes over a period of weeks. When the first large storm of winter arrives in November, cohesive layers of wind-deposited snow accumulate on a very weak base, setting the scene for a widespread avalanche cycle. Figure 2-11 describes other combinations that result in brittle or cohesive layers of snow on a weak layer.

Mechanical Properties: How Snow Deforms on a Slope

Almost all physical properties of snow can be easily seen or measured. A snowpit provides a wealth of information regarding these properties, layer by layer, throughout the thickness of the snow cover. However, even detailed knowledge of these properties does not provide all the information necessary to evaluate avalanche potential. The current mechanical state of the snow cover must be considered. Unfortunately, for the average person these properties are virtually impossible to measure directly.

Mechanical deformation occurs within the snow cover just before its failure and the start of a slab avalanche. Snow cover has a tendency to settle simply from its own weight. When this occurs on level ground, the settlement is perpendicular to the ground and the snow layer increases in density and gains in strength. The situation is not so simple when snow rests on a slope. The force of gravity is divided into two components, one

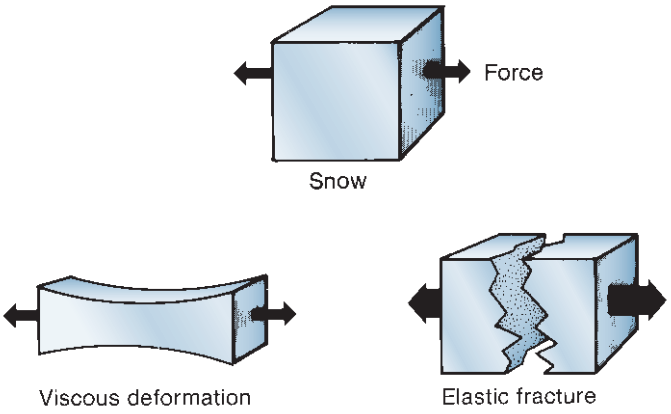


Figure 2-12. Depending on prevailing conditions, snow may deform and stretch in a viscous or flowing manner, or it may respond more like a solid, and fracture.

Figure 2-13. The consistent 90-degree angle between crown face and bed surface of the avalanche shows that slab avalanches result from an elastic fracture. (Photo by A. Judson.)

tending to cause the snow layer to shrink in thickness, and a new component acting parallel to the slope, which tends to pull the snow down the slope. Down-slope movement within the snow cover occurs at all times, even on gentle slopes. The speed of movement is slow, generally on the order of a few millimeters per day up to millimeters per hour within new snow on steep slopes. The evidence of these forces is often clearly visible in the bending of trees and damage to structures built on snow-covered slopes. Although the movement is slow, when deep snow pushes against a rigid structure, the forces are significant, and even large buildings can be pushed off their foundations.

Snow deforms in a highly variable fashion. It is generally described as a viscoelastic material. Sometimes it deforms as if it were a liquid (viscous) and at other times it responds more like a solid (elastic). Viscous deformation implies continuous and irreversible flow. Elastic deformation implies that once the force causing the deformation is removed, some small part of the initial deformation is recovered. The elasticity of snow is not so obvious, primarily because the amount of rebound is very small compared with that of more familiar materials.

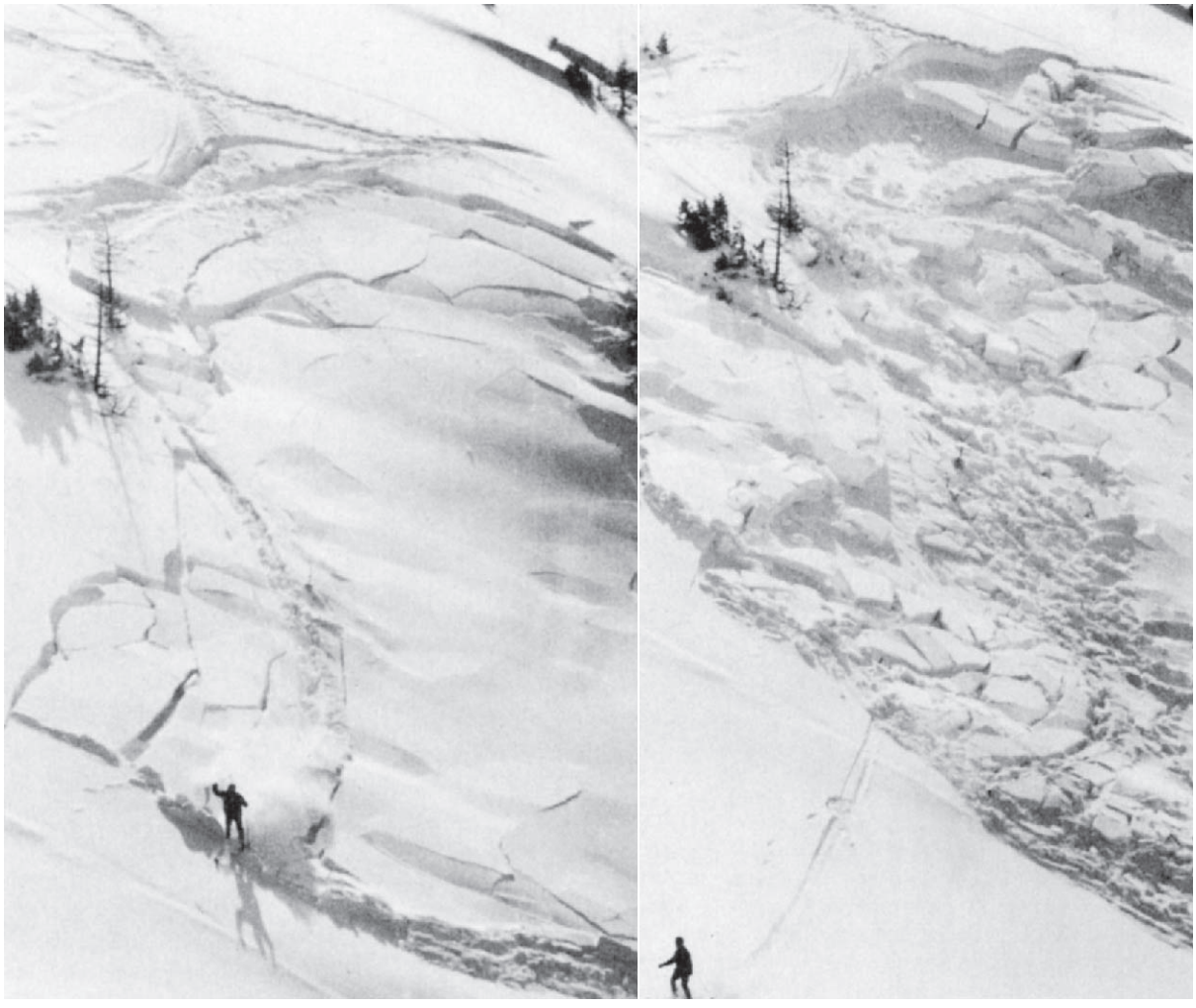
In regard to avalanche formation, it is important to know when snow acts primarily as an elastic material and when it responds more like a viscous substance. These conditions are shown in Figure 2-12. Laboratory experiments have shown that conditions of warm temperatures and slow application of force favor viscous deformation. We see examples of this as snow slowly deforms and bends over the edge of a roof or sags from a tree branch. In such cases, the snow deforms but does not crack or break. In contrast, when temperatures are very cold or when force is applied rapidly, snow reacts like an elastic material. If enough force is applied, it fractures. We think of such a substance as brittle; the release of stored elastic energy causes fractures to move through the material. In the case of snow cover on a steep slope, forces associated with accumulating snow or the weight of a skier may increase until the snow fails. At that point, stored elastic energy is released and is available to drive brittle fractures over great distances through the snow slab.

The slab avalanche provides the best example of elastic deformation in snow cover. Although the deformation cannot actually be seen, evidence of the resultant brittle failure is clearly present in the form of the sharp, linear fracture line and crown face of the slab release (Fig. 2-13). The crown face is almost always perpendicular to the bed surface, evidence that snow has failed in a brittle manner.

To fully understand the slab avalanche condition or the stability of the snow cover, its mechanical state must be considered. Snow is always deforming down-slope, but throughout most of the winter the strength of the snow is sufficient to prevent an avalanche. The snow cover is layered, and some layers are weaker than others. During periods of snowfall, blowing snow, or both, an additional load, or weight, is being applied to the snow in the starting zone, the snow is creeping faster, and these new stresses are beginning to approach the strength of the weakest layers. The weakest layer has a weakest point somewhere within its continuous structure. If the stresses caused by the load of the new snow or the weight of a skier reach the level at which they equal the strength of the weakest point, the snow fails completely at that point (Fig. 2-14). This means that the strength at that point immediately goes to zero. This is analogous to what would happen if someone on a tug-of-war team were to let go of the rope. If the remainder of the team was strong enough to make up for the lost member, not much would change immediately. The same situation exists with the snow cover. If the surrounding snow has sufficient strength to make up for the fact that the strength at the weakest point has now gone to zero, nothing happens beyond perhaps a local movement or settlement in the snow. If, however, the surrounding snow is not capable of doing this, the area of snow next to the initial weak point fails, and then the area next to it, and the chain reaction begins.

As the initial crack forms in the now unstable snow, the elastic energy is released, which in turn drives the crack further, releasing more elastic energy, and so forth. The ability of snow to store elastic energy is essentially what allows large slab avalanches to occur. As long as the snow properties are similar across the avalanche starting zone, the crack will continue to

A



B C

Figure 2-14. Slab avalanche released by a skier. (Photo by R. Ludwig.)

propagate, allowing entire basins, many acres in area, to be set in motion within a few seconds.

AVALANCHE DYNAMICS

The topic of avalanche dynamics includes how avalanches move, how fast they move, and how far and with how much destructive power they travel. The science of avalanche dynamics is not well advanced, although much has been learned in the past few decades. Measured data for avalanche velocity and impact pressure are still lacking. Although any environmental measurement presents its own set of problems, it is clear that opportunities for making measurements inside a moving avalanche are extremely limited. Although avalanche paths exist in a variety of sizes and shapes, they all have three distinct parts with respect to dynamics (Fig. 2-15). In the *starting zone,* usually the steepest part of the path, the avalanche breaks away, accelerates down the slope, and picks up additional snow. From the starting zone, the avalanche proceeds to the *track,* where it remains essentially constant and picks up little or no additional snow as it moves; the average slope angle has become less steep and frequently the snow cover is more stable than in the starting zone. (However, a study by Sovilla and colleagues[19a] from Switzerland in 2000 showed that a significant amount of snow could be entrained into the avalanche from the track.) Small avalanches often stop in the track. After traveling down the track, the avalanche reaches the *runout zone*. Here the avalanche motion ends, either slowly as it decelerates across a gradual slope, such as an alluvial fan, or abruptly as it crashes into the bottom of a gorge or ravine. As a general rule, the slope angle of starting zones is 30 to 45 degrees, of the track it

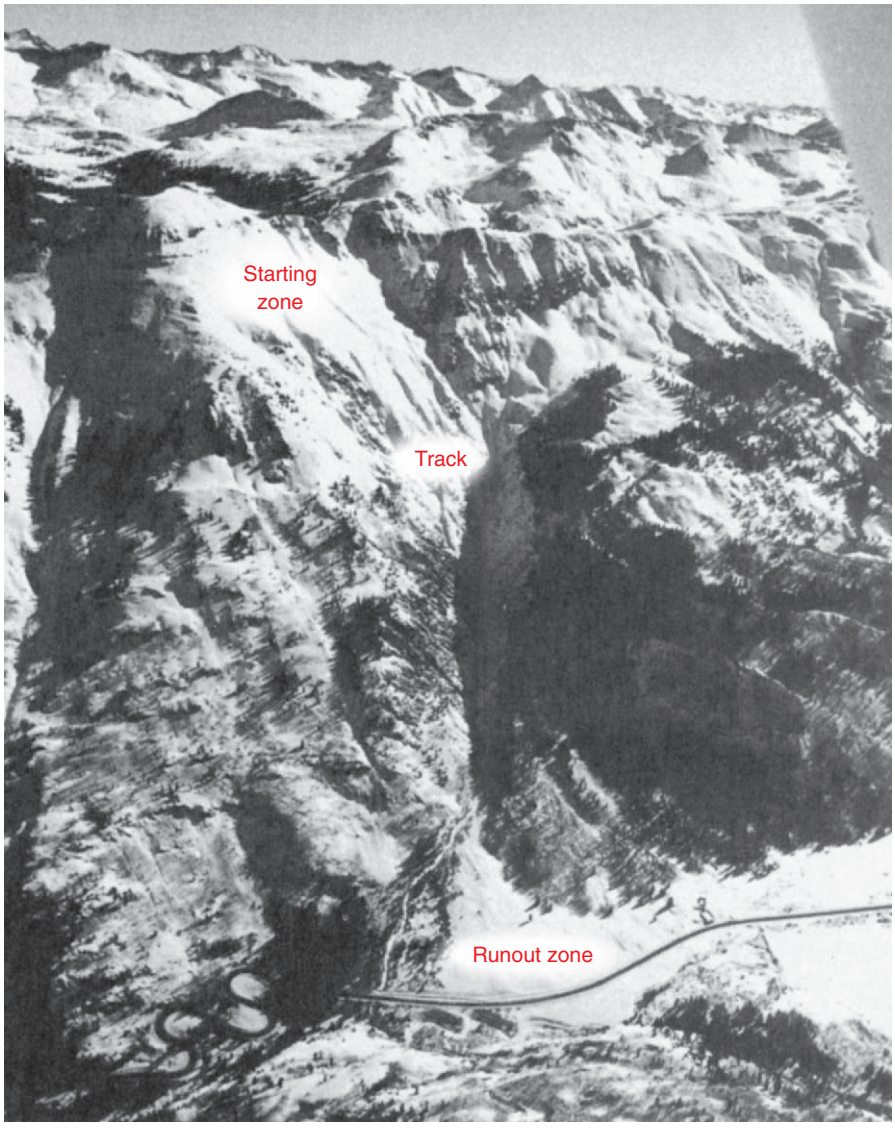


Figure 2-15. The three parts of an avalanche path: starting zone, track, and runout zone. (Photo by B. Armstrong.)

is 20 to 30 degrees, and of the runout zone it is less than 20 degrees.

Few actual measurements of avalanche velocities have been made, but enough data have been obtained to provide some typical values for the various avalanche types. For the highly turbulent dry-powder avalanches, the velocities are commonly in the range of 75 to 100 mph (34 to 45 m/s), with rare examples in the range of 150 to 200 mph (67 to 89 m/s). Such speeds are possible for powder avalanches because large amounts of air in the moving snow greatly reduce the forces resulting from internal friction. As snow in the starting zone becomes dense, wetter, or both, movement becomes less turbulent and a more flowing type of motion reduces typical velocities to the range of 50 to 75 mph (22 to 34 m/s). During spring conditions when the snow contains large amounts of liquid water, speeds may reach only about 25 mph (11 m/s) (Fig. 2-16).

In most cases, the avalanche simply follows a path down the steepest route on the slope while being guided or channeled by terrain features. However, the higher-speed avalanche may deviate from this path. Terrain features, such as the side walls of a gully, which would normally direct the flow of the avalanche around a bend, may be overridden by a high-velocity powder avalanche (Fig. 2-17). The slower-moving avalanches, which travel near the ground, tend to follow terrain features, giving them somewhat predictable courses.

Because avalanches can travel at very high speeds, the resultant impact pressures can be significant. Smaller and medium-size events (impact pressures of 1 to 15 kilopascals [kPa]) have the potential to heavily damage wood-frame structures. Extremely large avalanches (impact pressures of more than 150 kPa) possess the force to uproot mature forests and even destroy structures built of concrete.

Some reports of avalanche damage describe circumstances that cannot be easily explained simply by the impact of large amounts of fast-moving dense snow. Some observers have noted that as an avalanche passed, some buildings actually exploded, perhaps from some form of vacuum created by the fast-moving snow. Other reports indicate that a structure was destroyed by the "air blast" preceding the avalanche because there was no evidence of large amounts of avalanche debris in the area. However, this is more likely to be damage resulting from the powder cloud, which may be composed of only a few inches of settled snow yet it contributes significantly to the total impact force. The presence of snow crystals can increase the air density by a factor of 3 or more. A powder cloud traveling at a moderate dry-avalanche speed of 60 mph (27 m/s) could have the impact force of a 180-mph (80 m/s) wind, well beyond the destructive capacity of a hurricane.

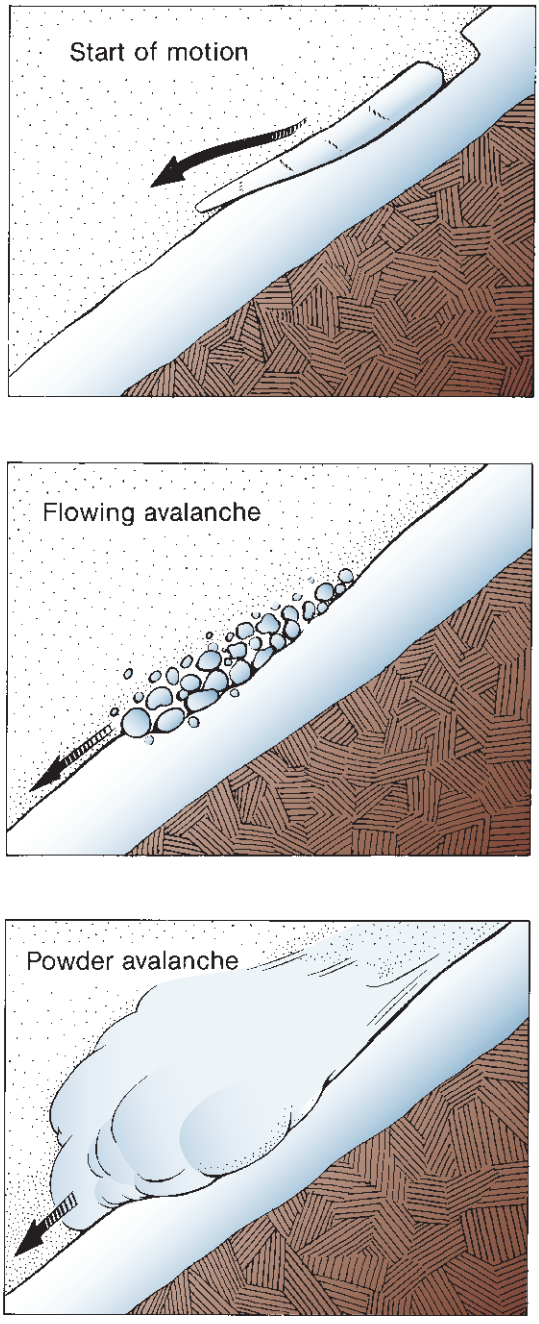


Figure 2-16. A dry-snow avalanche may have a slowing motion and travel near the surface or, with lower density snow and higher velocities, the turbulent dust cloud of the powder avalanche develops.

IDENTIFYING AVALANCHE PATH CHARACTERISTICS

Characteristics such as elevation, slope profiles, and weather determine whether a mountain can produce avalanches. The ingredients of an avalanche, snow and a steep-enough slope, are such that any mountain can produce an avalanche if conditions are exactly right. To be a consistent producer of avalanches, a mountain and its weather must work in harmony.

Elevation

Mountains must be at high-enough latitudes or must be high enough in elevation to build and sustain a winter snow cover before their slopes can become avalanche threats. Temperature drops steadily with elevation. This has the obvious effect of allowing snow to build up deeper and remain longer at higher elevations before melting depletes the snow cover. A less obvious effect of the temperature and elevation relationship on avalanche formation is the demarcation called the treeline.

Figure 2-17. The large powder cloud associated with a fast-moving dry-snow avalanche. (Photo by R. Armstrong.)

This is the level above which the combined effects of low temperature, strong winds, and heavy snowfall prevent tree growth.

The treeline can be quite variable in any mountain range, depending on the microclimates. On a single mountain, treeline is generally higher on south slopes than on north slopes (in the northern hemisphere), because more sunshine leads to warmer average temperatures on southern exposures. Latitudinal variation in the elevation of treeline ranges from sea level in northern Alaska to almost 3658 m (12,000 feet) in the Sierras of southern California and the Rockies of New Mexico.

Mountains that rise above treeline are more likely to produce avalanches. Dense timber anchors the snowpack, so avalanches can seldom start. Below treeline, avalanches can start on slopes having no trees or only scattered trees, a circumstance arising either from natural conditions, such as a streambed or rockslide area, or from human-made conditions, such as clearcuts. Above treeline, avalanches are free to start, and once set in motion, they can easily cut a swath through the trees below. The classic avalanche path is one having a steep bowl above treeline to catch the snow and a track extending below treeline. Avalanches run repeatedly down the track and ravage whatever vegetation grows there, leaving a scar of small or stunted trees that cuts through larger trees on either side.

Slope Angle

In snow that is thoroughly saturated with water, so that a slush mixture is formed, the slope needs only to have a slight tilt to produce an avalanche. For example, a wet-snow avalanche in Japan occurred on a beginner slope at a ski area. The slope was only 10 degrees, but the avalanche was big enough to kill seven skiers. This extreme applies only to a water-saturated snowpack, which behaves more like a liquid than a solid.

A more realistic slope is 22 degrees, the "angle of repose" for granular substances, such as sand and dry, unbonded snow. Round grains will not stack up in a pile having sides much steeper than 22 degrees before gravity rearranges the pile. Dry-snow avalanches have occurred on slopes of 22 to 25 degrees; these are rare because snow grains are seldom round and seldom touch without forming bonds. A useful minimum steepness for producing avalanches is 30 degrees. Avalanches occur with the greatest frequency on slopes of 30 to 45 degrees. These are the angles in which the balance between strength (the bonding of the snow trying to hold it in place) and stress (the force of gravity trying to pull it loose) is most critical. On even steeper slopes, the force of gravity wins; snow continually rolls or sloughs off, preventing buildup of deep snowpacks. Exceptions exist, such as damp snow plastered to a steep slope by strong winds.

Orientation

Avalanches occur on slopes facing every point of the compass. Steep slopes are equally likely to face east or west, north or south. There are factors, however, that cause more avalanches to fall on slopes facing north, northeast, and east than on those facing south through west. These relate to slope orientation with respect to sun and wind. The sun angle in northern hemisphere winters causes south slopes to get much more sunshine and heating than north slopes, which frequently leads to radically different snow covers. North slopes have deeper and colder snow covers, often with a substantial layer of depth hoar near the ground. South slopes usually carry a shallower and warmer snow cover, laced with multiple ice layers formed on warm days between storms. Most ski areas are built on predominantly north-facing slopes to take advantage of deeper and longer-lasting snow cover. At high latitudes, such as in Alaska, the winter sun is so low on the horizon and heat input to south slopes is so small that there are few differences in the snow covers of north and south slopes.

The effect of the prevailing west wind at midlatitudes is important. Storms most often move west to east, and storm winds are most frequently from the western quadrant: southwest, west, or northwest. The effect is to pick up fallen snow and redeposit it on slopes facing away from the wind—that is, onto northeast, east, and southeast slopes. These are the slopes most often overburdened with wind-drifted snow. The net effect of sun and wind is to cause more avalanches on north- through east-facing slopes.

Avalanche Terrain

The frequency with which a path produces avalanches depends on a number of factors, with slope steepness a major one. The easiest way to create high stress is to increase the slope angle; gravity works that much harder to stretch the snow out and rip it from its underpinnings. A slope of 45 degrees produces many more avalanches than one of 30 degrees. However, specific terrain features are also important.

Broad slopes that are curved into a bowl shape and narrow slopes that are confined to a gully efficiently collect snow. Those having a curved horizontal profile, such as a bowl or gully, trap blowing snow coming from several directions; the snow drifts over the top and settles as a deep pillow. On the other hand,

the plane-surfaced slope collects snow efficiently only if it is being blown directly from behind. A side wind scours the slope more than loads it.

The surface conditions of a starting zone often dictate the size and type of avalanche. A particularly rough ground surface, such as a boulder field, does not usually produce avalanches early in the winter, as it takes considerable snowfall to cover the ground anchors. Once most of the rocks are covered, avalanches pull out in sections, the area between two exposed rocks running one time, and the area between two other rocks running another. A smooth rock face or grassy slope provides a surface that is too slick for snow to grip. Therefore, full-depth avalanches are distinctly possible; if the avalanche does not run during the winter, it is likely to run to ground in the spring, once melt water percolates through the snow and lubricates the ground surface.

Vegetation has a mixed effect on avalanche releases. Bushes provide anchoring support until they become totally covered; at that point they may provide weak points in the snow cover, because air circulates well around the bush, providing an ideal habitat for the growth of depth hoar. It is common to see that the fracture line of an avalanche has run from a rock to a tree to a bush, all places of healthy depth hoar growth.

A dense stand of trees can easily provide enough anchors to prevent avalanches. Reforestation of slopes devoid of trees because of logging, fire, or avalanche is an effective means of avalanche control. Scattered trees on a gladed slope offer little if any support to hold snow in place. Isolated trees may do more harm than good by providing concentrated weak points on the slope.

FACTORS CONTRIBUTING TO AVALANCHE FORMATION

The factors that contribute to avalanche release are terrain, weather, and snowpack. Terrain factors are fixed; however, the states of the weather and of the snowpack change daily, even hourly. Precipitation, wind, temperature, snow depth, snow surface, weak layers, and settlement are all factors determining whether an avalanche will occur.

Snowfall

New snowfall is the event that leads to most avalanches: more than 80% of all avalanches fall during or just after a storm. Fresh snowfall adds weight to existing snow cover. If the snow cover is not strong enough to absorb this extra weight, avalanche releases occur. The size of the avalanche is usually related to the amount of new snow. Snowfalls of less than 6 inches (15 cm) seldom produce avalanches. Snows of 6 to 12 inches (15 to 30 cm) usually produce a few small slides, and some of these harm skiers who release them. Snows of 1 to 2 feet (30 to 60 cm) produce avalanches of larger size that present a considerable threat to skiers and pose closure problems for highways and railways. Snows of 2 to 4 feet (60 to 120 cm) are much more dangerous, and snowfalls greater than 4 feet produce major avalanches capable of large-scale destruction. These figures are guidelines based on data and experience and must be considered with other factors to arrive at the true hazard. For example, a snowfall of 10 inches (25 cm) whipped by strong winds may be serious; a fall of 2 feet of feather-light snow in the absence of wind may produce no avalanches.

Snowfall Intensity

The rate at which snowfall accumulates is almost as important as the amount of snow. A snowfall of 3 feet (90 cm) in 1 day is far more hazardous than 3 feet in 3 days. As a viscoelastic material, snow can absorb slow loading by deforming or compressing. Under a rapid load, the snow cannot deform quickly enough and is more likely to crack, which is how slab avalanches begin. A snowfall rate of 1 inch per hour or greater sustained for 10 hours or more is generally a red flag indicating danger. The danger worsens if snowfall is accompanied by wind.

Rain

Light rain falling on a cold snowpack invariably freezes into an ice crust, which adds strength to the snow cover. At a later time, the smooth crust could become a sliding layer beneath the new fall of snow. Heavy rain (usually an inch or more) greatly weakens the snow cover. First, it adds weight. An inch of rain is the equivalent in weight to 10 to 12 inches (25 to 30 cm) of snow. Second, it adds no internal strength of its own (in the form of a skeleton of ice, as new snow would), while it dissolves bonds between snow grains as it percolates through the top snow layers, reducing strength even further.

New Snow Density and Crystal Type

A layer of fresh snow contains only a small amount of solid material (ice); the large majority of the volume is occupied by air. It is convenient to refer to snow density as a percentage of the volume occupied by ice. New snow densities usually range from 7% to 12%. In the high elevations of Colorado, 7% is an average value; in the more maritime climates of the Sierras and Cascades, 12% is a typical value. Density becomes an important factor in avalanche formation when it varies from average values.

Wet snowfalls or falls of heavily rimed crystals, such as graupel, may have densities of 20% or greater. (Graupel is a snowflake that has been transformed into a pellet of soft ice because of riming inside a cloud.) A layer of heavier-than-normal snow presents a danger because of excess weight. Snowfall that is much lighter than normal, 2% to 4% for example, can also present a dangerous situation. If the low-density layer quickly becomes buried by snowfall of normal or high density, a weak layer has been introduced into the snowpack. By virtue of low density, the weak layer has marginal ability to withstand the weight of layers above, making it susceptible to collapse. Storms that begin with low temperatures but then warm up produce a layer of weak snow beneath a stronger, heavier layer.

Density is closely linked to crystal type. Snowfalls consisting of graupel, fine needles, and columns can accumulate at high densities. Snowfalls of plates, stellars, and dendritic forms account for most of the lower densities.

Wind Speed and Direction

Wind drives fallen snow into drifts and cornices from which avalanches begin. Winds pick up snow from exposed, windward slopes and drive it onto adjacent, leeward slopes, where it is deposited into sheltered hollows and gullies.

A speed of 15 mph (7 m/s) is sufficient to pick up freshly fallen snow. Higher speeds are required to dislodge older snow. Speeds of 20 to 50 mph (9 to 22 m/s) are the most efficient in transporting snow into avalanche starting zones. Speeds greater than 50 mph can create spectacular banners of snow streaming from high peaks, but much of this snow is lost to evaporation in the

air or is deposited far down the slope away from the avalanche starting zone.

Winds play a dual role in increasing avalanche potential. First, wind scours snow from a large area (of a windward slope) and deposits it in a smaller area (of a starting zone). Wind can thus turn a 1-foot snowfall into a 3-foot drift in a starting zone. The rate at which blowing snow collects in bowls and gullies can be impressive. In one test at Berthoud Pass, Colorado, the wind deposited snow in a gully at a rate of 18 inches (45 cm) per hour. Another wind effect is that blowing snow is denser after deposit than before. This is because snow grains are subjected to harsh treatment in their travels; each collision with another grain knocks off arms and sharp angles, reducing size and allowing the pieces to settle into a denser layer. The net result of wind is to fill avalanche starting zones with more and heavier snow than if the wind had not blown.

Temperature

The role of temperature in snow metamorphism is played over a period of days, weeks, and even months. The influence of temperature on the mechanical state of the snow cover is more acute, with changes occurring in minutes to hours. The actual effect of temperature is not always easy to interpret: whereas an increase in temperature may contribute to stabilization of the snow cover in one situation, it might at another time lead to avalanche activity.

In several situations, an increase in temperature clearly produces an increase in avalanche potential. In general, these include a rise in temperature during a storm or immediately after a storm, or a prolonged period of warm, fair weather such as occurs with spring conditions. In the first example, the temperature at the beginning of a snowfall may be well below freezing, but as the storm progresses, the temperature increases. As a result, the initial layers of new snow are light, fluffy, low density, and relatively low in strength, whereas the later layers are warmer, denser, and stiffer. Thus, the essential ingredients for a slab avalanche are provided within the new snow layers of the storm: a cohesive slab resting on a weak layer. If the temperature continues to rise, the falling snow turns to rain, a situation not uncommon in lower-elevation, coastal mountain ranges. Once this happens, avalanches are almost certain because as the rain falls, additional weight is added to the avalanche slope, but no additional strength is provided as it is whenever a layer of snow accumulates.

The second example may occur after an overnight snowstorm that does not produce an avalanche on the slope of interest. By morning, the precipitation stops and clear skies allow the morning sun to shine directly on the slopes. The sun rapidly warms the cold, low-density new snow, which begins to deform and creep down-slope. The new snow layer settles, becomes denser, and gains strength. At the same time, it is stretched downhill and some of the bonds between the grains are pulled apart; thus, the snow layer becomes weaker. If more bonds are broken by stretching than are formed by settlement, there is not enough strength to hold the snow on the slope and an avalanche occurs.

In these first two examples, the complete snow cover generally remains at temperatures below freezing. A third example occurs when a substantial amount of the winter's snow cover is warmed to the melting point. During winter, sun angles are low, days are short, and air temperatures are cold enough that the small amount of heat gained by the snow cover during the day is lost during the long cold night. As spring approaches, this pattern changes, and eventually enough heat is available at the snow surface during the day to cause some melt. This melt layer refreezes again that night, but the next day more heat may be available, so that eventually a substantial amount of melting occurs and melt water begins to move down through the snow cover. As melt water percolates slowly downward, it melts the bonds that attach the snow grains, and the strength of the layers decreases. At first the near-surface layers are affected, with the midday melt reaching only as far as the uppermost few inches, with little or no increase in avalanche hazard. If warm weather continues, the melt layer becomes thicker and the potential for wet snow avalanches increases. The conditions most favorable for wet slab avalanches occur when the snow structure provides the necessary layering. When melt water encounters an ice layer or impermeable crust, or in some cases a layer of weak depth hoar, wet slab avalanches are likely to occur.

Depth of Snow Cover

Of the snowpack factors contributing to avalanche formation, depth of snow cover is the most basic. When the early-winter snowpack covers natural anchors, such as rocks and bushes, the start of the avalanche season is at hand. North-facing slopes are usually covered before other slopes. A scan of the terrain usually suffices to weigh this clue, but another method can be used to determine the time of the first significant avalanches. Long-term studies show a relationship between snow depth at a study site and avalanche activity. For example, along Red Mountain Pass, Colorado, it is unlikely that an avalanche large enough to reach the highway will run until close to 3 feet (90 cm) of snow covers the ground at the University of Colorado's snow study site. At Alta, Utah, once 52 inches (130 cm) of snowpack has built up, the first avalanche to cover the road leading from Salt Lake City can be expected.

Nature of the Snow Surface

How well new snow bonds to the old snow surface is a key factor in determining whether an avalanche will release within the layer of new snow or deeper in the snowpack. A poor bond, usually new snow resting on a smooth, cold surface with snowfalls of 1 foot (30 cm) or more, almost always produces a new-snow avalanche. A strong bond, usually onto a warm, soft, or rough surface, may produce nothing at all, or if weaknesses lie at deeper layers of the snow cover, a large snowfall will cause avalanches to pull out older layers of snow in addition to the new snow layer. These avalanches have more potential for destruction. A cold, hard snow surface offers little grip to fresh, cold snow. Ice crusts are commonly observed to be avalanche-sliding surfaces. The crust could be a sun crust, a rain crust, or a hardened layer of firm snow that has survived the summer. Firm layers are especially dangerous in early winter when first snows fall.

Weak Layers

Any layer susceptible to collapse or failure because of the weight of the overburden is a weak link. Of the snowpack contributory factors, this is the most important, because a weak layer is essential to every avalanche. The weak layer releases along what is called the failure plane, sliding surface, or bed surface.

One common weak layer is an old snow surface that offers a poor bond for new snow. Another weak layer that forms on

the snow surface is hoar frost, or surface hoar (see Physical Properties, earlier). On clear, calm nights, it forms a layer of feathery, sparkling flakes that grow on the snow surface. The layer can be a major contributor to avalanche formation when buried by a snowfall. Many avalanches have been known to release on a buried layer of surface hoar, sometimes a layer more than 1 month old and 6 feet (180 cm) or more below the surface.

A weak layer that is almost always found in the snowpacks that blanket the Rocky Mountains and occasionally the Cascades and Sierra Nevadas is temperature-gradient snow, or depth hoar. The way to decide whether a temperature-gradient layer is near its collapse point is to test the strength of the overlying layers and the support provided around the edges of the slope. This is no easy task. One method is to try jumping on your skis while standing on a shallow slope. Collapse is a good indication that similar snow cover on a steeper slope will produce an avalanche. Often, skiers and climbers cause inadvertent collapses while skiing or walking on a depth hoar–riddled snowpack. The resulting "whoomf" sound is a warning of weak snow below.

Finally, a weak layer can be created within the snow cover when surface melting or rain causes water to percolate into the snow and then fan out on an impermeable layer, thereby lubricating that layer and destroying its shear strength. Combining the contributory factors on a day-by-day basis is the avalanche forecaster's art. Every avalanche must have a weak layer to release on, so knowledge of snow stratigraphy, or layering, and what sort of applied load will cause a layer to fail is the essence of forecasting.

SAFE TRAVEL IN AVALANCHE TERRAIN

The first major decision often faced in backcountry situations is whether to avoid or confront a potential avalanche hazard. A group touring with no particular goal in mind will probably not challenge avalanches. For this group, education to recognize and avoid avalanche terrain is sufficient. In the other extreme, mountaineering expeditions that have specific goals and are willing to wait out dangerous periods or take severe risks to succeed need considerably more information. Traveling safely in avalanche terrain requires special preparations, including education and possession of safety and rescue equipment. The group should have the skills required to anticipate and react to an avalanche.

Identifying Avalanche Terrain

Because most avalanches release on slopes of 30 to 45 degrees, judging angle is a prime skill for recognizing potential avalanche areas. An inclinometer can be used to measure slope angles. Some compasses are also equipped for this purpose; a second needle and a graduated scale in degrees can be used to measure slope angles. A ski pole may be used to judge approximate slope angle. When dangled by its strap, the pole becomes a plumb line from which the slope angle can be "eyeballed."

Evidence of fresh avalanche activity—the presence of fracture lines and the rubble of avalanche snow on the slope or at the bottom—identifies avalanche slopes. Other clues are swaths of missing trees or trees that are bent downhill or damaged, especially with the uphill branches removed. Above treeline, steep bowls and gullies are almost always capable of producing avalanches.

Route Finding

Good route-finding techniques are necessary for safe travel in avalanche terrain (Fig. 2-18). The object of a good route in avalanche country is more than avoiding avalanches. It should also be efficient and take into account the abilities and desires of the group to choose a route that is not overly technical, tiresome, or time consuming. The safest way to avoid avalanches is to travel above or below and well away from them. When taking the high route, the traveler should choose a ridgeline that is above the avalanche starting zones. It is safest to travel the windward side of the ridge. The snow cover is usually thinner and windpacked, with rocks sticking through—not the most pleasant skiing, but safe. Cornice collapses present a very real hazard; they should be avoided by staying on the roughened snow more to windward.

Skiers taking the low route in the valley should not linger in the runouts of avalanche paths. Even though it is unlikely that a skier traveling along the valley could trigger an avalanche high up on the slope, the skier should not boost the odds of getting caught in an avalanche released by natural forces far above. Slopes of 30 degrees or more should be avoided. By climbing, descending, and traversing only in gentle terrain, avalanche terrain can be avoided.

Stability Evaluation Tests

Skiers can perform several tests of stability. On a small slope that is not too steep (and therefore will not avalanche), the skier can try a *ski test* by skiing along a shallow traverse and then setting the ski edges in a hard check. Any cracks or settlement noises indicate that the same slope, if steeper, would have probably avalanched, and on the steeper slope it would have taken less weight or jolt to cause the avalanche.

Another test is to push a *ski pole* into the snow, handle end first. This helps to feel the major layering of the snowpack. For example, the skier may feel the layer of new snow, midpack stronger layers, and depth hoar layers, if the pole is long enough. Hard-snow layers and ice lenses resist penetration altogether. This test reveals only the gross layers; thin weak layers, such as buried surface hoar or a poor bond between any two layers, cannot be detected. Thus, the ski pole test has limited value.

A much better way to directly observe and test snowpack layers is to dig a hasty *snowpit*. (This is an excellent use of the shovel that, in the next section, we recommend the skier carry.) In a spot as near as possible to a suspected avalanche slope without putting the traveler at risk, a pit 4 to 5 feet (120 to 150 cm) deep and 3 feet (90 cm) wide should be dug. With the shovel, the uphill wall is shaved until it is smooth and vertical. Now the layers of snow can be observed and felt. The tester can see where the new snow touches the layer beneath, poke the pit wall with a finger to test hardness, and brush the pit wall with a paintbrush to see which layers are soft and fall away and which are hard and stay in place after being brushed. By grabbing a handful of depth hoar, the skier can see how large the grains are and how poorly they stick together.

The *shovel shear test* gauges the shear strength between layers and thus locates weak layers. First a column of snow is isolated from the vertical pit wall. Both sides and the back of the column are cut with the shovel or a ski, so that the column is free

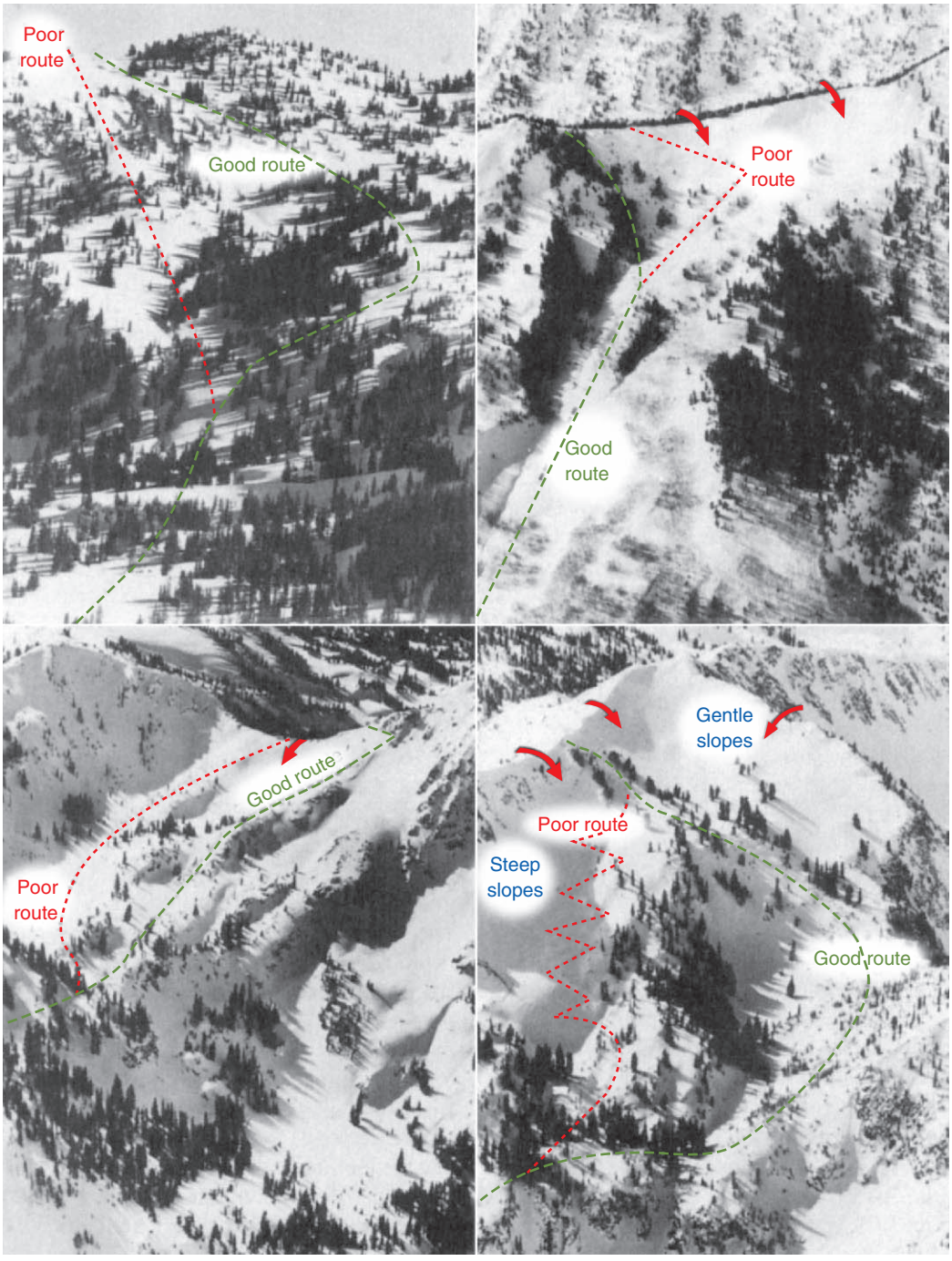


Figure 2-18. Four ski-touring areas showing the safer routes *(green dashed lines)* and the more hazardous routes *(red dotted lines)*. *Arrows* indicate areas of wind loading. (From USDA Forest Service: Avalanche Handbook, Agricultural Handbook 489, with permission. Photo by Alexis Kelner.)

A B

Figure 2-19. **A,** A small-size Avalanche ABS backpack with deployed airbags. The airbags are stowed in outside pockets of the backpack. **B,** Integrated into a backpack, the Avalanche ABS is deployed by pulling the white "T" handle. (Courtesy Peter Aschauer, GmbH.)

standing. The dimensions are a shovel's width on all sides. The tester inserts the shovel blade at the back of the column and gently pulls forward on the handle. An unstable slab will shear loose on the weak layer, making a clean break; the poorer the bond, the easier the shear. A five-point scale is used to rate the shear: "very easy" if it breaks as the column is being cut or the shovel is being inserted; "easy" if a gentle pull on the shovel does the job; "moderate" if a slightly stronger shovel-pry is required; "hard" if a solid tug is required; "very hard" if a major effort is needed to break the snow. Generally, "very easy" and "easy" shears indicate unconditionally unstable snow, "moderate" means conditionally unstable, and "hard" and "very hard" mean stable.

The value of the shovel shear test is that it can find thin weak layers undetectable by any other method. Its shortcoming is that it is not a true test of stability, as it does not indicate the amount of weight required to cause shear failure.

A test that does a better job of indicating actual stability is the *Rutschblock, or shear block, test.* This test is calibrated to the skier's weight and the stress he or she would put on the snow. Again, a snowpit is dug with a vertical uphill wall, but the pit must be about 8 feet (240 cm) wide. By cutting into the pit wall, the skier isolates a block of snow that is about 7 feet (210 cm) wide (a ski length) and goes back 4 feet (120 cm) (a ski pole length) into the pit wall. Both sides and the back are cut with a shovel or ski so that the block is free standing. Wearing skis, the skier climbs around and well uphill from the isolated block and carefully approaches it from above. With skis across the fall line, the skier gently steps onto the block, first with the downhill ski and then the uphill ski, so that he or she is standing on the isolated block of snow. If the slab of snow has not yet failed, gently flexing the knees applies a little more pressure. Next some gentle jumps are tried. The stress should be increased by jumping harder until the block eventually shears loose or crumbles apart.

The results are interpreted as "extremely unstable" if the block fails while the skier is cutting it, approaching it from above, or merely standing on it; "unstable" if it fails with a knee flex or one gentle jump; "moderately stable" if it fails after repeated jumps; and "very stable" if it never fails but merely crumbles. These are objective results that help answer the bigger question—will it slide?—and help the mountain traveler decide how much risk to take.

Avalanche Rescue Equipment

Shovel

The first piece of safety equipment the skier or climber should own is a shovel. It can be used to dig snowpits for stability evaluation, and snow caves for overnight shelter. A shovel is also needed for digging in avalanche debris, as such snow is far too hard for digging with the hands or skis.

The shovel should be sturdy and strong enough to dig in avalanche debris, yet light and small enough to fit into a pack. There is no excuse for not carrying a shovel. Shovels are made of aluminum or high-strength plastic and can be collapsible. Many good types are available in mountaineering stores.

Probe

Several pieces of equipment are designed specifically for finding buried avalanche victims. The first is a collapsible probe pole. Organized rescue teams keep rigid poles in 10- or 12-foot (3 to 4 m) lengths as part of their rescue caches. The recreationist can buy probe poles of tubular aluminum or carbon fiber that come in 18-inch (45-cm) sections that fit together to make a full-length probe. Ski poles with removable grips and baskets can be screwed together to make an avalanche probe. Survivors of an accident use probes to search for buried victims.

Avalanche Rescue Beacon

Avalanche rescue beacons, or transceivers, have become the most-used personal rescue device worldwide. When used properly, they are a fast and effective way to locate buried avalanche victims. In the United States, these have become standard issue

for ski area patrollers involved in avalanche work and for helicopter-skiing guides and clients. They are also commonly used by highway departments, search and rescue teams, and an increasing number of winter recreationists. Since beacons were introduced in the United States, they have saved at least 55 lives. Beacons save at least three or four lives per winter.

All transceivers act as transmitters that emit a signal on a world-standard frequency of 457 kHz. A buried victim's unit emits this signal, and the rescuers' units receive the signal. The signal carries 30 to 46 m (100 to 150 feet) and, once picked up, guides searchers specifically to the buried unit.

Beacon technology is evolving rapidly and the beacons on the market are improving. Two types of beacon have emerged: analog, which processes the signal in the traditional way to allow for a stronger (louder) signal as the receiving beacon approaches the sending beacon, and digital, which uses a computer chip to process the signal, displaying a digital readout of the distance and the general direction to the buried unit. Transceivers are not radio devices but are audio-magnetic-induction devices, so the directional arrows point only along a flux line of the sending unit's magnetic field. Likewise, the displayed distance is the distance along a flux line rather than a direct distance to the sending unit.

Both the analog and the digital types operate on the same frequency and therefore are compatible with one another. However, slightly different search techniques may be necessary to use each type most efficiently, and special training and practice are required before the user attains proficiency. The main brands available in the United States are ARVA, Barryvox, Ortovox, Pieps, Tracker, and SOS.

Merely possessing a beacon does not ensure its lifesaving capability. Frequent practice is required to master a beacon-guided search, which may not be straightforward. Skilled practitioners can find a buried unit in less than 5 minutes once they pick up the signal. Because speed is of the essence in avalanche rescue, beacons are certainly lifesavers. The best-proven rescue equipment is a beacon for a quick find, a collapsible probe pole to confirm and pinpoint the spot, and a shovel for a quick recovery (Box 2-1).

Avalanche Airbag

In 1995, a new rescue device made in Germany was introduced in Europe. The ABS Avalanche Airbag System (Fig. 2-19) was originally designed specifically for guides and ski patrollers but today can be used by anyone venturing into avalanche terrain. Since 1995, the system has undergone continued and significant improvements. The airbag works because of *inverse segregation*. An avalanche in motion is composed of many different-sized particles of snow. Because of gravity, granular flows segregate, with smaller particles sinking to the lower portion of the flow and larger particles rising to the surface. The process of inverse segregation depends primarily on the relative sizes of the particles in a granular flow. A person is already a large particle and the airbag makes the user an even larger particle, making the separation effect even greater. The airbag is integrated into a special backpack, and the user deploys it by pulling a ripcord-like handle. This releases a cartridge of nitrogen gas that escapes at high velocity and draws in outside air through jets that inflate two 75-L airbags in 2 seconds.

By 2004, there had been about 70 documented uses of the ABS Avalanche Airbag System, with three deaths. In one case the airbag failed to work, and in the second case the user did not or could not deploy the airbag. The third incident involved a user who successfully deployed the airbag, only to be completely buried by a second avalanche moments later. Despite these failures, Brugger and Falk in an expanded study found that the ABS reduces the likelihood of burial from 39% to 16.2%.[5]

The ABS system is the most effective piece of equipment for saving lives in avalanche terrain. Brugger and Falk showed that it reduces mortality significantly, from 23% to 2.5% compared with the 75.9% to 66.2% reduction for avalanche rescue transceivers.[5] This dramatic reduction in mortality occurs because the ABS system prevents burial, and very few partly buried or not buried victims (about 4% to 5%) die in avalanches.

Commercial sales of ABS system in the United States have been delayed because of problems obtaining certification for the nitrogen-gas cartridges. The German company hopes to have U.S. Department of Transportation approval in 2007.

AvaLung

In 1996, Dr. Thomas Crowley received a patent for an emergency breathing device to extract air from the snow surrounding a buried avalanche victim. The AvaLung functions as an artificial air pocket whose goal is to prolong survival time for the buried victim. Black Diamond Equipment, Ltd. (Salt Lake City, Utah) secured distribution rights in 2000 and redesigned the device in 2002. Called the AvaLung 2, it is worn outside the clothing like a bandoleer. If buried, the victim can breathe through a mouthpiece and inhale air from the surrounding snow. The carbon dioxide (CO_2)-rich exhaled air is redirected into another area of the snow, reducing re-inhalation of CO_2 (see Avalanche Victim Physiology and Medical Treatment after Rescue, later).

The AvaLung system has been proven in numerous simulated burials, allowing subjects to breathe for 1 hour in tightly packed snow, including dense, wet snow. As of 2004, there were three known uses in avalanches. In two cases, the burials were very short, but in a third case a helicopter skier survived a 4-foot (120-cm) burial of 35 to 45 minutes.

The ABS Avalanche Airbag and the AvaLung are designed to help avalanche victims and are an adjunct to the basic companion rescue equipment of transceiver, probe, and shovel. These devices should never be used to justify taking additional risks. Because surviving any avalanche is uncertain, this equipment should never replace good judgment.

Crossing Avalanche Slopes

Travel through avalanche country always involves risk, but certain travel techniques can minimize that risk. Proper travel techniques might not prevent an avalanche release but can improve the odds of surviving. The timing of a trip has a lot to do with safety. Most avalanches occur during and just after storms. Waiting a full day after a storm has ended can allow the snowpack to react to the new snow load and gain strength.

Before crossing a potential avalanche slope, the skier or hiker should get personal gear in order by tightening up clothing, zipping up zippers, and putting on hat, gloves, and goggles. Clothing should be padded and insulating. If a heavy mountaineering pack is carried, the straps should be loosened or slung over one shoulder only, so that the pack can be easily discarded if the person is knocked down. A heavy pack makes a person

Box 2-1. Avalanche Transceiver Search

INITIAL SEARCH

1. Have everyone switch their transceivers to "receive" and turn the volume to "high."
2. If enough people are available, post a lookout to warn others of further slides.
3. Should a second slide occur, have rescuers immediately switch their transceivers to "transmit."
4. Have rescuers space themselves no more than 30 m (100 feet) apart and walk abreast along the slope.
5. For a single rescuer searching within a wide path, zigzag across the rescue zone. Limit the distance between crossings to 30 m (100 feet).
6. For multiple victims, when a signal is picked up, have one or two rescuers continue to focus on that victim while the remainder of the group carries out the search for additional victims.
7. For a single victim, when a signal is picked up, have one or two rescuers continue to locate the victim while the remainder of the group prepares shovels, probes, and medical supplies for the rescue.

LOCATING THE VICTIM

With practice, the induction line search is more efficient than the conventional grid search for getting close to the sending beacon, but the conventional grid search is still necessary to pinpoint the buried victim. The induction line search is very similar to the flux line search used by digital transceivers. Users should always study the owner's manual to learn the best techniques for the specific brand of avalanche rescue beacon.

Induction Line Search (Preferred Method)

When an induction line search is used, the rescuer may initially follow a line that leads away from the victim (Fig. 2-30). Remember to lower transceiver volume if it is too loud, because the ear detects signal strength variations better at lower volume settings.

1. After picking up a signal during the initial search, hold the transceiver horizontally (parallel with the ground), with the front of the transceiver pointing forward (see Figure 2-30A).
2. Holding the transceiver in this position, turn until the signal is maximal (maximum volume), then walk five to seven steps (about 5 m), stop, and turn again to locate the maximum signal (see Figure 2-30B). When locating the maximum signal, do not turn yourself (or the transceiver) more than 90 degrees in either direction. If you rotate more than 90 degrees to locate the maximum signal, you will become turned around and follow the induction line in the reverse direction.
3. Walk another five steps, as just described, and then stop and orient the transceiver toward the maximum signal. Reduce the volume.
4. Continue repeating these steps. You should be walking in a curved path along the "induction line" toward the victim (see Figure 2-30C).
5. When the signal is loud at minimum volume setting, you should be very close to the victim and can begin the pinpoint search (see next).

Grid Search

1. When a signal is picked up, stand and rotate the transceiver, which is held horizontally (parallel with the ground), to obtain the maximum signal (loudest volume). Maintain the transceiver in this orientation during the remainder of the search.
2. Turn the volume control down until you can just hear the signal. Walk in a straight line, down the fall line from where the signal was first detected. (If the signal fades immediately, walk up the fall line.) If you are headed in the right direction, the signal will increase; turn the volume control down until the signal fades. Take an extra couple of steps to be sure the signal truly fades. If the signal increases, continue until it fades.
3. When the signal fades, mark the point and turn 180 degrees and walk back toward the starting position. The signal will increase in volume and then fade again; take two additional steps to confirm the fade. Walk back to the middle of the two fade points, this spot may or may not be the point of loudest volume/maximum signal. If you experience two maximum signals, go to the midpoint between the two maximums.
4. At this point, turn 90 degrees in one direction or the other. From that position, reorient the transceiver (held parallel with the ground) to locate the maximum signal. After orienting the transceiver to the maximum signal, reduce the volume, and begin walking forward. If the signal fades, turn around 180 degrees and begin walking again.
5. As the signal volume increases, repeat steps 3 and 4 until you have reached the lowest volume control setting on the transceiver. (Be sure to always take an extra step or two to confirm the fade point.) This time, when you return to the middle of the fade points, you should be very close to the buried victim and can now begin pinpointing him or her.
   a. While stationary, orient the transceiver to receive the maximum signal (loudest volume). At this point, turn the volume control all the way down.
   b. With the transceiver just above the surface of the snow, continue doing the grid search pattern two to four more times. Always sweep the transceiver a couple of feet beyond the fade point to confirm the fade point.
   c. Find the signal position halfway between fade points (i.e., at the loudest signal). At this point, you should be very close to the victim's position and can begin to mechanically probe. Speed is essential. With practice, the transceiver will be accurate to less than one fourth of the burial depth. A 4-foot burial should result in about a 1-foot square at the surface.
   d. Pinpointing with a digital transceiver: Depending on the brand, pinpointing with a digital transceiver will use a slight variation or combination of the induction line and grid techniques. Be sure to study the owner's manual.

top-heavy, making it difficult to swim with the avalanche. The skier should remove pole wrist straps and ski runaway straps, because poles and skis attached to the victim hinder swimming motions and only serve to drag the victim under. Finally, a person wearing a rescue beacon should be certain it is transmitting.

If possible, the person should cross low on the slope, near the bottom or in the runout zone. Crossing rarely causes a release in the starting zone far above. The greater risk is getting hit by an untimely natural release from above. If crossing high without reaching the safety of the ridge is necessary, the starting zone should be traversed as high as possible and close to rocks, cliff, or cornice. Should the slope fracture, most of the sliding snow will be below and the chance of staying on the surface of the moving avalanche will be better. Invariably, the person highest on the slope runs the least risk of being buried.

A person who must climb or descend an avalanche path should keep close to its sides. Should the slope fracture, escaping to the side improves the chance of surviving. Only one person at a time should cross, climb, or descend an avalanche slope; all other members should watch from a safe location. Two commonsense principles lie behind this advice. First, only one group member is exposed to the hazard, leaving the others available as rescuers. Second, less weight is put on the snow. All persons should traverse in the same track. This not only reduces the amount of work required but also disturbs less snow, which lowers the chance of avalanche release.

Skiers and climbers should never drop their guard on an avalanche slope. They should not stop in the middle of a slope, but only at the edge or beneath a point of protection, such as a rock outcropping. It is possible for the second, third, or even tenth person traversing or skiing down a slope to trigger the avalanche. Trouble should always be anticipated, and an escape route, such as getting to the side or grabbing a tree, should be kept in mind.

Avalanche Rescue

Self Rescue

Escaping to the Side. The moment the snow begins to move around the person, there is a split second in which to make a decision or make a move. The person should shout to alert companions and then close the mouth and breathe through the nose to prevent inhalation of a mouthful of snow. Whether on foot, skis, or snowmobile, the person should first try to escape to the side of the avalanche or try to grab onto a tree. Staying on one's feet or snow machine gives some control and keeps the head up. Escaping to the side gets the person out altogether or to a place where the forces and speeds are less. Turning skis or the snow machine downhill in an effort to outrun the avalanche is a bad move: the avalanche invariably overtakes its victims.

Swimming. For years, the command to swim if caught in an avalanche was dogma. However, in recent years the effectiveness of swimming is being called into question. It is the process of inverse segregation (see Avalanche Airbag, earlier) rather than swimming that keeps the victim on or near the surface. The most important thing to do when caught and being tumbled is to get a hand or both hands in front of the face to create an air pocket. Cumbersome or heavy gear should be discarded. Ski poles should be tossed away; with luck, the avalanche will strip away the skis. Skiers should try to discard gear, but snowmobile riders should try to stay on their snow machine. Once off their machine, riders are twice as likely to be buried as are their machines. Human-triggered avalanches tend to stop abruptly and almost instantly lock the person in place. There is no further chance to move hands to the face to create an air pocket, and without an air pocket, very few buried victims survive.

Reaching the Surface. Anyone caught in an avalanche should fight for his or her life. Creating an air pocket is the key to survival, but some victims, sensing themselves to be near the surface, have thrust out a hand or a foot. Any clue on the surface that gives the rescuers something to see greatly improves the odds of survival.

Companion Rescue

Marking the Last-Seen Area. A companion or eyewitness to an accident needs to act quickly and positively. The rescuer's actions over the next several minutes may mean the difference between life and death for the victim. First, the victim's last-seen spot should be fixed and marked with a piece of equipment, clothing, a tree branch, or anything that can be seen from a distance down-slope. It is usually safe to move out onto the bed surface of an avalanche that has recently run. It is dangerous when the fracture line has broken at midslope, leaving a large mass of snow still hanging above the fracture.

Searching for Clues. The fall line should be searched below the last-seen area for any clues of the victim. The snow should be scuffed by kicking and turning over loose chunks to look for anything that might be attached to the victim or that will reveal the victim's trajectory and thus narrow the search area. Shallow probes should be made into likely burial spots with an avalanche probe, ski, ski pole, or tree limb. Likely spots are the uphill sides of trees and rocks, and benches or bends in the slope where snow avalanche debris piles up. The toe of the debris should be searched thoroughly; many victims are found in this area.

Rescue Beacons. If the group was using beacons, all companions must immediately switch their units to receive mode. While making the fast scuff-search for visual clues, companions should at the same time search the debris, listening for the beeping sound from the buried beacon. When they detect a signal, companions must narrow the search area quickly. If skilled with a transceiver, companions can pinpoint the burial site in a few minutes.

Going for Help. A difficult question in rescues is when to seek outside help. If the accident occurs in or near a ski area and there are several companions, one person can be sent to notify the ski patrol immediately. If only one companion is present, the correct choice is harder. The best advice is to search the surface quickly but thoroughly for clues before leaving to notify the patrol. If a patrol phone is close, the companion should notify the patrol and wait to accompany the patroller back to the avalanche.

If the avalanche occurs in the backcountry far from any organized rescue team, all companions should remain at the site. The guiding principle in backcountry rescues is that companions search until they cannot or should not continue. When

deciding when to stop searching, the safety of the companions must be weighed against the decreasing survival chances of the buried victim.

Organized Rescue

Three-Stage Rescue. A full-scale avalanche search-and-rescue operation is divided into three stages. The goal of stage I is to find and extricate the buried victim or victims. Teams of rescuers dispatched to the avalanche are known as columns. The first team—known as the hasty search team—should consist of skilled and swift-traveling rescuers competent in not only avalanche rescue but also route finding and hazard evaluation. Basic rescue tools for stage I are probes, shovels, avalanche rescue dogs, the RECCO system (see later), and basic first-aid equipment. The hasty team performs the initial search, looking for clues with hopes of making a quick find. If unlucky, the team determines the most likely burial areas. The person reporting the avalanche should meet rescuers at the accident site, or the reporting person should be returned to the same vantage point from where they witnessed the accident to best guide rescuers to locate last-seen areas. Arriving rescuers continue the hasty search until sufficient clues can steer rescuers to positioning probe lines.

The goal of stage II is to provide emergency medical care and evacuation. This stage consists of one or two small teams to transport resuscitation and medical equipment along with sleeping bags and rescue sleds to the site. Ideally, stage II should begin 10 to 15 minutes after the start of stage I to ensure that necessary medical and evacuation equipment reach the site. Stage I often continues long after stage II has been deployed.

Stage III provides support for stages I and II when the rescue is prolonged. This support may include additional rescuers to take over for cold and tired searchers, hot food and drink, tents, warm clothing, and perhaps lights for nighttime searching.

Probing. Probing avalanche debris is a simple but slow method of searching for buried victims. For more than 40 years, the traditional probe line used by rescue teams was composed of about a dozen rescuers standing elbow to elbow with a probe pole 3 to 3.5 m (about 10 to 12 ft) long. The rescuers would probe once between their feet—each probe entering the snow about 75 cm (30 inches) from the neighbors on either side—and then advance 70 cm (about 30 inches) and probe again. The probability of detection (POD) was thought to be about 70% to 76%. If the probe line missed on the first pass—which tended to happen—the area was probed again and again. Behind the probe line, shovelers stood ready to check out any possible strike. The line did not stop in such an event but continued to march forward with a methodical "down, up, step" cadence.

Canadian avalanche workers modified this traditional probe line method in the late 1990s when they found it more efficient to probe three times per step rather than one time per step and slightly tightened the probe-grid spacing. However, experience showed that for all probe lines, the PODs were much less than described in the literature. In 2004, Ballard and colleagues developed a computer program that simulated a human body in an avalanche and compared the probabilities of detection for any probe-grid spacing.[1] The results showed the POD for the traditional probe line to be 59% rather than the 70% believed earlier. As probe-grid spacings were reduced, the POD increased, but the search times also increased. Rescuers must balance the speed of the search with the length of time of the search. At a grid spacing of less than 50 cm, the search time increased faster than the POD. Thus, a grid spacing of 50 by 50 cm (20 by 20 inches) was optimal, yielding the best combination of POD and search time. For a three-hole-per-step (3HPS) probe, probers stand with arms out, wrist to wrist. Probers first probe between their feet and then probe 50 cm to the right and then 50 cm to the left (Fig. 2-20). At a command from the leader, the line advances 50 cm (one step). This method gives an 88% chance of finding the victim on the first pass with an estimated time to discovery that is nearly identical to the traditional spacing. Ski patrols and mountain rescue teams that have adopted the 3HPS with a 50 × 50-cm grid have been pleased with the improved efficiency and effectiveness of the probe line.

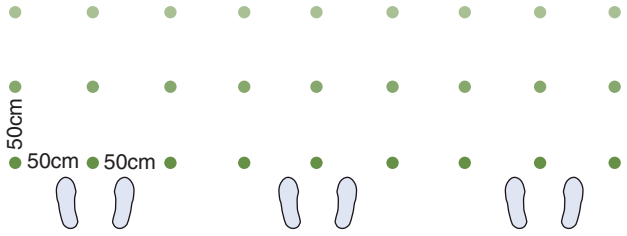


Figure 2-20. Three-probe spacing for 50 × 50-cm three-hole-per-step probe method.

Because rescuers insert more probes per rescuer, it is possible to have fewer rescuers on a probe line, and short probe lines are easier to manage and faster than long probe lines. Five rescuers doing 3HPS at 50-cm intervals form a slightly longer probe line than nine rescuers using the traditional method. To ensure proper spacing, it is most helpful to use a guidon cord with marked 50-cm intervals. Suspended between two rescuers, the lightweight guidon cord positions rescuers and probes to the right spots, allowing the line to move smoothly and efficiently without interruption. When a guidon cord is not used, the probe line must be frequently stopped and reassessed to ensure proper spacing. Rescuers managing the guidon cord should also place red flags every 3 to 5 m to mark the edge of the searched area.

If the victim is missed on the first pass, the second pass can be offset by 25 cm (10 inches) in each direction and the area researched. The second pass increases the POD to 99%.

Rescue teams use probe lines to find most avalanche victims not equipped with transceivers, RECCO reflectors, or when an avalanche rescue dog fails to locate the victim. However, these search methods should all be used concurrently. Because probe lines are time intensive, few victims are found alive by this technique alone (see Table 2-2).

RECCO. RECCO (RECCO AB, Sweden, www.recco.com) is an electronic rescue system that enables organized rescue teams to find victims who are equipped with a reflector (Fig. 2-21). The system consists of two parts: a detector used by the rescue teams (either on the ground or from helicopters) and the reflector worn by the recreationist. About the size and weight of a notebook computer, the detector is easily transported to the accident site. The detector transmits a directional radar signal. When it hits the reflector, the signal's frequency is doubled and reflected back to the detector, and the rescuer can follow the

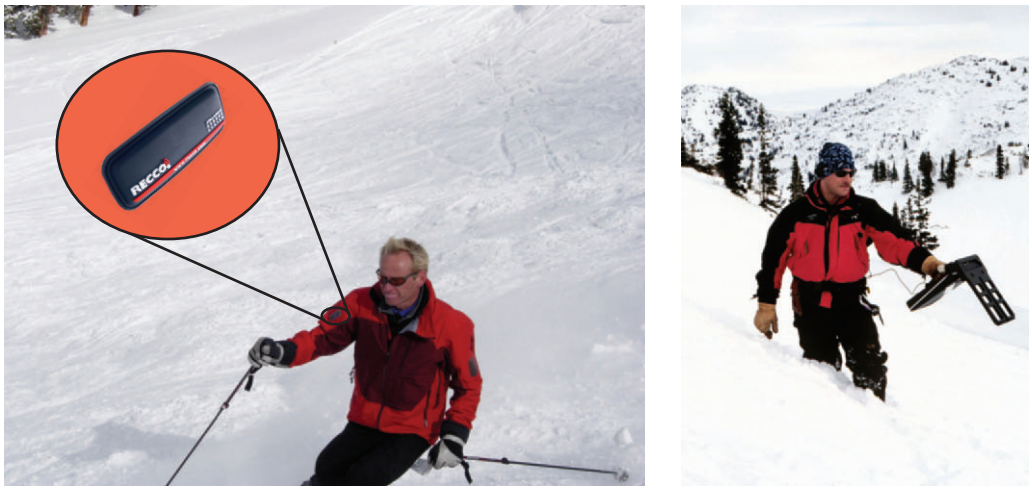


Figure 2-21. **A,** The RECCO reflector is a thin circuit card covered in soft plastic. It does not need batteries and does not need to be turned on or off. The reflector can be attached to jackets, pants, boots, and helmets. **B,** The RECCO detector consists of a transmitter and a receiver. It can also be used from a helicopter. (Courtesy RECCO AB, Sweden.)

signal to the buried person. The reflectors are small, passive (no batteries) electronic transponders that are fitted into outerwear, ski and snowboard boots, and helmets. The system will detect some electronic equipment (with diminished range) such as cell phones, electronic cameras, radios, and even turned-off avalanche rescue beacons, so RECCO should be used by rescue teams together with avalanche rescue dogs, rescue beacons, and probers in the first response to any avalanche rescue. In North America, more than 120 ski areas, mountain rescue teams, and National Parks have detectors. Worldwide, more than 500 ski resorts and helicopter rescue teams have them.

Avalanche Guard. If the threat of a second avalanche exists, one person should stand in a safe location to shout out a warning. This gives the searchers a few seconds to flee to safety. Rescues are often carried out under dangerous conditions, and self-preservation should be a major consideration.

THE MODERN AVALANCHE VICTIM

Avalanche deaths have increased in the United States each decade since 1950. Figure 2-22 shows annual deaths averaged over 5-year periods. From 1950 to 2004, 716 people have died in avalanches. Of these, 566 (79%) were men and 63 (9%) were women. (Interestingly, not all accident reports list the sex of the victim.) The average age of all victims is 30 years. The youngest was 6; the oldest, 67.

Figure 2-23 shows the activity groups for the victims. Most victims (86%) were pursuing some form of recreation at the time of the accident, with climbers, backcountry skiers, and snowmobilers heading the list. The backcountry-skiers category also includes ski mountaineers, helicopter skiers, and snowcat skiers. The distinction between backcountry skiers and lift skiers is that lift skiers pursue their sport in and around developed ski areas and rely on lifts to get them up the hill. Out-of-bounds lift skiers are skiers who leave the ski area boundary or ski into "closed" areas within the ski area. In-area skiers are those caught and killed on open terrain within the ski area boundary; it should be noted that only three deaths have occurred on open runs in the last 30 years. Miscellaneous recreation includes sledders and persons playing in the snow,

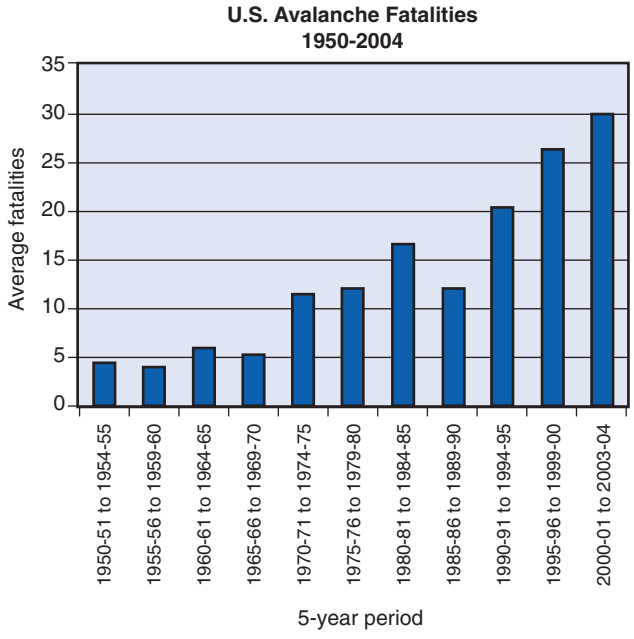


Figure 2-22. Avalanche fatalities in the United States averaged by five-winter periods, 1950–51 to 2003–04.

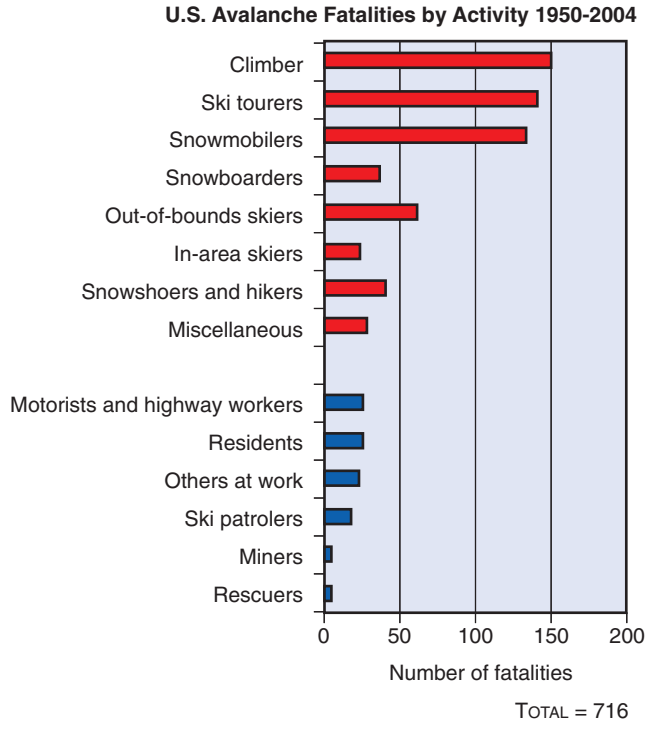


Figure 2-23. Avalanche fatalities in the United States from 1950–51 to 2003–04 by activity categories.

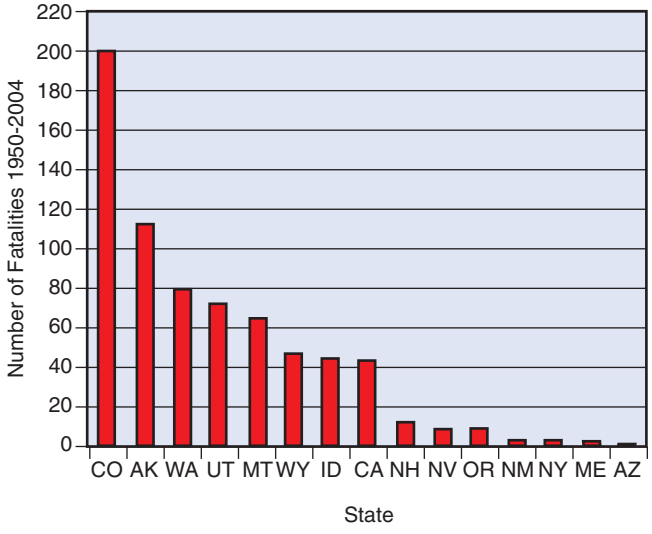


Figure 2-24. Avalanche fatalities in the United States from 1950–51 to 2003–04 by state.

campers, and even a most unlucky ski kayaker. Among non-recreation groups, avalanches strike houses (residents), highways (motorists and plow drivers), and the workplace (ski patrollers and others whose job puts them at risk). Since 1950, 15 states have registered avalanche fatalities (Fig. 2-24).

Statistics of Avalanche Burials

Numerous factors affect a buried victim's chances for survival: time buried, depth buried, clues on the surface, rescue equipment, injury, ability to fight the avalanche, body position, snow density, presence and size of air pocket, and luck. A victim who is uninjured and able to fight on the downhill ride usually has a better chance of being only partly buried, or, if completely buried, a better chance of creating an air pocket for breathing. A victim who is severely injured or knocked unconscious is like a rag doll being rolled, flipped, and twisted. Being trapped in an avalanche is a life-and-death struggle, with the upper hand going to those who fight the hardest.

Avalanches kill in two ways. First, serious injury is always possible in a tumble down an avalanche path. Trees, rocks, cliffs, and the wrenching action of snow in motion can do horrible things to the human body. About one quarter of all avalanche deaths are caused by trauma, especially to the head and neck. Second, snow burial causes asphyxiation (either obstructed airway or hypercapnia) in three quarters of avalanche deaths, and a very small percentage of avalanche victims succumb to hypothermia (see Avalanche Victim Physiology and Medical Treatment after Rescue, later). The problem of breathing in an avalanche does not start with being buried. A victim swept down in the churning maelstrom of snow has an extraordinarily hard time breathing. Inhaled snow clogs the mouth and nose; asphyxiation occurs quickly if the victim is buried with the airway already blocked. Snow that was light and airy when a skier carved turns in it becomes viselike in its new form. Where the snow might have been 80% air to begin with, it might be less than 50% air after an avalanche, and it is much less permeable to airflow, making it harder for the victim to breathe (but see AvaLung, earlier.)

Snow sets up hard and solid after an avalanche. It is almost impossible for victims to dig themselves out, even if buried less than a foot deep. Hard debris also makes recovery very difficult in the absence of a sturdy shovel. The pressure of the snow in a burial of several feet is sometimes so great that the victim is unable to expand the chest to draw a breath. Warm exhaled breath freezes on the snow around the face, eventually forming an ice lens that cuts off all airflow. This takes longer than snow-clogged airways, but the result is still death by asphyxiation.

Another factor that affects survival is the position of the victim's head—that is, whether the person is buried face up or face down. Data from a limited number of burials show the victim is twice as likely to survive if buried face up. In the face-up position, an air pocket forms around the face as the back of the head melts into the snow; in the face-down position, an air pocket cannot form as the face melts into the snow.

The statistics on survival are derived from a large number of avalanche burials (Figs. 2-25 and 2-26). In compiling these figures, the authors have included only persons who were totally buried in direct contact with the snow. Victims buried in the wreckage of buildings or vehicles are not included, as they can be shielded from the snow and can find sizable air pockets. Under such favorable circumstances, some victims have been able to live for days. In 1982, Anna Conrad lived for 5 days at Alpine Meadows, California, in the rubble of a demolished building, the longest survival on record in the United States.

A completely buried victim has a poor chance of survival, which is related to both time and depth of burial, as shown in

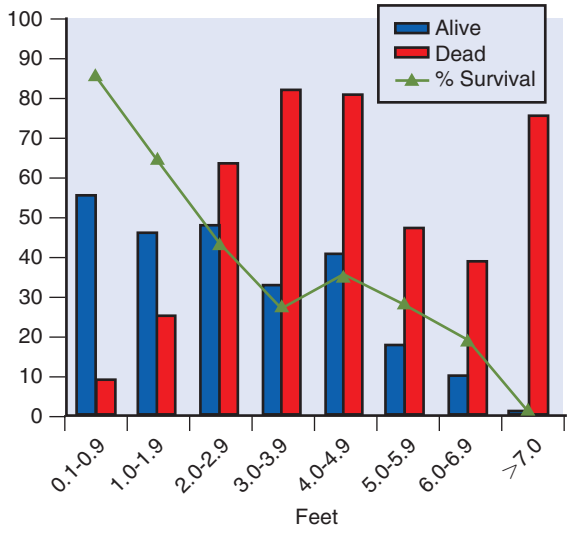


Figure 2-25. Percent survival versus depth of burial for U.S. avalanche fatalities and survivors in direct contact with the snow (not in a structure or vehicle) from 1950–51 through 2003–04.

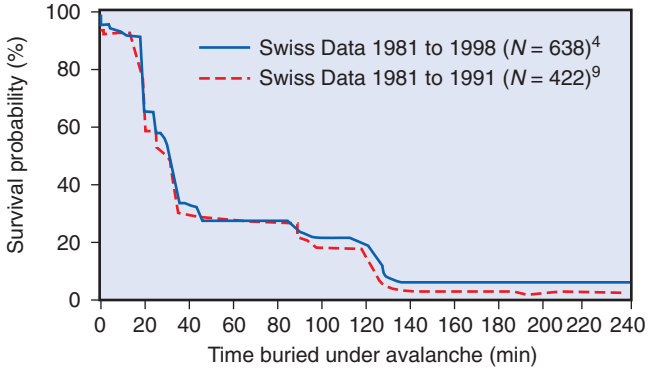


Figure 2-26. The solid blue line indicates data from Switzerland for survival probability for completely buried avalanche victims in open areas from 1981 to 1998 ($N = 638$) in relation to time (minutes) buried under the snow. Median extrication time was 37 minutes. The dashed red line represents survival probability for completely buried avalanche victims in open areas ($N = 422$) based on the Swiss data for 1981 to 1991 as calculated by Falk and colleagues.[9] (From Brugger H, Durrer B, Adler-Kastner L, et al: Resuscitation 51:7–15, 2001, with permission.)

Figure 2-25. Survival probabilities diminish with increasing burial depth, partly because it takes longer to uncover the more deeply buried victim. To date, no one in the United States who has been buried deeper than 2.1 m (7 feet) has been recovered alive; however, in Europe, two victims survived burials of 6 to 7 m (20 to 23 ft).[7,23] As important as burial depth may be in survival, time is the true enemy of the buried victim.

Survival statistics in the United States are very similar to those in Europe. Figure 2-26 presents the European survival probability function, showing decreasing survival with increasing burial time. In the first 15 minutes, more persons are found alive (>90%) than dead. At 30 minutes, an equal number are found dead and alive. After 30 minutes, more are found dead than alive, and the survival rate continues to diminish with increasing time. Speed is essential in the search. Because under favorable circumstances, buried victims can live for several hours beneath the snow, rescuers should never abandon a search prematurely. A miner in Colorado buried by an avalanche near a mine portal was able to dig himself free from nearly 1.8 m (6 feet) of debris after approximately 22 hours. In 2003, two snowshoe hikers caught near Washington's Mt. Baker survived burials of 22 and 24 hours. Such long survival times are a reminder that no rescue should be abandoned prematurely on the assumption that the victim is dead.

TABLE 2-1. Rescue Results as a Function of Rescuer, 1950–2004

	SELF-RESCUE	COMPANIONS	RESCUE TEAMS	TOTAL
Found alive	48 (14%)	198 (60%)	87 (26%)	333
Found dead	—	116 (23%)	383 (77%)	499

Type of rescue for buried avalanche victims in direct contact with snow, based on a sample of 832 burials in the United States from 1950–51 to 2003–04.

TABLE 2-2. Rescue Results as a Function of Locating Method, 1950–2004

	ALIVE	DEAD	TOTAL
Attached object or body part visible	140	54	194
Hasty search or spot probe	26	46	72
Coarse or fine probe	23	163	186
Transceiver	55	83	138
Avalanche dog	6	60	66
Voice	30	1	31
Other (e.g., digging, bulldozer)	20	14	34
Found after a long time	0	42	42
Not found, not recovered	0	35*	35
Not known	19	34	53
Inside vehicle	30	10	40
Inside structure	23	30	53
Totals	372	572	944

Method of locating (first contact) buried avalanche victims, based on a sample of 944 avalanche burials in the United States from 1950–51 to 2003–04.
*Eleven climbers died in an ice avalanche on Mt. Rainier (June, 1981); bodies not recovered.

Rescue Statistics

A buried victim's chance of survival directly relates not only to depth and length of time of burial but also to the type of rescue. Table 2-1 shows the statistics on survival as a function of the rescuer. Buried victims rescued by companions or groups nearby have a much better chance of survival than those rescued by organized rescue teams, time being the major influencing factor. Of those found alive, 60% were rescued by companions and 26% by an organized rescue team.

Table 2-2 compares the results of rescue by different techniques. Seventy-two percent of victims (140 of 194) who were

buried with a body part (such as a hand) or an attached object (such as a ski tip) protruding from the snow were found alive. In most cases, this was simply good luck, but in some cases it was the result of actively fighting with the avalanche or of thrusting a hand upward when the avalanche stopped. Either way, this statistic shows the advantages of a shallow burial: less time required to search, shorter digging time, and the possibility of attached objects or body parts being visible on the debris. Of the fatalities in this category, many were skiing or snowboarding alone with no companion to spot the hand or ski tip and provide rescue. Organized probe lines have found more victims than any other method, but because of the time required, most victims (88%) are recovered dead. Only 23 people were found alive by this method, with 163 recovered dead.

Rescue transceivers are an efficient technique for locating victims, but two problems have limited the number of survivors even among those wearing them. First, few beacon wearers are well practiced in using the beacon instantly and efficiently, and second, even after a quick pinpointing of the burial location, extricating the victim from deep burials may take too long to save the life. To recover a victim buried 1 m (3 ft) deep, companions have to remove at least 1.5 tons of snow. Since the first transceiver rescue in 1974, only 40% (55 of 138) of buried victims found with transceivers have been recovered alive. As dismal as this statistic is, there is a bright spot within the data. Prior to 2000, only 30% of transceiver users survived, but since 2000, 57% have survived burials (P = .003). The year 2000 marks the beginning of widespread use of digital avalanche rescue transceivers, which most people find easier to use. However, transceivers still do not guarantee a live rescue, which requires regular practice and training along with a dose of good luck.

Despite the sound-insulating properties of snow, 31 victims who were shallowly buried were able to yell and be heard by rescuers (voice contact). An unfortunate case was the man whose moans were heard but who was dead when uncovered 20 minutes later.

Trained search dogs are capable of locating buried victims very quickly, but because they are often brought to the scene only after extended periods of burial, there have been few live rescues. In the March 1982 avalanche disaster at Alpine Meadows, California, a dog made the first live recovery of an avalanche victim in the United States. Since then, dogs have made five additional live recoveries. Search and rescue teams and law enforcement agencies work closely with search dog handlers, and trained avalanche dogs are becoming common fixtures at ski areas in the western United States. A trained avalanche dog can search more effectively than 30 searchers. It moves rapidly over avalanche debris using its sensitive nose to scan for human scent diffusing up through the snowpack. Dogs are not 100% effective, however: they have found bodies buried 10 m deep but have also passed over some buried only 2 m (6 ft) deep.

These statistics point out the extreme importance of rescue skills. Organized rescue teams, such as ski patrollers and mountain rescuers, must be highly practiced. They must have adequate training, manpower, and equipment to perform a hasty search and probe the likely burial areas in a minimal time. For backcountry rescues, a buried victim's best hope for survival is to be found by the companions. The need to seek outside rescue units practically ensures a body recovery mission.

Avalanche Victim Physiology and Medical Treatment after Rescue

Avalanche Victim Morbidity and Mortality

Asphyxiation is the most common cause of death during avalanche burial. About 75% of avalanche deaths result from asphyxiation, about 25% from trauma, and very few from hypothermia.[13,16,22,24] Because asphyxiation is the major cause of death during avalanche burial, time to extrication is a major determinant of survival. Fully buried avalanche victims have a greater than 90% chance of survival if extricated within 15 minutes, but only 30% after about 35 minutes (see Figure 2-26),[4,9] emphasizing the need for companion rescue at the avalanche site. Survival beyond 30 minutes of burial requires an air pocket for breathing, and if the air pocket is large enough, avalanche victims may survive for hours and develop severe hypothermia.

Traumatic injury to avalanche victims depends on the terrain where the avalanche occurred. If the victim is carried through trees or over rock bands, then traumatic injury is more likely and may result in death. Grossman and colleagues, using data that included both partial and complete burials in Utah and Europe, reported that traumatic injuries occurred in 25% of survivors of avalanche accidents[13] (Table 2-3). The most common traumatic injuries were major orthopedic, soft tissue, and craniofacial injuries. Johnson and colleagues reviewed autopsy reports from 28 avalanche deaths in Utah over a 7-year period. Among 22 avalanche victims who died from asphyxiation, half had mild or moderate traumatic brain injury, which the authors argued could cause a depressed level of consciousness and contribute to death from asphyxiation.[14] All six of the avalanche deaths that were due to trauma had severe traumatic brain injury.

Respiratory Physiology of Avalanche Burial

Asphyxiation occurs during avalanche burial because inhaled snow occludes the upper airway or because expired air is rebreathed. Acute upper airway obstruction resulting in asphyxiation is one of the causes of early asphyxiation—that is, during the first 15 to 30 minutes of avalanche burial. Asphyxiation due to rebreathing expired air may also occur during this time if

TABLE 2-3. Injuries in Survivors of Avalanche Burial (Partial and Total)

	UTAH	EUROPE
Total Injuries	9 (Total, 91 avalanche accidents)	351 (Total, 1447 avalanche accidents)
Major orthopedic	3 (33%)	95 (27%)
Hypothermia requiring treatment at hospital arrival	2 (22%)	74 (21%)
Skin/soft tissue	1 (11%)	84 (25%)
Craniofacial	—	83 (24%)
Chest	3 (33%)	7 (2%)
Abdominal	—	4 (1%)

From Grossman MD, Saffle JR, Thomas F, Tremper B: J Trauma 29:1705–1709, 1989.

there is no air pocket for breathing, or it may be delayed if an air pocket is present. Inspired air contains 21% oxygen (O_2) and less than 0.03% CO_2, whereas expired air contains about 16% O_2 and 5% CO_2. Rebreathing expired air in an enclosed space results in progressive hypoxia and hypercapnia that will eventually result in death from asphyxiation. The larger the air pocket, the greater is the surface area for diffusion of expired air into the snowpack and for diffusion of ambient air from the snowpack into the air pocket, and thus the longer is the survival time before death occurs from asphyxiation. An ice mask is formed when water in the warm, humid expired air freezes on the snow surface in front of the face. Because this barrier is impermeable to air, it accelerates asphyxiation by preventing diffusion of expired air away from the air pocket in front of the mouth.

The physiology of asphyxiation from breathing with an air pocket in the snow was demonstrated in a study by Brugger and colleagues.[6] Subjects sat outside a snow mound and breathed through an air-tight mask connected by respiratory tubing to 1- or 2-L (0.9- to 1.8-quart) air pockets in snow. The snow had a density similar to that of avalanche debris (i.e., 150 to 600 kg/m^3, or 15% to 60% water). The initial fraction of inspired oxygen (F_{IO_2}) in the air pocket was 21%, and the initial fraction of inspired CO_2 (F_{ICO_2}) was near 0%. As expired air was rebreathed in the air pocket, F_{IO_2} decreased to about 10% and F_{ICO_2} increased to about 6% over 30 minutes (Fig. 2-27). These changes in O_2 and CO_2 in the air pocket resulted in a decreased arterial O_2 saturation as measured by a pulse oximeter (Spo_2%) and an increased end-tidal CO_2 ($ETCO_2$) partial pressure. Most subjects were not able to complete the entire 30 minutes of the study and had to stop secondary to dyspnea, hypercapnia, and hypoxia. Hypoxemia and hypercapnia occur as expired air is rebreathed. A smaller air pocket or denser snow causes a more rapid development of hypoxia and hypercapnia. Larger air pockets and less-dense snow allow more mixing of expired air with ambient air in the snowpack and result in longer survival before hypoxia and hypercapnia become severe enough to cause death from asphyxiation. Brugger and colleagues suggest that an equilibrium may occur where the F_{IO_2} and F_{ICO_2} in an air pocket reach a plateau within a physiologically tolerable range, and the avalanche victim may survive prolonged burial. This may occur even with small air pockets, as has been observed in the extrication of survivors of avalanche burials of up to 2 hours in duration.[4]

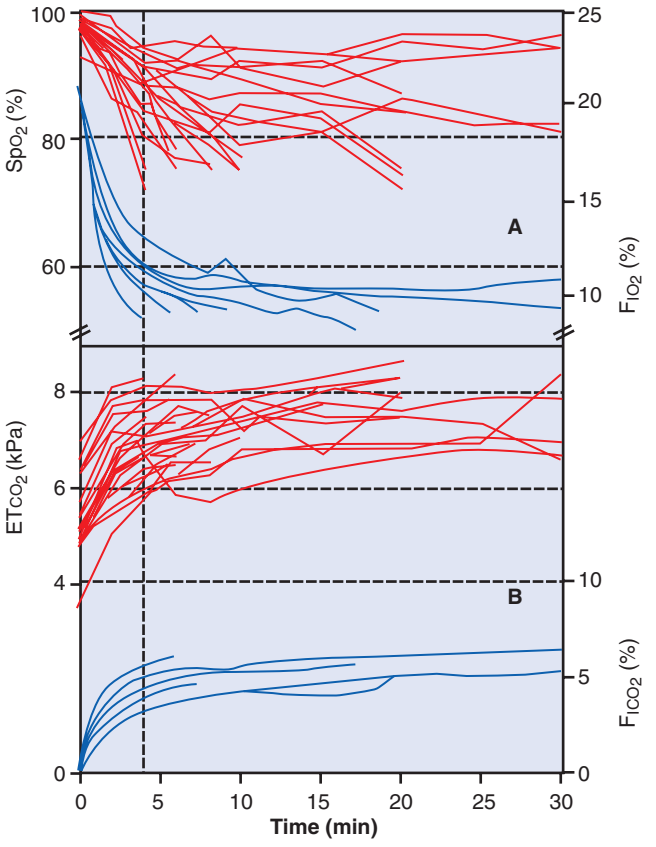


Figure 2-27. Curves of individual respiratory parameters in subjects breathing with a tight-fitting face mask connected to respiratory tubing running into 1- or 2-L air pockets in dense snow ($N = 28$). The x-axis represents time in minutes. Some subjects did not complete the 30-minute study because of dyspnea or hypoxia. **A,** Arterial oxygen saturation (Spo_2%) as measured by a digital pulse oximeter on the left y-axis *(red lines),* and fraction of inspired oxygen (FIO_2%) on the right y-axis *(blue lines).* **B,** Partial pressure of end-tidal CO_2 ($ETCO_2$) (kPa) on the left y-axis *(red lines)* and fraction of inspired CO_2 ($FICO_2$%) on the right y-axis *(blue lines).* (From Brugger H, Sumann G, Meister R, et al: Resuscitation 58:81–88, 2003, with permission.)

Radwin and colleagues[18] demonstrated that there is sufficient ambient air in densely packed snow to permit normal oxygenation and ventilation as long as all expired air is diverted out of the snowpack. They studied subjects totally buried in dense snow who inhaled air directly from the snowpack (density, 300 to 680 kg/m^3, or 30% to 68% water) through a two-way nonrebreathing valve attached to respiratory tubing that diverted all expired air to the snow surface. Subjects maintained normal oxygenation and ventilation for up to 90 minutes. This study demonstrated that there is sufficient air for breathing in snow with a density similar to that of avalanche debris, as long as expired air is not rebreathed. This is the principle behind the AvaLung (see Avalanche Rescue Equipment, earlier), designed to prolong survival during avalanche burial. Figure 2-28 describes its operation. Although the device prevents ice mask formation, the expired air permeates around the buried person's body and through the snow and eventually contaminates inspired air. Grissom and colleagues[11] compared breathing with this device while buried in dense snow, and breathing without the device but with a 500-cc (0.45-quart) air pocket in the snow. Mean burial time was 58 minutes when breathing with the device, and 10 minutes when breathing with a 500-cc air pocket in the snow. Development of hypoxia and hypercapnia were significantly delayed by breathing with the device. The AvaLung has resulted in survival from actual avalanche burials.[17]

Medical Treatment and Resuscitation of Avalanche Burial Victims

The key points regarding assessment and treatment of an extricated avalanche burial victim are presented in an algorithm in Figure 2-29. An initial impression of the level of consciousness is made as the head is exposed and cleared of snow. Opening the airway and ensuring adequate breathing are the primary medical interventions. Every effort should be made to clear the

Figure 2-28. The AvaLung 2 (Black Diamond Equipment, Ltd., Salt Lake City, UT), a breathing device intended to prolong survival during avalanche burial by diverting expired air away from inspired air drawn from the snowpack, is worn over all other clothing. *White arrows* show flow of inspiratory air, and *red arrows* show flow of expiratory air. The subject breathes in and out through the mouthpiece (A). Inhaled air enters from the snowpack through the one-way inspiratory valve on the side of the housing inside the mesh-protected harness on the chest (B). Expired air leaves the lungs via the mouthpiece and travels down the respiratory tubing to the housing and then passes through an expiratory one-way valve located at the bottom of the housing (B) and travels via respiratory tubing inside the harness around to the back (C). (AvaLung photo courtesy Black Diamond Equipment, Ltd.)

airway of snow as soon as possible and to provide assisted ventilation if the breathing is absent or ineffective. These measures should not wait until the entire body is extricated. If traumatic injury to the spinal column is suspected, or if there is evidence of head or facial trauma, then the spinal column is immobilized as the airway is opened, adequate breathing ensured, and oxygen provided. When the avalanche burial victim is unconscious, maintenance of the airway may be challenging because of the space limitations of the snow hole. If endotracheal intubation is required for the unconscious apneic patient not yet fully extricated from snow burial, then the inverse intubation technique[19] may be required. In this technique, the laryngoscope is held in the right hand while straddling the patient's body and facing the head and face. While facing the patient, insert the laryngoscope blade into the oropharynx with the right hand so that the larynx and cords can be visualized by leaning over and looking into the patient's mouth; the endotracheal tube is then passed through the cords with the left hand.

After an adequate airway and breathing are established and supplemental oxygen is provided, the circulation is assessed. The conscious patient is assumed to have a perfusing rhythm, and further treatment is directed at treating mild hypothermia and traumatic injuries. A patient found unconscious but with a pulse may have moderate or severe hypothermia and should be handled gently to avoid precipitating ventricular fibrillation (VF). Medical treatment of this patient is focused on ensuring adequate oxygenation and ventilation, either noninvasively with a bag-valve-mask device or by endotracheal intubation if clinically indicated, and on immobilizing the spinal column for transport and treating any obvious signs of trauma. Intravenous access may be obtained and warmed isotonic fluids infused. The treatment of hypothermia is described later.

If a pulse is not present after opening the airway and ventilating the patient, cardiopulmonary resuscitation (CPR) is begun. Before CPR is initiated, however, careful evaluation for the presence of a pulse should occur. Avalanche burial victims are hypothermic, which causes peripheral vasoconstriction and makes the pulse difficult to palpate. In addition, moderate to severe hypothermia causes depression of respiration and bradycardia. Before initiating CPR, palpation for a pulse should be done for a period of 30 to 45 seconds after the airway is opened and assisted ventilations are begun. CPR initiated on a severely hypothermic patient who actually has a perfusing rhythm may cause VF. If electrocardiographic monitoring is available, the cardiac rhythm is assessed, or alternatively an automatic external defibrillator may be applied. If the patient has VF, up to three defibrillations are attempted in the moderate or severely hypothermic patient with a core body temperature less than 30°C (86°F).[8] If these are unsuccessful, further attempts at defibrillation are done only after rewarming. Drugs usually administered as part of advanced cardiac life support (ACLS) are not effective below a core body temperature of about 30°C, and they may accumulate to toxic levels with a rebound effect as rewarming occurs.[8] If the patient is hypothermic but

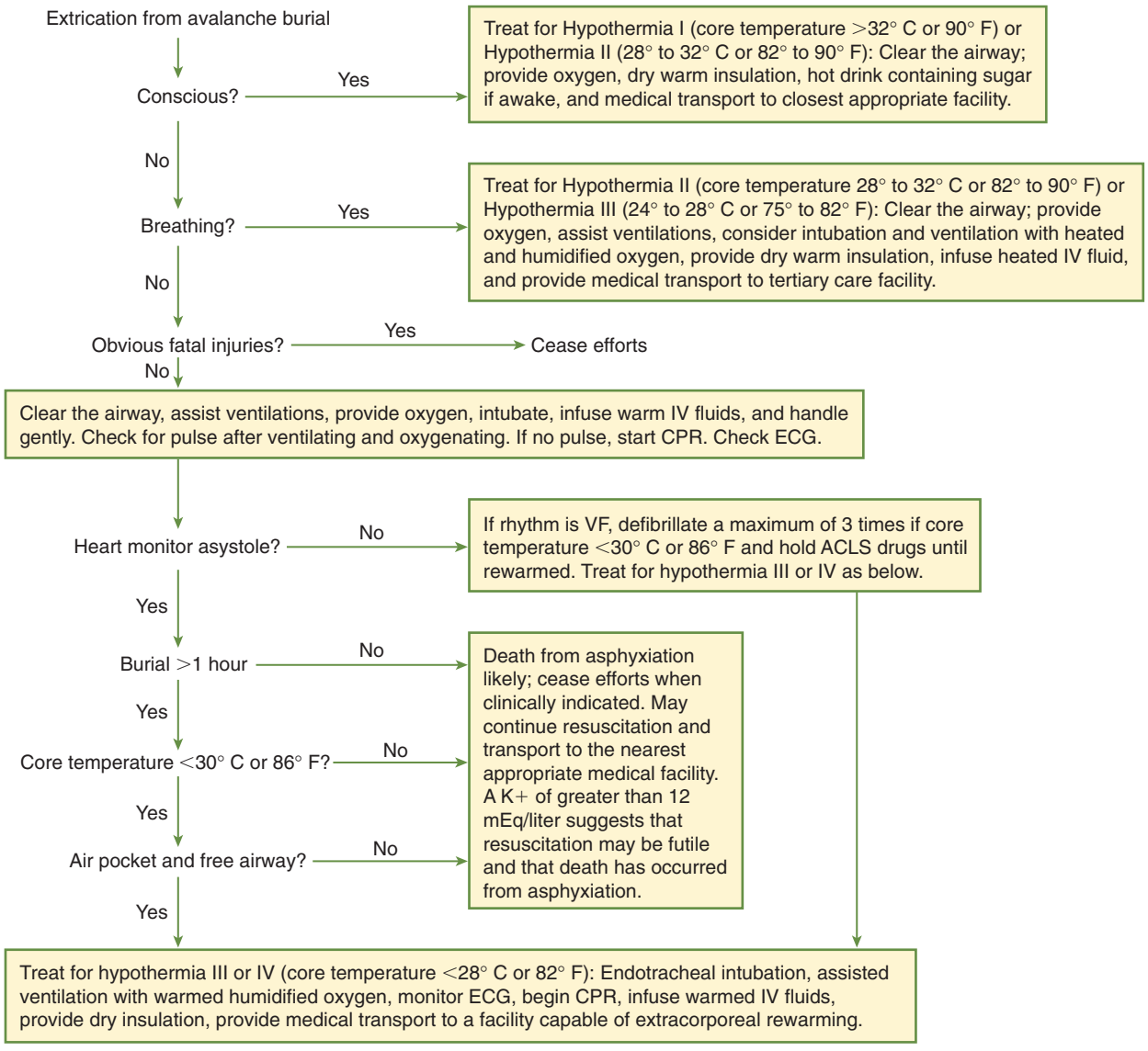


Figure 2-29. Assessment and medical care of extricated avalanche burial victims.

has a core body temperature greater than 30° C, the standard ACLS protocol is followed with longer intervals between administrations of drugs.

The likelihood of successful resuscitation of an avalanche burial victim who is in cardiac arrest at the time of extrication depends on whether cardiac arrest occurred from asphyxiation or from hypothermia. In burials of less than 1 or 2 hours with a core body temperature of greater than 30° C, resuscitation is unlikely to be successful because death has most likely occurred from asphyxiation. Avalanche victims extricated from burials of greater than 1 or 2 hours who have no signs of life but who are severely hypothermic (core temperature <30° C) and have an air pocket for breathing, may be considered for transport to a medical facility with capability for extracorporeal rewarming (see Figure 2-29). Most avalanche victims extricated in cardiac arrest, however, have died from asphyxiation, and continuing resuscitation efforts until rewarming occurs is unlikely to result in survival. This is especially true if no air pocket for breathing is present and burial time is greater than 1 hour. In a study by Locher and Walpoth, there were no survivors among 13 avalanche victims who were found in cardiac arrest after burials of 30 to 165 minutes' duration, suggesting that cardiac arrest was due to asphyxiation rather than hypothermia.[16] An air pocket for breathing must be present for an avalanche burial victim to survive long enough to develop severe hypothermia. If an air pocket for breathing is not present, the avalanche victim extricated from snow burial in cardiac arrest has most likely died from trauma or asphyxiation.

TABLE 2-4. Hypothermia Clinical Pre-Hospital Evaluation Using Swiss Society of Mountain Medicine Definitions

Hypothermia I	Patient alert, shivering (core temperature about 35° to 32° C [95° to 90° F])
Hypothermia II	Patient drowsy, non-shivering (core temperature about 32° to 28° C [90° to 82° F])
Hypothermia III	Patient unconscious (core temperature about 28° to 24° C [82° to 75° F])
Hypothermia IV	Patient not breathing (core temperature <24° C [<75° F])

From Brugger H, Durrer B, Adler-Kastner L: Resuscitation 31:11–16, 1996; Brugger H, Durrer B, Adler-Kastner L, et al: Resuscitation 51:7–15, 2001.

Hypothermia in the Avalanche Burial Victim

In avalanche burial victims who are extricated alive, hypothermia is the major medical problem that will require treatment.[2,3,4] Clinical definitions of hypothermia severity correlated with core body temperature are shown in Table 2-4. As the patient is extricated from snow burial, warm dry insulation is provided during packaging, and wet clothing is removed as soon as is practical. If the patient is unconscious, moderate or severe hypothermia is suspected and handling should be gentle to avoid precipitating VF. When treatment of moderate or severe hypothermia is clinically indicated, intravenous access is obtained, and warmed (42° C [107.6° F]) isotonic fluid is infused. If the patient is unconscious, intubation is appropriate to provide adequate oxygenation and ventilation with heated (40° to 45° C [104° to 113° F]) humidified oxygen. The goal of treatment of moderate to severe hypothermia before reaching the hospital is to limit temperature afterdrop during medical transport to a hospital, where definitive rewarming can occur. The shivering patient with mild hypothermia can rewarm in the field if dry insulation is provided. If the patient is conscious and alert, warm fluids containing sugar may be given by mouth. If possible, patients with mild hypothermia should remain supine until rewarming has occurred in order to prevent a greater temperature afterdrop from ambulation. Optimally, the severity of hypothermia is determined by a core body temperature measurement in the field, preferably using an esophageal probe, but tympanic membrane sensors or rectal probes may also be used. If core body temperature cannot be measured in the field, the severity of hypothermia may be estimated using the Swiss hypothermia stages I to IV as determined by the clinical presentation (see Table 2-4).[2]

The severity of hypothermia may also be estimated from the time the victim was buried and the average rate of core temperature cooling during the burial. Grissom and colleagues measured core temperature cooling rate during snow burial in a controlled experiment.[12] In subjects buried in dense snow for up to 60 minutes wearing a lightweight clothing insulation system while breathing with an artificial device (the AvaLung), core body temperature cooling rate was about 1.3° C (2.3° F) per hour.

Retrospective studies of core temperature on hospital arrival by Locher and Walpoth[16] suggest that the average core temperature cooling rate is 3° C per hour (range, 0.75° to 4.75° C [1.4° to 8.6° F] per hour in survivors), greater than the 1.3° C per hour estimated by Grissom and colleagues.[12] The higher rate of cooling reported by Locher is based on a known time of burial and a known time of arrival at the hospital where core body temperature was recorded, and it does not differentiate between core temperature cooling rate during snow burial and cooling rate during medical transport. Grissom and colleagues report that core body temperature cooling rate increases transiently after extrication from snow burial.[12] Core body temperature cooling rate accelerated by 50% for 25 minutes after extrication in subjects buried in snow for 60 minutes.[10] An accelerated cooling rate during and after extrication places avalanche burial victims at greater risk of complications due to hypothermia. Every effort should be made by rescue personnel to prevent further heat loss in avalanche burial victims, starting as soon as possible during extrication from the snow.

Core temperature cooling rate during avalanche burial may also be estimated from anecdotal reports of prolonged survival during avalanche burial. In one case, a 25-year-old male snowboarder with a large air pocket in front of his body survived 20 hours of avalanche burial.[21] At the time of extrication, he had a core body temperature of 25.6° C (78.1° F) (tympanic) in snow at −5° C (23° F) and was spontaneously breathing with a Glasgow Coma Scale score of 8, heart rate of 35, and palpable pulse. Core body temperature cooling rate in this anecdotal report was about 0.6° C (1.1° F) per hour. The large air pocket probably allowed adequate diffusion of expired air away from inspired air and prevented asphyxiation. In that same rescue, the survivor's companion was found dead from asphyxiation after 20 hours of burial with a core body temperature of 6° C (42.8° F) (tympanic), which is an average core body temperature cooling rate of about 1.5° C (2.7° F) per hour. Another case of survival after prolonged burial occurred in a 21-year-old female snowshoe hiker.[15] She survived 24 hours of avalanche burial with a large air pocket around her head, and was extricated unconscious. After transport to a local ski lodge, where she was placed in warm insulation and warmed intravenous fluids were infused, she regained consciousness and had a core body temperature of 29.3° C (84.7° F). These anecdotal reports demonstrate that core body temperature cooling rate during avalanche burial may vary significantly depending on adequacy of ventilation as determined by the size of an air pocket.

SUMMARY

Asphyxiation is the major cause of death during avalanche burial and is time dependent. Avalanche burial victims extricated within 15 minutes have a greater than 90% chance of survival, but this decreases to about a 30% chance of survival if extricated after 30 minutes, emphasizing the need for companion rescue using avalanche transceivers and shovels. Survival beyond 30 minutes of burial depends on an air pocket for breathing. The larger the size of the air pocket, the longer survival is possible during the burial. This is because asphyxiation during avalanche burial is caused by rebreathing expired air that contains 16% O_2 and 5% CO_2. A larger air pocket provides more surface area for diffusion of expired air away from an avalanche burial victim, allowing more ambient air from the snowpack to diffuse into the air pocket for inspiration. Opening the airway and ensuring adequate ventilation and providing supplemental oxygen are the primary medical interventions for an extricated avalanche burial victim. If the victim

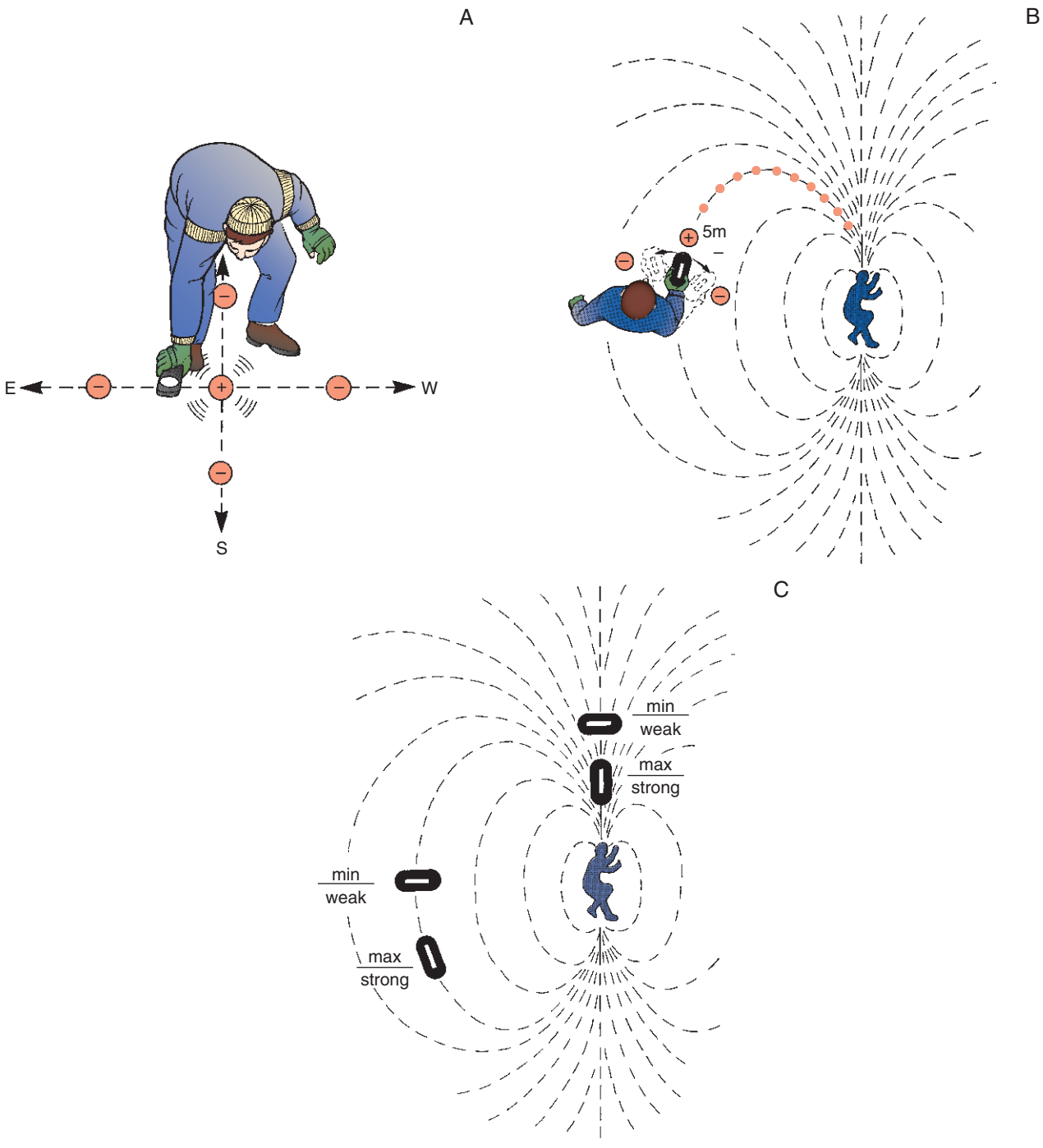


Figure 2-30. Induction line search. (From Auerbach PS, Donner HJ, Weiss EA: Field Guide to Wilderness Medicine, St Louis, Mosby, 1999, with permission.)

is conscious, mild hypothermia is most likely and treatment consists of proving warm dry insulation and warm, sugar-containing liquids. Rewarming will occur through shivering. Avalanche burial victims who are not shivering have progressed to moderate or severe hypothermia and require medical transport to a hospital for definitive rewarming. Unconscious avalanche burial victims who are breathing may require intubation for airway control and assisted ventilation, and they are most likely moderately to severely hypothermic. Victims extricated in asystolic cardiac arrest have most likely died from asphyxiation, but if burial time is greater than 1 or 2 hours and core body temperature is less than 30°C (86°F), they may be severely hypothermic and resuscitation efforts may be continued while the patient is transported to a medical facility capable of extracorporeal rewarming.

ACKNOWLEDGMENT

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SUGGESTED READINGS

The Avalanche Review, P.O. Box 2831, Pagosa Springs, CO 81147 (official publication of the American Avalanche Association).
Daffern T: Avalanche Safety for Skiers and Climbers, 2nd ed. Seattle, Cloudcap, 1992.
Fraser C: Avalanches and Snow Safety. New York, Charles Scribner, 1978.
Fredston J, Fesler D: Snow sense: A guide to evaluating avalanche hazard, ed 4. Anchorage, Alaska Mountain Safety Center, 1994.
LaChapelle E: The ABC's of Avalanche Safety, 2nd ed. Seattle, Mountaineers Books, 1985.
LaChapelle E: Secrets of the snow: Visual clues to avalanche and ski conditions. Seattle, University of Washington Press, 2001.
Logan N, Atkins D: The snowy torrents: Avalanche accidents in the United States 1980–86. Denver, Colorado Geological Survey Spec Pub 39, 1996.
McClung D, Schaerer P: The Avalanche Handbook. Seattle, Mountaineers Books, 1993.
Tremper B: Staying Alive in Avalanche Terrain. Seattle, Mountaineers Books, 2001.

APPENDIX A: PUBLIC INFORMATION

Twenty-four-hour regional avalanche information is available, generally from November through April, via the following Internet websites or telephone numbers with recorded messages.

California
- Internet: www.fs.fed.us/r5/tahoe/avalanche/
- Truckee 530-587-2158
- Mammoth Lakes 760-924-5500
- Internet: *www.shastaavalanche.org/*
- Mount Shasta 530-926-9613

Colorado
- Internet: www.geosurvey.state.co.us/avalanche/
- Denver/Boulder 303-275-5360
- Fort Collins 970-482-0457
- Colorado Springs 719-520-0020
- Summit County 970-668-0600
- Crested Butte 970-349-4022
- Aspen 970-920-1664
- Durango 970-247-8187

Idaho
- Internet: www.avalanche.org/~svactr/
- Sun Valley 208-622-8027

Montana
- Internet: www.mtavalanche.com/
- Bozeman 406-587-6981
- Missoula 406-549-4488

New Hampshire
- Internet: www.mountwashington.org/avalanche/
- North Conway 603-466-2713

Utah
- Internet: www.avalanche.org/~uac/
- Salt Lake City 801-364-1581
- Provo 801-378-4333
- Ogden 801-626-8600
- Park City 435-658-5512
- Logan 801-797-4146
- Alta 801-742-0830
- Moab 801-259-7669

Washington and Oregon
- Internet: www.nwac.us
- Seattle 206-526-6677
- Portland 503-808-2400

Wyoming
- Internet: www.jhavalanche.org/
- Jackson 307-733-2664

Canada
- Internet: www.avalanche.ca

APPENDIX B: AVALANCHE EDUCATION

Several organizations teach basic and advanced avalanche awareness and offer training courses. Beyond those listed here, many local colleges and universities, ski patrols, and recreation departments offer courses.

Alaska Avalanche School Anchorage, AK, www.alaskaavalanche.com
American Avalanche Institute Wilson, WY, www.avalanchecourse.com
Canadian Avalanche Association Training Courses Revelstoke, BC, Canada, www.avalanche.ca
National Avalanche School National Avalanche Foundation, Lakewood, Colo, www.avalancheschool.org
National Outdoor Leadership School Lander, WY, www.nols.edu
Northwest Avalanche Institute Enumclaw, WA, www.avalanche.org/~nai/
Silverton Avalanche School Silverton, CO, www.silvertonavalancheschool.com
Telluride Avalanche School Telluride, CO, www.sanjuanrivers.com

Colleges and universities that offer avalanche and snow-related courses (mostly graduate level) and the email contacts:

University of Arizona (Tucson)
Department of Hydrology and Water Resources
Dr. Roger Bales: roger@hwr.arizona.edu

Arizona State University (Tempe)
Department of Geography
Dr. Andrew Ellis: andrew.w.ellis@asu.edu
University of California, Santa Barbara
Donald Bren School of Environmental Science and Management
Dr. Jeff Dozier: dozier@bren.ucsb.edu
University of British Columbia (Vancouver)
Department of Geography & Civil Engineering
Dr. Dave McClung: mcclung@geog.ubc.ca
University of Calgary (Calgary)
Department of Geology and Geophysics
Department of Civil Engineering
Dr. Bruce Jamieson: jbjamies@ucalgary.ca
University of Colorado (Boulder)
Department of Geography
Institute of Arctic and Alpine Research (INSTAAR)
Dr. Mark W. Williams: markw@snobear.colorado.edu
Colorado State University (Fort Collins)
Department of Geosciences
Dr. Steven Fassnacht: srf@cnr.colostate.edu
Montana State University (Bozeman)
Department of Civil Engineering
Dr. Ed Adams: eda@ce.montana.edu
Department of Earth Sciences
Dr. Katherine Hansen: ueskh@montana.edu
Northern Arizona University (Flagstaff)
Department of Geography
Dr. Lee Dexter: Lee.Dexter@nau.edu
University of South Carolina (Columbia)
Department of Geography
Dr. Cary Mock: mockcj@sc.edu
Rutgers University (Piscataway, NJ)
Department of Geography
Dr. David A. Robinson: drobins@rci.rutgers.edu
University of Utah (Salt Lake City)
Department of Meteorology
Dr. John Horel: jhorel@met.utah.edu
Department of Geography
Dr. Bill Harrison: w.l.harrison@m.cc.utah.edu
Utah State University (Logan)
Department of Forest Resources
Dr. Michael J. Jenkins: mjenkins@cc.usu.edu
University of Washington (Seattle)
Geophysics Program
Dr. Howard Conway: conway@geophys.washington.edu

APPENDIX C: AVALANCHE SAFETY EQUIPMENT MANUFACTURERS AND SUPPLIERS

Backcountry Access, Inc., Boulder, CO 80301, www.bcaccess.com
- Backcountry ski equipment, Tracker rescue beacons

Black Diamond Equipment, Ltd., Salt Lake City, UT, www.bdel.com
- Backcountry ski equipment, clothing, survival gear, rescue beacons, AvaLung

Cascade Toboggan, Sandpoint, ID, www.cascadetoboggan.com
- Shovels, probes, rescue beacons, other rescue equipment

Climb High, Shelburne, VT, www.climbhigh.com
- Backcountry ski and expedition equipment, rescue beacons, shovels, probes, and clothing

Eastern Mountain Sports, Peterborough, NH (or local retail store), www.ems.com
- Backcountry ski equipment, clothing, survival gear, rescue beacons

Life Link International, Jackson Hole, WY, www.life-link.com
- Snowpit instruments, shovels, probes, rescue beacons, airbag systems, other rescue equipment

Mountain Safety Research, Seattle, www.msrcorp.com
- Survival gear, probes, other rescue equipment

Ortovox USA, Inc., Hopkinton, NH, 603-746-3176, www.ortovox.com
- Ortovox rescue beacons, shovels, rescue equipment

Recreational Equipment, Inc., Sumner, WA (or local retail store), www.rei.com
- Backcountry ski equipment, clothing, survival gear, rescue beacons

Survival on Snow, Inc., St. Albert, AB, Canada, www.sos-find.com
- SOS rescue beacons, rescue equipment

Wasatch Touring, Salt Lake City, UT, www.wasatchtouring.com
- Snowpit instruments, probes, shovels, rescue beacons

The references for this chapter can be found on the accompanying DVD-ROM.

3 Lightning Injuries

Mary Ann Cooper, Christopher J. Andrews, and Ronald L. Holle

HISTORICAL OVERVIEW

Lightning has caused injuries to humans since man evolved on Earth. Lightning played a major part in nearly every ancient religion and culture and continues to engender stories, perceptions, and myths in modern cultures.[91,170]

Priests, the earliest astronomers and meteorologists, became proficient at weather prediction, interpreting changes in weather as omens of good or bad fortune, sometimes to the advantage of their political mentors. Greek artists incorporated lightning symbols representing Zeus's tool of warning or favor. Romans killed by lightning were considered disfavored and were denied burial rituals. Many Roman emperors wore laurel wreaths or sealskin to ward off lightning strikes. Thursday is named for Thor, the Norse god of thunder. Chinese mythology had a goddess of lightning in the "Ministry of Thunderstorms." Lightning played a role in Buddhist symbolism.

Although lightning is most frequently rendered as fire, it also has been represented in French and Asian histories as stone axes hurled from the heavens. Africans and some Native American tribes picture lightning as a Thunderbird. The Navajos credit the creation of the Grand Canyon to lightning.[20] Native Australian stories incorporate lightning symbols, where the lightning spirit is depicted as having axes attached to his joints, beating together to make thunder.

People in both developed and third world countries often regard lightning and thunder with a great deal of fear as mysterious, uncontrollable, and unmanageable. In the 1990s and early 2000s, there was a series of lightning injuries at soccer games in Africa. In one incident, where only members of one team were injured, it was ascribed to witchcraft on the part of the other team. Even in "civilized" Western societies, lightning can take on mystical significance. When lightning struck an English cathedral just before enthronement of a controversial bishop in the late 20th century, some regarded it as an omen.

MYTHS, SUPERSTITIONS, AND MISCONCEPTIONS[58]

The Roman author Pliny noted that a man who heard thunder was safe from the lightning stroke. This implies that the person who hears the thunder is far enough away from the lightning strike to survive it, although we now know they may still become injured. Persons so close that they cannot hear the thunder are much more likely to be injured. Persons who receive ground current are more likely to report both seeing the flash and hearing the stroke, indicating that the main stroke was some distance away.

Many myths about lightning, including the notion that lightning strikes are invariably fatal, persist (Box 3-1). According to an American study of cases reported in the lightning literature since 1900, lightning strike carries a mortality rate of 30% and morbidity rate of 70%.[52] A slightly different statistical interpretation of the same data yielded a mortality figure of 20%.[5,11] Because literature reports are usually biased toward the severe or interesting cases, case reviews tend to overestimate mortality rate. In reality, mortality rate may be as low as 5% to 10%.[44]

Most people suspect that the major cause of death is from burns. However, the only cause of immediate death is from cardiac arrest.[52] Persons who are stunned or lose consciousness without cardiopulmonary arrest are highly unlikely to die, although they may still have serious, especially long-term, sequelae.[54,62,156] Delayed causes of death include suicide induced by the life changes from disability wrought by lightning.[55,59]

Most people know to seek shelter when storm clouds roll overhead. Few realize that one of the most dangerous times for a fatal strike is before the storm.[58] Lightning may travel nearly horizontally as far as 10 miles or more in front of the thunderstorm and seem to occur "out of a clear blue sky," or at least when the day is still sunny where the person is located. The faster the storm is traveling and the more violent it is, the more likely a fatal strike will occur. Another time underestimated for the potential danger of lightning is the end of a thunderstorm, which has been shown to be as dangerous as the start of the storm.

The "30-30 rule" is now recommended for lightning safety.[66] If you see lightning and count fewer than 30 seconds before you hear the thunder, you are already in danger and should be seeking shelter. Activities should not be resumed for at least 30 minutes after the last lightning is seen and the last thunder heard.[66,191] To calculate your distance from lightning, take the number of seconds between the "flash" and the "bang" (flash-to-bang method) and divide by 5 to find the number of miles.[189]

The problem with the flash-to-bang method is that it is sometimes difficult to match the correct thunder to the correct lightning flash in an active storm. In addition, many people forget to divide by 5 and so overestimate the miles (and their safety factor) by a factor of five. Therefore, it is best to count to 30 seconds as a reasonable precaution and not worry about the distance involved or, better yet, seek shelter at the first sound

Box 3-1. The Most Common Myths and Facts

NO TRUTH

Lightning injuries are always fatal.
The cause of death from lightning injury is burns.
Burns are a major component of lightning injury.
Nothing is left of a person after a lightning strike except a pile of ashes.
Lightning victims have "entry" and "exit" points.
Lightning victims have internal burns.
One can predict the degree of lightning injury from the voltage, amperage, and polarity of the strike.
Metal (on the body or not) attracts lightning.
Lightning does not hit outside the rainstorm.
It is safe to wait until the rain arrives to evacuate.
It is safe to finish the game if lightning is nearby.
After the rainstorm passes and rain stops falling, it is safe to resume activity.
If you can see blue sky, lightning danger is minimal.
Lightning injuries cannot occur inside a building.
Rubber tires (shoes, raincoats, sitting on a backpack) protect a person from lightning.
Cellular phones attract lightning.
Golf, picnic, and other electrically grounded shelters are safe.
Grounding a building makes it safe from lightning damage.
Lightning victims remain electrified and dangerous to touch.
Lightning victims are easier to resuscitate than are other cardiac arrest victims.
If there are no outward signs of lightning injury, the damage cannot be serious.
Lightning victims usually require treatment similar to that for high-voltage electrical injuries with aggressive fluid resuscitation, urine alkalinization, and fasciotomies.
Lightning survivors have few permanent problems.
Lightning never strikes the same place twice.
It is safe to seek shelter and dryness under a tree.
Tall objects provide a 45-degree cone of protection.
The majority of persons injured are golfers.

SOME TRUTH

Lightning always hits the highest object.
The "pointier" an object, the more likely it will be hit.

TRUE

No place outside is safe near thunderstorms.
It is better to be "proactive" rather than "reactive" with lightning injury avoidance.
Outdoor sports have the fastest rising lightning casualty rate.
The top activities and places for lightning casualties in the United States are:
1. Open fields and elevated locations
2. Under trees (or other tall isolated object)
3. Water-related activities (e.g., swimming, boating, fishing)
4. Golfing
5. Open vehicles (e.g., farm, construction vehicles)
6. Telephone
7. Radio and radio equipment

The best shelter is a typical house or other fully enclosed and substantially constructed building with properly earthed plumbing and wiring.
The second best shelter is a vehicle with a solid metal roof and metal sides.
Open picnic, bus, golf, and rain shelters, as well as tents and other camping cover, offer absolutely no protection from lightning.
Having and following a lightning safety plan can decrease the number of lightning deaths and injuries.
One cannot make up for too many bad decisions by choosing between bad options when "caught" outside.
The primary cause of death is cardiac and respiratory arrest at the time of the injury.
After all the proactive options for safety have been ignored, the lightning "safety" crouch may be the only option, but probably makes little difference in risk.
Ninety percent of lightning victims survive, but often with disability.
Lightning victims are safe to touch.
Automated external defibrillators (AEDs) have been useful in some resuscitations.

of thunder, especially if you are responsible for the safety of a group. Thunder is seldom audible more than 10 miles away and often much less audible in a city, on a mountainside opposite the thunderstorm, or in a noisy sports stadium.

The distance between successive lightning flashes may be as little as a few yards or as much as 5 miles plus or minus another 5 miles (a count of 50 seconds) depending on the terrain and other local geographic factors.[146] One way to teach children lightning safety is to teach them: "If you see it, flee it; if you hear it, clear it." Even better is "If thunder roars, go indoors."

Most people believe they are immune from lightning strikes when inside a building. However, a number of injuries occur to persons who are in their homes or places of employment.[4,9,78,168,181] Side flashes strike people through plumbing fixtures, telephones, and other appliances attached to the outside of the house by metal conductors.[4,78] Earth potential rise (EPR) can be significant, and in one study was thought to account for 80% of telephone-related injuries. Cellular or portable phones offer complete protection from the electrical effects, although victims may suffer acoustic damage from static in the earpiece similar to having a firecracker go off next to the ear.[9,153] With a hard-wired phone, persons may suffer neurocognitive deficits,[55,63,167] death, or a myriad of other lightning-related problems because the phone system in most houses is not grounded to the house's electrical system and so acts as a conduit for lightning either to come into the home or to exit from it. Telephone companies include warnings in their directories against using telephones during thunderstorms.

Taking shelter in small sheds, such as hikers' lean-tos or those on golf courses, can be especially dangerous when lightning splashes onto the inhabitants. Tent poles offer similar danger. Unfortunately, the most recently published *National Fire Pro-*

tection Association (NFPA) Journal discusses protection that may be effective for the structural safety of the shelter but not adequate to protect people sheltered within from side flashes. Being in an inadequately protected open shelter may actually increase the lightning risk to any inhabitants who seek shelter in them by increasing the inhabitants' effective height.

The "crispy critter" myth is the belief that the victim struck by lightning bursts into flames or is reduced to a pile of ashes.[58] In reality, lightning often flashes over the outside of a victim, sometimes blowing off the clothes but leaving few external signs of injury and few, if any, burns.

Two other myths held by the public and many physicians are that "If you're not killed by lightning, you'll be OK" and "If there are no outward signs of lightning injury, the damage can't be serious."[58] Medical literature, because of lack of follow-up case reports, also implies that there are few permanent sequelae of lightning injury. Several permanent sequelae may occur.* In addition, many lightning victims with significant sequelae have no evidence acutely of burns. Peripheral neuropathy, chronic pain syndromes, and neuropsychological symptoms, including severe short-term memory difficulty, difficulty processing new information, attention deficit, depression, and posttraumatic stress disorder, may be debilitating.[63,166,167] It is regrettable that victims still feel that these very serious complications are not believed, especially by medical practitioners.

A myth still prevalent is that the lightning victim retains the charge and is dangerous to touch, because he or she is still "electrified." This myth has led to unnecessary deaths by delayed resuscitation efforts.[58]

Medical literature and practice are plagued by myths that grew out of misread, misquoted, or misinterpreted data and continue to be propagated without further investigation. Not the least of these is the tenet that lightning victims who have resuscitation for several hours may still successfully recover. This belief seems to be grounded in the old idea of suspended animation—the concept that lightning is capable of shutting off systemic and cerebral metabolism, allowing rescuers a longer period in which to resuscitate the patient. This concept, credited to Taussig,[185] actually appeared some time before her article. In addition, the case recounted by Taussig that is the basis for this myth, when searched to its source, was a case report by Morikawa and Steichen.[154] The case does show a somewhat longer resuscitation period than usual, but not as miraculous as reported in Taussig's paper or as propagated in subsequent references to her paper.

In a study of lightning survivors, Andrews, Colquhoun, and Darveniza[7] have shown increasing prolongation of the QT interval, bringing up the theoretic possibility of torsades de pointes as a mechanism for the suspended animation reports. There is new evidence from animal experiments to support the teaching that respiratory arrest may persist longer than cardiac arrest.[5,68,69] One study, in which Australian sheep were hit with simulated lightning strokes, showed histologic evidence of damage to the respiratory centers located beneath the fourth ventricle.[5] Prolonged assisted ventilation may in some cases be successful after cardiac activity has returned.[119,122,163]

Another series of animal experiments[68,69] with hairless rats has shown that it is possible to obtain the skin changes (keraunographic markings), primary and secondary arrest with prolonged respiratory arrest, and temporary lower extremity paralysis with simulated lightning strike.

Several booklets listing precautions for personal lightning protection appeared in the late 1700s and early 1800s. One of the superstitions listed was that humans, by their presence, could attract lightning to a nearby object. A book of the times, *Catechism of Thunderstorms*, illustrated other myths. Lightning was said to follow the draft of warm air behind a horse-drawn cart, so that coachmen were cautioned to walk their horses slowly through a storm.

Historically, many remedies for resuscitation of lightning victims have been offered. On July 15, 1889, Alfred West testified in a New York court that he was revived by "drawing out the electricity" when his feet were placed in warm water while his rescuer pulled on Mr. West's toes with one hand and milked a cow with the other.[17]

Other early attempts at resuscitation included friction to the bare skin, dousing the victim with a bucket of cold water, and chest compression. An early attempt at cardiopulmonary resuscitation was given in 1807 when mouth-to-mouth ventilation was used for lightning victims, and it was proposed that gentle electric shocks from galvanic batteries passed through the chest might be successful in resuscitating a victim of lightning.[24] Before that, Benjamin Franklin had purposely electrocuted a chicken during a lightning experiment and reported successful, albeit temporary, resuscitation with mouth-to-beak ventilation.[88]

A myth in current treatment is that lightning injuries should be treated like other high-voltage electrical injuries. Although lightning as an electric phenomenon follows the same laws of physics, the injuries seen with lightning are very different from high-voltage injuries and should be treated differently if iatrogenic morbidity and mortality are to be avoided.[12,57]

"Lightning never strikes the same place twice." In reality, the Empire State Building and the Sears Tower are hit dozens of times a year, as are mountaintops and radio-television towers. If the circumstances facilitating the original lightning strike are still in effect in an area, the laws of nature will encourage further lightning strikes.

Other Myths[58]

1. Victims may have "internal burns." False. There may be cellular damage and certainly nervous system damage but rarely, if ever, internal burns such as those suffered with high-voltage electrical injuries. However, some physicians use this euphemism with patients to explain their pain and neurologic injuries.
2. Wearing rubber-soled shoes, raincoats, sitting on a foam pad or backpack, or the like will protect a person. False. If lightning has burned its way a mile or more through the air, which is an excellent insulator, it is foolish to believe that a fraction of an inch of rubber or that a few inches of any material will serve as an adequate insulator.
3. The rubber tires on an automobile are what protects a person from lightning injury. False. See number 2. Electrical energy travels along the outside of a metal conductor (the car body) and dissipates through the rainwater to the ground or off the axles or bumper of the car.
4. Wearing metal around the head or as cleats on shoes will increase the risk or "attract" lightning. False. There is no evidence to support this. Lightning victims often receive the

*References 2, 8, 49, 53, 56, 63, 156, 161, 166, 167, 181, 188, 199.

impact of a nearby strike as the current traveling across the ground. The current then comes up through the feet, sometimes causing shoes to be blown off. As the current travels across the skin, metal objects along the way are heated as an effect of the strike, but never because they caused or attracted the strike.

5. Carrying an umbrella increases the risk. This is somewhat true if a person's height becomes greater by holding an umbrella but provides an insignificant effect.
6. Lightning always hits the highest object. False. Lightning only "sees" objects about 30 to 50 meters (90 to 150 feet) from its tip. In addition, several pictures exist of lightning hitting halfway down a flagpole or striking in a parking lot next to light poles. Someone standing in the middle of a football field will not be protected by the goalposts if lightning is coming down over his head. Similarly, there are many photographs of lightning branching to meet the earth in several places.
7. There is no danger of lightning injury unless it is raining. False. Although lightning only occurs as a result of thunderstorms, it can travel 16 km (10 miles) or more in front of the thundercloud to strike a person or object long before the rain comes down. Nearly 10% of lightning occurs when there is no rain falling in the area of the strike. It has also been known to reach over a mountain ridge and hit "out of the blue" from the thunderstorm that was on the other side of the peak and was neither visible nor audible to the victim.
8. Lightning may occur without thunder. False. Whenever there is lightning, there is thunder, and vice versa. Sometimes it will appear that there is lightning without thunder because thunder is seldom heard more than 16 km (10 miles) from the lightning stroke or may be blocked by buildings or mountains. In addition, survivors who are very close to the strike may not hear the thunder.

INCIDENCE OF INJURY

Spatial Distribution of Lightning in the United States

The distribution of cloud-to-ground lightning across the United States is well known because of the operation of real-time lightning detection networks. On average, more than 20 million cloud-to-ground flashes are detected each year in the United States.[110,164] On a shorter time scale, more than 50,000 flashes per hour are sometimes detected during summer afternoons over the United States.[73] Existing technology provides detection of at least 90% of all cloud-to-ground flashes within the contiguous United States.

A multiyear climatology of lightning from detection network data shows that central Florida has the greatest number of flashes per area in a given year (Fig. 3-1A). Flash density decreases to the north and west from there. Flash densities over Missouri, Iowa, and Illinois during the 1993 Mississippi River flood rivaled those over Florida.[153] In addition to the general features in Figure 3-1, important local variations occur along the coast of the Gulf of Mexico, where sea breezes enhance lightning frequency.[141,195]

Additional important maxima and minima are found in and around the regions in the western United States with mountains and large slopes in terrain.[141,149]

Lightning Around the World[45,46]

Lightning detection systems similar to the U.S. network have been installed over part or all of more than 40 countries on every continent except Antarctica. Some of these ground-based networks have been in operation for up to 2 decades. However, there is no compilation of cloud-to-ground lightning flashes from such networks covering more than one country at a time. In the last decade, ground-based systems have been developed and deployed that detect the full horizontal and vertical extent of flashes.[77] Flashes detected with such systems are identified as three-dimensional, cloud, in-cloud, or total lightning. Total lightning networks have shown that five or more cloud flashes occur for every cloud-to-ground flash, and cloud flashes can have horizontal extents of 161 km (100 miles) or more. Total lightning networks sometimes show that cloud-to-ground flashes are connected to a complex lightning structure. Cloud flashes do not directly affect people on the ground. However, their wide range of relationships with ground flashes makes understanding ground strikes somewhat more complex than had been the case before these data became available. No ground-based maps of total lightning have been developed to date. However, a general view of worldwide total lightning activity as detected by the satellite-borne Lightning Imaging Sensor (LIS), which measures both cloud-to-ground and cloud flashes,[46] is shown by Figure 3-1B.

Most lightning occurs over tropical and subtropical continents. There is far more lightning over land than over the oceans. Some flash frequencies in South America, southeast Asia, and especially equatorial Africa are much greater than found in the United States over Florida and other Gulf Coast locations. In some tropical and subtropical regions, maximum lightning activity during the year is influenced primarily by the monsoon and coincides generally with the months of heaviest rainfall (Table 3-1).

Temporal Distribution of Lightning in the United States[140,141,196,202]

Lightning is most common in summer months (Fig. 3-2A). About two thirds of flashes occur in June, July, and August. In the southeastern states, lightning occurs quite often during all months of the year. A primary ingredient for lightning formation is a significant amount of moisture in the lower and middle levels of the atmosphere; this fuel for thunderstorms is consistently found in humid subtropical and tropical regions. The other necessary ingredient to produce a thunderstorm is a mechanism to lift the moisture. Along coastlines and mountain slopes during the summer months, updrafts are produced almost daily that provide favored locations for frequent thunderstorms.

Lightning is most common in the afternoon (see Figure 3-2B).[202] Nearly half of all lightning occurs from 1500 through 1800 local standard time (LST). Figure 3-2B combines regional results during the summer in several regions of the United States.[140,141,196] Lightning is at a maximum in the afternoon because the updrafts necessary for thunderstorm formation are strongest during the hours of the day when surface temperatures are highest, resulting in the greatest vertical instability.

U.S. Lightning Casualties in *Storm Data*[44,86,103,148,152,182]

Every month, each National Weather Service (NWS) office in the United States compiles a list of damaging or notable weather

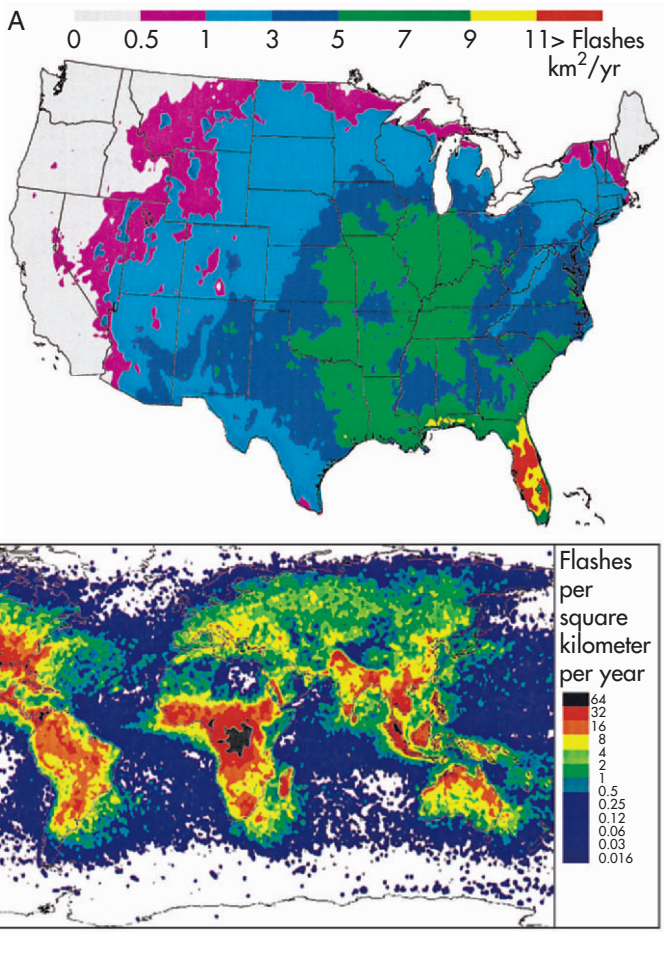


Figure 3-1. **A,** Cloud-to-ground flashes per square kilometer per year in the United States from a network of lightning detection antennas from 1996 to 2000. **B,** Total flashes per square kilometer per year for the world from 12 April 1995 through 1999 from the Optical Transient Detector. (**A** courtesy Vaisala, Inc., Tucson, AZ; **B** courtesy Hugh Christian, NASA/Marshall Space Flight Center.)

TABLE 3-1. Top 10 Areas of Lightning Density in the World Based on the Optical Transient Detector Satellite

LOCATION	FLASHES (km^2/yr)
1. Kamembe, Rwanda	82.7
2. Boende, Democratic Republic of Congo	66.3
3. Lusambo, Democratic Republic of Congo	52.1
4. Kananga, Democratic Republic of Congo	50.3
5. Kuala Lumpur, Malaysia	48.3
6. Calabar, Nigeria	47.3
7. Franceville, Gabon	47.1
8. Posadas, Argentina	42.7
9. Ocana, Colombia	39.9
10. Concepcion, Paraguay	37.0
. . .	
14. Orlando-Tampa, Florida	35.4

Christian HJ: Global lightning activity. Proceedings, 12th International Conference on Atmospheric Electricity, Versailles, France, 2003.

phenomena occurring within the office's area of responsibility. This list is sent to National Oceanic and Atmospheric Administration (NOAA) headquarters and then to NOAA's National Climatic Data Center (NCDC) in Asheville, NC. These lists are combined at NCDC, and then *Storm Data* is published.

Since 1974, *Storm Data* has reported an average of 67 deaths per year from lightning. Table 3-2 shows that lightning was second only to flash floods and floods in weather-related deaths during the 30 years since 1974. Lightning-related casualties and damages are often less spectacular and more dispersed in time and space than other weather phenomena. Therefore, lightning deaths, injuries, and damages have been found to be underreported.[103,148,152,182] Factors contributing to the underreporting include the fact that most casualty events involve only one person or object, the NWS only uses newspaper clipping services to compile *Storm Data,* there is a lack of a uniformly applied definition of lightning versus lightning-related deaths, and there is inconsistency in listing medical diagnoses.[86,152,182]

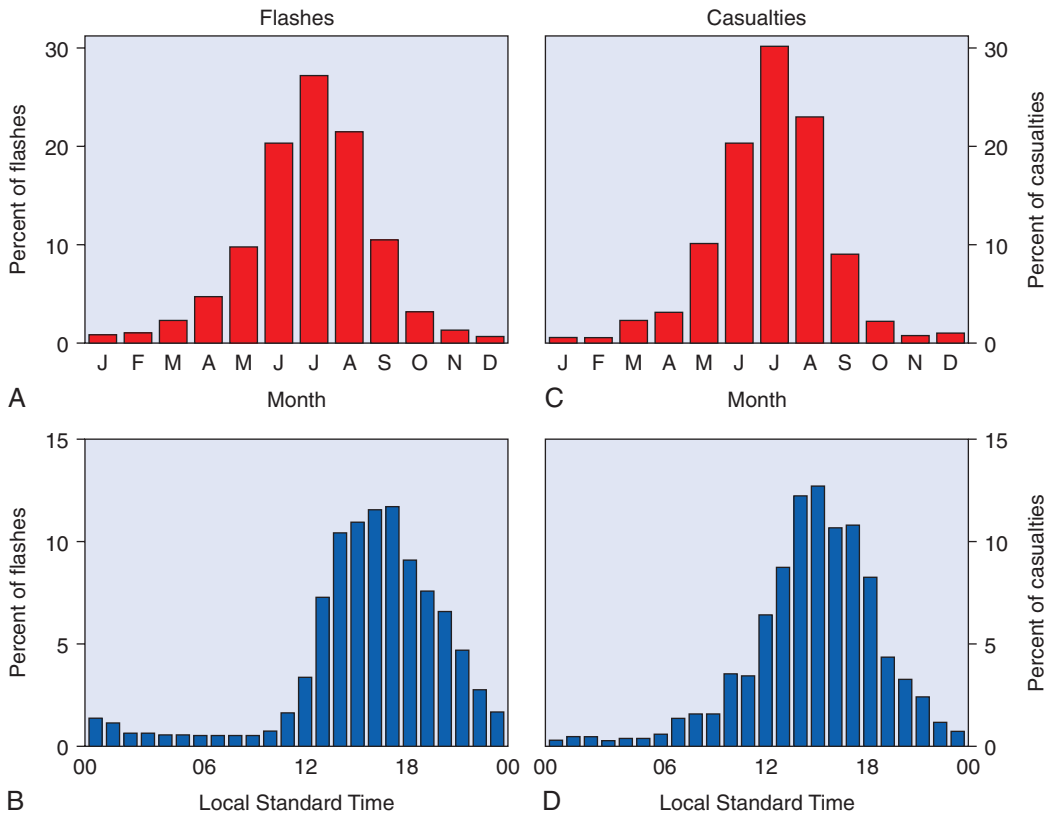


Figure 3-2. **A,** Monthly distribution of U.S. cloud-to-ground lightning from 1992 to 1995 from a lightning detection network. **B,** Hourly distribution of cloud-to-ground lightning from networks in four U.S. locations. **C** and **D,** Monthly and hourly distribution of lightning casualties from 1959 to 1994 in the United States. (**A** from Orville RE, Silver AC: Monthly Weather Review 125:631, 1997; **B** from López RE, Holle RL: Monthly Weather Review 114:1288, 1986; **C** and **D** from Curran EB, Holle RL, López RE: J Climate 13:3448, 2000, with permission.)

TABLE 3-2. Weather-Related 30-Year Average Deaths (1974–2003), and 2003 Weather Casualties

WEATHER TYPE	1974–2003 DEATHS PER YEAR	2003 DEATHS	2003 INJURIES
Flash and river floods	107	86	70
Lightning	67	43	236
Tornado	65	54	1087
Tropical cyclone	14	14	233
Marine		55	53
Winter		37	119
Heat		36	174
High wind		24	156
Cold		20	14
Thunderstorm wind		19	226
Mud slide		15	16
Hail		0	121
Other		35	419
TOTALS	253	438	2924

Order is by deaths from storm types with 30-year records, then by 2003 deaths from other natural hazards. Source: www.nws.noaa.gov/om/hazstats.shtml#.

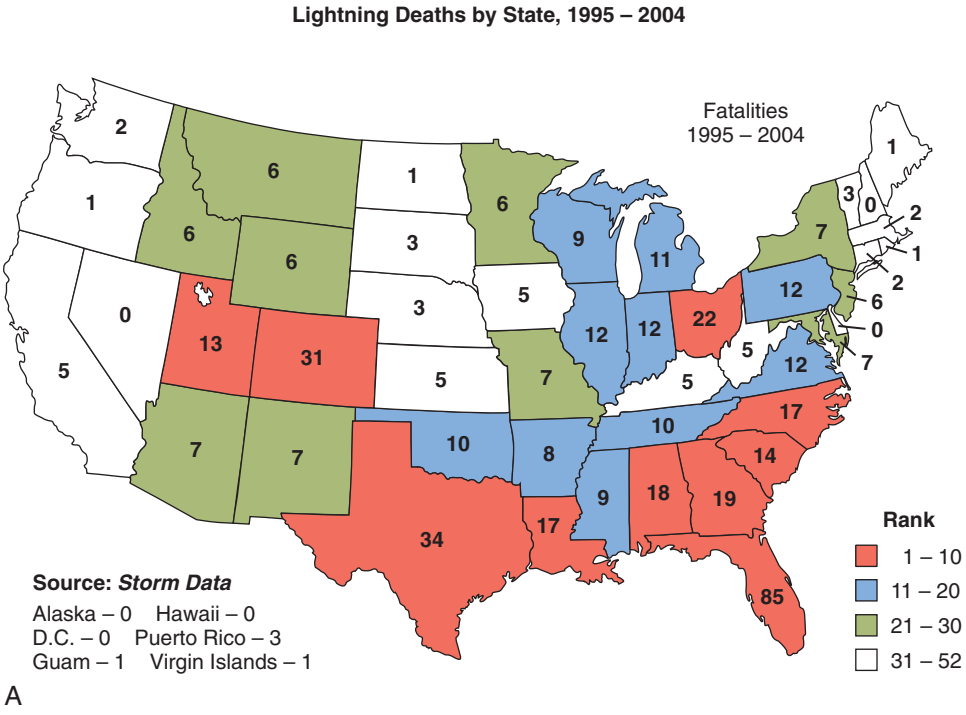
Likewise, nonfatal injuries are also underreported. Even though some studies have shown a mortality rate as high as 20% to 30%, these were retrospective reviews of the medical literature, which tended to overreport more severe injuries. In a thorough search of Colorado hospital and emergency department visits, Cherington and coworkers[44] found that a ratio of 10 injuries to every death was probably more reasonable. There are additional injuries to persons whose visits are not reported by a medical facility or who do not seek immediate treatment.

Even though *Storm Data* includes many property damage reports caused by lightning, it represents an extremely small portion of the actual total.[103] Each *Storm Data* report has some or all of the following: year, month, day, time, state, and county, as well as number, gender, and location of fatalities and injuries, and amount and type of damage. Despite its underreporting of lightning impacts and sometimes incomplete nature, *Storm Data* is a consistent national data source concerning weather impacts across several decades and is likely the best system in the world for collecting weather-related hazard information.

Distributions of Lightning Deaths by State*

Lightning casualty deaths in the United States by state from 1990 to 2003 are shown in Figure 3-3A. The general pattern

*References 48, 74, 82, 83, 86, 94, 109, 127, 142, 147, 152, 165.



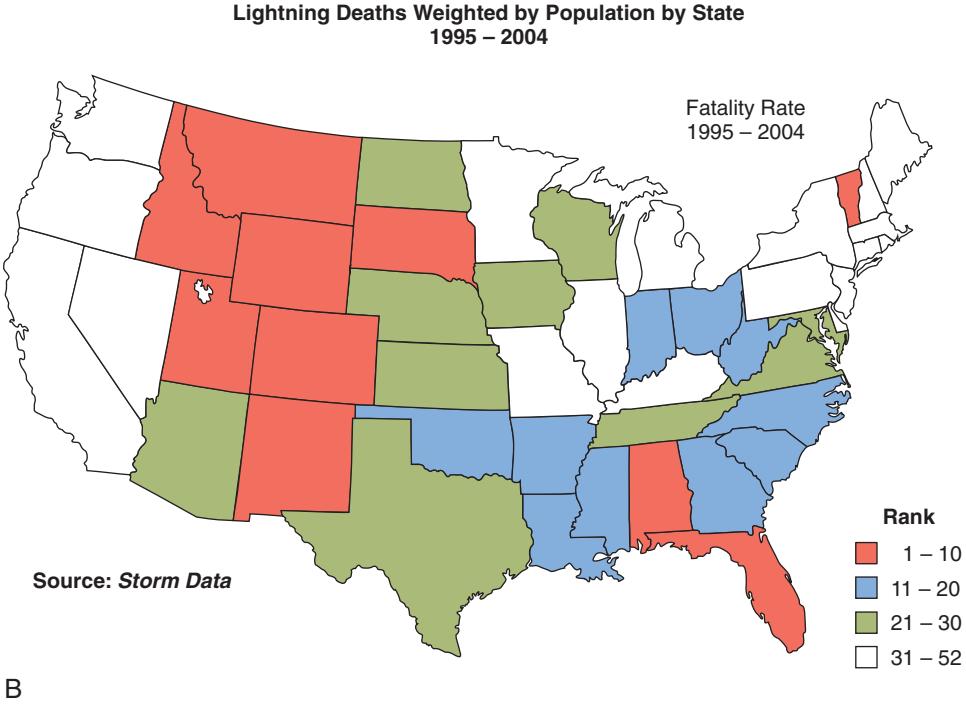


Figure 3-3. Rank of each state in lightning deaths from 1995 to 2004. **A,** Deaths per state. **B,** Deaths weighted by state population. (Updated from Curran EB, Holle RL, López RE: J Climate 13:3448, 2000, with permission.)

has similarities to the distribution of lightning in Figure 3-1, but Florida has twice as many deaths as any other state. Many of the other high numbers of fatalities are from populous states. It is preferable to use deaths for these results because the number of injuries is somewhat unevenly collected.

The lightning hazard is shown better when population is taken into account (see Figure 3-3B). The maximum rate of lightning fatalities shifts away from the more populous states, mainly in the eastern part of the country, to the Rocky Mountain and Great Plains states. Some southeastern states often have high rankings in both deaths and death rates (see Figure 3-3). The only states in the top 10 of both fatality and fatality rate are Florida, Colorado, Alabama, and Louisiana. Detailed state listings of deaths and injuries, and their rates, from 1959 to 1994, are in Curran and coworkers.[74]

Two U.S. lightning fatality studies had similar results to Figure 3-3 for earlier time periods. Duclos and Sanderson[82] used data from the National Center for Health Statistics, and Mogil and coworkers[152] used *Storm Data*. Single-state maps by county were compiled for Florida,[82,83] North Carolina,[127] Michigan,[86] and Colorado.[142,147] Outside the United States, studies showing the spatial distribution of fatalities by political boundaries have been developed for Canada,[109] Singapore,[165] Australia,[48] and France.[94] Many additional studies have included national totals over periods from several to many years.

Monthly Variations of U.S. Casualties*

By month, lightning casualties peak during July (see Figure 3-2C). The percentages increase gradually before July, then decline more quickly after the maximum. Cloud-to-ground flashes show similar features (see Figure 3-2A). Summer maps of lightning casualty rates in works by Curran and coworkers[74] are similar to annual maps. During other seasons, casualty rates are higher in southern states. Casualty rates in the northeast are low except during the summer, whereas they are highest on the West Coast during autumn and winter.

A July maximum was also found in prior *Storm Data* studies, as well as a slower increase before and a faster decrease after July.[82,83,86,142,147,152] August maxima were found in Florida by Duclos and coworkers[82] and Holle and coworkers.[107] Away from the tropics, most casualties occur during summer months. For example, January has the largest number of Australian fatalities due to the reversal of seasons from the Northern Hemisphere.[48] In the equatorial location of Singapore, fatality maxima in November and April are similar to the annual cycle of local thunderstorms.[165]

Time-of-Day Variations of U.S. Casualties[74,82,86,147]

Most lightning casualties occur in the afternoon (see Figure 3-2D); two thirds occur between 1200 and 1800 LST. They show a steady increase toward a maximum at 1600 LST, followed by a slower decrease after the maximum. Lightning flashes in Figure 3-2B showed a faster increase to the afternoon maximum than shown for casualties. Lightning occurs most often in the afternoon because the ground is heated most strongly by the sun during that time period. As a result, vertical cumulus clouds form and produce lightning when they are tall enough to have tops colder than freezing temperatures. Narrower distributions of casualties centered in the afternoon are apparent in the Rockies, Southeast, and Northeast compared with the broader time series in the plains and Midwest.[74]

*References 48, 74, 82, 83, 86, 107, 142, 147, 152, 165.

TABLE 3-3. Casualty Information in *Storm Data* from 1959 to 1994

TOPIC	DEATHS (%)	INJURIES (%)	CASUALTIES (%)
Males	84	82	83
One victim per event	91	68	68

In the evening and at night (1800 to 0559 LST), casualties are most frequent in the plains, upper Midwest, and some populous eastern states.[74] More than half of the deaths from 0000 to 0559 LST occurred when people were in a house set on fire by lightning, and 21% occurred when people were camping in tents.

Outside of the summer months, lightning casualties are distributed more widely through the day. Lightning is less concentrated during the afternoon in those months because the ground is not heated as much as in summer. As a result, more thunderstorms are formed by large-scale traveling disturbances during all hours of the day and night.

Maximum lightning impacts from 1400 to 1600 LST were documented by Duclos and Sanderson,[82] Ferrett and Ojala,[86] and López and Holle.[147] Duclos and Sanderson[82] found an 1800 LST peak in North Carolina deaths.

Gender and Number of Casualties†

Table 3-3 shows that males are much more frequent lightning casualties than are females. Similar ratios were found in the U.S.,[82,83,107,127] Singapore,[165] and England and Wales.[84] During the recreation activities of soccer, baseball, golf, and camping, the male percentage of casualties was as low as 57% during camping to 94% during golf.[101] The camping rate was 76% male, if a South Africa event with 23 Girl Scouts and adult leaders is omitted.[1,26]

The most common situation was for only one victim to be involved in a lightning incident. This fact is an important contributor to the underreporting of lightning casualties.[148] The largest single death total in one U.S. event resulted from the 1963 crash of an airliner in Maryland that killed 81 people. The largest number of injuries at one event was 90 at a Michigan campground.[86] The same tendency for single victims was noted in the United States,[147] Singapore,[165] and Australia.[48]

20th Century Trends in U.S. Lightning Deaths[48,82,84,92,152,165]

The number of lightning deaths reported since 1900 is shown in Figure 3-4A. During the first 20 years, annual reported deaths increased from less than 100 to about 450 because of an increase in reporting states. During the 1920s and 1930s there were about 400 lightning deaths per year, whereas recently it has been less than 75 per year. There has been a persistent drop in deaths since 1944. The same dramatic decline since 1940 was noted by others.[82,152]

The effect of changes in population is taken into account by dividing by the population. Figure 3-4B shows lightning deaths per million people per year. In the earlier part of the series, year-to-year fluctuations are relatively large as reporting states with different demographic and climatic conditions were included. Since 1925, the fluctuations are consistently smaller and more

†References 1, 26, 48,82–84, 86, 101, 107, 127, 147, 148, 165.

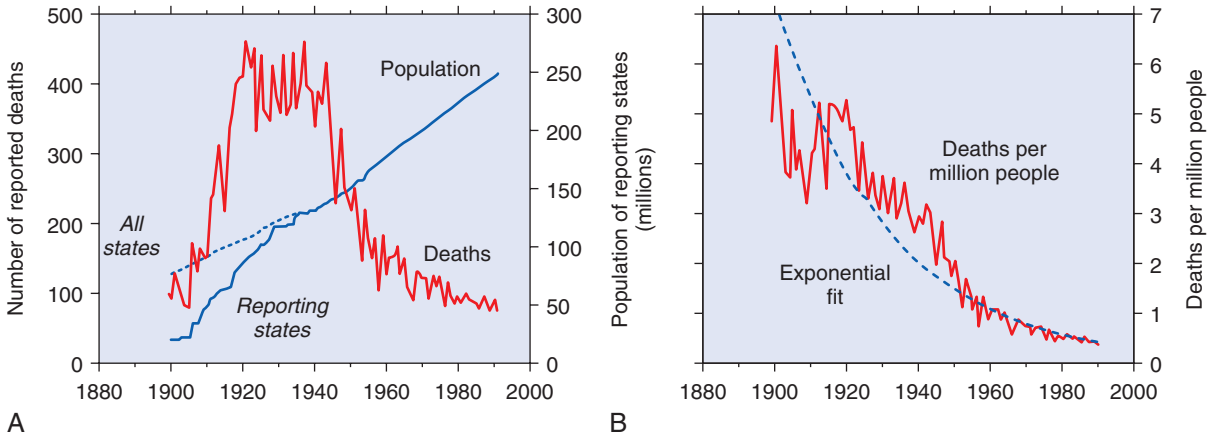


Figure 3-4. **A,** *Red line* is annual lightning deaths reported by Bureau of the Census and Public Health Service from 1900 to 1991. *Dashed blue line* is total population of United States and District of Columbia; *solid blue line* is population of reporting states and District of Columbia. **B,** *Solid red line* is adjusted yearly lightning deaths normalized by population. *Dashed blue line* is percent of population in rural areas. (From López RE, Holle RL: J Climate 11:2070, 1998, with permission.)

regular as the death rate decreases. The normalized time series indicates a decrease during the 20th century from more than six at the start to as low as 0.4 deaths per million people late in the century.

Notable decreases in deaths were also documented with long-term data sets in England and Wales,[84] England and Wales compared with Australia,[92] and Singapore.[165] Australian deaths increased during the years 1825 to 1918, then decreased through 1991.[48]

Effect of Rural-to-Urban Migration*

Before the turn of the last century, lightning deaths appeared to occur often in rural settings.[105] Since then, the percentage of the U.S. population in rural areas (but not the actual population) has dramatically decreased.[144,145] Figure 3-4B shows that the percentage of the population living in rural areas since 1890 decreased from 60% in 1900 to 25% in 1990. Departures from the exponential decrease are seen as a slowing in the 1930s and early 1940s during the Great Depression, and an acceleration of the trend in the 1950s and 1960s with increased urbanization after World War II and the Korean War.*

The adjusted normalized lightning-death plot is superimposed on the rural population curve in Figure 3-4B. The remarkable agreement leads to a conclusion that the decrease in population-adjusted deaths is closely related to the relative reduction in rural population. This link between long-term decreases in both lightning deaths and rural population has been hypothesized, as well as being attributed to improved home electrical systems that include substantial grounding, and improved medical treatment and communications, education, and meteorologic warnings.[83,143,147,152] This trend has been replicated with normalized population data from Canada and Spain.[62] The trend toward fewer lightning deaths in the United States since 1959 is divided into three segments in Fig. 3-5, which shows a steady decrease in fatality rate from 1959 to 1978, and two populations of lower fatality rates in the 1980s and since 1991. There were 20% more deaths reported in the National Center for Health Statistics than in *Storm Data*.[135] The most recent decreases could correspond with efforts in lightning safety education beginning in the early 1990s, but this factor is difficult to isolate.[64,65,135,136]

*References 62, 64, 65, 82, 105, 135, 136, 143–145, 147, 152.

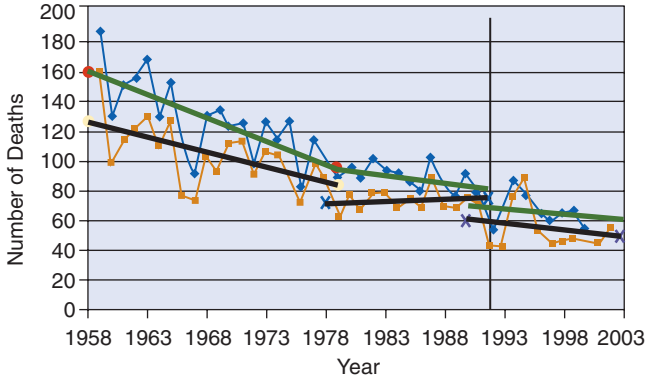


Figure 3-5. *Blue plot* shows annual lightning deaths from National Center for Health Statistics; *orange* is from National Oceanic and Atmospheric Administration's *Storm Data. Green and black lines* are regression curves that show a general rapid decrease from 1959 to 1978, a lower mean with few changes in the 1980s, then another drop without much change in the 1990s. (From Lengyel MM: Thesis, Master of Science in Meteorology, University of Oklahoma, Norman, OK, 2004, with permission.)

Types of Lightning Casualty Incidents[33,48,101,105,124,142,147]

The preceding analyses suggest a link between the shift from rural to urban settings and the number of lightning casualties. Kretzer[124] documented lightning deaths and injuries from 1891 to 1894, and an analysis was made of entries in *Storm Data* 100 years later (Fig. 3-6).[105]

In the 1890s, rural deaths were much more frequent than urban. Indoor fatalities were the most frequent; 23% of all deaths were inside houses. The next largest types were outdoors and agricultural incidents, whereas recreation and sports incidents were virtually nonexistent.[105]

In the 1990s, rural settings and agricultural incidents were much less frequent. Only 2% of modern deaths were attributed

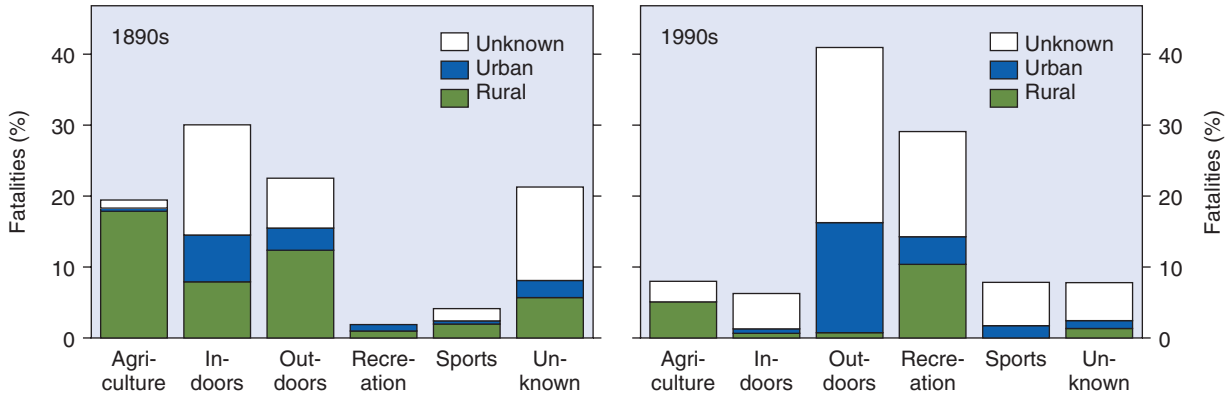


Figure 3-6. Types of U.S. lightning casualty incidents (%) from 1891 to 1894 compared with 1991 to 1994 (From Holle RL, López RE, Navarro BC: J Appl Meteorol 44:1563, 2005, with permission.)

to people inside houses, one tenth the percentage of a century earlier. The largest percentage of incidents occurred outdoors, often under or near trees (15% of all lightning deaths) and in the yard of a house. A high percentage also occurred during recreation, especially in beach, water, and camping situations. There also have been many more sports incidents in recent years than a century ago.[105] Specific recreation incidents have been summarized for recent years during soccer, baseball and softball, golf, and camping and tent situations around the world.[33,101]

These comparisons agree with the influence of the rural-to-urban migration on lightning casualties in the United States. Rural casualties are now half as frequent as urban cases. Being inside of a house is no longer as much of a risk for lightning strike. This trend is most likely a result of grounding by modern wiring and plumbing. Recreation and sports have become relatively greater contributors to the population at risk from lightning injury.[48,142,147]

Worldwide Lightning Fatalities[102]

Extrapolation of these results to the world can be considered with caution. The U.S. lightning death rate early in the 20th century exceeded 6 deaths per million people (see Figure 3-4B), whereas the rate is now less than 1 death per million. Earlier lightning deaths often occurred in agricultural incidents in rural settings or inside buildings before widespread installation of wiring and plumbing. The recent rates also can be considered typical of much of Europe and other industrialized, urbanized countries.

However, many people in the populous tropical and subtropical areas of Africa, South America, and Southeast Asia, including China and the Indian subcontinent, continue to rely on labor-intensive agriculture and live in dwellings with minimal or no grounding, and are serviced by less developed infrastructures for medical response and meteorologic information. The earlier rates from the United States could be considered to be appropriate in these regions, where lightning is generally as frequent as in the United States. In fact, some of these areas have higher flash densities than the Florida maximum in the United States (see Table 3-1).[45,46] Although there is almost no systematic information on lightning deaths in these regions, frequent reports of multiple lightning casualties per event show that rural and agricultural events continue to be dominant settings of casualties, together with some recreational cases involving soccer. When these regions with a population of more than 4 billion people are considered, and the rate of 6 deaths per million people per year is applied, a total of 24,000 lightning deaths per year is obtained.[102] If the ratio of 10 injuries for every death is applied,[44] 240,000 people are injured by lightning per year in these regions.[102]

EARLY SCIENTIFIC STUDIES AND INVENTION OF THE LIGHTNING ROD[47,87,88,170]

The study of electric phenomena is often traced to the publication of Gilbert's De Magnete in London in 1600. Experiments in France and Germany and by members of the Royal Society of London led to the invention of the Leyden jar in 1745.

Benjamin Franklin is generally regarded as the father of electric science and during his lifetime was known as the American Newton. The reason he was accepted into the French and English courts around the time of the American Revolution was not because he was an ambassador from America but because he was considered to be one of the foremost scientists of his time. Franklin was elected to every major scientific society at the time and received medals of honor from France and England for his scientific contributions.[47,170]

Before his work, it was thought that two distinct types of electric phenomena existed. Franklin's work unified these two forces, and he is responsible for renaming them as positive and negative charges.[88] He also proved with numerous experiments that lightning was an electric phenomenon and that thunderclouds are electrically charged, as demonstrated by the famous kite and key experiment.[87] He invented the lightning rod and announced its use in 1753 in *Poor Richard's Almanack:*

> *It has pleased God in his Goodness to Mankind, at length to discover to them the Means of securing their Habitation and other Buildings from Mischief by Thunder and Lightning. The Method is this: Provide a small Iron Rod (It may be made of the Rod-iron used by the Nailers) but of such a Length, that one End being*

three or four Feet in the moist Ground, the other may be six or eight Feet above the highest Part of the Building. To the upper End of the Rod fasten a Foot of brass Wire the Size of a common Knitting-needle, sharpened to a fine Point; the Rod may be secured to the House by a few small Staples. If the House or Barn be long, there may be a Rod and Point at each End, and a middling Wire along the Ridge from one to the other. A House thus furnished will not be damaged by Lightning, it being attracted to the Points, and passing thro the Metal into the Ground without hurting any Thing. Vessels also, having a sharp pointed rod fix'd on the Tops of their Masts, with a Wire from the Foot of the Rod reaching down, round one of the Shrouds, to the Water, will not be hurt by Lightning.

In the 1750s and 1760s, the use of lightning rods became prevalent in the United States for protection of buildings and ships. Some scientists in Europe urged the installation of lightning rods on government buildings, churches, and other tall buildings. However, religious advocates maintained that it would be blasphemy to install such devices on church steeples, in that the churches received divine protection. Because of this divine protection, some towns chose to store munitions in their churches, leading on more than one occasion to significant destruction and loss of life when the churches were hit by lightning.

Part of the delay in installing lightning rods in England may have been due to British distrust of the scientific theories originating in the upstart, newly independent United States. Years and numerous unsuccessful trials with English designs were required before the Franklin rod became accepted on Her Majesty's ships and buildings.[24]

At one time, lightning rods were theorized to be diffusers of electric charges that could neutralize a storm cloud passing overhead, thus averting a lightning stroke. This theory was in part an outgrowth of the observation of St. Elmo's fire, an aura appearing around the tip of lightning rods and ships' masts during a thunderstorm. This phenomenon is caused by an electron discharge that results from the strong electromagnetic field induced around the glowing object.

Properly installed lightning rods and lightning protection systems do not "attract" lightning, but rather protect a building by allowing the current from a lightning strike that would have occurred, regardless of the protection system, to flow harmlessly through the system to the ground instead of into or through the building, which often causes more extensive damage.[90,91] It has not been uncommon for charlatans to take advantage of the fear of lightning and the danger of lightning-caused fires. In the past, they drove from farm to farm offering to "discharge" the lightning rods on barns and homes for a fee. Lightning protection still remains an area of controversy, with only some of the lightning codes and protective devices verified by objective research. Some of the recent codes (written by the lightning protection industry) now do more to protect buildings and shelters but unfortunately may actually increase the risk for those seeking "shelter" in bus, pool, rain, or golf types of structures, not only by increasing the chances of side-flash from lightning protection downconductors, but also by increasing the sheltered person's effective height. Systems that claim to "predict" where and when lightning may strike an object, rather than detect the flash, have yet to have the scientific validity of their technology proven. The public is recommended to follow the caveat emptor principle, whether it applies to protection of shelters or detection and prediction of lightning. It remains salutary that the most effective protection still centers around the simple Franklin rod.

The first Lightning Rod Conference was held in London in 1882. Recommendations from this conference were published that year and again in 1905. Further progress in the study of the properties of lightning came with development of Sir Charles Vernon Boy's rotating camera and Dufour's high-speed cathode ray oscillograph, which helped delineate physical properties of lightning, including direction and speed of the strokes.

Certain countries developed codes of practice for lightning protection (Germany, 1924; United States, 1929; Britain, 1943; British colonies, 1965).[13] A variety of materials, including copper, aluminum, and iron, are recommended by these codes, which also specify the measurements and construction of the protective system, depending on the height, location, and construction of the structure to be protected. The most recent U.S. code revision was the National Fire Protection Act of 2004, written by lightning protection practitioners. Lightning strokes vary in power and frequency, depending on terrain and geographic location.[126] Complicated formulas have been devised to take into account relative frequency of strikes in an area; height, construction, and design of the building; and degree of protection desired, depending on whether it is a storage shed, house, school, hospital, or munitions factory.[118]

A lightning protection system should be designed to take into account these factors plus the economic considerations of construction. Including a system in the initial design and construction is always easier and less expensive than modifying a completed building. In addition, except where required by code, the owner may decide that a lightning protection system is not worth the expense, for example, for a mountain retreat that is seldom visited.[118,126] An excellent noncommercial source for discussion of these risks is www.lightningsafety.com.

PHYSICS OF LIGHTNING STROKE

Lightning Discharge[1,60,91,125,187,193]

The study of lightning discharge and formation is extremely complex and involves an entire branch of physics and meteorology. We therefore illustrate here the simplified and most common mechanism of thundercloud formation and lightning strike.

Thunderstorms can be created by a number of factors that produce vertical updrafts. These ingredients are usually caused by cold fronts, large-scale upward motions, sea and lake breezes, lifting by mountains, and afternoon heating of warm, moist air (Fig. 3-7A).[91,187]

As warm air rises, turbulence and induced friction cause complex redistribution of charges within the cloud (see Figure 3-7B). Water droplets and ice crystals within the cloud acquire and increase their individual charges. A complex layering of charges, with large potential differences between the layers, results from the interaction between charged particles and internal and external electrical fields within the cloud.

Generally, lower layers of the thundercloud become negatively charged relative to the earth, particularly when the storm occurs over a flat surface. The earth, which normally is negatively charged relative to the atmosphere, has a strong positive

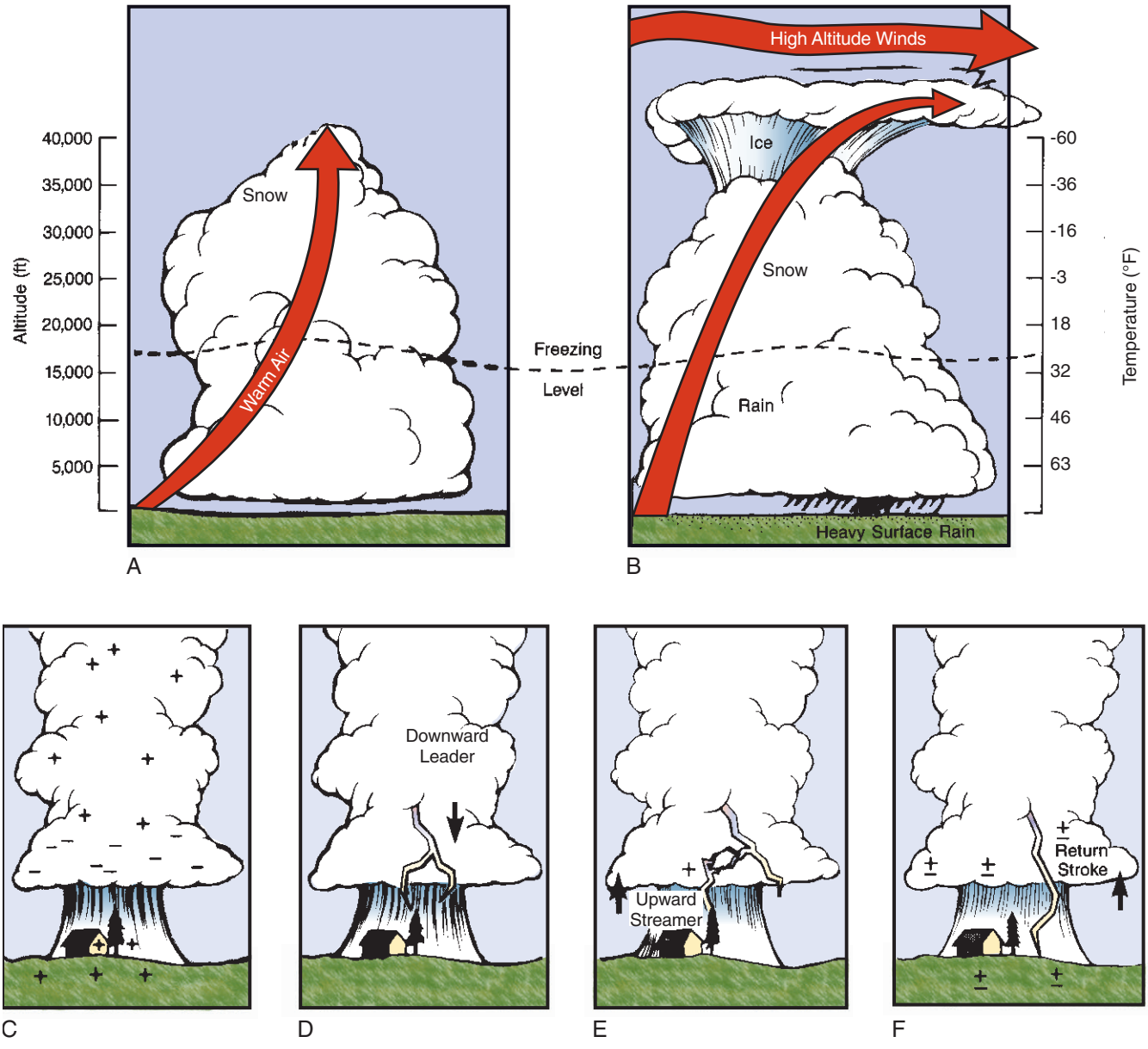


Figure 3-7. **A,** Air rises and condenses into a cumulonimbus cloud. **B,** Typical anvil-shaped thundercloud. **C,** Water droplets within the cloud accumulate and layer charges. **D,** Stepped downward leader initiates the lightning strike. **E,** Positive upward streamer rises from the ground to meet the stepped leader. **F,** Return stroke travels back up the channel made by the stepped leader.

charge induced as the negatively charged thunderstorm passes overhead. The induced positive charge tends to flow as an upward current into trees, tall buildings, or people in the area of the thunderstorm cloud and may actually course upward as "upward streamers."[1,60]

Normally, discharge of the potential difference is discouraged by the strong insulatory nature of air. However, when the potential difference between charges within the clouds or between the thundercloud and ground becomes sufficient, the intervening air may break down under the influence of the electric field created and the charge may be dissipated as lightning.

A lightning stroke begins as a relatively weak and slow downward leader from the cloud (see Figure 3-7C). Although the tip of the leader may be luminous, the stepped leader itself is barely discernible with the unassisted eye. The leader travels at about one-third the speed of light (1×10^8 m/sec), and the potential difference between the tip and the earth ranges from 10 to 200 million volts. The leader ionizes a pathway that contains superheated ions, both positive and negative, thus forming a plasma column of very low resistance. It travels with relatively short branched steps, going down about 50 m (150 feet) and then retreating upward. The next time it goes down, it fills the

original ionized path but branches at the end to go down another 50 m (150 feet) and then retreat again. This up-and-down, polybranching process continues until the leader comes to within 30 to 50 m (100 to 150 feet) of the ground. Because lightning follows this ionized path, its tip "sees" only objects within about a 30- to 50-m (100- to 150-foot) radius, meaning that the hill or tower 60 m (200 feet) away from a person will not be "seen" by the lightning as a potential target.

As the tip of the lightning gets closer to the earth with the large potential at its tip (Fig. 3-7D), more concentrated areas of induced charge accumulate on the earth, particularly at the peaks of tall, relatively sharp objects. Several upward streamers (see Figure 3-7E) may rise vertically from these objects toward the downward leader head. Ultimately one, or a small number, of the upward streamers will contact the downward leader, thus completing a lightning channel of low resistance between cloud and ground. The process of the downward leader joining with the upward streamers is called *attachment* and occurs when the tip of the stepped leader is within the "striking distance" of the point of attachment. There is often more than one point of attachment to the ground.[125]

As the low-resistance channel is formed by attachment, the potential difference between cloud and ground effectively disappears and the energy available is dissipated in an avalanche of charge between cloud and ground. This avalanche is referred to as the *return stroke* (see Figure 3-7F) and is highly luminous. Lightning is therefore a current, or charge dumping, phenomenon rather than a voltage phenomenon, and is analyzed accordingly. Subsequent to the discharge through the return stroke, the channel remains attached for a small amount of time, and with quick redistribution of charge from other regions of the cloud to the top of the channel (via J- and K-intracloud streamers), further return strokes may occur. A lightning flash may be made up of multiple strokes (1 to 30, mean 4 to 5) and is perceived by the eye as flickering of the main channel.

When a very tall building is involved, or when high mountains rise into the clouds, the leader stroke may initiate from the building or mountain rather than from the cloud. In such cases, a joining stroke is rarely seen initiating from the cloud. The channel of ions formed by the leader stroke is maintained as a continuous stroke, as the return stroke (misnamed in this instance) travels in the same direction from the ground or object to the cloud, dissipating the charge difference.

The tip of the downward leader is the most luminous of the sequence of strokes in each lightning discharge, in that a huge amount of energy must be expended to overcome air resistance and ionize a channel. Because of the relative slowness and brilliance of the leader, lightning is perceived as traveling from the cloud to the earth, although the vast majority of energy is actually dissipated in the opposite direction with the return strokes. The direction of the return stroke is not visually perceived because of its tremendous speed and is recognized merely as an instantaneous brightening or flickering of the ionized pathway. Lightning may vary in apparent color due to the amount and type of particles in the air between the observer and the lightning channel, from the excitation of nitrogen atoms in the atmosphere, or because the particles of dust through which the lightning passes are high in ion or mineral content.

Diameter and Temperature of Lightning[91,125,187]

Many techniques could be used to measure the diameter or temperature of the lightning stroke. Unfortunately, all measurement techniques have artifact problems. Visual measurement of the stroke using standard photography usually shows the diameter of the main body of the stroke to be about 2 to 3 cm.

The diameter of the arc channel is sometimes measured indirectly, using measurements of holes and strips of damage that lightning produces when it hits aluminum airplane wings, buildings, or trees. Measurements vary from 0.003 to 8 cm, depending on the material destroyed, with hard metallic structures sustaining smaller punctures than do relatively softer objects, such as trees. The ionized sheath around the tip of the bright leader stroke has never been measured but is estimated to be 3 to 20 m (10 to 66 feet) in diameter.

The temperature of the lightning stroke varies with the diameter of the stroke and has been calculated to be about 8000° C (14,000° F). Others estimate the temperature to be as high as 50,000° C (90,000° F). In a few milliseconds the temperature falls to 2000° to 3000° C (3600° to 5400° F), that of a normal high-voltage electric arc.

Forms of Lightning[12,24,77,171,180,190]

Lightning can be divided into cloud-to-ground and cloud (intracloud) flashes (Fig. 3-8). Cloud-to-ground flashes contact the surface of the earth at one or more locations, depending on the number and type of return strokes. Cloud lightning can travel between clouds, within clouds, from cloud to cloud, and in all combinations of these paths. The same flash can both (1) strike the ground at one or more places and (2) travel a long distance in clouds. Numerous single continuous flashes lasting 1 to 2 seconds have been measured to exceed 100 km (62 miles) in length.[77] There are several times as many cloud flashes as reach the ground. From the point of view of a person on the ground, cloud flashes may appear to travel in long lines streaking across the sky, or be totally within the clouds.

The most unusual, least understood, and least predictable type of lightning is ball lightning.[190] Ball lightning is usually described as a softball-sized orange to white globe. It may enter a plane, ship, or house, travel down the hallway, injure some people and objects and not others that it encounters, and exit

Figure 3-8. Example of cloud-to-ground and cloud lightning. (From Krider EP, Ladd CG: Weather 30(3):77–81, 1975, with permission.)

out another door, chimney, or window, explode with a loud bang, or exhibit other bizarre behavior.[12,24,180,190]

Lightning may be either positive or negative in charge.[171] Cloud-to-ground lightning flashes usually lower negative charge to ground. Positive cloud-to-ground flashes tend to occur during the winter, at the end of thunderstorms, and in relatively shallow thunderstorms. It is not known if positive lightning has a different injury profile. Positive lightning may be more likely to occur when there is particulate matter in the air, including smoke from forest fires. Laboratory positive flashes are more erratic than negative flashes.

Thunder[91,104,187,189]

Thunder is formed when shockwaves result from the almost explosive expansion of air heated and ionized by the lightning stroke.[91,187] The following are accepted statements:

1. Cloud-to-ground lightning flashes produce the loudest thunder.
2. Thunder is seldom heard over distances greater than 16 km (10 miles).
3. The time interval between the perception of lightning and the first sound of thunder can be used to estimate distance from the lightning stroke.
4. Atmospheric turbulence reduces audibility of the thunder.
5. Wind, rain, manmade noise, and vegetation, as well as hills and mountains, can reduce audibility of the thunder.
6. The pitch of thunder deepens as the rumble persists.

The thunder clap from a close lightning flash is heard as a sharp crack. Distant thunder rumbles as the soundwaves are refracted and modified by the thunderstorm's turbulence.[187] Because there is a large difference between the speed of light and the speed of sound, the distance to lightning can be estimated by a person on the ground. The estimation is made by the flash-to-bang method of counting the seconds between seeing a flash and hearing the thunder from the same flash. The time interval between light and thunder is 5 seconds per mile, which is the same as 3 seconds per kilometer.[104,189] For example, if the difference is 30 seconds from when a flash is seen until its thunder is heard, the flash is 6 miles away (10 km). This distance is found by taking the fact that thunder travels a mile every 5 seconds, so a flash that is 6 miles away takes 30 seconds to reach an observer.

MECHANISMS OF INJURY BY LIGHTNING[9,12,28,56]

Concepts in Electrical Mechanisms

A voltage can be applied to a body. It can be regarded as the pressure applied to a body to force it to conduct electric current, like water pressure applied at one end of a pipe to cause water flow. It is an external force, and after it is applied, a current (amps) flows as a result through the body, like the water in the pipe. The amount of current is inversely proportional to the resistance of the body, that is, for a given applied voltage, the higher the resistance of the body, the smaller the resulting current.

Resistance is a given property of a body. A piece of metal has a predictable and exact resistance, for our purposes invariable. It might be thought of as analogous to the diameter of the water pipe, or friction within the pipe. The resistances of various tissues have been measured and characterized as properties of the tissues concerned. If it is assumed that resistance is predictable, then resistance, voltage, and current can be related linearly, making modeling possible. Voltage, current, and resistance are related by Ohm's law. However, tissue resistance is not constant, but is affected by a number of factors that do not depart from the principles of linear analysis. Alterations in tissue resistance are predictable, so analysis can proceed on a "piece-wise linear" basis.

Alternatively, if a current is forced to flow through a body of a given resistance, as is the case with lightning, a voltage directly proportional to the resistance of the body can be measured across the body. That is, for a given current forced through a body, the higher its resistance, the higher will be the measured voltage. The relationship is governed by Ohm's law. The terms *voltage* and *potential* tend to be used interchangeably.

The remaining concept is that of an electric field. If we consider an air gap, we can apply a voltage to that air gap. Air has a known resistance, which is generally high. Thus air only conducts a tiny current. In time, the air "breaks down," and an avalanche of current flows across the gap. A large noisy flashover is the observed result. When in time this occurs varies with the voltage applied and the width of the gap, whether it is electrical charge built up from walking across a carpet in the winter or the electrical energy built up in a cloud. The gap is submitted to the electrical stress of an electric field. The electrical field is defined as the voltage across the gap divided by its width. Flashover in air occurs at about 4000 V/cm or 10,000 V/inch.

Electrical Mechanisms of Injury

Lightning is dangerous because of electrical effects, heat production, and concussive force. In addition, lightning may injure indirectly via forest fires, house fires, explosions, or falling objects such as trees. Only injuries directly caused by lightning are discussed here.

Many factors contribute to how lightning injures a person, and there may be interplay between these at any given time. Exact prediction in an individual case is impossible.

The later discussion on pathophysiology states that, when lightning current is injected into an individual, the current is initially transmitted directly through the individual. Then, as internal structures (capacitances) charge, flashover occurs over the surface of the individual, and internal current reduces dramatically.[119,122,163]

Lightning current may initially be inflicted on a person in one of several ways, described in more detail later (Box 3-2 and Fig. 3-9). In each of these mechanisms, the processes indicated by Kitigawa and coworkers can occur[119,122]:

Box 3-2. Mechanisms of Lightning Injury

Direct strike
Contact potential
Side flash, sometimes called "splash"
Step voltage (EPR)[193]
Ground current
Surface arcing
Upward streamer current[1,98]
Blunt or concussive injury

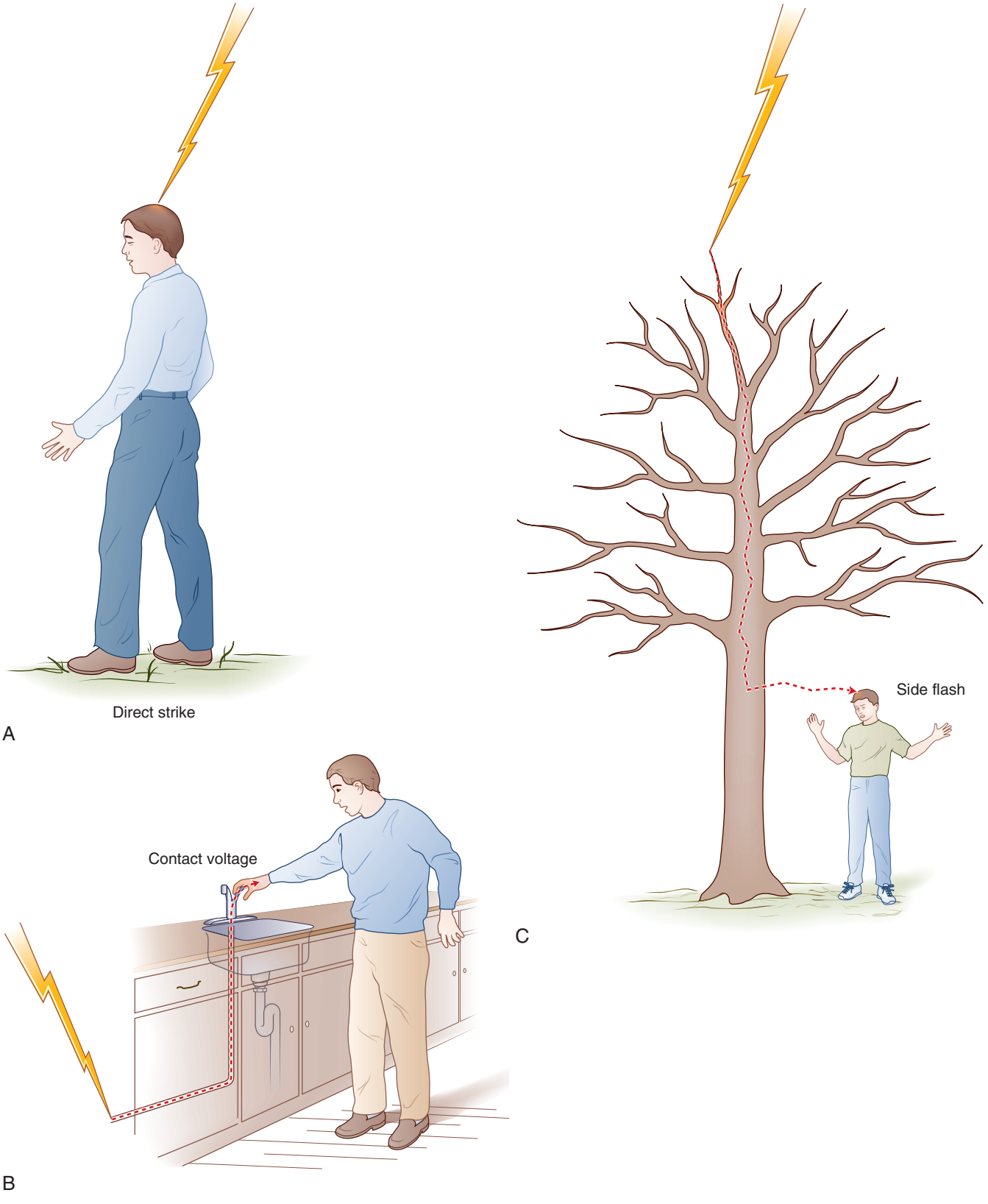
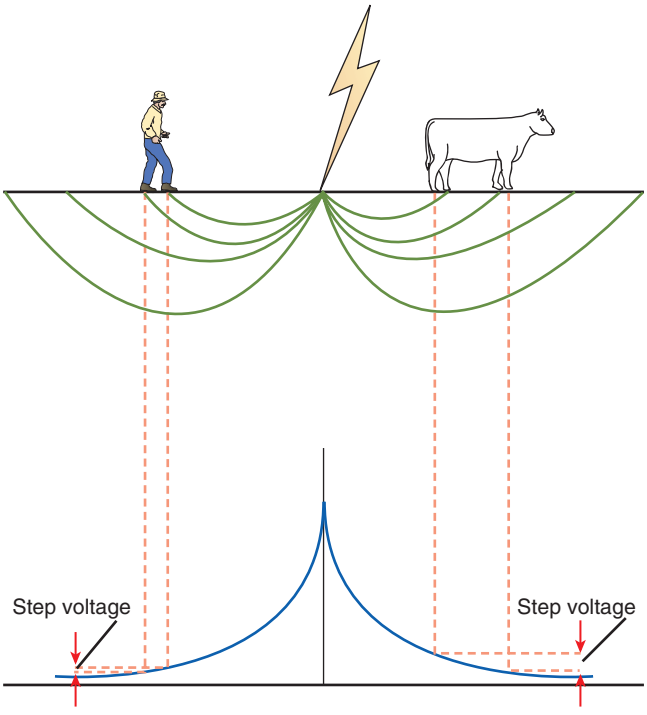
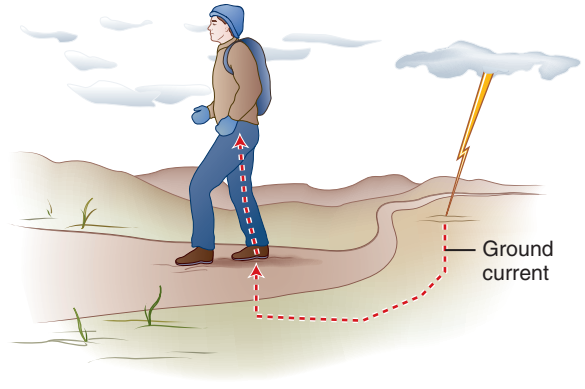


Figure 3-9. Mechanisms of lightning current. **A,** Direct strike. **B,** Contact potential. **C,** Side flash.



D1



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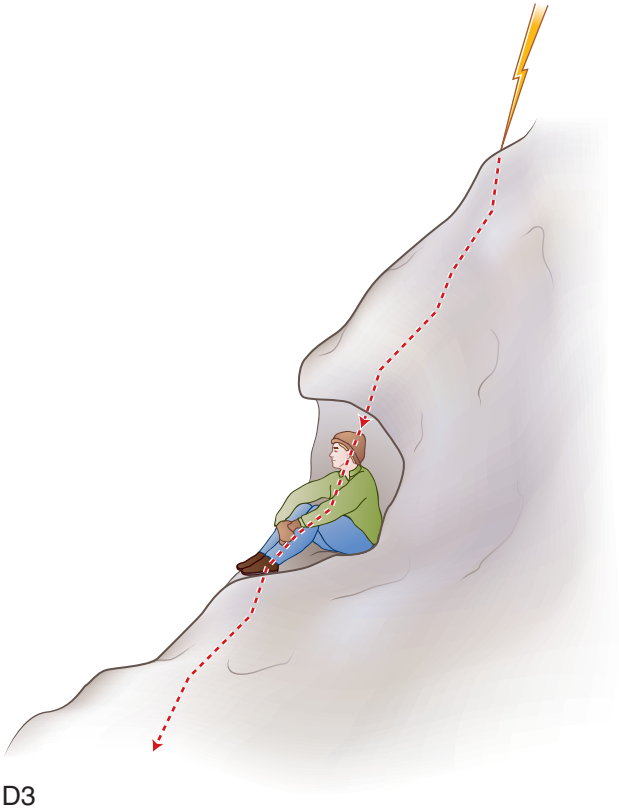

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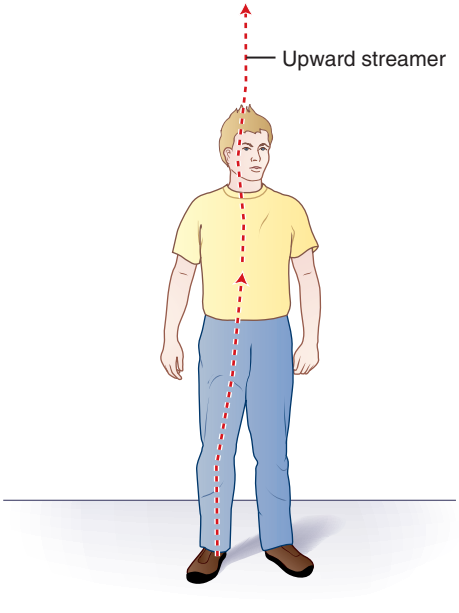


E

Figure 3-9 cont'd. **D.** Earth potential rise. **E.** Upward streamer.

1. The internal current phase may be the most causative for the development of cardiac arrest and respiratory arrest.
2. As the body potential builds up in response to the current, it produces an electric field over the surface of the body. At a certain level (about one-half the air breakdown field of 4000 V/cm [10,000 V/inch]), current can emerge or escape the body at various points of the body surface (also documented by Darveniza, *Electrical Properties of Wood and Line Design*). Metal pieces on the body trigger and enhance these surface discharges and surface flashovers.
3. As the current continues to increase, the surface flashover bridges the strike point and the ground. At this level, most of the lightning current flows as an arc current through the air outside the body (flashover effect). Only a very low fraction flows through the body at this point and may be too little to cause cardiac and respiratory arrest.

A direct strike occurs when the lightning stroke attaches directly to the victim (Fig. 3-9A). This is most likely in the open when a person has been unable to find a safer location, and probably occurs no more often than in 3% to 5% of injuries. Even though it seems intuitive that direct strike might be the most likely to cause fatalities, this has not been shown in any studies.

Contact, or touch potential, injury occurs when the person is touching or holding onto an object to which lightning attaches. A voltage gradient is set up on that object from strike point to ground, and the individual in contact with the object is subject to the voltage between their contact point and the earth (see Figure 3-9B). A current therefore flows through them. Contact injury probably occurs in about 1% to 2% of injuries.

A more frequent cause of injury, perhaps as much as 30% to 35%, is a side flash, also termed "splash." Side flashes occur when lightning that has hit an object such as a tree or building travels partly down that object before a portion "jumps" to a nearby victim (see Figure 3-9C).[92] Standing under or close to trees and other tall objects is a very common way in which people are splashed. Current divides itself between the two paths in inverse proportion to their resistances. The resistance of the "jump" path represents an additional path separate from the path to the earth from the stricken object. Side flash may also occur from person to person.

Earth potential rise (EPR) occurs because the earth, modeled ideally as a perfect conductor, is not so in reality. When lightning current is injected into the earth, it travels through the earth just like it would in any other conductor. Earth has a finite resistance, and so voltages are set up in the ground, decreasing in size with distance from the strike point. The voltage (or potential) of the earth is raised, hence the term EPR.

There are several consequences of EPR. If a person is standing in an area where EPR is active, that is, near the base of a strike, a voltage will appear between their feet and current will flow via the legs into the lower part of the body. This is more significant between front and back legs of animals, where the path may involve the heart (see Figure 3-9D1).

A special case occurs when a person is injured inside a building as lightning hits nearby and is transmitted through the land line of the telephone, the pipes of plumbing and faucet handles, via electrical wiring as one uses a computer, or attempts to dispatch an ambulance.[4,78,168,181] This is caused, for example, when the person, along with the environment around them, is raised in potential via EPR. If the telephone line is not locally earthed (grounded), it is at the same voltage as the environment. The fact that the line is earthed remotely away from the local EPR environment causes the person to be subjected to a shock with current flowing between the local earth at high potential and the distant unaffected earth. This highlights that local electrical apparatus, including telephones, should be well grounded locally. The grounds of all local structures (power, telephone, plumbing, structural steel) should have a common grounding point (i.e., be bonded) to eliminate any voltage differences developing between separated ground points for each system. For the special case of indoor or telephone injury, EPR may account for 80% or more of the injuries.

Static electrical discharges may occur when a person reaches for a car door or stands close to a metal window or door frame in a thunderstorm, because the surrounding electrical field induces static electrical charges. These are not lightning injuries. While people may be startled when this happens, these discharges are unlikely to be any more dangerous than static discharges experienced in the winter months from shuffling across the carpet and reaching for a door handle.

Kitigawa has identified further subdivisions of the EPR phenomenon.[119,121,122] He notes that not only can EPR occur as discussed, but it can also occur in a manner similar to the surface flashes over a body, with arcs developing over a ground surface (Fig. 3-10). Despite modeling to the contrary, the grounding earth is not homogeneous and provides arc generation points.

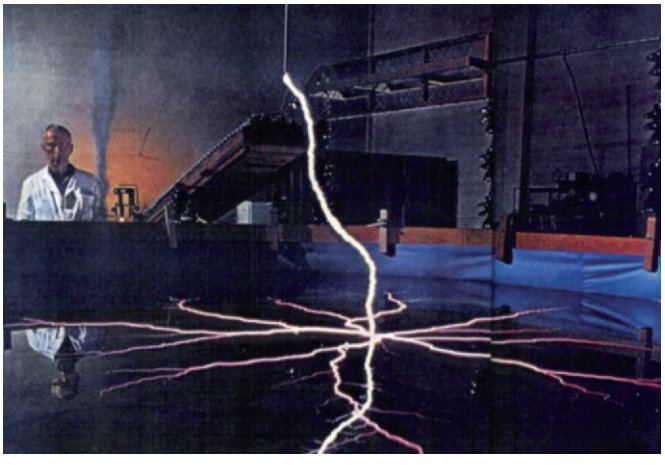
Ground current effects are possibly more likely to be temporary, slight, and less likely to produce fatalities. However, multiple victims and injuries are frequent. Large groups have been injured on baseball fields, at racetracks, while hiking, and during military maneuvers.[24,137] Shocks via telephones can produce significant long-term problems.[8,78,156]

Irregularities are highlighted on mountainsides. If the terrain is markedly irregular, the spreading lightning current may reach the surface, and a surface arc discharge develops together with the flow of the conduction current in the ground. Because arcs carry considerable energy, a person exposed to a surface arc discharge is more likely to have a more severe effect, including thermal injuries, temporary paralysis, or even death. This mechanism of injury makes it particularly dangerous for someone on a mountainside to shelter inside a shallow cave or under a small cliff or outcropping of terrain where surface arcing is much more likely to occur, injuring the person just as they feel some degree of safety has been achieved (see Figure 3-9D3).

The danger of upward streamers has recently been documented.[1,26,60,72] Injury may occur when a victim serves as the conduit for one of the usually multiple upward leaders induced by a downward stepped leader and its field (see Figure 3-9E). Streamers occur even when there is no attachment between them and the stepped leader. While one might think that these are weak in energy compared to the full lightning strike, and although upward leaders are poorly characterized, they may carry several hundred amperes of current to be transmitted through the victim. This mechanism has been mentioned by many engineering and physicist lightning experts in their writings, and a case report has been published in the medical literature.[60] Upward streamer injury is probably a much underestimated mechanism of injury and may account for as much as 30% to 35% of injury cases.

Finally, persons may suffer from (nonelectrical) blunt injury, either by being close to the concussive force of the shockwave produced as lightning strikes nearby or if ground current or

A



B

Figure 3-10. **A,** Lightning injury and ground current effect to a golf green—photographed a few days after the strike when the grass had died. **B,** Laboratory lightning onto a pool surface.

some other mechanism induces an opisthotonic contraction. Victims have been witnessed to have been thrown tens of yards by either mechanism. In addition, some have theorized that a person struck by lightning may suffer from explosive and implosive forces created by the thunderclap, with resulting contusions and pressure injuries, including tympanic membrane rupture. Another mechanism of blunt injury is blast injury resulting from vaporization of water on the body surface from a surface flashover spark. Lightning blast injury to the skull, brain, and viscera has been elegantly demonstrated in animals.[162]

Many cases of multiple injuries are likely a combination of many of these effects, with the majority of them from EPR and

Figure 3-11. Example of contact injury.

Figure 3-12. Example of side flash or "splash."

upward streamers, sometimes complicated by side flashes if people or animals are standing too close together (Figs. 3-11 and 3-12). Information on the exact mechanisms remain poorly documented and understood.

PATHOPHYSIOLOGY OF LIGHTNING INJURY[10,12,52,53,56,112,131,132]

Electrical Injury Physics Revisited

Kouwenhoven determined six factors that affect the type and severity of injury encountered with electrical accidents: frequency, duration of exposure, voltage, amperage, resistance of the tissues, and pathway of the current. However, several inconsistencies appear when these are applied too literally.

Electric field strength, not listed as one of the factors, is a more useful and accurate concept in explaining and predicting injuries from technical or manmade electricity than the classical Kouwenhoven factors that have traditionally been cited in the medical literature. Figure 3-13 shows 20 kV applied to a 1.8-m (6-foot) man, causing current to ground. This produces an internal electrical field strength of approximately 10 kV/m. When a child chews on an electric cord and suffers a lip burn, the field strength is approximately the same: 110 V applied to 1 cm of a child's lip generates a field strength of 11 kV/m. Even though no one would classify the child's injury as a "high" voltage, it is a high electrical field strength and produces the same tissue destruction in a small localized area, much as would a high-voltage injury. Lee indicates the importance of internal electric field calculations in describing electroporation damage.[128–133]

A similar inconsistency involves the breakdown strength of air (the force needed to cause a spark of electricity to cross a gap), which is roughly 4000 V/cm, or 10,000 V/inch. Most people are familiar with the shock experienced from walking across a carpet in the winter, although few appreciate this phenomenon would be classified as "high-voltage" injury by the 500- to 1000-V criterion used in medical literature.

Thus, terms used in the medical literature to categorize electrical injuries, such as *high* versus *low* voltage and *exit* versus *entry* in alternating current injuries, as well as the simplistic application of Kouwenhoven's six factors, do not reflect either medical or engineering reality and are poor predictors of injury.

Lightning Injury Physics

It is necessary to distinguish between lightning and generator-produced high-voltage electrical injuries, in that there are significant differences between the mechanisms of injuries and their treatment. Although lightning is an electrical phenomenon and is governed by the laws of physics, it accounts for a unique spectrum of induced signs and symptoms that are best understood relative to specific physical properties of lightning.

Frequency, Voltage, Amperage, and Resistance

Lightning is neither a direct nor an alternating current. At best description, lightning is a unidirectional massive current impulse. The cloud-to-ground impulse results from breakdown of a large electric field between cloud and ground, measured in millions of volts. Once connection is made with the ground, the voltage difference between cloud and ground disappears and a large current flows impulsively in a very short time. The study of massive electrical discharges of such short duration, particularly their effects on the human body, is not well advanced. Lightning is said to be a "current" phenomenon rather than a "voltage" phenomenon. Examining the particular voltage in these equations becomes difficult because the voltage between cloud and ground disappears after lightning attachment, and equations such as Ohm's law ($V = I \times R$) and power calculation ($P = V \times I$) cannot be accurately applied. Thus, we must resort to alternative formulations of the equations.

The energy dissipated in a given tissue is determined by the current flowing through the tissue and its resistance by:

$$\text{Energy (heat)} = \text{Current}^2 \times \text{Resistance} \times \text{Time}$$

where a current flows through a resistance for a time T.

As resistance goes up, so does the heat generated by passage of the same current. In humans when low energy levels are encountered, much of the electric energy may be dissipated by the skin, so that superficial burns are often not accompanied by internal injuries.

Although lightning occasionally creates discrete entry and exit wounds, these are rare. Lightning more commonly causes only superficial streaking burns. The exception to this is when "hot lightning," or long continuous current, occurs. This is a prolonged stroke lasting up to 0.5 second that delivers a tremendous amount of energy, capable of exploding trees, setting fires, and acting like high-voltage electricity to produce injuries. Other factors not understood may contribute to the formation of deep burns, although deep burns similar to those of high-voltage electrical injuries generally are quite rare with lightning.

Pathway, Duration of Current, Flashover Effect, and Time

It takes a finite amount of time for the skin to break down when exposed to heat or energy. Generally, lightning is not around long enough to cause this skin breakdown. Probably a large portion travels along the outside of the skin as "flashover." There is some experimental evidence that a portion of the current may enter the cranial orifices—eyes, ears, nose, and mouth.[5,6,10] This pathway would help explain the myriad eye and ear symptoms that have been reported with lightning injury.

Andrews[5] further examined the functional consequences of lightning on cardiorespiratory function and concluded that entry of current into cranial orifices leads to passage of current directly to the brainstem. In a sheep study, he was able to demonstrate specific damage to neurons at the floor of the fourth ventricle in the location of the medullary respiratory control centers. It is postulated that current travels from the caudad via cerebrospinal fluid (CSF) and blood vessel pathways to impinge directly on the myocardium. Andrews[5] also showed histologic damage to the myocardium, consistent with a number of autopsy reports of inferior myocardial necrosis.[9]

An alternative hypothesis can be tested with mathematical modeling.[5,12] Certain assumptions are made in any model, usually based on principles accepted in the literature.[18,131] Figure 3-14A shows a model for skin resistance, and its connection to the internal body milieu is shown in Figure 3-14B. Note that the internal body structures are regarded as purely resistive, whereas the skin contains significant elements of capacitance.[18,131]

In the model, the sequence of events during the strike starts with the postulate that the stroke attached initially to the head of the victim. From our knowledge now, it seems that this is an uncommon point of attachment, but the model is still useful in illustrating lightning energy flow. For a small fraction of time, current flowed internally as the skin capacitance elements became charged. At a voltage taken as 5 kV, the skin was assumed to break down. (A lightning stroke is modeled as a current wave, building to a maximum value in around 8 msec, although this may be "modulated lightning," that is, lightning that has passed through other structures, such as wiring. Others have measured the rise time of direct lightning as 1.2 to 1.5 msec.) Once the internal current increased, the voltage across the body to earth built up, and external flashover across the body occurred when the field reached the breakdown strength of air.

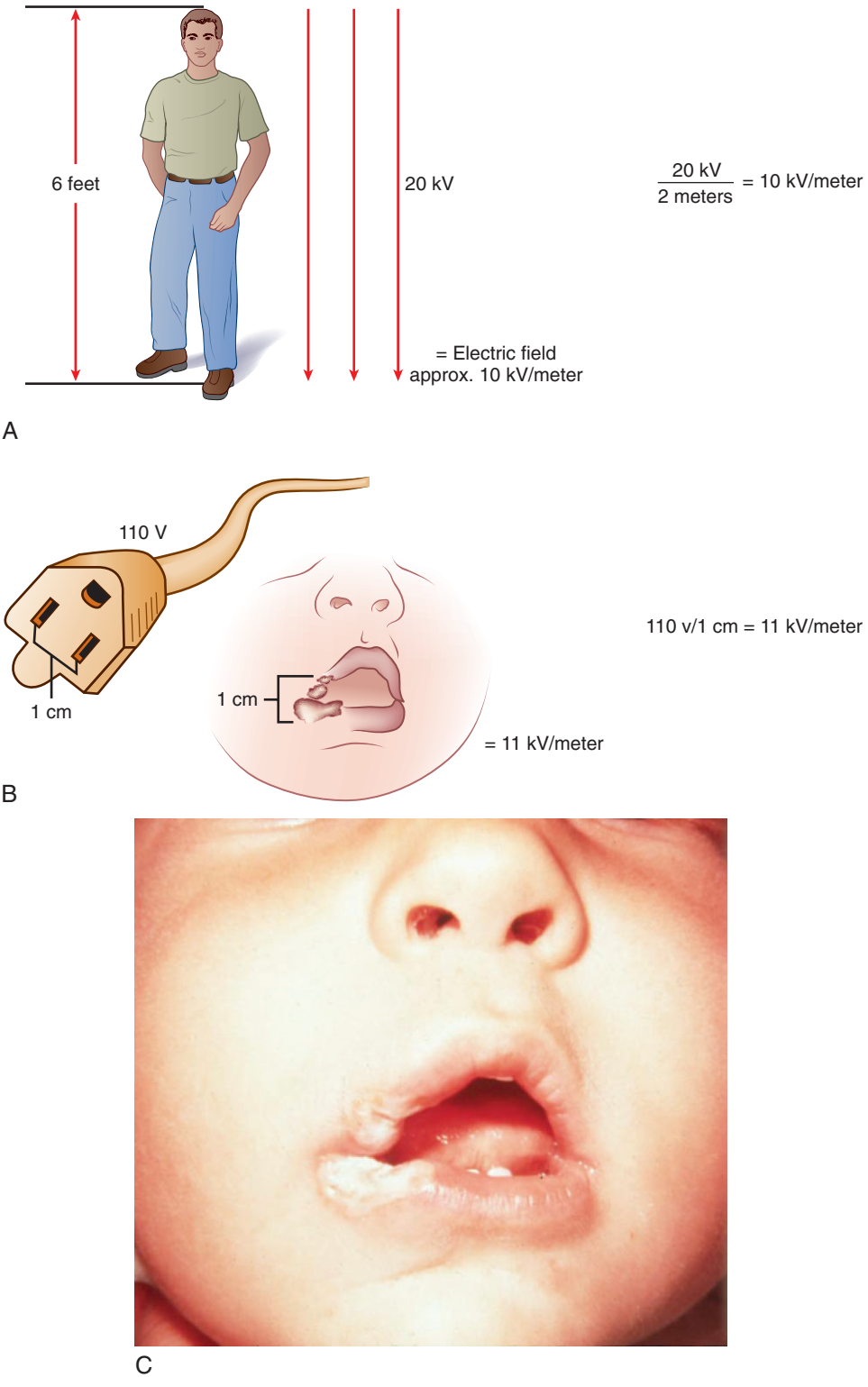


Figure 3-13. **A,** If 20 kV is applied to a 6-foot (1.8-m) man source to ground, an electric field strength of approximately 10 kV/m is produced. **B,** When a child chews on the end of an extension cord, the applied voltage of 110 volts across 1 cm produces an electric field strength of 11 kV/m—higher than the classic "high-voltage" injury. **C,** This explains the deep full-thickness burns the child receives which one would not predict given the "low-voltage" source.

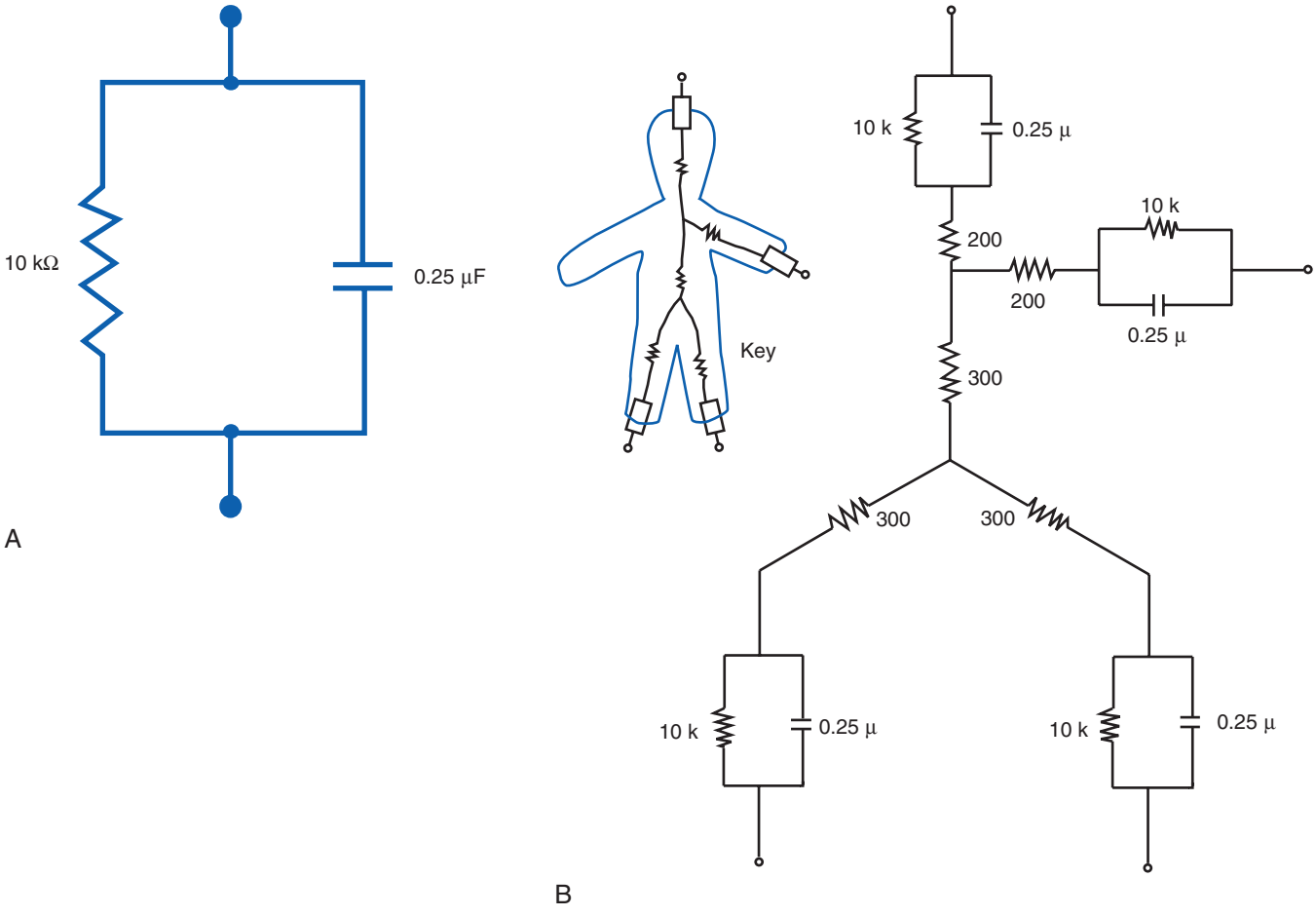


Figure 3-14. **A,** Electrical model for human skin impedance. **B,** Model of human body for the purpose of examining current flow during lightning strike.

The results of mathematical modeling of these events are shown in Figure 3-15, and the relative magnitudes of the various voltage components can be seen with their time scale. On this time scale, the times to breakdown are short and most events occur early in the course of the stroke. In summary, in this model, lightning applies a current to the human body. This current initially is transmitted internally, following which skin breaks down. Ultimately, external flashover occurs. Andrews[5] draws support for this model from measurements made in the experimental application of lightning impulses to sheep. Further modeling of step voltage injury verified that, for the erect human, this mechanism is less dangerous than is a direct strike.[121]

Experimental evidence suggests that "a fast flashover appreciably diminishes the energy dissipation within the body and results in survival." In addition, Ishikawa obtained experimental results with rabbits similar to the human data found by Cooper's study.[52] Cooper[68–70] has carried her studies to animals in developing an animal model of lightning injury and has successfully shown primary cardiac arrhythmias, prolonged ventilatory arrest, secondary cardiac arrest, keraunographic skin changes, and temporary lower extremity paralysis.

As current flashes over the outside of the body, it may vaporize moisture on the skin and blast apart clothes and shoes, leaving the victim nearly naked, as noted by Hegner[98] in 1917:

> *The clothing may not be affected in any way. It may be stripped or burned in part or entirely shredded to ribbons. Either warp or woof may be destroyed leaving the outer garments and the skin intact. . . . Metallic objects in or on the clothing are bent, broken, more or less fused or not affected. The shoes most constantly show the effects of the current. People are usually standing when struck, the current then enters or leaves the body through the feet. The shoes, especially when dry or only partially damp, interpose a substance of increased resistance. One or both shoes may be affected. They may be gently removed, or violently thrown many feet, be punctured or have a large hole torn in any part, shredded, split, reduced to lint or disappear entirely. The soles may disappear with or without the heels. Any of the foregoing may occur and the person not injured or only slightly shocked.*

The amount of damage to clothing or to the surface of the body is not an index to the severity of injuries sustained within

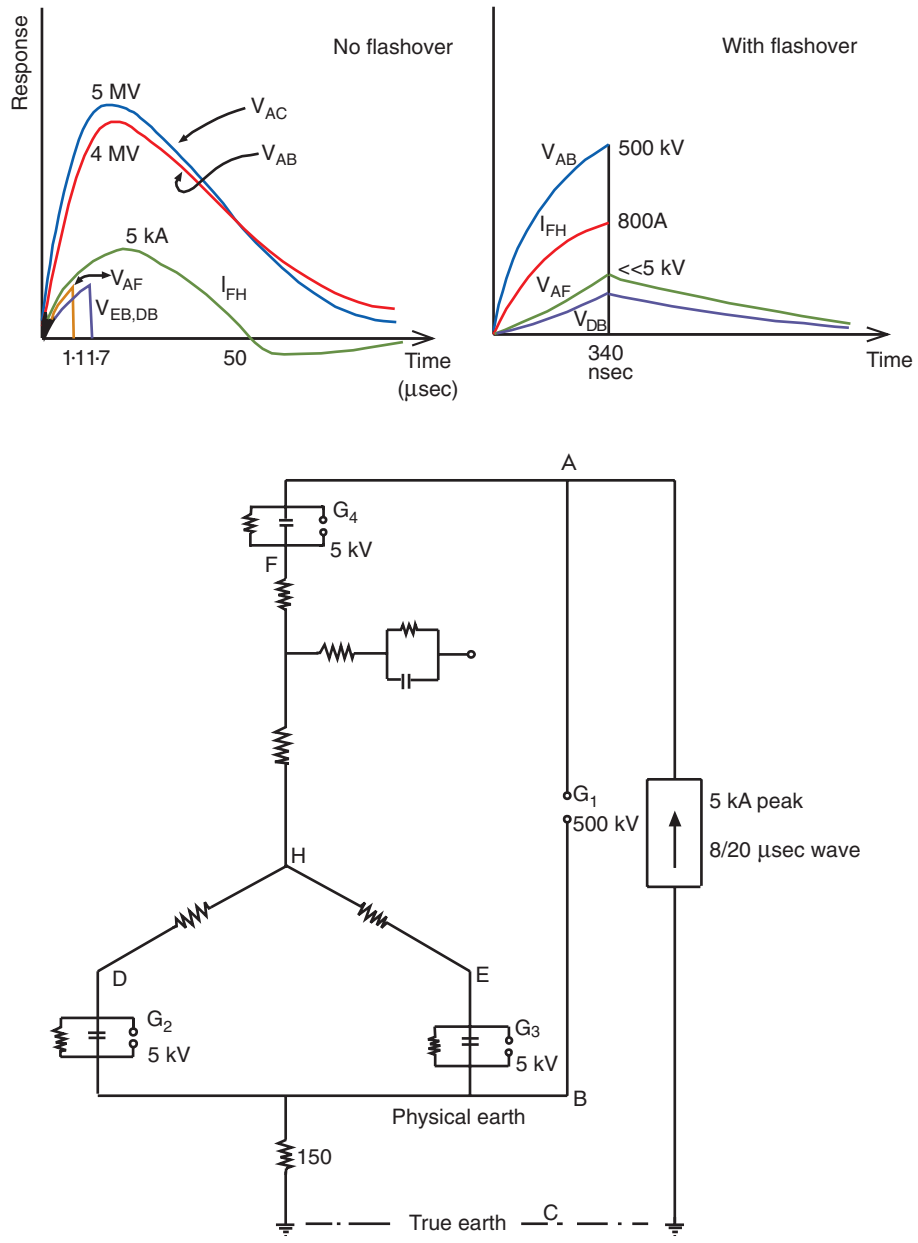


Figure 3-15. Model of human body adapted for the circumstance of direct lightning strike. Responses of the body model are shown for cases of direct strike with and without subsequent flashover.

a human. Either may be disproportionately great or small. However, in unwitnessed situations, Cooper and others have found that forensic evidence of damage to shoes and clothing may be the most important and reliable indicator in determining whether lightning caused a person's death.[116,198]

The factor that seems most important in distinguishing lightning from high-voltage electrical injuries is the duration of exposure to the current, both because lightning is not around long enough to cause tissue breakdown in the classic burn sense and because of the results of the mathematical modeling describing the path of the energy and how long it is in contact with the body.

Behavior of Current in Tissue

High- or low-voltage electric current may be carried through tissue in a direct conduction fashion, obeying simple linear equations such as Ohm's law. The result is heating of tissues under Joule's law, with thermally induced cellular death and dysfunction. Simple passage of current may interfere with neural and muscular function.[131]

Earlier in the previous century, electrical injury was thought to occur not only because of thermal effects but also because of some mysterious cellular effects.[17,113] Unfortunately, the technology was not available to investigate these effects and this idea was largely forgotten. In the last few years, the theory of electroporation has been examined. Cell wall integrity, enzyme reactions, protein shape and structure, and cell membrane "gates" and pumps all operate by changes on the order of microvolts. It is not beyond the realm of imagination that passage of an electric current too small to produce significant thermal damage may cause irreversible changes in these functions, leading to cell death or dysfunction.[131] Induction of electric charges by external electromagnetic fields has been shown to force water molecules into cell walls, causing the occurrence of fatal "pores."

Magnetic Field Effects

It has been stated that some effects of lightning might be magnetically mediated.[43] A case cited in support of this contention was of a golfer under a tree in the company of three other persons. It was stated that death occurred without evidence of current entering or leaving the index case. On the other hand, one accompanying golfer showed evidence of current traversal but survived. It is stated that three methods of shock may have existed—direct strike, side flash, and ground potential—but no evidence of any was seen. It was considered that contact potential was not relevant. In this case, with persons under a tree, it would seem possible to explain deleterious effects without resorting to a magnetic hypothesis, but the hypothesis bears examination, because it is a recurrent question.

In the case under consideration, the stroke was considered as a line current 1 m (3.3 feet) distant from the victim, and calculation of peak fields and their effects were given.

In considering the contention, it is useful to consider the stroke as a single line current as referenced; however, one must also gain a feeling for how far from a victim such a stroke will act. If the stroke is close to a victim, then attachment to the victim takes place and electrical effects apply as described earlier. If further away, the magnetic field is operative without attachment and magnetic effects need to be examined. Ground potential at this 1-m (3.3-foot) distance also exists.

It is necessary to find the minimum distance away from a victim that a stroke can reach ground without attachment to the victim. This gives the worst case distance from a victim (the worst case being the closest) at which a pure magnetic field acts without attachment.

The standard striking distance formula gives such a distance.[76] The formula is:

$$d_s = 10I^{0.65}$$

where d_s (m) is the striking distance and I is the stroke current in kA. This represents the distance at the last turn of the downward stepped leader, such that if an object lies inside this distance, attachment of the leader to the object will take place.

For illustrative purposes, let a stroke have a peak current of 18,000 A (the 50th percentile) so that d_s is 65.5 m. Pure magnetic effects are applicable at this distance and beyond. Inside this distance, the victim will be subject to electrical current effects. By comparison, the ground potential between two points 1 m (3.3 feet) apart at 60 m (197 feet) from a stroke of 18 kA is about 60 V, assuming earth resistivity of 100 ohm-meters.

In examining the magnetic fields involved at this distance, assume an 18 kA stroke at a distance of 65 m (213 feet) from an individual. The peak magnetic B field (the "magnetic induction" formally quantifying the force on a moving charge in its influence) is:

$$B_{peak} = \frac{\mu_0 I_{peak}}{2\pi d_s}$$

At 65 m (213 feet), the peak B field is 88 μT.

For comparison, the earth's magnetic field is about 1 μT, and the magnetic fields causing concern for powerline fields are in the 1 to 100 μT range. The magnetic fields used in magnetic resonance imaging (MRI) scanning are around 2,000,000 to 5,000,000 times these levels. Powerline fields are, however, held to be dangerous only if chronic. If one is concerned about a lightning stroke magnetic field, he or she should be entirely concerned about MRI fields. This concern is not seen in practice. Certainly, the time-varying nature of any B field is important, both in terms of the rate of change of the field and of movement of a conductor within this field. If one assumes that the above B field is generated in about 2 μsec, then the time rate of change for the B field is about 22 T/sec. Andrews applied this field to a model heart and found an entirely noninjurious current resulting.

It is concluded that magnetic field danger in normal circumstances does not seem to exist. Certainly special circumstances might exist, such as the presence of a pacemaker or the presence of an arrhythmic pathway, but in normal terms, magnetic effects would not seem to be clinically significant during occurrences of lightning strike.

Estimates of Streamer Currents

A fifth mechanism of current impingement on an individual has been recently proposed.[2,51] This mechanism recognizes that as a stepped leader steps toward the earth from a cloud, an upward leader will emanate from several objects that are possible points of attachment. Of these, a return stroke will evolve perhaps from only one. Alternatively stated, there will be several upward leaders that dissipate without attachment.

The current needed to establish and maintain any upward leader nonetheless must be supplied from the earth, and if a person is the source of an upward leader, then current must flow through the person as a first approximation.

Estimating the magnitude of this current is fraught with difficulty because so little is known about upward streamer characteristics. To gain a ballpark estimation of the current, however, the initial resistance of the body might be taken as 1000 ohms, with capacitances being initially uncharged. Minimal investigations have been made, however, to estimate that upward streamer current would seem to be around 200 A.[1] The initial head-foot voltage would therefore seem to be 200 kV, yet flashover is not seen at this stage. This may be explained by the decrease (time constant approximately 2 ms) of head-foot voltage as capacitances become charged, whence it may be well decreased to around 20 kV if upward streamer current decreases to a minimum. Clearly, more information on streamer behavior is needed. The field giving rise to the streamer is therefore in the range 100 to 1000 V/cm, which is below the breakdown threshold in air.

The contribution of air conduction may be estimated from the conductance of air, approximately 120 micro siemens/cm, and the field values stated. These give an air current density of 12 to 120 mA/cm^2. This represents only a small value compared with body current.

The speed with which a stepped leader makes its traverse is around 5×10^5 m/sec, with a large statistical variation, implying that for a 5-km (3.1-mile) distance, the traverse takes about 10 msec. The time course of current for an upward leader is not known, although it is likely to be less than 10% of this (i.e., 1 msec).

A rough estimate of the exposure of a body to current can be up to 200 A for 1 msec. Time/duration curves indicate that this current is in a range where effects are noticed, but there is not a risk for cardiac arrest. While this supports the contention of the fifth mechanism, the major thrust of the argument is that much more study of the parameters of upward leaders is needed.

INJURIES FROM LIGHTNING[53,63]

Severity of Injury

Some of the most common signs and symptoms are listed in Box 3-3. For prognostic purposes, victims generally can be placed in one of three groups.

Minor Injury

These victims are awake and may report dysesthesia in the affected extremity from a lightning splash or, in more serious strokes, a feeling of having been hit on the head or having been in an explosion. They may or may not have perceived lightning or thunder. They often suffer confusion, amnesia, temporary deafness or blindness, or temporary unconsciousness at the scene.[24] They seldom demonstrate cutaneous burns or paralysis but may complain of confusion and amnesia lasting from hours to days. Paresthesias, muscle pain, and headaches may last for days to months. Victims may suffer tympanic membrane rupture from the explosive force of the lightning shockwave. Vital signs are usually stable, although occasionally victims demonstrate transient mild hypertension. Recovery is usually gradual and may or may not be complete. Permanent neurocognitive damage may occur. Some victims may suffer posttraumatic stress disorder.

Box 3-3. Lightning Injuries

IMMEDIATE
- Cardiac arrest
- Neurologic signs
- Seizures
- Deafness
- Confusion, amnesia
- Blindness
- Dizziness
- Contusion from shockwave
- Chest pain, muscle aches
- Tympanic membrane rupture
- Headache, nausea, post-concussion syndrome

DELAYED
- Neuropsychological changes
- Memory deficits
- Coding and retrieval problems
- Distractibility
- Personality changes
- Irritability
- Chronic pain
- Seizures

Moderate Injury

Moderately injured victims may be disoriented, combative, or comatose. They frequently exhibit motor paralysis, particularly of the lower extremities, with mottled skin and diminished or absent pulses. Nonpalpable peripheral pulses may indicate arterial spasm and sympathetic instability, which should be differentiated from hypotension. If true hypotension occurs and persists, the victim should be scrutinized for fractures and other signs of blunt injury. Spinal shock from cervical or other spinal fractures, although rare with lightning, may also account for hypotension.

Occasionally, victims have suffered temporary cardiopulmonary standstill, although it is seldom documented. Spontaneous recovery of the pulse is attributed to the heart's inherent automaticity. However, respiratory arrest that often occurs with lightning injury may be prolonged and lead to secondary cardiac arrest from hypoxia or some other yet-to-be-elucidated cause. Seizures may also occur.

First and second degree burns not prominent on admission may evolve over the first several hours. Rarely, third degree burns may occur. Tympanic membrane rupture should be anticipated and, along with hemotympanum, may indicate a basilar skull fracture.

Whereas the clinical condition often improves within the first few hours, victims are prone to have permanent sequelae, such as sleep disorders, irritability, difficulty with fine psychomotor function and attention, paresthesias, generalized weakness, sympathetic nervous system dysfunction, and sometimes posttraumatic stress syndrome. A few cases of atrophic spinal paralysis have been reported.

Severe Injury

Victims with severe injury may be in cardiac arrest with either ventricular standstill or fibrillation when first examined. Cardiac resuscitation may not be successful if the victim has suffered a prolonged period of cardiac and central nervous system (CNS) ischemia. Direct brain damage may occur from the lightning strike or blast effect (Fig. 3-16). Tympanic membrane rupture with hemotympanum and CSF otorrhea is common in this group.

The prognosis is usually poor in the severely injured group, complicated by any delay in initiating cardiopulmonary resuscitation with resultant anoxic injury to the brain and other organ systems. There are anecdotal reports of successful resuscitation of these victims with automatic external defibrillators (AEDs).

Differences between Injuries from High-Voltage Electricity and Lightning[12,57]

There are marked differences in injuries caused by high-voltage electric accidents and lightning (Table 3-4). Lightning contact with the body is almost instantaneous, often leading to flashover. Exposure to high-voltage generated electricity tends

Figure 3-16. Injury to brain tissue, seen on autopsy, caused by a direct lightning strike to the head. (Photo courtesy Joanne Richardson, Summit County, CO, Coroner.)

TABLE 3-4. Lightning Injuries Compared with High Voltage Electrical Injuries

Factor	Lightning	High voltage
Energy level	30 million volts, 50,000 Å	Usually much lower
Time of exposure	Brief, instantaneous	Prolonged
Pathway	Flashover, orifice	Deep, internal
Burns	Superficial, minor	Deep, major injury
Cardiac	Primary and secondary arrest, asystole	Fibrillation
Renal	Rare myoglobinuria or hemoglobinuria	Myoglobinuric renal failure common
Fasciotomy	Rarely if ever necessary	Common, early, and extensive
Blunt injury	Explosive thunder effect	Falls, being thrown

to be more prolonged, particularly if the victim "freezes" to the circuit. In this context, "long" may mean only a few to several seconds of contact. With skin breakdown, electrical energy surges through the tissues with little resistance to flow, causing massive internal thermal injuries that sometimes necessitate major amputations. Myoglobin release may be pronounced, and renal failure may occur. In addition, compartment syndromes requiring fasciotomy may occur. This is not the case with lightning injuries in which burns and deep injury are uncommon and fluid restriction and expectant care are usually the rule.

Cardiopulmonary Arrest[14,52,67]

The most common cause of death in a lightning victim is cardiopulmonary arrest. In fact, a victim is highly unlikely ($P < .0001$) to die unless cardiopulmonary arrest is suffered as an immediate effect of the strike.[52] In the past, nearly 75% of persons who suffered cardiopulmonary arrest from lightning injuries died, many because cardiopulmonary resuscitation was not attempted.

Primary and secondary cardiac arrests had previously been hypothesized and confirmed with animal studies.[5,9,68–70,185] Injury first occurs with immediate asystolic cardiac arrest and respiratory standstill. Because of the heart's automaticity, contractions generally resume within a short time. Unfortunately, respiratory arrest caused by paralysis of the medullary respiratory center may last far longer than cardiac arrest. Unless the victim receives immediate ventilatory assistance, attendant hypoxia may induce arrhythmias and secondary cardiac arrest. Alternatively, the respiratory arrest and secondary cardiac arrest may be from more severe injury and not cause and effect.

The course has been verified experimentally in sheep, with initial asystole followed by resumption of a short run of bradycardia, then tachycardia, followed by an eventual atrioventricular block or bradycardia, and finally with second asystolic arrest.[5] Prolonged respiratory arrest has also been confirmed in hairless rats.[123]

It is unknown whether cardiac arrest and arrhythmias induced by lightning are a result of damage to central cardiac and respiratory centers in the brain, to the carotid body and other pacemakers along the cardiac control paths, to feedback control mechanisms within the autonomic nervous center, to the heart, or to a combination of these.[61,68–70] Certainly, clinical evidence of general damage to autonomic nervous system regulation has been well documented. Recently, cardiac effects from autonomic nervous system damage have been confirmed in the animal laboratory.[70]

Both asystole and ventricular fibrillation[6] have been reported with lightning strike. As noted in the animal work, asystole seems to be both the first and last response to the strike, as the secondary agonal arrest rhythm of ventricular fibrillation deteriorates.[5] Premature ventricular contractions, ventricular tachycardia, and atrial fibrillation have been reported.[67,138]

It is not uncommon to find electrocardiographic (ECG) ST changes consistent with ischemia and damage in subepicardial, posterior, inferior, or anterior patterns.[9,138] Creatine phosphokinase MB-isoenzyme elevation has been reported. The first report of troponin level elevations and cellular damage in a lightning survivor was published by a high-altitude pulmonary physiologist who suffered injury while climbing in the Alps, using himself as a control.[15]

Changes on an ECG may not occur until the second day, making the initial ECG a poor screening tool for ischemia. Several authors stress that cardiac symptoms may not be apparent on initial presentation. Premature ventricular contractions were reported in one patient nearly 1 week after presentation. Whereas most ECG changes resolve within a few days, some may persist for months.[17]

The QT interval may be prolonged following lightning strike[7] and may have implications for the cessation of metabolism hypothesis if torsades de pointes is involved.[7]

Some authors have theorized that vascular spasm is a cause of cardiac damage.[165] However, ECG changes are not always consistent with cardiac vascular supply patterns.[143] Areas of focal cardiac necrosis have been reported in autopsies, and histologic changes have been shown in sheep hearts.[5]

Pulmonary edema may accompany severe cardiac damage. Pulmonary contusion, with severe hemoptysis and pulmonary hemorrhage, may result from blunt injury or direct lung damage.[183]

Neurologic Injuries[13,32,34,39–42,54–56,117]

Although cardiac arrest may be the only immediate cause of death, lightning injury is primarily a neurologic insult with damage possible to central, peripheral, and sympathetic nervous systems. Injury to the nervous system far and away causes the greatest number of long-term problems to survivors. Tools commonly used in evaluation and treatment include functional scans, such as single photon emission computed tomography (often positive); positron emission tomography; anatomic scans, such as computed tomography (CT) and MRI (usually negative); neuropsychological assessment; cognitive retraining; pharmacotherapy as already mentioned; and psychotherapy. Electroencephalography is often mentioned in other literature, but it is seldom helpful.[9,57]

Central Nervous System Injury[13,32,34,39–42,54–56,117]

Gross structural changes to the brain, such as coagulation of brain substance, formation of epidural and subdural hematomas, paralysis of the respiratory center, and intraventricular hemorrhage, have been reported but are all rare and may be more from blunt trauma than from electrical injury. Autopsy findings include meningeal and parenchymal blood extravasation, petechiae, dural tears, scalp hematomas, and skull fractures.[186] While MRI or CT may show diffuse edema, intracranial hemorrhage, or other injury, they are more commonly normal.[38,41,42] There have been reports of MRI findings in a few acutely injured victims.[41]

In animals, direct cellular damage to the respiratory center beneath the fourth ventricle, as well as the anterior surface of the brainstem, has been shown.[5] Lightning energy has been theorized to enter through the orifices of the head, pass through the area of the pituitary and hypothalamus, and through the CSF into the retropharyngeal area, so that signs and symptoms of endocrine dysfunction, respiratory or cardiac arrest, and sleep disturbances could be reasonably expected and do, in fact, occur.

Seizures may accompany initial cardiorespiratory arrest as a result of hypoxia or intracranial damage.

Electroencephalographs may show epileptogenic foci in the acute phase. These patterns may be focal or diffuse, varying with the site and type of injury. However, most patients do not experience seizures during hospitalization. Some victims, however, including children, develop delayed seizures, some of which present as "absence spells," memory losses, or blackouts that are often diagnosed as "pseudoseizures."

Obviously, the victim of prolonged cardiopulmonary arrest may have anoxic brain injury that is not specific to lightning injury.

In Cooper's study[52] of severely injured victims, nearly two thirds had some degree of lower extremity paralysis (keraunoparalysis), usually demarcating around the waist or pelvis, and about one third of victims had upper extremity paralysis.[52] The affected extremities appear cold, clammy, mottled, insensate, and pulseless.[12] This is probably the result of sympathetic instability and intense vascular spasm, similar in appearance to Raynaud's phenomenon. It usually clears after several hours.[12,57] Fasciotomies are seldom indicated for lightning injuries, because any signs of compartment syndrome or distal ischemia usually clear with patient observation. Pulses can sometimes be elicited with a Doppler examination. Atrophic spinal paralysis has been reported, as have persistent paresis, paresthesias, incoordination, delayed and acute cerebellar ataxia, hemiplegia, aphasia, quadriplegia (immediate or delayed), and one report of progressive muscle atrophy of the upper extremities.

Nearly 72% of the victims in Cooper's study suffered loss of consciousness.[52] Nearly three fourths of these also suffered a cardiopulmonary arrest.[52] Those with cranial burns were two to three times more likely to suffer immediate cardiopulmonary arrest and had a three to four times greater probability of death.[52] Persons who are stunned or lose consciousness without cardiopulmonary arrest are highly unlikely to die,[52] although they may still suffer serious sequelae.

Whether or not victims have suffered loss of consciousness, they almost universally demonstrate anterograde amnesia and confusion, which may last for several days. Retrograde amnesia is less common. While the victim may carry on a conversation and remember his or her actions before the strike, he or she is often unable to assimilate new experiences for several days, even when there is no external evidence of lightning burns on the head or neck.

Survivors may have persistent sleep disturbances, difficulty with fine mental and motor functions, dysesthesias, headaches, mood abnormalities, emotional lability, storm phobias, decreased exercise tolerance, and posttraumatic stress disorder.[63,181]

The basal ganglia and cerebellum may be affected.[41,42] This agrees with older reports of localization. CNS damage may also eventually present as parkinsonism, extrapyramidal syndrome, or other involuntary movement disorders or signs of intracranial hematomata and hemorrhage, infarction, hypoxia, edema, cerebellar signs of infarction, dysfunction and atrophy, and centrally derived pain and psychological syndromes.[30]

Peripheral Nerve Injury[199]

The peripheral nervous system may be affected. Pain and paresthesias are prominent features of the injury, particularly in the line of the current passage. It would seem that the majority of ongoing pain and dysfunction may be accounted for in the peripheral nervous system.

Symptoms may be delayed by weeks to months. Paresthesias are frequently seen, often mirroring the area of keraunoparalysis. There is also evidence of autonomic neuropathy.

Autonomic Dystrophy[49,99,160,169,178,179,181]

Chronic pain syndromes may occur. Autonomic dystrophy, also called sympathetic dystrophy or sympathetically mediated pain syndrome, may occur. Such chronic pain syndromes are now subsumed under the classification of the complex regional pain syndromes, which are of types I (previously causalgia) or II (reflex sympathetic dystrophy).

The complex regional pain syndromes are long-term neurologic sequelae that may be caused by even minor injuries to nerves, and are characterized by pain, edema, autonomic dysfunction, trophic changes, including atrophy secondary to disuse from pain, and movement disorder.[160,178]

Post-traumatic Headache[53]

Many victims of lightning injury exhibit severe, unrelenting headaches for the first several months after lightning injury. Cooper has found acupuncture to be effective for at least some of the headaches. Many victims complain of nausea and unexpected, frequent vomiting spells early in their recovery period.

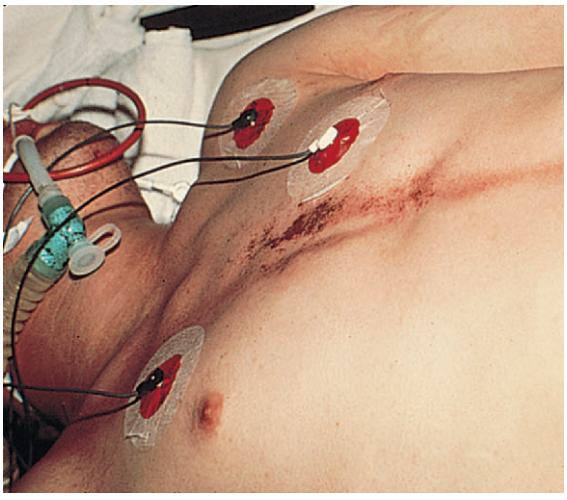


Figure 3-17. Linear burns from lightning injury.

Figure 3-18. Punctate burns from lightning injury. (Courtesy Art Kahn, MD.)

Dizziness and tinnitus are also common complaints, especially with telephone-transmitted lightning strikes.[8,53,55,59,62,63]

Burns[62,151,180]

Most people assume that, because of the tremendous energy discharge involved, a lightning victim will be flash-cooked.[24,58] Fortunately, the flashover effect saves most victims from suffering more than minor burns. Although extensive third and fourth degree burns may occur in combination with skeletal disruption, these are quite rare. Often there are no burns, especially with ground current effects. Another factor is the incredibly short period of exposure, which may also explain the lack of significant burn injury in most cases.

As shown in mathematical models, a portion of the lightning current may travel through the tissues.[5] If the electric field in the tissue becomes too large, electrons can be freed from their atoms. Referred to as dielectric breakdown,[131] this can cause a large increase in the flow of current and become manifest as an electric arc. This may occur internal or external to the body, the latter when the breakdown strength of air (2×10^6 V/m) is exceeded. Arclike burns can result. This happens more often with high-voltage electrical injury, but may also occasionally occur with lightning.

Cooper has related the prognosis of the victim to the location of the burns.[52] Persons who suffer cranial burns are four times more likely to die than those who do not have cranial burns ($P < .25$). Victims with cranial burns are two and a half times more likely to have a cardiopulmonary arrest than those who do not exhibit burns around the head and neck ($P < .025$).[52] Persons with leg burns are five times more likely to die than those who have no leg burns, perhaps because of a ground current etiology ($P < .05$).[52]

Discrete entry and exit points are uncommon with lightning. The burns most commonly seen may be divided into five categories: linear burns; punctate, full-thickness burns; feathering or flowers; thermal burns from ignited clothing or heated metal; and combinations.

Linear burns (Fig. 3-17) often begin at the victim's head and progress down the chest, where they split and continue down both legs. The burns generally are 1 to 4 cm ($^1/_2$ to $1^1/_2$ inches) wide and tend to follow areas of heavy sweat concentration, such as beneath breasts, down the midchest, and in the midaxillary line.[12,57] Linear burns are usually first and second degree burns that may be present initially or develop as late as several hours after the lightning strike. They are probably not primary lightning injuries, but are steam burns secondary to vaporization of sweat or rainwater on the victim's skin.

Punctate burns (Fig. 3-18) are multiple, closely spaced, and discrete circular burns that individually range from a few millimeters to a centimeter in diameter. They may be full thickness and resemble cigarette burns, but are usually too small to require grafting.

Feathering burns (Fig. 3-19) are pathognomonic of lightning and are known by such names as Lichtenberg's flowers, filigree burns, arborescent burns, ferning, and keraunographic markings.[35,37,50,81,100,172,192,197] These markings are not true burns, but usually appear as transient pink to brownish, sometimes lightly palpable, arborescent marks that follow neither the vascular pattern nor the nerve pathways. The pattern found is similar to that on a photographic plate exposed to a strong electric field and has been compared to fractals.[100] Sometimes the most superficial skin over the areas will slough or flake off after a few days. Although many pictures exist of these marks, they have never been described histologically. They may represent blood cells forcefully extravasated into the superficial layers of the skin from contracting capillaries. At least experimentally, they follow the current lines seen in flashover in Cooper's animal model.[68,69]

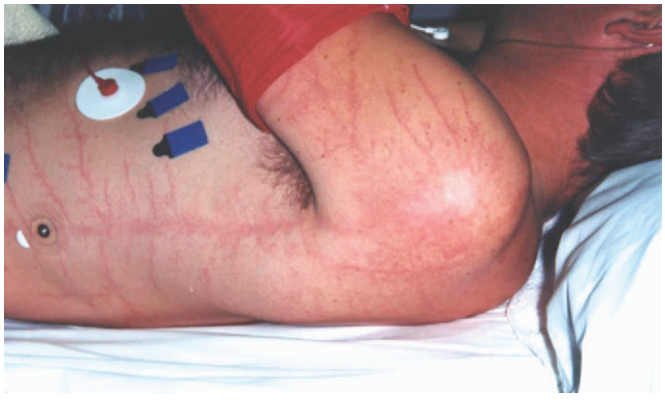
On rare occasions, clothing is ignited by lightning, causing severe thermal burns.[12,57] A victim wearing metal, such as a necklace or belt buckle, or carrying coins in his or her pocket may suffer second and third degree burns to adjacent skin as the objects become heated by the electric energy.[62,115] Fig. 3-20 shows the burn resulting from a metal belt buckle or athletic supporter worn by a young man who was struck while playing softball.

Victims of lightning may exhibit a combination of burns.

Blunt and Explosive Injuries

The recipient of a lightning strike may be injured directly from the explosive force of lightning or from a fall (as from a horse or mountain ledge, out of a vehicle, or being hurled by endogenous opisthotonic force). As might be predicted, back and spinal

A



B

Figure 3-19. **A,** Feathering burns. **B,** Ferning developed after the victim received a side flash lightning strike. (**A** courtesy Mary Ann Cooper, MD; **B** courtesy Sheryl Olson, RN.)

Figure 3-20. Burn resulting from metal belt buckle or athletic supporter worn by a man struck by lightning.

injuries unrelated to the electrical effects of lightning injury may occur from these mechanisms. A variety of fractures, including skull, ribs, extremities, and spine, have been reported in lightning victims but are uncommon.[139] Rarely, a burstlike injury of soft tissue occurs and discloses extensive underlying injuries, especially in the feet, where boots or socks may explode due to vapor expansion (Figs. 3-21 and 3-22).

Figure 3-21. Socks blown off during explosive lightning stroke.

Hemoglobinuria and myoglobinuria are seldom reported. When they occur, they are usually transient. Myoglobinuric renal failure has not been reported in the literature, although one case has been verbally relayed to the first author.

Persistent hypotension should alert the physician to blunt injuries to the chest, spine, lungs, heart, and intestines that may lead to complications of prolonged coma, pulmonary contusions,[183] heart failure, and ischemic bowel.

Several victims have complained of jaw pain. A number have suffered loss of teeth or fillings or necrosis of the jaw and teeth, and many describe a metallic taste in the mouth for months after the acute injury. At least one was found to have a styloid process fracture. Many recovering victims believe that premature arthritis may be a result of their injury.

Eye Injuries*

Ocular injuries may be due to direct thermal or electrical damage, intense light, contusion from the shockwave, or combinations of these factors.

Although cataracts most commonly develop within the first few days, they may occur late and are often bilateral.[23,78–80,85] Whereas the cataracts may be the typical anterior midperipheral type, posterior subcapsular opacities and vacuolization seem to occur more often with lightning injuries.[23] Corneal lesions, hyphema, uveitis, iridocyclitis, vitreous hemorrhage, choroidal rupture, chorioretinitis, retinal detachment, macular degeneration, optic atrophy,[23] diplopia, loss of accommodation, and decreased color sense have also been reported.

*References 27, 78–80, 85, 93, 96, 97, 111, 114, 158, 159, 184, 201.

A

B

Figure 3-22. The heat from a lightning strike caused water in the wet boot to instantaneously turn to steam, exploding the boot off the foot **(A)** and causing burns **(B).** (Photos courtesy Sheryl Olson, RN.)

Autonomic disturbances of the eye, including mydriasis, Horner's syndrome, anisocoria, and loss of light reflexes, may be transient or permanent. Transient bilateral blindness of unknown etiology is not uncommon. Intense photophobia may be present as the victim recovers. An interesting paper conjectures that the conversion of St. Paul on the Damascus Road and his subsequent blindness was due to lightning.[21]

Dilated or unreactive pupils should never be used as a prognostic sign or as a criterion for brain death in a lightning victim until all anatomic and functional lesions have been ruled out.[29]

Ear Injuries[89,93,96,114,154,161,168]

Temporary deafness is not uncommon.[114] It has been postulated that the intense noise and shockwave accompanying thunder may be responsible for sensorineural hearing loss. Newer work shows that entry of lightning current through cranial orifices could account for some of the injuries.[5,10] Telephone-transmitted lightning strikes also may account for otologic damage.[4,8,9]

Between 30% and 50% of more severely injured lightning victims may have rupture of one or both tympanic membranes from the shockwave effect, concomitant basilar skull fracture, or direct burn damage because of current flow into this orifice.[11,52] Otorrhea of the CSF or hemotympanum rarely occur. Disruption of the ossicles and mastoid has been reported. Many cases of permanent deafness are noted in older literature but are seldom found in our time. Facial palsies, both acute and delayed, may occur from direct nerve damage by lightning. Vertigo and tinnitus are common, and nystagmus and ataxia may occur.

Fetal Survival

The fetus of a pregnant woman who has been struck by lightning has an unpredictable prognosis.[52] Of 11 cases reported, nearly one half of the pregnancies ended in full-term live births, with no recognizable abnormality in the child. Approximately one fourth resulted in live births with subsequent neonatal death; the remainder were stillbirths or deaths in utero. There has been one report of ruptured uterus after lightning strike.

Hematologic Abnormalities

Several unusual hematologic complications have been attributed to lightning injuries in isolated case reports. These include disseminated intravascular coagulation, transiently positive Coombs' test, and Di Guglielmo's syndrome, a type of erythroleukemia characterized by erythroblastosis, thrombocytopenia, and hepatosplenomegaly. Although there have been anecdotal reports of increased hypersensitivity, development of allergies, and increased risk of cancer in lightning victims, perhaps indicating an immunologic component to lightning injury, these have not been studied.

Endocrine and Sexual Dysfunction

Decreased libido for both men and women and impotence for men are common complaints. Sexual dysfunction may be due to neural, spinal cord, endocrine, autonomic, or neuropsychological injury, drug effects, and possibly other causes, and should not be discounted. A report of male hypersexuality after a lightning strike has not been authenticated but can also be explained from a specific brain injury mechanism. One 32-year-old victim reported amenorrhea and premature menopause as a result of her injury. Others have reported menstrual irregularities lasting for 1 to 2 years.

Psychological and Neurocognitive Dysfunction[63]

It is not uncommon for a person hit by lightning to rest at home for a few days, assuming that he or she is supposed to feel "bad" after being hit by lightning. The victim may not see a physician until family members insist or symptoms do not abate. Neurocognitive deficits may not become apparent until a victim attempts skilled mental functions. Often, the person will attempt to return to work after the injury, but because of decreased work tolerance, short-term memory problems, and difficulty assimilating new information, he or she will be unable to continue in the prestrike occupation.

Although some medical authors were historically suspicious of victims' complaints of psychological and neurocognitive dysfunction, evaluation of many such patients appears to have confirmed a vast commonality of psychological symptoms. This

should begin to reverse a regrettable tendency, both medically and legally, to discount complaints as evidence of malingering, excessive reaction, conversion reaction, personality problems, or manifestations of "weak" coping strategies.

The syndrome is described first from a clinical viewpoint and then discussed in light of the extant literature.

Functional Issues

1. Memory disturbance. Individuals show marked diminution of short-term memory ability. They require shopping lists and reminder lists. Memory for recent names and places is diminished to the point of disability. Individuals tend to self-isolate, stop mixing socially, or avoid going into new circumstances.[55,59]
2. Concentration disturbance (adult attention deficit disorder). Individuals show deficits in their working memory, are unable to focus attention for more than a short period, and are easily distracted. In particular, reading and understanding are poor. Job training is detrimentally affected. This is worsened by sleep disturbance.
3. Cognitive function. Individuals report diminished mental agility. The keeping of accounts is a noteworthy example. Calculation and estimation become erratic and affect work performance. Ability at mental manipulation and problem solving is markedly decreased.
4. Higher executive functioning. Individuals are neither able to coordinate multiple tasks simultaneously nor able to follow orders for complex tasks they used to perform easily before the injury. One victim described it as if "the office manager of my brain had quit."

Behavioral Issues

1. Emotional lability and aggression. Individuals find that they are more aggressive than before. They are easily frustrated and are liable to have outbursts of temper. The strains on a partnership are significant, and marital and relationship dysfunction is common. An increased state of arousal and anxiety may further complicate distractibility as well as the proper recognition and assimilation of new learning.
2. Sleep disturbance. Extreme fatigue, sleep disturbance, or hypersomnolence is common and may last for years. Flashbacks and nightmares may be experienced.
3. Phobic behavior. Avoidance of the precipitant circumstances is demonstrated, in some cases to phobic proportion tantamount to posttraumatic stress disorder.
4. Depression is almost always present in classic biologic form and should be anticipated. Although the aforementioned symptoms can certainly arise secondary to a depressive state, it would also be reasonable for a victim to react with depression to the decrements in work power and lifestyle engendered by chronic pain, sleep deprivation, or decreased personal performance. A third reasonable possibility is concurrent neuropsychological syndrome and biologic endogenous depression. It is the view of the authors that the psychological disturbance exists as an organic entity and that part of that syndrome is also depressive as a primary organic entity. Both elements "feed" each other, compounding to a mixed picture.

Antidepressant medication may be useful. Formal neuropsychological testing may be used to attempt to validate the injury, quantify a functional baseline, and design cognitive therapy. Very few studies have formally examined the syndrome.[166,167,188] Primeau and coworkers point to research difficulties, including sample bias and heterogeneity, methodology (cross-sectional rather than longitudinal or prospective), and the essential difficulties of determining premorbid status or current independent psychiatric status. It is also noted that the magnitude of insult does not correlate with psychological disability.[55,59] Duration, type, and severity of the syndrome are therefore unpredictable.

Disturbances of verbal memory, attention, concentration, and new learning are very frequently identified.[166,167] This commonality among victims is noteworthy.

Primeau and coworkers also draw attention to the similarity of some facets of the disorder and those of other etiologies. They generally find the head injury model a useful one that can guide treatment. Other syndromes for which the lightning and electrical injury syndrome shows similarities include posttraumatic stress disorder, depression, anxiety, and obsessional and adjustment disorders. Features of these are seen to be present, although the syndrome is not simply an example of these dysfunctions per se.

The authors have considered somatoform disorders as a cause, noting the tendency to be preoccupied with the injury and to overattribute subsequent symptoms to the lightning injury. This may be due, in part, to the general lack of knowledge by physicians of the problems and likely outcomes of the injury, so that victims do not know what to expect in the future.

Conversion reaction may also be considered, although it cannot account for the symptomatology of lightning and electrical disability.[59] Good neuropsychological testing can detect this facet. One difficulty with neurocognitive testing is use and interpretation of the Minnesota Multiphasic Personality Inventory, which, although it was not developed to characterize patients with chronic problems, particularly those with chronic pain, has been applied to them, often resulting in erroneous conclusion of conversion reaction or preoccupation with physical complaints. It should be a surprise to no one that anyone suffering from chronic pain and neurologic injury that hampers their normal preinjury activities will rank higher on the "preoccupation with physical complaints" and "conversion" scales than will uninjured normal individuals.

Andrews and coworkers[8] recognized three postinjury syndromes and correlated these with the postinjury periods of 1 week, 1 week to 3 months, and 3 months to 3 years. Primeau and coworkers[166] add a fourth, which is persons experiencing longer and perhaps lifetime dysfunction.[96]

The first 12 months after injury are crucial to recovery. It is in this period that the most recovery is seen, with possible mild improvement up to 3 years after injury. Beyond this time, chronic dysfunction may be assumed, although it is also at this time that the survivor may decide to move on with life, accept limitations, and even sometimes be able to successfully pursue a new career.

Van Zomeren and coworkers[188] provide one of the few thorough examinations of the syndrome in lightning-injured patients. Fatigue and energy loss were the main complaints; poor concentration, irritability, and emotional lability were other common themes confirmed. Impairment of memory, attention, and visual reaction times were documented. Depression and "convincing signs of posttraumatic stress disorder" were seen. The syndrome was highly differentiable from normal ($P < .001$), and no parallel model was available for comparison. They stated that the lasting complaints and mild cognitive impairments could not be explained on the basis of anxiety reac-

tions or depression. They summarized their findings under the heading of "vegetative dystonia."

RECOGNITION AND TREATMENT OF LIGHTNING INJURIES[53,57,59,63]

Diagnosis

Diagnosis of lightning injury may be difficult. History of a thunderstorm, witnesses who can report having seen the strike, and typical physical findings in the victim make diagnosis easier but are not always present.

Lightning can strike on a relatively sunny day, thunder may not be appreciated, and sometimes the victim is alone when injured. A diligent historical effort and careful physical examination at the earliest opportunity may help determine the true cause. Cloud-to-ground lightning data for past times and places are available to verify the possibility of thunderstorms. Any person found with linear burns and clothes exploded off should be treated as a victim of lightning strike (Fig. 3-22).

Diagnosis is made doubly difficult as burns, which most people expect to accompany lightning injuries, are often absent. Feathering marks, also called Lichtenberg's flowers, are pathognomonic of lightning strike but are infrequently encountered.[35,37,50,81,172,192,197] Another sign/symptom complex includes linear or punctate burns, tympanic membrane rupture, confusion, and outdoor location. Because there have been several cases of side flash or contact injury from indoor plumbing and telephones, the physician should suspect lightning strike in persons found confused and unconscious indoors after or during a thunderstorm.[4,8,12,57,62]

Initial First Aid and Triage of Victims[12,57,67]

As in any other emergency, the first steps are the ABCs: airway, breathing, and circulation. If the victim has suffered a cardiac arrest, cardiopulmonary resuscitation should be started immediately and a rescue vehicle called for transportation; automated external defibrillators (AEDs) have been helpful in some cases.

If the strike occurs far from civilization and evacuation is improbable, the victim will probably die unless pulse and respirations resume spontaneously in a short period of time. The heart may resume activity but may slip into secondary arrest. It is unknown whether the secondary cardiac arrest is due to primary brain damage, hypoxia from prolonged respiratory arrest, primary cardiac damage, or autonomic nervous system damage. If no pulse is obtained within 20 to 30 minutes of starting resuscitation, it is reasonable to stop further resuscitation efforts. If a pulse is obtained, ventilation should be continued until spontaneous adequate respirations resume, the victim is pronounced dead, continued resuscitation is deemed unfeasible owing to rescuers' exhaustion, or there is danger to rescuers' survival.

When lightning strike involves multiple victims, resources and rescuers may not meet the demand and triage must be instituted. Normally the rules of triage in multiple-casualty situations dictate bypassing dead persons for those who are moderately or severely injured and can benefit from resuscitation efforts. However, "resuscitate the dead" is the rule in lightning incidents, because victims who show some return of consciousness or who have spontaneous breathing are already on the way to recovery. The most vigorous attempts at cardiopulmonary resuscitation should be directed to the victims who appear to be dead because they may ultimately recover if properly resuscitated. Survivors should be routinely stabilized and transported to the hospital for more thorough evaluation.

The probability that lightning victims can recover after prolonged cardiopulmonary resuscitation (several hours) is not high. There is no evidence to suggest that survival after a longer period than normal without resuscitation is possible in the setting of lightning injury. If the victim has not regained a pulse after 20 to 30 minutes of resuscitation, the chances of recovery are slim and the rescuer should not feel guilty about stopping resuscitation. Often in a remote setting, the rescuer is emotionally tied to the victim by age and friendship and may tend to continue resuscitation past the point of futility. In pronouncing a victim dead, the rescuer must be sure that other problems, such as hypothermia, are not contributing to the victim's response to resuscitation efforts.

Other stabilization procedures, including splinting of fractures, airway control or intubation, spinal precautions, and institution of intravenous fluids and oxygen, should be accomplished whenever indicated and feasible before transport.

History and Physical Examination

An eyewitness report is helpful, because victims are often confused and amnesic. The history should include a description of the event and the victim's behavior following the strike.

Like any other trauma victim, the victim must be completely undressed to facilitate examination. Special note should be taken of the vital signs, temperature, and level of consciousness. Because many victims are struck during a thunderstorm, they may be wet and cold. Hypothermia should be anticipated and treated appropriately (see Chapter 5).

The awake patient should be assessed for orientation and short-term memory. A cursory mental status examination of the lightning victim may reveal good ability to carry on a "social conversation" that easily hides deficits in fine neurocognitive skills. It is easier to detect deficits when the victim cannot assimilate information, perseverates, or asks repetitive questions. Continuing confusion or a deteriorating level of consciousness mandates CT or MRI of the head to rule out an intracranial injury.

Examination of the victim's eyes is essential to establish pupillary reactivity and ocular injury. However, lack of pupillary reaction or pupillary dilatation should not be taken as a sole indicator of death. Tympanic membrane rupture is an important indicator of lightning strike. Ossicular disruption may be one explanation for a victim's lack of appropriate response to verbal stimuli.

Although the pulmonary system may be affected by cardiac arrest, pulmonary edema, or adult respiratory distress syndrome, it is uncommon to witness these initially. The cardiovascular examination should include distal pulses in all extremities, appreciation of arrhythmias, and evaluation of cardiac damage, including ECG changes. Cardiac enzyme elevations have rarely been reported with lightning injury.

The victim's abdominal examination occasionally demonstrates absent bowel sounds, which suggests ileus or indicates acute traumatic injury, such as contusion of the liver, bowel, or spleen.

The examiner should document any skin changes. The victim's skin may show mottling, especially below the waist. Burns are not universally present or may take a period of hours

to evolve. Notation of pulses, color, and movement and sensory examination of the victim's extremities are important.

The physical findings and mental state of minimally and moderately injured victims tend to change considerably over the first few hours; careful observation and documentation delineate the course so that therapy can be modified if appropriate. The minimally injured victim can almost always be discharged to a responsible person or require only overnight observation, whereas the severely injured person may require intensive care with mechanical ventilation, antiarrhythmic medications, invasive interventions and monitoring techniques.

Laboratory and X-ray Tests

Minimum laboratory examination includes urinalysis (including a test for myoglobin on fresh urine). The more severely injured victim may require tests such as complete blood count, electrolytes, blood urea nitrogen, creatinine, serial cardiac enzymes, and troponin. If the victim is to be placed on a ventilator, arterial blood gases will be necessary; if intracranial pressure monitoring is used, serum osmolality may be required.

An ECG is desirable for most lightning victims. While several types of acute arrhythmias have been reported, they are uncommon. Curiously, QT prolongation is an abnormality sometimes associated with lightning injury and requires careful assessment of the ECG. Radiographs and other imaging studies may be obtained, depending on the presentation and history. Cervical spine imaging should be performed if there is evidence of cranial burns, contusions, loss of consciousness, or change in mentation that would make the physical examination unreliable, or if there are other considerations, such as a fall. The victim who is unconscious, confused, or has a deteriorating level of consciousness requires a CT or MRI scan of the brain to identify trauma or ischemic injury. X-ray studies to rule out fractures, dislocations, and other bony injuries are obtained as indicated.

Treatment[57]

Fluid Therapy

An intravenous lifeline is mandatory for the victim who shows unstable vital signs, unconsciousness, or disorientation. If the victim is hypotensive, fluid resuscitation with normal saline or Ringer's lactate solution is required, with the caution that cerebral edema may develop. Fluid restriction in normotensive or hypertensive victims is recommended because of this risk.

Arterial or central venous pressure monitoring may be indicated. Careful intake and output measurements are necessary in the severely injured patient and require placement of an indwelling urinary catheter. Myoglobinuria is rare and usually transient, so that mannitol diuresis, alkalinization, and aggressive fluid loading used with high-voltage electrical injuries are rarely necessary. However, if burns are severe and extensive, which is rarely the case, fluid resuscitation may be required.

Fasciotomy

Intense vascular spasm with lightning usually is caused by sympathetic instability. The presence of keraunoparalysis, paralyzed and pulseless extremities seen with lightning injuries, should not be treated like similar-appearing traumatized extremities caused by high-voltage electrical injury, but should be treated expectantly.[12,57,151] Steady improvement in the mottled, cool extremity, with return of pulses in a few hours, is the rule rather than the exception. Fasciotomies are rarely indicated unless the extremity shows no signs of recovery and raised intracompartmental tissue pressures are documented. Only one case necessitating fasciotomy has been reported as of this writing.

Antibiotics and Tetanus Prophylaxis

Prophylactic antibiotics are not indicated. Standard therapy should follow culture and identification of pathogens. Exceptions to this rule include open extremity fracture or cranial fracture that violates the dura. Appropriate tetanus prophylaxis is mandatory if burns or lacerations are present.

Cardiovascular Therapy[67,138]

Management of cardiac arrest is standard, including the use of an AED if available. In the victim who is not in cardiac arrest, vasospasm may make peripheral pulses difficult to palpate. Usually, femoral, brachial, or carotid pulses may be appreciated. A Doppler examination may be necessary to locate peripheral pulses and record blood pressure.

If a victim remains hypotensive, fluid resuscitation may be necessary to establish adequate blood pressure and tissue perfusion. Causes of hypotension include major fractures, blood loss from abdominal or chest injuries, spinal shock, cardiogenic shock, and occasionally deep burns similar to high-voltage electric burns. As soon as an adequate central blood pressure is obtained, fluids should probably be restricted because of the high incidence of cranial injuries and cerebral edema.

The victim who is without spontaneous or adequate respirations should be mechanically ventilated until he resumes adequate ventilation, brain death is declared legally, or the physician and family decide appropriately to cease efforts.

Cardiac monitoring and serial isoenzyme and troponin measurements are indicated if there is any sign of cardiac ischemia or arrhythmia or if the victim complains of chest pain. Injury patterns, as well as arrhythmias, have been reported.[57] The indications for antiarrhythmic drugs and pressor agents are the same as for a suspected myocardial infarction.[57]

Transient hypertension may be so short-lived as to require no acute therapy. However, several cases of hypertension have occurred 12 to 72 hours after lightning strike and seemed to respond well to beta-adrenergic blockers and other antihypertensive medications. Use of newer antihypertensive agents with lightning injuries has not been studied.

Central Nervous System Injury[12,30,32,34,39,41,42,54–56,134]

Every lightning victim should have a good neurologic examination. If there is a history of loss of consciousness or if the victim exhibits confusion, hospital admission is warranted. The victim with tympanic membrane rupture, cranial burns, or loss of consciousness, or who shows a decreasing level of consciousness, should undergo cervical spine imaging, brain CT, and possibly brain MRI.

Intracranial pressure monitoring may be a useful adjunct in persons with elevated intracranial pressure.[134] Cerebral edema may be managed with mannitol, furosemide, fluid restriction, and other standard therapies.[134] Although hypothermia was reported to contribute to complete recovery in one victim with prolonged cardiac arrest before resuscitation efforts, there is no evidence that this would benefit all victims.

Early seizures are probably due to anoxia. If there is evidence of CNS damage or if seizures continue after adequate oxygenation and perfusion have been restored, standard pharma-

cologic intervention with diazepam, phenytoin, phenobarbital, and/or other anticonvulsants should be considered.

If paralysis does not improve, causes other than lightning, including blunt injury from a fall, may be responsible. Spinal artery syndrome has been reported, perhaps caused by arterial spasm. A physical therapy program should be initiated before discharge. Unfortunately, in a number of patients, peripheral neuropathies and autonomic pain syndromes develop with continuing dysesthesias, weakness, and trophic changes. These may respond to chronic pain management therapies such as combinations of nonsteroidal antiinflammatory drugs, antiepileptic agents, tricyclic antidepressants, and ganglionic blocks.[8,12,181] Acupuncture may be helpful for resistant post-traumatic headaches that often accompany lightning injury.

In recent years, neuropsychological deficits from lightning have become better appreciated.[63,166,167,188] Heightened anxiety states, hyperirritability, memory deficits, attention deficit, aphasia, sleep disturbance, post-traumatic stress disorder, and other evidence of brain damage should be assessed.[8] Some of these deficits are much like those suffered by victims of blunt head trauma. A rehabilitation program should be instituted early for these patients to return them to as functional a state as possible.[200]

Often, the victim's family and coworkers have difficulty understanding the change in personality that affects many of these victims. Neuropsychological testing to define the injury, establish a baseline, and plan appropriate cognitive therapy may be helpful. Certain personality types may predispose victims to more pronounced neuropsychological symptoms.

The feeling of isolation and change that victims sometimes experience may lead to depression, substance abuse, and thoughts of suicide. Because of unfamiliarity with lightning injuries and their sequelae, many physicians are ill equipped to manage long-term care of lightning victims or may be so skeptical that the victim and family become frustrated and angry with their care.

Referral to Support Groups and Other Information Sources

Referral to Lightning Strike and Electric Shock Survivors, International (LSESSI), a support group founded in the late 1980s, can be of substantial help to victims and families. LSESSI is located at P.O. Box 1156, Jacksonville, NC 28541-1156 (Tel: 910-346-4708, email: lightning1@ec.rr.com, website: http://lightning-strike.org).[71] Other useful websites are www.uic.edu/labs/lightninginjury, www.struckbylightning.org, and www.lightningsafety.noaa.gov.

Burns[62,151]

Lightning burns may be apparent at the time of admission but more commonly develop within the first few hours. They occur in less than half of lightning survivors and are generally superficial, unlike high-voltage electrical burns, and seldom cause massive muscle destruction. Myoglobinuria is infrequently seen, so vigorous fluid therapy and mannitol diuresis are not usually indicated. In the rare instance of myoglobinuria, intravenous mannitol and alkalinization of the urine with bicarbonate may be used. Overzealous hydration from the presumption of similarity to high-voltage injuries with resultant cerebral edema has probably harmed more lightning victims than has myoglobinuric renal failure.

Lightning burns are generally so superficial that they do not require treatment with topical agents. In the unusual instance of deep injury, topical therapy should be standard. The findings that lightning burns are superficial and do not require active surgical intervention is reinforced by Matthews and coworkers.[151] Their paper provides a useful guide to the remote need for surgical intervention in lightning injury.

Eye Injuries[27,78–80,85,97,111,158,159,184,201]

Visual acuity should be measured and the victim's eyes thoroughly examined. Cataracts are not uncommon. Eye injuries should be treated in standard fashion and may require referral to an ophthalmologist. Dinakaran and coworkers[78] report a case in which cataract can be ascribed to a telephone-transmitted lightning strike. Case reports of successful treatment of optic neuritis with high-dose steroids similar to those used with spinal cord injury have been reported, but because there were no controls, it is not known if recovery would have occurred without their use.

Ear Injuries[25,89,93,96,114,153,154,161]

Loss of hearing mandates otologic evaluation. Simple tympanic membrane rupture is usually handled conservatively with observation until the victim's tissues heal. Sensorineural damage to the auditory nerve resulting in hearing changes, dizziness, and permanent tinnitus and facial nerve palsies are not uncommon. Ossicular disruption or more severe damage may necessitate surgical repair. Otorrhea and hemotympanum suggest basilar skull fracture. Complaints of pain around the angle of the victim's jaw should lead the physician to a search for occult fracture of the styloid process and other musculoskeletal damage. Gordon and coworkers[93] hypothesize that the injury must include components other than blast and consider burns the most likely. The present authors concur and cite the possibility of the cranial orifices being portals of entry for electrical current.[6] Mora-Magana and coworkers[153] provide a review of the otic effects of lightning.

Pregnant Victims

If a pregnant woman is struck, fetal viability should be assessed, including fetal heart tones, ultrasonography to observe fetal activity, and other standard methods.

Other Considerations

Gastric irritation is occasionally seen. A nasogastric tube is appropriate if ileus or hematemesis occurs. An abdominal CT scan may be indicated in comatose patients who remain hypotensive, because intestinal contusions and hemorrhage have been reported. It is common for the survivor to report continuing nausea and other signs of postconcussive syndrome for several weeks to months.

Endocrine dysfunction, perhaps as a result of pituitary or hypothalamic damage, including amenorrhea, impotence, and decreased libido, has occurred in some victims. Spinal cord or sympathetic nervous system injury or postinjury depression as well as side effects of medication used for pain control may also be a cause of impotence, decreased libido, and sexual dysfunction after lightning injury.

Pronouncing the Victim Dead[22,29]

Dilated pupils should not be taken as a sole sign of brain death in the lightning victim. It is always necessary to exclude other causes of dysfunction and eliminate them before death is

declared. Hypothermia with lightning injury may cloud end-of-life decisions. Charleton[29] states that lightning injury carries a high survival rate, so persistence in resuscitation should be the norm. His notion seems sensible, but his statement has drawn substantial correspondence. Campbell-Hewson and coworkers,[22] for example, raise the notion that it is a falsehood to claim that resuscitation after prolonged arrest is more likely with lightning-induced arrest. The authors entirely agree with this and agree that the notion arose from one misreported case. Certainly, if the victim has not regained a pulse after 20 to 30 minutes of resuscitation, it is reasonable to cease cardiopulmonary resuscitation.

PRECAUTIONS FOR AVOIDING LIGHTNING INJURY*

Lightning strike usually involves individuals, and as previously discussed, injuries are also usually individual. Prevention is more important than cure, and guidelines for individual safe practice have been produced (see www.lightningsafety.noaa.gov, for example).

Other dangerous circumstances, however, include the possibility of strikes to areas where large crowds have gathered, for example, at major sporting events. The possibility for multiple injuries exists, not only to the players but also to the observers.

Lightning Safety Guidelines

A multidisciplinary group of internationally recognized lightning experts updated existing lightning safety guidelines in 1998.[66,106] The new guidelines are available at www.uic.edu/labs/lightninginjury and address varying sizes of groups and evacuation times. Most lightning deaths and injuries occur in isolated events affecting only one person. Because 25 million cloud-to-ground flashes strike the United States every year, it is impractical for the NWS to warn of every potentially dangerous lightning event, so the key to safety is individual education and responsibility.[64,65,135,136]

The exceptions to this approach of personal responsibility are (1) when adults are in charge of groups of children, and (2) situations where responsibility is assumed by organizers and operators of facilities where large crowds are expected such as sporting or entertainment events. For groups of children, adults must assume responsibility and have a plan for evacuation. Struck by Lightning (www.struckbylightning.org), an organization formed to disseminate safety information, seeks to make "When thunder roars, go indoors" as well known a phrase to children as "Stop, drop, and roll" is for fire safety.

Operators of recreation facilities and promoters of large events carry the responsibility to be aware of threatening weather, determine when events should be canceled, and have a plan of action that includes proper warning, shelter guidelines, and all-clear signals.[157]

Studies of *Storm Data* casualties relative to cloud-to-ground flashes from the National Lightning Detection Network have shown many to occur during infrequent lightning. Casualties in Florida occurred before, during, and after the peak lightning activity in a thunderstorm.[107] Lengyel at the University of Oklahoma found that half of the lightning victims had enough warning of cloud-to-ground lightning near them before being struck so that application of planning and the 30-30 rule would have been beneficial. The rest of the victims were struck with less warning of the imminent threat of lightning, so lightning detection data and other methods of thunderstorm forecasting can be helpful in addition to direct local observations.

Lightning Safety Plan

Make a plan that will identify a series of steps.[157] The plan should identify safer places to go, who makes the decisions, how far in advance such actions are taken, how long to stay at the safe location, and backup people to make decisions at nonstandard times such as weekends.

Before working in the open or going on excursions, be aware of weather forecasts and conditions. If thunderstorms are forecast later in the day for the time and place of interest, pay attention to updated forecasts and weather conditions. Use NOAA weather radio or other weather-specific source of information. Seek a safe location if a severe thunderstorm or tornado warning is issued. Appoint a spotter to watch for lightning and provide appropriate warnings to people. Severe thunderstorm and tornado warnings issued by the NWS identify specific counties and periods of less than an hour when high winds, large hail, and/or tornadoes are expected, but such warnings do not identify the lightning threat at a particular place or time. So, a warning from NOAA weather radio or a similar source should be considered only as a wake-up call to the general threat of thunderstorms. Then, careful visual attention needs to be paid to nearby lightning and thunder.

Approaching Thunderstorm[31,106,108,146]

As a thunderstorm approaches, use the 30-30 rule.[106] This rules states that when the time between seeing lightning and hearing the thunder from that flash is 30 seconds or less, people are in danger and should be actively seeking shelter (the first 30 of the 30-30 rule). A longer count should be used by persons who have a long evacuation time, such as those involved in group sports, mountain climbers, the elderly or infirm, hikers, and golfers, as well as those who are responsible for others. A count of 30 seconds indicates that the lightning was 10 km (6 miles) away. There is still some risk of lightning with a count of more than 30 seconds, but this measure covers about 80% of all subsequent cloud-to-ground flashes that occur after a cloud-to-ground flash.[108,146]

If you are wearing a backpack that protrudes above your head, it increases your height and risk. It may be worthwhile to shed it because you can run faster to a safer place. Otherwise, it is a myth that wearing or carrying metal attracts lightning.

It is not uncommon for a portion of the sky to be blue when lightning hits. At least 10% of all cloud-to-ground lightning strikes occur with no rain at the location of the strike, so that people should not delay until the rain sets in. Pay more attention to the lightning than the rain.

End of Thunderstorm[108,146]

At the end of the storm, do not underestimate the danger of lightning. Outdoor activities should not be resumed until at least 30 minutes (the second 30 in the 30-30 rule) after the last lightning is seen or the last thunder is heard during the day.[108,146]

At night, flashes may be visible low on the horizon inside tall thunderstorms 80 km (50 miles) or more away. These are not of much concern unless the lightning channel itself is visible to

*References 64–66, 101, 104, 106, 120, 150, 157, 173–177, 191, 194, 203.

the ground. Waiting less than 30 minutes may be appropriate when limited number of people can very quickly reach a safe place, but the full 30 minutes should be used by persons who have a long evacuation time, as mentioned for the prestorm stage. This measure offers nearly complete safety from cloud-to-ground lightning. Every year, a number of people are killed while running to their vehicles during a lull in the thunderstorm instead of staying in the safety of the mall or grocery store.

Safer Places Inside[16,101,173,203]

Only two places are "safe" from lightning: a substantial building and a metal-topped vehicle. A substantial building in this context is a structure where people normally live or work. Such buildings have wiring and plumbing that take a direct strike to the structure and facilitate its pathway to ground, although both structural and equipment damage may be high in the process. Another common entry point into a substantial building occurs when a flash strikes an outside power or telephone pole or tower, sometimes resulting in the current being carried inside the building through the wiring. Conducting paths to be avoided include electrical appliances, plumbing fixtures, and corded telephones and headsets. As long as a person is not in contact with these conducting paths inside the building, or within several feet of them, this is a safer place from lightning.

In contrast, small buildings, such as golf shelters, rain shelters, bus shelters, car ports, and garages, may actually increase the person's risk, depending on the size and height of the building, because side flashes can occur to the occupants. Side flashes from nearby trees to buildings are especially dangerous; 76% of side flashes occur when a tree is within 20 meters (66 feet) of a building.[16] Tents offer no protection because there is no safe path for the current to travel around a person when a flash strikes at or near the tent.[203] Material such as a foam pad inside a tent offers no protection against the intense current traveling across the ground. Tent pole material—e.g., metal versus fiberglass—has no effect on protection.

Vehicles with metal tops provide a safer haven because the current diffuses around the occupants through the metal, creating a Faraday Cage, and usually arcs to ground from the body of the vehicle. Vehicles with a cloth top, such as a convertible, or recreational vehicle with only a roll bar are not safe. Antennas and other tall portions of a vehicle may be the most frequently struck features. When the current travels around the outside of the vehicle, a person should not be in contact with conducting metal parts along the path, just as a person inside a house should not be contact with wiring or plumbing. When a car, truck, bus, or van is struck by a flash, a person inside may be injured and the vehicle may be damaged, but being inside is greatly preferred to being in the open when the strike occurs. It is a myth that rubber tires provide insulation inside a vehicle; it is the metal body that generally affords protection.

A small group of people can be protected by using cars and vans around a youth soccer field, for example. Some golf tournaments place rented school buses around courses for lightning protection involving larger numbers of people. But a substantial building is preferable to a vehicle. During an after-school activity near a school, an alternative is to assure access to nearby school buildings where the playing fields are located.[173]

Darveniza has patented two options for lightning safety (Fig. 3-23).[75] Figure 3-23A shows a pyramid made of three telescoping poles, each of which is short enough to fit into a golf bag. Figure 3-23B shows a proposed lightning conduction cage that

A

B

Figure 3-23. **A,** Lightning protection for a small group made from telescoping rods that are small enough to fit into a golf bag or large backpack. **B,** Four-cornered lightning protection cage for a golf cart. (**A** courtesy M. Darveniza, pictured L with white beard; **B** courtesy M. Darveniza, pictured at right with hand on chin.)

could be prospectively built into golf carts, but the inhabitants would have to be trained not to touch any of the down conductors, including putting their arm across the back of the cart.

Unsafe Places Outside*

No place outside is safe during lightning. Consider the situation of a group of trees with some open spaces between them. Lightning tends to strike the tallest object, such as a tree. The tree acts as the connection to bring current to the ground. A person near the tree will be in danger because the current tends to travel in several arcs outward across the ground from the trunk. It is unknown where these arcs will travel across the ground; their paths do not appear to be related to tree roots or any other surface or subsurface feature. Trees are also dangerous because lightning can reach the ground through overhanging branches. In addition, lightning can send a side flash outward from a tree trunk.[16]

Similarly, a person standing in an open space between trees is in danger because, as mentioned earlier, a leader attaches to the tallest object when its lowest tip reaches down to within 27 to 46 m (30 to 50 yards) of the surface of the earth. Seeking a clearing to avoid trees makes a person the tallest object in the clearing and therefore more likely to be struck. Similar approaches apply to poles and other tall objects. For these reasons, it is not possible to identify where lightning will travel in order to avoid it, so that no place outdoors provides safety.

On or near a body of water, a person is often the tallest object, or close to one. Lightning tends to strike a person or the tallest parts of a boat projecting above the surface of the water. Plan ahead to avoid thunderstorms that are expected to occur while swimming or during other water-based activities. Compare how long it will take to reach safety on shore with how long the storm will take to reach you while on or in the water. A large sailboat or powerboat can be protected with lightning rods and grounding equipment attached to a metal keel or understructure (www.marinelightning.com).

Small caves, ditches, and valleys provide no protection from lightning. Sheltering under a small outcropping or overhang may actually increase a person's risk of injury, because lightning that has hit a hill tends to literally "drip" onto the person with the rain as it arcs over the ground, especially over small jagged prominences and gaps.

There is no easy remedy to prevent people from getting "caught" in a thunderstorm; more often than not, they have ignored lightning safety guidelines and made several bad decisions. Nothing can substitute for taking the proper initial precautions: know the weather, have a lightning safety plan, and follow the plan.

Wilderness†

No action will achieve safety from lightning in the wilderness away from a substantial building or metal-topped vehicle. There are only two general approaches to lightning safety in the true wilderness. These approaches are to avoid the risk in the first place, and the other is to accept the fact that a lightning risk exists. None of the following are known to lower the lightning risk: being inside a tent, sitting on a pad, removing metal objects from a person, or any configuration of the body.

Avoidance refers to taking into account the lightning threat as it exists. For example, there is a very real threat of lightning during the afternoon in summer over high terrain in the western United States The threat in this case can be avoided by climbing very early in the day before lightning begins over higher terrain, choosing to climb during seasons when lightning is much less frequent, and postponing the climb on a day with thunderstorms in the forecast to another day without the threat of lightning. Lightning safety, like most other safety precautions, may be inconvenient but necessary if injury is to be avoided.

The pressure of a schedule or other factors may be such that a wilderness activity will take place in the presence of a very real lightning threat. Then, a hiker or climber has accepted the personal risk in the same manner as the risks from dangerous animals or other natural hazards. Nothing can be done in the open to be safe from lightning on a mountain top on a summer afternoon where there is more lightning than in surrounding valleys.[36] A knowledgeable and careful hiker will no more seek routes and times that are prone to lightning than enter an area with a high risk of avalanches, bears, or hypothermia.

Large Group Safety

Crowd Safety and Protection‡

Special factors applying to crowd safety have been discussed briefly and rarely.[3,150] Reports of multiple casualties exist[2,119,122,137,163]; however, concerted strategies for crowd protection are scant. Gratz discusses crowd and stadium protection strategies in some detail, and rightly points out the competing issues in crowd protection.[95] He highlights the need for a proper management strategy. Andrews[3] examined the matter from the perspective of large crowds (up to 100,000) at a major event (The Olympic Games, Sydney, 2000) and while Makdissi and Bruckner[150] supported these views, they also discussed smaller groups (order of 100s). Both stress the importance of a responsible person having authority over institution of procedures.

There will be a gradation of strategy depending on crowd size. For example, a weekend sporting match with 100 people present may require practices akin to the individual practices above. However, a major event with crowds of 100,000 may require special considerations. Which of the strategies to implement will be a matter of judgment for each circumstance, but the "responsible person" and a preexisting safety plan are common themes to both.

An initial risk assessment is useful. Factors include the keraunic level of the region, and also recent past history of storms and lightning. In the Sydney case, while the probability of strikes could be predicted based on thunderday levels, it was also noted that 11 severe storms had occurred in the area at the relevant time of year in the past 10 years. Thus, the history transcended the more usual analysis. Other factors that affect the risk include time of year, time of day, local terrain, and the presence of structures providing a degree of "natural" protection. For example, the risk on a city street surrounded by skyscrapers is much less than on an open plain in Florida. Consideration of each factor leads to overall risk assessment.

*References 31, 33, 36, 64–66, 101, 104, 106, 120, 150, 157, 173–177, 191, 194, 203.

†References 26, 33, 36, 64–66, 101, 104, 106, 120, 150, 157, 173–177, 191, 194, 203.

‡References 3, 64–66, 95, 101, 102, 106, 120, 150, 157, 173–177, 191, 194, 203.

Preliminary Factors in Crowd Protection[3,150]

A thorough appraisal of the circumstances of each event must be made to identify risk, not only from lightning but also from items other than lightning, for example, the ability to withstand accompanying natural forces other than lightning, such as wind, driving rain, and hail. These are beyond our present scope but are part of a total risk management strategy.

Special guiding principles for lightning protection of a crowd include:

1. Crowd management, as well as the provision of "simple" technical protection from a strike itself. Psychological and behavioral management strategies are equally important in a crowd environment.
2. Any form of Franklin rod placed in a position to protect a venue or a crowd. This generally needs to be aesthetically appealing or invisible to media observation.

Protection of Crowds Compared with Individuals[3,95,150]

With a single person, for example, a competitor, or even a small team of competitors, placement in a protected zone may be all that is needed if risk eventuates. When to do so, however, is the important question. Such a person or group has easy and quick mobility, and the protected space requirement is small. The strategy that can be used is similar to that for individual protection.

With a crowd, managing a protection strategy and its implementation is as important as the technical nature of the strategy itself. Crowd behavior, possibly encompassing panic, can be threatening and dangerous. This can impede an orderly safe activation of a strategy, and a stampede could endanger life. Crowd education is therefore important, and information and maps should be provided, with reassuring explanations, both written and verbal, for what is to happen at each stage.

The psychological impetus to continue a high-level competition in the face of risk is high. Convincing an individual or a team to cease playing may be difficult, so authority to do so needs to be clearly identified. The possibility of competitive advantage to one side when compulsorily stopping competition needs to be considered. Authority needs therefore to be firmly vested and impartial.

There are two options for lightning safety in the large crowd context:

1. The first option is to move an entire crowd (evacuate the crowd) to a protected space in the face of risk. This depends on the size of the particular crowd, availability of protected space in close proximity, and sufficient egress pathway for the crowd to move in an orderly fashion. This latter orderliness also necessitates that the procedure be within the competence of marshals and their ability to maintain order. Such evacuation is appropriate for smaller bleacher seats, perhaps with an individual capacity of 500 people. Examination of a particular site may indicate the presence of protected spaces. In some cases this could be an adjacent warehouse or gymnasium. In other cases, it is possible to evacuate a crowd from its spectating position into the zone protected by the (then empty) stand, including underneath it. Examination of any possible side flash or contact potential or earth potential rise is required. For these reasons, ultimately, evacuation is not always a preferred option.
2. In the second case, it may not be realistically possible to evacuate a very large crowd, for example those in a stadium with a capacity of tens of thousands and fixed entrances and exits. The option, therefore, is to protect the crowd "in situ." That requires technical protection so that these crowds are not at risk (statistically) of being struck while remaining in place, with lightning being intercepted by a mechanical installation. This requires the design of protection structures. If this strategy is adopted, then one has to be assured that the stands will withstand any wind or hail that is possible, and to accept that such a crowd might get wet.

Details of the Alternative Strategies

Evacuation

Evacuation is mostly appropriate for smaller "crowds." Evacuation sites need to be available and signage appropriate. Sturdy buildings, warehouses, and gymnasia all provide suitable sites. The site needs to be of such a size that the numbers can be accommodated in relative comfort. It is inappropriate, for example, for 500 people to be crushed into a single room. Event organizers may wish to establish goodwill by providing refreshments. Sanitary facilities must exist. The final consideration is that evacuation must be achieved with appropriate haste, given the exit paths from the stands, transit paths to the protected site, and entrance and settling time into the protected domain.

Protection in Situ[3]

Protection in situ is often the most realistic option for many sites, given that crowds may be large, spare building space may not be available for the numbers required, and exit routes are generally easily saturated and become mobility-limiting factors. This tacitly accepts that being wet in a stand is an unfortunate but accepted consequence of storm activity.

In addition, individuals in transit between stadiums and fields need to be considered if multiple venues are involved. This implies that the venues themselves not only require protection, but transit paths also require protection.

When to Protect*

A key to an evacuation protocol, and certainly a key to advising players when an event is to be compulsorily paused, is the question of when to activate an evacuation protocol. The following is an action strategy.

1. Infrastructure. It is proposed that there should be a Lightning Location System (LLS) display unit present within a venue if the event is of large enough size to warrant it. It should be manned by persons skilled in its interpretation and with absolute authority to implement the phases of a management protocol as outlined below. Such authority could conceivably be shared between one or two individuals. Activation of a strategy is not a matter for a committee debate, but rather the application of firm principles known in advance.

From the display, the operator is required to obtain the speed and direction of travel of lightning activity. The important parameter is whether, given speed and direction of travel, a given storm will pass within 10 km (6.2 miles) of a competition venue. This is applied below.

*References 3, 64–66, 95, 101, 104, 106, 120, 150, 157, 173–177, 191, 194, 203.

If the event is not large enough to justify installation of LLS equipment, then the decision-maker may resort to visual observation, public meteorology advice, and websites with real-time weather and lightning information.

2. Principles. All lightning protection should be in place and operative when there is any lightning activity within 10 km (6.2 miles) of a venue. Let us then say that a half hour is the time necessary to activate the protocol before this distance threshold is breached. This time varies, depending on the time required to activate the protocol (i.e., evacuate). Thus, phases of a management protocol are required to lead up to the highest level of alert, this being at the stage when lightning activity crosses the 10-km (6.2-mile) threshold from a venue.

The operator of the LLS observes the developing lines, cells, and clusters of thunderstorms, and estimates the speed and direction of each to predict if and when they will enter the 10-km (6.2-mile) zone. If this is satisfied, then the half hour (or appropriate time) leadup plan is activated.

The risk of lightning injury depends on distance from lightning activity. In this case, all protection strategies need to be in place when lightning activity reaches 10 km (6.2 miles) from a venue, this being considered the maximum distance at which a lightning strike constitutes a risk. Just when to start implementing the strategy, however, depends on the leadup time required to have the strategy, such as evacuation, in place. The controller's tasks are therefore:

- To observe areas of lightning and watch their location, travel direction, and speed
- To determine when lightning will cross the 10-km (6.2-mile) threshold. When the lightning is still an hour away, a formal "watching" phase is declared
- To calculate when lightning will cross the 10-km (6.2-mile) threshold and to activate the evacuation strategy
- To be aware when lightning crosses the 10-km (6.2-mile) threshold, and to declare the highest level of activation; all strategies are then in place and active

3. Protocol for Activation of Lightning Risk Management. The action protocol is divided into three phases:

- A *yellow* phase alert, which is a warning that lightning activity has been detected and may impinge on the event
- An *orange* phase alert, when it has been determined that the activity is a half hour from crossing the 10-km (6.2-mile) threshold and requires definite action
- A *red* phase alert, when lightning is an immediate and imminent risk on or inside the 10-km (6.2-mile) threshold

A yellow phase alert is declared when lightning activity is 1 hour away from a venue. An orange phase alert is declared when lightning activity is one half hour away from a venue. Depending on the venue's requirements, all competitors in competition should cease activity, and evacuation is required.

Patrons in stands protected in situ should be advised to remain in position and to expect rain. Individuals in transit should be advised to complete their transit as soon as possible, and those at cars and buses should be advised not to commence transit, but to remain in their vehicles. Individuals outdoors should be advised to move to safe areas as shown on maps provided for the purpose.

A red phase alert is declared when lightning activity is within 10 km (6.2 miles) of a venue. By this stage, all movements and evacuations, if necessary, should be complete, with the venue now secure from lightning risk.

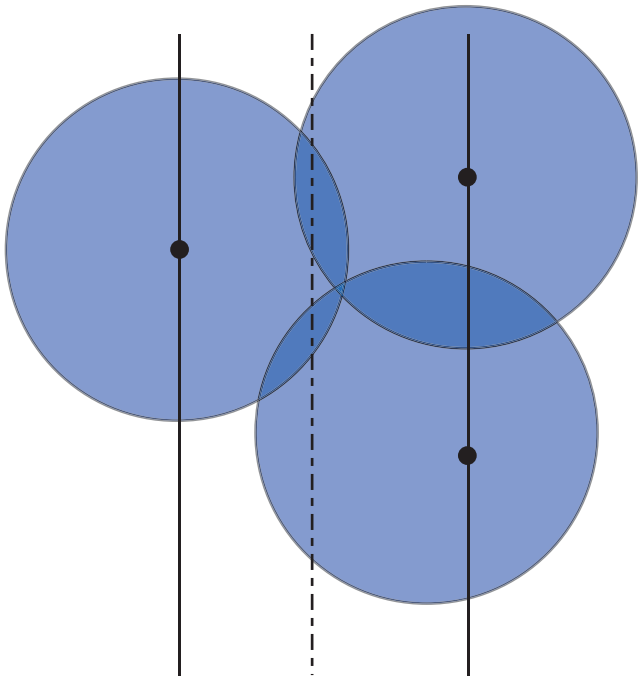


Figure 3-24. Protected zones on a pathway from light poles of varying height. (Redrawn from Crowd protection strategies: Experience from the Sydney Olympic Games, 2000. Presented at International Conference on Lightning and Static Electricity, Blackpool, UK, 2003.)

Protection in Situ[3]

The Franklin rod is often the basis of lightning protection schemes. Many existing structures can be turned into such rods with ease without detracting from appearances. Examples are given below. It is emphasized that these are illustrative only, and proper design is needed when these are being implemented.

Figure 3-24 shows a pathway with light poles of a given height. The spacing of these poles may be selected to protect the pathway, depending on the height of the poles and width of the path.

Figure 3-25A shows the "natural" protected zone around a temporary stand that might form an evacuation region if other risks are accepted. Franklin rods (see Figure 3-25B) added along the back of the stand extend this zone, and also add some in situ protection. Useful Franklin rods are formed by flagpoles that have the required "look." A horizontal wire at a given height above a stand also provides in situ protection (see Figure 3-25C). These can be made all but invisible, especially to media cameras. Alternatively, they can carry banners, lights, or media cameras.

Figure 3-26 shows horizontal wires providing protection to a group of stands. The width of the protective corridor depends on the height of the catenary, and the position of the corridor can be altered as a design parameter for effectiveness.

Figure 3-27 shows a stadium for a field game like baseball, where an existing stand provides some protection, as do lighting pylons. The addition of overhead wires can complete the coverage.

Figure 3-28 shows the main stadium stylistically and shows how open stands at either end of the stadium can be protected by fine wires, which are all but invisible.

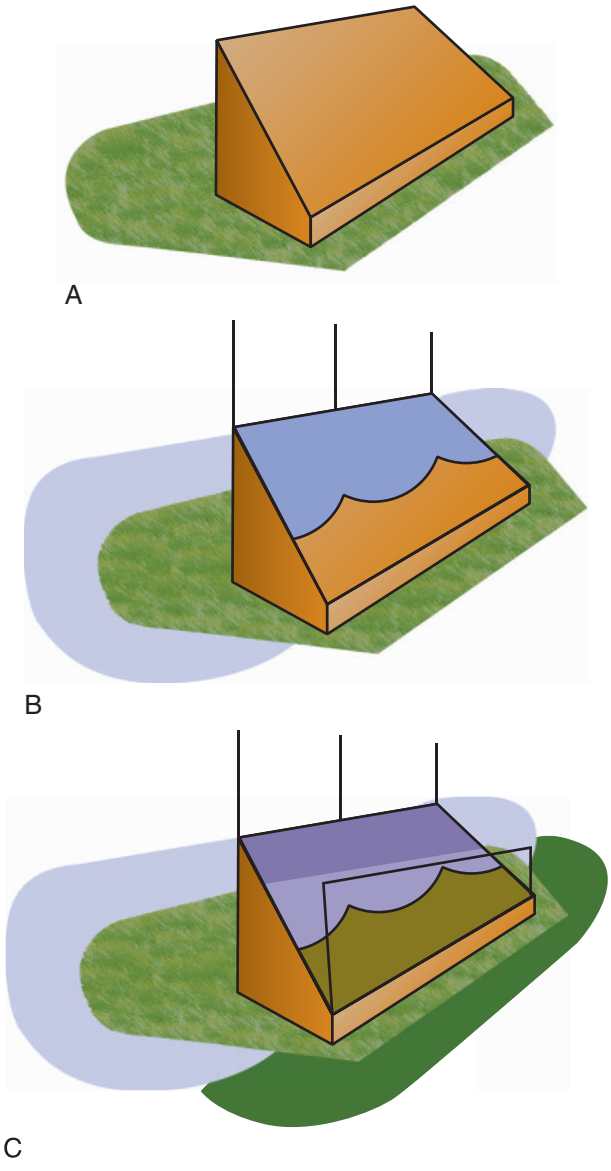


Figure 3-25. **A,** Natural protection zone of a stand. **B,** The effect of flagpoles implementing Franklin rods. **C,** The added effect of a catenary added to a stand. (Redrawn from Crowd protection strategies: Experience from the Sydney Olympic Games, 2000. Presented at International Conference on Lightning and Static Electricity, Blackpool, UK, 2003.)

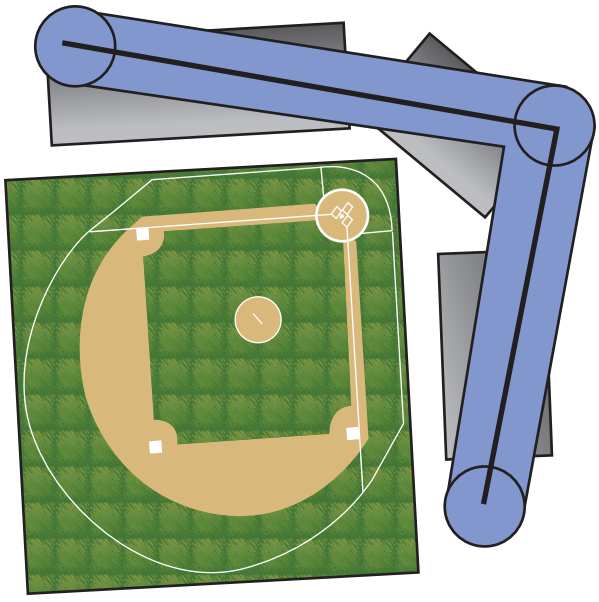


Figure 3-26. Use of catenaries to protect several stands. (Redrawn from Crowd protection strategies: Experience from the Sydney Olympic Games, 2000. Presented at International Conference on Lightning and Static Electricity, Blackpool, UK, 2003.)

CONTROVERSIES IN LIGHTNING INJURY[61]

Keraunomedicine is an expanding and developing discipline. Much remains to be learned, and knowledge at present remains partial.

Problems with Expert Reporting

This is especially evident when expert and legal assessments are made of victims of the injury. For example, the area of the psychological effects of lightning injuries is under active research and still needs clarification. The effects are well documented in the literature. Nonetheless, psychiatric and psychological standards (e.g., the *Diagnostic & Statistical Manual of Mental Disorders* as well as other standard teaching and textbooks) have not yet incorporated the syndrome into their formularies. Specialties move slowly, of course, but this also highlights that victims represent a numerically small group in routine practice, and routine practitioners, specialists in their own discipline though not in keraunomedicine, are rarely aware of the literature in the subject. Consequently, we see established practitioners without the benefit of seeing many of these cases, or familiarity with the literature, struggling for a diagnosis and trying to fit the "new" syndrome into existing categories.

Research Methodology Problems

Cooper[51] has drawn attention to further features of what is not yet known in lightning injury and documents methodology difficulties. She states that without knowledge of the basic physiology, only general symptomatic aftercare can be rendered. It may be more desirable to have specific treatments or even early interventions that might stop or change the course of the injury cascade that is precipitated by the initial injury.

Research on lightning injuries is difficult (Box 3-4). There are no public health regulations that require reporting of lightning injuries, so cases are difficult to collect and survivors are difficult to locate for studies in any systematic fashion.

Although it may be convenient to use such populations as the membership of LSESSI as a study group, people who join support groups differ from those who do not; they represent only a subset of survivors, and they may have systematic biases developed from the services and materials that LSESSI supplies to its members.[71] In addition, limited research can be done with them due to ethical limitations on human research, national

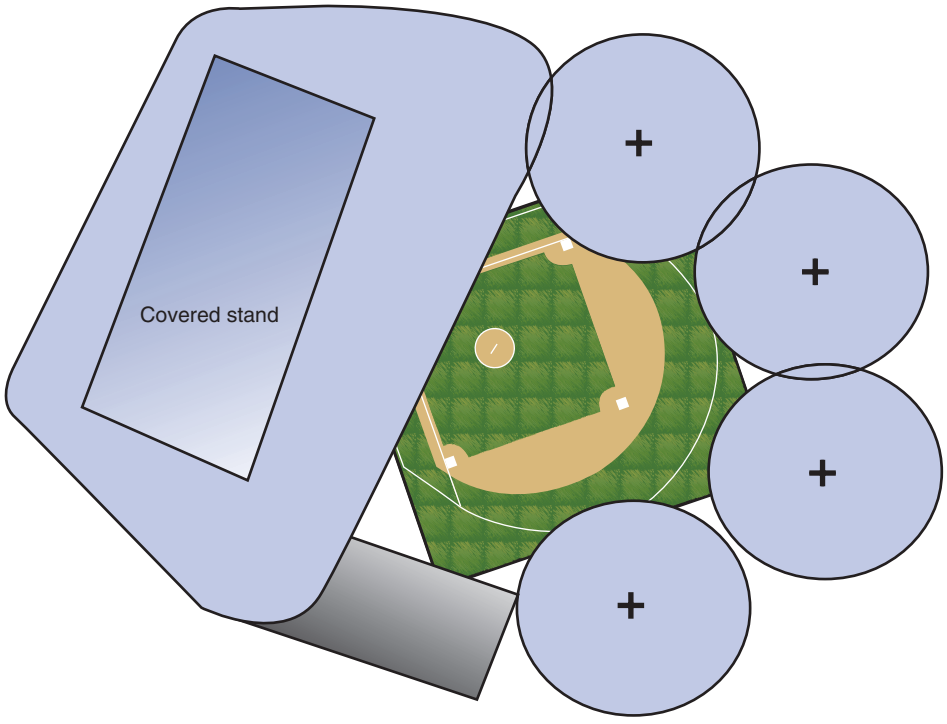


Figure 3-27. Effect of existing covered stands and lighting stanchions. (Redrawn from Crowd protection strategies: Experience from the Sydney Olympic Games, 2000. Presented at International Conference on Lightning and Static Electricity, Blackpool, UK, 2003.)

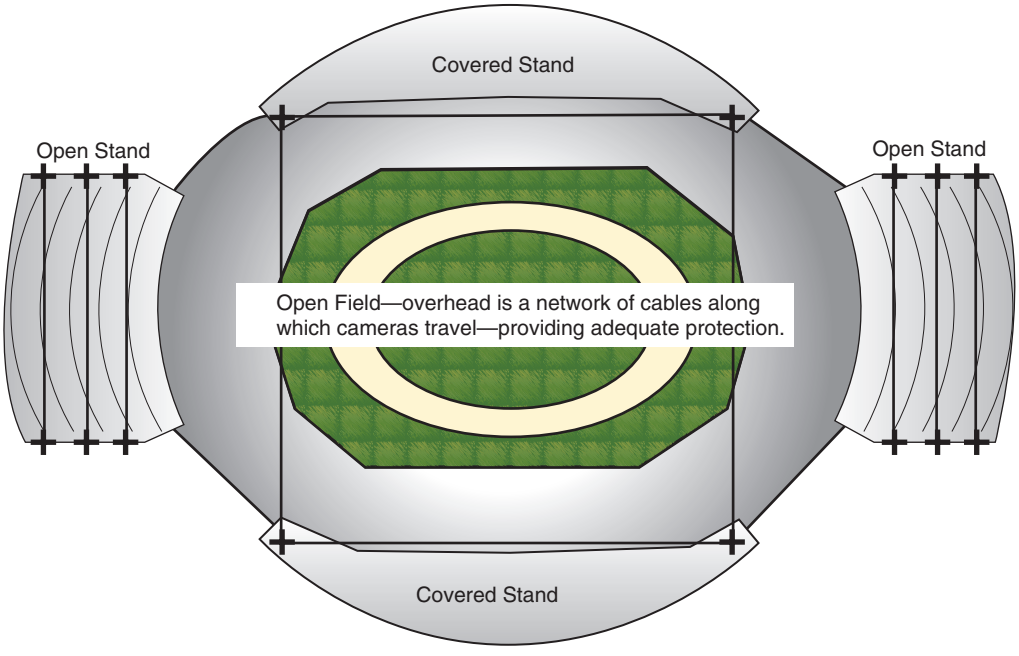


Figure 3-28. Open field—a properly installed network of overhead cables that can double for moveable cameras can also provide adequate protection. (Redrawn from Crowd protection strategies: Experience from the Sydney Olympic Games, 2000. Presented at International Conference on Lightning and Static Electricity, Blackpool, UK, 2003.)

Box 3-4. Research Problems

HUMAN RESEARCH
Recruitment of cases
Study biases
Dispersion of subjects
Cases must be free from
- Diabetes and other neuropathic illnesses
- Drug history
- Psychiatric history
- Blunt head trauma

ANIMAL RESEARCH
Expensive—both animals and equipment
Large number of animals for some studies
Difficult signal processing problems
Monitoring equipment design
Shock timing control considerations
Definition of dose
Standardization of dose
Flashover effect

MOLECULAR BIOLOGY
Cell culture, blood levels, and so on.
Requires specialized techniques
Bioengineering, collaboration
Expensive

dispersion, and lack of funding for the research, among other difficulties.

In order to sort out the lightning electrical effect from other etiologies, it is necessary to obtain relatively recently injured patients (no more than 6 to 12 months) with a "clean" history. There must be no past history of alcohol or drug abuse, head injury, psychiatric problems, concurrent medical problems that cause neuropathic pain or psychiatric symptoms, and no blast effect during the injury that would confound the investigation of the electrical effects of lightning. Unfortunately, this further narrows the available pool. Difficulties therefore compel the selection of other models, raising problems of applicability.

Because it is difficult to recruit volunteers to stand in a thunderstorm, animal work is a reasonable alternative. Kitagawa, Ishikawa, Ohashi, and Andrews have all done credible research using animals.[6,112] Cooper has built a reasonable rat model, inducing most of the clinical signs that are seen in humans.[68–70] Unfortunately, such research is expensive, time consuming, and sometimes requires large numbers of animals to show significant differences. Most problematic is the delivery of a "standardized dose" that can reliably reproduce a specific pattern of injury.

Many hurdles must be overcome in research equipment design, anesthetic choice, and animal care, as well as shock timing and delivery, given instrumentation requirements. Monitoring equipment must be electrically isolated from the animal and shocking platform, or the connections may preferentially transmit the lightning shock to the equipment, not the animal, clouding the experiment and destroying the equipment. Some anesthetics are neuroprotective, while others affect cardiac function. Temperature control in anesthetized rats is a significant issue. Animals that are studied for neurocognitive injury using water maze and other behavioral models need a standardized quiet environment, which is not always possible in the cramped quarters of most animal facilities. In survival or cardiac studies, timing of the shock to either target or avoid the "vulnerable period" must be done on a statistically predictive basis, because direct sensing of the specific targeted cardiac cycle would again result in damage to the equipment.

Box 3-5. Why Do 90% Survive?

Lightning characteristics?
  "Type" of lightning
  "Dose" of lightning delivered
  Timing of hit
Other meteorologic conditions?
Physical characteristics of those "hit"?
Where people were/what they were doing?

Areas of Specific Controversy

Survival Statistics

Box 3-5 lists factors concerning the unknown issues as to why 90% of casualties survive.[44] It is possible that characteristics of a lightning strike or possibly the mechanism by which the current impinges on the individual may be different for lightning fatalities. It is possible that timing of the lightning strike may be the important factor, particularly if it hits during a more vulnerable portion of the cardiac cycle. Although it is possible, it is unlikely that the physical characteristics of people differ sufficiently to cause a difference in mortality. It is unknown if the "lightning desperation position" (formerly known as the lightning safety position or the lightning crouch) changes outcome or, for that matter, if any position is useful.

Remote and Psychological Symptoms

A further area of active research is the etiologic origin of symptoms not directly in the line of passage of current. This is as much for electrical injuries as for lightning injuries.[155] Neuropsychological testing consistently suggests an organic origin to the deficits. Imaging, imperfect as it is, suggests unspecified organic impairment. Yet the exact nature of the impairment, its relation to functional pathologies, and its origin remain obscure.

Increasing awareness of the role of the spinal cord in nociception and existence of proximally directed neural pathways may provide a fruitful connection. Release of humoral transmitters may play a role. The importance of premorbid personality predisposition is unknown.

It has been noted by some that the neuropsychological constellation bears resemblance to the psychological effects of other syndromes, like head injury and the response to autoimmune disorders. It is possible the symptom complex that is seen represents a "final common pathway" of brain injury due to many etiologies.

Box 3-6 lists other possible factors yet to be verified.

Technical Matters

The degree of lightning injury or "dose" may vary considerably with the mechanisms of injury (e.g., direct, splash, contact), few of which have been modeled or quantified. Other technical factors regarding the interaction of a lightning stroke with the

Box 3-6. Possible Injuring Forces

Blunt trauma——explosive injury
Structural changes—direct damage
  Pathway of the injury
  Orifice entry
Flashover—how much goes through versus around
Neurochemical changes
  ANS effects
Electrical effects
  Electroporation
  Cellular level mechanical effects
  Cellular level enzymatic effects
  Subcellular organelle damage

body are poorly known, such as the effect of multiple return strokes. Although flashover is known to occur, the course of energy going through the victims versus around them, and the duration of energy flow initially or with return strokes is unknown. While these factors have been calculated based on engineering assumptions, actual measurements have never been done on research subjects or simulated subjects to test the assumptions.

Resources

Because common imaging techniques like CT and MRI are anatomic tests, results are limited by resolution of the equipment. As anatomic tests, they do not show functional deficits in how the brain, heart, nerves, or other structures behave when injured. Although it has been hypothesized that deficits are at a cellular, subcellular, synapse, or enzymatic level, this has not been actually investigated or measured, either clinically or in an experimental model. Lee has corroborated electroporation of muscle cell walls with high-voltage electrical injury, but the physics of such injury are quite different from lightning physics, and certain important symptoms are different as well.[128,131,132] Electroporation has not been investigated in the lightning model or investigated in nerve or brain cells that are more affected clinically than is muscle tissue. Our knowledge of very short (sub-millisecond) impulses and their effects on the body is poor, and it is not a priori obvious that results for longer durations (electrical) are immediately transferable to short (lightning) durations.

CONCLUSION

Much remains to be done in defining lightning injury and helping those who have been injured. Because no health research funding agency currently recognizes lightning as a risk significant enough to require research funding, it is likely that many of these questions may never be fully examined in a research setting. In the meantime, probably the best answer is to prevent as many injuries as possible and to help those who have been injured the best we can—however empirically that may be.

The references for this chapter can be found on the accompanying DVD-ROM.

4 Thermoregulation

Larry I. Crawshaw, Helen L. Wallace, and Samhita Dasgupta

A warm body has long been recognized as one of the primary conditions for, and signs of, life. Humans have the capacity to maintain a viable body temperature under many climatic conditions, but thermal extremes and injury can rapidly lead to dangerous internal temperatures. For a physician operating in primitive circumstances, maintaining or restoring a patient's body temperature can require both ingenuity and knowledge of physiology.

Because the thermal environment can be extremely complicated and the thermoregulatory system of humans is complex and only partially understood, making decisions about body temperature maintenance in the field can be difficult. This chapter is designed to aid the decision process by providing a basic understanding of the relationships among the ambient thermal environment, thermal characteristics of the body, and thermoregulatory system. First, the heat balance equation is used to quantify the thermal relationship between the body and the environment. Second, peripheral neuronal inputs, the nature of the central regulator, and methods for assessing core temperature are elucidated. Third, various organ systems that the body uses as effectors to regulate body temperature are outlined. Finally, a number of special circumstances that affect temperature regulation are noted. When values are given for a "person," they refer to a 70-kg (154-lb) man.

PHYSICAL FACTORS GOVERNING HEAT EXCHANGE: THE HEAT BALANCE EQUATION

The physical laws governing heat transfer determine the net energy flux into or out of the body.[29,57,103,110,129] The heat balance equation is a convenient method for partitioning and quantifying the flow of energy between the environment and the body. A high rate of metabolic heat production is critical for maintaining a constant body temperature in mammals. This is represented by total heat production (H_{tot}) on the left side of the following equation. For a person whose body is at thermal equilibrium, the equation is balanced, and

$$H_{tot} = \pm H_d \pm H_c \pm H_r \pm H_e,$$

where

H_{tot} = total metabolic heat production
H_d = conductive heat exchange
H_c = convective heat exchange
H_r = radiative heat exchange
H_e = evaporative heat exchange.

H_{tot} is always positive. The various modes of heat exchange from the right side of the equation can be positive or negative, depending on the situation. Positive values refer to net heat loss from the body. If the equation does not balance, the body either loses or gains heat. When the sum of the net heat exchange through the various channels exceeds H_{tot}, heat content of the body will decrease and mean body temperature will fall. On the other hand, if H_{tot} is greater than the net heat exchange, heat content of the body will increase and mean body temperature will rise.

CONDUCTIVE HEAT EXCHANGE

Heat transfer between objects in direct contact is termed *conduction* (H_d). The direction of heat flow is always from the higher to the lower temperature. Because conduction involves a direct interaction (contact) between molecules, this type of heat transfer is minimal except under certain circumstances, such as when sitting on a cold rock with little insulation. Under such conditions, the heat lost to the rock would be similar to that lost from the remainder of the body surface by radiation and convection. *Adequate insulation should be placed under patients in contact with hot or cold substrates.*[102] The equation governing heat exchange by conduction is

$$H_d = \frac{kA(T_{sk} - T_a)}{L},$$

where

k = thermal conductivity
A = area of contact
T_{sk} = skin temperature
T_a = ambient temperature
L = distance between the two surfaces.

The thermal conductivity of a number of substances is given in Table 4-1. Note that water has 25 times the conductivity of air, but only one-fifth that of granite. Muscle tissue has about twice the conductivity of fat. The conduction of heat through a tissue is termed *thermal diffusivity*. This expression is obtained by dividing the thermal conductivity by the product of the density and the specific heat. The specific heats of various sub-

TABLE 4-1. Thermal Characteristics of Selected Substances

SUBSTANCE	CONDUCTIVITY (cal • s–1 • cm–1 • °C–1)	SPECIFIC HEAT (cal • g–1 • °C–1)	VOLUMETRIC HEAT CAPACITY (cal • L–1 • °C–1)
Air	0.000057	0.24	0.29
Water	0.0014	1.0	1000
Granite	0.007	0.2	540
Muscle tissue	0.0011	0.8	850
Fat tissue	0.00051	0.5	460

Data from Schmidt-Nielsen K: Animal Physiology: Adaptation and Environment, 4th ed. Cambridge, Cambridge University Press, 1990; Cossins AR, Bowler K: Temperature Biology of Animals. New York, Chapman & Hall, 1987; and Hodgman CD (ed): Handbook of Chemistry and Physics: A Ready-Reference Book of Chemical and Physical Data, 43rd ed. Cleveland, Chemical Rubber, 1962.

stances are also given in Table 4-1. Water and muscle tissue (consisting mostly of water) have particularly high values. Specific heats can be misleading, however, so the volumetric heat capacities are also listed in Table 4-1. Although the specific heat of water is four times that of air, it takes about 3500 times as much heat to raise the temperature of a given volume of water by 1°C (1.8°F) as it does to accomplish the same feat with a similar volume of air. For someone in the water, the consequence of these properties is that skin temperature is within 1°C (1.8°F) of water temperature, and heat transfer to or from the environment is greatly facilitated. In cool water during rest, skin blood flow is minimized because of peripheral vasoconstriction. Heat loss is importantly determined by the subcutaneous fat layer; an average-size fat person (36% body fat by weight) begins shivering at a water temperature of about 27°C (81°F), whereas a lean person (with below 10% body fat) starts shivering at about 33°C (91°F).[109]

CONVECTIVE HEAT EXCHANGE

Convection (H_c) can be seen as a facilitation of conduction caused by the movement of molecules in a gas or liquid. This movement decreases the functional value of L, the denominator in the conduction equation. Convection can be either forced or natural (free). Forced convection results from gas or liquid movement due to the application of an external force, such as movement of a fan or pumping of a heart. Natural convection results from density changes that are produced by heating or cooling molecules adjacent to the body. These density changes cause the molecules to move with respect to the body surface. For humans, natural convection predominates at air speeds of less than 0.2 m sec^{-1}, whereas forced convection is more important at greater air speeds.[103]

The relationships defining heat exchange as a result of convection can be complicated. They depend on surface temperature profiles, surface shape, flow dynamics, density, conductivity, and specific heat. Any factor that impedes movement of the boundary layer (the molecules immediately adjacent to the body) greatly retards convective heat transfer.

Brengelmann and Brown[11] have noted that under relatively neutral conditions (T_a = 29°C [84.2°F], wind velocity = 0.9 m sec^{-1}), about 40% of heat loss from a nude human is mediated by convection. Increases in air or fluid velocity greatly increase convective heat transfer. *Fanning a minimally clothed patient will greatly augment heat loss in a cool environment.*

RADIATIVE HEAT EXCHANGE

All objects at temperatures above absolute zero emit electromagnetic radiation. This energy transfer occurs through space and does not require an intervening medium. In any given situation, the object is both transmitting and receiving infrared thermal radiation. In some cases, the object also receives solar radiation. The net heat transfer depends on the absolute temperatures, the nature of the surfaces involved, and solar input. Surfaces that are effective absorbers of radiation are also effective emitters of radiation. The idealized "black body" illustrates this property: such bodies absorb all and reflect none of the incident radiation. Conversely, poor absorbers (such as a polished silver surface) are also poor emitters. Heat transfer due to infrared (first-term) and solar (second-term) radiation is given by:

$$H_r = \sigma \varepsilon_{sk} \varepsilon_a (T_{sk}^4 - T_a^4) + a(1 + r)s,$$

where

H_r = radiative heat exchange
σ = Stefan-Boltzmann proportionality constant
ε_{sk} = emissivity of the skin
ε_a = emissivity of the environment
T_{sk} = skin temperature (K)
T_a = ambient temperature (K)
a = absorptance
r = reflectance
s = solar radiation.

For temperatures in the physiologic range, and where ($T_{sk} - T_a$) is less than 20°C (68.0°F), several authors have noted that infrared radiation heat exchange is roughly proportional to $T_{sk} - T_a$.[9,129] Also of note is that the spectrum of emitted radiation depends on the temperature of the object. At physiologic temperatures, the predominant wavelengths of emitted radiation are longer (infrared), whereas at higher temperatures, like that of the sun's surface, the predominant wavelengths are shorter (visible radiation) and can be detected by the human

eye. This difference leads to some important consequences. The middle infrared radiation that is emitted by mammals is maximal regardless of skin pigmentation or the color of clothing. Solar radiation, however, peaks in the visible portion of the spectrum and is differentially absorbed. *Dark clothes absorb more heat from solar radiation than do light clothes, but they emit similar amounts of radiation energy.*

Incident radiation can vary drastically under different environmental conditions and may severely tax the body's ability to respond. Heat input from solar radiation on a cloudless day may exceed by several times the heat produced by basal metabolism; on a cloudless night, there is a significant net loss of radiation to the sky. Under the relatively thermoneutral conditions noted earlier by Brengelmann and Brown,[11] radiant heat loss accounts for about 45% of the total.

EVAPORATIVE HEAT EXCHANGE

When water changes state, a large amount of energy is either absorbed or given off. Evaporation of 1 g (0.035oz) of water at 35°C (95.0°F), the usual skin temperature of a person sweating,[129] requires the input of 0.58 kcal of thermal energy. *In the field, the preferred cooling measure is to splash water on the patient, coupled with air fanning.*[53] *Heat absorbed by the evaporation of 100 cc of water will lower the body temperature by about 1°C (1.8°F).* In a neutral thermal environment, sweating does not occur and evaporation accounts for only 15% of the total heat loss. Of this, slightly more than half is the result of evaporation from the respiratory tract, with the remainder coming from water that passively diffuses through the skin and evaporates.[11]

Although it is unusual, the evaporation term (H_e) of the heat balance equation can become negative (meaning that heat is being introduced into the body). This occurs during airway rewarming when water-saturated oxygen is introduced into the respiratory system at about 43°C (109.4°F). Because the victim's body is considerably colder than 43°C, water condenses in the airways. For every gram of liquid water that is formed, the body heat content increases by 0.58 kcal.

THERMOREGULATORY NETWORK

A regulatory system requires sensing the controlled variable, comparing it with an ideal value, and producing an appropriate output signal. In the following paragraphs, the role of the nervous system in the maintenance of a stable body temperature is outlined.

Peripheral Thermal Sensors

The entire outer surface of the body is well supplied with sensitive thermoreceptive structures. Because one destination of information from these receptors is the sensory cortex, many properties of the receptors can be gleaned from direct experience. Afferent thermal information produces both hot and cold sensations and is particularly rate sensitive. In addition to cortical input that arrives via the medial lemniscus and ventrobasal thalamus, the brain receives a large amount of thermal information from pathways that synapse in the reticular area.[15] Although cortical thermal input is part of the sensory information that is used to reconstruct the external thermal environment, reticular inputs are more important in behavioral and autonomic regulation of body temperature.[33] This distinction was pointed out by Cabanac,[19] who found that internal body temperature determined whether a particular surface temperature was perceived as pleasant or unpleasant. However, altered body temperature did not affect the discriminative (cortically mediated) aspects of the thermal stimulus; subjects had no problem correctly identifying the actual peripheral temperature. This study also confirms the intimate relationship between the thermoregulatory network and the pleasure–pain system.[113]

The structure, location, and properties of peripheral thermoreceptors are well documented. Thermal sensors are free nerve endings and are categorized as either warm or cold. Cold receptors are found immediately beneath the epidermis, whereas warm receptors are located slightly deeper in the dermis. The hallmark of both types of receptors is their extremely high rate sensitivity (Fig. 4-1).[60] Although the static firing rate of cold receptors is usually less than 10 impulses per second, under conditions of rapid temperature change firing rates are often higher by an order of magnitude. Cold receptors are excited by cooling, inhibited by warming, and have static maxima at about 25°C (77.0°F). These receptors are active from about 10°C (50.0°F) to 40°C (104.0°F). Warm receptors are excited by warming, inhibited by cooling, and have static maxima above 40°C. They are active from about 30°C (86.0°F) to 45°C (113.0°F). At both ends of the spectrum, more extreme temperatures activate neuronal responses that are phenomenologically reported as "cold pain" and "warm pain."[59]

Recent work utilizing cloning and ion channel characterization has elucidated peripheral temperature transduction mechanisms. A family of related temperature-activated transient receptor potential (TRP) ion channels is highly sensitive to temperature. The cloned receptors TRPv3 and TRPv4 respond over a range similar to that of the warm receptors just described, whereas TRPm8 responds similarly to the cool receptors. TRPm8 also responds to menthol. Other receptors respond similarly to the heat-pain- and cold-pain-sensitive neurons. The heat-pain channels (VR1) also respond to low pH and capsaicin (the active ingredient of chili peppers).[115]

Psychophysical and physiologic studies indicate that thermal receptors are not uniformly distributed across the body surface and that there are far more cold receptors.[59] Because peripheral thermal input is intimately involved in regulation of body temperature, heating and cooling different body sites can differentially affect the magnitude of the restorative physiologic response produced. In one study, for example, cooling the forehead was more than three times as effective (per unit area) in decreasing ongoing sweating as was cooling the lower leg.[32] A separate study evaluated regional trunk and appendage sensitivity to cooling by assessing the magnitude of the gasping response that occurs at the onset of immersion. In this case, exposing various parts of the body to water at 15°C (59.0°F) indicated that the upper torso had the greatest cold receptor density or sensitivity, or both. The lower torso was somewhat less sensitive, with the arms and legs exhibiting similar but considerably lower sensitivity.[17]

Central Thermal Sensors

Many sites within the body are capable of eliciting generalized thermoregulatory responses. Such areas include the abdominal viscera, spinal cord, hypothalamus, and lower portions of the brainstem.[7,57] The genesis of input to the regulator resulting

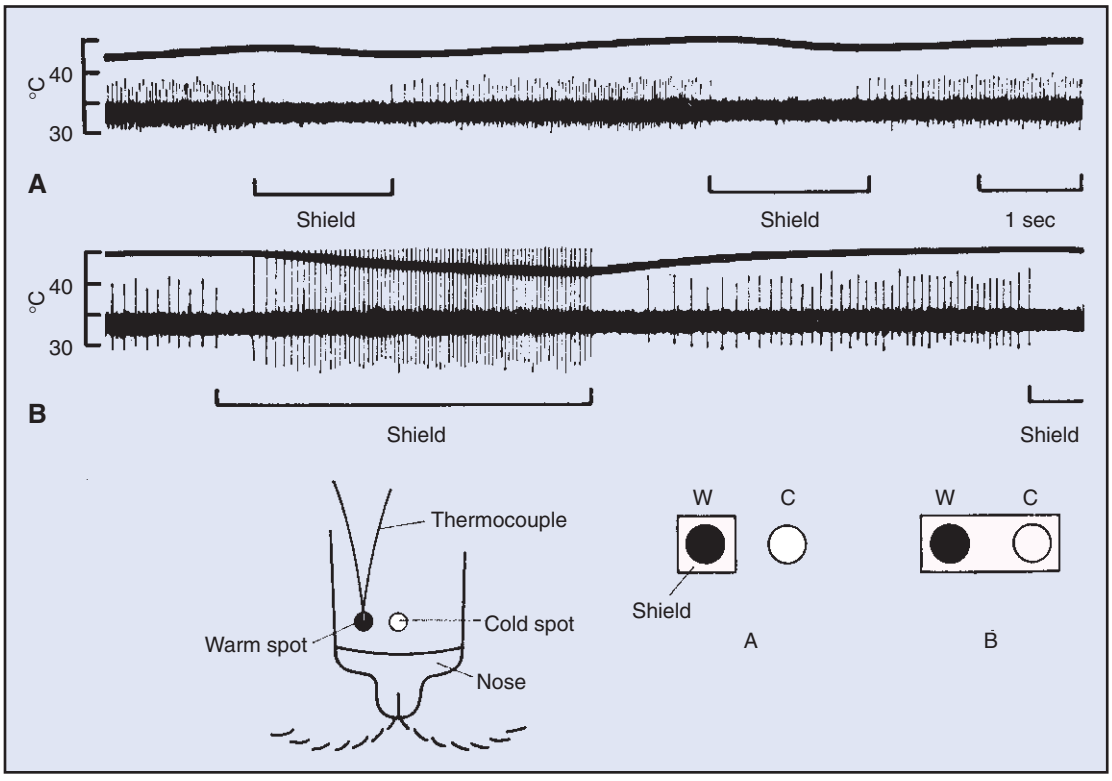


Figure 4-1. Impulses from a recording that includes a single warm fiber and a single cold fiber. **A,** In this recording, a shield was periodically placed in front of and then moved away from the skin site that was innervated by the warm fiber. The discharge stops immediately when the skin is shielded from the radiation source. **B,** In this recording, the shield was simultaneously placed in front of the skin site innervated by both the warm fiber and the cold fiber. This caused excitation of the cold fiber and inhibition of the warm fiber. (From Hensel H, Kenshalo DR: J Physiol [Lond] 204:99, 1969, with permission.)

from heating or cooling these areas is poorly understood. Some of the effects may result from modulation of synaptic connections rather than stimulation of specific thermodetectors per se. Input from central detectors is not rate sensitive but is rather a direct reflection of the absolute temperature. The area with the highest thermal sensitivity, and that has received the greatest amount of experimental attention, is the preoptic nucleus–anterior hypothalamic area (PO/AH). Heating or cooling this portion of the brainstem elicits the entire array of autonomic and behavioral heat loss and heat gain responses, respectively.[55] Neurons in this portion of the brain exhibit both warm sensitivity and cold sensitivity.[7] Recent work on hypothalamic slice preparations using synaptic blockers has indicated that warm sensitivity may be an inherent property of some of the PO/AH neurons, whereas cold sensitivity in this area of the brain requires synaptic input.[7,35] Figure 4-2 illustrates the effect of temperature on firing rates of three representative types of hypothalamic neurons. The high level of temperature sensitivity shown by one of the cells (labeled C) results from the temperature-dependent characteristic of the prepotential. Voltage clamp experiments indicate that the altered rate of depolarization is most likely the result of an effect on hyperpolarizing (K^+) conductances.[51] Work using hypothalamic slices has also established that about half of the thermosensitive neurons also respond to nonthermal stimuli such as osmotic pressure, glucose concentration, and steroid hormone concen-

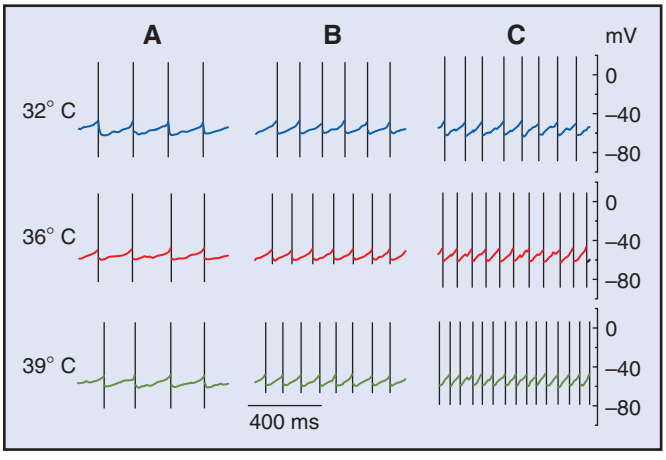


Figure 4-2. Effect of temperature on the activity of three different types of hypothalamic neurons. **A,** Low-slope temperature-insensitive neuron. **B,** Moderate-slope temperature-insensitive neuron. **C,** Warm-sensitive neuron. The thermosensitivity in each case was 0.06 **(A)**, 0.5 **(B)**, and 1.1 impulses $s^{-1} \cdot °C^{-1}$ **(C)**. All three types of neurons displayed depolarizing prepotentials, and action potentials occurred when the prepotentials reached threshold. As exemplified in **C**, putative postsynaptic potentials (especially inhibitory ones) were often observed in all neuronal types. (From Griffin JD, Kaple ML, Chow AR, Boulant JA: J Physiol 492:231-242, 1996, with permission.)

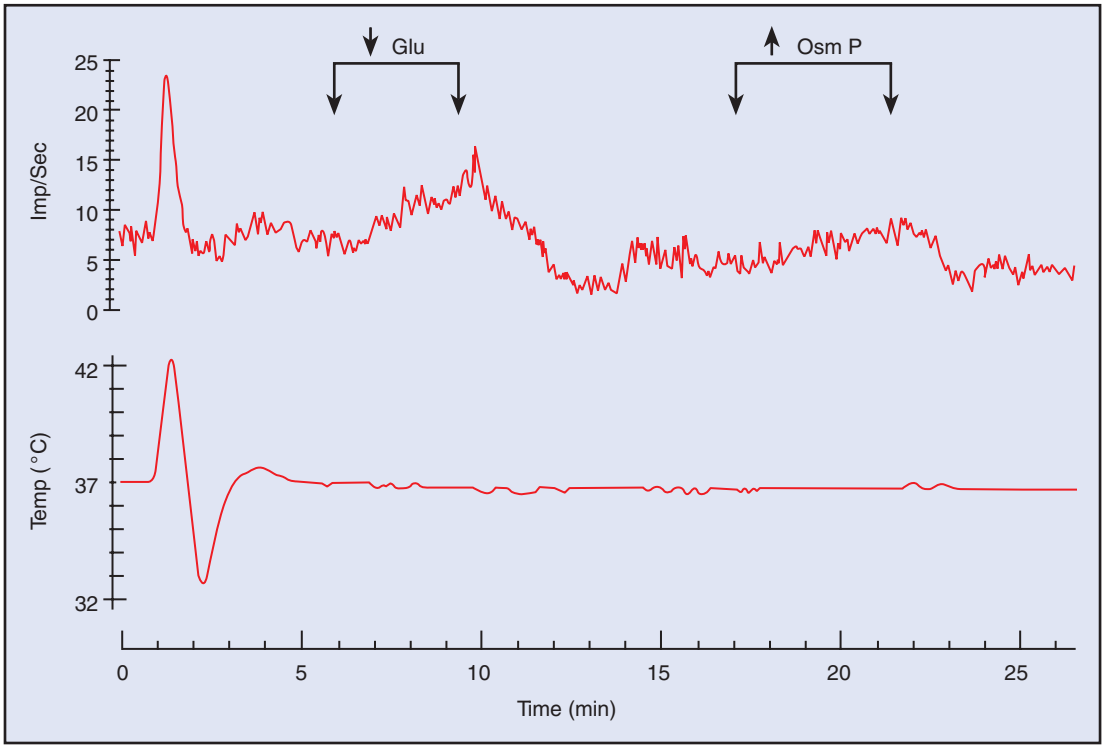


Figure 4-3. The response of a warm-sensitive preoptic nucleus–anterior hypothalamic area neuron to changes in temperature, glucose concentration, and osmotic pressure. The *large downward arrows* indicate media changes. (From Boulant JA, Silva NL: Brain Res Bull 20:871, 1988, with permission.)

tration. Such neurons could form the basis for the interactions between homeostatic systems described later. Figure 4-3 illustrates the response of a warm-sensitive PO/AH neuron in a slice preparation. This cell is excited by increased temperature, low glucose, or increased osmotic pressure.[8] Because TRPv protein expression has been detected in the PO/AH area, it has been proposed that the TRPv channels previously mentioned may also underlie the thermosensitivity found in PO/AH neurons.[115] However, TRPv channels respond to warming with persistent inward cationic currents, and this would produce a change in the resting membrane potential. In the PO/AH, both warm-sensitive neurons and temperature-insensitive neurons show identical membrane potential responses to temperature change.[156] Therefore, TRPv channels do not provide an explanation for PO/AH neuronal thermosensitivity. It is more likely that PO/AH warm sensitivity is caused by brief ionic currents that determine the rate of change in depolarizing prepotentials that occur between two successive action potentials.[51]

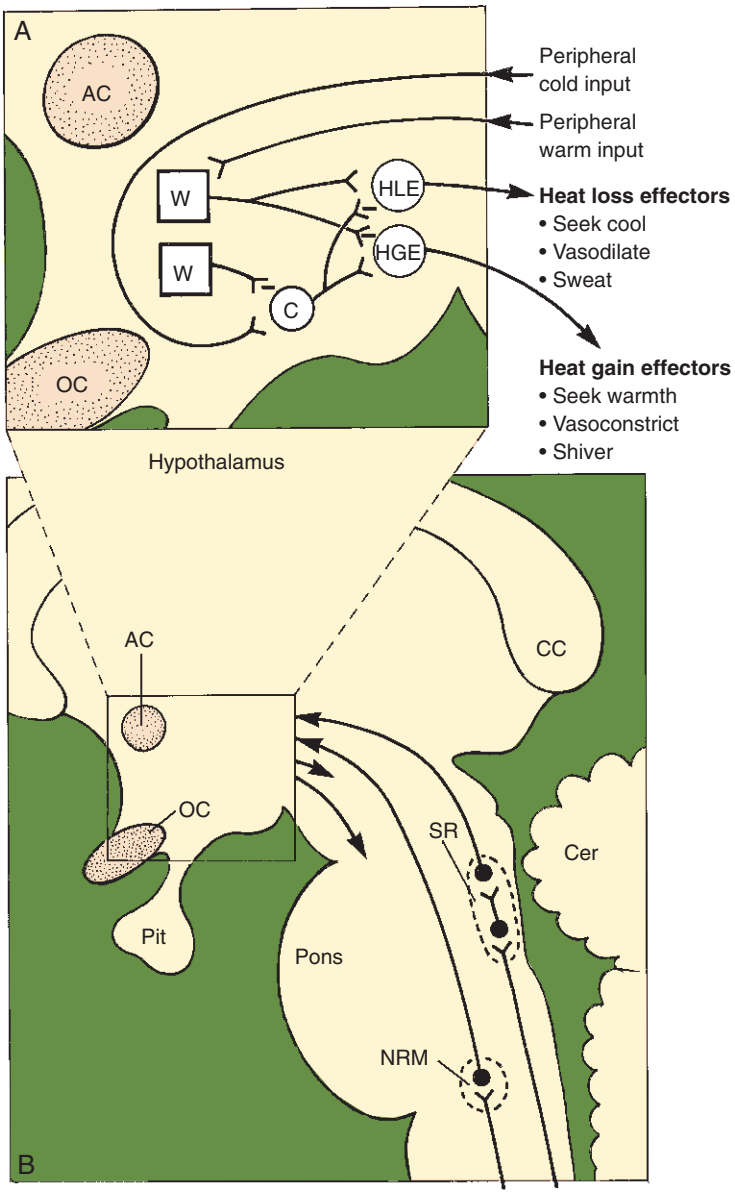
Regulator

The neuroanatomic structures that establish the regulated body temperature include portions of the spinal cord, lower brainstem, hypothalamus, and septum. The PO/AH and the caudal hypothalamus, in particular, are recognized as important integrating areas.[56] The PO/AH has been assigned a unique role as the center of central nervous thermosensitivity and thermointegration,[55] but other sites are also involved. As noted by Blatteis,[5]

Autonomic thermoregulatory functions may be attributed to brain areas outside of the PO/AH. These mechanisms appear to be subsidiary controls, may have their own (direct?) links to thermoeffectors, and are capable of operating independently of the PO/AH, although they normally may be influenced by it through ascending and/or descending connections. Thus, the role of the PO/AH in temperature regulation may be seen as not essential, but nevertheless preeminent, in the control of appropriate, coordinated, and low-threshold thermoregulatory responses. In this role, it receives afferent information from thermosensitive regions throughout the body and is connected with all the thermoeffectors.

A model for the regulation of body temperature is depicted in Figure 4-4. Such models have the advantage of clearly illustrating how the regulatory system could work, but as has been emphasized,[155] they greatly oversimplify the complexity and dynamic balance of the elements of the system. Some of the many inputs to this system include those illustrated in Figure 4-3 (glucose concentration and osmotic pressure), as well as factors covered later in this chapter such as time of day, hormone levels, pyrogen titer, and oxygen concentration in the blood.

The network in Figure 4-4 is based on the work of Hammel,[55] but it also incorporates other sources.[7,15] This system depends on input from peripheral warm and cold receptors and central thermodetectors. Input arising from cold-sensitive peripheral



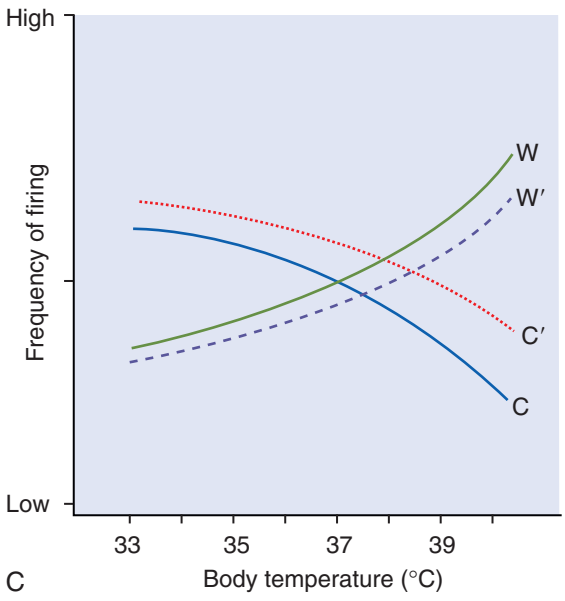


Figure 4-4. A schematic diagram denoting a suggested neuronal network to account for thermoregulation. **A,** The peripheral inputs and the site of the preoptic nucleus–anterior hypothalamic area (PO/AH)—the rectangle. **B,** Neuronal connections in the PO/AH. **C,** The firing rates of the warm (w) and cold (c) sensitive PO/AH neurons at different body (brain) temperatures. Synapses not shown as inhibitory are assumed to be excitatory. Details about the functioning of this system are in the text. ac, Anterior commissure; cc, corpus callosum; cer, cerebellum; cr, relays input from peripheral cold receptor; nrm, nucleus raphe magnus; oc, optic chiasm; pit, pituitary; sr, subceruleus region; wr, relays input from peripheral warm receptor. (Concepts and information for this figure from Boulant JA, Curras MC, Dean JB: In Wang LCH [ed]: Advances in Comparative and Environmental Physiology, vol 4. Berlin, Springer-Verlag, 1989; Brück K, Hinckel P: In Schönbaum E, Lomax P [eds]: Thermoregulation: Physiology and Biochemistry. New York, Pergamon Press, 1990; and Hammel HT: Annu Rev Physiol 30:641, 1968, with permission.)

neurons impinges on cells that excite noradrenergic neurons in the subceruleus region projecting to cold-sensitive neurons in the PO/AH. Peripheral warm-sensitive neurons provide input to serotonergic cells in the nucleus raphe magnus, which project to warm-sensitive cells in the PO/AH. The warm (W in Figure 4-4) and cold (C in Figure 4-4) sensitive neurons of the PO/AH provide reciprocal innervation that forms the basis for the thermostat. In this scheme, central cold sensitivity is postulated to be derived from inhibitory input via inherently warm-sensitive PO/AH cells. The interconnections of these neurons are shown in Figure 4-4B.

The firing characteristics of the warm and cold cells are illustrated in Figure 4-4C. At 37°C (98.6°F), the firing rates of warm and cold cells are balanced and there is no net output to either the heat loss or the heat gain effectors. If internal temperature increases, the warm cells fire faster, stimulating vasodilation, sweating, and seeking of a cool environment. On the other hand, if the internal temperature falls below 37°C, cold cells predominate and vasoconstriction, shivering, and seeking of a warm environment are stimulated.

Without the aforementioned peripheral input, body temperature would have to change considerably to create the error signal necessary to compensate for varying ambient thermal conditions. This does not occur: core temperature remains remarkably constant under most climatic conditions. This is accomplished by incorporating information from the peripheral thermodetectors. As illustrated in Figure 4-4C, if an animal moves into a cold environment, the peripheral cold receptors

are stimulated and the peripheral warm receptors are inhibited. This produces the altered firing rates shown by the W′ and C′ curves. Now, even though the body temperature is still 37°C (98.6°F), the hypothalamic cold cells are firing faster than the hypothalamic warm cells. This leads to activation of the heat gain effectors. If the system is properly designed (it is), the augmentation of vasoconstriction and metabolism is just sufficient to match the increased heat loss caused by the new, cooler thermal environment. The reverse occurs when a warmer environment is encountered. Peripheral warm receptors are stimulated and peripheral cold receptors are inhibited; the greater firing rate of the hypothalamic warm cells initiates sweating and vasodilation, even though the body temperature remains at 37°C. In this way, the regulator is able to maintain a remarkably constant internal temperature despite wide variations in the ambient temperature.

ASSESSING THE SYSTEM

The overall status of the thermoregulatory system is determined by measuring the core temperature. This can be done at a number of sites with several types of instruments (thermometers). In the following paragraphs, the relative merits of locations and instruments are discussed. In addition, an overview of the causes of altered body temperatures and the consequences of abnormally high and low tissue temperatures is given.

Monitoring Core Temperature

A recent history of clinical thermometry is available,[91] as are good overviews of the assessment of core temperature.[6,24,154] Sites, in order of increasing invasiveness, include the forehead, axilla, oral cavity, tympanum, and rectum. There is no clear-cut choice of the best site to monitor; particular situations demand different techniques. Thermometers that have been employed clinically include mercury-in-glass thermometers, electronic thermometers, tympanic radiation thermometers, and liquid crystal. Whatever instrument is utilized should have an accuracy of ±0.1°C (0.2°F). *The hand-held electronic thermometer is a good choice for field emergencies.*

Measuring Instruments

Clinical mercury-in-glass thermometers are widely available and easily used, but they have a number of disadvantages. In some situations, breakage can pose a problem, and equilibration time is at least 3 minutes. These thermometers read the maximum temperature. It is therefore important that they be shaken down fully or a core temperature below the initial reading is missed. In addition, the lowest reading on the typical clinical mercury-in-glass thermometer (34.5°C [94.1°F]) is insufficient when hypothermia is a possibility.[6,90] Although typically accurate when new, mercury thermometers may lose precision over time. In one evaluation, after 10 months, 24% of the thermometers tested failed to meet the accuracy standard just referred to.[24]

The hand-held electronic thermometer has replaced the mercury-in-glass thermometer in many applications. These instruments can utilize either thermistors or thermocouples as sensors, have the requisite degree of accuracy, and are very flexible in application. Although an equilibration time of 1 minute is specified for the typical probe, this is largely because of the need for a stiff casing for ease of insertion. Smaller probes are available that can equilibrate in seconds. The digital display of these instruments reduces errors, and the probes can be left in place for continuous monitoring. A quality instrument with a wide range of interchangeable probes is important. Even then, these devices are subject to the usual problems inherent with electronic instruments. *A mercury thermometer for backup and calibration, an alternate probe, and a spare battery are essential.*

Tympanic infrared radiometers are often used in hospital settings, but even in this relatively predictable environment some controversy exists over their ability to accurately assess core temperature. These instruments monitor electromagnetic radiation emanating from the ear canal, and various manufacturers utilize different, complicated electronic circuitry to produce a temperature display. An advantage is that the reading takes only a few seconds.[24] Questions remain, however, about the overall accuracy of the measurement displayed. In a laboratory situation where the auditory canal is plugged with a sponge and the probe measures only radiation emanating from the tympanum, infrared tympanic thermometry provides an excellent estimate of the core temperature.[136] In clinical settings, the results are less consistent. In one study, infrared tympanic thermometry produced core temperatures that were much more variable than rectal temperatures. Even after correcting for the higher rectal values (0.5°C [0.9°F]), tympanic measurements still inaccurately displayed one third of the temperatures above 37.7°C (99°F). An extended training program did not significantly alter accuracy of the readings.[117]

In one instance, a child arriving at an emergency department presented with tachycardia and skin vasoconstriction. Separate tympanic infrared thermometers gave core temperatures of 36.4° and 37.6°C (97.5° and 99.7°F). The rectal temperature was determined to be 42.2°C (108°F).[123] On the other hand, in a hospital setting with a trained operator and immobile patients, two brands of infrared tympanic thermometers produced readings that were closer to pulmonary artery readings than were those of the axilla or rectum.[122]

The potential benefits of a continuous, easily applied core temperature monitor have led to repeated attempts to validate liquid-crystal thermometers, typically placed on the head or neck surface. Unfortunately, the temperature readings produced by this method are not reliable.[6,91] Since these measurements are compromised by thermoregulatory vascular changes associated with heat conservation and heat dissipation, as well as by changes in ambient temperature,[66] they are particularly unsuited for field emergency measurements.

Measurement Sites

Although the deep internal temperatures of normothermic humans are reasonably similar, no specific anatomic site represents the core temperature. The temperature at each location is a consequence of a combination of local metabolic rate, local perfusion rate, proximity to the outer shell, and proximity to other locations with differing rates of metabolism and perfusion. Nevertheless, because of the generally high overall rates of tissue perfusion in mammals, deep core temperatures rarely differ by more than 0.5°C (0.9°F). The temperature of the pulmonary artery is a good reference temperature for the overall status of the thermal core. At steady state, accepted sites for assessing core temperature differ in varying amounts from this temperature. Esophageal and tympanic temperatures are essentially the same as the temperature of the pulmonary artery,[122,127] whereas rectal temperature averages about 0.4°C (0.7°F)

higher, and axillary and oral temperatures about 0.2° and 0.4°C (0.4° and 0.7°F) lower, respectively.[6,90,122]

Although the esophageal temperature is somewhat difficult to obtain, this is the site most likely to accurately reflect the temperature of the pulmonary artery. Measurement at this location accurately follows changes in core temperature and is reasonably noninvasive. For placement, the probe is lubricated and a small amount of local anesthetic is applied. It is then passed via a nasal passage into the distal portion of the esophagus at the level of the heart. The probe can be moved up and down slightly to obtain the highest temperature. Although this procedure is routinely utilized in physiology experiments, it is somewhat unpleasant for conscious patients. Esophageal temperature is transiently (for 30 sec) affected by swallowing.

Tympanic temperature as an estimation of core temperature has long been controversial. Because the tympanic membrane is highly vascular and is supplied by branches of the external and internal carotid arteries, it should be an ideal site. Nevertheless, over the years many studies have indicated that tympanic temperature is affected by ambient temperature and local facial cooling.[127] Conditions under which these complications can be avoided have now been clarified. If (1) the ear canal is insulated, (2) the thermocouple is made of fine wire and insulated except at the active junction, and (3) the thermocouple is in direct contact with the tympanum (which causes the patient to "hear" a continuous, low-pitched sound), then alterations in core temperature are detected more rapidly than by an esophageal probe, are not affected by facial skin temperature, and are otherwise identical to esophageal temperature.[127] In fact, if these conditions are adhered to, during facial fanning tympanic temperature provides a better estimate of brain temperature than do measurements made in the esophagus or rectum.[95] Accurate results were also obtained when an insulated probe was used in conjunction with an optical sensor to detect infrared radiation from the tympanum.[136] Whether the conditions met in these carefully controlled studies can be duplicated in the field is problematical.

In steady-state conditions, rectal temperature is a good index of core temperature and can also be utilized to estimate brain temperature.[157] However, when the heat content of the body or of the internal thermal compartments is in flux, rectal temperature changes more slowly than temperatures measured in other commonly used sites.[122] There is a thermal gradient along the rectum, so all measurements should be made at a standard depth; 4 cm is recommended.[6] The higher temperatures recorded in this region may be caused by a combination of low perfusion rates, digestive reactions, and bacterial activity, but there is not clear evidence in this regard.[92]

Oral temperature is an excellent index of core temperature, provided that the mouth is kept closed. The sublingual pocket is well perfused, and it responds quite rapidly to alterations in core temperature. Mastication, smoking, fluid intake, and mouth breathing can affect the sublingual temperature. These should be avoided in the period immediately preceding the measurement.[6,90,92] The use of an electronic thermometer with a rapidly responding sensor makes this measurement considerably more accurate and rapid than when performed with a mercury-in-glass thermometer.

Although axillary temperature does reflect core temperature, it has a number of negative characteristics and should be utilized only as a last resort. The axillary temperature is affected by local blood flow as well as thermal and nonthermal sweating.[6] Changes in core temperature are slow to affect the axillary temperature, and there is high interpatient variability.[122] This measurement, however, has proven particularly useful for assessing core temperature in infants.[5,92]

Heterothermy

Core temperature, as assessed in the previous paragraphs, provides a good estimate of the temperature of critical internal organs. This temperature is vigorously defended by the body. At low temperatures, regional heterothermy resulting from peripheral vasoconstriction forms an important aspect of this defense, increasing insulation by decreasing skin temperature. This decreases the thermal gradient for heat loss from the skin to the environment. Thus, at cooler temperatures there can be a large amount of tissue that is well below core temperature, and as a consequence the overall heat content of the body is greatly decreased. A nude human resting at 35°C (95°F) or 20°C (68°F) exhibits similar temperatures at various locations within the core. However, because of decreased temperatures in the outer shell, a person resting at 20°C will have a total heat content that is about 200 kcal lower than the same person resting at 35°C.[129] If the peripheral vessels were suddenly dilated, an immediate drop in core temperature of about 3.5°C (6.3°F) would result. In a hypothermic individual, the discrepancy between core and shell temperatures could be considerably greater. The temperature of the outer shell can be estimated by measuring skin temperature at a number of sites, with the sensor in contact with the skin on one side and insulated on the other.[6] An equation has been developed to estimate the mean temperature of the body using measurements of ambient temperature, several skin temperatures, and core temperature.[47] Although this approach is not feasible in the field, several measurements along the distal extremities can be used to obtain an estimate of outer shell cooling.

A different type of heterothermy may be present in hyperthermic humans. As the brain temperature reaches high levels, blood flow that is normally outward from the intracranium to the face via the ophthalmic vein is redirected and flows from the face inward.[63] This results in the brain being cooler than the remainder of the core. Although brain cooling is clearly documented and accepted in many mammals,[4] it remains controversial in humans.[10,18] Nevertheless, there is unanimous agreement that the head is an extremely important area for heat loss.[10,18] Thus, for hyperthermic patients, it is important to optimize the heat loss from that region, and, when necessary, to augment cranial heat dissipation by fanning and moistening.

Reasons for Altered Core Temperature

A variety of conditions can lead to an abnormal core temperature. Under some circumstances, the regulator may be defending an altered core temperature for reasons to be described. Alternatively, the thermal load posed by the environment or by heavy exercise may be too great for the capacity of the effectors. Finally, the regulator could be deranged due to substance abuse, extreme temperatures, side effects of prescription drugs, or other factors. When interpreting a particular core temperature, it is important to evaluate all of these alternatives. Accidents in the wilderness often involve a compromise of many aspects of thermal balance, and an altered body temperature is very likely.

Consequences of Altered Body Temperatures

When tissue temperatures change, there are immediate and important effects on metabolism, as well as on other physiologic mechanisms. With a 10°C (18°F) increase in temperature, the metabolism of typical human tissue increases by a factor (the Q_{10}) of about 2.7. The metabolic rate of the entire organism (apart from thermoregulatory responses) responds similarly. For temperature differences other than 10°C (18°F), these effects can be calculated with the equation[129]

$$R_2 = R_1 \cdot Q_{10}^{([T_2-T_1]/10)},$$

where

R_2 and R_1 = the two rates of physiologic response
T_2 and T_1 = the two temperatures
Q_{10} = increase in rate caused by a 10°C (18°F) increase in temperature.

Within the normal range of body temperatures, higher temperatures favor speed at the expense of tissue resources, whereas lower temperatures conserve resources. Although both high and low temperature extremes pose a threat to humans, increased temperatures greatly accelerate the development of serious complications and pose a much more immediate danger. A deviation of about 2°C (3.6°F) above or below the normal core temperature is well tolerated by the various regulatory systems of the body, but a discrepancy of about 3°C (5.4°F) begins to disrupt these systems, including those involved in temperature regulation. At this level of deviation, if there is no intervention, physiologic problems compound very rapidly.

Mild hypothermia (a core temperature of 34° to 36°C [93.2° to 96.9°F]) disrupts many important physiologic functions, which, taken together, may significantly affect patient outcome. Mild hypothermia impairs recovery from surgical procedures due to, among other things, impaired peripheral blood flow and oxygen availability, increased possibility of cardiovascular complications, decreased antibody and cellular immune defenses, impaired coagulation, and increased metabolic expenditure for heat production.[37,46,84,87] *In most situations, it is very important to maintain the patient at normothermic levels.*

Traumatic brain injury can be present in wilderness accidents, and it may be accompanied by hyperthermia. *Heightened temperatures can exacerbate cerebral inflammation and lead to increased neuronal damage.*[145] There is current interest in invoking mild hypothermia to minimize damage to the central nervous system following neurologic injury.[110] However, when this approach is used, care must be taken to deal with the side effects mentioned in the previous paragraph.[118]

EFFECTOR RESPONSES

The following paragraphs examine the properties of the response systems that heat or cool the body according to the output demands of the regulator. These effectors are influenced both by the output of the thermoregulatory network and by the temperature of the effector organ itself. Because patients away from medical facilities often exhibit whole-body or regional hyperthermia or hypothermia, both central outputs and local effects are relevant, so they are discussed separately.

Vascular Adjustments

Excellent overviews of the role of the vasculature in coping with thermal stresses are available.[11,25] One function of the circulatory system is to maintain a relatively homogeneous internal body temperature. Heat from metabolically active organs is convectively distributed to portions of the body where less heat is produced. More commonly appreciated are alterations of blood flow patterns that increase or decrease overall thermal conductivity of the body during exposure to hot or cold environments, respectively. Some of these alterations in conductivity result from preferentially shunting peripheral blood flow superficial or deep to the subcutaneous fat layer. Indeed, fat has about half the tissue conductivity of muscle. Nevertheless, shunting of blood away from major portions of the body is at least as important in determining overall conductivity as is the conductive property of the tissue itself. For example, during immersion in cold water, muscle accounts for about 90% of the total tissue insulation of the forearm.[38] Thus, directing blood away from poorly insulated (more highly conductive) regions reduces heat loss and preserves core temperature.

In addition to capillaries, microcirculatory units contain arterioles, metarterioles, and arteriovenous anastomoses. Flow through all of these vessels is under the control of smooth muscle. The smooth muscle of precapillary sphincters is largely influenced by local factors such as P_{O_2} and P_{CO_2}, whereas the other vessels are well supplied with receptors that respond to both neuronal and endocrine inputs. Glabrous skin (palms, lips, and soles) contains numerous arteriovenous anastomoses and is innervated only by sympathetic vasoconstrictor fibers. Nonglabrous (hairy) skin is innervated by sympathetic vasoconstrictor and vasodilator fibers and contains few arteriovenous anastomoses. The vasoconstrictor system acts primarily through α-1 and α-2 adrenoceptors, whereas the active vasodilator system utilizes acetylcholine, an unidentified cotransmitter, and nitric oxide. About 90% of the elevation in skin blood flow during heat stress is due to input from the active vasodilator system, and of this about 30% is due to nitric oxide.[68] *Thermoregulatory skin blood flow can reach up to 8 L/min and involve 60% of the cardiac output.*[25]

Operation of the vasomotor effector system is affected by excessive exposure to ultraviolet B radiation. Moderate sunburn impairs the vasoconstrictor response to cold; an associated, uncontrolled increase in thermal conduction is still present 1 week after exposure, although the original erythema has disappeared.[112]

Central Signal

Vascular changes are bioenergetically the least costly thermoregulatory autonomic effector response. Because of high sensitivity of the vasomotor system, ambient temperatures between the thresholds for sweating and shivering are often referred to as being in the zone of vasomotor regulation. If a particular vascular bed is kept at a relatively constant temperature, output from the central nervous regulator can be assessed. Under these conditions, in dogs, manipulations of hypothalamic temperature confirm a high level of vasomotor activity between the thresholds for the activation of panting and shivering.[58] In humans, forearm blood flow increases rapidly as core temperature rises; a sixfold increase in blood flow can occur with a core temperature that has risen to only 38°C.[148] Within the vasomotor zone (skin temperatures of 33° to 35°C [91.5° to 95.1°F]), core and skin temperatures linearly combine to

control skin blood flow. Skin blood flow responds accurately and rapidly to changes in skin temperature, which leads to a very stable core temperature.[12]

Although most peripheral arterioles are well supplied with adrenergic receptors, output from the thermoregulatory centers is not homogeneously distributed. Extensive nervous inputs from the thermoregulator occur only in the lips, ears, and distal extremities. Thus, immersing the feet in cold water leads to marked vasoconstriction in the hands and forearms, but not in the abdomen or upper arms.[50]

Local Modulation

Local temperature has a great effect on the vasomotor status of the peripheral vessels, and in some cases it may be largely responsible for the observed thermal conductivities. Local heating produces a biphasic response. The initial dilation occurs in a few minutes and depends on the release of various factors from the nerve ending. The VR1 receptors mentioned earlier are involved in this response. The longer-lasting second phase of dilation is not dependent on local nerve endings and involves nitric oxide. *Local cooling can decrease superficial cutaneous blood flow to almost zero.*[25] Although vascular beds on the skin surface constrict in response to cooling, other vascular beds dilate when cooled.[41] The specific response to cold shown by cutaneous vessels follows from the observed distribution and properties of the alpha-adrenergic vascular receptors. Although in most of the vasculature α_1-receptors predominate, in the superficial cutaneous areas α_2-receptors constitute a clear majority. The usual predominance of α_1-receptors is found in the deeper blood vessels. Local temperature affects the α_2- and α_1-receptors in a reciprocal manner. Although cooling augments the response of the α_2-receptors, it either inhibits or does not affect the response of the α_1-receptors. Cooling the skin, then, not only constricts the superficial vessels but also concomitantly dilates many underlying vessels. The ensuing flow pattern increases tissue insulation and augments countercurrent exchange between incoming cool blood and outgoing warm blood.[41,43] Although initial work was done on canine vessels, subsequent studies using α-adrenergic agonists and antagonists have demonstrated that a similar mechanism exists in human fingers.[39,42]

The responsiveness of cutaneous blood vessels is diminished in people with type 2 diabetes mellitus. The increased incidence of this disorder among the general population presages a greater number of patients exhibiting severe hyperthermia.[25]

Evaporative Responses

At high workloads and at environmental temperatures approaching 37°C (98.6°F), the only way to maintain thermal balance is to augment evaporative cooling by mobilizing the eccrine sweat glands. This sympathetic, cholinergically innervated organ system is spread over the entire body surface, but it is more profuse in some areas than in others. *A person acclimatized to heat can produce several liters of sweat per hour.*[64] High rates of sweating occur on the forehead, neck, anterior and posterior portions of the trunk, and dorsal surfaces of the hand and forearm. Low rates occur on the medial femoral regions, lateral trunk areas, palms, and soles.[108] Sweat is secreted in these latter two areas in response to emotional, but not thermal, inputs.[11] Sweating is cholinergically mediated and can be completely abolished by atropine. Sweat gland activity interacts with the regional vasculature; metabolic products of active sweat glands increase blood flow in areas of active sweating. *In well-hydrated individuals, the degree of anhidrosis is correlated with the severity of generalized autonomic failure.*[50,89] Several reviews discuss the many aspects of sweating disorders.[26,89]

Central Signal

By controlling the local milieu at different skin sites, it has been possible to separate the central thermoregulatory drive to sweat glands from local effects on the glands themselves. The central thermoregulatory system provides a proportional output that is influenced by both internal and whole-body skin temperatures. Per degree increase above thermoneutral values, internal temperature is about 10 times as important as mean skin temperature in eliciting an output to the sweat glands.[104,106]

Local Modulation

Local effects are important in determining the output of sweat glands. Temperature exerts a multiplicative effect on sweat secretion; the Q_{10} for this augmentation is about 3.70. Skin wetness also has an important local effect on sweat glands. The wetter the skin, the greater the suppression of sweating.[105]

Moderate sunburn disrupts evaporative cooling. This effect is locally mediated and involves decreases in both the responsiveness and capacity of the sweat glands.[111]

Metabolic Responses

Heat is an inevitable byproduct of the inefficiencies in the body's metabolic reactions. In oxidizing foodstuffs to carbon dioxide and water during ATP production, and transferring the ATP produced to the functional systems of the cells, about 75% of the original chemical potential energy appears as heat. Excepting that excreted or used to perform physical work, the remaining 25% of the original energy is also converted to heat when ATP is utilized in the numerous metabolic reactions of the body.[130] Mammals, relative to poikilotherms such as reptiles or fish, utilize much more ATP to maintain ionic and electrochemical balance of the cells[141] as well as for many other functions. This leads to a greatly increased metabolic heat production, which forms the basis for homeothermy. It also creates the need to maintain a substantial thermal gradient between the body and the environment to dissipate the high levels of heat that are produced.

An increased rate of metabolism above basal levels is critical for maintenance of body temperature in cold environments. The elevated heat production is derived largely from simultaneous rhythmic excitation of agonistic and antagonistic skeletal muscles (shivering), but other domains such as the gastrointestinal tract or adipose tissue[50,138] may be involved to some degree. Under normal circumstances, carbohydrate oxidation provides the major substrate for shivering. In glycogen depleted individuals, shivering levels are maintained by greatly increased oxidation of lipid and protein reserves.[54]

There is evidence that both epinephrine and thyroid hormones are released in humans following cold exposure.[45] Because these hormones augment overall tissue metabolism, they are components of the response to cold environments. In the absence of thyroid hormone, metabolic heat production is reduced by 30% or more. Thyroid hormone acts by both accelerating ATP turnover and reducing the efficiency of ATP synthesis.[137]

The basal metabolic rate, when calculated on a weight-specific basis, decreases with body size. This relationship holds within, as well as across, species. *Because drug potency (determined by the rate of inactivation) is most closely related to metabolic rate, when drug dosing is extrapolated to persons of different sizes it should not be done on a per-kilogram (or per-pound) basis unless the weights are similar.* Basal metabolic rate relates to size according to the equation[129]

$$\dot{V}o_2 = 0.676M_b^{0.75},$$

where

$\dot{V}o_2$ = oxygen consumption in L/hr
M_b = body mass in kg.

To calculate the correct drug dose for patients of different sizes, such as calculating the drug dose for a small child from a dose given for a large adult, the following equation should be used:

$$\frac{\dot{V}o_2 \text{ person A}}{\dot{V}o_2 \text{ person B}} = \frac{\text{Drug dose person A}}{\text{Drug dose person B}}$$

Thus,

$$\text{Drug dose person B} = \frac{(\dot{V}o_2 \text{ person B})(\text{Drug dose person A})}{\dot{V}o_2 \text{ person A}}$$

Central Signal

Of the various thermoregulatory outputs, metabolism is the easiest to evaluate quantitatively; the most complete documentation is available for this response, and most models of the thermoregulatory system are based on this information. Experiments on medium-size mammals have allowed the separate thermal manipulation of various parts of the brain, body core, and skin. This work has made it clear that the thermoregulatory centers act as a proportional controller and that skin temperature provides a feed-forward input to the system.[55,69] Thus, greater decreases in either core or skin temperature, or both, below neutral values elicit proportionally larger compensatory increases in metabolism. In addition to an impaired ability to generate metabolic heat, hypothyroidism is also associated with a decrease in regulated core temperature of about 1°C (1.8°F).[152]

Evidence indicates that humans have a similar control system. In a summary of their data and of that collected previously, Hong and Nadel[65] noted that the central output for shivering is augmented by an increased rate of skin cooling. They also concluded that a given decrease in core temperature elicits 10 to 20 times the metabolic response of an equivalent decrease in mean skin temperature. Exercise is not incompatible with shivering, but increased levels of exercise exert increasing degrees of suppression on the shivering response, possibly as a consequence of an increased arousal response.[65]

Local Modulation

Although the central and local effects of decreased core temperature on shivering have not been directly partitioned, both inputs are important. Slight decreases in core temperature create large compensatory responses, as delineated previously. However, even moderate hypothermia decreases the metabolic response to cold, and at about 30°C (86.0°F), the shivering response is lost.[11] This decrement must involve nervous system malfunction, because the muscles themselves are quite responsive below 30°C. For example, limb muscles and diaphragm muscles develop peak tensions that are not greatly affected by temperatures down to 25°C (77.1°F), and fatigue resistance is considerably increased at 25°C (77.1°F).[119,131] Likewise, altering skin and superficial muscle temperature of the anterior thigh through a range of temperatures between 12° and 40°C (between 53.6° and 104.0°F) for 30 minutes had little effect on subsequent isometric peak torque production during isometric knee extensions to exhaustion. Time to fatigue was longer at the coolest temperature.[146]

Behavioral Responses

In most wilderness situations, a variety of ambient temperatures are available and external insulation is easily adjusted. Under these conditions, the choice of thermal microenvironment and clothing provides a far higher gain than any of the autonomic effector systems discussed previously. Whole-body adjustments are achieved by all motile animals and are particularly well developed in vertebrates.[31] In addition to moving the body, the somatic effectors are important for optimizing autonomic responses to thermal stress. Thus, spreading out the arms and legs during heat stress increases the surface area available for autonomic augmentation of conductive, convective, evaporative, and radiative heat losses.

Central Signal

Available evidence indicates that behavioral responses are elicited by the thermoregulatory controller through outputs similar to those delivered to the autonomic effector organs.[31,33] Severe deviations in core temperature disrupt this system, and when this occurs, the person no longer feels too hot or too cold and the desire to take corrective action is lost.

Local Modulation

As with shivering, most problems involving behavioral temperature regulation probably emanate from disruption of the centrally generated output. The muscles used to move the body are fairly resistant to thermal incapacitation (see previous discussion), but if this occurs, major disruption of the body's thermal defenses ensues.

IMPORTANT MODIFICATIONS OF THERMOREGULATORY RESPONSES

In addition to the importance of a stable body temperature for normal physiologic function, monitoring body temperature provides a significant diagnostic indicator for many pathologic conditions. Whether the goal is to stabilize or to monitor body temperature, it is necessary to understand the many conditions that affect both the regulated temperature and effectiveness of the thermoregulatory system. In the following paragraphs, these circumstances are elucidated.

Normal Variation in the Regulated Temperature and in the Ability to Maintain Body Temperature

The same body temperature can represent a different state of affairs even under regularly encountered circumstances. Some of these conditions are noted here.

Level of Activity

Activity normally leads to increases in body temperature. However, the level of activity does not appear to provide direct input to the regulator of body temperature, unlike the situation with peripheral temperature. Rather, the magnitude of the error signal for increased heat dissipation is determined simply by the increase in body temperature.[142] Thus, someone exercising heavily (or having just exercised) in a neutral environment has an unusually high body temperature, whereas someone sleeping or resting quietly has a relatively low body temperature. At a given level of exercise, core temperature plateaus 30 to 40 minutes after the exercise is initiated; higher levels of exercise result in higher plateau levels of core temperature. For someone working at 50% of their maximal aerobic capacity, the increase in core temperature is about 1°C (1.8°F).[109]

Circadian Changes

Body temperature shows cyclic changes throughout the day. Some of this variation is due to the daily cycle of activity, as described earlier. However, there also exists a circadian rhythm for the body temperature set point. This sinusoidal rhythm accounts for much of the observed variation in body temperature. In a study involving 700 observations on 148 healthy individuals, the daily mean oral reading was 36.8°C (98.2°F). However, this was only a midpoint; the mean early-morning low was 36.4°C (97.6°F), and the mean late-afternoon high was 36.9°C (98.4°F).[93] These diurnal changes definitely reflect alterations in the controller, because the body temperature thresholds for eliciting sweating and peripheral vasodilation are significantly lower in the early morning than in the afternoon or evening, whereas the sensitivities and maximal response levels remain unchanged.[1,2] Melatonin may be an important factor in the rhythm of body temperature; light exposure produces similar shifts in both temperature and melatonin rhythms,[135] and artificial reductions in melatonin levels attenuate the circadian decline in body temperature.[21]

Thermoregulatory responses to exercise are affected by the circadian time. At the daily low (5:00 AM), the perceived exertion for a standardized task is the greatest, and thermoregulation is less effective than at the time of maximum temperature (5:00 PM). At the time when the body temperature is rising at the fastest rate (11:00 AM), heat loss mechanisms are less responsive, whereas at 11:00 PM, when the body temperature is falling at the fastest rate, heat loss mechanisms are much more responsive.[147] An excellent overview of body temperature cycles is available.[121]

Interindividual Differences

Normally, there is surprisingly little interindividual variability in core body temperature. In the study involving 700 observations described previously, 90% of the early morning values were between 36.0° and 37.1°C (between 96.9° and 98.9°F); corresponding values for the late afternoon were 36.3° and 37.4°C (97.4° and 99.4°F). On the basis of these interindividual differences, and of the diurnal changes described earlier, it has been suggested that the upper limit for a normal oral temperature should be 37.2°C (98.9°F) in early morning, increasing gradually to 37.8°C (99.9°F) by early afternoon, and remaining at that level until the early evening.[93] These values delineate the 99th percentile for body temperature observed during the respective time periods.

On the other hand, it is important to be aware that the normal body temperature of some individuals does fall outside of population norms. In one person the authors encountered, core temperature was consistently 35.5° to 36°C (95.9° to 96.8°F). He mentioned that on one occasion he had felt chills and malaise, but he was told by his physician that his temperature of 37°C (98.6°F) was normal. It was not. For this person, a core temperature of 37°C (98.6°F) represented a febrile state. Many individuals with atypical body temperatures are aware of their condition; it is prudent to ask about this possibility.

Age

The circadian rhythm of body temperature develops soon after birth. Although newborns display small-amplitude rhythms, the patterns are not circadian. Circadian rhythmicity begins to develop during the second and third weeks of life, and after a progressive increase in amplitude, the typical adult temperature rhythms are reached at 2 years of age.[121] Under thermoneutral conditions, rectal temperatures for older adults are similar to those of younger people, whereas oral and axillary temperatures are slightly lower.[73]

Of the major regulatory systems, temperature regulation is unique in the extent to which the effector organs are "borrowed" from other systems. This makes developmental assessments difficult, as functional changes may be secondary to changes in primary systems such as skeletal muscles or blood vessels. Other difficulties, detailed by Cooper,[28] include inconsistencies between chronological age and physiologic viability, and the increased incidence of interfering disease states and cerebral microinfarcts as aging progresses.

Thermoregulatory capacities of the young show a progressive increase but are not fully developed until after puberty. Effectors more important to infants than adults include certain behavioral responses (call for help) and the ability to activate thermogenic brown adipose tissue. Although brown adipose tissue is of little consequence in adults, it may aid in the production of heat in infants; adults may have the capacity to develop brown adipose tissue if subjected to chronic cold stress.[62] Shivering is not present in infants and develops fully only after several years as the nervous system matures. Metabolism in infants is increased to some degree by an increase in motor activity, which accompanies cold stress.[79]

Sweating is present and effective in children, but increases in sweat gland output during puberty lead to the typical high capacity for evaporative heat loss present in adults.[40] Factors affecting the loss of body heat during cold stress throughout the adult years have been investigated by using a multiple regression analysis to evaluate fitness, fatness, and age (from the twenties to the early fifties). Fitness has no effect, but fatness retards heat loss. Aging during this period is correlated with a progressive weakening of the vasoconstrictor response to cold.[16]

Individuals in their late sixties and beyond have a definite decrease in thermoregulatory capacity. Sweating is lessened in response to passive heating,[67] vascular responses to heating and cooling are significantly reduced,[75,78] and a distinct shivering tremor is rarely observed.[79] In older adult patients, thermoregulatory sympathetic nerve impulses to the skin are reduced by 60%,[49] and there is a lower resting metabolic rate.[149]

The young and older adults, without doubt, are particularly vulnerable to thermal extremes and should be given treatment priority when possible. Both groups are susceptible to climatic

heat injury, and when core temperatures exceed 40°C (104°F) and are accompanied by an altered mental status, treatment should be immediate, even at the risk of misdiagnosing a febrile condition. As for all such cases, most clothing should be removed and ice, if available, placed around the groin, axillae, and neck. Cool water should be sprayed on the skin and fanned. Concurrently, blood gas and electrolyte status should be determined and appropriate measures taken.[20,22]

Sex

Although less work has been done on thermoregulation in women, evidence indicates that their thermoregulatory responses are qualitatively similar to those of men.[52] Taken as a group, women have a number of physiologic and morphologic characteristics that produce subtle differences in the regulation of body temperature. Such attributes include a smaller blood volume, lower hemoglobin concentration, smaller heart, smaller lean body mass, greater percent of subcutaneous and total body fat, greater surface-area-to-mass ratio, higher set point for cutaneous vasodilation and sweating onset, greater resting vasoconstriction in hands and feet, geometrically thinner extremities, and cyclic hormonal changes.[100]

When age, thermal acclimation, body size, maximal aerobic capacity, and relative workload are matched, thermoregulatory sex differences are negligible. Nevertheless, in situations such as those encountered in the U.S. Navy, men and women do not tend to be matched for such nonthermoregulatory factors, and different thermal exposure standards may be required for the sexes.[52]

The menstrual cycle, menopause, and pregnancy are all associated with important effects on the thermoregulatory system. Relative to the early follicular phase of the menstrual cycle, core temperature is typically 0.3°C (0.5°F) higher in the late follicular phase and 0.7°C (1.3°F) higher in the luteal phase.[83] Postmenopausal hot flashes are experienced by most women and involve increases in sweating, vasodilation, and heart rate. Heat and exercise may be particularly stressful during and after these episodes. The proximate mechanism appears to involve a central noradrenergic activation.[44] Some information about hormonal effects has been gained by studying postmenopausal women undergoing hormone replacement therapy. Administered estrogen acts to lower the core temperature at which heat loss effector mechanisms are activated and results in a lowered core temperature. The addition of exogenous progestins blocks these effects.[13]

Pregnancy is of special concern to the physician in the wilderness. This concern does not apply to well-hydrated women exercising at submaximal levels, because the thermoregulatory system makes adaptive adjustments as pregnancy proceeds. Over the course of a pregnancy, basal body temperature shows a continuous decline and heat-loss responses are elicited at progressively lower levels, so that near term, steady-state temperature during exercise is about 1°C (1.8°F) lower than before conception. This reduces thermal stress on the fetus, which is typically 0.5°C (0.9°F) warmer than the mother.[27,140]

Concern is warranted, however, if hyperthermia develops in pregnant women. *Animal experiments and epidemiologic analysis both indicate that during pregnancy it is dangerous for body temperatures to exceed 39°C (102.2°F).* During the first half of pregnancy, excessive body temperatures are likely to produce birth defects, whereas during the latter half, birthweight is more likely to be affected. The fifth week after conception, the period of neural tube closure, is a particularly vulnerable time for the fetus.[88] Because women in the early stages of pregnancy may be unaware of their condition, it is critical that heat stress be promptly treated in women of child-bearing age. When exercising, it is also important that pregnant (or potentially pregnant) women acclimatize gradually to extreme thermal environments, remain well hydrated, wear loose-fitting clothing, exercise at a comfortable pace, and avoid swimming in warm water or immersion in hot tubs.[140]

Induced Alterations of the Regulated Temperature

The optimal body temperature is not always the same. In certain conditions of stress or vulnerability, the regulated temperature of the body may be altered. This is often an adaptive response to a particular physiologic crisis. Some of these situations are delineated here. In such circumstances, altered body temperature may be beneficial and should not necessarily be manipulated until the underlying condition is improved.

Fever

Increased body temperatures have been associated with illness for thousands of years. Based on the population data presented earlier, febrile body temperatures for resting, young adults include morning temperatures equal to or exceeding 37.3°C (99.2°F), increasing gradually to 37.8°C (100.0°F) for early afternoon and evening. Such elevated temperatures need to reflect a regulated increase to be considered a true fever. Pathogens and cancers are the usual causes of such increases in the regulated temperature, but they are not directly responsible for the increased body temperature. Rather, they interact with components of the immune system such as macrophages, T cells, monocytes, and Kupffer's cells, as well as with glial, epithelial, and many other types of cells. This interaction stimulates the cells to produce pyrogenic cyotokines,[93] including interleukin-1, interleukin-6, and macrophage inflammatory protein-1. Cytokines act on cells in the vicinity of the anterior hypothalamus, inducing them to release prostaglandin E_2, which leads to an increase in the regulated temperature.[82] Important avenues by which cytokine-mediated pyrogenic signals reach the brain include peripheral inputs from the abdominal vagus[124] nerve and central inputs via the subfornical organs.[143] Aspirin and related drugs block fever by inhibiting prostaglandin synthesis.[82] Interleukin-1 and other cytokines have many effects in addition to causing fever; these include decreased appetite, hypoferremia, activation of B and T lymphocytes, and increased slow-wave sleep.[80]

The increase in body temperature during fever aids many immune functions; neutrophil migration, release of reactive oxygen intermediates and nitric oxide by neutrophils, and interferon production are all augmented. It has been suggested that the most important aspect of fever is to greatly increase temperatures of the peripheral tissues via selection of a warmer microclimate, increased insulation, and postural changes. As temperatures increase from typical peripheral tissue temperatures (29° to 33°C [84.2° to 91.4°F]) to those approximating core temperature, activation, proliferation, and effector production in peripheral cells involved in cell-mediated and humoral immunity show temperature coefficients (Q_{10}) of 100 to 1000. In contrast, the Q_{10} for the effectiveness of the newly created effectors themselves, as well as for antigen-

nonspecific defense systems, are much lower, at about 1.5 to 5.36.[80,81,126]

The presence and beneficial effects of fever have been documented in a variety of cold- and warm-blooded vertebrates, and even in some invertebrates. *Under most conditions, it is probably not advisable to alleviate a fever. Exceptions include malignant hyperthermia, and particularly high fevers during pregnancy.*[80,81] *In addition, for patients with limited fluid, oxygen transport, or cardiopulmonary reserves, a febrile response should be treated with antipyretics.*[76] Consequences of the decreased immune response can be treated after the emergency.

Alcohol, Anesthetics, and Toxins

Increases in the blood concentrations of ethanol, anesthetics, and a number of toxic substances lead to substantial decreases in body temperature.[34,133] In many cases, this fall is caused by a decrease in the regulated temperature. In the case of high concentrations of alcohol and certain toxins, the reduction appears to be an adaptive adjustment that promotes survival. These chemicals disrupt protein structures within the cell membrane, an effect that is counteracted by a lower temperature.[34] Indeed, mouse studies have shown that a decreased body temperature counteracts ethanol toxicity.[94] A decrease in the regulated temperature of humans is caused by increases in blood ethanol concentration. After the ingestion of ethanol (3.0 mL/kg body weight) at 33°C (91.4°F), sweat rate increased and body temperature fell. Although the skin temperature did not increase, the subjects reported a warm sensation that paralleled the increase in sweat rate.[153] However, the lower blood ethanol levels associated with moderate consumption in humans have minimal, inconsistent effects on thermal balance of the whole body.[36,71]

Excellent overviews of the effects of general anesthetics on perioperative thermoregulation are available.[132,134] Many of these substances (halothane, fentanyl–nitrous oxide, enflurane, and isoflurane), in anesthetic doses, act in a similar manner. Heat loss thresholds are increased by about 1°C (1.8°F), and heat maintenance thresholds are lowered by approximately 2.5°C (4.5°F). Interestingly, in the typical clinical dose range, the gain (sensitivity) of the effector responses is nearly normal. *In the conditions under which general anesthetics are normally administered, body temperature decreases significantly.* An initial rapid drop is due to redistribution of heat; cool blood from the periphery lowers the central core temperature. A second, slower decrease results from a fall in body heat content. Finally, a plateau is reached, either because heat production and heat loss are passively balanced or because heat maintenance thresholds are reached. During postanesthetic recovery, there is vigorous shivering. Anesthetic-induced hypothermia is reduced in patients with higher preoperative systolic blood pressure. This difference is associated with higher preoperative plasma norepinephrine levels, which may intensify the vasoconstriction response.[74] *Cutaneous warming before and during anesthesia prevents the development of hypothermia and decreases the incidence of infectious complications.*[84,132]

Severe Hypoxia and Endotoxin Shock

When the inspired oxygen concentration falls to 10% to 12%, a substantial decrease in the regulated temperature occurs. This reaction has been documented, using behavioral responses, in fish, amphibians, reptiles, and mammals.[48,150] For humans exercising in 28°C (82.4°F) water under eucapnic conditions, decreasing the inspired oxygen to 12% lowers the core temperature thresholds for vasoconstriction and shivering, and increases the rate of core cooling by 33%.[72] The value of the resultant lowered body temperature is clear: the affinity of hemoglobin for oxygen is increased and the overall metabolic rate is decreased. The mechanism underlying the change in the regulated temperature may involve differential sensitivities of central neurons; hypoxia specifically increases activity of the warm-sensitive neurons (denoted by W in Figure 4-4) in the PO/AH.[144]

A somewhat similar regulated hypothermic response occurs when an animal is exposed to very high levels of pyrogens; the same response occurs under less extreme conditions in weak or malnourished animals. The lowered body temperature may serve to decrease the energetic costs of maintaining a high body temperature for a severely compromised animal.[125]

Altered System Responsiveness

A number of situations alter responsiveness of the thermoregulatory system. An awareness of these conditions is important in assessing the thermoregulatory capabilities of a particular person and in determining possible causes for hyperthermia or hypothermia.

Thermal Acclimation

Thermoregulation is affected by chronic exposure to very cold environments as well as by chronic exercise in cool or warm environments. Such exercise in a cool environment greatly increases the responsiveness of the sweat glands; if the exercise is in the heat, the central temperature at which sweating is initiated is also lowered. The net consequence of these adjustments is that a heat- and exercise-acclimated individual can work at a given level with far less increase in core temperature.[107] Exercise in humid heat appears to decrease the resting core temperature in acclimated individuals.[116] Increases in both core and skin temperatures contribute to the various changes involved in heat acclimation.[120] Physical training also increases the skin blood flow at any given increase in core temperature.[70] Although acclimation to warm conditions produces many changes in the cardiovascular system, the basic baroreflex responses are not altered.[151]

Chronic cold exposure alters many thermoregulatory systems and involves long-term evolutionary changes as well as thermal acclimation. In many cases, resting metabolic rate is increased after repeated cold exposure,[15,157] but repeated exposure to very cold environments may produce the opposite effect. For example, eighty 30-minute sessions at 5°C (41°F) decreased the metabolic response to a standard cold air test and often led to lower internal temperatures in these cold-acclimated individuals.[61]

When extremities are initially exposed to cold, strong vasoconstriction is seen. At some point, however, cold-induced removal of the superficial a-receptor inhibition leads to vasodilation of the fingers, which warms the extremities and protects against frostbite. This response has a genetic component; for individuals who are not cold acclimated, this response is very strong among Inuit Eskimos, moderate among Caucasians, and minimal among Chinese from Hong Kong. *Individual differences in the vasodilation response to cold determine the relative likelihood of the development of frostbite.* An environmental influence in this response is also likely, as fishermen in north-

eastern Canada did not exhibit vasoconstriction of the fingers when exposed to cold.[50]

Competition with Other Homeostatic Systems

In addition to a constant core temperature, the body has many other requirements. *When fluid balance or energy requirements are not met, thermoregulatory responses can be compromised.* For heat production and heat conservation, an adequate energy supply, a patent nervous system, and functional effector organs are critical. Thus, hypoglycemia decreases the core temperature at which shivering is initiated, while leaving the thresholds for sweating and vasodilation unaffected.[114]

For maintaining exercise performance and body temperature in a warm environment, body water status is critical.[101,128] It is common for a person working in the heat to lose 1 L of water per hour, and even when fluids are readily available, maintaining a euhydrated state may be difficult. *For hypohydration during activity, each percent decrease in body weight leads to a core temperature increase of about 0.15°C (0.27°F).* This decreased heat dissipation is mediated by two mechanisms. At a given core temperature, hypertonicity decreases the sweating response and hypovolemia reduces skin blood flow.[128] Hyperhydration has no effect on thermoregulation during compensable exercise-heat stress.[85] During uncompensable exercise-heat stress, hyperhydration slightly increases the time to exhaustion, but only by delaying hypohydration; thermoregulation is not affected.[86] It is important to be alert to the possibility of dehydration in many atypical situations. For example, swimmers training in an outdoor pool with a water temperature of 26°C (78.8°F) lost sufficient fluid in 3 hours (2.5% of their body weight) to compromise thermoregulatory responses.[139]

Hypoxia can undermine thermoregulatory vascular responses because of demands that compete or operate at cross purposes. Thus, in a hot environment, hypoxia reduces skin blood flow at a given sweat rate. In the cold, hypoxia enhances core cooling by delaying the onset of vasoconstriction and cooling.[99]

Alcohol, Drugs, Anesthetics, and Toxins

Although moderate doses of anesthetics and toxins may elicit adaptive changes in the regulated body temperature, *elevated doses of these substances impair or abolish both autonomic and behavioral aspects of thermoregulation.* Body temperature then changes passively, depending on the thermal environment. This can be particularly dangerous when elevated levels of alcohol or similar substances are combined with heat stress. An impaired ability to dissipate heat is then combined with the enhanced toxicity of increased tissue temperature. Drugs that increase metabolic heat production, such as amphetamine, ecstasy (MDMA), and cocaine are also particularly dangerous in the heat. In addition, cocaine interferes with heat dissipation mechanisms and the perception of warmth.[14,30]

Severe and Unusual Situations

The frequency and intensity of prior exposure in extreme thermal conditions affects subsequent thermoregulatory responses. While well-hydrated, sedentary individuals showed no carryover effects from exercise conducted the preceding day,[97] the sweating threshold was increased by 0.33°C (0.6°F) after intense exercise (85% of V_{O_2} peak). Some of the effect on the sweating response may have been due to a post-exercise decrease in blood pressure.[77] In the cold, similar effects occurred. After 3.5 days of sustained military operations, the threshold for shivering decreased by 1°C (1.8°F), and the steady state metabolic heat production at 10°C (50.0°F) was lower (55 $W \cdot m^{-2}$ versus 67 $W \cdot m^{-2}$).[23] At the other extreme, 14 days of bed rest increased the threshold and decreased the sensitivity of both sweating and cutaneous vasodilation in response to heat stress.[98]

In some situations, physiologic responses are minimally affected by exercise and thermal stress. After 1 hour of exercise (60% V_{O_2} peak) in a hot (38°C [100.4°F]) or cold (10°C [50.0°F]) environment, normal immune system function showed similar alterations during the recovery period for the two conditions. However, it was concluded that the levels of thermal stress used did not compromise overall immune function.[96] Likewise, patients with healed third-degree burns over as much as 60% of their body surface area showed normal changes in sweat rate, rise in core temperature, and increase in heart rate during moderate exercise in the heat.[3]

ACKNOWLEDGMENTS

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The references for this chapter can be found on the accompanying DVD-ROM.

Accidental Hypothermia

5

Daniel F. Danzl

Although used for medical purposes for millennia, cold modalities were not scientifically evaluated until the 18th century. The hemostatic, analgesic, and therapeutic effects of cold on various conditions were well known. Biblical references cite truncal rewarming of King David by a damsel; various remedies were mentioned by Hippocrates, Aristotle, and Galen.[233] Folklore from Maine still recounts the buggy ride of "Frozen Charlotte."

The effects of cold on human performance are perhaps best documented in military history.[216] Frosty conditions have decided many battles.[123] Numerous casualties occur even in training, such as four students of the U.S. Army Ranger School in 1995. Most cold injuries encountered today affect the urban destitute and the wilderness and sports enthusiasts, such as skiers, hunters, sailors, climbers, and swimmers.[5,144,256] The popularity of Arctic and mountain expeditions has increased the number of persons at risk.[2,137] Among those challenging the environment and at risk of cooling are climbers of Mt. Everest, Mt. Hood, and Mt. McKinley.[114,204]

EPIDEMIOLOGY

In most countries, primary hypothermia deaths are considered violent and are classified as accidental, homicidal, or suicidal.[34] Deaths from secondary hypothermia are usually considered natural complications of systemic disorders, including trauma, carcinoma, and sepsis. The true incidence of secondary hypothermia throughout the world is unknown because hypothermic persons found indoors usually have other serious and diverting secondary medical illnesses. In addition, delays are common between hospital admission and death, so secondary hypothermia is significantly underreported. In contrast, death certificate data more accurately quantify primary hypothermia.

Hypothermia occurs in various locations and in all seasons.[213,271,312] In a multicenter North American survey of 428 cases of civilian accidental hypothermia, 69 occurred in Florida.[53,293] Urban settings account for the majority of cases in most of the industrialized countries.[53,148,286] There is an annual average of 600 deaths attributed to primary hypothermia in the United States. Around half of these fatalities are in patients over age 65 years.[35]

The reason for any year-to-year decline in fatalities is unknown. Of note, the incidence of secondary hypothermia fatalities is far greater, but there are no reliable histologic criteria to confirm that hypothermia is the cause of death.[239]

Classifications

Accidental hypothermia is best defined as the unintentional decrease of around 2°C (3.6°F) in the "normal" core temperature of 37.2° to 37.7°C (98.9° to 99.9°F) without disease in the preoptic and anterior hypothalamic nuclei (Table 5-1). Classically, hypothermia is defined as a core temperature below 35°C (95°F). Hypothermia is both a symptom and a clinical disease entity. When sufficient heat cannot be generated to maintain homeostasis and the core temperature drops below 30°C (86°F), the patient becomes poikilothermic and cools to the ambient temperature.[178]

Among the clinical classifications, the most practical division includes healthy patients with simple environmental exposure (primary), those with specific diseases that produce hypothermia (secondary), and those with predisposing conditions. Other divisions that reflect the etiology of hypothermia include immersion versus nonimmersion, and acute versus chronic heat loss.[198,217]

Various physiologic stressors and other factors can impair thermoregulation.[264] Age extremes, state of health and nutrition, type of exposure, and a multitude of intoxicants or medications can jeopardize thermostability by decreasing heat production or increasing heat loss. Physiologic stressors also include dehydration, sleep deprivation, and fatigue. These challenges increase heat loss via evaporation, radiation, conduction, and convection, and compensatory responses often fail.[198] The resulting mortality rates range to well over 50% in many clinical series, depending largely on the severity of risk factors and on patient selection criteria.[53,213,247,312]

For safety, experimental investigations of induced hypothermia in human volunteers usually terminate cooling at about 35°C (95°F). Naturally, this precludes analysis of some of the more significant pathophysiologic features of moderate or severe hypothermia. Design limitations also occur in studies of anesthetized animals, because the results of these experiments require variable degrees of extrapolation to humans. For example, large differences exist both in the cardiovascular responses to interventions and in the amounts of peripheral musculature that are present, particularly in nonporcine animal models. As a result, clinical treatment recommendations must

TABLE 5-1. Fahrenheit-to-Celsius Conversion Scale*

FAHRENHEIT	CELSIUS*
95	**35.00**
94	34.44
93	33.89
92	33.33
91	32.78
90	32.22
89	31.67
88	31.11
87	30.56
86	**30.00**
85	29.44
84	28.89
83	28.33
82	27.78
81	27.22
80	26.67
79	26.11
78	25.56
77	**25.00**
76	24.44
75	23.89
74	23.33
73	22.78
72	22.22
71	21.67
70	21.11
69	20.56
68	**20.00**
67	19.44
66	18.89
65	18.33
64	17.78
63	17.22
62	16.67
61	16.11
60	15.56
59	**15.00**
58	14.44
57	13.89
56	13.33
55	12.78
54	12.22
53	11.67
52	11.11
51	10.56
50	**10.00**
49	9.44
48	8.89
47	8.33
46	7.78
45	7.22
44	6.67
43	6.11
42	5.56
41	**5.00**
40	4.44
39	3.89
38	3.33
37	2.78
36	2.22
35	1.67
34	1.11
33	0.56
32	**0.00**

*C = (F − 32) × 5/9.

be predicated on the degree and duration of hypothermia and on the predisposing factors that are subsequently identified.[112]

NORMAL PHYSIOLOGY OF TEMPERATURE REGULATION

Warm-blooded animals maintain a precariously dynamic equilibrium between heat production and heat loss.[146,147] The normal diurnal variation in humans is only 1°C (1.8°F). Because physiologic changes occurring in humans are modified by predisposing or contributory factors, the normal responses to severe temperature depression require significant extrapolation.[200,222]

Basal heat production usually averages 40 to 60 kcal/m^2 of body surface area per hour, which approximates the heat from a 100-watt incandescent bulb. This increases with shivering thermogenesis,[212] food ingestion, fever, activity, and cold stress. Normal thermoregulation in vertebrates involves transmitting cold sensation to hypothalamic neurons via the lateral spinothalamic tracts and the thalamus (Fig. 5-1). The physiologic characteristics of the four zones of hypothermia appear in Table 5-2. A complete discussion of the physiology of cold exposure and thermoregulation is presented in Chapter 7.

PATHOPHYSIOLOGY

Nervous System

Numbing cold depresses the central nervous system, producing impaired memory and judgment, slurred speech, and decreased consciousness. During cold-weather expeditions, leaders are prone to risk-taking behavior and its attendant trauma. Temperature-dependent enzyme systems in the brain do not function properly at cold temperatures that are well tolerated by the kidneys. As a result, most patients are comatose below 30°C (86°F), although some remain amazingly alert.

Neurons are initially stimulated by a 1°C (1.8°F) drop in temperature, but the brain does not always cool uniformly during accidental hypothermia. After the initial increase, there is a linear decrease in cerebral metabolism by 6% to 10% per degree centigrade from 35°C (95°F) to 25°C (77°F). Hypothermia can afford cerebral protection because of the diminished cerebral metabolic requirements of oxygen.[96]

The electroencephalogram is abnormal below 33.5°C (92.3°F) and becomes silent at 19° to 20°C (66.2° to 68°F). The triphasic waves commonly noted in hypothermia are also observed in various metabolic, toxic, and diffuse encephalopathies. Visual evoked potentials, another objective measure of cerebral function, become smaller as the mercury drops. After cerebral cortical function becomes impaired, lower brainstem functions are also deranged.

Cerebrovascular autoregulation is protectively intact until the temperature drops below 25°C (77°F). Although vascular resistance is increased, blood flow is disproportionately redistributed to the brain. In canine studies, blood flow in the brain, muscle, kidney, and myocardium recovers quickly to control levels after rewarming. Flow deficits persist in the pulmonary, digestive, and endocrine systems for up to 2 hours after rewarming.

Chilling the peripheral nervous system increases muscle tension and preshivering tone, eventually leading to shivering.

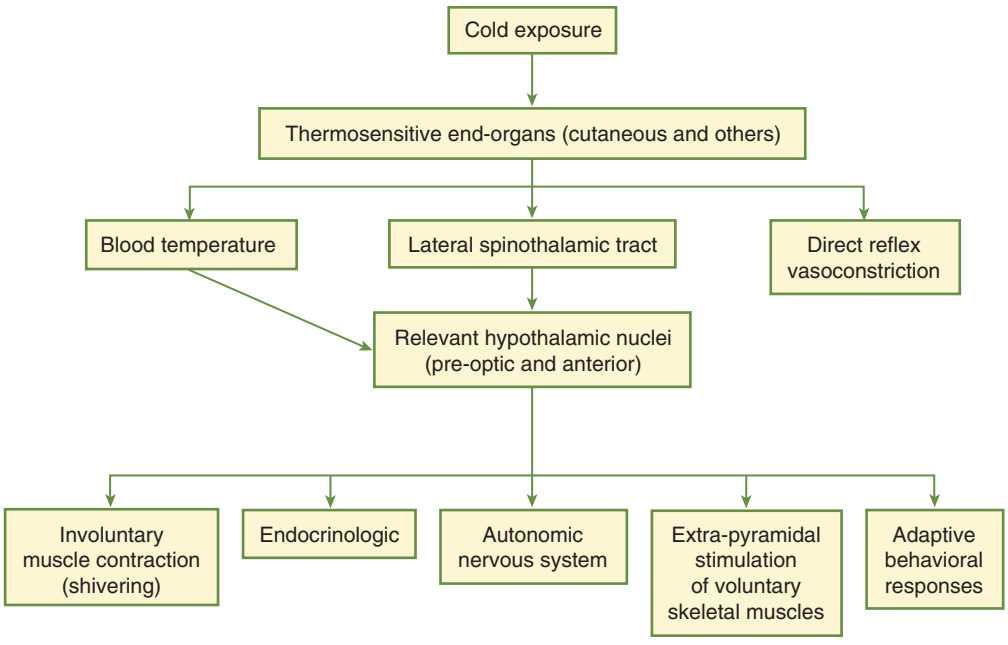


Figure 5-1. Physiology of cold exposure.

TABLE 5-2. Characteristics of the Four Zones of Hypothermia

STAGE	CORE TEMPERATURE °C	CORE TEMPERATURE °F	CHARACTERISTICS
Mild	37.6	99.6 ± 1	Normal rectal temperature
	37.0	98.6 ± 1	Normal oral temperature
	36.0	96.8	Increase in metabolic rate, blood pressure, and preshivering muscle tone
	35.0	95.0	Urine temperature 34.8°C (94.6°F); maximal shivering thermogenesis
	34.0	93.2	Development of amnesia, dysarthria, and poor judgment; maladaptive behavior; normal blood pressure; maximal respiratory stimulation; tachycardia, then progressive bradycardia
	33.30	91.4	Development of ataxia and apathy; linear depression of cerebral metabolism; tachypnea, then progressive decrease in respiratory minute volume; cold diuresis
Moderate	32.0	89.6	Stupor; 25% decrease in oxygen consumption
	31.0	87.8	Extinguished shivering thermogenesis
	30.0	86.0	Development of atrial fibrillation and other arrhythmias; poikilothermia; pupils and cardiac output two thirds of normal; insulin ineffective
	29.0	85.2	Progressive decrease in level of consciousness, pulse, and respiration; pupils dilated; paradoxical undressing
Severe	28.0	82.4	Decreased ventricular fibrillation threshold; 50% decrease in oxygen consumption and pulse; hypoventilation
	27.0	80.6	Loss of reflexes and voluntary motion
	26.0	78.8	Major acid–base disturbances; no reflexes or response to pain
	25.0	77.0	Cerebral blood flow one third of normal; loss of cerebrovascular autoregulation; cardiac output 45% of normal; pulmonary edema may develop
	24.0	75.2	Significant hypotension and bradycardia
	23.0	73.4	No corneal or oculocephalic reflexes; areflexia
	22.0	71.6	Maximal risk of ventricular fibrillation; 75% decrease in oxygen consumption
Profound	20.0	68.0	Lowest resumption of cardiac electromechanical activity; pulse 20% of normal
	19.0	66.2	Electroencephalographic silencing
	18.0	64.4	Asystole
	13.7	56.8	Lowest adult accidental hypothermia survival[94]
	15.0	59.2	Lowest infant accidental hypothermia survival[227]
	10.0	50.0	92% decrease in oxygen consumption
	9.0	48.2	Lowest therapeutic hypothermia survival[222]

Shivering, which is also centrally controlled, is a much more efficient heat producer than voluntary muscle contractions of the extremities.

Cardiovascular System

Many cardiovascular responses caused by or associated with hypothermia are well described but not well understood.[180,181] Cold stress increases the consumption of myocardial oxygen. Autonomic nervous system stimulation causes tachycardia and peripheral vasoconstriction, both of which increase systemic blood pressure and cardiac afterload.

As the core temperature drops, there is a fairly linear decrease in pulse rate. After a premonitory tachycardia, decremental bradycardia produces a 50% decrease in heart rate at 28°C (82.4°F). Because this bradycardia is caused by decreased spontaneous depolarization of the pacemaker cells, it is refractory to atropinization. If there is a "relative" tachycardia not consistent with the degree of hypothermia, the clinician should be concerned. Associated conditions include occult trauma with hypovolemia, drug ingestion, and hypoglycemia.

During hypothermic bradycardia, unlike normothermia, systole is prolonged longer than diastole. In addition, the conduction system is much more sensitive to cold than is the myocardium, so the cardiac cycle is lengthened. Cold-induced changes in pH, oxygen, electrolytes, and nutrients also alter electrical conduction.[197]

Hypothermia progressively decreases mean arterial pressure and cardiac index. Cardiac output drops to about 45% of normal at 25°C (77°F). Systemic arterial resistance, determined by invasive hemodynamic monitoring, is increased. Even after rewarming, cardiovascular function may remain temporarily depressed, with impaired myocardial contractility, metabolism, and peripheral vascular function.[292]

Mild, steady hypothermia in patients with poikilothermic thermoregulatory disorders causes electrocardiographic (ECG) alterations and conduction abnormalities. First the PR, then the QRS, and most characteristically the QTc intervals are prolonged. Clinically invisible increased preshivering muscle tone can obscure the P waves; ST segment and T wave abnormalities are inconsistent.[297]

The J wave (Osborn wave or hypothermic hump; Fig. 5-2), first described by Tomaszewski in 1938, occurs at the junction of the QRS complex and the ST segment. It is not prognostic but is potentially diagnostic.[100,228] J waves occur at any temperature below 32.2°C (90°F) and are most frequently seen in leads II and V_6. When the core temperature falls below 25°C (77°F), J waves are found in the precordial leads (especially V_3 or V_4). The size of the J waves also increases with temperature depression, but it is unrelated to arterial pH. J waves are usually upright in aV_L, aV_F, and the left precordial leads.[154]

J waves may represent hypothermia-induced ion fluxes, resulting in delayed depolarization or early repolarization of the left ventricle, or there may be an unidentified hypothalamic or neurogenic factor. J waves are *not* pathognomonic of hypothermia but occur also with central nervous system lesions, focal cardiac ischemia, and sepsis. They may also be present in young, healthy persons. When pronounced, J waveform abnormalities can simulate a myocardial infarction. Computer software is not widely available that can successfully recognize and suggest the diagnosis of hypothermia.

The capability to differentiate between J waves and current that results from injury is important in rural and wilderness settings, because prehospital thrombolytic therapy will expand.[51] Thrombolysis is unstudied in hypothermia but would be expected to exacerbate coagulopathies.[98]

Below 32.2°C (90°F), all types of atrial and ventricular arrhythmias are encountered.[66] The His-Purkinje system is more sensitive to cold than is the myocardium. As a result, conduction velocity decreases and electrical signals can disperse. Because conduction time is prolonged more than the absolute refractory period, reentry currents can produce circus rhythms that initiate ventricular fibrillation (VF).

In addition to causing bradycardia, widening the QRS complex, and prolonging the QT interval, hypothermia increases the duration of action potentials (Fig. 5-3).[17,18] During rewarming, nonuniform myocardial temperatures can disperse conduction and further increase the action potential duration, another mechanism to develop the unidirectional blocks that facilitate reentrant arrhythmias. At temperatures between 25° and 20°C (77° and 68°F), myocardial conduction time is prolonged further than the absolute refractory period. Another arrhythmogenic mechanism is the development of independent electrical foci that precipitate arrhythmias.

Various electrolyte abnormalities can further complicate the situation during hypothermic conditions, because they exacerbate the effects of prolonged action potentials. Most conspicuously, hypothermia-induced cellular calcium loading mimics digitalis toxicity and may predispose to a forme fruste of torsades de pointes.

Hypothermia-induced VF and asystole often occur spontaneously below 25°C (77°F). The VF threshold and the transmembrane resting potential are decreased. Because the heart is cold, the conduction delay is facilitated by the large dispersion of repolarization, and the action potential is prolonged. The increased temporal dispersion of the recovery of excitability is linked to VF. In a way, nature's model of resistance to VF is the heart of hibernating animals during rewarming.[143] Animals with this capacity seem to be protected by a shortened QT duration and a calcium channel handling system that prevents intracellular calcium overload.

Asystole and VF may both result from hypovolemia, tissue hypoxia, therapeutic manipulations, acid–base fluxes, autonomic dysfunction, and coronary vasoconstriction coupled with increased blood viscosity. Other causes may include rough handling or jostling, sudden vertical positioning, and acute metabolic stress from exertion or very rapid rewarming.

Core Temperature Afterdrop

Core temperature afterdrop refers to the continued decline in a hypothermic patient's temperature after removal from the cold (see also Chapter 6). Contributing to afterdrop is the simple temperature equilibration between the warmer core and the cooler periphery. Circulatory changes account for another set of observations. The countercurrent cooling of blood that perfuses cold extremities results in a core temperature decline until the existing temperature gradient is eliminated. In cold-water immersion, post-rescue collapse may also result from abrupt hypotension after loss of the hydrostatic squeeze in the water.

The incidence and magnitude of core temperature afterdrop vary widely in clinical experiments and in surgically induced

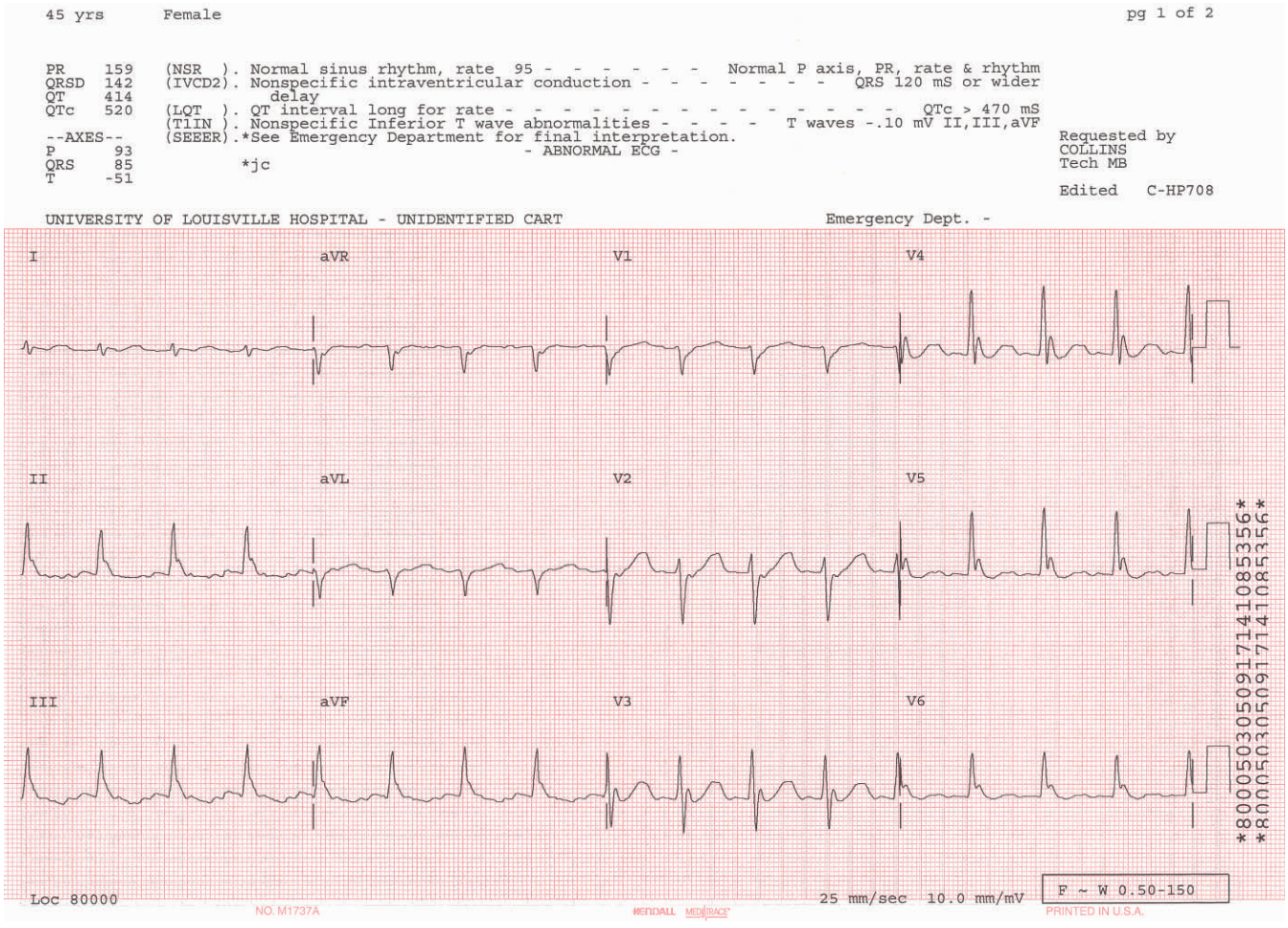


Figure 5-2. The J or Osborn wave of hypothermia. Note that the obvious J waves are not computer interpretable.

hypothermia.[90–92,112] Hayward measured his own esophageal, rectal, tympanic, and cardiac temperatures (via flotation tip catheter) during rewarming after being cooled in 10°C (50°F) water.[115] On three different days, rewarming was achieved via shivering thermogenesis, heated humidified inhalation, and warm bath immersion. Coincident with a 0.3°C (0.5°F) afterdrop during warm bath immersion, his mean arterial pressure fell 30% and his peripheral vascular resistance fell 50%. Therefore, the circulatory mechanism is another major contributor to afterdrop.

A human study of peripheral blood flow during rewarming from mild hypothermia suggests that minimal skin blood flow changes can also lead to afterdrop (Fig. 5-4). The largest core temperature afterdrops occur when subjects are rewarmed with plumbed garments and heating pads.

In summary, core temperature afterdrop appears to become most clinically relevant when a large temperature gradient exists between the periphery and the core, particularly in dehydrated, chronically cold patients. Both conductive and convective mechanisms are responsible for afterdrop.[92] Stimulating peripheral blood flow can increase afterdrop. Major afterdrops are also observed when frostbitten extremities are thawed before crystalloid volume resuscitation and thermal stabilization of the core temperature.

Respiratory System

Any exposure to a big chill initially stimulates the respiratory drive, which is followed by progressive depression of the respiratory minute volume as cellular metabolism is depressed. The respiratory rate often falls to 5 to 10 breaths/min below 30°C (86°F), and ultimately brainstem neurocontrol of ventilation fails. An important physiologic observation is that carbon dioxide production drops 50% for each 8°C (14°F) fall in temperature. In severe hypothermia, carbon dioxide retention and respiratory acidosis reflect the aberrant responses to normothermic respiratory stimuli.[86]

Other pathophysiologic factors contributing to a ventilation–perfusion mismatch include decreased ciliary motility, increased quantity and viscosity of secretions, hypothermic acute respiratory distress syndrome, and noncardiogenic pulmonary edema.[287] The thorax loses elasticity and pulmonary compliance drops. The respiratory "bellows" stiffen and fail

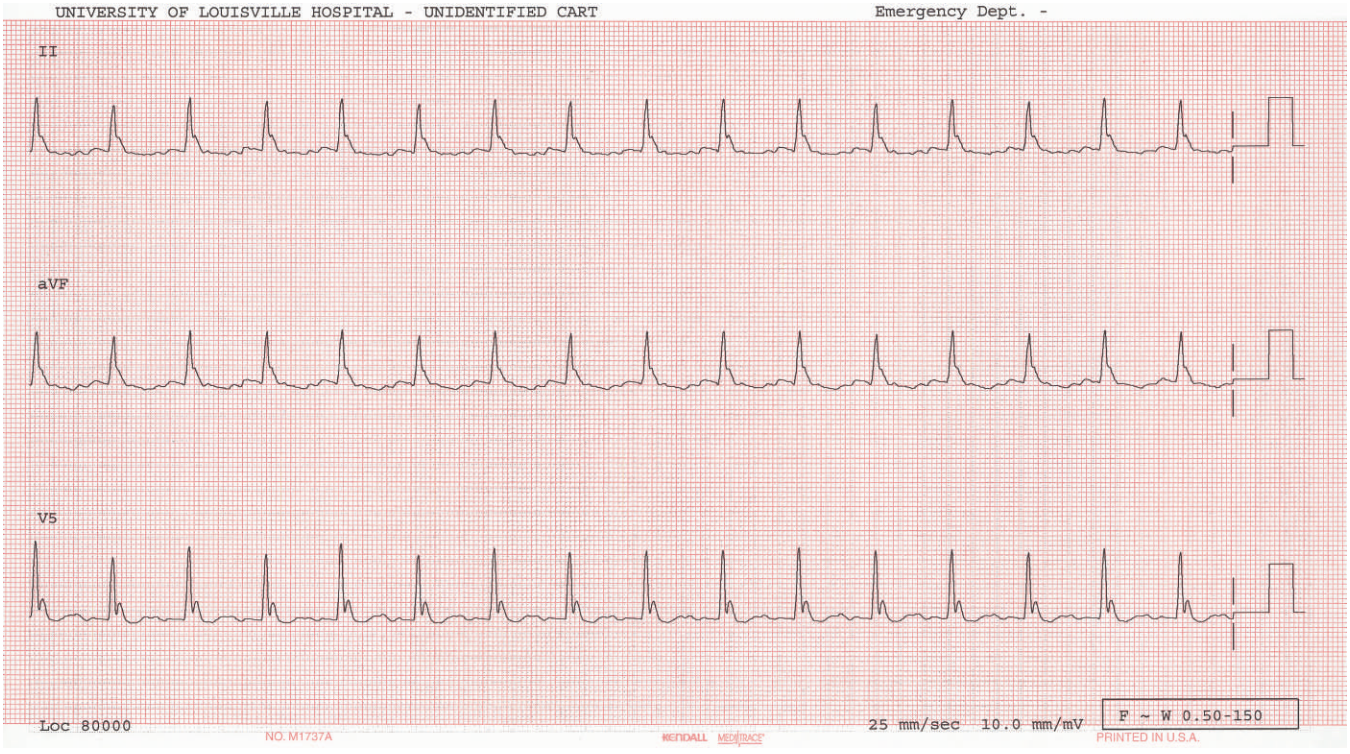


Figure 5-2, cont'd. The J or Osborn wave of hypothermia. Note that the obvious J waves are not computer interpretable.

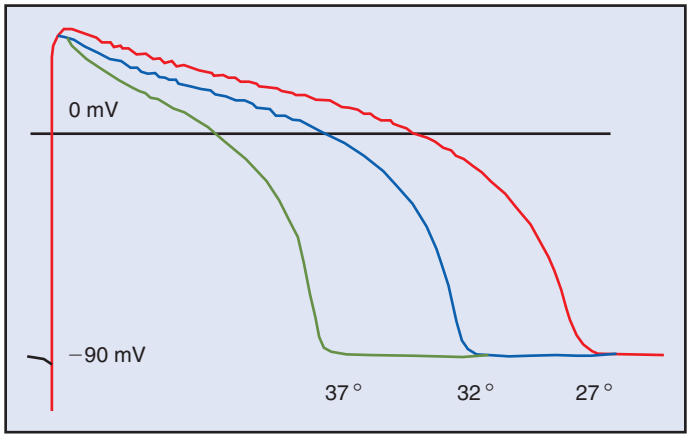


Figure 5-3. Example of the effects of temperature on action potentials in cardiac cells.

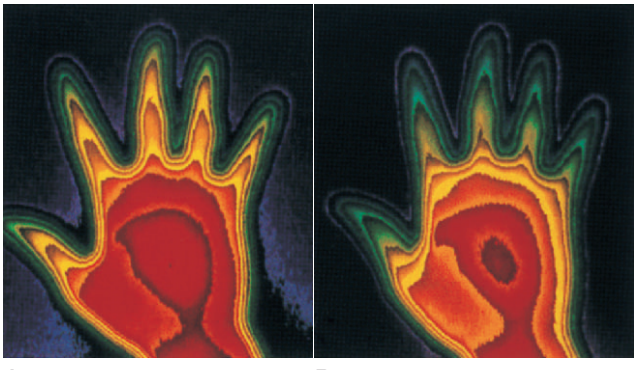


Figure 5-4. Infrared scan of the palmar hand surface. *Blue* = 43°C (109.4°F); *red* = 68°C (154.4°F). **A,** At room temperature. **B,** After 5 minutes in a cold room, with evidence of vasoconstriction. (Courtesy Naval Health Research Center, San Diego, CA.)

because the contractile efficiency of the intercostal muscles and diaphragm declines.

Pertinent potentially protective or detrimental factors that affect tissue oxygenation in endothermic humans are listed in Box 5-1.

Renal System

The kidneys respond briskly to hypothermia-induced changes in the vascular tree's capacitance. Peripheral vasoconstriction can result initially in a relative central hypervolemia, producing diuresis, even with mild dehydration. In addition, renal blood flow is depressed by 50% at 27° to 30°C (80.6° to 86°F), which decreases the glomerular filtration rate. Nevertheless, there is an initial large diuresis of this dilute glomerular filtrate, which does not efficiently clear nitrogenous wastes.

The etiology of the cold diuresis is multifactorial.[102,103] Some of the suggested mechanisms include inhibition of antidiuretic hormone (ADH) release and decreased renal tubular function.

Box 5-1. Oxygenation Considerations during Hypothermia

DETRIMENTAL FACTORS

- Oxygen consumption increases with rise in temperature; use caution if rewarming is rapid; shivering also increases demand
- Decreased temperature shifts oxyhemoglobin dissociation curve to the left
- Ventilation–perfusion mismatch; atelectasis; decreased respiratory minute volume; bronchorrhea; decreased protective airway reflexes
- Decreased tissue perfusion from vasoconstriction; increased viscosity
- "Functional hemoglobin" concept: capability of hemoglobin to unload oxygen is lowered
- Decreased thoracic elasticity and pulmonary compliance

PROTECTIVE FACTORS

- Reduction of oxygen consumption by 50% at 28°C (82.4°F), 75% at 22°C (71.6°F), 92% at 10°C (50°F)
- Increased oxygen solubility in plasma
- Decreased pH and increased $PaCO_2$ shift oxyhemoglobin dissociation curve to right

Neither hydration nor ADH infusions seem to influence the diuretic response, which appears to be an attempt to compensate for initial relative central hypervolemia caused by vasoconstrictive overload of capacitance vessels.

The diuresis may also be pressure related, caused by impaired autoregulation in the kidney. Cold diuresis has circadian rhythmicity and correlates with periods of shivering. Cold water immersion increases urinary output 3.5 times, and the presence of ethanol impressively doubles that diuresis.

Coagulation

Coagulopathies often develop in hypothermic patients because the enzymatic nature of the activated clotting factors is depressed by cold.[76] In vivo, prolonged clotting is proportional to the number of steps in the cascade. For example, at 29°C (84.2°F), a 50% to 60% increase in the partial thromboplastin time (PTT) would be expected. Kinetic tests of coagulation, however, are performed in the laboratory at 37°C (98.6°F). As the blood warms in the machine, the enzymes between the factors in the cascade are activated. The sample of warmed in vitro blood then clots normally.[68]

The reversible hemostatic defect created by hypothermia may not be reflected by the "normal" prothrombin time (PT), PTT,[250] or International Normalized Ratio (INR). This coagulopathy is basically independent of clotting factor levels and cannot be confirmed by laboratory studies performed at 37°C. Treatment is rewarming and not simply administration of clotting factors.[246]

Thrombocytopenia as a cause of bleeding becomes progressively significant in severe hypothermia. Proposed mechanisms include direct bone marrow suppression and splenic or hepatic sequestration. Thromboxane B_2 production by platelets is also temperature dependent, so cooling skin temperature produces reversible platelet dysfunction. Thrombocytopenia is a common but poorly recognized corollary of hypothermia in older adults and neonates.

Coagulopathy in trauma patients is attributed to enzyme inhibition, platelet alteration, and fibrinolysis.[276] The critical temperature at which enzyme activity slows significantly is 34°C (93.2°F).[306] In addition, clot strength weakens as a result of platelet malfunction. Fibrinolysis is not significantly affected at any temperature in the range measured (33° to 37°C [91.4° to 98.6°F]) (see Trauma, later).

Physiologic hypercoagulability also develops during hypothermia, with a sequence similar to that seen in disseminated intravascular coagulation (DIC). This produces a higher incidence of thromboembolism during hypothermia. Causes include thromboplastin release from cold tissue, simple circulatory collapse, and release of catecholamines and steroids. Because fibrin split-product levels can be normal, bleeding is not always considered a hematologic manifestation of DIC.

Whole blood viscosity increases with the hemoconcentration seen after diuresis and the shift of fluid out of vascular compartments. Red blood cells (RBCs) simply stiffen and have diminished cellular deformity when chilled.[237] The elevated viscosity of hypothermia is also exacerbated with cryoglobulinemia. Cryofibrinogen is a cold-precipitated fibrinogen occasionally seen with carcinoma, sepsis, and collagen vascular diseases. Blood viscosity is also increased by the transient increase in platelet and RBC counts seen with mild surface cooling. This could explain the increased mortality from coronary and cerebral thromboses that occur in winter.[198]

PREDISPOSING FACTORS

The factors that predispose to hypothermia can be separated into those that decrease heat production, those that increase heat loss, and those that impair thermoregulation.[198] There is significant overlap between these groups (Box 5-2).

Decreased Heat Production

Thermogenesis is decreased at both extremes of age. In older adults, neuromuscular inefficiency and decreased physical activity impair shivering. Aging progressively diminishes homeostatic and cold adaptive capabilities. Although most older adults have normal thermoregulation, they tend to develop conditions that impair heat conservation.[241,244]

Older adults are physiologically less adept at increasing heat production and the respiratory quotient (the ratio of the volume of carbon dioxide produced to the volume of oxygen consumed per unit of time). Impaired thermal perception, possibly caused by decreased resting peripheral blood flow, leads to poor adaptive behavior. Metabolic studies also demonstrate that in severely hypothermic older adults, lipolysis occurs in preference to glucose consumption.[221,234]

Neonates have a large surface area–to–mass ratio, a relatively deficient subcutaneous tissue layer, and virtually no behavioral defense mechanisms. Newborn "unadapted" infants attempt to thermoregulate with initial vasoconstriction and acceleration of metabolic rate. In contrast, "adapted" infants who are older than 5 days can increase lipolysis immediately and burn oxidative brown adipose tissue.

No cause-and-effect relationship has been found between hypothermia and the mortality rate of premature infants.[116,259] Although the smaller infants in a neonatal intensive care unit are at the greatest risk of hypothermia, mortality is related to hypothermia only in larger neonates.

Box 5-2. Factors Predisposing to Hypothermia

DECREASED HEAT PRODUCTION

Endocrinologic Failure
- Hypopituitarism
- Hypoadrenalism
- Hypothyroidism
- Lactic acidosis
- Diabetic and alcoholic ketoacidosis

Insufficient Fuel
- Hypoglycemia
- Malnutrition
- Marasmus
- Kwashiorkor
- Extreme physical exertion

Neuromuscular Physical Exertion
- Age extremes
- Impaired shivering
- Inactivity
- Lack of adaptation

IMPAIRED THERMOREGULATION

Peripheral Failure
- Neuropathies
- Acute spinal cord transection
- Diabetes

Central or Neurologic Failure
- Cardiovascular accident
- Central nervous system trauma
- Toxicologic effects
- Metabolic failure
- Subarachnoid hemorrhage
- Pharmacologic effects
- Hypothalamic dysfunction
- Parkinson's disease
- Anorexia nervosa
- Cerebellar lesion
- Neoplasm
- Congenital intracranial anomalies
- Multiple sclerosis
- Hyperkalemic periodic paralysis

INCREASED HEAT LOSS

Environmental
- Immersion
- Nonimmersion

Induced Vasodilation
- Pharmacologic effects
- Toxicologic effects

Erythrodermas
- Burns
- Psoriasis
- Ichthyosis
- Exfoliative dermatitis

Iatrogenic
- Emergency childbirth
- Cold infusions
- Heatstroke treatment

MISCELLANEOUS ASSOCIATED CLINICAL STATES
- Multisystem trauma
- Recurrent hypothermia
- Episodic hypothermia
- Shapiro's syndrome
- Infections: bacterial, viral, parasitic
- Pancreatitis
- Carcinomatosis
- Cardiopulmonary disease
- Vascular insufficiency
- Uremia
- Paget's disease
- Giant cell arteritis
- Sarcoidosis
- Shaken baby syndrome
- Systemic lupus erythematosus
- Wernicke-Korsakoff syndrome
- Hodgkin's disease
- Shock
- Sickle cell anemia
- Sudden infant death syndrome

Emergency deliveries and resuscitations are responsible for most acute neonatal hypothermia. Other common risk factors are prematurity, low birth weight, inexperienced mother, perinatal morbidity, and low socioeconomic status. In babies with more chronically induced subacute hypothermia, lethargy, a weak cry, and failure to thrive are common.[289]

Many cold infants have "paradoxical rosy cheeks," looking surprisingly healthy. After the first few days of life, hypothermia frequently indicates septicemia and carries a high mortality rate. Low weight and malnutrition are common. Hypothermia in a low-birthweight neonate should suggest the possibility of intracranial hemorrhage; hypothermia is also observed in shaken baby syndrome.

Endocrinologic failure, including hypopituitarism, hypoadrenalism, and myxedema, commonly decreases heat production. Interestingly, congenital adrenal hyperplasia with mineralocorticoid insufficiency is more common in cold climates, possibly an adaptive response to prolonged exposure to cold temperatures, because "normal" cold diuresis is reduced in these patients.

Hypothyroidism is often occult, with no history of cold intolerance, dry skin, lassitude, or arthralgias. The physician should check for a thyroid scar or any history of thyroid hormone replacement. The degree of temperature depression correlates fairly directly with mortality. Eighty percent of patients in myxedema coma, which is several times more common in female patients, are hypothermic.

The effects of insufficient nutrition extend from hypoglycemia to marasmus to kwashiorkor. Kwashiorkor is less often associated with hypothermia than is marasmus, because of the

insulating effect of hypoproteinemic edema. Central neuroglycopenia distorts hypothalamic function. Many alcoholic patients with hypothermia are hypoglycemic. Malnutrition decreases insulative subcutaneous fat and directly alters thermoregulation. Poor nutrition predisposes to hypothermia and its attendant clumsiness in older adult women with femoral neck fractures. Partly because of fuel depletion, hypothermia is as great a threat as hyperthermia in marathon races run in cool climates. Runners slowing from fatigue or injury late in a race are at serious risk of hypothermia.

Increased Heat Loss

Poorly acclimated and insulated individuals often have high diaphoretic, convective, and evaporative heat losses during exposure to cold. Because the skin functions as a radiator, any dermatologic malfunction increases heat loss. Such erythrodermas include psoriasis, exfoliative dermatitis, and toxic epidermal necrolysis. Hypothermia with hypernatremic dehydration is also seen in congenital lamellar ichthyosis.

Burns and inappropriate burn treatment cause excessive heat loss, as do other iatrogenic factors, including massive cold intravenous (IV) infusions and overcooling heatstroke victims.[165] When carbon dioxide is used for abdominal insufflation before laparoscopy, warming the gas before administration helps prevent hypothermia. Environmental immersion exposure is discussed in detail in Chapter 6.[89]

Many pharmacologic and toxicologic agents both increase heat loss and impair thermoregulation.[146] The most common is ethanol, which interacts with every putative thermoregulatory neurotransmitter. Although ingestion of ethanol produces a feeling of warmth and perhaps visible flushing, it is the major cause of urban hypothermia.[53,213] In fatal cases of accidental hypothermia, many victims are under the influence of ethanol. In children with ethanol intoxication, hypothermia is common.

Ethanol is also a poikilothermia-producing agent that directly impairs thermoregulation at high or low temperatures.[145] Body temperature is lowered both from cutaneous vasodilation with radiative heat loss and from impaired shivering thermogenesis. Chronic ethanol ingestion damages the mamillary bodies and posterior hypothalamus, which modulates shivering thermogenesis.[238] Ethanol also increases the risk of being exposed to the environment, by modifying protective adaptive behavior. The ultimate example is paradoxical undressing, or removal of clothing in response to a cold stress. As an organic solvent, ethanol confers a few theoretically redeeming qualities in freezing cold injuries by lowering the cellular freezing point.[102,103]

The neurophysiologic effects of ethanol are modified by duration and intensity of exercise, food consumption, and applied cold stress.[81] Aging increases sensitivity to the hypothermic actions of ethanol in some primate experiments. Chronic ingestion yields tolerance to its hypothermic effects, and rebound hyperthermia may be seen during withdrawal. Conditions associated with ethanol ingestion that adversely affect heat balance include immobility and hypoglycemia.

Inhibited hepatic gluconeogenesis coexists with malnutrition. Hypothermic alcoholic ketoacidosis is reported.[312] Intravenous thiamine is diagnostic and therapeutic for Wernicke's encephalopathy, another cause of reversible hypothermia. The acute triad of global confusion, ophthalmoplegia, and truncal ataxia is often masked by hypothermia, and temperature depression may persist for weeks.

Impaired Thermoregulation

Various conditions that impair thermoregulation can be considered as having central, peripheral, metabolic, and pharmacologic or toxicologic effects.

Central

Central conditions may directly affect hypothalamic function and mediate vasodilation. Traumatic lesions include skull fractures, especially basilar, and intracerebral hemorrhages, most commonly chronic subdural hematomas. Pathologic lesions include neoplasms, congenital anomalies, and Parkinson's disease. Patients with Parkinson's disease or Alzheimer's disease, because of global neurologic impairment, are particularly at behavioral risk. Finally, cerebellar lesions also impede heat production because of inefficient choreiform shivering.

Hypothermia can occur with Reye's syndrome. In Hodgkin's disease, hypothermia is seen only in previously febrile patients with advanced disease, independent of cell type. This seems to be a disease-associated functional disorder of thermoregulation, similar to that seen in anorexia nervosa. Centrally induced hypothermia is completely antagonized with thyrotropin-releasing hormone.

Peripheral

Peripheral thermoregulation fails after acute spinal cord transection. Victims are functionally poikilothermic as soon as peripheral vasoconstriction is extinguished.[211] Other peripheral impediments to thermostability include neuropathies and diabetes mellitus. Hypothermia is more common in older adult diabetics than in the general population, even after excluding patients with diabetic metabolic emergencies. The common denominator in metabolic derangements may be abnormal plasma osmolality that interferes with hypothalamic function. Similar causes of hypothermia include hypoglycemia, diabetic ketoacidosis, and uremia. Remarkably, the pH was 6.67 in one hypothermic survivor with lactic acidosis, and 6.41 in another.[219]

Pharmacologic or Toxicologic

Numerous medications and toxins in therapeutic or toxic doses impair centrally mediated thermoregulation and vasoconstriction.[151,235,321] The usual offenders are barbiturates, benzodiazepines, phenothiazines, and the tricyclic antidepressants. Reduced core temperature may be a prodrome of lithium poisoning. Organophosphates, narcotics, glutethimide, bromocriptine, erythromycin, clonidine, fluphenazine, bethanechol, atropine, acetaminophen, and carbon monoxide all cause hypothermia. In experimental studies, hypothermia after acute carbon monoxide poisoning is associated with increased mortality.

Recurrent Hypothermia

Recurrent and episodic hypothermias are widely reported. The recurrent variety is more common and is usually secondary to ethanol abuse, with one person having survived 12 episodes.[50] Severe, recurrent presentations are also caused by self-poisoning and anorexia nervosa.

Persons with episodic hypothermia can be divided into two groups, albeit with significant overlap, as follows:

Group 1: Diaphoretic episodes precede the temperature decline, which lasts several hours, and this group includes those

with hypothalamic lesions and agenesis of the corpus callosum (Shapiro's syndrome) and persons with spontaneous periodic hyperthermia. Resultant hyperhidrosis and hypothermia are successfully treated with clonidine, a centrally acting alpha adrenergic agonist. The hypothermia of corpus callosum agenesis is also seen with hypercalcemia and status epilepticus; because no hypothermia results from experimental sectioning of the corpus callosum, associated lesions, including lipomas, probably cause thermoinstability. Spontaneous periodic hyperthermia may reflect a diencephalic autonomic seizure disorder and can accompany paroxysmal hypertension. Vasomotor and thermoregulatory mechanisms are successfully treated with anticonvulsants. Florid psychiatric symptoms often mask these intermittent hypothermic episodes.

Group 2: The second group consists of persons who remain cold for days to weeks, rather than hours. These people have more seizure disorders, and the central hypothalamic thermostat is set abnormally low.

Patients with intermittent hypothermia usually show some characteristics of both groups.[198] Circadian rhythm disturbances are also seen in persons with neurologic disorders who have chronic hypothermia.

Predisposing Infections or Conditions

Among the infestations and infections that not only elevate but often depress core temperature are septicemia, pneumonia, peritonitis, meningitis, encephalitis, bacterial endocarditis, typhoid, miliary tuberculosis, syphilis, brucellosis, and trypanosomiasis.[188] Other diseases, in addition to cerebrovascular and cardiopulmonary disorders, that produce secondary hypothermia include systemic lupus erythematosus, carcinomatosis, pancreatitis, and multiple sclerosis. Hypothalamic demyelination may explain the episodic hypothermia observed in some patients with multiple sclerosis.

Hypothermia can also result from low cardiac output after a major myocardial infarction, which can be reversed by intraaortic balloon counterpulsation. Finally, causes include vascular insufficiency, giant cell arteritis, uremia, sickle cell anemia, Paget's disease, sarcoidosis, and sudden infant death syndrome. Magnesium sulfate infusion during preterm labor can produce hypothermia with fetal and maternal bradycardia, and hypothyroidism can be manifested as hypothermia after preeclampsia (see Box 5-2).

Trauma

Hypothermia protects the brain from ischemia but can result in arrhythmias, acidosis, and coagulopathies, and it can extract a high metabolic cost during rewarming.[84] Hypothermia hinders the protective physiologic responses to acute trauma and affects the pharmacologic and therapeutic maneuvers necessary to treat the injuries.[15,23]

An inverse relationship usually exists between the injury severity score and the core temperature of traumatized patients on arrival in the emergency department (ED). This observation does not settle whether hypothermia is just another risk factor for increased mortality or reflects the fact that the most severely injured patients are in hemorrhagic shock.[118]

One study assesses the impact of hypothermia as an independent variable during resuscitation from major trauma.[84] Patients not aggressively rewarmed with continuous arteriovenous rewarming (CAVR) had increased fluid requirements, increased lactate levels, and increased acute mortality.

Of the clinical entities associated with hypothermia, traumatic conditions causing hypotension and hypovolemia most dramatically jeopardize thermostability. Hypothermia is often obscured by obvious hemorrhaging and injuries. On the other hand, traumatic neurologic deficits, including paresis and areflexia, can be misattributed to hypothermia. In trauma patients requiring surgery, the mean temperature loss was greater in the ED than in the operating room.[105] Thermal insults are often added during a trauma resuscitation. The patient is completely exposed for examination, and resuscitative procedures cause further heat loss.

In a study stratifying subjects with the anatomic Injury Severity Score (ISS), hypothermic patients had a higher mortality rate than similarly injured patients who remained normothermic.[150] Another study did not corroborate this finding,[278] but those investigators stratified using trauma revised injury severity score (TRISS) methodology, which is probably less valid during hypothermia because its physiologic components overestimate injury severity. To illustrate this point, some component of hypotension is normal for a given degree of hypothermia.

Various adverse physiologic events accompany hypothermia with trauma. Decreased skin and core temperatures without compensatory shivering thermogenesis are reported in patients with major trauma as defined by the ISS.

Hypothermia directly causes coagulopathies in trauma patients through at least three avenues (see Coagulation, earlier).[28] The cascade of enzymatic reactions is impaired and plasma fibrinolytic activity is enhanced, producing a clinical presentation similar to that of DIC. Also, platelets are poorly functional and become sequestered.

Hypothermia protects only when induced before shock occurs. This reduces ATP utilization while ATP stores are still normal, as during elective surgery. ATP stores in traumatized patients are already depleted. Hypothermia worsens the effects of endotoxins on clotting time in vitro and may synergistically exacerbate the coagulopathy seen in trauma.[77] The average temperature of 123 initially normothermic trauma patients in whom lethal coagulopathies developed was 31.2°C (88.2°F). In another study, postinjury life-threatening coagulopathy in the seriously injured who require massive transfusion was predicted by persistent hypothermia and progressive metabolic acidosis.[44]

The appropriate target core temperature for a hypothermic patient with an isolated severe head injury may be 32° to 33°C (89.6° to 91.4°F). This target temperature balances neuroprotection against the adverse hematologic and physiologic consequences of hypothermia.[16,202,203] (See Cerebral Resuscitation, later.)

PRESENTATION

The patient's history may suggest hypothermia.[28] Diagnosis becomes fairly simple when exposure is obvious, as with avalanche victims. Subtle presentations, however, predominate in urban settings. Victims often complain only of vague symptoms, including hunger, nausea, fatigue, and dizziness. Predisposing underlying illness or ethanol ingestion is also common, as are major trauma, immersion, overdose, cerebrovascular accidents, and psychiatric emergencies (Box 5-3).

During the head, eye, ear, nose, and throat examination, abnormal findings can include decreased corneal reflexes, mydriasis, strabismus, flushing, erythropsia, facial edema, rhin-

Box 5-3. Signs of Hypothermia

HEAD, EYE, EAR, NOSE, THROAT
- Mydriasis
- Decreased corneal reflexes
- Extraocular muscle abnormalities
- Erythropsia
- Flushing
- Facial edema
- Epistaxis
- Rhinorrhea
- Strabismus

CARDIOVASCULAR
- Initial tachycardia
- Subsequent tachycardia
- Arrhythmias
- Decreased heart tones
- Hepatojugular reflux
- Jugular venous distention
- Hypotension
- Peripheral vasoconstriction

RESPIRATORY
- Initial tachypnea
- Adventitious sounds
- Bronchorrhea
- Progressive hypoventilation
- Apnea

GASTROINTESTINAL
- Ileus
- Constipation
- Abdominal distention or rigidity
- Poor rectal tone
- Gastric dilation in neonates or in adults with myxedema
- Vomiting

GENITOURINARY
- Anuria
- Polyuria
- Oliguria
- Testicular torsion

NEUROLOGIC
- Depressed level of consciousness
- Ataxia
- Dysarthria
- Amnesia
- Anesthesia
- Areflexia
- Poor suck reflex
- Hypoesthesia
- Antinociception
- Initial hyperreflexia
- Hyporeflexia
- Central pontine myelinolysis

PSYCHIATRIC
- Impaired judgment
- Perseveration
- Mood changes
- Peculiar "flat" affect
- Altered mental status
- Paradoxical undressing
- Neuroses
- Psychoses
- Suicide
- Organic brain syndrome
- Anorexia
- Depression
- Apathy
- Irritability

MUSCULOSKELETAL
- Increased muscle tone
- Shivering
- Rigidity or pseudo–rigor mortis
- Paravertebral spasm
- Opisthotonos
- Compartment syndrome

DERMATOLOGIC
- Erythema
- Pallor
- Cyanosis
- Icterus
- Scleral edema
- Ecchymosis
- Edema
- Pernio
- Frostnip
- Frostbite
- Panniculitis
- Cold urticaria
- Necrosis
- Gangrene

orrhea, and epistaxis. Mild hypothermia usually does not depress pupillary light reflexes.

Cardiovascular findings after initial tachycardia include bradyarrhythmias and hypotension. Heart sounds may be muffled and distant. Tachypnea, an early respiratory finding, is usually followed by progressive hypoventilation with bronchorrhea and adventitious sounds. Because the gastrointestinal tract is depressed, abdominal distention or rigidity, ileus, obstipation, and poor rectal tone are often present. Gastric dilation is common in neonates and myxedematous adults. Genitourinary output ranges from initial polyuria resulting from cold diuresis to anuria. Interestingly, the incidence of testicular torsion increases because of cremasteric contractions.

Diffuse neurologic abnormalities vary widely. Some persons can still converse at 32°C (89.6°F) and are normoreflexic. The level of consciousness generally declines proportionate to the degree of hypothermia. The presence of ataxia and dysarthria may mimic a cerebrovascular accident. Speed of reasoning

and memory registration are also impaired. Amnesia, antinociception, anesthesia, or hypesthesia can develop. Cranial nerve abnormalities are present after bulbar damage from central pontine myelinolysis. These extraocular muscle movement abnormalities, like extensor plantar responses, do not directly correlate with the degree of hypothermia.

Hyperreflexia predominates from 35° to 32.2°C (95° to 90°F) and is followed by hyporeflexia. The plantar response remains flexor until 26°C (78.8°F), when areflexia develops. The knee jerk is usually the last reflex to disappear and the first to reappear during rewarming. From 30° to 26°C (86° to 78.8°F), both contraction and relaxation phases of reflexes are prolonged equally. In myxedema, however, the relaxation phase of the ankle reflex is more prolonged than the contraction phase.[198] Most important, spinal cord and other central nervous system lesions may be obscured by depressive neurologic changes that normally accompany hypothermia.

Psychiatric presentations and suicide attempts associated with hypothermia are commonly misdiagnosed initially. Preexistent psychiatric disorders can blossom in the cold, even if they were stabilized in temperate climates.[25] Mental status alterations include anxiety, impaired judgment, perseveration, neurosis, and psychosis.[137] Leaders of expeditions can become moody, apathetic, uncooperative, and risk taking. Older adult patients often withdraw in confusion, become silent, and display lassitude and poor judgment. A peculiar flat affect is common, and psychomotor impairment can resemble an organic brain syndrome.

Early in hypothermia, simply losing effective use of the hands can be devastating. Appropriate behavior adapted to the cold, such as seeking a heat source, is often lacking. An extreme example is paradoxical undressing.[162] The clothing is removed in a preterminal effort to address impending thermoregulatory collapse, and many persons are mistakenly identified as sexual assault victims. This phenomenon is also seen in hypothermic children. Undressing may also occur due to Alzheimer's disease before wandering in the cold.[161]

Musculoskeletal posturing can extend to pseudo–rigor mortis. Preshivering muscle tone is increased before core temperature drops to 35°C (95°F), and muscular rigidity, paravertebral spasm, and even opisthotonos may occur. Extremity compartment syndromes often develop because of associated conditions causing prolonged compression and immobility, in addition to compartment hypertension seen during reperfusion of frostbitten extremities.

Dermatologic presentations of hypothermia include erythema, pallor, edema, and scleral edema. Cold urticaria, frostnip, frostbite, and gangrene should also cause the clinician to consider this diagnosis. Pernio is also observed with chronic myelomonocytic leukemia.

LABORATORY EVALUATION

Acid–Base Balance

The strategy for achieving and maintaining acid–base balance in hypothermia differs from that of normothermia.[58,113,135,159] After initial respiratory alkalosis from hyperventilation when a person first becomes chilled, a common underlying disturbance is mixed acidosis. The respiratory component of the acidosis is caused mainly by direct respiratory depression. In addition, as the temperature decreases, solubility of carbon dioxide in the blood increases. Further contributors to the metabolic component of this acidosis include impaired hepatic metabolism and acid excretion, lactate generation from shivering, and decreased tissue perfusion.[120] Nevertheless, reliable clinical prediction of the acid–base status in accidental hypothermia is not possible. In one series of 135 cases, 30% were acidotic and 25% alkalotic.[213]

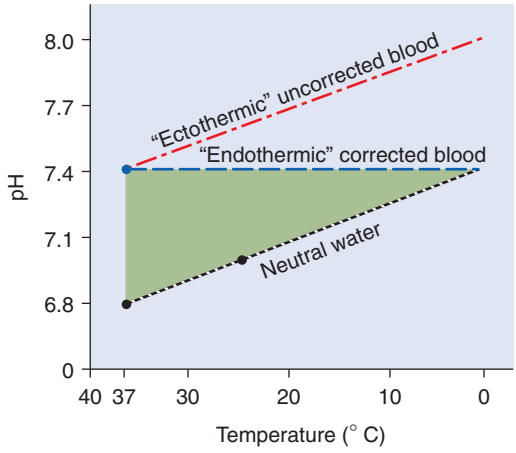


Figure 5-5. Neutrality is the pH of water at any given temperature. At 25°C (77°F), the neutral pH of water is 7.0; at 37°C (98.6°F), it is 6.8. An ectotherm's physiologic 0.6 pH offset from neutral water progressively diminishes if the arterial blood gases are temperature corrected. After the in vitro sample is warmed in the analyzer to 37°C, do not mathematically correct the reported values to reflect the in vivo temperature.

Circulatory changes also prevent adequate mobilization and delivery of organic acids to buffer systems. As in normothermia, mixed venous blood may best reflect acid–base status during resuscitation.[283] Despite flow changes in a moderately hypothermic canine model, a significant correlation persisted between arterial and mixed venous pH. The arteriovenous change in pH is 0.03 to 0.04 pH unit.

The buffering capacity of cold blood is also markedly impaired. In normothermia, when the $Paco_2$ increases 10 mm Hg, a decrease in pH of 0.08 unit occurs. At 28°C (82.4°F), the decrease in pH doubles to 0.16.

The accepted earlier assumption was that 7.42 was the ideal "corrected" patient pH at all temperatures and that therapy should be directed at maintenance of the corrected arterial pH at 7.42.[311] A better intracellular pH reference is electrochemical neutrality, at which pH equals pOH. Because the neutral point of water at 37°C (98.6°F) is pH 6.8, Rahn[240] has hypothesized that this normal 0.6 unit pH offset in body fluids should be maintained at all temperatures. Because the neutral pH rises with cooling, so should blood pH (Fig. 5-5).[245]

Relative alkalinity of tissues makes physiologic sense. Intracellular electrochemical neutrality ensures optimal function of the enzyme systems and transport proteins at all temperatures and allows excretion of the neutral intracellular waste product urea.[10]

Depressed metabolism and carbon dioxide generation are the physiologic responses to temperature depression, because each temperature has its associated metabolic rate. Ventilation is intrinsically adjusted to maintain a net charge on the defended

parameter, the peptide-linked histidine imidazole buffering system.

One homeostatic approach to maintain a steady pH is to keep the bicarbonate content constant. This is achievable only if the total blood carbon dioxide content does not change. Because carbon dioxide solubility increases with temperature depression, alveolar ventilation must increase to compensate by lowering the $Paco_2$. Active ectotherms exhibit this respiratory adaptation and do not depress their respiratory minute volume when cold. This response, termed the *ectothermic,* or *alpha-stat, strategy,* allows them to maintain total bicarbonate and carbon dioxide content while increasing pH.[232] Hibernating mammals are far more acidic due to respiratory acidosis and use an acid–base strategy that suppresses metabolism, termed the *endothermic,* or *pH-stat, strategy.*

The ideal acid–base strategy continues to be debated.[124,159,251] Some of the surgical literature about induced hypothermia, which clearly differs from accidental hypothermia, suggests that the alpha-stat approach improves neurologic outcome and also both cellular and myocardial function.[13,58,159,176,245,283] On the other hand, various animal models suggest that the pH-stat strategy may be better.[65]

In summary, it seems that accidental hypothermia in a human should not be considered a protective form of hibernation. The ectothermic (alpha-stat) approach appears to ensure adequate alveolar ventilation and acid–base balance at any temperature when the uncorrected pH is 7.4 and the uncorrected $Paco_2$ is 40 mm Hg.

Hematologic Evaluation

The severity of blood loss is easily underestimated. The hematocrit value increases because of a decline in plasma volume and a 2% increase per 1° C (1.8° F) fall in temperature. In addition, total RBC mass might already be low because of preexisting anemia, malnutrition, leukemia, uremia, or neoplasm.

The white blood cell count is frequently normal or low, even if sepsis is present. As a result, systemic leukopenia does not imply absence of infection, especially if the patient is at either age extreme, is debilitated, is intoxicated, is myxedematous, or has secondary hypothermia.[188] The leukocyte count also drops during hypothermia because of direct bone marrow depression and hepatic, splenic, and splanchnic sequestration.

Serum electrolyte levels must be continuously monitored and rechecked during warming. There are no safe predictors of electrolyte values.[112] Serum electrolytes fluctuate with temperature, duration of exposure, and rewarming technique selected. Both membrane permeability and sodium–potassium pump efficiency also change with temperature. Isolated temperature depression per se has no consistent effect on sodium and chloride levels until well below 25° C (77° F). Plasma electrolyte levels are also affected by ongoing fluid shifts, prehydration, rehydration, and endocrine or gastrointestinal dysfunction.

The plasma potassium level is independent of temperature. Empirical potassium supplementation during hypothermia often results in normothermic toxicity. From a clinical perspective, hypokalemia occurs as potassium moves into the musculature and not simply out of the body through kaliuresis. The physiologically illogical discrepancy of decreasing potassium level with decreasing pH results from greater intracellular than extracellular pH changes. Hypokalemia is much more common in prolonged or chronically induced hypothermia.

Systemic potassium deficiencies can also be exacerbated by prior diuretic therapy, alcoholism, diabetic ketoacidosis, hypopituitarism, and inappropriate antidiuretic hormone secretion. Hypokalemic digitalis sensitivity can be masked by hypothermia, and gradual correction of persistent and severe hypokalemia during rewarming is necessary for optimal cardiac and gastrointestinal function.

When hyperkalemia is identified, the physician should search for other causes of metabolic acidosis, crush injury or rhabdomyolysis, renal failure, postsubmersion hemolysis, or hypoaldosteronism. Temperature depression can increase hyperkalemic cardiac toxicity. An important caveat is that the well-known diagnostic ECG changes are often obscured by hypothermia, and VF can occur with serum potassium levels of less than 7 mEq/L.

Hypothermia has no consistent effect on magnesium or calcium levels. Severe hypophosphatemia can occur during treatment of profound hypothermia. Although increases in serum enzyme levels are not seen in mild experimental hypothermia, numerous serum enzymes are elevated when diffuse intracellular structural damage occurs in severe accidental hypothermia. Creatine kinase (CK) levels over 200,000 IU are observed, and rhabdomyolysis is often present in these instances.[50]

Inhibition of cellular membrane transport decreases glucose utilization. In addition, insulin release and activity are markedly reduced below 30° C (86° F). Because target cells are insulin resistant, hyperglycemia is commonly seen initially. Markedly elevated glucose levels often correlate with hyperamylasemia and increased cortisol secretion.[312]

Acute hypothermia initially elevates the serum glucose level through catecholamine-induced glycogenolysis. Chronic exposure after exhaustion and glycogen depletion leads to hypoglycemia. The symptoms often resemble those of hypothermia. Cold-induced renal glycosuria is common and does not imply normoglycemia or hyperglycemia. When hypoglycemia and central neuroglycopenia are present, correction improves the level of consciousness only to that expected for the current core temperature. Cholesterol and triglyceride levels are also often below normal.

Hyperglycemia that persists during and after rewarming should suggest diabetic ketoacidosis or hemorrhagic pancreatitis. Insulin is ineffective until the core temperature is well above 30° to 32° C (86° to 89.6° F) and therefore should be withheld to avoid iatrogenic hypoglycemia after rewarming. Although prior renal disease should be a consideration, blood urea nitrogen (BUN) and creatinine concentrations are often elevated because of decreased nitrogenous waste clearance by the cold diuresis. Because of ongoing fluid shifts, BUN is a poor reflector of circulatory volume status.[87]

The relationship between primary accidental hypothermia and hyperamylasemia appears to correlate with the severity of temperature depression, but preexisting or hypothermia-induced pancreatitis is present in up to 50% of patients in some urban series. Because the abdominal examination is frequently unreliable, amylase and lipase levels should be measured, except in minor cases. Ischemic pancreatitis is attributable to microcirculatory collapse in hypothermia. Decreased pancreatic blood flow activates many proteolytic enzymes. The extent of hyperamylasemia can correlate with mortality rates. Hypothermia with hypothalamic astrocytoma and pancreatitis also occurs.

TREATMENT

Four decisive factors should be considered when assessing nonperfusing, severely hypothermic victims at the site. Rescuers should reconsider the decision to resuscitate if there is evidence of asphyxia or lethal injuries. A rigid thorax precludes cardiopulmonary resuscitation (CPR), and a probable field core temperature below 10° to 12° C is ominous.

The combination of cold and exhaustion is a common cause of hypothermia in the field.[214] The individual's cold tolerance depends on temperature, wind, clothing worn, and wetness. It does not take an extremely cold temperature to produce hypothermia after energy depletion. As mental function decreases, the victim cannot respond to the rising threat. Judgment is impaired, and the victim seldom takes necessary precautions to prevent further disaster.[21]

Field presentation of victims who are awake covers a wide spectrum. Some persons are obtunded but conscious. Victims may or may not be shivering and often have a distant gaze and slurred speech.[88]

The type of power available in the transport vehicle determines the options for active rewarming. These include forced-air warming devices, heated IV fluids, and resistive heat.[90,93,99]

Comatose victims require careful handling because they are extremely likely to develop VF and asystole with rough handling (Fig. 5-6). Gurneys should be carried or rolled slowly to avoid jostling, and "code 3, full lights and sirens" transport should be avoided if victims are perfusing adequately.

It is often impossible for rescuers to separate primary from secondary hypothermia when an unwitnessed cardiac arrest victim is found in a frigid environment. Because patients with cold, stiff, and cyanotic primary hypothermia and fixed and dilated pupils have been "reanimated," the treatment dictum for prehospital personnel remains, "No one is dead until warm and dead."[7] A succinct summary of prehospital care of the hypothermic patient is rescue, examine, insulate, and transport.

The initial rescuer and first responder often encounter obstacles to preventing further heat loss.[215,307] In certain imposing geographic settings, treatment protocols are helpful to standardize treatment while tacitly acknowledging that available health care facilities may offer limited expertise and equipment. All treatment recommendations must be adapted to local rescue systems and facilities.[67,253] Aeromedical transport is often ideal in these circumstances.[316] In difficult environments, proper protocols with rehearsal and critique of mass-casualty plans in the cold are far more important than similar adjuncts in flat and consistently sunny terrain.

The history obtained at the scene helps determine optimal treatment. The pertinent past medical history regarding prior cardiopulmonary, endocrinologic, and neurologic conditions is particularly helpful. The circumstances of discovery, duration of exposure, associated injuries or frostbite, and obvious predisposing conditions should be recorded, as should the Glasgow Coma Scale score, although none of these is predictive. No prognostic neurologic scale during hypothermia is valid, but trends are often useful.

Accurate field measurement of core temperature is difficult. The International Commission for Mountain Emergency Medicine recommends tympanic or esophageal field measurements.[253]

Prolonged field treatment should be avoided whenever possible, although the rescuer must attempt to prevent further heat loss (Box 5-4). The rescuer should anticipate the presence of an

Box 5-4. Preparing Hypothermic Patients for Transport

1. The patient must be dry. Gently remove or cut off wet clothing and replace it with dry clothing or a dry insulation system. Keep the patient horizontal, and do not allow exertion or massage of the extremities.
2. Stabilize injuries (i.e., the spine; place fractures in the correct anatomic position). Open wounds should be covered before packaging.
3. Initiate intravenous infusions (IVs) if feasible; bags can be placed under the patient's buttocks or in a compressor system. Administer a fluid challenge.
4. Active rewarming should be limited to heated inhalation and truncal heat. Insulate hot water bottles in stockings or mittens and then place them in the patient's axillae and groin.
5. The patient should be wrapped. Begin building the wrap by placing a large plastic sheet on the available surface (floor, ground), and on it place an insulated sleeping pad. A layer of blankets, a sleeping bag, or bubble wrap insulating material is laid over the sleeping pad. The patient is then placed on the insulation. Heating bottles are put in place along with IVs, and the entire package is wrapped layer over layer, with the plastic as the final closure. The patient's face should be partially covered, but a tunnel should be created to allow access for breathing and monitoring.

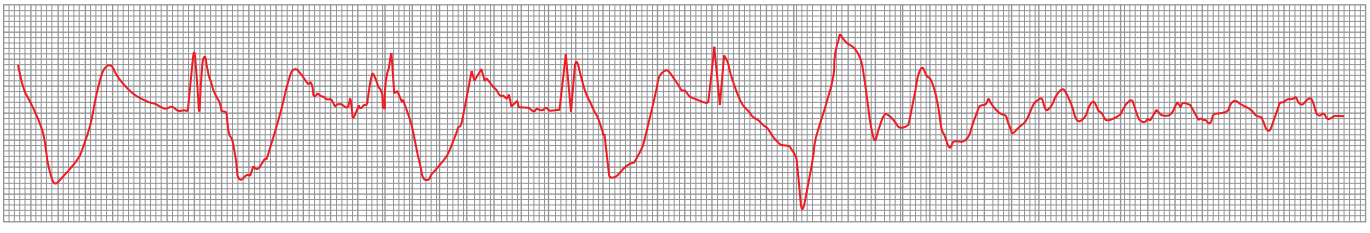


Figure 5-6. In this patient, ventricular fibrillation developed during a code 3 transport by emergency medical services to the emergency department. Note the pronounced J wave after the QRS complex.

irritable myocardium, hypovolemia, and a large temperature gradient between the periphery and the core.

The crux of the prehospital quandary surrounds the safety of providing prehospital heat. On one hand, the original "metabolic icebox" concept[214,215] implies that the risk of inducing a nonperfusing rhythm in a hostile environment justifies simple stabilization of the core temperature. On the other hand, the warmer the heart is, the better. The core temperature could decrease beyond reversal. Operant factors impacting this decision include core temperature, fluid status, predisposing factors, extent of frostbite, length and type of exposure, available expertise and position of transport, and type of active rewarming.

The rescuer should stabilize injuries, protect the spine, splint fractures, and cover open wounds. Rescuer safety factors commonly include unstable snow or ice and falling rocks.

Passive external rewarming with dry insulating materials minimizes conductive, convective, evaporative, and radiant heat losses. Remove any wet clothing from awake victims and insulate them with sleeping bags, insulated pads, bubble wrap, metallicized or regular blankets, or even newspaper. One device, the Hypothermic Stabilizer Bag (available at www.emssupply.com), has a nylon shell and a polyester-pile liner to wick moisture. In a survey of common field rewarming methods used by mountain rescue teams, the most common techniques include chemical pads, sleeping bags, body-to-body contact, and hot water bottles.[110]

If extrication will be delayed, give mildly hypothermic patients warm, sweet drinks, warm gelatin (Jell-O), Tang, juice, tea, or cocoa, because carbohydrates fuel shivering. Avoid heavily caffeinated drinks. Because a significant diuresis is usually associated with the cooling process, the victim's fluid balance must be assessed. Once mildly hypothermic victims are well hydrated, they can be walked out to safety.

Handle severely hypothermic victims gently, immobilizing them to prevent exertion. Although exercise can rewarm a person more rapidly than shivering, it also markedly increases afterdrop.[87,92] Because of autonomic dysfunction, victims should be kept in a horizontal position whenever possible to minimize orthostatic hypotension. Vigorously massaging cold extremities is also contraindicated, because skin rubbing, like ethanol, suppresses shivering thermogenesis and increases cutaneous vasodilation.

Field management of the comatose victim first involves ventilation to raise oxygen saturation. Rescue breathing may be difficult because of chest stiffness and significant resistance to diaphragmatic motion. Forced ventilation increases oxygenation, which helps stabilize the heart electrically. The single greatest factor in maintaining perfusion is oxygenation provided during severe hypothermia.[88]

During a storm, prolonged field rewarming may be the only viable option until meteorologic conditions become more favorable for land or, preferably, aeromedical evacuation.[316] Many prehospital medications freeze in solution. If this occurs, their pharmacologic activity after thawing is indeterminate (see Appendix at end of this book).

The rescuer should administer an IV fluid challenge with 250 to 500 mL heated (37° to 41°C [98.6° to 105.8°F]) 5% dextrose in normal saline solution. Empiric 50% dextrose is indicated only in select cases. The safety of several agents, such as naloxone or flumazenil, used in the field is unknown. Concerns regarding these agents prior to warming include precipitation of acute withdrawal, seizures, and markedly increased oxygen utilization.

Most patients are volume depleted. Nevertheless, iatrogenic volume overload should be avoided, because myocardial contractility is impaired. Improvisation during transport is often helpful. For example, a plastic IV container can be placed under the patient's back, shoulders, or buttocks to add warmth and infusion pressure. Taping heat-producing packets to IV bags is another option. These heating agents may be chemical packets or phase-change crystals, which produce heat for up to several hours. IV fluid compressors are bulb-inflating cuffs that surround IV pouches to maintain flow. The Israeli army has a spring steel compressor system for IV bags. Portable IV fluid heaters are commercially available. The devices fit in-line and are DC powered. Commercial heaters are available that are powered by a Physio-Control battery or from a DC converter plugged into an AC outlet.

Peripheral vessels may be difficult to locate, and IV lines are hard to maintain during transport. Ideally, IV fluids should be warmed to body temperature or slightly higher, but total body warming is not accomplished with IV fluids in the field. Using intraosseous infusions may provide a reasonable pathway for fluid replacement in the field when peripheral vessels have collapsed and cannot be entered.

The methods selected to stabilize core temperature should be tailored to the severity of hypothermia and the field circumstances. Gently removing or cutting off wet clothing while the patient remains prone may be the best option to limit heat loss and prevent orthostasis. Passive external rewarming with waterproof insulation suffices for mild chronic hypothermia. In their series of patients requiring cardiopulmonary bypass (CPB), Walpoth and colleagues[303,304] "maintained" hypothermia during transport. Whether supplemental active field rewarming should be initiated en route to the ED, and in which subsets of patients, is not definitively established. Intentionally maintaining hypothermia during transport seems wise only for patients with an isolated closed-head injury. For long helicopter or ambulance transports, the ideal ambient temperature in the vehicle is unclear. Rescue personnel comfort is important to maximize performance, and it is unlikely that heating the interior above 25°C will accelerate the rate of rewarming or induce vasodilation.

"Field rewarming" is a misnomer, because adding much heat to a hypothermic patient in the field is difficult.[71] Warmed IV solutions, heated sarongs, or heated humidified oxygen can provide only a small amount of heat input. Hot water bottles or heat packs can be placed in the axillae, in the groin, and around the neck. Casualty evacuation bags are available in many models and designs. Some have more insulation than others, and some have specialized zippers and openings that allow access to the victim during transport. Most bags are windproof and waterproof. Experimentally, convective air warming is more effective than resistive heating[139] and might be a viable option in some transport situations.

Inhalation therapy is safe for active rewarming of victims with profound hypothermia in the field.[112] It helps prevent respiratory heat loss, which represents an important percentage of heat production when the core temperature is below 32°C (89.6°F). Technical difficulties have been experienced while using inhalation rewarming devices in volunteers, however, which suggests the importance of proper instruction for the rescuer.[280] Under certain conditions, the impact of inhalation

therapy on the rate of rewarming may not be significant.[99] In shivering, mildly cold patients, the thermal advantage is minimal.[62,64,210]

One rewarming device weighs 3 kg, including oxygen tank, and consists of an oxygen cylinder, demand valve, 2-L reservoir bag, soda lime, and pediatric water canister. An in-line thermometer measures mean air temperature at the face mask. In the field, heat and moisture exchangers are practical, light, and inexpensive. They are, however, less efficient than are active humidifiers.[191,192]

The Res-Q-Air (RES-Q Products, Inc., Sooke, BC, Canada) is a lightweight, portable, noninvasive core-rewarming system that delivers heated humidified air or oxygen. It operates from any 12-volt DC power source. This system consists of a heating chamber connected via a corrugated hose to a one-way flow valve and an oronasal mask. The temperature is controlled by a transducer in the one-way flow valve that provides feedback to the electronic control circuits.

Although surface rewarming suppresses shivering, it may be the only option when the victim is isolated from medical care. Active external rewarming options include radiant heat, warmed objects placed on the patient, and body-to-body contact. Care must be exercised not to burn victims with hot objects, including commercially produced hot packs. Total body contact rewarming may carry some risk,[112] but logistical impediments limit the use of this method unless extrication will be significantly delayed.

A hydraulic sarong or vest, in which heated water is circulated via a hand pump, is also used occasionally.[110] Immersion rewarming is dangerous in the field because monitoring and resuscitation capabilities are limited.

The Norwegian Heatpac Personal Heater (Heat Pac, Oslo, Norway) can deliver 100 watts for 8 hours. The charcoal "vest" burns a briquette and has a favorable heat-to-weight ratio, unlike most rewarming devices.[42] This device uses a single D battery that must be inserted correctly to avoid a CO exhaust hazard. It is durable, lightweight (1 kg), efficient, and effective. It circulates hot air within a blanket or sleeping bag and provides a comfortable, warm, and dry environment for transporting victims.

Another option under development is a modified forced-air warming system for field use.[62,87,93] The Portable Rigid Forced-Air Cover is heated with a Bair Hugger 505 Heater/Blower (Augustine Medical, Inc., Eden Prairie, MN). It covers the patient's trunk and thighs and can adapt to various transport vehicle power sources. Experimentally, convective warming is more efficient than is resistive heating.[139]

Prehospital Life Support

A patent airway and the presence of respirations must be established (Fig. 5-7). The victim may appear apneic if respiratory minute ventilation is significantly depressed. A common error in tracheally intubated patients is overzealous assistance of ventilation, which can induce hypocapnic ventricular irritability. The indications for prehospital endotracheal intubation are identical to those under normothermic conditions. Appropriate ventilation with 100% oxygen may protect the heart during extrication and transport. Avoid overinflation of the tracheal cuff with frigid ambient air. As the victim warms, air in the cuff can expand and kink the tube. Careful protection and fixation of the tubing of the cuff port during extremely cold conditions are necessary to prevent breakage and cuff leak.

Palpation of peripheral pulses is often difficult in vasoconstricted and bradycardic patients. Apparent cardiovascular collapse may actually reflect cardiac output that is depressed just enough to meet minimal metabolic demands. The rescuer should auscultate and palpate for at least 1 minute to find pulses. Iatrogenic VF can easily result from chest compressions that were not indicated.

If a cardiac monitor is available, use maximal amplification to search for QRS complexes. If the victim is in VF, the rescuer should attempt defibrillation initially with 2 watt sec/kg up to 200 watt sec. The energy requirement for defibrillation does not increase in hypothermia. If the patient does not respond, defer further attempts, and assume that the victim must be rewarmed at least past 30°C (86°F) before further attempts.[5a] Do not perform defibrillation if electrical complexes are seen on the monitor. Most monitors and defibrillators have not been tested for operation at temperatures below 15.5°C (59.9°F). Standard monitor leads do not always stick well to cold skin, so benzoin may be useful. If this fails, needle electrodes may be necessary. Another option is to puncture the Gelfoam conventional monitor pad with a small-gauge injection needle.[308]

Unresponsive victims should be carefully assessed for a central pulse before assuming they have pulseless electrical activity (PEA). The lowest temperature at which mechanical reestablishment of cardiac activity has been successful is 20°C (68°F),[55] and defibrillation attempts rarely succeed below 30°C (86°F). If resuscitation in the field is unsuccessful, rewarming and CPR should be continued during transport to the ED.

Management in the Emergency Department

The history obtained from a hypothermic patient is often unreliable, so it is prudent to confirm hypothermia and monitor with continuous core temperature measurements. Diagnostic errors in the ED usually result from incomplete monitoring of vital signs. Doppler ultrasound may be necessary to locate a pulse and should be supported by continuous ECG monitoring. The physician should also address the requirements for resuscitation and initiate advanced life support when necessary (Box 5-5).

Temperature Measurement

Most hospitals no longer lack adequate equipment for accurate core temperature measurement.[223] Rectal measurements are most practical clinically for mild cases but may not reflect cardiac or brain temperatures.[119] An indwelling thermistor probe placed to a depth of 15 cm is fairly reliable unless adjacent to cold feces. Rectal temperature lags behind core temperature fluctuations and is also affected by lower extremity temperatures (Table 5-3).[313]

Esophageal temperature measurements are far preferable in severe cases and when airway protection is provided with endotracheal intubation. Esophageal temperature approximates cardiac temperature. Because the upper third of the esophagus is near the trachea, the probe may read falsely high during heated inhalation therapy if the probe is positioned too proximally. The probe should be placed 24 cm below the larynx. The greatest discordance between rectal and esophageal temperatures is noted during the transition phase between cooling and rewarming.

Tympanic temperature theoretically approximates hypothalamic temperature and can be accurately measured with ther-

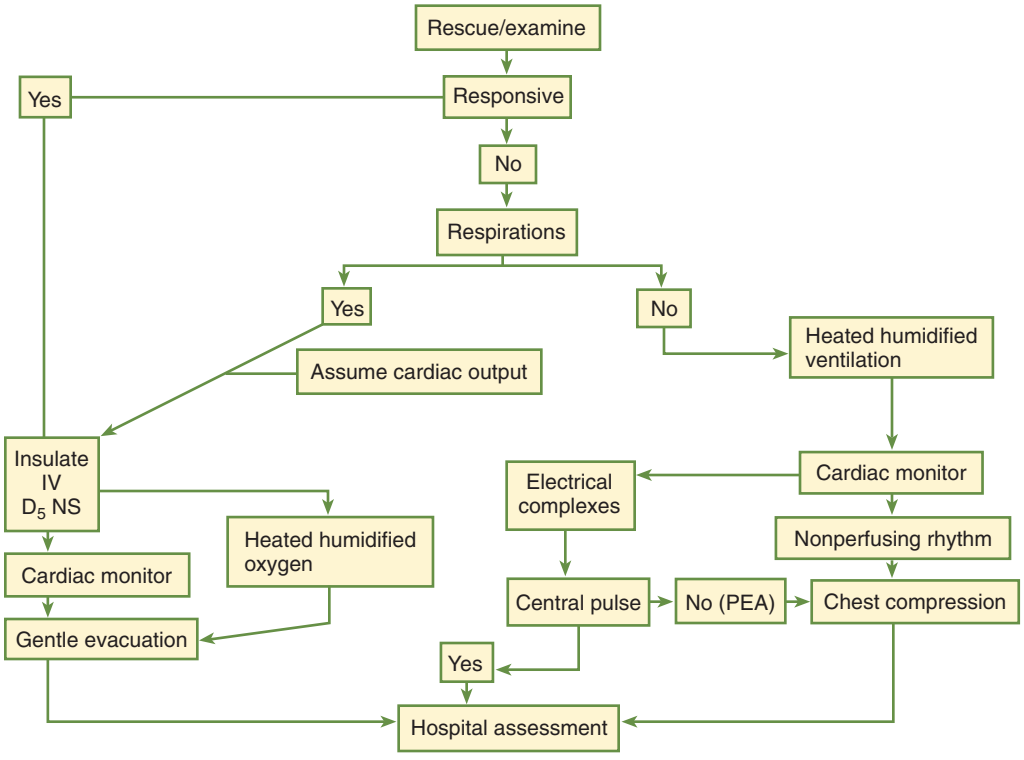


Figure 5-7. Prehospital life support. PEA, pulseless electrical activity.

Box 5-5. Resuscitation Requirements

Thermal Stabilization
- Conduction
- Convection
- Radiation
- Evaporation
- Respiration

Maintenance of Tissue Oxygenation
- Adequate circulation
- Adequate ventilation

Identification of Primary versus Secondary Hypothermia

Rewarming Options
- Passive external rewarming
- Active external rewarming
- Active core rewarming

mometers that are in contact with the tympanic membrane. Because these are impractical except in anesthetized patients, external auditory canal thermometers that measure infrared emission from the tympanic membrane have been developed. These are not actually "tympanic" thermometers, but this term is used extensively.[323] Unfortunately, the external auditory canal thermometers are not very sensitive.

Although bladder temperature measurement devices can be incorporated into the urinary drainage catheter, the readings are frequently unreliable. They are falsely elevated with peritoneal lavage and, more commonly, low as a result of cold-induced diuresis and crystalloid resuscitation.

Initial Stabilization

After core temperature measurement, all clothing should be gently removed or cut off with minimal patient manipulation.[231] Immediately insulate the patient with dry blankets. Apply a cardiac monitor, and insert intravenous catheters as needed. Arterial catheter insertion may help in managing selected profoundly hypothermic patients. Pulmonary artery catheters can precipitate cardiac arrhythmias and should be reserved for complex cases. Insertion of pulmonary artery catheters into cold vessels may perforate the pulmonary artery.[41] Central venous catheters should not be inserted into the right atrium, because this could precipitate arrhythmias.[268] A better option is temporary catheterization of the femoral vein.

The accuracy of pulse oximetry during conditions of poor perfusion is unclear.[167] In one study of hypothermic patients on CPB, the finger probes were inaccurate.[38] The probe may be made more reliable if a vasodilating cream is applied first.

In theory, combining pulse oximetry with near-infrared spectroscopy could provide tissue perfusion information during hypothermic vasoconstriction. More practically, an accurate core and peripheral muscle temperature would prove just as useful. The value of end-tidal carbon dioxide measurements to assess adequacy of tissue perfusion and tracheal tube placement is established at normal temperatures. Most of these devices, however, measure only the carbon dioxide content of dehumidified air and thus cannot be used during airway rewarming.

TABLE 5-3. Core Temperature Measurements

TYPE	ADVANTAGES	CONSIDERATIONS
Rectal	Convenient Continuous monitoring	Insert 15 cm Lags during transition from cooling to rewarming Falsely elevated with peritoneal lavage Falsely low if probe is in cold feces or when lower extremities are frozen
Esophageal	Convenient Continuous monitoring	Insert 24 cm below larynx Tracheal misplacement Aspiration Falsely elevated with heated inhalation
Tympanic	Approximates hypothalamic temperature via internal carotid artery	Probe: tympanic membrane perforation; canal hemorrhage Infrared: unreliable; cerumen effect
Bladder	Convenient Continuous monitoring	Unreliable Falsely elevated with peritoneal lavage Falsely low with cold diuresis

Laboratory evaluations to be considered, except in some cases of mild hypothermia, include blood sugar, arterial blood gases, complete blood cell count, electrolytes, BUN, creatinine, serum calcium, serum magnesium, serum amylase and lipase, PT, PTT, INR, platelet count, and fibrinogen level. A toxicologic screen should be considered if the level of consciousness does not correlate with the degree of hypothermia. Selective studies of thyroid function, cardiac isoenzyme levels, and serum cortisol levels are indicated.[52]

The indications for radiography should be liberalized from normothermia. Radiologic evaluation of poorly responsive patients must include cervical and other spine images to detect occult trauma. Chest radiographs may predict lung collapse during rewarming when cardiomegaly and redistribution of vascularity are already present. Abdominal imaging should be obtained when the physical examination is unreliable. Bowel sounds are usually diminished or absent in severe hypothermia, and rectus muscle rigidity is frequently present. Pneumoperitoneum, pancreatic calcifications, or hemoperitoneum may be noted. Small bowel dilation is associated with cold-induced mesenteric vascular occlusion, and colonic dilation is often present in conjunction with myxedema coma. The use of focused abdominal sonography may be helpful.[48]

Nasogastric tube insertion should be performed after endotracheal intubation in moderate or severe hypothermia, because gastric dilation and poor gastrointestinal motility are common. Indwelling bladder catheters with urine meters are needed to monitor urine output and the cold diuresis.

FLUID RESUSCITATION

Most fluid shifts are reversed by rewarming, and mild hypothermia usually requires only modest amounts of crystalloids. In more severe cases, volume shifts and elevated blood viscosity from hemoconcentration, lowered temperature, increased vascular permeability, and low flow state mandate aggressive fluid resuscitation. The viscosity of blood increases 2% per degree centigrade drop in temperature; therefore, hematocrit values over 50% are commonly seen. Low circulatory plasma volume is often coupled with elevated total plasma volume during rewarming. Hemodilution is usually not a problem and is seen only during massive crystalloid resuscitation of actively hemorrhaging patients.[50]

Most patients are significantly dehydrated, with free water depletion elevating serum sodium concentration and osmolality. During the descent into a hypothermic state, normal physiologic cues for thirst become inactive and access to water is often difficult. Because hypothermia results in natriuresis, saline depletion may be present. Further causes of sodium losses include prior diuretic therapy and gastrointestinal losses. Preexisting total body sodium excess is seen with congestive heart failure, cirrhosis, and nephrosis. In these cases, serum sodium and osmolality values are often normal. Rarely, serum sodium level is low because of free water excess. Other causes include myxedema, panhypopituitarism, and inappropriate antidiuretic hormone secretion.

Most adult patients with a core temperature below 32.2° C (90° F) should receive an initial fluid challenge with 250 to 500 mL of warmed 5% dextrose in normal saline solution. Theoretically, Ringer's lactate solution should be avoided because a cold liver cannot metabolize lactate. In severe cases, some clinicians favor a mixture of crystalloids and colloids.

The victim should be monitored for standard clinical signs of fluid overload, including rales, jugular venous distention, hepatojugular reflux, and S_3 cardiac gallop. Persistent cardiovascular instability often reflects inadequate intravascular volume. As mentioned, a properly placed central venous pressure catheter that does not enter and irritate the right atrium, such as a femoral line, has a role. Pulmonary wedge pressure measurements are not indicated. The need for RBC transfusions is determined by the corrected hematocrit; blood dilution with warmed infusate does not cause significant hemolysis.

In many cases, rapid volume expansion is critical. The circulatory volume is decreased, and peripheral vascular resistance is increased. In neonates, adequate fluid resuscitation markedly decreases mortality. Adults receiving hemodynamic monitoring show improvement of cardiovascular efficiency during crystalloid administration. VF immediately after rescue is attributed to both core temperature afterdrop and vascular imbalance in patients who are moved from a horizontal supine position.[193] The fluxing relationship between active vas-

Box 5-6. Rewarming Techniques

PASSIVE EXTERNAL
- Thermal stabilization

ACTIVE

External
- Radiant heat
- Hot water bottles
- Plumbed garments
- Electric heating pads and blankets
- Forced circulated hot air
- Immersion in warm water
- Negative-pressure rewarming

Core
- Inhalation rewarming
- Heated infusions
- Gastric and colonic lavage
- Mediastinal lavage
- Thoracic lavage
- Peritoneal lavage
- Diathermy
- Hemodialysis
- Venovenous extracorporeal blood rewarming
- Arteriovenous extracorporeal blood rewarming
- Cardiopulmonary bypass

Box 5-7. Indications for Active Rewarming

- Cardiovascular instability
- Moderate or severe hypothermia (<32.2° C [90° F]) (poikilothermia)
- Inadequate rate or failure to rewarm
- Endocrinologic insufficiency
- Traumatic or toxicologic peripheral vasodilation
- Secondary hypothermia impairing thermoregulation
- Identification of predisposing factors (see Box 5-2)

cular capacity and circulating fluid volume depends not only on the mechanism of cooling but also on the method of rewarming.

Hypothermic patients, particularly those with frostbite, are at high risk for extremity compartment syndromes and rhabdomyolysis. Pelvic fracture belt slings should be considered only to temporarily stabilize coexistent exsanguinating major pelvic fractures.

REWARMING OPTIONS

Passive External Rewarming

Because hypothermia is an extremely heterogeneous condition and evidence-based treatment guidelines do not exist, rigid treatment protocols are ill advised.[253,257,324] A versatile approach to rewarming can be developed after careful consideration of the observations from animal experiments, human experiments on mild hypothermia, and various clinical reports (Box 5-6).[106,107] Treatment should be predicted on the presenting pathophysiology and the available resources and expertise.

The initial key treatment decision is whether to use active or passive rewarming (Box 5-7). Noninvasive passive external rewarming (PER) is ideal for most previously healthy patients with mild hypothermia. The patient is covered with dry insulating materials in a warm environment to minimize the normal mechanisms of heat loss. When the wind is blocked, less heat escapes via radiation, convection, and conduction. Conditions with higher ambient humidity slightly limit respiratory heat loss.

Aluminized body covers also reduce heat loss.[72,129] Nevertheless, endogenous thermogenesis must generate an acceptable rate of rewarming for PER to be effective.[224] Humans are functionally poikilothermic below 30° C (86° F), and metabolic heat production is less than 50% of normal below 28° C (82.4° F). Shivering thermogenesis is also extinguished below 32° C (89.6° F). This thermoregulatory neuromuscular response to cold normally increases heat production from 250 to 1000 kcal/hr unless glycogen is depleted during cooling.

Older adult patients in whom mild hypothermia develops gradually are less acceptable candidates for PER. When rewarming times are markedly prolonged (over 12 hours), complications tend to increase.

Patients who are centrally hypovolemic, glycogen depleted, and without normal cardiovascular responses should be stabilized and rewarmed at a conservative rate. In a multicenter survey, the rewarming rates for older adults in the first (0.75° C), second (1.17° C), and third (1.26° C) hours far exceeded 0.5° C per hour, with no increase in mortality rate (Fig. 5-8).[53]

Active Rewarming

Active rewarming, which is the direct transfer of exogenous heat to a patient, is usually required with temperatures below 32° C (89.6° F).[182] Rapid identification of any impediment to normal thermoregulation, such as cardiovascular instability or endocrinologic insufficiency, is essential.[183] Intrinsic thermogenesis may also be insufficient after traumatic spinal cord transection or pharmacologically induced peripheral vasodilation. Some patient populations generally require active rewarming.[56,172] For example, aggressive rewarming of infants minimizes energy expenditure and decreases mortality. In these circumstances, vigorous monitoring for respiratory, hematologic, metabolic, and infectious complications is essential.[299]

When active rewarming is needed, heat can be delivered externally or to the core. Active external rewarming (AER) techniques deliver heat directly to the skin. Examples include forced-air rewarming, immersion, arteriovenous anastomosis rewarming, plumbed garments, hot water bottles, heating pads and blankets, and radiant heat sources.

During rewarming of hypothermic patients, there are metabolic pH and inflammatory interleukin fluxes.[207,294] Cytokine production may be activated by accidental hypothermia.[1]

Active External Rewarming

The interpretation of survival rates with AER is affected by various risk factors and patient selection criteria.[56,273] Some experimental and clinical reports link AER with peripheral vasodilation, hypotension, and core temperature afterdrop, but previously healthy, young, and acutely hypothermic victims are usually safe candidates for AER.[27] Heat application confined to

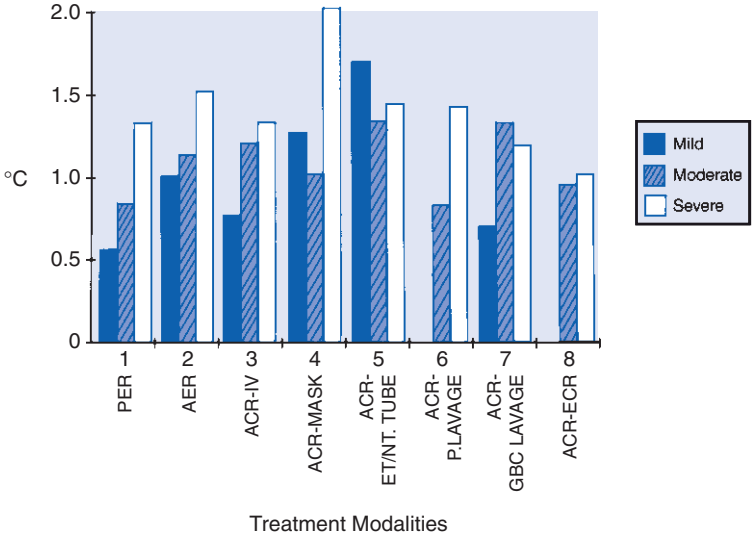


Figure 5-8. First-hour rewarming rates from a large multicenter survey. ACR, active core rewarming; AER, active external rewarming; ET, endotracheal tube; GBC, gastric-bladder-colon; IV, intravenous; NT, nasotracheal tube; P, peritoneal; PER, passive external rewarming. (Data from Danzl DF, Pozos RS, Auerbach PS, et al: Ann Emerg Med 16:1042–1055, 1987, with permission.)

the thorax may mitigate many of the physiologic concerns pertaining to the depressed cardiovascular and metabolic systems, which are unable to meet accelerated peripheral demands.[291] Combining truncal AER with active core rewarming may further avert many potential side effects.[63]

Forced-Air Surface Rewarming

Forced-air surface warming systems efficiently transfer heat.[8,90,141,173] The Bair Hugger (Augustine Medical, Inc.) uses hot forced air circulated through a blanket. The air exits apertures on the patient side of the cover, permitting the convective transfer of heat.[206,263] In one study that rewarmed accidental hypothermia victims in the ED, rewarming shock and core temperature afterdrop were not noted[277] with the use of heated inhalation and warmed IV fluids. A group also treated with a convective cover inflated at 43° C (109.4° F) rewarmed 1° C (1.8° F) per hour faster than a group covered with a cotton blanket (1.4° C [2.5° F] per hour). Experimentally, resistive external heating is more effective than passive metallic-foil insulation.[108]

A study of full-body forced-air warming compared a commercially available convective blanket with simple air delivery beneath bed sheets.[160] Directed 38° C (100.4° F) warm air under the sheets warmed standardized thermal bodies containing water very efficiently. Commercially available convective air rewarming devices (Warm Touch, Mallinckrodt, whole-body blanket) are also effective.

The use of forced-air surface warming systems is most practical in the ED.[170,185] Although these devices decrease shivering thermogenesis, afterdrop is minimized and heat transfer can be significant. Thermal injury to poorly perfused, vasoconstricted skin using some of the other external heat application techniques is a hazard in both adults and children.[110,165] In particular, avoid resistance-heat electric blankets on which a patient lies, because vasoconstricted capillaries are compressed and burns occur easily.

Immersion

Immersion in a 40° C (104° F) circulating bath presents difficulties in monitoring, resuscitation, treatment of injuries, and maintenance of extremity vasoconstriction to prevent core temperature afterdrop. In normothermic men with coronary artery disease, the cardiovascular stress and arrhythmogenic response to immersion in a hot tub are mild, less than those induced by exercise.[3] In contrast, placing the hands and feet in warm water theoretically opens arteriovenous shunts and accelerates rewarming in acute hypothermia. The attraction to Scandinavian palmar heat packs may reflect this physiology.

Arteriovenous Anastomosis Rewarming

The original description of this noninvasive AER technique was by Vangaard[295] in 1979. Exogenous heat is provided by immersion of the lower parts of the extremities (hands, forearms, feet, calves) in 44° to 45° C (111.2° to 113° F) water. The heat opens arteriovenous anastomoses (AVAs). These organs are 1 mm below the epidermal surface in the digits.[14,62,212] Countercurrent heat loss is minimized because the superficial veins are not close to the arterial tree.

To be efficacious, the cutaneous heat exchange area must include the lower legs and forearms, and the water must be 44° to 45° C. Advantages with AVA rewarming include patient comfort and decreased afterdrop after cooling.

A permutation of AVA rewarming is negative pressure rewarming. Under hypothermic conditions, the AVAs remain closed during peripheral vasoconstriction. In combination with localized heat application, application of subatmospheric pressure theoretically distends the venous rate and increases flow through the AVAs.

To initiate negative pressure rewarming, the forearm is inserted through an acrylic tubing sleeve device fitted with a neoprene collar. This allows an airtight seal to form around the forearm. After a vacuum pressure of –40 mm Hg is created, heat is applied over the dilated AVAs. The thermal load can be provided via an exothermic chemical reaction or a heated perfusion blanket.

The clinical efficacy of AVA rewarming in accidental hypothermia is unclear. Studies concluding that heat exchange is ineffectual used cooler water applied only on the hands and feet.[32,46] The potential for superficial burns of anesthetic, vaso-

constricted skin is a consideration. Another caveat is hypotension precipitated in hypovolemic patients who remain semiupright with this technique. In one study, the rate of core rewarming increased dramatically,[101] but another study comparing negative-pressure rewarming to forced-air warming failed to replicate these results.[284]

Active Core Rewarming

Various techniques that can effectively deliver heat to the core[248,261,295] include heated inhalation, heated infusion, diathermy, lavage (gastric, colonic, mediastinal, thoracic, peritoneal), hemodialysis, and extracorporeal rewarming. Average first-hour rewarming rates reported with some of these techniques in one multicenter study are listed in Figure 5-8. Although hemodynamic instability impacts the rewarming strategy, noninvasive techniques often succeed unless there are significant comorbidities.[249]

Airway Rewarming

The effectiveness of the respiratory tract as a heat exchanger varies with technique and ambient conditions.[210] Because dry air has low thermal conductivity, complete humidification coupled with an inhalant temperature of 40° to 45° C (104° to 113° F) is required.[244] The main benefit of airway rewarming is prevention of respiratory heat loss. Heat yield can represent 10% to 30% of the hypothermic patient's heat production when respiratory minute volume is adequate.[24]

The rate of rewarming is greater using an endotracheal tube (ETT) than by mask. In one series, the reported rewarming rate with a 40° C aerosol was 0.74° C (1.33° F) per hour via mask and 1.22° C (2.2° F) per hour via ETT.[213] In a multicenter survey,[53] the average first- and second-hour rewarming rates in severe cases were 1.5° to 2° C (2.7° to 3.6° F) per hour. Because of the decremental efficiency at higher temperatures, the rate is slower (10 kcal/hr) in mild cases.[53]

Thermal countercurrent exchange in the cerebrovascular bed of humans[57] affects the efficiency and influence of heated mask ventilation during hypothermia. Known as the *rete mirabile*, this system could preferentially rewarm the brainstem. Heated inhalation via face mask continuous positive airway pressure (CPAP) may correct the ventilation–perfusion mismatch.[33] Heated humidified oxygen via face mask is not feasible in some patients with coexistent midface trauma.

Heat liberated during airway rewarming is produced mainly from condensation of water vapor. The latent heat of vaporization of water in the lung is slightly lower than 540 kcal/g H_2O. This is multiplied by the liters per minute ventilation to calculate the quantity of heat transfer. When core temperature is 28° C (82.4° F), the rate of rewarming with heated ventilation at 42° C (107.6° F) equals endogenous heat production. Although the effect on overall thermal balance can be minimal, there may be preferential rewarming of thermoregulatory control centers.[50]

Heated humidified inhalation ensures adequate oxygenation, stimulates pulmonary cilia, and reduces the amount and viscosity of cold-induced bronchorrhea. Although preexisting premature ventricular contractions (PVCs) may reappear during rewarming, there is no evidence that inhalation rewarming precipitates new, clinically significant ventricular arrhythmias. Vapor absorption does not increase pulmonary congestion or wash out surfactant. When the pulmonary vasculature is heated, warmed oxygenated blood that returns to the myocardium could attenuate intermittent temperature gradients. The amplitude of shivering is also lowered, an advantage in more severe cases. This suppression could decrease heat production in mild hypothermia, although experimentally the core temperature continues to rise.[46]

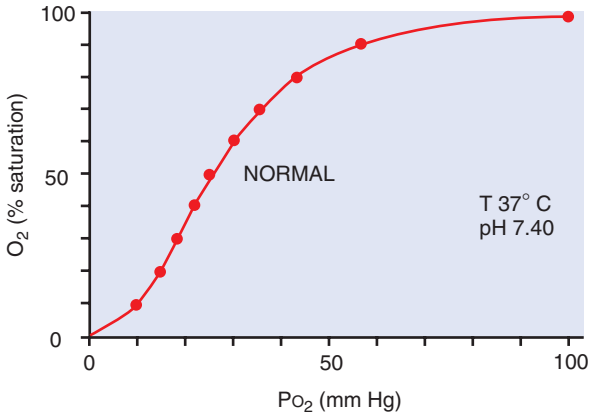


Figure 5-9. Oxyhemoglobin dissociation curve at 37° C (98.6° F). At colder temperatures, the curve shifts to the left.

There are numerous oxygenation considerations in hypothermia (see Box 5-1). The "functional" value of hemoglobin at 28° C (82.4° F) is 4.2 g/10 g in patients on CPB.[80] The oxyhemoglobin dissociation curve also shifts to the left (Fig. 5-9). This impairs release of oxygen from hemoglobin into the tissues. Although some patients can self-adjust their respiratory minute volume (RMV) for current carbon dioxide production, this may not be possible if there are additional toxins or metabolic depressants.[50]

Most humidifiers are manufactured in accordance with International Standards Organization (ISO) regulations. The humidifier will not exceed 41° C (105.8° F) close to the patient outlet with a 6-foot tubing length.[302] If the decision to alter the equipment is made, carefully monitor the temperature and do not exceed 45° C (113° F). The only report of thermal airway injury was in a patient ventilated via endotracheal tube for 11 hours with 80° C (176° F) inhalant.

Strategies to circumvent the 41° C ceiling include reduction of tubing length, adding additional heat sources, disabling the humidifier safety system, and placing the temperature probe outside the patient circuit.[302] Label all modified equipment to avoid routine use. A volume ventilator with a heated cascade humidifier can also deliver CPAP or positive end-expiratory pressure (PEEP) if needed during rewarming. The airway rewarming rates clinically range from 1° to 2.5° C (1.8° to 4.5° F) per hour.[53] In stable patients, circumventing the 41° C ceiling may not be worth the effort because the clinical benefit is modest.[159]

Heat and moisture exchangers function like artificial nares by trapping exhaled moisture and then returning it. They provide inadequate humidification to treat accidental hypothermia. With prolonged use, ETT occlusion and atelectasis are both problems.[40]

Airway rewarming is indicated in the ED when core temperature is lower than 32.2° C (90° F) on arrival. Although airway

rewarming provides less heat than other forms of active core warming, it prevents normal respiratory heat and moisture loss and is safe, fairly noninvasive, and practical in all settings.

Heated Infusions

Cold fluid resuscitation of hypovolemic patients can induce hypothermia. In one series of previously normothermic patients with major abdominal vascular trauma, the average postresuscitation temperature was 31.2°C (88.2°F) in those with refractory coagulopathies. IV fluids are heated to 40° to 42°C (104° to 107.6°F), although higher temperatures may be safe. The amount of heat provided by solutions becomes significant during massive volume resuscitations.[23,269] One liter of fluid at 42°C provides 14 kcal to a 70-kg patient at 28°C (82.4°F), elevating the core temperature almost 0.33°C (0.6°F).

Significant conductive heat loss occurs through IV tubing, so long lengths of IV tubing increase heat loss, especially at slow flow rates.[73] IV tubing insulators are available. There are various methods to achieve and maintain ideal delivery temperature of IV and lavage fluids in hypothermia, but there is no standardized approach.[111,255]

Blood preheated to 38°C (100.4°F) in a standard warmer is useful, but clotting and shortened RBC life are hazards with blood-warming packs. Local microwave overheating hemolyzes blood. An alternative is to dilute packed RBCs with warm calcium-free crystalloid. The Level 1 Fluid Warmer (Life Systems, Inc., Southfield, MI) warms cold crystalloid and blood via a heat exchanger at flow rates of up to 500 mL/min.

A more portable and compact option is the Ranger (Arizant Healthcare, Inc., Eden Prairie, MN). Unlike the Level 1, however, this unit must remain above the level of the patient, and the infusion capability is much lower. The Thermal Angel (Estill Medical Technologies, Inc., Dallas) is a portable, lightweight, battery-powered infusion warmer that has the advantage of portability to the initial point of rescue or trauma.

Microwave heating of IV fluids in flexible plastic bags is another option when more standardized heaters are unavailable. The plasticizer in the polyvinyl chloride containers is stable to microwave heating. Heating times should average 2 minutes at high power for a 1-L bag of crystalloid. The fluid should be thoroughly mixed before administration, to eliminate hot spots.

Rapid administration of fluid into the right atrium at a temperature significantly different from that of circulating blood may produce myocardial thermal gradients.[268] In one study, heated IV fluid, up to 550 mL/min, was administered through the internal jugular vein without complication. In an experimental canine model with adequate cardiac output, central infusion of extremely hot (65°C [149°F]) IV fluids accelerates rewarming without hemolysis.[78,265]

Using amino acid infusions may accelerate energy metabolism.[262] Fever is common in patients receiving hyperalimentation. In patients recovering from elective surgery, however, amino acids have no significant thermogenic effect.[125] The results might differ in energy-depleted patients with chronically induced accidental hypothermia.

In summary, intravenous solutions and blood should be routinely heated during hypothermia resuscitations. Various blood warmers are available commercially, but countercurrent in-line warmers are the most efficient techniques.[138] A mathematical model indicates that infusion heating devices are essential in trauma patients with high fluid requirements.[23]

Heated Irrigation

Gastrointestinal Irrigation

Heat transfer from irrigation fluids is usually limited by the available surface area, so do not use irrigation as the sole rewarming technique. The irrigant should not exceed 45°C (113°F). Direct gastrointestinal irrigation is less desirable than irrigation via intragastric or intracolonic balloons because of induced fluid and electrolyte fluxes. Exceeding 200- to 300-mL aliquots may force fluid into the duodenum; therefore, frequent fluid removal via gravity drainage minimizes "lost" fluid. A log of input and output is essential. This facilitates estimation of fluid balance during resuscitation and helps determine if irrigation should be abandoned in anticipation of dilutional electrolyte disturbances.

To avoid these limitations, a double-lumen esophageal tube is available, as are other modified Sengstaken tubes.[175] Patients should be tracheally intubated before gastric lavage. Because of the proximity of an irritable heart, overly vigorous placement of a large gastric tube seems ill advised. In a multicenter survey, gastric, bladder, and colon lavage rewarmed severely hypothermic patients at 1° to 1.5°C (1.8° to 2.7°F) for the first hour and 1.5° to 2°C (2.7° to 3.6°F) for the second hour.[53]

Commercially available kits designed for gastric decontamination are convenient (Fig. 5-10). The use of a Y connector and

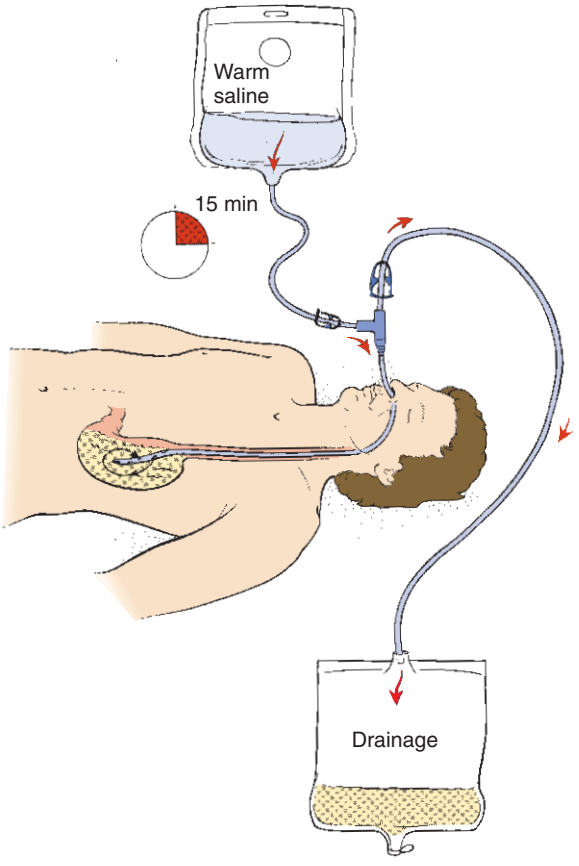


Figure 5-10. Gastric lavage.

clamp simplifies the exchanges. Ideal dwell times for thermal exchange depend on flow rates and may average several minutes. In direct gastric lavage, warmed electrolyte solutions, such as normal saline or Ringer's lactate, are administered via nasogastric tube.[187] After 15 minutes, the solution is aspirated and replaced with warm fluids. Disadvantages include the small surface area available for heat exchange and the large amount of fluid escaping into the duodenum.[31] Regurgitation is common, and the technique must be terminated during CPR. Esophageal heat exchange is very limited.[177,243,308,309] Aesthetic obstacles aside, heat transfer through bladder and colon irrigation is negligible.

Mediastinal Irrigation

Mediastinal irrigation and direct myocardial lavage are alternatives in patients lacking spontaneous perfusion. A standard left thoracotomy is performed while CPR is continued. Opening the pericardium is unnecessary unless an effusion or tamponade is present. The physician bathes the heart for several minutes in 1 to 2 L of an isotonic electrolyte solution heated to 40° C (104° F), then suctions and replaces warm fluids.[29]

The physician may attempt internal defibrillation after myocardial temperature reaches 26° to 28° C (78.8 to 82.4° F). Unless a perfusing rhythm is achieved, lavage is continued until myocardial temperature exceeds 32° to 33° C (89.6° to 91.4° F). A standard post-thoracotomy tube in the left side of the chest could provide an avenue for continued rewarming via thoracic irrigation.

A median sternotomy also allows ventricular decompression and direct defibrillation.[186] One potential disadvantage of both these techniques is that open cardiac massage of a cold, rigid, contracted heart may not generate flow.[4,47] Unless immediate CPB is an option, mediastinal irrigation and direct myocardial lavage are indicated only if cardiac arrest has occurred. In this circumstance, personnel skilled in the technique should also initiate all other available rewarming modalities.

Thoracic Lavage

Irrigation of the hemithoraces is a valuable rewarming adjunct.[11,29,30,166,305] An important semantic issue is that closed thoracic lavage via two thoracostomy tubes differs from open mediastinal and direct myocardial lavage. With the latter, closed-chest CPR is not possible. Two large-bore thoracostomy tubes (36 to 40 fr in adults; 14 to 24 fr, ages 1 to 3; 20 to 32 fr, ages 4 to 7) are inserted in one or both of the hemithoraces. One is placed anteriorly in the second to third intercostal space at the midclavicular line, and the other in the posterior axillary line at the fifth to sixth intercostal space. Normal saline heated to 40° to 42° C (104° to 107.6° F) is then infused via a nonrecycled sterile system (Fig. 5-11A).

A high-flow countercurrent fluid infuser (Level 1 Fluid Warmer) heats to 40° C and delivers normal saline in 1-L or preferably 3-L bags into the afferent chest tube.[272] This author prefers connecting into the Level 1 tubing with standard $^{3}/_{16}$-inch-internal-diameter suction connection tubing and a sterilized plastic graduated two-way connector, because this facilitates adaptation to any size chest tube (see Figure 5-11A). The effluent is then collected in a thoracostomy drainage set. The reservoir must be emptied frequently. Alternatively, when a single chest tube is used, 200- to 300-mL aliquots are used for irrigation, and suctioning is achieved through a Y connector. The Y connector is also useful for irrigating both hemithoraces with a single fluid warmer (see Figure 5-11B).

Fluid can be infused into the anterior higher chest tube (afferent limb) and suctioned or gravity-drained out the lower posterior tube (efferent limb) into a water-seal chest drain.[109] Infusion inferoposteriorly with suction anteriorly can increase dwell times.[140] The efficiency of thermal transfer varies with flow rates and dwell times. Once the patient is successfully rewarmed, the upper tube should be removed and the lower one left in place to allow residual drainage.

Closed sterile thoracic lavage is a natural choice in the ED during potentially salvageable cardiac arrest resuscitations.[315] Thoracic lavage is an option either as a bridge to CPB or when CPB is unavailable. In patients who are perfusing, this technique should be considered hazardous unless extracorporeal rewarming capability is immediately available. Many hypothermic trauma patients are irrigated successfully during surgery.

The clinically reported rates range from 180 to 550 mL/min. The overall rate of rewarming should easily equal or exceed that achievable with peritoneal lavage and is often 3° to 6° C (5.4° to 10.8° F) per hour. The surface area of the pleural space is well perfused. An added benefit is preferential mediastinal rewarming. In addition, closed chest compressions during cardiac arrest can maintain perfusion. Open cardiac massage of a rigid, contracted heart may not be possible in severe cases before bypass, which is a problem with mediastinal irrigation.[4,47,52]

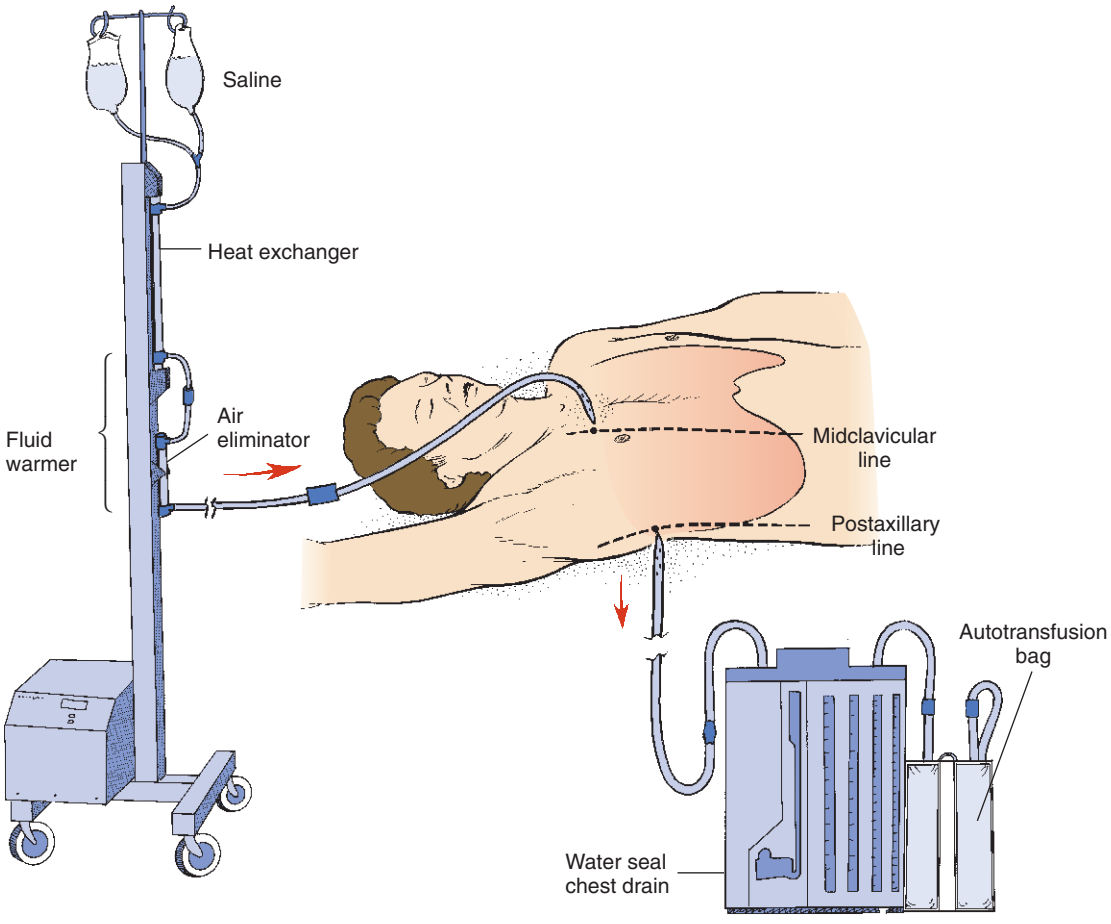
Various complications should be considered. Left-sided thoracostomy tube insertion into patients who are perfusing could easily induce VF. Patients with pleural adhesions or a history of pleurodesis have poor infusion rates, and subcutaneous edema may develop. If the fluids are infused under pressure without adequate drainage, intrathoracic hypertension or even a tension hydrothorax can develop and cause expected adverse cardiovascular effects.[153]

Peritoneal Lavage

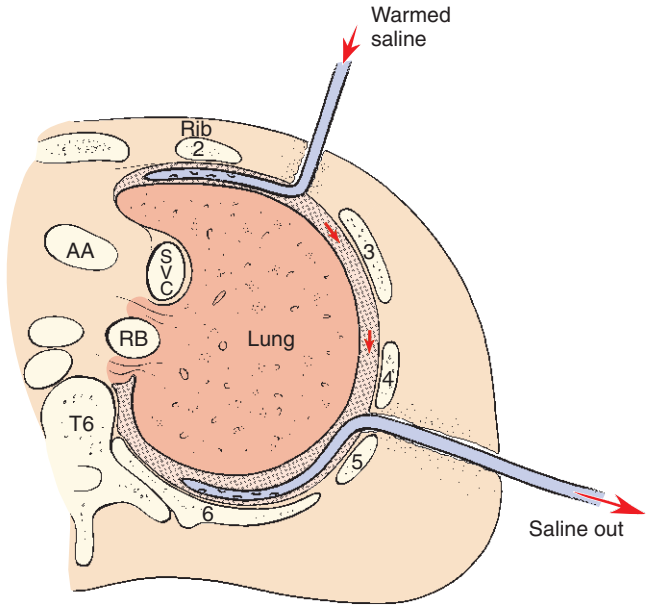
Heated peritoneal lavage is a technique available in most facilities (Fig. 5-12). Heat is conducted intraperitoneally via isotonic dialysate delivered at 40° to 45° C (104° to 113° F).[298]

Before lavage is initiated, chest and abdominal radiographs should be obtained because subsequent films may reveal subdiaphragmatic air introduced during the procedure. The bladder and stomach must be emptied before insertion of the catheter. The two common techniques for introducing fluid into the peritoneal cavity are the minilaparotomy and the percutaneous puncture.

The "minilap" requires an infraumbilical incision through the linea alba. A supraumbilical approach is necessary if previous surgical scars, a gravid uterus, or pelvic trauma is identified. The peritoneum is punctured under direct visualization and dialysis catheter(s) inserted. A much simpler and more rapid technique is the guidewire, or Seldinger, variation of the percutaneous puncture. The site is infiltrated if necessary with lidocaine, and a small stab incision is made. An 18- to 20-gauge needle penetrates the peritoneum, and a guidewire is introduced. Entry into the peritoneum is usually recognizable by a distinct "pop." A disposable kit is available (Arrow Peritoneal Lavage Kit [AK-0900], Arrow International, Inc., Reading, PA). The 8-fr lavage catheter is inserted over the wire and advanced into one of the pelvic gutters. Double-catheter systems with outflow suction speed rewarming.



A



B

Figure 5-11. Thoracic lavage. **A,** Cycle. **B,** Cross section.

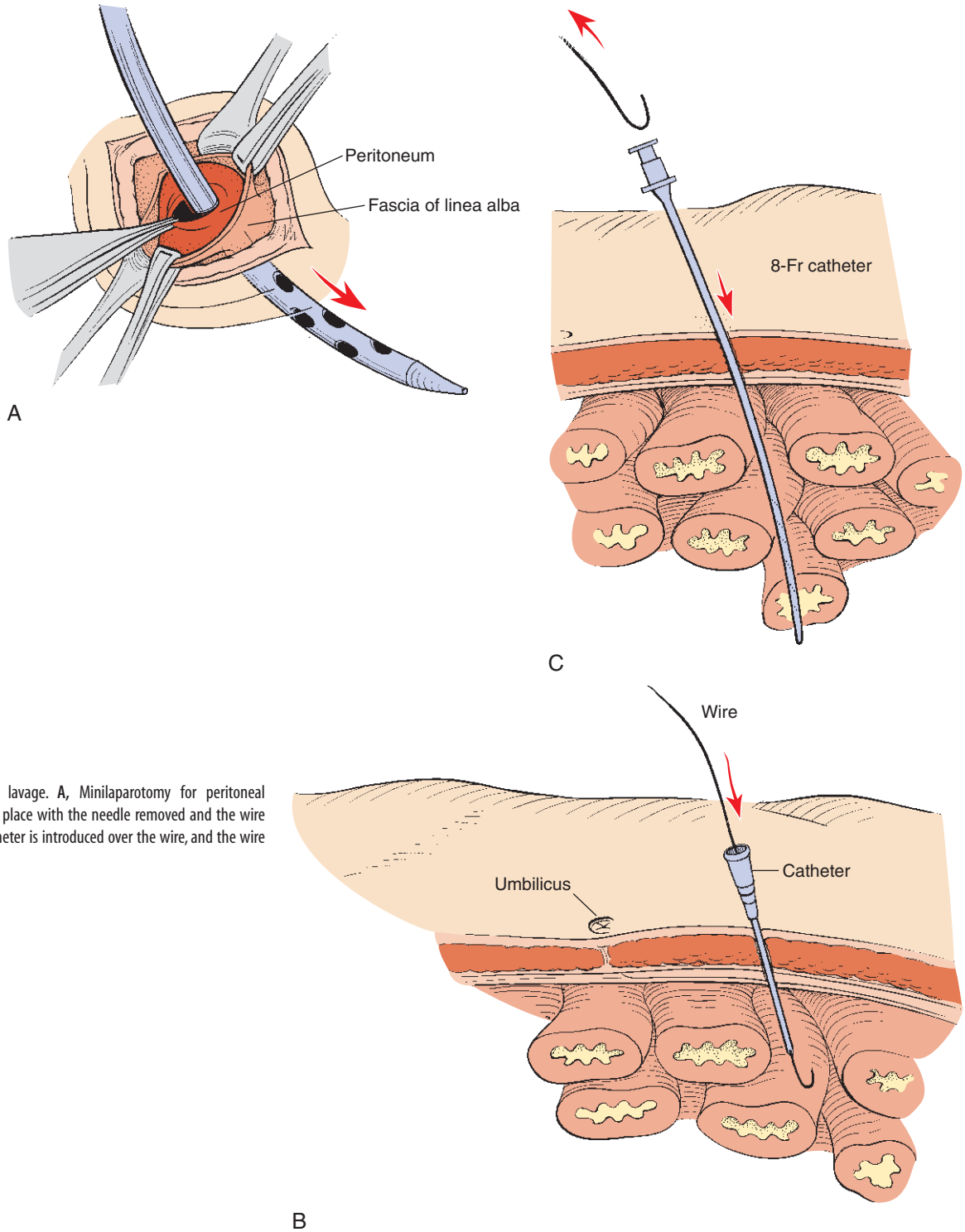


Figure 5-12. Peritoneal lavage. **A,** Minilaparotomy for peritoneal lavage. **B,** The catheter in place with the needle removed and the wire introduced. **C,** An 8-fr catheter is introduced over the wire, and the wire is then removed.

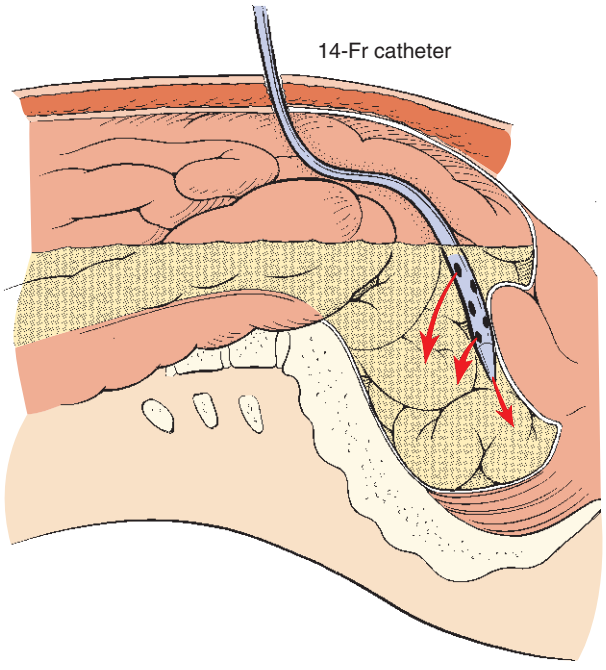


Figure 5-13. Peritoneal lavage. A 14-fr catheter is of greater caliber and can infuse fluids more rapidly.

TABLE 5-4. Techniques for Extracorporeal Blood Rewarming

TECHNIQUE	COMMENTS
Cardiopulmonary bypass	Full circulatory support Perfusate temperature gradient: consider 5°–10° C Flow rates of 2–7 L/min (average, 3–5 L/min) Rate of rewarming, up to 9.5° C/hr Consider if K < 10 mmol/L, pH > 6.5, at least 10°–12° C
Continuous arteriovenous warming	Percutaneous Seldinger technique to insert catheters Requires blood pressure of ≥ 60 mm Hg No need for perfusionist/pump/anticoagulation Average flow rates of 225–375 mL/min Rate of rewarming, 3°–4° C/hr Consider if trauma
Venovenous rewarming	Circuit not complex Efficient nonbypass modality Volume infusion to augment cardiac output Flow rates of 150–400 mL/min Rate of rewarming, 2°–3° C/hr
Hemodialysis	Widely available Portable and efficient Single or dual catheter Exchange cycle volumes, 200–500 mL/min Rate of rewarming, 2°–3° C/hr Consider if there are electrolyte/toxicologic derangements

Adapted from Danzl DF: Semin Resp Crit Care Med 23:57, 2002.

Normal saline, lactated Ringer's solution, or standard 1.5% dextrose dialysate solution with optional potassium supplementation can be used.[272] Isotonic dialysate is heated to 40° to 45° C (113° F). Up to 2 L is then infused (10 to 20 mL/kg), retained for 20 to 30 minutes, and aspirated. The usual clinical exchange rate is 6 L/hr, which yields rewarming rates of 1° to 3° C (1.8° to 5.4° F) per hour. An alternative for severe cases is a larger catheter, as found in cavity drainage kits (Arrow 14 fr [AK-01601]) (Fig. 5-13). The catheter can be placed with the Seldinger technique. The higher drainage capability markedly increases exchange rates and minimizes the dwell times necessary for maximal thermal transfer. The flow rate via gravity through regular tubing is approximately 500 mL/min, which can be tripled under infusion pressure.

A unique advantage of peritoneal dialysis is drug overdose and rhabdomyolysis detoxification when hemodialysis is unavailable. In addition, direct hepatic rewarming reactivates detoxification and conversion enzymes. Peritoneal dialysis worsens preexisting hypokalemia. Vigilant electrolyte monitoring is essential before empirical modification of the dialysate. The presence of adhesions from previous abdominal surgery increases the complication rate and minimizes heat exchange.

Peritoneal dialysis during standard mechanical CPR is as effective as partial cardiac bypass in resuscitating severely hypothermic dogs.[313] In contrast to AER, peritoneal lavage rewarming did not require significantly greater quantities of crystalloids and bicarbonate. This exchange rate is rarely possible in humans. Bowel infarction may be a concern when using prolonged warm peritoneal dialysis in patients with severe hypothermia with inadequate visceral perfusion during CPR.

Peritoneal lavage is invasive and should not be routinely used in treating stable, mildly hypothermic patients. Extracorporeal rewarming should be available in case of rare major complications, including VF. This technique is indicated in combination with all available rewarming techniques in cardiac arrest patients.

Extracorporeal Blood Rewarming

Table 5-4 summarizes the techniques for extracorporeal blood rewarming.

Hemodialysis

Standard hemodialysis is widely available, practical, portable, and efficient, and it should be strongly considered in patients with electrolyte abnormalities, renal failure, or intoxication with a dialyzable substance.[220,230] Two-way flow catheters allow the option of cannulation of a single vessel.[179] A Drake-Willock single-needle dialysis catheter can be used with a portable hemodialysis machine and an external warmer. After central venous cannulation, exchange cycle volumes of 200 to 250 mL/min are possible.

Although heat exchange is less than with standard two-vessel hemodialysis, the ease of percutaneous subclavian vein placement is a major advantage. Hemodialysis via two separate single-lumen catheters placed in the femoral vein can achieve continuous blood flow at 450 to 500 mL/min.[121,122] In-line hemodialysis also simplifies correction of electrolyte abnormalities. Local vascular complications, including thrombosis of

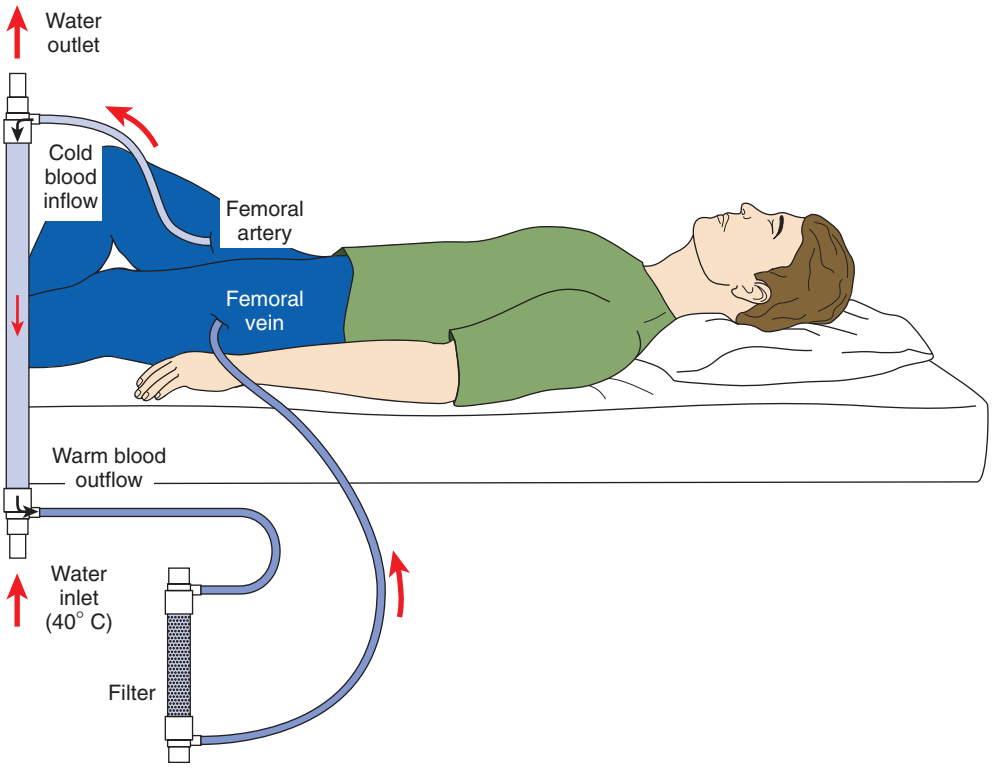


Figure 5-14. Schematic of continuous arteriovenous rewarming.

vessels and hemorrhage secondary to anticoagulation, may occur.

Venovenous Rewarming

In extracorporeal venovenous rewarming, blood is removed, usually from a central venous catheter, heated to 40°C (104°F), and returned via a second central or large peripheral venous catheter. Flow rates average 150 to 400 mL/min.[104,117,275]

The circuit is not complex and is more efficient than many other nonbypass modalities. There is no oxygenator, and because the method does not provide full circulatory support, volume infusion is the only option to augment inadequate cardiac output. Although the use of continuous arteriovenous rewarming (CAVR) is limited by profound hypotension, high-flow venovenous rewarming may prove to be an alternative.[142]

In another variation of the extracorporeal venovenous circuit, blood is removed from the femoral vein, heparinized, and sent through a blood rewarmer via an infusion pump accelerator. It is neutralized with protamine before re-injection into the subclavian or internal jugular vein, which would preferentially rewarm the heart. Another option is to insert a femoral vein dual-lumen dialysis catheter.

Continuous Arteriovenous Rewarming

CAVR involves the use of percutaneously inserted femoral arterial and contralateral femoral venous catheters.[82,83,85] This technique requires a blood pressure of at least 60 mm Hg. The Seldinger technique is used to insert 8.5-fr catheters. Heparin-bonded tubing circuits obviate the need for systemic anticoagulation. CAVR has principally been performed on traumatized patients (Fig. 5-14).

The blood pressure of spontaneously perfusing traumatized hypothermic patients creates a functional arteriovenous fistula by diverting part of the cardiac output out of the femoral artery through a countercurrent heat exchanger (e.g., Level 1; Flo Temp-IIe). The heated blood is then returned with admixed heated crystalloids via the femoral vein. The additional fluids are titrated and infused by piggyback until hypotension is corrected.

The rate of rewarming exceeds that of hemodialysis. CAVR does not require the specialized equipment and perfusionist necessary for CPB. The average flow rates are 225 to 375 mL/min, resulting in a rate of rewarming of 3° to 4°C (5.4° to 7.2°F) per hour. Because the catheters are 8.5 fr, the patient must weigh at least 40 kg. Coagulation begins to appear in the heparinized circuits at around 3 hours.

Cardiopulmonary Bypass

Partial or complete CPB should be considered in unstable, severely hypothermic patients.[12,105,164,247,318] Favorable considerations include absence of severe head injury or asphyxia.[270] Some centers initiate CPB only if the presenting arterial pH is above 6.5, serum potassium is below 10 mmol/L, and core temperature is above 10° to 12°C (50° to 54°F).[128,290,302]

Fischer's considerations for CPB include a potassium level under 10 mmol/L in adults or under 12 mmol/L in children, coupled with a core temperature below 30°C. Patients are not resuscitated if extreme hyperkalemia is identified in a patient with a temperature over 30°C (86°F).[79]

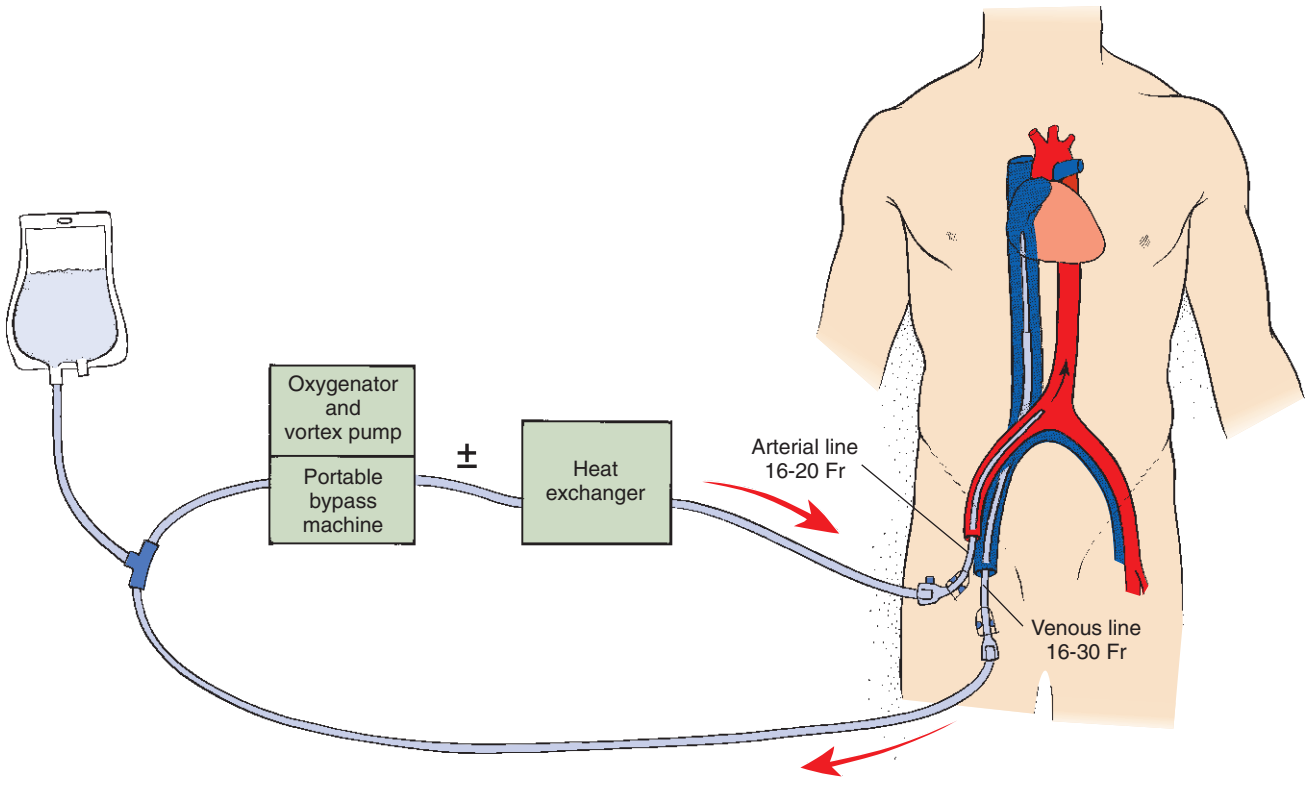


Figure 5-15. Femoral–femoral bypass.

A major advantage of CPB is preservation of oxygenated flow if mechanical cardiac activity is lost during rewarming.[61,134,136,205] CPB is three to four times faster at rewarming than other active core rewarming (ACR) techniques, and it reduces the high blood viscosity associated with severe cases. CPB should also be considered when severe cases do not respond to less invasive rewarming techniques, in patients with completely frozen extremities, and when rhabdomyolysis is accompanied by major electrolyte disturbances.[149]

Various extracorporeal rewarming techniques can be lifesaving in selected profound cases of hypothermia.[114] Althaus[4] describes complete recovery in three severely hypothermic tourists after prolonged periods of cardiac arrest and CPR. In another review of 17 cases, there were 13 survivors.[274] Walpoth and coworkers[303,304] report rewarming 32 patients with CPB, of which 15 are long-term survivors. The average age was 25.2 years. Their mean presenting esophageal temperature was 21.8°C (71.2°F), and the mean interval between discovery and CPB was 141 minutes.

The standard femoral–femoral circuit includes arterial and venous catheters, a mechanical pump, a membrane or bubble oxygenator, and a heat exchanger (Fig. 5-15). A 16- to 30-fr venous cannula is inserted via the femoral vein to the junction of the right atrium and the inferior vena cava. The tip of the shorter 16- to 20-fr arterial cannula is inserted 5 cm or proximal to the aortic bifurcation. Antretter and colleagues[6] use 32-fr venous and 28-fr arterial cannulas with the open surgical technique, and 21-fr venous and 19-fr arterial cannulas if inserted percutaneously. Closed chest compressions can be maintained during percutaneous or open surgical technique insertion and may help decompress the dilated nonbeating heart. Transesophageal echocardiography can help evaluate ventricular load and valve function.

Heated, oxygenated blood is returned via the femoral artery. Femoral flow rates of 2 to 3 L/min can elevate core temperature 1° to 2°C (1.8° to 3.6°F) every 3 to 5 minutes. In a review by Splittgerber and coworkers,[274] the mean CPB temperature increase was 9.5°C (17.1°F) per hour. Most pumps can generate full flow rates up to 7 L/min. Long[196] recommends considering the use of vasodilator therapy with IV nitroglycerin to facilitate perfusion. He initiates bypass flow rates at about 2 L/min and gradually increases to 4 to 5 L/min. Vasoactive agents may be needed to maintain the cardiac index at 30 L/min/m^2 or more and a (low) systemic vascular resistance of 1000 dynes/sec × cm^{-5} or less.

The optimal temperature gradient and bypass rewarming rates are unknown. One study of rewarming via CPB in a swine model cooled to 23°C (73.4°F) addresses this concern. An excessive temperature gradient between brain tissue and circulant adversely affected electroencephalographic (EEG) regeneration. The other theoretical concern is the possibility of increased bubbling if high perfusate temperature gradients are used. Most investigators use 5°C (9°F) gradients[19] or 10°C (18°F) gradients.[20] Eventually, neuromonitoring during rewarming will provide some answers. In severe cases, evoked cerebral responses before EEG regeneration could help assess the recovering brain.

Box 5-8. Extracorporeal Rewarming

COMPLICATIONS
- Vascular injury
- Air embolism
- Pulmonary edema
- Coagulopathies (hemolysis, disseminated intravascular coagulation)
- Frostbite tissue damage
- Extremity compartment syndromes

CONTRAINDICATIONS
- Lack of venous return
- Intravascular clots or slush
- Complete heparinization would be hazardous*
- Cardiopulmonary resuscitation is contraindicated (see Box 5-9)

*Unless with athrombogenic tubing or adequate physiologic fibrinolysis.

A variety of techniques decrease the need for IV anticoagulation with heparin, which previously limited clinical applicability.[163] Heparin-coated perfusion equipment was used successfully without systemic heparinization in a patient with hypothermic cardiac arrest (23.3°C [73.9°F]) and intracranial trauma.[300] The use of nonthrombogenic pumps, coupled with enhanced physiologic fibrinolysis seen in the first hour of CPB, has also succeeded experimentally.[59]

Complications with the standard technique include vessel damage, air embolism, hemolysis, DIC, and pulmonary edema (Box 5-8). Endothelial leakage increases compartment pressures and exacerbates frostbite. If adequate flow rates of 3 to 4 L/min (50 to 60 mL/kg/min) cannot be maintained, thoracotomy or a venous catheter with side holes, augmenting intravascular volume, should be considered.

With all four of these techniques, there is no proof that rapid acceleration of the rate of rewarming improves survival rates in patients being perfused. Potential complications of uncontrolled rapid rewarming in severe hypothermia include DIC, pulmonary edema, hemolysis, and acute tubular necrosis. As an alternative, a conservative core-rewarming approach can be highly effective for patients with severe hypothermia, despite hemodynamic instability.[249]

In hypothermic cardiac arrest, rewarming should be attempted via CPB and hemodialysis when CPR is not contraindicated (Box 5-9), unless frozen intravascular contents prevent flow. Clotted atrial blood or failure to obtain venous return indicates that these techniques will be futile. If experienced personnel and necessary equipment are unavailable, all other rewarming techniques should be used in combination.[19,316]

Diathermy

Diathermy, the transmission of heat by conversion of energy, is a potential rewarming adjunct in accidental hypothermia.[126,194] Large amounts of heat can be delivered to deep tissues with ultrasonic (0.8 to 1 MHz) and low-frequency (915 to 2450 MHz) microwave radiation. Short-wave (13.56 to 40.68 MHz) modalities are high frequency and do not penetrate deeply. Contraindications include frostbite, burns, significant edema, and all types of metallic implants and pacemakers.

Box 5-9. Contraindications to Initiating Cardiopulmonary Resuscitation in Accidental Hypothermia

- Do-not-resuscitate status is documented and verified.
- Obviously lethal injuries are present.
- Chest wall depression is impossible.
- Any sign of life is present.
- Rescuers are endangered by evacuation delays or altered triage conditions.

From Danzl DF, Pozos RS, Auerbach PS, et al: Ann Emerg Med 16:1042–1055, 1987. Developed in conjunction with the Wilderness Medical Society.

Under ideal conditions in a laboratory study, radiowave frequency (13.56 MHz) electromagnetic regional heating of hypothermic dogs after immersion did not damage tissue at 4 to 6 watts/kg and rapidly elevated the core temperature.[314] Zhong and colleagues[325] successfully rewarmed 16 piglets with microwave irradiation "until they squealed and suckled." Subsequently, 20 of 28 human infants who were rewarmed with microwave irradiation at 90 to 100 watts survived. The temperature rose an average 1°C (1.8°F) after 6 to 7 minutes, and the average infant required 45 minutes to achieve a rectal temperature of 36°C (96.8°F). In an experimental study of men cooled to 35°C (95°F), warm water immersion rewarming was more rapid than radiowave rewarming with 2.5 watts/kg.[157]

Both ultrasonic and low-frequency microwave diathermy can deliver large quantities of heat below the skin. As dosimetry guidelines are developed, potential complications and ideal application sites for this experimental technique deserve further study in the hospital. In the field setting, potential problems with power supply and electronic and navigational interference compound the physiologic problems.

CARDIOPULMONARY RESUSCITATION

Basic and advanced life support recommendations in hypothermia continue to evolve (Fig. 5-16).[159,279] Cardiac output generated with closed-chest compressions maintains viability in selected patients with hypothermia.[281] The optimal rate and technique are unknown. Definitive prehospital determination of cardiac activity requires a cardiac monitor, because misdiagnosis of cardiac arrest is a hazard. Peripheral pulses are difficult to palpate when extreme bradycardia is present with peripheral vasoconstriction.

Asystole may be as common a presenting rhythm as VF. In the field, differentiating VF from asystole may be impossible. Possible causes of VF include acid–base fluxes, hypoxia, and coronary vasoconstriction with increased blood viscosity. Chest compressions and various therapeutic interventions are also implicated. The role of acid–base fluxes is not clear. Mild alkalosis appears somewhat protective against VF during controlled, induced hypothermia.[176]

Respiratory Considerations

When cardiopulmonary arrest develops during resuscitation, noncardiac causes are often pulmonary emboli or progressive

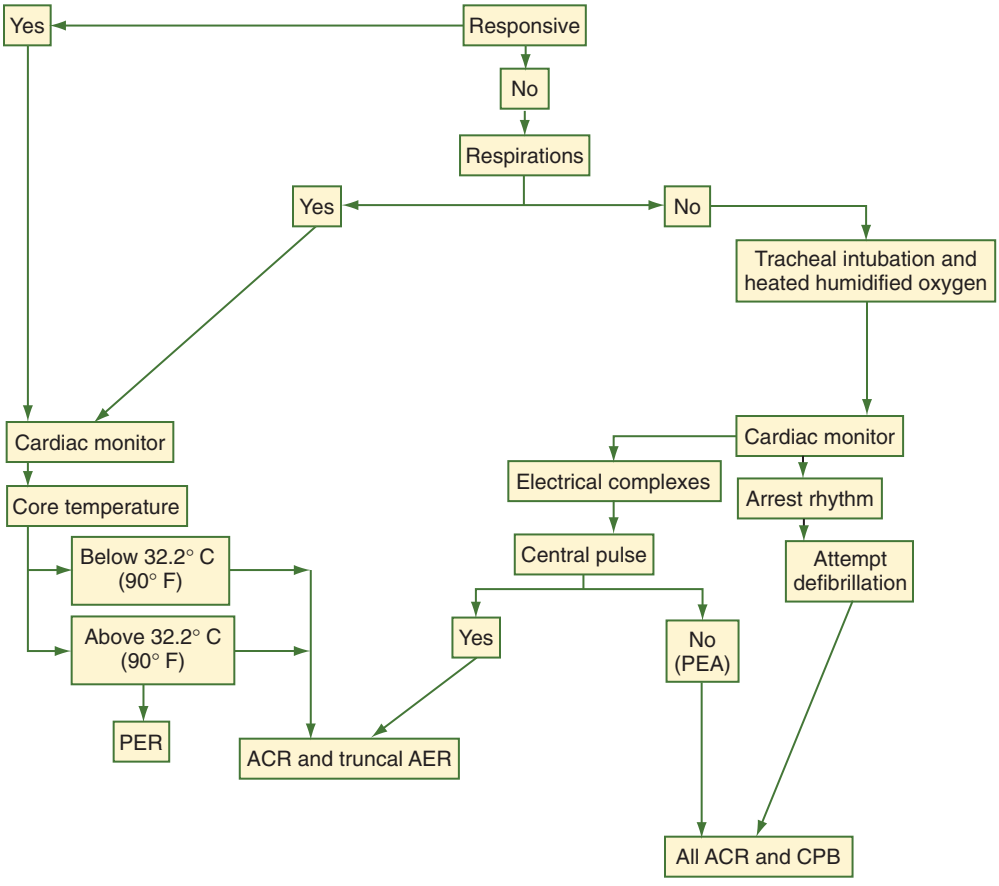


Figure 5-16. Emergency department algorithm. ACR, active core rewarming; AER, active external rewarming; CPB, cardiopulmonary bypass; PEA, pulseless electrical activity; PER, passive external rewarming.

respiratory insufficiency. Provision of adequate oxygen supply is essential during rapid rewarming. For each 1° C (1.8° F) rise in temperature, oxygen consumption increases up to three times (see Box 5-1).[80]

Endotracheal intubation and ventilation decrease atelectasis and ventilation–perfusion mismatch. Complete airway protection averts aspiration, which is otherwise common with depressed airway reflexes, bronchorrhea, and ileus. Carbon dioxide production also drops by half with an 8° C (14.4° F) fall in the temperature. During induced hypothermia, carbogen (1% to 5% carbon dioxide added to oxygen) facilitates acid–base management by allowing adjustment of the fractional inspired CO_2 concentration ($FICO_2$) while adjusting the ventilation. Carbogen will undoubtedly play a future role in accidental hypothermia.

Past controversy regarding the hazards of endotracheal intubation reflected coincidental episodes and a mis-citation by Fell and coworkers[75] of a series of hypothermic overdoses by Lee and Ames.[184] Fell stated that "endotracheal intubation was followed by cardiac arrest in a large proportion of cases," whereas Lee and Ames merely cautioned of that possibility. In a multicenter survey, endotracheal intubation was performed on 117 patients by multiple operators in various settings.[53] No induced arrhythmias were recognized, which is consistent with several reports.[183,213] Potential arrhythmogenic factors include hypoxia, mechanical jostling, and acid–base or electrolyte fluctuations.

The indications for endotracheal intubation in hypothermia are identical to those in normothermia.[48,95] It is required unless the patient possesses intact protective airway reflexes. Ciliary activity is depressed in hypothermia, frothy sputum produces chest congestion, and bronchorrhea resembles pulmonary edema. Fiberoptic or blind nasotracheal intubation may be preferable to cricothyroidotomy when cold-induced trismus or potential cervical spine trauma is present. Oral rather than nasal intubation is advisable in patients with coagulopathy, to avoid causing major epistaxis.

Rapid-sequence intubation may be impossible when severe cold-induced trismus is present and the mandible will not move. In the milder cases, hypothermia prolongs the duration of neuromuscular blockade. Drug metabolism is also prolonged with vecuronium and atracurium. As a result, avoid using neuromuscular blockers.[159] Efficacy, metabolism, and clearance are very unpredictable and assessment of sedation adequacy is difficult.

Blood Flow during Chest Compressions

During normothermic conditions, blood flow partially results from phasic alterations in intrathoracic pressure and not just from direct cardiac compression. Niemann and colleagues[225] demonstrate that antegrade flow occurs without left ventricular compression in a normothermic canine model. Closed-chest

compressions increase intrathoracic pressure.[199] When thoracic inlet venous valves are competent, the resultant pressure gradient between arterial and venous compartments generates supradiaphragmatic antegrade flow.

Clinical observations challenge the "cardiac pump" model. This model is predicated on closure of the mitral valve and opening of the aortic valve during chest compression. This allows a forward stroke volume. During release of compression, transmitral flow can fill the left ventricle. Optimal cardiac output is thus generated by achieving the maximal compression rate that allows maximal left ventricular end-diastolic filling. Interestingly, transesophageal echocardiography in a canine model demonstrates mitral valve closure during chest compression except during low-impulse (downstroke momentum) compressions. Compression of a cold, stiff chest wall may be equivalent to low impulse.[47]

In hypothermia, the role of a "thoracic pump" with the heart as a passive conduit is an attractive hypothesis. The phasic alterations in intrathoracic pressure generated by compressions are equally applied to all of the cardiac chambers and thoracic vessels. The mitral valve remains open during compression, and blood continues to circulate through the left side of the heart.

Myocardial compliance can be severely reduced in hypothermia. Althaus and coworkers[4] note at thoracotomy in one of three survivors of hypothermia that "the heart was found to be hard as stone and it is hardly conceivable how effective external cardiac massage could have been." Open cardiac massage can be combined with mediastinal irrigation when immediate cardiopulmonary bypass is an option.[29–31]

Chest wall elasticity is also decreased with cold, as is pulmonary compliance. If the abdominal muscles are not kneadable, anticipate that the stiff chest wall will prevent adequate ventilation. Lastly, more force is needed to depress the chest wall sufficiently to generate intrathoracic vascular compartment pressure gradients. Despite these potential physiologic explanations, a large number of neurologically intact survivors remain after prolonged closed-chest compressions. The explanation lies in measuring intrathoracic pressures during hypothermic closed-chest compressions.

Perfusion enhancement maneuvers remain largely unstudied in hypothermia. Ancillary abdominal binding, as with the abdominal compartment of antishock trousers, might favorably inhibit paradoxical diaphragmatic motion.[47,225] Simultaneous compression and ventilation can increase flow in the heart's left side. Ventilation with the proper carbogen concentration also allows high ventilatory rates while maintaining uncorrected $Paco_2$ at 40 mm Hg. Placement of a counterpulsation intraaortic balloon is another option. Mechanical thoracic compression devices could be useful during prolonged resuscitations with limited availability of personnel and no bypass capabilities.

During hypothermic cardiac arrest in swine, the cardiac output, cerebral blood flow, and myocardial blood flow averaged 50%, 55%, and 31%, respectively, of those achieved during normothermic closed-chest compressions.[201] Blood flow to these areas did not decrease with time, unlike in the normothermic group. No significant difference in flow generated appeared between normothermic and hypothermic swine at 20 minutes. Hypothermic rheologic changes, including increased viscosity, also affect flow. Peripheral vascular resistance is expected to increase during vasoconstriction, but in the swine there was no difference in systemic and organ vascular resistance between normothermic and hypothermic CPR.[201]

In a multicenter survey of 428 cases, 9 of the 27 patients receiving CPR initiated in the field survived, as did 6 of 14 patients with ED-initiated CPR.[53] Based on these cases and a literature review, CPR standards in hypothermia are proposed (see Box 5-9).

Because cardiac output is the product of heart rate and stroke volume, the optimal closed-chest compression rate should be the fastest rate allowing optimal ventricular filling. Many patients have recovered neurologically after slow compression rates. One recovered after 220 minutes at half the normal compression rate.[226] The optimal rate probably has a direct linear relationship with the degree of hypothermia.

Tissue decomposition, apparent rigor mortis, dependent lividity, and fixed, dilated pupils are not reliable criteria for withholding CPR. In addition, intermittent flow may provide adequate support during evacuation. Although one position paper implies otherwise, in the opinion of this author CPR should not be withheld only because continuous chest compressions cannot be ensured.[37,67] Intermittent flow may be preferable to no flow. The lowest temperature documented in an infant survivor of accidental hypothermia is 15.2°C (59.4°F); in an adult, 13.7°C (56.8°F); and in induced hypothermia, 9°C (48.2°F).[45,55,94,222,227] One patient recovered after $6^1/_2$ hours of closed-chest compressions.[189] In Saskatchewan in 1994, a 2-year-old child reportedly recovered from a core temperature of 13.9°C (57°F). Remarkably, a physician who authored this report was successfully resuscitated from 13.9°C (57°F) after she received 165 minutes of CPR and then CPB.[94]

CEREBRAL RESUSCITATION

The ability to reestablish cerebral blow flow with CPR and improve neurologic outcome is inversely proportional to the duration of cardiac arrest. Significant delay can markedly elevate the cerebral perfusion pressure (CPP) required to establish cerebral blood flow (CBF).[16]

It appears that thermal stabilization rather than aggressive field rewarming should be the focus, except for severely hypothermic victims or those in cardiac arrest. Mild hypothermia may be therapeutic and improve the neurologic outcome after global cerebral ischemia. Hypothermia decreases glutamate release, metabolic demand, free radical formation, and release of inflammatory cytokines.[97,252]

In the prehospital setting, addressing disproportionate hypotension, hypoperfusion, and hypoxia is paramount. Once the airway is secured, elevating the head 5 to 10 degrees can attenuate intracerebral hypertension induced by global cerebral ischemia. Normally, CBF is independent of CPP over a range of pressures. After cerebral autoregulation is extinguished, CBF becomes passively dependent on the arterial blood pressure. When venous access is difficult, medications may be administered intratracheally or via the intraosseous route. Of interest, large internal jugular catheters may partially obstruct cerebral venous outflow.[203]

Because hypoxemia is the rule, supplemental oxygen should be initiated when available. The goal is normoxia. The prolonged use of 100% oxygen after the return of spontaneous circulation may increase oxidative brain injury by generating reactive oxygen species, including superoxide and hydrogen peroxide. The rescuer should avoid positive end-expiratory

pressure unless it is essential to maintain normoxia, because it decreases cerebral venous outflow.

Cerebral vasculature autoregulation may be lost after global brain ischemia, and yet hypocarbia-induced vasoconstrictive mechanisms remain intact. Therefore, avoid protracted hyperventilation except in the rare case of a cranially traumatized hypothermic victim with impending uncal herniation. In the field, attempt to ventilate the patient at a rate that would maintain the uncorrected P_{CO_2} between 35 and 40 mm Hg.[202]

Postischemic hyperglycemia is potentially detrimental, because glucose levels over 250 mg/dL are associated with lactate generation and exacerbation of metabolic acidosis.

Cerebral resuscitation strategies in hypothermia reflect complex neurophysiology.[252] The brain, while making up only 2% of the total body weight, requires 15% of the cardiac output and consumes 20% of the available oxygen at 37° C. Cerebral blood flow depends on the CPP, which equals the mean arterial pressure minus the intracranial pressure. When the CPP drops below 50 to 60 mm Hg, CBF drops. Once CBF is below 20%, a cascade is initiated that includes the failure to maintain ionic gradients in neurons, cellular depolarization, and acidosis. Even when cold, neurons are more susceptible to hypoxic insult than are glial or endothelial cells.[133,152]

When reperfusion is reestablished, the initial transient hyperemic period is characterized by increased CBF with low oxygen consumption. This lasts for minutes and is followed by the "no reflow" phase, which lasts for hours. Reperfusion is also characterized by both cytotoxic edema during ischemia, and vasogenic edema due to blood–brain barrier injury. Intraluminal vascular damage is caused by nitric oxide (NO) and reactive oxygen species (ROS), and proteases inflict abluminal injury.[39]

The decrease in CBF decreases mitochondrial ATP generation. Ionic gradients collapse across the neuronal membrane, due to failure of the Na–K pump. Sodium influx results in depolarization, which causes axonal release of the excitatory neurotransmitter glutamate. Glutamate is the ultimate trigger of neuronal injury due to *N*-methyl-D-aspartate receptor activation, because it allows cellular calcium influx.[169]

Second messengers in the cascade are signals during ischemia from trigger events that activate other second messengers or perpetrators of damage. Intracellular overload of calcium inhibits mitochondrial respiration. In addition, nitric oxide synthase generates NO and ROS, both of which damage DNA and increase cytokine levels. In the field, emphasis should be placed on the avoidance of factors known to exacerbate the adverse neuronal responses to cerebral ischemia: disproportionate hypotension, hypoperfusion, hyperglycemia, hypoxia, and hyperoxia.[208]

RESUSCITATION PHARMACOLOGY

The pharmacologic effects of medications are temperature dependent. The lower the temperature, the greater is the degree of protein binding. Enterohepatic circulation and renal excretion are also altered, so abnormal physiologic drug responses should be anticipated. The usual clinical scenario consists of substandard therapeutic activity while the patient is severely hypothermic, progressing to toxicity after rewarming.[178] Medications are not given orally because of decreased gastrointestinal function, and intramuscular medications are avoided because they may be erratically absorbed from vasoconstricted sites.

The pharmacologic manipulation of respiratory drive, pulse, and blood pressure is generally not indicated except in profound cases. When a relative tachycardia is not consistent with temperature depression, the possibility of hypovolemia, hypoglycemia, or a toxic ingestion should be considered. Vasopressors are potentially arrhythmogenic, might lower VF threshold, and cannot increase peripheral vascular resistance if the vasculature is already maximally constricted.[183]

If intra-arterial pressure is not consistent with the degree of hypothermia, judicious use of inotropic agents may be necessary.[229] Dopamine can be a successful adjunctive treatment.[258] Dopamine reverses cardiovascular depression under hypothermic conditions, equivalent to up to 5° C (9° F) rewarming. Dopamine decreases systemic vascular resistance at low infusion doses and increases it at high infusion rates.[229] Shock requiring vasoactive drugs is a risk factor for mortality.[296]

In severe cases, the target mean arterial pressure is around 60 mm Hg. Epinephrine and vasoconstrictors should probably be avoided.[171,174] Balancing the need for flow versus pressure, Zandstra recommends labetalol and IV nitroglycerin. This can be coupled with 2 to 4 µg/kg of dopamine, which is less arrhythmogenic than exogenous catecholamines.[322]

Epinephrine is not recommended by the American Heart Association for hypothermic cardiac arrest below 30° C (86° F). In porcine models, there is no evidence that repeated or high-dose epinephrine during hypothermic CPR improves outcome.[171,174] In the same model, a single 0.4 units/kg dose of vasopressin below 30° C (86° F) improved defibrillation success.[260]

There is conflicting evidence regarding cardiovascular function after rewarming. In some studies, there is complete reversal of cold-induced changes, whereas in another, the physiologic response to norepinephrine is paradoxical.[310]

Patients with profound hypothermia after ethanol ingestion have been resuscitated with low-dose dopamine support. In frostbite victims, however, the use of catecholamines may jeopardize the extremities.[196] Catecholamines also exacerbate preexisting occult hypokalemia.

The effect of temperature depression on the autonomic nervous system is complex. In primates, cooling produces a biphasic response in plasma catecholamine concentrations. After an initial increase, the autonomic nervous system switches off at 29° C (84.2° F),[36] suggesting that catecholamine support might be useful below that temperature. The initial rise in catecholamine levels could also be caused by acute respiratory acidosis, which stimulates the sympathetic nervous system.[26] Low-dose dopamine (1 to 5 µg/kg/min) or other catecholamine infusions are generally reserved for disproportionately severe hypotensive patients who do not respond to crystalloid resuscitation and rewarming.

Thyroid

The most dramatic Rip Van Winkle type of rude awakening from hypothermic myxedema coma was Dr. Richard Asher's patient in the 1950s. He was successfully metabolically aroused from a 7-year, 30° C (86° F) slumber. Ironically, so was a quiescent oat cell carcinoma.

Cold induces stimulation of the hypothalamic-pituitary-thyroid axis. Unless myxedema is suspected, empirical therapy is not recommended. A history of neck irradiation, radioactive

iodine, Hashimoto's thyroiditis, or surgical treatment of hyperthyroidism should heighten the clinician's suspicion of myxedema. Failure to rewarm despite an appropriate course of therapy is a further clue.[198]

Myxedema coma is usually precipitated in older adult patients with chronic hypothyroidism who are stressed by trauma, infection, anesthesia, or medication ingestion. Typical nonspecific laboratory abnormalities include hyponatremia, anemia, and liver enzyme and lipid elevations. If myxedema coma is suspected, thyroid function studies, including serum thyroxine (T_4) by radioimmunoassay, triiodothyronine (T_3) resin uptake, and thyroid-stimulating hormone level, should be obtained and serum cortisol level measured.

Given the appropriate index of suspicion, the physician should administer 250 to 500 µg levothyroxine (T_4) intravenously over several minutes without waiting for confirmatory laboratory results. Daily injections of 100 µg are required for 5 to 7 days. The clinician should consider adding at least 100 to 250 mg of hydrocortisone to the first 3 L of intravenous fluid. Absorption of T_4 is erratic if the drug is given orally or intramuscularly. The onset of action of T_3 is more rapid, which jeopardizes cardiovascular stability; therefore, T_3 is avoided in acute replacement therapy. The onset of action of T_4 is 6 to 12 hours, evidenced by continuous improvement of the vital signs during rewarming. Up to one half of the T_4 is eventually converted by the peripheral tissues into T_3.

Steroids

Acute cold stress and many coexisting disease processes stimulate cortisol secretion. The free active fraction of cortisol decreases as the temperature drops because of increased protein binding, and cortisol utilization is similarly decreased. The increase in adrenocorticotropic hormone (ACTH) and adrenal steroid secretion may also be a neurogenic or an emotional response in the conscious subject to an unpleasant environment.

Experimentally, inhibition of ACTH secretion during hypothermia is mediated by decreased hypothalamic secretion of arginine vasopressin. This decreases pituitary responsiveness to corticotropin-releasing factor, inhibiting corticotropin release. Thus, exogenous arginine vasopressin could prove helpful during rewarming.

Cold exposure also induces adrenal unresponsiveness to ACTH. As a result, false diagnosis of decreased adrenal reserve is possible and does not represent functional adrenal insufficiency, because ACTH levels return to normal after rewarming. Serum cortisol levels are commonly elevated. Secondary adrenal insufficiency resulting from panhypopituitarism may also coexist with myxedema. Empirical administration of steroids is indicated if hypoadrenocorticism is suspected on the basis of a previous history of steroid dependence, suggestive physical findings, or an inexplicable failure to rewarm.

The use of narcotic antagonists in hypothermia is reported. Naloxone may reduce the severity of hypothermia in drug overdoses and in spinal shock and appears to have activity at the µ-opioid receptor sites.[50]

RESUSCITATION COMPLICATIONS

Atrial Arrhythmias

All atrial arrhythmias, including atrial fibrillation, should have a slow ventricular response with the temperature depression. Atrial fibrillation is commonly noted below 32° C (89.6° F) in accidental hypothermia victims. Atrial fibrillation usually converts spontaneously during rewarming, and digitalization is not warranted. Electrophysiologic studies show that the interval prolongation present on His bundle electrocardiography is unresponsive to atropine. Mesenteric embolization is a potential hazard when the rhythm converts back to sinus rhythm. Hypothermia renders the negative inotropic effects of calcium channel blockers redundant.

In summary, all new atrial arrhythmias usually convert spontaneously during rewarming and should be considered innocent. Attention is directed toward correcting acid–base, fluid, and electrolyte imbalances and avoiding administration of atrial antiarrhythmics.

Ventricular Arrhythmias

Because preexisting chronic ventricular ectopy may be suppressed in a cold heart, the physician noting ectopy during rewarming is placed in a quandary. The history from the hypothermic patient may be unproductive, and the past cardiac history is often unavailable.

Transient ventricular arrhythmias should generally be left untreated. In one study of 22 continuously monitored patients with hypothermia, supraventricular arrhythmias were common (nine cases) and benign.[242] Ventricular extrasystoles developed in 10 patients, but none experienced ventricular tachycardia or VF during rewarming. The terminal rhythm in the eight who died while being monitored was asystole and not VF. The energetics of the fibrillating hypothermic ventricle suggest that asystole may consume less energy and be more protective.

Pharmacologic options are limited, because hypothermia induces complex physiologic changes that result in abnormal responses.[319] Drug metabolism and excretion are both progressively decreased. In normothermia, class IA ventricular antiarrhythmics have negative inotropic and indirect anticholinergic effects, and they moderately decrease conduction velocity (Box 5-10). Procainamide increases the incidence of VF during hypothermia. Quinidine has been useful during induced profound hypothermia, preventing VF during cardiac manipulation at 25° to 30° C (77° to 86° F). No reports detail the effects of disopyramide.

The role of the class IB agent lidocaine for prophylaxis or treatment is unresolved. In animal studies, lidocaine and propranolol have minimal hemodynamic effects in hypothermia. If normothermic effects persisted during hypothermia, the class IB agents would appear attractive because they minimally slow conduction while shortening the action-potential duration (APD).

The class III agent bretylium tosylate, a unique bromobenzyl quaternary ammonium compound, has been effective in several animal studies and ineffective in others.[69,282] It is not commercially available. This class of agents seems most ideal pharmacologically because it possesses direct antifibrillatory properties. The ability to prolong the APD is temperature dependent. Ideally, a drug would lengthen the APD only in warmer regions to reduce dispersion (Box 5-11). Two cases of chemical ventricular defibrillation after infusion of bretylium (10 mg/kg) in accidental hypothermia are reported.[48,168]

In the first study to evaluate the effects of bretylium administered after induction of hypothermia, Murphy and coworkers[218] noted that only one of 11 dogs given bretylium (mean, 40.5 mg/kg) before five invasive maneuvers developed

Box 5-10. Antiarrhythmic Agents

Class I. Sodium Channel Blockers
- IA. Conduction and depolarization moderately slowed, and action potential duration (APD) and repolarization prolonged
  - Disopyramide
  - Procainamide
  - Quinidine
- IB. Conduction and depolarization minimally slowed, and APD and repolarization shortened
  - Lidocaine
  - Mexiletine
  - Moricizine
  - Phenytoin
  - Tocainide
- Conduction and depolarization markedly slowed, and APD and repolarization prolonged
  - Encainide
  - Flecainide

Class II. β-Adrenergic Blockers

Class III. Antifibrillatory Properties
- APD prolonged
  - Amiodarone
  - Bretylium
  - D-Sotalol

Class IV. Calcium Channel Blockers
- Diltiazem
- Verapamil

Unclassified
- Adenosine
- Magnesium sulfate

Box 5-11. Antiarrhythmic Characteristics

The ideal ventricular antiarrhythmic would do the following:
- Cause no further decrease in conduction velocity.
- Shorten the action potential duration (APD).
- Lengthen the APD only in warmer regions of the myocardium to reduce dispersion.
- Possess direct antifibrillatory properties.

VF. No dog, including control subjects, showed fibrillation during endotracheal intubation. Of note in discussions regarding prophylaxis, three of the 11 dogs converted to VF during the drug infusion. The effect of bretylium on plasma catecholamines and electrically induced arrhythmias has also been studied. Because catecholamine levels increase during cooling, the demonstrated protection appears to result from alteration of electrophysiologic properties of the cardiac tissues.

Amiodarone is another class III drug that possesses direct antifibrillatory activity. In a canine model of severe hypothermic VF, neither bretylium nor amiodarone improved resuscitation.[282] The safety of the class III antiarrhythmic agent D-sotalol is problematic.[18] It has temperature-dependent effectiveness and lengthens prolonged action potentials more efficiently at long pacing cycle lengths. Also of note, magnesium sulfate at a dose of 100 mg/kg IV can spontaneously defibrillate most patients on CPB at 30° C (86° F) with induced hypothermia.

Emergency transvenous intracardiac pacing of bradyarrhythmias is extremely risky with cold hearts because it commonly precipitates VF. New arrhythmias that develop after rewarming may require pacing on rare occasions. Transcutaneous pacing (TCP) with low-resistance electrodes seems far preferable before stabilization. In a canine model, TCP restored and maintained hemodynamic stability and rewarmed hypothermic animals twice as rapidly as it did the controls.[60] In summary, there are no commercially available ventricular antiarrhythmics proven safe and effective in accidental hypothermia.

Sepsis

In hypothermia, the classic signs of infection, including erythema and fever, are absent.[200] Rigors and shakes resemble shivering. The initial history, physical, and laboratory data are often unreliable, so repeated evaluations and comprehensive culturing are mandatory in the ED. The 10% subset of patients with sepsis syndrome who manifest a hypothermic response have a significantly increased frequency of shock and death, and this secondary hypothermia does not appear to be protective.

Hypothermia compromises host defenses and results in serious bacterial infections. These significant infections can be accompanied by minimal inflammatory response. Some common causative organisms include gram-negative bacteria, gram-positive cocci, oral anaerobes, and Enterobacteriaceae.[188]

The core endotoxin components of gram-negative bacteria normally signal macrophages. At a normal or elevated temperature, active cytokine triggers include tumor necrosis factor, interleukin-1, and interleukin-6. The potential role of anti-endotoxin monoclonal antibodies in patients with accidental but not cytokine-induced hypothermia is unresolved. Bone marrow release and circulation of neutrophils are compromised for up to 12 hours. In addition, human and porcine neutrophils are susceptible to hypothermia. In vitro, neutrophil migration and bacterial phagocytosis are reduced at 29° C (84.2° F). Neutrophilic extermination of various bacteria, including *Staphylococcus aureus* and *Streptococcus faecalis,* is also impaired.

Acquired neutrophil dysfunction occurs. In addition, hypothermia is associated with decreased neutrophil levels in near-drowned children. As a clinical demonstration of the importance of these factors, therapeutic maintenance of hypothermia to control cerebral edema in near drowning is abandoned because of the substantial incidence of infectious complications.

The incidence of infection varies dramatically with the victim's age and the clinical series reported.[188,312] In one large group of infants, over half were septic. Although there were no reliable indicators of infection, some suggestive clues emerged. Serum glucose and leukocyte abnormalities, anemia, uremia, and bradycardia were often identified. In addition to *Staphylococcus* and *Streptococcus,* the predominant organisms were *Haemophilus* and Enterobacteriaceae.

Lung infections, usually in the right upper lobe, were reported in many hypothermic infants by El-Radhi and colleagues.[70] Evaluation of the gastric aspirate was another diagnostic

predictor of sepsis in the majority of infected infants. In several studies, sepsis was found in a large percentage of hypothermic infants. In this age group, empirical broad-spectrum antibiotics are warranted.

In adults, the incidence of infection ranges from less than 1% to over 40%, depending on patient selection criteria.[53] In the urban series of Lewin and coworkers,[188] serious soft tissue or pulmonary infections were present in half the patients. Occult bacteremia was present in less than 1% of White's series[312] of 102 patients, and results were negative in all of the 46 lumbar punctures. In other studies of hypothermic older adult patients admitted to hospitals, most have had evidence of probable or definite infections.[54]

Invasive hemodynamic monitoring, although risky, may suggest an infection. Before patients are rewarmed, the combination of an elevated cardiac index and decreased systemic vascular resistance should suggest bacteremia. Right heart catheterization is more hazardous in hypothermia than during normothermia.

In summary, unlike children and older adults, most previously healthy young adults do not need empirical antibiotic prophylaxis. Nevertheless, treatment indications should be liberalized from normothermia. They should include failure to rewarm, and any suspicion or evidence of aspiration, myositis, chest film infiltrate, bacteriuria, or persistent altered mental status. In choosing broad-spectrum coverage, the physician should consider altered drug interactions, volumes of distribution, protein binding, hepatic metabolism, and renal excretion.

FORENSIC PATHOLOGY

Macromorphologic and micromorphologic lesions are variable and nonspecific in hypothermia, and there is no single pathognomonic finding at autopsy.[190] Establishing hypothermia as the primary cause of death requires an adequate history of exposure and the absence of other lethal findings at necropsy.[4] Unnatural deaths in nursing home patients, for example, may be significantly underreported for these reasons.[43]

Macroscopic skin changes can suggest the diagnosis. Hyperemia of the dorsa of the hands and knees is commonly found. Nonpathognomonic findings have been identified in the pancreas, lungs, and heart. Pancreatic findings include fat necrosis, aseptic pancreatitis, and hemorrhage. Pulmonary changes consist of intra-alveolar, interstitial, and intrabronchial hemorrhages.

An eye that has been directly exposed to the environment could be a chemical indicator of both the environmental and victim temperature at the time of death. Vitreous humor chemistry profiles at autopsy can reveal that glucose concentration and total carbon dioxide content vary inversely with temperature, with values significantly higher in the winter. An elevated vitreous glucose in a nondiabetic victim should suggest hypothermia.

The total urinary catecholamine content, particularly epinephrine, was high in one group of casualties known to be hypothermic. Erosions of the gastric mucosa, termed Wischnewsky spots, are also frequently found.[285,317] In addition, exposure to extreme cold should be suspected when unusual intravascular hemolysis, which is seen after freezing of blood, is observed in a corpse. Fatty degeneration in renal tubule epithelia may help confirm the diagnosis.[239]

Prevention

To function optimally "as the water stiffens" requires an understanding of the principles of heat conservation and loss.[155] Well-trained and educated urban adults can participate in prolonged Arctic maneuvers safely.[266,267,288]

To maintain core temperature in the narrow band necessary for peak functioning in cold environments, appropriate adaptive behavioral responses are essential.[130] Autonomic and endocrinologic mechanisms are only supplemental.

Studies of human cold adaptation reach highly variable conclusions. Explanations for these discrepancies include changes in core and shell temperatures and in metabolic rates before and after cold adaptation. Hypothermic, insulative, isometabolic cold adaptation may be associated with local cold adaptation of the extremities.[320]

Excellent physical conditioning with adequate rest and nutrition is paramount.[22] Hikers and skiers must be accompanied by a partner and should wear effective thermal insulation. Wet inner garments must be changed promptly. Persons who exert themselves, including long-distance skiers, should switch garments depending on current exertional heat production.[131,133] Because dehydration must be avoided, drinking from a cold stream is preferable to snow ingestion. Significant energy is needed to convert ice at 0°C (32°F) into water at 0°C.

All areas with a large surface area-to-volume ratio should be well insulated.[286] Excellent synthetic insulating materials include Gore-Tex, Flectalon, Thinsulate, and taslanized nylon.

Under certain circumstances, insidious hypothermia may develop during exposure to cold water because of the effects of increased insulation on compensatory physiologic events.[9,156] The army mnemonic COLD, in reference to insulation with clothing, means *clean, open* during exercise to avoid sweating, *loose* layers to retain heat, and *dry* to limit conductive heat losses. For a complete discussion of preventive measures, refer to Chapters 8 and 9.

Prevention of urban accidental hypothermia requires continuous public education. For example, the optimal safe indoor temperature recommendation for older adults has risen to 21.1°C (70°F). Energy assistance and temporary sheltering are effective measures, and selective heating of sleeping quarters and use of electric blankets are economical suggestions. Prewarming bed and bedroom at night may be the best overall advice to older adults.

OUTCOME

Partially in reaction to the dramatic reports of reanimations, the historic standard of care has been that "no one is dead until they are warm and dead." Clearly, some victims are indeed "cold and dead," and it would be useful to safely identify them.

Survival is difficult to predict because human physiologic responses to temperature depression vary so widely.[209,236] The type and severity of the underlying or precipitating disease process are two determinants. Age extremes, although not statistically correlated with survival, are commonly associated with severe illnesses. In a multicenter survey, however, there were no significant age differences in mortality.[53]

Sex, trauma, infection, and toxin ingestions affected survival differently in multiple, uncontrolled clinical studies.[127] There were no clinically significant differences in male versus

female profiles in the multicenter survey.[53] From a large hypothermia database, a hypothermia outcome score could enable multiple observers at different sites to assess treatment modalities and outcome predictors.[249] Prehospital cardiac arrest, low or absent blood pressure reading, elevated BUN, and the need for either endotracheal or nasogastric intubation in the ED were significant predictors of outcome after multivariate analysis.[49,158]

In a multiple regression analysis of 234 cases in Swiss clinics,[195] the biggest negative survival factors were asphyxia, slow rate of cooling, invasive rewarming, asystole, and development of pulmonary edema or adult respiratory distress syndrome. The largest single-hospital series of adult hypothermic cardiac arrest patients rewarmed with CPB is reported from Finland. Patients with cold exposure or immersion without suffocation or asphyxia tolerated prolonged CPR before CPB.[270] Positive predictors of survival include rapid cooling rate, presence of VF during cardiac arrest, and narcotic or ethanol intoxication. In a study of 29 patients below 30°C (86°F), mode of cooling was the only independent risk factor.[236]

In a study of CPB survivors, 15 of the 32 patients are long-term survivors.[304] Their neuropsychological functioning after prolonged prehospital circulatory arrest is very encouraging. These patients were not asphyxiated before becoming hypothermic. Vretenar and colleagues[301] reviewed the literature regarding the outcomes of 68 other patients resuscitated with CPB. The survival rate was 60%, and the coldest survivor had been 15°C (59°F). In a series of 26 patients given extracorporeal rewarming, those with nonasphyxiated deep accidental hypothermia had a reasonable prognosis.[74]

There is a need for a valid triage marker of death, because vital organ damage is difficult to predict. In one retrospective analysis of primarily avalanche burial victims, extreme hyperkalemia was noted on initial examination, and resuscitation proved fruitless.[254] In the Mt. Hood tragedy, the nonsurvivors were also hyperkalemic (serum potassium greater than 10 mmol/L).[114] Although a serum potassium of 10 mmol/L appears to be a reasonable ceiling for viability, a child at 14.2°C with a potassium level of 11.8 mmol/L did well.[61] In both of these reports, asphyxia and compression injury may have been contributory. Other indicators of a grave prognosis include a core temperature below 12°C (53.6°F), arterial pH below 6.5, or evidence of intravascular thrombosis (direct visualization; fibrinogen below 50 mg%).

The references for this chapter can be found on the accompanying DVD-ROM.

6 Immersion in Cold Water

Gordon G. Giesbrecht and Alan M. Steinman

Immersion in cold water is a hazard for anyone who participates in recreational, commercial, or military activities in the oceans, lakes, and streams of all but the tropical regions of the world. Recreational aquatic activities include swimming, fishing, sailing, power boating, ocean kayaking, white-water rafting, canoeing, ocean surfing, wind surfing, water skiing, diving, hunting, and using personal watercraft. In addition, although snowmobiling is not technically a water sport, it can involve cold-water exposure as a result of accidental entry into lakes and streams.[1] Commercial activities involving water include fishing, shipping, offshore oil drilling, and diving. Military operations over cold water include Coast Guard (Fig. 6-1), Navy, and Marine Corps missions. Army, Air Force, and Marine Corps forces may also encounter cold-water exposure during winter operations on land.

The definition of cold water is variable (Box 6-1). The temperature of thermally neutral water, in which heat loss balances heat production for a nude subject at rest (i.e., not shivering), is approximately 33° to 35°C (91.4° to 95°F).[18,20] Hypothermia eventually results from immersion in water below this temperature. For practical purposes, significant risk of immersion hypothermia usually begins in water colder than 25°C (77°F).[110,158,159] Table 6-1 shows the variation of water temperatures throughout the year at various sites in North America,[147] and Figure 6-2 shows typical world-wide sea-surface temperatures for April using data from 2005.[148] Using 25°C (77°F) as the definition of cold water, the risk of immersion hypothermia in North America is nearly universal during most of the year.

Cold-water immersion is associated with two significant medical emergencies: near-drowning and hypothermia. This

female profiles in the multicenter survey.[53] From a large hypothermia database, a hypothermia outcome score could enable multiple observers at different sites to assess treatment modalities and outcome predictors.[249] Prehospital cardiac arrest, low or absent blood pressure reading, elevated BUN, and the need for either endotracheal or nasogastric intubation in the ED were significant predictors of outcome after multivariate analysis.[49,158]

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Cold-water immersion is associated with two significant medical emergencies: near-drowning and hypothermia. This

chapter will discuss the physiologic responses to and treatment of immersion hypothermia. In addition, this chapter will discuss the risk of near-drowning with respect to the physiologic consequences of sudden immersion in cold water and the problems of survival in rough seas. Chapter 5 discusses land-based hypothermia, Chapter 68 provides a more complete discussion of drowning, and Chapter 70 discusses diving injuries.

HISTORY AND EPIDEMIOLOGY

The history of human association with the sea and with inland waters provides abundant examples of the effects of accidental cold-water immersion. The following examples demonstrate the scope of the problem.

Perhaps the most famous occurrence was the sinking of the *Titanic* in 1912. After striking an iceberg at approximately 11:40 PM on April 14, the ship sank in calm seas. Water temperature was near 0° C (32° F). Of the 2201 people on board, only 712 were rescued, all from the ship's lifeboats. The remaining 1489 people died in the water, despite the arrival of a rescue vessel within 2 hours. Nearly all of these people were wearing life preservers, yet the cause of death was officially listed as drowning.[116] More likely the cause of death was immersion hypothermia; survivors reported hearing victims' screams for periods long enough to result in severe hypothermia (i.e., longer than 1 hour).[98,107]

In 1963, the cruise ship *Lakonia* caught fire and sank in approximately 18° C (64.4° F) water off the coast of Madeira in fairly calm seas. Two hundred people abandoned ship into the sea; all wore self-righting life jackets but 120 died. The cause

Figure 6-1. U.S. Coast Guard rescue boat in rough seas. (Photo courtesy Allen Steinman, U.S. Coast Guard.)

Box 6-1. The Official Canadian Temperature Conversion Chart

The authors of this chapter hail from two countries that view and measure temperature differently. The United States uses °F (Fahrenheit), whereas that other country up north uses °C (Celsius). The author from up north has provided a conversion chart to allow neighbors from both sides of the border to get on the same thermal wavelength.

10° C (50° F)	Americans shiver uncontrollably.	Canadians plant gardens.
0° C (32° F)	American water freezes.	Canadian water gets thicker.
–18° C (0° F)	New York City landlords finally turn on the heat.	Canadians have the last cookout of the season.
–51° C (–60° F)	Mount St. Helens freezes.	Canadian Girl Guides sell cookies door-to-door.
–114° C (–173° F)	Ethyl alcohol freezes.	Canadians get frustrated when they can't thaw the keg.
–273° C (–460° F)	Absolute zero; all American atomic motion stops.	Canadians start saying, "Cold, eh?"
–295° C (–500° F)	Hell freezes over.	A U.S. hockey team beats Canada in an Olympic gold medal game.

TABLE 6-1. Mean Water Temperatures (°C)

SITE	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
Kodiak, AK	**0**	**0**	**2**	**4**	**7**	**9**	**12**	**12**	**10**	**7**	**4**	**2**
Victoria, BC	**7**	**7**	**7**	**9**	**10**	**11**	**11**	**11**	**11**	**10**	**9**	**8**
Astoria, OR	**4**	**5**	**7**	**10**	**13**	**16**	**19**	**19**	**17**	**13**	**9**	**5**
San Francisco, CA	**11**	**11**	**12**	**12**	**12**	**12**	**13**	**13**	**12**	**12**	**11**	**11**
San Diego, CA	**14**	**14**	**15**	**16**	**17**	**18**	**20**	**20**	**20**	**18**	**16**	**15**
Mobile, AL	**10**	**10**	**14**	**21**	**23**	28	29	29	27	**23**	**19**	**14**
Miami, FL	**21**	**21**	**23**	25	27	29	30	30	29	27	25	**23**
Norfolk, VA	**17**	**16**	**15**	**18**	**20**	24	26	26	25	25	**21**	**19**
Cape May, NJ	**3**	**3**	**5**	**10**	**14**	**19**	**23**	**23**	**21**	**17**	**11**	**6**
Traverse City, MI	**2**	**1**	**1**	**1**	**1**	**3**	**5**	**10**	**10**	**6**	**5**	**4**
Puerto Rico	26	26	26	26	26	27	27	27	28	27	27	26
Honolulu, HI	**24**	**24**	**24**	**24**	25	25	26	26	27	26	25	25

Temperatures less than 25° C are in boldface print.

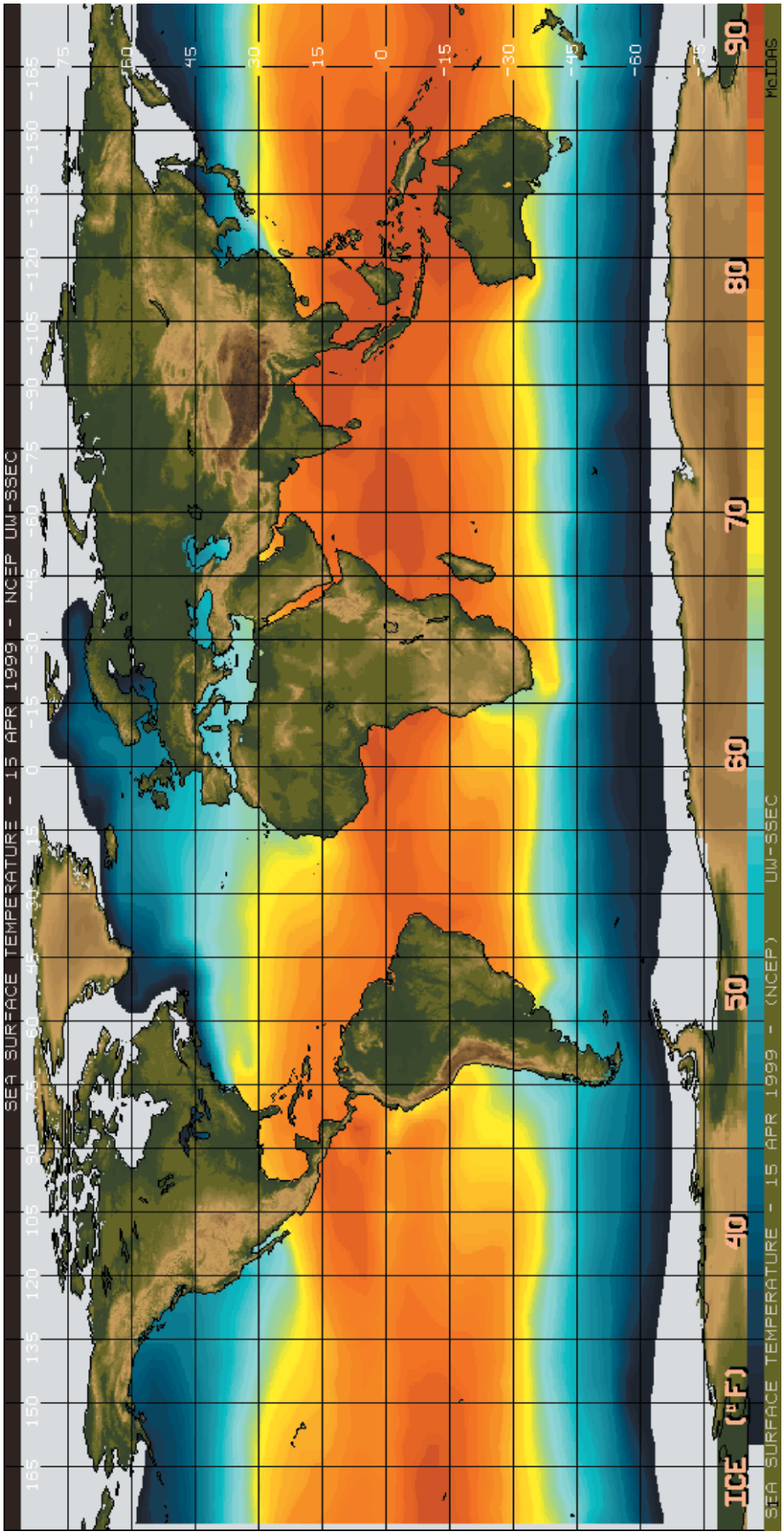


Figure 6-2. World-wide sea surface temperatures for April 2005. (From Space Science and Engineering Center, University of Wisconsin-Madison: National Center for Environmental Prediction: Sea Surface Temperature—25 April 2005, with permission. Available at http://www.ssec.wisc.edu/data/sst/latest_sst.gif.)

of death for most victims was hypothermia. Because rescuers reported that small waves washed over the faces of unconscious people, drowning was probably the terminal event.[97]

In 1994, the "roll on/roll off" vehicle and passenger ferry *Estonia* took on water and rapidly sank in stormy weather in the northern Baltic Sea. Of the 989 people on board, only 137 survived. Water temperature was 8° C (46.4° F). Only 300 passengers were able to get to the outer decks of the vessel before it sank stern first. Most of those rescued were in lifeboats. All the others either drowned in the heavy seas or went down with the vessel.[92]

Recreational Activities and Cold Water

Recreational aquatic activities are a major source of immersion incidents. Coast Guard statistics indicate that in 1997, there were 8047 recreational boating accidents, which produced 821 deaths, 4555 injuries, and over $29 million in property damage. Over two thirds of these recreational boating fatalities involved exposure of the occupants to water as a result of falling overboard or because the vessels capsized, flooded, swamped, or sank. Hypothermia or drowning accounted for 74.3% of these fatalities.[176]

A Canadian Red Cross report stated that from 1991 to 2000, there were 5900 water-related deaths in Canada. Of the recreational boating drownings, 283 (21%) occurred in "extremely cold" water of below 10° C (50° F). Of the 14% of all boating drownings in which the victims were wearing a personal flotation device (PFD), most were in cold water and death was due to hypothermia. In addition, 232 snowmobilers drowned after breaking through the ice, and 218 people died after falling through the ice during other activities.

The following events illustrate the potential problems presented by immersion hypothermia as it relates to recreational boating and other activities: In December 1996, three young adults died from hypothermia after their boat sank in 7.3° C (45.14° F) water off East Moriches, New York. The three were experienced boaters and well versed in cold-water survival and safety. Because the weather was unusual for a winter's day in the Northeast (calm winds and seas with an air temperature of 7.3° C [45.14° F]), the three left their safety gear and protective clothing at home. When the boat sank in the cold seas, their lack of immersion protective clothing led to their deaths from hypothermia.[9]

In October 1997, three persons died from immersion hypothermia when their 15-foot bass boat was swamped by the wakes of passing boats in Lake Mille Lacs, Minnesota. The four-person crew was forced to spend the night hanging onto their overturned boat in 15.5° C (59.9° F) water. They did not have a radio to call for assistance. Although they were wearing life jackets, they did not have immersion protective clothing for insulation against hypothermia.[127]

In the winter of 1997/1998, four people died when their snowmobile broke through the thin ice of Lake Scugog, near Toronto, Ontario. The deaths occurred, ironically, during Snowmobile Safety Week in North America. The Canadian Safety Council reported that in 1997, 101 people were killed in snowmobile mishaps, the majority of them from drowning and hypothermia after crashing through ice on frozen rivers and lakes.[1]

In the winter of 1988, two men died from hypothermia when their nine-man rowing scull sank in a winter storm on a small lake in western Canada. The nine rowers were immersed in 4° C (39.2° F) water, struggling to hang on to their overturned vessel in 0.6- to 1.0-m (2.0- to 3.3 ft) waves and 30-km/hr winds for over 50 minutes before rescuers could reach them. One man drowned just before rescue; the others were all conscious when pulled from the water. However, during the 13-minute boat transport to shore, three of the thinnest victims lost consciousness and one of these men suffered cardiopulmonary arrest. His core temperature in the hospital, 44 minutes after rescue, was 23.4° C (74.1° F), and subsequent resuscitation efforts were unsuccessful. The remaining seven survivors were resuscitated from core temperatures as low as 25° C (77° F). This case illustrates the rapidity of onset of immersion hypothermia in lean subjects, and the potentially serious consequences of cardiovascular instability, circum-rescue collapse, and post-rescue core temperature afterdrop in immersion hypothermia.[55]

Commercial Activities on Cold Water

Numerous examples of maritime occupational accidents exist. In 1982, the offshore mobile oil-drilling platform *Ocean Ranger* collapsed in mountainous seas near Newfoundland, Canada. Water temperature was –1° C (30° F). Eighty-four workers were plunged into the water, and despite the presence of a rescue vessel, all died. Immersion hypothermia was a significant factor in these deaths, although the inability of the workers to combat the rough seas and maintain airway freeboard (the distance between the water surface and the mouth) was also a major problem.[111]

Fishing vessel mishaps are a common cause of immersion hypothermia. The following incidents illustrate the problems facing the commercial fishing industry:

In December 2004, the fishing vessel *Northern Edge* capsized near Nantucket, Massachusetts, in 4° C (39.2° F) water and 3- to 4-m (9.8- to 13.1-ft) seas. Five of the six crewmen lost their lives.[179] In several similar cases in the same region, seven fishermen, on three different vessels, lost their lives in just a 30-day period in the winter months of 2004/2005.[175] In June 2003, the charter fishing vessel *Taki Tooo,* carrying 17 passengers and crew, capsized in the turbulent waters of the Tillamook River bar on the Oregon coast with the loss of 11 lives. Water temperature was 10° C (50° F).[177] In April 2001, the fishing vessel *Arctic Rose* sank in the Bering Sea of Alaska with the loss of 15 crewmen.[178] The vessel sank so quickly that there was no time for either a distress call or for the crewmen to don survival suits.

A 1999 U.S. Coast Guard Fishing Vessel Casualty Task Force report[175] found that in just 2 months (December 1998 and January 1999), 20 commercial fishing vessels were lost at sea. In these mishaps, 21 persons on board died, mainly from exposure to cold water because the vessel sank or capsized, or the people fell overboard. From 1994 through January 1999, U.S. Coast Guard statistics show that 396 fishermen lost their lives while fishing. Of these, 298 died from cold-water immersion secondary to falling overboard, or to the vessels' sinking, capsizing, or flooding. In 1998, the death rate for fishermen was 179 per 100,000 workers, a rate 16 times higher than that for firefighters or for police officers.

Military Activities and Cold Water

The extensive use of combat ships and aircraft in World War II, particularly in the northern Pacific and North Atlantic oceans, provided many examples of accidental immersion in cold water. Molnar reviewed several hundred of these cases.[121] Among them was the following: "A ship was rammed, and it sank in 3 minutes. Thirty survivors were picked up from rafts after exposure of 1.5 to 4 hours. Some drowned and others died of expo-

sure because the water was 4°C (39°F) with high seas and a wind velocity of scale 7. Some of the survivors who held on to ropes couldn't let go and rescuers had to cut frozen rope to release them. It appears miraculous how the survivors could have endured such cold water. Most of those who were rescued were in an unconscious state and, when they became conscious complained of numbness of extremities and hands."

The Falklands War in 1982 provided further examples of immersion hypothermia related to naval combat. The Argentine cruiser *General Belgrano* sank in approximately 5°C (41°F) water, resulting in the deaths of many sailors. Some of the deaths from cold occurred even after the sailors had managed to emerge from the water into life rafts.

Maritime military operations are not the only source of hypothermia casualties in the armed forces: land operations, either in combat or in training, also have immersion hypothermia risks. In March 1995, four U.S. Army Rangers died and four others were hospitalized during a training mission in a Florida swamp. The water at the exercise site was deeper and colder than was customary for this exercise. Consequently the Rangers were forced to spend more than 6 hours in cold, wet conditions, much of the time partially immersed in the 15°C (59°F) water. Night conditions and difficulties encountered in the medical evacuation of the accident victims contributed to the hypothermia deaths.[19,181]

In March 2001, two U.S. Coast Guard crewmen drowned following immersion in cold water when their four-man vessel capsized in the waves of the Niagara River bar in Lake Ontario. Water temperature was 2°C (36°F). All of the crewmen were wearing drysuits with insulation, and lifejackets, but they wore no insulated head gear, and leakage of cold water into two of the suits resulted in severe immersion hypothermia and subsequent drowning.[180]

The relatively high fatality rate from accidental immersion in cold water in various military, commercial, and recreational settings has stimulated technologic advances in protective clothing and rescue devices. The current availability of commonplace items of survival equipment (such as wetsuits, drysuits, survival suits, and inflatable life rafts) is primarily a response to the needs of people who work in cold-water environments. The value of various types of protective clothing is examined later in this chapter.

In summary, numerous case histories and statistical evidence document the significance of cold-water immersion as a cause of the environmental emergencies of drowning and hypothermia. Although drowning is relatively easy to prevent (for example, through the use of PFDs, water safety training, or restriction of alcohol use), hypothermia is not. Hypothermia is now more widely recognized than in the past, but prevention of immersion hypothermia is still a difficult and often expensive proposition. Therefore, in regions of cold water, which includes most of the North American continent, cold-water safety, the knowledge of cold-water risks, and the use of appropriate flotation devices and protective clothing are essential.

PHYSIOLOGIC RESPONSES TO COLD-WATER IMMERSION (HEAD ABOVE WATER)

The effect of sudden cold-water immersion on the human is poorly understood both by the general public and by medical and rescue personnel. Many individuals believe that an individual who falls in ice water will die of hypothermia within minutes, even if dressed in winter clothing. In a series of surveys of continuing medical education attendees (unpublished results), 75% of respondents felt that under these conditions, a victim would experience life-threatening hypothermia in less than 30 minutes (only 4% of responses had the correct answer). As discussed later, most adults could survive for an hour or more if they take appropriate action. The improper assumption of impending death only serves to induce panic, poor decisions, and exhaustion from thrashing about, causing problems for both the primary victims and those who are trying to rescue them.

The primary pathophysiologic effects of hypothermia are a decrease in tissue metabolism and a gradual inhibition of neural transmission and control. However, in the *initial* stages of cooling of an intact, conscious victim, secondary responses to decreased skin temperature predominate. Sudden immersion in cold water results in an immediate decline in skin temperature, which, in turn, initiates shivering thermogenesis with increases in metabolism ($\dot{V}O_2$), ventilation ($\dot{V}E$), heart rate (HR), cardiac output (CO), and mean arterial pressure (MAP). As body temperature declines and shivering ceases, $\dot{V}O_2$, HR, MAP, and CO decrease proportionately with the fall in core temperature, whereas hematocrit and total peripheral resistance increase. Renal diuresis and extravascular fluid shifts can lead to a considerable loss of intravascular volume, thus decreasing systemic perfusion. A more detailed description of the pathophysiologic effects of systemic hypothermia is provided in Chapter 5. These paragraphs will describe the pragmatic effects of the pathophysiologic changes accompanying cold-water immersion and their impact on survival.

The body's responses and the mechanisms of death during cold-water immersion can be divided into four phases: (1) initial immersion and the cold-shock response, (2) short-term immersion and loss of performance (cold incapacitation), (3) long-term immersion and the onset of hypothermia, and (4) circum-rescue collapse just prior to, during, or after rescue. Each phase is accompanied by specific survival hazards for the immersion victim from a variety of pathophysiologic mechanisms. Deaths have occurred during all four phases of cold-water immersion.

Phase 1: Cold Shock Response (Initial Immersion, 0 to 2 Minutes)

The cold-shock response occurs within the first few minutes of cold-water immersion and depends on the extent and rate of skin cooling. The responses are generally those affecting the respiratory system and those affecting the heart and the body's metabolism (Fig. 6-3).[36,171] Rapid skin cooling initiates an immediate gasp response, the inability to breath-hold, and hyperventilation. The gasp response may cause drowning if the head is submersed during the initial entry into cold water. Subsequent inability to breath-hold may further potentiate drowning in high seas. Finally, subsequent hyperventilation normally diminishes within seconds to minutes but could be increased and exaggerated indefinitely as a result of emotional stress and panic. Uncontrolled hyperventilation can cause numbness, muscle weakness, or even fainting, leading to drowning. Any of these respiratory responses can lead to aspiration of water into the lungs, panic, and subsequent drowning.

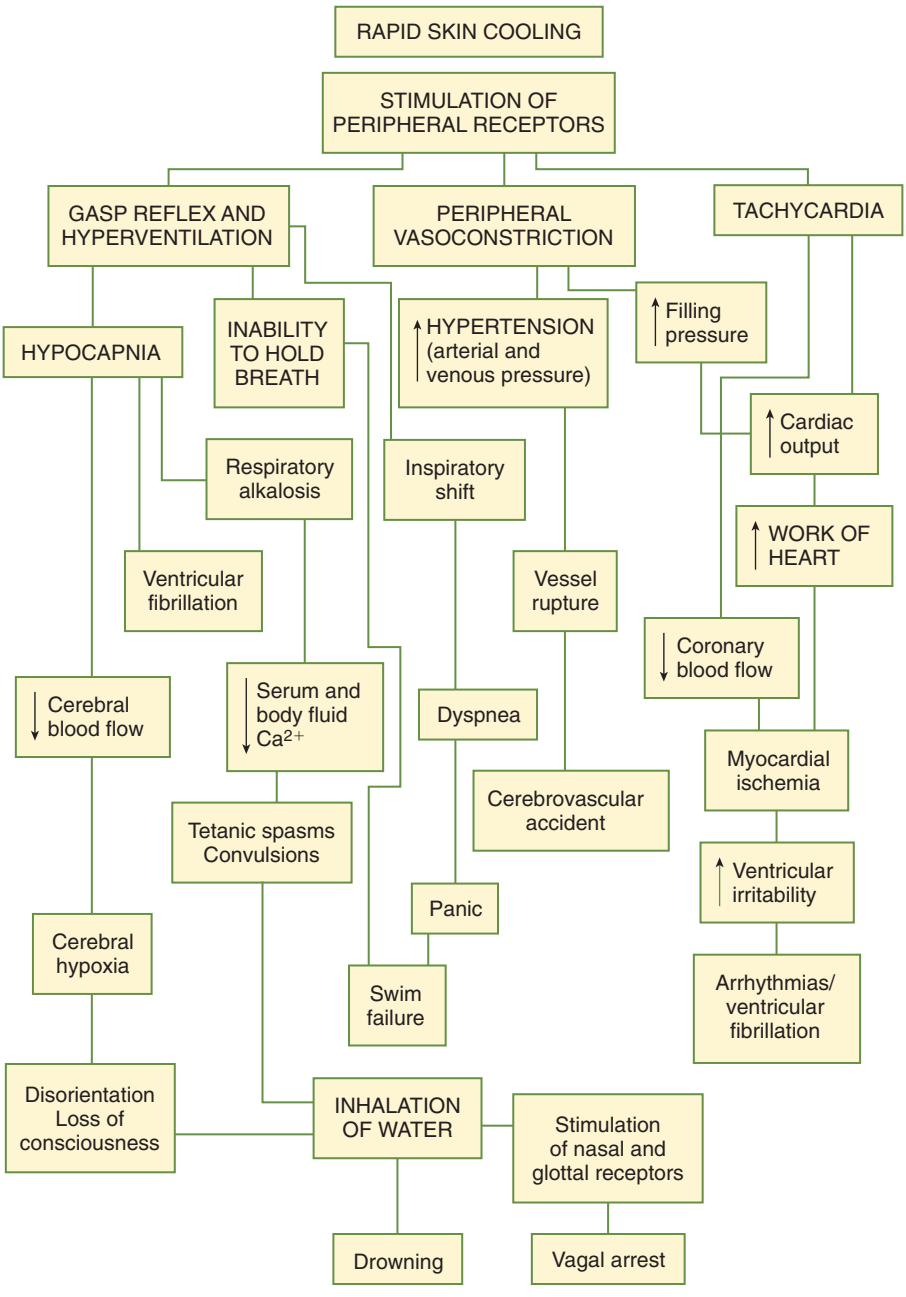


Figure 6-3. Possible cold-shock responses. ↑, increase; ↓, decrease. (From Edmonds C, Lowry C, Pennefather J: Diving and Subaquatic Medicine. 3rd ed. Oxford, Butterworth-Heinemann, 1992, with permission.)

Skin cooling also initiates peripheral vasoconstriction as well as increased CO, HR, and arterial blood pressure. The increased workload on the heart may lead to myocardial ischemia and arrhythmias, including ventricular fibrillation. Thus, sudden death can occur either immediately or within a matter of minutes after immersion (i.e., due to syncope or convulsions leading to drowning, vagal arrest of the heart, and ventricular fibrillation) in susceptible individuals.[25,44,99,115,163]

Cold shock can occur in water colder than 20° C (68° F), with symptoms increasing as water temperature decreases toward freezing. Staged entry into cold water attenuates the respiratory responses because of the gradual increase in thermal stimulation. The best advice to combat this phenomenon is to control the entry into cold water, when possible, by entering slowly and most importantly by keeping the head from being submersed. The next step is to focus on just surviving the first minute by not panicking and by consciously getting breathing under control.

Phase 2: Cold Incapacitation (Short-Term Immersion, 5 to 15 Minutes)

For those surviving the cold-shock response, significant cooling of peripheral tissues continues, especially in the extremities, with most of the effect occurring over the first 15 minutes of

immersion. This cooling has a direct and deleterious effect on neuromuscular activity.[182] This effect is especially significant in the hands, where blood circulation is negligible,[35] leading to finger stiffness, poor coordination of gross and fine motor activity, and loss of power.[40,49,128,129,187] It has been shown that this effect is primarily caused by peripheral and not by central cooling.[60] The loss of motor control makes it difficult, if not impossible, to execute survival procedures, such as grasping a rescue line or hoist. Thus, the ultimate cause of death is drowning, either through a failure to initiate or maintain survival performance (i.e., keeping afloat, swimming, grasping onto a life raft) or excessive inhalation of water under turbulent conditions.

These phenomena have clear survival implications. It is, of course, advisable to avoid cold-water exposure completely. If cold-water immersion does occur, however, it is best to quickly determine and execute a plan of action: (1) try to enter the water without submersing the head; (2) escape (e.g., pull oneself out of the water, inflate and board a life raft); (3) minimize exposure (e.g., get as much of one's body as possible out of the water and onto a floating object); (4) ensure flotation if one must remain in the water (e.g., don or inflate a PFD); and (5) call for assistance (e.g., activate signaling devices). It may be difficult to execute these actions while the cold-shock responses predominate. However, once the respiratory effects are under control, immediate action should be taken. If self-rescue is not possible, actions to minimize heat loss should be initiated by remaining as still as possible, by keeping as much of the body on top of a floating object as possible, by adopting the heat-escape-lessening position (HELP) (where arms are pressed against the chest, and legs are pressed together), or by huddling with other survivors (Fig. 6-4).[83] Drawstrings should be tightened in clothing to decrease the flow of cold water within clothing layers.

Phase 3: Onset of Hypothermia (Long-Term Immersion, Longer Than 30 Minutes)

Most cold-water deaths likely result from drowning during the first two phases of cold-water immersion, as discussed earlier. In general, true hypothermia usually becomes a significant contributor to death only if immersion lasts longer than 30 to 60 minutes. The individual who survives the immediate and short-term phases of cold-water immersion faces the possible onset of hypothermia as continuous heat loss from the body eventually decreases core temperature (T_{co}). Many predictive models to determine the core temperature response to cooling are based on the relationships between body composition, thermoregulatory response (i.e., shivering thermogenesis), clothing/insulation, water temperature, and sea conditions (see Cold-Water Survival, later).[86,159,193,197,201]

Even in ice water, a victim may not become unconscious due to hypothermia (T_{co} ~30°C [86°F]) if a PFD is worn or some other factor prevents the need for vigorous exercise to keep from drowning. Furthermore, if the head is kept above water after unconsciousness occurs, the victim could still survive for up to 1 hour more before the heart stops, as long as the sea is relatively calm and waves do not wash over the mouth. The public should be taught that death does not necessarily follow sudden immersion in cold water (Box 6-2).

Phase 4: Circum-Rescue Collapse

Anecdotal cases of fatalities among victims of immersion hypothermia have been noted in the immediate pre- and post-

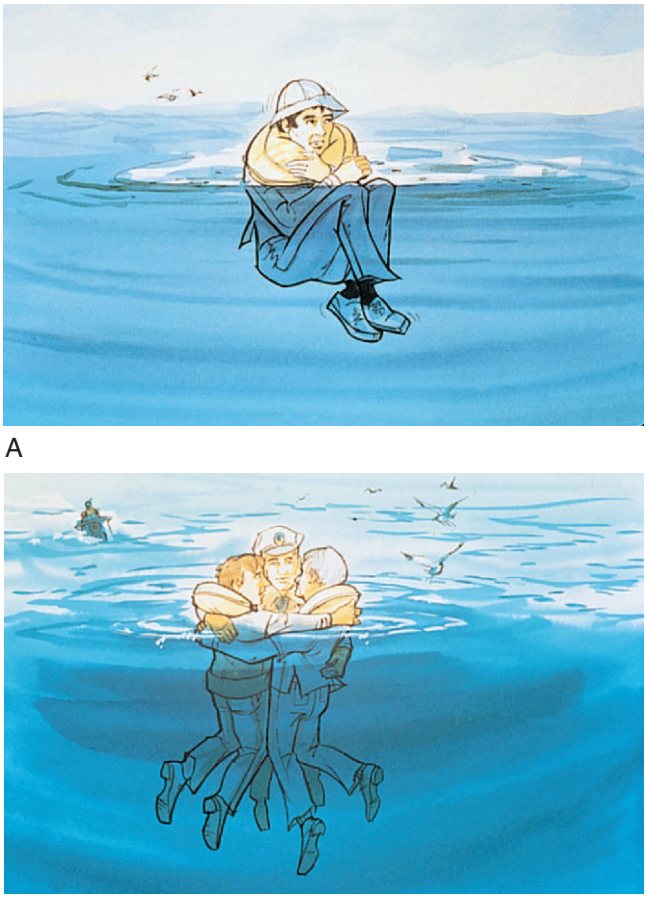


Figure 6-4. Heat-escape-lessening position (HELP) and huddle techniques for decreasing cooling rates of survivors in cold water. (From Hayward JS, Eckerson JD, Collis ML: J Appl Physiol 38:1073–1077, 1975, with permission.)

Box 6-2. Time Available if You Fall into Ice Cold Water: 1-10-1 Principle*

1 Minute	You have 1 minute to get your breathing under control, so don't panic.
10 Minutes	You have 10 minutes of meaningful movement to get out of the water or attain a stable situation.
1 Hour	You have up to 1 hour before you become unconscious from hypothermia, *if* you don't panic and struggle unnecessarily. Note: If you are wearing a personal flotation device, you may have an additional hour before your heart stops due to hypothermia.

*Times are subject to individual variability and factors such as water temperature and amount of the body immersed.

rescue periods despite the survivor's being recovered in an apparently stable and conscious condition. The hypothermic victim may experience symptoms ranging from fainting to cardiac arrest. These events are often referred to as cases of rewarming shock or post-rescue collapse. Golden and colleagues[71] have noted that deaths can occur either shortly before rescue, during rescue, or after rescue, and they have used the term *circum-rescue collapse* to describe these events. Deaths have occurred within minutes before rescue, while climbing out of the water, while being hoisted to a helicopter, within a few minutes after entering a warm compartment of a rescue vessel, 20 to 90 minutes after rescue, in hospital after transport, and during the 24-hour period after the rescue.[68,71,97,113]

Three causes of circum-rescue collapse have been proposed: (1) afterdrop—a continued drop in T_{co} after recovery, (2) collapse of arterial pressure, and (3) factors that potentiate the risk of ventricular fibrillation (e.g., hypoxia, acidosis, rapid changes in pH). These causes are discussed later.

Prior to imminent rescue, mental relaxation and decreased output of stress hormones may result in a drop of blood pressure that results in fainting and drowning. The act of rescue itself may also cause sudden collapse. Pulling a victim out of the water in a vertical position removes the hydrostatic squeeze around the lower limbs and may cause blood pooling in the these extremities and subsequent decreased blood pressure. This extra cardiac work or rough handling may induce a reflex cardiac arrest of the cold heart.

Afterdrop is a well-known phenomenon that occurs after removal of a victim from cold-water immersion. Core temperature continues to decline (i.e., afterdrop occurs) in both animate and inanimate objects, even though cold-water immersion has ended.[3,15,22,54,70,137,143,190] An afterdrop of as much as 5° to 6°C (9° to 10°F) has been observed in humans.[7,43,157] This afterdrop was long proposed as the major cause of post-rescue death because further cooling of the heart might result in a temperature at which ventricular fibrillation or cardiac arrest could occur.[15]

Golden and coworkers[71] discount the importance of this phenomenon and propose collapse of arterial pressure as the cause of death during rescue. They point out (correctly) that an afterdrop occurring in victims who were hypothermic, yet warm enough to climb on board a ship without assistance, would be unlikely to cause enough myocardial cooling to result in cardiac arrest or ventricular fibrillation. Rather, they propose that removal from cold water results in a precipitous fall in blood pressure, inadequate coronary blood flow, and myocardial ischemia, possibly precipitating ventricular fibrillation. This mechanism is most likely the main factor in collapse both just before and during rescue. The fall in blood pressure *prior to* removal from the water may be due to reduced sympathetic tone or catecholamine secretion when rescue is imminent. Hypotension *during* and *shortly after* rescue may be caused by the sudden decrease in the hydrostatic pressure on the body (i.e., removal of the hydrostatic squeeze of water pressure during immersion), hypovolemia, or impaired baroreceptor reflexes. However, this mechanism cannot fully account for all other post-rescue deaths occurring 20 minutes to 24 hours after rescue.

The importance of continued myocardial cooling cannot be discounted. Fibrillation of a cold heart can be initiated by mechanical stimuli,[130] by hypoxia and acidosis,[124] and by rapid changes in pH.[98] In dogs, myocardial cooling from 30° to 22°C (86° to 71.6°F) has been shown to cause a fivefold decrease in the electrical threshold for fibrillation.[27] It is also likely that an increase in the rate of myocardial cooling may stimulate fibrillation. On the basis of the large afterdrop values described earlier, it is plausible that continued core cooling in significantly hypothermic victims could result in ventricular fibrillation, either primarily, due to spontaneous fibrillation of the cold myocardium, or secondarily, due to cold-induced increase in sensitivity to other fibrillation stimuli.

Finally, death may occur within minutes to hours after rescue. A rescued victim may be severely compromised with cold alkaline or acidic blood in the extremities, a heart extremely prone to failure, decrease or loss of consciousness, and low blood volume (hypovolemia). Sudden redistribution of blood to the extremities (especially the lower extremities) may cause collapse through decreased blood pressure and cardiovascular instability, sudden return of metabolic byproducts to an irritable heart, or continued decrease in temperature (afterdrop) of an irritable heart. Core temperature continues to drop and the heart reacts with tachycardia (high heart rate) or fibrillation. Up to 20% of those recovered alive die due to circum-rescue complications, either before and during rescue or within hours after.

FACTORS AFFECTING BODY CORE COOLING

Normal core temperature fluctuates around 37°C (98.6°F). The clinical definition of hypothermia is a T_{co} of 35°C (95°F) or lower; however, any exposure to cold that lowers the core temperature below normal levels results in the body's becoming hypothermic. Although various temperatures and terms have been used to classify different levels of hypothermia, the following classification is used here and in Chapter 5 (Fig. 6-5). In mild hypothermia (T_{co} = 32° to 35°C [90° to 95°F]), thermoregulatory mechanisms continue to operate fully, but ataxia, dysarthria, apathy, and even amnesia are likely. In moderate hypothermia (T_{co} = 28° to 32°C [82° to 90°F]), the effectiveness of the thermoregulatory system (e.g., shivering thermogenesis) diminishes until it fails, there is a continued decrease in level of consciousness, and cardiac dysrhythmias may also occur. In severe hypothermia (T_{co} < 28°C [82°F]), consciousness is lost, shivering is absent, acid–base disturbances develop, and the heart is susceptible to ventricular fibrillation or asystole. Death from hypothermia is generally from cardiorespiratory failure.

If a victim of cold-water immersion can avoid drowning during the initial few minutes after water entry, prevention of hypothermia becomes an important problem. Survival time in cold water, based on the pathophysiologic effects of decreasing core temperature, is not a precise calculation. Among survivors, the large individual variations in body morphology and state of health and fitness, combined with many exogenous variables affecting cooling rate (e.g., clothing, water temperature, sea state, flotation, behavior), preclude exact predictions of survival time. However, sufficient experimental data and case history findings exist to allow generalizations. At a core temperature of 34°C (93°F), there is a significant deleterious effect on manual dexterity and useful function in cold water.[26,126] If a survivor is trying to combat rough seas, this level of dysfunction may potentiate drowning. At a core temperature of 30°C (86°F), unconsciousness is probable.[20,31,154,155,159] Even if a survivor is

Classifications	Core temp	Patient's ability to rewarm without external heat source	Clinical presentation	
Normal	**Above 35° C (95° F)**		**Cold sensation; shivering**	
Mild	35-32° C (95-90° F)	Good	Physical impairment • Fine motor • Gross motor	Mental impairment • Complex • Simple
Moderate	32-28° C (90-82° F)	Limited	**Below 30° C (86° F) shivering stops; loss of consciousness**	
Severe	Below 28° C (82° F)	Unable	Rigidity Vital signs reduced or absent Severe risk of mechanically stimulated ventricular fibrillation (VF) (rough handling)	
	Below 25° C (77° F)	Unable	**Spontaneous ventricular fibrillation (VF); cardiac arrest**	

Bold text shows the major thresholds between stages of hypothermia.

Figure 6-5. Classification of levels of hypothermia.

wearing a self-righting PFD designed to maintain airway freeboard in an unconscious person, drowning is probable at this core temperature in all but the calmest seas. At core temperatures below 25°C (77°F), ventricular fibrillation or asystole often occurs spontaneously.[31] Of these three core temperatures, 30°C (86°F) is the most practical for defining the limits of survival in cold water.

The rate of body core cooling during cold-water immersion depends on the following variables: water temperature and sea state, thermal protection, body morphology, amount and area of the body immersed in water, behavior (e.g., excessive movement) and posture (e.g., HELP, the huddle technique) of the body in the water, shivering thermogenesis, and other, nonthermal factors.

Water Temperature and Sea State

The variables of water temperature and sea state are critically important in the intensity of the cold-shock response, the onset of physical and mental impairment, and the rate of core cooling. Figure 6-6 graphically illustrates the inverse linear relationship between water temperature and T_{co} cooling rate.[78] Water has a specific heat 4000 times that of air and a thermal conductivity approximately 25 times greater.[122] Immersion in cold water is thus associated with a high rate of heat transfer from skin to water, at least 100 times greater than in air of the same temperature.[122]

Thermal Protection

A large number of studies over the past few decades have evaluated the relationship of different types of protective clothing

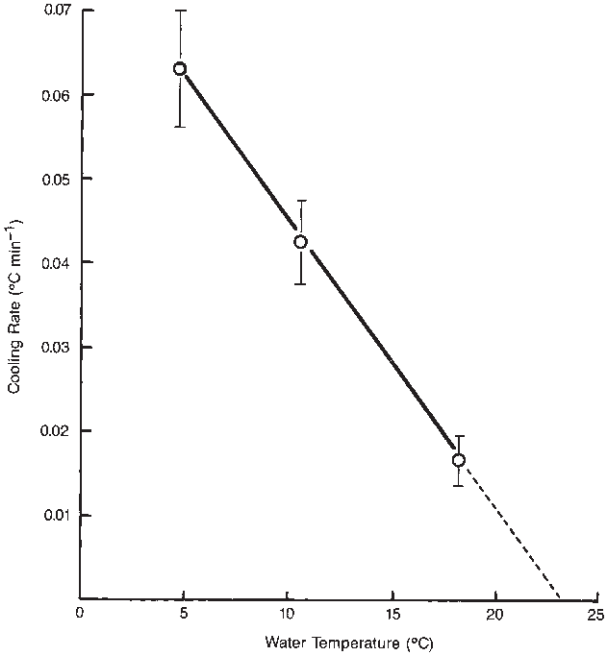


Figure 6-6. The relationship between water temperature and the mean cooling rate (by rectal temperature) in lightly clothed, nonexercising men and women during immersion in seawater. (From Hayward J: In Pozos R, Wittmers LE (eds): The Nature and Treatment of Hypothermia. Minneapolis, University of Minnesota Press, 1983, with permission.)

TABLE 6-2. Mean Linear Cooling Rates for Lean Men Dressed in Various Types of Garments in Calm Water at 10° C (50° F)

TYPE OF PROTECTIVE CLOTHING	COOLING RATE (°C/HR ± SD)
Control (equivalent to ordinary street clothes)	3.2 ± 1.1
"WET DESIGN"	
Thermal "float coat" (loose-fitted, 5.4-mm closed-cell foam-insulated jacket)	1.6 ± 0.6
Short "wet suit" (custom-fitted, 3.2-mm closed-cell foam covering arms, trunk, and upper thighs)	1.2 ± 0.4
"Insulated coveralls" (loose-fitted, 3.2-mm closed-cell foam covering extremities and trunk)	1 ± 0.4
Full "wet suit" (custom-fitted, 4.8-mm closed-cell foam covering extremities and trunk)	0.7 ± 0.3
"DRY DESIGN"	
"Immersion suit" (loose-fitted, 4.8-mm closed-cell foam with sealed openings)	0.5 ± 0.3

From Steinman AM, Hayward JS, Nemiroff MJ, Kubilis PS: Aviat Space Environ Med 58:550–558, 1987.

TABLE 6-3. Comparison of Mean Cooling Rates for Thin Men (Mean Body Fat = 9.1%) Wearing Various Types of Protective Clothing in Calm Water at 11.8° C (53° F)

CLOTHING TYPE	MEAN COOLING RATE (°C/HR)	*RATIOS TO CONTROL*	
		DIRECT	INVERSE
Dry, closed-cell foam insulation (4.8 mm thick)	0.31	0.14	7.35
Wet, closed-cell foam insulation (4.8 mm thick)	0.54	0.23	4.26
Dry, uninsulated (watertight shell over lightweight clothing)	1.07	0.47	2.15
Control (lightweight clothing alone)	2.3	1	1

From Hayward JS: Design concepts of survival suits for cold-water immersion and their thermal protection performance. In 17th Symposium of the SAFE (Survival and Flight Equipment) Association. Van Nuys, CA, 1979.

to heat loss and cooling rates.* Nearly all of these studies have been conducted in calm water or in laboratory settings. As illustrated by the cooling rate data in Tables 6-2 and 6-3, such studies have generally shown that in calm water, intact, "dry," insulated garments provide better protection than "wet" insulated garments, and well-insulated garments provide significantly better protection than poorly insulated garments. Figure 6-7 shows various types of cold-water protective garments.

Although calm-water studies have value for comparing the relative degree of protection afforded by different types of protective clothing, many immersion accidents occur in rough water.[139,164,165] In this environment, a survivor's cooling rate may be affected by swimming to maintain airway freeboard, by passive body movements caused by waves, by flushing of cold water through wetsuits, and by leakage of cold water into drysuits. For example, subjects in a wave tank demonstrate higher energy expenditure and faster cooling rates than subjects in calm water.[77] Several experimental studies have demonstrated significantly faster cooling rates for human volunteers wearing "wet" protective garments in rough or turbulent water[138,154,198] than for persons in calm water. Even drysuits have shown degradation of protection in rough water. In a recent study of dry immersion suits in 16° C (61° F) water, wave heights up to 70 cm resulted in a 14% decrease in total suit insulation and an average 45% decrease in thermal resistance at the head and trunk regions of the suits.[33]

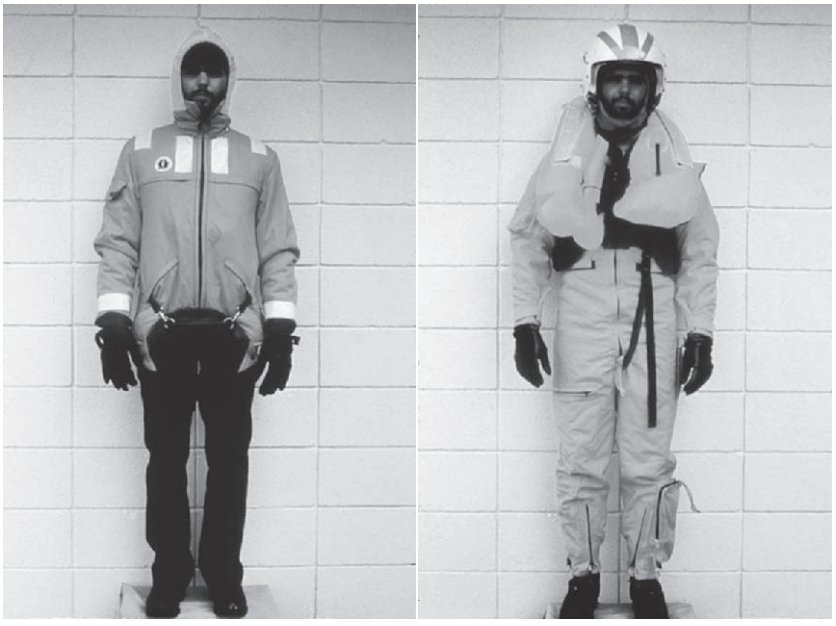
*References 6, 33, 79, 80, 95, 96, 105, 126, 145, 154, 155, 162, 166, 168, 169, 171, 194, 198.

Figure 6-8 shows a comparison of cooling rates for lean men dressed in the various types of protective clothing shown in Figure 6-7 in both calm and rough water at approximately 10° C (50° F).[154] The most dramatic differences occurred in the loose-fitted, "wet" protective clothing (such as the float coat and insulated coveralls), where cooling rates in rough seas were nearly double those in calm seas. This was primarily due to flushing of cold water through the garments. However, even the tight-fitted, full wetsuit allowed a 30% faster cooling rate in rough water over that in calm water. The drysuit, which did not leak, showed no significant difference between calm and rough seas.

Estimated survival times in rough seas, based on experimental data, were published for thin men wearing different types of protective clothing in 6° C (43° F) water.[155] Table 6-4 shows these times for three different levels of survival. The following assumptions underlie these estimates:

- Cooling rates are linear.[79,154,155,159]
- Initial core temperature is 37.5° C (99° F).
- Survivors are able to maintain airway freeboard until unconsciousness occurs at a rectal temperature of 30° C (86° F).
- Self-righting flotation maintains airway freeboard when survivors are unconscious.

In comparing these estimated survival times with those of Figure 6-9 (calm-water survival times), the reader must recall that Figure 6-9 concerns only survival to a core temperature of 30° C (86° F). Furthermore, the zone in the graph must be adjusted downward for rough seas and for survivors wearing



A B

Figure 6-7. Antiexposure garments. **A,** Float coat. **B,** Aviation coveralls with personal flotation device.

only light clothing, and upward for survivors wearing insulated clothing. For 6° C (43° F) water, the survival times (to a core temperature of 30° C [86° F]) correlate well between Figure 6-9 and Table 6-4. It must be emphasized that the estimates in Table 6-4 pertain to lean individuals with a mean body fat content of only 11.1%. Because many populations of adults (such as offshore oil workers) in the 30- to 50-year age range average 25% to 30% body fat, the estimates of time to unconsciousness and time to cardiac arrest must be considered conservative for a broader spectrum of adults.

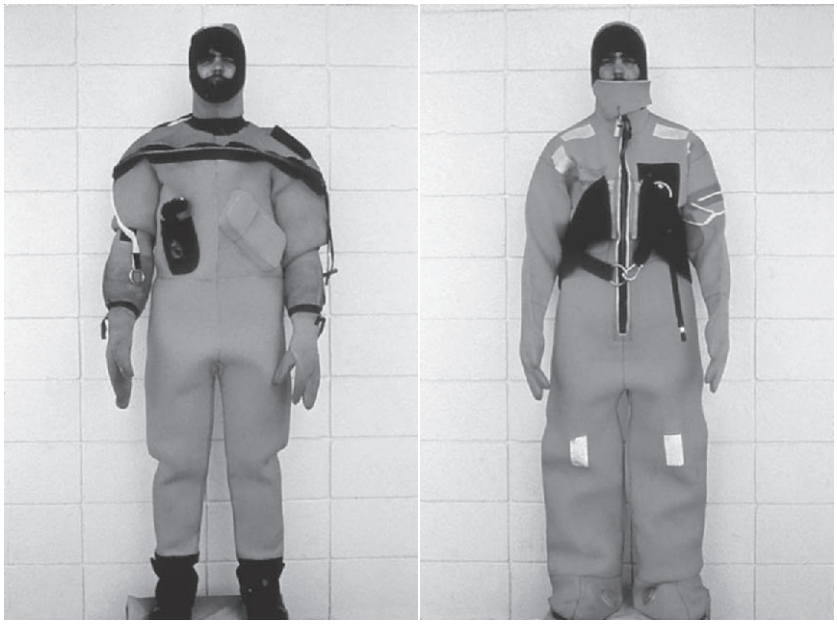
Body Morphology (Size and Composition)

Children cool faster than adults because children have a greater surface-area-to-mass ratio. Cooling rate is generally proportional to surface area, whereas the amount of heat that can be lost is proportional to mass. Thus, a large surface-area-to-mass ratio favors cooling. Similarly, smaller adults generally cool faster than larger adults, and tall, lanky individuals cool faster than short stout individuals. Body composition is also important. Subcutaneous fat is a very efficient insulator against heat loss, and cooling rate is inversely related to skinfold thickness. For example, subjects in the 10th percentile for skinfold thickness wearing light clothing in 5° C (41° F) water have nine times the cooling rate of 90th percentile subjects.[126] Figure 6-10 shows the linear relationship between change in core temperature and mean skinfold thickness.[98] Shivering, which is a primary defense against core cooling, also varies with skinfold thickness. At a given skin temperature, the shivering response is less in people with greater amounts of subcutaneous fat.[103] In moderately cold water temperatures (18° to 26° C [64° to 79° F]), core cooling has been shown to proceed at the same rate in individuals with high and with low fat because of the greater shivering thermogenesis in the low fat subjects. However, at colder water temperatures (8° C [46° F]), core cooling is attenuated by greater amounts of subcutaneous fat and body mass, because of increased insulation.[50]

In general, for a survivor immersed in cold water, the core cooling rate is fairly linear once T_{co} begins to decline. This has been shown to be true for mildly hypothermic experimental subjects. The only data that exist for severely hypothermic humans are those from the infamous and reprehensible Dachau concentration camp atrocities, in which conscious victims, cooled to death in ice water, had linear cooling rates.[3] Because these unfortunate victims were emaciated and ill, these data may not apply to healthy cold-water immersion subjects.

The advantage of body fat as an insulator against cold has been discussed (see Physiologic Responses to Cold-Water Immersion). Using an extrapolation of linear cooling rate to 30° C (86° F), Figure 6-9 shows predicted *calm-water* survival times of lightly clothed, nonexercising humans in cold water. The graph shows a line for the average expectancy and a broad zone that indicates the large amount of individual variability associated with different body size, build, and degree of fatness. The zone would include approximately 95% of the variation expected for adults and teenagers under the conditions specified. In the zone where death from hypothermia is highly improbable, cold water still potentiates death from drowning from cold shock (as discussed earlier) in the first few minutes of immersion, especially for those not wearing PFDs. Figure 6-9 discusses *only* calm-water survival times. Because rough-water conditions decrease survival times, as will be discussed later, Figure 6-9 may be useful for estimating *maximum* survival times for an individual immersed in cold water. Search-and-rescue organizations might find such a maximum survival time helpful because they often use the longest possible survival time in deciding when to terminate a search effort.

C D E



F G

Figure 6-7, con't. **C,** Boat-crew coveralls or snowmobile suit. **D,** Short wetsuit worn as an undergarment. **E,** Full wetsuit. **F,** Insulated drysuit. **G,** Immersion suit. Garments **A** and **C** are loose-fitting, closed-cell-foam-insulated wetsuits; garments **D** and **E** are tight-fitting, closed-cell-foam-insulated wetsuits; garments **F** and **G** are closed-cell-foam-insulated drysuits.

Amount of Body Immersed

Because of the difference in thermal conductivity between air and water, heat loss from body surfaces immersed in cold water is usually far greater than from body surfaces exposed to cold air, even considering the effect of windchill. Thus, immersed victims should attempt to get as much of their bodies out of the water as possible (see Cold-Water Survival, later).

Head Immersion

Exposure of the dorsal head to cold water may increase a survivor's rate of cooling and adversely affect mental performance. Survival may depend on the ability to conduct multiple tasks to avoid drowning and hypothermia. The ability to carry out these tasks involves both physical components (affected by local muscle and nerve temperature)[60] and mental components

(affected by brain temperature)[47]; both may be adversely affected by hypothermia.

Head immersion has traditionally been of concern in survival situations.[66] In the absence of water in the airways, heat is lost through the head via convection (heat loss from the blood that perfuses the scalp) or conduction (direct heat loss through bone and soft tissue).[200] One hypothesis predicts a substantial heat loss through the head because of abundant scalp vascularity and because the scalp vasculature does not constrict in response to cold as do superficial vessels in other body areas.[45] Alternatively, heat loss from the back of the head might be minimal because dorsal head and neck immersion would involve only an additional 3% to 5% of the body surface area (BSA).[102] As well, conductive heat loss directly through the scalp and skull is likely to be minimal.[200]

A few studies have addressed head cooling in animals in water[24] or in humans in cold air,[45,135] but only one human study has included cold-water exposure of the head. Alexander[3] reported the earliest data on head cooling from studies carried out on prisoners of war during World War II. These studies indicated that cooling the back of the head caused discomfort and significantly hastened the onset of hypothermia.[3] However, these studies were grossly unethical, and the results are considered invalid and unusable because of the emaciated condition of the prisoners as well as questions regarding the protocol and accuracy of the results. The experiments were inhuman and deplorable.

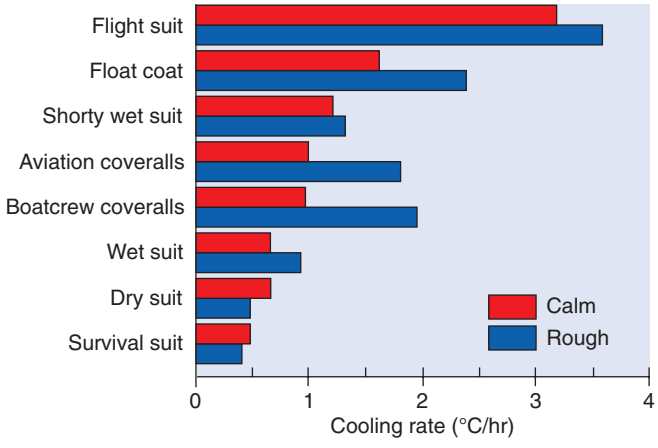


Figure 6-8. A comparison of mean rectal temperature cooling rates in lean male subjects in calm versus rough seas at 10.7° C (51° F). The flight suit, used as a control garment, is equivalent to light clothing. (From Steinman AM, Hayward JS, Nemiroff MJ, Kubilis PS: Aviat Space Environ Med 58:550–558, 1987, with permission.)

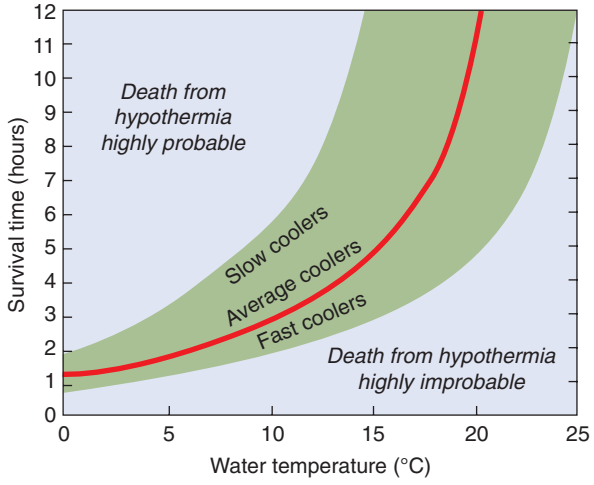


Figure 6-9. Predicted calm-water survival time (defined as the time required to cool core temperature to 30° C (86° F) for lightly clothed, nonexercising humans in cold water. The *line* is the average expected, and the *broad zone* indicates the large amount of individual variability associated with body size, build, fatness, physical fitness, and state of health. The zone includes approximately 95% of the variation expected for adult and teenaged humans under the conditions specified. The zone would be shifted downward by physical activity (such as swimming) and upward slightly for heavy clothing or protective behaviors (such as huddling with other survivors or adopting a HELP position in the water). Specialized insulated protective clothing (e.g., survival suits, wetsuits) can increase survival time from 2 to 10 times (or more) the basic duration shown. In the zone where death from hypothermia is highly improbable, cold shock on initial immersion can potentiate death from drowning, particularly for those not wearing flotation devices. (From U.S. Coast Guard: Addendum to the National Search and Rescue (SAR) Manual. COMDTINST M16120.5 and COMDTINST M16120.6, 17 August 1995, with permission.)

TABLE 6-4. Estimated Survival Times for Lean Subjects (Mean Body Fat = 11.1%) Wearing Various Types of Protective Clothing in Rough Seas

CLOTHING TYPE	*ESTIMATED SURVIVAL TIME (HR) (95% CONFIDENCE RANGE)*		
	TIME TO INCAPACITY (T = 34° C)	TIME TO UNCONSCIOUSNESS (T = 30° C)	TIME TO CARDIAC ARREST (T = 25° C)
Control (lightweight clothing)	0.4–1.3	0.8–2.6	1.3–4.3
Torn, non-foam–insulated dry coverall (2-inch tear in left shoulder)	0.9–2.7	1.6–5.2	2.5–8.4
Closed-cell foam-insulated, wet coverall (3.2-mm thick insulation in a loose fitted coverall)	1–2.9	1.9–6	3–9.9
Closed-cell foam-insulated, custom-fitted wet suit (4.8-mm thick insulation; tight-fitted)	1.6–4.7	3.1–9.9	4.9–16.2
Intact, non-foam–insulated dry coverall (watertight shell over thick, fiberfill, insulated underwear)	2.9–8.8	5.7–18.2	9.1–30

From Steinman AM, Kubilis P: Survival at sea. Report No. CG-D-26-86, USCG, National Technical Information Service, 1986.

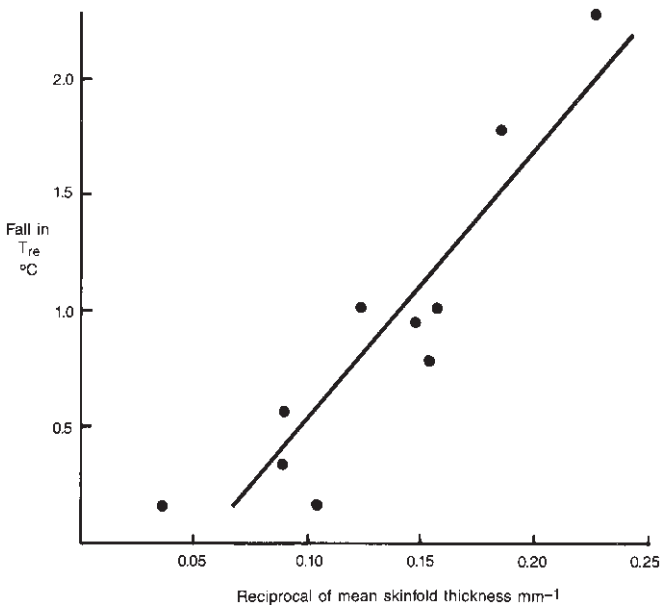


Figure 6-10. The relationship between subcutaneous fat thickness in 10 men and the decline in rectal temperature during 30-minute immersions in stirred water at 15° C (59° F). Skinfold thickness is the mean of measurements at biceps, abdominal, subscapular, and subcostal sites. T_{re}, rectal temperature. (From Keatinge WR: Survival in Cold Water. Oxford, Blackwell Scientific, 1969, with permission.)

Few experimental data are available to evaluate the effects of mild hypothermia on mental performance. A few medical studies have evaluated impairment of memory in divers during cold-water dives.[11,186,189] Two other studies have involved cognitive testing during hypothermia without direct head cooling.[21,47]

Lockhart and colleagues[108] recently evaluated the effects of dorsal head and neck immersion in human subjects immersed in 10° C (50° F) water on both core temperature cooling rates and on mental performance. Dorsal head and neck immersion (with the body insulated) resulted in a cooling rate of 0.4° ± 0.2° C/hr (0.7° ± 0.4° F/hr); body immersion (with the head, neck, and upper thorax out of the water) resulted in a cooling rate of 1.5° ± 0.7° C/hr (2.7° ± 1.3° F/hr); but immersion of *both* the whole body and dorsal head and neck nearly doubled the cooling rate, to 2.8° ± 1.6° C/hr (5.0° ± 2.9° F/hr). In addition, there were significant correlations between diminished cognitive performance and decreasing core temperature. The time required to correctly complete the Stroop Word-Color test increased as core temperature decreased. The number of correct responses decreased with core temperature for Digit Symbol Coding, Backward Digit Span, and Paced Auditory Serial Addition test.

Giesbrecht and coworkers[56] repeated the head-cooling study using subjects immersed in 12° C (54° F) water whose shivering thermogenesis had been inhibited with intravenous (IV) meperidine.[64] After 30 minutes' immersion, there was no significant difference in core temperature decline between head-only exposure and control (neither head nor body exposed). However, with the body immersed, there was a significant increase in cooling rate (5.0° C [9.0° F]/hr) in subjects whose dorsal head and neck and anterior thorax were immersed compared with subjects with head, neck, and anterior thorax out of the water (3.6° C [6.5° F]/hr).

These results did not confirm the supposition of proportionately greater heat loss from the head. The measured heat loss from the head in both head-immersed conditions was only about 60 kJ (compared with 18 to 33 kJ in the two head-out conditions). In contrast, total body heat losses in the body-exposed configurations were about 1100 and 1260 kJ, respectively, for head-out and back-of-the-head-in conditions. Thus, the head accounted for only about 3% and 5% of the total body heat losses, respectively, in the body-exposed conditions. The majority of the difference in heat loss in the body-immersed conditions came from immersion of the anterior thorax rather than of the dorsal head and neck.

It can thus be concluded that the head is not an area of exceptionally high heat loss, and heat losses from the head are not disproportionately increased over what would be expected from the head's contribution to total body surface area. This confirms the prediction of Xu, who hypothesized minimal heat loss from the back of the head on the basis of modeling and experimental findings.[200] However, these studies show the importance, from the standpoints of minimizing heat loss to the environment and maintaining cognitive function, of protecting the head in a survival situation.

Behavior and Posture of the Body in Cold Water

Behavioral variables also affect the core cooling rate. Hayward and coworkers used infrared thermography to demonstrate that, despite marked peripheral vasoconstriction, heat losses are high in the groin, lateral and central thorax, and neck.[82] In the groin and neck, regions with a relatively thin layer of peripheral soft tissue, blood flow through the large, relatively superficial femoral vessels, carotid arteries, and jugular veins potentiates heat flow to the cold water. In the lateral and central thorax, the relative absence of tissue insulation (muscle and subcutaneous fat), combined with high thermal conductivity of rib bone, potentiates heat loss from relatively warm lungs to the cold environment. Furthermore, exercise or excessive movement in the water greatly increases heat loss from active musculature.

The effect of activity on total heat balance depends on the balance between the many factors illustrated in Figure 6-11. In normothermic circumstances, heat produced locally in peripheral muscles is transferred to the core via venous return. In cold-water immersion, by contrast, physical activity may actually increase heat loss through increased blood flow to the periphery. This is especially relevant when immersed victims engage in excessive movement in the water (e.g., swimming or vigorous extremity movements necessary to maintain airway freeboard in rough seas).

Hayward and colleagues[83] demonstrated that minimizing both voluntary activity and the exposure of major heat loss areas of the skin to cold water is the most effective way to minimize the decline in core temperature. They showed that treading water and drown-proofing significantly increased the cooling rate. In spite of increased metabolic heat production during exercise, the increased surface heat loss resulted in faster core cooling during exercise in cold water. They also developed two well-known cold-water survival techniques: the heat escape lessening posture (HELP) and the group huddle (see Figure 6-4). These adaptive behaviors reduced core cooling by 69%

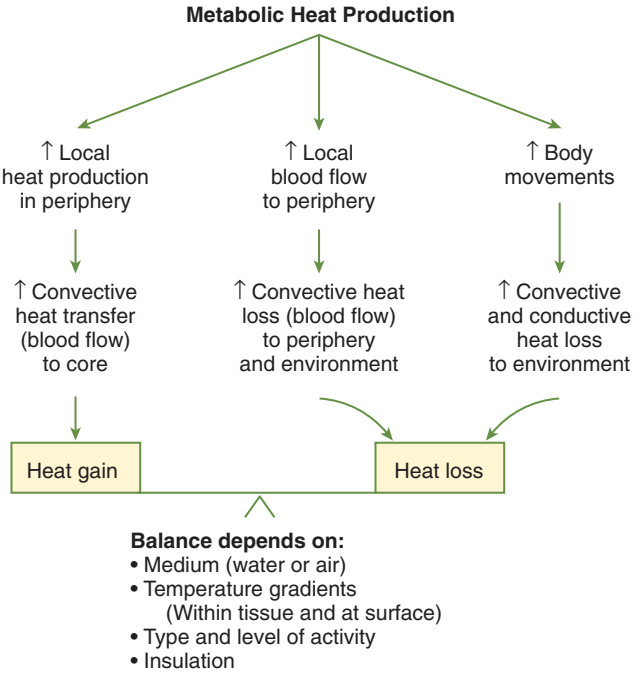


Figure 6-11. Factors influencing total thermal balance during increases in metabolic heat production (e.g., voluntary exercise and involuntary shivering).

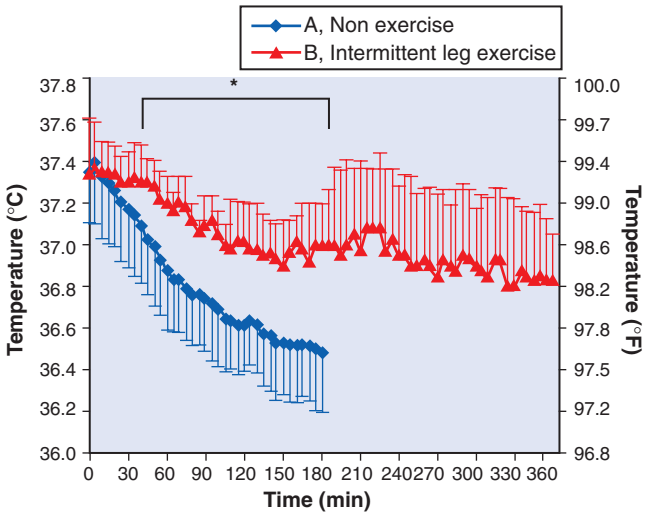


Figure 6-12. Rectal temperature for six subjects during immersion in 0° C (32° F) water and –5° C (23° F) air (mean ± SD). **A,** No exercise. **B,** Intermittent leg exercise for 5 minutes every 20 minutes. *Asterisk* indicates a significantly higher rectal temperature in condition **B** compared with **A** from 40 to 180 minutes. (From Færevik, Reinertsen, and Giesbrecht, unpublished data.)

and 66%, respectively.[78] When the sea is not calm, it may be difficult to perform the group huddle with complete thermal efficiency. However, other advantages of this position include maintenance of group contact and morale. One viable alternative in rough seas is to tether individuals together, with built-in or improvised tethers, allowing each survivor to perform the HELP position and maintain proper orientation to the waves. Sagawa and coworkers concluded that the lowest water temperature in which humans could maintain normal core temperature by generating body heat through muscular activity is 25° C (77° F).[141]

Exercise

Normally, the advice is to not exercise while awaiting rescue in cold water. This is because of the understanding that, although metabolic heat production will increase, exercise will increase blood flow to exercising muscles and cause increased heat loss to the water with a net result of increased core cooling. A recent study by Færevik and colleagues (unpublished observations) was conducted in subjects wearing neoprene survival drysuits in a wave tank with a water temperature of 0° C (32° F) and an air temperature of –5° C (23° F). When they remained still, core temperature dropped more in 3 hours of immersion than it did during 6 hours of immersion while they performed leg exercises for 5 minutes every 20 minutes (Fig. 6-12). Thus, if enough insulation is worn, the heat of exercise can be contained within the insulation, which attenuates the drop in core temperature.

Shivering

Shivering is a thermoregulatory function in which involuntary muscle contraction increases heat production in an effort to prevent or minimize body core cooling. Shivering intensity increases as core and skin temperatures decrease. Generally, shivering intensity is maximal at T_{co} = 32° to 33° C (90° to 91° F) and T_{skin} = ~20° C (68° F). However, shivering heat production is lower, at any given skin temperature, in individuals with higher levels of subcutaneous fat.[103,195]

Thermal balance during shivering depends on the same factors that are involved during voluntary activity. Figure 6-13 illustrates how heat production by shivering can maintain core temperature in cold air and can arrest the fall in core temperature in both warm and cool water. In colder water, the combination of shivering thermogenesis and body insulation may result (because of peripheral vasoconstriction) in the maintenance of a steady-state core temperature, albeit below normothermic levels. At even lower water temperatures, T_{co} will continue to decrease, and this decrease accelerates when shivering thermogenesis eventually stops because of hypothermia-induced thermoregulatory impairment (below T_{co} = ~30° C [86° F]). The power of shivering is especially important when considering the clinical classification of hypothermia and rewarming therapies, because this valuable heat source is an efficient mechanism for rewarming the core during recovery and resuscitation (see Rescue, and Medical Management, later).

Nonthermal Factors

Underwater divers often experience "symptomless" or undetected hypothermia. Several factors contribute to increased cooling. First, heat is lost through direct conduction to cold water and through breathing compressed air. Breathing compressed air at depth also alters human thermoregulation. Mekjavic studied the effects of hyperbaric nitrogen at 6 atm[114] and, with Passias and coworker, simulated inert gas or "nitrogen" narcosis with inhalation of 30% nitrous oxide.[132] They demonstrated a decrease in the core temperature threshold for shivering and an increase in the rate of core cooling of up to twofold under these conditions. In a separate study, Mekjavic

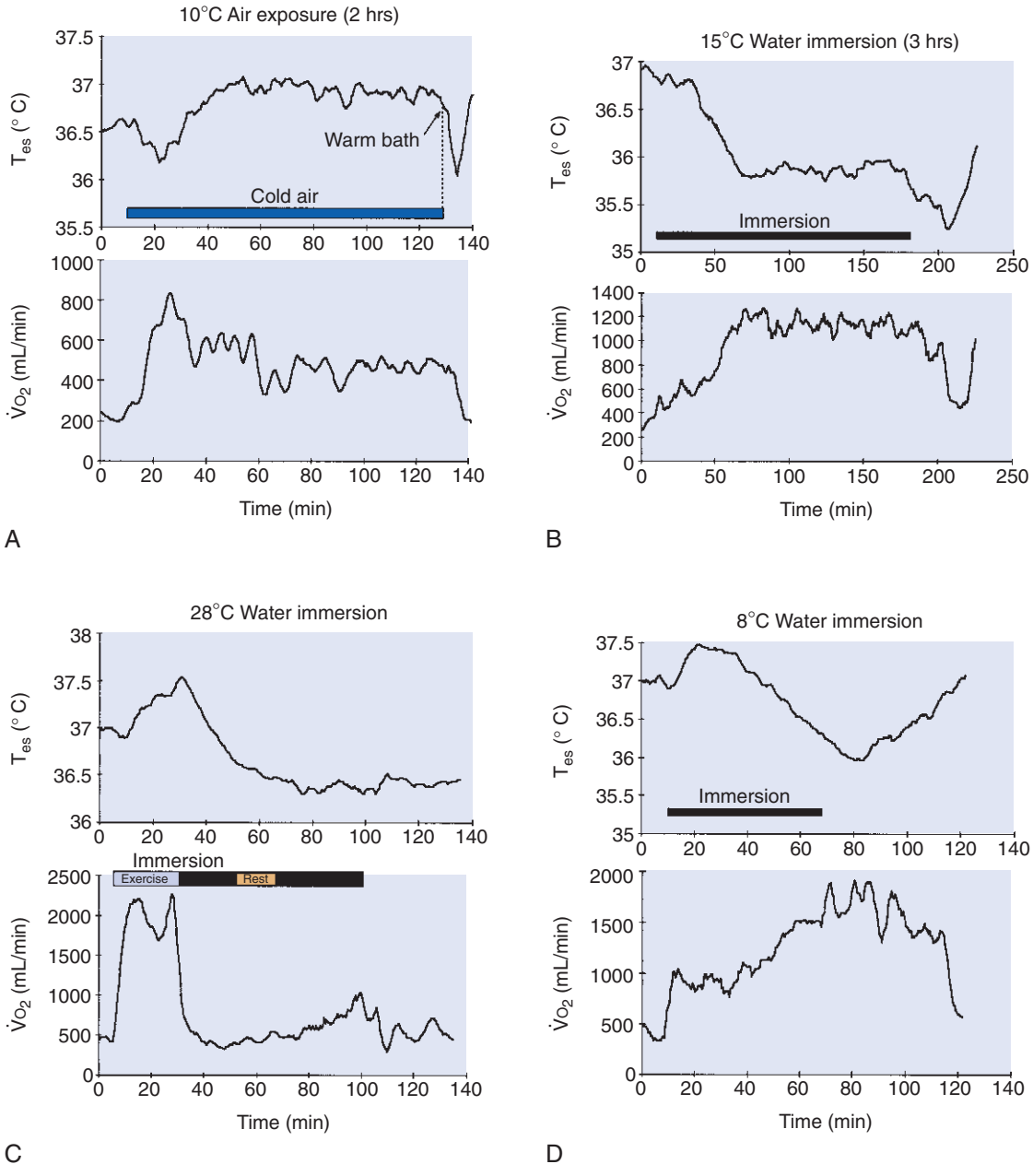


Figure 6-13. Effectiveness of shivering heat production in preventing onset of hypothermia during exposure to **A,** 10° C (50° F) air; **B,** 28° C (82° F) water; **C,** 15° C (59° F) water; and **D,** 8° C (46° F) water. T_{es}, esophageal temperature.

and colleagues demonstrated qualitatively similar effects of insulin-induced hypoglycemia.[132a]

Hypercapnia and hypoxia may also be present in various underwater scenarios. Hypercapnia has been shown to lower the shivering threshold[90] and transiently inhibit shivering itself.[109] Both hypercapnia[90] and hypoxia[91] have also been shown to accelerate core cooling.

Alcohol consumption is frequently associated with immersion hypothermia, as ethanol impairment of mental and motor performance is often the cause of accidental immersion. Social drinking can result in carelessness. Intoxicated mariners or others near water often fall from a boat, ship, gangway, wharf, or bridge into the water. Drunken drivers capsize or collide. On the basis of frequency of occurrence alone, the consequences of alcohol ingestion warrant special considerations.

Studies on the effect of moderate doses of ethanol (blood alcohol levels of 50 to 100 mg/dL, the range associated with legal impairment) on cold stress have established the following:

- Rate of heat loss is not significantly increased. Alcohol has a primary vasodilatory effect under normothermic conditions.[39,87,106] Under hypothermic conditions, when vasoconstriction predominates, alcohol lowers the vasoconstriction

threshold during the moderate cold stress of water immersion at 28° C (82° F) but does not impact the shivering threshold or rate of core cooling.[89]

- Rate of heat production is slightly decreased. For immersion in waters colder than 28° C (82° F), a moderate ethanol dose inhibits the metabolic response to cold.[42,112] Shivering thermogenesis is reduced in cold water by approximately 10% to 20%.
- Cooling rate is not significantly increased.[42,89,112] Because the body's cooling rate in cold water is influenced more by rate of heat loss than by rate of heat production, the slight reduction in shivering thermogenesis induced by moderate ethanol ingestion is outweighed by factors affecting heat loss (such as peripheral vasoconstriction and body fat). Because these do not vary with alcohol use, when the person is cold stressed, cooling rate does not change.
- Fatigue potentiates thermoregulatory impairment by alcohol. Exhaustive exercise leading to fatigue (characterized by hypoglycemia and depletion of glycogen reserves), combined with a moderate dose of ethanol, significantly reduces resistance to cold.[74,98] Alcohol inhibits gluconeogenesis,[73,93] so the ability to provide glucose to maintain shivering is reduced. If a person enters cold water in this condition, cooling rate is likely to be greater than in the absence of ethanol.
- Perception of cold is diminished. Experimental studies of humans in cold water show that moderate alcohol dosage to some extent relieves feelings of intense cold.[42,98] This cognitive alteration may be functionally related to the reduced shivering response.
- Cold-induced diuresis is increased. Alcohol inhibition of antidiuretic hormone augments immersion diuresis.[28] During the first hour of cold-water immersion, urine flow rate can be more than six times normal (approximately 8 mL/min). For longer immersions, alcohol potentiates the development of dehydration and hypovolemia.

For most people, high doses of alcohol (blood levels greater than 200 mg/dL) have an anesthetic effect. Major impairment of mental, motor, and involuntary function (including thermoregulation) occurs. Alcoholics who pass out in cold locations ("urban hypothermia") rapidly and passively become hypothermic (see Chapter 5).[30,118] When highly intoxicated persons enter cold water (usually by falling in), hypothermia is seldom a problem because they usually drown quickly.

COLD-WATER SURVIVAL

Cold-water survival depends on the avoidance of drowning and hypothermia, and on the many factors related to these risks,[150,151,155] including the ability to control the cold-shock response, the ability to swim and maintain airway freeboard, the availability and type of PFD, the availability of a life raft or other floating object to increase buoyancy, the behavior of the survivor in water, the decision to swim for shore or wait for rescue, the availability of signaling devices (whistles, flares, strobe lights, radios, mirrors) and the ability to use these devices, and the proximity of rescue personnel.

Ability to Control the Cold-Shock Response

As previously discussed, sudden immersion in cold water initiates a cardiorespiratory cold-shock response that significantly potentiates the risk of drowning. These reflexes and the resulting incapacitation that can accompany them have been suspected as the primary cause of drowning after short-term (<10 minutes) immersion in cold water.[161] The abrupt tachycardia and hypertension induced by sudden immersion in cold water can produce incapacitating cardiac dysrhythmias in susceptible individuals, and myocardial infarction or cerebrovascular accident in persons with arterial disease or hypertension.[161] In addition, reflex gasp response and hyperventilation[161,170] significantly shorten breath-holding duration.[81,85,161] Figure 6-14 illustrates this phenomenon. Loss of breath-holding capacity can have severe consequences for survivors attempting underwater egress from a submerged vehicle, capsized vessel, or aircraft, or for survivors simply trying to maintain airway freeboard in a rough sea or white-water river.[85,140,165]

The respiratory difficulties induced by the cold-shock reflexes make breathing while swimming extremely difficult. Golden and Hardcastle have demonstrated a "swim stroke/respiration asynchrony" leading to water inhalation and swimming failure,[67] and Golden and coworkers have shown that even persons who are considered good swimmers (at least in warm

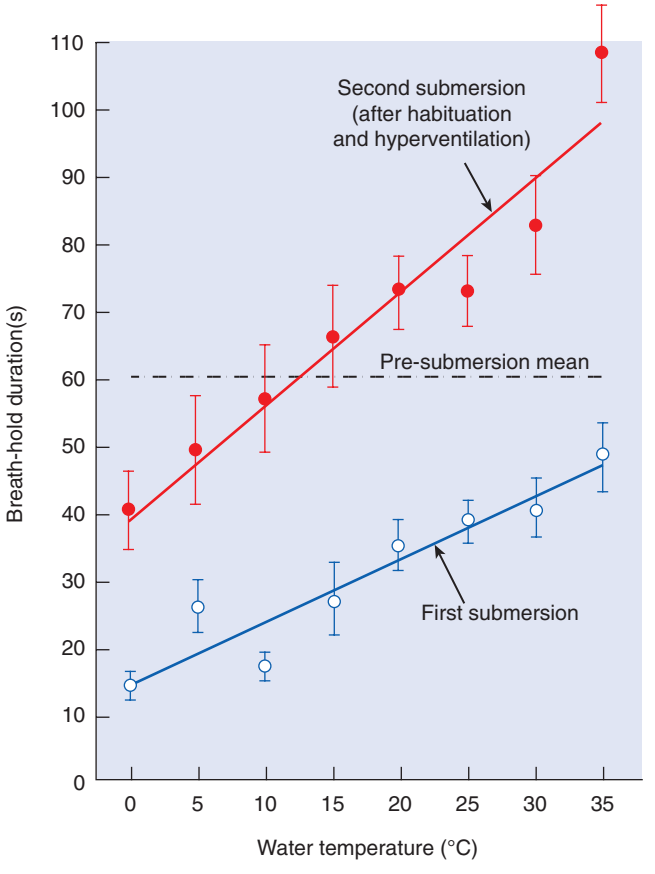


Figure 6-14. Effect of water temperature on maximum breath-hold duration in young, physically fit subjects (80 men and 80 women). First submersion was sudden after sitting comfortably in air at a mean temperature of 11.3° C (52° F). Second submersion followed 2 minutes of acclimatization to the water. The last 10 seconds of the acclimatization was accompanied by 10 seconds of hyperventilation. (From Hayward JS, Hay C, Matthews BR, et al: J Appl Physiol 56:202–206, 1984, with permission.)

water) can swim in cold water for only a few minutes.[67] Swimming ability and survival time in cold water is further diminished by subjective perception of shortness of breath and by panic reactions from unexpected cold-water immersion.[161] Furthermore, the work required to swim in cold water is greater than that in warm water, because of the former's higher viscosity (e.g., water at 4.7°C [40°F] has a viscosity 67% higher than water at 23.7°C [75°F]).[100] The increased work of swimming in cold water potentiates the onset of fatigue. The combination of all these factors may incapacitate even physically fit, capable swimmers. Keatinge and colleagues observed sudden incapacitation in two experimental subjects (who were good swimmers) immersed in 4.7°C (40°F) water. One inhaled water because of respiratory difficulty and fatigue after swimming for 7.5 min and had to be pulled out of the water. The second subject lasted only 1.5 minutes before he "floundered and sank without managing to reach the side of the pool, which was about 1 meter from his head, and had to be pulled" out of the water.[100] The observed frequency of such swimming failures and unexpected submersions has led the U.S. Coast Guard to coin the term *sudden disappearance syndrome* to describe the phenomenon.[173]

The magnitude of the cold-shock response can be attenuated through habituation to cold immersion. Habituating subjects to 10°C (50°F) water resulted in a 16% reduction in respiratory rate during the first 30 seconds of immersion in 10°C (50°F) water and a 26% reduction in respiratory rate during the 30- to 180-second interval after immersion in 10°C (50°F) water. Tidal volume and heart rate response showed similar declines in habituated subjects.[167] Habituation through cold-water survival training may thus be an important safety measure for workers in cold-water environments.

Protective clothing also diminishes the cold-shock response from initial immersion in cold water. Clothing that limits the amount and ingress velocity of water reaching a subject's skin (e.g., well-fitted wetsuits, and drysuits with adequate seals) significantly decreases the cardiorespiratory reflex responses to sudden immersion.[115,168,169,171] Furthermore, subjects wearing a wetsuit that protected the torso but left the limbs exposed had a significantly reduced respiratory reflex response but not a reduced heart rate response compared with control subjects with neither torso nor limbs protected. Tipton and Golden concluded that the limbs may be more important than the torso in the cardiac response to sudden cold-water immersion.[168] Tipton and coworkers also demonstrated that even loose-fitting, poorly insulated clothing can attenuate the magnitude of the cold-shock response compared with immersion in swimming trunks only. When volunteers were immersed in 10°C (50°F) water wearing either conventional clothing or conventional clothing plus windproof/shower-proof foul-weather clothing, the cardiopulmonary and thermal responses were significantly less than those found in those wearing swimming trunks alone.[169]

Mental preparation should be used to avoid panic, to decrease the emotional response, and to make a conscious effort to control breathing.

Ability to Swim and Maintain Airway Freeboard

Drowning is the most immediate survival problem after entry into cold water. To maintain airway freeboard and to avoid drowning, a survivor must possess the physical skills and psychological aptitude to combat the effects of wave action.[150,165] Although a PFD assists in maintenance of airway freeboard, waves can still submerge a survivor's head, even in moderately calm seas.[62,151,173] To reduce the risk of drowning in rough seas, a survivor can increase effective airway freeboard by partially exiting the water (e.g., by clinging to an overturned vessel or other debris floating in the water) or by climbing totally out of the water into a life raft or onto a capsized vessel. In both these environments, the survivor may still have to cope with the effects of cold wind, spray, and waves.

Behavior of the Survivor in the Water

Survivor location also has a significant effect on core temperature cooling rate and survival time. The U.S. Coast Guard and other rescue organizations recommend that a survivor of a maritime accident in cold seas get as much of the body out of the water as possible to minimize cooling rate and maximize survival time.[155,173] This recommendation derives from the higher thermal conductivity of water compared with air at the same temperature. However, survivors exposed to cold air are still at risk from hypothermia secondary to convective, evaporative, and radiant heat losses. In a rough sea environment, wind increases the magnitude of convective heat loss, and spray and periodic wetting from breaking waves result in conductive heat loss.[158,160] Steinman and colleagues confirmed the earlier observations.[155] The cooling rates of thin male subjects, wearing different types of protective clothing, were compared for three survival situations:

1. Immersion in 6°C (43°F) water with 1.5-m (4.9-ft) breaking waves
2. Exposure to 7.7°C (46°F) air, continuous water spray at 6°C (43°F), continuous 28 to 33 km/hr (17.4 to 20.5 miles/hr) wind, and occasional breaking waves while sitting atop an overturned boat (Fig. 6-15)
3. Exposure to 7.7°C (46°F) air and occasional breaking waves while sitting in an open, one-man life raft

The results of the study are shown in Figure 6-16. For each type of garment worn, cooling rates were considerably faster in the water than atop the boat (despite the effects of wind, spray, and breaking waves) or within the raft.

Survivors should therefore attempt to get as much of their bodies out of the water as possible, even if it means exposure

Figure 6-15. Testing protective clothing atop an overturned boat while exposed to wind, spray, and breaking waves. (Photo courtesy Alan Steinman, U.S. Coast Guard.)

Figure 6-16. Mean linear cooling rates (° C/hr) for lean men in three survival environments. *Water:* Immersion in 6.1° C (43° F) breaking waves. *Boat:* Immersion for 5 minutes in 6.1° C (43° F) water followed by exposure to 7.7° C (46° F) air atop an overturned boat with continuous 28 to 33 km/hr (17.4 to 20.5 miles/hr) wind, water spray, and occasional breaking waves. *Raft:* Exposure to 7.7° C (46° F) air in an open, one-man life raft, preceded by 5-minute immersion in 6.1° C (43° F) water. AC, aircrew coveralls; BC, boat-crew coveralls; FS, flight suit (lightweight clothing); NI, intact, non-foam-insulated, "dry" coveralls; NX, NI with a 5-cm tear in the left shoulder, permitting water to leak into the suit and degrade its insulation; WS, wetsuit. *Blank squares* indicate combinations of garment and environment that were not tested. (From Steinman AM, Kubilis P: Report No. CG-D-26-86, USCG, National Technical Information Service, 1986, with permission.)

to cold wind and spray. Even rescue and medical personnel who frequently work in wilderness environments poorly understand this recommendation.

A widespread misunderstanding of the concept of windchill[125] causes many to conclude that survivors have higher heat losses if they are exposed to wind, especially if they are wet, than if they are immersed in water.[155] Windchill (a term originally used by Siple and Passel[146] to describe the increase in heat loss from unprotected skin exposed to wind) is frequently used in the communication media without regard to the difference between exposed and unexposed skin. This misleads many to believe that the windchill temperature applies to both clothed and unclothed areas of the body. Furthermore, common experiences during recreational activities at the beach, lake, or swimming pool, where people subjectively feel colder after leaving the water (because of evaporative heat loss from the skin) than they do while swimming, reinforce the misunderstanding. This has occasionally led survivors to abandon a position of relative safety atop a capsized vessel and reenter the water, usually with tragic results. The sensation of coldness (which is skin dependent) does not reliably convey information about rate of heat loss when two radically different environments such as air and water are compared.

Decision to Swim for Shore or Wait for Rescue

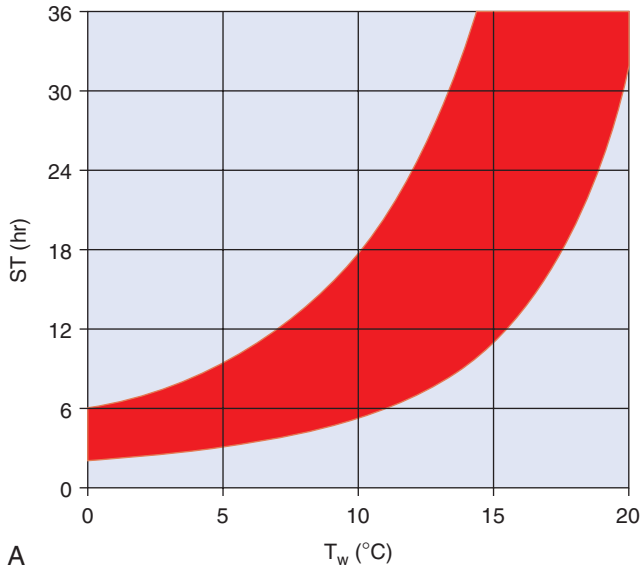
In cold-water immersion, physical activity increases heat loss through increased blood flow to the periphery. This is especially pertinent when immersed victims engage in excessive movement in the water (e.g., swimming or vigorous extremity movements necessary to maintain airway freeboard in rough seas).

The decision to swim for shore or wait for rescue is crucial for a survivor of cold-water immersion. The increased cooling rate associated with swimming might lead one to conclude that holding still is preferable to an attempt to swim for safety. Yet if the survivor can make it to shore or even to shallow water, survival is likely to be prolonged, despite the risk of lower core temperature. Lonsbury and coworkers (unpublished master's thesis) specifically evaluated this issue by comparing the cooling rates and swimming distances for both novice and expert swimmers in a swimming plume. Experimental subjects, wearing average clothing (e.g., no specific insulation against cold-water immersion) and a PFD, were immersed in 10° C (50° F) water and either remained still in a HELP position or swam against a current matched to their swimming ability. Swimming resulted in a 17% faster rate of core cooling compared with holding still. However, the novice swimmers traversed about 800 m and the expert swimmers traversed about 1400 m during their swims. Thus, swimming might well have allowed the subjects to achieve safety ashore, despite the faster cooling rate. They also found that the probability of a successful survival outcome was dependent on a decision to swim for safety early in a survival event. If the subjects had cooled for 30 minutes prior to attempting to swim, the likelihood of success was significantly reduced.

The decision to swim or remain still ultimately depends on a survivor's understanding the many variables affecting a successful outcome: body morphology and cooling rate, water temperature, wave conditions and currents, proximity of shore, proximity of rescue personnel, swimming ability, and availability of signaling devices, flotation devices, and protective clothing. There is no uniformly correct answer to the question, "Should I swim for it?" Each survival situation must be evaluated individually.

Survival Modeling

Tikusis recently devised a more sophisticated set of survival time estimates for individuals immersed in cold, rough seas and for survivors partially immersed or exposed to cold wind under wet conditions.[158,159] Using computer modeling of human cooling physiology and the experimental data of Steinman and colleagues[154,155] shown earlier, he developed a series of survival-time charts for a range of individual body morphologies while wearing various types of protective clothing.[159] Figure 6-17 shows his estimates for lean and fat individuals wearing a loose-



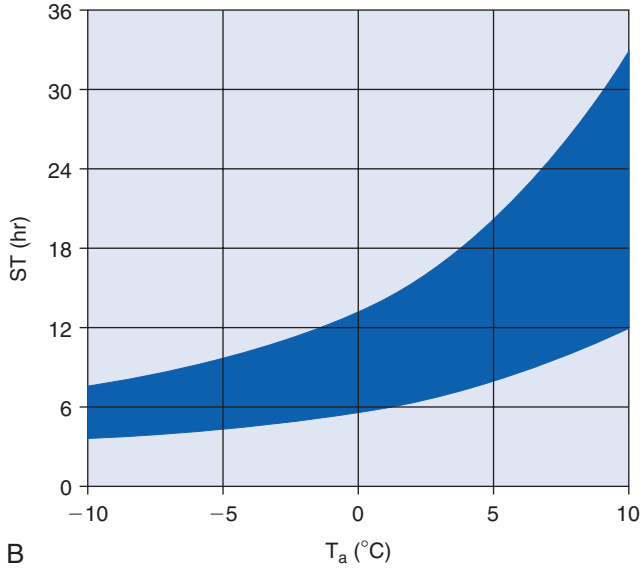
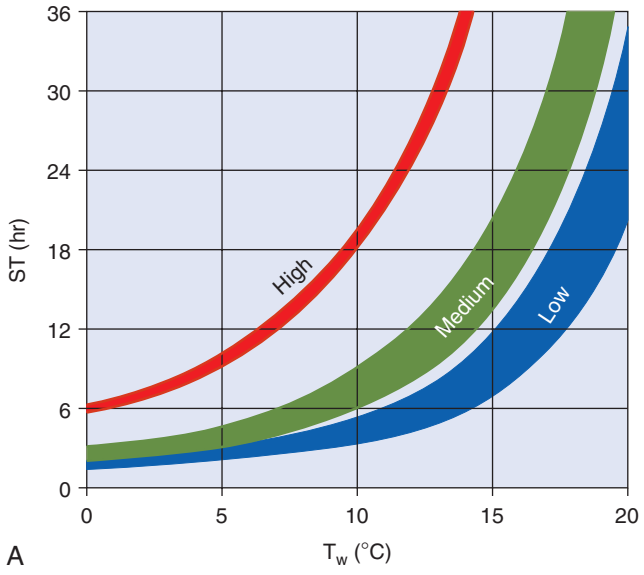


Figure 6-17. Predicted survival time for individuals wearing the boat-crew coverall (see Figure 6-7) for immersion in rough seas versus water temperature **(A),** and exposure to air at 20 km/hr under wet conditions (clothing wetness of 1550 g/m^2) versus air temperature **(B).** The lower and upper boundaries of the *shaded regions* represent predictions for lean and fat individuals, respectively. ST, survival time; Ta, time in air; Tw, time in water. (From Tikuisis P: Aviat Space Environ Med 68:441–448, 1997, with permission.)



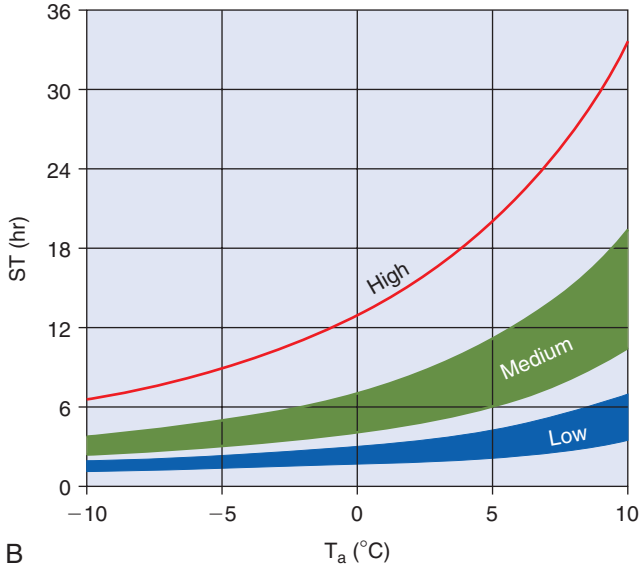


Figure 6-18. Prediction of survival time for an average individual under various degrees of clothing protection (see Figure 6-7). **A,** Immersion in rough seas plotted against water temperature. *Low,* Nude, flight suit, float coat, aviation coverall, boat-crew coverall, torn coverall. *Medium,* Short wetsuit, full wetsuit. *High,* Dry coverall, drysuit. **B,** Exposure to air at 20 km/hr under wet conditions plotted against air temperature. *Low,* Nude, flight suit, aviation coverall. *Medium,* Float coat, boat-crew coverall, short wetsuit, full wetsuit, dry coverall, torn coverall. *High,* drysuit. ST, survival time; Ta, time in air; Tw, time in water. (From Tikuisis P: Aviat Space Environ Med 68:441–448, 1997, with permission.)

fitted, insulated coverall (e.g., boat-crew coverall, snowmobile suit; see Figure 6-7). Tikusis defined the lean, average, and fat survivors as having the following morphologic characteristics: height, 1.77 m; weight, 66.3, 73.9, and 88.2 kg, respectively, for lean, average, and fat; and body fat, 11.2%, 17.7%, and 28.6%, respectively, for lean, average, and fat. Note that the lean definitions fit the experimental subjects used by Steinman and Kubilis[155] (see Table 6-4). Tikusis also published a series of survival-time charts for the average individual (as just defined) wearing various types of clothing protection. Figure 6-18 shows his results for low-insulation clothing (nude, float coats, average clothing, boat-crew coveralls, leaky drysuits), medium-insulation clothing (partial or full wetsuits), and high-insulation clothing (dry, insulated coveralls and survival suits). Both the Canadian and the U.S. Coast Guard in their search and rescue missions are now using these survival charts.

PHYSIOLOGIC RESPONSES TO COLD-WATER SUBMERSION (HEAD UNDER WATER)

Cold-water submersion or near-drowning is covered in detail in Chapter 68, but a brief summary is included here to complete the discussion of cold-water immersion. There have been several recent advances in our understanding of why individuals can survive cold-water submersion for as long as 66 minutes[10] with full or partial neurologic recovery (cold-water near-drowning). The most important factor in these unusual cases is the low water temperature and the subsequent brain cooling that results. This principle has been used in clinical practice for years. For example, cardiopulmonary bypass was used to cool neurosurgical patients to a core temperature of about 9° C (48° F), and it was then possible to arrest brain blood flow for at least 55 minutes with full neurologic recovery.[131] The full explanation for these recoveries relates to both (1) the mechanisms for, and amounts of, brain/body cooling that occurs, and (2) the mechanisms for the protective effect of this cooling.

Mechanisms for Brain/Body Cooling

Children have an advantage in cold-water submersion incidents because their greater surface-area-to-mass ratio allows faster conductive cooling, which provides cerebral protection based on decreased cerebral metabolic requirements of oxygen ($CMRO_2$). The mammalian dive reflex, which initiates intense bradycardia and shunts blood flow to important core organs such as the heart and brain, has also been implicated. A third and recently explored factor is the possibility that cold-water ventilation may result in a rapid and extensive cooling during submersion (see later).

The effectiveness of the human dive reflex, especially the breath-hold response, is controversial. Nemiroff, who has one of the largest series of successful resuscitations of near-drowning victims, writes that the dive reflex plays an important role, particularly in children and infants, and especially neonates.[123] Hayward and colleagues, on the other hand,[85,134] believe that the enhanced success of resuscitation associated with cold-water near-drowning is due more to hypothermia than to the dive reflex.

The protective effect of cooling depends on the T_{co} at cessation of oxygen delivery and the subsequent rate and extent of the decrease of T_{co}. Because T_{co} is likely to be near normal at the onset of an accidental submersion, rapid cooling after onset of ischemia is important for survival. Conduction alone probably cannot account for the rapid decrease in T_{co} that occurs in cold-water submersion incidents. Conn and coworkers[24] studied cold-water [4° C (39° F)] drowning in shaved, anesthetized dogs. They found that submersed dogs continued to breathe the cold water for an extended period. Core temperature decreased 11° C (52° F) in 4 minutes in the completely submersed dogs, but only 3° C (37° F) in the control (immersed with head out) dogs who did not breathe cold water. It is likely that the rapid cooling was caused by convective heat exchange in the lung as opposed to only surface conduction in the control dogs. The general conclusion that brain cooling is accelerated considerably by respiration of cold water has been proposed by others[51,65,72] and mathematically predicted by Xu and colleagues.[200] Although breath-holding may occur during submersion, a physiologic break point occurs, where involuntary breathing movements predominate.[5] This factor, coupled with unconsciousness, could reasonably be expected to result in respiration of water under at least some circumstances. Experimental and anecdotal evidence in humans is rare. However, one helicopter crash survivor reported that, after being trapped underwater for some time, he recalled feeling that he was about to die and that he was breathing water in and out just prior to escaping the cockpit.[14]

Mechanisms for the Protective Effect of Brain Cooling

Hypothermia provides an advantage during anoxic periods such as cold-water submersion because of what is known as the metabolic icebox. Whole-body or focal hypothermia has long been used to extend the biologic survival time during surgery under ischemic conditions. Cerebral protection under hypothermic conditions has commonly been attributed to the decreased cerebral metabolic requirements of oxygen according to the Q_{10} principle (Q_{10} being the factor by which the metabolism of tissue increases with a temperature increase of 10° C [18° F]). Although the Q_{10} of the whole body is about 2, the Q_{10} of the brain increases from about 3 between 27° and 37° C (81° and 99° F) to 4.8 between 18° and 27° C (63° and 81° F).[117] Thus, if the brain could survive an ischemic insult for 5 minutes at 37° C (99° F), cooling to 27° C (81° F) or to 17° C (63° F) would provide 15 and 72 minutes of protection, respectively, solely on the basis of the decreased $CMRO_2$ (Fig. 6-19). Although long survival times at brain temperatures below 20° C (68° F) may be predicted on the basis of increasing Q_{10} and diminishing $CMRO_2$, some additional mechanism(s) may be required to explain intact survival after prolonged submersion in which reported core temperatures are often above 30° C (86° F) (these factors are schematically shown in Figure 6-20). Over the past decade, several studies, directed mainly toward protection of the brain during or after cerebral ischemic events, have demonstrated that even moderate brain cooling of 3° to 5° C

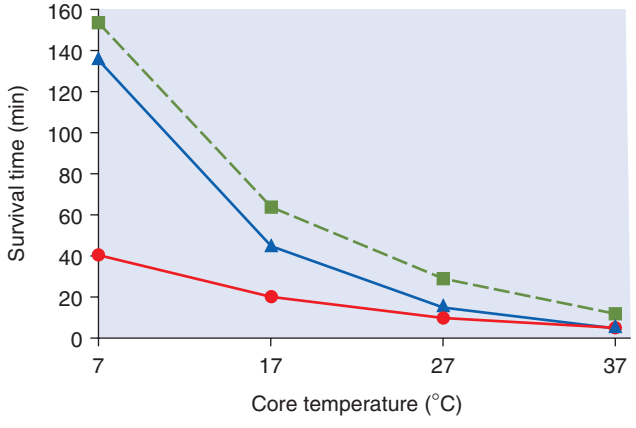


Figure 6-19. Schematic presentation of survival time at different core/brain temperatures. **A** (*red line*), Prediction based on a Q_{10} of 2. **B** (*blue line*), Prediction based on a Q_{10} of 3. **C** (*green line*), Prediction based on a Q_{10} effect plus other factors such as changes in neurotransmitter release (e.g., glutamate and dopamine). Actual survival times are from both neurosurgical and accidental hypothermia cases. Most of these cases fall outside the range predicted based on Q_{10} alone, so some other factors must also contribute to survival. Q_{10} is the factor by which the metabolism of tissue increases with a temperature increase of 10° C (18° F).

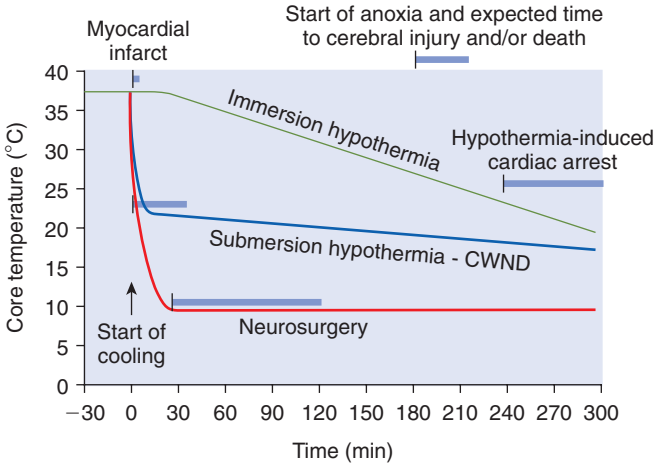


Figure 6-20. Schematic representation of theoretical times to cerebral injury or death after sudden onset of anoxia. Examples include anoxia induced by myocardial infarction at normal core temperatures, hypothermia-induced cardiac arrest during immersion hypothermia, ischemia-induced cardiac arrest after cold-water near-drowning (CWND), and electrically induced arrest after protective cooling for neurosurgery. Note: The times shown are representational; actual survival times may vary.

(5° to 9°F) provides substantial cerebral protection from ischemic insult.[16,17,61,120,142,199]

Submersion probably promotes cerebral death, as tissue ischemia depletes high-energy phosphates and leads to membrane depolarization. This stimulates release, into the extracellular space, of excitatory neurotransmitters such as glutamate and dopamine, which mediate postsynaptic depolarization, causing calcium entry into the cell. Calcium influx mediates the production of oxygen and hydroxyl free radicals (which may be involved in reperfusion injury), and the release of free fatty acids, resulting in eicosanoid synthesis.[172] Various animal studies have shown that cooling the brain by only 3° to 5°C (5° to 9°F) prior to ischemia (1) delays terminal depolarization and reduces the initial rate of rise of extracellular potassium,[94] (2) results in complete suppression of glutamate release and a 60% reduction in the peak release of dopamine,[17] (3) attenuates the ischemia-induced damage to endothelial cells,[32] (4) reduces hydroxyl radical production,[63] and (5) improves postischemic glucose utilization.[61] These results relate to mechanisms other than decreased $CMRO_2$, which may explain cerebral protection during cold-water near-drowning.

In summary, these findings indicate that (1) if the brain cools even by only 3° to 5°C (5° to 9°F), it is protected in excess of what would be predicted from decreased $CMRO_2$ alone; (2) protection results from additional protective mechanism(s) of cooling related to neurotransmitter release, calcium flux, eicosanoid synthesis, and so on; (3) the mammalian dive reflex may result in some cold-induced circulatory adjustments that favor conservation of oxygen for the heart and brain; and (4) any cold-water ventilation may accelerate brain cooling and provide further protection.

Implications for Survival

The paradox of whether core cooling is an advantage or a disadvantage depends on whether there is a cessation of oxygen delivery, and if so, what T_{co} is when anoxia occurs and how much T_{co} subsequently declines. These factors are schematically illustrated in Figure 6-20. If oxygen delivery is *not* compromised (e.g., in immersion hypothermia), then core cooling will eventually lead to death from cardiac arrest. However, if oxygen delivery *is* compromised (e.g., in submersion hypothermia and cold-water near-drowning), core/brain cooling will prolong the survival compared with a condition where core cooling does not occur (e.g., in myocardial infarction). These factors are important in understanding whether children have a survival advantage over adults during cold stress. Because children cool faster, their body size would be an advantage when oxygen supply is compromised during cold-water submersion. However, when the oxygen supply is uninterrupted in head-out immersion or in cold air, the small body size becomes a disadvantage because the onset of severe hypothermia will be faster.

RESCUE

Self-Rescue from Open Water or an Ice Hole

Most people have a poor understanding of how their body will react during cold water immersion. Although there is individual variability, the following general principles apply. First, get breathing under control, as gasping and hyperventilation may result in gulping in water and even drowning. One should be able to control breathing within 30 seconds to 2 minutes. In ice-cold water, loss of arm control and loss of the ability to grasp is progressive and begins to occur within the first 3 to 15 minutes. In open water, self-rescue may involve pulling oneself onto a floating object and out of the water as much as possible. If a person has fallen through the ice, it is usually best to turn and face the direction from which he came. (The ice traveled over has already been proved capable of holding your weight.) Swim to the ice ledge on the entry side of the hole.

The self-rescue exit is illustrated in Figure 6-21. It is initiated by putting the arms up on the ice and getting as much of the upper body out of the water as possible. Kick the legs vigorously to bring the body into a horizontal position. Now, kick and pull yourself forward and get on top of the ice. Once up on the ice edge, it is best to crawl or roll away from the hole until the ice is thick enough to stand on. It is possible to use ice picks, car keys, a knife, or any other available object to get a better grip in the ice.

Once on solid ice, remember that hypothermia and frostbite pose a threat to survival. The first thing to do is to wring the water out of clothing as soon as possible. It might seem that this would make one colder, but wringing out the clothing increases its insulation value, which is best in the long run. Then decide whether to stay and try to get warm and dry by a fire, or to go and seek shelter. If shelter is reasonably close (i.e., on average, less than 30 minutes of exposure to the cold air), it may be beneficial to walk to safety; if it is farther than 30 minutes away, it may be better to stay and build an emergency fire and campsite.

It is extremely important to protect hands from becoming frostbitten. Wring water from gloves/mitts. If the fingers are still very cold, place each hand in the opposite armpit where it will be insulated. If necessary, pull arms inside the jacket to place hands directly against the skin of the armpit. Once hands have recovered, it is imperative to start a fire to warm up and dry the clothing.

Although a person who cannot get out of the cold water unaided might not yet be very hypothermic, physical incapaci-

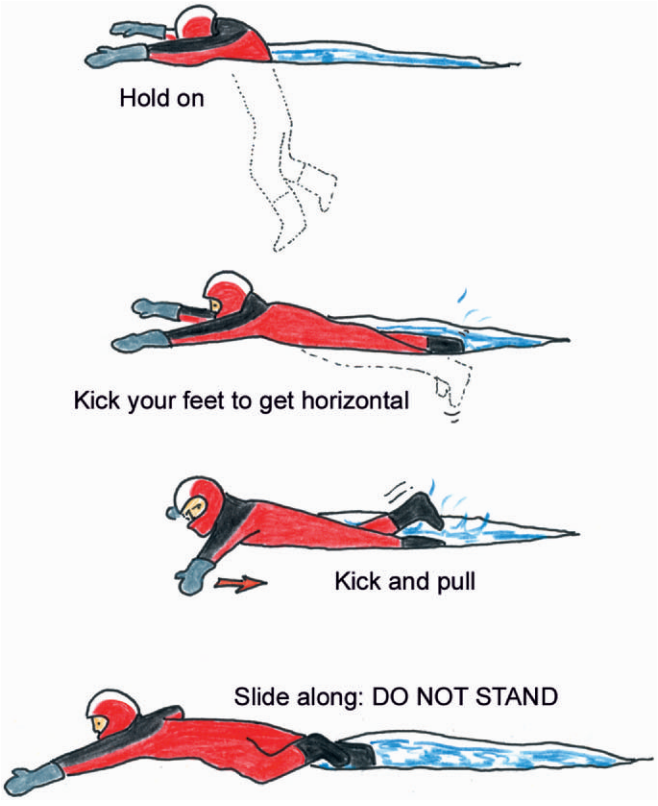


Figure 6-21. Self-rescue technique after falling through ice. Place arms on the ice and kick feet (flutter kick) until the body is horizontal on the water surface. Then kick vigorously with the legs and pull the body along the ice surface. Slide or roll away from the hole, and do not stand up until sure that the ice is thick enough to hold body weight. (Courtesy Wilderness Medicine Newsletter, available at http://www.wildernessmedicinenewsletter.com.)

tation will only get worse within 10 to 15 minutes. However, consciousness will remain for an hour or so, until core temperature decreases from 37° to about 30°C (99° to about 86°F), so survival time must be extended to widen the window of opportunity for rescue. The following principle illustrates cold water responses during cold water immersion (time estimates are subject to individual variability):

1-10-1 Principle

1. You have 1 minute to get your breathing under control.
2. You have 10 minutes of meaningful movement.
3. You have 1 hour until you become unconscious due to hypothermia.

Swimming should be minimized, because it causes the body to lose heat much faster than remaining as still as possible, and it also causes exhaustion. Clothes should be kept on in the water, as they help conserve body heat, and although the air initially trapped in the clothing will eventually be forced out, it helps with flotation while it remains. Wet clothing does *not* cause a person to sink. The main effect of water in clothing is that its inertia makes movement difficult. The weight of water in clothing becomes a problem only when trying to exit the water.

In all cases (even if wearing a thermal protection suit or a PFD), use the ice edge for support. Extend the arms over the ice and press the legs together to help conserve body heat. Keep the head and upper body as far out of the water as possible to conserve as much body heat as possible. Remain as still as possible. This will decrease heat loss, but it may also result in arms freezing to the ice. After about 1 hour, when consciousness is lost from severe hypothermia, drowning occurs unless the head is prevented from slipping beneath the water. If one is frozen to the ice, drowning will be prevented, and a victim might survive yet another hour before the heart stops beating (generally at a core temperature below 28° or 25°C [82° or 77°F]). Thus, this could double the survival time until rescue comes.

Assisted Rescue of a Victim Who Has Fallen through the Ice

The self-rescue and the assisted rescue both have three guidelines: acknowledge the danger of the situation, assess the situation, and follow the rescue sequence.

Recognize the Danger

1. Recognize that ice conditions are unsafe and that no one else should approach the area.
2. Recognize that the victim is in an urgent situation that is potentially life threatening, but that if proper action is taken, there is more time than might be expected to make safe, effective decisions.
3. Recognize your own limitations in terms of training and equipment.

Assess the Situation

1. Assess the victim's physical and emotional condition.
2. Assess the ice and water conditions (e.g., ice thickness and water current).
3. Assess the equipment you have that might be useful in a rescue.
4. Assess the rescue skills you (and those with you) have.

Follow the Rescue Sequence

The rescue sequence progresses from lowest-risk actions to high-risk actions. Low-risk actions should be taken first, and progression to higher-risk actions should be dictated by training level and equipment available.

1. Shore-assisted rescues involve talking, throwing, and reaching (untrained bystanders should limit themselves to these techniques).
2. Platform-assisted rescues involve working from, or with, some flotation platform such as inflatable boat or other buoyant device.
3. Finally, in "Go" rescues, the rescuer approaches and contacts the victim. This should only be done by trained personnel wearing flotation devices and a thermally protective drysuit.

Rescuers should always perform the lowest-risk rescue first, and contact professional authorities if possible.

Untrained Bystander Rescue

Every year, newspaper articles describe the death of well-meaning bystanders who rush to help victims who have fallen through the ice. It is extremely important that the untrained bystander apply the acronym STOP: stop, think, observe, and plan.

Stop and resist the urge to rush up to help the victim, as you are likely to fall through the ice and become a secondary victim. *Think* and *observe* the victim and the surrounding conditions, including any material that might be used in a rescue attempt. Finally, *plan* how to assist the victim out of the water. Untrained bystanders should limit themselves to techniques progressing from talking to throwing and then to reaching.

Talk. Encourage the victim to calm down and not panic. Direct the victim to an area of strong ice and instruct the victim to execute a self-rescue by placing the arms on the ice, kicking until the body is horizontal on the water surface, and then kicking and pulling up onto the ice. Direct the victim to crawl and roll away from the hole until stronger ice is reached.

Throw. Any buoyant item can be thrown to the victim to assist in remaining buoyant, especially if the ice is continually breaking and is not providing a secure base of support. If possible, tie a rope or cord to the object. This could allow you to pull the victim out of the water. If only a cord or rope is available, tie a large loop at the end to make it easier for the victim to hold on to it. Once the rope has been grabbed, instruct the victim to put the loop over the body and under the arms, to put an arm through the loop and bend the elbow around the rope, or to simply hold on to the loop.

Reach. It may be possible to reach a victim with some implement, such as a tree branch, a ladder, or anything else that can be pushed out on the ice. Care must be taken not to get too close to the hole when trying to reach the victim with any implement. Any rescue that requires the rescuer to approach the hole in the ice should be left to trained personnel.

An untrained bystander who cannot effect a rescue by these techniques should help the victim widen the window of opportunity for trained rescuers to arrive. Instruct the victim to stop struggling and to hold the arms still on the ice; the object is to let them freeze to the ice. Make sure that someone contacts emergency personnel. Wait with the victim and provide continuous encouragement that they can survive and that help is coming.

Rescue by Trained Personnel

These paragraphs are not meant to provide in-depth training for professional rescue workers but to describe some of the basics of some high-risk rescues. The techniques used by any professional personnel should follow the policies and procedures of their local jurisdictions.

Trained personnel should first use the lower-risk techniques described earlier. If these do not work, they should resort to the higher-risk techniques described by many ice-rescue training institutions.[104] Briefly, however, trained rescuers must not go on the ice unless they are wearing a thermal protective drysuit and a PFD. They should also be secured by a safety rope, which is operated by a trained colleague.

Platform-assisted rescues involve working from, or with, some flotation platform such as an inflatable boat or other buoyant device. These techniques involve pushing, or being transported on, the buoyant platform. The first choice is to push the platform toward the victim and use it as a reaching device. Alternatively, the rescuer can make contact with the victim directly from the platform.

Finally, in "Go" rescues, the rescuer approaches and contacts the victim directly. Standard lifesaving techniques should be used to ensure that a panicking victim does not pose a threat to the rescuer. Once the victim is securely contacted, the victim and rescuer can be pulled toward shore by the attendants on shore.

Rescue from Open Water

Retrieval of a victim from cold-water immersion must be performed with caution. Sudden reduction of the hydrostatic squeeze applied to tissues below the water's surface may potentiate hypotension, especially orthostatic hypotension.[71] Because a hypothermic patient's normal cardiovascular defenses are impaired, the cold myocardium may be incapable of increasing cardiac output in response to a hypotensive stimulus. A victim's vertical posture may also potentiate hypotension. Hypovolemia, secondary to combined cold- and immersion-induced diuresis, and increased blood viscosity potentiate these effects.[29] Peripheral vascular resistance may also be incapable of increasing, because vasoconstriction is already maximal as a result of cold stress. The result of sudden removal of a hypothermic patient from the water is similar to sudden deflation of antishock trousers on a patient in hypovolemic shock: abrupt hypotension. This has been demonstrated experimentally in mildly hypothermic human volunteers,[69] and it has been suspected as a cause of post-rescue death in many immersion hypothermia victims.[71,153] Accordingly, rescuers should attempt to maintain hypothermic patients in a horizontal position during retrieval from the water and aboard the rescue vehicle (Figs. 6-22 and 6-23).[4,71,153] If rescuers cannot recover the patient horizontally, they should place the victim in a supine posture as quickly as possible after removal from cold water.[153]

The patient's core temperature may continue to decline (depending on the quality of insulation provided, the patient's endogenous heat production, active or passive manipulation of extremities, and the site of core temperature measurement) even after the rescue. To diminish this, the patient's physical activity must be minimized. If practical, conscious patients should not be allowed to assist in their own rescue (e.g., by climbing up a scramble net or ship's ladder) or to ambulate once out of the water (e.g., by walking to a waiting ambulance or helicopter).[46,119,153] Physical activity increases afterdrop, presumably by increasing perfusion of cold muscle tissue with relatively warm

Figure 6-22. U.S. Coast Guard crew keep a victim of hypothermia in a horizontal position during rescue operations.

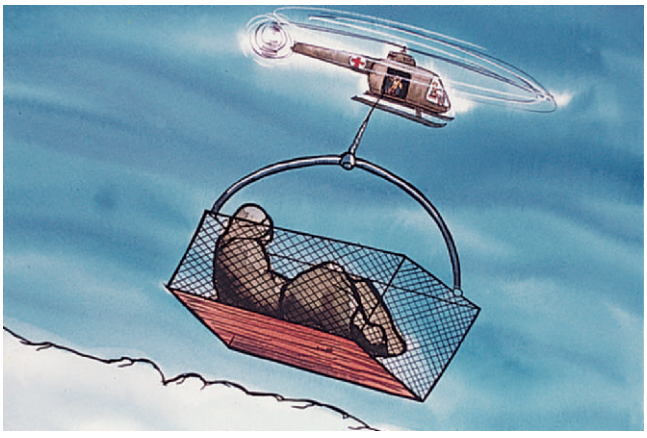


Figure 6-23. A victim of immersion hypothermia in a semi-horizontal posture during a helicopter basket hoist.

blood.[13,48,50,52] As this blood is cooled, venous return (the circulatory component of afterdrop) contributes to a decline in myocardial temperature, increasing the risk of ventricular fibrillation.[84] Mildly hypothermic volunteers (with an esophageal temperature of 33°C [91°F]) demonstrated a threefold greater afterdrop while walking on a treadmill than while lying still.[12,53] Such an exercise-induced enhancement of afterdrop could precipitate post-rescue collapse. Throughout the rescue procedures and during subsequent management, hypothermic patients must be handled gently.[4,119] Excessive mechanical stimulation of the cold myocardium is another suspected cause of death after rescue.[107,188]

MEDICAL MANAGEMENT

The prehospital management of hypothermia described here focuses specifically on immersion pathophysiology. Hospital management of both accidental immersion and land-based hypothermia is described in Chapter 5 and in cold injury guidelines.[2] Prehospital management of patients with hypothermia, both in the field and during transportation to a site of definitive medical care, varies with the patient's level of hypothermia, rescuer's level of training, resuscitative equipment available, type of transportation, and time required for delivery to definitive care. Medical personnel must exercise good clinical judgment in balancing all these factors to select appropriate therapeutic modalities.

The primary goals in prehospital management of victims of accidental immersion hypothermia are prevention of cardiopulmonary arrest, prevention of continued T_{co} decline, moderate core rewarming if practicable, and transportation to a site of definitive medical care.[34,75,153] Aggressive rewarming in the field is contraindicated, because the means to diagnose and manage the many potential complications of severe hypothermia are unavailable in this setting.[119] However, in the prehospital scenario of cold-water immersion, there is no way to supply heat that would be considered aggressive. Figure 6-24 indicates the level of aggressiveness and the effectiveness of various sources of heat for hypothermia and frostbite treatment. When transportation to a site of definitive care will take longer than 30 minutes or is impossible, rewarming in the field, using the principles and techniques of management described in the following paragraphs, may be appropriate.

Examination and Life Support

Because hypothermia affects virtually every physiologic process, rescuers should manage a severely hypothermic patient as they would a victim of multiple trauma.[152,153] They should not focus solely on core temperature to the exclusion of other potentially life-threatening problems. Conversely, when a trauma incident includes cold exposure, care must be taken to not focus solely on the trauma injuries. Patients have died of hypothermia when treatment was focused only on non–life-threatening physical injuries. In accordance with standard emergency medical procedures, the ABCs (airway, breathing, and circulation) of first aid are essential.[4,153] Rescuers should ensure an open airway and confirm the presence of adequate ventilation and circulation. If the patient is severely hypothermic, respiration and pulse may be slow, shallow, and difficult to detect.[4,152] Therefore, rescuers should take 60 seconds to assess these vital signs. If vital signs are not detected, 3 minutes of artificial ventilation should be administered; increased oxygenation my strengthen cardiac activity to detectable levels. Another 60 seconds should then be used to detect vital signs.[4] If neither pulse nor breathing is detectable, rescuers should commence cardiopulmonary resuscitation (CPR) in accordance with basic life-support protocols.[4,152]

Cardiac rhythm should be carefully monitored, if possible. Percutaneous electrodes may be required to overcome interference from muscle fasciculation or shivering.[4] Endotracheal intubation and the administration of heated, humidified air or oxygen are useful in the management of apnea or hypoventilation and in reducing further respiratory heat loss.[4] Mouth-to-mouth or mouth-to-mask ventilation also provides heated, humidified air. If oxygen is available, it may supplement either type of ventilation. Mouth-to-mouth or mouth-to-mask ventilation has the added advantage of providing a small amount of carbon dioxide to the patient's inspired gases. This may be useful in preventing hypocapnea secondary to relative hyperventilation in a severely hypothermic victim whose metabolic production of CO_2 is diminished.[30] This is important because hypocapnea may decrease the threshold for ventricular fibrillation.

Hypothermic patients with *any* detectable pulse or respiration do not require the chest compressions of CPR, even though severe bradycardia and bradypnea may be present.[4,152] This differs from normothermic CPR protocols, where chest compressions may be indicated if bradycardia fails to provide sufficient cardiac output or systolic pressure.[4] Because the metabolic requirements of hypothermic patients are reduced, the observed bradycardia and bradypnea may still meet tissue oxygen requirements.[31,46] Inappropriate administration of chest compressions in an attempt to augment cardiac output may precipitate ventricular fibrillation from mechanical stimulation of the cold, irritable myocardium.[31]

If a victim of immersion in cold water is found floating face-down, near-drowning should be suspected and managed accordingly (see Chapter 68). In this case, correction of anoxia is paramount, and consideration of hypothermia is of secondary importance.[4,123] Normal advanced cardiac life support (ACLS) protocols should not routinely be applied to severely hypothermic patients in cardiac arrest, because management beyond basic life support differs from that used in normother-

Power source	Heat source		Heat level/ Aggressive?	Treatment effectiveness	
	Pre-hospital	Hospital		Hypothermia	Frostbite
Non-electric	Fire		Low-to-moderate heat **Non-aggressive**	XX	XXX
	Chemical pack			–	√
	IV fluid			√	–
	Warm sweet drink			√	–
	Inhalation warming			–	–
	Warm water bottles			√	√
	Warm body			√	√
	Charcoal HeatPac			√√	√
Electric 120 VAC	Electric blanket	Electric blanket		√	√
	Water blanket	Water blanket		√	√
	Forced air warming	Forced air warming		√√	√
	Warm shower		High heat **Aggressive**	XX	–
	Warm bath	Warm bath		XXX	–
		Lavage		√√√	–
		CAVR		√√√	–
		Fem-fem		√√√	–
		Bypass		√√√√	–

√, Effectiveness –, Not effective or applicable X, Harmful

Figure 6-24. Heat sources that could be used for hypothermia rewarming, classified by location, heat level or aggressiveness, and treatment effectiveness for hypothermia and frostbite.

mia.[4,31] Defibrillation and pharmacologic interventions are usually ineffective for myocardial temperatures below 30°C (86°F).[4,8,31] Furthermore, repeated defibrillatory shocks may damage the myocardium.[4,152] Defibrillation should be limited to three shocks at 200 joules (J), 300 J, and 360 J, consecutively, in patients colder than 30°C (86°F).[4] Administered medications are not only ineffective but may accumulate to toxic levels, because drug metabolism by the hypothermic liver and kidneys is reduced.[31,192] For hypothermic patients with a core temperature of greater than 30°C (86°F), normal ACLS protocols may be used.[4] All IV fluids should be warmed before administration. However, it may be necessary to extend the recommended interval for IV medications because of the patient's reduced metabolic rate.

For the unconscious hypothermic patient who is not in cardiopulmonary arrest, endotracheal or nasotracheal intubation should be performed gently. Insertion of pacemaker wires and central venous catheters has been suspected of precipitating ventricular fibrillation, but prior ventilation with 100% oxygen has been associated with decreased risk for this complication.[29,30] If intubation is indicated, rescuers should not withhold it out of fear of precipitating ventricular fibrillation.[4]

If the patient does not require immediate life support intervention, a thorough and systematic examination must be performed as quickly as possible before initiation of hypothermia therapy. Because severely hypothermic patients may have a markedly depressed mental status, they may not respond normally to painful stimuli. Victims of immersion hypothermia may have suffered trauma before entering or while in the water. Central nervous system, skeletal, and soft tissue injuries may be overlooked without a careful examination.

Attention should be paid to the patient's mental status and other central nervous system signs. Rescuers should evaluate the level of consciousness, the presence or absence of shivering, and the pupillary size and light reflex. Pupils may appear fixed and dilated in an unconscious, severely hypothermic patient, simulating the appearance of death. The diagnosis of death should not be made in a hypothermic patient, particularly in a field setting, unless resuscitation efforts fail following adequate rewarming efforts.

Vital signs should be carefully measured, with particular attention to core temperature. A low-reading thermometer (capable of recording down to 20°C [68°F]) is required. Esophageal temperature (at the level of the atria) is the most clinically useful core temperature obtainable, as this site most closely parallels cardiac temperature.[53,84] However, most rescue personnel are not equipped to measure esophageal temperature. Rectal temperature is the most easily obtained core temperature, yet rescuers often show reluctance to obtain a recording from this site in the field, and incorrect thermometer placement may cause an erroneous reading. If neither esophageal nor rectal temperature can be taken, oral or axillary temperature may be of value, but neither is an accurate reflection of core temperature in a victim of immersion hypothermia. Facial cooling affects oral temperature, and cold skin temperature affects axillary recordings. However, because the patient's true temperature will not be lower than indicated by an oral or axillary recording, rescuers can use even these superficial temperatures

in a limited way to monitor the patient's status, although this is not ideal.

Hemorrhage should be controlled in the usual manner. Antishock trousers are normally contraindicated, however, because they are likely to be ineffective in the face of the maximal vasoconstriction already present in a severely hypothermic patient.[31] Because hypothermia itself can cause hypotension without massive fluid losses, antishock trousers should be used only if the patient's hypotension is severe and secondary to hemorrhage, or if required for the temporary stabilization of major pelvic fractures.[31]

Insulation, Stabilization, and Rewarming

After recovering the patient from the water, and after managing the immediate life-threatening emergencies, the next objectives are to prevent further heat loss and attempt moderate rewarming (e.g., 1° to 2° C [2° to 3° F]/hr). Strategies for moderate rewarming in the field vary with the equipment available, the training of rescue personnel, the environmental conditions, and the length of time required for transport to a site of definitive care. Maximal insulation of the whole body from any further cooling by the environment is a first requirement. The main goal for rescue personnel is to maintain or improve cardiorespiratory stability and to minimize core temperature afterdrop, which can decrease cardiac temperature and potentiate ventricular fibrillation, as previously discussed. All sources of heat loss (evaporation, conduction, convection, respiration, and radiation) should be controlled. Rescuers should maintain the patient in a horizontal posture to prevent hypotension. Movement of the patient must be kept to a minimum; clothing should be cut away if it is wet. The patient's skin should be dried, and the patient should be protected by dry, insulating clothing and blankets, sleeping bag, or specialized rescue bag. This protection must include the head and neck. Incorporation of a windproof and waterproof layer is of great value in preventing convective and evaporative heat loss. This is particularly important for patients requiring helicopter evacuation, as downwash from the rotor blades can reach wind speeds in excess of 160 km/hr (100 mph), potentiating significant heat loss from windchill.[125]

Spontaneous Rewarming

Patients who are only mildly hypothermic (exposed to cold water for a relatively short time, fully conscious, and vigorously shivering with a core temperature greater than 32° to 33° C [90° to 91° F]) are usually capable of rewarming themselves without difficulty. Heat production from shivering can reach levels five to six times that of resting metabolic rate.[38] Shivering is thus a highly effective means of rewarming, particularly if rescuers insulate the patient's head and body and include a vapor barrier to minimize evaporative heat loss.[53] Vigorous shivering has been shown to be capable of producing rewarming rates as high as 3° to 4° C (4° to 6° F)/hr.[46,53,59] Patients who are conscious and vigorously shivering are generally not a medical emergency and usually do not require immediate transportation for definitive care. However, rescuers should be aware that prolonged periods of shivering consume the patient's endogenous energy reserves, and they should administer oral glucose-containing fluids to conscious patients who have adequate cough and gag reflexes. Attention to energy reserves in unconscious, severely hypothermic patients is also important. Warmed IV glucose solutions are useful in these patients as part of definitive resuscitative and rewarming protocols (see later).[4,31] Alcoholic beverages are contraindicated in all cases.

Inhalation Warming

Rescuers should attempt to minimize respiratory heat losses. Hypothermic patients lose up to 11 to 13 kcal/hr (46 to 54 kJ/hr) through the inhalation of relatively cold, dry air and through the exhalation of water-saturated air at near-core temperature.[62,196] The administration of heated, humidified air or oxygen at approximately 42° to 46° C (107° to 115° F) can result in a net positive gain of 17.1 kcal/hr (71.5 kJ/hr).[175] Although this is a relatively small amount of heat compared with the total kilocalories required to completely rewarm the hypothermic patient, heated and humidified ventilation is of value in insulating the airway from further evaporative heat loss. Some studies have shown, however, that inhalation rewarming reduces the metabolic heat production achieved through shivering.[51] Also, in experimental subjects whose shivering was pharmacologically inhibited, inhalation warming did not appear to provide any core rewarming advantage over spontaneous rewarming.[64] Other suggested benefits of inhalation warming include rehydration, stimulation of mucociliary activity in the respiratory tract, and direct heat transfer from the upper airways to the hypothalamus, brainstem, and other brain structures. Any resultant warming of the respiratory, cardiovascular, or thermoregulatory centers would be of benefit. Although research continues on the efficacy of airway rewarming, most hypothermia treatment protocols continue to recommend inhalation warming as an adjunct to overall rewarming strategies.[4,29,31,34,46,153,191]

Warmed Intravenous Fluids

Administration of warmed IV fluids is beneficial in reversing hypothermia-induced dehydration and hypovolemia.[31,152] Replacement of fluids before rewarming has been shown in dogs to augment cardiac output.[136] Dextrose 5% in water or normal saline, or normal saline alone, is preferable to lactated Ringer's solution, because a hypothermic liver may be unable to metabolize lactate normally.[153] The first 300 to 500 mL should be administered fairly rapidly, with the remainder of the liter administered over the next hour.[6] In no case should cold IV fluids be administered. Plastic IV bags can be easily carried inside a rescuer's clothing (preferably next to the skin) to keep the fluids warm and to supply some perfusion pressure.

Body-to-Body Rewarming

Body-to-body warming of the patient has long been advocated as an acceptable field treatment of the hypothermic patient. Sir John Franklin, in his 1823 text, *Narrative of a Journey to the Shores of the Polar Sea in the Years 1819, 20, 21,* describes this technique for the resuscitation of an expedition member recovered from ice water.[41] When wrapped together in a blanket or sleeping bag, a rescuer can donate body heat to a hypothermic patient. Experimental studies on human volunteers, however, have failed to demonstrate a rewarming advantage of this technique in shivering individuals. When subjects were cooled to an average core temperature (as measured by T_{es}) of 34.6° C (94° F), body-to-body rewarming proved no better than shivering alone.[59] In this study, the heat donated by the rescuers was only sufficient to offset the heat lost by inhibition of shivering. Several theoretical arguments mediate against the routine use of body-to-body rewarming in shivering patients in the field:

(1) the large heat loss of an immersion hypothermic victim (e.g., 300 kcal [1254 kJ] in cooling an average normothermic adult to a core temperature of 33° C [91° F]) is unlikely to be reversed by the relatively small amount of heat provided by the donor (a resting, nonshivering adult produces only about 100 kcal/hr [418 kJ/hr])[59]; (2) rescuers provide a relatively small surface area of direct contact with the hypothermic victim through which to donate heat; (3) peripheral vasoconstriction in the victim limits heat flow to the core[37]; and (4) external heat from body-to-body contact inhibits the hypothermic victim's endogenous heat production through shivering.[53] However, if the victim is severely hypothermic and nonshivering, or if shivering thermogenesis is inhibited by alcohol, medications, or age, body-to-body rewarming may be indicated.[88] Furthermore, body-to-body rewarming may provide important psychological support to the patient. For these reasons, rescuers should weigh resource implications against the advantages and disadvantages of body-to-body rewarming in devising a field rewarming strategy for any particular hypothermic patient.

Heating Pads

The application of external moderate heat sources to hypothermic patients has been a traditional method of field rewarming. Particularly when applied to the patient's neck, thorax, and groin (areas with high potential for conductive heat transfer),[82] hot water bottles, chemical or charcoal heat packs, heating pads, or other warmed objects have been used in an attempt to stabilize the patient's core temperature. Experimental evidence is lacking, however, to demonstrate the efficacy of this treatment modality compared with spontaneous shivering thermogenesis alone.[23,53,76,156] Only when combined with a source of heated, humidified ventilation has a heating pad been experimentally shown (on mildly hypothermic, shivering volunteers) to be marginally effective in shivering subjects.[133] For more severely hypothermic patients who are not shivering, these types of external heat sources may be indicated to help stabilize the patient's core temperature. Indeed, a recent study has demonstrated that charcoal heating provides a significant rewarming advantage in hypothermic subjects when shivering was pharmacologically inhibited.[88]

Rescuers should be aware of several potential hazards with this rewarming technique. Very cold skin is very sensitive to heat and is easily injured. All sources of external heat must be separated from direct contact with the patient's skin to prevent severe thermal burns.[152] Third-degree burns have resulted from the application of a lukewarm hot-water bottle directly to a hypothermic child's skin. Furthermore, heat packs that use burning charcoal as a heat source can create a carbon monoxide hazard within an unventilated enclosed space.[156]

Arteriovenous Anastomosis Rewarming

One rewarming technique relies on the arteriovenous anastomoses (AVAs) that exist in human digits, and in the superficial venous rete of the forearms and lower legs. Warming these areas opens the AVAs and increases the superficial venous return via the rete. Warmed venous blood thus reaches the core without excessive countercurrent heat loss to cold arteries (i.e., superficial veins are not in close contact with arteries). Since the pioneering work of Vanggaard and Gjerloff,[185] who proposed AVA rewarming, the Royal Danish Navy has been using this technique since 1970,[183] and recent experimental studies have confirmed its efficacy.[184] Mildly hypothermic subjects (T_{es} = 34.2° C [94° F]), whose hands, forearms, feet, and lower legs were immersed in either 42° C (107° F) or 45° C (113° F) water, had a smaller postcooling afterdrop than from shivering alone (0.4° C [0.6° F] compared with 0.6° C [0.9° F]) and a significantly faster rate of rewarming (6.6° C [12° F]/hr and 9.9° C [18° F]/hr), respectively, compared with shivering alone (3.4° C [6° F]/hr). Although this technique may be difficult to implement in some field settings, it may be practical in mildly hypothermic victims on rescue vessels or other locations where a source of warm water is available. As with heating pads and other sources of external heat, however, rescuers should be concerned about the possibility of thermal burns. In the study just described, no burns were observed in the experimental subjects, even when their extremities were immersed in 45° C (113° F) water, although some subjects complained of initial discomfort at this temperature. Rescuers opting to use AVA rewarming should perhaps start with a 42° C (108° F) water temperature and gradually raise the temperature to 44° C (111° F).[184]

A concern with AVA rewarming concerns potential cardiovascular instability. Hypotension may occur secondary to an increase in peripheral blood flow in a hypovolemic patient. If the patient is required to be in a semi-upright posture to receive AVA rewarming, orthostatic hypotension could potentially add to cardiovascular instability. No experimental data support these potential cardiovascular problems, but studies on AVA rewarming have been performed only on mildly hypothermic subjects.[184] AVA warming should not be used on severely hypothermic patients.

Forced-Air Warming

Forced-air warming (FAW) is a technique derived from a treatment modality used to prevent or reverse hypothermia in surgical patients.[101,144] Convective heat transfer is provided by warm air blown into warming covers over the patient's body. Both experimental and clinical data support the efficacy of this technique on mild-to-moderate and on severely hypothermic patients. In mildly hypothermic experimental subjects, FAW was associated with a 30% smaller afterdrop than was found with shivering alone. Furthermore, in the initial 35 minutes of rewarming, shivering patients experienced a net heat loss of 30 to 50 W, compared with a net heat gain from FAW of 163 to 237 W.[58] In moderately-to-severely hypothermic patients (mean rectal temperature, 28.5° C [83° F]) treated in an emergency room, FAW (when used in conjunction with warmed IV fluids and inhalation warming) achieved a rewarming rate of 2.4° C (4.3° F)/hr, nearly twice that of patients treated with only warmed IV fluids and inhalation warming (1.4° C [2.5° F]/hr).[149]

In another series of experiments using human subjects, an FAW device designed for field use was associated with a significantly higher core rewarming rate (5.8° C [10.4° F]/hr) than that of shivering alone (3.4° C [6.1° F]/hr), but it showed no advantage over shivering in decreasing afterdrop.[33] When shivering was inhibited with meperidine in human volunteers,[54] FAW was associated with a 50% decrease in afterdrop and a 600% increase in rewarming rate compared with spontaneous rewarming alone.[64] A different prototype FAW device, using a collapsible rigid patient cover and evaluated experimentally on nonshivering human volunteers, was associated with a smaller afterdrop and faster rewarming rate than spontaneous rewarming alone.[57]

In summary, FAW has shown significant promise as both a field and a hospital treatment modality. It is a safe, noninva-

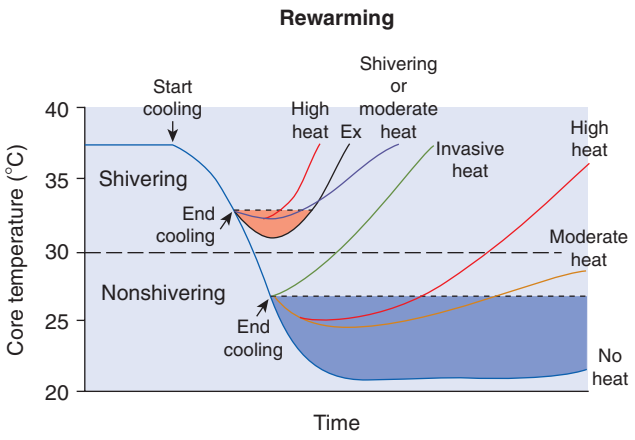


Figure 6-25. Schematic illustration of the effectiveness of various rewarming techniques under shivering and nonshivering hypothermic conditions. *Dashed line* indicates approximate core temperature at which shivering is spontaneously abolished.

sive technique that can both decrease the afterdrop and increase the core warming rate of immersion hypothermic patients. Figure 6-25 schematically illustrates core temperature responses to various rewarming methodologies under mild (shivering intact) and severe (shivering absent) hypothermic conditions.

Transportation

Stabilization, insulation, and rewarming of the hypothermic patient should be started during transport to an appropriate medical center for definitive rewarming. If possible, the receiving facility should be selected on the basis of knowledge and experience in managing hypothermic patients. In the same manner that victims of multiple trauma are most appropriately managed in trauma centers, severely hypothermic patients are best managed in hospitals equipped to handle potential complications and to provide core rewarming therapies. For example, a hospital with cardiac bypass rewarming capabilities may be a better choice for managing a severely hypothermic patient than a hospital without such qualifications, even though the former may require a longer transport time.

During transport to a site for definitive medical care, emergency medical personnel should frequently monitor the patient's core temperature and other vital signs. In addition, they should attach an electrocardiographic monitor, continue the administration of warmed IV fluids and heated, humidified air or oxygen, and continue the other rewarming efforts discussed earlier. Rescuers should maintain the hypothermic patient in a supine posture and should restrict the voluntary movements of conscious patients. At the receiving facility, rescuers should ensure that the patient is carried, and not allowed to walk, to the treatment location.

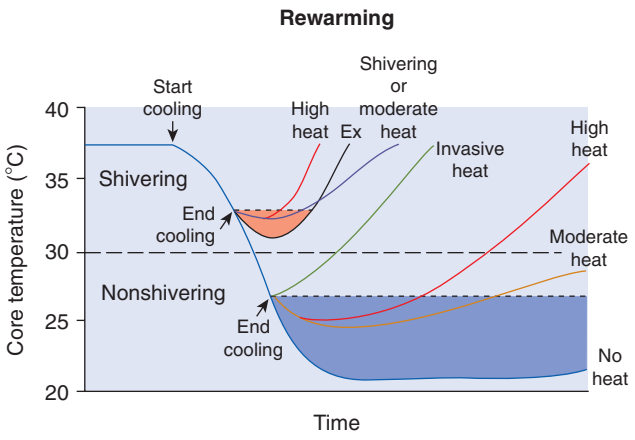
In summary, prehospital rescue and management of immersion hypothermia require an understanding of the physiologic events occurring during both cooling and recovery. With such understanding, medical personnel can better prepare for and manage this potentially difficult environmental emergency. When prehospital care is performed safely and effectively, definitive hospital care is more likely to have a successful outcome.

The references for this chapter can be found on the accompanying DVD-ROM.

7 Nonfreezing Cold-Induced Injuries

Grant S. Lipman and John W. Castellani

Nonfreezing cold injury (NFCI) is the clinical syndrome resulting from damage to tissues exposed to cold temperatures at or above freezing. NFCI does not involve tissue freezing, which distinguishes it both clinically and pathologically from frostbite.[17] Medical documentation of this syndrome has its origin in the military. Baron Dominique Jean Larrey, Napoleon's chief surgeon, used the word "congelation" to describe nonfreezing injuries together with frostbite casualties during the 1812 assault on Russia.[49] Historically, infantry have been decimated by cold and wet conditions; many medical advances in understanding the pathophysiology and clinical course of NFCI have occurred after wars.[2,16] However, continuity in research lags during the periods between major military campaigns.[54] Developments in the prevention of cold injury have flourished as new clothing and footwear are designed, but little progress has been made in the treatment of NFCI.



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As the number of people pursuing recreational activities in harsh environments increases, NFCIs are becoming more prevalent. However, because many physicians are unfamiliar with them, they may go undiagnosed during assessment of the cold-exposed victim.[54] This results in unnecessary hospital admissions and potentially harmful and expensive therapy.[21] With proper education and an awareness of the hazards innate to the cold environment, nonfreezing cold injury is preventable in most circumstances. This chapter explores the history, epidemiology, pathophysiology, and current prevention and treatments of NFCI.

EPIDEMIOLOGY

Individuals suffering cold and wet extremities for extended periods are at risk for nonfreezing cold injuries. During the 1800s, NFCI was observed more frequently when the temperature hovered around the freezing point—when the ground was muddy rather than frozen.[23,50] Standing or sitting for long periods, wearing constrictive footwear, malnutrition, fatigue, or the blunt trauma of marching on cold, wet feet all added to the severity of injury.[49] Original animal studies that modeled NFCI demonstrated that cold temperatures near the freezing point were more likely to cause injury when the extremities were wet than when they were dry.[6,50]

In combat settings, there is rarely the time, equipment, or opportunity to apply appropriate remedies to NFCI. In the 1854 Crimean War, cold injury was documented more often among "the men in the trenches [who] were so restricted in their movements. . . . Frequently this position happened to be the bottom of a trench knee-deep in mud and water or half-filled with snow."[50] In November 1944, during World War II, American forces sustained 11,000 cases of trench foot.[62] In the 1990s, both the United States Army and the Israeli Defense Forces recorded that the majority of nonfreezing cold injuries occurred during routine training exercises rather than combat operations.[12,39]

Circumstances producing large numbers of NFCI in military settings can also be found in the context of wilderness medicine. Outdoor recreation may lead to cold, dehydrated, exhausted, and wet hikers exposed to the elements for an extended period. These individuals may be unwilling or unable to take the time and effort to care for their wet boots and socks, and they may be unaware of the risks inherent to the situation. Other civilian populations at risk for NFCI include the homeless,[67] older adults,[46] and alcoholics.[54]

TRENCH FOOT (IMMERSION FOOT)

Clinical Presentation

Trench foot and immersion foot are clinically and pathologically indistinguishable, but they have different etiologies. The term *trench foot* originated during the trench warfare of World War I,[62] when soldiers wore wet boots and socks for prolonged periods. Immersion foot was first medically documented during World War II among shipwreck survivors whose feet had been continuously immersed in cold water.[10] Both injuries occur when tissue is exposed to cold and wet conditions at temperatures ranging from 0° to 15°C (32° to 59°F). Colder temperatures decrease the time required to induce NFCI.[6,49] Severe nerve damage from immersion foot has been seen after exposure periods of 14 to 22 hours.[57,58] Immersion foot injury may extend proximally and involve the knees, thighs, and buttocks, depending on the depth of immersion.[63] Clinically, NFCI is insidious in onset, with a progression from initial exposure through three distinctive phases (pre-hyperemic, hyperemic, post-hyperemic). These phases have variable time courses and may overlap.

Figure 7-1. Pre-hyperemic phase of immersion foot. These feet are still mostly numb and very cold to the touch. (British Crown Copyright/MOD.)

Pre-hyperemic Phase

During the pre-hyperemic phase, the affected limb both during and immediately after cold exposure appears blanched, yellowish white, or mottled but seldom blistered (Fig. 7-1).[58] Whayne and DeBakey state that the degree of edema during this pre-hyperemic stage is less severe if the feet are intermittently rewarmed during the course of exposure.[62] Whereas muscle cramps are common, pain is rare.[23,49] With further exposure, the cold sensation leads to complete anesthesia with loss of proprioception, resulting in numbness and gait disturbances. This sensation has been described as "walking on air" or "walking on cotton-wool."[58] Capillary refill is sluggish and pedal arterial pulses are usually absent, except through Doppler examination.[38] Intense vasoconstriction is the predominant feature of this stage.[26]

Hyperemic Phase

Within several hours after rewarming, the extremities become hot, erythematous, painful, and swollen (Figs. 7-2 through 7-5), with full bounding pulses.[61] Impairment of the microcirculation is evident through delayed capillary refill[54] (Fig. 7-6) and petechial hemorrhages.[63] Sensation returns first to proximal regions and then extends distally, rapidly progressing to a severe, burning, or throbbing pain, and reaching a maximal intensity in 24 to 36 hours.[57,58] Affected areas have marked hyperalgesia to light touch. This pain is aggravated by heat and

Figure 7-2. Hyperemic phase of nonfreezing cold injury. This person spent 18 hours in winter bailing out a boat that threatened to capsize in Prince William Sound, Alaska. (Photo courtesy James O'Malley, MD, 1995.)

Figure 7-3. Hyperemic phase of immersion foot in mild nonfreezing cold injury. Recruit of the British Royal Marines just returned from a field exercise feeling well. While showering, his feet rapidly became swollen, red, and painful. (British Crown Copyright/MOD.)

Figure 7-4. Hyperemic phase of immersion foot. The characteristic redness of the stage is absent due to the pigmented skin, but the feet are swollen and painful. (British Crown Copyright/MOD.)

Figure 7-5. Hyperemic phase in moderately severe nonfreezing cold injury. Swollen, red, and persistent pain in the feet of an infantry soldier from the Falklands War. (British Crown Copyright/MOD.)

dependent positioning, and it often worsens at night when even the pressure of sheets may be unbearable.[26] After 7 to 10 days, the nature of the pain changes to "shooting or stabbing."[57] The sensory deficits usually diminish, but paresthesias continue and anesthesia may be extensive on the toes and plantar surfaces.[63] Vibratory sensation is reduced or lost, whereas proprioception is usually retained. Anhidrosis coincides with the extent of sensory loss.[54]

Vascular injury is evident in vessel reactivity. Skin temperature gradients are absent, with digits often as warm as or warmer than the groin or axillae. When the affected limbs are lowered, blood pools, turning the extremity a deep purple-red color, whereas blanching occurs when the limb is raised. Tense edema becomes marked during this stage. Blisters containing serous or hemorrhagic fluid may form, indicating more severe injury.[54] The superficial epidermis becomes thick, indurated, and desquamated. Eschars form (Figs. 7-7 and 7-8) and eventually slough, leaving a pink dermis (Fig. 7-9). In more severe

Figure 7-6. Hyperemic phase in mild nonfreezing cold injury. There is delayed capillary refill in the dorsum of the foot. The examiner's two fingers resting on the skin for 10 seconds was sufficient to blanch the capillaries. When the pressure was removed, the blanched patches disappeared very slowly, reflecting impaired microcirculation. (British Crown Copyright/MOD.)

Figure 7-7. Severe nonfreezing cold injury. This Argentinian soldier had been unable to care for his feet for many weeks. (British Crown Copyright/MOD.)

cases, the skin may become gangrenous (Fig. 7-10); this is rare, and with appropriate care the gangrene is usually minimal.[2,61,62]

Muscles may show weakness with impaired electrical responses, slowing of plantar deep tendon reflexes, and intrinsic muscle atrophy.[57,58] In milder cases, this stage peaks at 24 hours; in more severe cases, the hyperemic phase may take 6 to 10 weeks to resolve.[54]

Figure 7-8. Severe nonfreezing cold injury in an Argentinian mine layer who wore his boots for 47 straight days during the Falklands War. (Courtesy M. P. Hamlet.)

Figure 7-9. Severe nonfreezing cold injury in a British sailor during World War II. (Courtesy M. P. Hamlet.)

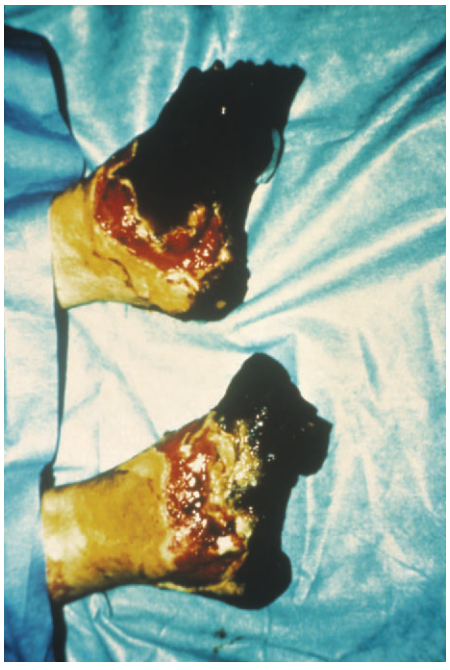

Figure 7-10. The Argentinian mine layer seen in Figure 7-8, several weeks later. (Courtesy M. P. Hamlet.)

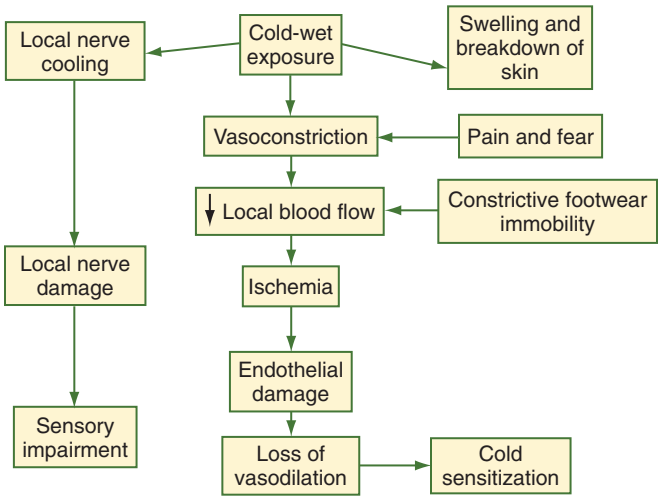

Figure 7-11. Schematic of factors and mechanisms that contribute to nonfreezing cold injuries.

Post-hyperemic Phase

The post-hyperemic phase lacks obvious physical signs. In mild cases, this phase may be absent;[58] in other cases, it may last weeks, months, or years after the hyperemic phase has subsided.[38,57] The extremities transition from a consistent warmth to noted coolness, with affected areas having an abnormal cooling response. Extremities become cold sensitive, remaining so for hours after exposure despite normal warming processes.

After 6 to 10 weeks, patients often complain of spontaneous hyperhidrosis, and sweat rashes are common in areas with heavy perspiration.[57] On a warm day, socks are quickly soaked; extremities may sweat excessively, even when cold. Hyperhidrosis predisposes to chronic paronychial infections. Sweating may be more pronounced at the margins of anhidrotic and analgesic areas.[58]

During this post-hyperemic phase, the paresthesias and extreme pains consistent with the hyperemic phase have usually resolved, replaced by dull aches and anesthesias that may persist for months to years.[62] Recurrent edema of the feet, return of paresthesias, and further blistering are common—especially after long walks. Intrinsic muscle and ligament atrophy tends to resolve,[58] but in severe cases, fibrous scarring may lead to rigidity and permanent contracture of the toes.[63] Decalcification of bones as seen in osteoporosis is frequently observed, but this tends to reverse.[56] Immobility and pain in severe cases may lead to prolonged convalescence of 6 months or more.[63]

Pathophysiology

Continual exposure to a cold and wet environment causes breakdown of the skin, directly cools nerves in the area of exposure, and causes prolonged vasoconstriction. NFCI is primarily caused by prolonged vasoconstriction, which in turn causes direct injury to the vessels (and endothelium) that supply blood to nerve, fat, and muscle cells.[54] Pain, fear, constrictive footwear, and immobility all interact to maintain vasoconstriction through a heightened sympathetic nervous system response or by mechanically limiting blood flow (Fig. 7-11). Nerve cooling has been suggested as contributing to the etiology of NFCI. Large myelinated fibers (C fibers) are most susceptible to prolonged cold exposure.[28,32,33,48] The prolonged cold injury affects blood vessels serving these large myelinated fibers with subsequent ischemia, the effect being a decrease of oxygen to the nerve, resulting in the appearance of a primary nervous system injury.

Nonfreezing cold injuries affect many different types of tissues. Examination of specimens displays a variety of lesions to the skin, muscle, nerve, and bone.[5,18] Muscles exhibit separation of the cells and damage to the muscle fibers, described as acidophilic and hyalinized (Zenker's hyaline degeneration).[5] Myoplasm within muscle loses the cross striation; the healing muscle then appears to undergo fibrous tissue replacement.

Vasoconstriction is mediated by presynaptic vesicle release of norepinephrine and neuropeptide Y from sympathetic nerve fibers, interacting postsynaptically on smooth muscle with α_2 (Frank) and Y1 (Stephens) receptors, respectively. Prolonged decrease in blood flow because of vasoconstriction causes direct injury to capillary endothelium. Studies indicate that the endothelial lining separates from the underlying cells, leaving gaps.[14] Leukocytes and platelets fill in these gaps and accumulate to further decrease capillary blood flow, leading to ischemia and eventual tissue hypoxia (Fig. 7-12). Severity of tissue injury is determined by the degree and duration of cold exposure.

Reperfusion of ischemic tissues results in free radical formation, leading to further endothelial damage and subsequent edema. Das and colleagues demonstrated that quinacrine, an antioxidant, decreased damage to cell membrane phospholipids on rewarming and reperfusion,[11] although more recent studies[55] have shown no benefit from antioxidant use. Most important,

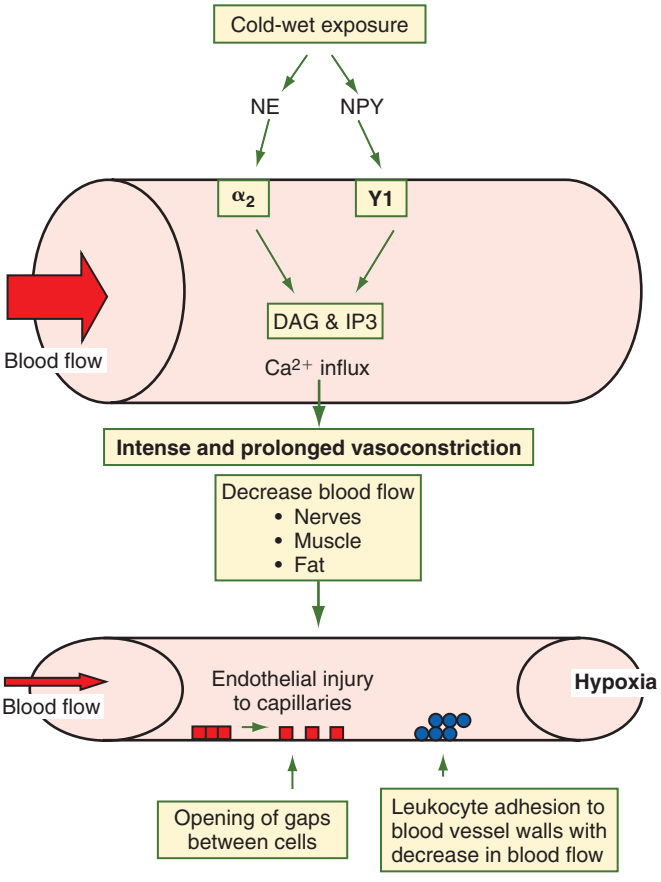

Figure 7-12. Proposed hypothesis for the etiology of nonfreezing cold injuries. α_2, norepinephrine (NE) alpha-adrenergic receptor; Y1, neuropeptide Y (NPY) receptor; DAG, diacylglycerol; IP3, inositol triphosphate; Ca^{2+}, calcium.

both these studies used models that assumed that cold-induced nerve injury, rather than capillary damage, was the primary etiologic factor for NFCI. Therefore, other experimental models need to be developed.

Cold-induced vasodilation (CIVD) is characterized by periodic oscillations of skin temperature after the initial decline during cold exposure. The absence of CIVD with prolonged cold exposure is a prominent and consistent finding in NFCI. CIVD is initiated by direct cooling of peripheral tissues and results from transient increases in local blood flow.[35] This increased blood flow supports higher tissue temperatures, which is theorized to be protective against cold injury.[65] Although this was originally thought to be only a local effect of cooling, new evidence suggests that a central nervous system mechanism also mediates CIVD. When core temperatures are low, the CIVD response is blunted,[42] so hypothermic individuals are at increased risk for NFCI. CIVD may remain blunted for weeks or months after the injury.

Treatment and Prevention

Treatment is limited to symptomatic relief and reversal of ischemia to minimize progression of tissue damage. Rewarming injured tissues increases the metabolic demand of damaged cutaneous cells to such an extent that the injured subcutaneous blood vessels cannot supply enough oxygen.[64] Tissue hypoxia and endothelial cell injury, coupled with reflex vasodilation, lead to fluid transudation, increasing edema and skin necrosis and worsening the pain.[61,63]

The treatment required for the general effects of cold is different from that needed for localized NFCI. Core temperature must be raised while the extremities are kept cool.[61–63] Injured feet should be elevated and exposed to steady cool air from a fan. Extremity cooling lowers the metabolic requirements of the tissue so that the oxygen demand can be met. Continuous cooling brings rapid improvement in pain, edema, and vesiculation.[61,63] Local cooling should be continued until pain is relieved, circulation has recovered, and hyperemia subsides.[63] The affected extremities should never be rubbed, as this may compound the injury.[1]

Recovery during the post-hyperemic phase may be hastened with physiotherapy and exercise to rehabilitate atrophied intrinsic muscles.[62,63] Lumbar sympathectomy has been theorized to reduce disabling contracture by decreasing vascular tone, increasing circulation, and hastening collagen and fibrous tissue absorption. In severe cases of NFCI involving atrophic rigid feet, small case studies have shown symptomatic improvement after sympathectomy,[63] but other authors feel that there is little therapeutic advantage to the procedure.[61]

The simplest way to prevent NFCI is to avoid prolonged exposure to cold and wet environments. However, sometimes this is quite difficult to implement. In military conflicts, completing the assigned mission is the most important goal, and it may require performing in a cold and wet environment for sustained periods in a cramped, immobile position. During mountain rescues, individuals may be so focused on helping to save others that they do not take adequate care of their own well-being.

Prevention can be achieved by encouraging people to remain active and increase blood flow to the feet, rotating personnel out of inclement environments on a regular basis, keeping feet dry by continually changing socks, maintaining body core warmth by limiting sweat accumulation into clothing and dressing in layers, and educating personnel about the early signs and symptoms of nonfreezing cold injuries. Changing socks two to three times throughout the day is mandatory in cold and wet environments. The military suggests that optimal care entails air-drying feet for at least 8 hours out of every 24.[8,67] Vapor-barrier boots do not allow sweat from the foot to evaporate, and in some situations this increases maceration.[3] These boots should be taken off each day, wiped out, and allowed to dry. Footwear should not constrict blood flow, so sizing is important, as is educating the user not to tie shoelaces too tight.

Prophylactic treatment with silicone preparations has proved effective in clinical studies.[20] This protective effect is theorized to result from prevention of hyperhydration of the stratum corneum.[8,13] However, the stickiness of these preparations, their tendency to increase adherence of sand and grit to the foot, and the bulkiness of the product made them only marginally acceptable to infantrymen in combat situations.[20] In small clinical trials, silicone ointment applied only to the sole of the foot instead of to the entire foot (thus reducing the surface area exposed to dirt retention, the amount of material transported by the soldier, and the dollar cost) was sufficient to prevent NFCI.[13]

PERNIO (CHILBLAINS)

Pernio (perniones) or chilblains are localized, inflammatory, bluish red lesions caused by an abnormal reaction to a cold, damp environment. This mild form of cold injury is prevalent in the temperate climates of northwestern Europe,[36] and it is found worldwide throughout temperate and northern zones.[21,37,43] Pernio is less common in very cold climates where well-heated houses and adequate warm clothing are common.[44]

Acute pernio has a seasonal incidence, with reversible symptoms more common in cold weather. The acute form is seen primarily in schoolchildren and young adults under the age of 20, with the highest incidence in adolescent females.[36] It can occur in mildly cold settings such as logging, kayaking, snowmaking,[26] winter horseback riding, and hiking.[45] Pernio can be caused by brief (30 minutes) cold exposure, often appearing several hours after exposure, with the skin lesions fully developed within 12 to 24 hours.[43] Characteristic locations for these lesions are the feet, hands, legs, and thighs. Single or multiple, erythematous, purplish, edematous lesions form, with vesicles in severe cases. Symptoms include intense pruritus, burning, or pain, often worsened by subsequent warmth. The lesions of acute pernio are self-limiting and usually resolve within a few days to 3 weeks,[44] occasionally leaving residual hyperpigmentation.[9] Although the healing process appears to occur as the plaques resolve, pain often persists. Subsequent mild cold exposure may trigger paresthesias, edema, and skin scaling.[26]

Chronic perniosis usually progresses over several winters after repeated episodes of acute pernio, rarely progressing from the initial injury to chronic irreversible skin changes within a single season.[36] Repeated episodic seasonal lesions may become edematous, with permanent discoloration and subcutaneous nodule formation. The nodules are firm and painful, ultimately rupturing, which provides pain relief and leaves a shallow ulcer with pigmented atrophic skin. These ulcers may grow larger and coalesce, remaining open, which leads to permanently swollen extremities, scaly pigmented skin, and unremitting pain aggravated by light pressure.

Pernio is thought to be caused by prolonged cold-induced vasoconstriction with subsequent hypoxemia and vessel wall inflammation.[21,29] Subcutaneous arterial vasoconstriction is documented by both pathologic[36] and arteriographic[51] studies. Histologic examinations show a lymphocytic vasculitis and papillary dermal edema with pervasive inflammatory changes.[21,29,36] The differential diagnosis includes lupus erythematosus, Raynaud's disease, polycythemia vera, atheromatous embolization, erythema nodosum, and livedo vasculitis with ulcerations.

Treatment of pernio is accomplished by drying and gently massaging the affected skin. Active warming above 30°C (86°F) significantly worsens the pain and should be avoided.[26] Although therapeutic regimens in the literature include nicotinic acid,[22] ultraviolet irradiation,[27] thymoxamine,[30] intravenous calcium combined with intramuscular vitamin K,[15] corticosteroids,[19] and sympathectomy in severe cases,[36] few have proven to be either effective or universally accepted. Recently, nifedipine (20 mg, three times daily) has shown to be effective for the treatment of severe perniosis. Patients treated had a significantly reduced time for the clearance of lesions, decreased pain and irritation of existing lesions, and less development of new pernio.[21,47]

Preventing pernio is relatively simple. Recommended prophylactic measures include minimizing cold exposure with suitable clothing when outdoors and maintaining adequate warm temperatures indoors.

CRYOGLOBULINEMIA

Cryoglobulins are a group of serum proteins that reversibly precipitate in the cold.[34] The vast majority of cryoglobulins are either intact monoclonal immunoglobulins or, more frequently, mixed immunoglobulin complexes in which one component (usually IgM) is active against IgG.[24] These cryoimmunoglobulins were first reported in a patient with multiple myeloma,[66] and they have subsequently been recognized to occur in a diverse group of hematologic malignancies, acute and chronic infections, and collagen vascular diseases.[52,59]

In general, greater serum concentrations of proteins precipitate at warmer temperatures. Of clinical relevance is the composition of the cryoprecipitate. For example, IgM is intrinsically more viscous than IgG, so patients with monoclonal cryo-IgM have an amplified hyperviscosity. Because extremity temperatures can reach 30°C (86°F), in vivo cryoprecipitation may directly contribute to impaired capillary blood flow.[24,26]

A large clinical trial showed that two thirds of patients diagnosed with cryoglobulinemia initially presented with skin lesions or Raynaud disease–like vasomotor attacks. Mucosal bleeding, visual disturbances, and abdominal pain were less common. Cold sensitivity occurred in fewer than half of these patients.[7] Symptoms associated with cryoglobulins include typical Raynaud's phenomenon, dependent purpura, cutaneous vasculitis with ulceration, retinal hemorrhages, coagulopathies, glomerulonephritis, renal failure, and cerebral thrombosis. Inexact correlations exist between cryoglobulin concentrations and the presence of symptoms.[24]

The primary treatment of cryoglobulinemia should be directed at minimizing cold exposure. Also, avoiding prolonged standing can minimize dependent purpura. Initiating active treatment is a difficult decision that should be based on the severity of symptoms and the presence of underlying disease. Small sample trials have shown that cyproheptadine (4 mg, three to four times daily) for 1 to 2 weeks is effective in relieving the associated symptoms of cold urticaria.[60] Adrenocorticotropic hormone (ACTH) or corticosteroids have provided limited therapeutic success, possibly due to their anti-inflammatory and antihistamine activity rather than to the serum reduction of cryoprecipitate.[59] With severe manifestations of the disease, such as renal failure, neurologic impairment, disabling paresthesias, and myalgias, emergency plasmapheresis may be helpful in reducing cryoglobulin concentration below a critical level.[7,24]

COLD URTICARIA

Cold urticaria is a fairly common physical urticaria, characterized by development of either localized or general wheals and itching after skin exposure to solid or liquid cold.[40] Primary cold urticaria can occur at any age, has an equal incidence in the sexes, and tends to be self-limiting, although it may become chronic.[26,53,60]

Local symptoms include erythema, itching, wheals, or edema in exposed skin. The wheals last approximately 30 minutes.

Figure 7-13. Cold urticaria. The hive occurred within minutes of holding an ice cube against the skin. (From Habif TP: Clinical Dermatology, 4th ed. Philadelphia, Mosby, 2004, with permission.)

Urticaria may occur in the oropharynx after drinking cold fluids.[25] Systemic symptoms include fatigue, headache, dyspnea, tachycardia, and, rarely, hypotension. Swimming in cold water is the most common precipitant of severe reactions. This may lead to massive transudation of fluid into the skin, resulting in hypotension, syncope, shock, and even death.[4]

Secondary urticaria occurs in 5% of patients with cold urticaria.[25] In these cases, the wheals are more persistent, and they may be associated with purpura and a demonstrable vasculitis on skin biopsy. This disorder is associated with underlying diseases such as cryoglobulinemia, cold agglutinins, paroxysmal hemoglobinuria, or connective tissue disease. In addition, there is a rare autosomal dominant familial form with onset in infancy, associated with arthralgias and leukocytosis.[60]

The cause of cold urticaria is unknown. It has been reported to be mediated by IgE and IgM, involving the release of histamine and other inflammatory factors.[31,40,53,60] In the majority of patients, the diagnosis of cold urticaria is made through the ice cube test (Fig. 7-13)[40]: a hive is induced by holding an ice cube to the skin for 3 to 5 minutes. If the results are equivocal, a cold-water immersion test establishes the diagnosis by submerging a forearm for 5 to 15 minutes in water at 0° to 8°C (32° to 46°F) to produce a hive.

Cold avoidance is not always effective in preventing cold urticaria, as it is the rate of cooling rather than the absolute temperature that appears to be the principal stimulus.[43,51] Small clinical trials have shown the efficacy of cyproheptadine, hydroxyzine, doxepin, and ketotifen for shortening the duration as well as for suppressing the clinical symptoms of cold urticaria.[41,53,60]

The references for this chapter can be found on the accompanying DVD-ROM.

8

Frostbite

Robert L. McCauley, Garry W. Killyon, David J. Smith, Jr., Martin C. Robson, and John P. Heggers

Cold-induced injuries are almost exclusively a result of humans' inability to properly protect themselves from the environment. Cold injuries may be either systemic or localized, depending on the temperature and length of exposure. Systemic injury is hypothermia. Although much confusion exists in the literature regarding the myriad of clinical terms used to describe localized injury, the correct term is *frostbite*.

HISTORICAL ASPECTS

Numerous historical accounts discuss armies exposed to cold and sustaining large numbers of cold-induced casualties. Although Hippocrates described some of the symptoms and sequelae of frostbite, cold injuries were probably not prevalent in ancient Greece. In 218 BC, Hannibal lost nearly half of his army of 46,000 to cold injuries in only 15 days when they crossed the Pyrenean Alps. In 210 BC, Xenophon described cold injuries suffered by Spartan soldiers during a retreat across the Carduchion mountains after leaving Alexander's armies. In 1778, Dr. James Thatcher wrote that 10% of George Washington's army had been left to perish in the winter cold during his campaign against the British soldiers.[69]

Despite these accounts, it was not until 1805 that the first official report was published on the effect of cold injury.[90] The first authoritative account of mass casualties was the descrip-

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tion by Baron Larrey, surgeon-in-chief of Napoleon's army during the invasion of Russia in the winter of 1812–1813.[35] Larrey wrote, "When some external part of the body is caught by cold, instead of submitting it to heat, which provides gangrene, it is necessary to rub the affected part with substances containing very little calorick—for it is well known that the effect of calorick on an organized part that is almost deprived of life is marked by an acceleration of fermentation and putrefaction."[35]

Larrey introduced the concept of friction massage with ice or snow, the avoidance of heat in thawing, and the idea that cold injuries were similar to burn injuries. These concepts are better understood against the background as viewed by Larrey.[35] Soldiers with cold injuries rapidly rewarmed their extremities over roaring fires (150° to 170°F) after long marches, only to renew the trek and refreeze their extremities the next day. Larrey recognized that warming was good, but he cautioned against the use of excessive heat and ultimately recognized the freeze–thaw–freeze cycle. Napoleon left France with 250,000 men and returned 6 months later with only 350 effective soldiers. The remainder were casualties to cold or starvation.[35]

During the Crimean War (1854–1856), 300,000 French troops sustained 5000 cases of frostbite and 1000 deaths. During World War I (1917–1918), World War II (1941–1945), and the Korean conflict (1950–1953), at least 1 million cases of frostbite occurred.[60,61,77,80] In November and December of 1942, the Germans performed 15,000 amputations for cold injuries.[77,78] High-altitude frostbite, first described in 1943, was recognized from the treatment of aviators in World War II.[80] The most prevalent form of this injury was seen in B-17 and B-24 crews flying between 25,000 and 35,000 feet in temperatures of –32° to –43°C (–25° to –45°F). When attacked, the only way the waist gunners of the aircraft could operate their machine guns was to open the large waist pots through which the guns were fired. The gunners often took off their bulky mittens and jackets to improve the dexterity they felt was crucial to saving their lives.[80]

Until the 1950s, treatment of cold injuries basically followed Larrey's guidelines. In 1956, experimental laboratory work by Merryman encouraged Hamill, the Public Health Service district medical officer in Tanana, Alaska, to try rapid rewarming at 100°F (37.7°C) on a patient with frostbite and hypothermia.[9,50] This was the genesis and has become the cornerstone of the method currently used in Alaska and popularized by Mills.[50]

TEMPERATURE HOMEOSTASIS IN HUMANS

No comprehensive statistical data are available on the incidence of frostbite. The Royal College of Physicians in London estimated that 10% of older adult British home dwellers were hypothermic, but no comparable data were available for frostbite.[18] Frostbite is much more prevalent during military campaigns and is a known hazard for mountain climbers and explorers. In the nonadventurer civilian population, Mills collected 500 cases in Alaska by 1963; Cook County Hospital in Chicago recorded 843 cases from 1962 to 1972; Maria Hospital in Helsinki, Finland, reported 110 cases in 1968; and Detroit Receiving Hospital reported on 154 patients treated from 1982 to 1985.[4,23,33,51]

Physiologically, humans are tropical beings, better suited to losing heat than retaining it. When naked and at rest, a person's neutral environmental temperature is 28°C (82°F); with an environmental drop of only 8°C (14.5°F), the metabolic rate must double to avoid a lowering of body temperature. In comparison, the Arctic fox can maintain a steady thermal state down to an external temperature of –40°C (–40°F), and by doubling its metabolic rate could cope with an outside temperature of –120°C (–184°F).

Putting on clothes in response to cold is not a reflex but requires a conscious decision. When the ability to decide or to act is impaired, there is a risk of cold-induced injury.[12] Alcohol has been implicated in many cases of frostbite.[28,29] Alcohol- or drug-intoxicated persons whose consciousness, judgment, or self-protective instincts are depressed often expose themselves to dangerous environmental hazards. Knize and colleagues[29] noted that alcohol intake or mental instability led directly to cold injury in 50% of their patients. Once the injury had occurred, alcohol intake probably did not significantly alter the course of events. Barillo and associates[1] experimentally demonstrated increased mortality and a detrimental effect of ethanol on tissue perfusion associated with severe murine frostbite. Alcohol consumption promotes peripheral vascular dilation and increases heat loss. This may make an exposed part more susceptible to frostbite.

The type and duration of cold contact are the two most important factors in determining the extent of frostbite injury.[80,82,83] Touching cold wood or fabric is not nearly as dangerous as direct contact with metal, particularly by wet or even damp hands. Air alone is a poor thermal conductor, and cold air alone is not nearly as dangerous a freezing factor as a combination of wind and cold.[89] Wind velocity in combination with temperature establishes the windchill index. For example, an ambient temperature of –6.7°C (20°F) with a 45-mph wind has the same cooling effect as a temperature of –40°C (–40°F) with a 2-mph breeze.[80,82,83] Thus, it is important to think in terms of heat loss, not cold gain. Frostbite occurs when the body is unable to conserve heat or protect against heat loss.

Because many serious cases of frostbite originate at high altitude, it has been assumed that physiologic changes occurring with increased altitude make persons more prone to frostbite. However, present data suggest that reduction in atmospheric pressure has little or no effect on susceptibility to frostbite. Increased red blood cell concentration does not increase blood viscosity, impede capillary circulation, or have any other apparent bearing on cold injury.[80,82,83] On the other hand, deep, loose snow, which traditionally has been thought to insulate from the cold, may actually contribute to frostbite. Temperature measured beneath deep snow is frequently much lower than that on the surface. Washburn[83] recounts one expedition to Mt. McKinley, Alaska, when members of his party found it extremely difficult to keep their feet warm, despite a clear, sunny, –16°C (3°F) day with little wind. One member inadvertently dropped a thermometer in the snow and noted that it registered –25.6°C (–14°F). Feet must be dressed for the temperature at their level, not for surface temperature protection.[83]

Development of frostbite does not depend simply on ambient temperature and duration of exposure. Along with windchill, humidity and wetness also predispose to frostbite. Skin wetting adds an increment of heat transfer through evaporation and causes wet skin to cool faster than dry skin.[57] More important, water in the stratum corneum can terminate supercooling by trig-

gering water crystallization not only in this layer but also in the underlying tissue. Skin wetness is therefore conducive to frostbite because it allows crystallization to terminate supercooling after approximately half of the exposure time required by dry skin. This substantiates the clinical observation, "It has been found that supercooling displays itself in greater degree in skin that remains unwashed. Washing the skin encourages freezing, while rubbing the skin with spirit and anointing it with oil discourages it. The capacity to supercool greatly would seem to be connected with relative dryness of the horny layers of the skin. It is well known that Arctic explorers leave their skins unwashed."[37]

The degree of inadequacy of protective clothing varies with conditions and may contribute to insufficient conservation of body heat. Tight-fitting clothing may produce constriction, which hinders blood circulation and lessens the benefit of heat-retaining air insulation. Wet clothing transmits heat from the body into the environment because water is a thermal conductor superior to air by a factor of about 25.[28] Clothing that transmits moisture away from the body may be protective if an outer wind-resistant layer decreases heat loss. However, this wind-resistant layer must retain the same transmission capabilities; otherwise, clothing will still become moist. Clothes that decrease the amount of surface area may decrease frostbite risk. Mittens are more protective than are gloves, because gloves have a greater surface area and prevent air from circulating between fingers. Eighty percent of total body heat loss can occur through exposed head and neck areas. Poorly fitted boots notoriously generate frostbite injuries.

During World War II and the Korean conflict, clinical studies indicated that cold injuries occurred with higher frequency among soldiers in retreat.[60,61,87] Fatigue and apathy also increase the incidence of cold injury. When warfare is proceeding toward defeat, or in conditions of starvation, soldiers often become indifferent to personal hygiene and clothing, and the frequency of frostbite increases.[27] Overexertion increases heat loss. A large amount of body heat can be expanded by panting, and perspiration further compounds the problem of chilling.[80,82,83] Both panting and sweating consume energy, which compounds the fatigue factor.

Impaired local circulation is a primary contributor to frostbite. Cigarette smoking causes vasoconstriction, decreased cutaneous blood flow, and tissue loss in random skin flaps.[36] Reus and colleagues[66] documented that smoking induced arteriolar vasoconstriction and decreased blood flow in a nude subject. Although red blood cell velocity increased, the net effect was a decrease in blood flow in the cutaneous microcirculation during and immediately after smoking. Curiously, habitual heavy smokers do not appear to be more prone to frostbite. Other drugs known to have vasoactive properties may also predispose to or compound a frostbite injury. Disease states that alter tissue perfusion, such as atherosclerosis or arteritis, predispose to frostbite.

On the basis of clinical observations, an individual who has experienced prior cold injury is placed in a high risk category during subsequent cold exposure.[54,87] For undefined reasons, cold injury sensitizes an individual, so that subsequent cold exposure, even of a lesser degree, produces more rapid tissue damage.[28] Military studies emphasize that long periods of immobility contribute to the extent of cold injury.[28,29] Motion produces body heat and improves circulation, especially in endangered limbs.

Civilian clinical studies are inadequate for statistical evaluation of factors such as race and previous climatic environmental exposure.[28,45] Military studies suggest that dark-skinned soldiers are more susceptible than whites to cold injury under the same combat conditions.[89] Individuals from warmer climatic regions in the United States tend to be more susceptible.[87] There are no data to suggest influence of age or sex in the incidence of frostbite.

THE SKIN

The skin is an individual's interface with the environment, serving as an organ of protection. It controls the invasion of microorganisms, maintains fluid balance, regulates temperature, protects against injury from radiation and electricity, and provides immunologic surveillance. Each function is specifically related to a cell or an area within the skin.[25]

The skin is a highly specialized bilaminate structure resting on a subcutaneous layer of padding. Generally, skin thickness is about 1 to 2 mm, but it varies between 0.5 and 6 mm. The highly cellular epidermis and underlying dermis are in contact via multiple interpapillary ridges and grooves. The outermost layer of the epidermis, the stratum corneum, functions for protection. The innermost layer, the stratum germinativum or basal layer, contains the only proliferating cells in the epidermis. Between these two layers are keratinocytes in various stages of differentiation. Other cells derived from the neural crest (melanocytes) and mesenchyme (Langerhans' cells), as well as cells of unknown etiology (Merkel's cells), migrate into the epidermis and become organized in specific association with certain keratinocytes.

The dermal–epidermal junctions undulate in most areas of the body, increasing surface contact between the two layers to provide resistance to shearing. The dermal–epidermal junction is a complex of structures referred to as the basement membrane zone, which functions as a filter to inhibit or prevent passage of molecules greater than 40 kDa.

The dermis, or deeper layer of skin, is 20 to 30 times thicker than the epidermis. It contains nervous, vascular, lymphatic, and supporting structures for the epidermis and harbors the epidermal appendages. Regions of the dermis differ in structural organization and biochemistry, and each responds uniquely to systemic disease, genetic disease, and environmental assault. The papillary dermis and the reticular dermis are the two main dermal zones. The dermis contains fibrous and nonfibrous matrix molecules. Fibrous proteins, primarily collagen, impart bulk, density, and tensile properties to skin, but they also allow compliance and elasticity. Nonfibrous matrix molecules, glycosaminoglycans and glycoproteins, form ground substance, which influences the osmotic properties of skin, permits cellular migration in a more fluid milieu, and serves as an integrative, continuous medium for all of the other structural elements.[24]

The papillary dermis is only slightly thicker than the overlying epidermis. Generally, it is separated from the underlying reticular dermis by a horizontal plexus of vessels. This plexus provides the overlying papillary dermis with a rich blood supply. The papillary dermis is more commonly altered in environmentally induced skin lesions (such as actinic damage) than in systemic disease or inherited diseases of connective tissue metabolism.

Most of the dermis is reticular dermis. Epidermal appendages either terminate in the lower levels of the reticular dermis or

penetrate even deeper into the subcutaneous tissue. Blood vessels pierce the dermis, yielding branches to hair follicles and sweat glands.

Thermoregulation is one body function in which the stratum corneum is not important. The rate at which heat is lost by radiation is a function of the temperature of the cutaneous surface, which in turn is a function primarily of the rate of blood flow through the skin. Heat is poorly conducted from warmer internal tissue to the cutaneous surface because adipose tissue is a good heat insulator.

Thus, cutaneous circulation is the key to the genesis of frostbite. Because of its role in thermoregulation, the normal blood flow of skin far exceeds its nutritional obligation. The skin holds a complex system of capillary loops that empty into a large subcapillary venous plexus containing the majority of the cutaneous blood volume. Under normothermic conditions, 80% of an extremity's blood volume is in the skin and muscle veins. Skin blood volume depends in part on the tone in the resistance and capacitance vessels, and tone in turn depends largely on ambient and body temperature. Under basal conditions, a 70-kg human has a total cutaneous blood flow of 200 to 500 mL/min. With external heating to maintain skin temperature at 41° C (105.8° F), this may increase to 7000 to 8000 mL/min, while cooling the skin to 14° C (57° F) may diminish it to 20 to 50 mL/min.

Blood flow through apical structures such as the nose, ears, hands, and feet varies most markedly because of richly innervated arteriovenous connections. Blood flow to hand skin can be increased from a basal rate of 3 to 10 mL/min/100 g of tissue to a maximum of 180 mL/min/100 g of tissue. This cutaneous vascular tone is controlled by both direct local and reflex effects. Indirect heating (warming a distant part of the body) results in reflex-mediated cutaneous vasodilation, whereas direct warming results in vasodilation dominated by local effects. When both types (central and peripheral) of heating or cooling are present, their effects are additive.

Cutaneous vessels are controlled by sympathetic adrenergic vasoconstrictor fibers. Vascular smooth muscles have both α- and β-receptors, although the significance of the β-receptors is yet to be defined. Vasodilation in the hands and feet is passive, so maximal reflex vasodilation occurs after sympathectomy. After sympathectomy, residual local control of vascular tone persists, so that direct heat or cold continues to alter blood flow.

When the hand or foot is cooled to 15° C (59° F), maximal vasoconstriction and minimal blood flow occur. If cooling continues to 10° C (50° F), vasoconstriction is interrupted by periods of vasodilation and an associated increase in blood and heat flow. This cold-induced vasodilation (CIVD), or "hunting response," recurs in 5- to 10-minute cycles to provide some protection from the cold. There is considerable individual variation in the amount of CIVD, which might explain some of the variation in susceptibility to frostbite. Prolonged repeated exposure to cold increases CIVD and offers some degree of acclimatization. Eskimos, Lapps, and Nordic fishermen have a very strong CIVD response and very short intervals between dilations, which may contribute to maintenance of hand function in the cold environment.[21]

There is little evidence that humans have any significant physiologic adaptation to cold. They remain homeothermic, warm weather, tropical animals who neither tolerate nor adapt well to the cold. They must find a well-defined microclimate to keep skin temperature close to 32.8° C (91° F). Normal skin maturation and tissue function rely on the maintenance of permeability and integrity of all tissue membranes. A steady rate relationship of prostaglandins (PGs), particularly PGE_2 (vasodilator) and $PGF_{2\alpha}$ (vasoconstrictor), is crucial for normal skin function. An imbalance may disrupt cell membrane equilibrium. This relationship is controlled through PGE_2-9-ketoreductase and nicotinamide adenine dinucleotide phosphate (NADPH). Low concentrations of PGE_2-9-ketoreductase found in normal skin emphasize an active biologic presence.

FROSTBITE INJURY

The frostbite injury has classically been divided into four pathologic phases.[31]

1. Prefreeze phase. This is secondary to chilling and prior to ice crystal formation. Changes are caused by vasospasticity and transendothelial plasma leakage. Tissue temperature ranges from 3° to 10° C (37° to 50° F). Cutaneous sensation is generally abolished at 10° C (50° F).
2. Freeze–thaw phase. This is caused by actual ice crystal formation. Tissue temperature drops below the freezing point as the ambient environmental temperature dips below –15° to –6° C (5° to 21° F). Because of the underlying radiation of heat energy, skin must be supercooled to –4° C (24.8° F) to freeze. With no circulation, skin temperature may drop at a rate in excess of 0.5° C (1° F) per minute. After it is completely frozen, the tissue rapidly exhibits poikilothermy. The susceptibility of tissue to freezing varies, with endothelium, bone marrow, and nerve tissue more sensitive than muscle, bone, and cartilage.
3. Vascular stasis phase. This involves changes in blood vessels, including spasticity and dilation, and includes plasma leakage, stasis coagulation, and shunting.
4. Late ischemic phase. This is a result of thrombosis and arteriovenous shunting, ischemia, gangrene, and autonomic dysfunction.

Overlap occurs among these phases.[32] The changes during each phase vary with rapidity of freezing and duration and extent of injury. It is therefore conceptually clearer to divide pathologic changes occurring in frostbite into those resulting from direct cellular injury and those from indirect cellular effects. Changes caused by direct injury include the following[91]:

1. Extracellular ice formation
2. Intracellular ice formation
3. Cell dehydration and shrinkage
4. Abnormal intracellular electrolyte concentrations
5. Thermal shock
6. Denaturation of lipid–protein complexes

Cells subjected to a slow rate of cooling (hours) develop ice crystals extracellularly in the cellular interspaces. Rapid cooling (seconds to minutes) produces intracellular ice crystals, which are more lethal to the cell and less favorable for cell survival. In a clinical cold injury, the slower rate of freezing does not produce intracellular crystals[47]; however, the extracellular ice formed is not innocuous. It draws water across the cell membrane, contributing to intracellular dehydration. The theory of cellular dehydration was originally proposed by Moran[58] in 1929 and subsequently supported by Merryman's study of "ice-crystal nucleation."[46–48] Cellular dehydration produces modifi-

cation of protein structure by high electrolyte concentration, alteration of membrane lipids, alteration of cellular pH, and imbalance of chemical activity.[42,43,48] This phenomenon subsequently permits a marked and toxic increase of electrolytes within the cell, leading to partial shrinkage and collapse of its vital cell membrane. These events are incompatible with cell survival.

Not all the water within a cell is freezable. A small amount of unfrozen water, "bound water," constitutes up to 10% of the total water content and is held tightly in the protein complex within the cell. No matter how rapid or marked the cold injury, this bound water remains liquid. At temperatures below –20° C (–4° F), approximately 90% of available water is frozen.[89] Although the theory of ice crystal disruption of cell structure is attractive, it has yet to be conclusively proven.

Thermal shock defines the phenomenon of sudden and profound temperature change in a biologic system.[20] Precipitous chilling has been theorized to be incompatible with life, but the severity of this phenomenon is debatable. Another poorly understood concept is the manner in which subzero temperatures produce denaturation of lipid–protein complexes. One theory hypothesizes detachment of lipids and lipid protein from cell membranes as a consequence of the solvent action of a toxic electrolyte concentration within a cell.[38,39] There is no direct evidence supporting an alteration of enzyme activity during freezing, but DNA synthesis is inhibited.[26] On the other hand, there is indirect evidence of ox liver catalase inactivation caused by denaturation and structural alteration of lactic acid dehydrogenase after freezing and thawing.[40,76]

Indirect cellular damage secondary to progressive microvascular insults is more severe than the direct cellular effect. This is supported by the observation that skin tissue subjected to a standard freeze–thaw injury, which consistently produced necrosis in vivo, survived as a full-thickness skin graft when transplanted to an uninjured recipient site.[84] Conversely, uninjured full-thickness skin did not survive when transferred to a recipient area pretreated with the same freezing injury. Thus, direct skin injury is reversible. The progressive nature of injury is probably secondary to microvascular changes.

Approximately 60% of skin capillary circulation ceases in the temperature range of 3° to 11° C, whereas 35% and 40% of blood flow ceases in arterioles and venules, respectively.[67] Capillary patency is initially restored in thawed tissue, but blood flow declines 3 to 5 minutes later. Three nearly simultaneous phenomena occur after thawing: momentary and initial vasoconstriction of arterioles and venules, resumption of capillary circulation and blood flow, and showers of emboli coursing through microvessels.[91] Ultimately, there is progressive tissue loss caused by progressive thrombosis and hypoxia. This is similar to the tissue loss seen in the distal dying random flap and the no-reflow phenomenon. For both of these, in addition to the effect of arachidonic acid metabolites, oxygen free radicals have been shown to be detrimental and to contribute to tissue loss. This may be the case in the frostbite injury.[8] Emerging concepts of reperfusion injury will no doubt be incorporated into our understanding of frostbite injury as the scientific understanding evolves.

Considerable evidence points to the primary alteration of the cold injury being injury to vascular endothelium.[91] Seventy-two hours after a freeze–thaw injury, there is loss of vascular endothelium in the capillary walls, accompanied by significant fibrin deposition. The endothelium may be totally destroyed, and fibrin may saturate the arteriole walls.[91] Ultrastructural derangement of endothelial cells after the thaw period has been observed by electron microscopy in capillaries of the hamster cheek pouch following subzero temperatures.[64] The endothelial injury was confirmed by demonstrating fluid extravasation from vessels almost immediately after thawing.[91] As in other forms of trauma, vascular endothelial cells swell and protrude inward into the lumen until they lyse.

Venules appear more sensitive to cold injury than other vascular structures, partly because of lower flow rates. Arterioles, with a rate of flow almost twice that of venules, are less damaged by freezing and develop stasis later than venules. Capillaries manifest the fewest direct effects of cold injury, but their flow is quickly arrested as a result of their position between arterioles and venules. Generalized stasis and cessation of flow are noted at the point of injury within 20 minutes after freeze and thaw. "White thrombi" (blood cells and fibrin) follow platelet thrombi as blood flow progressively slows. Sludging and stasis result in ultimate thrombosis. Microangiography after cold injury shows that although spasm of the arterioles and venules exists, it is not marked enough to completely account for the decreased flow of progressive microvascular collapse.[2] It has been postulated that defects seen in angiograms are caused by local factors, possibly thrombi. It has also been observed that vascular thrombosis after cold injury advances from the capillary level to that of the large vessels and ultimately results in ischemic death of progressively larger areas.[30,31] Viable dermal cells may be observed histologically in cold-injured tissues for up to 8 days or until occlusion of local vessels occurs. This emphasizes that a major role is played by vascular insufficiency, and that direct injury to cellular structures and mechanisms may be reversible.

Because Cohnheim had shown changes in cold injury to be similar to changes seen in other inflammatory states, Robson and Heggers[68] postulated that the progressive ischemia seen in frostbite might be caused by the same inflammatory mediators responsible for progressive dermal ischemia in the burn wound. They evaluated blister fluid from patients with hand frostbite, measuring levels of PGE_2, $PGF_{2\alpha}$, and thromboxane B_2 (TXB_2). Levels of the vasoconstricting, platelet-aggregating, and leukocyte-sticking prostanoids ($PGF_{2\alpha}$ and TXA_2) were markedly elevated. The investigators postulated that massive edema after cold injury was due either to leakage of proteins caused by release of these prostanoids or to leukocyte sludging in the capillaries and increased hydrostatic pressure. Recent studies have confirmed the similarity between cold injury and the burn wound.[8]

Severe endothelial damage was observed by researchers studying a minimal cold injury model in the hairless mouse.[5] In addition, the sequence of endothelial damage, vascular dilation, vascular incompetence, and erythrocyte extravasation was confirmed. This led to speculation that arachidonic acid metabolites, which may originate from severely damaged endothelial cells, are important in progressive tissue loss. Significantly absent from in vivo and microscopic observations were vascular spasm, thrombosis, and fibrin deposition, all of which have previously been implicated as pathophysiologic mechanisms. A rabbit ear model demonstrated increased tissue survival after blockade of the arachidonic acid cascade at all levels.[65] The most marked tissue salvage resulted when specific TXA_2 inhibitors were used. This has now been shown to be effective in clinical situations.[23]

A B C

Figure 8-1. **A,** Vesiculation of right ear with clear fluid, characteristic of second-degree frostbite. **B,** Minor first-degree frostbite of the ear, at 1 day post-thaw. Note blister formation on the back of the pinna. **C,** First-degree frostbite of the knee from kneeling to change a tire, at 12 hours post-thaw. (**B** and **C** courtesy Murray P. Hamlet, DVM.)

Skin Changes in Response to Frostbite

Reports in the 1940s documenting the histopathology of frostbite injury to the skin have been scarce and incomplete. Historically, studies by several investigators have been limited to skin biopsies without documentation of location, exposure time, temperature, or time elapsed since the injury.[71] More recently, experimental studies have been able to document the histopathology of skin changes under controlled conditions.

In 1988, Schoning and Hamlet[73,74] used a Hanford miniature swine model for frostbite injury (–75° C [–103° F] exposure for up to 20 minutes) to note progressive epithelial damage. Early changes included vacuolization of keratinocytes, and there was loss of intercellular attachments and pyknosis over a period of 1 week or more. This subsequently progressed to advanced cellular degeneration and formation of microabscesses at the dermoepidermal junction. Later changes included epithelial necrosis and regeneration, either separately or together within the same tissue. Such histopathologic data favor the current standard of conservative management of frostbite injury.

However, Marzella and associates[41] used a New Zealand white rabbit ear model of frostbite injury and proposed that the skin necrosis induced by frostbite injury was merely a reflection of damage to the target cell, the endothelial cell. After submersion of a shaved rabbit ear in 60% ethyl alcohol at –21° C for 60 seconds, the entire microvasculature demonstrated endothelial damage within 1 hour; erythrocyte extravasation occurred within 6 hours. These early vascular changes in the rabbit ear model are in contradistinction to the timing of vascular changes in the Hanford miniature swine model reported by Schoning and colleagues. These authors performed biopsies on animals exposed to frostbite injury (–75° C for up to 20 minutes) and evaluated the specimens for vascular inflammation, medial degeneration, and thrombosis.[74] The earliest change documented both grossly and microscopically was hyperemia. Within 6 to 24 hours, leukocyte migration and vasculitis were noted. However, the most severe vascular changes of thrombosis and medial degeneration were not documented for 1 to 2 weeks after the injury.

Whether or not the changes in the epidermis are primary or secondary to damage to the underlying endothelial cells, it is clear that these tissues have a potential, although limited, capacity for regeneration. This further supports a conservative approach to the management of frostbite injury.

Clinical Presentation

Classically, frostbite has been described by its clinical presentation. Initially it is difficult to predict the extent of frostbite damage.[29,53] Mills[52] favors the use of two simple classifications: mild (without tissue loss) and severe (with tissue loss). Historically, frostbite has been divided into "degrees" of injury based on acute physical findings after freezing and rewarming. First-degree injury shows numbness and erythema. There is a white or yellowish, firm plaque in the area of injury. There is no tissue loss, although edema is common. Second-degree injury results in superficial skin vesiculation (Fig. 8-1). A clear or milky fluid is present in the blisters, surrounded by erythema and edema. Third-degree injury shows deeper blisters, characterized by purple, blood-containing fluid (Fig. 8-2). This indicates that the injury has extended into the reticular dermis and beneath the dermal vascular plexus. Fourth-degree injury is completely through the dermis and involves the relatively avascular subcuticular tissues (Fig. 8-3B). This tends to cause mummification, with muscle and bone involvement (Fig. 8-4). Less severe bone injury in children may affect the growth plate and result in developmental digital deformities.[7,10]

More recently, Cauchy and colleagues[11] proposed a different classification of frostbite injury that is based on the risk of amputation of the affected part for the hand and foot. An early prognosis for frostbite patients may be delayed by the lack of useful clinical guidelines, and this new classification scheme (Tables 8-1 and 8-2) is intended to help resolve such issues. The four severity levels proposed provide earlier prediction of the final outcome of frostbite injury by using a technetium-99 bone scan in conjunction with the clinical findings on presentation. Grades 1 and 2 lesions do not require hospitalization or bone scans. Rapid rewarming and treatment with antibiotics and oral vasodilators appear to be sufficient for healing. Grade 3 lesions are connected with a significant risk of amputation and require rapid rewarming, antibiotics, aspirin, and intravenous vasodilators. Grade 4 lesions have a high risk of amputation and a high

Figure 8-2. **A,** Edema and blister formation 24 hours after frostbite injury occurring in an area covered by a tightly fitting boot. **B,** Deep second- and third-degree frostbite with hemorrhagic blebs, at 1 day post-thaw. (**A** courtesy Cameron Bangs, MD; **B** courtesy Murray P. Hamlet, DVM.)

Figure 8-3. **A,** Gangrenous necrosis of the frostbite injury shown in Figure 8-2A, 6 weeks later. **B,** Fourth-degree frostbite, at 4 weeks post-thaw. Note sharp line of demarcation under eschar, depigmentation of third-degree-frostbitten skin proximally, and indurated distal fingertips. Fingertips will auto-amputate. (**A** courtesy Cameron Bangs, MD; **B** courtesy Murray P. Hamlet, DVM.)

risk of complications such as thrombosis, sepsis, and other systemic problems (Table 8-3). Although this classification scheme may appear useful, it is important to note that the technetium-99 bone scan is crucial to the accuracy of the classification and subsequent therapy. In addition to the four severity levels, Cauchy and coworkers also noted that the probability of bone amputation for the hand and foot could also be correlated to the extent of frostbite injury seen at the time of initial presentation. In a review of 70 cases of severe frostbite injury, the probability of bone amputation was 1% for the distal phalanx, 31% for involvement of the middle phalanx, 67% for the proximal phalanx, and 98% and 100%, respectively, for involvement of the metacarpal/tarsal and carpal/tarsal bones (see Table 8-3).

Frostbite Symptoms

Severity of frostbite symptoms generally parallels severity of the injury. In the civilian population, frostbite most frequently occurs on the extremities, with the lower extremities being far more common. It can also appear on the nose or ears and has been reported on the scrotum and penis in joggers.[83]

Mere numbness followed by tingling after rewarming does not constitute bona fide frostbite. Even in its mildest form, true frostbite damages the affected tissue. In most patients, the initial clinical observation is coldness of the involved extremity, with numbness present in more than three fourths of patients. This ultimately causes the involved part to feel clumsy or absent and has been attributed to ischemia following intense vasoconstriction. When numbness is present initially, it is frequently followed by extreme pain (76% of patients) during rewarming. Throbbing pain begins 2 to 3 days after rewarming and continues for a variable period, even after the tissue becomes demarcated (22 to 45 days). After about a week, the victim usually notices a residual tingling sensation, probably caused by ischemic neuritis. For this reason, this sensation tends to persist longer than other symptoms. The severity of the injury usually defines the extent of neuropathologic damage. There may be a great deal of variation in symptoms, with some patients never noticing pain at the onset of the injury. In patients without tissue loss, symptoms usually subside within 1 month, whereas in those with tissue loss, disablement may exceed 6 months. In all cases, symptoms are intensified by a warm environment. Other sensory deficits include spontaneous burning and electric current–like sensations. The burning sensation, which is frequently early in presentation, subsides within 2 to 3 weeks. This sensation is not present in victims with tissue loss. In victims without tissue loss, the burning sensation may resume on wearing shoes or increasing activity. The electric current–like shock is almost universal (97%) in patients with tissue loss. It usually begins 2 days after injury, lasts for about 6 weeks, and is particularly unpleasant at night. All frostbite victims experi-

ence some degree of sensory loss for at least 4 years after injury and perhaps indefinitely.

The clinical appearance of frostbite may be deceiving.[4,60] Only a few patients arrive with tissue still frozen. At first, the extremity appears yellowish white or mottled blue. It may be insensate and may appear frozen solid. Regardless of the depth of the injury, the extremity may have the appearance of being frozen. With rapid rewarming there is almost immediate hyperemia, even with the most severe injury. Sensation returns after thawing and persists until blebs appear. At this point, some effort may be made to assess the severity of the injury. Although four degrees of injury have been defined, the differentiation usually made is between superficial (first- or second-degree) and deep (third- or fourth-degree) injuries. Even these distinctions are somewhat artificial, because the initial treatments of both are identical. The degree system may have some importance in retrospective analysis of cases, however.

Figure 8-4. **A,** Severe frostbite injury 3 days post-thaw. **B,** Severe frostbite injury 3 months post-thaw. (Photos courtesy Lanny Johnson.)

After the extremity is rewarmed, edema appears within 3 hours and lasts 5 days or longer, depending on the severity of the case.[60] Vesicles or bullae form 6 to 24 hours after rapid rewarming.[47,61] During the first 9 to 15 days, severely frostbitten skin forms a black, hard, and usually dry eschar, whether or not vesicles are present (Fig. 8-5).[61] Mummification forms an apparent line of demarcation in 22 to 45 days.[60]

Favorable prognostic signs include sensation to pinprick, normal color, warmth, and large, clear blebs that appear early and tend to extend to tips of the digits. Unfavorable signs include small, dark blebs that appear late and do not extend to digit tips, absence of edema, and presence of cyanosis that does not blanch with pressure.[52]

Techniques that have been used to estimate extent of injury and to predict ultimate tissue loss include intravenous radioisotope scanning (^{131}I radioiodinated serum albumin [RISA], ^{133}Xe, ^{99m}Tc-MDP, ^{99}Tc stannous pyrophosphate), angiography, routine roentgenography, and digital plethysmography. These modalities have also been recommended for estimating the vascular response to vasodilators and identifying tissue boundaries for surgical management. No prognostic technique is absolutely accurate in the immediate post-thaw period; a delay of 2 to 3 weeks is necessary to exceed the period of transitory vascular instability.

Ophthalmic Injuries

Ophthalmic injuries, particularly freezing of corneas, occur in individuals who try to force their eyes open in high windchill situations. Snowmobilers and cross-country skiers are particularly susceptible. Blurred vision, photophobia, blepharospasm, tearing, and pain on rewarming suggest such an injury. Rapid rewarming and patching are the only effective treatments. Corticosteroids are contraindicated. Corneal transplantation may

TABLE 8-1. Proposed Classification for Severity of Frostbite Injuries of the Extremities

	GRADE 1	GRADE 2	GRADE 3	GRADE 4
Extent of initial lesion at day 0 after rapid rewarming	Absence of initial lesion	Initial lesion on distal phalanx	Initial lesion on intermediary (and) proximal phalanx	Initial lesion on carpal/tarsal
Bone scanning at day 2	Useless	Hypofixation of radiotracer uptake area	Absence of radiotracer	Absence of radiotracer uptake area on the carpal/tarsal
Blisters at day 2	Absence of blisters	Clear blisters	Hemorrhagic blisters on the digit	Hemorrhagic blisters over carpal/tarsal
Prognosis at day 2	No amputation	Tissue amputation	Bone amputation of digit	Bone amputation of the limb ± systemic involvement ± sepsis
Sequelae	No sequelae	Fingernail sequelae	Functional sequelae	Functional sequelae

From Cauchy E, et al: Retrospective study of 70 cases of severe frostbite lesions: A proposed new classification scheme. Wilderness Environ Med 12:248–255, 2001.

TABLE 8-2. Management of Frostbite Injuries of the Extremities

On day 0, treatment consists of rapid rewarming for 2 hours in 38°C waterbath, with intravenous infusion of 400 mg chlorohydrate of vasodilator (buflomedil) and 250 mg aspirin. Subsequent treatment depends on the extent of the initial lesion, as follows:

GRADE 1* ABSENCE OF INITIAL LESION	GRADE 2 INITIAL LESION OVER DISTAL PHALANX	GRADE 3 INITIAL LESION OVER INTERMEDIARY (AND) PROXIMAL PHALANX	GRADE 4 INITIAL LESION OVER CARPAL/TARSAL
No hospitalization	Hospitalization 2 days	Hospitalization 8 days	Hospitalization intensive care unit
Oral treatment for 1 week (aspirin, vasodilator)	Possibly a bone scan at day 2	Bone scan at day 2	Bone scan at day 2
—	Oral treatment for 3 weeks (aspirin, vasodilators), dressing	IV administration for 8 days (aspirin, vasodilators), dressing	IV administration for 8 days (aspirin, vasodilators), dressing
—	—	Bone scan near day 8	Possibly antibiotics
—	—	Bone amputation near day 30	Early bone amputation near day 3 if sepsis
Recovery	Recovery with moderate sequelae	Bone amputation of the digit with functional sequelae	Bone amputation of the limbs with systemic involvement

*See Figure 8-1.
From Cauchy E, et al: Retrospective study of 70 cases of severe frostbite lesions: A proposed new classification scheme. Wilderness Environ Med 12:248–255, 2001.

TABLE 8-3. Probability of Amputation Based on the Extent of the Initial Lesion

	EXTENT (LEVEL OF INVOLVEMENT)	PROBABILITY OF BONE AMPUTATION (95% CI)
Hand	5 (carpal/tarsal)	100 —
	4 (metacarpal/metatarsal)	100 —
	3 (proximal phalanx)	83 (66;100)
	2 (intermediary phalanx)	39 (25;52)
	1 (distal phalanx)	1 (00;03)
Foot	5 (carpal/tarsal)	100 —
	4 (metacarpal/metatarsal)	98 (93;100)
	3 (proximal phalanx)	60 (45;74)
	2 (intermediary phalanx)	23 (10;35)
	1 (distal phalanx)	0 —
Hand and foot	5 (carpal/tarsal)	100 —
	4 (metacarpal/metatarsal)	98 (95;100)
	3 (proximal phalanx)	67 (55;79)
	2 (intermediary phalanx)	31 (22;41)
	1 (distal phalanx)	1 (00;02)

From Cauchy E, et al: Retrospective study of 70 cases of severe frostbite lesions: A proposed new classification scheme. Wilderness Environ Med 12:248–255, 2001.

Figure 8-5. Frostbitten toes demarcated at 3 weeks after injury. Note the cotton padding between the toes. Minimal tissue loss occurred. (Photo by Paul Auerbach, MD.)

be required if keratitis and corneal opacification are irreversible and debilitating. Snowblindness caused by ultraviolet exposure is discussed in Chapter 25.

Management

In 1947, Hurley stated, "Tissue cells can be affected by freezing in three different ways: (1) a certain number of cells are killed; (2) a certain number remain unaffected; and (3) a large number are injured but may recover and survive under the right circumstances."[25] Clearly, the major treatment effort must be to salvage as many cells in the third group as possible.

Frostbite treatment is directed separately at the pre-thaw and post-thaw intervals. If a patient is referred from a nearby location, no attempt at field rewarming may be indicated. Vigorous rubbing is ineffective and potentially harmful. The extremity should not be intentionally rewarmed during transport and should be protected against slow partial rewarming by keeping the patient away from intense campfires and car heaters. All constrictive and wet clothing should be replaced by dry, loose wraps or garments. The extremity is padded and splinted for protection, but no other treatment is initiated. Although there is correlation between the length of time tissue is frozen and the amount of time required to thaw that tissue, there is no direct correlation between the length of time tissue is frozen and subsequent tissue damage. Therefore, rapid transport of frostbite patients (within 2 hours) is appropriate. Otherwise, rapid

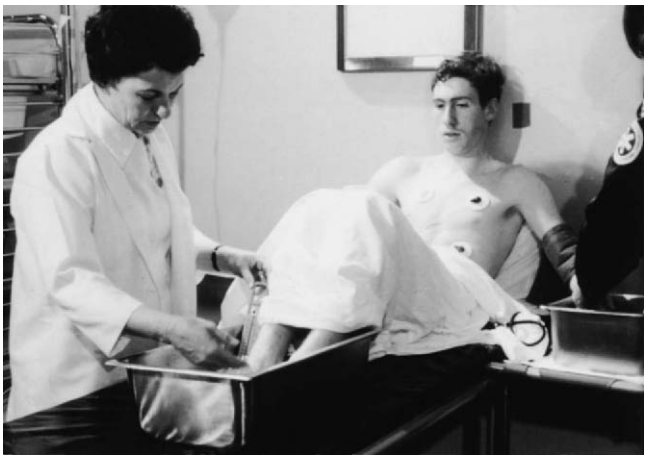

Figure 8-6. Technique to rapidly warm all four extremities simultaneously. The water is 40° to 42° C (104° to 108° F), monitored by a thermometer and circulated manually. Thawing is usually complete in 30 minutes, when the extremities are pliable and color and sensation have returned.

rewarming should be instituted and the victim transported with protective dressings to prevent refreezing. Blisters should be left intact. Patients with long transport times are at greater risk for recurrent injury. All efforts should prevent subsequent refreezing, as this creates an infinitely worse result than delayed thawing. A victim who must walk through snow should do so before thawing frostbitten feet. During transport, the extremities should be elevated and tobacco smoking prohibited.[51] Alcohol ingestion is contraindicated.

Once in the emergency department, rapid rewarming should be started immediately. Associated traumatic injuries or medical conditions should be identified. Systemic hypothermia should be corrected to a core temperature of at least 34° C (93° F) before frostbite management is attempted. Fluid resuscitation is usually not a problem with frostbite injuries, although one case of rhabdomyolysis and acute renal failure has been reported.[70]

Treatment is directed at the specific pathophysiologic effects of the frostbite injury, either blocking direct cellular damage or preventing progressive microvascular thrombosis and tissue loss. Direct cellular damage is treated by rapid thawing of all degrees of frostbite with immersion in gently circulating water warmed to between 40° and 42° C (104° and 108° F) (Fig. 8-6).[28] Adherence to this narrow temperature range is important, as rewarming at lower temperatures is less beneficial for tissue survival,[18,23] and rewarming at higher temperatures may compound the injury by producing a burn wound.[55] Heated tap water (50° to 60° C [122° to 140° F]) is too hot and will cause profound discomfort and possible tissue damage. Frozen extremities should be rewarmed until the involved skin becomes pliable and erythematous at the most distal parts of the frostbite injury. This usually takes less than 30 minutes. Active motion during rewarming is helpful, but massage may compound the injury. Extreme pain may be experienced during thawing, and unless otherwise contraindicated, parenteral analgesics are administered. Rapid return of skin warmth and sensation with the presence of an erythematous color is a favorable sign, whereas the persistence of cold, anesthetic, and pale skin is unfavorable.

Rapid rewarming reverses the direct injury of ice crystal formation in the tissue. However, it does not prevent the progressive phase of the injury. McCauley and associates[44,45] have designed a protocol based on the pathophysiology of progressive dermal ischemia that has been quite successful in minimizing production of local and systemic thromboxane by injured tissues (Box 8-1). All but the most minor frostbite cases should be admitted to the hospital. Victims with minor injuries should be admitted if, after rapid rewarming, a warm environment cannot be ensured for the patient. No patient should ever be discharged into subfreezing weather. Even with a warm car waiting, the patient should be allowed to leave only with proper clothing (such as stocking cap, wool mittens, and wool socks).

Because the majority of frostbite injuries necessitate admission to the hospital, a discussion of the protocol is warranted. White or clear blisters, which represent more superficial injury, are debrided to prevent further contact of $PGF_{2\alpha}$ or TXA_2 with the damaged underlying tissues. Unlike the clear blisters, hemorrhagic blisters reflect structural damage to the subdermal plexus. It may be worthwhile to aspirate the thromboxane-containing fluid out of these blisters, but debridement may allow desiccation of the deep dermis and allow conversion of the injury to a full-thickness injury. Therefore, hemorrhagic blisters are left intact. A specific thromboxane inhibitor is placed on the wounds to prevent further formation of this vasoconstricting mediator (Fig. 8-7). Aspirin was originally given systemically to block production of $PGF_{2\alpha}$ and TXA_2, but because the correct dose of aspirin is not known, it has been replaced by ibuprofen.[80] Ibuprofen not only inhibits the arachidonic acid cascade but has the additional benefit of fibrinolysis. Elevation of the extremity minimizes dependent edema. Because edema inactivates the normal streptococcidiocidal properties of the skin, parenteral penicillin is administered during the edema phase of the injury.[12] Antitetanus prophylaxis is given if indicated by the patient's history. Tetanus killed thousands of Napoleon's soldiers in the Russian campaign. There was a report as recently as April 1985 documenting tetanus associated with a frostbite injury. Because frostbite generally involves injuries to the extremities, appropriate physical and occupational therapy should be initiated early. Daily hydrotherapy for active and passive range of motion is extremely valuable in the preservation of function.[15]

Between 1982 and 1985, 56 patients were treated with the previous frostbite protocol and 98 were treated by other "standard" methods. Of the 56 patients treated with the protocol, 18 were admitted with first-degree frostbite, 25 with second-degree frostbite, and 13 with third-degree frostbite. Thirty-two suffered acute frostbite, and 24 had subacute (presenting for treatment more than 24 hours after injury) frostbite. The mean length of hospital stay for the acute protocol patients was 8.5 days and for the subacute group, 14.9 days. Overall, 67.9% of the protocol group healed without tissue loss, 25% healed with some tissue loss, and 7% required amputation. Of the nonprotocol patients, 11 were admitted with first-degree frostbite, 51 with second-degree frostbite, and 36 with third-degree frostbite. Fifty-six were acute cases, and 42 were subacute. The mean length of hospital stay was 17.5 days for the acute, nonprotocol patients and 19 days for the subacute group. Of this group, 32.7% healed without tissue loss, 34.6% healed with tissue loss, and 32.7% required amputation ($P<.001$, includes all groups of protocol-treated patients). These data indicate thera-

Box 8-1. Protocol for Rapid Rewarming

1. Admit frostbite patients to a specialized unit if possible.
2. Do not discharge or transfer to another facility victims of acute frostbite requiring hospitalization unless it is necessary for specialized care. Transfer arrangements must protect the victim from cold exposure.
3. On admission, rapidly rewarm the affected areas in warm water at 40° to 42° C (104° to 108° F), usually for 15 to 30 minutes or until thawing is complete.
4. On completion of rewarming, treat the affected parts as follows:
   a. Debride white blisters and institute topical treatment with aloe vera (Dermaide aloe) every 6 hours.
   b. Leave hemorrhagic blisters intact and institute topical aloe vera (Dermaide aloe) every 6 hours.
   c. Elevate the affected part(s), with splinting as indicated.
   d. Administer antitetanus prophylaxis.
   e. For analgesia, administer morphine or meperidine (Demerol) intravenously or intramuscularly as indicated.
   f. Administer ibuprofen (400 mg orally) every 12 hours.
   g. Administer penicillin G (500,000 units intravenously) every 6 hours for 48 to 72 hours.
   h. Perform hydrotherapy daily for 30 to 45 minutes at 40° C (104° F). The solution should meet the following specifications:
      (1) Large tank capacity, 425 gal
      Fill-level estimate, 285 gal
      Sodium chloride, 9.7 kg
      Calcium hypochlorite solution, 95 mL
      (2) Medium tank capacity, 270 gal
      Fill-level estimate, 108 gal
      Sodium chloride, 3.7 kg
      Calcium hypochlorite solution, 36 mL
      (3) Small tank capacity, 95 gal
      Fill-level estimate, 72 gal
      Sodium chloride, 2.5 kg
      Potassium chloride, 71 g
      Calcium hypochlorite solution, 24 mL
5. For documentation, obtain photographs on admission, at 24 hours, and serially every 2 to 3 days until discharge.
6. Discharge patients with specific instructions for protection of the injured areas to avoid re-injury and follow up weekly until wounds are stable. If the patient is being discharged with no open lesions, instruct him or her to use wool socks, wear a hat, and use mittens instead of gloves to decrease the loss of heat between the fingers. Explain to patients that they are now more susceptible to refreezing, so they should avoid exposure to cold and should wear warm clothing and shoes or boots when going outside is necessary. Give similar instructions to patients who are discharged with open lesions. Also instruct these patients to keep the affected extremity elevated and to take ibuprofen (400 mg orally) every 12 hours. Aloe vera should be applied to the involved areas, or scarlet red ointment used if the open areas are small.

peutic efficacy of the protocol by virtue of less tissue loss, lower amputation rate, and significantly shorter hospital stay.[23]

Other Therapies

A number of other therapeutic modalities have been suggested in an attempt to prevent progressive thrombosis and tissue loss. Most were proposed before the pathophysiology of the injury was found to be related to inflammatory mediators. These therapeutic attempts deserve comment.

Dextran

It has been observed that cold-injured vessels, shortly after thawing, become dilated and filled with clumps of erythrocytes. These clumps can be easily dislodged by gentle manipulation and do not represent true thrombosis. Although the mechanism that leads to erythrocyte clumping is not completely understood, it may reflect a cold-induced increase in blood viscosity. This suggests that the use of low-molecular-weight dextran may be of benefit in the early treatment of frostbite. Although no controlled clinical trial of low-molecular-weight dextran has been reported, there has been experimental evidence to support its use. Weatherly-White and colleagues[84] demonstrated that the use of low-molecular-weight dextran, 1 g/kg/day, protected against tissue loss in the rabbit ear model. This led to the suggestion that the use of 1 L of 6% dextran intravenously on the day of injury, followed by 500 mL on each of 5 successive days, might be of benefit.[69] With our present understanding of the etiology of frostbite, there appear to be few instances when it may be used and have demonstrable benefit.

Heparin

Although true thrombi are not present in dilated, erythrocyte-filled vessels immediately after thawing, they do form over the next few days. Heparin has been suggested as a possible treatment for frostbite. Lange and Loewe[34] demonstrated its usefulness in experimental frostbite. Subsequent investigations have been unable to substantiate these findings, and there is no evidence that heparin alters the natural history of the frostbite.[75]

Reserpine

Intra-arterial reserpine has also been shown experimentally to be of use in frostbite. Porter and coworkers[62] reported its clinical use in five patients, three of whom were treated within 2 weeks of injury. Angiography of the involved extremities was performed before and after injection (Fig. 8-8). Reserpine appeared to be effective in relieving vasospasm. However, treatment did not retard progression of tissue loss. A multiarmed clinical study compared slow rewarming and intravenous dextran, intra-arterial tolazoline, and intra-arterial reserpine, separately and in various combinations.[78] All of the drug treatments were superior to simple slow rewarming. However, rapid rewarming was as effective as any of the drug treatments. Therefore, reserpine might be effective when patients are brought for treatment late and have not been rapidly rewarmed. To date, no anticoagulant or vasodilation treatment has proved useful in controlled clinical trials.

Pentoxifylline

As noted, a number of articles have detailed the mechanism by which tissue damage occurs with cold exposure. Pentoxifylline is a methylxanthine-derived phosphodiesterase inhibitor commonly used in the treatment of intermittent claudication, arterial disease, and peripheral vascular disease. Miller and Koltai

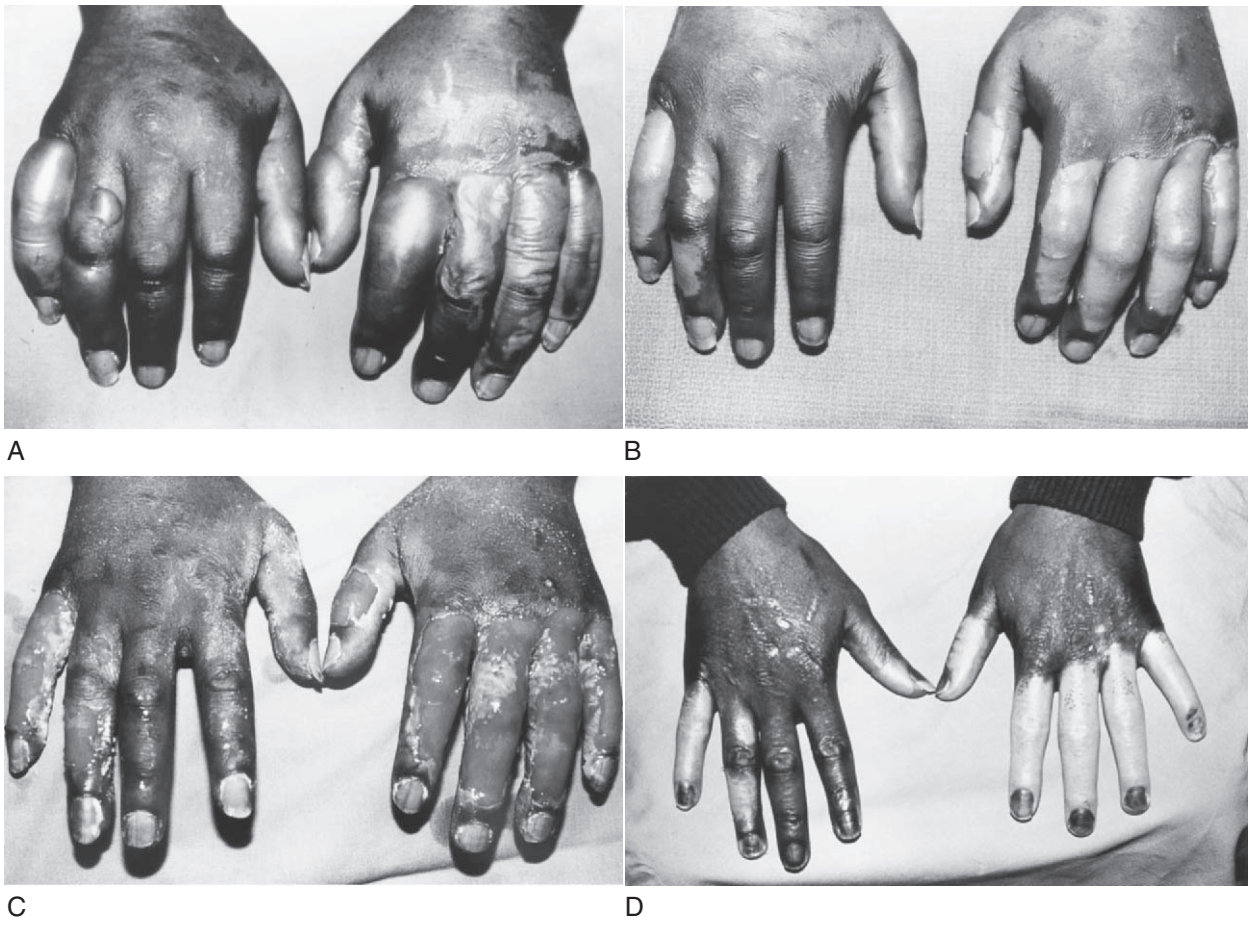

Figure 8-7. **A,** Acute frostbite injury with clear fluid in superficial blisters. **B,** Blisters before treatment but after debridement. **C,** After 48 hours of therapy with topical aloe vera (Dermaide aloe). **D,** Sixteen days after injury.

suggested that it may be effective in the experimental treatment of frostbite.[49]

Several investigators believe that pentoxifylline increases tissue survival by increasing red blood cell flexibility, decreasing thrombus function, decreasing blood viscosity, and blocking platelet activity.[49] In addition, pentoxifylline is believed to act by increasing leukocyte flexibility and impairing leukocyte ability to adhere to endothelium. Such actions, in return, protect the vascular endothelium from damage induced by polymorphonuclear leukocytes. Theoretically, this appears to be an alternative solution to both erythrocyte clumping and cold induced erythrocyte clumping, and should enhance survivability of tissues.[49] Hayes and coworkers[21a] have proposed a treatment using pentoxifylline in conjunction with the traditional therapy of rewarming, soaks, pain management, and vesicle debridement. They recommend pentoxifylline in the controlled-release form of one 400-mg tablet three times a day with meals. The duration should be from 2 to 6 weeks. Controlled studies of pentoxifylline in the management of frostbite have yet to be performed.

Sympathectomy

Several reports from Europe and the United States after World War II supported the use of sympathetic block and sympathectomy.[6] However, this remains controversial. If the sympathetic nervous system is involved, it seems reasonable that early sympathectomy would be beneficial. However, experimental evidence indicates that sympathectomy performed within the first few hours of injury increases edema formation and accelerates the pathologic process of tissue destruction.[19,84] On the other hand, sympathectomy performed 24 to 48 hours after thawing seems to hasten resolution of edema and decrease tissue loss.[19] Taylor[79a] presented a series of patients in which sympathectomy was used to control the associated symptoms of frostbite, such as pain, paresthesias, numbness, and hypesthesias. A continuous bupivacaine epidural blockade was performed for 48 hours, and relief of symptoms was obtained. When the epidural blockade did not work, bilateral lumbar sympathectomy was performed and relief was obtained. This series was small and the follow-up was for only 1 year, but it was observed that there was no recurrence of symptoms. The role of sympathectomy will not become clear until there is a verifiable clinical rating to evaluate results.[14]

Hyperbaric Oxygen

Similar problems exist in evaluating the use of hyperbaric oxygen (HBO). Although some experimental data support its

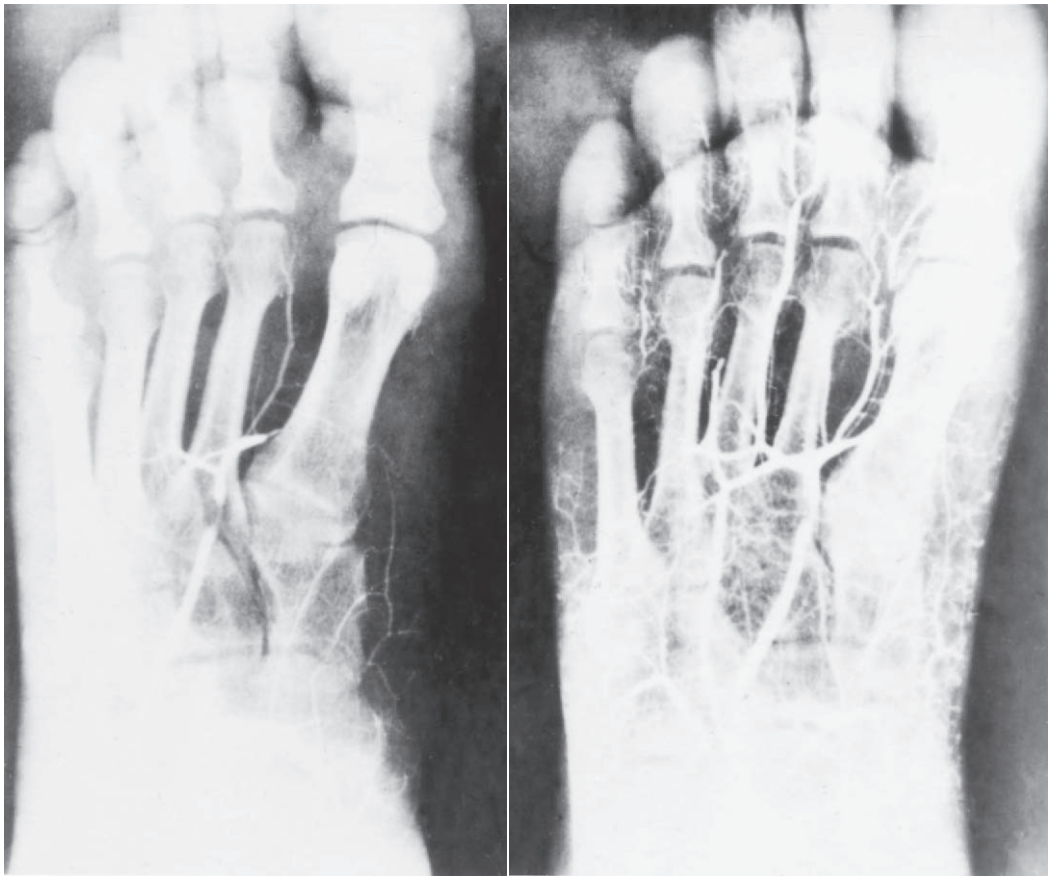

Figure 8-8. Angiograms of the left foot of a 30-year-old man with cold injury. **A,** Initial angiogram. **B,** Angiogram taken 2 days after intra-arterial injection of reserpine. (From Gralino BJ, Porter JM, Rosch J: Radiology 19:301–305, 1976, with permission.)

use, only occasional anecdotal cases are reported.[59] A number of anecdotal reports expound the beneficial effects of HBO on the preservation of tissue viability in frostbite patients. Although the proposed mechanism of action is multiple, the major changes are postulated to occur in the microcirculation. HBO reportedly increases erythrocyte flexibility and decreases edema formation in postischemic tissues, and it is bacteriostatic. Such actions may counteract the vascular congestion, platelet aggregation, and infiltration of leukocytes seen in the microcirculation of frostbite patients. Finderle and Canker[16] report successful HBO treatment of a patient at 2.5 atm for 90 minutes daily for 28 sessions in a multiplace chamber, without significant tissue loss. Treatment started 12 days after injury. Such success in the delayed treatment of frostbite injuries has been reported by other investigators. It is interesting that in one case report, von Heimburg and colleagues[81] noted that hyperbaric treatment was useful in preventing the late changes in growing bones. To date, no series of randomized patients with frostbite injury treated with HBO has been reported.

Early Surgical Intervention

Early surgical intervention has no role in the acute care of frostbite unless there is ischemia from a constricting eschar or subeschar infection that cannot be controlled by topical antimicrobials. Decompressing escharotomy incisions are rarely necessary to increase distal circulation. If such escharotomies are necessary to decompress digits and facilitate joint motion, incisions along the transaxial line are best. It is important that incisions avoid injury to the underlying structures. If uncontrolled infection is present early, escharotomy may be necessary. However, this is rare with the use of penetrating antibacterial agents such as mafenide acetate.

Surgical intervention is properly reserved for late treatment of frostbite. This is most often necessary if frostbite is very severe or if treatment has been delayed. Normally, aggressive therapeutic measures can prevent progressive injury and gangrene. If gangrene ensues, amputation or debridement with resurfacing may be necessary (Fig. 8-9).

Surgery should be accomplished only after the area is well demarcated, which generally requires 3 to 4 weeks. Historically, aggressive early debridement and attempted salvage have been thought to jeopardize recovering tissue and add to tissue loss. Gottlieb and coworkers[20] have taken a more aggressive approach to coverage of severe frostbite injury. Using technetium-99 phosphate and bone scans, they identify nonperfused tissue by the tenth day after injury and surgically remove

A B

Figure 8-9. **A,** Gangrene after third-degree frostbite. **B,** After partial hand amputation in preparation for toe-to-hand transfer.

necrotic tissue. The remaining nonvascularized, nonviable, yet non-necrotic and noninfected tissue is salvaged by early coverage with well-vascularized tissue. Theoretically, if nonvascularized tissue has not undergone autolysis and is not infected, it should behave like a composite graft. Preliminary reports are promising.[20]

Late Sequelae

Until 1957, little was recorded about the long-term sequelae of frostbite injuries. Blair and colleagues[3] studied 100 veterans of the Korean conflict 4 years after their injuries. In order of decreasing frequency, the victims reported excessive sweating, pain, coldness, numbness, abnormal skin color, and stiffness of the joints. In addition, the investigators noted frequent asymptomatic abnormalities of the nails, including ridges and inward curving of the edges. In general, the degree of long-term disability was related to the severity of the original injury. Symptoms were worse in cold than in warm weather. This is attributed to the fact that vessels do not react as well to stress.[77] Previously injured blood vessels do not constrict when exposed to cold as effectively as normal vessels do, and they do not dilate as effectively when vasoconstriction is blocked.

Hyperhidrosis is probably both a cause and a result of frostbite. Hyperhidrosis suggests the presence of an abnormal sympathetic nervous response induced by cold injury and is abolished by sympathetic denervation. Sensitivity to cold and predisposition to recurrent cold injury should suggest hyperhidrosis. Blanching and pain on subsequent cold exposure may be a nuisance or may be dramatic enough to suggest a diagnosis of Raynaud's phenomenon (Fig. 8-10). Almost without exception, a painful, shiny, cyanotic, and sweaty limb becomes warm, dry, and useful with sympathetic interruption.[73,74] Schoning[72] examined the changes in the sweat glands of Hanford miniature swine after experimental frostbite injury to determine the etiology of hyperhidrosis. She noted that severe sweat gland changes were of two types: degeneration with necrosis and squamous metaplasia. Clearly, if hypohidrosis were a sequela of frostbite injury, morphologically normal and active sweat glands would be an expected finding. We can probably conclude that hyperhidrosis lacks histologic documentation.

The late abnormalities of change in skin color, including depigmentation of dark skin and an appearance resembling erythrocyanosis in light skin, are most likely the result of ischemia.[3] Similarly, the nail abnormality is comparable to that seen with ischemia. Usually, neither of these sequelae requires treatment.

Late symptoms of joint stiffness and pain on motion are relatively common and are undoubtedly related to the underlying scars and mechanical problems occasioned by the variety of amputations. "Punched-out" defects in subchondral bone of involved limbs have been noted. These localized areas of bone resorption generally appear within 5 to 10 months after injury and may heal spontaneously. Vascular occlusion is the probable cause of these lesions. Such bone involvement in close proximity to joint surfaces may help to explain joint symptoms.

The effects of frostbite on premature closure of epiphyses in the growing hand have recently been reemphasized.[86] Extent of premature closure has been correlated with severity of frostbite and noted in partial-thickness injuries. In the digits, premature closure is more frequent from a distal to proximal direction (distal interphalangeal greater than proximal interphalangeal greater than metacarpophalangeal). The thumb has been less often involved. In only 2% of cases does partial epiphyseal closure cause angular deformity.

One of the characteristic features of frostbite is the surprising salvage possible in an apparently badly injured extremity. Amputation should therefore be performed with great conservatism and as late as possible. The line of demarcation may not be decided definitively for weeks or months after injury. As long as secondary sepsis does not intervene, patience often rewards both the patient and the surgeon with maximal length of the useful limb. This is particularly true for the upper extremities.

Figure 8-10. A 54-year-old man had an acute primary episode of Raynaud's disease after surfing for 80 minutes in water that was 21°C (70°F). The distinctive asymmetric pallor of the terminal phalanges of the fourth digit on the right hand persisted for 40 minutes. A sharp demarcation between the second and third phalangeal joints was evident, but no other symptoms were apparent. The pallor spontaneously resolved without cyanosis or redness of the affected area. Medical evaluation subsequently revealed no disorder known to cause secondary Raynaud's phenomenon. (From Bluto MJ, Norman DA: Acute episode of Raynaud's disease. N Engl J Med 347:992, 2002.)

Conditioning and Prevention

Frostbite can usually be prevented by experienced leadership, good physical condition, adequate food, and intelligently used equipment. Probably the two most important basic factors in prevention of frostbite are the heat-producing capacity of the body and the effectiveness of measures to conserve heat once it has been produced. The most fundamental defense against frostbite is a healthy body.

As previously noted, there are no data to support the belief that high altitude increases susceptibility because of changes in blood viscosity. On the other hand, living conditions at high altitude may contribute to increasing danger from frostbite. Cold becomes more intense, as do the violence and severity of storms. Shelter is less appealing and protective. Loss of sleep, inadequate diet, altered digestion, and nervous tension all contribute to a greater degree of fatigue than normally experienced at lower heights. Hypoxia reduces reasoning powers and leads to a tendency toward laziness, carelessness, indecision, and lack of normal insight and judgment.[83]

The body requires the same amount of oxygen for various tasks regardless of the altitude. At higher altitudes, however, the body must process more air to gain that same amount of oxygen. Individuals must carefully regulate their activity, or they will not only exhaust the body but will also cool it. Large quantities of heat can be lost through the lungs by panting and overexertion,[63] which also adds to fatigue.

At higher altitudes, the natural protective mechanism of shivering is impaired and heat production during rest can be significantly reduced. If an individual becomes unable to exercise, the body cannot maintain adequate heat production. The body reduces blood flow to its surface and extremities in an effort to maintain core temperature. In such a situation, no amount of insulation can prevent frostbite and nothing but vigorous warming and administration of oxygen can avert serious results. Since the use of auxiliary oxygen was introduced in mountaineering, frostbite has become less common.

Cold weather increases caloric needs, and variations in diet can have an effect on tolerance.[56] In the cold, cooking, dishwashing, thawing food, and melting water are arduous tasks. Fluid requirements far exceed what is empirically thought to be necessary.

Frostbite is rarely experienced by healthy individuals who are standing still and adequately clothed. It seems to be related almost always to factors such as fatigue, a sudden storm, an accident, or combinations of these. Alcohol intoxication is a frequent forerunner of frostbite. Injury to any part of the body, combined with fear or panic, can allow frostbite in a situation where climatic effects alone would not cause trouble to a seasoned, uninjured person.[83]

Because impaired local circulation is the primary cause of frostbite, anything with even a mildly adverse effect on normal peripheral circulation, such as tobacco or alcohol, should be avoided. Smoking results in varying degrees of diffuse vascular spasm, reducing normal peripheral circulation. Alcohol is not recommended at any time on the trail.[22]

Previous frostbite injury usually results in a prolonged reduction in tolerance to cold by the injured part. However, the concept of physiologic adaptation to cold persists, although it is extremely controversial. There can be no argument that Eskimos and Tibetans in cold, rugged climates "feel" cold much less than do non-natives. They certainly seem to resist cold more than others and clearly tolerate cold better than those who live in temperate and tropical climates. However, many medical experts allege that this "resistance" is really no more than the result of experience in Arctic survival and day-to-day living. Furthermore, Eskimos, even though they do not feel cold as fast, freeze just as fast and badly as others, given the same actual contact situation. There is increasing evidence that tolerance exists, but even if it is proved, it may be of only academic interest. The basic reason most outdoorsmen are not frostbitten is that they tend to be in good physical condition and know how to act and dress properly.

Many physicians enjoy mountain climbing. Those involved in Everest exploration wrote about the effects of cold and how to protect against it.[45,83] They suggested that overall physical well-being, good clothing, and intelligent operations in the field were by far the best insurance against frostbite. When a person is exhausted, hungry, ill, injured, or hypoxic, the chances for frostbite injury are increased.[22]

The following measures for frostbite protection are based on Washburn's recommendations[83]:

1. Dress to maintain general body warmth. In cold, windy weather, the face, head, and neck must be protected, as enormous amounts of body heat can be lost through these parts.
2. Eat plenty of appetizing food to produce maximal output of body heat. Diet in cold weather at low altitude should tend toward fats, with carbohydrates intermediate and proteins least important. As altitude increases above 10,000 feet, carbohydrates become most important and proteins remain least.
3. Do not climb under extreme weather conditions, particularly at high altitudes on exposed terrain, or get too early

a start in cold weather. The configuration of a mountain can help the climber find maximal shelter and solar warmth.

4. Avoid tight, snug-fitted clothing, particularly on the hands and feet. Socks and boots should fit closely, with no points of tightness or pressure. In putting on socks and boots, a person should carefully eliminate all wrinkles in socks. Old matted insoles are to be avoided.
5. Avoid perspiration under conditions of extreme cold; wear adequately ventilated clothing. If perspiring, remove some clothing or slow down.
6. Keep the feet and hands dry. Even with vapor-barrier boots, socks must not become too wet. All types of boots must be worn with great care during periods of inactivity, especially after exercise has resulted in damp socks or insoles. Wet socks in any type of boot soften the feet and make the skin more tender, greatly lowering resistance to cold and simultaneously increasing the danger of other foot injuries, such as blistering. Extra socks and insoles should always be carried. Light, smooth, dry, clean socks should be worn next to the skin, followed by one or two heavier outer pairs.
7. Wear mittens instead of gloves in extreme cold, although gloves can be worn for short intervals when great manual dexterity is required for specialized work such as photography or surveying. In these situations, a mitten should be worn on one hand and a glove temporarily on the other. If bare-finger dexterity is required, silk or rayon gloves should be worn, or metal parts that must be touched frequently should be covered with adhesive tape. Occasionally, the thumbs should be pulled into the fists and held in the palm of the mittens to regain the warmth of the entire hand.
8. Be careful while loading cameras, taking pictures, or handling stoves and fuel. The freezing point of gasoline (–57° C [–70° F]) and its rapid rate of evaporation make it very dangerous. Metal objects should never be touched with bare hands in extreme cold, or in moderate cold if the hands are moist.
9. Mitten shells and gloves worn in extreme cold should be made of soft, flexible, dry-tanned deerskin, or moose, elk, or caribou hide. Horsehide is less favorable because it dries stiffly after wetting. Removable mitten inserts or glove linings should be of soft wool.
10. Mittens should be tied together on a string hung around the neck or tied to the ends of parka sleeves. Oiled or greased leather gloves, boots, or clothing should never be used in cold weather operations.
11. Keep toenails and fingernails trimmed.
12. Hands, face, and feet should not be washed too thoroughly or too frequently under rough weather conditions. Tough, weather-beaten face and hands resist frostbite most effectively.
13. Wind and high altitude should always be approached with respect. They can produce dramatic results when combined with cold. Exercise should not be too strenuous in extreme cold, particularly at high altitude, where undue exertion results in panting or very deep breathing. Cold inspired air will chill the whole body and under extreme conditions may damage lung tissue and cause internal hemorrhage.
14. When a person becomes thoroughly chilled, it takes several hours of warmth and rest to return to normal, regardless of superficial feelings of comfort. A person recovering from an emergency cold situation should not venture out again into extreme cold too soon.
15. Avoid tobacco or alcohol at high altitudes and under conditions of frostbite danger.
16. A person who is frostbitten or otherwise injured in the field must remain calm. Panic or fear results in perspiration, which evaporates and causes further chilling.
17. Tetanus immunity should be current.

For information on nonfreezing cold-induced injuries, see Chapter 7.

FROSTNIP

Frostnip is superficial and reversible ice crystal formation associated with intense vasoconstriction. It is characterized by discomfort in the involved parts. The symptoms usually resolve spontaneously, and no tissue is lost. There is some question whether this qualifies as cold-induced injury, as neither frozen extracellular water nor progressive tissue loss is routinely demonstrated.

SUMMARY

The morbidity of progressive dermal ischemia in frostbite may be decreased by the therapeutic use of specific inhibitors of the eicosanoid cascade. Additionally, continual surveillance of the injured site is essential in salvaging the frostbitten limb. Strict adherence to the therapeutic protocol is an important component in saving any injured tissue.

We postulate that utilization of the triple–phase scanning technique combined with the therapeutic approach outlined in Box 8-1 would act synergistically and enhance tissue survival of frostbitten tissue.

ACKNOWLEDGMENTS

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The references for this chapter can be found on the accompanying CD-ROM.

9 Polar Medicine

Betty Carlisle and Ian Davis

Like much of wilderness medicine, polar medicine's identity derives to a large extent from its setting. *Polar,* however, can be defined in several different ways. Geographically, the Arctic and Antarctic circles, at latitudes 66°33′ north and south, delimit areas in which the sun does not rise or set on at least 1 day of the year. A more functional definition is the 10° isotherm, which joins those areas in which the average temperature in the warmest month of the year is 10°C (50°F); this correlates roughly with the tree line.

For medical purposes, the definition of *polar* is more complex. Although climate and geography are clearly important, the salient features of polar medicine are logistic and experiential. Unlike high-altitude medicine or hyperbaric medicine, for example, polar medicine is not unified by an underlying pathophysiology. High-altitude medicine is a medical field because of the environment's effect on the human organism, but polar medicine is largely medical practice within the setting.

Medical practice in isolated settings is paradigmatic of wilderness medicine: the patient population is essentially, if not literally, a small demographic island. A provisional definition of *polar medicine,* then, could be "the practice of medicine in isolated settings within an extreme cold environment."[78] It is the cold, remoteness, hostility, and unforgiving nature of the environment in which humans struggle to survive, let alone work, that makes medicine in polar areas, and in particular Antarctica, so challenging.[73]

THE DISTINCTION BETWEEN ARCTIC AND ANTARCTIC MEDICINE

Although the polar regions share the predominant attributes of cold, dark, isolation and severe weather, they display many important contrasts. The Arctic has been described as a frozen sea nearly surrounded by land, whereas Antarctica is a land and ice mass circumscribed by ocean. The moderating effect of the North Polar waters compared with the elevation (2835 m [9302 feet]) of the South Polar plateau accounts for the roughly 40°C (72°F) differences in wintertime low temperatures. On a plot of temperature versus humidity, the polar plateau is more similar to Mars than to the rest of the earth. The flora and fauna are unique to each area. The Arctic has an abundance of land- and sea-based animals as well as migratory birds and plant life. On the other hand, the Antarctic has no permanent land-based animals, although there are migratory birds, as well as a rich marine life and a limited plant life.

The differences in population patterns mark an important north–south disjunction in polar studies in general and in polar medicine in particular. The northern polar or frigid areas above 66° latitude comprise a frozen sea that is surrounded by eight countries, each with its unique set of populations and medical care services. The countries of the Arctic are Canada, Finland, Greenland (Denmark), Iceland, Norway, Russia, Sweden, and the United States. Human population is scattered throughout the Arctic, with several relatively large urban areas, but for the most part the Arctic is sparsely populated.[169] The overall estimated population of the Arctic as defined by the Antarctic Monitoring and Assessment Programme (AMAP) is approximately 4.5 million; indigenous peoples make up 30% of that population.[92,168] Within the Arctic, there are eight major indigenous peoples and more than 30 minority groups. Some of these peoples have separate governments, languages, and socioeconomic possibilities.[92,203]

There are no indigenous peoples in Antarctica, which is regulated by the Antarctic Treaty, with 12 original signatory countries and currently a total of 45 signatories.[42,173,174] This complex treaty helps delineate the relationships of the countries that are active in Antarctica and how the continent is to be best preserved and used for peaceful purposes. Seven countries claim territorial rights.[39,174] Of the 77 stations in Antarctica, 43 are year round.[173] The total population living in Antarctica, all seasonal, now exceeds 4000 persons in summer and well over 1000 in winter.[173,174]

In the Arctic, there are two overlapping but distinct spectra of medical problems—those of indigenous Arctic people and those of visitors and immigrants to the polar region. Many of the medical problems among the Inuit, Sami, Chukchi, Nenet, and other northern groups are those of populations making the often Faustian demographic and cultural transition to a Western industrialized society. Thus, medical practice among these groups is similar to that among many other displaced indigenous populations.[25] In a sense, the current illnesses and health risks of the indigenous Arctic populations are the result of contamination from distant sources and too much contact with other cultures.[25] In contrast, medical problems in Antarctica among the usually young and fit scientific, commercial, tourist, or expedition support personnel are often related to trauma and the environment and thus fall more appropriately under the purview of travel or expedition medicine. Furthermore, tourist ships often bring older adults, whose complex medical problems can create logistical dilemmas. Medical problems of sojourners to either polar region are largely the result of too little connection with the culture and medical facilities of home.

Thus there is a disjunction in polar medicine between the indigenous and sojourner populations that poses a challenge to a meaningful synthesis. It makes sense to focus not only on the health of elite visitors to these remote areas but also on the health of the local population and the effects that visitors can have.

IMPORTANCE OF POLAR MEDICINE

Increase in Tourism and Expeditions

The combined geographic polar regions cover about one sixth of the earth's surface, and their territory in the imagination has been enlarged by several remarkable expeditions and recent popular magazine articles and books on polar tourism. In the past decade, there has been a veritable explosion of tourist activities in both the Arctic and the Antarctic. Despite or because of the forbidding environment, tourism of various sorts is increasing.[91,131,204] The Arctic is a trendy destination. An estimated 1 million tourists visit the Arctic each year.[164,205] In 1980, it was estimated that a total of 31,000 paying tourists, adventurers, or guests of national scientific expeditions had ever visited Antarctica, but its increasing lure has led to explosive growth. The International Antarctic Association of Tour Operators (IAATO) reports a nearly 53% increase in visitors from the 1999–2000 season to the 2003–2004 season. In the latter season, there were 27,537 tourist visits alone, and more than 33,000 persons landed, including staff and crew. Although the bulk of tourists visit aboard ships, there is an increasing number of overland participants who engage in camping, kayaking, diving, skydiving, mountaineering, Arctic or Antarctic traverses, polar challenges, and adventure tourism.[91,131,205]

Concerns about the dramatic increase in tourism and its effect on the ecosystem have led to a movement to limit the total number of tourist visits per year. The World Wildlife Fund has proposed guidelines to developing ecofriendly tourism in the Arctic. The guidelines cover its impact on the wildlife as well as on local populations. In Antarctica, the IAATO has encouraged monitoring its members and has proposed voluntary adherence to limiting the number of passengers per vessel and the impact of visitors on the local areas.[91,131,205]

This increasing number of visitors and expeditioners has created interest in polar medicine, in particular as it relates to land-based and extreme expeditions. Wilderness medicine courses specific to polar medicine are now offered. Tourist vessels generally carry a physician and sometimes a nurse. Although facilities vary in level of care, all have preparations for the most frequently encountered medical problems. Most illnesses are upper respiratory infections, gastrointestinal (GI) problems, motion sickness, and mild bruises or strains. As might be expected given the age of many visitors, an occasional cardiac event or serious fracture occurs. The occasional expeditioner who needs rescue or assistance from a scientific station utilizes scarce resources.[48] An Australian tourist who suffered a massive myocardial infarction at the McMurdo facility had to be evacuated to New Zealand by the U.S. program. An Australian climber injured in an avalanche required the combined efforts of his support yacht, a tourist ship, and the Chilean national program.[131]

Perhaps the most notable tourist fatalities were the ill-fated sky diving venture at the South Pole, which resulted in the death of three of the four participants,[191] and the Air New Zealand flight that crashed into Mt. Erebus with the death of all 257 people aboard.[137]

Hazards are inherent in any expedition and may well be the attraction for some participants. To be allowed to participate in risky activities, some tourists may fail to declare serious medical problems that later precipitate emergency medical evacuation. Thus, one real concern is increased risk taking and the consequences thereof, or the dilemma between what is reasonable and what is foolhardy. From a medical perspective, making a risk assessment is multifactorial and differs for people, places, and goals.[87] Hazards of an evacuation need to be assessed for both the patient and the rescuers. Setting guidelines and anticipating needs in pre-event preparation are the key to a successful evacuation.[63,136,196] Because of the occasional tourist in need of expeditious medical evacuation, IAATO and other governmental groups have suggested requiring insurance to defray the costs associated with these evacuations.[9,107] There is also discussion of the ethical versus medicolegal implications of whether to render aid. The overriding issue should be care of the patient.[91] Polar Challenges and expeditions will be discussed later (see Medical Aspects of Arctic Racing).

Geopolitical Concerns

The interests of governments in polar regions have not lagged behind those of tourists. Political and territorial concerns have been important in both polar regions. With increasing exploration of mineral and oil reserves and fishing potential, migrating humans will continue to bring medical problems with them.[26] Although the Antarctic Treaty of 1959 prohibits territorial or commercial claims, seven nations have made sectoral claims on the continent.[206] Some of these claims overlap and in the future may lead to less than peaceful resolution.[199] Concern about the environmental impact of increasing human activity in polar regions seems to be tempering the pace of development, but as national resources diminish, the Antarctic may come under increasing pressure to open up to extraction similar to what has been the case in the Arctic.

Increase in Research Activities

Scientific research has been a part of polar exploration throughout this century. There are scientific and medical journals devoted to the polar regions, and thousands of articles are indexed each year in the *Antarctic Bibliography*. The *International Journal of Circumpolar Health* is a richer source for health issues in polar regions. A 45,000-square-foot science facility has been completed at McMurdo, and the replacement of the current South Pole Station and the construction of new and more powerful telescope facilities are expected to be completed by 2007. The Australian Stations have completed expansion and updating of research and medical facilities. British Antarctic Survey (BAS) has rebuilt Bonner Lab, which was destroyed in a winter fire in 2001.[30] BAS has completed an environmental report to replace Halley Station.[179] The French-Italian coalition has completed and occupied the first truly international station, Concordia, on the polar plateau at Dome C.[42,61]

Some observers have discerned a north–south split, perceiving research in Antarctica as having more of a political motivation, and that in the Arctic as being more practical. For both scientific and political reasons, an important part of the scien-

tific research in polar regions concerns environmental issues. The remoteness of these regions enhances their value as benchmarks for studies of pollution, and indeed several worrisome facts have been revealed about the contamination of formerly pristine wilderness, such as the widespread radioactive contamination,[130] the prolonged effective half-life of radioactive fallout deposits,[185,203] and the increased chemical contamination, particularly in animals high on the food chain, with persistent organic compounds (POCs), heavy metals, and other contaminants affecting the environment and the food supply.[156,203,204] Widening of the ozone hole carries implications of increased ultraviolet (UV) radiation.[37] As occupational and environmental health issues draw more attention, this aspect of polar medicine assumes greater importance.[65,66,156]

Interest in Analogies to Space Missions

Several articles and conferences have explored analogies between the isolated and confined environments of polar research stations and those of space stations. Data from polar (primarily Antarctic) stations tend to be more available and recent than from other space station analogs, such as nuclear submarines or the Biosphere.[6] International cooperation in polar health care and human research is coordinated by the Scientific Committee on Antarctic Research (SCAR). Polar studies may give planners more information relevant to these future long-distance human space missions. Studies in crew selection,[25] social compatibility, leader selection, health, neuroendocrine disruption, sleep, and potential psychosocial difficulties, as well as how to identify and effectively deal with abnormal behaviors, are ongoing.[52,58,59,61,115]

BRIEF HISTORY OF HUMAN HABITATION IN POLAR REGIONS

A perspective on the contrasts between Arctic and Antarctic medicine may be sharpened by a brief summary of human habitation in polar areas. Humans are known to have inhabited Arctic regions for at least 4500 years. Anthropologists have uncovered evidence for several waves of population migration from Siberia through northern Canada to Greenland. Each of these migrations was probably linked to climatic conditions, and each resulted in a distinct set of cultures. The general pattern was of a nomadic life with population densities of approximately 1 person per 400 km^2 (154.4 sq mi).[93,189] In more recent times, this long-established and remarkable adaptation to a hostile environment has been disturbed. The pace of cultural change increased dramatically during the second half of the 19th century, when the whaling industry moved into Hudson Bay, leading to sustained contact between Europeans and Inuit. In the early part of the 20th century, religious missions, trading company posts, government stations, church missions, and eventually medical clinics and schools began to encourage permanent settlements, roughly quadrupling the population density.[93] In some areas, such as Eurasia, large industrial and mining cities arose. This change in population distribution has at times led to conflict between the indigenous and European cultures, and it has had significant environmental and medical consequences.[204]

The known history of human exploration of Antarctica, unlike that of the Arctic, is quite recent. Recorded sightings of the continent date only to around 1800, and "winter-over" sojourns did not occur for another century. Waves of settlement can also be observed to have occurred in Antarctica: there was sealing in the early 19th century and whaling in the early 20th, and there has been scientific exploration since the mid-20th.[189] The heroic era of Antarctic exploration occupied the early years of the 20th century, with exploits such as the highly publicized race for the South Pole between Roald Amundsen and Robert Scott in 1911, the extraordinary survival of the crew of the 1914 Endurance expedition, led by Ernest Shackleton,[106] and the heroic survival of Douglas Mawson, an Australian geologist who wintered alone after his other expedition members perished.[120] Perhaps because of these and other dramatic events, and perhaps because of the absence of an indigenous population and a scarcity of easily exploited resources, human activities in the Antarctic have retained a somewhat more expeditionary flavor than in the Arctic. This has helped shape the contrasts in medical practice between northern and southern polar regions.

ARCTIC MEDICAL PROBLEMS

Effects of Cultural and Demographic Transition

The Arctic is not a homogeneous region. Although the population is small, a bit over 4 million, it is very diverse, with few features in common except the latitude of residence and hours of daylight. Medical problems among indigenous populations in the Arctic, approximately 30% of the total population, are characteristic of those of displaced aboriginal people elsewhere in the world. There is a wide range of interrelated factors, including demographics; the social, economic, and physical environments; personal health practices; and availability of good-quality and culturally appropriate health services.[24,94] Although increased contact with industrialized cultures has brought benefits, it has also brought many problems.[181] For many years, health care has lagged behind national norms, with infant mortality in the Canadian Arctic 2 to 3.5 times the national average.[182] Age-adjusted death rates are also telling, with 9.7 per 1000 indigenous people compared with 5.8 per 1000 for the Canadian national average. Other striking statistics reveal a suicide rate in Nunavik of six times that of the southern provinces of Canada,[197] and an unintentional accidental death rate that is 3 to 4 times higher than the national average. Tuberculosis has decreased but is still unacceptably prevalent at 47 versus 7.1 per 100,000 persons. Diabetes mellitus has increased to 5% of the population of 30- to 39-year-olds compared with 1% in the general Canadian population. In a population in which dental caries were previously almost unknown, the need for restorative dental work is up to 60%. Widely prevalent alcoholism and tobacco use greatly contribute to these problems.[22,82,156,182] As the Arctic environment and indigenous Arctic populations make the cultural and demographic transition to a Western industrialized way of life, the spectrum of medical problems has shifted. Changing social patterns have resulted in disturbing trends: increasing numbers of young adolescent mothers, rising prevalence of gonorrhea and syphilis among Greenlanders, and concerns about other transmissible diseases, such as viral hepatitis and acquired immunodeficiency syndrome (AIDS).[128,215] Change from the traditional diet and lifestyle has led to increased obesity, diabetes, cardiovascular disease, and mental health problems, including

depression, binge drinking, alcoholism, and a higher suicide rate.[24,182,192]

Environmental and Occupational Health Problems

Trans-boundary pollutants, primarily from Eurasia and the North American continent, have made a deep and lasting change.[22] The Arctic, once viewed as pristine, is now considered a pollutant sink.[156,182,185,204] The nuclear accident at Chernobyl in May 1986 led to cesium 134 and 137 levels in reindeer meat 50 to 100 times those considered safe, forcing destruction of the Sami reindeer herds. During that same time, lake fishing and berry picking were curtailed because of contamination.[78,204] Industrial emissions from neighboring regions, including an estimated 100 million tons of sulfur dioxide, have led to the phenomenon of the "Arctic haze," a gradual whitening of the historically deep blue Arctic sky.[78,156]

Environmental impacts have had direct health consequences.[22,82,156] During the last 500 years, industrial pollution has led to a sevenfold increase in lead levels in human tissues in the Arctic.[78] Another survey found blood mercury levels above the normative limit in over 10% of Sami reindeer herders in northern Finland. In North Greenland, a study revealed that in 84% of human mothers, blood mercury levels were above the World Health Organization (WHO) provisional limit.[79] Oceans, rivers, and lakes reveal contamination and concentration of heavy metals in fish.[204] Further contamination of the food chain with long-lasting POCs, including polychlorinated biphenyls (PCBs) and many organic pesticides, is increasingly identified.[65,156,204] The long-term consequences of these environmental pollutants on the health of the Arctic dwellers has only recently been studied.[65,66,156,209] Recent work suggests that the endocrine-disrupting effects of PCBs may have changed the normal ratio of male-to-female births from 1.05 to 1.02 in the past 30 years.[24] It has been documented that the concentrations of PCBs in breast milk are seven times higher in the Arctic than in Quebec.[182] As previously noted, fallout from radioactive testing has caused river pollution, and other environmental degradations have affected the food chain such that it is estimated that 750 fatal cases of cancer per year are the result.[202,204] Fear of the effect of pollutants has caused many to abandon or significantly limit their consumption of traditional foods. Unfortunately, this can have negative nutritional effects, such as increased obesity and increased cardiovascular disease, and in some instances relative malnutrition. In some areas, consumption of traditional marine foods has led to blood levels of mercury exceeding current recommendations. Local health departments have to weigh overall nutritional benefits of traditional land and marine food sources against the potential long-term risks of neuropsychological and other potential health problems, particularly in newborns and infants.[38,156,204]

Nontoxicologic factors also make important contributions to Arctic morbidity and mortality. Accidents are more prevalent in the Arctic. The drowning mortality rate for the circumpolar area is about 50 per 100,000 persons compared with 6 per 100,000 in Scotland.[192] It is striking that Alaska's occupational death rate is five times that of the U.S. national average of 7.6 deaths per 100,000 workers.[44,183] With establishment of the Alaska surveillance system in 1991, there has been a 50% reduction in work-related deaths, including a significant reduction in commercial fishing deaths and a very sharp decline in helicopter logging–related deaths.[43] These data reflect in part the inherently hazardous working conditions and occupations and possible demographic biases of a young population. Other factors, however, may play a role. Recently, aircraft safety has attracted increasing attention.[176] Because aircraft are a predominant mode of transportation in polar regions, it is perhaps not surprising that Alaska accounted for almost 39% of all air crashes in the United States, and 24% of the fatalities.[138] Nonscheduled or commuter plane (less than 30 seats) flights, commonly used in isolated areas, may pose a risk of a fatal crash that is six times that of scheduled airline flights, a figure not surprising given the often extremely challenging flying conditions.[138,176] Significantly, air crashes are now the number one cause of work-related fatality in Alaska and the United States.[138] This is largely a result of terrain, weather, and isolated landing sites with limited navigational aids and operator error.[138] Overall, the mortality rate for Alaska is 3,185 per 100,000 per year as compared with the overall U.S. rate of 831 per 100,000.[64]

Psychosocial Health Problems

With social disruption and increased environmental and occupational health risks, it comes as no surprise that psychological problems in the Arctic have achieved higher visibility in recent years. For a variety of reasons, stress seems to be higher in winter.[80] One survey of over 7000 adults living north of the Arctic Circle found a prevalence of midwinter mental distress of 14% in men and 19% in women.[80] Most other studies suggest that this figure may be low. One study among Inuit found that 22%, or one in five, experience depression during the winter months, and 7% appeared to have seasonal affective disorder (SAD).[77,182] Cultural stress, erosion of traditional lifestyles, and substance abuse are all contributory factors.[24,182] Studies investigating scientific staff at Arctic research stations are relatively uncommon, but they show patterns of sleep disturbances, depression, and alcohol use reminiscent of those in Antarctic stations. The double apparent risk factors of high latitude and displaced indigenous population have made alcohol abuse and concomitant violence a serious problem in the Arctic. In Greenland, one in three Inuits dies a violent death, and roughly 25,000 adults consume 28 million cans of beer a year, one of the highest per capita consumptions of alcohol in the world. Prevalence of "binge drinking" among Arctic peoples ranges from 12% to 39%, with male rates being greater than female rates. Fetal alcohol syndrome is 13% higher in Alaska than in the continental United States.[24,182] One study compared alcohol consumption by the Greenland Inuit to nonindigenous local inhabitants and found that alcohol consumption was less among the Inuit. However, other studies found the reverse.[182] Accidents are the leading cause of death in the Arctic, and one third of these are estimated to be alcohol related. In the Canadian Arctic, alcohol consumption is one and a half times the national average, and Inuit and Indians between 15 and 24 years of age have a suicide rate six times the national average. In some communities, for boys and men aged 15 to 29, suicides are the most common form of injury leading to death.[182,192,197]

Current and Future Trends

Fortunately, there are encouraging signs related to Arctic health care.[192] The incidence of low birthweight, 5.5% in the central Canadian Arctic, has decreased,[24] although it is still higher than that among non-Arctic dwellers.[24,182] Tuberculosis, which incapacitated up to 20% of the Canadian Inuit by 1950, has largely

been brought under control. Unfortunately, the age-adjusted incidence of diabetes mellitus is rising.[182]

An important development in recent years has been institution of trauma registries to track and target significant causes of morbidity and mortality.[138] Recent research emphasizes that injury prevention is not solely a function of safer design of equipment but is a complex interplay of environment, activity, and people, notably including personal risk-taking behavior.[78,138] If current trends toward greater economic independence and education of Arctic peoples continue, such potentially modifiable adverse health behaviors may recede in importance in coming years.[92,101,102]

ANTARCTIC MEDICAL PRACTICE

Medical practice, problems, and their study in the Antarctic are somewhat different from those in the Arctic. Extreme isolation makes Antarctic medicine unique. Because of the remoteness, it may not be possible to medically evacuate (medevac) a patient for weeks or even months. It might be easier to medevac from the international space station than from Halley station.[73] In general, the population is adult and is physically and mentally healthy, although in the summer, health waivers are given for some individuals with certain chronic diseases so that they can visit or work for a limited time. The winter population is in good health by requirement, and all are adults. There are two exceptions: Chile and Argentina have families living at their principal bases and have even had children born on them. All stations have some form of medical care, usually a station doctor. The more temporary summer camps have varied medical support, ranging from first-aid wilderness responders to paramedics and limited medical supplies. In general, the permanent year-round bases have a more sophisticated facility, with x-ray, laboratory, surgical, and dental provisions and a reasonably well stocked pharmacy for expected emergencies. There is one physician, sometimes more, and sometimes nurses and ancillary medical personnel.

Medical Stations in Antarctica

The medical facilities in the Antarctic polar regions can be conveniently divided into permanent and expeditionary facilities. As a result of community expectations and the long-term commitment to research in Antarctica, a notable shift, from an expeditionary to an operational attitude, has occurred in many of the Antarctic programs.[219]

What type of doctor is needed? To paraphrase Grant, the doctor cannot be too specialized in approach; a broad knowledge and wide range of practical skills are necessary to provide good Antarctic medical care. In small bases, there is no room for the luxury of anesthetists and surgeons, dermatologists and psychiatrists. These roles reside within the same person. However, modern communication modalities help the polar physician be less isolated from advice, counsel, and guidance. Yet, for a significant proportion of the Antarctic population, it is still impossible to evacuate patients. For these reasons the doctor must strive to maintain the highest practicing standards.[73]

The following is a description of the personnel and facilities in the U.S. Antarctic Program; other nations maintain similar facilities. McMurdo station (MCM) has the largest population: 250 in the winter and over 1200 in the summer. In the summer season, the medical component is staffed with two physicians, a physician's assistant or nurse practitioner, a National Guard flight surgeon, a dentist, an x-ray/laboratory technician, and a physical therapist. In the winter, it is staffed by one physician, a physician's assistant, and one other medical person, who can be cross trained. Additionally, MCM has a full fire department with paramedics who respond to emergencies on station. The South Pole population peaks at 250-plus in summer and has a nadir of 75 in the winter. There is one physician, a physician's assistant or nurse practitioner, and a volunteer trauma team trained on station. Palmer station is the smallest, with a population of 45 in the summer and a low of 10 to 20 in winter. There is one physician, and volunteers are trained on site to provide assistance. The Australian stations have one physician. Before deployment, there is a 2-week training course for two of the crew in operating-theater nursing skills, and for two persons in anesthesia assisting. The pre-deployment training has proved very valuable, as was clearly demonstrated in the case of a scientist who fell into a crevasse and suffered serious injuries, including internal bleeding, that required on-site surgery (Fig. 9-1).[158]

Figure 9-1. Trauma and rescue teams are frequently composed of volunteers and at least one experienced, trained individual. This rescuer is practicing for possible extrication of a victim from a crevasse. (Photo courtesy Betty Carlisle.)

All permanent U.S. medical facilities have digital x-ray and ultrasound capabilities, laboratory equipment for blood chemistries and cardiac enzymes, and the capability to perform immunologic testing for beta-hemolytic *Streptococcus*, infectious mononucleosis, and influenza A. Resuscitative equipment, including a Life Pak 12, portable ventilator, and crash cart is similar to that found in most emergency departments. The pharmacies are well stocked and cover a broad range of potential medical problems. There is no blood storage, but in the event of significant bleeding, a walk-in donor system is activated.

Figure 9-2. Historic midwinter rescue at the South Pole. Twin Otter flown by pilots from Canadian Ken Borek Service.

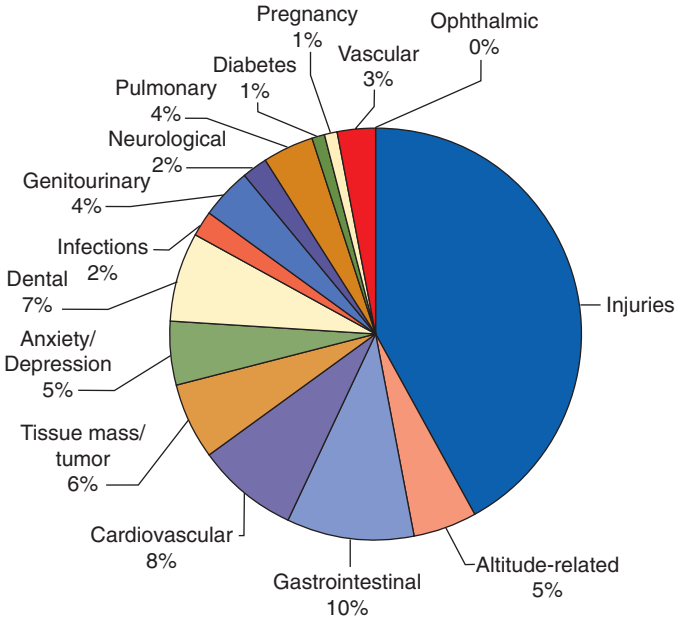

Figure 9-3. Evacuations for medical reasons, with percentages of 5-year totals (1998-2003). (From Mahar H [Safety and Health Officer, OPP/NSF]: Medical clearance criteria: Revalidation study. Personal communication, 2003.)

All winter-over personnel are screened for hepatitis B, C, and human immunodeficiency virus (HIV) and have been blood typed as well. After an outbreak of acute cholelithiasis in the winter, South Pole winter-over personnel are now screened with ultrasound for gallbladder disease. The Australian physician undergoes prophylactic appendectomy. Otherwise, the experience has been that early antibiotic therapy can abort an acute appendicitis attack, allowing the appendix to be removed electively at a later time.

The South Pole does not have 24-hour satellite coverage, but year-round stations have facilities for telemedicine, including live camera, still photos, fax, and digital x-ray films for transmission and consultation via satellite and the Internet. Telemedicine, used for many years in Antarctica by multiple programs,[73] came of age at the South Pole in 2002 with a midwinter patellar tendon rupture repair. The tendon was surgically reattached by the base physician under the direction of consultants at major medical centers via live video and voice connection. In addition, telemedicine has been used for diagnosis and consultation in the treatment of a wide variety of ailments, including acute cholecystitis, ophthalmologic lesions, pancreatitis, pericarditis, fracture treatment, psychological consults, radiologic reports, and many other situations. Telemedicine has improved the quality and availability of sophisticated care in these and other remote, hostile environments.

Medevacs are frequently dangerous and costly. Summer is the only season during which some stations can be reached for an evacuation. Winter evacuations are infrequent, very dangerous, and very costly. Logistically, it may be 2 or more weeks before a rescue can take place, and for some stations it may not even be possible. A historic medical evacuation from the South Pole was undertaken in the winter of 2001 with the first successful landing of a twin Otter aircraft in full darkness at a temperature of –92°F (–68.9°C) (Fig. 9-2). This event, while fraught with danger, has opened new possibilities and in fact has been repeated.[40] Certainly, though, prevention through careful screening of candidates and increased use of telemedicine as a way to avoid medevacs still remains the ultimate goal.

To this effect, all participants must pass a physical qualification test. Medical clearance criteria are used as a screening tool. To establish appropriate guidelines, evidence-based criteria from medical literature, historical data of the Antarctic Program, and the guidelines of other programs that are responsible for activities in remote areas, such as NASA, the Peace Corps, and the Navy, were used. However, up to 30% of *all* medical evacuations (both emergent and urgent) have been for trauma—that is, they were not related to the evaluation criteria. Of the emergent and urgent evacuations, 19% were for GI and 16% for cardiovascular reasons (Fig. 9-3). If considering only truly emergent medical evacuations, then 28% were for cardiovascular and another 28% were for GI reasons (Table 9-1).

Somatic Health Problems

Many of the primarily somatic health problems in Antarctica have to do with cold, altitude, and trauma and are thoroughly covered in the chapters on those topics. In a midwinter setting where a tossed mug of coffee freezes before the liquid hits the ground, the dangers of frostbite are clear. Anyone gasping for breath after climbing the six flights of stairs with a total of 92 steps from the tunnel to the living area of the new station at the South Pole, or who has lost 5 kg (11 lb) in the first 2 weeks merely from resting tachycardia and tachypnea, can appreciate the physiologic stresses of rapid ascent to an equivalent of 3200 m (10,499 feet). Likewise, the dangers of UV exposure at altitude with an ozone hole and snow surface reflectance of 80% to 90% can become painfully evident to the unwary visitor.[41,180] A few points, however, deserve further mention.

Cold-Related Problems

Cold is a dominant factor in polar medicine. It can be a source of humor, such as the infamous "300 Degree Club" at the South

TABLE 9-1. Emergent Evacuations

REASON FOR EVACUATION	98-99	99-00	00-01	01-02	02-03	5-YR TOTAL	PERCENT OF 5-YEAR TOTAL
Injury	1	1	—	—	1	3	17
Tissue mass/tumor	—	1	—	—	—	1	6
Cardiovascular	—	1	—	1	3	5	28
Genitourinary	—	—	1	—	—	1	6
Gastrointestinal	1	—	—	3	1	5	28
Altitude related	—	—	—	—	1	1	6
Pulmonary	—	—	—	—	1	1	6
Vascular	1	—	—	—	—	1	6
TOTALS	3	3	1	4	7	18	—

From Mahar H (Safety and Health Officer, OPP/NSF): Medical clearance criteria: Revalidation study. Personal communication, 2003.

Pole. Membership requires sprinting while somewhat less than completely clothed, from a 200°F (93.3°C) sauna to the Pole outside at an ambient temperature of –100°F (–73.3°C) or lower. Similarly, the quintessential polar first-aid story involves creative solutions to the problem of finding warm fluids,[3,53] but it would be reckless to forget the ever-present danger of such a hostile environment. Windchill commonly drops the effective temperature far below negative 73°C (–100°F). It has been estimated that under the most severe winter conditions, an inactive person in full polar clothing could undergo a life-threatening drop in core temperature in only 20 minutes.[68] Airplane refueling crews and others working with liquids at polar ambient temperatures are constantly reminded that even a small splash can mean instant frostbite.[53]

Rescue and treatment are complicated by the additional need for both victim and rescuer to avoid hypothermia and frostbite.[93,96] Disorientation and confusion from hypothermia, clumsiness from bulky clothing, and degraded performance characteristics of equipment and intravenous (IV) fluids can complicate otherwise straightforward procedures. A recent case report describes almost immediate freezing of fluid in IV lines and shattering of the plastic IV tubing despite vigorous attempts to warm them.[53,96] Simple devices commonly used in warmer settings, such as air splints and pneumatic antishock garments, would be similarly unusable. In more sophisticated equipment, batteries rapidly fail, unwinterized mechanical moving parts seize up, and metal objects become dangerous sources of frostbite.

Clothing, not normally considered a medical topic, assumes special importance in the polar environment (Fig. 9-4).[86,122,155] Although the clothing used for many polar research programs in the summer seems adequate, a strong argument can be made for specialized winter gear, possibly adapted from the space program, that would allow relatively delicate manipulation of scientific equipment while offering protection from the cold. In fact, at temperatures below those of most military specifications, material properties of equipment, including plastic electrical insulation, create unexpected hazards, such as splitting of insulation and other plastics, and extreme rigidity of cables and hoses so that they cannot be used. Fortunately, the importance of ergonomic issues in the design of machinery and clothing for polar conditions is receiving increased attention.[86,210] There is new research on use of personal heaters, which are particularly effective for extreme cold and for sedentary work in the cold.[171] Phase-Change Materials (PCM), now in their trial phases, can store latent heat and then release it during periods of low activity.[155] These newer materials will allow thinner gloves with high thermal protection to be worn for work in the cold that requires dexterity, and they will be particularly effective for persons whose efforts alternate between heavy work that can produce sweating and light work with associated cooling (Fig. 9-5).

Figure 9-4. Clothing is an important topic in polar medicine. A researcher prepares to leave the station for an extended time at –56.7°C (–70°F). The total weight of clothes is 11.5 kg (25 lb). Notice the multiple layers for each area of the body. (Photo courtesy Stephen Warren.)

Figure 9-5. Frostbite on thigh with eschar. Newer phase-change materials would have prevented this injury. This skier across Antarctica suffered frostbite when he removed one layer of insulating clothing to avoid sweating on a calm sunny day. When a wind came up, it caused frostbite through the wind-pants and light insulation. (Photo courtesy Borge Ousland.)

Nutrition Studies

Nutrition has occupied a key niche throughout the history of polar expeditions. Early expeditions in both polar regions provided several examples of nutritional illnesses, including scurvy and possible hypervitaminosis A and lead poisoning.[8,197] More sophisticated nutritional analyses have been possible during recent expeditions,[180,186] and advances continue to be made in development of lightweight but nutritionally dense rations.[117] When participants in endurance events are well trained, it is possible to compete without loss of lean body mass.[117] Although many prolonged unsupported expeditions include an element of malnutrition or even starvation, protein synthesis is still active.[186,187]

During a Canadian-Soviet transpolar ski trek, participants skiing some 20 km/day (12.4 mi/day) at a speed of about 3.5 km/hr (2.2 mi/hr) while carrying 37- to 45-kg packs (81.5 to 99 lb) showed increased strength, decreased body fat, and increased high-density lipoprotein cholesterol. However, a paradoxical drop in aerobic power was noticed, perhaps because of intensive pre-training and conditioning, as well as the increased efficiency of skiing as the trip progressed.[170] More recently in a nonsupported, two-man, 86-day trans-Greenland trip, well-trained and experienced skiers eating an adequate caloric diet had little if any significant impairment. Their average energy intake in rugged terrain was 28 to 34 MJ/day (6927 to 8121 cal/day) and in level terrain 14 to 16 MJ/day (3344 to 3822 cal/day).[69] Although applicability of these figures to more sedentary polar sojourners is probably limited, work is being done to alter the diet of the more sedentary expeditions to improve the lipid profile of the participants.[118] Better availability of common foods and fresh fruits and vegetables during the summer, and frozen foods in the winter at the permanent stations allow participants to choose a healthier diet. However, frequent fried and high-fat food intake leads to elevation of lipid profiles and the associated increase in cardiovascular risks. Some studies have demonstrated that it is possible to make simple and acceptable changes in food preparation that have a significant impact on lowering cardiovascular risk factors of winter-over personnel.[17,118]

An area that has not received wide attention involves the possibility that the prolonged low dose of sunlight may have long-term effects on bone metabolism. One study showed a decrease in the 25-hydroxy metabolite of vitamin D (25OHD) as UV radiation ceased. However, the active metabolite 1,25-dihydroxyvitamin D (1,25[OH]2D) did not show such variation.[157] A few studies have demonstrated significant decreases in serum vitamin D as measured by 1,25(OH)2D, and associated increases in serum osteocalcin during the dark period, possibly indicating bone remodeling.[223] Another study in which low doses of vitamin D were given showed increased bone mass in lumbar vertebrae and leg bones, but no bone mass increase in the pelvis or upper extremities. This was felt to be the result of vigorous activity over the winter. The study also showed a decrease in serum calcium and 25OHD levels during the winter.[141] Because many participants return for successive winters, further studies are in order to delineate possible long-term effects of bone mass after redeployment.

Infection and Epidemiology

With particular reference to the space station analogy, a number of studies have taken advantage of the physical isolation of Antarctic stations as a natural laboratory for infectious disease epidemiology.[115] Because microorganisms are thought not to survive the extreme cold long enough to be carried in by winds, midwinter respiratory tract infection outbreaks in the absence of outside contacts have suggested that such organisms could persist in clothing or other fomites.[153]

Despite a widely reported leukopenia, decrease in cell-mediated immunity, and decreases in salivary IgA and IgM during the isolation of over-wintering, it is not clear whether persons emerging from Antarctic stations are more susceptible to infection[67,130] or whether the changes in immunity are related to the stress associated with a winter-over.

Circadian Rhythms, Endocrine Studies, and Sleep Research

Pioneering studies of Natani and colleagues[134] and Shurley and coworkers[184] on sleep electroencephalograms at the old South Pole station demonstrated clear patterns of sleep disturbances and "free cycling" of the sleep–activity cycle. Some of these findings have been extended in more recent studies. For example, among four subjects at a small winter-over camp, summer sleep cycles synchronized within the group and with clock time. During 126 days of sunless winter, rhythms free cycled in all four people and then resynchronized with reappearance of the sun.[98] The degree of synchrony versus free cycling appears to depend on a number of factors, notably zeitgeber strength (an environmental agent or event that provides the cue for setting or resetting a biological clock—that is, activities, social contacts, and the most important zeitgeber in nature, light).[70]

A number of studies have examined the effect of prolonged polar residence on diurnal variations in the level of melatonin. Its important role in free radical scavenging and sleep regulation is beginning to be elucidated.[97] Melatonin has been studied as a chronobiotic and is used as a supplement to treat circadian rhythm disorders.[11,13] Of the various studies using melatonin to treat sleep disorders, including delayed sleep phase and for nightshift workers, most have only small numbers of participants, and although the initial studies are promising, more research needs to be done.[224] It appears that exogenous melatonin can be used effectively for most persons. The short-term

side effects (headache, nausea, and drowsiness) are few. There are no long-term studies.[225a] Melatonin in dosages of 1 to 5 mg given 1 to 2 hours before sleep time has been used successfully to reset the sleep–wake cycle for persons who tend to free cycle and for some shift workers as well.[224] Therapy with full-spectrum bright light (greater than 2500 lux) seems to facilitate daily resetting of the melatonin cycle.[13,127]

Other endocrine fluctuations also appear to be correlated with the length of the day.[201] Some endocrine studies have examined effects of prolonged residence in polar regions with a longer than diurnal time constant. Twenty-four-hour urinary excretion of catecholamines increases in the cold, but social stresses appear to overwhelm climatic determinants of catecholamine metabolism, correlating with increases in diastolic blood pressure and pulse during the year.[81] A more consistent pattern of elevated thyroid-stimulating hormone (TSH), decreased free thyroid hormones, and increased triiodothyronine (T_3) clearance after several months has been demonstrated by several groups.[76,167,168] It has been suggested that rapid clearance of T_3 is caused by the cold. A pattern of increased pituitary release of TSH in response to IV thyrotropin-releasing hormone and increased serum clearance of orally administered T_3 has been identified and dubbed the polar T_3 syndrome.[55,146,167] The full clinical significance of these findings remains unclear, but it appears that the T_3 may well be related to the winter-over syndrome (see later).[149,166] One study that used thyroxine supplementation noted improvement in mid-winter cognitive function, mood, and vigor.[168]

Environmental Health Issues

Because of Antarctica's isolation, it seems a particularly disturbing site for litter and pollution. As in the Arctic, events such as the early problems with a nuclear reactor at McMurdo (since removed) and the 1989 oil spill from the Bahia Paraiso near Palmer Station,[109] and studies by groups such as Greenpeace, have focused attention on environmental health risks in the Antarctic.[132,133,203] Environmental inspections at a broad range of Antarctic stations have raised concern about toxic effluents and heavy metal residues,[160] as well as about radioisotopes used for research. To address these issues, a number of countries have begun to implement more responsible waste-management and water-monitoring policies and are taking corrective actions. Most have active recycling programs in place, and many cleanups are in progress or completed. Currently, most stations, including all U.S. stations, discharge raw sewage. Proposals are in place to find a way to ameliorate the contamination from raw sewage that is discharged into the sea, and/or into large bubble holes in the ice for the inland stations.[88] The BAS Rothera Station installed a sewage treatment plant with significant improvement in the near-shore marine environment.[88,125]

Occupational Health and Injury Prevention

Rigorous studies of risk are important for injury prevention. Unfortunately, they are complex and often bedeviled by challenges, including controversial data, multiple variables, and biases. For example, for purposes of occupational epidemiology, it may ultimately prove useful to stratify the Antarctic sojourner population into groups such as sport expeditions, military, commercial, and scientific, but the health behaviors of these groups overlap, and few studies have gone beyond aggregate descriptive statistics. There is no current system for archiving these data, except by individual programs or organizations, so much of the data is not available for review. There are plans to address this issue at the Council of Managers of National Antarctic Programs (COMNAP) at the forthcoming planning meeting for the International Polar Year of 2007–2008.[21]

Even from these broad statistics, some figures emerge that may be useful cognitive anchors. There were 16 fatalities from 1947 to 1999 in the Australian Antarctic Program, but only one fatality in the past decade. Among 3500 scientists and support personnel (2000 person-years), there were four medical deaths, two myocardial infarctions, one case of appendicitis, one perforated gastric ulcer, and one cerebral hemorrhage. The remainder of the events were accidents involving falls, head injuries, hypothermia, drowning, crush injury, and burns.[112,114]

There have been 60 deaths in the U.S. Antarctic Program since 1946.[2,116] These can be ranked by cause: aviation, 61%; vehicles, 11%; ships, 7%; recreational, 7%; station/industrial, 5%; field activities, 5%; and other, 4%.[116,177,178] Although it is difficult to adhere to standard occupational safety practices when in a remote location,[177] new guidelines and serious efforts in some of the programs have led to improved worker and participant safety.

A review of data for the Australian National Antarctic Research Expedition (ANARE) 2002–2003 season listed the following reasons for consultation: medical (e.g., upper respiratory, GI), 39%; injuries (sprains, strains, dermatologic, lacerations, fractures), 27%; follow-up visits, 24%; and preventive (immunizations, midyear examinations, wellness programs), 10%.[20] For the 2004–2005 season, Antarctic New Zealand (ANZ) reported the following: musculoskeletal, 42%; medical (e.g., upper respiratory infections, GI), 22%; other injuries, 20%; dermatologic, 15%; and preventive, dental, and other, 8%.[154] Usually, most visits are related to injuries, and the next most frequent medical clinic visits are attributable to upper respiratory infections. Environmental problems include dry skin and fissures, particularly on fingertips as a result of cold and very dry conditions. Cyanoacrylate glue hastens healing of disabling fingertip and heel fissures.[19] Cold-induced urticaria, frostnip, and frostbite are frequently underreported except for moderate to severe cases. Small nasal bleeds occur frequently, but they rarely require treatment beyond instruction to use petrolatum or antibiotic/antiseptic ointment once or twice a day to protect the delicate nasal epithelium, which easily becomes dehydrated or can suffer frostnip. Although the bulk of care is limited to routine visits, the pathology encountered even among this carefully screened population has included myocardial infarctions, massive GI bleeds, unreported Crohn's disease, bowel obstructions secondary to cancer, pulmonary emboli, massive head injuries, fractures, crush injuries, newly diagnosed schizophrenia, and cancer.

Fire Safety

In light of the extreme cold, it is somewhat counterintuitive that fire is a major concern and principal danger at all Antarctic stations. This surprising assessment rests on a number of factors. Cold temperatures severely limit the utility of water for fire suppression. Firefighting equipment may become inoperative in extreme cold, and frequent high winds can fan fires. The extremely low absolute humidity in many polar regions results in an even lower relative humidity when outside air is warmed in station dwellings. This in turn leads to increased static electricity and dryness of combustibles, already at risk from fre-

quent use of space heaters. Finally, alternative food, shelter, and fuel are limited.

Elaborate pre-deployment training, frequent on-site drills, and keen awareness of potential fire hazards have correlated with a satisfactory fire safety record to date, but this good fortune cannot be taken for granted. At least six major fires occurred in Antarctica in the past 21 years.[178] One of the most life threatening was the explosion and fire at Vostok in 1982, leaving the station basically without power or adequate heat. Fortunately, only one person died, but there were several injuries, and the winter crew had to endure 8 months without a power plant to supply heat.[161] In 2001, the BAS Rothera Station lost its new Bonner Lab building when an electrical short started a fire. No one was injured, but with 50- to 70-knot winds, little could be done to save the station.[31] Fire-suppression systems are built into the newer buildings, but fire is an ever-present threat.

Tourist Safety

Polar regions are imbued with a sense of the exotic, conjuring up "images of hardship, personal valor, danger, adventure, and of course, the hero."[178] As transportation increasingly opens up previously inaccessible areas, this image attracts growing tourist traffic and, with it, hot debate from both legal and environmental viewpoints.[9] The essential medicolegal issue is the extent of governmental organizations' responsibility for medical care of participants in nongovernmental activities, whether scientific, political, or commercial. Although this discussion started much earlier, it is still ongoing.[9,131,178] When a tourist group requires aid beyond its own capabilities, whose responsibility is it? To what extent should a research station with limited medical supplies be required to divert some of those supplies to an individual who presumably bears responsibility for planning his or her own medical coverage? Although the ethical course of action seems clear in the preceding scenarios, there is still uncertainty in our litigious society regarding the relative strengths of the Good Samaritan principle and the doctrine of assumed risk.[84] The Antarctic Treaty XII Consultative Meeting provided attendees with a list of documents that were recommended before their trip to Antarctica, including travel insurance that would cover rescue in Antarctica.[9,131] These are voluntary guidelines.

Air Safety

As in the Arctic, reliance on aircraft for transportation has highlighted the critical importance of air safety. The worst Antarctic air accident was the crash of an Air New Zealand tourist "flightseeing" DC-10 on Mt. Erebus in November 1979.[137] This accident killed all 257 people aboard and temporarily ended such flights, although IAATO records indicate that overflights are now happening with new guidelines in place.[91] There have been 35 air-related fatalities in the U.S. Antarctic Program since 1946 from crashes related to either fixed-wing or rotor aircraft.[116,178] A ski-equipped LC-130 Hercules crashed at the remote D-59 camp in 1971 (Fig. 9-6). Fortunately, none of the 10 crew members aboard was injured, but during a salvage operation in 1987, another LC-130 bringing supplies to D-59 crashed nearby, killing two and injuring nine. More recently, a helicopter lost control while working in the dry valleys; both the pilot and passenger were seriously injured. After a weather delay, they were extricated and evacuated to New Zealand for definitive care.[35,135] The review of literature for air crashes in Antarctica is exhaustive. With better weather prediction and increased safety standards, the accident profile is improving.

Figure 9-6. LC-130 Hercules partially stuck in hidden crevasse, demonstrating the inherent hazards of working in Antarctica. (Photo courtesy National Science Foundation photo archive.)

Psychosocial Health Problems

For many, polar living arrangements and psychosocial issues may be more important than the strictly medical or environmental factors. Psychiatric problems are mentioned even in reports of early expeditions,[171] and almost any participant in expeditions will recognize Thor Heyerdahl's observation: "The most insidious danger on any expedition where men have to rub shoulders for weeks is a mental sickness that might be called "expedition fever"—a psychological condition that makes even the most peaceful person irritable, angry, furious, absolutely desperate because his perceptive capacity gradually shrinks until he sees only his companions' faults while their good qualities are no longer recorded by his grey matter."[225]

Most psychological problems, primarily sleep disturbances, are minor and temporary.[184] Many disturbances are more related to the social environment than to the physical environment.[138,142,151,172] Appropriate lighting or changes in photo periodicity (the duration of daylight and darkness in a 24-hour period) help improve mood and sleep.[12,13,147,211] In addition to appropriate exercise facilities, opportunities for quality mental stimulation and frequent contact with friends and family via the Internet and telephone can help maintain stability. Except for insomnia, which is perhaps endemic, the incidence of psychiatric problems is lower than expected in the overall population.

Alcohol Abuse

Adjustment to psychosocial stresses in polar communities depends on a complex array of sociocultural factors. One

coping mechanism is alcohol use. As in many isolated communities at high latitudes, alcohol use can disrupt and lubricate social interactions. At one station, annual per capita absolute alcohol consumption was calculated at 16.9 L (17.9 qt).[7] Alcohol use at the station was studied, and blood alcohol levels during social functions ranged from 0 to 0.22 mg/dL. On the nights of social events, 44% to 61% of members were intoxicated.[7] At another, a summer support-staff member had to be terminated because of alcohol abuse, and two winter-over staff had to be prohibited from purchasing alcohol from the station store; one of these actually required disulfiram to ensure abstinence. In some instances, interventions with group support are found to be very effective. At many Antarctic stations, alcohol is easily available and may be subsidized. In some stations, rations can be excessive or nonexistent. Although relatively few serious incidents involving intoxicated staff have been reported, the potential for serious accidents remains real, particularly during winter when medical evacuation flights are impossible or fraught with extreme danger. Recommendations have called for routine monitoring of alcohol levels of anyone involved in an accident[178] or even prohibition of alcohol at Antarctic stations. However, as with prohibition in other countries, this might create more problems than it might solve. Judicious use of monitoring can help identify persons who may need intervention before an accident happens.

Psychoneuroimmunology

Psychological stress at research stations may arise from several sources. One is the environment itself; environmental severity has been found to be an independent predictor of hostility and anxiety after wintering over.[145] Perhaps more important is the perception of the environment. Newcomers to the South Pole station are usually observed wearing a full 12-kg (26.5-lb) polar outfit at –20° C (–4° F) for several weeks after arrival. By midwinter, the same individuals think little of walking short distances in only workout shorts and T-shirt at –60° (–76° F).

Stress can also result from disjunction between a person's original motivation for joining the program and the realities of life in the station—that is, the sociocultural environment. This may be the greatest determinant of difficulties.[145] Studies suggest that people who go to Antarctic stations to seek thrills or to challenge themselves often have more difficulty sustaining their motivation and performance than those who go to accomplish a scientific mission or even those primarily motivated to earn money. In general, older individuals who are somewhat introverted are able to set work goals and work toward them, and persons who have broadly defined hobby interests seem to cope better with the social isolation and dynamics of wintering over.[57] One study found that members with low social coherence reported significantly more depression, anxiety, and anger than those individuals belonging to expeditions with high social cohesion.[142,150]

Small-Group Dynamics

In addition to external climate and internal motivational factors, stress can arise from interpersonal interactions. Although there are many exceptions, participants in the Antarctic program often speak of a tendency for personnel to form cliques or microcultures. People may cluster according to ordinary personal chemistry, along lines of OAE (Old Antarctic Explorer) versus novice, winter-over versus summer status, or according to scientific, civilian, or military affiliation. With time, such social clusters can be a source of support or of friction. In an attempt to unify the core group, winter-overs participate in a mini Outward Bound type of group bonding experience before deployment.

Social isolation is of course an important factor in polar communities, although this isolation has been lessened by modern communications and transportation. A number of studies have drawn parallels between group processes in polar stations and those in other isolated and confined environments, such as submarines or in space. As space travel becomes a reality, studies on group selection, leadership style, and interaction between the isolated group and headquarters take on urgency.[24,52,108,115,143,150] Collaborative efforts are being made between many stations and space agencies, in particular the European Space Agency (ESA) and the NASA program.

Winter-Over Syndrome

Stressors such as those mentioned previously can result in what has been termed the winter-over syndrome. The historically oft-mentioned "Big Eye" or "20-foot stare in a 10-foot room" seems to be less common now, perhaps because the stations are increasingly comfortable and have stimulating environments. But the constellation of depression, hostility, sleep disturbances, and impaired cognition still seems to be both common and underreported.[148,149,159,168,214] The winter-over syndrome is not a static condition but a time- and individual-dependent process. At the South Pole research station, for example, winter-over staff characteristically report a recognizable pattern of fluctuating activity and mood. There is often a mixture of relief, pride, and fearful anticipation as the last LC-130 flight departs in mid-February, followed by a period of frenetic activity to beat inventory deadlines, file resupply orders, launch projects, and prepare the station for winter. As work and recreation routines develop, mood generally drops with the setting sun. A rise in mood generally occurs toward sunrise but is followed with anticipation, increase in anxiety, and stress as the time of the arrival of the first return flight draws near. Studies at the Australian stations of Mawson and Macquarie Island suggest that the influx of new staff at station opening may actually be associated with increased depression.[7]

Seasonal Affective Disorder

A probable contributor to and confounder of the winter-over syndrome is the apparently fairly consistent exacerbation of stress and depression under conditions of winter or night, leading to seasonal affective disorder (SAD). Subsyndromal SAD (S-SAD) is perhaps a better definition of what is experienced by many who winter over in the dark latitudes.[146] Further studies indicate that S-SAD may be part of the T_3 syndrome.[149] Correlations with melatonin levels have been shown to be associated with the frequent sleep disturbances at higher latitudes, both with advanced or delayed phase, and with increased latency.[12,14,211] In some studies, melatonin's chronobiotic properties are used to resynchronize the individual's sleep.[12,14] Other studies have demonstrated the effectiveness of light therapy in helping the body resynchronize.[211] SAD may be no more prevalent in Antarctica than at lower latitudes; some of the depressive symptoms may well be the result of social isolation.[152]

Beneficial Effects of Isolation

An interesting point is that isolation may have positive effects as well as negative ones.[188] Isolation is not synonymous with

loneliness and depression, as Amundsen reported from his sojourn at Framheim. In fact, a subset of "professional isolates" may actually prefer polar stations to "normal" society.[193] In some cases, reentry and adaptation to the rest of the world can take up to 6 months. The positive effect of isolation may be reflected in a study of somatic complaints, published in 1992, that compared Navy volunteers who qualified for winter duty in Antarctica and wintered over, with those who were physically qualified but were assigned elsewhere because of the limited number of winter assignments; those who spent the winter in Antarctica had fewer illness and hospitalizations after the winter-over period.[226] These volunteers were followed an average of 5.4 years after the winter period.[142] It was also noted that the complex psychophysiologic symptoms experienced during the winter-over period were probably adaptive coping mechanisms. Coping led to a process of negotiation leading to compromise, which frequently led to finding new strategies or resources that could be used for subsequent stressful experiences.[142]

Screening and Selection

Based on the preceding findings, several attempts have been made to refine the selection and screening process for successful polar and space sojourners.[25,115,175] It is not surprising that previous successful polar experience seems to be among the best predictors of subsequent high performance. In general, biographic data, peer ratings, psychometric testing, and interviews all seem helpful in selection, although each has weaknesses and inconsistencies. Recent studies have suggested that team climate significantly related to the perception of good leadership. More research needs to be done to explore the characteristics and behaviors that constitute effective leadership, in order to guide selection of crew and leader.[25,115,175] Early studies led to the tripartite "ability, stability, compatibility" criteria for successful participation in the Antarctic programs.[74] This simple but useful scheme recognizes that technical skill, emotional equilibrium, and interpersonal skills all play important roles in an individual's performance and internal satisfaction. It would be desirable to improve measurement and predictive usefulness of these factors; such studies remain at the active frontier of Antarctic research.

OVERVIEW AND FUTURE DEVELOPMENTS

A point made early in this chapter was the challenge inherent in meaningfully synthesizing Arctic and Antarctic medicine. Reflection on some of the issues raised previously suggests several common areas of linkage and directions for future work.

Issues of Methodology and Medical Epistemology

The state of knowledge about the human factors involved in living in the Antarctic is still rudimentary, although renewed interest by NASA and the ESA in Antarctica as a space analog has led to increased research funding and should lead to better information. Studies in polar settings are often handicapped by extremely difficult research conditions and may never achieve the statistical power and validity expected of counterparts in more forgiving climes. Commonly encountered methodologic problems include, most notably, small sample sizes, unknown effect sizes, measurement of proxy variables, and multiple confounders.

Fourth World Medical Decision Making

Polar medical lore is replete with stories like that of the physician of the 6th Soviet Antarctic Expedition who performed an appendectomy on himself.[222] Although the basic principles of emergency medicine, epidemiology, occupational health, psychology, and other disciplines still apply in polar settings, the spectrum of health problems and some important aspects of their management are undoubtedly skewed. However, with newer and improved technical communication modalities and transportation, delivery of advanced care in the polar latitudes has improved, and many lessons learned in Antarctica are applicable to space exploration and settlement.

Fourth World medicine requires distillation of medical practice into a compact yet comprehensive package that is sufficiently robust to travel well. Novel constraints and the inherently unpredictable nature of a hostile environment further stretch the usual, already-tentative rules and thresholds of medical decision making. Perhaps to a greater extent than in most settings, prevention and preparation are paramount in polar medicine. As might be imagined, the risks from even minor mishaps are exacerbated by cold and its effects on both humans and equipment, by altitude and impaired tissue oxygenation and wound healing, and by restricted dexterity and vision from bulky clothing. Isolation from medical facilities exerts a multiplier effect on these risks. Victim extrication and resuscitation that would be routine in most urban or suburban settings presents an overwhelming challenge in polar settings.[158]

With lives at stake, arguments have been made for planning for worst-case scenarios rather than only for likely situations.[177,178] On the other hand, a realistic balance point on the cost-versus-utility curve, based on estimated risks, must be set for each situation. Given existing fiscal and logistic constraints, preventive measures should take priority over higher-cost options.[20]

A recurrent medical issue in polar stations involves contingency planning and the optimal level of inventory. This question applies equally to drugs, equipment, training, and personnel, and it is faced immediately by any incoming physician. Given finite (even scarce) resources, space, and resupply capability, does the physician have enough X at the station, enough training to handle Y, enough help available to take care of Z? "Just in time" principles of inventory management are not likely to be appropriate.[20,176,178]

The combined improvements in communications systems, the Internet, and the powerful tool of telemedicine with its many modalities have transformed what was once an extremely isolated practice of medicine. The advantages of rapid access to organized databases and remote consultations for radiology and other specialties via video are a reality. Telemedicine is in place and functioning in many northern Arctic regions. The Australian, British, Italian, and United States Antarctic Programs and many others have been using it successfully for a number of years.[73,87,109,165] Physiologic, psychological, and occupational health data can be monitored or retrieved, ultimately linking geography and information flow, and allowing nearly real-time consultation and guidance from multiple specialists. These promising tools are real solutions to the problems of isolation faced by earlier physicians.

MEDICAL ASPECTS OF ARCTIC RACING

In the spring of 2003, the first-ever Polar Challenge race, from Resolute Bay in northern Canada to the Magnetic North Pole (1996 position), took place, with Dr. Ian Davis as Senior Medical Officer. The Polar Challenge is run annually as an ultra-extreme endurance race covering 580 km (360 miles) in temperatures down to –48° C (–80° C with windchill) (54° F, –112° F with windchill). Teams of two or three persons race the distance on foot and skis, pulling pulks, which are small sleds weighing up to 80 kg attached to the skier. Teams pass through two manned checkpoints during the race.

The medical needs of the competitors and support staff are unique in such a challenging and harsh environment. Provision of medical care has been perfected and refined over the preceding 3 years.

The Medical Team

Before the start of the race, and after consultation with local hospitals, an emergency evacuation plan is written for extrication of racers to the most appropriate trauma/emergency medical facility, depending on the type of injury. At each checkpoint, a doctor is available to provide treatment and consultation to the racers. A more experienced polar doctor forms part of the mobile medical team (contacted via satellite pager), which also includes a navigator and a local Inuit guide/mechanic. This team is placed strategically on the ice for the duration of the race. It responds to any medical event either by snowmobile or on foot, with full air evacuation support on standby at all times. The race doctors carry an extensive range of equipment and drugs (Box 9-1) designed to handle any eventuality in this environment. This kit is refined annually after each post-race injury debriefing.

Box 9-1. Doctor's Medical Bag

DRUGS

Multiples of the drugs found in the competitor's medical bags, plus the following doctor-use-only drugs:

- Ketamine, 100 mg/mL, 10-mL vials
- Morphine, 10 mg/mL, 1-mL vials
- Midazolam, 5 mg/mL, 2-mL vials
- Flumazenil, 100 µg/mL, 5-mL vials
- Adrenaline, 1 in 1000 (1 mg/mL), 1-mL vials
- Hydrocortisone, 100 mg/mL, 1-mL vials
- Prednisolone, 5-mg tablets
- Salbutamol, 100 µg/metered inhalation
- Metoclopramide, 10 mg/mL, 1-mL vials
- Lidocaine 1%, 10 mg/mL, 10-mL vials
- Oxybuprocaine hydrochloride eye drops, 0.4%
- Norethisterone, 5-mg tablets
- Dexamethasone drops for ears

MEDICAL EQUIPMENT

- Trauma kit, including chest tube
- Suture kit
- Urethral catheter kit
- Airway kit, including bag valve mask

Pre-Race Selection and Training

Competitors for the race range in ability from experienced and very well trained arctic travelers to complete novices. Ages so far have ranged from 20 to 65 years with a mix of two-thirds men and one-third women, totaling 80 racers in 3 years. All are offered the same training program (Box 9-2). If required, experts are available for extra tutoring specifically tailored to individual needs.

When they join the race, competitors are sent a standard medical questionnaire that is used as a tool for excluding medically unsuitable applicants. Conditions such as cardiac disease and insulin-dependent diabetes would prove very difficult to treat in such a remote and hostile environment. The practicalities of racing with such conditions are discussed in detail with applicants and then a decision is made as to whether they will take part in the event. Through a process of reflective practice, the training program has been refined, with the result that the current program achieved a 98% competitor completion rate.

Team Medical Training

Each team is required to carry a pre-packed medical bag. The bags and their contents are identical for all teams. Constructed

Box 9-2. Training Program for Polar Challenge Competitors

In the 6 months before the race, training is carried out in three locations: the United Kingdom (UK), Austria, and northern Canada.

UK TRAINING

Fitness training
Navigation
Firearms
Polar bear training
Medical training for cold-weather injury avoidance and treatment

AUSTRIA TRAINING

Polar clothing and equipment
Pulk packing (small sleds for gear, attached to skier by a flexible harness)
Arctic food
Stove use
GPS navigation
Medical training for cold-weather injuries
Firearms
Ski technique and tent craft
Cold-water immersion exercise

NORTHERN CANADA TRAINING

Cold-weather injuries
Communications
Safety
Evacuation
Firearms
Polar bears
Navigation
Mini 3-day expedition (fully guided) to start line of race

Box 9-3. Polar Challenge Medical Advice Sheet 2005

SUSPECTED FRACTURES OR SERIOUS TRAUMA OR ILLNESS—TENT UP AND CALL FOR HELP

PAINKILLERS

- Begin with regular simple pain relief. Use acetaminophen (paracetamol), 500 mg, 1 to 2 tabs, maximum of 8 per day.
- If needed, add in acetaminophen with codeine (paracetamol plus codeine), maximum of 4 per day.
- For increased pain relief, take an anti-inflammatory (do not take if have stomach ulcers or asthma). Try either (but not together in the same 24 hours) diclofenac (50-mg tablets, 3 per day), or piroxicam (1 per day). Take with food.
- For headache, try the above. If this does not work, take 1 aspirin plus one metoclopramide tablet, repeated twice a day. If headache is increasingly severe, contact the physician.

ANTIBIOTICS

- If worried about infection, call physician, giving the site of the infection and symptoms. We will then advise.
- If your communications break down, take Augmentin (625 mg) for any suspected infection. Take 1 tablet, 3 times a day for 1 week. Do not take Augmentin if allergic to penicillin. If you have an allergy to penicillin, take erythromycin (500 mg, 1 tablet 3 times a day).

EYES

- Any problems with eyes, please try contacting physician first. Otherwise, chloramphenicol eye drops is a good general antibiotic for eyes. If your eyes generally become very sore/red and watery, you may have snow blindness so call for advice.

GUT

- Diarrhea—Take Imodium (1 tablet after each explosion!!).
- Heartburn—Try Gaviscon. If this doesn't work, take 1 lansoprazole (Zoton) fast-tab per day.
- Constipation—Drink lots and take senna, 2 tablets at night.
- Abdominal pain—Contact physician.

ALLERGY

- Piriton (chlorpheniramine maleate 4 mg) 1 tablet 3 times/day

NAUSEA AND VOMITING

- Metoclopramide tablets (1 tablet 3 times/day)

SKIN

- Any general rashes (groin, etc.)—Apply clobetasone 0.05% cream (Trimovate, green tube) twice a day.
- Injuries to finger tips and burns—Use silver sulfadiazine (Flamazine) cream twice a day.

COLD SORES

- Acyclovir cream, apply 5 times a day over affected area.

BLISTERS

- Try using DuoDerm (hydrocolloid dressing), which can be cut to size. Leave at least a 2-cm margin of the dressing around the blister. Leave on for 3 days before changing it. It is normal while the DuoDerm is on to get a blob of fluid building up over the affected area.

GENERAL WOUND CARE

- Lacerations—Depending on size, use either MedLogic skin protectant (similar to Steri-Strips) with glue, or just glue on its own (octyl cyanoacrylate [Dermabond, Liquibond]).
- Larger wounds—Apply direct pressure with a dressing and call for advice.

of lightweight yet tough material, they have two outer pockets and internal color-coded pouches, whose contents are listed in Box 9-3. Each bag contains a color-coded contents list with instructions for use. This makes giving advice over the satellite phone much easier, reducing the likelihood of error. Each race doctor knows which drugs are in which color-coded pouch and so can instruct racers accordingly. The external pouches contain emergency dressings and wound and blister treatments.

Prior to the race, competitors are given both group and individual instruction on the contents of the medical pack and their use. They receive lectures on cold-weather injuries and how to avoid them, as well as instructions on what to do in a crisis. The medical packs can be individualized if racers have specific medical needs.

Arctic Racing Injuries Encountered to Date

Frostnip in a variety of degrees has been seen in most competitors during the race, regardless of their previous cold-weather experience. It has affected mainly the extremities, such as the nose, ears, and fingertips (Fig. 9-7). All cases were treated by rewarming and diligent future protection. The number of frostnip injuries decreased after a team buddy system was instituted, which led to rapid prediction of impending injury and timely avoidance.

Figure 9-7. Frostnip ear. (Photo courtesy Ian Davis.)

Figure 9-8. Frostbitten fingers recovering several weeks after the injury. (Photo courtesy Ian Davis.)

A

B

Figure 9-9. **A,** Frostbitten fingers. **B,** After recovery from frostbite. (Photos courtesy Ian Davis.)

Frostbite was seen in six competitors, five of whom had finger injuries (Fig. 9-8). All the fingers made a complete recovery largely as a result of early detection and rapid evacuation of affected competitors from the race to a rewarming and treatment facility.

One case of fingertip frostbite was caused by a fuel spill onto the fingertips, which caused rapid freezing and subsequent injury. This competitor was a highly experienced Arctic traveler and was therefore allowed to continue, subsequently making a full recovery during the race (Fig. 9-9).

The final case of frostbite was more unusual, affecting a competitor's penis (Fig. 9-10). After urinating, he forgot to close the zipper correctly on his wind suit. After walking into a headwind with a windchill factor of –80° C (–112° F) for an hour, he discovered his foreskin to be frozen. Rapid evacuation prevented further damage and, after a circumcision on his return to the United Kingdom, he made a complete recovery both in sensation and function. During the event, this competitor could easily have gone into urinary retention and would have been catheterized accordingly, making it essential to carry such equipment in the doctor's medical kit.

Musculoskeletal injuries, most commonly of the lower back, shoulders, knees, and wrists (related to cross-country skiing and pulk pulling), were seen in most of the competitors. They were all treated with appropriate analgesia and advice from both the mobile and static medical teams. This enabled most competitors to finish, with only 5% of the total having to withdraw for this reason.

The most serious musculoskeletal injury was a dislocated shoulder, which was relocated by a teammate using a slow Kocher's method. He was talked through this maneuver by the race doctor over the satellite phone. This competitor went on to finish the race.

Figure 9-10. Frostbitten foreskin. (Photo courtesy Ian Davis.)

A

B

Figure 9-11. **A,** Snowblindness. **B,** Eye patches. (Photo courtesy Ian Davis.)

Snowblindness was seen in 4% of racers (Fig. 9-11A). Diagnosis in the field was achieved with the aid of fluorescein stain, and the eyes were treated with topical local anesthetics, antibiotics, oral analgesia, rest, and eye patches (see Figure 9-11B). All affected racers went on to complete the race.

Dermatologic problems, including sweat rashes, were seen mainly in the groins of male racers. All were treated successfully using appropriate topical creams.

All the female racers (and none of the male racers) developed a cold-induced urticarial rash isolated to the anterior surfaces of the thighs (Fig. 9-12). This was treated with oral antihistamines and topical steroids but was difficult to cure, taking days to weeks to resolve in a warmer environment after the race. Steps are being taken to design clothing for female competitors to prevent this from happening in the future.

Blisters were seen in 30% of competitors. All were treated effectively using DuoDerm hydrocolloid dressings. This type of dressing is extremely adherent and can be cut to any shape and applied for up to 5 days. Extra adherence at the edges of these dressings on high-friction areas like the heels can be obtained by using zinc oxide tape.

Trench foot was reported in two competitors at a late stage in the race (Fig. 9-13). Both were treated after completion of the race. Diligent foot care during the race would help prevent this injury.

Respiratory disease was seen once in a hypothermic competitor who collapsed during a storm with a severe case of asthma. The victim was unable to speak in complete sentences and was so cold that he had stopped shivering. He was rewarmed in a pre-warmed tent and treated with inhaled salbutamol and intravenous steroids, followed by oral steroids and oral antibiotics, with air evacuation as soon as there was a weather window. The competitor made a full recovery.

Fatigue and hypothermia affected all racers to varying degrees. Despite consuming 29,307,600 J (7000 cal) and drinking 4 L (4.2 qt) per day, most competitors are likely to lose 10% of their bodyweight. Training is given to all the teams on how to treat a teammate with severe fatigue and hypothermia. Only two competitors have withdrawn for such reasons (Fig. 9-14).

Gastrointestinal problems were mainly diarrhea, which was treated with loperamide and adequate hydration. Constipation, anticipated in the medical packs with senna tablets, has not yet been a problem.

Psychological problems, such as fear of polar bear attack or of becoming lost or injured, have led to at least one person pulling out of the race.

Lacerations and split skin were seen in some competitors; the lacerations were minor. All victims were treated very successfully using tissue glues (octyl cyanoacrylate tissue adhesive). This proved much more effective than teaching competitors suturing techniques.

Other potential injuries that are anticipated include major trauma from polar bear attack or firearms injury; burns, both minor (from stoves) and major (from tent fires); carbon monox-

Figure 9-12. Cold-induced rash. (Photo courtesy Ian Davis.)

Figure 9-14. Team racing. (Photo courtesy Ian Davis.)

Figure 9-13. Trench foot. (Photo courtesy Ian Davis.)

ide poisoning; long-bone fractures; medical emergencies (e.g., anaphylaxis, cardiac problems, infection, and pneumothorax); surgical emergencies (e.g., abdominal sepsis from appendicitis); dental problems; and problems unique to female racers, including vaginal candidiasis, menstrual problems, and urinary tract infections.

The Use of Frozen Medications

The medical literature is relatively sparse on this topic.[155] The author has rewarmed frozen fluorescein stain, oxybuprocaine hydrochloride 0.4%, ophthalmic local anesthetic, steroid creams, antifungal creams, inhaled salbutamol, and intravenous hydrocortisone, all of which worked normally after rewarming. All forms of tablet medication warmed from frozen have also been fine after rewarming. Medical devices, such as intravenous cannulae, worked normally after rewarming, as did all forms of tape and dressings. Frozen intravenous fluids expanded when frozen, perforated the bags, and thus leaked when rewarmed.

The references for this chapter can be found on the accompanying DVD-ROM.

10 Pathophysiology of Heat-Related Illnesses

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Developments in immunology, molecular biology, and systemic and cell physiology have broadened our understanding of heat-related illnesses. A brief outline of a few advances that are opening new avenues of investigation serves as an introduction to a more detailed discussion. Systemic mechanisms underlying heatstroke, based on studies of induced cellular tolerance to heat,[247] organ blood flow,[218] and hemodynamics in heatstroke,[280] are presented. Explanations of the causes and consequences of voluntary dehydration[255] are explored, along with the expected impact of water and salt depletion on physiologic parameters and exercise performance.

Because research into hyperthermia requires reproducible, systematic experiments, which are difficult to carry out in the wilderness in a random population, much research is carried out by military organizations. During basic and advanced training in various schools, students are routinely subjected to heat and physical stresses that are similar to those seen in the wild, but that can be reproducibly studied on military volunteers who are chosen because they are similar, or who can be rendered similar, in characteristics such as age, body mass index, diet, acclimation state, and hydration level. Furthermore, some military organizations maintain complex chambers to regulate ambient temperature and relative humidity, with nearby high-quality laboratories where volunteers can be studied using ergometers and oximeters. Results obtained by these groups can have great relevance for the civilian population in the wilderness and are described here in detail.

The demonstration that strenuous exercise can produce systemic endotoxemia,[54,381] and that endotoxins and the cytokine cascade have been implicated in acute heatstroke[59] and in other stressors, such as fear and even depressed mood,[289] may open new avenues of diagnosis and treatment. An update to the classic systemic approach to understanding the physiologic dysfunction in heatstroke is presented here and, where possible, related to emerging shock models involving cytokines and bacterial toxins. Furthermore, alterations in the activities of dozens to hundreds of genes have been found in human and other mammalian tissues subjected to heat and other stresses.[486] These studies have changed our understanding of physiologic stress pathways. Contrary to the usual textbook descriptions that reflect a relative simplicity and commonality of stress pathways, each stress type upregulates or downregulates hundreds of genes, with very few overlaps of changes in gene expression among several different stressors. Furthermore, unexpected upregulation of certain genes has pointed the way toward investigations of pathways, such as chitinase in human peripheral blood monocytes, that might otherwise not be suspected of involvement in heat stress.[486]

OVERVIEW OF HEAT ILLNESS

A characteristic property of warm-blooded animals is the ability to raise body temperatures higher than ambient temperature (T_{amb}). However, this property is not limited to vertebrates. For example, tent caterpillars can raise their body temperatures above T_{amb} to 30°C (86°F) or more using solar radiation.[109] Even certain plants can produce heat: in the winter, a philodendron flower can develop a temperature as high as 46°C (115°F) at a T_{amb} of 4°C (39.2°F).[464]

Accidental overheating is a danger to all organisms, but sometimes in nature, overheating is intentional and may be used as a weapon. Hornets occasionally attack the hives of Japanese honeybees. In response, scores of bees crowd closely around the invading hornet to form a ball of bees several layers thick. The bees vibrate their muscles rapidly, raising their own core temperature (T_c), which in turn heats the hornet to approximately 43.3°C (110°F). The hornet then dies of "heatstroke," and the relatively heat tolerant bees survive.

In humans and probably most animals, hyperthermia is usually not painful and may even induce a pleasant euphoria. It is this lack of a pain warning that is the major facilitator of heat illnesses. As a result, highly motivated athletes or soldiers may choose to continue performing severe exercise in the heat, even as the risk of heat injury increases.

Although most heat illnesses represent points on a continuum of disorders, it is convenient to classify the more familiar heat disorders into separate, well-defined categories such as heat syncope, heat exhaustion (including that induced by exertion, and by water or salt depletion), heat cramps, and classic and exertional heatstroke (EHS). Because they are part of a continuum of physiologic dysfunctions, the symptoms often overlap.

HEAT STRESS AND THERMOREGULATION

Mechanisms of Heat Transfer

The human body obeys the laws of thermodynamics, and heat is transferred from a higher to a lower temperature. Applying these principles, Santee and Gonzalez[448] and Gonzalez[201] provide an excellent discourse on characteristics of the thermal

environment, the biophysics of heat transfer, the heat balance equation, and clothing considerations. When environmental temperature is higher than skin temperature (T_{sk}), the body gains heat; when it is lower, the body loses heat. As a result, elevated environmental temperature adds to the heat burden of the body and interferes with heat dissipation. The four fundamental mechanisms of heat transfer in humans are conduction, convection, radiation, and evaporation of sweat, with a minor component of humidification of inspired air. The environmental parameters that determine the potential for heat exchange constitute the thermal environment, which has been defined as "a biophysical aggregate of air temperature, wind speed, relative humidity, and radiation."[508] Humans have another important way to effect thermoregulation—their behavior (i.e., by wearing appropriate clothes, entering an air-conditioned or a heated environment, using shade in the heat or solar energy in the cold).

Conduction

Conduction is heat exchange between two surfaces in direct contact. Because the areas are usually small (feet in contact with the ground) during the day in most mammals, conduction is generally the least important in quantitative terms, and behavioral thermoregulation (e.g., wearing of insulated boots while walking on hot desert sand) generally intervenes. However, lying uninsulated while sleeping on either hot or cold ground can result in significant heat exchange, especially for a person under the influence of vasodilating drugs or alcohol. A person may comfortably hold the handle of a very hot cooking pot with a dry towel (containing a large number of tiny cells filled with air). However, if the towel becomes wet (the cells are filled with water), it rapidly heats up, because conduction of heat through water is much faster (approximately 30 times faster) than through air, and within a few seconds, the heated wet towel becomes too hot to grip.

Convection

Convective heat exchange refers to heat transferred from a surface to a gas or fluid, usually air or water, whether induced by thermal currents, body motion, or the natural movement of air (wind) or water. It is a more complex process than conduction because the medium of heat transfer (air or water) is usually moving. The rate of heat exchange by convection depends on many variables, including density of the fluid (e.g., water versus air), temperature gradient, surface area exposed, and heat capacity and flow rate of the fluid. Heat loss during immersion in cold water is faster than when standing nude in air because of the high thermal capacity of water, the greater body surface exposed, and the greater thermal conductivity of water compared with air.[415,421] Convective heat exchange varies with the velocity of air movement. As T_{amb} of air rises to approach T_{sk}, heat loss becomes minimal. Once air temperature exceeds mean skin temperature, heat is gained by the body. Loose-fitting clothing maximizes convective and evaporative heat loss. By the same token, body motion alters heat exchange by convection, both within (the air pocket between the skin surface and fabric) and on the surface of clothing.

Radiation

Radiation refers to transfer of heat between the body and its surroundings by electromagnetic waves. All matter absorbs and emits thermal radiation,[488] which is the "radiant energy emitted by a medium that results solely from the temperature of the medium."[488] Clothing reduces radiant heat impinging on the skin from industrial sources and from solar load in broad daylight. Solar load is a major source of heat gain in hot climates: up to 250 kcal/hr (1050 J/hr) in a seminude man[195] (approximately the same as walking; see later discussion) and 100 kcal/hr (420 J/hr) in a clothed man. The amount of solar radiation varies with the angle of the sun, the season, clouds, and other factors. At the same time, clothing affects the evaporative process.

Highly pigmented skin is protected from ultraviolet radiation, but it absorbs approximately 20% more heat than nonpigmented or relatively nonpigmented skin.[287] Increasing blood flow to the skin (BF_{sk} at $T_{sk} > T_{amb}$) maximizes heat loss from the skin[231,436] by convection and radiation. Although heat is carried by convection and conduction from the body core to the skin, the main role of elevated BF_{sk} in a warm environment is to deliver the heat necessary to vaporize sweat.[198] Radiation from the body is quantitatively large and may account for 50% of the total heat loss of a nude human in the shade at neutral ambient temperature.

Evaporative Cooling

Evaporative, "wet," or insensible heat exchange is usually a one-way heat flow from a body surface to the environment. The heat of vaporization (0.59 kcal/kg [2.45 J/kg]) is absorbed slowly, with a small or undetectable change in temperature of the skin or blood. It may be sensed as chilling of the skin, causing goose bumps (piloerection) when coming out of the water at a swimming pool or beach. As water is converted from liquid to a gaseous state during heat exchange, the rate of evaporation of water from the skin, and hence the cooling effect, is proportional to the difference between vapor pressures of water in sweat on the skin surface and that of surrounding air. The presence of sodium chloride (NaCl) lowers the vapor pressure of water. Therefore, a low NaCl concentration in sweat increases this difference, increasing the rate of evaporation and cooling. Because acclimatization leads to lower NaCl concentrations in sweat, acclimatization increases sweat evaporation rate and rate of cooling.[245]

If the body is unable to maintain thermal equilibrium by a combination of radiation, convection, and conduction, and the core temperature rises, sweat is secreted to remove heat by evaporation of water.[198] Evaporation of 1 L of water is accompanied by the loss of 580 kcal/L, or approximately 1 kcal of heat is lost by evaporation of 1.7 mL of sweat.

The ability to maintain a constant sweat rate depends on a constant total body water volume. This in turn requires ingestion of water at the same rate as the sweat rate, and it must be within the stomach's capacity to empty water into the intestine. Because rates of gastric emptying and delivery of water to the intestines can exceed 1 L/hr, a sweat rate of 1 L/hr appears sustainable without significant dehydration if fluid is consumed. Higher sweat rates (e.g., 1500 mL/hr) could theoretically achieve greater rates of heat loss (1500 mL/1.7 mL/kcal = 882 kcal), but this is almost never achieved because some sweat drips off the skin and thus has no cooling effect. Moreover, such high sweat rates may be greater than the rate of gastric emptying, so sweating may occur at the expense of total body water. Losing 650 kcal/hr is a more reasonable figure for an upper limit of heat dissipation.[301]

Certain parts of the body surface are more important than others in providing cooling through sweating. The scalp, face, and upper torso are most important, and only about 25% of total sweat is produced by the lower limbs.[17] Sweat that drips from the body provides no cooling, and sweat evaporated from clothing is considerably less efficient than sweat evaporated directly from skin.[153] Therefore, wearing a shirt may be critical in causing heat illness, whereas wearing long or short pants is much less important. In particular, wearing protective headgear may be most important in the development of heat illness, as shown by a training exercise in the British Army. To bareheaded persons, the exercise was merely grueling, but among those wearing hats, 33% became heat illness casualties[238] (as cited in Porter[413]).

The maximal rate of sweat vaporization depends on air dryness and movement.[96,455] The environment's capacity to vaporize sweat varies primarily with humidity and also with wind velocity. As humidity approaches 100%, evaporative heat loss is minimized. The major effect of wind in speeding evaporation of sweat occurs in humid environments at a velocity between 0.5 and 5 m/sec (1.8 and 18 km/hr).[153]

The risk of developing hyperthermia increases with air temperature and humidity. As a result, military trainers and coaches tend to conduct runs in the early morning to avoid high T_{amb} and the increased incidence of heat illness in humid climates, despite the lower dry-bulb temperatures (T_{db}). Exertional heatstroke (EHS) is reported with increasing frequency during "fun runs" and marathons when the temperature is not particularly hot.[498] This reinforces the simple definition that exertion-induced heatstroke occurs whenever the rate of heat production of an exercising individual exceeds the rate of heat loss, causing body temperature to rise to critical levels.

Heat Stress Indexes

The ability to work in a hot environment is inversely related to the prevailing heat stress level; the higher the heat stress level, the shorter is the ability to carry out work and the greater is the risk of heat illness. Safety in a hostile environment depends on following strict rules and limitations concerning exposure time and work intensity. The need for a quantitative index that combines environmental heat stress factors to provide a reliable and consistent correlation with the induced physiologic strain was recognized 100 years ago.[216]

In 1905, Haldane suggested the use of wet-bulb temperature (Tw) as an index of the severity of a warm environment in Cornish tin mines.[216] Tw is lower than dry-bulb temperature because of the cooling effect of evaporation of water from the thermometer, which, in turn, varies inversely with relative humidity (RH). However, at high humidity levels and in hot dry climates, the relationship between Tw and physiologic strain (i.e., rises in T_c, HR, respiratory rate) was not valid.[34] In 1923, Houghton and Yaglou[252] developed the effective temperature (ET) index to define thermal comfort limits. This index was a combination of Tw, T_{amb}, and wind velocity (V_a), and it produced an equivalent thermal sensation. However, several deficiencies were recognized, including overestimation of the effects of T_{amb} at high temperatures and underestimation of V_a in hot wet climates.[34]

In 1957, Yaglou and Minard[549] suggested the wet-bulb globe temperature (WBGT) index, which consisted of combining T_{amb}, Tw, and black globe temperature (T_g) in unequal weights:

$$\text{for indoor use, WBGT} = 0.7\,\text{Tw} + 0.3\,T_{amb}, \text{ and}$$
$$\text{for outdoor use, WBGT} = 0.7\,\text{Tw} + 0.2\,T_g + 0.1\,T_{amb},$$

in which the effect of solar radiation was included (T_g) in the index.

However, calculation of WBGT involves measuring T_g from a thermometer surrounded by a 6-inch-diameter blackened sphere, which is inconvenient and not practical under many circumstances. Nevertheless, the WBGT index is today the most widely used index to describe environmental heat stress for outdoor and indoor use and is used to set limits for U.S. military training exercises in hot weather.[73,124] It is also used to set limits in industrial plants,[380] by sports associations as guidance to prevent heat injury,[34,353] and by workers in different occupations as a safety index.[90,127,212,476] However, WBGT is limited for two main reasons: (1) it cannot be applied to persons wearing different types of clothing (e.g., protective clothing), and (2) measuring T_g is inconvenient. Wearing protective clothing imposes a higher heat stress, equivalent to adding 6° to 11°C (10.8° to 19.8°F) to the WBGT index.[409] As a result, for WBGT to accurately reflect environmental stress, corrections and adjustments must be made for type of clothing worn and intensity of physical activity (metabolic rate).[243,282]

Attempts were made to develop alternatives to WBGT. In 1971, Botsford[56] suggested the wet globe thermometer (WGT), known also as the Botsball, which combined measuring T_{amb}, Tw, and radiation into a single reading. Unfortunately, the WGT index did not provide precise enough values in hot climates, where its readings were significantly lower than the WBGT index.[352]

In 1962, Sohar and colleagues[484] suggested the discomfort index (DI), using a combination of T_{amb} and Tw, which has been used extensively in Israel. A modified DI (MDI) was proposed, in which the coefficients to the parameters T_{amb} and Tw were altered to achieve a better correlation with the WBGT index.[365] However, uncertainties in the radiation component in any heat stress index (e.g., solar radiation measurement for the shade versus open sky) limit its value. Because the most widely used WBGT index is computed from three separate environmental measurements, the instrumentation to measure this index is cumbersome and more suited to a fixed-site station than to a mobile situation. Furthermore, about 30 minutes is required for the instrument to reach equilibrium when measuring T_g by a black globe thermometer.

Recently, a new environmental stress index (ESI) was developed[367] and evaluated.[368] It was calculated from T_{amb}, RH, and solar radiation measurements, the sensors for which are all available in a microsensor format.[368]

Physical activities in a hot environment require a fairly accurate measurement of the stress index to prevent heat illnesses and to determine safety behavior patterns (e.g., water consumption rates and work–rest cycles). To implement the current guidelines and limitations for exercise in a hot climate, there is great need for development of an accurate, portable heat stress measurement device. Current technology (e.g., very small electronic sensors to measure T_{amb} and RH, a miniature display, and a miniature programmable microprocessor for calculating and storing the MDI index) is already available in a wristwatch format, which should facilitate the devising of a new portable heat stress monitor. Such a tool could help in the decision-making process relating to permissible strain in a hostile environment and could be used by medical monitors, leaders, and

others directly exposed to heat stress. These measures would clearly help prevent heat illnesses and decrease dependency on weather-station reports.

In a Marine training base with 17,000 to 25,000 recruits per year, clinical studies from the 1950s reported a large number of heat casualties occurring as a result of strenuous physical exercise when the WBGT was higher than 26° C (78.8° F).[359] Therefore, new regulations and guidelines were implemented for different heat categories, which reduced the numbers of casualties.[215] However, Kark and coworkers[277] reported 1425 cases of exertional heat illness from 1982 to 1991 and showed that 25% of the cases occurred between 7:00 to 9:00 AM at a WBGT level of 26.7° C. These cases occurred in spite of the preventive measures taken on Parris Island, South Carolina, which involved rescheduling training and physical activity according to newly categorized WBGT values. In addition, further attention was given to clothing, equipment, workloads, and hydration. Exertional heat illnesses continue to be a common problem during training in a warm environment despite preventive measures taken.[277] The WBGT index provides for measurement of heat load, but its practical application in current army doctrine is limited. Its utility would be improved if the weather station were close to the trainees at the training site, and if the environmental measurements were taken continuously and not once per hour.

In the civilian population, dehydration is one of the most common risk factors when exercising in the heat, with fluid loss occasionally as high as 6% to 10% of bodyweight.[106] For each 1% of bodyweight lost to dehydration during exercise, T_c rises an additional 0.15° to 0.2° C (0.27° to 0.36° F). Other risk factors for heat illness include hot and humid climate, obesity, low physical fitness, lack of acclimatization, previous history of heat stroke, sleep deprivation, medications (especially diuretics or antidepressants), sweat gland dysfunction, and upper respiratory or gastrointestinal illness. Close monitoring of signs, symptoms, and hydration status, coupled with early intervention with fluids, may be the most important factor in preventing severe heat illness.

Temperature Regulation

To prevent or appropriately manage heat illness, the clinician must understand both thermal stress[171] and human thermoregulation (see Chapter 4).[198,226,496] Temperature regulation refers not only to behavioral factors but also to autonomic thermoregulatory processes that modify the rates of heat production via shivering and variations in basal metabolism, and of heat loss by sweating and variations in peripheral vasomotor tone.[198] These processes act to maintain the temperature of the body within a restricted range with a variable internal or external heat load. The regulated temperature is generally considered the T_c.

Heat Exchange and Skin Blood Flow

Body temperature is not regulated directly by changes in a single physiologic variable. Like blood pressure (which is the product of cardiac output and resistance), body temperature is a derived function that increases with metabolic rate and decreases with cooling rate. The latter, in turn, depends in a nonlinear manner on a number of factors: rate of blood flow to the skin; ambient temperature; heat gain or loss by conduction, convection, radiation, and sweat evaporation; relative humidity; and wind speed.

The body core (brain, heart, lungs, kidneys, liver, spleen, gastrointestinal tract), though small (only 6% to 10% of body mass), accounts for most (about 75%) of the resting metabolic rate.[23,204] In contrast, the body periphery (skin, fat, muscle, bone), though large (>90% of body mass), has a low resting metabolism (<35% of the total). That is, at rest, peripheral tissue is relatively silent.

At high T_{amb} or during severe exercise, it is the temperature of the core organs and not that of the periphery that is defended by heat-dissipating mechanisms (radiation, convection, conduction, and evaporation). Those cooling mechanisms occur at skin surfaces, and therefore, for efficient cooling, blood flow to the skin must increase during hyperthermia. Control of BF_{sk}, hence control of T_c, involves a complex interaction of regulatory systems (body temperature, blood pressure, metabolism) and efferent mechanisms (passive withdrawal of constrictor tone, reflex vasoconstriction, active vasodilation).[284]

At the same time that T_c is rendered relatively constant during hot or cold T_{amb}, the temperature of peripheral tissues can vary substantially with environmental temperature, over a range from about 1° to 44° C (33.8° to 111.2° F). The metabolic rate of most body tissues at any given moment determines its own regional distribution of blood flow. In resting humans, the core is metabolically the most active; hence, it receives most of the blood flow.[167] However, during exercise, muscle metabolic rate may rise 10-fold and muscle blood flow increases accordingly.[311] Because only a fraction of the heat is dissipated instantaneously, most of the heat returns to the core.[285]

Most mammals have extensive regions of hair or fur insulation on their skin. Thus, heat is most rapidly lost from skin regions lacking hair. The rate of heat loss depends on several factors, among them rate of heat flow (blood flow) to the skin, and the thermal gradient (difference between environmental and blood temperatures).

Body Temperature Shells

Under normal resting conditions at neutral T_{amb}, body temperature is not uniform at 37° C (98.6° F) but instead may be considered to be a series of shells of uniform temperatures, highest in the core and decreasing in the extremities (Fig. 10-1). During cold ambient temperatures, only the temperature of the core organs is defended at 37° C (98.6° F), and the temperature of the greater mass of the periphery at cold T_{amb} may fall to below 20° C (68° F) and even, during severely cold environmental conditions, to below 0° C (32° F), leading to frostbite (see Figure 10-1).

A practical aspect of distribution of body temperature in shells is seen during races run on cool or cold days. Because the rate of chemical reactions increases with temperature, the intrinsic speed of shortening of skeletal muscle is less at 25° C (77.0° F) than at 37° C (98.6° F), placing an athlete with a peripheral temperature of 25° C (77° F) at a disadvantage compared with one at 37° C (98.6° F). Therefore, there is a need in cool or moderate T_{amb} for warming-up exercises that warm the extremities, thus increasing the intrinsic speed of muscle contraction. At temperatures 3° or 4° above 37° C (5° or 6° above 98.6° F), however, proteins denature and muscle function is decremented. During hot weather or severe exercise, the temperature of the core is defended at 37° C (98.6° F), but the temperature of blood returning from large leg muscles can be higher. Consequently, the temperature of blood flowing through the skin may be elevated to increase the rate of heat loss across the skin by maximizing the

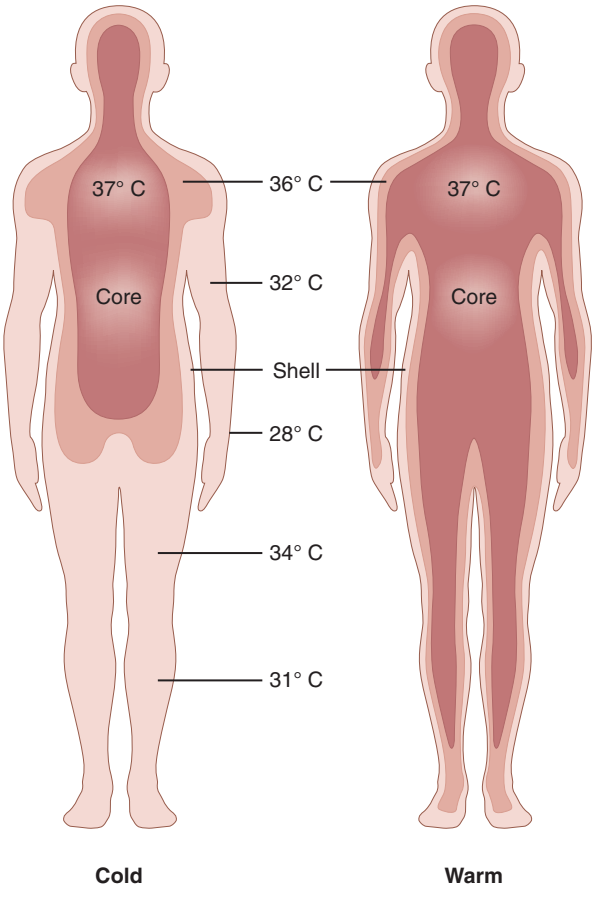


Figure 10-1. Distribution of temperatures within the human body into core and shell during exposure to cold and warm environments. The temperatures of the surface and the thickness of the shell depend on the environmental temperature: the shell is thicker in the cold and thinner in the heat. (From Elizondo RS: In Rhoades RA, Pflanger RG [eds]: Human Physiology, 3rd ed. Philadelphia, Saunders College, 1996. Reprinted with permission of Brooks/Cole, a division of Thomson Learning: www.thomsonrights.com. Fax: 800-730-2215.)

thermal gradient. Water immersion cools the skin and body core by the returning cooled blood, but it also cools the tissues immediately below the skin by conduction.

Blood flow through the skin has two main functions: to deliver oxygen and nutrients to the skin for its own metabolism, and, at elevated temperatures, to dissipate excess heat from the core to the environment. To efficiently carry out both of these skin functions, within the skin are two different types of blood vessels, nutritive units and heat exchange units, which are structurally and functionally different from one another. Nutritive vascular units are distributed everywhere throughout the skin. On the other hand, heat exchange units (also known as AVAs, for arteriovenous anastomoses) are present only in the noninsulated skin regions, including the palms of the hands, soles of the feet, ears, and nonhairy regions of the face (Fig. 10-2). In the hand, heat exchange units are present under the nail beds, on the tips of digits, in the palm, and on the palmar surface of the fingers, but they are absent from the dorsal surface of the fingers and hand.[430] The flow of heat and blood through the heat exchange units can range from almost zero during cold stress to very high levels (60% of cardiac output), depending on the body's metabolic rate and environmental temperatures. Blood and heat from the core are delivered directly to the heat exchange units, and at elevated T_c, they return cooled to the heart (see Figure 10-2).

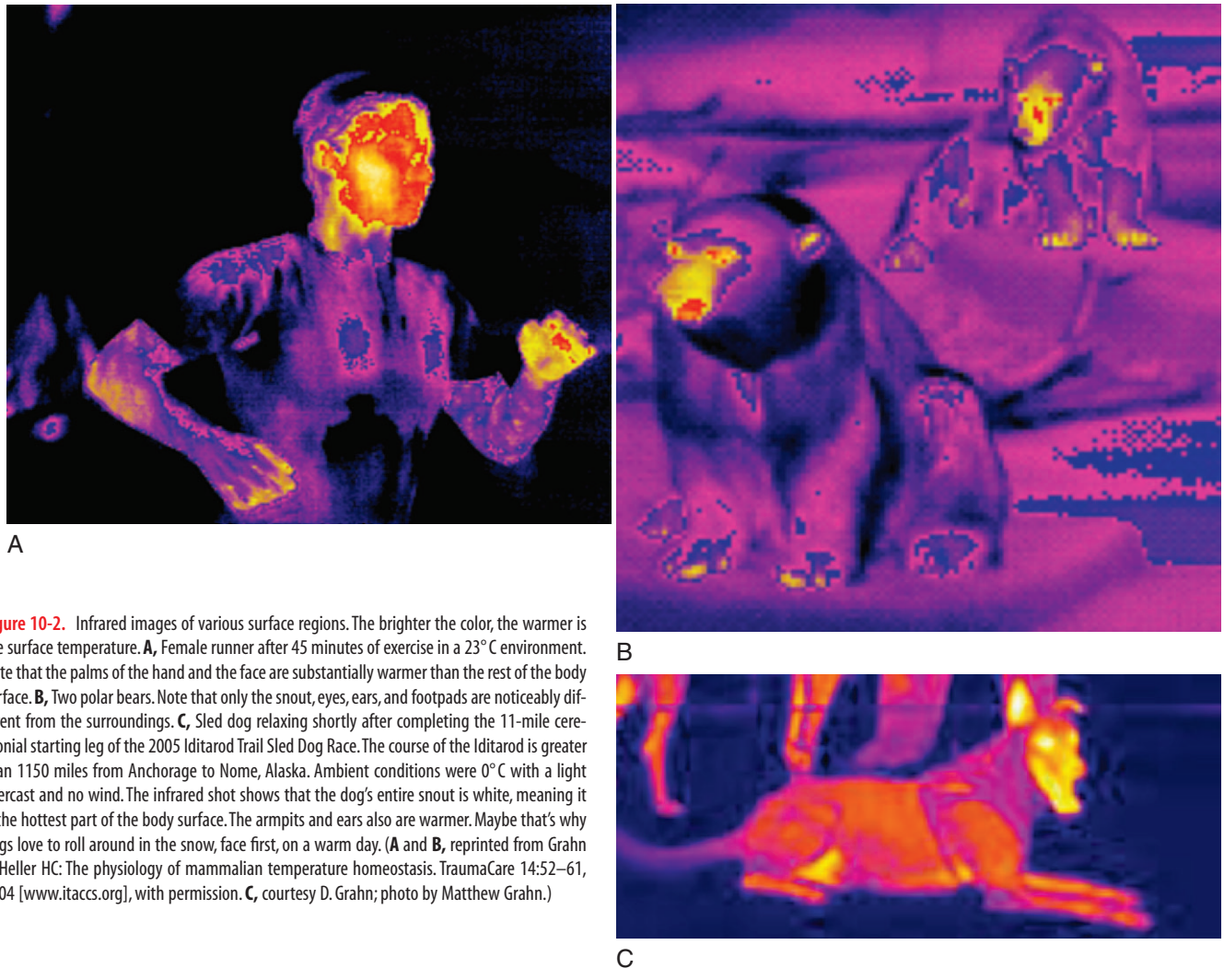
For patients with heat injury, cooling strategies that are based on cooling large areas of the skin, such as immersion in tepid or ice water, can lead to imbalanced thermal signals in preoptic and anterior hypothalamic brain regions.[72] Prior to active cooling of a heat injury victim, hyperthermic blood passing through the hypothalamus stimulates increased flow of blood through the skin. However, during cooling with ice or cold water, cold receptors present in the skin would be expected to send a stimulus to the hypothalamus to reduce BF_{sk}, leading to skin vasoconstriction. The complex interactions between the factors increasing or decreasing blood flow through the skin are not well understood.

For quite some time, there has not been complete agreement on the most rapid way to cool a victim of heat illness. It had been widely believed that immersing the victim in tepid water or using a spray of warm water to cool the victim by evaporation are the fastest methods, because they would not cause much skin vasoconstriction. These techniques avoid cooling by immersion in cold or ice water, on the belief that immersion causes skin blood vessels to constrict, thus potentially slowing the rate of cooling. No side-by-side comparison of the various means of cooling hyperthermic humans has been reported.[214] In one study, several hundred consecutive Marine recruits were cooled by being covered with sheets drenched with ice and cold water while maintaining continuous vigorous hand massage for several minutes, until T_c fell to 39° C (102.2° F). No fatality was reported. Ice water drenching of heat injury victims has become the standard cooling method of the U.S. Army and Marines and the Israel Defense Forces (IDF).[179,214]

The fundamental mechanism by which this cooling procedure is so effective is not fully clear. Does the rapid cooling rate result from the high thermal gradient across the skin caused by the ice and water despite the vasoconstriction? Or does the manual massage during the drenching procedure increase local arteriolar blood pressures and physically overcome vasoconstriction normally caused by skin cooling?

A new series of studies by Grahn and colleagues[205] touches on this matter. They developed a novel cooling device to lower or raise core temperature without cooling peripheral tissues. The idea is based on the preferential blood flow and heat exchange through the specialized regions of nonhairy skin—the palms of the hands, the soles of the feet, the ears, and regions of the face. In a hand-held device (AVAcore Technologies, Ann Arbor, MI), the hand passes through a sealing wrist cuff, the palm grips a curved metal heat sink adjusted to 22° C (71.6° F) by thermoregulated circulating water, and a moderate vacuum (−40 mm Hg) is applied. The low vacuum maintained within the chamber of the device serves to maintain an elevated blood flow under the local cool conditions that might otherwise cause vasoconstriction. This chamber behaves as if it opens the AVAs or maintains their open position when they might otherwise be vasoconstricted. This allows cooling of venous blood, which returns to the core.

By cooling one hand, this device was capable of slowing the rise in core temperature during treadmill exercise at increasing intensities and thus allowed prolonged performance (Fig. 10-3). It also shortened the period of recovery from hyperthermia due



A

B

C

Figure 10-2. Infrared images of various surface regions. The brighter the color, the warmer is the surface temperature. **A,** Female runner after 45 minutes of exercise in a 23° C environment. Note that the palms of the hand and the face are substantially warmer than the rest of the body surface. **B,** Two polar bears. Note that only the snout, eyes, ears, and footpads are noticeably different from the surroundings. **C,** Sled dog relaxing shortly after completing the 11-mile ceremonial starting leg of the 2005 Iditarod Trail Sled Dog Race. The course of the Iditarod is greater than 1150 miles from Anchorage to Nome, Alaska. Ambient conditions were 0° C with a light overcast and no wind. The infrared shot shows that the dog's entire snout is white, meaning it is the hottest part of the body surface. The armpits and ears also are warmer. Maybe that's why dogs love to roll around in the snow, face first, on a warm day. (**A** and **B,** reprinted from Grahn D, Heller HC: The physiology of mammalian temperature homeostasis. TraumaCare 14:52–61, 2004 [www.itaccs.org], with permission. **C,** courtesy D. Grahn; photo by Matthew Grahn.)

to exercise in the heat (Fig. 10-4). The device contains a cooling thermoregulated water bath and therefore is not highly portable. At present, a commercialized version is being used by professional sports teams for athletes resting after periods of intense physical activity.

Behavioral Thermoregulation

From an evolutionary perspective, hyperthermia appears to represent a more serious problem than does hypothermia, because humans have developed a greater capacity for heat elimination (vasodilation, sweating) than for heat conservation (vasoconstriction).[169,226] To live, work, and reproduce successfully in arid or tropical climates, all land animals, including humans, depend not only on physiologic mechanisms to acclimatize to heat but also on behavioral responses to assist temperature regulation. Thus, humans possess two control systems (behavioral and physiologic) to regulate body temperature.

Although behavioral responses (e.g., seeking shelter from the cold, wearing clothes, building fires) that augment shivering seem obvious in a cold environment, behavioral factors for thermoregulation in the heat (resting in the heat of the day, seeking shade or water when thirsty, reducing the intensity of exercise or ceasing to exercise, removing clothes) are also important. Behavioral responses are conscious actions: the behavior is induced by subjective sensations of thermal discomfort that anticipate stressful changes in T_c.[111]

Heat Production

The basal metabolic rate of the average 70-kg man in a sitting position is approximately 50 to 60 kcal/hr/m^2 of body surface area, or 100 kcal/hr.[305] With increased physical activity, metabolic heat production increases significantly (walking produces 250 to 300 kcal/hr; walking rapidly with a load, up to 400 to 450 kcal/hr) and may reach 20 times baseline level with strenuous exertion. Depending on the exercise task, usually 70% to 85% of the metabolic rate is released as heat that must be dissipated to maintain thermal balance. If there were no means to dissipate heat, the addition of 70 kcal to a 70-kg person would

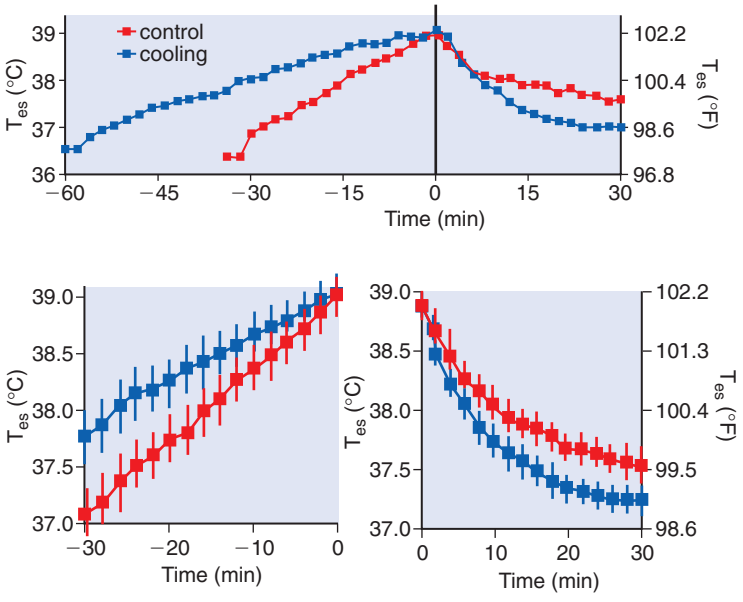


Figure 10-3. Esophageal temperature (T_{es}) during exercise and recovery in a heated room, while the Grahn heat-extraction device was used. The rate of rise of T_{es} during exercise was decreased with cooling, which enabled a longer duration of the exercise. *Blue symbols,* cooling through one hand; *red symbols,* control treatment. **Top:** A single subject. **Bottom:** Mean results of $N = 5$, during the last 30 minutes of exercise (*left*) and after exercise recovery (*right*). (Reprinted from Grahn D, Heller HC: The physiology of mammalian temperature homeostasis. TraumaCare 14:52–61, 2004 [www.itaccs.org], with permission.)

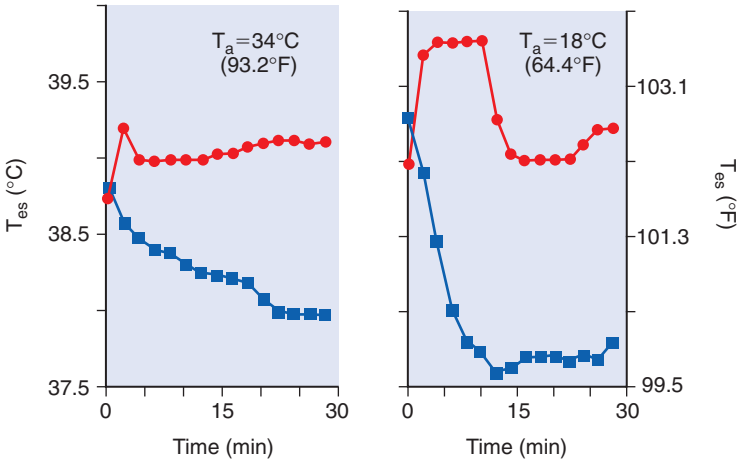
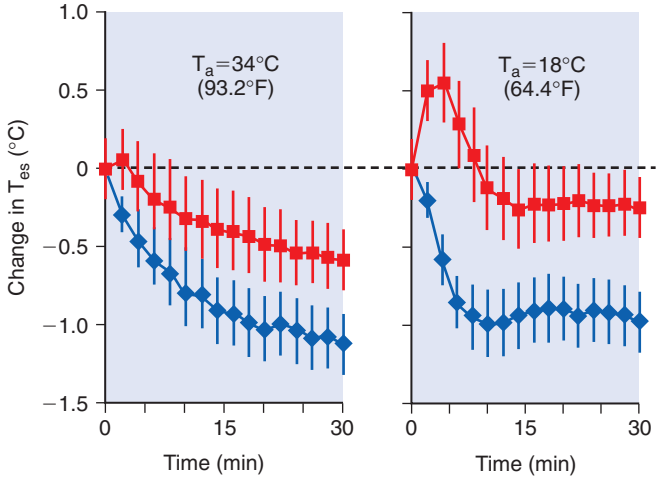


Figure 10-4. Effect of postexercise heat extraction on esophageal temperature (T_{es}). **Top:** Individual examples of T_{es} versus time from end of exercise. **Bottom:** Group results. *Left:* Recovery in a cold room. *Right:* Recovery in a cool room. *Blue symbols,* heat extraction; *red symbols,* control treatment. (Reprinted from Grahn D, Heller HC: The physiology of mammalian temperature homeostasis. TraumaCare 14:52–61, 2004 [www.itaccs.org], with permission)



theoretically increase core temperature approximately 0.8°C per minute, assuming the average specific heat of the human body to be 0.8 kcal/kg/°C. (The specific heat is not the 1.0 kcal/kg/°C of water because of the presence of bone, fat, and protein.) Cellular metabolism increases 13% for each 1°C (1.8°F) rise in temperature until the body approaches heatstroke temperature; at that point, cellular metabolism increases more rapidly. It is 50% above normal at 40.6°C (105.1°F).[75]

Body Temperature Ranges

Normal internal temperatures range from 36° to 38°C (96.8° to 100.4°F), whereas the limits of body temperature for efficient thermoregulation are approximately 35° to 40°C (95° to 104°F).[495] However, during athletic events, T_c increases to 40° to 42°C (104° to 107.6°F).[130,374,546] One marathoner was able to maintain a T_c greater than 41.5°C (106.7°F) for at least 44 minutes during a race.[346] Survival limits of body temperatures are exceptions to these ranges. For example, one heatstroke victim survived a measured T_c of 46.5°C (115.7°F).[480]

Sensing, Relaying, and Central Integration Functions

Physiologic temperature regulation involves detecting changes in body temperature by sensory mechanisms and relaying thermal signals from central and peripheral locations to a central integrative area. This directs effector organs to increase or decrease heat storage[198] and is mediated by the autonomic nervous system. Sensitive nerve endings within the hypothalamus and near the skin surfaces over most of the body act as thermoreceptors[238,379] to monitor T_c and T_{sk}.

The preoptic area of the anterior hypothalamus of the brain appears to be the primary site for integration and generation of a thermal command signal. This area contains neurons that alter their firing rate in response to warming or cooling.[61] The brain is well perfused relative to its mass and responds rapidly to changes as small as about 0.1°C (0.18°F) in blood temperature.[42,43]

The primary means of regulating T_c are vasomotor alterations in blood flow and its regional distribution, shivering plus activation of uncoupling protein (nonshivering thermogenesis), and sweating. The threshold temperatures for initiating increased BF_{sk} and forearm venous volume and sweating depend on both T_c and T_{sk}. Heating the skin lowers these threshold temperatures.[100,450,532] At any given T_c, heating the skin increases the effector response. Mathematical modeling suggests that T_c is approximately nine times as important as T_{sk} in the reflex control of BF_{sk}.[452] However, the importance of T_{sk} should not be minimized. Whereas T_c varies over a narrow range of 7°C (12.6°F), the variation in T_{sk} is threefold to fourfold greater because of the wide range of environmental temperatures.

During exercise, the rise in body temperature is proportional to the increased metabolic rate and, over a wide range, does not depend on T_{amb}. Increase in T_c elicits an effector response (sweating) with only minimal change in T_{sk}.[387] However, this relationship does not hold under extremely hot or humid conditions or conditions of maximal effort; consequently, heat exhaustion becomes manifest. This disparity between theory and common experience gave rise to the concept of a prescriptive zone, or a set of conditions in which the magnitude of the core temperature response for everyday work was independent of environmental temperature.[331]

Sweating and skin vasodilation increased linearly above the set-point temperature of approximately 37°C (98.6°F).[42,43] Local heating of the preoptic area accelerates sweating. Thus, central and peripheral mechanisms cause sweating when skin temperature is elevated. Local heating may result in a greater release of neurotransmitter for a given sudomotor signal,[149] or heating increases sensitivity of the hypothalamus to a given dose of neurotransmitter.[393] On the other hand, lower skin temperatures may inhibit sweating during exercise.[42] In addition, spinal centers for temperature regulation appear to contribute, as sweating is not completely abolished by transection of the cervical spinal cord.[505]

Set-Point Hypothesis

The concept that the central nervous system is a functional interface (a central integrative area) between the thermosensors and thermoregulatory effectors is not accepted by all physiologists.[474] However, it provides a rationale for envisioning a thermostat, or set point, that shifts all effector thresholds in the same direction.[198] This concept of a central thermostat provides a conceptual framework that fits a variety of situations.

The principal mechanisms involved in temperature regulation during exercise in the heat are venodilation, increased BF_{sk}, and sweating. Dilation of superficial veins increases efficiency of heat flow from the core to skin and increases time available for heat transfer between the blood and skin. The cutaneous vasculature is therefore an effector system in thermoregulation because BF_{sk} controls the rate of heat transfer between the body core and the surface.[198,436] Unless the rate of heat storage (i.e., a rise in T_c) exceeds the capacity of the thermoregulatory system (e.g., a breakdown of heat dissipation mechanisms), effector responses will increase until heat balance is restored.

Vasomotor System

The term *vasomotor system* refers only to the arterioles that control organ blood flow, vascular resistance, and arterial blood pressure.[436] Because arterioles and the heart (or cardiac output [CO]) together regulate blood pressure, a change in blood pressure caused by alterations in regional blood flow is corrected by changes in vascular resistance or CO.[198,227,436] Vasomotor adjustments optimize and regulate distribution of CO within and between different organ systems. Central thermal receptors alter vasomotor outflow to redirect blood flow to the skin. With exposure to cold, BF_{sk} in humans can be reduced to as little as 1 mL/100 g of skin per minute and during maximal heat stress can increase to 150 mL/100 g of skin per minute, greater in the extremities than in the trunk.[245]

Blood flow to the skin is under dual vasomotor control. In the cold, adrenergic vasoconstrictor fibers reduce skin blood flow.[170] However, during heat exposure, blood flow increases to the hands, lips, nose, and ears as a result of vasodilation, largely because of withdrawal of vasoconstrictor tone.[170] Active sympathetic vasodilation over most of the skin area[240] reduces vascular resistance below basal tone. There is a relationship between active sympathetic vasodilation and sweating, perhaps through release of vasoactive intestinal polypeptide (VIP).[515] Simultaneous release of both transmitters (VIP and acetylcholine) could help explain the apparent relationship between eccrine sweat secretion and cutaneous vasodilation.[337]

Venomotor System

The venomotor system controls the veins. It should be noted, however, that there is dual venous drainage of the limbs. The

deep veins draining mainly from the muscles have relatively poor sympathetic innervation,[526] whereas superficial veins draining the skin are richly innervated. Although venodilation and increased BF_{sk} enhance heat transfer,[200] they also increase the amount of blood pooled in compliant peripheral vessels. As a consequence, filling of these veins reduces central blood volume; at some point, redistribution of blood could compromise venous return and cardiac filling.[240,436] This system may be altered by fundamental changes in smooth muscles of the various vessels. Heat increases the speed and extent of contraction of femoral arteries in response to K^+ and catecholamines (i.e., increases vascular resistance) and may therefore inappropriately slow blood flow to and from the extremities.[400]

CLASSIC HEATSTROKE

The pathologic features of heatstroke, which are similar regardless of the cause of the heat illness, are manifested by swelling and degeneration of tissue and cell structures, and by widespread, microscopic to massive hemorrhages.[472] Most organs are congested, with increased masses and swollen cells. In the gastrointestinal tract, postmortem examination often shows massive ulcerations, hemorrhages, and engorged intestinal vessels.[472] A discussion of the gut is important in any discussion of heat illness for two reasons: (1) its function determines whether ingested fluid and solutes are delivered to the systemic circulation to correct losses and thereby attenuate hyperthermia, dehydration, and reductions in splanchnic blood flow and gut distress, and (2) heatstroke may be exacerbated by gastrointestinal dysfunction, leading to leakage of gut-derived endotoxin (lipopolysaccharide [LPS]) into the circulation, endotoxemia, and consequent circulatory collapse. Optimally, there is an equivalence between the volume of the rehydration demand, the rate at which the stomach can deliver water (gastric emptying under optimal conditions can provide 1.8 L/hr), and the rate at which the intestine can absorb ingested fluids (1.4 to 2.2 L/hr).

Classic heatstroke does not much relate to metabolic rate but occurs when environmental heat stress is near maximum and greater than the rate of heat dissipation.[146,228,492] Populations at risk include older adults, the poor (who lack adequate air conditioning), those who suffer from malnutrition, and those who have chronic diseases or substance addiction.[228] In classic heatstroke, in contrast to EHS, physical effort is not a primary determinant of excessive heat storage, and therefore the onset of classic heatstroke is substantially slower, commonly occurring over days rather than minutes or hours. As a result, there is often ample time for imbalances to develop in fluids and electrolytes.

Under some circumstances, passive hyperthermia can develop rapidly in extremely hot environments, such as when infants and small children are left in locked vehicles in the summer heat. This is also true for adults who are passengers in improperly ventilated or nonairconditioned vehicles. Under summer desert conditions, the temperatures of cabins and cargo spaces of vehicles can rapidly reach 54° to 60° C (129.2° to 140° F), depending on environmental temperature and solar radiation.[256] Similar high temperatures may occur in closed or confined spaces such as enclosed attics, or in places where there is a high radiant load from machinery or power plants, such as boiler rooms.[305] Individuals who depend on others for fluid intake because of age (older adults, the very young) or illness are at risk for involuntary dehydration. Infants are more heat labile because of their immature thermoregulatory systems and smaller thermal masses.[457]

Epidemiology: Heat Waves

Heat waves are usually defined as at least three consecutive days with air temperatures of 32.2° C (90° F) and are associated with heat illnesses, including classic heatstroke.[84] In California, heat waves associated with El Niño events were associated with hospitalizations among older adults for acute myocardial infarction, angina pectoris, congestive heart failure, and stroke.[142]

When environmental heat stress is maximal, strenuous exercise is not necessary to produce heat illness.[146,203,228,492] In Peking in 1743, a heat wave reportedly caused 11,000 deaths[472]; 411 cases of severe heatstroke in Nanjing were reported in 1988.[343] During the July 1996 Atlanta Olympics, there were 1059 heat casualties reported, of which 88.9% were spectators and volunteers.[535] In a separate heat wave, within 8 consecutive days,[160] 42 of 44 cases occurred at a time when the maximal ambient temperature varied from 38.9° to 41.1° C (102° to 106° F). The temperatures of the patients' local environments were probably much higher, as air conditioning was not widely available at that time. Only 7 of the 44 persons were less than 50 years of age, and most were in the range of 60 to 70 years.

In Boulder City, Nevada, July 8 to 18, 1936, T_{amb} reached higher than 38.3° C (100.9° F) daily. Forty-four heatstroke victims were admitted to the Boulder City Hospital, and the daily admission rates increased progressively during that period.[129] This suggests that prolonged hot weather causes progressive deterioration of the body. In a different heat wave near Dallas, Texas, lasting 26 days, the first case occurred on day 10 of the heat wave and the last case occurred 10 days after the end of the heat wave, with half the cases (14 of 28) occurring over a 3-day period on days 20 to 22. Age was a factor in both heat waves, with 8 of 72 victims older than 80 years, and with mean ages of 59 years in Boulder City and 70.5 years in Dallas. Alcoholism and degenerative diseases were contributing factors in both waves. The victims characteristically had high rectal temperature (T_{re}) and dry skin; half of the Boulder City patients and 24 of 28 Dallas patients were comatose. Of the 44 Boulder City patients, 23 showed a fiery red skin rash over the body, particularly over the chest, abdomen, and back.[129] The most common presentation in Dallas patients was that of respiratory alkalosis, often accompanied by metabolic acidosis.[228] All Dallas patients with blood lactate greater than 3.3 mMol/L (normal range, 0.6 to 1.8 mMol/L) suffered a poor outcome, whereas those with initial lactate less than 3 mMol/L did well. So, modest elevations (related to exercise studies) in blood lactate become adverse prognostic indicators in classic heatstroke. Furthermore, 9 of 28 classic heatstroke patients arriving with normal serum potassium subsequently became hypokalemic, and all victims were hypokalemic at some point in their course.[228]

A heat wave in Chicago in July 1995 resulted in more than 600 deaths, with more than 3300 emergency department visits and intensive care unit admissions for near-fatal heatstroke. In a group of 58 of these victims with classic heatstroke, 100% experienced multiorgan dysfunction with neurologic impairment, 52% showed moderate to severe renal insufficiency, 45% had disseminated intravascular coagulopathy, and 10% had acute respiratory distress syndrome. Fifty-seven percent of the

TABLE 10-1. Core Temperature (T_c) of Heatstroke Victims in Nanjing, July 1988

T_c ON ADMISSION (°C)	DEATH RATE (%)	NUMBER OF DEATHS*
<39	11.5	52
39–39.9	13.4	112
40–40.9	31.0	126
41–41.9	40.0	85
≥42	83.3	36

*Overall deaths, 124/411 (30.2%). Victims older than 60 years accounted for 77.4% of all deaths.
Data from Zhi-cheng M, Yi-tang W: Chin Med J 104:256–258, 1991.

Box 10-1. Complications and Symptoms of Heatstroke Victims in Nanjing, July 1988

COMPLICATIONS (%)
Diabetes mellitus (24.8)
Various chronic diseases (24.5)
Hypertension (24.3)
Coronary artery disease (6.1)
Obesity (5.3)
Cardiac insufficiency (4.6)
Cerebral vascular accidents (4.1)
Chronic bronchitis (1.7)
Neoplasm (1.7)

SYMPTOMS (% OF VICTIMS)
T_c > 40°C (60.1)
Xerosis and hot skin (46.7)
Coma (45.3)
Headache (35.5)
Convulsions (31.6)
Dizziness (29.2)
Weakness (29.2)
Incontinence (19.2)
Thirst (19)
Delirium (14.8)
Numbness of extremities (14.4)
Dysphagia and dysphonia (7.1)
Moist skin (3.2)
Kinetic imbalance (2.4)

Data from Zhi-cheng M, Vi-tang W: Chinese Med J 104:256, 1991.

victims had infections on admission. In-hospital mortality was 21%.[119]

During a July 1988 heat wave in Nanjing, China, at least 4500 victims of heat illness were treated, with 411 of them classified as severe[553] (Table 10-1 and Box 10-1). The mean ages for men and women were 69.5 and 75.6 years, respectively, and persons over 60 years of age accounted for 77.4% of the total deaths. Interestingly, some of these victims showed severe overhydration, with electrolyte values falling to as low as Na^+, 98; K^+, 2.1; Ca^{2+}, 1.52; and Cl^-, 97.4 mMol/L.

During five heat wave years (1952 to 1955 and 1966), the average number of heatstroke or heat exhaustion deaths in the United States jumped from 179 to 820 deaths per year.[150,151] In addition, the number of deaths classified as heat-precipitated was more than 8000 during these heat wave years.

There appears to be a reluctance to certify heatstroke or heat exhaustion as a cause of death, and epidemiologic estimates of the incidence are probably conservative.[472] As an example, heat-related death rates in the Chicago heat wave of July 1995 were calculated by two methods: (1) classification of heat-related deaths by the Cook County Medical Examiner's Office (CCMEO), and (2) excess mortality rates based on total mortality differentials during and before the heat wave. This analysis showed underestimation of heat-related deaths in areas with high death rates, which indicates that current practice does not adequately measure actual heat-related deaths. Nevertheless, it may still be practical to use those numbers as an indicator to target specific communities for prevention and relief efforts.[469]

Hyperthermia commonly occurs in the presence of numerous and varied host factors.[28,256] These include many that affect thermoregulation by altering heat loss mechanisms (e.g., lack of acclimatization, fatigue, lack of sleep, dehydration, skin disorders), whereas others contribute to heat production (e.g., obesity, lack of physical fitness, dehydration, febrile illness, sustained exercise).

The most lethal heat wave in modern times occurred in Europe, from August 4 to 12, 2003, with ambient temperatures exceeding 35°C (95°F) and sometimes 40°C (104°F). More than 10,000 heatstroke deaths occurred in France and additional thousands in other countries.[39,134] Although the exact number is unknown, the French undertaker's group Pompes Funèbres Générales claimed 13,600 excess deaths in August, and other sources claimed 14,800 heatstroke deaths.[127] There were so many dead that morgues overflowed and bodies were stored in a food market's refrigerated building near Paris and in refrigerated food trucks. These deaths were from classic heatstroke and were almost exclusively among older adults (81% of deaths were of persons older than 75 years, often with concurrent illnesses). For the aged, prolonged high temperatures in the face of depressed organ function leads to electrolyte disturbances, dehydration, and overheating through failure of heat-dissipating mechanisms.

Many or most of these classic heatstroke deaths could have been avoided with good public health measures, as remedies are well known. Public health authorities failed to adequately advise the government, the public, and especially older adults that heatstroke could be avoided by simple procedures: remain in an air-conditioned environment (air-conditioned public buildings such as schools could have been made available), spend the days in a bathtub or swimming pool or frequently shower with tepid water, and drink plenty of water, rather than wine. If global temperatures rise, heat waves will increase in frequency and severity.

EXERTIONAL HEATSTROKE (EHS)

Classic heatstroke tends to be a disease of older adults, alcoholics, and the infirm. EHS is different, typically affecting young, healthy, and even euhydrated men and women during exercise. It is a syndrome involving hyperventilation and respiratory alkalosis.[63]

Physical training increases tolerance to heat, whereas a sedentary lifestyle decreases it. A sedentary lifestyle leads to a loss of muscle protein and strength and to a gain of fat. As a result, more energy is required to carry out any given physical task per gram of body mass, consequently increasing metabolic heat production. Physical training improves tolerance to heat mainly by improving efficiency of the cardiovascular system, increasing the rate of sweat production, and reducing the threshold T_c at which cooling mechanisms are activated.[21]

Within certain limits of body temperature, normal thermoregulatory mechanisms are capable of restoring T_c to 37° C (98.6° F) from either hyperthermia or hypothermia. However, if T_c rises to approximately 42° C (107.6° F), metabolic pathways may be so altered that inappropriate physiologic responses occur. Vascular collapse, shock, and death can follow unless countermeasures such as cooling and volume therapy are initiated. It is not certain whether there is a single critical intracellular derangement occurring at 42° C (107.6° F) that ultimately leads to activation of many harmful metabolic pathways and heatstroke death, or if several harmful pathways become established independently at about the same time in response to the given T_c. EHS often affects fit and highly motivated individuals participating in sporting events or undergoing performance tests, such as military recruits, firefighters, and police officer candidates. It is expected that there will be long-term increases in exertional heat illness because of increased numbers of individuals participating in sports[228,301,302] and fitness activities, such as jogging,[225] fun-running,[259,386,423,499] bicycling,[472] and long-distance sporting events.[227,546] For example, an estimated 25 million Americans and 1 million Canadians participate in organized road races annually.[228]

EHS is not a summer illness only; it has been reported during every season, even winter. During a winter night, a father totally covered his 9-month-old infant with a blanket and a thick quilt because its crying disturbed his sleep. In the morning the child was found dead with many petechial hemorrhages in the upper chest and thoracic viscera, the blood was concentrated (indicating dehydration), and the bedclothes were extremely wet with sweat. This was listed as an accidental death resulting from exertional self-overheating in bed.[554]

Heat Storage Rate

A temperature of 40.4° C (104.7° F) represents the hyperthermia threshold above which heatstroke mortalities occur in exercised, heat-stressed rats.[104,258] The increase in an individual's heat storage, as shown by a rise in T_c, may be rapid (minutes to hours) and metabolic in origin because of overloaded heat loss mechanisms. For brief periods of intense effort, heat production may exceed 1000 kcal/hr[351]; an Olympic marathon runner produced metabolic heat in excess of 1400 kcal/hr.[18] Under conditions that limit heat dissipation, such as high humidity or wearing impermeable clothing, this rate of heat storage would cause body temperature to rise each minute approximately 0.5° C (0.9° F) and produce heatstroke within 10 to 12 minutes. Even under a moderate workload (300 kcal/hr), a person who cannot sweat effectively and thereby dissipate heat can experience a rise in T_c of 5° C/hr (9° F/hr).[351,374]

Occupations associated with increased risk of exertional heat illness include mining,[545] heavy industry,[132,140] and the military.[74,339,340,359] The yearly pilgrimage to Mecca has resulted in hundreds of fatalities from heat illness.[290] Wearing impermeable garments during physical activity leads to rapid loss of sweat, but without efficient cooling, and to consequent overloading of heat loss mechanisms and hyperthermia. These events may occur voluntarily (e.g., dieters, wrestlers trying to attain a lower weight class) or as a result of occupation (e.g., firefighters, hazardous waste handlers). Some cases of EHS have a genetic component.[399]

Failed thermoregulation can have its origin in the dysfunction of either central (hypothalamic) control or peripheral responses of heat loss mechanisms (sweating and vasodilation). A variety of drugs and toxins can produce hyperthermia and are implicated in some cases of severe heat illness.[3,4]

Individuals at Higher Risk

To preserve body temperature in its optimal range of 36.5° to 38.5° C (97.7° to 101.3° F), and to prevent an increase above 40.5° C (104.9° F), it is essential that the body delicately balance metabolic heat production, dry heat exchange, and evaporative heat dissipation. This requires highly developed and effective mechanisms of thermoregulation and temperature sensation. Heatstroke can be prevented by providing proper health education and following some simple regulations.[465,467] Prompt recognition, attention, and treatment usually result in complete recovery from heatstroke.[235]

Preventive measures include acclimation to environmental conditions, adjusting physical efforts to match physical fitness, scheduling training activities to avoid warmer hours of the day, establishing regulations to induce proper rehydration, and commanding adequate rest periods during activities. The medical team supervising an expedition or sporting or training event should play a major role in preventing exertional heat illness during strenuous physical activity. It should also have the authority to cancel any strenuous activity whenever weather conditions are adverse. Medical personnel must examine and evaluate each subject predisposed to heatstroke before exercise and, if necessary, exclude him or her from the activity. These measures were successfully implemented in the Israel Defense Forces and have been in practice there for many years. These criteria also became an official position offered by the American College of Sports Medicine.[6]

Heat-susceptible persons include those who are obese, unfit, dehydrated, or unacclimated; individuals with a previous history of heatstroke; and those who suffer from acute febrile illness, diarrhea, or chronic disturbances of the sweating mechanism. These persons should be prevented from participating in strenuous exercise, particularly in hot environments.[154] Prescription drugs or drug abuse are among the acquired factors that underlie heat intolerance and may predispose individuals to excessive heat strain by altering thermoregulatory functions physiologically or behaviorally. Potentially harmful drugs include diuretics, anticholinergics, vasodilators, antihistamines, central nervous system inhibitors, muscle relaxants, tranquilizers and sedatives, β-blockers, amphetamines, and tricyclic antidepressants.[398]

Most victims of EHS are highly motivated, young, healthy individuals who exert themselves beyond their physiologic capacity (Box 10-2). In a review of 82 cases of EHS in soldiers,[152] it was found that most cases (57%) occurred during basic training and an additional 21% occurred during screening tests for special operations forces in which motivation is a key factor. Most of the EHS cases occurred in highly motivated but relatively unfit soldiers. Overly motivated soldiers lied to the medical team about various issues that otherwise would

Box 10-2. Exertional Heatstroke in the Field

The following mid-19th century narrative, excerpted from "A Cavalry Detachment Three and a Half days without Water" by Captain J. H. T. King, Assistant Surgeon, U.S. Army Post Surgeon, Fort Concho, Texas, describes vividly the devastating impact of heat and dehydration on a military unit in Texas during the Indian Wars:

> *The next day found them still marching onwards, and the mid-day tropical heat causing great suffering. The desire for water now became uncontrollable. The most loathsome fluid would now have been accepted to moisten their swollen tongues and supply their inward craving. The salivary and mucous secretions had long been absent, their mouths and throats were so parched that they could not swallow the Government hard bread. . . .*
>
> *Vertigo and dimness of vision affected all; they had difficulty in speaking, voices weak and strange sounding, and they were troubled with deafness, appearing stupid to each other, questions having to be repeated several times before they could be understood; they were also very feeble and had a tottering gait. Many were delirious. . . . As the horses gave out they cut them open and drank their blood . . . [which] was thick and coagulated instantly on exposure; nevertheless, at the time it appeared more delicious than anything they had ever tasted.*
>
> *This horse blood quickly developed into diarrhea, passing though the bowels almost as soon as taken; their own urine which was very scanty and deep colored, they drank thankfully, first sweetening it with sugar. The inclination to urinate was absent and micturition performed with difficulty. A few drank the horses' urine, although at times it was caught in cups and given to the animals themselves. They became oppressed with dyspnea and a feeling of suffocation as though the sides of the trachea were adhering . . . prolonging the intervals between each inspiration as much as possible, . . . their lips . . . were . . . covered with a whitish, dry froth and had a ghostly, pale, lifeless appearance as though they would never be opened again. Their fingers and the palm of their hands looked shriveled and pale; some who had removed their boots suffered from swollen feet and legs.*

Box 10-3. Risk Factors for Exertional Heat Illness

FUNCTIONAL-PHYSIOLOGIC
Dehydration
Poor physical fitness
Lack of acclimatization
Heat illnesses history (controversial)
Obesity
Age
Fatigue
Pregnancy

CONDITIONAL CIRCUMSTANCES
Hot climate
External load
Inadequate rest periods
Impermeable clothing
Insulated materials
Missed meals

CONCURRENT DISEASES AND CONGENITAL ABNORMALITIES
Central nervous system lesions
Sweat gland dysfunction
Infectious diseases
Diabetes mellitus
Skin disorders
Diarrhea

DRUGS
Drug abuse
Medications
Alcohol
Caffeine

PSYCHOLOGIC STRESS
Overmotivation
Peer pressure

have prevented their participation in the activity.[465] Shapiro and Moran[465] concluded that in the young, active population, physiologic maladaptation is associated mainly with lack of acclimatization, and with dehydration, presence of infectious diseases or skin disorders, fatigue, and overweight. Although an internal "alarm system" should warn the human body to cease physical activity, during thermoregulatory maladaptation, it fails in the individual who is overmotivated and/or under peer pressure. The most common underlying factors for heat sensitivity that may lead to heatstroke are summarized in Box 10-3.

Mechanisms of temperature regulation have close functional relationships with other homeostatic mechanisms, such as the regulation of blood pressure, of body fluid volumes, electrolytes, and of acid–base balance.[465] When these are intact, the thermoregulatory system has the physiologic potential to cope appropriately with any strain resulting from exercise-induced metabolic heat production and will protect the body from the hazards involved with excessive heat accumulation and elevated body temperature. Defense from the dangerous combination of intense physical effort (high metabolic rate) and a low environmental potential to evaporate sweat (high humidity) is expressed by several body alarms that ultimately culminate in cessation of physical activity. The subjective alarm may be caused by the thermoregulatory system and expressed by elevated body temperature, or it may be caused by the cardiovascular system and manifested by elevated HR.[465] Heatstroke is a maladaptive state whereby the body temperature exceeds a temperature of 40° C (104° F) or greater, depending on the intensity of any recent exercise, as a result of the mismatch between heat production and heat dissipation.

Imbalances in fluids and electrolytes are common during hot spells, especially when salt losses in sweat are compounded by loss of appetite. Fluid and electrolyte losses resulting from illness (diarrhea or vomiting) often contribute to heat illness. In some cases, dehydration is the primary cause of death.[472]

Sleep deprivation has an insidious effect. Prolonged deprivation of sleep is fatal in animals, involving immunosuppression and a sepsis-like death. That is, sleep is essential for optimal

immune function.[40] During hyperthermia, sleep deprivation increases the T_c threshold for the onset of sweating and, in women, reduces BF_{sk}.[126,319] During moderate-intensity exercise, this delayed sweating reduced BF_{sk} in the heat, decreased total body sweat rate, reduced evaporative and dry heat loss, and led to elevated T_c.[451] On the other hand, hyperthermia may disrupt the sleep-state pattern, decreasing the duration of rapid eye movement (REM) episodes, thereby degrading the quality of sleep and imposing a psychological stress.[185]

Combinations of factors that create a threat may be subtle. The presence of air conditioning in the home, workplace, and transportation may decrease the risk for development of classic heatstroke. However, by diminishing the state of heat acclimatization and respect for the environment, air conditioning may increase a person's risk for developing EHS. Intense and prolonged exposure during weekends and holidays accounts for many cases of sunburn and heat illness.

Military Training

From 1942 to 1944, there were at least 198 deaths from heat illness during military training in the United States.[455] Gardner and colleagues[188] studied exertional heat illnesses in Marine recruits at Parris Island. During a 12-week basic training, the rate of exertional heat illness depended on fitness. These rates correlated with body mass index (BMI) and the time to complete a $1^1/_2$-mile run during the first week of the training. Recruits with a BMI greater than 22 kg/m^2 and a run-time greater than 12 minutes had eight times the risk of developing heat illness than those with lower values.[188] In this study of 217,000 Marine recruits (90% male, 80% age 18 to 20 years), there were 1454 cases of exertional heat illness (89% in men and 11% in women),[188] with a peak summer rate of 2% of recruits. The frequency of reported cases closely correlated with daily heat load (a combination of Tw, T_{db}, and solar radiation) but surprisingly did not correlate well with T_{amb}. Most cases occurred between 7:00 and 9:00 AM, during training that would have been restricted at elevated T_{amb} later in the day. During those hours, the rate of heat casualties increased substantially as WBGT increased, beginning at levels as low as 18.3° C (65° F). At a WBGT of 23.9° to 26.7° C (75° to 80° F), heat illness rates increased 26-fold over baseline rates at T_{amb} less than 18.3° C (65° F), and 39-fold over those on the day before the exercise. Exposure to WBGT of greater than 80° F (26.7° C) was infrequent (25%) among the early morning cases at the time of illness. However, the fascinating observation was made that on the day before developing heat illness, high T_{amb} was common (87%), suggesting a prolonged biochemical physiologic effect of heat exposure.[277] Regardless of the current temperature, if the prior day was cool, the frequency of heat cases was low; if the prior day was hot, the frequency was high. Because the recruits were forced to drink large amounts of water, enough to keep them urinating during the night, the elevated rate of heat casualties could not have been related to accumulated dehydration from the day before. The presence of a high environmental temperature caused an unspecified physiologic predisposition to heatstroke that lasted for at least a day.

Thus, adhering to the published guidelines based on WBGT for physical activity does not necessarily prevent heat illnesses. A better index must be developed. High rates of metabolic heat production (e.g., running) combined with conditions that limit evaporative heat loss (e.g., high relative humidity, impermeable garments) present a significant risk regardless of T_{db}. Furthermore, prevention is complicated by the observation that weather conditions on the previous day contribute to the current risk for heatstroke.

Seasonal Effects

Not unexpectedly, the incidence of heat illness was found to vary with the season. Rates for females were higher during the early hot season (May), and male rates were higher in the late hot season (September). Although May was cooler than September, there was a higher overall rate in May, probably reflecting less acclimatization of subjects in the spring. Comparing heat illness rates during days of the week, only 1.1% of casualties occurred on Sundays, reflecting decreased physical activity. In another study, obesity increased the risk of heat disorders in soldiers training in a hot and humid environment.[95]

In the IDF, 82 cases (of 150 suspected) were positively diagnosed as EHS from 1988 to 1996.[152] More than 50% of these cases occurred during the first 6 months in service, mainly in summer (June to September), but 30% of the cases occurred during the spring, and some cases occurred during the winter season. EHS was not related to time of day. Many cases occurred during the night or early morning, even under mild heat load. Interestingly, evaluated by the heat load index,[484] 40% of the cases occurred during very short activities, whereas 60% occurred within the first 2 hours of exercise. The mechanism of temperature regulation is normally highly efficient and the potential core-to-periphery conductance and sweat rates in the 82 heatstroke victims were higher than required. Therefore, EHS is more likely to occur as a consequence of disturbances in the homeostatic mechanisms aggravated by one or more predisposing factors (see Box 10-3).[465]

Although heatstroke may be due to accident, lack of knowledge, poor judgment, or neglect, it is still a preventable illness. Therefore, cases of fatal heatstroke should be investigated for criminal intent. For example, in the IDF, almost all cases of EHS occurred when regulations were not strictly followed.[152] In a most unusual incident, a herbalist in Australia was convicted of manslaughter[354] because he "treated" a young boy by immersing him for 40 minutes in a heap of fermenting horse manure; the boy died of heatstroke. Educational efforts by both the media and health care community aimed at parents of small children during heat waves should be made with the same enthusiasm as public service announcements of windchill factors during subzero temperatures. If participants develop heat injuries during sporting events that occur in hot weather and outside the heat stress guidelines, coaches and organizers of the event should be held legally responsible. The military has long recognized the preventable nature of heat injuries that result from intense physical activity in hot weather. A passage in the June 10, 1865, *Lancet* reads: "Commanding Officers of volunteers are very apt to err in this particular; and the spirit of their men is such that they shrink from complaint and persevere in efforts which may easily, under a burning sun, become dangerous to life."

Sports

EHS is a true sports emergency. There are several reports each year of heat illnesses, some with fatal results, especially in competitive games. Incidence and mortality statistics for athletes are difficult to ascertain, but some have been reported. Heat illness is the third leading cause of death of U.S. high school athletes.[322] Undoubtedly, the incidence of unreported nonfatal heatstroke

is higher. From 1990 to 1995, there were 84 reported deaths resulting from heatstroke in athletes participating in American football.[507] In 1995, five American high school football players were reported to have died of heatstroke.[447] Interior linemen are at higher risk because of their large muscle bulk and relatively small skin surface area. The uniform worn by football players covers almost the entire body and adds to heat load. In a 10-year study, 10% of 136 deaths among American high school and college athletes were caused by EHS.[517] In the Falmouth, Massachusetts, road race, there are between 10 and 15 heatstroke victims every year.[447] Cyclists and tennis players, in particular, tend to have great problems with dehydration—the cyclists because their sweat evaporates rapidly as they ride, causing them to underestimate fluid loss.[236] In tennis, the duration of the game varies considerably and drinking is permitted only during changeovers. The players' sweat rates can reach 2 L/hr (2.1 qt/hr), which presents a practical challenge for adequate fluid volume replacement.[45] In soccer, 4.5% of injuries in 480 games were related to heat illnesses.[292,294] Marathon runners thermoregulate at elevated core temperature (about 40°C [104°F]) during a run, and approximately 0.6% of the runners suffer from various heat illnesses.[412]

Training Injuries

Although the rate of heat illnesses is greater in summer than in winter, development of musculoskeletal and other injuries is a less obvious consequence of seasonal variations in climate. During basic military training in summer, the incidence of musculoskeletal injuries severe enough to cause a time loss is higher than in fall.[299] That is, the injury rate increases with temperature, and at a T_{amb} of 33.3°C (92°F) it is 2.6 times as high as at a T_{amb} of 16.7°C (61°F) for men and 1.8 times as high for women. This effect is also seen in elite athletes[309] and in British professional rugby players, with summer seasons showing almost double the injury rate of winter (696.8/1000 hours and 363.6/1000 hours, respectively).[410] In several studies, women had 1.5 to 3 times the injury rate of men.[300] Other risk factors for injury are poor physical fitness, cigarette smoking, physical inactivity, longer running distances, and use of old running shoes (those 6 months to 1 year old more than doubled the injury rate).[187] Additional risk factors for injury include older age, high-arched feet, and being knock-kneed.[299]

Heat Strain Indexes

How can one best quantify dyshomeostasis and body injury, or the inability to maintain T_c at the level prescribed by the thermoregulatory center,[216] resulting from exercise and heat? Heat strain is not simply a matter of T_c alone, because heatstroke and other heat illnesses can occur at T_c less than 41°C (105.8°F). During the past century, environmental parameters and physiologic variables were combined to develop a unified heat strain index. Although more than 20 heat strain indexes have been described, none is accepted as a universal physiologic strain index. The main reasons for their lack of general application are the number and complexity of the interactions among recognized determining factors and the limitations of their validity.

The existing indexes can be divided into two main categories: (1) "effective" temperature (ET) scales,[252] based on meteorologic parameters only (e.g., ambient temperature, Tw, and T_g) in which the WBGT index is derived,[549] and (2) "rational" heat scales that comprise a combination of environmental and physiologic parameters (e.g., radiative and convective heat transfer, evaporative capacity of the environment, and rate of metabolic heat production). ET indexes have been widely applied to both assess and predict heat strain, but they cannot accurately account for different levels of metabolic rate and different types of clothing (e.g., encapsulating).[282,409]

In 1937, the operative temperature index was developed,[542] which combined rates of metabolic heat production (M), heat transfer between the body and the environment (Hr + c) and evaporative capacity of the environment to dissipate heat (Emax).[35] Its best known modification is the heat strain index (HSI), which relates total evaporation required (Ereq, the sum of M and Hr + c) to attain Emax. It is widely accepted because it combines environmental variables and body activity. However, in certain situations, the heat strain was seriously in error and further modifications have not been fully satisfactory.[243] HSIs based on physiologic parameters were also suggested but found unsatisfactory.[320] In sum, although many HSIs were developed, some simple to apply and others highly complex, all were found to be valid only under certain specific conditions.

Moran and coworkers[370] introduced a new physiologic strain index (PSI) based on adding with equal weighting the individual strains of T_{re} and heart rate (HR), which represent the combined strains of thermoregulatory and cardiovascular systems. Each strain system was scaled 0 to 5. Thus, the PSI is scaled 0 to 10 and can be calculated continuously on-line or during data analysis. The PSI, unlike some of the other indexes, can be applied at any time that T_c and HR are measured, including during rest or recovery periods.[370] In a series of studies, PSI successfully evaluated the strains generated by different clothing ensembles, different climatic conditions, different levels of hydration, and different exercise intensities. The evaluations compared the sexes during heat stress, and different age groups during acclimatization, during acute exercise heat stress, and during environmental stress.[364,366,369] Furthermore, this index, when adjusted for animal values, successfully rated and correctly discriminated between trained and acclimated rats exposed to exercise heat stress.[363]

The PSI differs from previous indexes. It is easier to interpret and use than other indexes available, and it can be used to assess rest and recovery periods. The PSI overcomes the shortcomings of previously described indexes and can be used over a wide range of conditions.

Heat strain is well known to correlate with environmental heat load and metabolic heat production, which result in higher values of T_{re}, HR, and T_{sk} and a reduced ability to maintain exercise. Various types of clothing with different degrees of water permeation create microclimates differing from that of the environment, and the impermeable garments lead to higher strains. The combination of different climates, impermeable garments, and hydration levels during different exercise intensities is a challenge for assessment of the individual physiologic strain. However, in two studies, when different HSIs were applied to quantify heat strain, PSI was successful at all levels of heat exposure, whereas the success of the other indexes was limited.[370] The applicability of PSI was further successfully shown for various sweat rates and relative exercise intensities in different climates, and for exercise heat stress related to sex at different combinations of exercise intensity and climate.[364]

Gender

In many experiments, women exposed to acute heat stress and exercise thermoregulated less effectively than did men, resulting in the general conclusion that women are less heat tolerant than are men.[356,457,466] However, in most studies, women generally had lower cardiorespiratory fitness, higher percentage of body fat, lower body weight, lower body surface area, and higher surface-area-to-mass ratio than men.[333,405,408] Those physiologic differences are probably not intrinsically specific to the gender but result from inequalities in fitness and acclimatization attributable to differences in lifestyle.[390] When such variables were controlled, most of the physiologic differences were narrowed, although mean sweat rate (msw) still remained lower for women.[9,196,356,548] Stephenson and Kolka[490] suggested that the apparent physiologic differences in msw were based on comparing unmatched genders, mainly aerobically fit men to relatively unfit women. They argued that most of the studies comparing responses of men and women were not controlled for menstrual cycle phase and thus were limited in their conclusions. Furthermore, Sawka and coworkers[453] concluded that when men and women were matched for aerobic fitness, they had similar heat tolerances and rises in body temperature during exercise in the heat. For example, acclimatized men and women have comparable responses to rest and exercise in a desert environment.[528] However, hormonal fluctuations of estrogen and progesterone concentrations associated with the menstrual cycle may alter women's performances and tolerance to exertional heat stress.[333] Moran and colleagues[363] applied the PSI in three climates to evaluate the strain on men and women who were matched for maximal aerobic power ($\dot{V}O_2max$) and intensity for 60 minutes. All subjects underwent a matrix of nine experimental combinations of three different exercises.[25,272,453] The PSI confirmed previous studies that when men and women were matched for $\dot{V}O_2max$ and selected physiologic parameters, there is no difference in strain between the two groups.

For middle-aged women, the effects of hormone replacement therapies on the thermoregulatory system were studied.[69,70,196,503] The luteal phase, with its increased progesterone-to-estrogen ratio, was associated with an elevated T_c, whereas elevated levels of unopposed estrogen were associated with a lower T_c both at rest and during exercise heat stress.[503]

In summary, although mean physiologic responses to exercise heat stress suggest poorer thermoregulation for women, this is not necessarily an intrinsic sex difference but reflects a combination of social factors serving to reduce fitness in women.

Aging

In general, middle-aged and older men and women are less tolerant of heat and exercise, suffering more physiologic strain than do younger individuals.[251,522] They developed higher HR, T_{sk}, and T_c and lower msw than younger men and women during exercise heat stress. However, it is uncertain whether this is related to age per se or to other factors, such as disease states, decreased physical activity, and lowered aerobic fitness.[403]

A subgroup of middle-aged men who were "habitually active" displayed the same strains to acute exercise heat tolerance and acclimated to heat at about the same rate and degree as when they were younger.[429] Some studies on aging emphasize the importance of aerobic fitness and physical characteristics, such as percent body fat and body weight in maintaining work heat tolerance. In one study, old and young men and women were matched for $\dot{V}O_2max$, surface area, and surface-area-to-mass ratio, but they differed in age by as much as 35 years.[283] During 75 minutes of light exercise at 37°C (98.6°F) and 60% RH, all groups showed the same degree of physiologic strain. In 1988, Pandolf and coworkers[404] compared responses to acute heat tolerance between young and middle-aged men who were matched for $\dot{V}O_2max$ and morphologic factors. The middle-aged men's tolerance times were actually half an hour longer than the younger men's during the first day of acclimation (49°C [120.2°F], 20% RH), and they were also at a thermoregulatory advantage during the few days of heat acclimation, although both groups eventually acclimated to the same absolute degree. However, in these studies the middle-aged men were more chronically active than were the younger men before heat acclimation. Under different environmental conditions, such as 30°C (86°F) with 80% RH, and 40°C (104°F) with 20% RH, no differences were seen between the two groups.[481] In summary, when properly matched subjects are studied, heat stress of middle-aged men resulted in either the same or reduced physiologic strain as that of younger men.

CELLULAR HEATSTROKE

Subcellular Disruption

Heating alters subcellular structures in many cell types, including detachment of cortical microfilaments from the plasma membrane,[108,145] collapse of the cytoskeleton, swelling of the mitochondria and endoplasmic reticulum,[52] and disaggregation of polyribosomes and nucleoli.[85,355] Furthermore, heating increases membrane fluidity, grossly distorting plasma membranes and forming bulges known as blebs.[31,120,417] Blebs alter membrane function,[13] increasing membrane permeability[511] and leading to solute leakage.[419,439] Such changes are not necessarily lethal; up to a point, the bleb formation may be adaptive, increasing cell survival.[53,273] Red blood cells (RBCs) also undergo rearrangements in their cytoskeleton with heat, but rather than forming blebs at elevated temperatures, the cells change shape from biconcave discs to spheroids.[219] These enlarged spherical cells are much less efficient at gas exchange than are normal red blood cells and probably contribute to reduced P_{O_2} in the tissues at elevated temperatures. Spheroid-shaped RBCs have been found in athletes during long-distance running and may contribute to their physical collapse during exercise.[219]

Apoptosis

Cells are destroyed within the body by the processes of apoptosis and necrosis. Apoptosis occurs because certain individual cells within the body are genetically programmed to die. In this process, cells are broken into small vesicles containing condensed chromatin surrounded by intact cell membranes, and they are phagocytosed by neighboring cells. During necrosis, a variety of factors, including inflammation, cause swelling, destruction of the plasma membrane, and spewing of cell contents into the environment.[135] Apoptosis may be induced by a variety of stressors, including hyperthermia, aging, and the presence of certain toxins (*Pseudomonas* endotoxin, diphtheria toxin, and ricin).[143] A few minutes' exposure to temperatures of 41.5° to 42°C (106.7° to 107.6°F) triggers apoptosis in both cultured mammalian cells and experimental animals by activating Jun N-terminal kinase (JNK).[110,443] The organs producing

the greatest number of apoptotic cells caused by whole-body hyperthermia are the thymus, spleen, lymph nodes, and mucosa of the small intestine. The heat-shock protein HSP-70 (see later) inhibits this activation and contributes to acquired thermotolerance in mammalian cells.[175]

Heat-Induced Changes in Intracellular Ions

Ions do not readily cross lipid bilayers despite their large concentration gradients across plasma membranes. In general, they require specialized channels or carriers to do so. Membrane channels are proteins that contain hydrophilic pores that penetrate the lipid bilayer, permitting diffusion of specific ions down their electrochemical gradients to enter or leave cells.[543] Cotransporters are fundamentally different types of pathways that move ions across cell membranes up their electrochemical gradients by coupling energy stored in preformed gradients, usually the Na^+ gradient.[543]

Potassium ions enter cells by at least two pumping mechanisms, the membrane Na^+,K^+-ATPase pump, and the ouabain-resistant pump.[220,552] K^+ exits cells through several types of membrane channels, including voltage-gated, Ca^{++}-gated, ATP-gated, and arachidonic acid–gated K^+ channels, and K^+-leak channels.[294,383,489] Although the rate of K^+ transport by each pathway varies with temperature, the overall resultant flux during heating is a net loss of intracellular K^+ and a primary rise in plasma K^+.[177,180,440,491]

Hyperthermia increases the rate of Na^+ influx, leading to a net rise in intracellular sodium (Na_i) that is prolonged and not rapidly reversed by cooling. At a sufficiently high temperature, the Na transport mechanisms become denatured and, because of increased leakiness of the plasma membrane and the inward-directed Na^+ concentration gradient, Na_i rises progressively until death.[183] Acidifying cells leads to an explosive influx of Na^+ on heating. Hyperthermia leads to at least mild cytoplasmic acidification,[538] and therefore the combined stresses of heating and acidification lead to increased intracellular Na^+.

Moderate heat activates a plasma membrane–bound Ca^{2+}-ATPase pump to temporarily lower intracellular calcium (Ca_i). However, severe heating causes a net rise in Ca^{2+} through activation of at least two different transport systems, and the elevated concentration of free cytoplasmic Ca^{2+} is expected to alter a number of metabolic pathways.[307] Depending on the temperature, Ca^{2+} enters the cell from either the extracellular fluid by means of the reversed mode of the plasma membrane sodium–calcium exchanger or from intracellular Ca^{2+} stores, although it is not clear when either is activated, or in what sequence.

Some ionic changes in mammalian cells induced by heating (e.g., Ca^{2+} and Na^+ changes) are mediated by inhibition of the Na-H exchanger, activation of Na^+,K^+-ATPase, and changes of membrane conductance for ions.[478] See Gaffin[177] for a review of these mechanisms.

Although heating acts directly on the rates of fundamental cell processes, heat also has indirect effects by causing release of a variety of stress hormones. These hormones, singly or in combination, further alter the activities of virtually all ionic pathways. For example, catecholamines, especially in the presence of insulin, strongly stimulate the muscle membrane Na^+,K^+-ATPase pump, increasing intracellular K^+ concentration (K_i),[98] which hyperpolarizes the resting membrane potential in excitable cells and increases the magnitude of subsequent action potentials.[164] This stimulating effect of hormones on physical performance may be so powerful that, for a time, it masks a primary decrementing effect of the heat-induced hyperkalemia on performance.

In addition to the effect of heat on pathway activities, the rise in activity of the various ion pumps provides more metabolic heat, which, near the limit of sweat secretion, further elevates body temperature.

Increased membrane permeability from hyperthermia leads to rises in circulating enzymes. Creatine kinase is the first elevated enzyme detected at a T_c as low as 39.5°C (103.1°F) in monkeys (S. Constable and S. L. Gaffin, 1995, unpublished observations) and 42.5°C (108.5°F) in rats, followed by lactate dehydrogenase.[341]

Stress Pathways

Early unicellular forms of life were preyed on by their neighbors and, over geologic time, developed an immune system for protection. This nonspecific immunity is basic to all phyla today. One of the earliest mechanisms of host defense was production of active oxygen species, including free radicals, that destroy the invader's cytoplasm. In modern mammalian species, specialized inflammatory cells, such as macrophages and natural killer (NK) cells, are enriched in the enzymes that allow chemical pathways to produce toxic free radicals. They also contain additional pathways that allow the inflammatory cells to recognize invaders and to penetrate the host's tight epithelia, so that they may easily reach and destroy these invaders. Immune cells are activated by circulating and locally produced cytokines, such as tumor necrosis factor (TNF) and interleukin-1 (IL-1), as part of the stress reactions mediated in part by the hypothalamus. All vertebrates share common inflammatory stress reactions that are activated by stresses other than infections, such as hyperthermia, and that activate pathways that produce toxic free radicals, which may now be inappropriate and toxic to the organism[178] (see Genomics, next).

Severe hyperthermia in mammals leads to a common response pathway involving secretion of corticotrophin-releasing hormone (CRH) by parvicellular neurons of the paraventricular nucleus of the hypothalamus[32,514] and activation of the sympathetic nervous system (Fig. 10-5). CRH is directly transported via the hypothalamo-hypophyseal portal system to the anterior pituitary, where it has two important effects: (1) it downregulates cells that secrete immunostimulants (growth hormone, prolactin, gonadotropin releasing hormone, luteinizing hormone, follicular stimulating hormone),[16,138] and (2) it upregulates the pro-opiomelanocortin (POMC) gene in corticotropes, leading to secretion of immunosuppressors (adrenocorticotropic hormone [ACTH], β-endorphin, and α-melanocyte-stimulating hormone, in turn leading to secretion of the immunosuppressor cortisol) (see Figure 10-5). A rise in IL-1 occurs that is part of a positive feedback loop, causing a further rise in CRH and ACTH.[261,268]

These are not just theoretical predictions based on animal studies. In human subjects, circulating levels of CRH, ACTH, cortisol, and arginine vasopressin (AVP) rose during severe exercise. It appears that high-intensity exercise favors AVP release, whereas prolonged duration favors CRH release.[269]

GENOMICS

The physiologic activities of a tissue depend on the concentrations and activities of the protein components of the cell, and

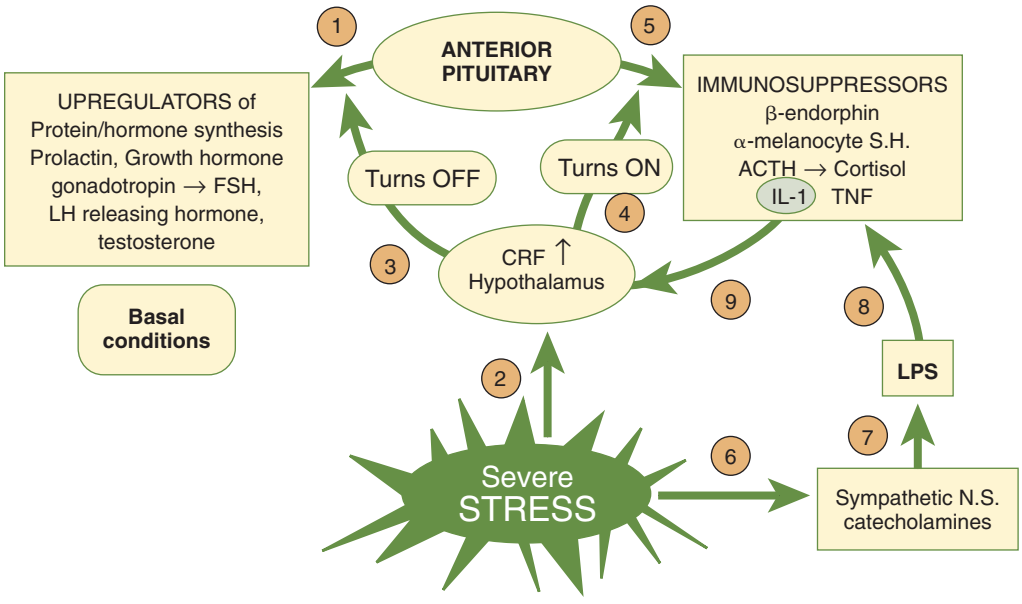


Figure 10-5. Stress and the hypothalamic-pituitary-adrenal axis. Under basal conditions, (*1*) the anterior pituitary secretes the immunostimulants prolactin, growth hormone, thyroid-stimulating hormone (TSH), and gonadotropin-releasing hormone, leading to the secretion of luteinizing hormone (LH), follicle-stimulating hormone (FSH), and testosterone. Severe stress (*2*) acts on the paraventricular nucleus in the hypothalamus, producing corticotrophin-releasing hormone (CRH) (*3*), downregulating the secretion of the immunostimulants and gonadal hormones, and (*4, 5*) upregulating the secretion of the immunosuppressors β-endorphin, α-melanocyte-stimulating hormone, and adrenocorticotropic hormone (ACTH), the latter of which upregulates the secretion of the immunosuppressor cortisol. Stress also activates (*6*) the sympathetic nervous system, and catecholamines are secreted. These further downregulate the production of immunostimulants and (*7*) may reduce splanchnic blood flow sufficiently to cause ischemia of the intestinal vascular bed, local damage to the gut wall, and the leakage of lipopolysaccharide (LPS) into the circulation. LPS in turn (*8*) causes the production of the powerful immunosuppressors tumor necrosis factor (TNF) and interleukin (IL)-1. TNF and IL-1 are also secreted within the hypothalamus in direct response to stress, leading to their participation (*9*) in a positive feedback loop, augmenting the secretion of CRF and resulting in even greater immunosuppression. NOTE: Moderate stress may have the opposite effect and increase the secretion of immunostimulants. (From Gaffin SL, Hubbar RW: Wilderness Environ Med 4:312, 1996, with permission.)

ultimately on the genes involved in the synthesis of those proteins. Important knowledge of the pathophysiology of heat and other stressors on a cell, tissue, or organism can be gained from knowledge of which particular genes are active at any given time. In the past, such information could only be gleaned one experiment at a time in a very slow, labor-intensive, and costly fashion. The development of gene chips has changed the world of medical research and diagnosis,[16] leading to the term *physiologic genomics*.

In this approach, cells or tissues are treated to allow RNA (usually) extraction. Messenger RNA (product of gene activity) is converted to complementary DNA and attached to a bound fluorescence dye for quantification.[16] The sample is then transferred to gene chips. Gene chips are slightly larger than glass microscope slides and have thousands of different individual microdots of DNA probe permanently bound to the surface in a rectangular array. Each microdot is a synthetic DNA of a short sequence of a single gene, containing a bound fluorescence dye of a different color from that of the recombinant DNA. If the sequences of the cDNA (synthesized from the extracted mRNA) and the gene probe are complementary, then the cDNA will bind tightly to the microdot and not wash off during processing. After processing and washing, the gene chip is placed on a computer-driven microfluorescence recorder, and the fluorescence intensities of the two dyes are measured for each microdot. A substantial rise in the fluorescence intensity at a single microdot of the cDNA of the treated cells is an indication that this particular gene has been upregulated in response to the treatment. If the intensity is reduced, then that gene was downregulated. The time course of rises and falls of gene activity must be examined, as well as rises or falls in response to any treatment.

Although the principles of gene chip technology are straightforward, the technical aspects have many pitfalls. Because of the very low amounts of cDNA being measured, the distribution of results is broad, so standards as to the presence of a significant rise or fall in gene activity and what constitutes a biologically meaningful change must be addressed. There is no universally accepted standard, but usually a biologically meaningful change in gene activity is defined as a 50% to 400% fall or rise in the ratio of fluorescence intensities.

Another problem relates to the large amount of data produced from the results of thousands of individual gene activities. Currently, computers must be relied on excessively to explain the results, because no one can intellectually understand, integrate, and assimilate the altered activities of hundreds to thousands of genes. Furthermore, there is no guarantee that a rise in specific mRNA activity results in the production of more active proteins; instead, proteins may not be synthesized from any mRNA products immediately but may require an additional signal.

Given the problems intrinsic to gene chip technology, Sonna and colleagues attempted to study which genes are up- or downregulated by stressors.[486] In the case of heat stress, prior to these

TABLE 10-2. Top 10 Upregulated Genes in Heat-Shocked* Human Peripheral Blood Mononuclear Cells[485]

RANK	GENE	FUNCTIONAL CLASS
1	CDF2RB	Immune
2	Immunoglobulin receptor	Immune
3	Integrin	Immune
4	HSP27/28 (α crystalline)	Chaperone
5	GOK	Cell cycle/proliferation
6	G22P	RNA/DNA structure repair
7	DDAH protein	Cell cycle/proliferation
8	Amyloid A4	Immune
9	Photolyase	RNA/DNA structure repair
10	Immunophilins	Immune

*Cells were heat shocked for 20 min at 43° C (109.4° F) and then incubated at 37° C (98.6° F) for 2 hr 40 min.

Box 10-4. Lack of Commonality of Upregulated Genes in Three Stress Models

Among three stress models (in vitro heat shock of human peripheral blood mononuclear cells, exertional heat injury in Marine recruits, and hypoxia of liver cells in tissue culture), there was a lack of commonality of upregulated genes. Only the following seven gene transcripts out of hundreds in each model were upregulated in all three models.[485–487]

- Ubiquitin C
- Dual-specificity phosphatase-1
- Colligin 2 (HSP-47)
- Phosphatidylserine receptor
- S100A2 (calcium binding protein)
- Pyruvate carboxylase
- Lysine hydroxylase (collagen synthesis)

experiments, an extensive literature search indicated that heat altered the activities of a total of 28 known genes in mammalian cells. From blood samples obtained from volunteers, peripheral blood mononuclear cells were isolated and suspended in two tubes in RPMI solution. The cells were heated for 30 min at 37° C (98.6° F). Then, one tube was placed at 43° C (109.4° F) for 20 min, then returned to 37° C for 3 hours. RNA was isolated, checked for quality and, if of acceptable purity, processed for analysis on gene chips containing more than 6000 gene probes.

The results of this in vitro heat-shock study were stunning.[486] Instead of only the 28 altered genes previously noted, 227 sequences were found to be upregulated and 168 decreased.[486] Furthermore, these genes represented all functional classes of proteins, not only the expected various heat-shock proteins, but also genes involved in apoptosis, cardiovascular function, cell growth and proliferation, blood coagulation, cytokines, cytoskeleton, DNA repair, histones, immune function, membrane transport and trafficking, metabolism, protein degradation, redox control, RNA processing and transport, signaling, transcription, translation, and several unknown functions. Table 10-2 shows the ten most quantitatively upregulated genes. These cellular genomic effects probably contribute to the whole-body physiologic responses to heat stress, such as coagulation, maintenance of blood pressure, and immune function.

The results of an in vitro experimental model may have important implications, but do similar changes in gene activities occur in humans suffering from heat illnesses? Exertional heat injury was investigated in four Marines with T_c between 39.3° and 42.5° C (102.7° and 108.5° F).[487] The results were compared with results from other Marines who underwent the same intense training in the heat but did not experience exertional heat injury. Blood samples were taken at presentation, 2 to 3 hours after cooling, and 1 to 2 days later. When peripheral blood mononuclear cells were processed by gene chip analysis with 12,600 gene probes, 361 gene sequences were considered upregulated and 331 downregulated in a time-dependent manner. The affected transcripts included sequences shown to be heat-shock responsive in the prior in vitro study (including both heat-shock proteins and non–heat-shock proteins). New sequences noted to have changes in expression included several interferon-induced sequences and nonspecific stress response genes (including ubiquitin C and dual-specificity phosphatase-1). Of the downregulated genes, two thirds do not have any known function at the current time.

Because the final stage of heatstroke involves severe hypotension and ischemic injury, it is of interest to investigate the effect of hypoxia on cell genomics. This was carried out in HepG2 liver cells rendered hypoxic by 24-hour exposure to 11% oxygen. Using a 12,600-probe gene chip, 213 gene sequences increased and 174 decreased twofold or greater.[485] These sequences were substantially different from the heat-shocked sequences.

When the upregulated gene sequences were compared among the three different stress models (in vitro heat shock of human blood cells, exertional heat injury, and hypoxic injury of cultured cells), only seven specific genes were upregulated in all of the models (Box 10-4).[485–487] However, among downregulated genes, 11% to 18% of all genes were identical. This small number of common upregulated genes seen in the three stress models is a very important observation. Most textbooks describe human physiologic responses to stress as largely involving a small number of pathways. Various levels of stress cause activation of the sympathetic nervous system and upregulation of CRH in the hypothalamus, leading to (1) downregulation of gonadotropin-releasing hormone, luteinizing hormone, follicle-stimulating hormone, growth hormone (if stress is severe and prolonged), and prolactin, (2) activation of the pro-opiomelanocortin gene and upregulation of ACTH, α-melanocyte-stimulating hormone, and β-endorphin, leading to upregulation of cortisol, and (3) activation of the sympathetic nervous system.[479,510]

Contrary to expectations, different stressors produce largely different genomic responses and very few common pathways. The apparently simple physiologic responses to stress disguise activation of a wide range of very different biochemical pathways induced by each stressor. Through the genomics approach, we can now clarify which genes are upregulated and devise experiments to understand their functions. For example, chitinase was upregulated by heat shocking human white blood cells.[486] Chitinase destroys chitin, which humans do not produce. Chitin is found in the exoskeleton of arthropods, certain parasites, and molds. Perhaps chitinase is an adaptation of the immune system to destroy fever-producing parasites and microorganisms. If so, perhaps response mechanisms of the

human immune system to elevated core temperature cannot differentiate between fever (due to invading organisms) and hyperthermia (due to excess metabolic rate, ambient temperature, or depressed cooling mechanisms). The immune system may therefore be inappropriately upregulated by hyperthermia. Heatstroke patients might one day be evaluated for the presence of chitinase in their blood cells, whether chitinase concentration correlates with tissue injury markers, and whether infectious organisms or LPS can also do so. Perhaps chitinase may become a marker for heat injury, which might indicate that immunomodulators should be investigated for their ability to reduce tissue injury and improve survival from heatstroke.

Energy-Depletion Model

During hyperthermia and continued heat stress, cellular Na^+,K^+-ATPase pumps operate at elevated rates, hydrolyzing ATP more rapidly and liberating waste heat into the body faster than normal. If the body cannot dissipate this heat through radiation, conduction, convection, and sweat evaporation, then according to the laws of thermodynamics, T_c must rise further, leading to still greater rates of the Na^+,K^+-ATPase pumps, a hazardous positive-feedback loop. The amount of energy available to a cell is limited. Therefore, at a certain elevated temperature, ATP utilized by the activated Na^+,K^+-ATPase pump can no longer be resynthesized rapidly enough to be available for normal cellular processes. The cell becomes energy-depleted. Experimental support for this concept is the presence of swollen cells (implying a slowing of ion pumps, which affect water transport) and rapid development of rigor mortis (caused by depletion of ATP) at the end stage of heatstroke pathophysiology. Persons exercising in the heat develop lethal heatstroke at lower T_c than do those at rest. This can be explained by the concept that exercise lowers ATP stores (i.e., creatine phosphate) and therefore, less is available for other important cellular processes.[128]

CARDIOVASCULAR STRAIN AND EXERCISE

Severe heat illness involves every system and affects regulatory (cardiovascular), integrative (neuroendocrine), and ultimately basic cellular systems of the body. Probably no greater strain is put on the human body than heavy physical exertion in the heat. This impact of heat stress on the cardiovascular system represents strain resulting from increased demand for cardiac output to transfer heat and water to the skin for evaporative cooling.

Severe and prolonged exercise leads to changes in the sizes of body compartments and the cardiovascular system, persisting for hours to days. For example, a group of men carrying 20-kg (44-lb) backpacks during a 110-km (68.3-mile) march under warm conditions, eating and drinking ad libitum, lost 3.4% of body weight, and plasma volume (PV) fell by 6.1%. The next day, PV dropped still further to minus 8.4%, even though they were not exercising. During the third day, PV rose to +3.7% above baseline and remained elevated 4 days after the march.[22] The hemodynamic displacement of blood to the periphery is aggravated by gravitational displacement of blood volume resulting from upright posture. In an upright human, about 70% of the blood volume is below heart level. Venous pooling of blood in the skin and in the great veins below the level of the heart leads to reduced venous return to the heart and consequent reduced cardiac filling.[240] If exercising skeletal muscle then vasodilates to supply the increased demands for blood flow in support of muscle metabolism, the competing demands for blood flow between vascular beds translate into a major regulatory problem.[436]

The potential conductance of the vasculature (skin, 8 L/min [8.45 qt/min]; viscera, 3 L/min [3.2 qt/min]; and muscle, 65 to 70 L/min [74 qt/min]) is enormous and far exceeds the pumping capacity of the normal human heart (about 22 L/min).[266,437] Because the combined blood flow requirements of these vascular beds cannot be met, an inherent competition takes place between the mechanisms that maintain blood pressure and those that maintain blood flow to support metabolism and thermoregulation.[257]

The existence of a variety of types of heat illnesses suggests (1) physiologic strain resulting in homeostatic failure produces heat illness, (2) a certain biologic variability is expressed in the response to heat and exercise, (3) hemodynamic stability often takes precedence over thermoregulation, (4) volitional behavior, expressed as exercise performance, is often maintained even as the risk of heat injury increases, and most important (5) the onset and increase in hyperthermia are not painful.[256] Not surprisingly, the physiologic threat to homeostasis is worsened when accompanied by a fluid–electrolyte imbalance.[14,255] Treatment is then directed at the major sources of homeostatic failure: cease the physical activity, lie down, cool down, and rehydrate. Sometimes the cause of collapse or the other symptoms involved (such as headache, nausea, vomiting, vertigo) is obvious; other times it is not readily appreciated.

Sweating

Although human eccrine sweat glands generally behave physiologically and pharmacologically as if under parasympathetic or cholinergic control, they also respond to adrenergic stimulation.[449] There are 2 to 3 million sweat glands in the skin distributed with decreasing density from the palms and soles, to head, to trunk, and to the extremities, with an average density of about 100 to 200 per square centimeter (645 to 1290 per square inch). One gram of sweat glands can secrete up to 250 g of sweat per day. Eccrine sweat is always hypotonic, it contains variable concentrations of sodium, and it is generally 99.5% water by weight.[153] Nevertheless, the remaining 0.5% of sweat solids is important, as 1 L of 0.5% sodium chloride contains 5 g of salt, a potential cause of serious salt depletion, if sweating persists over several hours.

Sweat secretion and rate depend on activation of the sweat center in the hypothalamus, which discharges over cholinergic fibers of the sympathetic nervous system. Rising blood temperature increases both the number and rate of sweat glands responding, until the body reaches thermal equilibrium or maximal sweat rate.[198] Sweat rates are reduced by dehydration and by hyperosmolality,[454] with hyperosmolality more effective (Fig. 10-6).

One of the highest sweat rates ever recorded in a human was in a world-class Olympic runner.[18] He produced sweat at a rate of 3.71 L/hr (3.9 qt/hr) after 19 days of heat acclimation training. Because the maximal rate of gastric emptying (1.2 to 1.5 L/hr [1.3 to 1.6 qt/hr]) is much less than maximal sweat rate, rehydration cannot keep pace with sweat losses under those conditions,[19] and an athlete faces significant risk of dehydration despite frequent drinking.[17,20,257] Because rates of gastric emptying can exceed 1 L/hr, a sweat rate of 1 L/hr appears sustainable without significant dehydration.

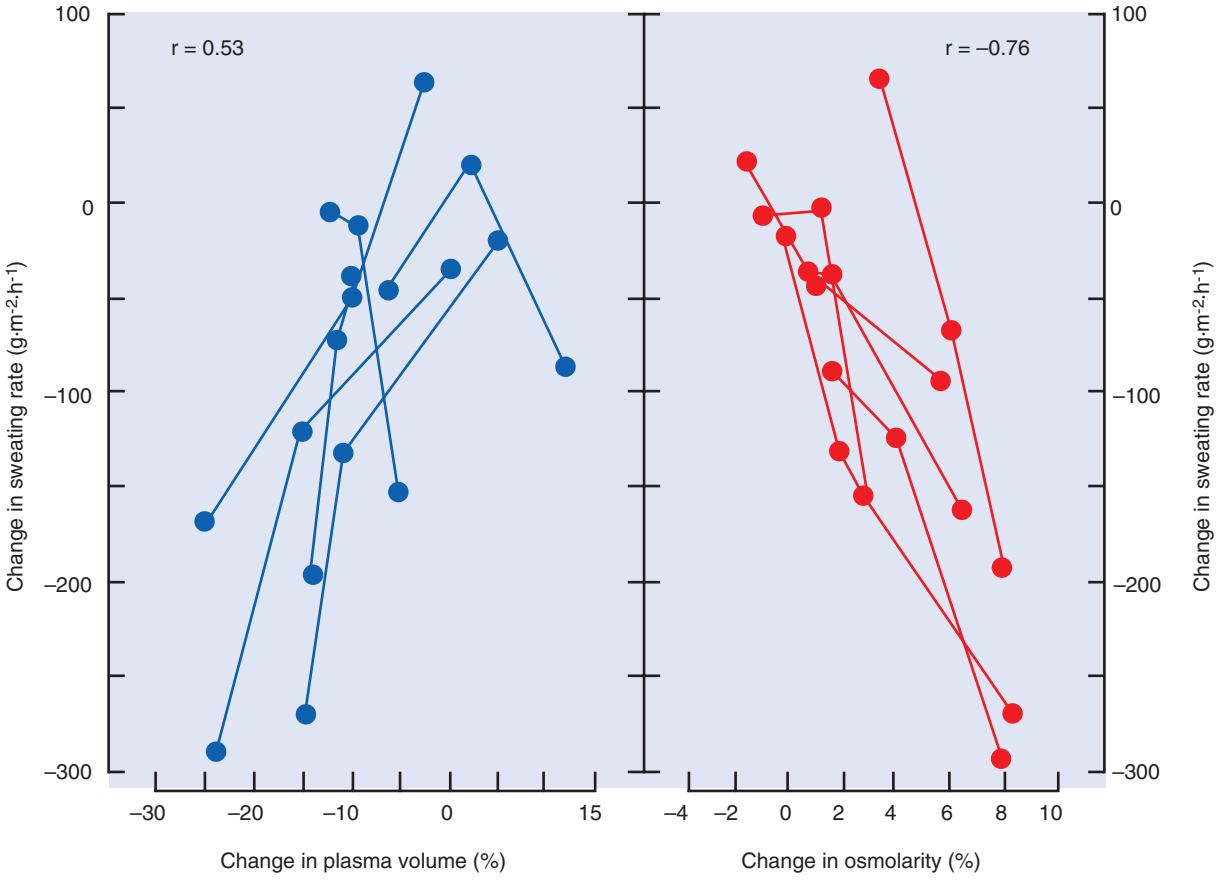


Figure 10-6. Effect of reduced plasma volume or increased osmolality on sweat rates in six individuals. (Modified from Sawka MN, Young AJ, Francesconi RP, et al: J Appl Physiol 59:1394–1401, 1985.)

Fluid and Electrolyte Imbalance

Heatstroke Model

Determining the time courses of changes in various physiologic and biochemical parameters caused by heatstroke is difficult or impossible to study in humans for ethical reasons. Use of rodents precludes determining many parameters simultaneously because they lack sufficient blood volume. Miniswine have been increasingly used as models because of their practical size and physiologic similarities to humans. When anesthetized miniswine were passively heated to a T_{amb} of 43° C (109.4° F), HR and mean arterial pressure (MAP) rose only slightly until T_c reached 42° C (107.6° F) (Fig. 10-7A). At this T_c, HR rose to a peak and rapidly fell until death, whereas MAP gradually fell until death but with a shoulder corresponding to the HR peak. The respiratory rate rose, peaking at approximately 42° C (107.6° F), and then continuously fell until death (see Figure 10-7B). Because of increased ventilation, P_{CO_2} fell until shortly before death, but then it rose to very high levels immediately before death. As ventilation increased, plasma pH rose, but as ventilation fell, pH also gradually fell and CO_2 rose. Renal function was stable until 41° to 42° C (105.8° to 107.6° F), the same T_c that reduced MAP (see Figure 10-7C). Serum K^+ continuously rose with T_c (see Figure 10-7D).

A schematic model of normal thermoregulation and pathophysiology of heat stress and heatstroke (based on most of the preceding considerations) is shown in Figure 10-8. Core temperature has been divided into two regions to clarify physiologic mechanisms.

Hydration

"Water discipline," meaning voluntary fluid deprivation, is an attempt to reduce the body's water requirements during activities in the desert or jungle. This is an outdated, incorrect, and dangerous concept. Voluntarily or involuntarily withholding water from persons in the heat in the face of severe thirst leads to unnecessary deaths and permanent neurologic damage in the survivors of heatstroke. Frequent spitting and withholding of water are often done by collegiate wrestlers to qualify for a lower weight class. This behavior must be regarded as a serious risk for developing heat illnesses.[407] For further details, see Chapter 64.

Mental Performance

An athlete who is hypohydrated when commencing strenuous physical exercise in the heat will be at a disadvantage compared with a euhydrated person. Overheating occurs earlier, physical and mental performance decline sooner, and there is more

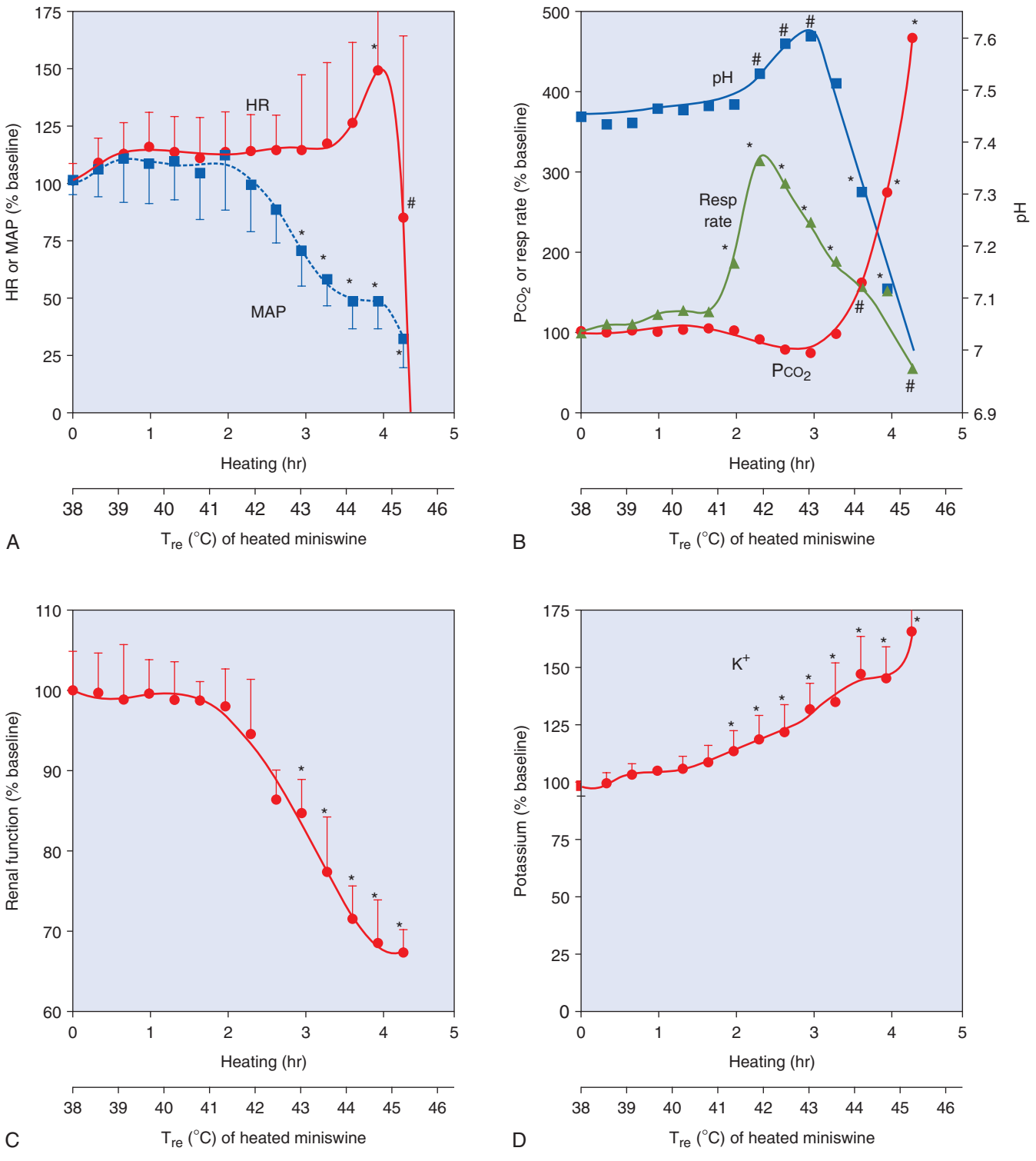


Figure 10-7. Experimental heatstroke in miniswine. **A,** Effect of hyperthermia on mean arterial pressure (MAP) and heart rate (HR) in anesthetized miniswine. After the 1-hour baseline, environmental temperature was raised to 42° to 43° C (107.6° to 109.4° F). **B,** Effect of hyperthermia on respiratory rate (resp rate), P_{CO_2}, and arterial pH in anesthetized miniswine. Changes from baseline levels. **C,** Effect of hyperthermia on renal function. Percent baseline level. **D,** Effect of hyperthermia on arterial potassium, calcium, and inorganic phosphate in anesthetized miniswine. *, $P < .05$ compared with controls; #, $P < .05$ compared with own baseline values. (From Gaffin SL, Gentile B, Koratich M, et al: J Therm Biol 23:341–352, 1998, with permission.)

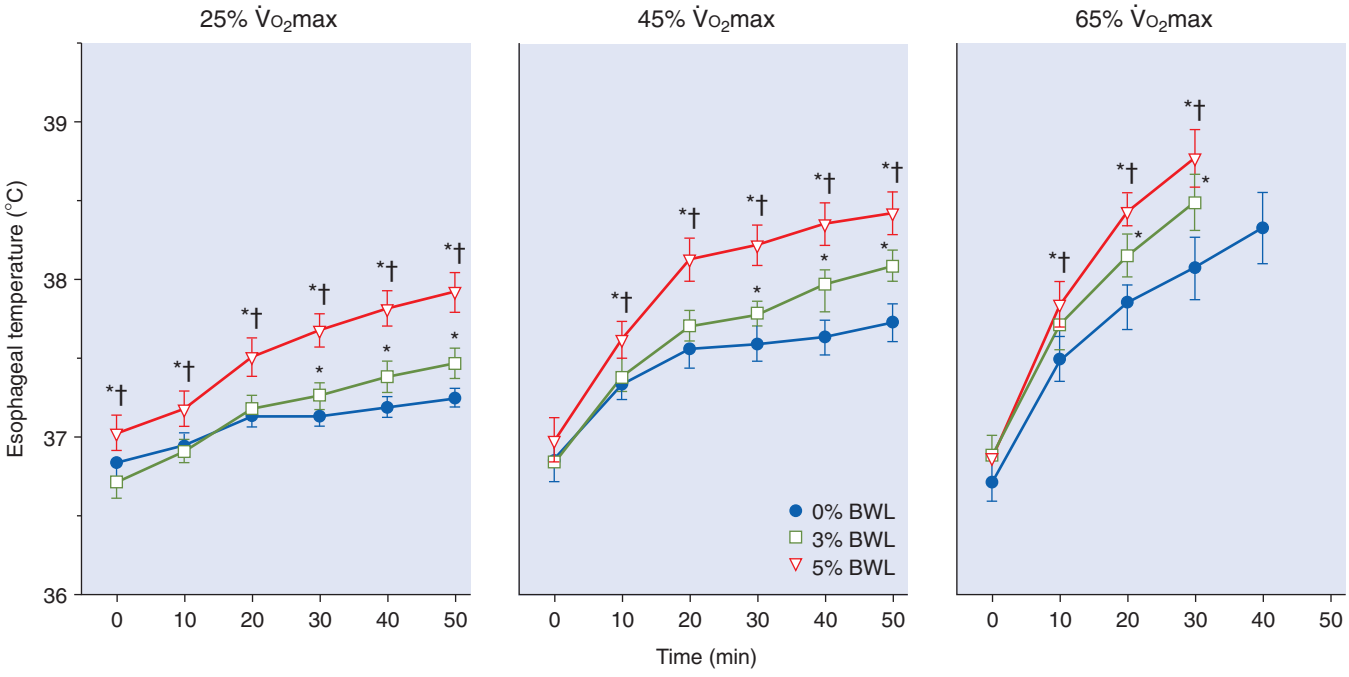


Figure 10-8. The effect of hydration level and exercise intensity on esophageal temperature. (Data from Montain SJ, Latzka WA, Sawka MN: J Appl Physiol 79:1434, 1995.)

susceptibility to hemorrhage and trauma. Furthermore, heat stress slows reaction and decision times. Routine tasks are done more slowly, and errors of omission are more common.[233] Heat stress degrades attention to detail, concentration, and short-term memory.

With normal nutrition, ingesting commercial electrolyte- or carbohydrate-containing beverages offers no advantage over water in maintaining PV or electrolyte concentration or in improving intestinal absorption. However, under conditions of caloric restriction or repeated days of sustained sweat losses, there might be a benefit to consuming an electrolyte-containing beverage. For athletes, use of glucose polymer sorbitol may be considered. For the vast majority of individuals, however, the primary advantage of using electrolyte- or carbohydrate-containing drinks appears to be their flavor, which enhances voluntary consumption of fluids. This factor could become important if regulated intake is impossible.

Dehydration, as a dynamic change from euhydration to hypohydration, reduces or compromises the physiologic advantages of any physical training or heat acclimation. Involuntary dehydration can produce serious water deficits, such as are found in persons cast adrift on tropical seas or stranded in the desert without water. Survival under these conditions requires knowledge of the tradeoffs between the benefits of increased physical activity to obtain water and the consequences of increased sweat and dehydration. Adolph and colleagues[1] described both the symptoms and the expected dehydration rates under these circumstances.

The minimal unavoidable daily water loss is approximately 1.5 L (1.6 qt) or about 2% of body weight as body water.[347] Although hard work in the heat can elicit this sweat loss in 1 hour (1.5 L/hr), resting in the shade can reduce sweat losses to minimum values (50 to 300 mL/hr at air temperatures from 26.7° to 37.8° C [80° to 100° F]). Assuming that circulatory shock occurs when water loss reaches 12% body weight loss as water loss, then survival time could vary between 1 and 5 days. In the desert, for every 20 miles walked at night, one should carry 1 gallon of water and, during the day, 2 gallons.[1]

Normal oxidation of carbohydrates produces a small amount of metabolic water that may have survival value. Approximately 500 mL (0.53 qt) may be produced per day, which represents 30% of the daily obligate water loss at rest. However, converted to running time in the heat, this represents only about 20 minutes.

Thirst and Voluntary Dehydration

The sensation of thirst does not become prominent until osmotic dehydration exceeds the renal capacity to deal with it physiologically.[255] Therefore, inadequate fluid intake can also occur where drinking is possible. That is, the delay in thirst is a manifestation of the body's osmotic control. Although there is wide variation in the individual thirst threshold, it has an average value close to the upper normal value of 295 mOsm/kg H_2O (normal range, 280 to 295 mOsm/kg H_2O).[345,426,428]

Dehydration is the most potent stimulator of both thirst and antidiuretic hormone (ADH).[11] For any one individual, the plasma vasopressin response (ADH release) is linearly related to plasma osmolality[428] and therefore to plasma sodium. For example, at an osmolality of 280 mOsm/kg H_2O, ADH release is almost completely inhibited and osmolality of the urine is minimal (100 mOsm/kg H_2O). As dehydration occurs, plasma Na rises, causing a rise in plasma ADH concentration (0.5 to 5 pg/mL). ADH causes conservation of water by causing insertion of the aquaporin water pore into the basolateral ductal

membranes of the nephrons, increasing renal reabsorption of solute-free water.[427] This resorption of water into the body increases urine concentration. A rise in plasma osmolality by 1 mOsm increases urine osmolality by almost 100 mOsm/kg and decreases its rate of formation. At the thirst threshold of 295 mOsm/kg H_2O, urine flow is reduced 10-fold to 20-fold.[345]

In summary, for most people, small rises in osmolality resulting from water loss do not normally initiate the sensation of thirst. During work in the heat, men never voluntarily drink as much water as they lose even when initially thirsty, and they usually replace only two thirds of the net water loss.[411] This phenomenon is called voluntary dehydration,[434] and it increases with increasing sweat rates (induced by higher T_{amb} or work rate), inadequate time allowed for rehydration, and greater effort involved in acquiring water. All persons in the heat should be considered dehydrated (except immediately after a meal) unless they have recently been forced to drink more water than they desired. In the past, heatstroke was almost always accompanied by some degree of dehydration.

In the past decade or so, medical authorities have fairly successfully educated athletes to drink adequate amounts of fluids during physical exercise in the heat, even if they are not thirsty. This has led to routine establishment of water stations along marathon routes for participants to obtain drinks on the run and to on-command drinking in the military. In a small number of cases, however, too much water was ingested, leading to overhydration, hyponatremia, physical collapse, and even death.[189] Therefore, to avoid exacerbating this condition, any administration of intravenous infusions to all collapsed marathon runners must be recorded, and all subsequent medical teams must be notified about the infusion. Because most collapsed athletes superficially appear similar, Noakes[389] emphasized that there is a significant danger of overhydrating people who collapsed in the heat. It is essential to determine the patient's hydration status unless it is certain that the victim has not ingested adequate water.

Chronic Hypohydration

There is an important delay between the thresholds for release of ADH (>280 mOsm) and for thirst (>295 mOsm). This delay is important and has a practical benefit. It frees the individual from being forced to drink immediately in response to changes in osmolality. On the other hand, certain individuals and groups[255] maintain a chronic state of hypohydration just below the thirst threshold (294 mOsm/kg), which represents a deficit of some 2L in total body water (TBW), according to the following example:

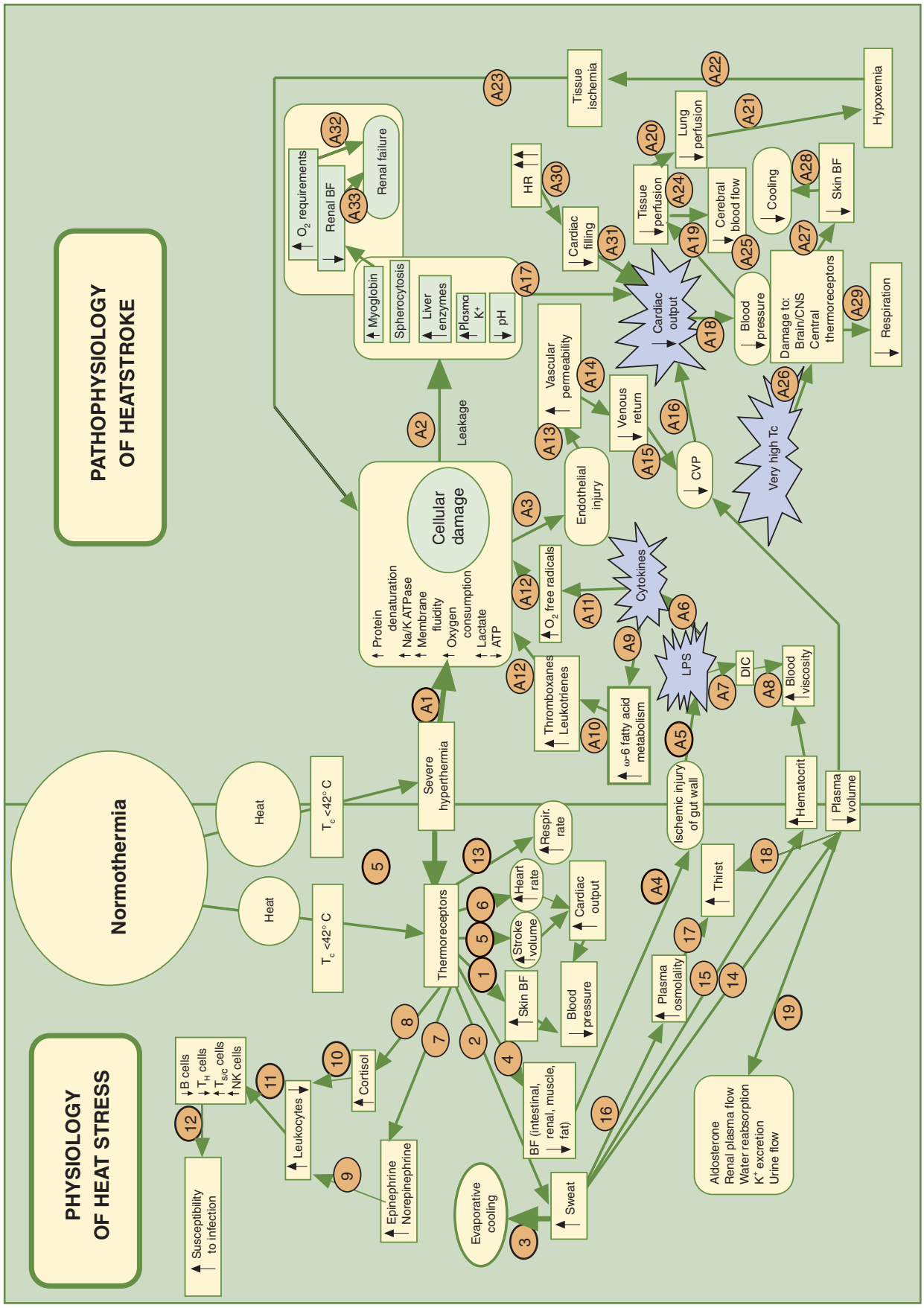
By exercising in the heat for only about 2 hours, one can incur additional water loss of 2.1 L (or 4.2 L total), which then results in a water deficit equivalent to 6% of body mass. This 6% TBW deficit decreases performance and increases risk for heat illness. The relationship between hydration state, rise in T_c, and decrement in physical performance is best shown in a classic work by Montain and colleagues,[361] who measured performance at three work rates and in three states of hydration (Fig. 10-9).

ACCLIMATIZATION

Prior heat exposure enhances heat tolerance by expanding circulating PV, increasing the maximal capacity of cutaneous vasodilatation and sweating, and reducing the threshold temperature for increased BF_{sk} and initiation of sweating (commencing cooling mechanisms at lower temperatures).[12,531] An acclimatized person shows a lower T_c, T_{sk}, and HR and a higher msw in response to a standard heat stress. A further important benefit of heat acclimation is reduced loss of sodium (both urinary and in sweat) and therefore reduced likelihood of developing salt-depletion heat illnesses from high sweat losses. When these physiologic adjustments occur in response to heat exposure in a controlled laboratory setting, they are termed acclimation, and if induced by the natural environment, acclimatization. However, the terms are often interchanged. The physiologic adaptations in the acclimation to heat and to severe physical exercise (training) are very similar: acclimation to one partly, but not completely, acclimates to the other.

The process of human acclimatization to heat produces both short-term and long-term changes in thermoregulatory

Figure 10-9. Model of the physiology and pathophysiology of heat stress and heatstroke.
Normal thermoregulation: Under a moderate heat load, as skin or core temperature rises, thermoreceptors increase skin blood flow (BF) (*1*) and cause the secretion of sweat (*2*), resulting in evaporative cooling (*3*). To prevent a drop in blood pressure, blood flow to the splanchnic regions and to muscle is reduced (*4*), and stroke volume (*5*) and then heart rate (*6*) are increased. Then catecholamines are secreted (*7*), followed by corticotropin-releasing hormone, which leads to the secretion of adrenocorticotropic hormone followed by cortisol (*8*). Catecholamines cause leukocytosis (*9*), whereas cortisol causes leukopenia (*10*), leading to changes in amounts of subsets of leukocytes (*11*). If the heart stress is prolonged and severe, the immunosuppression could lead to increased susceptibility to infections (*12*) and decreased physical and mental performance. As temperature rises, the respiratory rate increases (*13*). As a result of sweating, plasma volume decreases (*14*), and hematocrit (*15*) and plasma osmolality (*16*) rise, leading to the sensation of thirst (*17*, *18*) and the release of antidiuretic hormone. Reduced plasma volume (*14*), particularly in the presence of reduced renal blood flow (*4*), leads to rises in water-sparing hormones and reduced kidney function (*19*).
Impaired homeostasis: As temperature continues to rise above approximately 40° C (104° F), direct hyperthermic damage to cells commences with increases in membrane fluidity and permeability, increases in metabolic rate, including the activity of the Na^+,K^+-ATPase pump, increases in a variety of metabolites, and decreases in cellular ATP content (*A1*). Defects form in plasma membranes with leakage of proteins and potassium from cells, and red blood cells may change shape (*A2*). At about the same time, the reduction in intestinal BF (*4*) becomes more severe, leading to ischemic injury of the gut wall (*A4*). This in turns leads to rises in circulating lipopolysaccharide (LPS) (*A5*) and cytokines (*A6*). By activating a blood factor, LPS causes disseminated intravascular coagulation (*A7*) and a consequent rise in blood viscosity (*A8*). Thermal injury of endothelia (*A3*) together with cytokines (*A6*) leads to enhanced metabolism of ω-6 fatty acids (*A9*), as well as the production of thromboxanes and leukotrienes (*A10*), oxygen free radicals (*A11*), and further cellular injury (*A12*), and the probable production of the highly toxic nitric oxide and increased vascular permeability (*A13*). This leads to the loss of fluids into the tissues, and thus to reduced venous return (*A14*) and consequent reduced central venous pressure (*A15*). Following Starling's law of the heart, cardiac output begins to fall (*A16*). This is made more serious by electrolyte changes in the blood (*A17*). Eventually, blood pressure falls (*A18*), leading to reduced tissue perfusion (*A19*). In the case of the lung, reduced perfusion (*A20*) leads to systemic hypoxemia (*A21*) and eventually ischemia of various tissues and organs (*A22*) and its consequent contribution to further cellular damage (*A23*). The reduced tissue perfusion (*A24*) also leads to reduced BF to the brain (*A25*), and thus to probable direct thermal denaturation and damage to centrally mediated homeostatic mechanisms (*A26*), a reduced skin BF (*A27*), and a drop in cooling rate (*A28*), and eventually a fall in respiration (*A29*). In a separate pathway, cardiac output is also depressed as a result of too rapid a pulse rate (*A30*), causing incomplete cardiac filling (*A31*). Electrolyte derangements are made more severe by an increased metabolic rate (*A32*) and a reduction of renal blood flow in the kidney (*A33*).


PHYSIOLOGY OF HEAT STRESS
PATHOPHYSIOLOGY OF HEATSTROKE
Normothermia
Heat
Heat
T_c <42° C
T_c <42° C
Severe hyperthermia
Thermoreceptors
Susceptibility to infection
↓B cells
↓T_H cells
↓$T_{S/C}$ cells
↑NK cells
Leukocytes
Cortisol
Epinephrine
Norepinephrine
Evaporative cooling
Sweat
BF (intestinal, renal, muscle, fat)
Skin BF
Blood pressure
Stroke volume
Heart rate
Cardiac output
Respir. rate
Plasma osmolality
Thirst
Aldosterone
Renal plasma flow
Water reabsorption
K^+ excretion
Urine flow
Hematocrit
Plasma volume
Blood viscosity
Ischemic injury of gut wall
LPS
DIC
Cytokines
ω-6 fatty acid metabolism
Thromboxanes
Leukotrienes
O_2 free radicals
Protein denaturation
Na/K ATPase
Membrane fluidity
Oxygen consumption
Lactate
ATP
Cellular damage
Leakage
Endothelial injury
Vascular permeability
Venous return
CVP
Cardiac output
Blood pressure
Very high Tc
Damage to: Brain/CNS Central thermoreceptors
Respiration
Hypoxemia
Tissue ischemia
Lung perfusion
Tissue perfusion
Cerebral blood flow
Cooling
Skin BF
HR
Cardiac filling
O_2 requirements
Renal BF
Renal failure
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Spherocytosis
Liver enzymes
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responses involving sweating, skin circulation, and thermoregulatory set point. See Horowitz[248] for detailed experimental studies on rats showing these changes during short-term and long-term acclimation (30 days). For example, in some studies, short-term heat-adapted people sweated more rapidly, whereas those who were long-term adapted sweated more slowly.[245] Other equally impressive changes, although not specifically thermoregulatory, include cardiovascular alterations (HR, stroke volume, and PV) and endocrine adjustments. In the fully acclimatized state, a given heat or exercise load leads to reduced physiologic responses—for example, lowered T_c, lowered HR, elevated cardiovascular reserve, and increased cooling capacity through a reduced threshold for vasodilation.

Importantly, even within a single person the rates of heat and exercise acclimatization are not uniform among various tissues but vary from tissue to tissue. As a result, there are periods when compensation processes in different organs are out of phase with one another, and the overall effect may be negative.[462] For example, in rats, the immediate heat adaptation response of both the salivary glands and the heart is a temporary period of increased autonomic inputs but decreased sensitivity of those organs to their respective neurotransmitters, causing reduced intrinsic secretion of saliva and HR. These intrinsic failures may be masked during the acclimation processes by overstimulating those organs through especially high autonomic activities.

Over time, different genes are activated within each organ, leading to replacement of certain metabolic enzymes with isoforms more appropriate to elevated temperatures. These new isoforms have improved thermostability, altered reaction rates, and specificities, leading to development of more efficient dynamic processes with reduced heat absorption by the body. In the final adaptive state, these altered enzymes have the most efficient cellular performance, even with normal, rather than elevated, autonomic activities.[12,127,326]

Depending on a given individual's constitution, state of physical fitness, and thermal history, a specific thermal stress and intensity of exercise will produce some degree of thermal strain. However, each person acclimatizes to heat stress in his or her own individual way, with considerable variation in indexes of heat strain (e.g., weakness, rapid pulse, narrow pulse pressure, flushing of the face and neck, headache, shortness of breath, dizziness, cramps, nausea, vomiting).[462] Many early studies were found to be unreliable because of small sample size, incomplete acclimation, or inappropriate measurements. Furthermore, a good deal more is known about the time course for the acquisition of acclimation than about its decay or loss.[402] In recent years, heat-shock proteins have been shown to participate in the fundamental processes involved in acclimation.[247]

Is there a single, fundamental event during exercise heat stress that causes biochemical and physiologic alterations in the body that ultimately result in heat tolerance? At the moment, this is unknown. In humans, exercise in the heat causes a rise in aldosterone concentration within a few minutes. Elevated aldosterone concentration increases the number of Na^+,K^+-ATPase pumps present on plasma membranes.[172] Simply stimulating rabbit muscle over several days increased the number of Na^+-K^+ pumps on the sarcolemma, and exercise training in humans raised the number of Na^+,K^+-ATPase pumps on sarcolemma within 1 week.[207] Consequently, severe exercise no longer caused a "normal" rise in plasma K^+, because the increased number of Na^+-K^+ pumps attenuated the loss of K^+ from working muscle.[207]

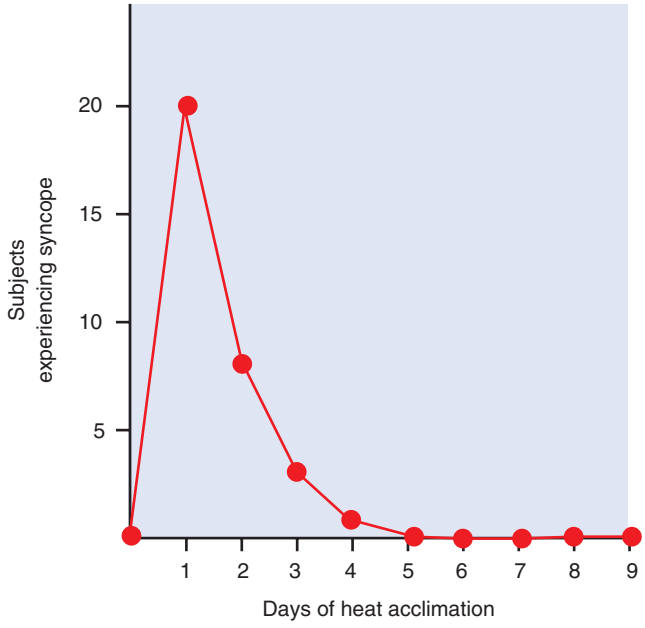

Figure 10-10. Incidence of syncope among 45 subjects who lived in and trained at cool ambient temperatures and could complete a physical training regimen without mishap. Then they were relocated to a hot environment where they carried out the same physical training regimen. (Modified from Bean WB, Eichna LW: Fed Proc 2:144–153, 1943, by Hubbard RW, Armstrong LE: In Pandolf KB, Sawka MN, Gonzalez RR [eds]: Human Performance and Environmental Medicine at Terrestrial Extremes. Indianapolis, Benchmark Press, 1988, pp 305–359.)

Physiologic Changes

One benefit of adaptation is clearly shown by the incidence of heat syncope among persons suddenly exposed to living in a hot environment (Fig. 10-10). Syncope, as described later, is caused by reduced cerebral blood flow resulting from a combination of peripheral blood pooling, reduced CO, and orthostatic hypotension. Syncope peaks on the first day of heat exposure and falls to zero by day 5. Persons can acclimatize to hot environments over a period of 1 to 2 weeks, and they also deacclimate over approximately the same period of time.

In general, symptoms lessen as acclimation progresses. Nearly complete acclimation to a given level of daily exercise and heat stress can be achieved in 7 to 10 days (Fig. 10-11).[402] The physiologic changes produced by heat or physical training include (1) lowered threshold temperature for the onset of BF_{sk}, (2) sweating (see Figure 10-11),[425] (3) increased PV, (4) increased stroke volume, (5) decreased HR, (6) decreased skin temperature, (7) decreased T_{re}, and (8) increased $\dot{V}O_2max$.[21] Physical training also increases aerobic capacity. Practically, this means that a fit but untrained person who runs a 500-m race and develops a T_{re} of 40° to 43°C (104° to 109.4°F) could, after acclimation, run the same race under the same environmental conditions and have a T_{re} of only 37.5°C (99.5°F).[301]

Although many controversial issues remain, a general rule is that either strenuous interval training or continuous exercise at an intensity greater than 50% $\dot{V}O_2max$ accounts for about 50% of the improvements found with classic acclimation procedures.[197] This may be important to the individual who wants to prepare for an acute change in thermal climate but does not

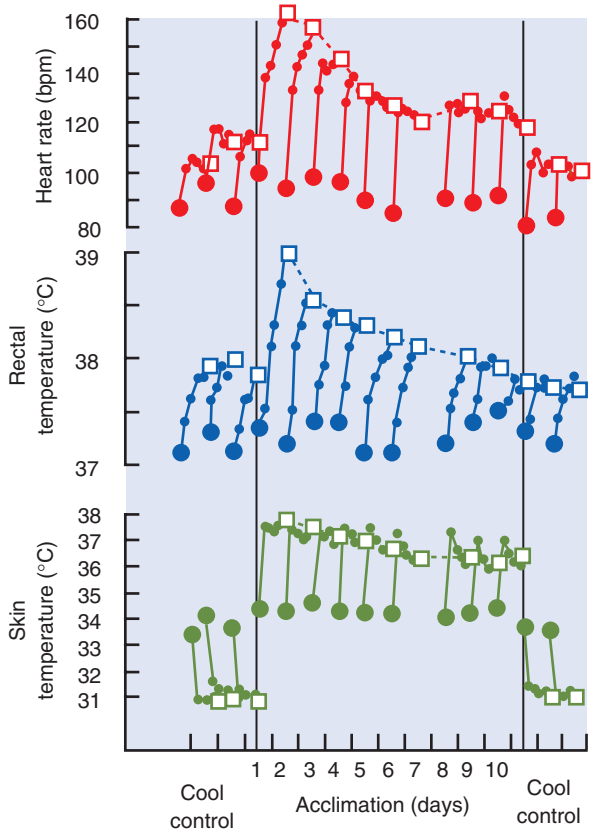

Figure 10-11. Effect of 10 days' acclimation on heart rate and rectal and skin temperatures during a standard exercise (five 10-minute periods of treadmill, separated by 2-minute rests) in dry heat. *Large circles,* Values before start of the first exercise period each day; *small circles,* successive values; *squares,* the final values each day. Controls of exercise in cool environment before and after acclimation. (Modified from Eichna LW, Park CR, Nelson N, et al: Am J Physiol 163:585–597, 1950.)

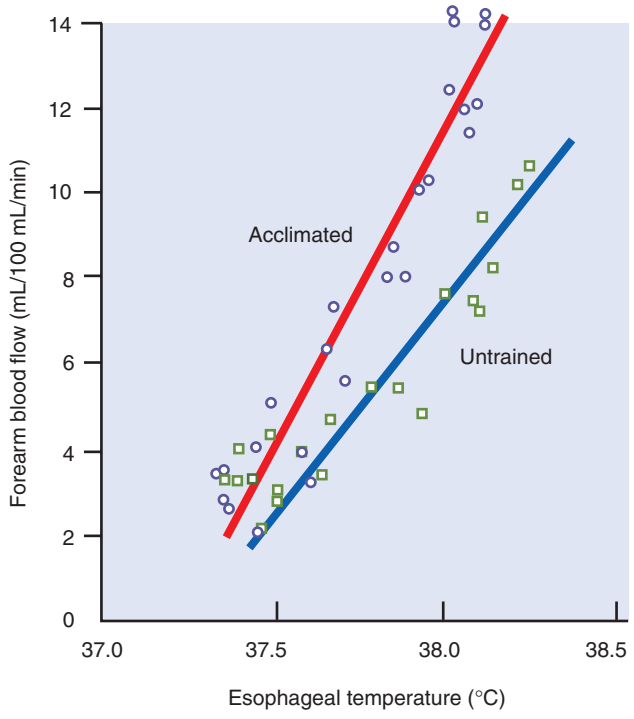

Figure 10-12. Effect of acclimation on forearm blood flow versus esophageal temperature in one subject. (Modified from Roberts MF, Wenger CB, Stolwijk JAJ, Nadel ER: J Appl Physiol 43:133–137, 1977.)

have the means to safely elevate his or her core temperature in the heat.

The primary mechanism for initiating the processes of heat acclimation is raising internal body temperature sufficiently to induce moderate sweating. This can be achieved by heat strain with moderate exercise in the heat or by exercise while wearing more clothing in a cooler environment. In the unacclimated individual, usually an hour per day of exercise in the heat is sufficient to produce an effect over days of exposure. As a rule, 10 days of successive treadmill walks in dry heat lower final exercise HR by about 40 beats/min; lower rectal and skin temperatures by 1° and 1.5°C (1.8° and 2.7°F), respectively; and increase sweat production by about 10%.[362] Generally, the degree of acclimation is related to the amount of daily heat stress. That is, daily 100-minute bouts in the heat conferred more acclimation after 9 days than did 50-minute bouts.[332] Most of the improvement in HR occurs in 4 to 5 days, and the improvement is nearly complete after 7 days (see Figure 10-11). As heat acclimation progresses, the onset of sweating occurs at lower core temperatures and the peak sweat rate is higher or even doubled (from 1.5 to 3 L/hr). Furthermore, the ability to sustain sweating for prolonged periods improves, along with the ability to increase sweating in skin areas that sweated the least before acclimation. Acclimation tends to make the sweating response more uniform over different skin areas.

These effects result, in part, from central mechanisms, as well as from intrinsic fundamental changes in pharmacologic activity of the sweat glands.[103] Heat acclimation increases the sweat glands' capacity to reabsorb sweat sodium at any given sweat rate.[5] Depending on the state of acclimation, the sodium concentration in sweat varies between approximately 5 and 60 mEq/L (10 to 20 mOsm/kg H_2O) and increases with sweat rate but declines with the state of acclimation. The salt-sparing effect of acclimation seems to depend on secretion of aldosterone, triggered by the combined effects of sodium depletion and heat stress.[308]

Skin vasodilation, especially in the forearm where it can be measured, occurs at a lower core temperature after acclimation (Fig. 10-12). Because these changes parallel the threshold T_c for sweating, they suggest that alterations are occurring in skin blood vessels at the same time as in skin sweat glands.[171] During acclimatization, despite vasodilation of skin in the heat, BP is maintained for a long period and without reducing splanchnic blood flow (BFspl). Improved vascular contraction results from improved responsiveness of α-adrenoreceptors and decreased responsiveness of β-receptors.

Changes in HR and PV decrease in parallel during the first week of acclimation.[462] The decrease in HR and increase in stroke volume may begin as early as the second day of work in the heat.[547] These changes are nearly complete by day 5, whereas final rectal temperatures are still declining with subse-

quent exposures. Many authors have reported that PV increases with the days of heat exposure. However, the mechanisms involved, including salt retention, addition of protein to the vascular space, and fluid shifts between compartments, are unclear. What is interesting, however, is a return of PV to baseline levels after about 2 weeks without apparent thermoregulatory impairment.[30] Horowitz and coworkers[250] have shown that long-term acclimation increased the compliance of rat ventricles in isolated hearts, resulting in a greater diastolic volume at any end-diastolic pressure, hence greater Starling contractility.

Climatic Effects

There is a seasonal effect on the threshold temperature (T_{th}) for the onset of sweating; T_{th} is highest in winter and lowest in summer. Therefore, for a given heat stress, BF_{sk} and sweat rate are highest in summer and lowest in winter.[376] Rapid air travel to a desert or jungle climate leads to incomplete acclimatization, or to none at all, and a very much increased rate of heat illnesses. For example, military personnel have higher heat illness rates in summer than in winter in a number of locations.[67,315] In a 4-year period, there were 10 severe heat casualties among soldiers visiting Cyprus during military training, largely because of incomplete acclimatization. The rate was highest during summer months when the WBGT was 26° to 29°C (78.8° to 84.2°F).[67]

The time course of acclimation is similar in hot wet and hot dry climates that are equivalent in WBGT. Acclimation to a defined hot environment confers equal acclimation to any other equivalent climate.[210] In groups of unacclimated healthy men subjected to heat for 9 days, those exercising under humid conditions produced better and more complete acclimatization than those exercising under dry conditions at similar T_{amb}. Practically, when training at any given T_{amb}, a humid environment and consequent higher heat load leads to more rapid acclimatization than training in a dry environment.[375]

Athletes have thinner skinfold thickness than do nonathletes. This leads to an increased rate of heat loss from the core via transport across the skin barrier into air by radiation, convection, and conduction. Exercise training improves heat dissipation not only by the reflex physiologic changes previously described but also by causing reduction in the thickness of subcutaneous fat. Importantly, skinfold thickness is thinner in summer than in winter, thereby improving heat dissipation when it is most needed.[245]

Biochemical Changes

Heat-Shock Proteins

The metabolic heat produced by severe exercise can raise the temperature of exercising human muscle to 45°C (113°F), and T_c up to 44°C (111.2°F). These temperatures can damage muscle mitochondria so that they generate O_2^- and H_2O_2, causing oxidative damage in addition to thermal stress.[445]

All cells have the potential of being stressed by heat and other stressors. To survive, they have developed the ability to respond to moderate stresses and rapidly synthesize specialized proteins known collectively as heat-shock proteins (HSPs), which protect them against ordinarily lethal conditions. Many cell types are ordinarily destroyed by 5 minutes at a T_{amb} of 49°C (120.2°F). However, if they are first heated to 42°C (107.6°F) for 15 minutes, protective HSPs are induced within the cells so that they can then survive exposure to a T_{amb} of 49°C.[442] Among their many properties, HSPs can refold denatured proteins into their normal conformation and stabilize normal proteins against thermal injury. The binding of HSPs to denatured proteins does not cause immediate global conformational change of the unfolded proteins. Instead, HSPs act locally on small regions of exposed hydrophobic amino acids through repeated cycles of binding, folding, and release reactions that are energized by hydrolysis of ATP.[71] HSPs are produced as a general response to all known stresses, if sufficiently intense, including hypoxia, free radicals, cold, acidic environment, starvation, ionizing radiation, a variety of chemical toxins, and even electromagnetic fields.[71,158,282,330] HSPs are induced not only by physical and chemical stresses, including sepsis, cocaine, and tobacco smoke,[47,118,520] but apparently even by psychologic stresses, such as loud noise, isolation in darkness, restraint, water immersion, inescapable irritation, seizures, and sleep deprivation.[93,175,230,241,252] Indications of elevated levels of HSP-60 were found in one small study of schizophrenics.[459] Furthermore, HSPs may protect parasites against host defenses.[456]

Heat shocking a variety of cells and animals increased HSP content and protected them against not only heat but also apoptosis, ischemia/reperfusion injury to the mucosa, and ionizing radiation.[373,396,446,494] In mammalian tissues, the presence of heat-shock proteins appears to provide an overall protective advantage of at least 2°C (3.6°F) against morbidity and mortality caused by high temperatures.[349]

Production of high levels of HSPs is not without cost—otherwise, they would be present in cells all the time. HSPs may interfere with spontaneous folding of new proteins, and the stresses sufficient to induce HSPs redirect cellular machinery to produce HSPs at the expense of producing other proteins. This would be particularly damaging to rapidly growing cells, such as during fetal maturation.[158]

HSPs are grouped into a series of families on the basis of their molecular masses. For example, the most important HSP-70 family contains several proteins of 68 to 75 kDa. In each cell, certain HSPs from each family are normally present and increase only moderately following heat—for example, HSP-73, which is sometimes referred to as constitutive HSP-70. Other HSPs are normally absent from the cell or present in only very low amounts but are rapidly induced by heat (T_{amb} > 41°C [105.8°F]) to become the most common protein in the cell—for example, HSP-72. The widely used, but ambiguous, term *HSP-70* refers to any or all members of the family.

The threshold temperature for the induction of HSPs is not a constant but depends on the species and cell type and is approximately 4°C (7.2°F) above the normal environmental temperature encountered by the cell. For example, an ambient temperature as low as 5°C (41°F) induces HSPs in Antarctic algae, which normally live at –1° to 1°C (30.2° to 33.8°F).[519] Most investigators suggest that the threshold in mammalian cells is approximately 41°C (105.8°F), although temperatures as low as 39°C (102.2°F) have been reported.[96] Because production of HSPs and cell survival also depend on the rate of heating, this rate may also be an important factor in thermal injury.[48,239] HSP production is also organ dependent. In heat-shocked rats (42°C [107.6°F]), the amount of HSP-72 was tissue specific and was produced quantitatively in the order of liver >> small intestine > kidney, quadriceps muscle > brain.[163] In rat brain, lung, and skin, HSP-70 concentration peaked at 1 hour and returned to baseline at 3 hours after the stress. In liver, however, HSP-70 peaked 6 hours after the stress.[48] The

determination of HSP-72 may be useful as a diagnostic probe of recent heat injury, as an enzyme-linked immunosorbent assay (ELISA) for HSP-72 determination has become commercially available (Stress-Gen, Vancouver).[65]

Most studies on HSPs have been carried out in isolated cells and in rodent models. In humans, the minimal conditions of threshold T_c and duration for a single heat shock to induce HSPs is not certain. Some cells can survive very brief exposures (1 second) to temperatures as high as 60° C (140° F), because they induce HSPs that render them thermotolerant.[62] After a single heat shock in human peripheral blood mononuclear cells, HSP-72 concentration is unchanged for 1 to 2 hours, then rises rapidly to plateau at 4 hours. It gradually falls after 12 to 18 hours but remains above baseline at 24 hours.[184] Overall, however, on a single supra-threshold stress, HSPs in many or most tissues rise within an hour or two of the stress and remain elevated for 12 to 48 hours and possibly for as long as a week.[224] During sports activities or exercise in summer, however, T_c can become elevated several times over a few hours. Such stimuli may have profound effects on the time course of HSP elevation and protection, but this has not been well studied.[247,313]

This temporary thermoprotection by HSPs to heat and exercise is not long-term acclimatization. Long-term acclimatization is a different process, although it may in part involve HSPs. It develops over a period of several days in response to several periods of moderate heat and may persist for weeks.

In the work of Horowitz,[247] acclimatization involves alterations in autonomic, cellular, and molecular responses to heat, varying in their intensities and interrelationships over time. Some studies suggest that increased HSP-72 during rigorous exercise is one of the adaptive mechanisms to cope with increased stress.[249,328] Rats run on a treadmill for 30 minutes per day at 75% $\dot{V}O_2max$ showed increases in HSP-72 within 10 weeks.[123] Endurance exercise–trained rats had higher levels of HSP-72, as well as lower levels of lipid peroxidation, in their ventricles and could develop higher systolic pressures.[122,414]

During exercise of an unacclimatized individual in moderate and warm temperatures or at rest in tropical or desert heat, T_c may rise to levels that severely decrement performance or may lead to heatstroke, collapse, and even death. Therefore, the presence of HSPs is extremely important. A better understanding of the role of HSPs may ultimately lead to methods for faster acclimatization to heat. The ability to produce HSPs may depend on diet, because vitamin D–deficient animals showed reduced HSP production.[281]

Protection. In a number of different systems, HSP levels elevated by a variety of stresses (heat shock, brief cyanide exposure, arsenite, peroxide, hypoxia, and toxins) were beneficial to membranes, cells, tissue, and organs. HSP-70 protected cells and their ultrastructure against these or other stressors.[7,51,291,523] They protected against membrane damage caused by toxins (ionomycin) and cytokines.[211,316] Well-healing wounds show high levels of hsp70mRNA, whereas poorly healing wounds have lower levels.[392] Overexpression of HSP-70 in transgenic mice rendered their hearts more resistant to ischemic injury.[344] Human cell types (proximal tubular epithelia) that normally exhibit great resistance to hypoxia also contain high basal levels of HSP-70.[512] Elevated HSP-72 (by heat shock or arsenite) protected rats against heatstroke morbidity and mortality.[551]

Elevated HSP-72 blunted liberation of inflammatory mediators (interleukin-6 and thromboxane B_2) in LPS-challenged swine and improved the course of hemodynamic variables; it decreased peak pulmonary arterial pressure and pulmonary vascular resistance index values, increased systemic arterial pressure and systemic vascular resistance index values, and favorably altered hypodynamic/hyperdynamic CO.[296] Elevated HSP-70 (by heat shock) protected rat intestine against toxin (ricin)-induced acute intestinal inflammation, with reduced generation of leukotriene B4 and neutrophilic infiltrate in their ileums.[493] Monocytes and granulocytes constitutively express more HSP-70 and are more heat resistant than lymphocytes. LPS raises HSP-70 content even more and may be the reason that those cells can survive and function in hostile inflammatory microenvironments.[161]

Not only does raising HSP levels protect against heat, but reducing them is damaging during stress. For example, reducing HSP-72 induction by 40% (by a specific toxin) rendered cardiac myocytes more susceptible to hypoxic injury.[377]

In principle, it may be possible in humans and experimental models to elevate HSPs by one stress (e.g., temporary hypoxia) to render athletes more tolerant to heat, cold, or toxins. This would be expected to benefit not only athletes but also soldiers deployed to hot climates and patients soon to undergo surgery.

Age. In normal adult rats, heat and exercise can each induce HSP-72. However, in aged rats, only exercise could induce HSP-72.[314] That is, aging caused them to lose their ability to induce HSPs by heat. Among humans, older adults have lower levels of HSP-70 in their peripheral blood mononuclear cells (PBMCs) than do the young.[116] This loss of HSP protection may in part account for increased susceptibility of the aged to classic heatstroke, as is seen in heat waves.

Race. HSP production may be race related. In one study, heat-induced HSP levels were very intense in cells isolated from native Turkmen men living in Turkmenistan, the hot desert of middle Asia, but very weak in Russians living in moderate climatic regions of European Russia. At the same time, cells isolated from Turkmen men better survived heat stress.[264] Not surprisingly, organisms living many generations in hot climates become better adapted to it over time. No large-scale systematic study of racial influences on HSPs has been reported. However, one report indicates a difference in basal and heat induced levels of HSP-70 between Europeans and non-Europeans in South Africa, and it suggests that there may be different susceptibilities to stress and disease.[55]

Training. Long-term exercise training in the heat induced HSPs. In one study of rat brains, HSP-70 content rose only if the exercising rats became hyperthermic, whereas exercise alone did not induce central HSP-70 expression.[523] On the other hand, in another study in rats, in spleen cells, peripheral lymphocytes, and soleus muscles, exercise alone induced HSP-60, HSP-70, HSP-90, and HSP-100.[336] In male rowers in training, the HSP-70 content in an active muscle rose each week (181%, 405%, 456%, and 363%) with maximal HSP production at the end of the second training week.[536] In humans walking 30 minutes on a treadmill at their individual anaerobic thresholds, mRNA for HSP-70 rose but not the HSP-70 protein itself. That is, a single bout of exercise in humans may not be sufficient to induce HSP-70 protein.[416]

Combined stressors may be additive or synergistic in their effects. Exposure of cells to either a moderately elevated tem-

perature or to a low level of ethanol does not induce HSPs. However, when the two stresses were applied at the same time, the cells induced large amounts of HSPs. Thus hyperthermia and ethanol acted synergistically to increase HSP gene expression.[431] Practically, this implies that HSPs may be induced by overlapping periods of exercise, heat, hypoxia, and sleep deprivation.

Consequences of Elevated HSP-70. There is a downside to the production of HSPs during stress injury. While HSPs are being synthesized, the cell ceases or slows production of many other proteins. As a result, in cells at normal temperatures, synthesis of HSPs (induced by a short heat stress) retards cell growth,[157] even as it protects against subsequent heat injury. Not only is growth retarded but heat-shocked immune cells secreted reduced amounts of cytokines in response to LPS.[422] Therefore, persons with elevated HSPs may have reduced or inappropriately altered immune function. This was clear in immune gene activities shown in a recent genomics study.[486] Clearly, the potential benefits of deliberately inducing HSPs, with presumed improved resistance to heat, hypoxia, and certain chemicals and toxins, must be weighed against a potentially decreased ability to resist infections.

Thyroid Hormone and Protein Isoforms

Thyroid gland function is an important component of heat acclimation. IL-1 inhibits release of thyroid hormone from the thyroid gland.[113,138] Because IL-1 becomes elevated in response to heat and many other stresses (see Figure 10-5),[182] its ability to inhibit thyroid hormone release may be part of the overall protective mechanisms and responses to stress.[278]

A reduction in thyroid hormone concentration has a number of biochemical and physiologic consequences. Deficiency of thyroid hormone has a negative cardiac chronotropic effect, reducing cardiac contractility and increasing systemic vascular resistance.[66] Basal metabolic rate (BMR) falls with thyroxine deficiency, and because its secretion rate is lower in summer and higher in winter, there is corresponding seasonal alteration in BMR and all other processes influenced by thyroxine concentrations.[245] One component of heat acclimatization is reduction in BMR.[245] Chronic heat stress decreases blood flow to the thyroid gland[217] and reduces the rate of thyroid hormone production, leading to decreased food intake, growth rate, oxygen consumption, and BMR.[435] Deficiency in thyroid hormone during acute stress may be fatal. A young woman was discovered unconscious in a sauna and later died with a diagnosis of heatstroke. On autopsy, she was found to have preexisting Hashimoto's thyroiditis.[473]

Muscle contains characteristic isoforms of myosin, each with different intrinsic metabolic activities, such as the rate of its actin-activated ATPases. Myosin molecules are composed of heavy and light subunits that associate in a specific manner, each of which is specific to muscle type during development and maturation; that is, ventricular myosin in a fetal heart is different from that of an adult heart.[89] In some muscles, acclimation leads to a higher proportion of "slow" myosin ATPase isoforms, such as the replacement of myosin heavy chain type IIb (MHCIIb) with type Iix.[133,250] The hearts of heat-acclimated rats become more efficient (amount of oxygen required per unit force time per gram of tissue)[246] as a result of the presence of altered isoforms of contractile proteins. Thirty to 90 minutes of daily exercise for 10 weeks reduced the percentage of MHCIIb fibers in rat hind-limb muscles and increased the slower, but more efficient, MHCIIa fibers. That is, increasing the training duration increases the fast-to-slow shift in myosin isoforms.[121] Excess thyroid hormone increases the amount of myosin isozyme VI, with its high rate of ATPase and contractile speed, at the expense of the normal V3 isozyme, leading to greater speed and strength but reduced efficiency. In the case of sweat secretion, on acclimation there is an intrinsically higher rate of sweat secretion because of isoform alterations along the secretory tubules.

Administration of thyroid hormone to neonatal rats rapidly replaces fetal cardiac ventricular myosin with its adult isoform. On the other hand, if the synthesis of thyroid hormone is suppressed, then the slower, fetal isoform predominates.[66] Thyroid hormone concentration regulates protein isoforms, activities, and therefore the output of metabolic heat.[253]

The speed and extent of contractility of heart muscle depend on the number of Ca release channels in the sarcoplasmic reticulum (SR) (more channels permit faster Ca^{2+} entry into the cytoplasm and faster contraction) and on the rate of Ca uptake by the SR (faster Ca^{2+} uptake means faster relaxation). In ventricles, low thyroid hormone levels reduce the number of Ca release channels, which depresses contractility. In the atria, low thyroid hormone also increases the density of muscarinic receptors, rendering them more sensitive to negative chronotropic agents. In summary, low thyroid hormone levels depress cardiac function and render the heart more sensitive to agents that decrease atrial contraction, and less sensitive to agents that increase HR and contraction.[15,295,312,432]

Acclimation occurs under conditions of relative or actual hypothyroidism. As a result, the acclimated heart shows altered myosin isoforms and increased phospholamban content but lowered contractile velocity, rate of Ca uptake by the SR, and relative oxygen consumption. These changes increase overall efficiency of the heart at the expense of contractile velocity.[79,360]

Thyroid hormone increases the number of Na^+,K^+-ATPase pumps[97] and decreases the density of voltage-dependent calcium channels on plasma membranes (leading to reduced intracellular Ca^{2+} content).[159] Thyroid hormone and sustained aldosterone, such as from excess sweat loss and elevated plasma Na^+, also alter metabolic processes and electrical activity of cells by regulating intracellular Na^+ and K^+ concentrations. In the short term, they increase the number of functional Na^+,K^+-ATPase pumps by recruiting preformed but inactive pumps and their subunits to the plasma membrane. In the long term, they induce synthesis of new pump subunits.[97,155] Therefore, the prolonged and reduced levels of thyroid hormone seen during acclimatization decrease the number of those pumps, hence reducing metabolic activity.[463]

Almost any traumatic insult to the body alters hormonal level by elevating corticotropin releasing hormone, in turn decreasing thyroid hormone, growth hormone (GH), gonadotropin-releasing hormone, luteinizing hormone, follicle-stimulating hormone, and gonadal steroid concentrations, while increasing ACTH, cortisol, and prolactin levels (see Figure 10-5).[46] Reduction in thyroid hormone concentration commences within a few hours, may be maximal after 1 to 4 days, and persists for the duration of the illness.[338] Among heatstroke victims, decreases in serum thyroid hormone correlated with severity of heatstroke, according to peak T_c.[91] That is, severity of heatstroke was related to depression of thyroid function. After the patients

completely recovered, thyroid function tests returned to normal. The hypothyroid state may protect by preventing undesirable catabolic effects. Therefore, thyroid replacement therapy is not currently recommended.[91]

In summary, long-term acclimatization to heat and exercise involves changes on a molecular level, involving alterations in intracellular HSP concentrations, increased IL-1, reduced thyroid hormone, increased aldosterone, increased number of Na^+,K^+-ATPase membrane pumps, and alterations in protein isoforms, altogether contributing to the physiologic adaptations in organ function.[246]

IMMUNE SYSTEM

In the processes of digestion and absorption of ingested food, chyme remains in the intestines for hours to days. Although we absorb a large share of the nutrients, bacteria present in the gut lumen also utilize nutrients from the chyme and reproduce rapidly, reaching concentrations of 10^9 to 10^{12} organisms per gram.[168] Dead gram-negative bacteria slough off into their milieu large amounts of the highly toxic cell wall component LPS, which may reach concentrations of 1 mg/g in the feces, a million times the lethal concentration if it were in plasma.

As long as LPS remains within the intestines, it is not harmful. Small amounts that leak into the circulation are rapidly inactivated by several mechanisms. Some LPS is phagocytosed by bound Kupffer cells within the liver reticuloendothelial system (RES), where it is partly detoxified and then bound by hepatocytes for further degrading[509]; some LPS binds to circulating antilipopolysaccharide antibodies[176]; some binds to high-density lipoprotein (HDL)[513]; and some binds to LPS-binding protein (LBP).[544]

If large amounts of LPS rapidly enter the circulation, it could overwhelm the protective systems, allowing LPS to express its toxic effects rapidly. At plasma concentrations between approximately 10 and 100 pg/mL, LPS initiates a cascade of molecular events, leading to nausea, vomiting, diarrhea, fever, and headache.[44] Higher concentrations can lead to conditions identical to those of gram-negative bacteremia, including vascular collapse, shock, and death.[80] In fact, during gram-negative bacteremia, normal immune mechanisms may destroy all viable circulating bacteria so that at the time of death there may be zero live bacteria in the plasma. The circulating LPS appears to be the immediate cause of septic shock.[534]

Intestinal Ischemia and Lipopolysaccharide Release

During exercise, blood flow to muscle may rise from a resting value of 1 to 2 mL/100 g/min to as high as 300 mL/100 g/min to deliver oxygen and nutrients. As T_c rises, blood flow to the skin also rises to provide cooling. This strains the cardiovascular system's ability to maintain blood flow to the heart, brain, and liver.[304,350] During heavy exercise, it is important that blood flow to the liver be substantially maintained to remove lactate and other metabolites from the blood and to provide glucose for energy.[304]

To maintain blood pressure under still more intense exercise and thermal stress, blood flow is reduced to those organs less immediately critical—that is, the intestines and kidneys. If splanchnic blood flow drops sufficiently, reduced delivery of oxygen leads to regions of local ischemia and transitory damage to the barrier function of the gut wall. If this is prolonged, there may be permanent ischemic injury to the gut wall.[415,418] Occurrence of several transitory bouts of local hypoxia and metabolic stress in splanchnic tissues may, during reperfusion, generate free radicals, exacerbating the ischemic injury.[221] Exercise at 80% $\dot{V}O_2$max for 30 minutes at 22°C (71°F) increased permeability of human intestines to small molecules.[401] Ischemic injury of the intestines causes the diarrhea or water intoxication occasionally encountered during a marathon run as a consequence of the inability to reabsorb water ingested during the race.[304] For example, in one extreme case, after winning a marathon in 1979, Derek Clayton stated that "two hours later . . . I was urinating quite large clots of blood, and I was vomiting black mucus and had a lot of black diarrhea."[166] Another athlete, Dr. Peter Rogers, stated that during training for two Olympiads (gymnastics, 1972, 1976) and one world championship, many of the athletes developed bloody diarrhea after long periods of intense training (personal communication, 2005). Intense and prolonged running is a common cause of gastrointestinal bleeding; up to 85% of ultramarathoners demonstrate guaiac-positive stools from a 100-km (62.1-mile) race.[29] Endotoxemia, the ultimate consequences of reduced intestinal blood flow, may be severe.

Because of the high LPS gradient across the gut wall, almost any insult to the integrity of the gut wall leads to a rise in plasma LPS. Severe hemorrhage reduces splanchnic blood flow and oxygen delivery to the walls of the stomach, small intestine, and sigmoid colon.[128] This has led to local elevations in the permeability barrier of the gut wall and caused endotoxemia.[181] In a swine model, blood flow through the superior mesenteric artery was progressively occluded, leading to progressive tissue hypoxia and a local shift to anaerobic metabolism, producing lactate and a fall in the pH of the gut wall.[162] At about the same time, LPS entered the circulation. This may lead to an ominous positive-feedback loop, because infusing LPS itself causes hypotension, reduced splanchnic blood flow, and an increase in gut permeability so severe that whole bacteria can be translocated into the circulation.[162,529]

The size of putative holes in the gut wall accounting for the rise in LPS permeability depends on the duration of the ischemia. When the superior mesenteric artery of canines was occluded, LPS (molecular or micellar) leaked out into the circulation within 20 minutes, but whole live bacteria (several orders of magnitude larger) required 6 hours of occlusion.[406] In other species, these times may be much shorter.[117]

In a different model, nonhuman primates breathed a hypoxic gas mixture for an hour.[17] The resultant hypoxemia rapidly initiated a reflex response designed to maintain oxygenation of the heart and brain at the expense of the rest of the body. This reflex caused intestinal blood flow to fall and was so intense that it resulted in transient ischemic injury to the gut wall and translocation of LPS into the circulation within only 5 to 10 minutes of breathing the hypoxic gas mixture. When the immune system was suppressed by whole-body ionizing radiation, the same hypoxic stress caused LPS to rise to higher levels and persist in the blood for a longer period. On the other hand, when the gut flora had been reduced three to four orders of magnitude by administration of nonabsorbable antibiotics for a week, there was no detectable translocation of LPS and bacteria by hypoxia.[117,168]

There is a fitness component to alterations in splanchnic blood flow. When experimental animals (as compared with

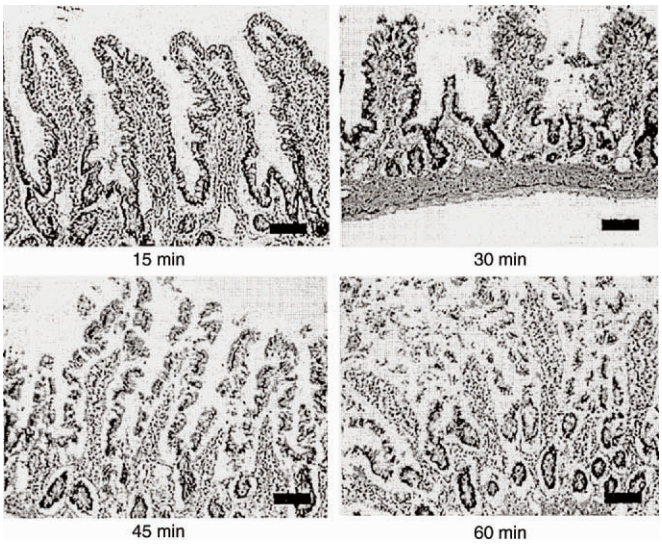

Figure 10-13. Effect of heating on villus structure. Representative light micrographs of rat small intestinal tissue over a 60-minute course at 41.5° to 42°C. Note the generally normal appearing villi at 15 minutes (slight subepithelial space at villous tips), compared with initial sloughing of epithelia from villous tips at 30 minutes, massive lifting of epithelial lining at top and sides of villi at 45 minutes, and completely denuded villi at 60 minutes. *Bars* represent 100 µM. (From Lambert GP, Gisolfi CV, Berg DJ, et al: J Appl Physiol 92:1750–1761, 2002, with permission.)

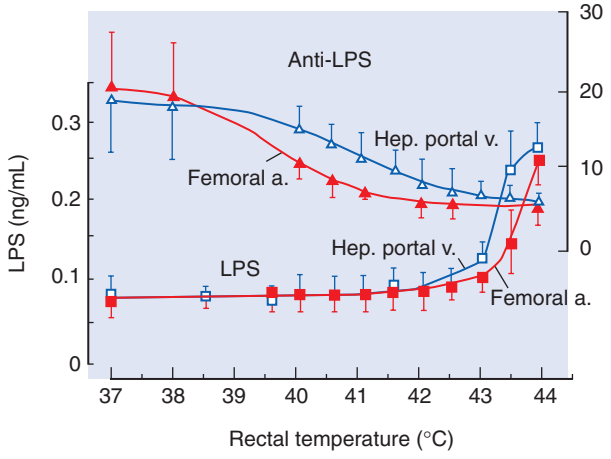

Figure 10-14. Endotoxemia caused by heatstroke in anesthetized nonhuman primates. At T_{re} of 42° to 43°C (107.6° to 109.4°F), plasma lipopolysaccharide (LPS) concentration rose first in the hepatic portal vein, and 10 to 15 minutes later in the systemic circulation. However, a decline in consumed anti-LPS antibodies occurred at temperatures as low as 39° to 40°C (102.2° to 104°F). (Modified from Gathiram P, Wells MT, Brock-Utne JG, Gaffin SL: Circ Shock 25:223–230, 1988, with permission.)

sedentary animals) were heat stressed, the fit ones with their greater cardiovascular capacity better maintained BFspl, they had reduced amounts of ischemic damage to the gut wall, and reduced quantities of LPS translocated into the circulation.[444] That is, fitness may reduce the LPS load during intense exercise. Such studies show that the permeability barrier in the gut wall is rapidly damaged by hypotension, reduced blood flow, and hypoxia, thus permitting LPS to enter the portal and systemic circulations at a high rate.

It has been reported that after heatstroke patients are cooled, they may develop secondary fever and infection carrying a high death rate.[470] The susceptibility of such patients to infections[470] may result from a combination of changes in lymphocyte subpopulations, together with the increase in gut wall permeability to LPS and bacteria caused by hyperthermia and associated hypotension.[117]

Role of Lipopolysaccharide in Heatstroke Pathophysiology

Heatstroke temperatures greater than 43°C (109.4°F) cause a large increase in permeability of isolated rat intestinal walls to LPS that persists even after the temperature is reduced to 37°C (98.6°F).[384] This suggests that heat causes major direct thermal injury of the gut wall at approximately 42° to 43°C (107.6° to 109.4°F) (Fig. 10-13).[318] The time course of the movement of LPS through the intestinal wall into the circulation resulting from hypoxia, ischemia, and ionizing radiation was determined in nonhuman primates[125] and compared with hyperthermia (T_{amb} = 41°C [105.8°F], RH = 100%, 3 to 4 hours).[327] As T_c rose, plasma LPS concentration remained low until 42° to 43°C (107.6° to 109.4°F) (Fig. 10-14). At this temperature, there was a sudden rise in LPS concentration, first in the portal vein, and 10 to 15 minutes later in the systemic circulation. This sequence appears to be the main route of LPS: out of the lumen of the intestines, through the portal vein to the liver, and into the vena cava and systemic circulation as a result of heatstroke and intestinal ischemia.[192]

In a previous study of infection by injecting live gram-negative bacteria, Gaffin and coworkers[534] noted that when the concentration of live gram-negative bacteria or LPS rose, the concentration of measurable circulating LPS-specific antibodies fell because they became bound to circulating LPS and thus were no longer detectable by an immunoassay.[534] It had been expected therefore that in the heatstroke experiments, the concentration of natural (i.e., background) anti-LPS antibodies would also immediately fall at 42° to 43°C (107.6° to 109.4°F). Contrary to expectations, natural anti-LPS began to decline at temperatures as low as 39° to 40°C (102.2° to 104°F) (see Figure 10-14).[192] This suggests that as T_c rose to only 39° to 40°C, LPS actually began to leak into the circulation at a slow rate, gradually consuming the anti-LPS antibodies. As T_c continued to rise, at a certain point massive damage to the gut wall occurred, leading to rapid leakage of LPS into the portal vein. It is not clear how much of this damage is caused by reduced oxygen delivery from reduced intestinal blood flow, how much by direct thermal damage of the gut wall, and how much by other causes.

Anti-LPS antibodies were protective against heatstroke in vervet monkeys up to a T_c of 43.5°C (110.3°F), but no higher.[191] This suggests that LPS-induced toxicity is important in the pathophysiology of heatstroke death only up to 43.5°C (108.5°F). Above this temperature, other mechanisms are more important, such as direct thermal damage to nervous tissue.

LPS had previously been implicated as a factor in heatstroke death by indirect observations. Injection of very low doses of LPS leads to rapid tolerance to ordinarily lethal doses of LPS.[136,208] Administration of low doses of LPS protected rats against subsequent heatstroke. When activity of the reticuloen-

dothelial system (the main mechanism for removal of LPS from the circulation) was reduced, the mortality of heatstroke increased.[137] Ryan and colleagues[441] found the inverse effect. Rats were heated to a T_c of 42.5°C (108.5°F) and then were passively cooled. The next day they were challenged with a lethal dose of LPS, and mortality rate dropped from 71% in the control rats to zero in the previously heat-stressed rats.

The importance of LPS in fatal heatstroke death was confirmed in a canine model of heatstroke.[232] T_c was raised to 42°C (107.6°F) for 3 hours and then cooled to 38°C (100.4°F). Deaths occurred only in the animals with rises in plasma LPS concentration.

Exertional Heatstroke

Several studies support the idea that the immune system and LPS are involved in the pathophysiology of heatstroke. Leukocytosis is a general response to most forms of stress, including muscular activity,[190] administration of epinephrine or glucocorticoid, and excitement. Prolonged or severe exercise initiates mobilization and activation of neutrophils and causes proteolysis of skeletal muscle and production of acute phase proteins by the liver.[76] Exercise leads to local disruption of tissues and sloughing of tissue fragments that circulate and activate the complement system. This activation primes monocytes for further activation by LPS or by fragments of tissue subsequently damaged.[78]

Severe exercise causes reduction in splanchnic blood flow and impaired renal function, with an increase in urinary excretion of proteins so profound (>100-fold) that it led to an actual depletion of circulating proteins.[33,415] To understand the relationship between splanchnic shutdown and heatstroke pathophysiology, it is necessary to consider the contents of the intestinal lumen and the likely results of their leakage into the systemic circulation.

Many of the clinical signs in a heatstroke victim, including blood-clotting disturbances, are similar to those seen in septic shock cases.[202] LPS activates a blood factor leading to disseminated intravascular coagulation,[39,555] a common complication of septic shock. It was suggested that LPS participates in the pathophysiology of heatstroke. Similarities between heatstroke and septic shock are described in Box 10-5.

Because core temperatures of long-distance runners may rise above 40°C (104°F), a study of runners who collapsed during an ultramarathon (89.5 km [55.6 mi]) run on a warm day was conducted.[68] Those runners reached or were carried into the medical tent at the finish line, where blood samples were taken from 98 of them before initiating volume therapy (Fig. 10-15). Eighty of 98 runners had plasma LPS levels above normal values, including two in the lethal range. It should be noted that the body can tolerate short periods of high LPS concentrations.[534] Although hypovolemia and hemoconcentration resulting from sweating may have caused a few high normals to cross into the elevated range, this was not the case for the majority. Of those who finished the race, the smaller group with normal, low LPS levels finished faster. Furthermore, this normal group had higher levels of natural anti-LPS in their plasma. That is, the presence of high levels of anti-LPS antibodies correlated with low levels of LPS and better performance. This high anti-LPS antibody and low LPS group also had reduced indexes of nausea, vomiting, and headache and recovered faster (within 2 hours) than did the larger high-LPS and low-anti-LPS group (up to 2 days).

Box 10-5. Common Factors in Heat Illnesses and Sepsis

CLINICAL

Neurologic symptoms
- Fatigue, weakness, confusion, delirium, stupor, coma, dizziness, paralysis, amnesia

Tachycardia
Nausea, vomiting, diarrhea
Headache
Myalgia
Hypotension
Spasm, rigors
Oliguria, renal failure
Hyperventilation
Organ failure
Shock

LABORATORY

Metabolic acidosis
Hematocrit elevated
Urea elevated
Lactate elevated
Disseminated intravascular coagulation
Cytokines elevated
Hepatic dysfunction
Lipopolysaccharides elevated

In triathlon participants, concentrations of LPS rose and of anti-LPS antibodies fell at the end of the third race.[54] Some athletes were found to have higher levels of natural anti-LPS antibodies than others. When individuals were questioned about their training regimen, it became clear that those who trained the hardest (miles swum, bicycled, and run the 3 weeks before the triathlon) had the highest levels of anti-LPS antibodies. It may be that one component of the benefit of physical training is increased levels of natural anti-LPS antibodies. We proposed that as a result of severe training, temporary periods of intestinal ischemia occurred, leading to entry of low to moderate levels of LPS into the circulation, which was enough to stimulate the immune system and induce anti-LPS antibodies. When a marathon was run on a cold day, no elevations in plasma LPS were seen (T. Noakes and S. Gaffin, 1988, unpublished observations). It is not clear what combinations of heat load and exertional factors are required to decrease BFspl sufficiently to damage the gut wall for translocation of LPS into the circulation.

Classic Heatstroke

Survival of hospitalized heatstroke patients depends in large part on the rapidity of cooling on entry to a hospital intensive care unit. This time factor may be important because of the time required for the production of cytokines, which is in the range of minutes to hours. Seventeen Hadj patients with classic (nonexertional) heatstroke were admitted to a hospital an average of 2 hours after the onset of heatstroke.[59] Each victim's T_c was greater than 40.1°C (104.2°F). They suffered from delirium, convulsions, and coma. Plasma LPS concentrations ranged from 8 to 12 ng/mL, extremely high and in the potentially lethal range.[524] Furthermore, TNF and IL-1 concentrations were also

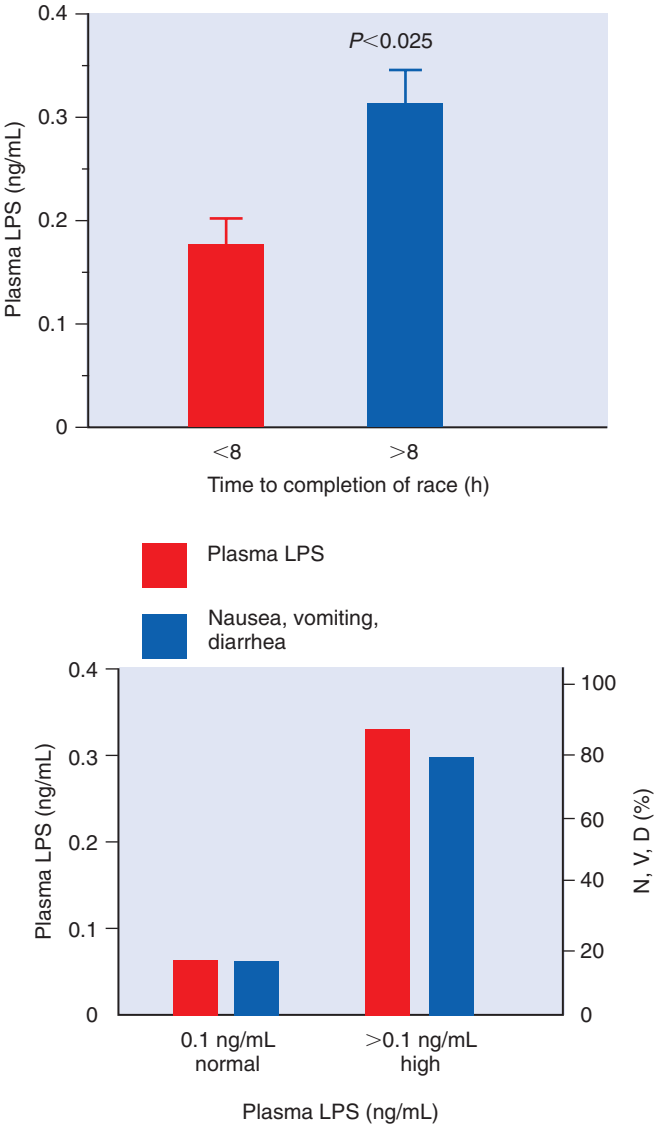

Figure 10-15. Role of lipopolysaccharide (LPS) and anti-LPS IgG in long-distance races. The Comrades' Marathon is an 89.5-km race between Pietermaritzburg and Durban, Republic of South Africa, run on a warm day. Blood samples were obtained from collapsed runners prior to treatment. On examination, they all looked the same. However, of 89 samples analyzed, 80% had elevated levels (>0.1 ng/mL) of LPS, and 20% had normal low levels. **Top:** Those with low levels of circulating LPS ran the double marathon significantly faster before they collapsed. **Bottom:** Those with low levels of circulating LPS had higher morbidity indexes of nausea, vomiting, and diarrhea and all recovered within 2 hours. Those with elevated levels of LPS had more serious symptoms and required up to two days to recover. (Redrawn from Brock-Utne JG, Gaffin SL, Wells MT, et al: S Afr Med J 73:533–536, 1988.)

very high. The authors suggested that these cytokines exacerbated the hyperthermia of heatstroke through induction of prostaglandins.

Nine of 11 heatstroke patients showed marked leukocytosis resulting from a large increase in the number of T suppressor cytotoxic cells (CD8) and NK cells (CD16/CD56).[58] This leukocytosis increased with increasing T_c. There were also substantial decreases in helper T cells (CD4) and B cells (CD19) as a result of heatstroke. Heatstroke is known to elevate catecholamine levels.[84] Because epinephrine causes leukocytosis with an increase in NK and T suppressor/cytotoxic cells, these authors suggested that heatstroke raised catecholamines, which in turn altered lymphocyte subpopulations.

On the other hand, hyperthermia causes elevated cortisol,[276] which causes the opposite effect, lymphocytopenia.[58] In the preceding study, two of the 11 heatstroke patients had a decreased number of lymphocytes. To account for the reduction in lymphocytes, the authors suggested that in those two patients the effects of cortisol, rather than of catecholamines, were dominant. That is, changes in subpopulations of lymphocytes in heatstroke may depend (on an individual basis) on the relative rises in concentration of catecholamines and cortisol, as well as on individual sensitivities to them. However, this is not yet clearly established.

TABLE 10-3. Elevated Cytokines Due to Heatstroke in 17 Military Recruits Who Developed Exertional Heatstroke*

	EXERTIONAL HEAT STROKED	CONTROLS†
Rectal temperature‡	41.2 ± 1.2°C (99.7 ± 1.4°F)	37.6 ± 0.8°C (106.2 ± 2.1°F)
IL-1β‡	3.1 ± 1.5 pg/mL	1.2 ± 0.8 pg/mL
TNF-α‡	4.9 ± 4.1 pg/mL	1.2 ± 2.4 pg/mL
IL-6‡	15.8 ± 3.2 pg/mL	1.2 ± 1.2 pg/mL
IFN-γ‡	7.3 ± 4.9 pg/mL	2.4 ± 4.1 pg/mL
IL-2r‡	1568 ± 643 pg/mL	610 ± 214 pg/mL
IL-4	2.5 ± 1.2 pg/mL	1.2 ± 0.8 pg/mL
IL-10	12.9 ± 9.4 pg/mL	2.5 ± 4.9 pg/mL
IL-8‡	84.2 ± 79.9 pg/mL	10.4 ± 3.2 pg/mL
Monocyte chemoattractant protein 1‡	959 ± 589 pg/mL	158 ± 217 pg/mL
RANTES‡	12,464 ± 10,505 pg/mL	5570 ± 2894 pg/mL

*The severity of exertional heatstroke was evaluated using a simplified acute physiology (SAP) score. Interleukin (IL)-6, interferon-gamma (IFN)-γ, and IL-2 receptor positively correlated with the SAP score. Among chemokines, only serum monocyte chemoattractant protein 1 was positively correlated with the SAP score ($r = .78$, $P < .001$). There was no correlation between either cytokines or chemokines and body temperature.

†Controls were 17 military recruits undergoing exercise who did not develop heatstroke.

‡*Conclusions:* Proinflammatory cytokines IL-1β, tumor necrosis factor (TNF)-α, IL-6; Th1 cytokines INF-γ and IL-2 receptor; and chemokines IL-8, monocyte chemoattractant protein 1, and RANTES (regulated on activation, normal T cell expressed and secreted) are increased in patients with exertional heatstroke. Helper T-2 cytokines may play a role as anti-inflammatory cytokines.

From Lu KC, Wang JY, Lin SH, et al: Crit Care Med 32:399–403, 2004.

Adaptation and the Immune System

Cytokines and Shock

Cytokines are a class of protein cell regulators produced by a wide variety of cell types throughout the body. They control timing, amplitude, and duration of the immune response.[99] They are relatively low molecular weight proteins (<80 kDa) and usually act at short range in a paracrine or autocrine manner, but they can also act as circulating hormones.[12] Cytokines interact with high-affinity cell-surface receptors regulating tran-

scription of several cellular genes, resulting in changes in cell behavior.[26] Various cytokines are extraordinarily active, have important and overlapping activities, and may induce each other. In the past, it was difficult to establish which cytokine had which critical function. Local concentration probably determines the regulatory influence a cytokine has at a particular time. After induction by a single stress, various cytokines may remain active for hours to days. Proinflammatory cytokines are largely cytotoxic. They appear designed primarily to destroy invading microorganisms by producing free radicals, and to increase vascular permeability by loosening contacts between adjacent cells in tissue to permit entry of macrophages. Nonspecific (innate) immunity is the default setting of the human immune system and provides for rapid, nonspecific actions against viruses, bacteria, and tumors, prior to the development of specific immunity. Reactive oxygen species produced in response to these cytokines may damage host cells at the same time they are destroying bacteria, thus slowing wound healing. Such nonspecific immune responses are regulated by antigen presenting cells (APCs)—monocytes/macrophages, dendritic cells, and NK cells. For a review of cytokines, see Elenkov.[148]

Antiinflammatory cytokines facilitate B-cell activation and production of antibodies, and they promote wound healing. Specific immunity is largely regulated by the Th1 and Th2 subclasses of helper T cells. These cells are mutually inhibitory so that, in general, the ascendant actions of one lead to the diminishing of activities of the other. Precursor cells to Th1 and Th2 cells are naive CD4 cells (not yet sensitive to antigens) or T0 cells. IL-12 is a powerful inducer of T0 cells into mature Th1 cells, and it increases nonspecific cellular immunity.

Th1 cells mainly secrete interferon-gamma (IFN-γ), IL-2, and TNF-α, which promote nonspecific cellular immunity. Cytokine expression altered by heatstroke is shown in Table 10-3. IL-12, TNF-α, and IFN-γ also stimulate synthesis of the powerful vasodilator nitric oxide, which aids in development of the inflammatory response. This group of cytokines (IL-12, TNF-α, and IFN-γ) is considered the main proinflammatory group to inhibit Th2 cell activities.

Th2 cells secrete different cytokines, mainly IL-4, IL-10, and IL-13, which promote humoral and specific immunity. IL-4 and IL-10, the major anti-inflammatory cytokines of the Th2 cells, promote humoral and specific immunity by stimulating (1) growth and activation of mast cells and eosinophils, (2) differentiation of B cells into antibody-secreting cells, and (3) B cells to switch to IgE production. Cytokines produced by Th2 cells also inhibit activation of macrophages, proliferation of T cells, and production of proinflammatory cytokines.

The end products of stress-induced activation of the hypothalamus-pituitary-adrenal axis, cortisol and other glucocorticoids, are selective immunosuppressors. Glucocorticoids produced in response to stresses such as heatstroke (but not necessarily the milder heat stress) increase Th2 activity mainly indirectly by inhibiting IL-12 production of APCs and causing loss of IL-12 responsiveness of NK and Th1 cells. To a lesser extent, glucocorticoids also directly suppress activity of Th1 cells by inhibiting their production of TNF-α, IFN-γ, and IL-2. Other hormonal products, such as epinephrine and norepinephrine, of heatstroke and other stresses also induce a shift from Th1 to Th2 cytokine production. Altogether, these end products of stimulation of the HPA axis are highly likely to increase susceptibility of a heatstroke victim to infections and influence the course of the infections.

Endogenous LPS leaking out of the lumen of ischemic intestines can cause septic shock and has a number of properties in common with glucocorticoids. But LPS itself is not a contact poison. Rather, LPS present in the bloodstream is first bound by LPS-binding protein (LPB) to form a complex, which circulates until it encounters a specific high-affinity LPS receptor, CD-14, on a macrophage.[460] Binding of CD-14 by the LPS-LBP complex initiates a cascade of reactions within the macrophage involving hypersecretion of proinflammatory cytokines. Because of their inappropriate and excessive local concentrations, inflammatory cytokines become highly toxic. Although the correlation between mortality and plasma concentration of any single cytokine is low in persons with sepsis, mortality correlates closely ($P < .001$) with a score consisting of a summation of the concentrations of individual cytokines.[83]

Most of the symptoms of gram-negative bacterial shock can be induced by injection of some purified cytokines alone. The first two (and most important) cytokines induced by LPS are TNF and IL-1.[104] TNF administration to humans causes fever, tachycardia, increase in stress hormones, and leukocytosis,[104] with the intensity of symptoms closely correlating with peak concentrations of TNF.

TNF rapidly induces IL-1, which causes fever, sleep, anorexia, and hypotension.[342] IL-1 acts directly on vascular endothelium to increase local concentrations of nitric oxide and to raise circulating prostaglandin concentrations, ultimately resulting in vasodilation, hypotension, and, possibly, shock.[12] IL-1 may also have a more subtle effect. It alters arterial responses to norepinephrine in different vascular beds.[33] It decreases norepinephrine-induced contraction by about 50% in the aorta, carotid, and pulmonary arteries, but it increases contraction in femoral arteries. By so doing, IL-1 may cause abnormalities in regional blood flow during sepsis.[424] Heatstroke raises circulating catecholamine concentrations, which, in the presence of IL-1, may redirect blood flow inappropriately.[1]

Summary of Cytokine Responses in Heat Illness. Collectively, several studies suggest that early in the pathophysiology of heat illnesses, proinflammatory cytokines rise and inflammatory processes occur; later, inflammatory cytokines decline and anti-inflammatory cytokines rise.

Kark and colleagues reported in Marine recruits that the frequency of heat illnesses during training increased not with the highest ambient temperature reached during exercise but with the temperature of the previous day.[277] That is, high T_{amb} may lead to a prolonged rise in persistent cytokines within the body, which in turn may lower the threshold for developing exertional heat illness during exercise the following day. High T_{amb} leads to elevated T_c with apoptosis of inappropriate cells, including intestinal mucosal cells, over the next several hours, permitting leakage of toxic bacterial contents from the lumen into the circulation, rendering the person at risk from exercise. The winners and losers of athletic competitions or battles are determined as much by the activity of the immune system through cytokine expression and susceptibility to heat illnesses as by skill, strength, and speed. A sick athlete or professional soldier may be defeated by an inferior but healthy opponent. Through appropriate training, an athlete may reach his or her peak muscular strength, cardiovascular capacity, coordination, and $\dot{V}O_2max$. However, at the same time, other environmental, psychological, or nutritional factors may degrade the immune system, and the athlete becomes more susceptible to an oppor-

tunistic infection, very rapidly becomes unfit for competition, and becomes at risk for heat illnesses.

Mammals possess a programmed universal response to various stressors known as the general adaptation syndrome. This includes responses to heat, trauma, hemorrhage, toxins, cold, infections, and nervous irritation.[461] Genomic studies described earlier may require reexamination of this excessively simplified point of view. Nevertheless, prolonged heat provokes the general adaptation syndrome, leading to shrinkage of immune tissues and organs, with a decrease in immune function.[173] Among various lymphoid subclasses, human NK cells and their precursors are especially sensitive to heat and are completely inactivated by a T_{amb} of 42°C (107.6°F) for 60 minutes.[271]

There are relatively few published studies of cytokine secretion following exercise and heat, although the numbers have increased recently because of the availability of commercial cytokine ELISAs. Early studies may have been hampered by problems in sampling tissue. Urine samples taken after a race showed large increases in interferon, TNF, IL-1, IL-6, and soluble IL-2r.[135] Failure to detect changes in plasma cytokine concentrations may have resulted from rapid renal clearance from the circulation. Rises in IL-1 were observed in human skeletal muscle after muscle-damaging eccentric exercise.[77] Ingestion of vitamin E prevented a rise in IL-1 in vitro from leukocytes obtained from persons subjected to severe exercise.[78] IL-1 causes muscle proteolysis by inducing branched-chain keto acid dehydrogenase. This is a rate-limiting enzyme for the oxidation of amino acids in skeletal muscle. As this enzyme increases in concentration, amino acids are progressively oxidized, leading to muscle protein breakdown. Severe exercise suppressed mouse lung macrophages and increased their susceptibility to infections, with greater morbidity and mortality than in moderately exercised mice.[84]

Plasma cytokine determinations in 17 military recruits who developed exertional heatstroke were compared with 17 undergoing the same exercise but who did not develop heatstroke.[338] Proinflammatory cytokines IL-1β, TNF-α, IL-6, INF-γ, IL-2 receptor, IL-8, monocyte chemoattractant protein 1, and RANTES (regulated on activation, normal T cell expressed and secreted) were elevated in the patients with heatstroke. IL-6, IFN-γ, IL-2, and serum monocyte chemoattractant protein 1 receptor positively correlated with a heatstroke severity index.

Fourteen consecutive patients with heatstroke were found to be immunosuppressed from depressions in the number of T and helper T cells, depressions in the number of CDIIa-, CDIIc-, CD44-, CD56-, and CD54-positive cells, and rises in T suppressor/cytotoxic T cells. (The many subpopulations of immune cells are usually defined by CD markers.) Cooling led to partial or complete normalization, further derangements, or overcorrection.[222] After a marathon race, leukocyte levels approximately tripled and cortisol concentration rose approximately fivefold.[101] The concentrations of NK and B cells fell, although no changes occurred in number of lymphocytes.

Adequate lymphocyte activity is required for normal muscle function. Overtraining may be harmful by lowering lymphocyte concentrations. A combination of intense physical training and the stress of competition renders some athletes immunosuppressed and more susceptible to infection. One possible mechanism of overtraining muscle injury is that glutamine induces HSPs that protect muscle cells, and overtraining reduces the amount of glutamine available for normal lymphocyte function.[468] There may be a point at which laboratory techniques can quantitatively determine that the disadvantage of immunosuppression is greater than any benefit from exercise training, and that exercise should be curtailed.[101]

HEAT-RELATED ILLNESSES: VARIANTS OF HEATSTROKE

A number of clinical situations arise in which the hyperthermia that develops is sufficiently severe (>40°C [104°F]) that it should be considered a subgroup of EHS. Neuroleptic seizures and overdose of recreational drugs share with EHS the features of massive muscle contractions (with consequent overuse of high-energy compounds) and rhabdomyolysis.[303] Because the use of recreational drugs is not expected to decline and the number of persons using neuroleptic drugs is probably on the increase, the involvement of heatstroke pathophysiology should be considered in treating those cases. In an unusual situation during mountaineering in summer, two persons died of heatstroke and acute rhabdomyolysis. Both patients had received treatment with antipsychotic drugs, including a phenothiazine.[306] Ergogenic aids are also a problem. A highly trained, heat-acclimatized infantry soldier suffered from exertional heatstroke during a 12-mile road march shortly after taking an ephedra-based supplement. Because there are no clear ergogenic benefits in using ephedra alone, clinicians and military commanders should strongly discourage the use of ephedra-containing substances in active duty soldiers undergoing strenuous exercise.[395] In summary, heatstroke in a summertime vacation area might be complicated by the use of therapeutic or recreational drugs or ergogenic aids.

Malignant Hyperthermia

Malignant hyperthermia is a rare life-threatening disorder involving hypermetabolism, rapid rise in body temperature, and rigidity of skeletal muscle. It is induced by exposure to volatile anesthetics during surgical procedures in affected patients. In about half these patients, mutations were seen in the gene for the Ca^{2+} release channel (RyR).[107,213,323] The anesthetic binds to RyR and activates the Ca^{2+} release channel, causing massive calcium entry into the cytoplasm. This activates contractile proteins, calmodulin, and a variety of calcium-sensitive enzymes, which leads to muscle rigidity, hypercatabolism, fulminating hyperthermia, and metabolic acidosis.[107]

Rhabdomyolysis, hyperkalemia, and myoglobinemia[458] are commonly associated with malignant hyperthermia, with plasma K^+ rising as high as 10 mmol/L.[357] Underlying illnesses in five cases of rhabdomyolysis included heatstroke, high fever, and grand mal seizures with associated hyperthermia. Nevertheless, there were multiple factors responsible for rhabdomyolysis in each case, such as hypokalemia, hypophosphatemia, shock, and arteriosclerosis.[378] A 41-year-old man susceptible to malignant hyperthermia developed an infection and self-medicated with a cold medicine. He presented with high fever, dysarthria, dysphagia, and progressive weakness of his muscles and developed massive rhabdomyolysis with acute renal failure.[279]

Neuroleptic Seizure

Treatment of psychiatric patients with neuroleptic drugs, as well as with antidepressants, antiemetics, and others,[141] may lead to

the uncommon but often fatal neuroleptic malignant syndrome, characterized by hyperthermia as high as 42° C (107.6° F), "lead pipe" (skeletal muscle) rigidity, dyspnea, coma, extrapyramidal syndrome, rhabdomyolysis, severe metabolic acidosis, leukocytosis, and elevated creatine kinase.[104,234,270] A number of factors predispose to neuroleptic seizure, including dehydration, exhaustion, aggression, and restraints[265]; high environmental temperature; high doses of neuroleptics; abrupt discontinuation of antiparkinsonism agents; and administration of lithium.[141] Successful treatment of these cases includes immediate withdrawal of the drug, administration of dantrolene, and either oral bromocriptine or the combination of levodopa and carbidopa.[141]

Drug Overdose

Although the toxicity of drug overdose is well recognized, it is not often appreciated that the hyperthermia attained can be in the range reported for heatstroke. Such hyperthermia has been induced with cocaine[130] and amphetamine derivatives, such as 3,4-methylenedioxymethamphetamine (MDMA, Ecstasy) and 3,4-methylenedioxyethamphetamine (MDEA, Eve).[504] Other components of this syndrome include hyperkalemia, rhabdomyolysis,[475] sympathetic hyperactivity, convulsions, rectorrhagia, psychosis, disseminated intravascular coagulation in the absence of positive blood cultures, and acute renal failure.[237]

Susceptibility to Heatstroke

There may be an inherited susceptibility to EHS. Muscle biopsy specimens taken from two men in military service who had recovered from EHS had abnormal responses to halothane, a well-known cause of malignant hyperthermia.[213] Furthermore, muscles from members of their families had abnormal responses to halothane or ryanodine, a drug that binds to the Ca^{2+} release channels of the sarcoplasmic reticulum.[8] A ryanodine contracture test has been proposed as an in vitro diagnostic test to screen for surgical patients susceptible to malignant hyperthermia.[8] This test might be useful in identifying, retrospectively, a possible subgroup of patients with EHS.

Changes in Cognitive Function

Changes in cognitive function appear to occur before development of physical symptoms associated with heat stress.[82] Typically, heat stress causes distortion of the sense of time,[36,37,112] memory impairment,[540] deterioration in attention, and decreased ability to calculate mathematical problems.[92,209,539] Health care personnel should be trained to recognize that confusion, changes in affect, and impaired ability to function in the work environment can be early signs of heat injury under heat stress conditions.[82]

Vasovagal Syncope

Syncope is the cause of about 3% of emergency department visits and 6% of hospital admissions.[193] Vasovagal syncope is responsible for 28% to 38% of syncopal episodes in patients 35 to 39 years old.[115,274,348] Benign presyncope or syncope may result from diminished venous return to the heart because of blood pooling in the peripheral circulation. Syncope encompasses psychological disturbances activating an autonomic vasodilation response; reflex syncope caused by heavy coughing, micturition, or pressure on an irritable carotid sinus; and reduced vasomotor tone caused by hypotensive drugs or alcohol.[144]

The frequency of vasovagal syncope is greater in the young than in older adults, whereas orthostatic hypotension is more common in older persons.[334] Propranolol does not prevent the vasovagal reaction in response to head-up tilt.[334] Therefore, after the age of 40, presyncope may suggest a more serious condition, such as gastrointestinal bleeding, myocardial or valvular heart disease, or severe anemia. Cardiovascular syncope resulting from arrhythmia carries a 1-year mortality rate of about 30%.[275]

Hyperventilation Dizziness

A slight but prolonged increase in respiratory rate or tidal volume may accompany an increase in anxiety.[144] This can lead to increased blood oxygen content and decreased P_{CO_2}, with accompanying alkalosis. Altogether, these lead to generalized cerebrovascular vasoconstriction with ischemia and dizziness.

Heat-Induced Syncope

The associated clinical syndromes vary in severity depending on the cause of hyperthermia and, therefore, so does the duration of central nervous system (CNS) dysfunction. Transient or temporary loss of consciousness associated with a mild form of heat syncope has its origins primarily in the cardiovascular system. It is a consequence of a reduced effective blood volume rather than an actual loss of volume.

In an upright and stationary person, blood volume is displaced into the dependent limbs by gravity. If that person is also heat stressed, more blood is displaced into the peripheral circulation to support heat transfer at the body surface. These combined reductions in effective blood volume can temporarily compromise venous return, cardiac output, and cerebral perfusion. Patients are usually erect at the outset and sometimes report prodromal symptoms of restlessness, nausea, sighing, yawning, and dysphoria.[293] Hypotension results predominantly from vasodilation and bradycardia. This systemic disorder is self-limited because when the person faints and assumes a horizontal position, central blood volume is restored, cardiac filling rises, blood pressure is restored, and the problem is remedied.

Transient loss of consciousness in syncope has a metabolic basis within ischemic cells of the brain. Despite this, the effects, although startling to onlookers and frightening to the patient, appear to be readily reversible. There is no risk of direct thermal injury to brain cells complicating the circulatory origin of this sudden decline in effective arterial volume.

The incidence of syncopal attacks falls rapidly with increasing days of work in the heat (see Figure 10-10), suggesting the importance of salt and water retention in preventing this disorder (Table 10-4). Individuals medicated with diuretics would be at high risk. Furthermore, potassium depletion and hypokalemia may lower blood pressure and blunt cardiovascular responsiveness.[305] In stark contrast to simple syncope is the profound CNS dysfunction dominating the early course of heatstroke. Thus, if a person faints in a setting where hyperthermia is possible and does not rapidly return to consciousness, heatstroke should be suspected, cooling measures instituted, and body temperature monitored.

Exertion-Induced Syncope, Cramps, and Respiratory Alkalosis

During basic military training, a cluster of 17 syncopal episodes was associated with a seldom-described form of heat exhaus-

TABLE 10-4. Signs and Symptoms of Salt and Water Depletion Heat Exhaustion

SIGNS AND SYMPTOMS	SALT DEPLETION HEAT EXHAUSTION	WATER DEPLETION HEAT EXHAUSTION	DILUTIONAL HYPONATREMIA
Recent weight gain	No	No	Yes
Thirst	Not prominent	Yes	Sometimes
Muscle cramps	In most cases	No	Sometimes
Nausea	Yes	Yes	Usually
Vomiting	In most cases	No	Usually
Muscle fatigue or weakness	Yes	Yes	No
Loss of skin turgor	Yes	Yes	No
Mental dullness, apathy	Yes	Yes	Yes
Orthostatic rise in pulse rate	Yes	Yes	No
Tachycardia	Yes	Yes	No
Dry mucous membranes	Yes	Yes	No
Increased rectal temperature	Yes	In most cases	No
Urine Na^+/Cl^-	Negligible	Normal	Low
Plasma Na^+/Cl^-	Below average	Above average	Below average

*Data from Armstrong LE, Curtis WC, Hubbard RW, et al: Med Sci Sports Exerc 25:543–549, 1993; Shopes E: Water intoxication: Experience from the Grand Canyon (abstract). Presented at the 10th Annual Meeting of the Wilderness Medical Society, August 1994, Squaw Valley, Idaho, p 265.

TABLE 10-5. Clinical Data for 17 Patients with Heat Exhaustion

CASE NO.	AGE	Activity	SYNCOPE	CRAMPS	T_{re} (°F)	RR (bpm)	Na^+ (mEq/L)	pH	P_{CO_2} (mmHg)
1	19	Marching	Yes	Abd	99.6	24	142	—	—
2	20	Running mile	Yes	Legs/abd	98.4	30	145	—	—
3	20	Rifle range	No	No	99.4	24	143	—	—
4	21	Marching/running	Yes	Hands	100.4	22	162	7.47	34.0
5	21	Marching	No	Severe abd/legs	102.4	35	141	7.50	32.4
6	22	Marching	No	Legs	100.0	22	152	7.70	14.8
7	20	Rifle range	No	Mild	100.0	22	140	—	—
8	20	Marching	Yes	Abd/legs	100.8	30	145	7.52	28.8
9	20	Marching	No	Abd	101.4	24	—	7.69	19.8
10	18	Marching	Yes	Chest	100.8	18	140	7.56	29.4
11	19	Marching	Yes	Tetany	101.5	30	160	7.44	34.2
12	18	Marching	Yes	Severe	100.6	30	130	7.71	17.2
13	20	Marching	No	Mild	98.6	26	141	7.77	15.2
14	19	Marching	Yes	Abd/legs	100.7	30	145	7.76	16.3
15	23	Rifle range	Yes	Abd/legs	101.0	32	148	7.66	19.7
16	18	Marching	Yes	Chest/legs	101.2	28	148	7.78	14.7
17	17	Marching	Yes	Abd	101.6	22	146	7.53	28.4

Abd, abdomen; RR, respiratory rate; T_{re}, rectal temperature.
From Boyd AE, Beller GA: Acid-base changes in heat exhaustion during basic training. Proc Army Sci Conf 1:114, 1972.

tion (Table 10-5).[260] This heat exhaustion, in contrast to hypovolemic salt depletion, was characterized by hyperventilation, respiratory alkalosis, syncope, and tetany. Most victims also experienced abdominal cramps, yet this was independent of lactic acidosis and hyponatremia. These descriptions were unique in that the heat syncope episodes were not those classically described as the venous pooling or postural hypotension variety.[32,387] The incapacitated trainees arrived at a heat ward within 10 to 30 minutes of the onset of symptoms, and blood samples were drawn immediately on admission. They exhibited moderate to marked respiratory alkalosis, but only two appeared to be severely dehydrated; nearly all (16 of 17 patients) had severe cramps of the abdominal or extremity muscles.

Clinical data recorded on admission are shown in Table 10-5. Almost all the casualties occurred in the afternoon during July, 1971, at Fort Polk, Louisiana. All were diagnosed with heat exhaustion resulting from training in the field (12 of 17 while speed marching). Rectal temperatures on admission were elevated, even though most of the victims had been doused with water before evacuation from the field. Serum electrolytes were in the normal range in the majority of victims. However, hemoconcentration with elevated serum sodium level was observed in four patients. One of 17 patients had a low serum sodium level and also experienced severe muscle cramps.

The majority of these patients were not water or salt depleted, and 15 of the 16 remaining patients with cramps had normal to elevated serum sodium and chloride levels. The mean arte-

TABLE 10-6. Protective Effect of Hypertonic 10% Albumin versus Saline on Experimental Heatstroke-Induced Cerebral Ischemia-Hypoxia Injury in Amelioration of Rises in Glutamate, Glycerol, Lactate, and Free Radicals in Brain

	SALINE	10% ALBUMIN
Mean arterial pressure	42 ± 3 mm Hg	64 ± 6 mm Hg
Intracranial pressure	33 ± 3 mm Hg	10 ± 2 mm Hg
Cerebral perfusion pressure	9 ± 3 mm Hg	54 ± 5 mm Hg
Cerebral blood flow	109 ± 20 blood perfusion units	452 ± 75 blood perfusion units
Brain Po_2	6 ± 1 mm Hg	15 ± 2 mm Hg
Striatal glutamate	51 ± 7 nM	3 ± 2 nM
Striatal glycerol	24 ± 3 nM	4 ± 2 nM
Striatal lactate/pyruvate ratio	124 ± 32	7 ± 3
Striatal hydroxyl radicals	694 ± 22% rise	119 ± 7% rise
Striatal neuronal damage score	2.25 ± 0.05	0.38 ± 0.05

Adapted from Chang CK, Chiu WT, Chang CP, Lin MT: Clin Sci (Lond) 106:501–509, 2004.

rial pH for this group of patients was 7.62 ± 0.03 (SEM), and five had a pH of 7.67 or greater. Arterial Pco_2 was reduced to a mean value of 23.5 ± 2 mm Hg. Thus, all patients had moderate to marked respiratory alkalosis, and nine had obvious tetany with carpopedal spasm.[64] The presence of carpopedal spasm and paresthesias in the distal extremities and perioral area helps distinguish this form of cramps from the classic variety.

These data associate exertion-induced heat exhaustion with a form of respiratory alkalosis characterized by syncope, tetany, and muscle cramps and may possibly be the result of "an exaggeration of the normal physiologic ventilatory response to thermal extremes."[64] Hyperventilation with its resulting decrease in cerebral blood flow[288,471,525] could account for a significant number of cases of exercise-induced heat syncope. Recumbency, rest, and oral replacement of fluid and electrolyte deficits are usual recommendations. Rebreathing of expired air is directed at alleviating carpopedal spasm, but it should be done with extreme caution because of its hypoxemic effect.

Classic syncope is usually associated with postural hypotension, whereas heat exhaustion and heat cramps are usually associated with water and electrolyte imbalance. Most literature suggests that unacclimated workers have higher salt losses in the heat than those who are acclimated.[317,325] Thus this series is a good example of the real world with a mixed bag of heat illness symptoms. To explain these clinical results, one should recall that acclimated individuals have higher sweat rates (2.5 L/hr versus 1.5 L/hr) than unacclimated persons, but they also have increased tolerance to exercise. If both groups voluntarily work at maximal sweat rates for any given task, those who are heat acclimated could produce higher salt losses despite their reduced sweat sodium concentrations. In such a scenario, the acclimated individuals would be predicted to be the more prone to heat cramps.[10] However, Table 10-6 indicates that there are higher salt losses for unacclimated individuals at any given sweat rate or volume of sweat lost. The differential diagnosis of heat cramps should also include exercise-induced peritonitis.[500]

Heat-Induced Tetany

In excessively hot environments, men at rest hyperventilate.[216] Adolph and Fulton[2] described dyspnea and tingling in the hands and feet of men being dehydrated in the heat. In 1941, during a voyage through the intense heat of the Persian Gulf, a ship's engineer was reported to experience spontaneous hyperventilation and attacks of tetany.[541] He could reproduce these symptoms simply by deliberately overbreathing. This appeared to be the first clinical description of heat-induced hyperventilation tetany.

The exposure of male test subjects to hot, wet conditions led to physiologic changes and the onset of symptoms ranging from slight tingling of the hands and feet to more severe carpopedal spasm.[260,261] The frequency and severity of symptoms were apparently not related to the absolute change in the four measured parameters (Pco_2, CO_2, pH, and T_{re}) but rather to the *rate* of change. When the subject's tolerance time was short, changes occurred rapidly and the incidence of symptoms was high; conversely, when the tolerance time was long, the same degree of change occurred but the incidence of symptoms was low. It was suggested that rapid changes of the four parameters lead to imbalance between intracellular and extracellular compartments and that this imbalance may be one of the factors inducing symptoms. Again, treatment consists of rest, cooling, and rebreathing expired air.

Heat Cramps

Heat cramps typically occur in conditioned athletes who compete for hours in the sun. They can be prevented by increasing dietary salt and staying hydrated.[147] Heat cramps are brief, intermittent, and often excruciating; muscle contractions are a frequent complication of heat exhaustion and occurred in about 60% of 969 cases of heat exhaustion.[102,325,495] The term *heat cramps* is a misnomer because heat itself does not cause them; rather, they occur in muscles subjected to intense activity and fatigue. The victim with salt depletion at the time of heat exhaustion is clearly ill and has numerous symptoms other than cramps. Furthermore, fatigue, giddiness, nausea, and vomiting are common and may occur before and more prominently than cramps. Sometimes, heat cramps occur as the only complaint, with minimal systemic symptoms. Furthermore, there is a difficulty in distinguishing abdominal heat cramps from gastrointestinal upset.

During the 1930s, steel workers, coal miners, sugar cane cutters, and boiler operators were among the most common victims of classic heat cramps.[502] Three factors common to most reports are that cramps are preceded by several hours of sustained effort, they are accompanied by heavy sweating in hot surroundings, and they are combined with ingestion of large volumes of water. A fourth factor (see later discussion) may be cooling of the muscles. Serum Na^+ levels ranged from 121 to 140 mEq/L (normal, 135 to 145 mEq/L).[502] In an industrial setting, heat cramps occur most commonly late in the day, after physical activity has ceased; they sometimes occur while a person is showering and occasionally occur in the evening.[325] Hyponatremia and hypochloremia are diagnostic of heat cramps that might be due to salt deficit or some degree of water intoxication.[305] If overdrinking causes gastric distention, nausea[420] could trigger vasopressin release and contribute to renal water retention.

Classic heat cramps are distinguished from hyperventilation-induced tetany in that they are not generalized but are limited to contractions of voluntary skeletal muscles subjected to prior exertion, and they usually affect only a few muscle bundles at a time. Nevertheless, the pain can be excruciating in severe cases. As one bundle relaxes, an adjacent bundle contracts for 1 to 3 minutes. The cramp thus appears to wander over the affected muscle. Three precipitating conditions (exhaustive work, hemodilution, and cooling the muscle) can each depolarize muscle cells.[305] This could explain the association of cramps with showering in cool water, because cooling slows the Na^+,K^+-ATPase pumps and depolarizes the cell, which may thus reach excitation threshold.[477] Heat cramps do not occur at the same frequency in all populations. For example, the Indian Armed Forces has a very low incidence[340] and Shibolet observed no cases within Israeli Defense Forces.[472] These data suggest that heat-acclimated individuals are less likely to experience them. This is consistent with the observation that the incidence was greatest during the first few days of a heat wave.[502]

Heat cramps generally respond rapidly to sodium chloride solutions. Mild cases may be treated orally with 0.1% to 0.2% NaCl solutions (two to four 10-grain salt tablets [56 to 112 mEq] or $^1/_4$ to $^1/_2$ teaspoon of table salt dissolved in a quart of water). Cooling and flavoring enhance palatability. Oral salt tablets are gastric irritants and not recommended. In severe cases, IV isotonic saline (0.9% NaCl) or small amounts of hypertonic saline (3% NaCl) are administered by physicians for rapid relief.

Heat Exhaustion

Classic heat exhaustion is a manifestation of cardiovascular strain resulting from maintaining normothermia in the heat. Symptoms of heat exhaustion include various combinations of headache, dizziness, fatigue, hyperirritability, anxiety, piloerection, chills, nausea, vomiting, heat cramps, and heat sensations in the head and upper torso.[20,21,256] Clinical descriptions include tachycardia, hyperventilation, hypotension, and syncope. Although the boundary between heat exhaustion and heatstroke is usually defined as 39.4° to 40°C (102.9° to 104°F), the differential diagnosis is often tenuous[229] or even considered artificial.[472] The victim may collapse with either a normal or an elevated temperature (severe cases, around 40°C), usually with profuse sweating. Spontaneous body cooling can occur, which is not prominent in severe heatstroke. The clinical determination of heat exhaustion is primarily a diagnosis of exclusion.

Classic heat exhaustion, like classic heatstroke, tends to develop over several days or longer and presents ample opportunity for development of imbalances in electrolytes and water. The hyponatremia and hypochloremia of patients with either heat cramps or salt-depletion heat exhaustion often develops over 3 to 5 days[324] and usually in the unacclimatized individual who has not fully developed salt-conserving mechanisms.[482,483] In salt-depletion heat exhaustion, muscle cramps, nausea, and vomiting may be intense, but victims do not feel very thirsty.[14,256] The major cause of fluid and electrolyte imbalance (salt depletion, water depletion, water intoxication) involved in a particular case of heat exhaustion can be discovered from the history of events surrounding collapse.[75,305,325,516]

Other forms of heat exhaustion are characterized by the type of fluid or electrolyte deficit (primarily pure water or salt deficiency), their underlying causes (prolonged heat exposure versus intense, short-term exertion), the intensity of hyperthermia, and the absence or form of CNS disturbance. If external cooling does not rapidly lower T_c to normal or, in fact, precipitates severe shivering, intercurrent illness is suspected. Anecdotal experience in the field suggests that approximately 20% of persons with suspected heat exhaustion have some form of viral or bacterial gastroenteritis. This is especially likely if nondisinfected water or ice has been consumed.

At any given loss of body weight, the decrement in plasma volume increases with the salt content of sweat. This would be the case for relatively unacclimated individuals. On the other hand, the more dilute the sweat (approaching a pure water deficit) and therefore the greater the retention of salt, the greater the increase in osmolality, plasma sodium, and thirst. Table 10-4 compares and contrasts the signs and symptoms of salt and water depletion heat exhaustion with dilutional hyponatremia. It is clear that at some point both syndromes share many symptoms. Vomiting and cramps appear to signal a significant sodium deficit, in addition to some degree of water deficit.

Heat Illness and Coexistent Disease

Coexistent illness or infection predisposes an individual to heatstroke.[195] In one study of heat illnesses, 11.2% of patients[537] also had gastrointestinal (choleraic) illness.[537] The reverse is also true: heat waves produce excess deaths of people with all categories of disease. For example, in one heat wave week in New York in the late summer of 1948, deaths from cardiovascular diseases and diabetes more than doubled (1364 versus 585), and pneumonia deaths tripled.[151]

Infection predisposes to heat illness, and heat stress exacerbates infections, leading to greater morbidity and mortality.[329] Relatively few studies have been reported on the susceptibility to infection during heat exposure, or the influence of infection on heat tolerance; there is need for further research into the effects of diseases on thermoregulation.[286]

POSSIBLE NEW THERAPIES

IL-1 Receptor Antagonist

Heatstroke of rats causes hypotension that may be related to the production of inflammatory cytokines such as IL-1. Therefore, interference with the IL-1 pathways by blocking the IL-1 receptor appeared to be a promising means of improving heatstroke symptoms and survival. A single injection of IL-1 recep-

tor antagonist (IL-1ra) immediately after the onset of heatstroke in rats blunted the hypotension response to heat, and the rats survived much longer (91 minutes versus 17 minutes) than controls.[94] With continuous perfusion of IL-1ra, the survival time increased to 10 hours from the onset of heatstroke. Because heatstroke may lead to a time-dependent shift of Th1 to Th2 cytokine production, timing of such a therapy would be critical, and difficult to perform safely in the field. Currently, the beneficial effects of IL-1ra have not been proven in humans, and the authors do not recommend such therapy. However, if clinical trials should prove its effectiveness, then after initiating cooling procedures and volume therapy, and after transportation to a hospital, administration of IL-1ra may be become part of heatstroke therapy.[94]

Insulin

Rats were rendered diabetic (by streptozotocin) and then heatstroked by exposure to a T_{amb} of 43°C (109.4°F) for 60 min. Administration of insulin to the diabetic rats attenuated the high core temperature and heart rate, and improved cerebral blood flow and hypotension.[388]

Free Radical Scavengers

Part of the pathophysiology of heatstroke involves production of highly toxic free radicals, or reactive oxygen species. Therefore, administration of scavengers of free radicals seems an obvious choice for therapy.[550] Magnolol, obtained from the plant *Magnolia officinalis,* is a potent free radical scavenger that is 1000-fold more active than α-tocopherol in inhibiting lipid peroxidation in rat mitochondria. In rats heatstroked by exposure to a T_{amb} of 42°C (107.6°F), magnolol (20 mg/kg, IV) attenuated the pathophysiology associated with heatstroke.[87] This pharmaceutical is not recommended for therapy until it undergoes clinical trials and has been proven successful in humans.

Hypertonic Albumin

Heatstroked rats were treated with either 10% albumin or an equal volume of saline, and cooled by exposure to a T_{amb} of 24°C (75.2°F) for 12 min. As seen in Table 10-6, hypertonic albumin had a neuroprotective effect compared with saline controls in reducing elevations of glutamate, glycerol, lactate, and free radicals in the brain.[86] The mechanism for this is not understood. It is somewhat counterintuitive, as a rat would normally be expected to be rendered hyperosmotic by heatstroke.

ω-3 Fatty Acids

At the biochemical level, binding of IL-1 to its specific membrane receptor activates G proteins, which increase intracellular concentration of cyclic adenosine monophosphate (cAMP), which in turn activates membrane phospholipases. Activation of the phospholipases produces arachidonic acid, leukotrienes, prostaglandins, and thromboxanes, ultimately leading to cell damage and organism pathophysiology.[88,199] The phospholipases hydrolyze phospholipid esters of fatty acids, which in western diets are largely ω-6 fatty acids.[263,321] The ω-6 fatty acids are hydrolyzed into the key metabolite, arachidonic acid.

Cells contain two major enzyme classes that can act on arachidonic acid: lipoxygenases and cyclooxygenases. Lipoxygenase acts on arachidonic acid to enter a pathway that results in the formation of 5-hydroperoxyeicosatetraenoic acid (5-HPETE) and a series of toxic leukotrienes. Of these, LTB_4, LTC_4, and LTD_4 are the most important. LTB_4 induces inflammation, increases capillary leakage, and causes leukocytes to aggregate. LTC_4 and LTD_4 are potent bronchoconstrictors involved in asthma.[105,223] Cyclooxygenase converts arachidonic acid into prostaglandin G_2 (PGG_2), which is converted into PGH_2 with the formation of toxic free radicals. PGH_2 is a central metabolite on which a variety of enzymes act to form mainly toxic products, such as thromboxane A_2 (TxA_2) and many different prostaglandins, including the toxic PGD_2. TxA_2 causes platelets to aggregate, is a strong vasoconstrictor, and increases capillary leakage. To a person in shock or with another circulatory disorder, such agents could convert a severe but treatable condition into a lethal one. In summary, eating a normal Western diet results in the presence of large amounts of ω-6 fatty acids in phospholipid cell membranes, predisposing to the formation of arachidonic acid and a large number of its toxic metabolites. For a review of prostaglandin and thromboxane biochemistry, see references.[57,371,391]

In fish-enriched diets laden with ω-3 fatty acids, a high proportion of the ω-6 fatty acids in plasma membranes are replaced by the ω-3 fatty acids, phospholipases hydrolyze the phospholipids into eicosapentaenoic acid, and no arachidonic acid is produced. Therefore, no strongly toxic thromboxanes, prostaglandins, and leukotrienes are produced. Instead, only slightly toxic leukotriene B_5 is formed.

A diet enriched in fish oil (rich in ω-3 fatty acids) dramatically downregulates key immunoregulatory cytokines involved in autoimmune disease.[267] Mice fed fish oil for 6 weeks showed reduced fever and weight loss caused by LPS injection and did not have the rise in PGE_2 that normally results from LPS activity. These changes suggest that the pathophysiology induced by toxic arachidonic acid metabolites can be reduced or prevented by dietary replacement of ω-6 fatty acids with ω-3 fatty acids.[310] However, there was an exaggerated rise in TNFα, a toxin in itself, possibly because of the lack of the negative feedback from PGE_2. Therefore, although these studies appear promising, they must be interpreted with extreme caution and are not recommended to guide prophylaxis for heatstroke. There has not yet appeared a clinical study showing that injection or ingestion of ω-3 fatty acids protects humans against heat illnesses. However, supplementing a normal Western diet with fish oil capsules replaces a significant proportion of ω-6 fatty acids by ω-3 fatty acids in human cell membranes within 6 weeks, and possibly much sooner.[153]

Cyclooxygenase Inhibitors

Fever, in contrast to exercise hyperthermia, represents a physiologic state in which the hypothalamic thermostat has been reset above 37°C (98.6°F) by exogenous pyrogens released from bacteria or viruses[131] or by IL-1,[150,270,372] IL-2, or interferon-α and interferon-β.[50]

Cytokines may be responsible in part for other clinical symptoms of fever, including fatigue, malaise, and edema. α-Melanocyte-stimulating hormone inhibits IL-1-induced fever and the acute phase response.[335] Neutralizing antibodies to IL-1 and TNF have been found in the sera of both normal and sick individuals and may play a role in their regulation.[501] Current evidence suggests that aspirin-like cyclooxygenase inhibitors interfere with IL-1-induced fever or shock responses by inhibiting prostaglandin synthesis.[397] Nevertheless, in an experimental model of heatstroke, rats were pretreated with aspirin and survived longer than did controls.[254]

Defervation

Circulating LPS reaches the thermoregulatory control center in the anterior hypothalamus, activates cyclooxygenase, and induces prostaglandins.[244,358] LPS is also bound by the liver, where it stimulates the vagus nerve to signal the hypothalamus to produce prostaglandins.[433] At the onset of fever, a patient often feels chilled and shivers to elevate T_c by additional metabolic heat. A new, higher, preferred ambient temperature is behaviorally established.[393] The physiologic change is even more important. Once this new set point temperature is established, the thermoregulatory center uses all available thermoregulatory mechanisms to maintain it. As a result, attempts at whole-body cooling are met with sensations of extreme discomfort and violent shivering. When unsuccessful attempts to cool patients who have suspected heat illness result in chills and violent shivering, coexistent infection or disease is suggested.

This prostaglandin-mediated pathway may be responsible for fever, normal circadian temperature variation, pathologic temperature elevations, and temperature elevations related to stress.[41,298] Although there may be pyrogens that do not act via prostaglandins,[113,262] treatment, if necessary, should be directed at agents that block the action of the pyrogen at the hypothalamic receptor sites.

External application of cold to reduce true fever may be counterproductive[497] and is often ineffective, even after antipyretic therapy.[27,385] The body defends the higher temperature set point against environmental cooling. Therapy for fever that uses agents to block the causative molecular interaction is the most rational and clinically effective approach. Aspirin and other antipyretic agents, such as acetaminophen, indomethacin, ibuprofen, and other newer nonsteroidal anti-inflammatory compounds, are effective and act either directly or indirectly through inhibition of the prostaglandin mechanism.[114,372] The normal febrile response is generally self-limited in both magnitude and duration.[533] Vasopressin[521] and melanotropin[382] appear to act centrally to suppress temperature elevation and may be important in preventing extreme hyperthermia.

Should Antipyretic Therapy Be Routine?

High temperatures enhance resistance to viral and bacterial infections in experimental animals.[38,81,174] For example, replication of DNA viruses is inhibited by mild hyperthermia,[194,242,337] and measles virus membrane protein is selectively blocked by heating cultures to 39°C (102.2°F).[394] Some host defense functions[438,518] that become more effective at 40°C (104°F) than at 37°C (98.6°F) level off or diminish at 42° or 43°C (107.6° or 109.4°F).[497] Although fever has long been recognized as a manifestation of disease[24] and may be identified as a debilitating problem even in the absence of other signs or symptoms,[527] antipyretic therapy should not be instituted routinely for every febrile episode.[165,206] Furthermore, although administration of aspirin lowers fever by altering the thermoregulatory set point in the hypothalamus, it also leads to a greater rise in T_c in response to a standardized heat stress and therefore is not a universal temperature-lowering agent.[156]

In summary, at T_c up to approximately 40°C (104°F), the febrile process has a role in host defense, and routine antipyretic therapy for fever is generally unnecessary and may be harmful,[497] especially because of the link between aspirin and Reye's syndrome.[497] Instead, treatment should be based on evaluation of relative risks.[49,139,297]

The references for this chapter can be found on the accompanying DVD-ROM.

11 Clinical Management of Heat-Related Illnesses

Daniel S. Moran and Stephen L. Gaffin

This chapter discusses clinical observations of heatstroke victims and management of heat-related illnesses. In heatstroke, the most severe heat illness associated with excess body heat, early clinical signs are nonspecific. A common picture of heatstroke is sudden collapse of an individual during physical activity in a warm environment. This is usually followed by loss of consciousness with elevated core temperature (T_c) greater than 40°C (104°F), rapid heart rate (HR), tachypnea, hypotension, and, possibly, shock. Severity of heat illness depends on the degree of elevation in T_c and its duration. Heatstroke is an extreme medical emergency that can be fatal if not diagnosed and treated promptly. Therefore, to prevent and minimize complications and save lives, proper management and clinical care are essential.

This chapter focuses on the three different phases of heatstroke (acute, hematologic and enzymatic, and late), problems with recognition of heat illnesses, diagnosis and complications of heatstroke, treatment, and awareness of risk factors.

Defervation

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This chapter focuses on the three different phases of heatstroke (acute, hematologic and enzymatic, and late), problems with recognition of heat illnesses, diagnosis and complications of heatstroke, treatment, and awareness of risk factors.

TABLE 11-1. Comparison of Classic and Exertional Heatstroke

CHARACTERISTICS	CLASSIC	EXERTIONAL
Age group	Older adults	Men (15–45 yr)
Health status	Chronically ill	Healthy
Concurrent activity	Sedentary	Strenuous exercise
Drug use	Diuretics, antidepressants, antihypertensives, anticholinergics, antipsychotics	Usually none
Sweating	May be absent	Usually present
Lactic acidosis	Usually absent; poor prognosis if present	Common
Hyperkalemia	Usually absent	Often present
Hypocalcemia	Uncommon	Frequent
Hypoglycemia	Uncommon	Common
Creatine phosphokinase	Mildly elevated	Markedly elevated
Rhabdomyolysis	Unusual	Frequently severe
Hyperuricemia	Mild	Severe
Acute renal failure	<5% of patients	25% to 30% of patients
Disseminated intravascular coagulation	Mild	Marked; poor prognosis
Mechanism	Poor dissipation of environmental heat	Excessive endogenous heat production and overwhelming of heat-loss mechanisms

Modified from Knochel JP, Reed G: In Kleeman CR, Maxwell MH, Narin RG (eds): Clinical Disorders of Fluid and Electrolyte Metabolism. New York, McGraw-Hill, 1987, pp 1197–1232.

CLINICAL AND LABORATORY OBSERVATIONS IN HEATSTROKE

Clinical manifestations of heatstroke vary, depending on whether the victim suffers from classic heatstroke, which is a common disorder of older adults during heat waves and occurs in the form of epidemics, or exertional heatstroke (EHS), which occurs when excess heat generated by muscular exercise exceeds the body's ability to dissipate it (Table 11-1). Some overlap in presentation may occur; treatment with a medication that places an older adult at risk for classic heatstroke also places an exercising individual at risk for EHS. The clinical picture of heatstroke usually follows a distinct pattern of events with three phases.[61]

EHS is usually associated with prolonged exertions, warm climate, body T_c above 40.5°C (104.9°F), and dry skin. Performance of strenuous physical exercise in the heat has been notorious as a cause for EHS. However, in many instances, EHS occurs within the first 2 hours of exercise and not necessarily at high ambient temperatures.[60] Use of a T_c of 40.5°C (104.9°F) as a critical temperature to define heatstroke is arbitrary. In many instances, lower temperatures are recorded because the first measurements are delayed, executed by untrained individuals, or measured incorrectly.

At the stage of collapse, profuse sweating is still likely to be present unless heatstroke develops in an already anhidrotic individual. Dry skin might be evident either in situations where the climate is very dry and sweat evaporates easily, or when heatstroke coincides with a severe degree of dehydration.[59]

Acute Phase

The acute phase is characterized by central nervous system (CNS) disturbances. Because brain function is very sensitive to hyperthermia, this phase is present in all heatstroke patients. Signs of CNS depression often appear almost simultaneously in the form of irritability, aggressiveness, stupor, delirium, and coma.[4,29,164] Seizures occur in 60% to 70% of heatstroke cases. After a return to normothermia, persistence of coma is a poor prognostic sign.[99,164] Other symptoms include fecal incontinence, flaccidity, and hemiplegia. Cerebellar symptoms, including ataxia and dysarthria, are prominent and may persist.[123,164,182] In over 60% of heatstroke cases, pupils were constricted to pinpoint size.[99,183] Papilledema was found in cases of cerebral edema. However, cerebrospinal fluid and pressure were usually within normal values.[2,4,99,164]

Other common disturbances during the acute phase occur in the gastrointestinal and respiratory systems. Gastrointestinal dysfunction, including diarrhea and vomiting, often occurs. The latter, however, may reflect translocation of toxic gram-negative bacterial lipopolysaccharide (LPS) from the lumen of the intestines because of poor splanchnic perfusion secondary to hypotension resulting from increased skin blood flow (BF_{sk}), as well as from CNS impairment.[27,72,164] Hyperventilation and elevation of T_c primarily lead to respiratory alkalosis, which in EHS may be masked by metabolic acidosis as a result of increased glycolysis and hyperlacticacidemia.[31,133] Hypoxemia may be present in patients with respiratory complications.[36,133,168]

Hematologic and Enzymatic Phase

Hematologic and enzymatic disorders peak 24 to 48 hours after collapse. In the hematologic and enzymatic phase of heatstroke, hematologic, enzymatic, and other blood parameters are altered. In humans and experimental animals, hyperthermia results in temporary leukocytosis[79] and changes in lymphocyte subpopulations, both in absolute numbers and in percentages.[1] Leukocytes may range from 20 to $30\times10^3/mm^3$ and higher.[15,86] In one study, all fatal cases of exertional heatstroke had disturbances in the blood coagulation system.[15,162] Prothrombin time, partial thromboplastin time, and the level of fibrin split products increased with a fall in thrombocytes.[61] Clotting dysfunc-

tion peaked 18 to 36 hours after the acute phase of heatstroke, and 2 to 3 days after heatstroke, prothrombin levels fell to 17% to 45% of normal. Depending on the severity of heatstroke, thrombocyte values range between $110\times10^3/mm^3$ and zero.[162,163] These clotting disturbances resemble those of gram-negative bacterial sepsis, and it appears that LPS participates in the pathophysiology of heatstroke.[74]

Significant lay interest, scientific research, and commercial product development and marketing have focused on electrolyte abnormalities associated with physical exertion under various conditions. The marked variability in electrolyte content of sweat and intensity of physical activity, along with ambient temperature (T_{amb}), humidity, and preexisting state of hydration in the individual, all contribute to the degree of electrolyte abnormalities observed in plasma and sweat.

Plasma Volume Changes

Physical exertion alters plasma volume (PV), depending on environmental conditions, intensity and duration of exercise, preexisting and coexisting states of hydration, and level of heat acclimatization. In a cool environment, PV is maintained or even increased with exercise. However, in the heat, a few minutes' burst of supramaximal exercise causes biphasic reduction in PV.[17] During severe exercise, there is rapid efflux of K^+ and metabolites from contracting muscles, causing a local rise in osmotic pressure in the interstitial fluid. As a result, obeying Starling's law, fluid is initially drawn across capillary walls from the circulation into the extravascular space. This increases the transcapillary osmotic gradient, leading to the observed secondary reduction in PV. The combined processes result in an overall sustained reduction of PV by 10% to 15% within 30 seconds. The volume of sweat loss associated with intense physical activity of 30 minutes or more decreases total body water; if these fluid losses are not replaced, a further reduction in PV results.

Potassium

The literature is contradictory regarding the effect of heat stress on plasma potassium concentration. Generally, most patients with classic heatstroke appear to have normal serum K^+ levels at admission, whereas EHS patients often show hyperkalemia and rhabdomyolysis, although a range from hypokalemia to hyperkalemia is seen.[162]

Mechanisms. Early studies suggested that hypokalemia predisposes to heatstroke,[63,92] and that the subsequent resultant cellular damage with its elevated K^+ efflux would raise plasma values back to near normal or above.

Only about 2% of total body K^+ is extracellular, and the intracellular environment acts like a large K^+ reservoir. K^+ passively diffuses out of the cells down its concentration gradient through K^+ "leak" channels in the plasma membrane. It requires a significant amount of ATP to pump it back into the cell. Agents that act on the K^+ transport system can cause rapid rises or falls in plasma K^+ concentrations. Elevated extracellular K^+ concentrations depolarize excitable cell membranes. During hyperthermia in several animal models, ranging from crustaceans to miniswine, serum K^+ rose substantially.[12,25,64,102] In some, the higher the K^+, the shorter the survival time. If the animals had been heat acclimated, the rise in K^+ was blunted, even with prolonged exercise, when elevated serum K^+ is usually seen.[155] In some human subjects, serum K^+ levels rose during heat and exercise.[65,140] This would be expected to cause vasodilation of terminal blood vessels within active skeletal muscles. Several days' training lowered this increase in extracellular K^+ for a given workload, and it improved cardiovascular stability and the ability to dissipate heat. An increase in serum K^+ from exercise may result in part from the large number of depolarizations of muscle membranes. This elevated K^+ concentration was reduced by insulin injection, but performance was not improved.[120]

In the early stage of heatstroke, victims admitted to a hospital may present with hypokalemia, normokalemia, or hyperkalemia. Rhabdomyolysis, cellular damage, and decreased renal perfusion may in part account for hyperkalemia.[1]

Loss of K^+ in sweat usually cannot account for hypokalemia in EHS because the normal K^+ concentration of 4 to 5 mmol/L is not significantly affected by prolonged sweating, heat acclimation, or dietary intake of potassium.[43] However, earlier studies suggested that repeated bouts of intense exercise in the heat lead to K^+ deficiency resulting from sweat loss.[107] Some reports under similar conditions, however, demonstrate normal K^+ levels in plasma and muscles, associated with reduced urinary K^+ excretion. Development of hyperkalemia in heatstroke victims appears to be a consequence of increased K^+ permeability, resulting from failure of the Na^+,K^+-ATPase pump at the cell membrane level, as described in Chapter 10.

One group concluded that hyperthermia in humans leads first to hypokalemia, probably because of respiratory alkalosis, increased renal secretion, and loss in sweat.[156] This would be followed by metabolic acidosis and hyperkalemia when a potential combination of impaired renal function and general cellular damage develops. Most heatstroke patients admitted during the Hadj (the pilgrimage to Mecca, Saudi Arabia) exhibited normokalemia or hypokalemia, with K^+ concentrations of 2 mEq/L or less.[103] Furthermore, once the characteristic hypovolemia had been corrected, serum K^+ fell still further. During exercise in the heat, humans can lose as much as 0.225 mEq K^+ per 100 g body mass (70% into urine and 30% into saliva).[130] Those differences are not simply a matter of experimental error. Gaffin and colleagues[68] explained these discrepancies in a miniswine model, described in Chapter 10.

Sodium

Failure to maintain normal salt intake can suppress triggering of the thirst mechanism, thereby reducing voluntary rehydration. Model experiments indicate that unlike K^+, intracellular Na^+ content does not change significantly in response to heat treatment until a few minutes before death, at which point there is a small rise.[178,184] However, in some clinical cases, serum Na^+ levels were low, reflecting a state of hyperhydration or rhabdomyolysis.[69,77]

The concentration of sodium chloride in sweat may range from 10 to 60 mEq/L, depending on physical activity, heat exposure, state of heat acclimatization, and amount of salt ingested.[148] Whereas the Na^+ concentration in sweat does not vary with sex, children show lower concentrations than do adults.[12,51] Values rarely reach 40 mEq/L in prepubertal boys. The average American diet contains about 170 mEq of Na^+ per day, an amount usually adequate to replace all Na^+ lost through sweating by the trained athlete. However, an athlete with an inadequate caloric intake may suffer from dietary salt deficiencies, and soldiers in combat usually have insufficient food

TABLE 11-2. Determination of Wet Bulb Globe Temperature (WBGT) Heat Index

TEMPERATURE (T, in °F)	FACTOR	EXAMPLE
Wet bulb T	×0.7	78 × 0.7 = 54.6
Dry bulb T	×0.1	80 × 0.1 = 8.0
Black globe T	×0.2	100 × 0.2 = 20.0
HEAT INDEX		82.6

Wet bulb reflects humidity. Dry bulb reflects ambient air temperature. Black globe reflects radiant heat load. Heat index is the sum of the three.
Alternative equation: WBGT = (0.567 T_{db}) + (0.393 P_a) + 3.94, where T_{db} = dry bulb temperature, P_a = water vapor pressure.

intake. In such cases, supplementation may be required after moderate sweat losses.

Effect of Physical Activity on Sodium Sweat Concentration. A sudden increase in exercise intensity rapidly increases the electrolyte concentrations of sweat and more slowly increases electrolytes in serum.[21,62,114] The electrolyte concentration of sweat is highest immediately after commencing physical activity and gradually decreases with prolonged exercise, possibly as a passive result of falling salt content in the body.

Thermal stress alone raises Na^+ and Cl^- concentrations less than changes induced by running.[66] In a variety of sports (yachting, rowing, handball, and cycling), initial sweat electrolyte concentrations were dependent on type of sport, duration of performance, and level of training in elite athletes.[21] Cyclists and rowers, whose sports produce high-endurance capability, had a significantly lower content of sweat Na^+ content than did yachtsmen. In addition, participation in an intensive training program led to a further reduction in sweat electrolyte concentration.

The sweat of a trained athlete may show a Na^+ content as low as 17 mmol/L (34 mOsm/kg), a level one-third that of a person who is untrained and unacclimated to the heat. On the other hand, the untrained, unacclimatized "weekend warrior" or vacationer is at greatest risk for significant Na^+ losses when exercising in the heat (Table 11-2).

Effect of Heat Exposure on Renal Absorption of Sodium. After repeated days of exercise in the heat, there is increased production of aldosterone with consequent enhanced renal reabsorption of Na^+; thus, Na^+ is progressively conserved. After prolonged strenuous exercise and sweating, urinary excretion of Na^+ may temporarily almost cease.[42]

Sodium Deficiency. In temperate thermal environments, Na^+ deficit due to exercise is unlikely to be so severe as to require Na^+ supplementation of the normal diet. Similarly, a brief (<2 hours) episode of moderate exercise in the heat rarely causes clinically significant hyponatremia upon voluntary rehydration. However, when sweat loss exceeds 5 L/day (1.1 gal/day), hyponatremia may develop upon rehydration and occur during the initial stage of acclimation to heat, during prolonged and repeated exercise in the heat (training camps), or during ultra-endurance events, such as marathons, 50-mile runs (80.5 km), or triathlons.[132]

In one report, four athletes developed hyponatremia and water intoxication during endurance events lasting more than 7 hours.[135] The cause was thought to be voluntary hyperhydration with hypotonic solutions, combined with moderate losses of Na^+ and Cl^- in sweat. These athletes were slower runners who competed at a lower intensity than the top finishers.

An interesting case of symptomatic hyponatremia resulting from excessive voluntary intake of water showed paradoxical secretion of antidiuretic hormone (ADH), leading to renal shutdown instead of increased output of urine.[5] Ordinarily, ADH is secreted in response to dehydration but not to hyponatremia. However, volume overload of the stomach is known to cause nausea, which is a strong stimulus for production of ADH.

Calcium

In isolated cultured cells, heating may lead to increased permeability of the plasma membrane[178] and an increase to approximately 0.1 µM in cytoplasmic Ca^{2+} concentration.[63] It is unclear if the rise in Ca^{2+} contributes to the pathophysiology of heatstroke, because elevating the total cell content of Ca^{2+} sixfold had no effect on survivability at 45°C (113°F).[178]

Several studies suggest that hyperthermia leads to a reduction in serum Ca^{2+} as a result of deposition of calcium phosphates and calcium carbonates in injured skeletal muscles.[156] Other studies, however, indicate that high temperatures may cause acute renal failure or rhabdomyolysis, which in turn releases Ca^{2+} into serum.[106] Moreover, in microswine with heatstroke, Araki and colleagues reported a biphasic reduction in Ca^{2+} concentration.[12] At approximately 41°C (105.8°F), Ca^{2+} concentration declined, reaching a minimum at 44°C (111.2°F), and then rapidly rose to near baseline levels. This may account for both the rises and the falls in serum Ca^{2+} reported for heatstroke victims. Fluorescence imaging[111] and tracer techniques[178] show that heating cultured cells raises cytoplasmic Ca^{2+} concentration.

Magnesium

Concentrations of magnesium in sweat vary from 0.02 to 5 mmol/L and might be expected to cause hypomagnesemia during prolonged exercise in the heat.[38,40,44,175] Significant loss of total body magnesium is prevented, however, because there is a concomitant rapid decrease in renal magnesium excretion.[43,175] Losses during endurance exercise have been estimated to be about 1% of total body magnesium content.[44] Occasionally, an acute fall in serum magnesium occurs after prolonged intense exercise,[18,149] which may be caused by temporary uptake of Mg^{2+} by erythrocytes,[146] mononuclear cells,[158] and actively contracting muscle,[41] in addition to losses in sweat.[42] Although some studies suggest that these losses are severe enough to cause symptoms, the consensus is that development of clinical hypomagnesemia secondary to losses in sweat is unlikely.[119]

Phosphate

Hypophosphatemia is frequently seen in EHS.[23] Heat stress often causes respiratory alkalosis, which increases the rate of cellular uptake of phosphate and reduces its excretion.[131] Other studies suggested that hypophosphatemia was caused by increased renal clearance of phosphorus with decreased renal threshold, plus contributions from metabolic acidosis and increased uptake of phosphorus by cells.[156] However, hypophosphatemia is not always reported in humans or animal models,

and respiratory alkalosis does not always occur at the same time as does hypophosphatemia.

Enzymes

One of the prominent and almost pathognomonic characteristics of EHS is the appearance of exceptionally high levels of certain cellular enzymes, implying cell damage or death. Most EHS patients show elevation of serum creatine phosphokinase (CPK) activity and myoglobinuria, suggesting damage of skeletal muscles.[60] CPK activity in the range of 10^3 to 10^4 IU was commonly found, with peak values 24 to 48 hours after collapse.[48,157] At a T_c of 41.8° to 42.2° C (107.24° to 107.96° F), aspartate aminotransferase (AST) and alanine aminotransferase (ALT) concentrations rose by factors of 25 and 8, respectively, and bilirubin concentration approximately doubled to 1.56 mg/dL.[140]

These rises in enzyme levels are related to tissue damage, which in turn depends on both T_c and its rate of rise and its duration.[129] CPK, an indicator of skeletal and cardiac muscle damage, was the most sensitive to increases in T_c, followed by lactate dehydrogenase, an index of generalized tissue damage. In microswine with heatstroke, however, Gaffin and colleagues found rises in CPK and lactate dehydrogenase (LDH) only about 20 minutes before death, and a rise in AST about an hour before death, but no change in ALT concentration.[68]

During the 1994 Hadj, 26 heatstroke victims admitted to a heatstroke treatment unit had elevated levels of CPK, AST, ALT, and LDH, and these remained high after 24 hours. Those who died had higher enzyme levels than the survivors. LDH concentration was useful in distinguishing between those who died and those who had a rapid recovery. The enzymes were better prognostic indicators than T_c, anion gap, and serum K^+.[7]

Glucose

Catecholamines and cortisol are secreted during heatstroke, they stimulate glycogenolysis, and they raise plasma glucose concentrations.[3] Thus, patients with classic heatstroke are usually hyperglycemic.[23,76] Approximately half of miniswine with heatstroke studied also developed hyperglycemia, but 20 to 30 minutes after a rise in circulating insulin, glucose concentrations sharply declined. In accordance with these observations, patients suffering from heat exhaustion were normoglycemic, but some of those with heatstroke were hypoglycemic.[46]

Late Phase

The late phase of heatstroke manifests 3 to 5 days after collapse and is characterized by disturbances in renal and hepatic functions. High bilirubin levels, which may last for several days, reflect both hepatic dysfunction and hemolysis.

Acute renal failure is a common complication of severe cases and occurs in approximately 25% to 30% of EHS patients.[108,122,162] Oliguria and anuria are characteristic features. At this phase, urine has been described as being like machine oil, with a low specific gravity.[139,144,154,162] Usually present in the urine are red and white cells, hyaline and granular casts, and mild to moderate proteinuria.[154] The etiology involves multiple causes,[139] but a major one is reduced renal blood flow subsequent to heat-induced hypotension, hypohydration, and peripheral vasodilation. Furthermore, direct thermal injury may also lead to widespread damage of renal tissue.[154] Myoglobinuria and elevated blood viscosity resulting from disseminated intravascular coagulation (DIC) may further contribute to acute oliguric renal failure.[144,154,167,176]

EHS is usually manifested by increased serum levels of liver enzymes, but acute liver failure has also been reported. Liver injury in most cases of EHS is usually asymptomatic and exhibits reversible elevation in plasma aminotransferase levels.[83] Acute liver failure is documented in 5% of EHS patients.[110] Despite limited experience and the observation that even those EHS patients with extensive liver damage may recover spontaneously, orthotopic liver transplantation had been suggested as a potential treatment.[19,82,151] Among 16 reported cases of EHS-induced liver failure,[78] 3 patients underwent liver transplantation. In the conservatively managed group, eight patients recovered spontaneously and five died. Concomitantly, all three patients who received transplantation died. Hadad and coworkers[78] concluded that because of the poor and limited results of liver transplantation after heatstroke, interpretation of the prognostic criteria is crucial before listing a patient for such surgery.

Recovery

CNS dysfunction becomes increasingly severe with prolonged duration of hyperthermia and its associated circulatory failure. Nevertheless, coma persisting for 24 hours, even with subsequent seizures, is usually followed by complete recovery without evidence of mental or neurologic impairment.[4,150] However, chronic disability may prevail for several weeks or months in the form of cerebellar deficits, hemiparesis, aphasia, and mental deficiency.[4,108,150] Only in exceptional cases, when coma persisted for more than 24 hours, did mental and neurologic impairment become chronic and prevail for some years. However, in one study of classic heatstroke, 78% of patients had minimal to severe neurologic impairment.[48] Shapiro and Moran[157] studied 82 cases of EHS in Israeli soldiers and concluded that associated with each case was at least one factor predisposing to heatstroke (e.g., diarrhea, lack of acclimatization, poor fitness). Correcting those individual risk factors should lead to strategies capable of preventing heatstroke. Indeed, in more than 90% of the victims in whom predisposing background factors were identified and eliminated, and after waiting 6 to 8 weeks to ensure full recovery, the patients were permitted to return to their routine training schedule.

Heat Tolerance Test

In the Israeli Defense Forces, all heatstroke victims must undergo an exercise–heat tolerance test after recovery, to rule out any effect of concurrent disease and abnormality in thermoregulation.[58] The patients (usually highly motivated soldiers from a combat unit) are scheduled for the test 6 to 8 weeks after the collapse. Before taking the test, they are not permitted any activity related to heat stress. During the test, the individuals wear only shorts and sport shoes. After 10 minutes of rest, they walk on a treadmill at 1.34 m/sec (4.39 ft/sec) at a 2% grade in a hot and dry environment (40° C [104° F], 40% relative humidity [RH]) for 120 minutes.[6] Analysis of multiple physiologic variables during the test helps to diagnose any congenital or acquired factors that might compromise body temperature regulation.[58,127] The measurements obtained determine whether the individual should continue to serve in a combat unit or be removed from it.

CLINICAL MANAGEMENT OF HEAT-RELATED ILLNESS

Classic heatstroke occurs when environmental heat load is very high.[53,81,170] Usually it develops over a few days (e.g., during a heat wave) and affects mainly older adults, especially those with underlying infection, liver disease, or limited access to air conditioning. In classic heatstroke, physical effort is not the primary cause and most cases are accompanied by dehydration. However, the pathologies of different heat illnesses (e.g., classic or EHS, heat exhaustion) share many similarities, regardless of the cause (see Chapter 10).

As previously discussed, individuals exercising in the heat develop a spectrum of clinical illnesses ranging from syncope to heatstroke. Further discussion of the clinical management of heat illness focuses on heat exhaustion and heatstroke. The distinction between these two clinical diagnoses is somewhat artificial and may be difficult to establish in the field or at the medical facility.

Exertional Heat Illness

A universal feature of heatstroke is a state of collapse that puts an abrupt end to the physical effort. As activity ceases and body temperature falls, victims may spontaneously regain consciousness temporarily, and they may recover. However, if high T_c persists long enough, heat-induced tissue injuries may already be lethal unless the patient is rapidly cooled.

Although strenuous physical exercise (e.g., athletic events, military training, hard labor) in the heat has been notorious as a cause of heatstroke, the disorder has also occurred at relatively low T_{amb}.[10] In principle, diagnosing exertional heat illness (EHI) is clear: if a previously healthy individual collapses while exercising, usually in a warm environment for more than 1 hour, with a rectal temperature (T_{re}) of greater than 40.0°C (104.0°F), the diagnosis of heatstroke is virtually certain.[36,73,95,117,166] Although EHI might be diagnosed immediately, EHI and its severity can also be diagnosed retrospectively from clinical laboratory data after proper clinical management.[20,50,80,138] Occasionally, a T_{re} of less than 40°C (104°F) has been seen in individuals diagnosed as having heatstroke, which is later confirmed by blood chemistry. A T_{re} measurement should be taken immediately after the collapse to obtain an immediate and definite diagnosis.

The presentation of heatstroke is usually acute, with approximately 25% of all casualties exhibiting prodromal symptoms lasting minutes to hours. These include dizziness, weakness, nausea, confusion, disorientation, drowsiness, and irrational behavior. Failure to recognize the first signs of disability (or in some cases even an assumption that the subject is malingering) has led to incorrect assessment of true physiologic status. Because of delay in transportation, a heatstroke victim is likely to arrive at the emergency department partially rehydrated and with a T_c that is lower than it was at the site of collapse, and thus only mild CNS disturbances are evident. Therefore, a correct diagnosis of heatstroke depends on understanding the entire clinical and pathologic picture (see Chapter 10).

At the time of heatstroke collapse, sweat glands are usually still active and profuse sweating is usually present, in contrast to earlier beliefs about heatstroke.[46,95,117,166] The appearance of dry skin occurs mainly in a very dry climate where sweat evaporates very rapidly, or when dehydration has become so severe that secretion of sweat is depressed[93]; it is more often seen in classic heatstroke than in EHS.

It is essential to determine body temperature as soon as possible after collapse, bearing in mind that intense hyperthermia occurs immediately after exercise. Heatstroke has been misdiagnosed when certain conventional criteria, such as a warm climate, very high body temperature, and lack of sweating, are depended on. The widely held criterion that T_{re} must be high is incorrect. At the moment of collapse, body temperature probably exceeds a certain critical temperature that usually leads to heatstroke. However, delaying the first measurement of body temperature may yield misleading lower temperatures. In one instance, during transportation between the site of collapse and the hospital emergency room, body temperature dropped from 41.1° ± 0.8°C to 37.8° ± 1.2°C (106° to 100°F), a fall of 3.3°C (6°F).[172] Furthermore, temperatures may be taken by untrained individuals or measured incorrectly.[94,108,172]

Heat Exhaustion

Heat exhaustion occurs in individuals who sweat profusely while participating in an endurance event or while engaging in repeated bouts of exercise. This results in significant and unreplaced sweat losses. Prolonged insensible sweat losses, such as occur while sitting in the sun in a moderate breeze, predispose to heat exhaustion if fluid and electrolyte losses are significant.

Problems with Recognition

Early symptoms of heat illnesses are often unrecognized or mistaken for malingering. Failure to attend to these symptoms puts the individual at risk of significant thermal injury. Headache, confusion, drowsiness, and even euphoria may be important warning signals. Individuals who anticipate exposure to hot environmental conditions should be thoroughly versed in the manifestations of heat illness.

The victim of heat exhaustion may arrive at the medical facility with other symptoms that can be misread. Nausea and visual disturbances mimic the prodrome of a migraine headache. Dizziness or syncope may suggest arrhythmia or seizure. Symptoms of chilling, piloerection, sweating, and moderately elevated temperature may suggest an infectious process. Development of altered mental status or ataxia raises the possibility of meningitis, encephalitis, or drug intoxication.

Treatment

In treatment of heatstroke, cooling should be initiated vigorously and immediately on collapse, and any delay of vital resuscitation measures should be minimal. The most practical and efficient method of cooling is drenching the victim with large quantities of crushed ice and water, with vigorous massage. If ice is unavailable, then use tap water alone. Treatment is based on symptoms and focuses on both rehydration and reduction in temperature. The victim must immediately cease exercising or carrying out any other physical activity. Although this appears to be intuitively obvious, some persons develop euphoria and may resist this measure. Furthermore, later return to physical activity on the same day is contraindicated. When possible, the victim should be removed from direct sunlight (which could have a heat load of hundreds of kilocalories per hour) and restrictive clothing should be loosened. A victim who is unconscious or whose T_{re} is greater than 39°C (102.2°F) should be treated as an incipient heatstroke victim and immediately evac-

Box 11-1. Current Cooling Method for Exertional Heatstroke

At the U.S. Marine Corps training base at Parris Island, South Carolina, victims of exertional heatstroke are routinely cooled using the following procedures. Note: These victims are young men and women who are otherwise healthy. Victims of classic heatstroke are usually older adults with concurrent diseases, and those factors must be taken into consideration.

1. During physical training, the clinic routinely maintains a dedicated room with two bathtubs full of cold water and ice.
2. In the field, the blouse and pants are removed from a collapsed patient with suspected heatstroke, leaving on the shorts and T-shirt. Rectal temperature is measured, ice is packed around the groin and axillary areas, and the patient is immediately transported to the clinic on a stretcher. On arrival, the stretcher is placed on top of the iced bathtub, above the water and ice, with the carrying handles sticking out at both ends.
3. Mental status and other vital signs are assessed and blood is drawn for laboratory analyses.
4. One liter of normal saline is administered as a bolus.
5. Sheets are dipped into the tub's icy water and are used to cover and drench the patient. Copious ice is added to the top of the sheet to cool still further, and the skin is massaged to improve skin blood flow. The sheets are frequently rewetted with the icy water.
6. Concurrently, the head is constantly irrigated with more ice water and a fan is directed at the patient.

Routinely, the victim is not immediately immersed in the ice water in case cardiopulmonary resuscitation becomes necessary. However, if rectal temperature is not lowered sufficiently, then the victim is immersed directly into the ice and water. With this procedure, rectal temperature usually falls to 39.5° C (102.5° F) within 15 to 20 minutes, and the patient is removed from the stretcher, rinsed, and placed on a gurney, and IV fluids and laboratory studies are reviewed.

From Gaffin SL, Gardner J, Flinn S: Ann Intern Med 132:678, 2000.

uated to a medical facility, where the guidelines in the next paragraphs should be followed.

Body temperature may be lowered by a variety of means. Immersion in ice water (when it is available), especially when combined with massage through ice water–drenched sheets, facilitates the most rapid drop in body temperature.[46,67] In the field, alternative measures include moving the victim to a shaded area, pouring large amounts (20 to 30 L [4.4 to 6.6 gal]) of water all over the victim's body, especially the face and torso, and fanning, or placing ice bags over the superficial great vessels (axillae and groin).[37] Periodic toweling of the skin renews the evaporative surface and facilitates evaporative cooling (Box 11-1). Cooling should be continued until T_c reaches 38.5° C (101.3° F).[142]

The RTX rapid thermal exchange device (AVAcore Technologies, Inc., Ann Arbor, MI; www.avacore.com) (Fig. 11-1) amplifies blood flow through the human hand by utilizing a slight vacuum, which allows it to then extract heat. Designed for athletes to enhance cooling and improve performance, it has potential applications for firefighters (Fig. 11-2) and other persons who are detrimentally affected by high body temperature.

Figure 11-1. The RTX rapid thermal exchange device. The device subjects the hand to a slight vacuum, which dilates small arteriovenous anastomoses and maintains high local circulation. The device then extracts the heat produced. (Courtesy of AVAcore Technologies, Inc., Ann Arbor, MI.)

If the patient is alert, oral rehydration can be instituted with cold water or a fluid-replacement beverage. The concentration of carbohydrates in such a beverage should not exceed 6%; otherwise, gastric emptying and fluid absorption by the intestines may be delayed. Initial target intake of 1 to 2 L (0.9 to 1.8 qt) over 2 to 4 hours is reasonable; a greater rate results in limited absorption with inappropriate diuresis. The victim should continue to rest and drink over the next 24 hours. As a general rule, for every pound of weight lost by sweating, 0.5 qt (2 cups or 500 mL) of fluid should be consumed. It may require 36 hours to completely restore lost electrolytes and fluid volume to all body compartments via oral intake. After the acute episode, the medical care provider should determine any possible host risk factors for heat illness and review with the victim signs of heat illness and preventive measures.

Heatstroke

Heatstroke is a true medical emergency. Before 1950, the mortality rate was 40% to 75%.[11,57] Long-term survival is directly related to rapidly instituting resuscitative measures.[85]

Problems with Recognition

Earlier diagnosis of heatstroke requires recognition of three signs: severe hyperthermia (T_c > 41° C [105.8° F]), CNS disturbance, and cessation of sweating.[34] This symptom complex represents the extreme or full-blown heatstroke presentation and is now recognized to be too rigid and to possibly promote delays in critical interventional measures. These criteria will be considered in turn.

The body temperatures reported in the field for heatstroke victims may be significantly higher (41.1° C [106.9° F], as opposed to 37.8° C [100° F]) than those documented in the hospital emergency department, because T_c may fall during trans-

Figure 11-2. Firefighter utilizing the RTX rapid thermal exchange device.

port to the hospital.[161] Failure to obtain or record an on-site rectal temperature may hinder prompt diagnosis; similarly, documentation of only mild elevation in body temperature should not exclude the diagnosis of heatstroke.

The victim's altered mental status may adversely affect the ability of emergency department personnel to obtain a detailed history of precipitating events. Lack of such information may also delay diagnosis. Emergency medical transport personnel should attempt to obtain this history before evacuating the victim and should communicate the information to medical staff.

Central Nervous System

The brain is particularly susceptible to thermal injury. CNS dysfunction is directly related to duration of hyperthermia and to circulatory failure. The underlying pathology observed on autopsy is brain edema and congestion, with petechial hemorrhages and neuronal degeneration.[122] Cerebrospinal fluid pressure and hematologic features are normal.[164] In Shibolet's study,[161] all patients presented with confusion or agitation or both, and 72% had convulsions, but all eventually became comatose. Agitation, delirium, and hallucinations reflect CNS hyperirritability. Pupillary constriction was present in 66% of cases.[100] Seizures occur in approximately 50% of cases.[100] Decerebrate rigidity, oculogyric crisis, and opisthotonos may develop. Other possible findings are loss of rectal sphincter tone, loss of skeletal muscle tone, and hemiplegia.[164] Cerebellar findings of dysarthria and ataxia are common.

Complete recovery occurs in most cases. However, chronic disability may develop in the form of mental deficiency, dysarthria, ataxia, aphasia, and hemiparesis.

Two important points: (1) Some investigations have shown that cessation of sweating is a late phenomenon of heatstroke.[147,162] *At the time of collapse, most heatstroke victims continue to sweat profusely.* Failure to consider the diagnosis of heatstroke in a diaphoretic patient could be fatal. (2) Unless an alternative cause is obvious, the previously healthy individual who collapses after physical exertion in hot weather should be considered to have EHS.[33,164]

Diagnosis

Shibolet[161] noted that a delay in measurement of T_c or inaccurate methodology in obtaining initial temperature values often led to inappropriately low initial temperature determinations when compared with actual T_c at the time of collapse. Loss of consciousness is an unvarying feature of heatstroke. With cessation of physical activity, rate of metabolic heat production markedly decreases, and the T_c falls if skin temperature (T_{sk}) is greater than T_{amb} and sweating persists. Shapiro and Seidman[160] proposed that the diagnosis of heatstroke should be considered in any person who has lost consciousness during exertion and who demonstrates clinical and laboratory signs of heatstroke, even if body temperature was not markedly elevated several hours after collapse.

Epstein and Moran[59] suggested that because early clinical signs of heatstroke are not specific, any systemic disease or condition that occurs with elevation in T_c and manifestations of brain dysfunction should be considered only after the diagnosis of heatstroke is excluded. A history of prodromal symptoms as described for heat exhaustion should markedly increase the suspicion for heatstroke.

CNS disturbances (coma, convulsions, confusion, or agitation) accompanying hyperthermia may also result from CNS infections, sepsis, or other disease processes. Evaluation for these disorders should proceed only after the diagnosis of heatstroke is ruled out. Markedly elevated levels of AST, ALT, and LDH may help in the differential diagnosis.[96] However, the clinician should bear in mind that elevation of these enzymes may not occur for 24 to 48 hours after heat injury.[164]

Complications

Observed complications of heatstroke reflect the results of both direct thermal injury and cardiovascular collapse. Autopsy of persons with EHS showed multiple hemorrhages, congestion, and cellular degeneration in most or all of the organs examined.[162] In particular, rhabdomyolysis is usually present in EHS.[109] A brief overview of the specific pathology found should provide a framework for discussion of the therapeutic and supportive management of heatstroke.

Cardiovascular System. Hypotension does not always occur in heatstroke but is usually present in fatal or severe cases.[162] This hypotension, which may persist even after large volumes of fluid therapy along with vasopressors, is an ominous late feature of heatstroke and may reflect failure of compensatory vasoconstriction of mesenteric blood vessels. As vital organs are hypoperfused, shock develops. Left ventricular subendocardial

hemorrhage and focal necrosis of cardiac muscle fibers are commonly found on autopsy.[122,164]

The extreme stress placed on the cardiovascular system of victims with heatstroke results in a universal finding of sinus tachycardia, often with rates exceeding[115] 140 beats/min.[162] Pulse pressure is elevated in the face of low cardiac output and low diastolic blood pressure.[164] Electrocardiographic findings include ST segment and T wave abnormalities and conduction disturbances.[46] In microswine and monkeys during heating, HR rose to a plateau shortly after heating and commenced to rise still further at T_c of 41°C (105.8°F), peaking a few minutes before death. Blood pressure was stable or even rose until HR peaked at a T_c of 41°C and then declined until death.[68]

Pulmonary System. Hyperventilation is a common finding, particularly in EHI. Persistent hyperventilation may lead to respiratory alkalosis and tetany. Coma or seizures may predispose to pulmonary aspiration with consequent lung injury.[164] Pulmonary edema may become severe or fatal; DIC may lead to adult respiratory distress syndrome.[56] Pulmonary infarctions have been found on autopsy.[122]

Renal System. Acute renal failure develops in approximately 25% of heatstroke victims.[105] Hypotension and the resultant decreased renal blood flow constitute the primary underlying etiology. As in the CNS, thermal injury may cause direct cellular damage.[154] DIC and myoglobinuria exacerbate the insult to renal tissue. Hematuria, pyuria, proteinuria, and hyaline and granular casts are seen on urinalysis. Eventually, oliguria and anuria may develop.

Hepatic System. Within 12 to 24 hours after heatstroke collapse, elevated AST, ALT, and serum bilirubin levels can be detected.[98] Levels of AST greater than 1000 U/mL are commonly seen in severe heatstroke.[177] Prothrombin level reaches its nadir 48 to 72 hours after heat injury.[153] Cholestasis and hepatocellular necrosis may develop.

Gastrointestinal System. Vomiting and diarrhea are common symptoms of heatstroke. Compensatory mesenteric vascular constriction may produce localized areas of gut ischemia and mucosal injury with consequent breakdown of its permeability barrier and translocation of LPS into the circulation.[71] Direct thermal damage to the gut wall may also contribute to gut wall pathophysiology. As previously discussed, the subsequent increase in serum LPS and its role as inducer of potentially damaging cytokines may play a critical part in both morbidity and potential therapy of heatstroke. In the presence of DIC, hematemesis and melena may develop.[164]

Hematologic System. White blood cell counts may be elevated to as high as 20,000 to 30,000/mL. Platelet count is depressed, as are levels of clotting factors V and VIII. The commonly seen bleeding diathesis may be manifested as conjunctival hemorrhage, melena, purpura, or hematuria.[164] The primary causes of this coagulopathy are believed to be release of thromboplastic substances secondary to endothelial damage, and endotoxemia resulting in intravascular thrombosis and secondary fibrinolysis.[161] Other contributing factors include direct inactivation of clotting factors by thermal injury, decreased hepatic production of clotting factors, and decreased production of platelets secondary to thermal injury to marrow megakaryocytes. Low levels of fibrinogen and elevated levels of fibrin split products, in the presence of thrombocytopenia, herald the onset of DIC. Clotting abnormalities most often peak 18 to 36 hours after acute heat injury.[161]

Acid–Base and Electrolyte Abnormalities. Early in the course of heatstroke, respiratory alkalosis secondary to hyperventilation may be present. As anaerobic or even aerobic glycolysis increases, serum lactate levels rise, generating metabolic acidosis.

Hypernatremia reflects associated dehydration common in heatstroke; however, Na^+ levels may appear normal. In the face of dietary deficiency of Na^+ or profuse sweat losses, initial normonatremia may develop into hyponatremia upon rehydration. Potassium levels may be low or elevated; hyperkalemia is associated with tissue damage and renal compromise. Hypocalcemia, hypomagnesemia, and hypophosphatemia may also occur. Although hypoglycemia may develop in cases of EHS, hyperglycemia secondary to elevated catecholamine release may also be seen.

ON-SITE EMERGENCY MEDICAL TREATMENT

Heatstroke is a medical emergency. Rapid reduction of elevated body temperature is the keystone of management of heatstroke; duration of hyperthermia may be the primary determinant of outcome.[105,162] Nevertheless, it is important to follow the ABCs of stabilization while cooling efforts are initiated. See Box 11-2 for basic first aid.

Early diagnosis of heat exhaustion can be vital. Early warning signs include flushed face, hyperventilation, headache, dizziness, nausea, tingling arms, piloerection, chilliness, incoordination, and confusion.[113] Pitfalls in the diagnosis of heat illness include confusion, preventing self-diagnosis.[55] Mainstays of therapy include emergency on-site cooling, intravenous (IV) fluids, treating hypoglycemia as needed, IV diazepam for seizures or severe cramping or shivering, and hospitalization if response to therapy is slow or atypical.[55,112]

Cooling should be energetically initiated immediately upon collapse. To prevent or minimize expected complications, it should be minimally delayed for vital resuscitation measures. In the field, the sick individual should be placed in the shade and any restrictive clothing removed. The victim's skin should be kept wet by applying large quantities (20 to 30 L [5.3 to 7.9 gal]) of tap water or water from any source, and the body should be constantly fanned. However, it is of utmost impor-

Box 11-2. Basic First Aid for Heat Illnesses

1. Place the victim in the shade.
2. Remove restrictive clothing.
3. Apply large amount of water onto the victim and keep the skin wet.
4. Improvise a fan and cool the victim.
5. Measure rectal temperature to confirm diagnosis.
6. Evacuate to the nearest medical facility.

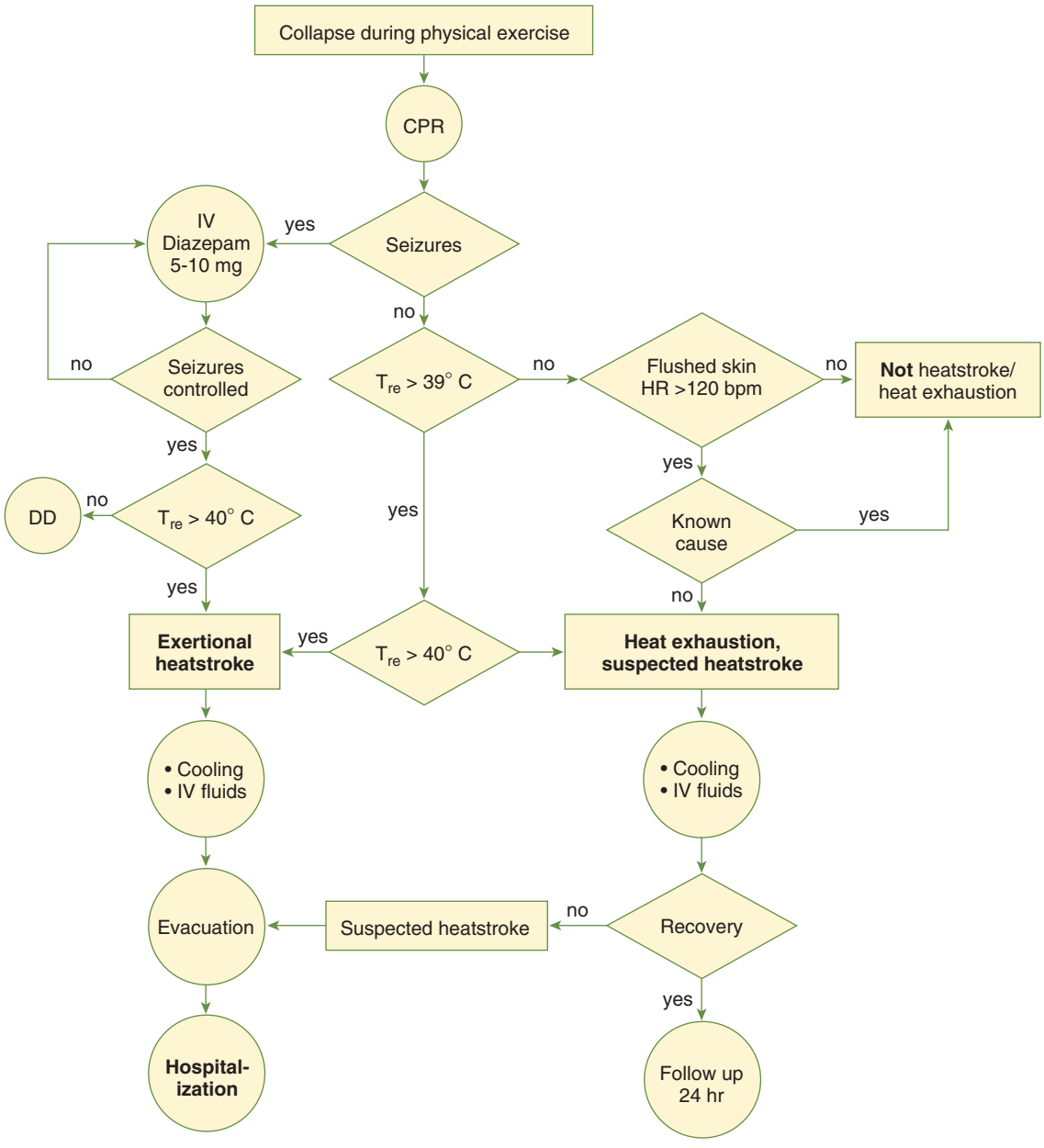


Figure 11-3. Flow chart for on-site emergency medical treatment of exertional heat illnesses. CPR, cardiopulmonary resuscitation; DD, differential diagnosis; HR, heart rate; IV, intravenous; T_{re}, rectal temperature. (Modified from Shapiro Y, Seidman DS: Med Sci Sports Exerc 22:6, 1990.)

tance that these measures only barely delay evacuation of the victim to a hospital or the closest medical facility.

In a comatose victim, airway control should be established by insertion of a cuffed endotracheal tube. When available, administration of supplemental oxygen may help meet increased metabolic demands and treat the hypoxia commonly associated with aspiration, pulmonary hemorrhage, pulmonary infarction, pneumonitis, or pulmonary edema.[56,122] Positive-pressure ventilation is indicated if hypoxia persists despite supplemental oxygen administration (Fig. 11-3).

As discussed previously, resuscitative measures may rapidly lower body temperature. Monitoring and recording T_{re} on site may be important for the correct diagnosis of heatstroke. Vital signs should be monitored, with attention to blood pressure and pulse. Although normotension should not be taken as a reassuring sign, hypotension should be recognized for the ominous sign it always represents. If possible, urine and blood samples should be obtained for electrolyte evaluation, especially Na^+ to avoid hyponatremia, before fluid infusion.

Vascular access should be established without delay by insertion of a large-gauge IV catheter. Administration of normal saline or lactated Ringer's solution should be started. Recommendations regarding the rate of administration of fluids vary. Some authors advise a rate of 1200 mL (1.26 qt) over 4 hours,[136] but we consider this to be too conservative. Others encourage a 2-L (2.11-qt) bolus over the first hour and an additional liter of fluid per hour for the next 3 hours.[160]

Vigorous fluid resuscitation may precipitate development of pulmonary edema, so careful monitoring is indicated. Ideally, 1 to 2 L (1.05 to 2.11 qt) of fluid should be administered during the first hour after collapse and additional fluids administered according to the level of hydration.[61]

Cooling measures should be initiated immediately. However, cooling techniques are ineffective when the victim suffers seizures that increase storage of body heat. Therefore, convulsions should be controlled by IV administration of 5 to 10 mg of diazepam, as necessary.

As a result of drastic cooling, T_{sk} may decrease enough to cause shivering. IV administration of chlorpromazine (50 mg)[97] or diazepam (5 mg) is effective to suppress shivering and prevent an additional rise in body temperature from metabolic heat production.

Heat-Induced Syncope

In an upright and stationary person, blood volume is displaced into the dependent limbs by gravity. If that person is also heat stressed, more blood is displaced into the peripheral circulation to support heat transfer at the body surface. These combined reductions in effective blood volume can temporarily compromise venous return, cardiac output, and cerebral perfusion. Fainting is usually brief and responds to horizontal positioning and improved venous return. The patient should be allowed to rest in cooler, shadier surroundings (as the solar heat load can be >200 kcal/hr) and should be offered cool water. The patient should be cautioned against protracted standing in hot environments, advised to flex leg muscles repeatedly while standing to enhance venous return, and warned to assume a sitting or horizontal position at the onset of warning signs or symptoms, such as vertigo, nausea, or weakness. Normally, muscles in the legs act as a "second heart" and, in concert with venous valves, promote venous return, thereby counteracting orthostatic pooling and the predisposition to syncope. Consistent with this, nonfainters have higher intramuscular pressure than do fainters.

In stark contrast to simple syncope is the profound CNS dysfunction dominating the early course of heatstroke. Thus, if a person faints in a setting where hyperthermia is possible and does *not* rapidly return to consciousness, heatstroke should be suspected, cooling measures instituted, and body temperature monitored.

Cooling Methods

Much debate exists in the literature regarding the best approach to cooling heatstroke victims.[37,45,75,165,180,181] Morbidity and mortality are directly related to duration and intensity of elevated T_c. Therefore, the rate at which any given method lowers body temperature is extremely important. Another consideration in choosing a cooling modality is the need for access to the victim for continuous monitoring.

Proulx and colleagues[142] used cooling water immersion at 2°C (35.6°F) to achieve a cooling body rate of 0.35°C/min. At water temperatures of 8, 14, and 20°C (46.4°, 57.2°, and 68°F), cooling rate was 0.19°, 0.15°, and 0.19°C/min, respectively. Shivering was seldom observed during the 20°C water immersion. In contrast, Clements and coworkers[35] found no significant differences between the cooling rates of water immersion at 5.2°C and at 14°C (41.4° and 57.2°F). According to these two studies and others,[78] a clear recommendation regarding optimal water temperature for cooling cannot be stated.

Khogali and coworkers[180] developed a body cooling unit designed to maximize evaporative cooling by maintaining cutaneous vasodilation and minimizing shivering. The patient is suspended on a net and sprayed from all sides with water at 15°C (59°F). Warm (45° to 48°C [113° to 118.4°F]) air is blown over the victim. Cooling rates of 0.06°C/min (0.11°F/min) have been obtained. Although this method is widely recommended as the treatment of choice, the rate of cooling is actually much less than that accomplished by ice-water immersion.

Although not always available, ice-water or cold-water immersion is an effective and easily available method of rapidly lowering core body temperature. However, its use is one of the more hotly debated topics in the heatstroke literature. In most cases, increased thermal conductivity of water results in reduction of T_c to less than 39°C (102.2°F) in 10 to 40 minutes.[46] This reflects a mean rate of cooling of 0.13°C/min (0.23°F/min)—that is, twice the rate of the body cooling unit. Use of cold water rather than ice water resulted in the same rate of cooling—0.13°C/min.[136] Cold-water immersion is less uncomfortable for the victim than is immersion in ice water. In several hundred EHS victims in a military population, there were no fatalities or permanent sequelae after treatment with ice-water immersion and massage.[46,136] Although other cooling methods reduce the rate of mortality, none has been as successful as ice water–soaked sheets or immersion.[67]

Poulton and Walker[141] treated heatstroke patients by using a light helicopter as a large powerful fan to provide surface cooling and enhance evaporation of water sprayed over the patients. These authors found the method to be an efficient cooling concept. However, the helicopter rotary blade downdraft carries potential risks for the patient and medical staff attendants.

In discussing an alternative cooling method, Khogali[101,180] summarizes the most commonly offered criticisms of ice-water immersion:

- Exposure to severely cold temperatures may cause peripheral vasoconstriction with shunting of blood away from the skin, resulting in a paradoxical rise in core temperature.
- Induction of shivering (in response to the cold) may cause additional elevation in temperature.
- Exposure to ice water causes marked patient discomfort.
- Working in ice water is uncomfortable for medical attendants.
- Accessing the patient to monitor vital signs or administer cardiopulmonary resuscitation is more difficult.
- It is difficult to maintain sanitary conditions if vomiting or diarrhea develops.

Although the first two criticisms may appear physiologically appropriate, review of the medical literature fails to provide documentation that a rise in body temperature after ice-water immersion or shivering is a problem.[46] In fact, vascular resistance decreased during ice bath cooling and persisted until normothermia was achieved.[136] This is an expected observation. The hypothalamic set point for temperature regulation is not

raised during heatstroke (unlike during febrile illness), and brain temperature accounts for approximately 90% of the thermoregulatory response, compared with the skin's 10%.[152] The shivering response should occur only if body temperature is allowed to fall below normal. When shivering occurred, IV chlorpromazine treatment (25 to 50 mg) was effective.[90] Heatstroke victims rarely require cardiopulmonary resuscitation, so this concern should not preclude use of ice baths to treat heatstroke. The documented efficacy of ice-water immersion in rapidly reducing body temperature, and therefore morbidity and mortality, overrides any consideration of transient personal discomfort for the patient or medical attendants.

If other methods are used initially, any victim whose T_c does not reach 38.9°C (102.2°F) within 30 minutes after beginning treatment should be placed in a tub containing ice water or on a stretcher above the tub and covered with ice water–drenched sheets and massaged.[67] The tub should be deep enough for submersion of the neck and torso. Rapidly falling T_c may not be accurately reflected by measured T_{re},[36] so, with any cooling technique, active cooling should be discontinued when core body temperature falls to 39°C (102.2°F) to prevent inducing hypothermia. Ducharme and colleagues,[143] however, suggested that to avoid hypothermia, the cooling of body core temperatures of hyperthermic individuals should not go below 38.5°C (101.3°F)—that is, it is not necessary to eliminate all of the heat gained.

In summary, ice-water treatment cools EHS patients fastest, can be easily set up with little training, is available in most hospitals without purchasing capital equipment, and may also be used on classic heatstroke victims. However, when treating older adults with classic heatstroke, a case-by-case decision should be made that balances the risk of a theoretical, but never shown, harmful stress by ice-water treatment, against the clear benefit of rapid cooling. Otherwise, cold-water or ice-water cooling is the method of choice.

Various ancillary modalities have been proposed to facilitate cooling, including administration of cold IV fluids, gastric lavage with cold fluids, and inhaling cooled air. Although these therapies lower body temperature, their effects are minimal compared with ice-water immersion. Cooling blankets are ineffective for inducing the rapid lowering of body temperature required in treatment of heatstroke. Use of antipyretics is inappropriate and potentially harmful in heatstroke victims. Aspirin and acetaminophen lower temperature by normalizing the elevated hypothalamic set point caused by pyrogens; in heatstroke, the set point is normal, with temperature elevation reflecting failure of normal cooling mechanisms. Furthermore, acetaminophen may induce additional hepatic damage, and administration of aspirin may aggravate bleeding tendencies. Alcohol sponge baths are inappropriate under any circumstances, because absorption of alcohol may lead to poisoning and coma.

HOSPITAL EMERGENCY MEDICAL TREATMENT

If airway control was not previously established, a cuffed endotracheal tube should be inserted to protect against aspiration of oral secretions (Fig. 11-4). Supplemental oxygen and, when necessary, positive-pressure ventilation should be provided. Temperature should be monitored at 5-minute intervals by means of an esophageal or rectal probe. Cooling measures should be maintained for T_c greater than 38°C (100.4°F).

IV access should be obtained as quickly as possible. In the emergency department, IV fluid should be administered to EHS victims as a bolus of 1 L (1.05 qt). Administration of additional fluid should be based on the clinical situation after laboratory results are obtained; the object is to support the circulatory system without risk of inducing pulmonary or cerebral edema. Most heatstroke victims arrive with high cardiac index, low peripheral vascular resistance, and mild right-sided heart failure with elevated central venous pressure. Only moderate fluid replacement is indicated if effective cooling results in vasoconstriction and increased blood pressure. A Swan-Ganz pulmonary artery catheter may be necessary to assess appropriate fluid supplementation. Some victims have low cardiac index, hypotension, and elevated central venous pressure. These persons have been successfully treated with an isoproterenol drip (1 mg/min).[136] Patients with low cardiac index, low central venous pressure, hypotension, and low pulmonary capillary wedge pressure should receive fluid.

Cardiac monitoring should be maintained during at least the first 24 hours of hospitalization. Use of norepinephrine and other α-adrenergic drugs should be avoided because they cause vasoconstriction, thereby reducing heat exchange through the skin. Anticholinergic drugs that inhibit sweating, such as atropine, should also be avoided.

As previously discussed, chlorpromazine may be used to treat uncontrollable shivering that might lead to rising body temperature. However, chlorpromazine should be used advisedly because it may cause hypotension or seizures, and its anticholinergic effects may interfere with sweating. For these reasons, some physicians prefer to use diazepam to control shivering.

A Foley catheter should be placed to monitor urine output. Myoglobinuria and hyperuricemia can be prevented by promoting renal blood flow by administering IV mannitol (0.25 mg/kg) or furosemide (1 mg/kg).[160] Early dialysis should be reconsidered if anuria, uremia, or hyperkalemia develops. Cooling and hydration usually correct any acid–base abnormality; however, serum electrolytes should be monitored and appropriate modifications of IV fluids made. Glucose should be monitored repeatedly because either hypoglycemia or hyperglycemia may occur after heatstroke.[156] Oral and gastric secretions are evacuated via a nasogastric tube connected to continuous low suction. Although antacids and histamine-2 blockers have been used to prevent gastrointestinal bleeding, no studies to date demonstrate their efficacy in heatstroke victims.

As previously discussed, clotting disturbances peak 18 to 36 hours after onset of heat injury.[164] Coagulation tests (platelet count, prothrombin time, fibrinogen levels, fibrin split products) should be obtained on admission and after 24 hours. DIC may develop 24 to 72 hours after admission and is marked by acute onset of bleeding from venipuncture sites, gingivae, nasal mucosae, lungs, or the gastrointestinal tract. DIC is best prevented by rapid cooling of initial hyperthermia and replacement of clotting factors and platelets by transfusion of fresh frozen plasma and platelets.

Acute hepatic dysfunction is exhibited by elevated levels of aminotransferases and bilirubin. Peak levels are seen 36 to 72 hours after collapse. These high levels may last for several days.[61,125,162] Muscle damage is displayed primarily by marked elevation of serum CPK activity levels, which peak 24 to 48

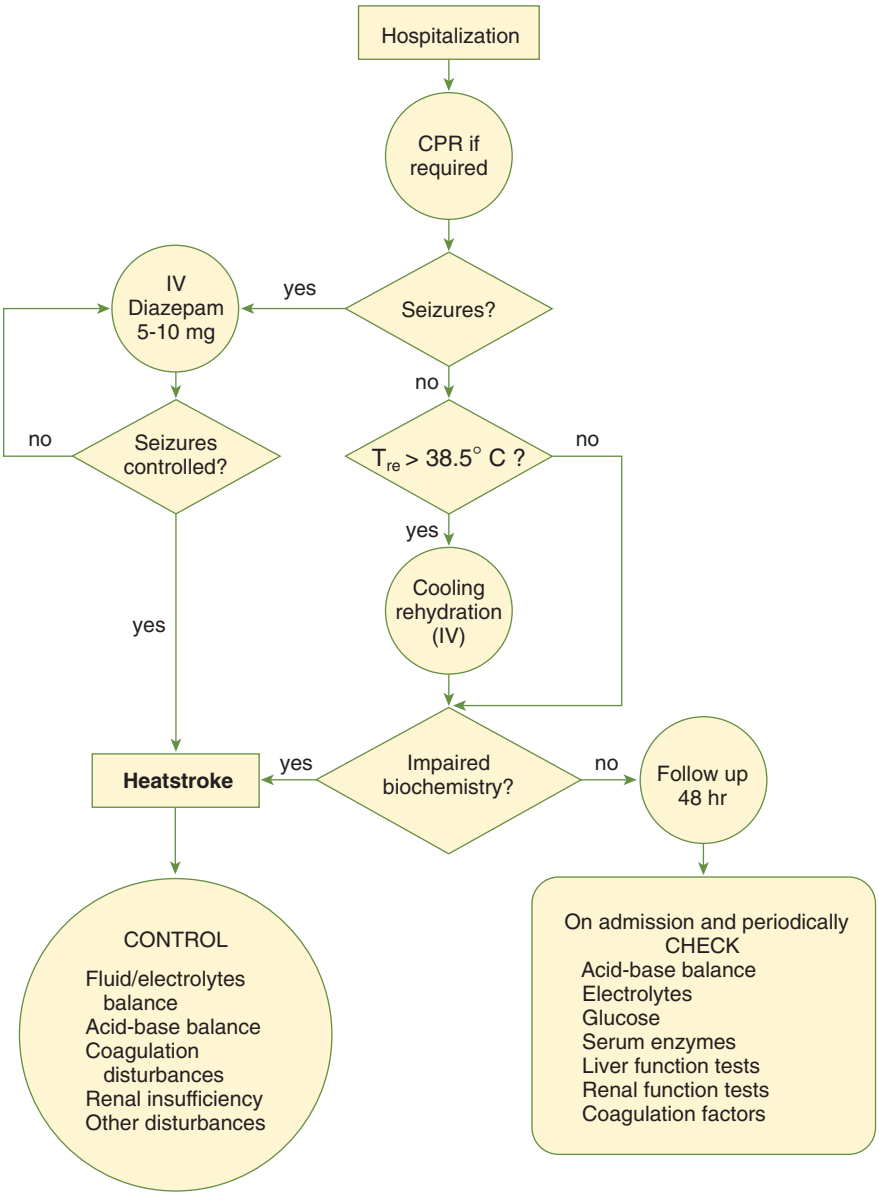


Figure 11-4. Flow chart for hospital medical treatment of exertional heat illnesses; CPR, cardiopulmonary resuscitation; IV, intravenous; T_{re}, rectal temperature. (Modified from Shapiro Y, Seidman DS: Med Sci Sports Exerc 22:6, 1990.)

hours after collapse and usually return to normal spontaneously within 5 days. Muscle and liver enzymes and bilirubin values should be carefully followed, but drastic intervention (e.g., liver transplant) is rarely necessary.

Prognosis

The combination of rapid reduction of body temperature, control of seizures, proper rehydration, and prompt evacuation to an emergency medical facility results in a 90% to 95% survival rate in heatstroke victims, with morbidity directly related to duration of hyperthermia.[159] A poor prognosis is associated with a T_c of greater than 41°C (105.8°F), prolonged duration of hyperthermia, hyperkalemia, acute renal failure, and elevated serum levels of liver enzymes. Therefore, misdiagnosis, early inefficient treatment, and delay in evacuation are the major causes of deterioration in the patient's condition. Full recovery without evidence of neurologic impairment has been achieved even after coma of 24 hours' duration and subsequent seizures.[162] Persistence of coma after return to normothermia is a poor prognostic sign.[164] Neurologic deficits may persist, but usually for a limited period of 12 to 24 months, and only rarely for longer. One recent study of classic heatstroke reported that 33% of patients left the hospital with some neurologic impairment.[48]

Dantrolene

No drug has been found to have a significant effect in reducing body temperature. Antipyretics are ineffective because the thermoregulatory set point is not affected in heatstroke. Furthermore, antipyretics might be harmful, as they cannot be readily

metabolized in the heat-affected liver. However, dantrolene has been used quite successfully in the treatment of several hypercatabolic syndromes, such as malignant hyperthermia, neuroleptic malignant syndrome, and other conditions characterized by muscular rigidity or spasticity.[171,179] Dantrolene stabilizes the Ca^{2+} release channel in muscle cells, reducing the amount of Ca^{2+} released from cellular calcium stores. This lowers intracellular Ca^{2+} concentrations, muscle metabolic activity, and muscle tone, and thus heat production.[32,134] In some studies, dantrolene was claimed to be effective in treating heatstroke, but in others it improved neither the rate of cooling nor survival.[30,49,121,173] In six rhabdomyolysis patients, intramuscular Ca^{2+} concentrations were 11 times higher than in controls, and dantrolene successfully lowered this elevated Ca^{2+}.[118] Collectively, the limited data available are at best inconsistent. In spite of growing evidence for a possible benefit of dantrolene treatment in heatstroke, justification for its routine use in such cases is not proved, although future clinical trials may change this assessment.[9,22,116,144]

Moran and colleagues[126] studied dantrolene in a hyperthermic rat model. They found it effective as a prophylactic agent in sedentary animals only. Dantrolene induced more rapid cooling by depressing Ca^{2+} entry into the sarcoplasm. This led to relaxation of peripheral blood vessels with attenuated production of metabolic heat. Dantrolene also may be effective in treating heatstroke by increasing the cooling rate. However, in other animal models, dantrolene was not superior to conventional cooling methods.[185]

Neuroleptic Seizure

Psychiatric patients treated with high doses of neuroleptic drugs, antidepressants, antiemetics, and other drugs[52] may develop fatal neuroleptic malignant syndrome, characterized by hyperthermia as high as 42°C (107.6°F), "lead pipe" (skeletal muscle) rigidity, dyspnea, coma, extrapyramidal syndrome, rhabdomyolysis, severe metabolic acidosis, leukocytosis, and elevated creatine kinase.[39,84,91] Successful treatment of these cases includes immediate withdrawal of the offending drug, administration of dantrolene, and either oral bromocriptine or the combination of levodopa and carbidopa.[52]

PREVENTION

Prevention of heat illness relies on awareness of host risk factors, altering behavior and physical activity to match these risk factors and environmental conditions, and a requirement for appropriate hydration during physical exercise in the heat. More aggressive educational activity of the media explaining heat illness and its prevention to the public is to be strongly promoted. Primary care physicians should incorporate this information in the anticipatory guidance of routine health assessment. Despite a wealth of medical literature on heat injury, some athletic coaches continue to use physical or psychological methods to force athletes to compete or run under intolerably hot conditions. This practice should be viewed as irresponsible, dangerous, and possibly criminally negligent.

Awareness of Host Risk Factors

Any underlying condition that causes dehydration or increased heat production, or that causes decreased dissipation of heat, interferes with normal thermoregulatory mechanisms and predisposes an individual to heat injury. Older individuals are less heat tolerant than are younger persons to EHI, and are more susceptible to classic heatstroke because of decreased secretory ability of sweat glands and decreased ability of the cardiovascular system to increase blood flow to the skin. When healthy young adults exercise strenuously in the heat, EHS may occur despite the absence of host risk factors. In particular, persons with type II muscle fiber predominance are more susceptible to EHS because these fibers are "faster" but less efficient than are other fiber types.[89] In principle, because women have a thicker subcutaneous fat layer and a T_c that is 0.4° to 0.5°C (0.7° to 0.9°F) higher during the luteal phase than in the follicular phase, they may be at greater risk for heat injury during the luteal phase, but this has not been documented in controlled studies.[169]

Elite and professional athletes, the general public, and the military have widely used ergogenic aids, such as the herb ephedra (*ma huang*) containing ephedrine, to improve performance and lose weight. Because ephedra increases metabolic rate, it has caused numerous cases of heat illnesses and deaths worldwide and is banned. Because there are no clear ergogenic benefits in using ephedra alone, use of ephedra-containing substances should be discouraged.[137]

In children, the ratio of basal metabolic rate to surface area is higher than it is in adults. As a result, the child's T_{sk} is higher. Although the secretory rates of sweat glands are lower in children, they have greater numbers of active sweat glands per area of skin than do adults and overall greater sweat rates per unit area.[88] Any reduction in sweat rates would therefore put children especially at risk.

Endocrine abnormalities, such as hyperthyroidism and pheochromocytoma, cause a marked increase in heat production. Acute febrile illness, by virtue of the elevated hypothalamic set point caused by pyrogens, also leads to increased heat production. Muscular activity associated with uncontrolled gross motor seizures or delirium tremens also releases significant metabolic heat.

The primary means of heat dissipation is production and evaporation of sweat. Any condition that reduces this process places the individual at risk for thermal injury. Poor physical conditioning, fatigue, sleep deprivation, cardiovascular disease, and lack of acclimation all limit the cardiovascular response to heat stress. Obesity places an individual at risk from reduced cardiac output, increased energy cost of moving extra mass, increased thermal insulation, and altered distribution of heat-activated sweat glands.[128] Older adults and the young show decreased efficiency of thermoregulatory functions and increased risk of heat injury.

Several congenital or acquired abnormalities affect sweat production and evaporation. Ectodermal dysplasia is the most common form of congenital anhidrosis. Widespread psoriasis, scleroderma, miliaria rubra ("prickly heat," caused by plugging of the sweat ducts with keratin), or deep burns may also limit sweat production.

Dehydration affects both central thermoregulation and sweating. A mere 2% decrease in body mass through fluid loss produces an increase in HR, an increase in T_c, and a decrease in PV. In an otherwise healthy adult, gastrointestinal infection with vomiting and diarrhea may cause sufficient dehydration to place the individual at risk for EHS. Chronic conditions that may contribute to dehydration include diabetes mellitus, diabetes insipidus, eating disorders (especially bulimia), and mental retardation. Alcoholism and illicit drug use are among the 10

Box 11-3. Drugs That Interfere with Thermoregulation

DRUGS THAT INCREASE HEAT PRODUCTION
- Thyroid hormone
- Amphetamines
- Tricyclic antidepressants
- Lysergic acid diethylamide (LSD)

DRUGS THAT DECREASE THIRST
- Haloperidol

DRUGS THAT DECREASE SWEATING
- Antihistamines (diphenhydramine)
- Anticholinergics
- Phenothiazines
- Benztropine mesylate

From references 16, 46, 81, 108, 162, 164.

major risk factors for heatstroke in the general population.[104] An important effect of alcohol consumption is inhibition of ADH secretion, leading to relative dehydration.

Despite evidence that hypohydration limits physical performance, voluntary dehydration continues to be routine in certain athletic arenas.[9,13,28,174] Wrestlers, jockeys, boxers, and bodybuilders commonly lose 3% to 5% of their body mass 1 to 2 days before competition. In addition to restricting fluid and food, they use other pathogenic weight control measures, such as self-induced vomiting, laxatives and diuretics, and exposure to heat (saunas, hot tubs, and "sauna suits"). Athletes undergoing rapid dehydration are at risk not only for heat injury but also for other serious medical conditions, such as pulmonary embolism.[47]

Box 11-3 highlights common medications that interfere with thermoregulation. Special attention should be paid to the role of antihistamines in reducing sweating. This class of medications is commonly obtained over the counter, and the general population should be warned of the dangers of exercising in the heat when taking antihistamines.

Although it has been widely believed that sustaining an episode of heatstroke predisposes the individual to future heat injury, this has been refuted in a recent study of heatstroke victims.[14] Ten heatstroke patients were tested for their ability to acclimate to heat; by definition, the ability to acclimate to heat indicates heat tolerance. Nine of these patients demonstrated heat tolerance within 3 months after the heatstroke episode; the remaining patient acclimated to heat a year after his heat injury. In no case was heat intolerance permanent. Although individuals may show transient heat intolerance after thermal injury, evidence for permanent susceptibility to thermal injury is lacking.

Adaptation to Environmental Conditions

Appropriate adaptation to hot environmental conditions encompasses many forms of behavior, including modification of clothing, degree of physical activity, searching for shade, anticipatory enhancement of physical conditioning, acclimation to heat stress, and attention to hydration.

Clothing

Different regions of the body are not equivalent in their sweat production.[87] The face and scalp account for 50% of total sweat production, whereas the lower extremities contribute only 25%. When exercising under conditions of high heat load, maximal evaporation of sweat is facilitated by maximal exposure of skin. Clothing should be lightweight and absorbent. Although significant improvement has been made in the fabrication of athletic uniforms, the uniforms and protective gear required by certain branches of the military and public safety officers continue to add to the risk of heat injury. Development of protective clothing that permits more effective heat dissipation is indicated.

Activity

Behavioral actions can effectively minimize the occurrence of classic heatstroke. Lack of residential air conditioning places indigent persons at risk during heat waves. By sitting in a cool or tepid bath periodically throughout the day, the individual can decrease the heat stress and thereby prevent heat injury. The more than 10,000 deaths in the 2003 heat wave in Europe could have been reduced by simple announcements by public health officials of exactly this preventive measure.

Modification of physical activity should not be based solely on any individual parameter of T_{amb}, wet bulb temperature or relative humidity, or solar radiation, as all of these contribute to heat load. The wet bulb globe temperature (WBGT) is an index of heat stress that incorporates all three factors. This value may be calculated (see Table 11-2) or obtained directly from portable heat stress monitors that measure all three parameters simultaneously to compute the WBGT. Alternatively, the heat index may be obtained from national weather stations. Current recommendations from the American College of Sports Medicine (ACSM) for prevention of thermal injuries during distance running are based on the WBGT.[8] It is stated that "distance races (≥16 km or 10 miles) should not be conducted when the WBGT exceeds 28° C (82.4° F). During periods of the year when the daylight T_{amb} often exceeds 27° C (80° F), distance races should be conducted in the early morning or in the evening to minimize the heat load from T_{amb} and solar radiation."[8] In the British Army, the strenuous Combat Fitness Test (CFT) occasionally leads to heat casualties. To prevent a mean rise in T_c of 0.7° C (1.26° F) and to minimize heat illnesses, calculations indicate that the CFT should not be undertaken when the end WBGT is expected to be greater than 25° C (77° F).[26]

Table 11-3 presents a suggested modification of sports activity that is also based on the WBGT. Although ACSM guidelines for summer indicate that vigorous physical activity should be scheduled in the mornings or in the evenings, it should be cautioned that the highest humidity of the day is usually during early morning. In 1999, Montain and coworkers[124] updated the replacement guidelines for warm weather training (see Chapter 64, Table 64-7). It is important to note that compliance with these recommendations does not remove all risk of heat injury. Developing another index of heat stress that provides a better basis for prevention of EHS is indicated. Recently, a new, user-friendly, miniaturized 5.1 × 2.5 × 1.3 cm (2- × 1- × 0.5-inch) device based on measuring T_{amb} and RH with microsensors was developed for assessment of heat stress.[129] However, further miniaturization and evaluations of this device are required.[3]

TABLE 11-3. Modification of Sports Activity on the Basis of Wet Bulb Globe Temperature (WBGT)

INDEX (°F)	LIMITATION
<50	Low risk for hyperthermia but possible risk for hypothermia
<65	Low risk for heat illness
65–73	Moderate risk toward end of workout
73–82	Those at high risk for heat injury should not continue to train; practice in shorts and T-shirt during the first week of training.
82–84	Care should be taken by all athletes to maintain adequate hydration.
85–87.9	Unacclimated persons should stop training; all outdoor drills in heavy uniforms should be canceled.
88–89.9	Acclimated athletes should exercise caution and continue workouts only at a reduced intensity; light clothing only.
90 or above	Stop all training.

Fatal Exertional Heatstroke. Rav-Acha and colleagues[145] assembled a case series of six fatal cases of EHS in the Israeli Defense Forces and examined the circumstances that led to the deaths. A significant association between accumulation of predisposing factors and EHS totality was found. In almost all the fatal cases, seven predisposing factors were noticed: low physical fitness, sleep deprivation, high heat load, high solar radiation, physical exercise unmatched to physical fitness, absence of proper medical triage, and training during the hottest hours. These fatality factors primarily concerned organizational training regulations and not individual factors, which emphasizes the importance of proper guidelines and safety measures in a warm climate, rather than individual characteristics, for preventing EHS fatalities. It follows that a combination of predisposing factors that were already found to impair heat tolerance[70] is a strong predictor of a grave prognosis. Dehydration was found in only two of the six cases reported.

Pathologic findings for EHS were seen only in the three victims that were exposed to heat for longer times and to longer durations of exercise in the heat. These pathologic pictures revealed that EHS is a function of the dynamic development of the syndrome, including the precollapse duration (period of time prior to hyperthermic collapse), and not simply the time elapsed from collapse to death, as was suggested by Shibolet and coworkers.[164] This perspective[145] is in line with the pathophysiology model suggested by Bouchama and Knochel,[24] who emphasized the cascade of events that contribute to the progression from heat stress to heatstroke. According to their study, the characteristic clinical and pathologic picture of heatstroke is a complex interplay among the physiologic and biochemical alterations during the precollapse period, which include thermoregulatory failure, exaggeration of the acute phase response, and the expression of heat shock proteins.

Conditioning

The contribution of cardiovascular conditioning to thermoregulation is discussed in Chapter 4. Ideally, an individual should train under temperate or thermoneutral conditions before exercising in the heat. For the previously sedentary individual, an exercise regimen incorporating 20 to 30 minutes of aerobic activity 3 to 4 days a week will improve cardiovascular function after 8 weeks.

It is important to remember that even physically active individuals may lack physical conditioning relative to a particularly stressful competition or activity. Heat illness in runners commonly occurs when novices exceed their training effort during races or when well-trained athletes increase their pace above normal during long-distance events.

Acclimatization

On initial exposure to a hot environment, workouts should be moderate in intensity and duration. A gradual increase in the time and intensity of physical exertion over 8 to 10 days should allow optimal acclimatization.[54] Children require 10 to 14 days to achieve a similar response. Acclimitization can be induced by simulating hot environmental conditions indoors. If symptoms of heat illness develop during the acclimation period, all physical activity should be stopped and appropriate interventions begun. Acclimitization is not facilitated by restricting fluid intake; in fact, conscious attention to fluid intake is required to prevent dehydration. As with physical conditioning, there are limits to the degree of protection that acclimitization provides from heat stress. Given a sufficiently hot and humid environment, no one is immune to heat injury.

The references for this chapter can be found on the accompanying DVD-ROM.

12 Wildland Fires: Dangers and Survival

Martin E. Alexander, Robert W. Mutch, and Kathleen Mary Davis

There can be few natural physical phenomena with the scope and complexity of a forest fire. The fuel that powers it is found in a huge range of sizes, quantities, and arrangements in space. The weather affects the current condition of this fuel array in a bewildering maze of drying and wetting effects, each fuel component responding to a "different drummer." The combustion process itself, once under way, responds to a complex blend of fuel variation, moisture status, topography, wind speed, and other atmospheric factors. Its frontal intensity varies over an immense range, from tiny flickers easily stepped over, to dense sheets of flame whose fierce radiation keeps the observer at a distance.

—Van Wagner (1985)

In describing the 13 wildland firefighter fatalities that occurred on the Mann Gulch fire near Helena, Montana, on August 5, 1949, author Norman Maclean[226] wrote in his seminal book *Young Men and Fire*, published in 1992, that "They were still so young they hadn't learned to count the odds and to sense they might owe the universe a tragedy." Three years later, Canadian folk singer-songwriter James Keelaghan, inspired by Maclean's book, paid tribute to the fallen firefighters in a haunting ballad entitled "Cold Missouri Waters." The Mann Gulch fire has been called "the race that couldn't be won."[328] Although the crew increased their pace ahead of the fire, the fire accelerated faster than they did until fire and people converged. Miraculously, three people survived the fire. Smokejumper foreman Wag Dodge ignited an "escape fire" by burning off a patch of cured grass into which he tried to move all of his crew, whereas two others found a route to safety and escaped injury on a nearby rockslide.

Many improvements in a firefighter's odds of surviving an entrapment or burnover encounter with a wildland fire have occurred since 1949. These advances include improved understanding of fire behavior, increased emphasis on fire safety and fire training, and development of personal protective equipment. However, as incidents such as the 14 firefighter fatalities on the 1994 South Canyon fire in Colorado (described by Norman Maclean's son John 50 years later[223]) have shown, tragedies continue to occur.[141] Grief over lost loved ones can sometimes lead to further tragedies. For example, one of the smokejumpers killed on the 1949 Mann Gulch fire, Stan Reba, had been married less than a year. His widow grieved for him and never remarried, and then 10 years later took her own life.[195]

There are very few comprehensive summaries of deaths due to wildland fires globally. The compilation of U.S. wildland firefighter fatalities begun in the mid-1970s[416] is unique.[274] Statistics on the number of civilian fatalities due to being trapped or overrun by wildland fires on a global basis are unfortunately not kept in any systematic manner.[193] More than 300 deaths were reported to have occurred in the 20th century in the state of Victoria in Australia.[205] Wildland fires are as much a threat to human life, property, and natural resources on the North American continent and many other regions of the world (Fig. 12-1) as are hurricanes, tornadoes, floods, and earthquakes.[69] Of course, the total number of deaths among the general public due to wildland fires in modern times pales in comparison to the death toll and destruction resulting from the tsunami of December 26, 2004 in southeast Asia or in the wake of Hurricane Katrina along the U.S. Gulf Coast on August 29, 2005.

This chapter describes the current look of fire as a historical force and discusses fire management policies, the nature and scope of wildland fire hazards, behavior of fires, typical injuries, fatality fire statistics, several fatality fire incidents, and survival techniques. Although the emphasis is on North America, reference is made to other regions of the globe, most notably Canada and Australasia. Wildland fire, like many other disciplines and subjects, has its own unique terminology, so readers may need to consult glossaries.[240,279]

WILDLAND FIRE MANAGEMENT AND TECHNOLOGY

Programs for dealing with the overall spectrum of fire are collectively termed *fire management*.[44] They are based on the concept that fire and the complex interrelated factors that influence fire phenomena can and should be managed. The scientifically sound fire management programs that respond to the needs of people and natural environments must also maintain full respect for the power of fire.[44]

Since the early 1900s, federal, state, and local fire protection agencies in the United States, for example, have routinely extinguished wildland fires to protect watershed, range, and timber values, as well as human lives and property. The basic methods of fire suppression[174,220] have changed very little, although new technologies have gradually come on board (Fig. 12-2). However, improvements in fire detection, fire danger rating systems, and fire suppression methods have been developed by fire science laboratories and two equipment development

Figure 12-1. A wildland–urban interface fire in the Amarante region of Portugal on August 5, 2005. Drought conditions contributed to an exceptionally severe fire season in Southern Europe during 2005. Twelve firefighters and 10 civilians were killed in Portugal. Eleven firefighters were killed in one separate incident in a nature reserve in the Guadalajara area east of Madrid, Spain on July 17, 2005. (Courtesy *Publico* newspaper. Photo by Paulo Ricca.)

centers maintained by the U.S. Department of Agriculture (USDA) Forest Service, as well other organizations (e.g., state agencies and universities). Patrol planes, some with infrared heat scanners, and other fixed-wing aircraft (Fig. 12-3), including helicopters (Fig. 12-4), can deliver firefighters, equipment, and fire-retarding chemicals or water to the most remote fire. These firefighting resources are organized under an Incident Command System that can easily manage simple to complex operational, logistical, planning, and fiscal functions associated with wildfire suppression actions.[94,153,267] Many other countries have followed a somewhat similar path in their evolution with regard to wildland fire suppression and supporting research and development.[309–311,314]

Modern fire suppression technology, however, cannot indefinitely reduce the area burned by wildfires, as demonstrated, for example, by numerous large fires and major fire seasons in the past 25 years or so in North America: Mack Lake fire in Michigan in 1980; the "Siege of '87" in California in 1987; Silver Complex in Oregon in 1987; Greater Yellowstone Area fires in 1988; Stanislaus Complex in California in 1989; Foothills fire near Boise, Idaho, in 1992; Garnet fire near Penticton, British Columbia, in 1994; Millers Reach fire in Alaska in 1996; Virginia Hills fire in Alberta in 1998; Florida fires of 1998; Silver Creek fire in British Columbia in 1998; Cerro Grande fire in New Mexico in 2000; Valley Complex in Montana in 2000; Chisholm fire in Alberta in 2001; Biscuit fire in Oregon in 2002; Hayman fire in Colorado in 2002; Rodeo/Chedeski fire in Arizona in 2002; House River fire in Alberta in 2003; "Firestorm 2003" season in British Columbia; and record-setting fire seasons in Alaska and the Yukon Territory in 2004 (Fig. 12-5), to name a few. Similar incidents have occurred in other regions of the world. The hard lesson learned from these and other fires is that wildfires are inevitable. Several factors have coincided to produce massive forest mortality, including drought, epidemic levels of insects and diseases,[197,258,261] and unnatural accumulations of fuels at the stand level and at a

A

B

Figure 12-2. A helitorch or flying drip torch is a specialized aerial ignition device slung below and activated from a helicopter **(A).** It dispenses and ignites gelled fuel. The use of helitorch in wildfire suppression operations can take many forms (e.g., to reduce the intensity, slow, or steer a fire; to remove potentially dangerous fuel concentrations; to widen and strengthen control lines; to expedite mop-up operations). Regardless of its specific purpose, the main objective of this application of "fighting fire with fire" is to speed up or strengthen control actions **(B).** Aerial backfiring and burning out with a helitorch can be one of the safest, most economical, fastest, and least damaging means of widening control lines and constitutes one of the major innovations in wildland fire suppression technology in the past 30 years. The helitorch is also used in prescribed burning operations. (Courtesy Alberta Sustainable Resource Development and Wildfire Consulting Ltd., Edmonton, Alberta. Photos by O. Spencer and W. Bereska.)

Figure 12-3. Specialized fixed-wing aircraft like the amphibious CL-415 "superscooper" are capable of delivering water and chemical fire retardants on or near a wildfire. Although such planes are used in sustained-action situations on isolated sectors of large fires, they are most effective in initial attack operations during the incipient phase of fire growth. In such cases, their primary purpose is to slow down the progress of the fire until such time as ground suppression resources are able to arrive and effect direct containment. Wing-tip vortices are created in the wake of low-flying aircraft. These vortices gradually dissipate as they descend downward to the ground level. They can cause a sudden, unexpected increase in fire activity as a result of the momentary wind gust. This can pose a threat to anyone located near the fire perimeter. (Photo by T. Nebbs.)

Figure 12-4. Several different sizes and models of rotary-wing aircraft or helicopters are used in a wide variety of direct roles as well as support functions in wildland fire suppression, including the dropping of water or fire-retarding chemicals directly on the fire. Helicopters also create wake vortices while in forward motion just like airplanes, but generally much weaker. A more common safety issue with helicopters is the downward induced air flow or rotary "downwash." Some downwash effects are unavoidable when helicopters are engaged in direct attack on the fire with water or chemicals. Anyone located near the fire perimeter during such operations could be at risk. Even with a "long line" delivery system operated from a Bell 212 helicopter, as illustrated here on the Anarchist Mountain fire near Osoyoos in southern British Columbia on July 16, 2003, rotary downwash can still be a significant issue. (Courtesy Alberta Sustainable Resource Development.)

growing landscape scale in some but not necessarily all vegetation types[198] as a result of attempted fire exclusion.[17,175] This result in the dry forest types of the western United States was foreshadowed by a forester, Harold Weaver,[402] in the early 1940s. Many agencies are now using prescribed fire more frequently, deliberately burning under predetermined conditions to reduce accumulations of fuels and to protect human life, property, and other values that are at risk by wildfire.[241,259,288]

Research has indicated that fires are not categorically bad. In fact, many plant communities in North America are highly flammable during certain periods in their life cycles. For example, annual grasses, ponderosa pine, and chaparral plant communities are flammable almost every dry season. Other communities, such as jack pine or lodgepole pine forests, although fire resistant during much of their life cycle, eventually become fire prone when killed by insects, diseases, and other natural causes. The spread of non-native grasses, such as cheatgrass and red brome, in the arid regions of the western United States has increased the frequency of fires in desert shrub lands.

Wildland fires can benefit plant and animal communities. Evolutionary development produces plant species well adapted to recurrent fires. Fire tends to recycle ecosystems and maintain diversity.[219,420] Thus, there is growing consensus that fire should be returned to wildland ecosystems where appropriate to perpetuate desirable fire-adapted plant and animal communities and to reduce fuel accumulations.

A landmark report in 1963 to the National Park Service by the Advisory Board on Wildlife Management[213] described how the western slope of the Sierra Nevada had been transformed by fire protection:

> *When the forty-niners poured over the Sierra Nevada into California, those that kept diaries spoke almost to a man of the wide-spaced columns of mature trees that grew on the lower western slope in gigantic magnificence. Today much of the west slope is a dog-hair thicket of young pines, white fir, incense-cedar, and mature brush—a direct function of overprotection from natural ground fires. Not only is this accumulation of fuel dangerous to the giant sequoias and other mature trees, but the animal life is meager, wildflowers are sparse, and, to some at least, the vegetative tangle is depressing, not uplifting.*

It must also be acknowledged that in some cases, past logging practices contributed to the resulting fuel structure. Neverthe-

Figure 12-5. Swan Lake fire in northern British Columbia (near the border with the Yukon Territory) during mid-afternoon on June 23, 2004. Such high-intensity fire events continue to spread until such time as burning conditions ameliorate, for example, when a change in weather conditions (e.g., winds die down) or fuel type (e.g., leafed-out hardwood stand) occurs, or a wide, natural barrier to fire spread is encountered (e.g., large lake or river). The natural fire regime in the boreal forest of Canada and neighboring Alaska is characterized by the periodic occurrence of large crown fires. In the Yukon, for example, the average area burned over the past 25 years has been about 120,000 hectares (about 297,000 acres) per year. During the 2004 fire season, a record-setting 1.7 million hectares (4.2 million acres) and 2.7 million hectares (6.7 million acres) was burned over in the Yukon and Alaska, respectively. (Courtesy Yukon Government, Department of Energy, Mines, and Resources. Photo by M. Clark.)

less, the board recommended that the Park Service recognize in management programs the importance of the natural role of fire in shaping plant communities.

Wildland Fire Management Policies

Recent tragedies in the western United States have focused attention on the need to reduce hazardous fuel accumulations.[359] The events associated with the 1994 fire season in particular created a renewed awareness and concern among federal land management agencies about wildfire impacts, leading to a combined review of fire policies and programs. The result was enactment of a new interagency federal wildland fire management policy, which provided a common approach to wildland fire among federal agencies and called for close cooperation with tribal, state, and other jurisdictions.[272] The principal points of the new federal wildland fire policies are as follows:

1. Firefighter and public safety remain the first priorities in wildland fire management. Protection of natural and cultural resources and property is the second priority.
2. Wildland fire, as a critical natural process, must be reintroduced into the ecosystem, accomplished across agency boundaries, and based on the best available science.
3. Where wildland fire cannot be safely reintroduced because of hazardous fuel accumulations, pretreatment must be considered, particularly in the wildland–urban interface.
4. Wildland fire management decisions and resource management decisions are connected and based on approved plans. Agencies must be able to choose from the full spectrum of actions—from prompt suppression to allowing fire to have an ecologic function.
5. All aspects of wildland fire management will involve all partners and have compatible programs, activities, and processes.
6. The role of federal agencies in the wildland–urban interface includes firefighting, hazardous fuel reduction, cooperative prevention and education, and technical assistance. Ultimately, the primary responsibility rests at the state and local levels.
7. Structural fire protection in the wildland–urban interface is the responsibility of tribal, state, and local governments.
8. Federal agencies must better educate internal and external audiences about how and why we use and manage wildland fire.

The 1995 U.S. federal wildland fire management policy was reviewed and updated in 2001[277] following a particularly severe fire season in the western United States the year before; about 2.8 million hectares (about 7 million acres) were burned over by about 89,000 fires during the 2000 fire season in the United States.[386] Although similar to the 1995 policy,[387] the new policy calls for using the full range of fire management activities to achieve ecosystem stability, including fire use. The 2001 policy stresses the need to complete or revise fire management plans that are more effectively and directly integrated with other natural resource goals.[65] One of the other outgrowths of the 2000 fire season was the National Fire Plan (www.fireplan.gov), which calls for the following actions:

1. Ensuring that necessary firefighting resources and personnel are available to respond to wildland fires that threaten lives and property.
2. Conducting emergency stabilization and rehabilitation activities on landscapes and communities affected by wildland fire.
3. Reducing hazardous fuels (dry brush and trees that have accumulated and increase the likelihood of unusually large fires) in the country's forests and rangelands.
4. Providing assistance to communities that have been or may be threatened by wildland fire.
5. Committing to the Wildland Fire Leadership Council, an interagency team created to set and maintain high standards for wildland fire management on public lands.

It is clear from these objectives that one of the major focuses of the U.S. National Fire Plan is the human dimension of wildfire (i.e., rural homeowners and communities).[194,222]

Similar fire management policies exist in other regions of the world.[159] On October 4, 2005, the Canadian Council of Forest Ministers signed off on the Canadian Wildland Fire Strategy Declaration,[89] which is based on principles of risk management and hazard mitigation. This strategy will strive to strike a balance between public safety, forest protection and health, and fire management expenditures that maintains a strong and effective fire suppression organization, but also includes innovative hazard mitigation, preparedness, and recovery programs—in other words, a more holistic view of wildland fire envisioned some 35 years ago as land management agencies made the transition from fire control to fire management. This strategy recognizes the need for shared responsibility among the various stakeholders (i.e., property owners, industries, and local, provincial, and federal governments). One of the guiding principles of this new declaration was that "public safety—including the safety of firefighters—is paramount."

Figure 12-6. Lightning-ignited fires in some wilderness areas and national parks in the United States that meet certain criteria have been allowed to burn freely under observation since the early 1970s as part of a fire management strategy to allow natural ecosystem processes to operate more freely. (Courtesy USDA Forest Service.)

Prescribed Fire and Wildland Fire Use

Prescribed fire, the intentional ignition of grass, shrub, or forest fuels for specific purposes according to predetermined conditions, is a recognized land management practice.[29,399] The objectives of such burning vary: to reduce fire hazards after logging, expose mineral soil for seedbeds, regulate insects and diseases, perpetuate natural ecosystems, and improve range forage and wildlife habitat. In some areas managed by the National Park Service, USDA Forest Service, and Bureau of Land Management, naturally ignited fires may be allowed to burn according to approved prescriptions; fire management areas have been established in national parks and wildernesses from the Florida Everglades to the Sierra Nevada in California (Fig. 12-6). Planned-ignition prescribed fires are also carried out. Similar policies exist in Canadian National Parks. Visitors are increasingly aware that wildland fires can be an important part of the natural cycle of an ecosystem and that the future health of parkland may depend in part on today's managed fires.

WILDLAND–URBAN INTERFACE: NEW LOOK OF A HISTORICAL PROBLEM

Just as resource agencies are attempting to provide a more natural role for fire in wildland ecosystems, the general public is increasingly living and seeking recreation in many of these areas. The area where houses and other human-made structures meet or are intermingled with wildland vegetation is regarded as the wildland–urban interface or intermix. It is a growing problem from a wildland fire perspective in the United States[60,318,380] and many regions of the world.[188] Past fire exclusion practices in the United States, for example, have allowed abnormal fuel accumulations in some forest types and regions, and this fact has combined with the sacrifice of relatively safe perimeter fire suppression strategies in favor of directly protecting people and their possessions.[416] Direct suppression actions within the fire's perimeter place firefighters at a greater disadvantage from a safety standpoint. The new interagency policy that emphasizes firefighter and public safety as the first priority will result in less effort to save structures in dangerous situations. Thus, what is known of fire behavior and fire survival principles must be readily available to emergency medical personnel, wildland dwellers, and recreationists. In fact, fire protection agencies and others have been making such information more readily available to the general public for some time now,[96,136,160,403] although much more needs to be done.[18]

Nature of the Problem

Hot, dry, windy conditions annually produce high-intensity fires that threaten or burn homes where wildland and urban areas converge (Box 12-1). Can the historical levels of destruction, injury, and fatality be repeated today in the face of modern fire suppression technology? The answer to this question requires an analysis of the conditions that created such high-intensity fire behavior events in the forests of northern Idaho and northwestern Montana during late August 1910 (Fig. 12-7) in which 85 people (Fig. 12-8), including 78 firefighters, were killed.[116,312,354]

The earliest fire incident of note involving a large loss of human life was the series of conflagrations in the Miramichi region of New Brunswick and adjacent areas in Maine in early October 1825 in which an estimated 160 people perished.[119,301] This was followed by several fatality fires in the U.S. Lake States region, including the Peshtigo and area fires in eastern Wisconsin in October 1871 (about 1300 fatalities),[187,214,409] the Lower Michigan fires of September 1881 (169 fatalities),[191] the Hinckley fire in east-central Minnesota in September 1894 (418 fatalities),[209] the Baudette fire in northern Minnesota in October 1910 (42 fatalities),[301] and the Cloquet fire in Minnesota in October 1918 (538 fatalities).[90,191] The western United States experienced other fatality fires, such as in western Washington and Oregon in mid-September 1902 (38 fatalities).[64,237] Canada suffered similar tragedies. For example, the province of Ontario, like the U.S. Lake States region, experienced several significant settler-related wildfire fatalities in 1911, 1922, and 1938, including the 1916 "Matheson Holocaust" in which an estimated 400 people were killed.[42,236] In 1908, a forest fire destroyed the city of Fernie, British Columbia, killing nine people.[371]

Other countries, such as New Zealand[239] and France,[160] are reported to have suffered similar but varying losses in the 20th century. In Australia, for example, possessing some of the world's most fire-prone environments,[97] 71 lives were lost in the state of Victoria on "Black Friday" in January 1939.[160,285] On August 1, 1959, 48 people were reported killed by a forest fire northeast of Massif des Aures, Algeria.[193] In early September 1963, forest fires swept about 2 million hectares (about 4.9 million acres) in Paraná State in southern Brazil, destroying more than 5000 homes and killing 110 people.[350] More than 100 people were killed by extensive forest and savannah fires in Cote D'Ivoire (Republic of Ivory Coast) on the southern coast

Box 12-1. What Is It Like to Experience a Wildland–Urban Interface Fire? A Town That Nearly Went . . . Up in Smoke by D. Blasor-Bernhardt[56]

Tok, Alaska, July 1, 1990, 15–30 minutes after the lightning strike that started the Tok fire. (Photo by D. Blasor-Bernhardt.)

July 1, 1990, started out like any other Sunday, but it quickly turned Tok, Alaska, into a near raging inferno. I was working at the Chamber of Commerce Main Street Visitor Centre when an otherwise beautiful day suddenly turned black and foreboding. Blowing in, a huge thundercell hung just southeast of town.

I stepped outside just in time to see a long, crooked finger of electricity split from an ominous cloud and drill its way into earth. Minutes later, where the lightening had hit, a mushroom-shaped cloud began to grow. I grabbed my camera and snapped a few shots.

Five days later, and in spite of several thousand firefighters and equipment from all over Alaska, Canada, and the States, Tok was completely under siege and at risk of being cremated. The wind-blown inferno licked greedily at several unoccupied dwellings, then consumed them. East-side residents were evacuated. We, on the west side, were told to pack our things, to be ready to leave, and to await further instructions.

Tok was enveloped on two sides by fire—from the eastern and northern directions. My log home was about a mile from the north front of the fire. I stood on the cabin's sod roof, watching the fire, when the kids and extended family converged there about the same time, looking to me for instructions. All day, I had the sprinklers and hoses running full blast over the cabins, garage, and surrounding area. By now, the area looked like a swamp. Good. I'd leave them on, even if we evacuated, in the hope that it would help save some of our place from destruction.

Smoke stifled each breath. Helicopters screamed back and forth carrying people, equipment, and large buckets of water.

There was no fear, no panic, in anyone—just resignation. And there was no way our town would be destroyed.

I told the kids to first pack up whatever was closest to their hearts—it didn't matter how silly an item seemed if that made them feel good. The first thing I placed in my own car was my late husband's ashes. I couldn't bear to leave him behind. Next, we would take irreplaceable things: photo albums, mementos. Then down to brass tacks: the necessary things for survival and rebuilding in the event we were burned out. Bombers loaded with retardant flew directly overhead. We were in a war zone—man against fire.

The girls loaded their cars with personal items. Mine was loaded with blankets, groceries, and clothing. The boys readied our tandem-axle trailer, loading it with items from the garage—chainsaws, tools, portable generator, water jugs. If the fire drew too near and we had to evacuate, the plan was for the girls and I to load our pets while the boys quickly cut the trees down around the cabins, hopefully providing a bare, wet perimeter between them and the fire.

Fully loaded, we assembled in the driveway, ready for word to come. Besides myself, six young adults, a baby, half a dozen dogs, numerous cats, a snake, six vehicles, and a trailer comprised the Bernhardt entourage. We were ready. We waited in the foul air, our eyes smarting from the smoke. An ember hit me on the shoulder, burning a hole though my shirt. Sadly, I turned to look at our homes one last time. Tomorrow they would be gone.

Imperceptibly at first, a slight shift in the wind began. I held my breath. "Mom?" my son said. "Yes," I answered, "but I'm afraid to hope." The wind, definitely shifting now, was no longer blowing toward us. A sweet, light rain began to fall. While the eight of us stood together, soaking wet in the rain, a trooper came by to tell us the worst was over.

In all, the fire burned 109,501 acres (44,314 hectares) at a cost of over $35 million (U.S.), yet not one person had been injured, not one occupied dwelling destroyed, and Tok had been spared by a miracle wind.

From Blasor-Bernhardt D: A town that nearly went . . . up in smoke. Guide to the Goldfields and Beyond (Harper Street Publishing, Dawson City, Yukon Territory) Summer: 52, 1997

of western Africa during the 1982–1983 fire season.[339] Greece has reported a number of civilian and wildland firefighter fatalities in recent years.[193]

On October 8, 1871, the same day that fire wiped out the town of Peshtigo, Wisconsin, and surrounding communities, the Great Chicago fire devastated urban Chicago. Comparative statistics for those two fires highlight the destructive potential of wildland fires. In every way, the Peshtigo conflagration was far worst than the Chicago blaze. The Peshtigo fire covered about 518,000 hectares (about 1,280,000 acres) and killed about 1300 people, whereas 906 hectares (2240 acres) burned and some 200 lives were lost as a result of the Chicago fire.[180] The Great Chicago fire is generally acknowledged as the birthplace of modern urban fire prevention. As Lloyd[218] notes, "mention the name Peshtigo to most people and all you get is a blank stare. Mention Mrs. O'Leary's cow to the same person and they will think right away of the Great Chicago fire" and "Some of the same social misconceptions that allowed Peshtigo to be all but forgotten persist today." So it is perhaps fitting to consider the following poem entitled "The Peshtigo Calamity"[191]:

Figure 12-7. These burned-over and wind-thrown trees resulted from the intense fire behavior associated with a forest fire near Falcon, Idaho, in August 1910. (Courtesy USDA Forest Service. Photo by J. B. Halm.)

Figure 12-8. Burned ruins of the foundry in Wallace, Idaho, furnish mute testimony to the destructive force of the 1910 fires. The cottage on the terrace was the only one left standing in that part of town. Of the 85 people reported killed in the "big blowup of 1910," 78 were firefighters. (Courtesy USDA Forest Service. Photo by R. H. McKay.)

As the years roll along and the ages have sped
O'er the charred, blackened bones of the Peshtigo dead,
And the story is told by the pen of the sage,
In letters immortal on history's page—
No fancy can compass the horror and fright
The anguish and woe of that terrible night.

The August 1910 wildfires in northern Idaho and Montana had several elements in common with the Peshtigo fires of 1871: many uncontrolled fires burning at one time; prolonged drought, high temperatures, and moderate to strong winds; and mixed conifer and hardwood fuels with slash from logging and land clearing. These large fires occurred primarily in conifer forests north of the 42nd meridian, or roughly across the northern quarter of the contiguous United States.[64] One of these critical elements that is not as likely to occur today as formerly is the simultaneous presence of many uncontrolled fires. The effectiveness of modern fire suppression organizations is now able to reach even the most remote wildland locations. High-velocity winds and more than 1600 individual fires contributed to the spread of the fires in 1910; it is unlikely that a multifire situation of that magnitude would occur today.[67,97] However, multiple fire starts are still possible owing to human-caused ignitions (e.g., arson, power lines). For example, on October 16, 1991, 92 separate fires occurred in the Spokane region of eastern Washington[264] in the space of a few hours under the influence of exceedingly strong winds.[25] Prolonged drought, high winds, and flammable fuel types, however, remain significant to the behavior of high-intensity fires.

Some of the fires most potentially damaging to human lives and property occur in areas rich in chaparral shrub fuel in southern California. Wilson[415] and Phillips[299] described the severe 1970 fire season in California, in which official estimates showed that 97% of 1260 fires occurring between September 15 and November 15 were held to less than 121 hectares (300 acres). The other 3% of the fires, fueled by a prolonged drought and fanned by strong Santa Ana winds, produced 14 deaths, destroyed 885 homes, and burned about 243,000 hectares (about 600,000 acres), as chronicled in the 1971 film *Countdown to Calamity.* Ten years later, the situation recurred over 9550 hectares (23,600 acres) in southern California, the Panorama fire in November 1980, which resulted in the deaths of 4 people and loss of more than 325 structures.[86]

On October 20, 1991, a devastating fire "of unprecedented force blew out of control"[4] in the hills above Oakland and Berkeley, California. Burning embers carried by high winds from the perimeter of a small fire resulted in a major wildland–urban interface conflagration. The impact on people and their possessions was enormous.[4,369] The fire resulted in direct deaths of 25 people, including a police officer and a firefighter, injured 150 others, destroyed 2449 single-family dwellings and 437 apartment and condominium units, burned about 650 hectares (about 1600 acres), and did an estimated $1.5 billion in damage.[263] The scenario for disaster included a 5-year drought that had dried out overgrown grass, shrubs, and trees, making them readily ignitable. Other factors included untreated wood shingles, unprotected wooden decks that projected out over steep terrain, low relative humidity, high temperatures, and strong winds that averaged 32 km/hr (20 mph) and gusted up to 56 to 80 km/hr (35–50 mph). The area had not experienced any rain for 67 days before the major fire run, although mean monthly maximum temperatures in the 6 months preceding the fire were not appreciably high (i.e., 18° to 23° C, or 64° to 73° F).[26] These severe conditions produced a voracious fire that consumed 790 homes in the first hour. Winds lessened to 8 km/hr (5 mph) by the first evening, which

TABLE 12-1. Approximate Probabilities of a House Surviving a Wildland Fire*

Scenario A: Total disregard for any fire safety precautions
Scenario B: Fire safety precautions taken only in regard to fuels surrounding house
Scenario C: Fire safety precautions taken only in regard to the house itself
Scenario D: All reasonable fire safety precautions to and near the house taken

	WITHOUT PERSONS IN ATTENDANCE FIRE INTENSITY CLASS (kW/m)				WITH PERSONS IN ATTENDANCE FIRE INTENSITY CLASS (kW/m)		
SCENARIO	500–1,500	1,500–10,000	10,000–60,000	SCENARIO	500–1,500	1,500–10,000	10,000–60,000
A	14.4%	1.2%	0.3%	A	29.9%	10.3%	2.3%
B	16.0%	4.9%	1.0%	B	63.6%	32.1%	8.9%
C	26.1%	8.7%	1.9%	C	76.5%	46.8%	15.3%
D	59.1%	28.1%	7.4%	D	93.0%	78.2%	42.6%

kW/m × 0.29 = BTU/sec-ft

The following attributes were considered in the above scenarios:

	SCENARIO			
HOUSE AND SURROUNDING FUEL CONDITIONS	A	B	C	D
Flammable objects nearby (e.g., firewood heap)?	Yes	No	Yes	No
Wooden shingle roof?	Yes	Yes	No	No
Nonwooden or nontile roof with pitch >10°?	No	No	Yes	Yes
External walls made of brick, stone, or concrete?	No	No	Yes	Yes
Trees (5+m high) within 40 m of house?	Yes	No	Yes	No

m × 0.3 = ft.
*Based on a logistic regression model as developed from an analysis of 455 houses which were completely destroyed, threatened, or sustained minor damage by the bushfire that swept through the township of Mount Macedon in Victoria, Australia on the evening of Ash Wednesday (February 16) in 1983.[412,414]
From Alexander ME: Proposed revision of fire danger class criteria for forest and rural areas in New Zealand. Circular 1994/2. Wellington, New Zealand, National Rural Fire Authority, 1994.

assisted with the containment of the fire. Firefighters had the situation under control by the fourth day, but not before they had been given an awful glimpse of the nature of fires in the future. In late October 2003, fires in southern California burned over 300,000 hectares (742,000 acres) in one week alone, taking 26 lives and destroying 3361 homes.[85,199]

In recent times, wildland fire fatalities among the general public have not been restricted to just California, or the United States for that matter. Four civilians were killed as a result of the massive forest conflagration that converged on the outskirts of Canberra, Australia in January 2003.[3] During the summer of 2003 in Portugal, 21 people lost their lives in 18 different fire incidents.[395,398] On January 11, 2005, nine people (including four children and two firefighters) perished on the Eyre Peninsula of South Australia as a result of being overrun by an extremely fast-spreading grass fire in what is now referred to as "Black Tuesday" in the state.[347,353] Even more recently, numerous civilian fatalities due to entrapments and burnovers were reported in Texas and Oklahoma by the media during late 2005 and the early part of 2006.

In many parts of the world, but especially in the United States and Canada, the primary response is to evacuate all people threatened by wildfires.[1,117,317] The pros and cons of evacuation are extensively discussed in Chapter 12 of Webster's seminal work, the *Complete Australian Bushfire Book*,[403] entitled "The Decision—Evacuate or Stay? Safety or Suicide?" In this regard, Phil Cheney,[101] a renowned Australian bushfire research scientist, feels that "What it comes down to is a civil right to risk your own life to save your house. I personally am in favor people taking risks in their lives provided that they also bear the responsibility for the consequences."

Some amazing logistical feats have taken place to date without incident using both ground and air transportation. A good example of the latter occurred in early June 1995 as the residents of Fort Norman and Norman Wells in Canada's Northwest Territories were airlifted to Yellowknife, the territorial capital.[134] However, statements later on from the public like "I think they took us away just in time" suggest that this approach is not infinitely infallible. Coupled with this reality is the fact that modern fire records indicate that the vast majority of area burned by wildfires is the result of a small percentage of fires.[363] Thus, on some days, adverse fuel and weather conditions, coupled with an ignition source, will conspire to produce conflagrations that occur in spite of our best fire prevention, detection, and initial attack efforts. Climate change will only exacerbate the situation.[157] Communities need to ensure that they are fully prepared well in advance of a threatening wildfire occurrence by developing individual and local government emergency action plans.[296] Taylor[371] has emphasized that managing the fire risk at the wildland–urban interface is a shared responsibility involving both private citizens and numerous government agencies.

Fire experiences in Australia[292,403] have demonstrated repeatedly that "houses protect people and people protect houses." Research has demonstrated that a well-prepared house can provide protection from fire and that the presence of people prepared to defend against their home burning down is the most significant factor in determining its survival (Table 12-1).

Obviously, zones of defendable space around homes must be established in advance of fires; the young, elderly, and infirm generally are evacuated well ahead of the fire. Communities at risk from wildfires should be encouraged to be responsible for their own safety because fire service personnel may not be available when burning conditions are severe. The major issue is whether able-bodied residents should stay and defend or "fight" for their homes, or "flee" in the hopes of escaping an advancing fire.[413]

The Tasmania Fire Service had a policy that provided guidance on bushfire safety and evacuation decision making called "Prepare, Stay, and Survive."[169] When there was a threatening wildfire, people were told to go home and assist in the protection of their property. Because human lives and property values are at risk when threatened by wildfires, exemplary cooperation and teamwork are required to ensure adequate safety margins. Team members identified for reducing the loss of life and property include state agencies, local government, the communities, and individuals.

The "Stay or Go" policy concerning evacuation now advocated by the Australasian Fire Authorities Council[38] is regarded as a fundamental component of community bushfire safety in Australia.[184] Variants to this general theme exist (e.g., "stay and defend or go early"). However, the essence of the approach urges people to make the decision to prepare themselves and their properties to "stay" and defend when a wildfire is likely, or to "go" well before a fire is likely to arrive. Although early evacuation may contribute to increased personal safety, bushfire property losses are likely to increase. Conversely, late evacuation may put people at greater risk than had they stayed in the house as the fire passed around them. An evaluation of this policy is the focus of Project C6.1—Evaluation of Stay or Go Policy currently being undertaken by the Australian Bushfire Cooperative Research Centre (www.bushfirecrc.com). Fire management agencies and the public alike will no doubt find the final results of this research project of great interest. Preliminary results emerging from the project strongly support the Australian position on the grounds of both improved safety and reduced property loss. Handmer and Tibbits[183] state, "The evidence also shows that the most dangerous option—and the cause of most fatalities—is the last minute evacuation." This subject is considered worthy of more open debate and discussion in North America.[18]

In many regions of the world, people are warned that wildfires are dangerous and that they always need to evacuate. This scare tactic approach reinforces the concepts that wildland fires are always dangerous and that people must leave their homes. Perhaps one solution to dealing with fires in the wildland–urban interface in the future would be to embrace the concept of "A Dream, a Team, and a Theme."[260] The "dream" would be one where houses are able to survive fires even when fire services personnel are not available. The "team" would consist of the effective partnership between the fire services and home dwellers. The "theme" would comprise the dual strategy of adequate defendable space coupled with the home dweller's motivation to remain on site as an important factor in suppressing fires initiated by ember attack. In order for this approach to work, rural homeowners must understand that although wildfires can be dangerous, with proper precautions, people can remain with their home and be an important part of the solution to the wildland–urban interface fire problem. Empowering communities at risk from wildfire to play an active part in their own protection is viewed as a viable long-term strategy to enable safe and harmonious coexistence with fire as an element of nature.[99]

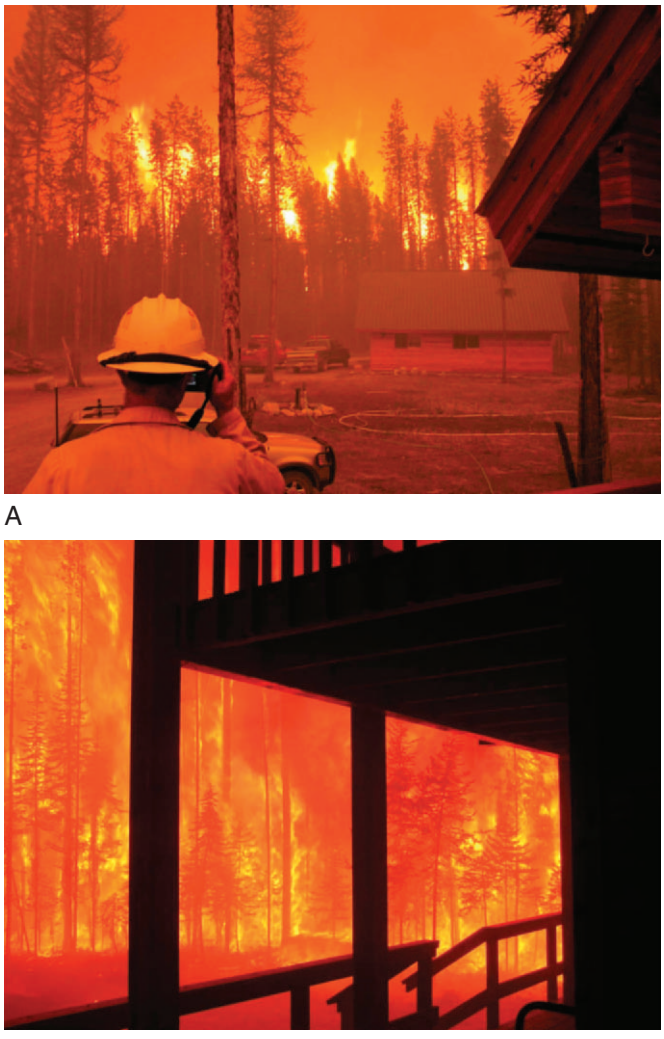


Figure 12-9. Homeowners and firefighters **(A)** successfully prepared, stayed, and defended this rural home in western Montana from a high-intensity flame front **(B)** associated with the Wedge fire in July 2003. (Courtesy Big Fork Volunteer Fire Department.)

During the 2000 fire season in western Montana, some individuals took it upon themselves to stay with their homes as flame fronts advanced. They created defensible space, installed sprinkler systems, engaged in fire suppression activities, and provided local intelligence to incoming fire service personnel. No home was lost as people demonstrated responsibility for their personal well-being. A similar "prepare, stay, and defend" action was successfully carried out during the 2003 fire season on the Wedge fire along the North Fork of the Flathead River in western Montana. The property was prepared in advance to be "fire safe," the homeowner remained with volunteer fire department officials, and the home survived the passage of a high-intensity crown fire (Fig. 12-9).

A

B

C

Figure 12-10. Old man's gorse *(Ulex europaeus)* is probably the worst fuel type **(A)** in the world from the standpoint of wildland fire management for two main reasons. The density of the vegetation and its sharp spines or thorns **(B)** make it extremely difficult if not nearly impossible to physically engage in any kind of fire suppression activity. Vigorous stands grow outward, crowding out all vegetation and forming a center of fine, dry, dead, elevated fuel. This fuel structure, coupled with the plant's oil content, readily contributes to the development of high-intensity flame fronts even under relatively mild fire weather conditions **(C).** Gorse is native to central and western Europe, where it has long been cultivated for hedgerows. It has been introduced to Australia, New Zealand, the west coast of the United States (California, Oregon, and Washington), and southwestern British Columbia. It has also been reported in Costa Rica and the Hawaiian Islands. In the northeastern United States, it has established itself along the Atlantic Coast from Virginia to Massachusetts. (Courtesy Ensis Bushfire Research Group—New Zealand.)

Wildland fires that threaten human lives and property are not exclusively located in southern California because the exodus to wildland regions has become a national phenomenon. Fires burned about 81,000 hectares (about 200,000 acres) in Maine in October 1947, killing 16 people[82,143]; another area of about 81,000 hectares (about 200,000 acres) burned in New Jersey in April 1963.[41] Wildland fire disasters have not always been large in size. On September 26, 1936, most of the coastal town of Bandon in western Oregon was destroyed, and 11 people were killed by a wildfire that covered probably no more than about 4000 hectares (about 10,000 acres).[191] The prevalence of gorse (Fig. 12-10), a non-native plant, was considered a key contributing factor to the resulting devastation. More recently, on July 16, 1977, the Pattee Canyon fire in Missoula, Montana, destroyed 6 homes and charred about 500 hectares (about 1200 acres) of forests and grasslands in only a few hours.[156] Similarly, more than 70 homes were damaged or destroyed by fires in the southern Cape Peninsula region of South Africa that burned about 8000 hectares (about 19,800 acres), during January 16 to 20, 2001.[87,393] A whole host of other cases have occurred in North American[190] and are steadily accumulating.

Fires at the wildland–urban interface are also increasing internationally. For example, the Ash Wednesday fire disaster in southeastern Australia on February 16, 1983 burned about 340,000 hectares (about 840,000 acres) of urban, forested, and pastoral lands in the states of Victoria and South Australia, killing 77 people, injuring some 3500, and destroying more than 2500 homes.[46,186,286] Phenomenal fire spread rates in both forests and grasslands occurred,[200,320] and flame heights of almost 200 meters (almost 650 feet) were observed.[370] In May 1987, wildfires in northeastern China[333] added a new perspective regarding the devastating impact wildland fires may have on human lives, property, and natural resources. These fires reportedly burned about 1.34 million hectares (about 3.31 million acres), killed 212 people, seriously injured another 226, and left about 56,000 homeless.[139] Clearly, it was a disaster of major proportions. These fires resulted from a combination of heavy dry fuels, sustained drought, and strong winds.[362] Rates of spread during the major runs reached 20 km/hr (12 mph). Protecting lives and property from wildfires at the wildland–urban interface represents one of the greatest challenges faced by wildfire protection agencies.

Large forest fires during the intense El Niño drought conditions of 1997 and 1998 focused public and media attention once again on the need to evaluate public policies and practices in the forestry and nonforestry sectors that directly or indirectly contribute to the impact of wildland fires. The size and damage attributed to these fires were so immense that the *Christian Science Monitor* termed 1998 "The Year the Earth Caught Fire." At times, that seemed to be literally true, as smoke palls blanketed large regional areas, disrupted air and sea navigation, and caused serious public health threats.[170,316,338,419] At least 60 people were killed in Mexico as a result of the fires,[381] and ecosystems that generally are not subjected to fires, such as the Amazon rain forest and the cloud forest of Chiapas, Mexico, sustained considerable damage. A global fire conference sponsored by the Food and Agricultural Organization of the United Nations in October 1998 brought together specialists from 33 countries to review the serious nature of worldwide fires.[159] Participants at the conference in Rome concluded that governments needed to enact more sustainable land use policies and practices to reduce the impacts of wildfires on people and natural resources.

It is becoming increasingly rare to have a wildland fire situation or incident that does not involve people and their homes. However, people are not fully aware of the fire risks and hazards of living and traveling in or near wildlands.[51,177] Risk, in the jargon of the wildland fire specialist, is the probability that a fire will occur. Hazard is the likelihood that a fire, once started, will cause unwanted results. Risk deals with causative agents or ignition sources; hazard deals with the fuel complex.[162] The results of two surveys carried out in the western United States during the 1970s indicated a general feeling of overconfidence by most residents toward the potential danger or threat of a wildland fire. Eighty percent of Seeley Lake, Montana forest residents interviewed thought that the forest fire hazard was low to moderate in their area.[161] Seventy-five percent of Colorado residents interviewed thought that the forest fire hazard was low or moderate in mountain subdivisions of their state.[192] Forest fire hazards in these two areas were much higher than these public estimates. In a survey of attitudes toward bushfires in the Dandenong Ranges of Victoria, Australia, it was found that people who had experienced bushfires tended to rank their area with a higher rating than did people without such experience.[146] However, the survey also revealed that 40% of the people with recent severe bushfire experiences still did not rate bushfires as one of the three most important environmental problems in their area. In more recent surveys carried out in Florida and Minnesota[282] of homeowners' preferences for vegetation and defensible space near their homes, people interviewed recognized the wildfire threat but varied in their perceptions of effective wildfire prevention measures and willingness to take actions to reduce the potential threat to themselves and their homes. Most supported the use of planned fire to reduce fire potential, especially if wildland fire experts who understand the local ecology and fire behavior conducted the prescribed burns. In-depth interviews conducted with homeowners along the northern part of the Colorado Front Range revealed that these people face difficult decisions regarding implementation of wildfire mitigation measures.[63] Perceptions of wildfire mitigation options were found to be possibly as important as perceptions of risk in determining likelihood of implementation. These mitigation options were often viewed as tradeoffs between wildfire risk and preferred landscapes. The study participants also reported, however, that one-on-one information sharing with wildland fire experts, as well as increased understanding of the flexibility of mitigation options, encouraged implementation. Personalized contact appears to be a key factor in educating homeowners about the realities of living in a wildland environment.[235]

Participants in a survey conducted in Michigan[418] considered wildland fires as inherently uncontrollable and the resulting damage essentially random. Thus, they only weakly supported investments in fire suppression infrastructure but strongly supported fire prevention programs that reduced the number of fire starts, were unlikely to take all possible steps to safeguard their own properties, and exhibited a negative view of prescribed fire to the extent that it could preclude its use as a risk management tool.

There is a growing need for the general public, emergency medical personnel, and fire suppression organizations to be well prepared to deal with wildland fire encounters (Box 12-2). As Reitz and Geissler[322] have recently noted, "The old model of individual homeowners and neighborhoods depending solely on government provided fire fighting resources is gone. Recent wildland fires have demonstrated that community firefighting resources are easily outpaced when multiple structures are burning simultaneously. The cure is to move most structure protection responsibility to the homeowner and community."

In 2000, 450 residents of Strathcona County, Alberta, Canada were randomly selected to test which three educational methods would produce the greatest change in homeowner behavior with respect to the wildland–urban interface.[360] The study showed that residents could be influenced to change their behaviors when provided with the information materials that enabled them to select between fire-safe alternatives.

A recent study evaluated workshops for the adult public featuring experimental learning about wildland fire.[295] Participants used hands-on activities to investigate fire behavior and ecology and to assess hazards in the wildland–urban interface. Effectiveness was examined using a pretest, a post-test following the program, and another post-test 30 days later. Participant's knowledge increased after the workshop, and attitudes and beliefs became more supportive of fire management. The study concluded that hands-on activities can help adults become better informed about wildland fire and more positive about fire management.

Wildland Fire Lessons

Recommendations to reduce the loss of life and property in the wildland–urban interface will be useless unless they are implemented at the grassroots level by all stakeholders. An excellent example of a community-based program is one implemented at Incline Village and Crystal Bay in the Lake Tahoe basin.[348] The objective of this program is to "reduce the potential for natural resource, property, and human life losses due to wildfire by empowering the communities' residents with the knowledge to address the hazard, providing the resources necessary to correct the problem, and encouraging the cooperative efforts of appropriate agencies." The three major components of this defensible-space program include neighborhood leader volunteers, a slash removal project, and agency coordination. The key to protecting life and property in the wildland–urban interface is property owners' realization that they have a serious problem and that their actions embody a significant part of the solution.

Box 12-2. Recommendations to Reduce Loss of Life and Property in the Wildland–Urban Interface

FIRE PROTECTION SERVICES

- Remember that firefighter and public safety is the first priority in every fire management activity.
- Ensure that all personnel receive regular cross-training in fighting wildfires and structural fires.
- In urban departments, in particular, recognize the need to extinguish fires in wildland fuels by using thorough mop-up procedures.
- Recognize the need for close coordination of response efforts among neighboring departments or agencies.
- Develop specific mutual aid plans for coordinating resources to attack fires in the wildland–urban interface.
- Schedule and conduct regular mutual aid training exercises.
- Regularly schedule and conduct fire prevention and fire preparedness education programs for the general public and homeowners.
- Conduct an assessment of fire risks and prepare a strategic plan to reduce these risks.
- Work effectively with lawmakers and other government officials to help prevent unsafe residential and business development.

LEGISLATORS

- Examine existing laws, regulations, and standards of other jurisdictions that are applicable locally in mitigating hazards associated with wildland fires.
- Adopt National Fire Protection Association (NFPA) guidelines in the form of NFPA 1144, (formerly NFPA 299, 1991 edition) *Standard for the Protection of Life and Property from Wildfire*.[265] The purpose of this standard is to provide criteria for fire agencies, land use planners, architects, developers, and local government for fire-safe development in areas that may be threatened by wildfire. NFPA 1144 provides minimum planning, construction, maintenance, education, and management elements for the protection of life, property, and other values that could be threatened by wildland fire. It is designed to assist local, state, and federal fire agencies in dealing with the escalating challenges presented by the proliferation of wildland–urban interface communities and the monetary losses of structures in wildland–urban interface areas. These guidelines address the following topics: assessment and planning; access, ingress, egress, and evacuation; fuel modification area; water supply; and residential development design, location, and construction.
- Provide strong building regulations that restrict untreated wood shingle roofs and other practices known to decrease the fire safety of a structure in the wildlands.

PLANNERS AND DEVELOPERS

- Create a map of potential problem areas based on fuel type and known fire behavior.
- Evaluate all existing or planned housing developments to determine relative wildland fire protection ratings and advise property owners of conditions and responsibilities.
- Ensure that all developments have more than one ingress–egress route.
- Offer options for fire-safe buildings.
- Provide appropriate firebreaks, fuel breaks, or green belts in developments.
- Ensure that adequate water supplies exist in developments.
- Follow specifications in NFPA 1144 *Standard for the Protection of Life and Property from Wildfire*.[265]

PUBLIC AND HOMEOWNERS

- Determine the wildfire hazard potential of the immediate area before buying or moving into any home.
- Contact federal, state, and local fire services for educational programs and materials regarding fire protection.
- Provide a fuel-modified area or defensible space around vulnerable structures to reduce the likelihood of ignition by an advancing wildfire.
- Design and build nonflammable homes.
- Urge lawmakers to respond with legislative assistance to require appropriate fire safety measures for communities.

In the Incline Village and Crystal Bay community fire plan, neighborhood leader volunteers are trained in defensible-space techniques and are expected to teach these techniques to their neighbors and to coordinate neighborhood efforts. Such concerted community action will greatly minimize threats from the southern California–type "fires of the future" as discussed earlier.

It is also wise to have sensible land development practices because tragedies arise not only from ignorance of fuels and fire behavior (Fig. 12-11) but also from a greater concern for the aesthetics of a home site than for fire safety.[266] Several aspects of development detract from fire safety in the wildland–urban interface[118,156]:

1. Lack of access to adequate water sources.
2. Firewood stacked next to houses.
3. Slash (i.e., branches, stumps, logs, and other vegetative residues) piled on home sites or along access roads.
4. Structures built on slopes with unenclosed stilt foundations.
5. Trees and shrubs growing next to structures, under eaves, and among stilt foundations.
6. Roads that are steep, narrow, winding, unmapped, unsigned, unnamed, and bordered by slash or dense vegetation that makes them extremely difficult if not impossible for fire appliances to negotiate.
7. Subdivisions on sites without two or more access roads for simultaneous ingress and egress.
8. Roads and bridges without the grade, design, and width to permit simultaneous evacuation by residents and access by firefighters and emergency medical personnel and their equipment.
9. Excessive slopes, continuous or heavy fuel situations, structures built in box canyons, and other hazardous situations.
10. Lack of constructed firebreaks and fuel breaks around home sites and in subdivisions.
11. Living fuels that have not been modified by pruning, thinning, landscaping, or other methods to reduce cured

Figure 12-11. This permanent residence, located in west-central Alberta, Canada, is unlikely to survive exposure to even a low-intensity wildland fire, and it would be difficult to stay and defend it for a number of reasons. There is dense forest vegetation immediately adjacent to the structure. Cedar shakes have been used for roofing. This has been shown time and time again to be the "kiss of death" in wildland–urban interface fires (at the time this photo was taken in 1997, the shingles were in such bad shape that they had lichens growing on them). The homeowner also liked to store flammable materials under his deck. (Courtesy Yukon Wildland Fire Management. Photo by A. K. Beaver.)

vegetation and litter that readily contribute to spot fire development and fire intensity.

12. Homes constructed with flammable building materials (e.g., wooden shake shingles).
13. Propane tanks exposed to the external environment.
14. Inability to deliver water effectively before and during passage of a fire front around the home property (e.g., lack of proper equipment, such as sprinklers).

In a lot of cases, simply cutting the grass and keeping the yard cleaned up will dramatically increase the chances of the house surviving a wildfire.[107]

What Some Organizations Have to Offer

The National Disaster Education Coalition is a group of U.S. federal agencies and nonprofit organizations that support common goals in disaster and hazards education. This group worked hard with the nation's leading social scientists in this area to produce a state-of-the-art guide for public risk education. The group developed a standardized guide on hazard safety messages, providing information that all organizations agreed on for national use. The guide covers many different hazards as well as general preparedness issues. It is in the public domain and can be used by anyone. It is available from any local Red Cross chapter or can be downloaded from the national Red Cross website (www.redcross.org).

Firewise Communities is a national mitigation planning program that encourages communities to include land use planning, building codes, landscaping codes, zoning, and fire protection in developing new communities and in retrofitting existing communities.[40,303] *Firewise Communities* is a national initiative designed to reach beyond the fire service and involve homeowners, community leaders, planners, developers, and others in the effort to protect people and property from the dangers of wildland fire. *Firewise Communities* programs include the *Firewise Communities* Workshop series, *Firewise Communities*/USA Recognition Program, and support for fire organizations and community groups. The *Firewise Communities* program is part of the Wildland–Urban Interface Working Team of the National Wildfire Coordination Group (NWCG), a consortium of wildland fire agencies that includes the USDA Forest Service, Department of Interior, National Association of State Foresters, U.S. Fire Administration, Federal Emergency Management Agency, and National Fire Protection Association. *Firewise Communities* emphasizes that everyone in every community is responsible for fire protection. *Firewise Communities* workshops help define responsibilities for a network that includes many partners. The intent is to undertake planning and actions to ensure that people live more compatibly in every neighborhood situation in a wildland fire environment. Towards this end, a website has been developed (www.firewise.org), and several publications and books[266,345] have been published to assist homeowners living in the wildland–urban interface.

A team of scientists from across the United States has been visiting communities to identify the activities they need to undertake in order to increase their wildfire preparedness and the resources necessary to support these activities. This research is funded by the National Fire Plan and is led by the Social and Economic Dimensions of Ecosystem Management Research Work Unit of the USDA Forest Service's North Central Research Center. Research partners include USDA Forest Service's Pacific Northwest Research Station, University of Minnesota, Southern Oregon University, and University of Florida. Sixteen community preparedness case studies were completed between 2002 and 2004 in a variety of locations throughout the continental United States (www.ncrs.fs.fed.us).

Partners in Protection, based out of Alberta, Canada, is a coalition of professionals representing national, provincial, and municipal associations and organizations committed to raising awareness, providing information, and developing forums with a view to encouraging proactive, community-based initiatives with respect to fires at the wildland–urban interface. Partners in Protection has produced a comprehensive manual with respect to the wildland–urban interface fire problem entitled "FireSmart: Protecting Your Community From Wildfire."[296] This well-illustrated guide focuses on how individuals and communities can work together to reduce the risk for loss from fires in the wildland–urban interface. It provides practical tools and information for use by residents and by individuals and organizations that operate in the wildland–urban interface. The primary topics are description of interface issues, evaluation of hazards, mitigation strategies and techniques, emergency response for agencies and individuals, training for interface firefighters, community education programs, and regional planning solutions. The manual, available in both book form and CD-ROM, can be viewed online at the *Partners in Protection* website (http://partnersinprotection.ab.ca), and covers nearly every facet of fire preparations in the wildland–urban interface. In the chapter on communications and public education, for example, several principles are cited for effective communication:

- Begin with clear, explicit objectives.
- Don't make assumptions about what people know, think, or want done. Take time to find out what people are thinking by using surveys, focus groups, or other research.
- Involve all parties that have an interest in the issue. Identify and address the particular interests of different groups.

- Identify with your audience. Put yourself in their place and recognize their emotions.
- Take time to coordinate with other organizations or groups.
- Choose your spokespeople carefully and ensure they have the training to communicate your messages effectively.
- Practice and test your messages.
- Do not either minimize or exaggerate the level of risk.
- Promise only what you can do. Do what you promise.
- Plan carefully and evaluate your efforts.

Kumagai and colleagues[207] also offer some excellent advice with respect to wildland fire education and communication with the general public.

Nowadays most local, state, provincial, territorial, and federal fire management agencies have some sort of outreach program related to the wildland–urban interface. For example, the state of Alaska offers teachers both face-to-face wildland fire workshops and an online training course.[366] Beginning in 1993, the Country Fire Authority of Victoria, Australia, developed a program called "Community Fireguard," which assists communities in developing wildfire survival strategies.[58]

FIRE BEHAVIOR

Urban and Wildland Fire Threats

Safety precautions for wildland firefighting crews are continually upgraded as new knowledge is gained about fire behavior and human behavior. The sites where people were injured or killed by fire are routinely examined[230,254,411] afterward to assess fuel conditions, terrain features, probable wind movements at the time of the fire, and actions of firefighters (Fig. 12-12). In this regard, author Norman Maclean[226] has noted:

> *It is hard to know what to do with all the detail that rises out of fire. It rises out of a fire as thick as smoke and threatens to blot out everything. Some of it is true but doesn't make any difference. Some of it is just plain wrong. And some doesn't even exist, except in your mind, as you slowly discover long afterwards. Some of it, though, is true—and makes all the difference.*

The information gleamed from past wildland firefighter fatalities, as well as data about hazards in the wildland–urban interface, is now included in training programs and safety briefings.

In reviewing many past wildland fire tragedies, a sobering observation is that crew members are almost always experienced and well-equipped firefighters, trained to anticipate "blowup" fire conditions (i.e., a temporary escalation in fire behavior). However, when visibility is lowered to 6 m (20 ft), noise levels preclude voice communication, eyes fill with tears, and wind blows debris in all directions, a person's judgment is badly impaired. For many members of the general public, the first wildland fire they may experience will in all likelihood be a high-intensity conflagration. As Cheney[100] notes:

> *The approach of a major fire can appear ominously threatening to those directly in its path, but is often viewed with curiosity and with little concern if it appears that the wind will direct the fire past their properties. Thus, when a combination of circumstances, often associated with a frontal wind passage, exposes residents to a high-intensity fire environment, they are very much unprepared. Their senses are assaulted by sudden darkness, thick blinding and choking smoke, buffeting winds and unexpected calm periods, searing heat and flames which illuminate the smoke-obscured scene and a roaring noise of wind and explosive combustion. In all this activity they observe flames which change from a metre or two to engulfing whole trees. In such a dramatic and emotional situation, many people are apt to invent phenomena (such as, houses exploding, the air burning, fantastic spread rates and the like) to explain the extent of the devastation that confronts them when the smoke clears away.*

Figure 12-12. In an attempt to avoid the intense heat of this brush fire in southwestern Colorado, four firefighters took refuge in the fire line, in the foreground at point A. Affected by intense convective and radiant heat and dense smoke, one individual ran into the fire and died at point B. Another individual ran about 300 meters (about 1000 feet) down the ridge, where his body was found at point C. The third fatality was a person who remained at point A; he died a short time after this position was overrun by fire. The only survivor also remained in a prone position at point A with his face pressed to the ground. At one point he reached back and threw dirt on his burning pants legs. The survivor sustained severe burns to the back of his legs, buttocks, and arms. The deaths of the other three individuals were attributed to asphyxiation. (Courtesy USDA Forest Service.)

Even for experienced firefighters, previous training can give way to panic-like attacks and poor decision making, leading to actions that can result in serious injury or death.[289]

This scenario is most evident in urban fires; the pattern of hysteria affecting people trapped in burning buildings is all too familiar to urban firefighters.[351] The way fire kills in the urban setting can be compared with wildland fires, as shown here[291]:

- Heat rises rapidly to upper stories when a fire starts in the basement or on the ground floor. Toxic gases and smoke rise to the ceiling and work their way down to the victim—a vital lesson for families planning protective measures. Smoke poses the double problem of obscuring exit routes and contributing to pulmonary injury and oxygen deprivation.
- As the fire consumes oxygen, the ambient oxygen content drops, impairing neuromuscular activity. When the oxygen content drops below 16%, death by asphyxiation will ensue unless the victim is promptly evacuated. Asphyxiation, not fire itself, is the leading cause of fire deaths.
- Ambient temperatures may rise extremely rapidly from even small fires. Temperatures of 150° C (300° F) will cause

rapid loss of consciousness and, along with toxic gases, will severely damage lung tissues. Warning devices may offer the only possibility for survival owing to the rapid onset of debilitating symptoms.

Obvious similarities and differences are seen between wildland fires and urban fires:

1. Smoke, heat, and gases are not as concentrated in wildland situations as in the confined quarters of urban fires.
2. Flames are not a leading killer in either the urban or wildland situation.
3. Although oxygen levels may be reduced near wildland fires, there is usually sufficient replenishment of oxygen in the outdoor environment to minimize deprivation. Asphyxiation, however, can also be an important cause of death in wildland fires.
4. Inhalation of superheated gases poses as serious a threat to life in wildland fires as in urban fires.
5. Wildland smoke does not contain toxic compounds produced by combustion of plastics and other household materials, but it does impair visibility, contain carbon monoxide, and carry suspended particulates that cause severe physical irritation of the lungs.
6. Sprinkler systems and automatic early-warning devices to detect smoke or heat may protect people from serious injury or death in the urban environment, but in the wildland environment, people must rely on their experience, senses, knowledge, and skills or ingenuity to provide early warning of an impending threat to life.

The general public needs to appreciate that fires occurring under certain fuel, weather, and topographic situations may result in explosive fire growth on an area basis (Fig. 12-13). The fire that burned through Faro, Yukon, on June 13, 1969 is a good example of this type of behavior.[201] The Faro fire, which burned about 15,500 hectares (about 38,300 acres) resulted from a lightning strike that occurred 8 km (5 miles) west of town at 5:04 PM. The fire started to spread fairly quickly in an easterly direction. Fire crews were dispatched from town by vehicle to take suppression action, but little could be done because of crowning activity. By 8:30 PM, the fire had burned through town and was continuing to spread further east. In other words, the fire had traveled 8 km (5 miles) in less than 3.5 hours. This equates to a rate of spread of around 2.3 km/hr (1.4 mph) or 40 m/min (130 ft/min) or 2.3 km/hr (1.4 mph). This would not be considered an unusual situation in many regions of the North American boreal forest.[23,131] How wide would the fire have likely been when it hit the edge of town? A rough rule of thumb is to divide the separation distance by a factor of 2 to 4 based on an assumed wind-driven fire shape.[374] The fire would have thus been roughly 2 to 4 km (1.2 to 2.5 miles) wide when it hit the western edge of Faro.

Figure 12-13. View of the Crutwell fire as it burned toward the city of Prince Albert, Saskatchewan during its initial major run that started during the late afternoon of June 28, 2002. This photo taken from a building in the downtown area when the fire was about 18 kilometers (about 11 miles) away. The fire was started by lightning during the morning of a day of extreme fire danger. Upon escaping initial containment, this wind-driven fire advanced by crowning through jack pine forests at rates of about 2.3 km/hr (1.4 mph) at the height of activity. Spotting up to 1 km (0.6 miles) was observed. Fortunately, changes in fuel types and less severe fire weather conditions during the night contributed to the effectiveness of the fire suppression operations. (Courtesy Saskatchewan Health. Photo by G. Matchett.)

Fire Behavior Knowledge: A Wildland Fire Early-Warning System

To the lay person, a wildland fire can be a perplexing event. As Countryman and Schroeder note[130]:

> *A casual observer seeing a forest fire for the first time would probably decide there is little rhyme or reason to the way a fire burns. Its sudden variations in direction of travel, its quick change from a seemingly mild, slow-burning fire to a raging inferno, all seem to add up to erratic behavior without local explanation. Actually a fire behaves in accord with changes in its surroundings—the character and condition of the burnable vegetation, the topography, and the meteorological factors—that affect fuel flammability, fire intensity, and fire spread. This complex of environmental factors that control the behavior of fire is what we call "fire environment."*

Understanding one's fire environment[125] is considered a key factor for safe and effective action in dealing with wildland fires. For example, 4 of the 32 civilians killed in Victoria during the 1983 Ash Wednesday fires in southeastern Australia were caught outside their homes tending to or attempting to herd up livestock,[205] apparently unaware of the change in wind direction and speed that accompanied passage of the cold front through their area.[320] In such situations in southeastern Australia, the strong prefrontal winds are from the northwest. With the passage of the front, the wind direction changes sharply to the southwest, and winds increase markedly in strength. Had these people understood or been aware of the implications of the cold front passage on fire behavior and received some information on the timing of the change, they may have chosen to stay inside their homes.[205]

In the conclusions to their detailed case study report on the 1994 South Canyon fire, USDA Forest Service fire researchers[78] noted:

> *None of the findings and observations discussed in this study represent new breakthroughs in wildland fire behavior understanding. Rather, the findings support the need for increased understanding of the relations between the fire environment and fire behavior. We can also conclude that fire managers must continue to monitor*

and assess both present fire behavior and potential future fire behavior given the possible range of environmental factors.

An excellent overview of the fire environment concept has been prepared by the National Wildfire Coordinating Group.[271]

Fire behavior is defined as the manner in which fuel ignites, flame develops, and fire spreads and exhibits other related phenomena as determined by the interaction of fuels, weather, and topography. The science of fire behavior describes and predicts the performance of wildland fires in terms of ignition probabilities, rates of spread, intensity levels, spotting distances and densities, and crowning potentials. Experienced firefighters routinely monitor the fire and the fire environment and assess the probable behavior of fires using fire behavior guidelines and experienced judgment based[28,43] on current and expected fire weather conditions in relation to local fuel type information, topographic conditions, and moisture levels.[71,215,374] This is also referred to as "situational awareness" (i.e., understanding what the fire is doing and what you are doing in relation to the fire and being able to predict where the fire and you will be in the future).[304]

The emergency medical person, backcountry recreationist, and wildland homeowner must also understand basic fire behavior principles to provide for adequate personal safety. A cardinal rule is to base all actions on current and expected fire behavior.[232,247] Attention to simple principles, indicators, and rules should enable wildland users to anticipate and avoid wildfire threats.

Heat, oxygen, and fuel are required in proper combination before ignition and combustion will occur[43,165] (Fig. 12-14). If any one of the three is absent, or if the three elements are out of balance, there will be no fire (Box 12-3). Fire control actions are directed at disrupting one or more elements of this basic fire triangle.[15]

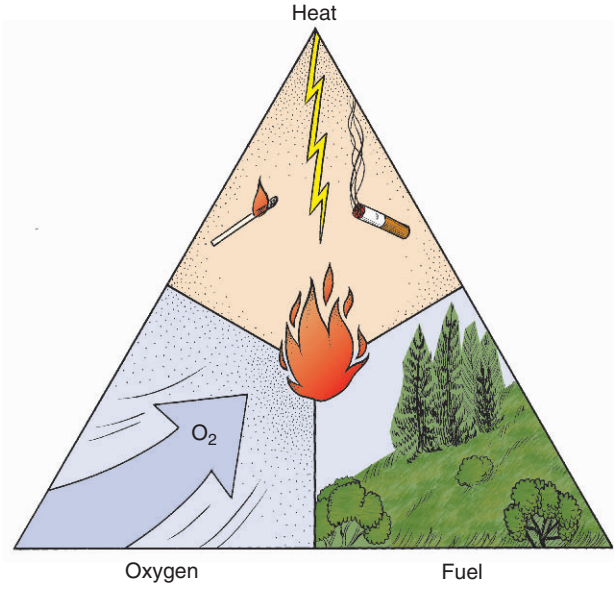


Figure 12-14. Combustion is a process involving the combination of heat, oxygen, and fuel. An understanding of the variation of these three factors is fundamental to an understanding of wildland fire behavior. (Adapted from Barrows JS: Fire Behavior in Northern Rocky Mountain Forests, Station Paper No. 29. Missoula, MT, 1951, USDA Forest Service, Northern Rocky Mountain Forest and Range Experiment Station, 1951.)

Box 12-3. Requirements for Wildland Fire Spread

Certain universal principles apply to all spreading fires in vegetation, living or dead. These really have nothing to do with biology but are based purely on the physics and chemistry of combustion:

1. There must be sufficient fuel of appropriate size and arrangement in space.
2. This fuel must be of sufficient dryness to support a spreading combustion reaction.
3. There must be an agent of ignition.

In practical terms, the primary requirement is a continuous layer of finely divided fuel or minor vegetation on the surface of the ground. This material may be conifer needles, hardwood leaves, grass, lichen, moss, finely divided shrubs, or other minor vegetation. However, it must be present and it must be continuous, or the possibility of spreading fire does not really exist. The second requirement is that the material be dry enough. The maximum moisture content at which fire will spread is hard to specify; for any given fuel or vegetation complex, it will depend on the amount of fuel, its arrangement in space, and the wind speed. Thus, fire spreads poorly or not at all in surface litter of various kinds at moisture contents over 25% or 30% (dry weight basis), whereas fine shrubby fuels or conifer foliage may support fast-spreading fires at moisture contents of 100% or more.

The two limiting criteria common to all spreading fires are as follows:

1. The fire must transfer enough heat forward to dry out the unburned fuel and raise it to ignition temperature by the time the flame front arrives.
2. Enough fuel must pass through the moving flame front to produce a continuous solid flame.

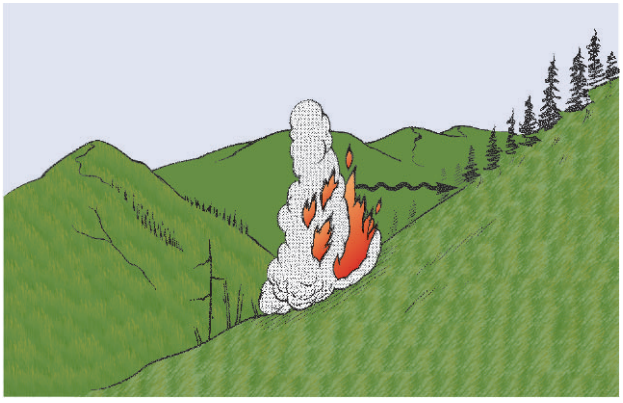
The behavior of any fire is the result of a complex process that results in a dynamic equilibrium among all elements of mass and energy flowing in and out of the flame zone. However difficult this whole process may be to describe and predict, the above two criteria run like threads through the whole range of fires in vegetation, whether the fuel itself is dead or live.

From Van Wagner CE: Fire behaviour in northern conifer forests and shrublands. In Wein RW, MacLean DA (eds): The Role of Fire in Northern Circumpolar Ecosystems, Chichester, England, J. Wiley & Sons, 1983.

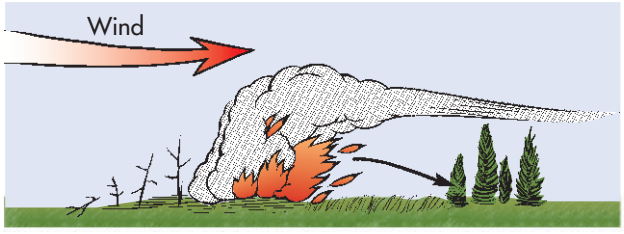
Physical Principles of Heat Transfer

Heat energy is transferred by conduction, convection, radiation, and spotting, but generally only the last three processes are significant in a wildland setting.[84] Although conduction through solid objects is important in the burning of logs, this process does not transfer much heat outward from a flaming front.

Convection, or the movement of hot masses of air, accounts for most of the heat transfer upward from the fire. Convective



A



B

Figure 12-15. Fuels and people upslope **(A)** or downwind **(B)** from a fire receive more radiant and convective heat than on the downslope or upwind sides of a spreading fire. (Adapted from Barrows JS: Fire Behavior in Northern Rocky Mountain Forests, Station Paper No. 29. Missoula, MT, USDA Forest Service, Northern Rocky Mountain Forest and Range Experiment Station, 1951.)

Figure 12-16. Most of the heat felt from a fire is radiant heat, which is largely a function of the height, depth, and angle of the flame front. Wildland firefighters work far enough from the radiating flames to avoid pain to exposed skin (e.g., face and ears). This also limits the amount of radiant heat absorbed through clothing. Wildland firefighters working near an active fire edge with hand tools typically adjust their distance from the flames so that the radiant heat they receive from the fire is little more than that of direct sunlight. (Courtesy British Columbia Forest Service.)

currents usually move vertically unless a wind or slope generates lateral movement (Fig. 12-15). Convection preheats fuels upslope and in shrub and tree canopies, which contributes further to a fire's spread and the onset of crowning forest fires.

Through radiation, heat energy is emitted in direct lines or rays; about 25% of combustion energy is transmitted in this manner. Radiated heat on exposed skin can cause discomfort and severe pain (Fig. 12-16), and even death at elevated levels.[96,122] The amount of radiant heat transferred decreases inversely with the square of the distance from a point source. More radiant heat is emitted from a line of fire than from a point source. Radiant heat travels in straight lines, does not penetrate solid objects, and is easily reflected. It accounts for most of the preheating of surface fuel ahead of the fire front and poses a direct threat to people who are too close to the fire (see Figure 12-15).

Most organized fire crews in the United States carry protective fire shelters[33,34] as part of their personal protective equipment (PPE) (Fig. 12-17). These aluminized "pup tent"–type structures are intended to protect against radiant heat.[81,307] The U.S. Forest Service fire shelter was never designed to mitigate against sustained, direct flame contact.[243] Certain materials that fail when in direct contact with flames, such as "emergency space blankets,"[160] are also not appropriate for use in wildland fires[70] and in fact could cause serious burn injuries. Similarly, "emergency evacuation smoke hoods," intended for use in urban situations or other human-made environments (e.g., airplanes), have absolutely no application in a wildland fire environment despite manufacturers' suggestions to the contrary.

Spotting is a fire spread or mass transport heat transfer mechanism by which wind currents carry flaming firebrands or glowing embers land beyond the main advancing fire front to start new fires (Fig. 12-18). In this manner, fire spread may accelerate very quickly, unexpected new fire starts can occur, and fire intensity and in-draft winds can dramatically increase as spot fires coalesce (Fig. 12-19). High-density, short-range spotting up to 100 m (328 ft) is a common feature of many wildfires, especially when fine fuels are very dry.[31] Low-density, intermediate- to medium-range spotting up to about 1 to 2 km (0.6 to 1.2 miles) is a common occurrence with high-intensity crown fires.[23] Longer-range spotting occurs very infrequently in most fuel types, although spot fire distances of 16 km (10 miles) have been reported in North America.[32] The native eucalyptus forests of Australia have a notorious reputation when it comes to spotting behavior owing to the bark characteristics of many species, including an authenticated spot fire distance of 24 km (15 miles).[221]

Fundamental Wildland Fire Behavior Characteristics

One important aspect of fire behavior that distinguishes urban or structural fires from wildland fires is the latter's horizontal spread potential. Although urban or structural fires may exhibit some vertical fire spread potential, for practical purposes, they are considered stationary fire sources. Wildland fires on the other hand characteristically involve moving flame fronts.

Figure 12-17. The personal protective equipment (PPE) for a firefighter includes hard hat and safety goggles, fire-resistant shirt and trousers, leather boots and gloves, and an aluminized fire shelter carried in a waist pouch **(A).** Firefighters also carry canteens to ensure an adequate water supply in a heat-stressed environment. The fire shelter **(B)** is deployed **(C)** by firefighters as a last resort to provide protection from being exposed directly to radiant heat and superheated air **(D).** (Courtesy USDA Forest Service.)

Whereas ground or subsurface fires may spread very slowly (about 1 m [about 3 ft] per hour),[408] surface fires in open grown forests can reach rates of about 15 to 25 m/min (about 50 to 80 ft/min); conversely, surface fires in closed forest types typically spread at 5 to 6 m/min (16 to 20 ft/min) before the onset of crown combustion. Crowning forest fires generally advance at rates between 30 to 60 m/min (100 to 200 ft/min), occasionally higher.[23,131] Grass fires spread at rates up to around 385 m/min (1263 ft/min) or 23 km/hr (about 14 mph).[106,284] It is thus quite possible for forest fires to advance up to 80 km (50 miles) in a single day[7,330] and for grass fires to travel correspondingly even further.

The threat to life and property largely depends on a fire's residence time (Table 12-2). This represents the length of time any object overrun by a fire will be heated by direct flame contact. Coupled with the fire's rate of spread, the residence time determines the depth of the active or continuous flaming front (Fig. 12-20). The length of time that a fire will continue to burn by smoldering combustion depends on the composition, compaction, moisture content, and quantity of fuel present.

The most basic features of a wildland fire are that (1) it spreads or moves, (2) it consumes or "eats" fuel, and (3) it produces heat energy and light in a visible flaming combustion reaction.[6] Byram[83] defined fire intensity as the rate of heat energy

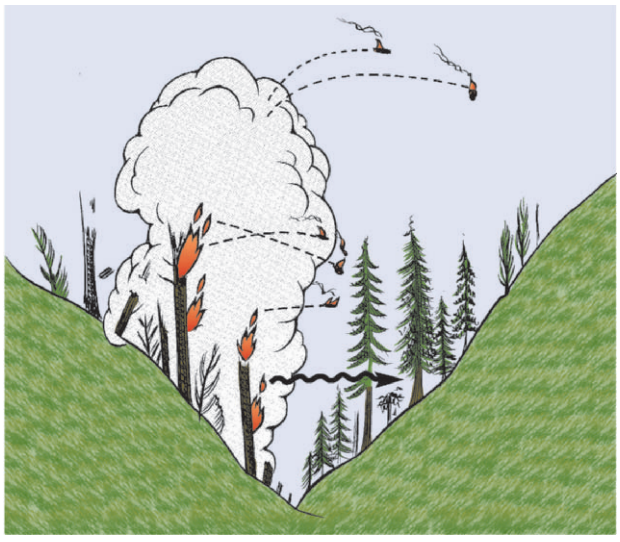


Figure 12-18. Fires can easily cross narrow canyons. Fuels and people on the slope opposite a fire in a narrow canyon are subject to intense radiant heat and spot fires from airborne embers. (Adapted from Barrows JS: Fire Behavior in Northern Rocky Mountain Forests, Station Paper No. 29. Missoula, Mont, USDA Forest Service, Northern Rocky Mountain Forest and Range Experiment Station, 1951.)

Figure 12-19. The occurrence of numerous spot fires is a direct indication that the moisture content of fine fuels has attained a critically dry level and in turn an early warning signal that the potential for extreme fire behavior exists or is imminent. This sequence of photos (**A** to **D**) spanned a period of just over 100 seconds taken late in the afternoon of July 9, 2005 near Coimbra, Portugal. The fuel type is blue gum *(Eucalyptus globulus)*—maritime pine *(Pinus pinaster)* stand. (Courtesy Associação para o Desenvolvimento da Aerodinâmica Industrial. Photos by M. G. Cruz.)

TABLE 12-2. Nominal Residence Times for Four Broad Fuel Complexes and Computed Maximum Theoretical Flame Depths* Associated with Variable Rates of Fire Spread

			RATE OF FIRE SPREAD					
	RESIDENCE TIME		10 m/min (33 ft/min) FLAME DEPTH		20 m/min (66 ft/min) FLAME DEPTH		40 m/min (131 ft/min) FLAME DEPTH	
BROAD FUEL COMPLEX	(sec)	(min)	(m)	(ft)	(m)	(ft)	(m)	(ft)
Grassland	10	0.17	1.7	5.5	3.4	11	6.8	22
Shrub land	20	0.33	3.3	11	6.6	22	13	43
Forest stand	45	0.75	7.5	25	15	49	30	98
Logging slash	90	1.5	15	49	30	98	60	197

*Numerically equal to Residence Time multiplied by the Rate of Fire Spread (in compatible units).

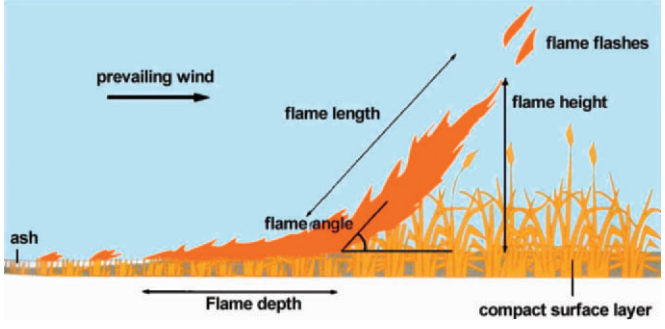


Figure 12-20. Cross-sectional view of a stylized free-burning fire in a grass fuel bed. (From Cheney P, Sullivan A: Grassfires: Fuel, Weather and Fire Behaviour. Collingwood, South Australia, CSIRO Publishing, 1997.)

release per unit time per unit length of fire front, regardless of its depth. Numerically, it is equal to the product of the quantity of fuel consumed in the flaming front, a fire's rate of fire spread, and a fuel heat combustion value. Flame size is its main visual manifestation. The fastest spreading part of a fire is the head; the back of the fire is the slowest spreading part, with the flanks being intermediate between the two (Fig. 12-21). It is for this reason that the fire intensity, flame length, and flame height (see Figure 12-20) are greatest at the front of the fire and least at the rear[91] (Fig. 12-22).

The amount of radiation received from a flame front is determined by the size and geometry (e.g., height, depth, and tilt angle) of the flame front. Radiation levels coupled with residence times determine the nature of burn injuries.[70,144]

Environmental Factors Influencing Wildland Fire Behavior

A wildland fire behaves according to variations in the fire environment (i.e., fuels, weather, and topography). Some of the fire environment factors associated with adverse burning conditions that can signal the potential or onset for severe fire behavior are listed in Box 12-4. These factors or indicators are subject to assessment, measurement, and observation.

When a person encounters a wildland fire, the first step should be to review the principles and indicators of fire behavior, sizing up the situation in terms of fuel, weather, topographic factors, and observed fire behavior. After the fire's probable direction and rate of spread are estimated, travel routes that avoid hazards to human life can be planned (see Figure 12-21). The direction of the main body of smoke is often a good indicator of the direction the fire will take.

Fuel. Because wildland fuels vary so widely in their distribution, their physical characteristics or properties (i.e., moisture content, size and shape, compactness or arrangement, load, horizontal and vertical continuity, chemical content), and their effect on fire behavior, some means of classification is needed for their systematic assessment. Fuels are commonly classified into four groups or strata (Fig. 12-23). Some fuel types may exhibit only one or two of the strata (e.g., grasslands, shrub lands, and logging slash lack the ladder and crown fuel stratum).

An increase in fuel available for combustion affects fire intensity. In other words, the more fuel that is burning, the hotter the fire will be. As Brown and Davis[64] note:

> *The ignition, buildup, and behavior of fire depend on fuels more than any other single factor. It is the fuel that burns, that generates the energy with which the fire fighter must cope, and that largely determines the rate and level of intensity of that energy. Other factors that are important to fire behavior (that is, moisture, wind, etc.) must always be considered in relation to fuels. In short, no fuel, no fire!*

Certain types of fuel, such as chaparral, pine, and eucalyptus, burn more intensely because their foliage contains flammable oils and resins (see Figure 12-10). The size and arrangement of fuel also influence fire behavior. Small, loosely compacted fuel beds, such as dead grass, long pine needles, and shrubs, burn more rapidly than does tightly compacted fuel. Large fuels burn best when they are arranged so that they are closely spaced, such as logs in a fireplace. Scattered logs with no small or intermediate fuel nearby seldom burn unless they are decomposed. Seasonal changes in the moisture content and live-to-dead ratio (e.g., degree of curing in grasslands) of conifer tree foliage,

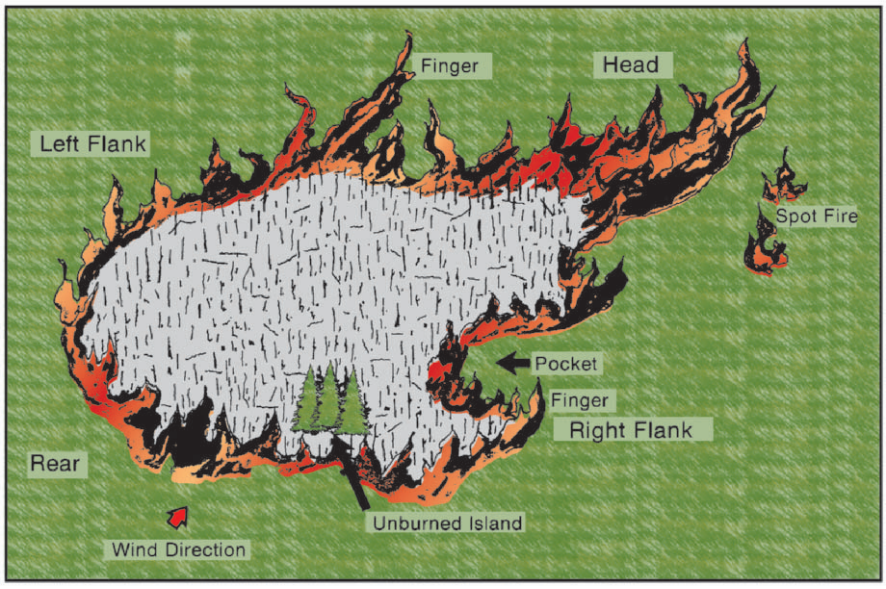


Figure 12-21. The parts of a fire are described in terms of its left flank, right flank, head, and back or rear. There may also be unburned islands within the fire and spot fires ahead of the fire. The safest travel routes generally involve lateral movement on contours away from the fire's flank or movement toward the rear of the fire. Moving in front of a head fire should be avoided. The burned area inside the fire's perimeter can offer a safe haven provided smoldering or glowing combustion levels are low and the flaming perimeter can be safely penetrated by an individual; falling trees or snags and rolling rocks could, however, still pose a hazard. (Adapted from Moberly HE, et al: Planning for Initial Attack, revised ed. Forestry Report SA-FR-2. Atlanta, Ga, USDA Forest Service, Southeastern Area State and Private Forestry, 1979.)

Figure 12-22. Elliptical fires resulting from multiple, wind-driven, point source ignitions associated with a prescribed burning operation in a southeastern U.S. pine plantation. As indicated by the smoke drift, the winds are blowing roughly from lower right to the upper left. (Courtesy USDA Forest Service. Photo by C.W. Adkins.)

shrubs, grasses, and other herbaceous plants can result in gradual changes in fuel flammability over the course of a fire season.[127,221]

Wildland fire research has identified a wind speed threshold in fuel types with a discontinuous, combustible surface layer (e.g., due to the presence of areas of bare, mineral soil).[68,72] In these discontinuous fuel types, fire spread is exceedingly limited until a certain or threshold wind speed level is attained, and then the rate of advance can be quite unexpectedly rapid[163] (Fig. 12-24). This kind of situation could lead a person into a false sense of security regarding fire potential, until unfortunately it's often too late.

Various weather phenomena (e.g., wind, hail, snow, ice, and frost) and insect and disease epidemics can drastically elevate the flammability of a fuel complex, creating decidedly new fire safety concerns.[27] For example, on July 9, 1999, heavy rains and winds in excess of 145 km/hr (90 mph) blew down about 181,000 hectares (about 447,000 acres) of sub-boreal forest in northeastern Minnesota, resulting in heavy dead and downed woody fuel loads.[164]

Weather. The greater the wind, the more rapid the spread of fire. Thunderstorm downdrafts and winds associated with dry cold frontal passages can be especially hazardous.[106,335] Low relative humidity and high air temperatures decrease the moisture content of fine, dead fuels and thereby increase ignition ease and rate of spread. Prolonged drought makes more medium and heavy fuels available for combustion, leading to increased intensity levels. When fuels reach critically dry levels, fires become very responsive to minor changes in wind and slope. Fires tend to burn more vigorously under unstable atmospheric conditions.

The North American continent has been classified into 15 fire climate regions based on geographic and climatic factors[335] (Fig. 12-25). Major fire seasons, or periods of peak fire activity, can be used to warn emergency medical personnel and wildland users of the most probable times for life-threatening situations. Exceptions occur. For example, rapidly spreading grass and forest fires can occur during the winter months in the northern latitudes, such as happened in southwestern and west-central Alberta on December 14, 1997 under Chinook conditions and no snow cover.[176] One of the grass fires advanced 33 km (20.5 miles) in less than 4 hours for a spread rate of around 8 km/hr (about 5 mph), seriously threatening the community of Granum.[57] A forest fire near the town of Hinton in west-central Alberta on the same day posed a similar threat.

Although the fire season for the southern Pacific coast is shown in Figure 12-25 as June through September, critical fire

Box 12-4. Early Warning Signals or Indicators Associated with Extreme Fire Behavior

FUEL
- Continuous fine fuels, especially fully cured (dead) grasses
- Large quantities of medium and heavy fuels (e.g., deep duff layers, dead-down logs)
- Abundance of bridge or ladder fuels in forest stands (e.g., branches, lichens, suspending needles, flaky or shaggy bark, small conifer trees, tall shrubs, etc., extending from ground surface upward)
- Tight tree crown spacing in conifer forests
- Presence of numerous snags
- Significant amounts of dead material in elevated, shrubland fuel complexes
- Seasonal changes in vegetation (e.g., frost kill)
- Fire, meteorologic or insect and disease impacts (e.g., preheated canopy or crown scorch, snow-, wind-, or ice-damaged stands, drought-stressed vegetation, or mountain pine-beetle–killed stands)

WEATHER
- Extended dry spell
- Drought conditions
- High air temperatures
- Low relative humidity
- Moderately strong, sustained winds
- Unstable atmosphere (visual indicators include gusty winds, dust devils, good visibility, and smoke rising straight up)
- Towering cumulus clouds
- High, fast-moving clouds
- Battling or shifting winds
- Sudden calm
- Virga (i.e., a veil of rain beneath a cloud that does not reach the ground)

TOPOGRAPHY
- Steep slopes
- South- and southwest-facing slopes in northern hemisphere
- North- and northeast-facing slopes in southern hemisphere
- Gaps or saddles
- Chutes, chimneys, and narrow or box canyons

FIRE BEHAVIOR
- Many fires that start simultaneously
- Fire that smolders over a large area
- Rolling and burning pine cones, agaves (a desert plant found in the southwestern United States), logs, hot rocks, and other debris igniting fuel downslope
- Frequent spot fires developing and coalescing
- Spot fires occurring out ahead of the main fire early on
- Individual trees readily candling or torching out
- Fire whirls that cause spot fires and contribute to erratic burning
- Vigorous surface burning with flame lengths starting to exceed 1 to 2 m (3 to 6 ft)
- Sizeable areas of trees or shrubs that begin to readily burn as a "wall of flame"
- Black or dark, massive smoke columns with rolling, boiling vertical development
- Lateral movement of fire near the base of a steep slope

weather can occur year round in the most southerly portion. Fire seasons are most active during spring and fall in the Great Plains, Great Lakes, and North Atlantic regions. The pattern of seasonal fire occurrence in Australia has also been mapped,[221] as has the frequency of large fire occurrences.[97]

Topography. The steeper the slope, the more rapid the spread of fire. Fire usually burns uphill, especially during daylight hours. Changes in topography can cause rapid and violent changes in fire behavior (Fig. 12-26). On steep terrain, rolling firebrands may cause a fire to initially spread downhill, followed by upslope runs. Mountainous terrain can modify wind speed and direction and in turn influence fire behavior in exceedingly complex ways.[335] Slope exposure or aspect has a very pronounced diurnal effect on fine fuel flammability, a fact directly incorporated into the Campbell Prediction System of wildland fire behavior (www.dougsfire.com).[88]

Extreme Fire Behavior

Extreme fire behavior is generally considered a level of fire activity that often precludes any fire suppression action by conventional means.[240] It usually involves one more of more of the following characteristics:
- High rates of spread and intensity
- Crowning
- Prolific spotting
- Large fire whirls
- Well-established convection column

Fires exhibiting such phenomena often behave in an erratic, sometimes dangerous manner. Fire whirls appear frequently in and around wildland fires.[124] Most fire whirls are small and short-lived, but occasionally one becomes large and strong enough to do tornado-like damage and cause serious injury, such as occurred at Mt. Kuki, Japan in 1977[151]; death is also a distinct possibility. Their occurrence is commonly associated with unstable air, moderate winds, and large heat sources created by plentiful and dry fuel concentrations (Fig. 12-27) or terrain configurations that concentrate the heat from the fire (e.g., lee side of a ridge).[171,181] These fire-induced tornado-like whirlwinds can travel up to 2.5 km (1.6 miles) from the main fire[358] and can cause considerable damage and even lead to fatalities. On March 7, 1964, fire whirlwind that formed on the Polo fire near Santa Barbara, California cut a 1.6 km (1 mile) path, injuring four people, destroying two houses, a barn, and four automobiles, and wrecking a 100-tree avocado orchard.[300] The fringe of the fire whirl passed over a fire truck and sucked out the rear window of the vehicle. A firefighter standing on the rear platform of the vehicle was pulled up vertically so that his feet were pointing to the sky while his hands clasped the safety bar. A small piece of plywood was rammed 7.6 cm (3 in) into an oak tree.

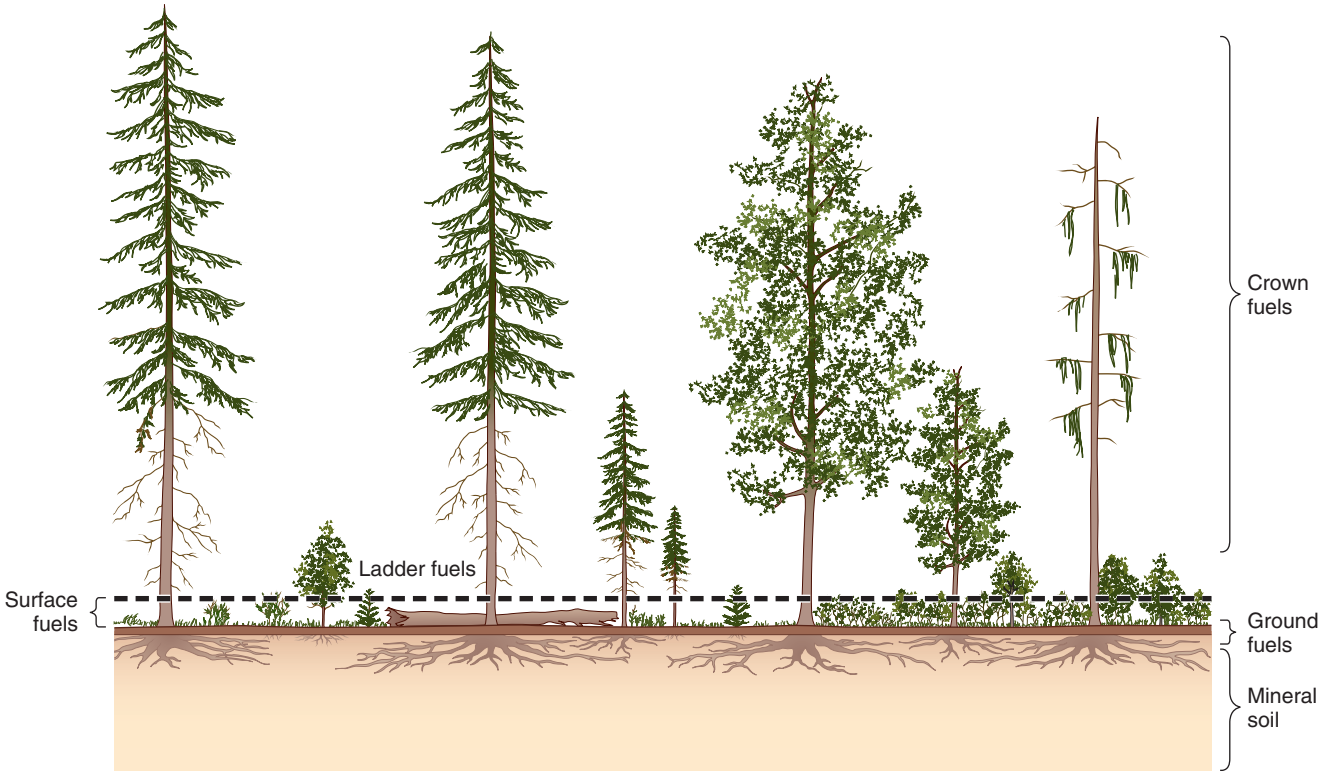


Figure 12-23. Profile of a stylized forest stand showing the location and classification of fuel complex strata. Wildland fuels contain energy, stored over extended periods through photosynthetic processes, that is released rapidly, occasionally explosively, in combustion. (Adapted from Brown AA, Davis KP: Forest Fire: Control and Use, 2nd ed. New York, McGraw-Hill, 1973.)

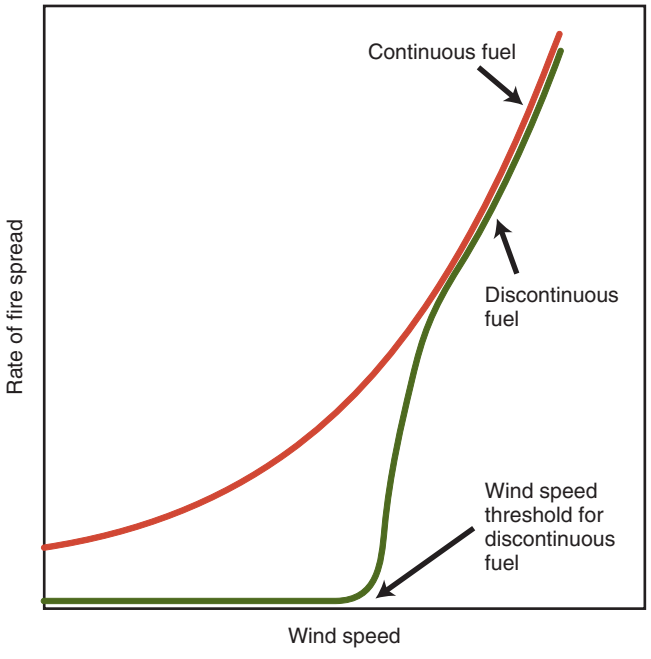


Figure 12-24. Discontinuous fuel types will not carry a fire until wind speed exceeds a particular threshold value. For example, a wind of 12 km/hr (7.5 mph) has been identified from conducting experimental fires as a critical threshold for fire spread in spinifex grasslands of central Australia. In contrast, fire spread in continuous fuel types is possible even under calm conditions and, predictably, steadily increases with increasing wind. (Adapted from Gill AM, Burrows ND, Bradstock RA: Fire modeling and fire weather in an Australian desert. CALMScience Suppl 4:29, 1995.)

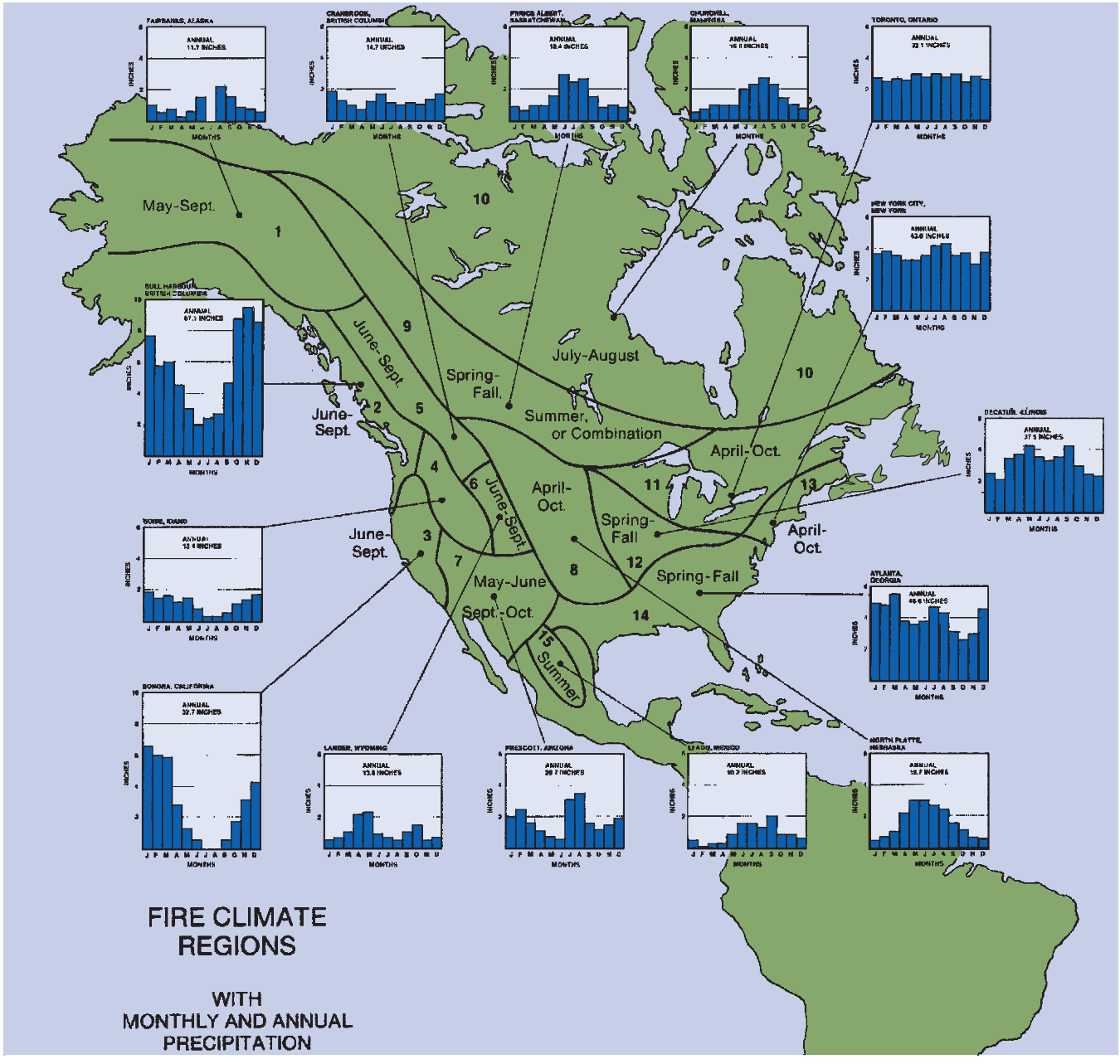


Figure 12-25. Fire climate regions of North America, based on geographic and climatic factors: (1) interior Alaska and the Yukon, (2) north Pacific Coast, (3) south Pacific Coast, (4) Great Basin, (5) northern Rocky Mountains, (6) southern Rocky Mountains, (7) Southwest (including adjacent Mexico), (8) Great Plains, (9) central and northwest Canada, (10) sub-Arctic and tundra, (11) Great Lakes, (12) Central States, (13) North Atlantic, (14) Southern States, and (15) Mexican central plateau. The bar graphs show the monthly and annual precipitation for a representative station in each of the fire climate regions. Months on the map indicate fire seasons. The graph for Decatur, Illinois, obscures the provinces of Newfoundland and Prince Edward Island on the map. (Adapted from Schroeder MJ, Buck CC: Fire Weather. Agriculture Handbook 360. Washington, DC, USDA Forest Service, 1970.)

Several years of drought combined with a national forest health issue that has produced many dead and dying forests set the stage for extreme fire behavior conditions that threaten people, property, and natural and cultural resources. Protection from these conditions requires understanding of the crown fire process. Crowning involves a fire spreading horizontally as a "wall of flame" from the ground surface up through and above the canopies of trees (Fig. 12-28) or tall shrubs. The first views inside a crown fire were documented on videotape during the International Crown Fire Modelling Experiment near Fort Providence in Canada's Northwest Territories in 1997.[373] The onset of crowning is significant from both a safety and suppression perspective. At a minimum, when a fire crowns, the rate of spread and intensity doubles, and the area burned quadruples.[13]

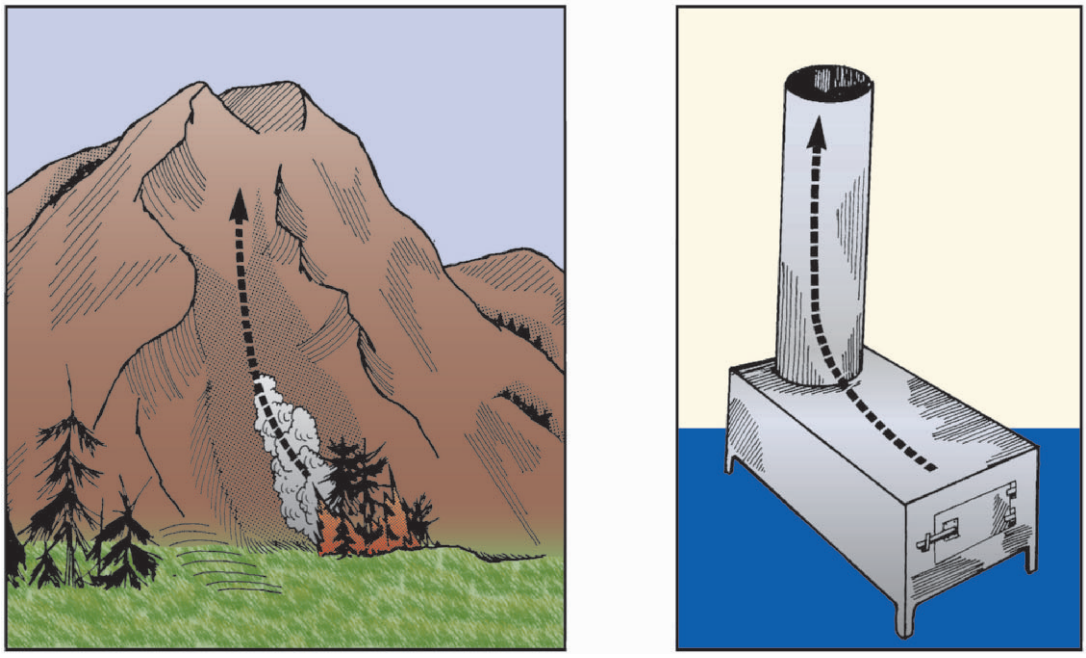


Figure 12-26. Chutes, chimneys, and box canyons created by sharp ridges provide avenues for intense updrafts (like a fire in a wood stove) and rapid rates of spread. People should avoid being caught above a fire under these topographic conditions. (Adapted from Barrows JS: Fire Behavior in Northern Rocky Mountain Forests, Station Paper No. 29. Missoula, MT, USDA Forest Service, Northern Rocky Mountain Forest and Range Experiment Station, 1951.)

Figure 12-27. Large fire whirl associated with the burning of logging slash debris near Whitecourt, Alberta. (Courtesy University of Alberta. Photo by M.Y. Ackerman.)

Figure 12-28. Experimental crown fire in a northern jack pine *(Pinus banksiana)* and black spruce *(Picea mariana)* forest near Fort Providence, Northwest Territories. The average height of flames from crowning forest fires is generally one to one and a half times taller than the tree height. Flame flashes will extend considerably higher into the fire's convection column. (Courtesy Canadian Forest Service.)

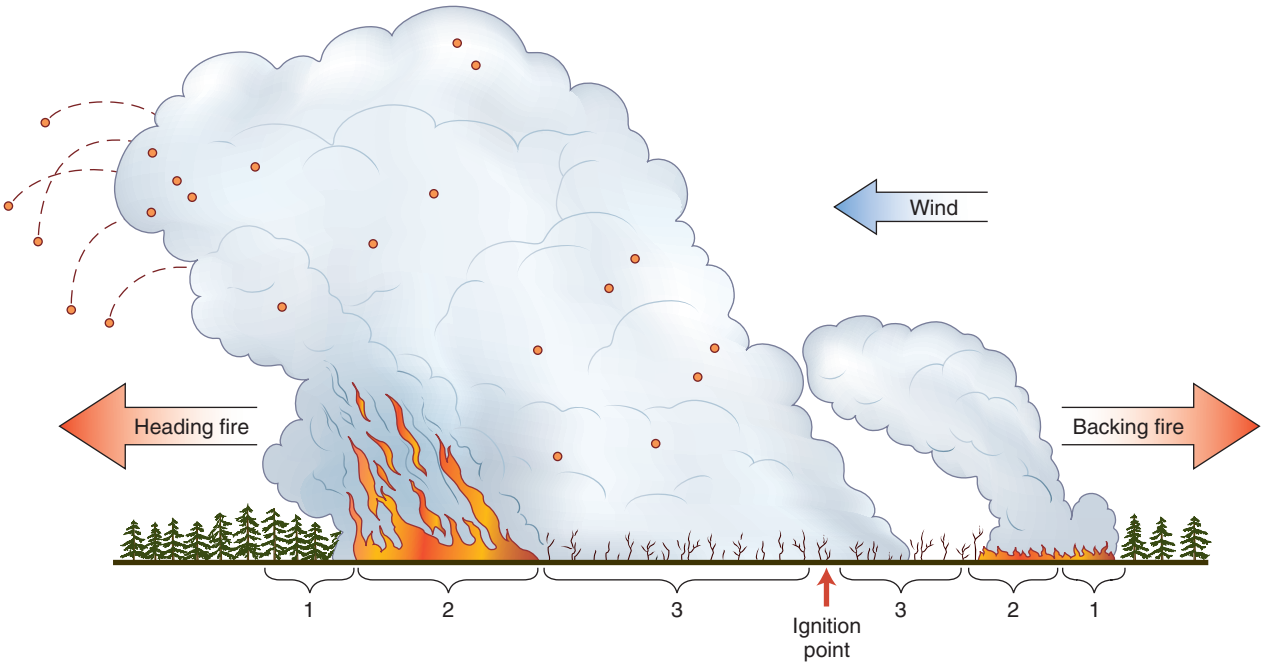


Figure 12-29. Cross-sectional view of a wind-driven crown fire, illustrating the various stages of combustion: pre-ignition (1), ignition and flaming combustion (2), and glowing or smoldering combustion (3). People are most at risk on the downwind side and upper flanks of such a free-burning fire. This type of fire is caused by winds that increase in velocity with increasing elevation above the ground. (Modified from Cottrell WH: The Book of Fire. Missoula, MT, Mountain Press, 1989.)

Author Norman Maclean[225] gives an indication of how crown fires were handled in the early part of the 20th century by the USDA Forest Service:

> *By the time they reached the fire, it had spread all over the map, and had jumped into the crowns of trees, and for a lot of years a prospective ranger taking his exam had said the last word on crown fires . . . When asked on his examination, "What do you do when a fire crowns?" he had answered, "Get out of the way and pray like hell for rain."*

This wisdom seems still valid today. Rothermel[327,329] described the conditions that produce a crown fire:

1. Dry fuels
2. Low humidity and high temperatures
3. Heavy accumulations of dead and downed fuels
4. Small trees in the understory, or "ladder fuels"
5. Steep slope
6. Strong winds
7. Unstable atmosphere
8. Continuous crown layer

The two most prominent behavior patterns of crown fires are wind driven and plume (or convection) dominated. Each type of crown fire poses a distinct set of threats to people. Free-burning wildland fires are seldom uniform and well behaved, so these descriptions of wind-driven and plume-dominated crown fire behavior may not be readily apparent. The behavior of these types of fires can be expected to change rapidly as environmental, fuel, and topographic conditions change.[327]

Wind-Driven Crown Fire. A running crown fire can develop when winds increase with increasing elevation above the ground, driving flames from crown to crown (Fig. 12-29). Steep slopes can produce the same effect. Spread rates in conifer forests can reach up to 12 km/hr (7.5 mph) for brief periods of time[200,344] and are possibly faster in mountainous terrain,[327] especially in open forest or nonforested or shrub land fuel types.[78,110] A running crown fire is accompanied by showers of firebrands downwind, fire whirls, smoke, and rapid development of a tilted convection column. As long as the wind remains fairly constant from one direction, the flanks of the fire can remain relatively safe. The greatest threat is to people who are immediately at the head, or downwind side, of the fire, although medium and long-distance spot fires can also pose an unexpected risk.[5,55]

Plume-Dominated or Convection-Dominated Crown Fire. An alternative form of crown fire develops with relatively low wind speeds or when wind speed decreases with elevation above the ground. This type of crown fire is referred to as *plume dominated* or *convection dominated* because it is characterized by a towering convection column that stands vertically over the fire (Fig. 12-30). This type of fire poses a unique threat to people because it can produce spot fires in any direction around its perimeter. It can also spread rapidly as the combustion rate accelerates.

One form of a plume-dominated crown fire that can be especially dangerous is when a downburst of wind blows outward near the ground from the bottom of the convective cell. These

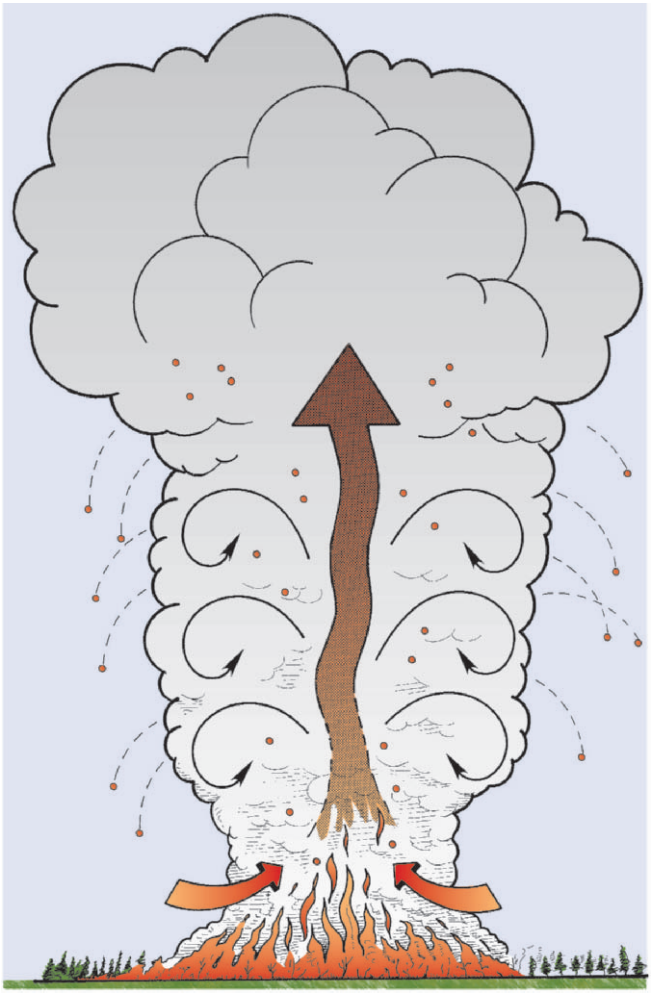


Figure 12-30. Cross-sectional view of a plume-dominated or convection-dominated crown fire. People are at risk around the complete perimeter of this type of fire because it has the potential to spread intensely or spot in any direction around its perimeter with little or no warning. This form of crown fire develops when wind velocities are relatively low or when velocities decrease with elevation above the ground coupled with critically dry and abundant fuel conditions. The convective plume associated with this type of fire may rise to 7600 to 9100 m (25,000 to 30,000 feet) above the ground. (From Cottrell WH: The Book of Fire. Missoula, MT, Mountain Press, 1989.)

winds can be extremely strong[178] and can greatly accelerate a fire's spread. This type of wind event occurred during the major run of the Dude fire near Payson, Arizona on June 26, 1990, when six firefighters were killed.[172]

Some indicators help signal the onset of a downburst from a plume-dominated crown fire. The surest indicator is the occurrence of precipitation of any amount, even a light sprinkle, or the appearance of virga (evaporating rain) below the base of a cloud formation.[327,335] Another indicator is rapid development of a strong convection column above the fire, or nearby thunder cells. A third and very short warning is the calm that develops when the in-draft winds stop before the turnabout and outflow of wind from the cell. This brief period of calm may be accompanied by a humming sound just before the reversing wind flow arrives. If any of these indicators is present, the area should quickly be evacuated and a safe refuge sought. The downburst may also break or uproot trees, creating an additional hazard for people.[327]

Value of Fire Danger Ratings

Fire management agencies employ fire danger rating systems to help them gauge ignition and fire behavior potential based on fuel characteristics, past and current weather conditions, and certain topographic variables that encapsulate fire behavior knowledge garnered from research.[10,221,372] Such systems have been demonstrated to be of value in accurately predicting fire behavior in the wildland–urban interface.[11] In commenting on the historic fatality fires around the turn of the 20th century, Brown and Davis[64] pointed out that in general terms, these situations reflected the public's indifference to forest fires that had persisted for so many years and still lingers on to some degree today. They noted:

> *The Maine fires of 1947 were a sobering reminder in this respect. They occurred at a time when fire danger measurement was well understood and means for mass public dissemination of this information were well developed. Responsible officials were aware of the mounting fire danger. Nonetheless, some fifty fires were reported to have been burning in Maine at the time the major break occurred on October 23. A contributing circumstance was the general feeling that, as it was late October, the fire season was over.*

Fire danger rating outputs are being applied directly to the task of community fire safety and protection, including early warning or alert systems linked to fire weather forecasts that operate during the fire season, such as developed for the Northwest Territories, Canada.[24] This information is now commonly accessible to the general public on the Internet regarding countries like the United States (www.fs.fed.us/land/wfas), Canada (http://cwfis.cfs.nrcan.gc.ca), and New Zealand (http://nrfa.fire.org.nz/fire_weather). Forecasts of fire weather conditions are by themselves useful; the information can also be used to forecast fire danger indices and potential fire behavior characteristics.[50,132,335] In Australia, television and radio are relied on extensively to alert the public of "total fire ban" days (i.e., extreme fire danger) based on current fine fuel moisture levels, drought status, and forecasted fire weather conditions.[104] Fire danger rating and fire behavior predictions can be used to judge whether evacuations are required, or to assist people to prepare for the eventuality of a fire arriving.[189] Evacuation routes must be established in advance, and the actual time taken to evacuate to a safe zone needs to be determined in much the same manner as a wildland firefighter uses an escape route to reach a safety zone or an urban dweller evacuates a building fire using preidentified travel routes and exit locations.

FIRE-RELATED INJURIES AND FATALITIES

Most fatalities in wildland fires occur on days of extreme fire danger when people are exposed to abnormally high heat stress caused by weather or proximity to fires. Loss of life is dramat-

TABLE 12-3. Location of Bodies of 53 Persons Who Died in Tasmanian Fires, February 7, 1967

LOCATION	NO. OF DEATHS
Mustering stock	2
Firefighting	11
Traveling in a vehicle	2
Escaping from and found at some distance from houses	11
Within a few meters of houses	10
In houses	17

From McArthur AG, Cheney NP: Report on Southern Tasmania Bushfires of 7 February 1967. Hobart, Australia, Government Printer, 1967.

TABLE 12-4. Age Distribution of 53 Persons Who Died in Tasmanian Fires, February 7, 1967

AGE GROUP (yr)	NO. IN GROUP	AVERAGE AGE (yr)
1–25	1	23
26–50	13	38
51–75	26	64
76–88	13	82

From McArthur AG, Cheney NP: Report on Southern Tasmania Bushfires of 7 February 1967. Hobart, Australia, Government Printer, 1967.

ically highlighted under extreme burning conditions; however, many more people are injured than are killed by fires.

One of the worst fire disasters in Australia occurred on February 7, 1967, when 62 people died in Tasmania.[233,410] Analysis of location and age of 53 individuals at the time of death is instructive (Tables 12-3 and 12-4). Most people whose bodies were found within or near houses were old, infirm, or physically disabled. More than half of the houses vacated by the 11 people who traveled some distance before being killed were not burned. Most of these victims would probably have survived if they had remained in their homes. Most of the 11 firefighters who died were inexperienced. Many might have survived if they had observed fire behavior and safety rules.

Krusel and Petris[205] investigated the circumstances surrounding the 32 civilian deaths that occurred during the 1983 Ash Wednesday bushfires in Victoria. As a result, they identified three categories of victims, suggesting ways to address the deficiencies in the manner individuals respond to the wildfire threat:

- Victims who recognized the real threat to their safety with enough time to save their lives, but chose an ineffective survival strategy.
- Victims who did not recognize the real threat to their safety in time to implement an effective survival strategy.
- Victims who were physically incapable of implementing an effective survival strategy.

Similar situations have been reported around the world. For example, during the 1993 fire season in the Tarragona Province of eastern Spain, five people were killed trying to escape from their home, built in the woods and surrounded by a fire; four of the victims were elderly and did not dare to flee until it was impossible to escape.[394]

Common Denominators of Fire Behavior on Fatality Fires

A review of USDA Forest Service records between 1926 and 1976 shows that 145 men died in 41 fires from fire-induced injuries.[274,276,416] Besides the 1949 Mann Gulch and 1990 Dude fires, large losses occurred on the 1933 Griffith Park fire in California (29 deaths and 150 others injured),[145] the Blackwater fire in Wyoming in 1937 (15 deaths),[62] the Pepper Hill fire in Pennsylvania in 1938 (8 deaths),[337] the Hauser Canyon fire in California (11 deaths),[92,349] the Rattlesnake fire in California in 1953 (15 deaths),[224] the 1956 Inaja fire in California (11 deaths),[336,382] the 1966 Loop fire in California (12 deaths),[128] and the 1968 Canyon fire in California (8 deaths).[129] The United States is not the only country to have had firefighters killed by entrapments and burnovers. For example, 25 soldiers were killed while involved in wildland firefighting operations near Lisbon, Portugal on September 6, 1966.[193] Australia has experienced a number of incidents,[97,193] as has Greece. On December 2, 1998, five volunteer firefighters were killed near the town of Linton, Victoria when the tanker they were traveling in was overrun by fire.[120]

Wilson's analysis of people killed by wildfires in areas protected by other federal, state, county, and private agencies indicated 77 fire-induced fatalities in 26 fires. Wilson[416] identified some common features connecting these fatal fires:

1. Relatively small fires or isolated sectors within larger fires seemed associated with most fatal incidents.
2. Flare-ups of presumed controlled fires were particularly hazardous. Fatalities occurred in the mop-up stage.
3. Unexpected shifts in wind direction or speed occasionally caused flare-ups in deceptively light fuels.
4. Gullies, chimneys, and steep slopes directed fires to run uphill.
5. The violent wind vortices left by helicopters and air tankers may have caused flare-ups in previously controlled areas.

Many firefighters were surprised to learn that tragedy and near-miss incidents occurred in fairly light fuels, on small fires, or on isolated sectors of large fires, and that the fire behavior was relatively quiet just before the incident, even in the cases involving aircraft.[129,135,179] Many have been lead to believe that it is the conflagration or large, high-intensity crown fire in timber and heavy brush that traps and kills firefighters. Yet, with some exceptions, most fires were innocent appearing just before the fatal moment. Case studies of wildland firefighter fatality incidents from other regions of the world suggest similar patterns and circumstances—for example, the 25 fatalities at Puerto Madryn, Argentina on January 21, 1994,[137] 10 fatalities in the Sabie district of the Mpumalanga Province of South Africa in 1994,[138] and 5 fatalities associated with three different incidents that occurred in Portugal during the 1999 and 2000 fire seasons.[397]

Wilson concluded that the hairline difference between fatal fires and near-fatal fires was determined by the individual's reaction to a suddenly critical situation. Escapes were due to luck, circumstances, advance planning, a person's ability to avoid panic, or a combination of these factors. Frequently, poor visibility and absence of concise fire information threatened

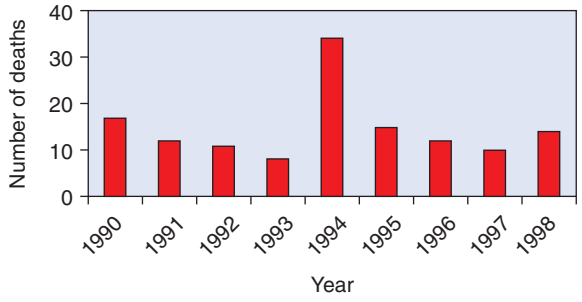


Figure 12-31. The annual death toll for persons who died from all causes while involved in fighting wildland fires in the United States from 1990 to 1998 (133 total deaths). (From Mangan R: Wildland Fire Fatalities in the United States: 1990–1998, Technical Report 9751-2817-MTDC. Missoula, MT, USDA Forest Service, Missoula Technology and Development Center, 1999.)

survival opportunities by creating confusion and panic. Wilson's findings are summarized in a popular booklet published by the National Wildfire Coordinating Group.[273] The British Columbia Forest Service found many of the same factors identified by Wilson in their investigation of a number of close calls or near misses that occurred during the 1994 fire season.[39] Although there have been few wildland firefighter fatalities in Canada,[30,377] those that have occurred are also in alignment with Wilson's findings.[49]

Most fatality fires are the result of a temporary escalation in fire spread, an increase in flame dimensions, crowning and spotting activity, or the development of large fire whirls, often occurring suddenly and with very little warning. A gentle surface fire will rapidly develop into a high-intensity fire. This could occur, for example, as a result of thunderstorm downdraft winds,[106,335] a change in fuel types,[39] fire whirl development upon the fire reaching a ridgeline with an opposing gradient wind,[124] or a "slope reversal" (e.g., fire slowly backs down a northerly aspect, crosses the drainage, and then rapidly spreads up the south-facing slope),[315] such as documented with time-lapse photography on the 1979 Ship Island fire in central Idaho.[271] As the moisture content of dead and live fuels decreases, fires become increasingly more responsive to slight changes in wind strength and slope steepness. Once "critically" dry levels are reached, fire behavior becomes exceedingly unstable.[22,71,221]

Nature of Injuries and Fatalities

Fire-related injuries and fatalities are a direct consequence of heat, flames, smoke, critical gas levels, or indirect injuries[229,255] (Fig. 12-31). Injuries and fatalities associated with wildland fires fall into one of five categories: heat (direct thermal injury, inhalation, and heat stress disorders), flames (direct thermal injury and inhalation), smoke (inhalation and mucous membrane irritation), critical gas levels (oxygen, carbon monoxide, and carbon dioxide), and indirect effects (acute and chronic medical disability and trauma).

Intense fires that produce very high temperatures generally last for only a short time. The duration of intense heat increases with fuel load, being greater in a forest fire where heavy fuels[368] are burning than in a grass[106] or shrub fire (see Table 12-2). Temperatures near the ground are lower because radiant heat is offset somewhat by inflow of fresh air and the fact that gases of combustion rise and are carried away by convection.[96] Close to the ground, within a few meters of flames reaching up to 11 m (36 ft), air temperatures may be less than 15° C (59° F) above ambient temperature. The breathing of heated air can be tolerated for 30 minutes at 93° C (199° F) and for 3 minutes at 250° C (482° F).[221] Death or severe pulmonary injury occurs when these limits are exceeded. Thermal injuries of the respiratory tract frequently contribute to the clinical picture of smoke inhalation. People trapped in a fire may have no choice but to breathe flame or very hot gases. This usually injures the tissues of the upper airway and respiratory tract, most commonly the nose, nasopharynx, mouth, oropharynx, hypopharynx, larynx, and upper trachea. These injuries may result in edema that obstructs the airway and produces asphyxia or that causes tracheitis and mediastinitis.

Signs of thermal injury to the airway include thermal injuries to the head, face, and neck; singed facial hair; burns of the nasal, oral, or pharyngeal mucosa; and stridor or dysphonia.[251,365,423] Associated with a history of exposure to flame and hot gases in a closed space, these clinical findings strongly suggest the presence of a thermal injury to the airway. With the potential for acute airway obstruction, there is obvious urgency in establishing this diagnosis. Most experts who treat thermal injuries of the tracheobronchial tree advocate early visualization of the vocal cords by laryngoscopy and bronchoscopy.[252] Bronchoscopy is a useful predictor of the clinical course and urgency of intensive care unit intervention. In addition, one report shows a significantly greater incidence of pneumonia and late mortality in people with facial burns than in those without them.[421]

Burns of the lower trachea are rarely reported. In fact, injuries to and beyond the carina are difficult to produce when the trachea is cannulated and hot gases are delivered in the anesthetized dog.[249,422] Air has a very low specific heat and is therefore a poor conductor of thermal energy. In addition, the thermal exchange systems of the upper airway are quite efficient. The hot gas or flame is cooled sufficiently in the upper airway so that it does not burn the bronchi or more distal structures. However, although water or steam in the hot gas mixture is probably rare, it is a far more efficient conductor of heat and permits significant thermal injury to the lower trachea and bronchi.

A delayed onset (2 to 24 hours after smoke inhalation) of pulmonary edema and adult respiratory distress syndrome is widely reported and should be anticipated. Whether this results from direct injury to alveoli, prolonged hypotension, or cerebral hypoxia and cerebral edema is unclear.

Heat stress[340] occurs when air temperature, humidity, radiant heat, and poor air movement combine with strenuous work and insulative clothing to raise body temperature beyond safe limits. Sweating cools the body as moisture evaporates. When water lost through sweating is not replaced, physiologic heat controls can deregulate, and body temperature may rise, leading to heat exhaustion or heatstroke (see Chapters 10, 11, and 64).

Direct contact with flames causes thermal injury, and death is inevitable with exposure for long periods. Burns may be superficial, partial, or full thickness (see Chapter 13). Immediate death results from hypotension, hyperthermia, respiratory failure, and frank incineration.

As mentioned earlier, the common cause of asphyxia in wildland fire is smoke. Danger increases where smoke accumulates because of poor ventilation, as in caves, box canyons, narrow valleys, and gullies.

Dense, acrid smoke is particularly irritating to the respiratory system and eyes. Excessive coughing induces pharyngitis and

Figure 12-32. USDA Forest Service Ranger Ed Pulaski led 42 men and two horses to this mine tunnel near Placer Creek in northern Idaho to seek refuge from the 1910 firestorm. One man failed to get into the tunnel and was burned beyond recognition. All the men in the tunnel were evidently unconscious for a period of time. Five died inside, apparently from suffocation. The remainder of the crew was evacuated to the hospital in Wallace, where all recovered. For a fuller account of this incident, see reference 312. (Courtesy USDA Forest Service. Photo by J.B. Halm.)

vomiting. Keratitis, conjunctivitis, and chemosis may make it impossible to keep the eyes open.

There are concerns about the levels of oxygen, carbon monoxide, and carbon dioxide associated with wildland fires. Critical levels readily occur in a closed space and near burning or smoldering heavy fuels, but the open space associated with wildland fires usually contributes to continual mixing of air. Misconceptions or myths about lack of oxygen or excessive carbon monoxide and carbon dioxide in a wildland fire abound in the popular literature.[106]

Flaming combustion can be maintained only at oxygen levels that exceed 12%, a level at which life can also be supported.[96,221] With continued in-drafts of air that feed the flames, a fresh source of oxygen is usually present. Even mass fires, in which large tracts of land are burning, rarely reduce oxygen to hazardous levels. Low oxygen levels may occur, however, where there is little air movement, such as in caves or mine shafts (Fig. 12-32) or in burned-over land that continues to smoke from smoldering fuels.

Concentrations of carbon monoxide exceeding 800 parts per million (ppm) can cause death within hours. Most fires produce small quantities, but atmospheric concentrations rarely reach lethal levels because of air movement. High concentrations appear to be associated with smoldering combustion of heavy fuels, such as fallen trees or slash piles, and carbon monoxide may also collect in low-lying areas or underground shelters (e.g., root cellars).[98] Outdoors, the danger lies in continual exposure to low concentrations that can increase blood carboxyhemoglobin levels. Prolonged exposure affects the central nervous system, resulting in headache, impaired judgment, progressive lethargy, decreased vision, and other psychomotor deficits.[416]

TABLE 12-5. Human Response to Decreased Ambient Oxygen at Sea Level

AMBIENT OXYGEN (%)	HUMAN RESPONSE
20.9	Normal function
16–18	Decreased stamina and capacity for work
12–15	Dyspnea with walking; impaired coordination; variable impaired judgment
10–12	Dyspnea at rest; consciousness preserved; impaired judgment, coordination, and concentration
6–8	Loss of consciousness; death without prompt reversal
<6	Death in 6 to 10 minutes

Carbon monoxide levels of 50 ppm were measured close to a prescribed burn in grass.[123] In another estimate, concentrations of 30 ppm were found roughly 61 m (200 ft) from the fire front. Studies on the 1974 Deadline and Outlaw forest fires in Idaho showed that firefighters were exposed to levels above the standards proposed by the National Institute of Occupational Safety and Health (35 ppm over an 8-hour period).[378] However, more recent measurements of carbon monoxide levels and particulates made on wildfires between 1992 and 1995[321] indicate that wildland firefighters are seldom exposed to smoke that exceeds the Occupational Safety and Health Association's permissible exposure limits.

Decreased ambient oxygen may contribute to hypoxia and the overall picture of smoke inhalation (Table 12-5). This mechanism is at least variably operant. When standing gasoline was ignited in a closed bunker, the fire self-extinguished, whereas the ambient oxygen level remained at 14%, a survivable level.[249] Injecting burning gasoline or napalm into bunkers produced nearly complete and prolonged exhaustion of ambient oxygen. Conflicting data make it difficult to classify definitively situations in which decreased ambient oxygen and subsequent hypoxia of exposed individuals contribute to the clinical picture of smoke inhalation. Studies in which ambient oxygen was measured by scientists did not show significant depletion at the scene of the fire.[173]

Few data are available on levels of carbon dioxide around wildland fires. Although it may be produced in large quantities, it apparently never reaches hazardous concentrations, even in severe fire situations.[96,221]

The quantity of burning fuel and type of topography affect levels of oxygen and toxic gases. Danger is greater in forest fires where heavy fuels burn over long periods of time than in quick-moving grass and shrub fires. Topography has a major influence: caves, box canyons, narrow canyons, gulches, and other terrain features can trap toxic gases or hinder ventilation, thereby preventing an inflow of fresh air. Although most fatalities result from encounters with smoke, flames, and heat, critical gas levels can induce handicaps sufficient to render the victim more vulnerable to other hazards. Whereas respirators[231] are not routinely used on wildland fires or on wildland–urban

interface fires, they could possibly play a role in the future as a means of protection against airborne toxic materials.

Wildland Fires, Air Toxins, and Human Health

In the United States each year, about 80,000 firefighters are involved with suppression activities on about 70,000 wildland fires that burn an average of more than 0.8 million hectares (2 million acres). In 1988, more than 2 million hectares (5 million acres) of land were burned, with a total combined suppression cost exceeding U.S. $600 million. The firefighting effort has another cost that has not been quantified: the effect of smoke on firefighter health and productivity. Over the 4 months of the Greater Yellowstone Area fires, about 40% of the 30,000 medical visits made by wildland firefighters were for respiratory problems. More than 600 firefighters required subsequent medical care. In the Happy Camp area during the Klamath fire complex in California in 1987, ambient concentrations of carbon monoxide measured as high as 54 ppm on a volume basis.[400] A better understanding of the long-term effects of wildland fire smoke on people is clearly needed, although as Dr. Brian Sharkey,[343] a wildland firefighter health and safety specialist with the USDA Forest Service's Missoula Technology and Development Center who coordinated an 8-year study of the health hazards of smoke on wildland firefighters,[341,342] has noted, "firefighters just keep coming back year after year." The combustion products of concern include these classes of materials:

1. Particulate matter
2. Polynuclear aromatic hydrocarbons
3. Carbon monoxide
4. Aldehydes
5. Organic acids
6. Semivolatile and volatile organic compounds
7. Free radicals
8. Ozone
9. Inorganic fraction of particles

Large variances are associated with the development of smoke combustion products and exposure to the materials of concern.[400] Ward and colleagues[401] indicated that the toxicity of the combination of combustion products depends on the relative concentrations of the individual compounds, as well as the overall concentration and length of exposure. Individual toxicities are associated with many of the compounds found in smoke. The combined toxicity of these substances is not known. A detailed set of studies is being carried out to provide answers so that risk management options can be exercised. One of these studies suggested that wildland firefighters experience a small cross-seasonal decline in pulmonary function and an increase in several respiratory symptoms.[332] Eye irritation, nose irritation, and wheezing were associated with recent firefighting.

The strenuous work of fighting or escaping a fire magnifies chronic illnesses, age disabilities, exhaustion, and cardiovascular instability. Common trauma is induced by falling trees or limbs, rolling logs or rocks, vehicular accidents, poor visibility, panic, falling asleep in unburned fuels that later ignite, and leaving the safety of buildings and vehicles. Cuts, scrapes, scratches, lacerations, fractures, and eye injuries (foreign particles, smoke irritation, sharp objects) are other common afflictions. Poison oak, poison ivy, stinging insects, and poisonous snakes are additional sources of trauma during wildland fires. To avoid fire-related injuries and fatalities, a person must keep attuned to mental and physical stress levels and be aware of cumulative effects.[340] Ignorance of this simple principle is disastrous.

WILDLAND–URBAN INTERFACE FIRE SURVIVAL PRINCIPLES AND TECHNIQUES

History has demonstrated repeatedly that individuals simply were not prepared to make correct choices of survival alternatives under stressful situations. At least 420 wildland firefighters have been killed in the United States since 1910 as a direct result of a burnover or entrapment by a free-burning wildfire (www.nifc.gov). Overconfidence, complacency, ignorance, bad habits, lack of preparation, poor decision making, and a whole host of other human factors quickly lead to improper and unsafe actions during fire emergencies.[289,406,416] "Learning from mistakes" in these settings is not a reasonable education strategy because second chances are frequently unavailable in the wildland firefighting profession.

The USDA Forest Service organized a task force in 1957 to "study how we might strengthen our ways and means of preventing firefighting fatalities."[248] A major recommendation was to adopt service-wide standard firefighting orders. Ten standard firefighting orders[232,247] summarize the fundamental principles of safety on the fireline (Box 12-5). Although these were written

Box 12-5. Ten Standard Firefighting Orders Adapted for the General Public

1. Keep informed of fire weather conditions, changes, and forecasts and how they may affect the area where you are located.
2. Know what the fire is doing at all times through personal observations, communication systems, or scouts.
3. Base all actions on current and expected behavior of the fire.
4. Determine escape routes and plans for everyone at risk and make certain that everyone understands routes and plans.
5. Post lookouts to watch the fire if you think there is any danger of being trapped, of increased fire activity, or of erratic fire behavior.
6. Be alert, keep calm, think clearly, and act decisively to avoid panic reactions.
7. Maintain prompt and clear communication with your group, firefighting forces, and command and communication centers.
8. Give clear, concise instructions and be sure that they are understood.
9. Maintain control of the people in your group at all times.
10. Fight fire aggressively, but provide for safety first. (Nonqualified or untrained and improperly dressed or equipped persons should only engage in firefighting operations when it is absolutely necessary to assist injured persons. Wildland firefighting is a physically demanding task and should not be attempted by persons who are not in good physical condition.)

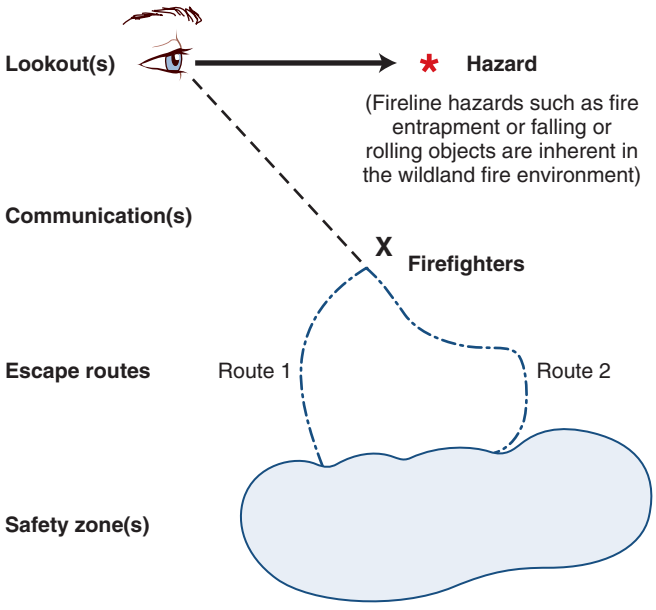


Figure 12-33. Concept of the LCES wildland fire safety system. (From Gleason P: LCES—the key to safety in the wildland fire environment. Fire Manage Notes 52(4):9, 1991.)

for wildland firefighters, they apply to all people working, living, or traveling near wildland fires and are adapted here to remind emergency medical personnel, wildland homeowners, and recreationists of safety precautions to be taken around wildland fires. Much of this material is summarized in the handy "Incident Response Pocket Guide."[280]

LCES: The Key to Safe Procedures in Wildland Fire Environments

LCES stands for Lookout(s), Communication(s), Escape routes, Safety zone(s).[167] These variables are key components in evaluating the threat posed by fireline hazards and determining the best course of action (Fig. 12-33). Some fire management agencies have added an "A" to LCES, thus LACES, for a variety of meanings—Attitude, Awareness, and Anchor points.[208,375] The importance of an anchor point is quite vividly highlighted in the video, *A Firefighter's Return from a Burnover: The Kelly York Story,* which details the severe burn injuries received by a volunteer firefighter while attempting to suppress a grass fire in central California during the 1995 fire season.[148]

The wildland fire environment's basic hazards are lightning, volcanoes, falling of fire-weakened trees and snags, rolling rocks and logs, entrapment by free-burning fires, respirable particulates, air toxins, and heat stress. When these hazards exist, there are two options:

1. Do not enter the environment, ***or***
2. Adhere to safe procedures according to LCES.

LCES should be viewed from a "systems" point of view, stressing their interdependence. For example, the best safety zone is worthless if the escape route does not offer access at the point of need. People must be familiar with the LCES plan well before it is needed. In addition, the nature of wildland fires dictates that LCES be redefined in pace with changing conditions.

The LCES wildland fire safety system is implemented as follows:

1. *Lookout*(s). Fixed lookouts or roving lookouts must be where both the hazard and the people can be seen. A lookout is trained to observe the wildland fire environment and to recognize and anticipate changes in fire behavior. When the hazard becomes a potential threat or danger, the lookout relays this information so people can depart for the safety zone.
2. *Communication*(s). Communication refers to alerting people to the approaching or pending hazard. Promptness and clarity are essential.
3. *Escape routes.* These paths lead from a currently threatened position to an area free from danger. More than one escape route must be available. Escape routes are probably the most elusive component of LCES because they change continuously. Timely access to safety zones is the most important component.[52] Recent research on wildland firefighter travel rates by the Forest Engineering Research Institute of Canada[21,45] and others suggest that although civilians could momentarily advance at a rate in excess of what most wildland fires are capable of achieving, even the most fit individuals would not be able to sustain such a pace for more than a few minutes before being burned over by an advancing fire, especially on steep slopes—escaping uphill is thus a very questionable concept, unless a safety zone is known to very near by. Furthermore, some fire researchers believe there is a common tendency to overestimate the distance to a fire when observing through the forest, which may lull firefighters into thinking there is more time available for an orderly exit than is actually the case.[105]
4. *Safety zone*(s). In a safety zone, threatened persons find adequate refuge from danger. The size of the safety zone must be sufficient to provide protection from flames, radiant heat, convective heat, falling trees and snags, and rolling rocks and logs. The necessary size varies with changes in fuels, topography, wind conditions, and fire intensity. The question of whether a previously burned area will serve as a safety zone depends not only on the size of the area but also on the degree of completeness of fuel consumption, both vertically and horizontally.[78,297] What constitutes a safety zone varies widely among individuals.[357] Based on theoretical consideration for radiation emitted from a "wall of flame" under idealized conditions (e.g., level terrain, steady-state fire conditions),[75] it has been suggested, from the standpoint of flame radiation, that a safety zone should be large enough that the distance between the human occupants and flame front be at least 4 times the maximum expected flame height at the edge of the safety zone[77] (Table 12-6). It should be emphasized that in deriving this rule of thumb, several assumptions had to be made that could ultimately limit the usefulness of this simple guide,[367] although some field verification has been accomplished.[76] Furthermore, both the modeling and resultant radiation-based guide do not consider flame impingement or horizontal reach into a clearing, fire whirls, or any allowance for convective heat transfer, which will be significant under windy conditions in sloping terrain.[106,326] Obviously, further study, including field measurements, and development are warranted (e.g., allowance for convective heating on a slope), which is

TABLE 12-6. Minimum Safety Zone Separation Distances and Sizes (Circular Shape) in Relation to Flame Height*

FLAME HEIGHT		SEPARATION DISTANCE		AREA†	
METERS	FEET	METERS	FEET	HECTARES	ACRES
2	6.5	8	26	0.02	0.05
5	16	20	64	0.13	0.31
10	33	40	132	0.5	1.2
20	66	80	264	2.0	5.0
40	128	160	512	8	20
60	262	240	1048	18	45

*For a single person on flat topography based on the "4-times-flame-height" rule of thumb for radiant heat only (i.e., no convection) and no allowance for flame impingement or other fireline hazards.[75,77] It is assumed that the person is standing upright and is properly clothed, including headgear and gloves.

†Assuming the area of a circle is equal to ~3.14159 times the Separation Distance,[2] where the Separation Distance is deemed to be the radius of a circle. For perspective, a typical North American ice hockey rink is 0.16 hectares (0.39 acres) in size, whereas Canadian and American football fields are about 0.6 hectares (1.5 acres) and 0.4 hectares (1.1 acres) in size, respectively. In turn, the maximum sizes of outdoor soccer and rugby fields would be approximately 0.8 hectare (2.0 acres) and 1.0 hectare (2.5 acres), respectively. A full-size basketball court is about 0.04 hectares in size (0.1 acres).

indeed the case.[73] Nevertheless, while acknowledging these limitations, this rule of thumb has been incorporated into existing operational guidelines,[280] training course material,[377] and computer programs.[35] According to Butler and Forthofer,[80] additional modeling (e.g., radiation on a sloping surface), subsequent observations, and other field measurements support "4 times flame height" for minimum safety zone size.

While serving as the ignition boss during the International Crown Fire Modelling Experiment (ICFME) in the Northwest Territories,[19,361] one of the authors (MEA) was able to informally evaluate the Butler and Cohen[75,77] safety zone guideline. Based on observations and the experience of being on the backside or upwind edge of all the ICFME experimental crown fires,[364,373] the 4-times-flame-height rule of thumb seems to be reasonably valid. As Butler and Forthofer[80] point out, "It's important to realize that this should be considered a minimum—meaning that in all cases larger is better."

In using Butler and Cohen's[75,77] rule of thumb for safety zones or in turn Table 12-6, the logical question is: How does one go about estimating flame height in advance of a fire's occurrence? On the basis of various fire behavior observations and various analyses, we can say with some degree of certainty that the maximum flame heights in grasslands, shrub lands, and hardwood stands would vary, depending on the burning conditions, from about 2 to 10 m (6 to 33 ft). Thus, a separation of at least 40 m (130 ft) should be adequate in most fuel types that do not contain conifer trees. The average flame height of crown fires in conifer forests is generally 2 to 2.5 times the stand height.[364]

Any area smaller than a safety zone should be regarded as a possible "survival zone" or site. Unfortunately, no quantitative description of what constitutes a survival zone presently exists. Nevertheless, specific case study examples (e.g., 1937 Blackwater fire in Wyoming, 1958 Wandilo fire in South Australia, and 1990 Dude fire in Arizona) suggest that survival is possible in relatively small areas even when exposed to a highly hostile thermal environment provided a person maintains a prone position and uses every possible bit of protection against radiation, convection, and direct flame contact. Movement within the survival zone may also be possible and necessary as the flame front surrounds the site.

Eighteen "Watch Out!" Situations in the Wildland Fire Environment

The following list of 18 "Watch Out!" situations, adapted from the wildland fire community's safety guidelines,[246,250,346] are of particular relevance to emergency medical personnel and wildland users; each of these situations have come about as a result of one or more wildland firefighter fatalities:

1. You are moving downhill toward a fire, but must be aware that fire can move swiftly and suddenly uphill. Constantly observe fire behavior, fuels, and escape routes, assessing the fire's potential to run uphill.
2. You are on a hillside where rolling, burning material can ignite fuel from below. When below a fire, watch for burning materials, especially cones and logs, that can roll downhill and ignite a fire beneath you, trapping you between two coalescing fires.
3. Wind begins to blow, increase, or change direction. Wind strongly influences fire behavior, so be prepared to respond to sudden changes.
4. The weather becomes hotter and drier. Fire activity increases, and its behavior changes more rapidly as ambient temperature rises and relative humidity decreases.
5. Dense vegetation with unburned fuel is between you and the fire. The danger in this situation is that unburned fuels can ignite. If the fire is moving away from you, be alert for wind changes or spot fires that may ignite fuels near you. Do not be overconfident if the area has burned once because it can reignite if sufficient fuel remains.
6. You are in an unburned area near the fire where terrain and cover make travel difficult. The combination of fuel and difficult escape makes this dangerous.
7. Travel or work is in an area you have not seen in daylight. Darkness and unfamiliarity are a dangerous combination.
8. You are unfamiliar with local factors influencing fire behavior. When possible, seek information on what to expect from knowledgeable people, especially those from the area.
9. By necessity, you have to make a frontal assault on a fire with tankers. Any encounter with an active line of fire is dangerous because of proximity to intense heat, smoke, and flames, along with limited escape opportunities.
10. Spot fires occur frequently across the fireline. Generally, increased spotting indicates increased fire activity and intensity. The danger is that of entrapment between coalescing fires.
11. The main fire cannot be seen, and you are not in communication with anyone who can see it. If you do not know the location, size, and behavior of the main fire, planning becomes guesswork, which is an unfavorable response.
12. An unclear assignment or confusing instructions have been received. Make sure that all assignments and instructions are fully understood.

13. You are drowsy and feel like resting or sleeping near the fireline in unburned fuel. This may lead to fire entrapment. No one should sleep near a wildland fire. If resting is absolutely necessary, choose a burned area that is safe from rolling material, smoke, reburn, and other dangers or seek a wide area of bare ground or rock.
14. Fire has not been scouted and sized up.
15. Safety zones and escape routes have not been identified.
16. You are uninformed on strategy, tactics, and hazards.
17. No communication link with crew members or supervisor has been established.
18. A line has been constructed without a safe anchor point.

Humorous artwork has even been developed to help illustrate or visualize the 10 standard fire orders and 18 "Watch Out!" situations.[256]

Fifteen Structural "Watch Out!" Situations for the Wildland–Urban Interface

Continuing development creates ever more wildland–urban interface; therefore, fires are tending to occur more often in that interface. These 15 structural "Watch Out!" situations have been defined to increase awareness of structural fire dangers[379]:

1. Access is poor (e.g., narrow roads, twisting, single lane with inadequate turning).
2. Load limits of local bridges are light or unknown; the bridges are narrow.
3. Winds are strong, and erratic fire behavior is occurring.
4. The area contains garages with closed, locked doors.
5. You have an inadequate water supply to attack the fire.
6. Structure windows are black or smoked over.
7. There are septic tanks and leach lines. These are found in most rural situations.
8. A house or structure is burning with puffing rather than steady smoke.
9. Inside and outside construction of structures is wood with shake shingle roofs.
10. Natural fuels occur within 9 m (30 ft) of the structures.
11. Known or suspected panicked individuals are in the vicinity.
12. Structure windows are bulging, and the roof has not been vented.
13. Additional fuels can be found in open crawl spaces beneath the structures.
14. Firefighting is taking place in or near chimney or canyon situations.
15. Elevated fuel or propane tanks are present.

These are also known as the "Wildland–Urban Watch Outs!"[280]

Taking Refuge in Vehicles, Buildings, and Protective Fire Shelters Used by Wildland Firefighters

The radiant energy of a fire, although highly intense at a given location, typically lasts for only a short time. Because radiant heat travels in straight lines, does not penetrate solid substances, and is easily reflected, seeking refuge in vehicles, buildings, or protective fire shelters should be viewed as a lifesaving solution.

Vehicles

Wildland firefighters have survived severe fire storms or the passage of fire fronts by taking refuge in vehicles.[227,234,293,294] Sadly, there are reported cases in which civilians would have survived or avoided serious injury had they remained in their vehicles.[193,290] Bereska[54] has indicated that he and two others were forced to drive a $^3/_4$-ton Dodge 4 × 4 truck at a moderate speed (25 to 30 km/h or 8 to 9 mph) through the flame front of a grass fire during a prescribed burning operation carried out in north-central Alberta in early April 1980. Winds at the time of the incident were light (less than 12 km/h [7 mph]) and the flames, although high (3 to 4 m [10 to 13 ft]), were not deep (1 to 2 m [3 to 7 ft]). He remembers "it got very hot instantly and as we came out of the flames we opened the windows and appreciated the cool fresh air."

The following three U.S. case histories serve as examples of intense burning situations in which lives were saved because people stayed inside vehicles while the fire front passed by their location[346]:

- In 1958, a veteran Field Section Fire Warden and two young men were fighting forest fires that burned in heavy fuels near the Bass River State Forest in southern New Jersey. A 90-degree wind shift transformed the flank fire into a broad head fire, with the advancing flames reaching up to 12 m in height (40 ft). The men entered their vehicle, a Dodge W300 Power Wagon, which stood in the middle of a 4-m (12-ft) wide sand road. Simultaneously, the engine and radio failed. The fire warden repeatedly admonished the crewmen, who wished to flee, to stay in the truck. Subsequently, the truck was rocked violently by convection currents and microclimatic changes generated by the flames. The men could neither see nor breathe because of smoke, and the cab began to fill with sparks that ignited the seat. The men stayed with the truck for only 3 or 4 minutes during passage of the head fire, but they indicated later that the interval involved seemed more like 3 or 4 hours. At the first opportunity, all of them left the vehicle on the upwind side and crouched beside it to escape the searing heat and burning seats. The warden proceeded to burn his hand severely while disposing of a flaming gas can located in the truck bed. Although the young men escaped virtually unscathed, the older man suffered lung damage and remained on limited duty for 5 years. He has since recovered completely and subsequently retired.
- In a 1962 California Division of Forestry fire in Fresno County, three men, followed by a flank fire that had turned into a head fire, raced back to their truck only a few feet ahead of the flames. The truck would not restart. After the main body of flames passed over the vehicle, the men jumped out in order to breathe because the truck was burning. Almost completely blinded by smoke and heat, they stumbled headlong into matted fuels, and two received first- and second-degree burns. One man was not burned but had to be treated for smoke inhalation. The truck was a loss.
- In 1976 a firefighter died while fighting a grass fire near the town of Buhler in Reno County, Kansas. A flashover occurred from buildup of gases on the lee side of a windbreak. A fire truck was caught in the flashover, and the firefighter working from the back of the vehicle ran and was killed. Although the truck burned, the driver was not seriously hurt.

Sitting in a vehicle during a passing fire front is often perilous, but when a person is trapped, it is almost certain doom to attempt escape by running from the fire. The preceding case

histories illustrate a few facts that, if remembered, may prevent panic-like reactions:

1. The engine may stall and not restart.
2. The vehicle may be rocked by convection currents.
3. Smoke and sparks may enter the cab.
4. The interior, engine, or tires may ignite. (Tires exploded during the Crank fire burnover in California in August 1987 involving several wildland fire engines.[269] This is obviously unsettling in an already stressful situation that could lead to panic-like actions.)
5. Temperatures increase inside the cab because heat is radiated through the windows.
6. Metal gas tanks and containers rarely explode.
7. If it is necessary to leave the cab after the fire has passed, keep the vehicle between you and the fire.

The type of vehicle determines the amount of protection afforded. Two travelers died in a fire in 1967 in Tasmania, Australia when they were caught in a canvas-topped vehicle.[221] A later fire in Australia led to further research on various vehicles' protection and the explosiveness of gasoline tanks. In 1969 near Lara, Victoria, Australia, a fast-moving grass fire crossed a four-lane expressway.[160,221] Several cars stopped in the confusion of smoke and flames. Seventeen people left the safety of their cars and perished. Six people stayed inside their vehicles and survived, even though one car ignited.

Investigations were carried out by the Forest Research Institute (now the Commonwealth Scientific and Industrial Research Organization [CSIRO], Division of Forestry and Forest Products) in Canberra, Australia to collect accurate data and dispel the misconceptions that make people flee a safe refuge if trapped by fire.[96] Cars were placed between two burning piles of logging slash to study a car's ability to shield against radiation.[221] The test was a hotter, longer-duration fire than would normally be encountered in a wildland setting.

Car bodies halved the external radiation transmitted at the peak of the fire, but a person inside would have suffered severe burns to bare skin. Although air temperatures inside the car did not reach hazardous levels until well after the peak radiation had passed, smoke from smoldering plastic and rubber materials would have caused discomfort and made the car uninhabitable. In this study, metal gasoline tanks did not explode, whether intact on cars or separated and placed on a burning pile of slash. Apparently, when tanks are sealed, the space above the liquid contains a mixture too deficient in oxygen vapor to support an explosion.

Cheney[96,98] offered the following advice for survival when in a car and trapped by fire:

1. If smoke obstructs visibility, turn on the headlights and drive to the side of the road away from the leading edge of the fire. Try to select an area of sparse vegetation offering the least combustible material.
2. Attempt to shield your body from radiant heat energy by rolling up the windows and covering up with floor mats or hiding beneath the dashboard. Cover as much skin as possible.
3. Stay in the vehicle as long as possible. Unruptured gas tanks rarely explode, and vehicles usually take several minutes to ignite.
4. Grass fires create about 30 seconds (maximum) of flame exposure, and chances for survival in a vehicle are good. Forest fires create higher-intensity flames lasting 3 to 4 minutes (maximum) and lowering chances for survival. Staying in a vehicle improves chances for surviving a forest fire. Remain calm.
5. A strong, acrid smell usually results from burning paint and plastic materials, caused by small quantities of hydrogen chloride released from breakdown of polyvinyl chloride. Hydrogen chloride is water soluble, and discomfort can be relieved by breathing through a damp cloth. Urine is mostly water and can be used in emergencies.

Buildings

The decision to evacuate a house or remain and defend it is not an easy one. Fire services generally prefer that residents evacuate the threatened area so that agencies can concentrate on protecting structures. Authorities also agree that evacuation of elderly, very young, infirm, and fearful people is usually a good idea.[205] People should evacuate only if it can be accomplished safely, well in advance of any danger. Several principles should guide the evacuation decision[403]:

1. A fire within sight or smell is a fire that endangers you.
2. More unattended houses burn down.
3. Evacuation when fire is close is too late; evacuation must be done well before danger is apparent.
4. More people are injured and killed in the open than in houses.
5. Learn beforehand about community refuges.
6. Evacuate only to a known safe refuge.

Whether people can find refuge in buildings depends on construction materials and amount of preparation in reducing fuels around the structure. If a home is constructed amid flammable vegetation, plans and procedures to safeguard the home and its occupants are essential. A building usually offers protection while the fire passes, even if it ignites later, because it shields against radiant heat and smoke. After the fire passes, it may be necessary to exit if the building is burning.

Numerous wildfire case histories from Australia since the 1939 Black Friday fires in Victoria[212] demonstrate that homes provide safe havens.[205,221,290,414] In 1967 in Tasmania, 21 people left their houses as fire approached. All died, and some were within a few meters of the buildings. Many houses did not burn and would have been refuges.

When taking refuge in a building, give people useful jobs, such as filling vessels with water, blocking cracks with wet blankets, and tightly closing windows and doors. If possible, assign lookouts to keep watch for spot fires on the outside of the building until the last minute. Before fire approaches the house, take the following precautions[346]:

1. If you plan to stay, evacuate your pets and livestock and all family members not essential to protecting the home well in advance of the fire's arrival.
2. Be properly dressed to survive the fire. Wear long pants and boots, and carry for protection a long-sleeved shirt or jacket made of cotton fabrics or wool. Synthetics should not be worn as they can ignite and melt. Wear a hat that can offer protection against radiation to the face, ears, and neck areas. Wear leather or natural fiber gloves and have a handkerchief handy to shield the face, water to wet it, and safety goggles, if possible.
3. Remove combustible items from around the house, including lawn and poolside furniture, umbrellas, and tarp coverings. If they catch fire, the added heat could ignite the house.

4. Ensure that anything that might be tossed around by strong fire-induced winds is secured (e.g., sheet metal, lumber, and plywood).
5. Ensure that the areas around any external propane tanks are fuel free for a considerable distance.
6. Close outside attic, eave, and basement vents to eliminate the possibility of sparks blowing into hidden areas within the house. Close window shutters.
7. Place large plastic trash cans or buckets around the outside of the house and fill them with water. Soak burlap sacks, small rugs, and large rags to use in beating out burning embers or small fires. Inside the house, fill bathtubs, sinks, and other containers with water. Toilet tanks and water heaters are an important water reservoir.
8. Place garden hoses so that they will reach any place on the house. Use the spray gun type of nozzle, adjusted to spray.
9. If you have portable gasoline-powered pumps to take water from a swimming pool or tank, make sure they are operating and in place.
10. Place a ladder against the roof of the house opposite the side of the approaching fire. If you have a combustible roof, wet it down or turn on any roof sprinklers. Turn on any special fire sprinklers installed to add protection. Do not waste water. Waste can drain the entire water system quickly.
11. Back your car into the garage and roll up the car windows. Disconnect the automatic garage door opener (otherwise, in case of power failure, you cannot remove the car). Close all garage doors.
12. Place valuable papers and mementos inside the car in the garage for quick departure, if necessary. In addition, place all pets in the car.
13. Close windows and doors to the house to prevent sparks from blowing inside. Close all doors inside the house to prevent drafts. Open the damper on any fireplace to help stabilize outside–inside pressure, but close the fireplace screen so that sparks will not ignite the room. Turn on a light in each room to make the house more visible in heavy smoke.
14. Turn off pilot lights.
15. If you have time, take down drapes and curtains. Close all Venetian blinds or noncombustible window coverings to reduce the amount of heat radiating into the house. This provides added safety in case the windows give way because of heat or wind.
16. As the fire front approaches, go inside the house. Stay calm; you are in control of the situation.
17. After the fire passes, check the roof immediately. Extinguish any sparks or embers. Then check the attic for hidden burning sparks. If you have a fire, enlist your neighbors to help fight it. For several hours after the fire, recheck for smoke and sparks throughout the house.

If a person expects to take refuge in his home and in turn survive to ensure its defense then it is critical that steps are taken to reduce the ignition potential of the structure as much as possible. Recent research on home ignition involving field experiments, coupled with modeling and observations of recent wildland–urban interface fire incidents, has provided valuable new insights into the means of ensuring home survival.[111,112,114] The research has shown that the characteristics of the home (e.g., exterior building materials) and in an area referred to as the "home ignition zone" located within 30 to 60 m (100 to 200 ft) of the home (e.g., surface fuel accumulations immediately adjacent to the structure that would be receptive to ignition by airborne firebrands) principally determines its ignition potential and thus its survivability during exposure to a wildland fire (Fig. 12-34); videos that summarize this research are available from *Firewise Communities*.[113,154,155] It is quite conceivable, for example, that a home could have little or no natural surface fuel within the immediate vicinity of the dwelling but because cedar shake shingles were used in the roof construction, it is vulnerable to ignition from airborne embers or firebrands from an approaching wildfire. If such a burning structure is not handled soon after ignition, it can contribute to the demise of neighboring houses if left unattended[253] or push the limits of fire protection to effectively keep up if too many structures become involved.[115]

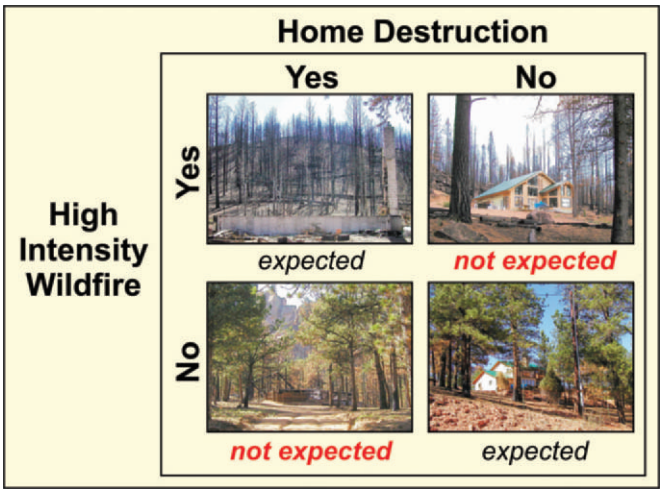


Figure 12-34. Expectations of home destruction as a result of exposure to a wildfire. Home survival is expected if low fire intensities occur (lower right cell) and unexpected if the home is destroyed (lower left cell). Conversely, home survival is not expected if high fire intensities occur (upper left cell) and unexpected if the home survives (upper right cell). (From Cohen JD: Home Destruction. In Graham RT, tech ed: Hayman Fire Case Study: Summary. General Technical Report RMRS-GTR-115. Ogden, UT, USDA Rocky Mountain Research Station, 2003.)

The use of external sprinklers systems on a home[121,283,352] should not be viewed as a panacea for a lack of fuel treatment in the home ignition zone and beyond or the use of flammable building materials. There is a perception that sprinklers should be capable of providing thermal protection from a high-intensity wildfire assault when in fact their real value is in preventing ignitions from airborne firebrands,[244] direct flame contact, and radiation. The Forest Engineering Research Institute of Canada (FERIC) has been engaged in a number of research studies related to the use of sprinkler systems for home and cabin protection since 2002, including outdoor field trials, using simulated structures, involving both prescribed and experimental fires (http://fire.feric.ca). Ray Ault,[37] the FERIC Wildland Fire Operations Research Group Supervisor, and his colleagues maintain that the greatest use of commercially available "Rain Bird" type sprinklers and small portable pumps for individual home structure protection is in applying water in sufficient quantities and locations to inhibit ignitions from airborne firebrands by an approaching or passing wildfire well in

advance. If the presoaking or application of water for wetting down fuels is applied for too long, it can lead to other problems (e.g., flooded basements).

Irrigation systems have also been occasionally applied to the task of wildland–urban interface protection. Other more elaborate systems have been devised. For example, the Saskatchewan Fire Management and Forest Protection Branch has successfully adapted and applied operationally a manure drag hose transportation system to the task of creating wet "breaks or belts" in values protection or to facilitate backfiring or burnout operations.[206]

Protective Fire Shelters Used by Wildland Firefighters

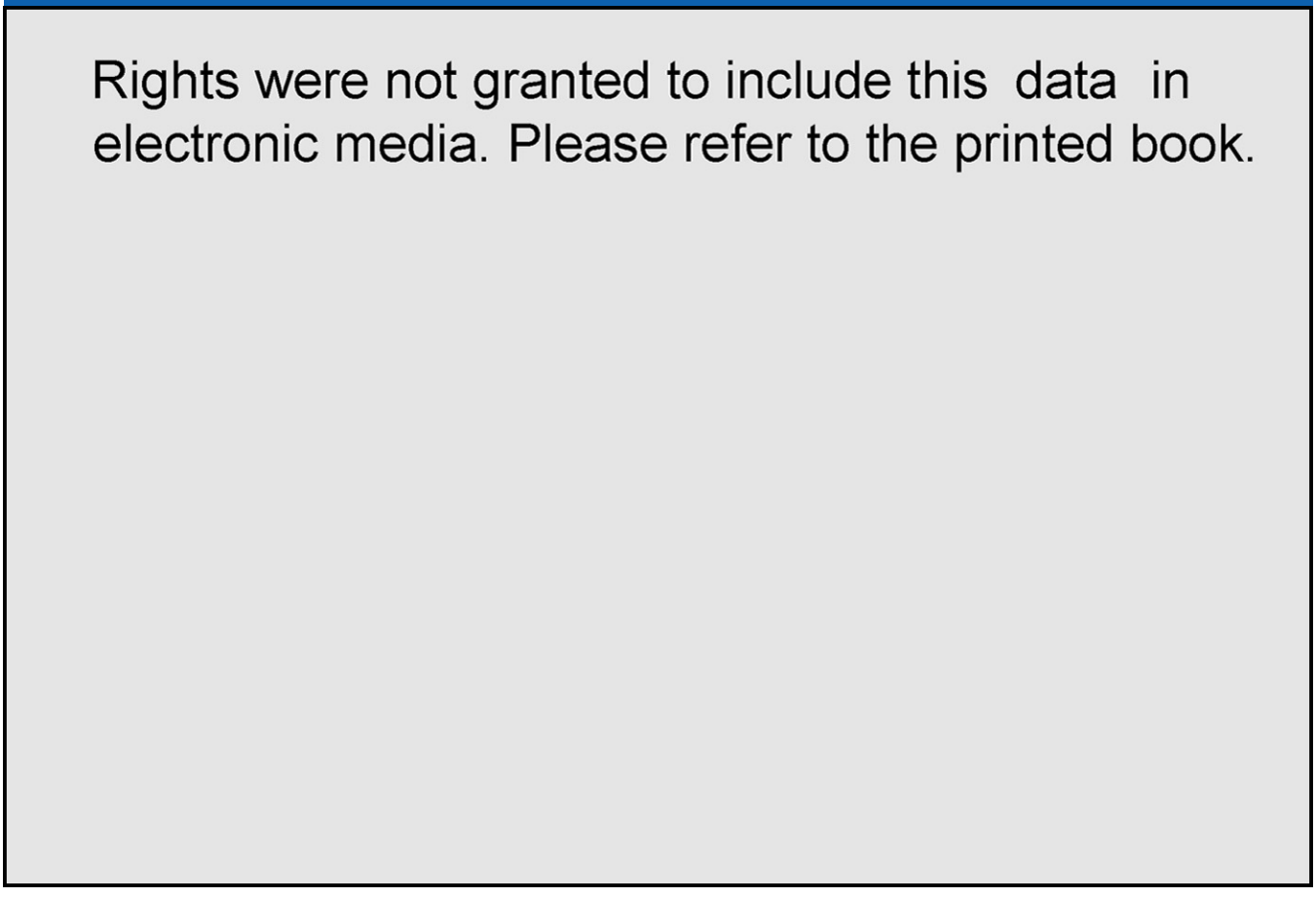
The concept of a protective fire shelter for wildland firefighters described earlier (see Figure 12-17) was originally conceived by Australian bushfire researchers in the late 1950s.[202,221] The USDA Forest Service equipment development center in Missoula, Montana (now the Missoula Technology and Development Center) initiated research and development of protective fire shelters around the same time as the Australians,[383] and this work has continued to this day[81,307] with a view to reducing the number of serious burn injuries and fatalities among firefighters who become trapped while fighting wildland fires.[33] Shelters designed in the shape of a "pup tent" protect the firefighter by reflecting radiant heat. Constructed of an aluminum-foil and fiberglass cloth laminate, the shelter reflects about 95% of the radiant heat emanating from a fire. These shelters were never designed for direct flame contact. Fire shelters were first introduced operationally on the wildland fire scene in the United States in the mid to late 1960s and became a required PPE item for federal wildland firefighters in 1977. More than 1100 U.S. wildland firefighters have deployed their fire shelters to date.[34] The fire shelter is credited with having saved the lives of more than 300 firefighters and preventing hundreds of serious burn injuries,[33] although it is not known with any degree of certainty how many would have actually survived and suffered no burn injuries without the fire shelter. Some personal accounts of fire shelter deployments (Box 12-6) have been published.[95,323]

Why protective fire shelters work well was demonstrated dramatically on August 29, 1985 when 73 firefighters were forced to take refuge in their shelters for about 1.5 hours while a severe crown fire burned over them.[8,262,331] The incident took place during the Butte fire in the Salmon National Forest in central Idaho. Observers described the crown fire that overran the firefighters as a standing wall of flame that reached about 60 m

Box 12-6. What Is It Like to Experience a Wildfire Entrapment in a Protective Fire Shelter?

Rights were not granted to include this data in electronic media. Please refer to the printed book.

Box 12-6. What Is It Like to Experience a Wildfire Entrapment in a Protective Fire Shelter?—cont'd



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(about 200 ft) above the treetops. Within the shelters, firefighters experienced extreme heat for as long as 10 minutes. Shelters were so hot that they could be handled only with gloves. After leaving the shelters, some firefighters showed symptoms of possible carbon monoxide poisoning, including vomiting, disorientation, and difficulty breathing. Emergency medical technicians administered oxygen to several individuals before evacuation from the site after the incident. Five firefighters were hospitalized overnight for heat exhaustion, smoke inhalation, and dehydration. The consensus of those interviewed was that without the shelters, none would have survived.[268]

It should be emphasized that the fire shelter was intended to be used as a "last resort" when it became impossible to escape to a safety zone before being overrun by a fire. However, despite the emphasis placed on this basic tenet and extensive training in entrapment avoidance over the years,[246,247,270,275,377] the results from a recent study[61] suggest a possible link to what has been suspected for many years—that some firefighters are likely to engage in behavior that neutralizes the gain in safety afforded by fire shelters. In other words, fire shelters can lead to additional risk taking[334] or what's called "risk homeostasis."[166]

Putnam[305] maintains that "you are always better with a fire shelter in an entrapment situation than not." That's fair enough advice, so it seems. Fire shelters are, after all, relatively light to carry—the current model weighs 1.9 to 2.2 kg (4.2 to 4.8 lbs) depending on the size[34,298]—and a minor inconvenience to pack around. Even when fire shelters become compromised, they still provide some protection. One of the entrapment survivors of the 1990 Dude fire, Dave LaTour deployed his fire shelter before the arrival of the fire front and stayed under his shelter even after a hole was kicked in it by one of the firefighters who got out of his shelter and attempted to evade the flames.[278] This allowed hot gases to enter his shelter. Depite this development, LaTour stayed down on the ground as flat as possible. He did sustain some burn injuries but fully recovered. LaTour was able to mentally concentrate his attention on his family to help him through the ordeal of being burned over by the passage of a high-intensity flame front.

One of the authors (MEA)[12] is of the opinion that the main issue regarding their use, besides not knowing what constitutes an adequate clearing size for a shelter deployment in a particular situation,[12,204] is the time taken for their efficient deployment.[53] In trying to avoid a wildland fire entrapment or burnover, seconds can mean the difference between life and death.[304] Without a regular, formalized training program, carried out under realistic environmental conditions, the precious time taken to deploy a shelter might be better spent attempting to evade the fire's encroachment on one's position

in order to reach a safer location.[12] The British Columbia Forest Service, the only Canadian fire management agency to use protective fire shelters, recently carried out a risk analysis[48] and decided to no longer have its firefighters carry fire shelters,[185] relying instead on situational awareness and entrapment avoidance.

Emergency Procedures During a Wildland Fire

Entrapment or Burnover

The danger of being entrapped or burned over and possibly killed or seriously injured by a wildfire is a very real threat for people living, working, or visiting[389] in rural areas subject to wildfires. Arnold "Smoke" Elser,[150] an accomplished Montana outfitter, described how he helped guests avoid entrapment by a forest fire in the mid-1960s:

> *The fire began at the bottom of the canyon and proceeded up canyon as fires do. However, the wind currents carried the smoke to the east and not up the drainage to the north; therefore, we received no warning of the fire. The Monture Creek trail goes through some very old mature timber which was not burning as we approached. As my stock, the guests, and I arrived at the fire site and realized that we were in danger, we felt we should fall back and try to flank the fire to the east. Starting back toward this trail, we found a ground fire that made it very hazardous to travel in this direction. Because of my knowledge of the trail and terrain, I knew that our best bet would be to wet down the stock, guests, saddles and outer clothing and try to break through the head of the fire. We successfully did this, receiving only a few minor burns on the horses and the loss of some apparel tied to the backs of the saddles. Some lessons that I learned in this experience were that in handling livestock in a fire situation you must have a very close, firm hand on them. It is also very important that no one panics or shows any excitement, as this alarms the livestock and begins the panic run that is so well known. I found that by talking in very low monotone, keeping the pack stock and saddle stock very close together (head to tail), and moving on a good trail, we were able to come through this fire with virtually no harm.*

Elser[150] had these additional suggestions for wildland recreationists:

> *Campers, whether they be livestock-oriented, hikers, or boaters, should know where to camp to provide adequate fire barriers around campsites. All campers should consider at least one, and preferably two, safe escape routes and havens (such as rock piles, rivers, and large green meadows) away from heavy fuel areas. Campers should be alert to canyon air current conditions in critical fire seasons. The safety of many recreationists is threatened by nylon and other synthetic fabrics used in the manufacture of most backpacking equipment. These materials melt upon contact with heat. The very nature of good horse packing equipment is a deterrent to fire; canvas mantles that cover the gear and the canvas pack saddles are easily wet down. Leather items such as chaps, good saddle bags, and western hats [that] can shield against heat blasts all provide important protection for the horse user.*

Sometimes there may be no chance to easily escape an approaching wildfire. Injuries can be minimized or avoided and possible death adverted by adhering to certain fundamental principles and procedures (Box 12-7). There are, however, four simple concepts that one must try to adhere to at all times[36,96,103,403]:

- Select an area that won't burn—the bigger the better—or, failing that, with the least amount of combustible material, and one that offers the best microclimate (e.g., depression in the ground).
- Use every means possible to protect yourself from radiant and convective heat emitted by the flames (e.g., boulders, rock outcrops, large downed logs, trees, snags).
- Protect your airways from heat at all costs and try to minimize smoke exposure.
- Try to remain as calm as possible.

The first requirement will limit the flame dimensions and in turn the potential heat energy from flame radiation. It will also limit the time of exposure, an important factor in thermal injuries. Radiant heat can kill you long before direct flame contact.[122,403] The more exposed skin, the greater the likelihood of death.

Obviously, the last requirement—to remain as calm as possible—may seem difficult to establish and maintain. The expectation that people will panic (i.e., a sudden uncontrollable fear or alarm leading to unthinking behavior) during an emergency situation such as wildfire entrapment or burnover is very strong. Admittedly, with the benefit of 20/20 hindsight, it is easy to point to some decisions that were not optimal and played a negative role in the outcome of the fire.[289] Structural fire researchers feel that most people faced with a fire situation react in a rational manner considering the ambiguity of the initial cues about the fire, their limited knowledge about fire development and fire dynamics, and the restricted time to make a decision and to take action.[351]

Panic is viewed as being synonymous with a frightened, scared, nervous, or anxious response.[351] In actual fact, panic in the form of irrational or crazed behavior (e.g., aimlessly trying to flee) is rare during fires. Social scientists long ago rejected this concept to explain human behavior in urban fires.[108,302,351] Instead of panic, what is commonly observed is an increased level of stress. Stress is not panic. As Dr. Guylene Proulx, a human factors specialist with the National Research Council Canada's structural fire research program, suggests, "This stress is not an abnormal reaction or a negative response; on the contrary, stress is regarded as a necessary state to motivate reaction and action" and that "Decision-making under stress is often characterized by a narrowing of attention and focusing on a reduced number of options. This explains why training is so important since the person is unlikely to develop new solutions under heightened stress; a well-run decision plan learned and practiced beforehand is easier to apply under stress."

There are four fundamental or basic survival techniques or options (see Box 12-7) available to an individual who is caught out in the "open" and is likely to be entrapped or burned over by a wildfire and is not able to take refuge in a vehicle or building[160,221,403] or have a protective fire shelter. These four survival options are:

- Retreat from the fire and reach a safe haven.
- Burn out a safety area.
- Hunker in place.
- Pass through the fire edge into the burned-out area.

Box 12-7. Surviving a Wildland Fire Entrapment or Burnover

When entrapment or burnover by a wildland fire appears imminent, injuries or death may be avoided by following these basic emergency survival principles and procedures.

- **Acknowledge the stress you are feeling.** Most people are afraid when trapped by fire. Accept this fear as natural, so that clear thinking and intelligent decisions are possible. If fear overwhelms you, judgment is seriously impaired, and survival becomes more a matter of chance than good decision making.
- **Protect yourself against radiation at all costs.** Many victims of forest fires actually die before the flames reach them. Radiated heat quickly causes heatstroke, a state of complete exhaustion. Find shielding to reduce heat rays quickly in an area that will not burn, such as a shallow trench, crevice, large rock, running stream, large pond, vehicle, building, or the shore water of a lake. Do not seek refuge in an elevated water tank. Avoid wells and caves because oxygen may be used up quickly in these restricted places; consider them a last resort. To protect against radiation, cover the head and other exposed skin with clothing or dirt.
- **Regulate your breathing.** Avoid inhaling dense smoke (which can impair both your judgment and eyesight). Keep your face near the ground, where there is usually less smoke. Hold a dampened handkerchief over the nose. Match your breathing with the availability of relatively fresh air. If there is a possibility of breathing superheated air, place a dry, not moist, cloth over the mouth. The lungs can withstand dry heat better than moist heat.
- **Do not run blindly or needlessly.** Unless a clear path of escape is indicated, do not run. Move downhill and away from the flank of the fire at a 45-degree angle where possible. Conserve your strength. If you become exhausted, you are much more prone to heatstroke and may easily overlook a place of safe refuge.
- **Burn out fuels to create a safety zone if possible.** If you are in dead grass or low shrub fuels and the approaching flames are too high to run through, burn out as large an area as possible between you and the fire edge. Step into the burned area and cover as much of your exposed skin as possible. This requires time for fuels to be consumed and may not be effective as a last-ditch effort, nor does this work well in an intense forest fire.
- **Lie prone on the ground.** In a critical situation, lie face-down in an area that will not burn. Your chance of survival if the fire overtakes you is greater in this position than standing upright or kneeling.
- **Enter the burned area whenever and wherever possible.** Particularly in grass, low shrubs, or other low fuels, do not delay if escape means passing through the flame front into the burned area. Move aggressively and parallel to the advancing fire front. Choose a place on the fire's edge where flames are less than 1 m (3 feet) deep and can be seen through clearly, and where the fuel supply behind the fire has been mostly consumed. Cover exposed skin and take several breaths, then move through the flame front as quickly as possible. If necessary, drop to the ground under the smoke for improved visibility and to obtain fresh air.

These four survival options are presented in no particular order of priority. Such factors as the size of the fire, the fire environment, the size and location of safety areas or zones, the prevailing fire behavior, and the location of the person with respect to the head of fire will ultimately dictate which option or options (should the first one of these options selected become compromised) should be selected. Each of these options has its own unique advantages and disadvantages.

Survival Option 1: Retreat from the Fire and Reach a Safe Haven

When people are under pressure, they fall back on habitual, first-learned, and overlearned responses.[406] The natural tendency when a person is threatened by a hazard such as a wildfire is to try and move away from the danger as quickly as possible to a place of safe refuge. If the distance between the fire and safe area is short, the fire's advance slow, the path to the safe area easily traversed, and the person able bodied, then selection of this survival option is appropriate. Bear in mind that a safe refuge may be very near by, so one should not avoid the most obvious place even though it may temporarily be uncomfortable to reach owing to smoke or low to moderate radiation levels, for example. In some cases, this might mean just taking a few steps into the "black" or recently burned-over ground. The question of whether a previously burned area will serve as a safety zone will depend not only on the size of the area but also on the degree of completeness or fuel consumption, both vertically and horizontally.[78,297] Furthermore, a recently burned-over area may not immediately serve as a safe refuge owing to the burnout time of woody fuels and duff,[368] in contrast to grassland fuels in which a person can enter the recently burned area in a matter of a few seconds.[106]

There are a good many cases in which firefighters have escaped injury and death by being able to outpace or outmaneuver a spreading fire.[297,377] There have also been many well-publicized cases or incidents during which attempting to initially out pace and then ultimately outrun an advancing flame front have ended in tragedy, including several incidents already mentioned in this chapter (i.e., 1938 Pepper Hill fire, 1949 Mann Gulch fire, 1953 Rattlesnake fire, 1956 Inaja fire, 1966 Loop fire, 1968 Canyon fire, and 1994 South Canyon fire). Sadly, there are many other examples,[324] one of the most recent being the 2003 Cramer fire in central Idaho involving two firefighter fatalities.[110] Dr. Ted Putnam,[306] a retired wildland fire safety specialist with the USDA Forest Service, considers that many of the above-noted fatality fires were in fact escapable had better decision making been employed (e.g., dropped tools and packs earlier on to maximize rate of advance, put the fastest pace setters at the lead, and used fire shelters as shields against radiant and convective heat).

Similar incidents involving civilians have also taken place. For example, on November 30, 1957, four members of a group of nine young hikers perished while trying to outrun a bushfire in the Blue Mountains of New South Wales, Australia, as it

advanced upslope.[160,221] Another incident occurred on August 26, 1995, near the community of São Domingos in the district of Sandtarém, Portugal.[396] Three civilians who had been assisting local firefighters in suppression operations eventually ended up being killed while trying to run ahead of the fire when it blew up. A fourth individual received severe burns to his feet, which eventually led to them being amputated, and he died some months later.

Simulations carried out by the FERIC Wildland Fire Operations Research Group based on their research on firefighter travel rates,[133] coupled with case study information gleaned from the 1949 Mann Gulch and 1994 South Canyon fires,[79] clearly indicate that a person is not able to sustain a maximum pace for even a relatively short period of time without being overrun by a rapidly advanced flame front, even on a moderately steep slope.[21,45] For example, a fire spreading at 60 m/min (197 ft/min) up a 26% slope would, depending on the fuel type, overrun someone in about 6 to 7.5 minutes or after about 360 to 460 m (about 1200 to 1500 ft) once the "race" had started. Chandler and colleagues[93] state, "In most firefighter fatalities . . . the unsuccessful strategy has been to try and run away from the fire and continue running until exhaustion or the radiant heat load from the fire front fells the victim and allows the flame front to pass over him or her." Thus, trying to outpace a fire for any significant distance, but especially uphill, is "courting disaster."[221] For this reason, escape routes involving travel upslope should generally not be selected.

Survival Option 2: Burn Out a Safety Area

Burning out fuels to create an area of safety or to enlarge an existing burned area is a viable survival technique or option in some situations (e.g., light fuels and having sufficient time to implement). As Australian bushfire research pioneers Harry Luke and Alan McArthur[221] have noted, "Carrying a box of matches is part of survival planning" (in this regard, windproof-type matches would be ideal); most wildland firefighters carry fuses, which would infinitely more reliable and effective than a match. Undoubtedly the most publicized example of this survival strategy being used in modern times occurred on the 1949 Mann Gulch fire, mentioned at the start of this chapter.[226,328] However, the technique was known to have been used by American Indians in the early 1800s[308,384] and undoubtedly by aboriginals in other parts of the world as depicted, for example, in the 1989 movie, *The Gods Must Be Crazy II*. Wag Dodge described his escape fire during his testimony at the board of review investigation into the 1949 Mann Gulch fire as follows:

> *After setting a clump of bunch grass on fire, I made an attempt to start another one, but the match had gone out and upon looking up, I had an area of 100 feet square that was ablaze. I told the man nearest to me that we would wait a few seconds to give it a chance to burn out inside, and then we would cross through the flames into the burned area, where we could make a good stand and our chances of survival were more than even.*

Interestingly, Dodge's statement was printed in the letters to the editors section of the September 1949 issue of *Life* magazine following publication of an article on the Mann Gulch fire entitled "Smokejumpers Suffer Ordeal by Fire" from the previous issue. In that letter, the reader stated, "I am sure that there are many people throughout the country who would appreciate and perhaps benefit sometime by a more detailed account of how Foreman Wagner Dodge, who kept calm and did not become panic-stricken, saved himself."

Survival Option 3: Hunker in Place

When caught in the open, survival may depend on taking advantage of every possible source of cover or protection from radiant and convective heat (e.g., depressions in the ground, large rocks or logs).[160] If a cave (see Figure 12-32) or root cellar[417] is used to take refuge in, it is important to vacate into the open at the earliest opportunity owing to potential problems associated with accumulations of smoke and carbon monoxide.

In selecting this option, the importance of staying as flat as possible with one's nose and mouth pressed down into the ground cannot be overemphasized. Lying prone minimizes one's radiation profile, and cooler, denser air will always be present at ground level. In selecting this option, bear in mind the following advice[103]:

> *When a fire passes over a point, the air temperature near the ground is higher than the air above it and remains higher for longer. So if someone is sheltering from radiation at ground level, they need to stand up as soon as possible after the fire passes to breathe cool, fresh air. This is most apparent in grass fires where air at ground level is hot and smoky for several minutes whereas at 2 m [6.6 feet] it is cool and breathable within 10 to 15 seconds of the flames passing.*

Although there have been reported cases of firefighters surviving on large rockslides during a wildfire entrapment or burnover, most notably two smokejumpers on the 1949 Mann Gulch fire,[226,328] there have also been instances in which these apparently safe, fuel-free areas contained enough combustible materials to cause injury or death (e.g., Shephard Mountain fire in western Montana on September 4, 1996). Four firefighters died on a rockslide during the 2001 Thirtymile fire in north-central Washington, due in part to the accumulation of duff and rotting wood lodged in the rock crevices that ignited from airborne firebrands.[66,388]

In selecting this option, maximum use should be made of any clothing or other readily nonburnable material to protect exposed skin. You may have to improvise.[385] *Again, it must be emphasized that synthetics, including undergarments, should not be used.* During the 1983 Ash Wednesday fires in Victoria, Australia, two individuals wearing only summer clothing who covered themselves with a synthetic blanket perished, while two other individuals right next to the two victims covered themselves with a wet woolen blanket and survived the burnover.[205]

A good example of using this option was the Wandilo fire that occurred in an exotic pine plantation near Mount Gambier, South Australia on April 5, 1958. Eleven firefighters found themselves trapped on a narrow firebreak during a "blowup."[221,234] Eight in the group attempted to run back along the firebreak, but perished after exposure to extreme radiant heat levels and direct flame contact. Of the three who survived, two remained in the cab of their firefighting truck to shelter from the worst of the firestorm and only left this cover when the vehicle was well alight and the fire's peak intensity had abated. The remaining survivor sheltered in a deep wheel rut in the soft sand of the firebreak with his coat over his face during the peak period of extreme fire behavior.

Taking refuge in a natural water body such as a pond, lake, or river must be done with caution, but for other reasons (e.g., swimming ability). For example, in 1986, three firefighters in the province of Quebec, Canada drowned as a result of being forced to enter a lake with a steep drop-off when their camp location was overrun by fire.[14] The risk for hypothermia must also be considered.[80] Dion[140] described an incident that occurred in west-central Saskatchewan, Canada in May 1919 in which 11 Cree Indians perished because they were not able to reach a nearby lake or find deep enough water to avoid radiation burns. Of the 12 who survived the ordeal, 11 "bore the marks of their burns for life." One adult member of the group "escaped severe burns by staying under the water" as much as was possible.

Survival Option 4: Pass Through the Fire Edge into the Burned-Out Area

Luke and McArthur[221] have suggested that "running through flames cannot be generally recommended and should certainly not be attempted when flames are more than 1.5 m [5 ft] in height or depth." Nevertheless, a number of firefighters have done this very thing and survived (e.g., 1991 Tikokino grass fire in New Zealand[319]). In fact, the five members of the group of young hikers that survived the 1957 bushfire in the Blue Mountains of New South Wales mentioned earlier on did so successfully but suffered considerable discomfort. A similar incident occurred on the Warm Springs Indian Reservation in central Oregon in June 1985. Wildland fire behavior analyst Jim Roessler[325] has indicated that those individuals who tried to outrun a grass fire on a moderately steep slope were killed, whereas at least one person who passed through the flame front survived. In contrast, during the blowup of the 1937 Blackwater fire, the "five horsemen" made the decision to move downhill through the advancing flames.[62] Three didn't make it, no doubt due in part to the heavy fuel conditions. Of the two that did survive, one died later of burn injuries. A group of 41 on another section of the fire rode out the blowup in a ridgeline clearing as the fire progressed upslope; 3 were badly burned and eventually died of their injuries.

It has been suggested that a person could theoretically survive passing through flames 3 m (9.8 feet) high and 37 m (121 feet) in depth and still survive.[93] It is presumed that the person would be immersed in flames for less than 7.5 seconds and would require ideal running conditions (e.g., good footing, no obstructions) and be properly clothed to withstand the direct flame contact. Although it is reasonable to expect a person to be able to hold his or her breath for this long, the very notion of attempting such a drastic or draconian [93] feat seems unimaginable. Nevertheless, it is worth noting that firefighters have lived, albeit while suffering severe burns as result, by running through high-intensity flame fronts. One of the most notable examples of this involved a prescribed fire (PB-3/79) in heavy logging slash near Geraldton, Ontario, Canada on August 22, 1979.[29,238,257] As a result of a complex set of unforeseen circumstances, eight members of the firing crew found themselves encircled by fire. One member of the party, a local fire technician, realizing that there was no other option except to run through the advancing flame front or face what appeared to be certain death, tried to get seven seasonal employees to follow him. They failed to heed his urgings and were eventually engulfed by the fire, whereas he survived but did suffer serious burn injuries. This outcome is hauntingly reminiscent of the 1949 Mann Gulch fire and Wag Dodge's escape fire in which 15 firefighters failed to follow his lead and 13 ended up perishing. In this regard, Dr. Karl Weick,[407] a renowned professor of organizational behavior and psychology, points out that there is good evidence to support the notion that when people are threatened, their thinking becomes much more rigid and difficult to change. Furthermore, they tend to seek out and talk to only those who are most familiar to them.[356]

The survival technique or option of moving through the flame front to previously burned ground would logically be most suitable in light, discontinuous fuel types that fail to produce deep, uniform flame fronts, and significant postfrontal smoldering or isolated flaming, such as afforded by certain grasslands (e.g., heavily grazed areas). Furthermore, if one has the good fortune to take advantage of a lull in the wind, this would lessen the momentary rate of spread and in turn the flame depth (see Table 12-2). An area along the flanks of the fire (see Figure 12-21) would be preferable to the head. One may only have to travel a relatively short distance before reaching previously burned ground that has "cooled" down sufficiently to serve as a safe area.

Having reached the burned area, a person would still have to be cognizant of the danger posed by fire-weakened trees and falling snags, hot ash pits and burned-out stump holes, and rolling rocks or logs.[217] This would apply to the other survival options as well.

It is worth emphasizing that wildland firefighters as well as members of the general public have been killed and seriously burned while engaged in using fire as a land management tool.[29,242,358,395,398] Thus, the survival principles and options discussed earlier are equally applicable to prescribed fires or controlled burns.

Although radiant heat emanating from a wall of flame is generally viewed as the principal killer in wildland fire entrapments and burnovers,[122] under certain conditions convective gases or superheated air can have lethal consequences with little or no warning, most notably upslope of a fire source. Under strong winds, a fire's convection column will not lift away from the surface of a steep slope.[106] As slope steepness increases, flames gradually "attach" themselves (Fig. 12-35), thereby enhancing fire spread by both direct flame contact and convective heat transfer.[326] This is the very reason that fires spread faster upslope than on level ground given the same fuel and weather conditions.

WILDLAND FIRES AND HUMAN BEHAVIOR

As discussed earlier, the reality of human behavior in fires is somewhat different from the panic scenario "nourished by the media and movie industry who like to play on strong emotional images."[302] What is regularly observed in urban fire situations is a lethargic response to fire alarms or even the initial cues of a fire (e.g., smoke and visible flame).

What has been most troubling to the wildland fire community in recent years is the fact that the same mistakes seem to be made time and time again,[141] rather than using "the lessons of the past so we don't have to keep relearning them the hard way."[276] Since the mid-1990s, there has been a growing recognition among the wildland fire community of the role that human factors play in firefighter fatalities.[74,142,304]

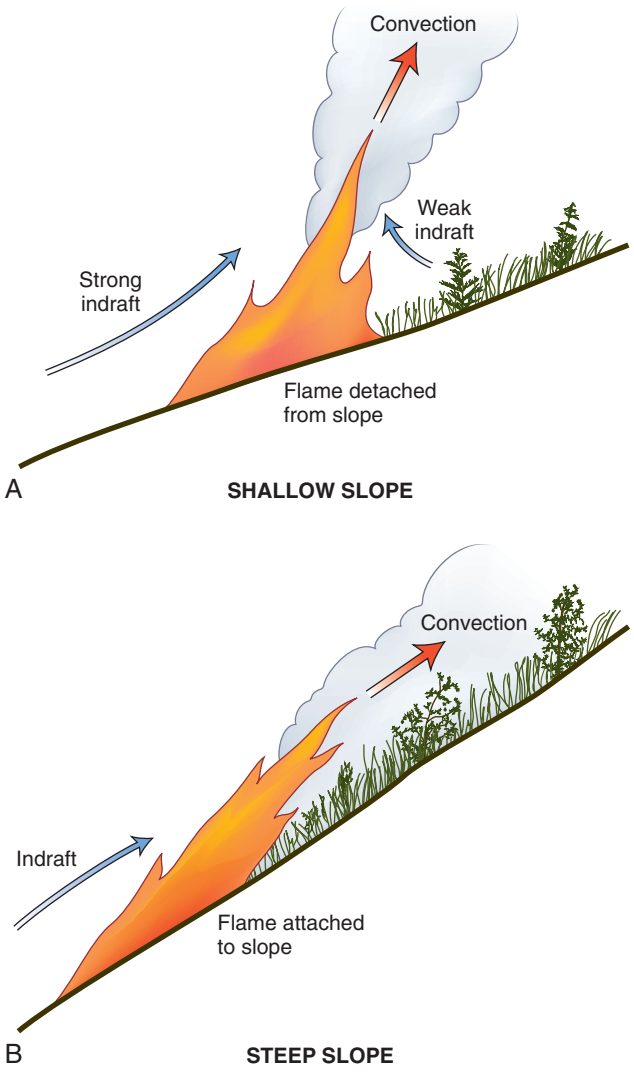

Figure 12-35. Effect of slope steepness on degree of flame attachment and convective heat transfer for shallow sloping **(A)** and steep sloping **(B)** terrain. (Redrawn from Rothermel RC: Fire behavior considerations of aerial ignition. In Mutch RW (tech coord): Prescribed Fire by Aerial Ignition. Proceedings of a Workshop, 1985.)

Morse[250] considered the development of a mental "scotoma" among firefighters to be a major contributing factor in many wildland fire fatality incidents, noting, "Scotoma is, literally, a blind spot. In a psychological sense, it is that condition which occurs when a person tends to block out from his or her consciousness anything considered not important—or critical—to survival." In other words, blindness to danger perceived as routine takes hold and blocks out sensitivity to hazardous events or conditions present in the wildland fire environment.

The smokejumpers involved in the 1949 Mann Gulch fire would appear to have suffered from a psychological scotoma or overconfidence.[405] By the time they had landed and gathered their gear together, it was nearly 5:00 PM. At the time, "They did not feel the fire threatened them then."[328] Less than an hour later, 12 smokejumpers and a local firefighter were overrun and killed by the fire. Air temperatures reached a maximum of 36° C (97° F) on the day in question, and in turn, the fully cured grasses were in a critically dry state.[328] It was a day of extreme fire danger[16] One of the survivors, Bob Sallee, commented afterwards[328]:

> *I took a look at the fire and decided it wasn't bad. It was burning on top of the ridge and I thought it would continue on up the ridge. I thought it probably wouldn't burn much more than night because it was the end of the burning period (for that day) and it looked like it would have to burn down across a little saddle before it went uphill any more.*

One crew member was reported to have been taking photographs of the fire less than 10 minutes before the first fatalities occurring.[226,328]

Even on the major run of the Thirtymile fire in north-central Washington on July 10, 2001,[66] the firefighters behaved more as spectators than as potential victims until it was too late for some. A total of 16 people (14 firefighters and 2 civilians) were entrapped and required to deploy protective fire shelters. Four firefighters were killed, and 2 were injured, 1 with severe burns. Earlier on during the day in question, the feeling was that the fire was "basically a mop-up show."[66] Some of the firefighters who were overrun by the fire "spent time taking photos or making journal entries" just a half hour before the incident.[66]

Although wildland firefighters may be subject to a whole host of human failings or other psychological phenomena,[110,406] openly acknowledging and constantly being aware of the potential for entrapment or a burnover, regardless of the situation, is the initial key step in reducing the risk for injury or death from wildland fires. Complacency may be the most difficult factor to overcome.[255] The late Paul Gleason, a veteran wildland firefighter and accomplished rock climber, noted that three major causes of climbing accidents are ignorance, casualness, and distractions.[168]

Although firefighters are expected to engage a wildfire, civilians are not. Nevertheless, the general public could also become complacent about wildfires and fail to fear the worst, until, unfortunately, it's too late. This may be due in part to a fascination with fire in general (Fig. 12-36), denial, indifference, or a complete ignorance about free-burning fire behavior. In any case, there is a perception that there is no real sense of urgency until the threat is imminent. As Proulx points out,[302] "People are often too cool during fires, ignoring or delaying their response to the initial cues of an actual emergency. Once occupants decide that the situation requires moving to an area of safety, the time left could be minimal." This is probably best exemplified by an urban fire case study. On May 11, 1985, 56 people burned to death and more than 200 were injured when a rapidly growing fire, caused by a cigarette, engulfed the wooden main grandstand at Valley Parade Stadium in Bradford, England, during a soccer match. Filmed documentation of the fire indicates that the blaze spread the entire 88-m (290-ft) length of the stand in less than 5 minutes—a rate of 18 m/min (58 ft/min). Although people in the immediate vicinity of the developing fire moved toward exits, "Persons in remote sections of the grandstand continued to watch the match, apparently unconcerned about (or perhaps unaware of) the developing fire."[203] One of the seven factors that contributed to the loss of life was "The failure of patrons to perceive the danger of the developing fire in the early stages and begin evacuation."[203]

A

B

Figure 12-36. Rural homeowners **(A)** observing the Haeckel Hill fire **(B)** near the city of Whitehorse, Yukon Territory, Canada, on June 22, 1991. (Courtesy Yukon Wildland Fire Management.)

A

B

Figure 12-37. Fires in fully cured grasslands are very responsive and can attain exceedingly rapid rates of spread. The flank of a grass fire, with a change in wind direction, can quite quickly become the head and catch the unwary off guard. This scene from the multifire situation that occurred in the Spokane, Washington, area on October 16, 1991 **(A)**, reinforces the importance of having well-established escape routes that can be easily navigated **(B)** to a designated safety zone. (Courtesy Spokane Statesman Review/WorldPictureNews. Photos by K. King.)

It is hard sometimes to believe that a wildland fire could become life-threatening, especially if it is far away from the observer or small in size and isn't burning very vigorously at the time. However, danger is inherent in every wildland fire occurrence, be it a wildfire or a prescribed fire. Some wildland fire situations are simply more hazardous than others and can catch a person or persons off guard (Fig. 12-37), possibly leading to severe injuries or even death.[43,71]

Tom Leuschen[216] is an experienced fire manager and wildland fire behavior analyst who worked for many years in north-central Washington, within sight of the area struck in 2001 by the Thirtymile fire. He was involved in the accident investigation and had the following to say about the incident:

> *When I stand at the deployment site and look at their pictures, knowing what I know about the fuels, barriers, and interactions between fires, I honestly do not expect that they will have to shelter either. I am as wrong as they were. Things got very complex as the fires joined at their location. The obvious lesson is that we must prepare for the worst when trapped, regardless of what we may anticipate. They were entrapped at 1634 and deployed at 1724. They had time to get organized.*

Anyone involved in a wildland fire can become a victim of an entrapment or burnover at any time given the precarious nature of wildland fires and their environmental influences. People need to be constantly mindful or conscious of this fact because, as Weick[404,406] says, "safety is not bankable" and it's "an ongoing struggle for alertness." Plan for and expect the worst every time out.

Proper Clothing

There are documented cases in which homeowners, who were seemingly prepared to stay and defend their property, have died because they were not properly dressed to do so (e.g., clad only in shorts or a bathing suit and slippers).[403] Clothing protects against radiant heat, embers, and sparks, so it is sensible to dress appropriately.[121,290] The cover of a recent book on the wildland–urban interface fire situation in southern California[182]

Figure 12-38. An inappropriately clad and positioned homeowner attempting to defend his property from ember attack during a wildfire in Rancho Penasquitos, San Diego, California, on August 25, 1995. The fire at the moment appears largely dominated or supported by a plentiful fuel source. However, given the capricious nature of fire and winds, this fire could have easily turned into a more wind-dominated event at any time. Thus, this individual could have been incapacitated by a momentary blast of superheated air from the convection or radiant heat produced by the spreading fire. In the absence of any outside assistance, this would then have left him vulnerable to burning by direct flame contact along with his home. Fortunately for this individual, shortly after the photo was taken, an air tanker dropped a load of fire retardant near the advancing flames and effectively nullified the fire's spread and intensity. (Courtesy California Chaparral Field Institute. Photo by R.W. Halsey.)

depicts a lightly clad homeowner on the roof of his house presumably attempting to extinguish sparks or embers with a garden hose as a high-intensity flame front approaches (Fig. 12-38). This is a potential recipe for disaster in many respects. This person is not properly attired to stay and defend his property, nor is this the correct approach.

Closely woven material is more resistant to radiation and less likely to ignite than is open-weave material. Natural fibers are best. Wool is more flame resistant than cotton, although cotton can be improved by chemical treatment to retard flammability. As mentioned earlier, synthetic materials are a poor choice because they readily absorb heat or, worse yet, can ignite and melt onto the skin.

Closely woven materials that provide protection also restrict airflow, so clothes should fit loosely so that they do not interfere with dissipation of body heat. Cotton long johns or undergarments absorb sweat, aid evaporation, and do not melt; however, underwear made of synthetic materials does. Wearing excessive layers of clothing generally contributes to heat stress.

As little skin as possible should be exposed to fire. Long trousers and a long-sleeved shirt should be worn. For maximum protection, the shirt should be kept buttoned with sleeves rolled down.

Brightly colored (yellow or orange) coveralls or shirts are worn by organized firefighting crews. These colors improve safety and communications because they are visible in smoke, vegetation, and blackened landscapes.

Other essential apparel includes a safety helmet (hard hat), gloves (leather or natural fiber), leather work boots, woolen or cotton socks, a warm jacket for night wear, goggles, and a handkerchief. Clothing, backpacks, tents, and other camping equipment made of synthetics should be discarded when a person is close to a fire.

Water Intake

Sweating is the primary method for body cooling, although exhaling warm air and inhaling cooler air also decreases body temperature (see Chapter 64). Because the cooling effect of sweat evaporation is essential for thermoregulation, fluids lost during strenuous work must be replaced. In firefighting, water losses of 0.5 L/hr (1 pint/hr) are common, with losses of up to 2 L/hr (2 quarts/hr) under extreme conditions.[221] Unless water is restored regularly, dehydration may contribute to heat stress disorders, reluctance to work, irritability, poor judgment, and impatience.

Thirst is not a good indicator of water requirements during strenuous work, so additional drinking of small quantities of water at regular intervals is recommended. A useful signal of dehydration is dark, scanty urine.

An excessive amount of electrolytes may be lost through sweating, leading to nausea, vomiting, and muscle cramps. When meals are missed or unseasoned foods are eaten, electrolyte supplements may be needed to replace lost salts. Sweetened drinks should be used as a source of energy if solid food is not available.

Personal Gear

Some rescue and medical missions take a few days. Therefore, it is necessary to be prepared for extended periods and changing conditions in the backcountry (Box 12-8).

Box 12-8. Personal Gear for a Rescue Mission on a Wildland Fire Incident

1. Boots (leather, high-top, lace-up, nonslip soles, extra leather laces)
2. Socks (cotton or wool, at least two pairs)
3. Pants (natural fiber, flameproof, loose fitting, hems lower than boot tops)
4. Belt or suspenders
5. Shirt (natural fiber, flameproof, loose fitting, long sleeves)
6. Gloves (natural fiber or leather, extra pair)
7. Hat (hard hat and possibly a bandanna, stocking cap, or felt hat)
8. Jacket
9. Handkerchiefs or scarves
10. Goggles
11. Sleeping bag and ground cover
12. Map
13. Protective fire shelter
14. Food
15. Canteen
16. Radio (AM radio will receive better in rough terrain; FM is more line-of-sight; emergency personnel should have a two-way radio)
17. Bolt cutters (carried in vehicles to get through locked gates during escape from flare-ups or in the rescue of trapped people)
18. Miscellaneous items (mess kit, compass, flashlight, extra batteries, toilet paper, pencil, notepaper, flagging tape, flares, matches (windproof), can opener, wash cloth, toiletries, insect repellent, plastic bags, knife, first aid kit, and lip balm)

How to Report a Wildland Fire to Local Fire Protection Authorities

A caller should be prepared to provide the following information when reporting a fire:

1. Name of person giving the report.
2. Where the person can be reached immediately.
3. Where the person was at the time the fire was discovered.
4. Location of the fire; orient the fire to prominent landmarks, such as roads, creeks, and mileposts on highways.
5. Description of the fire: color and volume of the smoke, estimated size, and flame characteristics if visible.
6. Whether anyone is fighting the fire at the time of the call.

The phone number to call varies locally. For example in Alberta, Canada, the number for reporting wildland fires is 310-FIRE or 310-3473 in both the northern and southern halves of the province, even though they have different area codes. In other locations, it may simply be 9-1-1. Rural homeowners should make a point of finding out the number to call before the beginning of fire season.

Portable Fire Extinguishers

People should know which extinguisher to select for a specific hazard in the home or recreational vehicle and be trained in its use. When a fire is discovered, first evacuate occupants to safety and promptly report the fire to appropriate authorities. If the fire is small and poses no direct threat, an extinguisher should be used to fight it.

The three major classes of fires are as follows:

Class A fires: fueled by ordinary combustible materials, such as wood, paper, cloth, upholstery, and many plastics

Class B fires: fueled by flammable liquids and gases, such as kitchen greases, paints, oil, and gasoline

Class C fires: fueled by live electrical wires or equipment, such as motors, power tools, and appliances

The right type of extinguisher must be used for each class of fire. Water extinguishers control Class A fires by cooling and soaking burning materials. Carbon dioxide or dry chemical extinguishers are used to control class B fires by smothering flames. Multipurpose dry chemical or liquefied gas extinguishers control class A, B, or C fires by a smothering action. Liquefied gas extinguishers also produce a cooling effect. A dry chemical or liquefied gas extinguisher is recommended for recreational vehicles.

Basic Wildland Fire Materials, Training Courses, and Other Information Resources

A large number of reference or text books on wildland fire suppression currently exist, many of which have been cited or referenced in this chapter. *Wildland Fire Fighting for Structural Firefighters* is a comprehensive text written specifically for firefighters whose primary focus is fighting structure fires, but who are also have wildland and wildland-urban interface responsibilities. This book[174] provides an excellent overview on wildland fire behavior, fire apparatus and communications equipment for wildland fires, wildland firefighting tools and PPE, extinguishing agents, wildland incident management, fire suppression methods, wildland and interface fire suppression, firefighter safety and survival, fire prevention and investigation, and fire protection planning. The booklet *Planning for Initial Attack* published by the USDA Forest Service[245] is a handy reference on basic fire behavior and fire suppression principles. Other reference books on the basics of wildland fire suppression exist.[220] For a good general reference book on forest fires, see the recent contribution by Omi.[287]

Wildfire magazine, the official publication of the International Association of Wildland Fire, is an excellent source of information on current fire management topics (www.iawfonline.org), as is *International Forest Fire News* (www.fire.uni-freiburg.de/iffn/iffn.htm) and *Fire Management Today* (www.fs.fed.us/fire/fmt). For other sources, see the list of Suggested Readings for this chapter.

Other information that would be valuable to emergency response personnel includes reference materials and materials associated with the Publications Management System (PMS) and "S," or suppression skills, training courses available through the U.S. National Wildfire Coordinating Group's National Fire Equipment System (NFES) based at the National Interagency Fire Center in Boise, Idaho.[281] These include, for example, "Firefighter Training" (S-130, 2003), "Look Up, Look Down, Look Around" (PMS 427, 1992),[270,271] "Lookouts, Communications, Escape Routes and Safety Zones (LCES)" (S-134, 2003), "Fire Operations in the Wildland–Urban Interface" (S-215, 2003), and "Lessons Learned: Fatality Fire Case Studies" (PMS 490, 1998)[275,276] The latter course uses nine past fatality fires as a learning tool to help fireline tactical decision makers avoid similar mistakes. It is worth noting that some NFES publications are available on-line (www.nwcg.gov).

There are also several CD-ROM–based training courses available on fireline safety,[30,377] fire behavior,[376] and fire danger rating.[355] For additional information on introductory to advanced wildland fire training courses available at the local area, geographic area, and national levels in the United States, visit the Wildland Fire Training website (www.nationalfiretraining.net). For national wildland fire training courses in Canada, visit the website of the Canadian Interagency Forest Fire Centre (www.ciffc.ca).

One of the initiatives of the U.S. National Wildland–Urban Interface Fire Program was a firefighter safety program in the wildland–urban interface series. This training package, which includes three videos, was developed for small community fire departments to address the problems faced by structural and wildland firefighters when fighting fires, especially those threatening structures in the wildland–urban interface.[211] Copies can be ordered from *Firewise Communities* online catalog.

The Wildland Fire Lessons Learned Center (www.wildfirelessons.net) is a good source for information on nearly all aspects of wildland fire management. An annotated reading list and bibliography on wildland firefighter safety management is available[210] on their "My Fire Community" website (www.myfirecommunity.net).

CONCLUSIONS

Fire suppression efforts in the late 1800s and early 1900s were largely ineffective because of limited access, absence of trained firefighting organizations, and lack of a fire detection network. During these times, many residents and numerous firefighters died in wildland fires in the United States and Canada. In the recent past, firefighters were more vulnerable to injuries and fatalities from wildfires than was the general public. Today, with many people living and seeking recreation in wildlands, the odds for serious fire encounters are shifting toward an inexpe-

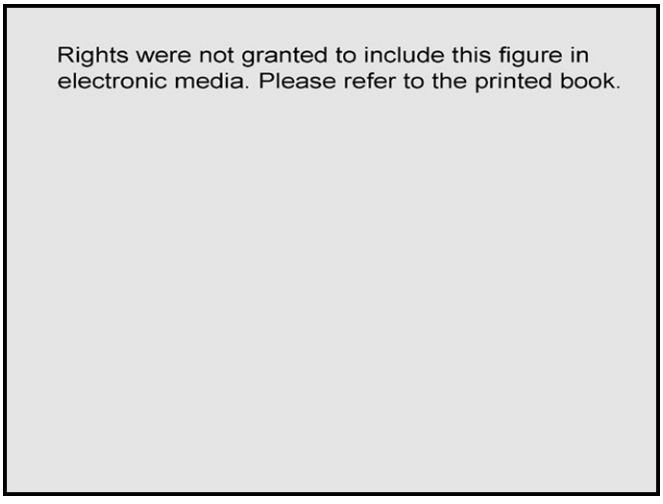

Figure 12-39. The Strawberry Hill fire near Kamloops, British Columbia, about 10 minutes after ignition on August 1, 2003. The white plume in the background is the convection column associated with the McLure fire. Although it does seem theoretically possible to completely eliminate the threat of conflagrations, this is unlikely to happen. Even with the most highly effective fire prevention, fuel management, and fire suppression programs, the likelihood that members of the general public will encounter a high-intensity wildfire event at some point in their lives is gradually increasing as long as they continue to live, recreate, and work in fire-prone environments. Coexistence or living with fire involves taking a proactive stance, including being prepared for when wildfire arrives in a community. (Photo by D. Christie.)

rienced populace. Large property losses and direct injuries are being reported in increasing numbers in the wildland–urban interface. It has now become not a question of "if" a wildfire will come or of it "can't happen here," but rather "when and where" the next major wildland–urban interface conflagration will take place (Fig. 12-39). Emergency medical personnel will probably have increasing exposure to wildland fires in the future and will need to know more about fire-related injuries, fire safety, and fire survival.

Many wildland firefighters are in turn feeling compelled to save homes,[158] due in part to the expectations placed on them by the general public and homeowners in particular, coupled with a culture derived from more than a century of wildland fire suppression.[47] Not surprisingly, many wildland firefighter fatalities in recent years have been associated with wildland–urban interface incidents (e.g., the 2003 Cedar fire in southern California).[85] Given even the best practices,[228] there is an overwhelming need to impress on rural homeowners that no home is worth a firefighter's life.

Although wildland fires have not yet posed a serious threat to backcountry recreationists in the United States to any appreciable degree, the prospect for such confrontations is growing. For example, two recreationists were involved in entrapment and burnover associated with the major run of the 2001 Thirtymile fire[149]; fire investigators concluded they would probably not have survived, although they did suffer minor injuries, had they not joined up with the firefighters just before (about 14 minutes) their location was overrun.[389]

Many years of practical experience with wildland fires allow us to conclude the following:

1. Many indicators point to the fact that financial support and programs that address only emergency responses to wildfires will result in more damaging and expensive wildfire emergencies in the future. Public policies and public education that link sustainable land use practices with emergency preparedness are likely to be most successful in the long run.
2. Residential shifts to the wildland–urban interface will increase exposures to life-threatening situations.
3. Expanded use of fire in managing national parks and wilderness areas will increase the likelihood that backcountry recreationists will encounter free-burning fires.
4. The general public tends to underestimate existing fire hazards and is usually not experienced in avoiding fire threats.
5. In some instances, attempted fire exclusion practices have contributed to development of hazardous wildland fuel situations in terms of quantity and continuity, setting the stage for extreme fire behavior in some plant communities. The national ecosystem health issue has compounded this problem by producing vast expanses of dead and dying forests, increasing the threat to people from fast-moving, high-intensity fires.
6. Knowledge of fire behavior principles and survival guidelines will prepare people to take appropriate preventive measures in threatening situations.

The general public must share responsibility with suppression organizations to minimize fire hazards created by humans. Care with fire, proper cleanup of debris, fuel reduction efforts on wildland property, fire-safe construction guidelines, and application of survival skills will minimize fire threats. Such precautions should become as commonplace in the wildland environment as smoke alarms and fire extinguishers have become in urban settings.

Wildland fire suppression agencies will continue to provide fast, safe, and energetic initial attack responses to protect human life, property, and natural resources.[15] Many fires will start and burn under environmental conditions that permit their control at a very small size. However, in certain situations (e.g., critically dry fuels or strong winds), some fires[9,25,26] will defy control until burning conditions ameliorate. The major runs of the 1985 Palm Coast fire in south Florida[2] and the Millers Reach fire that occurred near Anchorage, Alaska on June 2, 1996[109] are good examples of this kind of fire behavior and associated suppression problems. No radically new concept of fire suppression for stopping the head of a hot, fast-running wildland fire should be anticipated in the future as evident, for example, by the 2003 fire season in California.[126,199]

Strategic fuel management and land use planning could potentially reduce the total number and size of wildfire occurrences as well as influence their geographic distribution, and thereby mitigate the impacts of too much of the "wrong kind of fire."[313] Science-based-only solutions[175] are considered insufficient; effective wildland fire policy must integrate ethics, economics, aesthetics, and values.[313] Furthermore, as Cheney[100] points out, if we are to encourage people to live safely in the wildland environments, they need to be fully aware of the full potential for fire behavior and what can be done to protect themselves and their assets. He goes on to say that "they must be individually persuaded to recognize what constitutes the fuel for a forest fire and be convinced that, since they own the fuel, they also own the fire it produces and are responsible for

the damage they or others nearby incur." The public and fire management agencies themselves must fully appreciate that fuel management is not a panacea for conflagration mitigation, but rather a prerequisite to aid successful fire suppression.[20,59] An excellent source of information on fuel management is the USDA Forest Service Fuels Synthesis Project (www.fs.fed.us/fire/tech_transfer/synthesis/synthesis_index).[390]

After the 1983 Ash Wednesday fires in southeastern Australia, the editor of *Australian Forestry* commented on what could be learned from studying such wildland fire disasters[147]:

> *Advances in technical areas will be of great value. It would be valuable too to make comparable advances in the areas of public policy. People forget, community attitudes change and policies erode. Perhaps one of the greatest challenges, given the realities of southeastern Australia, is to learn how to ensure the permanence of public interest policies where permanence is required.*

Twenty years later and following the "mega-fires" that engulfed Canberra, Australia, Cheney[102] offered some advice on this matter:

> *. . . I believe that there is strong evidence that the policies and institutional arrangements of organizations, both those directly related to fire management and those completely unrelated, will have the result of perpetuating major fire disasters. I also believe that the legal framework in which we're working is one that [militates against] individuals taking responsibility for their own actions and lifestyle choices. In this country wildfire is inevitable but the impact of disastrous fires can be reduced. But this will only be achieved if policies focus on the physics of fire spread and foster a climate of awareness where fuel reduction, from the backyard to beyond, is encouraged and supported by legislation.*

At the end of the day, though, what can an individual do to improve matters when it comes to living with fire? Kaufmann, Shlisky, and Marchand[196] offer some very practical advice:

- Get involved in a community-based conservation group working on local landscape restoration projects.
- Educate yourself about the role of fire in your local ecosystems.
- Provide feedback on agency land management plans.
- Consult with regional experts on how to safely reintroduce fire on to your land holding.
- Participate in local workshops (e.g., *Firewise Communities*) to learn how to treat fuels around your home and create defensible space.

Their final suggestion: "Start simply. But start." Local forestry and fire management agencies are more than willing to offer technical advice and information. Just give them a call to get started today before wildfire comes knocking!

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SUGGESTED READINGS

Agee JK: Fire Ecology of Pacific Northwest Forests. Washington, DC, Island Press, 1993.

Alexander ME: The staff ride approach to wildland fire behavior and firefighter safety training: A commentary. Fire Manage Today 62(4):25, 2002.

Alexander ME: What triggered the Brewer Fire blowup remains the mystery. Fire Manage Today 64(2):56, 2004.

Alexander ME: You are about to be entrapped or burned over by a wildfire: What are your survival options? In Proceedings of V International Conference on Forest Fire Research, 2006.

Alexander ME, et al (tech coords): People, Ecosystems and Wildland Fires—Working in Harmony. Abstracts and Program of the 2000 Interior West Fire Council Annual Meeting and Workshop, held October 3–5 in Edmonton, Alberta. Alberta Environment, Land, and Forest Service, Forest Protection Division and Natural Resources Canada, Canadian Forest Service, Northern Forestry Centre. Available at: http://www.interiorwestfirecouncil.org.

Alexander ME, Fogarty LG: A Pocket Card for Predicting Fire Behaviour in Grasslands Under Severe Burning Conditions. Fire Technology Transfer Note 25, Rotorua and Wellington, New Zealand, and Ottawa, Ontario, 2002, Forest Research Institute, Rotorua, National Rural Fire Authority, and Natural Resources Canada, Canadian Forest Service.

Alexander ME, Thomas DA (issue coords): Wildland Fire Behavior Case Studies and Analyses: Part 1. Fire Manage Today 63(3):1, 2003.

Alexander ME, Thomas DA (issue coords): Wildland Fire Behavior Case Studies and Analyses: Part 2. Fire Manage Today 63(4):1, 2003.

Alexander ME, Thomas DA (issue coords): Forecasting Wildland Fire Behavior: Aids and Guides. Fire Manage Today 64(1):1, 2004.

Alexander ME, Thomas DA (issue coords): Prescribed Fire Case Studies, Decision Aids, and Planning Guides. Fire Manage Today 66(1):1, 2006.

Arno SF, Allison-Bunnell S: Flames in Our Forest. Washington, DC, Island Press, 2002.

Arno SF; Fiedler CE: Mimicking Nature's Fire: Restoring Fire-Prone Forests in the West. Washington, DC, Island Press, 2005.

Arrowood JC: Living with Wildfires: Prevention, Preparation, and Recovery, Denver, Colo, Bradford Publishing Company, 2003.

Beck J, et al: Fire ecology and management. In Watts SB, Tolland L (eds): Forestry Handbook of British Columbia. Vancouver, BC, University of British Columbia, Faculty of Forestry, 2005.

Blanchet P: Forest Fires: The Story of War. Montreal, Que, Cantos International Publishing, 2003.

Blonski KS, Miller C, Rice CL: Urban-Wildland Fire: A Practical Guide for Local Governments, Fire Authorities, Developers, and Property Owners. Port Arena, Calif, Solano Press, 2007.

Bradford G: Structure Protection in the I-Zone: Focusing Your Wildland Experience for the Urban Interface. Tulsa, Okla, Pennwell Books, 2000.

Brown JK, Smith JK (eds): Wildland Fire in Ecosystems: Effects of Fire on Flora. General Technical Report RMRS-GTR-42 volume 2, Ogden, Utah, USDA Forest Service, Rocky Mountain Research Station, 2000.

Budd G, et al: Safe and Productive Bushfire Fighting with Hand Tools. Canberra, Australian Capital Territory, Australian Government Publishing Service, 1996.

Burrows ND: Describing Forest Fires in Western Australia. Technical Paper No. 9. Perth, Western Australia, Forests Department of Western Australia, 1984.

Carle D: Burning Questions: America's Fight with Nature's Fire. Westport, Conn, Praeger Publishers, 2002.
Carroll MS, et al: Fire as a galvanizing and fragmenting influence on communities: The case of the Rodeo-Chediski fire. Soc Nat Resour 18:301, 2005.
Cary G, Lindenmayer D, Dovers S (eds): Australia Burning: Fire Ecology, Policy and Management Issues. Collingwood, South Australia, CSIRO Publishing, 2003.
Cash P, et al: Health, Safety and Workload Challenges of the Okanagan Mountain Fire, 2003. Okanagan, British Columbia, University of British Columbia Okanagan, Faculty of Health and Social Development, School of Nursing, 2005.
Close KR: 20 Minutes at H-2-linear decision making in an exponential fire environment. In Proceedings of the Ninth International Wildland Fire Safety Summit, 2006.
Cottrell WH: The Book of Fire. Missoula, Mont, Mountain Press, 1989.
Daniel TC, Ferguson IS (eds): Proceedings of the U.S.–Australian Workshop Integrating Research on Hazards in Fire-Prone Environments-Reconciling Biological and Human Values in the Wildland/Urban Interface, U.S. Man and the Biosphere Program. Washington, D.C., 1991.
DeBano LF, Neary DG, Ffolliott PF: Fire's Effects on Ecosystems. New York, John Wiley & Sons, 1998.
DeSorcy G, Partington L, Cohen J: Chisholm Fire Review Committee Final Report. Edmonton, Alberta, Alberta Sustainable Resource Development, 2001.
Filmon G, Leitch D, Sproul J: Firestorm 2003: Provincial Review. Victoria, British Columbia, Government of British Columbia, 2004. Available at: http://www.2003firestorm.gov.bc.ca.
Fire and Emergency Services Authority of Western Australia: The Homeowner's Bush Fire Survival Manual (4th ed). Perth, Western Australia, Fire and Emergency Services Authority of Western Australia, 2004.
Fischer WC, Arno SF (comps): Protecting People and Homes From Wildfire in the Interior West: Proceedings of the Symposium and Workshop, General Technical Report INT-251. Ogden, Utah, USDA Forest Service, Intermountain Research Station, 1988.
Fowler CT: Human health impacts of forest fires in the Southern United States. J Ecol Anthropol 7:39, 2003.
Fuller M: Forest Fires: An introduction to Wildland Fire Behavior, Management, Firefighting, and Prevention. New York, J. Wiley & Sons, 1991.
Gaylor HP: Wildfires: Prevention and Control. Bowie, Md, RJ Brady, 1974.
Gilmer M: California Wildfire Landscaping. Dallas, Tx, Taylor Publishing, 1994.
Gomes D, Graham Jr OL, Marshall EH, Schmidt AJ: Sifting Through the Ashes: Lessons Learned from the Painted Cave Fire. Santa Barbara, Calif, University of California-Santa Barbara, 1993.
Graham RT (tech ed): Hayman Fire Case Study. General Technical Report RMRS-GTR-114. Fort Collins, Colo, USDA Rocky Mountain Research Station, 2003.
Graham RT (tech ed): Hayman Fire Case Study: Summary. General Technical Report RMRS-GTR-115. Fort Collins, Colo, USDA Rocky Mountain Research Station, 2003.
Hardy CE, et al: Smoke Management Guide for Prescribed and Wildland Fire (2001 ed). National Fire Equipment System NFES Publication 1279. Boise, Idaho, National Wildfire Coordinating Group, 2001.
Heinselman ML: The Boundary Waters Wilderness Ecosystem. Minneapolis, Minn, University of Minnesota Press, 1996.
Huggard CJ, Gomez AR (eds): Forests Under Fire: A Century of Ecosystem Mismanagement in the Southwest. Tucson, Ariz, University of Arizona Press, 2001.
Institute for Business and Home Safety: Is Your Home Protected from Wildfire Disaster? A Homeowner's Guide to Wildfire Retrofit. Tampa, Fla, IBHS, 2001.
Keller P (tech ed): Managing the Unexpected in Prescribed Fire and Fire Use Operations: A Workshop on the High Reliability Organization. General Technical Report RMRS-GTR-137. Fort Collins, Colo, USDA Forest Service, Rocky Mountain Research Station, 2004.
Kumagai Y, Carroll MS, Cohn P: Coping with interface wildfire as a human event: Lessons from the disaster/hazards literature. J For 102(6):40, 2004.
Kumagai Y, et al: Research on causal attribution of wildfire: an exploratory multiple-methods approach. Soc Nat Resour 17:113, 2004.
Mangan RJ: Lessons Learned: The Use of Personal Protective Equipment on Wildland Fire Entrapments in 1993. Fire Tech Tips 9451-2335-MTDC. Missoula, Mont, USDA Forest Service, Missoula Technology and Development Center, 1994.
Miller C, Landres P: Exploring Information Needs for Wildland Fire and Fuels Management. General Technical Report RMRS-GTR-127. Fort Collins, Colo, USDA Forest Service, Rocky Mountain Research Station, 2004.
Monroe MC, Long AJ, Marynowski S: Wildland fire in the Southeast: Negotiating guidelines for defensible space. J For 101(3):14, 2003.
Moore HE: Protecting Residences from Wildfires: A Guide for Homeowners, Lawmakers, and Planners. General Technical Report PSW-50, Berkeley, Calif, USDA Forest Service, Pacific Southwest Forest and Range Experiment Station, 1981.
Murphy PJ, et al: The Alberta Forest Service, 1930–2005: Protection and Management of Alberta's Forests. Publication No. I/33. Edmonton, Alberta, Alberta Sustainable Resource Development, 2006.
National Fire Protection Association: Black Tiger Fire Case Study. Quincy, Mass, NFPA, 1991.
National Fire Protection Association: Stephan Bridge Road Fire Case Study. Quincy, Mass, NFPA, 1992.
National Fire Protection Association: American Perspectives on the Wildland/Urban Interface. Quincy, Mass, NFPA, 2004.
O'Bryan D: The Science of Bushfire Behavior: Search for Answers. Scarsdale, Victoria, Australia, Papyrus Publishing, 2005.
Ombudsman Province of British Columbia: The Sliver Creek Fire Review. Victoria, B.C., Public Report No. 39 to the Legislative Assembly of British Columbia, 1999.
Pattison L: The Garnet Fire. Altona, Manitoba, Friesen Printing, 1995.
Perry DG: Managing a Wildland Fire: Practical Perspective. Bellflower, Calif, Fire Publications, 1989.
Perry DG: Wildland Firefighting: Fire Behavior, Tactics & Command (2nd ed). Bellflower, Calif, Fire Publications, 1990.
Phillips CB: Fire safety for individual home owners. In Proceedings of the 1992 Society of American Foresters National Convention, 1992.
Platt RH: To Burn or Not to Burn: Summary of the Forum on Urban/Wildland Fire, Washington, DC, January 26, 2001. Washington, DC, National Academy of Sciences, 2001. Available at: http://www.nap.edu.
Pringle L: Natural Fire: Its Ecology in Forests. New York, William Morrow, 1979.
Pyne SJ: America's Fires: Management on Wildlands and Forests. Durham, NC, Forest History Society, 1997.
Pyne SJ: Fire: A Brief History. Seattle, University of Washington Press, 2001.
Pyne SJ: Smokechasing. Tucson, AZ, University of Arizona Press, 2003.
Pyne SJ: The Still-Burning Bush. Carlton, Victoria, Australia, Scribe Short Books, 2006.
Queen PL: Fighting Fire in the Wildland/Urban Interface. Bellflower, Calif, Fire Publications, 1993.
Radtke KWH: Living More Safely in the Chaparral-Urban Interface. General Technical Report PSW-67. Berkeley, Calif, USDA Forest Service, Pacific Southwest Forest and Range Experiment Station, 1981.

Radtke KWH: A Home Owner's Guide to Fire and Watershed Management at the Chaparral/Urban Interface. San Diego, Calif, City of San Diego Water Department, 2004. Available at: http://www.sandiego.gov.
Ramsay GC, Dawkins D (comps and eds): Building in Bushfire-Prone Areas: Information and Advice. Melbourne, Victoria, Australia, CSIRO and Standards Australia, 1999.
Ramsay GC, Rudolph L: Landscape and Building Design for Bushfire Areas. Collingwood, Victoria, Australia, CSIRO Publishing, 2003.
Rothermel RC: How to Predict the Spread and Intensity of Forest and Range Fires. General Technical Report INT-143. Ogden, Utah, USDA Forest Service, Intermountain Forest and Range Experiment Station, 1983.
Schindler B, Toman E: Fuel reduction strategies in forest communities: A longitudinal analysis of public support. J For 101(9):8, 2003.
Schwab J, Meck S, Simone J: Planning for Wildfires. Washington, DC, American Planning Association, 2005.
Slaughter R (ed): California's I-Zone: Urban/Wildland Fire Prevention and Mitigation. Sacramento, Calif, California Department of Forestry and California State Fire Marshall, 1996.
Smith JK, McMurray NE: FireWorks Curriculum Featuring Ponderosa, Lodgepole and Whitebark Pine Forests. General Technical Report RMRS-GTR-65. Fort Collins, Colo, USDA Forest Service, Rocky Mountain Research Station, 2000.
Teie WC: Firefighter's Handbook on Wildland Firefighting: Strategy, Tactics and Safety. Rescue, Calif, Deer Valley Press, 1994.
Teie WC: Fire Officer's Handbook on Wildland Firefighting. Rescue, Calif, Deer Valley Press, 1997.
Thoele M: Fire Line: The Summer Battles of the West. Golden, Colo, Fulcrum Publishing, 1995.
Titus M, Hinderman J: Safety in numbers. Wildfire 15(4):8, 2006.
TriData Corporation: Wildland firefighter safety awareness study, phase III. Arlington, Va, 1998. Available at: http://www.nific.gov.
USDA Forest Service: Dude Fire staff ride. Fire Manage Today 62(4):1, 2002.
Viegas DX: [Surrounded by Fire: The Forest Fires of 2003 and Associated Mortal Incidents] [in Portuguese]. Coimbra, Portugal, MinervaCoimbra, 2004.
Wade D, Ewel J, Hofstetter R: Fire in South Florida Ecosystems. General Technical Report SE-17. Asheville, NC, USDA Forest Service, Southeastern Forest Experiment Station, 1980.
Wallace LL: After the Fires: The Ecology of Change in Yellowstone National Park. New Haven, Conn, Yale University Press, 2004.
Weick KE: The collapse of sensemaking in organizations: The Mann Gulch disaster. Admin Sci Quart 38:828, 1993.
Weise DR, Martin RE (tech coords): The Biswell Symposium: Fire Issues and Solutions in Urban Interface and Wildland Ecosystems. General Technical Report PSW-GTR-158. Berkeley, Calif, USDA Forest Service, Pacific Southwest Research Station, 1995.
White SM: Bridging the Worlds of Fire Managers and Researchers: Lessons and Opportunities from the Wildland Fire Workshops. General Technical Report PNW-GTR-599. Portland, Ore, USDA Forest Service, Pacific Northwest Research Station, 2004.
Wilson-Millar G: Critical Incident Stress in Wildland Firefighting. Fairfield, Wash, International Association of Wildland Fire, 1992.
Winter GJ, Vogt C, Fried JS: Fuel treatments at the wildland-urban interface: Common concerns in diverse regions. J For 100(1):15, 2002.
Winter G, Vogt CA, McCaffrey S: Examining social trust in fuel management strategies. J For 102(6):8, 2004.

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Emergency Care of the Burned Victim

13

Roberta Mann and David M. Heimbach

EPIDEMIOLOGY

Each year in the United States, approximately 2 million individuals are burned seriously enough to seek the care of a physician, and about 70,000 of these require hospitalization. Fortunately, the number of deaths attributable to burns decreased from 12,000 in 1979 to 6000 in 1990. Although treatment facilities would like to take credit for this decrease, the most likely reason is the widespread use of smoke detectors in both public and private dwellings. It has been estimated that more than 90% of burns are preventable and that most are caused by carelessness or ignorance. During the next few years, the biggest contributions in decreasing burn mortality and morbidity may come not from medical scientists but rather from improved engineering design and more successful programs in teaching burn prevention to the public.

Like other forms of trauma, burns frequently affect children and young adults. The hospital expenses and the social costs related to time away from work or school are staggering. Although most burns are limited in extent, a significant burn of the hand or foot may prevent a manual laborer from working for a year or more, and in some cases may permanently prevent a return to former activity. The eventual outcome for the burned victim is related to the severity of the injury, individual physical characteristics of the victim, motivation toward rehabilitation, and quality of treatment of the acute burn.

Most persons with burns who are seen by physicians visit emergency departments, where judgment in triage, care plan for small burns, and initial management for major burns can influ-

Radtke KWH: A Home Owner's Guide to Fire and Watershed Management at the Chaparral/Urban Interface. San Diego, Calif, City of San Diego Water Department, 2004. Available at: http://www.sandiego.gov.
Ramsay GC, Dawkins D (comps and eds): Building in Bushfire-Prone Areas: Information and Advice. Melbourne, Victoria, Australia, CSIRO and Standards Australia, 1999.
Ramsay GC, Rudolph L: Landscape and Building Design for Bushfire Areas. Collingwood, Victoria, Australia, CSIRO Publishing, 2003.
Rothermel RC: How to Predict the Spread and Intensity of Forest and Range Fires. General Technical Report INT-143. Ogden, Utah, USDA Forest Service, Intermountain Forest and Range Experiment Station, 1983.
Schindler B, Toman E: Fuel reduction strategies in forest communities: A longitudinal analysis of public support. J For 101(9):8, 2003.
Schwab J, Meck S, Simone J: Planning for Wildfires. Washington, DC, American Planning Association, 2005.
Slaughter R (ed): California's I-Zone: Urban/Wildland Fire Prevention and Mitigation. Sacramento, Calif, California Department of Forestry and California State Fire Marshall, 1996.
Smith JK, McMurray NE: FireWorks Curriculum Featuring Ponderosa, Lodgepole and Whitebark Pine Forests. General Technical Report RMRS-GTR-65. Fort Collins, Colo, USDA Forest Service, Rocky Mountain Research Station, 2000.
Teie WC: Firefighter's Handbook on Wildland Firefighting: Strategy, Tactics and Safety. Rescue, Calif, Deer Valley Press, 1994.
Teie WC: Fire Officer's Handbook on Wildland Firefighting. Rescue, Calif, Deer Valley Press, 1997.
Thoele M: Fire Line: The Summer Battles of the West. Golden, Colo, Fulcrum Publishing, 1995.
Titus M, Hinderman J: Safety in numbers. Wildfire 15(4):8, 2006.
TriData Corporation: Wildland firefighter safety awareness study, phase III. Arlington, Va, 1998. Available at: http://www.nific.gov.
USDA Forest Service: Dude Fire staff ride. Fire Manage Today 62(4):1, 2002.
Viegas DX: [Surrounded by Fire: The Forest Fires of 2003 and Associated Mortal Incidents] [in Portuguese]. Coimbra, Portugal, MinervaCoimbra, 2004.
Wade D, Ewel J, Hofstetter R: Fire in South Florida Ecosystems. General Technical Report SE-17. Asheville, NC, USDA Forest Service, Southeastern Forest Experiment Station, 1980.
Wallace LL: After the Fires: The Ecology of Change in Yellowstone National Park. New Haven, Conn, Yale University Press, 2004.
Weick KE: The collapse of sensemaking in organizations: The Mann Gulch disaster. Admin Sci Quart 38:828, 1993.
Weise DR, Martin RE (tech coords): The Biswell Symposium: Fire Issues and Solutions in Urban Interface and Wildland Ecosystems. General Technical Report PSW-GTR-158. Berkeley, Calif, USDA Forest Service, Pacific Southwest Research Station, 1995.
White SM: Bridging the Worlds of Fire Managers and Researchers: Lessons and Opportunities from the Wildland Fire Workshops. General Technical Report PNW-GTR-599. Portland, Ore, USDA Forest Service, Pacific Northwest Research Station, 2004.
Wilson-Millar G: Critical Incident Stress in Wildland Firefighting. Fairfield, Wash, International Association of Wildland Fire, 1992.
Winter GJ, Vogt C, Fried JS: Fuel treatments at the wildland-urban interface: Common concerns in diverse regions. J For 100(1):15, 2002.
Winter G, Vogt CA, McCaffrey S: Examining social trust in fuel management strategies. J For 102(6):8, 2004.

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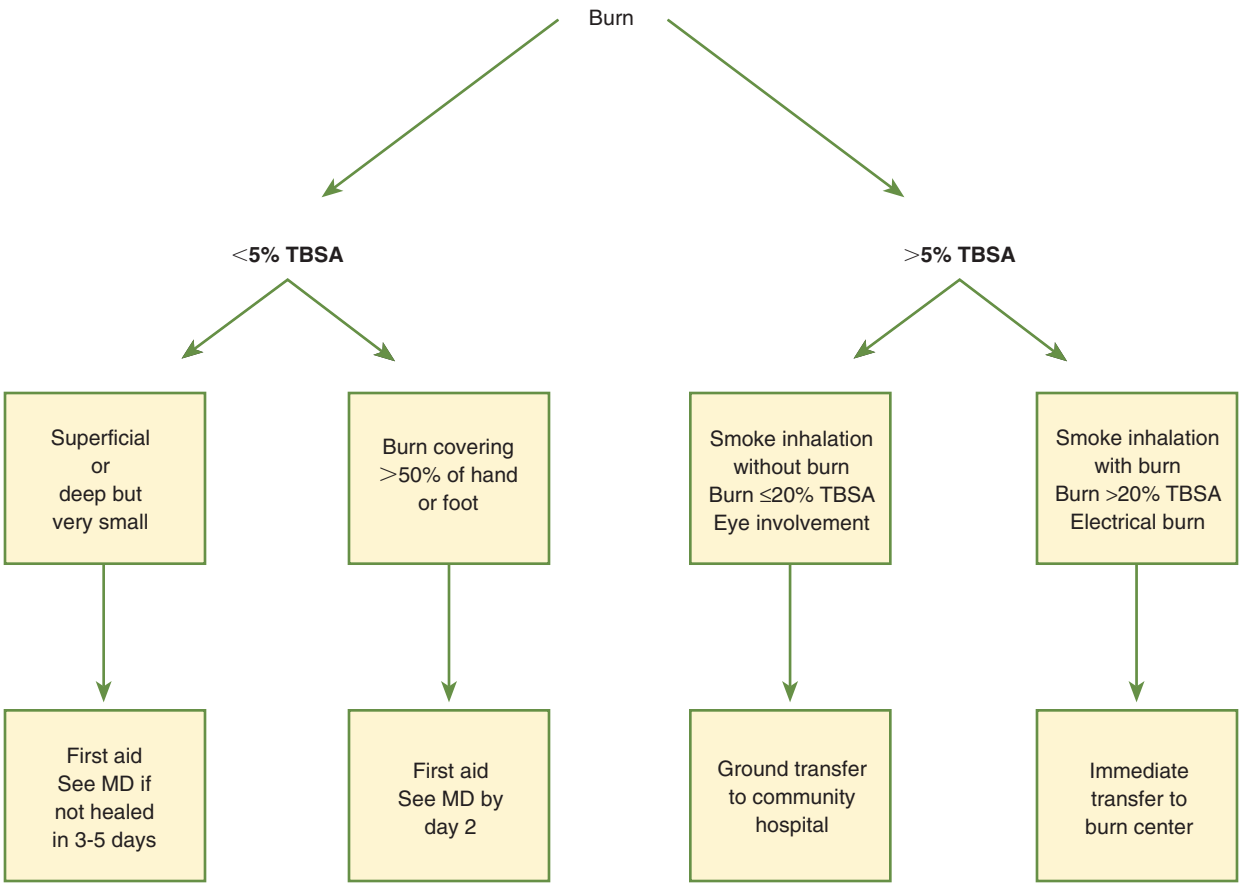

Figure 13-1. Algorithm for decision making at the scene. TBSA, total body surface area.

ence survival of the victim and eventual cosmetic and functional results. Because most victims are young and about one third are children, they will live with the consequences of the acute treatment for an average of 50 years.

The decisions made at the initial contact with the victim require answers to the algorithm shown in Figure 13-1. This chapter describes first-responder care for major burns and is organized to guide assessment of burn severity and initial management of the serious burns, as well as to provide an initial treatment plan for minor burns.

PHYSIOLOGY

For burns other than chemical burns, the primary events of injury occur during the time of heat contact. Coagulation necrosis takes place within cells, and denaturation of collagen occurs in the dermis. Either blood vessels are completely destroyed or the endothelium is damaged severely enough to cause clotting, which leads to ischemic necrosis of remaining viable cells. The burn wound is not static. Surrounding the zone of coagulation is a zone of capillary and small vessel stasis. Red cells form into rouleaux, platelet and white cell aggregates form, and the circulation becomes stagnant. Over the next hours or even days, the ultimate fate of the burn wound depends on the resolution or progression of this zone of stasis. Cells and tissue stroma release mediators to initiate the inflammatory response. Histamine, serotonin, prostaglandin derivatives, and the complement cascade all have roles. In victims with burns of less than 10% of the total body surface area (TBSA), the actions of these mediators are generally limited to the burn site itself. Capillary permeability increases, neutrophils marginate, and additional inflammatory cells (monocytes, macrophages) are attracted by chemotaxis to the site of injury, initiating the healing process.

As burns approach 20% TBSA, the local response becomes systemic. The capillary leak, permitting loss of fluid and protein from the intravascular compartment into the extravascular compartment, becomes generalized. Cardiac output falls as a result of the markedly increased peripheral resistance, decreased intravascular fluid volume from the capillary leak, and accompanying increase in blood viscosity. Decreased blood volume and cardiac output, accompanied by an intense sympathetic response, lead to decreased perfusion to the skin and viscera. Decreased flow to the skin can convert the zone of stasis to one of coagulation, increasing the depth of the burn. The capillary leak and depressed cardiac output lead to depressed central nervous system (CNS) function, and in extreme cases they result in severe cardiac depression with eventual cardiac failure in

healthy patients or in myocardial infarction in patients with preexisting coronary artery disease. The first sign of CNS change is restlessness, followed by lethargy and finally coma. Without adequate resuscitation, burns of 30% TBSA frequently lead to acute renal failure, which in a victim with a severe burn almost invariably leads to a fatal outcome.

Cardiovascular changes begin immediately after a burn. The extent of these changes depends primarily on the size of the burn and to a lesser extent on the depth of the burn. Most victims with uncomplicated burns of less than 15% TBSA can undergo oral fluid resuscitation with some salt-containing solution. As the burn extent passes 20% TBSA, massive shifts of fluid and electrolytes occur from the intravascular into the extravascular (extracellular) space. Reversal of the shifts begins during the second postburn day, but normal extracellular volume is not completely restored until 7 to 10 days after the burn. Unless the intravascular volume is depleted, classic hypovolemic shock occurs. An untreated person will die of cardiovascular collapse; if treatment is poor, irreversible acute tubular necrosis and renal failure will develop.

TYPES OF BURNS

Scald Burns

In civilian practice, scalds, usually resulting from hot water, are the most common cause of burns. Water at 60°C (140°F) creates a deep partial-thickness or full-thickness burn in 3 seconds. At 68.9°C (156°F), the same burn occurs in 1 second. Freshly brewed coffee from an automatic percolator is generally about 82°C (180°F). Boiling water always causes deep burns, and soups and sauces, which are thicker in consistency, remain in contact longer with the skin and often cause deep burns. In general, exposed areas tend to be burned less deeply than areas covered with thin clothing. Clothing retains the heat and keeps the liquid in contact with the skin for a longer time.

Immersion scalds are deep and severe burns.[21,31,97] Although the water may be cooler than with a spill scald, the duration of contact is longer, and these burns frequently occur in small children or older adult victims with thin skin. Consequently, many states have passed legislation to set home and public hot water heaters to maximum temperatures well below 60°C (140°F).

Scald burns from grease or hot oil are generally deep partial thickness or full thickness. Cooking oil and grease, when hot enough to use for cooking, may be in the range of 204.4°C (400°F). Tar and asphalt burns are a special kind of scald. The "mother pot" at the back of the roofing truck maintains tar at a temperature of 204.4° to 260°C (400° to 500°F). Burns caused by tar directly from the mother pot are invariably full thickness. By the time the tar is spread on the roof, its temperature has decreased enough that most of the burns are deep partial thickness. Unfortunately, the initial evaluator cannot usually examine these burns because of the adherent tar. The tar should be removed by application of a petroleum-based ointment (such as Vaseline) under a dressing. The dressing may be removed and the ointment reapplied every 2 to 4 hours until the tar has dissolved. Only then can the extent of the injury and the depth of the burn be accurately estimated.[43,96]

Flame Burns

Flame burns are the next most common burn injuries. Although injuries in house fires have decreased with the advent of smoke detectors, a significant number of burn injuries still result from careless smoking, improper use of flammable liquids, automobile accidents, and clothing ignited from stoves or space heaters. Victims whose bedding or clothes have been on fire rarely escape without some full-thickness burns. Outdoor misadventures result from using cooking stoves fueled by white gasoline, taking lanterns into tents, smoking in a sleeping bag, and starting (or improving) charcoal fires with gasoline or kerosene.

Flash Burns

Flash burns are next in frequency. Explosions of natural gas, propane, gasoline, and other flammable liquids cause intense heat for a very brief time. For the most part, unignited clothing protects the skin in flash burns. Flash burns generally have a distribution over all exposed skin, with the deepest areas facing the source of ignition. Flash burns are partial thickness, with the depth dependent on the amount and kind of fuel that explodes. Although such burns generally heal without requiring extensive skin grafts, they may be very large and associated with significant thermal damage to the upper airway.

Contact Burns

Contact burns result from hot metals, plastic, glass, or hot coals. Such burns are usually limited in extent but deep. Victims involved in industrial accidents commonly have both severe contact burns and crush injuries, because these accidents often occur from presses or from hot, heavy objects. With the increased use of wood-burning stoves, an increasing number of toddlers are burned each year. The most common injuries are deep burns on the palms because the child falls with hands outstretched against the stove. Contact burns, especially in unconscious persons or those dealing with molten materials, are frequently fourth degree.[19,56,84] In the wilderness setting, the most common contact burn is from hot coals. Intoxicated campers dance around and then into the campfire, architects of "river saunas" mishandle hot rocks, children fall into fires, and beach walkers may sustain deep burns when coals are buried in sand overnight. Even though the injured areas may be small, they can be deep and devastating when the hiker must walk a considerable distance on burned feet.[27]

Electrical Burns

Electrical burns are actually thermal burns from very high-intensity heat. As electricity meets the resistance of body tissues, it is converted to heat in direct proportion to the amperage of the current and the electrical resistance of the body parts through which it passes. The smaller the body part through which the electricity passes, the more intense the heat is and the less it is dissipated. Therefore, fingers, hands, forearms, feet, and lower legs are frequently totally destroyed, whereas larger-volume areas, such as the trunk, usually dissipate the current enough to prevent extensive damage to the viscera, unless the contact point is on the abdomen or chest. Although cutaneous manifestations may appear limited, massive underlying tissue destruction may be present.[18,36,37,68]

Arc burns occur when current takes the most direct path rather than the one of least resistance. These deep and destructive wounds occur at joints that are in close apposition at the time of injury. Most common are burns of the forearm to the arm when the elbow is flexed, and from the arm to the axilla if the shoulder is adducted when current passes from the upper extremity to the trunk.

Electrical burns cause a particular set of other injuries and complications that must be considered during the initial evaluation. As mentioned previously, injuries related to a fall are common. The intense associated muscle contractions may cause fractures of the lumbar vertebrae, humerus, or femur and may dislocate shoulders or hips.

Electrical cardiac damage may have symptoms like those of a myocardial contusion or infarction. Alternatively, the conduction system may be deranged. There can be actual rupture of the heart wall or of a papillary muscle, leading to sudden valvular incompetence and refractory cardiac failure. Household current at 110 volts generally either does no damage or induces ventricular fibrillation. Alternating current is more likely to induce fibrillation than is direct current. If no cardiac abnormalities are present when a victim is first seen after shocks of 110 to 220 volts, the likelihood that they will appear later is small.

The nervous system is particularly sensitive to electricity. The most severe brain damage occurs when current passes through the head, but spinal cord damage is possible any time current passes from one side of the body to the other.[47,51] Myelin-producing cells are susceptible. The devastating effects of transverse myelitis may develop days or weeks after injury. Conduction remains normal through existing myelin, but as the old myelin wears out, it is not replaced and conduction stops. Peripheral nerves are commonly damaged and may demonstrate severe permanent functional impairment.[23,33] Every victim with an electrical injury must have a thorough neurologic examination as part of the initial assessment. Myoglobinuria is a frequent accompaniment of severe electrical burns. Disruption of muscle cells releases cell fragments and myoglobin into the circulation to be filtered by the kidney. If untreated, this can lead to permanent renal failure.

Lightning strikes are discussed in Chapter 3, and reviews are available.[24,25,28,29,57,68]

Chemical Burns

Chemical burns, usually caused by strong acids or alkalis, are most often the result of industrial accidents, home use of drain cleaners, assaults, and other improper use of harsh solvents.[26,38,62,64,76,79,86] In contrast to thermal burns, chemical burns cause progressive damage until the chemicals are inactivated by reaction with the tissue or by dilution by flushing with water. Although individual circumstances vary, acid burns may be more self-limiting than are alkali burns. Acid tends to tan the skin (as leather is tanned), creating an impermeable barrier that limits further penetration of the acid. Alkalis combine with cutaneous lipids and saponify the skin until they are neutralized. A full-thickness chemical burn may appear deceptively superficial, appearing as only a mild brownish surface discoloration. The skin may appear intact during the first few days after the burn and then begin to slough spontaneously. Unless the observer can be absolutely certain, chemical burns should be considered deep partial thickness or full thickness until proven otherwise.

CLINICAL PRESENTATION

Cutaneous burns are caused by the application of heat or caustic chemicals to the skin. When heat is applied to the skin, the depth of injury is proportional to the temperature applied, duration of contact, and thickness of the skin.

The severity of the burn injury is related to the size of the burn, depth of the burn, and part of the body that is burned.

Estimation of Burn Size

Burns are the only quantifiable form of trauma. The single most important feature in predicting mortality, need for specialized care, and the complications expected from the burn is the overall burn size in proportion to the victim's TBSA. Treatment plans, including initial resuscitation and subsequent nutritional requirements, are derived directly from the size of the burn.

A general idea of burn size is provided by the "rule of nines." Each upper extremity accounts for 9% of TBSA, each lower extremity accounts for 18%, the anterior and posterior trunk each account for 18%, the head and neck account for 9%, and the perineum accounts for 1% (Fig. 13-2). Although the rule of nines provides a reasonably accurate estimate of burn size, a number of more precise charts have been developed. A diagram of the burn can be drawn on a chart so that a relatively precise calculation of burned area can be made from the accompanying TBSA estimates given. Children less than 4 years old have much larger heads and smaller thighs in proportion to body size than do adults. In an infant, the head accounts for approximately 18% of the TBSA; body proportions do not fully reach adult percentages until adolescence. To further increase accuracy in burn size estimation, especially when burns are in scattered body areas, the observer might calculate the unburned areas on a separate diagram. If the calculations of the unburned areas and the burned areas do not add up to 100%, the observer should begin again with a new diagram to recalculate the burned areas. For smaller burns, an accurate assessment of burn size can be made by using the victim's hand. The hand amounts to 2.5% TBSA. The dorsal surface accounts for 1%, the palmar surface for 1%, and the vertical surface for 0.5%.

Depth of Burn

An understanding of burn depth requires an understanding of skin anatomy (Fig. 13-3). The epidermis, an intensely active layer of epithelial cells under layers of dead keratinized cells, is superficial to the active structural framework of the skin, the dermis. Although metabolically very active, the dermis has no regenerative capacity, and epithelial cells must eventually cover the surface of the dermis before the burn is healed. The skin appendages (hair follicles, sebaceous glands, and sweat glands) all contain an epithelial cell lining, so when the surface epidermis has been killed, epithelial covering must take place from overgrowth of the epithelial cells lining the skin appendages. As these cells reach the surface, they spread laterally to meet their neighbors, creating a new epithelial surface. As the burn extends deeper into the dermis, fewer and fewer appendages remain, and the epithelial remnants must travel farther to produce a new surface covering, sometimes taking many weeks to produce coverage. When the burn extends beyond the deepest layer of the skin appendages, the wound can heal only by epithelial ingrowth from the edges, by wound contraction, or by surgical transplantation of skin from a different site.

The thickness of skin varies both with the age and sex of the individual and with the part of the body considered. Although the thickness of the living epidermis is relatively constant, keratinized epidermal cells may reach a height of 5 mm on palms of hands and soles of feet. The thickness of the dermis, on the

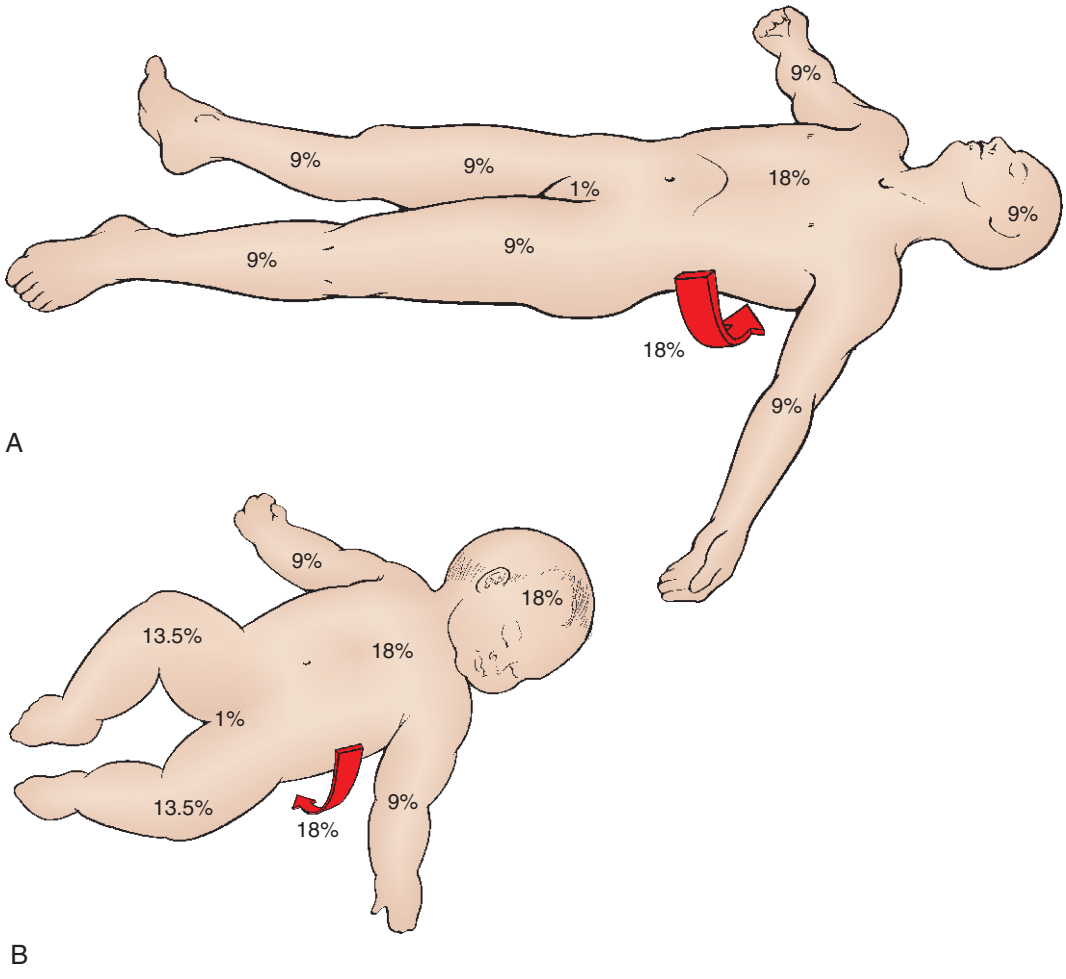

Figure 13-2. Rule of nines used for estimating burned surface area. **A,** Adult. **B,** Infant.

other hand, may vary from less than 1 mm on eyelids and genitalia to more than 5 mm on the posterior trunk. Although the proportional thickness of skin in each body area is similar in children, infant skin thickness in each specific area may be less than half that of adult skin; the skin does not reach adult thickness until adolescence. Similarly, in patients older than 50 years, dermal atrophy causes all areas of skin to become quite thin.

Burns are classified by increasing depth as first degree, superficial partial thickness, deep partial thickness, full thickness, and fourth degree. The following descriptions appear to separate burns into clearly defined categories, but many burns have a mixture of characteristics that give the observer an imprecise diagnostic ability. Considerable research is under way to devise instruments that will more precisely measure the depth of injury. Much of current burn treatment depends on knowledge of the depth of the burn.

First-Degree Burns. First-degree burns involve only the epidermis. The prototype is mild sunburn. First-degree burns do not blister. They become erythematous because of dermal vasodilation and are quite painful, both spontaneously and when touched. The erythema and pain subside over 2 to 3 days. By the fourth day, the injured epithelium desquamates in the phenomenon of "peeling."

Superficial Partial-Thickness Burns. Superficial partial-thickness burns (Fig. 13-4) include the upper layers of dermis and characteristically form blisters with fluid collection at the interface of the epidermis and dermis. Blistering may not occur for some hours after injury. Burns initially thought to be first degree may therefore be diagnosed as superficial partial thickness by day 2. When blisters are removed, the wound is pink and wet, and it is quite painful as currents of air contact it. The wound is hypersensitive to touch and blanches with pressure, and blood flow to the dermis is increased over that of normal skin. If infection is prevented, superficial partial-thickness burns heal spontaneously within 3 weeks without functional impairment. They rarely cause hypertrophic scarring, but in pigmented individuals the healed burns may never completely match the color of the surrounding normal skin.

Deep Partial-Thickness Burns. Deep partial-thickness burns (Fig. 13-5) also blister, but the wound surface is usually a mottled pink and white color immediately after the injury.

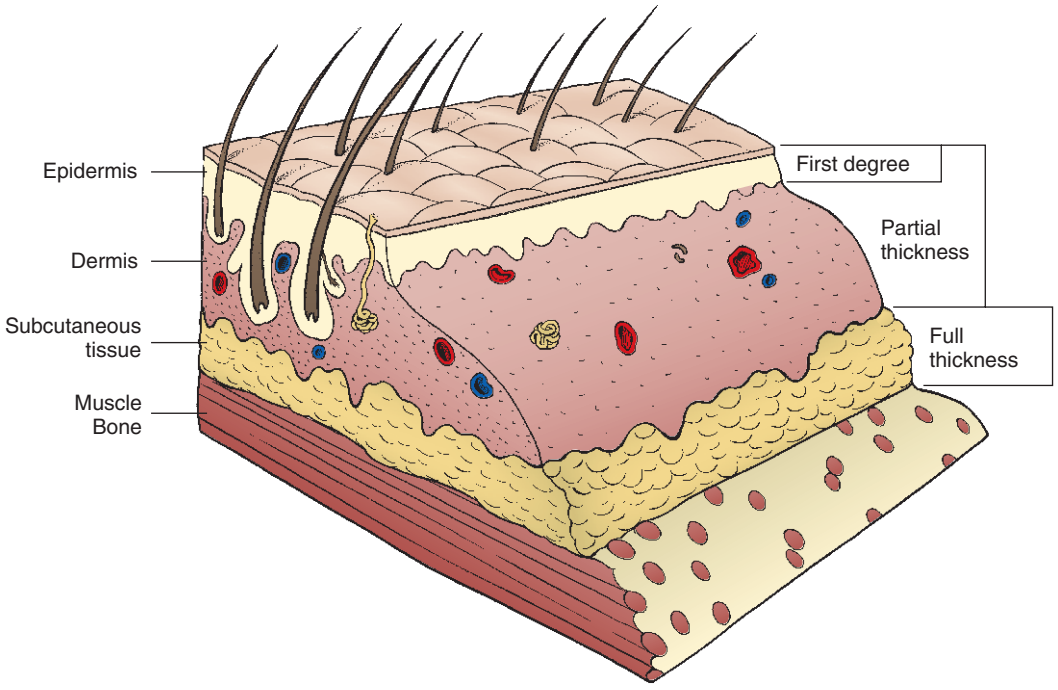

Figure 13-3. Skin anatomy.

Figure 13-4. Superficial partial thickness burn. Note pink color and moist surface.

Figure 13-5. Deep partial-thickness burn. Note cherry red color.

Figure 13-6. Full-thickness burn. Note thick, leathery, white eschar.

Alternatively, the burned dermis may be dry, with a cherry red color. The victim complains of discomfort rather than pain. When pressure is applied to the burn, capillary refill returns slowly or may be absent. The wound is often less sensitive to touch than the surrounding normal skin. By the second day, the wound may be white and is usually fairly dry. If infection is prevented, such burns heal in 3 to 9 weeks but invariably do so with considerable scar formation. Unless active physical therapy is continued throughout the healing process, joint function may be impaired and hypertrophic scarring, particularly in pigmented individuals and children, becomes inevitable.

Full-Thickness Burns. Full-thickness burns (Fig. 13-6) involve all layers of the dermis and can heal only by wound contracture, epithelialization from the wound margin, or skin grafting. Full-thickness burns are classically described as leathery, firm, depressed when compared with the adjoining normal skin, and insensitive to light touch or pinprick. Unfortunately, the difference in depth between a deep partial-thickness burn and a full-thickness burn may be less than 1 mm. Full-thickness burns are easily misdiagnosed as deep partial-thickness burns, because the two types have many of the same clinical findings. For example, they may be mottled in appearance. They rarely blanch with pressure, and they may have a dry, white appearance. The burn may be translucent with clotted vessels visible in the depths. Some full-thickness burns, particularly immersion scalds, have a red appearance and can be confused with superficial partial-thickness burns by the uninitiated. However, these red, full-thickness burns do not blanch with pressure. Full-thickness burns develop a classic burn eschar. An eschar represents the structurally intact but dead and denatured dermis that, over days to weeks, separates spontaneously from the underlying viable tissue.

Fourth-Degree Burns. Fourth-degree burns involve not only all layers of the skin but also subcutaneous fat and deeper structures. These burns almost always have a charred appearance, and frequently only the cause of the burn gives a clue to the amount of underlying tissue destruction.

TREATMENT

Care at the Scene

Flame Burns

The first responder must remove the injured person from the source of heat. Because of the potential dangers of smoke inhalation in closed areas, the rescuer in a fire must take extreme caution not to become a victim. Persons with burning clothing should be prevented from running and should be made to lie down to keep flames and smoke away from the face. If water is not immediately available, the flames can be smothered with a coat or blanket. If nothing is available, the victim should be rolled slowly on the ground. Once the burning has stopped, the clothes should be removed; some fabrics will continue to smolder and synthetic fabrics may melt, leaving a hot adherent residue on the victim that will continue the burning process.

Scalds and Grease Burns

The victim of a scald or grease burn must be removed from the source of heat. Any wet clothing or wrap should be removed, because fabric retains moist heat and may continue to burn skin that is in contact with the hot material. Accidents resulting from cooking indoors with grease are particularly hazardous. The startle response to a grease splatter may cause the victim to drop a pan of grease onto the fire, starting a kitchen fire that can rapidly become a dwelling fire.

Airway

Once flames are extinguished, primary attention must be directed to the airway. Any person rescued from a closed space or involved in a smoky fire should be considered at risk for smoke inhalation injury. If compressed oxygen is not available, a victim who is coughing independently should be encouraged to continue to do so. If the victim is unconscious or the airway status is in question, the victim should be placed supine and the airway manipulated manually via the chin lift or jaw thrust maneuver. When possible, 100% oxygen should be administered by a tight-fitting mask if smoke inhalation is suspected. If the victim is unconscious and if personnel are trained to insert an endotracheal tube, such a tube should be passed and attached to a source of 100% oxygen. If the airway has to be supported by a tight mask, the rescuers must be aware of the significant danger of aspiration of gastric contents. Air forced into the stomach distends it and causes vomiting. The mask prevents expulsion of the fluid, and the victim rapidly aspirates vomitus into the tracheobronchial tree. No unconscious supine victim should ever be left unattended.

Other Injuries and Transport

Once an airway is secured, the first responder should quickly assess for other injuries and then transport the victim to the nearest hospital.[9,23,68] Victims should be kept flat and warm and should be given nothing by mouth. Aside from establishment of an airway, further resuscitation is unnecessary if the victim will arrive at a hospital within 30 minutes. For transport, the victim should be wrapped in a clean, dry sheet and blanket. Sterility is not required.

Cold Application

Smaller burns, particularly scalds, may be treated with immediate application of cool water in hopes of limiting the extent

of injury. The application of cold water is controversial, but immediate cooling does decrease the pain, possibly by a decrease in thromboxane production. By the time several minutes have passed, or after arrival in the emergency department, further cooling is not likely to alter the pathologic process. Ice water should not be used except on the smallest burns. Using ice on larger burns can easily induce systemic hypothermia and associated cutaneous vasoconstriction that can extend the thermal damage.

Swelling

During transport, constricting clothing and jewelry should be removed from burned and distal parts, because local swelling begins almost immediately. Constricting objects increase the swelling, and the removal of tight jewelry in the presence of distal edema is time consuming once swelling has occurred.

Electrical Burns

Electrical burns are particularly dangerous, both to the victim and to the rescuer. If the victim remains in contact with the source of electricity, the rescuer must avoid touching the victim until the current can be turned off or the wires cut with properly insulated wire cutters. Once the victim is removed from the source of current, airway, breathing, and circulation must be checked. Ventricular fibrillation or standstill is a common accompaniment to a major transthoracic current; cardiopulmonary resuscitation should be instituted if carotid or femoral pulses are not palpable. If pulses are present but the victim is apneic, mouth-to-mouth resuscitation alone may be lifesaving. Cardiopulmonary resuscitation should continue until a cardiac monitor can be obtained, which will direct treatment with epinephrine for cardiac standstill or defibrillation for ventricular fibrillation. Once an airway is established and pulses return, a careful search must be made for associated life-threatening injuries. Electrocuted victims frequently fall from heights and may have serious head or neck injuries. The intense tetanic muscle contractions associated with electrocution may fracture vertebrae or cause major joint dislocations. Patients with high-voltage electrical injuries should be treated with spinal precautions, and splints if necessary, until fractures can be ruled out.

Chemical Burns

Whenever possible, chemical burns should be thoroughly flushed with copious amounts of water at the scene of the accident. Chemicals will continue to burn until removed; washing for 5 to 10 minutes under a stream of running water may limit the overall severity of the burn. No thought should be given to searching for a specific neutralizing agent. Delay deepens the burn, and neutralizing agents may cause burns themselves; they frequently generate heat while neutralizing the offending agent, adding a thermal burn to the already potentially serious chemical burn.

First Aid at the Scene for Smaller Burns

Not all burns need immediate medical attention. Burns less than 5% TBSA (excluding deep burns of the face, hands, feet, perineum, or circumferential extremity) can be treated successfully in a wilderness setting if adequate first-aid supplies are available and wound care is performed diligently. Except for the very shallow burn that heals within a few days, most burns should be seen by a physician within 3 to 5 days after injury.

Burns should be washed thoroughly with plain soap and water and dried with a clean towel. The water used should be suitable for drinking, but it need not be sterile or bottled. Any obviously dead skin should be peeled off (which may be painful) or trimmed with sharp manicure scissors (usually painless). Large (greater than 2.5 cm), thin, fluid-filled blisters should be drained and the dead skin trimmed to prevent a potential closed-space infection. Deep burns, as from a flame, are firm and leathery, usually do not blister, and do not require immediate debridement. A small tube of silver sulfadiazine cream and tubes of antibiotic (such as bacitracin) ointment should be available in the first-aid kit. Either may be used and should be spread lightly over the wound. The wound may then be wrapped in dry, clean gauze, which need not be sterile. Simple dressings (one type of topical cream, plain gauze) are sufficient. Some patients prefer nonadherent dressings, such as Telfa or Adaptic (sometimes known as "greasy gauze"), because they are less likely to stick to the wound during dressing changes. The same effect can be achieved by soaking (with water) a plain gauze dressing that appears stuck to the wound, waiting a few minutes, and then removing the dressing with additional water if necessary. Other dressings (hydrogels, silver-coated dressings, silicone gel sheets, calcium alginate) are available but are not necessary. The authors recommend simple antibiotic ointments and plain gauze dressings rather than "specialty" dressings for inclusion in first-aid kits. Although patients may prefer one dressing over another for various reasons, no dressing has been shown conclusively to accelerate the healing of burn wounds. The first-aid kit should be stocked with general-use supplies; soap and dressings designed specifically for burns are expensive and unnecessary.

Mobility of the wound area must be actively maintained, and concentrated efforts should be made to avoid dependent positioning, especially when the victim is resting or sleeping. Focal edema in a small burn wound can be painful and alarming and should be prevented with extremity elevation and active range-of-motion exercises several times a day. Wound care should be performed once a day if the outer dressing remains dry. A wet, sticky outer dressing needs more frequent wound care. If only the outer dressing is dirty, it may be changed as often as needed. For the quickest healing of superficial burns, daily wound care should remove all exudates and crust, both of which significantly retard wound healing.

When determining the type of dressing to apply to a burn wound, the following considerations are important:

- The dressing should cover the entire burned area (an enclosed dressing), leaving no burned skin exposed to air or air currents.
- The burn wound surface should stay moist, whereas the unburned skin surrounding the burn should not macerate.
- The dressing should be relatively light and not bulky; it should not limit the patient's ability to actively flex and extend all burned extremities and digits. Frequent flexion and extension prevent the burned skin from tightening and thereby assist with pain control and prevention of edema.

These principles are far more important than the choice of topical agent to cover the burn. Commonly used topical agents that might be carried in first-aid kits include antiseptic and antibiotic ointments and creams (e.g., bacitracin, Neosporin, Polysporin, double antibiotic ointment, triple antibiotic ointment, silver sulfadiazine) and nonantibacterial ointments (e.g., Aquaphor, Vaseline, A&D ointment). All of these agents are

Figure 13-7. Contact burn from stepping on hot coals. Small, flat blisters do not require debridement.

Figure 13-8. Contact burn from stepping on hot coals. Leaking blisters that have burst should be debrided (see text).

A

B

Figure 13-9. Partial-thickness burn to the dorsal hand from hot oil while cooking at camp. **A,** Blister is debrided 5 days after the injury and **B,** bacitracin ointment applied. (Photos courtesy Tim Platt-Mills, MD.)

acceptable and the use of a topical antimicrobial is not essential in the early postburn setting.

Technique of Burn Wound Debridement. Small intact blisters do not need debridement (Fig. 13-7). The blister environment serves as a sterile biologic dressing, preventing desiccation and (usually) pain. The intact blister should be protected from trauma and observed every few hours to ensure that the blister has not popped. If the blister opens, debridement is usually recommended because the leaking blister fluid may lead to crusting that can effectively "seal over" the wound, leading to a closed-space infection (Fig. 13-8).

Debridement of blisters is essentially painless so long as the blistered skin is cut and not peeled or torn. The burn should be washed with soap and water. Forceps are used to grasp the blistered skin and small (manicure) scissors can be used to cut off the blister roof (Fig. 13-9). Care should be taken to remove as much of the blistered skin as possible, leaving a uniform surface that can then be treated with a topical agent (Fig. 13-10). There will be no bleeding during this procedure. To ensure that the procedure will be painless, the body part to be debrided should be stabilized against a solid surface. This prevents motion on the part of the patient, and it optimizes control for the person performing the procedure.

Technique of Escharotomy. If an extremity burn is circumferential, and if the burn eschar is dry and leathery, performance of escharotomy is justified. This is even true in a wilderness

Figure 13-10. The burn seen in Figure 13-8, after debridement.

setting where monitoring equipment (such as Doppler ultrasound) is not available (but see later, under Emergency Department Care).

Escharotomy does not require an anesthetic because only insensate full thickness burn is incised. The burned extremity should be rinsed well and cleansed with soap and water if available. A scalpel is used to perform an incision through the eschar into the subcutaneous tissue. Ideally, only the eschar is incised, because subcutaneous fat is often viable and can cause troublesome bleeding. The change in tissue texture is usually easy to discern—a "give" is felt as the scalpel passes through the eschar and into the fat. After the long initial incision is made, a short, push-type maneuver is performed by laying the belly of the scalpel blade along the entire length of the incision to ensure that all constricting tissue has been freed. Frequently, a popping open of the incision occurs as the scalpel moves from one end of the incision to the other. The first incision is made along the lateral aspect of the extremity. The extremity should soften, and any signs or symptoms of ischemia should resolve within a few minutes. If this does not occur, a second incision should be made along the medial aspect of the extremity. The incision is made only through eschar—not through unburned skin. Bleeding is usually scant and can be controlled with digital pressure and, if necessary, topical clotting agents (e.g., oxidized cellulose). On completion of the procedure, a moist dressing, such as antibiotic cream or ointment or gauze soaked in normal saline, should be applied. A compression wrap and elevation of the extremity after the procedure will assist in maintaining hemostasis.

Emergency Department Care

The primary rule for the emergency physician is to "forget about the burn." Although a burn is usually readily apparent and is often a dramatic injury, a careful search for other life-threatening injuries must take priority. Only after an overall assessment of the victim's condition should attention be directed to the specific problem of the burns. Assessment of the nonthermally injured victim is presented in Chapter 20; the following paragraphs consider the specific problems encountered in the victim with burns.

Resuscitation

Since the 1970s, more than a dozen resuscitation plans have been suggested. The goal of all of them is to complete treatment with a living victim who has normally functioning kidneys and does not have cardiac failure or pulmonary edema. Nearly all of these plans use a combination of colloid and crystalloid solutions, but they vary considerably in the ratio of colloid to crystalloid, the timing of colloid administration, the sodium concentration of crystalloid solution, and, to a much lesser extent, the total volume of fluid given.[4,16,32,35,41,59,63,65] Some require frequent changing of solutions, others require mixing of solutions, and some require careful monitoring of the victim's electrolytes. Intense controversy rages over which resuscitation plan is best, but this need not concern the physician without a special interest in burn physiology. There is general agreement on a few facts. A victim with very large burns will probably need both colloid and crystalloid. Initially, capillaries are permeable to both crystalloid and colloid solutions. The capillary leak to albumin and other large molecules repairs itself between hours 6 and 24.

The choice of formulas for initial resuscitation is probably of relatively little consequence as long as the rate of fluid administration is modified according to the victim's changing requirements as the hours pass. Because of its simplicity, ease of administration, and need for little blood chemistry monitoring, the formula developed by Baxter, known as the Baxter formula, or Parkland formula, has been adopted by most hospitals and has been recommended officially by the American College of Surgeons Committee on Trauma.

According to the Baxter formula, crystalloid is given during the first 24 hours while the capillaries are still permeable to albumin, and then colloid is given during the second 24 hours when the capillary leak has presumably sealed. Rapid administration of crystalloid solution results in early expansion of depleted plasma volume, which will return cardiac output toward normal. Once the capillary leak has sealed, colloid (usually in the form of albumin) remains the most effective solution to maintain plasma volume without further increasing edema. The Baxter formula was derived to provide specific replacement of known deficits measured by simultaneous determinations of red blood cell volume, plasma volume, extracellular fluid volume, and cardiac output during burn shock.

The first 24-hour and second 24-hour calculations are shown in Box 13-1. The formula calls for the administration of lactated Ringer's solution, 4 mL/kg body weight per percent body surface burned during the first 24 hours after the injury. Half of this fluid should be given in the first 8 hours and the second half during the next 16 hours. Fluid therapy during the second 24 hours consists of the administration of free water in quantity sufficient to maintain a normal serum sodium concentration, as well as plasma (or other colloid) to maintain a normal plasma volume.

The adequacy of resuscitation can best be judged by frequent measurements of vital signs, central venous pressure, and urine output, and by observation of general mental and physical responses. Despite myriad new monitoring devices, urine output remains one of the most sensitive and reliable assessments of fluid resuscitation. In the absence of myoglobinuria, a urine output of 0.5 mL/kg/hr in adults and 1 mL/kg/hr in children

Box 13-1. Baxter (Parkland) Formula

FIRST 24 HOURS—RINGER'S LACTATE
4 mL/kg/% burn in 24 hours
One half in first 8 hours
One half in second 16 hours
Example: 70 kg man with 50% burn
4 mL × 50% (70 kg) = 14,000 mL in 24 hours
7000 mL in hours 1–8
3500 mL in hours 8–16
3500 mL in hours 17–24

SECOND 24 HOURS—ALBUMIN OR PLASMA AT MAINTENANCE
Maintain normal vital signs
Adequate urine output
Example: 70 kg man with 50% burn
250–500 mL plasma
2000–2500 mL D_5W

weighing less than 10 kg ensures that renal perfusion is adequate. The victim's sensorium gives an indication of the state of cerebral perfusion and oxygenation. The victim should be alert and cooperative; confusion and combativeness are signs of inadequate resuscitation or warn of other causes of hypoxia.

Patients with burns that involve less than 10% TBSA generally do not require any fluid resuscitation. They should stay well hydrated but should not be encouraged to force fluids. Patients with burns that involve between 10% and 20% TBSA usually do not require intravenous (IV) fluid resuscitation. They should be encouraged to drink fluids that contain electrolytes (e.g., Gatorade) and should be discouraged from drinking large amounts of water or sodas. Hydration status in these patients should be monitored simply by ensuring that the oral mucous membranes are moist and that urine output is roughly the same as it was before the injury occurred. Patients with burns that involve more than 20% TBSA should receive intravenous fluid resuscitation with crystalloid solution until they reach a facility where medical professionals can evaluate the need for other types of fluids.

Victims with burns of less than 50% TBSA can usually be resuscitated with a single large-bore peripheral IV line. Because of the high incidence of septic thrombophlebitis, lower extremities should be avoided as IV portals. Upper extremities are preferable, even if the IV line must pass through burned skin. Victims with burns larger than 50% TBSA or who have associated medical problems, are at the extremes of age, or have concomitant smoke inhalation should have additional central venous pressure monitoring. Because of the extremely unstable state of the circulation in victims with burns over 65% TBSA, such victims should be monitored in an intensive care setting where a Swan-Ganz catheter for measuring pulmonary capillary wedge pressure and cardiac output is available.

The presence of myoglobinuria alters the resuscitation plan. Myoglobinuria results from the destruction of muscle cells, with release of myoglobin (the red muscle pigment). This is most often a problem when there are associated crush injuries, electrical burns, or extremely deep thermal burns. Characteristic cola-colored urine is an indication to increase the amount of fluid given and to establish a diuresis of 70 to 100 mL urine per hour. An initial bolus of 12.5 g of mannitol with a repeat dose in 15 to 30 minutes should be considered.

Escharotomy

Careful monitoring of the peripheral circulation is required in victims with circumferential full-thickness burns of the extremities. The edema that forms beneath inelastic eschar increases tissue pressure to a point at which it exceeds lymphatic pressure, thereby further increasing edema. When the edema exceeds venous pressure and eventually approaches arterial pressure, it stops all circulation to the extremity distal to the constricted area.

The classic findings of compartment syndrome, usually considered to be pain, paresthesias, pulselessness, and tense swelling, may or may not be present in the burned extremity. Therefore, distal pulses should be carefully monitored with Doppler ultrasound, and if any of the clinical signs mentioned earlier occur, or if Doppler signals disappear, an escharotomy should be performed immediately.

Escharotomy performed in the hospital does not require an anesthetic, because only insensate full-thickness burn is incised. An incision is made through the eschar into subcutaneous tissue, first along the lateral aspect of the extremity and, if symptoms or signs do not improve, along the medial aspect. The incisions need not be as deep as the investing muscle fascia, and bleeding can usually be easily controlled with electrocautery and topical clotting agents. If arrival to the hospital will be within 6 hours, escharotomies should not be done in the field because the victim may bleed to death without proper equipment to control bleeding.

Circumferential full-thickness burns of the trunk in small children occasionally demand an escharotomy to improve pulmonary function. Chest wall escharotomies are made in the anterior axillary line bilaterally, extending from the clavicle to the costal margin. If the abdomen is involved with the burn, the inferior margins of the escharotomy may be connected transversely.

Fasciotomies are rarely needed in victims with thermal burns. However, if distal pulses do not return after medial and lateral escharotomies, fasciotomy should be considered. On the other hand, victims with electrical injuries frequently need fasciotomy. Careful monitoring is mandatory for all victims with electrical burns and with burns associated with soft tissue trauma or fractures. In these circumstances, loss of pulses is a strong indication for urgent fasciotomy under general anesthesia in the operating room.

The need for escharotomy in the burned hand is somewhat controversial. Fingers burned badly enough to require escharotomy are frequently mummified, and the lack of muscles in the fingers puts less tissue at ischemic risk. Escharotomy done in fingers that may not require it runs the risk of exposing the interphalangeal joints, leading to subsequent infection that may ultimately require joint fusion or finger amputation. Both the palmar arch and the digital vessels should be monitored with Doppler ultrasound in any significant hand burn. If the signals disappear over the palmar arch or in the digital vessels, consideration should be given to performing a dorsal interosseous fasciotomy.

Burn Wound Management

The burn wound should be cleansed initially with a surgical detergent. All loose, nonviable skin should be gently trimmed.

Debridement should be done gently; small doses of IV narcotic are sufficient analgesia for this procedure. General anesthesia and operating room debridement should be avoided until resuscitation is complete, unless other surgical procedures are necessary.

Once the wound is cleansed, a topical chemotherapeutic agent should be applied. A detailed description of all the agents available is beyond the scope of this chapter, but those most commonly used in the United States and other industrialized countries contain silver sulfadiazine. It comes as a white cream, is soothing to the wound, has a good antimicrobial spectrum, and has almost no systemic absorption or toxicity.[7,40,81,90] The victim should be carefully questioned about allergy to sulfa drugs before its use, however, because allergic reactions are encountered in about 3% of patients. These reactions may be manifested clinically as pain after application rather than the soothing feeling that silver sulfadiazine usually provides. If an allergy is suspected by history, a small (10 × 10-cm) patch of silver sulfadiazine should be applied as a test. The remainder of the wound can be dressed in bacitracin. If no local reaction occurs after 2 to 4 hours, an allergy is unlikely and silver sulfadiazine is the dressing of choice. If an allergy is confirmed, the victim should be referred to a burn center, where the next choice of topical antimicrobial would probably be silver nitrate solution.

Outpatient Burns

The vast majority of victims with burns do not require hospitalization. In many cases, the burn, if merely kept clean, heals spontaneously in less than 3 weeks with acceptable cosmetic results and no functional impairment. Unfortunately, good results in treating superficial minor burns may entice the unwary physician to treat more complex burns by the same methods. For the victim, the consequences of such a mistake can be the need for subsequent hospitalization, joint dysfunction, and hypertrophic scarring that may be difficult to correct, as well as considerable loss of time from work or school.

First-Degree Burns

Although first-degree burns are very painful, victims rarely seek medical attention unless the burned area is extensive. These victims do not require hospitalization, but control of the pain is extremely important. Aspirin or codeine may be adequate for small injuries, but for large burns, liberal use of a more potent narcotic for 2 to 3 days is indicated.

For topical medication, the authors recommend one of the many proprietary compounds containing extracts of the aloe vera plant in concentrations of at least 60%. Aloe vera has antimicrobial properties and is an effective analgesic.[49,77] Anecdotal evidence suggests that it may decrease subsequent pruritus and peeling.

Burns from ultraviolet rays (sunlight, sunlamp) may initially appear to be only epidermal, but the injury may in fact be a superficial partial-thickness burn with blistering apparent only after 12 to 24 hours. Therefore, the victim with such a burn should be cautioned about blisters and should be asked to return if they form, as wound management then becomes more important because of the potential for infection and subsequent scarring.

Superficial Partial-Thickness Burns

Treatment of superficial partial-thickness burns presents little problem. If the wound is kept clean, the victim is kept comfortable, and the joints are kept active, these wounds heal in less than 3 weeks with minimal scarring and no joint impairment.

Initially, the wound should be cleansed and debrided as described previously. Small blisters may be left intact. Biochemical analysis of the protein-banding pattern of burn blister fluid, obtained by polyacrylamide gel electrophoresis, has shown it to be similar to that of serum.[93] The authors suggest that the fluid is an exudate mainly from the vascular system, that it provides a good environment for the fibroblasts in the damaged site, and that it facilitates the healing process. Larger blisters are difficult to protect, however, and blister fluid is a rich culture medium for bacteria that live in the skin appendages. Therefore, large blisters and small blisters in large burns should usually be totally removed with forceps and scissors. In some instances the blister fluid can be aspirated with a large-bore needle, allowing the blistered epidermis to remain on the wound as a biologic dressing. This dead epidermis, however, is fragile, tends to contract, and rarely stays in place except over small areas.

After debridement, these wounds can sometimes be managed with a biologic dressing, such as porcine xenograft.[5,95] Pigskin is available in frozen or lyophilized forms. After a biologic dressing is applied, the burn pain is markedly diminished, and if the xenograft "sticks," no further treatment is necessary except for a periodic wound check. When the burn reepithelializes, the xenograft desiccates and peels away from the new epidermis. Other synthetic dressings, such as those made from plastic film (Op-Site or Epigard) have achieved some popularity.[11,69,88] We find that these dressings adhere poorly (a leaking dressing is not an occlusive one) and provide no advantage. The most commonly used treatment is wound coverage with silver sulfadiazine and application of a light dressing to promote active range of motion. Some very small burns do not require topical agents. For small facial burns, bacitracin ointment may be a better choice than silver sulfadiazine cream because it is less drying.

Pain is managed as for first-degree burns, and the patient usually should return every 2 to 3 days until the wound heals or the patient has demonstrated the ability to manage the wound without supervision.

One home treatment regimen is to have the patient cleanse the wound once daily with tap water and reapply the topical agent and light dressing. During dressing changes, and as often as possible, all involved joints should be put through a full range of motion. The dressing may be unnecessary while the patient is at home, but the patient should dress the wound before leaving the house. This method is highly successful, but it is inconvenient and fairly painful and requires good patient cooperation.

Another treatment regimen advocated by some physicians is a single initial debridement and application of a bulky dressing to be left in place for several days without intermediate cleansing or dressing change. This method reduces pain, but the patient must be careful of the bandage. If the bandage is allowed to become saturated with fluid draining from the wound or to become dirty, it may promote infection.

The "exposure" method has little to recommend it. This method involves leaving the wound open, allowing the wound drainage to desiccate and form a scab. Controlled studies in animals have shown that desiccation and crust formation interfere with wound healing. Our experience has also shown that

crusts crack over joints, cause considerable discomfort, and can hide infection.

Deep Partial-Thickness and Full-Thickness Burns

Treatment of deep partial-thickness and full-thickness burns is a matter of grave concern. Full-thickness burns heal only by contraction and epithelialization from the periphery. Epithelium does not begin to migrate until the eschar is removed; the growth rate then is only about 1 mm/day. Thus, healing of even a small full-thickness burn may involve many weeks of discomfort and disability.

Deep partial-thickness burns may take 4 to 8 weeks to heal and then leave an unacceptable scar. If a joint is involved, some loss of joint function is the rule. The authors have adopted a policy of early excision and grafting for such wounds.

Initial outpatient treatment can be followed by elective surgery as soon as it can be scheduled. Small wounds can be treated through day surgery; larger wounds located over dynamically important areas can be closed with only a day or two of hospitalization. The excision and grafting procedures should be done by a surgeon experienced in tangential wound excision.

The advantages of this aggressive approach—a pain-free patient with normal joint function, a better cosmetic result, and a rapid return to work or school—more than compensate for the brief hospitalization and the very small risk associated with minor operation.

Should the excision and grafting plan be unacceptable to the patient or the treating physician, the standard method of daily cleansing and application of silver sulfadiazine cream is used. Most full-thickness burns need grafting at about 3 to 4 weeks after the injury. Deep dermal burns should be seen by the physician frequently during the healing process, and active physical therapy is crucial to ensuring a successful outcome.

Rehabilitation

Physicians who regularly care for people with burn injuries recognize that treatment goals extend far beyond healing of the wounds and survival of the victim. The aim is to return victims at least to their preburn functional status physically and to ensure a smooth and timely reentry into family and social situations, including work or school. Awareness that recovery from burn injury often depends on a number of nonphysician health care workers is essential. Depending on the severity of the burn and the associated social situation, participation is commonly required of nurses, nutritionists, occupational therapists, physical therapists, recreational therapists, social workers, vocational rehabilitation counselors, psychologists, pain management specialists, and clergy.

Burn rehabilitation should be initiated by the first physician to see the victim. Once all systemic and wound issues have been addressed, proper positioning of wounded extremities or digits should be assessed by an occupational therapist who has been specially trained in burn management. Splints should be made immediately if deemed appropriate. Range-of-motion exercises should be started on the day of injury, and frequent follow-up by a physical therapist is essential. The best functional outcomes result from meticulous attention to early mobility. Patients almost universally choose not to move a burned body part, and an active ancillary burn staff is essential for satisfactory results. Burn scars require approximately a year to fade, soften, and mature. Physical therapy may be required throughout this time period or longer. Pressure garment therapy may be used in certain cases in an attempt to prevent the development of hypertrophic scars. The interested reader is referred to books dealing with acute burn care, reconstructive plastic surgery, and burn rehabilitation for further discussion.[1,8,14]

INHALATION INJURY

Of the nearly 500,000 fire victims admitted to hospitals each year, smoke or thermal damage to the respiratory tree may occur in as many as 30%.[71] Carbon monoxide poisoning, smoke poisoning, and thermal injury are three distinctly separate aspects of clinical inhalation injury. Inhalation injury rarely occurs in an outdoor setting.

Carbon Monoxide Poisoning

Pathophysiology. Carbon monoxide (CO) is a colorless, odorless, tasteless gas that has an affinity for hemoglobin 200 times greater than that of oxygen. The most simply explained mechanism of action of CO is reversible displacement of oxygen on the hemoglobin molecule. Although worsening hypoxia is important, and the percentage of carboxyhemoglobin in the blood represents in large measure the degree of victim hypoxia, this simple mechanism cannot account for all of the experimental and clinical findings seen with exposure to CO. For example, an experimental group of dogs was exchange-transfused with blood containing 80% carboxyhemoglobin (COHb). They showed no symptoms. In a control group with COHb levels of 80% produced by inhalation of CO, all animals died. Furthermore, the degree of enzyme and muscle impairment may not correlate accurately with the levels of blood COHb.[15,20,30,50]

In vitro, CO combines reversibly with cardiac muscle myoglobin and heme-containing enzymes, such as cytochrome oxidase (a_3).[15] Despite its intense affinity, it readily dissociates according to the laws of mass action. The half-life of COHb in humans breathing room air is 4 to 5 hours. In humans breathing 100% oxygen, the half-life is reduced to 45 to 60 minutes.[54] In a hyperbaric oxygen chamber at 2 psi, the half-life is 30 minutes, and at 3 atmospheres in a chamber it is reduced to 15 to 20 minutes.[92]

Clinical Presentation. Blood levels of COHb provide a laboratory measure to correlate with associated symptoms of CO poisoning. Levels less than 10% do not cause symptoms, although victims with exercise-induced angina may show decreased exercise tolerance. At levels of 20%, healthy persons complain of headache, nausea, vomiting, and loss of manual dexterity. At 30%, they become confused and lethargic and may show depressed ST segments on an electrocardiogram. In a fire situation, this level may lead to death as the victim loses both the interest and the ability to flee the smoke. At levels between 40% and 60%, the victim lapses into coma, and levels much above 60% are usually fatal.

Therapy. Victims who have not been unconscious and who have a normal neurologic examination on admission almost always recover completely without treatment beyond administration of 100% oxygen. Victims who remain comatose once COHb levels have returned to normal have a poor prognosis,

and in the authors' experience they rarely awaken. Although enthusiasts for hyperbaric oxygen treatment (HBOT) consider it a standard of care for CO poisoning,[2,3,52,61] many physicians are skeptical.[78] One controlled study indicated that hyperbaric oxygen made no difference for moderate poisoning, and multiple treatments were no better than a single treatment for victims with severe poisoning.[72] Furthermore, when associated with a major burn, transport to a chamber delays definitive care and is associated with numerous complications, including emesis, seizures, eustachian tube occlusion, aspiration, hypocalcemia, agitation requiring restraints or sedation, arterial hypotension, tension pneumothorax, and cardiac arrhythmia or arrest.[34,87] The issue of whether, and at what time in the progression of CO poisoning, HBOT may be of value will undoubtedly be continued to be studied and debated.

Thermal Airway Injury

Pathophysiology. The term *pulmonary burn* is a misnomer. True thermal damage to the lower respiratory tract and lung parenchyma is extremely rare unless live steam or exploding gases are inhaled. The air temperature near the ceiling of a burning room may reach 540°C (1000°F) or more, but air has such poor heat-carrying capacity that most of the heat is dissipated in the nasopharynx and upper airway. The heat dissipation in the upper airway, however, may cause significant local thermal injury.

Clinical Presentation. Victims who have been in explosions (propane, natural gas, or gasoline) and have burns of the hands, face, and upper torso are particularly at risk for pharyngeal edema.

Therapy. Maintenance of the airway is the main concern with potential thermal airway injury. Victims injured in explosions should be examined for oropharyngeal erythema and edema. If these are present, the victim should be intubated for 24 to 72 hours until edema subsides. A simple test to determine if intubation should continue is to see if the victim can breathe around the endotracheal tube when the cuff is deflated. If so, the airway edema has probably resolved and extubation should be safe. If doubt exists, extubation should be performed over a fiberoptic bronchoscope or nasogastric tube, which allows easy replacement of the endotracheal tube if necessary. Since this is not a pulmonary parenchymal injury, the purpose of intubation is to protect the airway and not necessarily to assist with ventilation. Ventilator settings should be adjusted accordingly, and vigorous pulmonary toilet should be instituted to prevent the pulmonary problems (atelectasis and pneumonia) commonly seen in intubated victims.

Smoke Poisoning

Pathophysiology. Some 280 separate toxic products have been identified in wood smoke. Modern petrochemical science has now produced a wealth of plastic materials in homes and automobiles that when burned produce nearly all of these and many other products not yet even characterized.[45,66,85,99] Prominent byproducts of incomplete combustion are oxides of sulfur, nitrogen, and many aldehydes. One such aldehyde, acrolein, causes severe pulmonary irritation and edema in concentrations as low as 10 ppm. Although the chemical mechanisms of injury may be different with different toxic products, the overall end-organ response is reasonably well defined.* There is an immediate loss of bronchial–epithelial cilia and decreased alveolar surfactant. Microatelectasis, and sometimes macroatelectasis, results in and is compounded by mucosal edema in small airways. Wheezing and air hunger are common symptoms at this time. After a few hours, tracheal and bronchial epithelia begin to slough and hemorrhagic tracheobronchitis develops. In severe cases, interstitial edema becomes prominent, resulting in a typical picture of the adult respiratory distress syndrome (ARDS). Pulmonary alveolar macrophages are poisoned, causing severe impairment of chemotaxis, which undoubtedly contributes to the high incidence of late pneumonia seen in victims with associated cutaneous burns. The activated neutrophils release superoxides and free radicals of oxygen, which together with other inflammatory mediators aggravate alveolar-capillary damage, leading to increased interstitial edema and impaired oxygenation.

Clinical Presentation. Any victim who has been indoors or in an enclosed space with a smoky fire and has a flame burn should be assumed to have smoke poisoning until proven otherwise. The acrid smell of smoke on the victim's clothes should raise suspicion. Rescuers are often the most important historians and should be carefully questioned.

Careful inspection of the mouth and pharynx should be done early. Hoarseness and expiratory wheezes are signs of potentially serious airway edema or smoke poisoning. Copious mucus production and carbonaceous sputum are sure signs, but their absence should not raise false hopes that injury is absent. COHb levels should be obtained; elevated carboxyhemoglobin levels or any clinical symptoms of CO poisoning are presumptive evidence of associated smoke poisoning. In very smoky fires, COHb levels of 40% to 50% may be reached after only 2 to 3 minutes of exposure.[92]

Anyone with suspected smoke poisoning should have a set of arterial blood gases drawn. One of the earliest indicators is an improper ratio of Pao_2 to Fio_2 (the P/F ratio). Normally the ratio is 400 to 500, whereas patients with impending pulmonary problems have a ratio of less than 300 (e.g., a Pao_2 of less than 120 with an Fio_2 of 0.40). A ratio of less than 250 is an indication for vigorous pulmonary therapy and not an indication for merely increasing the inspired oxygen concentration.

A number of authorities suggest the routine use of fiberoptic bronchoscopy.[12,55,74,81] It is inexpensive, quickly performed by an experienced clinician, and useful for accurately assessing edema of the upper airway. Aside from the presence of tracheal erythema, however, it does not materially influence the treatment for smoke poisoning. Therefore, it should be used only when the diagnosis is in doubt or for experimental studies.

We have correlated by multivariate analysis a constellation of history, signs, and symptoms with bronchoscopic findings in 100 consecutive patients with suspected smoke inhalation admitted to the University of Washington Burn Center in Seattle. If the patient had the combination of history of closed-space fire, carbonaceous sputum, and a COHb level greater than 10%, there was 96% correlation with positive bronchoscopy. Presence of two of the above features dropped correlation to 70%, and if only one was present, the correlation dropped to 36%. As discussed previously, upper airway edema was best correlated with an explosion (flash burn) that involved both the

*References 10, 17, 39, 42, 53, 54, 75, 83, 98, 99.

face and the upper torso. Nearly 50% of such victims had significant upper airway edema and underwent prophylactic airway intubation.

Therapy. All victims burned in an enclosed space or having any suggestion of neurologic symptoms should be given 100% oxygen while awaiting measurement of COHb levels. This should be administered through a tight-fitting mask in the field. If the victim demonstrates labored breathing or if a prolonged transport time is anticipated, endotracheal intubation should be performed by trained personnel. Oxygen at 100% can then be administered by ventilator.

Mucosal burns of the mouth, nasopharynx, and larynx respond with edema formation and may lead to upper airway obstruction at any time during the first 24 hours after the burn. Red or dry mucosa or small mucosal blisters should alert the observer to the possibility of subsequent airway obstruction; they also should raise suspicion that significant smoke inhalation may have occurred. Any victim with burns of the face should have a careful visual inspection of the mouth and pharynx; if abnormalities are found, the larynx should be examined immediately on arrival at the hospital. The presence of significant intraoral and pharyngeal burns is a clear indication for early endotracheal intubation, because the progressive edema may make later emergency intubation extremely hazardous, if not impossible.

The mucosal burns themselves are rarely full thickness and can be successfully managed with good oral hygiene. Once inserted, the endotracheal tube should remain in place for 2 to 5 days until the edema subsides.

Pulmonary functions early in the course of smoke poisoning are variably affected. Typically, there are decreased lung volume (functional residual capacity) and vital capacity, evidence of obstructive disease with reduction in flow rates, increased dead space, and rather rapid decrease in compliance. Surprisingly, much of the variability in pulmonary response appears to correlate with the severity of the associated cutaneous burn.[14] Without associated burns, the mortality from smoke poisoning is low, the disease rarely progresses to ARDS, and symptomatic treatment usually leads to complete resolution of symptoms in a few days. In the presence of burns, smoke poisoning appears to approximately double the rate of mortality from burns of any size. Pulmonary symptoms (hypoxia, rales, rhonchi, wheezes) are seldom present on admission but may appear 12 to 48 hours after exposure. In general, the earlier the onset of symptoms, the more severe is the disease.[91]

No standard treatment has evolved to ensure survival after smoke poisoning; each recommended treatment modality is tempered by opinion and the individual experience of the treating physician. In the presence of increasing laryngeal edema, nasotracheal or orotracheal intubation is indicated. A tracheostomy is never an emergency procedure and certainly should be avoided as initial airway management in victims with burns to the face and neck. A soft-cuffed endotracheal tube should be left in place for 3 to 5 days until the generalized oropharyngeal edema subsides.

Mild cases of smoke poisoning are treated with highly humidified air, vigorous pulmonary toilet, and bronchodilators as needed. Blood gases are drawn at least every 4 hours, and the P/F ratio is calculated. Worsening symptoms, difficulty in handling secretions, and a falling P/F ratio are all indications for intubation and respiratory assistance with a volume ventilator. If oxygenation is impaired (P/F ratio 250 or less), positive end-expiratory pressure (PEEP) or continuous positive airway pressure (CPAP) is initiated and increased by increments of 3 to 5 cm H_2O until no further improvement in the P/F ratio occurs or there is evidence of decreased cardiac output.

The physician must carefully search for other mechanical causes of poor ventilation (e.g., restricted chest wall motion from full-thickness burns, pneumothorax from high ventilator pressures, or mechanical difficulties with the endotracheal tube).

Prophylactic antibiotics have no value in this chemical pneumonitis, and the subsequent burn management and treatment of eventual bacterial pneumonia can be made more difficult by the development of resistant organisms if antibiotics are used early.

Steroids are commonly used in victims with severe asthma. Clinicians dealing with smoke poisoning often use them for their spasmolytic and anti-inflammatory actions. Several authors have studied the use of steroids, but a most convincing study comes from Moylan, who showed in a prospective blinded study of patients with smoke poisoning and associated major burns that the rates of mortality and infectious complications were higher in the steroid-treated patients. In patients with associated burns, Robinson and Seward[76] found that steroids did not alter the hospital course of patients admitted to the hospital after the MGM Grand and Hilton Hotel fires in Las Vegas in 1981.

The decision for hospital admission and the need for specialized care rest on the severity of symptoms from the smoke and the presence and magnitude of associated burns. Any victim who shows symptoms of smoke inhalation and has more than trivial burns should be admitted. If the burns are greater than 15% TBSA, the victim should probably be referred to a special care unit. In the absence of burns, admission depends on the severity of symptoms, the presence of preexisting medical problems, and the social circumstances of the victim. Otherwise healthy victims with mild symptoms (only a few expiratory wheezes, minimal sputum production, COHb level less than 10%, and normal blood gas levels) can be watched for an hour or two and then discharged if they have a place to go and someone to stay with them. Victims with preexisting cardiovascular or pulmonary disease should be admitted for observation if they have any symptoms related to the smoke. Victims with moderate symptoms (generalized wheezing, mild hoarseness, moderate sputum production, COHb levels 5% to 10%, and normal blood gas levels) may be admitted for close observation and treated as for asthma. The patient with severe symptoms (air hunger, severe wheezing, and copious [usually carbonaceous] sputum) requires immediate intubation and ventilatory support in an intensive care unit setting.

OTHER CONSIDERATIONS

Burns are tetanus-prone wounds. The need for tetanus prophylaxis is determined by the victim's current immunization status. The treating physician should follow the recommendations of the American College of Surgeons.

All victims undergoing IV resuscitation should have an indwelling urinary catheter placed for hourly monitoring of urine output. Arterial lines are useful in victims who need frequent assessment of blood gases or who will need repeated blood sampling. Necessary laboratory work during the resusci-

tation phase is relatively minimal. Blood should be drawn for baseline blood chemistries. If major operative procedures, such as fasciotomy or multiple escharotomies, are expected, blood should be sent for type and crossmatching in anticipation of needing several units of whole blood. Blood gases are mandatory in any victim with a suspected inhalation injury, and arterial pH measurement is useful as an assessment of the overall treatment of shock. If the Baxter formula is used for resuscitation, frequent electrolyte determinations are not necessary because levels will remain in the normal range. By 48 hours, however, careful monitoring of serum sodium and potassium becomes important. High levels of circulating aldosterone result in an increase in renal potassium excretion, and evaporative water loss to varying degrees through eschar dramatically increases the free water requirements of burned victims. Hemoglobin and hematocrit levels are initially high and remain high or normal until the third or fourth postburn day. The blood glucose level commonly is elevated because of the glycogenolytic effect of elevated catecholamines, the gluconeogenic effect of elevated glucocorticoid and glucagon levels, and relative insulin resistance.[44,82,94,100] This well-described "stress diabetes" can become a problem in normal patients if glucose-containing solutions are given during resuscitation, and it is frequently a serious problem in patients with preexisting diabetes. All diabetic patients require careful monitoring of blood and urine glucose, and most require supplemental insulin during resuscitation.

All medications during the shock phase of burn care should be given intravenously. Subcutaneous and intramuscular injections are unreliably absorbed systemically, and their use should be avoided. Pain control is best managed with small IV doses of morphine given until pain control is adequate without affecting blood pressure.

Before the discovery of penicillin, 30% of burn victims died during the first week after the injury from overwhelming β-hemolytic streptococcal sepsis. The availability of penicillin decreased streptococcal infections but had no influence on mortality or the incidence of bacterial sepsis. Victims then survived the first postburn week only to die of gram-negative penicillin-resistant bacterial sepsis during the second or third week after the burn. The advent of effective topical chemotherapeutic agents applied directly to the burn wounds made possible the control of streptococcal infection, obviating the need for prophylactic penicillin. The use of prophylactic antibiotics in outpatient burns has not been carefully evaluated, so opinion is divided regarding its utility in victims not requiring hospitalization.

Stress ulceration of the stomach and duodenum was once a dreaded complication, occurring in nearly 30% of victims with burns. Protection of the gastric mucosa by immediate feeding through a small nasogastric tube or, failing that, with instillation of antacids or sucralfate has made stress ulcers rare.[48,61,70,73,89]

Psychosocial care should begin immediately. The victim and family must be comforted, and a realistic outlook regarding the prognosis of the burns should be given, at least to the victim's family. In house fires, the victim's loved ones, pets, and many or all possessions may have been destroyed. If the family is not available, some member of the team, usually the social worker, should find out the extent of the damage in hopes of comforting the victim. If the victim is a child and the circumstances suggest that the burn may have been deliberately inflicted or resulted from negligence, physicians in most states are required by law to report their suspicion of child abuse to local authorities.

Burn Severity and Categorization

Severity of injury is proportionate to the size of the total burn, the depth of the burn, the age of the victim, and associated medical problems or injuries. Burns have been classified by the American Burn Association and the American College of Surgeons Committee on Trauma into categories of minor, moderate, and severe.[6] Moderate burns are defined as partial-thickness burns of 15% to 25% of TBSA in adults (10% to 20% in children), full-thickness burns of less than 10% of TBSA, and burns that do not involve the eyes, ears, face, hands, feet, or perineum. Because of the significant cosmetic and functional risk, all but very superficial burns of the face, hands, feet, and perineum should be treated by a physician with a special interest in burn care in a facility that is accustomed to dealing with such problems. Major burns, as described previously, most full-thickness burns in infants and older adults, and burns combined with diseases or injuries should also be treated in a specialized facility. Moderate burns can be treated in a community hospital by a knowledgeable physician as long as the other members of the health care team have the resources and knowledge to ensure a good result. Newer techniques of early wound closure have made burn care more complex, and an increasing number of victims with small but significant burns are being referred to specialized care facilities to take advantage of these concepts.

The criteria for admission to the hospital of victims with minor and moderate burns vary according to physician preference, the victim's social circumstances, and the ability to provide close follow-up. In some circumstances, superficial burns as large as 15% can be successfully managed on an outpatient basis. In other circumstances, burns as small as 1% may require admission because of the victim's inability or unwillingness to care for the wound. In general, the threshold for admission of older adults and infants should be low. Any victim (child or adult) whom the physician suspects of having been abused must be admitted.

Transport and Transfer Protocols

Once an airway is established and resuscitation is underway, burn victims are eminently suitable for transport.[9,22,46] Resuscitation can continue en route, because for the most part patients remain stable for several days. This was well proven during the Vietnam War, when burn victims were transported from Vietnam to Japan and then from Japan to the military burn center in San Antonio, Texas. The transport was generally accomplished during the first 2 weeks after the burn, with few complications occurring in about 1000 patients transferred.

Hospitals without specialized burn care facilities should decide where they will refer patients and work out transfer agreements and treatment protocols with the chosen burn center well in advance of need. If this is done, definitive care can begin at the initial hospital and continue without interruption during transport and at the burn center. In general, transfer should be from physician to physician, and contact should be established between them as soon as the patient arrives at the initial hospital.

The mode of transport depends on vehicle availability, local terrain, weather, and the distances involved. For distances of less than 50 miles (80 km), ground ambulance is usually satis-

factory. For distances between 50 and 150 miles (80 and 241 km), many people prefer helicopter transport. It should be noted, however, that monitoring, airway management, and any changes in therapy are more difficult to achieve in a helicopter. All victims transported by air should have a nasogastric tube inserted and be placed on dependent drainage, because nausea and vomiting usually result during the flight. Two large-bore IV lines should be functional in case one stops working.

For distances over 150 miles, fixed-wing aircraft are usually satisfactory. Modern air ambulances are completely equipped flying intensive care units, and the personnel are usually well trained for both critical care and the peculiarities of victim care during the flight (see Chapter 35).

The referring physicians must ensure that the victim's condition is suitable for a long transport and prepare the victim for the flight. The victim's airway must be secure. At 9144 m (30,000 feet), planes can be pressurized to an altitude of about 1676 m (5500 feet). Although supplemental oxygen can be given in flight, if the victim's oxygenation is marginal, performing intubation and starting mechanical ventilation before the transport may be preferable. Intubation is difficult en route, so if there is any question of upper airway edema, the victim should be intubated at the referring hospital. Burned victims have difficulty maintaining body temperature, and they should be wrapped warmly before transport. Bulky dressings, a blanket, and a Mylar sheet (usually available from the flight team) can help maintain body temperature. In case the victim has any cardiac irregularities, the plane must be equipped with electronic monitoring capability, because noise and vibrations in flight make clinical monitoring difficult.

Only after all other assessments are complete should attention be directed to the burn itself. If the victim is to be transferred from the initial hospital to a definitive care center during the first postburn day, personnel at the referring hospital can leave the burn wounds alone, merely calculating the size of burn for resuscitation, and monitoring pulses distal to circumferential full-thickness burns. The victim can be wrapped in a clean sheet and kept warm until arrival at the definitive care center.

The references for this chapter can be found on the accompanying DVD-ROM.

Exposure to Radiation from the Sun

14

Lee A. Kaplan

Sun exposure, sun damage, and sun protection are increasingly the focus of medical, scientific, and public attention. Adverse effects of sunlight overexposure are well documented,[84,143] but the relative value and safety of sun-protective strategies are debated. These issues are of particular concern to wilderness enthusiasts who spend considerable time in the sun. Sun protection against acute phototrauma, especially sunburn, is more easily judged than protection against chronic phototrauma—photoaging, cataracts, and photocarcinogenesis. Yet these chronic effects are increasingly relevant to modern societies with demographically aging populations. The economic concerns are huge: billions of dollars are spent annually in the cosmetic and medical industries to repair photodamage, photoaging, and skin cancer. Consumer interest in photoprotection is rapidly expanding. With better sunscreens and photoprotective clothing, even more investment will be focused on sun protection in the future.

SOLAR RADIATION

Electromagnetic Spectrum

The sun produces a continuous spectrum of electromagnetic radiation (Fig. 14-1). The most energetic rays, those with wavelengths shorter than 10 nm (cosmic rays, gamma rays, x-rays), do not appreciably penetrate to Earth's surface. Phototrauma is primarily the result of ultraviolet radiation (UVR). UVR (10 to 400 nm) accounts for about 10% of the incident radiation at Earth's surface; visible light (400 to 760 nm), 50%; and infrared (IR, 760 to 1700 nm), 40%.[84]

UVR has four components. Vacuum UVR (10 to 200 nm) is readily absorbed by air and does not penetrate Earth's atmosphere. UVC (200 to 290 nm) is almost entirely absorbed in the stratosphere, 15 to 50 km above Earth's surface, by oxygen and ozone. Man-made sources of UVC—germicidal lamps and arc welding devices—are rarely medically relevant. UVB (290 to 320 nm) is biologically quite active and principally responsible for tanning, burning, and nonmelanoma skin cancer (NMSC) formation.[84,143] Beneficial effects of UVB include vitamin D production from a cutaneous precursor, 7-dehydrocholesterol, to form previtamin D_3, which is quickly converted to vitamin D_3 and then metabolized, first by the liver, to form 25-hydroxyvitamin D_3, and then by the kidneys to form active 1,25-dihydroxyvitamin D_3.[129] Approximately 90% of 25-hydroxy vitamin D_3 is formed in this manner.[227] Of the UVR that reaches Earth's surface, UVB accounts for roughly 10%, whereas UVA (320 to 400 nm) accounts for about 90%, depending on the

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The sun produces a continuous spectrum of electromagnetic radiation (Fig. 14-1). The most energetic rays, those with wavelengths shorter than 10 nm (cosmic rays, gamma rays, x-rays), do not appreciably penetrate to Earth's surface. Phototrauma is primarily the result of ultraviolet radiation (UVR). UVR (10 to 400 nm) accounts for about 10% of the incident radiation at Earth's surface; visible light (400 to 760 nm), 50%; and infrared (IR, 760 to 1700 nm), 40%.[84]

UVR has four components. Vacuum UVR (10 to 200 nm) is readily absorbed by air and does not penetrate Earth's atmosphere. UVC (200 to 290 nm) is almost entirely absorbed in the stratosphere, 15 to 50 km above Earth's surface, by oxygen and ozone. Man-made sources of UVC—germicidal lamps and arc welding devices—are rarely medically relevant. UVB (290 to 320 nm) is biologically quite active and principally responsible for tanning, burning, and nonmelanoma skin cancer (NMSC) formation.[84,143] Beneficial effects of UVB include vitamin D production from a cutaneous precursor, 7-dehydrocholesterol, to form previtamin D_3, which is quickly converted to vitamin D_3 and then metabolized, first by the liver, to form 25-hydroxyvitamin D_3, and then by the kidneys to form active 1,25-dihydroxyvitamin D_3.[129] Approximately 90% of 25-hydroxy vitamin D_3 is formed in this manner.[227] Of the UVR that reaches Earth's surface, UVB accounts for roughly 10%, whereas UVA (320 to 400 nm) accounts for about 90%, depending on the

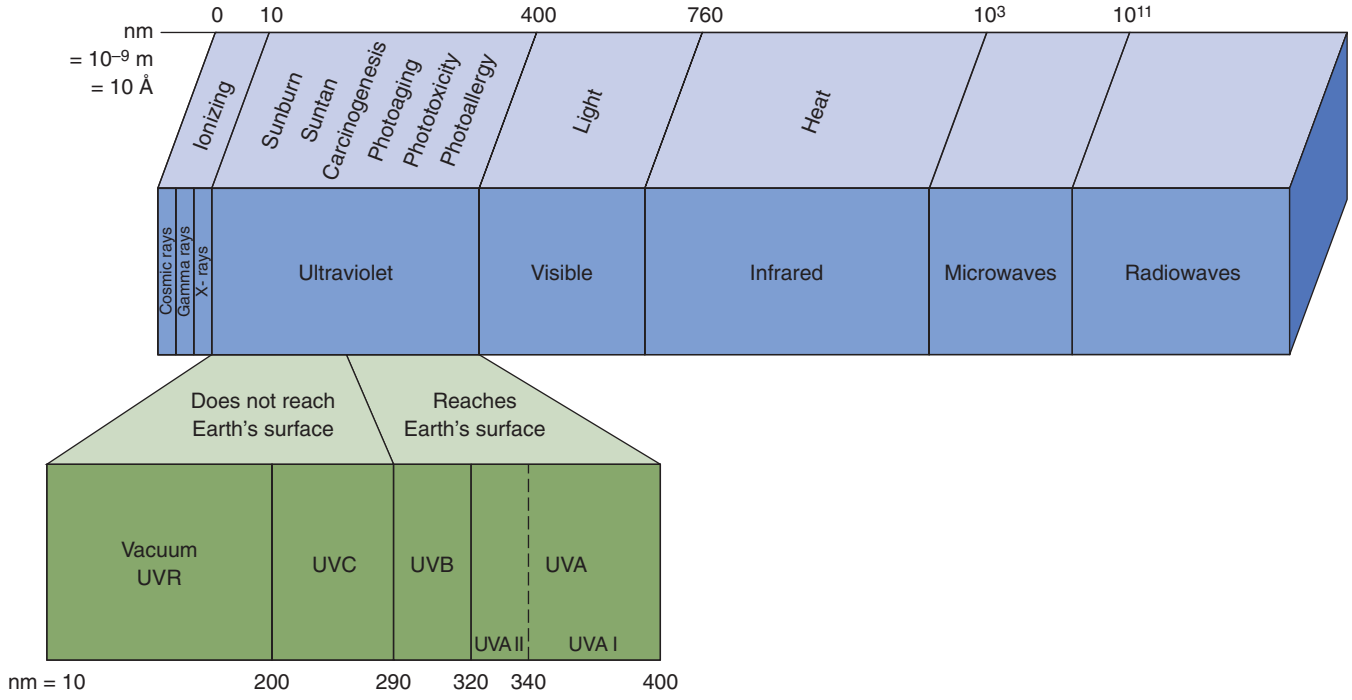

Figure 14-1. Electromagnetic spectrum.

season and time of day. UVA is typically subdivided into "near" UVA, or UVA II (320 to 340 nm), and "far" UVA, or UVA I (340 to 400 nm). These subdivisions, although somewhat arbitrary, are based on photobiologic responses. UVA is biologically active, contributing to tanning, burning, photoaging, and carcinogenesis. UVA penetrates the skin more deeply than does UVB, with less energy lost in the superficial layers of the stratum corneum and epidermis. In addition, UVA is the principal trigger for photodrug reactions. Longer-wavelength visible light and IR may also cause cutaneous phototrauma, albeit rarely. Solar urticaria has been reported to occur on exposure to visible wavelengths. IR alone, and along with UVR, may produce epidermal and dermal alterations.[150]

Environmental Influences on UVR Exposure

UVR intensity is substantially affected by latitude, altitude, season, time of day, surface reflection, atmospheric pollution, and ozone levels. Most UVR reaches Earth in midday: 80% between 9 AM and 3 PM, and 65% between 10 AM and 2 PM.[84] UVB peaks at midday when the sun is at its zenith.[175] UVB is absorbed, reflected, and scattered by the atmosphere, and at midday it has less atmosphere to traverse. In early morning and late afternoon, when the sun nears the horizon, UVB decreases considerably. Latitude and season have similar effects: peak UVB exposure is approximately 100 times greater in June than in December.[175] For each degree of latitude away from the equator, UVB intensity decreases an average of 3%. UVA varies considerably less than UVB with latitude, time of day, and season, as predicted by Rayleigh's law:

$$\text{Atmospheric light scattering} \propto 1/\lambda^4,$$

where λ = wavelength. The shorter the wavelength, the greater is the atmospheric scattering, so UVB is scattered much more readily than UVA. Additionally, UVA but not UVB is transmitted through window glass, allowing indoor exposure to UVA but not to UVB.

UVR may be increased by surface reflection. Water, surprisingly, is a relatively poor reflector. UVR at midday penetrates water up to 60 cm.[175] Reflection from water may increase as the sun nears the horizon, but at that time there remains little UVB to reflect because of atmospheric attenuation. Ice and snow are considerably better reflectors. Clean snow may reflect up to 85% of UVR,[175] accounting for oddly distributed sunburns in spring skiers. Sand, metal, concrete, and other surfaces reflect UVR to varying degrees.

Clouds may attenuate UVR exposure. Absorption by clouds varies from 10% to 80% but rarely exceeds 40%.[143] Polluted clouds—those containing the greatest concentration of hydrocarbons—are the most effective at absorbing UVR. Clouds are more effective at absorbing heat in the form of infrared radiation, occasionally seducing hikers and bathers into excessively lengthy midday exposures.

Wind and water augment sunburn. In mice, exposure to wind plus UVR results in more erythema than does exposure to UVR alone.[217] In humans, wind may reduce heat perception, encouraging longer exposure. Water increases UVR exposure because moist skin reflects less UVR, resulting in greater absorption of UVB. Consequently, swimmers and hikers in humid environments may be at risk for increased UVR absorption.[84]

Altitude profoundly influences UVB exposure. Until recently, it was generally accepted that UVB exposure rises 4% for each 305-m (1000-foot) rise above sea level. However, Rigel and

colleagues[237] demonstrated an 8% to 10% increase in UVB for each 305-m rise above sea level. Beginning at the 3353-m (11,000-foot) summit of a ski run in Vail, Colorado, these investigators measured UVB readings every 152 m (500 feet) as they skied to the base of the run at 2500 m (8200 feet). This study showed that UVB exposure in Vail at 2591 m (8500 ft) is similar to exposure levels in Orlando, Florida, 775 miles farther south.

Snow, wind, and altitude may act simultaneously to greatly augment UVB exposure for skiers and climbers. Singh and coworkers[260] reported that 36% of climbers (24 of 67) developed significant sunburns during three consecutive expeditions up to 7000 m (23,400 feet) in the western Himalayas, despite the application of sunscreen. In a year-long study of professional Alpine mountain guides using continuous dosimetry monitoring, Moehrle and colleagues[203] confirmed exceedingly high cumulative UVR exposures. These data emphasize the difficulties in providing appropriate UVR protection for high-altitude outdoor enthusiasts.

Ozone Depletion and UVR Exposure

Stratospheric ozone, 15 to 50 km above Earth's surface, provides a thin, fragile shield against UVR. The combination of ozone and oxygen absorbs virtually all of the incident UVC. Ozone attenuates UVB and modestly reduces UVA II, but it allows transmission of all UVA I.[304] Ozone is continuously created and removed from the stratosphere by natural physicochemical processes that are in turn significantly affected by human-made pollution. Molina and Rowland[204] first suggested that chlorofluorocarbons (CFCs) could cause ozone depletion. CFCs are organic chemicals containing carbon, chlorine, and fluorine, initially developed in the 1970s as refrigerants. Since then, CFCs have been used in several technologies and industrial processes: air conditioning systems, insulation, cleaning solvents, degreasing agents, and metered dose inhalers. Related compounds, halons, contain bromine, which also depletes stratospheric ozone. Halons arise from seawater, fire extinguishers, and various industrial processes.[125] CFCs and halons belong to the group of chemicals known as halocarbons.

The remarkable stability of CFCs allows them to rise into the stratosphere, where, catalyzed by solar radiation, they release Cl^- and chlorine monoxide (ClO^-), which in turn degrade ozone. One prominent chemical pathway[125] by which CFCs deplete ozone is as follows:

$$Cl^- + O_3 \rightarrow ClO^- + O_2$$
$$O_3 + UVR \rightarrow O + O_2$$
$$ClO^- + O \rightarrow Cl^- + O_2$$
$$\text{Net reaction: } 2O_3 + UVR \rightarrow 3O_2$$

Note that Cl^- is preserved in this reaction. The half-life of Cl^- is approximately 75 years,[291] although CFCs may reside in the stratosphere for 50 to 200 years.[125] During that time, each Cl^- ion may destroy 100,000 molecules of ozone.[291] Over Antarctica, additional and augmented ozone-depleting mechanisms may be operative; molecular halogens coat the surface of ice clouds, making them even more reactive and able to degrade ozone.[125]

Ozone losses were first reported over Antarctica in 1985[76] by the British Antarctic survey. Ozone showed large declines in the austral springtime (September/October), decreasing a total of 35% during springtimes from 1975 to 1984.[125] Ozone depletion is now documented at all latitudes except the equator. This depletion is uneven, with more loss at the poles and less at middle latitudes.[291] Using its total ozone mapping spectrometer (TOMS) in satellite orbit, the National Aeronautics and Space Administration (NASA) has documented ozone depletion over continental Europe, North and South America, South Africa, New Zealand, and Australia.[165]

Significant increases in UVB have been documented, even in middle latitudes, because of ozone depletion. In Toronto from 1989 to 1993, Kerr and McElroy[147] documented surface UVB increases of 35% per year in winter and 7% per year in summer, corresponding to ozone decreases of 4% per year and 1.8% per year, respectively. Their experimental design allowed the assignment of all of the UVB increase to ozone depletion rather than to cloud coverage or pollution. Similar increases in ground-level UVB, paralleling decreases in stratospheric ozone, have been documented in Scotland.[208] Typically, in the northern hemisphere's middle latitudes, ozone losses are greater in winter (about 6% per decade) than in summer (3% per decade).[125] Ironically, pollution—smog and particulates—may mitigate UVR increases by absorbing UVB.[291]

The effects of ozone depletion on the biosphere and human skin cancer rates have been estimated. Although estimates vary, data suggest that for every 1% decrease in ozone, there is a corresponding 2% increase in the incidence of basal cell cancer (BCCa) and 3% increase in the incidence of squamous cell cancer (SCCa).[58] Melanoma incidence may increase 1%.[291] However, even if UVB triples near the poles, as suggested in worst-case scenarios, polar areas will still receive less UVB than current equatorial levels.[291] Ozone depletion may have its greatest effects on nonhuman biosystems. Plant and plankton yields may be diminished significantly, with uncertain, potentially severe, detriment to terrestrial and marine life.[304]

To prevent ecologic disturbances and restore ozone levels, international agreements have been negotiated. The Vienna Convention in 1985 and the subsequent Montreal Protocol in 1987 were among the first and most notable. The Montreal Protocol, now signed by 184 of the 191 U.N. member nations,[282] agreed to limit, and then reduce, CFC production, leading to a 50% reduction by 1998.[125] Three later amendments were made to accelerate progress in response to increasing awareness of ozone depletion in middle latitudes. The London Amendment in 1990 required complete phaseout of halocarbon production. The Copenhagen Amendment in 1992 accelerated the timetable for complete phaseout, stopping production in developed countries by 1996 and undeveloped nations within 10 years, and it extended the protocol to include hydrochlorofluorocarbons. The Vienna Amendment in 1995 revised the phaseout schedule of hydrochlorofluorocarbons.[125,261]

Although the Montreal Protocol is working—CFC production has been decreased by 90%[282]—its modest limitations would allow elevated stratospheric chlorine levels through most of the 21st century.[125] In contrast, the stricter limitations of the Copenhagen Amendment will result in ozone levels rising again within the first decade of the 21st century.[261] Among the remaining sources of CFC production are metered dose inhalers and cleaning agents for rocket motor manufacturers.[125] American factories could produce 53,500 tons of CFCs annually for export until 2005.[23] CFC substitutes are more costly (leading to a black market in CFCs), with an estimated 22,000 tons produced in 1994.[23] A significant portion of this black market is in Freon smuggled to automobile air-conditioner rechargers. In 1996, an estimated 10,000 tons of CFCs were smuggled into

the United States from Mexico and elsewhere.[83] Despite these black market sources, initial projections suggested that atmospheric chlorine would begin to fall in this decade, with resultant UVB levels gradually falling by 2050 to levels recorded in the 1950s. Unfortunately, recent data suggest that current global warming trends exacerbate ozone depletion and may delay recovery of the ozone layer.[282]

ACUTE EFFECTS OF ULTRAVIOLET RADIATION ON SKIN: SUNBURN AND TANNING

The effects of UVR on skin depend primarily on wavelength, length of exposure, intensity of exposure, repetition of exposure, age at time of exposure, site of exposure, and genetic factors of the individual exposed. To have a biologic effect, UVR must be absorbed. Absorbing molecules in the skin are known as chromophores, and different wavelengths of UVR are absorbed by different chromophores. Among these are nucleic acids, especially pyrimidine bases, various amino acids in cutaneous proteins, and lipoproteins in cell membranes. These chromophores are at various depths in the skin, accounting in part for the differing photobiologic responses to UVR. To date, we have only imprecise, but improving, knowledge of how a given photochemical reaction results in a specific biochemical product, leading to an observable clinical change.

UVC

UVC has no appreciable impact on human health, as it is effectively screened from Earth's surface by stratospheric ozone and oxygen. Even with continued ozone depletion, levels of UVC are not expected to rise. As mentioned, human-made sources of UVC—germicidal lamps and arc welding devices—have been only rarely associated with cutaneous pathology, as might be predicted, because UVC is effectively absorbed by the stratum corneum, the outermost cutaneous layer.[84]

UVB

UVB acutely induces a cutaneous inflammatory response that is at least partially definable clinically, histologically, and biochemically.[134] Clinically, erythema, or sunburn, is the hallmark of acute overexposure to UVB. The action spectrum for erythema peaks in UVB.[210] Generally, UVB is considered 1000-fold more effective than UVA at inducing erythema. In a human model, 300 nm UVB is 1280-fold more effective at inducing erythema than 360 nm UVA.[307] We usually define erythemogenic doses as multiples of the minimal erythema dose (MED)—the lowest dose that elicits perceptible erythema. In a typical fair-skinned individual, the MED might range from 15 to 70 mJ/cm^2 for UVB and 20 to 80 J/cm^2 for UVA.

One MED of UVB for a typical fair-skinned individual would require 20 minutes of midsummer exposure in San Diego, whereas an MED of UVA would require 2 to 3 hours of exposure. In a day's time, a person can receive 15 MEDs of UVB but only 2 to 4 MEDs of UVA.[194] Consequently, although we are exposed to 10-fold to 100-fold more UVA than UVB, more than 90% of sunlight-induced erythema is attributable to UVB. The erythema action spectrum, which peaks in UVB, is remarkably similar to the absorption spectrum of DNA,[210] suggesting that DNA is a principal target chromophore for UVB-induced erythema[306] and pyrimidine dimer formation. Supporting this is the finding that pyrimidine dimer yields correlate with erythema.[114]

Sunburn reflects a local vascular reaction. The causes are multifactorial: DNA damage, prostaglandin activation, cytotoxicity, and other mechanisms are implicated. UVB erythema has its onset 2 to 6 hours after exposure; it peaks at 12 to 36 hours and fades at 72 to 120 hours.[84,134,143] Acute histologic changes accompanying UVB exposure include edema with vasodilation of upper dermal vasculature[134] and endothelial cell swelling, most likely caused by the release of vasoactive mediators.[111] Delayed histologic changes include appearance of sunburn cells in as little as 30 minutes after exposure. These dyskeratotic cells have enlarged nuclei and vacuolated cytoplasm. Initially, sunburn cells are localized in the lower half of the epidermis, but by 24 hours they are also found in the upper half. These sunburn cells may represent actively cycling, proliferating basal cells that cannot adequately repair UVR-induced DNA lysosomal damage.[54] Beginning 1 hour after exposure, stainable Langerhans' cells—cutaneous antigen-presenting cells—are reduced by 25%, and by 72 hours after exposure only 10% remain.[134] In mice exposed to repetitive suberythemogenic doses of UVB, normal numbers of Langerhans' cells return by 8 days after exposure.[134] Vacuolization of melanocytes is seen after 1 hour, returning to normal 4 to 24 hours after exposure.[134] Mast cells decrease in number and granularity within 1 hour, returning to normal in 12 to 72 hours.[134] By 24 to 48 hours after exposure, there is increased melanin synthesis, epidermal proliferation, and thickening of the stratum corneum. Functionally, the epidermal permeability barrier diminishes after UVB exposure because of the altered kinetics of lamellar body–containing cells in the exposed epidermis.[130]

Biochemical changes accompanying sunburn include increased levels of histamine,[134] which return to normal within 74 hours. However, histamine is unlikely to be the sole or even the principal mediator of vasodilation and erythema, because antihistamines are ineffective in preventing sunburn. UVR induces increased phospholipase activity, with accompanying increases in prostaglandins (PGs). PGD_2, PGE_2, PGF, and 12-hydroxyeicosatetraenoic acid (12-HETE) are increased in suction blister aspirates immediately after UVB, peaking in 18 to 24 hours.[134] Topical and intradermal indomethacin, a prostaglandin inhibitor, blocks UVB-induced erythema for 24 hours after exposure,[263] supporting the thesis that eicosanoids (PGs and leukotrienes) are significant mediators of UVR-induced inflammation.[156] UVB stimulates induction of proinflammatory and mutagenic cytokines: tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and IL-12.[130] UVR generates free radicals in the skin that probably contribute to the sunburn reaction[188] by causing peroxidative chromosomal, membrane, and protein damage.[16] Topical antioxidants may mitigate sunburn when applied before but not after exposure. Melatonin[16] and vitamin C[56] act protectively by scavenging UVR-generated free radicals; neither absorbs UVR.

UVA

UVA penetrates more deeply into the skin than does UVB. Whereas 95% of incident UVB is reflected or absorbed by the epidermis, nearly 50% of UVA reaches the dermis.[75] UVA contributes modestly to sunburn and may cause clinical erythema. Prolonged daily UVA exposure can approach 125 J/cm^2, significantly exceeding the threshold erythema dose of 20 to 80 J/cm^2.[266] Clinically, UVA erythema has an onset within 4 to 6

Box 14-1. Sunburn Treatments

PAIN CONTROL
Acetylsalicylic acid
Nonsteroidal anti-inflammatory drugs

SKIN CARE
Cool soaks, compresses
Nonmedicated moisturizers
Topical anesthetics
- Prax lotion (pramoxine)
- Sarna anti-itch lotion (menthol plus camphor)
- Aveeno anti-itch concentrated lotion (pramoxine plus camphor plus calamine)
- Neutrogena Norwegian Formula soothing relief moisturizer (lidocaine plus camphor)

STEROIDS
Topical
Systemic

hours, peaks in 8 to 12 hours, and fades in 24 to 48 hours.[134,143] Erythema resulting from UVA may have a distinct pathophysiologic mechanism. UVA-induced erythema may be caused by keratinocyte cytotoxicity.[134] Histologically, UVA erythema displays more epidermal spongiosis, fewer sunburn cells, and more dermal changes than does UVB-induced erythema, with a denser, deeper mononuclear cell infiltrate and more vascular damage.[134]

Infrared

IR radiation plays a less well defined role in photodamage. Near-IR pre-irradiation prevents UVR-induced cytotoxicity,[194] suggesting that a possible evolutionary protective mechanism for IR is daily preparation of skin cells to resist UVR-induced damage. However, no data indicate whether IR protects against UVR's mutagenic and carcinogenic effects.

Sunburn Treatment

Sunburn is self-limited, and its treatment is largely symptomatic, involving local skin care, pain control, and anti-inflammatory agents (Box 14-1).[15] Cool water soaks or compresses may provide immediate relief, and topical anesthetics are sometimes useful. It is generally preferable to use the nonsensitizing anesthetics menthol, camphor, pramoxine, and lidocaine rather than potentially sensitizing anesthetics containing benzocaine and diphenhydramine. Refrigerating topical anesthetics before application provides added relief. A legion of topical remedies have been suggested anecdotally, including aloe, baking soda, and oatmeal, but controlled studies are lacking. Topical steroids may blanch the reddened skin. Diclofenac gel alleviates pain, erythema, and edema for up to 48 hours when applied after exposure.[148] Oral nonsteroidal anti-inflammatory drugs (NSAIDs) provide analgesia and may reduce sunburn erythema.[84] According to a recent *Medical Letter,* topical steroids and oral NSAIDs slightly decrease erythema in the first 24 hours if administered before exposure or shortly after exposure before sunburn becomes clinically apparent.[228] Few published studies support the value of systemic steroids, but they enjoy considerable anecdotal support. In a metastudy of all published articles related to sunburn treatment from 1966 to 2001, Han and Maibach[118] found no therapy to be unequivocally or consistently effective and no consensus for the treatment of sunburn. Moreover, education for acutely sunburned patients is lacking. Warnings regarding skin cancer are rarely given to sunburned patients when seen in the emergency room.[15]

Tanning

Tanning, like sunburning, is caused by UVR. Consequently, persons seeking a tan risk sunburn. Tanning is biphasic. After sun exposure, there is immediate pigment darkening (IPD) within minutes, followed by delayed pigment darkening (DPD) in 3 days. IPD is predominantly due to the action of UVA on preformed melanin precursors and occurs in as little as 5 minutes after exposure, peaks in 60 to 90 minutes, and then fades quickly. However, DPD represents new melanin synthesis within melanocytes and the subsequent spread of the richly melanized melanosomes into surrounding keratinocytes. After UVB exposure, DPD is notable by 72 hours, peaks in 5 to 10 days, and slowly fades. DPD is primarily a response to UVB. Most tanning studies have been performed with erythemal doses of UVR. However, multiple suberythemal exposures to UVA are significantly more melanogenic than similarly dosed UVB.[20] The mechanism of DPD is uncertain, but it is likely to be multifactorial. UVB stimulates tyrosinase release and arachidonic acid metabolites and releases α-melanocyte-stimulating hormone (α-MSH) from keratinocytes.[7] It also increases the binding affinity of melanocytes for MSH, resulting in increased melanocyte proliferation, melanization, and arborization.[30] UVB increases melanocytes in both exposed and protected human skin,[270] suggesting the possibility of a UVR-stimulated circulating factor that promotes melanocyte proliferation.

CHRONIC PHOTOTRAUMA

Natural Defenses and Skin Type

The absence of erythema does not preclude cutaneous photoreactions. Chronic exposure to UVR is accompanied by insidious cumulative biologic and clinical changes. Although some of these changes are due to broadband UVR, UVA and UVB often have distinct and different effects on the epidermis, dermis, extracellular matrix, cytokines, and immune response.[293] In response to UVB, the stratum corneum thickens[109] and melanin increases, both protective mechanisms to mitigate further UVB photodamage. The stratum corneum, the outermost layer of skin, is composed of flattened anucleate keratinocytes. It reflects, scatters, or absorbs up to 95% of incident UVB, depending largely on its thickness.[120] With repeated exposure to UVB, the stratum corneum can increase its thickness up to sixfold.[84] As a consequence, the stratum corneum is the main photoprotective factor in whites.[109] Repeated UVA exposures may cause some thickening,[174] but to a much lesser degree. Consequently, UVA tans are not as photoprotective as UVB tans.

Melanin reflects, scatters, and absorbs throughout the UVR spectrum; acts as an antioxidant; and reduces UVR-induced photoproducts.[153] Consequently, constitutive (racial) skin color is a principal determinant of an individual's erythemal response to UVR. Although blacks and whites have similar numbers of melanocytes, these pigment-forming cells are differently melanized and distributed in black and white skin. Increased melanin in blacks can decrease the dermal penetration of UVR up to fivefold[52] and increase the MED up to 30-fold. In con-

Box 14-2. Skin Types

I	Always burns. Never tans.
II	Often burns. Tans minimally.
III	Sometimes burns. Tans moderately.
IV	Burns minimally. Tans well.
V	Rarely burns. Tans deeply. Moderately pigmented (brown).
VI	Never burns. Deeply pigmented (black).

trast, tanning is much less protective. After an entire summer of tanning, the MED in whites increased only 2.3-fold.[52] Clinically, these racial differences in melanization are reflected in lower rates of burning, photoaging, and skin cancer in blacks.

Susceptibility to photodamage is typically defined by six distinct skin types (Box 14-2). Racial pigmentation alone does not account for differences in skin type. Some redheads tan easily, whereas some blacks burn readily. Reporting errors, typically overstating sun tolerance, are common, further complicating the interpretation of skin types.[229]

Skin type correlates well with MED. Additional factors that influence MED include age and anatomic site. Lower MEDs are recorded in the very young and the very old.[143] Differences in stratum corneum thickness and melanocyte concentration may account for body site–specific differences in MED. For example, the MED of the back is typically less than the MED of the lower leg.

Chronic suberythemal UVA exposures also cause photodamage. Repetitive, low-dose exposures to UVA result in histologic changes,[166,174] including thickening of the stratum corneum and the granular and stratified cell layers, decreased elastin, vascular dilation, and inflammation.

Aside from stratum corneum thickening and increased pigmentation, intrinsic mechanisms of photoprotection include antioxidants, such as the glutathione peroxidase-reductase system, that mitigate damage from UVR-induced reactive oxygen species. DNA repair enzymes correct most UVR-induced mutations. Carotenoids stabilize biologic membranes from singlet oxygen attack. Urocanic acid absorbs some of the UVR that penetrates the stratum corneum.

Photoaging

Long-term repetitive exposures to sunlight result in photoaging.[79] This process, known as dermatoheliosis, is distinctive clinically and histologically from chronologic aging; it is not merely accelerated chronoaging. Photoaged skin is characterized by dryness, roughness, mottling, wrinkling, atrophy, and pebbling and may be studded with precancers (actinic keratoses) or cancers (Fig. 14-2). Most of what we consider "old-looking" skin is in fact the result of photoaging and not chronoaging. Although age can be estimated from observing sun-exposed sites, it cannot be estimated from photoprotected sites.[289] The action spectrum for photoaging includes UVB, UVA, and infrared.[174]

Chronically sun-exposed sites have fewer Langerhans' cells. Keratinocytes and fibroblasts from sun-damaged skin have diminished lifespans in culture.[104] The most notable histologic change with chronic UVB exposure is deposition of thickened amorphous elastic fibers high in the dermis, demonstrable in photoexposed white skin by age 30. In a transgenic mouse model, UVB but not UVA produces this solar elastosis.[279] Another mouse model suggests that the action spectrum for "photosagging" peaks in UVA at 340 nm,[119] and that this action spectrum is remarkably similar to that for the generation of singlet oxygen by excitation of transurocanic acid. Those authors conclude that UVA causes photoaging because of its interaction with transurocanic acid, which releases reactive oxygen species.

With photoaging, elastin gene expression appears to be activated, although data are conflicting. Tropoelastin and fibrillin synthesis actually diminish with chronic UVB exposure.[292] Transcription of other extracellular matrix genes is enhanced. In particular, photoaged skin demonstrates increased matrix metalloproteinases, potent mediators of connective tissue damage.[79] These arise within hours of UVB exposure, even suberythemal exposure.[78] Interestingly, tretinoin inhibits induction of these UVB-induced proteinases,[78,79] perhaps explaining in part its clinical utility against photoaging.[215,290] Tretinoin also normalizes photoaltered epidermal differentiation and deposits new type I collagen in the upper dermis.[113]

Ocular Effects

Acute overexposure to UVB may cause photokeratitis, especially in skiers and climbers (snowblindness),[308] whereas chronic exposure to UVR may cause or contribute to pterygia, cataracts, and macular degeneration. A full discussion is presented in Chapter 25.

Sun and Skin Cancer

Cutaneous precancers and skin cancers (Fig. 14-3) are overwhelmingly caused by UVR exposure. In particular, UVR is the principal cause for NMSC—basal and squamous cell cancers—the most common of all cancers. UVR also contributes significantly to melanoma but is not a sine qua non. The incidence of skin cancer is staggering. One in five Americans[237] and two thirds of all Australians[13] born today will develop skin cancer. In addition, the incidences of both NMSC[107] and melanoma continue to increase.[236] Three causative factors seem to predominate:

- Lifestyle trends, including sun exposure, sunbathing, and tanning bed use
- Demographic aging of the population
- Ozone depletion

Since the 1890s, epidemiologic evidence has accumulated linking sun exposure and NMSC in humans. The incidence of skin cancer increases with increasing proximity to the equator.[53] NMSC has a far greater incidence in whites than in blacks and occurs primarily on sun-exposed areas. The risk of NMSC increases with increasing sun exposure, and repeated sunburn is an independent risk factor.[160] Adoption of a sun-protected lifestyle is associated with decreased risk.[127] Patients with xeroderma pigmentosum (XP), with deficient ability to repair UVR-induced DNA damage, have a 1000-fold greater risk of developing NMSC, typically at a very early age.[156]

In the 1920s, laboratory data confirmed that UVR induces and promotes NMSC in mammalian animal models.[143,275] UVC and UVB are effective inducers of SCCa in mice.[134] Although UVB is primarily implicated, UVA augments UVB-induced carcinogenesis,[275] and it can be a carcinogen on its own.

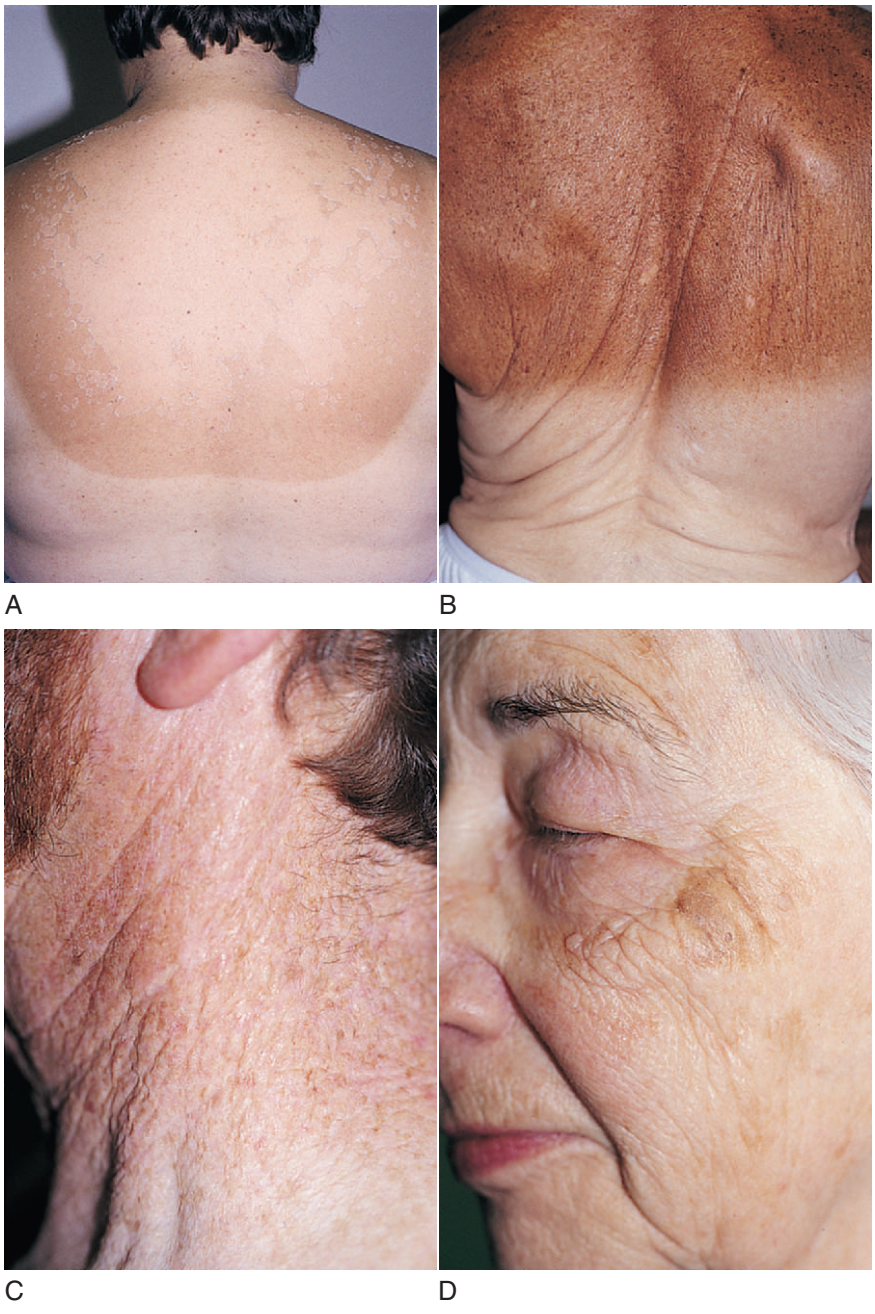

Figure 14-2. Phototrauma. **A,** Tan and peeling after sunburn. **B** to **D,** Dry, mottled, wrinkled, and pebbled skin with photoaging.

Development of NMSC in humans is related to the time and intensity of exposure. Sun exposure in childhood and adolescence is more predictive of later BCCa.[88] British immigrants to Australia assume the much higher Australian risk of NMSC only if they emigrate before age 18; after 18, immigrants retain the lower British risk.[184] A separate study suggests the risk decreases after age 10.[159] Gallagher and coworkers[88] found that BCCa is associated with sun exposure up to age 19 but not associated with mean annual cumulative summer sun exposure. In this study, risk for BCCa was also associated with fair complexion and freckling. Kricker and colleagues[160] suggest that intermittent, rather than continuous, sun exposure in poor tanners is the most important factor in the development of BCCa.

The relationship of photoexposure and SCCa is distinct and different from that of sun and BCCa. There is significantly increased risk of SCCa with chronic occupational exposure, especially in the 10 years before diagnosis.[89] Although cumulative lifetime photoexposure and SCCa are not associated, risk factors include periodic recreational exposure, pale complexion,

Figure 14-3. **A,** Actinic keratoses: thin, vague, scaly papules. **B,** Basal cell carcinoma: smooth, pearly papule. **C,** Squamous cell carcinoma: keratotic red/tan papule. **D,** Melanoma: note the asymmetry, border irregularity, color variegation, and diameter greater than 6 mm.

and red hair. Persons who develop SCCa seem phenotypically sensitive to UVR with chronic exposure as adults. Household and office exposure to nonsolar UVR does not increase the risk of BCCa and SCCa.[14] In select mouse models, suberythemal UVR causes SCCa.[210] In these models, gradual suberythemal exposures to UVR may actually be more carcinogenic than are erythemal doses.[87] This may be relevant to humans and explain why persons without any prior sunburns may develop SCCa.

Melanoma

Melanoma has a less well defined relationship to sun exposure. Clearly, however, sun exposure plays a significant role in its development.[6] The ultraviolet action spectrum for melanoma remains uncertain, with different results in different animal models. In *Monodelphis domestica*, a South American marsupial, both UVA and UVB are implicated[62]; in *Xiphophorus* (platyfish and swordtails), UVA, UVB, and visible (blue) light are causative[62]; and in transgenic mice Tyr-SV40E (C57BL/6 strain), UVB is the etiologic waveband.[149] In humans with XP, the 1000-fold increased incidence of melanoma strongly supports UVB as a causative agent.[157] A unique and elegant laboratory experiment documented that UVB induces melanocytic hyperplasia, atypia, and melanoma in newborn human foreskin xenografts on RAG-1 (immunodeficient) mice.[7]

Melanoma incidence increases with proximity to the equator in white populations[145] in the United States, Australia, and

Scandinavia, as well as in the nonwhite population of India.[163] In the U.S. Surveillance, Epidemiology, and End Results (SEER) Program, 1992 to 2001, the incidence of melanoma increased with lower latitudes in non-Hispanic whites; no association was noted in Hispanics or blacks.[69] In contrast, Hu and coworkers[135] noted a positive association of lower latitude and UVR exposure with melanoma in blacks and Hispanics in the United States, although statistical significance was reached for black men only. Intermittent intense exposures pose a particularly high risk for melanoma.[71] A meta-analysis of 57 studies published prior to September 2002 supports a significantly increased risk of melanoma with a history of intermittent sun exposure and sunburn.[91] By contrast, there is only a small increased risk for total sun exposure and there is a decreased risk with heavy occupational exposure.[73,91] Patients with melanoma are twice as likely to relate a history of a prior sunburn than are age-matched controls, and three times as likely to relate a history of multiple prior sunburns.[73] Persons who tan poorly and burn readily are at higher risk for melanoma.[176] The distribution of melanomas on the trunk (in both sexes) and lower legs (in women) is consistent with the hypothesis that intermittent sun exposure is provocative. However, melanomas also arise in sun-protected sites, especially in nonwhite populations, suggesting an additional cause separate from sun exposure.

A high level of outdoor activity in college is associated with a fourfold risk of later melanoma.[72] Servicemen who served in the Pacific theater during World War II have higher risks of melanoma than do those who served in Europe.[40] The incidence of melanoma is increased in indoor workers with higher socioeconomic status, again reflecting the role of intermittent intense sun exposure.[145] Fluorescent lights raise some concern[287] but do not significantly increase melanoma risk, especially when these lights have covers or diffusers.[132]

Sunburns in childhood may be particularly relevant to the later development of melanoma. Celtic migrants to Australia arriving before age 10 assume the high melanoma risk of native Australians; migrants older than 15 at arrival have only one-fourth that risk.[245] Europeans who live more than 1 year in a sunny climate have an increased relative risk (RR = 2.7) of melanoma, and the risk increases substantially if they arrive before age 10 (RR = 4.3).[10] However, one study suggests that childhood sun exposure contributes a serious risk only if there is subsequent and significant sun exposure as an adult.[8]

This association of increased melanoma risk with youthful sun exposure may be in part attributable to the effect of sun on the development of melanocytic nevi (moles) in children. Several studies confirm that the number of nevi in children increases with increasing acute and chronic sun exposure.[38,92,93,122,123,285] In Australia, the number of nevi up to age 12 increases with increasing proximity to the equator.[146] Increased nevus counts with sun exposure are demonstrable at early ages. In Queensland preschoolers aged 0-35 months, increased nevus counts are associated with more time spent outdoors and a history of sunburn.[122] These children have the highest number of nevi in the world, and, not surprisingly, Queensland has the highest incidence of melanoma in the world[123] at 55.8/100,000 for males and 42.9/100,000 for females.[13,27] The number of nevi is a strong predictor of melanoma risk. Melanoma increases with increasing numbers of benign acquired nevi. In a large European case-controlled study, the most important risk factors for melanoma are the number of nevi and the number of atypical nevi.[93] Established nevi may develop histologic changes transiently, simulating melanoma after a single UVR exposure,[278] and some data suggest that sunburn can induce malignant transformation in benign nevi.[47]

In the United States, melanoma incidence has increased since records were first kept in the 1930s, rising 121% in the 20 years between 1973 and 1994.[115] The American Cancer Society estimates that there were 54,200 new cases of invasive melanoma in 2003.[49] Today, the lifetime risk of melanoma for a male child born in the United States is 1 in 57; for a female child 1 in 81.[4] Similarly large increases in melanoma incidence have been noted in Europe, Australia, and even Japan.[145] However, melanoma incidence and death rates show signs of stabilizing and perhaps decreasing for younger cohorts,[106,115,145] probably as a result of improved sun protective behaviors.

Significant national efforts have been made to educate and protect populations at risk. In Australia, taxes have been removed from sunscreen sales, hats are typically required for children in outdoor sports, and artificial shade is increasingly available in public parks. In addition to improved photoprotection, early detection has been widely promoted. The Skin Cancer Foundation encourages monthly self-examinations, with special attention to pigmented lesions displaying atypical clinical features, the so-called ABCDEs of melanoma[225]: asymmetry, border irregularity, color variegation, diameter greater than 6 mm, and evolving features (see Figure 14-3). Nevi with these clinical features, symptomatic nevi, or nevi that bleed should be evaluated by a physician.

Molecular Basis of Photocarcinogenesis

Several photomolecular events are associated with NMSC. UVR causes characteristic photoinsults at sites of adjacent pyrimidines on DNA.[37,206,307] UVR photons are absorbed at the 5-5 double bonds, resulting in cyclobutane dimers if both bonds open and 6-4 (pyrimidine-pyrimidone) photoproducts if a single bond opens.[37] Cyclobutane dimers predominate 3:1, with thymine (T-T) dimers most common.[156] Suberythemal, as well as erythemal, doses of UVR can induce T-T dimers.[306] High-performance liquid chromatography can quantitatively measure these UVB photoproducts.[44] Such measurements indicate a 30-fold interindividual variation in photoproduct yields.[44]

Resultant UVR-induced mutations have a distinctive signature: two thirds display cytosine → thymine (C → T) substitutions at dipyrimidine sites; 10% show CC → TT substitutions.[37] These mutations are relatively unique to UVR damage and allow UVR-induced mutations to be distinguished from chemical mutations.[37]

Data suggest that cyclobutane dimers and 6-4 photoproducts are primarily responsible for the mutagenic[167] and carcinogenic[156] properties of UVR. Many genes may be targets for UVR mutations, including genes involved in tumor suppression and promotion. Tumor-suppressor genes (TSGs) that are known to play a role in photocarcinogenesis include p53, p16, and PTCH.[36,131] Mutation of p53, the most common genetic alteration identified in human cancers,[131] is found in nearly 50% of all cancers.[37,156] Approximately 50% of BCCa, 60% of actinic keratoses, and 90% of SCCa contain p53 mutations.[37,310] Although p53 mutations may be found in non–sun-damaged skin at a low frequency, 10^{-2} to 10^{-3}, mutations are much more common in sun-exposed sites.[37]

Normal p53 protein is a transcription factor, regulating the cell cycle. DNA damage stimulates p53 protein production, which leads to cell cycle arrest in G_1 (a premitotic phase), allowing time for DNA repair.[37] Irreparable damage leads to apoptosis, or programmed cell death. Sunburn cells are examples of apoptotic cells.[310] Cells with mutated p53 are more resistant to apoptosis with subsequent UVR exposures, explaining why p53 inactivation reduces sunburn cells in irradiated mouse skin.[310]

Within the p53 gene there are "hot spots," codons where mutations frequently occur.[37,43] Most p53 mutations result in a single amino acid substitution, typically cytosine to thymine (C → T).[310] Although normal, wild-type p53 has a short half-life and is generally unstable and unstainable, mutated p53 is significantly more stable and consequently stainable by immunohistochemical techniques.[43,237] Both UVA and UVB upregulate p53 expression in human skin.[46] After a single UVR exposure to the forearms, p53 protein expression peaks in 24 hours, returning to baseline after 360 hours.[116]

Mutations in p53 occur as an early, initiating event in photocarcinogenesis.[156] In actinic keratoses, unique p53 mutations are present throughout the lesion,[310] confirming that they occur before, not after, lesion formation. Different actinic keratoses display different p53 mutations, again supporting the role of p53 mutations as initial causative events, followed by clonal expansion of mutated cells to form clinically visible lesions of precancerous actinic keratosis.[310] Altered p53 provides a survival advantage to mutated cells. In response to chronic UVR, neighboring nonmutated cells become apoptotic and die, allowing space for further expansion of the mutated clone with ongoing UVR exposure[37] and ultimately resulting in the development of actinic keratosis or SCCa.

However, p53 mutations alone may be insufficient to produce NMSC. Patients with Li-Fraumeni syndrome, who inherit a mutated form of the p53 gene, have an increased incidence of sarcomas, adenocarcinomas, and melanomas, but not NMSC.[156] Consequently, other factors, such as decreased DNA repair and UVR-induced immunosuppression, must play a permissive role. In patients with XP, for example, there are increased numbers of p53 mutations[37] and defective gene repair mechanisms, and greatly increased numbers of NMSC. DNA repair may also be defective in normal patients who do not have the syndrome who develop BCCa at an early age,[288] and in older adult patients[206] who have a higher incidence of NMSC.

Cutaneous lymphomas may also show a higher frequency of UVR signature p53 mutations.[192] In mycosis fungoides, these mutations are found in the tumor stage but not the plaque stage,[192] suggesting that UVR promotes clinical progression of this lymphoma. These findings are particularly interesting in view of epidemiologic data demonstrating an increased incidence of non-Hodgkin's lymphoma in those living closer to the equator.[1]

Other TSGs are less well studied but have increasingly defined roles in photocarcinogenesis. In melanoma, p16 is frequently inactivated[136] and increasingly downregulated as melanoma progresses.[265] Another TSG, PTCH, also located on chromosome 9, is frequently mutated in familial, as well as sporadic, BCCa. Mutations of PTCH are especially notable in patients with XP and multiple BCCa.[136]

In addition to downregulating TSGs, UVR can activate proto-oncogenes to form functional oncogenes.[136] Included in this group are the proto-oncogenes *bcl*-2, c-*fos,* and *ras*. In response to UVR, the Bcl-2 protein is overexpressed, with resultant suppression of apoptosis and consequent permissive expansion of malignant clones. Additionally, UVR alters c-*fos,* disrupting transcription of nuclear proteins involved in cell proliferation. UVB also causes mutations in *ras,* disrupting mitogenic signaling pathways. In particular, mutations in BRAF, a critical component of the Ras protein kinase pathway, are found in a large percentage of nevi[164] and melanomas,[221] especially melanomas that arise on intermittently sun-exposed skin.[181] However, BRAF mutations occur only rarely in melanomas arising in chronically sun-exposed or completely sun-protected skin,[181] suggesting multiple genetic pathways for melanoma induction. Moreover, mutations of BRAF and p53 may interact to form melanoma.[221]

Oxidative damage is another mechanism, in addition to mutation, by which UVR contributes to photocarcinogenesis. UVA, although less directly mutagenic than UVB, is more potent at causing cellular oxidative damage, producing reactive molecular oxygen and nitrogen species, which, in turn, damage DNA, proteins, and lipids. This damage contributes to carcinogenesis via inflammation, immunosuppression, and, ultimately, mutation.[117] In addition, a group of zinc-dependent enzymes, known as matrix metalloproteinases (MMPs), increase in response to UVR, altering the cutaneous extracellular environment to favor tumor invasion and spread.[39,136]

Photoimmunology

UVR produces local and systemic immunosuppression.[212] Locally, UVB depletes Langerhans' cells, which are immunocompetent antigen-processing cells (APCs), for up to 2 weeks after exposure.[201] In addition, UVB functionally alters Langerhans' cells, interfering with their presentation of antigens to T cells,[110] diminishing helper T cell (Th)-1 responses (which promote contact hypersensitivity) while preserving Th2 responses[247] (which suppress contact hypersensitivity), effectively converting Langerhans' cells from immunogenic to tolerogenic. Antigen-specific suppressor T cells arise, their appearance possibly mediated by UVR-induced synthesis of IL-10 from keratinocytes.[247] As a consequence, contact hypersensitivity and mixed lymphocyte reactions are diminished with UVR exposure. This may explain, at least partially, why UVR-induced skin cancers, which are antigenic, progress to clinical lesions. In mice, this UVR-induced immunosuppression can be transferred with irradiated T lymphocytes.[212] Other proposed mediators of UVR-induced immunosuppression include cis-urocanic acid,[57] TNF-α, prostaglandins,[210] and DNA photoproducts.[202]

Immunoregulatory failure contributes to skin cancer formation.[162] Skin cancer is often more aggressive and occurs at an earlier age in immunosuppressed patients.[74,216] In Australia, 45% of kidney transplant patients develop skin cancer within 11 years and 70% within 20 years.[33] For heart transplant patients in Australia, the incidence of skin cancer is 31% at 5 years and 43% at 10 years, and skin cancer accounts for 27% of patient deaths in this group after the fourth post-transplant year.[216] In these patients, the ratio of SCCa to BCCa is 3:1, precisely opposite the ratio present in nonimmunosuppressed patients. These figures are unusual only because most studies suggest that the risk of skin cancer is higher after heart transplant than after kidney transplant because of higher doses of immunosuppressive drugs with the former. Chronic sun exposure and fair complexion further increase risks of skin cancer

in transplant patients,[216] possibly resulting from overexpression of p53.[101]

PHOTOPROTECTION

Sunscreens

Chemical sunscreens were discovered in 1926. By 1928, the first commercial sunscreen, containing benzyl salicylate and benzyl cinnamate, was marketed in the United States.[84] Subsequent sunscreen evolution was primarily directed toward UVB protection to mitigate development of sunburn from overexposure to the sun. Para-aminobenzoic acid (PABA) was first advocated as a sunscreen in 1942, patented in 1943, and made commercially available in 1960. By then, national advertisements for sunscreens were blazing the message, tan without burning. Sun protection factor (SPF)-15 formulations became available only around 1980. More recently, as UVA photodamage has been increasingly appreciated, UVA sunscreening agents such as avobenzone have been introduced. Micronized preparations of titanium dioxide (TiO_2) and zinc oxide (ZnO) have become popular, providing broad-spectrum protection throughout the UVR range. Previously considered "physical" (reflective) as opposed to "chemical" (absorptive) sunscreens, these micronized preparations blur the traditional distinctions. Micronized TiO_2 and ZnO are largely transparent and absorb, reflect, and scatter UVR.[155] Consequently, chemical and physical labels for sunscreens are of diminishing value and perhaps should be dropped.

In the United States, sunscreens are regulated over-the-counter (OTC) drugs. The U.S. Food and Drug Administration (FDA) *Final Over-the-Counter Drug Products Monograph on Sunscreens,* published in the Federal Register on May 21, 1999, establishes the allowable sunscreening agents, testing procedures, and labeling claims for efficacy, water resistance, and safety.[81]

The current FDA-approved sunscreening ingredients are listed in Table 14-1. Two names are sometimes given for the same agent because the U.S. Pharmacopeia (USP) changed the names of several sunscreening agents, effective September 1, 2002, to better conform to international standards.[280] PABA is an effective UVB absorber that has fallen out of favor. Poorly water soluble, PABA must be formulated in an alcohol vehicle. On the skin, it binds epidermal proteins, enhancing its resistance to water wash-off (substantivity) but provoking contact and photocontact dermatitis in approximately 4% of exposed subjects. Further limiting its acceptability, PABA can permanently stain fabrics a dull yellow color. As a consequence, PABA has been largely replaced by PABA esters—amyl dimethyl PABA (padimate A) and octyl dimethyl PABA (padimate O). These absorb well in the UVB range, are easier to formulate in nonalcoholic vehicles, and are less staining and less allergenic.

Cinnamates are the next most potent UVB absorbers. They often replace PABA in PABA-free sunscreens, but octyl methoxycinnamate (octinoxate; Parsol MCX) is an order of magnitude less potent than padimate O.[171] Octocrylene, a cinnamate derivative, is a weak UVB absorber that also absorbs UVA modestly up to 360 nm. Cinnamates are poorly bound to the stratum corneum and may cause contact dermatitis. Cinoxate is the most frequent contact sensitizer, with cross-sensitization to related cinnamates in coca leaves, balsam of Peru, and cinnamon oil.[65]

TABLE 14-1. Sunscreening Agents Approved in the United States

SUNSCREEN	MAXIMAL SCREEN (%)
Aminobenzoic acid	15
Avobenzone	3
Cinoxate	3
Dioxybenzone	3
Ecamsule*	2
Homosalate	15
Menthyl anthranilate (meradimate)	5
Octocrylene	10
Octyl methoxycinnamate (octinoxate)	7.5
Octyl salicylate (octisalate)	5
Oxybenzone	6
Padimate O	8
Phenylbenzimidazole sulfonic acid (ensulizole)	4
Titanium dioxide	25
Trolamine salicylate	12
Zinc oxide	25

Adapted from U.S. Food and Drug Administration: Sunscreen Drug Products Final Monograph, May 21, 1999.
*Approved by FDA July 21, 2006.

Salicylates, including homosalate and octyl salicylate (octisalate), are relatively weak absorbers of UVB and are consequently most often used in combination with other sunscreening agents. They have the advantages of being nonsensitizing and water insoluble, and they help solubilize benzophenones in commercial products.[84]

Anthranilates are similarly weak UVB absorbers that also filter UVA. They display peak absorption at 340 nm.[171] The single commercially available agent from this class is methyl anthranilate (meradimate).

Phenylbenzimidazole sulfonic acid (PBSA; ensulizole) is a unique UVB absorber. Unlike other chemical absorbers, which solubilize in the oil phase of emulsion formulations, PBSA is water soluble. This physiochemical property has resulted in its increasing use in oil-free cosmetic sunscreens.[171]

Benzophenones are broader-spectrum sunscreening agents, with good absorption in the UVB and UVA ranges up to 360 nm.[171] The three benzophenones available in the U.S. market are oxybenzone, dioxybenzone, and sulisobenzone.

In the United States, dibenzoylmethanes are represented by a single agent—avobenzone (Parsol 1789, butyl methoxydibenzoylmethane). It is a potent UVA absorber, with little or no absorption in the UVB range.[171] Its absorption peak at 358 nm falls nearly to zero at 400 nm.[242] Concerns have been raised that photodegradation may limit its effectiveness. Under simulated solar light, avobenzone can be degraded 36% in as little as 15 minutes.[251] However, these data are disputed, and there is debate whether any significant decrease occurs with typical use, especially given the mitigating effect of combined sunscreening agents in commercially available preparations.[251] For example, a patented complex known as Helioplex—a combination of avobenzone, oxybenzone, and diethyl 2,6-naphthalate—is remarkably photostable.[50a] Additionally, select stabilizing compounds, such as vitamin C, vitamin E, and iron chelators, may retard photodegradation.[193] Ecamsule (Mexoryl SX) is the

newest sunscreening agent approved by the FDA, although it has been available in Europe and elsewhere for several years. It is an excellent UVA filter, with maximal absorbance at 345nm, and with modest absorption in the UVB range as well. Ecamsule is distinguished by being highly photostable and thermostable. It effectively protects skin from repeated exposures to UVA, preventing the histologic changes associated with photoaging.[256]

Physical sunscreens traditionally refer to opaque agents that primarily reflect, scatter, and, to a lesser extent, absorb UVR. These include calamine, ichthammol, iron oxide, kaolin, red veterinary petroleum, starch, talc, TiO_2, and ZnO. In the wilderness, extemporaneous physical blockers can be made from ashes, mud, and leaves. A sunscreen with a physical filter typically protects throughout the UVR and visible spectra and may even protect against IR-induced erythema.[218] However, classic physical blockers are messy, uncomfortable, and cosmetically undesirable.

Preparations of TiO_2 and ZnO with a smaller (micronized or microfine) particle size are now available. Their particle size of 0.015 to 0.035 µm[63] makes them more soluble in their vehicle base and minimally reflective of visible light, which makes them transparent, or nearly so, in thin coats. The lower refractive index of ZnO in the visible range makes it less white and more transparent than TiO_2.[200] These micronized preparations significantly absorb UVR, providing broad-spectrum protection from UVB and UVA and blurring the distinction between chemical and physical sunscreens (as mentioned previously).[63,171,250] TiO_2 and ZnO are sometimes marketed as "chemical-free" sunscreens, clearly a misnomer. Additionally, products containing "physical" sunscreening agents are often marketed as "sunblocks," a misleading term eliminated in the 1999 FDA *Final Monograph*.[81]

New sunscreens are on the horizon, with imaginative observation leading to future sources of sunscreening agents. For example, Saikawa and colleagues[248] determined that the viscous "red sweat" of the hippopotamus is an excellent, broad-spectrum sunscreen, with absorption in the 200 to 600 nm range.[248]

Sunscreen Vehicles. Sunscreen vehicles affect efficacy and acceptability. The ideal vehicle spreads easily, maximizes skin adherence, minimizes interaction with the active sunscreening agent, and is noncomedogenic, nonstinging, nonstaining, and inexpensive. In practice, the best vehicle is highly dependent on personal preference. Creams and lotions (emulsions) are most popular. Both are oil-in-water or water-in-oil preparations, although lotions spread more easily. Most sunscreening agents are lipid soluble, accounting for an objectionable, greasy feel. Increasingly popular "dry lotions" minimize the lipid component and often include at least one water-soluble sunscreening agent to reduce oiliness.[171] In contrast, sunscreen oils contain only a lipid phase. Oils spread easily, but thinly, limiting the achievable concentration of active sunscreening agent and thereby limiting protectiveness. In addition, oils are oily and cosmetically less acceptable. Other anhydrous vehicles—ointments and waxes—may be desirable for climbers and winter campers, reducing the risks of chapping and frostbite. Gels are generally nongreasy but tend to wash or sweat off easily. In addition, gels seem to produce more stinging and irritation. Sticks typically incorporate sunscreening agents into wax bases, but application of stick preparations to larger areas is not easy. Aerosols are wasteful, with spray lost to the air, and usually form an uneven film,[171] and they need to be rubbed in to provide adequate, uniform protection.[18] Increasingly, sunscreens are being incorporated into cosmetics, including foundations, lipsticks, and moisturizers.

Sun Protection Factor. The ability of a sunscreen to protect the skin from UVR-induced erythema is measured by the SPF. The SPF is defined as the ratio of UVR required to produce minimal erythema (1 MED) in sunscreen-protected as opposed to sunscreen-unprotected skin.[81,143] It can be represented by the following formula:

$$\text{SPF} = \frac{\text{MED of sunscreen-protected skin}}{\text{MED of unprotected skin}}$$

Testing conditions are standardized by the FDA.[81] The agent to be tested is applied at a standard concentration of 2 mg/cm^2. Testing is performed indoors with a solar simulator on the back between the beltline and scapulae. The SPF is typically determined on a panel of 20 (maximum, 25) subjects with skin types I, II, or III (see Box 14-2). The mean is then determined to be the approved SPF. Using indoor testing with a solar simulator, although more reproducible than outdoor natural sun exposure, may yield a falsely high SPF value. In outdoor testing of more than 30 sunscreens labeled SPF 15, none had an SPF greater than 12.[220] Several factors are responsible for lower SPF values with outdoor testing: sweating; clothing, towel, and sand abrasion; and application variability. In addition, solar simulators generally have reduced outputs of UVA compared with sunlight.[251]

SPF determinations use erythema as their measurable endpoint. Erythema is predominantly a result of UVB, not UVA, exposure. Consequently, SPF is primarily a measure of UVB protection. The relationship of SPF to UVB absorption is given in Table 14-2.

How high an SPF is necessary? Given that SPF 15 blocks 93% of UVB, some argue that SPF 15 is sufficient[182] and that higher labeling claims are misleading and costly for consumers. However, in several studies, higher SPF sunscreens conferred clinical and histologic benefits. In children on Florida beaches, a single application of SPF 25 sunscreen protected just as well as multiple applications of SPF 15 for up to 6 hours of exposure.[213] Histologically, an SPF 30 sunscreen provides better protection against sunburn cell formation than does an SPF 15 sunscreen.[141]

TABLE 14-2. Skin Protection Factor (SPF) and Ultraviolet B (UVB) Absorption

SPF	UVB ABSORPTION (%)
2	50.0
4	75.0
8	87.5
15	93.3
30	96.7
50	98.0

Sunscreen Application. Perhaps the most persuasive argument in favor of higher-SPF sunscreens derives from variations in application technique. In the real world, sunscreens are applied at much lower concentrations than the 2 mg/cm^2 at which they are tested. Used ad lib, sunscreens are typically applied in concentrations of 0.5 to 1 mg/cm^3.[21,253] The resultant SPF is considerably reduced, typically to about 50% of the labeled SPF for chemical sunscreens.[41] Some suggest the reduction is even greater and that the actual SPF is only 20% to 50% of the labeled value.[271] Physical sunscreens tend to be applied even more thinly, typically at two-thirds the concentration of chemical sunscreens, most likely because of their cosmetic visibility.[61] For TiO_2 products, application concentrations of 0.65 mg/cm^2 result in SPF values of only 20% to 30% of the intended, labeled value.[271] Not surprisingly, persons who burn more easily tend to apply thicker concentrations of sunscreen.[61] There is no difference in the applied concentrations of higher versus lower SPF formulations.

Uneven application further reduces sunscreen protection. Using a fluorescent dye, a pattern of spotty use emerges. Individuals typically cover the forehead adequately, but the temples, ears, and posterior neck are often undertreated or missed entirely.[99,173,234] Newly available sunscreens that contain disappearing colorants are popular, allowing visible assurance of complete coverage. Adequate coverage of just the chronically exposed areas (face, ears, and dorsal hands) requires 2 to 3 g,[171] requiring an 8-oz bottle of sunscreen every 80 to 120 days.

Application delay imposes a further decrease in protectiveness. In a study of families on Chicago beaches in summer,[239] sunscreen was applied only after arriving at the beach in 98% of families. The median delay from arrival to application to the last family member was 51 minutes.

Underapplication, uneven application, and delayed application of sunscreens result in unnecessary photoexposure and photodamage. In addition, for hikers, the concomitant use of sunscreen and insect repellent containing diethyltoluamide (DEET) lowers the effective SPF by 34%.[205] Balancing these issues with consumer concerns over SPF inflation, the FDA set a labeling limit of SPF 30+ in the 1999 *Final Monograph,*[81] effective May 21, 2001. This was subsequently extended to December 31, 2002, and later deferred indefinitely until the FDA develops a more comprehensive set of requirements to address UVA testing and labeling as well.

UVA Protection Factors. With our increased understanding of UVA-induced photodamage and the recent addition of better UVA blocking agents (avobenzone, ecamsule, and micronized TiO_2 and ZnO), more attention has been focused on measuring UVA protection. Several measures of UVA protectiveness have been suggested, but none have been widely accepted or used. Among them are the UVA protection factor (APF), analogous to the SPF.[243] The principal drawback to APF determination is related to the lengthy exposure times required to induce erythema. A second method, the phototoxic protection factor (PPF), shortens exposure times by sensitizing the skin to UVA with oral or topical psoralens. However, the relevance of PPF data to real-world conditions has been challenged. Other UVA protection factors involve measurement of UVA-induced immediate pigment darkening, suppression of UVA-induced sunburn cell formation in psoralen-treated skin, and UVA-induced photocurrent transmission through various media.[84] In its 1999 *Final Monograph,* the FDA chose not to impose a specific UVA protection factor but allows a sunscreen to make UVA protection claims as long as it contains an approved UVA sunscreening agent.[81] However, Rosenstein and coworkers[245] found that sunscreens with a labeling claim of UVA protection allow transmission of 6% to 52% of UVA.

Substantivity. The ability of a sunscreen to resist water washoff is referred to as substantivity. "Water-resistant" sunscreens maintain their SPF after 40 minutes of immersion, "waterproof" sunscreens after 80 minutes. The 1999 FDA *Final Monograph*[81] changed these labels to "water resistant" and "very water resistant," respectively. Select sunscreening agents, primarily PABA and PABA-esters, are intrinsically substantive, a result of bonding to stratum corneum proteins. Other sunscreens must be incorporated into vehicles that confer substantivity. By applying sunscreen 15 to 30 minutes before water exposure, substantivity can be increased. Reapplication after swimming or sweating helps ensure protection. Today, dozens of affordable, high-SPF, waterproof (very water resistant) sunscreens are commercially available (Table 14-3).

Conditions for testing substantivity are set by the FDA.[81] Freshwater immersion is required in a pool, whirlpool, or Jacuzzi at 23° to 32°C (73.4° to 89.6°F). This raises some concerns about the relevance of substantivity labeling for saltwater ocean enthusiasts, including surfers, sailors, and scuba divers. The MED for skin bathed in salt water is less than the MED for skin bathed in fresh water, and both are lower than the MED of dry skin.[90] Furthermore, cold, churning water, sand abrasion, and toweling may add to sunscreen loss, begging the question of whether waterproof sunscreens are really surfproof. The concerns over saltwater substantivity, amplified by surfer folklore, have led to an almost cultish market of "surfshop" sunscreens, although few published data indicate whether these are more substantive in a saltwater environment.

Stability. Photostability—a sunscreen's ability to remain intact and effective after sun exposure—has been questioned for a number of sunscreens, most notably avobenzone, as noted earlier. However, questions regarding photostability have also been raised about octyl dimethyl PABA and octyl methoxycinnamate. Marot and colleagues demonstrated that the photodegradation of octyl methoxycinnamate allows histologic and enzymatic injury to the skin,[185] and that failure of a photolabile sunscreen may result in genotoxicity.[186] In a European study of 27 photoprotective lipsticks, 14 showed photoinstability—they became partially photoinactive after moderate UVR exposure.[180] Twelve were photolabile in the UVA range, one in UVB, and one in both UVA and UVB.

Photostability can be significantly improved by using combinations of sunscreening agents. For example, octocrylene stabilizes avobenzone in a number of sunscreen formulations, whereas 4-methylbenzylidene camphor can be used to stabilize octyl methoxycinnamate.[31] Vehicle formulations also affect stability; butyloctyl salicylate and butyloctyl benzoate contribute to photostability.[31]

There are few published data regarding the packaging, storage, and shelf life of sunscreens. Packaging may affect sunscreen acceptability and stability. For example, octyl methoxycinnamate turns PVC a bright yellow color. In a recent

TABLE 14-3. Select Sunscreen Products

PRODUCT	SPF	ACTIVE INGREDIENTS
High-SPF Waterproof Broad-Spectrum Creams/Lotions		
Bain de Soleil Oil-Free Protecteur Sunscreen Lotion	35	A, B, OC, S
Banana Boat Sport 50	30	B, C, OC, S
Blue Lizard Australian Sunscreen Lotion	30+	B, C, OC, ZO
Bullfrog SuperBlock Lotion	45	B, C, MA, OC, S, TI
Coppertone Sport Ultra Sweatproof	30	B, C, S
Dermatone Sunscreen Lotion	36	C, PBSA, ZO
Durascreen Lotion	30	B, C, PBSA, S, TI
Hawaiian Tropic Ozone Sunblock	70	B, C, PB, S, TI
Kiss My Face Sunblock Lotion	30	C, TI
Neutrogena Ultra Sheer Dry-Touch Sunblock	55	A, B, OC, S
Ocean Potion Anti Aging Sunblock	50	A, B, C, S
Ombrelle Sunscreen Lotion Extreme	40	A, B, OC, S
Panama Jack Sunscreen Lotion	45	B, C, OC, S
Sea & Ski Sport	50	B, C, S, ZO
Solbar PF Cream	50	B, C, OC, S
TI Screen Moisturizing Sunscreen Lotion	30	A, B, C, OC, S
Water Babies Sunblock Lotion	45	B, C, S
High-SPF Gels		
Bullfrog Quik Gel	36	B, C, OC, S
Coppertone Sport Sunblock Gel	30	A, B, OC, S
Pre-Sun Ultra Gel	30	A, B, C, S
Ti-Screen Sports Gel	20	B, C, S
High-SPF Sprays		
Banana Boat Sport Quik Block Sunblock Spray	48	A, B, C, OC, S
Bullfrog Fast Blast Spray Sunblock	36	B, C, OC, S
Coppertone Sport Sunblock Spray	30	B, C, S
Neutrogena Healthy Defense Oil-Free Spray	30	A, C, S
Ti-Screen Sunscreen Spray	23	B, C, MA, OC
High-SPF Sticks		
Banana Boat Sport Sunscreen Stick	30	B, C, OC, S
Bullfrog Quik Stick	36	B, C, OC, S
Hawaiian Tropic Ozone Oil-free Sunblock Stick	50+	A, B, OC, S
Neutrogena Healthy Defense Sunblock Stick	30	A, C, S
Ocean Potion Sport Stick	60	A, B, S
Shade Sunblock Stick	30	B, C, S
Specialty Sunscreens		
AloeGator Super Waterproof Gel	40+	B, C, OC, S
Bullfrog Surfer Formula Gel	36	B, C, OC
Dermatone Skin Protector Pomade	23	B, C, PB
SolRx ProSport	44	B, C, OC, S
Lip Screens		
Banana Boat Aloe Vera Lip Balm	30	B, C, PB, S
Coppertone Sport Lip Guard	15	B, C
Dermatone Medicated Lip Balm	23	B, PB
Hawaiian Tropic Aloe Vera Sunblock Lip Balm	45+	C, OC, S
Neutrogena Lip Moisturizer	15	B, C
Ti-Screen Lip Protectant	15	B, C
"Physical" Sunscreens		
Neutrogena Sensitive Skin Sunblock Lotion	30	TI
Pre-Sun Sensitive Skin Sunblock	28	TI
Vanicream Sunscreen	15	TI, ZO
Moisturizers Containing Higher SPF Sunscreen		
Antihelios SX Daily Moisturizing Cream	15	A, E, OC
Aveeno Positively Radiant Daily Moisturizer	15	A, C, S
Eucerin Extra Protective Moisture Lotion	30	C, OC, PBSA, TI, ZO
Lubriderm Daily Moisture Lotion	15	B, C, S
Neutrogena Healthy Defense Daily Moisturizer	30	C, OC, PBSA, ZO
Oil of Olay Complete Defense Daily UV Moisturizer	30	C, OC, S, ZO
Purpose Dual Treatment Moisture Lotion	15	C, MA, TI

A, avobenzone; B, benzophenones; C, cinnamates; E, ecamsule; MA, methyl anthranilate (meradimate); OC, octocrylene; PB, PABA or PABA ester; PBSA, phenylbenzimidazole sulfonic acid (ensulizole); S, salicylates; TI, titanium dioxide; ZO, zinc oxide.

study by Santoro and coworkers,[249] benzophenone-3 and octyl methoxycinnamate maintained stability for 60 days in both glass and plastic containers at room temperature (25°C [77°F]) and 35°C (95°F) with only minor changes in concentrations at 10°C (50°F) and 45°C (113°F). The generally accepted wisdom is to keep sunscreens out of glove compartments and similar locations where they are exposed to extremes in temperature for long periods. Shelf life is presumed to be at least a year or more for most commercially available sunscreens, though, again, data are lacking.

Sunscreen Prevention of Chronic Photodamage. Sunscreens not only prevent sunburns but also mitigate UVR-induced histologic damage, UVR-induced immunosuppression, photoaging, and photocarcinogenesis. Sunscreens reduce UVR-induced DNA damage,[283] but not entirely.[44] Reductions are seen in pyrimidine photoproducts in humans,[44] as well as in UVR-induced p53 mutations and skin cancer in mice.[5] Sunscreen nearly eliminates the overexpression of p53 in mice after acute[226] and chronic[90] UVR exposure. In humans, an SPF 15 sunscreen reduces p53+ cells by 33% after chronic UVR exposure.[24]

The published effects of sunscreen on immunosuppression are contradictory, largely resulting from the complex relationships of UVR dosing and sensitivity in different experimental models. SPF is not a reliable measure of a sunscreen's ability to block UVR-induced immunosuppression; two sunscreens with identical SPF values may vary considerably in their immunoprotectant effects.[232] SPF and IPF (immune protection factor) are not synonymous or equivalent.[305] Overall, sunscreens mitigate but do not abrogate UVR-induced immunosuppression. In mice, sunscreens prevent UVR-induced depletion of Langerhans' cells, with higher-SPF sunscreens conferring greater protection.[19] However, earlier studies noted little or no immunoprotectant effect from sunscreens.[19] A broad-spectrum SPF 15 sunscreen prevents UVR-induced suppression of contact hypersensitivity to dinitrochlorobenzene (DNCB), a potent topical allergen, in humans.[257] In C3H mice transplanted with NMSC, sunscreen blocks UVR-induced immunosuppression, allowing tumor rejection, again showing increased protection with higher SPF values.[238] However, in susceptible mice injected with melanoma, sunscreens fail to adequately suppress UVB enhancement of melanoma growth.[295] This finding has been overinterpreted and misinterpreted by some to support a peculiar anti-sunscreen stance. The most appropriate interpretation, however, is that sunscreens by themselves are not sufficient to prevent all of the immunosuppressive sequelae of UVR exposure, and that sunscreens should be used as part of a larger sun-protective strategy, including avoidance and clothing protection.

Sunscreens reduce sunburn cell formation and solar elastosis[34] in humans. Histologic changes of photoaging in mice are prevented by pretreatment with SPF 15 sunscreen.[151] In mice exposed to UVB for 10 to 20 weeks, subsequent sunscreen use prevents further photodamage and promotes repair of previous damage, even with continued exposure.[152] Higher-SPF sunscreens provide increasing protection against UVB-induced wrinkling in mice.[29] Similarly, histologic and clinical signs of UVA-induced photoaging are prevented by broad-spectrum sunscreens.[121]

Sunscreen use reduces the formation of precancerous actinic keratoses[209,276] and promotes resolution of preexisting lesions.[276] In a 2-year trial of sunscreens in humans with actinic keratoses, those who benefited most had the greatest number of keratoses at enrollment,[209] underscoring the value of continuing sunscreen use in adults. The daily use of SPF 15 sunscreen in adults reduces the incidence of SCCa but not BCCa.[112] Stern and colleagues[269] estimated that use of an SPF 15 sunscreen from birth until age 18 would reduce the lifetime risk of NMSC by 78%.

Liquid-base makeup, even without sunscreen, provides an SPF of approximately 4 because of pigments in the foundation.[171] For women who use lipstick, the incidence of SCCa is lower than in nonusers or men.[224]

Sunscreens, Melanoma, and Vitamin D. Controversy regarding the effects of sunscreen use on the incidence of melanoma has intensified. With melanoma incidence and sunscreen sales both rising over several decades, some have suggested that sunscreens increase the incidence of melanoma. Epidemiologic studies have generally failed to demonstrate decreased melanoma incidence with sunscreen use. Paradoxically, several studies have shown an increased risk of melanoma with sunscreen use.[12,62] In 1996, Donawho and Wolf[62] reviewed retrospective studies evaluating melanoma risk in sunscreen users. Only one showed a decreased risk,[132] whereas seven showed an increased risk. Subsequent studies continue to show contradictory results; a case-control study in southern Spain, published in the same year as the metastudy by Donawho and Wolf, showed a decreased melanoma risk with sunscreen use in a Mediterranean population.[241] In 1993, Garland and coworkers[94] proposed that sunscreens, by primarily blocking UVB and sunburn, permit longer exposures to UVA in sunlight, and that this increased UVA exposure results in an increased incidence of melanoma. Confounding all of these studies, however, are the following points:

- Persons at highest risk for melanoma (those with fair complexion who burn easily) are precisely the same persons who use sunscreens.
- Earlier sunscreen products had lower SPFs and narrower spectrums. Modern sunscreens are typically more substantive, with higher SPFs and substantially broader, better UVA protection.
- Because sun exposure in childhood appears most provocative for melanoma, surveying adults about their sunscreen habits may be irrelevant and misleading.

The effect of sunscreens and clothing protection on the numbers of nevi in children and adolescents, a prominent risk factor for melanoma,[93] is controversial as well. In Australian children, aged 0 to 35 months, sunscreen use is associated with modestly decreased nevus counts.[122] However, in a study of adolescent twins in the United Kingdom,[285] sunscreen use did *not* decrease nevus counts. In fact, there was a modest increase in benign nevi in adolescents who regularly used high-SPF sunscreens, possibly because of increased exposure times by sunscreen users. In this U.K. study, clothing protection had no effect on nevus counts. However, in a recent study of 1st and 4th graders in British Columbia, those who regularly used sunscreens developed fewer nevi, especially on intermittently sun-exposed sites.[169]

It remains unclear why intermittent sun exposure, especially in childhood, is associated with an increased risk of melanoma, whereas chronic occupational exposure is partially protective.[91] An increasingly favored explanation is that vitamin D, formed

by the action of sunlight on the skin, may be protective. Data are amassing to support this hypothesis. Mortality rates of several common cancers in the United States—breast, colon, and prostate—increase with increasing latitude.[68] In 1990, Garland and colleagues[95] suggested that decreased cutaneous synthesis of vitamin D at higher, less sunny latitudes, might explain increased mortality from breast cancer in northern states. Recently, Smedby and coworkers[262] reported that sunlight reduces the risk of non-Hodgkin's lymphoma. Among men in the Navy, Garland and colleagues[96] determined that regular sun exposure reduces melanoma risk. In a recent, population-based study of 528 patients with melanoma, Berwick and coworkers[25] found that increased survival is associated with sun exposure. These data suggest that melanoma may be among the tumors for which vitamin D has a salutary effect. Supporting this hypothesis are laboratory data that show the presence of vitamin D receptors on melanoma cells, with growth inhibition in response to this vitamin.[51] In a case-control study of 502 melanoma patients and 565 controls, Millen and colleagues[196] found that diets rich in vitamin D and carotenoids were associated with a reduced risk of melanoma.

These findings beg the question, Do sunscreens, by absorbing UVB, reduce vitamin D levels sufficiently to pose an increased risk of melanoma and perhaps other cancers as well? Holick[129] states that there is an "unrecognized epidemic" of vitamin D deficiency in the United States. Nonetheless, concerns regarding sunscreen-induced vitamin D depletion may not be warranted. Although regular sunscreen use can decrease cutaneous synthesis of vitamin D_3[189] and circulating levels of 25-hydroxyvitamin D,[190] even the most conscientious sunscreen users appear to maintain normal levels of vitamin D. One hundred thirteen subjects using an SPF 17 sunscreen throughout an Australian summer maintained adequate vitamin D levels,[183] and 24 subjects using SPF 15 sunscreen for 2 years maintained normal parathyroid hormone levels and normal bone metabolic markers.[77] Moreover, eight patients with XP maintained normal vitamin D levels for the 6 years observed, despite rigorous photoprotection with sunscreens, clothing, and sun avoidance.[264]

Part of the controversy surrounding vitamin D stems from unclear or varying recommendations regarding the minimum daily allowance and the difficulty in getting adequate amounts from diet and supplements. Current recommendations suggest a daily minimum of 200 IU to age 50 years, 400 IU for ages 50 to 70, and 600 IU for ages greater than 70. Food sources for vitamin D—milk, orange juice, fish—are often insufficient, and supplements most often contain vitamin D_2, derived from plants and fungi, which is less potent than the human, D_3 form of the vitamin. It is clear that attention needs to be focused on fortifying more foodstuffs and providing better vitamin D supplementation, especially for persons living in northern climates in the winter, for older adults who are at higher risk for osteopenia and fractures,[108] and for infants, especially breast-fed infants, who get inadequate vitamin D in human milk and may consequently be at risk for rickets.[97,158] Given the remarkably brief sun exposures needed to synthesize vitamin D,[128] Holick suggests an additional approach—"sensible sun exposure," 5 to 10 minutes two or three times weekly, in conjunction with dietary intake of vitamin D and vitamin supplements.[129] This recommendation has raised considerable controversy and concern in the dermatologic community, where the rise in melanoma incidence and sunscreen use has been considered a reflection of increased recreational sun exposure.[210] Hopefully, research in the next decade will put this controversy to rest.

Sunscreens and Herpes. UVB is a potent stimulus for reactivation of herpes labialis in outdoor enthusiasts.[138] Sunscreen, although ineffective at preventing recurrences in skiers on the slopes,[198] is quite effective in preventing recurrences in the clinical laboratory,[244] perhaps reflecting a difference in application techniques.

Sunscreen Side Effects. Sunscreen side effects are generally mild and limited. In a prospective study of 603 subjects who applied SPF 15 sunscreen or vehicle control,[80] 19% in both groups had adverse reactions. Most reactions were irritant in nature, and fewer than 10% were allergic. More than half who developed irritation were atopic. These data support the notion that most contact irritant dermatitis to sunscreens is attributable to excipients. Similarly, allergic reactions to sunscreens are more often due to preservatives and fragrances than to active sunscreening agents.[65] As a consequence, and contrary to popular belief, higher-SPF sunscreens are not more irritating than lower-SPF sunscreens.[258]

Nonetheless, most sunscreening agents can cause allergic or photoallergic contact dermatitis.[143] For example, PABA is now rarely used because it sensitizes approximately 4% of exposed subjects. Once sensitized, cross sensitization may occur to thiazides, sulfonamides, benzocaine, and hair dyes containing paraphenylenediamine.[235]

Paradoxically, sunscreens have become the leading cause of photoallergic contact dermatitis.[277] Oxybenzone is the most commonly implicated. In 283 cases of photodermatitis, 35 people were photoallergic to oxybenzone and 17 to PABA.[274] In a separate study of 108 patients with photodermatitis, four were photoallergic to oxybenzone and four to other sunscreening agents.[277]

Stinging or burning without accompanying erythema, scaling, or dermatitis is common, especially in periocular areas and particularly in patients with rosacea, although addition of skin "protectants" (dimethicone or cyclomethicone) to the sunscreen vehicle can mitigate this.[211] Certain vehicles, such as alcoholic gels, may be more stinging. Even when periocular application is avoided, perspiration, water immersion, and rubbing may cause sunscreen to migrate, producing symptoms.

Comedogenicity is primarily related to ingredients in the vehicle base. Certain common excipients (almond oil, cocoa butter, isopropyl myristate, isopropyl palmitate, and olive oil) are possible comedogens.

TiO_2 and ZnO can generate reactive molecular species, called free radicals, with sun exposure.[63] TiO_2 is more photoactive than ZnO.[200] Electromagnetic radiation with λ less than 412.5 nm has sufficient energy to excite TiO_2 electrons from the valence band to the conduction band.[155] Photoactivated TiO_2 can damage DNA in vitro.[126] In vivo, however, it is unlikely that TiO_2 particles penetrate the stratum corneum to reach underlying epidermal cells containing DNA. Transmission electron microscopy fails to demonstrate TiO_2 penetration of the stratum corneum.[66] Data also demonstrate that ZnO is not absorbed, and Zn levels are unchanged after application.[98] Coating TiO_2 or ZnO with silicone halts photoproduction of reactive

species.[63] Still, some concerns have been raised regarding the use of these agents on broken skin.

Some sunscreening agents have weak estrogenic and/or antiandrogenic activity.[255] In particular, benzophenone-3, octylmethoxycinnamate, and 3-(4-methylbenzylidene) camphor may have estrogenic effects on human breast cancer cells in culture.[254] To assess their endocrinologic safety, Janjua and coworkers[138] studied whole-body application of these three sunscreening agents on humans daily for 1 week, using a liberal concentration of 2 mg/cm^2. Plasma and urine levels of all three agents were detectable in men and postmenopausal women; luteinizing hormone and follicle-stimulating hormone were unchanged; there were only minor changes in testosterone and estradiol (in men only). Janjua and colleagues conclude that these endocrinologic alterations are clinically insignificant, and that these sunscreen products are safe for use, at least for adults.

Clothing Protection

Clothing is a crucial part of sun protection, providing substantive coverage to broad surface areas. However, clothing varies considerably in its ability to block UVR. Robson and Diffey[240] determined an SPF for 60 different clothing fabrics, ranging from 2 (polyester blouse) to 1000 (cotton twill jean). The most important factors in determining SPF are tightness of the weave,[102,175,240] and then the actual fabric. An extreme example of this is Lycra, which blocks nearly 100% of UVR when lax and only 2% when maximally stretched.[175] Other determinants include wetness and color. Dry, dark fabrics have a higher SPF than otherwise identical, wet, white fabrics.[102] A typical dry, white cotton T-shirt has an SPF of 5 to 9.

Accumulating data show that clothing prevents chronic photodamage. In European children, clothing coverage is associated with fewer nevi.[9] Blue denim reduces UVR-induced p53+ cells twice as effectively as an SPF 15 sunscreen.[24] Denim greatly reduces the formation of skin cancers in patients with XP.[22]

Standardized testing of clothing fabrics has produced a new measure, the ultraviolet protection factor (UPF), analogous to the SPF for sunscreens. A collimated UVR source is passed through flat, tensionless fabric, with resultant transmission measured by a spectroradiometer or broad-band sensor.[102,240,309] The resultant UPF, however, is generally much lower than actual use values, because natural sunlight is scattered and reflected to a far greater extent than is a collimated beam.[231]

In the United States, sun-protective clothing is regulated as a medical device (Fig. 14-4). Several manufacturing strategies are used to achieve high SPFs. One approved product, Solumbra, is made of tightly woven nylon with an SPF of 30+. In hairless mice, this fabric is significantly better than cotton in reducing the formation of UVR-induced SCCa.[195] By contrast, Solarknit uses chemically treated cotton and cotton–synthetic blends to achieve an SPF of 30+. Rayosan, a UVR-absorbing agent, bonds to various fabrics, increasing their SPF by up to 300%, and lasts through several washings.[175] A unique chemical UVR protectant, Tinosorb FD, may be incorporated into detergents, increasing the SPF of clothing with each wash.

Ladies' hosiery provides a surprisingly low SPF: black hose has an SPF of 1.5 to 3.0 and beige hose has an SPF less than 2.[259] Perhaps this explains in part why skin cancers are more common on the lower legs of women than of men.

Hats are also protective, with protection varying as a function of brim diameter and wearing style. Small-brimmed (<2.5 cm [about 1 inch]) hats adequately protect the forehead and upper nose; medium-brimmed (2.5 to 7.5 cm [about 1 to 3 inches]) and wide-brimmed (>7.5 cm) hats protect increasing proportions of the nose, cheeks, chin, and neck,[60] with large-brimmed hats providing an equivalent SPF of 7 for the nose, 3 for the cheeks, and 2 for the chin. Baseball-style caps are especially useful as a part of an overall sun-protective strategy in children. When hats are used to protect the forehead and periocular areas, sunscreen application can be limited to areas below the cheekbones, mitigating the risk of stinging near the eyes.

A B

Figure 14-4. Photoprotective clothing. **A,** Solumbra hiking apparel. **B,** Coolibar swimwear. (**A** courtesy Sun Precautions, Inc.; **B** courtesy Coolibar, Inc.)

Glasses, contact lenses, and sunglasses protect the corneas from most UVB and variable amounts of UVA.[246] Surprisingly, price is unrelated to protection.[3] A complete discussion is contained in Chapter 25.

Sun Avoidance

An indoor lifestyle is protective but undesirable for most persons. More practical is avoidance of excessive midday sun, from 10 am to 3 pm, significantly reducing UVB exposure.[175] Shade provides variable and often less protection than generally assumed. In one study, shade beneath leafy trees provided an SPF of less than 4.[219] Shade cloths allow significantly more UVB exposure than clothing of the same fabrics, largely because of atmospheric scattering and surface reflection.[296]

Automobile windshields typically block UVB and some UVA, whereas side windows block only UVB.[278a] This may explain why photodamage is more prominent on the left side of the face in Americans (and on the right side in Australians) who drive a lot.[64] Transparent plastic films, meeting legal requirements in all 50 states, can be applied to block more than 99% of UVR (Llumar UV Shield, CPFilms, Martinsville, VA).

Sunless Tanning

Bronzers. Most artificial tanners and bronzers contain dihydroxyacetone (DHA, or $C_3H_6O_3$). DHA reacts with amino groups of keratin proteins via the Maillard reaction to form brown-pigmented products known as melanoidins.[170] With DHA, bronzing can occur in as little as 1 hour. Often, however, it requires multiple applications to achieve the desired depth of color. Maintaining this bronzed look requires reapplication every few days, as the stained stratum corneum is shed from the skin surface. The depth of color is related to the thickness of the stratum corneum and the amount and frequency of application.[171] Cosmetic complaints include difficulty in obtaining an "even" tan and yellowing of the palms.

DHA plus lawsone (a henna dye) was previously used as a sunscreening agent. However, DHA alone is an inadequate sunscreen. It absorbs no UVB; rather, DHA absorbs higher-wavelength UVA I and lower-wavelength visible light,[85] making it useful in select photosensitivity disorders, such as porphyrias and polymorphous light eruption. Some commercial bronzing products now contain sunscreen in addition to DHA. Although the artificial tan in these combination products lasts for days, photoprotection lasts for only hours. Consequently, the FDA *Final Monograph* requires that bronzers without sunscreens display a warning that they do not protect against sunburn.

Certain other products to promote indoor tanning have been used, but few are safe and effective. Tan accelerators, containing melanin precursors such as tyrosine, have no discernible benefit.[139] Sunscreen preparations containing psoralens, most commonly 5-methoxypsoralen (oil of bergamot), are available in Europe. Although these stimulate melanin synthesis, they are tumorigenic in mice.[48] Psoriasis patients treated with psoralen plus UVA (PUVA) have a higher incidence of SCCa[267] and, with prolonged use, melanoma.[268] Users of psoralen-containing sunscreen have an increased risk of subsequent melanoma.[12] Oral carotenoids, especially canthaxanthin, have been promoted as tanning pills but are potentially toxic and, consequently, not approved in the United States.

Tanning Salons. Although precise data are lacking, approximately 25 million North Americans, many of them adolescents, use tanning salons each year.[273] In a survey of high school students in St. Paul, Minnesota, 34% admitted to using tanning salons at least four times in the previous year.[214] Until 1980, most sunlamps emitted mostly UVB.[12] Today's tanning salons utilize high-output UVA tanning beds and market their services as a way to tan without burning, with a subtle but incorrect message that such tans are safe. However, chronic suberythemal UVA contributes to photoaging, immunosuppression, and carcinogenesis. In addition, there is significant variation in the radiation output of tanning beds. In a study of 50 tanning beds in England,[299] UVA outputs varied by a factor of 3 and UVB output varied by a factor of 60. Typically, tanning bed outputs are contaminated with 2% to 10% UVB,[286] more than the average UVB content of natural sunlight. In 38 Scottish tanning units, 10 minutes of exposure yielded the same carcinogenic risk as 30 minutes of peak summer sun at the same latitude.[207]

Tanning bed exposure for 15 to 20 minutes induces DNA damage in fibroblasts.[297] Several case-control studies document a positive association between tanning bed use and the later development of melanoma.[10,287,294] In a Swedish study of 400 patients and 640 controls, persons who used sunbeds or sunlamps before age 30 had a 7.7-fold increased risk of melanoma.[294] In 420 patients from Germany, France, and Belgium, melanoma risks increased 8.97-fold when sunlamps or sunbeds were used for tanning.[11]

Ironically, UVA tans are less effective than natural tans in conferring protection against subsequent sunburn.[142] In addition, maintenance of a yearlong tan provides no protection against subsequent melanoma development.[132] Too often, tanning salons ignore photosensitizing risks in their patrons.[42] Furthermore, the damage from tanning salons is insidious. Public awareness of the danger needs to be heightened, and health and government regulators are now taking the lead.[298] Recently, the World Health Organization stated that no one younger than 18 should use tanning beds. Increasingly, states and individual counties within the United States are restricting access to tanning parlors by minors. As of May 2005, 21 states and four counties had restrictions limiting access to tanning salons by minors.[82]

Melanin and Thymine Dimers. Cyclobutane T-T dimers, produced by the action of UVR on skin, stimulate melanin synthesis and subsequent pigmentation.[105] Eller and coworkers[70] exposed melanocyte cultures to synthetic T-T dimers and noted twofold to threefold increases in tyrosinase messenger ribonucleic acid (mRNA) and sevenfold increases in melanin production. Topical application to guinea pigs increases pigmentation. This raises the question of whether induction of protective pigmentation in humans might be possible with T-T dimers. Is it possible to induce a safe tan?

Unique Photoprotectants

Systemic Sunscreens. Safe and effective oral sunscreens remain elusive. Clearly, these would have an advantage in convenience, coverage, and substantivity. Several oral agents have been evaluated, but few are significantly effective or have favorable risk-to-benefit ratios in normal individuals. However, three botanicals appear promising: green tea, turmeric, and extracts of the fern *Polypodium leucotomos*.[233] Currently, a few oral agents are used in patients with photosensitivity disorders: beta-carotene is helpful in erythropoietic protoporphyria, chloroquine in lupus erythematosus, and psoralens in polymorphous light eruption.

Antioxidants. UVR exposure results in reactive oxygen species that contribute to DNA damage[222] and the peroxidative destruction of membrane lipids.[134] These effects contribute to DNA structural abnormalities, inflammation, and immune alterations. Protecting the skin from reactive species are various natural antioxidants (vitamins A, C, and E; reduced glutathione; urocanic acid; melanin) and enzymatic systems (catalase, superoxide dismutase).[134] However, chronic exposure to UVR depletes the skin of these antioxidants.[178]

Application of antioxidants before UVR exposure delays skin damage in hairless mice.[28] Select forms of vitamin E, especially tocopherol sorbate, diminish photoinduced free radicals and skin wrinkling in mice.[140] Topical application of vitamins C and E provide protection against the histopathologic changes of photodamage in swine skin.[56] In this animal model, sunscreen plus vitamins C and E provide more protection than sunscreen alone.[56] Although vitamin C does not act as a sunscreen—it does not absorb UVR—it protects against erythema and sunburn cell

formation by quenching free radicals that are at least partially causative.[55] Interestingly, the beneficial effect of topical vitamin C in swine skin persists for 72 hours after application, suggesting a potential strategy for longer-term photoprotection.

Resveratrol, a potent antioxidant found in grapes, nuts, and red wine, mitigates UVB associated damage in mouse skin. It reduces UVB-induced edema, inhibits lipid peroxidation, and decreases the production of enzymes associated with tumor promotion.[2] Silmarin, a flavanoid derived from milk thistle, also has antioxidant, anti-inflammatory, and salutary immunomodulatory effects when applied topically.[144] Similarly, topical applications of aloe and tamarind xyloglucan have been shown to protect against UVR-induced immunosuppression by acting as antioxidants rather than sunscreens.[272]

Oral antioxidants may also be of value in preventing photodamage. In humans, a combination of 2 g of vitamin C combined with 1000 IU of vitamin E, given orally for 8 days, increased MED, albeit modestly.[67] A more recent, larger study of human volunteers, using a combination of oral vitamins C and E for 3 months, documents a significant decrease in the clinical signs of sunburn and thymine dimer formation following UVB irradiation.[223] In a mouse model, the dietary addition of lutein, a carotenoid antioxidant, decreases edema and suppression of contact hypersensitivity following UVB exposure.[168] Green tea given orally to mice reduces cutaneous oxidative damage and abnormal MMP formation in response to UVB.[284]

There are some concerns, however, about the routine use of antioxidants. For example, topical vitamin E may provoke contact sensitization and may occasionally serve as a tumor promoter.[199] In addition, both topical and oral antioxidants might interfere with the normal functioning of p53 protein to produce apoptosis of photodamaged keratinocytes via oxidative enzymes.

Repair Enzyme T4N5. Recently, there has been considerable interest in postexposure therapy for sunburn with topical application of a DNA repair enzyme, T4 endonuclease V (T4N5). Applied in liposomes, it localizes in the epidermis and appendages,[302] enhancing repair of UVR-induced DNA damage in mice and humans.[303] T4N5 prevents UVR-induced immunosuppression[161] and reduces UVR-induced skin cancer in mice.[301] In a study of 30 patients with XP, T4N5 in a liposomal base significantly reduced the incidence of new actinic keratoses and basal cell carcinomas after 1 year.[300] Given these qualities, T4N5 may serve in the future as a "morning-after" cream.

PHOTOSENSITIVITY DISORDERS

Endogenous Photosensitivity Disorders

Sun protection is especially important for persons with endogenous photosensitizing disorders, persons who take photosensitizing medications (Box 14-3), and persons exposed to topical photosensitizers (Box 14-4). The most common endogenous photodermatosis is polymorphous light eruption (PLE), affecting 10% to 14% of whites, predominantly females under age 30. Clinically this presents with pruritus, erythema, macules, papules, or vesicles on sun-exposed skin, arising 1 to 2 days after exposure and resolving spontaneously over the next 7 to 10 days. It is most common with initial sun exposures in spring or early summer; "hardening" of the skin may occur with subsequent exposures. The action spectrum is unclear, although it is most commonly 290 to 365 nm.[84] Sunscreens are only sometimes helpful, although a broad-spectrum sunscreen containing padimate O and avobenzone was quite effective in one study.[86] Sun avoidance and protective clothing are valuable. PUVA, oral antimalarials, and beta-carotene have also been used with varying success.

Box 14-3. Common Oral Photosensitizing Medications

Antihistamines
Fluoroquinolones
Nonsteroidal anti-inflammatory drugs
Oral contraceptives
Phenothiazines
Sulfonamides
Sulfonylureas
Tetracyclines
Thiazides
Tricyclic antidepressants

Lupus erythematosus may be exacerbated by sunlight. Although UVB is typically causative, UVA can contribute in some patients. Consequently, broad-spectrum sunscreens, combined with sun avoidance and clothing protection, are most appropriate. A broad-spectrum sunscreen containing padimate O and avobenzone diminished the clinical severity in a 4-week study.[45] Systemic antimalarials may also be used in more serious cases.

Porphyrias are caused by inherited abnormalities in heme synthesis. Clinical manifestations vary with the genetic subtype, but photosensitivity is common to this group of disorders. Typically, the action spectrum is in the Soret band, 400 to 410 nm. Consequently, sunscreens other than "physical" blockers are generally inadequate, although topical DHA and oral beta carotene may be useful adjuncts to sun avoidance and protective clothing.

Chronic actinic dermatitis presents as persistent macules and plaques, often infiltrated and lichenified, in chronically sun-exposed sites on older men. These patients may demonstrate dermatitic flares on involved areas with even modest photoexposure to UVR and shorter-wavelength visible light. Histologically, the disorder can demonstrate progression from dermatitis to T-cell lymphoma. Broad-spectrum sunscreens, sun avoidance, and clothing protection, including brimmed hats, are essential. For advanced cases, therapy with azathioprine, cyclosporine, PUVA, and retinoids has been used.

Persons with persistent light reaction (PLR) display a peculiar and persistent overreaction to sun from a prior topical photoallergy. Musk ambrette, found in many perfumes, has been reported to provoke PLR.[235] UVB is most often causative, although UVA may be involved as well. Broad-spectrum sunscreens, sun avoidance, and clothing protection provide appropriate prophylaxis.

For disorders in which melanocytes are defective or absent, such as albinism and vitiligo, and disorders in which DNA repair mechanisms are deficient, such as xeroderma pigmentosum, the protective triad of sunscreens, sun avoidance, and clothing protection is required.

Box 14-4. Topical Phototoxins and Photoallergens

TOPICAL PHOTOTOXIC COMPOUNDS

Dyes
- Eosin
- Methylene blue

Medications
- Phenothiazines
- Sulfonamides

Psoralens
- Methoxypsoralen
- Trimethylpsoralen

Tars
- Creosote
- Pitch

TOPICAL PHOTOTOXIC PLANTS
- Angelica
- Carrot
- Celery
- Cow parsley
- Dill
- Fennel
- Fig
- Gas plant
- Giant hogweed
- Lemon
- Lime
- Meadow grass
- Parsnip
- Stinking mayweed
- Yarrow

TOPICAL PHOTOALLERGENIC COMPOUNDS

Antiseptics
- Chlorhexidine
- Hexachlorophene

Fragrances
- Methylcoumarin
- Musk ambrette

Phenothiazines

Salicylanilides

Sulfonamides

Sunscreens
- Benzophenones
- Cinnamates
- Dibenzoylmethanes
- Para-aminobenzoic acid (PABA)
- PABA esters

Phototoxicity

Most oral photodrug eruptions are phototoxic, clinically presenting as exaggerated sunburn on exposed skin. Typically, there are sharp cutoffs at the V of the neck and short-sleeve line. Less commonly, there may be photo-onycholysis, in which the distal fingernails separate from their underlying beds. Photodrug eruptions are usually triggered by UVA. The most common offending drugs are listed in Box 14-3.

Topical photosensitizers may cause phototoxicity, photoallergy, or both. Phototoxicity results in an exaggerated sunburn, typically followed by postinflammatory pigmentation. Phototoxicity is nonimmunologic. The action spectrum for most topical phototoxic sensitizers is in UVR.

A surprisingly large number of plants contain photosensitizers that can produce phototoxic reactions in exposed human skin (see Box 14-4), resulting in a condition known as phytophotodermatitis. The most common chemical precipitants are furocoumarins, especially psoralens found in limes, lemons, and certain other plants. Phytophotodermatitis is common in farmworkers, bartenders, cannery packers, and vacationers to sunny climates. Oil of bergamot, containing 5-methoxypsoralen, is a frequent cause of phototoxicity in perfumes (berloque dermatitis). Streaks of erythema and subsequent pigmentation on exposed skin of the face, neck, and wrists typify this eruption. Meadow-grass dermatitis in hikers is another example of phytophotodermatitis. Various common weeds, including meadow parsnip, contain furocoumarins, causing whiplike erythematous streaks and pigment on skin after exposure to sunlight.

Psoralens have been incorporated into sunscreens in Europe to promote tanning, although their use is associated with increasing risks of burns and carcinogenicity.[48] Psoralens, in combination with UVA (PUVA), are used therapeutically for psoriasis and selected other dermatoses.

Photoallergy

Although phototoxic reactions may occur in anyone, photoallergic reactions occur only in previously sensitized individuals. Photoallergic reactions are uncommon. They are typically provoked by the interaction of UVA with a topical proallergen, resulting in contact dermatitis within 48 hours. Unlike phototoxic reactions, which are limited to exposed skin, photoallergic reactions can spread to sun-protected adjacent sites. Paradoxically, sunscreens are the current leading cause of photoallergy.[59,133] In the 1970s, a fragrance, 6-methylcoumarin, added to a suntanning product was responsible for a number of cases of photoallergy.[235] In the 1960s, there was a virtual epidemic of photoallergic reactions resulting from antibacterial salicylanilides in soaps.[235] Prevention requires excellent UVA protection, including broad-spectrum sunscreens.

CHANGING ATTITUDES TOWARD PHOTOPROTECTION

In the 20th century, a tan became desirable, equated with health, wealth, and stylishness. Characterizing the times was Coco Chanel's pronouncement in 1929 that "a golden tan is the index of chic." Sadly, this attitude led to dangerous behaviors, resulting in an epidemic of skin cancer.

Several studies document current attitudes toward photoprotection. In a study of Connecticut beachgoers, 70% were there to get or maintain a tan.[312] College students questioned in North Carolina rarely, if ever, used sunscreens.[281] In a U.S. study of 2459 whites, 59% admitted to sunbathing and 25% to sunbathing more than 10 times in the preceding year.[154] Among

spring skiers, only two thirds used sunscreens, and, of those, one third were sunburned at the time of the survey.[193] A survey in Leicester, England, found that half of all children sunburn at least once a year.[32] A 2003 Fact Sheet from the Centers for Disease Control and Prevention reports that 43% of white children under 12 years of age in the United States had one or more sunburns within the past year.[49] Even patients with sun-related problems often do not adopt adequate sun protection habits. One study documented that patients with dysplastic nevus syndrome, who are at higher risk for melanoma, do not avoid sunburning.[35] Among Israeli patients 1 year after treatment for BCCa, only 49% wear hats or long sleeves in summer, and 62% use less than two bottles of sunscreen per year.[124]

Sunscreen use correlates with knowledge of the harmful effects of sun exposure and understanding of SPF values.[26] In a survey of 1001 adults in the United States, only 42% were aware of the term *melanoma,* and only 34% knew it was some type of skin cancer.[197] Many mistakenly use sunscreens in the belief that sunscreens will promote tanning. Clearly, more education is needed. In 146 college students in Southern California, a 12-minute videotape on photoaging in conjunction with ultraviolet facial photography resulted in improved sun protective behaviors 1 month after the education.[179] In Australia, where a significant educational effort is underway, three fourths of adults visiting a family physician report using sunscreens.[187] Interestingly, Australian state and privately sponsored programs stress sun avoidance and clothing protection over sunscreens, and increasingly Australian fashion magazines feature more hats and more models who are less tan.[50] Prevention and early diagnosis programs have yielded results: sun-seeking behaviors and sunburns have decreased. More importantly, the incidence of melanoma has decreased and melanoma survival has increased in younger Australian cohorts.[191]

Photoprotection education needs to begin in childhood. Considerable sun exposure occurs by age 18, and childhood exposure may be more significant than lifetime exposure in determining the subsequent risks of BCCa and melanoma. Children who use, or are compelled to use, sunscreens at an early age are more likely to use sunscreens as adolescents.[17] This requires educating parents. Sadly, parents too often ignore, or are ignorant of, photoprotective measures. On beaches in Galveston, Texas, in midday July, only 42 of 82 parents interviewed applied sunscreen to their children.[177] Only a history of prior painful sunburns was correlated with sunscreen use, a finding documented in other studies as well.[311] Physicians should try to alter parental behavior through education, beginning with the first well-baby visit. In the Galveston study, photoprotection was discussed in only 18% of well-baby checkups. Sun avoidance and clothing protection should be advocated for infants under age 6 months, and sunscreen should be added to the photoprotective regimen for older toddlers and children. Consistent use of sun protection in childhood would not only decrease sunburns but would provide lifelong reductions in photoaging and NMSC.[269]

Advertisers and the fashion industry continue to glorify tans with bronzed models. Even sunscreen advertisements seem to have a dual and medically incompatible message: tan beautifully while protecting your skin. No wonder the public is confused by oxymoronic enticements to a "healthy tan."[230] Recently, in the United States, there is a glimmer of progress toward photoprotection education in the public media. In a review of models in six popular American fashion magazines between 1983 and 1993, there is a trend toward lighter tans, more sunscreens, and more articles about sun awareness, although men's magazines did not show this trend.[100]

CONCLUSION

Sun exposure is associated with deleterious sequelae, both acute and chronic. Sun protection significantly mitigates these sequelae. Appropriate sun-protective strategies include a combination of sun avoidance, clothing protection, and sunscreens. Sunscreens have evolved to become increasingly elegant and effective. Despite occasional concerns, sunscreens have enjoyed an excellent safety record for more than 5 decades.[103,172] Still, some issues remain controversial and unanswered. For example, further research is needed to answer concerns about vitamin D deficiency with strict sun protection. It is conceivable that minimal, "sensible" sun exposure in adults may be part of that answer. For now, the best advice remains to use sun protection. Sun protection should begin in infancy and continue throughout life; education of parents and children is the key to its success. With the introduction of high-SPF, broad-spectrum, waterproof sunscreens and high-SPF clothing fabrics, the wilderness enthusiast can enjoy the outdoors more safely.

The references for this chapter can be found on the accompanying DVD-ROM.

15 Volcanic Eruptions

Joanne Feldman and Robert I. Tilling

Volcanic eruptions are spectacular, violent, and often quite dangerous expressions of Earth's dynamic internal processes (Fig. 15-1). More than 80% of the earth's surface above and below sea level is of volcanic origin, and gaseous emissions from volcanoes helped form the earth's oceans and atmosphere.[14] On average, about 50 to 60 eruptions occur worldwide each year, half of which are continuations of previously erupting volcanoes, and the others are new eruptions.[37,44] There are approximately 600 active volcanoes in the world, and probably another 800 that have erupted at least once during the past 10,000 years.[44] Some volcanoes erupt only once in their lifetime, whereas others erupt repeatedly or even continuously. The largest explosive eruptions occur infrequently; in general, the longer the time interval between eruptions, the larger the next eruption tends to be. Sometimes, the worst volcanic catastrophes in history have occurred at volcanoes believed to be extinct, including the famous eruption of Vesuvius in AD 79, which destroyed the cities of Pompeii and Herculaneum (see later).[44] In fact, in the last 2 centuries, 11 of the 16 largest eruptions were the first eruptions known in historical times.[34,37] Table 15-1 lists some notable historical eruptions.

Volcanic eruptions have been responsible for the deaths of approximately 300,000 people in the past 400 years. In recent years, the average is two to four fatal volcanic events per year.[38] Cause of death and number of fatalities are not well documented.[38] Most deadly of the volcano hazards, pyroclastic flows have claimed the most lives, whereas tephra is the most common killer (Box 15-1). Long after the eruption, famine and disease epidemics are responsible for up to a third of the total fatalities attributed to explosive eruptions (Tables 15-2 and 15-3).[38]

Compared with other natural disasters, volcanic eruptions occur infrequently, affect few people, and are responsible for only a small percentage of fatalities. Only some 2% of all natural disasters are from volcanic activity.[20,39] The deadliest volcanic eruption in history, Tambora, Indonesia, in 1815, killed 92,000 people, whereas 1 million people were killed in the worst hurricane (Ganges Delta in Bangladesh, 1970) and 830,000 were killed in the worst earthquake (Shaanxi earthquake in China, 1556).[22,36,44] Nevertheless, volcanoes have the potential to unleash one of the most destructive forces on Earth. Approximately 74,000 years ago, an Indonesian volcano named Toba exploded and ejected 2800 km^3 (670 cubic miles) of ash, dust, and volcanic gases high into the stratosphere, where it was carried around the world by high-altitude winds. The particulate matter interfered with solar radiation and is thought to have led to a 10°C (22°F) temporary global cooling of the earth's surface.[36,50] Such a degree of cooling today would be catastrophic on a global scale.

We can expect more fatalities from volcanic eruptions as human settlements inexorably encroach on areas with high-risk volcanoes.[2] Current estimates suggest that about 500 million people, or 10% of the world's population, live within 100 km (62 miles) of a volcano that has been active in the historical record.[2,30] Auckland, New Zealand, occupies an area of young volcanoes. Seattle–Tacoma, Washington, is on land that could be devastated by mudflows from an eruption of Mount Rainier. Naples, Italy, is built on the flanks of Vesuvius, where in the first minutes of a major eruption more than 100,000 people could be killed (Fig. 15-2).[2,7,34] Latin America and the Caribbean are areas of particularly high risk because of population density. During the 20th century, 76% of all fatalities from volcanic eruptions occurred in this region, and in the past 10 years, half of the most powerful eruptions occurred in this geography. In general, most fatalities occur in densely populated, less developed countries (Table 15-4).[30]

Although volcanic eruptions constitute a significant natural hazard, other processes and products of volcanism can be highly beneficial to society, explaining in part why so many people live on or near volcanoes.[36] Volcanic ash rejuvenates soil and can prevent the loss of phosphorus, resulting in highly productive agricultural land (Fig. 15-3). Volcanic ore deposits supply diamonds, copper, gold, silver, lead, and zinc. Products of volcanic activity are used as building materials, as abrasive and cleaning agents, and for many chemical and industrial uses.[14] Geothermal heat and steam can drive turbines to generate electricity, heat homes and industries directly, and be enjoyed by people at hot spring resorts (Fig. 15-4). The beauty of volcanoes and volcanic activity also generates income for communities through tourism.

VESUVIUS, AD 79

The best-known volcanic eruption was Vesuvius on August 24, AD 79. This eruption killed thousands of people, devastated the surrounding countryside, and destroyed at least eight towns, most notably Pompeii and Herculaneum. Prior to this eruption, Vesuvius was seen as a benign mountain with lush vineyards planted on the slopes and human settlements on the mountain's flanks. Unlike the nearby often-erupting volcanoes Etna and Stromboli, Vesuvius was not considered volcanic. Even a series of pre-eruption earthquakes, as is typical before eruptions, did little to raise concern that Vesuvius was an active volcano. The eruption was witnessed and documented by Pliny the Younger. In two letters written to the Roman historian Tacitus, Pliny wrote that the first explosion began in the early afternoon and

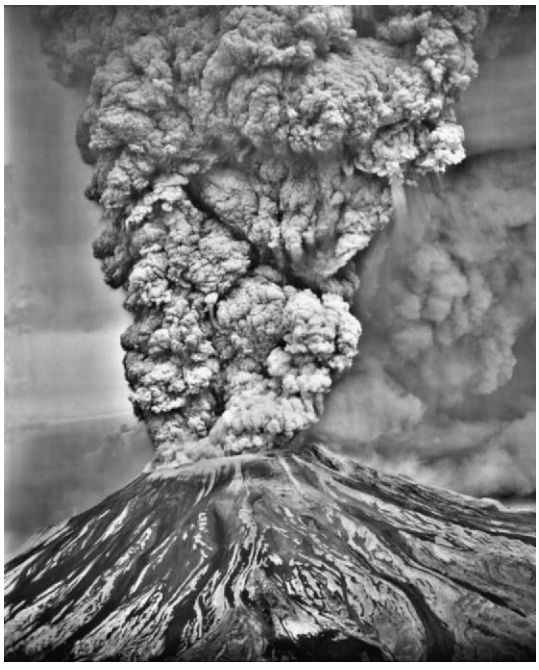

Figure 15-1. Eruption of Mount St. Helens on May 18, 1980. (Courtesy Robert M. Krimmel, U.S. Geological Survey.)

Box 15-1. Glossary of Terms

Lahar	Volcanic mudflow
Lava	Magma that has erupted at the earth's surface
Magma	Molten rock inside the earth
Pumice	Lightweight solidified fragments of magma ejected from an eruption
Pyroclastic flow	Hot ash and rock clouds that sweep down slopes at hurricane speeds
Tephra	Ejected volcanic material such as ash, pumice, and rocks
Tsunami	Seismic sea wave generated by an earthquake or eruption
Volcanologist	Scientist who studies volcanoes

ended in the evening of the following day. At first, Vesuvius produced an enormous eruption cloud that ejected ash, pumice, and volcanic gases vertically up to 30 km (19 miles) high. Then, from a darkened sky, ash and pumice rained down on the towns of Pompeii and Straide, causing roofs to collapse under the weight of the ash fall and burying the towns (Fig. 15-5).

The town of Herculaneum, lying at the foot of Mount Vesuvius on a cliff overlooking the sea, was initially spared from burial. The prevailing wind blew away from the town and toward Pompeii. But in the early hours of August 25, Vesuvius

TABLE 15-1. Notable Historical Eruptions

VOLCANO	COUNTRY	YEAR	SIGNIFICANCE
Santorini	Greece	1650 BC	One of the largest explosive eruptions on Earth; may have contributed to the decline of Minoan civilizations
Vesuvius	Italy	AD 79	First major historical eruption to occur within range of major cities; first eruption to be well documented by an eyewitness (Pliny the Younger)
Laki	Iceland	1783	Largest historical lava flow eruption on Earth; catastrophic impact on Iceland's population from famine caused by loss of livestock from poisoning by hydrogen fluoride emissions
Tambora	Indonesia	1815	Largest historical volcanic eruption on Earth; highest estimated eruption column (43 km [29 miles]); largest known death toll of over 92,000 (10,000 directly by pyroclastic flows* and tephra*; 82,000 indirectly from starvation and disease); global sulfuric acid aerosols caused worldwide climate change resulting in the "Year Without a Summer" in 1816; abnormally low temperatures in the northern hemisphere caused famine from widespread crop failure
Krakatau	Indonesia	1883	First large eruption of the modern age; $^2/_3$ of the island destroyed; tsunami* killed 36,000 people living in adjacent coastal areas
Montagne Pelée	Martinique	1902	Classic example of a moderate-size eruption causing severe loss of life: 29,000 people died
Mount St. Helens	United States	1980	First major explosive eruption to be monitored intensively with modern technology; worst volcanic disaster in U.S. history; $1 billion worth of damage but only 57 people died because timely forecasting prompted evacuation
El Chichón	Mexico	1982	Erupted violently three times; first eruption came as a complete surprise; pyroclastic flows killed 2000; worst volcanic disaster in Mexican history
Nevado del Ruiz	Colombia	1985	Example of small eruption causing severe loss of life; lahar* caused 23,000 deaths and $212 million of damage; worst volcanic disaster in Colombian history; disaster demonstrates that in densely populated areas, even very small eruptions can cause widespread devastation and kill thousands
Mount Pinatubo	Philippines	1991	Second largest eruption of the 20th century; major societal impact in Philippines; important but temporary global atmospheric effects; despite its huge size, eruption directly caused only 300 deaths because of timely evacuations prompted by precise forecasts by scientists

*See definitions in Box 15-1.

Modified from Sigurdsson H: The history of volcanology. In Sigurdsson H, Hougthon BF, McNutt SR, et al (eds): Encyclopedia of Volcanoes. San Diego, Academic Press, 2000; Tilling RI: Rev Geophys 27:239, 1989; Clemens JA: Volcano! Evacuation and military medical implications. ADF Health 3:25, 2002.

TABLE 15-2. Causes of Fatalities from Notable Volcanic Disasters since AD 1000

VOLCANO	COUNTRY	YEAR	NO. OF DEATHS ACCORDING TO PRIMARY CAUSE				
			PYROCLASTIC FLOW	LAHAR	TSUNAMI	LAVA FLOW	POST-ERUPTION STARVATION
Merapi	Indonesia	1006	1,000	—	—	—	—
Kelut	Indonesia	1586	—	10,000	—	—	—
Vesuvius	Italy	1631	—	—	—	18,000	—
Etna	Italy	1669	—	—	—	10,000	—
Merapi	Indonesia	1672	300	—	—	—	—
Awu	Indonesia	1711	—	3,200	—	—	—
Oshima	Japan	1741	—	—	1,480	—	—
Cotopaxi	Ecuador	1741	—	1,000	—	—	—
Makian	Indonesia	1760	—	3,000	—	—	—
Papadajan	Indonesia	1772	2,960	—	—	—	—
Laki	Iceland	1783	—	—	—	—	9,340
Asama	Japan	1783	1,150	—	—	—	—
Unzen	Japan	1792	—	—	15,190	—	—
Mayon	Philippines	1814	1,200	—	—	—	—
Tambora	Indonesia	1815	12,000	—	—	—	80,000
Galunggung	Indonesia	1822	—	4,000	—	—	—
Nevado del Ruiz	Colombia	1845	—	1,000	—	—	—
Awu	Indonesia	1856	—	3,000	—	—	—
Cotopaxi	Ecuador	1877	—	1,000	—	—	—
Krakatau	Indonesia	1883	—	—	36,420	—	—
Awu	Indonesia	1892	—	1,530	—	—	—
La Soufriere	St. Vincent	1902	1,560	—	—	—	—
Montagne Pelée	Martinique	1902	29,000	—	—	—	—
Santa María	Guatemala	1902	6,000	—	—	—	—
Taal	Philippines	1911	1,330	—	—	—	—
Kelut	Indonesia	1919	—	5,110	—	—	—
Merapi	Indonesia	1951	1,300	—	—	—	—
Lamington	Papua New Guinea	1951	2,940	—	—	—	—
Hibok-Hibok	Philippines	1951	500	—	—	—	—
Agung	Indonesia	1963	1,900	—	—	—	—
Mount St. Helens	United States	1980	57	—	—	—	—
El Chichón	Mexico	1982	>2,000	—	—	—	—
Nevado del Ruiz	Colombia	1985	—	>25,000	—	—	—
	Totals:		>65,200	>53,840	53,090	28,000	89,340
	GRAND TOTAL				289,470		

From Tilling RI: Rev Geophys 27:239, 1989.

TABLE 15-3. Fatalities from Volcanic Eruptions, 1783–2000

VOLCANIC HAZARD	FATALITIES	
	No.	%
Post-eruption famine and disease epidemics	75,000	30
Pyroclastic flows	67,500	27
Lahars	42,500	17
Volcanogenic tsunamis	42,500	17
Debris avalanches	10,000	4
Air fall	10,000	4
Volcanic gases	1,750	<1
Lava flows	750	0.3
TOTAL	250,000	—

Modified from Baxter PJ: In Sigurdsson H, Hougthon BF, McNutt SR, et al (eds): Encyclopedia of Volcanoes. San Diego, Academic Press, 2000.

exploded again, this time ejecting hot gases, ash, and pumice down the mountain's slopes as a pyroclastic flow. Herculaneum was destroyed, buried beneath more than 20 m (66 feet) of pumice and ash. Whatever remained of Pompeii was also destroyed at that time.[33] At about that time, Pliny the Younger wrote of the death of his uncle, Pliny the Elder, who probably died of asphyxiation from being caught too close to a pyroclastic flow.[2]

The remains of more than 2000 people found amid the ruins offer clues as to the causes of death. People unwilling or unable to flee their homes were instantaneously suffocated as hot volcanic ash and gases entered buildings. Others had multisystem trauma as the force of the explosion and pyroclastic flows scooped up rocks and building materials and smashed them into anything in their path. Skeletons recently uncovered in beach caves in Herculaneum suggested that the cause of death was the intense heat, roughly 500°C (930°F) (Fig. 15-6).[9,25,42]

A

B

C

Figure 15-2. **A,** Mount Eden, one of 50 geologically young but dormant volcanoes in the Auckland Volcanic Field, within the city of Auckland, New Zealand. **B,** Mount Rainier sits just 137 km (85 miles) southeast of Seattle-Tacoma, Washington. (Courtesy Lyn Topinka, U.S. Geological Survey.) **C,** Bay of Naples, Italy, with Mount Vesuvius in background. (**A** courtesy Lloyd Holmer, Institute of Geological and Nuclear Sciences, New Zealand; **B** courtesy Robert I. Tilling, U.S. Geological Survey.)

Figure 15-3. Farmer plowing a lush rice paddy in central Java, Indonesia. Sundoro Volcano looms in the background. The most highly prized rice-growing areas have fertile soils formed from the breakdown of young volcanic deposits. (Courtesy Robert I. Tilling, U.S. Geological Survey.)

TABLE 15-4. Fatalities from Volcanic Eruptions by Region, 1600-1982

	FATALITIES	
REGION	No.	%
Indonesia	161,000	67
Caribbean	31,000	13
Japan	19,000	8
Iceland	9,400	4
Everywhere else	19,000	8
TOTAL	238,900	100

Modified from Blong RJ: Volcanic Hazards: A Sourcebook on the Effects of Eruptions. Orlando, FL, Academic Press, 1984.

VOLCANOES AND THEIR GLOBAL DISTRIBUTION

Different images are associated with the word *volcano*—for example, a violently erupting Mount St. Helens, a quiet snow-capped Mount Villarica in Chile, or rivers of lava flowing down the flanks of Kilauea Volcano in Hawaii (Fig. 15-7). *Volcano* also means the opening, or vent, in the earth's crust through which molten rock, ash, and gases are ejected. Molten rock, while underground, is called *magma*. It becomes *lava* once it reaches the surface. Whether a volcano erupts explosively (like Vesuvius or Mount St. Helens) or nonexplosively (like the lava flows of Kilauea) depends on the magma—its composition, tem-

Figure 15-4. Blue lagoon in Iceland, with Svartsengi power plant in the background. Clean geothermal energy heats 87% of the homes in Iceland, including all of the capital city, Reykjavík. Excess geothermal water (which is absolutely clean) is ejected into the lagoon, and where there is swimming, the temperature averages about 40° C (104° F). The lagoon is a very popular tourist destination. (Courtesy Mary Dagold.)

Figure 15-5. View from the main square of the well-preserved ruins of Pompeii, with Vesurius in the background. (Courtesy Harvey E. Belkin, U.S. Geological Survey.)

perature, gas content, viscosity, and crystal content.[36] Magma that is fluid and hot tends to erupt frequently (every few years) and generally nonexplosively. This type of volcano produces fountains or rivers of red-hot lava.[46] In contrast, magma that is less fluid and cooler, and that contains trapped gases, tends to rise sluggishly and can plug up the volcanic vent. If enough pressure builds up as trapped gases expand during ascent, the pent-up pressure can blow the plug, abruptly unleashing the expanding gases and producing a violent eruption.[21]

The most volcanically and seismically active zone in the world, called the Ring of Fire, coincides roughly with the borders of the Pacific Ocean (Fig. 15-8).[21] The volcanically active countries in this zone include Russia, Japan, the Philippines, Indonesia, Papua New Guinea, New Zealand, and the countries on the Pacific coasts of North, Central, and South America. Volcanoes are also scattered in the Atlantic and Pacific Oceans—in Hawaii, Iceland, and the Galapagos.

Figure 15-6. The "Garden of the Fugitives," in Pompeii, Italy, reveals 13 adults and children huddled together in a futile attempt to shield themselves from pyroclastic flows from the eruption of Vesuvius in AD 79. The human casts are obtained by filling cavities left in hardened ash with liquid chalk. (Courtesy Lancevortex.)

Theory of Plate Tectonics

The global distribution of volcanoes, as well as the origin and distribution of mountain ranges and earthquakes, is explained by plate tectonics.[31,44] This theory suggests that the earth's outermost layer is broken into a number of rigid plates that move relative to one another as they float atop the hotter, semisolid, more mobile material of the mantle. The nature and distribution of volcanic activity depends on the formation, movement, and destruction of these plates at their margins (Fig. 15-9).

When two tectonic plates move away from each other, or diverge, new crust is created as molten rock pushes up from the mantle and oozes out onto the earth's surface or the seafloor. One well-known, mostly oceanic, divergent boundary, the Mid-Atlantic Ridge, is a submerged mountain range in the Atlantic Ocean that extends from the Arctic Ocean to beyond the southern tip of Africa. Along the Mid-Atlantic Ridge are numerous volcanoes, including those that rise above sea level on Iceland (Fig. 15-10). Another prominent divergent boundary, entirely continental, is the 11,000-km-long (6835-mile-long) Great African Rift. About 75% of Earth's volcanism occurs unseen along divergent boundaries, deep on the ocean floor.

Two plates can also move toward one another, or converge. How two converging plates interact depends on the type of crust—continental or oceanic. Continental parts of plates, com-

A

B

C

Figure 15-7. **A,** Violently erupting Mount St. Helens on May 18, 1980. **B,** Villarica Volcano, Chile, looks peaceful and even has a ski lift on its flanks, but the mountain is actually the most active volcano in Chile. **C,** A long river of lava flowing downhill from Kilauea Volcano in 1983. It ultimately enters the ocean 12 km (7.5 miles) away. (**A** courtesy Keith Ronnholm; **B** courtesy Robert I. Tilling, U.S. Geological Survey; **C** courtesy J. D. Griggs, U.S. Geological Survey.)

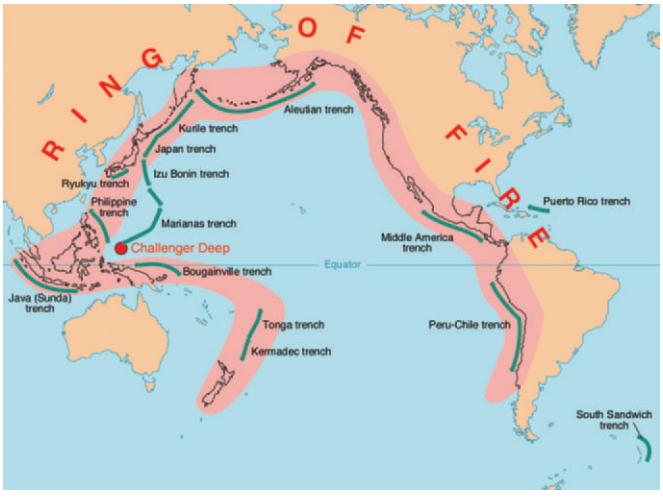

Figure 15-8. The Ring of Fire, a zone of frequent earthquakes and volcanic eruptions encircling the Pacific Ocean. Where the plates come together, they form deep oceanic trenches *(green)*. (From Kious WJ, Tilling RI: This Dynamic Earth: The Story of Plate Tectonics. Washington, DC, U.S. Government Printing Office, 1996.)

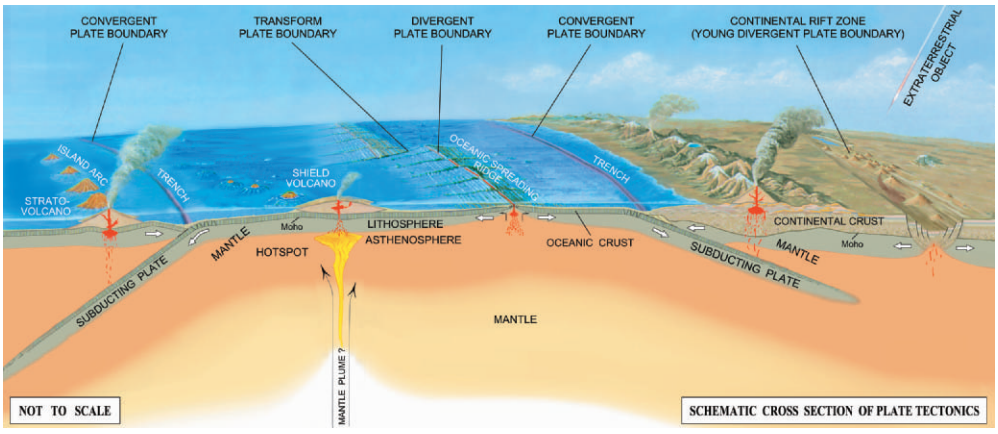

Figure 15-9. Cross section of the main types of plate boundaries. (From "This Dynamic Planet," a wall map produced jointly by the U.S. Geological Survey, the Smithsonian Institution, and the U.S. Naval Research Laboratory.)

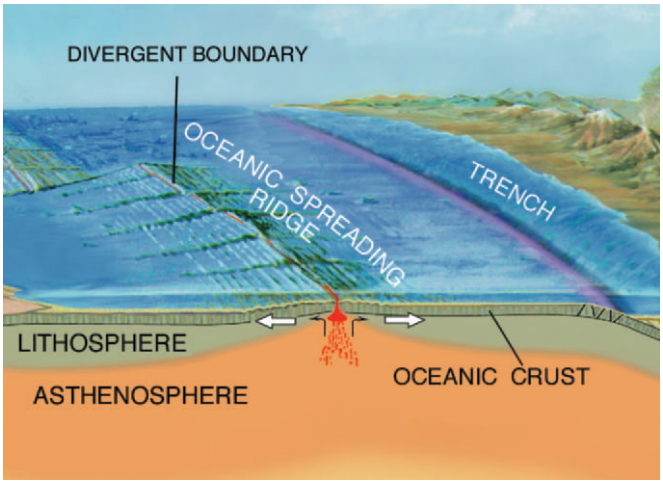

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Figure 15-10. **A,** Cross section of an oceanic divergent boundary. **B,** Map of the Mid-Atlantic Ridge along which the Eurasian and African Plates are pulling away from the North American and South American Plates. Note the ridge rising above sea level in Iceland. **C,** Exposed segment of the Mid-Atlantic Ridge at Thingvellir, Iceland. Left of the fissure, the North American Plate is pulling westward away from the Eurasian Plate (right of the fissure). (**A** from "This Dynamic Planet," a wall map produced jointly by the U.S. Geological Survey, the Smithsonian Institution, and the U.S. Naval Research Laboratory; **B** and **C** from Kious WJ, Tilling RI: This Dynamic Earth: The Story of Plate Tectonics. Washington, DC, U.S. Government Printing Office, 1996.)

posed largely of granite rocks, are relatively lightweight compared to the much denser and heavier oceanic parts of plates, which are composed of basalt. When two continental plates collide, both buckle upward to form a mountain range. The Himalaya Mountains are the result of the Indian plate colliding against the Eurasian plate (Fig. 15-11). Few volcanoes are located in zones of continental collisions.

Many of the world's volcanoes are located at the convergent boundary between a continental plate and an oceanic plate. When these plates converge, the heavier oceanic plate dives, or subducts, below the lighter continental plate. This produces tremendous pressure and heat, melting the rock deep in the mantle. This molten rock, or magma, traps gases, such as CO_2 and SO_2, making it buoyant in the surrounding denser, solid

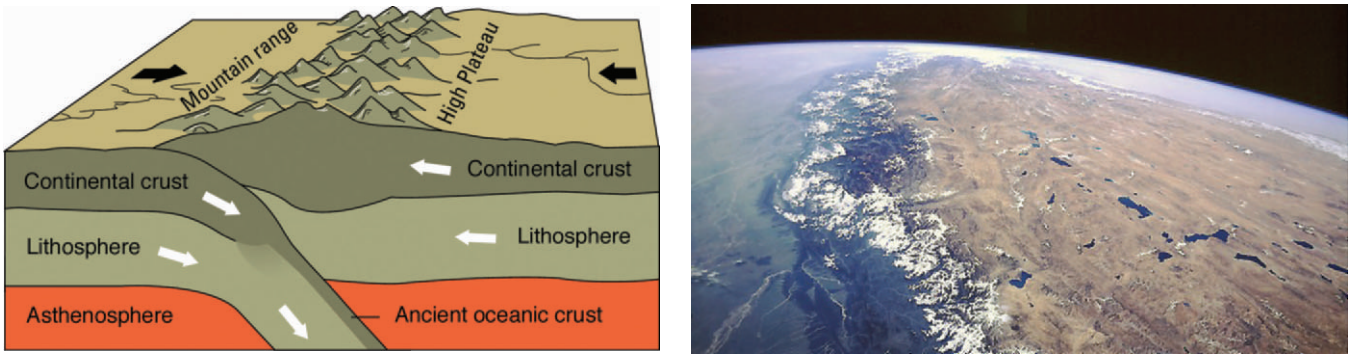


Figure 15-11. **A,** Cross section of two continental parts of plates converging, resulting in the formation of a mountain range. **B,** The snow-capped Himalayas and the Tibetan Plateau are produced by the collision of the Indian-Australian Plate *(left)* against the Eurasian Plate *(right)*. (**A** from Kious WJ, Tilling RI: This Dynamic Earth: The Story of Plate Tectonics. Washington, DC, U.S. Government Printing Office, 1996; **B** courtesy NASA, photo STS41G-120-22, available at http://eol.jsc.nasa.gov/sseop/EFS/printinfo.pl?PHOTO=STS41G-120-22.)

rock, and it begins to rise through the surrounding solid rock. Magma movement causes earthquakes that can be recorded with sensitive volcano-monitoring equipment. If the rising magma finds an area of weakness at the earth's surface, a volcano is created either just inland from the coast—for example, the volcanoes in eastern Russia, the Cascades in North America, and the Andes in South America—or just off the coast, such as the volcanic island-arcs of the Aleutian Islands in Alaska, and Indonesia (Fig. 15-12). Because of their proximity to a continent's coastline or to an island, subduction volcanoes are often near populated regions and thus can have a significant human impact. Eruptions tend to be violent and explosive, such as the 1980 eruption of Mount St. Helens or the 1991 eruption of Mt. Pinatubo in the Philippines.[36]

Some volcanoes are found far from plate boundaries. The Hawaiian volcanoes are more than 3200 km (1988 miles) from the nearest plate boundary, and Yellowstone, with over 10,000 geysers, hot springs, and boiling mud-pools, is located in the interior of the North American plate. At both of these locations, an inferred hotspot below the plate melts overriding rock to produce magma that can rise toward the surface and ultimately erupt onto the seafloor or land. It has been assumed that the hotspot is stationary while the tectonic plate above it moves. However, recent studies have questioned the fixedness of hotspots, prompting substantial debate among earth scientists.

Examination of oceanic hotspots shows that in a series of islands created by plate movement, the islands farthest from the hotspot are the oldest. For example, a 6000-km (3728-mile) chain of volcanoes stretches from the older Emperor Seamounts (underwater sea mountains) off Alaska to the younger Hawaiian Islands. These were all created by passage of the Pacific Plate over the Hawaiian Hotspot. This hotspot, currently located under the Big Island of Hawaii, has remained stationary for about 45 million years, whereas the Pacific Plate has been slowly moving to the northwest. According to the stationary-hotspot model, as the plate continues to move over the hotspot, a new Hawaiian island will eventually emerge above sea level. In fact, just 35 km (22 miles) southeast of the Big Island there is an underwater volcano, Loihi, that has risen 3 km (1.9 miles) above the seafloor and is within 1 km (0.6 miles) of the ocean surface (Fig. 15-13). At hotspot volcanoes, eruptions tend to be nonexplosive and are rarely life threatening.[21] Huge volumes of fluid lava pour out, often creating large volcanic mountains, such as Mauna Loa, the largest single volcano in the world, standing 8851 m (5.5 miles) above the sea floor.

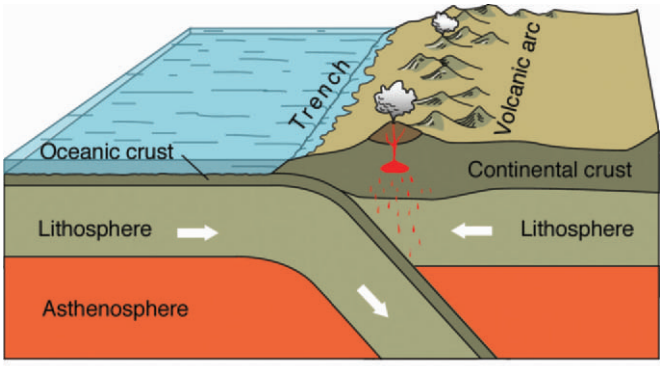
TYPES OF VOLCANOES

As erupted material accumulates around a volcanic vent, a volcano is formed and progressively grows. Classified by structure, the most common types of volcanoes are composite volcanoes, calderas, shield volcanoes, subglacial volcanoes, and flood basalts. These can be roughly grouped as either explosive or nonexplosive volcanoes. However, volcanoes can show both explosive and nonexplosive behavior during their lifespan. For example, Kilauea typically erupts nonexplosively, spewing out fountains and rivers of lava, yet a 1924 eruption was spectacularly explosive, and new studies show that Kilauea's explosive eruptions are as frequent as those of Mount St. Helens.

Generally Explosive Volcanoes

Composite Volcanoes

Composite volcanoes, or stratovolcanoes, have steep sides and are typically associated with subduction zones at converging plate boundaries. Mount Fuji in Japan, Pinatubo in the Philippines, and Mount Rainier in the United States are classic examples. However, composite volcanoes can also be formed at divergent plate boundaries (e.g., Hekla in Iceland and Kilimanjaro in Kenya) or at hotspots (e.g., Mt. Erebus in Antarctica and Tenerife of the Spanish Canary Islands).[33,34] The steep-sided shape results from the deposition of viscous lava flows alternating with pyroclastic flows and air-fall deposits. The explosive nature of composite volcanoes comes from the high viscosity and volatility of the magma (Fig. 15-14).



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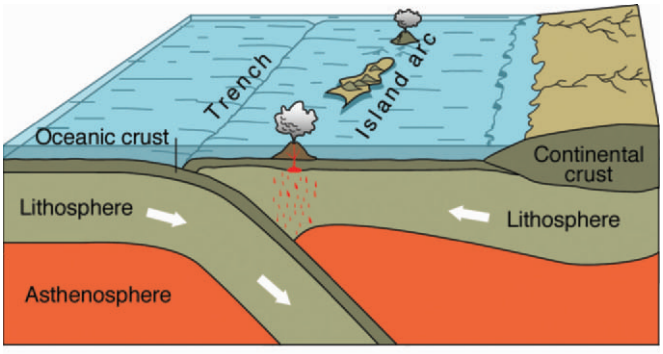

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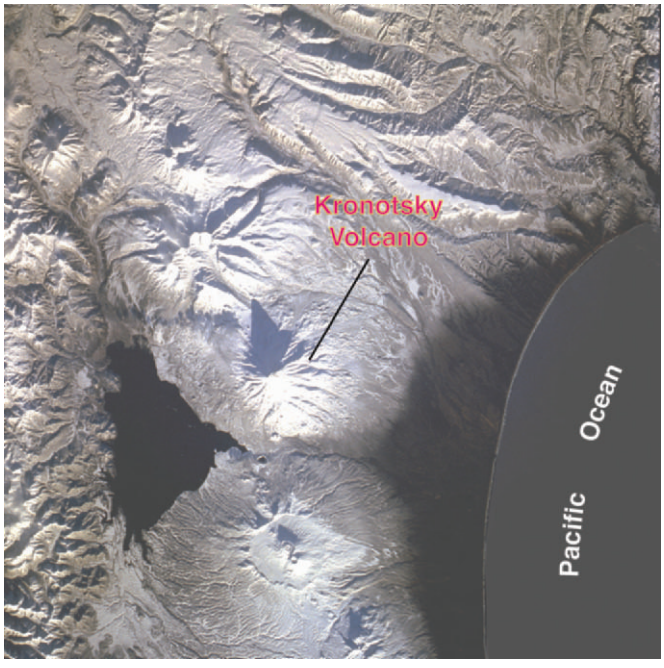

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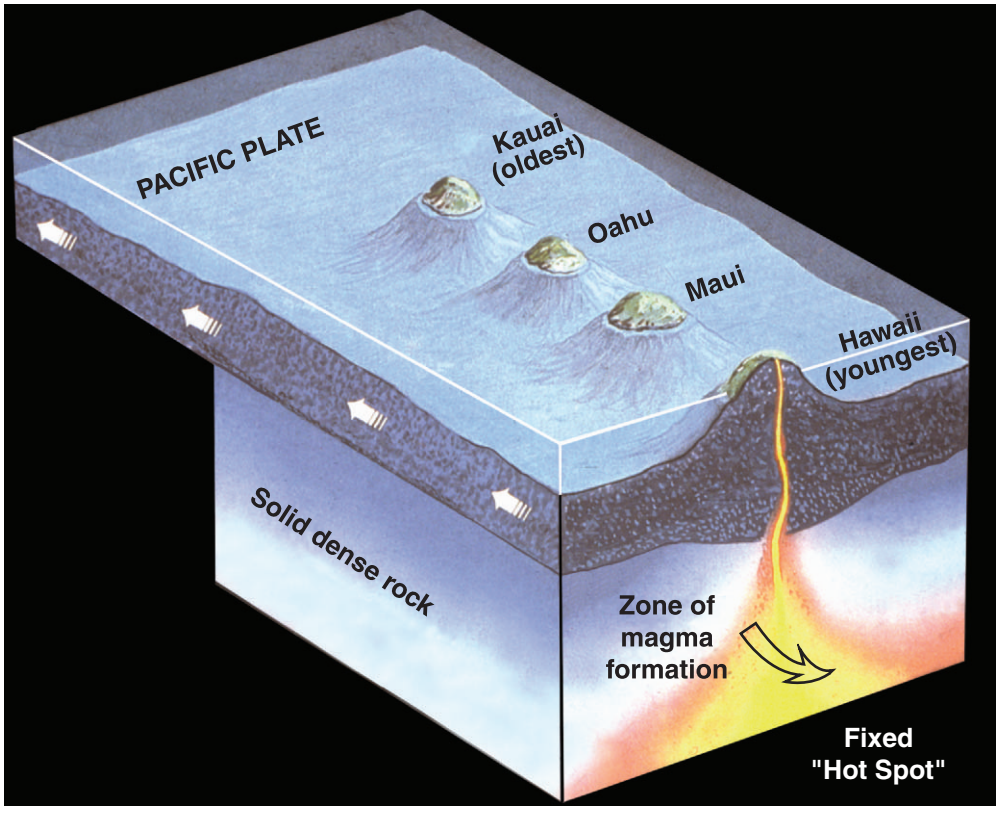
Figure 15-12. **A,** Cross section showing subduction of an oceanic plate resulting in the formation of continental volcanoes and mountains. **B,** Kronotsky Volcano, at 3525 m (11,570 feet) above sea level, lies at the margin of the Pacific Ocean on the Kamchatka Peninsula of Russia. It was formed from the subduction of the Pacific Plate *(right)* under the Eurasian Plate *(left)*. **C,** Cross section illustrating subduction of an oceanic plate with formation of volcanic island arcs. **D,** View of steep-sided, symmetrical Carlisle volcano on Carlisle Island in the central Aleutian Islands. The 1620-m (5315 feet) high stratovolcano has erupted several times since the late 1700s. (**A** and **C** from Kious WJ, Tilling RI: This Dynamic Earth: The Story of Plate Tectonics. Washington, DC, U.S. Government Printing Office, 1996; **B** modified from NASA, photo STS61A-45-0098, available at http://eol.jsc.nasa.gov/sseop/EFS/photoinfo.pl?PHOTO=STS61A-45-98; **D** courtesy M. Harbin, University of Alaska Fairbanks, U.S. Geological Survey.)

Calderas

Formed by the collapse of a volcanic structure, calderas are circular or elliptical depressions, generally over 1 km in diameter (0.62 mile). Crater Lake in the U.S. Cascades is a large, partially filled caldera 10 km in diameter and 600 m deep (6.2 miles wide by 1970 feet deep), formed when Mount Mazama exploded 7700 years ago. Krakatau, in Indonesia, was created by an 1883 eruption that involved rapid emptying of the magma chamber and subsequent collapse of the volcano.[33] Kilauea Crater is another well-known caldera (Fig. 15-15).

Very large calderas fed by huge active magma chambers have been called supervolcanoes and are capable of producing enormously explosive eruptions. Yellowstone Caldera, 85 km long by 45 km wide (46 × 24 miles), the largest and most worrisome in the United States, has a magma chamber that is 40 km long, 20 km wide, and 10 km deep (22 × 11 × 5 miles).[34] Yellowstone has produced explosive eruptions 1000 times larger than the 1980 Mount St. Helens eruption. Two other geologically young, large calderas located in the United States are Long Valley Caldera in California and Valles Caldera in New Mexico. Outside the United States, very large calderas include Campi Flegrei in Italy, Kos Caldera in the eastern Aegean, and Rabaul Caldera in Papua New Guinea. Fortunately, very large caldera-forming eruptions are rare events,



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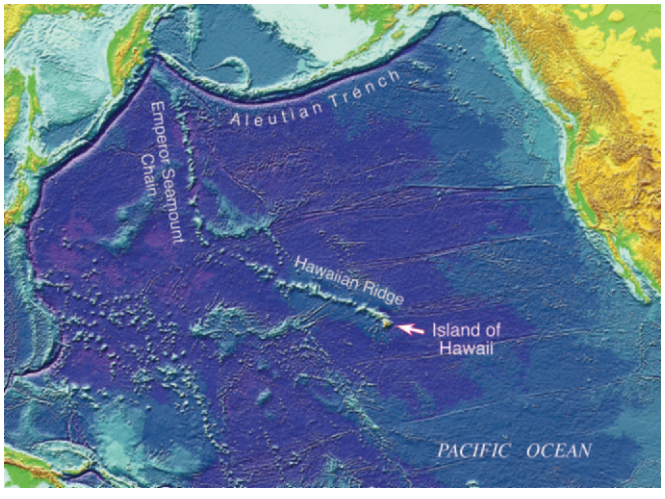


B

Figure 15-13. **A,** Cross section of the Hawaiian hotspot. **B,** Map of the 6000-km (3000 mile)-long Emperor Seamount–Hawaiian Ridge formed by the passage of the Pacific Plate over the Hawaiian hot spot over a span of 70 million years. (**A** from Kious WJ, Tilling RI: This Dynamic Earth: The Story of Plate Tectonics. Washington, DC, U.S. Government Printing Office, 1996; **B** from "This Dynamic Planet," a wall map produced jointly by the U.S. Geological Survey, the Smithsonian Institution, and the U.S. Naval Research Laboratory.)

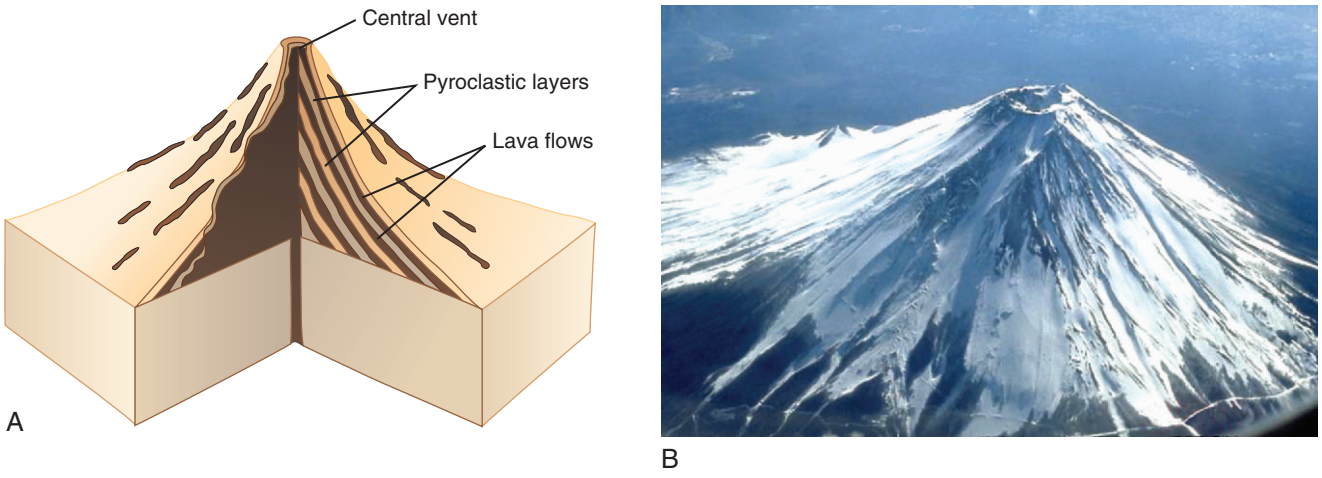


Figure 15-14. **A,** Cross section of a composite volcano (also called a stratovolcano). **B,** Mount Fuji, Japan, is a perfect example of a composite volcano. (Courtesy Thomas C. Pierson, U.S. Geological Survey.)

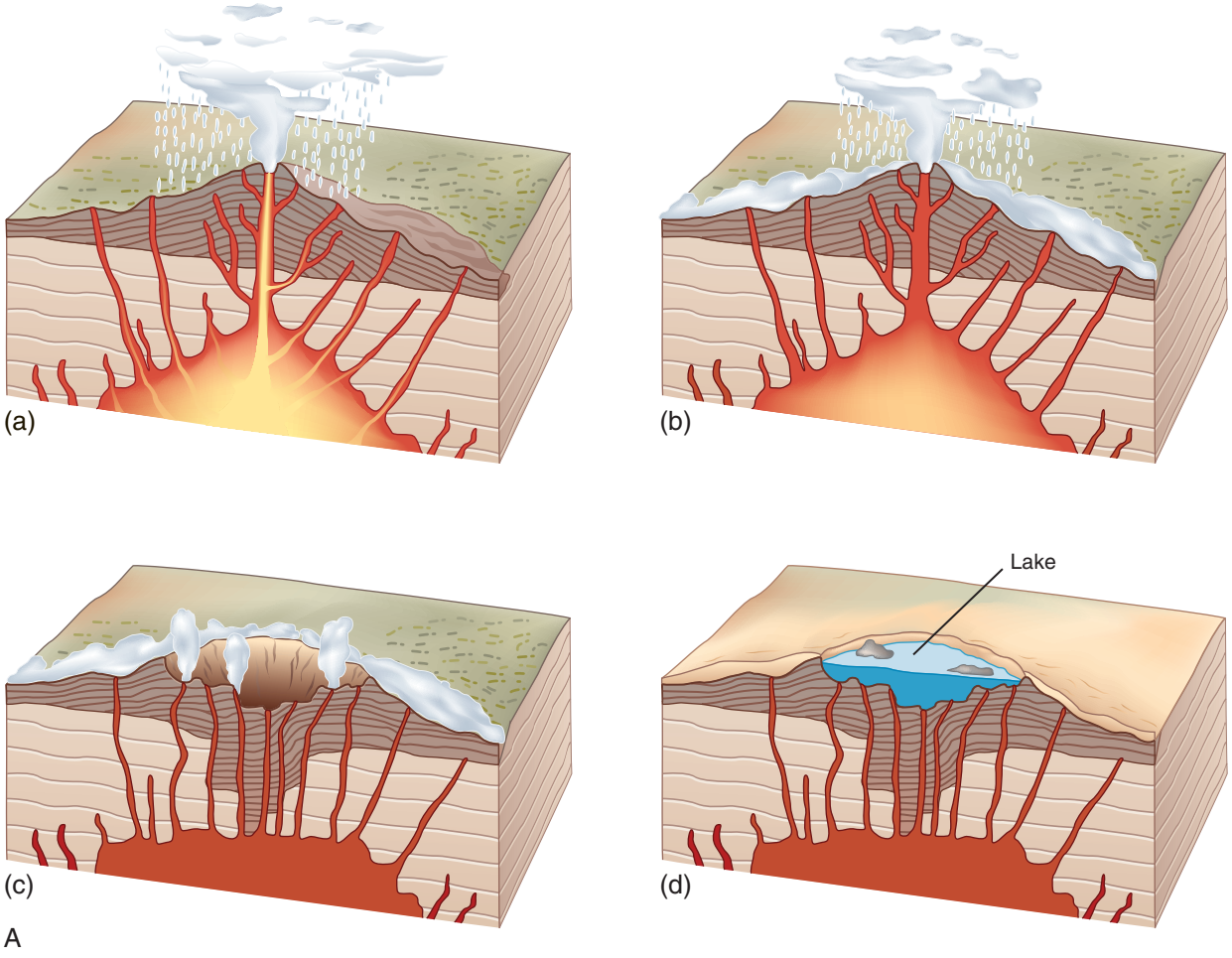


Figure 15-15. **A,** Cross section of the formation of a caldera.

B

C

D

Figure 15-15, cont'd. **B,** Crater Lake, Oregon, was formed by the caldera-forming eruption of Mount Mazama 7700 years ago. **C,** When that mountain erupted cataclysmically, the summit collapsed, forming a caldera that eventually filled with water to form Crater Lake. **D,** Kilauea Caldera, Hawaii, in 1954 after an eruption within the caldera (*dark area* is the new lava). (**B** courtesy Peter Dartnell, U.S. Geological Survey; **C** courtesy Crater Lake Natural History Association; **D** courtesy U.S. Geological Survey.)

occurring approximately once in hundreds of thousands of years (Fig. 15-16).[44]

Generally Nonexplosive Volcanoes

Shield Volcanoes

Shield volcanoes have gentle slopes and are formed almost exclusively of layers of lava that have often flowed great distances from the eruptive vents. The magma, unlike that of explosive volcanoes, is predominantly of low viscosity and low volatility. Shield volcanoes are mostly formed at hot-spots, although some are located at convergent or divergent plate boundaries—for example, Erta Ale in Ethiopia and Etna in Italy. The largest volcanoes on Earth, Mauna Loa and Mauna Kea of Hawaii, are classic examples of shield volcanoes (Fig. 15-17).

Subglacial Volcanoes

Volcanoes located under glaciers, called subglacial volcanoes, can erupt explosively, but the ice cover inhibits material from being ejected high into the atmosphere. Instead, lava flows out, often melting ice and creating subglacial lakes and rivers. If there is a sudden escape of this subglacial water onto the glacier's surface (a glacial burst, or jökulhlaup), a huge river can form and destroy anything in its path. Grímsvötn volcano, in Iceland, lies largely beneath the vast Vatnajökull icecap. The caldera lake is covered by an ice shelf 200 m (656 feet) thick, and only the southern rim of the 6- by 8-km (3.7- by 5-mile) caldera is exposed (Fig. 15-18).[34]

Flood-Basalt Plateaus

Flood-basalt plateaus are massive areas of hardened lava produced from the largest volcanic events known on Earth.[18] Found on all continents and ocean floors, flood-basalt plateaus are created when basaltic magma erupts rapidly from fissures to form sheets of lava flows, typically tens of meters thick and covering tens of thousands of square kilometers.[18] The rate of erupting lava can be 20 times the average eruption rate of a hotspot volcano, and the eruption is on average is 1000 times bigger than that of a supervolcano.[34] Not surprisingly, the copious release of volcanic gases during eruptions of flood-basalts can severely affect the climate, and there is a strong correlation between mass extinction on Earth and the eruption of flood-basalts (Fig. 15-19).[34]

Undersea Volcanoes

Most undersea volcanoes are found at oceanic divergent plate boundaries. Others are found at undersea hotspots or near island-arcs at convergent plate boundaries. Where two plates diverge, magma is injected along the space created. If the magma reaches the seafloor, pillow lava (from slow effusions) or sheets of lava (from more rapid effusions) form. Sometimes, if the magma supply to a single point is sufficiently large and persistent, a cone-shaped volcano, called a seamount, rises from the seafloor. Seamounts found at undersea hotspots become island shield volcanoes, like the Hawaiian volcanoes, if they rise above sea level. Seamounts at subduction zones become island-arc volcanoes, like the volcanoes of Indonesia, above sea level. It is not uncommon for hotspot seamounts to emerge above sea level and then later sink as the seamount moves away from the hotspot and the seafloor subsides under the weight of the lava.

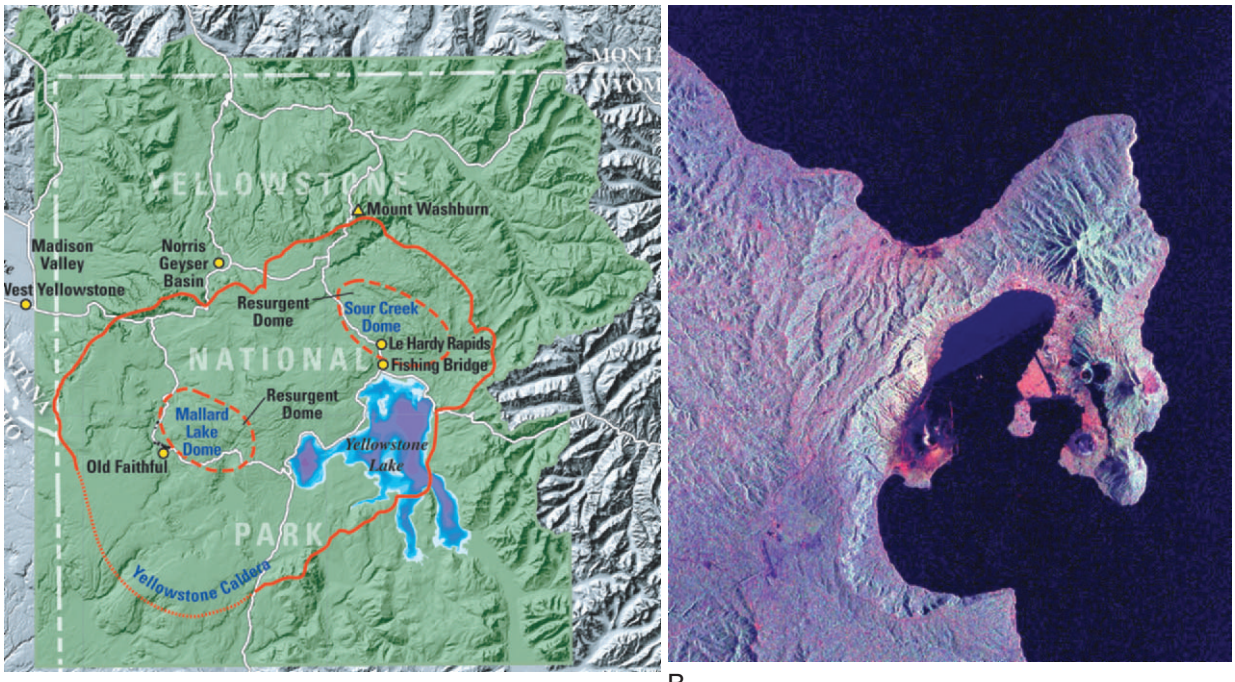


Figure 15-16. **A,** Map of Yellowstone National Park and the 650,000-year-old caldera *(outlined in red)* that is 45 km wide and 85 km long (28 by 53 miles). **B,** View from space of Rabaul Caldera, Papua New Guinea. The caldera is 9 km wide by 14 km long (6 by 9 miles) and filled by the sea. (**A** courtesy U.S. Geological Survey; **B** courtesy NASA.)

Atolls, found in equatorial regions, are the coral reefs around drowning island volcanoes, after they become inactive and sink below sea level (Fig. 15-20).[34]

VOLCANO HAZARDS

Some volcanic eruptions cause injury, death, and destruction within minutes to hours. Others produce hazards that pose risks to human life, livestock, and the environment for months to years after the eruption. Explosive and nonexplosive volcanoes generate unique hazards, but explosive eruptions are usually associated with a larger number of deaths.

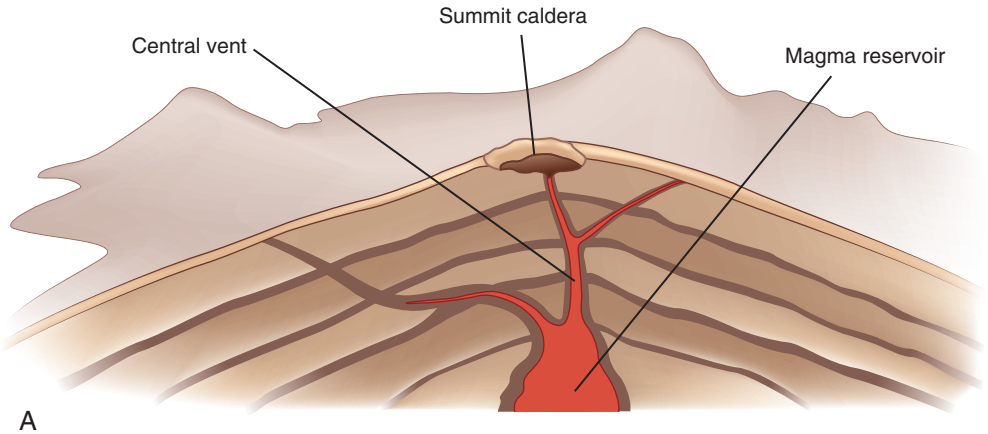
Hazards from Explosive Volcanoes

Volcanoes with explosive characteristics are extremely dangerous. They can cause thermal injury from hot gases and ash; mechanical injury from mud flows, debris avalanches, and falling tephra; chemical injury from toxic gases, and, rarely, electrical injury from lightning or ionizing radiation injury from radon gas (Fig. 15-21).[2] Most volcano-related deaths, injuries, and psychosocial effects occur because the volcano erupts violently and rapidly, leaving little or no time for people to escape or take shelter.[28] However, significant fatalities have been associated with mud flows and release of toxic gases during noneruption periods. Unfortunately, the global distribution of explosive volcanoes, mostly along convergent plate boundaries, places them in densely populated areas.[5] Fortunately, explosive eruptions occur infrequently, and technologic advances have increased the ability of volcanologists to predict an eruption. Prior to the 1980 and 2004 eruptions of Mount St. Helens and the 1991 eruption of Mt. Pinatubo, high-risk zones around the volcanoes were evacuated because of timely warnings.

Pyroclastic Flows and Surges

Commonly, when an explosive volcano erupts, a volcanic cloud of gas, ash, and lava fragments rises vertically into the atmosphere. If this rising eruption cloud becomes denser than the surrounding air, it collapses and falls back toward the ground. Such gravitational collapse of the cloud leads to the formation of a hot, high-speed avalanche of ash, volcanic gases, lava fragments, and heated air, called a pyroclastic flow.[33] Pyroclastic surges are similar to flows but are more dilute and less dense (Fig. 15-22).[2]

There is little chance for survival in the direct path of a pyroclastic flow traveling at 300 km/hr (186 mph) or even greater, with temperatures of 600° to 900° C (1000° to 1650° F).[34] Everything living is carbonized by the extreme temperatures and swept away by the hurricane force of gas, ash, and rock sweeping downhill. Most human victims die as a result of asphyxiation (from breathing overwhelming amounts of ash or from oxygen deprivation), exposure to the intense heat, or burial under volcanic debris. Moreover, fatal traumatic injuries can result from associated devastating winds that can flatten trees and throw rocks. Survivors might have severe and extensive burns to their skin and respiratory tracts, or blunt trauma injuries.[30] Although the main impact lasts a minute or two, ash deposits can remain extremely hot for weeks and pose a



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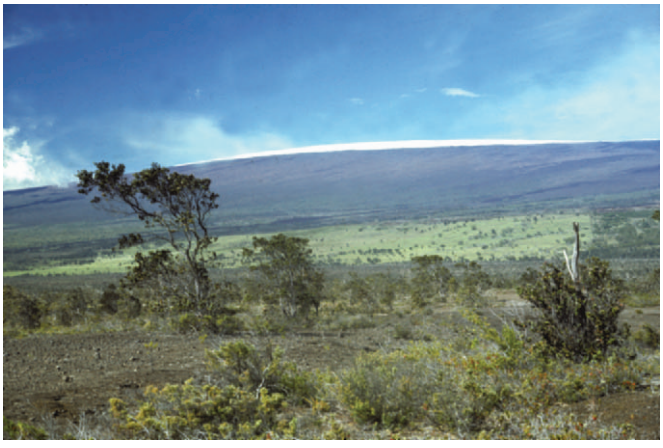

B

Figure 15-17. **A,** Cross section of a shield volcano. **B,** Snow-capped Mauna Loa as seen from the Hawaiian Volcano Observatory on the Big Island of Hawaii. (**B** courtesy Robert I. Tilling, U.S. Geological Survey.)

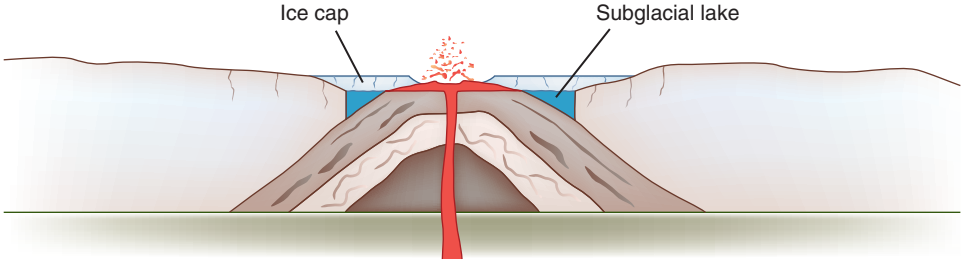

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Figure 15-18. **A,** Cross section of a subglacial volcano. **B,** Subglacial volcano Grímsvötn in Iceland lies largely beneath the vast Vatnajökull icecap. The caldera lake is covered by a 200-m–thick (656 feet) ice shelf. A volcanic plume rises, during the November 2004 eruption, from the exposed southern rim of the 6- by 8-km (3.7- by 5-mile) caldera. (**B** courtesy Freysteinn Sigmundsson, Nordic Volcanological Center.)

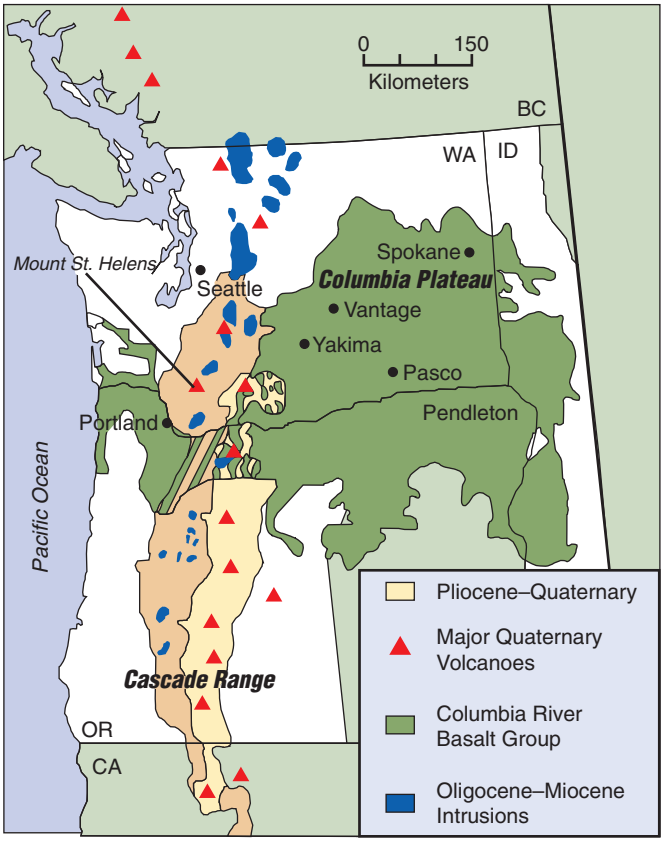


Figure 15-19. The Columbia River Flood-Basalt Plateau *(green)* covers approximately 163,000 km^2 (63,000 square miles) of the Pacific Northwest. In areas, it is up to 1800 m (6000 feet) thick. (Courtesy U.S. Geological Survey.)

lingering danger.[2] Subsequent fires could cause further injury and death.

Some of the most famous volcanic disasters have involved pyroclastic flows and surges. The AD 79 eruption of Vesuvius sent pyroclastic flows down the volcano flanks, killing thousands in nearby towns. The 1980 eruption of Mount St. Helens created a pyroclastic surge cloud that enveloped over a hundred people as it moved at locally supersonic speeds. The 57 people closest to the crater died. Many were buried by volcanic debris, and others died from exposure to intense heat or from asphyxia. One man was killed by a falling tree and another by a rock that flew through his car windshield. Two men died in the hospital from lung injury (acute respiratory distress syndrome) caused by inhaling fine ash.[2] Two others, near the edge of the pyroclastic surge, survived with minimal injury. More people would have died, but the area surrounding Mount St. Helens had been evacuated after swarms of earthquakes and changes in the shape of the mountain signaled an impending eruption. Two weeks after the eruption, ash deposits on the volcano were still 300° to 400° C (570° to 785° F).[45] Gophers were the only animals to survive, because they were below ground.

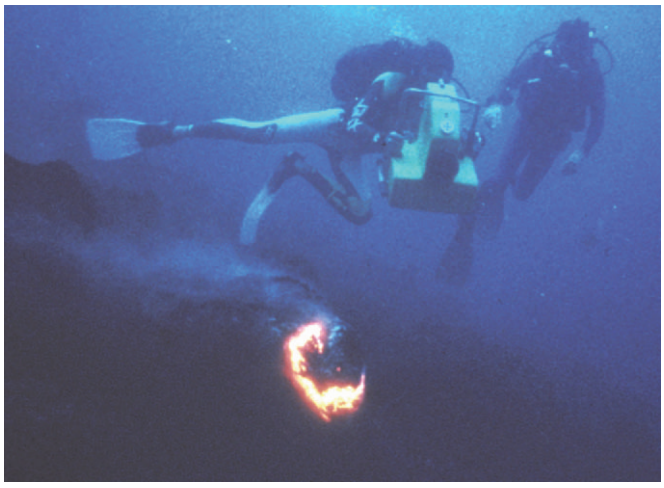
The most devastating eruption of the 20th century occurred on May 8, 1902, when Montagne Pelée, on the Caribbean island of Martinique, sent pyroclastic flows moving greater than 100 km/hr (62 mph) down the mountainside. Within minutes, the city of Saint Pierre, 6 km (3.8 miles) away, was annihilated and 29,000 people killed. Only two survived in the town. At sea, ships were capsized and demasted, and people burned by hot air fall.[34] Since then, Montagne Pelée has been relatively quiet. Saint Pierre has been rebuilt, but the population now is only 5000, much below the 1902 level (Fig. 15-23).

Mitigation. Mitigation of future disasters from pyroclastic flows depends on evacuation prior to an impending eruption. Failure for any reason to evacuate in time will result in many deaths. Unfortunately, many countries with human settlements close to active volcanoes do not have the resources to closely monitor volcanic activity, as the United States did for Mount St. Helens. In Latin America and the Caribbean alone, nearly 60% of all deaths from volcanic eruptions are caused by pyroclastic flows.[30] Survival is possible without evacuation far from the volcano, if there is adequate shelter to protect against high temperatures, asphyxiating gases, falling trees, and projectiles. Once persons are sheltered, windows, doors, and dampers should be closed. Skin should be completely covered, the head swathed in cloth and, to prevent severe lung damage, the breath should be held during the seconds to minutes when the hot cloud passes by. Emergency medical personnel should be prepared to treat burns and blunt trauma injuries.

Lahars (Volcanic Mudflows)

Lahars, the Indonesian term for volcanic mudflows, can accompany most active, explosive volcanoes (particularly those with snow or ice cover), and historically they have been almost as devastating as pyroclastic flows.[30] They occur when hot or cold volcanic debris mixes with water. Because the mixture has a high proportion of sediment, lahars behave like liquid cement and may cover 20 times the area of equivalent rock avalanches.[21] The mass of rock, mud, ash, and water sweeps down valleys, and when it reaches lowland plains, it slows down and spreads out to cover hundreds of square kilometers. Lahars destroy buildings, bridges, and other structures, and leave thick deposits behind (Fig. 15-24). Most fatalities occur when victims are caught in the flow and are killed by trauma resulting from this destruction, by flying debris, and by burial.[2] More deaths occur later from famine and disease as people are displaced from their homes and local water sources become contaminated.

Lahars are especially dangerous because they come unheralded. They can form during or after an eruption, or they may be completely unrelated to eruptive activity.[2] Water is the key ingredient. Lahars need at least 10% water by weight to become mobile.[34] During an eruption, hot pyroclastic material can melt glacial ice or snow, producing water that mixes with ash on the volcano's flank. The water may come from a crater lake drained during an eruption, or from the volcano's own subsurface geothermal system. A subglacial eruption can form a subglacial lake that subsequently breaks out to the surface and mixes with glacial debris. Heavy rainfall occurring during an eruption can initiate lahar formation. Shortly after an eruption, when new volcanic material is on the volcano's flank and there is no groundcover, rainfall can mix with the ash to create a lahar. Even long after an eruption, under circumstances such as a sus-



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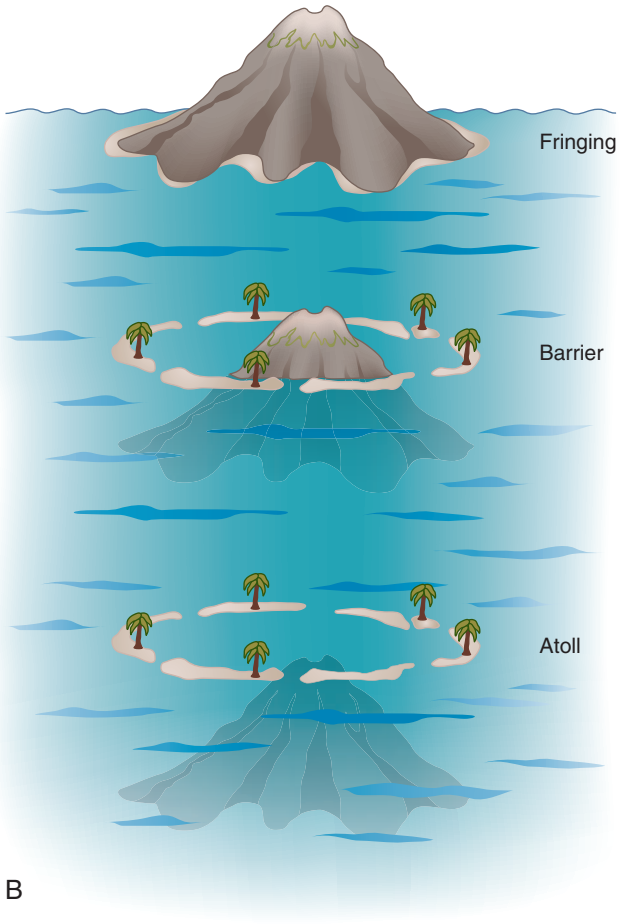


B

Figure 15-20. **A,** Scuba divers filming undersea volcanic activity offshore of Kilauea Volcano. **B,** Formation of an atoll. **C,** Bora-Bora (top right), Society Islands, French Polynesia. The island of Bora Bora includes the remnants of an extinct volcano surrounded by a barrier coral reef. Tupai Atoll (bottom left) is all that remains of a sunken volcano. (**A** courtesy Sharkbait Productions, Kona, HI; **C** courtesy NASA, photo STS068-258-042, available at http://eol.jsc.nasa.gov/sseop/EFS/photoinfo.pl?PHOTO=STS068-258-42.)

tained intense rainfall, water may mix with ash and a lahar (now called a secondary lahar) can start to flow.

Lahars can flow far from the site of the eruption. It is not necessarily the largest ones that produce the highest fatality rate; other factors include the victims' proximity to the hazard and the efficacy of a warning system. The greatest volcano tragedy of recent times began late at night on November 13, 1985, with a small eruption of the Colombian volcano Nevado del Ruiz. Small pyroclastic flows melted the snow cap and glacial ice on the mountain at 1656 m (5432 feet). The water mixed with volcanic ash, forming lahars that swept down the steep valleys and destroyed the town of Armero 50 km (31 miles) away, killing 23,000 inhabitants (Fig. 15-25). Around midnight, the first lahar struck the town, completely unaware of the danger, and covered it with 3 to 4 m (9 to 12 feet) of mud, burying three quarters of the population.[2] Causes of death included suffocation from mud aspiration, trauma, hypovolemic shock, and, later, gangrene. Of the 834 survivors, 578 (69.3%) had lacerations, 343 (41.1%) had penetrating injuries, 312 (37.4%) had fractures, and 272 (32.3%) had eye injuries; many had multiple injuries.[49] Armero had been rebuilt on the site of previous settlements destroyed by similar lahars in 1595 and 1845. Local officials were warned by an on-site scientific team but failed to act.

Another devastating lahar occurred in 1953 when Ruapehu volcano in New Zealand claimed 151 lives after an ash and ice dam retaining its crater lake collapsed. The resultant lahar swept away the Tangiwai railway bridge just minutes before the arrival of the Wellington-Auckland express, and five train cars plunged into the river.[34,46]

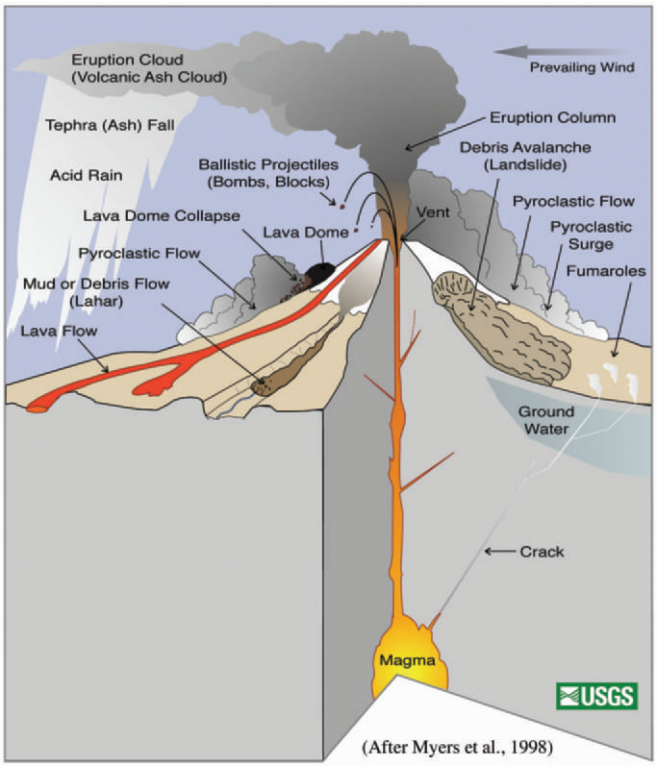


Figure 15-21. Common volcanic hazards. (Modified from Bobbie Meyers, U.S. Geological Survey.)

Figure 15-22. Pyroclastic flow sweeping rapidly down-slope from Unzen Volcano, Japan in 1991. (Courtesy Shigeru Suto, Geological Survey of Japan.)

Mitigation. For communities at risk of being struck by a lahar, evacuation is as important as when the threat is a pyroclastic flow. Early warning systems include using trip wires stretched across lahar channels, seismic instrumentation (e.g., acoustic flow monitors), video cameras, automated rain gauges, and human observers. Also, lahars might be partly diverted or contained by creating channels, and they might be held back by dams and retention basins.[34] To reduce the water source, reservoirs or crater lakes can be drained. Survival is possible without complete evacuation if the population can be moved to higher ground. Caution should be taken when crossing bridges. Emergency medical personnel in high-risk areas need to be prepared to treat blunt and penetrating trauma (crush injuries, lacerations, fractures, eye injuries), hypovolemic shock, and wounds with gangrene complications. Access to victims buried in the thick cementlike mud can be difficult and prolonged.

Tsunamis

Tsunami, the Japanese term for harbor wave, is a seismic sea wave triggered by an earthquake or by volcanic activity. Specific triggers include major earthquakes along subduction zones, submarine landslides, underwater explosions, eruptions of island volcanoes, and avalanches on the flanks of a volcano.[21] Waves can move at speeds more than 800 km/hr (500 mph), and travel hundreds or even thousands of kilometers away from the source. In deep water, a tsunami is generally less than a meter (3 feet) high and of no threat, but as the wave approaches the shore, its speed falls and its height increases until it becomes a steep breaking wave.

Though relatively infrequent, tsunamis pose a severe risk that cannot be ignored, as was illustrated by the 2004 Indian Ocean Banda Aceh undersea earthquake, magnitude 9.0, which generated a tsunami that devastated the shores of Indonesia, Sri Lanka, India, Thailand, and other countries, with waves up to 15 m (50 feet) high, killing more than 165,000 people, though it is hard to know the exact figure (Fig. 15-26).

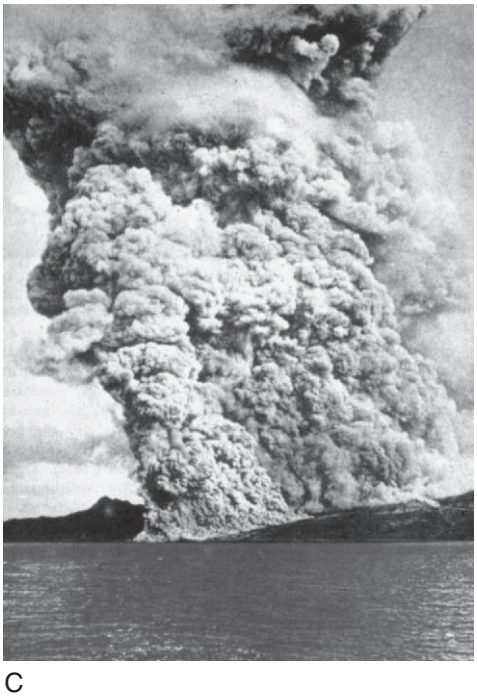
Tsunamis caused by volcanic activity have directly killed more than 42,500 people, which is as many as have been killed by lahars. Tsunamis, like lahars, can come unheralded to settlements close to the source but can also affect distant coastal regions. Fortunately, many of these distant communities can now receive forewarning from the International Tsunami Warning System, which covers the Pacific Basin. Death and destruction occur when a tsunami slams ashore and destroys low-lying coastal communities (Fig. 15-27).

Fatalities associated with volcanogenic tsunamis include those triggered by Mount Unzen, Japan, in 1792 and Krakatau, Indonesia, in 1883. Mount Unzen, on the Japanese island of Kyushu, is an island-arc volcano created above a subduction zone. In 1792, part of the volcano collapsed, probably as a result of seismic activity. The subsequent debris avalanche swept 6 km (3.7 miles) down the volcano to devastate the city of Shimabara, killing over 9500 people. Most of the debris then emptied into the sea and triggered a tsunami that struck much of the seacoast of Ariake, killing another 5000 people in villages and on farms, even those as high as 30 m (98 feet) above sea level, along nearly 100 km (62 miles) of coastline.[33] The famous eruption of Krakatau in 1883, involving volcano collapse and sending pyroclastic flows into the sea, set off a series of tsunamis that washed away 165 coastal villages on Java and Sumatra, killing 36,000 people.[21,34]

Mitigation. Volcanic islands at risk of being hit by a tsunami include the islands of Hawaii, the Marquesas, the Canaries,

A

B



C

Figure 15-23. **A,** City of Saint Pierre, Martinique, with Montagne Pelée in the background, before the 1902 eruption. **B,** City of Saint Pierre showing the destruction from pyroclastic flows from Montagne Pelée, May 8, 1902. **C,** Six months later, another, smaller pyroclastic flow sweeps down the flank of Montagne Pelée, on December 16, 1902. (**A** from an anonymous source; **B** and **C** courtesy Academy of Sciences, France.)

Tristan da Cunha, and Réunion.[34] The International Tsunami Information Center was created in 1965 to warn of impending tsunamis in the Pacific Basin.[21] Hawaii's warning system consists of sirens throughout the islands, and the lobbies of coastal high-rise buildings are designed to allow water to pass through them without causing structural damage.[34] In addition, tsunami warning-system buoys are scattered throughout the Pacific Ocean, and many coastal communities are educated to flee to high ground in case of earthquake or volcanic eruption (Fig. 15-28). Unfortunately, outside the Ring of Fire there is no coordinated tsunami warning system, but since the Banda Aceh earthquake and tsunami, the United Nations and other organizations have been working to establish a global tsunami warning system.

Evacuation to higher ground is essential to reduce the morbidity and mortality associated with tsunamis. Emergency medical personnel need to be prepared to treat near-drownings, the effects of trauma, and disease epidemics.

Debris Avalanches

Volcanoes, especially steep-sided composite volcanoes, can be the site of catastrophic debris avalanches at any time.[33] In 1792 at Mount Unzen, mentioned previously, it was a debris avalanche that swept down the side of the volcano, killing

Figure 15-24. Structure buried by mudflows (lahars) along the Sacobia-Bamban River during the eruption of Mount Pinatubo. (Courtesy R. S. Punongbayan, Philippine Institute of Volcanology and Seismology.)

Figure 15-25. Armero, Colombia, after devastating lahars from Nevado del Ruiz swept down the steep-sided valley *(top center)* on November 13, 1985. (Courtesy Richard Janda, U.S. Geological Survey.)

Figure 15-26. Hundreds of dead bodies and debris are piled along the coast, a dramatic demonstration of the devastation caused by tsunamis affecting the Indian Ocean basin, this one triggered by the Banda Aceh earthquake, December 26, 2004. (Photographer unknown.)

Figure 15-27. Early in the morning on April 1, 1946, an earthquake (magnitude 7.1) in the Aleutian Islands off Alaska sent large, destructive waves toward the Hawaiian Islands. Five hours later, the waves struck the Big Island, killing 159 people, including many curious school children who ventured into the exposed reef area, not knowing the receding water to be a sign of an approaching tsunami. Pictured are people fleeing the approaching tsunami (seen in background) in Hilo, Hawaii. (Courtesy the Pacific Tsunami Museum Archives. Photo by Cecilio Licos.)

A

B

Figure 15-28. **A,** Tsunami buoy contains pressure sensors for determining a wave's size by gauging the weight of the water column passing over it. **B,** "TsunamiReady" sign. (**A** and **B** Courtesy National Oceanic and Atmospheric Administration.)

thousands before emptying into the sea and setting off a devastating tsunami.[33,34] Also in Japan, in 1888, an avalanche from the north flank of Bandai volcano sent 1.5 km^3 (0.36 cubic mile) of debris down the mountainside, killing 400 people below.[33,34] A debris avalanche also contributed to the eruption of Mount St. Helens in 1980. Triggered by an earthquake, a debris avalanche on the north flank of the mountain removed rock overlying a shallow magma chamber. Pent-up volatile gases in the magma, no longer contained by the overlying pressure of the rock, quickly expanded, causing explosive fragmentation of the magma and the first eruption.

Mitigation. Mitigating danger from debris avalanches includes mapping the zones of potential slope collapse and avoiding travel to and construction in those areas. Fatalities and injuries are associated with blunt trauma and burial beneath rock debris. A victim caught in a rock fall should immediately seek some sort of shelter, and roll into a ball to protect the head. Health professionals need to be prepared for victims of blunt trauma.

Tephra

Tephra is the general term for fragmented rock of any size (volcanic blocks, glass shards, pumice, ash) ejected into the air by volcanic explosions. Tephra can range in size from large ballistic ejecta (over 1 m [3 feet]) to ash (less than 2 mm). The largest tephra fall back to the ground at high speeds in the immediate vicinity of the volcano. Smaller fragments stay airborne longer, and the smallest are carried away by prevailing winds before ultimately being deposited.

Ballistic Ejecta. An erupting volcano can eject huge ballistic fragments at high velocities (Fig. 15-29). They can travel kilometers away from the volcanic vent and can cause significant damage (skull injuries, lacerations, blunt trauma of the chest and abdomen) to humans on impact.[2,34] The zone of damage from volcanic ballistics is generally limited to about 5 km (3 miles)—the maximum recorded range—so this is rarely a human health hazard.[2] However, in Italy in 1944, three men were killed at Terzigno by falling volcanic rocks from Mount Vesuvius 5 km away. In 1924, an explosive eruption at Kilauea knocked a photographer down with flying stones and severed his leg. He died later that day, probably from toxic gas exposure.[7] Fire is a more common hazard, as hot flying ejecta can set fire to wooden structures, forests, or grassland.

Volcanic Ash. Smaller-sized tephra, especially ash, pose a much greater danger than blocks and bombs. Drifting volcanic ash can be deposited hundreds to thousands of kilometers downwind from a volcano. Heavy accumulations of ash fall can collapse roofs, interfere with driving, clog machinery, and damage vegetation (Fig. 15-30).

A layer of ash only 10 cm (4 inches) thick is enough to collapse a flat roof, especially if it is wet with rainfall.[30,34] Approximately 10,000 fatalities have been directly associated with ash fall, and the majority of these are from collapsed roofs. In 1991, Mt. Pinatubo in the Philippines erupted during an untimely typhoon. Subsequent ash mixed with rain collapsed roofs and killed at least 300 people.[34]

Figure 15-29. Ballistic ejecta thrown up onto the shore from an explosion caused by hot lava entering the ocean in Hawaii. (Courtesy Christina Heliker, U.S. Geological Survey.)

Decreased visibility from airborne ash and slippery ash-coated roads poses a danger to drivers of motor vehicles. During and after an eruption, the incidence of motor vehicle accidents and associated injuries rises. Moderate amounts of ash fall can also directly damage motors and other machinery, as fine ash clogs engine filters and lubrication systems.[34] Damaged machinery is rarely a risk to human health, except in the case of jet engines. Volcanic ash clouds from powerful explosive eruptions are extremely dangerous to aircraft. The ash can be sucked into the jet engine, melt, and produce glass that chokes fuel nozzles and coats turbine engines, leading to reduced engine performance and ultimately total engine failure. Gritty ash can also abrade aircraft surfaces, including cockpit windows, and contaminate the interiors.

At night, clouds of volcanic ash cannot be distinguished from ordinary meteorologic clouds. In the last 25 years, over 80 commercial aircraft have accidentally flown into eruption clouds, and seven of these encounters caused in-flight loss of the jet engine power.[27] Fortunately, no lives have been lost. In 1982, two jetliners flew into the eruption cloud from Galunggung volcano on Java. The first, a British Airways jumbo jet carrying 240 people, lost all four engines and plummeted 7500 m (25,000 feet) in 16 minutes before cold air chilled and shattered the volcanic glass, and the engines could be restarted. The jet made an emergency landing at Jakarta airport with three functioning engines and flying blind because the windshield was opaque from ash abrasion. A few weeks later, a Singapore Airline jumbo jet flew into a volcanic ash cloud from the same volcano, lost three engines and dropped 2400 m (7900 feet) before one engine restarted and the aircraft made an emergency landing (Fig. 15-31).[7]

A

B

C

Figure 15-30. **A,** An enormous ash cloud from the 1980 eruption of Mount St. Helens passing over the small town of Ephrata, Washington, 233 km (145 miles) to the west. **B,** Ash-covered village of Parentas, Java, after one of the eruptions of Galunggung Volcano in 1982. **C,** In the city of Yakima, central Washington, ash made roads slippery and decreased visibility after the 1980 eruption of Mount St. Helens. (**A** courtesy Douglas Miller; **B** courtesy Maurice Kraft; **C** courtesy U.S. Geological Survey.)

A

B

Figure 15-31. **A,** Eruption cloud from Rabaul Volcano, New Britain Island, Papua New Guinea, September 1994. Note the difference in appearance between the eruption cloud and the ordinary meteorologic clouds. **B,** Aircraft damage after flying into an eruption cloud from Redoubt Volcano in Alaska, December 1989. (**A** courtesy NASA, photo STS064-116-47, available at http://eol.jsc.nasa.gov/sseop/EFS/photoinfo.pl?PHOTO=STS064-116-47; **B** courtesy Joyce Warren.)

Heavy ash can strip vegetation from leaves and coat surviving leaves. Destroyed vegetation and crops result in starvation of animals and can ultimately lead to famine in areas dependent on agriculture for subsistence. Famine from ash-related destruction of crops was the number one cause of fatalities associated with volcanic eruptions before the 20th century. With global communication and world relief organizations, famine is less common.

Most volcanic ash poses little direct danger to humans. Eye irritation may cause conjunctivitis and corneal abrasion, especially in persons wearing contact lenses, but treatment is simple.[30] Inhalation of ash, typically thought to be a significant health hazard, is for the most part benign, except for people with preexisting lung disease, and except for heavily exposed workers, such as gardeners, road workers, and police.[4,6,8,10,15,24,35,48] Most ash particles are too large to be respired, high-dust concentrations are of brief duration, and ash is biologically inert.[30,46]

However, fine ash particles of less than 10 µm in diameter can irritate the lungs. They are especially dangerous for people with asthma and chronic bronchitis, and they result in an increase in visits to emergency departments for exacerbation of chronic lung disease. For example, after Mount St. Helens erupted in 1980, there was a fourfold increase in the number of asthma patients and a twofold increase in the number of bronchitis patients visiting emergency departments in two large hospitals.[4] However, no study has shown long-term consequences of regularly breathing ash, even in the densely populated regions around Mount Sakurajima in Kyushu, Japan, where eruptions occur hundreds of times each year.[48]

Mitigation. To prevent injury from large tephra, danger zones around the volcano should be mapped, and they should be closed when eruption is a possibility. If closure is not possible, concrete shelters can be built in danger zones for the public to access. Hardhats should be worn at all times, and anyone caught near an erupting volcano should watch for missiles and dodge them.[2] Emergency medical personnel need to be prepared for blunt trauma to the head and torso, and for burn victims.

To minimize injury from ash, drive slowly, minimize engine use, and wear eye protection and a dust mask. All machinery should be put inside a garage or barn. Animals and livestock should be brought into closed shelters. Consider evacuating children and high-risk individuals (asthmatics and those with chronic bronchitis) until ash levels are lower. Although communities used to be advised to keep flat roofs clear of ash buildup, this is not now recommended, as people have been injured or killed from falling off roofs while cleaning off ash (Fig. 15-32). Health professionals should be ready for an increase in emergency visits from motor vehicle collisions, eye injuries, and respiratory problems.

To mitigate the dangers associated with aircraft travel near volcanic ash clouds, pilots are now trained in what to do at the first sign of engine failure, and a global system of Volcanic Ash Advisory Centers warns air traffic of drifting eruption clouds.

Volcanic Gases

Gases dissolved in magma can separate explosively or passively from the molten rock and be discharged into the atmosphere. During an explosive eruption, 10 million to a billion tons of volcanic gases can be released into the atmosphere over a period of a few hours to a few days. During noneruptive periods, gas can also escape continuously from fissures, geysers, and other sites of volcanic activity. The most abundant gas released from magma is water. Second is carbon dioxide, closely followed by sulfur dioxide (SO_2). Carbon monoxide, hydrochloric acid, hydrogen, and hydrogen sulfide (H_2S) are other common gases released in appreciable quantities.[33] CO_2 and H_2S are by far the most dangerous. Because they are both denser than air, they collect in low-lying areas and cause harm if inhaled by unsuspecting individuals.[30] CO_2 is an asphyxiant, and at concentrations higher than 20%, even a few breaths can very quickly lead to unconsciousness and death from acute hypoxia, severe acidosis, and respiratory paralysis. H_2S, the colorless gas that smells like rotten eggs, is so toxic that a high-level exposure can

Figure 15-32. Ash removal in Moses Lake, eastern Washington after the 1980 eruption of Mount St. Helens. Keeping roofs clear of ash is not advised because of the risk of falling off. (Photographer unknown.)

kill a human after a single breath.[34] H_2S, much like cyanide, arrests cellular respiration and thus aerobic metabolism.

There are numerous accounts of fatalities associated with carbon dioxide emissions from volcanic activity. Perhaps the most lethal tragedy occurred in 1986 at Lake Nyos, Cameroon, when dangerous amounts of dissolved CO_2, once trapped in the lower levels of this volcanic crater lake, suddenly rose to the surface and were abruptly released like champagne suddenly uncorked.[17] The gas rose 80 m (262 feet) before the CO_2 settled to the ground and rolled down valleys and into villages up to 20 km (12 miles) away, instantly suffocating 1746 people and 3000 livestock. About 1500 others survived and suffered transiently from burns, headache, nausea, vomiting, cough, dyspnea, hemoptysis, and chest pain (Fig. 15-33).[1] Only 2 years earlier, at nearby Lake Monoun, a similar but smaller cloud of CO_2 welled up from the lake, flowed downhill, and killed 39 people.[17] In Indonesia in 1979, a release of CO_2-rich volcanic gas from Sinila Crater (Dieng Plateau) killed 142 local inhabitants as they evacuated the area via a low-lying valley.[23] Less dramatically, CO_2 leaking from underground volcanic systems in Italy has been linked to the deaths of animals and two children, and CO_2 trapped under snow may have contributed to the death of a cross-country skier in the Mammoth Lakes region in eastern California.[16]

There are a few reports of H_2S-associated fatalities. Six downhill skiers on Kusatsu-Shirane volcano in Japan died in 1971, and four hikers who wandered into the lowest part of the summit crater on Adatara volcano in Japan died in 1997. Neither volcano was erupting at the time.[19,34]

Sulfur dioxide is an irritant to mucous membranes and can induce respiratory distress, but it has never been proven to directly cause death.[34] However, in Japan, SO_2 emissions from Mount Oyama on the island of Miyake forced evacuation of Miyake's 3900 residents to the mainland from September 2000 to February 2005.[12] Similarly, there are no fatalities associated with hydrogen fluoride, but indirectly HF is responsible for the death of 10,000 Icelanders in 1783. HF from the prolonged Laki fissure eruption poisoned much of the pastureland and crops, causing the death of half the livestock and crop failure, which ultimately led to famine and death.[32–34]

Figure 15-33. Village of Fulani down-slope from Lake Nyos in Cameroon, Africa, where 25 people died from CO_2 poisoning in 1986. Clusters of white dots on slope are dead cattle, also killed by the CO_2. (Courtesy John P. Lockwood, U.S. Geological Survey.)

Mitigation. Mitigating the danger associated with toxic volcanic gases depends on the type of gas and how it is released. Since the Lake Nyos disaster, scientists have placed 12 pipes into the lake at different depths to allow CO_2 to escape continuously rather than accumulating to dangerously high levels. Danger zones can be mapped and warnings signs can be posted. Equipment can be stationed to monitor gas emissions. Recreationists should be educated to avoid certain activities at times of gas release or buildup under snow. Low-lying areas where poisonous gases can collect should be avoided. Emergency medical personnel should remove victims from the source of toxic gases, place them on high-flow oxygen, and treat other injuries accordingly.

Hazards from Nonexplosive Volcanoes

In general, nonexplosive volcanoes pose little direct danger to humans. Few fatalities are associated with them because lava usually flows slowly enough for people to avoid it.[2,3] Generally, those who died either lingered to watch the lava or returned to their homes too early. On the other hand, rivers of lava can cause significant property damage as they set fire to, knock down, or bury buildings and roads. On rare occasions, flows of low-viscosity lava have been witnessed to travel at up to 100 km/hr (62 mph).

Lava Flows

Fatalities associated with lava flows account for only about 0.3% of total deaths from volcanic activity. The most deadly lava flow took place at Mount Nyiragongo, Congo.[2] Nyi-

Figure 15-34. A rapidly advancing lava flow from Puu Oo vent, Kilauea Volcano, Hawaii, engulfs vehicles. (Courtesy Robert W. Decker, U.S. Geological Survey.)

ragongo is among the few volcanoes with an active lava lake inside its summit crater, and during an eruption in 1977, this lake suddenly drained through fissures in the crater wall. A very fluid lava flow, estimated to be traveling at 100 km/hr (62 mph), swiftly engulfed small towns, killing hundreds of people.[2,29,33,34] In 1947, a scientist filming a lava flow at Mount Hekla in Iceland was killed by a glowing lava block rolling down-slope.[7] Other hazards include methane-fueled explosions when lava overruns vegetation, and explosions or scalding steam when lava flows over ice or snow or into water (Fig. 15-34).[34]

Mitigation. Because lava flows rarely pose a significant health hazard during volcanic activity, they are not a high priority for health planning.[30] However, to limit property damage, lava-flow diversion is a possibility. In 1973, after 6000 years of inactivity, Eldfell volcano on the island of Heimaey in Iceland reawakened and lava poured out, threatening the most important port of the Icelandic fishing industry. To stop or divert the lava flow, cold seawater was sprayed onto the advancing lava front, and after 2 weeks of nonstop work, the port was saved.[33,34] Other attempts at diversion, including building rock and dirt barriers, and aerial bombings,[44] have not been successful. Health professionals should be prepared to treat burns from contact with lava or scalding steam.

Jökulhlaups

A jökulhlaup is a large outburst flood associated with the eruption of a subglacial volcano. The heat from the volcano melts overlying ice, forming a subglacial lake. As the lake rises, the overlying glacier is lifted and begins to melt, forming a subglacial river. If this river bursts to the surface, disastrous flooding can occur. When this happened in 1996 from a subglacial eruption at Grímsvötn in Iceland, water escaped from a 3.5-km (2.2-mile) crevice at a maximum rate of 45,000 m^3/sec (12 million gal/sec) to form, temporarily, the second-largest river on Earth.[34] Miles of road and bridges were destroyed. A smaller jökulhlaup emerged in November 2004, from volcanic activity at Grímsvötn, with a peak discharge of up to 4000 m^3/sec (1 million gal/sec), but this time no roads or bridges were destroyed.[40]

Post-Eruption Hazards

Major eruptions can be hazardous long after the eruption is over. Large quantities of gas released into the atmosphere can cause air pollution, produce acid rain, and deplete ozone, possibly affecting global climate for a few years.[2] Water supplies can be contaminated and crops destroyed, leading to disease epidemics and famine (Fig. 15-35). Ongoing smaller eruptions can also cause problems. In Italy, Etna is the largest continuous source of SO_2 globally, and Stromboli is probably the second largest in Europe.[13]

During the 8 months that the Laki fissure in Iceland was erupting in 1783, at least 122 megatons of SO_2, 7 MT of HCl, and 15 MT of HF were released into the atmosphere.[47] HF poisoned Icelandic sheep and destroyed crops, contributing to the death of about a fifth of Iceland's entire population.[32] In addition, the eruptions at Laki are probably responsible for the death of more than 10,000 people in England during the summer of 1783 and winter of 1784.[32,47] Noting that the mortality rate was 16.7% above normal for this time period, experts in the United Kingdom suggest that a cloud of volcanic gases and particles swept south from Laki into England and was responsible for a very hot 1783 summer and the subsequent severe winter.[32,47] Combing climatic data, burial records, and written accounts, they read of a summertime "volcanic haze" or "dry fog" that shrouded the moon and sun, reduced visibility, withered vegetation, and caused health problems.[13,32] Like smog, the dry fog caused headache and eye irritation, exacerbated lung disease, and irritated mucous membranes. Wintertime deaths were associated with unusually cold temperatures. Benjamin Franklin, as U.S. ambassador to France in Paris, was the first to link the effect of Laki's eruption on climate, and presented a scientific paper on the topic.

Eruption clouds that penetrate the upper atmosphere can spread volcanic particles and gases across an entire hemisphere.[34] During the 3 months it can take fine ash particles to settle out, solar heating is reduced and the lower atmosphere is cooled. The 1991 eruption of Mount Pinatubo in the Philippines created a large volcanic cloud that drifted around the world and caused an average global temperature drop of 0.5°C (0.9°F).[21] The largest eruption in recorded history, in 1815 at Tambora, Indonesia, reduced the average global temperature by 3°C (5.4°F) for several years.[21] This eruption directly or indirectly caused the death of an estimated 92,000 people in Indonesia and was responsible for a typhus epidemic in Great Britain, cholera in India, and mass migration from northern Europe and Russia. It ruined farms in New England

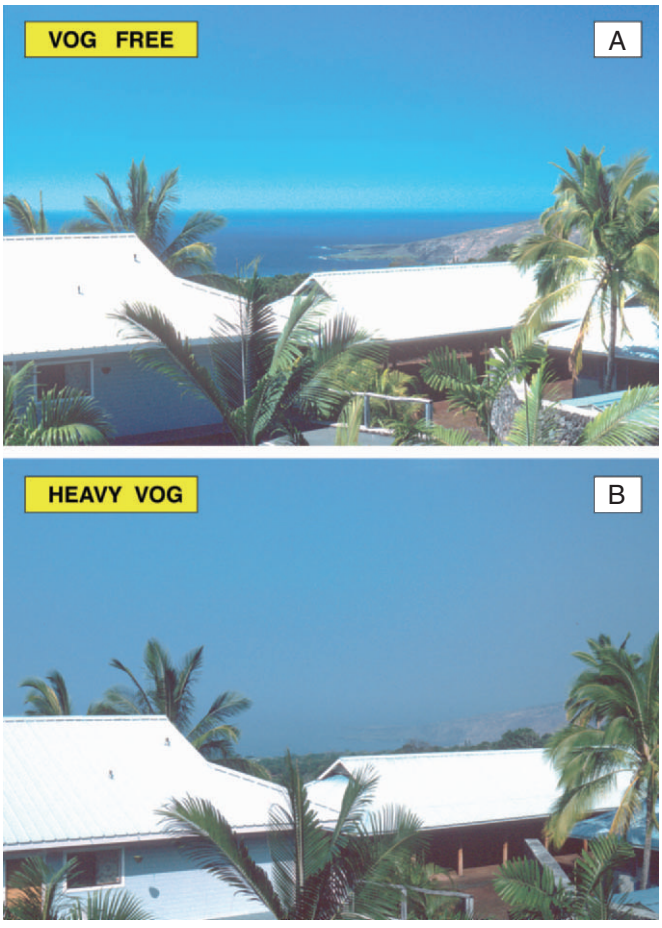


Figure 15-35. The ongoing eruption of Kilauea's east rift zone (which began in 1983) has put a huge amount of natural pollutants, including sulfur oxides, into the air. Introduced vegetation, such as Kona coffee plants, can be scorched, and downwind of Kilauea, only native species, tolerant of the natural acid rain, survive. The two images compare atmospheric clarity between a volcanic-smog (vog)-free day (**A**) and a heavy-vog day (**B**). (Courtesy Jeff Sutton, U.S. Geological Survey.)

and spurred westward migration.[34] The greatest eruption since humans evolved was that of Toba in Indonesia, about 2 million years ago. The global temperature dropped 5° C (9° F) for many years, causing a global environmental catastrophe. It may have resulted in an estimated decrease in the population of *Homo sapiens* from over 100,000 to less than 2000.[34]

RISK REDUCTION FROM VOLCANIC HAZARDS

Humans are vulnerable to severe, unpreventable volcanic eruptions. Risk reduction starts with comprehending the seriousness of volcanic hazards and being prepared for the associated dangers. Nearly 99% of fatalities associated with volcanic eruptions are from pyroclastic flows and lahars, yet one tenth of the world's population live in cities and homes near volcanoes,

Figure 15-36. U.S. Geological Survey scientists sampling volcanic gases within the Mount St. Helens crater. (Courtesy Kathy Cashman, University of Oregon.)

sometimes on the volcano slopes or on the remains of a previous pyroclastic flow, lahar, or debris avalanche.[30,34] Fortunately, most volcanic eruptions are preceded by premonitory events, such as earthquakes and other measurable phenomena, far enough in advance to enable scientists and emergency workers to plan for diaster. However, a few of the severest eruptions have occurred with little or no warning, and most of the world's dangerous volcanoes are in densely populated countries that lack the resources or political interest to monitor them.[3,44] Nevertheless, emergency planning for an eruption, including creation of hazard-zone maps and hazard evaluation, should be routine for all populated areas near volcanoes, regardless of their location or apparent state of activity.[3] Risk reduction includes volcano monitoring, eruption prediction, and effective coordination between volcanologists, scientists, health professionals, and the community at risk.

Volcano Monitoring

Scientists monitor volcanoes for seismic activity (earthquakes), changes in volcano shape (ground deformation), surface temperature, magma level, gas emissions, and other chemical and physical attributes (Fig. 15-36).[28] Earthquakes and ground deformations are the most reliable diagnostic observations for helping scientists predict when a volcano might erupt or when it is no longer likely to erupt.[26,43] The importance of monitoring cannot be overstated. During the last half of the 20th century, most eruptions from monitored volcanoes were anticipated weeks or months in advance. These include the eruptions of Mount St. Helens in 1980 and 2004, and Mount Pinatubo in 1991 (see next). Often, the problem is not predicting when the volcano will erupt but convincing government officials to act on the warning. Before 1985, authorities in Armero, Colombia, were warned that another lahar could devastate their town, but the warning was disregarded, and the eruption at Nevado del Ruiz and the subsequent lahar destroyed the town and killed over 25,000 people.[34]

Figure 15-37. Volcano Disaster Assistance Program "SWAT" team lands near Rabual Volcano, Papua New Guinea, in 1994. (Courtesy Elliot Endo, U.S. Geological Survey.)

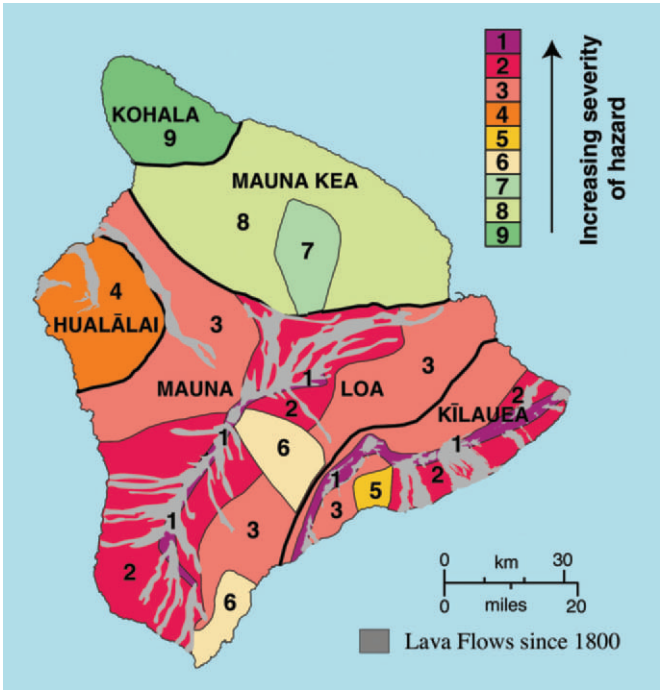


Figure 15-38. Map of the Hawaiian Big Island showing the volcanic hazards from lava flows. Severity of the hazard increases from zone 9 to zone 1. (Courtesy Christina Heliker, U.S. Geological Survey.)

Eruption Prediction and Mount Pinatubo

In April 1991, the Philippine volcano Mount Pinatubo began to erupt weakly for the first time in recorded history. Ultimately, the activity escalated into the world's largest eruption in the preceding 8 decades, yet only 300 people were killed, even though at least 600,000 people lived in cities, villages, and military bases around the volcano. The larger tragedy was prevented by effective cooperation between local authorities and scientists of the Philippine Institute of Volcanology and Seismology (PHIVOLCS) and the U.S. Geological Survey (USGS).

Two months prior to the climatic eruption on June 15, PHIVOLCS consulted the USGS's Volcano Disaster Assistance Program (VDAP), a mobile volcano-response team (i.e., an eruption SWAT team) of experienced volcanologists and other scientists who rapidly respond to a developing volcanic crisis with state-of-the-art portable monitoring equipment (Fig. 15-37). The request for assistance came after there was an increase in the number of earthquakes, up to 500 per day, explosions, and new cracks opening in the volcano. The VDAP team set up a base camp at Clark Air Force Base and began to monitor and interpret seismic activity, ground deformation, and gas emissions. They also gathered data about Pinatubo's previous eruptions to predict the types of hazards that could be expected, and to create a map of the area, delineating zones at risk during an eruption. Finally, the PHIVOLCS-USGS team worked with local authorities to develop an evacuation plan and educate the people. A 30-minute video, "Understanding Volcanic Hazards," produced after the tragedy of Armero, Colombia, in 1985, was shown to local officials and populations to raise awareness of hazardous volcanic processes.

On June 7, a month and a half after the USGS team arrived, a warning was sent out of a possible major eruption within 24 hours. Villages and cities were evacuated. Five days later, the first in a series of powerful explosive eruptions began. An eruption cloud rose 19 km (12 miles) into the air and small pyroclastic flows started down to the north and northwest. Further eruptions took place over the next 2 days. On June 15, a cataclysmic eruption took place during a typhoon. The eruption cloud rose 12 km (7.5 miles) into the atmosphere and spread out like an umbrella more than 200 km (124 miles) in all directions. Wet ash covered a 4000-km^2 (1500-square-mile) area, burying crops and collapsing roofs, and many destructive lahars were generated.[11] Ash fell as far away as the Indian Ocean. The warning saved at least 5000 lives and at least $250,000 in property damage.

Coordination between Volcanologists and Health Professionals

Coordination between geologists, other scientists, and health professionals is essential to prepare communities for volcanic emergencies. Planning for future eruptions requires determining the history of a volcano's past eruptions and their impacts, developing a hazard-zone map, anticipating when a volcano might erupt, and describing the types of hazards to be expected and their potential effects on health (Fig. 15-38). Furthermore, health professionals need to identify high-risk populations, prepare for disaster victims, and help volcanologists educate the community about potential health effects for each volcanic hazard.[28] Evacuation of communities, especially in low-lying areas and river valley regions, is of utmost importance. Goggles and masks should be distributed, especially to individuals who must work in dusty conditions.

Emergency Medical Response

Emergency medical care plays a small role in severe volcanic eruptions. The number of injured who could benefit from treatment is much smaller than the number of victims killed within minutes of a catastrophic eruption (Table 15-5).[3] Therefore, prevention is of utmost importance, and evacuation is the key

TABLE 15-5. Casualties Caused by Pyroclastic Flows in 20th-Century Explosive Eruptions

ERUPTION	YEAR	DEATHS (*N*)	RATIO OF DEAD TO INJURED	SURVIVORS AFTER TREATMENT
Montagne Pelée, Martinique	1902	28,000	230:1	163 treated, 123 survived
La Soufrère, St. Vincent	1902	1,565	11:1	194 treated, 120 survived
Taal, Philippines	1911	1,335	10:1	Not known
Lamington, Papua New Guinea	1958	2,942	44:1	70 treated, 67 survived
Mount St. Helens, United States	1980	58	16:1	130 airlifted, 9 treated, 7 survived
Unzen, Japan	1991	43	5:1	17 treated, 4 survived (minor burns)
Merapi, Indonesia	1994	63	3:1	86 treated, 11 dead on arrival
Soufriere Hills, Montserrat	1997	19	4:1	7 treated, all survived

From Baxter PJ: In Sigurdsson H, Hougthon BF, McNutt SR, et al (eds): Encyclopedia of Volcanoes, San Diego, Academic Press, 2000.

Box 15-2. Summary of Health Effects from Volcanic Eruptions

PHYSICAL

Blunt trauma from pyroclastic material, lahars, debris avalanches, tsunamis, and tephra
Burns, wounds, and gangrene complications
Asphyxiation from lack of oxygen or inhaled ash
Acute irritation of the respiratory tract caused by ash
Exacerbation of prior respiratory disease caused by inhaled particles
Respiratory tract and lung burns caused by inhalation of hot steam
Conjunctivitis and corneal abrasions
Toxic effects of gases such as CO_2, H_2S, SO_2, HF, CO, and radon
Gastroenteritis
Skin irritation from acid water
Drowning in lahars or tsunamis

PSYCHOLOGICAL

Depression
Anxiety
Nightmares
Psychomotor disorders
Irritability
Insomnia
Confusion
Neurosis
Stress

Modified from Zeballos JL, Meli R, Vilchis A, et al: World Health Stat Q 49:204, 1996.

to decreasing morbidity and mortality.[3] Health professionals should be prepared to treat a variety of medical problems in those who survive (Box 15-2), and they must be aware that access to victims will be limited by high ash conditions, burial beneath volcanic debris, and ongoing hazards. Transient increases in emergency department visits and hospital admissions will require additional resources.[6]

People working or traveling near an active volcano should heed these safety recommendations:

- Read about the volcano, including past eruptions and accidents.
- Know the current volcano warning level, and obey local authorities.
- Travel with a guide experienced in local conditions.
- Leave travel details with a responsible person.
- Wear a hardhat and take a gas mask.
- Beware of the sources of danger on a volcano:
  - Rock falls
  - Avalanches
  - Hazardous gases
- Look for warning signs of an eruption.
- Immediately leave the area if it becomes dangerous.
- Do not approach lava flowing through vegetation.

The references for this chapter can be found on the accompanying DVD-ROM.

16 Injury Prevention

Ariel D. Marks

The feasibility of terrestrial and underwater wilderness exploration and the medical management of related emergencies have changed dramatically as availability, performance, and distance capabilities of off-road vehicles, watercraft, and vertical-flying, long-range aircraft have allowed access to the most remote and extreme recesses of the planet's environment. Advances in mountain climbing, backpacking, and recreational scuba equipment; affordable cellular telephones; global positioning systems with satellite tracking; computer communications; infrared sensors; radio beacons; and night vision aids have made such exploration safer. Advances in water purification and disinfection technology, food processing, and preservation techniques extend the potential duration and distance of expeditions. Armed with technologic innovations, novices, children, elders, and persons with disabilities or impaired health now venture into areas once explored only by skilled and experienced wilderness enthusiasts. Accompanying increased access is a rise in the number of associated injuries and fatalities.

Some of these pursuits into the wilderness have become increasingly extreme, with participants pushing the boundaries of safety and security, making rescue concomitantly more challenging and hazardous. Delay in access and definitive treatment becomes a critical determinant in the management of wilderness injuries. A national wilderness Emergency Medical System (EMS) system does not exist, despite the fact that the practice of wilderness medicine poses unique challenges and requirements that are distinct from those encountered in the urban, and even most rural EMS systems.[15]

Imperative to the health care provider practicing in the wilderness are the physical skills to practice in a challenging environment. Providers must have the proper training and equipment to enter the chosen setting, and the mental and emotional stamina to engage in potentially long-term care of patients who have limited physical resources. Rescue of these patients will extend well beyond simple medical management. Broader-based specialties are best equipped to address the breadth of wilderness medical injuries and illnesses, but providers must often rely on the expertise of their specialized colleagues, including wilderness guides and rescue personnel proficient in nonmedical skills.

Because of the lack of diagnostic equipment, in-field practitioners of wilderness medicine must rely solely on their clinical judgment. In addition, illnesses seldom seen in the urban setting, such as acute mountain sickness, decompression sickness, and frostbite, may be encountered. Despite appropriate treatment in the field, practitioners are still left with the daunting task of coordinating evacuation to a hospital setting. Without the modern conveniences of rapid transportation and resources of traditional medicine, providers are forced to improvise and are often able to provide only suboptimal care. The absence of a mode of transport can turn a relatively minor injury such as an ankle sprain into a protracted wilderness rescue. The environment often dictates what, if any, care can be delivered. Frequently, the environment is the cause of the problem. Hazards such as exposure to high altitude, snow, rain, wind, and cold can have a profound impact on everyone involved in a wilderness medical emergency.

Evacuation of a nonambulatory casualty in the wilderness requires a tremendous number of rescuers and is a dangerous and exhausting task. Thus, the skills of injury prevention are paramount in the practice of wilderness medicine.

CAUSES OF WILDERNESS INJURIES

Contrary to popular belief and media reports, most U.S. wilderness injuries are not due to exotic causes (e.g., wild animal attacks, rock climbing, hang gliding) but occur during common events, such as hiking, swimming, walking, skiing, and driving. In one review, fighting and substance abuse accounted for more than three times as many wilderness injuries as did rock climbing,[29] and purely environment-related activity fatalities (e.g., avalanche, shark attack, volcanic fume inhalation) accounted for less than 4% of all deaths.[29] Surprisingly, suicide ranked fourth in a field of 12 activities leading to death.

In one study, researchers found that sprains, strains, and soft tissue injuries accounted for 80% of the injuries; 60% of illnesses were due to nonspecific viral illnesses or diarrhea; and 39% of injuries required evacuation. Researchers concluded that wilderness medical efforts should concentrate on wilderness hygiene and management of musculoskeletal injuries and soft tissue wounds.[18] Other studies have shown that a significant number of wilderness mortalities are related to incidents involving alcohol. A 13-year retrospective study of wilderness mortality in Pima County, Arizona, found alcohol in 50% and drugs of abuse in 12% of the deceased.[20] Drowning appears to be the overwhelming cause of death associated with recreational aquatic activities, with alcohol detected in the blood in 30% to 70% of persons who drowned while involved in these activi-

ties. Alcohol consumption significantly increases the likelihood of immersions resulting in drowning during aquatic activities.[14]

There is additional anecdotal evidence to suggest that primary efforts to reduce accidents in the wilderness should be directed toward diminishing alcohol use (Fig. 16-1).

MORBIDITY AND MORTALITY STATISTICS

Injury rates in traditional sporting activities are well studied, but wilderness-related injuries are not.[18] In the United States,

Blood Alcohol Concentration

The measure of impairment is directly related to several factors such as weight, gender, and body metabolism. Women are more susceptible to the effects of alcohol than men. The following charts are organized based on weight and gender only.

Drinks / hour	Male: Estimated Percentage of Blood Alcohol Content %BAC — Weight in Pounds								
	100	120	140	160	180	200	220	240	
1	0.04	0.03	0.03	0.02	0.02	0.02	0.02	0.02	Safe Zone
2	0.08	0.06	0.05	0.05	0.04	0.04	0.03	0.03	Impairment begins
3	0.11	0.09	0.08	0.07	0.06	0.06	0.05	0.05	Impairment zone (legally intoxicated certain states)
4	0.15	0.12	0.11	0.09	0.08	0.08	0.07	0.06	Impairment zone (legally intoxicated certain states)
5	0.19	0.16	0.13	0.12	0.11	0.09	0.09	0.08	Legally intoxicated
6	0.23	0.19	0.16	0.14	0.13	0.11	0.1	0.09	Legally intoxicated
7	0.26	0.22	0.19	0.16	0.15	0.13	0.12	0.11	Legally intoxicated
8	0.3	0.25	0.21	0.19	0.17	0.15	0.14	0.13	Legally intoxicated
9	0.34	0.28	0.24	0.21	0.19	0.17	0.15	0.14	Legally intoxicated

Drinks / hour	Female: Estimated Percentage of Blood Alcohol Content %BAC — Weight in Pounds								
	100	120	140	160	180	200	220	240	
1	0.05	0.04	0.03	0.03	0.03	0.02	0.02	0.02	Impairment begins
2	0.09	0.08	0.07	0.06	0.05	0.05	0.04	0.04	Impairment zone (legally intoxicated certain states)
3	0.14	0.11	0.1	0.09	0.08	0.07	0.06	0.06	Impairment zone (legally intoxicated certain states)
4	0.18	0.15	0.13	0.11	0.1	0.09	0.08	0.08	Legally intoxicated
5	0.23	0.19	0.16	0.14	0.13	0.11	0.1	0.09	Legally intoxicated
6	0.27	0.23	0.19	0.17	0.15	0.14	0.12	0.11	Legally intoxicated
7	0.32	0.27	0.23	0.2	0.18	0.16	0.14	0.13	Legally intoxicated
8	0.36	0.3	0.26	0.23	0.2	0.18	0.17	0.15	Legally intoxicated
9	0.41	0.34	0.29	0.26	0.23	0.2	0.19	0.17	Legally intoxicated

Figure 16-1. Alcohol consumption chart.

no general database tracks the incidence of wilderness-related morbidity and mortality. Thus, only a sample of the total number of participants and injury rates can be examined. Data reveal that more than 11 million people participated in backpacking or wilderness camping in 1990. The morbidity and mortality figures from eight national parks in California between 1993 and 1995 reported 1708 injuries and 78 fatalities.[29]

A study looking at wilderness deaths in Pima County, Arizona from 1980 to 1992 determined that 100 fatalities occurred during the 13-year study period.[20] There were 59 unintentional traumas, 18 suicides, 9 homicides, 12 medically related deaths, and 2 deaths of unknown causes. Toxicology tests performed on body fluids yielded positive findings for alcohol in a total of 50 (50%) cases and positive findings for drugs of abuse in 12 (12%) cases. It was estimated that alcohol was "a very probable" or "a probable" causative factor in 23 (40%) of the 59 unintentional trauma deaths, and in 1 (8.3%) of the 12 medically related deaths.

There are more than 9 million certified scuba (self-contained underwater breathing apparatus) divers in the United States, with approximately 650,000 annual certifications.[11] The Divers Alert Network (DAN) at Duke University Medical Center (Durham, NC) reported approximately 1000 serious diving-related injuries and approximately 90 diving-related fatalities annually between 1995 and 2002.[13]

From 1990 through 1995, an estimated 32,954 persons were involved in injuries related to personal watercraft (PWC). No estimate of total participants has been recorded.[5]

In western Washington, 40 pediatric wilderness-activity related deaths were recorded between 1987 and 1996.[33] Ninety percent of the victims were male, and 83% were 13 to 19 years of age. The most common cause of death was by drowning (55%), followed by closed head injury (26%). Injuries or deaths resulted from lack of preparation, lack of training for wilderness activities, and inadequate basic safety equipment; alcohol use and rescue delays were contributing factors. Of note, the presence of adults did not appear to be significant in reducing the incidence of mortality among the pediatric population.

INJURY PREVENTION IN MEDICAL PRACTICE

Relatively few physicians recognize injury as a disease process with predictable patterns based on patient demographics, geographic locations, and temporal variation. Moreover, most physicians do not yet embrace injury prevention as a realistic dynamic of medical practice. Traditional medical training emphasizes intervention over prevention, and it does not often approach injury as a "preventable disease."

Minimizing the risks of wilderness environments and effectively evaluating and treating illnesses and injuries in remote settings constitute the central mission of wilderness medicine. Because wilderness injuries occur in remote settings, distant from usual sources of medical care, injury control is very important. This chapter distinguishes accidents from injuries, presents the principles of wilderness injury prevention, examines equipment designed to protect or limit injury, lists injury prevention practices in wilderness settings, and identifies vehicular safety strategies.

ACCIDENTS VERSUS INJURIES

Accidents are defined as unpredictable acts of fate or chance events. Injuries are defined as corporeal damages resulting from such events.[35] Injuries are prevented by stopping or reducing the number of accidents causing them. By applying this principle, practitioners of injury prevention estimate that 90% of all injuries are predictable and preventable. For example, by inspecting, testing, and replacing rappelling gear, mountain climbers consciously attempt to prevent accidents related to equipment failure. If equipment-related accidents occur, mountain climbers then minimize injury by safeguarding their bodies through use of protective devices, such as helmets, harnesses, and ropes.

Accidents are the result of situations. Prevention of accidents requires greater focus on the situation that produced the error (e.g., driving too close to the car ahead or entering the wilderness with inadequate exposure protection). Identifying the scenarios that produced error in judgment helps avoid accidents (e.g., allowing greater braking distance or dressing to survive).

Active versus Passive Injury Prevention Strategies

Injury prevention strategies are either active or passive.[35] Active injury prevention strategies require wilderness enthusiasts to change their behaviors when exposed to risks.[44] For example, wilderness enthusiasts must be convinced that wearing seatbelts reduces the risk of injury when riding in off-road vehicles, then must take the action of buckling up every time they ride.

Passive injury prevention strategies require no action. Rollover bars on off-road vehicles are passive injury prevention devices because, unlike seatbelts, they do not rely on the beliefs or behaviors of passengers. Making off-road vehicles safer by equipping them with rollover bars has done more to decrease injuries from accidents than warnings to wear seatbelts.[36] Another example of passive injury prevention would be to select a campsite well away from steep drops or water hazards to protect children from potential dangers.[22] Passive injury prevention strategies are generally more likely to decrease injury rates than are active injury prevention strategies.[25]

PRINCIPLES OF WILDERNESS INJURY PREVENTION

Planning, preparation, and problem anticipation are the three principles of wilderness injury prevention.

Planning

Careful planning builds the foundation for a safe wilderness outing. This multifaceted task includes thoroughly researching all aspects of the wilderness activity or destination. Today, critical and up-to-date information regarding weather, trail conditions, best times to travel, permit requirements, and so forth can be easily and readily obtained. Planning includes developing a comprehensive equipment checklist, realistic activity timeline, acceptable alternative plans, and emergency procedures. A critical determinant for trip planning is weather, but even the most recent detailed forecast can be wrong, so one must be prepared for unexpected weather.

Planning includes identifying and understanding potential hazards and risks associated with a wilderness activity or des-

tination. For example, the injury most often associated with kayaking is anterior shoulder dislocation caused by the "high brace" maneuver. Knowing this, kayakers can incorporate shoulder strengthening exercises into fitness regimens and practice "low brace" maneuvers that minimize the risk of shoulder dislocation when paddling in white water.

Planning for health and safety includes establishing limitations that directly minimize any known potential for harm in specific wilderness activities. High-altitude mountain and rock climbers may plan to limit daily ascent rates, not only to allow for acclimatization but to avoid headaches, fatigue, and dizziness, all symptoms of acute mountain sickness.[21]

Preparation

When traveling, many people focus on the logistical aspects of a trip, taking health for granted and assuming standards of health and hygiene similar to those at home. They should be prepared for the worst possible eventuality and expect poor hygiene practices and substandard medical services when traveling to remote locations.

Physical Preparation. Physical preparation includes maintaining excellent fitness and health in anticipation of the added stress of outdoor activities. Because wilderness activities take place in remote settings, distant from usual sources of medical care, a much higher level of medical self-sufficiency is required. Reading a book is a far cry from actually performing these physical functions under duress and challenging environmental conditions, so it is a good idea to rehearse hands-on first-aid procedures.

Mental Preparation. Mental preparation emphasizes being confident about the planned trip. Confidence is based on knowing that one has the skills and experience necessary to handle the situation and not exceed limits. Possible evacuation routes and emergency procedures should be established, and one must be aware of each participant's level of comfort with self-treatment and specific abilities. Determine the location of and distance to known medical care facilities. Notify a friend or neighbor of departure date, planned length of stay, and anticipated return date and time. Leave a face-down note of your planned excursion on the front seat of the vehicle so that authorities can find it. Plan each trip, establish priorities, and carry out the plan. Be prepared to take charge in the event of an emergency situation.

Material Preparation. Material preparation involves acquiring proper equipment, testing and organizing equipment, and having backup equipment should something fail. Have sufficient equipment to survive the night out under the worst possible environmental conditions for that time of the year.

The wilderness explorer must have all equipment maintained regularly and inspected carefully before a trip. Have emergency bivouac equipment. Carry equipment (in excess of personal needs) that could be given to a person overcome by sudden illness or injury.

If traveling with partners, review each person's responsibility. Try to "streamline" equipment so that it does not get caught on things, will be easier to handle, and will be maneuverable when traveling under windy conditions or in water.

Use battery-powered items that require the same size and type of batteries. Keep a gear checklist to avoid forgetting essential items. Depending on your wilderness activity, essential equipment may include an avalanche beacon, compass, duct tape, emergency blankets, emergency bag, extra clothing, extra food, firestarter, first-aid kit, flashlight, personal locator beacons, rescue lasers, ground insulation, knife, maps, matches, sunglasses, sunscreen, shelter (e.g., emergency tube tent), survival whistle, water, water disinfection system, and timepiece. If the risk of human immunodeficiency virus (HIV) or hepatitis B is great, carry a small kit containing sterile needles, syringes, intravenous catheters, and supplies for suturing.

Having adequate light in the wilderness setting is essential. Headlamps are particularly useful because they free up the hands. Light-emitting diode (LED) flashlights are small and lightweight, and have extended burn times that are far superior to those of conventional flashlights.

Problem Anticipation

Discuss and agree on a travel route. Choose a leader, and review each member's capabilities and what equipment and supplies are necessary. Bring adequate protection from environmental and recreational hazards. Be sure to have all the appropriate immunizations. Maintain safe drinking and food hygiene practices. Make sure everyone has a working knowledge of first aid relevant to the environment at hand.

If using specialized equipment, take a course in its proper use and maintenance. Specialty courses available through retail outlets, clubs, and wilderness organizations prepare and train participants for most outdoor activities. For example, a course in avalanche safety is essential if one plans to travel through avalanche areas.

INJURY PREVENTION

Scuba Diving and Snorkeling

DAN reported 1063 cases of decompression illness treated in hyperbaric chambers and 83 recreational dive fatalities in 2002.[13] Other data collected by DAN reveal approximately 1000 serious scuba diving accidents per year, of which approximately 10% are fatal.

Common causes of injury while diving or snorkeling include drowning, barotrauma, environmental exposure, bites, and stings. Unique to diving are arterial gas embolism, decompression sickness, and nitrogen narcosis.

Preventing drowning includes always wearing a buoyancy control device and a quick-release weight belt, as well as not diving or snorkeling under hazardous weather conditions. Diving with a buddy ensures that one will have someone to assist if a muscle cramp, exhaustion, entanglement, or other adverse situation occurs. Carrying an underwater knife helps in case of entanglement in monofilament fishing line or seaweed. Carrying a whistle, beacon, or other attention-getting device, such as an "emergency sausage" (a flexible plastic tube 1.8 m [6 feet] long that can be filled with air), visible or audible from a great distance, provides a help signal if ocean currents carry a person far from the point of entry. Newer laser devices encased in waterproof housings have been found to be very effective in identifying people during aerial searches. Commercially available "rescue laser flares" are small, lightweight, rugged, handheld laser devices with a wide-focused beam. They

Figure 16-2. The Rescue Laser Flare Magnum. (© Greatland Laser, Anchorage, AK, www.greatlandlaser.com.)

allow the user to sweep across an area with a dispersed red light beam that can be seen from 32 km (20 miles) away. The device is powered by two 1.5-V batteries that when used continuously will last for 15 hours[30] (Fig. 16-2).

Personal locator beacons (PLBs) transmitting on 121.5 MHz have had a high false-alarm rate and are of limited benefit because rescues have been delayed until legitimacy of the signal could be determined. Beacons transmitting on 406 MHz are available to the marine community and more useful because the owner's name is registered at the time of purchase. When activated, the 406-MHz beacon transmits a digital signal with a unique identification code that instantly identifies the owner, and rescuers can be dispatched. As of July 1, 2003 it is legal to purchase these PLBs in the United States for land use.

Because of higher conductive heat loss in water, divers and snorkelers should wear insulating wet suits or dry suits. This outerwear also protects against scrapes, bites, and stings. Booties can protect the feet when walking along a rocky beach while carrying heavy scuba gear. When immersed for any length of time, skin of the hands and feet becomes soft and more prone to injury. Gloves and booties guard these areas from cuts caused by rock or coral.

When in an underwater environment, pay attention to marine life that bites or stings. Avoid direct interactions with marine organisms because their aggressive behavior is usually made in self-defense.

Decompression sickness is prevented by following dive table guidelines to limit accumulation of excessive tissue nitrogen. Divers should attend a training certification course to understand how dive tables operate.

Preventing arterial gas embolism requires that divers breathe continuously when using compressed air. Persons with certain pulmonary contraindications, such as severe asthma, are generally advised not to dive.

Nitrogen narcosis impairs judgment and can result in disorientation and drowning. Usually occurring at depths exceeding 30 m (100 feet) of salt water, nitrogen narcosis is rapidly reversible by swimming into shallower water. To prevent nitrogen narcosis, avoid diving to depths exceeding 30 m (100 feet).

The most common anatomic site of barotrauma is the tympanic membrane external to the middle ear space; barotrauma results from inadequate equalization of pressure during descent. Preventing barotrauma is achieved by "equalizing" properly.

Injury prevention behaviors for diving include the following:

1. Breathe continuously. Never hold your breath.
2. Clear your ears. Do not dive if sinuses are congested. Do not dive if there is a medical contraindication to diving.
3. Know how to use and follow the dive tables.
4. Dive with a buddy. Review underwater and topside communications.
5. Inspect all dive gear regularly.
6. Know training and physical limitations.
7. Procure air refills from a reputable air station.
8. Get a predive orientation. Learn about the environment and be prepared to abort a dive if conditions are hazardous.
9. Do not use drugs or alcohol before, during, or immediately after diving.
10. Leave a dive plan with a friend or neighbor so that rescue procedures can be initiated if you do not return when expected.

Kayaking, Canoeing, and Rafting

Kayaking, canoeing, and rafting have together become the third largest outdoor recreational industry in the United States,[19] with 76 million people participating in these activities. New equipment designs, such as self-bailing inflatable rafts and lightweight, plastic polymer kayaks, have opened up more remote and difficult waterways for exploration and commercial recreation. The American Canoe Association reports approximately 130 white-water fatalities each year. The most common contributing factor is failure to wear a personal flotation device (PFD).[43]

Exposure to cold water stimulates cardiorespiratory reflexes that make it difficult for submersed swimmers to keep their heads above water.[26] The preferred type of PFD, therefore, when participating in kayaking, canoeing, or rafting, is a vest-type jacket (Coast Guard Type III PFD) that provides heads-up buoyancy. A PFD should fit snugly and not ride up over the head when the user is in the water. Some PFDs include crotch straps as an added safety feature.

A PFD also provides some protection from hypothermia. Closed-cell foam flotation material protects the thorax from impact injuries during falls on slippery rocks or when swimming rapids. A PFD can also be used as an improvised splint.

Helmets should also be worn by all white-water enthusiasts to protect against head trauma after capsizing. From 10% to 17% of all kayaking accidents involve head injury.[27,42] White-water rafting enthusiasts have used polypropylene helmets similar to those used by bicyclists, with foam liners, nylon chin straps, and drainage holes. The helmet should have a hard outer shell to protect the skull from impact with sharp rocks. Ears should always be covered and protected. Portholes allow rapid exit of water from the inside of the helmet and prevent the

helmet from becoming waterlogged, which would add to drag when submerged. The chin strap should be secure and waterproof so that it does not stretch when wet. Brightly colored, highly visible helmets aid in locating an injured individual in the water.

An athlete is 60 times more likely to sustain a dental injury when not wearing a mouth guard.[32] Mouth guards shield the teeth and protect the lips, cheek, tongue, mandible, neck, temporomandibular joint, and brain.[45] The three types of mouth guards currently available are stock, mouth-formed, and custom models, in order of increasing cost and protective benefits. The American Dental Association "recognizes the preventive value of orofacial protectors and endorses the use of orofacial protectors by all participants in recreational sport activity." This endorsement includes recommendations for water sports, such as water skiing, surfing, and water polo.[1,19] In the future, this recommendation should be extended to include high-velocity water sports, such as operating a PWC.

Rope should be readily accessible and secured so that it can be rapidly deployed without entanglement. The type of rope used should not absorb water and should allow slight stretch. Rope can be used to rescue a paddler who has fallen out of a vessel, or can be thrown to a paddler who is pinned or broached.

Injury relating to kayaking, canoeing, and rafting commonly occurs when portaging vessels. If proper lifting techniques are not used, portaging a kayak or canoe can cause back strain. Careful attention to using leg muscles rather than those of the lower back can prevent lower back injury. Injuries resulting from poor conditioning, improper stroke technique, or muscle overuse, include shoulder dislocation, low back strain, tendinitis in the wrist, extensor tenosynovitis of the forearm, muscle strains, and sprains. Prevention and correction involve maintaining physical fitness, engaging in a conditioning program, relaxing the grip on the oar, and taping the wrist in a neutral position. Blisters are common and can be prevented by wearing properly fitted gloves. Warm-up exercises, including stretching, are beneficial.

All paddlers should watch where they step to avoid ankle sprains and fractures, especially when stepping out of vessels onto wet rocks. Reef walking shoes or neoprene booties with antiskid soles are helpful.

Enthusiasts should apply sunscreen often, carry a first-aid kit, and avoid drinking river water and eating unrecognizable plants. They should watch the weather and modify activities accordingly. If paddling in cold water, paddlers should dress appropriately, wearing wet suits, gloves, booties, and woolen pullover hats to avoid hypothermia in case of capsizing. They should also avoid ingesting alcohol or illicit drugs because these intoxicants impair judgment, balance, and heat production. Paddlers should apply insect repellent and keep tetanus immunization current.

Backpacking and Hiking

A common injury that occurs during hiking and backpacking is low back pain. It is frequently caused by carrying excessive or poorly distributed loads. Several improvements in backpack design have been developed to prevent low back injury.

The two basic types of backpacks are those with external frames and those with internal frames. The frames are designed to transfer the weight of the backpack from the shoulders and back to the hips and legs. An ideal backpack weight distribution is 20% on the shoulders and 80% on the hips. This distribution lowers the body's center of gravity, making it more stable (Fig. 16-3).

Figure 16-3. To prevent injuries, a backpacker should limit the contents of a backpack to 25% of body weight. (Courtesy Jandd Mountaineering, Vista, CA.)

Backpacks with External Frames. An external frame pack allows a larger amount of weight to be carried, using a ladder-like frame made of aluminum or plastic. A hip belt and shoulder strap are attached to the frame, usually with clevis pins and split rings. Some backpacks are adjustable to fit the length of the spine. Hikers should look for lumbar padding, a conical hip belt, recurved shoulder pads/straps, and a chest compression strap that improves distribution of weight and increases comfort.

A backpack with an external frame allows air space between the back and the pack, reducing sweating. With an external frame pack, the weight is carried higher in the pack, allowing for more upright posture. However, the pack frequently wobbles side to side during walking.

Backpacks with Internal Frames. The internal frame backpack conforms more to the body, allowing for better balance, and can be worn comfortably for longer periods of time. However, back perspiration can be a problem. Because the bulk of the weight is carried lower, one must bend more, which in turn alters proper posture and can predispose to low back strain. Compression straps can compact the contents of the pack, keeping it more stable.

Backpack Considerations. Backpack size is an important consideration for injury prevention. Hikers need to be sure that all equipment necessary for the duration of the trip can be carried without putting excessive strain on the shoulders and back

muscles. The amount of weight that can be carried in a backpack is determined by the size, body weight, and fitness level of the backpacker. For multiday trips, one should attempt to limit the contents to 25% of body weight. When loading the backpack, 50% of the weight should be in the upper third of the pack. To accomplish this distribution, lighter and bulkier items should be packed in the bottom and heavier items should be packed in the top. The heaviest items should be packed at the top, closest to the frame.

Backpack Lifting. The least injurious method for donning a backpack is to have someone hold and stabilize the pack while the carrier slips his or her arms into the shoulder straps. If a second person is not available, the backpacker can lift and rest the backpack on an object that is waist-high, and then slip into it.

If a backpack must be donned from the ground, it is not recommended that the carrier sit down, slip into the backpack, and stand up. Rather, he or she should lift the backpack onto a bent knee and slide one arm through the shoulder strap. After adjusting the strap so that the backpack rests on the shoulder, the carrier should lean forward and rotate the body slightly, allowing the free arm to slide through the other shoulder strap. While still leaning forward with bent knees, the backpacker adjusts the second strap and hip belt.

To prevent low back strain, a backpacker should use the knees rather than the lower back to bend. Good abdominal muscle tone helps limit back strain.

Backpacks and Children. Backpacks that carry small children can be dangerous. Risks include strangulation if a child becomes entangled in the carrier's harness, and head and body injury if a child wiggles out of the harness and falls or is struck by an overhead obstruction, such as a tree branch. Be certain to use a well-structured carrier with a design that restrains the ambitious, mobile child.

Hiking Footwear. Blisters are a common backpacking and hiking injury, but careful selection of shoes and socks limits this problem. The purposes of footwear are protection, cushioning, support, and grip. Selecting, fitting, breaking in, and caring for footwear help it last a long time and maximizes comfort. Size footwear in the afternoon because feet swell during the day. Also, try footwear on both feet before purchase. If one foot is larger than the other, select the size in which the larger foot has the best fit.

Size feet using a Brannock device, which measures not only the length and width of a foot but the ball-to-heel (arch) length. When selecting footwear, check the length first. If toes touch the end, leaving no space for the heel, the shoe is too small. When you slide the foot forward into an unlaced shoe or boot, you should be able to insert a finger between the footwear and the heel. If this is not possible, the footwear is too short and will bruise toes, especially on long downhill walks. When hiking, wear shoes that extend above the ankle to reduce the likelihood of an ankle sprain.

Footwear should fit comfortably with moderate tension on the laces so that they can be tightened or loosened as needed. To avoid blister formation, seams should not rub against any part of the foot. The tongue of the footwear should be aligned and laced properly; otherwise the tongue can slide into an incorrect position and cause blisters. Ankles should be comfortably supported by stiff heel counters or heel cups and should not slip with toe flexion, causing blisters because of repetitive rubbing. With the foot on the ground, there should be no more than 6 to 12 mm of heel lift.

The soles are protected with layers of cushioning thick enough to prevent bruising but pliable enough to allow natural heel-to-toe flexion. Thick soles insulate against cold and heat. The tread provides grip. Support comes from a fit that stops the foot from slipping inside the shoe but that is not so tight that it prevents the foot from expanding when it swells.

Socks. Many boots feature sock liners or booties made from vapor-permeable membranes, such as Gore-Tex and Sympatex, hung between the lining and outer aspect of the boot. These make boots waterproof when they are new, but most boots leak after a few weeks in wet weather. In addition, lack of breathability means that water vapor cannot penetrate the barrier, resulting in hot, sweaty feet. Socks cushion the feet, preventing abrasion. They also wick away moisture, keeping the feet at the right temperature and humidity. Newer synthetic materials, such as polypropylene, Capilene, and Thermax, wick away moisture quickly, making them good choices for an inner layer.[17] Pile or fleece socks do not wick away moisture very well.

Wool offers many advantages. It is warm in winter, cool in summer, absorbs and wicks away sweat, and keeps the feet warm when wet. Furthermore, during long trips, it rinses well in cold stream water, can be worn for days at a time without wear, and does not mat down like cotton socks with terry loop liners.

It is worth the time to check socks for loose threads, knots, or harsh stitching that might cause blisters or sore spots. Flat seams at the toes are important. Bulky seams rub and cause blisters. Consider wearing one pair of fairly heavy socks (rag wool) and one pair of light liner socks next to the skin. With one sock, the boot and the outer sock tend to move as a unit. They rub against the heel and the top of the toes at the metatarsal heads. A lightweight liner sock tends to cling to the foot. As the boot moves, the socks rub against each other, not against foot skin. Consider using powder (Fig. 16-4) to keep the feet dry.

Figure 16-4. Foot powder is applied to avoid moisture and skin maceration.

Figure 16-5. Blist-O-Ban bandage (SAM Medical Products) applied to prevent a heel blister.

Figure 16-6. Blist-O-Ban (SAM Medical Products) bandage applied to a bunion to prevent blister formation.

Figure 16-7. Sun protection for the eyes is critical in outdoor activities to prevent premature cataracts, photokeratitis, and other ocular injuries. (Courtesy Black Diamond Equipment, Ltd., Salt Lake City, UT.)

Foot Hygiene. Keep toenails short and cut them square. Keep feet dry to avoid skin softening. Immediately stop walking and attend to the first sign of a sore or "hot spot" to prevent further injury. Protect any reddened areas with moleskin, tape, or molefoam. Be sure that the covering extends beyond the reddened area. An excellent new product for blister prevention is the Blist-O-Ban bandage (SAM Medical Products, Newport, OR), which is an artificial blister applied to a hot spot before true blister formation occurs (Figs. 16-5 and 16-6).

Avoid getting blisters. Once formed, they must be protected to prevent rupture and infection. If a blister has already formed, relieve external pressure by applying a doughnut-shaped piece of molefoam. A blister should not be unroofed unless absolutely necessary. If this is required, wash the area with soap and water and insert a decontaminated needle ("sterilized" by a flame or with rubbing alcohol) into the edge of the blister. Gently press out the fluid. Apply a sterile dressing. If the blister has already broken, cleanse and cover the area. Topical antiseptic ointment may be beneficial.

Exposure. Overexposure caused by inadequate skin covering results in several problems. An essential area to protect is the head. From preventing heat loss to blocking ultraviolet (UV) exposure, hats are an integral component of the wilderness wardrobe. The uncovered head is a source of considerable heat loss because it can dissipate up to 70% of total body heat production.[4] Because there is no substantial decrease in blood flow to the head in cold conditions, hats should be worn to stay warm. Hats large enough to cover the ears, face masks, and neck warmers (gaiters) are popular among skiers. The preferred type of hat for wilderness expeditions is the stocking type.

In warm climates, hats with visors and brims provide considerable protection from UV exposure to the face, including the nose, and ears. Hats also provide some degree of insect protection, especially if sprayed with insect repellent, and reduce radiant heat exposure. They repel light rain, keep leaves and twigs out of the hair, and hold a mosquito net in place. In addition, when worn in the jungle where larger arboreal animals reside, hats can protect the head and hair from airborne droppings. Hats should be lightweight, light-colored, and preferably vented. It is helpful to have a retention neck or chin strap for windy days and to have snaps to fasten the brim up when traveling on a trail. Hats do not need to be waterproof. They should be loose-fitting.

Eye protection is also essential. Sunglasses provide protection against injury caused by UV exposure, which can contribute to ocular complications, including photokeratitis (snowblindness), cataracts, pterygium, and macular degeneration (Fig. 16-7).

Major variables to consider in selecting sunglasses are light transmission, coloration, polarization, and lens construction. Lenses for wilderness exposure should absorb 99% of UV radiation. If used in high solar reflection areas, lenses should also absorb 85% to 95% of visible light.

The preferred lens is made of glass or polycarbonate. Polycarbonate lenses are lightweight, scratch resistant, and shatterproof (a major consideration if impact is possible). Plastic lenses scratch easily and are generally not recommended.

When wearing dark lenses in a bright environment, the pupils dilate and allow more harmful radiation exposure to the retina. Photochromic lenses, which darken with increasing sun intensity, are potentially dangerous when driving. If fully darkened in bright sunlight, photochromic lenses limit vision considerably when traveling through a tunnel. It is preferable to have a less dark pair of sunglasses for driving. Amber lenses or rose lenses enhance contrast. Polarization is particularly useful in reducing reflective solar glare from water, snow, and ice. UV-absorbing contact lenses do not provide adequate protection from UV radiation and should not be used as substitutes for sunglasses. Sunglasses are needed to cover the entire eye area, including eyelids.

Choose sunglasses that are close-fitting and have relatively large lenses, or are of the wraparound variety, to prevent UV rays from entering from the side. Children, including infants, should wear sunglasses. Glasses should be secured with some type of strap. Commercial lens "defoggers" are available, although any surfactant, such as soap, rubbed or washed onto lenses will have the desired defogging effect.

In severe blizzard conditions, goggles improve visibility. The goggles' foam mesh vents help reduce fogging. A wide elasticized headband helps keep them in place. For protection from impact and foreign bodies, safety glasses or goggles should be worn when traveling on bicycles, horses, or any off-road motorized vehicle without a windshield.

Hiking Accessories. A useful injury prevention adjunct is a hiking pole or staff to provide stability on rough ground and diminish impact on knees and ankles. A staff can support a good walking rhythm and prevent imbalance when carrying a heavy load. Hiking poles allow for probing and identifying hidden rocks and deep spots and can hold back bushes, barbed wire, stinging plants, and other trail obstructions. They can even be used to fend off aggressive dogs or wildlife.

Using two staffs or poles takes the load off the legs and hips and redistributes part of the weight to the upper body musculature. On a steep area, hiking poles enable tripoding, or having three points of contact with the ground.

Injury Prevention for the Genitourinary Tract. To prevent genitourinary infections during wilderness outings, enthusiasts should maintain fastidious genital hygiene and ensure adequate hydration. To counteract the tendency to bathe less frequently during an extended outing, a shower should be part of the daily schedule (Fig. 16-8).

Infections and irritation of the genitourinary tract are more common in women than in men because the female urethra is shorter, leaving the bladder more predisposed to colonization by bacteria. Infrequent urination and urethral trauma from vigorous activity or bruising from a bike seat, saddle, or climbing harness are common contributing factors to urinary tract infections.

Vaginitis. Vaginitis can occur with increased stress, sweating, or strenuous and vigorous activities, as well as with changes in the environment, including heat and humidity. To avoid vaginitis, women should keep themselves cool and dry by wearing loose-fitting clothing that maximizes air circulation and minimizes the optimal conditions for bacterial and fungal growth. Avoid nylon undergarments because they retain humidity. Cotton underwear is preferable.

Figure 16-8. Several new and effective methods of transporting water into the wilderness have been developed. The biking hydration system shown here can help prevent urinary tract infection by facilitating the opportunity for adequate hydration. (Courtesy Jandd Mountaineering.)

Sanitary Protection for Menstruation. If menstruating while in the wilderness, women should change their tampons or sanitary napkins just as frequently as at home to avoid infection or toxic shock syndrome. Women who use tampons should make sure their hands are scrupulously clean before insertion. For environmental reasons, tampons with cardboard rather than plastic applicators should be used if wilderness disposal is a possibility.

Perianal Hygiene. Whenever possible, cleanse the anal area with fresh water. If pruritus ani develops, medicated wipes, such as witch hazel tucks or moisture-blocking ointments, have been shown to be beneficial for some individuals. "Porta-showers" or "sunshowers" are particularly helpful to maintain good anal hygiene. Baby wipes are often quite soothing.

For wilderness excursions, biodegradable toilet paper is recommended. Select toilet paper that is dye and perfume free. Use double zip-lock bags for packing to keep the toilet paper dry and clean. Burning toilet paper creates a fire hazard, so, ideally, it should be packed out. If buried, it should be placed 6 inches from the ground surface and at least 60 m (~200 feet) from any natural water supply. Toilet paper decomposes more rapidly if wet and not buried too deeply.

Trail Safety. Finding one's way in the wilderness, sometimes in unfavorable and extreme weather conditions; negotiating cliffs, loose scree, uneven and hazardous terrain, including snow, ice, and mud; and facing other hazards, such as wild animals, can be challenging. Do not assume that because a trail is marked that it is safe, clear, and well maintained. Take a switchback route across slopes when possible. Use rest areas.

Balance is key to crossing rough terrain. Watch for unstable rocks and boulders. Keep body weight over feet, with knees bent, and do not lean backward. When traveling along scree, do not have anybody traveling directly below or above you because stones can dislodge and fall. If this occurs, shout a warning, but the person below should not look up. For rock climbers, do not climb what you cannot descend.

At altitude, strong winds can make walking impossible. Be prepared to descend early or take an alternate route if weather worsens. It may be necessary to "sit out" bad weather.

When fording rivers and streams, remember that water is very powerful. The use of safety ropes is recommended.

Hammock Safety. Small, lightweight minihammocks are popular in camping and backpacking. These hammocks have no spreader bars and can be folded easily. The U.S. Consumer Product Safety Commission (CPSC) warns that children can become trapped and strangle on certain minihammocks; two deaths and one nonfatal incident have involved minihammocks or backpacker hammocks. If a hammock is rigged too high off the ground, a child will have difficulty climbing into it and may become trapped or entangled, leading to strangulation. To prevent injuries, install a minihammock near the ground.[40]

Maps. Knowing where you are is a key wilderness skill. Carry maps and know how to read them. Always establish entry, check, and exit points on your maps. Even on marked trails, an inadvertent turn can rapidly create confusion and allow you to become lost.

Two types of maps are available—planimetric (two-dimensional) and topographic (three-dimensional). Planimetric maps identify features on the ground. Topographic maps provide information about the shape of the ground itself. Topographic maps use contour lines to identify points of equal elevation. The standard U.S. Geological Survey 1:24,000 scale maps have contour intervals of 40 feet (12 m). More recent Bureau of Land Management maps use a metric 50-m (166-foot) interval. The closer together the contour lines, the steeper the slope.

Global Positioning Devices. The global positioning system (GPS; see Chapter 85) is a government-operated system consisting of approximately 28 satellites at a 12,000-mile (19,200-km) altitude. Each satellite orbits the earth twice a day, emitting continuous signals of time and position. By carrying a GPS receiver on the ground, position can be triangulated.

Line of sight is required between the receiver and satellite. Therefore, position determination may be impossible in dense forests and jungles, or below steep cliffs. Furthermore, electronic GPS receivers are sensitive to mishandling, extremes of temperature, and battery failure. They should be an addition to, and not a replacement for, traditional maps and compasses. Natural references, such as sun position, wind, and topography, can provide useful clues to position.

Cellular Telephones. Cellular telephones can provide ready access to the outside world, as well as an additional form of security when traveling in remote locations. They are sensitive to mishandling and have limited battery life. Because signal transmission relay stations are infrequent in the wilderness, there is a real chance of telephone failure.

If emergency assistance is needed during a wilderness activity, dialing 911 can connect one to a service that will assist with wilderness rescue. Calling the local National Park Service office, a search and rescue organization, or even the local sheriff's office may result in an earlier and more efficient rescue response.

For the past few years in Grand Teton National Park and Teton County, approximately 70% of initial mountaineering and recreational accident reports have been received by cell phone.[24] This trend has had a significant impact on rescue response. Rescuers are able to obtain an accurate, on-site victim report with information on location and the mechanism of injury. This helps establish an "urgency factor." Continued communication with the reporting party can guide rescuers to the viction and ensure arrival of appropriately equipped medical personnel. In addition, contact with the victim permits first-aid advice to be offered by dispatch before the arrival of rescue teams.

Other Emergency Forms of Communication. The international distress signal (SOS) consists of three short signals, followed by three long signals, and then three short signals, repeated at intervals. This signal can be transmitted with light, sound, or Morse code. Ground-to-air signals can be used with any material visible from the air.

Climbing

Common climbing injuries involve falls, often from great heights. One of the most critical pieces of safety equipment is a helmet because the head is a major area of potential injury.

Helmet Composition and Fit. The purpose of a helmet is to absorb the energy of an impact, minimizing or preventing head injury. Usually, crushable expanded polystyrene foam is used for this purpose. Most helmets now have a hard outer shell to protect the head in a collision with a sharp object. It is prudent to wear a hard-shelled helmet when climbing because falling rocks can cause significant damage. It is also beneficial to have a visor attached to the helmet to protect the face and eyes.

Helmets should have a snug, but comfortable, fit. Some helmets have varying thicknesses of internal padding to custom fit the helmet to the user. Ideally, helmets should be lightweight and have a buckle that stays securely fastened. No combination of twisting or pulling should remove the helmet from the head or loosen the buckle on the strap (Fig. 16-9).

Additional guidelines for effective helmet wearing practices include the following:

1. Wear the helmet flat atop the head, not tilted back at an angle.
2. Make sure the helmet fits snugly and does not obstruct the field of vision.
3. Make sure the chin strap fits securely and the buckle stays fastened.

Climbing Equipment. To prevent trauma to the torso when climbing, a harness should be worn. Harnesses secure the wearer's center of gravity to a safety line. If footing is lost, free-fall injuries are abated by the harness and safety line. To be effective, a harness must fit securely and be made of strong material with reinforced stitching. It should be inspected regularly for wear.

Figure 16-9. Helmets should be worn by all wilderness enthusiasts involved in activities that might result in head injury. (Courtesy Black Diamond Equipment, Ltd.)

A useful accessory if climbing in avalanche country is an "AvaLung." The AvaLung is worn like a lifejacket and can help keep a person alive in an avalanche (see Chapter 2). A victim buried in snow can breathe for approximately 10 minutes without and up to 1 hour with the AvaLung vest. The additional time is gained by increasing the surface area for air exchange, using the vest as a "breathing membrane" (see Chapter 81, Figure 81-3).

Knee guards, elbow guards, and wrist guards are useful protective devices when traveling with heavy packs or on slippery surfaces. Crampons are metal devices that attach to the outside bottom of footwear to provide traction on ice. Although crampons allow crossing of ice and hard snow without slipping, walking with them involves a change in gait. Because they have sharp metal serrated edges, stress injury to the medial aspect of the knee can occur unless a wider gait is maintained during walking.

Altitude. Along with the limitation of oxygen because of reduced partial pressures, a major consideration at altitude is hypothermia. Hypothermia is prevented through proper exposure protection and common sense. Potential hypothermic situations must be managed before they become serious (see Chapter 5).

Recognize the environmental conditions that lead to hypothermia. Wear appropriate clothing for cold and wet conditions and always wear a hat. Wear polypropylene underclothing to wick water away from the skin. Wear multiple layers, including, but not limited to, wool and a windproof and waterproof covering.

Physical fitness, continuous exercise, high-calorie food intake, and consumption of hot beverages will prevent onset of mild hypothermia. Avoid exhaustion, panic, and energy-depleting activities. Carry fire-starting materials and materials for an improvised shelter. Pack a VHF radio and know how to use it. Learn to recognize warning signs of hypothermia.

An ice ax is imperative for traversing snow and ice at altitude. A walking staff or ski pole is entirely inadequate for crossing hard-packed snow or ice. On these surfaces, a slip can quickly become an out-of-control fall. Limiting this fall (self-arrest) involves proper use of an ice ax, which can also be used to cut steps into ice.

Boating

The single most useful injury prevention device for boating is the PFD[3] (see Chapter 76). A PFD or lifejacket should be used when participating in any water-related activity, such as canoeing, kayaking, or jet skiing, but it is not a replacement for knowing how to swim. Brightly colored so that it can be seen at a distance, the PFD is designed to provide the wearer a stable, face-up position in calm water when the head is tilted back. However, most PFDs do not hold up the face of an unconscious wearer, although some newer models ensure face-up flotation even with an unconscious user. In rough water, the wearer's face may often be covered by waves because the PFD is not designed for extended survival in rough water. The styles that ensure face-up flotation and are designed for the body habitus and weight of the user are safest.

Personal flotation devices require routine minor maintenance. Check the PFD frequently for rips, tears, and holes. Confirm that the seams, fabric, straps, and hardware are in good working order. If caught in riptides, undertows, or strong currents, ride the current out and work your way back to the shore in the area adjacent to the current where the water is calmer.

Personal Watercraft

The use of PWC, such as jet skis or waverunners, in water recreation has increased rapidly since 1990. Over 1 million PWC were operated in 1999.[37] PWC are less than 4 m (13 feet) in length and designed to be operated by persons sitting, standing, or kneeling on the craft, instead of within the confines of the hull.[39] These watercraft allow swifter movement and attain faster speeds in less time than do larger, motor-driven boats.

From 1990 through 1995, there were an estimated 33,000 injuries associated with PWC use. Injury rates increased fourfold from an estimated 2860 in 1990 to more than 12,000 in 1995. Injuries and deaths to both the watercraft drivers and bystanders occurred disproportionately among the very young or intoxicated.[23] Almost 86% of injured persons were 15 to 44 years of age. The median age was 25 years. Of injured persons, 71% were male. An estimated 7% of the persons injured in the cited report were 14 years of age or younger. The rate of injuries related to PWC treated in emergency departments was about 8.5 times higher than the rate of emergency department–treated injuries from motorboats. In California, PWC account for only 16% of all vessels registered in that state. However, according to the Department of Boating and Waterways statistics in 1997, PWC were involved in 45% of all accidents, 55% of injuries, 14% of fatalities, and 23% of property damage.[12]

Kinetic energy is the pathogen of PWC injuries.[36] Increased speed equals increased crash fatality risk and large increases in death tolls.[34]

Given the high speeds that can be achieved with PWC, training requirements and enforcement may reduce the number of injuries considerably. Parental or adult supervision is recommended for minor children. Right-of-way guidelines currently in place for boat operators, including maintaining safe distances and reduced speeds, should be extended to PWC users. PWC

should not be used where others are swimming or wading. PWC offer no protection from impact to the driver or the bystander. No federal laws govern the safe conduct of these vehicles, and many injuries involve swimmers and other unprotected water enthusiasts. The impact of a PWC on a swimmer can be equivalent to a pedestrian being hit by a small truck at high speed.

Hunting

More than 35,000 firearm fatalities have occurred in the United States each year since 1989, and it is estimated that there are 3 nonfatal firearm injuries for each death associated with a firearm[2] (see Chapter 22). Hunter safety courses are available in every state and are a prerequisite in most states to obtaining a hunting license. Key recommendations include warning nonhunters of hunting season, limiting hunting to hunting areas, and wearing international orange clothing articles when traveling in areas frequented by hunters. Hunters should always be sure of their target before shooting. A safety harness should be worn when using a tree stand. Care should be taken to use appropriate techniques when cleaning game. Eye protection should always be worn with impact-resistant lenses to prevent injury from ricocheting fragments and shotgun pellets. Ammunition should always be stored separately from guns.

High-frequency hearing loss is common among hunters because of the loud report of the firearm. Earplugs and headsets, although impractical for most hunting situations, can provide good protection for the ears. In most scenarios, a single earplug for the ear closest to the muzzle will protect the ear most likely to be injured.

The greatest potential serious injury from hunting is a gunshot wound. Every effort should be made to ensure that inadvertent discharges do not occur. Most injuries related to firearms occur during common gun-related activities, such as cleaning and inspection, and are therefore self-inflicted. Further studies are needed to evaluate the efficacy of gun safety training courses and to assess the potential role of various gun safety devices, such as trigger locks and loading indicators.

Vehicular Safety

Off-road travel is challenging for both vehicle and driver. Off-road path conditions are usually poor, with deep potholes, large boulders, and mud that limit traction. Because of a lack of clearing and the absence of street lighting, visibility is usually low. There is also the chance of encountering wildlife, which may jump out in front of the vehicle.

Off-road four-wheel-drive vehicles have high centers of gravity to allow for ground clearance so that boulders and rocks do not damage the vehicle's underside. This means that the vehicles are top-heavy and prone to tipping, especially when turning corners at high speeds. Care should be taken to travel slowly.

Because most four-wheel-drive vehicles have stiff suspensions (for better maneuverability), always wear a three-point harness and be cognizant of bumping and jarring that can injure the spine. Secure all equipment.

Many off-road vehicles have no air bags or cabs, and many have only have lap-belt restraint systems. They often have thin-walled bodies and frames and are not well designed for impact.

Rollover protective structures (ROPS) are essential. These can be enclosed in the cab or unenclosed (resembling one or more exposed rollover bars). ROPS are structural components attached to vehicles designed to protect the operator from being crushed if the vehicle overturns. Although ROPS offer some degree of protection, passengers must be securely fastened with seat belts so they are not ejected from their seats if the vehicle rolls over. Passengers must remain confined within the space protected by the ROPS.

All-Terrain Vehicles. All-terrain vehicles (ATVs) are motorized recreational cycles with four (and sometimes three) large, soft tires. These vehicles are designed for off-the-road use on various terrains.

All-terrain vehicles have become enormously popular, particularly among the young, with approximately 2.5 million vehicles in use. Although ATVs give the appearance of stability, the three-wheeled design is unstable, especially on hard surfaces. Stability is further compromised by a high center of gravity. The hazard is compounded by the fact that ATVs can achieve speeds up to 50 to 80 km per hour (30 to 50 miles per hour). Moreover, their use by youths has sometimes been promoted.[36] Recent studies by the CPSC showed that the risk of injury is 2.5 times higher when children younger than 16 years of age drive ATVs than for drivers 16 to 34 years of age, and 4.5 times higher than for drivers 35 to 54 years of age. Over 2400 deaths associated with 3- and 4-wheel ATVs occurred from 1982 to 1993.[9] In 2002, approximately 111,000 people sustained injuries related to ATVs.[38]

The majority of injuries associated with ATVs occur when the driver loses control, the vehicle rolls over, the driver is thrown off the vehicle, or the driver collides with a fixed obstacle. Children are often injured when struck by a fence wire or tree branch while traveling at a high speed. In a recent study of three-wheeled ATVs in Alaska,[16] risk factors were found to include intoxication with alcohol, excessive speed, lack of proper protective helmet use, and rider inexperience.

All-terrain vehicles should be used only in daylight or driven very slowly at night. These vehicles should never be used on unfamiliar terrain or on public roadways. ATVs should not carry more than one person at a time. Riders should always wear helmets, eye protection, and protective clothing, such as gloves and hand guards.

Drivers should always use the buddy system, driving with other ATVs. In case of injury in a remote area, a buddy ATV can go for help.

In 1982 an estimated 12,000 children 14 years of age and younger suffered motorized minibike- and trailbike-related injuries.[31] Children aged younger than 16 years account for more than 30% of all ATV-related injuries.[41] Children have a high incidence of trauma to the central nervous system when involved in ATV crashes.[10] During the past 2 decades, approximately 5000 adults and children were killed in ATV crashes. In a recent 4-year period, the annual number of injuries caused by ATV crashes nearly doubled to more than 110,000 in 2001. The number of ATV-related deaths has also been on a steep rise.[41]

All-terrain bikes and mountain bikes are a popular form of recreation for wilderness enthusiasts. The rider may be traveling on uneven trails with multiple obstacles, including logs, rocks, and streams. Falls usually cause the rider to lunge forward and sustain concomitant injury to the face and head. Consequently, riders should wear full-face helmets with chin guards to limit maxillofacial injuries. Other forms of injury prevention gear include gloves and hand, elbow, knee, and shin guards. The rider should also wear eye protection if it is not a part of the helmet.

Snowmobiles. In New Hampshire, between January 1989 and February 1992, there were 12 fatal and 165 nonfatal snowmobile injuries.[6] None of the snowmobile drivers who died had taken an off-highway recreational vehicle (OHRV) course. Overall, 76% of the fatal incidents were associated with alcohol use and 67% with excessive speed. Of 165 persons nonfatally injured, 104 (63%) were reported to have been wearing helmets. Helmets were reported to have been worn by 31 (57%) of 54 persons with nonfatal head injuries, compared with 4 of 6 persons (67%) with fatal head injuries. In Maine, from fall of 1991 through spring of 1995, 25 (81%) of the 31 who died from snowmobile-related causes were wearing helmets at the time of the incident.[7] The observation that a large percentage of deaths occurred in persons wearing helmets demonstrates that this behavior modification does not ensure survivability. Other contributing factors, such as excessive speed, alcohol, and lack of other corporeal protection, have a significant impact on outcome. During the 2002–2003 winter season in northern New England, 28 deaths in 3 states were associated with the use of snowmobiles, more than reported during any of the previous 12 winter seasons.[8]

In Alaska, between 1993 and 1994, injury, death, and hospitalization rates were greater for snowmobiles than for on-road motor vehicles.[28] A total of 26 snowmobile injury deaths were reported. Seven decedents drowned after breaking through ice, and eight were ejected from vehicles. More than half (58%) of the snowmobile injury deaths involved a natural object, such as a boulder, ravine, or river. Of the 17 decedents for whom blood alcohol concentrations were available, 11 (65%) had blood alcohol concentrations of at least 100 mg/dL. Thus, natural obstacles, excessive speed, and alcohol intoxication contribute to the high risk of injury death associated with snowmobile use.

It is irresponsible to venture into the wilderness assuming that, if an accident occurs, someone else will solve the problem. Preparation for emergencies and rescue is essential when planning a wilderness excursion. With proper planning and application of injury prevention principles discussed in this chapter, rescue situations can be averted, thereby avoiding risk to others.

INTERNET RESOURCES

Internet sources related to wilderness injury prevention can be found online at www.WildernessMD.com.

The references for this chapter can be found on the accompanying DVD-ROM.

17 Principles of Pain Management

Bryan L. Frank

Safe and effective pain management in the wilderness often necessitates special considerations because of remote location, extrication considerations, and concomitant illness or injury that may impact the effects of physical modalities, anesthetics, analgesics, and other pharmacologic agents. More adventurers are traveling into wilderness and remote settings with chronic pain syndromes that pose additional considerations. World travelers are often exposed to various indigenous therapies for acute and chronic conditions. Many of these therapies are becoming more available to American wilderness travelers as complementary and alternative modalities. These therapies can be integrated well with conventional medical therapies and occasionally may be superior to conventional approaches for the treatment of acute or chronic pain in the wilderness.

The old adage "nobody ever died of pain" may be very inappropriate in a wilderness setting. Effective pain management may dramatically enhance a rescue effort and thus minimize morbidity and mortality. Pain management advances that over the past decade have dramatically altered care in emergency departments and operating rooms also have an impact on care in the wilderness.

PATHOPHYSIOLOGY OF PAIN

Perception of pain is critically important for the survival of most organisms. Detection of tissue threat or injury leads to activation of the sensory nervous system. Nociception, which is the interpretation of pain, initiates protective behavior, including withdrawal, a sympathetic response, and mood changes. The simple nociceptive pain response seen with acute injury becomes much more complex in chronic pain syndromes, where the original insult is long past. Numerous distinct clinical pain entities have been recognized, including acute nociceptive pain (somatic and visceral), postoperative pain (post-traumatic), neuropathic pain, terminal pain, chronic pain, and psychogenic pain. The

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wilderness physician who is informed and prepared can generally manage acute nociceptive pain. Chronic pain may pose greater difficulty.

Acute Nociceptive Pain

Designed to protect us from environmental dangers, acute pain is critical to human survival and well-being. To respond to acute trauma, the human nervous system contains peripheral receptors that detect noxious events, peripheral transmission to relay information centrally, central processing mechanisms, and response apparatuses.[58]

The body's external-most element, skin, is richly innervated by polymodal nociceptors, which respond to mechanical, thermal, and chemical stimuli. These nerve fibers are generally A-delta and unmyelinated C fibers and are most prevalent in the skin. In the normal state, greater stimuli lead to greater frequency of action potentials and increased pain perception. Acting as transducers, these nociceptors transmit their signals to the spinal cord via primary afferent nerve fibers,[59,60] where the signals may be amplified or dampened before transmission to the brain.

The peripheral nerve terminals experience a change in milieu following tissue damage and subsequent plasma extravasation due to increased capillary permeability. These events lead to a red flush at the site of trauma secondary to arteriolar dilation, local edema caused by increased capillary permeability, and primary and secondary hyperalgesia. In addition to this decreased pain threshold, there may be pain from normally non-noxious stimuli (allodynia), and an exaggerated response to noxious stimuli (hyperpathia). Tissue substances released in response to tissue injury may greatly magnify the nociceptive response of the body. Histamine and serotonin may further lead to stimulation of free nerve endings and vasodilation. Kinins, including bradykinin, are also released after tissue trauma and stimulate free nerve endings, as can lipidic acids, cytokines, and various primary afferent peptides (calcitonin gene-related peptide, substance P, and others) (Box 17-1).[59,60]

In the dorsal horn of the spinal cord, connections are made from peripheral nerves to one of five zones, know as lamina I to lamina V. Connections within these laminae are differentiated on the basis of cell type, interneural connections, and histochemistry. Neurochemicals within the dorsal horn include substance P, beta-endorphin, enkephalins, dynorphin, serotonin, dopamine, norepinephrine, vasoactive intestinal peptide, gamma-aminobutyric acid (GABA), neurotensin, thyrotropin-releasing factor, oxytocin, and others.[58,60]

Somatic afferent fibers and large-diameter myelinated primary afferent axons, with cell bodies in the dorsal root ganglia, ascend within the spinal cord and terminate in the cuneate and gracile nuclei. Other branches of these primary afferents terminate in the anterior horn of the spinal cord. The spinothalamic tracts (neo- and paleo-) ascend the anterolateral pathways after crossing from the contralateral dorsal horn. These fibers then terminate in the thalamic nuclei or with collateral connections in the brainstem and midbrain, where they may modulate sensory transmission.[58]

Descending inhibitory pathways that modulate acute painful signaling involve mechanisms utilizing enkephalins, serotonin, noradrenaline, and probably other neurotransmitters. Central structures involved in this activity include the somatosensory cortex, periventricular gray matter, periaqueductal gray matter, dorsal raphe nuclei, reticular formation, medulla, and nucleus raphe magnus.[58] These mechanisms and others are involved in modulation of the nociceptive experience.

Box 17-1. Neurochemicals of Pain Transmission

NEUROPEPTIDES
Adrenocorticotropin
β-Endorphin
Bombesin
Calcitonin gene-related peptide
Cholecystolium
Corticotropin-releasing factor
Dynorphin
Enkephalins (leu-, met-)
Galanin
Neurotensin
Oxytocin
Somatostatin
Substance P
Substance K
Vasoactive intestinal peptide
Vasopressin

MONOAMINES
Dopamine
Norepinephrine
Serotonin

AMINO ACIDS
Aspartate
Gamma-aminobutyric acid (GABA)
Glycine
Glutamate
Taurine

Chronic Pain

In the experience of chronic pain, the sensation of pain no longer serves to warn the organism of trauma and danger. The duration of pain is not the important factor in characterizing pain as chronic. Rather, it is the prevalence of cognitive-behavioral aspects that supersede nociception. Prolonged pain that is traumatic, postoperative, neuropathic, or terminal is not called chronic if the pathophysiologic and psychological characteristics of the acute nature are maintained.[58]

Chronic pain is poorly defined through co-involvement of peripheral and central mechanisms. The initial stress response seen with acute pain is no longer present. Psychologically, the patient with chronic pain is commonly depressed. The sufferer may seek secondary gain and demonstrate psychosis, neurosis, or a premorbid personality. More so than with acute pain situations, pharmacologic interventions for patients with chronic pain are often very challenging. Anti-inflammatory agents, neural blockade, sedative-hypnotics, and narcotics are often ineffective or contraindicated, whereas antidepressants, cognitive coping strategies, and behavioral modification may be useful.

Recent therapies for patients with chronic pain that may be utilized by wilderness travelers include implantable infusion pumps that deliver narcotic and non-narcotic medications, dorsal column spinal stimulators, and transcutaneous electrical nerve stimulators (TENSs).

GOALS OF WILDERNESS PAIN MANAGEMENT

Wilderness pain control may be difficult to achieve because of complex physiologic and environmental challenges. The primary goal should be safety, and the secondary goal effective pain relief. However, failure to provide adequate analgesia may place a sick or injured patient, as well as the rescue party, at grave risk.

It is important to assess the nature of the injury or illness and determine the diagnosis while addressing the need for and choice of pain therapy. It is often important to withhold pain therapy until a reasonable diagnosis can be determined, which may be difficult without sophisticated diagnostic equipment. Generally, however, the time-tested skills of history taking and physical examination should lead to an appropriate diagnosis by an experienced physician.

Evaluation of the Pain Patient

The cornerstone of accurate pain evaluation is observation, examination, and focused history taking. This should determine the location of the pain, time of onset, precipitating or aggravating factors, frequency and duration, character, severity, and previous treatment. General medical history includes current medications, past medical and surgical history, menstruation and pregnancy, environmental exposures, dietary intake, and associated symptoms of nausea or vomiting, fever, vertigo, dyspnea, weakness, or numbness. Many injuries in the wilderness may be reasonably straightforward to diagnose, but pain related to medical illness may be a diagnostic dilemma. Open-ended questioning may provide greater insight than the direct questioning necessitated by constraints of time and safety. Changes in cognition may compromise patient evaluation. Pre-travel history forms and information or observations of traveling companions may be critical to the evaluation of a painful condition.

If pressed by critical environmental or patient conditions, physical examination should be performed quickly and systematically, targeted by clues obtained during the history. When possible, a focused examination, including a general assessment of contributing factors such as obvious blood loss, tissue derangement, ecchymoses, rash, other skin disturbance, animal bite, blood pressure and pulse, mental status, cranial nerve function, motor and sensory function, deep tendon reflexes, and strength, may provide vital information.[13]

PHYSICAL METHODS FOR TREATMENT OF PAIN

Wilderness pain management includes simple physical measures such as applying pressure, cold, heat, or splinting, which are important adjuncts to narcotic analgesics, anesthetics, and other pharmaceutical agents that utilize topical, oral, intranasal, rectal, or parenteral administration (Box 17-2). Additionally, pain treatment modalities characterized as complementary and alternative therapies may provide significant pain relief with less physiologic risk.

Compression Analgesia

Although compression is taught more as a method for establishing hemostasis than for pain management, it has been known for centuries to reduce pain. An injured extremity is wrapped firmly with an elastic cloth wrap or a rubber Esmarch bandage from a point on the extremity just proximal to the injury site and extending more proximally on the extremity for at least 3 to 4 inches (7–10 cm) and up to approximately 12 inches (30 cm) of compression wrapping. Alternatively, a simple nonvascular occlusive constriction bandage may be placed on the limb proximal to the site of the injury. Resultant anesthesia is thought to occur due to compression of the peripheral nerves.[38] Distal pulses should be evaluated and the compression wrap should allow for a palpable pulse. If the wrap (or a tourniquet to stop bleeding) obliterates the pulse, the compression must be released periodically to prevent ischemia in the limb. In cold environments, where the risk of ischemia is increased further, release of the compression should be more frequent. Compression anesthesia may be safe and appropriate in a wilderness setting if other methods or pharmacologic agents are unavailable or contraindicated.

Cryoanalgesia

Hippocrates first recorded use of ice and snow packs to relieve pain in the 4th century BC. Cryoanalgesia experienced a significant modification by Richardson in 1866 with the introduction of refrigerant ether spray, replaced by the use of ethyl chloride

Box 17-2. Methods of Pain Management

PHYSICAL MODALITIES
Compression
Cryoanalgesia
Heat therapy
Splinting
Transcutaneous electric nerve stimulator (TENS)
Pharmacologic modalities

ANALGESICS
Narcotic agonists
Narcotic agonist/antagonists
Salicylates
Para-aminophenols
Nonsteroidal anti-inflammatory drugs (NSAIDs)
Dissociative analgesics
Novel nonopioid, noncyclooxygenase inhibitor analgesics

LOCAL ANESTHETICS
Amide local anesthetics
Ester local anesthetics

ADJUNCTIVE PHARMACEUTICALS
Antidepressants
Anticonvulsants
Antihistamines
Muscle relaxants
Intra-articular injections of hyaluronan substances

COMPLEMENTARY AND ALTERNATIVE THERAPIES
Acupuncture
Herbal and botanical remedies
Magnetic therapy

spray in 1890. Cooper advanced cryoanalgesia in 1961 through the development of the liquid nitrogen probe, which served as prototype for the nitrous oxide or carbon dioxide cryoanalgesia devices in common use today.[38,45]

Wilderness cryoanalgesia may be applied with ice, snow, or frigid water. Additionally, metal cylinders containing gasoline or ethyl alcohol (freezing point –174.1°F [–114.5°C])[10] remain liquid at temperatures below water's freezing point and may be used to provide a dry cold compress.[56] However, these containers of subzero-degree liquids may lead to serious frostbite injury. As cold absorbs heat from the adjacent tissues, nerve conduction is reversibly blocked. Conduction ceases in the larger myelinated fibers before the unmyelinated fibers; all nerve conduction ceases at 32°F (0°C). On rewarming, nerve conduction resumes, unless the intracellular contents have turned to ice crystals. Extreme cryotherapy, such as that experienced with frostbite, leads to wallerian degeneration of axons and myelin sheaths, although the perineurium and epineurium may remain intact, which sometimes allows regeneration.[45]

Although a selective cryolesion is the goal of many pain management treatments in chronic pain clinics, this deliberate cell injury is not appropriate in a wilderness setting. Prevention of iatrogenic frostbite and generalized hypothermia while using cold therapy is critical. The specific length of time that tissue can tolerate a cold compress before experiencing cellular damage is variable, depending on preexistent tissue hypothermia, the peripheral versus central nature of the tissue, and the temperature and pressure of the cold compress. Cold-water immersion may induce frostbite in persons with snakebite because of venom-compromised tissues. Cold packs may be beneficial for certain marine envenomations. Commercial cold packs typically contain a gel of water and propylene glycol, or other similar antifreeze and heat-exchange substances, which may be cooled in cold water or snow.[24] A reasonable guide is to place a dry, thin cotton cloth or foam between the skin and the cold metal cylinders, ice or snow, or cold packs and to remove cold therapy every 15 minutes to assess tissue status.

Heat Therapy

Application of heat, including hot-water immersion, is not usually recommended for initial (up to 48 hours) pain management of acute trauma when there is a risk of edema or bleeding. There are, however, times when it is reasonable to use heat for pain management in the wilderness—for example, for muscle strains and some stable fractures. Muscle relaxation, attributed to decreased gamma-fiber activity, has been demonstrated after applying heat to the overlying skin. Collagen tissues become more extensible with heat therapy, requiring less force for movement and resulting in less mechanical damage with stretching. These effects may be useful for patients pursuing wilderness travel, especially with chronic pain conditions. Furthermore, heat applied to the skin of the abdomen may markedly reduce gastrointestinal (GI) peristalsis and uterine contractions and thus decrease pain associated with these organs.[52]

Application of heat need not be extreme. Temperatures in the range of 100° to 104°F (37.8° to 40°C) for 10 to 20 minutes will generally be comforting, without leading to thermal injury. Heat therapy should be avoided in patients who are cognitively impaired, dehydrated, or intoxicated and should be avoided on tissue that is anesthetic or ischemic, to prevent further unintended tissue injury. Heat therapy may worsen or improve certain marine envenomations or lead to lymphangitis.[19]

Liniments and balms are not true heat transfer agents; rather, they typically consist of multiple botanical or chemical substances. Application of small amounts of these drugs may make the tissue feel warm through counterirritant effects and subsequent vasodilation. Some contain capsaicin or menthol, which activates small numbers of thermal nociceptors. Through vasodilation, these substances may help a traveler's soreness and stiffness abate. Common ingredients include menthol, camphor, mustard oil, eucalyptus oil, methyl nicotinate, methyl salicylate, and wormwood oil.[6] Use of these products is generally recommended only on intact skin with a light cloth or plastic covering, and they should not be placed on mucous membranes. They also should not be used with tight compresses or external heat sources.

Splinting

Splinting allows positioning and immobilization of injured body parts and prevents further damage to soft tissues, blood vessels, nerves, and bones. Preventing bony fragments from damaging surrounding tissue diminishes pain and often facilitates mobilization and extrication of a victim.

Splints should be padded to prevent further surface trauma. They may be accompanied by pressure dressings or cold compresses for additional pain management. Regular reevaluation of tissue circulatory status is critical to prevent damage from swelling, frostbite, or ischemia in immobile, splinted limbs.

Transcutaneous Electrical Nerve Stimulation

Shealy and Liss developed TENS as a trial for patients considering spinal cord electrical stimulation to relieve chronic pain.[1] TENS units are in such common use that it is likely that wilderness physicians will encounter persons utilizing these devices. The mechanism of action is believed to be stimulation of large, myelinated A-fibers, which modulate some C-fiber pain sensation by keeping it from reaching the brain. This process is explained at least in part by the gate control theory of Melzack and Wall.[41] Additionally, Pomeranz and others have shown the neurohumoral effects of electrical stimulation to involve endorphins, enkephalins, serotonin, GABA, adrenocorticotropic hormone (ACTH), and other modulators. High-intensity, low-frequency electrical stimulation elicits an endorphinergic response, whereas low-intensity, high-frequency electrical stimulation leads to spinal segmental analgesia, and monoamine-based pre- and post-synaptic inhibition of pain signals.[49] Some studies have demonstrated increased plasma and cerebrospinal fluid (CSF) levels of endorphins with high- or low-frequency electrical stimulation, but other studies have failed to demonstrate these changes. Conventional TENS units utilize stimulation frequencies of 50 to 80 Hz, whereas acupuncture-like TENS devices utilize stimulation frequencies of less than 10 Hz to greater than 100 Hz.

Wilderness physicians familiar with TENS units may find them to be very useful for treating acute traumatic pain. More likely, they will encounter a traveler with a TENS unit. Most persons have developed personal preferences for TENS unit settings and skin electrode placement. Rigorous physical activity or sweating may cause difficulty maintaining electrode placement, as may excessive hair or deformities. Application to clean, dry skin is important. Preparing the skin with topical benzoin may significantly increase the duration of adhesiveness. Because benzoin is an iodine-based topical solution, it should be avoided in persons with sensitivity or allergy to iodine.

TABLE 17-1. Common Oral Analgesics: Dosage Recommendations in 70-kg Adults

DRUG	DOSAGE (mg)	INTERVAL (hr)	RISKS, PRECAUTIONS
Salicylates			
Acetylsalicylic acid	325–650	4–6	Gastrointestinal (GI) distress, inhibits platelet function, contraindicated in children with viral illness
Diflunisal	300–600	8–12	Similar to aspirin
Para-Aminophenol			
Acetaminophen	300–1000	4–6	Hepatic toxicity in overdose
Indoles			
Indomethacin	50	4–6	Similar to aspirin
Sulindac	50–75	4–6	Similar to aspirin
Ketorolac	10	4–6	Similar to aspirin, 5 days or less due to GI bleed
Propionic Acids			
Fenoprofen	400–600	4–6	Similar to aspirin
Ibuprofen	300–400	4–6	Similar to aspirin
Ketoprofen	25–50	6–8	Similar to aspirin
Dexketoprofen	25	8	Similar to aspirin, less GI distress
Naproxen	200–275	6–8	Similar to aspirin
COX-2 Inhibitors			
Celecoxib	100–200	12–24	GI distress, skin rash
Valdecoxib	10	24	GI distress, hepatic side effects
Narcotic Agonists			
Codeine	15–60, max 360 mg/24 hr	4–6	Narcotic side effects; note cumulative acetaminophen dosage
Hydrocodone	5–10	4–6	Narcotic side effects; note cumulative acetaminophen dosage
Hydromorphone	7.5	3–4	Narcotic side effects
Levorphanol	4	6–8	Narcotic side effects
Meperidine	300	2–3	Narcotic side effects
Methadone	2.5–150	4–12	Narcotic side effects
Morphine	30–60	3–4	Narcotic side effects
Oxycodone	5–10 Extended release, 10	4–6 12	Narcotic side effects; note cumulative acetaminophen dosage
Propoxyphene	HCl 32–65, max 390 mg/24 hr Napsylate 100, max 600 mg/24 hr	4–6 4–6	Narcotic side effects; note cumulative acetaminophen dosage
Miscellaneous			
Tramadol	50–100	4–6	Seizure risk at >400 mg/24 hr; reduce dosage in older adults; patients with cirrhosis, 50 mg every 12 hr

COX-2, cyclooxygenase-2.
From Burnham T, et al (eds): Drug Facts and Comparisons. St. Louis, Facts and Comparisons, 1999; Emermann CL, Spenetta J: Pain management in the emergency department. Emerg Med Rep 23:53–67, 2002; and Lawrence R, Rosch P, Plowden J: Magnet Therapy: The Pain Cure Alternative. Rocklin, CA, Prima, 1998.

PHARMACOLOGIC TREATMENT OF PAIN

Analgesics

See Tables 17-1 and 17-2 for oral and parenteral dosage recommendations.

Opioid Analgesics

Opium emerged as the first widely used narcotic analgesic by the time of the Renaissance, generally in the form of a powder or sticky gum. It was often combined with alcohol to form laudanum. Prussian pharmacist Frederich Sertürner isolated morphine from opium in the 19th century. Development of the hypodermic needle and syringe by Rynd in Ireland and Pravaz in France greatly enhanced morphine's clinical utility in pain management.[38]

Narcotic agonists affect the mu (μ), kappa (κ), and delta (δ) opiate receptors in the central nervous system (CNS) and periphery.[44] Morphine and other opioids are administered orally, intranasally,[48] sublingually, transdermally, subcutaneously, intramuscularly, intravenously, and rectally. Morphine is metabolized primarily in the liver; approximately 10% is excreted through the kidneys. Hepatic and renal damage impact the recipient's response, so tolerance for a given dosage must not be presumed with these conditions. Bioavailability depends on tissue perfusion. Morphine is a potent analgesic with sedative and euphoric effects that also depresses the CNS and respiratory drive. Critical patient assessment is always required when using narcotic analgesics to detect respiratory depression and hypotension. This is particularly true in situations of hypoxia, such as occur at high altitude.

Other opioid agonists include codeine, which is a controlled substance in the United States but available over the counter in many other countries, and meperidine, methadone, propoxyphene, and hydromorphone. Semisynthetic narcotics that exhibit much shorter durations of action include fentanyl and sufentanyl.

TABLE 17-2. Common Parenteral Analgesics: Dosage Recommendations in 70-kg Adults

DRUG	DOSAGE (mg)	INTERVAL (hr)	RISKS, PRECAUTIONS
Narcotic Agonists			
Codeine	15–75 IM	4–6	Narcotic side effects
Fentanyl	50–100 µg	0.5–1	Narcotic side effects, wide range of dosages
Hydromorphone	1–2 IM	3–4	Narcotic side effects, choice over morphine in hepatic impairment
Levorphanol	4	6–8	Narcotic side effects
Morphine	10–20 IM, 2.5 IV	3–5	Narcotic side effects
Meperidine	50–100 IM, 25–50 IV	2–4	Narcotic side effects, active metabolite accumulates in renal impairment and may cause seizures
Oxymorphone	1	3–4	Narcotic side effects
Narcotic Agonists/Antagonists			
Buprenorphine	0.3–0.6 IM	6–8	May precipitate narcotic withdrawal
Butorphanol	2–4 IM	3–4	May precipitate narcotic withdrawal
Dezocine	5–20 IM, 5–10 IV	2–4	May precipitate narcotic withdrawal
Nalbuphine	10–20 IM, 1–5 IV	3–6	May precipitate narcotic withdrawal
Nonsteroidal Anti-Inflammatory Drugs (NSAIDs)			
Ketorolac	15–30 IM, 2–5 IV	4–6	Similar to aspirin
Dissociative Analgesic/Anesthetic			
Ketamine	50–75 IM, 15–30 IV	2–4	Increased intracranial pressure

IM, intramuscularly; IV, intravenously.
From Burnham T, et al (eds): Drug Facts and Comparisons. St. Louis, Facts and Comparisons, 1999; Emermann CL, Spenetta J: Pain management in the emergency department. Emerg Med Rep 23:53–67, 2002; and Lawrence R, Rosch P, Plowden J: Magnet Therapy: The Pain Cure Alternative. Rocklin, CA, Prima, 1998.

Potential adverse side effects may include obtundation, sedation, anaphylaxis, nausea, vomiting, rash, respiratory depression, and hypotension. Narcotic antagonists such as naloxone and other emergency resuscitative medications should be available when using opioids in the wilderness, just as in the contemporary clinical setting. Potent opioid analgesics should be given with care to victims with suspected head injury or neurologic illness.[57]

Narcan (Naloxone) is an opioid antagonist that may reverse narcotic effects. Doses of 0.2 mg intravenously (IV), or 0.4 mg IV, intramuscularly (IM), or subcutaneously, may be given and repeated every 2 to 5 minutes until CNS, respiratory, or hypotensive narcotic symptoms are reversed, to a maximum dosage of 10 mg. Nalmefene is a similar narcotic antagonist but with a longer duration of action. Intravenous dosages of 0.25 µg every 2 to 5 minutes to a total dosage of 1 mg (four doses) may be given.[6] Continuous monitoring of blood pressure, mental status, and respiratory status is critical, with possible repeat doses necessary in 1 to 2 hours. Pain may return with narcotic reversal; a balance between pain alleviation and physiologic stability is desired. Additionally, narcotic antagonists may lead to acute narcotic withdrawal symptoms in persons with a tolerance to and dependence on narcotics.

Narcotic agonist-antagonist combinations first became popular with pentazocine, which causes less euphoria, but it is abused and so has addictive potential. These agents in general cause less addiction but may depress the brain and respiration,[39] which may have special significance in a high-altitude setting where impaired cerebral and pulmonary function may occur. These medications do not provide the narcotic agonist effects needed in narcotic-dependent persons, so they may experience withdrawal symptoms without narcotic agonists. Familiar drugs in this class include buprenorphine, butorphanol, and nalbuphine.

Non-narcotic Analgesics

Salicylates. Non-narcotic analgesics provide mild to moderate pain relief and are generally safer than narcotics. Acetylsalicylic acid (aspirin) is a mild analgesic that reduces fever, inhibits platelet function, and diminishes inflammation. It has significant GI effects including gastric irritability, erosion, and bleeding ulcers, so it is often better tolerated in an enteric-coated formulation. Persons who have a history of GI ulcers or severe indigestion should avoid aspirin. Dosage should not exceed 650 mg orally every 4 hours. Other salicylate analgesics include diflunisal, choline magnesium trisalicylate, and salsalate. The latter two have minimal GI toxicity and antiplatelet effects. Salicylates are contraindicated in persons with known allergy to the class of drugs, and in children (less than age 15 years) with viral respiratory illnesses, because their use has been linked to Reye's syndrome.[1]

Para-Aminophenols. Acetaminophen is a para-aminophenol that is a mild analgesic and antipyretic.[11] It has no antiplatelet, anti-inflammatory, or antiprostaglandin effect, and it is less likely than aspirin to cause serious gastric irritation. The dosage should not exceed 650 mg every 4 hours. Ingestion of more than 10 g over 24 hours may lead to severe hepatic damage; the drug should be used with extreme caution in anyone with preexisting liver disease. Many over-the-counter medications contain acetaminophen, so these should not be used in combination with pure acetaminophen or in persons with liver disease.

Nonsteroidal Anti-inflammatory Drugs

Nonsteroidal anti-inflammatory drugs (NSAIDs) are effective for mild to moderate pain and may provide ample clinical benefit without concern for the respiratory depression seen with narcotic analgesics. The mechanism of action is most likely a result of inhibition of prostaglandin-mediated amplification of

chemical and mechanical irritant effects on the sensory pathways.[37] NSAIDs undergo rapid absorption after oral or rectal administration, followed by hepatic metabolism and renal excretion of their conjugated metabolites. Injectable forms of various NSAIDs, such as ketorolac, have also been developed in recent years. This nonsteroidal class of medications includes chemically unrelated compounds that are grouped together because of their therapeutic actions. These include acetic acid, propionic acid, oxicam, and pyrazolone derivatives.

Adverse effects of NSAIDs include GI distress and bleeding, CNS disturbances (vertigo, drowsiness), renal dysfunction, and prolonged bleeding from platelet function inhibition. In certain circumstances, NSAID-induced immunosuppression increases propensity to bacterial infection.

Indomethacin is an indoleacetic acid derivative with analgesic, antipyretic, and anti-inflammatory (antiprostaglandin) effects. Both rectal and oral administrations lead to rapid absorption. Reasonable caution should be used in persons with hepatic or renal dysfunction. Gastrointestinal toxicity has been reported with its use, including gastric ulceration and perforation, as well as bone marrow suppression. Other acetic acid derivatives include sulindac, tolmetin, ketorolac, and diclofenac.

Ibuprofen is a nonsteroidal propionic acid derivative anti-inflammatory agent with prostaglandin antagonist activities. It has analgesic and antipyretic properties similar to those of aspirin and acetaminophen, but it may be better for women with dysmenorrhea because its antiprostaglandin effects somewhat relax the uterus. Its anti-inflammatory properties may be very useful for arthritis and acute injuries. Like aspirin, ibuprofen can be a gastric irritant and should be avoided in persons with a history of GI ulcer, indigestion, or hiatal hernia. Oral and rectal administrations lead to rapid absorption. Ibuprofen has been reported to cause nonspecific fluid retention. Although not studied in wilderness medicine research to this point, its potential to aggravate high-altitude illnesses, from acute mountain sickness to high-altitude pulmonary or cerebral edema, should be considered. The usual dosage is 400 to 600 mg every 4 to 6 hours. Other propionic acid derivatives include naproxen, fenoprofen, ketoprofen, and flurbiprofen.[11]

A newer class of NSAID medications available in the United States is the cyclooxygenase-2 (COX-2) inhibitor group, consisting of celecoxib (Celebrex) and valdecoxib (Bextra), as well as the recently withdrawn medication rofecoxib (Vioxx). Rofecoxib was withdrawn in 2004 as a result of studies showing significant increases in adverse cardiovascular events in patients taking the medication continuously for 18 months.[69] These medications inhibit inflammation and also demonstrate analgesic and antipyretic properties. The analgesia offered by COX-2 inhibitors is similar to that of other NSAID medications, but the COX-2 inhibitors have less GI toxicity.[47] The mechanism of action is believed to be through inhibition of prostaglandin synthesis via inhibition of cyclooxygenase-2. These medications may be taken orally once per day, but they are not cleared for patients younger than 18 years or for patients with advanced renal disease. Elimination is primarily via hepatic metabolism, with little of the drug recovered in the urine. Side effects primarily include GI distress, hypertension, skin rashes, and peripheral edema. The recommended dosage for rofecoxib is 12.5 to 25 mg once daily. Celecoxib may be given either 100 mg twice a day or 200 mg daily (neither method has a clinical advantage over the other). The recent demonstration of adverse cardiac events shown in patients who are given high dosages of COX-2 may become extended to naproxen and other classes of pain medications as more data are evaluated.

Dexketoprofen (Keral) is a recently developed stereoisomer of the NSAID ketoprofen. It is currently available in most of Europe and Central America, and it is being considered for inclusion in the pharmacopoeia of Africa and Asian countries, although it was removed from the market in the United Kingdom in March 2004.[7,66] It is a rapidly acting analgesic with fewer GI distress symptoms than most NSAID medications. The eutomer has been separated and the inactive isomer (distomer) has been discarded to potentially reduce unwanted side effects. These changes have resulted in halving the dosage relative to ketoprofen, with similar clinical effects. The usual dosage is 25 mg three times a day, which has been tolerated on an empty stomach. Taking the medication 30 minutes prior to eating may further speed its onset of action.[50,65]

Dissociative Analgesia

Ketamine is a dissociative anesthetic agent that has been demonstrated to provide significant analgesia. Its mechanism of action is probably related to its agonist effects on the glutamate receptor of the *N*-methyl-D-aspartate (NMDA) subtype.[45] NMDA is thought to affect central or peripheral neuropathic pain transmission more than it does nociceptive transmission in tactile or thermal modalities.[3,28] Ketamine is believed to activate μ-opioid receptors responsible for analgesia and δ-opioid receptors for dysphoria.[21] As a consideration in wilderness settings where respiratory depression may be a significant concern, ketamine may provide significant analgesia, and its dissociative state, with less respiratory depression or loss of glossopharyngeal reflexes. Ketamine possesses sympathomimetic activity that may prove useful in injured persons with depressed cardiac function or shock, and it may also provide bronchodilation for victims with reactive airway disease. It is contraindicated with head injury because it increases intracranial pressure,[27] which is caused by increased cerebral blood flow and direct cerebral vasodilation. Cerebral metabolic oxygen requirements increase as well.[4] Ketamine is ideally administered by a trained physician with adequate monitoring capability.

An intravenous dose of 0.2 to 0.4 mg/kg of ketamine provides analgesia, and 1 to 2 mg/kg IV or 2 to 4 mg/kg IM leads to profound analgesia and a dissociative state. A quiet and calm setting will diminish unpleasant dissociative experiences for the recipient.

Novel Nonopioid, Noncyclooxygenase Inhibitor Analgesics

Capsaicin, the crystalline alkaloid in chili peppers (*Capsicum* species) that causes the heat sensation, has been used increasingly in recent decades for pain management. This odorless, colorless, tasteless alkaloid retains its potency despite time, cooking, or freezing. Capsaicin was discovered by Bucholtz in 1816 and was first synthesized by Spath and Darling in 1930.[64] In fact, capsaicin refers to a complex of one synthetic and six naturally occurring and related compounds known as capsaicinoids. These compounds range in potency from mild to extremely hot to the taste or with stimulation of the mucous membranes, potentially producing severe irritation and a numbing sensation. One milligram of pure capsaicin can blister the skin and can give the feeling that a burning hot metal probe has been placed on the skin. On ingestion, the alkaloids are

quickly metabolized in the liver and are excreted in the urine within a few hours.[64]

Sensory neuron depolarization occurs following topical application of capsaicin to the skin, with binding to the vanilloid receptor subtype 1 (VR-1) and a deluge of calcium ions entering the neuron. With continuous application, substance P is depleted at the nerve ending. Thus, although the first topical application of capsaicin usually produces a burning sensation, repeated applications become better tolerated, and analgesia ensues. This commonly requires a day or two of being applied four times per day until the burning sensation ceases. Some find using lower concentrations of the commercial applications allows a more tolerable process until the analgesia is established. Once established, it is important that the patient continue the daily use of capsaicin preparations to maintain the analgesia. If the application is not tolerable, a true detergent or rubbing alcohol is generally needed to remove the cream or gel preparation from the skin.[61]

Clinical research and practice have shown usefulness of capsaicin product creams and gels for many painful conditions including postherpetic neuralgia, trigeminal neuralgia, atypical facial pain, diabetic neuropathy, arthritis pain, postmastectomy pain, psoriasis, and epicondylitis. Wilderness medical practitioners may encounter travelers using the product on an ongoing basis for these or other conditions or may consider its use for various painful conditions experienced on an expedition. Commercial preparations are commonly found in concentrations of 0.025%, 0.05%, and 0.075%. Contact of these products with mucous membranes should be avoided.

Neurotropin is an oral nonopioid, noncyclooxygenase inhibitor analgesic developed in Japan. The preparation contains an extract that is isolated from the inflamed cutaneous tissue of rabbits injected with vaccinia virus. Each tablet contains four neurotropin units of extract; the recommended dosage is one tablet twice daily.[29,32] The parenteral preparation has been available in Japan for 50 years, and the oral tablets have been available for 15 years. Although it is not currently available in the United States, it is increasingly marketed and available in other countries and is a viable option for analgesia in patients engaged in wilderness and travel activities in these countries. Neurotropin is indicated in chronic and acute pain, including low back pain, neck-shoulder-arm syndrome, and postherpetic neuralgia. The most common side effects include rash and GI distress, nausea, vomiting, and anorexia. Studies have shown this medication to compare favorably in analgesic quality to ketoprofen. Its mechanism of action is thought to be inhibition of bradykinin release, as well as through the descending pain inhibitory system with effects on serotonergic 5-HT_3 and noradrenergic a_2 receptors in the spinal dorsal horn. It has demonstrated an increased skin temperature effect in peripheral tissues, but the mechanism of action of this effect is as yet unknown.[32]

Local Anesthetics

Local Anesthetic Pharmacology

For centuries, indigenous healers have used coca shrub leaves native to the mountains of the Andes to create a mouth-numbing effect. Gaedicke extracted the alkaloid erythroxylin in 1855, from which Niemann isolated cocaine in 1860.[9] Carl Koller first reported using ophthalmic cocaine anesthesia in 1884. Subsequent enthusiasm led to the use of cocaine for anesthesia of the nasopharynx and oropharynx for surgery of the ear, nose, and throat. Erdtman of Sweden synthesized lidocaine in 1943.[9]

TABLE 17-3. Comparable Anesthetic Dosages* for Peripheral Blocks and Local Infiltration

	DOSAGE (mg/kg)
Amide Anesthetics	
Lidocaine	5
Prilocaine	5
Etidocaine	4
Mepivicaine	5
Bupivacaine	2
Ester Anesthetics	
Procaine	5
Tetracaine	1–2
2-Chloroprocaine	5

*No epinephrine included.

Local anesthetics (Table 17-3) bind to sodium channels on sensory, motor, and sympathetic fibers and block nerve conduction through sodium-blocking properties in free nerve endings, peripheral nerves, spinal roots, and autonomic ganglia. Normally, the cell's sodium-potassium pump constantly pumps sodium out of and potassium into the cell to restore membrane ionic gradients. The anesthetic renders the membrane impermeable to the influx of sodium during depolarization, and the nerve cell thus remains polarized. Anesthetic drug binding occurs within the sodium channel after the drug enters the channel from the intracellular side of the nerve membrane.[12] Variables such as nerve length, rate of nerve impulse transmission, myelination, and the concentration and volume of local anesthetic determine the rate of onset and extent of therapeutic effect. Anesthetic potency and onset and duration of action depend on factors that include volume, perfusion, edema, lipid solubility, protein binding, and ionization. In general, the S stereoisomer has less toxicity and greater duration than does the R stereoisomer. Anesthetic metabolism and elimination are functions of the specific anesthetic's chemical structure.[12]

Lidocaine was the first of the amino amide class of local anesthetics. It is metabolized by hepatic microsomal enzymes. Since its metabolites do not include para-aminobenzoic acid (PABA), allergic reactions are rare. Procaine is a synthetic amino ester local anesthetic. This class of anesthetic is hydrolyzed by cholinesterase to form PABA, which is responsible for the allergic reactions seen with ester anesthetics. Because the esters are relatively unstable, they do not tolerate repeated autoclaving for sterilization.[8] Although no studies have specifically addressed the effects of temperature extremes on supplies in wilderness settings, it is prudent to protect these medications from heat as much as is reasonably possible.

A local anesthetic may provide relief in a topical application prior to more invasive cleansing and debridement. TAC, a mixture of tetracaine (0.5%), adrenaline (1:2000), and cocaine (11.8%) in saline, may be soaked into a sterile bandage and placed directly over a wound. This combination may provide good analgesia and moderate vasoconstriction.[42] However, pediatric deaths and morbidity resulting from cocaine absorption from the aqueous mixture of the original 11.8% cocaine

solution have been reported, especially when there was mucosal absorption of the mixture. Subsequent studies demonstrated that a 5.9% cocaine mixture was also effective, and further, that a viscous preparation may decrease the likelihood of inadvertent mucosal contact. Vinci and colleagues demonstrated that the 5.9% viscous preparation was safe in 25 pediatric emergency patients when contact with mucosal surfaces was avoided .[53]

EMLA (the acronym for eutectic mixture of local anesthetics) is a mixture of 2.5% lidocaine and 2.5% prilocaine. After this cream is applied to intact skin under a nonabsorbent dressing for at least 45 minutes, an invasive procedure such as intravenous needle insertion or suturing of a small laceration may be more easily tolerated. EMLA is now available in 5- and 30-g tubes and as a 1-g anesthetic disc for single application.[63]

Anesthetic Toxicity

The blood level of anesthetic after tissue injection depends on absorption, dosage, metabolism, and elimination. Increased tissue vascularity and vasoactivity of the anesthetic greatly affects blood concentration after injection. Infiltration into a highly vascular site, such as around an intercostal nerve and pleura, leads to a more rapid escalation of the blood level than injection into the less vascular subcutaneous tissues. A mixture of anesthetic and epinephrine leads to slower absorption, but this must be avoided when injecting distal extremities and digits, where epinephrine-induced vasoconstriction may lead to acute ischemic injury. Because of the possibility of unintentional direct intravascular injection, all local and regional anesthetic infiltrations should be made after negative aspiration for blood and in small aliquots between aspiration attempts.

As anesthetic toxicity levels are approached, common early symptoms include circumoral numbness, tinnitus, and cephalgia. CNS toxicity in the form of seizures occurs at lower anesthetic blood levels than does cardiotoxicity in the form of ventricular arrhythmias and cardiovascular collapse. The CNS effects of lidocaine on the brain are paradoxical. At blood concentrations of 3 to 5 µg/mL, lidocaine is an anticonvulsant, whereas blood concentrations of 10 to 12 µg/mL are associated with seizures. Generally, cardiotoxicity is achieved at approximately 150% of the blood level concentration required for anesthetic CNS toxicity. Bupivacaine has demonstrated increased cardiotoxicity, out of proportion to its increased potency relative to lidocaine.[12]

Anesthetic allergy per se is uncommon. It is estimated that perhaps 99% of all adverse anesthetic reactions are related to pharmacologic toxicity of the anesthetic or to epinephrine mixed with the agent.[12] Ester anesthetic allergies are related to PABA metabolites; therefore, intolerance to PABA-containing sunscreens may indicate allergic tendency to ester anesthetics, although this is somewhat controversial. Allergies to amide anesthetics in preservative-free vials are rare. There is no known evidence of cross-sensitivity between amide and ester anesthetic classes.

Anesthetic Infiltration Techniques and Nerve Blocks

Soft tissue analgesia may be accomplished with local injection of 1% lidocaine. Generally, the maximal injectable dosage for lidocaine is 4 mg/kg. Care should be taken to inject from the wound periphery toward the center of the wound to decrease the chance of spreading bacteria or foreign matter to adjacent tissue.[42] In larger wounds, injections should proceed from an area that is previously anesthetized to lessen the discomfort of subsequent injections.

Local anesthetic injection typically causes temporary pain because of the pH of the solution. Buffered solutions are available or may be created by the addition of sodium bicarbonate. Sodium bicarbonate (1 mEq/mL) is added to lidocaine or other anesthetic in a bicarbonate to anesthetic ratio of 1:10. Tolerance to the injection will also be improved by gentle and slow injection, which also allows the physician to be prudent with the total dosage of anesthetic. The addition of epinephrine is not generally recommended for soft tissue, although it may provide useful hemostasis, especially in lacerations of the head and scalp. Epinephrine should be avoided on nose tips, ear lobes, distal extremities, and digits to avoid ischemic injury and even subsequent necrosis.

Many central and regional nerve blocks require special training, including a thorough knowledge of anatomy and management of potential complications. However, several blocks may be appropriate in a wilderness setting if the physician is cautious and limits the amount of anesthetic injected. All infiltrations should be made after aseptic preparation of the skin whenever possible.

Digital Nerve Block

Anesthesia to the digits is easily accomplished with a low-volume field block to the medial and lateral aspects of the digit at the base of the respective phalanx (Fig. 17-1). The digital nerves should be approached from the dorsum of the hand or foot rather than from the palm or sole. The dorsal digital nerves and proper digital nerves course along the medial and lateral aspects of the digits roughly at the 10 and 2, and at the 4 and 8 o'clock positions, respectively, in a sagittal section of the finger. A volume of 3 to 5 mL lidocaine (0.5% to1.0%) injected as a field block with a 25-gauge (or 27-gauge) needle to the medial and lateral aspects of the proximal digit will give a satisfactory digital block. An epinephrine-containing anesthetic should not be used, as this could lead to circulatory compromise and possible necrosis of the digit.

Wrist Block

The entire hand may be anesthetized by blocking the three main nerves at the wrist (Fig. 17-2). The radial nerve at the wrist supplies the cutaneous branches of the dorsum of the hand, the dorsal and palmar aspects of the thumb, and distally to the dorsal aspects of the distal interphalangeal joint of the index finger, the long finger, and the radial aspect of the ring finger. Median nerve sensory distribution includes the palmar surface of the hand, the ulnar aspect of the thumb, and the palmar aspects of the index finger, the long finger, and the radial portion of the ring finger. Median nerve innervation extends dorsally over the index, long, and ring fingers to the distal interphalangeal joint. The ulnar nerve gives sensation to the palmar and dorsal surfaces of the lateral hand, the fifth finger, and the ulnar half of the ring finger.[5]

To perform this block, a 25-gauge needle is used to inject 2 to 4 mL of 1% lidocaine into the subcutaneous tissue overlying the radial artery. A superficial subcutaneous injection from this point and over the radial styloid will anesthetize cutaneous branches that have emerged from the proximal forearm and that extend into the hand. The median nerve is blocked with 2 to 4 mL of 1% lidocaine just proximal to the palmar wrist crease

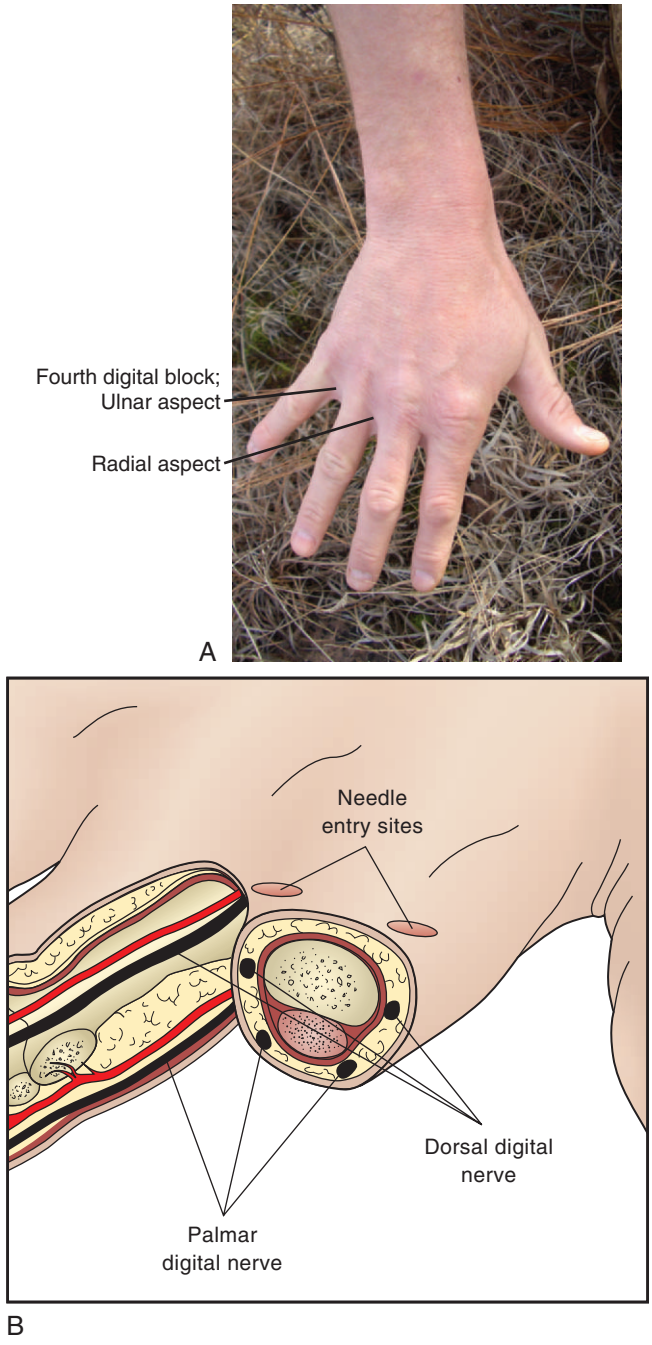


Figure 17-1. **A,** Site of digital nerve block. **B,** Digital nerve anatomy. (**A,** photo by Bryan L. Frank, MD.)

between the tendons of the palmaris longus and the flexor carpi radialis muscles. Injection is made deep to the volar fascia. If a paresthesia is elicited (as a result of contact with the nerve), the needle is withdrawn slightly prior to injection. The ulnar nerve is blocked with 2 to 4 mL of 1% lidocaine injected just lateral

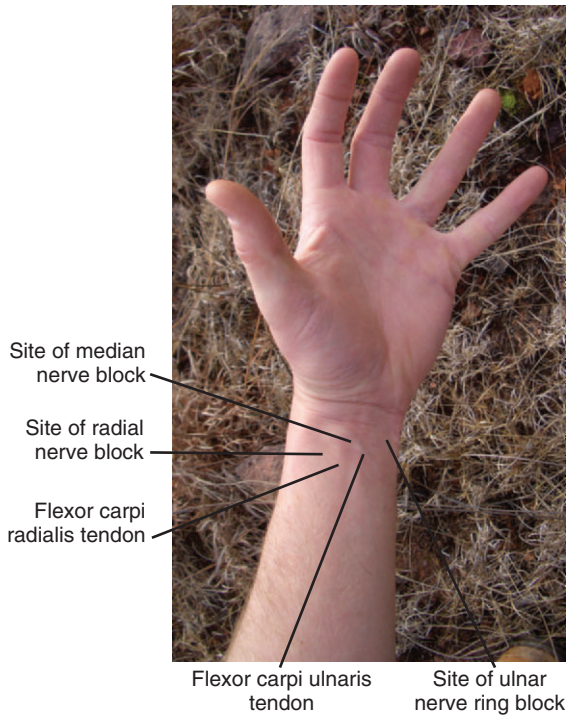


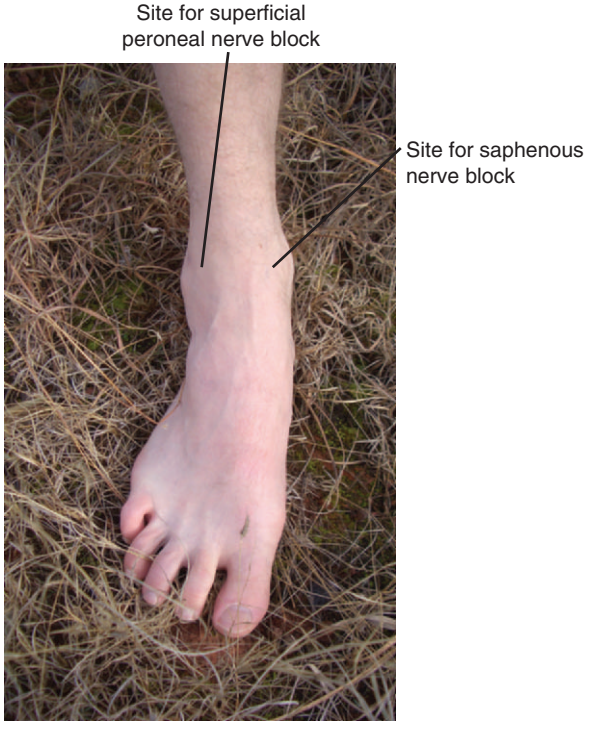
Figure 17-2. Landmarks for wrist block. (Photo by Bryan L. Frank, MD.)

to the ulnar artery, which is radial to the flexor carpi ulnaris tendon at the level of the ulnar styloid. Again, a superficial subcutaneous injection from this site and over the ulnar styloid will block cutaneous branches that have emerged more proximal in the forearm and that extend to the hand.[46]

Ankle Block

Anesthesia of the foot is easily accomplished with blocks of the sensory nerves at the ankle (Fig. 17-3). Using a 25-gauge needle, the deep peroneal nerve, providing sensation between the great and second toes, is blocked with 5 mL of 1% lidocaine between the tendons of the tibialis anterior and the extensor hallucis longus at the level of the medial and lateral malleoli. The needle may be passed to the bone just lateral to the dorsalis pedis artery. The superficial peroneal nerve is injected with 5 mL of 1% lidocaine with a superficial ring block between the injection of the deep peroneal nerve and the medial malleolus. This will block sensation to the medial and dorsal aspects of the foot. The posterior tibial nerve is injected with 5 mL of 1% lidocaine just posterior to the medial malleolus, adjacent to the posterior tibial artery. As the needle is slowly advanced toward the nerve, eliciting a paresthesia by slightly contacting the nerve with the needle will increase the likelihood of success of the block.[31] Posterior tibial nerve distribution includes the heel and plantar foot surface. Paresthesias are followed with a slight withdrawal of the needle prior to injection. The sural nerve, providing sensation to the posterolateral foot, is blocked with 5 mL of 1% lidocaine between the lateral malleolus and the Achilles tendon, followed by a subcutaneous infiltration originating from this site and over the lateral malleolus.[46]

A



B

Figure 17-3. Landmarks for ankle block. (Photo by Bryan L. Frank, MD.)

Trigger Point Injections

Persons who suffer from neck and shoulder or lower back strain may benefit greatly from deactivation of myofascial trigger zones (Fig. 17-4). The pain relief may be profound and may enable an adventure to continue without disruption. Travell and Simons have extensively described primary and secondary painful points and their referral patterns. Myofascial pain may be intense and may be referred to a large zone of the body.[51] Successful deactivation of trigger zones may be accomplished with either dry needling (acupuncture) or injection of 1% lidocaine. In the absence of anesthetic or acupuncture needles, trigger point deactivation may be accomplished with a 27-gauge needle.

Injection of a trigger zone is typically performed directly into the painful myofascial point and in a four-quadrant zone from the center of the trigger point. As the needle is advanced in the central point, 0.5 mL of anesthetic is injected and then the needle is withdrawn to where the needle tip is just subcutaneous, and it is then advanced 1 to 2 cm into the adjacent tissue at a 45° to 60° angle from the skin surface in each quadrant from the center point, with 0.5 mL of anesthetic injected at each quadrant. Muscle twitches or fasciculations may accompany the injections, but they need not be sought. A volume of 2 to 3 mL of 1% lidocaine is usually ample for each trigger zone. There is no benefit to adding corticosteroids to the anesthetic. Using an acupuncture needle (or a 27- or 30-gauge needle) may deactivate the trigger zones nicely using a similar four-quadrant pecking of the myofascial zone, without injection of anesthetic. In this technique, the acupuncture needle is simply advanced centrally into the trigger zone and then briskly advanced and retracted several times. Then, as with the injection technique, the needle is partially withdrawn so that the needle tip is just subcutaneous, and then it is redirected to each of the four quadrants around the central point, briskly pecking in each quadrant as at the central point. A 1- to $1^1/_2$-inch needle may be used to peck briskly several times in each direction, and also at a 45° to 60° angle from the center of the trigger zone. Depth of insertion is typically 1 to 2 cm. Soiled skin should be prepared as for an intramuscular injection.

Intravenous Regional Anesthesia by Bier Block

The Bier block may provide sufficient anesthesia for a physician unfamiliar with the anatomy and technique of a more sophisticated proximal nerve block to stabilize an arm or a leg fracture (Fig. 17-5). Because a tourniquet is necessary, a Bier block should not be used for longer than 60 minutes.

A vein of the hand or foot on the extremity to be blocked is cannulated with a 20- to 22-gauge intravenous or butterfly catheter, which is then capped and taped in place. The extremity is then raised above the level of the heart for 1 to 2 minutes to diminish the volume of blood in the distal limb. If tolerated, an Esmarch (rubber) or elastic bandage is wrapped from the hand or foot toward the proximal arm or leg to exsanguinate the limb. If a compression bandage is not tolerated, adequate exsanguination may occur by elevating the extremity for 5 to 10 minutes, or by applying an inflatable splint. A tourniquet is applied and the pressure is held at 50 to 100 mm Hg above systolic blood pressure. In the wilderness, a blood pressure cuff would be an effective tourniquet, or a strap, rope, or tubing may be used. The limb is then placed horizontally for the injection, using 25 mL of 1% lidocaine for the upper extremity and 35 mL for the lower extremity. The anesthetic is slowly infused through the previously inserted intravenous or butterfly catheter at a rate of approximately 0.5 mL/sec. Caution should be taken to avoid extravasation of the anesthetic into the surrounding tissues.

Anesthesia will ensue over the first 10 to 15 minutes as the lidocaine diffuses from the intravascular space and binds to the soft tissue. It is important to discontinue the injection if the tourniquet pressure is not maintained during the injection to prevent anesthetic toxicity from an unrestricted intravenous bolus injection. The tourniquet should remain inflated for 60

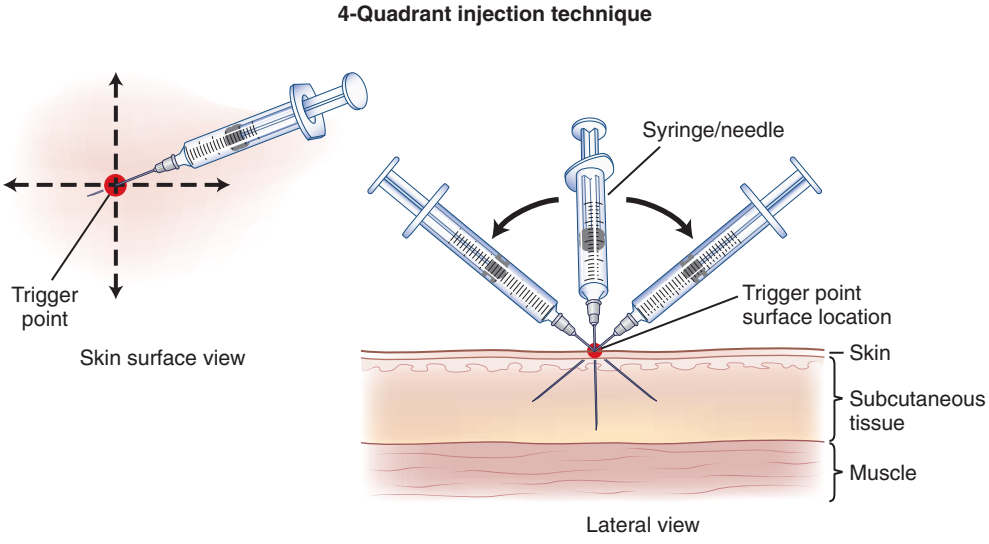


Figure 17-4. Trigger point injections.

Figure 17-5. Preparation for intravenous regional block using field supplies. (Photo by Bryan L. Frank, MD.)

minutes maximum, at which time most of the anesthetic should be protein bound in the soft tissues. The tourniquet may then be released on a single occasion, whereupon the anesthetic effect will diminish within 5 to 10 minutes.

Adjuvant Pharmaceuticals

Pharmaceutical agents other than analgesics and local anesthetics may offer significant relief and may avoid or decrease the potential adverse effects of analgesics and anesthetics.

Antidepressants

Wilderness travelers may be taking antidepressant medications for chronic pain or psycho-emotional dysfunction. Continuation of these medications is important to avoid intensification of pain or psycho-emotional lability. Chronic pain conditions that may benefit from antidepressants include migraine cephalgia, postherpetic neuralgia, and diabetic neuropathy. The tertiary amine tricyclic drugs, such as amitriptyline, imipramine, doxepin, and clomipramine, are commonly used for pain problems. Antidepressant medications are presumably effective for treating chronic pain problems because they block presynaptic reuptake of serotonin and/or norepinephrine by the amine pump.[2] The drugs are rapidly absorbed after oral administration, although this may be decreased by antimuscarinic effects. They are highly protein bound and have half-lives of 1 to 4 days. They are generally oxidized by the hepatic microsomal system and conjugated with glucuronic acid. Elimination occurs via urine and feces.[25]

Side effects may include dry mouth, urinary retention, constipation, and hyperactivity.[20] Photosensitivity may place wilderness travelers at risk for sunburn, and orthostatic hypotension may lead to falls. Patients with overdoses present with excessive sedation, anticholinergic effects on the cardiac conduction system, significant hypotension, respiratory depression, arrhythmias, and coma.

Anticonvulsants

Of the heterogeneous group of drugs classified as anticonvulsants, nine have been useful as adjuvant medications in pain management: phenytoin, valproic acid, topiramate, levetiracetam, lamotrigene, neurontin, pregabalin, carbamazepine, and clonazepam. Their chemical structures bear little relationship to each other. Serum levels have not been clearly established as useful parameters in pain management. The mechanism of action of each of these drugs is unique and varies from altering sodium, calcium, and potassium flux, to enhancing GABA activity or binding.[25]

Side effects vary but may include nausea, vomiting, and CNS effects such as drowsiness, ataxia, and confusion. It is important to rule out drug effects as a factor in wilderness-related illnesses. It is not recommended that these drugs be used for acute management of pain in the wilderness.

Antihistamines

Antihistamines may be useful as adjuvants in a wilderness travel setting. Specifically, hydroxyzine seems to provide an additive

TABLE 17-4. Skeletal Muscle Relaxants

DRUG	DOSAGE (mg)	INTERVAL (hr)	RISKS, PRECAUTIONS
Baclofen	3–5 start, increase 5 mg every 3 days	8	Dizziness, ataxia, confusion, severe abrupt withdrawal
Carisoprodol	350	4–6	Dizziness, ataxia, headache, tremor, syncope, severe abrupt withdrawal, contraindicated with acute intermittent porphyria
Chlorphenesin	400	6–8	Confusion, headache, dizziness (8 weeks or less); avoid in patients with hepatic disease
Chlorzoxazone	250–750	4–6	Dizziness, paradoxical stimulation, rare severe hepatotoxicity
Cyclobenzaprine	10	12–24	Anticholinergic; may cause hypotension, arrhythmias; avoid with MAOI use and in patients with hypertension, CHF, arrhythmia, glaucoma, urinary retention, severe depression, or suicide attempts
Diazepam	2–10	6–8, variable	Dizziness, paradoxical stimulation, cardiopulmonary depression; use only in patients with severe anxiety
Metaxalone	800	6–8	Hepatic impairment, rash, dizziness, headache; avoid with drug-induced anemia
Methocarbamol	1000–1500	4–6	Dizziness, headache, rash
Orphenadrine	100	8–12	Anticholinergic; headache, dizziness

CHF, congestive heart failure; MAOI, monoamine oxidase inhibitors.

analgesic effect in combination with narcotic analgesics, while also lending antiemetic and sedative properties. Hydroxyzine may cause ataxia and disinhibition, and it has, rarely, been associated with convulsions in high dosages.[25] A dosage of 50 to 100 mg IM may improve clinical pain management. Similarly, oral antihistamines, such as diphenhydramine 25 to 50 mg, may provide useful adjuvant effects.

Muscle Relaxants

Muscle relaxants (Table 17-4) are a diverse group of drugs with similar clinical effects but different pharmacologic properties. They are not true skeletal muscle relaxants in the sense of blocking neuromuscular transmission. Rather, they act by depressing reflexes in a general fashion in the CNS. They are indicated for the relief of muscle spasm related to acute, painful, musculoskeletal injuries. Side effects of these centrally acting muscle relaxants include decreased alertness, motor coordination, and physical dexterity, as well as nausea, vomiting, and abdominal pain. Recent studies have shown muscle relaxants to be effective in the management of low back pain. Efficacy is probably increased when muscle relaxants and NSAID analgesics are used in combination.[17]

Common centrally acting muscle relaxants include carisoprodol (Soma, 350 mg PO three times a day to four times a day), metaxalone (Skelaxin, 800 mg per os [PO] three times a day to four times a day), and methocarbamol (Robaxin, 1.5 g PO four times a day for the first 48 to 72 hours, then 1 g PO four times a day). The specific mechanism of action of these agents in humans is unknown, but it appears to be related to centrally acting sedation. Cyclobenzaprine (Flexeril, 10 to 20 mg PO three times a day) is structurally related to the tricyclic antidepressant medications. Diazepam (Valium, 2 to 10 mg PO, IM, or IV) is a benzodiazepine that induces calm and anxiolysis via effects on the thalamus and hypothalamus.[6] Tizanidine (Zanaflex, 8 mg three times a day) is an α-2 agonist with muscle-relaxing properties.

Intra-articular Injections of Hyaluronan Substances

Travelers with degenerative osteoarthritis (OA) of the knees may find hiking, trekking, and other wilderness activities very painful, reducing their ability to participate in these activities. However, some may engage in these activities and experience exacerbation of their symptoms while in a remote or wilderness setting. These exacerbated symptoms may impact the pleasure and safety of the travelers and their companions.

OA is felt to be related to decrease in the amount or quality of hyaluronan. Synvisc is a gel-like mixture of hylan A and hylan B polymers (derived from the hyaluronan of chicken combs) and saline. Injection of this "drug-free" hylan complex has proved to be useful to many sufferers of OA, with significant reductions in oral analgesic use. Studies have not demonstrated efficacy in less than a series of three weekly injections; thus, immediate response to injection in the wilderness setting should not be expected.[66]

HIV/AIDS—SPECIAL WILDERNESS CLINICAL CONSIDERATIONS

With advances in treatment of patients with acquired immunodeficiency syndrome (AIDS) and human immunodeficiency virus (HIV) infection, many are pursuing more activities and living healthier lives than a decade ago. These activities include travel into wilderness and remote locations. For many of these travelers, HIV/AIDS is undertreated and misdiagnosed. An estimated 85% of HIV/AIDS patients receive inadequate therapy, which can significantly reduce their quality of life and options for wilderness travel.[68]

Patients with HIV/AIDS commonly experience pain, which may include headaches, oropharyngeal pain and odynophagia, earaches, abdominal and chest pain, myalgias, and arthralgias. New or unexplained pains should initiate a medical investigation so that pain therapeutic agents do not mask emerging associated illnesses. Associated diseases that may lead to pain in these patients include neuropathies, postherpetic neuralgia, avascular necrosis of the hip, osteopenia, myopathies, renal calculi, herpes simplex, candida esophagitis, and pancreatitis. Additionally, various diagnostic and therapeutic procedures may initiate or increase pain for these patients. As seen in the peripheral neuropathies of patients with HIV/AIDS, the virus may attack the nerve endings in the extremities, with resulting

burning, numbness, and dysesthesias. This is often referred to as distal symmetrical polyneuropathy (DSP), and it may prevent or complicate these patients' wilderness endeavors. Additionally, therapeutic medications may also lead to pain in patients with HIV/AIDS, as is seen with didanosine, dicalcitabine, isoniazid, Videx (ddI), and Zerit (d4T).[67,68]

Treatment of pain in patients with HIV/AIDS commonly includes oral non-narcotic and narcotic medications. Aspirin, acetaminophen, and NSAIDs are commonly utilized for mild to moderate pain, whereas opioids are the primary therapeutics for severe pain. Many physicians are reluctant to prescribe narcotics to patients because of potential addiction, studies show the risk for addiction in patients with HIV/AIDS to be small. Longer-acting medications, including methadone and time-release morphine, may offer greater pain management and allow increased wilderness activities. The fentanyl patch transdermal system providing medication release over three days may significantly help those who poorly tolerate oral administration.

Adjuvant medications, including caffeine, antihistamines, anticonvulsants, and antidepressants, should be considered so that significant pain relief can be obtained with lower narcotic dosages and thus lower opioid side effects. Persons with a narcotic addiction either prior to or subsequent to narcotic pain management will need to have very strict limits and guidelines set by a specialist in addiction medicine.

Drug interactions are of concern, as various anti-HIV medications may interfere with narcotic medications. Ritonavir (Norvin) increases levels of meperidine, propoxyphene, and fentanyl, and efavirenz and nevirapine lower the methadone level. Phenytoin lowers the methadone level and NSAIDs increase the lithium level. These medication interactions are also important to consider for over-the-counter medications, for botanical and herbal remedies, and for illicit drugs as well. Tobacco smoking shortens the half-life of NSAIDs and increases the metabolism of meperidine, morphine, and propoxyphene. Cannabis is known to increase the effect of morphine, ritonavir increases the level of MDMA (methylenedioxymethamphetamine; Ecstasy) and alcohol increases the abacavir (Ziagen) level.[67]

COMPLEMENTARY AND ALTERNATIVE MEDICINE THERAPIES

Complementary and *alternative* are terms used to denote therapies and modalities that may not be supported by Western-designed prospective, randomized studies, that are not commonly taught in U.S. medical colleges, or that are not generally covered by traditional health insurance plans.[55] Some of these therapies can provide a significant contribution to the management of pain in the wilderness.

Acupuncture

Acupuncture developed over the past 3 to 5 millennia in Asia, and it has been practiced over the past several hundred years in the Western world. In many cases, acupuncture developed in geographic areas and under social conditions very similar to the primitive or undeveloped conditions familiar to wilderness travelers. The use of acupuncture by physicians to treat trauma and illness in wilderness settings has been described recently in medical literature.[22,24] Properly administered, acupuncture should have a very low risk of morbidity and may be extremely effective in alleviating pain and even restoring function to an injured wilderness traveler (Fig. 17-6 through Fig. 17-9).

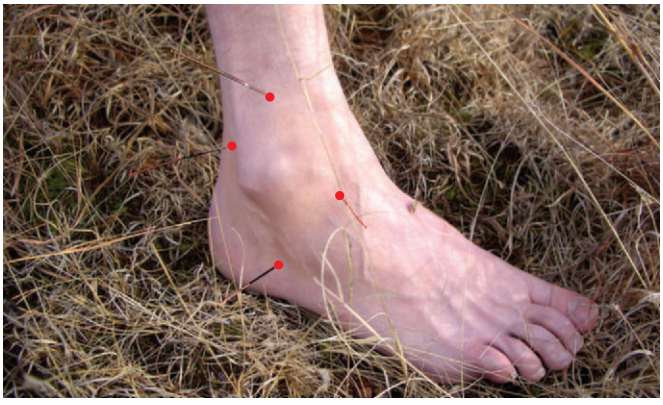


Figure 17-6. Typical acupuncture needle placement for lateral ankle strain. (Photo by Bryan L. Frank, MD)

Figure 17-7. Typical acupuncture needle placement around injured knee. (Photo by Bryan L. Frank, MD.)

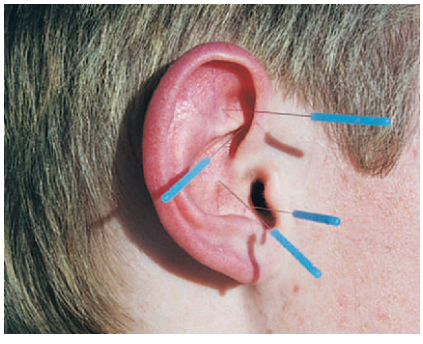


Figure 17-8. Patient with auricular needles in place. (Photo by Bryan L. Frank, MD.)

Stux and Pomeranz, and others, have demonstrated endorphin, enkephalin, monamine, and adrenocorticotropic hormone (ACTH) release with acupuncture stimulation.[49] Furthermore,

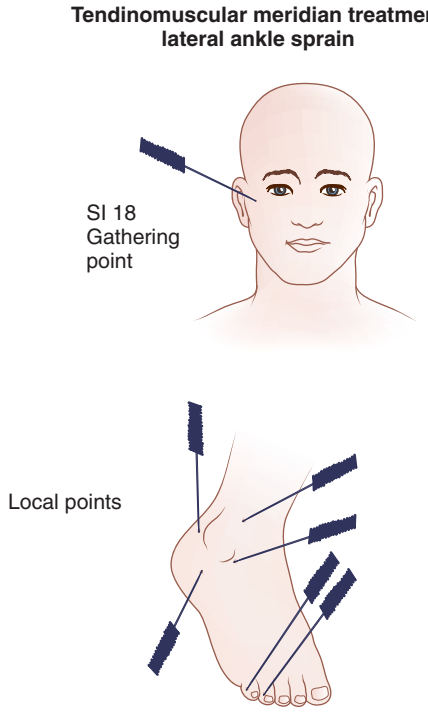


Figure 17-9. Tendinomuscular meridian treatment for lateral ankle sprain. (From Frank BL: Medical acupuncture in wilderness and Third World settings. Wilderness Med Lett 14:1, 1997.)

Box 17-3. Pain Management First Aid Kit*

BASICS
Esmarch bandage, 3 × 36 inches
Tourniquet
Hot and cold gel packs

ORAL MEDICATIONS
Acetylsalicylic acid (ASA), 500 mg
Acetaminophen, 500 mg
Carisoprodol (Soma), 350 mg, or metaxalone (Skelaxin), 500 mg
Diazepam (Valium), 5-mg tabs
Hydrocodone, 5-mg tabs

INJECTABLE MEDICATIONS
Naloxone, two ampules, 0.4 mg each
Ketamine, 50 mg/mL, 5 to 10 mL
Lidocaine 1%, 30 mL
Procaine 1%, 30 mL
Midazolam (Versed), 5 mg/mL, 5-mL vial
Morphine, 5 mg/mL, 5 mL
Meperidine (Demerol), 50 mg/mL, 5 mL

TOPICAL THERAPIES
Arnica cream, 5-mL tube
Capsaicin ointment, 2-g tube
TAC (mixture of tetracaine [0.5%], adrenaline [1:2000], and cocaine [11.8%] in saline), 10 mL†
Biomagnets (200 to 800 gauss), two to four small

ADDITIONAL SUPPLIES
Intravenous (IV) cannula, 20-gauge, three
IV cannula, 18-gauge, three
IV tubing, two
Normal saline, 500 mL
IV D_5LR (5% dextrose in lactated Ringer's), 500 mL
Acupuncture needles, 50

*Items subject to training and scope of practice. This pain kit is in addition to regular first-aid kit.
†EMLA (2.5% lidocaine and 2.5% prilocaine) should be used if there is a concern for cocaine toxicity.

the gate theory of pain modulation and altered sympathetic activity[33] may apply as well. Clinically, improved microvascular circulation may lead to decreases in tissue edema, which in turn may diminish pain and aid in restoring function. Release of ACTH leads to increased circulating corticosteroids; decreased inflammation may contribute to decreased pain and improved healing.[30]

Contemporary medical acupuncturists are typically trained in a variety of styles or traditions. Many physicians utilize a combined approach of acupuncture point selections based on neuroanatomy and those that are felt to have energetic effects in the body. Additionally, acupuncture microsystems are often employed, in which the entire body is represented in a small area such as the ear, scalp, or hand. These microsystems are often quite beneficial for acute pain relief (see Figure 17-8).[24]

Sterile acupuncture needles are compact, lightweight, and easy to include in a daypack or first-aid kit (Box 17-3). Integration of acupuncture into the biomedical care of wilderness trauma, pain, and illness may dramatically enhance patient comfort and facilitate extrication from a remote setting.

For example, a common clinical case that often responds to acupuncture is a sprained ankle. An energetic style of acupuncture that is especially useful for common trauma utilizes the tendinomuscular meridians (TMMs) of the acupuncture energetic subsystems. The indications for activation of the TMMs treatment include acute strains, sprains, abrasions, and hematomas. The method of activation for this style of treatment is to place a needle in the "Jing-well" or "Ting" point of one to three of the meridians involved in the lesion. This is followed by placing a needle in the "gathering point" for the meridians, then by placement of needles around the area of induration, swelling, or bruising, approximately 1 cm out from the edge. All needles in this treatment are placed only 2 to 3 mm deep and are left in place for 20 to 45 minutes.

Commonly, a lateral ankle sprain involves both the gallbladder and bladder TMM zones. The Ting (Jing-well) points for these meridians are at GB 44 and BL 67 on the lateral angles of the fourth and fifth toes, respectively. The gathering point for these meridians is at SI 18, just below the zygoma in line with the lateral canthus. Locally, four to six needles are typically placed around the swelling or ecchymosis of the ankle sprain (see Figure 17-9). Recovery from the sprain may proceed much more rapidly with this acupuncture input than with the conventional therapies of rest, ice, compression, and elevation (RICE) alone. Failure to achieve 70% to 85% or greater decrease in pain over 24 to 48 hours may alert the medical

acupuncturist that an injury is more serious than initially appreciated.

Most acupuncture treatment requires substantial training to be responsibly integrated with conventional Western therapies. National and international standards of training have been established for Western-trained physicians who desire to incorporate acupuncture into their traditional medical practices. The American Academy of Medical Acupuncture (AAMA) is the professional organization representing U.S. physician acupuncturists whose training meets or exceeds standards established by the World Health Organization's affiliate, the World Federation of Acupuncture and Moxibustion Societies. Physicians interested in learning acupuncture may contact the AAMA (www.medicalacupuncture.org) for information on training programs designed specifically for physicians. The National Commission for Certification of Acupuncture and Oriental Medicine provides testing and resources primarily for U.S. nonphysician acupuncturists.[23] The International Council of Medical Acupuncture and Related Techniques (ICMART) is composed of approximately 84 physician acupuncture organizations from around the world and provides international educational congresses and symposia.

Herbal and Botanical Remedies

The term *herb* is broadly defined as a nonwoody plant that dies down to the ground after flowering. The term *botanical* is a more general description of flora, although common interpretation describes any plant used for medicinal therapy (see Chapter 60), nutritional value, or food seasoning or dyeing (coloring).[14] The use of botanicals, like the use of acupuncture, may be encountered in wilderness travels as a part of the indigenous culture and medical care. Botanicals are often prepared as infusions, with the plant's soft portions placed in a pot and covered by boiling water to create a supernate. Herbal decoctions are traditionally prepared in special earthen crocks or in containers of stainless steel, ceramic, or enamel by contemporary practitioners, specifically avoiding aluminum and alloyed metal pots. The herb is placed in the container and covered with cold water that is then boiled, covered, and simmered. Travelers may encounter an herbal remedy intended to be applied to the skin, dispensed as a poultice, a botanical liniment, an ointment, or an oil. Alternatively, many modern herbalists utilize capsule forms of herbs for ease of administration. Combinations of several herbs are often more effective than a single herb, and common formulas have been recorded worldwide for centuries.[33] Appropriate application or prescription of botanical products rarely leads to toxicity or adverse reactions, although such are possible if botanicals are used excessively or carelessly.

Botanical products that have been used for pain include morphine, isolated from the opium poppy, and cocaine, from coca leaves *(Erythroxylum coca)*. Often used as a seasoning or food, oregano *(Origanum vulgare)* has been reported to be beneficial for rheumatic pain.[33] Sunflower *(Helianthus annuus)* is a source of phenylalanine, useful for general pain. Turmeric *(Curcuma longa)* contains curcumin, an anti-inflammatory substance that is beneficial for rheumatoid arthritis, and ginger *(Zingiber officinale)* is beneficial for rheumatoid arthritis, osteoarthritis, and fibromyalgia. Clove *(Syzygium aromaticum)* is endorsed by the German botanical resource, Commission E, topically for dental pain, and red peppers (*Capsicum* species) contain substance P–depleting capsaicin and also salicylates. Often taken as an infusion or decoction, kava kava *(Piper methysticum)* contains both dihydrokavain and dihydromethysticin, which have analgesic effectiveness similar to that of aspirin. Evening primrose *(Oenothera biennis)* is a great source of tryptophan and has been demonstrated to relieve pain associated with diabetic neuropathy. Lavender (*Lavandula* species) contains linalool and linalyl aldehyde, which appear to be useful, in topical and aromatherapy form, for pain of burns and other injuries.[14]

Willow (*Salix* species), which has been used to treat pain since 500 BC, contains salicin and other salicylate compounds. Commission E has recognized willow as an effective pain reliever for headaches, arthritis, and many other pains.

Other salicylate-containing plants include wintergreen and birch bark. All botanicals containing salicylates should be avoided in persons who are sensitive or allergic to aspirin products. Furthermore, children who have viral infections such as a cold or influenza should avoid these products, as salicylates have been implicated in the development of Reye's syndrome.[14]

Chamomile *(Matricaria chamomilla)* contains chamazulene, which is reportedly beneficial for abdominal pain related to GI spasm or colic. As an antihistaminic, it has mild calming or sedative properties. It is used in Europe to treat leg ulcers and may be beneficial for painful, irritated bites and stings.[14] Plantain major *(Plantago major)* is also commonly useful for bites and stings, poison ivy discomfort, and toothache, and it has been used traditionally by Native Americans as a wound dressing. Aloe gel *(Aloe vera)* has been used since ancient times to treat burns and sunburn and to promote wound healing.

Especially useful for sprains and strains is the mountain daisy or arnica *(Arnica montana)*, which is also endorsed by the German Commission E. Arnica was in the U.S. Pharmacopoeia from the early 1800s to the 1960s, and it has long been used by Native Americans and others for relieving back pain and other myofascial pains and bruising. It is used topically or internally, often in homeopathic form. Comfrey *(Symphytum officinale)* has been used since ancient Grecian times for skin problems.[15] It contains alloin, which is anti-inflammatory, and is endorsed by Commission E to topically treat bruises, dislocations, and sprains. Comfrey has experienced a controversial safety record because oral ingestion of its pyrrolizidine alkaloids has been associated with hepatotoxicity and carcinogenicity.[14] For this reason, only topical use of comfrey is recommended.

Magnet Therapy

Magnet therapy has been described for approximately 4000 years in the Hindu Vedas and the Chinese acupuncture classic, *Huang Te Nei Ching*. The application of magnetic stones, or lodestones, is said in ancient legends of Cleopatra and others to have decreased pain and preserved youth. Early Romans used the discharge of the electric eel to treat arthritis and gout.[35]

Danish physicist Oerstad proved in 1820 that an electric current flowing through a wire had its own magnetic field. (In modern medicine, the most familiar use of magnetic energy is in magnetic resonance imaging.) There are various theories about the effectiveness of permanent magnets for use in pain and healing. Lawrence and coworkers reported increased blood circulation and increased macrophage activity,[35] and others have hypothesized effects on peripheral nerves, including blockage or modification of sensory neuron action potentials and enhanced regeneration of peripheral nerves.[36,40,43,54]

Most permanent magnets marketed at this time for management of pain are approximately 200 to 1200 gauss in strength. At this level, there is little, if any, risk in trying a magnet for

pain reduction, assuming the patient is properly evaluated to provide other care when appropriate. This author has seen the benefits of magnets in his pain medicine practice over many years. Many patients report significant pain reduction within hours; others report relief after wearing the magnets for a week or more. In wilderness travel, it is reasonable to carry a few therapeutic magnets, as they are usually lightweight, compact, and unbreakable. Magnets marketed for pain therapy range in size from a 2-inch (5 cm) circle to a 6- by 12-inch (15–30 cm) rectangular pad. Most are only a few millimeters thick, and they are often flexible. These are becoming more readily available through multilevel marketing, television infomercials, health food stores, and the internet. Some people have also experienced pain relief with simple, small magnets such as those used to place notes and photographs on a refrigerator. The magnet should be placed directly over the area of pain, and it can be held in place with tape, clothing, or straps.

The references for this chapter can be found on the accompanying DVD-ROM.

18 Bandaging and Taping

Daniel Garza

Taping and bandaging are both useful skills in wilderness medicine. Taping can be used to support injured joints and soft tissues; bandaging is most often used to secure a wound dressing (Fig. 18-1). Bandaging with an elastic wrap is an alternative to taping and, over larger joints, such as the knee, is often preferable.

In general, taping requires practice, but some simple techniques can be easily mastered. It is most often utilized in mild to moderate sprains and strains, where some functional capacity such as weightbearing and lifting are maintained. Although taping offers dynamic support, it is in no way comparable to splinting, which can immobilize an extremity. The most common tape applied is white athletic (or adhesive) tape, often used by trainers in organized sports. Athletic tape may be applied to skin, although it may lose adhesion if the body part is not shaved and tape adhesive not applied.

Some keys to successful taping include the following:

- Avoid leaving any gaps in the tape because these will lead to blisters.
- Avoid excessive tension on tape strips that serve to fill these gaps.
- Apply tape in a manner that follows the skin contour to avoid wrinkles.
- Try to overlap a half-width on successive strips.

Bandaging is accomplished by either elastic wraps or gauze rolls of various widths. Once a dressing is applied to a wound, appropriate bandaging allows the patient to feel confident that it will remain secure throughout reasonable amounts of activity.

Regardless of the method used, it is important to remember that taping and bandaging, especially when circumferential, should not be so tight as to limit circulation. Signs and symptoms of overly tight application are similar to a mild compartment syndrome, classically characterized by the five P's:

Pain
Pallor
Paralysis
Pulselessness
Paresthesias

TAPING

Types of Tape

Athletic tape is composed of fibers woven into strips that are coated with zinc oxide, an adhesive compound. Although most commonly colored white, athletic tape is available in a variety of colors. This is the most commonly used tape in athletics and first aid for support and prevention of injury. It is available in a variety of widths. Although the major advantage of athletic tape is versatility, its major disadvantage is the tendency of zinc oxide to lose adhesive properties with heat and moisture, thus resulting in loss of support when the patient sweats. There are a variety of techniques used to increase the durability of athletic tape under these conditions, described later in this section.

Elastic tape (e.g., Elastikon by Johnson & Johnson) is cotton elastic cloth tape with a rubber-based adhesive. The elasticity of the tape allows for greater flexibility and is particularly useful for large joints such as the knees or shoulders.

Skin Preparation

Skin preparation involves measures meant to increase longevity of tape adhesion and patient comfort. If tape is to be applied directly to the skin, the area is usually shaved to remove hair that may interfere with direct contact. Care must be taken to avoid small abrasions in the skin when shaving because these can serve as sites of infection. If the area cannot be shaved in a clean and deliberate manner, it may be advisable to avoid. Any

pain reduction, assuming the patient is properly evaluated to provide other care when appropriate. This author has seen the benefits of magnets in his pain medicine practice over many years. Many patients report significant pain reduction within hours; others report relief after wearing the magnets for a week or more. In wilderness travel, it is reasonable to carry a few therapeutic magnets, as they are usually lightweight, compact, and unbreakable. Magnets marketed for pain therapy range in size from a 2-inch (5 cm) circle to a 6- by 12-inch (15–30 cm) rectangular pad. Most are only a few millimeters thick, and they are often flexible. These are becoming more readily available through multilevel marketing, television infomercials, health food stores, and the internet. Some people have also experienced pain relief with simple, small magnets such as those used to place notes and photographs on a refrigerator. The magnet should be placed directly over the area of pain, and it can be held in place with tape, clothing, or straps.

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obvious abrasion should be covered with a thin layer of gauze or small adhesive strip before taping.

There are a variety of commercially available skin adhesives in aerosolized form. These preparations use benzoin as the adhesive. One example is Cramer's Tuf-Skin. Skin adhesives are applied after the skin has been shaved and abrasions dressed.

If the area is not shaved, a foam underwrap or prewrap is used to protect body hair. Prewrap is generally supplied in 3-inch rolls in a variety of colors. After applying a topical skin adherent, such as Tuf-Skin, prewrap is applied over the part to be taped in a simple, continuous circular wrap. The prewrap is sufficiently self-adherent that it does not need to be taped down.

Heel-and-lace pads and foam pads are used to provide greater comfort by relieving potential pressure points. When tape is applied over bony prominences, it can create tension on the skin surface that leads to blistering. Heel-and-lace pads are prefabricated pieces of white foam that are stuck together with petroleum jelly and then applied to the anterior and posterior aspects of the talus when the ankle is taped. Pads of foam can be cut to size to fit over painful areas that need to be taped, as in medial tibial stress syndrome, or to be used for support in special cases, such as taping for patellar subluxation.

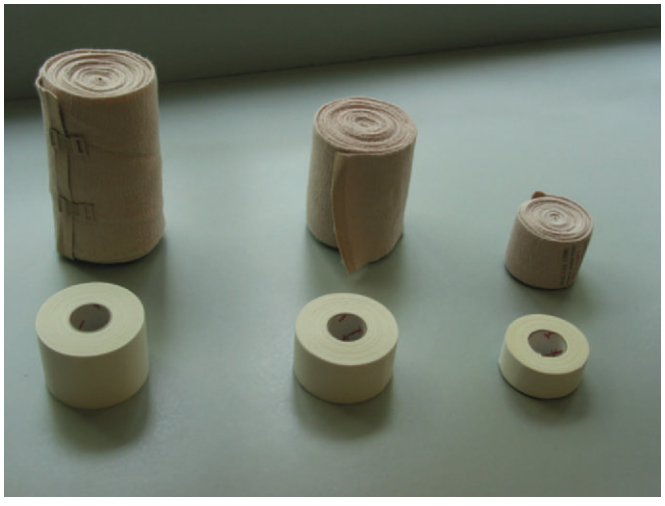


Figure 18-1. Athletic tape (*front row*) and elastic bandages (*back row*) come in various sizes.

Ankle Taping

The most common injury to the lower extremity while hiking is a sprained ankle. It is usually the result of inverting the ankle on an unstable surface. Pain and swelling linger for several days, and taping can help offer support if the patient is able to bear weight. Because most injuries occur to the lateral ligaments, taping supports the lateral surface by restricting inversion. In general, taping of the ankle consists of anchor strips on the lower leg and foot, stirrups that run in a medial to lateral direction underneath the calcaneus, and support from either a figure-8 or heel-lock technique (Fig. 18-2). The heel lock requires some expertise to perform, so most operators are more comfortable with the figure-8 initially.

Toe Taping

Taping toes that are sprained or fractured is simple and effective. This treatment involves "buddy-taping" to the adjacent toe with one or two pieces of tape to provide support. A piece of gauze, cotton, or cloth can be placed between the toes to avoid skin breakdown.

A sprain of the first metatarsophalangeal joint, also known as "turf toe," can be a chronic and painful condition. Taping for turf toe attempts to support and stabilize the joint and is described in Figure 18-3.

Lower Leg Taping

Medial tibial stress syndrome, commonly referred to as "shin splints," can be taped for support and comfort. Tape is brought

A B

Figure 18-2. Ankle taping. **A,** (1) Ankle at 90 degrees; (2) apply anchors of 1.5-inch tape at the lower leg and distal foot. **B,** (3) Apply 3 stirrups from medial to lateral in a slight fan-like projection.

C

D

E

F

G

H

Figure 18-2, cont'd. **C,** (4) Fill in gaps with horizontal strips. **D,** (5) Begin figure 8. Apply tape across front of ankle in left-to-right direction. **E,** (6) Continue under the foot to the opposite side and cross back over the top of the foot. **F,** (7) Complete by wrapping around the leg and end at the anterior aspect of ankle. **G,** (8) Apply heel locks for both feet (omit if not familiar with this technique). Start in left-to-right direction and apply tape across front of joint. **H,** (9) Wrap around the heel (bottom margin of tape should be above the superior edge of the calcaneus) to form the first heel lock.

I

J

K

Figure 18-2, cont'd. **I,** (10) Continue under the foot to the opposite side and cross back over the top of the foot. **J,** (11) The tape is then brought back around the superior margin of the calcaneus and down and around the heel. **K,** (12) Finish by wrapping around the ankle. Repeat figure 8 or heel lock as desired.

from a lateral to medial direction, and a small foam pad can be cut to cover the area of tenderness. Underwrap should be used over a foam pad to secure it in place (Fig. 18-4).

Knee Taping

Because it is a large joint, taping the knee requires expertise and special consideration. Underwrap should not be used because adequate traction to support the joint can only be achieved by taping directly to the skin. The patient's knee should be shaved 6 inches above and below the joint line. In addition, standard athletic tape should not be used because it cannot provide enough support. Three-inch elastic tape provides the foundation. Taping for injuries to the medial aspect of the knee is described in Figure 18-5.

Patella Taping

Subluxation of the patella is exacerbated by the stress of walking long distances across uneven terrain. Incorporating a piece of foam into taping the knee can help relieve symptoms. As with all taping around the knee, underwrap should not be used (Fig. 18-6).

Finger Taping

Injuries to the fingers are common in a variety of outdoor settings. Both simple fractures and sprains can be initially treated by taping. The most common scenarios involve fingers that are hyperextended or that are "jammed." Injuries in this scenario are often to the palmar ligaments and tendons. Patients may find it difficult to flex the finger against the resistance of an examiner's finger or may demonstrate tenderness over the palmar aspect of the finger. Swelling is almost always present and may be difficult to localize. This presentation is also seen after reduction of a dorsal dislocation of the proximal interphalangeal joint. In all these cases, it is always best to splint or tape the finger in slight flexion to avoid further injury to the flexor apparatus.

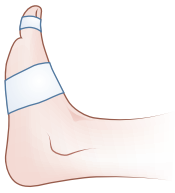
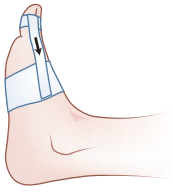
1. Apply two anchors: First anchor around IP joint of first toe with 1-inch tape. Second anchor around midfoot with 1½-inch tape.	
2. Apply strip of 1-inch tape from distal to proximal anchor along medial aspect.	
3. Continue with a strip of 1-inch tape from lateral edge of distal anchor along plantar aspect of first MTP joint to medial aspect of proximal anchor.	
4. Cross with a strip of 1-inch tape extending from the medial aspect of the toe to the plantar aspect of the proximal anchor.	
5. Begin dorsal strips by applying 1-inch tape from medial aspect of distal anchor across the dorsal aspect of the MTP joint to the proximal anchor.	
6. Cross over the previous strip by applying 1-inch tape from lateral aspect of distal anchor to medial aspect of proximal anchor.	
7. Close with 1-inch strips around the toe and 1½-inch tape around the forefoot.	

Figure 18-3. Toe taping.

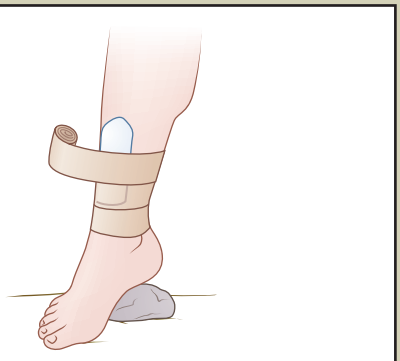
1. (Optional) Underwrap is applied over a foam pad.	
2. With the patient placing his or her heel on a rock or roll of tape, begin applying 1½-inch tape from the superior margin of the malleoli to the calf.	

Figure 18-4. Lower leg taping.

Fingers are buddy-taped to the adjacent finger as a natural splint (Fig. 18-7). The second and third fingers and fourth and fifth fingers are always paired. If the third and fourth fingers are paired, this makes injury to the second and fifth fingers more likely with subsequent activity.

A small piece of gauze, cotton, or cloth should be placed between the fingers to avoid blistering or pressure on a tender joint. Strips of tape should be applied around fingers but not over the joints.

Although not as common, injuries to the extensor tendons can occur. Typically these occur with hyperflexion, but they can also occur with hyperextension and axial loading.[1]

A mallet finger results from fracture of the base of the distal phalange, the site of attachment for the extensor tendon. The resulting inability of the distal phalange to extend fully results in a partially flexed "mallet" finger. Injuries in which the extensor mechanism is clearly disrupted should be treated with the finger taped in full extension. Often a straight splint, such as a tongue blade or smooth stick, can be placed on the dorsal surface and the finger taped to it for additional extensor support (Fig. 18-8).

Any injury to the fingers or hands should always be evaluated by a physician, who can determine whether radiographs are necessary. Given the importance of maintaining optimal function of the hands for one's personal and professional activities, this point cannot be overemphasized.

Thumb Taping

The thumb is frequently injured when placed in extreme extension or abduction, such as occurs when it is caught in the strap of a ski pole when falling. Taping can prevent reproducing the mechanism of injury, particularly when grasping an object (Fig. 18-9).

Wrist Taping

Wrist sprains generally occur during falls and initially can be difficult to distinguish from fractures. Although splinting is initially the most desirable treatment, there are two basic taping approaches that can be used, depending on the nature of the injury. As with the finger, the most important factor is whether the injury occurred in hyperextension or hyperflexion. Anchors are placed around the palm and distal wrist, whereas support strips to prevent undesirable movements are placed on the palmar aspect for hyperextension injuries or dorsal aspect for hyperflexion injuries (Fig. 18-10).

Elbow Taping

The two most common soft tissue injuries to the elbow result from hyperextension and from excessive valgus force. Each of these injuries can result in significant ligament and tendon injury. Taping techniques are meant to prevent reproducing painful movements while maintaining function. Because these techniques allow for substantial joint movement, underwrap

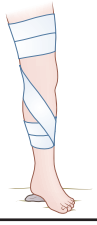
1. The patient maintains the knee in slight flexion (10–15 degrees) by placing the heel on a small stone or cap of a spray can.	
2. Apply two anchor strips of 3-inch elastic tape 6 inches above and below the joint line.	
3. Apply a strip of 3-inch elastic tape from the anterolateral aspect of the lower leg, across the knee joint and up to the posteromedial aspect of the thigh.	
4. Apply a second strip from posterior calf to anterior thigh, forming an X.	
5. Repeat steps 3 and 4 twice.	
6. Apply two additional anchor strips of 3-inch elastic tape 6 inches above and below the joint for closure.	
7. (Optional) Wrap a 6-inch elastic bandage from mid-calf to mid-thigh to cover the tape and provide additional support.	

Figure 18-5. Knee taping.

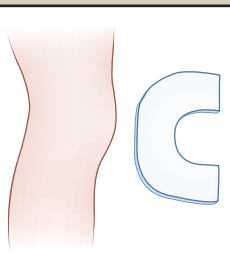
1. Cut a piece of foam into a C shape, measured to encircle half of the patient's patella.	
2. The patient maintains the knee in slight flexion (10–15 degrees) by placing the heel on a small stone or cap of a spray can.	
3. Apply two anchor strips of 3-inch elastic tape 4 inches above and below the patella.	
4. Apply the foam pad cut to fit the patient's patella. Elastic tape (3-inch) is applied in a manner that reproduces the curvature of the foam pad.	
5. Starting from the medial aspect of the lower leg anchors, bring the elastic tape around the lateral aspect of the patella and back to the medial aspect of the upper leg anchors.	
6. (Optional) Wrap a 6-inch elastic bandage from mid-calf to mid-thigh to cover the taping and provide additional support.	

Figure 18-6. Patella taping.

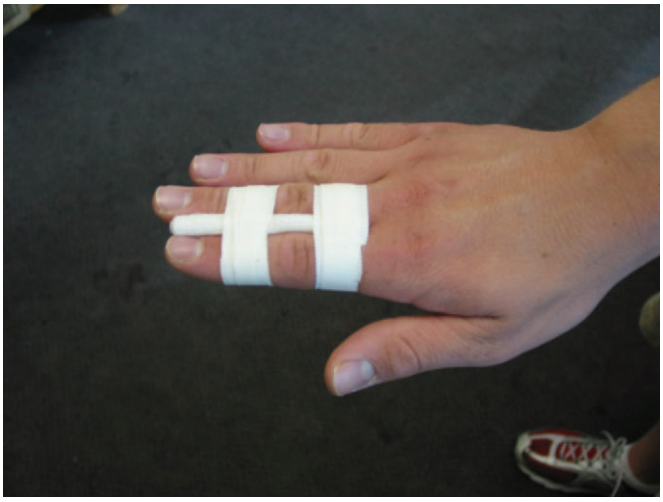
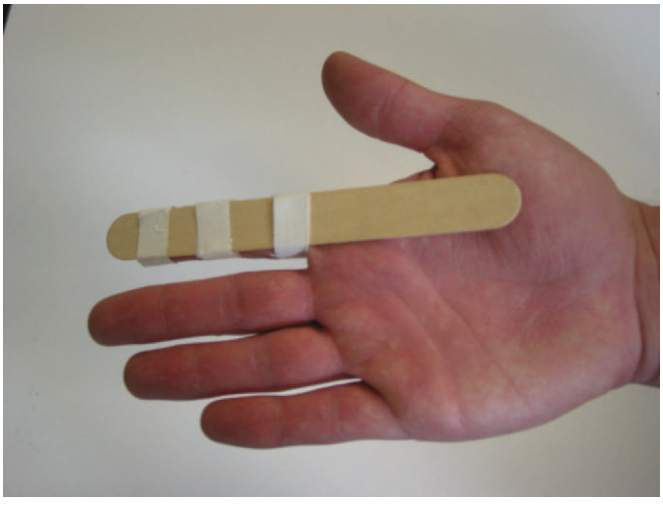


Figure 18-7. Buddy-taping of fingers.

A



B

Figure 18-8. **A and B,** Extension taping of finger with small splint.

A

B

C

Figure 18-9. Thumb taping. **A,** (1) Using 1.5-inch athletic tape, wrap an anchor strip around the wrist. **B,** (2) Using 0.75-inch tape, start at volar aspect and continue along the dorsal aspect of the thumb towards the first web space. **C,** (3) Allow the patient to crimp the tape as it comes across the web space and continue around base of thumb.

D

E

F

G

H

I

Figure 18-9, cont'd. **D,** (4) Bring the tape around to the volar aspect of the wrist and tape at that point. To complete a thumb spica, apply several more strips in succession. To reinforce, rather than repeating a series of strips, continue as follows. **E,** (5) Apply an anchor strip from volar to dorsal aspects of wrist through the first web space (note crimping). **F,** (6) Apply strip from dorsal to volar aspect of anchor strip. **G,** (7) Apply successive strips until at wrist. **H,** (8) Add a finishing anchor strip through first web space. **I,** (9) Complete with anchor strip at wrist.

Figure 18-10. Wrist taping. **A,** (1) With the hand wide-open, apply one anchor across the palm of the hand and two to three anchors across the distal forearm. **B,** (2) Measure out the distance between the two anchors and construct a fan of three strips at varying angles on a smooth surface. **C,** (3) For hyperextension injuries, apply these support strips to the palmar aspect. For hyperflexion injuries, apply them to the dorsal aspect. **D,** (4) Apply another set of anchors over the support strips.

should not be used, and tape should be applied directly to skin to allow for maximal adhesion. Taping for a hyperextension injury employs a fan of elastic or tape, similar to that used in the wrist, to prevent excessive extension (Fig. 18-11).

Individuals who have suffered a valgus stress injury require reinforcement from elastic tape placed on the medial aspect of the elbow (Fig. 18-12).

Shoulder Taping

Taping the glenohumeral joint is rarely done, primarily because it results in so significant a restriction of movement that the patient cannot function effectively. As such, taping offers little advantage over a sling. Taping of the acromioclavicular joint, however, can be effective in reducing pain and maintaining adequate function at the glenohumeral joint.

Injuries to the acromioclavicular joint most commonly occur when a patient falls on the lateral aspect of the shoulder. Enough force is transmitted through the acromioclavicular ligament to stretch or tear it, resulting in a sprain, or "separated shoulder." Tape must be applied directly to the skin (Fig. 18-13).

BANDAGING

Bandaging may be used to wrap and support an injury or to help dress a wound. Many of the techniques described in the section on taping, such as figure-8 patterns, are used in bandaging.

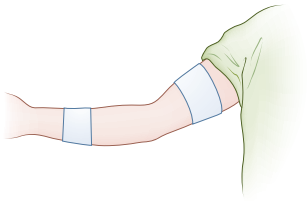
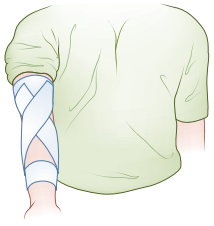
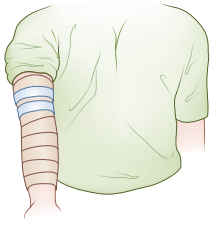
1. The arm is supinated and extended just short of where pain is experienced. Apply anchors with 3-inch elastic tape 4 inches above and below the elbow.	
2. Apply 3-inch elastic tape from distal to proximal anchors that form an X over the antecubital fossa. A total of four strips should be applied.	
3. Apply closure strips above and below the elbow over the original anchors.	
4. (Optional) Apply a 4- or 6-inch elastic bandage from distal to proximal to secure the taping.	

Figure 18-11. Elbow taping.

Types of Bandages

The type of bandage used depends on its purpose. Elastic bandages (e.g., Ace wrap) come in a variety of widths and are used to wrap injuries such as sprains and strains. These bandages generally come with separate clips or clips built into the bandage to secure it. Of note is the double-length 6-inch elastic bandage that is useful for wrapping large joints such as the knee and shoulder.

Bandaging wounds generally involves rolled gauze or cotton-based wraps that secure a dressing in place. These wraps are more desirable than elastic bandages in wound care because they do not place as much tension on the wound dressing. A triangular bandage, which is often used to create a sling, can be folded two to three times into a strap, called a *cravat* (Fig. 18-14). Cravat dressings are useful for applying pressure to a wound that is bleeding to promote hemostasis.

In the discussion of bandaging different parts of the body later in this chapter, the method for using an elastic bandage is described. When securing a wound dressing, the same methods may be used, except that rolled gauze or cotton bandages should be substituted. If there is a special technique for wound care, it will be described separately.

Securing Bandages

Because bandages are not adhesive, they must be secured with tape or clips, or by tying them to the body. Two techniques for tying off a bandage are as follows:

1. As you are finishing wrapping with a bandage, bend the free end backward over your fingers, creating a loop. Now double back around the body part and tie the remaining free end to the loop to secure the bandage[2] (Fig. 18-15).

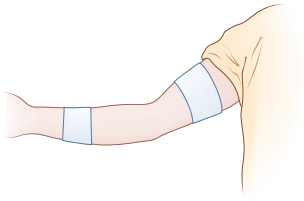
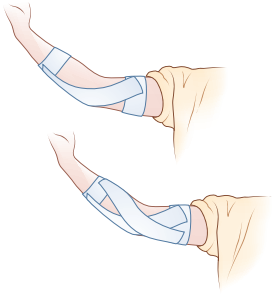
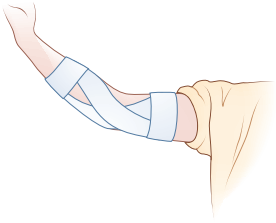
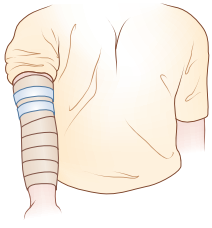
1. The arm is supinated and flexed to approximately 30 degrees. Apply anchors with 3-inch elastic tape 4 inches above and below the elbow.	
2. Apply 3-inch elastic tape that forms an X over the medial joint line. The first strip begins at the posterior aspect of the proximal anchor and crosses to the anterior aspect of the distal anchor. The second strip begins at the anterior aspect of the proximal anchor and crosses to the posterior aspect of the distal anchor. Apply a total of four strips.	
3. Apply closure strips above and below the elbow over the original anchors.	
4. (Optional) Apply a 4- or 6-inch elastic bandage from distal to proximal to secure the taping.	

Figure 18-12. Elbow taping (valgus stretch injury).

2. As you are finishing wrapping, tear or cut the remaining portion of bandage lengthwise down the middle. Double back with one of the resulting strips and tie off.

Ankle and Foot Bandaging

Ankle bandaging with a 2- to 3-inch elastic wrap can be used to support a sprain. The bandage can be applied over a sock or directly to the skin. It is usually simplest to use a series of figure-8 wraps, or, if preferable, a series of heel locks as described in the section on ankle taping. Anchors and stirrups are not used. When bandaging the foot, the same technique should be carried out to the metatarsophalangeal (MTP) joint. Circumferentially bandaging the foot by itself will result in the bandage slipping, as opposed to bandaging the ankle as well.

Knee Bandaging

A double-length 6-inch elastic bandage can provide support to the knee. Ask the patient to hold the knee in slight flexion by placing his heel on a small stone or piece of wood (Fig. 18-16A). The elastic wrap is then applied circumferentially from mid-quadriceps to mid-calf (see Fig. 18-16B). If using gauze to secure a dressing or a smaller elastic wrap, then a series of figure-8 wraps can be applied, leaving the patella exposed.

Thigh and Groin Bandaging

Quadriceps, hamstring, and hip adductor ("groin") strains can all be treated with an elastic bandage in a hip spica. The bandage is modified slightly for the groin strain (Fig. 18-17).

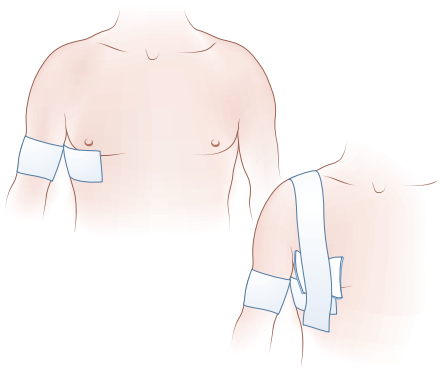
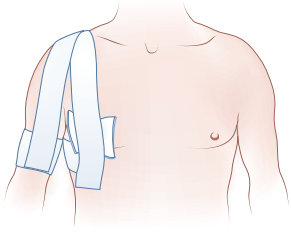
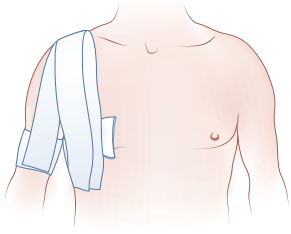
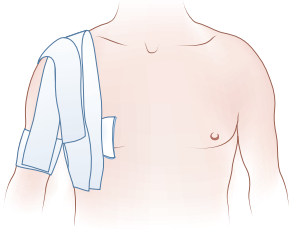
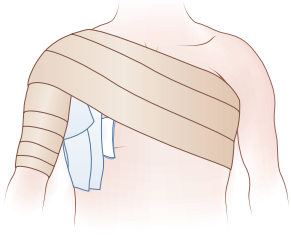
1. Apply three anchor strips with 3-inch elastic tape. Place the first anchor around the mid-humerus. Place the second anchor beginning inferior to the nipple anteriorly and around to the inferior tip of the scapula in the posterior. Apply the third anchor from the anterior portion of the second anchor, over the trapezius and extending to the posterior portion of the second anchor. Note that a 3-inch gauze pad is incorporated into this anchor to protect the nipple.	
2. Apply a longitudinal strip of 3-inch elastic tape from the anterior aspect of the first anchor, over the acromioclavicular joint and ending on the posterior aspect of the third anchor.	
3. Apply the next strip overlapping two thirds of the third anchor laterally. It should extend from posterior to anterior portions of the second anchor and cross over the acromioclavicular joint.	
4. Apply the next strip from the posterior aspect of the first anchor, over the acromioclavicular joint and ending at the anterior aspect of the third anchor.	
5. Repeat steps 2 through 4 two more times. (Optional) Wrap a 6-inch elastic bandage in a shoulder spica for additional support. (See Bandaging section.)	

Figure 18-13. Shoulder taping.

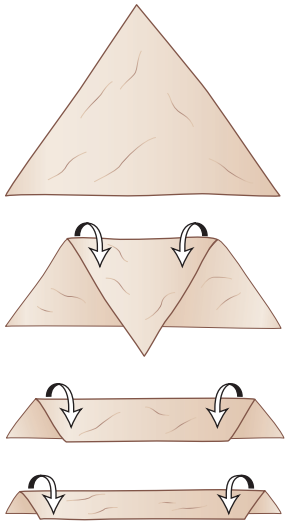


Figure 18-14. Making a cravat from a triangular bandage. (Redrawn from Auerbach PS: Medicine for the Outdoors, 4th ed. Guilford, CT, Lyons Press, 2003. p 262.)

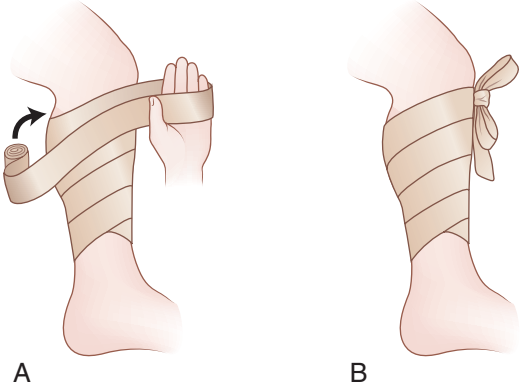


Figure 18-15. **A and B,** Securing a bandage. (Redrawn from Donelan S: That's a wrap: Wound bandaging made easy. Ski Patrol Magazine 2005; Winter: 63.)

A

B

Figure 18-16. **A,** Knee positioned in slight flexion with heel-lift while bandage is applied. **B,** Completed knee bandage.

Although the quadriceps and hamstring can be supported by wrapping only the leg with a 6-inch elastic bandage, the hip spica helps prevent slipping and provides additional support.

Wrist and Hand Bandaging

Support to the wrist can be supplied by a 2- to 3-inch elastic wrap using a continuous technique (Fig. 18-18). This same technique can be used with gauze to secure a dressing to a wound that can occur when falling on an outstretched hand.

A hand cravat bandage can be used for wounds that continue to bleed despite manual pressure[2] (Fig. 18-19).

Finger Bandaging

Finger wounds are generally easily treated with adhesive bandages. However, if size or degree of bleeding necessitates a larger dressing, then the following method may be used:

Fold a 1-inch rolled gauze back and forth over the tip of the finger to cover and cushion the wound (Fig. 18-20). Then wrap the gauze around the finger until the gauze is snug. On the last turn around the finger, pull the gauze over the top of the hand so that it extends beyond the wrist. Split this lengthwise; tie the ends around the wrist to secure the bandage.

Thumb Bandaging

Application of a bandage or dressing to the thumb usually involves a thumb spica, as described in the taping section. Rather than apply individual strips, the gauze or elastic bandage is looped continuously.

Shoulder Bandaging

A shoulder spica is used to support shoulder sprains, strains, and subluxations (Fig. 18-21).

A triangular bandage can be used to dress a shoulder wound (Fig. 18-22).

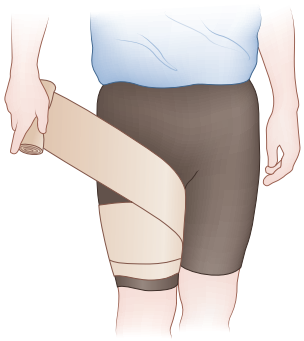
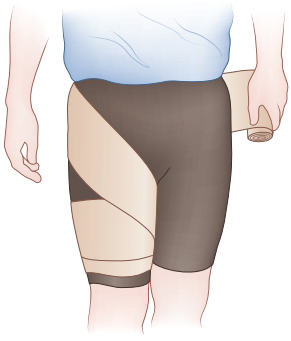
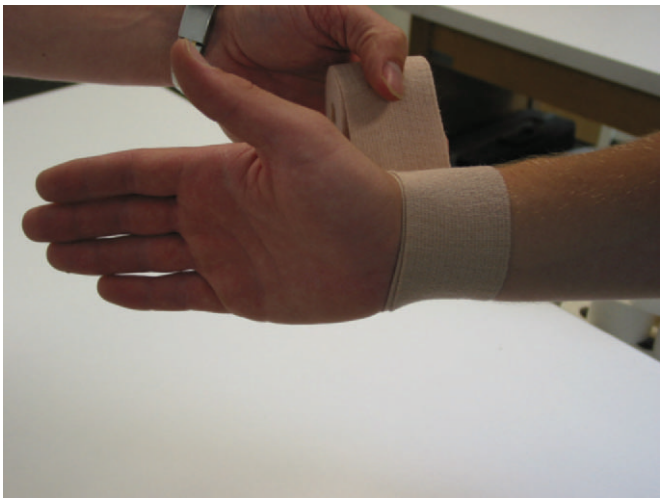
1. Wrap a double-length 6-inch elastic bandage around the mid-thigh in a medial to lateral direction and continue proximally.	
2. At the groin crease, continue up and around the waist once to help anchor the bandage.	
3. Return to the thigh to complete the figure-of-eight. For quadriceps and hamstring strains, concentrate on wrapping the leg, using an additional figure-of-eight to anchor the wrap. For groin strains, concentrate on supporting the hip adductors by alternating wrapping the leg with figure-of-eight wraps.	
4. Finish wrap on the leg.	

Figure 18-17. Thigh and groin bandaging.



A

B

C

D

E

Figure 18-18. Wrist bandaging. **A,** (1) Begin by encircling the wrist 2 to 3 times. **B,** (2) Continue across the dorsum of the hand, through the first web space and around the base of the proximal phalanges. **C,** (3) Continue down and across the dorsum of the hand. **D,** (4) Circle the wrist and bring across the dorsum of the hand to form a figure-8. **E,** (5) Repeat, alternating figure-8 patterns on the dorsum of the hand and secure at the wrist.

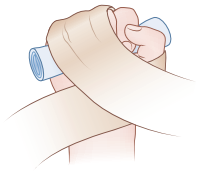
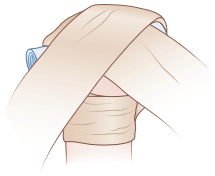
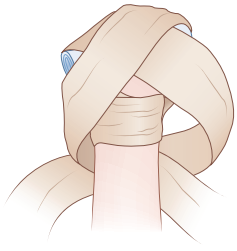
1. After dressing the wound, the patient closes his fist around rolled gauze.	
2. Starting from the anterior aspect of the wrist, wrap one end around the dorsum of the hand, over the fingers and back to the wrist.	
3. With tension, wrap the other end around the dorsum of the hand, over the fingers and back to the wrist, creating an X.	
4. Cross both ends around the wrist.	
5. Tie the ends to secure the dressing.	

Figure 18-19. Hand cravat bandage. (Redrawn from Donelan S: That's a wrap: Wound bandaging made easy. Ski Patrol Magazine 2005; Winter: 66.)

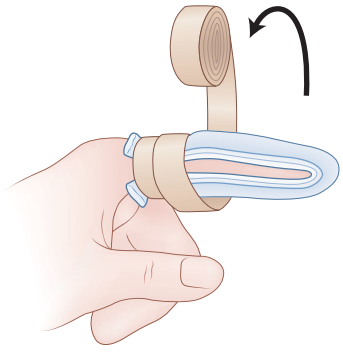
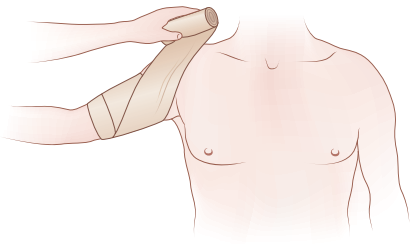
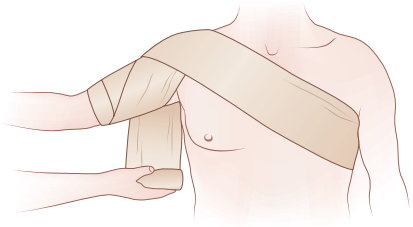


Figure 18-20. To begin a finger bandage, place layers of gauze over the fingertip. (Redrawn from Auerbach PS: Medicine for the Outdoors, 4th ed. Guilford, CT: Lyons Press, 2003, p 263.)

1. Begin by encircling the mid-humerus with a double-length 6-inch elastic bandage and continue proximally while wrapping. Once near the axilla, wrap over the acromioclavicular joint and around the posterior thorax.



2. Continue under the opposite axilla, across the chest and bring down over the acromioclavicular joint and onto the upper arm.



3. Repeat the figure-of-eight pattern as the length of the bandage allows and finish on the upper arm.

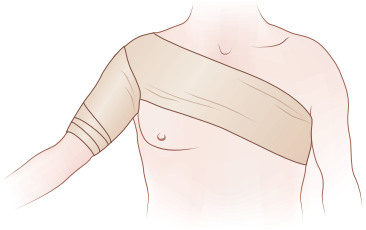
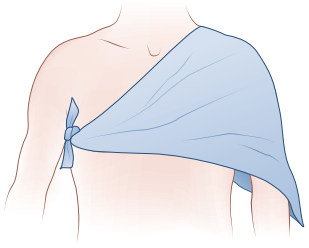


Figure 18-21. Shoulder bandaging.

1. Lay the base of the bandage over the shoulder with the apex pointed toward the arm. Tie the two free ends just anterior to the axilla.



2. Roll the apex up the arm to the desired point of coverage and tie off.

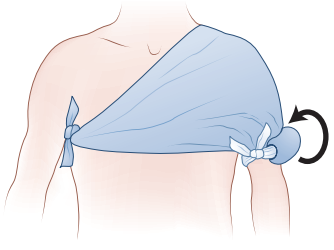


Figure 18-22. Shoulder bandaging (triangular bandage). (Redrawn from Auerbach PS: Medicine for the Outdoors, 4th ed. Guilford, CT: Lyons Press, 2003, p 264.)

Scalp Bandaging

Wounds to the scalp often require a dressing placed over hair, making adhesion very difficult. The dressing can be secured with a triangular bandage in a method that allows for considerable tension should pressure be necessary to stop bleeding (Fig. 18-23).

Ear Bandaging

A wound to the pinna may require a compression dressing. If so, gauze should be placed both anterior and posterior to the ear to allow it to maintain its natural curvature. A cravat is used to secure the dressing (Fig. 18-24). This method may be used for wounds anywhere along the side of the head or under the chin.

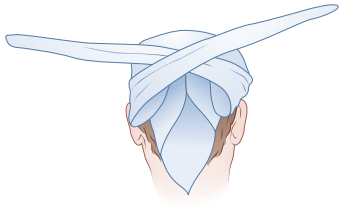
1. Drape a triangular bandage just over the eyes and fold the edge 1 inch under to form a hem. Allow the apex to drop over the back of the neck.	
2. Cross the free ends over the back of the head and tie in a half-knot.	
3. Bring the free ends to the front of the head and tie a complete knot. At the posterior aspect of the head, tuck the apex into the half-knot.	

Figure 18-23. Scalp bandaging. (Redrawn from Auerbach PS: Medicine for the Outdoors, 4th ed. Guilford, CT: Lyons Press, 2003, p 265.)

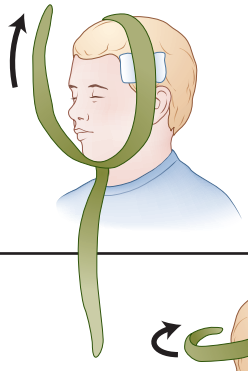
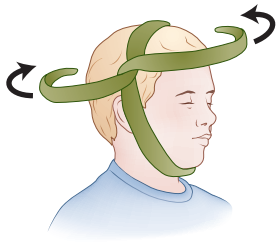
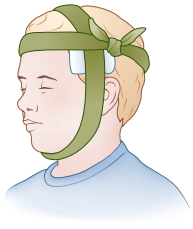
1. Place the cravat over the wound at the cravat's midpoint. Wrap one end over the head and the other under the chin.	
2. Cross the cravat just above ear level and wrap ends in opposite directions.	
3. Tie off the ends.	

Figure 18-24. Ear bandaging. (Redrawn from Auerbach PS: Medicine for the Outdoors, 4th ed. Guilford, CT: Lyons Press, 2003, p 266.)

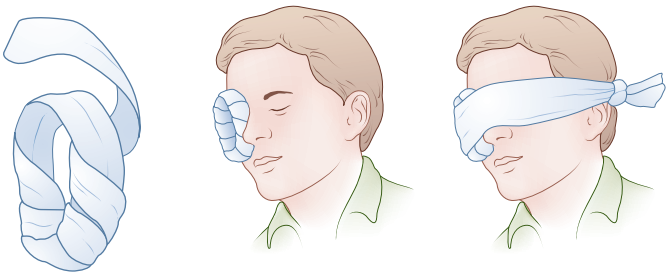


Figure 18-25. Bandage for the injured eye. A cravat or cloth is rolled and wrapped to make a doughnut-shaped shield, which is fixed in place over the eye. (Redrawn from Auerbach PS: Medicine for the Outdoors, 4th ed. Guilford, CT: Lyons Press, 2003, p 175.)

Figure 18-26. Holding an eye patch in place with a cravat. Hang a cloth strip over the uninjured eye. Hold the patch in place with the cravat. Tie the cloth strip to lift the cravat off the uninjured eye (Redrawn from Auerbach PS: Medicine for the Outdoors, 4th ed. Guilford, CT: Lyons Press, 2003, p 262.)

Eye Bandaging

When bandaging an eye, a shield is placed over the eye socket to protect the globe, followed by application of a bandage over the shield. The shield may be commercially available sterile pads, cut foam or felt, stacked gauze, or a shirt or cravat fashioned into a doughnut shape (Fig. 18-25). The bandage is fashioned from a cravat and a spare piece of 15-inch cloth or shirt. The spare cloth is placed over the top of the head from posterior to anterior such that the anterior portion lies over the unaffected eye. A cravat is then applied horizontally to hold the shield over the injured eye. To expose the uninjured eye, pull up both ends of the spare cloth and tie at the top of the head (Fig. 18-26).

The references for this chapter can be found on the accompanying DVD-ROM.

19 Emergency Airway Management

Swaminatha V. Mahadevan

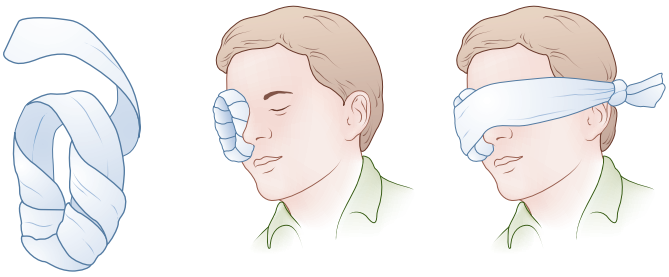
Emergency airway management encompasses the assessment, establishment, and protection of the airway in combination with effective oxygenation and ventilation. Timely, effective airway management can literally mean the difference between life and death, taking precedence over all other clinical considerations.

Airway management in the wilderness must often be provided in austere or unusual environments under less than ideal circumstances. Many of the resources and equipment readily available in a hospital or emergency department setting are not accessible in the wilderness. As with other aspects of wilderness medicine, improvisation may prove invaluable.

AIRWAY ANATOMY

A full understanding of airway anatomy is essential for airway evaluation and management. Internally, the airway is made up of many structures and well-defined spaces, and originates at the nasal and oral cavities (Fig. 19-1). The nasal cavity extends from the nostrils to the posterior nares or choanae. Resistance to airflow through the nose is almost twice that of the mouth, explaining why patients mouth-breathe when they require high flow rates (e.g., with exercise). The *nasopharynx* extends from the end of the nasal cavity to the level of the soft palate. The tonsillar lymphoid structures are the principal impediments to airflow through the nasopharynx. The *oral cavity* is bounded by the teeth anteriorly, hard and soft palate above, and tongue below. The *oropharynx*, which communicates with the oral cavity and nasopharynx, extends from the soft palate to the tip of the epiglottis. The tongue is the principal source of obstruction in the oropharynx. This obstruction results in part from decreased muscle tone of the genioglossus muscle, which contracts to move the tongue forward during inspiration and dilate the pharynx.

The oropharynx continues as the *laryngopharynx (hypopharynx)*, which extends from the epiglottis to the upper border of



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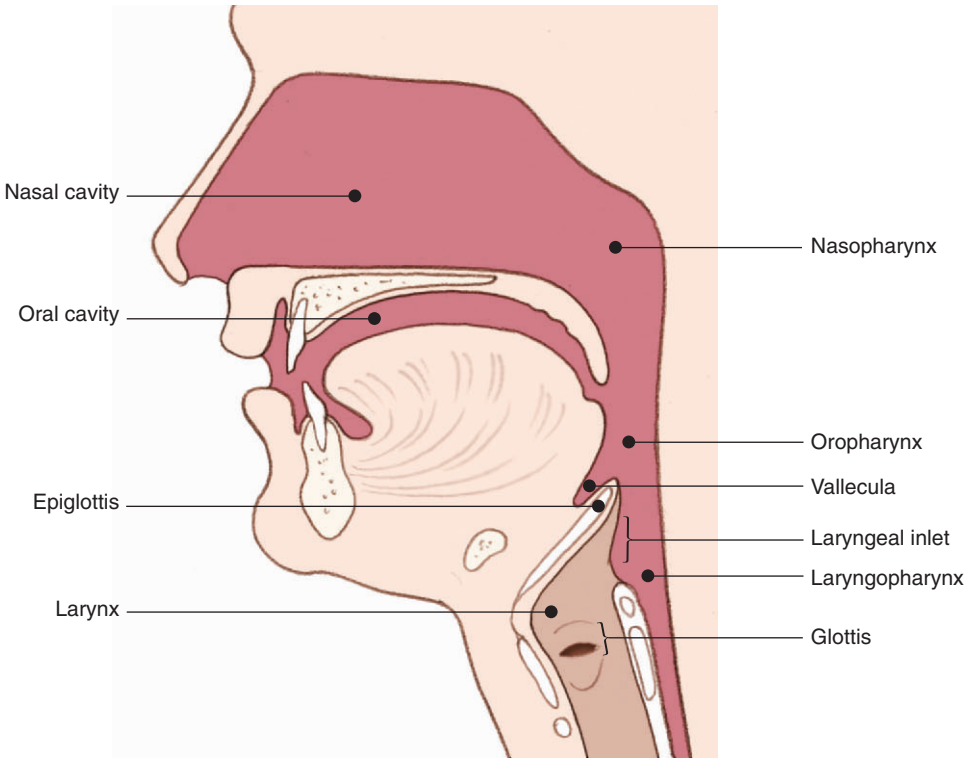


Figure 19-1. Lateral airway anatomy. (Redrawn from Mahadevan SV, Garmel GM [eds]: An Introduction to Clinical Emergency Medicine: Guide for Practitioners in the Emergency Department. Cambridge, UK, Cambridge University Press, 2005. © Chris Gralapp, www.biolumina.com.)

the cricoid cartilage (at the level of the C6 vertebral body). The *larynx*, which lies between the laryngopharynx and trachea, serves as an organ of phonation and a valve to protect the lower airway from aspiration. The larynx is made up of muscles, ligaments, and cartilages, including the thyroid, cricoid, arytenoids, corniculates, and epiglottis.

The flexible *epiglottis*, which originates from the hyoid bone and base of the tongue, covers the glottis during swallowing and provides protection from aspiration. During laryngoscopy, the epiglottis is as an important landmark for airway identification and laryngoscopic positioning. The *vallecula* is the space at the base of the tongue formed posteriorly by the epiglottis and anteriorly by the anterior pharyngeal wall. The *laryngeal inlet* is the opening to the larynx bounded by the epiglottis, aryepiglottic folds, and arytenoid cartilages. The *glottis* is the vocal apparatus including the true and false vocal cords and the glottic opening. The triangular fissure between these vocal cords is the *glottic opening*, the narrowest segment of the larynx in adults.

Externally identifiable landmarks are also important to airway assessment and management (Fig. 19-2). The *mentum* is the anterior aspect of the mandible, forming the tip of the chin. The *hyoid bone* forms the base of the floor of the mouth. The *thyroid cartilage* forms the laryngeal prominence ("Adam's apple") and thyroid notch. The *cricoid cartilage*, lying inferior to the thyroid cartilage, forms a complete ring that provides structural support to the lower airway. The *cricothyroid membrane* lies between the thyroid and cricoid cartilage and serves as an important site for surgical airway management.

Knowledge of the anatomic differences between adults and children is integral to effective pediatric airway management. These important differences are summarized in Table 19-1 and Figure 19-3.

ASSESSMENT OF THE AIRWAY AND RECOGNITION OF AIRWAY COMPROMISE

Assessment of the airway begins with evaluation of airway patency and respiratory function. The goal of this assessment is to determine whether the airway is patent and protected, and whether breathing is present and adequate. This is accomplished by inspection, auscultation, and palpation, commonly known as the "look, listen, and feel" approach.

Visual signs of airway compromise include agitation, obtundation, and cyanosis. Blue, gray, or ashen skin, especially around the eyes, lips, and nail beds, is a worrisome finding. Significant airway compromise may present without cyanosis, such as an allergic reaction with upper airway edema and vasodilation (causing flushed red skin), or unconsciousness resulting from carbon monoxide poisoning.

Bradypnea, tachypnea, or irregular respirations may be a sign of impending respiratory compromise. Breathing that is shallow, deep, or labored may indicate respiratory insufficiency. Respiratory muscle fatigue may result in recruitment of the accessory muscles of respiration, clinically manifested

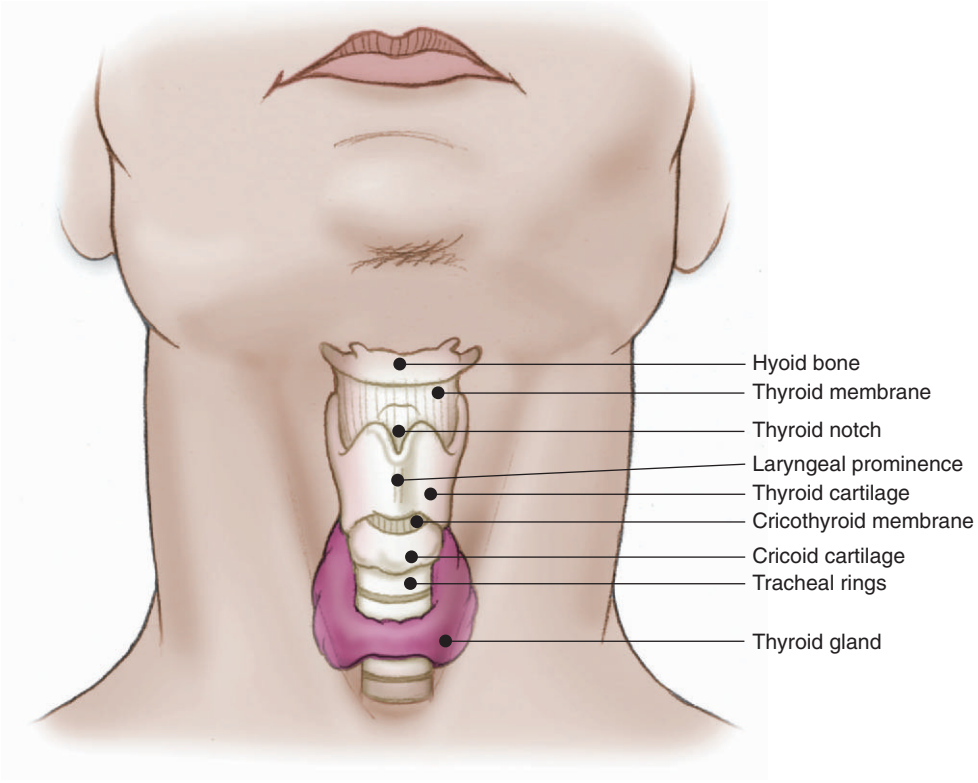


Figure 19-2. External airway anatomy. (Redrawn from Mahadevan SV, Garmel GM [eds]: An Introduction to Clinical Emergency Medicine: Guide for Practitioners in the Emergency Department. Cambridge, UK, Cambridge University Press, 2005. © Chris Gralapp, www.biolumina.com.)

TABLE 19-1. Anatomic Airway Differences between Children and Adults

ANATOMY	CLINICAL SIGNIFICANCE
Large intraoral tongue occupying relatively large portion of the oral cavity	1. High anterior airway position of the glottic opening compared with that in adults
High tracheal opening: C1 in infancy versus C3–4 at age 7, C4–5 in adults	2. Straight blade preferred over curved to push distensible anatomy out of the way to visualize the larynx
Large occiput that may cause flexion of the airway; large tongue that easily collapses against the posterior pharynx	Sniffing position is preferred. The large occiput actually elevates the head into the sniffing position in most infants and children. A towel may be required under shoulders to elevate torso relative to head in small infants.
Cricoid ring narrowest portion of the trachea as compared with the vocal cords in adults	1. Uncuffed tubes provide adequate seal as they fit snugly at the level of the cricoid ring. 2. Correct tube size is essential because variable expansion cuffed tubes are not used.
Consistent anatomic variations with age, with fewer abnormal variations related to body habitus, arthritis, chronic disease	<2 years, high anterior 2–8 years, transition >8 years, small adult
Large tonsils and adenoids may bleed. More acute angle between epiglottis and laryngeal opening results in nasotracheal intubation attempt failures.	Blind nasotracheal intubation not indicated in children Nasotracheal intubation failure
Small cricothyroid membrane	Needle cricothyroidotomy difficult and surgical cricothyroidotomy impossible in infants and small children

From Walls RM, Murphy MF, Luten RC, Schneider RE (eds): Manual of Emergency Airway Management, 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2004.

as suprasternal, supraclavicular, or intercostal retractions. Traumatic injury to the chest (e.g., flail chest) or an aspirated foreign body may result in paradoxical or discordant chest wall movement.

In children, visual signs of airway compromise and respiratory distress include tachypnea, cyanosis, drooling, nasal flaring, and intercostal retractions. A child with severe upper airway obstruction may sit upright with the head tilted back ("sniffing" position) to straighten the airway and reduce occlusion. A child with severe lower airway obstruction may sit up and lean forward on outstretched arms ("tripod" position) to augment accessory muscle function.

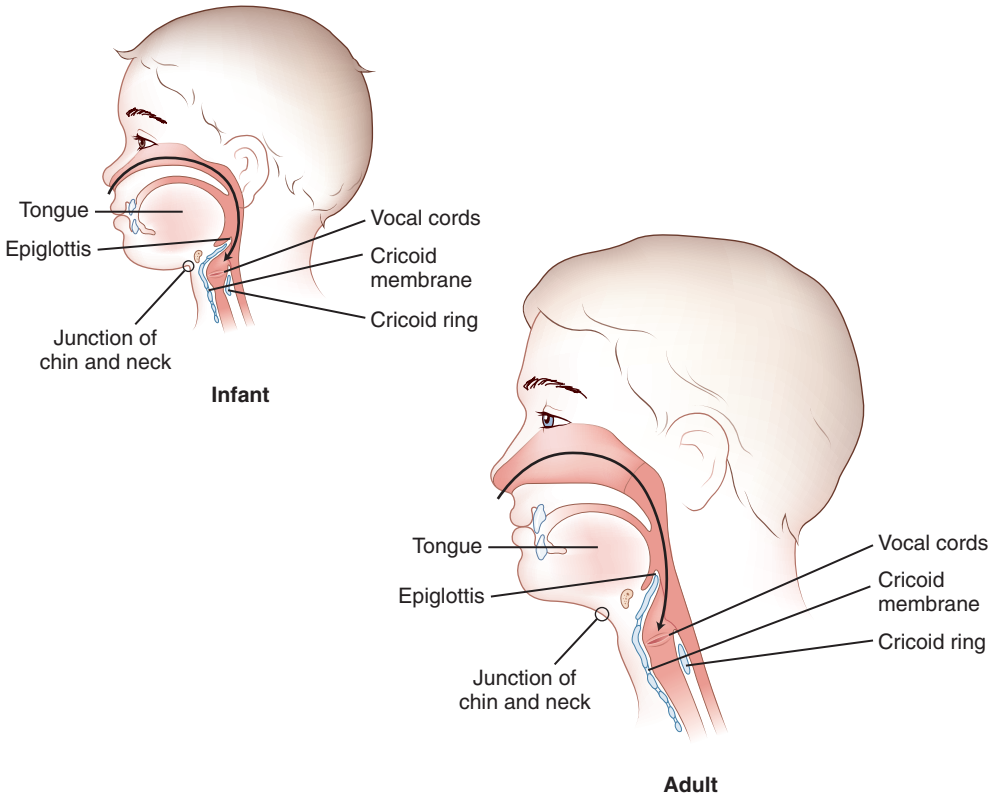


Figure 19-3. Anatomic airway differences between children and adults. The anatomic differences particular to children include: 1. Higher, more anterior position for the glottic opening. (Note the relationship of the vocal cords to the chin–neck junction.) 2. Relatively larger tongue in the infant, lying between the mouth and the glottic opening. 3. Relatively larger and more floppy epiglottis in the child. 4. Cricoid ring is the narrowest portion of the pediatric airway; in adults, the narrowest portion is the vocal cords. 5. Position and size of the cricothyroid membrane in the infant. 6. Sharper, more difficult angle for blind nasotracheal intubation. 7. Larger relative size of the occiput in the infant. (Redrawn from Walls RM, Murphy MF, Luten RC, Schneider RE [eds]: Manual of Emergency Airway Management, 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2004.)

Under most circumstances, hearing the victim speak with a normal voice suggests that the airway is adequate for the moment. Unusual sounds or noisy respirations may be present with partial airway obstruction. Snoring indicates partial airway obstruction at the pharyngeal level; gurgling may be heard with blood or secretions in the airway; stridor may be associated with partial airway obstruction at the level of the larynx (inspiratory stridor) or the level of the trachea (expiratory stridor); hoarseness suggests a laryngeal process.

The central face and mandible should be assessed for structural integrity because injuries to these structures may lead to airway distortion and compromise. The anterior neck should be carefully inspected for penetrating wounds, asymmetry, or swelling that may herald impending airway compromise. Palpation of subcutaneous air suggests direct airway injury.

In the unconscious victim, feel for air movement at the mouth and nose. Open the mouth to inspect the upper airway, taking care not to extend or rotate the neck. Identify and remove any vomitus, blood, or other foreign bodies. Look for swelling, bleeding, or other abnormalities of the oropharynx. The gentle use of a tongue blade may facilitate this task. The victim's ability to spontaneously swallow and handle secretions is an important indicator of intact airway protective mechanisms. In the unconscious victim, absence of a gag reflex has traditionally been linked to loss of protective airway reflexes.

Auscultation of the lung fields should demonstrate clear and equal breath sounds. Diminished breath sounds may result from a pneumothorax, hemothorax, or pleural effusion. Wheezing and dyspnea are often associated with lower airway obstruction.

OPENING THE AIRWAY

Opening the airway and ensuring airway patency are essential for adequate oxygenation and ventilation. These are the first priorities in airway management. The conscious victim uses the musculature of the upper airway and protective reflexes to maintain a patent airway and protect against aspiration of foreign substances, gastric contents, or secretions. In the severely ill, compromised, or unconscious victim, these protective airway mechanisms may be impaired or absent. Upper airway obstruction in the unconscious victim is most commonly the result of posterior displacement of the tongue and epiglottis at the level of the pharynx and larynx. This occlusion results directly from the loss of tonicity of the submandibular muscles, which provide direct support to the tongue and indirect support to the epiglottis. Upper airway obstruction may be alleviated by head positioning, manual airway techniques, and mechanical airway adjuncts.

Head Positioning

If the mechanism of injury or physical examination suggest possible of cervical spine injury, the head should be placed in the neutral position, and efforts to stabilize the neck and head should be initiated. Care should be taken not to flex, extend, or rotate the victim's head. After cervical spine immobilization, the airway should be reevaluated for obstruction.

The optimal head position for airway alignment and patency varies with age. For an infant in the supine position, the large

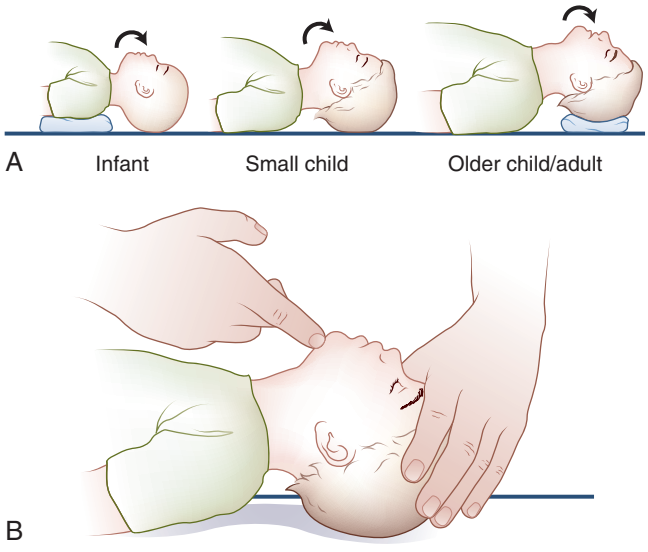


Figure 19-4. **A,** Clinical determination of optimal airway alignment using a line passing through the external auditory canal and anterior to the shoulder (see text for details). **B,** Application of the line to determine optimal position. In this small child the occiput obviates the need for head support, yet the occiput is not so large as to require support of the shoulders. Note that the line traversing the external auditory canal passes anterior to the shoulders. With only slight extension of the head on the atlanto-occipital joint, the sniffing position is achieved. (Redrawn from Walls RM, Murphy MF, Luten RC, Schneider RE [eds]: Manual of Emergency Airway Management, 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2004.)

occiput contributes to flexion of the head and neck and resultant airway obstruction. This may be alleviated by elevating the shoulders with a small towel (Fig. 19-4). In children, slightly extending the head into the sniffing position helps relieve airway obstruction. In adults, placing a folded towel or article of clothing under the occiput, which flexes the neck at the torso, followed by gentle hyperextension of the head at the atlanto-occipital joint, provides optimal alignment of the air-way axes.

Manual Airway Techniques

Although the following manual airway techniques are effective, they often require continuous involvement by a single provider to maintain airway patency.

Head Tilt with Chin Lift

The **head tilt with chin lift** (Fig. 19-5) is a simple, effective technique for opening the airway. This maneuver is accomplished by placing the palm of one hand on the victim's forehead and then applying firm backward pressure to tilt the head back. Simultaneously, the fingers of the other hand are then placed under the bony part of the chin and lifted, bringing the chin forward. These fingers support the jaw and maintain the head-tilt position. This maneuver extends the neck; therefore, it should not be used if there is concern about a cervical spine injury.

Jaw Thrust with Head Tilt

If a cervical spine injury is not suspected, the **jaw thrust with head tilt maneuver** may be used to gain additional forward displacement of the mandible. Position yourself at the top of the victim's head. Then, grasp the angles of the mandible with both hands and lift, displacing the jaw forward while tilting the head back.

Jaw Thrust without Head Tilt

If a cervical spine injury is suspected or cannot be excluded, the **jaw thrust without head tilt** (Fig. 19-6) can be performed while maintaining neutral cervical spine alignment. In this maneuver, the jaw thrust is performed without extending or rotating the neck.

Tongue Traction

If the patient is unconscious or in extremis, the airway may be opened temporarily by attaching the anterior aspect of the victim's tongue to the lower lip with two safety pins (Fig. 19-7). An alternative to piercing the lower lip is to pass a string through the safety pins and exert traction on the tongue by securing the end of the string to the victim's shirt button or jacket zipper (Fig. 19-8).

Mechanical Airway Adjuncts

Several airway adjuncts are available to maintain airway patency while freeing up the health care provider to perform other duties.

Oropharyngeal Airway

The oropharyngeal airway (OPA) is an S-shaped device designed to hold the tongue off the posterior pharyngeal wall (Fig. 19-9). When properly placed, it prevents the tongue from obstructing the glottis. It also provides an air channel and suction conduit through the mouth. These devices are most effective in unconscious and semiconscious victims who lack a gag reflex or cough. The use of an OPA in a victim with a gag reflex or cough is contraindicated because it may stimulate retching, vomiting, or laryngospasm.

The OPA is made of disposable plastic and comes in various sizes to accommodate children and adults. The size is based on the distance (in millimeters) from the flange to the distal tip. The proper OPA size is estimated by placing the OPA's flange at the corner of the mouth so that the bite block segment is parallel to the victim's hard palate; the distal tip of the airway should reach the angle of the jaw.

Two types of OPAs are commonly employed. The Guedel uses a tubular design, whereas the Berman is distinguished by airway channels on each side.

Technique for Insertion:

1. First, open the mouth and clear the pharynx of any secretions, blood, or vomitus. Remove dentures or partial plates if present.
2. In an adult or older child, insert the OPA upside down or at a 90-degree angle to avoid pushing the tongue posteriorly during insertion. Slide it gently along the roof of the mouth. As the oral airway is inserted past the uvula or crest of the tongue, rotate it so that the tip points down the victim's throat.
3. In a child, grasp the tongue and gently pull it forward or use a tongue blade to displace the tongue inferiorly and anteriorly. Then, insert the OPA with the tip pointing toward the tongue and throat (i.e., the intended position following placement).
4. The flange should rest against the victim's lips, and the distal portion should rest on the posterior pharyngeal wall. If the OPA is too short, it may displace the tongue

Figure 19-5. Head tilt with chin lift. (Redrawn from Mahadevan SV, Garmel GM [eds]: An Introduction to Clinical Emergency Medicine: Guide for Practitioners in the Emergency Department. Cambridge, UK, Cambridge University Press, 2005. © Chris Gralapp, www.biolumina.com.)

into the hypopharynx and occlude the airway. If the OPA is too long, it may displace the epiglottis and result in an airway obstruction.

Nasopharyngeal Airway

The nasopharyngeal airway (NPA) is an uncuffed trumpet-like tube that provides a conduit for airflow between the nares and pharynx (Fig. 19-10). The NPA is inserted through the nose rather than the mouth. It has a flange at the outer end to prevent displacement or slippage beyond the nostril. These devices are better tolerated than OPAs and are commonly used in intoxicated or semiconscious victims. They are also effective when trauma, trismus ("clenched teeth"), or other obstacles (e.g., wiring of the teeth) preclude placement of an oropharyngeal airway. NPAs are contraindicated in victims with basilar skull or facial fractures because inadvertent intracranial placement may occur.

The NPA is made of soft, pliable rubber or plastic and comes in various sizes to accommodate children and adults. Sizes (internal diameter) vary from 12 to 36 French. Proper NPA length is determined by measuring the distance from the tip of the nose to the tragus of the ear.

Technique for Insertion:

1. Lubricate the nasopharyngeal airway with a water-soluble lubricant to minimize resistance in the nasal cavity. Do not use petroleum jelly or a non–water-based lubricant.
2. If the NPA has a beveled (angled) edge, place the airway in the nostril with the bevel directed toward the nasal septum.
3. Gently insert the NPA straight back along the floor of the nasal passage (perpendicular to the coronal plane of the face).
4. If you meet resistance, rotate the tube slightly, reattempt insertion through the other nostril, or try a smaller-diameter tube. Do not force the tube because injury to the nasal mucosa can result in bleeding.
5. Following insertion, the flange should rest on the victim's nostril, and the distal portion of the airway should rest in the posterior pharynx, behind the tongue.

Any flexible tube of appropriate diameter and length can be used as an improvisational substitute for the NPA. Examples include a Foley catheter, radiator hose, solar shower hose, siphon tubing, or inflation hose from a kayak flotation bag or sport pouch. An endotracheal tube (ETT) can be shortened and softened in warm water to substitute for a commercial nasal trumpet. The flange can be improvised using a safety pin through the nostril end of the tube (Fig. 19-11).

Although OPAs and NPAs help to establish artificial airways, they do not provide definitive airway protection from aspiration.

Figure 19-6. Jaw thrust without head tilt. (Redrawn from Mahadevan SV, Garmel GM [eds]: An Introduction to Clinical Emergency Medicine: Guide for Practitioners in the Emergency Department. Cambridge, UK, Cambridge University Press, 2005. © Chris Gralapp, www.biolumina.com.)

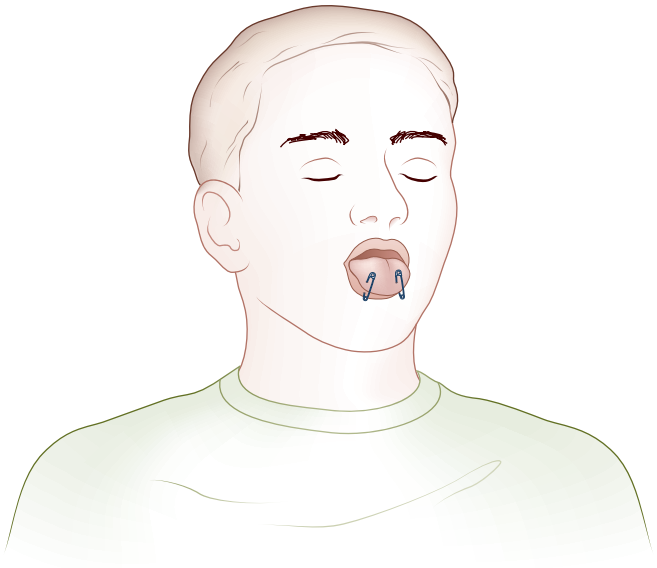


Figure 19-7. Tongue traction. The airway may be opened temporarily by attaching the anterior aspect of the victim's tongue to the lower lip with two safety pins.

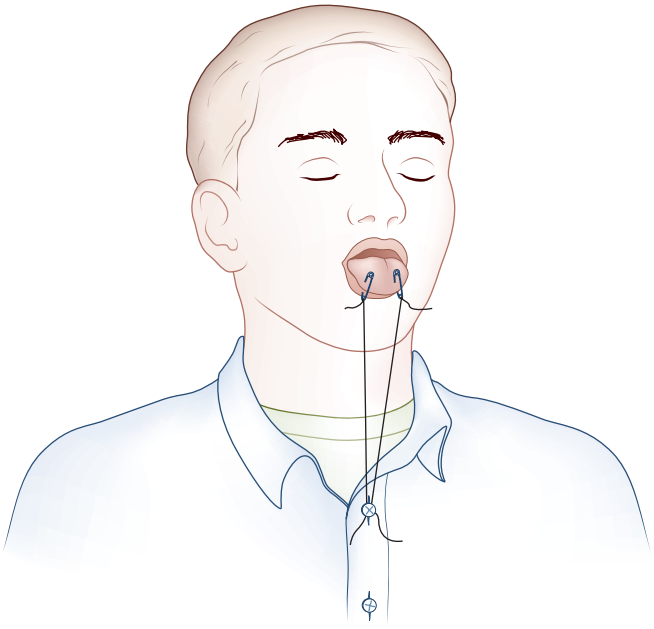


Figure 19-8. Tongue traction. An alternative to piercing the lower lip is to pass a string through the safety pins and exert traction on the tongue by securing the end of the string to the victim's shirt button or jacket zipper.

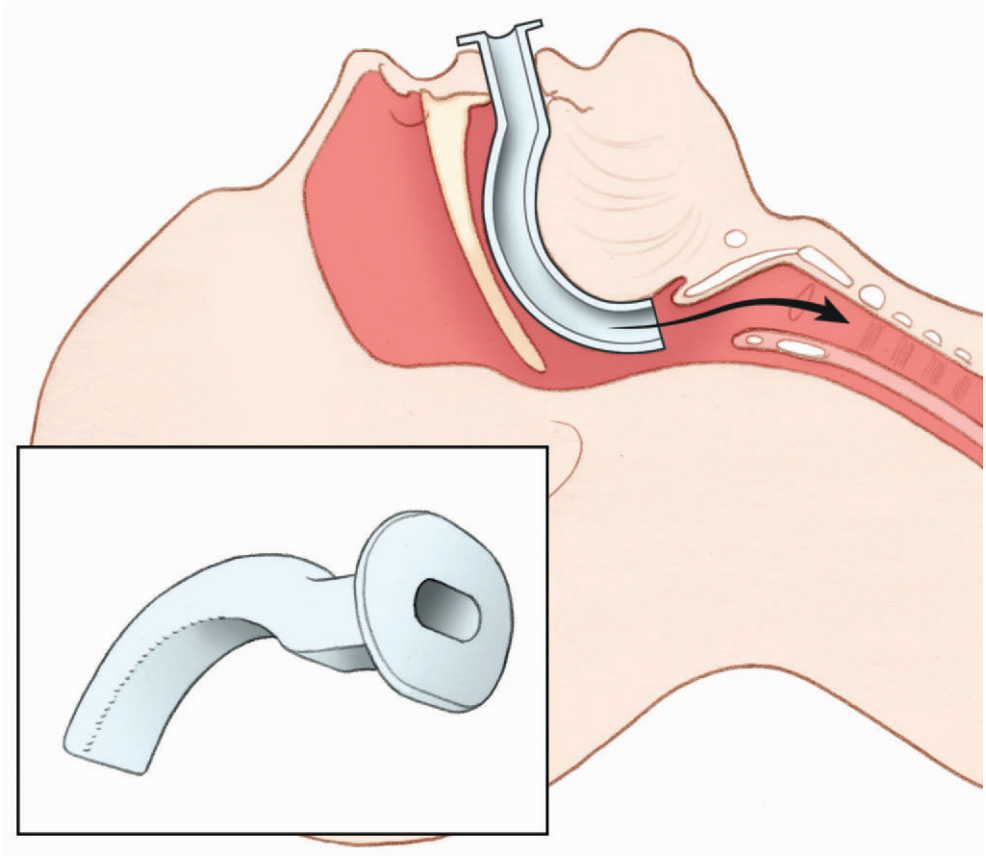


Figure 19-9. Oropharyngeal airway. (Redrawn from Mahadevan SV, Garmel GM [eds]: An Introduction to Clinical Emergency Medicine: Guide for Practitioners in the Emergency Department. Cambridge, UK, Cambridge University Press, 2005. © Chris Gralapp, www.biolumina.com.)

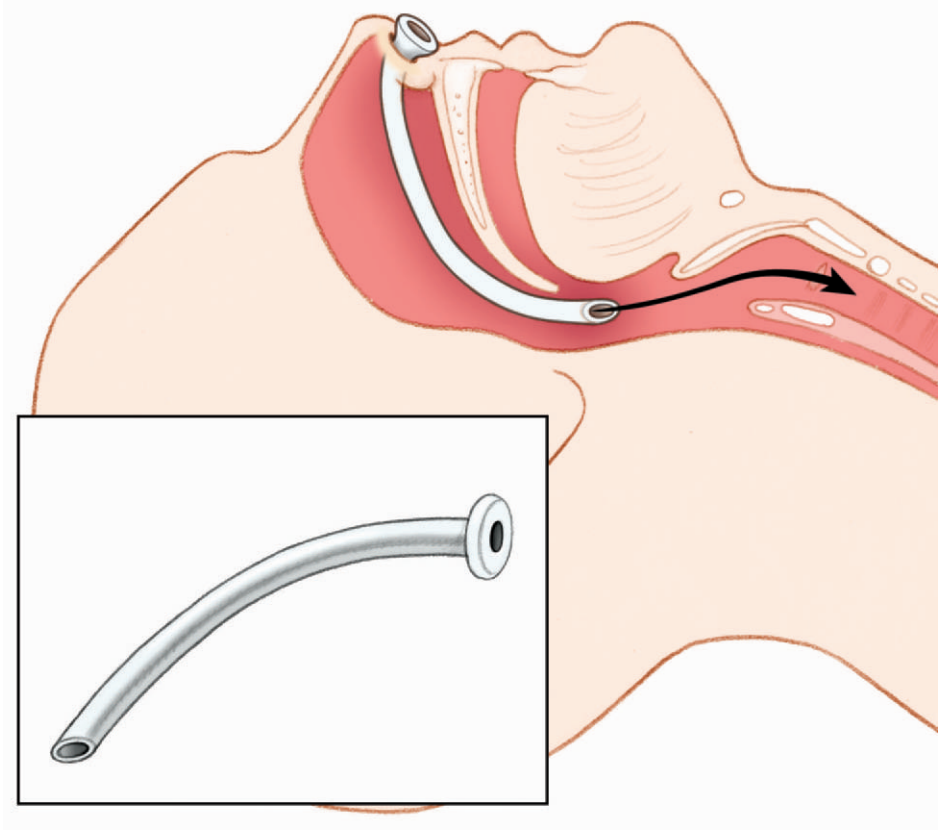


Figure 19-10. Nasopharyngeal airway. (Redrawn from Mahadevan SV, Garmel GM [eds]: An Introduction to Clinical Emergency Medicine: Guide for Practitioners in the Emergency Department. Cambridge, UK, Cambridge University Press, 2005. © Chris Gralapp, www.biolumina.com.)

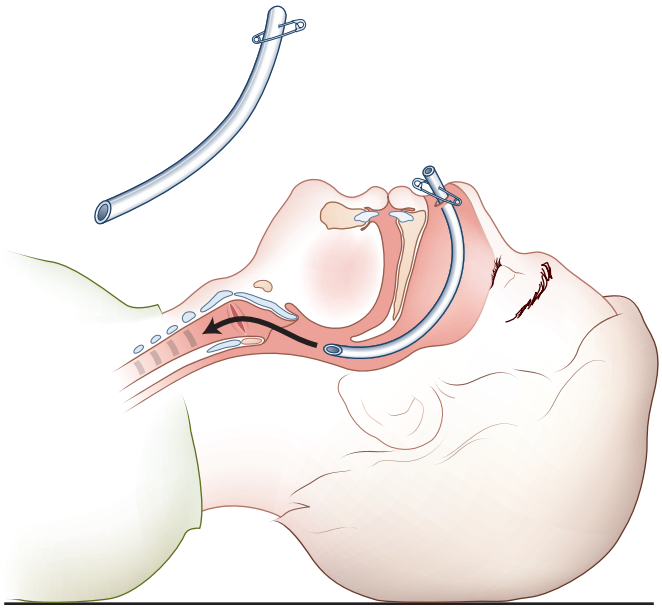


Figure 19-11. Improvised nasal trumpet.

Recovery Position

In the spontaneously breathing, unconscious victim who is not at risk for cervical spine injury, placement in the recovery position (Fig. 19-12) assists with maintaining a clear airway and reduces the risk for aspiration. In the recovery position, the tongue is less likely to fall back and occlude the airway, and vomitus is more likely to be expelled than inhaled. Even a diminutive rescuer can place a large person in the recovery position if the proper technique is employed.

FOREIGN-BODY AIRWAY OBSTRUCTION

Foreign bodies, most commonly meat, may cause partial or complete airway obstruction. A victim with partial airway obstruction can usually phonate or produce a forceful cough in an attempt to expel the foreign body. When encountering a victim with a partially-obstructed airway, if air exchange is adequate, do not interfere with the person's attempts to clear the airway. Encourage forceful coughing and closely monitor the victim's condition. If the obstruction persists or air exchange worsens or becomes inadequate, the victim should be managed as though a complete airway obstruction exists. Worrisome findings that should prompt immediate management include a weak or ineffective cough, increased respiratory difficulty, decreased air movement, and cyanosis.

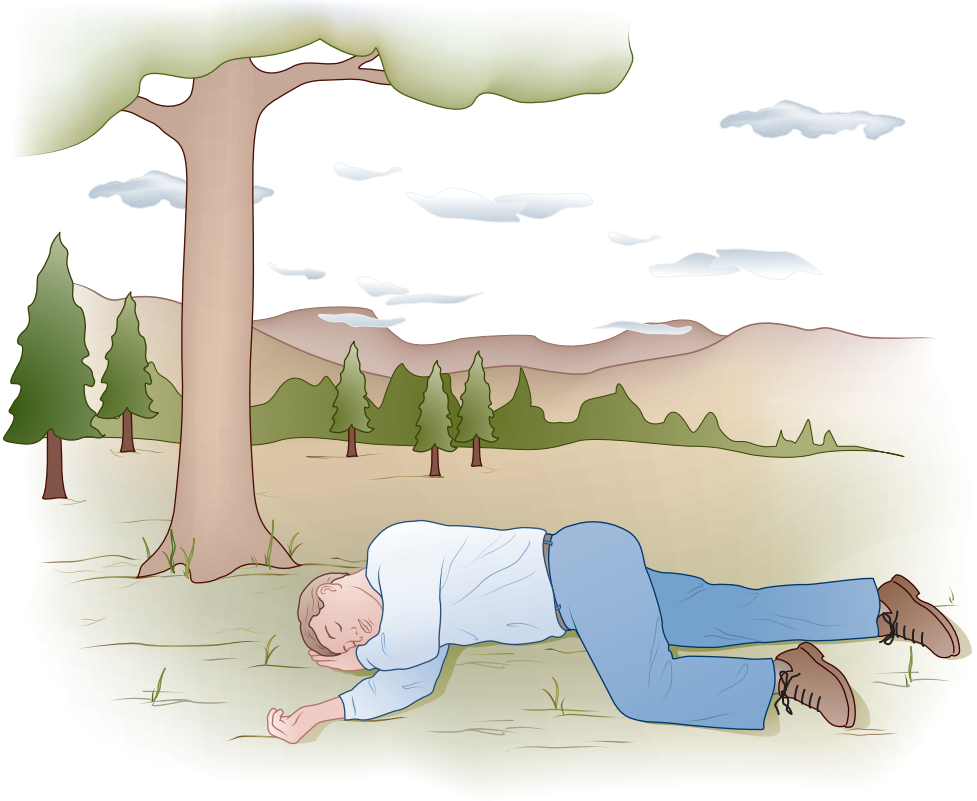


Figure 19-12. Recovery position.

TABLE 19-2. Relief of Choking

	ADULTS AND CHILDREN <1 YEAR OF AGE		CHILDREN <1 YEAR OF AGE	
	SIGNS	RESCUER ACTIONS	SIGNS	RESCUER ACTIONS
Mild airway obstruction	Good air exchange Responsive and can cough forcefully May wheeze between coughs	1. As long as good air exchange continues, encourage the victim to continue spontaneous coughing and breathing efforts. 2. Do not interfere with victim's own attempts to expel the foreign body, but stay with the victim and monitor his or her condition. 3. If mild airway obstruction persists, activate EMS.	Good air exchange Responsive and can cough forcefully May wheeze between coughs	1. Do not interfere with victim's own attempts to expel the foreign body, but stay with the victim and monitor his or her condition. 2. If mild airway obstruction persists, activate EMS.
Severe airway obstruction	Poor or no air exchange Weak, ineffective cough or no cough at all High-pitched noise when inhaling or no noise at all Increased respiratory difficulty Possible cyanosis (turning blue) Unable to speak Clutching neck with thumb and fingers (universal choking sign) Unable to move air	1. Ask the victim if he or she is choking. If the victim nods yes and cannot talk, severe airway obstruction is present and you must activate EMS. 2. Perform abdominal thrusts (Heimlich maneuver). It may be necessary to repeat the thrusts several times to clear the airway. 3. Repeat until the object is expelled from the airway or the victim becomes unresponsive. 4. If the patient is pregnant or obese, perform chest thrusts instead.	Poor or no air exchange Weak, ineffective cough or no cough at all High-pitched noise when inhaling or no noise at all Increased respiratory difficulty Possible cyanosis (turning blue) Unable to cry Unable to move air	1. If the victim cannot make any sounds or breathe, severe airway obstruction is present and you must activate EMS. 2. Deliver 5 forceful back slaps between the victim's shoulder blades using the heel of your hand. 3. If the airway remains obstructed, perform 5 quick downward chest thrusts just below the nipple line at a rate of 1 per second. 4. Repeat back slaps and chest thrusts until the object is removed or the victim becomes unresponsive.
Choking in the unresponsive victim	Choking victims may be initially responsive and then become unresponsive. In this circumstance, you know the choking has caused the victim's symptoms.	**Adults** 1. Activate EMS. 2. Open the airway. 3. Remove the object if you see it. **Children from 1 yr to puberty** 1. Open the airway. 2. Remove the object if you see it. 3. Begin CPR. 4. After 2 minutes (or 5 cycles) of CPR, activate EMS.	Chocking infants may be initially responsive and then become unresponsive. In this circumstance, you know the choking has caused the victim's symptoms.	1. Place the infant onto a flat surface. 2. Open the airway. 3. Remove the object if you see it. 4. Do not perform blind finger sweeps because the foreign body may be pushed back into the airway, causing further obstruction or injury. 5. Begin CPR. 6. Each time you open the airway, look for the object and remove it if you see it. 7. After 2 minutes (or 5 cycles) of CPR, activate EMS.

CPR, cardiopulmonary resuscitation; EMS, emergency medical services.
Adapted from American Heart Association: BLS for Healthcare Providers. Dallas, American Heart Association, 2006.

A person with a complete airway obstruction cannot speak (aphonia), exchange air, or cough. The person will often grasp the neck, the universal distress signal for choking, and open the mouth widely. The unconscious victim with complete airway obstruction will not demonstrate any typical chest movements or other signs of good air exchange. Individuals with complete airway obstruction from a foreign body require immediate medical attention. Failure to rapidly relieve the obstruction can lead to cardiac arrest.

A complete summary of the treatment for complete foreign-body airway obstruction in adults and children is provided in Table 19-2.

SUCTION

All sick or injured victims are at risk for airway obstruction and pulmonary aspiration. Life-saving interventions, such as bag-valve-mask (BVM) ventilation, may increase this risk. Suction is essential for removal of vomitus, secretions, blood, and foreign bodies that may occlude the airway or increase the risk for pulmonary aspiration.

Portable suction devices (available from a number of manufacturers) are ideal for the wilderness setting. These portable units should provide enough vacuum flow for adequate pha-

A

B

Figure 19-13. **A,** RES-Q-VAC suction device. **B,** S-SCORT Jr Quickdraw battery-powered, hand-held device. (**A** redrawn from Repro-Med Systems, Inc. product information; **B** redrawn from SSCOR, Inc. product information.)

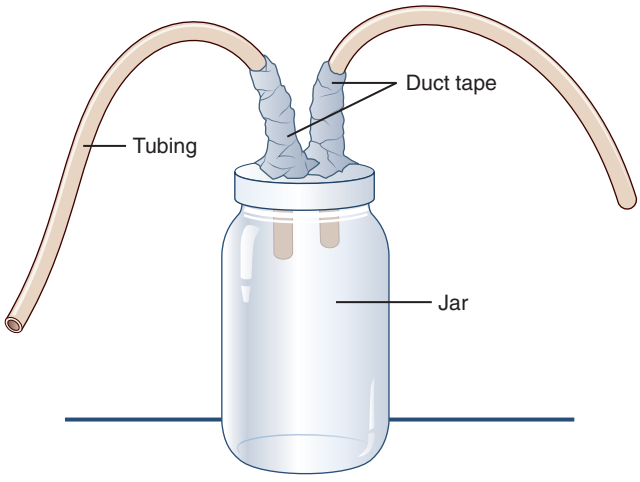


Figure 19-14. Improvised mucus trap suction device.

ryngeal suction. Portable devices may be powered by oxygen, air, or electricity, or may be manually powered (Fig. 19-13). Hand-operated units are popular because they are lightweight, compact, reliable, and inexpensive. All units should have large-bore, nonkinking suction tubing, an unbreakable collection container, and a sterile disposable suction catheter.

Flexible ("French") suction catheters are used to suction the nose, mouth, and oropharynx, whereas rigid suction catheters are used to suction the mouth and oropharynx. These suction catheters should not be inserted beyond the base of the tongue. Adults should not be suctioned for more than 10 to 15 seconds to prevent oxygen deprivation; children should be suctioned for less time. Care should be taken when using rigid suction catheters in children because stimulation of the oropharynx may lead to bradycardia. A bulb syringe may be used to suction the nose and mouth of infants up to 4 months of age because they are obligate nose-breathers.

If time permits and supplies are available, a "mucus trap" suction device can be improvised from a jar with two holes poked in its lid and two tubes or straws duct-taped into the holes (Fig. 19-14). One straw goes to the rescuer, who provides suction, and the other is directed toward whatever has accumulated in the airway. The jar serves to trap the removed secretions, preventing the rescuer from suctioning bodily fluids or foreign substances, such as mud, directly into his or her own mouth.

In the absence of suction or in the presence of large pieces of foreign material, visible debris can be swept from the mouth of an unconscious victim using a finger that is gloved or wrapped in cloth. When using this "finger sweep" technique on infants and children, be sure to use your little finger. Never attempt to blindly sweep the mouth if you do not see debris. Also, do not probe the mouth of a conscious victim as this may precipitate gagging, vomiting, and aspiration. Victims without risk for cervical spine injury may be placed in the recovery position (with their head turned to one side) to facilitate clearing their mouths.

SUPPLEMENTAL OXYGEN

Supplemental oxygen therapy should be provided to all sick or injured people with cardiac disease, respiratory distress, shock, or trauma, even if their measured arterial oxygen tension is normal. Start with a high oxygen concentration and then titrate downward. Although oxygen should never be withheld from a hypoxic patient with respiratory distress, care should be exercised when administering oxygen to patients with chronic hypercarbia (e.g., chronic obstructive pulmonary disease patients). Unmonitored treatment of such patients with high oxygen concentrations can result in respiratory depression from diminished hypoxic ventilatory drive.

A variety of oxygen delivery techniques may be employed, depending on the desired oxygen concentration and clinical circumstances. Please refer to Chapter 69, Emergency Oxygen Administration, for more details.

VENTILATION

Even with an open airway and supplemental oxygen, a person who is not adequately ventilating cannot conduct meaningful gas exchange. Adequate ventilation implies inhalation of enough air to deliver oxygen to the alveoli and exhalation of enough air to facilitate removal of carbon dioxide (CO_2). The sequence of interventions for the inadequately ventilating victim is opening the airway followed by positive-pressure ventilation: mouth-to-mouth, mouth-to-nose, mouth-to-pocket mask, or BVM.

It is advisable to use barrier protection such as a face shield when performing mouth-to-mouth or mouth-to-nose ventilation. Face shields are clear plastic or silicon sheets that feature a centrally located one-way valve. They are placed over a victim's face to allow rescue breathing while preventing direct contact with the victim. Face shields are compact, flexible, and portable. They are manufactured by a number of companies.

Rescue Breathing

Rescue breathing techniques using exhaled air from the rescuer will deliver about 16% to 17% inspired oxygen concentration to the victim, ideally producing an alveolar oxygen tension of 80 mm Hg.

Mouth-to-Mouth

Mouth-to-mouth ventilation is an efficient approach to assisting ventilation. Failure to use a barrier device during mouth-to-mouth ventilation places the rescuer at risk for exposure to infectious bodily fluids.

Technique:

1. Open the airway by the head-tilt with chin-lift approach (if no cervical spine trauma is suspected).
2. If needed, clear the airway of vomitus, secretions, and foreign bodies.
3. Pinch the victim's nostrils closed with the finger and thumb of one hand; the heel of that same hand may be placed on the forehead to maintain head tilt.
4. Support the chin with the other hand, holding the mouth slightly open.
5. Take a deep breath.
6. Place your mouth around the victim's mouth, which should be open, and make a tight seal with your lips against the victim's face.
7. Exhale slowly into the victim's mouth until you see the chest rise and feel resistance to the flow of your breath.
8. Break contact with the victim's mouth to allow passive exhalation.

Mouth-to-Nose

Some people have conditions (e.g., injuries to the mouth or mandible, or missing teeth or dentures) that make mouth-to-mouth ventilation ineffective. Under such circumstances, mouth-to-nose ventilation should be considered. The mouth-to-nose technique is similar to mouth-to-mouth with a few specific differences.

Technique:

1. Open the airway by the head-tilt with chin-lift approach (if no cervical spine trauma is suspected).
2. The victim's nose is left open.
3. Maintain a head tilt with the heel of one hand placed on the forehead.
4. Support the chin with the other hand while sealing the victim's lips (closing the mouth) with the thumb and forefinger.
5. Take a deep breath.
6. Place your mouth around the victim's nose and make a tight seal with your lips against the victim's face.
7. Exhale slowly into the victim's nose until you see the chest rise and feel resistance to the flow of your breath.
8. Break contact with the victim's nose and open the victim's mouth to allow passive exhalation.

Mouth-to-Mask

Mouth-to-mask ventilation is the safest, most effective technique for rescue breathing (Fig. 19-15). The pocket face mask (or similar barrier device) allows the rescuer to provide ventilation without direct contact with the victim's mouth and nose. The mask has a one-way valve in the stem to prevent exhaled gases and body fluids from reaching the health care provider. Additionally, a disposable high-efficiency particulate air (HEPA) filter may be inserted into the pocket mask to trap infectious air droplets and secretions.

The pocket face mask is made of a soft plastic material and can be folded and carried in a pocket. Some masks are available with an oxygen inlet, allowing supplemental oxygen administration. These devices are produced by a number of manufacturers and are available in a number of sizes.

Technique:

1. Open the airway by the head-tilt with chin-lift approach (if no cervical spine trauma is suspected).
2. Connect the one-way valve to the mask.
3. If available, connect oxygen tubing to the inlet port and set the flow rate at 15 L/minute.
4. Position yourself at the head of the victim.
5. If needed, clear the airway of vomitus, secretions, and foreign bodies.
6. Insert an oral or nasopharyngeal airway.
7. Place the mask on the victim's face.
8. Apply pressure to both sides of the mask with the thumb side of the palms, creating an airtight seal. Apply upward pressure to the mandible (jaw thrust) using the index, middle, and ring fingers of both hands while maintaining a head tilt.
9. Take a deep breath and exhale into the port of the one-way valve, observing for chest rise.
10. Allow the victim to passively exhale between breaths.

Bag-Valve-Mask Ventilation

The self-inflating ventilation bag with face mask, the BVM device, provides an emergent means of ventilation with high concentrations of oxygen. The device is equipped with several valves that allow for the coordinated flow of air into and out of the victim. This includes a one-way valve that prevents exhaled air from reentering the victim's lungs (Fig. 19-16). When attached to a high-flow oxygen source (15 L/min), the BVM can supply an O_2 concentration of nearly 100%. The adapter for the face mask is interchangeable with an ETT, so the same bag can be used after intubation.

Competence with the BVM is a vital emergency skill and a prerequisite to using paralytic agents to intubate a person.

Figure 19-15. Rescuer using mouth-to-mask ventilation. The rescuer is providing rescue breathing using face mask with supplementary oxygen during CPR. The rescuer is using cephalic technique (rescuer positioned at top of victim's head). Both of the rescuer's hands are used to hold the mask securely in position while keeping the victim's airway open. The rescuer's thumbs and forefingers hold the mask in place while the third, fourth, and fifth fingers of each hand lift the jaw (jaw thrust) and maintain an open airway with the head tilted (as shown). Alternatively, the thumbs and a portion of the rescuer's palms can anchor the mask and the index and remaining fingers can lift the jaw, holding it against the mask. (Redrawn from Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Part 6: Advanced cardiovascular life support. Section 3: Adjuncts for oxygenation, ventilation and airway control. The American Heart Association in collaboration with the International Liaison Committee on Resuscitation. Circulation 2000;102[8 Suppl]:I95–I104.)

When using the single-hand mask hold technique, substantial proficiency is required to use only one hand to maintain an adequate mask seal, position the victim's head, and assure airway patency, while using the other hand to ventilate. Although mastery of solo BVM technique is imperative, recruitment of another individual allows one person to perform a jaw thrust and ensure a good mask seal with both hands while the second individual squeezes the bag.

Successful BVM ventilation depends on an adequate mask seal and a patent airway. Placement of an oral airway should always be considered before BVM ventilation. Slow, gentle ventilation minimizes the risk for gastric inflation and subsequent regurgitation. The effectiveness of BVM ventilation can be determined by watching the chest rise and fall, feeling the resistance in the bag, and monitoring the victim's oxygen saturation. Smaller bag-valve devices are employed for infants and children to prevent overinflation and ensuing barotrauma.

DEFINITIVE AIRWAY MANAGEMENT

The presence of a definitive airway implies "patency and protection." Providing a definitive airway requires a tube in the trachea with the cuff inflated, secured in place, and attached to an oxygen-rich ventilation device. Whether in the wilderness or at the hospital, the inability or failure to secure a definitive airway in a timely manner can have disastrous or fatal consequences for the victim. Approaches to definitive airway management include immediate oral intubation, rapid sequence oral intubation, awake oral intubation, nasotracheal intubation, and surgical airways (e.g., cricothyrotomy).

Although the ultimate decision to intubate a patient is often complicated and may depend on a variety of clinical factors, there are a number of clinical situations that mandate definitive airway management: (1) failure of ventilation or oxygenation, (2) inability to maintain or protect the airway, (3) potential for deterioration based on the patient's clinical presentation, (4) delivery of specialized treatment, and (5) patient protection.

Immediate Oral Intubation (Crash Intubation)

Individuals who present with respiratory arrest, agonal respirations, or deep unresponsiveness require immediate oral intubation without the use of any paralytic or sedative medications. The advantages of the crash intubation are technical ease and rapidity. Disadvantages include the potential for increasing intracranial pressure from the stress of intubation and inducing vomiting and subsequent aspiration.

Awake Oral Intubation

Awake oral intubation is an approach that makes use of titrated intravenous sedation and liberal topical airway anesthesia before inspection or intubation of an awake victim's airway. The approach conveniently allows for preservation of the victim's protective airway reflexes and spontaneous respirations, while the laryngoscopist takes a gentle "awake" look at the glottis, vocal cords, and internal airway anatomy. The decision to intubate the victim immediately or perform a controlled rapid

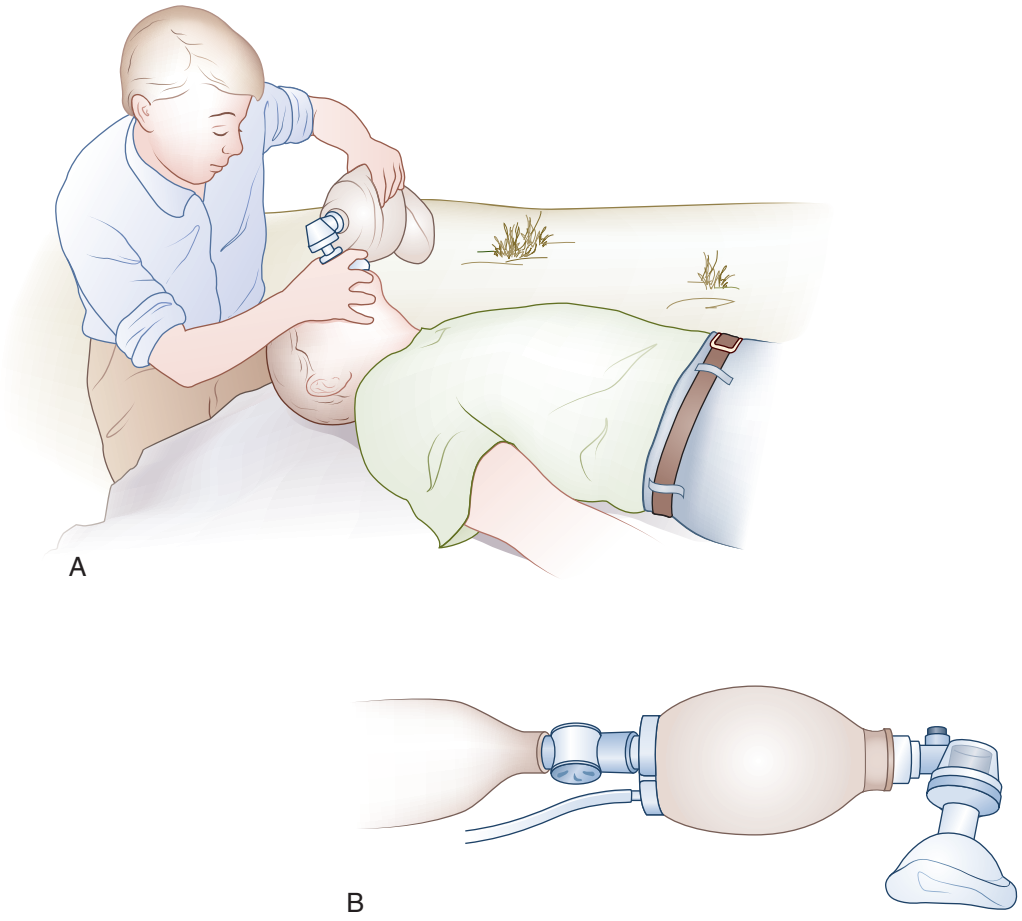


Figure 19-16. Detailed view of bag-valve-mask device (BVM). **A,** Rescuer provides ventilation with bag and mask attached to oxygen supply. The rescuer is using the E-C technique to hold the mask to the face (creating a C with the thumb and forefinger) while lifting the jaw along the bony portion of the mandible with the last three fingers of the same hand (these fingers make the E). The second hand squeezes the bag while the rescuer watches the victim's chest to ensure that the chest rises with each ventilation. The rescuer is keeping the victim's airway open with both a head tilt and a jaw thrust. **B,** Details of a BVM system with supplementary oxygen. The system consists of a self-refilling bag with an oxygen inlet, either no pop-off valve or (as shown) a pop-off valve that can be disabled during resuscitation to ensure delivery of adequate tidal volumes despite high pressures, and standard fittings (in this case, the bag is joined with a standard fitting to a mask). This system is capable of delivering high concentrations of oxygen because it contains an oxygen reservoir. If additional gas is required during ventilation, the gas is drawn from the oxygen reservoir rather than from room air, so a high concentration of oxygen can be delivered during ventilation. (Redrawn from Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Part 6: Advanced cardiovascular life support. Section 3: Adjuncts for oxygenation, ventilation and airway control. The American Heart Association in collaboration with the International Liaison Committee on Resuscitation. Circulation 2000;102[8 Suppl]:I95–I104.)

sequence oral intubation depends on the potential for progression of airway difficulty (or compromise). During the "awake" look, the clinician might elect to immediately intubate a victim with an airway burn or anaphylaxis with progressive airway swelling because that may be the most timely opportunity for airway management.

Rapid Sequence Oral Intubation

Rapid sequence intubation (RSI) is a coordinated series of procedures that allows rapid intubation of a victim without interposed BVM ventilation. Because victims requiring emergent intubation may not have fasted, BVM ventilation can inadvertently lead to gastric distention, regurgitation, and increased risk for aspiration.

To perform RSI, the victim is first preoxygenated to allow for a period of apnea without interposed assisted ventilation. Next, sequential administration of an induction agent and a rapidly acting neuromuscular blocking agent induces a state of unconsciousness and paralysis, respectively. This allows the victim to be intubated without the need for BVM ventilation. The steps that make up RSI can be thought of as the "eight Ps" (Table 19-3).

Preparation

Before RSI, careful preparation is essential to achieving success. The **SOAP ME** mnemonic summarizes the necessary preparatory steps and essential equipment (Table 19-4).

Preoxygenation

During RSI, the process of direct laryngoscopy and ETT placement precludes delivery of oxygen to the paralyzed, apneic victim. This could potentially lead to rapid arterial oxygen

desaturation (below 90%). However, preoxygenating the victim, which establishes an oxygen reservoir within the victim's lungs and body tissues, allows for a prolonged period of apnea without detrimental arterial oxygen desaturation. A victim may be preoxygenated by receiving 100% oxygen for 5 minutes before paralysis. In circumstances in which time is limited, a person can be quickly preoxygenated by taking eight vital capacity (the largest possible) breaths in rapid succession from a 100% oxygen source. Preoxygenation effectively leads to "nitrogen washout," or replacement of any room air (about 80% nitrogen, 20% oxygen) within the lungs with near 100% oxygen.

The time to desaturation after preoxygenation is determined by the duration of preoxygenation as well as the type of victim. Young children, pregnant women, and obese adults tend to desaturate more rapidly than do normal-sized adults.

TABLE 19-3. The Eight Ps of Rapid Sequence Intubation

TIME	ACTION
Zero minus 10 minutes	Preparation
Zero minus 5 minutes	Preoxygenation
Zero minus 3 minutes	Pretreatment
—Time zero—	Paralysis with induction
Zero plus 20–30 seconds	Protection and positioning
Zero plus 45 seconds	Placement
Zero plus 45 seconds	Proof
Zero plus 1 minute	Postintubation management

Pretreatment

During RSI, the act of laryngoscopy, as well as use of succinylcholine, a depolarizing neuromuscular blocking agent, can lead to a number of adverse effects, such as bronchospasm in patients with reactive airway disease, increased intracranial pressure, increased intraocular pressure, increased intragastric pressure, increased sympathetic discharge, and bradycardia (especially in children). Select pretreatment medications may be administered before laryngoscopy to mitigate these adverse effects; they may be remembered using the mnemonic **LOAD: l**idocaine, **o**pioid, **a**tropine, **d**efasciculation. These medications and their indications, mechanisms of actions, and doses are summarized in Table 19-5.

TABLE 19-4. SOAP ME Mnemonic

Suction	Suction should be tested and readily available at the bedside.
Oxygen	A high-flow oxygen mask and bag-valve ventilation device should be ready for use.
Airway equipment	At least two functioning laryngoscope handles and the appropriately sized and shaped laryngoscope blades should be obtained. The anticipated blade of choice should be clicked into position to ensure that the light functions properly. An endotracheal tube (ETT) should be chosen based on the patient's anatomy, and one smaller size should be prepared as well. The average adult male will require a 7.5 or 8.0 sized ETT, the average adult female a 7.0 or 7.5. In children, the ETT size may be estimated by the formula ETT size = 4+(age in years/4). The ETT cuff should be inflated to test for an air leak. A stylet should be inserted within the ETT to shape it into a configuration that will facilitate insertion into the airway. Care must be taken to ensure that the tip of the stylet does not protrude from the end of the ETT or through the small distal side port (Murphy's eye).
Pharmacy	The patient should have at least one IV, and patency of the line should be verified and ensured. The specific rapid sequence intubation medications, proper dosing, and sequence of administration should be determined, and the agents should be drawn up and labeled.
Monitoring Equipment	Cardiac monitoring, blood pressure monitoring, and pulse oximetry are mandatory for all patients. If available, an end-tidal CO_2 monitor should be readied as well. In a wilderness setting, many of these hospital-based amenities may not be available.

TABLE 19-5. Pretreatment Medications: LOAD Mnemonic

DRUG	INDICATION	MECHANISM	ADULT DOSE (IV)	PEDIATRIC DOSE (IV)	NOTES
Lidocaine	↑ ICP, RAD	↓ Intracranial response to intubation, mitigates bronchospasm in RAD	1.5 mg/kg	1.5 mg/kg	
Opioid (fentanyl)	↑ ICP, ischemic heart disease, aortic dissection	Blunts sympathetic response to laryngoscopy	3–6 µg/kg	1–3 µg/kg	Use with caution in young children
Atropine	Children < 10 yr; adults receiving a second dose of SCh	Mitigates bradycardic response to SCh	2.0 mg	0.02 mg/kg (minimum dose 0.1 mg)	
Defasciculation (pancuronium, vecuronium)	↑ ICP or globe injury	Defasciculates and mitigates ICP response to SCh	0.01 mg/kg	0.01 mg/kg	Only for adults and children > 20 kg

ICP, intracranial pressure; LOAD, lidocaine, opioid, atropine, defasciculation; RAD, reactive airway disease; SCh, succinylcholine.

From Mahadevan SV, Garmel GM (eds): An Introduction to Clinical Emergency Medicine: Guide for Practitioners in the Emergency Department. Cambridge, UK, Cambridge University Press, 2005.

TABLE 19-6. Clinical Characteristics of Induction Agents

INDUCTION AGENTS	INDUCTION DOSE (IV)	ONSET OF ACTION	DURATION OF ACTION	BENEFITS	PRECAUTIONS
Barbiturates					
Thiopental	3–6 mg/kg (adult) 1–3 mg/kg (elderly)	<30 sec	5–10 min	↓ ICP	↓ BP Laryngospasm
Methohexital	1–3 mg/kg	<30 sec	5–10 min	↓ ICP Short duration	↓ BP Laryngospasm, Seizures
Benzodiazepines					
Midazolam	0.2–0.3 mg/kg	30–60 sec	15–30 min	Reversible Amnestic Anticonvulsant	Apnea No analgesia Variable dosing
Etomidate	0.3 mg/kg	15–45 sec	3–12 min	↓ ICP Rarely ↓ BP	Myoclonic jerks Vomiting No analgesia
Ketamine	1–2 mg/kg	45–60 sec	10–20 min	↑ BP Bronchodilator Dissociative amnesia	↑ Secretions ↑ ICP Emergence phenomenon
Propofol	1.5–3 mg/kg	15–45 sec	5–10 min		

BP, blood pressure; ICP, intracranial pressure.
From Mahadevan SV, Garmel GM (eds): An Introduction to Clinical Emergency Medicine: Guide for Practitioners in the Emergency Department. Cambridge, UK, Cambridge University Press, 2005.

Paralysis (with Induction)

The next step in RSI is rapid intravenous (IV) administration of an induction agent to produce complete loss of consciousness, followed immediately by administration of a neuromuscular blocking agent (NMBA) to induce complete motor paralysis.

Induction Agents. All patients, with few exceptions, should receive an induction agent before neuromuscular blockade. Paralyzing a patient without providing adequate sedation can lead to detrimental physiologic and undesirable psychological sequelae. Additionally, when induction agents are combined with NMBAs, they enhance muscle relaxation, thereby creating improved intubating conditions.

There is no single induction agent of choice for RSI in the field or emergency department. The choice of an induction agent is based on the victim's clinical circumstances and the agent's own specific attributes. The clinical characteristics of the most commonly used induction agents are summarized in Table 19-6.

Neuromuscular Blockade. NMBAs are used to completely paralyze the patient, facilitating rapid endotracheal intubation. These agents do not provide any analgesia, sedation, or amnesia. The ideal NMBA should have rapid onset of action, short duration of action, and few adverse side effects. Succinylcholine (SCh), a depolarizing NMBA, comes closest to meeting all these traits and is the most commonly used NMBA. At the neuromuscular junction, SCh binds tightly to acetylcholine receptors, causing depolarization of the motor endplate and ensuing muscle contraction. Clinically, this initially manifests as muscle fasciculations followed by complete paralysis. IV administration of SCh results in muscle fasciculations in 10 to 15 seconds, followed by complete paralysis after 45 to 60 seconds. Because of its short duration of action, patients may begin spontaneously breathing again within 3 to 5 minutes.

The dose of SCh is 1.5 mg/kg rapid intravenous pulse (IVP) in adults; 2 mg/kg rapid IVP in children less than 10 years of age; and 3 mg/kg rapid IVP in newborns. Giving an insufficient dose of SCh can lead to an inadequately paralyzed patient and can adversely affect laryngoscopy and intubation. The main disadvantages to the use of SCh are its side effects, including muscle fasciculations, bradycardia, hyperkalemia, prolonged neuromuscular blockade, malignant hyperthermia, and trismus (masseter muscle spasm).

Nondepolarizing NMBAs, such as rocuronium, cause paralysis by competing with acetylcholine for receptors at the neuromuscular junction. Although these agents are most commonly used as defasciculating agents or for postintubation patient paralysis, they may also be used as the primary RSI paralytic agent in specific patient populations or in patients who have a contraindication to SCh. Although they possess fewer side effects than SCh, these agents are generally less effective for intubation because of their delayed time to paralysis, prolonged duration of action, or both. Specific attributes of the depolarizing and nondepolarizing NMBAs are listed in Table 19-7.

Protection

After sequential administration of the induction and paralytic agents, the patient will predictably lose consciousness and become apneic. Perform Sellick's maneuver, the application of cricoid pressure, just as the patient is noted to lose consciousness. This application of firm pressure (10 lbs) to the cricoid cartilage compresses the esophagus, preventing passive regurgitation of gastric contents and aspiration (Fig. 19-17). Sellick's maneuver should be maintained until the ETT has been inserted into the trachea, its position verified, and the cuff inflated. If Sellick's maneuver is applied too early, the patient may find it uncomfortable or vomit. If the patient begins actively vomiting, Sellick's maneuver should be discontinued, and the patient should be log-rolled and suctioned.

TABLE 19-7. Neuromuscular Blocking Agents

NEUROMUSCULAR BLOCKING AGENT	INTUBATING DOSE (IV)	ONSET	DURATION
Depolarizing			
Succinylcholine	1.5 mg/kg (adult) 2 mg/kg (child) 3 mg/kg (infant)	45–60 sec	6–12 min
Nondepolarizing			
Rocuronium	1.0 mg/kg	50–70 sec	30–60 min
Vecuronium	0.15 mg/kg	90–120 sec	60–75 min
Pancuronium	0.1 mg/kg	100–150 sec	120–150 min

From Mahadevan SV, Garmel GM (eds): An Introduction to Clinical Emergency Medicine: Guide for Practitioners in the Emergency Department. Cambridge, UK, Cambridge University Press, 2005.

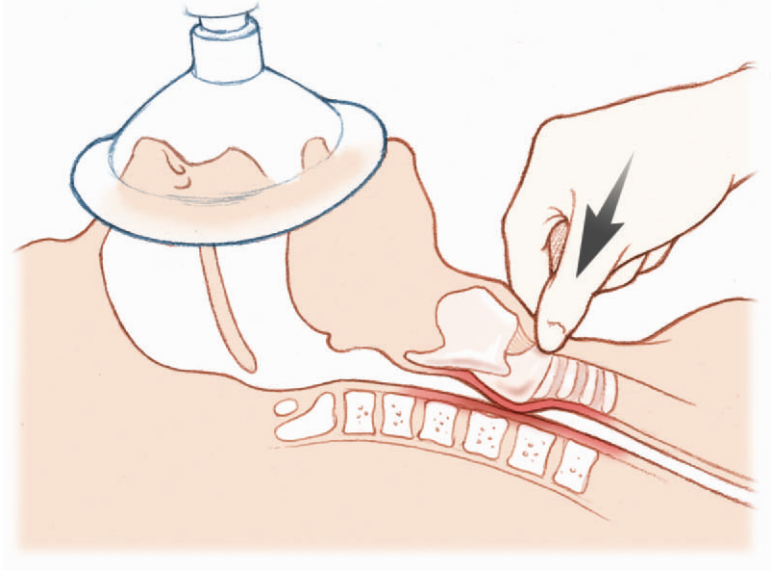


Figure 19-17. Cricoid pressure (Sellick's maneuver). (Redrawn from Mahadevan SV, Garmel GM [eds]: An Introduction to Clinical Emergency Medicine: Guide for Practitioners in the Emergency Department. Cambridge, UK, Cambridge University Press, 2005. © Chris Gralapp, www.biolumina.com.)

Cricoid pressure should also be applied when the victim requires positive-pressure ventilation before, during, or after intubation attempts. Positive-pressure ventilation, whether from rescue breathing or BVM device, may lead to gastric distention, compromised diaphragmatic excursion, and increased risk for emesis or gastric rupture. By employing Sellick's maneuver during positive-pressure ventilation, air will be directed to the lungs rather than the stomach, avoiding these complications.

Positioning

The airway may be thought of as having three separate axes: oral, pharyngeal, and laryngeal. Proper positioning of the head before laryngoscopy can help to align these three axes, improving visualization of the glottis and greatly increasing the likelihood of a successful intubation. In the neutral position, these axes are misaligned (Fig. 19-18).

Placing a small pillow or similar-sized object under the patient's occiput flexes the lower cervical spine relative to the torso and aligns the pharyngeal and laryngeal axes (Fig. 19-19). Positioning the patient in the sniffing position with extension of the head on the neck aligns all three axes (Fig. 19-20).

Patients with potential cervical spine injury should be maintained in the neutral position.

Placement

During RSI, administration of a paralytic agent will predictably lead to paralysis and apnea. With adequate preoxygenation, arterial oxygen saturations remain normal despite this apnea. Complete muscular paralysis can be confirmed by gently grasping the patient's jaw and checking for flaccidity. Wait until the patient is completely relaxed before proceeding with intubation.

With the laryngoscope in the left hand, gently open the mouth with the right hand. Insert the laryngoscope into the right side of the patient's mouth and sweep the tongue to the left. The curved (Macintosh) blade is slid into the vallecula while the straight (Miller) blade is positioned below the epiglottis. The handle is pushed along the axis of the handle at a 45-degree angle to the patient's body. Care should be taken not to

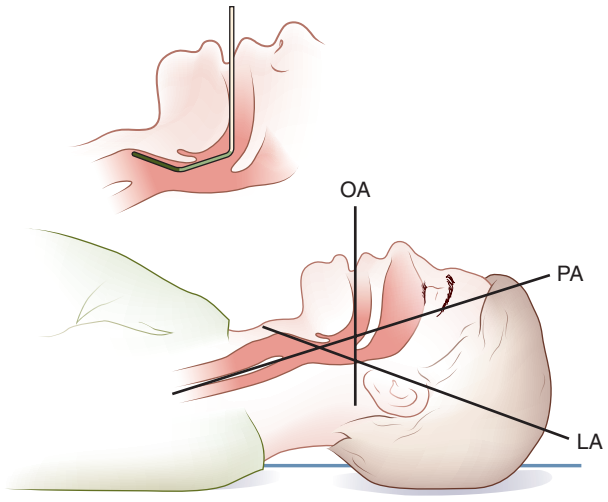


Figure 19-18. Head on bed, neutral position. PA, pharyngeal axis; OA, oral axis; LA, laryngeal axis. (Redrawn from Walls RM, Murphy MF, Luten RC, Schneider RE [eds]: Manual of Emergency Airway Management, 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2004.)

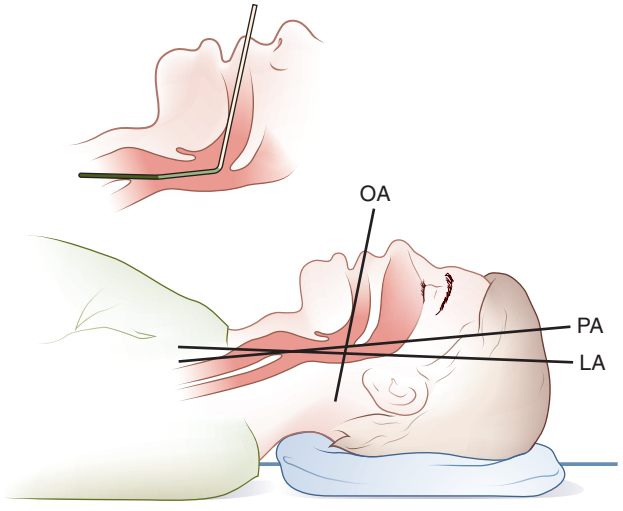


Figure 19-19. Head elevated on pad, neutral position. PA, pharyngeal axis; OA, oral axis; LA, laryngeal axis. (Redrawn from Walls RM, Murphy MF, Luten RC, Schneider RE [eds]: Manual of Emergency Airway Management, 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2004.)

"crowbar" the laryngoscope because this may fracture the patient's teeth.

If the glottic aperture is not readily visible, the intubator or an assistant may elect to perform the BURP maneuver. This involves placement of the right hand on the thyroid cartilage, followed by application of **b**ackward, **u**pward, **r**ightward **p**ressure (BURP) to help bring the glottis into view (Fig. 19-21). Resulting displacement of the thyroid cartilage backward against the cervical vertebrae, upward or as superiorly as possible, and laterally to the right has been found to significantly improve the view of the glottis during laryngoscopy.

When the glottis is clearly visible, the right hand gently inserts the ETT until the cuff is about 2 to 3 cm past the vocal cords. For adult males, the 23-cm marker of the ETT should be located

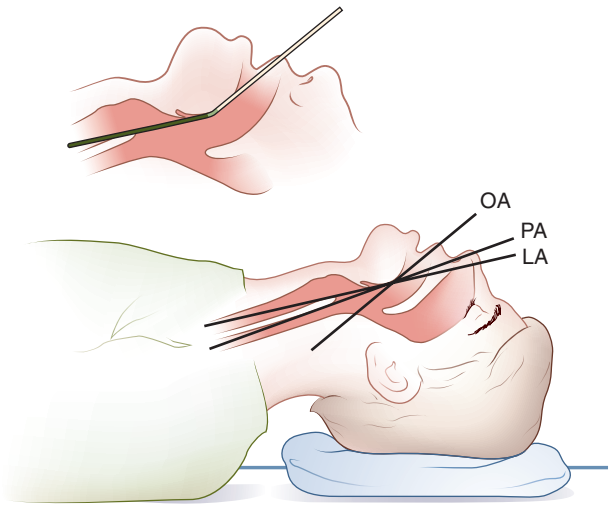


Figure 19-20. Head elevated on pad, head extended on neck. PA, pharyngeal axis; OA, oral axis; LA, laryngeal axis. (Redrawn from Walls RM, Murphy MF, Luten RC, Schneider RE [eds]: Manual of Emergency Airway Management, 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2004.)

at the corner of the mouth (21 cm for adult women). Once the ETT is in place, remove the stylet and inflate the cuff. An air leak should not be audible with bag ventilation.

If the patient is adequately preoxygenated, the laryngoscopist may make multiple attempts at intubation before arterial oxygen desaturation occurs. If available, a dedicated team member should focus on the patient's cardiac rhythm, blood pressure, and oxygen saturation, alerting the intubator to any abnormalities. After any unsuccessful intubation attempt, it is important to recheck the patient's position and make any needed adjustments. Consider changing the size or type of laryngoscope blade. It is important to "change something" before taking a second look to ensure the same mistake or problem is not encountered again.

Proof (Confirmation of Endotracheal Tube Placement)

Proper ETT positioning within the trachea needs to be confirmed after every intubation. Failure to recognize an inadvertent esophageal intubation can be disastrous for both the patient and physician. Sellick's maneuver should not be released until confirmation of correct ETT placement.

Methods used to detect ETT placement include clinical assessment, pulse oximetry, end-tidal carbon dioxide ($ETCO_2$) detection, and aspiration techniques. Chest radiography assesses ETT position but does not confirm ETT placement within the trachea.

Clinical Assessment

Classic clinical observations used to confirm correct ETT placement include (1) seeing the ETT passing through the vocal cords during intubation, (2) auscultation of clear and equal breath sounds over both lung fields, (3) absence of breath sounds when auscultating over the stomach, (4) observation of symmetrical chest rise during ventilation, and (5) observation of "fogging" of the ETT during ventilation. These clinical findings are subject to failure and should not be the sole means for confirming ETT placement.

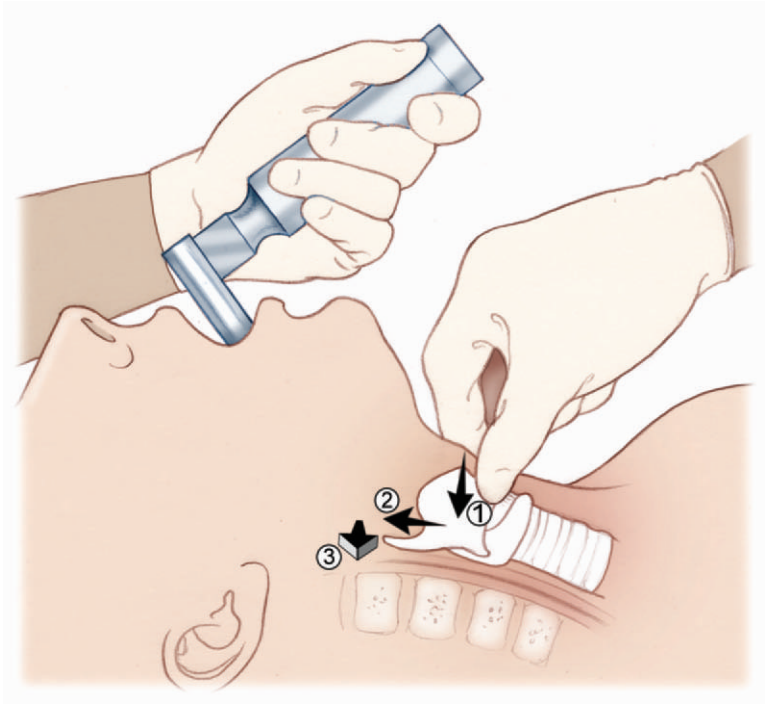


Figure 19-21. BURP maneuver. (Redrawn from Mahadevan SV, Garmel GM [eds]: An Introduction to Clinical Emergency Medicine: Guide for Practitioners in the Emergency Department. Cambridge, UK, Cambridge University Press, 2005. © Chris Gralapp, www.biolumina.com.)

Pulse Oximetry

A drop in oxygen saturation following intubation should raise concern for an esophageal intubation; however, this drop may be delayed for several minutes if the patient was adequately preoxygenated. For certain patients (e.g., hypotensive), the oxygen saturation measurement may be unreliable or difficult to detect. Although pulse oximetry is important to assess after intubation, it should not be the primary indicator of successful ETT placement.

End-Tidal Carbon Dioxide Detection

Detection of exhaled CO_2 is a highly reliable method for identifying proper placement of the ETT within the trachea. A colorimetric $ETCO_2$ detector is a small disposable device that connects between the bag and the ETT. When this device detects $ETCO_2$, its colorimetric indicator changes from purple to yellow; absence of this color change indicates the tube is incorrectly placed in the esophagus. A false-positive color change may occur if the tube is placed just above the glottis. A false-negative color change may occur (even with correct ETT placement) in some cases of cardiac arrest and profound circulatory collapse as CO_2 production and delivery to the lungs abruptly declines.

Aspiration Devices

Aspiration appliances, such as the bulb aspiration and syringe aspiration devices, may also be used for confirmation of ETT placement. The bulb aspiration device is a round, compressible plastic globe ("turkey baster") that is first compressed and deflated, then placed within the ETT and released. If the bulb reexpands rapidly, the ETT is likely in the trachea, whereas failure to reexpand or delayed reexpansion suggests the ETT is in the esophagus. Syringe aspiration devices are large syringes (usually 30 mL) that are inserted into the ETT. The syringe plunger is then drawn back rapidly to allow brisk aspiration of a large amount of air. The rapid, easy flow of air suggests a tracheal intubation, whereas resistance suggests esophageal intubation.

Postintubation Management

After verification of correct placement of the ETT within the trachea, the tube should be secured (taped or tied) in place to ensure it does not move or migrate. The patient's blood pressure and other vital signs should be repeated. Bradycardia following intubation is worrisome for hypoxia and should prompt exclusion of an esophageal intubation. Hypertension after intubation suggests inadequate sedation. Hypotension after intubation may be the result of a tension pneumothorax, decreased venous return, the induction agent, or a cardiac cause.

After intubation, consideration should be given to long-term sedation and paralysis. A benzodiazepine such as lorazepam (0.05–0.1 mg/kg) may be given initially for sedation and repeated for any signs of awareness. Nondepolarizing NMBAs, such as pancuronium (0.1 mg/kg) or vecuronium (0.1 mg/kg), may be used for long-term paralysis; a repeat dose (one third of the initial dose) may be given after 45 to 60 minutes if any motor activity is detected. An opioid agent such as morphine sulfate (0.1 mg/kg) may also be administered for additional patient comfort.

Nasotracheal Intubation

There are certain clinical circumstances, such as the spontaneously breathing patient presenting with a difficult airway, in which RSI may not be advisable. In the patient with anatomic features that may pose a challenge to RSI and BVM ventilation, awake nasotracheal intubation (NTI) may provide a better

alternative because it can be performed while preserving the patient's spontaneous respirations.

Technique:

1. Prepare the mucosa to facilitate tube passage and minimize the risk for epistaxis. Instill 2 to 3 drops of a topical vasoconstrictor agent such as Neo-Synephrine (0.25%) or oxymetazoline (0.05%) in each nare. Anesthetize the nasal mucosa using a 4% cocaine pack or 2% lidocaine jelly.
2. Select a cuffed ETT size 0.5 to 1.0 mm smaller than would be used for orotracheal intubation. The tube should be lubricated with a water-soluble lubricant to facilitate passage.
3. The patient may be seated or reclined while the clinician stands to the side of the patient.
4. The tube is inserted into the more patent of the two nares. The right side is preferable because the tube bevel will face the septum, thus avoiding Kiesselbach's plexus. If going through the left nares, consider inserting the tube "upside down," then rotating it such that the curve follows the posterior nasopharynx once the turbinates are passed.
5. Direct the tube straight back along the nasal floor toward the occiput, rotating it 15 to 30 degrees with advancement. Once the tube nears the glottis, listen for airflow within the tube with each breath.
6. When maximal airflow is heard, quickly and gently advance the tube. A cough will likely be heard with passage of the tube into the trachea. The tube should be advanced to 32 cm at the nares in the adult male and 27 to 28 cm in the adult female.
7. Confirm position with an $ETCO_2$ detection device. Because the tube is placed blindly, it may also be misdirected into the esophagus, piriform sinus, or vallecula (rare). When this occurs, withdraw the tube slightly, redirect, and retry. If the tube becomes caught on the vocal cords, rotate the tube slightly to realign the bevel with the cords. Occasionally, external manipulation of the larynx posteriorly or laterally with one's nondominant hand will facilitate successful passage.

NTI is absolutely contraindicated in the apneic patient because air movement is essential to the placement of the ETT. NTI is also contraindicated in patients with the possibility of cribriform plate injury, basilar skull fracture, or midface fracture, out of concern that the tube may enter the cranial vault. Patients with bleeding disorders or coagulopathy may develop massive epistaxis from NTI. Other contraindications include combativeness; increased intracranial pressure; neck hematoma; upper airway obstruction or anatomic alteration from trauma, edema, or infection; and need for immediate airway management.

Epistaxis and nasal turbinate injury from blind NTI can be greatly reduced by pretreatment with vasoconstrictor agents and proper technique. Long-term complications, such as sinusitis or turbinate destruction, are uncommon and result from multiple intubation attempts or prolonged intubation.

ALTERNATIVE AIRWAY ADJUNCTS AND TECHNIQUES

In certain wilderness conditions and settings, tracheal intubation may be difficult or impossible. Under such circumstances, alternative airway adjuncts or techniques may be employed to provide an airway. Alternative airways that require blind passage of the device into the airway may be simpler to master than passage of an ETT under direct vision. To achieve good outcomes with these devices and techniques, health care providers must maintain a high level of knowledge and skills through frequent practice and field use.

Laryngeal Mask Airway

The laryngeal mask airway (LMA) is a modified ETT with an inflatable, oval cuff ("laryngeal mask") at its base (Fig. 19-22). The LMA is blindly inserted into the pharynx and advanced until resistance is felt as the distal portion of the tube locates in the laryngopharynx. Inflation of the collar provides a seal around the laryngeal inlet, facilitating tracheal ventilation.

Although the LMA does not provide a definitive airway or ensure absolute protection against aspiration, studies have shown that aspiration is uncommon and regurgitation is less likely with an LMA than with the BVM device. The LMA provides equivalent ventilation to the tracheal tube. The LMA may have advantages over traditional endotracheal intubation when access to the victim is limited, the possibility of unstable neck injury exists, or appropriate victim positioning for tracheal intubation is impossible. However, studies report that a small proportion of patients cannot be ventilated, even when the LMA is successfully inserted.

An updated version of the original LMA, called the intubating laryngeal mask airway (ILMA), facilitates blind endotracheal intubation by allowing passage of an ET through the device and into the trachea with a high degree of success.

Esophageal-Tracheal Combitube

The esophageal-tracheal Combitube (ETC) is a double-lumen, dual-cuffed airway; one lumen functions as an esophageal airway, whereas the other performs as a tracheal airway (Fig. 19-23). The ETC is typically blindly inserted and advanced until the victim's teeth lie between two guide marks printed on the tube. The distal end of the tube most commonly finds its way into the esophagus. The pharyngeal and distal balloons are then inflated, isolating the oropharynx above the upper balloon and the esophagus (or trachea) below the lower balloon. The location (i.e., esophagus or trachea) of the distal orifice is then ascertained, and the victim is ventilated through the appropriate opening. One lumen contains ventilating side holes at the hypopharyngeal level and is closed at the distal end; the other lumen has a distal open end with a cuff similar to a tracheal tube.

The advantages of the ETC over the BVM include isolation of the airway, reduced aspiration risk, and more reliable ventilation. Because laryngoscopy and visualization of the vocal cords are not necessary for insertion, the ETC is easier to teach, learn, and practice than tracheal tube placement. Additionally, ventilation and oxygenation with the ETC compare favorably with those of the tracheal tube.

Fatal complications with the ETC may result from incorrect identification of the position (trachea or esophagus) of the distal lumen. For this reason, use of an $ETCO_2$ or esophageal detector device in conjunction with the ETC is recommended. Another possible complication is esophageal trauma.

Lighted-Stylet Intubation

Utilizing transillumination of the soft tissues of the neck, a lighted-stylet apparatus can be used to facilitate blind ETT placement within the trachea. Devices such as the Trachlight

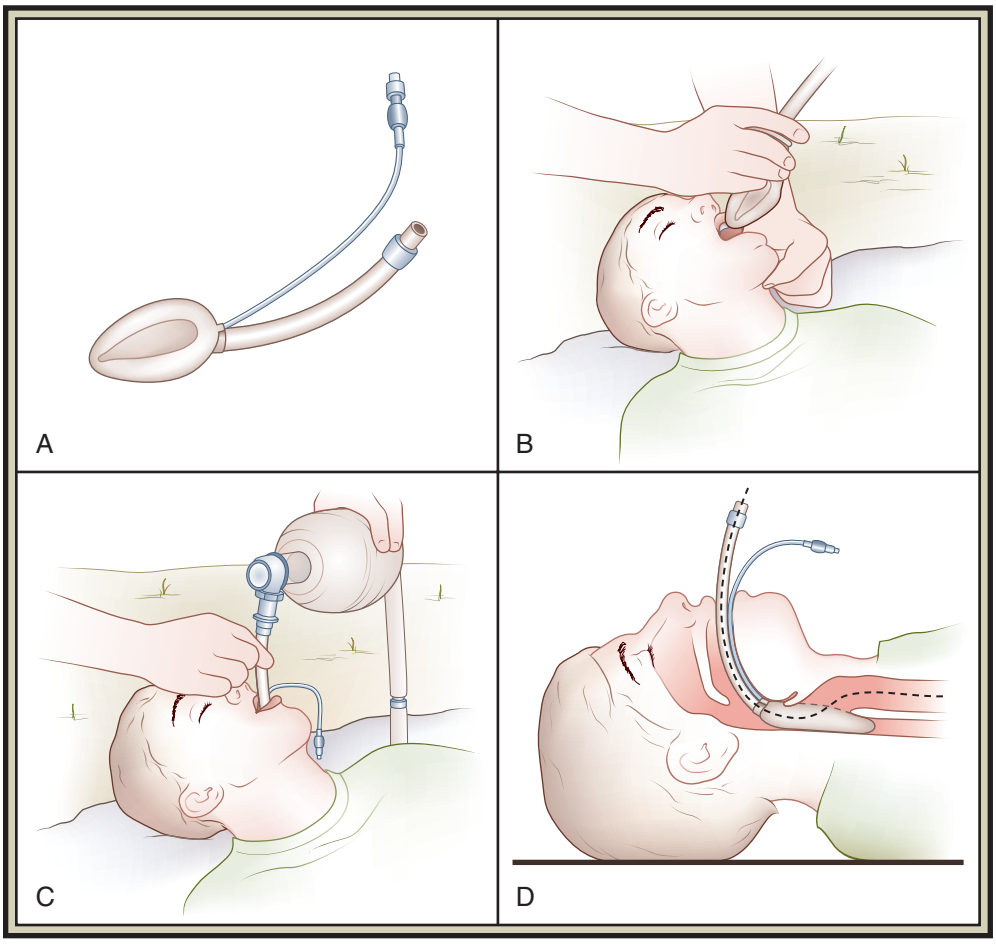


Figure 19-22. Laryngeal mask airway (LMA). **A,** LMA is an adjunctive airway that consists of a tube with a cuffed mask-like projection at the distal end. **B,** LMA is introduced through the mouth into pharynx. **C,** Once the LMA is in position, a clear, secure airway is present. **D** (Anatomic detail), During insertion, the LMA is advanced until resistance is felt as the distal portion of tube locates in the hypopharynx. The cuff is then inflated. This seals the larynx and leaves the distal opening of the tube just above the glottis, providing a clear, secure airway (*dotted line*). (Redrawn from Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Part 6: Advanced cardiovascular life support. Section 3. Adjuncts for oxygenation, ventilation and airway control. The American Heart Association in collaboration with the International Liaison Committee on Resuscitation. Circulation 2000; 102[8 Suppl]:I95–I104.)

(Laerdal Medical, Armonk, NY) take advantage of the anterior location of the trachea relative to the esophagus. When the ETT and lignted stylet correctly enter the glottic opening, a well-defined, circumscribed glow can readily be seen in the anterior neck. If the tip of the tube is correctly placed in the esophagus, the light glow is diffuse and not readily seen. Lighted stylets may have a role in difficult airways owing to anatomic difficulties (e.g., restricted mouth opening), severe facial trauma, and victims with limited neck mobility. Despite the absence of absolute contraindications to the use of the lighted stylet, this device should probably be avoided in any victim with an unknown cause of upper airway obstruction.

Retrograde Intubation

Retrograde intubation is an invasive technique that involves needle puncture of the cricothyroid membrane followed by threading of a guidewire retrograde through the vocal cords and into the mouth or nose. The wire is then used to guide the ETT through the glottis. Kits are commercially available. Retrograde intubation may have a role in difficult airways owing to anatomic abnormalities, upper airway trauma, and victims with limited neck mobility as a result of cervical spine immobilization.

Digital Intubation

Digital intubation, or tactile intubation, is a technique in which the index and long fingers of the nondominant hand are used to identify the epiglottis and then manually direct an ETT into the larynx. It may be useful when poor lighting, poor positioning, copious airway secretions, or equipment failure make laryngoscopy difficult or impossible. Digital intubation requires a profoundly unresponsive victim and is relatively contraindicated in the semiconscious victim with intact oropharyngeal reflexes. Other relative contraindications include caustic ingestions, thermal burns, and upper airway foreign bodies.

Fiberoptic Intubation

Fiberoptic techniques for endotracheal intubation have emerged as invaluable tools for difficult airway management and include the use of fiberoptic intubating bronchoscopes and rigid fiberoptic laryngoscopes. These devices may allow awake intubation in selected victims for whom RSI or insertion of a laryngoscope blade made be disadvantageous. The use of fiberoptic devices may be difficult in the setting of uncontrolled secretions or active bleeding as visualization is impaired.

SURGICAL AIRWAY MANAGEMENT

Surgical airway management entails the creation of an opening directly into the trachea by surgical means and includes both

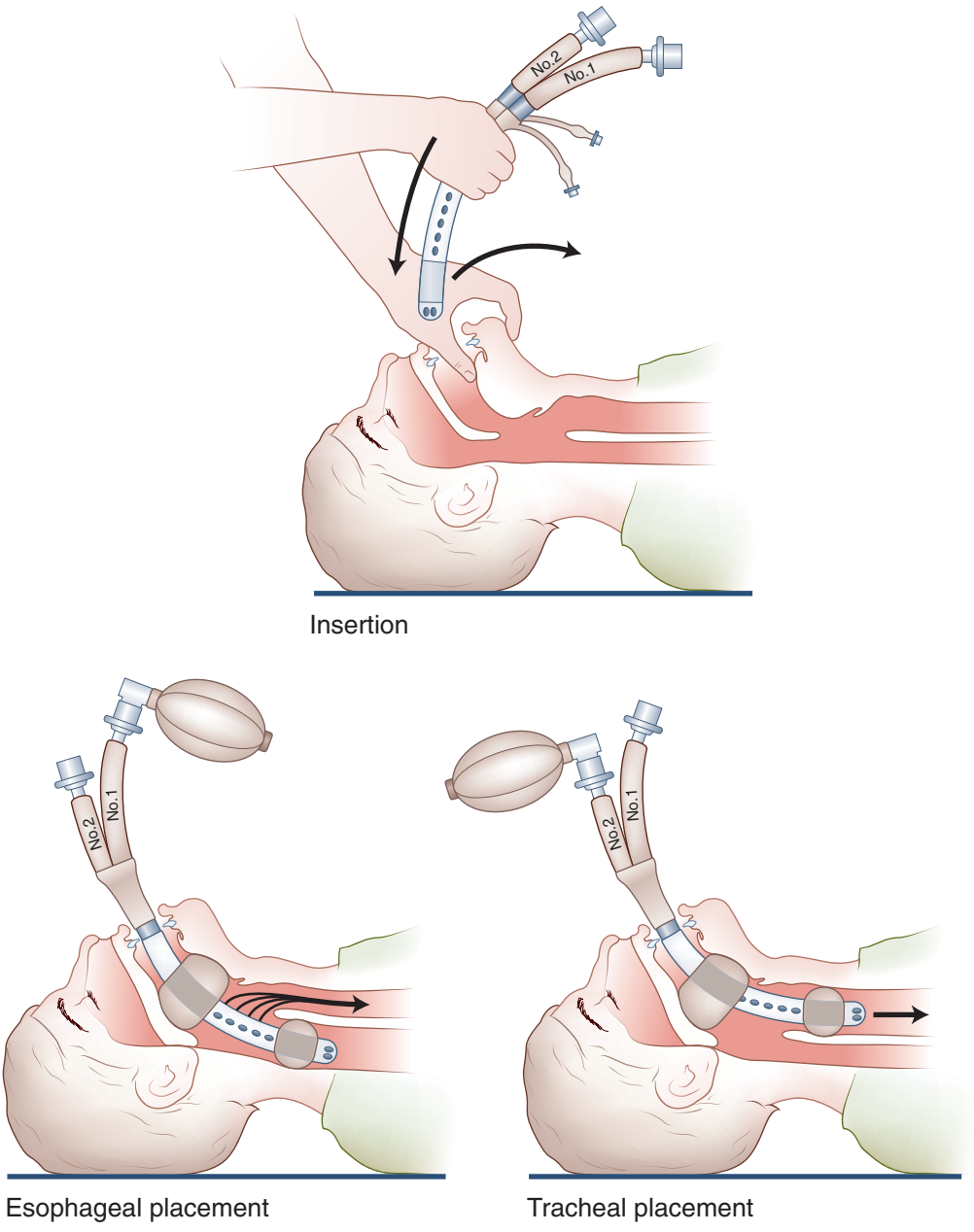


Figure 19-23. Esophageal-tracheal Combitube. (Redrawn from Skinner D, et al: Cambridge Textbook of Accident and Emergency Medicine. Cambridge, UK, Cambridge University Press, 1997.)

cricothyrotomy and needle cricothyrotomy with percutaneous transtracheal ventilation.

Cricothyrotomy

Cricothyrotomy is the creation of a surgical opening through the cricothyroid membrane to allow placement of an ETT or cuffed tracheostomy tube. The proximal ends of these tubes can be connected to a BVM for oxygenation and ventilation. The primary indication for cricothyrotomy is the need for a definitive airway in the victim in whom orotracheal or blind nasotracheal intubation (BNTI) has failed, is contraindicated, or is extremely difficult. Clinical scenarios include the victim with severe facial trauma for whom conventional airway management is extremely complicated or unfeasible, the victim with severe laryngeal edema, or the victim with upper airway foreign-body obstruction that cannot be alleviated by the Heimlich maneuver or direct laryngoscopy.

The primary contraindication to cricothyrotomy is young age. Because of anatomic considerations, the procedure is extremely difficult in children younger than 10 to 12 years of age and is generally avoided in this population. Other contraindications to cricothyrotomy include preexisting tracheal or laryngeal pathology, anatomic obliteration of the landmarks (e.g., hematoma), coagulopathy, and operator inexperience with the procedure. Complications of cricothyrotomy include incorrect airway placement, hemorrhage, tracheal or laryngeal injury, infection, pneumomediastinum, subglottic stenosis, and voice change.

Standard Surgical Cricothyrotomy

Indications

- Failure of orotracheal or nasotracheal intubation in a patient requiring definitive airway management
- Contraindication to orotracheal or nasotracheal intubation in a patient requiring definitive airway management
- Chemically paralyzed patient who cannot be BVM ventilated
- Destruction of the central face, mouth, or nose without means of maintaining the airway
- Severe supraglottic airway obstruction

Contraindications

- No absolute contraindications.
- Relative contraindications:
  - Age younger than 8 years
  - Anterior neck hematoma or infection
  - Previous cricothyrotomy
  - Coagulopathy
  - Tracheal tumor or mass

Equipment

- Scalpel with no. 11 blade
- Tracheal hook
- Trousseau dilator
- No. 4 Shiley cuffed tracheostomy tube with introducer and riser
- Betadine antiseptic solution
- 4×4 gauze pads
- 3-0 silk stay sutures or twill tape

Procedure (Fig. 19-24)

1. Identify the thyroid cartilage, the only V-shaped structure encountered when palpating up the neck. The cricothyroid space is the gap immediately caudad to the thyroid cartilage.
2. Prepare the neck in sterile fashion. With the nondominant hand holding the thyroid cartilage, make a vertical midline incision from the thyroid cartilage caudad about 3 to 4 cm (**A**).
3. Place a tracheal hook through the cricothyroid space and hold cephalad traction with the nondominant hand (**B–D**).

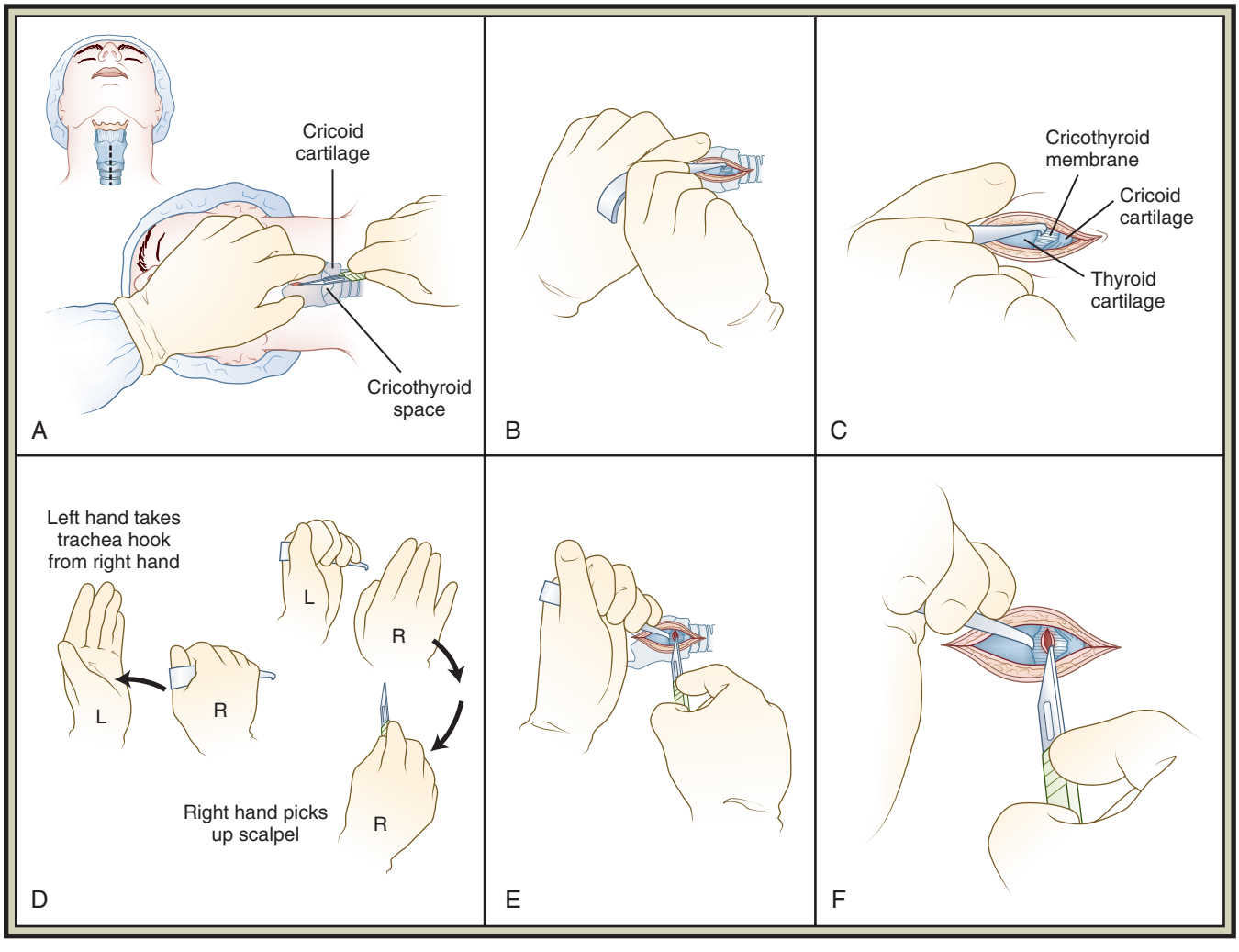


Figure 19-24. Standard surgical cricothyrotomy. (Redrawn from Walls RM: Cricothyrotomy. In Rosen P, Chan TC, Vilke GM, Sternbach G [eds]: Atlas of Emergency Procedures. St. Louis, Mosby, 2001.)

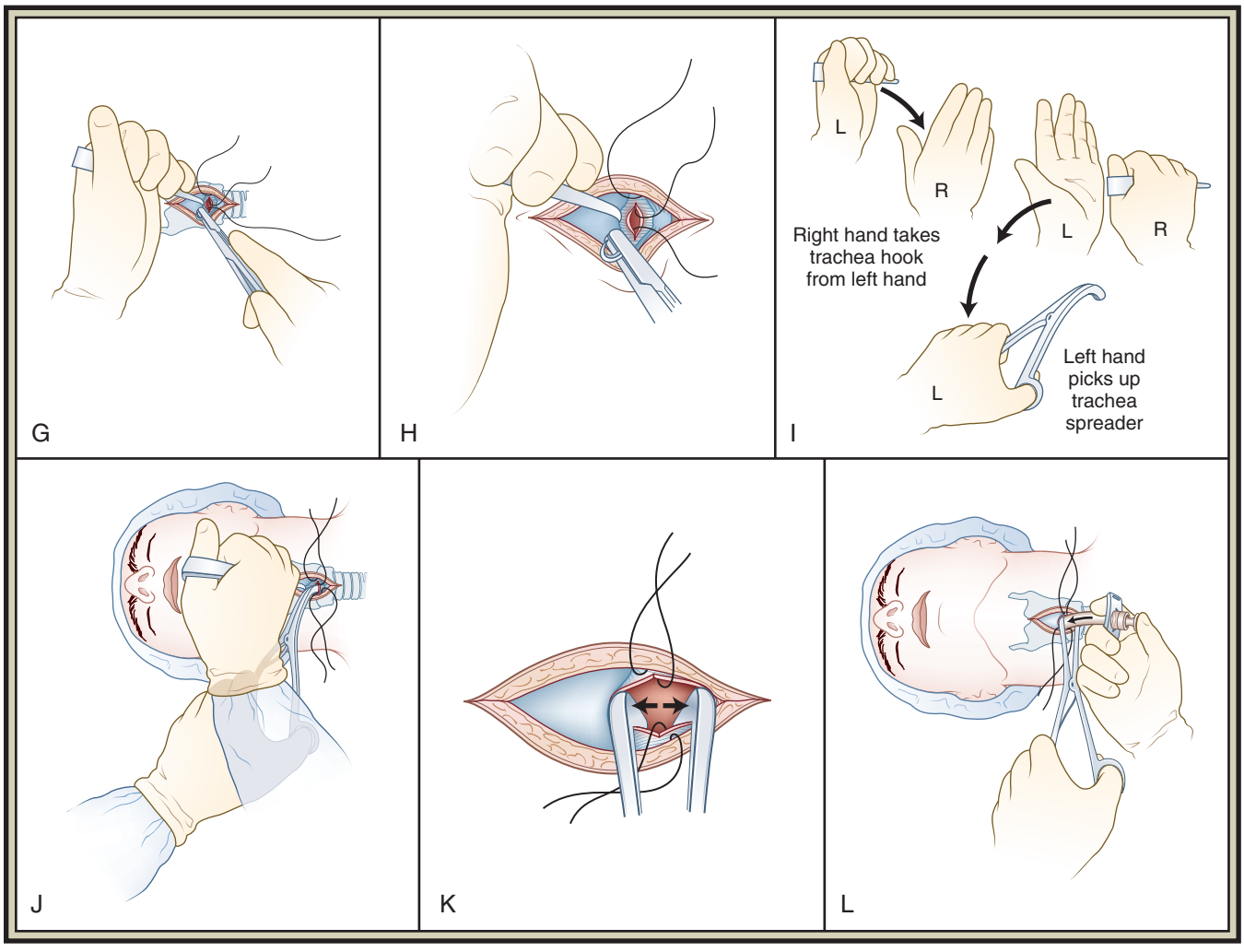


Figure 19-24, cont'd. Standard surgical cricothyrotomy.

4. Make a transverse incision across the exposed cricothyroid membrane (**E, F**). If time permits, insert stay sutures in the trachea (**G, H**). Then dilate the space using the Trousseau dilator, while maintaining traction with the hook (**I–K**).
5. Place a Shiley cuffed tracheostomy tube through the cricothyroid membrane, initially aiming posteriorly, then redirecting caudad once through the membrane (**L**). The hook may be removed before insertion to avoid damaging the tube.

Rapid Four-Step Cricothyrotomy

Technique:

1. From the position at the head of the victim, palpate and identify the landmarks, especially the cricoid and thyroid cartilages and the cricothyroid membrane (Fig. 19-25).
2. Incise the cricothyroid membrane and overlying skin simultaneously with a single horizontal 1.5-cm incision, using a no. 20 scalpel. While maintaining the blade within the airway, slide a tracheal hook along the caudal side of the blade into the wound.
3. Orient the hook caudally and place gentle traction on the cricoid ring. This typically widens the incision. Then remove the blade from the airway.
4. The ET is then placed through the incision into the airway and secured.

Percutaneous Transtracheal (Translaryngeal) Jet Ventilation

An alternative surgical airway procedure is needle cricothyrotomy with percutaneous transtracheal jet ventilation (TTJV). In this technique, a transtracheal catheter is inserted through the cricothyroid membrane into the trachea and connected to a jet ventilation system that includes high-pressure tubing, an oxygen source at 50 psi, and an in-line one-way valve for intermittent administration of oxygen. One hundred percent oxygen is then

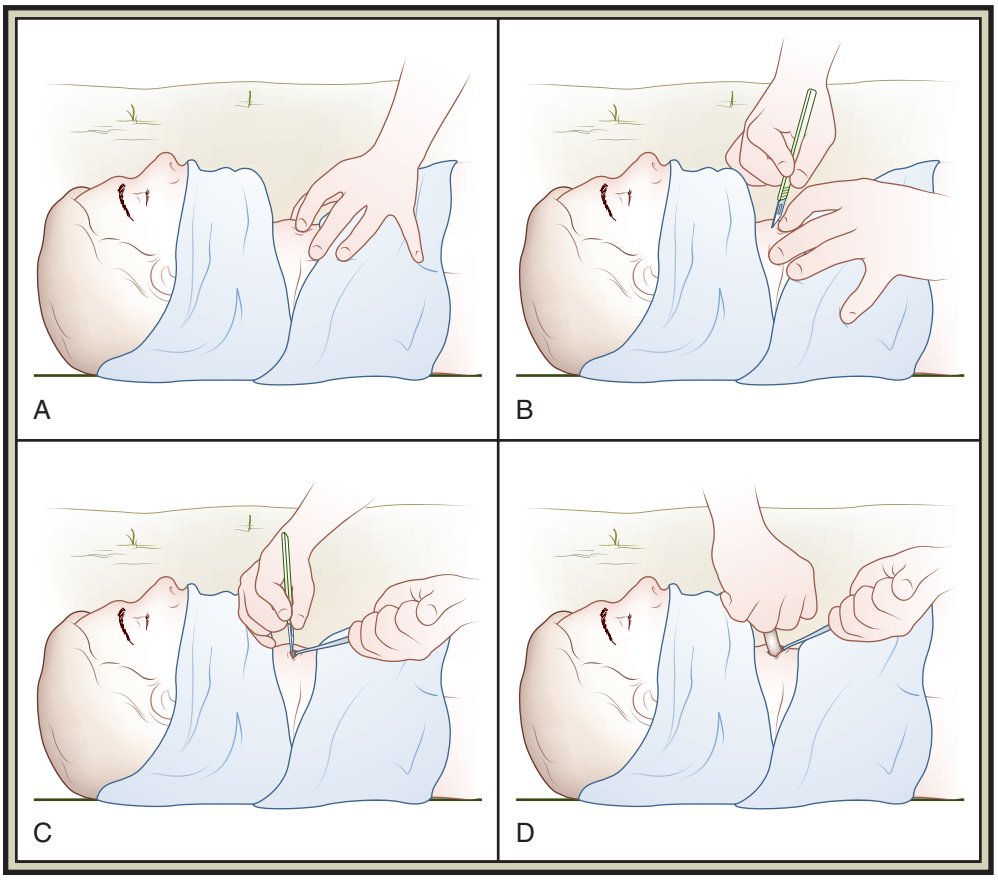


Figure 19-25. Rapid four-step cricothyrotomy. **A,** Step 1: Palpation (location of cricoid membrane externally). **B,** Step 2: Incision (horizontal incision of skin and soft tissues through cricoid membrane). **C,** Step 3: Traction (caudal traction on cricoid ring). **D,** Step 4: Intubation (passage of tracheal tube). (Redrawn from Brofeldt BT, Panacek EA, Richards JR: An easy cricothyrotomy approach: the rapid four-step technique. Acad Emerg Med 1996;3:1060.)

delivered at 12 to 20 bursts per minute. The inspiratory phase should last 1 second, whereas the expiratory phase lasts 2 to 4 seconds. Advantages of this technique include its simplicity, safety, and speed. There is typically less bleeding when compared with cricothyrotomy, and age is not a contraindication, making it the preferred surgical airway technique in children younger than 12 years. During TTJV, the upper airway must be free of obstruction to allow for complete exhalation, otherwise the victim is at risk for barotrauma from air stacking. All victims receiving TTJV should have an oral and nasal airway placed. Unlike cricothyrotomy, TTJV does not provide complete airway protection. Therefore, it should be considered a temporizing measure until a definitive airway can be established.

Technique:

1. Stand to one side of the victim at the level of the neck. Expose the neck and identify the landmarks, especially the cricoid and thyroid cartilages and the cricothyroid membrane.
2. Prepare the neck with a antiseptic solution and provide local anesthesia if time permits.
3. Immobilize the larynx between your thumb and middle finger while your index finger identifies the cricothyroid membrane.
4. Attach a large-bore (12- to 16-gauge) needle-over-catheter to a 20-mL syringe. Hold the needle and syringe in the dominant hand. Direct the needle caudally through the cricothyroid membrane at a 30-degree angle to the skin while maintaining negative pressure on the syringe. As soon as the needle enters the trachea, the syringe should fill easily with air.
5. Advance the catheter to the hub while simultaneously removing the needle. Reconfirm catheter position within the trachea.
6. Connect the catheter to the jet ventilation system.

Commercial kits with cannulas designed for cricothyrotomy or percutaneous translaryngeal ventilation are available. Several of these employ the Seldinger technique for catheter placement, whereas others rely on direct percutaneous placement of the airway device. These kits include Nu-trake (Smiths Medical, Keene, NH), Pertrach (Engineered Medical Systems, Inc., Indianapolis, IN), Quick trach (Rusch, Duluth, GA), and Melker emergency cricothyrotomy catheter (Cook Critical Care, Bloomington, IN).

In terms of expedition kit portability, three transtracheal puncture emergency airway devices deserve special mention. LifeStat (New Orleans, LA) manufactures a keychain emergency airway set that consists of a sharp-pointed metal trocar introducer that fits through a straight metal cannula that screws into a metal extension with a universal 15-mm male adaptor. Lightweight and less than 3 inches long, the three-component apparatus is attached to a separate and detachable keychain (Fig.

19-26). Cook Critical Care offers a 6-Fr reinforced-catheter emergency transtracheal airway catheter (order number C-DTJV-6.0-7.5-BTT) with a molded Luer-Lok connection for jet ventilation or added assembly of a 15-mm adaptor for standard modes of positive-pressure ventilation. Cook Critical Care also offers the Wadhwa Emergency Airway Device (order number C-WEAD-100). This lightweight, impact-resistant assembly disassembles to yield a 12-Fr Teflon-coated cricothyrotomy catheter with a removable metal stylet (with a molded plastic Luer-Lok connection for oxygen or jet ventilation), plus a flexible nasopharyngeal airway adhered to a molded plastic flange (Fig. 19-27). Both the cricothyrotomy catheter and the NPA screw into the Wadhwa case to provide a low-resistance extension and a 15-mm (male) connection for standard positive-pressure ventilation equipment.

Under circumstances when a jet ventilator is not readily available, the clinician in the wilderness may improvise by using a self-inflating bag-valve device to ventilate the victim through the transtracheal catheter. The bag-valve device may be connected to a 3.0-mm-ID ETT adapter inserted directly into the transtracheal catheter or to a 7.5-mm-ID ETT adapter inserted into a 3-mL syringe barrel and then the transtracheal catheter (Fig. 19-28). Ventilation using such an apparatus is temporary at best, but may have utility in children younger than 5 years.

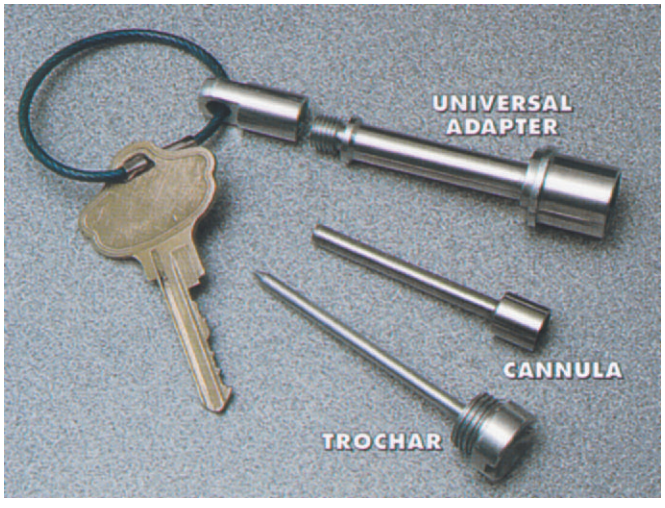


Figure 19-26. LifeStat emergency airway device. (Redrawn from LifeStat product information.)

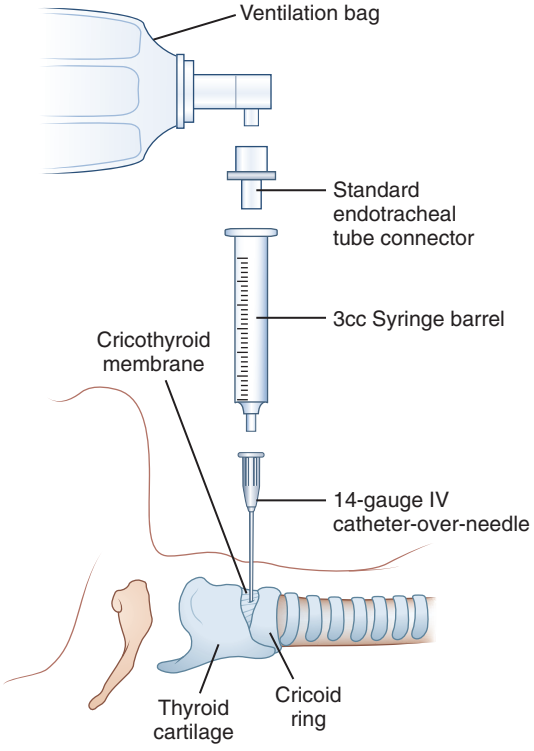


Figure 19-28. A simple setup for translaryngeal ventilation using standard equipment found in any emergency department. This setup is inadequate for adults. High-pressure (50 psi) ventilation systems are optimal. Even with the pressure relief valve on the bag-valve device turned off, only a suboptimal pressure can be developed. However, this technique may be satisfactory in infants and small children. (Redrawn from Roberts JR, Hedges JR [eds]: Clinical Procedures in Emergency Medicine, 4th ed. Philadelphia, Saunders, 2004.)

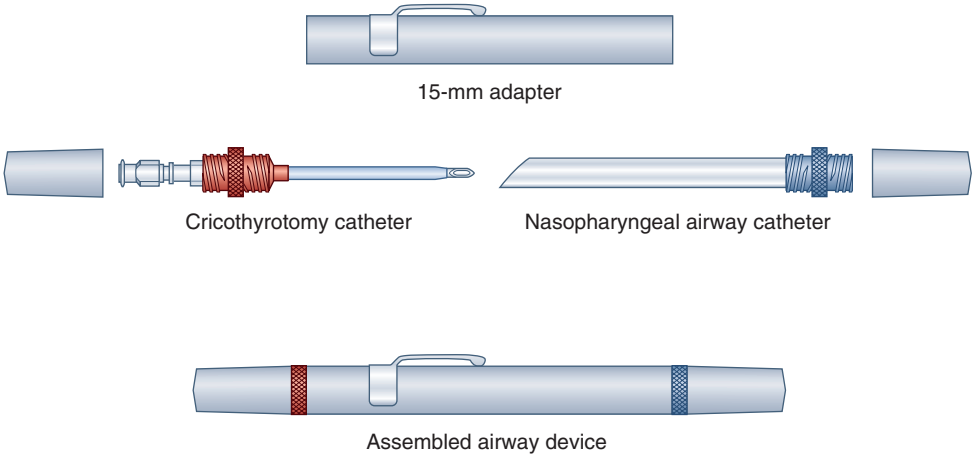


Figure 19-27. Wadhwa emergency airway device. (Redrawn from Cook Critical Care product information.)

Box 19-1. Sample Contents of a Wilderness Airway Management Kit

BASIC AIRWAY EQUIPMENT
Laerdal pocket mask
CPR Barrier MicroShield
Nasal airway kit
Oral airway kit
Stethoscope
Bulb suction device

ADVANCED AIRWAY EQUIPMENT
Bag-valve-mask (BVM) device with pediatric and adult masks
Manual suction device
Endotracheal tubes with stylet
Laryngoscope handles and blades
Magill forceps
Esophageal detector device
Colorimetric end-tidal CO_2 detector
Laryngeal mask airway and/or esophageal-tracheal Combitube
Needle cricothyrotomy catheter or device
Commercial cricothyrotomy kit
Oxygen cylinder with toggle handle
Nasal cannula, oxygen mask with strap, non-rebreather bag
Oxygen tubing

AIRWAY EQUIPMENT FOR THE WILDERNESS

Although it is neither practical nor possible to bring every type of airway device to the wilderness, the airway equipment listed in Box 19-1 is worth considering. Emergency medical kits with basic or advanced airway equipment, such as the StatKit (Banyan International Corp., Abilene, TX), are commercially available.

SUGGESTED READINGS

ATLS: Advanced Trauma Life Support for Doctors. Chicago, American College of Surgeons, 1997.

Bergeron JD, Bizjak G: First Responder, 6th ed. Upper Saddle River, NJ, Prentice Hall, 2000.

Blanda M, Gallo UE: Emergency airway management. Emerg Med Clin North Am 2003;21(1):1–26.

Butler KH, Clyne B: Management of the difficult airway: Alternative airway techniques and adjuncts. Emerg Med Clin North Am 2003;21(2):259–289.

Cummins RO (ed): ACLS Provider Manual. Dallas, American Heart Association, 2002.

Daniel JW, Pinosky ML: A simple suction device to aid in transportation of the critically ill pediatric patient. Anesthesiology 1996;85(1):220–221.

Danzl DF: Tracheal intubation and mechanical ventilation. In Tintinalli JE (ed): Emergency Medicine: A Comprehensive Study Guide, 5th ed. New York, McGraw-Hill, 2000.

Dickson AE: Emergency airway management. In Auerbach PS (ed): Wilderness Medicine: Management of Wilderness and Environmental Emergencies, 4th ed. St. Louis, Mosby, 2001.

Dieckmann R (ed): Pediatric Education for Prehospital Providers. Elk Grove Village, IL, American Academy of Pediatrics, 2000.

Doak SA: Airway management. In Hamilton G (ed): Emergency Medicine: An Approach to Clinical Problem-Solving, 2nd ed. Philadelphia, WB Saunders, 2003.

Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Part 6: Advanced cardiovascular life support. Section 3: Adjuncts for oxygenation, ventilation and airway control. The American Heart Association in collaboration with the International Liaison Committee on Resuscitation. Circulation 2000;102(8 Suppl):I95–I104.

Hazinski MF (ed): PALS Provider Manual. Dallas, American Heart Association, 2002.

Kaide CG, Hollingsworth JH: Current strategies for airway management in the trauma patient, parts 1 and 2. Traum Rep 2003;4(1, 2).

Mahadevan SV, Sovndal S: Airway management. In: Mahadevan SV, Garmel GM (eds): An Introduction to Clinical Emergency Medicine: Guide for Practitioners in the Emergency Department. Cambridge, UK: Cambridge University Press, 2005.

McGill JW, Clinton JE: Tracheal intubation. In Roberts JR, Hedges JR (eds): Clinical Procedures in Emergency Medicine, 3rd ed. Philadelphia, WB Saunders, 1998.

Miller RD: Miller's Anesthesia, 6th ed. Philadelphia, Elsevier/Churchill Livingstone, 2004.

Parr MJA, et al: Airway management. In: Skinner D, Swain A, Peyton R, Robertson C (eds): Cambridge Textbook of Accident and Emergency Medicine, 5th ed. Cambridge, UK, Cambridge University Press, 1997.

Roman AM: Non-invasive airway management. In: Tintinalli JE (ed): Emergency Medicine: A Comprehensive Study Guide, 5th ed. New York, McGraw-Hill, 2000.

Rubin M, Sadovnikoff N: Pediatric airway management. In Tintinalli JE (ed): Emergency Medicine: A Comprehensive Study Guide, 5th ed. New York, McGraw-Hill, 2000.

Sanders MJ: Mosby's Paramedic Textbook, 2nd ed. St. Louis, Mosby, 2001.

Stoy WA: Mosby's EMT—Basic Textbook, 2nd ed. St. Louis, Mosby, 1996.

Vanstrum GS: Airway. In Vanstrum GS (ed): Anesthesia in Emergency Medicine. Boston, Little, Brown, 1989.

Walls RM: Airway. In Marx JA (ed): Rosen's Emergency Medicine: Concepts and Clinical Practice, 5th ed. St. Louis, Mosby, 2002.

Walls RM, Murphy MF, Luten RC, Schneider RE (eds): Manual of Emergency Airway Management, 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2004. *This book is the standard companion manual for the airway course administered by the Airway Management Education Center (AMEC) (www.theairwaysite.com).*

Ward KR: Trauma airway management. In Harwood-Nuss A (ed): Clinical Practice of Emergency Medicine, 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2001.

Weiss EA, Donner HJ: Wilderness improvisation. In Auerbach PS (ed): Wilderness Medicine: Management of Wilderness and Environmental Emergencies, 4th ed. St. Louis, Mosby, 2001.

Wilderness Trauma, Surgical Emergencies, and Wound Management

20

Bryan R. Collier, William P. Riordan, Jr., Roger J. Nagy, and John A. Morris, Jr.

PURPOSE

This chapter is written to provide physicians and health care practitioners with a concise, pragmatic approach to the management of trauma and surgical emergencies encountered in a wilderness environment. Our focus is directed primarily at the health care professionals responsible for the well-being of expedition members.

Wilderness expedition health care providers will have varied capabilities and experience, but it is the environment (location, time from medical facilities, and conditions) and available resources that will most influence patient outcome. Complex interventions in the field are frequently impractical. With ongoing debate concerning issues ranging from the value of field resuscitation for internal hemorrhage to the method (if any) of closure of animal bite wounds, it remains clear that simple things, such as identifying injuries, establishing an airway, keeping the patient warm, and making expedient evacuation plans, most strongly influence survival.[22,25,75]

The key to successful management of wilderness emergencies is preparedness. Knowledge of the advanced trauma life support (ATLS) protocols provides a scaffold for preparation for wilderness travel. The simple, concise principles contained in ATLS concepts are well suited to the management of wilderness emergencies, where resources are scant and prompt response is essential to victim survival. Newer training programs, such as advanced wilderness life support (AWLS), will provide additional wilderness-specific learning opportunities.

WILDERNESS TRAUMA EMERGENCIES

Overview

Management of wilderness trauma is based on military and civilian prehospital principles, including rapid assessment and diagnosis of injury, treatment, and, if necessary, evacuation. These principles and those promulgated by the Committee on Trauma of the American College of Surgeons (ACS) are formally taught in the ACS ATLS program. Any physician planning to be a medical officer on a wilderness expedition should be familiar with the material taught in the ATLS program. Optimally, the complete ATLS course should be successfully completed before assuming responsibility for the care of participants in a wilderness expedition.[1–3,47]

Background

Trauma remains the leading cause of death in the first four decades of life, and in terms of trauma-related expenditures and significant long-term disability, the societal cost is staggering. Epidemiologic data indicate that death from trauma occurs in a trimodal time distribution.[5,98] The first peak occurs within seconds to minutes and represents catastrophic injury. The second peak occurs within minutes to hours after injury. Injuries responsible for the second mortality peak are those associated with significant head injury, pneumothorax, or significant blood loss. This distribution emphasizes the concept of the "golden hour," the postinjury time interval when rapid and aggressive intervention may significantly influence mortality. Unfortunately, the location of the wilderness patient and the complexity of the evacuation often prohibit definitive care within the golden hour.

The medical literature is limited regarding the incidence of injury incurred during wilderness-related activities. It is estimated that greater than 10 million Americans participate in wilderness backpacking and camping activities annually. A study by Gentile and colleagues[34] documented the injury and evacuation patterns recorded by the National Outdoor Leadership School over a 5-year period. Injuries occurred at a rate of 2.3 per 1000 person-days of exposure, with orthopedic and soft tissue injuries most frequent. Montalvo[65] analyzed case incident report files from 8 California National Park Service parks and found an injury incidence of 9.2 nonfatal events per 100,000 visits, with 78 fatalities reported in a 3-year period. In a prospective surveillance study evaluating 38,940 days of wilderness exposure on the Appalachian Trail,[12] foot blisters and diarrhea were the most common reasons a hike was prematurely ended. Leemon and Schimelpfenig[53] showed over 50% of evacuated participants of the National Outdoor Leadership School were able to return and finish their course. These studies document a low risk of injury but highlight the possible morbidity resulting from wilderness injury or illness and the need for rapid, uniform intervention.

A 2001 study from the University of Arizona[36] highlighted wilderness mortalities over the past 13 years. Alcohol was the

most common causative factor, involved in 40% of the 59 unintentional trauma deaths. In addition, 80% of these victims died immediately or before evacuation could be completed. This again emphasizes the importance of sound judgment and preparedness as keys to maximizing care to expedition members.

Establishing Priorities

There are three immediate priorities in managing wilderness trauma:

1. *Control oneself.* It is normal to feel anxious when confronted with an injured victim. However, anxiety must not be transmitted to the victim or other members of the expedition team. One must be in control of oneself to take control of the situation.
2. *Control the situation.* The first priority in controlling the situation is ensuring the safety of the uninjured members of the party. Expeditious evacuation of a victim requires that all expedition members function at maximal efficiency; even minor injuries to other members in the group can jeopardize physical strength, functional manpower, and the success of the evacuation. Although the physician member of the team may not be the expedition leader, his or her position is automatically elevated during a medical crisis. However, this does not mean that the physician should dominate the evacuation process. Although the expedition leader must rely on the medical assessment provided by the physician, the leader is best prepared to plan the evacuation.
3. *Obtain an overview of the situation.* The victim's general condition should be evaluated. Is the victim in immediate distress from a condition that requires relatively simple management, such as airway control? Is the victim in such a precarious environmental situation that he or she needs to be moved before resuscitation? Scene security may be integral to the safety of the injured and caregiver. Is the victim properly protected from the elements, including sun, wind, cold, and water? Hypothermia remains one of the most important contributors to trauma-related coagulopathies.[95]

In addition to these three priorities, McSwain and Kerstein[61] have described the golden principles of prehospital trauma care. Although not intended for wilderness medicine, several key components are applicable: ensure scene and provider safety, use primary and secondary surveys, provide cervical spine immobilization, control external hemorrhage, keep the patient warm and use warm intravenous (IV) fluid resuscitation if possible, initiate early transport, and, above all, do no further harm.

After the victim has been placed in the most stable and safe environment, the examining physician is ready to implement the ATLS-based five steps of wilderness trauma management:

1. Primary survey
2. Resuscitation
3. Secondary survey
4. Definitive plan
5. Packaging and transfer preparation

The purpose of the primary survey is to identify and begin initial management of life-threatening conditions by assessing the following (called the ABCDE of trauma care):

1. Airway maintenance and cervical spine stabilization
2. Breathing
3. Circulation, with control of significant external hemorrhage
4. Disability: neurologic status
5. Exposure/Environmental control: completely undress the victim with careful attention to the prevention of hypothermia

After the primary survey is performed, resuscitation efforts are initiated. The level of resuscitation depends on the equipment and expertise available (see Chapters 21, 81, and 83). At a minimum, resuscitation consists of control of external hemorrhage and, when available, administration of oxygen and provision of warm IV fluids.

The third step is the secondary survey, a head-to-toe evaluation of the trauma victim that uses inspection, percussion, and palpation techniques to evaluate each of the body's five regions: head and face, thorax, abdomen, skeleton, and skin. A history should be taken at the same time as performance of the secondary survey. The specifics of the mechanism of injury, if unknown to the physician or caregiver, may be of vital importance. Loss of consciousness, head injury, the height of a fall, or the species of attacking animal may influence treatment and evacuation plans, as well as contribute to the stability of the scene. The ATLS AMPLE method of rapid history taking is discussed in detail in the Secondary Survey section, later. After this survey, the examining physician should formulate a definitive plan. It is useful to record all observations on paper if the circumstances permit. Such data may prove of critical importance to evacuating or hospital personnel.

The first step in formulating a plan is to compile a list of the injuries present. The next step is to determine if any injury warrants evacuation. A determination needs to be made as to the route of evacuation: air, land, or water. Aeromedical evacuation is expensive and, depending on the environment, could pose a risk to both the victim and medical evacuation team. Aeromedical evacuation should be considered only for victims with potentially life- or limb-threatening injuries where the terrain and environment allow safe access to the patient.

Packaging the victim for evacuation is the final step. The evacuation effort requires organization, coordination, and great effort on the part of the expedition team. Transfer protocols are discussed within the respective injury sections.

Preparation for wilderness patient care should address the challenges associated with varying levels of personnel help, minimal medical resources, prolonged prehospital care, and difficult evacuation proceedings. However, with ATLS knowledge, improvisation, and an organized effort, the patient's outcome can be maximized.

Fluid Precautions in the Wilderness

A number of life-threatening viruses are transmitted through contact with bodily fluids. The Centers for Disease Control and Prevention (CDC) have established a set of standard precautions to be applied in all cases of contact with human body fluids:

1. Goggles
2. Gloves
3. Fluid-impervious gowns
4. Shoe covers and fluid-impervious leggings
5. Mask
6. Head covering

Created in 1983 and revised in 1987, the recommendations for universal precautions and the use of the aforementioned equipment have undergone no additional changes over the past

decade. Multiple types of body fluids (blood, semen, vaginal secretions, cerebrospinal, pleural, synovial, pericardial, peritoneal, and pericardial) can place a caregiver at risk. In the wilderness setting, the materials necessary for universal precautions are rarely available. However, every victim in the wilderness must be assumed to carry a communicable disease. Every effort should be made to approximate universal precautions, particularly covering the hands and eyes.

Primary Survey

Persons injured in the wilderness should be assessed and their treatment priorities established based on their mechanism of injury, vital signs, and specific injuries incurred. The victim's vital signs must be assessed quickly and efficiently, with restoration of life-preserving vital functions.

Airway

The upper airway should be assessed for patency (see Chapter 19). This rapid assessment should include inspection for signs of airway obstruction, including foreign bodies and signs of facial or tracheal fractures that may lead to airway obstruction. The chin lift (see Figure 19-6) or jaw thrust (see Figure 19-3) may be helpful in establishing an airway. If the victim can speak, the airway is likely not jeopardized, but this is not an absolute. Frequent repeated evaluation of the patient's airway, breathing and circulatory status in the familiar ABC format is mandatory. In addition, a Glasgow Coma Scale (GCS) score of 8 or less indicates the possibility of a precarious airway and requires establishment of a definitive airway.

Specific attention should be directed toward the possibility of cervical spine injury. The victim's head or neck should never be hyperextended, hyperflexed, or rotated to establish or maintain an airway. Approximately 10% of victims with head injuries or facial fractures have a concomitant cervical spine fracture. Such a fracture should be assumed to exist in any person with a significant injury above the level of the clavicle. If a situation requires removal of immobilizing devices, in-line stabilization, not traction, must be maintained.

Breathing and Ventilation

The victim's chest should be exposed and chest wall movement observed. Because oxygenation and carbon dioxide elimination require adequate function of the diaphragm, chest and abdominal musculature, and ribs, any trauma sustained by these structures will alter normal pulmonary mechanics and increase the work of breathing. Establishment of an airway in the primary survey, although critical to patient survival, does not ensure adequate oxygenation or ventilation. All trauma victims must be continuously monitored for the signs and symptoms of hypoxemia and hypercarbia.

Auscultation, observation, and palpation of the chest after establishment of the airway are integral parts of the primary survey. Absent breath sounds may indicate improper placement of an endotracheal tube. This can occur with inadvertent esophageal intubation or intubation of a single lung (right or left mainstem bronchial intubation). Diminished breath sounds or asymmetric chest wall movement can occur with pneumothorax (simple and tension), hemothorax, and tracheobronchial obstruction. Observation and palpation of the chest may identify thoracic injuries such as rib fractures, fractures with flail segments, or pneumothorax (by the presence of subcutaneous emphysema).

Circulation

Evaluation of circulation is divided into assessment of cardiac output and control of major external hemorrhage. Manometric blood pressure measurement is not easily performed in the field, although it may provide useful data. Important information regarding perfusion and oxygenation can be obtained rapidly by determining the victim's level of consciousness, assessing peripheral and central pulses, looking at skin color, and evaluating capillary refill time.

The pulse should be assessed first. Although the following are only general estimates and carry some inaccuracy, approximations of systolic blood pressure can be used if a palpable pulse is present:

1. Radial artery—80 mm Hg
2. Femoral artery—70 mm Hg
3. Carotid artery—60 mm Hg

When circulating blood volume is reduced, cerebral perfusion pressure may be critically impaired, resulting in altered level of consciousness. Do not assume that altered level of consciousness is attributable to head injury. Victims in class III shock and near death may be conscious.

Peripheral perfusion can be assessed using skin color and capillary refill. Pressure applied to the thumbnail or hypothenar eminence causes underlying tissue to blanch. In a normovolemic person, color returns to the nail bed within 2 seconds. In a hypovolemic, poorly oxygenated person, capillary refill requires more than 2 seconds.

If hypovolemia is suspected on the basis of either absent pulses or prolonged capillary refill, the examiner should immediately assess the neck veins. Distended neck veins, although a nonspecific sign, may suggest tension pneumothorax or pericardial tamponade in the context of hypotension. Flat neck veins suggest hypovolemia and hemorrhagic shock.

Major hemorrhage can occur in five anatomic areas:

1. Chest
2. Abdomen
3. Retroperitoneum
4. Thigh
5. External environment

Exsanguinating external hemorrhage should be identified and controlled during the primary survey. Blood loss should be controlled by direct pressure on the wound. Tourniquets should not be used to control external bleeding unless direct pressure fails to control the bleeding. The proper stabilization of long bone fractures (femur fractures specifically) minimizes blood loss into soft tissues. In the wilderness, little can be done about significant intrathoracic or intra-abdominal hemorrhage. In a modern trauma center, however, less than 5% of blunt trauma patients with intra-abdominal or intrathoracic injuries require surgery to control bleeding. Consequently, even significant blunt injuries in the wilderness carry a real probability for survival if basic ATLS principles are followed.

Disability and Neurologic Assessment

The neurologic assessment of the injury victim conducted during the primary survey should be rapid and efficient. The victim's level of consciousness should be established. In addition, pupillary size and reactivity should be assessed. Level of consciousness should be assessed using the GCS (Box 20-1). It is critical that the neurologic assessment be repeated hourly, particularly if evacuation is delayed. Deterioration in mental status portends a poor prognosis, although a variety of condi-

tions other than intracranial injury can affect mental status. Hypoxia, hypovolemia, and hypothermia should be expeditiously corrected.

Exposure and Environmental Control

The victim should be fully undressed and exposed, if possible in a protected environment. Garments and gear should be removed if necessary by cutting them away, unless the garments can be dried and are essential for future protection from the environment. Wet clothing must be removed early to prevent hypothermia. It is mandatory to visualize the entire victim to document and assess injury. However, this step of the primary survey should be performed with caution. First, it is imperative to cover the victim immediately after removal of clothing. Hypothermia and its effects on mental status, cardiovascular function, and coagulation is one of the most underappreciated entities in care of the trauma victim. The possibility of hypothermia should be entertained in all environments because it is most prevalent in trauma victims during warm summer months. Second, clothing and gear should not be pulled from the victim unless complete immobilization of injuries can be achieved. Expedition members may be wearing a variety of gear and clothing, including biking, skiing, or climbing helmets. Helmets should be removed with in-line stabilization of the cervical spine.

Box 20-1. Glasgow Coma Scale

This scale evaluates the degree of coma by determining the best motor, verbal, and eye opening response to standardized stimuli.

EYE OPENING	
Spontaneous	4
To voice	3
To pain	2
None	1
VERBAL RESPONSE	
Oriented	5
Confused	4
Inappropriate words	3
Incomprehensible words	2
None	1
MOTOR RESPONSE	
Obeys command	6
Localizes pain	5
Withdraw (pain)	4
Flexion (pain)	3
Extension (pain)	2
None	1
Total	**21**

Shock

The shock state has been defined in many ways. Essentially, *shock* describes the physiologic condition in which oxygen delivery to the tissues of the body is not adequate for the metabolic demands of those tissues. This results in cellular hypoxia, anaerobic metabolism, and, eventually, cell death. Shock was defined historically by John Collins Warren as "a momentary pause in the act of death." Nowhere is this adage more applicable than in the wilderness setting. Any physician who cares for trauma victims has witnessed an apparently "stable" victim rapidly deteriorate and die. With only clinical acumen, the wilderness physician must be sufficiently astute to identify the subtle signs of shock.

The treatment for shock is resuscitation, which may be defined as any intervention that restores blood flow and cellular oxygenation. The power of the primary survey lies in the fact that it is essentially a form of resuscitation. Rapid identification and treatment of conditions leading to hypoperfusion facilitate their reversal. Although the majority of trauma victims exhibiting signs of shock in the wilderness have sustained acute blood loss, a simple entity such as a pneumothorax may be responsible for shock.

Hemorrhagic shock is classified according to the blood volume that has been lost. A central question in caring for victims of blood loss in the wilderness is the reliability of vital signs in quantifying degree of hemorrhage. Associated with each class of hemorrhage are clinical signs that allow a caregiver to quantify blood loss (Table 20-1). It is evident that assessing blood pressure alone is a poor way to predict impending deterioration. When hypotension is present, a normal individual has already lost 30% of his or her blood volume and salvage may be impossible. Resuscitation should be initiated at the first sign of shock, equating to class II hemorrhage, and evacuation planned accordingly.

TABLE 20-1. Classes of Hemorrhagic Shock

	CLASS I	CLASS II	CLASS III	CLASS IV
Blood loss (mL)	Up to 750	750–1500	1500–2000	>2000
Blood loss (% blood volume)	Up to 15%	15%–30%	30%–40%	>40%
Pulse rate	<100	>100	>120	>140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure (mm Hg)	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate	14–20	20–30	30–40	>35
Urine output (mL/hr)	>30	20–30	5–15	Negligible
CNS/mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic
Fluid replacement (3:1 rule)	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

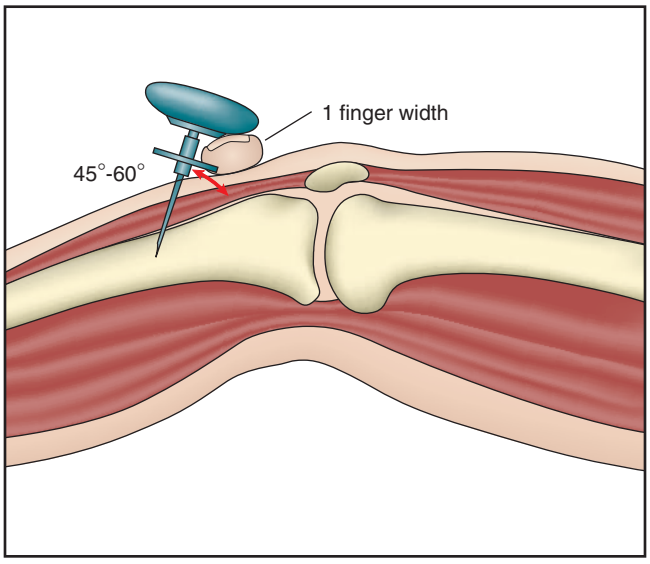


Figure 20-1. Intraosseous resuscitation performed by introducing a needle through the periosteum of the tibia inferior to the tibial tuberosity.

Vascular Access

Vascular access must be obtained promptly. The standard method of obtaining access is by insertion of two large-bore (16-gauge or larger) catheters, preferably into peripheral veins of the upper extremity. Alternatives include using lower extremity veins and obtaining central venous access. If peripheral access cannot be secured, the femoral vein should be the next site attempted. Advantages of femoral vein access include ease of cannulation relative to jugular and subclavian access, and fewer complications.[96]

Depending on expertise, the internal jugular and subclavian veins may be accessed. Despite a higher incidence of complications compared with peripheral access, central access complication rates in trauma centers have been demonstrated to be less than 5% in most studies.[96] If peripheral access is inadequate or unobtainable, it is clear that the next site attempted should be determined by the provider's level of confidence and expertise. As a last resort, venous cutdown may be considered. Despite fewer complications than central access, cutdowns are experience and equipment dependent and not recommended in the wilderness environment.

Children in the wilderness younger than 6 years of age in whom venous access cannot be obtained should undergo intraosseous resuscitation. This is accomplished by introducing a needle through the periosteum of the tibia just inferior to the tibial tuberosity (Fig. 20-1). Needles that are 18- to 20-gauge are preferable, but any needle strong enough to penetrate the periosteum without bending can be used.

Of the resuscitative therapies that potentially may be initiated in a well-prepared expedition, volume resuscitation deserves considerable attention. Fluid resuscitation in trauma has been a contentious topic, perhaps overly so, relative to fluid type and amounts used. A number of recent studies focusing on the prehospital administration of fluids in trauma victims not only rehashed the fluid composition debate but called into question the efficacy of prehospital resuscitation.[75] Although further prospective trials are needed relative to fluid type and prehospital use, an impressive compilation of data has been amassed looking at resuscitative fluids in the trauma victim. Past studies have not only compared colloids with crystalloids[91] but explored the use of blood and plasma substitutes, and hypertonic saline. An analysis of the details of such studies is beyond the scope of this chapter, but a summary of fluid recommendations is in order.

In small volumes, hypertonic saline has been shown to be an effective resuscitative fluid, and its efficacy in closed head injury is under evaluation. Currently, no improvement in survival has been demonstrated using hypertonic saline compared with crystalloid, and its use has been associated with hypokalemia, pulmonary edema, and dramatic increases in serum sodium and osmolarity.[6] The significance of these reported complications in trained hands is questionable, and hypertonic fluids may have a future role in resuscitation. Further study is warranted on the use of hypertonic saline, but its use in the wilderness setting is not recommended at this time.

Artificial blood products, such as perfluorocarbons and diaspirin cross-linked hemoglobin, have been shown to be efficient resuscitative fluids in animal studies.[20,80,89] However, they are expensive and not yet available for humans.

Based on the current literature, it is clear that both colloids (including hetastarches and albumin) and crystalloids are efficient volume expanders.[82] Larger volumes of crystalloids than colloids are needed to achieve similar resuscitative end points, usually in a ratio of 3 to 1. However, no benefit in survival using colloids has been demonstrated, and recent studies indicate that their use in critically ill patients may increase mortality.[19,72] In addition, no proven detriment, including increased extravascular lung water, impaired wound healing, or decreased tissue oxygen diffusion, has been demonstrated with the use of large volumes of crystalloids. Crystalloids are safe, nonantigenic, easily stored and transported, effective, and inexpensive. Most experts in trauma care agree that crystalloid is preferable to colloid infusion in the prehospital, early resuscitative phase of trauma care. Accordingly, the resuscitative fluid recommended by ATLS protocol is normal saline.

Several animal studies and recent human clinical trials in trauma victims have found that treatment with IV fluids before control of hemorrhage resulted in increased mortality rates.[9,59] Although these data are compelling, they have been accumulated in victims with penetrating injuries and short prehospital times, and the definition of prehospital resuscitation in these studies comprised widely varying volumes. Prehospital resuscitative protocols remain in evolution. However, application of these data to the wilderness setting at the current time is dangerous for a number of reasons. First, the leading cause of death in wilderness trauma is head injury. Many multiple trauma victims have a head injury, and it may be impossible to discern whether an intracranial lesion is present. Although under continued study, the current approach to management of head injury is aggressive maintenance of cerebral perfusion pressure to control intracranial pressure (ICP). Under-resuscitation in the context of an intracranial injury could be catastrophic. Second, the multisystem-injured victim frequently presents with associated orthopedic injuries. A victim with closed extremity fractures with significant contained hemorrhage benefits from fluid resuscitation. Third, a victim with significant external hemorrhage that can be controlled before evacuation benefits from intravascular repletion.

In summary, resuscitation with IV fluids should be initiated in the field, particularly in victims with head injury and unquantified multiple trauma. Victims should have vascular access secured and resuscitative fluids given in the form of normal saline as dictated by severity of injuries and hemodynamics.

Secondary Survey

The secondary survey is an extension of the primary survey and should not be undertaken until the primary survey is complete and the victim has been stabilized. In addition, resuscitative regimens, if available, should have been initiated. The secondary survey is a head-to-toe assessment of the victim, including history and physical examination. The face, neck, chest, abdomen, pelvis, extremities, and skin should be examined in sequence. A more detailed neurologic examination should be completed, including reassessment of the GCS. The neck should be examined independently of the thoracolumbar spinal cord. Examination of the pelvis should not include the traditional "rocking" to determine stability because in the presence of pelvic fractures, this action may exacerbate existing comminution.

The detailed secondary survey should not delay evacuation packaging. As in the nonwilderness setting, it is imperative to repeat the primary survey as the victim's condition warrants. Specific examinations are discussed in the sections covering regional injuries.

History

The victim's history should be assessed during the secondary survey. Knowledge of the mechanism of injury and any comorbid medical conditions or allergies may enhance understanding the victim's physiologic state.

The ATLS "AMPLE" history is a useful and rapid mnemonic for this purpose:

*A*llergies
*M*edications currently used
*P*ast medical history/*P*regnancy
*L*ast meal
*E*vent or *E*nvironment related to the injury

Adjuncts

Resuscitation should be initiated simultaneously with the primary survey. The degree of resuscitation depends on available resources, experience of the rescuer(s), and environmental conditions. Under the best circumstances, initial management of the wilderness trauma victim provides for airway control, adequate oxygenation and ventilation, appropriate fluid resuscitation, and stabilization of cardiac function while continuing to monitor and reassess the patient's vital signs. As adjuncts to the secondary survey, it also includes placement of a urinary catheter and nasogastric (NG) tube. This degree of resuscitation will be almost universally unavailable in the wilderness setting. Here, resuscitation may be limited to oral administration of warm, high-calorie fluids and maintenance of victim comfort and body temperature.

An NG tube and indwelling urinary (Foley) catheter should be placed if available and appropriate. Aspiration of gastric contents can be catastrophic in terms of patient survival after trauma. Gastric decompression with an NG tube may help prevent this type of adverse event in patients with a depressed level of consciousness. It should be remembered that children can exhibit significant hemodynamic consequences secondary to massive gastric distention. In this setting, decompression becomes critical. If possible, NG tubes should be placed in persons who are endotracheally intubated in the field. The tube can be aspirated sequentially with a syringe or left open to gravity drainage. Any suspicion of facial fracture should deter attempts to place an NG tube, and orogastric decompression should be chosen instead.

A Foley catheter can assist in volume assessment and hemodynamic status determination in a critically injured victim. Hourly urine output typically does not decrease until the onset of class III hemorrhagic shock, with loss of 30% to 40% of blood volume. Contraindications to urinary catheter placement in the field are blood at the urethral meatus, high-riding prostate, scrotal hematoma, and personnel not experienced in placement.

Pneumatic Antishock Garment. The pneumatic antishock garment (PASG) is a noninvasive device inflated around the lower extremities and abdomen to augment peripheral vascular resistance and increase blood pressure. It was widely instituted as a treatment for shock in the 1980s, largely based on anecdotal data. Prospective data relevant to penetrating chest and abdominal trauma[44,60] and retrospective data in blunt trauma[7] indicate an increase in mortality with its use. Hemodynamically unstable pelvic fractures can be stabilized using a simple sheet wrap. Use of the PASG is not indicated in the wilderness setting.[75] A new device for stabilization of a pelvic fracture in a wilderness setting is the SAM Sling (SAM Products, Newport, OR).

Injuries to the Head, Face, and Neck

The secondary survey begins with examination of the entire head and scalp for evidence of skull or facial fractures, ocular trauma, lacerations, and contusions. The scalp is thoroughly palpated for tenderness, depressions, and lacerations. The bones of the face, including the zygomatic arch, maxilla, and mandible, are palpated for fractures. Detailed discussion of orofacial and eye injuries is presented in Chapters 25 and 26). Elements of the GCS are repeated.

The wilderness eye is discussed in detail in Chapter 25, but general examination principles are simple. Significant periorbital edema may preclude examination of the globe, so assessment should be carried out early. The globe should be evaluated for visual acuity, pupillary size, conjunctival hemorrhage, lens dislocation, and entrapment. Persons with significant facial trauma have a high incidence of associated ocular or orbital injuries.[78] Recent studies of ocular injuries in trauma victims have emphasized underappreciation by many disciplines involved in the victim's care of ocular and periocular signs indicative of significant underlying injury.[74]

Head Injuries

Approximately 500,000 to 2 million cases of head injury occur in the United States yearly.[35] Of these, approximately 10% result in the patient's death before reaching a hospital.[5] Long-term disability associated with head injury is significant, with more than 100,000 persons suffering varying degrees of permanent impairment. Because of the high-risk nature of traumatic brain injury (TBI) and the impact of initial management on disability and survival, clinical management objectives must address both immediate survival and long-term outcome. Management guidelines for head injuries in a wilderness do not exist,

and a wide range of clinical approaches are used in hospital settings.[35] However, the literature suggests that morbidity and mortality can be reduced by means of a protocol that includes early airway control with optimization of ventilation,[40] prompt cardiopulmonary resuscitation, and rapid evacuation to a trauma care facility.

Initial management of head injury in the wilderness should follow established ATLS protocols. Prompt attention must then be given to victim triage, evacuation strategies, and ongoing resuscitative needs to prevent or minimize secondary brain injury. Expeditious evacuation to a neurosurgery-capable trauma center is essential.

Multiple clinical and experimental studies have demonstrated the detrimental effects of hypoxia on the injured brain. A definitive airway should be established if any degree of neurologic or respiratory compromise exists. Cervical spine injuries are common in patients with TBI. Therefore, cervical spine immobilization is paramount in prevention of further devastating neurologic injury.

After immobilization, attention is directed to prevention of secondary brain injury. The purpose of the wilderness head injury protocol is to allow individuals with widely varying levels of experience and expertise to identify signs of significant head injury, begin proper resuscitation in the context of prevention of secondary brain injury through airway maintenance and hemodynamic support, and evacuate appropriately.

Anatomy

The scalp comprises five layers of tissue that cover the calvaria: skin, connective tissue, galea aponeurotica, loose areolar tissue, and periosteum of the skull. The galea is a fibrous tissue layer with important ramifications in closure of scalp wounds, discussed later in this chapter. Loose areolar tissue beneath the galea represents the site of accumulation of blood in scalp hematomas. A rich vascular network located between the dermis and the galea supplies the scalp. When lacerated, these vessels can be a significant source of hemorrhage, which may be important if evacuation is impossible or delayed.

The skull is composed of two groups of bones that form the face and cranium. Cranial bones are divided into the calvaria and skull base. The calvaria is composed of frontal, ethmoid, sphenoid, parietal, and occipital bones. Within the skull, the brain is covered by three membranous layers that may be of pathophysiologic importance after injury. However, in the wilderness environment, these layers have little clinical relevance (except in terms of defining an open versus closed brain injury).

Pathophysiology of Traumatic Brain Injury

Traumatic brain injury can be divided into primary and secondary brain injury. Primary injury consists of the physical or mechanical insult at the moment of impact, and the immediate and permanent damage to brain tissue. Little can be done in the wilderness setting relative to primary brain injury. Secondary brain injury is the biochemical and cellular response to the initial mechanical trauma and includes physiologic derangements that may exacerbate effects of the primary trauma. Such pathophysiologic alterations include hypoxia, hypotension, and hypothermia. Compounding these pathophysiologic alterations is elevation of ICP after TBI. Increased ICP increases cerebral ischemia and exacerbates secondary brain injury.

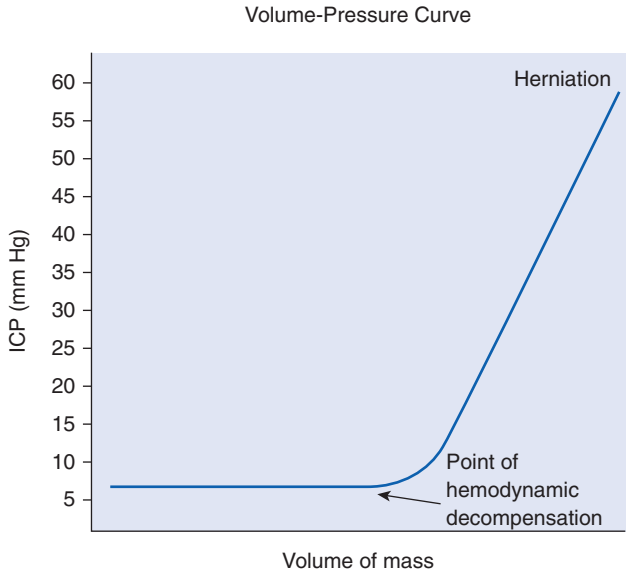


Figure 20-2. Critical time period between decompensation and brainstem herniation after traumatic brain injury.

Many forms of head injury result in elevated ICP, the duration of which is significantly correlated with poor outcome. The Monro-Kellie doctrine states that the volume of intracranial contents must remain constant because the cranium is a rigid container. The normal compensatory response to increased intracranial volume is to decrease venous blood and cerebrospinal fluid (CSF) volume within the brain. If this normal response is overwhelmed, small increases in intracranial volume result in exponential increases in ICP. A rigid bony cranium cannot expand to accommodate increases in brain volume and the resultant increase in ICP. Brain parenchyma becomes compressed and eventually displaced from its anatomic location. In the most devastating circumstances, the brain parenchyma herniates toward the brainstem through the largest cranial opening (the foramen magnum) and death rapidly follows. The volume–pressure curve in Figure 20-2 relates the small, but critical, time period between neurologic symptoms, hemodynamic decompensation, and brainstem herniation. Elevation in ICP directly correlates with secondary brain injury. Therefore, the field provider must attempt to minimize ICP of head-injured patients to the greatest extent possible.

The most important priority in minimizing secondary brain injury in the field is optimizing cerebral perfusion pressure (CPP). CPP is related to ICP and mean arterial pressure (MAP) as follows:

$$CPP = MAP - ICP$$

A CPP less than 70 mm Hg after head injury correlates with increased morbidity and mortality.[5,39] Cerebral blood flow (CBF) should be maintained at approximately 50 mL/100 g brain tissue per minute.[57] At 5 mL/100 g per minute, irreversible damage and potential cell death occur.[5] Study data have shown a correlation between low CBF and poor outcome.[57] At a MAP between 50 and 160 mm Hg, cerebral autoregulation maintains

CBF at relatively constant levels. Not only is autoregulation disturbed in injured regions of the brain, but a precipitous fall in MAP can further impair autoregulatory function, decreasing CBF and exacerbating ischemia-induced secondary injury. The field provider is able to combat a rise in ICP by simply optimizing MAP through aggressive IV fluid resuscitation.

Diagnosis

The three useful descriptions of head injury that may be applied to field recognition are history, severity, and morphology. History includes mechanism of injury, timing of the event, and related circumstances. This knowledge assists in the decision-making process with regard to resuscitation and evacuation.[5] Mechanism of injury is identified as blunt or penetrating trauma. The anatomic demarcation between blunt and penetrating injury is traditionally defined by violation of the outer covering of the brain (dura mater). Blunt injuries in the wilderness setting most often result from falls, falling objects, or assaults. Penetrating injuries are most commonly gunshot, or other projectile, wounds. Severity of injury can be estimated by quantifying the GCS and pupillary response. The generally accepted definition of coma is a GCS score of less than or equal to 8; these patients often require endotracheal intubation. Although GCS score does not directly correlate with a need for intubation, it is essential that all head-injured patients be provided a stable, secure airway by the most appropriate means available. It is important to note the TBI victim's best initial motor response because this is most predictive of long-term neurologic outcome. Any victim with a GCS score less than 15 who has sustained a head injury should be evacuated, if possible. A low or declining GCS score suggests increasing ICP. Abnormal pupil size or asymmetric pupillary responses suggest increased ICP. These clinical deteriorations demand the rapid attention of rescue or evacuation personnel to optimize MAP and CCP, minimize secondary brain injury, and prevent brainstem herniation. Injury morphology may be difficult to assess in the wilderness setting and relies on level of suspicion and clinical signs and symptoms. After attention to the primary survey, including airway provision and spinal immobilization, the physical examination of the secondary survey is imperative and can provide information about the presence of a TBI.

Injury Classification

Intracranial injuries range from concussion to massive subdural hematoma. Subdural hematomas are more common than epidural hematomas, comprising 20% to 30% of mass lesions. "Subdurals" result from torn bridging veins between the cerebral cortex and draining venous sinuses. Their prognosis is worse than that of "epidurals," although prompt recognition and drainage improves patient outcome. Epidermal hematomas are most commonly located in the temporal region and result from injury to the middle meningeal artery, often associated with a fracture. These patients may present with loss of consciousness followed by a lucid interval and subsequent rapid neurologic deterioration. This sequence, however, is not frequently observed. Hemorrhagic contusion is also quite frequent, constituting 35% of traumatic injuries, and has the propensity to increase ICP significantly. Diffuse axonal injury (DAI) is the term used to describe prolonged post-traumatic coma not resulting from a mass lesion or ischemic insult. Similar to hemorrhagic contusion, DAI may result in elevated ICP.

Physical Examination

After the primary survey and initial attempts to resuscitate the victim, a more complete physical examination should be done. However, this examination should not delay patient evacuation. A hallmark of TBI is altered level of consciousness. Determination of the GCS score aids in recognition of TBI and should be regularly reassessed to provide a mechanism for quantifying neurologic deterioration. Physical signs that may denote underlying brain injury include significant scalp lacerations or hematomas, contusions, facial trauma, and signs of skull fracture. Findings specific for basilar skull fracture include ecchymosis behind the ears (Battle's sign) and periorbital ecchymosis (raccoon eyes). Blood behind the tympanic membrane on otoscopic examination (hemotympanum), frank bleeding from the ears, and CSF rhinorrhea/otorrhea also suggest skull fracture and underlying TBI.

The pupillary examination may provide valuable data in assessment of underlying TBI. Herniation of the temporal lobe of the brain may be heralded by mild dilation of the ipsilateral pupil with sluggish response to light. Further dilation of the pupil followed by ptosis (drooping of the upper eyelid below its normal level), or paresis of the medial rectus or other ocular muscle, may indicate third cranial nerve compression by a mass lesion or herniation. Table 20-2 relates pupillary examinations to possible underlying brain lesions. Most dilated pupils (mydriasis) are on the ipsilateral side to the mass lesion. With direct globe injury, traumatic mydriasis may result, making evaluation of TBI more difficult. In addition, 5% to 10% of the population has congenital anisocoria (a normal difference in pupillary size between the eyes). Casual inspection may overlook a prosthetic eye, which is mistaken for a fixed pupil. Neither direct trauma nor congenital anisocoria should be assumed in a head-injured victim exhibiting mental status change in the wilderness.

After quantification of GCS score, pupillary examination, and examination of the head and face for signs of external trauma, a concise neurologic examination should be performed. The goal of the field neurologic examination is to identify motor or sensory focal deficits suggestive of intracranial injury. Sensory

TABLE 20-2. Interpretation of Pupillary Findings in Head-Injured Victims

PUPIL SIZE	LIGHT RESPONSE	INTERPRETATION
Unilaterally dilated	Sluggish or fixed	Third nerve compression secondary to tentorial herniation
Bilaterally dilated	Sluggish or fixed	Inadequate brain perfusion; bilateral third nerve palsy
Unilaterally dilated or equal	Cross-reactive (Marcus-Gunn)	Optic nerve injury
Bilaterally constricted	Difficult to determine; pontine lesion	Opiates
Bilaterally constricted	Preserved	Injured sympathetic pathway

deficits follow general dermatome patterns shown in Figure 20-3. Unilateral hemiplegia may signify uncal herniation resulting from mass effect in the contralateral cortex because of compression of the corticospinal tract in the midbrain. Ipsilateral pupillary dilation associated with contralateral hemiplegia is a classic and ominous sign of tentorial herniation. Reflex changes in the absence of altered mental status are not indicative of TBI. Detailed evaluation of brainstem function cannot be undertaken in the wilderness setting. Performance of gag and corneal reflex evaluations may provide some information helpful in triage and evacuation planning, but their presence would not automatically obviate the need for prompt evacuation.

Resuscitation

Resources and circumstances permitting, resuscitation should be initiated as an adjunct to the primary survey. The primary focus for the head-injured victim, similar to any traumatized victim, is the airway. During the primary survey and performance of the ABCDE sequence, IV access should be established. If IV resuscitation is impossible, it is not advisable to adminis-

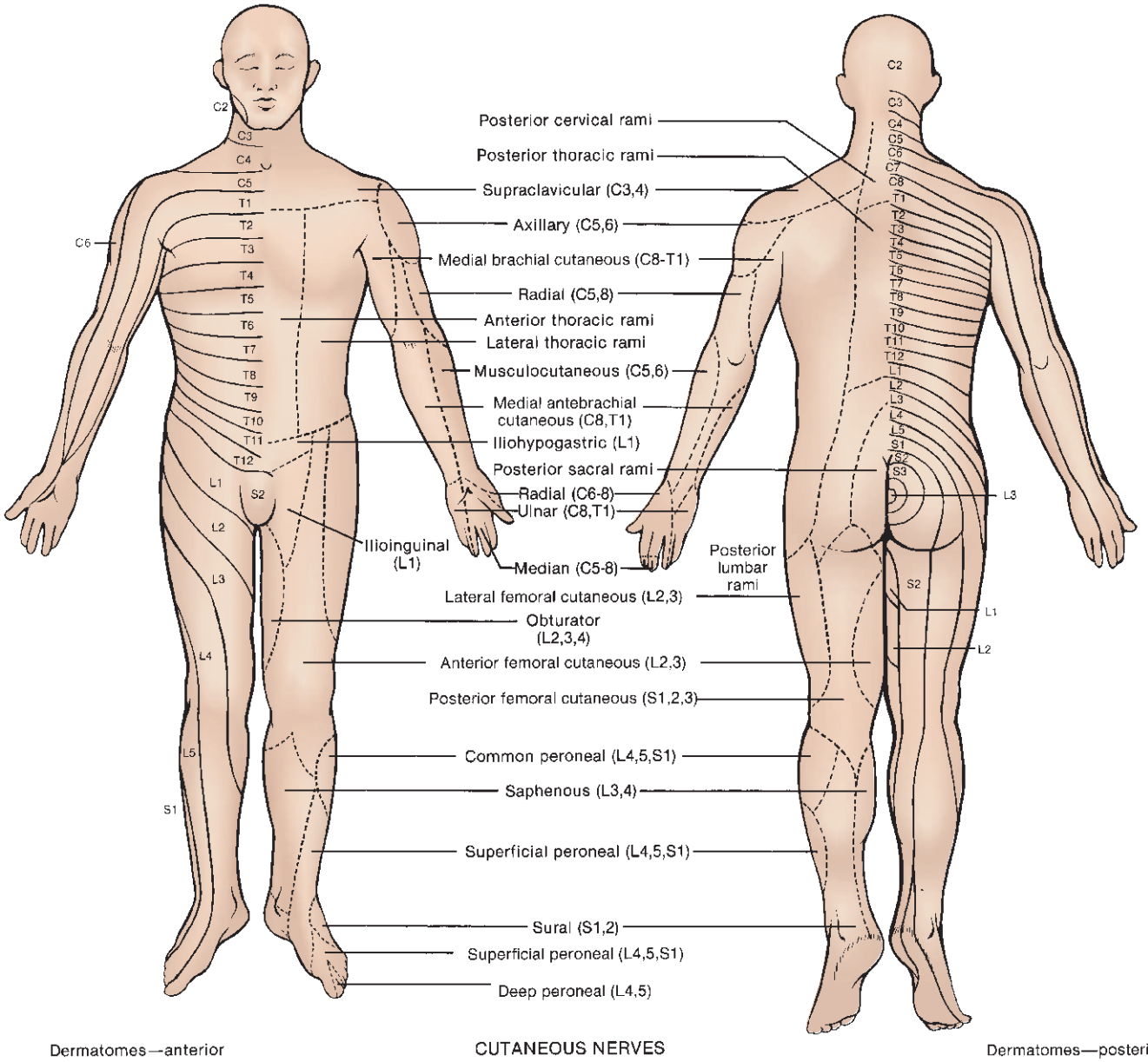


Figure 20-3. Dermatome pattern, showing the skin area stimulated by spinal cord elements. Sensory deficits follow general dermatome patterns.

ter fluids orally to the victim with head injury because of the likelihood of vomiting, airway compromise, and aspiration.

Individuals sustaining head trauma have a high incidence of concomitant injuries. Up to 32% of persons with severe head injury have a long bone or pelvic fracture, 20% to 25% a chest injury, and 10% an abdominal injury. A victim who does not have a palpable femoral pulse or manifests other signs of hypotension in the context of suspected head injury must not be assumed to have a neurogenic etiology of shock, so other etiologies must be thoroughly and aggressively investigated. Resuscitation is critical in the setting of head injury for multiple reasons. Management of a head injury should be secondary to other life-threatening injuries, which, if not addressed, may preclude survival. As previously discussed, maintenance of MAP (and thus CPP) is critical in preventing secondary brain injury.

The type of resuscitative fluid administered to trauma victims continues to be controversial. Previously, recommendations warned of the dangers of overhydration in head injury leading to recommendations restricting fluids. Restriction of fluid has not been shown to reduce ICP or edema formation in laboratory models of TBI. Theories of limiting cortical free water content in TBI by using hypotonic IV solutions have not been borne out in animal studies.[90] The need for resuscitation and intravascular volume support has been well established. Possible resuscitative fluids include isotonic crystalloids, hypertonic crystalloids, or colloid solutions. There is convincing evidence that hypotonic fluids are not appropriate in TBI secondary to an increase in whole-brain water content and subsequent elevation in ICP. Recent data from animal studies of TBI suggest that colloid solutions offer no advantage over isotonic crystalloids, such as lactated Ringer's solution, in terms of augmenting CBF or preventing cerebral edema.[101] As previously noted, no clear prospective trial has documented any advantage of colloid over crystalloid administration in the victim with multiple systemic injuries. Evidence is accumulating that hypertonic solutions, particularly hypertonic saline, may be beneficial in TBI.[92,97] However, an advantage has not been demonstrated in trauma victims overall, and expertise is necessary for their use. The recommended resuscitative fluid for the head-injured victim in the wilderness setting is isotonic crystalloid, with a target MAP of 85 to 95 mm Hg based on cuff blood pressure determinations or extrapolation from distal pulses evaluation.

Further Management

Numerous adjuncts exist in the management of the head-injured victim, few of which are applicable in the wilderness setting. Once the primary and secondary surveys are complete, the airway is secured, resuscitation has been initiated, and spine immobilization has been achieved, the victim should be placed in a 30-degree head-up position. This position assists in control of ICP, and thus CPP, through augmentation of venous outflow. This maneuver should not be attempted if the spine cannot be adequately immobilized.

If endotracheal intubation is possible, ventilation should be optimized without hyperventilating the victim. Hyperventilation has been used aggressively in the past to promote hypocarbia-induced cerebral vasoconstriction, theoretically to decrease brain swelling. However, if the $Paco_2$ falls below 25 mm Hg, severe vasoconstriction ensues, effectively reducing CBF, promoting ischemia, and possibly augmenting secondary brain injury. Studies have demonstrated worse outcomes in victims with severe head injury who were hyperventilated.[68] The inability to measure or titrate $Paco_2$ in the wilderness mandates that respiration be controlled to approximate near-normal minute ventilation.

All bleeding from the scalp or face should be controlled with direct pressure. Scalp hematomas, regardless of size, should not be decompressed. Open wounds, particularly skull fractures, should be irrigated and covered with the most sterile dressing available. Fragments of displaced cranium overlying exposed brain tissue should not be replaced. If signs of skull fracture are present, immunization against tetanus and broad-spectrum antibiotic prophylaxis are recommended as soon as possible.

Although diuretics have been widely used in the intensive care management of intracranial hypertension, no rationale exists for their use in the field. The wilderness trauma victim may have many injuries that are impossible to evaluate fully in the field. In this setting, particularly in the presence of hemorrhagic shock, attempts to induce osmotic diuresis to decrease ICP may be life-threatening. Diuretics such as furosemide or mannitol may exacerbate hypotension, cause metabolic alkalosis, and induce renal complications in the absence of physiologic monitoring.[4] Steroids have no role in head injury in the field or intensive care unit. Studies have documented no beneficial impact on ICP or survival. Attempts at brain preservation by slowing metabolic rate and oxygen consumption have no role in the wilderness setting. Barbiturates have been used for elevated ICP refractory to other measures, but may induce hypotension, depress myocardial function, and confound the neurologic examination.[4] Compared with minimizing ICP, these interventions offer no significant benefit.[39]

Approximately 15% of persons with severe head injury experience post-traumatic seizures. Phenytoin, if available, can be safely administered in the field, but only after a witnessed seizure. Prophylactic administration has not been shown to decrease long-term seizure activity.

Skull Fracture

Skull fracture in the wilderness mandates evacuation. Therapeutic options in the field are few, with intervention limited to identifying the injury and arranging rapid transport. Skull fractures may be open or closed, linear or stellate, and may occur in the vault or skull base. These fractures are associated with a high incidence of underlying intracranial injury. In an awake and alert victim with a skull fracture, the chance of brain injury is increased 400-fold.[5] Skull fractures with depression greater than the thickness of the skull may require elevation. No attempt at elevation should be made in the field. Any exposed brain surface should quickly be covered with the most sterile covering available, preferably moistened with crystalloid solution. Loose bone or brain fragments should not be manipulated. If a broad-spectrum antibiotic is available, it should be administered. After attention to the wound and stabilization of associated injuries, the victim should be rapidly evacuated.

Penetrating Head Injuries

The majority of penetrating head injuries in the wilderness are gunshot wounds, although knives and arrows may penetrate the cranium. Such penetrating injuries are usually catastrophic. However, examples of survival exist with small-caliber, low-velocity injuries and tangential wounds.[58] As with closed head injury, management priorities consist of maintenance of airway, prevention of secondary brain injury, and rapid evacuation. If the cranium has been violated, the victim should receive antibi-

otics and tetanus immunization in the same manner as for open skull fracture. In the rare instance that the projectile is embedded in the skull, no attempt at removal should be undertaken. If the length of the projectile makes immobilization or transport cumbersome, excess length may be removed, but only if this can be done easily and without displacement of the intracranial segment.

Evacuation

Survival and outcome of head injury in the wilderness correlate directly with rapidity of evacuation. Certain situations dictate immediate evacuation. Any person with evidence of an open or closed skull fracture should be evacuated. The incidence of TBI associated with skull fracture is variable but significant throughout the literature. Recent data predict that 30% to 90% of persons with raccoon eyes or Battle's sign will show abnormalities on computed tomography (CT) scan.[11,18] Similarly, any person who sustains a penetrating injury should be evacuated. Decisions concerning evacuation of victims who have sustained closed head injuries can be simplified by dividing the victims into three groups based on probability of injury. A high-risk group, defined as patients with GCS score of 13 or less, focal neurologic signs, or evidence of decreasing level of consciousness, requires evacuation. The low-risk group includes persons who have suffered a blow to the head but are asymptomatic, did not lose consciousness, and complain only of mild headache or dizziness. Data from recent studies suggest that persons who meet low-risk criteria (including GCS of 15, no loss of consciousness, minimal symptomatology, and unlikely mechanism) have a minimal chance of having significant TBI and may be closely observed.[11,18]

The group for which the evacuation decision is most difficult is the moderate-risk group. These persons have a history of brief loss of consciousness or change in consciousness at the time of injury, or a history of progressive headache, vomiting, or posttraumatic amnesia. If any of these signs is present in the face of concurrent systemic injury, the victim should be evacuated immediately. Studies associating clinical variables and abnormal results on CT scan have demonstrated the significance of decreased GCS score, symptoms, and loss of consciousness. If these signs are present in isolation and the evacuation can be completed in less than 12 hours, the evacuation should proceed. If the evacuation is impossible or will require longer than 12 hours, the victim should be closely observed for 4 to 6 hours. If the examination improves to normality during the observation period, it is reasonable to continue observation.

Neck Injuries

Blunt Neck Injuries

Injuries to the neck may be classified as blunt or penetrating. Significant blunt injuries include cervical spine injuries and laryngotracheal injuries. Seventy-five percent of injuries to the trachea are confined to the cervical region.[73] Fracture of the larynx and disruption of the trachea usually require surgical intervention unavailable in the wilderness. The sooner laryngeal repair is accomplished, the better the outcome with respect to phonation.[21] Victims present with a history of a significant blow to the anterior neck. Physical examination findings include difficulty with phonation, subcutaneous emphysema that may extend as far inferiorly as the abdominal wall, stridor, odynophagia, and, often, acute respiratory distress.

Treatment is focused on establishing and maintaining an airway until evacuation can occur. Frequently, the airway is in jeopardy. Because of the propensity for injuries of this type to result in significant and progressive edema, endotracheal or nasotracheal intubation is often necessary. If these options are unavailable, airway maintenance techniques as described in the Primary Survey section should be used. In the event of intubation failure or lack of availability with impending hypoxic death, a surgical cricothyrotomy may be necessitated. A recent study of prehospital cricothyrotomy demonstrated that success rates were high regardless of medical specialty as long as previous training had been instituted.[54] For further descriptions of airway management, refer to Chapter 19.

Background. Vertebral column injury, with or without neurologic deficits, must be identified in any wilderness multiple trauma victim. Approximately 2.6% of victims of major trauma suffer acute injury of the spinal cord.[16] Fifteen percent of victims sustaining an injury above the clavicles and 5% to 10% of persons with a significant head injury have a cervical spine injury. In addition, 55% of spinal injuries occur in the cervical region.[58] In the wilderness setting, fractures or dislocations of the cervical spine are a result of falls from significant heights, or of high-velocity ski or vehicular injuries. Twenty-eight percent of persons with cervical spine fractures have fractures elsewhere in the spine.[10]

Anatomy. The cervical spine consists of seven vertebrae. The anteriorly placed vertebral bodies form the weight-bearing structure of the column. The bodies are separated by intervertebral discs and held in place anteriorly and posteriorly by longitudinal ligaments. The paraspinal muscles, facet joints, and interspinous ligaments contribute as a whole to the stability of the spine. The cervical spine, based on its anatomy, is more susceptible to injury than are the thoracic and lumbar spine. The cervical canal is wide from the foramen magnum to C2, with only 33% of the canal comprised by the spinal cord itself. The clinically relevant tracts in the spinal cord include the corticospinal tract, spinothalamic tract, and posterior columns.

Classification and Recognition. Fractures of the cervical spine may result in neurologic deficit, with total loss of function below the level of injury.[52] Resultant spinal cord injuries should be classified according to level, severity of neurologic deficit, and spinal cord syndrome. Fractures of the C1-C2 complex generally result from axial loading (a C1 ring fracture, or Jefferson's fracture) or an acute flexion injury (a C2 posterior element fracture, or hangman's fracture). Approximately 40% of atlas fractures have an associated fracture of the axis. The atlas fracture, if survived, is rarely associated with cord injury but is unstable and requires strict immobilization. Usually, a complete neurologic injury at this level is unsurvivable owing to paralysis of respiratory muscle function. One third of victims sustaining an upper cervical spine injury die at the scene. The most common mechanism of injury is flexion, and the most common level of injury is C5-C6.[10]

Fractures and dislocations may result in partial or complete neurologic injury distal to the fracture or in no neurologic injury at all. Partial injuries to the spinal cord result from typical patterns of injury. Because flexion injuries are the most common type of injury to the cervical spine, the anterior cord syndrome (see later) is the most commonly seen serious neurologic picture.

A careful neurologic examination in the field to grade motor strength and document sensory response to light touch and pinprick yields important information that should be documented and reported to the treating physician at the definitive care facility. The presence or absence of Babinski's reflex should be noted, as well.

When appropriate resources are available, a rectal examination should be performed. Complete lack of tone and failure of the sphincter muscles to contract when pulling on the penis or clitoris (the bulbocavernosus reflex) indicate the presence of spinal cord injury.

When individuals with cervical spine fractures or dislocations are transported, the neck must be stabilized to prevent further injury to the spinal cord or nerve roots at the level of the fracture or dislocation. Approximately 28% of persons with cervical spine fractures have fractures elsewhere in the spine[10]; therefore, the entire spine must be protected during transport.

Occasionally, a pure flexion event can result in dislocation of one or both of the posterior facets without fracture or neurologic injury. The victim may complain only of neck pain and limitation of motion. If so, the victim should be transported with the neck rigidly immobilized. With this injury, posterior instability is present (because the interspinous ligament is ruptured), and any further flexion stress could produce a spinal cord injury.

Physical Examination. A thorough neurologic examination should be performed. Initial documentation of deficits and frequent repeat examinations are critical to follow-up care. The classification of injury in the field begins with determination of the level of injury. Knowledge of sensory dermatomes (see Fig. 20-3) and motor myotomes is invaluable. The sensory level is the lowest dermatome with normal sensation and may differ on each side of the body. C1 to C4 are variable in their cutaneous distribution, so assessment should begin at C5. The examiner should not be confused by the occasional innervation of the pectoral skin by C1 to C4, known as the "cervical cape." Light touch and pinprick should be assessed.

Motor function should be assessed by the myotomal distribution listed in Box 20-2. Each muscle should be graded on a six-point scale:

0—Total paralysis
1—Palpable or visible contraction
2—Full range of motion without gravity
3—Full range of motion against gravity
4—Full range of motion with decreased strength
5—Normal strength

Each muscle must be tested bilaterally and documented. The reflexes alluded to in the classification section must be tested, as well as anal sphincter tone.

Box 20-2. Sensory and Motor Deficit Assessment

SENSORY
C5: Area over deltoid
C6: Thumb
C7: Middle finger
C8: Little finger
T4: Nipple
T8: Xiphisternum
T10: Umbilicus
T12: Symphysis
L3: Medial aspect of thigh
L4: Medial aspect of leg
L5: First toe web space
S1: Lateral foot
S4 and S5: Perianal skin

MOTOR
C5: Deltoid
C6: Wrist extensors
C7: Elbow extensors
C8: Finger flexors, middle finger
T1: Small finger abductors
L2: Hip flexors
L3: Knee extensors
L4: Ankle dorsiflexors
L5: Great toe extensors
S1: Plantar flexors

Syndromes.

There are three clinically useful spinal cord syndromes:

Central cord syndrome is characterized by a disproportionate loss of motor power between the upper and lower extremities, with greater strength retained in the lower extremities. Sensory loss is variable. The mechanism of injury usually involves a forward fall with facial impact and hyperextension of the spine.

Anterior cord syndrome is characterized by paraplegia and loss of pain and temperature sensation. It is the most common presenting syndrome caused by cervical spine injury and carries a poor prognosis.

Brown-Séquard syndrome results from hemisection of the cord. It consists of ipsilateral motor loss and position sense with contralateral sensory loss two levels below the level of injury. It is usually secondary to penetrating injury.

Immobilization. After identification of injury, the caregiver faces a critical decision with important ramifications—whether to immobilize.[32] Victims who would as a matter of course be immobilized in an urban setting might not be appropriate candidates for immobilization in the wilderness. The decision to immobilize converts an otherwise ambulatory victim who can actively participate in his or her own evacuation to one requiring more involved evacuation procedures. The subsequent evacuation can be dangerous to the victim and rescuers and demands significant expense and resource utilization.

Risk criteria for cervical spine injury and the need for immobilization have been defined.[42,56] All criteria for the exclusion of immobilization must be satisfied. These include normal mental status without chemical influence; lack of distracting injury; normal neurologic examination; and a reliable neck examination without midline neck pain, deformity, or tenderness. Figure 20-4 presents an evidence-based algorithm for determining need for immobilization. Although the need for immobilization poses hazards for the evacuation process, if criteria are met, immobilization takes precedence over ease of evacuation.[62] A difficult balance must be struck in the wilderness between the likelihood of true injury and the danger to the expedition members and rescuers that may ensue when the victim is immobilized.

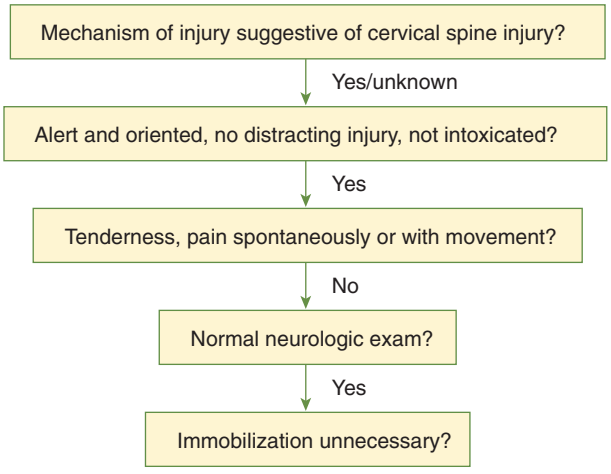


Figure 20-4. Clinical assessment of cervical spine stability. Failure of any criterion suggests need for immobilization.

If a rigid litter is not available, the victim should be maintained on the flattest surface possible. A rigid cervical collar should be placed. All collars allow some degree of movement, particularly rotation. Soft collars offer the least immobilization.[30] The Philadelphia collar has been shown to allow 44% of normal rotation and 66% of normal lateral bending.[58] To achieve 95% immobilization, a halo and vest are necessary. Any number of materials may be used to improvise an immobilizing device (see Chapter 21). Restriction of flexion, extension, and rotation must be achieved to the greatest degree possible. Optimal immobilization consists of a long spine board or litter, rigid collar, bolsters to the sides of the head, and tape or straps restricting movement (Fig. 20-5).

Treatment. The issue of pharmacotherapy for spinal cord injury is under continuous study. Currently, based on data accumulated by the National Acute Spinal Cord Injury Study Group, documented blunt spinal cord injury should be treated with a bolus of 30 mg/kg of methylprednisolone within 8 hours of injury followed by a continuous infusion of 5.4 mg/kg/hr over the next 23 hours.[14] Steroids are not recommended in the field unless a victim clearly manifests a spinal cord injury in the absence of head injury.

Because little definitive treatment for cervical spine injury can be accomplished in the field, survival and outcome depend on speed of transport and maintenance of airway. This is particularly true considering the association of cervical spine injury with head injury and major systemic trauma. Transport all victims with proven or suspected cervical spine injury to a definitive care facility.

Penetrating Neck Injuries

Similar to penetrating head injury, penetrating neck injury is usually due to gun or knife wounds. Most penetrating injuries do not confer bony instability; however, stability should not be assumed. Neurologic deficits, if present, can progress with further movement of an unstable spine. Projectiles should not be removed if embedded in the neck. Penetrating injuries to the neck may not directly injure the spine, but neurologic sequelae may result from blast effect. The same immobilization criteria should be implemented as when dealing with blunt injuries.

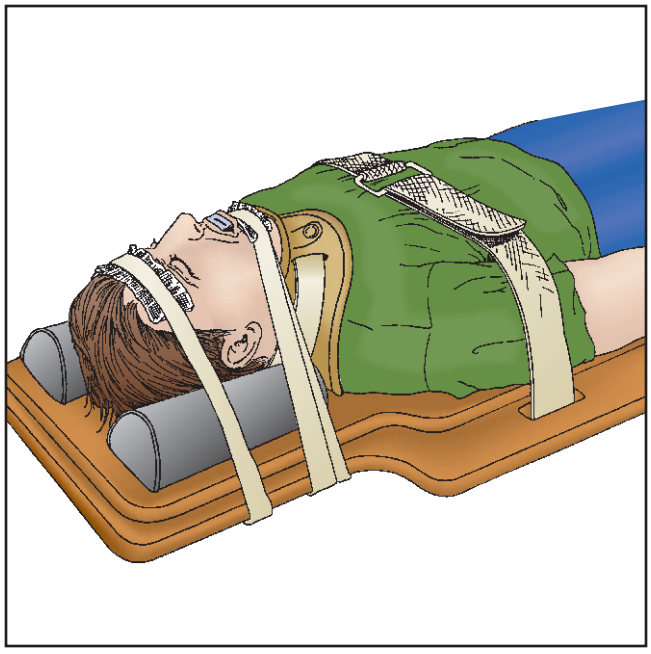


Figure 20-5. Proper spine immobilization.

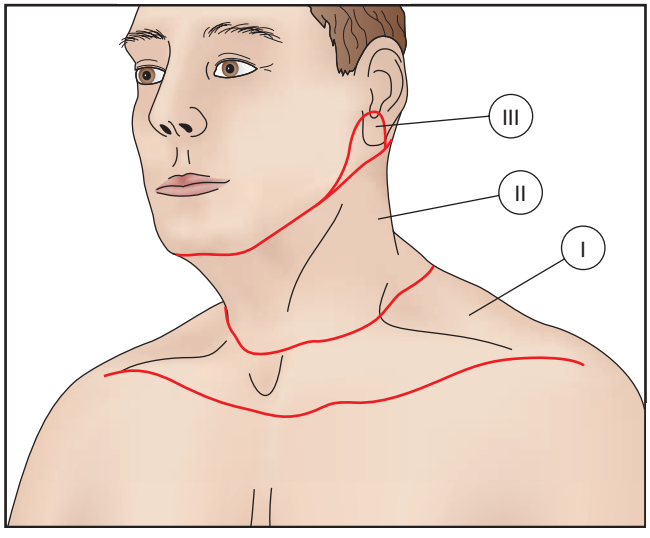


Figure 20-6. Zones in penetrating neck trauma (see text).

Penetrating injuries to the neck are classified according to anatomic zones of injury (Fig. 20-6). Zone I injuries extend from the clavicles to the cricoid cartilage. Zone II injuries occur between the cricoid and the angle of the mandible. Zone III injuries occur superior to the angle of the mandible.

Historically, treatment has been based on penetration of the platysma muscle. In the wilderness setting, if the examiner is

confident that platysmal penetration has not occurred, the victim may be observed and the wound considered a laceration. Much debate has occurred over management of platysmal penetration within respective topographic zones, with treatment arms consisting of surgical exploration versus radiographic evaluation. In the wilderness setting, such considerations remain relevant. A penetrating injury violating the platysma muscle indicates the possibility of significant neurovascular, esophageal, or tracheal injuries, so the victim should be evacuated with close attention to the airway.

Injuries to the Thorax

Background

The mortality rate from thoracic trauma is approximately 10%. Approximately 25% of all trauma deaths in the United States are attributable to chest injury.[5] However, only 15% of persons with penetrating thoracic trauma require thoracotomy.

In the wilderness environment, blunt thoracic injuries usually result from falls or direct blows to the chest. Penetrating injuries result from gun, knife, or arrow wounds, or from impalement after a fall. Immediate, life-threatening thoracic injuries include airway obstruction, tension pneumothorax, flail chest, and cardiac tamponade.

Pathophysiology

Chest injuries often result in hypoxia, hypercarbia, and acidosis. The tissue hypoxia of thoracic trauma can be multifactorial. Inadequate delivery of oxygen can be from hemorrhagic shock, direct lung injury with ventilation–perfusion mismatch (pulmonary contusion, atelectasis, hematoma), or changes in normal intrathoracic pressure dynamics (tension or open pneumothorax). Hemodynamic instability and inadequate oxygen delivery may also result from cardiac tamponade or contusion.

Physical Examination

Thorough physical examination begins with visualization and inspection of the chest. Exposure of the chest should be completed in the primary survey. The airway is assessed for patency and air exchange, and the pattern of breathing is noted. In the immediate postinjury period, most trauma victims are tachypneic, partly from pain and anxiety. Dyspnea, cyanosis, the use of accessory muscles of respiration, and intercostal muscular retraction are abnormal and may give clues to the underlying injury.

Chest wall movement during respiration should be symmetrical. Paradoxical chest wall movement is associated with flail chest. The chest wall should be inspected for contusions and abrasions, which may herald underlying bony or visceral injury.

Distention of the external veins in a person who has just suffered thoracic trauma and is hypotensive or tachycardic (heart rate greater than 130 beats per minute) suggests impaired venous return to the heart. This finding may be seen in situations of increased intrathoracic or intrapericardial pressure and is associated with tension pneumothorax and pericardial tamponade. In tension pneumothorax, deviation of the trachea is in a direction opposite the lesion. Significant sternal bruising may herald fracture or cardiac contusion.

The thorax should be palpated systematically for bony tenderness, starting at the distal clavicles and working medially toward the sternum. The sternum is divided into the manubrium, gladiolus (body), and xiphoid cartilage. The manubrium is joined to the gladiolus by fibrocartilage, but mobility at this joint is minimal.

Each rib should be palpated individually. Ribs 1 to 7 are vertebrosternal; their costal cartilages join the sternum. Ribs 8 through 10 are vertebrochondral, with each costal cartilage commonly joining the cartilage of the rib above. Ribs 11 and 12 are vertebral ribs without attachment to the sternum. Point tenderness over a rib can be associated with contusion or fracture. Displaced fractures can be palpated; occasionally, bone grating can be palpated during respiration.

Subcutaneous emphysema may extend up into the neck and down to the level of the inguinal ligaments. In the trauma situation, subcutaneous emphysema is invariably associated with pneumothorax.

Vocal fremitus describes palpation of vibrations transmitted through the chest wall. During speech, the victim's vocal cords emit vibrations in the bronchial air column that are conducted to the chest wall. Diminished vocal fremitus is associated with pneumothorax or hemothorax. To test for vocal fremitus, the examiner applies the palmar arch of the examining hand against the person's anterior chest wall. The person is asked to repeat "one, two, three" using the same pitch and intensity of voice with each repetition. If the vibrations are not well perceived, the patient is asked to lower the pitch of the voice. The chest should be symmetrical, left to right.

Percussion is used to detect changes in the normal density of an organ. Percussion of the chest is performed by placing the examining fingertips on the chest wall and sequentially striking the fingertips with the tip of the index or middle finger of the other hand. In the trauma victim, dullness replacing resonance in the lower lung suggests hemothorax. Hyperresonance or tympani replacing resonance occurs only with a large pneumothorax or tension pneumothorax.

If a stethoscope is not available, primitive chest auscultation can be performed using a rolled piece of cardboard or paper. Any cylinder that can transmit sound through a column of air accentuates breath sounds when placed against the chest wall. The absence of sounds normally produced by the tracheobronchial air column indicates blockage in the airways or abnormal filtering of sound by fluid in the pleural cavity. In the trauma victim, this is invariably associated with pneumothorax or hemothorax.

Blunt Chest Trauma

Blunt chest trauma in the wilderness is most often associated with either a direct blow or a deceleration injury. The mechanism usually relates to a fall from a height. Compression of the chest wall by moving or falling debris may also contribute to intrathoracic injuries, as may be seen in traumatic asphyxia associated with burial in an avalanche or earthquake.

Rib Fractures. Rib fractures range in severity from an isolated nondisplaced single fracture, which causes only minor discomfort, to a major flail segment, which can be associated with an underlying hemopneumothorax and pulmonary contusion. Rib fractures are characterized by painful respiration, most severe on inspiration. Victims often breathe in a characteristically rapid, shallow pattern. Point tenderness is palpated over the fracture, and displacement can occasionally be detected. Rib fractures are detected with a compression test, in which pressure is exerted on the sternum while the victim lies supine. This will elicit pain over the fracture site.

Most patients with rib fractures can be managed with oral analgesics and rest. Thoracic taping and splinting are contraindicated. Multiple rib fractures are significant because of the potential seriousness of associated injuries and increased pain. However, if extreme and compromising respirations, this pain responds well to an intercostal nerve block. Victims with multiple rib fractures need to be evacuated as conditions permit. After administration of an intercostal block, a person may regain the ability to hike out of the wilderness.

The morbidity of rib fractures relates to decreased inspiratory tidal volume secondary to pain and splinting. In the wilderness setting, management must focus on pain control and pulmonary toilet. If oral analgesia is insufficient to control pain, an intercostal block is ideal. Depending on the anesthetic used, varying durations of analgesia can be attained, perhaps allowing transient ambulation for evacuation. Deep breathing should be encouraged 10 times hourly to help prevent atelectasis.

Costochondral Separation. It is difficult to distinguish between a rib fracture and costochondral separation. With the latter, pain is more likely to be predominantly anterior over the costochondral junction. Pain increases with inspiration and worsens with direct palpation. Costochondral separation also responds to intercostal nerve block and oral and IV analgesics.

Sternal Fracture. A sternal fracture is usually associated with a direct blow to the anterior chest wall. The injury is characterized by severe, constant chest pain that worsens with direct palpation. Sternal instability is unusual and can be associated with a significant underlying visceral injury, including pulmonary or myocardial contusion. If the sternum is unstable, the victim should immediately be evacuated by litter or helicopter.

Pneumothorax. Simple pneumothorax can occur from an injury that allows air to enter through the thoracic wall or, more frequently, from an injury to the lung that permits air to escape into the pleural space. Symptoms include tachypnea, dyspnea, resonant hemithorax, absence of breath sounds, and tactile fremitus. A person with chest pain after a blunt blow to the chest, particularly with accompanying rib fracture, should be suspected of having a pneumothorax.

Treatment of pneumothorax involves decompression of the pleural space. In the wilderness environment, tube thoracostomy is rarely possible. Fortunately, although victims with isolated pneumothorax may complain of chest pain or dyspnea, they are not completely disabled. With analgesia to control pain, ambulation facilitates evacuation. It may be easier and more prudent to set a slow pace with frequent rest periods than to perform an unnecessary litter evacuation.

If resources and expertise allow placement of a thoracostomy tube, it should be performed only when clinically indicated. Considering possible morbidity in a remote area, prophylactic decompression should never be undertaken. Suspicion of a pneumothorax alone on physical examination does not warrant a catheter or chest tube. When a high index of clinical suspicion is accompanied by incapacitating symptoms, such as shortness of breath, decompression should be considered. The key to saving a victim's life is understanding that a condition exists that can rapidly progress from a nondisabling condition to a life-threatening condition. Once the diagnosis of pneumothorax is entertained, vigilant observation and a high index of clinical suspicion are necessary in the event of progression to a tension pneumothorax. Symptoms should be closely monitored and frequent repeat examinations should be performed.

Tension Pneumothorax. A tension pneumothorax develops when a one-way air leak follows lung rupture or chest wall penetration. Air is forced into the thoracic cavity with no means of escape, and pressure mounts within the hemithorax. With sufficient increases in intrathoracic pressure, the mediastinum is shifted to the contralateral side, which impedes venous return from both the superior and inferior venae cavae. Cardiac output is diminished and the victim soon exhibits signs and symptoms of shock. Victims with tension pneumothorax manifest distended neck veins and tracheal deviation away from the side of the lesion. There is unilateral absence of breath sounds, and the hemithorax is hyperresonant or tympanitic. Respiratory distress, cyanosis, and frank cardiovascular collapse may occur.

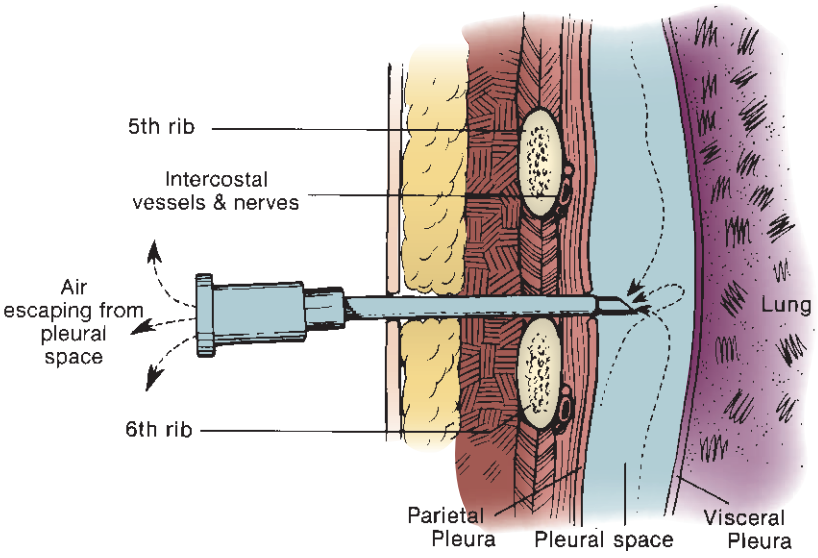
Tension pneumothorax is life-threatening and frequently associated with additional serious injuries. It mandates rapid chest decompression, followed by evacuation to a medical facility.

Decompression is performed by inserting a needle or catheter into the chest and converting the tension into an open pneumothorax. Ideally, a 14-gauge catheter is inserted percutaneously over the second rib in the midclavicular or anterior axillary line (Fig. 20-7). Once the rib is identified with the tip of the needle, the needle is marched over the anterior superior surface of the rib and inserted through the intercostal muscles and pleura into the thoracic cavity. As the pressure within the hemithorax is released, a distinct rush of air is heard. The plastic catheter is advanced over the tip of the needle, the needle withdrawn, and the catheter left in place to ensure continued decompression. The needle should not be reintroduced into the catheter because it may damage or sever the catheter. Because tension pneumothorax is commonly associated with severe injury, the victim should be evacuated to a medical facility as rapidly as possible. A rubber glove or a finger cot can be attached to the external catheter opening to create a unidirectional flutter valve that allows egress of air from the pleural space.

If resources are limited and treatment is needed, any number of devices can be used to decompress the chest. A sharp instrument and hollow tube sterilized as well as possible are all that is needed. Rapid cleansing of the skin surface is accomplished with antiseptic, alcohol, or water. A Heimlich valve kit is ideal for decompression and represents a valuable addition to the expedition first aid arsenal.

If resources permit placement of a thoracostomy tube, adequate anesthesia and expertise are required. The skin should be sterilized if possible, and local anesthesia should be infiltrated into the skin and periosteum of the rib. Insertion is most effectively accomplished through the fifth intercostal space at the anterior axillary line. A small incision is made and the subcutaneous tissue bluntly separated with a finger or clamp. A blunt instrument, preferably a clamp, is forcefully inserted into the pleural space closely adhering to the superior surface of the rib to avoid the inferiorly located intercostal neurovascular bundle. Once the pleural space is entered, a tube (36 fr or greater in size) is inserted apically and posteriorly.

The tube should then be secured with suture or tape and 10 to 20 cm H_2O suction or underwater seal applied. A tube open



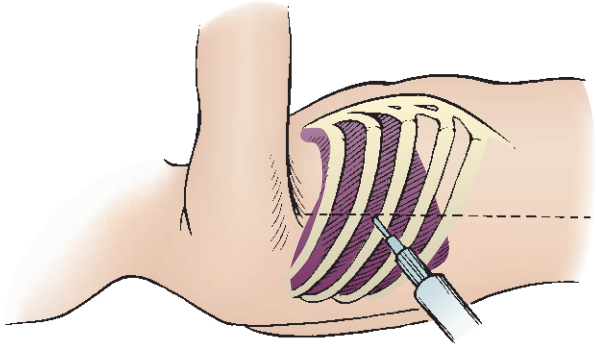


Figure 20-7. Needle decompression of tension pneumothorax. This procedure is performed only for tension pneumothorax in patients with hemodynamic instability.

to the atmosphere can accomplish decompression. The end of the tube can be covered with a rubber glove, finger cot, or plastic bag. One-way flow evacuating the chest is the goal. This procedure is not without morbidity and should be used only by trained personnel under optimal conditions. Antibiotics with gram-positive coverage should be initiated if the pleural space is penetrated with an indwelling catheter or tube.

Hemothorax. Hemothorax is usually associated with multiple rib fractures resulting from a direct blow to the chest. The primary cause of a hemothorax is laceration of the lung, intercostal vessel, or internal mammary artery. The victim complains of chest pain, tenderness associated with rib fractures, inspiratory pain, and dyspnea. Vocal fremitus is absent, percussion may be flat or dull, and breath sounds are diminished or absent.

A chest tube for hemothorax is rarely required in the wilderness setting, but may be placed if proper equipment is available, the patient is symptomatic, and evacuation will be prolonged. Needle aspiration of a hemothorax is unnecessary in the immediate postinjury period and may precipitate a pneumothorax.

Flail Chest. When a series of three or more ribs is fractured in both the anterior and posterior plane, a portion of the chest wall may be mechanically unstable. As negative intrathoracic pressure develops during inspiration, the unstable segment paradoxically moves inward and inhibits ventilation. A flail segment indicates a severe direct blow to the chest wall with associated multiple rib fractures and decreased tidal volumes, often with associated underlying pulmonary contusion. The contusion can be expected progressively to impair ventilation and oxygenation over the succeeding 48 hours. Victims often tolerate a flail segment for the first 24 to 48 hours, after which they require mechanical ventilation.

Any victim with a flail segment should be rapidly evacuated. Because the victim is usually incapable of participating in evacuation, a litter should be prepared or aeromedical evacuation considered. Intercostal nerve block may assist in short-term management of pain and pulmonary toilet. Restrictive (to chest wall expansion during inhalation) external chest wall supports, including taping or extensive stabilization with sandbags, are contraindicated. These measures hinder chest wall movement, decrease vital capacity, and are less effective than intercostal nerve block in pain control. However, focal stabilization or cushioning of the flail segment only to control unnecessary motion and pain may provide minimal relief from the discomfort.

Blunt Cardiac Injuries. Blunt cardiac injuries leading to pericardial tamponade or cardiac contusion are rare. Pericardial tamponade is life-threatening. The pericardial sac is fibrous and expands little. A small amount of intrapericardial blood can severely restrict diastolic function. Blunt injury resulting in tamponade is usually from chamber rupture and rarely survivable, particularly in a remote setting.

The diagnosis of tamponade can be difficult, particularly in the wilderness. Beck's triad, which consists of distended neck veins, hypotension, and muffled heart sounds, is present in less than 33% of cases of tamponade and is particularly difficult to ascertain under nonoptimal conditions. Pulsus paradoxus, an increase in the normal physiologic decrease in blood pressure with inspiration, may be indicative of tamponade. Kussmaul's sign, or a rise in venous pressure with spontaneous inspiration, is possible to assess outdoors.

Once pericardial tamponade is diagnosed, immediate evacuation is required. Treatment consists of median sternotomy in a hospital operating room. The only temporizing measure pending evacuation is pericardiocentesis. This procedure can be lifesaving, particularly if a cardiac injury with a slow leak exists. However, its application in the wilderness setting should occur only if there is a high index of suspicion, coupled with shock and impending death unresponsive to resuscitative efforts. A long (approximately 15 cm [6 in]), 16- to 18-gauge needle with an overlying catheter is introduced through the skin 1 to 2 cm (0.5 to 0.75 in) below and to the left of the xiphoid. The needle is advanced at a 45-degree angle with the tip directed at the tip of the left scapula. When the pericardial sac is entered, aspiration with a syringe follows. The catheter is left in place and secured for possible repeat aspirations as the victim's condition warrants. Immediate evacuation should follow.

Cardiac contusion is a rare condition resulting from a severe blow to the precordium. An overlying sternal contusion or fracture may be present. The diagnosis should be suspected in an isolated high-velocity blow to the precordium with unexplained evidence of increased venous pressure, arrhythmias, and hemodynamic instability. Chest pain is invariably present, usually resulting from musculoskeletal contusion. Morbidity results from ensuing arrhythmias. Diagnosis can be definitively made only at autopsy. Electrocardiographic abnormalities after injury have correlated with subsequent arrhythmias.[55] In the wilderness setting, any person who is unstable or symptomatic from an arrhythmia should be evacuated. If evacuation is not possible, it is noteworthy that fatal arrhythmia potential decreases significantly after 24 hours.

Traumatic Asphyxia. Traumatic asphyxia is a rare syndrome of craniocervical cyanosis, facial edema, petechiae, subconjunctival hemorrhage, and occasional hypoxemia-related neurologic symptoms that results from severe thoracic crush injury. In the wilderness environment, it is associated with land or mudslides, avalanches, or falling debris. Any significant blunt compressive force to the thorax can result in the syndrome. Children are particularly susceptible because of high compliance of the chest wall.[16] Traumatic asphyxia is not a benign condition, as a result of a high incidence of serious associated injuries,[71] and the mortality rate in natural disasters is consequently high.[33] A number of studies have documented the severity of associated injuries, with the syndrome useful as an indicator of potentially lethal injury.[24] As documented in natural disasters, a significant crush injury component may accompany traumatic asphyxia.[33] Crush injuries and rhabdomyolysis are discussed later in the Extremity Trauma section of this chapter.

Figure 20-8. Typical clinical facial appearance of traumatic asphyxia.

The pathophysiology of traumatic asphyxia involves two elements. The crush injury results in acute increases in intrathoracic pressure and thus inferior and superior vena caval pressures. Venous flow is reversed in the veins of the head, which contain no valves. Venous hypertension leads to capillary rupture and the characteristic facial edema and petechiae. Recognition of the physical findings is imperative in diagnosing the syndrome and identifying concomitant injuries (Fig. 20-8).

Treatment consists of carefully extracting and, if necessary, immobilizing the victim. Rapid extrication is the single most important factor in improving survival. Establishment and maintenance of an airway is critical because significant facial and laryngeal edema may rapidly develop. Associated injuries should then be addressed in the primary survey. Subsequent care is supportive, consisting of airway control, administration of oxygen, head elevation of 30 degrees, treatment of associated injuries, and possible evacuation.

The mortality rate is low in civilian environments but higher in wilderness disaster settings. Mortality is due to pulmonary dysfunction and associated injuries. Morbidity is secondary to neurologic damage; however, the majority of neurologic sequelae clear within 24 to 48 hours. If the victim survives, long-term sequelae are rare.

Penetrating Chest Trauma

Penetrating chest trauma above the nipple line is associated with hemopneumothorax and may also be associated with significant visceral injury. A victim with penetrating chest trauma below the nipple line often has intra-abdominal penetration in addition to possible thoracic injury. Such a victim requires immediate rapid evacuation.

The open ("sucking") chest wound produces profound intrathoracic physiologic alterations. Normal chest expansion creates negative intrathoracic pressure, which pulls air into the trachea and allows the lungs to expand. When the diaphragm and chest wall relax, positive pressure creates expiration. If the chest wall sustains an injury approximately two thirds the tracheal diameter, negative intrathoracic pressure for inspiration is lost, the ipsilateral lung collapses, and loss of negative

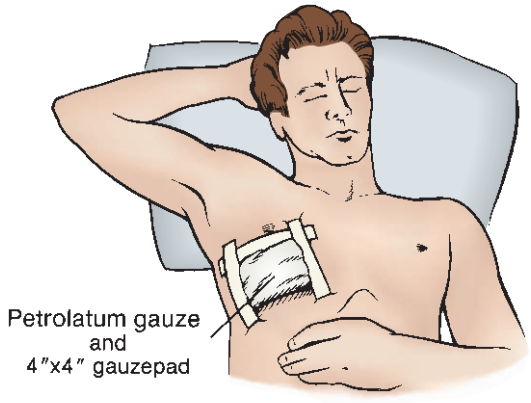


Figure 20-9. Treatment of a sucking chest wound. Sealing the wound with a gel defibrillator pad works best because this pad adheres to wet or dry skin. Petrolatum gauze or Saran Wrap also works well. Note that one side is not sealed to allow egress of air.

intrathoracic pressure affects the good lung. Consequently, it is important rapidly to reconstruct chest wall integrity. Initially, this is most easily done by placing a hand over the sucking chest wound. Field treatment includes placing petrolatum gauze on top of the wound, covering it with a 4 × 4 gauze pad, and taping it on three sides (Fig. 20-9). The untaped fourth side serves as a relief mechanism to prevent tension pneumothorax. Persons with sucking chest wounds should be rapidly evacuated to sophisticated medical care.

Injuries to the Abdomen

Intra-abdominal injuries in the wilderness setting are difficult to recognize. However, if recognized, all intra-abdominal injuries require rapid resuscitation and immediate evacuation. The abdomen represents the most frequent site of life-threatening hemorrhagic shock; however, in the wilderness setting, few diagnostic and treatment options exist.

Blunt Abdominal Trauma

Blunt intra-abdominal injury is commonly associated with falls. Abdominal injuries are often associated with fractures or closed head injuries. Often, the decision for evacuation is made on the basis of other injuries; however, the wilderness physician must be attuned to the potential for intra-abdominal hemorrhage as an occult injury.

Anatomy. For descriptive purposes, the abdomen may be divided into thoracic, true, and retroperitoneal compartments. The thoracic abdomen contains the liver, spleen, stomach, and diaphragm. The liver, spleen, and, more rarely, stomach may be injured by direct blows to the ribs or sternum. Twenty percent of persons with multiple left lower rib fractures have a ruptured spleen. A direct blow to the epigastrium may result in increased intra-abdominal pressure with subsequent rupture of the liver or diaphragm. The true abdomen contains the small bowel, large bowel, and bladder. Isolated bowel injuries are rare in the wilderness setting. Blunt bladder or rectal injury usually occurs in conjunction with severe pelvic fracture and carries high mortality. The retroperitoneal abdomen contains the kidneys, ureters, pancreas, and great vessels. It is notoriously difficult to evaluate by physical examination. Life-threatening hemorrhage can occur into the true abdomen or retroperitoneal space.

Diagnosis. Although much progress has been made in the last decade to evaluate for the presence of blunt intra-abdominal injury, modalities such as CT, ultrasonography, and diagnostic peritoneal lavage are irrelevant in the wilderness setting. The wilderness physician must have a high index of suspicion and perform a superlative history and physical examination.

Physical Examination. The physician should look for signs of early shock: tachycardia, tachypnea, delayed capillary refill, weak or thready pulse, and cool or clammy skin. Physical examination of the abdomen begins with visualization and inspection. Contusions and abrasions may be the only harbingers of occult visceral injury. Periumbilical ecchymosis associated with abdominal hemorrhage (Cullen's sign) is virtually never present in a victim with acute abdominal trauma. Abdominal distention secondary to hemorrhage is a very late sign and never present before shock and cardiovascular collapse. Abdominal inspection should survey the flanks, lower chest, and back. Inspection of the back should follow palpation of the spine while the victim is supine. The victim should be very carefully logrolled if there is any suspicion of spinal injury.

Looking for muscle guarding, the examiner gently palpates the abdomen in all four quadrants. Any persistent guarding or tenderness after wilderness trauma mandates rapid evacuation. Percussion tenderness is an indicator of peritoneal irritation, also mandating evacuation. The presence or absence of bowel sounds has little prognostic significance. Bowel sounds may be present in the face of significant intra-abdominal hemorrhage or, conversely, absent in victims when extra-abdominal injuries induce ileus.

Referred pain to the left shoulder (Kerr's sign) strongly suggests the presence of a ruptured spleen. This pain is often exaggerated by placing the victim in Trendelenburg's position, increasing the amount of left upper quadrant blood irritating the diaphragm. Pain from the retroperitoneal abdomen associated with injuries to the kidney or pancreas may be referred to the back. However, referred pain is usually a late finding and not helpful in the evaluation of acute trauma.

Gross hematuria that does not clear immediately or is coupled with an associated injury, such as pelvic fracture or abdominal or back pain, requires immediate evacuation. To minimize blood loss, the victim should be kept stationary and the evacuation team brought as close to the victim as possible.

In a wilderness setting, rectal and vaginal examination adds little to the evacuation decision when evaluating for abdominal trauma. The unstable pelvic fracture associated with rectal and vaginal injuries is usually the determinant for evacuation.

Penetrating Abdominal Trauma

Penetrating intra-abdominal injuries may result from gunshot, stab, or arrow wounds. The social context in which these injuries occur (accidental, intentional, or self-inflicted) makes little difference in the wilderness setting. Recrimination, guilt, and blame only interfere with the paramount goal of immediate evacuation.

Gunshot Wounds. Low-caliber gunshot injuries often present with small entrance and no exit wounds. High-caliber, high-

velocity gunshot injuries may have relatively innocuous entrance wounds but may be associated with large, disfiguring exit wounds and extensive internal injuries. No matter what the caliber or trajectory and no matter where the entrance and exit, all gunshot wounds from the nipple line to the inguinal ligament should be presumed to have penetrated the abdominal cavity and created an intra-abdominal injury. These injuries mandate immediate surgical intervention. A victim of gunshot wounds to the head, neck, chest, abdomen, or groin should undergo immediate evacuation accompanied by the administration of a single-agent broad-spectrum antibiotic, such as an oral fluoroquinolone (e.g., ciprofloxacin, 750 mg PO bid). Hunting injuries are discussed in Chapter 22.

Shotgun Injuries. Shotgun injuries to the torso are managed in the same manner as gunshot wounds. Shotgun injuries have a potentially lower incidence of underlying visceral injury than gunshot wounds, but there is often extensive soft tissue damage requiring surgical debridement. The potential exists for delayed development of peritonitis from a single penetrating pellet to the viscera. Consequently, shotgun injuries should also be treated with emergency evacuation and a broad-spectrum antibiotic, as recommended previously for gunshot wounds.

Occasionally, a close-range shotgun blast results in a soft tissue defect large enough for the injured bowel to extrude through the wound. The injured bowel should not be placed back into the abdomen. Injured bowel displaced from the abdominal cavity conceptually should be treated as though it were an enterocutaneous fistula. Because evacuation is often delayed in the wilderness, it is better to have fecal contents outside, rather than inside, the peritoneal cavity. The exteriorized bowel should be kept moist and covered at all times. Uncovered bowel outside the peritoneal cavity rapidly desiccates and becomes nonviable, mandating later surgical resection. Exposed bowel should be covered with an abdominal pack or cloth moistened with sterile saline at best, or at worst with potable water. The dressing should be checked and remoistened at least every 2 hours.

Stab Wounds. The penetrating object is usually a knife but may be as varied as a piton, ski pole, or tree limb. Any deep skin laceration from the nipple line to the groin should be considered to have damaged an intra-abdominal organ. Whereas the odds of an abdominal gunshot wound injuring a visceral organ exceed 85%, the odds of a stab wound injuring a visceral organ are less than 50%.

In certain urban hospitals, the high incidence of negative surgical explorations for stab wounds had led to a more selective approach toward patients with abdominal stab wounds.[69] This approach uses local wound exploration and frequent physical examination.

Although there are no data addressing the management of stab wounds in the wilderness environment, the following approach is practical and reasonable. If the wound extends into the subcutaneous tissue, the evacuation decision depends on local wound exploration. This procedure is simple to perform, even in the wilderness environment. The skin and subcutaneous tissue are infiltrated with local anesthetic, and the laceration is extended several centimeters to clearly visualize the underlying anterior fascia. It is helpful to use lidocaine (Xylocaine) 1% with epinephrine to minimize slight but annoying bleeding that can impair visualization. The wound should never be probed with any instruments, particularly if overlying the ribs.

Wound exploration is confined to the area from the costal margin to the inguinal ligament. Local wound exploration is contraindicated in wounds that extend above the costal margin, because it is possible for such exploration to communicate with a small pneumothorax, potentially exacerbating respiratory distress.

If thorough exploration of the wound shows no evidence of anterior fascial penetration, and if the victim demonstrates no evidence of peritoneal irritation, the wound can be closed with tape (Steri-Strips) or adhesive bandages, dressed, and the evacuation process delayed. Physical examination should be performed every few hours for the next 24 hours. If no peritoneal signs develop and the victim feels constitutionally strong, a remote expedition may resume with caution and an eye to evacuation should the victim become ill.

In the wilderness environment, it is prudent to have a low threshold for evacuation because of technical difficulties in performing wound exploration, such as insufficient light and inadequate instruments. Persons who have been impaled by long objects, such as tree limbs or ski poles, should have the object left in place and carefully shortened, if possible, to facilitate transport.

Pelvic Trauma

In the wilderness setting, fractures of the pelvis are generally associated with falls from significant heights, high-velocity ski accidents, or vehicular trauma.

Pelvic fractures can be lethal. With opening of the pelvic ring, there may be hemorrhage from the posterior pelvic venous complex and occasionally from branches of the internal iliac artery. For hemodynamically unstable victims with severe pelvic fracture, resuscitative efforts should be instituted. In addition, simple techniques to reduce any increased pelvic volume through the application of sheets or slings may slow bleeding.

The key factor in initial management of pelvic fractures is identification of posterior injury to the pelvic ring. Posterior ring fractures or dislocations are associated with a greater incidence of significant hemorrhage, neurologic injury, and mortality than are other pelvic fractures. The diagnosis of a posterior ring fracture is based on instability of the pelvis associated with posterior pain, swelling, ecchymosis, and motion. Persons with posterior ring fractures must be immediately evacuated on backboards, with care taken to minimize leg and torso motion.

The flank, scrotum, and perianal area should be inspected for blood at the urethral meatus, swelling or bruising, or a laceration in the perineum, vagina, rectum, or buttocks suggestive of an open pelvic fracture. The pelvis should be examined carefully once, without any aggressive rocking motion. The first indication of mechanical disruption is leg length discrepancy or rotational deformity in the absence of an obvious leg fracture or hip location. For more information on pelvic fracture, see Chapter 24.

Extremity Trauma

The majority of wilderness-related extremity injuries involve fractures and sprains, which are discussed in Chapter 24. This section focuses on the general field management of significant extremity vascular injury, traumatic amputation, and recognition and treatment of rhabdomyolysis.

Vascular Injuries

Injury to the major vessels supplying the limbs can occur with penetrating or blunt trauma. Fractures can produce injury to the vessels by direct laceration (rarely) or by stretching, which produces intimal flaps. Penetrating injuries can be devastating if transection of a vessel occurs. Significant vascular injuries, from both penetrating and blunt causes, can result in multiple vessel injury subtypes, each of which may be limb-threatening. Injury subtypes include laceration, transection, contusion with spasm, thrombosis, or aneurysm formation (true and false), external compression, and arteriovenous fistula. An accurate history, expeditious physical examination, and swift evacuation are the keys to life and limb salvage.

History. A complete history of the time and mechanism of injury is invaluable in planning further management. Although no absolute ischemia time has been established, a goal of less than 6 hours to reperfusion is prudent.[30] The amount of blood present at the scene should be quantified. A history of bright pulsatile blood that abates is suggestive of arterial injury. Thirty-three percent of victims with arterial injuries have intact distal pulses.

Physical Examination. Vascular examination in the field can be highly variable. Hypovolemia, hypothermia, and hostile conditions make an accurate examination challenging. Skin color and extremity warmth should be assessed first. Distal pallor and asymmetric hypothermia are suggestive of a vascular injury. Pulses should be palpated. In the upper extremity, the axillary, brachial, radial, and ulnar arteries should be assessed. In the lower extremity, the femoral, popliteal, posterior tibial, and dorsalis pedis pulses should be assessed. Location and direction of the wound should be determined, hemorrhage quantified, and the presence of hematomas or a palpable thrill noted.

A good neurologic examination that quantifies motor and sensory deficits is critical. Because of the high metabolic demands of peripheral nerves, disruption of oxygen delivery makes neuronal cells highly susceptible to ischemic death. Conversely, skeletal muscle is relatively resistant to ischemia. Loss of sensation or limb paralysis is an alarming sign of impending anoxic necrosis.

Treatment of Vascular Injuries. Significant hemorrhage should be identified and controlled in the primary survey. All hemorrhage should be controlled with direct pressure at the site of injury. Tourniquets should be applied only when direct pressure fails to control bleeding. Tourniquets should be released every 5 to 10 minutes to prevent further ischemia.

Hematomas should never be explored or manually expressed. Attempts to clamp or ligate vessels are not recommended. Frequent repeat neurovascular examinations are mandatory.

Once bleeding is controlled and the wound is covered with a sterile but noncompressive dressing, completion of the primary survey, identification and stabilization of associated injuries, and appropriate resuscitation with normal saline should follow. The extremity should be splinted to prevent further movement. The need for evacuation depends directly on the results of the physical examination. Examination results can be grouped into "hard signs," indicative of ischemia or continued hemorrhage, and "soft signs" that are suggestive but not indicative of ischemia (Boxes 20-3 and 20-4).

Box 20-3. Vascular "Hard Signs"

Pulsatile bleeding
Palpable thrill
Audible bruit
Expanding hematoma
Six "P's" of regional ischemia
- Pain
- Pulselessness
- Pallor
- Paralysis
- Paresthesia
- Poikilothermia

Box 20-4. Vascular "Soft Signs"

Injury in proximity to major vessel
Diminished but palpable pulses
Isolated peripheral nerve deficit
History of minimal hemorrhage

All victims with hard signs should be evacuated emergently. Based on current data, an isolated soft sign may warrant observation alone, depending on the remoteness of the expedition and the risks of evacuation. The data for observation of soft signs have emerged from hospital settings and must be applied with great caution in the wilderness. If soft signs are present, clinical suspicion is high, and evacuation can be accomplished safely, the victim should be transported and observed in a medical facility.

Traumatic Amputation

In the wilderness environment, amputation victims require immediate evacuation. Hemorrhage is controlled during the primary survey with direct pressure, and resuscitation is instituted. Tourniquets are rarely required. The victim should be kept warm and calm. Reassurance and analgesics should be administered. Amputations should be completed only if minimal tissue bridges exist and it is clear that the neurovascular supply has been interrupted.

Amputation of a mangled extremity, defined as an extremity with high-grade open fracture and soft tissue injury, should not be carried out in the wilderness except to free a trapped victim in order to avoid further severe injury or even death, or in the case of uncontrollable hemorrhage threatening the life of the victim, and then only by experienced surgical personnel. All other severely injured extremities should be wrapped in available sterile materials, splinted, and kept moist.

Amputated extremities should be cooled if possible, optimally in a plastic bag in ice or ice water. Avoid placing the extremity in direct contact with ice. Without cooling, the amputated extremity remains viable for only 4 to 6 hours; with cooling, viability may extend to 18 hours. The amputated extremity should accompany the victim throughout the course of the evacuation.

Crush Injuries and Rhabdomyolysis

Rhabdomyolysis is a potentially fatal syndrome that results from lysis of skeletal muscle cells. In its fulminant form, rhabdomyolysis can affect multiple organ systems. Compartment

syndrome, renal failure, and cardiac arrest represent the major complications.

Any condition resulting in significant acute or subacute striated muscle damage can precipitate rhabdomyolysis. Crush injuries of the extremities and pelvis, revascularization of ischemic tissue, ischemic extremities, animal bite and snakebite,[17,23] frostbite, and traumatic asphyxia[33] can all result in rhabdomyolysis in a wilderness setting. Crush injuries are frequently a result of avalanches, falls from heights, or rock slides.

The pathophysiology of rhabdomyolysis remains controversial. The exact mechanism of muscle injury appears not to be simple direct force or isolated ischemia and is probably multifactorial.[33] The common cellular derangement is interference of the normal function of muscle cell membrane sodium-potassium adenosine triphosphatase (ATPase) with intracellular calcium influx and cell death.[77] After cell death, multiple intracellular constituents, including myoglobin, creatine kinase, potassium, calcium, and phosphate, are released into the systemic circulation.

The metabolic derangements of rhabdomyolysis depend directly on release of intracellular muscle constituents. Myoglobinemia, hypercalcemia, hyperphosphatemia, hyperkalemia, hyperuricemia, metabolic acidosis, coagulation defects, and contracted intravascular volume result.

The clinical presentation of rhabdomyolysis may include muscle weakness, malaise, fever, tachycardia, abdominal pain, nausea and vomiting, or encephalopathy. Symptoms may mimic those of persons with spinal cord injury.[8] The danger of the syndrome lies in the cardiovascular effects of electrolyte disturbances and renal failure[70] secondary to changes in renal perfusion and direct toxicity of myoglobin to tubular cells.

Successful treatment relies on prompt diagnosis based on clinical signs and urinalysis, aggressive hydration, and forced diuresis. Myoglobin turns urine tea-colored, which is an important indicator of significant muscle death and need for aggressive treatment.

Normal saline should be administered IV at 1 to 2 L/hr to achieve a urine output of 100 to 300 mL/hr. Victims who are trapped in rubble should have resuscitation initiated before extrication, if possible. The addition of agents to alkalinize the urine and promote diuresis has been shown to improve clearance of myoglobin but not to alter survival. In addition, diuretics may be detrimental in multisystem trauma victims who are hypovolemic. All victims demonstrating myoglobinuria should be evacuated.

Injuries to the Skin and Wilderness Wound Management

The goals of wilderness wound management are to minimize wound complications and promote healing. Treatment should begin with an approach to the victim as a trauma patient. Within the context of the primary survey, hemorrhage should be controlled. Then, the victim should be examined and the wound inspected. Steps to minimize infection should be undertaken, incorporating anesthesia, assessment of need for tetanus immunization and antibiotics, irrigation, and debridement. After attempts to minimize infection, a definitive plan should be established, including the need for evacuation (Box 20-5).

Box 20-5. Guidelines for Wilderness Wound Management

1. Identify and stabilize associated traumatic injuries
2. Control hemorrhage
3. Examine wound
4. Minimize infection
   a. Tetanus immunization as indicated
   b. Antibiotics for high-risk wounds
   c. Irrigation
   d. Debridement
5. Implement definitive care

Wound Morphology

Major lacerations are often the most obvious sign of trauma; however, injuries to the integument are rarely life-threatening. Contusions, abrasions, and lacerations should force the examiner to focus on areas of potential occult injury. Contusions often overlie extremity fractures or, when present on the torso, suggest the potential for underlying visceral injury. Extremity lacerations may be associated with fractures or may extend into the joint space.

The four basic types of skin injuries are lacerations, crush injuries, stretch injuries, and puncture wounds. Lacerations rarely require closure in the wilderness environment. Commonly, multiple wound morphologies are present in the same injury, and an array of wound presentations is possible.

Crush injuries may be associated with significant tissue necrosis, impaired healing, increased rates of infection, and underlying muscle damage with subsequent rhabdomyolysis. Fortunately, they are rare in the wilderness.

Stretch injuries produce a split in the skin but, more important, may be associated with underlying nerve or tendon damage.

Puncture wounds often appear innocuous but have a high propensity for infection. Animal bite wounds, discussed in detail in Chapters 51 and 52, can manifest any of these wound morphologies alone or in combination.

Primary Survey

Many skin and soft tissue wounds encountered in the wilderness setting accompany significant injuries. Therefore, the wound must never distract the physician from associated life-threatening injuries. The ATLS primary survey should be performed in the usual fashion.

Control of Hemorrhage

The vast majority of bleeding is controlled with direct pressure, applying the most sterile covering available. Applying pressure over major arterial pressure points is discouraged, as is the use of tourniquets. In the event of bleeding not controlled by direct pressure, tourniquets may be applied with the knowledge that limb sacrifice is possible. If applied, tourniquets should be released every 5 to 10 minutes if possible to restore perfusion transiently and to assess if they are still necessary. Clamping bleeding vessels is not advised because this may cause unnecessary neurovascular injury.

Physical Examination

Wound inspection and physical examination are critical in any setting. This phase of treatment may need to be abbreviated and should not delay packaging and evacuation. Although it is important to assess the extent of injury, including tissue loss and

underlying musculoskeletal and neurovascular injury, aggressive wound exploration may worsen existing injuries.

Detailed knowledge of regional anatomy is useful. The detailed neurovascular examination should be documented before anesthesia and definitive care, including assessment of pulses and regional perfusion. The neurologic examination should quantify sensory and motor function, with particular attention to functional assessment of muscle groups traversing the injured region. Two-point discrimination should be assessed in wounds involving the hands or fingers. Wounds over joints and tendons should be put through full range of motion.

Anesthesia

Pain management in the wilderness is discussed in Chapter 17. Administration of anesthesia occurs before mechanical wound cleansing and definitive care. The three methods of anesthetic administration briefly discussed here are topical, local, and regional.

Topical anesthesia was originally introduced for mucosal lacerations but has been shown to be effective for skin wounds. TAC (sterile tetracaine 0.5%, adrenalin [epinephrine] 1:2000, and cocaine 11.8% in saline) has been used with success as a topical anesthetic. Complications have included seizures and death.[93] An alternative preparation consisting of lidocaine, adrenalin, and tetracaine (LAT) has been shown to be as effective as TAC without the associated complications.[29] Topical anesthetics may be soaked into a sterile gauze and placed on the wound surface for 7 to 10 minutes. Disadvantages of topical anesthetics include potential for a slightly increased risk of infection and less versatility than locally injected lidocaine.

Local anesthesia is the standard method for achieving soft tissue analgesia. Typically, 1% lidocaine without epinephrine is used. In adults, the maximum injectable dose of lidocaine is 4 mg/kg. Lidocaine should not be injected directly from within the wound to the periphery because this increases the chance of introducing bacteria deeper into the soft tissue.[64] The injection should proceed from the periphery of the wound, with each successive needle stick entering the skin through a previously anesthetized area.

Local anesthesia can be administered with relatively little discomfort using a 25-gauge needle and a 1-mL tuberculin syringe. Although using a small syringe increases the time to anesthetize a larger wound, this method minimizes both the anesthetic dose and distortion of soft tissue planes, facilitating tissue repair. Pain associated with administration of local anesthesia is due to the acidity and stretching of nerve endings within the dermis and subcutaneous tissue. Burning sensation associated with lidocaine injection is directly proportional to the rate of administration. Warming the local anesthetic,[46] buffering the solution with sodium bicarbonate to a concentration of 1%, and administering anesthetic slowly in small doses all minimize pain.

Regional anesthesia, defined as sensory nerve blockade proximal to the wound, is an excellent mode of anesthetizing wounds of the upper and lower extremities. Two types are regional nerve block and Bier block. Regional nerve blocks require skill and a detailed knowledge of regional anatomy. They are not suitable for the first-time user in the wilderness environment.

The Bier block is reasonable to administer and is possible in the wilderness setting. It involves injection of local anesthetic into a cannulated hand or foot vein, with concurrent control of venous outflow using a tourniquet.

Irrigation

Once the wound is anesthetized, irrigation, debridement, and closure can proceed. Irrigation removes dirt, debris, foreign bodies, and bacteria from the wound. Irrigation has been extensively studied in traumatic wounds and clearly results in a decreased incidence of infection, reducing infection rates as much as 20-fold when proper technique is used.

The type of irrigation fluid and the technique used are resource dependent in the wilderness setting. The cleanest fluid available should be used. Wilderness fresh water sources that are not grossly contaminated can be boiled or filtered.[32] Any concentration of sterile crystalloid solution can be used, although normal saline remains the most readily available, economical, and cost-effective irrigation solution. Recent data suggest that tap water may be as effective as normal saline.[66]

The amount of irrigation necessary is difficult to quantify. Some authors use 60 mL (2 ounces) of irrigation solution per centimeter wound length as a guide,[46] but in the wilderness setting where precision is more difficult to attain, irrigation should be continued in amounts and time intervals sufficient to remove visible debris from the wound.

Many bactericidal and bacteriostatic irrigation solutions, including commercial soaps, ethyl alcohol, iodine solutions, and hydrogen peroxide, are available in wilderness first-aid kits. Many of these agents have been shown to result in significant microcellular destruction of tissues[15] and, when used in high concentrations, may impair wound healing. They offer no advantage over copious irrigation with sterile water or crystalloid. Although addition of antibiotics to irrigation solutions is an attractive concept, they are costly, difficult to store, and offer no advantage over irrigation with sterile water alone.

The method of wound irrigation, as well as the pressures used, have been studied extensively. The goals of irrigation are to remove bacteria, assist in the mechanical debridement of necrotic tissue, and remove foreign bodies that can impair subsequent wound healing. Optimal irrigation pressures are 5 to 8 psi, delivered through a syringe with a 16- to 20-gauge needle. A splash shield attached to the syringe should be used if available.

In summary, irrigation consisting of normal saline or sterilized potable water should be delivered in a continuous fashion by the most sterile implement available at a pressure sufficient to dislodge debris but not overtly damage tissue.

Debridement

Like irrigation, debridement has been shown to decrease the incidence of wound infection. In addition, debridement has the potential to improve long-term cosmesis. Debridement should be carried out sharply. Scrubbing the wound with abrasive materials does not improve infection rates and may cause damage to healthy tissue. Similarly, soaking the wound has never been shown to improve outcome. Hair removal should be undertaken only if it impairs visualization and inspection of the wound,[93] or if tape is to be used as a method of temporary closure.

The goal of debridement in the wilderness should be to remove grossly contaminated or devitalized tissue and to remove foreign bodies and bacteria embedded in such tissue.

TABLE 20-3. Tetanus Prophylaxis

HISTORY OF IMMUNIZATION (DOSES)	CLEAN MINOR WOUNDS		DIRTY MAJOR WOUNDS	
	TOXOID*	TIG†	TOXOID	TIG
Unknown	Yes	No	Yes	Yes
None to one	Yes	No	Yes	Yes
Two	Yes	No	Yes	No (unless wound older than 25 hr)
Three or more				
Last booster within 5 years	No	No	No	No
Last booster within 10 years	No	No	Yes	Yes
Last booster more than 10 years ago	Yes	No	Yes	Yes

*Toxoid: Adult: 0.5 mL dT intramuscularly (IM).
Child less than 5 years old: 0.5 mL DPT IM.
Child older than 5 years: 0.5 mL DT IM.
†Tetanus immune globulin (TIG): 250–500 units IM in limb contralateral to toxoid.

The extent of tissue removal should be based on the experience and training of the caregiver.

Tetanus Prophylaxis

Spores of *Clostridium tetani* are ubiquitous in the environment in such places as soil, animal teeth, and saliva. Any animal bite that penetrates the skin can be responsible for a tetanus infection. The majority of cases of tetanus infection in the United States follow failure to attain adequate immunization.[79] This fact accentuates the preventable nature of tetanus infections and essential role of proper immunization. If available in the wilderness setting, tetanus prophylaxis should be administered as outlined in Table 20-3.

Definitive Care of Lacerations

"Definitive" care may have many definitions, depending on the setting. The planned approach to management of wounds in the wilderness is determined by a combination of morphology of the wound, infection risk factors, available resources, level of expertise, and type of expedition.

Major lacerations or those associated with significant injury should be evacuated. If wound management cannot acceptably minimize infection risk factors, the victim should be evacuated. In general, wounds that can be closed or managed open and that do not impose excessive infection risk factors and do not immobilize the expedition member or group can be treated definitively in the wilderness. The wound must not impair physical ability in a way that the victim risks further injury or jeopardizes group safety.

Wound Closure. Lacerations can be closed if they are small to intermediate in size; have minimal infection risk factors; are on well-vascularized regions, such as the scalp and face; are less than 6 hours old; and have no anatomic contraindications.

Closure may be accomplished with suture, staples, tape and similar bandages, or adhesives. Tape and, less frequently, adhesives are viable alternatives to sutures. Healing and cosmetic outcome depend directly on dermal apposition, which is the goal of any closure method.

Advantages of sutures include meticulous closure and high wound tensile strength. The primary disadvantage is the skill necessary to place them. The suture selected is dictated by morphology of the wound. To simplify selection in the wilderness environment, absorbable sutures, such as chromic gut and polyglactin (Vicryl), should be used to close deep layers and for subcuticular closures. Nonabsorbable sutures, such as nylon (Ethilon) and polypropylene (Prolene), should be used on the skin. Silk is reactive, has poor tensile strength, and should be avoided. No wound should be sutured by an individual who is inexperienced in basic surgical technique. In addition, no wound incurred in the wilderness is truly risk-free regarding infection. In general, the safest management strategy for lacerations in the wilderness setting is open management or closure with nonsuture alternatives.

Surgical staples are easily placed, are nonreactive, have lower infection rates than sutures, and minimize time of closure.[93] They should be avoided on areas of cosmetic importance, such as the face.

Tapes and adhesives offer a preferable alternative to sutures. Tape and adhesive strips (e.g., Steri-Strips) are easily applied and require little technical ability. If a wound is appropriate for closure, tape offers a rapid, safe, painless, and inexpensive alternative to sutures and staples.[27] The only requirement of tape use is conformity to principles of dermal apposition. Disadvantages of tape include need for adhesive solutions, such as benzoin; low tensile strength; and lack of applicability over any region of tension.[93] A critical limiting factor in wilderness use of tape closure is the need for the wound to remain dry.

Tissue adhesives in wound closure have been studied for 20 years.[26] Recently, octylcyanoacrylate and similar synthetic agents have been shown to be equal in strength and cosmesis to sutures at 1 year.[81] Advantages of adhesives include ease of application, safety, patient comfort, and low cost.[94] Similar to tape, immediate tensile strength is poor and dehiscence is more likely compared with sutures.[93] If closure is possible and other means are unavailable or impractical, adhesives may be used.

Scalp Lacerations. The extent and severity of scalp lacerations are often initially obscured by surrounding hair that is matted with blood. Hydrogen peroxide and water effectively remove blood from hair, although hydrogen peroxide should not be

used to irrigate the wound. Interestingly, and most likely owing to the vascularity of the scalp, irrigation did not alter outcome in clean lacerations in one study.[43]

Hair surrounding the laceration is removed using a safety razor only if absolutely necessary to clean the wound. Hair removal should be limited to the immediate area of the laceration because the surrounding hair can later be twisted into strands and used to approximate the wound edges if necessary. Once wound margins have been identified, anesthetic should be applied.

The key to examination of the scalp is determining the integrity of the galea. Significant degloving injury or galeal laceration may mandate evacuation. The scalp is highly vascularized. An extensive scalp laceration bleeds freely, and if it follows a fall or direct blow to the head, may be associated with an underlying skull fracture. Superficial scalp lacerations often bleed freely and may require pressure dressings to achieve hemostasis.

Minor scalp lacerations can be effectively treated in the wilderness setting, after following the aforementioned infection-minimizing steps. Of note, debridement of scalp wounds should be kept to a minimum because it may be difficult to mobilize wound edges to cover the resulting soft tissue defect. In addition, cosmesis is not a significant concern on the hair-covered scalp. Acceptable closure of a minor scalp laceration can be performed using strands of hair to approximate wound edges. The hair on either side of the wound can be twisted into a thick bundle. The opposing bundles are then pulled together and crossed. They can be secured with a drop of tissue glue or tied together if glue is not available (see Figure 21-45). This method minimizes shaving.

Facial Lacerations. Facial lacerations are relatively simple to manage because they rarely damage underlying structures and are well vascularized. If suspicion exists regarding damage to cranial nerves or the parotid duct, the wound should be managed in an open fashion. Debridement should be limited to obviously necrotic tissue. Because of vascularity of the face, infection is rare, and most wounds can be closed. For small wounds, tape is a useful closure technique.

Torso Lacerations. Torso lacerations require evaluation for fascial penetration. Anterior fascial penetration in the torso converts the wound from a skin wound to one requiring management of underlying chest or abdominal structures. Tissue debridement may be more aggressive over the torso because surrounding tissue planes can be mobilized for closure. Adipose tissue should not be approximated with sutures, and subdermal dead space should be obliterated with deep, nonadipose approximating sutures.

Hand Injuries

Severe contusions to the hand commonly occur with crush or rope injuries. The hand should be carefully protected if marked swelling and pain with motion are present. If no joint instability or fracture is identified, a bulky hand dressing should be applied with the wrist dorsiflexed 10 degrees, the thumb abducted, and the metacarpophalangeal joints flexed 90 degrees, known as the position of function. Cotton wadding or bandages can be placed in the palm and between the fingers, and an elastic bandage can be used as an overwrap. A volar splint allows this position to be maintained until definitive care is reached.

Lacerations of the finger flexor or extensor tendons occur with accidents involving knives or other sharp objects. A flexor tendon laceration, partial or complete, can be a serious problem if not repaired early. The open wound should be cleansed and loosely taped closed if no infection risk factors are present, and the finger should be splinted in slight flexion at the interphalangeal joints and in 90 degrees of flexion at the metacarpophalangeal joint. To achieve optimal results, this injury should be managed by a hand surgeon within the first 3 to 5 days.

For an extensor tendon, the open wound should be cleansed and taped closed, and a splint should be applied with the metacarpophalangeal joint in slight flexion and the interphalangeal joint extended. The victim should be seen by an orthopedic surgeon within 7 days.

The nerves most commonly injured by laceration include the superficial radial nerve at the wrist, ulnar nerve at the elbow or wrist, and median nerve at the wrist. Digital nerves are commonly lacerated in accidents with knives. In general, the wound should be cleansed and taped loosely and a splint should be applied to the wrist and hand. The victim should see a hand surgeon within 7 days.

Puncture Wounds

Puncture wounds carry significant infection risk where organic contamination is frequent. Significant puncture wounds to the torso should be treated according to the guidelines outlined in the section on penetrating trauma to the chest and abdomen. Puncture wounds to the extremities should be unroofed if they are proximal to the wrist or ankle. The unroofed wound should be irrigated as previously described and then packed open with sterile gauze. Delayed primary closure with tape can occur at 48 to 96 hours. Puncture wounds to the hands and feet should not be explored in the absence of detailed knowledge of anatomy. If this expertise is not available, the wound should be cleaned and the victim started on antibiotics, such as cephalexin (Keflex) 500 mg PO q6h.

If the skin is punctured with a fishhook, the skin surrounding the entry point should be cleansed with soap and water. Fishhook removal techniques are discussed in Chapter 22.

Antibiotic Use and Infectious Complications

The use of prophylactic antibiotics for wounds incurred in the wilderness is not recommended. Antibiotic treatment usually begins after the injury has occurred and therefore is never truly "prophylactic." The use of antibiotics in lacerations and bite wounds should be confined to victims with significant infection risk factors, such as animal bites, heavily contaminated wounds, or comorbid medical conditions. This includes high-risk wounds, such as puncture wounds and those occurring on the hands. Debridement and irrigation are far more important than antibiotics in eliminating infection and remain the mainstay of risk reduction in any wound incurred in the wilderness.

If antibiotics are indicated, selection should be tailored by available resources and coverage of likely contaminating organisms. Many single-agent broad-spectrum oral antibiotics are available. If any sign of infection develops in a wound, closed or open, including pain, discharge, erythema, edema, or fever, antibiotics should be administered. In an infected closed wound, the adhesive or sutures should be removed and the wound

TABLE 20-4. Differential Diagnostic Features of Abdominal Pain

DISEASE	LOCATION OF PAIN AND PRIOR ATTACKS	MODE OF ONSET AND TYPE OF PAIN	ASSOCIATED GASTROINTESTINAL PROBLEMS	PHYSICAL EXAMINATION
Acute appendicitis	Periumbilical or localized generally to right lower abdominal quadrant	Insidious to acute and persistent	Anorexia common; nausea and vomiting in some	Low-grade fever; epigastric tenderness initially; later, right lower quadrant
Intestinal obstruction	Diffuse	Sudden; crampy	Vomiting common	Abdominal distention; high-pitched rushes
Perforated duodenal ulcer	Epigastric; history of ulcer in many	Abrupt; steady	Anorexia; nausea and vomiting	Epigastric tenderness; involuntary guarding
Diverticulitis	Left lower quadrant; history of previous attacks	Gradual; steady or crampy	Diarrhea common	Fever common; mass and tenderness in left lower quadrant
Acute cholecystitis	Epigastric or right upper quadrant; may be referred to right shoulder	Insidious to acute	Anorexia; nausea and vomiting	Right upper quadrant pain
Renal colic	Costovertebral or along course of ureter	Sudden; severe and sharp	Frequently nausea and vomiting	Flank tenderness
Acute pancreatitis	Epigastric penetrating to back	Acute; persistent, dull, severe	Anorexia; nausea and vomiting common	Epigastric tenderness
Acute salpingitis	Bilateral adnexal; later, may be generalized	Gradually becomes worse	Nausea and vomiting may be present	Cervical motion elicits tenderness; mass if tubo-ovarian abscess is present
Ectopic pregnancy	Unilateral early; may have shoulder pain after rupture	Sudden or intermittently vague to sharp	Frequently none	Adnexal mass; tenderness

irrigated. Wet-to-dry dressings with normal saline should be started and the wound closely observed. Elevation and splinting may assist in relieving pain.

Management of Animal Attacks and Bite Wounds

See Chapters 51 to 53.

WILDERNESS SURGICAL EMERGENCIES

The Acute Abdomen

In the wilderness, the critical distinction between the surgical and nonsurgical abdomen determines whether the victim should be evacuated. A myriad of nonsurgical conditions mimic a surgical abdomen. It has been extensively reported that the bite of the black widow spider *Latrodectus mactans* can induce abdominal pain indistinguishable from a surgical acute abdomen,[51] and mushroom ingestion can cause severe gastroenteritis.[85] Pain, anorexia, nausea, vomiting, and fever are characteristic manifestations of an acute abdominal disorder. Tenderness and guarding are the hallmarks of peritoneal irritation and suggest that operation is indicated.

The approach to someone with abdominal pain begins with a detailed history that includes age, sex, systemic symptoms, and past medical history. This information provides a framework for more detailed questioning about the character of the pain, its mode and time of onset, severity, and precipitating and palliating factors.

In persons 15 to 40 years of age, women are more likely to have abdominal pain, but men have a higher incidence of surgical disease. Common genitourinary causes for abdominal pain in men include epididymitis, renal colic, urinary retention, and testicular torsion. Common causes in women include pelvic inflammatory disease (PID), urinary tract infection, dysmenorrhea, ruptured ovarian cyst, and ectopic pregnancy.

Pain is the hallmark of a surgical abdomen (Table 20-4). It can be characterized by nature of onset, severity, location, and precipitating factors. The onset of abdominal pain can be explosive, rapid, or gradual. The person who is suddenly seized with explosive, agonizing pain is most likely to have rupture of a hollow viscus into the free peritoneal cavity. Colic of renal or biliary origin may also be sudden in onset, but seldom causes pain severe enough to prostrate the victim. If someone has rapid onset of pain that quickly worsens, acute pancreatitis, mesenteric thrombosis, or small bowel strangulation should be suspected. The person with gradual onset of pain is likely to have peritoneal inflammation, such as that accompanying appendicitis or diverticulitis.

Severity of the pain may be characterized as excruciating, severe, dull, or colicky. Excruciating pain unresponsive to narcotics suggests an acute vascular lesion, such as rupture of an abdominal aneurysm or intestinal infarction. Both conditions are unusual in the wilderness environment. Severe pain readily controlled by medication is characteristic of peritonitis from a ruptured viscus or acute pancreatitis. Dull, vague, and poorly localized pain suggests an inflammatory process and is a common initial presentation of appendicitis. Colicky pain characterized as cramps and rushes is suggestive of gastroenteritis. The pain from mechanical small bowel obstruction is also colicky but has a rhythmic pattern, with pain-free intervals alternating with severe colic. The peristaltic rushes associated with gastroenteritis are not necessarily coordinated with colicky pain.

Physical examination of the abdomen is initiated by inspection. Valuable clues to the underlying condition that may be obtained in this manner include stigmata of cirrhosis, distention, hyperperistalsis, or incarcerated hernia. The victim may not be able lie still, which is indicative of renal or biliary colic. Persons lying perfectly still frequently have peritoneal inflammation. Auscultation is helpful if the classic "rushes and tinkles" of small bowel obstruction are present. Palpation in the wilderness setting is most helpful in documenting the presence or absence of peritoneal signs. Shake or percussive tenderness, particularly in the context of fever, nausea, and or vomiting, indicates a need for evacuation. Rectal and vaginal examinations should be performed as dictated by the clinical presentation and setting.

General Field Treatment Principles

When evaluating someone with a possible emergent surgical condition in the wilderness, correctly identifying the etiology of a given condition is less important than identifying peritoneal inflammation and the need for operation.

When dealing with a surgical abdomen or other condition requiring evacuation, adjuncts to definitive hospital treatment can be initiated in the field.

Dehydration and intravascular volume depletion accompany many surgical conditions, particularly when the disease process has progressed and evacuation is delayed. Crystalloid resuscitation in the field is beneficial for patients who have developed or have the potential to develop septic or hypovolemic shock. Dehydration is common in the wilderness, and frank hypovolemia can result from vomiting associated with gastroenteritis, appendicitis, renal colic, and small bowel obstruction. Perforated viscus, pancreatitis, cholecystitis, small bowel obstruction, PID, and necrotizing soft tissue infections all may feature volume depletion. The goal in the wilderness setting is to recognize the signs of hypovolemia and initiate resuscitation to decrease perioperative morbidity.

Nasogastric tube decompression of the stomach may help alleviate emesis secondary to abdominal pain or obstruction. Large-bore (i.e., 18-fr) catheters are best and can be easily aspirated with a syringe. Placement should be confirmed by aspiration of gastric contents or auscultation of gastric air upon insufflation of the stomach.

Foley catheters are becoming increasingly more available in wilderness first-aid kits. Recording urine output provides an effective estimate of intravascular volume status. Foley catheter placement should never hinder the possibility of ambulatory evacuation.

Appendicitis

Acute appendicitis is the most common cause of a surgical abdomen in persons younger than 30 years of age. Acute appendicitis is really more than a single disease entity. In terms of physical signs and symptoms, appendicitis proceeds from inflammation to obstruction to ischemia to perforation, all within approximately 36 hours. Symptoms reflect the stage of the disease. Unfortunately, the time frame for the progression of clinical events is highly variable.

Differential diagnosis of appendicitis includes gastroenteritis and mesenteric adenitis, the most common inflammatory disorders in adults. The first symptom of gastroenteritis is typically vomiting, which precedes the onset of pain and is often associated with diarrhea; it is rarely associated with localizing signs or muscular spasm. Bowel sounds are usually hyperactive. A rectal examination rarely shows abnormalities in gastroenteritis but frequently does in adults with appendicitis.

Mesenteric adenitis is often preceded by an upper respiratory infection and is associated with vague abdominal discomfort that often begins in the right lower quadrant. Abdominal examination reveals only mild right lower quadrant tenderness that is often poorly localized.

The incidence of PID in young women with abdominal pain confounds the diagnosis of appendicitis. Some clinicians have documented a relationship between menses and onset of pain. If abdominal pain occurs within 7 days of menses, the incidence of PID is twice that of appendicitis. If onset of pain occurs greater than 8 days from menses, appendicitis is twice as likely as PID. This history with a pelvic examination may enable the examiner to differentiate between the two entities.

Acute appendicitis mandates evacuation because untreated perforation is associated with significant mortality. Broad-spectrum antibiotics (if IV capability, cefotetan [Cefotan] 2 g IV q12h, or as an alternative, piperacillin/tazobactam [Zosyn] 3.375 g IV q6h; if only oral antibiotic capabilities, a fluoroquinolone, such as ciprofloxacin [Cipro] 750 mg PO bid) should be initiated, attempting coverage against gram-negative and anaerobic organisms. IV crystalloid resuscitation should be initiated, particularly if the victim is older or perforation is suspected, and the victim should be placed at bowel rest.

Acute Cholecystitis and Biliary Colic

Biliary colic refers to pain induced by obstruction of the cystic duct, usually by gallstones. The label of this condition as "colic" is a misnomer because the pain is usually constant. The condition is rarely seen before adulthood and is several times more common in women than in men. A sufferer often relates a past history of gallstones and previous episodes of similar pain, which is described as constant right upper quadrant or epigastric pain, radiating to the right scapula and back. Onset ranges from insidious to acute and frequently follows a meal. The episode usually lasts 15 minutes to 1 hour and then abates.[67] If pain is severe, nausea and vomiting may be present (60% to 70%).

Acute cholecystitis is an infection of the gallbladder secondary to cystic duct obstruction, usually from gallstones. In the wilderness setting, it is useful to think of these interrelated conditions as a continuum of one unified disease process. Both biliary colic and cholecystitis present with right upper quadrant pain; however, biliary colic has the potential to be self-limiting and may not require evacuation. Not all persons with biliary colic develop cholecystitis, and signs of infection should be excluded.

Cholecystitis symptoms typically escalate in severity. Pain that persists more than 1 to 2 hours is suspect for cholecystitis, particularly when accompanied by fever, more significant nausea and vomiting, and right upper quadrant tenderness. Studies have shown that fever may be an unreliable predictor of severity of infection.[38] Acute cholecystitis mandates hospitalization, and evacuation plans should be instituted. The disease can progress to gangrenous changes in the gallbladder wall, leading to perforation and death if untreated.

The definitive treatment of cholecystitis is cholecystectomy. In the field, IV antibiotics (ampicillin/sulbactam [Unasyn] 3 g IV q6h) or an oral alternative (ciprofloxacin [Cipro] 750 mg PO

bid) should be given, directed at common biliary organisms, including *Escherichia coli* and *Klebsiella, Bacteroides, Enterobacter, Streptococcus,* and *Proteus* species. Oral antibiotics with optimal bioavailability should be initiated in the absence of parenteral forms, ensuring the broadest spectrum of coverage available. As with most abdominal infections, IV hydration should be started in the field. If significant nausea and vomiting are present, NG decompression may improve comfort and prevent aspiration.

Peptic Ulcer Disease

The incidence of peptic ulcer disease (PUD) is decreasing in the United States. With the advent of treatment of *Helicobacter pylori* infections and the variety of acid-reducing agents available, PUD is now rarely seen by surgeons. The exception is perforation of a gastric or duodenal ulcer, which should be considered in the differential diagnosis of acute abdomen in the wilderness. Victims frequently relate a history of PUD and need for medication. This history, in combination with acute onset of unrelenting epigastric pain radiating to the back, is suspect for perforation of an ulcer.

The physical examination greatly assists in differentiation between simple ulcer disease symptoms and perforation. Pain may be severe. Gastric secretions are caustic to the peritoneum and, as a result, the abdomen frequently displays a rigid, board-like character with associated diffuse peritoneal signs.

History and examination consistent with gastric or duodenal perforation mandate evacuation. Dehydration may be significant. IV resuscitation should be started and the victim placed on bowel rest.

Diverticulitis

Diverticulitis is localized infection of a colonic diverticulum. Impacted material in the diverticulum, usually feces, leads to a localized inflammatory process that can lead to abscess formation and perforation. Diverticulitis presents over a wide range of severity, from mild, localized infection to intra-abdominal catastrophe. It is more common in middle age; one third of the population older than 45 years of age has diverticula, 20% of whom will develop diverticulitis.[89]

Victims often relate a history of previous attacks. Pain is typically described as gradual in onset and localized in the left lower quadrant of the abdomen, although right-sided diverticulitis can occur. Diarrhea and fever are frequently associated complaints. Examination findings range from mild left lower quadrant abdominal tenderness to frank peritonitis, depending on the severity of the underlying infection.

Treatment in the wilderness setting consists of hydration, bowel rest, antibiotics, and evacuation. If evacuation is impossible or significantly delayed, oral broad-spectrum antibiotics may be effective. Mild cases of diverticulitis are frequently treated on an outpatient basis with broad-spectrum oral antibiotics,[83] such as ciprofloxacin 750 mg PO bid. Antibiotic therapy is directed primarily at gram-negative aerobic and anaerobic bacteria, and single-agent therapy covering these organisms has been demonstrated to be as effective as multiple-agent regimens.[50] Because of unpredictability of response to antibiotic treatment, evacuation is indicated.

Mechanical Small Bowel Obstruction

Small bowel obstruction is a true emergency in the wilderness. When the obstruction is complete, expedient surgery is the only treatment. Small bowel obstruction in the United States is almost invariably the result of adhesions from previous laparotomy or incarceration of abdominal hernias, and the history often makes the diagnosis. Victims complain of sudden onset of diffuse, crampy abdominal pain associated with vomiting and obstipation. With progression of the process to strangulation and infarction of bowel, fever and tachycardia develop.

Late physical examination findings reveal a distended, tympanitic abdomen. Although variable, high-pitched tinkling bowel sounds suggest obstruction. A thorough inspection for hernias should be performed.

Progression of examination findings to frank peritonitis is alarming and suggests ischemic bowel. The adage, "Don't let the sun set on a small bowel obstruction," is sound advice in the wilderness setting. All persons suspected of having a small bowel obstruction should be evacuated immediately. In the interim, the stomach should be decompressed with an NG tube to relieve vomiting and abdominal distention, and aggressive IV hydration should be started.

Incarcerated Abdominal Wall Hernias

Abdominal wall hernias are common; groin herniorrhaphy is the most common major general surgical operation performed in the United States.[63] Hernias can become incarcerated or strangulated, which constitutes a surgical emergency. Seventy-five percent of hernias occur in the groin[86]; the majority of incarcerated hernias presenting in the wilderness setting are inguinal hernias. Other common hernias with the capacity for incarceration are incisional and umbilical hernias.

Many people live with bulging asymptomatic hernias. Others manually reduce symptomatic hernias. New painful hernias or known hernias that can no longer be reduced are concerning. The pain of inguinal hernias is usually intermittent. A description of constant pain is suspect for incarceration. Associated symptoms of fever, tachycardia, nausea, and vomiting are indicative of possible incarceration or strangulation.

On physical examination, a mass should be sought along the course of the spermatic cord. Masses may present from the external inguinal ring to the scrotum. The differential diagnosis for painful inguinal or scrotal masses includes lymphadenopathy, testicular torsion, and epididymitis. Associated tenderness of the spermatic cord may be present. A painful mass at the umbilicus, a previous incision site, or below the inguinal ring could represent an incarcerated umbilical, incisional, or femoral hernia, respectively.

Bowel within an incarcerated hernia sac can become gangrenous in as little as 4 to 5 hours[63]; therefore, it is important to determine the time of incarceration. The decision to evacuate is influenced by the presence of contraindications to manual reduction (see later), which are essentially physical signs that suggest progression to strangulation.

Femoral hernias should not be reduced. The danger is en masse reduction of compromised or gangrenous bowel. Contraindications to reduction include associated signs of toxicity, such as fever, tachycardia, or evidence of bowel obstruction in association with a painful, irreducible mass. If the hernia itself is exquisitely tender to palpation and the overlying skin is erythematous and warm, the hernia should not be reduced. Victims with incarcerated hernias with signs of systemic or local toxicity should be evacuated. Similarly, victims with irreducible hernias—which require emergent surgery—should be evacuated.

A newly incarcerated hernia without contraindications to reduction may be reduced by personnel experienced in such techniques. Gentle pressure is exerted on the hernia mass toward the inguinal ring, optimally with the victim flat and hips elevated. Analgesia and sedation may aid in reduction. Gangrenous bowel can rarely be reduced by this method.[93] If successful, the victim should be closely observed for signs of recurrence or abdominal pain.

Urologic Emergencies

Renal colic describes a symptom complex resulting from acute obstruction of the urinary tract secondary to calculus formation. The goal of the wilderness physician is to recognize the symptom complex and institute treatment. After obstruction of the urinary tract, the pain crescendo of renal colic begins in the flank. The pain progresses anteriorly over the abdomen and radiates to the groin and testes in men and the labia in women. Because the autonomic nervous system transmits visceral pain, many abdominal complaints may manifest. Nausea and vomiting are common. With severe colic, the victim writhes in pain and is unable to find a comfortable position.

Physical findings are less revealing than is the review of systems. Tenderness to deep palpation of the region of obstruction or to percussion of the flank is present. Diagnosis in the wilderness is assisted by the presence of gross hematuria.

Management of ureteral colic is pain control. Although almost universally deployed, forced diuresis may reduce ureteral peristalsis. Thus, forced oral fluids or aggressive IV hydration are of questionable benefit.[100] The majority of calculi pass spontaneously in 4 to 6 hours. The goal of management is to control pain until passage of the stone has occurred. A number of pharmacologic approaches may be used. Nonsteroidal anti-inflammatory drugs, such as ibuprofen and ketorolac, have been shown to be effective in the management of renal colic.[100] For symptoms uncontrolled by anti-inflammatory agents, narcotics may be added. Narcotic analgesics are most effective given parenterally; however, agents such as meperidine (Demerol), codeine, and hydromorphone (Dilaudid) may be given orally. Anti-inflammatory agents and analgesics can be combined. An antiemetic may be added to relieve nausea. When administering pain medication in the field, particular attention should be given to airway maintenance and induced nausea and vomiting.

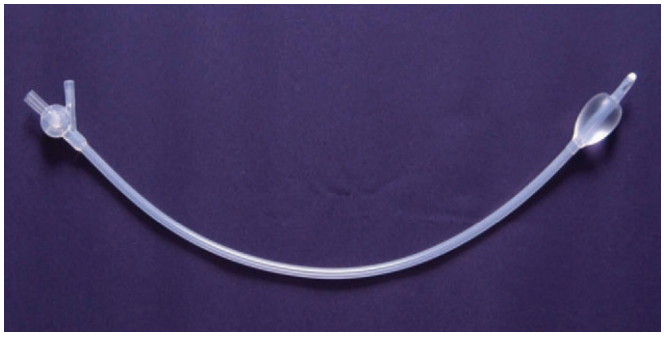
Any person whose symptom complex cannot be controlled must be evacuated. Additional indications for evacuation include calculus anuria and evidence of obstruction-induced infection.

Urinary Retention

Urinary retention is a painful experience that requires immediate medical, and often surgical, intervention.[13] The etiology of urinary retention ranges from prostatism[48] in men to atonic bladder in women. In general, causes have been broadly divided into four groups: obstructive, neurologic, pharmacologic, and psychogenic.[99] Twenty-five percent of men reaching 80 years of age will experience acute retention,[13] which has been shown to increase prostate surgery perioperative mortality rates.[76] Acute urinary retention can lead to incapacitating symptoms in the wilderness; prompt recognition and intervention are necessary.

Principal symptoms are bladder distention and pain that may mimic acute abdomen, overflow incontinence, dribbling, and hesitancy. Physical examination findings include prostatic enlargement in men and lower midline abdominal tenderness and distention. If painful distention of the bladder is present, decompression should be undertaken.

A



B

Figure 20-10. **A,** OPTION-*vf* (female) catheter. **B,** OPTION-*vm* (male) catheter. (Courtesy Opticon Medical, Dublin, OH)

Bladder decompression should be initially attempted with a standard Foley catheter. In men with prostatic hypertrophy, passage of the catheter may be challenging, and a large catheter or coudé catheter should be used if a standard Foley catheter cannot be passed. Instrumentation of the urethra with hemostats or dilators is dangerous and should not be attempted in the field.

Recently introduced are the OPTION-*vf* (female; Fig. 20-10A) and OPTION-*vm* (male; see Fig. 20-10B) (Option Medical, Dublin, Ohio), which are valved urinary catheters that eliminate the need for urine drainage bags and connecting tubes normally required with Foley catheters. These catheters incorporate a manually activated valve at the end of the catheter that allows the patient to store urine in the bladder and to mimic normal voiding behavior. The catheters may be used with a continuous drainage adapter when appropriate, so that a bag may be placed and urination rate and volume assessed.

If multiple attempts are unsuccessful and symptoms are severe, needle decompression is indicated. The skin of the suprapubic region should be anesthetized, if possible. The distended bladder is palpated to guide aspiration. A 22-gauge needle attached to a syringe is introduced through the skin of the lower abdomen two finger breadths above the pubic symphysis and directed at the anus. The needle is advanced with simultaneous aspiration of the syringe until free-flowing urine is visualized. Palpation of the bladder in combination with adherence to this technique should lead to successful decompression.

Complications related to decompression can occur.[84] Drainage of greater than 300 mL/hr can induce mucosal hemorrhage. In addition, 10% of victims develop postobstructive

diuresis that may lead to dehydration, in which case aggressive oral hydration or crystalloid repletion should be undertaken. Finally, it must be recognized that surgical decompression is temporizing and retention will recur. Treatment may need to be continued or repeated. Drainage of the bladder acutely relieves symptoms and may allow ambulatory evacuation, but the underlying etiology must be addressed in a medical facility.

The Acute Scrotum

Acute onset of scrotal pain and swelling requires immediate attention. Etiologies are multiple, but incarcerated hernia and testicular torsion are the most clinically significant in the field. Although any one aspect of the history and physical examination may not be diagnostic, when taken as a whole, they frequently suggest the etiology of the scrotal pathology.[49]

Testicular torsion can occur at any age, but it is more likely near puberty. The likelihood of testicular salvage is inversely proportional to elapsed time from torsion; this is a true surgical emergency. Acute onset of severe testicular pain is the hallmark. Mild to moderate pain is more suggestive of torsion of a testicular appendage or epididymitis. It has been stated that victims who can ambulate with minimal pain are less likely to have testicular torsion. In addition, nausea and vomiting may accompany torsion, whereas fever, dysuria, and frequency are associated with epididymitis.

Physical examination reveals a patient in extreme discomfort with a swollen scrotum and a tender testicle; the affected testicle may be higher than normal and have a horizontal lie.[49] Scrotal skin may be edematous and discolored. Unilateral scrotal swelling without skin changes is more indicative of a hernia or hydrocele. In testicular torsion, the affected testis is often larger than the unaffected side. Testicular torsion can be somewhat differentiated from acute epididymitis by Prehn's sign,[88] which is relief of pain accomplished by elevation of the testicle. Because torsion twists the spermatic cord and elevates the testicle, pain is not relieved by elevation (negative Prehn's sign). Conversely, pain is relieved in epididymitis with elevation (positive Prehn's sign). This maneuver has low sensitivity in distinguishing the two conditions, but may be helpful in conjunction with other findings.[88]

Treatment consists of surgical detorsion, which should be accomplished within 12 hours of torsion.[49] Manual detorsion is not the treatment of choice; however, remoteness of the wilderness environment may mandate manual attempts. Studies of manual detorsion are scant and the cohorts small.[41]

If manual detorsion is necessitated, the victim should be placed supine. Because testicular torsion typically occurs in the medial direction, detorsion is initially attempted with outward rotation of the testis (toward the ipsilateral thigh). Simultaneous rotation in the caudal to cranial direction may be necessary to release the cremasteric muscle.[31]

The surgical treatment of testicular torsion includes pexis of the testis to prevent recurrent torsion. Thus, although detorsion may temporize an acute situation in the field, all victims must be evacuated for definitive treatment.

Prostatitis

Fifty percent of men experience prostatic symptoms in their adult life.[77] A number of forms of prostatitis have been defined, including viral, bacterial (5%), nonbacterial (65%), and chronic forms, as well as prostatodynia.[79] The acute bacterial form may potentially lead to severe infection.

Bacterial prostatitis is an infection of the prostate caused primarily by gram-negative bacteria, with 80% attributable to *E. coli*. It is an acute, febrile illness characterized by perineal pain radiating to the low back, chills, malaise, and voiding symptoms, such as urgency, frequency, and dysuria. Urinary retention is common, and cystitis frequently accompanies the infection. On rectal examination, the prostate gland is usually boggy, warm, and tender and enlargement is variable.

In an ideal situation, treatment is individualized to the cause, which may be difficult to discern in the wilderness. The infection may respond to an oral antibiotic such as ciprofloxacin (750 mg PO bid), ampicillin (500 mg PO qid), or trimethoprim (80 mg with sulfamethoxazole 400 mg PO bid). Penetration of prostatic secretions has been shown to be best achieved by trimethoprim/sulfamethoxazole (TMP/SMX). The chosen antibiotic should be administered for 30 days. If retention is present, catheterization or suprapubic aspiration should be undertaken.

Acute bacterial prostatitis can escalate in severity to systemic toxicity. Persons with evidence of systemic toxicity unresponsive to a trial of oral or parenteral antibiotic therapy should be evacuated.

Urinary Tract Infection

Urinary tract infections (UTIs) are extremely common and include episodes of acute cystitis and pyelonephritis occurring in otherwise healthy individuals. These infections predominate in women; approximately 25% to 35% of women 20 to 40 years of age report having had a UTI.[45] Conversely, men between the ages of 15 and 50 years rarely develop a UTI.

Despite the striking difference in prevalence, symptoms are similar between men and women. The symptoms may represent urethritis, cystitis, or an upper UTI; the distinction is often difficult. Common symptoms include frequency, urgency, dysuria, suprapubic pain, flank pain, and hematuria. Flank pain with tenderness to percussion suggests pyelonephritis. On urinalysis, pyuria is nearly invariably present and hematuria may assist in the diagnosis. Definitive diagnosis is based on significant bacteriuria. The leukocyte esterase test has a screening sensitivity of 75% to 96% and a specificity of 94% to 98% in detecting greater than 10 leukocytes per high-power field.[45]

Treatment in the wilderness setting for both men and women should be directed at the most common causative agents, although 50% to 70% of cases resolve spontaneously if untreated. Causative bacteria include *E. coli* (70% to 95%); *Staphylococcus* species (5% to 20%); and, less frequently, *Klebsiella, Proteus,* and *Enterococcus*. Fortunately, oral antibiotics are highly effective. Although resistant *E. coli* strains are being reported, TMP/SMX (Bactrim DS) is an excellent first-line drug. Alternative regimens include nitrofurantoin, a fluoroquinolone, or a third-generation cephalosporin. A 3-day course of therapy has been shown to be more effective than single-dose therapy.[45] For pyelonephritis, a similar antibiotic in a 10- to 14-day course is acceptable initial treatment. Evacuation should be reserved for systemic toxicity unresponsive to oral antibiotics.

Gynecologic Emergencies

See Chapter 88.

Skin and Soft Tissue Infections

Poor hygiene, superficial skin wounds, blisters, dermatologically significant plants, and insect bites contribute to disruption of the skin barrier. Most often seen are superficial pyodermas

that do not extend beyond the level of the skin. These include erysipelas, impetigo, folliculitis, furunculosis, and carbunculosis. The majority of superficial skin infections are self-limited. However, with less than optimal hygiene and limited resources, skin infections may progress to deep soft tissue infections.

Cellulitis

Cellulitis is acute infection of the skin involving subcutaneous tissue. The superficial form of cellulitis, erysipelas, is identified by well-demarcated, warm, and erythematous plaques with raised borders. The face, scalp, hands, and lower extremities are most often affected. Cellulitis is frequently preceded by a superficial wound.

In the wilderness setting, there are ongoing infection risk factors. Members of expeditions may be physically stressed and nutritionally depleted. High altitude is associated with immunosuppression. *Clostridium* species and other pathogens are ubiquitous in the soil, and proper initial wound care may be suboptimal. Under such conditions, cellulitis can progress to abscess formation and tissue necrosis, leading to septicemia. Wounds that develop erythematous, warm, and boggy margins should be treated with antibiotics and closely observed. The margins of erythema should be marked to gauge progression.

Treatment consists of proper wound hygiene and antibiotic therapy. Local wound measures include elevation, application of moist heat every 4 to 6 hours, and immobilization. Antibiotic therapy is directed at common causative pathogens. Group A streptococci and *Staphylococcus aureus* are most commonly implicated. If complications occur and the cellulitis appears to be progressing, a mixed infection is likely.

Because Gram's stain and culture-directed therapy are not possible, the most broad-spectrum antibiotic available should be administered. Parenteral antibiotics are indicated for serious or mixed infections. Penicillin G (1 to 2 million units q2–3h) is recommended, with first-generation cephalosporins as an alternative. Because oral antibiotics may be the only available therapy, they should be initiated early in the field. Suggested agents include erythromycin (for the penicillin-allergic victim); cephalexin; a macrolide, such as clarithromycin; or a fluoroquinolone, such as ciprofloxacin or levofloxacin.

Lymphangitis

Acute lymphangitis is an infectious process involving subcutaneous lymphatic channels. Its recognition in the wilderness setting is important relative to its propensity to follow puncture wounds, hand wounds, infected blisters, and animal bite wounds. Clinical presentation involves linear erythematous streaks that originate in the lymphatic drainage basin of the wound and "point" to the draining nodal group. Causative agents are similar to those common for cellulitis, with the addition of *Pasteurella multocida* from animal bite wounds.

Treatment consists of antibiotics, warm, moist soaks every 4 to 6 hours, and immobilization and elevation of the extremity. Optimal treatment consists of parenteral antibiotics, but as with cellulitis, oral agents should be initiated if parenteral drugs are not available. Broad-spectrum agents with good oral bioavailability and gram-positive coverage are recommended.[87]

Abscess Formation

When untreated, many superficial skin infections convert to abscesses. Development of a raised, fluctuant mass with overlying warmth and erythema should raise suspicion that surgical drainage is necessary. Frequently, the lesion will "point" when purulent fluid has amassed below the dermis and rupture is impending. Warm soaks and observation are recommended by some clinicians, particularly if the presence of drainable pus is uncertain. However, the definitive treatment is drainage. Antibiotics are recommended if significant associated cellulitis is present, but penetration may be poor.

Local anesthesia should be administered before incision. The lesion should be incised in line with tissue planes over the point of maximum fluctuance. The value of cruciate incisions over linear incisions is debatable; the important feature is assurance of adequate drainage to prevent recurrence. If drainage is undertaken, the incision must be large enough to adequately drain the cavity. All purulent material should be evacuated and the cavity copiously irrigated with saline or water.

Packing is unnecessary if continued drainage is ensured. The wound should be covered with a sterile dressing, changed two to three times per day with concurrent irrigation, and closely observed for reaccumulation of purulence.

Necrotizing Infections

Necrotizing skin and soft tissue infections are life-threatening conditions caused by virulent, toxin-producing bacteria. Depth of tissue involvement is variable and may involve skin, fascia, or muscle. The etiology of necrotizing infections is related to breaks in normal cutaneous defenses associated with some form of injury. Although rare, such infections are of importance to the wilderness physician because of the array of documented inciting injuries and the reduction in mortality possible if diagnosis and treatment are rapid.[28] Necrotizing infections have developed after innocuous-appearing injuries, including simple scratches, insect bites, ankle sprains, and sore throats.[37]

The incidence of necrotizing soft tissue infections is unknown, and there is no age or sex predilection. They most commonly occur on the extremities, abdominal wall, and perineum, within 1 week of the inciting event. Common pathogens include *Streptococcus* species, *Staphylococcus, Vibrio* species, *Clostridium, Pseudomonas, Aeromonas, Enterobacter,* and fungi. Many infections become polymicrobial.

The clinical manifestations can be subtle. An area of cellulitis is commonly the first indication of infection. Pain is often excruciating, and fever is usually present. The infection then progresses to spreading erythema, induration, blue-black discoloration, and blister formation. Necrosis of skin, subcutaneous fat, muscle, or fascia follows, depending on the organism involved. Necrotic tissue exudes a foul-smelling, watery "dishwater" fluid. Subcutaneous emphysema may be present, particularly with clostridial infections, although enteric organisms may also produce air in tissues.

Unfortunately, treatment is limited in the wilderness environment. Debridement of infected tissue should be carried out to the greatest degree humanely possible, but further debridement in a hospital setting is invariably necessary. The extent of debridement necessary is often striking.

After debridement, parenteral antibiotics are necessary. Vancomycin and gentamicin are appropriate first-line agents. The most broad-spectrum oral antibiotic available should be initiated, and the victim expeditiously evacuated. Time is of the essence because the infection can be halted only by aggressive surgical intervention.

The references for this chapter can be found on the accompanying DVD-ROM.

Improvisation in the Wilderness

21

Eric A. Weiss and Howard J. Donner

At the heart of wilderness medicine is improvisation, a creative amalgam of formal medical science and common sense problem solving. Defined as "to fabricate out of what is conveniently at hand," improvisation encompasses many variations, is governed by few absolute rights and wrongs, and is limited more often by imagination than by personnel or equipment.

GENERAL GUIDELINES

When working with an improvised system, test the creation on a noninjured person ("work out the bugs") before applying it to a victim. Include materials that lend themselves to improvisation in the wilderness survival kit to enhance efficiency. Creativity is needed when searching for improvisational materials. The victim's gear can provide needed items (e.g., backpacks can be dismantled to obtain foam pads and straps). When possible, practice constructing improvised systems before they are required in an actual rescue.

IMPROVISED AIRWAY MANAGEMENT

Airway obstruction in the semiconscious or unconscious victim is usually caused by relaxation of the oropharyngeal muscles, which allows the tongue to slide back and obstruct the airway. If only one rescuer is present, maintaining a patent airway with the jaw-thrust or chin-lift technique precludes further first-aid management. You can improvise a nasal trumpet type of airway from a Foley catheter, radiator hose, solar shower hose, siphon tubing, or inflation hose from a kayak flotation bag or sport pouch.

Establish a temporary airway by attaching the anterior aspect of the victim's tongue to the lower lip with two safety pins (Fig. 21-1). An alternative to puncturing the lower lip is to pass a string through the safety pins and hold traction on the tongue by securing the other end to the victim's shirt button or jacket zipper.

Surgical Airway (Cricothyrotomy)

Cricothyrotomy—establishment of an opening in the cricothyroid membrane—is indicated to relieve life-threatening upper airway obstruction when a victim cannot be ventilated effectively from the mouth or nose and endotracheal intubation is not feasible. This may occur in a victim with severe laryngeal edema or with trauma to the face and upper larynx. Cricothyrotomy may also be useful when the person's upper airway is obstructed by a foreign body that cannot be extracted by a Heimlich maneuver or direct laryngoscopy.

In the wilderness, you can perform cricothyrotomy by cutting a hole in the thin cricothyroid membrane and placing a hollow object into the trachea to allow ventilation (Box 21-1). Locate the cricothyroid membrane by palpating the victim's neck, beginning at the top. The first and largest prominence felt is the thyroid cartilage ("Adam's apple"); the second prominence (below the thyroid cartilage) is the cricoid cartilage. The small space between these two, noted by a small depression, is the cricothyroid membrane (see Figure 21-4). With the victim lying on his or her back, cleanse the neck around the cricothyroid membrane with an antiseptic if one is readily available. Put on protective gloves. Make a vertical 1-inch (2.54 cm) incision through the skin with a knife over the membrane (go a little bit above and below the membrane) while using the fingers of your other hand to pry the skin edges apart. Anticipate bleeding from the wound. After the skin is cut apart, puncture the membrane by stabbing it with your knife or other sharp, penetrating object (see Figure 21-5A). Stabilize the larynx between the fingers of one hand, and insert the improvised cricothyrotomy tube through the membrane with your other hand (see Figure 21-5B). Secure the object in place with tape.

Complications associated with this procedure include hemorrhage at the insertion site, subcutaneous or mediastinal emphysema resulting from faulty placement of the tube into the subcutaneous tissues rather than into the trachea, and perforation through the posterior wall of the trachea with placement of the tube in the esophagus.

Improvised Barrier for Mouth-to-Mouth Rescue Breathing

A glove can be modified and used as a barrier shield for performing rescue breathing. Cut the middle finger of the glove at its halfway point and insert it into the victim's mouth. Stretch the glove across the victim's mouth and nose and blow into the glove as you would to inflate a balloon. After each breath, remove the part of the glove covering the nose to allow the victim to exhale. The slit creates a one-way valve, preventing backflow of the victim's saliva (see Figure 21-6).

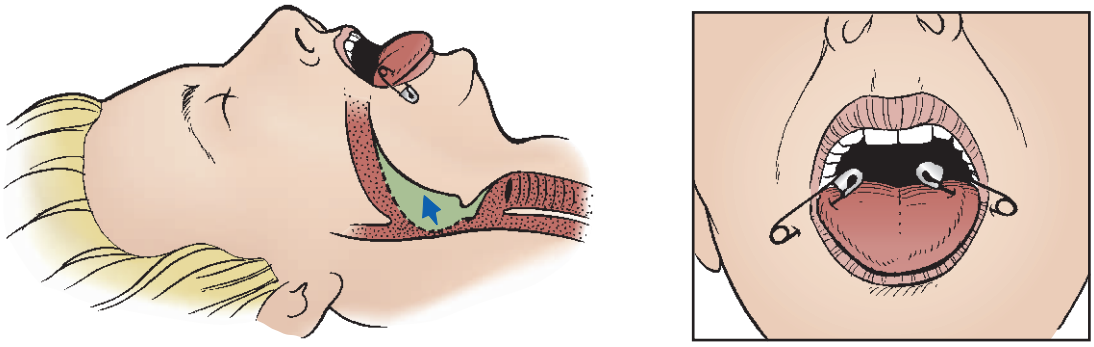


Figure 21-1. Safety pins on tongue to open airway.

Box 21-1. Improvised Cricothyrotomy Tubes

1. **IV administration set drip chamber:** Cut the plastic drip chamber of a macro drip (15 drops/mL) IV administration set at its halfway point with a knife or scissors. Remove the end protector from the piercing spike and insert the spike through the cricothyroid membrane. The plastic drip chamber is nearly the same size as a 15-mm endotracheal tube adapter and fits snugly in the valve fitting of a bag-valve device (Fig. 21-2).
2. **Syringe barrel:** Cut the barrel of a 1- or 3-mL syringe with the plunger removed at a 45-degree angle at its midpoint to create an improvised cricothyroid airway device. The proximal phalange of the syringe barrel helps secure the device to the neck and prevents it from being aspirated (Fig. 21-3).
3. **Any small hollow object:** Examples include a small flashlight or penlight casing, pen casing, small pill bottle, and large-bore needle or IV catheter. Several commercial devices are small and lightweight enough to be included in the first-aid kit.

EAR, NOSE, AND THROAT EMERGENCIES

Epistaxis

Epistaxis is a common problem in travelers. Reduced humidity in airplanes, cold climates, and high-altitude environments can produce drying and erosion of the nasal mucosa. Other etiologic factors include facial trauma, infections, and inflammatory rhinitis. Although most cases of epistaxis are minor, some present life-threatening emergencies (see Chapter 26).[42]

Anterior epistaxis from one side of the nasal cavity occurs in 90% of cases.[11] If pinching the nostrils against the septum for a full 10 minutes does not control the bleeding, nasal packing may be needed. Soak a piece of cotton or gauze with a vasoconstrictor, such as oxymetazoline (Afrin) nasal spray, and insert it into the nose, leaving it in place for 5 to 10 minutes. Petroleum jelly–impregnated gauze or strips of a nonadherent dressing can then be packed into the nose so that both ends of the gauze remain outside the nasal cavity (Fig. 21-7). This prevents the victim from inadvertently aspirating the nasal packing.[42]

Complete packing of the nasal cavity of an adult victim requires a minimum of 1 m (3 feet) of packing to fill the nasal cavity and tamponade the bleeding site.[7] Expandable packing material, such as Weimert Epistaxis Packing, Rapid Rhino (ArthroCare Corporation, Sunnyvale, CA), or the Rhino Rocket (Shippert Medical Technologies, Centennial, CO), is available commercially. A tampon or balloon tip from a Foley catheter can also be used as improvised packing.[42]

Anterior nasal packing blocks sinus drainage and predisposes to sinusitis. Prophylactic antibiotics are usually recommended until the pack is removed in 48 hours.[42]

If the bleeding site is located posteriorly, use a 14- to 16-Fr Foley catheter with a 30-mL balloon to tamponade the site.[14] Prelubricate the catheter with either petroleum jelly (Vaseline) or a water-based lubricant, then insert it through the nasal cavity into the posterior pharynx. Inflate the balloon with 10 to 15 mL of water and gently withdraw it back into the posterior nasopharynx until resistance is met. Secure the catheter firmly to the victim's forehead with several strips of tape. Pack the anterior nose in front of the catheter balloon as described earlier.

Esophageal Foreign Bodies

Esophageal foreign bodies may cause significant morbidity. Respiratory compromise caused by tracheal compression or by aspiration of secretions can occur. Mediastinitis, pleural effusion, pneumothorax, and abscess may be seen with perforations of the esophagus from sharp objects or pressure necrosis caused by large objects.[28]

The use of a Foley balloon-tipped catheter can be a safe method for removing blunt esophageal foreign bodies.[5,6,17] Success rates of 98% have been cited.[5] Associated complications include laryngospasm, epistaxis, pain, esophageal perforation, and tracheal aspiration of the dislodged foreign body.[28] Sharp, ragged foreign bodies or an uncooperative victim precludes use of this technique (Fig. 21-8).[37]

Lubricate a 12- to 16-Fr Foley catheter and place it orally into the esophagus while the victim is seated. After placing the victim in Trendelenburg's position, pass the catheter beyond the foreign body and inflate the balloon with water. Withdraw the catheter with steady traction until the foreign body can be removed from the hypopharynx or is expelled by coughing.

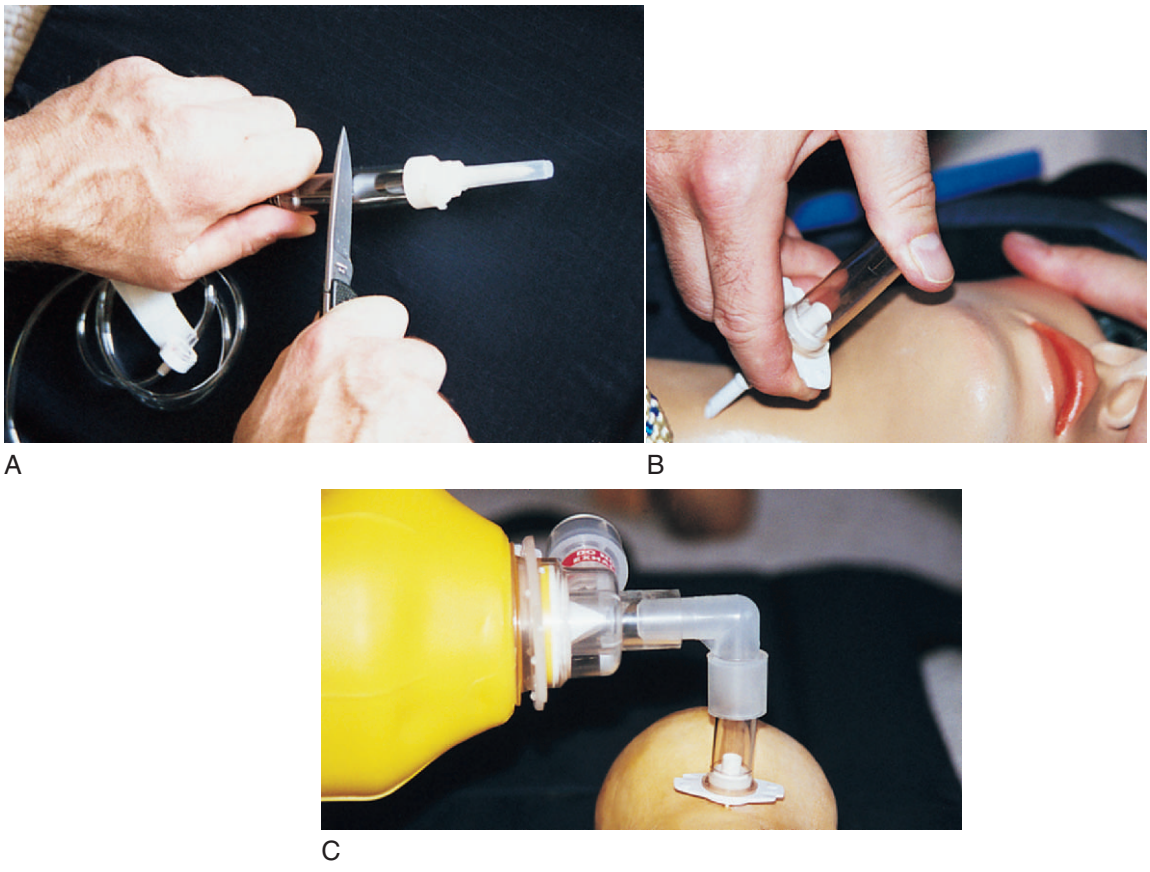


Figure 21-2. Improvised cricothyrotomy tube. **A,** Cut plastic drip chamber at halfway point. **B,** Insert spike from drip chamber into the cricothyroid membrane. **C,** Bag-valve device will fit over the chamber for ventilation.

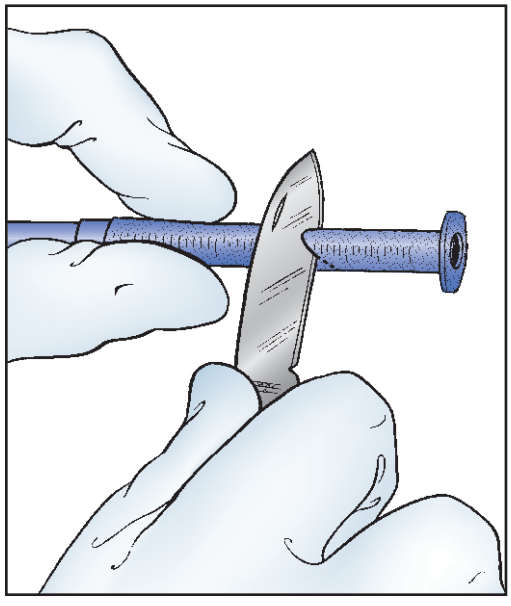


Figure 21-3. Improvised cricothyroid airway device can be created by cutting barrel of syringe at a 45-degree angle at its midway point.

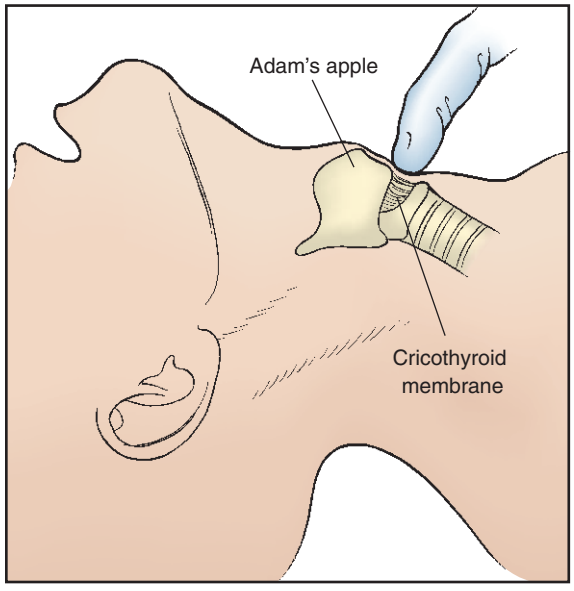


Figure 21-4. Cricothyroid membrane is found in the depression between the Adam's apple (thyroid cartilage) and the cricoid cartilage.

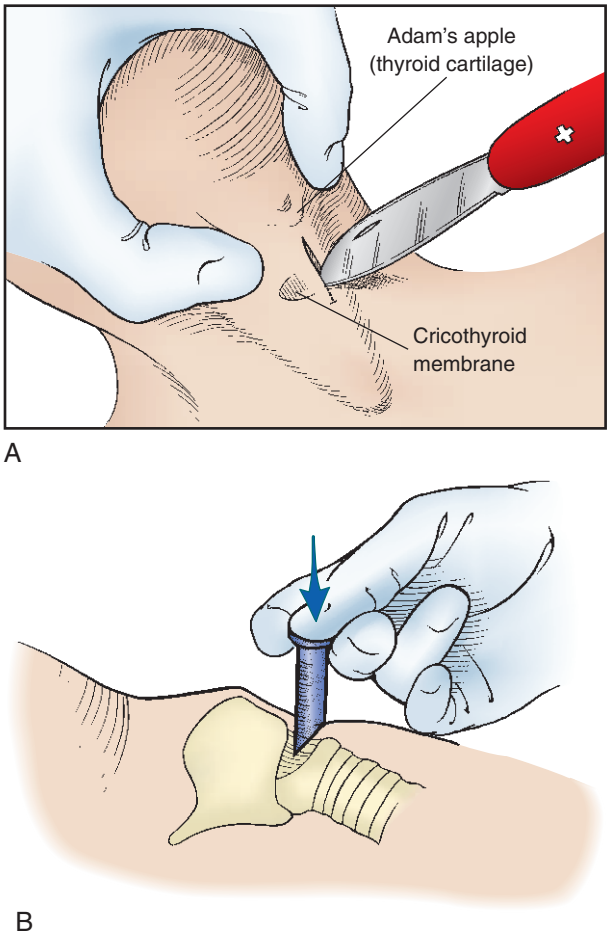


Figure 21-5. Cricothyrotomy. **A,** Locate cricothyroid membrane and make a vertical 1-inch incision through the skin. **B,** Insert pointed end of improvised cricothyrotomy tube through the membrane.

Take care to avoid lodging the foreign body in the nasopharynx. Any significant impedance to withdrawal should terminate the attempt.[37] Use of this technique is recommended only in extreme wilderness settings or when endoscopy is not available.

TENSION PNEUMOTHORAX

Overview

Signs and symptoms of a tension pneumothorax include distended neck veins, tracheal deviation away from the side of the pneumothorax, unilateral absent breath sounds, hyperresonant hemithorax to percussion, subcutaneous emphysema, respiratory distress, cyanosis, and cardiovascular collapse. Tension pneumothorax mandates rapid pleural decompression if the victim appears to be dying. Possible complications of pleural decompression include infection; profound bleeding from puncture of the heart, lung, or a major blood vessel; or even laceration of the liver or spleen.

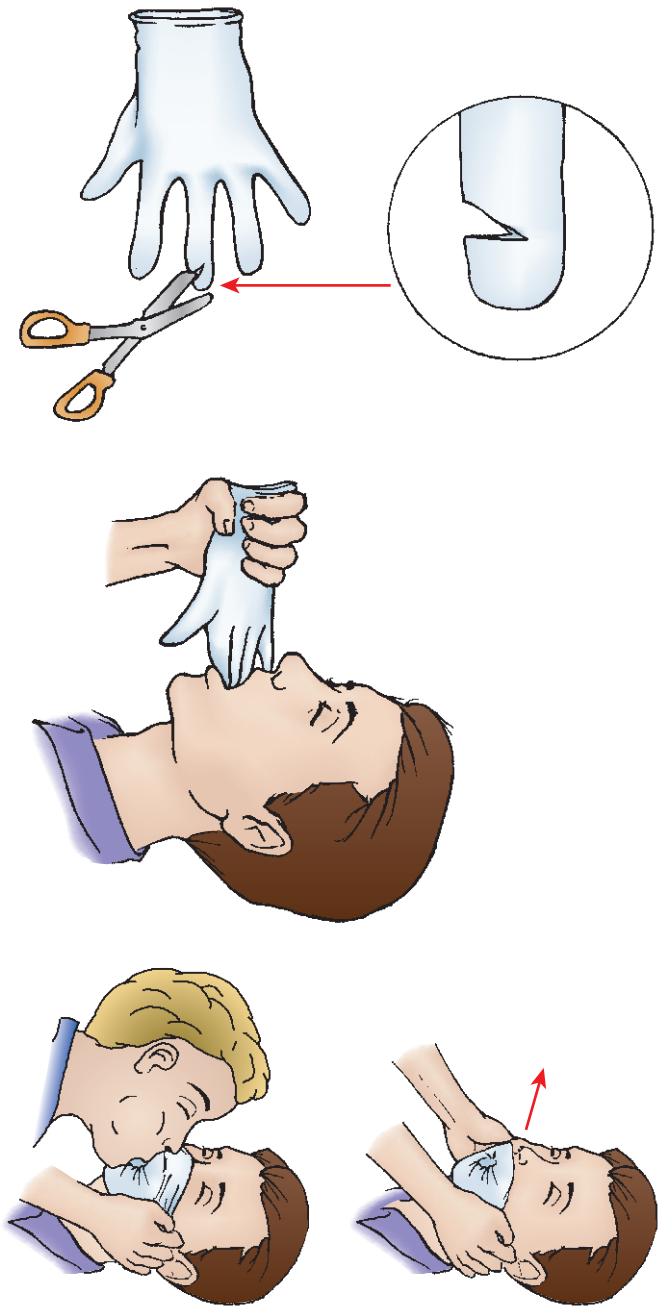


Figure 21-6. Improvised cardiopulmonary resuscitation (CPR) barrier is created using a latex or nitrile glove. Make a slit in the middle finger of the glove.

Improvised Pleural Decompression Technique

Swab the entire chest with povidone-iodine or another antiseptic. If sterile gloves are available, put them on after washing your hands. If local anesthesia is available, infiltrate the puncture site down to the rib and over its upper border.

Insert a large-bore intravenous (IV) catheter, needle, or any pointed, sharp object that is available into the chest just above

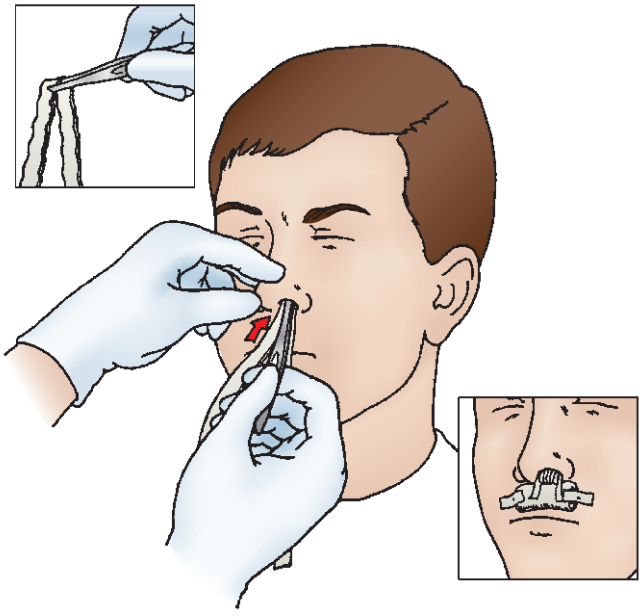


Figure 21-7. Anterior epistaxis from one side of the nasal cavity can be treated using nasal packing soaked in a vasoconstrictor. Petroleum jelly–impregnated gauze or strips of non-adherent dressing can be packed in the nose so that both ends of the gauze remain outside the nasal cavity.

Box 21-2. Improvised Pleural Decompression Devices

1. Large-bore (12- or 14-gauge) IV catheter or needle
2. Endotracheal tube
3. Foley catheter with a rigid support ("stylet"), such as a clothes hanger, placed into the lumen
4. Section of a tent pole
5. Hose from a hydration pouch

the third rib in the midclavicular line (midway between the top of the shoulder and the nipple in a line with the nipple approximates this location) (Box 21-2). If you hit the rib, move the needle or knife upward slightly until it passes over the top of the rib, thus avoiding the intercostal blood vessels that course along the lower edge of every rib. The chest wall is $1^1/_2$ to $2^1/_2$ inches (4–6 cm) thick, depending on the individual's muscularity and amount of fat present. A gush of air signals that you have entered the pleural space; do not push in the penetrating object further. Releasing the tension converts the tension pneumothorax into an open pneumothorax.

Leave the needle or catheter in place, and use a rubber glove to make a flutter valve. Cut out a finger portion from a rubber glove, making a tiny slit cut at the tip. Place this over the external opening to create a unidirectional flutter valve that allows continuous egress of air from the pleural space (Fig. 21-9). To create a one-way flutter valve, cut a finger portion of a latex glove off at the proximal end of the finger and insert the needle

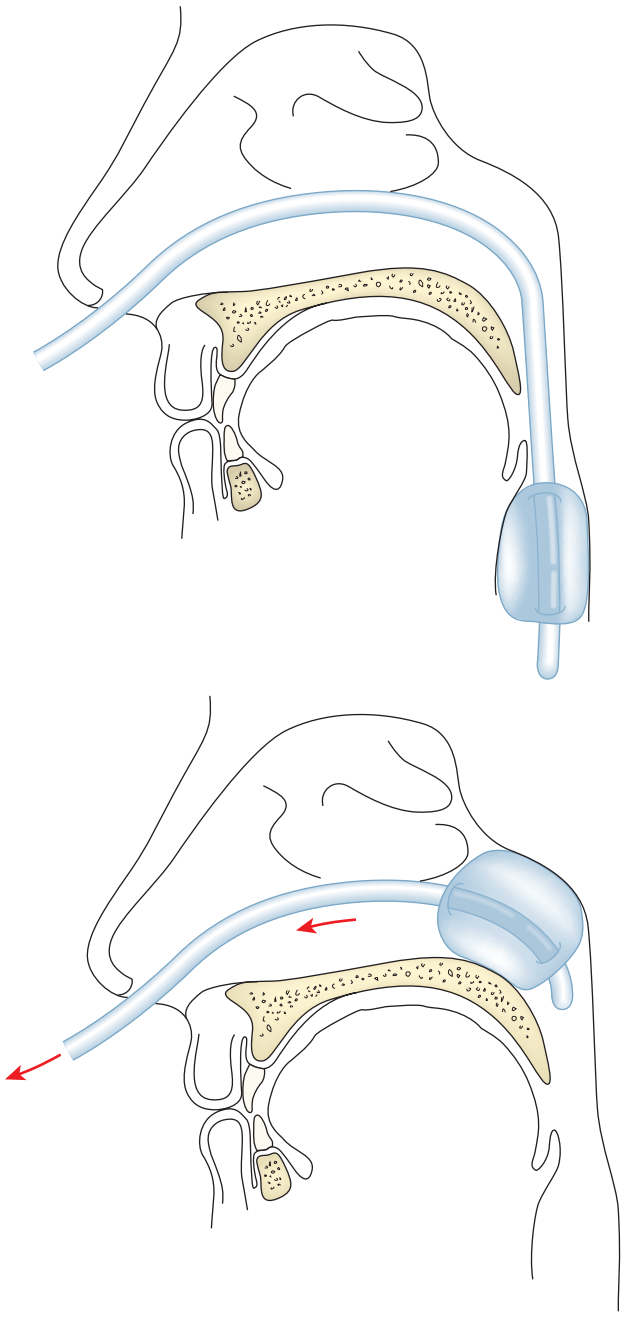


Figure 21-8. Packing the back of the nose. Insert a Foley catheter into the nose and gently pass it back until it enters the back of the throat. After the tip of the catheter is in the victim's throat, carefully inflate the balloon with 10 to 12 mL of air or water from a syringe. Inflation should be done slowly and should be stopped if painful. After the balloon is inflated, gently pull the catheter back out until resistance is met.

or catheter into the open end of the glove finger and through the tip as shown (see Figure 21-9A). The cut-out finger portion of the glove creates a unidirectional flutter valve that allows egress of air from the pleural space during expiration, but collapses to prevent air entry on inspiration (see Figure 21-9B, C).

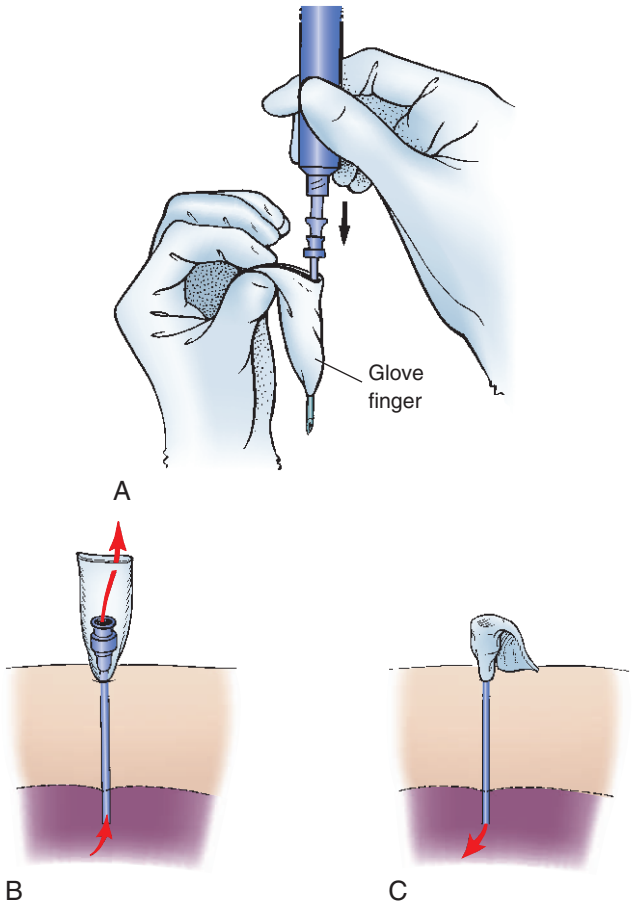


Figure 21-9. **A,** Finger of glove is attached to needle or catheter to create flutter valve. **B,** Flutter valve allows air to escape. **C,** Flutter valve collapses to prevent air entry.

OPEN ("SUCKING") CHEST WOUND

Penetrating trauma to the chest can produce a chest wound that allows air to be sucked into the pleura on inspiration. Place a piece of plastic food wrap, aluminum foil, or one side of a plastic sandwich bag on top of the wound and tape it on three sides. The untaped fourth side serves as a relief valve to prevent formation of a tension pneumothorax.

SPLINTING AND TRACTION*

Cervical Spine Injuries

Because of its mobility, the cervical spine is the spinal column area most commonly injured in trauma. Any obvious or suspected cervical spine injury demands full spinal immobilization with both a rigid or semirigid cervical collar and long-board immobilization. Historically, dogma about cervical spine injuries has specified a "splint 'em as they lie" approach. Transporting a victim who is not in anatomic position is arduous in the backcountry. It is uncomfortable for the victim, difficult for the rescuers, and increases the risk of further injury. In general, gentle axial traction back to anatomic position is indicated unless (1) return to anatomic position significantly increases pain or focal neurologic deficit or (2) movement of the head and neck results in any noticeable mechanical resistance.[24]

All cervical spine injuries (or suspected injuries) warrant full long-board immobilization. Movement of the pelvis and hips laterally is potentially more dangerous than anterior–posterior movement; therefore it is appropriate during extended transport to allow gentle flexion at the hip with immobilization in that position if the victim is more comfortable. Soft pads behind the knees and small of the back also add to the victim's comfort during a long transport.[9]

Improvised Cervical Collars

Cervical collars are always adjuncts to full spinal immobilization; they should never be used alone. The improvised cervical collar is used in conjunction with manual cervical spine stabilization followed by complete immobilization of the victim on a spine board. A properly applied and fitted collar is a primary defense against axial loading of the cervical spine, particularly in an evacuation that involves tilting the victim's body uphill or downhill. Improvised cervical collars have had a bad reputation, and textbooks continue to depict them made from a simple cravat wrapped around the neck. This type of system is no more effective than are the soft cervical collars often used by urban plaintiffs trying to impress a jury (i.e., not effective at all).

An improvised cervical collar works effectively only if it has the following features:

1. It is rigid or semirigid.
2. It fits properly (many improvised designs are too small).
3. It does not choke the victim.
4. It allows the victim's mouth to open if vomiting occurs.

Improvisational approaches to cervical collars are outlined below.

Closed-Cell Foam System. The best closed-cell foam systems incorporate a full-size or three-quarter-length pad folded longitudinally into thirds and applied centered over the back of the victim's neck and wrapped forward. The pad is crossed under the chin, contoured underneath opposite axillae, and secured. If the pad is not long enough, you can tape or tie on extensions. This system also works well with blankets, beach towels, or even a rolled plastic tarp. Avoid small flexible cervical collars that do not optimally extend the chin-to-chest distance.

Padded Hip Belt. A padded hip belt or fanny pack removed from a large internal or external frame backpack can sometimes be modified to work perfectly. Wider is usually better. Take up excess circumference by overlapping the belt, and secure the excess material with duct tape (Fig. 21-10).

Clothing. Bulky clothing, such as a fiber pile or fleece jacket, can be rolled and then wrapped around the victim's neck to make a cervical collar. The extended sleeves can be used to secure the collar. Prewrapping a wide elasticized (Ace) wrap

*Specific aspects of fracture care are covered in detail in other chapters. This chapter focuses on improvised systems, not on definitive orthopedic management. Improvised systems rarely provide the same degree of protection as commercial systems. Good judgment is needed.

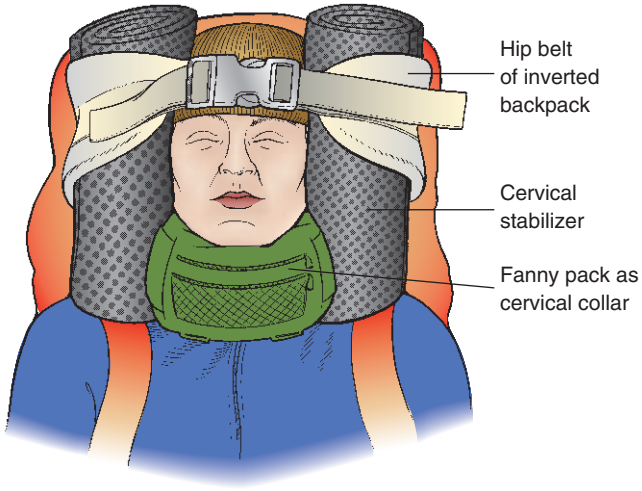


Figure 21-10. Inverted pack used as spine board.

around the jacket compresses the material to make it more rigid and supportive.

Malleable Aluminum Splint. A well-padded, aluminum splint (e.g., SAM Splint) can be adjusted to fit almost any size neck (Fig. 21-11).

Improvised Spinal Immobilization

As noted, the improvised cervical collar is only an adjunct to full spinal immobilization. Two immobilization systems are (1) short-board immobilization, which is useful for short-duration transport (that is, getting the victim out of immediate danger) or when used in conjunction with a long board; and (2) long-board immobilization, used for definitive immobilization during extensive transport.

Use either of these systems in conjunction with a rigid or semirigid cervical collar, as described previously. Improvised lateral "towel rolls" are often added to these systems for additional head and neck support. These rolls can be improvised from small sections of closed-cell foam (e.g., Ensolite [Armacell LLC, Mebane, NC]) sleeping pads. Alternatively, a U-shaped head support, or "horse collar," can be made from any rolled garment, blanket, tarp, or tent fly; this is placed over the victim's head in an inverted U and used with the improvised cervical collar and spine board. Hiking socks or stuff bags filled with dirt, sand, or gravel also work well for this purpose. Stuff bags filled with snow for support should never be used because the snow can melt during transport, which will cause excessive head and neck motion. However, snow-filled stuff bags can act as temporary support while more definitive systems are being constructed.

Improvised Short-Board Immobilization

Internal Frame Pack and Snow Shovel System. Some internal frame backpacks can be easily modified by inserting a snow shovel through the centerline attachment points (the shovel handgrip may need to be removed first). The victim's head is taped to the lightly padded shovel (Fig. 21-12); in this context, the shovel blade serves as a head bed. This system incorporates the remainder of the pack suspension as designed (i.e., shoulder and sternum straps with hip belt) and works well with other long-board designs, such as the continuous loop system (see Continuous Loop System section). Always assess the amount of flexion or extension the system will create on the cervical spine; if it is unacceptable, the system must be modified appropriately.

Inverted Pack System. An efficient short board can be made using an inverted internal or external frame backpack. The padded hip belt provides a head bed, and the frame is used as a short board in conjunction with a rigid or semirigid cervical collar (Fig. 21-13).

Turn the pack upside down, and lash the victim's shoulders and torso to the pack. Fasten the waist belt around the victim's head, as in the top section of a Kendrick extrication device. The hip belt is typically too large, but excess circumference can be eliminated with bilateral Ensolite rolls. Unlike the snow shovel system, this system requires that the victim be lashed to the splint.

Snowshoe System. A snowshoe can be made into a fairly reliable short spine board (Fig. 21-14). Pad the snowshoe and rig it for attachment to the victim as shown.[19]

Improvised Long-Board Immobilization

Continuous Loop System (also known as the daisy chain, cocoon wrap, or mummy litter). In the authors' opinion, this is the only improvised system that is adequate for immobilizing and transporting a patient with potential spine injuries. To construct the continuous loop system, the following items are needed:

1. Long climbing or rescue rope
2. Large tarp (or tent fly)
3. Sleeping pads (Ensolite or Therm-a-Rest [Cascade Designs, Inc., Seattle])
4. Stiffeners (such as skis, poles, snowshoes, canoe paddles, or tree branches)

Lay the rope out with even U-shaped loops as shown in Figure 21-15A. The midsection should be slightly wider to conform to the victim's width. Tie a small loop at the foot end of the rope and place a tarp on the rope loops. On top of the tarp, lay foam pads the full length of the system (the pads can be overlapped to add length). Then, lay stiffeners on top of the pads in the same axis as the victim (see Fig. 21-15B). Add multiple foam pads on top of the stiffeners, followed optionally with a sleeping bag (see Fig. 21-15C). Place the victim on the pads. To form the daisy chain, bring a single loop through the pre-tied loop, pulling loops toward the center, and feeding through the loops brought up from the opposite side. It is important to take up rope slack continuously. When the victim's armpits are reached, bring a loop over each shoulder and tie it off (or clip it off with a carabiner) (see Fig. 21-15D).

One excellent modification involves adding an inverted internal frame backpack. This can be incorporated with the padding and secured with the head end of the rope. The pack adds rigidity and padding, and the padded hip belt serves as a very efficient head and neck immobilizer (see Figures 21-10 and 21-13). If the victim vomits during transport, the entire unit can be turned on its side or even partially inverted.

Backpack Frame Litters. Functional litters can be constructed from external frame backpacks. Traditionally, two frames are

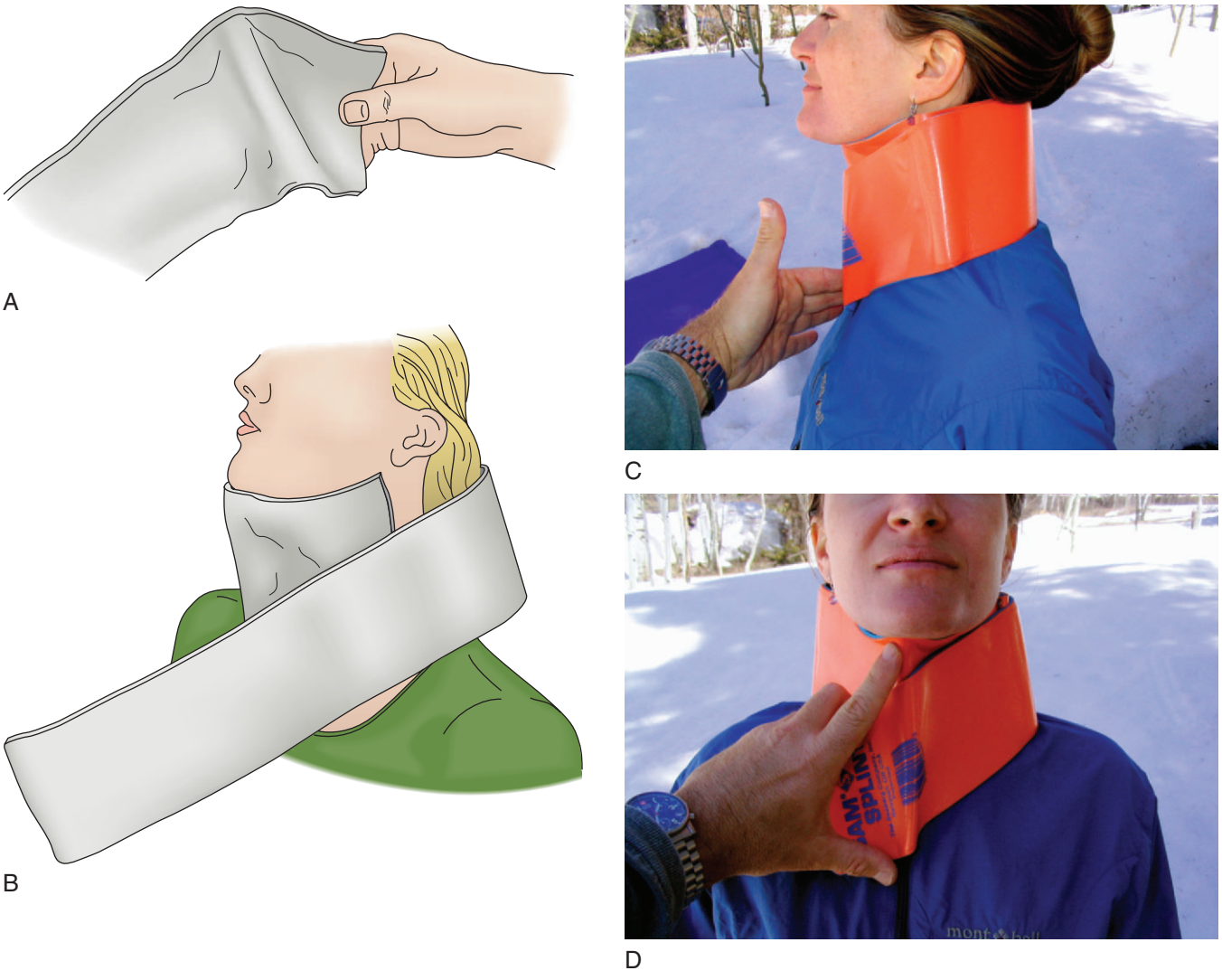


Figure 21-11. Padded aluminum splint cervical collar. **A,** Place a vertical bend in the malleable aluminum splint approximately 6 inches from one end to form a vertical pillar. Then, add bilateral flares to make the splint comfortable for the victim where it rides against the lower mandible. **B,** Place the anterior pillar securely beneath the victim's chin and wrap the remaining length of the splint around the victim's neck. **C,** Side view of cervical collar fashioned from a SAM Splint. Note the formation of lateral, and if possible, posterior pillars. **D,** Frontal view. The end is angled inferiorly to provide an adequate chin-to-chest distance. Note the formation of the anterior pillar.

used, but three or four frames (Fig. 21-16) make for a larger, more stable litter. Cable ties or fiberglass strapping tape simplifies this fabrication. These litters can be reinforced with ice axes or ski poles.

Kayak System. Properly modified, the kayak makes an ideal rigid long-board improvised litter. First, remove the seat along with sections of the upper deck if necessary. A serrated river knife (or camp saw) makes this improvisation much easier. Open deck canoes can be used almost as is once the flotation material has been removed.

Canoe System. Many rivers have railroad tracks that run parallel to the river canyon. The tracks can be used to slide a canoe by placing the boat perpendicular to the tracks and pulling on both bow and stern lines.

Improvised Head Bed. A padded aluminum splint can be formed as shown to create a head bed to assist in securing the immobilized head of an injured victim. Tape this head bed to a commercial or improvised backboard (Fig. 21-17).

Pelvic Fractures

Overview

Unstable pelvic fractures are associated with a high incidence of morbidity and mortality.[10,16,25] In one study, pelvic ring disruption in the trauma patient was associated with a mortality

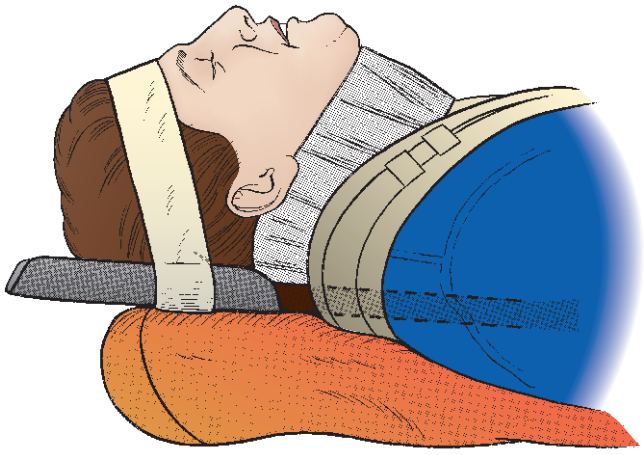


Figure 21-12. Head immobilized on a padded shovel.

Figure 21-13. Short board using an inverted pack system. The backpack waist belt can be seen encircling the head.

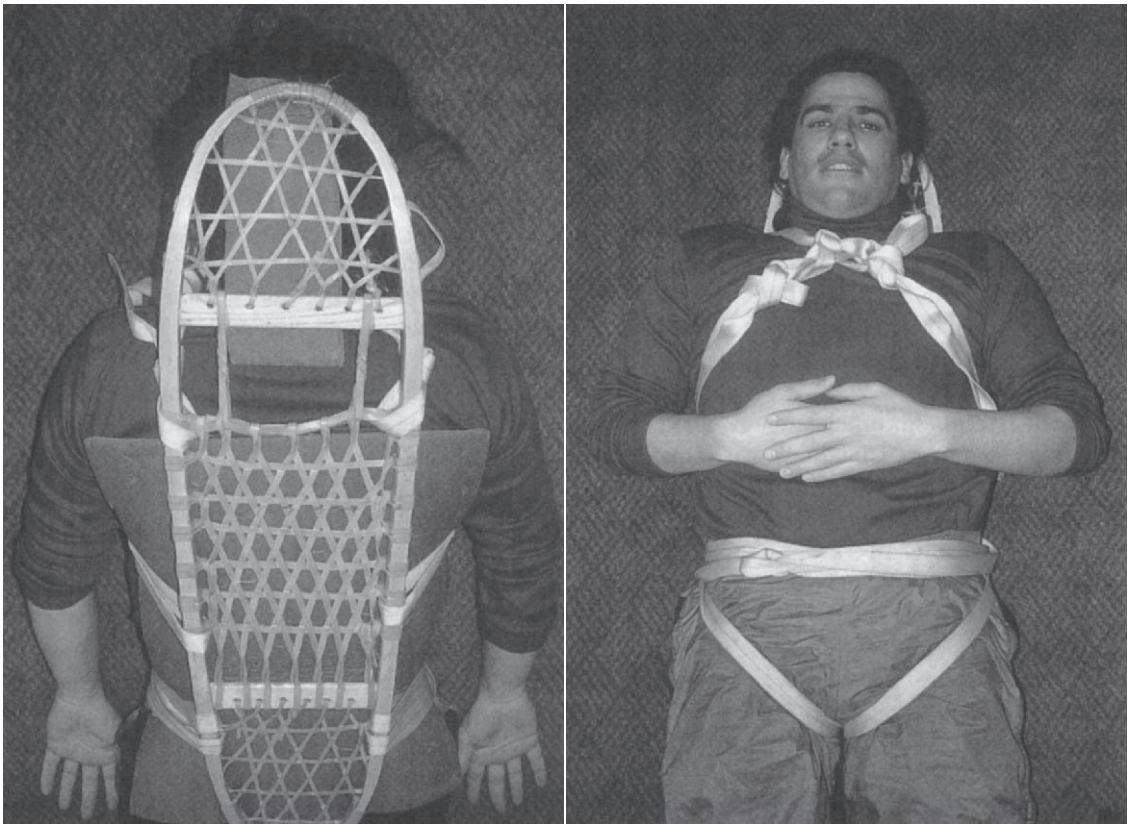


Figure 21-14. Improvised snowshoe short board. A well-padded snowshoe is prerigged with webbing and attached to the victim as shown. This system can also be used in conjunction with long-board systems, such as the continuous loop system.

rate of 25%.[1] Acute and uncontrolled hemorrhage and its complications are the leading causes of death in these patients.[22] Hemorrhage after pelvic injury results from fracture surfaces, venous plexuses, and major arteries, usually branches of the internal iliac artery. Pelvic reduction and stabilization in the early post-traumatic phase are reported to provide the most effective means of controlling venous hemorrhage.[9] Pelvic reduction realigns bleeding fracture surfaces and reduces pelvic volume, and pelvic stabilization facilitates clot formation by reducing fracture site motion.[23]

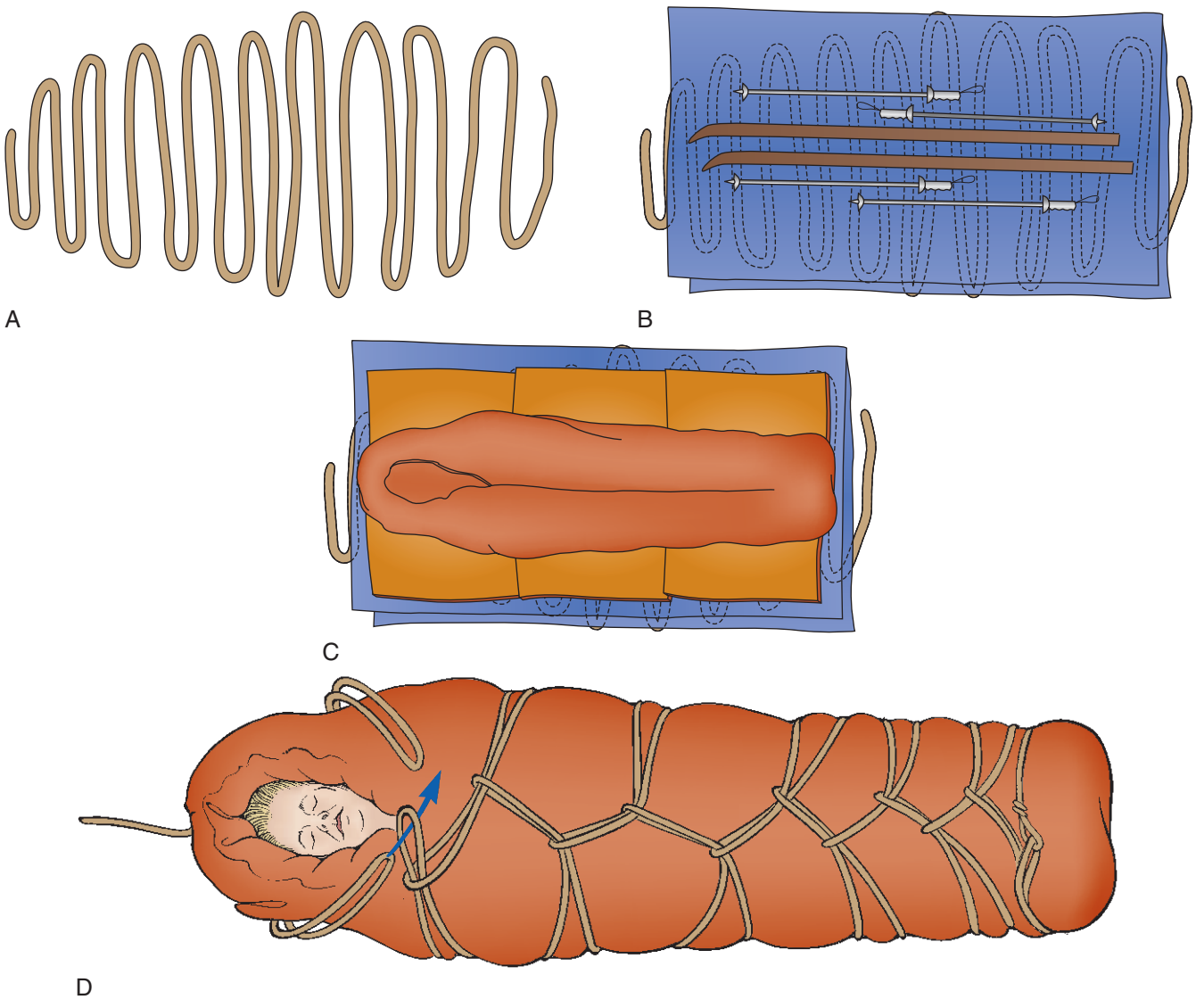


Figure 21-15. Continuous loop, or "mummy," litter made with a climbing rope. **A,** Rope is laid out with even U-shaped loops. **B,** Stiffeners such as skis and poles are placed underneath the victim to add structural rigidity. It is important to pad between the stiffeners and the victim. **C,** A sleeping bag may be used in addition to the foam pads. **D,** Loop of rope is brought over each shoulder and tied off (see text).

Circumferential wrapping and compression of the pelvic region with a sheet or strap has been studied as a means to stabilize and reduce open-book pelvic fractures.[8,10] In a pair of studies from Oregon, circumferential pelvic compression was evaluated with the use of cadaveric models. Circumferential compression was found to result in significant stabilization of open-book pelvic injuries. Optimal placement of compression was at the level of the greater trochanters and the symphysis pubis, while the desired tension was found to be around 180 N (40 lb).[8,10]

Vermeulen and colleagues reported application of pelvic straps to 19 patients in Switzerland.[40] Each strap was applied at the accident scene by emergency medical technicians (EMTs) upon suspicion of an unstable pelvic fracture. It took only 30 seconds to apply each strap. Application was subsequently found to produce significant reduction in both symphysis diastasis and pelvic inlet area. Routt and coworkers used a large circumferential sheet pulled tightly around the pelvis.[33] The sheet remained in place until further stabilization could be achieved with an external fixator. Application of the sheet achieved reduction and stabilization in a safe and time-effective manner and did not interfere with resuscitation efforts. The American College of Surgeons Advanced Trauma Life Support (ATLS) course now includes a protocol for emergent management of pelvic ring disruptions, advising circumferential application of a pelvic sheet.

Circumferential compression with a noninvasive pelvic sling should be considered for any patient with a suspected pelvic

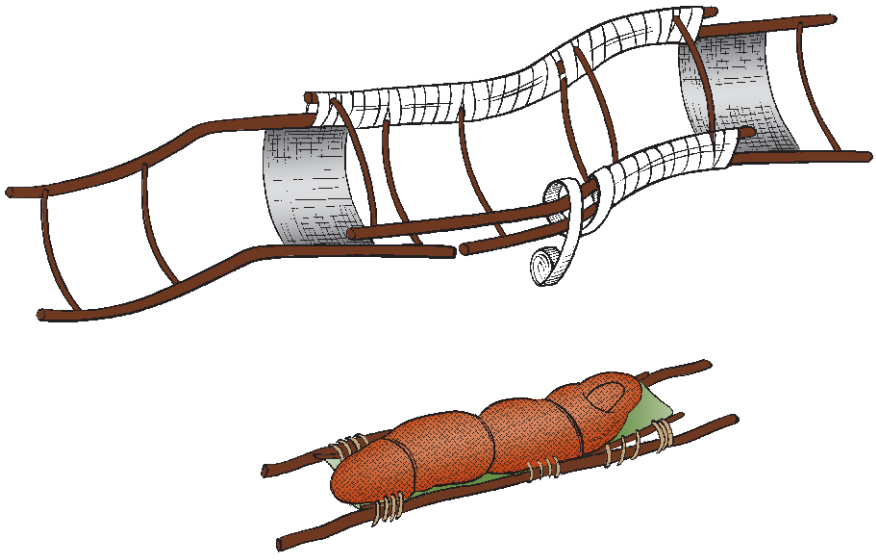


Figure 21-16. Backpack frame litter.

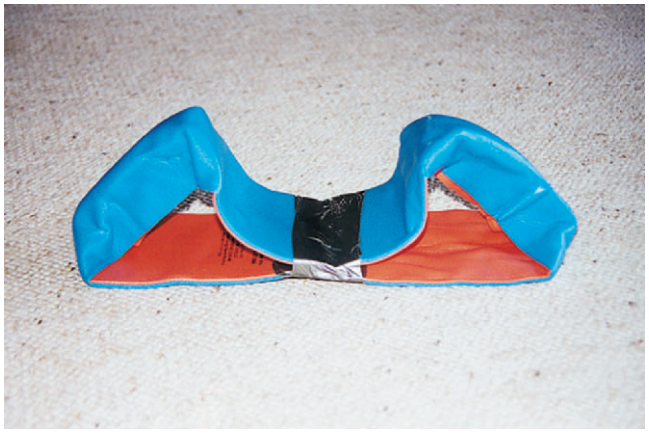


Figure 21-17. Improvised head bed.

Figure 21-18. Pelvic sling improvised with jacket.

fracture in the wilderness. The SAM Sling (SAM Medical Products, Newport, OR), is a commercial device that provides 150 N (33 lb) of pressure to the pelvis when applied according to the manufacturer's instructions. It is equipped with an "autostop" buckle, which comprises spring-loaded prongs that lock the buckle in place when the correct amount of force is applied.

Clothes, sheets, a sleeping bag, pads, air mattress, tent, or tent fly can be used to improvise a very effective pelvic sling in the backcountry. The object should be wide enough so that it does not cut into the victim when tightened.

Applying an Improvised Pelvic Sling

1. Ensure that objects have been removed from the patient's pockets and that any belt has been removed so that the pressure of the sheet or object doesn't cause additional pain by pressing items against the pelvis.
2. Very gently slide the improvised sling under the victim's buttocks and center it under the bony prominences at the outer part of the upper thigh or hips (greater trochanters/symphysis pubis) (Fig. 21-18). Cross the object over the front of the pelvis and tighten the sling by pulling both ends and securing with a knot, clamp, or duct tape. Another tightening technique is to wrap the sling snugly around the pelvis and tie an overhand knot. Place tent poles, a stick, or similar object on the knot and tie another overhand knot. Twist the poles or stick until the sling becomes tight. The sling should be tightened so that it is snug and the pelvis is returned to its normal anatomic position.
3. If a Therm-a-Rest pad or other inflatable sleeping pad is available, fold it in half so that it approximates the size of the pelvis. Gently slide the pad under the victim's buttocks and center it under the greater trochanters and symphysis pubis. Secure the pad with duct tape, and then inflate the

Figure 21-19. Pelvic sling improvised with inflatable sleeping pad and duct tape.

pad as you would normally until it produces a snug fit and the pelvis is reduced (Fig. 21-19).
4. Place padding between the legs and gently tie the legs together to further stabilize the fracture in a position that is most comfortable for the victim.

Traction

In the backcountry environment, traction is essential for two fundamental reasons: (1) the general inability to provide IV volume expansion and (2) prolonged transport time to definitive care.

The primary purpose of backcountry femoral traction is to limit blood loss into the thigh. For a constant surface area, the volume of a sphere is greater than the volume of a cylinder. Pulling (via traction) the thigh compartment back into its natural cylindrical shape limits blood loss into the soft tissue. Although the main objective is to control hemorrhage and prevent shock, enhanced comfort for the victim and decreased potential for neurovascular damage are important secondary benefits. Properly applied improvised femoral traction can save lives in the backcountry, particularly on extended transports where IV fluids are not available.[6]

General Principles of Traction

The potential variety of traction designs is unlimited, but five key design principles should be considered when evaluating any femoral traction system:

1. Does the splint provide inline traction or does it incorrectly pull the leg off to the side or needlessly plantar flex the ankle?
2. Is the splint comfortable? Be sure to ask the victim.
3. Does the splint compromise neurologic or vascular function? Constantly check distal neurovascular function.
4. Is the splint durable, or will it break when subjected to backcountry stresses? As stated earlier, it might help to try the traction design on an uninjured person and then knock the device around a bit to determine its strength.
5. Is the splint cumbersome? Many reasonable splint designs become so bulky and awkward that litter transport, technical rescue, or helicopter evacuation is impossible. For example, a full-length ski splint is not compatible with evacuation in a small helicopter.

Femoral Traction Systems

Every femoral traction system has six components:

1. Ankle hitch
2. Rigid support that is longer than the leg
3. Traction mechanism
4. Proximal anchor
5. Method for securing the splint to the leg
6. Additional padding

Ankle Hitch. Various techniques are used to anchor the distal extremity to the splint. Many work well, but some are impossible to recall in an emergency. Choose an easy-to-remember technique and practice it. It is best to leave the shoe on the victim's foot and apply the hitch over it. Cut out the toe section of the shoe to periodically check circulation.

Single Runner System. Loop a long piece of webbing, shoelace, belt, or rope over itself, bringing one end through the middle to create a stirrup. After rotating it away from the person by 180 degrees, slip the hitch over the shoe and ankle.

Double Runner System. In this very straightforward technique, lay two short webbing loops ("runners") over and under the ankle as shown (Fig. 21-20A). Pass the long loop sides through the short loop on both sides (see Fig. 21-20B) and adjust as needed (see Fig. 21-20C). One advantage of this system is that it is infinitely adjustable, enabling the rescuer to center the pull from any direction. As always, proper padding is essential, especially for long transports. The victim's boot can distribute pressure over the foot and ankle but will obscure visualization and palpation of the foot. A reasonable compromise is to leave the boot on and cut out the toe section for observation.

S-Configuration Hitch. This type of hitch is preferred if the victim also has a foot or ankle injury because traction is pulled from the victim's calf instead of the ankle. Lay a long piece of webbing or similar material over the upper part of the ankle (lower calf) in an S-shaped configuration. Wrap both ends of the webbing behind the ankle and up through the loop on the other side. Pull the ends down on either side of the arch of the foot to tighten the hitch and tie an overhand knot (Fig. 21-21).

Victim's Boot System. Another efficient system uses the victim's own boot as the hitch. Cut two holes into the side walls of the boot just above the midsole and in line with the ankle joint. Thread a piece of nylon webbing or cravat through to complete the ankle hitch (Fig. 21-22). Cutting away the toe may be necessary for neurovascular assessment.

Buck's Traction. For extended transport, Buck's traction can be improvised using a closed-cell foam pad (Fig. 21-23). Wrap the pad around the lower leg as shown and loop a stirrup below the foot from medial calf to lateral calf. Fasten this assembly with a second cravat wrapped circumferentially around the calf over the closed-cell foam (duct tape or nylon webbing can be used instead of cravats). This system greatly increases the surface area over which the stirrup is applied and decreases the

A

B

C

Figure 21-20. Double runner ankle hitch. **A** and **B,** Two webbing loops (runners) are laid over and under the ankle. **C,** Completed double runner ankle hitch. The beauty of this system is its infinite adjustability. The traction can be easily centered from any angle, ensuring in-line traction.

potential for neurovascular complications and dermal ischemia. In addition, improvised Buck's traction has been used to manage backcountry hip fractures. However, it has been suggested that this technique may have little benefit.[1] If Buck's traction is used for a hip injury, use smaller amounts of traction (roughly ≤5 lb [2.5 kg]).

Rigid Support. The rigid support can be fabricated as a unilateral support (similar to the Sager traction splint or Kendrick traction device) or as a bilateral support, such as the Thomas half ring or Hare traction splint. Unilateral supports tend to be easier to apply than bilateral supports. The following are some ideas for rigid support.

Double Ski Pole or Canoe Paddle System. This is fashioned like a Thomas half ring, with the interlocked pole straps slipped under the proximal thigh to form the ischial support. Some mountain guides carry a prefabricated drilled ski pole section or aluminum bar that can be used to stabilize the distal end of this system (Fig. 21-24).

Single Ski Pole or Canoe-Kayak Paddle. Use a single ski pole or paddle either between the legs, which is ideal for bilateral femur fractures, or lateral to the injured leg. The ultimate rigid support is an adjustable telescoping ski pole used laterally. Adjust the pole to the appropriate length for each victim, making the splint compact for litter work or helicopter evacuation (Fig. 21-25).

Tent Poles. This system uses conventional sectioned tent poles. Fit the poles together to create the ideal length rigid support. Because of their flexibility, tent poles must be well secured to the leg to prevent them from flexing out of position. Place a blanket pin or bent tent stake (Fig. 21-26) in the end of the pole

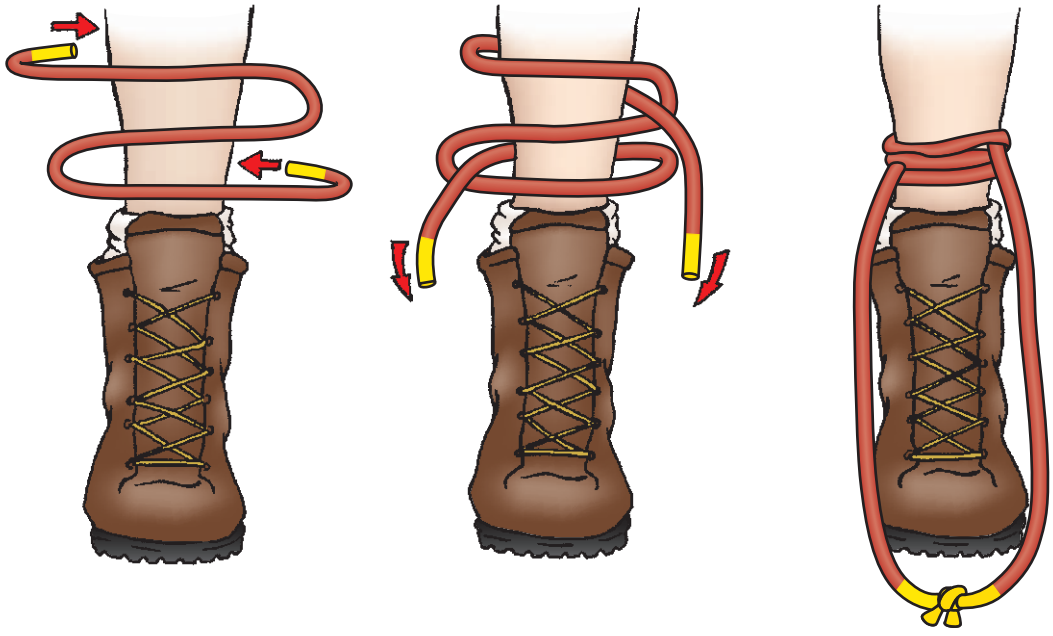


Figure 21-21. S-configuration hitch for traction splinting. The system should be configured distally over the boot to distribute the forces.

Figure 21-22. Traction using cut boot and cravat.

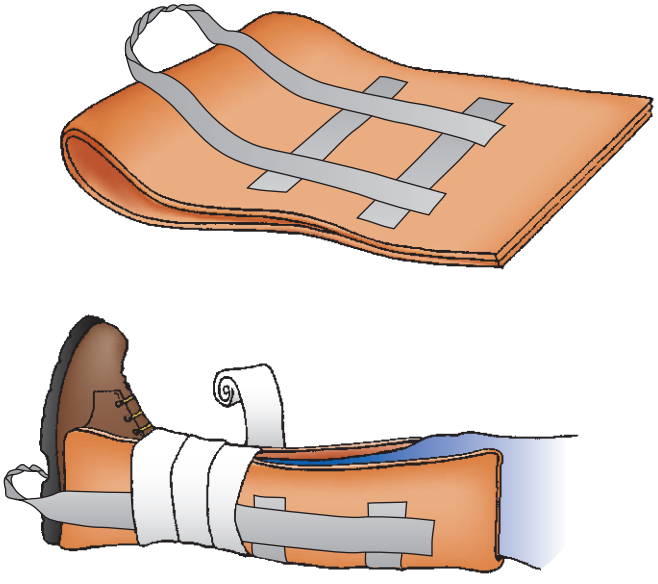
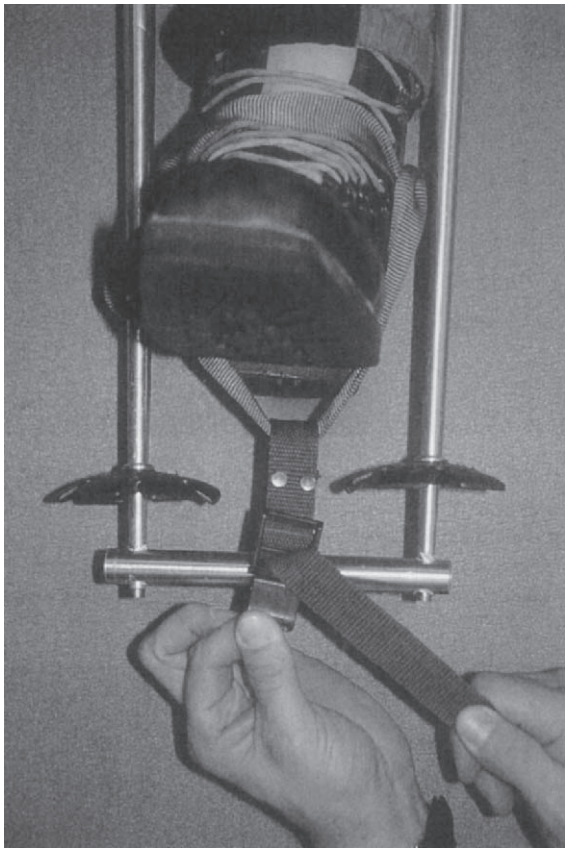


Figure 21-23. Buck's traction. Duct tape stirrups are added to a small foam pad that is wrapped around the leg. The entire unit is wrapped with an Ace bandage. This system helps distribute the force of the traction over a large surface area.



A

B

Figure 21-24. **A,** Double ski pole system with prefabricated cross-bar and webbing belt traction. A prefabricated drilled ski section is used to attach the ends of two ski poles. Traction is applied with a webbing belt and sliding buckle. **B,** A Fastex-type or cam-lock utility strap makes a very efficient traction device and can be adapted for most systems.

Figure 21-25. Single ski pole system. An adjustable telescoping ski pole is used as the rigid support. A stirrup is attached to a carabiner placed over the end of the pole. Traction is applied by elongating the ski pole while another rescuer provides manual traction on the victim's leg. Additional padding and securing follow (not shown).

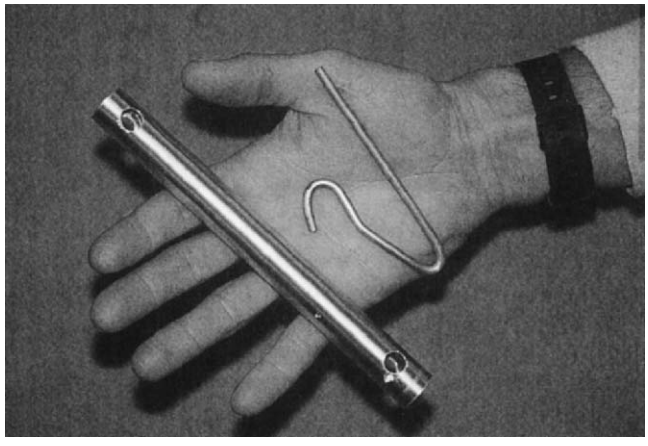


Figure 21-26. Prefabricated drilled tent pole section and bent tent stake. The ski pole section is used to stabilize the end of a double ski pole traction system. This can be improvised on site if necessary. The bent tent stake serves as a distal traction anchor if a tent pole is used as the rigid support.

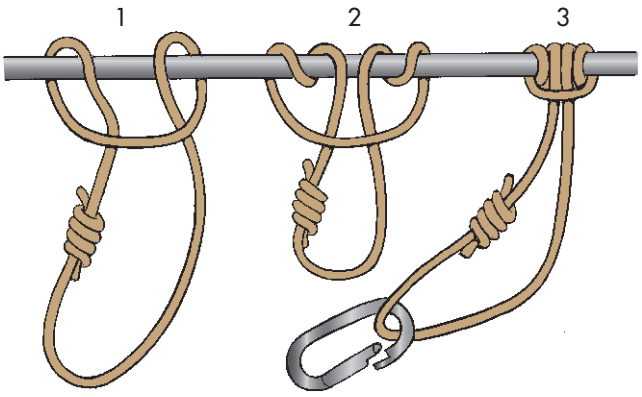


Figure 21-27. A Prusik knot made from a small-diameter cord is used as an adjustable distal traction anchor. Although two wraps are shown in the illustration, an additional wrap adds further security when applied to a smooth surface, such as a kayak paddle.

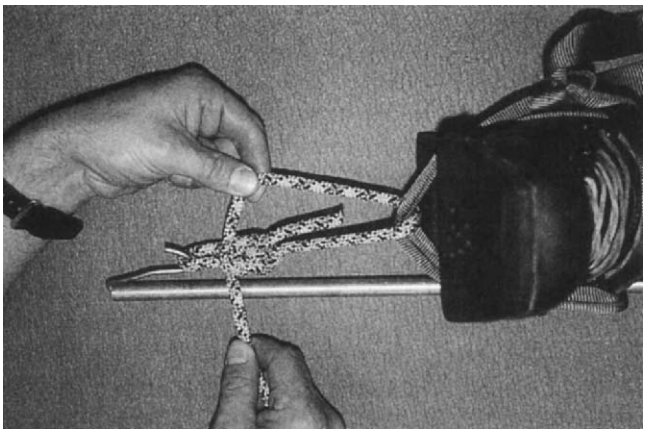


Figure 21-29. Tent pole traction with trucker's hitch. A bent tent stake is placed into the end of the tent pole as the distal traction anchor. A simple trucker's hitch is used to provide traction.

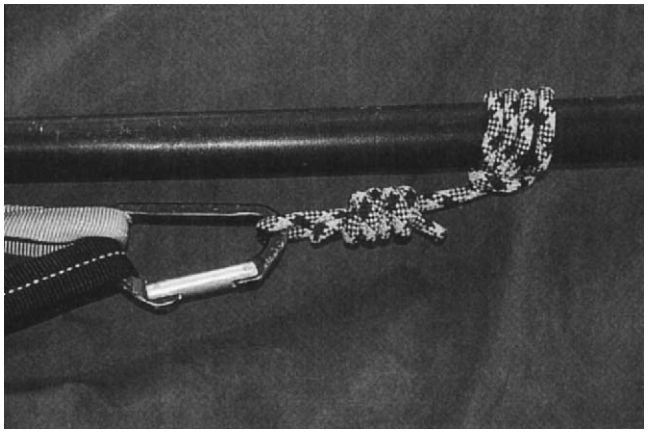


Figure 21-28. Two Prusik wraps are shown. Three or four wraps provide additional friction and security. If the Prusik knot slips, it can be easily taped in place.

to provide an anchor for the traction system. Alternately, use a Prusik knot (Fig. 21-27) to secure the system to the end of the tent pole (Fig. 21-28).

Miscellaneous. Any suitable object, such as a canoe or kayak paddle (see Figure 21-28), two ice axes taped together at the handles, or a straight branch, can be used to make a rigid support. Although skis immediately come to mind as suitable rigid components, they are too cumbersome to work effectively. Because of their length, skis may extend far beyond the victim's feet or require placement into the axillae, which is unnecessary and inhibits mobility (e.g., sitting up during transport). Premanufactured canvas pockets, available from the National Ski Patrol System, provide a ski tip and tail attachment grommet for use with the ski system.

Traction Mechanism. The first popularized modern improvised traction mechanism was the Boy Scout–style Spanish windlass. Although these systems work and look good in the movies, they can be awkward to apply and are often not durable. The windlass can unspin if it is inadvertently jarred and can apply rotational forces to the leg.

The amount of traction required may be difficult to estimate. A general rule is to use 10% of body weight or about 10 to 15 lb (4.5–7 kg) for the average victim. Consider practicing with a commercial system, such as a Sager Traction Device (Minto Research & Development, Inc., Redding, CA), ahead of time to get a feeling for what these forces look and feel like. Check the opposite extremity to compare length. After the traction is applied, always recheck distal neurovascular function (circulation, sensation, and movement).

Cam Lock or Fastex-Like Slider. This simple, effective system uses straps that have a Fastex-like slider (ITW Fastex, Des Plaines, IL). Such straps are often used as waist belts or to hold items to packs. Alternately, a cam lock with nylon webbing can be used. Attach the belt to the distal portion of the rigid support and then to the ankle hitch. Traction is easily applied by cinching the nylon webbing (see Figure 21-24).

Trucker's Hitch. A windlass can be easily fashioned using small-diameter line (parachute cord) and a standard trucker's hitch for additional mechanical advantage (Fig. 21-29).

Prusik Knot. Almost any system can be rigged with a Prusik knot (see Figure 21-27). Prusiks are ideal for providing traction from rigid supports with few tie-on points (such as a canoe paddle shaft or a tent pole). The Prusik knot can be used to apply the traction (by sliding the knot distally) or simply as an attachment point for one of the traction mechanisms already mentioned.

Litter Traction. If no rigid support is available and a rigid litter such as a Stokes is being used, apply traction from the rigid bar

at the foot end of the litter. If this system is used, ensure that the victim is immobilized on the litter with adequate countertraction, such as inguinal straps.

Proximal Anchor. The simplest proximal anchor uses a single proximal thigh strap, which can be made from a piece of climbing webbing or a prefabricated strap, belt, or cam lock (Figs. 21-30 and 21-31). A cloth cravat can be used in a pinch. On the river, a life jacket can be used (Fig. 21-32). When climbing, a climbing harness is ideal.

Securing and Padding. Check all potential pressure points to ensure that they are adequately padded. An excellent padding system can be made by first covering the upper and lower leg with a folded length of Ensolite (Fig. 21-33). This is preferred over a circumferential wrap because the folded system allows you to see the extremity. The victim is more comfortable if femoral traction is applied with the knee in slight flexion (padding placed beneath the knee during transport). Secure the splint firmly to the leg. Almost any straplike object will work, but a 4- to 6-inch (10–15 cm) Ace bandage wrapped circumferentially provides a comfortable and secure union. Finally, strap or tie the ankles or feet together (with adequate padding between the legs) to give the system additional stability. Tying the ankles together also prevents the injured leg from excessive external rotation during transport, which might otherwise greatly add to the victim's discomfort.

Extremity Splints

Splint all fractures before the victim is moved unless his or her life is in immediate danger. In general, make sure the splint incorporates the joints above and below the fracture. If possible, the splint should be fashioned on the uninjured extremity and then transferred to the injured one.

On ski trips, skis and poles can be used as improvised splints. On white-water trips, canoe and kayak paddles can be used in a similar manner. Airbags used as flotation for kayaks and canoes can be converted into pneumatic splints for arm and ankle injuries. The minicell or Ethafoam (Dow, Midland, MI) pillars found in most kayaks can be removed and carved into pieces to provide upper and lower extremity splints. A life jacket can be molded into a cylinder splint for knee immobilization or into a pillow splint for the ankle.

The flexible aluminum stays found in internal frame packs can be molded into upper extremity splints. Other improvised

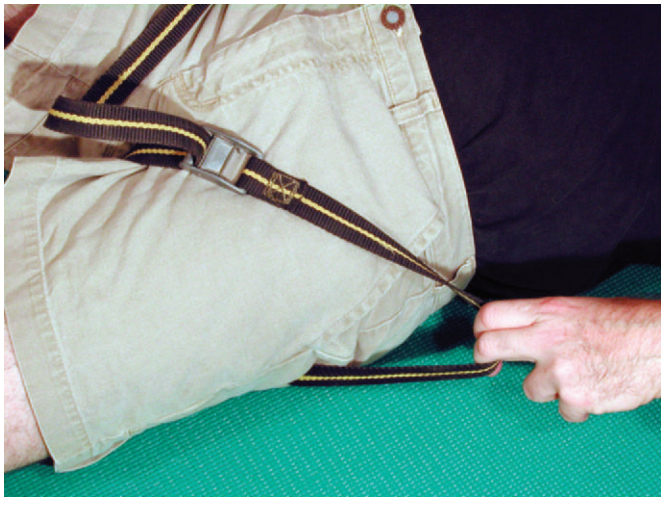
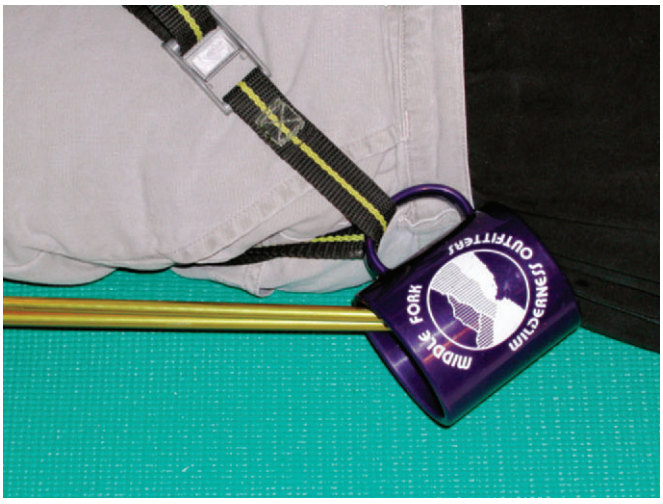


Figure 21-30. Proximal anchor using cam lock belt. The belt is applied as shown. The strap is adjusted loosely to allow the belt to ride up to the point of the hip. If the strap is improperly tightened, it can create pressure over the fracture, and it moves the traction point to a less optimal distal position. Padding is helpful, but not always necessary if the victim is wearing pants and the strap is properly adjusted.

A

B

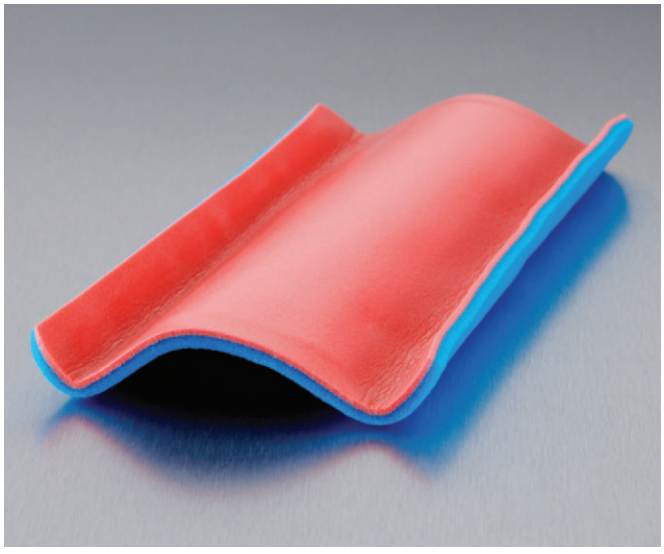
Figure 21-31. **A,** River dry bag used to create a simple proximal support with a kayak paddle. **B,** Here a paddle is pushed up against the proximal support strap. It is taped for security but the forces are pushing against the strap, not the tape.



C

Figure 21-31, cont'd. **C,** A similar technique could be used with a ski pole or other suitable object.

Figure 21-32. Life jacket proximal anchor. An inverted life jacket worn like a diaper forms a well-padded proximal anchor. A kayak paddle is rigged to the life jacket's side adjustment strap.



A

B

Figure 21-33. Folded Ensolite padding often provides better visualization of the extremity than does a circumferential wrap.

splinting material includes sticks or tree limbs, rolled-up magazines, books or newspapers, ice axes, tent poles, and dirt-filled garbage bags or fanny packs.

Ideally, a splint should immobilize the fractured bone in a functional position. In general, "functional position" means that the legs should be straight or slightly bent at the knee, the ankle and elbow bent at 90 degrees, the wrists straight, and the fingers flexed in a curve as if the person were attempting to hold a can of soda or a baseball.

Splints can be secured in place with strips of clothing, belts, pieces of rope or webbing, pack straps, gauze bandages, or elastic bandage wraps.

Padded aluminum can be molded into various configurations to splint extremity injuries. Padded aluminum splints (e.g., SAM Splint) are built from a thin core of aluminum, sandwiched between two layers of closed-cell foam. When molded into any of several "structural curves," padded aluminum splints become much more rigid and can be used to immobilize most fractured or injured extremities. If prolonged use (more than a few hours) is anticipated, place absorbent material, such as cotton cloth, between the splint and the skin to prevent skin irritation. Also, to prevent uncomfortable pressure points during prolonged use, place soft padding (such as

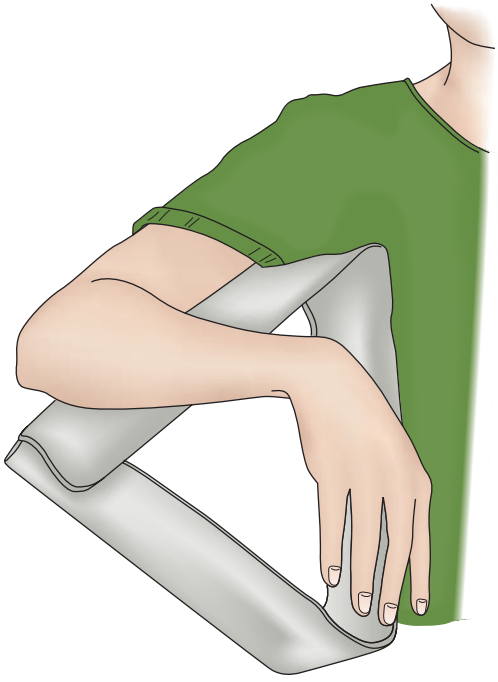


Figure 21-34. Tripod splint for unreduced anterior shoulder dislocation. This splint holds the arm in abduction when adduction is not possible. Additional padding should be added where necessary and the splint secured to the arm with an elastic wrap or other bandaging material.

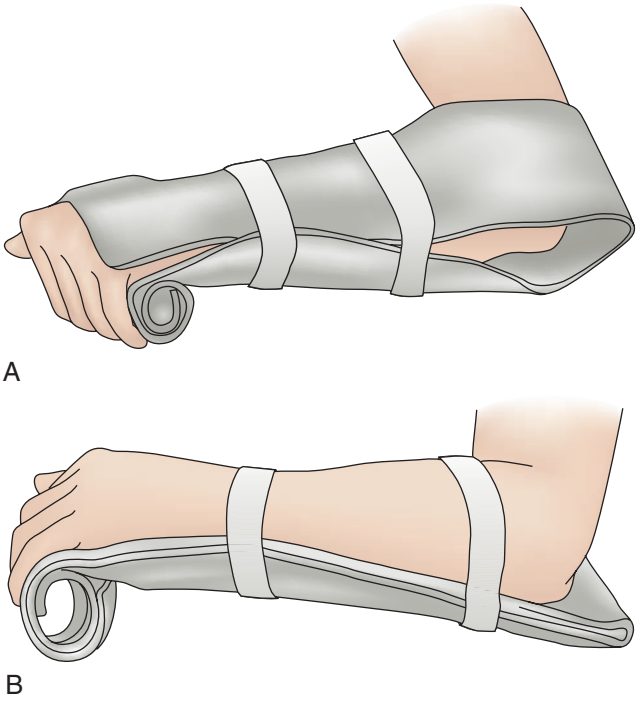


Figure 21-36. Forearm splint. These splints are used for treatment of wrist or forearm fractures. The sugar-tong splint **(A)** prevents pronation and supination and has the advantage of greater security and protection than the volar splint **(B)** because of its anterior-posterior construction.

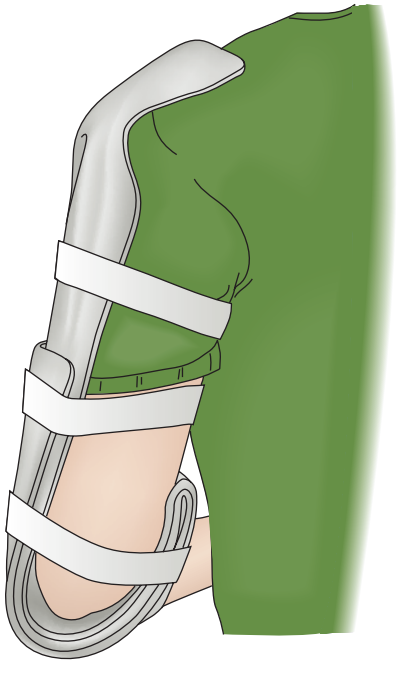


Figure 21-35. Humerus splint. Used in conjunction with a sling and swath, this splint adds extra support and protection for a fractured humerus.

gauze pads) around all bony prominences (Figs. 21-34 to 21-41).

Functional Splints

Although most splints are designed to immobilize an injured extremity completely, in the backcountry a splint may need to allow for a limited range of motion so the victim can facilitate his or her own rescue. Many functional splints can be improvised quickly using nothing more than a closed-cell foam sleeping pad and some tape or elastic wrap. With the advent of inflatable sleeping pads (e.g., Therm-a-Rest), foam pads are not as ubiquitous as they once were in the backcountry. However, many of these splints can be made using a partially inflated Therm-a-Rest. Once applied, these pads can be inflated to provide the necessary support, fit, and comfort (Fig. 21-42).

Other Wraps and Bandages

Functional Shoulder Immobilizer (Shoulder Spica Wrap)

After a dislocated shoulder is reduced, standard treatment is to completely immobilize the arm with a sling and swath. This, however, prevents the victim from using the extremity to facilitate his or her own evacuation. A more functional system can be made using a 6-inch (15 cm) elastic wrap. This method allows the victim limited function (e.g., ski poling or kayak

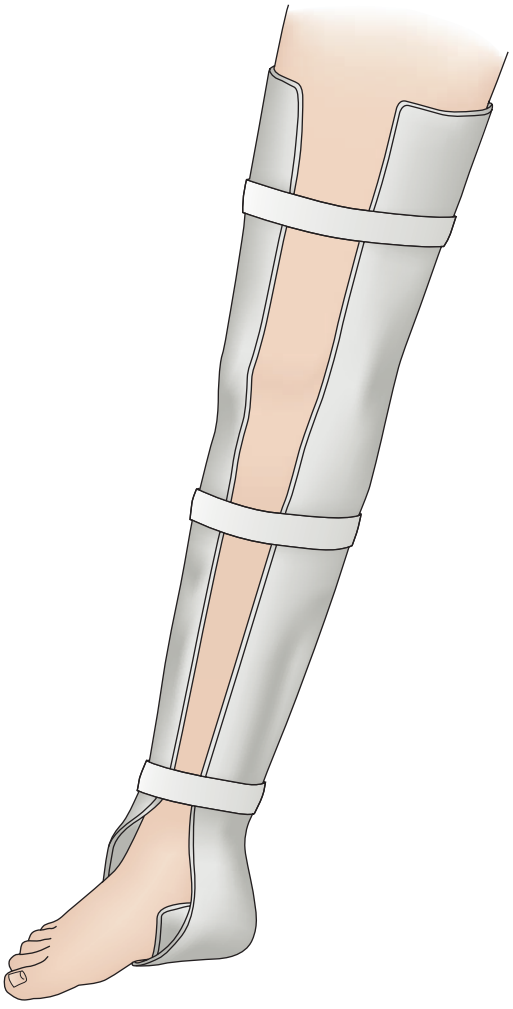


Figure 21-37. Lower leg or ankle splint. A sugar-tong splint can be used to immobilize fractures of the tibia, fibula, or ankle. When used on an adult, two splints should be used. A third splint may be placed posteriorly for additional support.

A

B

Figure 21-38. **A** and **B,** Padded aluminum thumb spica.

paddling) while still preventing complete abduction of the arm (Fig. 21-43).

Triangular Bandage

One of the most ubiquitous components of first-aid kits and one of the easiest to replace through improvisation is the triangular bandage. The need to carry this bulky item, which is commonly used to construct a sling and swath bandage for shoulder and arm immobilization, can be eliminated by carrying two or three safety pins. Pinning the shirt sleeve of the injured arm to the chest portion of the shirt effectively immobilizes the extremity against the body (Fig. 21-44A).

If the victim is wearing a short-sleeved shirt, the bottom of the shirt can be folded up and over the arm to create a pouch. This can be pinned to the sleeve and chest section of the shirt to secure the arm (see Fig. 21-44B).

Triangular bandages are also used for securing splints and constructing pressure wraps. Common items, such as socks, shirts, belts, pack straps, webbing, shoe laces, fanny packs, and underwear, can easily be substituted.

WOUND MANAGEMENT

The same principles that govern wound management in the emergency department apply in the wilderness. The main problem faced in the wilderness is access to adequate supplies. In deciding to close a wound primarily or pack it open, take into account the mechanism of injury, age of the wound, site of the wound, degree of contamination, and ability to effectively clean the wound.

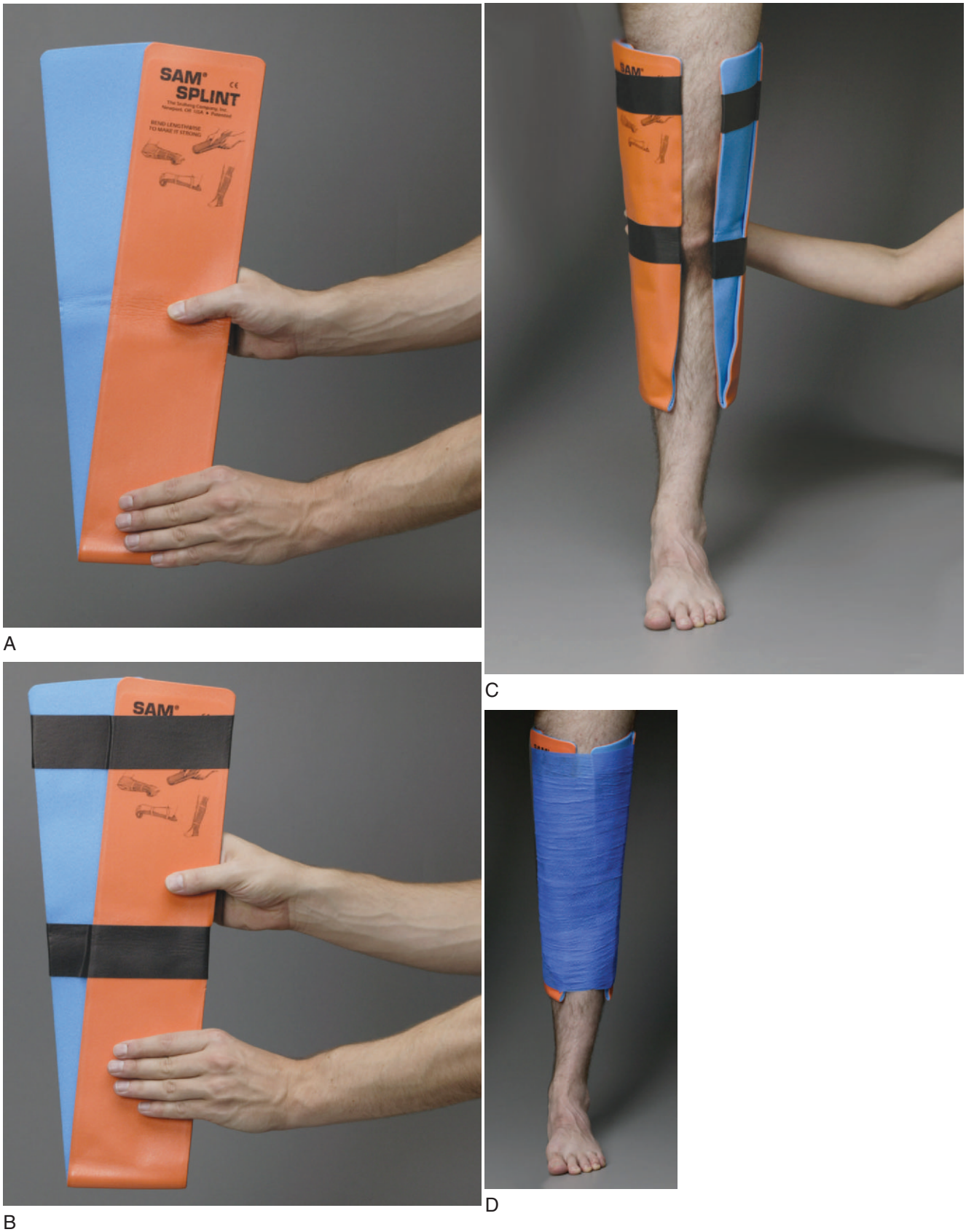


Figure 21-39. Knee immobilizer using two padded aluminum splints. The splints are folded in half, then fanned out (wider at the top for the thigh) and taped. Tape is applied to maintain the fan shape. The splint is then applied bilaterally and secured.

Figure 21-40. Posterior arm splint. This splint is cut from a 5- to 9-gallon (1.3–2.4 L) plastic fuel or water can. When used with appropriate padding, this forms an excellent splint for injured or fractured elbows.

Figure 21-41. Webbing sling. An 8-foot (2.5 m) length of 1-inch (2.5 cm) tubular or flat webbing is used to form a functional arm sling. A Crazy Creek Chair can be used to improvise both upper and lower extremity splints. Its inherent integral strapping system precludes the need for additional straps or tape.

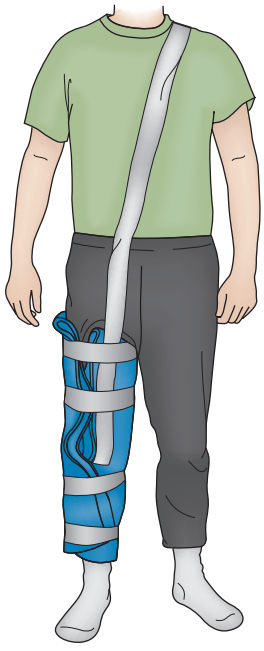


Figure 21-42. Functional knee and lower leg immobilizer. Wrap a sleeping pad around the lower leg from the mid thigh to the foot. Fold the pad so that the top of the leg is not included in the splint. This provides better visualization of the extremity and leaves room for swelling. A full-length pad forms a very bulky splint and may need to be trimmed before rolling. Because of the conical shape of the lower extremity and the effects of gravity, foam-pad lower extremity splints tend to work their way inferiorly when the victim ambulates. A simple solution is to use duct tape "suspenders" to keep the splint from migrating downward.

Wound Irrigation

The primary determinants of infection are bacterial counts and amount of devitalized tissue remaining in the wound.[20] Ridding a wound of bacteria and other particulate matter requires more than soaking and gentle washing with a disinfectant.[26] Irrigating the wound with a forceful stream is the most effective method of reducing bacterial counts and removing debris and contaminants.[29,36] The cleansing capacity of the stream depends on the hydraulic pressure under which the fluid is delivered.[19,35] Irrigation is best accomplished by attaching an 18- or 19-gauge catheter to a 35-mL syringe, or a 22-gauge needle to a 12-mL syringe. This creates hydraulic pressure in the range of 7 to 8 lb/in^2 and 13 lb/in^2 (3 to 3.5 and 6 kg/cm^2), respectively.[19,31,35] The solution is directed into the wound from a distance of 1 to 2 inches (2.5–5 cm) at an angle perpendicular to the wound surface and as close to the wound as possible. The amount of irrigation fluid varies with the size and contamination of the wound, but should average no less than 250 mL.[19] Remember: "the solution to pollution is dilution."

Which irrigation solution is best for open wounds? Those who subscribe to the theory that nothing should enter a wound that could not be instilled safely into the eye believe that normal saline is the best solution.[12,21] In a study of 531 patients with traumatic wounds, there was no significant variation in infection rates among sutured wounds irrigated with normal saline, 1% povidone-iodine, or pluronic F68 (Shur-Clens).[15]

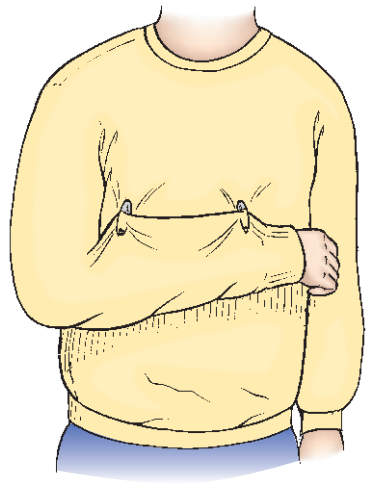
A

B

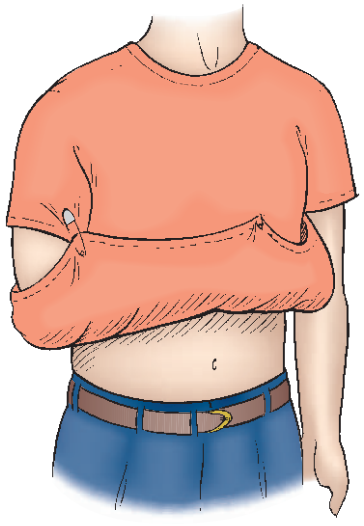
C

D

Figure 21-43. **A–D,** Shoulder spica wrap.



A



B

Figure 21-44. Techniques for pinning the arm to the shirt as an improvised sling. **A,** With a long-sleeved shirt or jacket, the sleeved arm is simply pinned to the chest portion of the garment. **B,** With a short-sleeved shirt, the bottom of the shirt is folded up over the injured arm and secured to the sleeve and upper shirt.

Tap water has been found to be as effective for irrigating wounds as sterile saline. In fact, the infection rate was significantly lower after irrigation with tap water, and no infections resulted from the bacteria cultured from the tap water.[3]

Improvised wound irrigation requires only a container that can be punctured to hold the water, such as a sandwich or garbage bag, and a safety pin or 18-gauge needle (Box 21-3).

Wound Closure

Before a wound is closed, remove all foreign material and grossly devitalized tissue. Debridement can be accomplished using scissors, knife, or any other sharp object, and wounds can be closed with sutures, staples, tape, pins, or glue. Although suturing is still the most widely used technique, stapling and gluing are ideal methods for closing wounds in the wilderness.

Box 21-3. Recommended Technique for Wound Irrigation

1. Fill a sandwich or garbage bag with disinfected water.
2. Disinfect the water with iodine tablets, iodine solution, or povidone-iodine or by boiling it.
3. Normal saline can be made by adding 2 teaspoons (9 g) of salt per liter of water.
4. Seal the bag.
5. Puncture the bottom of the bag with an 18-gauge needle, safety pin, fork prong, or knife tip.
6. Squeeze the top of the bag forcefully while holding it just above the wound, directing the stream into the wound.
7. Use caution to ensure that none of the irrigation fluid splashes into your eyes.

Box 21-4. Wound Taping Technique

1. Obtain hemostasis, and dry the wound edges.
2. Apply benzoin or cyanoacrylate glue to the skin adjacent to the wound. Benzoin should be allowed to dry long enough for it to become tacky, but tape should be applied to the glue while the glue is still wet.
3. Tape should be cut to $^1/_4$-inch or $^1/_2$-inch widths (6–13 mm), depending on the size of the laceration, and to a length that allows for $^3/_4$ to 1 inch (2 to 3 cm) of overlap on each side of the wound.
4. Secure one half of the tape to one side of the wound. Oppose the opposite wound edge with a finger while the tape is secured to the other side.
5. Wound tapes should have gaps of $^1/_{16}$ to $^1/_8$ inch (2–3 mm) between them to allow for serous drainage.
6. Cross-stays of tape can be placed perpendicular over the tape ends to prevent them from peeling off.
7. Additional glue can be applied to the tape edges every 24 hours to reinforce adhesion.

Clinical studies of the use of staples to close traumatic lacerations have found various advantages of stapling over suturing: wound tensile strength is greater, there is less inflammation, time required for closure is shorter, and fewer instruments are needed.[32] Most important, cosmetic outcome is not compromised.[18] Staplers are lightweight, presterilized, and easy to use.

Wound Taping

Skin Tapes. Skin tapes are useful for shallow, nongaping wounds and have several advantages over suturing, including reduced need for anesthesia, ease of application, decreased incidence of wound infection, and availability. Any strong tape can be used to improvise skin tape strips, but duct tape works especially well (Box 21-4). Puncturing holes in the tape before application helps prevent exudate from building up under the tape.

Wipe the skin with a solvent such as acetone to remove oil and sweat. Then, apply benzoin to the skin before the tape to augment adhesion. Wound taping does not work well over joints or on hairy skin surfaces unless the hair is first removed.

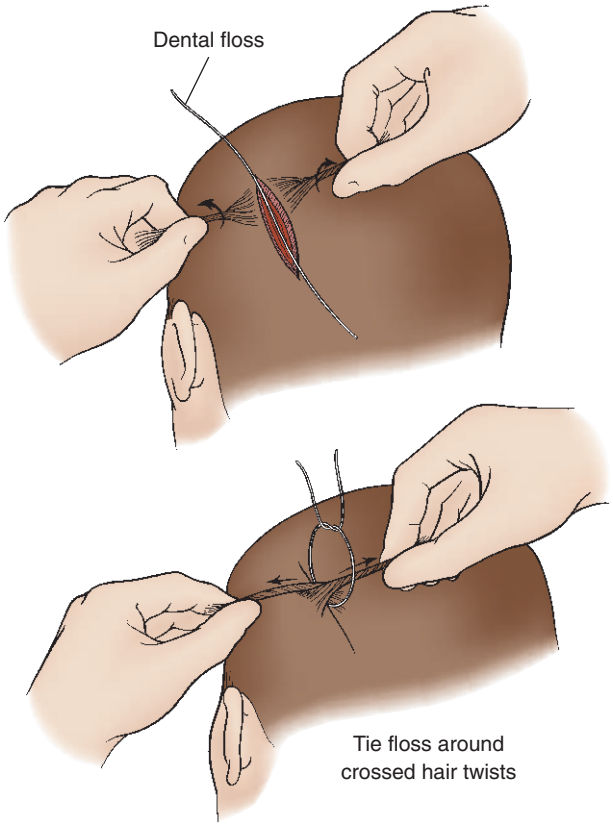


Figure 21-45. Scalp laceration closed using dental floss.

Hair-Tying a Scalp Laceration. If you are faced with a bleeding scalp laceration and the injured person has a healthy head of hair, you can tie the wound closed using the victim's own hair and a piece of suture (0-silk works best), dental floss, sewing thread, or thin string. Take the material and lay it on top of and parallel to the wound. Twirl a few strands of hair on each side of the wound and then cross them over the wound in opposite directions so that the force pulls the wound edges together. Have an assistant tie the strands of hair together with the material while you hold the wound closed with the strands of hair. A square knot works best (Fig. 21-45). Repeat this technique as many times as necessary along the length of the wound to close the laceration.

Gluing

The concept of gluing wounds is not new; the U.S. Army used a quick-sealing glue to treat battlefield wounds in Vietnam, and Histoacryl (*N*-butyl-2-cyanoacrylate) tissue adhesive has been used in Europe and Canada for sutureless skin closure for more than a decade.[41]

The U.S. Food and Drug Administration (FDA) has approved a topical skin adhesive to repair skin lacerations. Dermabond (2-octyl cyanoacrylate) is packaged in a small single-use applicator. Tissue glue is ideal for backcountry use because it precludes the need for topical anesthesia, is easy to use, reduces the risk of needle stick injury, and takes up much less room than a conventional suture kit. When applied to the skin surface, tissue glue provides strong tissue support and peels off in 4 to 5 days without leaving evidence of its presence.[31] It provides a faster and less painful method for closing lacerations than does suturing and has yielded similar cosmetic results in children with facial lacerations (Box 21-5).[30] Tissue glue evokes a mild acute inflammatory reaction with no tissue necrosis.[39]

Dermabond has four times the three-dimensional breaking strength of Histoacryl and forms a more flexible bond, thus providing a stronger and longer bond than its European counterpart. Petroleum-based ointments and salves, including antibiotic ointments, should not be used on the wound after gluing because these substances can weaken the polymerized film and cause wound dehiscence.

Tissue glue has also been used successfully to treat superficial painful fissures of the fingertips ("polar hands"), which commonly occur in cold climates and at high elevations.[4]

Box 21-5. Technique for Gluing Lacerations

1. Irrigate the wound with copious amounts of disinfected water.
2. Control any bleeding with direct pressure. Place a gauze pad moistened with oxymetazoline (Afrin) nasal spray into the wound to help control bleeding.
3. Once hemostasis is obtained, approximate the wound edges using fingers or forceps.
4. Paint the tissue glue over the apposed wound edges using a very light brushing motion of the applicator tip. Avoid excess pressure of the applicator on the tissue because this could separate the skin edges, forcing glue into the wound. Apply multiple thin layers (at least three), allowing the glue to dry between each application (about 2 minutes).
5. Glue can be removed from unwanted surfaces with acetone, or loosened from skin with petroleum jelly.

IMPROVISED BLISTER MANAGEMENT

To dress blisters without moleskin, molefoam, or other commercial blister dressing, you can improvise one with a piece of duct tape. Duct tape's smooth outer surface provides protection from friction, while its adhesive side adheres strongly to skin.

A sandwich bag can be used to improvise another type of blister dressing. It simulates the Blist-O-Ban, which was developed by SAM Medical Products as an innovative technique (BursaTek technology) to prevent blisters. The smooth, gliding surface of the bag helps to stop friction and reduce development of hot spots and blisters. Cut the corner of the sandwich bag and apply a lubricant between the two surfaces. Secure the piece of bag to the blister site with tape or glue (Fig. 21-46).

RING REMOVAL

Rings should always be removed quickly after injury to fingers and trauma to the hands; progressive swelling may cause rings to act as tourniquets. If a ring cannot be removed with soap or lubricating jelly, the string wrap technique can be used. Pass a

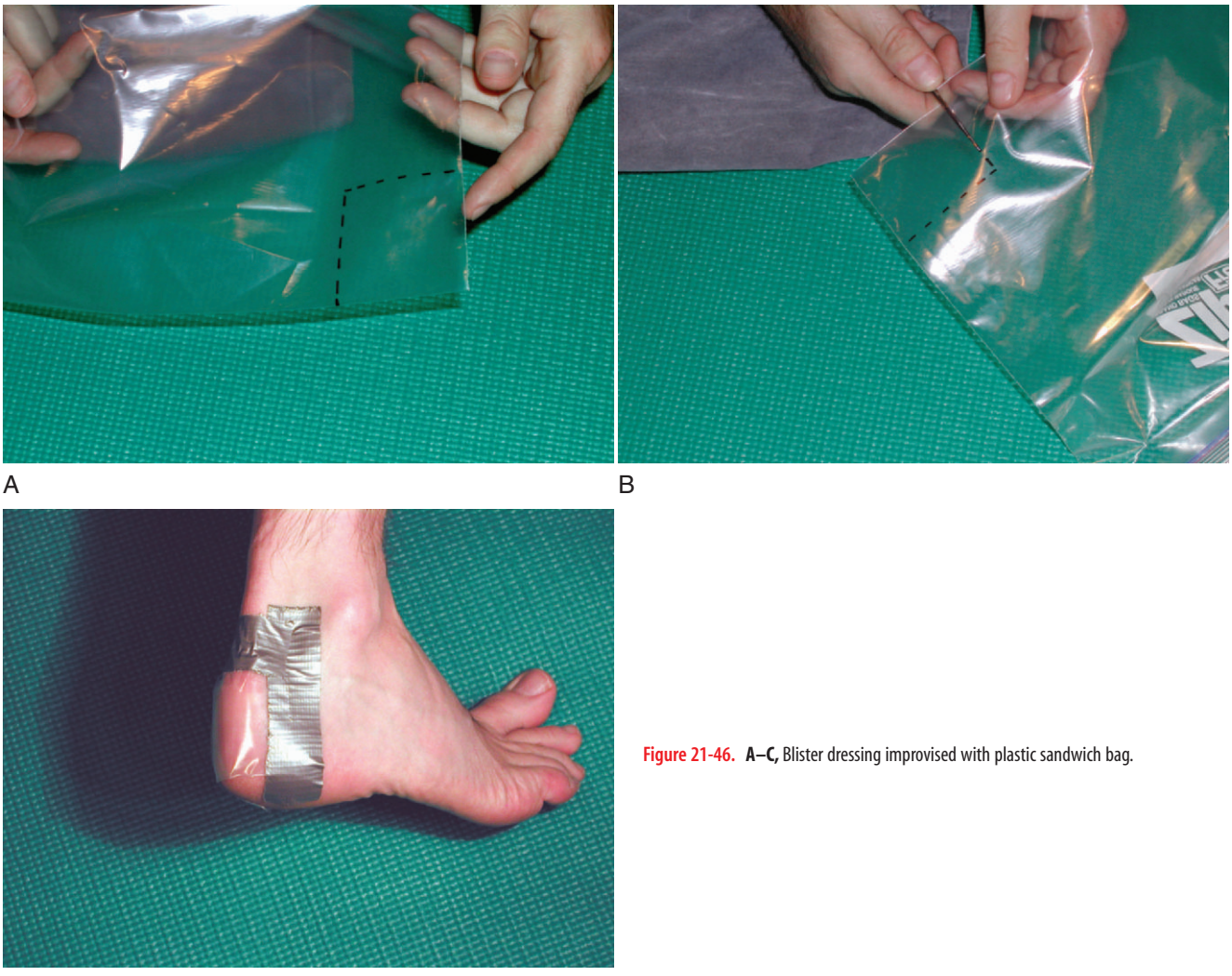


Figure 21-46. **A–C,** Blister dressing improvised with plastic sandwich bag.

20-inch (51 cm) length of fine string, dental floss, umbilical tape, or thick suture between the ring and the finger. Pull the string so that most of it is on the distal side of the digit. Then wrap it around the swollen finger from proximal to distal, beginning next to the ring and continuing past the proximal interphalangeal joint. Place successive loops of the wrap close enough together to prevent any swollen skin from bulging between the strands. Remove the ring by unwinding the proximal end of the string and forcing the ring over the distal string. If the string is not long enough, the technique may require repeated wraps (Fig. 21-47).

IMPROVISATIONAL TOOLKIT

Some people, convinced they could whittle a Swan-Ganz catheter from a tree branch, enter the wilderness with nothing more than a Swiss Army knife. However, a little foresight and preparation make improvisation much easier. Efficiency translates into speedy preparation and assembly, which ultimately results in better care. The following section lists items that facilitate improvisation in the field.

Knife

The knife can be a fairly simple model, but it should have an awl for drilling holes into skis, poles, sticks, and so on. The awl on a Swiss Army knife works quite well for this purpose. This allows one to create well-fitted components during improvisation (e.g., a drilled cross-bar attached to ski tips for an improvised rescue toboggan).

Tape

Carry some form of strong, sticky, waterproof tape. This item cannot be improvised. Use either cloth adhesive tape (already in the medical kit) or duct tape. Duct tape is ideal for nearly all tasks, even being useful on skin when needed (e.g., to close wounds, treat blisters, or tape an ankle). Note, however, that some persons may be sensitive to the adhesive. Fiberglass

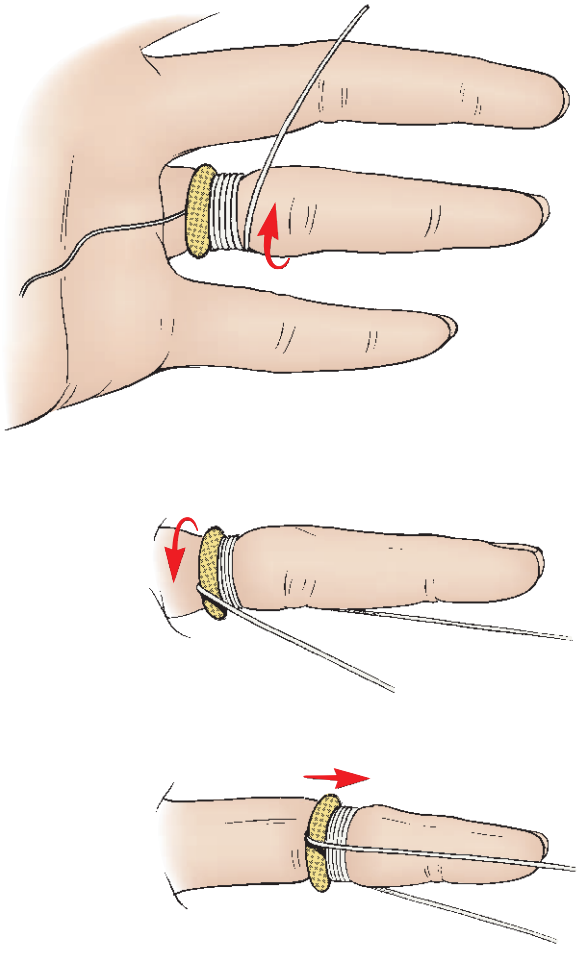


Figure 21-47. String technique for removing a ring from a swollen finger (see text).

Box 21-6. Uses of a Safety Pin

Safety pins can be used:

- To pin the anterior aspect of the tongue to the lower lip (use two pins) to establish an airway in an unconscious victim whose airway is obstructed (see Figure 21-1)
- To replace the lost screw in a pair of eyeglasses to prevent the lens from falling out
- To improve glasses: Draw two circles in a piece of duct tape where your eyes would fit. Use a pin to make holes in the circles, then tape this to your face. The pinholes will partially correct myopic vision and protect the eyes from ultraviolet radiation. Slits can also be used for improvised sunglasses.
- To perform neurosensory skin testing
- To puncture plastic bags for irrigation of wounds
- To remove embedded foreign bodies from the skin
- To drain an abscess or blister
- To relieve a subungual hematoma
- As a fishhook
- As a finger splint (mallet finger)
- As a sewing needle, using dental floss as thread
- To hold gaping wounds together
- To replace a broken clothing zipper
- To hold gloves or mittens to a coat sleeve
- To unclog jets in a camping stove
- To pin triage notes to multiple victims
- To remove a corneal foreign body (with ophthalmic anesthetic)
- In a sling and swath for shoulder or arm injuries
- To fix a ski binding
- To extract the clot from a thrombosed hemorrhoid
- To pin a strap or shirt tightly around the chest for rib fracture support
- To remove ticks

strapping tape has greater tensile strength and is ideal for joining rigid components, such as taping two ice axes together. However, it is less sticky than duct tape and not as useful for patching torn items. Extra tape can be carried by wrapping lengths of it around pieces of gear.

Plastic Cable Ties

Lightweight cable ties can be used to bind almost anything together (e.g., binding pack frames together for improvised litters or ski poles together for improvised carriers). They are also perfect for repairing many items in the backcountry.

Parachute Cord

Parachute cord has hundreds of uses in the backcountry. It can be used for trucker's hitch traction and for tying complex splints together. Parachute cord is light; carry a good supply.

Safety Pins

See Box 21-6 for various uses for safety pins.

Wire

Braided picture-hanging wire works well because it is supple and ties like line. Its strength makes it superior for repairing and improvising components under an extreme load, such as fabricating improvised rescue sleds or repairing broken or detached ski bindings.

Bolts and Wing Nuts

Bolts and wing nuts make the job of constructing an improvised rescue sled much easier (see Improvised Rescue Sled or Toboggan section). Bolts are useful only if holes can be created to put them through. Therefore, a knife with an awl is needed for drilling holes through skis, poles, or other improvised items.

Prefabricated Cross-Bar

The prefabricated cross-bar can be used for double ski pole traction splint systems. A cross-bar is easily fabricated from a branch or short section of a ski pole, but carrying a prefabricated device, such as a 6-inch (15 cm) predrilled ski pole section, saves time (see Figure 21-24).

Ensolite (Closed-Cell Foam) Pads

Since the introduction of Therm-a-Rest types of inflatable pads, closed-cell foam has become increasingly scarce; however, closed-cell foam (Ensolite) is still the ultimate padding for almost any improvised splint or rescue device. The uses for

closed-cell foam are virtually unlimited. Even die-hard Therm-a-Rest fans should carry a small amount of closed-cell foam, which is lightweight and doubles as a comfortable seat cushion. Furthermore, unlike inflatable pads, Ensolite will not puncture and deflate.

Therm-a-Rest pads also have their place, being useful for padding for long bone splints and immobilizers (e.g., an improvised universal knee immobilizer). An inflatable pad can also be used to cushion pelvic fractures. First, wrap the deflated pad around the pelvis. Then secure the pad with tape and inflate it, creating an improvised substitute for military antishock trousers (MAST device).

Fluorescent Surveyor's Tape

Surveyor's tape can be used much like Hansel and Gretel's breadcrumbs to help relocate a route into or out of a rescue scene. It is also ideal for marking shelters in deep snow and can serve as a wind sock during helicopter operations on improvised landing zones. Surveyor's tape is not biodegradable, so it should always be removed from the site after the rescue is completed.

Space Blanket or Lightweight Tarp

For improvising hasty shelters in times of emergency, some form of tarp is essential. In the snow, a slit trench shelter can be built in a matter of minutes using a tarp. Otherwise, the complex and time-consuming construction of improvised structures, such as snow caves, igloos, or tree branch shelters, might be necessary. Typically, little time or help is available for this task during emergencies. In addition, tarps are essential for "hypothermia wraps" when managing injured persons in cold or wet conditions. The only advantage of a space blanket over other tarps is its small size, which means there is a good chance it was packed for the trip.

IMPROVISED EYEGLASSES

Exposure of unprotected eyes to ultraviolet radiation at high altitudes may produce photokeratitis (snow blindness). Symptoms are delayed, and the victim is often unaware that an eye injury is developing. When sunglasses are lost at 14,000 ft (4267 m) in the snow, photokeratitis can develop in 20 minutes. One can improvise sunglasses from duct tape, cardboard, or other light-impermeable material that can be cut. Cardboard "glasses" with narrow eye slits can be taped over the eyes for protection.

Slits can also be cut into a piece of duct tape that has been folded over on itself with the sticky sides opposing. After a triangular wedge is removed for the nose, apply another piece of tape to secure the glasses to the head.

If a sunglass lens is broken or lost, the above technique can be used over the existing frame (Fig. 21-48).

Pinhole tape glasses can improve vision in a myopic person whose corrective lenses have been lost. With myopia, parallel light rays from distant objects focus in front of the retina. The pinhole directs entering light to the center of the cornea, where refraction (bending of the light) is unnecessary. Light remains in focus regardless of the refractive error of the eye (Fig. 21-49). Pinhole glasses decrease both illumination and field of vision, so puncture a piece of duct tape or cardboard repeatedly with a safety pin, needle, fork, or other sharp object until enough light can enter to focus on distant objects. Secure the device to the face (Fig. 21-50).

A

B

Figure 21-48. Improvised lens for sunglasses (see text).

IMPROVISED TRANSPORT

Carries

Two-Hand Seat*

Two carriers stand side by side. Each carrier grasps the other carrier's wrists with opposite hands (e.g., right to left). The victim sits on the rescuers' joined forearms. The carriers each maintain one free hand to place behind the back of the victim for support (support hands can be joined). This system places great stress on the carriers' forearms and wrists.

Four-Hand Seat

Two carriers stand side by side. Each carrier grasps his or her own right forearm with the left hand, palms facing down. Each

*Both the two-hand seat and the four-hand seat are useful only for very short carries over gentle terrain.

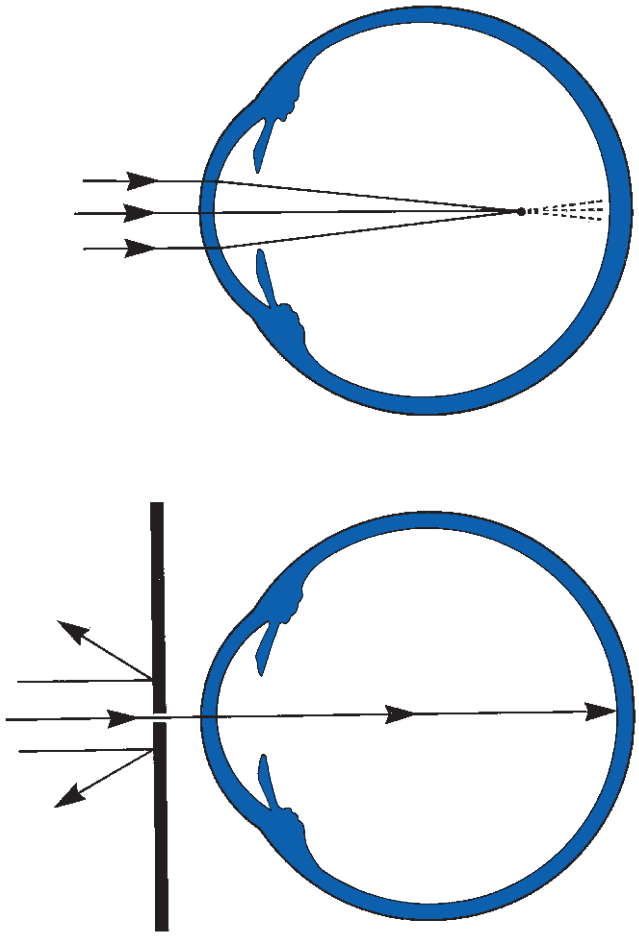


Figure 21-49. Pinhole in cardboard to improve vision in person with myopia.

Figure 21-50. Pinholes in duct tape to improve vision in person with myopia.

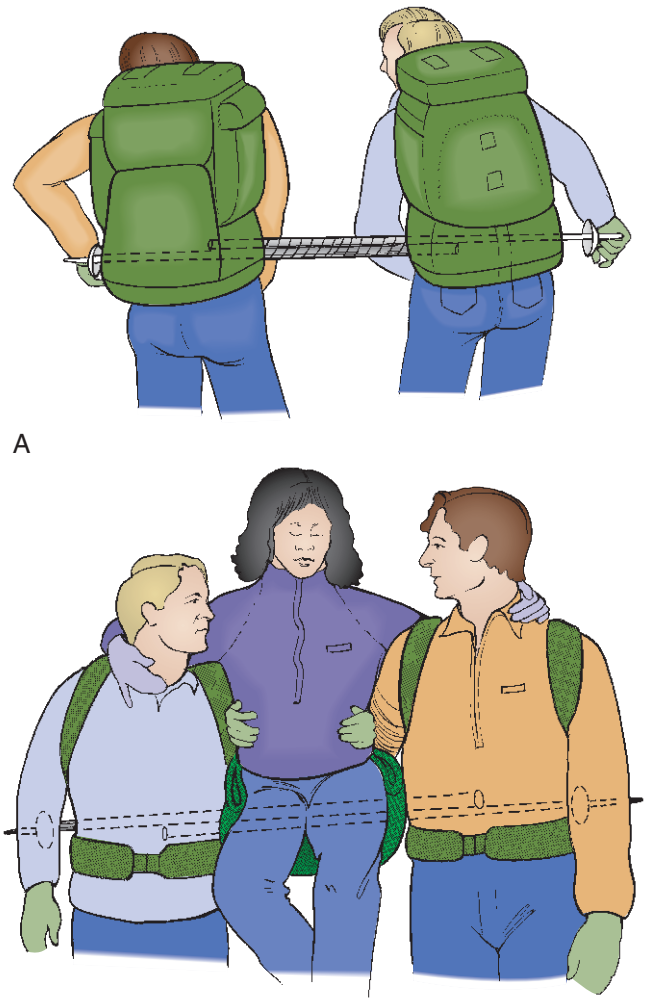


Figure 21-51. Ski pole seat. **A,** Ski poles are anchored by the packs. **B,** The victim is supported by the rescuers.

carrier then grasps the forearm of the other with his or her free hand to form a square "forearm" seat. With the forearm seat, the victim must support himself or herself with a hand around the rescuers' backs.

Ski Pole or Ice Ax Carry

Two carriers with backpacks stand side by side with four ski poles or joined ice ax shafts resting between them and the base of the pack straps (Fig. 21-51). The ski poles or ice ax shafts can be joined with cable ties, adhesive tape, duct tape, wire, or cord. Because the rescuers must walk side by side, this technique requires wide-open, gentle terrain. The victim sits on the padded poles or shaft with his or her arms over the carriers' shoulders.

Split-Coil Seat ("Tragsitz")

The split-coil seat transport uses a coiled climbing rope to join the rescuer and victim together in a piggyback fashion (Fig. 21-52). The victim must be able to support himself or herself to avoid falling back, or must be tied in.

Figure 21-52. Split-coil seat. **A,** Rope coil is split. **B,** Victim climbs through rope. **C,** Rescuer hoists the sitting victim.

Two-Rescuer Split-Coil Seat

The two-rescuer split-coil seat is essentially the same as the split-coil Tragsitz transport, except that two rescuers split the coil over their shoulders. The victim sits on the low point of the rope between the rescuers (Fig. 21-53). Each rescuer maintains a free hand to help support the victim.

Backpack Carry

A large backpack is modified by cutting leg holes at the base. The victim sits in it as would a child in a baby carrier. Some large internal frame packs incorporate a sleeping bag compartment in the lower portion of the pack that includes a compression panel. With this style of pack, the victim can sit on the suspended panel and place his or her legs through the unzipped lower section without damaging the pack, or the victim can simply sit on the internal sleeping bag compression panel without the need to cut holes.

Nylon Webbing Carry

Nylon webbing can be used to attach the victim to the rescuer like a backpack (Fig. 21-54). At least 15 to 20 feet (4.6 to 6.1 m) of nylon webbing is needed to construct this transport. The center of the webbing is placed behind the victim and brought forward under the armpits. The webbing is then crossed and brought over the rescuer's shoulders, then down around the victim's thighs. The webbing is finally brought forward and tied around the rescuer's waist. Additional padding is needed for this system, especially around the posterior thighs of the victim.

Three-Person Wheelbarrow Carry

This system is extremely efficient and can be used for prolonged periods on relatively rough terrain. The victim places his or her arms over two rescuers' shoulders (the rescuers stand side by side). The victim's legs are then placed over a third rescuer's shoulders. This system equalizes the weight of the victim very efficiently.

Litters (Nonrigid)

Many nonrigid litter systems have been developed over the years. These systems are best suited for transporting non–critically injured victims over moderate terrain. They should

Figure 21-53. Two-rescuer split-coil seat. Balance could be improved by using a longer coil to carry the victim lower.

Figure 21-54. Webbing carry. Webbing crisscrosses in front of the victim's chest before passing over the shoulders of the rescuer.

never be used for trauma victims with potential spine injuries.

Blanket Litter

A simple nonrigid litter can be fabricated from two rigid poles, branches, or skis and a large blanket or tarp. The blanket or tarp is wrapped around the skis or poles as many times as possible and the poles are carried. The blanket or tarp should not be simply draped over the poles. For easier carrying, the poles can be rigged to the base of backpacks. Large external frame packs work best, but internal frame packs can be rigged to do the job. Alternatively, a padded harness to support the litter can be made from a single piece of webbing, in a design similar to a nylon webbing carry.

Tree Pole Litter

The tree pole litter is similar to the blanket litter described previously. In the tree pole litter, instead of a blanket or a tarp, the side poles are laced together with webbing or rope and then padded. Again, the poles may be fitted through pack frames to aid carrying. To give this litter more stability and to add tension to the lacing, the rescuer should fabricate a rectangle with rigid cross-bars at both ends before lacing.

Parka Litter

Two or more parkas can be used to form a litter (Fig. 21-55). Skis or branches are slipped through the sleeves of heavy parkas, and the parkas are zipped shut with the sleeves inside. Ski edges should be taped first to prevent them from tearing through the parkas.

Internal Frame Pack Litter

The internal frame pack litter is constructed from two to three full-size internal frame backpacks, which must have lateral compression straps (day packs are suboptimal). Slide poles or skis through the compression straps; the packs then act as a support surface for the victim.

Life Jacket Litter

Life jackets can be placed over paddles or oars to create a makeshift nonrigid litter.

Rope Litter

On mountaineering trips, the classic rope litter can be used, but this system offers little back support and should never be used for victims with suspected spine injuries. The rope is uncoiled and staked onto the ground with sixteen 180-degree bends (eight on each side of the rope center). The rope bends should approximate the size of the finished litter. The free rope ends are then used to clove hitch off each bend (leaving 2 inches [5 cm] of bend to the outside of each clove hitch). The leftover rope is threaded through the loops at the outside of each clove hitch. This gives the rescuers a continuous handhold and protects the bends from slipping through the clove hitches. The rope ends are then tied off (Fig. 21-56). The litter is padded with packs, Therm-a-Rest pads, or foam pads. This improvised

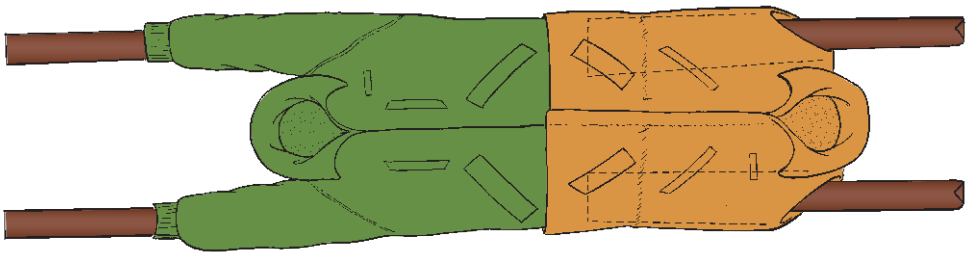


Figure 21-55. Parka litter. On the right, the sleeves are zipped inside to reinforce the litter.

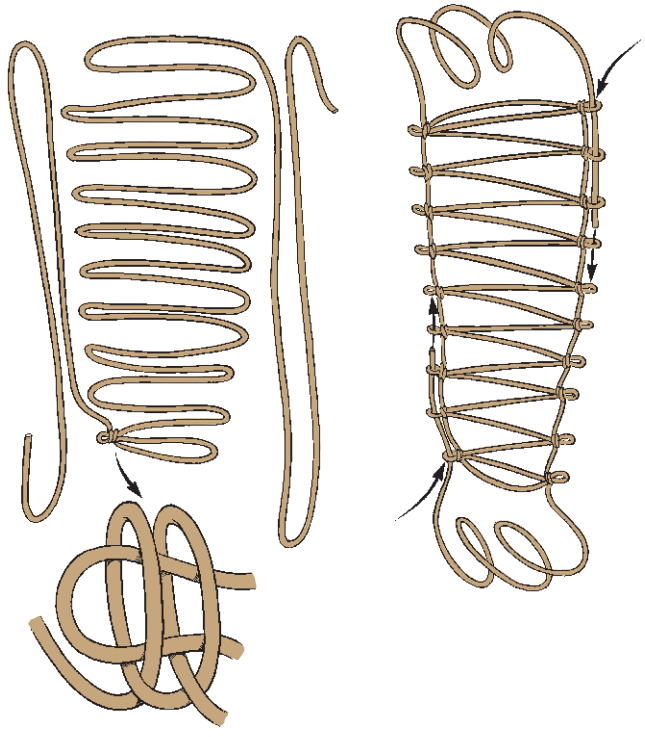


Figure 21-56. Rope litter (see text).

litter is somewhat ungainly and requires six or more rescuers for an evacuation of any distance. A rope litter can be tied to poles or skis to add lateral stability if needed.

Improvised Rescue Sled or Toboggan

A sled or toboggan can be constructed from one or more pairs of skis and poles that are lashed, wired, or screwed together. Many designs are possible. Improvised rescue sleds may be clumsy and often bog down hopelessly in deep snow. Nonetheless, they can be useful for transporting a victim over short distances (to a more sheltered camp or to a more appropriate landing zone). They have sometimes been used for more extensive transports, but they do not perform as well as commercial rescue sleds.

To build an improvised rescue sled or toboggan, the rescuer needs a pair of skis (preferably the victim's) and two pairs of ski poles; three 2-foot-long (61-cm) sticks (or ski pole sections); 80 feet (24.4 m) of nylon cord; and extra lengths of rope for sled hauling.

The skis are placed 2 feet (0.6 m) apart. The first stick is used as the front cross-bar and is lashed to the ski tips. Alternatively, holes can be drilled into the stick and ski tips with an awl, and bolts can be used to fasten them together. The middle stick is lashed to the bindings. One pair of ski poles is placed over the cross-bars (baskets over the ski tips) and lashed down. The second set of poles is lashed to the middle stick with baskets facing back toward the tails. A third rear stick is placed on the tails of the skis and lashed to the poles. The lashings are not wrapped around the skis; the cross-bar simply sits on the tails of the skis under the weight of the victim. Nylon cord is then woven back and forth across the horizontal ski poles. The hauling ropes are passed through the baskets on the front of the sled. The ropes are then brought around the middle cross-bar and back to the front cross-bar. This rigging system reverses the direction of pull on the front cross-bar, making it less likely to slip off the ski tips.[38]

Another sled design utilizes a predrilled snow shovel incorporated into the front of the sled. A rigid backpack frame can also be used to reinforce the sled. This requires drilling holes into the ski tips and carrying a predrilled shovel. This system holds the skis in a wedge position and may offer slightly greater durability.[34]

A FINAL NOTE

Under certain conditions, improvised systems are entirely suboptimal and may not meet standard-of-care criteria. It would, for example, be ill advised to fabricate a litter for transporting a victim with a suspected spine injury when professional rescue is only a few miles away. An improvised litter system might be entirely appropriate, however, if the injured person is 40 miles out and needs transport to a sheltered camp or potential helicopter landing zone. The context of the situation should be considered. At times, persons are obligated to do whatever they can, and a resourceful approach to problem solving combined with a little ingenuity could save a victim's life.

The references for this chapter can be found on the accompanying DVD-ROM.

Hunting and Other Weapons Injuries

22

Edward J. Otten and David G. Mohler

Even as Nimrod the mighty hunter before the Lord.
Genesis 10:9

Anthropologists have many theories concerning the origins and importance of hunting in the evolution of the human species. The physical attributes of bipedal locomotion, binocular vision, and an opposable thumb all make humans more efficient hunters. Whether these exist because humans have an innate compulsion to hunt or whether humans are hunters because of these traits is debatable. There is no debate, however, that human social evolution, language, the use of tools, and domestication of animals are directly related to more efficient hunting. In a survival situation, and in some ways with regard to evolution, hunter–gatherer animals have a distinct advantage over strictly vegetarian animals because of the relative food value of meat over plants. Hunters tend to be males. Approximately three fourths of all calories in modern hunter–gatherer groups are derived from plants, and this portion of the food is usually supplied by the women in the group. Even in Eskimo tribes where plants make up little of the diet, the women do most of the fishing while the men hunt.

Hominids were at a disadvantage, even in groups, when hunting large animals or driving off other predators from their kills until they began using stones, long bones, and sticks to enhance their relatively weak teeth and claws. Implements for hunting and skinning animals were the earliest tools found by anthropologists. Human cultural evolution followed closely the technological changes in weapons, although sports, business, and war had replaced the need for hunting in most cultures even by the time Nimrod walked the earth. Bows and arrows, slings, spear throwers, nets, harpoons, traps, and firearms were designed to extend the reach and increase the lethality of the human hand. Unfortunately, humans discovered that they could kill each other with these weapons. Since the discovery of gunpowder, the development of weapons technology has surpassed all other forms of human endeavor, including medicine and transportation.[6,7,15]

Only a few cultures still depend on hunting as their primary food-gathering method. Examples are the Mbuti tribe of the Ituri Forest in Zaire, Andaman Islanders in the Bay of Bengal, and Eskimos. Many cultures, however, use hunting to supplement agriculture, plant gathering, or raising livestock. Most hunting in the United States is done for sport or pleasure, although in some areas of the country hunting and trapping are still the primary source of income for a few people.

HUNTING IN THE UNITED STATES

The total number of hunters and trappers is unknown. Some participate illegally and are not licensed. In 2003, throughout the United States, 14.7 million individuals purchased hunting licenses at a cost of $679.8 million. Although hunting seasons are regulated and relatively short, hunters spent 16 million visitor-days in the national forests.

The North American Association of Hunter Safety Coordinators, a division of the New York State Office of Wildlife Management, reported 860 fatal hunting injuries in the United States during the 4-year period of 1983 to 1986, with a total of 6992 injuries from firearms.[35a] Interestingly, 34% of the total injuries and 89% of the handgun injuries were self-inflicted. Shotguns accounted for 106 of the fatalities and 906 of the total injuries, whereas rifles accounted for 79 fatalities and 465 injuries. The New York State Department of Environmental Conservation reported that the average number of hunting injuries decreased from an average of 137 per year in the decade of the 1960s to only 48 in 2001 and 37 in 2002.[24a] They credit the institution of hunter safety programs in 1960. In 2001 Colorado reported nine injuries and one death per 500,000 licensed hunters.[12a] Michigan reported 2 deaths per 2,665,952 hunters in 2003, making hunting one of the lowest injury and fatality rates of any recreational activity.[47a] The type of hunting also influences the rate of injury. Smith and colleagues reviewed 1345 hunting injuries in Pennsylvania from 1987 to 1999.[38] They showed that turkey hunters had the highest rate of injury (7.5 per 100,000 hunters) and grouse hunters the lowest (1.9 per 100,000 hunters). This was attributed to turkey hunters not wearing hunter orange clothing. Deer hunters had the highest case-fatality ratio at 10.3%, and pheasant hunters the lowest at 1.3%. This higher fatality rate was largely because most deer hunting injuries were due to wounds caused by rifle bullets. They also noted that younger hunters suffered the highest rate of injuries, and the largest percentage of incidents occurred on opening day. Hunting-related shootings represent a very small portion of the total number of accidental firearm deaths in the United States. Of 131 unintentional firearm deaths in California from 1977 to 1983, only eight were the result of hunting accidents.[8,12,27,35,44,47]

Hunting injury data may be inaccurate for a number of reasons. Many minor nonfatal injuries may go unreported, and

most states do not differentiate accidental firearm hunting deaths from deaths that occur during any other activity. Also, automobile and all-terrain vehicle accidents that occur while hunting, or gunshot wounds inflicted while "cleaning a gun" at home, may be classified as hunting or nonhunting injuries.

Types of Injuries Encountered

Most injuries to hunters are the same types of injury seen in backpackers, fishermen, and climbers. Frostbite, sprains, burns, and fractures occur with the same frequency in hunters as in others who visit wilderness areas. Prolonged extraction times may increase the risks of hypothermia, wound infection, dehydration, missed medications, and other time-dependent secondary complications.

Injuries that are unique to hunters are those caused by their weapons. Most hunting is done with firearms. Shotguns and rifles are more commonly used, although handguns are increasing in popularity. Use of bows and crossbows in hunting is also rapidly increasing. Hunters using these weapons frequently are permitted an extended hunting season that does not overlap with periods for rifle and shotgun hunting. Hunters who use bows and crossbows pose far less danger to people in the hunting area at long range compared with rifles and shotguns. Bow hunting requires more skill, use of camouflage, and stealth because of the short effective ranges of arrows and bolts. These factors place bow hunters at greater risk for being mistaken for a game animal at long ranges, which is why rifle and shotgun seasons rarely run concurrently with bow-hunting activity.

Other weapons are used for hunting but are less likely to be encountered. For example, spears, harpoons, and nets are used by some hunters in the Arctic, Australia, and Africa. Spear injuries from gas-powered spearguns or rubber-band powered Hawaiian slings have been associated with fatal injuries, especially when occurring in ocean or lake environments where secondary drowning or shark attack may be an additional hazard. Harpoon and fishing spearheads may separate from the shaft and, depending on the force used, may penetrate the skull or a body cavity. Slingshots are rubber-band–powered devices that use the energy in a stretched piece of rubber to hurl a projectile, often a small rock or ball bearing, at 200 to 300 feet (61 to 91 m) per second. Although this is considered a low-velocity and thus low-energy projectile, injuries to the head and face, especially the eyes, have been reported. Blowguns, while mainly used by aboriginal hunters, have become popular with some recreational hunters of birds and small game. The blowgun varies in length, and a variety of darts can be projected 20 to 50 feet (6 to 15 m) by the exhaled breath. The darts have low energy and do not penetrate very deeply. Modern blowguns rarely cause serious injury unless striking the eye or possibly a blood vessel. To effectively kill small game, the darts generally must carry an immobilizing or poisonous toxin. Darts used by some tribes contain toxins such as curare or batrachotoxin, which can be fatal to humans.

Trap injuries may be included in the definition of hunting injuries. Most traps are designed to catch and hold small game. Injuries usually occur when a trapper triggers a spring-loaded trap prematurely. Crush injuries and puncture wounds to the hands are most common. Hikers occasionally tread on unmarked traps, and domestic animals such as dogs are accidentally caught in poachers' traps. Another problem with traps occurs when an animal (wild or domestic) is caught in a trap and attacks the trapper while being released.

Figure 22-1. The wrong way to use a tree stand. This hunter is not wearing a safety harness, is drinking alcohol, and is pulling his firearm into the tree stand with the muzzle pointing upward.

Many knife lacerations occur when hunters clean game. Lack of familiarity with the process or techniques for field dressing and cleaning game is the likely cause. Failing to wear protective gloves; using the wrong type of knife; working with bloody, slippery material; and having cold hands all contribute to accidents.

Tree Stand Injuries

A frequent preventable cause of serious injury and death among hunters is not associated with firearms at all. It is the tree stand injury. Tree stands are small platforms designed to hold hunters high above the ground so they can more easily spot and kill large game while remaining undetected. Whether homemade or of commercial design, the platforms generally are small, portable devices that the hunter attaches to the trunk of a tree near game trails or water holes. The stands may have attached ropes or ladders for access, or the hunter may free-climb the tree for placement of the stand or fasten small climbing steps on the tree (Figs. 22-1 and 22-2). Hunters may fall asleep on the platforms and fall off, or fall while climbing up or down trees. At least half of these injuries could be prevented if all

Figure 22-2. A commercially produced tree stand can be used to climb the tree and obviates the need for a ladder or steps, which are the cause of many falls.

Figure 22-3. The correct way to bring a firearm or quiver into the tree stand, with the muzzle or arrowheads pointing down and the hunter wearing a safety harness at all times.

hunters wore tree stand safety harnesses (Fig. 22-3). Although most of the injuries are similar to those seen with any type of fall, occasionally a hunter drops a firearm, which discharges, or falls on an arrow or rifle, causing an additional weapons injury. Over 10 years, injuries of this type in Georgia accounted for 36% of reported hunting injuries and 20% of hunting fatalities.[9] A study from the University of Rochester, New York, looked at tree-stand injuries from 1996 to 2001.[34] The authors noted that 51 injuries occurred, all in men with a mean age of 42. Alcohol was present in 10% of patients and 2 of 3 deaths. Spinal fractures were the most common injury (51%), followed by extremity (41%), head (24%) and lung injuries (22%). Only two patients had been using a safety belt (4%). Sixteen spinal cord injuries were reported from 1987 to 1999 in Oklahoma; the mean height of fall was 16 feet, and 18% were related to alcohol ingestion.[41] Ninety percent resulted in paraplegia/paresis, and 12.5% were fatal.[34,41,43]

Arrow Injuries

Modern arrows are usually made from aluminum, graphite, or fiberglass, although many beginners still use inexpensive wooden arrows. A number of types of arrowhead are in use, such as field points and target points, but most injuries are due to specially designed hunting arrowheads called broadheads. These razor-sharp metal points come in a variety of sizes and shapes and are designed to kill game by lacerating tissue and blood vessels, causing bleeding and shock. Unlike hunting firearms projectiles, which are designed to kill quickly through massive tissue damage and rapid incapacitating hemorrhage, arrows usually kill more slowly with less tissue damage (Fig. 22-4).[4,22,24] Arrows are propelled by a conventional bow, which may be straight, recurved, or compound, or by a crossbow. Crossbow projectiles may be called arrows or *bolts* and generally are shorter and heavier than arrows fired from a bow. The force used to propel the arrow is usually measured in *draw weight,* which is the number of foot-pounds necessary to draw a 28-inch (71.1 cm) arrow to its full length. The higher the pound draw, the more powerful the bow and the deeper the penetration the same type of arrow will have.

Arrows have a much shorter range than bullets do, and arrows must be more accurately placed to kill the animal quickly; therefore, most shots are taken under 164 feet (50 m).

Figure 22-4. Types of arrows. *Top,* Aluminum shaft arrow with hunting broadhead. *Middle (left to right),* Four field points of various weights: two types of broadheads and small game blunt hunting head with spring claws to prevent arrow loss from burrowing into the ground. *Bottom,* Fiberglass shaft for interchangeable heads.

Because brush and tree branches can easily deflect an arrow, most shots are taken with a clear field of view. For these reasons, bow hunters rarely mistakenly shoot another hunter they presumed was a game animal. Most arrow injuries occur when hunters fire illegally at night in heavy brush and are not sure of their target. Another common injury occurs when a hunter runs after a wounded animal and falls on an arrow that was to be used for a second shot or falls out of a tree stand onto an arrow. A loaded crossbow is similar to a loaded gun. Hunters have been accidentally shot when dropping the weapon or snagging the trigger on a branch or fence. Hunting arrowheads are quite sharp; self-inflicted injuries may occur when a hunter is sharpening the blades of the broadhead or returning an arrow to the quiver.

Injuries from Firearms

Nonpowder Firearms. Although the word *firearms* technically defines guns that fire projectiles by ignition and burning of a propellant, similar designs referred to as "nonpowder" firearms using springs, compressed air, or compressed gas cartridges are in widespread use among sportsmen and children, and will be considered as firearms in practical use.

Whereas traditional firearms discharge a projectile by the contained expanding gases generated in the gun barrel by modern fast-burning powders or old-fashioned black powder, nonpowder firearms use a spring, compressed air, or a carbon dioxide cartridge to accelerate the projectile out the barrel. Although air guns are quite accurate at short distances and can develop muzzle velocities in excess of 1200 feet per second, the small lightweight projectiles cannot usually penetrate skin at distances greater than 328 feet (100 m). Nonpowder firearms are commonly used by children, who cannot legally obtain or use other types of firearms. Uninformed parents buy them as toys, erroneously believing them to be harmless by design. Without supervision and proper training in gun safety, severe injury and death can result. The wounds they cause can be lethal, especially from high-powered air rifles, which can send out pointed projectiles at sufficiently high velocities to penetrate the skull and body cavities. In a recent technical report, Laraque and colleagues estimated 21,840 injuries in 2000 from nonpowder firearms, with a 4% hospitalization rate.[28] There were 39 resultant deaths between 1990 and 2000. Care must be taken not to trivialize these injuries, especially in the pediatric patient where softer, thinner bone may lead to deep penetration of even lightweight projectiles.

Powder Firearms. In older style weapons *(black powder weapons),* gunpowder is loaded directly into the barrel. In modern weapons, gunpowder is contained in a cartridge.

Black powder weapons use a centuries-old slow-burning propellant that is ignited with a spark from flint striking steel or a percussion cap. The firearms are usually single shot and are loaded from the muzzle by pouring a measured amount of black powder down the barrel, and then inserting the projectile and tamping it down onto the powder charge. When ignited, the propellant is converted to a gas that expands and pushes the projectile out of the barrel of the weapon. With modern design and manufacturing techniques, these weapons are sufficiently accurate to hunt large game, such as deer and elk. The injuries from black powder weapons are similar to those from modern weapons and are discussed later. The same precautions should be used when hunting with or shooting any type of firearm, whether the propellant is air or gunpowder.[29,33,46]

The term *cartridge* is used to refer to the intact, unfired assembly of projectile and propellant loaded into the gun for firing. Rifle and pistol cartridges consist of a metal case that contains the gunpowder propellant and into which the bullet is seated and held by compressing the case around the bullet base at the time of manufacture. The base of the case contains a small metal primer filled with a small amount of high explosive that serves to ignite the fast-burning propellant when it is struck by the firing pin of the gun. The primer is in the center of the base of the case (*center-fire* ammunition) in all cartridges except in small-caliber .22 cartridges, where it is incorporated into the entire circumference of the cartridge base rim (known as *rimfire* ammunition). Rifle and pistol cartridges generally contain a single bullet, although some may be loaded with very small shot to increase the probability of hitting small objects at short distances. Shotgun cartridges consist of a center-fire metal base combined with a paper or plastic shell in the form of a closed-end tube. Within this tube is placed the propellant and then the projectile(s) along with associated plastic, cotton, or paper materials collectively referred to as *wadding.*

Shotgun projectiles consist of shot ranging in size from 1 mm to 10 mm (Fig. 22-5) or a single solid projectile known as a *slug.* Shot pellets used to be made of lead. Because of high lead levels in ducks and geese who ingested spent shot while feeding, lead shot for bird hunting was banned in 1991. Approved shot may be made of steel, tin, or various mixtures of tin, bismuth, and up to 15% iron. Steel shot can be identified on radiographs because it retains a perfect round shape, whereas lead and tin shot deforms inside the barrel during firing, resulting in nonspherical shapes. Determining the shot type can help clinicians decide about the safety and utility of MRI scans in the setting of steel projectiles, or the risk of lead toxicity.

The wadding is commonly a single plastic cup with a thickened expandable base designed to contain the shot and serve as a seal inside the barrel to contain the expanding gases behind the shot cup for maximal muzzle velocity. Slugs also have a type of wadding known as a *sabot,* which surrounds the slug inside the barrel of the shotgun. In all cases, the wadding is fired from

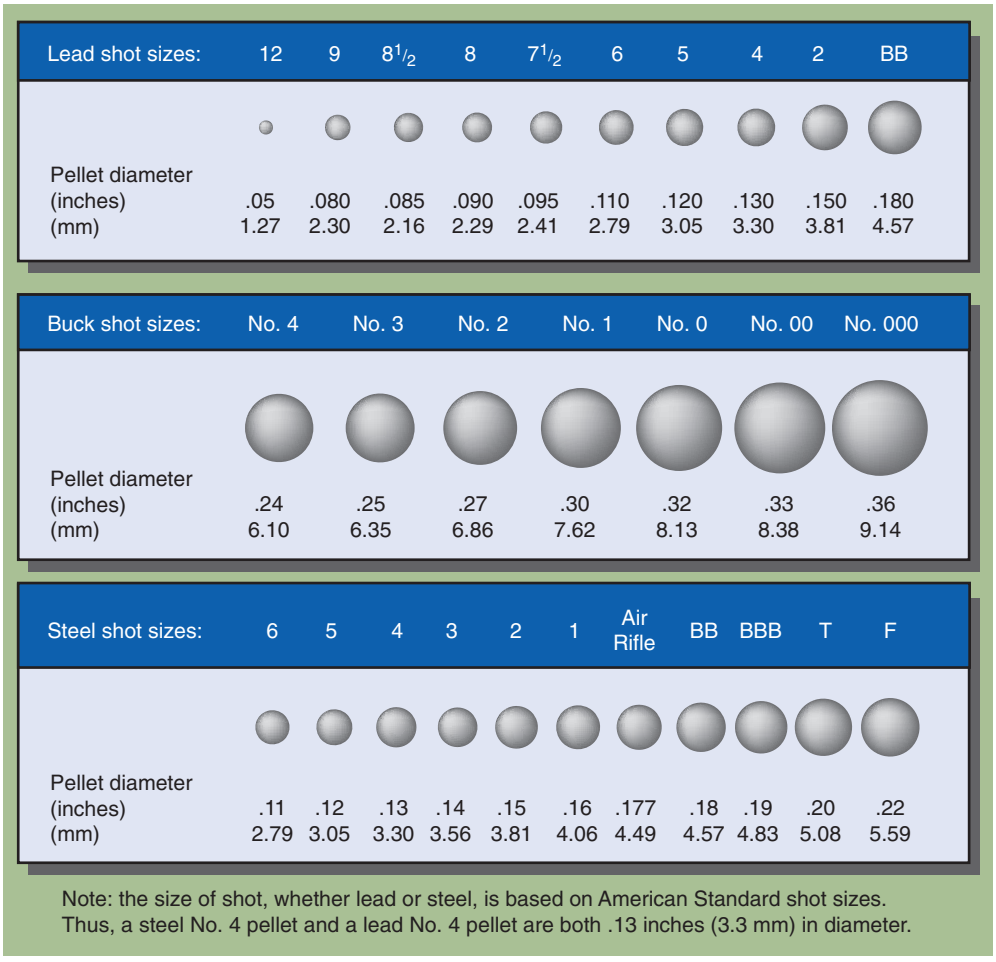


Figure 22-5. Standard shot size number and letter system (with corresponding metric measurements) of hunting shotgun shell projectiles. The smaller the shot size, the more pellets loaded in a single shotgun shell. Larger pellets are heavier, lose less velocity per unit of flight time, and penetrate more deeply than smaller pellets. (From Shotgunworld.com. Used with permission.)

the gun and immediately peels away from the slug or shot. Wadding is commonly found inside close-range wound channels but generally is not involved in wounds at ranges more than 16 to 23 feet (5 to 7 m).

Besides the wadding and projectile(s), hot gas and unburned powder also exit the muzzle. In cases of close-proximity wounds, usually under $3^1/_2$ feet (~1 m), powder stippling may appear on clothing or skin. The presence of wadding in a wound or powder stippling may have important forensic applications and should always be noted. With contact wounds where the muzzle is pressed into the skin at the time of firing, the escaping hot gases may enter the wound channel and expand inside the victim, causing burning, organ damage, bursting of the skin, and a stellate laceration around the point of entrance. Figures 22-6 to 22-8 show examples of gunshot wounds.

Hundreds of types of cartridges are available for firearms. They may be factory loaded or hand loaded, which adds the variables of propellant amount and type. Rifle and pistol cartridges are initially classified according to caliber, or diameter, of the bullet. For example, .22 caliber means the diameter of the bullet is 0.22 inch (5.6 mm); .45 caliber is 0.45 inch; and so forth. The caliber may be expressed in metric measurement; for

Figure 22-6. Gunshot wound to the face and mandible showing extensive bone and soft tissue injury. Patient was initially able to protect his airway, but later required endotracheal intubation because of edema and bleeding.

Figure 22-7. Shotgun wound to the upper arm. Initially, the wound looked benign. Both entrance and exit can be seen.

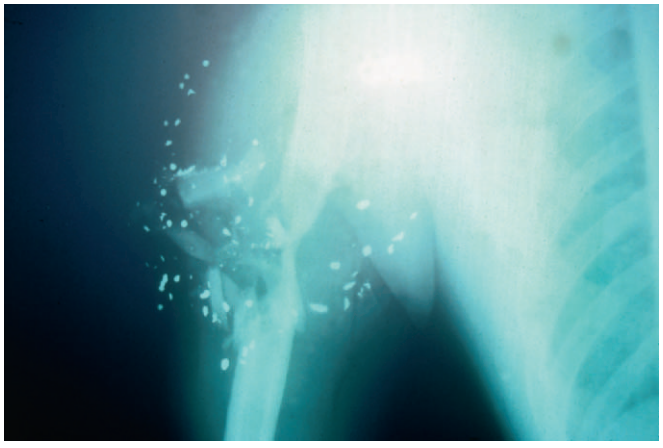


Figure 22-8. Radiograph of the same shotgun wound to the upper arm as seen in Figure 22-7. Extensive bone and soft tissue injury, as well as vascular and nerve damage, can be seen.

example, a 9-mm bullet is 9 mm in diameter, which also happens to be 0.357 inch. However, bullet diameter alone is insufficient to name a cartridge, because bullet length can vary, as do cartridge length and width. The U.S. Army M-16 service rifle fires a .22 caliber bullet weighing 62 grains at 3100 feet (945 m) per second. The common .22 rimfire rifle used by generations of young shooters fires a .22-caliber bullet weighing 40 grains at 1100 feet (335.3 m) per second initial velocity. Obviously, the wounding potential of these two projectiles is vastly different. Nomenclature for a particular cartridge is made more specific by including a measurement of cartridge length, name of the inventor or inventing company, amount of powder in the case, length of the entire cartridge, or the year the cartridge was invented. For instance, the M-16 round is generally referred to as a 5.56 × 45 mm (metric), or a .223 cartridge (English system of measurement), with the third digit differentiating it from the common low power .22 rimfire cartridge. Other common hunting cartridge names illustrating these variations are .45/70 (70 grains of powder), .30-06 (adopted in 1906), and .35 Whelen (the man who developed the round).

Figure 22-9. Examples of hunting bullets. *Left to right,* .50 caliber black powder lead bullet, .22 caliber lead bullet, .22 caliber long rifle lead bullet, .44 magnum semijacketed hollowpoint bullet, .44 magnum shotshell, .223 caliber (5.56 mm) full metal jacket bullet, .22/250 caliber semijacketed soft point bullet, .30/30 caliber soft point flat nose bullet, .270 caliber pointed soft point bullet, and .30-06 caliber round nose soft point bullet.

Figure 22-10. Examples of shotgun rounds. *Left to right,* 12-gauge slug round, empty 12-gauge plastic round, plastic 12-gauge wadding, and number six shotgun pellets.

The recent introduction of the term *magnum* refers more to the type and amount of powder than to the size of the bullet used. Magnum cartridges are designed to give hunters the ability to successfully hunt large game with pistols by improving terminal ballistic performance of the bullet. Figure 22-9 shows some examples of different bullets. Shotgun terminology is a little less complicated, based on the number of lead balls, the diameter of the barrel, and how many lead balls it takes to make a pound. For example, a 12-gauge shotgun has a barrel that is the same diameter as a lead ball that weighs $^{1}/_{12}$ pound; a 20-gauge, $^{1}/_{20}$ pound. The higher the gauge number, the smaller the barrel and the smaller the projectile. The only exception is the .410 shotgun, which is caliber .410 or 0.410 inch in diameter. Figure 22-10 shows some examples of shotgun rounds and the shot and wadding within them.

TABLE 22-1. Comparison of Bullet Caliber, Weight, Velocity, and Muzzle Energy

CALIBER	WEIGHT (GRAINS)	MUZZLE VELOCITY		MUZZLE ENERGY (FOOT-POUNDS)
		Feet/sec	M/sec	
.22	40	1080	329	90
.223	55	3250	991	1280
.44 magnum	180	1600	488	1045
.30/60	150	2750	838	2500

The type and severity of wounds inflicted by a firearm depend on several factors. The most often quoted factor, but the least important, is the amount of energy the bullet (projectile) has when leaving the firearm. The kinetic energy formula, KE = $^1/_2$ MV^2, can be applied to any moving object and can be used to calculate the muzzle energy for a particular type of firearm. Energy increases much more as a function of the velocity of the bullet than as a function of the mass. For this reason, most firearms are classified according to muzzle velocity. The higher the velocity of the bullet, the greater the energy and the greater the potential for injury. Firearms with muzzle velocities greater than 2500 feet (762 m) per second are considered high velocity, 1500 to 2500 feet (457–762 m) per second are medium velocity, and less than 1500 feet per second are low velocity (Table 22-1).

Bullets cause damage to tissue by crushing. The energy of a bullet may be transmitted to the tissue in part or in total depending on the surface area the bullet presents to the tissue. Bullets that yaw, expand, or fragment present more surface area than do bullets that stay in one axis and maintain one shape. By international agreement codified in the articles of the Hague Convention IV of 1907, military bullets are not to be designed in a manner to produce "superfluous" wounding effects by features that would encourage the bullets to flatten or expand on impact with tissue. They are typically completely encased in a copper jacket to prevent deformation of the soft lead core. Such rounds generally pass through an individual, leaving a permanent wound tract similar in diameter to that of the bullet. The ammunition is designed to wound a soldier and put him out of combat, but not to kill him. In contrast, hunting ammunition is designed to expand on impact up to 2 or 3 times its diameter, resulting in a much larger wound channel, greater tissue damage and rapid incapacitation and death (Fig. 22-11). This feature of planned deformation also promotes retention of the bullet within the target and reduces the risk of injury to unseen individuals downrange from the game animal. In fact, many states require the use of expanding ammunition for hunting large game, and this may increase the wound severity of hunting injuries compared to military and criminal shootings.

Figure 22-11. The .30-caliber Nosler 180 g Accubond Polymer Tip bullet fired into calibrated 10% ordnance gelatin is typical of the .30 caliber hunting bullets used for .30-06, .308, .300 Win Mag, and other cartridges. This photo shows this bullet fired from a .308 rifle with an 18-inch-long barrel. Muzzle velocity was 2,499 feet (762 m) per second. Penetration depth exceeded 20 inches (51 cm). Temporary cavity maximum width was 4.7 inches (11.99 cm) at a depth between 2.8 inches (7.11 cm) and 8.7 inches (22.1 cm). Diameter of the recovered bullet at the front surface was 0.56 inches. Weight of the recovered bullet was 149.5 g; 17% of the bullet turned into fragments. Terminal performance of this type is suitable for all medium to heavy game encountered in the lower 48 U.S. states, including moose, elk, black bear, pigs, and deer. Ten-cent coin (dime) is shown for comparison. (Courtesy of Gary K. Roberts, DDS.)

In addition to direct tissue destruction by the deforming bullet, fragmentation may occur when a bullet strikes bone and sends bone and bullet fragments in several directions. These secondary missiles cause injuries within the body similar to those from bullet fragments and may even exit the body to injure bystanders. A second injury mechanism of terminal ballistic bullet behavior is *temporary cavitation*, which occurs at all velocities to some degree but becomes a significant wounding mechanism factor only at high velocities. The temporary cavity is created by radial dispersion of tissue by the bullet surface as a result of acceleration of tissue away from its path. A *permanent cavity* occurs when a bullet or fragment crushes tissue. In high-velocity bullet wounds, the temporary cavity may be many times larger than the permanent cavity. This wave is well tolerated by most elastic tissue, such as muscle, bowel, and lung; however, inelastic tissues, such as liver or brain, do not tolerate it and may be severely damaged by the temporary cavity.

Bullet motion in flight is described by *rotation, yaw,* and *pitch*. The rifle grooves cut into the inside of the barrel cause the bullet to spin around its long axis, just like a football thrown in a tight spiral. *Yaw* and *pitch* describe bullet motion left or right, or up and down relative to the long axis of the bullet. In wound ballistics, yaw is used to describe both axes of motion. A rifle bullet will usually yaw after striking any object outside the body such as a tree branch, belt, or clothing and when striking skin and other tissue within the body. Yaw can result in complete end-over-end rotation of the bullet within tissue, causing the bullet to increase the area that is crushed as the entire side profile of the bullet passes through the tissue. Bullets with round ends, for example the .45 automatic Colt pistol bullet or round musket balls, routinely do not yaw, and produce wound channels equal to their caliber or expanded caliber. Yaw causes maximum damage at 90 degrees of rotation, when the entire side profile of the bullet crushes tissue. This factor, combined with any bullet expansion, can cause the exiting bullet to produce a much larger wound than when it entered.

In order to allow laboratory comparison of projectile designs and to study the effect of velocity and expansion, experimental *wounding profiles* have been described using ballistic gelatin, which accurately simulates human muscle tissue. These wounding profiles show the various aspects of potential ballistic injury including the cavitation, yaw, and fragmentation (Figs. 22-12 and 22-13).[13] The total effect of high energy, fragmentation, expansion, yaw, and temporary cavity formation results in tissue injury. Although the kinetic energy formula yields the total energy available to cause injury, the physical behavior of the projectile(s) and the transited tissues are the actual determinants of the complete injury pattern. The type of tissue struck is the most important factor. As can be seen from Table 22-1, the .22-caliber long rifle rimfire bullet has a low mass and velocity and thus a low muzzle energy, yet more fatalities have occurred from this round than from any other. It is very inexpensive, can be fired from a number of rifles and handguns, is commonly used to hunt game, and is not thought of as particularly dangerous by inexperienced hunters. For these reasons, more people are shot by this cartridge than any other single bullet type. The bullet is highly lethal when striking the brain, heart, or major blood vessel.[1,2,16–20,32,42,45] Rubber or plastic bullets, while generally not used for hunting, may be encountered. These bullets travel at about 200 feet (61 m) per second and will not usually penetrate skin, although at short ranges (under 50 feet [15 m]) can cause fractures, eye trauma, and other blunt injuries.[13]

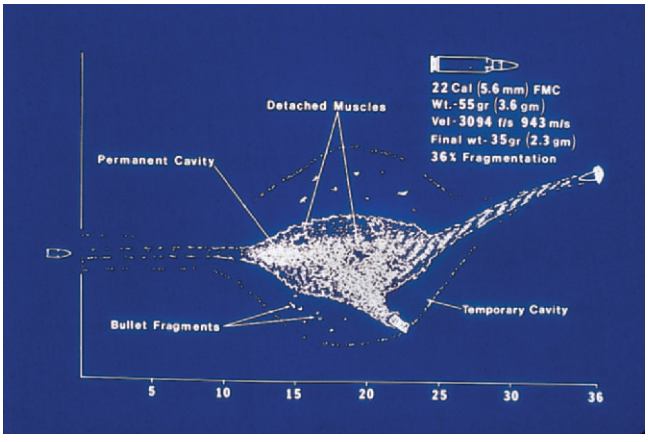


Figure 22-12. Wound profile of a .223 rifle bullet in 10% ballistic gelatin showing the permanent and temporary cavities and the effect of tumbling and fragmentation.

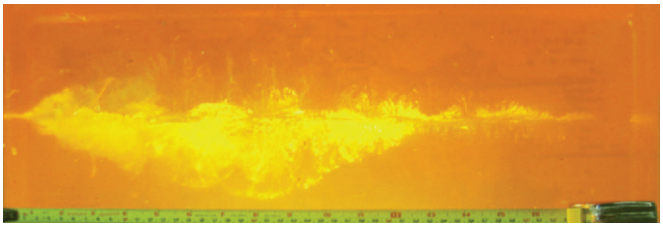


Figure 22-13. The path of a test bullet through ballistic gelatin suggests the amount of tissue damage that a hunting bullet can do inside the human body, even if entrance and exit wounds are small. The data for this bullet are given with Figure 22-11. (Courtesy of Gary K. Roberts, DDS.)

Other rare problems associated with firearms are explosions that occur within the firearm itself. These can cause burns or fragment types of injuries. When firearms are loaded with excessive amounts of powder or when the wrong powder is used in reloading bullets, the resultant detonation may cause the frame or cylinder of the firearm to explode. The burning powder or fragments of metal can cause injuries to the shooter. These injuries usually occur to the face and hands; penetrating eye injuries are also common. Obstruction of the barrel of the firearm by snow, mud, or other foreign material may cause a similar explosion.

Trap Injuries

Traps are designed either to kill animals or to capture them alive and uninjured. The latter type poses no risk to humans unless they should happen upon a trap and attempt to free the animal or otherwise approach the trap. The trapped animal often will bite or claw anyone within range. Leghold traps designed to kill or injure an animal may occasionally cause problems for unwary hikers or campers. These traps have a spring-loaded jaw that closes when triggered by something touching the trigger plate, usually involving only 1 to 2 pounds ($^1/_2$ to 1 kg) of pressure. Most injuries involve the foot, but any area of the body that can fit between the jaws potentially can be injured. The jaws can be released by compressing the spring controlling the jaws (Fig. 22-14). Very large traps used to trap poachers or to catch large animals, such as tigers or bears, cannot easily be released without help. These traps may also be attached to large weights, such as logs or concrete blocks, to prevent escape. Fortunately, most of these large traps are now collector's items and not used in the field.

Many injuries occur when the person setting the trap inadvertently causes the trap to spring before being set in the ground. This often causes hand and finger injuries, especially with amateur trappers unfamiliar with the type of trap (Fig. 22-15). Unconventional traps, such as snares, deadfalls, and pit traps, may rarely be encountered, but the mechanisms and types of injuries are quite variable. Trap guns are illegal in most areas of the world; injuries are similar to gunshot wounds.

Treatment of Hunting Injuries

Treatment of hunting injuries involves standard principles and priorities of trauma care. Airway, breathing, circulation, bleed-

ing control, immobilization of the spine and fractured extremities, wound care, and stabilization of the victim for transport should be performed in an expedient manner. The victim should always be disarmed to prevent accidental injury to the rescuer or further injury to the victim. Removing the firearm or arrow from the vicinity of patient care is usually sufficient, but ideally the firearm should be made safe by removal of the ammunition and opening of the firing chamber. Arrows should be placed in a quiver, or the points may be wrapped in cloth to prevent injury.

Figure 22-14. **A,** A leghold trap set. **B** and **C,** A leghold trap sprung. **D** and **E,** To release a trap that has been sprung, stand on each end of the trap and compress the spring.

Figure 22-15. Spring traps must be set carefully to avoid injury to the person setting the trap.

Figure 22-16. Arrow wound to the left side of the neck near the mandible. The shape of the wound resembles the blades of the broadhead as shown in Figure 22-4.

The management of common traumatic injuries and illnesses, such as hypothermia and mountain sickness, is no different except for one important point: always disarm the victim. A victim with a charged weapon and a head injury or change in mental status for any reason presents an immediate danger to a well-meaning rescuer. If the person attempting to offer aid to an injured hunter is not familiar with weapons, it is usually best to move the weapon several feet from the victim and point it in a direction where an accidental discharge will do the least harm.

Arrow Injuries

Lacerations from razor-sharp hunting points are not unusual and can be treated like any similar laceration. The wound should be irrigated, any foreign material removed, and the laceration closed primarily. Victims pierced by an arrow should be stabilized, and the arrow should be left in place during transport, if possible. Attempts to remove the arrow by pulling it out or pushing it through the wound may cause significantly more injury and should be avoided. It is acceptable to cut off the shaft of the arrow and leave 3 or 4 inches (8 to 10 cm) protruding from the wound to make transport easier if this can be accomplished with a minimum of arrow movement. A large pair of paramedic-type shears can usually cut through an arrow shaft if it is stabilized during cutting. The portion of the arrow that remains in the wound should be fixed with gauze pads or cloth and tape. A similar approach should be used for spears and knives. The victim should be transferred as quickly as possible to an operating room, where the arrow can be removed under controlled conditions. Radiographs are helpful to identify associated anatomic structures before removal is attempted in the operating room (Fig. 22-16).

Gunshot Wounds

Myths about Gunshot Wounds. Many myths associated with the management of gunshot wounds should be repudiated.

Myth 1: The size or caliber of the bullet can be determined by the size of the wound. In truth, the skin is quite elastic and has high tensile strength. Although a knife or arrow can cut the skin, a blunt bullet must crush the tissue by stretching. This stretching occurs until the bullet passes through and then retracts, causing the wound in the skin to be smaller than the caliber of the bullet.

Myth 2: The size of the wound determines whether it is an exit or entrance wound. Actually, the bullet usually tumbles or yaws after striking the skin and soft tissue; if the bullet exits while still tumbling, the exit wound may be larger than the entrance. This commonly occurs when a missile strikes an arm or leg where the bullet is in mid-tumble at 90 degrees when exiting. Often the bullet fragments, and only a small portion of the bullet exits, making the exit wound much smaller than the entrance. In addition, pieces of bone or tooth may exit, causing an odd-size wound.

Myth 3: The path of the bullet can be determined by connecting the entrance and exit wounds. This myth may lead to inappropriate intervention in gunshot wounds. A case example: A gunshot-wound victim had two wounds about 6 inches (15 cm) apart on his upper thigh, and they were initially thought to be an entrance and exit wound. The patient developed abdominal pain, and on chest x-ray it was noted that there were two bullets in the left chest. Subsequent surgical intervention revealed injury to the colon, spleen, bowel, stomach, diaphragm, lung, and subclavian vessels. So what appeared to be entrance and exit wounds were actually two gunshot wounds sustained while the patient was lying supine.

The outcome of most gunshot and blast wounds depends primarily on the body part that has been injured, and secondarily on the environment in which the injury occurred and the quality and timeliness of medical intervention. While knowing the type of weapon and the physics of ballistics and blast can help predict the extent of physical damage, there is no substitute for attention to detail when examining the patient.

Emergency Department Care. Emergency department care of the gunshot wound includes securing the airway, placing two intravenous lines in unaffected extremities, performing cardiac monitoring, and providing oxygen therapy. The patient with a neck wound and expanding hematoma should be endotracheally intubated as soon as possible. If endotracheal intubation is not possible, a needle cricothyrotomy followed by a tube

cricothyrotomy should be performed. Relief of tension pneumothorax with a needle or tube thoracostomy or occlusion of a sucking chest wound should be done immediately. Any external bleeding should be controlled by direct pressure. In a wilderness situation, a tourniquet may be the best means of controlling significant bleeding in a manner that is less labor intensive.

A radiograph should be obtained of the involved area, and where there is a presumed entrance wound without an exit wound, multiple x-ray studies may be needed to find the location of the bullet. On rare occasion, bullets have been observed to embolize from the chest area via the aorta to the lower extremity arteries or to the heart via the vena cava. A type and crossmatch and basic trauma laboratory tests should be performed. Tetanus toxoid and immunoglobulin should be administered as indicated by the victim's history.

Broad-spectrum antibiotics should be administered to cover the wide range of pathogens associated with gunshot wounds, especially with complex wounds to the abdomen and extremities. The bacteriology of gunshot and blast injury wounds is quite complex. There are a number of environmental pathogens, including soil and water bacteria, such as *Clostridium, Aeromonas, Staphylococcus, Streptococcus, Bacteroides,* and *Bacillus*. These bacteria and associated bacteria on the victim's clothing and skin account for the majority of infections in soft tissue. Wounds penetrating the abdomen increase the presence of *Pseudomonas, Proteus, E. coli,* and other coliforms. Systemic antibiotics should be started as soon as possible and continued for at least 24 hours. Although there is no specific antibiotic that will cover all organisms, selection of antibiotic should be on the basis of probable type of infection. Cellulitis and necrotizing soft-tissue infections can be treated with ceftriaxone plus metronidazole plus gentamicin. Intra-abdominal infections can be treated with the same regimen or cefotetan, and ampicillin–sulbactam can be substituted for ceftriaxone, clindamycin for metronidazole, and ciprofloxacin for gentamicin. There is no substitute for drainage of abscesses and empyemas, excision of devitalized tissue, and removal of foreign debris. Topical antibiotics have not been shown to be useful. Surgical debridement and systemic antibiotics are the mainstay of prevention and treatment of wound infection.

Victims in shock should be taken to the operating room immediately to control bleeding. If this is not possible, type O-negative or type-specific blood should be transfused. Autotransfusion, when available, can be an ideal way to replace lost blood in the victim in shock. Starch or other blood substitutes may raise the blood pressure temporarily, but large amounts of crystalloid fluids may cause increased bleeding. Hypotensive resuscitation, which allows the patient to remain relatively hypotensive as long as organ perfusion is adequate, may be the best intervention if an operating room is not readily available. A systolic blood pressure of 80 mmHg may not be "normal," but it may be sufficient for a supine patient. Increasing the blood pressure to 110 mmHg may seem "normal," but it may also cause rebleeding and irreversible shock. Military antishock trousers or pneumatic antishock garments have not been shown to be beneficial in the treatment of shock secondary to penetrating trauma.[5a] Emergency thoracotomy is indicated for victims who have lost vital signs shortly before reaching the emergency department or while in the emergency department. Injuries to the heart or great vessels can be occluded with Foley catheter balloons, pericardial tamponade can be relieved, and the aorta can be cross-clamped. Hypothermia is commonly unrecognized in the trauma victim and may lead to coagulopathy, cardiac arrhythmias, or electrolyte disturbances. Rectal temperatures should be obtained and only warmed fluids and blood given to the victim.[13]

Wounds from high-velocity bullets are similar to other types of wounds, and standard rules of debridement should be followed. Wide debridement of normal-appearing tissue is unnecessary and should not be done. In general, victims of gunshot wounds should be evacuated quickly and stabilized if possible. Most victims (80%) of gunshot wounds to the chest who survive the first 30 minutes can be treated with a thoracostomy tube and observation.[26a] The amount of blood that is drained from the thoracostomy tube determines whether operative intervention is necessary. Draining > 1500 mL of blood immediately or > 200 mL/hour for over 4 hours is an indication for thoracotomy. Signs of pericardial tamponade are an indication for immediate pericardiocentesis and operative repair. All gunshot wounds to the abdomen should be explored in the operating room. These include all penetrating injuries below the nipples and above the symphysis pubis.

Radiographs should be used to identify bullets, bullet fragments, and bony injuries. Extremity wounds can be treated conservatively unless signs of vascular injury are present. Signs of arterial injury include pulsatile bleeding, expanding hematoma, absent pulses, presence of a thrill or bruit, or an ischemic limb. Experience in combat has shown that vascular injuries do best when identified and treated immediately. However, life takes priority over limb, and a tourniquet may be needed to control vascular bleeding that may ultimately lead to loss of the limb. Obviously, major bony injuries and nerve injuries eventually need operative therapy, but immediate intervention is rarely necessary. Most important, the underlying injury cannot be determined by examination of the external wound.

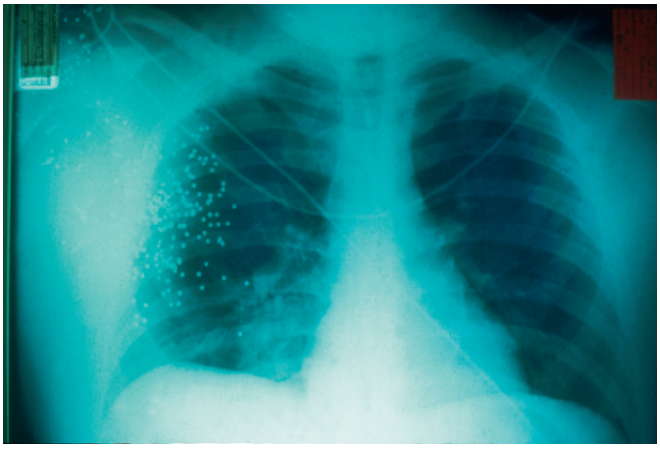
Vascular injuries may not be identified during the initial examination; therefore noninvasive, portable, Doppler ultrasound studies can be extremely valuable in the emergency department. Contrast angiography should be performed on any victim with a suspected vascular injury. The removal of the bullet or bullet fragment is not necessary unless the bullet is intravascular, intra-articular, or in contact with nervous tissue, such as the spinal cord or a peripheral nerve. Bullets found during exploratory laparotomy or wound debridement should be removed, but it is unnecessary to explore soft tissue, such as muscle or fat, solely to remove a bullet. Shotgun pellets that have minimal penetration can be removed from the skin with a forceps. Often plastic or cloth wadding is found in superficial shotgun wounds and should be removed. Shotgun blasts may produce large soft tissue defects that need extensive debridement and either skin grafting or surgical flap rotation to maximize coverage. Patients with powder burns should have as much of the powder residue removed as possible with a brush under local anesthesia. The powder will tattoo the skin if it is not removed, and the deep burns may need dermabrasion or surgical debridement (Fig. 22-17).[17,32,45]

Retained lead bullets and shotgun pellets for the most part are not hazardous; however, when they are within joint spaces or the gastrointestinal tract, significant amounts of lead can be absorbed and toxicity can occur.[40]

Prevention of Hunting Injuries

Most state fish and wildlife agencies have recognized that hunters are at risk for injuries and have tried to develop pro-

A



B

Figure 22-17. **A,** Close-range 12-gauge shotgun wound to the right side of the upper chest. The large central wound was caused by the plastic wadding, and the pellets have struck at an angle toward the shoulder. The patient was turning to the right when shot. The external appearance of the wound indicates a massive injury to the chest. **B,** Chest radiograph of the patient in **A.** No pellets have penetrated the chest, and there was no pneumothorax, pulmonary contusion, or vascular injury. The injury was totally superficial, and the patient was admitted for observation and local wound care.

Figure 22-18. Hunter wearing bright orange clothing, which is essential to distinguish a human from the background and avoid an accidental shooting. Many accidental shootings have occurred when one hunter's movement was mistaken by another hunter for the movement of an animal.

grams to minimize morbidity and mortality. National organizations such as the Boy Scouts of America and the National Rifle Association have been teaching firearm and hunting safety for decades. The Hunter Education Association and the North American Association of Hunter Safety Coordinators (NAAHSC) have attempted to identify high-risk groups and situations by collecting data on both fatal and nonfatal hunting-related injuries. NAAHSC-approved Hunter Safety Programs are available in every state, and all states except Alaska, Massachusetts, and South Carolina require the course before issuing a license to hunt. These courses are roughly 12 hours long and cover hunter responsibility, firearms and ammunition, bow hunting, personal safety, game care, and wildlife identification. They stress respect for the wilderness and a rational approach to game management. All hunters, potential hunters, and persons going into hunting areas should take one of these courses. Approximately 650,000 hunters complete a hunter safety course annually. Since the first course given in Kentucky in 1946, more than 18 million hunters have been certified.

Most injuries could probably be prevented by following a few simple rules. Nonhunters should be aware of hunting seasons and designated hunting areas and wear international orange clothing while in hunting areas (Fig. 22-18). Hunters should always be sure of their target before shooting, use safety harnesses in tree stands, and use appropriate technique and tools for cleaning game. Tree stands should be well constructed. Hunters should never consume alcohol or mind-altering drugs that might interfere with their judgment. Eye protection in the form of safety glasses should be worn while hunting or target shooting to prevent injuries from ricocheting fragments and shotgun pellets. High-frequency hearing loss is common in hunters because of the loud report of the firearm. Although earplugs and headsets can protect the hunter, they are impractical for most hunting and are used mainly for target shooting. Some hunters use a single ear plug for the ear closest to the muzzle of the firearm. This protects the ear most likely to be injured but still allows the hunter to hear approaching game

and other hunters. Bow hunters should always use wrist and finger protection to prevent injuries from the arrow fletching and the bowstring. All arrows should be carried in a quiver until ready for use. The broadhead arrow should always be pointed away from the hunter. These few steps would probably eliminate most hunting injuries.[36,37]

FISHING INJURIES

Sport fishing is associated with a large number of relatively minor injuries compared with hunting. The usual problems associated with outdoor recreation are common among fishermen: sunburn, frostbite, hypothermia, near drowning, sprains, fractures, motion sickness, and heat illness. Lacerations are relatively more common because of the use of knives to cut bait and fishing lines and to clean fish. These lacerations are often contaminated with a variety of marine and freshwater pathogens that may increase the incidence of wound infection. Thorough debridement of the wound and copious irrigation with sterile saline solution are the best initial methods to prevent infection.

Fishhook Injuries

Fishhooks are designed to penetrate the skin of fish easily and to hold fast while the fish is played and landed. To perform this dual role, they are extremely sharp at the tip, have a barb just proximal to the tip, and are curved so that the more force applied to the hook, the deeper it penetrates. Fishhooks may be single or in clusters of two, three, or four to increase the chance of catching the fish. Some state fishing laws limit the number of hooks allowed on a single line when fishing for certain game fish to make it more sporting. Unfortunately, the greater the number of hooks on a lure or line means an increased chance of catching a fisherman. The most common fishhook punctures occur when fish are removed from hooks. The combination of sharp hooks, slippery fish, and an inexperienced fisherman leads to puncture wounds or embedded fishhooks. Many fishermen use commercial fishhook removers or large Kelly forceps to remove hooks. Some fishing guides simply cut the hook with a side-cutting pliers; they believe the remaining segment of hook will eventually oxidize in the victim and disintegrate. Often, fishhooks are stepped on with a bare foot or fishermen catch themselves or another person on the backcast.

Fishhooks can penetrate skin, muscle, and bone, and they may pierce the eye or the penis. Care must be taken in removing a fishhook so that further damage to underlying structures is avoided. The first step is to remove the portion of the hook that is embedded from any attached lines, fish, bait, or lure. This is best done with a sharp side-cutting pliers. A bolt cutter may be needed for large, hardened hooks.

A number of techniques are used for removing embedded fishhooks, but all involve a certain amount of movement of the hook, which causes increased pain. A local anesthetic should be infiltrated around the puncture site to minimize pain and movement of the patient. The following method can be used if the hook is not deeply embedded (Fig. 22-19E). Pressure is applied along the curve of the hook while the hook is pulled away from the point. Because the barb is on the inside of the curve of the hook, this enlarges the entrance hole enough to allow the barb and point to pass through. Sometimes a string looped through the curve of the hook facilitates the process. If the hook is deeply

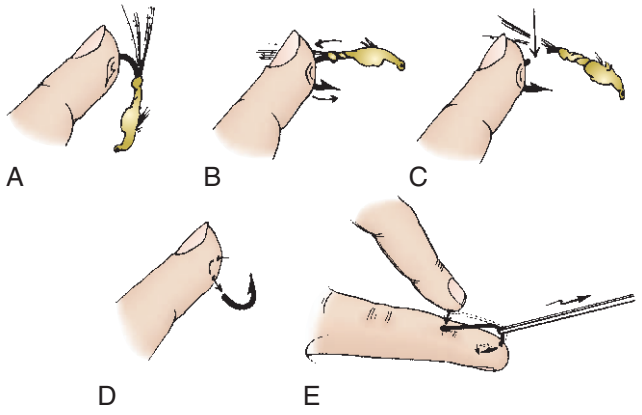


Figure 22-19. **A** to **D,** Removal of a fishhook that has deeply penetrated a fingertip. **E,** "Press-and-yank" method of fishhook removal.

embedded, pressure can be applied along the curve of the hook until the point and barb penetrate the skin at another place, and then the barb can be cut off and the remainder of the hook backed out (Fig. 22-19A–D).

Fishhooks embedded in the eye should be left in place, the eye covered with a metal patch or cup, and the victim referred to an ophthalmologist for further care. Rarely, hooks become embedded in bone or cartilage; this victim must be taken to the operating room to have the hook removed via a surgical incision.

Fishing Spear Injuries

Fishing spears, like fishhooks, are designed to penetrate and hold fish. They may be jabbed, thrown, or propelled by rubber straps or carbon dioxide cartridges. The more force used to propel the spear, the deeper the penetration into tissue. Although arrows are designed to cause bleeding and bullets to cause crushing, fishing spears are designed to hold the fish until it drowns or is otherwise dispatched. Spears may penetrate the human chest or abdominal cavity, skull, or any other anatomic area. Some bleeding may occur, especially if major blood vessels are struck. The victim should be removed from the water as soon as possible and immediate attention given to airway, breathing, and bleeding control. The spear should be stabilized in place, and the victim immediately transported to a medical facility. Penetrating neck and chest injuries may require endotracheal intubation and tube thoracostomy. If a spear is embedded in the victim's cheek and interferes with the his or her airway, cutting the spear off with a bolt cutter and removing it through the mouth is permitted. Spears in all other locations should be left in place, although they may be cut off to facilitate transportation or improve the victim's comfort (Fig. 22-20).

UNEXPLODED ORDNANCE

There are many areas in the world where unexploded ordnance can be found. Types of ordnance may include aerial bombs, rockets, artillery and mortar shells, grenades, and mines. Any area of the planet where a war has been fought within the past century or so has potential for harboring these items. Crews excavating streets in urban areas of England, France, and Germany often uncover unexploded ordnance from World War

A

B

Figure 22-20. **A,** Male patient with a multipronged fishing spear through the foot. He said he saw something move and he speared it. **B,** Same patient with the spear being cut off in the emergency department with a bolt cutter. The patient was taken to the operating room to have the remainder of the spear removed.

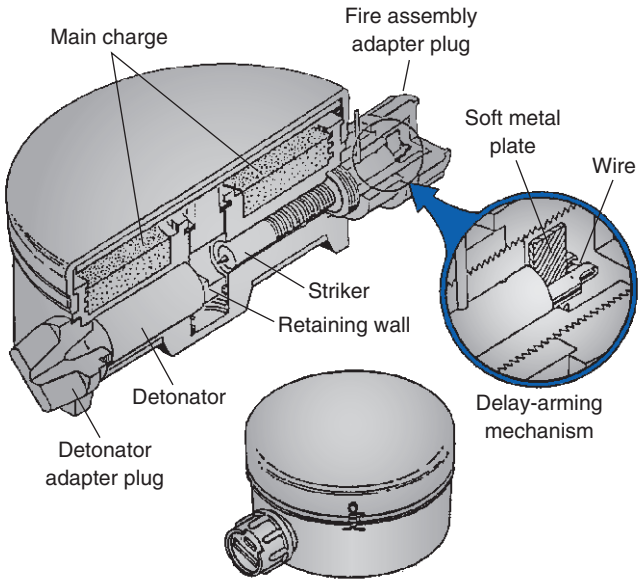


Figure 22-21. An example of an antipersonnel mine manufactured by the Soviet Union.

I or World War II. Many areas of the United States that have been used for bombing or artillery ranges are adjacent to wilderness regions and, although they are usually well marked as impact areas, still pose a risk for the unwary traveler. Many areas of the shallow ocean accessible to scuba divers have sunken munitions transports and warships that contain massive amounts of unexploded bombs, shells, and torpedoes. Another problem that has arisen is the use of mines and booby traps to protect marijuana and opium fields and illegal drug laboratories throughout the world.

Currently, unexploded land mines represent a significant health problem in Southeast Asia, the Balkans, Central America, Egypt, Iran, and Afghanistan. The International Red Cross estimates that someone is killed or injured by a land mine every 22 minutes. The average number of mines deployed per square mile in Bosnia is 152, in Iran 142, in Croatia 92, and in Egypt 59. There are a total of 23 million mines in Egypt alone.[39]

Land mines may be commercially manufactured or produced locally from available materials (Fig. 22-21). Commercial land mines currently produced in United States have a limited active life and self-destruct after their active life has expired. Unfortunately, this is not true of older types of mines or mines produced by other countries. Locally produced mines have no standard size, shape, or detonation pattern and may be very difficult to detect and defuse. These types of land mines are used extensively in El Salvador, Malaysia, and Guatemala.

Land mines have two primary functions, the first of which is to cause casualties, the so-called antipersonnel mines. These may be blast or fragmentation type. The fragmentation type may be either directional or nondirectional (Fig. 22-22). Antipersonnel mines may cause lethal or nonlethal injuries in several persons. Wounded soldiers require more care than killed soldiers do, and the tactical effect may be the same. The second primary function is to destroy vehicles, such as tanks, so these mines are usually much larger.

All mines have three basic components: (1) a triggering device, (2) a detonator, and (3) a main explosive charge. The triggering device differs depending on the type of mine. Blast mines usually involve a pressure-type trigger and occasionally are command detonated, especially for antitank purposes. Many antitank mines will not explode unless a pressure of 300 to 400 pounds (136 to 181 kg) is applied. The M14 blast antipersonnel mine needs only 20 to 30 pounds (9 to 14 kg) of pressure to trigger the detonation. Fragmentation mines are usually triggered by trip wires or similar "touch" devices. The M18A1 fragmentation mine, or "claymore" mine, is designed to be command detonated by an electronic trigger.

Booby traps other than land mines may be mechanical, chemical, or explosive. During the Vietnam War, venomous snakes

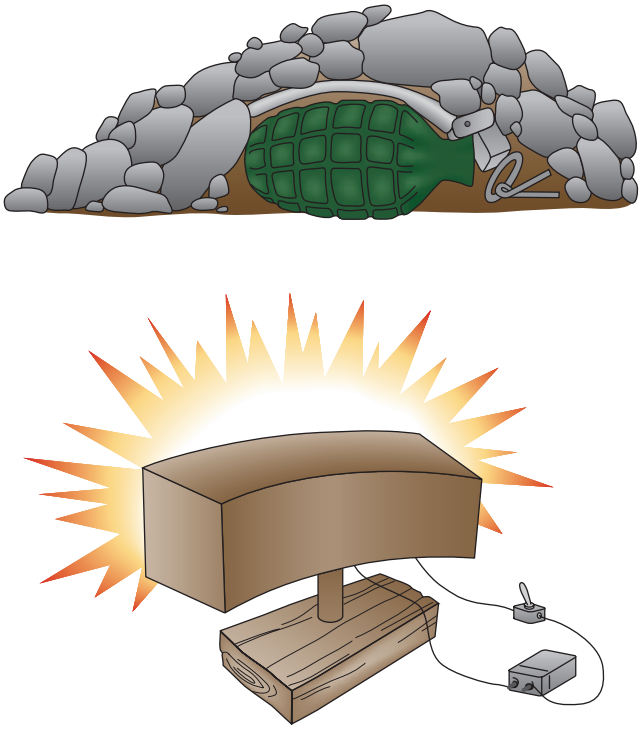


Figure 22-22. *Above,* A directional type mine used against unarmored vehicles or personnel. *Below,* An improvised mine, or booby trap, manufactured from a hand grenade and materials at hand.

were used, as well as the notorious sharpened bamboo spikes known as "punji" traps.

The distribution of mines usually entails spreading them on the surface of the ground; by air along roads, railways, and defensive positions; or hiding them by burying or camouflage along trails or suspected routes of approach.

Injuries from land mines depend on several factors: type of mine (blast vs. fragmentation), position on the ground, method of detonation, whether it explodes above the ground, position of the victim, environment, and type of soil. Four general patterns of injuries occur with land mines. *Pattern A injuries* occur with small blast mines, such as the U.S. M14 and the Chinese Type 72. These injuries usually involve only the leg below the knee. Complete or partial foot amputations are most common, and trunk or head injuries are rare. *Pattern B injuries* are caused by larger blast mines, such as the Russian PMN. These mines contain 4 to 6 times as much high explosive material, and the cone of explosion is much larger. The injuries seen with this type of land mine usually involve massive soft tissue injuries to both legs below and above the knee and commonly the pelvis, abdomen, and chest. *Pattern C injuries* are generally caused by Russian PFM-1, or "butterfly," mines. These mines are usually distributed by air, and the wings are designed to help spread the mines. They are triggered by pressure applied to the wings; handling the mine commonly does this. Most of the injuries involve amputation of the hand at the wrist, but often the head, neck, and chest are injured also. Unfortunately, the loss of one or both eyes is not uncommon with this mine. *Pattern D injuries* are caused by fragmentation mines. These may be bounding mines, such as the U.S. M16 or the Russian OZM, or directional mines, such as the U.S. M18A1 or the Russian MON. They are designed to spread metallic fragments over a wide area at the height of a person's waist. The fragments lose their energy much faster than a bullet projectile but at close range can be devastating. The lethal range is usually 82 to 164 feet (25 to 50 m), with casualties occurring out to 656 feet (200 m). The injuries are quite similar to gunshot wounds and are often multiple.

Large, unexploded artillery shells or bombs may cause a combination of blast and fragment injuries but on a larger scale, sometimes involving scores of victims. Many military and civilian casualties are caused by improvised explosive devices (IEDs), which may be conventional explosives; improvised from fertilizer, propane, or other unconventional explosives; artillery, mortar or other ordnance; or explosive charges removed from said ordinance. The IEDs are commonly used by terrorist groups, which may detonate the devices either by timing mechanisms or by command, sometimes in suicidal attacks. These devices are usually detonated in crowded areas or near important political or military targets to create the greatest impact. Examples are attacks on the Oklahoma City Federal Building, the Beirut Marine barracks, and multiple incidents in Iraq.

The treatment of these injuries can be very complex and involve vascular, orthopedic, soft tissue, abdominal, and craniofacial procedures. The wounds are usually highly contaminated with soil, clothing, and fragments that may be driven deep into tissue proximal to the obvious injuries.[48] In most of these injuries, massive debridement is necessary. Rarely, unexploded ordnance may be imbedded in soft tissues and body cavities and must be removed in the operating room, possibly endangering the lives of medical personnel. Most victims who survive never completely regain normal function, especially if the initial treatment was delayed or inadequate. Postsurgical infection of mine injuries is common and greatly increases morbidity and mortality. Initial treatment involves airway control, treating tension pneumothorax, and controlling hemorrhage. Tourniquets are often necessary to control the bleeding from amputated limbs. Splinting the injured extremity and covering the wound to prevent further contamination is necessary. Initial debridement must be done carefully; removal of fragments may cause bleeding to recur. Penetrating injuries of the pelvis and abdomen usually require laparotomy, and soft tissue injuries may require multiple reconstructive procedures. Broad-spectrum antibiotics and tetanus prophylaxis are appropriate in all cases, and fluid resuscitation is usually indicated with extremity injuries.

Blast injuries without fragmentation may cause tympanic membrane rupture, blast lung resulting from alveolar rupture, and intestinal rupture, although the latter is more common with underwater mine explosions. The mechanism is production of an overpressure wave that travels through tissue of various densities and causes tear injuries at membrane interfaces. These injuries must be suspected in any victim involved in a blast, whether from a land mine or other explosive device.

Scuba divers and swimmers who are involved in underwater explosions may have more serious injuries because of the increased speed of sound in a liquid medium. This can cause more severe tearing of membranes at the fluid–air interface and additional trauma secondary to a "water hammer" effect and spalling. The position of the victim and the number of shock waves caused by reflection of the blast wave off of walls and ground may increase the amount of damage. Victims in contact with solid objects, such as the hull of a ship or vehicle, may have increased injuries because of increased velocity of the blast wave through solids. Burns and translational injuries, whereby

the victim is thrown by the blast and has injuries similar to a fall or motor vehicle crash, also occur.

Generally speaking, the closer the victim is to the blast, the greater the injury. The tympanic membrane will rupture at overpressures of 5 psi. This causes acute hearing loss, pain, and tinnitus. Blast lung is caused by the overpressure wave passing through the chest wall and may involve one or both lungs. Chest pain, dyspnea, and hemoptysis may present immediately or be delayed up to 48 hours. Chest x-ray may show patchy or diffuse infiltrates, pneumothorax, subcutaneous air, and hemothorax. Implosion of air into the vascular system may cause air embolism and sudden death. Abdominal injuries in air blast are uncommon but in water blast may present as abdominal pain, nausea, vomiting, and tenesmus. Sigmoid and transverse colon injuries are most often seen, followed by small bowel and solid organ (such as liver and spleen) injuries from the water hammer effect. Abdominal injuries may have a delayed (up to several days) presentation. The key to therapy is to be suspicious of occult injuries in any victim of a blast, whatever the cause. Most injuries will present within the first hour; however, because injuries may be delayed in presentation, observation and close follow-up are critical. Treatment is generally supportive for ear and lung injuries and operative for abdominal injuries.[3,5,10,11,14,21,23,25,26,30,31]

The references for this chapter can be found on the accompanying DVD-ROM.

23 Tactical Medicine and Combat Casualty Care

Lawrence E. Heiskell, Bohdan T. Olesnicky, and Lynn E. Welling

Tactical medicine can be defined as both emergent and nonemergent care provided to victims of illness or injury related to law enforcement or military operations, often in a hostile environment.[56] Tactical medicine in the early years was often referred to as tactical emergency medical support (TEMS). The emergency medical services (EMS) and prehospital community called it tactical EMS, and the U.S. military coined the phrase *combat casualty care*. Numerous law enforcement agencies now have tactical medical teams composed of on-call physicians and prehospital care providers. Because many law enforcement agencies and branches of the U.S. military have embraced this concept, it is now commonly known as tactical medicine.

Prior to 2001, there was a perception of professional separation between doctors in traditional medical practice and the tactical medicine physicians involved in law enforcement. This was probably related to what might be seen as competing priorities for physicians when dealing with sick or injured patients who are suspects in a police investigation.

No other subspecialty in emergency medicine has experienced the growth rate of tactical medicine. In the past 15 years, more than 170 publications addressing tactical medicine issues have been written. Tactical medicine educational programs have trained thousands of emergency medical technicians (EMTs), paramedics, and physicians, who have responded to the call to provide on-scene emergency medical care to members of the law enforcement community or active duty military.[51] Tactical medicine is very similar for both military and civilian tactical providers. Techniques, strategies, protocols, and equipment are all virtually identical, with few differences. The military tactical medical provider must deal with long deployment times, therefore incurring a significant preventative medicine requirement. Although routine medical care and performance enhancement (e.g., conditioning, nutrition, rest) are important for both civilian and military tactical teams, they take on a longer-term function for the military tactical medicine provider. A civilian tactical operation typically takes hours to days. (The Waco and Ruby Ridge incidents were exceptions, more in concert with a military length of engagement). A typical tactical military operation may take days to months, and other aspects—disease and nonbattle injury—become as important as the tactical medical care (Table 23-1).

Tactical medicine has been a mainstay of military operations since the beginning of modern warfare. The hospital corpsman, or combat medic, was deployed on the front lines with the warriors to provide basic medical care. This care was provided under fire, sometimes in the harsh environments of the jungle, the desert, high mountains, and underwater. As the science of medicine improved, the need to move higher levels of care further toward the forward edge of the battle area was recognized. Shock trauma platoons, manned by emergency physicians and support staff, were sent to the front lines to provide advanced resuscitative support. These units could be fully operational and seeing patients in less than 30 minutes. Mobile surgical teams and forward resuscitative surgical teams developed the technology to put trauma surgical teams within minutes of the location of a combat casualty. These teams are fully mobile

the victim is thrown by the blast and has injuries similar to a fall or motor vehicle crash, also occur.

Generally speaking, the closer the victim is to the blast, the greater the injury. The tympanic membrane will rupture at overpressures of 5 psi. This causes acute hearing loss, pain, and tinnitus. Blast lung is caused by the overpressure wave passing through the chest wall and may involve one or both lungs. Chest pain, dyspnea, and hemoptysis may present immediately or be delayed up to 48 hours. Chest x-ray may show patchy or diffuse infiltrates, pneumothorax, subcutaneous air, and hemothorax. Implosion of air into the vascular system may cause air embolism and sudden death. Abdominal injuries in air blast are uncommon but in water blast may present as abdominal pain, nausea, vomiting, and tenesmus. Sigmoid and transverse colon injuries are most often seen, followed by small bowel and solid organ (such as liver and spleen) injuries from the water hammer effect. Abdominal injuries may have a delayed (up to several days) presentation. The key to therapy is to be suspicious of occult injuries in any victim of a blast, whatever the cause. Most injuries will present within the first hour; however, because injuries may be delayed in presentation, observation and close follow-up are critical. Treatment is generally supportive for ear and lung injuries and operative for abdominal injuries.[3,5,10,11,14,21,23,25,26,30,31]

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Figure 23-1. Forward resuscitative surgical team. (Courtesy Lynn Welling, MD.)

Figure 23-2. Patient transport. (Courtesy Lynn Welling, MD.)

TABLE 23-1. Nonbattle Conditions Encountered by Tactical Medical Personnel (Spring 2003)*

PRIMARY ICD-9 DISEASE CATEGORY	*n*	% OF TOTAL
Digestive	44	17.4
Symptoms ill defined	38	15.0
Mental disorders	29	11.5
Musculoskeletal	29	11.5
Genitourinary	21	8.3
Nervous system sense organs	17	6.7
Skin	15	5.9
Supplemental	15	5.9
Infectious and parasitic	10	4.0
Circulatory	10	4.0
Endocrine, nutritional	8	3.2
Neoplasms	6	2.4
Respiratory	5	2.0
Pregnancy	3	1.2
Congenital	3	1.2
Total	253	100.0

*Navy/Marines Operation Iraqi Freedom from 21 March to 15 May 2003.
ICD, International Classification of Diseases.

Figure 23-3. A high-velocity gunshot wound. (Courtesy Lawrence E. Heiskell, MD.)

and are able to set up or dismantle in 30 minutes, utilize tent shelters or shelters of opportunity within which to perform operations, and provide life-saving damage control surgery to multiple patients under the extreme conditions of modern warfare (Fig. 23-1).

Systems are designed so that staff can resuscitate, treat, and transport patients in extreme hot or cold temperatures, over rough terrain and hostile territory, while the patient is paralyzed and intubated, while wounds are still open, and while attempts are made to prevent the hypothermia, dehydration, and coagulopathy inherent in postsurgical patients (Fig. 23-2).

Tactical medicine has advanced to anticipate and react to changes in combat strategy. In Operation Iraqi Freedom, the initial injury patterns were primarily high-velocity penetrating wounds—that is, mostly gunshot wounds (Fig. 23-3 and Table 23-2).[30] As the war has progressed, the weapon of choice of the insurgents has become the improvised explosive device (IED) (Fig. 23-4).[79] This weapon produces significantly more trauma, including shrapnel, blast, and thermal injuries. It has also required a change in protective body armor as the injury patterns have changed to include more devastating extremity and head-and-neck wounds than torso wounds (Fig. 23-5).[29] The IED has also continued to cause problems with torso injuries, as the blast patterns cause shrapnel to angle up under traditional body armor and through arm openings. This has promoted development of armor that helps better protect these areas.

The terrorist attacks of September 11, 2001, and events such as the 1999 Columbine High School shootings heightened our nation's awareness of the real threats of terrorism and violence

on U.S. soil and diminished some of the resistance to medical providers being actively and closely involved in law enforcement special operations.[80] Today, hundreds of fire and EMS agencies provide tactical emergency medical support to federal, state, and local law enforcement special operations teams.

Law enforcement special operations, often referred to as SWAT (special weapons and tactics) teams, are intended to deal with a wide range of high-risk criminal problems and threats.[69] These include, but are not limited to, hostage rescues, terrorist acts, barricaded suspects, violent and suicidal suspects, takeover bank robberies, high-risk warrant services, and active shooter situations.[18,43,45]

Patient advocacy, with priorities of ensuring the best possible quality of care and patient confidentiality, can be at cross purposes with a police officer trying to gather important facts in

TABLE 23-2. Battle Conditions Encountered by Tactical Medical Personnel (Spring 2003)*

MECHANISM OF INJURY	*n*	% OF TOTAL
Gunshot wound	76	24.1
Shrapnel/fragmentation	65	20.6
RPG (handheld antitank grenade-launcher)/grenade	39	12.4
Motor vehicle accident	28	8.9
Fall	17	5.4
Explosion	16	5.1
Unknown/not recorded	16	5.1
Landmine	14	4.4
Blast	11	3.5
Mechanical/machinery	13	4.1
Other	10	3.2
Multiple (NOS [not otherwise specified])	4	1.3
Blunt	3	1.0
Debris	3	1.0
Total	315	100.0

*Navy/Marines Operation Iraqi Freedom from 21 March to 15 May 2003; wounded in action.

Figure 23-4. An example of an improvised explosive device (IED): a hand grenade wired to the undercarriage of a vehicle. (Courtesy Lawrence E. Heiskell, MD.)

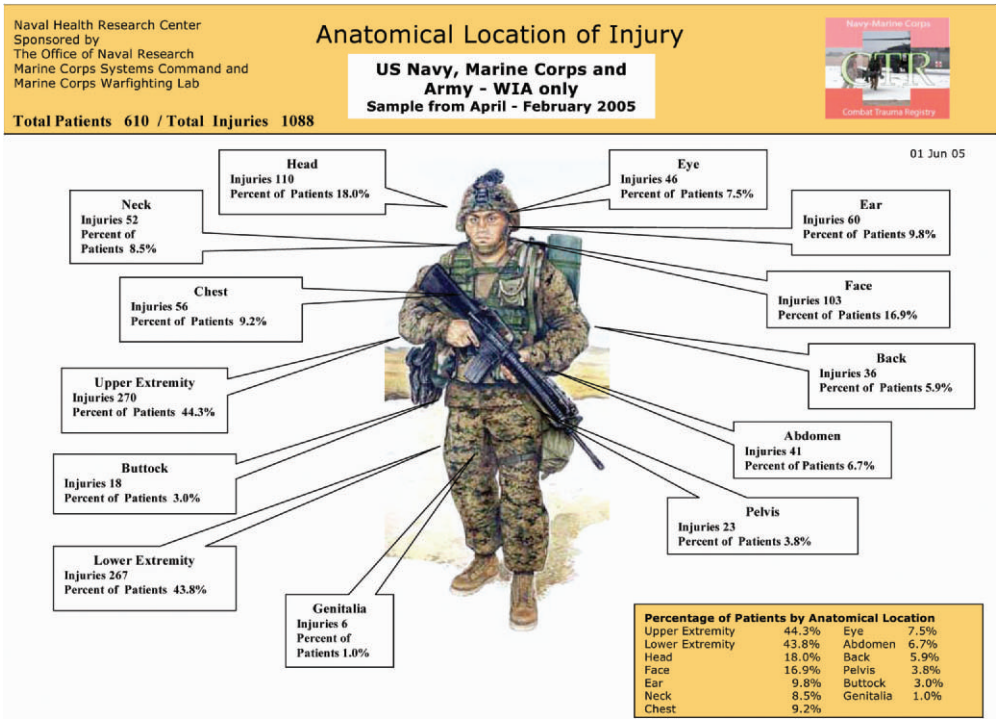


Figure 23-5. Anatomic location of injury (wounded in action [WIA] only). (Courtesy Naval Health Research Center.)

an investigation to ensure public safety and justice. Tactical medicine must respect both patient rights and mission goals.

SWAT teams are found in most midsize and larger law enforcement departments. In some areas, a number of small departments have banded together to form multi-jurisdictional or regional SWAT teams.[16] Harsh environmental conditions, including what many regard as wilderness, will increasingly provide a backdrop for incidents requiring tactical efforts.

HISTORY OF COMBAT CASUALTY CARE

Much of the training and tactics of civilian SWAT teams are based on the experience of military special operations teams. Such military teams have their origins in the U.S. Office of Strategic Services and the British Special Air Service during World War II (WWII). Some of the earliest military special operations teams incorporated tactical medical components.

German *Fallschirmjäger* (paratroopers) incorporated a well-organized medical support team with physicians. Dr. Heinrich Neumann jumped with the unit during the invasion of the island of Crete in 1942.[37] During the Normandy Invasion of June 6, 1944, at Pegasus Bridge on the Orne River, the British, led by Major R. J. Howard, landed with medical support accompanied by a physician, Captain J. Vaughan of the Royal Army Medical Corps.[2] The U.S. Armed Forces during WWII also incorporated physicians in their assault on fortressed Europe. Dr. Robert Franco and Dr. Daniel B. McIlvoy both parachuted into Sicily with the 82nd Airborne Division in April 1943 and jumped into Normandy in June 1944.[26]

During the 1950s, the Army Special Forces (77th Special Forces Group) was formed. As U.S. special operations teams evolved, other specialized teams, such as DELTA, America's elite counter-terrorist force, were formed.[36] Each of these special operations units has a plan for tactical medical support. The growth of terrorism in the 1970s resulted in the formation of other special operations groups worldwide. The Germans established a special unit within their border police, later presented to the world as GSG9 (Grenzschutzgruppe-9).[99] This unit emerged after the 1972 tragedy at the Olympic Village in Munich, Germany. The French formed the Groupe d'Intervention de la Gendarmerie Nationale in 1974, and many other countries have since developed similar units.

Medical providers in the combat environment were traditionally taught to perform with the principles of ATLS (advanced trauma life support). Although this training was instrumental in decreasing the morbidity and mortality of trauma victims in the noncombat scenario, it fell short of providing appropriate care for the patient and the combatant team members on the field of battle. Numerous reviews of past and recent conflicts have noted the inadequacies of this approach to battlefield medical care.[6,8,15,104]

Ninety percent of battle deaths occur in the field, prior to any medical intervention. Bellamy did a landmark review of wounds and death in battle.[7,73] In this study,[7] he noted that 31% of battlefield deaths resulted from penetrating head injury, 25% from surgically uncorrectable torso trauma, 10% from potentially correctable torso trauma, 9% from exsanguinating extremity wounds, 7% from mutilating blast trauma, 5% from tension pneumothorax, 1% from airway obstruction, and 12% from various wounds (sepsis and shock off the battlefield) (Fig. 23-6A). Potentially preventable battlefield causes of deaths include bleeding to death from extremity wounds, tension pneumothorax, and airway obstruction.[54]

These statistics have proven true in today's Global War on Terrorism conflicts and in most tactical medical scenarios. They gave rise to questions about the pure application of the basic advanced life support precepts of airway, breathing, and circulation (ABC) for battlefield and tactical situations. In 1993, led by the Naval Special Warfare Command, a multi-agency working group (Committee on Tactical Combat Casualty Care), including special operations physicians, medics, corpsmen, and operators, began a 2-year study of this issue. This led to the guidelines titled Tactical Combat Casualty Care in Special Operations.[15] The committee meets regularly and reviews new equipment, practices, and current operations for lessons learned, and then revises the guidelines as appropriate. These guidelines, which evolved from the special operations community, are currently being evaluated and implemented in most combatant units of the U.S. military, and of many other countries.[14]

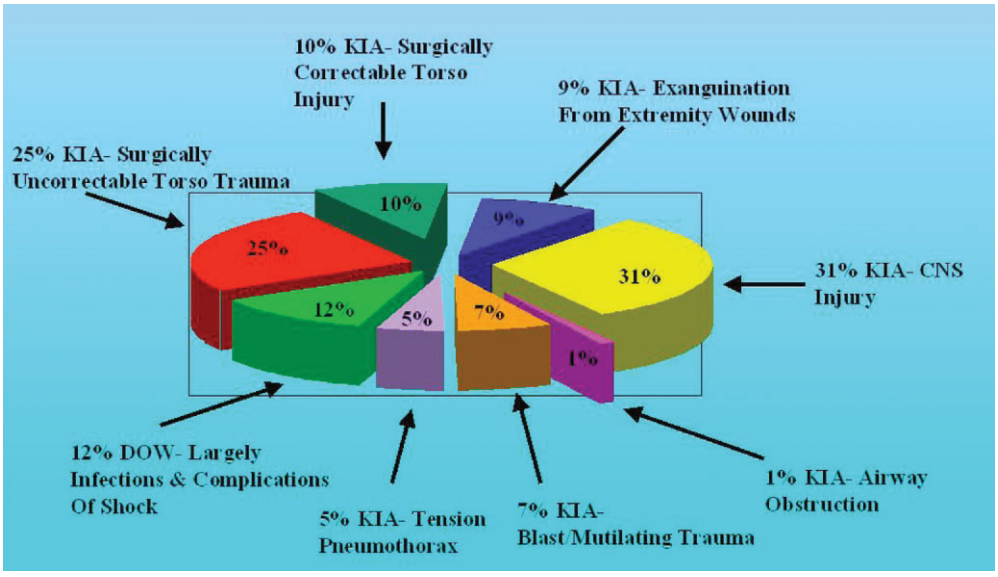
The need for civilian SWAT teams in the United States evolved from high-profile criminal acts that resulted in shocking losses of human life. The seminal incident involved a sniper at the University of Texas at Austin. On August 1, 1966, Charles Whitman shot and killed 15 people and wounded 31 others.[70] In the midst of this tragedy, it became apparent that the law enforcement agencies called out were ill equipped to deal with the threat, hampered by inadequate weaponry and not trained to respond in a timely and optimal fashion. After this incident, many law enforcement agencies began developing specially trained and equipped tactical units to respond rapidly to such threats to public safety.[31] The Los Angeles Police Department and the Los Angeles County Sheriff's Department were among the first law enforcement agencies in the United States to organize and develop full-time tactical units specifically trained to handle high-risk incidents.

Before 1989, there existed great diversity in the ways emergency medical care was provided during law enforcement tactical operations. Early on, most law enforcement agencies relied on regular civilian EMS providers staged at a safe location removed from the area of operation, or they simply called 911. Although this took advantage of an already established prehospital care system, care for injured officers was delayed.[103]

Other agencies trained full-time SWAT officers as EMTs or paramedics. This concept of getting medical care "close to the fight" was also realized in the Gulf War, and the military put this new concept in place during Operation Iraqi Freedom. Information obtained from interviews with military emergency physicians who served in Iraq has suggested success of the new model of battlefield care.[101]

PRINCIPLES OF TACTICAL COMBAT CASUALTY CARE

Tactical combat casualty care (TCCC) varies from ATLS in several distinct ways, primarily because the victim and the medical provider are not in a safe environment. Additionally, medical care of the victim may not be the highest priority, and the team may be hours from higher levels of care and operating in the open under extreme environmental conditions.



A

B

Figure 23-6. **A,** How people die in ground combat. **B,** Intubation. (**A** courtesy Col. Ron Bellamy; **B** courtesy Lawrence E. Heiskell, MD.)

The premise of TCCC is to do the right things at the right times. Underlying this basic statement is the suggestion that good hospital-based medicine is often not good battlefield medicine,[14] as logically follows from these three statements:

1. Good medicine can be bad tactics.
2. Bad tactics can get everyone killed.
3. Bad tactics can cause the mission to fail.

The ultimate goals of TCCC are the following:

1. Treat the casualty.
2. Prevent additional casualties.
3. Complete the mission.

TCCC is divided into three main stages of care[14]: care under fire, tactical field care, and combat casualty evacuation care. These are defined in the following paragraphs.

Care under Fire

Sometimes care is rendered by the medic or corpsman at the scene of the injury while he is still under effective hostile fire. The medical equipment available is limited to what the individual operator or the corpsman or medic can carry in the medical pack. The most effective medical care during this stage of TCCC is fire superiority—that is, winning the battle, or at least keeping enemy heads down and weapons ineffective. The medical provider (and the casualty if able) must work to suppress hostile fire and eliminate the threat as directed by the mission commander, and, if possible, to protect the injured fighter from further harm.

For many reasons, this is undoubtedly the most difficult phase of TCCC. First, the traditional provider, trained to be a "medic first," may find it hard to direct attention to the threat and not maneuver to respond to the casualty. Second, this phase usually occurs in the most exposed environment, where the provider cannot use his normal assessment tools. For example, during nighttime he cannot use a light, as it would draw more fire, and listening for lung sounds with a stethoscope in an explosion-rocked firefight is useless. In earlier conflicts, it was noted that

Figure 23-7. CATS (Combat Application Tourniquet System) tourniquet. (Courtesy Lawrence E. Heiskell, MD.)

Figure 23-8. Mechanical advantage tourniquet. (Courtesy Lawrence E. Heiskell, MD.)

many medics and corpsmen who responded to casualties instead of suppressing fire were wounded or killed, and that a significant number of the victims that they were trying to rescue were already dead. The priorities for the provider during this phase of care, therefore, are as follows[14]:

1. Return fire as directed or required.
2. Try to keep yourself from getting shot.
3. Try to keep the casualty from sustaining additional wounds.
4. Stop any life-threatening external hemorrhage with a tourniquet.
5. Take the casualty with you when you leave.

Airway and breathing problems are not addressed during this phase. The key action is to stop exsanguinating hemorrhage. A tourniquet is the primary means to stop the bleeding on an extremity (Figs. 23-7 through 23-10).

The tourniquet can be applied and left in place by the injured operator or medic, who can then return fire in support of the team. If a tourniquet cannot be placed because of the location of the wound, then direct pressure and a hemostatic dressing are recommended as the appropriate actions.

As soon as possible, the casualty is moved to a safer location, and the next phase of TCCC is instituted. This movement is performed with techniques dictated by the tactical situation. It can be done by, for example, vehicles, pack animals, buddy lifts, or dragging. Another departure from traditional ATLS teaching is that cervical spine protection is not routinely provided in this phase of care. Studies of penetrating neck injuries in Vietnam demonstrated that only 1.4% of patients with penetrating injuries would have benefited from cervical spine immobilization.[4] Although not all combat-related injuries are penetrating, the complexities of moving a patient in an environment where the patient and provider are under fire often preclude even rudimentary cervical spine immobilization.

Tactical Field Care

The tactical field care phase consists of care rendered once the medic or corpsman and the casualty are no longer under effective hostile fire. It also applies to situations in which an injury has occurred on a mission but there has been no hostile fire. Available medical equipment is still limited to that carried into the field by mission personnel. Time prior to evacuation to a medical treatment facility may vary considerably.

In this phase, the medic has a short time to evaluate and treat the wounded. The medic assesses injuries, performs medical

Figure 23-9. Chitosan hemostatic dressing. (Courtesy Lawrence E. Heiskell, MD.)

Figure 23-10. Emergency bandage. (Courtesy Lawrence E. Heiskell, MD.)

care as able (equipment still limited to what was carried onto the battlefield), and then informs the mission commander of the findings. The mission commander then determines what action will be taken (evacuation, abort, continue). This again may be a major departure from nontactical medical care, in that the medical provider is not the ultimate authority on patient disposition. The mission commander decides how much time is taken to care for the casualty in any phase of the operation, if and when MedEvac will occur, and what assets will be allocated from the primary mission toward care of the injured.

During this phase, the provider must assume not only that hostile fire may occur at any time but also that any injured team member with altered mental status may become a threat. The provider must therefore disarm the team member, an action that most warriors resist. This is the first step in the tactical field care phase.

The second step is to address airway compromise. Airway actions are usually rendered as follows: if the victim is unconscious without obstruction, utilize a nasopharyngeal airway (better tolerated and less likely to become dislodged with movement)[1] and a rescue position if able. If airway obstruction is present and cannot be alleviated with these maneuvers, the next recommended treatment is to move directly to a surgical cricothyrotomy. Endotracheal intubation is not recommended at this level of care for several reasons: (1) It requires the medic to carry onto the battlefield, equipment that has no other purpose, (2) the medic must practice regularly to maintain his skills, (3) success rates under austere conditions are believed to be significantly less than those done in a controlled or semicontrolled setting, and (4) the laryngoscope light may compromise team safety on the field.[84,96,97] Emergency cricothyrotomy is the best option in this phase of TCCC. Because of distorted anatomy, it is the best way to protect the airway of a patient with maxillofacial wounds. Blood and tissue in the airway preclude visualization of the cords and make endotracheal intubation difficult or impossible.[14,92]

The third step is to treat breathing difficulties. Any severe progressive respiratory distress is assumed to be due to a tension pneumothorax (the number-two cause of preventable battlefield deaths). One cannot wait for the classic signs (which are unreliable at best and most often impossible to ascertain on the battlefield) of diminished breath sounds, hyperresonance, and tracheal deviation to make this diagnosis.[76] Therefore, faced with victims in increasing respiratory distress and with unilateral penetrating chest trauma, the medic will go directly to a needle thoracostomy. This is the definitive procedure in this phase. A chest tube is not usually needed, it is difficult to perform on the battlefield, and it would only further complicate patient care, transportation, and mission completion.[14]

The fourth step is to readdress bleeding. The medic rapidly locates uncontrolled hemorrhage and any wounds where a tourniquet has been placed. If possible, a hemostatic dressing is placed; the tourniquet may be discontinued if the wound and tactical scenario permit. Even if the bleeding appears controlled, further "rough" evacuation may necessitate keeping a tourniquet in place to prevent rebleeding. Each action that the medic takes is designed to save life with minimal further care by the medic. For example, a medic who is holding pressure on a bleeding wound cannot return fire, take care of other casualties, or perform other procedures on this patient. The patient is not optimally prepared for transportation, which may consist of being thrown over someone's back and carried out. Thus, a tourniquet that would be a last-ditch effort in a *noncombat* environment becomes the method of choice in the tactical combat situation. Each operator in the field carries, and knows how to use, at least one tourniquet that can be self-applied. This allows the operator to self-administer life-saving bleeding control and then continue with the fight until treatment by the medic is possible.

The fifth step is for the medic to determine whether an intravenous (IV) line or a saline lock is beneficial. The advantages would be that the patient could receive fluid resuscitation and IV antibiotics. Disadvantages include a probable delay in transportation, the additional equipment required (and the bulky apparatus that could become dislodged or tangled during evacuation), and difficulty in placing a line under austere tactical conditions. If an IV is deemed necessary but cannot be expeditiously placed, the intraosseous route is utilized. Several devices can be used to achieve this, including large-bore hypodermic needles, traditional intraosseous needles, and devices such as the FAST-1 (fast access for shock and trauma) and BIG (bone-injection gun), which quickly and accurately place the needle in the sternum or in another appropriate location.

The medic's sixth step is to determine whether fluid resuscitation is required. In general, if the patient is not in shock (the best indicators of shock in the field are altered mental status in the absence of head injury, and weak or absent pulses), then no IV fluids are necessary. If the patient is conscious, oral rehydration is permissible and preferred in many tactical scenarios. If the patient is in shock, the medic can give Hextend[67] as a 500-mL bolus and reassess after 30 minutes. If the victim is still in shock, the Hextend is repeated once. Usually, no more than 1000 mL of Hextend is given, and further efforts at resuscitation are determined by the tactical scenario. If the patient has a traumatic brain injury and is unconscious and pulseless, fluid resuscitation is given to restore the pulse. This protocol maximizes survivability of the patient and limits the amount of equipment necessary to be carried onto the battlefield.

After exsanguinating hemorrhage, airway compromise, and breathing difficulties have been addressed, the seventh step in the tactical field care medical plan is to inspect and dress known wounds. The medic locates and appropriately treats wounds already identified but not yet treated because of tactical considerations and then proceeds with a quick but thorough head-to-toe assessment for additional wounds. This is analogous to the secondary survey of ATLS, with a couple of notable exceptions. First, the patient is not exposed. This is because the patient may have to be moved quickly if the tactical situation changes, and because the patient must be kept warm and protected from further injury for a much longer time than in an urban setting. The medic often does this examination by feel in order to avoid using white light, to keep the victim's body armor as intact as possible, and to avoid cutting off protective clothing that may be required during evacuation.

Step eight is to assess for pain control. Analgesia is administered in this phase of care with the following considerations. If the victim is able to fight, non-narcotic preparations are used. These do not affect mental status, allowing the victim to remain armed and responsive. If the victim is unable to fight, morphine and promethazine, IV or intramuscular (IM), are given as needed. Step nine: If not already done, fractures are splinted and neurovascular status is rechecked.

Step ten, the early administration of antibiotics for open combat wounds, significantly reduces the rate of infection.[12]

Again, oral medication is preferred if the patient is conscious, and gatifloxacin has been shown to be highly effective and with minimal risks. Gatifloxacin is a broad-spectrum fluoroquinolone that is active against gram-positive and gram-negative microbes, aerobes, anaerobes, and fresh and salt water pathogens, is a once-a-day drug, and has a long shelf life.

If the victim is unable to take oral medication or has significant abdominal trauma, IV/IM antibiotics are utilized, with cefotetan being the current drug of choice. In many units, the operators are given a "wound pack," consisting of an anti-inflammatory, acetaminophen, and gatifloxacin, and instructed to take the entire pack as soon as possible after being wounded.

Step 11 in the tactical care medical plan is to continue to communicate with the victim, giving encouragement, explaining the care given, and giving updates on the tactical scenario if appropriate.

Traumatic cardiac arrest is treated on the battlefield just as it is in the civilian setting. If the victim is pulseless, apneic, and has no sign of life, resuscitation is not attempted.

After these measures have been taken, or if medical evacuation is now available, the last phase of TCCC, combat casualty evacuation care, is entered.

Combat Casualty Evacuation Care

This phase of care is rendered once the casualty (and usually the rest of the mission personnel) has been picked up by an aircraft, land vehicle, or boat. Additional medical personnel and equipment that have been prestaged in these assets should be available at this stage of casualty management. The management plan aligns closely with that of the tactical field care phase, with the addition of more equipment and perhaps higher levels of medical providers. This phase is also the phase most similar to ATLS, although it may occur in the back of a moving conveyance and is still somewhat limited by available equipment and the tactical scenario. The basic medical plan for combat casualty evacuation care is as follows:

1. *Airway management:* Same as for tactical field care, with the addition of laryngeal mask airway (LMA)/Combitube/endotracheal intubation for definitive airway management prior to cricothyrotomy if the operators are trained and the patient can be intubated (e.g., has no midface injuries). Spinal immobilization is still not deemed necessary for casualties with penetrating trauma for the reasons stated earlier.
2. *Breathing:* Same initial considerations as for tactical field care. A chest tube can be placed if needle thoracostomy has produced no improvement in breathing, or if long transport is anticipated. Most combat casualties do not require oxygen, but its administration may be of benefit in the following situations: low oxygen saturation by pulse oximetry, injuries associated with impaired oxygenation, unconscious patient, and traumatic brain injury (to maintain oxygen saturation >90%). Sucking chest wounds should be treated with petroleum gauze applied during expiration, covering it with tape or a field dressing, placing the victim in the sitting position, and monitoring for development of a tension pneumothorax. Asherman seals are also ideal for quickly securing chest tubes and for sucking chest wounds.
3. *Bleeding:* Same as for tactical field care.
4. *IV:* Same as for tactical field care.
5. *Fluid resuscitation:* Same as for tactical field care, with blood and/or lactated Ringer's solution possibly available.
6. *Monitoring, wound care, re-inspection for additional wounds, analgesia, reassessment of fractures, antibiotics:* All the same as for tactical field care.

The following scenario exemplifies the concepts of TCCC and the various phases of care. Consider a Special Forces team on a night mission. They jump from an aircraft at night into hostile territory. They are then to travel by foot over 4 miles of rocky, mountainous terrain to secure the objective, and then move to the shoreline for a waterborne extraction. On the initial jump, one of the operators sustains an open femur fracture. The medic and the team leader must consider (at a minimum) the following issues:

- How to treat the injured member with the equipment they carried in (keeping in mind that a potentially long evacuation wait time precipitates the need to minimize injury, control pain, prevent infection, avoid shock, defend selves, and maintain concealment)
- How or whether to continue the mission

Option 1. MedEvac the injured member, breaking operational security and calling for helicopter extraction.
Option 2. Commandeering a local vehicle and driving to a pickup site, taking the patient to the planned extraction site, and continuing with the mission.
Option 3. Aborting the mission and leaving the injured member and possibly some of the team behind to provide security until they return with help.

Thus, a single injury throws a lot of variables into the mission commander's decision tree. Preplanning for medical situations is essential, and the medical member of the team is essential in the planning and execution of the mission.

A main precept of TCCC is to move the medical care from being the sole responsibility of the combat medic, to involve each operator and each level of leadership. Each fighter carries a tourniquet that can be self-placed. Each fighter is trained in basic combat lifesaving skills so that the effects of wounds can be minimized (within defined limits) and fire can be returned until the medic can arrive and perform the appropriate advanced medical care. Each leader, from the squad level up, is trained to evaluate medical concerns as an integral part of the mission execution and is able to decide when to abort the mission, when to continue, and when to alter the plan, based on the mission objective and the issues that the injured team member or members bring to the fight.

PRINCIPLES OF TACTICAL MEDICINE

The tactical environment presents unique challenges to law enforcement officers, and the same is true for personnel providing EMS in that environment. Tactical medical care providers must have an understanding of and consideration for law enforcement tactics and mission-specific objectives when planning and providing medical support (Fig. 23-11).[55]

Traditional EMS doctrine maintains that rescuer and scene safety are first priorities, and that patient care is a secondary concern.[78] The nature of tactical operations requires that law enforcement officers and tactical medical personnel operate in unsecured environments and situations with significant potential for violence and injury (Fig. 23-12).[74] Tactical scenes are

Figure 23-11. Tactical medics placing a suspect under arrest during a tactical operation. (Courtesy Lawrence E. Heiskell, MD.)

Figure 23-13. Advanced airway management in the tactical environment. (Courtesy Lawrence E. Heiskell, MD.)

Figure 23-12. At the International School of Tactical Medicine, law enforcement tactical medics train for high-risk vehicle assaults. (Courtesy Lawrence E. Heiskell, MD.)

rarely safe from the civilian standpoint, but tactical medical personnel are trained to conduct concise and limited medical evaluations and interventions in potentially threatening areas.[85]

What sets tactical EMS apart from standard EMS is the ability to render immediate care in an environment that may not be completely secured from threats (Fig. 23-13). When a SWAT team relies on traditional EMS to provide medical care and an operator or civilian is acutely injured during the mission, the EMS unit must wait until either the victim is brought out to the safe ("cold") zone (Fig. 23-14) or for the entire scene to be secured by law enforcement before moving to the patient. When a tactical medical unit is present, care can generally be rendered to the victim in a timely manner, and when the injuries involve acute airway issues or life-threatening hemorrhage, lives may be saved by faster access to care.

Other differences between tactical EMS and conventional EMS include limitations in medical equipment at hand, performing in adverse or austere environments (e.g., while maintaining light or sound discipline), and performing patient assessment from remote locations.[90] "Medicine across the barricade" involves remote evaluation and management of patients, such as when a hostage has become ill or injured and the provider attempts to assist the victim by using the eyes, ears, and hands of someone closer to the situation.[35] The tactical medical provider must use skills not unlike those of an EMS dispatcher handling an emergency over the radio. In addition, standard EMS medical care performed in specific clinical scenarios may require a different approach when the same situation is encountered under tactical conditions.[34]

Tactical medicine can be provided by EMTs, paramedics, registered nurses, mid-level providers (physician assistants, nurse practitioners), or physicians who serve on police tactical teams.[47] Mid-level providers and physicians traditionally have training in advanced surgical and medical procedures beyond what is normally allowed for traditional EMS personnel.[22]

The primary goal of tactical medicine is to assist a tactical team in accomplishing its mission. This is achieved through team health management—keeping the tactical team members healthy before, during, and after operations.[21,62] A full tactical medicine program encompasses the provision of preventive and acute medical and dental care, and for some teams even canine support veterinary care.[50] Ready access to such care has a positive effect on team morale.

One of the most important roles of the provider is to create a formal medical threat assessment for each training and operational deployment. This includes consideration of issues such as environmental conditions (heat, cold, wind) (Fig. 23-15),[41,42] fatigue (and the possible need for rotating operators), nutritional issues,[23] plant and animal threats, and a plan for extrication and transport of patients.[11] When operational, this medical plan should include any medical intelligence that can be gathered prior to or during the mission, including issues such as who is involved, ages of those involved, medical history and

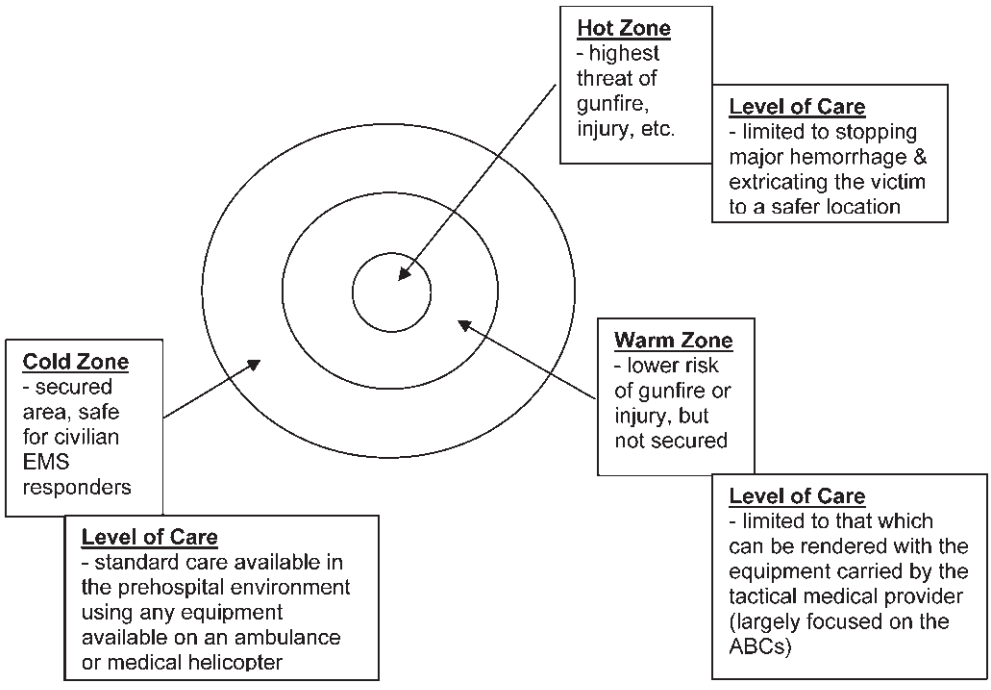


Figure 23-14. The zones-of-care concept of tactical emergency medical services. (Courtesy Bohdan T. Olesnicky, MD.)

Figure 23-15. Tactical operators overcome with heat exhaustion receive cooling measures from a tactical physician. (Courtesy Lawrence E. Heiskell, MD.)

background, preexisting medical conditions, geographic location, and even the weather.[20]

SWAT teams and their tactical medical teams are important community resources not only for their response to major emergencies (e.g., weapons of mass destruction) but also in planning for them. Tactical medical operators, in conjunction with local medical control, EMS, and public health officials, should take a leadership role to ensure that aggressive, proactive planning for these future threats is completed before the resources are needed.[17,105]

Although no one doubts that some terrorists, outlaw states, and even organized criminals have the capability to produce or access chemical and biological agents, the question is whether they will use them.[39] The use of explosive devices is on the rise worldwide. The potential for terrorist acts against the United States is immeasurable. As a result, domestic preparedness and proper training for blast injuries is essential.[27,32,38,75,98]

It is advantageous to have more than one provider as a part of a tactical team. In the event of a serious injury or when multiple casualties are involved (e.g., in a raid on a clandestine drug laboratory),[48,61] one of the team's responsibilities is to lessen the agency's liability exposure with adequate written, photographic, diagrammatic, or video documentation.[66] Another benefit of having multiple providers is the ability to send a provider to the hospital with an officer who becomes ill or injured during a mission. This provider can serve as a concerned advocate for the officer and as a go-between with hospital personnel, which provides significant reassurance to the entire tactical team.[44,46,71]

Team Health Management

Tactical medical providers ensure that everyone on the team is healthy and optimally fit for duty.[49] The team's medical officer is responsible for the team's physical fitness, diet, exercise, sleep, stress management, and preventive medicine. The tactical unit can be viewed as a group of elite operators who are "occupational athletes."

Strength training for many tactical operators consists of traditional bodybuilding exercises. However, this type of physical conditioning does not duplicate the actions needed to perform as a tactical operator. A SWAT team member never does a bench press on a tactical entry. Tactical operators, like professional

athletes, need a broad-based program of training and physical conditioning tailored to the specific actions they will perform.

The team's medical officer should stress regular physical conditioning. A comprehensive plan of proper nutrition and exercise must be established and maintained. This should include a balance of aerobic and anaerobic exercises and stretching. Cardiovascular fitness workouts, such as running or swimming, are excellent for the tactical team. Full-body or resistance circuit weight training is excellent for strength training, but it must be a total body workout. Training some parts of the body but ignoring others can lead to costly injuries and a lower level of fitness than desirable.[93]

Flexibility training is one of the best ways to prevent injuries in the field. Unfortunately, it is frequently ignored. Regular stretching or yoga has been long recognized as beneficial in athletic physical conditioning. In this respect, the tactical unit is no different from any other group of athletes. When the physical conditioning program encompasses all these points, the team operates at its peak potential with fewer injuries.

A sound diet should be stressed and maintained, but diet is often a controversial topic. In response to the obesity problem in the United States, the Food and Drug Administration (FDA) and many other researchers have looked at diets worldwide and their effects on the human body. Randomized clinical trials studying the risks and benefits of various individual diets are only recent works.[10,28,83,102] The FDA studied the traditional food pyramid (based on four food groups and no longer considered a valid nutritional program[72]), and Mediterranean, high-carbohydrate, high-fat, high-protein, low-fat, low-carbohydrate, Atkins, Zone, Weight Watchers, Ornish, and South Beach diets.[25] They revised their food pyramid to a more balanced program designed for variations in age, sex, and level of physical activity, and containing five food groups: grains, vegetables, fruits, milk, and meat and beans. Fats, sugars, sodium, and total caloric intake are restricted. The FDA has an interactive website for the new food pyramid (www.foodpyramid.gov). Although our knowledge of diet and exercise is improved, data continue to be gathered, and the perfect dietary program for the tactical operator is not yet known. Nonetheless, fast foods and simple sugars should be deemphasized or eliminated from the tactical operator's diet.

The team medical officer essentially becomes the family physician for the tactical unit and should be prepared for this role. Regardless of the level of training, the team physician will be viewed as the medical advisor to the tactical unit. It is this relationship that fosters better team health overall, and better performance of the unit as a result. Preventive medicine should be stressed with regular physical examinations and treatment where appropriate. Smoking cessation, alcohol and drug counseling, and stress management are the responsibility of the team medical officer.

A

B

Figure 23-16. **A,** Blackhawk Products Group Special Operations medical backpack. **B,** Ballistic vest, level IIIa, showing trauma plate worn for tactical operations. (Courtesy Lawrence E. Heiskell, MD.)

Tactical Medical Equipment

In general, tactical EMS equipment comes from other areas of emergency medicine and law enforcement and is combined into field-expedient, multifunction toolkits. Looking at the gear as a whole, a modular approach may be most helpful (Fig. 23-16A). The gear differs depending on the roles of the providers and the tactical unit.[33] Basic equipment for the operator includes essential items. Typically, an operator has a duty uniform consisting of a battle dress uniform or jumpsuit.[95] The uniform may undergo appropriate modifications depending on weather conditions. As with other areas of outdoor activity, it is wise to use a system of appropriate layers that can be easily adjusted to changing weather conditions. Waterproof and breathable outer layers may be a consideration, as are wicking underlayers. In addition to the standard duty uniform, a Nomex balaclava and gloves are worn to protect from exposure to pyrotechnic devices on many entries.

Because this is an environment where gunfire may be encountered, ballistic protection is needed. For the tactical medical

TABLE 23-3. U.S. Department of Justice Rating of Body Armor

	TEST VARIABLES				*PERFORMANCE REQUIREMENTS*		
ARMOR TYPE	TEST ROUND	TEST AMMUNITION	NOMINAL BULLET MASS	MINIMUM REQUIRED BULLET VELOCITY	REQUIRED FAIR HITS PER ARMOR PART AT 0° ANGLE OF INCIDENCE	MAXIMUM DEPTH OF DEFORMATION	REQUIRED FAIR HITS PER ARMOR PART AT 30° ANGLE OF INCIDENCE
I	1	38 Special RN Lead	10.2 g 158 gr	259 m/sec (850 ft/sec)	4	44 mm (1.73 in)	2
	2	22 LRHV Lead	2.6 g 40 gr	320 m/sec (1050 ft/sec)	4	44 mm (1.73 in)	2
II-A	1	357 Magnum JSP	10.2 g 158 gr	381 m/sec (1250 ft/sec)	4	44 mm (1.73 in)	2
	2	9 mm FMJ	8.0 g 124 gr	332 m/sec (1090 ft/sec)	4	44 mm (1.73 in)	2
II	1	357 Magnum JSP	10.2 g 158 gr	425 m/sec (1395 ft/sec)	4	44 mm (1.73 in)	2
	2	9 mm FMJ	8.0 g 124 gr	358 m/sec (1175 ft/sec)	4	44 mm (1.73 in)	2
III-A	1	44 Magnum Lead SWC Gas Checked	15.55 g 240 gr	426 m/sec (1400 ft/sec)	4	44 mm (1.73 in)	2
	2	9 mm FMJ	8.0 g 124 gr	426 m/sec (1400 ft/sec)	4	44 mm (1.73 in)	2
III	—	7.62 mm (308 Winchester) FMJ	9.7 g 150 gr	838 m/sec (2750 ft/sec)	6	44 mm (1.73 in)	0
IV	—	30–06 AP	10.8 g 166 gr	868 m/sec (2850 ft/sec)	1	44 mm (1.73 in)	0
Special requirement	—	*	*	*	*	44 mm (1.73 in)	*

*See section 2.2.7 of reference.
g, grams; gr, grains.
From U.S. Department of Justice National Institute of Justice: Ballistic Resistance of Police Body Armor. NIJ Standard 0101.03; 5.2.1, April 1987.

provider, levels I and IIa are not advised. Level II is the bare minimum if the body armor is concealed under a shirt or uniform, but levels IIIa to IV are better (Table 23-3, and see Figure 23-16B). These levels of protection have a good balance of bullet stopping power and ability to absorb blunt trauma. Many tactical operators combine body armor with ballistic plates made of metal or ceramic, which stop high-velocity rifle bullets. Body armor is chosen by the agency from a vast array of different types and styles produced by a host of manufacturers. Some tactical physicians also carry a Kevlar blanket or ballistic shield, which can be used to cover a patient in harm's way or can be used as a mobile source of cover when providing care or extracting a downed victim. These blankets, although effective, are extremely heavy and bulky.

The weight and bulk of all nonmedical tactical equipment hinders the ability to carry large amounts of additional material. The medic needs to decide how much can be carried and whether to wear a backpack or a load-bearing vest, or neither. In general, the tactical medical provider must be able to effectively carry equipment and operate in a tactical situation without hindering the rest of the team. Of the two general sets of medical equipment, one is carried for immediate care, typically worn in a small backpack or load-bearing vest, and the second is carried in a larger backpack or duffel bag in the support vehicle. The latter is used for more extensive treatment, multiple casualties, and prolonged transports.[59]

Communication

Communication between team members and members outside the area of the operation is often essential. Radios with throat microphones and headsets are fairly standard on most tactical units. Radios tend to be on secure channels to ensure the security of a mission. Some communications may even be encrypted. Simple communication between members may involve standard or specialized sign language.

Entry and Breaching Tools

Specialized entry tools are used to gain access to barricaded subjects or closed doors. Typical items, familiar to firefighters and EMS personnel, include pry bars, battering rams, sledge hammers, hooks on chains or rope, stop blocks, and halligan tools (Fig. 23-17). Ladders may be needed to gain access to an elevated or depressed point. In extreme cases, a variety of explo-

Figure 23-17. Entry tools: two rakes, a halligan tool, a ram, and a sledge hammer are shown. (Courtesy Bohdan T. Olesnicky, MD.)

Figure 23-18. At the International School of Tactical Medicine, law enforcement tactical medics train with the HK MP5 submachine gun. (Courtesy Lawrence E. Heiskell, MD.)

sive devices are available to trained explosive experts in the tactical unit to gain entry to an area.

Weapons Systems

Whether a team medic should be a sworn law enforcement officer or an armed civilian has been a subject of much debate. Regardless, a provider must be familiar with the unit's weapons systems.

For offensive or defensive purposes, weapons systems are constantly encountered in the tactical arena. A provider who is a sworn officer has a primary role as an operator on the unit and a secondary role as a medical provider.[77,81] However, a provider who is first a medical officer would still have to protect himself in a hostile situation and therefore would be armed defensively. In another scenario, the provider may be unarmed but may have to take charge of an officer's weapon in a medical or tactical situation. An armed officer who is disoriented may become a danger to the team, so the provider would need to take charge of all of the officer's weapons and render them safe. In the worst case, the provider may have to defend a downed officer using the officer's weapon.

Weapons system familiarity is paramount for the tactical medicine provider. A provider should be familiar with every handgun, shotgun, rifle, submachine gun, assault rifle, and smoke or chemical agent gun used by the team. All tactical team members, whether providers or not, should be able to use any weapon a team member carries and render it safe.[91] The provider should not be exempt from this requirement (Fig. 23-18).

Different weapons systems use different ammunitions (see Chapter 22). Typically, there is a duty handgun, which shoots low-velocity handgun ammunition. This same ammunition may be used by a submachine gun, such as the Heckler and Koch (HK) MP5 UMP 40, or UMP 45. Calibers of the handgun and submachine gun should be matched to avoid the wrong caliber ammunition going into the wrong weapon, causing malfunction in a crisis situation.

Assault rifles, such as the Colt AR 15 or Colt M16, shoot .223-caliber high-velocity cartridges. The provider may be exposed to shotgun ammunition, typically 00 buckshot. The team may have a sniper, who shoots with a .308 high-velocity rifle that is bolt action for precision shots. The flight characteristics and ballistics of shotgun, rifle, and handgun ammunition vary depending on the weight, shape, and velocity of the ammunition. The provider should be familiar with the effects in order to treat field wounds appropriately.

In addition to traditional ammunition, the provider may be faced with the use of distraction devices, chemical munitions, and less lethal munitions. Chemical munitions deliver a chemical agent to an intended target to disorient or incapacitate and facilitate capture or surrender without loss of life. Typical chemical agents used in tactical operations are tear gas or a derivative of pepper spray.

Less lethal munitions are also fired to incapacitate and facilitate capture. Unfortunately, with this use of force, injury is common, so the provider should be familiar with the injuries and their treatments. These munitions are low-velocity projectiles of wood, hard rubber, foam rubber, plastic, or bean bags. They have enough force to cause pain and usually make an assailant stop aggressive action on contact. They usually cause minor blunt trauma, but they may achieve enough force and velocity to penetrate into body cavities, causing penetrating injures (Figs. 23-19 through 23-21).

Explosive breaching techniques and distraction devices, such as "flash-bangs," are often deployed during tactical operations. The small explosives used to gain entry into an area can create injuries that the medic must be prepared to recognize and to treat effectively.[68] For example, the flash-bang is a device that is hand thrown into an area to deliberately disorient a suspect and divert attention toward the device and away from the entry team. These devices usually have a nonexploding canister and a small explosive charge. The device is activated and thrown much like a military hand grenade, but it causes a brilliant flash of light (6 to 8 million candlepower) and a thunderous noise (175 decibels). This is accomplished by venting explosive gases through multiple holes in the canister (Fig. 23-22). Medical problems caused by flash-bangs and explosive entries include the following:

Figure 23-19. A 37-mm less-lethal munitions launcher. (Courtesy Lawrence E. Heiskell, MD.)

Figure 23-20. The stinger grenade has a small explosive charge that disperses many small rubber balls in a spherical blast pattern, causing compliance through pain. (Courtesy Lawrence E. Heiskell, MD.)

- Burns, both minor and major
- Smoke causing bronchospasm
- Vestibular dysfunction
- Transient visual disorientation
- Emotional upset and anxiety

Of note, in general use, the flash-bang has not been reported to cause ear drum rupture.

Explosive breaching is the role of the team's explosives expert, with whom the medic should consult, as part of the medical threat assessment, about the types of explosives planned and the blast forces that may be encountered.

Vision

Covert operations and low-light situations dictate the use of visual adjuncts. Binoculars, tactical mirrors, spotlights, periscopes, strobe lights, chemical lights, and headlamps are often deployed in a low-light tactical environment (Fig. 23-23). Proper training and discipline are required for use of these devices in the tactical environment, as they may give away one's position and alert a hostile opponent to the team's position. Similarly, electronic night vision equipment, which operates outside of the range of visible light, is also extensively used.

MEDICAL PERSONAL PROTECTIVE EQUIPMENT

Universal precautions against infectious diseases must be deployed; tactical medicine is no different from any other venue in this respect. Medical personal protective equipment (PPE) includes masks, eye protection, gloves, and perhaps gowns. In remote locations, surgical treatment may be provided prior to transport to a tertiary care center. The basics for protection should be carried on the provider's person in a readily accessible location. Some don surgical gloves underneath their shooting gloves prior to an operation so they will be ready if the need arises. Although not sterile, they provide protection from blood and body fluid–borne pathogens.

Personal Supply Module

To reduce the amount of equipment carried by the medic and to help team members help themselves, each member of the tactical unit should carry a personal supply module (PSM), or "self-help kit," with medical supplies. A team member can thus provide self-help or aid another member, and the medic may not have to be summoned until the scene is more secure. A typical PSM should be vacuum sealed and contain supplies for basic trauma care and for IV access (Table 23-4 and Fig. 23-24). Vacuum sealing these contents provides protection from the elements and makes them last longer. It also cuts down greatly on bulk, but it adds some weight.

Basic Medical Module

In addition to a PSM, a basic medical module (BMM) should be carried by team medics. Because every team member in a tactical unit should have at least basic EMT certification, a BMM could be used by any team member to provide initial care to a victim. The BMM should have basic splinting material and dressing material (Table 23-5).

Basic airway tools, such as nasal airways and pocket mask, should be included. A bag-valve-mask or a more compact alternative is advisable in the tactical environment. A simple bag-valve-mask alternative device (BVMAD) can be constructed out

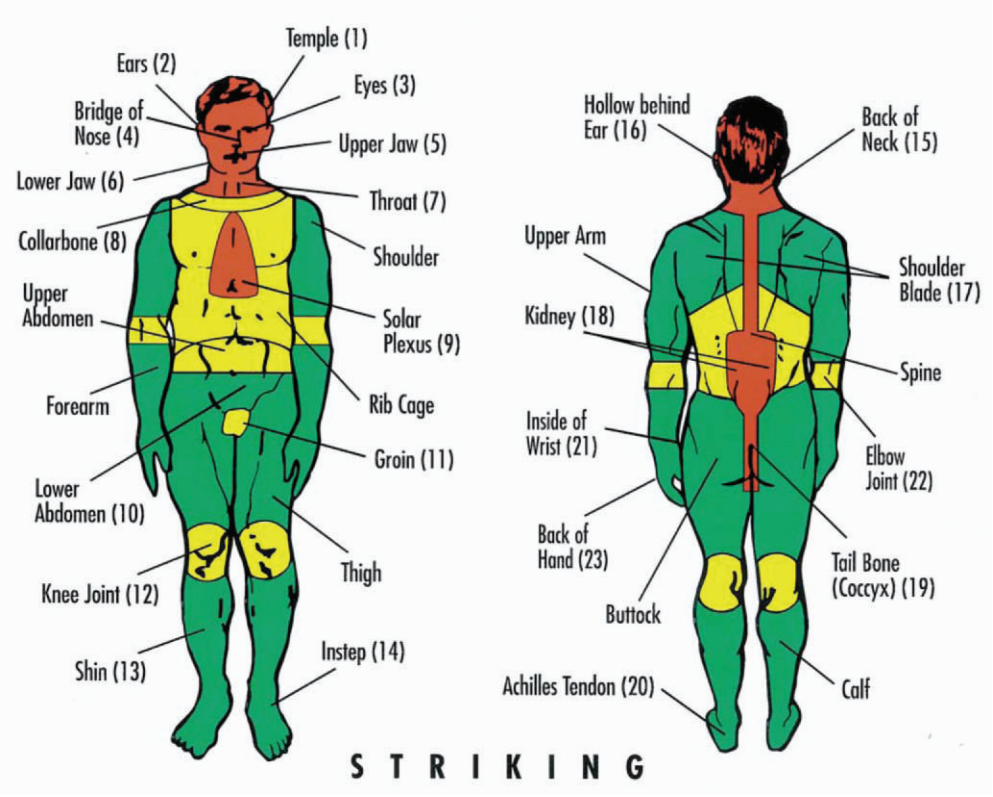


Figure 23-21. Target areas for direct-fire or skip-fire less-lethal projectiles. The *orange areas* are nontargets, the *green areas* are preferred targets, and the *yellow areas* should be targeted with caution only. (Courtesy Armor Holdings, Inc.)

Figure 23-22. The Def Tec 25 flash-bang device. (Courtesy Lawrence E. Heiskell, MD.)

Figure 23-23. A Surefire headlamp setup can use white light or LED light in low-light tactical situations. (Courtesy Lawrence E. Heiskell, MD.)

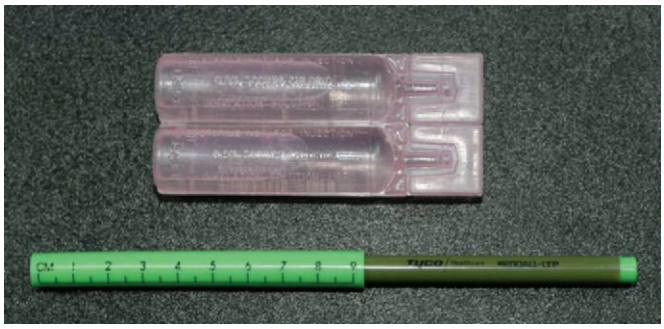


Figure 23-24. Saline bullets remove the most common injury: foreign bodies in the eyes. (Courtesy Bohdan T. Olesnicky, MD.)

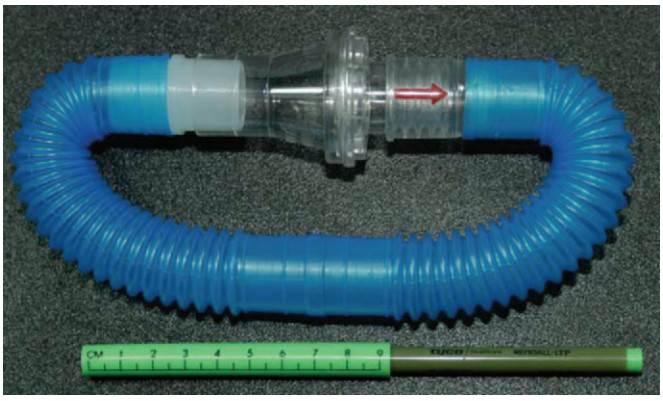


Figure 23-25. The bag-valve-mask alternative device (BVMAD) as it is stored, with the mouthpiece *(red arrow)* over the exhaust port. (Courtesy Bohdan T. Olesnicky, MD.)

TABLE 23-4. Sample Contents of Personal Supply Module (PSM)

Trauma dressing	Bandages such as the Israeli bandage can be easily self-administered by the victim on most extremity wounds.
IV start kit	100 mL IV fluid, an alcohol wipe, tourniquet, IV catheter (3), IV tubing, tape, flush and saline lock
Minor dressings	Adhesive bandages should be carried by all team members.
Saline bullets	Foreign bodies in the eyes are very common on entries, and a bottle of eye drops (saline bullets) may allow an operator to continue on a mission (see Figure 23-24).
Medicines	Pain: acetaminophen, ibuprofen, narcotic analgesics Antibiotics: ciprofloxacin, metronidazole, cephalexin
Other wound items	Surgical staples or liquid tissue adhesive

TABLE 23-5. Sample Contents of Basic Medical Module (BMM)

Splints	Two SAM splints
Airway	Pocket mask, bag-valve-mask (BVM) or BVM alternative device, oral and nasal airways
Litter	Fold-up stretcher
Wound care	Various trauma dressings
Other	Elastic (ACE) wraps and cravats

of respiratory supplies from a one-way valve, flexible tubing, and a mouthpiece (Fig. 23-25).[5] This is the preferred ventilatory device in the tactical environment, as it allows a rescuer to provide ventilation without unnecessary bulk.

Oxygen is rarely useful in the immediate tactical environment, so O_2 cylinders are left in the support vehicle with a regular bag-valve-mask and retrieved when necessary. An automated external defibrillator (AED) should be carried in the support vehicle.[3] A small collapsible litter for extrication of a downed person should be a part of every BMM.

Intermediate Medical Module

An intermediate medical module (IMM) is intended to be used by paramedics and registered nurses. Unlike the BMM, it contains equipment and supplies suitable for advanced life support (ALS). Under standing orders from a team physician, ALS providers may provide advanced cardiac life support to a victim. An IMM is much more extensive than a BMM. It contains medications, IV tubing, IV fluids, an endotracheal tube, a Combitube, a laryngoscope, a light wand, and, if protocol allows, a cricothyrotomy kit (Table 23-6 and Figs. 23-26 and 23-27). These facilitate placement of a definitive airway prior to transport, which, when used with a BVMAD, permits hands-free ventilation of a patient, allowing extrication by one or two team members. Proficiency with the airway toolkit is of high priority, as conditions in the tactical environment are difficult at best.

TABLE 23-6. Sample Contents of Intermediate Medical Module (IMM)

Airway	Endotracheal tubes, laryngoscope, stylette, bougie, bag-valve-mask alternative device (BVMAD)
Medications	IV setup and tubing, pain medication, rapid sequence intubation medications, antibiotics (see Figures 23-26 and 23-27)

Advanced Medical Module

The most complex module is the advanced medical module (AMM), intended for independent practitioners, such as nurse practitioners, physician assistants, and physicians. These practitioners can perform advanced surgical procedures and medical interventions that can make the difference between life and death on a long transport. Transport to definitive care should not be delayed unless medically necessary. However, if advanced care in the field is indicated, it can be provided by an independent practitioner with an AMM.

Major Trauma Module

Lengthy surgical interventions in the field are not advised and have extremely poor prognoses. Although rapid transport to a trauma center should not be delayed, some surgical procedures may be of benefit when performed in the field: laceration repair to stop bleeding or facilitate evacuation, cricothyrotomy, and,

Figure 23-26. Vacuum-sealed intravenous start kit. (Courtesy Bohdan T. Olesnicky, MD.)

Figure 23-27. A well-stocked and functional intermediate medical module (IMM) airway kit, containing a laryngoscope, a bag-valve-mask alternative device (BVMAD), oral airways, a bougie, a stylette, endotracheal tubes, tape, and a surgical airway kit. (Courtesy Bohdan T. Olesnicky, MD.)

in critical situations, chest tube insertion. These types of procedures can usually be performed using only the essential equipment found in a vacuum-sealed minor-surgery tray (Fig. 23-28), which can be one component of the major trauma module (MTM) for advanced providers (Table 23-7).

Many users include hemostatic dressings in trauma kits, but their use is not yet well studied.[60,86] The efficacy versus the potential harm of hemostatic dressings is a subject of debate.

Direct pressure with a sterile dressing is the initial approach to hemorrhage control. The traditional and time-tested approaches of pressure-point compression and tourniquets are useful adjuncts to any bleeding problem, so these materials should be included in the MTM. A set of combine dressings, gauze, petrolatum gauze, and Israeli dressings should also be carried.

Figure 23-28. A vacuum-sealed minor-surgery tray. Many procedures can be accomplished with a minimum of equipment. (Courtesy Bohdan T. Olesnicky, MD.)

TABLE 23-7. Sample Contents of Major Trauma Module (MTM)

Israeli dressing	May be self-administered by the patient with one hand; combines an ACE wrap, combine dressing, and tourniquet in one device
Combine dressings	To control heavy bleeding or cover eviscerated bowel and open fractures
Gauze pads	Multiple uses
Antibiotic packs	Pre-prepared medication packs for major trauma, containing pain medication and antibiotics
Tourniquet	Several excellent devices are available for tactical use
Wound closures	Sutures, staples, and wound adhesives
Minor-surgery tray	For performing surgical procedures that cannot wait for extrication or transport
ACE wraps	Strains, sprains, and fractures are common
Splinting material	SAM splint or a short sealed roll of fiberglass casting material. Bullet wounds frequently fracture long bones
IV fluids	For infusion of medication and management of shock as needed. Also used for wound irrigation and eye irrigation. Several IV start kits should also be on hand
Supplies	Gloves, tape, trauma shears, tweezers, adhesive bandages, roll gauze, cravats, nasal airways, 60-mL syringes, headlamp
Other	Duct tape, biohazard bags, medical record sheets, and trauma tags for multiple casualties

Support Vehicle Module

Additional supplies and equipment are kept in the support vehicle module (SVM). Consumable items should be kept in the SVM, so that other modules can be restocked from it (Table 23-8). The SVM contains equipment such as O_2 cylinders, an AED, airway adjunct devices, fiberoptic scopes, nebulizers, surgical trays, chest tubes, cervical collars, backboards, peroxide, povidone-iodine, liter bags of crystalloid IV fluid, replacement filters for gas masks, and fiberglass splinting material.

TABLE 23-8. Sample Contents of Support Vehicle Module (SVM)

Biohazard container	Disposal container for used sharps and medical waste
Saline eye flush	Foreign bodies in the eyes are common on entries
Elastic (ACE) wraps	Strains, sprains, and fractures are common
Splinting material	Fairly bulky and difficult to carry in the medical pack. C-collars are frequently kept here and may be retrieved when needed
IV fluids	For prolonged transport or massive hemorrhage, more IV fluid should be kept in the SVM and not carried on entries. Multiple IV start packs for use when necessary
Ice packs	Ice packs are commonly used, as ice is not always available in the field. If the location has a freezer, bags should be kept to use existing ice
Wound dressings	Additional adhesive bandages, Israeli dressings, combine dressings, ABD pads, and burn dressings
Advanced airway tools	Difficult-airway tools may be needed in the cold zone prior to transport, to secure a definitive airway
Spare uniforms	If decontamination is needed, the victim will need to be reclothed in a clean, dry uniform, particularly in cold or wet environments
Oxygen cylinders	Best left in the support vehicle because of their weight
Bag-valve-mask (BVM)	Replaces the BVM alternative device when hooked up to oxygen in the cold zone
Automated external defibrillator (AED)	The AED is proven to save lives but is too bulky to carry on entry

Field care is limited only by the equipment that can be transported and the training of the providers. Items carried by advanced providers include the following:

- Central line
- Tracheotomy set
- Retrograde intubation set
- Laryngeal mask airway
- Chest tube set
- Fiberoptic intubation set
- Blood products or blood substitutes

CBRN Specialty Modules

Depending on the role of the tactical unit, chemical, biologic, radiologic, or nuclear (CBRN) threats may be encountered. These incidents require tactical emergency medical care because they involve large crime scenes with casualties. Individuals trained in tactical emergency medicine are much more familiar with evidence collection and preservation, and they usually already have necessary security clearance to enter such an area. Until the scene is cleared, the tactical physician may be the only one who can provide medical care to victims inside.

The chemical and biologic environments are specialized depending on the agent released. Various civilian and military protective gear and respirators or supplied air sources may need to be worn. Operating in CBRN protective gear requires extensive training in addition to regular tactical training. Antibiotic prophylaxis with ciprofloxacin, as well as agent detection equipment, may be carried by the medic in this case. Several biologic and chemical diagnostic kits and meters are available but costly.

Radiologic incidents involve the dispersal of a radiologic agent with conventional explosives, the combination often referred to as a dirty bomb. Nuclear detonations refer to the splitting of a radioisotope and the resultant massive energy release from a nuclear bomb. Geiger counters are available for radiologic and nuclear situations. These situations require a great deal of additional training, but they are not beyond the realm of tactical emergency medicine.

Hazardous material (hazmat) situations are frequently seen in civilian law enforcement raids on clandestine drug laboratories. Level A, B, or C protective suits with gas masks or supplied air may be required in these situations as well. Hazmat and CBRN situations are highly specialized in their nature and require extensive training, beyond the scope of this text.

Medical Threat Assessment

Any mission planning must include a medical threat assessment (MTA). The SWAT commander uses information from many sources to create a tactical plan prior to execution of a mission, including the manpower available, building layouts, street layouts, support equipment needed, nature of the mission, available weaponry, and various sources of intelligence.[65]

An MTA is an important component of the intelligence the commander needs to properly execute the mission. It is the responsibility of the tactical medic to provide a concise and accurate medical briefing to the commander. MTA forms should be used on every mission to ensure systematic planning, as scenarios and problems may be unique. Only a systematic approach ensures complete assessment of the situation.

The tactical medical team and its MTA are key factors in dealing with apocalyptic terrorist events, such as the Columbine school shootings and the school hostage crisis in Beslan, Russia.[87] Other venues, such as protection details and the war on drugs, rely heavily on a team's internal capacity for medical care, as evidenced by the U.S. Marshals Service Judicial Protection Training Program.[88]

A complete threat assessment should include these elements:

1. Location of the operation, with a brief description of the goals of the mission and the other teams involved, with their needs and resources (Table 23-9).
2. Locations of all surrounding hospitals and medical care facilities, such as designated burn and trauma centers, with phone numbers to facilitate communication. Local EMS numbers should be listed.
3. Helicopter flight plan. Before the mission, it should be ascertained that a helicopter is available and that there is an acceptable landing zone (LZ) for day or night conditions. This should include the exact global positioning system (GPS) coordinates of the LZ. Obstructions and debris should be cleared prior to the mission (Table 23-10 and Fig. 23-29).
4. Weather. Factors to be evaluated include temperature, rain, wind, humidity, wet-bulb temperature (T_W), and windchill. Sunrise and sunset times should be recorded and logged. The T_W is the lowest temperature to which air can be cooled by the evaporation of water into the air at a constant pressure, so it reflects the limit to which a person can shed heat

TABLE 23-9. Sample Form to Provide Operational Information

Location						
Type of operation	Hostage #	Suspect #	Warrant #	Protection #	Open terrain search #	Terrorist #
Other teams	Tactical	EMS/medics	K9	Patrol	Detective	FBI/other

TABLE 23-10. Sample Form for Helicopter Information

Helicopter	Obstructions? LZ cleared prior to mission start?	LZ coordinates	
Landing zone (LZ)	Address:	Latitude	Longitude

Figure 23-29. Air ambulance helicopter preparing for tactical casualty evacuation. (Courtesy Lawrence E. Heiskell, MD.)

through sweating in a hot environment. The T_W is used to determine fluid requirements and the need for work–rest cycles. The weather-related components of the MTA are used to determine the appropriate uniform to wear and the shelter required to prevent overheating or hypothermia (Table 23-11). Water sources should be recorded prior to the mission as part of the MTA.

5. Plant and animal threats. Plant exposures, especially to poison ivy and poison oak, are common to snipers. Snakebites are common to team members working with police dogs. Anticipated animal threats should be recorded, along with telephone numbers for the police veterinarian, animal control, and poison control, including sources of antivenom (Table 23-12).

Forms and Documentation

Medical records should be kept for the team and for anyone treated or evaluated as a TEMS patient. Records should be stored for a minimum of 10 years and have proved to be indispensable as defense documents in several antipolice liability lawsuits. Without a medical record, there is no proof that appropriate medical care was given.

THE TACTICAL MISSION

Each mission has a number of phases[55,94]:

1. Warning order (issued when the tactical team is first requested, and establishes the situation and chain of command)
2. Gathering of intelligence
   a. Building intelligence (targets location and surrounding areas and includes avenues of approach, escape routes, and rally points, as well as natural and man-made obstacles, fields of fire, opportunities for cover and concealment)
   b. Suspect or hostage intelligence (as detailed as possible)
   c. Medical threat assessment (complete)
3. Operation order
4. Briefing phase
   a. Detailed planning
   b. Detailed briefing
   c. Equipment selection
   d. Move to staging
5. Execution phase
   a. Entry
   b. Secondary search
   c. Transfer to arrest team and investigation team
   d. End of mission
6. Debriefing phase
   a. All persons, weapons, equipment, injuries, shots fired, and ammunition must be accounted for.
   b. Any problems must be discussed.

In general, a tactical mission follows this order, although it may differ somewhat between agencies and missions. Proper handling of each point is required in order for a mission to flow seamlessly. Without proper intelligence, a mission becomes hazardous.

RESERVE PROGRAMS

The ways a tactical medical team is utilized by a law enforcement agency can differ widely, especially between the East Coast and the West Coast. For example, in the western United States, especially California, Arizona, Nevada, Washington, Utah, and Oregon, there are many reserve programs in the police and sheriff's departments.[63] In these programs, the tactical medical provider has additional, formal law enforcement training, such as found in the Peace Officer Standards of Training (POST) program in California. This program allows the provider to be a sworn peace officer, bringing about an enhanced comfort level for the department and potentially mitigating some issues of civil liability. On the East Coast, reserve opportunities are less common, and medical providers typically serve as auxiliary units borrowed from traditional fire and EMS agencies. The liability issues and expenses in this type of relationship are often resolved via a written "memorandum of understanding" between the participating agencies.[82]

TABLE 23-11. Sample Form for Weather-Related Information

Temp high	Wet-bulb temp	<60	60–78	78–82	82–85	85–88	88–90	>90		Sunrise: AM
Temp low	H_2O qt/hr	0.5	0.5	0.5	0.5–1.0	1.0	1.0–1.5	2.0		Sunset: PM
Rain %	Rest Min/Hr	0	0	0	10	15	30	40		Night ops:
Wind: MPH	Cold casualties	Y/N							Work cycles	Duration:
	Heat casualties	Y/N							Yes/No	
Humidity %	Uniform adjustments	Y/N							Shelter: Y/N	Location:

TABLE 23-12. Animal and Plant Threats

Animal Threats

Yes/No	Animals present? Yes/No	Police dog? Yes/No #			
	Types of animals	Number:	Do you anticipate wild animals?	Yes/No	What type?
	Poisonous snake exposure: Yes/No		Veterinarian address:		Vet phone:
	Animal Control: 973-470-2242	Poison Control: 800-222-1222			

Plant Threats

Yes/No	Exposure to poisonous plants likely?	Yes No	Type
	Tecnu or Ivy Block available?	Yes No	
	Uniform adjustments needed?	Yes No	Recommendations:

Selecting appropriate providers must be accomplished through interviews, psychological testing, background investigations, and physical fitness testing. The tactical team leaders should use a careful approach in the selection process for each candidate, just as they do for other members of the team.[58]

MILITARY COMBAT FIELD UNITS

Field units vary significantly with the mission, service, and threat (Fig. 23-30). Most often, medical care is provided in the open, or in as secure a location as possible. It is done on the ground, on a table, in the back of a Humvee, or on an aircraft or a watercraft. Specific types of shelter and equipment are available, most often in the far-forward care under fire and the tactical field care phases, but generally the equipment is as noted earlier.

When CBRN threats or high explosives are added to the scenario, the forward elements are the same, with the exception of the appropriately protective clothing. Once evacuation occurs, the injured team member is taken to a standard unit with the capability for decontamination and treatment, much as is done in the civilian setting. Most larger military transport planes have the capability to accept a critical care air transport team (CCATT), which has a physician trained in critical care (emergency medicine, anesthesiology, internal medicine), a critical care nurse, and a respiratory technician. Most larger Navy ships are equipped with fully functioning operating rooms and intensive care units, or they can expand to provide this service quickly. All the services have basic medical units, from the self-aid or buddy-aide, to an aid station (with a physician), to forward resuscitative systems (surgical and nonsurgical), to surgical companies, combat surgical hospitals, and their equivalents. All services also have teams that are available to move far forward to provide surgical capabilities quickly should the need arise. These units can operate on any semi-flat surface, do resuscitative surgery, and package the patient for expeditious transport for further care.[13]

Figure 23-30. Law enforcement tactical medical team during a training exercise. (Courtesy Lawrence E. Heiskell, MD.)

Uniforms and Personal Protective Gear

The standard military tactical operator carries between 40 and 60 pounds of equipment when heading into battle. For longer

missions, gear is heavier. Clearly, the operator needs to be in excellent physical condition, and the mission commander must proactively manage nutrition, hydration, and exertion levels.

From anecdotal reports, the military has seen a significant decrease in truncal injuries because body armor has improved. The medical personnel have noted that the insurgent combatants in Operation Iraqi Freedom have changed their IEDs to target the head, neck, and extremities more than the whole body. Continued development is underway to produce body armor that will protect against higher-energy weapons, protect extremities, and be lightweight and flexible enough to allow fieldwork.

As protective clothing is not removed in the field, the medic must work under and around it. Vigilance is required to check all body areas for hidden wounds. This is clearly more difficult in the field, in the dark, with sound and light restrictions, and with clothing in place than when the patient is undressed on the trauma table.

Because the medic carries all the usual combat equipment, medical equipment is additional. Stethoscopes are usually left behind, as the medic cannot use the earpieces under the helmet, the environment is not conducive to being able to hear anything, and the victim is usually wearing body armor. Advanced medical gear is usually found on the MedEvac vehicle, or further to the rear where it is safe enough to remove protective equipment and further evaluate and treat the patient.

TABLE 23-13. Sample Curriculum for Basic Tactical Medicine (BTM) Training

Day 1	Administration and Introduction
	Introduction to Tactical Medicine
	Tactical Medical Equipment
	Tactical Equipment
	Team Concepts and Planning
	Slow and Deliberate Team Movement
Day 2	Introduction to Tactical Pistol
	Medical Aspects of Chemical Agents and Distraction Devices
	Forced Entry Techniques
	Dynamic Clearing Techniques
Day 3	Operational Casualty Care
	Wound Ballistics
	Hemostatic Techniques and Dressings
	Team Health Management
	Medical Aspects of Clandestine Drug Labs
	Tactical Medical Scenarios
Day 4	Introduction to MP5 Submachine Gun
	Special Operations Aeromedical Evacuation
	Medical Management of K-9 Emergencies
	Disguised Weapons and Street Survival
	Medical Threat Assessment
Day 5	Written Exam
	Safety Briefing
	Tactical Medical Scenarios

From International School of Tactical Medicine, copyright 1996–2005 (see www.tacticalmedicine.com).

EDUCATION AND TRAINING PROGRAMS

Tremendous advancement in tactical medicine education over the last decade has resulted in numerous training programs, many providing formal continuing medical education (CME) credits. These courses focus on the core issues of tactical medicine. A unique aspect of tactical medicine is application of ALS in an austere environment. The traditional approach to providing EMS is often not feasible in a tactical situation.[89]

Cost-effective training is available and should be afforded to all involved medical personnel, including prehospital care providers and physicians, who should be trained to the highest level possible. Such training provides emergency medical personnel with an understanding of tactical procedures and an appreciation for why some routine prehospital care techniques may not be appropriate in the tactical environment.[53,57]

Tactical medicine training needs to be as realistic as possible with live teaching scenarios in full tactical gear. This allows the medical providers to more fully understand the unique aspects of law enforcement tactical operations and the roles and responsibilities of each team member, along with the integration and application of EMS. With an established body of knowledge and skills, graduates of such training programs are better prepared to effectively perform as safe tactical medical providers.[64]

The International School of Tactical Medicine, a law enforcement agency program, is based at the Palm Springs Police Department Training Center in Palm Springs, California. This school has conducted high-quality realistic tactical medical training courses since 1996 and offers a 2-week, 80-hour program. The training and educational courses are designed for the U.S. military and federal and local law enforcement agencies to enhance their provision of medical care in the tactical environment. The standard curriculum for each course can be seen in Tables 23-13 and 23-14. Both the basic and advanced courses offer category 1 CME credit through the American College of Emergency Physicians. The school is approved by the State of California Commission on Peace Officer Standards and Training (POST) (see www.tacticalmedicine.com).

The Tactical EMS School of Columbia, Missouri, offers two TEMS educational programs. The Essentials of Tactical EMS is the basic entry-level course. The Tactical EMS Field Operations course is designed to augment the training offered in the essentials course and is scenario-based teaching with a focus on casualty care in the tactical environment (see www.tacticalspecialties.com).

The Counter Narcotics Tactical Operations Medical Support program (CONTOMS) at the Casualty Care Research Center (CRC) is a multidisciplinary injury-control research and training facility in Bethesda, Maryland. It is based in the Department of Homeland Security, Federal Protective Service, Special Operations Division, forming the Protective Medicine Branch (see www.casualtycareresearchcenter.org).

THE FUTURE OF TACTICAL MEDICINE

Tactical medicine will continue to grow as a medical discipline, and emergency medicine is the ideal specialty to lead its development. Since the fall of 2002, under leadership of the International Tactical EMS Association (ITEMS), a multidisciplinary group of subject matter experts has been working to achieve consensus on development of a standardized national curriculum for tactical medicine training.[19] Emergency medicine residents and surgeons with no special interest in participating in tactical medicine should have an understanding of this discipline, as they may well have the opportunity to treat an injured

TABLE 23-14. Sample Curriculum for Advanced Tactical Medicine (ATM) Training

Day 1	Administration and Introduction
	Pediatric Trauma Management
	Trauma Anesthesia
	Building Clearing Techniques Review
	Tactical Medical Scenarios
Day 2	Range Advanced Pistol–MP5
	Advanced Airway Management
	Advanced Airway Management Skills Stations
	Environmental Injuries
Day 3	WMD Biological Weapons Part 1
	WMD Biological Weapons Part 2
	Medical Issues of Less-Lethal Weapons
	Low Light Tactics and Team Movement
	Tactical Medical Scenarios
Day 4	Pistol–MP5 Field Courses
	Explosive Entry Demonstrations
	Medical Management of Blast Injuries
	WMD Chemical Weapons
	WMD Nuclear and Radiation Injuries
Day 5	Written Exam
	Safety Briefing
	Tactical Medical Scenarios

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operator or victim of violence associated with a tactical law enforcement action.[24,40]

There is a need for research into the unique aspects of civilian tactical medicine, such as injury prevention during operations, methods of ensuring optimal mental and physical preparedness for tactical operators, and evaluation of various standard EMS therapies for their feasibility and efficacy in tactical scenarios.

Figure 23-31. Tactical medics provide medical support under any and all operative conditions. (Courtesy Lawrence E. Heiskell, MD.)

Tactical medicine is wilderness medicine taking place in both the urban environment and some of the most remote places on earth.

In these times, when the threat of violence to civilians in our society is at its greatest, we rely on our law enforcement professionals and the military to do all they can to keep us safe and protected. It is the role of tactical medics, providing medical care under any and all operative conditions (Fig. 23-31), to give back to these professionals by ensuring that someone is there to care for them if they are injured in the course of doing their duty.

The references for this chapter can be found on the accompanying DVD-ROM.

Wilderness Orthopaedics

24

Julie A. Switzer, Thomas J. Ellis, and Marc F. Swiontkowski

Musculoskeletal and soft tissue injuries account for 70% to 80% of injuries that occur in a wilderness setting.[4,10] Being able to identify and provide initial, acute treatment of the most common types of injury is particularly important. In the initial management of a musculoskeletal injury, the following must be considered: the etiology and time of the injury, the direction of the causative force in relation to the individual or limb, and the environment where the accident occurred. These factors may indicate the severity of the injury and help determine examination and treatment priorities that can affect outcome. Special considerations for injuries to the skeletal system that occur in the wilderness include the effect of weather (exposure to wind, cold, or heat), lack of usual devices for stabilization of bone or joint injuries, and increased time to initiation of a victim's definitive care.

Stabilization of a victim's cardiovascular and pulmonary status is critical. Once this has been accomplished, examination of the musculoskeletal system should be undertaken in a sys-

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Stabilization of a victim's cardiovascular and pulmonary status is critical. Once this has been accomplished, examination of the musculoskeletal system should be undertaken in a sys-

tematic manner. Careful initial attention should be devoted to the spine. After the cervical, thoracic, and lumbar spine are evaluated and stabilized, the focus is brought to bear on the pelvis and extremities.

SPINAL INJURIES

Cervical Spine

In the wilderness, cervical spine fractures or dislocations often result from falls from a height or from high-velocity ski or vehicle crashes. Because head and cervical spine injuries are highly associated, victims with significant head injuries should be considered to have cervical spine injuries, especially if the individual is unconscious. Ideally, a person with a suspected cervical spine injury is placed on a backboard with neck immobilization and promptly evacuated. When transporting an individual with a cervical spine fracture or dislocation, the neck must be stabilized to prevent further injury. Approximately 28% of persons with cervical spine fractures also have other spinal fractures.[3] Therefore, the person providing care must protect the entire spine.

When a cervical spine injury is suspected, the field examination involves grading motor strength, documenting sensory response to light touch and pinprick, and noting the presence or absence of the Babinski reflex. When appropriate supplies are available, a rectal examination should be done. Complete lack of tone and failure of the sphincter muscles to contract when pulling on the penis or clitoris (the bulbocavernosus reflex) indicate spinal cord injury.

Neurologic deficit often results from cervical spine fracture. Complete neurologic injury from the occiput to the C4 level is usually fatal because of paralysis of the respiratory muscles. The corollary to this is that surviving victims generally have partial deficits or are neurologically intact. Axial cervical spine fractures may result from flexion forces (most common), extension forces, rotational forces, or a combination of these. They most commonly occur at C5-6.[3] Fracture of the C1-2 complex results from axial loading (a C1 ring fracture, or Jefferson fracture) or from an acute flexion injury (a C2 posterior element fracture, or hangman's fracture). A pure flexion event may dislocate one or both posterior facets, producing neck pain and limitation of motion. Because the interspinous ligament is ruptured, in this fracture dislocation victim transport must be done with the neck rigidly immobilized to reduce the risk of posterior motion in this highly unstable condition.

Fractures and dislocations may result in neurologic insult distal to the bony injury. Because flexion injuries are the most common cervical spine injuries, the neurologic deficit is generally an anterior cord syndrome. In this setting, the victim suffers complete motor and sensory loss but retains proprioception.

Thoracolumbar Spine

Thoracolumbar spine fractures occur most frequently at the thoracolumbar junction. Because the thoracic spine is well splinted by the thoracic cage, when an axial or flexion load is applied, the ribs diminish forces on the thoracic vertebral bodies and transmit the force to the upper lumbar levels. In the wilderness, falls from significant heights or a high-velocity sporting vehicle crash may produce these fractures (Fig. 24-1). Thoracolumbar spine fracture may also be associated with major hindfoot fractures (particularly of the calcaneus). These injuries commonly occur when there is an axially loaded injury, such as a fall onto the lower limbs from a height. With the identification of unilateral or bilateral calcaneus fracture, a concomitant spine fracture should be assumed to have occurred (Fig. 24-2). Therefore, an individual who sustains a calcaneus fracture as the result of a fall from a height generally should be transported under spinal precautions.

When a spine fracture is suspected, a careful neurologic examination should be performed as part of the secondary

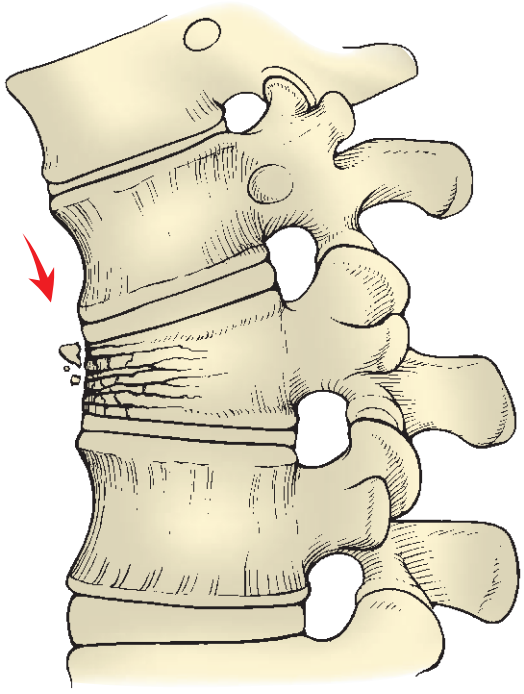


Figure 24-1. Wedge compression fracture from axial or flexion loading at the thoracolumbar junction.

Figure 24-2. Calcaneus fracture, sustained as a result of a fall from a height.

Figure 24-3. Bilateral sacroiliac joint dislocations and a pubic symphysis disruption make this pelvis injury unstable both anteriorly and posteriorly. Significant care should be exercised in transporting an individual with suspected pelvic ring disruption.

Figure 24-4. Three-dimensional computed tomographic scan of an acetabular fracture. One of the fracture fragments along the inner pelvic brim probably created a small bladder injury in this patient.

survey, with close attention paid to the dermatomal response to light touch and pinprick, motor function, and the presence or absence of cord level reflexes. Because significant fluctuations in sympathetic tone may occur, the rescuer should monitor blood pressure and body temperature, taking appropriate steps to cool or warm the victim. If evacuation cannot be performed immediately, hemodynamic and neurologic function should continue to be noted and documented. When there is significant head injury, a spinal injury should be assumed to be present. The victim should be logrolled, maintaining perfect spinal alignment, and carefully placed on a backboard. The scoop stretcher may be used in this situation (see Chapter 34).

PELVIC INJURIES

A study of mortality in the wilderness setting in Pima County, Arizona, between 1980 and 1992 demonstrated that most deaths occurred as a result of falling or drowning.[5] Pelvic fractures generally occur with a fall from significant height, high-velocity ski accident, or vehicle crash. The direction of force is directly related to the fracture and influences definitive management.[16,18] Pennal and colleagues[11] and Tile[18] divide pelvis fractures into anteroposterior (AP) compression injuries, lateral compression (LC) injuries, and vertical shear (VS) injuries. In addition, there are simple, nondisplaced inferior or superior ramus fractures and avulsion fractures. On clinical examination, these rami and avulsion fractures are generally appreciated as areas of tenderness without instability. Lateral compression injuries are usually stable, with impaction of the posterior structures. They are usually not emergent surgical or medical situations. AP compression injuries demonstrate anterior instability, palpable ramus fractures, or pubic symphysis gapping (Fig. 24-3). These fractures, which may include acetabular fractures, are often accompanied by bladder, prostate, or urethra injury (Fig. 24-4).

If a pelvic fracture is suspected, it should be determined whether there is posterior injury to the pelvic ring, as this is associated with significant hemorrhage, neurologic injury, and, ultimately, mortality. Posterior ring fractures are identified by instability of the pelvis associated with posterior pain, swelling, and ecchymosis. This victim should be immediately evacuated on a backboard, taking care to minimize leg and torso motion.

Figure 24-5. SAM Sling.

Hemodynamic instability may occur with pelvis fractures, especially if the injury is the result of translational or shear forces, or if the posterior pelvis elements are primarily involved. Bleeding associated with a pelvis injury is usually from cancellous bone at fracture sites, retroperitoneal lumbar venous plexus injury, or, rarely, pelvic arterial injuries. Medical antishock trousers (MAST), the portable SAM sling, or even a bed sheet wrapped around the pelvis of an individual with a suspected unstable pelvic fracture may provide stability and accomplish adequate tamponade of bleeding from the fracture.[12] The applied sling belt or similar contrivance should be left in place until definitive care is available (Fig. 24-5).

EXTREMITY INJURIES

Physical Examination

The physical examination should address circulatory, nerve, skeletal, and joint function.

Circulatory Function. Penetrating or blunt trauma can injure the major vessels that supply the limbs. Fractures can produce injury by direct laceration (rarely) or by stretching, which can produce intimal flap tears. These intimal injuries can occlude distal flow or lead to platelet aggregation and delayed occlusion. Thus, an examination of circulatory function should be done prior to the victim's arrival at the definitive care center. The color and warmth of the skin or distal extremity should be assessed; pallor and asymmetric regional hypothermia may indicate vascular injury. In the upper extremity, the brachial, radial, and ulnar arteries should be palpated. In the lower extremity, the femoral, popliteal, posterior tibial, and deep peroneal arteries should be palpated. If blood loss, hypothermia, or obesity makes these pulses difficult to assess, temperature and color must be relied on to determine vascular integrity. Any suspected major arterial injury mandates immediate evacuation after appropriate splinting.

Nerve Function. Nerve function may be impossible to assess in an unconscious or uncooperative person. In the conscious person, the results of light touch and pinprick tests should be carefully documented. For spinal and pelvic injuries, the dermatomal distribution of spinal nerves is assessed, and muscle function is evaluated by observing active function and by grading the strength of each muscle group against resistance. If possible, once the victim's condition has been stabilized, nerve function to the distal extremities is established. These initial findings should be compared periodically with repeat examinations during transport. Any change, and, in particular, any deterioration in condition should be noted.

Skeletal Function. The long bones of the lower extremity serve as the major structural supports for locomotion, whereas those of the upper extremity stabilize the soft tissues, enabling positioning of the hand in space. A visible angular deformity reveals a fracture; palpable crepitus confirms the diagnosis. The care provider in the field should perform appropriate splinting after aligning the limb using axial traction. After noting the degree and orientation of the limb's position when the victim is found, there should be no delay in aligning and splinting fractures. Distinguishing joint injuries and intra-articular or very proximal or distal fractures must wait for the definitive care facility, where radiologic studies can be done. Similarly, distinguishing a wrist or ankle ligamentous injury from a fracture is not required for initial treatment.

Joint Function. Muscle forces act across joints to improve the position of the lower limbs for ambulation and the hand for handling objects. Each joint has a normal range of motion and a certain minimal function to allow for stability. Making the diagnosis of a joint injury in the field allows appropriate splinting and prevents further damage during transport.

The palpation of long bones begins distally and proceeds across all joints. A splint should be applied if there is palpable crepitus, swelling, deformity, or a block to motion. If the victim can cooperate, each joint is taken through an active range of motion to quickly locate any injury. When this is not possible, passive motion of each joint is evaluated after palpation for crepitus and swelling.

Any dislocations are reduced after completing the neurocirculatory examination. This generally relieves the victim's discomfort considerably. Once reduction of the dislocation has occurred, stability is evaluated by careful, controlled motion. Joints with associated fractures or interposed soft tissues are frequently unstable after reduction. Great care is required in applying splints to prevent redislocation or further soft tissue injury. A report of details of the reduction maneuver, including orientation of the pull, amount of force involved, amount of sedation, and residual instability of the joint, should be provided to the definitive care physician.

Splinting Techniques

A victim with suspected cervical or thoracolumbar spine trauma should be transported on a hard surface. Backboards or scoop stretchers (see Chapter 34) are most effective, but improvisation with hard pieces of wood, fiberglass, or straight tree limbs lashed together may be needed. If cervical spine injury is suspected, a roll of clothes or a water bottle can be placed as high as the victim's mid face on either side of the head to prevent rotational movement. Tape applied from the supporting stretcher across the objects and the victim's forehead adds stability. Any victim with a suspected major pelvis injury is transported in a similar fashion, stabilizing the pelvis with a circumferential sheet or piece of clothing, and holding the lower extremities as immobile as possible, with the knees slightly flexed.

Many different extremity splints are available for use in the wilderness setting. These splints are lightweight, compact, and easy to use. They are designed to provide traction through the injured extremity, while using the intrinsic properties of the splint and the injured limb to apply the traction. With proper splint application, the injured limb can be immobilized securely in a functional position until definitive care is reached.

Air splints may be of some benefit, but they are generally manufactured in one shape. Especially in the setting of injured tissues and in environments that might include wide temperature variability, these splints can cause untoward damage to an already injured extremity. Therefore, in more extreme conditions, an air splint is used only if it has an automatic adjustment valve for heat and altitude. Also, these splints should be stored in a minimally inflated state when the temperature is below freezing, to prevent ice from creating a means for the splint material to freeze or adhere together. Beaded vacuum splints can also be used. However, temperature and altitude considerations can make adequate and consistent inflation less reliable. When beaded vacuum splints and air splints are used, vigilance is required to ensure that no excessive pressure is applied to already injured soft tissues.

Upper extremity splints may also be made from plaster or fiberglass, which can be applied over a soft cotton roll. Lightweight fiberglass splints, such as Orthoglass (Smith and Nephew), are easy to use and effective in the initial management of these injuries. These splints are pre-padded and can be applied with either cold or warm water. The warmer the water, the faster the fiberglass sets and the greater the exothermic reaction. Hot water should be avoided because it may generate an

Figure 24-6. Short leg splint, well padded and made with fiberglass stirrups. This type of immobilization, if materials are available, may be indicated for tibia–fibula, ankle, and severe foot injuries.

Figure 24-7. SAM splint.

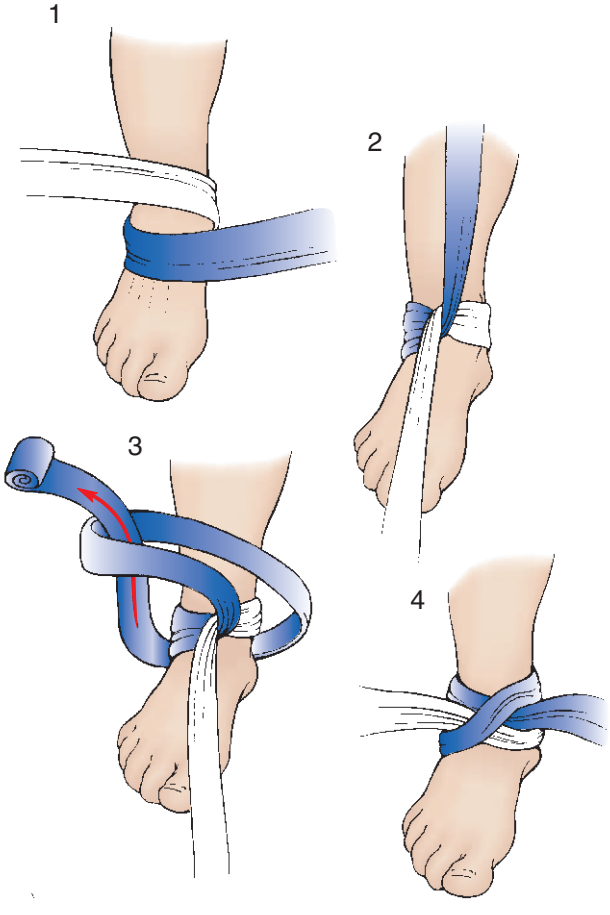


Figure 24-8. Improvisation of an ankle wrap to be used for traction.

excessively exothermic reaction and possibly burn the skin. The fiberglass is immersed in water, the excess water is gently squeezed out, and the splint is applied. An elasticized bandage helps hold the splint where desired until the fiberglass is hard (Fig. 24-6). Air splints, when inflated, can adequately splint the upper extremity in this position. Wooden or metal splints, custom made or improvised, also can be used to stabilize an injured extremity.

Hand splints are applied with the metacarpophalangeal (MCP) joints flexed 90 degrees and the interphalangeal (IP) joints extended. This position places the collateral ligaments at maximal length and prevents later joint contracture. Wrist or forearm splints are applied with the wrist in a neutral position—excessive wrist flexion or extension might compromise median or ulnar nerve function in an already compromised limb. The elbow is positioned in a splint or sling at 90 degrees, if possible.

For shoulder fractures or dislocations, a commercially available sling or improvised triangular bandage should be used to take the weight of the arm off the injured structures. Although it may be difficult to place an injured elbow in 90 degrees of flexion and neutral pronation–supination, the upper extremity should be splinted in the position of function whenever possible.

For the lower leg, air splints provide adequate immobilization of tibia or fibula fractures and of ankle fractures and dislocations. Splints made from plaster or fiberglass may be applied over cotton padding with elasticized wraps (Fig. 24-6). The SAM splint, an excellent first-aid item that may be molded to immobilize a wide variety of injuries, provides stability and strength through its aluminum and foam core (Fig. 24-7). The aluminum structure can be bent into three configurations to provide different degrees of stability, flexibility, and immobilization. The ankle is held in neutral position and the splint applied firmly. For transport, the lower extremity is positioned with the hip and knee extended and the ankle in neutral position. Victims with unstable lower extremity fractures or dislocations are transported in the recumbent position with the afflicted limb elevated.

For hip or femur fractures or dislocations, traction is applied whenever possible, improvising when necessary (Fig. 24-8). For suspected hip, femur, or knee injury, a Thomas splint with a Spanish windlass or the Kendrick traction device (a lightweight,

Figure 24-9. Slishman splint.

Figure 24-10. A femoral splint applied against the ischial tuberosity provides traction for this subtrochanteric femur fracture.

more conveniently packaged splint) has traditionally been used. Similar, but lighter-weight and more compact splints include the Slishman splint and Reel splint (Fig. 24-9). The design principles of the Slishman splint are similar to those of the Thomas and Kendrick splints. The ring of the Thomas splint rests against the victim's ischium and pubis, whereas the Slishman splint applies traction through the pubis (Fig. 24-10). The Kendrick splint employs a rigid, retractable pole and cuffs. The Reel splint uses a multi-angle and hinge system to provide traction in anatomic regions that are especially difficult to access (Fig. 24-11). If commercial splints are unavailable, the injured leg is strapped to the noninjured leg, with a tree limb or walking stick placed between them. If possible, the victim is transported on a backboard.

Open Fractures

Recognizing an open fracture is imperative; without prompt surgical treatment, the incidence of osteomyelitis in this setting is high.[7] In an open fracture, the fractured bone communicates with a break in the skin. With subcutaneous bones (e.g., tibia), open fractures are easily identified, but with other bones (e.g., humerus, femur, pelvis) that have more surrounding soft tissue, identification is more difficult because the fractured bone end usually retracts once it punctures the skin and is then covered by soft tissue. A laceration near a fracture may be an indication of an open fracture. Most open fractures persistently ooze blood or fat globules from the laceration, which may facilitate diagnosis.

General care of an open fracture outdoors depends on evacuation time. Open fractures require prompt operative irrigation, debridement, and stabilization. If evacuation can be completed within 8 hours, realign the fracture, administer a broad-spectrum antibiotic, and splint the extremity. If bone ends extrude through the skin, cover the exposed bone with a povidone-iodine solution–soaked gauze sponge, splint the extremity, and arrange for prompt evacuation. If evacuation time exceeds 8 hours, in addition to antibiotic administration and splinting, irrigation and debridement in the field may be attempted. Antibiotic options are listed in Box 24-1.

Amputation

In the wilderness, the amputation victim requires immediate evacuation. Control hemorrhage using direct pressure; tourniquets are virtually never indicated. Without cooling, an amputated part remains viable for only 4 to 6 hours; with cooling, viability may extend to 18 hours. Cleanse the amputated part with saline or water, wrap it in a moistened sterile gauze or towel, place it in a plastic bag, and transport it in an ice-water mixture. Do not use dry ice. Keep the amputated part with the victim throughout the evacuation.

A

B

Figure 24-11. **A and B,** Reel splint.

Box 24-1. Antibiotic Options

INTRAVENOUS
Cefazolin (Ancef) 1 g q8h and gentamicin (5 mg/kg) q24h
or piperacillin with tazobactam (Zosyn) 3.375 g q6h

INTRAMUSCULAR
Ceftriaxone (Rocephin) 1 g q24h
Oral ciprofloxacin 750 mg bid and cephalexin (Keflex) 500 mg qid

WATER EXPOSURE
Ciprofloxacin 400 mg IV or 750 mg PO bid; or a sulfonamide and trimethoprim conbination (Bactrim DS: 800 mg sulfamethoxazole and 160 mg trimethoprim) with either cefazolin (Ancef) 1 gm IV q8h or cephalexin (Keflex) 500 mg PO q6h

DIRT OR BARNYARD
Add penicillin 20 million units IV qd or 500 mg PO q6h.

IF PENICILLIN ALLERGY
Use clindamycin 900 mg IV q8h or 450 mg PO q6h in place of penicillins and cephalexin (Keflex).

ALTERNATIVES
Erythromycin 500 mg q6h or amoxicillin 500 mg PO q8h

Compartment Syndrome

A compartment syndrome begins when locally increased tissue pressure reduces arterial and, ultimately, capillary blood flow to a muscle compartment. When local blood flow is unable to meet metabolic demands of the tissue, ischemia ensues. In the wilderness, a compartment syndrome most frequently occurs in association with a fracture, crush injury, or severe contusion. It can also occur when the victim has been lying for some time across an extremity so that the body weight occludes the arterial supply. Elevated local tissue pressure (compartment pressure within 10 to 20 mm Hg of diastolic arterial blood pressure) can also occur with acute hemorrhage or after revascularization of an ischemic extremity. Because perfusion pressure is the most important variable in the development of compartment syndrome, hypotension can increase the risk of a compartment syndrome.

Compartment syndrome can occur in the thigh, hand, foot, and gluteal regions. It is most common, however, in the forearm and, especially, the lower leg, because of the tight fascia in these regions. The conscious victim complains of severe pain out of proportion to the injury. The muscle compartment feels extremely tight, and applied pressure increases the pain. There may be deceased sensation to light touch and pinprick stimuli in the areas supplied by the nerves traversing the compartment. Stretching muscles within the compartment produces severe pain. The most reliable signs of a compartment syndrome are pain, tight compartments, hypesthesia, and pain on passive stretch. Pulselessness, pallor, and slow capillary refill may not be observed, even with a severe compartment syndrome.

Emergency evacuation is required when compartment syndrome is suspected. The victim must be definitively treated in the first 6 to 8 hours after onset to optimize return of function to the involved limb. Emergency fasciotomy, the treatment of choice, relieves the pressure. If a compartment syndrome develops and evacuation cannot occur within 8 hours, it must be decided whether the treating individual possesses the skill to perform a fasciotomy and whether it can be performed in an aseptic manner. Fasciotomies can convert a closed fracture into an open fracture and can provide a conduit for limb- if not life-threatening infection.

If a fasciotomy is to be done, antibiotics should be administered. In the forearm, the procedure usually involves making volar and dorsal incisions and splitting the underlying fascia. In the lower leg, the procedure usually involves making two long incisions, one on the medial aspect and the other on the lateral aspect of the leg, and splitting, in a vertical fashion, the compartmental fascia in each of the four lower leg compartments (Fig. 24-12).

Fasciotomies for compartment syndrome that cannot be done within 12 hours of the syndrome's development ought not to be undertaken. A retrospective analysis of individuals who underwent late release for compartment syndrome (more than 35 hours after the injury) demonstrated significant complication

Figure 24-12. This photograph of a compartment syndrome demonstrates the degree of intracompartmental swelling that can occur in the leg. After fasciotomy for compartment syndrome, wound closure is often not possible, and skin grafting may be necessary.

and amputation rates. Therefore, even in an urban trauma hospital, delayed compartment release for compartment syndrome is not recommended.[8]

RICE Principle

The general principle in the acute management of extremity injuries is rest, ice, compression, and elevation (RICE). For unstable fractures, immobilization is also indicated. Avoid heat for the first 72 hours after injury. Chemical cold packs work well, but cold packs made from ice or snow suffice. If cold packs are unavailable, the extremity can be immersed intermittently in a cold mountain stream. If ice is used, mix some water in a bag with the ice to more evenly distribute the cold. The cold pack to the injured area may be held in place with an elasticized bandage. A thin piece of fabric is placed between the cold pack and the victim's skin to prevent frostbite. The ice is applied to the elevated extremity (above the level of the heart) for 30 to 45 minutes every 2 hours. A compressive dressing also helps decrease swelling but should not be used if development of a compartment syndrome is possible. In this situation, keep the limb at the level of the heart and avoid compressive dressings.

UPPER EXTREMITY FRACTURES

Clavicle

Fracture of the clavicle usually occurs in the middle or lateral third of the bone and is associated with a direct blow or with a fall onto the lateral shoulder (Fig. 24-13). Clavicle fractures are common with snow skiing and mountain bike riding. The victim complains of shoulder pain, which may be poorly localized. Arm or shoulder motion exacerbates the pain. To localize the problem, gently palpate the clavicle to identify the area of maximal tenderness. The presence of crepitus at the clavicle confirms the diagnosis. Although rare, a clavicle fracture can be associated with a pneumothorax if the cupula of the lung is punctured; therefore, auscultate the chest for breath sounds. Shortness of breath and deep pain on inspiration increase suspicion for a pneumothorax.

Clavicle fracture may also be accompanied by injury to the brachial plexus, axillary artery, or subclavian vessels. In an

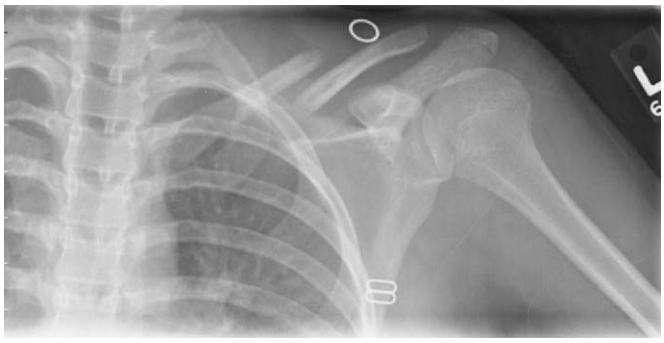


Figure 24-13. A 13-year-old sustained a left clavicle fracture in a boating accident. Significant shortening at the fracture site ultimately warranted open reduction and internal fixation.

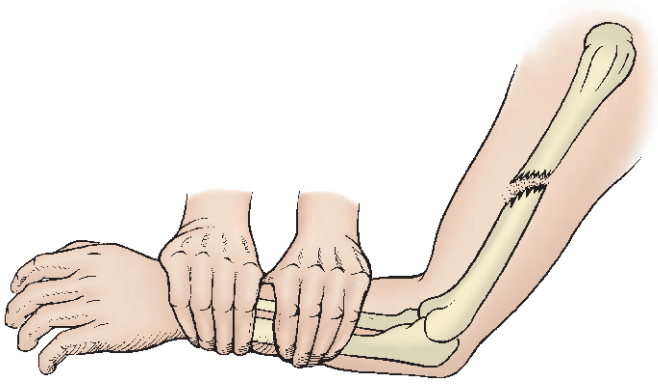


Figure 24-14. To control pain, a fractured humerus should be stabilized manually until a splint can be applied.

"individual with a fractured clavicle, a thorough neurocirculatory examination of the affected extremity is performed, and the skin is examined carefully. Approximately 3% to 5% of clavicle fractures may be open because of the bone's subcutaneous location. The victim should be evacuated if there is a significant open wound, suspected pneumothorax, or nerve or vascular injury. Field treatment for a clavicle fracture consists of a figure-8 bandage or sling and judicious use of analgesics.

Humerus

Fracture of the shaft of the humerus may result from a direct blow or torsional force on the arm. This fracture frequently occurs with a fall, rope accident, or skiing accident. Fractures of the midshaft and junction of the middle and distal third of the humeral shaft violate the spiral groove path of the radial nerve. If there is arm pain with deformity and crepitus, the arm is stabilized and the sensory and motor function of the radial nerve is carefully checked as part of the overall neurocirculatory examination (Fig. 24-14). Radial nerve function is evaluated by checking sensation in the dorsal thumb web space, and MCP extension with the proximal and distal IP joints flexed. When fracture of the humeral shaft is suspected, an appropriate coaptation splint made of plaster, fiberglass, or wood is firmly applied with an elastic bandage on the medial and lateral sides of the humerus. A sling is useful for comfort. Acute reduction of the fracture is not routinely required.

Figure 24-15. Comminuted proximal humerus fracture.

Figure 24-16. This woman attempted to break her fall with her outstretched right upper extremity, resulting in a proximal humerus fracture and posterior shoulder dislocation. Relocation of this injury in the field is nearly impossible and should not be attempted.

Fracture of the proximal humerus is often difficult to differentiate from shoulder dislocation in the acute phase. The mechanism is frequently a high-velocity fall onto an abducted, externally rotated arm, or a direct blow to the anterior shoulder. The victim complains of severe pain around the shoulder with palpation or with any arm motion. Palpable crepitus confirms the diagnosis. Although the inclination to attempt an emergent reduction may be compelling (it may be mistaken for a shoulder dislocation), this fracture does not routinely require acute reduction; application of an arm sling is appropriate field management (Fig. 24-15).

Fracture-dislocation of the proximal humerus can also occur. Most dislocations are anterior. Anterior or posterior fullness, with crepitus on the injured side compared with the uninjured side, suggests the diagnosis. This is a more severe injury than a simple shoulder dislocation, so a very careful neurocirculatory examination should be performed. Any significant nerve or vascular injury should prompt evacuation to a definitive care center (Fig. 24-16).

Fracture of the distal humerus is frequently extra-articular in children and intra-articular in adults. Children generally sustain supracondylar fractures after falls from heights. Extension-type injuries are much more common than the flexion type, and they most commonly occur in children aged 4 to 8 years. Deformity, swelling, pain, and crepitus are present, and the diagnosis of fracture is fairly obvious. A careful neurocirculatory examination should be performed, focusing on the motor examination of flexion of the thumb and distal IP joint of the index finger, because injury to the anterior interosseus nerve, which supplies innervation to these muscles, is frequently associated with these fractures. If the radial pulse is absent, an attempt should be made to flex or extend the elbow while palpating the radial pulse. If the pulse improves, the limb is splinted in that position for transport. If the pulse does not improve and definitive care is more than an hour away, a reduction is performed. After available sedation is given, the supinated elbow is extended with gentle longitudinal traction. The fracture is reduced by flexing the elbow while maintaining longitudinal traction, and the elbow is splinted in 90 degrees of flexion. Evacuation should be performed promptly.

For the adult with elbow pain, crepitus, deformity, and swelling after a fall, the neurocirculatory examination is performed, and then a splint is applied with the elbow at 45 or 90 degrees of flexion, depending on the victim's comfort. Reduction should not be attempted without radiographic confirmation because crepitus is more often associated with a fracture than with a dislocation. Evacuation should be performed promptly if there is an open fracture or neurocirculatory deficit.

Subluxation of the radial head in children (nursemaid's elbow) occurs when a longitudinal pull is applied to the upper extremity (Fig. 24-17). The orbicular ligament partially tears, allowing a portion of it to slip over the radial head. An audible snap may be heard at the time of the injury. The initial pain from the injury subsides rapidly, and the child does not seem distressed but refuses to use the extremity. Any attempt to supinate the forearm brings a cry of pain and distress. If a definitive care center is nearby, the injury is splinted and evacuation is arranged. Otherwise, if the history and examination are consistent with the diagnosis, a reduction can be attempted. First the slightly flexed forearm is supinated; if this fails to produce the characteristic snapping sensation of reduction, the elbow is maximally flexed in supination until the snapping sensation occurs (Fig. 24-18). If the reduction is successful, the child is usually content and playing within 5 to 10 minutes, and no immobilization of the joint is indicated. If the reduction is unsuccessful, the child continues to avoid using the involved arm and should be evacuated for definitive care.

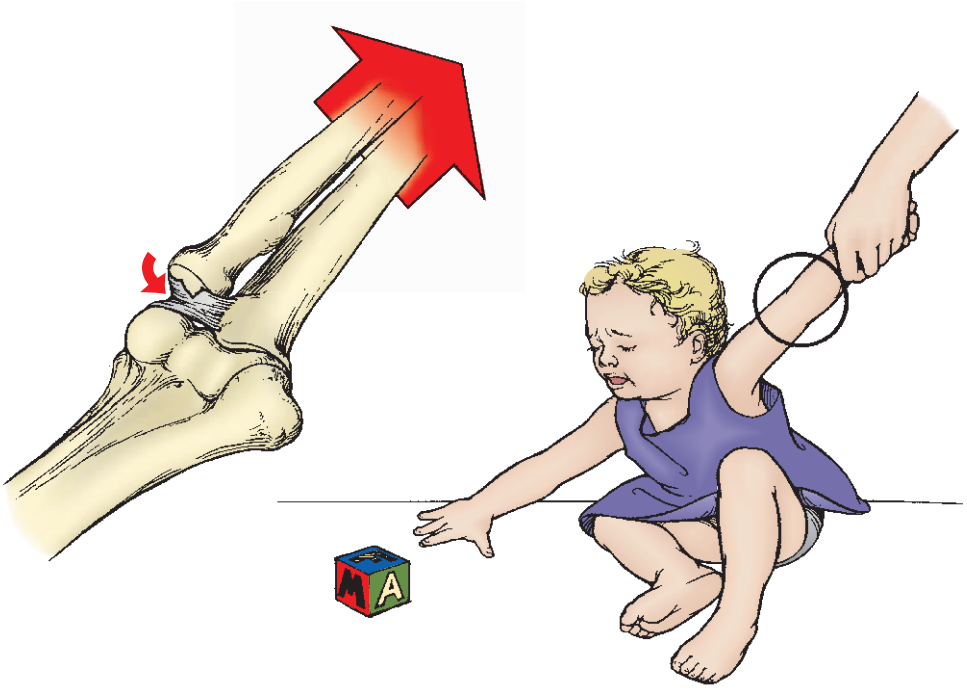


Figure 24-17. Nursemaid's elbow most commonly occurs when a longitudinal pull is applied to the upper extremity. Usually the forearm is pronated. There is a partial tear in the orbicular ligament, allowing it to subluxate into the radiocapitellar joint. (From Rockwood CA Jr, Wilkins KE, King RE [eds]: Fractures in Children, 3rd ed. Philadelphia, JB Lippincott, 1991.)

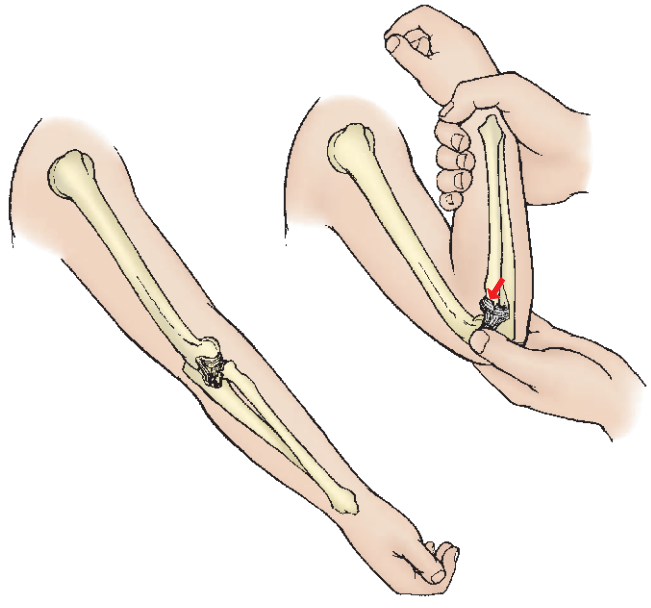


Figure 24-18. Reduction of nursemaid's elbow injury. *Left,* The forearm is supinated. *Right,* The elbow is then hyperflexed. The rescuer's thumb is placed laterally over the radial head to feel the characteristic snapping as the ligament is reduced. (From Rockwood CA Jr, Wilkins KE, King RE [eds]: Fractures in Children, 3rd ed. Philadelphia, JB Lippincott, 1991.)

Radius

Radial shaft fracture is commonly associated with a motor vehicle crash or an industrial accident but may occur with a fall involving angular or axial loading of the forearm. A radial shaft fracture may be associated with dislocation of the distal radioulnar joint (Galeazzi fracture). Therefore the wrist should be examined for tenderness, swelling, and deformity. The victim generally complains of pain, and deformity and crepitus are noted over the radial shaft after a fall or direct blow. Any arm motion exacerbates the pain. When both the radius and ulna are fractured, forearm instability is marked. The joint above (elbow) and the joint below (wrist) should always be examined for tenderness, crepitus, and deformity. Once a fracture of the radius or both bones of the forearm is identified, the wrist, forearm, and elbow are splinted in the position of function.

Fractures of the radial head generally occur in young to middle-aged adults who fall onto outstretched hands. The victim complains of pain about the elbow, with loss of full extension, and pain at the radial head on the lateral side of the elbow with direct gentle pressure and rotation of the forearm. Fracture of the radial head or neck frequently produces an elbow hemarthrosis, which is identifiable by fullness posterior to the radial head and anterior to the tip of the olecranon (Fig. 24-19). A fluid wave can sometimes be ballotted. If equipment is available and the diagnosis is certain, the hemarthrosis is aspirated and 5 mL of lidocaine is instilled. The elbow is gently moved through a range of motion and then placed in a posterior splint in 90 degrees of flexion with the forearm supinated. On a prolonged expedition when definitive care cannot be reached, the splint is removed after 5 days so that the victim can perform intermittent range-of-motion exercises (both flexion–extension and pronation–supination), and the splint is reapplied for comfort. With more comminuted radial head fractures, attempts at motion produce pain and crepitus, and motion remains restricted. These injuries require operative treatment. Although most individuals lose some extension and pronation–supination, early motion may prevent permanent loss of motion when the radial head fracture is nondisplaced or minimally displaced. The arm is splinted in supination to

Figure 24-19. Fracture of the neck of the radius, sustained in a fall. Pain on palpation at the lateral aspect of the elbow, in the region of the radial neck and head, would be anticipated.

Figure 24-20. Lateral plain radiograph of a 75-year-old hiker who fell onto her outstretched hand and sustained a distal radius fracture.

prevent contracture of the intraosseous ligament and loss of supination.

Fracture of the distal metaphyseal radius is generally associated with the fall of an older osteoporotic individual onto the outstretched hand (Fig. 24-20). In the wilderness, these fractures occur in younger adults with falls from significant heights onto outstretched hands. Ulnar styloid fracture often accompanies intra-articular fracture of the distal radius. Pain, deformity, and crepitus are obvious. When this injury is suspected, a distal neurocirculatory examination is performed, focusing on the sensory function of the median nerve. Median nerve injury or compression at this level of injury would manifest primarily as decreased sensation of the volar palm and fingers (the thumb, index finger, middle finger and the radial half of the ring finger). Weakness in the opponens and abductor pollicis muscles might also be noted. If there is neurocirculatory compromise and definitive care is more than 2 hours away, a gentle reduction is performed. One hand is placed on the forearm to provide countertraction and the other around the wrist of the involved extremity. The wrist is dorsiflexed and longitudinal traction is applied as the wrist is returned to a neutral position. A splint is applied to immobilize the wrist and elbow.

As seen with many extremity fractures, there is a bimodal age group distribution in distal radius fractures; distal radius and ulna fractures occur commonly in children, too. They are seen most frequently in girls aged 11 to 13 years and in boys aged 13 to 15. These fractures are not usually comminuted but can be difficult to reduce (Fig. 24-21). In cases involving an open fracture, a significant distal neurologic deficit, or an abnormal circulatory examination, splinting and evacuation should be prompt. The limb should be kept elevated above the heart during transport.

Ulna

Ulna shaft fracture is most often associated with fracture of the radial shaft at the same level. When isolated, it usually occurs as a result of a direct blow, the so-called nightstick fracture. Fracture of the ulnar shaft can be associated with dislocation of the radial head (Monteggia lesion), so elbow function is carefully assessed. In the wilderness, the most frequent mechanism of injury is bracing a fall or collision with the forearm. Pain, localized swelling, and crepitus are present. A long-arm splint is applied in the position of function. An open fracture is an indication for prompt evacuation.

Fracture of the proximal ulna (olecranon) results from a fall onto the posterior elbow, or from violent asymmetric contraction of the triceps muscle. The victim may be unable to extend the elbow actively against gravity if the triceps is dissociated from the forearm with a complete olecranon fracture. On initial examination, the victim has pain, significant swelling, and a palpable gap in the olecranon. With severe trauma, olecranon fracture may be associated with an elbow dislocation or an intra-articular fracture of the distal humerus, which can only be diagnosed radiographically (Fig. 24-22). A complete distal neurocirculatory examination should be performed. The shoulder and wrist should be examined, and a splint should be applied in the position of function and comfort. An open fracture, absent pulse, severe swelling, or neurologic deficit should prompt immediate evacuation.

Wrist/Carpus

Wrist fractures occur with significant rotational forces or high axial loading forces, as occur in falls onto the hand. The victim first complains of pain and later of wrist swelling. Hand use or forearm rotation produces significant pain. Many carpal bone fractures are associated with wrist dislocation. Reduction of dislocations is described later.

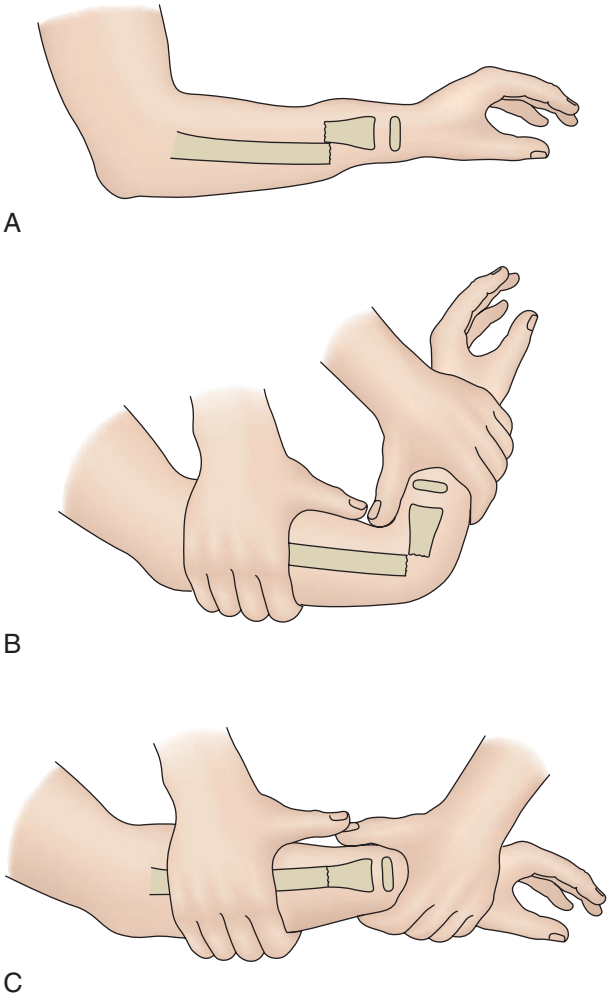


Figure 24-21. Technique for reduction of a complete fracture of the forearm. **A,** Initial fracture position. **B,** Hyperextend fracture to 100 degrees to disengage the fracture ends. **C,** Push with the thumb on the distal fragment to achieve reduction. (**A** to **C** from Green N, Swiontkowski MF: Skeletal Trauma in Children, vol 3, ed 2. Philadelphia, WB Saunders, 1998.)

Figure 24-22. Closed, displaced olecranon fracture dislocation. A sling or posterior splinting is appropriate for this injury type until definitive care can be provided.

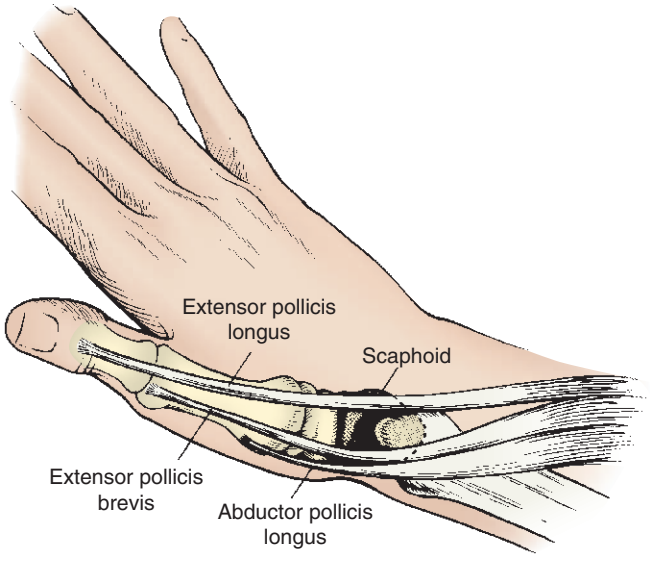


Figure 24-23. The scaphoid (navicular) bone sits in the "anatomic snuffbox" of the radial aspect of the wrist.

Carpal bone fractures cannot be diagnosed without radiographs. Scaphoid (navicular) fracture is the most common fracture and is suspected when the patient's area of maximal tenderness is in the "anatomic snuffbox" (Fig. 24-23). If appropriate splinting materials are available, a thumb spica splint is applied, immobilizing both the radius and the entire thumb. With fracture of the hook of the hamate bone, the victim complains of pain at the base of the hypothenar eminence. This injury occurs when the hand is used to apply significant force to an object with a handle on it, such as an ax or hammer, and great resistance is met. A short-arm splint suffices for this injury, and for other suspected carpal injuries, until definitive treatment is obtained. With open fractures or those accompanied by median nerve dysfunction, the victim should be promptly evacuated.

Metacarpals

Fracture of the metacarpal base or shaft occurs with crush injuries or with axial loads, such as when rocks or other immovable objects are struck. Fractures at the base of the metacarpals are suspected when tenderness, crepitus, and, occasionally, deformity are present. These should be managed with a short-arm splint or an ulnar gutter splint.

Fractures of the metacarpal necks also occur by the same mechanism and usually involve the fifth and fourth metacarpals. These fractures can be associated with significant flexion deformity. Up to 40 degrees of flexion in the fifth and fourth digits can be accepted without compromising hand function, so these fractures rarely require reduction. Rotational deformity of the metacarpal is poorly tolerated, however, and should be anticipated with suspected metacarpal fractures. With the MCP and the IP joints flexed 90 degrees, the fingernails should be parallel to one another and perpendicular to the orientation of the

palm. The terminal portions of the digit should point to the scaphoid tubercle. If this is not the case, a rotational deformity should be strongly suspected.

When malalignment or significant shortening with a suspected shaft fracture is noted, the fracture is reduced with longitudinal traction on the involved digit. A fractured metacarpal shaft or neck is immobilized by applying an aluminum splint (or stick) to the volar surface of the finger and palm and taping the involved digit to the adjacent digit, with the MCP joint at 90 degrees. This position provides maximal length of the collateral ligaments. Immobilizing the joint in this position prevents contractures that can lead to subsequent loss of motion.

Fracture of the base of the thumb metacarpal often occurs with an axial force directed against a partially flexed thumb metacarpal. If the fracture extends into the joint, it often requires operative fixation. If this fracture is suspected, the thumb and wrist are immobilized in a thumb spica splint. An open metacarpal fracture needs cleansing, debridement, and presumptive antibiotic therapy for 48 hours or until definitive care is obtained.

Phalanges

Fractures of the digital phalanges occur with crush injuries or when the digits are caught in ropes or in equipment being used to haul objects. Angular or rotational deformity and crepitus make these fractures obvious. Without radiographs, an intra-articular fracture with subluxation or dislocation is difficult to differentiate from an IP joint dislocation. Angular deformities in these fractures can be reduced using a pencil or thin stick placed in the web space as a fulcrum to assist in reduction. A fracture of the shaft of a phalanx is reduced by applying traction and correcting the deformity. The fractures are immobilized by taping the injured digit to the neighboring uninjured digit or to a volar splint. Nail-bed fractures or crushes are cleansed with soap, a sterile dressing is applied, and a protective volar splint is applied to prevent further injury.

UPPER EXTREMITY DISLOCATIONS AND SPRAINS

Sternoclavicular Joint

Traumatic dislocation of the sternoclavicular joint generally requires tremendous force, either direct or indirect, applied to the shoulder. Consequently, it is rare. Anterior dislocation is most common, with the medial head of the clavicle going anterior to the manubrium of the sternum. The victim complains of pain around the sternum and frequently has difficulty taking a deep breath. When the dislocation is posterior, significant pressure may be placed on the esophagus and superior vena cava. The victim may complain of difficulty swallowing and have engorgement of the veins of the face and upper extremities, representing superior vena cava obstruction syndrome. A step-off between the sternum and the medial head of the clavicle (compared with the uninjured side) confirms this diagnosis.

Unreduced anterior dislocation does not produce neurocirculatory compromise and is treated with a sling. Reduction of a posterior sternoclavicular dislocation should be attempted as soon as possible if any neurocirculatory compromise is present. The victim is placed supine with a large roll of clothing or other firm object between the scapulae. Traction is applied to the arm against countertraction in an abducted and slightly extended position. The medial end of the clavicle may need to be manually manipulated to dislodge the clavicle from behind the manubrium (Fig. 24-24). If this fails, sharp, firm pressure is applied posteriorly to both shoulders. This maneuver is repeated several times, with a larger object placed between the scapulae if reduction attempts are initially unsuccessful. Alternatively, with the victim seated and the caregiver's knee against the back between the shoulders, both shoulders are pulled back. If the victim remains in extremis, the midshaft clavicle is grasped with a towel clip or pliers and forcefully pulled out of the thoracic cavity. Once reduced, the injury is usually stable. The posterior sternoclavicular dislocation requires evacuation.

Acromioclavicular Joint

The acromioclavicular joint is usually injured by a blow on top of the shoulder (Fig. 24-25). Because using the hand increases pain, the arm on the affected side is placed in a sling. As long as the individual can tolerate the discomfort associated with such an injury, evacuation is not necessary.

Glenohumeral Joint

The head of the humerus at the shoulder joint is generally dislocated anteriorly and inferiorly. The usual mechanism of injury is a blow to the arm in the abducted and externally rotated position. This frequently occurs in skiing as the individual crosses the ski tips or goes forward on a mogul and lands face down with the arms in this position.

Recurrent dislocations and dislocations in younger patients may be easier to reduce than first-time dislocations in older patients. A thorough motor, sensory, and circulatory examination of the involved extremity is performed. Axillary and musculocutaneous nerves are assessed carefully because they are the nerves most commonly injured with an anterior dislocation. Serial examinations of distal pulses, capillary refill, and forearm compartments are performed.

The preferred method of reduction is linear traction along the axial line of the extremity while stabilizing the torso with a blanket or rope (Fig. 24-26). Narcotic or benzodiazepine premedication can be extremely helpful, but this should be avoided in a multiply injured victim if there is concern about altering mentation or adversely affecting blood pressure. A sheet, belt, webbed strapping, or avalanche cord can be tied around the caregiver's waist and the victim's bent forearm, so that the caregiver (standing or kneeling) can lean back to apply traction, leaving hands free to guide the head of the humerus back into position (Fig. 24-27). Padding is placed in the armpit and bend of the elbow to prevent pressure injury to sensitive nerves beneath the skin.

Many other relocation techniques have been described. In the Milch technique, the patient is prone or sitting upright. The caregiver places his or her right hand in the axilla for a dislocated right shoulder and holds the victim's hand with the left. The victim's arm is gently abducted and pressure is applied to the humeral head. When the arm is fully abducted, it is rotated externally and gentle traction is applied to reduce the humeral head. This slow process can be highly successful in the acute setting because it usually does not require analgesics or muscle relaxants (Fig. 24-28).[13] The success of this maneuver decreases as the time after dislocation increases.

Scapular manipulation is also minimally traumatic and highly successful.[2] The victim is placed prone and 5 to 15 pounds of traction are applied on the arm. Once relaxation is obtained,

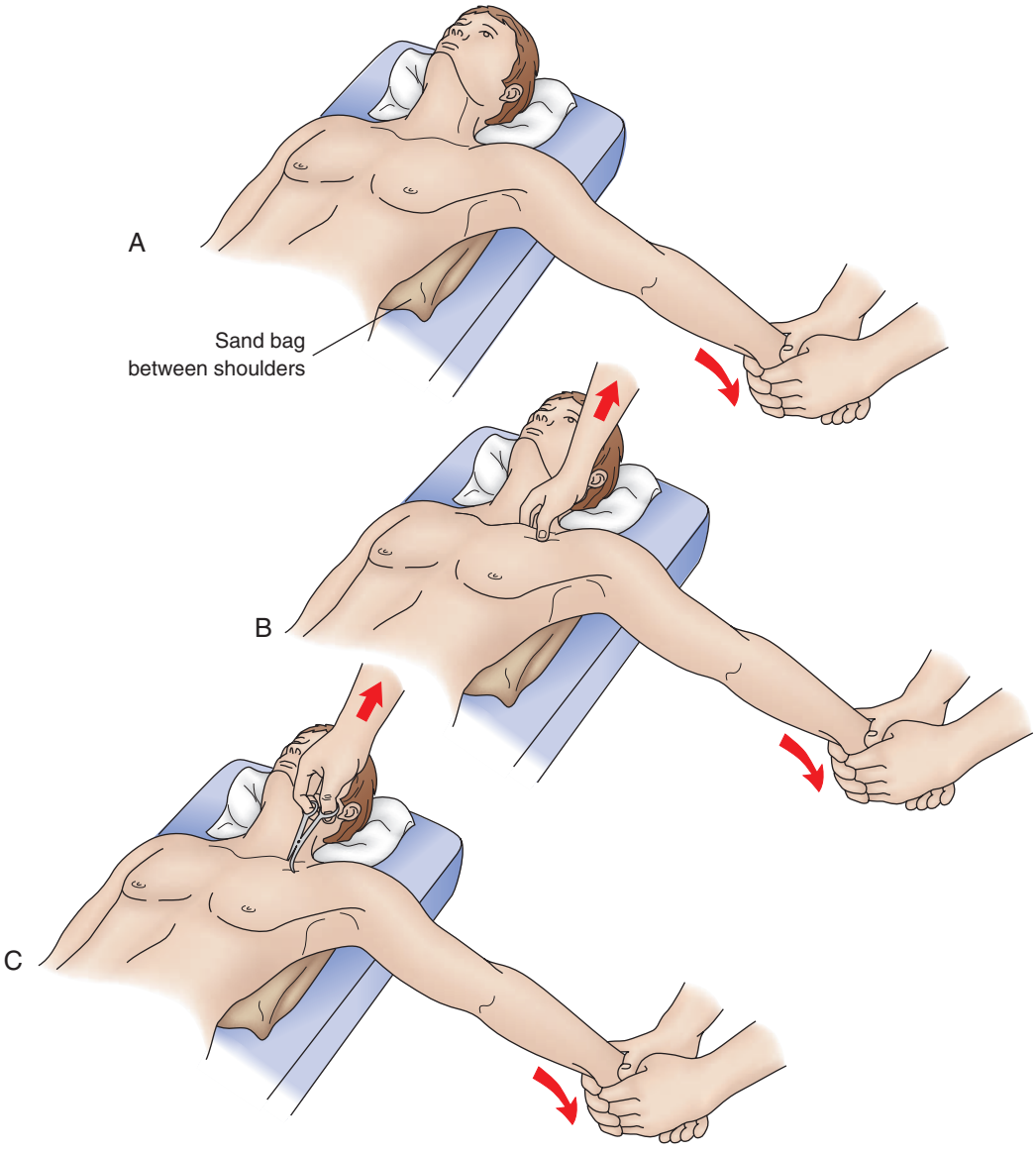


Figure 24-24. Technique for closed reduction of the sternoclavicular joint. **A,** The patient is positioned supine with a sandbag placed between the shoulders. Traction is then applied to the arm against countertraction in an abducted and slightly extended position. For anterior dislocation, direct pressure over the medial end of the clavicle may reduce the joint. **B,** For posterior dislocation, in addition to the traction it may be necessary to manipulate the medial end of the clavicle with the fingers to dislodge the clavicle from behind the manubrium. **C,** For a stubborn posterior dislocation, it may be necessary to sterilely prepare the medial end of the clavicle and use a towel clip to grasp around the medial clavicle and lift it back into position. (From Rockwood CA Jr, Green DP, Bucholz RW [eds]: Rockwood and Green's Fractures in Adults, 3rd ed. Philadelphia, JB Lippincott, 1991.)

the inferior angle of the scapula is raised and rotated toward the spine; the superior aspect is rotated away from the spine (Fig. 24-29). This can also be done with the victim in the standing position (Fig. 24-30). If the victim is standing, it may help to pull the arm forward as well as down. This technique generally requires excellent relaxation, but it can be highly successful.

An alternative method is to have the victim lie prone so that the injured arm dangles free. A thick pad is placed under the injured shoulder. A 10- to 20-lb (4.5- to 9-kg) weight is attached to the wrist or forearm (the victim should not attempt to hold the weight), and it is allowed to exert steady traction on the arm, using gravity to relocate the humeral head (Fig. 24-31). A standing victim can bend forward at the waist as the caregiver pulls steadily downward on the arm to simulate the gravity effect. A gentle side-to-side motion at the wrist can help with the reduction (Fig. 24-32). The Hippocratic maneuver of placing a foot in the axilla of the injured limb to achieve countertraction should be avoided because of increased pressure on the structures within the axillary sheath (Fig. 24-33).

Posterior dislocation of the glenohumeral joint makes up less than 5% of shoulder dislocations. It may occur with adduction and axial loading of the shoulder, a direct blow to the anterior aspect of the shoulder, or as a result of marked internal rotation accompanying a grand mal seizure. Frequently, the dislocation is associated with either a humeral head impaction injury or a glenoid fracture. The victim complains of significant pain and loss of shoulder motion. Usually, external rotation is completely lost. On palpation of the shoulder, posterior fullness that is not found on the uninjured side can usually be detected. This dislocation can be more difficult to reduce than an anterior dislocation, so excellent analgesia is generally required. The

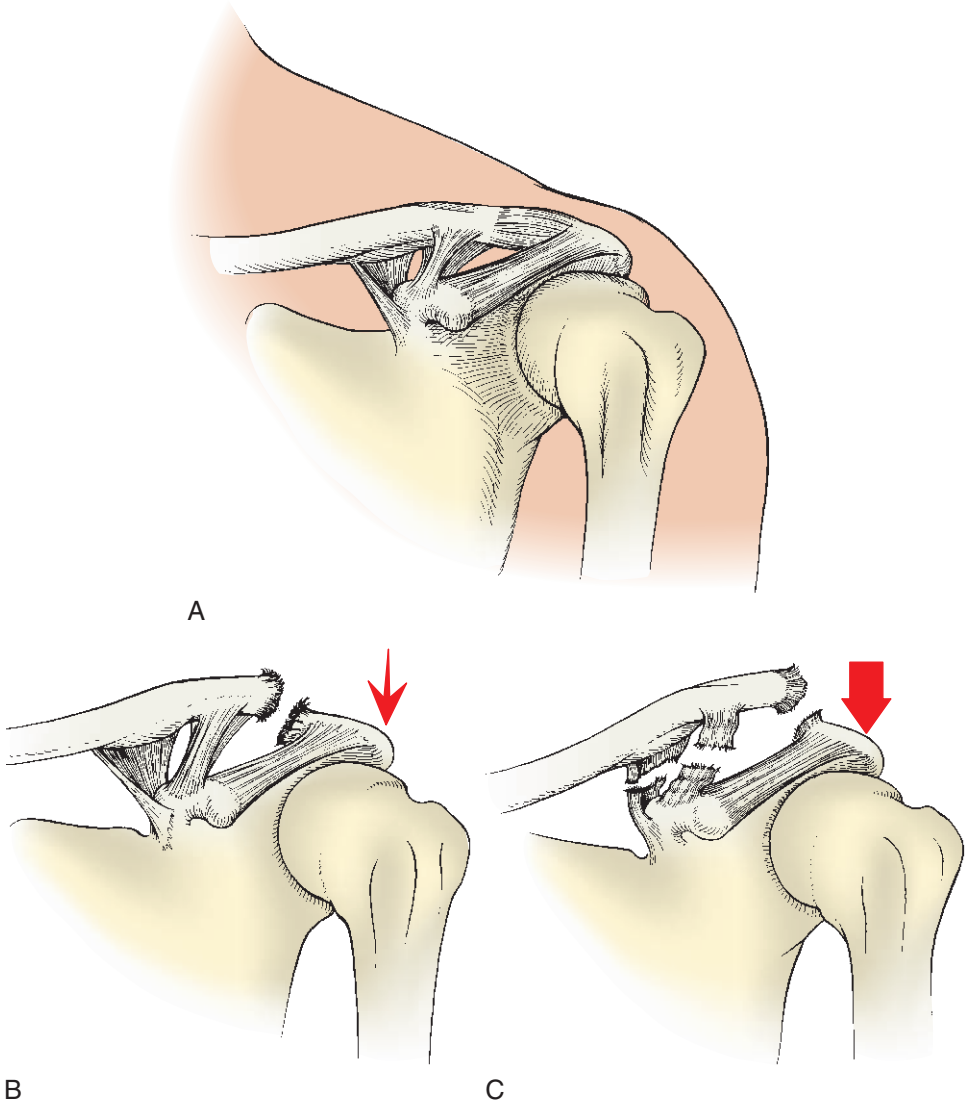


Figure 24-25. Acromioclavicular joint surgery. **A,** Normal anatomy. **B,** Second-degree injury. **C,** Third-degree injury.

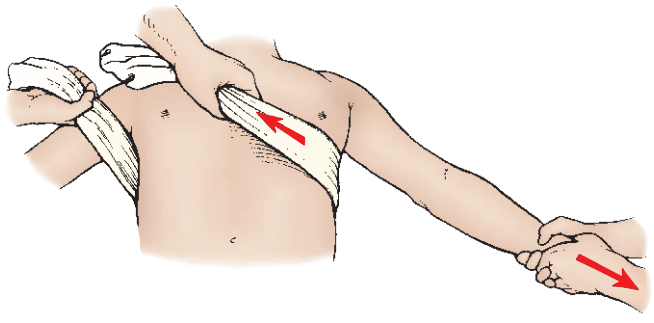


Figure 24-26. Traction and countertraction for dislocated shoulder reduction.

arm is flexed forward, rotated internally, and adducted to disengage the head from the posterior glenoid rim. Occasionally, lateral traction on the humeral shaft is also required. With longitudinal traction and anterior pressure on the humeral head from behind, reduction is achieved.

If the reduction maneuver is successful, the arm is placed in a sling until definitive care is reached. If possible, a posterior dislocation is held in neutral or slight external rotation. Because of the significant incidence of fractures with these injuries, radiologic examination is required to make the diagnosis, and evacuation is mandated. If the reduction maneuver is not successful, the injured extremity is placed in a sling and evacuation is arranged as soon as possible.

Elbow

Dislocation of the elbow occurs with hyperextension or axial loading from a fall onto an outstretched hand. The direction of

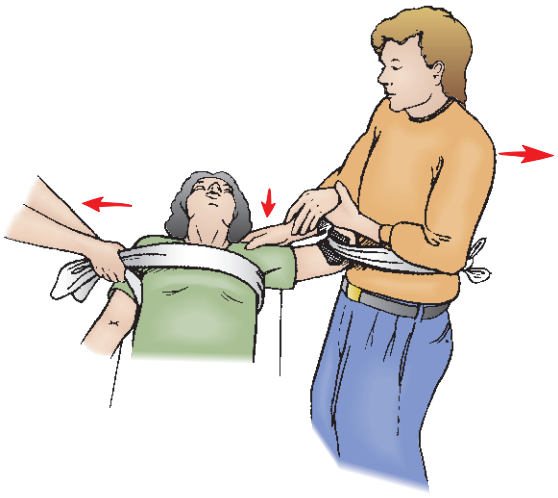


Figure 24-27. Repositioning a dislocated shoulder. Attached to the victim's forearm with a strap, rope, or sheet, the rescuer uses his body weight to apply traction, leaving his hands free to manipulate the victim's arm. A second rescuer applies countertraction, or the victim can be held motionless by fixing the chest sheet to a tree or ground stake. (From Auerbach PS: Medicine for the Outdoors: The Essential Guide to Emergency Medical Procedures and First Aid, 4th ed. Guilford, CT, Lyons Press, 2003.)

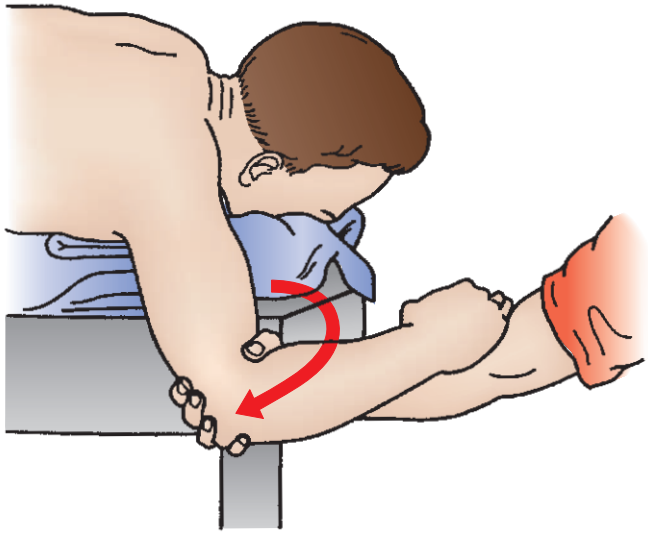


Figure 24-28. Milch technique of closed reduction of anterior glenohumeral dislocation with the patient prone. The arm can be manipulated in the same manner with the patient supine. (Redrawn from Lacey T II, Crawford HB: J Bone Joint Surg 34A:108, 1952. In Browner BD, Jupiter JB, Levine AM, Trafton PG: Skeletal Trauma, vol 2, 2nd ed. Philadelphia, WB Saunders, 1998.)

dislocation is generally posterior and lateral. The diagnosis is clear, with posterior deformity at the elbow and foreshortening of the forearm. After carefully assessing distal sensory, motor, and circulatory status, reduction is performed. With countertraction on the upper arm, linear traction is applied with the elbow slightly flexed and the forearm in the original degree of pronation and supination. Downward pressure on the proximal forearm to disengage the coronoid from the olecranon fossa may be helpful. Hyperextension should be avoided. Adequate

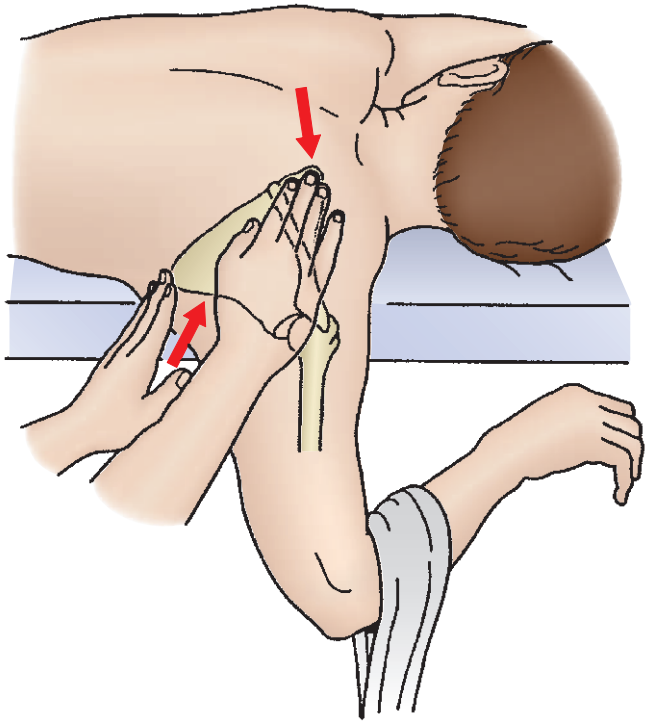


Figure 24-29. Scapular manipulation technique for closed reduction of anterior glenohumeral dislocation. (Redrawn from Anderson D, Zvirbulis R, Ciullo J: Clin Orthop 164:181, 1982. In Browner BD, Jupiter JB, Levine AM, Trafton PG: Skeletal Trauma, vol 2, 2nd ed. Philadelphia, WB Saunders, 1998.)

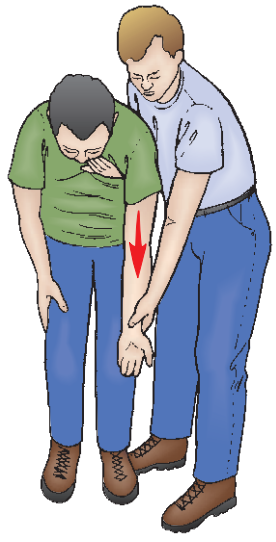


Figure 24-30. Pulling on the hanging arm to relocate a dislocated humerus. (From Auerbach PS: Medicine for the Outdoors: The Essential Guide to Emergency Medical Procedures and First Aid, 4th ed. Guilford, CT, Lyons Press, 2003.)

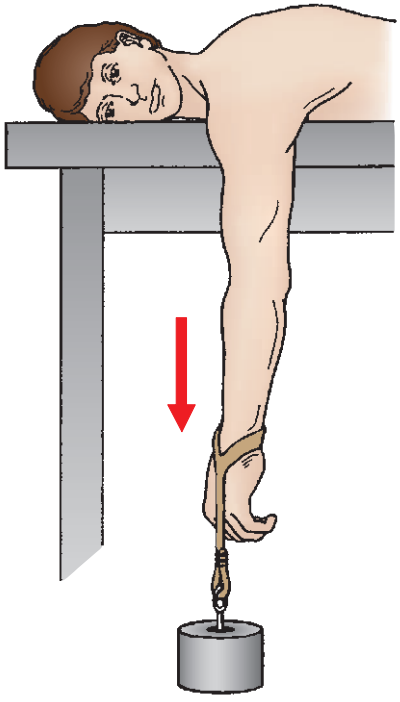


Figure 24-31. Stimson technique. (Redrawn from Rockwood CA, Green CP [eds]: Fractures in Adults, vol 1. Philadelphia, JB Lippincott, 1984. In Browner BD, Jupiter JB, Levine AM, Trafton PG: Skeletal Trauma, vol 2, 2nd ed. Philadelphia, WB Saunders, 1998.)

analgesia can be extremely helpful. An alternative method (Parvin's method) is to place the patient prone over a log or makeshift platform and apply gentle downward traction on the wrist for a few minutes. As the olecranon begins to slip distally, the arm is lifted up gently. No assistant is needed, and if the maneuver is done gently, no anesthesia is required (Fig. 24-34). A modification of this maneuver (Meyn and Quigley's method) is to hang only the forearm over the platform while applying gentle downward traction via the wrist and guiding the reduction of the olecranon with the opposite hand (Fig. 24-35). Reduction provides nearly complete relief of pain and restoration of normal surface anatomy. A posterior splint is applied with the elbow in 90 degrees of flexion and the forearm in neutral position. A sling is provided for comfort. If reduction is not successful after three attempts, or if a nerve injury is suspected, a splint is applied to the arm as it lies and evacuation is initiated.

Wrist

Wrist dislocations are frequently associated with carpal fractures and are generally produced by falls onto an outstretched hand. A wrist dislocation may be difficult to differentiate clinically from a fracture of the distal radius. However, in either case, a reduction maneuver is performed after careful assessment of distal neurocirculatory function, focusing on median nerve function. The reduction maneuver is similar to that for a distal radius fracture. One hand is used to stabilize the forearm and the other to grasp the hand. First, the wrist is dorsiflexed if the dislocation is dorsal (most common) or volarflexed if the

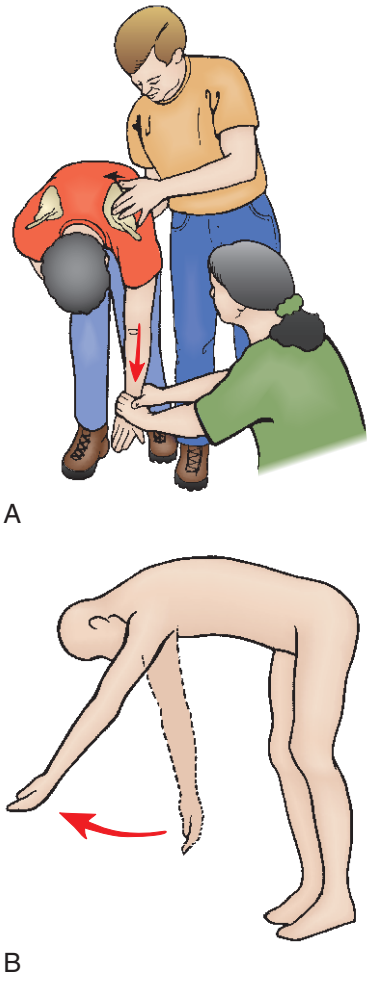


Figure 24-32. **A,** Pushing the lower edge of the scapula toward the spine while an assistant pulls downward on the hanging arm to assist in the relocation of a dislocated humerus. **B,** The downward pull on the arm may be slightly forward to help put the humerus back in the shoulder socket. (From Auerbach PS: Medicine for the Outdoors: The Essential Guide to Emergency Medical Procedures and First Aid, 4th ed. Guilford, CT, Lyons Press, 2003.)

dislocation is volar, and then longitudinal traction is applied. In general, significant dorsiflexion is required to obtain reduction, and premedication can be extremely helpful. If reduction is unsuccessful after three attempts, or if there is median nerve dysfunction, evacuation should be initiated. A short-arm splint is applied if reduction is successful, and the arm is elevated above the level of the heart until definitive care is procured. Pain and tenderness about the wrist with no significant deformity indicates a possible intercarpal ligamentous disruption or a carpal fracture, and a short-arm splint is applied.

Metacarpophalangeal Joint

MCP joint dislocation is rare and may be produced by a crush injury or when the hand is caught in a rope. This dislocation may be dorsal or volar, with dorsal dislocation more common. Clinically, the joint is hyperextended and the phalanx shortened. Most dorsal dislocations are easily reduced. First, the proximal phalanx is hyperextended 90 degrees on the meta-

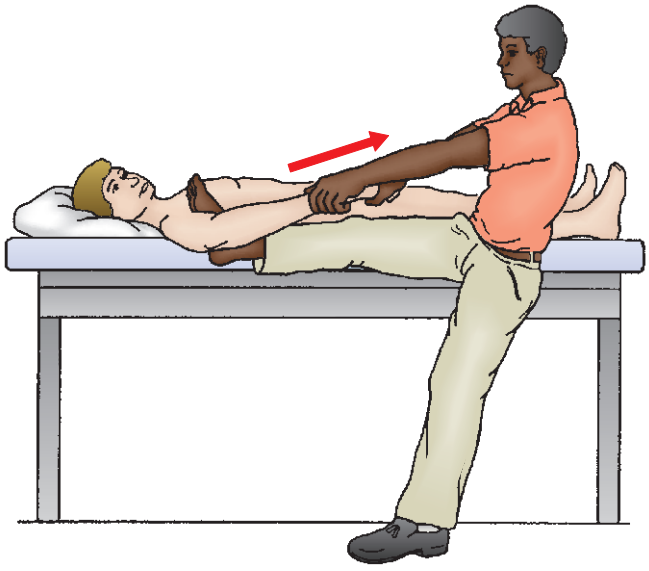


Figure 24-33. Hippocratic technique of closed reduction of anterior glenohumeral dislocation. The foot is placed against the proximal humerus, and longitudinal traction is applied to the upper extremity. (Redrawn from Rockwood CA, Green CP [eds]: Fractures in Adults, vol 1. Philadelphia, JB Lippincott, 1984.)

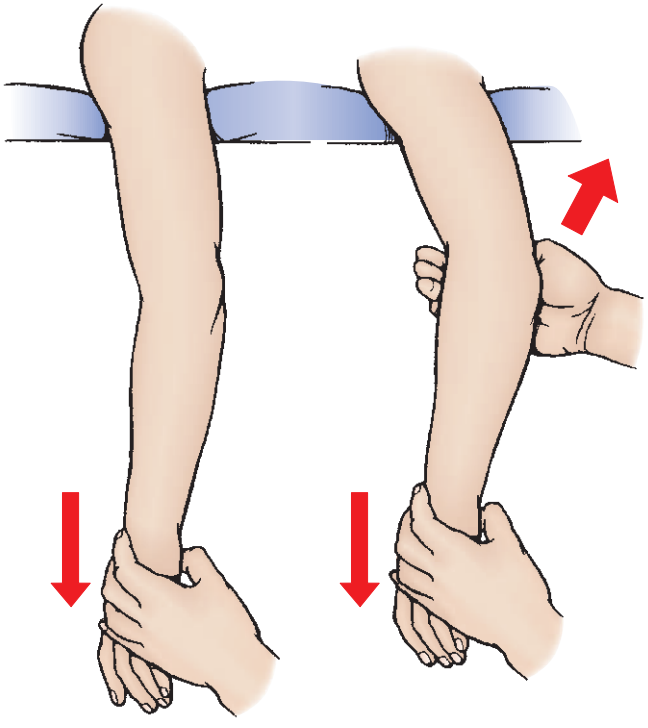


Figure 24-34. Parvin's method of closed reduction of an elbow dislocation. The patient lies prone on a stretcher, and the physician applies gentle downward traction on the wrist for a few minutes. As the olecranon begins to slip distally, the physician lifts up gently on the arm. No assistant is required, and if the maneuver is done gently, no anesthesia is required. (Redrawn from Parvin RW: Arch Surg 75:972, 1957. In Rockwood CA Jr, Green DP, Bucholz RW [eds]: Rockwood and Green's Fractures in Adults, 3rd ed. Philadelphia, JB Lippincott, 1991.)

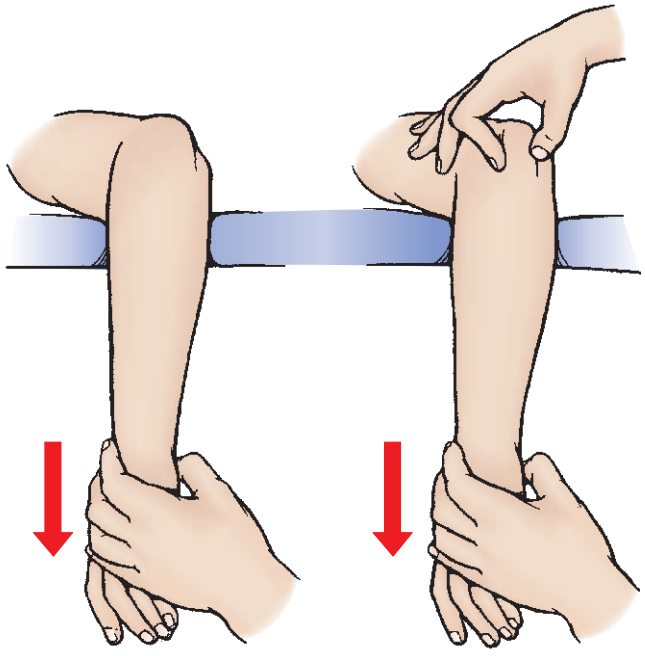


Figure 24-35. In Meyn and Quigley's method of reduction, only the forearm hangs from the side of the stretcher. As gentle downward traction is applied on the wrist, the physician guides reduction of the olecranon with the opposite hand. (Redrawn from Meyn MA, Quigley TB: Clin Orthop 103:106, 1974. In Rockwood CA Jr, Green DP, Bucholz RW [eds]: Rockwood and Green's Fractures in Adults, 3rd ed. Philadelphia, JB Lippincott, 1991.)

carpal, then the base of the proximal phalanx is pushed into flexion, maintaining contact at all times with the metacarpal head to prevent entrapment of the volar plate in the joint (Fig. 24-36). Straight longitudinal traction is avoided because it may turn a simple dislocation into a complex dislocation (see later). The wrist and IP joints are flexed to relax the flexor tendons. The joint usually reduces easily with a palpable and audible clunk. A dorsal–volar splint is applied with the joint held at 90 degrees of flexion.

Irreducible or complex dislocations occur when the volar plate is interposed in the joint. Clinically, the joint is only slightly hyperextended and the volar skin is puckered over the joint. These dislocations are most common in the index finger, thumb, and small finger. A single attempt at reduction using the technique just described is indicated, but these dislocations usually require open reduction. If reduction of an MCP joint dislocation is unsuccessful, the joint should be splinted in the position of comfort and definitive treatment obtained as soon as possible.

The thumb MCP joint is most commonly injured. Dislocations are reduced as already described. Injury to the ulnar collateral ligament of this joint (skier's or gamekeeper's thumb) results from a valgus stress, as may occur when an individual falls holding an object in the first web space. The victim complains of tenderness over the ulnar aspect of the MCP joint. There may be instability to radial stress with the joint held in 30 degrees of flexion, an indication for surgical repair. Often, the adductor aponeurosis becomes interposed between the ligament and its bony attachment, resulting in a Stener lesion

(Fig. 24-37). In the field, a thumb spica splint is applied and definitive care sought within 10 days. If splinting material is not available, the thumb is taped until definitive care can be obtained (Fig. 24-38).

Proximal Interphalangeal Joint

Proximal interphalangeal (PIP) joint dislocations may be dorsal, volar, or rotatory, with dorsal dislocation by far the most common. Dorsal dislocation occurs with hyperextension and volar plate rupture. It can be associated with fracture of the volar lip of the middle phalanx, creating instability after reduction. Reduction is performed as described for dorsal MCP dislocation. Straight longitudinal traction is avoided to prevent entrapment of the volar plate into the joint. After reduction, the finger is taped to an adjacent finger to avoid hyperextension and allow early motion (Fig. 24-39). As with MCP dislocation, a complex dislocation can occur, but it is rare. This is difficult to reduce closed and often requires open reduction.

Volar dislocations are rare. For this injury to occur, the central slip must be disrupted, and the potential for a boutonniere deformity is present. The digit is reduced by flexion of the PIP joint, pushing the base of the middle phalanx dorsally. The PIP joint is treated like a rupture of the central slip, with the PIP joint splinted in extension. The distal interphalangeal (DIP) and MCP joints are left free to allow motion.

Rotatory subluxation of the PIP joint occurring after a twisting injury is also rare (Fig. 24-40). The condyle of the head of the proximal phalanx is buttonholed between the lateral band and the central slip, both of which remain intact. This injury can be difficult to reduce. With both the MCP and PIP joints flexed, gentle traction is applied to the finger. This relaxes the volarly displaced lateral band and allows the lateral band to be disengaged and slip dorsally when a gentle rotary and traction force is applied. Further relaxation of the lateral band can be achieved by dorsiflexion of the wrist. After reduction, the finger is buddy taped and early motion is begun. With any dislocation of the PIP joint, definitive care should be sought promptly to ensure adequate reduction of the injury.

"Jammed" fingers may be just as debilitating and painful as dislocated digits. With these injuries, stress the involved joint both radially and ulnarly to ensure collateral ligament integrity. If the joint is stable, the finger can be buddy-taped to an adjacent digit and immediate care is not indicated. If the finger is unstable, definitive care should be sought.

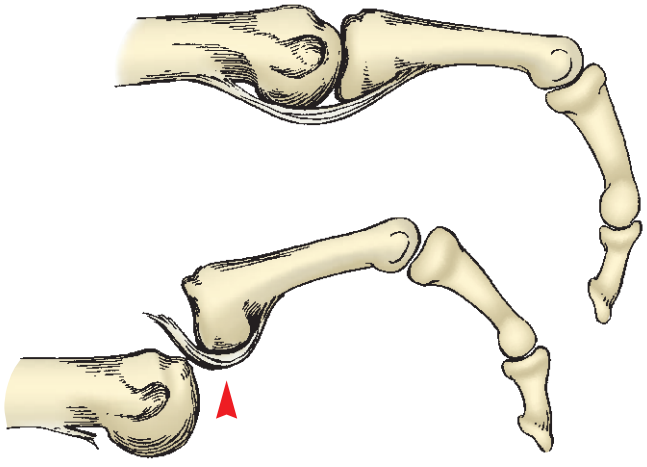


Figure 24-36. The single most important element preventing reduction in a complex metacarpophalangeal (MCP) dislocation is interposition of the volar plate within the joint space. It must be extricated surgically. (From Rockwood CA Jr, Green DP, Bucholz RW [eds]: Rockwood and Green's Fractures in Adults, 3rd ed. Philadelphia, JB Lippincott, 1991.)

Distal Interphalangeal Joint

The DIP joint is less frequently injured than the PIP joint. Pure dislocations are rare and are usually dorsal and associated with an open wound. The reduction maneuver is similar to that used for dorsal PIP joint dislocation. The injury is usually stable after reduction.

More commonly, a mallet-finger injury occurs when the extensor tendon is taut, as when striking an object with the finger extended. The basic injury is incompetence of the extensor tendon at its insertion into the distal phalanx. The three

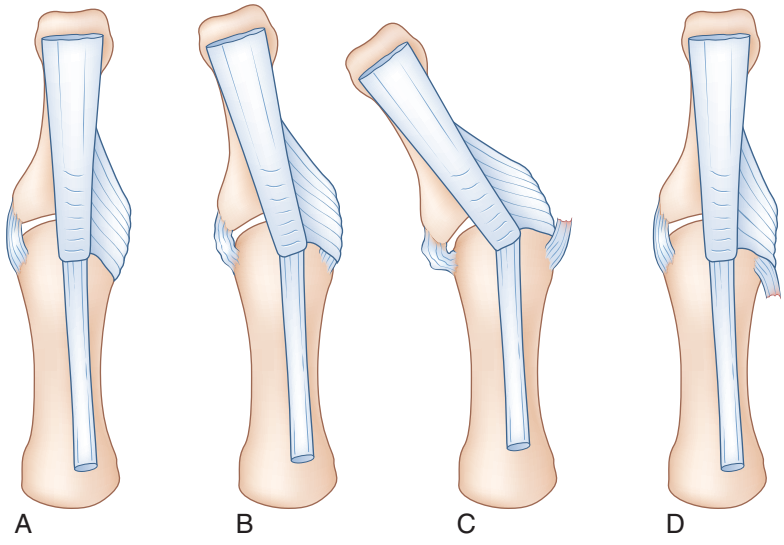


Figure 24-37. Diagram of the displacement of the ulnar collateral ligament of the thumb metacarpophalangeal joint. **A,** Normal relationship, with the ulnar ligament covered by the adductor aponeurosis. **B,** With slight radial angulation, the proximal margin of the aponeurosis slides distally and leaves a portion of the ligament uncovered. **C,** With major radial angulation, the ulnar ligament ruptures at its distal insertion. In this degree of angulation, the aponeurosis has displaced distal to the rupture and permitted the ligament to escape from beneath it. **D,** As the joint is realigned, the proximal edge of the adductor aponeurosis sweeps the free end of the ligament proximally and farther away from its insertion. This is the Stener lesion. Unless surgically restored, the ulnar ligament will not heal properly and will be unstable to lateral stress. (Redrawn from Stener B: Acta Chir Scand 125:583–586, 1963.)

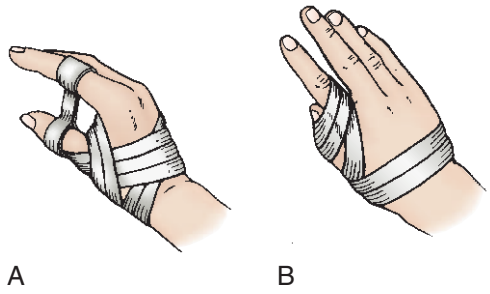


Figure 24-38. Taping the thumb for immobilization. **A,** The buddy-taping method. **B,** A thumb-lock. If possible, padding should be placed between the thumb and forefinger. (From Auerbach PS: Medicine for the Outdoors: The Essential Guide to Emergency Medical Procedures and First Aid, 4th ed. Guilford, CT, Lyons Press, 2003.)

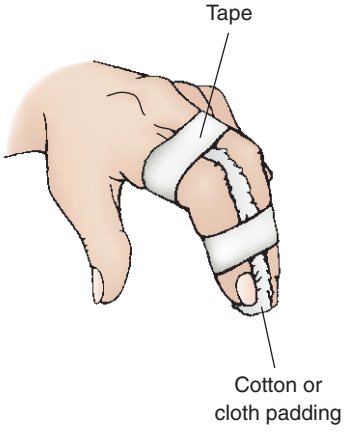


Figure 24-39. Buddy-taping method to immobilize a finger. (From Auerbach PS: Medicine for the Outdoors: The Essential Guide to Emergency Medical Procedures and First Aid, 4th ed. Guilford, CT, Lyons Press, 2003.)

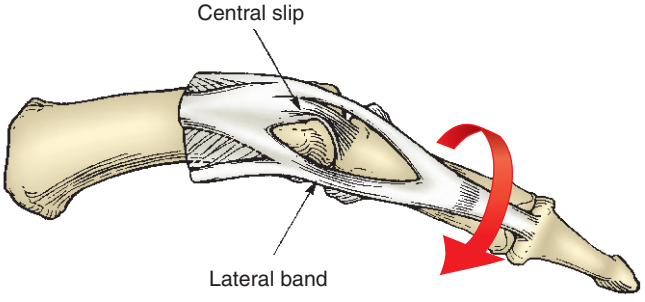


Figure 24-40. Rotary subluxation of the proximal interphalangeal (PIP) joint. The condyle of the head of the proximal phalanx is button-holed between the lateral band and the central slip, both of which remain intact. (From Rockwood CA Jr, Green DP, Bucholz RW [eds]: Rockwood and Green's Fractures in Adults, 3rd ed. Philadelphia, JB Lippincott, 1991.)

types of mallet injuries are shown in Figure 24-41. Individuals with this injury lack full extension of the DIP joint when the MCP and PIP joints are kept in extension. Holding these joints in extension isolates the extensor tendon by neutralizing the intrinsic muscles. If an extension lag is noted, the joint is

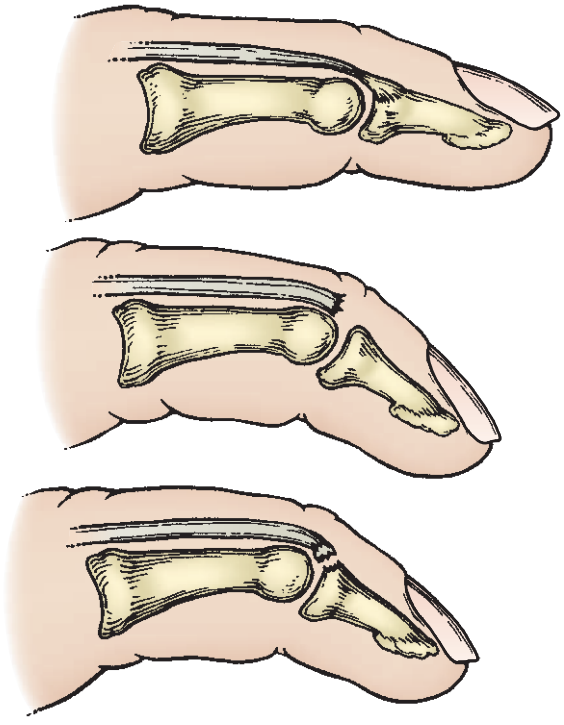


Figure 24-41. The three types of injury that cause a mallet finger of tendon origin. *Top,* The extensor tendon fibers over the distal joint are stretched without complete division of the tendon. Although there is some drop of the distal phalanx, the patient retains weak active extension. *Center,* The extensor tendon is ruptured from its insertion on the distal phalanx. There is usually a 40- to 45-degree flexion deformity, and the patient has loss of active extension at the distal joint. *Bottom,* A small fragment of the distal phalanx is avulsed with the extensor tendon. This injury has the same clinical findings as that shown in the center drawing. (From Rockwood CA Jr, Green DP, Bucholz RW [eds]: Rockwood and Green's Fractures in Adults, 3rd ed. Philadelphia, JB Lippincott, 1991.)

splinted in slight extension for 6 to 8 weeks, leaving the PIP and DIP joints free (Fig. 24-42). A radiograph is obtained within the first 10 days to ensure that the joint is reduced.

Occasionally, the flexor digitorum profundus (FDP) tendon is avulsed from its insertion on the distal phalanx. This occurs with forced hyperextension of the DIP joint while the FDP tendon is maximally contracted. The ring finger is most commonly injured. The diagnosis is made by demonstrating inability to flex the DIP joint with the PIP joint held in extension. Pain and local tenderness are more common over the PIP joint, where the retracted end of the tendon usually lies. The digit is splinted in flexion, and care should be sought within 7 days from a surgeon specializing in the upper extremities.

LOWER EXTREMITY FRACTURES

Femur and Patella

In general, healthy, active individuals sustain fractures of the proximal femur only in falls from significant heights or from high-velocity injuries sustained, for example, during water or snow skiing. These fractures occur in the femoral neck, intertrochanteric, or subtrochanteric region (Fig. 24-43). When

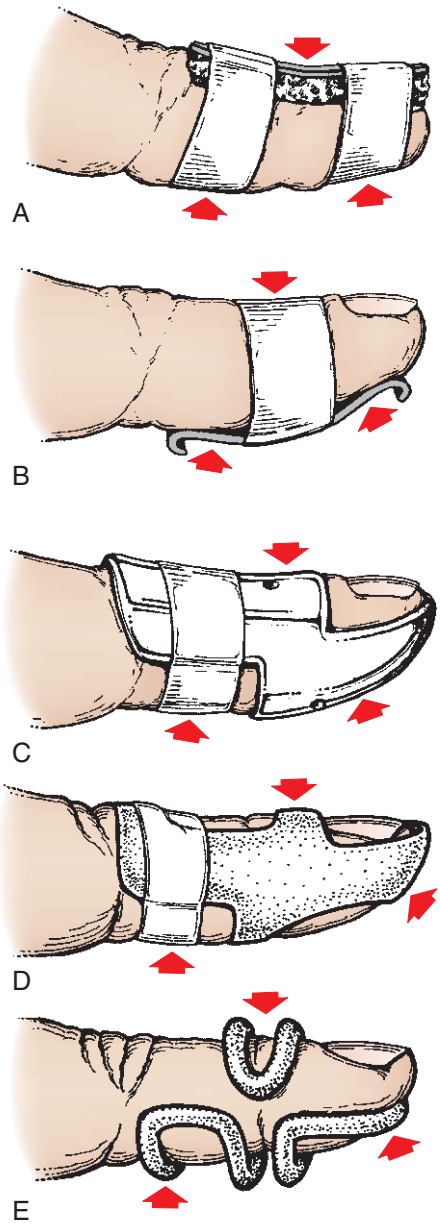


Figure 24-42. Mallet finger can be treated by immobilizing the distal interphalangeal (DIP) joint with a dorsal padded aluminum splint **(A),** a volar unpadded aluminum splint **(B),** a Stack splint **(C),** a modified Stack splint **(D),** or an Abouna splint **(E).** Each of these splints uses a three-point fixation principle. (From Rockwood CA Jr, Green DP, Bucholz RW [eds]: Rockwood and Green's Fractures in Adults, 3rd ed. Philadelphia, JB Lippincott, 1991.)

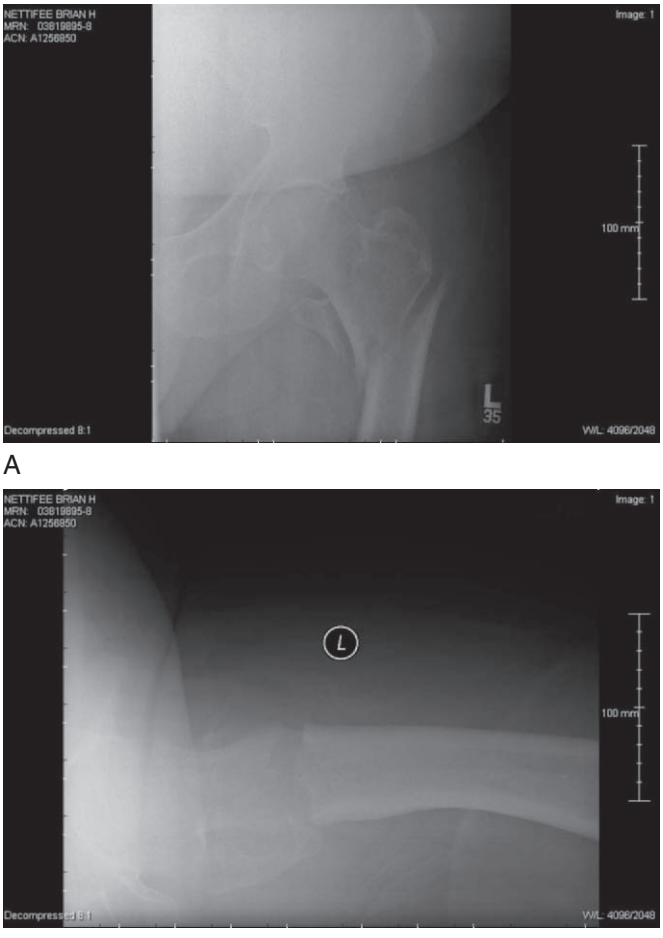
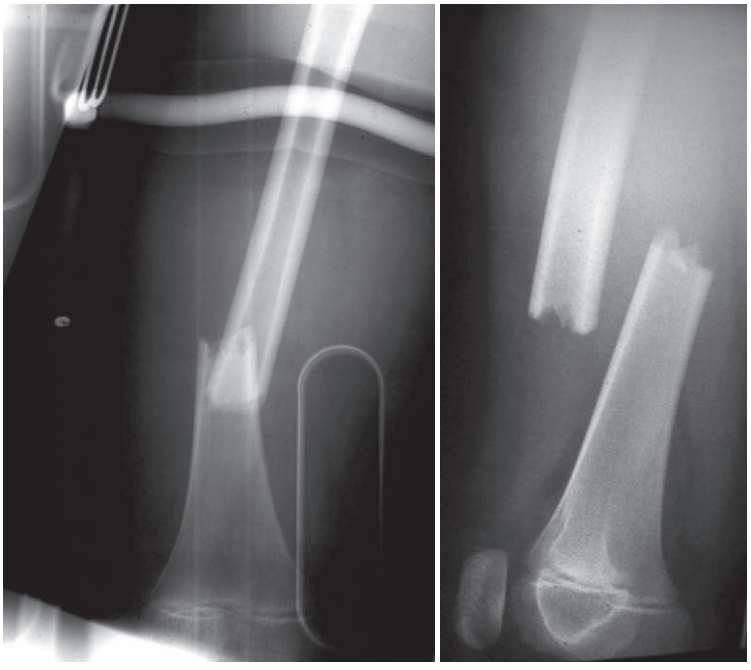


Figure 24-43. An intertrochanteric fracture caused by a fall from a height. Always check for other injuries that may result from a fall, such as lumbar spine fractures or calcaneus fractures.

the head and spinal cord are uninjured, the victim complains of pain within the proximal thigh. In all but the thinnest individuals, there is little local reaction in terms of swelling or deformity around the hip region to aid in diagnosis. Any movement of the affected limb produces significant pain. In many cases, the affected limb is noticeably shortened and externally rotated. After a careful sensory, motor, and circulatory examination, the limb is realigned and, if available, a Kendrick, Thomas, or Reel splint is applied. An improvised traction splint can be fabricated. If none is available, the victim is transported on a backboard, with the limbs strapped together or tied to a board with a tree limb placed between them.

Fracture of the femoral neck is associated with a significant risk of post-traumatic femoral head necrosis. Without a radiograph, this fracture is impossible to distinguish from a subtrochanteric or an intertrochanteric hip fracture. Because there is evidence that emergency treatment of a fracture of the femoral neck decreases the risk of post-traumatic avascular necrosis,[14,17] rapid evacuation should be arranged for any victim in whom this injury is suspected.

Fracture of the femoral shaft occurs by similar mechanisms (Fig. 24-44). Crepitus and maximal deformity are noted in the midportion of the thigh. After neurocirculatory examination, the limb is placed in traction or protected as noted previously. Any gross deformity of the shaft is corrected with gentle traction, and the neurocirculatory examination is repeated. This fracture may be an open injury, so the victim's pants should be



A B

Figure 24-44. A 12-year-old skier sustained this closed, midshaft femur fracture when he struck a tree. The anteroposterior view shows that the patient is on a backboard and in traction.

split to complete the examination. Discovery of an open wound should prompt rapid evacuation.

Fracture of the distal end of the femur is frequently intra-articular and occurs with high-velocity loading when the knee is flexed. With axial loading on the femur, the patella becomes the driving wedge and the femoral condyles suffer direct impact, producing either a patella fracture or a fracture of the femoral condyles or distal femoral metaphysis. Occasionally, in high-energy trauma, a distal femur fracture and an ipsilateral proximal tibia fracture can occur concurrently (Fig. 24-45). With a patella fracture, the injury may be obvious on deep palpation. This is often an open injury because there is very little soft tissue overlying this sesamoid bone. The definitive diagnosis is made radiographically. After initial neurocirculatory examination, the limb is realigned to avoid compression of the popliteal artery and vein. A posterior splint is applied to the realigned limb for transportation. As with all fractures, open wounds in the region of the fracture or an abnormal nerve or vascular examination should prompt immediate evacuation.

Tibia and Fibula

The tibial plateau is the broad intra-articular surface of the upper tibia that articulates with the distal femur. This area can be fractured in a fall or leap from a height (Fig. 24-46). A valgus moment of force produces a fracture of the lateral tibial plateau, whereas a varus moment of force produces a medial plateau fracture. Pain, swelling, and deformity are obvious on initial examination. With a tibial plateau fracture, significant hemarthrosis develops quickly. Because of anatomic tethering of the popliteal artery by fascia of the soleus complex, arterial injury may result from this fracture, especially when it is associated with a knee dislocation. Distal pulses and capillary refill should be assessed at 1-hour intervals. After initial examination, the limb is carefully realigned, and a posterior splint is applied for transportation.

Tibial shaft fractures are associated with fibular shaft fractures in 90% of cases. These fractures result from high-impact trauma. Before the development of higher, anatomically conforming ski boots, these fractures were the most common ski injuries (Fig. 24-47). The injury was sustained when the body rotated around a fixed foot (occurring with a ski caught against a rock or tree stump), which produced a torsional, spiral fracture of the tibia and fibula.

Tibial shaft fracture is the most common type of open fracture in the wilderness setting. When this injury is suspected, the entire limb should be inspected for distal sensory, motor, and circulatory function before realignment. A posterior splint is applied for transport. As this is the most common anatomic location for this problem, the limb is serially examined for the possibility of a compartment syndrome.

Ankle

The intra-articular distal tibia, medial malleolus, and distal fibula, or any combination of these, may be involved in an ankle fracture, which is generally produced by large torsional moments of force about a fixed foot. With the distal tibia fracture, axial loading from a fall or jump may also be involved. Any significant pain and swelling should be noted as the shoe is removed. Palpation along the medial and lateral malleoli confirms the clinical suspicion. After the shoe is removed to inspect the skin for open wounds, a neurocirculatory examination is performed.

If there is a rotational deformity in the ankle, the ankle is realigned with gentle traction before applying a posterior splint with the ankle in neutral position. During transport, the limb is elevated above the level of the heart.

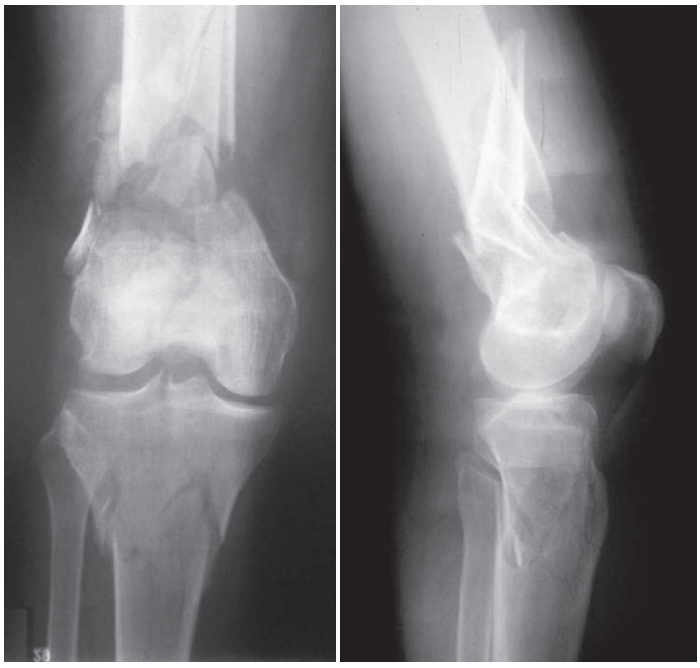


Figure 24-45. In higher-energy trauma, fractures of adjacent long bones can occur. These distal femur and proximal tibia fractures create the highly unstable situation of a "floating knee." Adequate and secure splinting is imperative to avoid further damage to neurovascular or soft tissue structures.

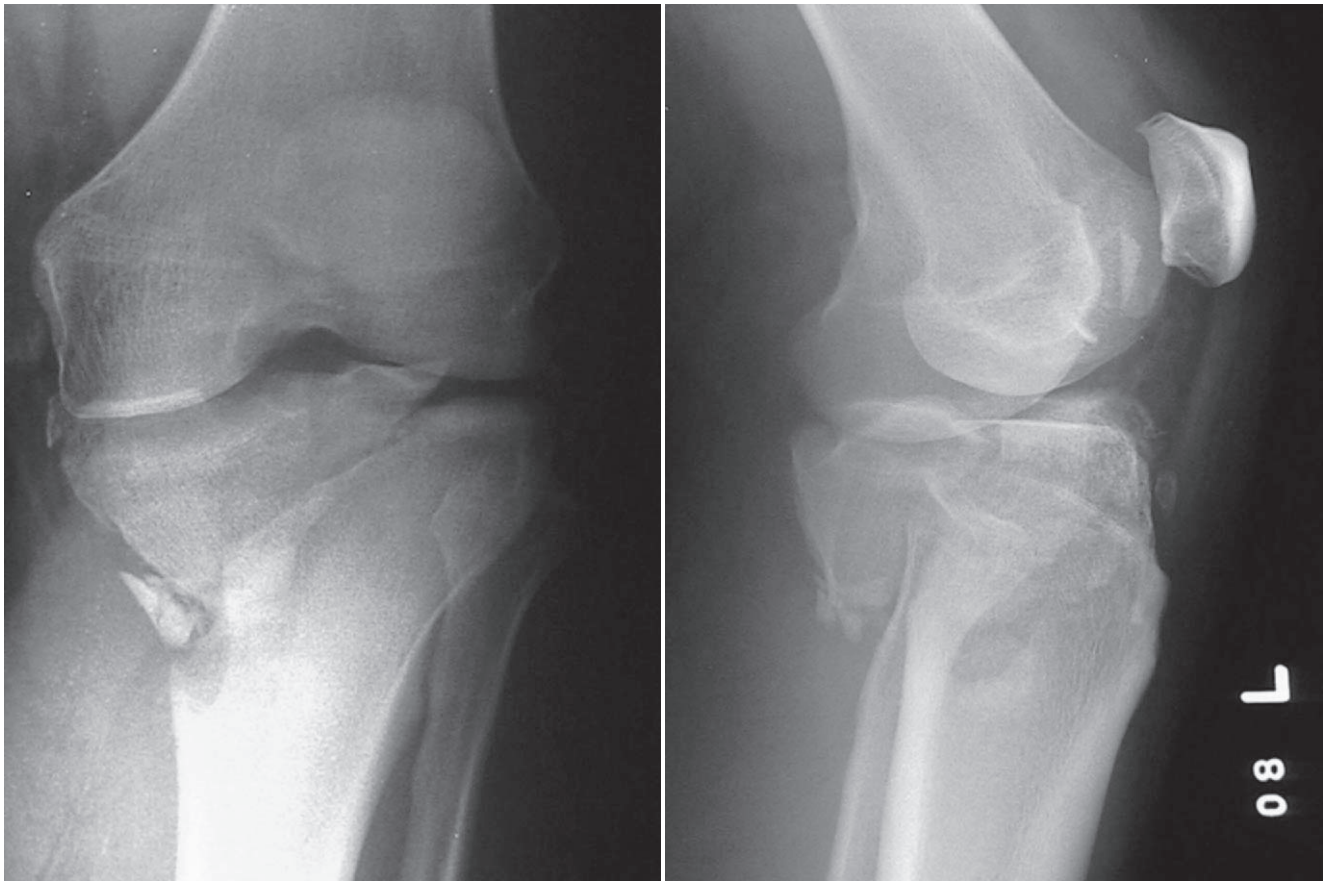


Figure 24-46. Anteroposterior and lateral plain radiographs of a tibial plateau fracture that occurred as a result of a fall. When a high-energy knee or lower extremity injury is sustained, always check for pulses, sensation, motor function, and indicators of compartment syndrome, such as pain with passive stretch.

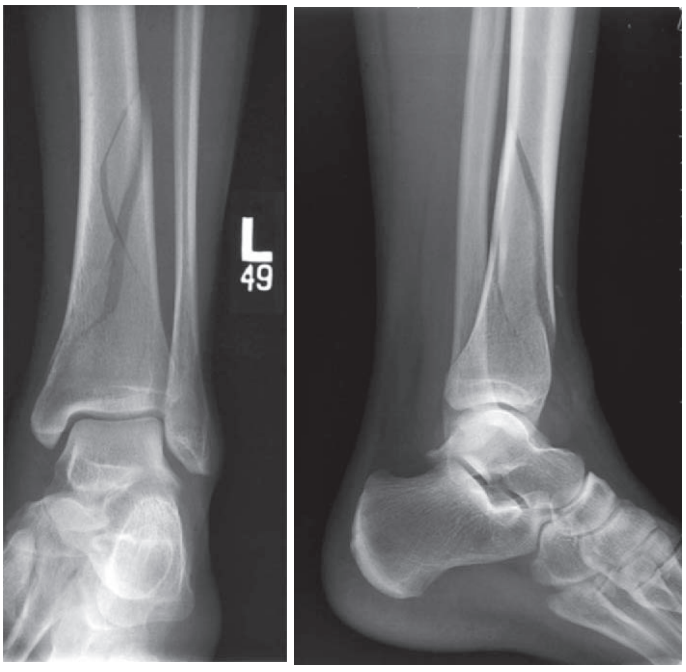


Figure 24-47. Anteroposterior and lateral plain radiographs of the distal tibia of a 30-year-old man after a twisting injury. This fracture should be well immobilized in a short leg splint, and the victim should be evacuated.

Figure 24-48. A talar neck fracture, when suspected, should be treated as an emergency. Restoration and fixation of alignment provides the greatest chance for maintenance of the tenuous blood supply of the talar dome.

Tarsal Bones

The calcaneus and talus are usually fractured during falls or jumps from significant heights when the victim lands on his or her feet. With a calcaneus fracture, significant heel pain, deformity, and crepitus are immediately evident after the boot is removed. A talus fracture may be impossible to differentiate from an ankle fracture on clinical grounds. An ankle fracture is tender at the malleolus level, whereas with a talus fracture, the tenderness is located distal to the malleoli. Talus fracture usually occurs when the foot is forced into maximal dorsiflexion. Knowing the point of the foot's impact with the ground is helpful in differentiating a talus fracture from an ankle fracture (Fig. 24-48).

Talus fracture may be associated with subtalar or ankle joint dislocation. Emergency evacuation should be arranged when this injury is suspected because these injuries are very difficult to reduce closed, and pressure on the skin from the displaced talar body can produce significant skin slough. If a talar neck fracture is suspected (admittedly difficult to diagnosis without plain radiograph), immediate evacuation is indicated; the blood supply to the talar dome, in part via the talar neck, is tenuous (Fig. 24-49). Therefore, to prevent post-traumatic avascular necrosis that may result from the fracture and disrupted blood supply, expedited evacuation is indicated.

Fractures of the other tarsal bones are exceedingly rare but can be defined by localizing the tenderness to a specific site. A short-leg splint with extra padding is applied and the limb is elevated during transportation.

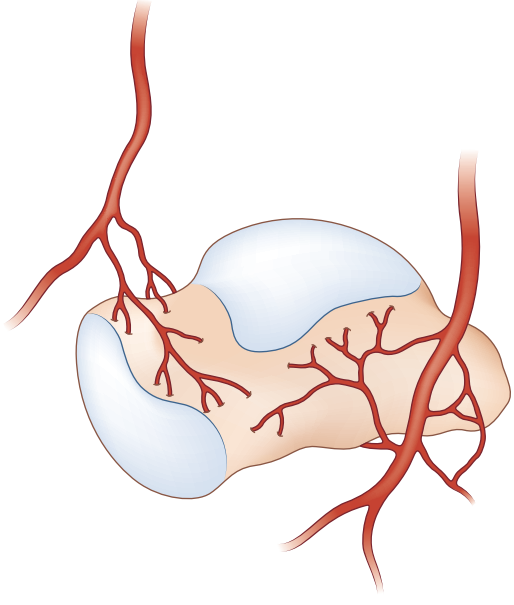


Figure 24-49. The talar blood supply. The talus receives contributions from the dorsalis pedis, posterior tibial artery, and perforating peroneal artery. (Redrawn from Myerson MS: Foot and Ankle Disorders. Philadelphia, WB Saunders, 2000, p 1325.)

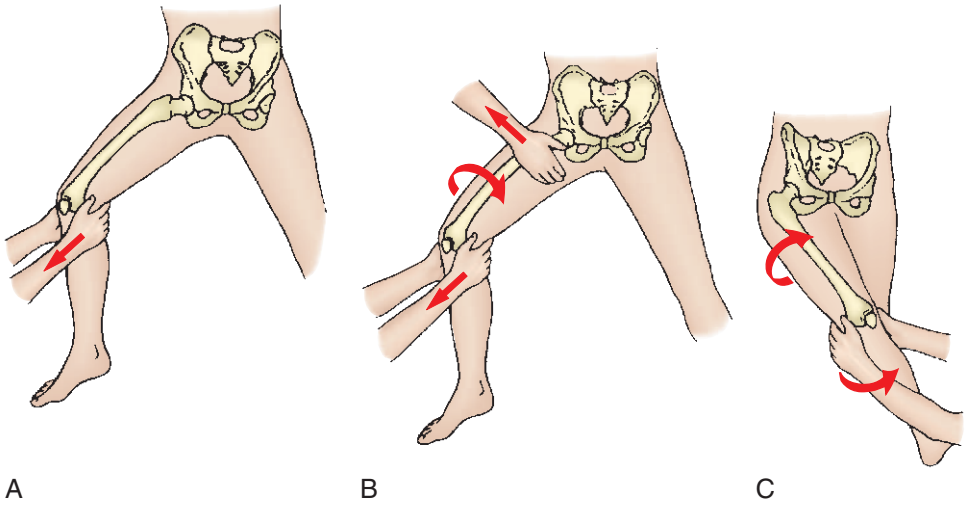


Figure 24-50. The Allis technique for reduction of a hip dislocation. The surgeon's position must provide a mechanical advantage for the application of traction. **A,** Internal and external rotations are gently alternated, perhaps with lateral traction by an assistant on the proximal thigh. **B,** In-line traction with hip flexed. **C,** Adduction is often a helpful adjunct to in-line traction. (**A** to **C** redrawn from DeLee JC: In Rockwood CA Jr, Green DP: Fractures, vol 2, 2nd ed. Philadelphia, JB Lippincott, 1985.)

Metatarsal Bones

Fractures at the base of the metatarsals often accompany midfoot dislocation (Lisfranc dislocation). The mechanism usually occurs with axial loading of the foot in maximal plantar flexion as a result of vehicle crash, most frequently snowmobiling. The victim complains of midfoot pain and swelling. On removing the shoe, crepitus and tenderness are noted at the base of the metatarsals (especially the first, second, and fifth metatarsals) and plantar ecchymosis may be present. Overall foot alignment is maintained, but stressing the midfoot by stabilizing the heel and placing force across the forefoot in the varus and valgus directions reveals instability. The foot is placed in a well-padded posterior splint and elevated whenever possible. The patient is not allowed to ambulate. Swelling associated with Lisfranc dislocation can produce a compartment syndrome.

Metatarsal shaft fracture occurs with a crush injury or a fall or jump from moderate height. Midshaft metatarsal fractures also occur as "fatigue" (or "march") fractures, which often result from prolonged hiking or running with poor preconditioning. Pain and localized tenderness are the hallmarks of this diagnosis. The dull pain at the midshaft of a metatarsal (often the second or fifth) may be converted to more severe pain with associated crepitus by a jump from a log or a rock. These fractures can be temporarily managed with a stiff-soled boot or orthotic insert. If there is fracture instability or extreme pain, a short-leg splint is applied and no further weight-bearing is allowed until more definitive evaluation and treatment can be accessed.

Phalanx

Toe phalanges are usually fractured by crush injuries that can be prevented by the use of steel-toed or hard-toed boots. A great-toe phalanx fracture can be a significant problem because force is placed on this digit during the toe-off phase of gait. Phalanx fractures are managed by buddy taping the toe to an adjacent uninjured digit with cotton placed between the toes. Displaced intra-articular fracture of the proximal phalanx of the great toe may need operative fixation. Stiff-soled boots minimize discomfort during weight bearing.

LOWER EXTREMITY DISLOCATIONS AND SPRAINS

Hip

Posterior hip dislocation is produced by axial loading of the femur with the hip flexed and adducted.[7] It generally occurs in a vehicle crash but can follow a fall or a sledding or skiing accident. With posterior dislocation, the victim complains of severe pain about the hip and the affected limb appears shortened, flexed, internally rotated, and adducted. Any hip motion increases the pain. It is not clinically possible to determine if there is an associated acetabular or femoral neck fracture. With the rare anterior dislocation, the limb is externally rotated, slightly flexed, and abducted. This type of dislocation is generally produced by wide abduction of the hip caused by significant force.

The victim is placed in the supine position for a complete survey of all organ systems. The distal limb is carefully examined for associated fractures, and a thorough sensory and motor examination is performed. The peroneal division of the sciatic nerve is most susceptible to injury with a posterior dislocation. Hip dislocations are an orthopaedic emergency because time to reduction is directly linked to the incidence of avascular necrosis of the femoral head. Immediate transfer to a definitive care center is desirable because hip radiographs may reveal an associated femoral neck fracture that could become displaced if closed reduction is attempted. However, if it will be more than 6 hours before the victim can be evacuated to a definitive care center, closed reduction should be attempted. To perform the Allis technique (Fig. 24-50), the victim is positioned supine on the ground or a stretcher. Analgesic medication is administered, if available. The caregiver stands above the victim and applies in-line traction on the extremity while an assistant applies counter-traction to the iliac wings. Posterior dislocations are reduced by flexing the hip 60 to 90 degrees. Internal rotation and adduction of the hip facilitates the reduction. With anterior dislocation, traction is applied with the leg slightly abducted and externally rotated and the hip gently extended. Successful reduction is usually indicated by an audible "clunk" and restoration of limb alignment. As with any reduction

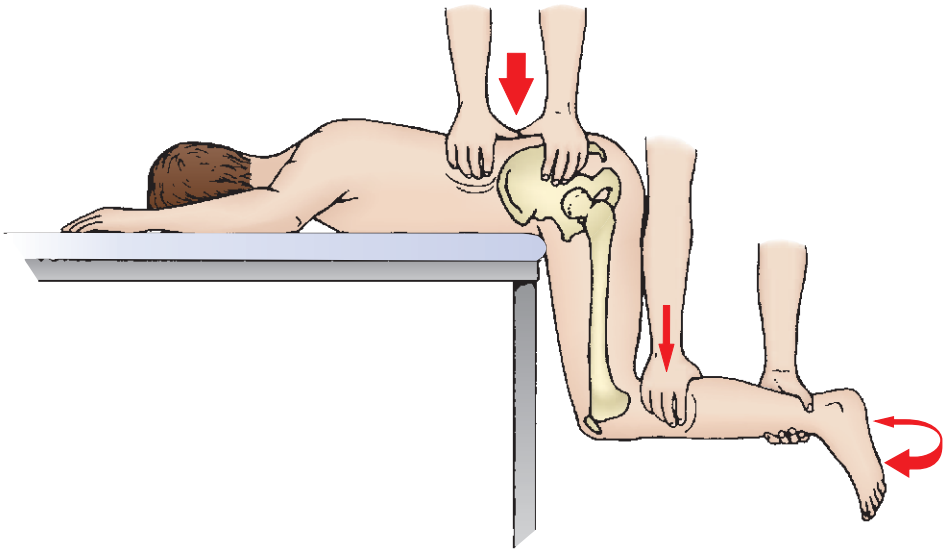


Figure 24-51. The Stimson gravity reduction technique. This method has limited application in patients with multiple injuries. (Redrawn from DeLee JC: In Rockwood CA Jr, Green DP: Fractures, vol 2, 2nd ed. Philadelphia, JB Lippincott, 1985. Copyright © 1990 by Jesse C. DeLee.)

maneuver, adequate analgesia and a slow, progressive increase in traction force are helpful. For the Stimson gravity technique (Fig. 24-51), which may not be as practical in the wilderness, the patient is positioned prone on a makeshift platform. With a posterior dislocation, the hip and knee are flexed 90 degrees. Longitudinal traction is applied in addition to adduction and internal rotation. The reduction maneuver is actually the same for both techniques, but one is performed with the victim supine and the other with the victim prone.

Knee

Knee dislocation is usually obvious because of the amount of deformity (Fig. 24-52). The tibia may be dislocated in one of five directions: anterior, posterior, lateral, medial, or rotatory. The most common directions are anterior and posterior. This represents a true emergency because 5% to 40% of knee dislocations have associated vascular injuries.[1,15,19] In a large series, Green and Allen[6] reported an above-knee amputation rate of 86% for vascular injuries associated with knee dislocations that were not repaired within 8 hours of injury. The vascular injury occurs because of tethering of the popliteal vessels along the posterior border of the tibia by the soleus fascia. A knee dislocation is a high-velocity injury usually produced by a vehicle crash or a fall. When this injury is suspected, a careful screening neurocirculatory examination must be performed. Intact distal pulses do not definitively rule out an arterial injury. Intimal flap tears can produce delayed thromboses of the popliteal artery. In addition, injury to the peroneal nerve can occur.

Many knee dislocations spontaneously reduce and may lead the examiner to underestimate the seriousness of the injury. Instability in extension to either varus or valgus stress indicates disruption of at least one of the cruciate ligaments and signifies the potential for a knee dislocation.

After the initial examination, the persistent dislocation should be reduced. Anterior dislocation is reduced with traction on the leg and gentle elevation of the distal femur. Posterior dislocation is reduced with traction in extension and anterior elevation of the tibia. Posterolateral rotatory dislocation can be very dif-

Figure 24-52. Anterior knee dislocation. Significant attention should be devoted to relocation, if possible, and to assessment of the neurovascular structures that run posterior to the knee joint, such as the popliteal artery and the tibial and common peroneal nerves.

ficult to reduce and usually requires open reduction. It occurs when the medial femoral condyle buttonholes through the medial capsule. A transverse furrow on the medial aspect of the knee is pathognomonic for this injury. For transport, a posterior splint is applied to the limb and the victim is moved on a backboard. The possibility of an arterial lesion or emerging compartment syndrome requires vigilance. Emergency evacuation is advised because of the risk of amputation related to vascular injury.

Frequently, the patellofemoral joint is dislocated. Because of the increased femorotibial angle in females, this injury is far more common in women. Generalized ligamentous laxity may also predispose to this problem. Dislocation of the kneecap may result from a twisting injury or an asymmetric quadriceps contraction during a fall. These mechanisms routinely occur with hiking, climbing, and skiing accidents. The patella dislocates to a position lateral to the articular surface of the distal femur. Although neurovascular injuries rarely occur in association with a dislocated patella, a screening examination should be conducted.

The patella can often be reduced by simply straightening the knee. If this is not successful, gentle pressure is applied to the patella to push it back up onto the distal femoral articular groove. A knee splint is applied with the joint in extension; weight bearing is allowed. The knee is kept in extension until definitive care can be obtained. A radiograph is ultimately required to rule out osteochondral fractures, which are frequently associated with this injury.

Ankle

Ankle dislocations are almost always accompanied by fractures of one or more malleoli. These dislocations generally occur with falls onto uneven surfaces or with twisting injuries of moderate velocity. The area about the ankle is carefully examined for open injuries, and a neurocirculatory examination is conducted to obtain a baseline status. Then, the ankle joint is aligned by grasping the posterior heel, applying traction with the knee bent (to relax the gastrocnemius–soleus complex), and bringing the foot into alignment with the distal tibia. After this maneuver, the foot is reexamined, any wounds are dressed, and a posterior splint is applied. During transport, the limb is elevated above the level of the heart.

The most common musculoskeletal injury occurring in the wilderness setting is an ankle sprain. Ligament sprain, or tearing of the fibers, is separated into three grades. Grade 1 injury is partial disruption of some of the ligament fibers. Grade 2 injury is complete disruption of a portion of the ligament fibers. The main substance of the ligament remains intact, and the injury is characterized by moderate hemorrhage with grossly visible torn ligament fibers. Grade 3 injury is complete disruption of the ligament fibers, which can result in instability of the ankle joint.

The medial ligament complex consists of the deltoid ligament, which runs from the medial malleolus to the talus (Fig. 24-53). The ligament complex on the lateral side is much more intricate and consists of three separate ligaments named for their origins and insertions: the calcaneofibular ligament, the anterior talofibular ligament, and the posterior talofibular ligament (see Figure 24-53). The lateral ligament complex is the most frequent site of an inversion injury. When such an injury occurs, the shoe and sock are removed and a screening neurocirculatory examination is conducted. Each ligament is palpated individually for tenderness, and the ankle is evaluated for instability with the anterior drawer test. This test is performed by stabilizing the tibia with one hand and grasping the posterior heel to pull the foot forward with the other hand. If the talus slides forward within the ankle mortis (using the uninjured side as a comparison), the injury represents a grade 3 injury. The foot and ankle are placed into a posterior splint or air splint. If possible, the victim is kept from bearing weight on the limb. If this examination does not reveal instability and is thus indicative of a grade 1 or 2 sprain, an elasticized bandage is applied or the ankle is taped (see Chapter 18). All injuries should be acutely treated following the RICE principle. Commercially available stirrup air splints also aid in ambulatory management of these injuries.

A more serious ankle sprain is the high ankle sprain, which affects the anterior inferior tibiofibular ligament (a portion of the syndesmotic ligament) and occurs in up to 10% of ankle sprains. The victim complains of pain to palpation over the distal tibiofibular joint and also with dorsiflexion and external rotation of the foot relative to the tibia. Compression of the fibula and tibia in the proximal half of the calf produces pain over the syndesmosis. Unlike stable lateral ankle sprains, these injuries take 4 to 6 weeks to resolve. Initial treatment is with a short-leg splint or walking boot. Failure to recognize this injury will produce prolonged disability.

In the field, the ankle is taped both to decrease pain and to limit swelling (Fig. 24-54, and see Chapter 18). During taping, the victim's ankle is kept perpendicular to the tibial shaft. This makes walking easier, because the ankle is not plantar flexed, and helps prevent development of an Achilles tendon contracture. If available, an Aircast ankle brace provides additional ankle support and can be used with a shoe or boot.

A fracture of the lateral process of the talus, which is a fairly common lower extremity fracture in snowboarders, may be confused with a lateral ankle sprain, so radiographs are generally needed to rule out this injury.[9] Inversion injuries are also infrequently associated with fractures at the insertion of the peroneus brevis tendon. This injury can be identified by the point tenderness at the base of the fifth metatarsal, but a radiograph is required for definitive diagnosis. Early management is the same as for an ankle sprain.

Hindfoot

The subtalar joint may infrequently be dislocated in a significant fall or jump when an individual lands off balance or on an uneven surface. The calcaneus may be dislocated medially or, more commonly, laterally relative to the talus (Fig. 24-55). The position of the heel relative to the ankle is assessed. With either dislocation, a reduction is attempted if it will be more than 3 hours until the victim will reach a definitive care center.

Medial dislocation is reduced more easily than lateral dislocation, in which the posterior tibial tendon frequently becomes displaced onto the lateral neck of the talus, blocking the reduction. The maneuver is the same for both: the heel is grasped with the knee flexed (relaxing the gastrocnemius–soleus complex), the deformity is accentuated, linear traction is applied, and the heel is brought over to the ankle joint. This maneuver is generally successful for medial dislocation, but lateral dislocation, especially when associated with open wounds, often requires open treatment. After reduction is attempted, a posterior splint is applied and the limb is elevated

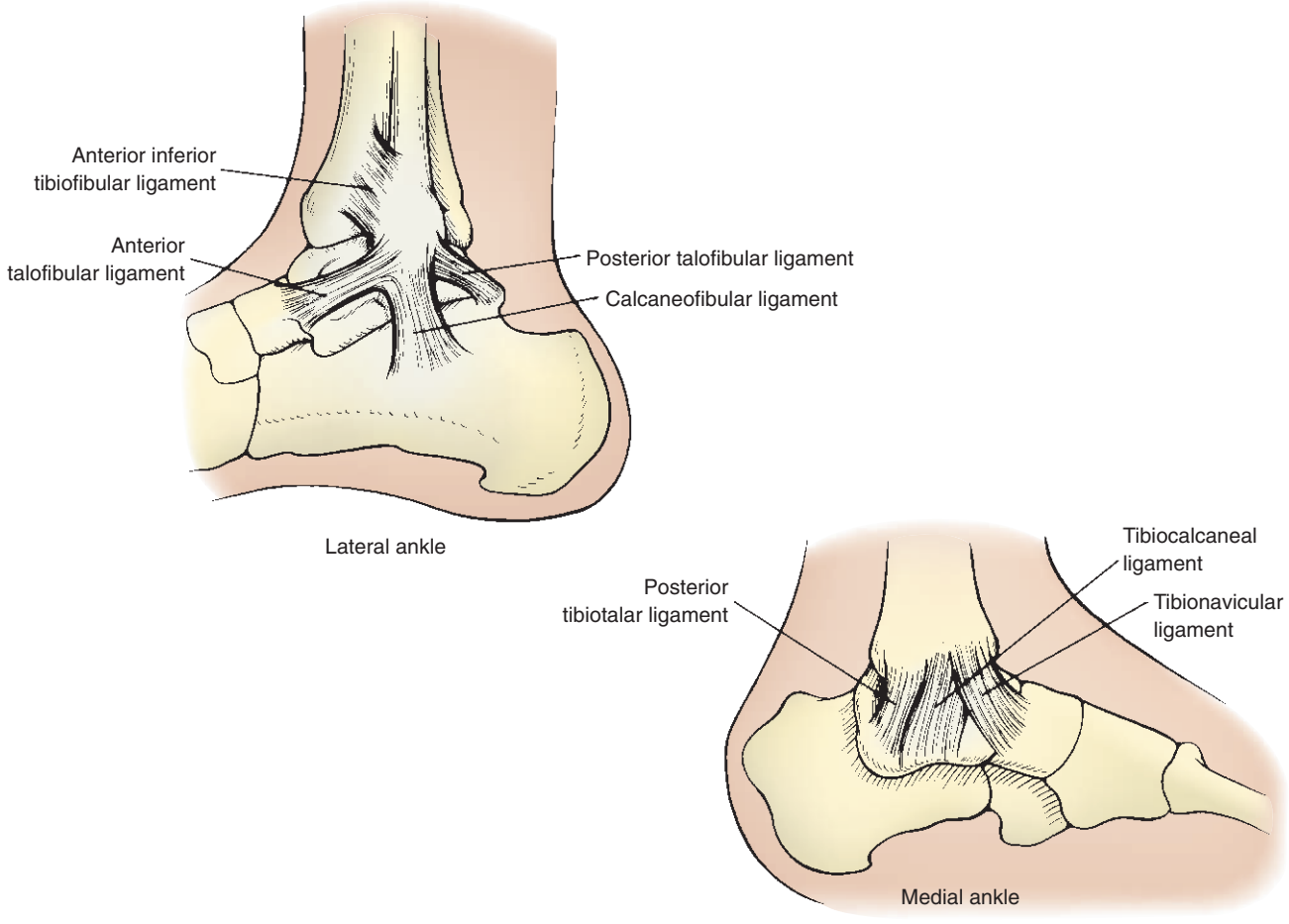


Figure 24-53. Ligament complexes of the ankle.

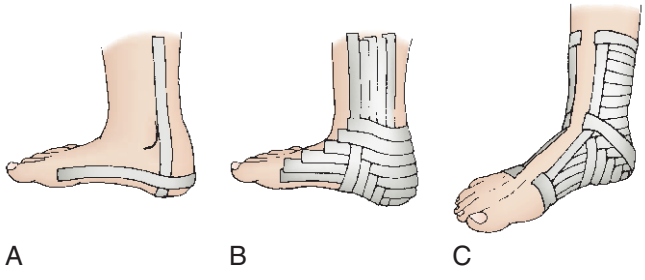


Figure 24-54. Taping a sprained ankle. Strips of adhesive tape are placed perpendicular to each other **(A)** to lock the ankle with a tight weave **(B).** The tape edges are covered to prevent peeling **(C).** (From Auerbach PS: Medicine for the Outdoors: The Essential Guide to Emergency Medical Procedures and First Aid, 4th ed. Guilford, CT, Lyons Press, 2003.)

above the heart. Even if the reduction is successful, the victim must not be allowed to bear weight until definitive care is obtained.

Midfoot

Midfoot fracture dislocation (Lisfranc injury) is described in the metatarsal fracture section.

Metatarsophalangeal and Interphalangeal Joints

Metatarsophalangeal (MTP) joint dislocations of the toes are relatively uncommon but can occur when a moderate axial force is directed at the great toe. Crush injuries and rock-climbing accidents while the victim is wearing flexible-sole shoes can produce this injury; wearing boots with reinforced toe boxes of adequate depth generally prevents it. Injuries of this type at the great toe may be associated with fractures of the metatarsal joint or phalanx. The dislocation is generally dorsal. Because these may be open injuries, the foot must be inspected carefully. The joint is reduced in a manner similar to that used for dorsal

Figure 24-55. Ankle and hindfoot of a man with a calcaneus fracture. Soft tissue swelling from a fracture or crush injury can result in swelling that contributes to a compartment syndrome.

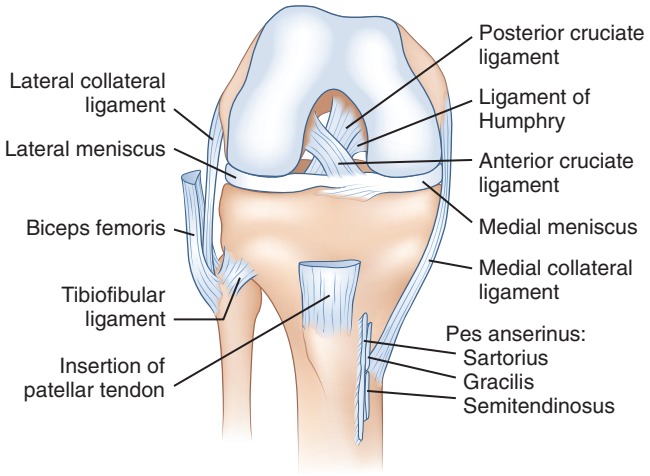


Figure 24-56. Anterior view of a partially dissected knee joint. (Redrawn from Insall JN, Scott WN [eds]: Surgery of the Knee, 2nd ed. New York, Churchill Livingstone, 1993, p 2.)

PIP joint dislocation of the hand. MTP dislocation of the great toe can occasionally require open reduction if the head of the metatarsal buttonholes through the sesamoid–short flexor complex.

The lesser MTP joints are generally dislocated laterally or medially. The most common mechanism for this injury is striking unshod toes on immovable objects. The toes are relocated by applying linear traction with the victim supine and using the weight of the foot as countertraction. Similar mechanisms produce dislocations of the IP joints, which are also reduced by applying linear traction with gentle manipulation. Once reduced, the injured toe is taped to the adjacent toe for 1 to 3 weeks, and the victim wears a protective boot with a stiff sole and deep toe box.

OTHER SOFT TISSUE AND MUSCULOSKELETAL INJURIES

Intra-articular Knee Disruption

Sprains or ruptures of ligaments about the knee (Fig. 24-56) are common occurrences in sporting events and in vigorous activities. They often involve a sudden fall or a twisting mechanism.

Anterior cruciate ligament (ACL) injuries often result from twisting or pivoting, or from hyperextension. The individual who sustains this injury often sustains concomitant knee injuries, such as a meniscus tear or an injury to another ligament in the knee. Tearing of the ACL may be accompanied by the sound of a pop in the knee. Usually, but not always, the knee becomes swollen. A sense of instability is common. On examination, there may be marked laxity to anterior stress on the tibia relative to the femur (Lachman test).

Although it may be easy to produce mobilization in extension, ACL injuries do not necessarily require immobilization. If the mechanism of the injury suggests that it is more likely to be ligamentous than a fracture, weight bearing, as tolerated, and range of motion, as tolerated, are safe recommendations. Nonsteroidal anti-inflammatory drugs (NSAIDs), compression dressing, and ice, if it is available, are all palliative and might enhance the victim's ability to leave the wilderness setting and obtain more definitive diagnosis and treatment.

An individual who incurs an ACL tear might also sustain a posterior cruciate ligament (PCL) injury, an MCL injury, or a lateral cruciate ligament (LCL) injury. Sprain or frank disruption of any of these ligaments may result in a sense of instability in the plane controlled by that ligament (for example, valgus instability in the setting of an MCL injury).

Meniscal tears in the setting of a ligamentous injury are common. Some tears—called bucket-handle tears—become caught in the joint. These can result in a locked knee. Manipulative procedures have been described for liberating an incarcerated meniscal fragment. However, unless evacuation is substantially hindered by the locked knee, it is generally better to get the injured individual as quickly as possible to a facility that can provide a magnetic resonance imaging (MRI) study. Sometimes, surgery is the sole solution for this problem.

Achilles Tendon Rupture

Achilles tendon ruptures are fairly common in the 35- to 55-year age group. These injuries usually occur as an individual applies an eccentric load to the calf and Achilles tendon. The affected individual often feels as though he has been struck by a baseball bat at the posterior aspect of the ankle. Active plantar flexion is impossible. In the Thompson squeeze test, the injured person kneels on an elevated surface without sitting on the haunches and allows the feet to hang over the edge of the surface. Squeezing the calf does not result in an involuntary plantar flexion on the injured side. This lack of motion suggests that there is no connection between the gastrocnemius and soleus muscle bellies and the Achilles attachment to the calcaneus.

If a short-leg splint can be applied as a temporizing measure, this should be done. The affected individual may bear weight as tolerated. This injury may be treated with or without surgery.

Hamstring Pull or Tear

A hamstring pull or tear can occur when there is a hyperflexion mechanism applied to the hip simultaneously with a hyperextension mechanism to the leg. This can also occur as an overuse injury. Generally, the best way to treat this is to apply ice, stretch gently, and bear weight, as tolerated.

Effusions

Joint or bursal effusions occur when there is excess production of synovial fluid. Effusions may result from trauma (for example, hemarthrosis as a result of an ACL tear, or lipohemarthrosis as the result of a tibial plateau fracture). If an effusion can be aspirated under sterile conditions in the wilderness, aspiration may be of value for comfort and for diagnosis. If it is not possible to perform aspiration under sterile conditions, the effusion should be treated with NSAIDs, compression dressing, and application of cold.

OVERUSE SYNDROMES

Overuse syndromes that affect the musculoskeletal system are a very common form of injury in the wilderness.

Plantar Fasciitis

Plantar fasciitis is inflammation of the fascia (the tough connective tissue) on the sole of the foot. An individual with plantar fasciitis complains of insidious onset of pain at the origin of the plantar fascia, which is located at the most anterior aspect of the heel pad. Any activities that stretch the plantar fascia elicit pain. The pain is worst when first getting up in the morning or after resting, and it is accentuated when the ankle and great toe are dorsiflexed (e.g., during push-off). Conservative treatment consists of (1) heel cord stretching 20 minutes twice a day, (2) NSAIDs, and (3) wearing an orthotic that cups the heel, has a soft spot under the tender area, and supports the arch. It may take several weeks for symptoms to improve, but conservative therapy is successful in 90% of cases. An ankle–foot splint worn at night may help because it holds the foot in a neutral position, keeping the plantar fascia slightly stretched. The orthosis also provides significant pain relief if used while walking.

In severe cases, taping the arch can provide pain relief (Fig. 24-57). A thin layer of benzoin or spray tape adhesive is applied to the bottom of the foot. An anchor strip of $^{3}/_{4}$-inch adhesive tape is fixed in a U shape around the heel from just under the malleoli (the prominences of the ankles) up to just behind the level of the "knuckles" of the toes (see Figure 24-57A). Next, fairly tight cross-strips of $^{1}/_{2}$-inch tape are laid across the bottom of the foot, with their ends torn to lie on the anchor strip (see Figure 24-57B). This creates a "sling" of tape under the foot for support. Finally, another U-shaped piece of tape is applied around the heel; it crosses under the center of the arch and locks down the crosspieces (see Figure 24-57C).

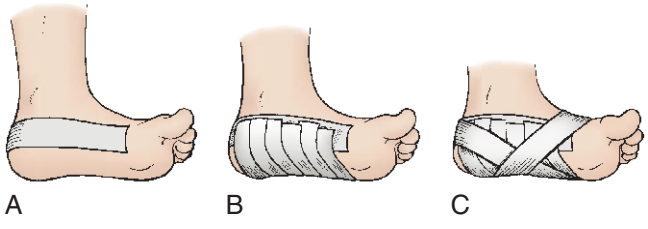


Figure 24-57. Taping for arch support. **A,** Fix an anchor strip under the heel. **B,** Attach strips across the bottom of the foot. **C,** Lock the crosspieces. (From Auerbach PS: Medicine for the Outdoors: The Essential Guide to Emergency Medical Procedures and First Aid, 4th ed. Guilford, CT, Lyons Press, 2003.)

Carpal Tunnel Syndrome

Carpal tunnel syndrome (CTS) occurs when the median nerve is compressed within the carpal tunnel (Fig. 24-58). The carpal tunnel, located on the palmar side of the wrist, is formed by the transverse carpal ligament volarly and the carpal bones dorsally. The flexor digitorum profundus and superficialis tendons to the second through fifth digits, the long thumb flexor, and the median nerve pass through this canal. Individuals with CTS complain of pain and paresthesias along the palmar aspect of the radial digits. They also complain of frequently dropping objects. Symptoms are worse at night and aggravated with prolonged wrist extension or flexion. The Phalen sign, which is numbness and tingling in the median nerve distribution after sustained wrist flexion, is suggestive of CTS. Thenar muscle atrophy is seen only in severe cases. Other causes should be considered, such as more proximal sites of nerve compression (especially the cervical spine), dialysis, pregnancy, and acute or chronic trauma. Treatment consists of wrist splinting in slight extension (especially at night), activity modification, and NSAIDs.

Tibial Fatigue Fractures

Tibial fatigue fractures can occur in individuals who suddenly increase their activity. Victims complain of pain with weight bearing, swelling, tenderness to palpation, and increased warmth at the fracture site. The most common site in the tibia is the proximal two thirds of the tibial diaphysis. Stress fracture of the distal third of the fibula can also occur. Treatment consists of activity reduction, protective weight bearing, and avoidance of activities that produce pain. Failure to decrease activity level complicates fracture of the tibia. As the pain subsides, the activity level can be increased. Two to 3 months may be required for resolution of symptoms.

EVACUATION DECISION

The decision to evacuate an orthopedically injured individual depends on the goals of and the support available to the expedition. The criteria for evacuation of an injured person in a family of four spending a week hiking in the Rockies differ from those of a group of 25 climbers in the Himalayas with physician support and a field hospital at the base camp. In all cases, party leaders should have a plan for contacting evacuation support teams if a serious injury occurs.

Musculoskeletal injuries that warrant immediate evacuation to a definitive care center are listed in Box 24-2. These include

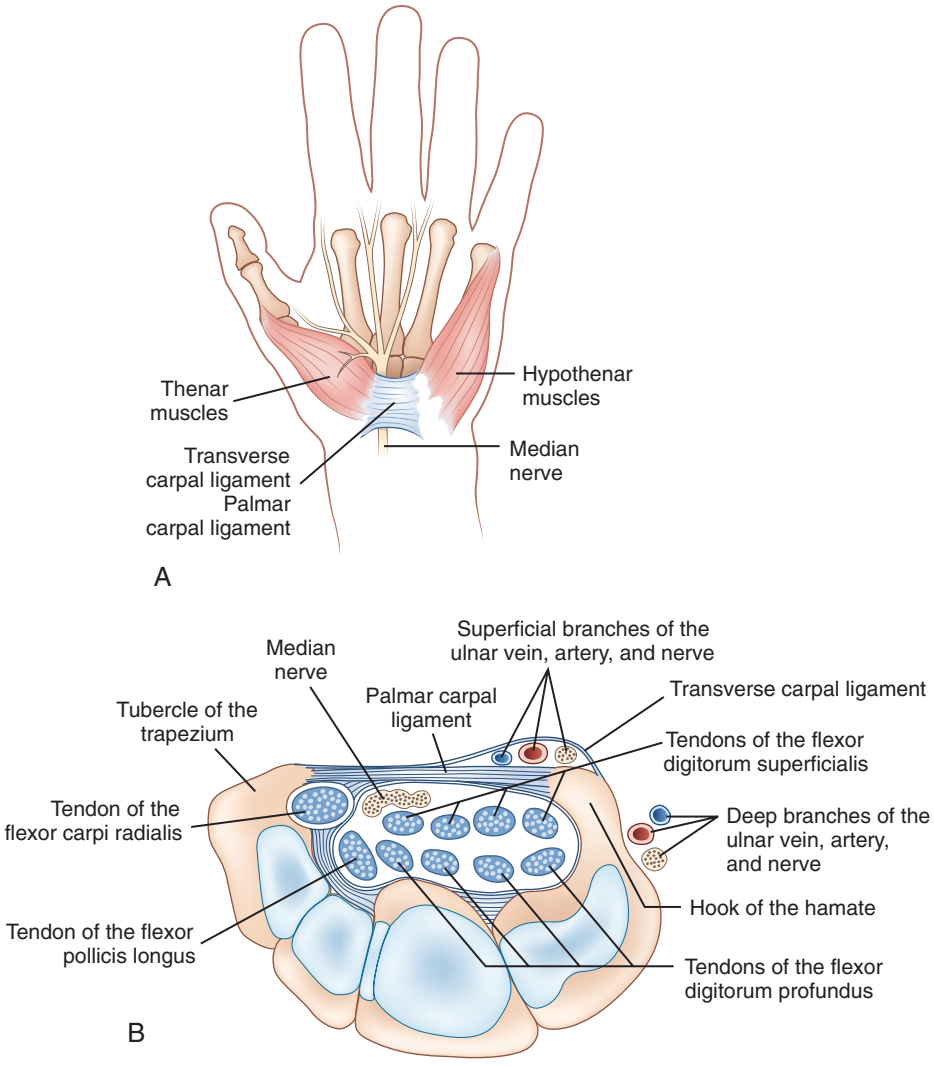


Figure 24-58. Anatomic basis of carpal tunnel syndrome. **A,** General view of the relationship between the median nerve and the flexor retinaculum. **B,** Cross section at the distal carpal row, showing the structures in the carpal tunnel. (Redrawing based on an illustration by Li-Guo Liang, in Yu HL, Chase RA, Strauch B: Atlas of Hand Anatomy and Clinical Implications. Philadelphia, Mosby, 2004, p 513.)

Box 24-2. Indications for Emergent Evacuation

Suspected spine injury
Suspected pelvic injury
Open fracture
Suspected compartment syndrome
Hip or knee dislocation
Vascular compromise to an extremity
Laceration with tendon or nerve injury
Uncertainty of severity of injury

any suspected cervical, thoracic, or lumbar spine injury. A victim who has a suspected pelvic injury with posterior instability, possibly significant blood loss, or injury to the sacral plexus should be emergently evacuated on a backboard. Any open fracture requires definitive debridement and care within 8 hours to prevent development of deep infection, so it should prompt emergency evacuation. Victims with suspected compartment syndromes must be evacuated on an emergency basis. Joint dislocations involving the hip or knee warrant immediate evacuation because of the associated risk of vascular injury or post-traumatic avascular necrosis of the femoral head.

Lacerations involving a tendon or nerve warrant urgent evacuation to a center where an extremity surgeon is available. In all but the most serious wilderness expeditions, arrangements should be made to evacuate the victim when the treating individuals are not reasonably sure of the injury with which they are dealing or its appropriate management.

The references for this chapter can be found on the accompanying DVD-ROM.

25 The Eye in the Wilderness

Frank K. Butler, Jr.

This chapter considers several commonly encountered types of eye disorders: periocular trauma; chemical injury to the eye; sudden vision loss in a white, quiet eye; acute orbital and periorbital inflammation; and the acute red eye. A diagnostic and therapeutic approach to these disorders suitable for the wilderness environment is presented. Finally, eye problems that are encountered in diving and altitude exposures are discussed.

PRELIMINARY PLANNING

Pertinent ocular items in a preliminary medical survey include contact lens wear; previous episodes of nontraumatic iritis; previous episodes of herpetic keratitis; and a history of corneal transplantation, retinal detachment, refractive surgery, or other ocular surgery. A positive response to these questions may alert the health care professional to specific ocular problems that may be encountered on the proposed trip. Congenital color vision deficiencies are common in the male population (8% in whites)[27] and typically pose no functional problems in the wilderness.

In addition to reviewing ocular health in the medical survey, a basic wilderness eye kit should be assembled and taken on the trip, as described in the next section.

THE WILDERNESS EYE EMERGENCY KIT

Box 25-1 lists the suggested items for a basic wilderness ocular emergency kit. A topical fluoroquinolone, such as moxifloxacin, is the antibiotic eye drop of choice. These medications are preferred for treatment of bacterial keratitis in a wilderness setting. Topical tetracaine and fluorescein strips are important for diagnosis. Topical prednisolone is an excellent ocular anti-inflammatory medication.

The choice of an oral antibiotic is based on the efficacy of the proposed antibiotic in treating preseptal cellulitis, orbital cellulitis, and penetrating trauma to the globe. The fluoroquinolone family of antibiotics offers several good choices for these indications. The new fourth-generation fluoroquinolone moxifloxacin provides good coverage against gram-positive, gram-negative, and anaerobic organisms, with a relatively mild side effect profile and once-daily dosing, and is the recommended first-line oral antibiotic.[92] Gatifloxacin has recently been withdrawn from the market because of dysglycemic side effects. Trovafloxacin (500-mg tablets) is a systemic fluoroquinolone with excellent coverage against gram-positive, gram-negative, and anaerobic bacteria.[54,130] It also offers the convenience of once-daily dosing, but there have been a number of unpublished reports of hepatotoxicity with trovafloxacin that limit the usefulness of this medication.[98] Levofloxacin (500-mg tablets) is another systemic fluoroquinolone with very good activity against a wide variety of gram-positive and gram-negative organisms but less anaerobic coverage than trovafloxacin.[98] Ciprofloxacin has been shown to have excellent ocular penetration when given orally,[73] but is less efficacious against gram-positive organisms than is levofloxacin. Bacitracin is an antibiotic ointment suitable for use in patching corneal abrasions. Ophthalmic ointments are best applied by using downward pressure on the lower lid to pull it away from the eye and then applying a 1-cm (0.4-inch) ribbon of ointment to the conjunctiva of the lower lid. When released, the lid returns to its normal position and normal blinking distributes the ointment over the corneal surface.

Oral prednisone has three possible treatment uses in the wilderness—refractory iritis, giant cell arteritis, and orbital pseudotumor. Topical scopolamine (0.25%) is used to reduce ciliary muscle spasm, which causes much of the discomfort associated with iritis and corneal abrasion. Scopolamine, however, has the disadvantage of dilating the pupil (making the eye very sensitive to bright light) and preventing accommodation (making reading very difficult) for 5 to 7 days. Artificial tears are used to treat ocular surface drying and to flush conjunctival foreign bodies from the eye.

Artificial tears are a better choice for these symptoms than are ophthalmic preparations containing vasoconstrictors because ocular decongestants have been shown to cause both acute and chronic conjunctivitis.[124] Diclofenac 0.1% drops have been shown to decrease corneal sensitivity, especially when multiple drops are used.[112,128,129] They have been found helpful in reducing the discomfort associated with traumatic corneal abrasion[60] and excimer laser refractive surgery.[40,131,135] In the unlikely event of angle-closure glaucoma in a wilderness setting, 2% pilocarpine may be used. The medications in Box 25-1 are listed in my recommended priority order. In the spirit of making do in the wilderness, all of the disorders mentioned in this chapter can be managed with only the aforementioned medications, but alternative therapies are also discussed.

Many individuals suffer from seasonal allergies, including allergic conjunctivitis, that may be exacerbated in the wilderness. Adding olopatadine 0.1%, lodoxamide 0.1%, nedocromil 2%, or ketotifen 0.025% to the wilderness eye kit provides treatment options for allergic conjunctivitis.[70] Because this condition is often a chronic one, however, individuals often carry their own medications on trips.

Box 25-1. The Wilderness Eye Emergency Kit

MEDICATIONS
Moxifloxacin 0.5% drops
Tetracaine 0.5% drops
Prednisolone 1% drops
Moxifloxacin 400 mg tabs
Levofloxacin 500-mg tabs
Bacitracin ointment
Prednisone 20-mg tabs
Artificial tears
Scopolamine 0.25% drops
Diclofenac 0.1% drops
Pilocarpine 2% drops

MISCELLANEOUS
Penlight with blue filter
Fluorescein strips
Cotton-tipped applicators
Metal eye shield
Eye patches (gauze)
Tape (1 inch, plastic or nylon)
Near vision card
Wound closure strips ($^1/_4$ inch)
Magnifying glass

Box 25-2. Differential Diagnosis of Acute Periocular Inflammation

Preseptal cellulitis
Orbital cellulitis
Dacryocystitis
Orbital pseudotumor
Insect envenomation

VISUAL ACUITY MEASUREMENT IN THE WILDERNESS

Evaluation of visual acuity is an essential element of the eye examination. Serial measurements of visual acuity are used to monitor an individual's progress while being treated for an eye disorder. Lack of an eye chart does not preclude the ability to obtain some quantitative measure of visual acuity. A near-vision card can be used for this purpose. It should be held the prescribed distance—usually 35 cm (14 inches)—from the eye. If a near card is not available, the ability to read print in a book is a useful alternative measure. If glasses have been lost, use a piece of paper with a pinhole created by the tip of a pen or pencil to help compensate for the lost refractive correction. Remember that individuals 40 years of age and older may need a pinhole or reading correction to help them focus on a near target. Although a marked decrease in visual acuity can be an important warning of a significant ocular disorder, visual acuity cannot always be considered a reliable indicator of the severity of disease. A corneal abrasion victim may initially have worse visual acuity than a person with a retinal detachment or corneal ulcer, despite the fact that the latter two entities are much more serious disorders.

GENERAL THERAPEUTIC APPROACH

The recommendations made in this chapter are not necessarily the preferred management of the disorders mentioned when one is not in the wilderness setting. Of special interest is the recommendation for a nonophthalmologist to use a topical steroid in the management of several of the disorders discussed. The use of topical ocular steroids is generally best undertaken by ophthalmologists for two reasons: first, steroids are usually indicated only for relatively serious ocular disorders, which should be followed by an ophthalmologist when possible. Second, topical steroid use may result in elevated intraocular pressure, cataracts, and exacerbation of certain eye infections. All of the disorders for which steroids are recommended in this chapter should be referred to an ophthalmologist for follow-up as soon as possible on return from the wilderness. Caution should be exercised in prescribing a topical steroid for longer than 3 days. Although cataracts are typically associated with long-term steroid use, a significant rise in intraocular pressure may occur within just a few days after initiation of topical steroid therapy.[70]

The requirement for expedited evacuation is one of the questions that must be answered when treating an eye disease in the wilderness. In the following sections, the need for evacuation may be considered nonurgent unless an emergent (as soon as possible) or expedited evacuation (as soon as is deemed reasonable given the resources required to accomplish the evacuation) is specified in the recommendations for treatment.

ACUTE PERIOCULAR INFLAMMATION

Causes of acute periocular inflammation are listed in Box 25-2. *Preseptal cellulitis* means that the infectious process is confined to the tissues anterior to the orbital septum. Preseptal cellulitis therefore presents as erythema and edema of the eyelids without restricted ocular motility, proptosis, pupillary change, or decrease in visual acuity. However, some of these findings may be difficult to appreciate in the presence of marked lid edema. Historical clues include antecedent periocular trauma or insect bite or sting. In the past, this disorder has been treated very aggressively because of the high incidence of *Haemophilus influenzae* infection, especially in pediatric patients, with subsequent septicemia and meningitis. The advent of *H. influenzae* type B vaccine has changed the microbiology of this disorder and may dictate changes in treatment strategies in the future.[31] Persons who present with this disorder may be treated with moxifloxacin 400 mg once a day and should have an expedited evacuation. Alternative antibiotic choices include levofloxacin 500 mg once a day, ciprofloxacin 750 mg twice a day, dicloxacillin 500 mg every 6 hours, or cephalexin 500 mg four times a day.

Dacryocystitis (infection of the lacrimal sac) may mimic the findings of preseptal cellulitis, but erythema, edema, and tenderness are localized to the area inferior to the medial aspect of

the eye, over the nasolacrimal sac and duct. The presence of this condition usually indicates obstruction in the opening between the lacrimal sac and the nasal cavity. Surgical intervention to restore the patency of this opening is usually undertaken after the acute infection is treated. The most common pathogens in acute dacryocystitis are *Staphylococcus aureus*, *Streptococcus* species, and (in children) *H. influenzae*.[91] Treatment should be initiated with moxifloxacin 400 mg once a day and warm compresses. Alternative antibiotic choices include levofloxacin 500 mg once a day, ciprofloxacin 750 mg twice a day, or amoxicillin/clavulanate 875 mg/125 mg every 8 hours. Worsening of the condition after 24 to 48 hours should be managed with an expedited evacuation.

Periocular insect envenomation is a preseptal cellulitis look-alike. Although secondary infection may follow envenomation, the envenomation itself may produce significant erythema and edema. Diagnostic clues include a history of insect bite or a periocular papular lesion at the site of the envenomation. Ice or cool compresses may be used to treat the envenomation, with moxifloxacin 400 mg once a day, levofloxacin 500 mg once a day, dicloxacillin 500 mg every 6 hours, or cephalexin 500 mg 4 times a day added if secondary infection is suspected.

The term *orbital cellulitis* means that the infection has spread to or originated in the tissues posterior to the orbital septum. This may be manifest as diplopia or restriction in ocular motility as the extraocular muscles are affected, proptosis as edema in the orbit pushes the globe forward, decreased vision as the optic nerve is affected, or pupillary change if innervation of the pupil is affected. Fever is suggestive of orbital cellulitis in the differential diagnosis of periocular inflammation. This condition is more commonly associated with sinusitis than with periocular trauma as an antecedent disorder.[76] Most series report a 50% to 75% incidence of sinusitis or other upper respiratory infection in association with orbital cellulitis.[137] The bacteria that most commonly cause orbital cellulitis are *S. aureus*, *Streptococcus pyogenes*, and *Streptococcus pneumoniae*.[91] Anaerobes are frequently present in cases of chronic sinusitis and should be suspected in orbital cellulitis associated with long-standing sinus disease.[91] If not treated aggressively, orbital cellulitis may be associated with life-threatening infection of the central nervous system. Before antibiotics became available, approximately 19% of persons with orbital cellulitis died of intracranial complications and 20% of survivors became blind in the involved eye.[91] This disorder requires hospitalization and intravenous antibiotic therapy. Interim therapy should include moxifloxacin 400 mg once a day. Alternative antibiotic choices are levofloxacin 500 mg twice a day, ciprofloxacin 750 mg twice a day, or amoxicillin/clavulanate 875 mg/125 mg every 8 hours. A decongestant should be added if sinusitis is present, and emergent evacuation should be undertaken.

Orbital pseudotumor is an inflammatory disease of the orbit that may present very much like orbital cellulitis. The differentiation between these two entities may be difficult.[76] There would typically not be a history of preceding sinusitis. A reasonable approach in the wilderness is to begin therapy with moxifloxacin 400 mg once a day and arrange for emergent evacuation. Prednisone (80 mg/day) should be added if there is no response to antibiotic therapy, there is no fever or sign of central nervous system involvement, and evacuation has not been possible by 24 to 48 hours after presentation. If prednisone therapy is initiated, its efficacy should be evaluated after 48 hours. If there has been a decrease in pain, erythema, edema, or proptosis, therapy should be continued until evacuation is accomplished. If there has been no response after 48 hours, the prednisone may be discontinued without tapering.

PERIOCULAR TRAUMA

Eyelid Laceration

The most important aspect of managing an eyelid laceration is carefully to exclude the presence of penetrating injury to the globe. Clues to the presence of an open globe are noted in the sections on Obvious Open Globe and Occult Ruptured Globe, later.

A lid laceration that is horizontally oriented on the eyelid, does not penetrate the full thickness of the lid, and does not involve the lid margin is relatively easily managed. In the absence of an ability properly to irrigate, disinfect, and suture the laceration, it should be managed by irrigation with the cleanest disinfected water available, application of topical antibiotic drops (ciprofloxacin, ofloxacin, or tobramycin) to the laceration, drying the surrounding skin surface, and then closing the laceration with tape strips. The wound may then be treated with antibiotic ointment (bacitracin or erythromycin) four times a day for 3 to 4 days. Alternatively, the laceration may be left open, treated with antibiotic ointment four times a day, and repaired 1 or 2 days later.[76] The laceration should be observed frequently while healing. If redness or discharge develops, the victim should be started on moxifloxacin 400 mg once a day, levofloxacin 500 mg once daily, or dicloxacillin 500 mg every 6 hours, the wound closure tape should be removed from the laceration, and evacuation should be expedited, especially if the response to oral antibiotics is poor.

Complicated lid lacerations, defined as stellate or complex, or involving the lid margin or the canthi (medial or lateral end of the palpebral fissure), are more difficult to manage. These lacerations may result in secondary functional difficulties if ocular lubrication or lacrimal drainage becomes impaired. In addition, cosmesis may be poor if a meticulous repair is not done. These wounds should be managed by irrigation with the cleanest disinfected water available, application of antibiotic ointment, and coverage with a sterile dressing. Evacuation for definitive repair should be expedited.

Corneal Frostbite

Corneal frostbite is an uncommon disease, but has been reported during such activities as snowmobiling without protective eye goggles when participants maintain their eyes open in conditions of high windchill factor.[5] Symptoms and treatment are the same as those described in the section on Corneal Abrasion, later.

Instillation of Adhesive Drops into the Eye

Inadvertent instillation of a "superglue" (cyanoacrylate-type adhesive) compound into the eye may bind the lids tightly together. Overnight application of a pressure patch with eye pads presoaked with water has been reported to make manual separation of the lids possible and eliminate the need for general anesthetic and surgical separation of the lids.[102] Ophthalmic ointment inserted through any small opening in the adherent lids has also been reported to facilitate resolution.[86] Once the lids are separated, the eye should be checked for a corneal abrasion.[116]

CHEMICAL INJURY TO THE EYE

The mainstay in the management of any chemical eye injury is immediate and copious irrigation of the ocular surface with water from whatever source is most readily available. In the wilderness, bottled water or intravenous solution is the best option. If neither of those is available, treated (filtered and disinfected) water from a drinking container is the next best option, with untreated water a last resort. Instillation of several drops of tetracaine will make the procedure much less uncomfortable for the victim. Irrigation should be continued for a minimum of 30 minutes.[76] The two most damaging chemicals are strong acids and alkalis.[76] Sulfuric acid from an exploding car battery is a typical acid, whereas cleaning products, such as drain cleaners, are typical alkalis. Caustic alkalis are more likely to damage the eye than are acids because of their profound and rapid ocular penetration. Do not attempt to neutralize the corneal surface with acidic or alkaline solutions. Chemicals other than acids and alkalis may be uncomfortable when they are encountered, but are less likely to produce significant long-term damage.

After a minimum of 30 minutes of flushing has been completed, the eye should be examined for retained particles. These should be removed with a moistened cotton-tipped applicator.[76] Treatment for an acid- or alkali-induced injury includes moxifloxacin 0.5% drops (1 drop four times a day) until fluorescein staining confirms that the corneal epithelial defect that typically accompanies these injuries has resolved. Topical prednisolone 1% should be added if there is significant inflammation. Prednisolone drops should be used every hour while awake for 3 days. Eye pain is managed with scopolamine drops four times a day for 3 days and oral pain medications.[114] Evacuation should be expedited if (1) the cornea is found to be opaque, (2) a large epithelial defect is found on fluorescein staining, or (3) significant pain persists after 3 days. Another sign of serious injury is blanching of the conjunctiva in the limbal area.[76]

A special type of chemical injury to the eye is ocular envenomation from a spitting cobra.[45,49,132] Ocular injury from cobra venom may be severe and result in blindness. Cobras can project this venom out to a range of 3 m (10 feet) and typically aim directly for the victim's eyes. Ocular surface envenomation causes severe conjunctival and corneal inflammation, edema, and erosions. Corneal erosions may progress to corneal ulceration, perforation, hypopyon, and endophthalmitis. Central corneal opacification may result despite aggressive therapy. One report described nine individuals who had suffered ocular surface cobra envenomations.[132] Five had only severe conjunctivitis, whereas corneal erosions developed in the other four. Two individuals were permanently blinded as a result. Therapy includes immediate flushing of the eyes as described previously. After this is done, check for a corneal abrasion and treat with moxifloxacin 0.5% drops if an erosion is present (1 drop four times a day until the erosion is healed). Note that healing may be prolonged in toxin-induced corneal surface injuries. Immediate consultation with a toxicology consult service and a corneal/external disease subspecialist should be obtained if possible and an expedited evacuation arranged. Topical steroid therapy should be considered (1 drop of prednisolone every hour while awake) and discussed with the consultant. The decision to treat is based on the severity of symptoms and the presence or absence of a corneal erosion or ulcer. The value of topical application of specific cobra antivenom has not been documented, so this therapy is not recommended. Systemic heparin therapy has been shown to be useful in preventing corneal opacification in animal models of this injury.[57,58]

Another type of chemical injury to the eye may occur when the eyes are sprayed with skunk musk. A PubMed search did not disclose any reports of human eye injuries from this type of event, but such an episode should be managed with immediate ocular irrigation as described previously. If discomfort persists beyond several hours, check for a corneal abrasion and treat with moxifloxacin 0.5% drops (1 drop four times a day until the abrasion is healed). If fluorescein staining does not show an abrasion but significant discomfort persists, a 3-day course of prednisolone 1% drops (1 drop four times a day for 3 days or until symptoms have resolved) may be of benefit. If pain is severe and unrelieved by prednisolone drops or persists after the 3-day course has been completed, consider an expedited evacuation and an ophthalmology consultation.

ACUTE LOSS OF VISION IN A WHITE, QUIET EYE

Vision may be variably decreased with many of the disease entities that are noted later in the section on Acute Red Eye. This section addresses sudden loss of vision that occurs in a white, quiet eye. A differential diagnosis is provided in Box 25-3. Disorders that cause this symptom are often difficult to diagnose without ophthalmic instruments (none of which is included in the kit shown in Box 25-1). There are few treatments for most of these disorders that are likely to be effective in a wilderness setting. Although an afferent pupillary defect (Marcus Gunn pupil) may be present, this is a nonspecific finding that would be expected with most of the disorders in Box 25-3, except for vitreous hemorrhage and high-altitude retinal hemorrhage.

An important question that must be asked in the face of acute loss of vision in a white, quiet eye is "Does this person have giant cell arteritis?" Giant cell arteritis (GCA), also called *temporal arteritis*, can cause devastating anterior ischemic optic neuropathy, often first noted on waking and that usually becomes permanent.[43] Subsequent involvement of the second eye is common if GCA in the first-stricken eye is not promptly treated.[43] Although visual loss has been reported to occur in both eyes simultaneously, there is typically a delay of 1 to 14 days before the second eye is affected.[1] Loss of vision in the

Box 25-3. Differential Diagnosis of Acute Loss of Vision in a White, Quiet Eye

Retinal detachment
Central retinal artery occlusion
Anterior ischemic optic neuropathy
Optic neuritis
Central retinal vein occlusion
Arteritic anterior ischemic optic neuropathy
Vitreous hemorrhage
High-altitude retinal hemorrhage

second eye can be prevented in most cases by prompt initiation of high-dose corticosteroid therapy.[1,43,52,97]

Arteritic anterior ischemic optic neuropathy is typically a disease of older individuals, with one large study reporting a mean age of 70 years and the age of the youngest patient as 53 years.[1] Clues to diagnosis are temporal headache, jaw claudication, fever, weight loss, transient visual obscurations, and polymyalgia rheumatica (generalized myalgias).[97] The visual obscurations seen in GCA usually last 2 to 3 minutes.[97] If a person is thought to be suffering from GCA, he or she should be started on prednisone 80 mg once a day and evacuation expedited.

If one suspects a retinal detachment based on a history of high myopia (extreme nearsightedness), floaters, or photopsias (flashing lights), expedited evacuation should be undertaken because of the need for surgical repair. Loss of central vision caused by a retinal detachment usually means that the macula is involved and that surgical repair is urgent rather than emergent. Ross and Kozy[109] found that a delay to surgery of up to 1 week in macula-off rhegmatogenous retinal detachments did not affect final visual acuity. Expedited evacuation to a facility that has retinal surgery capability allows for more precise determination of the urgency for surgical repair.

If the victim is at altitude (above approximately 3048 m [10,000 feet]), high-altitude retinal hemorrhage (discussed later) should be suspected and further ascent avoided.[17] Descent of at least 915 m (3000 feet) should be undertaken as soon as feasible.[17]

Another potentially treatable cause of sudden loss of vision in a white, quiet eye is central retinal artery occlusion (CRAO). Previous conventional therapy for CRAO of ocular massage, pentoxifylline, and anterior chamber paracentesis has been reported to be unsuccessful in restoring vision in 40 of 41 patients with CRAO, even though 11 patients presented within 6 hours of visual loss and 17 presented within 12 hours.[110] Primate retinas can tolerate no more than 100 minutes of ischemia caused by a complete blockage of retinal blood flow.[51] However, fluorescein angiography has shown that in humans, CRAO is seldom complete, and that therapy begun up to 6 hours after visual loss may be successful in restoring vision.[110]

Hyperbaric oxygen was reported successful in restoring vision on two separate occasions in one patient with recurrent branch retinal artery occlusions associated with Susac's syndrome.[74] Oxygen is supplied to the retina from both the retinal and choroidal circulations. Under normoxic conditions, approximately 60% of the retina's oxygen is supplied by the choroidal circulation. Under hyperoxic conditions, the choroid is capable of supplying 100% of the oxygen needed by the retina.[74] When retinal arterial flow is interrupted, the retinal tissue undergoes a period of ischemia. Blood flow may be spontaneously reestablished, as frequently happens with arterial obstruction, or ischemia may continue until cell death and necrosis occur.[80] The period of time during which the tissue is ischemic, yet capable of recovery, is called the *ischemic penumbra*.[80] Hyperbaric oxygen is not always required for a reversal of retinal ischemia. The author has treated a monocular patient who suffered a central retinal artery occlusion in his only seeing eye and presented to the emergency department within an hour of visual loss. The victim's vision improved from 20/400 to 20/25 within minutes on supplemental oxygen by mask. Unfortunately, however, his vision decreased rapidly to 20/400 whenever the supplemental oxygen was removed. The victim was heparinized and maintained on supplemental oxygen for approximately 10 hours, at which time the removal of oxygen no longer caused a decrease in vision. If oxygen is being carried for an extreme altitude summit attempt or for other purposes, a person with sudden painless loss of vision should be given a trial of oxygen administered in as high a concentration as possible to see if this therapy results in visual improvement.

Care should be taken when monocular visual loss occurs. Although central vision may still be normal in the fellow eye, depth perception may be impaired and the victim may therefore be at increased risk of a fall during evacuation.

Box 25-4. Differential Diagnosis of the Acute Red Eye

Obvious open globe
Corneal abrasion
Corneal ulcer
Subconjunctival hemorrhage
Traumatic iritis
Hyphema
Occult open globe
Herpes simplex virus keratitis
Corneal erosion
Acute angle-closure glaucoma
Iritis
Scleritis
Conjunctivitis
Blepharitis
Ultraviolet keratitis
Episcleritis
Conjunctival foreign body
Dry eye
Contact lens overwear syndrome

ACUTE RED EYE

Box 25-4 provides a partial list of disorders that can result in an acute red eye. In the absence of a slit lamp, the diagnosis must rely on the basic techniques of history, penlight inspection, fluorescein staining, response to administration of topical anesthesia, and pupillary status. The discussion of the differential diagnosis of the acute red eye that follows uses these clinical findings to establish the diagnosis. An algorithmic representation is shown in Figure 25-1. The term *fluorescein positive* is used to denote an eye with a discrete area of staining noted with cobalt blue light after instillation of fluorescein dye. Some conditions, such as blepharitis, viral keratoconjunctivitis, and ultraviolet (UV) keratitis, may cause a pattern of punctate staining referred to as *superficial punctate keratitis* (SPK).

Traumatic Ocular Disorders

Obvious Open Globe

If there is a history of trauma and penlight inspection of the eye reveals an obvious open globe (such as the eye in Fig. 25-2), the examination should be discontinued and a protective shield placed over the eye. Do not apply a pressure patch or instill any topical medication. There are two primary concerns in the management of this condition. The first is to minimize manipula-

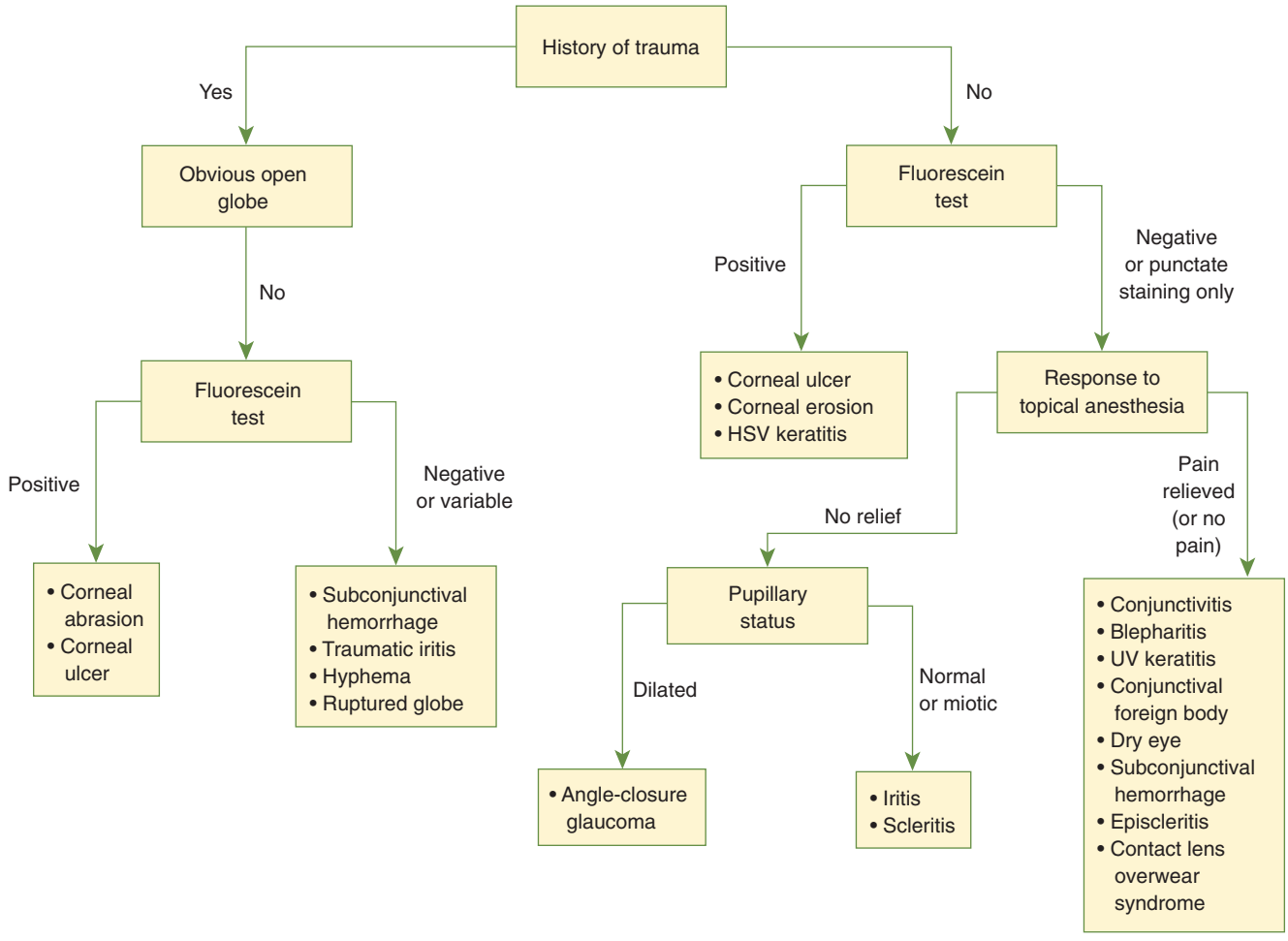


Figure 25-1. Algorithm showing wilderness diagnostic procedure for the acute red eye.

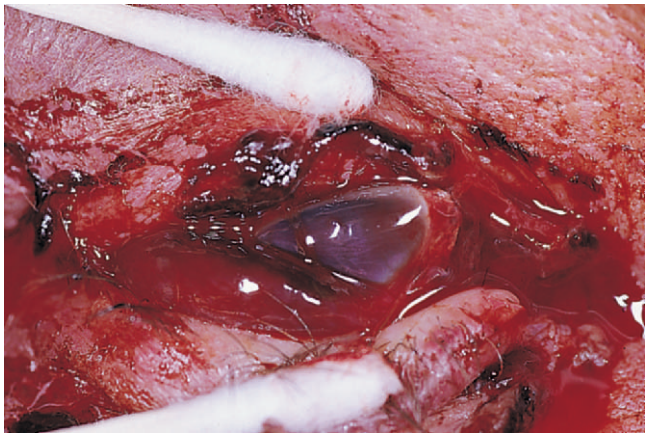


Figure 25-2. Obvious open globe (corneoscleral laceration). (Courtesy Steve Chalfin, MD, University of Texas Health Science Center, San Antonio, TX.)

tion or additional trauma to the eye that might raise intraocular pressure and result in expulsion of intraocular contents through the corneal or scleral defect. The second is to prevent development of post-traumatic endophthalmitis, an infection of the aqueous and vitreous humors of the eye. This typically has devastating visual results, with only 30% of victims in one study retaining visual acuity greater than or equal to 20/400.[69] *Staphylococcus epidermidis* is the most common pathogen implicated, but *Bacillus cereus* is a very aggressive pathogen often isolated in this condition.[69] After the shield is placed, the victim should be started on moxifloxacin 400 mg once a day or levofloxacin 500 mg once a day. Trovafloxacin 200 mg once a day is another alternative choice for prophylaxis because of its very broad antibacterial spectrum[54,130] and good ocular penetration.[89] Ciprofloxacin 750 mg twice a day is a fourth option. A person with an obvious open globe needs surgical repair as soon as possible and should be evacuated emergently. Because there is a possibility that air may have been introduced into the eye, barometric pressure changes during evacuation should be

Figure 25-3. Occult open globe with uveal pigment at the limbus and a peaked pupil. (Courtesy Steve Chalfin, MD, University of Texas Health Science Center, San Antonio, TX.)

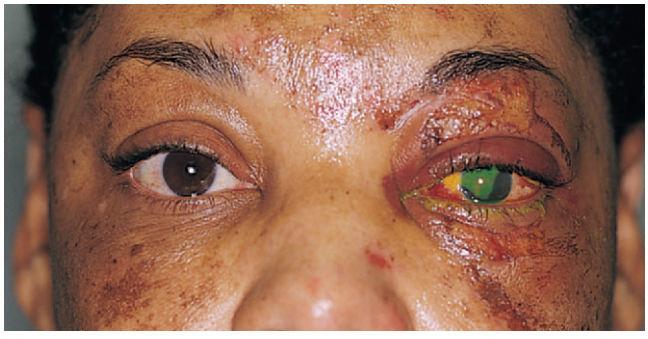


Figure 25-4. Corneal abrasion. (Courtesy Steve Chalfin, MD, University of Texas Health Science Center, San Antonio, TX.)

minimized, if possible. However, this consideration is secondary to the need for expeditious transport to a facility where surgical repair can be performed.

Occult Ruptured Globe

A penetrating injury to the eye or a ruptured globe may not always be obvious. Clues to occult rupture include large subconjunctival hemorrhage with chemosis, dark uveal tissue present at the limbus, distorted pupil (Fig. 25-3), fluorescein leak from a linear or punctate corneal epithelial defect, mechanism of injury (e.g., hammering metal on metal, impaling injury), or decrease in vision. If an occult globe rupture is suspected, the victim should be treated as described previously for an obvious open globe. The relatively less severe appearance of the injury does not eliminate the threat of endophthalmitis, so systemic antibiotic therapy as noted previously should be initiated.

Corneal Abrasion

A corneal abrasion is disruption of the protective epithelial covering of the cornea (Fig. 25-4). This results in intense pain, tearing, light sensitivity (photophobia), and increased susceptibility to infection until the defect has healed (usually in 2 to 3 days). There is typically a history of antecedent trauma or contact lens wear. The sine qua non for this diagnosis is an epithelial defect on fluorescein staining. Standard treatment in the recent past consisted of antibiotic ointment followed by application of a pressure patch. A recent study, however, has shown that small ($<10\,mm^2$), noninfected, and non-contact lens–related abrasions healed significantly faster with less discomfort when they were not patched.[62] In a wilderness setting, the nonpatching option has the additional advantage of not rendering the victim completely monocular and adversely affecting visual field and depth perception. If the nonpatching option is chosen, the victim should be treated with moxifloxacin 0.5% drops (1 drop four times a day) or bacitracin ointment four times a day until the corneal epithelium is healed. Diclofenac drops four times a day should be helpful in reducing discomfort. Sunglasses help alleviate photophobia. Repeated use of a topical anesthetic for pain control is contraindicated. If the abrasion is contact lens related, the eye should not be patched because of the increased risk of corneal ulcer present with a contact lens–related abrasion[104]; contact lens–related corneal abrasions should be treated with moxifloxacin 0.5% drops (1 drop every 2 hours while awake) until the epithelial defect has resolved. An abrasion associated with vegetable matter should also not be patched.[104] If the abrasion is large or the victim's discomfort severe, scopolamine drops once or twice a day may be added to the antibiotic (wait 5 minutes between each drop). Much of the pain associated with corneal abrasion and ulcer is due to ciliary muscle spasm, which is relieved by scopolamine. The rationale for using scopolamine only with a very painful abrasion in the wilderness is that this medication will cause the pupil to dilate (and the eye to become very sensitive to light) and accommodation to relax (with a resultant decrease in near visual acuity) for approximately 5 to 7 days.

An oral analgesic may be required for pain control. The victim should be monitored daily for development of a corneal ulcer (noted on penlight examination as a white or gray infiltrate on the cornea) and for progress in healing of the epithelium (as measured by resolution of fluorescein staining).

Corneal Ulcer

The term *corneal ulcer*, as used here, denotes acute bacterial, fungal, or protozoal infection of the cornea. Chronic corneal epithelial defects caused by a variety of autoimmune or inflammatory processes are also referred to as corneal ulcers at times, but these disorders are beyond the scope of this chapter. Although a corneal ulcer (Fig. 25-5) is an infectious process, it is often preceded by a traumatic corneal abrasion. The other predisposing condition for a corneal ulcer is contact lens wear, which results in microtrauma to the corneal epithelium and may allow bacteria or other microorganisms to infect the cornea. Corneal ulcers are typically significantly painful. A small white or gray infiltrate on the cornea can be appreciated by a careful penlight examination. If not treated aggressively, the small initial lesion may progress to the much larger infiltrate shown in Figure 25-5, with a correspondingly more severe impact on visual acuity. Fluorescein staining reveals an epithelial defect overlying the infiltrate. The associated pain is usually significantly decreased by application of a topical anesthetic, but ciliary spasm may cause pain relief to be incomplete.

An inadequately treated corneal ulcer may result in visual loss from dense corneal scarring or ocular perforation with subse-

Figure 25-5. Corneal ulcer. (Courtesy Steve Chalfin, MD, University of Texas Health Science Center, San Antonio, TX.)

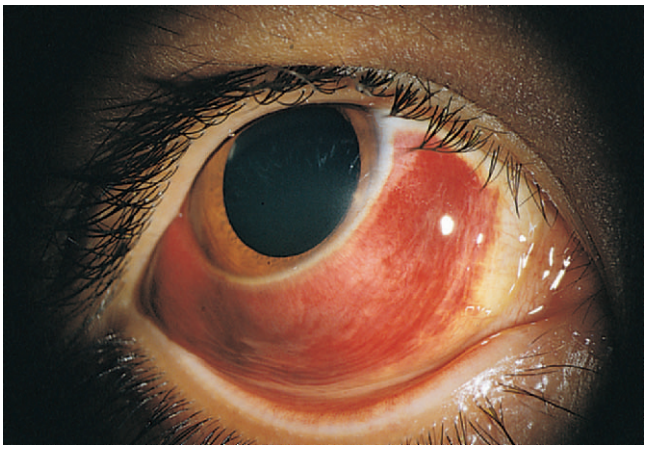


Figure 25-6. Subconjunctival hemorrhage. (Courtesy Steve Chalfin, MD, University of Texas Health Science Center, San Antonio, TX.)

quent endophthalmitis. Management of this disorder in the past included hospital admission for treatment with concentrated and frequently administered aminoglycoside and cephalosporin topical drops. More recently, outpatient therapy with fluoroquinolone eye drops has been shown to be comparable in efficacy to fortified antibiotic preparations.[56,93] Treatment for corneal ulcer, then, should be with moxifloxacin 0.5% drops, 1 drop every 5 minutes for five doses initially, then 1 drop every 30 minutes for 6 hours, then 1 drop every hour thereafter around the clock.[104] Scopolamine 1 drop two to four times a day may help relieve discomfort caused by ciliary spasm. Repeated use of topical anesthetics for pain control is contraindicated. Systemic analgesia may be required if pain is severe. Expedited evacuation is recommended.

Traumatic Iritis

Iritis refers to inflammation of the iris or, more accurately, the anterior uveal tract of the eye. It may also be called anterior uveitis or iridocyclitis. The sine qua non of this condition is inflammatory cells in the anterior chamber of the eye, which must be visualized with a slit lamp. In the wilderness, this diagnosis must be presumptive. Iritis may accompany a corneal abrasion or result from blunt trauma. The diagnosis rests primarily on the presence of significant post-traumatic pain without a corneal abrasion or ulcer noted on fluorescein staining, or on pain that persists after the abrasion is healed. Treatment is with prednisolone 1 drop four times a day for 3 days. Scopolamine 1 drop twice a day may be added if pain is severe enough to justify the blurred vision that scopolamine therapy will entail.

Subconjunctival Hemorrhage

Subconjunctival hemorrhage (Fig. 25-6) is a bright red area over the sclera of the eye that results from bleeding between the conjunctiva and the sclera. It is easily visible without the use of a slit lamp. This injury is innocuous and resolves over a period of several days to several weeks without treatment. In the presence of antecedent trauma, one should be alert for another, more serious injury. In particular, if hemorrhage results in massive swelling of the conjunctiva (chemosis), an occult globe rupture should be suspected.

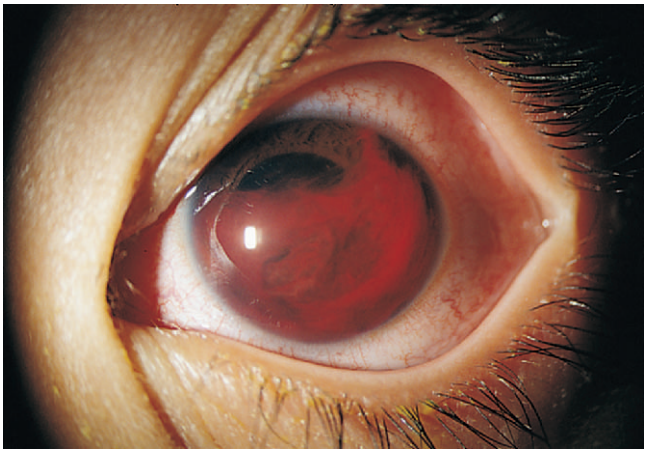


Figure 25-7. Hyphema. (Courtesy Steve Chalfin, MD, University of Texas Health Science Center, San Antonio, TX.)

Hyphema

The term *hyphema* is defined as blood in the anterior chamber. Although this is usually seen in the setting of acute trauma, it may also be caused by other conditions, such as iris neovascularization. The eye should be examined with the victim sitting upright. If enough blood is present, it collects at the bottom of the anterior chamber and is visible as a layered hyphema (Fig. 25-7). This may not be appreciated if the victim is examined while in a supine position or if the amount of blood is very small.

Although most hyphemas resolve without sequelae, this disorder may be complicated by an acute rise in intraocular pressure or corneal blood staining. Treatment in the wilderness consists of activity restriction (walking only), prednisolone drops four times a day, avoidance of aspirin or nonsteroidal anti-inflammatory drugs (NSAIDs), and use of an eye shield until the hyphema has resolved. Diamox 250 mg four times a day by mouth should be added if available to treat the poten-

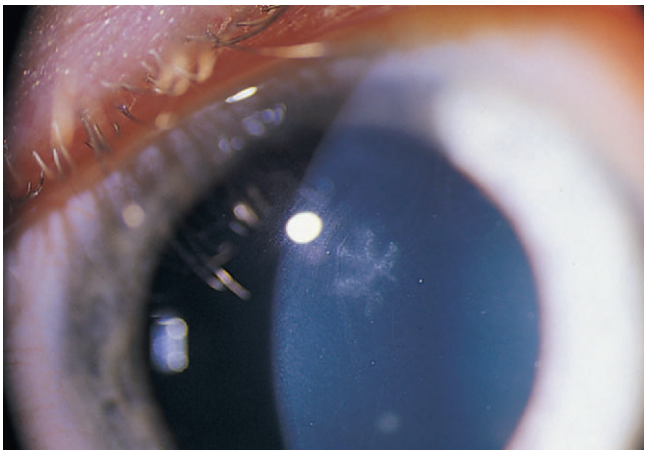


Figure 25-8. Herpes simplex virus keratitis. (Courtesy Steve Chalfin, MD, University of Texas Health Science Center, San Antonio, TX.)

tially increased intraocular pressure. Retinal injury or an occult ruptured globe may accompany traumatic hyphema. A hyphema victim requires ophthalmologic evaluation, so expedited evacuation should be undertaken.

Nontraumatic Fluorescein-Positive Acute Red Eye

Herpes Simplex Virus Keratitis

The essential element in this diagnosis is the characteristic dendritic epithelial pattern on fluorescein staining (Fig. 25-8). There will often be a history of previous episodes of herpes simplex virus (HSV) keratitis. Treatment is trifluorothymidine 1% drops nine times a day.[104] Treatment is continued until the corneal staining has resolved, at which time the frequency of dosing is reduced to four times a day for 1 week. Trifluorothymidine was not included in the list of eye medications to be taken on the expedition because trifluorothymidine drops require refrigeration. If a significant delay is anticipated before evacuation, HSV keratitis may be treated by using tetracaine 3 to 5 drops given 1 minute apart to anesthetize the cornea and then performing a gentle cotton-tipped applicator debridement of the epithelial lesion.[104] The resulting epithelial defect should then be treated as described in the Corneal Abrasion section.

Corneal Erosion

A corneal erosion is an epithelial defect caused by nontraumatic disruption of the corneal epithelium. The fluorescein staining pattern seen with corneal erosion may be identical to that seen with corneal abrasion; pain and photophobia are present with both disorders. The diagnosis is made when the apparent corneal abrasion has no history of trauma to explain its presence. There is often a history of previous similar episodes. The two primary causes for corneal erosion are corneal dystrophies and previous ocular trauma.[97] Recurrent corneal erosions are believed to be caused by a defect in healing between the hemidesmosomes of the corneal epithelium and the underlying basement membrane.[76] At night, the epithelium may become adherent to the closed eyelid during sleep. When the individual awakens and opens his or her eyes, the corneal epithelium is pulled away from the basement membrane by movement of the lid. This accounts for the typical history of acute onset of pain on awakening. Signs and symptoms include pain, tearing, and foreign body sensation.[76]

Treatment of these lesions may be difficult. The cornea should be inspected for a loose sheet of epithelium that remains partially attached to the corneal surface. If this is present, try to debride it with a cotton-tipped applicator after topical anesthesia with tetracaine. The lesion is then managed initially in the same manner as a corneal abrasion. There is a high rate of recurrence if follow-up treatment with 5% sodium chloride ointment each evening or anterior stromal puncture is not undertaken.

Corneal Abrasion and Corneal Ulcer in Contact Lens Wear

Both lesions may occur as complications of contact lens wear. Management has been described earlier. Contact lens–related corneal abrasions have a relatively high incidence of progressing to corneal ulcers.[62] Therefore they should not be patched and should receive topical antibiotic therapy with moxifloxacin 0.5% drops every 2 hours while awake until the epithelial defect has resolved. Contact lens wear in both eyes should be discontinued immediately. If an ulcer is related to contaminated lens solutions, infection in the first eye may be followed rapidly by a similar occurrence in the other eye.

Nontraumatic Fluorescein-Negative Acute Red Eye (Pain Not Significantly Improved by Topical Anesthesia)

Acute Angle-Closure Glaucoma

This diagnosis is easily made when the intraocular pressure can be measured with a tonometer. Intraocular pressure is normally between 10 and 21 mm Hg. With acute angle-closure glaucoma, the pressure may increase to 50 or 60 mm Hg or higher. Although handheld tonometers are available, they will not often be present outside of hospitals or clinics. The most important clues to the diagnosis of angle-closure glaucoma in the wilderness setting are the characteristics of the pain and the status of the pupil. Angle-closure glaucoma victims do not have the mild burning or foreign body–type pain typically seen with conjunctivitis, blepharitis, or other external eye diseases. Angle-closure glaucoma produces pain that is usually deep and severe. Although corneal abrasions and corneal ulcers may also be accompanied by severe pain, the pain in these two disorders is usually significantly relieved by topical anesthetics. This is not true of the pain seen with acute glaucoma, scleritis, or iritis. In these disorders, the pain is rarely if ever significantly relieved by topical anesthetics. In acute angle-closure glaucoma, the pupil is usually found to be mid-dilated (6 to 7 mm) and vision is usually decreased. This disorder generally affects persons older than 50 years of age[76] and is more common in persons of Asian ethnic origins. There is often a history of previous transient episodes of eye pain. Nausea and vomiting may be present. An eye with acute angle-closure glaucoma is shown in Figure 25-9. Some wilderness expeditions now carry portable instruments such as a Tono-Pen (Medtronic, Minneapolis) to measure intraocular pressure. Instructions for using this device are included in Box 25-5.

The usual treatment is topical antiocular hypertensive medications and laser iridotomy, which allows the aqueous humor to bypass the pupillary block. This relieves the angle closure with a resultant decrease in intraocular pressure.[76] In the wilderness setting, treatment should be with 2% pilocarpine one drop every 15 minutes for four doses,[97] then four times a day in both

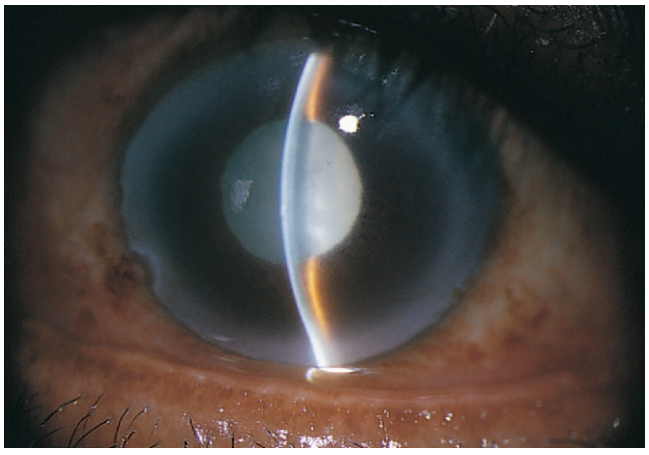


Figure 25-9. Angle-closure glaucoma. (Courtesy Steve Chalfin, MD, University of Texas Health Science Center, San Antonio, TX.)

eyes because there is a high rate of subsequent occurrence of angle-closure glaucoma in the fellow eye. Pilocarpine alone may be successful in relieving the angle closure and lowering the pressure, but this is not a reliably effective treatment because ischemia of the pupillary sphincter muscle may prevent pilocarpine from exerting its miotic effect. Diamox 250 mg four times a day by mouth should be added if available. Evacuation should be emergent if pain is not relieved because even 1 day of very high intraocular pressure may result in permanent damage to the optic nerve and loss of vision.

Nontraumatic Iritis

Signs and symptoms of nontraumatic iritis that may be appreciated without a slit lamp include pain, redness, photophobia, limbal flush, and decreased vision.[97] There is often a history of previous episodes. An eye with nontraumatic iritis is shown in Figure 25-10 (the pupil is iatrogenically dilated). Iritis not associated with corneal trauma is typically more severe than the traumatic variety but may range from mild to very severe and typically is not significantly relieved by topical anesthesia. Fluorescein staining is negative and the pupil is usually miotic, thus helping differentiate iritis from angle-closure glaucoma. Iritis is far more common than angle-closure glaucoma and may be associated with a number of systemic infectious and inflammatory diseases. In the wilderness setting, the emphasis should be on immediate treatment with prednisolone 1% 1 drop every hour while awake. Scopolamine 1 drop one to four times a day may be added for pain control and to prevent posterior synechiae (pupillary scarring) if inflammation is severe. If pain is not significantly decreased in 24 to 48 hours, oral prednisone 80 mg/day should be added to the treatment regimen and maintained until evacuation. Evacuation should be expedited because posterior synechiae and elevated intraocular pressure may develop.

Scleritis

It may be difficult to differentiate between nontraumatic iritis and scleritis without a slit lamp, but scleritis is much less common than iritis. The characteristics of the pain are similar, with pain, photophobia, scleral injection, and tearing.[97] Scleritis is often associated with rheumatologic disease and may be either nodular or diffuse.[97] The diffuse form of scleritis is shown in Figure 25-11. The initial treatment of scleritis in the wilderness setting is topical prednisolone 1 drop every hour while awake and an NSAID, if available. Prednisone 80 mg/day should be added if there is no improvement in 24 to 48 hours and maintained until an expedited evacuation is accomplished.

Box 25-5. Tono-Pen Instructions

Steps in setting up Tono-Pen:

1. Remove the Ocu-Film tip cover from the probe.
2. To help prevent buildup of debris around the probe post, spray the probe tip with compressed gas before the first use of the day.
3. Cover the Tono-Pen XL probe tip with a new Ocu-Film tip cover.
4. Check calibration only before the first use of each day.
   a. Depress the activation switch momentarily, then release.
   b. If the previous calibration check was good, the LCD will briefly display "----" followed by " = = = = ," accompanied by a beep.
   c. If the previous calibration was bad, a long beep sounds, after which "CAL" appears and a short beep sounds. The display then changes to "----" and another short beep sounds.
5. Hold the Tono-Pen vertically with the probe tip pointing straight down.
6. Press and release the activation switch twice in rapid succession. Two beeps will sound and "CAL" appears.
7. Wait (up to 20 seconds) until a beep sounds and "-up-" appears.
8. Quickly turn the probe straight up.
9. Wait a few seconds. A second beep will sound, indicating the end of the calibration check.
10. Instill a drop of topical anesthetic (proparacaine) into both eyes.
11. Instruct the patient to look straight ahead at the fixation target with his or her eyes fully open.
12. Brace the heel of your hand on the patient's cheek for stability
13. Activate the Tono-Pen by pressing the activation switch.
14. The LCD will display " = = = = " and a beep will sound.
15. Once activated, touch the Tono-Pen probe against the patient's cornea lightly and briefly. Repeat several times.
16. A click will sound and a digital intraocular pressure measurement is displayed.
17. Proceed to the other eye. Repeat steps 12 through 15.

Nontraumatic Fluorescein-Negative Acute Red Eye (No Discomfort or Discomfort Improved by Topical Anesthesia)

Conjunctivitis

One of the most common disorders in this diagnostic category is conjunctivitis. Etiologic agents of infectious conjunctivitis

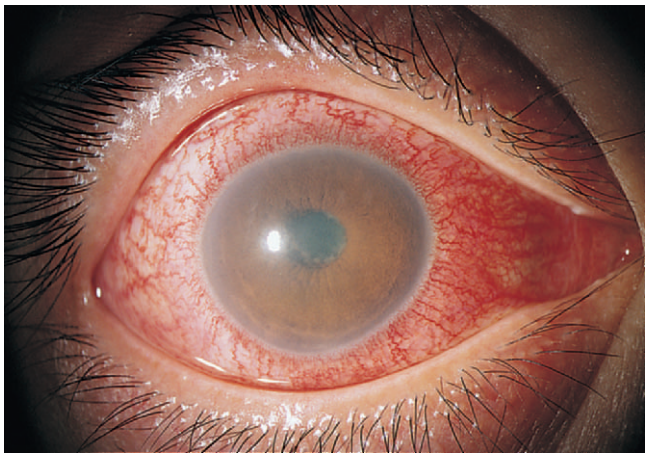


Figure 25-10. Nontraumatic iritis. (Courtesy Steve Chalfin, MD, University of Texas Health Science Center, San Antonio, TX.)

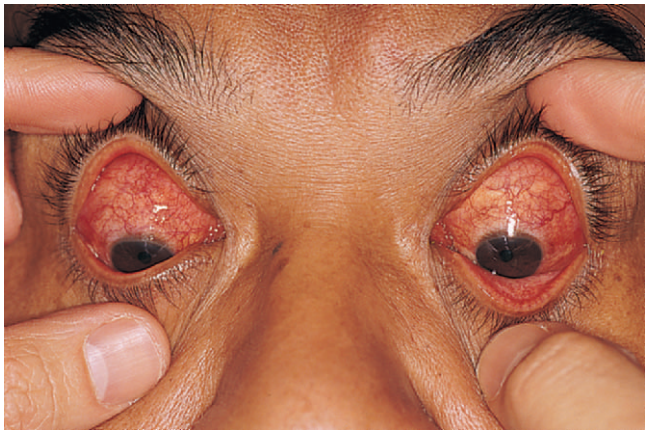


Figure 25-11. Diffuse scleritis. (Courtesy Steve Chalfin, MD, University of Texas Health Science Center, San Antonio, TX.)

include bacteria, viruses, chlamydiae, fungi, and parasites.[76] Acute allergic conjunctivitis may also be encountered, especially in a wilderness setting. The keys to diagnosis are the presence of discharge or tearing, relatively mild burning or foreign body–type discomfort that is relieved by topical anesthesia, and a negative (or SPK) fluorescein staining pattern. The diagnosis of conjunctivitis should be questioned in the absence of discharge or tearing. The primary exception to this statement is allergic conjunctivitis, which may not generate a significant discharge. In this case, bilaterality, significant ocular itching, and a history of ocular or systemic allergies help make the diagnosis. With infectious conjunctivitis, there are often signs or symptoms of an accompanying upper respiratory infection (URI) or a history of contact with other persons who have recently suffered from conjunctivitis.

Treatment of conjunctivitis in the wilderness setting is with gatifloxacin 0.3% drops or moxifloxacin 0.5% drops (1 drop four times a day for 5 days) if there is a yellowish discharge. Ciprofloxacin 0.3% drops, ofloxacin 0.3% drops, tobramycin 0.3% drops, or trimethoprim/polymyxin B drops are acceptable

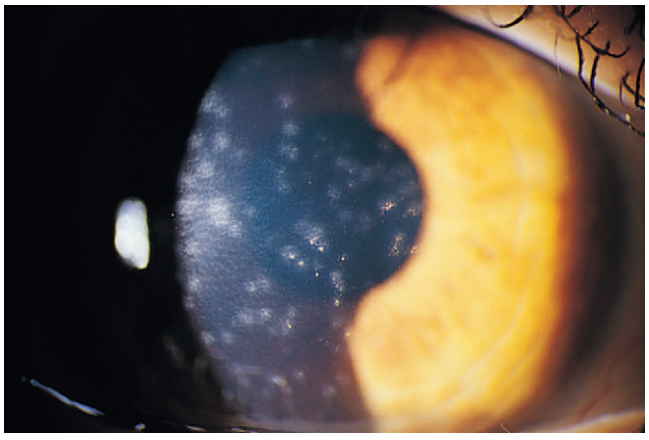


Figure 25-12. Epidemic keratoconjunctivitis (EKC). (Courtesy Steve Chalfin, MD, University of Texas Health Science Center, San Antonio, TX.)

alternatives.[76] This should be an adequate treatment time if conjunctivitis is bacterial. Symptoms that persist for more than 5 days suggest a viral etiology. These symptoms may take several weeks to clear, just as some viral URIs take several weeks to resolve. If there is only tearing or a watery discharge, suggesting a viral etiology, treatment with artificial tears and cool compresses may be substituted for antibiotic therapy. The victim should be instructed on infection precautions because viral conjunctivitis can spread rapidly through a group. This is especially true if the discomfort caused by conjunctivitis is severe and accompanied by significant photophobia. These symptoms suggest epidemic keratoconjunctivitis (EKC), which, in addition to having a prolonged course, is very uncomfortable because of the corneal involvement. The cornea of an eye with EKC is shown in Figure 25-12. Sunglasses should be part of the treatment. Scopolamine 1 drop two to four times a day may be required to reduce discomfort.

As noted previously, if the predominant ocular symptom is itching, allergic conjunctivitis should be suspected. Allergic conjunctivitis may be treated with cool compresses or systemic antihistamines. Severe ocular itching may be treated with a 3-day course of prednisolone, 1 drop four times a day.

Hyperacute conjunctivitis with marked lid edema, conjunctival hyperemia, chemosis, and copious purulent discharge should alert the care provider to the possibility of a gonococcal etiology.[76] Gonococcal conjunctivitis may progress to vision-threatening keratitis and should be treated aggressively with moxifloxacin 400 mg orally once a day, moxifloxacin drops four times a day, and expedited evacuation. If the conjunctivitis is chronic and associated with a preauricular node and conjunctival follicles, the individual will require additional therapy with doxycycline 100 mg twice a day for 14 days.[72]

Bacterial and viral conjunctivitis are highly contagious. Individuals with infectious conjunctivitis should be informed of this fact and the importance of infection precautions emphasized.

Blepharitis

Blepharitis is probably the most common external eye disease seen in the general ophthalmologist's office.[32] It is often mis-

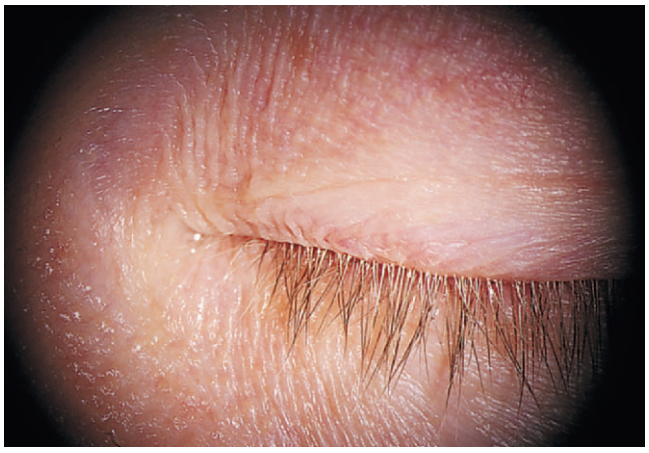


Figure 25-13. Blepharitis. (Courtesy Steve Chalfin, MD, University of Texas Health Science Center, San Antonio, TX.)

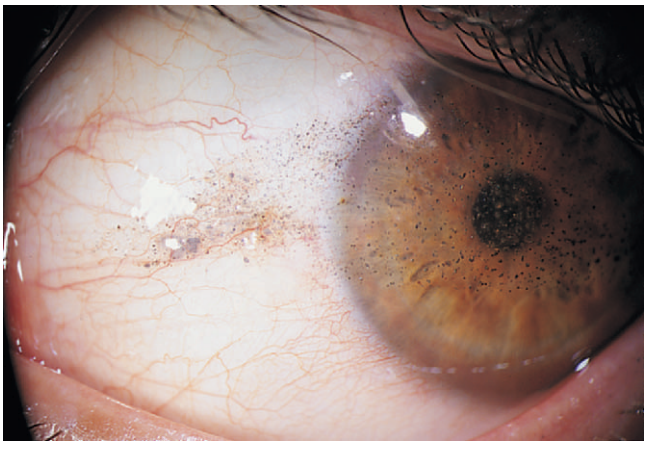


Figure 25-14. Conjunctival foreign body. (Courtesy Steve Chalfin, MD, University of Texas Health Science Center, San Antonio, TX.)

diagnosed as conjunctivitis. Differentiation may often be made by the history. Blepharitis tends to be a bilateral, chronic disease with recurrences and exacerbations. There is chronic flaking and irritation of the skin at the base of the eyelashes that may occasionally be complicated by bacterial superinfection. An eye with blepharitis is shown in Figure 25-13. There may be an association with skin disorders, such as seborrheic dermatitis or acne rosacea.[32] The victim often has a history of chronic ocular itching and burning. Fluctuating vision may be present as well.[32] There is often a history of excessive mucus discharge in the eyes on waking.

Treatment for blepharitis should focus on the lid margins. Bacitracin or erythromycin ophthalmic ointment should be applied thinly to the lid margins at bedtime for 4 weeks. Lid hygiene is directed at reducing the amount of debris on the lid margins and consists of warm, moist compresses applied to the eyelids one to four times a day for 5 to 10 minutes, followed by gently wiping away the moistened lash debris. Artificial tears may relieve the sensation of "dry eye" that often accompanies blepharitis.[32]

Ultraviolet Keratitis

The diagnosis of UV keratitis is easy to make in the presence of a severely sunburned face and bilateral red, painful eyes. The discomfort typically does not start until 6 to 10 hours after UV exposure and may awaken the victim.[97] Symptoms range from mild irritation and foreign body sensation to severe pain, photophobia, and lid spasm.[97] Fluorescein staining usually reveals a punctate staining pattern.

Treatment consists of moxifloxacin drops four times a day. Bacitracin or erythromycin ophthalmic ointment applied to the eye four times a day is an acceptable alternative. Sunglasses help with the severe light sensitivity that accompanies this disorder. In a severe case, one or both eyes may be patched to aid in pain control, although it may be better to patch only the more severely affected eye so that the victim is not deprived of vision in both eyes.[17] Scopolamine (1 drop twice a day) may be added for pain control. Systemic analgesia may also be required. The victim should be reexamined every day until there is no longer an SPK pattern present on fluorescein staining, at which time antibiotic therapy may be discontinued. The duration of discomfort from UV keratitis is typically 24 to 48 hours.[97]

Conjunctival Foreign Body

The symptom of ocular foreign body sensation does not necessarily mean that a conjunctival or corneal foreign body is present. Although the presence of a conjunctival foreign body may be strongly suspected based on the abrupt onset of discomfort after a gust of wind or other mechanism for depositing foreign material in the eye, definitive diagnosis requires visualization of the offending material, which may sometimes be quite difficult. Figure 25-14 shows an eye with a conjunctival foreign body. The victim is often able to help with foreign body localization before the instillation of topical anesthetic drops.

Treatment consists of a careful search for the foreign body using adequate lighting. Topical anesthesia makes the victim much more agreeable during the search and removal efforts. A handheld magnifying lens or pair of reading glasses provides magnification to aid in the visualization of the foreign body. Eyelid eversion with a cotton-tipped applicator helps the examiner identify foreign bodies located on the upper tarsal plate. Once located, the foreign body should be removed with a cotton-tipped applicator after the eye has been anesthetized and the cotton-tipped applicator moistened with tetracaine. The eye is then stained with fluorescein to check for a corneal abrasion. If no foreign body is visualized but the index of suspicion is high, vigorous irrigation with artificial tears or sweeps of the conjunctival fornices with a moistened cotton-tipped applicator after topical anesthesia may be successful in removing the foreign body.

Several types of foreign body merit special mention. If the foreign body identified is one that may have penetrated into the eye, such as a large thorn, the victim should be managed as described in the Obvious Open Globe section. Hot ashes or cinders from a campfire may strike the eye, resulting in both a foreign body and a thermal burn. Immediate instillation of topical anesthesia provides temporary pain relief to facilitate foreign body removal and also stops any ongoing thermal injury. After the foreign body has been removed, the corneal abrasion that typically results from a thermal injury to the

cornea should be managed as outlined in the Corneal Abrasion section.

Dry Eye

Symptomatic dry eye is commonly encountered in the wilderness, especially in mountainous areas where the air is very dry and significant wind is often present.[17] Dry eye is usually bilateral and may result in secondary tearing.[104] There may be a history of previous episodes of symptomatic dry eye. Individuals with chronic dry eyes are usually middle-aged or older and may have a history of autoimmune disorders.

Treatment is with artificial tears used as often as needed to relieve symptoms. Dehydration may contribute to this condition, and adequate fluid intake should be maintained. The use of sunglasses may provide protection from the wind and be of significant benefit in managing this disorder. High-viscosity drops such as Celluvisc and lubricating ointments used at bedtime may also help to relieve symptoms.[78] In more severe cases, reversible punctal occlusion may be considered as well.[78]

Contact Lens Overwear Syndrome

The considerations here are much as described in the Dry Eye section, except that the symptoms are magnified by the presence of contact lenses.

Contact lens rewetting drops and sunglasses are the first line of management. Should these measures be ineffective in relieving symptoms, the contact lenses should be removed. If significant SPK is present on fluorescein staining, moxifloxacin drops (1 drop four times a day) should be used until the SPK have resolved.[17] Contact lenses should not be replaced in the eye until the eye is symptom free. An individual who wears contact lenses in the wilderness should always carry a pair of glasses that can be used if contact lens problems arise.

Episcleritis

Episcleritis is a benign, self-limited, inflammatory condition of the lining of the eye between the conjunctiva and the sclera.[76] There is usually sectorial redness without discharge (Fig. 25-15). There is often a history of previous episodes. Discomfort is typically mild or absent. The presence of severe pain, photophobia, or decreased vision suggests another diagnosis. Episcleritis is often misdiagnosed as conjunctivitis, but the lack of a discharge and the sectorial redness usually seen in episcleritis help differentiate between the two disorders.

Episcleritis is usually self-limited and resolves without treatment over 3 to 4 weeks.[76] If symptoms are troublesome, it may be treated with prednisolone drops four times a day for 3 days.

SOLAR RETINOPATHY

The retina is protected from UV radiation damage because this high-energy radiation is absorbed by the cornea and the lens of the eye. However, visible and near-infrared light of sufficient intensity may reach the retina and cause photochemical damage.[76] In the wilderness setting, this would most likely occur as the result of staring at the sun or a solar eclipse. Shortly after such an exposure, the individual may experience blurred or distorted vision, a central scotoma, or a headache. Visual acuity is often reduced to the 20/40 to 20/70 range. There is no effective therapy for this injury, but visual acuity may return to normal over a period of several months in mild cases.

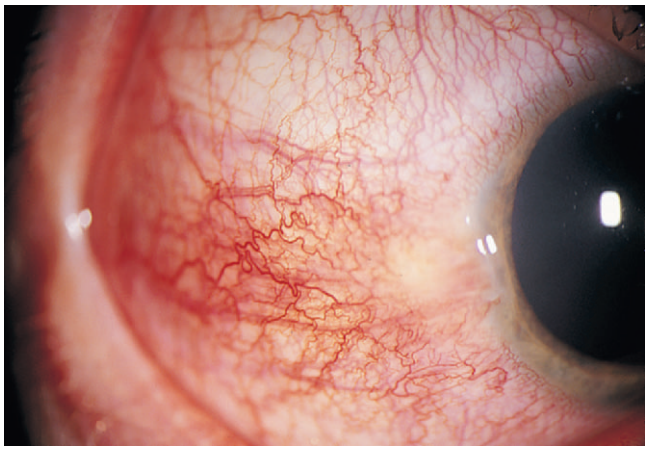


Figure 25-15. Episcleritis. (Courtesy Steve Chalfin, MD, University of Texas Health Science Center, San Antonio, TX.)

LOCATING A DISPLACED CONTACT LENS

Soft contact lens wearers may occasionally have one of their lenses become displaced, causing blurred vision and a foreign body sensation. Once the lens is displaced, it may be hard to locate. The conjunctival fornix of the lower lid is easily examined by distracting the lens from the globe with gentle downward finger pressure applied to the lower lid. If the contact lens has been displaced into the superior conjunctival fornix (usually the case), it may be more difficult to locate. If visual inspection with a penlight and a handheld magnifying lens is not successful in finding the lens, gentle digital massage over the closed upper lid directed toward the medial canthus often results in the contact lens emerging at that location. Several minutes of massage may be required. A few drops of artificial tears often facilitate the process. If this maneuver is unproductive, the eye may be anesthetized with a drop of tetracaine, the upper lid distracted from the globe with upward finger pressure, and the fornix swept with a moistened cotton-tipped applicator.

IMPROVISATION

See also Chapter 21.

If you encounter a person with known or suspected globe rupture, it is of paramount importance to ensure that subsequent inadvertent trauma does not cause extrusion of ocular contents. If an eye shield is not available, one may be fashioned with duct or rigger's tape and any available rigid flat or concave object that can be placed over the eye. Examples include small cups or bowls to provide a good standoff distance between the eye and the improvised shield. Improvised wound closure strips can be made by tearing rigger's or duct tape into $^1/_4$-inch widths. Spectacles and sunglasses can be improvised by making a pinhole in a piece of paper or cardboard. (Make the pinholes before placing the paper or cardboard in front of the eyes!) Take note of the restriction in peripheral vision caused by using pinhole glasses. If the purpose is to improvise sunglasses rather than to achieve a refractive effect, the pinhole can be larger or fashioned into a horizontal slit to improve peripheral vision.

TABLE 25-1. Pressures and Equivalent Oxygen Fractions at Altitude

FEET	METERS	ATMOSPHERIC PRESSURE (mm Hg)	EQUIVALENT OF OXYGEN (%)	PARTIAL PRESSURE OF OXYGEN (mm Hg)
Sea level	Sea level	760	20.9	159
4,000	1,219	656	18.0	137
7,000	2,134	586	16.1	122
10,000	3,048	523	14.4	109
15,000	4,572	429	11.8	89
20,000	6,096	347	9.5	73
25,000	7,620	282	7.8	60
29,028	8,848	253	7.0	53

Improvised magnifying glasses for examining the eye or other small areas may be made by simply using a pair of reading or hyperopic glasses. Another refractive improvisation technique involves a handheld magnifying lens. This convex lens with its plus refracting power can be used to provide a variety of hyperopic refractive corrections by moving the lens to various distances in front of the eye.

THE EYE AT ALTITUDE

Ocular disorders associated with altitude exposures have been addressed in several recent review articles.[17,63,78]

Altitude Exposures and Ocular Physiology

A number of significant effects on visual function resulting from the hypoxia of altitude were described by Wilmer and Berens[142] in their classic article, and later by Sharma.[113] The decrease in ambient pressure at altitude causes a hypobaric hypoxia despite a constant oxygen fraction of 0.21, as noted in Table 25-1. Retinal blood flow has been shown to increase by 128% after 4 days at 5300 m (17,384 feet).[42] This increase in blood flow results in clinically observable changes, such as increase in diameter and tortuosity of retinal vessels and optic disc hyperemia, which are seen in most unacclimatized persons at altitudes above 4573 m (15,000 feet).[42,65,81,103]

Because of its avascularity, the cornea receives most of its oxygen supply from the surrounding atmosphere,[120] so it may suffer hypoxic dysfunction even if the inspired gas mix is not hypoxic, as noted later in the section on Refractive Changes at Altitude after Refractive Surgery. This is an important factor when considering topics such as the suitability of contact lenses and refractive surgical procedures for mountaineers and aviators.

High-Altitude Retinal Hemorrhage

There are many reports of retinal hemorrhages (Fig. 25-16) in mountain climbers.* These have been described as high-altitude retinal hemorrhages (HARHs) or as part of the more inclusive term altitude *retinopathy*.[20] A classification for HARH has been developed by Weidman.[133,134] Butler and colleagues[20] reported a 29% incidence of HARH in climbers on a Mt. Everest expedition at altitudes ranging from 5300 to 8200 m (17,385 to 26,896 feet). McFadden and coworkers[81] found that 56% of their subjects had HARH at an altitude of 5360 m (17,581 feet), and that one had a retinal nerve fiber layer infarct (cotton wool spot; Fig. 25-17). They also reported that exercise at altitude was associated with both an increased incidence of HARH and fluorescein leakage from the retinal vessels. Hackett and Rennie[47] described HARH in 4% of 140 trekkers examined at 4243 m (13,917 feet) at Pheriche in the Himalayas. These authors also found a significant correlation of retinal hemorrhages with symptoms of acute mountain sickness (AMS). Kobrick and Appleton[65] found no retinal hemorrhages in eight subjects examined after 48 hours at 4573 m (15,000 feet) in a hypobaric chamber, although all subjects displayed the marked vascular engorgement and tortuosity typical of the eye at altitude (Fig. 25-18). HARH developed in 19 of 21 climbers who climbed above 7600 m (25,000 feet). Fourteen of 19 climbers who climbed to between 4880 and 7600 m (16,000 and 25,000 feet) were found to have retinopathy.[140] Differences in the incidence of HARH for exposures at similar altitudes may be due to differences in time at altitude before examination, acclimatization schedule, exercise levels, examination techniques, and the presence of concurrent conditions that may predispose to HARH.

High-altitude retinal hemorrhage has been reported to be associated with both altitude headache and a history of vascular headache at sea level.[111] Rimsza and colleagues[105] noted that HARH may occur at lower altitudes in individuals with chronic lung disorders that interfere with oxygenation. Their case report described a woman with cystic fibrosis who had climbed as high

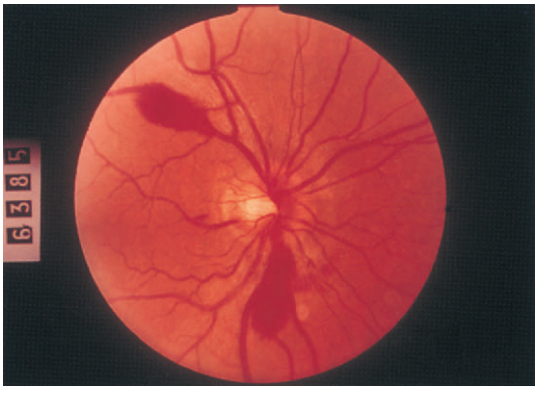


Figure 25-16. High-altitude retinal hemorrhages. (Courtesy Dr. M. McFadden, UBC, Vancouver, British Columbia, in association with Dr. C. Houston, Burlington, VT; Dr. G. Gray, DCIEM, Toronto, Ontario; and Drs. J. Sutton and P. Powells, McMaster University, Hamilton, Ontario, Canada.)

*References 20, 41, 47, 68, 71, 81, 103, 105, 111, 115, 141.

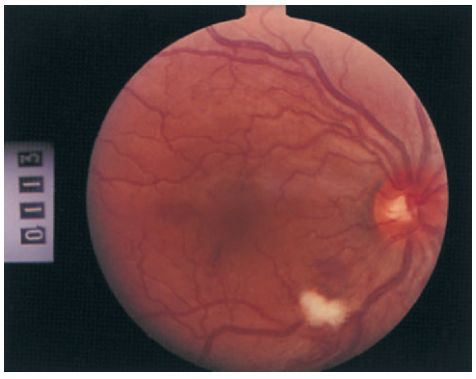


Figure 25-17. Cotton wool spots seen at 5400 m (17,712 feet). This occurred in a climber after Valsalva maneuver and represents the most severe form of retinopathy. (Courtesy Dr. M. McFadden, UBC, Vancouver, British Columbia, in association with Dr. C. Houston, Burlington, VT; Dr. G. Gray, DCIEM, Toronto, Ontario; and Drs. J. Sutton and P. Powells, McMaster University, Hamilton, Ontario, Canada.)

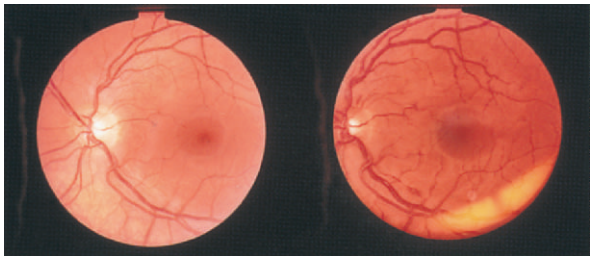


Figure 25-18. The normal fundus at sea level (*left*). The same fundus at 5400 m (17,712 feet; *right*). Note the vascular engorgement and tortuosity at altitude. (Courtesy Dr. M. McFadden, UBC, Vancouver, British Columbia, in association with Dr. C. Houston, Burlington, VT; Dr. G. Gray, DCIEM, Toronto, Ontario; and Drs. J. Sutton and P. Powells, McMaster University, Hamilton, Ontario, Canada.)

as 3049 m (10,000 feet) but was at 1677 m (5500 feet) when she noted a sudden decrease in vision associated with preretinal hemorrhage of the right eye. They also noted that the ocular findings of cystic fibrosis are similar to those seen with altitude retinopathy.[105]

Although HARHs are often not associated with acute visual symptoms,[20,103,141,147] they may result in a loss of visual acuity or paracentral scotomas.[41,71,115,141] Lang and Kuba[71] reported a person who experienced decreased visual acuity and central scotoma from HARH resulting from a 7000-m (22,960-foot) altitude exposure. Permanent deficits in visual function are uncommon but have been reported.[115,141] Shults and Swan[115] reported that four survivors of an ill-fated Aconcagua expedition in Argentina in 1973 were found to have severe HARH after an altitude exposure of 6860 m (22,500 feet). Two of the survivors suffered apparently permanent paracentral scotomas. No reports were found of a progressive decrease in visual acuity or progressive enlargement of paracentral scotomas as a result of remaining at altitude after the development of HARH.[17] Wiedman[141] reported a case in which further ascent after the development of HARH resulted in additional lesions. HARH that results in decreased visual acuity should be a contraindication to further ascent.[17] Butler and Harris[20] recommended that evacuation of individuals with decreases in visual function resulting from HARH (in the absence of high-altitude cerebral edema [HACE] or high-altitude pulmonary edema [HAPE]) be considered nonemergent unless reexamination indicates a progressive deterioration of vision or increasingly severe retinopathy. HARH resolves over a period of 2 to 8 weeks after the altitude exposure is terminated.[141] Recognizing advancing grades of HARH may allow physicians to recommend initiating treatment with oxygen, steroids, diuretics, and immediate descent to prevent HARH progression, macular involvement, or potentially fatal HACE. High-altitude retinopathy is both a significant component of and a predictor of progressive altitude illness.[140]

Intraocular Gas Bubbles at Altitude

The presence of an intraocular gas bubble presents a hazard for anyone exposed to a change in ambient pressure. Intraocular gases are frequently used to provide internal tamponade during and after vitreoretinal surgery. Three patients were described by Hart and associates[50] who lost vision in eyes with intraocular gas after subsequent nitrous oxide (N_2O) general anesthesia. All three patients suffered visual loss due to presumed retinal artery occlusion caused by expansion of the intraocular gas by N_2O during general anesthesia.[50] N_2O is a highly soluble inhalational anesthetic agent that diffuses rapidly down its concentration gradient into an intraocular gas bubble. N_2O is known to increase intraocular gas bubble volume during general anesthesia.[144] The resulting expansion of the bubble can lead to a rapid rise in intraocular pressure.[121]

Growth of intraocular gas bubbles can also be caused by exposure to the reduced atmospheric pressure in air travel.[66,84] Even small bubbles can cause symptomatic rises in intraocular pressure. The patient with residual intraocular gas who experiences pain or dimness of vision in an ascending airplane can obtain relief if the cabin altitude is decreased to 610 m (2000 feet).[75] A patient who had had a scleral buckle and pneumatic retinopexy with a residual gas bubble 2 weeks before a commercial airline flight experienced sudden blindness presumably because of expansion of the gas bubble with resultant central retinal artery occlusion when the aircraft ascended to a cabin pressure of 2440 m (8000 feet).[100] The mechanism of this injury is presumed to be a rise in intraocular pressure that exceeds the central artery pressure, thereby collapsing the artery. The patient's symptoms were relieved when an onboard flight surgeon recognized the problem and the cabin pressure was reset to 610 m (2000 feet).[100]

The same problem can occur if an individual with an intraocular gas bubble ascends to altitude during mountaineering.[48] Fang and Huang[35] reported a case of a 46-year-old man who ascended to an altitude of 1893 m (6210 feet) after vitreoretinal surgery with a residual perfluoropropane (C_3F_8) bubble in his right eye. He noted a sensation of fullness in his right eye accompanied by loss of vision. When he returned to sea level, his intraocular pressure was found to be 54 mm Hg. The etiology of this vision loss was believed to be central retinal artery occlusion caused by expansion of intraocular gas during mountain travel.[35]

Cortical Blindness at High Altitude

Hackett[46] reported six cases of cortical blindness at high altitude. These victims were found to have intact pupillary reflexes. Descent, Gamow (hyperbaric) bag recompression, or supplemental oxygen breathing should be used in persons with neurologic dysfunction at altitude.

Ocular Motility

Kramar and colleagues[68] reported that convergence insufficiency was found in women with altitude illness. Basnyat[6] noted that lateral gaze palsy and other focal neurologic deficits, often in the absence of other symptoms of AMS or HACE, are seen commonly by physicians working for the Himalayan Rescue Association. Rennie and Morrissey[103] reported a person with nystagmus at altitude that was associated with ataxia and intention tremor. Ocular motility abnormalities should be managed in the same way as cortical blindness at high altitude, as described in the preceding section.

Contact Lenses in Mountaineering

Contact lenses may be used successfully at high altitude.[23,147] Clarke[23] noted that contact lenses were used successfully by five members of the British Everest Expedition in 1975 up to altitudes as high as 7317 m (24,000 feet). Use of contact lenses at altitude during trekking or mountaineering entails several considerations beyond those encountered in normal use. In general, overnight use of extended-wear contact lenses is not recommended because of the associated increased rate of microbial keratitis. Even soft contact lenses decrease the oxygen available to the cornea. Lid closure during sleep further accentuates corneal hypoxia. Removing contact lenses at night, however, presents logistical problems in the mountaineering setting. Practicing acceptable lens hygiene during an expedition is difficult. The mountaineer who leaves contact lenses in a case filled with liquid solution in the tent outside of his or her sleeping bag at night may awaken to find the solution and lenses frozen solid. Finally, wearing contact lenses can make eyes more sensitive to glare.[38]

Guidelines for military personnel using contact lenses in austere environments have been developed and apply to the expedition setting[17]:

1. Disposable extended-wear lenses may be left in the eye for up to 1 week. If the wearer is still in the field at the end of this period, the lenses should be removed and discarded. After an overnight period without lenses, new lenses may be inserted, with strict attention to contact lens hygiene.
2. Contact lens wearers should always have backup glasses available for use in the wilderness in case a lens is lost or becomes painful.
3. Individuals who wear contact lenses on expeditions should carry both fluoroquinolone eye drops and contact lens rewetting solution. Both types of drops may freeze if not protected from the cold.
4. Contact lens wearers often note that their eyes become dry. This discomfort may be alleviated with contact lens rewetting drops.
5. Contact lens wearers often note increased sensitivity to sunlight. Individuals who wear contact lenses in the field during daylight hours should carry sunglasses.

Continuous wearing of disposable contact lenses for a week, followed by discarding of the lenses and insertion of fresh lenses after an overnight period without a lens, is a controversial approach to contact lens wear in an expedition setting. Whether the reduction in lens handling offsets the increased risk of microbial keratitis resulting from overnight wear is unknown. The decision to wear contact lenses while mountaineering should be made carefully. Microbial keratitis (corneal ulcers) can pose a significant threat to vision under the best of circumstances. Should this disorder occur with a 7- to 10-day delay to definitive ophthalmologic care, the danger of permanent loss of vision is great. Any eye pain that occurs in contacts lens wearers in the wilderness should be managed as described previously in the Acute Red Eye section. Contact lenses that block out harmful UV rays are now available (Acuvue, Precision UV), but sunglasses are still a good idea both to help protect the eyes from drying wind effects and the eyelids from UV exposure even if contacts are worn. Considering all the potential problems, a good pair of prescription glacier glasses or laser refractive surgery might be a more reasonable alternative than contact lenses as a long-term solution to the refractive needs of mountaineers.

Refractive Changes at Altitude after Refractive Surgery

An acute hyperopic shift in persons who have had radial keratotomy (RK) and then experience an altitude exposure was reported by Snyder and colleagues[122] in 1988 and White and Mader[139] in 1993. This effect has been observed at altitudes as low as 2744 m (9000 feet).[122] A dramatic example of this phenomenon was that experienced by Dr. Beck Weathers in the Mt. Everest tragedy of May 1996 in which eight climbers also lost their lives. Dr. Weathers had undergone bilateral RK years before the expedition. He noted a decrease in vision, which started early during his ascent.[67] Author Jon Krakauer recalls that ". . . as he was ascending from Camp Three to Camp Four, Beck later confessed to me, 'my vision had gotten so bad that I couldn't see more than a few feet.' "[67] This decrease in vision forced Dr. Weathers to abandon his quest for the summit shortly after leaving Camp Four and nearly resulted in his death. Another report describes two expert climbers who experienced hyperopic shifts of 3 diopters or more during altitude exposures of 5000 m (16,400 feet) or higher on Mt. McKinley and Mt. Everest.[24] Mader and White[79] found that the magnitude of the hyperopic shift was 1.03 ± 0.16 diopters after 24 hours at 3659 m (12,000 feet) and 1.94 ± 0.26 diopters at 5183 m (17,000 feet). Ng and coworkers[90] reported no refractive change after 6 hours in post-RK eyes at a simulated altitude of 3659 m (12,197 feet), suggesting that the hyperopic shift requires more than 6 hours to develop.

Further studies by Mader and colleagues[77] at 4299 m (14,100 feet) on Pike's Peak revealed that (1) a subject who had undergone RK demonstrated a progressive hyperopic shift associated with flattened keratometry findings during a 72-hour exposure; (2) control eyes and eyes that had undergone laser refractive surgery (photorefractive keratectomy [PRK]) experienced no change in their refractive state; (3) peripheral corneal thickening was seen on pachymetry in all three groups; and (4) refraction, keratometry, and pachymetry all returned to baseline after return to sea level. Winkle and associates[143] demonstrated that exposing post-RK corneas to 100% nitrogen through goggles at 1 atmosphere for 2 hours caused a significant hyperopic shift of 1.24 diopters and corneal flattening of 1.19 diopters. Corneal thickness increased in both post-RK and control eyes but was not associated with a hyperopic shift in control eyes. This is strong evidence that the effect of altitude exposures on post-RK eyes is caused by hypoxia rather than by hypobarism and further illustrates that breathing a normoxic inspired gas mix will not protect against the development of hypoxic corneal changes in post-RK eyes.

The effect of the post-RK hyperopic shift seen at altitude depends on the postoperative refractive state (undercorrected

patients may actually have their vision improve) and the accommodative abilities of the individual.[79] The work of Mader and colleagues has provided compelling evidence that for myopic mountaineers, PRK instead of RK is the refractive surgical procedure of choice. Individuals who have undergone RK and plan to undertake an altitude exposure of 2744 m (9000 feet) or higher while mountaineering should bring multiple spectacles with increasing plus lens power.[79] To my knowledge, a post-RK hyperopic shift has not been reported in airline passengers or flight crew. This may be because the latent period required for this phenomenon to develop exceeds the duration of most commercial flights or because the approximate 2440-m (8000-foot) cabin pressure on most commercial flights does not produce sufficient corneal hypoxia for a hyperopic shift to occur.

The altitude at which RK is performed also affects the surgical outcome. RK done at altitude (1753 m or 5750 feet) produces more of a hyperopic shift than the same procedure done at sea level.[22,29,119] The average changes in spherical equivalent cycloplegic refraction were 5.09 ± 1.29 diopters and 6.50 ± 2.24 diopters in subjects who had undergone the same procedure by the same surgeon at sea level and 1753 m (5750 feet), respectively.[119] In another study, the mean spherical equivalent cycloplegic refraction changes were 4.40 ± 0.92 diopters and 6.03 ± 1.13 diopters in patients who had had RK at sea level (Istanbul, Turkey) and 1720 m (5641 feet) (Van, Turkey), respectively ($P < .001$). The procedures at both altitudes were done by the same surgeon using the same technique, demonstrating the potential need to adjust surgical nomograms for altitude.[22]

The most commonly performed laser refractive surgery at present is laser in-situ keratomileusis (LASIK).[78] White and Mader[138] reported that a climber who had undergone LASIK developed blurred vision after summitting Alpamyo (5945 m or 19,500 feet). He noticed the blurred vision several days later after spending two nights at 5488 m (18,000 feet). His vision cleared 3 days later after returning to 3048 m (10,000 feet).[138] Recent studies have examined the effects of altitude exposure on individuals who have undergone this procedure. Six climbers who had bilateral LASIK subsequently undertook an ascent of Mt. Everest.[29] Visual acuities were measured at base camp (5365 m or 17,600 feet); subjective symptoms (but not measured acuities) were obtained at higher altitudes. All climbers ascended to at least 7927 m (26,000 feet) and four of the six summitted at 8852 m (29,035 feet). Five of the six climbers reported no visual changes at altitudes up to 7927 m (26,000 feet). One climber reported mild blurring with ascent above 4878 m (16,000 feet) that improved with descent or prolonged stay at altitude. Two additional climbers reported visual blurring at altitudes of 8231 m (27,000 feet) and 8689 m (28,500 feet). The authors' conclusion was that LASIK may be a good choice for individuals involved in high-altitude activities, but those achieving extreme altitudes of 7927 m (26,000 feet) and above should be aware of possible fluctuation of vision.[29] In another study, two physicians who had undergone myopic LASIK 8 and 14 weeks previously subsequently undertook an ascent of Mt. Aconcagua (6964 m or 22,841 feet). They prospectively assessed their vision at 610-m (2000-foot) intervals during ascent. Both climbers described moderate loss of distance acuity with preserved near and pinhole acuity. These findings are suggestive of a myopic shift at altitude. Blurred vision began to occur at 5488 m (18,000 feet) in the left eye and at the summit (6964 m or 22,841 feet) in the right eye in one climber; blurring was first noticed at 5488 m (18,000 feet) in the right eye in the second climber. These myopic shifts resolved after return to sea level.[12] These data suggest that a small refractive shift in the myopic direction may be present at extreme altitudes. Climbers who do not ascend beyond moderate altitudes should not experience a post-LASIK refractive shift.

Ultraviolet Radiation Damage

Ultraviolet radiation is divided into UV-A (320 to 400 nm), UV-B (290 to 320 nm), and UV-C (100 to 290 nm). Almost all UV-C radiation is absorbed by the earth's ozone layer.[94,126] The cornea absorbs all radiation with wavelengths of less than approximately 300 nm, and the lens absorbs almost all of the remaining UV-B radiation that reaches it.[126] UV radiation is increased with high-altitude, low-latitude, and highly reflective environments.[94] Altitude exposures associated with mountaineering entail exposures to increases in the amount of both incident and reflected UV light (see also Chapter 14).

Acute exposure to high levels of UV radiation may result in UV photokeratitis,[30,126] whereas chronic exposures may be associated with cortical lens opacities,[25] posterior subcapsular lens opacities,[11] pterygia,[9] and squamous cell carcinoma of the conjunctiva.[88] The Salisbury Eye Evaluation project[136] quantified ocular UV-B exposure using a UV-B pyranometer. This study found that cataract incidence increased (odds ratio 1.1; 95% confidence interval 1.02 to 1.20) with the degree of UV-B sun exposure, from the lowest to the highest quartile.

Diagnosis and management of UV keratitis were discussed previously in this chapter. The best strategy for dealing with UV radiation–induced disorders is prevention. Most experienced mountaineers and trekkers are well aware of the need for sunglasses with high rates of UV absorption. UV attenuation in sunglasses depends on the size, shape, and wearing position, as well as the absorption properties of the optical material used.[107] The use of a brimmed hat is another effective means of decreasing UV exposure to the eye.[38,94,108] In conjunction with the use of topical sun-blocking agents, a hat may also help prevent a series of cutaneous neoplasms from being a lasting reminder of previous mountaineering expeditions.

Glaucoma at Altitude

Mader and Tabin[78] discuss the hazards of going to altitude with severe glaucoma. Even individuals who have mild, well-controlled glaucoma may suffer adverse effects at altitude if they are being treated with topical beta blockers. These drugs may produce systemic side effects of lethargy and exercise intolerance that may be exacerbated at altitude.[78] Consideration should be given to other choices for topical therapy in persons who will be traveling to significant altitude. The hypoxia of altitude may also worsen glaucomatous optic neuropathy. One person with moderately severe glaucoma who was well controlled on two topical medications ascended to 6707 m (22,000 ft) on Cho Oyu in the Himalayas. He noticed a decrease in vision and descended. Intraocular pressure was normal when measured in Kathmandu, but a marked increase in optic nerve damage was later confirmed by visual field and optic nerve imaging studies.[78]

Sunglasses Selection in Mountaineering

In selecting sunglasses for use in mountaineering, absorption of essentially all UV radiation is a key consideration because radiation in this portion of the electromagnetic spectrum is not visible and serves only to produce adverse effects in the eye. Another critical consideration is the amount of visible light transmitted. Sunglasses that suffice for everyday use may not be

TABLE 25-2. Sunglasses Selection Criteria for Mountaineering

SUBJECT	CHARACTERISTICS
Ultraviolet light absorption	99%–100%
Visible light transmittance	5%–10%*
Lens material	Polycarbonate or CR-39†
Optical quality	Clear image without distortion‡
Frame design features	Large lenses; side shields or wraparound design; fit close to face; good stability on face during movement; lightweight; durable
Color	Gray§

*Glasses with less than 8% transmittance of visible light should not be worn while driving. Sunglasses or any tinted lenses with a visible light transmittance of less than 80% should not be worn while driving at night.[126]

†Glass lenses typically have very good optical clarity and scratch resistance but are heavier and more expensive.

‡Hold the sunglasses at arm's length and move them back and forth. If the objects are distorted or move erratically, the optical quality is probably less than desirable. Also, compare the image quality between several different pairs of sunglasses to get a basis for comparison.

§The use of colored lens tints can alter color perception and possibly compromise the visibility of traffic signals. Neutral gray absorbs light relatively constantly across the visible spectrum and avoids these problems.[126]

adequate for use in the mountains, especially while on snow or glaciers. The comfort zone for luminance is approximately 350 to 2000 candelas/m^2.[126] An outdoor environment consisting of sunlit fields and foliage may have a luminance of 3000 to 7000 candelas/m^2; a bright beach may have a luminance of 6000 to 15,000 candelas/m^2. Standard sunglasses that transmit 15% to 25% of visible light reduce the luminance in these situations to within the comfort range. In contrast, bright sun reflected off snow or clouds may result in luminances of 15,000 to 30,000 candelas/m^2. Sunglasses with visible light transmittance in the 5% to 10% range are needed to reduce luminance to a comfortable range in these circumstances. (The 1986 American National Standards Institute [ANSI] standards for nonprescription sunglasses recommend that tinted lenses with visible light transmittances of less than 8% not be used for driving.[126]) Side shields or deeply wrapped lens designs should be used.[126] Infrared absorption is important in certain industrial occupations, such as glass, iron, and steelworkers,[126] but is less important in protective eyewear to be used outdoors. Table 25-2 provides desirable characteristics in selecting sunglasses for mountaineering or other environments with high levels of luminance and UV radiation.[17] Individuals involved in mountaineering expeditions should always carry spare sunglasses in case the primary pair is lost or broken.

Photochromic Lenses

Photochromic lenses change transmittance or color when exposed to light or UV radiation.[126] They are designed to transmit a greater percentage of incident light when indoors or in conditions of reduced illumination and a reduced amount of light when exposed to higher levels of illumination. This is accomplished in one example of glass lenses by incorporating an inorganic silver halide into the lens. When the lens is exposed to sunlight, this compound decomposes into its component silver and halide ions and the lens turns dark gray.[126] When the lens is removed from sunlight, the process reverses. When selecting photochromic lenses, the adequacy of indoor light transmittance can be judged while trying them on. The outdoor transmittance should be approximately 5% to 10%, as noted in Table 25-2, if the glasses are to be used for mountaineering. Most photochromic lenses have outdoor transmittances of approximately 20%, which should suffice for less highly reflective outdoor environments.[126]

Several plastic photochromic lenses are now available. The darkening process in plastic lenses is accomplished by organic light-sensitive compounds that are suspended in a thin layer near the front of the lens. Plastic photochromic lenses generally do not darken well enough to be used as sunglasses in bright environments. In addition, unlike glass photochromic lenses, the photochromic reaction typically used in plastic lenses usually fades in 1 to 2 years.[126]

THE EYE AND DIVING

The ocular aspects of scuba diving and other hyperbaric exposures were reviewed in a 1995 paper.[16] The sections from that paper that address the hyperbaric environment, ocular barotrauma, the ocular manifestations of decompression sickness (DCS) and arterial gas embolism, ophthalmic considerations in fitness-to-dive evaluations, and the differential diagnosis of decreased vision after diving are discussed here. An expanded discussion of these issues, as well as additional material on diving after eye surgery, the effect of common eye medications on fitness to dive, and the use of hyperbaric oxygen to treat ocular disorders, may be found in the review article.

The Hyperbaric Environment

See also Chapter 71.

At sea level, the body is exposed to 1 atmosphere (atm) of pressure. This magnitude of pressure may also be expressed as 760 mm Hg, 33 feet of sea water (fsw), and 14.7 pounds per square inch (psi).

The normal atmospheric pressure of 1 atm is often used as a reference point from which other pressures are measured. When one states that the intraocular pressure is 15 mm Hg, what is meant is that the intraocular pressure is 15 mm Hg greater than the surrounding environment. In fact, the absolute pressure inside the eye at sea level is 775 (760 + 15) mm Hg. The intraocular pressure that is measured with a tonometer is therefore a "gauge" pressure, meaning that the pressure displayed is the actual pressure minus the atmospheric pressure.

Ophthalmic Considerations in the Fitness-to-Dive Evaluation

A diver should have adequate visual acuity to be able to read his or her gauges and function safely underwater. Possession of a driver's license is a convenient indication that a potential diver has sufficient visual acuity to meet this standard.[16] A person who has recently undergone ophthalmic surgery should refrain from diving until the recommended convalescent interval has passed.[16] Individuals who suffer from glaucoma may dive safely unless they have had glaucoma filtering surgery performed.[16] Systemic carbonic anhydrase inhibitors are best avoided in patients with glaucoma who wish to dive because of possible confusion between medication-induced paresthesias and DCS.[16] Any individual who is suffering from an acute ocular disorder that causes significant pain, decreased visual acuity, or other disabling symptoms should refrain from diving until these symptoms have resolved.[16] Individuals who have undergone surgical

repair of retinal detachments may occasionally have gas bubbles placed in the eye to stent the retina against the retinal pigment epithelium. Intraocular gas bubbles are an absolute contraindication to both diving and hyperbaric chamber exposures. A study was done in which rabbit eyes were injected with perfluoropropane (C_3F_8) to a bubble size of 60% of the vitreous cavity. When the eyes containing the bubbles were exposed to increased atmospheric pressure, intraocular pressure dropped in comparison to the surrounding environment and then rose to more than 50 mm Hg when the atmospheric pressure was returned to normal.[59] The individual who dives with a gas bubble may experience severe pain on descent owing to compression of the globe as the gas phase is compressed,[99] or on ascent after extra gas has diffused into the bubble and the absolute pressure is reduced on surfacing, causing the bubble to grow and intraocular pressure to rise.

Underwater Refractive Correction

If contact lenses are to be used for diving, soft contact lenses are preferred.[26,33,61,82] Hard (polymethylmethacrylate) contact lenses have been associated with corneal edema during decompression and after dives.[117,118] These changes are caused by formation of nitrogen bubbles in the precorneal tear film during decompression, which interferes with normal tear film physiology and results in epithelial edema. Bubble formation under contact lenses would be expected to be more common during dives with significant decompression stress.

Although the increased gaseous diffusion properties of rigid gas-permeable contact lenses theoretically decrease the chance of bubble formation in the tear film, use of these lenses while diving has been demonstrated to cause bubble formation under the lens, leading to secondary corneal epithelial disruption.[123] The author has treated one diver with foreign body sensation and blurred visual acuity that occurred during ascent while wearing gas-permeable contact lenses. Symptoms resolved on removal of the lens at the surface.

Corneal edema was not observed in one series in which soft contact lenses were studied.[117] The most frequent complication of soft contact lens use in diving is loss of the lens.[61,64] The risk of lens loss can be minimized by ensuring a good seal on the face mask and minimizing the amount of water that gets into the air space of the mask. Should the mask become displaced during the dive, narrowing of the palpebral fissures helps decrease the chance of the contact lens floating off the surface of the eye.[61]

Betts[10] reported an incident in which bubbles formed under "micro-corneal" contact lenses. On leaving the hyperbaric chamber after a simulated dive, the diver complained of blurred vision in the right eye, which appeared to have a white film over it. The left eye was similarly affected, but to a lesser extent. On examination with an ophthalmoscope, a haze of fine bubbles could be seen under the contact lens. These bubbles persisted on the surface of the cornea after the lens was removed. The bubbles did not move with blinking and were felt to be in the superficial layers of the cornea. They slowly resolved over 30 minutes. The author speculates that the observed difference in bubble formation between the two eyes may have been due to a difference in lens fit.[10]

A prescription ground face mask is another refractive alternative, as is a face mask with a lens bonded onto the surface of the mask. Masks and lenses may be lost in high swells or rough surf, however, leaving a diver without refractive correction. When contemplating the purchase of an expensive prescription face mask, one needs to be mindful of the corollary of Murphy's Law that applies to diving: "Weight belts always fall on the face masks with prescription lenses."

Refractive corrections for presbyopia present special challenges underwater. Presbyopic divers with contact lens–corrected myopia may require greater near-vision correction underwater than when viewing the same objects in air because of the increased percentage of shorter-wavelength light rays underwater. Divers with presbyopia should consider monovision correction to facilitate underwater visual tasks.[13]

Refractive Surgery and Diving

Laser refractive surgery is a safe and effective means of correcting refractive errors in divers.[16] PRK has been allowed in U.S. Navy divers since 1996. LASIK is now a more commonly performed procedure. Although this procedure presents the potential for both inflammation and infection under the corneal flap as well as traumatic dislocation of the flap, these conditions have not to date been reported as complications of diving. Visual acuity appears to be maintained in the hyperbaric environment. Huang and colleagues[55] reported three patients who had undergone bilateral myopic RK, two who had had bilateral myopic LASIK, and three control subjects. Eye examinations were done at baseline, at 4 atm in a dry chamber, and on return to the surface. Acute hyperbaric stress did not appear significantly to alter refractive power after corneal surgery.[55]

Ocular and Periocular Barotrauma

The eye is normally filled with noncompressible fluid and solid tissues and is therefore protected from barotrauma. However, once a mask is placed over the face, a different circumstance exists. The face mask is an air-filled space bounded on one side by the eyes and ocular adnexa. As a diver descends, if he or she does not expel gas through the nose into the airspace of the face mask, a relative negative pressure develops in this space. If this negative pressure becomes great enough, the eyes and ocular adnexa are drawn toward the space. Marked lid edema with ecchymosis and subconjunctival hemorrhage may develop as tissues and blood vessels are disrupted by this distention. These signs may be alarming to the diver but typically resolve without sequelae. In a more severe case, such as that which may occur when an unconscious diver sinks a significant distance in the water column, more serious injury, including hyphema, may occur.[39] A diver with face mask barotrauma is shown in Figure 25-19.

In some settings, facial barotrauma may be quite severe. The first reported "squeeze" in diving took place on October 4, 1841 on the Royal George salvage work off Portsmouth Harbor, England. Ten months later, on the same project, another diver suffered a severe squeeze when his air hose burst. Colonel Pasley, director of salvage operations, told of removing the diver's helmet: "his face and neck were much swollen, and blood was issuing from his mouth and ears profusely, his eye closed and protruding; he vomited a great deal of blood on being laid on the deck." Severe squeezes in the early days of hard-hat diving resulted from a combined failure of both nonreturn valves and air supply hoses.[96]

Barotrauma is also possible in persons with gas bubbles in the anterior chamber or vitreous cavity. Pressure-induced changes in the volume of this bubble may result in retinal, uveal, or vitreous hemorrhage, as well as in partial collapse of the globe.

Figure 25-19. Mask squeeze in a diver who descended to 45 fsw without exhaling into his mask. (Courtesy Kenneth W. Kizer, MD.)

Permanent loss of vision may ensue. One person who attempted to dive while an iatrogenic bubble was present in the vitreous cavity noted immediate onset of very severe eye pain on descent and quickly aborted his dive.[99] Persons with intraocular gas should not be allowed to dive as long as the bubble remains in the eye. The necessity of adding extra gas to the face mask during descent makes it obvious that swim goggles, which cover only the eyes and not the nose, should never be used for diving.

Facemask barotrauma may also result in damage to the periocular tissues. Bove and Butler[19] reported a diver who noted numbness and paresthesias in the distribution of the infraorbital nerve that resulted from maxillary sinus barotrauma. Butler[18] reported a diver who noted the onset of pain during a dive. After the dive, she noted a bulging sensation of the eye and diplopia. Ophthalmologic examination revealed a tropia and the diver was subsequently found to have sustained a subperiosteal orbital hemorrhage on computed tomography scan.[18]

Decompression Sickness

Ocular involvement in DCS was first reported by Sir Robert Boyle, who observed gas bubbles in the anterior chamber of the eye of a viper that had been experimentally exposed to increased pressure.[14] Ocular manifestations of DCS are infrequently reported in the ophthalmic literature,[16] but there are a number of reports of ocular involvement with DCS in the dive medicine literature.[15,16,34,106] Reported manifestations include nystagmus, diplopia, scotomas, homonymous hemianopias, orbicularis oculi pain, cortical blindness, convergence insufficiency, optic neuropathy, and central retinal artery occlusion.[16] The incidence of visual symptoms in patients with DCS was found to be 7% in one large series.[106]

Physicians should also be aware of the possibility of DCS occurring after altitude exposures.[15,125] Altitude DCS presenting as optic neuropathy has been reported.[15] The risk of DCS may be increased if an altitude exposure occurs after diving without allowing sufficient time for excess nitrogen taken up during the dive to leave the body.[16] Fitzpatrick[36] reviewed 143 cases of altitude-induced type II neurologic DCS. Visual disturbances were identified in 38 cases, for a prevalence of 26.6%. The most commonly reported visual symptoms were blurred vision (61%), loss of vision (34%), and scintillation/scotoma (32%). Forty-five percent of the cases occurred at altitude, 24% occurred within the first hour after the exposure, and 21% occurred 1 to 6 hours after the exposure. No cases were reported to have begun more than 24 hours after the exposure.[36] Special attention should be paid to the possibility that DCS may present with symptoms after a hypobaric exposure that subsequently resolve, only to recur days to even weeks later. Steigleman and coworkers[125] reported a 20-year-old aircrew student who had pain on ascent during altitude exposure. The pain resolved on descent, but recurred 3 weeks later when he experienced severe retro-orbital pain and moderate vision loss in the left eye. His signs and symptoms resolved completely on the first U.S. Navy Treatment Table Six, but recurred once more several days later. He eventually underwent a total of four treatment tables, but had complete resolution of his signs and symptoms.[125]

Decompression sickness is treated with oxygen breathing and recompression on an emergent basis. Ophthalmologists seldom encounter this disease in an acute setting because most divers know to seek recompression therapy for signs or symptoms of DCS. Because treatment usually results in resolution of all symptoms, most persons with visual symptoms before treatment are asymptomatic after recompression treatment and are therefore not referred to ophthalmologists.[14] Should an ophthalmologist encounter a person with acute ocular disturbances consistent with DCS after a hyperbaric or hypobaric exposure, the victim should be referred emergently to the nearest available recompression chamber and diving medicine specialist because DCS may worsen rapidly if not treated. Physicians unsure of the location of the nearest diving medicine specialist or recompression chamber can call the Divers Alert Network at Duke University, Durham, North Carolina (919-684-8111).

Incomplete response to treatment or recurrence of symptoms after treatment may bring a victim with ocular DCS to the ophthalmologist on a less emergent basis. The victim should be managed in conjunction with a dive medicine specialist. Recompression therapy and hyperbaric oxygen should be administered even when a significant delay has occurred between the onset of symptoms and initial evaluation of the victim because treatment may be effective despite delays of up to several weeks.[15,16]

The Ocular Tear Film as an Indicator of Decompression Stress

There has been recent interest in observing bubble formation in the ocular tear film and using the presence and numbers of bubbles observed after hyperbaric exposures as an indicator of the magnitude of decompression stress encountered. Nine healthy adult volunteers made a series of dives to 50 fsw and 90 fsw.[85] In the first series, all subjects spent the 15-minute bottom time resting, whereas on the second they exercised on a bicycle ergometer. Dive profiles were accomplished in accordance with the Canadian Defense and Civil Institute of Environmental Medicine (DCIEM) tables. On the resting profiles, the divers were found to have no bubbles on postdive eye examination, whereas on the exercising profiles bubbles were present.[85] In another study, 11 divers completed two series of

chamber dives according to the Professional Association of Diving Instructors (PADI) tables.[83] The first dive series was 70 fsw for 15, 29, and 40 minutes. The second was maximum-duration no-stop diving at 40 fsw (140 minutes), 70 fsw (40 minutes), 90 fsw (25 minutes), and 120 fsw (13 minutes). Tear film bubble assessment was observed to be more sensitive in detecting decompression stress than was the conventional Doppler ultrasonic surveillance of the precordial region.[83] Bennett and colleagues[8] observed 42 recreational divers after a single day of diving and 11 divers after repetitive, multi-day diving. After diving, bubble counts increased significantly ($P < .01$) from predive values. Single-day divers reached a maximum bubble count at 48 hours of one (0 to 2.25) bubble/eye. Bubble counts were not significantly correlated with inert gas load, body mass index, age, or diving experience.[8] Another study subjected 11 volunteers to pressures of 4 bar for 15 minutes. In the predive examinations, there was only one individual with one bubble observed. Postdive examination found a mean of 6 bubbles per eye (range, 3 to 12; $P < .001$)[127] Whether these observations will eventually be useful for clinicians or decompression researchers remains to be determined.[7]

Ocular Fundus Lesions in Divers

Fluorescein angiography of divers has documented retinal pigment epithelial abnormalities indistinguishable from those seen in eyes with choroidal ischemia.[101] These changes were attributed to decompression-induced intravascular gaseous microemboli. The incidence of these lesions was related to the duration of diving and a history of DCS. Other abnormalities noted more frequently in divers were low retinal capillary density at the fovea, microaneurysms, and small areas of capillary nonperfusion. Retinal pigment epithelium defects were seen in 1 of 23 nondivers and half of the divers. All observed changes were noted by the authors to be consistent with obstruction of the retinal and choroidal circulations. Although no diver was reported to have suffered a loss of visual acuity from these abnormalities, the long-term effects of this phenomenon remain to be studied. Another study[53] reported fundus fluorescein angiography performed on 26 divers who had reportedly used safe diving practice for at least 10 years, and on 7 control subjects. There was no significant difference in the incidence of macular abnormalities between divers and nondivers in this study. The authors concluded that adherence to safe diving practice confers some protection against the macular abnormalities known to occur in divers with a history of DCS.[53] A third study[87] compared the fluorescein angiograms of 55 Royal Navy divers with those of 24 nondivers. No differences were found between divers and nondivers, and the prevalence of abnormalities was not correlated with diving experience. Visual fields were normal in all control subjects and divers. Twenty-five percent of control subjects and 25% of divers had retinal pigment epithelium defects. The authors believed that their divers, as members of the Royal Navy, may have followed safer diving practices and thus avoided the lesions observed in other studies.[87]

Arterial Gas Embolism

Retrochiasmal defects, such as hemianopias or cortical blindness, may be seen with arterial gas embolism. Central retinal artery occlusion may also result from gas emboli in the ophthalmic artery.[16] Management is similar to that for DCS, with emergent recompression and hyperbaric oxygen therapy indicated in all cases.

Hyperoxic Myopia

Individuals who undergo prolonged exposures to hyperoxic gas mixtures may experience lenticular oxygen toxicity that is manifest initially by a myopic refractive shift. This myopic shift is progressive and usually reversible if the hyperoxic exposures are discontinued. Although this condition is most common in patients undergoing repeated hyperbaric oxygen exposures in a chamber for medical conditions,[2,4] it has also been reported in a scuba diver doing a series of prolonged dives using a gas mix with a constant partial pressure of oxygen of 1.3 atm.[21] This condition has also been noted to occur in caisson workers.[95]

Ocular Jellyfish Stings

Divers and swimmers may occasionally be stung by jellyfish on and around the eyes. The pain from this injury may be severe, but is typically self-limited, usually resolving within 24 to 48 hours. Glasser and colleagues,[44] however, reported a series of five cases associated with unusually severe and prolonged iritis and elevation of intraocular pressure. Intraocular pressures ranged from 32 to 48 mm Hg and were treated successfully with topical beta blockers and oral carbonic anhydrase inhibitors. One patient developed chronic unilateral glaucoma. The iritis persisted for up to 8 weeks. This report illustrates the potential for serious long-term sequelae from ocular jellyfish stings. All patients with ocular jellyfish stings that do not resolve within 24 to 48 hours should be seen urgently by an ophthalmologist.[44]

Differential Diagnosis of Decreased Vision after Diving

Decompression sickness and arterial gas embolism should be considered whenever vision is acutely decreased after diving because of the possible emergent need for recompression therapy, especially if any other manifestation of DCS or arterial gas embolism is present. Other disorders may also affect vision after a dive. Corneal edema resulting from the formation of gas bubbles under polymethylmethacrylate and rigid gas-permeable contact lenses may cause decreased vision. A soft contact lens wearer who complains of blurred vision after a dive may have a lost or displaced lens.

Another possible cause of nondysbaric decreased vision after a dive is epithelial keratopathy induced by chemical agents used to reduce face mask fogging. The time-honored application of saliva or toothpaste to the interior surface of the mask reduces but does not eliminate fogging. This led to the development of commercial antifog agents designed to be applied to the inside surface of face masks. These agents may contain volatile compounds potentially toxic to the corneal epithelium, including glycols, alcohols, and phenol derivatives. Exposure to these compounds may result in blurred vision, photophobia, tearing, and blepharospasm that may not develop until several hours after the dive.[145] Slit-lamp examination typically reveals diffuse superficial punctate keratopathy. Development of this syndrome commonly results from improper use of the antifog agent, such as overly generous application or failure to rinse the mask before use.

The author has treated several persons with recurrent mild ocular irritation and blurring of vision after dives on which soft contact lenses were worn. The lenses were noted to be tightly adherent to the cornea, probably as a result of a decrease in water content in the lens after contact with hypertonic sea water. Symptoms were relieved with a few drops of isotonic artificial tears. Decreased movement of soft contact lenses on the

cornea has been reported to occur from exposure to swimming pool water as well.[28]

Divers sometimes use a transdermal scopolamine patch (placed behind the ear) to prevent motion sickness. This may result in mydriasis, decreased accommodation, and blurred vision in the ipsilateral eye. Hyperoxic myopia may be a possible etiology for the vision loss if the diver has had recurrent or prolonged exposures to elevated partial pressures of oxygen.[21] Divers often use oxymetazoline nasal spray to assist in shrinking mucous membranes in order to equalize the pressure in their middle ears. Vasoconstriction caused by this medication has been reported to cause anterior ischemic optic neuropathy. Fivgas and Newman[37] reported a 43-year-old woman evaluated for sequential bilateral vision loss after the use of oxymetazoline nasal spray. Visual loss in both eyes was temporally associated with the use of the medication. A thorough evaluation for other causes of optic neuropathy was negative.[37]

Migraine-like phenomena have been reported after hyperbaric exposures. Anderson and coworkers[3] reported four individuals who had migrainous signs or symptoms after a hyperbaric exposure. Three of the four had a past history of migraine headaches. They noted sudden appearance of scintillating scotomas 10 to 90 minutes after surfacing with no other manifestations of DCS. Three of the four patients had resolution of symptoms with surface oxygen only. One patient was recompressed as well. These events may represent a migraine event temporally associated with diving, or they may be an atypical manifestation of DCS.[3]

Finally, loss of vision may be caused by ocular disorders that occurred during or shortly after the dive, but were not a direct result of the dive itself. A differential diagnosis of decreased vision after diving is presented in Box 25-6.

Box 25-6. Differential Diagnosis of Decreased Vision after Diving

Decompression sickness
Arterial gas embolism
Bubbles under contact lenses
Displaced contact lens
Antifog agent keratopathy
Contact lens adherence syndrome
Transdermal scopolamine
Hyperoxic myopia
Oxymetazoline optic neuropathy
Diving-induced migraine phenomena
Eye disorders not related to diving

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The author thanks Drs. Steve Chalfin, Dave Harris, and Dave Perlman for their much-appreciated contributions to the preparation of this chapter.

The references for this chapter can be found on the accompanying DVD-ROM.

Wilderness Dentistry and Management of Facial Injuries

26

Henry J. Herrmann and Daniel M. Laskin

Acute conditions involving the mouth and related structures can disrupt outdoor recreational activities. This chapter discusses diagnosis and treatment of a wide variety of emergencies, from the merely annoying to the life-threatening. In addition, local anesthesia for dental emergencies, the dental first-aid kit, and prevention are covered.

HISTORY AND EXAMINATION

History taking for dental emergencies proceeds as with any other condition. In emergency situations, the chief complaint and history of the present illness deserve greater emphasis than family, personal, and social history or review of systems. However, the presence of complicating medical factors or chronic illness may prove very important in choosing the proper treatment. Begin with open-ended questions before focusing attention with more specific questions. It is useful to know how long ago the symptoms started, how frequently they occur, how long they last, how they have changed over time, how severe they are, what brings them on, and what brings relief.

The focus of the examination will depend somewhat on the examiner's suspicions. However, in all cases a systematic approach to examination in oral and maxillofacial emergencies

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The focus of the examination will depend somewhat on the examiner's suspicions. However, in all cases a systematic approach to examination in oral and maxillofacial emergencies

allows efficient collection of all relevant data. Imagine you are a cinematographer, and that you want to start with a wide-angle view of the victim and gradually zoom in to an extreme close-up of the oropharynx. Begin the examination with a general appraisal of the victim that may include temperature, blood pressure, and pulse rate. How sick does he or she appear to be? Next observe the head, neck, and face, especially noting any asymmetry. Palpation may reveal point tenderness, crepitus, foreign bodies, or swelling. Palpate the temporomandibular joints, muscles, lymph nodes, and areas of suspected injury. Observe the victim slowly open and close his or her mouth, observing for degree of opening, range of motion, and deviation on opening.

Begin the intraoral examination with the lips, then move back to the cheeks, floor of the mouth, tongue, hard palate, soft palate, and pharynx. In addition to inspection, palpation is very important. A swelling may turn out to be rock hard (bone) or fluctuant (abscess). Bimanual palpation is helpful when examining the lips, cheeks, and floor of the mouth. The gingiva is observed for color, firmness, recession, and swelling. Gentle probing reveals the depth of the gingival pocket and the presence or absence of bleeding. The teeth are inspected for caries, fractures, presence of plaque and calculus, wear, and loose restorations. Test for tooth mobility by using moderate force in a side-to-side direction. Gentle percussion of the teeth with a metal instrument reveals injury or disease of the supporting structures. Inspect occlusion by retracting the lips and then having the victim bite.

Radiographs and other technology-dependent diagnostic tests are valuable, but for purposes of this chapter are assumed to be unavailable.

Box 26-1. Some Conditions That Cause Facial Pain

- Pulpitis (acute, chronic, hyperplastic)
- Periapical osteitis (acute, chronic)
- Cracked tooth syndrome
- Periodontal infections
  - Periodontal abscess
  - Acute necrotizing ulcerative gingivitis
  - Primary herpetic gingivostomatitis
  - Pericoronitis
- Temporomandibular disorders
- Maxillary sinusitis
- Neurologic pain
  - Trigeminal neuralgia
  - Trigeminal neuritis
  - Herpes zoster neuritis
  - Postherpetic neuralgia
- Atypical facial pain
- Psychogenic facial pain
- Dental causalgia (atypical odontalgia)
- Vascular pain
  - Migraine headache
  - Temporal arteritis
  - Cluster headache
- Referred pain
  - Pulpitis
  - Subacute thyroiditis
  - Myocardial infarction or angina pectoris

MAXILLOFACIAL PAIN

The causes of maxillofacial pain are myriad, and the diagnosis of head and neck syndromes can be exceedingly difficult.[26] Box 26-1 is a partial listing of pain-producing conditions. Fortunately, most of these syndromes are relatively rare. Only the most commonly encountered are covered in this chapter.

Pulpitis

The common toothache is caused by inflammation of the dental pulp. It may be difficult for the victim to identify the offending tooth, because the pain often radiates to the eye or ear region, or is referred from one dental arch to the other. The painful tooth is rarely sensitive to percussion or palpation. An obvious cause, such as a large carious lesion, is sometimes found on examination of the mouth, but often all of the teeth appear intact. If inflammation is mild, the condition is characterized by pain that is elicited only by cold or sweets, and disappears within seconds when the stimulus is removed. Moderate pulpitis is characterized by sensitivity to hot as well as cold, greater discomfort, and an increasing interval between the removal of the stimulus and resolution of the pain. In its most severe form, pulpitis causes intense, continuous, and debilitating pain.[4,6,26] Emergency treatment recommendations are given in the following sections.

Mild Pulpitis (Characterized by Transient Thermal Sensitivity). Examine the mouth visually. Structures will likely appear within normal limits. However, if a defect in a tooth is found, it can be temporarily filled. Reassure the victim that although this condition is annoying, rapid progression is unlikely.

Moderate Pulpitis (Longer Episodes of Pain). Proceed as for mild pulpitis. Treat with a non-narcotic analgesic (ibuprofen 600 mg orally [PO] every 6 hours as needed for pain) and reduce physical activity.

Severe Pulpitis (Intense, Continuous Pain). The preferred approach is pain relief using a local anesthetic, followed by evacuation of the victim.[28,38] A nerve block with bupivacaine 2% with 1:200,000 epinephrine (Marcaine) can provide up to 8 hours of excellent pain relief without central nervous system depression (see section on Local Anesthesia). Large doses of narcotics should not be used because they are likely to compromise the victim's ability to participate in evacuation. Sometimes application of cold (continuously sipping cold water) provides relief to the hyperemic pulp. In an extraordinary circumstance, an experienced rescuer could locate the offending tooth, expose the pulp, remove the inflamed pulpal tissue with a barbed broach, and cover the opening with a temporary filling material. Extraction is also an option in a case of severe pulpitis, but is usually contraindicated for a number of reasons (see section on Exodontia).

Periapical Osteitis

Inflammation of the supporting structures at the root of a tooth is characterized by constant, often throbbing, pain. Unlike pulpitis, the affected tooth is easily located. The victim can usually point to the exact source of the pain, or the examiner may gently

tap individual teeth, observing for tenderness. The area over the apex of the tooth is usually tender to palpation, but there is no frank swelling. Although trauma to a tooth can result in periapical inflammation, the most common cause is egress of bacteria and breakdown products from the necrotic pulp. Minor swelling around the apex extrudes the tooth slightly, causing increased forces on the tooth during occlusion, and thus intensifies the pain. Emergency treatment includes an analgesic (ibuprofen 600 mg PO every 6 hours as needed for pain) or a local anesthetic and a soft diet. Ideally, the contacting areas of the opposing tooth are reduced to relieve occlusal forces.[4,6] Because this usually is impractical in the field, the victim can be given a strip of webbing or something similar to place between the teeth on the nonpainful side. This will keep the offending tooth out of occlusion and reduce the pain.

Cracked Tooth Syndrome

The victim complains of momentary, sharp pain when chewing certain foods, or on releasing the bite. Often, the victim reports that the tooth feels "weak" or that "it only hurts when I bite on something hard just the right way." These symptoms occur when forces of the proper magnitude and direction open the incomplete fracture in the tooth.[30] Significantly, there is no pain on chewing soft foods. This condition usually progresses slowly. The victim should be advised to avoid chewing on the affected side and to seek definitive dental treatment as soon as possible.

Maxillary Sinusitis

The pain of maxillary sinusitis can be referred to a maxillary posterior tooth. It is usually described as a relatively continuous throbbing ache that is intensified by postural change. A typical statement is "My tooth really hurt when we were hiking down that hill. I could feel it pound with every step. When we got to camp, I lay down, but it got even worse." The pain may be unilateral or bilateral. It is usually located in the infraorbital region and is often referred to the cheek, frontal region, and the maxillary premolars and molars. A complaint of multiple toothaches in the maxilla, with no evidence of carious teeth, should immediately raise suspicion for maxillary sinusitis. In addition to pain, there is tenderness elicited by pressure infraorbitally or over the bony prominence above the first molar. The victim also generally has an elevated temperature and nasal or postnasal discharge.

Treatment of maxillary sinusitis includes an analgesic (ibuprofen 600 mg PO every 6 hours as needed for pain), inhalation of steam, oxymetazoline (Afrin) 0.05% 1 spray in each nostril twice a day to shrink the nasal membranes and improve sinus drainage, and an antibiotic. Appropriate choices include amoxicillin 875 mg with clavulanic acid 125 mg (Augmentin) PO twice a day for 10 days or trimethoprim 160 mg with sulfamethoxazole 800 mg (Septra DS) PO twice a day for 10 days. Azithromycin (Zithromax) 500 mg PO the first day and then 250 mg PO for 4 days is a convenient alternative.

Temporomandibular Disorder

Temporomandibular disorder (TMD) is actually a cluster of conditions, multifactorial in origin, and with overlapping symptoms that often respond to a variety of therapies, including placebos.[25] Included under the term TMD are two groups of sufferers: those with primary masticatory muscle involvement (myofascial pain and dysfunction [MPD]) and those with internal derangements of the temporomandibular joint (TMJ). Patients also may present with a combination of both types. A brief summary follows.

Myofascial Pain and Dysfunction

Muscle hyperactivity is an important etiologic factor in MPD. In some persons this may result from parafunction (gum chewing, clenching or grinding the teeth). Occlusal interferences can also cause muscle hyperactivity. This occurs when a lower tooth contacts an upper tooth prematurely during mouth closure and a reflex causes jaw muscle contraction that shifts the mandible in such a way as to avoid the premature contact. For this reason this condition is often referred to as an "occlusomuscle disorder." Psychological stress is also an important factor in causing excessive muscle tension.[22]

Participants in wilderness activities are exposed to many of the risk factors for MPD. The high physiologic and psychological demands of many expeditions lead to considerable stress. Increased jaw function, such as that required to chew granola, jerky, and other dried foods common on wilderness expeditions, is another factor that may precipitate an acute episode of MPD.

Symptoms of MPD include pain in the muscles of mastication that is usually unilateral and increased with chewing, headache, earache, limitation of jaw movement, and a change in the bite. The victim may have a history of acute onset, or a long saga of exacerbation, remission, and various treatments.

The examiner may find objective signs, such as tenderness of the jaw muscles to palpation, muscle spasm or "knots," and abnormal jaw movements, such as inability to open the mouth widely, or deviation of the chin to one side on opening. Tenderness to palpation of the TMJ or joint noise suggest the victim has an internal derangement of the joint.[22]

Emergency treatment consists of resting the muscles (soft diet and control of tooth clenching and grinding habits) and the application of moist heat. Holding a soft material such as a folded gauze between the front teeth often gives immediate relief because it keeps the teeth from touching and allows the muscles to relax. An analgesic should be given on a scheduled basis, rather than as needed, to break the cycle of muscle pain and spasm (ibuprofen 600 mg PO every 6 hours). A muscle relaxant, such as cyclobenzaprine HCl (Flexeril) 10 mg PO three times a day, or sedatives, such as diazepam (Valium) 2 to 10 mg PO three times a day, may be helpful if primary treatment is ineffective. Muscle relaxants and sedatives, especially in higher doses, can cause significant central nervous system depression, so they should be used in the lowest effective dosage and only if more conservative therapy has failed.[7,25,33]

Mandibular Dislocation

Dislocation of the mandible and inability to close the mouth can result from external trauma or sudden wide opening of the mouth, such as occurs with yawning. The condition may be unilateral or bilateral. If there is a history of trauma, a condylar fracture should be suspected (Fig. 26-1). A dislocated mandible is reduced by placing the rescuer's thumbs on the victim's lower molars and moving the mandible down, then posteriorly, and then up. If muscle spasm is severe, sedation might be necessary.[6] After reduction of the mandible, the victim must avoid wide mouth opening. The victim should place one hand under the chin or position the chin against the chest when yawning. If recurrent dislocation occurs, place a bandage around the head and under the chin for several days to limit jaw movement.

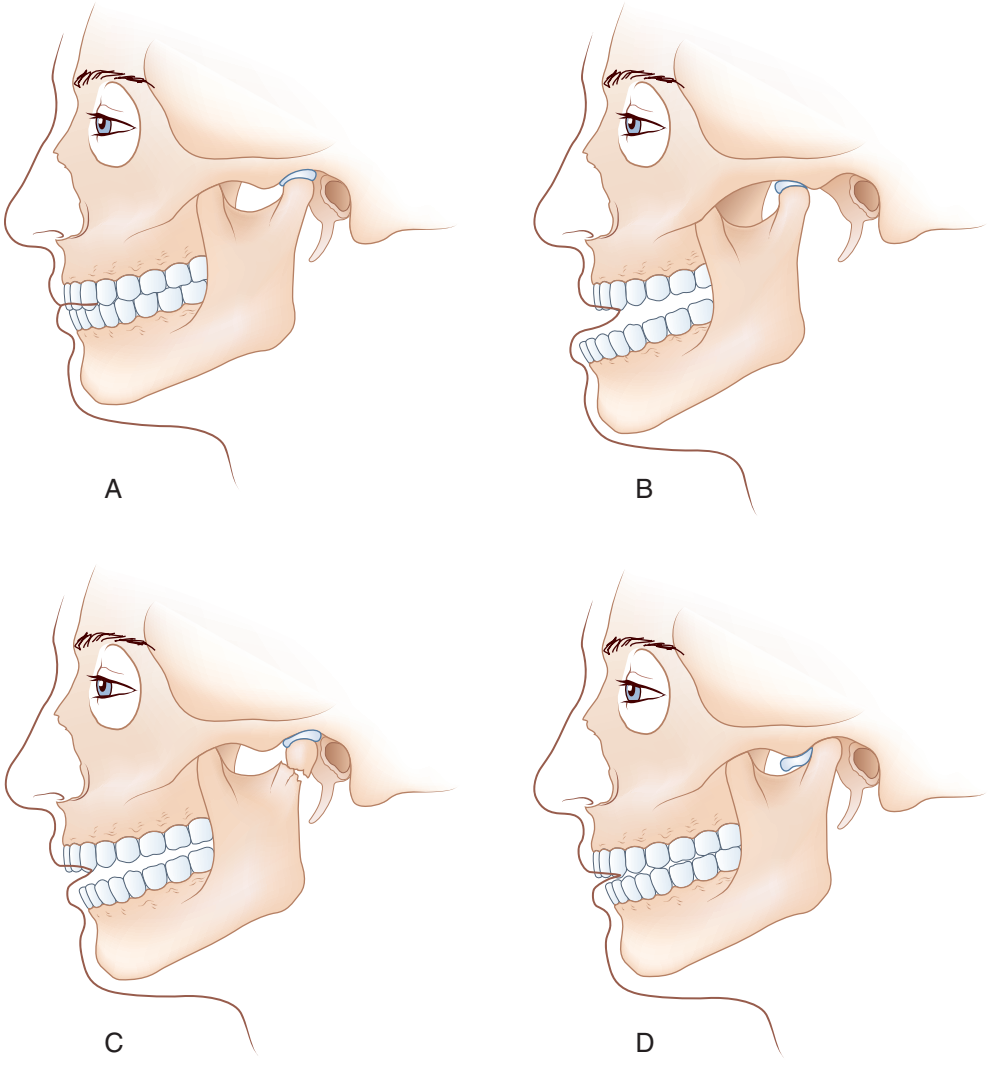


Figure 26-1. Open and closed lock versus condylar fracture. **A,** The normal temporomandibular joint with cartilaginous disc. **B,** In a mandibular dislocation, the condylar head and disc are anterior to the articular eminence. No teeth are touching. **C,** In a condylar fracture, the joint is positioned normally, but the muscles of mastication have pulled the posterior portion of the mandible upward, creating premature contact of the posterior teeth. **D,** In a closed lock, the ligaments have stretched and the disc is trapped anterior to the condyle.

Anterior Disc Displacement

Anterior displacement of the TMJ's cartilaginous disc with reduction to a normal position on mouth opening causes clicking in the joint. Usually this is not associated with pain or severe dysfunction. However, if the disc is displaced and does not reduce on mouth opening, the victim may have not only pain, but limited jaw movement (closed lock). Closed lock can occur suddenly while eating or talking, may be present on awakening, or can be associated with trauma. There is joint tenderness and the chin deviates toward the affected side on attempted mouth opening.[7] Similar restriction of mandibular function may be caused by muscle spasm. However, with myospasm the affected muscles are firm and extremely tender, whereas in closed lock the muscles are usually normal.

The victim can often reduce a closed lock. Have the victim close the mouth until the teeth almost touch, move the mandible as far as possible to the affected side, and finally swing the mouth fully open. If these maneuvers fail after three attempts, consider manual reduction. The rescuer places his or her thumbs on the victim's lower molars, presses downward, pulls the mandible forward, and then gradually moves the mandible up and back.[7] This may require sedation with oral diazepam (Valium), 2 to 10 mg.

MAXILLOFACIAL INFECTIONS

Aphthous Ulcers

The etiology of aphthous ulcers is unclear. One opinion is that they represent an autoimmune attack on the oral mucosa, followed by secondary infection. The lesions are round, superficial, and have a red halo (Fig. 26-2). They occur on movable mucosa and can be quite painful. The victim usually gives a history of similar ulcerations. The lesions typically last 10 to 14 days.

Figure 26-2. An aphthous ulcer.

Many treatments have been proposed, but none has been found predictably effective. The best approach appears to be application of a topical steroid to reduce pain and hasten healing by 3 to 4 days. Mix fluocinonide 0.05% (Lidex) ointment with Orabase and place the mixture gently over each ulcer six to eight times per day, especially after meals and before bedtime. Do not mix the medications until you are ready to apply them, and do not rub the mixture into the lesions. Other options include premixed preparations, such as Kenalog in Orabase, applied to the ulcer six to eight times per day, which are more convenient but deliver only about 10% of the anti-inflammatory effect; dexamethasone (Decadron) elixir (0.5 mg/5 mL, rinse with 5 mL for 2 minutes and expectorate four times a day); or a systemic steroid (prednisone, 40 mg PO once daily for 3 days, then taper) for a very severe case.[35] If these preparations are not available, tincture of benzoin or a topical anesthetic (viscous lidocaine 2%) can be applied to the dried surface of the ulcer before meals and at bedtime to control the pain.

Viral Infections

Herpes labialis (cold sore, fever blister) is the most common oral viral infection. It is characterized by yellow, fluid-filled vesicles that rupture to leave ragged ulcers on the lips. Other locations for recurrent herpetic outbreaks include the palate, tongue, and buccal mucosa. The victim can be given valacyclovir (Valtrex) 2 g PO twice daily for 1 day, with doses taken about 12 hours apart.[34] It is important to begin treatment as soon as the victim becomes aware of a prodromal "tingle" or paresthesia. There is some evidence that the same regimen of valacyclovir can prevent outbreaks from occurring.[5,24] However, more conservative methods, such as the use of sunblocking agents on the lips, are preferred.

Primary herpetic gingivostomatitis is characterized by a thin zone of very red, painful gingiva just next to the teeth. Other areas of mucosa, such as the tongue, may also be involved, and close inspection may reveal tiny vesicles or ulcers. Sore throat, lymphadenopathy, and low-grade fever are also present. This and other viral infections of the oral cavity are self-limited. The victim should be reassured that the condition will resolve in about 10 days. Treatment involves the use of an analgesic (ibuprofen 600 mg PO every 6 hours as needed for pain) and soothing mouth rinses, such as warm saline or a mixture of equal amounts of diphenhydramine (Benadryl) elixir 12.5 mg/5 mL with kaolin-pectin (Kaopectate) and viscous lidocaine 2% (rinse and expectorate 5 mL every 2 hours).

Yeast Infections

Oral yeast infections occur most commonly in persons who are debilitated, immunocompromised, or taking an antibiotic. Classic oral candidiasis (thrush) is characterized by white patches on the mucosa that can be rubbed off, leaving a red and raw surface. Candidiasis can also present as an erythematous mucosa, without any white patches, or as chronic angular cheilitis. Candidiasis is treated with an antimycotic mouth rinse (nystatin oral suspension 100,000 U/mL, rinse with 5 mL for 2 minutes and swallow, four times a day for 10 days) or lozenges (clotrimazole [Mycelex] troche, 1 four times a day, leave in mouth 5 minutes and expectorate remains). In the field, nystatin preparations meant for vaginal treatment (Mycostatin vaginal suppository used as an oral lozenge three times a day for 10 days) can be used.

Bacterial Infections

A bacterial infection in the maxillofacial region can become a serious health threat. In a wilderness setting, it should be taken seriously. Most odontogenic infections are caused by mixed populations of aerobic and anaerobic bacteria. These organisms are almost always present as normal oral flora, but because of a change in the relative numbers of various bacteria or because of a change in the oral environment, an infection can become aggressive.

The behavior of an organism, such as the production of collagenase or hyaluronidase, determines the clinical presentation. Thus, an infection may present as diffuse cellulitis or localize as an abscess. Oral infections usually spread slowly, but rapid spread to a deep facial space can occur. Regional lymphadenopathy is common, whereas severe systemic symptoms are rare. Although bone is often involved, osteomyelitis is uncommon.[31]

Acute Apical Abscess/Cellulitis

An acute apical infection begins with bacteria invading the dental pulp. The infection then spreads to surrounding bone through the apical foramen and then along the path of least resistance. Because the apices of most teeth are located closer to the facial aspect of the jaw, swelling is much more common in the facial soft tissues, as opposed to those on the lingual or palatal side.

The victim presents with pain and swelling, often fluctuant and usually in the buccal vestibule (Fig. 26-3). There is often a history of prior toothache, but at this stage tooth pain is often absent. The offending tooth can be localized by percussion, site of the swelling, condition of the teeth, and by radiographs if available. The affected tooth does not respond at all to hot or cold. The victim may be dehydrated because of decreased fluid intake.

Primary treatment for an apical abscess is drainage.[23,32] This can be accomplished with incision, extraction, or endodontic therapy. The treatment chosen depends on equipment and personnel available, advisability of retaining the offending tooth, and ultimately on clinical judgment. An antibiotic is necessary only if complicating factors exist[8,23,32,36] (Box 26-2). Penicillin

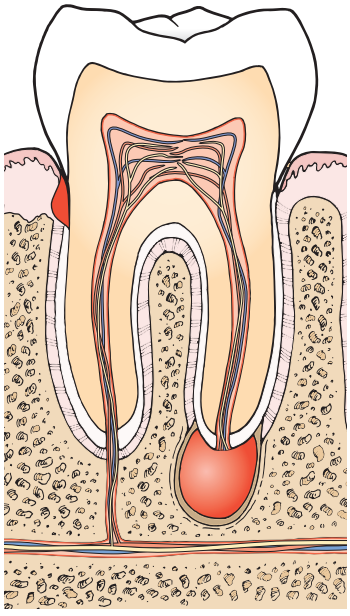


Figure 26-3. The left side of the figure shows a periodontal abscess. A periapical abscess is depicted on the right.

Box 26-2. Indications for Antibiotic Use in Dental Emergencies

Prophylaxis for persons at risk of bacterial endocarditis
Prophylaxis for persons having prosthetic joint implants within the past 2 years
Local infections
- If the victim is immunocompromised
- If drainage cannot be established
- If there will be a long delay before definitive care
- If infection persists after local treatment

Disseminated infections
- Lymphadenopathy
- Fascial plane involvement
- Trismus
- Systemic symptoms (fever, chills, malaise)

Compound maxillofacial fractures, including all fractures of tooth-supporting bone
Exarticulation (avulsion) of teeth
Soft tissue wounds open for 6 hours or more before closure
Surgical procedures under nonsterile conditions

(Pen VK 500 mg PO four times a day) is the most commonly used antibiotic in dental practice, but a cephalosporin, such as cephalexin (Keflex) 500 mg PO four times a day, typically carried on wilderness expeditions, is acceptable. Clindamycin 150 mg PO four times a day is a good alternative.[32,36] Combination antibiotic therapy is not indicated except for life-threatening sepsis or when organisms particularly sensitive to combination therapy have been identified. There is some evidence that hesitation to obtain drainage combined with over-reliance on chemotherapy can lead to serious exacerbation of dental infections.[8]

Incision and Drainage

Incision and drainage (I&D) is often the treatment of choice in an emergency situation. An I&D can be performed by a non-dentist using commonly available supplies. It is indicated for fluctuant swelling due to an apical abscess and may also be effective for nonfluctuant swelling associated with infection. Infiltration of a local anesthetic helps to reduce the pain of incision. If unavailable, adequate anesthesia can often be obtained by applying cold to the area to be incised, using ice, snow, or frigid water. An incision is made down to bone in one swift motion and a knife handle or the beak of a hemostat is used to spread the incision. A T-shaped drain may be improvised from a piece of latex glove (Fig. 26-4) and retained without sutures. Hydration, a soft diet, an analgesic (ibuprofen 600 mg PO every 6 hours as needed for pain), and warm saline rinses are helpful postoperative measures.

Deep Fascial Space Infections

An acute apical infection occasionally spreads beyond the local region. The rescuer should be suspicious of any dental infection that causes swelling or tenderness in the floor of the mouth, swelling of the tongue, dysphagia, breathing difficulty, or trismus, or that fails to respond to appropriate therapy. The most commonly involved fascial spaces are the canine, buccal, masticator, and submandibular spaces.[9]

The canine space is located lateral to the nose. Infection originates in the maxillary canine tooth and swelling causes the eye to close. The infection is drained through an intraoral approach over the root of the canine tooth.

Buccal space infection is characterized by rounded swelling of the cheek. The offending teeth are the maxillary and mandibular molars. Drainage is obtained extraorally through an incision below the lower border of the mandible, so that the scar will eventually be hidden or minimal.

The masticator space is divided into the masseteric, pterygoid, superficial temporal, and deep temporal spaces, all of which communicate. The hallmark of involvement is trismus. Swelling may be minimal owing to the deep location of the abscess. The masseteric and pterygoid spaces are drained at the angle of the mandible, whereas the temporal space may be drained from an intraoral approach or through an incision just superior to the zygomatic arch.

The mylohyoid muscle divides the floor of the mouth into the sublingual and submandibular spaces. These communicate posteriorly, and with their counterparts across the midline. An infection originating in a mandibular tooth can involve these spaces. If only the sublingual space is infected, an intraoral approach is used for drainage, avoiding damage to Wharton's duct. If the submandibular space is involved, an extraoral approach is used. All incisions in the facial area are made parallel to branches of the facial nerve.[9,21]

Fascial space infections are potentially life-threatening. The likelihood of widespread sepsis increases, cavernous sinus venous thrombosis and mediastinitis are possible, and the airway may become compromised.[10,16] The most feared infection is Ludwig's angina, a bilateral submandibular space infection that elevates the tongue, obstructs breathing, and is associated with a high mortality rate (Fig. 26-5). A mild dental

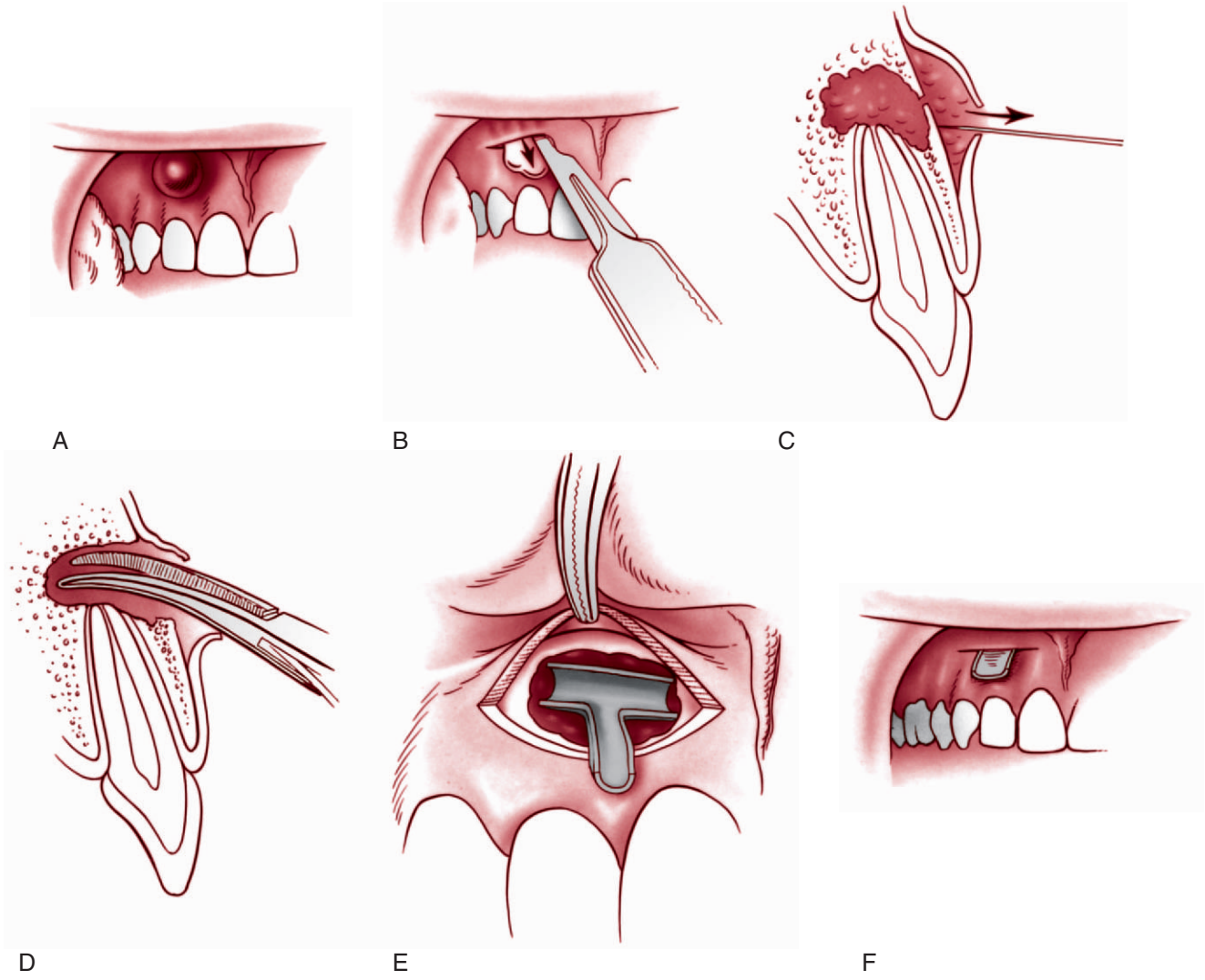


Figure 26-4. Incision and drainage technique. **A,** Fluctuant abscess. **B,** Abscess incised with scalpel. Purulent drainage is removed by suction or caught in gauze sponges. **C,** Cross section showing incision carried to the bone. **D,** The incision is spread with a hemostat. **E,** A "T" drain often stays in place without sutures. **F,** Drain in place. (Redrawn from Ingle JI, Beveridge EE: Endodontics, 2nd ed. Philadelphia, Lea & Febiger, 1976, with permission.)

infection can progress to a life-threatening emergency in as little as 48 hours.[16]

Treatment of fascial space infections includes airway management, proper hydration and electrolyte balance, aggressive incision and drainage, intravenous antibiotics, and pain control. Because these objectives are best met in a controlled environment, any person with a suspected fascial space infection should be evacuated immediately.

Chronic Apical Abscess

The hallmark of chronic apical infection is a draining fistula or "gum boil." Because the bacteria have a route to escape, they do not cause pressure or pain, although the tooth may be mildly sensitive when eating. Such an abscess is not an emergency except in the unlikely event of an acute flare-up.

An infected deciduous tooth also usually presents with a fistula, and a mild amount of swelling at most. Happily for the child, severe pain is rarely present. Emergency care involves an antibiotic, with the dosage adjusted for the child's weight, and I&D only if the abscess is not draining. Extract the tooth only if it is very loose and it will be weeks until comprehensive care is available.

Periodontal Abscess

A periodontal abscess is an accumulation of pus between the gingiva and the tooth (see Figure 26-3). Swelling is near the gingival margin rather than in the vestibule, as would be the case with a periapical abscess. The tooth is sensitive to percussion, but responds appropriately to hot and cold. There is always a potential communication between the abscess and the mouth.

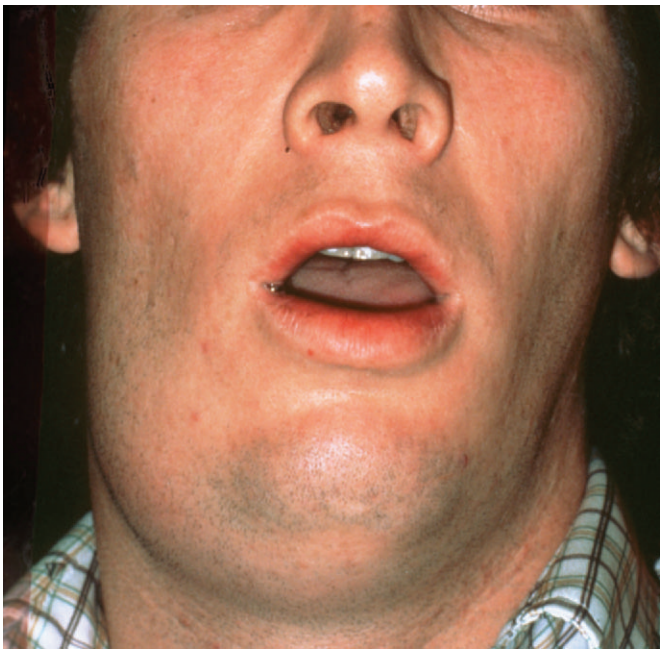


Figure 26-5. Ludwig's angina.

Box 26-3. Factors to Consider before Extraction of Teeth

- Desires of the victim
- Victim's medical history
- Available alternative treatments
- Difficulty/desirability of evacuation
- Certainty of diagnosis (Are you sure you have the correct tooth?)
- Possible complications if the tooth is not extracted
- Factors relating to the difficulty of the procedure
  - Mobility or immobility of the tooth
  - Position of the tooth
  - Condition of tooth structure above the gingiva
  - Patient's mouth-opening ability
  - Available supplies and instruments
  - Experience of rescuers
- Possible complications arising from the extraction
  - Fractured root(s)
  - Fractured alveolus
  - Soft tissue injury
  - Root tip lost in the sinus
  - Prolonged bleeding
  - Localized osteitis (dry socket)

The passage can be found by probing the gingival margin with a small, blunt instrument, using a local anesthetic if available. Gentle probing establishes drainage; no incision is necessary. Hot saline rinses are prescribed and a rapid recovery is almost invariable.

Pericoronitis

Pericoronitis is an infection of the gingival flap over a partially erupted tooth. The most common site is the mandibular third molar. Pericoronitis is usually caused by streptococci and seldom produces purulence. The condition may mimic streptococcal pharyngitis or tonsillitis. The primary site of infection is always tender, and trismus is a common sign. Field treatment consists of saline irrigation of the space under the flap using a syringe. Place the victim on hot saline rinses every 2 hours, and begin antibiotic therapy.

Acute Necrotizing Ulcerative Gingivitis

Acute necrotizing ulcerative gingivitis (ANUG), also known as *trench mouth*, is characterized by ulceration and blunting of the interdental papillae. The gingiva between the teeth appears punched out, and is covered by a gray/white pseudomembrane, whereas the surrounding gingival tissue is very red. The victim's primary complaint is pain, but he or she may also report gingival bleeding, metallic taste, and foul odor. This infection is caused by fusiform bacteria and spirochetes. It usually occurs in young adults with poor oral hygiene, stress, and suboptimal nutrition (conditions that may be present during difficult wilderness expeditions). The most important treatment is gentle debridement of plaque, calculus, and food from around the teeth. Resolution may require several sessions of careful cleaning 1 to 2 days apart. Each session of debridement results in some healing, which allows more aggressive treatment the next time. The victim is also given an antibiotic (metronidazole [Flagyl] 500 mg PO four times a day) and analgesics (ibuprofen 600 mg PO every 6 hours as needed for pain), and instructed to keep the area clean with good brushing, flossing, and rinsing (warm saline or diluted hydrogen peroxide, if available).[4]

EXODONTIA

A dental extraction is considered definitive treatment, and should be attempted in the field only under extraordinary circumstances (Box 26-3). Extraction requires trained personnel, specialized instruments, and profound anesthesia, which may be difficult to obtain. Premedication with a sedative or narcotic may be necessary, as may dental radiographs. Intraoperative and postoperative complications are common. Providers of emergency care should focus on treating pain and infection with local anesthetics, analgesics (ibuprofen 600 mg PO every 6 hours as needed for pain, or hydrocodone 7.5 mg and acetaminophen 750 mg [Vicodin ES] PO every 4 to 6 hours as needed for pain), I&D, and antibiotics (see section on Acute Apical Abscess/Cellulitis) as appropriate for each case.[4,28,32] Extraction or other definitive care can then be rendered after evacuation, and the tooth can often be saved.

Other factors to consider before exodontia are degree of mouth opening possible, relationship of the root to the maxillary sinus, condition of the clinical crown, alignment of the tooth in the dental arch, and history of previous endodontic treatment (after root canal treatment, a tooth is usually very brittle).[20] If, after careful consideration of all factors, extraction is deemed the best course of treatment, proceed as follows.

Review the victim's medical history. In a wilderness setting, you are unlikely to be faced with a case complicated by a blood dyscrasia, anticoagulant therapy, severe cardiovascular disease, and the like, but you must be certain before the surgery begins. Persons with most types of heart murmurs or recent prosthetic

Box 26-4. Recommendations for Antibiotic Prophylaxis before Intraoral Procedures Likely to Cause Significant Bleeding

CONDITIONS FOR WHICH PREMEDICATION IS RECOMMENDED

Prosthetic joint replacement *if* first 2 years after placement, victim has type 1 diabetes, previous prosthetic joint infection, or hemophilia
Prosthetic heart valve replacement
Previous endocarditis
Complex cyanotic congenital heart disease
Surgically constructed systemic pulmonary shunts or conduits
Most congenital cardiac malformations other than those listed below
Acquired valvular dysfunction (e.g., rheumatic heart disease)
Hypertrophic cardiomyopathy
Mitral valve prolapse with valvular regurgitation or thickened leaflets

CONDITIONS FOR WHICH PROPHYLAXIS IS NOT RECOMMENDED

Isolated secundum atrial septal defect
Surgical repair of atrial septal defect, ventricular septal defect, or patent ductus
Previous coronary artery bypass graft surgery
Mitral valve prolapse without valvular regurgitation
Physiologic, functional, or innocent heart murmurs
Previous rheumatic fever without valvular dysfunction
Cardiac pacemakers and implanted defibrillators
Coronary artery stents

Box 26-5. Regimens for Antibiotic Prophylaxis before Intraoral Procedures Likely to Cause Significant Bleeding

STANDARD REGIMEN

Amoxicillin 2 g PO 1 hour before procedure

CHILDREN

Amoxicillin 50 mg/kg PO 1 hour before procedure, not to exceed adult dose

UNABLE TO TAKE ORAL MEDICATIONS

Ampicillin 2 g IM/IV 30 minutes before procedure

PENICILLIN ALLERGY (CHOOSE ONE OF THE FOLLOWING)

Clindamycin 600 mg PO 1 hour before procedure
Clindamycin 600 mg IV 30 min before procedure
Clindamycin 20 mg/kg PO 1 hour before procedure (for children)
Clarithromycin 500 mg PO 1 hour before procedure
Cephalexin 2 gm PO 1 hour before procedure

From Dajani AS, Taubert KA, Wilson W, et al: JAMA 277:1794–1801, 1997, with permission.

implants should be premedicated with an antibiotic[11] (Boxes 26-4 and 26-5). Consider the emotional state of the victim and decide if sedation is prudent.

Plan the procedure and gather all necessary equipment. Secure a good light source and a means to keep the field dry—either suction or plenty of gauze. It is important to have the victim's head supported. Therefore, it may be best to place the victim in a supine position with the head slightly elevated. Obtain good local anesthesia and test by touching the soft tissues with a sharp instrument. A 10 × 10-cm (4 × 4-inch) gauze curtain placed in the rear of the victim's mouth prevents aspiration of teeth and debris.

Teeth are inflexible and brittle. Heavy forces, especially if applied quickly (high acceleration), break teeth. Bone has more flexibility, the degree of which depends on the individual patient. Judiciously applied, moderate forces slowly expand the bone. The tooth will eventually be delivered in the same direction as if it continued to erupt. However, do not attempt to "pull" the tooth in that direction initially. The direction of force needed to loosen a tooth depends on the anatomy of the root. A straight, conical root can be loosened by twisting forces. This technique often works well on the upper front teeth. Sometimes a tooth can be removed by alternating 30 seconds of steady pressure toward the cheek with 30 seconds of steady pressure in the opposite direction, until the root gradually loosens.[42]

A variety of instruments can be used to apply force to the tooth. Elevators are firmly wedged between tooth and bone in the interproximal area. Avoid putting pressure on the adjacent tooth. Forceps should be applied as far apically as possible. Spend plenty of time working the forceps well under the gingiva. Lower molars are often removed with "cowhorn" forceps, which, in addition to applying the usual forces, are designed to apply force to the tooth in a coronal direction simply by squeezing the handles. However, an experienced exodontist using careful technique and a single forceps (the no. 151 universal extraction forceps) will have success extracting almost any nonimpacted tooth, whereas a reckless operator using dozens of specialized instruments will still have difficulty.[20,42]

Despite the utmost care, a tooth may break during the extraction procedure. If this occurs, stop and take a minute to reevaluate the situation. The root canal will probably now be exposed. If you are treating a case of infection, perhaps drainage can be obtained through the root canal. If you are treating a case of severe pulpitis, perhaps the pulpal tissue can now be removed. Remember that you are rendering emergency care, and it may not be necessary to remove the remaining root at that time. Some teeth are impossible to remove without sectioning or can be removed in only one direction.

Once the tooth has been removed, compress the expanded socket using the thumb and forefinger. If the gingiva is quite loose, placing a suture may help speed healing. Have the victim apply direct pressure to the wound by biting firmly on a gauze pack for 30 minutes while sitting in an upright position. Complete hemostasis may require several hours of steady pressure. Caution the victim to avoid rinsing, spitting, tooth brushing, and smoking for 24 hours. Application of cold and the use of an appropriate analgesic reduce swelling and pain. Beginning the day after surgery, have the victim rinse with warm saline to cleanse the area.

A common postoperative complication is persistent bleeding several hours after the extraction, accompanied by a poorly organized clot that looks like a piece of raw liver growing out

of the socket. Remove the "liver clot" and have the victim apply firm, uninterrupted pressure to the socket by biting on a gauze pack for 20 minutes. If the victim cannot do this, the rescuer will have to apply manual pressure. If bleeding continues after 20 minutes, consider packing the socket (Gelfoam, Surgicel, or sterile gauze) or suturing. Then resume direct pressure. A dry teabag used as a compress may provide chemical hemostasis because of tannic acid as well as direct pressure.

Another postextraction complication is acute alveolar osteitis, or "dry socket." The victim reports moderate to severe pain, foul odor, and a bad taste, beginning about 3 days after a dental extraction. Examination reveals an empty socket and exposed bone due to loss of the blood clot, but no suppuration. Treatment consists of gentle irrigation with warm saline followed by packing with a strip of gauze dipped in eugenol. Administer an oral analgesic. Change the pack every 24 to 48 hours until the symptoms subside, which may take up to 10 days. The victim should avoid alcohol, smoking, and carbonated beverages during treatment.

TRAUMA TO THE FACE AND JAWS

Wilderness recreation creates many opportunities for traumatic injury. One recent survey of 9543 consecutive trauma patients registered with an oral and maxillofacial surgery service found that 31% were sports related. Of those, 44% could be considered "backcountry" trauma, mostly resulting from skiing and snowboarding. The study was done in Innsbruck, Austria.[40]

It is essential quickly to evaluate the general condition of an injured victim[17] (see Chapter 20). The primary survey identifies and corrects any inadequacy in respiration or circulation. The mouth and pharynx should be examined for foreign bodies such as blood clots, tooth or bone fragments, or dentures. If the airway is still obstructed, it may be necessary to perform a chin lift or jaw thrust, or insert an oropharyngeal airway to hold the tongue forward (see Chapter 19). Care should be taken not to hyperextend the neck because of the possibility of a cervical spine fracture. If there is no indication of a neck fracture, sometimes merely placing the victim on his or her side rather than in a supine position facilitates respiratory exchange. If all these measures fail, endotracheal intubation or performance of a cricothyrotomy or, less commonly, a tracheotomy may be necessary. Once a victim's airway is secured and he or she is hemodynamically stable, perform a secondary survey.

Injuries to the Teeth and Supporting Tissues

Question the victim about the time and nature of the accident, symptoms, loss of consciousness, nausea, vomiting, visual disturbances, and headache. A baseline mental status examination and cranial nerve assessment may be warranted. Clean the face, mouth, head, and neck of blood and debris. This unmasks soft tissue injuries and facilitates diagnosis. Gently reflect the lips with teeth closed to examine soft tissues and occlusion. Examine any lacerations carefully to determine if they penetrate through the lip or contain foreign material. Examine all of the teeth for fractures. A blow to the chin or whiplash injury may produce fractures of the posterior teeth as the mandible is forcibly closed. Examine fractured teeth carefully for pulp exposure. This will require drying the tooth with gauze. Tap each tooth with an instrument handle. Tenderness denotes injury to the periodontal ligament, whereas a high-pitched, metallic sound indicates ankylosis as occurs in lateral luxation or intrusion. Test each tooth for abnormal mobility. Electrical pulp vitality testing, dental radiographs, and soft tissue radiographs are obtained if available.[2,14,16]

Box 26-6. Classification of Dental Trauma

INJURIES TO THE HARD DENTAL TISSUES AND PULP
Crown infraction
Uncomplicated crown fracture
Complicated crown fracture
Uncomplicated crown-root fracture
Complicated crown-root fracture
Root fracture

INJURIES TO THE PERIODONTAL TISSUES
Concussion
Subluxation
Intrusive luxation
Lateral luxation
Extrusive luxation
Exarticulation

INJURIES TO THE SUPPORTING BONE
Comminution of alveolar socket
Fracture of alveolar socket wall
Fracture of alveolar process
Fracture of jaw

INJURIES TO SOFT TISSUES

From Andreasen JO, Andreasen FM: Textbook and Color Atlas of Traumatic Injuries to the Teeth, 3rd ed. St. Louis, Mosby, 1994, with permission.

A classification of traumatic injuries to the teeth and supporting structures is given in Box 26-6. Note that these injuries often occur in combination, with one tooth exhibiting two or more injuries, or several teeth exhibiting various sequela of trauma. Each injury requires definitive treatment, but proper emergency care improves the prognosis and makes the victim more comfortable. Treatment of most injuries requires, or is at least facilitated by, infiltration of a local anesthetic.

Specific Dental Injuries

Crown Infraction. Blows to the teeth sometimes produce small craze lines in the enamel. These superficial fractures look like tiny surface cracks on an old porcelain dish. Reassure the victim that minimal damage has occurred and treatment is not necessary.

Uncomplicated Crown Fracture. The tooth has been fractured, but no pulp tissue is visible. The tooth may be sensitive to cold, but otherwise all test results are within normal limits. Account for and save the missing fragment if possible. No emergency treatment may be necessary. Irritating sharp edges can be smoothed with a fingernail file. If thermal sensitivity is moderate to severe, a soothing topical anti-inflammatory dressing (Intermediate Restorative Material [IRM], L. D. Caulk Co., Milford, DE) can be held in place with aluminum foil or adhesive tape.

Uncomplicated Crown–Root Fracture. Diagnosis and treatment of this condition are identical to the uncomplicated crown fracture, except that the fracture is nearly vertical, leaving a small, chisel-shaped fragment attached only by the palatal gingiva (Fig. 26-6). Removal of this mobile fragment makes the victim much more comfortable.

Complicated Crown Fracture. In this injury, the pulp has been exposed. A small exposure that has not been grossly contaminated is capped with calcium hydroxide (Dycal, L. D. Caulk Co.) or IRM. If the exposure is larger than 1 mm or if the pulp tissue has been exposed for more than 24 hours, amputate approximately 2 mm of tissue with a sharp, sterile instrument. If bleeding continues for more than a few minutes, use a cotton pellet soaked in local anesthetic solution, hydrogen peroxide, or Dycal to obtain hemostasis. Fill the top of the canal with Dycal or IRM and protect the tooth as for a crown fracture (Fig. 26-7).

Complicated Crown–Root Fracture. The tooth has been fractured obliquely, resulting in a mobile fragment attached to the palatal gingiva as well as a pulp exposure. First, remove the mobile fragment as in a crown-root fracture, then treat the pulp exposure as in a complicated crown fracture.[2] It is possible to glue tooth fragments together with 2-octyl cyanoacrylate (Dermabond) for a day or two.[19a]

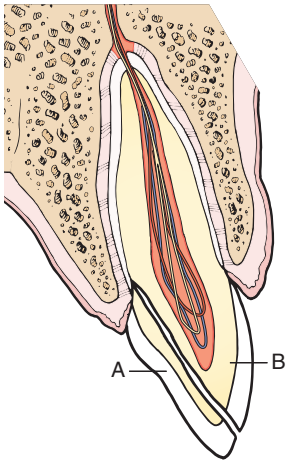


Figure 26-6. Crown-root fracture. **A,** The mobile fragment should be removed. **B,** The larger fragment should be left in place.

Root Fracture. A root fracture may be difficult to diagnose without radiographs. There is slight to severe malposition of the crown, but this could be caused by extrusion of the entire tooth or a root fracture with luxation of the coronal portion. Reposition the tooth as precisely as possible and splint rigidly (see section on Splinting, later). Hard tissue union of the fragments usually occurs within 3 months. If the coronal portion of the tooth proves impossible to stabilize and definitive treatment is days away, remove the mobile fragment, but do not attempt to extract the apical fragment.[2,14,15]

Concussion and Subluxation. Injuries to the tooth's supporting structures (periodontal ligament, bone, and gingiva) cause sensitivity to percussion. The tooth remains in its proper position. In subluxation the tooth is abnormally mobile, whereas mobility is normal with concussion. Hemorrhage from the gingival crevice sometimes occurs. Emergency treatment consists of shortening the opposing tooth so that the victim can occlude comfortably.

Intrusion. A tooth that has been driven into the bone by a vertical force demonstrates little mobility and a high-pitched, metallic tone on percussion. Emergency treatment is palliative only. Endodontic treatment (to prevent inflammatory root resorption) and orthodontic extrusion should begin within 2 weeks of injury. Intrusion is associated with a poor long-term prognosis.[2]

Extrusion. The extruded tooth is partially displaced from its socket and extremely mobile. Gentle, steady pressure is used to reposition the tooth, allowing time to displace the blood that has collected in the apical region of the socket. After reduction, the tooth is nonrigidly splinted for 2 to 3 weeks.

Lateral Luxation. In this injury, the tooth is often displaced by a horizontal blow, yet it is not mobile because the apex is locked into its new position in the alveolar bone. A high, metallic tone on percussion is another clue that this has occurred. This injury and its treatment are painful. Figure 26-8 shows how two

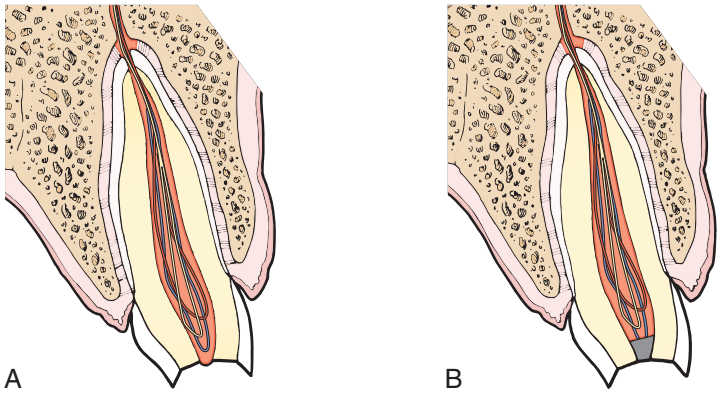


Figure 26-7. **A,** Complicated crown fracture. **B,** Treatment of a complicated crown fracture.

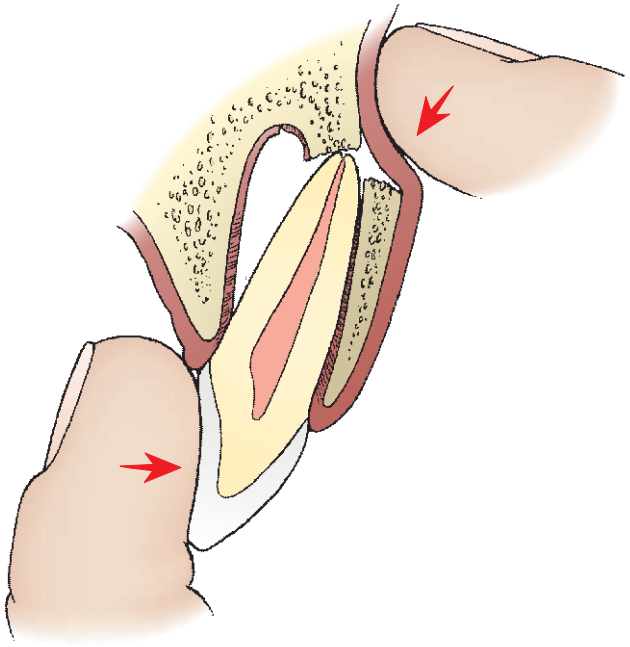
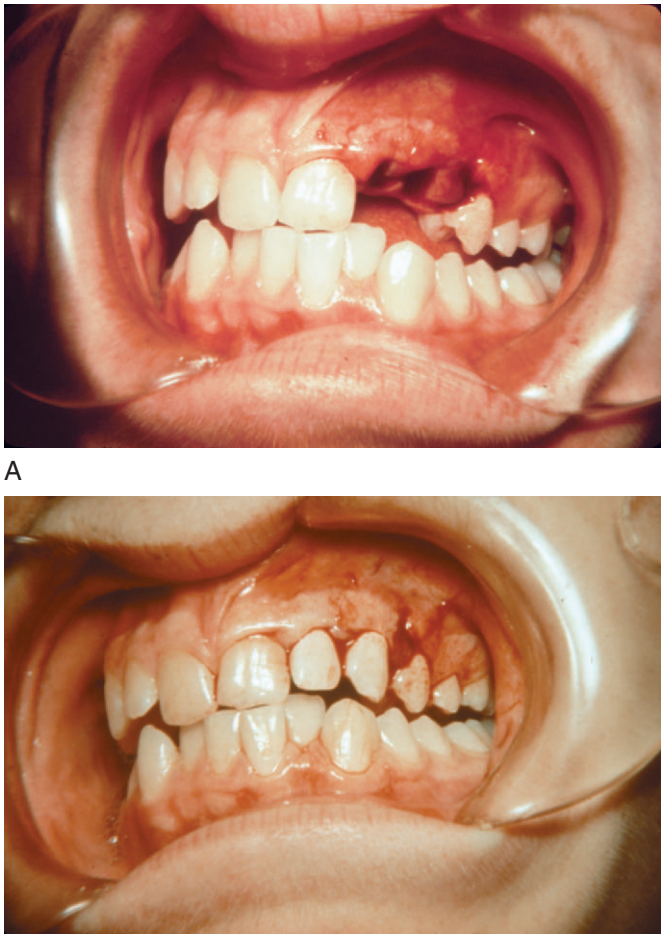


Figure 26-8. Technique used to reduce lateral luxation.

fingers are used to reduce the tooth. One finger guides the apex down and back while the other repositions the crown.[2] This requires judicious, but sometimes quite firm, application of force. The tooth may snap back into position and be quite stable. Check the tooth the next day. Breakdown of marginal bone and inflammation may cause the tooth to become mobile. Splinting is necessary if mobility is present, whether this occurs immediately after reduction or is detected later.[15]

Exarticulation. When a tooth is totally avulsed from bone, the prognosis after replantation depends on the health of the periodontal ligament cells, some of which are still attached to the root surface and some of which line the socket wall.[12,29] To preserve the vitality of these cells, certain guidelines should be observed. Keep the time before replantation to a minimum. Immediate replacement is ideal (Fig. 26-9). If the tooth must be stored, keep it moist. Less than 20 minutes in air is associated with a good prognosis and longer than 1 hour with a poor prognosis.[2,12] If possible, the tooth should be stored in a Save-A-Tooth (Smart Practice, Phoenix, AZ), which uses a soft mesh to suspend the tooth in Hanks' balanced salt solution. Alternative transport mediums are whole milk, saline, the victim's saliva, sports drinks, and water, in that order of preference. The concept is to preserve the health of the periodontal ligament cells in the most isotonic, pH-balanced solution at hand. If no container is available, use a plastic bag, plastic wrap, or saturated cloth to keep the tooth from drying. Handle the tooth by the crown only. Never scrub, curette, or use a disinfectant on the root surface; gently rinse with saline to remove debris. When removing clotted blood from the socket, use gentle irrigation and suction, and avoid scraping the socket walls. Examine the socket for fracture of the alveolar wall. If present, reduce the fractured bone with a blunt instrument. Ease the tooth back into place with slow, steady pressure. After replantation, splint the tooth nonrigidly for 1 week. Suture any gingival lacerations after splinting. Administer an antibiotic for 5 days and give appropriate antitetanus prophylaxis. Endodontic therapy should be instituted within 2 weeks.[2,29,39]



A

B

Figure 26-9. **A,** Two teeth have been avulsed. **B,** The teeth have been replanted.

The preferred approach to tooth avulsion or severe luxation is immediate reduction in the field, followed by evacuation for definitive treatment. The next most desirable option is to store the tooth properly and transport victim and tooth for replantation within a few hours of injury. If the extraoral dry time exceeds 60 minutes, the periodontal ligament cells are presumed necrotic and should be removed by curettage. The root is then treated before replantation. Sodium fluoride 2.4% with a pH of 5.5 has been shown to improve prognosis, as have enamel matrix proteins (Emdogain).[39]

If field conditions prevent all of the aforementioned treatments, a delayed replantation procedure is indicated. Store the tooth dry. Three weeks after the injury, the necrotic pulp tissue is removed and the tooth is disinfected, fluoridated, and surgically replanted. The aim of delayed replantation is to produce ankylosis between tooth and bone.[2]

Alveolar Segment Fracture. This condition is characterized by displacement of two or more teeth as a unit. The teeth are not mobile with respect to one another. The apices may be locked into their abnormal position as in lateral luxation. The segment is repositioned (this may be painful even with local anesthesia) and rigid splinting is placed for 4 to 6 weeks.

Patient Instructions

After all of the preceding intraoral injuries, the patient should be placed on a soft diet for 10 to 14 days and instructed to brush gently but thoroughly with a soft toothbrush after every meal. The patient should also rinse twice each day with a chlorhexidine-containing mouthwash if available, or warm saline if not.

Proper Reduction

It is sometimes difficult to know when a displaced tooth has been returned to its proper position. Usually a tooth should be positioned similarly to its contralateral mate. Sometimes asking the victim can help; it may be that one tooth has always been longer than the others, or in a different position. However, occlusion is always the best guide to proper position. If the victim bites and contacts only the injured tooth, further positioning is necessary. In many cases, it is not possible completely to reposition the tooth because of swelling or organized clot formation. Therefore, some adjustment of the opposing tooth (try using an emery board or pocket knife file) may be necessary to allow proper occlusion. If the injured tooth receives additional trauma each time the victim bites, it will be uncomfortable and healing will not occur.

Splinting

When the goal of splinting is to establish a normal, fibrous union between the tooth and the bone, a short-term, nonrigid technique is used. When hard tissue union is desired (e.g., root fracture or alveolar segment fracture), longer-term rigid splinting is used.[2] Ideally, a single avulsed or loosened tooth is usually bonded to the adjacent tooth or teeth with the acid etch and composite technique.[2,15] For fractures of the jaws or alveolar segments, arch bars and wire splints are often used. The rescuer lacking adequate materials must use ingenuity and improvisation to splint teeth. During a very short evacuation, the victim could hold the tooth in approximate position by closing (biting gently) on a gauze pad. Softened wax can be adapted to a loosened tooth and the teeth on either side to lend support. Figure 26-10 shows how a suture can be used to hold a tooth in place for a period of a few days. For a sturdier splint, a crude arch bar can be cut from a SAM splint or made from a paper clip, and dead-soft wire obtained from copper wiring or twist ties.

Injuries to Primary Teeth

Injuries to the deciduous teeth offer unique challenges, not the least of which is behavioral management of a young child. Splinting is very difficult because of the small amount of tooth structure available. In general, heroic efforts should not be made to save primary teeth.[2,14] Exarticulated deciduous teeth should not be replanted. Severely extruded teeth, infected teeth, or those intruded into the developing permanent tooth should be extracted. Because the permanent tooth follicle lies to the lingual side of the primary tooth root, the typical frontal impact displaces the crown toward the palate, but levers the root apex away from the permanent tooth (Fig. 26-11). Most minor subluxations and luxations require only symptomatic treatment. As long as the displaced tooth does not interfere with occlusion, reduction is contraindicated. Spontaneous repositioning often occurs over a period of weeks. Fragments of primary roots need not be extracted because normal resorption will still occur.

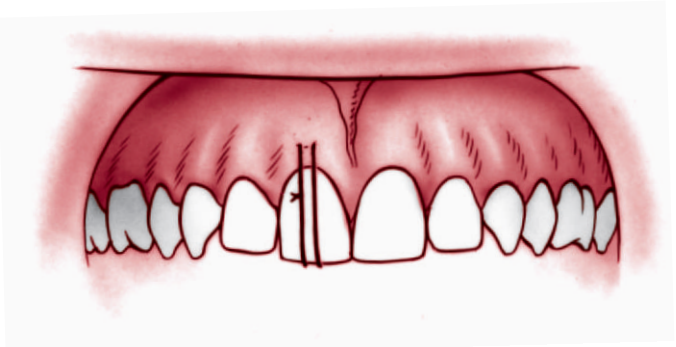


Figure 26-10. Suture used to stabilize a loose or avulsed tooth. The suture also passes through the palatal tissue (hidden from view).

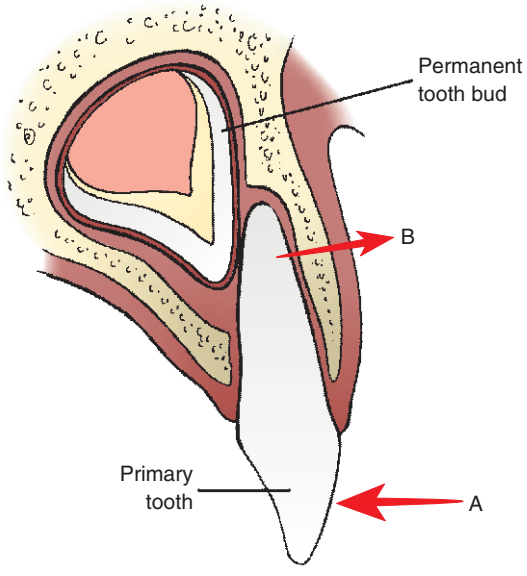


Figure 26-11. When a deciduous tooth is traumatized, the typical direction of force as the child falls forward is shown by *arrow A,* and the apex of the deciduous tooth is levered away from the developing tooth bud, as shown by *arrow B*.

Epistaxis

Although most cases of nosebleed are trivial, some can become life-threatening owing to respiratory compromise secondary to aspiration of blood, or extensive blood loss resulting in hypotension. Therefore, the condition should never be neglected. The nasal mucosa is laced with numerous superficial blood vessels that serve to warm and humidify inspired air. A particularly rich collection of vessels, and common site of anterior nosebleed, is Kiesselbach's plexus on the nasal septum (Fig. 26-12). Spontaneous epistaxis is more common in environments that are cold, dry, dusty, or smoke-filled.

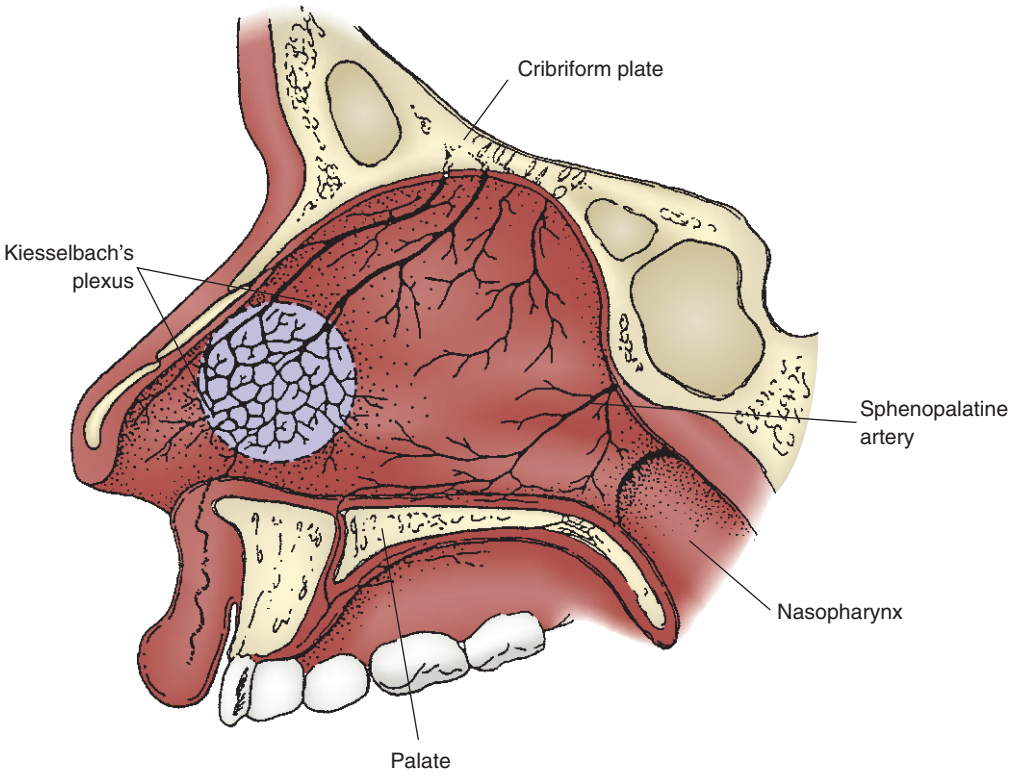


Figure 26-12. Nasal anatomy. Kiesselbach's plexus is the most common site of anterior epistaxis. The sphenopalatine artery is the most common site of posterior epistaxis. Insert tubes and instruments in the direction of the nasopharynx, not the cribriform plate. (From Fleisher GR, Ludwig S: Textbook of Pediatric Emergency Medicine. Philadelphia, Lippincott Williams & Wilkins, 1999, with permission.)

In evaluating a victim with epistaxis, first determine if the bleeding is unilateral or bilateral, and whether it is coming from an anterior or posterior site.[1] A nosebleed usually occurs on one side of the nasal cavity. However, in a victim with profuse bleeding, blood can pass behind the nasal septum and also appear on the unaffected side. Most victims bleed from an anterior site, which can be visualized on intranasal examination. Posterior epistaxis is usually caused by traumatic injury to the sphenopalatine artery (see Figure 26-12). The bleeding point cannot be seen on intranasal examination.

The first step in treating a person with a nosebleed is to have him or her sit upright with the head tipped slightly forward so that blood will drip passively out of the nose rather than flow posteriorly into the throat, causing choking and possible aspiration, or vomiting of swallowed blood. The victim is asked to blow the nose to remove any clots. If suction and a nasal speculum are available, the nasal cavity can then be examined to determine the site of bleeding. When there is minor bleeding from the anterior aspect of the nose, the victim can be instructed to pinch the nostrils together for at least 10 minutes. Hold the soft tissues tightly against the septum. Pinching the bony bridge of the nose will not provide direct pressure on the bleeding vessels.

If nose pinching does not stop the bleeding, apply a topical vasoconstrictor. Choices include cocaine 4%, ephedrine 5%, aqueous epinephrine 1:1000, phenylephrine 0.5% (Neo-Synephrine), or oxymetazoline 0.05% (Afrin). Vasoconstrictors can also be found in some local anesthetics, asthma medications, and southwestern U.S. desert plants of the genus *Ephedra* (Mormon tea, joint-fir, canatilla, tepopote). Vasoconstrictors can be applied by drip, spray, on a cotton pledget, or with a cotton-tipped applicator. Objects placed in the nose should have a string attached or other method of easy removal, and should be aimed posteriorly. Avoid pushing material laterally into the turbinates or superiorly toward the cribriform plate (see Figure 26-12). Leave the vasoconstrictor in place for 10 minutes to 24 hours.

When there is more vigorous anterior nasal bleeding, nose pinching or the topical application of a vasoconstrictor may not be effective. In such an instance, the bleeding site can be injected with 0.5 to 1 mL of lidocaine containing 1:100,000 epinephrine, which has a tamponading as well as a vasoconstrictive effect. Alternatively, a small piece of oxidized regenerated cellulose (Oxycel or Surgicel), gelatin sponge (Gelfoam), or microfibrillar collagen (Avitene) can be placed directly over the bleeding site. Once the bleeding has stopped, the victim should be instructed not to blow the nose or probe the area for 48 hours. Increasing the humidity of inspired air may help prevent a recurrence of the bleeding. Placing the victim in a tent with several other persons, or with a pot of boiling water, and placing a handkerchief over the victim's nose while outside, will humidify and warm the air before it reaches the nasal mucosa.

If the bleeding cannot be controlled by the previous methods, anterior nasal packing is required. This involves placement of 12-mm ($^1/_2$-inch) petrolatum- or antibiotic-impregnated strip gauze into the nasal cavity. The adult victim requires 90 to 120 cm (3 to 4 feet) of such gauze to pack the nose adequately and tamponade the bleeding. The gauze is layered in tiers starting on the nasal floor and proceeding to the roof of the nose. Both ends of the gauze are left outside the nose and taped to the face to prevent inadvertent aspiration. Expandable packing

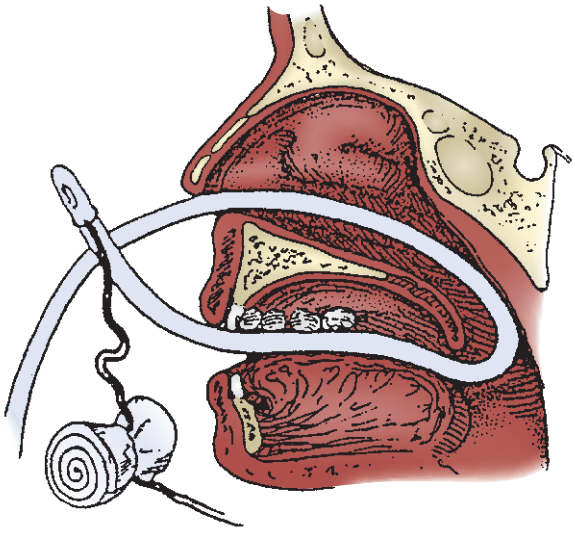


Figure 26-13. Inserting a posterior nasal pack. (From Fleisher GR, Ludwig S: Textbook of Pediatric Emergency Medicine. Philadelphia, Lippincott Williams & Wilkins, 1999, with permission.)

(Rapid Rhino, Weimert Epistaxis Packing, and Rhino Rocket are current varieties) with an applicator device for rapid deployment, or a Foley catheter, can also be used for anterior nasal tamponade. Because anterior nasal packing blocks sinus drainage and can predispose to sinusitis, the victim should be placed on prophylactic amoxicillin 875 mg with clavulanic acid 125 mg (Augmentin) PO twice a day until the packing is removed in 48 hours.

Because it is not possible to visualize the site of posterior epistaxis, the victim requires a posterior nasal pack or placement of a Foley catheter or nasal balloon.[1] Placing the conventional posterior nasal pack involves first gently inserting a lubricated soft tube into each nostril until the ends can be visualized in the back of the throat, grasped with a hemostat, and brought out through the mouth. Use Foley catheters, nasogastric tubes, chest tubes, or improvised substitutes such as one of the products used to hold sunglasses in place. A cylindrical pack of 10 × 10-cm (4 × 4-inch) gauze pads is prepared and held in shape by tying three silk sutures around it and leaving the ends long. The pack should be the same diameter as a circle made by the victim's thumb and forefinger (the "OK" sign). The two end sutures are attached to the oral ends of the catheters (Fig. 26-13). The nasal ends of the catheters are then pulled carefully back out of the nose until the pack is firmly positioned against the posterior aspect of the nasal cavity above the soft palate. The sutures are then detached from the catheters and tied over a bolster placed underneath the nose. The middle suture remains outside of the mouth and is secured externally to be used to remove the pack 48 hours later.

A 14- to 16-fr Foley catheter with a 30-mL balloon can also be used as a posterior pack. The lubricated catheter is inserted through the nose into the posterior pharynx, inflated 10 to 15 mL, and then gently pulled back into the nasopharynx and held in position by clamping the external end with a hemostat. Commercially available preshaped nasal balloons can also be used to treat posterior epistaxis.[1] Air is preferred over saline for inflating nasal balloons as a safety precaution in case of breakage.

Nasal Fracture

Because of the prominence of the nose, nasal fracture is the most common facial fracture. It is often accompanied by abrasions, lacerations, and epistaxis. Nasal bleeding should be managed first. Once this bleeding is controlled, any facial wound should be properly cleansed. Local anesthesia may be needed to accomplish removal of foreign material. Plain lidocaine or another local anesthetic without vasoconstrictor is recommended when injecting the nose. Particulate matter is removed either by irrigation or scrubbing with a sterile brush. This is essential to avoid infection and prevent tattooing.

Abrasions should be covered with antibiotic or antiseptic ointment (mupirocin, bacitracin) and washed gently twice daily with a mild soap and warm water to prevent crusting. Small laceration edges can be reapproximated with tape closures. Deeper lacerations require interrupted sutures. Remove sutures in 3 to 5 days, at which time tape strips can be placed if additional stabilization of wound margins is necessary.

Once soft tissue injuries have been managed, the nose should be assessed for possible fracture by observing it for symmetry from the front and from below. If swelling has already occurred, this may be difficult to determine. However, palpation of the bridge may reveal displacement that is not visible, and crepitus can sometimes be felt. Point tenderness may also be indicative of a fracture, but it can also be associated with soft tissue injury.

After external examination of the nose, the interior of the nasal cavity should be examined for lacerations and septal hematoma. Remove blood clots with a swab or suction to improve visibility. Small lacerations may not require treatment, but large lacerations should either be closed with resorbable sutures or covered with Oxycel, Gelfoam, Surgicel, or Avitene to control bleeding. Any deviation, bulging, or widening of the nasal septum may indicate a hematoma. However, septal deviation may have been present before the injury. To determine if a hematoma is present, the area can be pressed with a cotton-tipped applicator. A hematoma feels soft and the area may be temporarily indented by the pressure.

A septal hematoma needs to be drained to prevent pressure necrosis and loss of nasal support, or the formation of a septal abscess, which can also produce destruction of the septum.[13] The area is anesthetized by injection of a local anesthetic or application of a topical anesthetic (benzocaine 20%), and a small incision is made in the most inferior aspect of the hematoma. The nasal passage should then be packed with $^1/_2$-inch petrolatum- or antiseptic-impregnated gauze to prevent recurrence of the bleeding.

Because most surgeons prefer to treat nasal fracture after the swelling has subsided (5 to 10 days after injury), such an injury does not require immediate treatment.[13] As long as there is no active bleeding, and any abrasions and lacerations have been treated, the victim has at least 3 to 5 days before definitive management is indicated. If the nose appears straight as the swelling subsides, and the victim can breathe easily through both nostrils, there probably was no fracture or one with minimal displacement, and further treatment may not be necessary. In the interim, the nose can be protected from further injury by a splint made by cutting a triangular piece of a SAM splint large enough to fit the contour of the nose. Rest the splint on the adjacent part of the face without placing pressure on the nasal bridge. This shield is held in place by strips of adhesive tape. Nasal packing is not necessary unless it is used to control epistaxis or

Figure 26-14. Mandibular fracture. **A,** Note the malocclusion before reduction. **B,** Normal occlusion after reduction.

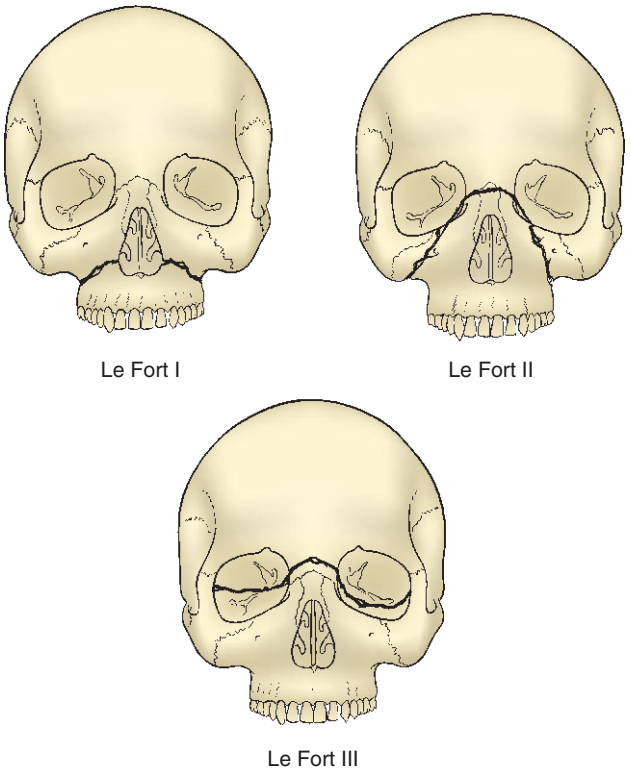


Figure 26-15. Classification of midface fractures. (Redrawn from American Association of Oral and Maxillofacial Surgeons: Oral and Maxillofacial Surgery Services in the Emergency Department. Rosemont, IL, American Association of Oral and Maxillofacial Surgeons, 1992, with permission.)

recurrent septal hematoma. Penicillin VK or amoxicillin 500 mg PO should be administered four times a day for 5 days.[13]

Jaw Fracture

Any person who has suffered a head or facial injury should be examined for a jaw fracture.[17,37] First, evaluate occlusion by having the victim bite the teeth together. Any deviation of the bite or change in the level of the occlusal plane, especially in the mandible, should raise the suspicion of a fracture (Fig. 26-14). Usually there will also be a tear in the gingival tissues and bleeding and ecchymosis at the site of the discontinuity. A sublingual hematoma is a common sign of a mandibular fracture. In edentulous areas, there will also be a discrepancy in the level of the bone, often accompanied by a disruption in the mucosa.

If no obvious tooth or bone displacement is noted, bimanual examination of the body of the mandible should determine if any abnormal movement can be detected. It is particularly important to evaluate any area of contusion in the soft tissue. In addition to preternatural movement, a grating sound can occasionally be heard when a fracture is present.

The mandibular condyles should be evaluated, especially if the chin has sustained a traumatic blow. A unilateral fracture is suspected when there is a shift of the midline of the mandible to the painful side on mouth opening. Bilateral fractures often result in an anterior open bite (see Figure 26-1). Normally, the condyles can be palpated by placing a forefinger in front of the external auditory meatus. If the condyle cannot be palpated, or if it does not move significantly when the mouth is opened, a fracture may be present.[17,37]

The maxilla is examined for possible fracture by grasping the anterior segment between the thumb and forefinger and gently rocking the jaw anteroposteriorly and laterally. If a complete (Le Fort I) fracture is present, the entire maxilla will move (Fig. 26-15). With a unilateral fracture, only one half of the maxilla will move. If a complete maxillary fracture is detected, the presence of a pyramidal (Le Fort II) fracture extending to the nasal area needs to be considered. The maxilla should again be rocked gently while the bridge of the nose is grasped between the thumb and forefinger of the opposite hand. Any movement of the nasal complex is indicative of a pyramidal fracture. Because this fracture also extends through the infraorbital rim, this area should be palpated for the presence of a step deformity. However, care must be taken not to confuse a fracture with the infraorbital foramen, which can also be felt in this region. If a fracture is present, the victim will feel numbness below the eye, as well as

in the upper lip and lateral aspect of the nose. This is due to injury to the infraorbital nerve.

The third type of midface fracture, the Le Fort III, involves detachment of the midface from the base of the skull. This is a complex injury often accompanied by intracranial trauma. There is usually subconjunctival hemorrhage and bilateral periorbital edema and ecchymosis. Usually, the eyelids are swollen shut. Laceration of the meninges causes cerebrospinal fluid to leak from the nose. To distinguish cerebrospinal fluid from mucus, hold a clean, white handkerchief under the nose for a moment and then allow the material to dry. Mucus will stiffen the fabric, whereas cerebrospinal fluid will dry as a double ring without stiffening the fabric. When cerebrospinal fluid rhinorrhea is suspected, an antibiotic (penicillin VK 500 mg PO four times a day) should be administered, but the nose should not be packed.

Fractures of the zygomaticomaxillary complex usually result from a blow to the cheek. The most common findings are subconjunctival hemorrhage and ecchymosis and swelling of the lids. There may be double vision because of displacement of the globe or entrapment of extraocular muscles. As with a Le Fort II fracture, there is numbness in the distribution of the infraorbital nerve. Because of the depression of the cheekbone, the victim's face has a flat appearance.

Fracture of the zygomatic arch can occur as an isolated injury. Such a fracture results in a depression on the lateral aspect of the face. Because the coronoid process of the mandible is located beneath the zygomatic arch, this type of fracture can also result in inability to open the mouth fully.[17]

The best treatment for a jaw fracture is immediate immobilization. Even if perfect alignment is not achieved, fixation makes the victim more comfortable, reduces bleeding, and avoids further displacement of the fragments. Fractures that pass through the tooth-supporting portion of the mandible may be quickly stabilized with a bridle wire, or more securely held with an arch bar. Fractures in more posterior locations can be temporarily immobilized with a bandage (Fig. 26-16), which should pull the mandible in a superior direction. Do not pull the chin posteriorly because this can displace the fracture and compromise the airway. More rigid fixation can be obtained with intermaxillary wiring, which involves placing arch bars on upper and lower arches, placing the teeth in proper occlusion, and then connecting the upper arch bar to the lower bar with elastics or thin wire.

A posteriorly displaced Le Fort fracture should be disimpacted using forward traction. If the mandible is intact, the maxilla can be held in place by forcing mouth props (commercially available or improvised) between the upper and lower molars.[3] Significant bleeding from the nasal cavities sometimes accompanies midface fractures. See the section on Epistaxis for victim positioning and treatment. However, any tamponade of the nasal cavities will displace the mobile maxilla inferiorly unless it is first stabilized from below with mouth props.

A victim with facial bone fractures should be taken to a hospital for definitive treatment. Indications for immediate evacuation include complicated or uncontrolled medical problems, such as type 1 (insulin-dependent) diabetes, extremes of age (younger than 5, older than 55 years), midface fractures, displaced mandibular fractures, and zygomatic complex fractures.[18] In the interim, an antibiotic (penicillin VK 500 mg PO four times a day) and pain medication should be given. However, strong narcotics should be avoided if there is an associated head injury, to avoid respiratory depression in an already obtunded victim. The victim should lie with the head raised above the heart to decrease bleeding and swelling, or in a sitting position with the head forward so that blood drains out the mouth or nose.[3] The victim should never be left unattended.

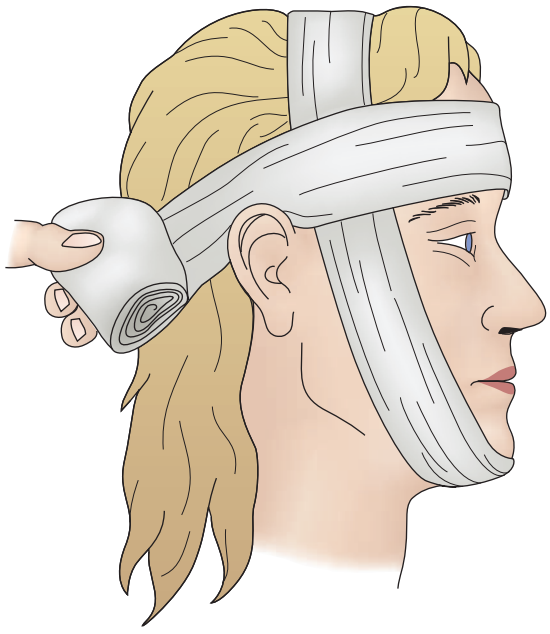


Figure 26-16. A simple bandage can be used to stabilize temporarily a jaw fracture.

Soft Tissue Injuries

Wounds of the oral mucosa and face should be treated after repair of the dental injuries and jaw fractures. Lacerations are likely to be reopened if closed before intraoral manipulations. Soft tissue injuries should be thoroughly irrigated and cleansed of foreign debris to prevent infection and tattooing. Tissue debridement should be very conservative. The excellent blood supply to this region means that wounds can be closed with sutures or other wound closures with little fear of infection, if treatment can be accomplished within 6 hours of the injury.[2,19] Sutures should be removed in 3 to 5 days, after which tape strips can be placed for additional stabilization of the wound margins. Facial abrasions should be covered with an antiseptic ointment and gently washed twice daily with mild soap and warm water to prevent crusting. Small lacerations of the oral mucosa need not be closed. Use direct pressure for hemostasis.

Through-and-through lacerations, which are common in the lower lip, are treated as follows. First, close the mucosal layer from an intraoral approach. Next, irrigate the wound thoroughly. If possible, obtain clean instruments and gloves. Finally, close the remainder of laceration from an extraoral approach. A laceration crossing the vermilion border of the lip requires careful alignment to avoid disfigurement. Deep lacerations of the soft palate involve the muscles, and must be closed in layers. Maintain traction on the tongue while lingual lacerations are sutured. Have an assistant or the victim grasp the tongue with gauze and hold it forward. After any intraoral repair, have the victim avoid rinsing for 24 hours and advise a liquid, nondairy

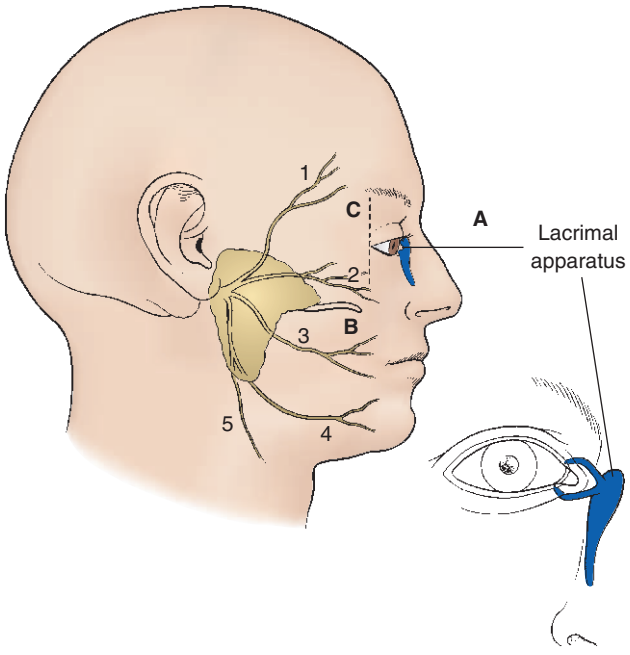


Figure 26-17. Structures that may be injured by facial lacerations. A, The lacrimal drainage system; B, the parotid duct; C, a line drawn through the lateral canthus of the eye; facial nerve injuries posterior to this line should be repaired as soon as possible; 1–5, branches of cranial nerve VII. (Redrawn from American Association of Oral and Maxillofacial Surgeons: Oral and Maxillofacial Surgery Services in the Emergency Department. Rosemont, IL, American Association of Oral and Maxillofacial Surgeons, 1992, with permission.)

diet. After this period, initiate rinsing four times a day with warm saline, chlorhexidine gluconate 0.12%, or diluted peroxide.

A facial laceration may be complicated by damage to associated structures (Fig. 26-17). Injury to the lacrimal drainage system is present if a probe inserted into the punctum at the medial corner of the lid emerges into the laceration. Damage to the parotid duct should be suspected if there is leakage from the wound when Stensen's duct is irrigated with saline. Facial nerve damage is sought by observing the victim move the eyebrows, eyelids, and mouth. If facial nerve injury occurs behind a vertical line through the lateral canthus of the eye, evacuation for immediate repair is recommended.[17]

Dislodged Fillings, Crowns, and Bridges

No emergency treatment is necessary for a dislodged filling, small piece of tooth, crown, or bridge in the absence of other symptoms. Often the best course of action is to file off any excessively sharp areas of the tooth that are irritating the victim's soft tissues, and otherwise leave the tooth alone. If you are going to attempt a temporary filling or crown recementation procedure, make sure you do not create an occlusal problem by leaving the restoration too high. This will only make the patient more uncomfortable.

Appliance Sores

Complete dentures, removable partial dentures, retainers, mouth guards, and the like can cause mucosal sores if their fit changes even a small amount. The mucosa will appear red or even ulcerated. Often the patient can do without the appliance until definitive treatment is available. If not, it is necessary to file or scrape away a small amount of material where the appliance is rubbing the mucosa. Finding the proper area of the appliance to relieve can be a challenge. Mark the sore spot (a tiny piece of adhesive tape, sticky side out, or a slurry of water and grape drink mix are options), insert the dried appliance, and remove. Hopefully the mark will have transferred to the appliance, showing the proper place to adjust.

Orthodontic Emergencies

Orthodontic appliances can cause soft tissue irritation or ulceration. Cover the offending fixture with soft wax. Sometimes a protruding wire can be bent until the sharp portion faces away from the soft tissues. If a bracket or wire becomes excessively loose, it can usually be removed with judicious tinkering. If a wire needs to be cut and small wire cutters are unavailable, try fingernail clippers or repeatedly bending the appliance until the metal fatigues and breaks.

LOCAL ANESTHESIA

Local anesthesia is a prerequisite to many emergency dental, oral, and maxillofacial procedures. Anesthesia of any upper tooth and the associated buccal soft tissues can be obtained by infiltration. Approximately 2 mL of a local anesthetic solution is placed as close to the apex of the tooth as possible, just above the periosteum. By holding the syringe parallel to the long axis of the tooth, the needle tip is guided in the proper direction (Fig. 26-18). Four percent articaine with 1:100,000 epinephrine (Septocaine) has become the most widely used local anesthetic in dentistry.[41] Two percent lidocaine with 1:100,000 epinephrine is also commonly used, and 2% bupivacaine with 1:200,000 epinephrine (Marcaine) is useful for long-term pain relief. Other available local anesthetics can be substituted.

The mental nerve block is a simple method for obtaining anesthesia of the teeth and buccal mucosa from the second premolar forward. Proceed as for the infiltration, except that the target area is the mental foramen, located between apices of the lower first and second premolars (Fig. 26-19). After depositing the solution, gently massage the area for about 30 seconds.

The inferior alveolar nerve block is more difficult to learn, but produces anesthesia of all the lower teeth up to the midline, as well as the buccal soft tissues forward of the mental foramen. The lingual nerve is also blocked by this injection, producing numbness of the anterior two thirds of the tongue and lingual gingiva. Figure 26-20A shows how the deepest concavity on the anterior border of the mandibular ramus is palpated by the thumb while the deepest concavity on the posterior border is palpated by the index finger. The target area is then halfway between the rescuer's thumb and index finger.[38] Figure 26-20B shows how the syringe is kept parallel to the plane of the lower teeth, and how to puncture the mucosa just medial to the rescuer's thumbnail. When bone is contacted in the target area, the anesthetic solution is deposited. Experienced dentists miss the target area, and thus fail to produce adequate anesthesia, in approximately 10% of injections.[38]

DENTAL FIRST-AID KIT

Items necessary to manage dental emergencies can be added to a wilderness first-aid kit without a large sacrifice of space or weight. Cavit (Premier Co., Norristown, PA) is temporary filling

material that requires no mixing and is easy to use. Squeeze a small amount of the material from the tube and place it in the tooth. Wet a dental packing instrument (or cotton-tipped applicator or toothpick) to prevent sticking and pack the Cavit well. Then remove any excess. Have the victim bite to displace material that would interfere with occlusion. The filling material will set in a few minutes after contact with saliva.

Zinc oxide/eugenol cements (Intermediate Restorative Material [IRM]; L. D. Caulk Co., Milford, DE) consist of a liquid and a powder. Start with two drops of the liquid and begin mixing in the powder. Keep adding powder to make a dough that is not sticky yet will hold together. Dip the instruments in some powder to keep the mixture from sticking. Insert and shape the filling material as explained previously. Zinc oxide/eugenol cements have several advantages compared with Cavit. They are significantly stronger and can be mixed to a doughy stage for filling or slightly thinner for use as cement. Most important is the soothing effect of eugenol on teeth with pulpitis. However, the liquid often leaks from its container, lending a pervasive odor to the backpack and tent; wind tends to blow the powder away; and the material is difficult to mix and is a sticky mess to insert into the tooth.

A more complete kit for extended expeditions should include a no. 151 universal extraction forceps and a straight elevator

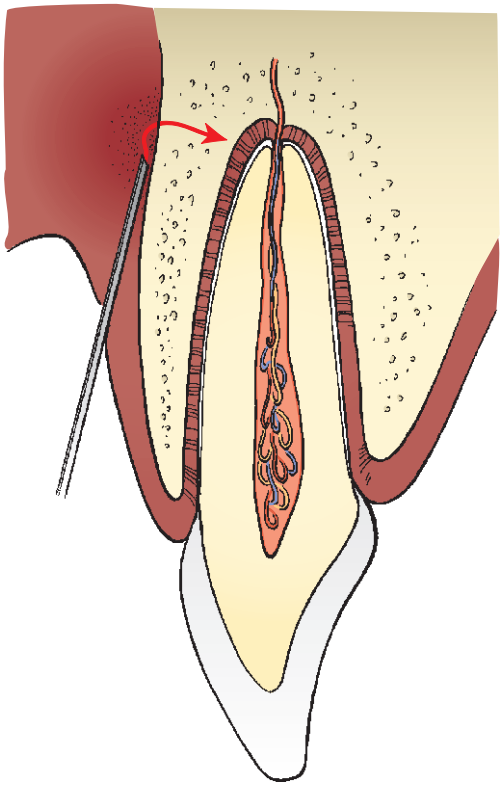


Figure 26-18. Technique for infiltration of a local anesthetic.

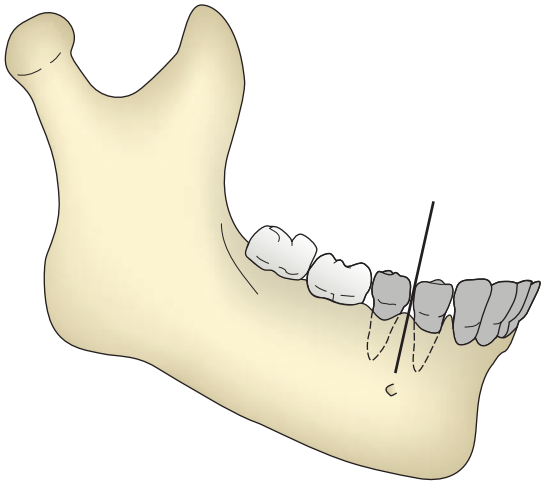
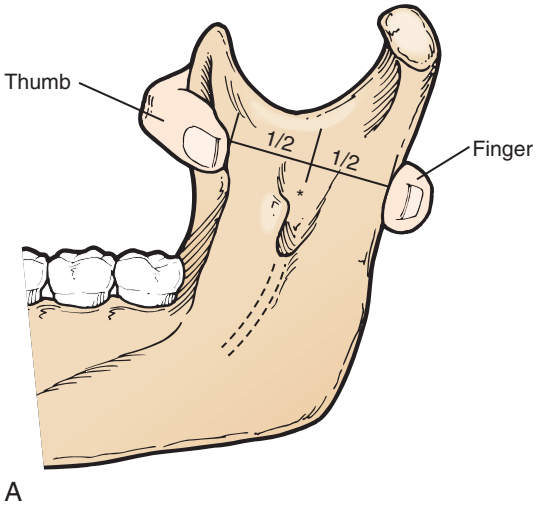


Figure 26-19. Technique for blocking the inferior alveolar nerve near the mental foramen. The shaded teeth will be anesthetized.



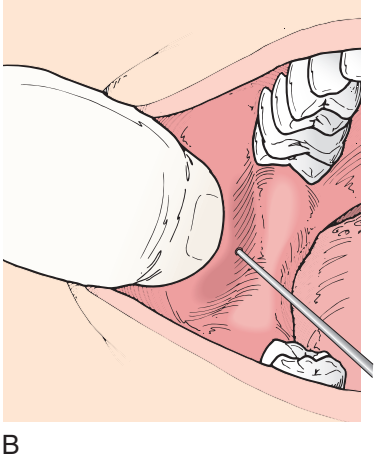


Figure 26-20. **A,** When administering the inferior alveolar block, the target area lies on the medial surface of the mandibular ramus, halfway between the rescuer's thumb and forefinger. **B,** Keep the syringe parallel to the occlusal plane.

for extracting teeth. A mouth mirror, orthodontic wax, dental floss, dental syringe, 30-gauge needles, and anesthetic cartridges complete the kit. These items fit in a small case and weigh about 14 ounces. A custom dental first-aid kit is preferred over commercial dental "travel kits," which contain unnecessary items and lack essential ones.

In an outdoor situation, techniques must often be adapted or improvised depending on the items available. For example, Figure 26-10 shows how a suture can be used to splint an avulsed or extruded tooth. A temporary filling can be fashioned from softened candle wax, an emery board can be used to smooth a sharp tooth, or a pocket knife can be used to perform a drainage procedure.

PREVENTION

The vast majority of dental emergencies can be prevented, beginning with pretrip planning. Before any extended travel in a remote area, all persons should have a thorough dental examination, radiographs, periodontal care, and treatment of potentially troublesome teeth. The Peace Corps requires certification that examination and treatment have been performed before assignment to developing countries. The National Science Foundation has similar requirements, and in addition requires impacted and unopposed third molars to be extracted before travel to Antarctica.

Once in the wilderness, minimal precautions can have significant effects. Lip balm with sunscreen can inhibit herpes labialis outbreaks. Routine personal oral hygiene will prevent many odontogenic infections and painful inflammatory conditions. Ideal care should include twice-daily tooth brushing and flossing. Toothpaste is not essential because mechanical removal of plaque and stimulation of the gingiva are the most important aspects of oral care. The fuzzy end of a hickory twig or even a finger can be used if a toothbrush is not available. In the wilderness, daily oral hygiene not only helps prevent dental emergencies but contributes to an overall sense of well-being and helps buoy morale when, for example, an expedition is tent-bound by bad weather.

Helmets are essential for preventing trauma in rock climbing, whitewater kayaking, and mountain biking. It is estimated that custom-made mouthguards prevent over 200,000 injuries every year in interscholastic sports.[27] Their use in backcountry recreation, now almost nonexistent, would probably also be very beneficial in appropriate circumstances.

The references for this chapter can be found on the accompanying DVD-ROM.

27 Wilderness Cardiology

Brett D. Atwater, Andrew Wang, and Matthew T. Roe

The wilderness environment places unique demands on the human body, particularly the cardiovascular system. The popularity of wilderness adventure sports continues to increase steadily, exposing many patients with underlying cardiovascular disease to risk. Certain wilderness activities, especially high-altitude trekking, diving, and those involving very high or low ambient temperatures, place significant stress on the cardiovascular system, stimulating dramatic physiologic, anatomic, and metabolic adaptations. In this chapter, we review these adaptations, present an appropriate cardiovascular screening protocol for use in patients before a wilderness excursion, and review basic therapeutic maneuvers that can be used in the event of a cardiovascular emergency in a wilderness setting.

HIGH ALTITUDE AND HYPOXIA

Myocardial tissue is profoundly dependent on an adequate arterial oxygen supply. Although systemic tissues use only approximately 25% of available arterial oxygen, myocardial tissues use 75% to 100% of available oxygen.[13] Atmospheric oxygen tension is reduced at increased elevation and arteriolar oxygen concentration declines in a near-linear relationship (Fig. 27-1). The heart responds to hypoxemia through a variety of physiologic, anatomic, and metabolic adaptations. These adaptations evolve if exposure to hypoxia is sustained.

Physiologic Adaptations to High Altitude and Hypoxia

Acute Hypoxia

Within the first few hours of ascent to high altitude, cardiac output increases proportionately to the degree of hypoxia, reaching a 75% increase at altitudes of 5000 m (16,400 feet).[36,41,42] This increase is due mostly to an increase in resting heart rate resulting from both an increase in sympathetic activation and a decrease in parasympathetic tone.[9,26] Changes in

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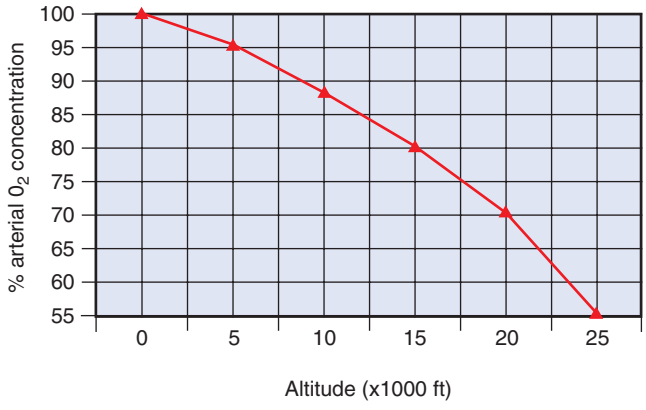


Figure 27-1. The relationship between mean arterial oxygen saturation (percentage) and altitude. (Adapted from McFarland R: Psychophysiological implications of life at altitude and including the role of oxygen in the process of aging. In Yousef MK, Horvath SM, Bullard RW [eds]: Physiological Adaptations: Desert and Mountain. New York, Academic Press, 1972, pp 157–182.)

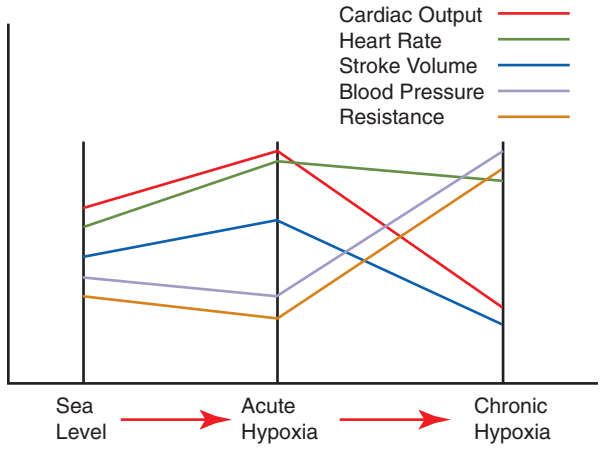


Figure 27-2. A summary of the cardiovascular physiologic adaptations to acute and sustained exposure to hypoxia. Cardiac output, heart rate, and stroke volume rise with acute hypoxia but decrease to levels at or below sea level values with sustained exposure. Blood pressure and peripheral vascular resistance fall with acute hypoxia before rising with sustained hypoxia. (Adapted from Wolfel E, Levine B: The cardiovascular system at high altitude: Heart and systemic circulation. In Hornbein T, Schoene RB [eds]: High Altitude: An Exploration of Human Adaptation. New York, Marcel Dekker, 2001, pp 235–292. Copyright 2001. Reproduced by permission of Routledge/Taylor & Francis Group, LLC.)

stroke volume play a minor role in the acute rise in cardiac output. Myocardial contraction velocity is augmented and left ventricular (LV) end-systolic volume (LVESV) is slightly reduced with high-altitude exposure, but the increase in contractility is offset by a dramatic reduction in preload and LV end-diastolic volume (LVEDV).[36,41,42] In addition, peripheral vascular resistance drops dramatically with hypoxia, driven primarily by peripheral vasodilation.[32] In fact, hypoxia can override the normal vasoconstrictive effects of the sympathetic nervous system, compromising the ability to maintain normal blood pressure. This may explain the increased incidence of orthostatic hypotension and syncope reported in otherwise healthy individuals early after ascent to high altitude.[29] In summary, acute high-altitude exposure results in increased cardiac output, increased heart rate, increased contractility, decreased LV filling pressure, and decreased peripheral vascular resistance (Fig. 27-2).

Chronic Hypoxia

The body acclimates to hypoxia within the first few days of exposure through further adaptation of normal physiology. The lungs maximize alveolar oxygen concentration (P_{AO_2}) by increasing alveolar ventilation, whereas red blood cells shift the oxyhemoglobin dissociation curve to the left through increased expression of 2,3-diphosphoglycerate. The kidneys stimulate hematopoiesis in the bone marrow by increasing production of erythropoietin, and further increase serum hemoglobin concentration through diuresis of extracellular volume. These changes significantly augment tissue oxygen delivery, reducing the hyperdynamic state of the circulatory system.

Resting heart rate remains elevated, regardless of the duration of hypoxia. Despite this chronically elevated heart rate, cardiac output is depressed with chronic hypoxia, often to below baseline sea level values.[22] This adaptive mechanism increases tissue capillary transit time, allowing more complete oxygen transfer. Because heart rate remains elevated regardless of duration of hypoxia, cardiac output is reduced through a reduction in stroke volume.[22] Numerous echocardiographic studies have shown a consistent reduction in LVEDV with preservation of ejection fraction in persons with sustained high-altitude exposure.[1,39] The reduced LVEDV is likely secondary to both a 20% to 30% reduction in plasma volume[20] and a significant impairment of right ventricular (RV) function at altitude.

As the body adapts to hypoxia and circulating arterial oxygen concentration rises, peripheral vascular resistance rises as well. This leads to a steady rise in both systolic and diastolic blood pressure during the initial days to weeks of acclimatization. Persons who live at low altitudes may experience transient hypertension after acclimatization to high altitude, but studies of native highlanders show that sustained systemic hypertension is relatively rare.[25]

Functional and Anatomic Changes of the Right Ventricle at High Altitude

Hypoxia causes significant pulmonary artery vasoconstriction and dramatically increased pulmonary artery and RV pressures.[18] Electrocardiographic (ECG) studies demonstrate development of rightward axis deviation and T-wave inversions in the right precordial leads both acutely and chronically after exposure to high altitude, indicating significant RV strain or overload.[12,19] ECG studies in groups of climbers at elevations above 6000 m (19,680 feet) demonstrate RV dilation in response to high altitude.[15] In one group of ultra-endurance athletes competing in a high-altitude ultra-distance marathon, 5 of 14 studied runners developed transient RV dilation and hypokinesia with paradoxic septal wall motion in response to high-altitude exercise.[8] These changes resolved at 1-day follow-up in all subjects. With chronic high-altitude exposure, people normally show ECG evidence of RV hypertrophy, suggesting that pulmonary hypertension and elevated RV pressures do not resolve with time.[31]

Metabolic Changes with Hypoxia

Hypoxia reduces entry of adenosine diphosphate (ADP) into the respiratory chain, reducing the rate of oxidative phosphoryla-

tion. This leads to increased intracellular ADP and decreased intracellular adenosine triphosphate (ATP). High intracellular ADP releases inhibition of phosphofructokinase, resulting in increased conversion of fructose-6-phosphate to fructose-1,6-diphosphate, stimulating glycolysis. Many other mitochondrial enzymes have increased activity during exposure to altitude, suggesting a further augmentation of the activity of glycolytic enzymes in the myocardium.[44] Some intriguing studies comparing high-altitude natives in Peru and Nepal with lowlanders suggest a greater reliance on carbohydrate metabolism for energy resources of resting myocardium and enhanced capacity for carbohydrate metabolism after prolonged exposure to high altitude.[16,28]

In summary, the human cardiovascular system makes numerous metabolic, hemodynamic, and physiologic adaptations to hypoxia associated with high-altitude exposure. These adaptations evolve with chronic exposure to high altitude.

DIVING

Diving places a unique combination of physical stressors on the cardiovascular system. Having an adequate cardiovascular reserve is essential to the safety and enjoyment of the sport. Depending on the age of the population, sudden cardiac death (SCD) and myocardial infarction (MI) account for 12% to 27% of the 100 annual diving fatalities.[10] Adequate screening before participation is essential because the existence of significant underlying cardiovascular disease should disqualify a candidate from diving.

Stressors

Immersion

Immersion significantly reduces the effect of gravity on the diver's lower extremity venous blood pool. This blood pool redistributes from the limbs to the thorax, increasing intrathoracic blood volume by up to 700 mL and increasing right atrial pressure by up to 18 mm Hg.[24] This immediately increases stroke volume and cardiac output by up to 100% before finally stabilizing at a lower level. Frequently, this increase in central arterial and venous blood volume results in diuresis and resultant dehydration on surfacing. Patients with congestive heart failure (CHF) or other cardiovascular states that are sensitive to cardiac pressure overload may do poorly with these dramatic shifts in volume status.

Exercise

Exercise directly increases myocardial oxygen demand through increases in heart rate, stroke volume, and cardiac output. Typically, divers have extended exercise sessions and use large muscle groups nearly continuously, resulting in a sustained elevation of cardiac output and myocardial oxygen demand. This increased myocardial oxygen demand may cause angina in patients with underlying coronary artery disease (CAD) and inadequate cardiovascular reserve.

Breath Holding and the Diving Reflex

The diving reflex was first described in 1870 by the French physiologist Paul Bert after he observed that heart rate dramatically slowed after forced water submersion. The response involves slowing of the heart rate, reduction in cardiac output, changes in arterial blood pressure, and peripheral vasoconstriction. It is accentuated by cooling of the face, particularly in the distribution of the ophthalmic branch of the trigeminal nerve.[33] The reflex is mediated vagally and typically reduces the heart rate to 20 to 30 beats per minute. Heart rates as low as 8 beats per minute have been measured in accomplished breath-hold divers.[11] This intense bradycardia frequently results in supraventricular and ventricular arrhythmias, although these rhythms are seldom fatal.[30]

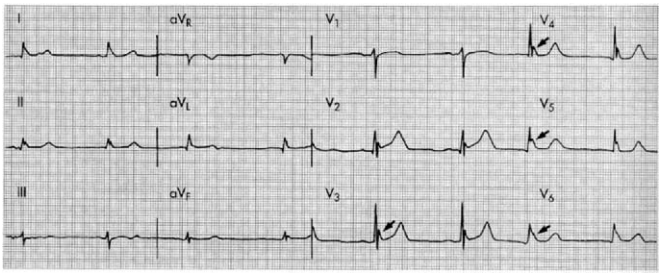


Figure 27-3. Systemic hypothermia. The arrows (V_3 through V_6) point to the characteristic convex J waves, termed Osborn waves. (From Goldberger AL: Clinical Electrocardiography: A Simplified Approach, 6th ed. St. Louis, CV Mosby, 1999.)

HYPOTHERMIA

The initial response to cold exposure is peripheral arterial constriction with subsequent reduction in peripheral blood flow and increase in central blood flow. There is an increase in arterial and venous blood pressure and an accompanying increase in heart rate with decreased coronary artery diastolic filling time. In mild hypothermia (core body temperature of 33° to 35° C [91.4° to 95° F]), this central volume shift results in diuresis, which can lead to dehydration. With moderate hypothermia (30° to 33° C [86° to 91.4° F]), heart rate and cardiac output fall and the ECG may show characteristic findings of prolonged QT interval or Osborn waves (Fig. 27-3). Atrial fibrillation also commonly occurs in this temperature range. In severe hypothermia (<30° C [86° F]), there is a significant reduction in stroke volume and heart rate, with a subsequent reduction in blood pressure. Ventricular fibrillation typically occurs at temperatures below 25° C (77° F), with cardiac arrest occurring at temperatures below 20° C (68° F). If found and resuscitated quickly, victims of hypothermia often have well-preserved brain function because hypothermia greatly reduces ischemic insult to the brain. In fact, at 25° C (77° F), the brain can tolerate 25 minutes of circulatory arrest without significant effect. In a review, Larach[23] describes cases of patients who were neurologically intact after almost 4 hours of cardiac arrest in the setting of hypothermia.

HYPERTHERMIA

Normally the hypothalamic thermoregulatory center controls core body temperature after exposure to high ambient temperature through augmentation of sweat production. Heat stroke occurs when this system fails, typically manifested by hyperpyrexia and central nervous system dysfunction. Cardiac manifestations of heat stroke include pulmonary edema, pericardial effusion, RV dysfunction, and hypotension.[46] Sinus tachycardia

TABLE 27-1. Summary of Commonly Prescribed Cardiac Medications and Potential Side Effects*

MEDICATION CLASS	USES	POTENTIAL SIDE EFFECTS
Beta blockers	CAD CHF Arrhythmia	Limit heart rate response to exercise, hypotension, fatigue, bronchospasm
Angiotensin-converting enzyme inhibitors	CAD CHF	Hypotension, renal failure
Antiplatelet agents	CAD	Excess bleeding with trauma
Anticoagulants	Atrial fibrillation	Excess bleeding with trauma
HMG-CoA reductase inhibitors	Hyperlipidemia CAD	Liver failure, muscle pain, rhabdomyolysis
Diuretics	CHF	Hypotension, renal failure
Antiarrhythmic agents	Arrhythmia	Arrhythmia, hypotension, CHF

CAD, coronary artery disease; CHF, congestive heart failure; HMG-CoA, hydroxymethylglutaryl coenzyme A.
*This list is not intended to be comprehensive and includes only those side effects that may be especially pertinent in a wilderness environment.

occurs in approximately 75% of patients, whereas atrial fibrillation occurs in 15%.[2] Ventricular arrhythmias occur very infrequently. Transient T-wave changes and prolongation of the QT interval and ST segment may also occur. Cardiac enzymes may transiently increase as well, although significant resultant heart dysfunction is rare.

SCREENING FOR FITNESS BEFORE WILDERNESS ADVENTURE

Physicians are often asked to evaluate the health risks associated with participation in wilderness activities. All patients should seek medical evaluation before embarking on a physically demanding wilderness excursion. A thorough history and physical examination should be elicited. A list of current medications should be obtained because certain cardiovascular medications may affect the safety of wilderness activities (Table 27-1). A general screening protocol can be used for most wilderness activities. Because of the special physiology of diving, additional restrictions apply when screening potential participants.

Screening before a General Wilderness Excursion

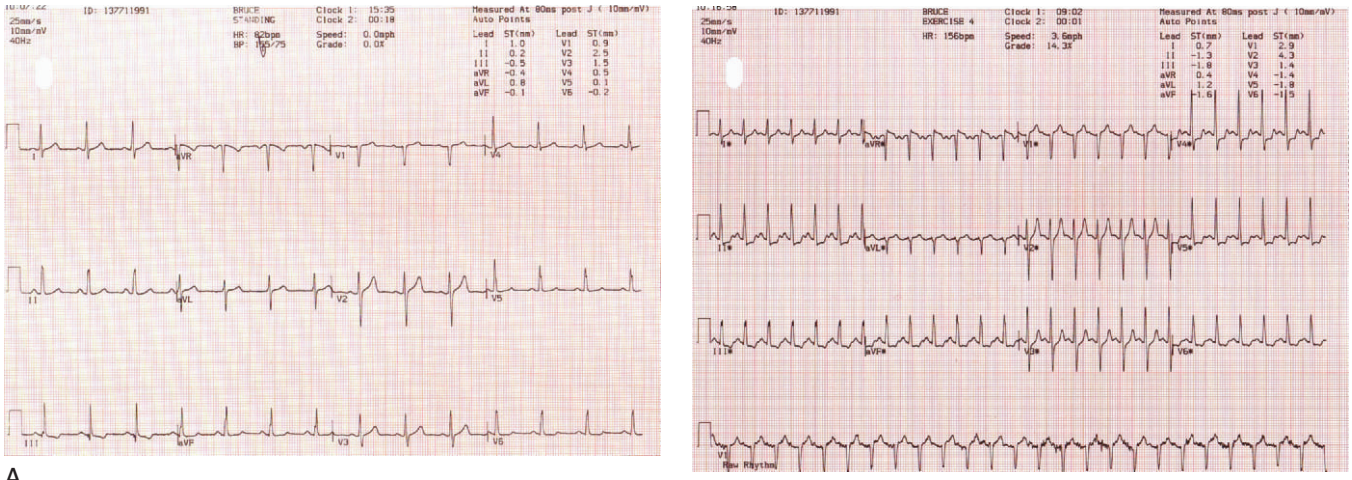
Coronary Artery Disease

Coronary artery disease is most commonly due to obstruction of the coronary arteries by atheromatous plaque. Characteristic signs and symptoms of CAD include exertional chest pain, CHF, cardiac arrhythmia, and SCD. Over 12,000,000 Americans have CAD,[5] many without having clinical symptoms.

As part of the initial general screening examination, risk factors for underlying CAD should be assessed, including history of previous CAD, previous coronary artery bypass graft (CABG) surgery, family history of early CAD in a first-degree relative, current smoking, renal insufficiency, hypertension, hyperlipidemia, or diabetes mellitus. All patients older than 50 years of age should also have a resting ECG study as part of their initial evaluation.[17] Asymptomatic patients younger than 50 years of age with normal results on a cardiac examination usually require no further testing. Asymptomatic patients older than age 50 years without risk factors for CAD and with a negative cardiac examination and normal ECG findings require no further testing. Asymptomatic patients older than age 50 years with an abnormal ECG result or one or more additional CAD risk factors should undergo exercise stress testing before embarking on a physically demanding wilderness excursion.[17] Similarly, patients with a known history of CAD, including prior CABG surgery or percutaneous coronary intervention (PCI), ECG evidence of a prior MI, stable angina, or history of prior unstable angina or acute MI, should undergo exercise stress testing within 2 years of embarkation.[17] A patient with a normal result on the follow-up stress test should have no restrictions on physical activities, including wilderness activities. Patients with evidence of residual ischemia on stress testing should be restricted to symptom-limited exercise and should be discouraged from participation in physically demanding wilderness activities in a remote setting. Patients who have recently had an MI, PCI, or CABG surgery should be treated in a cardiac rehabilitation program and evaluated by stress testing before participating in activities requiring strenuous physical exercise. Appropriate timing for return to exercise after cardiac events or procedures should vary on a case-by-case basis, but typically a period of at least 1 month is recommended.

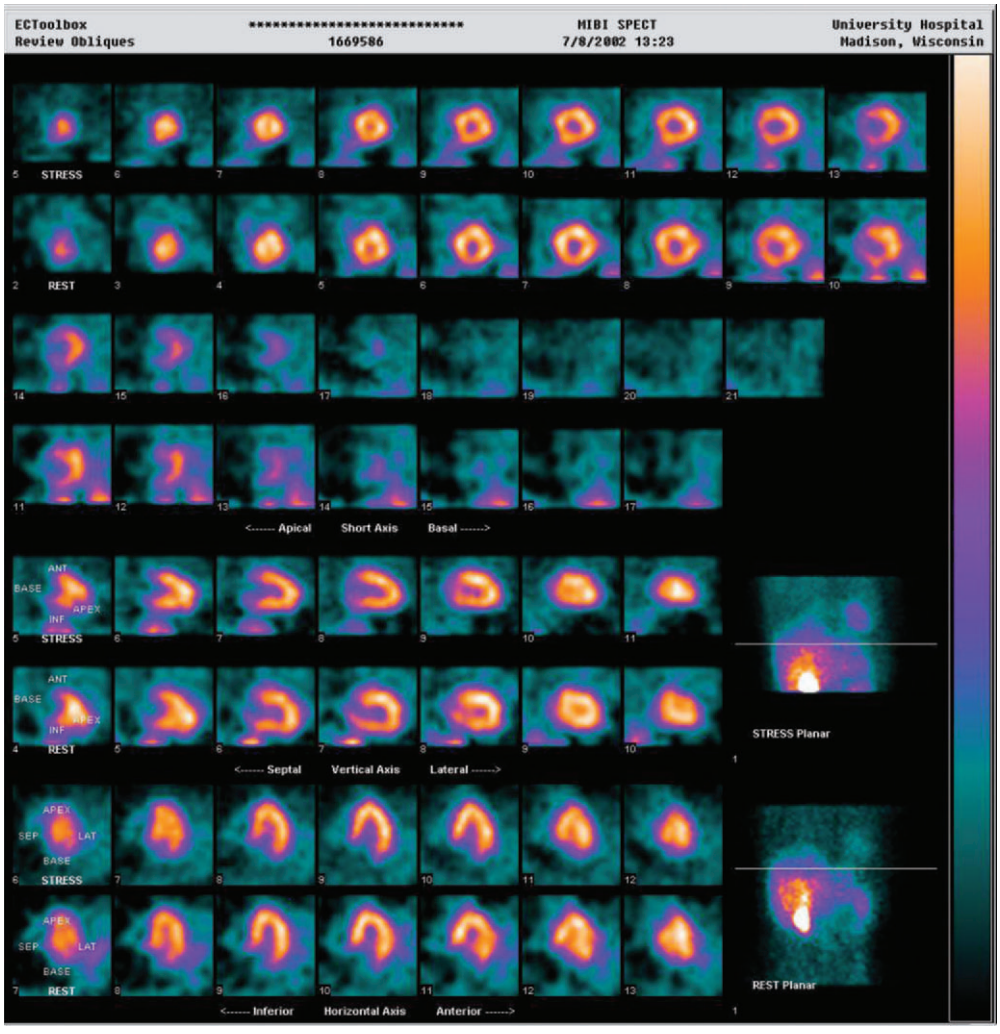
Exercise stress testing provides important prognostic information in patients at risk for ischemic heart disease. Cardiovascular reserve is assessed using a dynamic exercise protocol that includes a warm-up period, a period of continuous progressive exercise, and a cool-down period. ECG data, blood pressure, and heart rate are continuously monitored during exercise. Greater than 0.1 mV of ST segment depression or greater than 0.1 mV of ST segment elevation 60 to 80 msec after the J point are considered positive ECG results (Fig. 27-4). Other findings associated with a poor prognosis include failure to increase systolic blood pressure to a value greater than 120 mm Hg or a fall in systolic blood pressure during exercise, poor maximal work capacity, inappropriate heart rate response, chest discomfort with exercise, and ventricular arrhythmia with exercise.

Exercise stress testing may be performed with or without the use of additional imaging techniques, including nuclear imaging or echocardiography. Nuclear stress testing is performed by injection of a radionucleotide isotope at rest and again after stress. In areas of adequate perfusion, the radionucleotide is actively translocated from the bloodstream into the myocardium both at rest and with stress (Fig. 27-5A). In areas



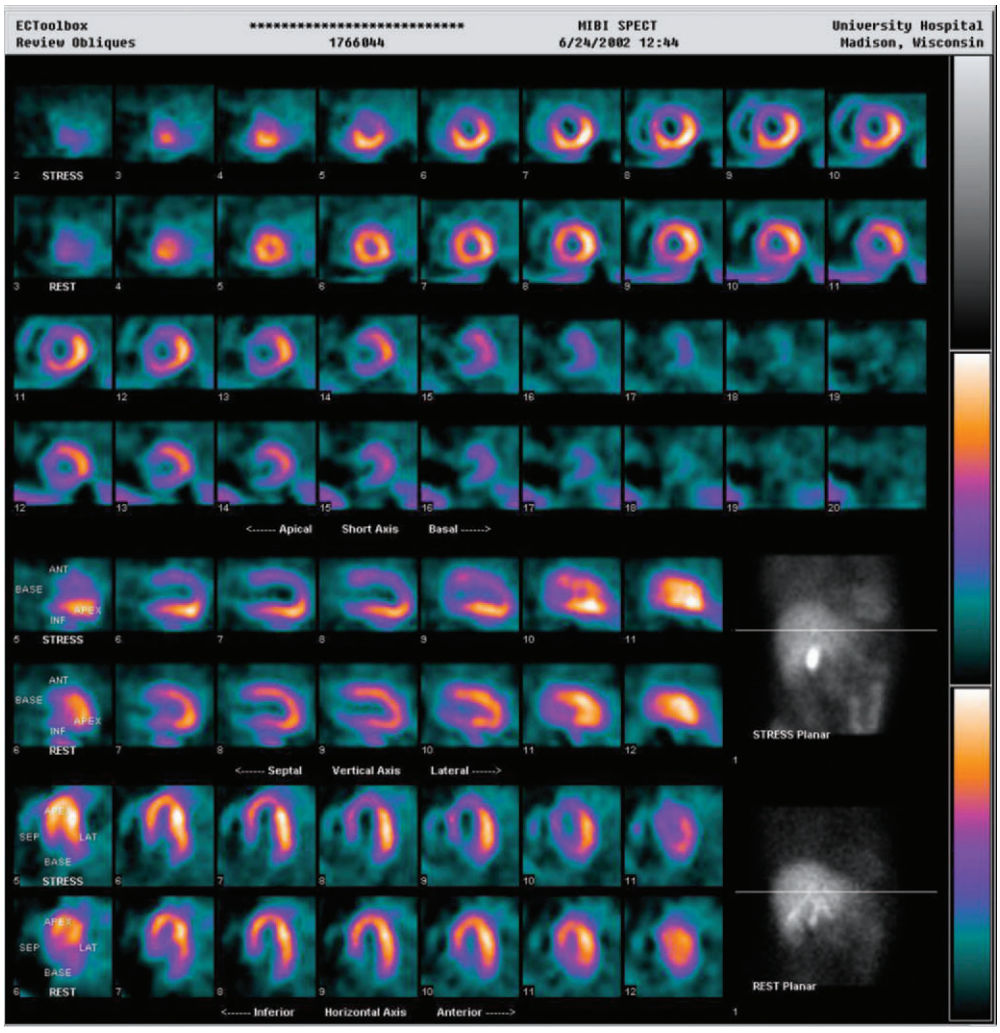
A B

Figure 27-4. Positive stress electrocardiogram results.



A

Figure 27-5. A, Negative nuclear stress test result.



B

Figure 27-5, cont'd. B, Positive nuclear stress test result.

with stress-induced ischemia, the radionucleotide cannot be actively translocated from the bloodstream to the myocardium, resulting in a perfusion defect on imaging (Fig. 27-5B). Resting and stress images are compared, and areas of stress-induced myocardial hypoperfusion are visualized. Stress echocardiography is performed by acquiring a standard resting echocardiogram followed by a repeat echocardiogram after stress. Areas with stress-induced hypoperfusion develop heart wall motion abnormalities that are visualized on the echocardiogram. The addition of imaging increases both the sensitivity and specificity of the stress test and provides valuable information about the location of coronary artery occlusions. This information is often helpful to interventional cardiologists and cardiac surgeons while planning revascularization.

It is well established that patients with documented CAD may perform moderate exercise with minimal risk. A survey of 518,000 diagnostic exercise tests performed in 73 medical centers revealed a mortality rate of 0.01%.[38] Another study evaluated the risk of SCD and MI in patients with known CAD participating in exercise rehabilitation programs.[40] A total of 51,303 patients exercised for 2,351,916 hours. Twenty-one cardiac arrests and eight MIs occurred for a cumulative event rate of approximately 0.02%. These reports suggest that moderate to heavy exercise is safe, even in patients with known CAD.

The screening physician should advise each patient about the early symptoms of unstable angina, MI, and CHF. Patients should be instructed to take along their prescription medications, including aspirin. Instructions should be provided detailing an appropriate course of action should the patient suffer a suspected cardiac event in the wilderness. Specifically, patients should be instructed to take an aspirin immediately after onset of symptoms and restrict themselves to bed rest while awaiting emergency medical evacuation. Chest pain should be controlled with nitrates, beta-adrenergic blockers or calcium channel blockers, and supplemental oxygen if available, and the patient should seek professional medical assistance as soon as possible.

TABLE 27-2. New York Heart Association (NYHA) Classification of Congestive Heart Failure

NYHA CLASS	SYMPTOMS
Class I	Patient with objective evidence of congestive heart failure without resulting limitations on physical activity.
Class II	Patient with objective evidence of congestive heart failure resulting in slight limitation of physical activity without symptoms at rest.
Class III	Patient with objective evidence of congestive heart failure resulting in marked limitation of physical activity without symptoms at rest.
Class IV	Patient with objective evidence of congestive heart failure resulting in inability to participate in physical activity, frequently accompanied by symptoms at rest.

Common symptoms of congestive heart failure include shortness of breath, exercise intolerance, lower extremity or pulmonary edema, dyspnea on exertion, and paroxysmal nocturnal dyspnea.
© New York Heart Association.

Congestive Heart Failure

Congestive heart failure is defined as a complex clinical syndrome resulting from a structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood.[3] Symptoms include dyspnea, fatigue, and fluid retention that may lead to pulmonary and peripheral edema. The severity of CHF symptoms is assessed using the New York Heart Association (NYHA) classification system (Table 27-2). Patients with CHF and significantly reduced LV function (ejection fraction <30%) are at high risk for SCD compared with the general population. In the past, patients with CHF were advised to limit their physical exertion in the belief that physical activity might lead to further deterioration of LV function. More recent studies have shown that physical inactivity leads to physical deconditioning and worsening CHF symptoms. Controlled trials have shown that exercise programs can lead to significant symptomatic improvement in patients with CHF.[7] Patients with CHF should be advised to avoid activities with particularly high cardiac demand, including diving and those involving high-altitude low-oxygen exposures, because they may precipitate CHF symptoms. Patients with severely impaired LV function are at high risk for SCD and should generally be advised to avoid all wilderness activities. Specific recommendations regarding participation in less strenuous activities should be made by a consulting physician on a case-by-case basis.

Valvular Heart Disease

Valvular heart disease refers to any disorder of one of the four cardiac valves and is commonly associated with either stenosis (reduced opening or orifice area) or insufficiency (impaired closing or coaptation of the valve leaflets) of the involved valve. Because either narrowing or impaired closure of a valve results in alteration or turbulence of normal blood flow through the heart chambers, a heart murmur may be a clue to the presence of a valvular lesion in the asymptomatic patient. Echocardiography is highly sensitive for the diagnosis of valvular heart disease and provides an accurate, noninvasive assessment of its severity. Patients found to have a significant systolic murmur or any diastolic murmur on physical examination should undergo transthoracic echocardiography (TTE) to evaluate its cause.[4]

Asymptomatic patients with TTE evidence of mild aortic stenosis, aortic insufficiency, mitral regurgitation, or mitral stenosis may participate in symptom-limited exercise.[4] Conversely, patients with TTE evidence of moderate or severe aortic stenosis, mitral stenosis, or mitral regurgitation should not participate in strenuous exercise. Patients with mild mitral regurgitation and associated LV dilation or atrial fibrillation should also avoid strenuous exercise. Patients with moderate to severe aortic insufficiency should undergo stress testing in addition to TTE before exercise. If these tests reveal normal LV ejection fraction and no evidence of exercise-induced myocardial ischemia, these patients can be cleared for symptom-limited exercise.[4]

Arrhythmia, Internal Cardioverter-Defibrillators, and Pacemakers

Heart arrhythmia is a common complication of underlying CAD or CHF. Arrhythmias are classified according to the location of their origin, either supraventricular or ventricular. Supraventricular arrhythmias may be benign and asymptomatic, or may cause significant hemodynamic compromise and symptoms of palpitations, chest pain, or CHF. Ventricular arrhythmias are usually unstable, rapid heart rhythms that lead to hemodynamic compromise, palpitations, chest pain, and frequently death.

Patients with a history of asymptomatic supraventricular arrhythmia, including multifocal atrial tachycardia, atrial flutter, and atrial fibrillation, may participate in symptom-limited exercise including wilderness activities. Patients receiving anticoagulation therapy are at a significant risk of bleeding with minimal trauma and should be advised to take appropriate precautions. Patients with a history of ventricular arrhythmia, including ventricular tachycardia or ventricular fibrillation, and those with an internal cardioverter-defibrillator (ICD) in place are at high risk of exercise-related SCD and should not participate in physically demanding wilderness activities.

Patients with a history of pacemaker placement usually have underlying disease of the native heart conduction pathway. The first implantable pacemakers were designed to pace at a fixed rate regardless of exercise level. These early-generation devices did not sense the patient's need for increased cardiac output during exertion, leading to characteristically poor exercise tolerance. Modern pacemakers adjust ventricular pacing rate according to the patient's level of physical activity by tracking the atrial rate, QT interval, or respiratory rate, or by tracking patient limb movement through a piezoelectric ceramic element. Some devices use a combination of methods to ensure an appropriate response.

Before embarking on a wilderness adventure, patients with a pacemaker should have routine CAD and CHF screening as well as a pacemaker interrogation to assess adequate battery life and appropriate sensing and pacing features. This will ensure maximal exercise capacity during their wilderness adventure.

Congenital Heart Disease

Congenital heart disease encompasses numerous distinct conditions that may or may not coexist with other cardiac or extracardiac manifestations. In broad terms, congenital heart disease can be categorized as either cyanotic (typically due to a relative reduction in pulmonary blood flow with direct systemic venous-to-arterial transit of blood) or acyanotic (normal arterial oxygen

saturation). Most patients with cyanotic congenital heart disease will have been previously diagnosed and treated. These patients typically have poor exercise tolerance and most should be restricted from strenuous wilderness activities. Patients with acyanotic congenital heart disease may have no signs or symptoms of disease until late in life, and therefore may not have been previously diagnosed with a cardiac condition. In the asymptomatic person, the presence of a murmur or abnormal ECG results may lead to further diagnostic testing, usually including an echocardiogram. Patients with acyanotic congenital heart disease may tolerate certain wilderness activities poorly, especially those occurring in high-altitude, low-oxygen environments. As discussed previously, hypoxia stimulates elevated RV and pulmonary artery pressures, potentially causing transient right-to-left shunting and sudden oxygen desaturation. Patients with a known acyanotic congenital abnormality should see their cardiologist before participation in such activities.

Patients with hypertrophic cardiomyopathy (HCM) are at higher risk of exercise-associated SCD and should generally be excluded from strenuous wilderness activities. Risk factors for SCD among patients with HCM include a family history of SCD, abnormal blood pressure response during exercise, recurrent syncope, nonsustained ventricular tachycardia on Holter recording, and extreme hypertrophy. Patients free of these five risk factors are considered at low risk for SCD. Patients with the obstructive form of HCM, termed *hypertrophic obstructive cardiomyopathy* (HOCM) or, formerly, *idiopathic hypertrophic subaortic stenosis* (IHSS), may have a dynamic worsening of LV outflow obstruction in the setting of higher heart rate or intravascular volume depletion that may result in syncope. Ultimately, a patient's decision to participate in sports, including diving, should be made after an informative discussion with their cardiologist regarding the perceived risks and benefits.

Heart Transplant

Compared with the general population, heart transplant recipients are at high risk for development of persistent or recurrent CHF symptoms, premature CAD, reduced exercise capacity, graft rejection, and side effects from immunosuppressant medications, including opportunistic infection, reduced bone mineral density, and muscle atrophy.[37] Functional capacity during peak exercise is reduced 40% to 50% compared with age-matched control subjects, and there is a diminished heart rate and cardiac output response to exercise. Exercise training in a supervised setting has been shown to reduce some of these abnormalities.[21] Exercise tolerance varies widely among patients and recommendations regarding the safety of participation in wilderness activities should be made on a case-by-case basis. Because transplant recipients are predisposed to opportunistic infection, they should take special precautions, including water boiling and strict attention to dental hygiene. They may consider bringing a medication kit along on an expedition that includes broad-spectrum antibiotics, diuretics, and additional immunosuppressants in the event they develop signs or symptoms of infection or acute rejection in the wilderness.

Additional Screening Procedures before Diving

Congestive Heart Failure

Patients with clinical signs or symptoms of CHF are at high risk of developing pulmonary edema with immersion and should therefore be excluded from diving. Patients with a history of cardiomyopathy and a reduced ejection fraction with no clinical symptoms of CHF are also at high risk of pulmonary edema and arrhythmia and should generally also be excluded. If these patients elect to dive against medical advice, they should be encouraged to limit dive stress and dive only in warm water.

Arrhythmia, Internal Cardioverter-Defibrillators, and Pacemakers

Benign supraventricular arrhythmia is a common occurrence with diving and should not be a contraindication to repeat diving. These patients should be encouraged to avoid decongestants, caffeine, tobacco, and alcohol before diving because these stimulants can precipitate arrhythmias. Chronic atrial fibrillation should exclude individuals from professional diving, but does not exclude sport diving at shallow depths. These patients are frequently on chronic anticoagulation and should be instructed to be especially careful to avoid trauma. Ventricular arrhythmias, such as premature ventricular contractions and short runs of nonsustained ventricular tachycardia, may also be benign in healthy individuals when these occur during diving, but there should be a low threshold for initiating a cardiac evaluation if arrhythmias are documented despite avoidance of stimulants. Conversely, in patients with a history of CAD or CHF, ventricular arrhythmias can lead to SCD, so a history of ventricular arrhythmias in this setting should be a contraindication to diving. Patients with ICDs should not participate in diving because these devices are implanted in patients at high risk of or with a known history of ventricular arrhythmias and SCD. Any patient with recent or recurrent syncope (fainting) should also be restricted from diving, particularly if a cardiovascular cause is suspected.

Left or right bundle branch blocks, first-degree heart block, and type I second-degree heart block are not contraindications to diving. Type II second-degree heart block and third-degree heart block are contraindications. These patients should be referred to a cardiologist for further care and consideration of a pacemaker.

The presence of a pacemaker usually implies underlying cardiovascular disease that may exclude a patient from diving. Pacemakers differ by manufacturer in their ability to withstand external pressure. It is advised that the patient or physician consult the available literature accompanying their device before diving to verify the depth tolerance. Pacemakers that feature a piezoelectric pressure sensor for rate responsiveness may misinterpret the pressures associated with submersion as increased activity, resulting in unnecessary tachycardia during diving. All patients with a pacemaker should have their devices interrogated before participation in diving.

Congenital Heart Disease

Because of the abnormally low arterial oxygen content in patients with cyanotic heart disease, these patients should be restricted from diving. Among the acyanotic congenital heart defects, intracardiac shunts are of particular relevance to diving. Atrial septal defects (ASDs) and patent foramen ovale (PFO) are typically characterized by a left-to-right shunt at sea level. As mentioned previously, diving causes increased intrathoracic, right atrial, and RV blood pressures that can cause transient right-to-left shunting. Precipitation of gas bubbles in the bloodstream with decompression may result in paradoxical embolization of gas bubbles or thrombus in the setting of an intracardiac right-to-left shunt. This may cause severe decompression sick-

ness or stroke. Several studies indicate that the incidence of PFO is higher among patients with severe decompression sickness than among the general population.[27,45] Although routine screening for asymptomatic PFO is not recommended before clearance for diving, recurrent or severe decompression sickness should prompt a search for PFO. In a small study involving six divers with a history of severe decompression illness, closure of the PFO by a percutaneous closure device resulted in no further episodes of decompression sickness on subsequent dives.[43] Therefore, it seems reasonable to grant clearance to patients with surgical or percutaneous closure of a PFO, assuming there is no residual shunt documented on echocardiography. Unrepaired PFO or ASD should exclude a patient from diving.

THERAPEUTIC MANEUVERS FOR CARDIAC EMERGENCIES

Numerous studies have documented the importance of short response times for the treatment of acute coronary syndrome and cardiac arrest.[34] In the event of a cardiovascular emergency, it is critical to evacuate the patient to an appropriate care facility as soon as possible. Almost by definition, wilderness adventure takes place away from the advanced medical care facilities that are available in populated areas, making fast response times difficult, if not impossible. Bedside diagnosis and rudimentary treatments may be the only available therapeutic maneuvers for extended periods of time. This section reviews basic bedside diagnosis and the treatments available for use in wilderness cardiovascular emergencies.

Syncope and Cardiac Arrest: A Summary of the ABCs of Basic Life Support

The initial evaluation of a patient who unexpectedly collapses focuses on confirming that the collapse is related to cardiac arrest.[34] The responder should first evaluate for response to voice or tactile stimulation. He or she should then palpate a major artery for the presence of a pulse while simultaneously checking for breathing effort and skin color. If a pulse is not palpable, the responder should instruct bystanders to begin efforts to contact professional medical responders immediately and start basic life support (BLS; Fig. 27-6).

If the patient appears to be struggling for breath but still has a pulse, the collapse is likely related to a respiratory cause such as choking. The Heimlich maneuver or clearance of the airway should be attempted, along with the provision of two breaths by mouth-to-mouth resuscitation (see later). Conversely, if the patient has no breathing effort and no pulse, the etiology may be either respiratory or cardiovascular and BLS should be initiated.

Precordial Thump

Given the importance of immediate defibrillation to outcome in the setting of cardiac arrest, the responder may initially attempt a precordial thump. It has been recommended that the use of precordial thump be restricted to advanced cardiac life support (ACLS); however, in the wilderness setting, time to defibrillation is likely to be prolonged and an early precordial thump may improve outcome. In one prospective study in 5000 patients, precordial thump successfully reverted ventricular fibrillation in 5 events, ventricular tachycardia in 11, asystole in 2, and undefined cardiovascular collapse in 2 others in which

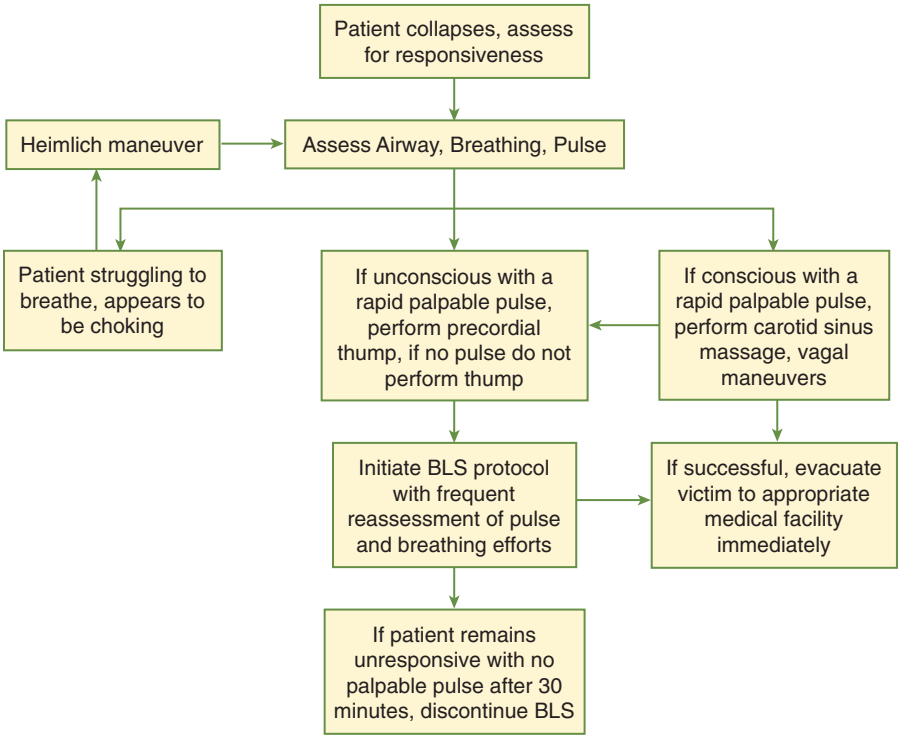


Figure 27-6. A response algorithm for evaluation and treatment of a person who collapses in the wilderness. BLS, basic life support.

the electrical mechanism was unknown. In no instance was conversion of ventricular tachycardia to ventricular fibrillation observed.[6] The precordial thump should be attempted only on an unconscious patient with ventricular fibrillation or a rapid palpable pulse consistent with ventricular tachycardia. Patients without a pulse are more likely to be in asystole or pulseless electrical activity, rhythms that are not typically responsive to defibrillation. Time should not be wasted on a precordial thump in these patients, and resuscitative efforts should be focused on initiation of BLS.

Technique. The patient's chest should be bared for examination of anatomic landmarks. From a height of 20 to 25 cm (8 to 10 inches) above the chest, one or two firm blows should be delivered to the middle to lower third of the sternum with a closed fist. A major artery should again be palpated for a pulse. If unsuccessful after one to two blows, the effort should be abandoned in favor of reinitiating the BLS protocol.

Basic Life Support

BLS is intended to maintain blood flow to the critical organs (central nervous system and heart) until definitive management at a hospital can be arranged. If a responder is alone with the victim, initial efforts should be made to contact help. In all other cases, BLS should be initiated immediately.

Airway (A). Clearing the airway is critical to the success of any resuscitation attempt. The patient's head should be tilted back and the chin lifted to maintain a patent airway. The mouth should be inspected for foreign bodies, including dentures, and these should be removed. If a foreign body is lodged in the airway, the Heimlich maneuver should be attempted. This is performed by wrapping the arms around the abdomen from the back and delivering several forceful thrusts inward and upward. If the rescuer is alone, the Heimlich maneuver can also be performed from an anterior position by placing a fist on the abdomen and delivering forceful thrusts inward and upward from a position above the patient. If the patient dislodges a foreign body and there is a strong suspicion that a respiratory arrest precipitated cardiovascular collapse, a second precordial thump may then be attempted before continuing BLS.

Breathing (B). After the airway is cleared, mouth-to-mouth resuscitation should be attempted. The rescuer positions himself at the side of the patient with his head positioned perpendicular to the victim's head. A seal is made between the lips, and two long forceful breaths should be administered. In two-rescuer BLS, a ratio of one breath per five chest compressions should then be continued. If the rescuer is alone, he or she should alternate 2 breaths for every 30 chest compressions.

Circulation (C). Chest compressions are intended to promote forward blood flow by repeatedly increasing and decreasing intrathoracic pressure. The heel of one hand is placed on the victim's lower sternum with the heel of the other hand resting on the back of the lower hand (Fig. 27-7). The arms should be

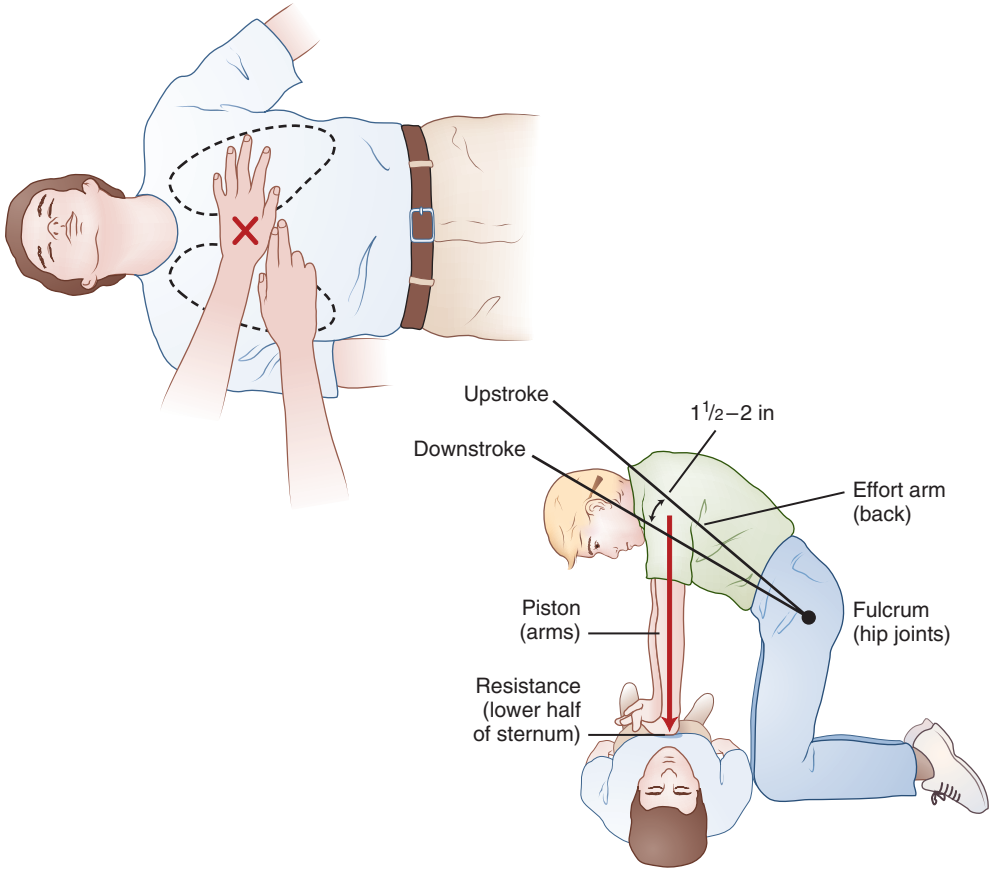


Figure 27-7. External chest compression. *Left*, Locating the correct hand position on the lower half of the sternum. *Right*, Proper position of the rescuer, with shoulders directly over the victim's sternum and elbows locked. (From Standards and guidelines for cardiopulmonary resuscitation [CPR] and emergency cardiac care [ECC]. JAMA 255:2905–2989, 1986, with permission.)

straight and the rescuer should bend at the hips to provide downward force on the chest wall. The rescuer should provide 100 chest compressions per minute and each compression should depress the chest wall 3 to 5 cm.

Duration of Cardiopulmonary Resuscitation

The success of BLS is largely related to the availability of access to ACLS facilities and equipment. Patients who do not respond during the first 15 to 30 minutes of BLS are very unlikely to recover any significant degree of brain function, even if a pulse and respiratory efforts are returned. In the urban environment, emergency medical services personnel are instructed to continue BLS until (1) spontaneous ventilation and circulation are restored, (2) the patient's care is transferred to another person who continues BLS, (3) the patient is pronounced dead by a physician, or (4) the rescuer is exhausted and unable to continue resuscitation.[35] A new wilderness protocol was proposed in the early 1990s that modified these instructions to maximize safety for the rescuer and the patient.[14] In the normothermic patient, rescuers should discontinue BLS after 30 minutes if a spontaneous pulse has not been restored. In the hypothermic patient, BLS should not be initiated if it would put rescuers at risk, if the core body temperature is less than 15° C (60° F), if the chest wall is frozen, if the victim has been submersed in cold water for more than 60 minutes, if obvious lethal injury is present, or if BLS would significantly delay evacuation to controlled rewarming.[14]

Treatment of Tachyarrhythmia in the Conscious Patient

Sudden-onset tachyarrhythmias may originate in the atrium, atrioventricular (AV) junction, or ventricle and may be a physiologic or pathologic response to the stress of a wilderness environment. As mentioned previously, extreme environments can stimulate abnormal heart rhythms in structurally normal hearts. The differentiation between normal sinus tachycardia and an abnormal tachyarrhythmia can be difficult in the wilderness setting. Typical symptoms of an abnormal tachyarrhythmia include palpitations, lightheadedness, chest pain, or new-onset symptoms of CHF. A medical history of Wolff-Parkinson-White syndrome, atrial fibrillation, or other known arrhythmia can be helpful. A brief examination of the patient's pulse should evaluate the regularity, rate, and beat-to-beat intensity. Evaluation of the jugular venous pulse may reveal cannon A waves, disclosing AV dissociation. Direct auscultation of the heart by simply placing the ear on a patient's bare chest can disclose a paradoxically split second heart sound, characteristic of the left bundle branch block that accompanies ventricular tachycardia, or supraventricular tachycardia with aberrancy.

If the patient is symptomatic, initial efforts should be made to seek professional medical assistance and transportation to medical facilities. While awaiting help, several therapeutic maneuvers can be attempted.

Carotid Sinus Massage

Carotid sinus massage can be both diagnostic and therapeutic for several types of tachyarrhythmias. This technique works by increasing vagal tone and slowing the rate of sinus nodal discharge, as well as increasing AV nodal conduction time and refractoriness. AV nodal reentrant tachycardia and AV reciprocal tachycardia will slow abruptly and frequently terminate with carotid sinus massage. Rapid ventricular response to atrial flutter and atrial fibrillation will slow momentarily but will not terminate. Rarely, ventricular tachycardia can be terminated by carotid sinus massage.

Technique. The patient should be placed in a supine position with the neck fully extended and turned away from the rescuer. The carotid pulse should be palpated at the angle of the jaw and, if palpable, the bifurcation should be localized. Firm pressure should then be applied with a gentle rotating motion for 5 seconds. If there is no response, the procedure should be attempted on the patient's other side. The procedure should never be performed on both sides simultaneously. External pressure on the carotid sinus stimulates baroreceptors that trigger increased vagal tone and decrease sympathetic outflow, potentially terminating the arrhythmia.

Vagal Maneuvers

In patients who do not respond to carotid sinus massage, an attempt at various alternative vagal maneuvers may terminate certain tachyarrhythmias. The diving reflex can be reproduced by suddenly dunking the patient's head in cold water. Breath holding or a strong cough against a closed glottis may also cause a strong vagal response that can terminate arrhythmia. Last, pressing on the closed eyelids can stimulate the ophthalmic branch of the trigeminal nerve. This elicits a strong vagal response, capable of terminating certain tachyarrhythmias. One must exhibit appropriate caution when massaging the carotid sinus or compressing the eyeball of an elder patient.

SUMMARY

A wilderness environment places unique stressors on the cardiovascular system. Appropriate cardiovascular screening before departure on a wilderness excursion can help ensure a safe and enjoyable adventure. Should an emergency arise in a wilderness environment, emphasis should be placed on the rapid evacuation of the victim to an appropriate medical care facility. BLS and vagal maneuvers can be successful in the short-term stabilization of certain cardiovascular emergencies.

The references for this chapter can be found on the accompanying DVD-ROM.

28 Wilderness Neurology

Karen Nolan Kuehl and Donald B. Nolan

Neurology comprises an entire specialty of medicine and includes disorders of the central nervous system (CNS) and peripheral nervous system. The CNS includes the brain and spinal cord, whereas the peripheral nervous system comprises motor and sensory nerves to the remainder of the body. The peripheral system includes the nerve roots of the brain and spinal cord, the brachial and lumbosacral plexuses, and the peripheral nerves that provide innervation to end organs and muscles. Neurologic disorders may affect one or both of the central or peripheral nervous systems. A neurologic problem may be caused by a primary neurologic disorder or could be the secondary result of an infection, medication, or trauma.

Although many of the individuals traveling to wilderness regions are relatively healthy, it is not uncommon for travelers to be affected by some of the more common neurologic problems, particularly headaches, seizure disorders, and trauma. In addition, as technology has improved, more individuals with chronic neurologic disorders are remaining active, traveling, and participating in wilderness activities. Medical providers in the wilderness should be able to recognize common neurologic problems and know when to transport these patients to a higher level of care.

NEUROLOGIC EXAMINATION

Each patient with a neurologic problem or complaint should be approached in a similar fashion, beginning with a thorough history and physical examination. An adequate neurologic history and examination can be achieved in a wilderness setting without availability of elaborate equipment or a neurologist. Focusing the history can limit the differential diagnosis, and should begin with questions about the complaint, the duration of symptoms, preceding symptoms, past medical history and medications, and other pertinent information such as recent travel history or hospitalizations. Attempt to obtain the patient's history, but use other resources as well, including family, available medical records, and medical alert jewelry.

At a minimum, the neurologic examination should include three elements that can provide a great deal of information about the patient's neurologic status. Many neurologic deficits may be elicited simply by listening to the patient speak, watching the patient walk, and looking at the patient's eyes. Deficits in one of these areas should lead to an additional, focused examination.

The neurologic examination should begin with a general physical examination, including vital signs. Abnormal vital signs may provide an answer to the cause of the neurologic complaint. For example, seizures may be caused by hyperthermia or fever, or altered mental status may be secondary to hypothermia, hypotension, or hypoxia. A generalized physical examination, including assessment of the head, neck, heart, lungs, and extremities, may give clues about the etiology of the neurologic problem. For example, bruising of the scalp may indicate head trauma, tongue laceration or urinary incontinence may indicate a recent seizure, neck stiffness may be a sign of meningitis, and extremity weakness could suggest a stroke or spinal cord injury.

A focused neurologic examination should begin with assessment of the patient's mental status, which can begin by obtaining a history from the patient. If the patient is able to provide an accurate history of the problem and give specifics about medications, symptoms, and past medical history, a fairly intact mental status can be assured. If the patient is confused, additional questions should be asked pertaining to orientation, including the person's name, the current location, and the time and date. Speech and memory should be assessed as well during this portion of the examination.

If a patient is altered in consciousness or comatose, alternative assessment methods must be used to grade severity of mental status deficits. Altered levels of consciousness should be noted in terms of severity using the following scale:

1. Lethargy—malaise
2. Obtundation—impaired function but awake
3. Stupor—arousable to stimulation
4. Coma—unresponsive to stimulation

The Glasgow Coma Scale (GCS) is a more detailed method of determining brain activity and is a standard formula, particularly when following mental status of a patient over a prolonged period of time, such as during extrication or transport to definitive care (Table 28-1). The GCS examination includes a scoring mechanism based on eye opening, verbal function, and motor function and should be followed on a serial basis.

In a medical facility, the necessary equipment for a complete neurologic examination includes an ophthalmoscope/otoscope, tuning fork, and percussion hammer. In the wilderness, assessment may include only mental status, pupillary reaction, facial symmetry, extremity strength, sensory examination, and gait (Box 28-1).

Although all 12 cranial nerves may be difficult to test in the backcountry, it is critical to examine the pupils for reactivity and equal size, as well as examine extraocular movements in all directions. In addition, note any facial asymmetry or abnormalities of speech, hearing, or smell.

TABLE 28-1. Glasgow Coma Scale (GCS)

Eye Opening		
Spontaneously	4	Eyes open
To verbal command	3	Will open to command
To pain	2	Will open to pain
None	1	Will not open
Verbal Response		
Oriented–conversant	5	Mostly intact CNS
Disoriented	4	Not oriented
Inappropriate words	3	Random words
Incomprehensible	2	Moaning, no coherent words
No response	1	No response/intubated
Motor Response		
Obeys commands	6	Moves limbs as ordered
Localizes to painful stimuli	5	Moves limb away from pain
Flexion withdrawal	4	Pulls from pain with flexion
Abnormal flexion	3	Decorticate posture
Extension	2	Decerebrate posture
No response	1	Flaccid; may indicate paralysis or loss of brain function

CNS, central nervous system.
The highest (best) possible score is 15.

Box 28-1. Neurologic Backcountry Medical Kit

EQUIPMENT
Thermometer
Blood pressure cuff
Pulse oximeter
Otoscope or alternative light source
Dermatome map
Glucometer

MEDICATIONS
Glucose (honey, glucose packets, glucagon, Glutose)
Analgesics
- Acetaminophen (adult and children's)
- Ibuprofen (adult and children's)
- Hydrocodone

Headache treatments
- Sumatriptan or other triptan (oral, dissolving, nasal)
- Anti-emetics (oral, dissolving, IV)

Antiseizure medications
- Benzodiazepines (rectal, oral, IV)
- Phenytoin (oral, IV)

Antibiotics
- Ceftriaxone (IV)
- Ampicillin (IV)
- Amoxicillin
- Azithromycin
- Levofloxacin

Antiviral
- Acyclovir (oral, IV)

Corticosteroids
- Prednisone
- Methylprednisolone

IV, intravenous.

Box 28-2. Types and Causes of Headache

Types
Migraine
Cluster
Tension

Causes
Dehydration
High altitude
Subarachnoid hemorrhage
Sinusitis
Temporomandibular joint pain
Tumor
Postconcussive

Each extremity should be examined for abnormalities in strength and sensation. When examining an extremity, it should be taken through its full range of motion, including flexion and extension at each joint to evaluate strength. Abnormalities should be localized to a specific muscle group, specifically the flexors or extensors. If strength is adequate and the patient can walk, gait testing gives a good assessment of cerebellar function. Inability to walk a straight line should be noted, as should the tendency to fall to one side or the other. Abnormalities in gait may indicate a lesion in the cerebellum.

Sensation should be assessed in each extremity for discrepancies in light touch, pain, temperature, and position sense. Tools for testing sensation of pinprick and hot/cold require only imagination. Asking the patient to close his or her eyes and move a digit in a specific direction may test position sense. Any deficits should be localized to a spinal cord level or a dermatome. For example, if a patient has no sensation to light touch or pain below the nipples, a lesion at T4 (the spinal cord between thoracic vertebrae 4 and 5) may be diagnosed (see Figure 20-3).

Deep tendon reflexes should be checked at the wrists, elbows, knees, and ankles, and should be symmetric and equal throughout. Any loss of reflexes may indicate a brain, spinal cord, or focal extremity injury. A reflex hammer is not required to test reflexes; alternatives include a stethoscope, a rounded, heavy stick, or even stiff fingers.

HEADACHE

Headaches are a common neurologic complaint and may represent a primary problem or be a symptom of something else (Box 28-2). More than 90% of all headaches can be diagnosed as migraines, tension, or cluster headaches, or due to dehydration or acute mountain sickness, and the majority of these can be treated effectively and easily in the backcountry.[12] Treatable, benign headaches must be differentiated from more serious, and potentially lethal, causes. Early diagnosis can prevent devastating outcomes when transport to appropriate medical care is delayed or unavailable. All patients with headache should be watched closely and encouraged to reduce activity until symptoms are improved. Evacuation should occur for any headache, despite etiology, if the patient has worsening pain, persistent vomiting, changed mental status, or generalized worsening of clinical status despite treatment.

Migraines

Migraines are a frequent cause of headaches in emergency departments and may be a chronic problem affecting otherwise healthy individuals. Migraines most often begin in young adults in their third and fourth decades, and are approximately three times more common in women than in men. It has been estimated that 18% of the general population will be affected by a migraine during their lifetime.[5]

Clinical features of a migraine can vary among patients but typically include a throbbing, unilateral headache associated with nausea, vomiting, and sensitivity to light (photophobia) and sound (phonophobia). Approximately 15% of individuals have a headache preceded by an aura of neurologic phenomena, which include changes in vision such as blurry vision, visual field cuts, or flashes of light. Rarely, individuals may have associated numbness or weakness, or problems with speech or cognition.

Diagnosis of a migraine headache can usually be made with a thorough history. Migraine sufferers typically have a prodrome of some kind, which may be a classic aura or may only be fatigue. The migraine headache is usually of gradual onset, and in most patients leads to a unilateral, pulsating headache. However, 40% of migraine patients describe a bilateral headache. Untreated, migraine headaches last from 4 to 72 hours and have been known to last weeks.[8] Diagnosis of a migraine is aided by a personal or family history of migraines. Differentiation of a migraine from a tension-type or cluster headache is made by the presence of nausea and vomiting, pulsating quality, photophobia and phonophobia, and aggravation of symptoms by physical activity.[10] Migraine headaches may mimic transient ischemic attacks (TIAs) and other cerebrovascular events, particularly when temporary neurologic deficits such as visual changes or numbness occur, and TIA should be strongly considered in middle-aged and elderly patients with no personal or family history of migraines. Although routine neuroimaging, such as head computed tomography (CT) or magnetic resonance imaging (MRI), is not required for all individuals with suspected migraines, it must be considered for those with neurologic deficits or with a new or changing headache.[13]

Treatment of migraine headache consists of both physical and pharmacologic interventions. Migraines may be prevented by regular sleep and meals and adherence to a diet that avoids dietary triggers such as caffeine, aged cheeses, and red wine. Patients with frequent migraines may take preventive medications, including beta-adrenergic blockers, epilepsy medications, and antidepressants. Acute migraines can be treated by over-the-counter and prescription medications. Over-the-counter medications, including ibuprofen, acetaminophen, and aspirin, have all shown effectiveness when taken in adequate doses.

Initial management of a patient with migraine includes placement in a quiet, dark area, such as a shaded tent, to minimize external stimuli. Patients should be encouraged to drink fluids, if possible, so that any dehydration component is removed. Ibuprofen 600 mg, acetaminophen 975 mg, and aspirin 324 mg are excellent first-line options for treatment, as long as the patient has no contraindications to these medications. Caffeine in some form (e.g., coffee, soda, as an additive in some over-the-counter medications) may be a helpful adjunct.

Prescription migraine medications vary in effectiveness and appropriateness. Narcotic medications have been shown to cause both a rebound effect and the possibility of addiction, and they should be avoided if possible. Ergot compounds have been used for decades but have questionable efficacy in many patients. Dihydroergotamine (DHE 45; Migranal) comes in a formulation that may be given intravenously (IV), intramuscularly (IM), and subcutaneously (SC), at a dose of 1 mg. This dose may be repeated after 1 hour if not effective, but it should not exceed 2 mg IV or 3 mg IM/SC in 1 day. There is a convenient intranasal formulation (0.5 mg/spray) that may be used up to 6 sprays/day.

The triptan class of medications, although more costly, have been shown to have more efficacy and fewer side effects than the ergot precursors. These medications, such as sumatriptan (Imitrex), naratriptan (Amerge), rizatriptan (Maxalt), and zolmitriptan (Zomig), come in various formulations. Tablet forms are the most durable for travel but may be ineffective in vomiting patients. Dissolving tablets, intranasal solutions, and SC formulations are excellent options for patients who cannot tolerate anything by mouth. Sumatriptan may be given 6 mg SC, 25 to 100 mg orally (PO), or as a 5- or 20-mg nasal spray. Doses may be repeated in 1 hour if not effective to a total of 12 mg SC, 200 mg PO, or 50 mg nasally in 1 day. Naratriptan comes in an oral formulation only in 1- and 2.5-mg tablets, with a maximum of 5 mg daily. Rizatriptan comes in tablet and oral disintegrating forms of 5 and 10 mg, with a maximum of 30 mg/day. Zolmitriptan also comes in tablet and oral disintegrating forms in 2.5- and 5-mg strengths, and may be given at a maximum dose of 10 mg/day.

The longer migraines go untreated, the less likely they are to respond to oral triptans. Patients who wake with a migraine would be best served with intramuscular triptan; patients who fail to respond to the first oral triptan may respond to intramuscular triptan for the next dose. The mechanism of action of the triptans is unclear but is theorized to be through vasoconstriction and reduced neural pain transmission. Side effects include flushing, tingling, and dizziness. There has been an incidence of angina symptoms and even myocardial infarction in some triptan users, so these medications should not be used in patients with cardiac disease.[5] They should also not be used in patients with risk of cerebrovascular disease, peripheral vascular disease, uncontrolled hypertension, or severe liver disease.

Antiemetics have long been used as a successful addition to migraine medications. Prochlorperazine (Compazine) 5 to 10 mg may be given PO, IM, or IV every 6 hours. A rectal suppository (25 mg) may be used every 12 hours. Promethazine (Phenergan) 12.5 to 25 mg may be given PO/IV/IM/rectally every 6 hours, but the lower dose should be tried first to avoid oversedation. Odansetron (Zofran) 4 mg may be given PO or IV every 8 hours and may be repeated if necessary. In addition, odansetron has recently been released in a wafer form of 4 and 8 mg, and it easily dissolves when placed on a patient's tongue. Although expensive, the wafers may be an excellent option to carry into the backcountry.

Tension Headaches

Tension headaches present after or during stress and may be associated with pain in the neck and shoulders resembling muscle tightness. These headaches are the most common type of headache, and occur in most individuals during their lifetime (prevalence in men is 69%, and 88% in women). Prevalence decreases with age. Tension headaches are not increased in persons with depression or anxiety disorders.

Tension headaches have gradual onset and worsen as the day progresses. The headache is typically bilateral and is described as viselike with pain in the forehead, temples, and occiput.

There should be no associated gastrointestinal symptoms or problems with sound or bright light. Tension headaches are commonly confused with migraine headaches, and vice versa, but must also be differentiated from brain tumor and sinusitis.

In a hospital setting, patients may be released without neuroimaging if they have a history of similar headaches and a negative neurologic examination. However, head CT or MRI should be completed in all patients with severe, unremitting headaches, neurologic changes, or a significantly different character to the headache.

Most commonly, patients use over-the-counter medications such as acetaminophen or ibuprofen as treatment and may present for medical advice only when the headache is unresponsive to common treatments. Although narcotics may help acutely, these must be limited, if used at all.

Cluster Headaches

Cluster headaches are a specific type of unilateral headache, commonly associated with eye and nasal symptoms. Symptoms include eye redness and tearing, nasal congestion, rhinorrhea, and eyelid edema. Cluster headaches occur with a male-to-female ratio of 6:1, and commonly develop in young adults. Commonly, these headaches occur in a "cluster" pattern, in that they are short-lived but recurrent and severe.

Diagnosis of cluster headaches is made by history and presence of associated eye and nasal symptoms. The headache is unilateral, severe, and short, lasting 30 to 90 minutes. During the attack, symptoms may radiate into the eye, jaw, neck, or shoulder. The attacks may occur multiple times daily, and are not usually associated with nausea or vomiting.

Differential diagnosis includes migraine, tension headaches, dehydration, high-altitude cerebral edema or headaches, and glaucoma. Other rare diagnoses include intracranial hemorrhage, trigeminal neuralgia, and temporal arteritis. A careful history is the most effective way of making the diagnosis, and neuroimaging such as head CT or MRI is not required unless there is suspicion of an intracranial lesion.

Treatment includes supplemental oxygen by face mask at 8 to 10 L/min and over-the-counter remedies such as acetaminophen and ibuprofen. Migraine treatments such as DHE and the triptans have been shown to help some patients. Because the headaches are usually brief and self-limited, prevention of future attacks is more helpful than symptomatic treatment in most patients.[26]

Dehydration and Altitude Headaches

Dehydration and altitude headaches are a common complaint in areas of low humidity or elevated altitude. Their appearance is nonspecific and they often are found in an otherwise healthy individual, but are best diagnosed by exclusion. Dehydration and high altitude may increase the incidence of the other types of headache (migraine, tension, and cluster), and should be considered in all patients in the wilderness because hydration is usually a simple treatment with no negative outcomes. If high altitude is a possibility, a headache sufferer should either avoid further ascent until the headache is resolved, or descend until there is improvement. The response to hydration or reduced altitude may not be immediate but should be evident within 24 hours.

Subarachnoid Hemorrhage

Subarachnoid hemorrhage (SAH) accounts for 5% of all strokes and affects nearly 30,000 individuals per year in the United States. Saccular or berry aneurysms at the base of the brain cause nearly 80% of all cases of SAH. Approximately 5% to 10% of patients with SAH have a family history positive for brain aneurysm; therefore, all patients with a severe headache should be questioned about family history. There are a number of other causes of SAH, the most common of which is trauma. Twelve percent of patients die before receiving medical attention, and another 20% die after admission to the hospital.

Subarachnoid hemorrhage usually commences with an explosive "thunderclap" headache followed by neck stiffness. Patients describe the headache as the worst in their life. Common associated symptoms include loss of consciousness, nausea and vomiting, and photophobia. In patients who lose consciousness, posturing movements may occur and may be difficult to differentiate from a seizure. Other symptoms may include fever, which may make the diagnosis difficult to distinguish from meningitis. Any patient suspected of having potential SAH should be transported to an emergency department with cerebral CT or MRI capability; a lumbar puncture may be necessary in addition to imaging.[16]

Sinusitis

Sinusitis is inflammation of the linings of the sinuses that may be caused by viruses or bacteria, with a viral etiology being at least 20 times more likely than bacterial. Patients at high risk for sinusitis include those with recent upper respiratory infections, nasal polyps, recent facial trauma, immunocompromise, and recent nasotracheal intubation. Symptoms include facial and tooth pain, headache, purulent nasal drainage, fever, and halitosis. To make a diagnosis of acute bacterial sinusitis, patients should have had symptoms for longer than 7 days. Initial treatment should be decongestants and analgesics. Decongestant options include oxymetazoline hydrochloride sprays (2 to 3 sprays in each nostril twice daily) or pseudoephedrine 60 mg PO every 4 to 6 hours. If symptoms persist for 7 days despite conservative treatment, antibiotics should be started. Initial options for treatment include amoxicillin 500 mg PO three times daily for 14 days, with amoxicillin-clavulanate 500 mg PO three times daily reserved for those treated unsuccessfully with antibiotics in the previous month or who fail initial treatment. Options for persons with penicillin allergy include azithromycin 500 mg on day 1 followed by 250 mg daily for 4 days or levofloxacin 500 mg daily for 10 days. Patients with mental status changes, seizures, focal neurologic signs, persistent vomiting, or any cranial nerve abnormalities should be transported to definitive care immediately.

Temporomandibular Joint Pain

Pain in the temporomandibular joint presents as jaw pain but may cause ipsilateral ear pain or headache. The pain is aching and dull and may be worsened at night. Diagnosis is made when movement of the mandible exacerbates symptoms. Treatment includes nonsteroidal anti-inflammatory drugs and narcotics, as well as reassurance that symptoms will improve.

Tumor

Tumor as a cause of headache is a diagnosis of exclusion and should be placed in the list of differential diagnoses for headaches. This headache is usually present for weeks or longer. It is often worse in the morning, and may worsen with changes in position, sneezing, or exertion. Associated vomiting may occur with no preceding nausea. Abnormalities in the neuro-

logic examination may or may not be present. Diagnosis must made be with radiologic studies, preferably MRI.

SEIZURES

Seizure activity is caused by excessive activity of cortical neurons in the brain leading to synchronous, abnormal discharge. The resultant seizure activity depends on the area of the cortex involved. If the increased activity occurs in both hemispheres of the brain and appears to have no focus of activity, the seizure is "generalized" and associated with a loss of consciousness. If the increased activity has focal onset in one region of the cerebrum, it is considered to be "partial." There is no correlation between increased physical activity and increased seizure activity in epileptic patients. All patients with seizures should be discouraged from activities that would be dangerous to themselves or others if they were to suffer a seizure, which includes driving a vehicle or working around any equipment with moving parts. Activities such as free climbing, swimming alone, or skydiving[3] are contraindicated as well.

Generalized seizures may be further differentiated into convulsive or nonconvulsive seizures. Convulsive seizures involve tonic or clonic muscle activity with associated loss of consciousness. Generalized tonic-clonic seizures have also been termed *grand mal seizures*. Patients usually have a postictal period during which they may be sleepy or confused. Generalized seizures without any major motor activity may include absence seizures, small clonic movements, or drop attacks. Absence seizures, which usually begin in childhood, may present with an episode that resembles "daydreaming" but may have associated eye-blinking, rapid hand movements, or mouth chewing.

Partial seizures are further subdivided into simple partial and complex partial seizures. Simple partial seizures should have no associated loss of consciousness, although they may evolve into generalized seizures. Patients may have motor, sensory, or visual complaints and should have no postictal state. Complex partial seizures typically involve a specific movement or activity and are associated with some change in mental status. These may be preceded by an aura, which is a specific taste, smell, hallucination, or feeling of emotion.

Seizure activity has multiple etiologies and must be classified accordingly. *Epilepsy* is the proper term for a primary seizure disorder, and occurs when an individual has recurrent seizures secondary to an underlying process. Approximately 5% to 10% of the U.S. population will suffer a seizure during their lifetime, but less than 1% have epilepsy.[22] Causes of seizure activity vary in frequency, according to the age of the patient (Box 28-3).

The most common causes of seizure activity can be summarized by age group. Newborns and infant seizures are often caused by hypoxia, infection, trauma, intracranial hemorrhage, maternal drug use, or congenital CNS abnormalities.

Seizures in young children are most often caused by fever, and are called *febrile seizures*. Febrile seizures occur in children between 3 months and 5 years of age, and are associated with the fever itself, not a CNS infection. A simple febrile seizure is an isolated, short event. Complex febrile seizures occur when there is more than one seizure, when a seizure lasts more than 15 minutes, or when the seizure localizes to one area of the body.[15] Children between the ages of 6 and 18 months need careful consideration for meningitis because they may not exhibit the typical signs of meningitis such as mental status changes, vomiting, and neck stiffness. Children older than 18 months of age with a suspected febrile seizure usually do not require additional evaluation if they appear well, and they may be managed conservatively by treatment of the fever with acetaminophen (15 mg/kg every 4 to 6 hours) or ibuprofen (10 mg/kg every 8 hours).

Epilepsy may present during childhood and should be investigated if more than one seizure occurs or if there is no associated fever. Other common causes in children include brain tumor, infection, or head trauma. In older individuals with late-onset seizures, additional causes include cerebrovascular disease, tumor, intracranial hemorrhage, or metabolic derangement. In all age groups, one must consider the possibility of medication effects, illicit drug use, withdrawal from alcohol, renal failure, hepatic failure, hypoglycemia or hyperglycemia, or physical abuse.

The approach to a patient with a seizure will be similar in the wilderness to the approach followed in the emergency department. The most important considerations include controlling the airway and protecting the patient from additional harm. If a patient has stopped seizing, a thorough history and physical examination should be completed to seek the initial cause of the seizure. The patient should be questioned about seizure history, recent medical problems, use of medications or illicit drugs, alcohol ingestion, or head trauma. Physical examination should include vitals signs with temperature, neurologic evaluation, examination of the head and neck, and a thorough examination for systemic illness. If the patient has a new diagnosis of seizure, he or she requires a brain CT, laboratory evaluation for electrolyte abnormalities, glucose check, and possibly a lumbar puncture for cerebrospinal fluid (CSF) analysis. If the patient has a known history of seizures, a serum level of anticonvulsants may be required to evaluate for noncompliance or inadequate dosing of medications. Any patient with a first-time seizure should be transported to a medical facility for further evaluation. Those patients with a known history of seizures who have a seizure in the backcountry may not require additional evaluation but should be monitored closely to ensure medica-

Box 28-3. Causes of Seizure

ADULTS
Trauma
Alcohol withdrawal
Illicit drug use
Metabolic abnormalities (low glucose, low sodium)
Infection
Brain tumor
Idiopathic

CHILDREN
Trauma
Infection
Developmental abnormalities
Metabolic abnormalities
Brain tumor
Idiopathic

tion compliance and for the development of any repeat or persistent seizure activity. Once a seizure has occurred in the backcountry, the individual should be prevented from participating in potentially dangerous activities such as scuba diving, swimming alone, lead climbing, or driving an all-terrain vehicle until cleared by a neurologist. These individuals could harm themselves and endanger others if an additional seizure occurred while they pursued an unsafe activity.

If the patient is actively seizing, the airway should be protected while an attempt is made to correct the cause of the seizure. Status epilepticus is a true emergency defined as continuous seizure activity lasting longer than 30 minutes or as two or more sequential seizures without a return to normal mental status between seizures. If status epilepticus is not treated quickly, death or major disability can result from hypoxia, respiratory or cardiac arrest, lactic acidosis, renal failure, or electrolyte abnormality.[27]

A seizing patient should be assessed for airway patency and receive supplemental oxygen if available. Fingers and other foreign bodies should *not* be inserted into a patient's airway to avoid tongue biting because the risk of aspiration or additional injury is high. Glucose or fructose in the form of honey, juice, or sugar granules should be provided immediately for mucosal absorption if hypoglycemia is a possible cause of the seizure. These may be placed directly on the patient's tongue or gums in small amounts, taking care to avoid injury to the care provider. Glutose gel (concentrated glucose in a tube) may also be used.

Benzodiazepines such as lorazepam (Ativan) and diazepam (Valium) are first-line agents against seizures. Intravenous administration is preferred but alternatives include rectal (Diastat), intramuscular, and mucosal administration if options are limited in the backcountry. Valium 5 to 10 mg IV or Ativan 2 to 4 mg IV has been used to stop seizures in most patients. However, rectal diazepam (Diastat) may be the only alternative in status epilepticus in the backcountry and may be given in doses of approximately 0.2 to 0.5 mg/kg. Side effects include respiratory depression and sedation. Do not force any oral medication in a patient having a seizure, but if the patient is experiencing lucid intervals between seizures and his or her usual medications are available, giving oral medications may prevent frequent seizures from turning into status epilepticus.

Other anticonvulsants may be used if there is no response to benzodiazepines. Phenytoin (Dilantin) is a common medication taken by many patients with epilepsy. Intravenous phenytoin or fosphenytoin should be given to patients with multiple seizures or in status epilepticus at a dose of 18 to 20 mg/kg. In addition, phenytoin may be given IV as seizure prophylaxis during transport of a patient with a severe head injury. Other common seizure medications include oral phenobarbital, oral valproic acid (Depakote), and oral carbamazepine (Tegretol). A patient may take his or her own medications if the seizure has stopped and the patient is sufficiently alert for oral administration.

Immediate transport out of the backcountry should be arranged for anyone with status epilepticus, seizure after a head injury, first-time seizure, seizure associated with headache, or seizure with associated fever in an adult or young child (<18 months). An older child (>18 months) with a suspected febrile seizure should be transported out of the backcountry in an urgent fashion, as opposed to emergently, in case additional seizures occur or the child becomes more ill.

INFECTION

CNS infections are rare but important syndromes that can present in the wilderness setting and need to be recognized quickly to avoid serious morbidity and mortality. The most common infectious processes are meningitis and encephalitis caused by bacteria, viruses, parasites, or fungi.

Meningitis

Meningitis is an infection in the CSF surrounding the brain and often occurs after colonization of the nasopharyngeal tissues by the infecting organisms. Other infectious routes include severe bacterial sinusitis, ear infections, or recent neurosurgical procedures. Persons at highest risk for illness include children, particularly those without childhood immunizations; the immunocompromised, including those with human immunodeficiency virus (HIV) infection, cancer, or alcoholism; long-term steroid users; and patients on hemodialysis. However, healthy adults can become sick with meningitis and, if traveling in a developing country, may develop meningitis from an unusual organism. In the United States, typical causative bacterial organisms include *Haemophilus influenzae, Neisseria meningitidis,* and *Streptococcus pneumoniae.*

Patients with meningitis commonly have severe headaches, fever, neck stiffness, and vomiting. Mental status changes, photophobia, and body aches (myalgias) also occur. Signs and symptoms may develop in 24 hours or less, or may develop over days. A rash with concomitant petechiae and purpura can signal *N. meningitides* meningitis, which may rapidly progress to death. In 5% of patients, there may be focal neurologic signs such as seizures or a dilated pupil, which worsen the prognosis of these patients.[1]

The only way definitively to diagnose meningitis is by lumbar puncture, which should be done under sterile conditions, so that a laboratory can analyze the CSF. Although it would be preferable to obtain CSF before the patient receives antibiotics, prophylactic antibiotics should be given in suspected cases of meningitis, particularly in wilderness situations when a lumbar puncture cannot be completed. Prophylactic antibiotics should be started in any patient with severe headache, mental status changes, neck stiffness, and fever. Antibiotic treatment should be focused on organisms most likely to cause meningitis, and should preferably be given within the first hour of symptoms to prevent possible neurologic sequelae. A third-generation cephalosporin such as ceftriaxone (1 g IV every 8 hours) with the addition of ampicillin (2 g IV every 4 hours) is an adequate antibiotic choice to use during transport to definitive medical care. In addition, acyclovir 10 mg/kg IV should be given every 8 hours for empirical treatment of herpes simplex virus meningitis or encephalitis.

Encephalitis

Encephalitis classically involves the brain. The most common causes of encephalitis are viruses, particularly herpes simplex virus and the arboviruses (which are spread by arthropods, including mosquitoes and ticks). The most publicized cause of encephalitis in recent years has been West Nile virus, which is transmitted by mosquitoes and is considered to be a subgroup of the larger worldwide group of Japanese encephalitis viruses. Most of these viruses appear to be transmitted to humans from animals such as birds and pigs through mosquitoes and have

been found on every continent except Antarctica. Children and immunocompromised adults are at high risk, as are individuals in regions endemic for the viruses with large numbers of mosquitoes. Rates of infection are greatly increased during the seasons with the most mosquito activity. Tick-borne encephalitis is particularly common in Russia and areas of Europe, and is especially problematic in heavily wooded areas with large numbers of ticks.[23]

It may be difficult to differentiate encephalitis from meningitis because many of the presenting signs and symptoms are similar. Symptoms may begin as soon as 2 to 3 days after exposure, although typically they begin in 5 to 15 days, usually with a fever and flulike symptoms. Patients may have headache, fever, and seizures, and almost all have a change in level of consciousness. Paralysis and Parkinson-type movements of extremities are not uncommon in the arthropod encephalitis presentations.[29] Personality changes that resemble psychiatric disturbance may precede other symptoms by several days, which can greatly confuse the clinical picture.[14]

Encephalitis is more difficult to diagnose than meningitis because the causative viruses are not always present in the CSF on lumbar puncture. Viral studies completed on serum may be diagnostic but certainly will not be completed in the field. Instead, diagnosis in the backcountry must be made clinically. Although herpes simplex encephalitis may be treated with antiviral medications such as acyclovir, there is no current pharmacologic treatment for infections caused by arboviruses. Treatment is thus supportive, and may include ventilator support, IV fluids, and prophylaxis against complications such as seizures. However, if transport is necessary, empirical treatment with antibiotics and antivirals, as stated previously, should be started for any patient suspected of meningitis or encephalitis.

Protection against insect bite is the best strategy for prevention of disease. Judicious use of DEET-containing insect repellents as well as avoidance of peak mosquito activity times can greatly limit transmission from infected mosquitoes and other insects.[30]

CEREBROVASCULAR DISEASE

Strokes secondary to cerebrovascular disease are the third leading cause of death in the United States. Patients with stroke symptoms must be recognized immediately and transported to a medical facility quickly if permanent disability or death is to be prevented. A stroke can be defined as any vascular injury that prevents sufficient blood flow to specific areas of the brain, leading to focal neurologic injury. Strokes may be further defined as ischemic or hemorrhagic. Ischemic strokes comprise 80% to 85% of all strokes and occur when an embolus or a focal plaque occludes a cerebral vessel. Hemorrhagic strokes comprise 15% to 20% of all strokes and occur when a cerebral blood vessel ruptures, leading to bleeding within the brain itself (intracerebral hemorrhage) or into the subarachnoid space around the brain (SAH). Transient ischemic attack (TIA) is a term used to describe transient symptoms that may precede an ischemic stroke. Symptoms may last less than 15 minutes but are important to recognize to prevent additional disease.

Patients suffering from ischemic stroke typically have sudden onset of one or more of the following symptoms, depending on location of the lesion: unilateral weakness or numbness, difficulty speaking, vision changes, difficulty walking, and occasionally headache. If ischemia is secondary to focal atherosclerosis, the patient may have similar or identical symptoms consistent with a previous TIA. Differential diagnosis of ischemic stroke includes intracranial hemorrhage, hypoglycemia, tumor, Todd's paralysis (after seizure activity), cerebral venous thrombosis, and infection. Ischemia can occur either in the anterior circulation (carotid arteries), which provide perfusion to 80% of the cerebrovascular system or in the posterior circulation (vertebral arteries). Anterior strokes lead to motor and speech disability, whereas posterior strokes typically cause problems with balance, vertigo, and vision.

Hemorrhagic strokes present with symptoms similar to those of ischemic strokes; however, associated mental status changes, particularly decreased level of consciousness and subsequent respiratory depression, can be life-threatening. Patients with SAH may have sudden onset of a severe headache with vomiting, syncope, hemiparesis, and neck stiffness. Differential diagnosis of hemorrhagic stroke includes trauma, accelerated hypertension, vasculitis, tumor, infection, drug overdose, metabolic derangement, and cardiac disease.

All patients with suspected cerebrovascular disease should be treated similarly. Airway, breathing, and circulation should be assessed quickly. Although the airway is not commonly compromised in ischemic stroke, any patient with suspected intracranial hemorrhage should have close airway observation. An electrocardiogram or cardiac monitor can help assess for arrhythmias that could have caused an embolus leading to ischemic stroke, or may indicate cardiac problems causing mental status changes. All patients should have a thorough neurologic examination, searching for asymmetry of strength or sensation and changes in pupil size or visual fields. Gait testing can show ataxia or help define lower extremity weakness. Mental status examination not only provides information about level of consciousness, memory loss, and confusion but also identifies speech and comprehension deficiencies. Emergency transport to a hospital is critical for definitive airway protection, neuroimaging, and definitive treatment. Severe ischemic strokes with associated motor and language deficits may be treated with intravenous tissue plasminogen activator (t-PA). Current indications for this therapy mandate treatment within 3 hours of symptom onset as well as a brain CT scan showing no evidence of intracranial hemorrhage. Other contraindications to treatment include severe hypertension, recent surgery or head trauma, use of oral anticoagulants, recent gastrointestinal bleeding, or improving stroke symptoms.

Treatment with other intravenous anticoagulants, such as heparin, has not been shown to be helpful except in the case of thromboembolism secondary to atrial fibrillation. Aggressive lowering of blood pressure may lead to worsening of cerebrovascular ischemia and should be done only in an intensive care setting under the advice of a neurologist.[4]

Patients with presumed intracranial hemorrhage may require close airway management and do require transport to a neurosurgical center. Surgical evacuation of an intracranial hemorrhage is determined by the severity and location of the bleed. Hyperventilation of certain patients and elevation of the head of the bed may be helpful in decreasing intracranial pressure during transport. Use of mannitol is sometimes recommended in cases of presumed brain herniation, but should be done carefully to avoid hypotension, and should be guided by the preference of the treating neurosurgeon. SAH is typically treated

with surgical clipping of the aneurysm and also requires neurosurgical consultation.[11]

If evacuation is to be delayed, patients should be monitored closely for any changes in mental status or progression of peripheral symptoms. All patients should be encouraged to rest, and they may benefit from sleeping with the head elevated at least 30 degrees. The patient's usual blood pressure medications may be administered if the patient is alert and oriented, and a daily aspirin may be beneficial in cases of embolic stroke if the patient is not complaining of headache and has no mental status changes. Any activity that could stress the patient should be prevented, such as carrying a pack or even straining to have a bowel movement. If these patients must evacuate under their own power, they should avoid carrying weight and should be allowed to rest frequently. Any worsening in signs or symptoms requires a formal extrication.

MOVEMENT DISORDERS

Movement disorders include hypokinetic disorders (Parkinson's disease), hyperkinetic disorders (prolonged movements of one muscle group or the entire body), tremors, and myoclonus (short, sudden jerks of one muscle group or the entire body). Although these disorders are important medical syndromes, they do not usually have severe, life-threatening presentations in the wilderness. However, they all must be differentiated from seizure activity so that appropriate treatment may occur.

Movement disorders often become obvious on physical examination, and patients are often able to describe their personal symptoms. An exacerbation could occur in the wilderness but should not be dangerous to the victim. However, the medications used to treat these disorders should be acknowledged because they often have a great number of side effects and may be part of the problem.

Parkinson's Disease

Parkinson's disease has a prevalence of 160 per 100,000 people overall and increases with age (500 per 100,000 at age 70 years). It may be diagnosed when the patient is in the early stages of the disease and thus remains fairly active. It is a neurodegenerative disorder thought to be secondary to genetic predisposition and probably yet-unspecified environmental factors. The disease is characterized by deficiency of effective dopamine in the substantia nigra in the basal ganglia, leading to changes in movements. The disease is more common in men than women, and greater than 85% of cases occur in persons older than 50 years of age.

Parkinson's disease is identified by the presence of resting tremor, bradykinesia (slowed movements), rigidity, and loss of postural reflexes. Loss of postural reflexes often causes poor balance and frequent falls. Commonly, an emergency presentation of the patient with Parkinson's disease will be after a fall that has injured the patient. Patients with Parkinson's disease may also suffer from a form of dementia and associated emotional depression. Differential diagnosis includes normal aging, essential tremor, drug-induced parkinsonism, severe depression, and other, less common movement disorders (e.g., Huntington's disease, chorea, myoclonus, Tourette's syndrome, and progressive supranuclear palsy). Because Parkinson's disease is progressive and chronic, emergency presentations do not occur often. However, side effects from antiparkinsonian medications might cause a patient to seek help in the wilderness. Many patients with Parkinson's disease take dopamine replacement or dopamine agonists, such as levodopa, which help replace inadequate dopamine stores and allow for improved movement, lessened tremor, and diminished rigidity. Common side effects of these medications include nausea, hypotension (especially postural), involuntary movements, and mental status changes.[24] Because of the increased rate of falls in this population, both from the disease itself and the medications, any mental status change should cause the care provider to consider the possibility of a subdural hematoma or other intracranial hemorrhage secondary to a recent fall. Falls can induce significant neurosurgical and orthopedic injury and should be treated as multiple organ system trauma.[25]

Another side effect unique to levodopa is secondary to its pharmacokinetic properties of erratic absorption and short duration of action. Although a patient is usually aware of the effects of levodopa wearing off, some patients experience sudden immobility after a period of mobility. The cycle of mobility and immobility is known as *on-off phenomenon* and can lead to effective paralysis of motion. Patients with Parkinson's disease are usually well educated about their personal medications and should be asked if their symptoms are familiar or recurrent.

Resting and Intention Tremors

Tremors should be differentiated between resting and intention tremors. Resting tremors are present when extremities are at rest and are caused by Parkinson's disease as well as by certain neuroleptic medications. Intention tremors may be hereditary, caused by cerebellar disease, or may have no known cause but may worsen when the patient makes a specific movement such as drinking from a glass or reaching for something. Tremors may worsen with fatigue, stress, or missed doses of medications. If a patient's tremor acutely worsens in the wilderness, the patient should be encouraged to hydrate and take any missed medication doses, as well as to rest. Removal from the backcountry is not required unless the patient becomes so disabled from the tremor that inability to walk or care for oneself occurs.

DEMENTIA

Dementia versus Delirium

Dementia and delirium both involve disruption in global cognition and may include changes in mental status, memory, and loss of social skills. Differentiation between delirium and dementia is critical because delirium has an organic cause that often may be treated, particularly in the acute phase. Delirium presents abruptly and may involve changes in level of consciousness over the course of the disease. Dementia presents over a period of time, and level of consciousness remains fairly stable.

Dementia

Dementia is a single term used to describe a clinical syndrome of progressive deterioration of cognitive function. Dementia may be a primary disorder, such as Alzheimer's disease, or secondary to a multitude of disease states, including Parkinson's disease, HIV infection, or a nutritional deficit such as thiamine deficiency. The approach to all patients with dementia in the wilderness is the same. Although most severely affected patients with Alzheimer's disease do not venture into the remote wilderness, it is not unusual to find elderly patients in national and state parks and other, more easily accessible wilderness areas.

It is thought that approximately that 6% of all individuals have at least mild dementia, and this proportion rises to 30% in persons 85 years of age and older.[7] Primary dementia is caused by either Alzheimer's or Pick's disease. Alzheimer's disease, the best understood dementia, is caused by neuronal death and subsequent atrophy. The amount of resulting atrophy is much greater than the atrophy found in the normal aged population. On brain biopsy or autopsy, histologic findings include specific changes such as senile plaques and neurofibrillary tangles, which are pathognomonic for Alzheimer's disease. Risk factors include advanced age, female sex, family history, lower education level, and history of head trauma. Dementia can be secondary to other causes, such as multiple infarcts, medication, tumor, infection, nutritional deficits, and metabolic derangements.

Diagnosis of dementia is suspected when individuals begin to show deficits in memory, completion of activities of daily living, and use of language. Patients may have difficulty initially with advanced activities of daily living such as use of maps, and gradually have increased difficulty with grooming and eating. Diagnosis of incurable dementia is completed by effectively ruling out all reversible causes. All medications should be reviewed and thorough physical and mental status examinations should be completed.[28]

Currently, treatment for dementia consists mostly of reversing treatable causes, such as infection or metabolic derangement. Although there is no single agent that truly reverses signs and symptoms of Alzheimer's disease, several treatments appear to slow advancement of the disease. Cholinesterase inhibitors, such as donepezil and rivastigmine, have become the standard of care for all patients with Alzheimer's disease, and should be started with early symptoms. Although the drugs have not yet been proven to improve symptoms, there is adequate evidence to suggest that they are effective in allowing individuals to perform more activities of daily living and to have fewer behavior changes, thus allowing them to live more independently for a longer time. Memantine, an antagonist to *N*-methyl-D-aspartate, has recently been approved for moderate or severe dementia, and has been shown to provide cognitive improvement and delay loss of daily activities in some patients compared with placebo. In large doses, vitamin E (α-tocopherol) has been shown to be an adjunctive treatment and is thought to act as an antioxidant. Anti-inflammatory agents and hormone replacement therapy have been proven to have no effect. Nonpharmacologic interventions such as exercise and music, as well as frequent interactions with loved ones, are also effective treatments.[2]

Dementia is not a diagnosis that will be made in the wilderness. If a person has dementia or suspected dementia and has abrupt worsening in mental status or physical condition, immediate transport should be made to a medical facility. Urgent evaluation for intracranial hemorrhage, cerebrovascular accident, or metabolic derangement is required. Hypoglycemia is one cause of mental status change that may be easily treated by providing glucose to the patient, usually through oral administration by placing sugar or honey on the mucous membranes for rapid absorption.

NEUROMUSCULAR DISORDERS

Although neuromuscular disorders are often chronic and not life-threatening, emergencies can arise if the airway and lungs are compromised. An uncommon but not rare example is Guillain-Barré syndrome (GBS).

Acute Inflammatory Polyneuropathy (Guillain-Barré Syndrome)

The vast majority of patients with GBS have progressive symmetrical weakness over several days or weeks. Early in the illness, reflexes may be reduced, and they often disappear as the disease progresses. Although sensory findings are often minimal throughout the illness, paresthesias and numbness may occur. In 90% of patients, symptoms begin in the lower extremities and ascend sequentially to the trunk area, upper extremities, and cranial nerves, reaching a peak in 2 weeks or less. In 10% of cases, symptoms may start in the face and descend. Even in the ascending variety, there may be early facial weakness. Cardiac conduction disorders, respiratory failure, and urinary retention are not uncommon and must be constantly monitored. Cardiac or respiratory failure causes death in 3% to 5% of cases.

Any patient with weakness should be removed from the backcountry immediately for further testing. CSF analysis and nerve conduction studies must be completed for this disease to be definitively diagnosed. The most important diagnosis to rule out is an acute spinal cord disease, such as transverse myelitis, with associated progressive extremity weakness, paresthesias, and rectal sphincter paralysis. Spinal cord compression, such as that caused by tumor, abscess, or hematoma, should be ruled out in every patient with GBS.

Treatment of GBS includes respiratory assistance at the first sign of respiratory compromise, as well as detection and treatment of cardiac arrhythmias and careful monitoring for deep venous thrombosis and pneumonia in ventilated patients. Patients who receive plasma exchange or intravenous immunoglobulin in the early stages of GBS have a decreased need for mechanical ventilation later on and tend to have shorter hospitalizations. GBS is a potentially life-threatening illness. When it is seriously considered in the wilderness, the patient should be transferred to a facility with an intensive care unit without delay.[6]

Tick Paralysis

Tick paralysis is a rare disorder caused by a toxin secreted by the salivary glands of a gravid tick during a blood feeding, and it may present very similarly to GBS. Forty-three species of ticks have been found to cause tick paralysis, with *Dermacentor* species causing most cases in North America and *Ixodes* species implicated most often in Australia. The causative toxin acts at the motor endplate to prevent potential transmission and requires that the tick be attached to the skin for several days. The tick is usually found in the hair; patients are mostly children. Symptoms include ascending motor weakness (beginning in the lower extremities), areflexia, and absent or minimal sensory changes. The symptoms clear rapidly (within hours) when the tick is removed. All patients with the suspected diagnosis of GBS should have a careful examination for ticks, with particular attention to regions with hair such as the scalp and pubic areas.[31] Ticks should be removed immediately if found, but transport to an appropriate medical facility is still warranted.

Myasthenia Gravis

Myasthenia gravis is an autoimmune disorder that results in a defect in neuromuscular transmission secondary to antibody-

mediated attack on the nicotinic acetylcholine receptors at the neuromuscular junction. The result is fluctuating weakness from antibody saturation of acetylcholine receptors on muscle. Ocular muscles are affected first in 40% of patients and ultimately involved in approximately 85%.[19] Myasthenia gravis should be a primary consideration in patients with double vision (diplopia) and drooping eyelids (ptosis).[20] Patients with generalized myasthenia may have weakness of limbs and respiratory muscles; therefore, if the diagnosis is strongly suspected, the first consideration is to ensure an adequate airway. The disease is characterized by fluctuation of weakness over a period of hours or days. Diagnosis is made by the finding of serum antibodies to acetylcholine receptors in 85% to 90% of cases.[21]

Botulism

Botulism is a poisoning caused by a polypeptide toxin of *Clostridium botulinum*. In contrast to myasthenia gravis, botulinum toxin affects release of acetylcholine and results in weakness of striated and smooth muscles, as well as autonomic dysfunction. In adults, the toxin is usually ingested when eating inadequately sterilized canned or prepared food. The toxin is easily destroyed by heat, and most cases result from eating uncooked food. A temperature of 115°C (240°F) must be achieved by regular cooking or pressure-cooking to eliminate botulinum toxin and spores. Other causes of botulism include injection of improperly and illegally manufactured forms of Botox and the use of contaminated heroin, neither of which should occur in the wilderness. (The commercial form of Botox used by medical professionals is a purified form of botulinum protein.) Infant botulism occurs in the first year of life, frequently between 2 and 4 months of age. Approximately 30% of cases follow ingestion of honey contaminated with botulism spores, which infants are unable to digest safely.[17]

Initial symptoms are usually nausea, vomiting, and diarrhea, followed by visual complaints. Ptosis and paralysis of extraocular muscles commonly occur, and pupils may be fixed and dilated. Ocular symptoms are followed by weakness of jaw muscles, difficulty speaking (dysarthria), and difficulty swallowing (dysphagia). Progressive paralysis can ensue. Smooth muscles of the intestines and bladder may be affected, and in severe cases cardiac and respiratory failure may develop.[17] Convulsions and coma may be preterminal. Treatment with antitoxin should begin as soon as the disease is suspected, which requires rapid transport to a medical facility.

NERVE DISORDERS

Multiple Sclerosis

Multiple sclerosis (MS) is a chronic disease that begins most commonly in young adults and is characterized pathologically by CNS white matter inflammation, demyelination, and glial scarring (sclerosis). MS has long been defined as a white matter disorder that is disseminated in "time and space" because lesions are scattered throughout the nervous system and exacerbations may occur weekly, monthly, or yearly. The etiology of MS is unknown. Genetic susceptibility, autoimmune mechanisms, and viral infections have been suggested as possible origins.

Patients with MS have variable disease courses. Although some have gradual, progressive worsening and are unable to remain as active as previously, others may go long periods with minimal symptoms, allowing them to continue with many outdoor activities. Common signs and symptoms of MS are weakness, spasticity, fatigue, bladder dysfunction, visual disturbances, and cognitive abnormalities, including depression. An acute exacerbation such as limb paralysis or loss of vision may result in immediate need for medical attention. Acute vertigo, urinary retention, and profound clumsiness, although less common, are problematic. Fatigue is a common complaint and may become so pronounced with exertion that recreational plans must be abandoned. Patients with MS are encouraged to exercise, but not to the point of severe fatigue.

Heat and other environmental stressors can cause acute exacerbations of MS. For many years, the "hot bath" test was used to diagnose MS. Heat produces a temporary worsening of symptoms and does not cause more actual tissue damage (demyelination or damage to the nerves). Symptoms are usually rapidly reversed when the source of increased temperature is removed. Keeping patients with MS well hydrated and avoiding heat-related illnesses are critical to maintaining health in the wilderness. Because fever can be a source of heat leading to worsening symptoms, any suspected infection should be treated aggressively. Some people with MS notice that symptoms, particularly spasticity, worsen in cold weather, but for the most part, patients with MS tolerate cold better than heat.[18]

Exacerbations may respond to steroid therapy, but this can be given only if there is no risk of infection. Transport to a medical facility should be instituted if signs or symptoms are a concern.

Bell's Palsy

Bell's palsy is the most common form of facial paralysis, involving paralysis of the facial muscles innervated by the seventh (facial) nerve (Fig. 28-1). Bell's palsy is rapidly progressive, with maximum weakness present within 24 to 48 hours. The patient may not be aware of early symptoms until they are noticed by others. The palsy is painless, although there may be mild pain and tenderness behind the ear when symptoms are first noted. This discomfort may precede the weakness by a day or so. Taste may be reduced or lost on the anterior two thirds of the tongue on the same side as the facial weakness. It is important to differentiate Bell's palsy from a stroke or tumor. Bell's palsy should cause weakness on one side of the face, including the forehead. If there is no weakness of the forehead, transport to a CT scanner is necessary to rule out more ominous causes of the facial deficit.

The etiology in most cases of Bell's palsy is idiopathic. The prognosis is excellent, with 80% of patients having complete recovery and 90% of patients showing some improvement within a few weeks or months. Herpes zoster (Ramsay-Hunt syndrome) should be ruled out by looking for painful herpetic lesions (vesicles) in the ear and mouth.

Treatment with acyclovir (800 mg PO five times daily for 7 days) and a corticosteroid (prednisone 40 to 60 mg/day PO for 5 to 10 days) is thought to improve symptoms if medications are started within 72 hours of symptoms. Other immediate management of Bell's palsy primarily involves eye protection. The patient is unable to blink and becomes a prime candidate for corneal abrasion or infection if the eye is not protected. If Bell's palsy develops in the wilderness, the person should wear an eye patch, particularly if there is exposure to wind or other potential eye irritants. Lacri-Lube or other eye lubricants should be used to provide moisture when the eye is not patched.

Figure 28-1. Bell's palsy: paralysis of the facial nerve. (Photograph courtesy of Dr. Stephen Bretz, University of California, San Francisco.)

Herpes Zoster

Herpes zoster is a common viral infection of the nervous system that can cause severe pain, and should be suspected early to make a therapeutic intervention. The cause is thought to be reactivation of latent varicella infection in the sensory nerve ganglia after a primary infection with chickenpox or immunization against varicella. The disease is more common in the elderly, persons with malignancies, and persons on immunosuppressive therapy but may occur in healthy adults, particularly during times of fatigue or stress.

The first symptoms may be malaise and fever with paresthesias, itching, or pain in the affected nerve root distribution (see Figure 20-3). Symptoms should be unilateral only, with sensory, not motor symptoms. There may initially be some redness of the involved painful skin, with development of clear vesicles 3 to 4 days after onset of pain. Vesicles crust after a week or so, and scabs are noted. The affected area can be severely painful and symptoms may last weeks to months after the rash has disappeared. All areas of the body are susceptible to zoster infection, but the chest wall is the most common site.

If herpes zoster affects the face, it usually affects the cranial nerves. Involvement of the eye (ophthalmic zoster) requires urgent consultation with an ophthalmologist. Involvement of the facial nerve (Ramsay Hunt syndrome) can lead to Bell's palsy and painful lesions in the ear.

If the diagnosis is in question, viral cultures can be obtained from vesicular fluid. Early in the course of the illness, radicular pain may suggest nerve root compression, especially if there is motor weakness. Sharp pain in the chest wall may be mistaken for pleurisy or cholecystitis. Herpetic abdominal wall pain can be mistaken for appendicitis.

There is usually no problem with making the diagnosis as long as the vesicles are apparent. The vesicles may not be obvious, however, and in rare cases may be absent. It is wise to check everyone with acute localized pain in a nerve root distribution for zoster vesicles.

Effective treatment for herpes zoster depends on prompt diagnosis. Acyclovir (800 mg PO five times daily for 7 days) should be given as soon as possible after appearance of the rash to be effective, and it has been found to prevent postherpetic pain only when started within 72 hours of symptoms. Narcotic pain medication may be required. Postherpetic pain is a difficult problem. It may last for months to years and may be resistant to most therapies. Zoster infection can recur. Children contract chickenpox from exposure to adults with shingles, but adults are subject to herpes zoster only if they had chickenpox earlier in life or were immunized against varicella.[9]

Trigeminal Neuralgia (Tic Douloureux)

Trigeminal neuralgia is a disorder of the sensory division of the trigeminal nerve whose cause remains unknown. It results in sharp, stabbing pain in one or more division of the nerve. Pain is episodic; the patient is symptom-free between attacks. It may occur in spells lasting a few seconds to 10 to 15 minutes and may recur several times during the day. It can be precipitated by talking, chewing, eating cold food, or having cold air contact a trigger area. It usually affects middle-aged or older patients. The younger the patient, the more likely there is to be concomitant MS, acoustic neuroma, basilar aneurysm, or other brainstem lesion. In the wilderness, trigeminal neuralgia should be differentiated from migraine headache, temporomandibular joint pain, and sinusitis.

The neurologic examination in these patients is negative, so diagnosis must be made by history. Treatment is often successful with neuroleptic medications such as carbamazepine and gabapentin, but this will not usually be available in the wilderness. Although narcotic pain medications may be requested, the symptoms, because they generally are of short duration, may not be helped by acute administration of analgesics. Instead, care must be taken to isolate and prevent the stimulus that causes the symptoms.

Peripheral Neuropathy

Peripheral neuropathy includes disorders of peripheral motor, sensory, and autonomic nerves. From a practical standpoint it is easier to separate the neuropathies into acute (traumatic, GBS, tick paralysis), subacute (occur over weeks from toxin, drug, or heavy metal poisoning), and chronic (diabetes). Patients with polyneuropathy (in multiple extremities) are likely to have diminished or absent reflexes, motor weakness in the distal extremities, and sensory changes. Sensory changes are often in a stocking-and-glove distribution, and include impaired sensation to pain, temperature, and light touch. The sensory changes are a result of damage to the myelin sheath surrounding the nerves or damage to the neuronal axons themselves. The most common complaint of peripheral neuropathy is burning paresthesias in the feet, which are often a result of diabetes or alcoholism.

TABLE 28-2. Neurologic Presentations in Wilderness Situations

SITUATION	NEUROLOGIC PRESENTATIONS
High altitude	Headache—acute mountain sickness, carbon monoxide poisoning Altered mental status—high-altitude cerebral edema (HACE), high-altitude encephalopathy, hypoxia Seizure—HACE Vision loss—retinopathy, keratitis
Cold environment	Headache—dehydration Motor and sensory changes—frostbite Altered mental status—hypothermia Vision loss—corneal freezing
Hot environment	Headache—dehydration, heat exhaustion Altered mental status—heat stroke Seizure—heat stroke, (hypotonic) hyponatremia from water ingestion
Diving	Headache—barosinusitis Motor and sensory changes—hyperventilation, decompression sickness, dysbaric spinal cord injury Altered mental status—arterial gas embolism, nitrogen narcosis Seizure—CNS oxygen toxicity, arterial gas embolism
Submersion	Altered mental status—hypothermia, hypoxia
Snakebite envenomation	Motor and sensory changes—neuromuscular weakness/paralysis, fasciculations, and paresthesias from neurotoxic snakes (Mojave rattlesnakes and elapids), ascending paralysis and loss of reflexes (sea snakes) Altered mental status—drowsiness and hallucinations (elapids)
Insect envenomation	Headache—rickettsial infections Altered mental status—arthropod-borne encephalitis, Lyme meningitis/encephalitis Motor and sensory changes—tick paralysis, facial nerve palsy and radiculoneuritis from Lyme disease, progressive encephalomyelitis years after Lyme infection Seizures—*Latrodectus* bites in elders and children
Toxic plant ingestions	Motor and sensory changes—hypotonia, decreased reflexes, paralysis (nicotinics such as *Solanaceae,* horse chestnut, milkweed) Altered mental status—anticholinergic syndrome (alkaloids such as jimson weed, mandrake, henbane, deadly nightshade), confusion (nicotinics), hallucinations (psychoactive agents such as psilocybin, mescaline, cannabis) Seizures—water hemlock
Mushroom toxicity	Motor and sensory changes—ataxia (*Amanita muscaria*, *Amanita pantherina* of isoxazole derivation), delayed paresthesias (*Cortinarius*) Altered mental status—intoxication (isoxazole derivatives), hallucinations (*Psilocybe*), delirium (*Gyromitra*) Seizures—*Gyromitra*
Scorpion envenomation	Motor and sensory changes—cranial nerve and skeletal muscle dysfunction (*Centruroides*) Seizures—*Leiurus, Androctonus, Mesobuthus*
Smoke inhalation	Headache—carbon monoxide poisoning Altered mental status—carbon monoxide poisoning Seizure—carbon monoxide poisoning
Lightning strike	Headache—occurs weeks to months after strike Motor and sensory changes—difficulty with fine motor movements after strike Altered mental status—intracranial hemorrhage or edema, amnesia, confusion, anoxia Seizure—delayed seizures, electroencephalographic abnormalities Vision loss—retinal detachment, vitreous hemorrhage, hyphema, cataracts
Animal bite	Altered mental status—rabies

Treatment is to reverse the cause of the neuropathy if possible. The ultimate goal of the patient, however, is relief from painful paresthesias. Tricyclic antidepressants and a number of neuroleptic medications, such as gabapentin, carbamazepine, and lamotrigine, may be useful, depending on the disorder. Acutely, narcotic pain medication may be required.

Carpal Tunnel Syndrome

Carpal tunnel syndrome is an entrapment neuropathy of the median nerve as it passes through the canal formed by the carpal bones and ligament. Painful paresthesias in the thumb and first three fingers that awaken the patient at night or in the early morning are the most common complaint. Often the symptoms are vaguely annoying and the patient thinks that all the fingers are involved. Carpal tunnel syndrome is frequently seen in patients with hypothyroidism and diabetes mellitus and is more common in women. Symptoms can be produced with wrist flexion (Phalen's sign). In some cases, tapping over the carpal tunnel may produce paresthesias (Tinel's sign). Activities such as driving and holding a newspaper with the wrist in flexion may elicit symptoms. Wilderness activities such as climbing may cause an acute exacerbation.

A wrist splint worn at night that holds the wrist in a neutral position may reduce symptoms by preventing extreme flexion and extension. Anti-inflammatory medications should be administered. A steroid injection using 40 mg of methylprednisolone and 2 mL of bupivacaine (Marcaine) 0.25% through a 25-gauge needle below the carpal ligament sometimes gives

relief. Care must be taken to avoid the median nerve, so the injection site should be on the ulnar side of the midline of the palmar surface (or on the ulnar side of the palmaris longus, if present). Full relief may not occur for up to 2 weeks, so this therapy should be reserved for patients on longer backcountry excursions. Surgery to release the nerve by removing the ligament overlying the carpal tunnel is the only treatment option if there is evidence of denervation or hand muscle atrophy.

WHEN TO TRANSPORT

Rapid transportation to definitive care is required when a patient has neurologic signs or symptoms that could lead to loss of the airway or ventilatory effort. Such patients include those with suspected intracranial hemorrhage (from stroke, tumor, or trauma), spinal cord injury, or decreased level of consciousness. In addition, new-onset seizures, persistent seizures, suspected meningitis or encephalitis, and limb weakness mandate prompt evacuation. If an individual with a chronic neurologic condition appears to be worsening, this may require evacuation (Table 28-2).

The references for this chapter can be found on the accompanying DVD-ROM.

Chronic Diseases and Wilderness Activities

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Colin K. Grissom and Thomas G. DeLoughery

The focus of this chapter is prevention and treatment of complications due to chronic medical conditions during wilderness activities. For the purposes of this chapter, wilderness activities are defined as any excursion into an outdoor environment where usual medical services are not available, and include outings ranging from a day to several months. Wilderness activities may occur close to home, in the country of residence, in foreign developed countries, or in foreign developing countries. Issues relating to chronic medical conditions during wilderness activities are important to primary care and specialty physicians because patients often seek advice on whether a certain wilderness trip or activity is appropriate, given their medical condition, and how they should manage their disease while in the wilderness.

Medical problems may occur during wilderness activities due to an acute injury, acquired infectious illness, or environmentally caused illness (heat, cold, or high altitude illness). A preexisting medical condition may also cause complications during a trip or activity, or may predispose the patient to environmentally caused illness. The few studies that report the epidemiology of medical problems in the wilderness show that most are due to traumatic injuries, and most deaths are due to falls or drowning.[38,52,95] Cardiac disease is the most common medical illness that causes death in the wilderness. Cardiac disease, asthma, and diabetes are also reported as causes of medical illness during wilderness activities. Medical illness due to environmental exposure, such as heat, cold, or altitude illness, depends on the environment.[38,75]

The first issue to consider in evaluating a patient with a chronic medical condition who is planning a wilderness trip or activity is how the environment will affect the patient and his or her medical condition. In a wilderness environment, patients with chronic medical conditions will potentially be exposed to heat, cold, high altitude, and a greater level of exercise. Wilderness activities occur in remote areas where medical care is impossible to obtain. These factors require evaluation in relation to the patient's chronic medical condition. First, the physician and patient need to address whether the planned wilderness activity is appropriate for the patient. Second, the physician should provide advice to the patient on how to manage his or her medical condition in the remote wilderness environment.

Deciding on whether a potential wilderness activity is appropriate for a patient is a shared responsibility between the physician and patient. "Wilderness" implies that medical care will be remote, so if a major medical problem arises, the consequence may be severe or even include risk of death. Risk is an inherent part of any wilderness activity, so persons who venture into the wilderness should make conscious decisions about acceptable levels of risk and how they can exercise good judgment to reduce those risks. The same process is used with regard to a chronic medical condition: acknowledging risk, deciding whether it is acceptable, and changing behavior to minimize risk. Although it is important for the physician to help the patient understand issues and to make medically responsible recommendations, he or she should recognize patient autonomy and leave most decisions to the patient.

relief. Care must be taken to avoid the median nerve, so the injection site should be on the ulnar side of the midline of the palmar surface (or on the ulnar side of the palmaris longus, if present). Full relief may not occur for up to 2 weeks, so this therapy should be reserved for patients on longer backcountry excursions. Surgery to release the nerve by removing the ligament overlying the carpal tunnel is the only treatment option if there is evidence of denervation or hand muscle atrophy.

WHEN TO TRANSPORT

Rapid transportation to definitive care is required when a patient has neurologic signs or symptoms that could lead to loss of the airway or ventilatory effort. Such patients include those with suspected intracranial hemorrhage (from stroke, tumor, or trauma), spinal cord injury, or decreased level of consciousness. In addition, new-onset seizures, persistent seizures, suspected meningitis or encephalitis, and limb weakness mandate prompt evacuation. If an individual with a chronic neurologic condition appears to be worsening, this may require evacuation (Table 28-2).

The references for this chapter can be found on the accompanying DVD-ROM.

Chronic Diseases and Wilderness Activities

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Colin K. Grissom and Thomas G. DeLoughery

The focus of this chapter is prevention and treatment of complications due to chronic medical conditions during wilderness activities. For the purposes of this chapter, wilderness activities are defined as any excursion into an outdoor environment where usual medical services are not available, and include outings ranging from a day to several months. Wilderness activities may occur close to home, in the country of residence, in foreign developed countries, or in foreign developing countries. Issues relating to chronic medical conditions during wilderness activities are important to primary care and specialty physicians because patients often seek advice on whether a certain wilderness trip or activity is appropriate, given their medical condition, and how they should manage their disease while in the wilderness.

Medical problems may occur during wilderness activities due to an acute injury, acquired infectious illness, or environmentally caused illness (heat, cold, or high altitude illness). A preexisting medical condition may also cause complications during a trip or activity, or may predispose the patient to environmentally caused illness. The few studies that report the epidemiology of medical problems in the wilderness show that most are due to traumatic injuries, and most deaths are due to falls or drowning.[38,52,95] Cardiac disease is the most common medical illness that causes death in the wilderness. Cardiac disease, asthma, and diabetes are also reported as causes of medical illness during wilderness activities. Medical illness due to environmental exposure, such as heat, cold, or altitude illness, depends on the environment.[38,75]

The first issue to consider in evaluating a patient with a chronic medical condition who is planning a wilderness trip or activity is how the environment will affect the patient and his or her medical condition. In a wilderness environment, patients with chronic medical conditions will potentially be exposed to heat, cold, high altitude, and a greater level of exercise. Wilderness activities occur in remote areas where medical care is impossible to obtain. These factors require evaluation in relation to the patient's chronic medical condition. First, the physician and patient need to address whether the planned wilderness activity is appropriate for the patient. Second, the physician should provide advice to the patient on how to manage his or her medical condition in the remote wilderness environment.

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CONSIDERATIONS FOR WILDERNESS TRAVEL

Increased Physical Activity

Many commercial adventure trips carry a difficulty rating in terms of level of exertion, remoteness, and exposure to high altitude. Ratings generally include easy, moderate, and strenuous categories. This can be useful to the physician evaluating the patient before the trip, especially if the physician is not familiar with the environment or the activity that will be pursued. Some companies that offer adventure trips require potential clients to complete a medical history questionnaire and may also have a consultant physician review the questionnaire and provide advice to selected clients.[59,60] Erb[39] has developed a scale of trip difficulty and correlated that with objective parameters of exercise capacity required to complete that type of activity (see Table 89-2, Chapter 89). Those requiring the highest exercise capacity are "Extreme Performance Ventures." These are the most physically demanding and include mountaineering at high altitude and alpine climbing. Above-average exercise capacity is required to complete such activities. The next most physically demanding wilderness trips are "High Performance Ventures," such as high-altitude trekking or hunting, or jungle trekking, and require an average exercise capacity. The third level of wilderness trips is "Recreational Activities." These include hiking in a variety of environments that require average, or just below average, exercise capacity. The last category of wilderness trips is "Therapeutic Activities," which may be appropriate for persons with chronic cardiovascular or pulmonary disease that limit activity. Even though Erb defines categories of wilderness trips and assigns objective exercise capacity parameters to them, formal cardiac stress testing or cardiopulmonary exercise testing is not required in most cases to determine if a patient has adequate exercise capacity to complete a planned wilderness trip. The most important predictor is successful completion by the patient of similar activities in the past. Still, formal cardiopulmonary exercise testing may be useful in patients with chronic medical conditions to define work capacity objectively in order to select the appropriate level of a wilderness trip. This helps to ensure a more enjoyable and safe experience for the patient.

Available Medical Care and Reliance on Other Trip Members

For the patient with a chronic medical condition, preparation for a wilderness trip or activity includes informing others involved in the activity or trip, or the leader in the case of a commercial trip, of medical issues that might affect the patient's ability to complete the planned activity or itinerary. This is important because chronic medical conditions may influence not only the patient but other members of the party. Awareness is important to help the patient avoid situations where problems may worsen if not recognized early. Other members of the group, or the leaders on a commercial trip, need to monitor the patient's medical problem and be prepared to alter the plans for the whole group and possibly assist with evacuation if a serious problem arises. It is also important that everyone in the group accept the limitations of a chronic medical condition and help make conservative decisions during the trip that prevent the patient from overexertion. Pre-trip preparation includes identifying the closest medical facility and resources available should evacuation become necessary. Rescue insurance is often available and should be considered by the patient. Patients should have an adequate supply of medications and carry medications with them at all times to ensure that these do not become lost.

ENVIRONMENTAL EXTREMES AND CHRONIC MEDICAL CONDITIONS

Thermoregulation, hypothermia, heat illnesses, and high altitude are extensively reviewed elsewhere in this textbook. For the purpose of understanding how these environmental extremes might affect chronic medical conditions, some key points are worth emphasizing.

Cold

Exposure to cold causes peripheral vasoconstriction, tachycardia, and increased systemic blood pressure. Afterload and myocardial oxygen consumption are increased. Shivering is the primary mechanism for thermogenesis if core body temperature drops below normal. Physiologic cold acclimatization is not as important as learned behavioral responses. Therefore, potential problems related to cold exposure may be prevented by behavioral adaptations that maintain thermal protection. Even with appropriate preparation, however, some chronic medical conditions still increase the risk of accidental hypothermia or frostbite, or exacerbation of the underlying chronic medical condition may occur because of effects of the cold. Some chronic medical conditions and complications associated with cold exposure are:

- Cardiovascular disease (angina or congestive heart failure may occur because of increased systemic vascular resistance, afterload, and myocardial oxygen demand)
- Hypertension (exacerbated by cold-induced peripheral vasoconstriction)
- Peripheral arterial disease (increased risk of frostbite)
- Raynaud's phenomenon (increased risk of frostbite)
- Asthma (cold air– and exercise-induced bronchospasm)
- Elderly (impaired thermogenesis)
- Endocrine disease (impaired thermogenesis)
- Iron deficiency (impaired thermogenesis)
- Neuromuscular disease (impaired shivering thermogenesis may precipitate hypothermia)
- Diabetes (hypoglycemia may impair thermogenesis; peripheral neuropathy may predispose to cold injury)

Heat

The primary physiologic mechanisms to maintain core body temperature during exposure to heat are peripheral vasodilation and sweating. Sweating is the most important cooling mechanism and increases evaporative heat loss. High humidity decreases effective heat loss from sweating. Other mechanisms of heat loss (radiant, conductive, and convective) are inadequate when environmental temperature exceeds skin temperature. Adequate hydration is required for heat tolerance. Compared with adaptation to cold, adaptation to heat depends on physiologic acclimatization rather than behavior. Prior heat exposure reduces the skin temperature at which vasodilation and sweating occur, thus initiating cooling earlier. Physical training improves heat tolerance through the same mechanisms, and improves efficiency of the cardiovascular system. Poor physical

fitness, obesity, lack of heat acclimatization, and dehydration decrease heat tolerance. Chronic medical conditions increase risk of heat stroke, and heat may exacerbate chronic medical conditions:

- Diabetes (may predispose to dehydration; diabetic patients are less tolerant of dehydration)
- Cardiovascular disease (limited cardiac output in the face of peripheral vasodilation decreases heat tolerance and may lead to syncope or presyncopal symptoms)
- Skin disease (inadequate sweating predisposes to heat stroke)
- Anticholinergic drugs or diuretics (decrease heat tolerance)
- Sickle cell trait (increased risk of heat stroke)

To maximize tolerance to a hot environment, a program for heat acclimatization is important.[5] Before a wilderness trip to a hot environment, exercising daily in the heat for limited periods of at least 1 hour's duration for at least several days will improve heat acclimatization. If this is not possible before a trip, then limited periods of exercise during cool parts of the day for the first week of the trip are necessary for heat acclimatization. Adequate hydration with water or electrolyte drinks to maintain body volume status is important. During periods of prolonged exercise lasting several hours or more, meals or salty snacks with hydration are necessary to maintain electrolyte balance and prevent hyponatremia that can result from salt losses in sweat and hydrating with water alone. A copious clear urine output is a good indicator of adequate hydration status.

High Altitude

The primary physiologic insult at high altitude is hypobaric hypoxia. The physiologic process of high-altitude acclimatization occurs over time. If sufficient time for acclimatization is not allowed, then high-altitude illness may occur, which is associated with fluid retention and increased microvascular permeability in the brain or lung. The primary physiologic mechanism for acclimatization to high altitude is increased ventilation, which occurs within hours of ascent to altitudes greater than about 2000 m (6562 feet). Increased ventilation decreases alveolar carbon dioxide partial pressure and increases alveolar oxygen partial pressure according to the alveolar gas equation. Increased ventilation is both acute and chronic during stay at high altitude. Persons with chronic diseases who cannot increase ventilation to defend partial pressure of arterial oxygen (Pa_{O_2}) at high altitude may suffer complications of hypoxemia with end-organ compromise, such as myocardial ischemia, or be more predisposed to high-altitude illness. Diseases with ventilatory limitation include chronic obstructive pulmonary disease (COPD), obesity hypoventilation syndrome, and neuromuscular disease with involvement of respiratory muscles.

Normal acclimatization after ascent to high altitude is associated acutely with diuresis and a decrease in plasma volume. Fluid retention after acute ascent is associated with high-altitude illness. Increased sympathetic tone occurs acutely after ascent to high altitude and increases heart rate and blood pressure. Heart rate and blood pressure gradually decrease over several days at high altitude, but remain higher than sea level baseline values for the duration of stay at high altitude. Despite the increase in sympathetic tone, most persons with mild to moderate cardiovascular disease do well after ascent to moderate altitudes of approximately 2500 m (8202 feet).[108] Increased erythropoiesis is a long-term adaptation to high altitude that increases red cell mass and oxygen-carrying capacity of the blood and requires weeks to complete. Persons with chronic marrow suppression are unable to increase erythropoiesis to complete the normal long-term hematologic acclimatization.

The primary physiologic insult at high altitude is a decrease in partial pressure of inspired oxygen (P_{IO_2}). Fraction of inspired oxygen (F_{IO_2}) remains approximately 21% as altitude is increased, but ambient barometric pressure decreases. Any chronic disease that might be directly exacerbated by hypoxia may worsen after ascent to high altitude. For persons with normal gas exchange, however, after acute ascent to high altitude, significant decreases in arterial oxygen saturation do not occur until altitudes greater than approximately 3000 m (9843 feet) because of the sigmoidal shape of the oxyhemoglobin dissociation curve. In acclimatized persons, arterial oxygen saturation (Sa_{O_2}) will decrease below 90% when the Pa_{O_2} decreases below 55 mm Hg above approximately 3500 m (11,482 feet). Coronary artery disease and congestive heart failure can be directly influenced by hypoxia at high altitude, although most persons with mild to moderate disease do well after ascent to moderate altitudes of approximately 2500 m (8202 feet).[78] Moderate or severe pulmonary hypertension can be significantly worsened by hypoxia after ascent to high altitude. Even mild pulmonary hypertension can cause serious problems at high altitude because any degree of preexisting pulmonary hypertension predisposes to high altitude pulmonary edema.

The following chronic diseases may worsen at high altitude:

- COPD (inadequate ventilatory acclimatization, worsened hypoxemia)
- Asthma (exacerbation due to breathing cold dry air)
- Cardiac disease (cardiac ischemia or congestive heart failure precipitated by hypoxia)
- Pulmonary hypertension (worsened by hypoxemia; predisposes to high-altitude pulmonary edema)
- Obesity (hypoventilation increases risk of high-altitude illness)
- Neuromuscular disease (inadequate acclimatization if there is involvement of respiratory muscles)
- Hematologic disease (severe anemia, sickle cell disease, thalassemia)

CHRONIC MEDICAL CONDITIONS AND WILDERNESS TRAVEL

Asthma

Asthma is a common medical problem of individuals who pursue activities in the wilderness. Asthma is a disease of episodic airflow limitation, with different triggers causing exacerbations among patients. Classic symptoms are cough, wheezing, chest tightness, and shortness of breath, although these may not all occur simultaneously. Treatment of a patient with asthma planning a wilderness activity should begin with the basic elements of treatment for any patient with asthma: monitoring of symptoms, pharmacologic therapy, avoidance of triggers, and patient education. A pre-trip evaluation for asthma is an opportunity to ensure that the patient has optimal baseline treatment for asthma, which will also help prevent asthma attacks during a wilderness trip.

Patient monitoring of symptoms of asthma includes establishing a baseline peak expiratory flow (PEF). This objective parameter is measured after the patient takes in a full inspiration and then forcefully exhales into a peak flow meter.

Establishing a best PEF requires that the patient perform the maneuver several times a day and record the results in a diary. Typically, PEF is highest in the morning and lowest in the afternoon. The highest measured PEF becomes the baseline for the patient. PEF can then be monitored relative to the baseline to help identify an exacerbation early and escalate therapy for exacerbations. Variability in PEF is also a measure of the severity of asthma. The National Institutes of Health's *Guidelines for the Diagnosis and Management of Asthma*[54] recommends using a zone scheme for categorizing results of PEF: green is a PEF greater than 80% of personal best, yellow is a PEF 50% to 80% of personal best, and red is a PEF less than 50% of personal best. A PEF in the green zone indicates the patient should continue maintenance medications, whereas a PEF in the yellow or red zone requires adjustments in treatment according to a predetermined plan as well as seeking evaluation by a physician. This type of patient self-monitoring could easily be used on a wilderness trip. For example, a predetermined treatment plan for PEF in the yellow zone associated with symptoms of asthma might include acute treatment with a short acting β_2-adrenergic agonist inhaled medication followed by reevaluation in 1 hour, and if not improved to the green zone, initiation of oral steroids and an increase in the dose of inhaled steroids. A treatment plan for PEF in the red zone associated with symptoms of an asthma exacerbation would specify immediate therapy with inhaled β_2 agonist medications, to be repeated at frequent intervals, and initiating an oral corticosteroid. Persistent symptoms associated with PEF in the red zone suggest that evacuation from the wilderness environment should be initiated.

Of interest is the PiKo-1 (Ferraris Respiratory, Louisville, CO), a new alternative to a portable hand-held PEF meter for monitoring asthma. The PiKo-1 measures both PEF and forced expiratory volume in 1 second (FEV_1) and is small and easy to pack. It is electronic with an estimated 2-year battery life. Both the PiKo-1 and PEF meters read significantly lower than actual PEF with cold temperatures and high altitude.[102]

Patient education is an important element of asthma treatment before a wilderness trip. It begins with instructing patients on recording symptoms and PEF in a diary. It is also imperative that the patients' technique in using inhaled medication be observed and instruction provided if necessary. Inhaled medications for asthma are of less benefit when they are not administered properly.

Identifying and avoiding asthma triggers is essential to preventing asthma exacerbations. Asthma triggers generally fall into six categories: allergens, respiratory infections, irritants, chemicals, exercise, and emotional stress. After triggers are identified, a plan is made to avoid them. The patient generally has three choices: entirely avoid the asthma trigger, minimize exposure to the asthma trigger, or pretreat with asthma medications. Patients with asthma pursuing a wilderness trip may not be able entirely to avoid an asthma trigger, such as breathing cold, dry air during exercise on a mountaineering expedition.

Some studies suggest that travel to high altitude may actually reduce bronchial hyperresponsiveness.[2,26] This is the anecdotal experience of patients with asthma who pursue climbing at high altitude. Many have an improvement in symptoms of asthma at high altitude, whereas some who have relatively mild asthma at low altitude experience asthma exacerbations exercising in the cold, dry air at high altitude. The difference in these two groups probably relates to triggers for asthma. Patients with asthma who improve at high altitude have primarily allergen triggers for their asthma that are absent in an environment with few allergens. Patients who have more exacerbations at high altitude have a primary trigger of exercise in cold, dry air.

Another key to preparation for a wilderness trip for an asthmatic patient is to ensure that appropriate pharmacologic treatment is administered ahead of time. The National Institutes of Health's *Guidelines for the Diagnosis and Management of Asthma*[54] provides definitions of categories of severity for patients with asthma and the appropriate treatment (Table 29-1). Pre-trip evaluation for a patient with asthma should include a review of these guidelines in relation to the patient's symptoms, and consideration of stepping up therapy before a wilderness trip. For example, a patient who is not usually maintained on montelukast, a leukotriene inhibitor, or an inhaled steroid might want to consider adding one of these agents to step up therapy. Because a primary trigger for asthma on a wilderness trip is exercise, adding montelukast may be helpful because of its efficacy as an adjunctive therapy for exercise-induced asthma,[76] in addition to inhaled β_2 agonists used before exercise.[90]

Golan and colleagues[51] reported the only prospective study in the literature of patients with asthma who engaged in adventure travel activities, including high-altitude trekking. Among 5835 consecutive patients who presented to their travel clinic, they identified 203 with asthma (3.5%). Most patients had mild to moderate asthma. Dust, exercise, and respiratory tract infection were the most common listed triggers. Asthma exacerbations occurred in 88 of the patients (43%) during their trip, most commonly associated with trekking or exercise. Among the patients with asthma exacerbations, 40 reported worsening asthma during travel, 32 experienced the worst asthma attack ever, and 11 reported a life-threatening asthma attack. Two independent risk factors for attacks during travel were identified: frequent use (more than three times weekly) of inhaled bronchodilators before travel, and participation in intensive physical exertion during treks. Interestingly, pre-trip exercise testing with pre-exercise and postexercise spirometry was not helpful in predicting which patients would experience an asthma exacerbation. This study is important because it emphasizes that patients with asthma who are pursuing wilderness trips that include an increased level of exercise may experience an asthma exacerbation, and it may be difficult to predict in which patients this will occur. All patients with asthma traveling to remote areas need to carry a supply of medications for treatment of an exacerbation, most notably bronchodilators and oral corticosteroids.

Chronic Obstructive Pulmonary Disease

Chronic obstructive pulmonary disease is a syndrome of progressive airflow limitation caused by chronic inflammation of the airways and lung parenchyma.[6] COPD has a prevalence of approximately 4 to 7 per 1000 persons in developed countries. The limitation that COPD imposes on a patient intending to pursue an activity in the wilderness depends on severity. The Global Initiative for Chronic Obstructive Lung Disease[42] (GOLD) defined a staging system to grade severity based on airflow limitation as measured by the FEV_1 (Table 29-2). The GOLD severity classification includes patients with COPD who have airflow obstruction, as defined by a ratio of FEV_1 to forced vital capacity (FVC) of less than 70%. Severity is then further categorized by the postbronchodilator FEV_1. An FEV_1 that is

TABLE 29-1. Stepwise Approach for Managing Asthma in Adults and Children Older than 5 Years of Age: Preferred Treatments

CLASSIFY SEVERITY: CLINICAL FEATURES BEFORE TREATMENT OR ADEQUATE CONTROL	DAILY MEDICATIONS REQUIRED TO MAINTAIN LONG-TERM CONTROL
Step 4: Severe Persistent Continual symptoms, limited physical activity, frequent exacerbations Nights with symptoms: frequent PEF or FEV_1: <60%–80% PEF variability: >30%	High-dose inhaled corticosteroid AND Long-acting inhaled β_2 agonist AND Oral corticosteroids if necessary
Step 3: Moderate Persistent Daily symptoms, daily use of inhaled short-acting β_2 agonist, exacerbations affect activity Nights with symptoms: ≥5/mo PEF or FEV_1: >60% to <80% PEF variability: >30%	Low- to medium-dose inhaled corticosteroid and long-acting inhaled β_2 agonist OR Low- to medium-dose inhaled corticosteroid and leukotriene modifier or theophylline
Step 2: Mild Persistent Symptoms >2 times a week but less than daily, exacerbations may affect activity Nights with symptoms: ≥2/mo PEF or FEV_1: >80% PEF variability: 20%–30%	Low-dose inhaled corticosteroid OR Leukotriene modifier, cromolyn, nedocromil, or sustained-release theophylline
Step 1: Mild Intermittent Symptoms ≤2 times a week, normal PEF between exacerbations Nights with symptoms: ≤2/mo PEF or FEV_1: >80% PEF variability: <20%	No daily medication needed Exacerbations may require treatment with a course of systemic corticosteroids. Long periods of normal lung function and no symptoms between exacerbations
All Patients	Quick relief treatment with a short-acting inhaled β_2 agonist

Percentage predicted values for forced expiratory volume in 1 second (FEV_1) and percentage of personal best for peak expiratory flow (PEF).
Adapted from Guidelines for the Diagnosis and Management of Asthma. Expert Panel Report 2: National Institutes of Health, National Heart, Lung and Blood Institute. NIH publication no. 97-4051. Bethesda, MD, National Institutes of Health, 1997.

TABLE 29-2. Global Initiative for Chronic Obstructive Lung Disease (GOLD) Classification of Severity of COPD and Therapy

STAGE 0: AT RISK	STAGE I: MILD	STAGE II: MODERATE	STAGE III: SEVERE	STAGE IV: VERY SEVERE
Chronic symptoms Risk factors Normal spirometry	$FEV_1/FVC < 70\%$ $FEV_1 \geq 80\%$	$FEV_1/FVC < 70\%$ $50\% \geq FEV_1 \leq 80\%$	$FEV_1/FVC < 70\%$ $30\% \geq FEV_1 \leq 50\%$	$FEV_1/FVC < 70\%$ $FEV_1 \leq 30\%$ *or* $FEV_1 \leq 50\%$ with chronic respiratory failure
Avoidance of risk factors; influenza vaccination				
	Add short-acting bronchodilator when needed			
		Add regular treatment with one or more long-acting bronchodilators and add rehabilitation		
			Add inhaled corticosteroids if repeated exacerbations	
				Add long-term oxygen if chronic respiratory failure

FEV_1, forced expiratory volume in 1 second, reported as percentage predicted; FVC, forced vital capacity; FEV_1/FVC, ratio reported as percentage predicted.
Adapted from Fabbri LM, Hurd SS: Eur Respir J 22:1–2, 2003, with permission.

greater than 80% predicted associated with symptoms of COPD is mild or stage I, an FEV_1 50% to 79% predicted is moderate or stage II, an FEV_1 30% to 49% predicted is severe or stage III, and an FEV_1 less than 30% predicted is very severe or stage IV. Patients in the mild category will probably do well on a wilderness activity that is selected to match their maximum exercise capacity. Patients in the moderate category need a careful evaluation to determine their baseline symptoms and exercise tolerance. Cardiopulmonary exercise testing may be helpful in these patients to determine exercise capacity and gauge the appropriateness of an intended wilderness trip.[3] Patients in the severe or very severe categories, or any patient

with chronic respiratory failure or right heart failure, should be advised against any wilderness trip where an increase in their current level of exertion might be required, or if the intended trip is at a higher altitude than where they live. Such patients warrant pulmonary consultation before significant travel to ensure that they are maximally medically treated and are provided with supplemental oxygen if indicated.

Patient education and optimal medical management for COPD should occur before any wilderness trip. Patient education includes counseling on smoking cessation (unlikely to be successful just before a wilderness trip) and ensuring that the patient has optimal inhaled bronchodilator therapy for COPD. Inhaled bronchodilators are the foundation of pharmacotherapy for COPD because of their capacity to alleviate symptoms, decrease exacerbations of disease, and improve exercise tolerance by decreasing hyperinflation and airflow obstruction.[123] Patients with COPD can have an increased work of breathing during exercise because hyperinflation (increase in functional residual capacity) places respiratory muscles at a mechanical disadvantage.[99] Paradoxically, improvement in function resulting from administration of bronchodilators is not always reflected by changes in FEV_1 and FVC.[98] Compared with patients with asthma, patients with COPD do not uniformly demonstrate airway hyperresponsiveness with methacholine challenge, and do not uniformly have a response to bronchodilators with an increase in FEV_1 of 12% or greater. Despite this, bronchodilator therapy results in improvement over time in FEV_1 or parameters of lung hyperinflation in patients with COPD,[19,123] and inhaled bronchodilator therapy with selective β_2 agonist medications or anticholinergics is the mainstay of therapy for COPD. In addition to prescribing appropriate bronchodilators, patient education should be provided for proper technique for inhaled bronchodilators administered by a metered-dose inhaler with a spacer, or by dry-powder inhaler. If proper technique is used, the therapeutic effect is similar to that with nebulized bronchodilator therapy.

Mild COPD can be treated effectively with short-acting bronchodilators used on an as-needed basis. The selective β_2 agonist albuterol and the anticholinergic ipratropium bromide are equally effective.[123] Moderate COPD requires scheduled therapy and as-needed rescue therapy. Albuterol has a rapid onset of action and is the main treatment for rescue therapy to treat acute bronchospasm. Maintenance therapy with β_2 agonists may be accomplished with scheduled treatments of short-acting albuterol or longer-acting salmeterol or formoterol. More frequent dosing of the short-acting albuterol may achieve a greater degree of bronchodilation, but may also cause side effects of tremor, tachycardia, and, in rare instances, hypokalemia. The long-acting anticholinergic tiotropium bromide may be used instead of a long-acting β_2 agonist for scheduled pharmacologic bronchodilation in moderate or severe COPD (the maximum effect may not occur for approximately 1 week after starting drug therapy with tiotropium). Combination bronchodilator therapy with a β_2 agonist and an anticholinergic should be used when the patient fails to achieve symptomatic relief with a single agent alone.[55] Either a combination of the short-acting albuterol and ipratropium, or the longer-acting salmeterol and tiotropium may be used. The shorter-acting combination can be used on a scheduled or as-needed basis. The longer-acting combination should be used on a schedule because the longer-acting bronchodilator salmeterol is not appropriate for acute, as-needed treatment. The longer-acting β_2 agonist formoterol can be used as needed because it has a rapid onset of action compared with salmeterol, but its long half-life precludes use of formoterol more frequently than every 12 hours.

Other adjunctive treatments for COPD include theophylline and inhaled corticosteroids. Theophylline is an effective adjunct to therapy with β_2 agonist and anticholinergic bronchodilators, but requires careful monitoring of levels to prevent toxicity. For this reason, patients on theophylline who intend to go on a wilderness trip should have their theophylline level checked to ensure that the drug is not overdosed. It is probably not wise to add theophylline as a new agent for treatment of COPD just before a wilderness trip because of the risk of toxicity. Inhaled corticosteroids are controversial in COPD, but are recommended as an additional therapy in patients with moderate to severe COPD who have persistent symptoms despite optimal bronchodilator therapy or who have frequent exacerbations.[42] Inhaled corticosteroids should be used for several weeks before a response to the treatment is evaluated, and if started before a wilderness trip should also be used for several weeks before the trip.

In addition to optimizing therapy for stable COPD before a wilderness trip, medications should be carried, and an evacuation route planned, in the event of an exacerbation of COPD during a wilderness trip. COPD exacerbations are characterized by increased dyspnea and sputum production and are most commonly caused by bacterial or viral infections. Hypoxemia, increased airflow obstruction, and, in the worst case, respiratory failure may occur. Inhaled bronchodilators, corticosteroids, antibiotics, and supplemental oxygen are the mainstays of therapy. These medications may not be practical to administer in a wilderness setting, but modifications can be made to treat a COPD exacerbation in a remote area. Although the β_2 agonist and anticholinergic bronchodilators albuterol and ipratropium are usually administered by nebulizer when treating a COPD exacerbation in a hospital setting, they may be administered by inhaler with equal efficacy using good technique. Corticosteroids may be administered orally rather than intravenously, and a supply of prednisone should be carried. The standard antibiotics for a COPD exacerbation may all be given orally: amoxicillin, doxycycline, or trimethoprim-sulfamethoxazole.

Availability of supplemental oxygen is a consideration for some patients with moderate to severe COPD when traveling to a remote area if prior exacerbations were associated with hypoxemia. Any patient with a baseline resting Pa_{O_2} of less than 55 mm Hg or an Sa_{O_2} less than 88% should be treated with chronic supplemental oxygen and should make plans to carry oxygen with them on any wilderness trip. The traditional supplemental medical oxygen delivery system is a continuous flow of 100% oxygen from a compressed gas cylinder delivered by nasal cannula. The disadvantage to this system is an inefficient use of oxygen because only a small percentage of the oxygen delivered to the nose actually reaches the lungs. In a wilderness setting, the weight and space required to carry medical oxygen cylinders create a significant burden and limit the duration of a trip. An alternative to provide more efficient use of a limited supply of oxygen is a pneumatic nonelectronic demand valve that delivers flow of oxygen only on inspiration.[127] Portable liquid oxygen units with demand valves are an alternative to oxygen cylinders and offer advantages of lighter weight for a comparable amount of oxygen, but are more expensive. Any patient using a demand valve system for a wilderness trip should

be evaluated during rest and during exercise to ensure that adequate oxygenation is maintained.[128] Patients with chronic hypoxemia requiring supplemental oxygen therapy are not capable of wilderness trips that require sustained increases in physical activity, but may be capable of a vehicle or horse-packing trip into a wilderness area for an activity such as fishing. Portable oxygen sufficient for the trip should be taken. Any patient with COPD who will travel to a higher altitude for a wilderness trip should be evaluated for the need for supplemental oxygen, or an increase in the current level of supplemental oxygen if he or she already is being treated with oxygen. Several methods may be used to estimate oxygen requirements at higher altitudes,[28,80,122] including having the patient breathe a decreased FIO_2 that would approximate the PIO_2 at the altitude of the planned trip. Adjustments may also be required for commercial airline travel, where physiologic cabin pressure is equivalent to an altitude of approximately 2000 to 2500 m (6562 to 8202 feet).

Diabetes

Approximately 17 million people with diabetes live in the United States (7% of the U.S. population), so it is likely that diabetes will be encountered in persons pursuing wilderness activities.[25] Diabetes encompasses the disorders of type 1 diabetes, previously known as insulin-dependent or juvenile-onset diabetes, and type 2 diabetes, previously known as non–insulin-dependent or adult-onset diabetes. Approximately 5% to 10% of diabetic patients have type 1 disease, and 80% have type 2, with the remainder of cases due to other causes. The pathophysiology of type 1 diabetes generally relates to inadequate insulin production, whereas the pathophysiology of type 2 diabetes generally relates to increased peripheral resistance to insulin. Patients with type 1 diabetes are usually treated with insulin, whereas type 2 diabetes may be treated with diet and exercise, oral hypoglycemic agents, or insulin.

Increased physical activity is beneficial for diabetic patients, particularly those with type 2 disease. However, engaging in nonroutine activities creates certain safety issues for the patient with diabetes, so advising the patient on how to manage diabetes appropriately during exercise in an outdoor environment is essential. High-risk wilderness activities, such as mountaineering or rock climbing, where loss of focus and concentration may result in death, would not be appropriate for a diabetic patient who is susceptible to hypoglycemia unless the person is constantly monitored by a climbing partner, does not climb in the lead, and is always secured by a rope. Similarly, solo wilderness activities may not be appropriate for diabetic patients who may become hypoglycemic.

Patients with diabetes require evaluation for several issues before a wilderness trip. The extent of increased exercise that will be undertaken on the trip needs to be determined in relation to the patient's current level of physical activity and his or her glucose control. In addition to exercise, the specific activity and associated travel are likely to effect a change in diet. Screening for conditions associated with diabetes, such as cardiovascular disease, peripheral neuropathy, proliferative retinopathy, and nephropathy, should be done. These conditions may significantly influence the ability of diabetic patients to pursue wilderness activities safely and may require further evaluation and testing before the trip. Last, the effect of the change in environment on the diabetic patient is considered, including potential problems with exposure to heat or cold.

The effect of increased exercise is important for diabetic patients because it may precipitate hypoglycemia or hyperglycemia, depending on the timing of the last dose of insulin.[89] The normal response to exercise in nondiabetic people entails a decrease in insulin secretion as serum glucose levels fall because of uptake of glucose by exercising muscle, and an increase in hepatic glucose release in response to catecholamines, glucagon, and growth hormone to maintain blood glucose levels. Patients with type 1 diabetes who are hypoinsulinemic when they exercise (as may occur if they decrease their insulin too much in an effort to accommodate the increased physical activity) may become hyperglycemic because of increased hepatic release of glucose into the blood, but insufficient insulin to allow glucose to enter the cells. Thus, hyperglycemia results in decreased exercise capacity because exercising muscle is depleted of glucose in the absence of insulin to enable glucose to enter the cells. Under these conditions, the substrate for fuel becomes free fatty acids released from adipocytes, with generation of ketone bodies by the liver. Hypovolemia may occur due to glycosuria; if the process persists, diabetic ketoacidosis may ensue.

Patients with type 1 diabetes who have exogenous insulin in their system when they exercise may become hypoglycemic. Exercise results in an increase in insulin entering the blood, rather than the expected physiologic decrease in insulin with exercise. The increased insulin level enhances glucose uptake by the muscles and decreases release of glucose from the liver. Both result in a fall in blood glucose to a greater degree than in nondiabetic people during exercise. Delayed hypoglycemia may occur hours after exercise as the resting muscle takes up glucose from the blood to replenish glycogen stores. Therefore, monitoring glucose levels, taking nutrition, and adjusting the dose of insulin are also important after exercise.

Another factor contributing to hypoglycemia in the exercising patient with type 1 diabetes is increased exogenous insulin mobilization from subcutaneous tissue because of increased blood flow.[46,67] Inadvertent intramuscular injection would exaggerate this phenomenon. It is important for patients with type 1 diabetes to administer their dose of subcutaneous insulin before exercise in a location away from exercising muscle. They should avoid injections into the arms and legs, instead using the abdomen. Insulin absorption is fastest and most consistent when it is injected into the abdomen. Absorption is generally slower from the arms and slowest from the thighs or buttocks, but the rate may be more inconsistent depending on the type of exercise. If the planned exercise uses primarily arm muscles, such as sport climbing, then it may be beneficial to give the insulin injection in the abdomen to avoid increased absorption from the exercising arm muscles. Likewise, if the planned activity is hiking or skiing, then it would be beneficial to use the arm or abdomen to avoid increased absorption from the exercising leg muscles. Absorption of subcutaneous insulin during exercise may also depend on the type of insulin used. A study of the long-acting insulin analog glargine injected subcutaneously into the thigh on the evening before an intense 30-minute exercise session in patients with type 1 diabetes did not show an increased rate of absorption, but plasma glucose did fall during exercise.[104]

Another measure to prevent exercise-associated hypoglycemia is to reduce the dose of insulin that will be in effect during exercise.[86] The best strategy for a patient with type 1 diabetes is to monitor blood glucose before, during, and after exercise to predict changes, and adjust insulin doses accord-

ingly. This means that before a wilderness trip, the type 1 diabetic patient should exercise daily at a similar level of physical activity as anticipated on the wilderness trip so that adjustments in insulin dosing can be made. Differences in nutritional intake, in addition to increased exercise, during the wilderness trip should also be anticipated when planning insulin regimens. Patients with type 1 diabetes may engage in strenuous physical activity without experiencing problems, but it is important for the patient to focus on the timing of exercise in relation to meals and insulin dosing.[68,125] Another consideration is whether regular insulin or a rapidly acting insulin analog (lispro or aspart) is used for prandial dosing. Rapidly acting insulin alters the timing of exercise-related hypoglycemia. Patients who exercise early in the postprandial period (1 to 3 hours after a meal) require a decrease in the dose of rapidly acting insulin, whereas those who exercise later (3 to 5 hours) require a smaller change or none.[61] A predictable exercise schedule on a wilderness trip would be useful for the patient with type 1 diabetes, although this may be difficult depending on the type of trip and the environment.

Other members of the group need to be aware of signs and symptoms of hypoglycemia in the diabetic patient, and how to render appropriate treatment. This is especially important for patients with type 1 diabetes, but also is significant for patients with type 2 diabetes who are on oral hypoglycemic drugs or insulin because this group of diabetic patients also experiences a decrease in serum glucose during exercise.[105] Preventive measures include ingesting extra food in the form of 15 to 30 g of quickly absorbed carbohydrate (e.g., glucose tablets, hard candies, or juice), which should be taken 15 to 30 minutes before exercise and approximately every 30 minutes during exercise. Patients are also at risk of late hypoglycemia (i.e., 4 to 8 hours after the termination of exercise) because of replenishment of depleted glycogen stores. This can usually be avoided by ingesting slowly absorbed carbohydrates (dried fruit, granola bars, or trail mix) immediately after exercise.[120] Fluid intake should also be increased to ensure adequate hydration.

The acute symptoms of hypoglycemia are mediated by the adrenergic and cholinergic nervous systems and are manifested by shakiness, tremors, palpitations, cold sweats, hunger, and nervousness/anxiety. If these are ignored, not sensed (owing to hypoglycemic unawareness), or cannot be treated immediately, the person may experience neuroglycopenic symptoms because of inadequate amounts of glucose reaching the brain. Neuroglycopenic symptoms consist of headache, loss of concentration, irritability, and, if allowed to proceed long enough, even seizures and loss of consciousness. All persons with diabetes should be questioned about their history of hypoglycemic reactions (frequency and severity) as well as their ability to sense the reactions (history of hypoglycemic unawareness). Travel into the wilderness should be discouraged in persons with a history of severe or frequent reactions or a history of hypoglycemic unawareness. Companions should be taught to recognize the signs and be knowledgeable in the treatment of hypoglycemia.

If the person with diabetes is alert and cooperative, he or she may be treated with 15 g of a rapidly absorbed carbohydrate (e.g., glucose tablets, juice, or hard candies). The blood glucose should be checked in 15 minutes to ensure that the glucose level has increased to a safe level (≥100 mg/dL) before continuing with the physical activity. The person should be closely watched for evidence of recurrent symptoms. If the person becomes uncooperative or loses consciousness, a glucagon injection kit should be available. Glucagon releases glucose stored in the liver and helps raise the glucose level to the point where the person becomes alert and treatment may be continued with an oral, rapid-acting carbohydrate. The dose of glucagon is 1 mg (1 ampule) given either subcutaneously or intramuscularly.

Activity limitations for diabetic patients also depend on associated complications. Patients with proliferative retinopathy should avoid activities that can rapidly or explosively elevate intraocular pressure because vitreous hemorrhage may occur. Bleeding from damaged retinal capillaries may also occur during a Valsalva maneuver, while lifting heavy objects, or during a collision. An expert should do an examination for early signs of retinopathy at least once a year.[34]

Patients with peripheral neuropathy are at risk from pressure ulceration to the feet and should avoid traumatic weight-bearing exercise (long-distance running or prolonged downhill skiing). Well-fitting protective footgear helps prevent this complication. Diabetic patients with peripheral neuropathy are also more susceptible to cold injury of the feet in a cold environment, and patients, as well as group members, need to monitor their feet meticulously. In a cold weather environment, it is especially important to have well-insulated warm footgear with dry socks.

Autonomic neuropathy also poses problems with increased activity in a wilderness environment. Cardiac responsiveness may be delayed during rapidly changing exercise levels. Gastroparesis may make absorption of carbohydrates and oral hypoglycemics unpredictable and could result in a mismatch in timing between insulin and glucose peak.

Ensuring that insulin does not freeze and glucose testing equipment works properly are also important for the insulin-dependent diabetic patient in the wilderness. Strategies to ensure that insulin does not freeze include carrying the medication inside a jacket next to the body or storing the insulin in an insulated container and putting the container in the sleeping bag at night. Glucose testing equipment should be reliable, rapid, and used frequently to help identify impending hypoglycemia and increase carbohydrate ingestion before incapacitation occurs.

Provided that the insulin-treated patient with diabetes has reviewed all the factors associated with a wilderness trip that may be complicated by diabetes, and has chosen to undertake the preparation required and accept the risk while on the trip, then a treatment plan needs to be generated. The most challenging wilderness trips for maintaining glucose control in diabetic patients are those where the level of exercise is consistently increased above usual. Intensive diabetes management can be achieved with multiple daily injections of insulin, or an insulin pump, and numerous checks of blood glucose throughout the day (Tables 29-3 and 29-4). A standard strategy to offset the increased efficiency of insulin due to exercise is to ingest 15 to 30 g of carbohydrate for every half hour of moderate aerobic exercise. Monitoring blood glucose during periods of exercise of several hours' duration is important. Adjusting the dose of insulin downward may be required if carbohydrate supplementation does not prevent hypoglycemia. It may be necessary to decrease the dose of insulin by 30% or more.[34,86] Blood glucose should be checked more often early during a wilderness trip to assess how to balance insulin, carbohydrate intake, and exercise. It is also critical to prevent delayed hypoglycemia that often occurs after muscle and liver glycogen stores have been depleted

TABLE 29-3. Matching Insulin Treatment Schedules with Exercise Schedules

TREATMENT TYPE	ADVANTAGES	DISADVANTAGES
Standard: two injections, mixed intermediate- and short-acting insulins	Easy to perform	Poor match with exercise, rigid time restraints, least likely to give good metabolic control and health
Intensive: three or more injections a day	Better control, more flexible timing, less hypoglycemia in the evening	More frequent testing, harder to learn
Extended: ultralente or glargine for basal plus lispro/aspart for meals	Least amount of time rigidity, most protection against hypoglycemia, excellent metabolic control	Much more effort to master and do well
Continuous infusion (pump)	Most flexible (no injections most days), low hypoglycemia risk overnight, best metabolic control	Needs expensive device, harder to master, must remove pump for some activities; need to carry syringes in case of mechanical failure

Adapted from Draznin MB: Physician Sportsmed 28(12):49–56, 2000, with permission.

TABLE 29-4. Types, Names, and Uses of Insulin

INSULIN	ONSET OF ACTION	PEAK ACTION	EFFECTIVE DURATION	USES	NOTES
Short-acting insulin analogs: lispro, aspart	5–15 min	30–90 min	4–6 hr	Meal coverage if taken up to 15 min before eating; adjust for elevated blood glucose levels	Insulin of choice for pumps Rapid action may not be desirable during periods of physical activity because it may lead to more hypoglycemia
Regular	30–60 min	2–3 hr	8–10 hr	Meal coverage if taken 30–45 min before eating	May last too long, leading to hypoglycemia
Intermediate: NPH	2–4 hr	4–10 hr	12–18 hr	Meal coverage (lunch) if taken in the morning	More active early in the dosage or can linger longer than anticipated; either can cause hypoglycemia
Intermediate zinc insulin: lente	2–4 hr	4–12 hr	12–20 hr	Basal, between meals	Requires snacks to cover extended peaks before or after lunch (both in young children)
Very long-acting zinc insulin: ultralente	6–10 hr	10–16 hr	18–24 hr	Basal, between meals	Can be used once a day, but dosage is often split; has a peak action about 10 hr after injection
Very long-acting insulin analog: glargine	2–4 hr	None	20–24 hr	Basal	Once-a-day injection; cannot be mixed with other insulins; soluble until injected

NPH, neutral protamine hagedorn.
A typical intensive schedule of injections would be a mixture of either NPH or lente with lispro before breakfast, lispro alone at dinner, and either NPH or intermediate insulin zinc suspension at bedtime.
Testing is done before meals and at bedtime regardless of the type of insulin used. To establish the dose (given by injection or by pump) for long-acting insulin plus lispro for meals, the patient also tests blood glucose 2 hr after each meal for 3–4 wk. Careful record keeping will determine if assumptions are correct and which doses work best. Testing should be done initially every 30–60 min during exercise to determine the effect of the activity on the blood glucose levels. Testing should be done every 2 hr after the completion of exercise for 6–12 hr to monitor for late-onset hypoglycemia.
Adapted from Draznin MB: Physician Sportsmed 28(12):49–56, 2000, and Hirsch IB: N Engl J Med 352:174–183, 2005, with permission.

and not replenished during exercise. The use of intermediate-acting insulin may need to be shifted from the afternoon or early evening to bedtime, or a long-acting insulin preparation that does not peak may be used.

Diabetic patients may pursue the same wilderness activities as other individuals, provided they have the experience necessary to participate safely, just as any other individual. This includes activities such as high-altitude mountaineering.[74] On an expedition to Cho Oyu, an 8201-m (26,906-ft) peak in the Nepal Himalaya, six type 1 diabetic patients participated in the expedition and one reached the summit.[100,101] These climbers were highly motivated and adhered to careful glucose monitoring, and all completed the expedition safely and free from long-term complications. Their cardiovascular parameters were comparable to those of nondiabetic control subjects on the same expedition, although the patients with diabetes did have a worsening in metabolic control and required higher insulin doses than at sea level.

Peripheral Arterial Disease

Peripheral arterial disease is caused by atherosclerotic occlusion of the arteries of the legs and affects approximately 10 million people in the United States, with an age-adjusted prevalence of 12%. Approximately 25% of patients with peripheral arterial

disease are symptomatic with claudication, and approximately 10% have critical ischemia.[121] Because peripheral arterial disease is common, it may be present in persons intending to pursue wilderness activities, especially the elderly and those with other manifestations of systemic atherosclerosis. The severity of peripheral arterial disease is closely associated with the risk of myocardial infarction, ischemic stroke, and death from vascular causes. The major risk factors for peripheral arterial disease are older age (>40 years), cigarette smoking, and diabetes mellitus. Hyperlipidemia, hypertension, and hyperhomocystinemia are also important risk factors.[58]

When evaluating a patient with peripheral arterial disease for a wilderness activity it is helpful to characterize the severity of the disease and how it affects functional capacity. The most useful method for evaluating severity is the ankle-to-arm ratio of systolic blood pressure (ankle–brachial index), which is easily obtained with a standard blood pressure cuff and a Doppler device.[58] Systolic blood pressure is measured by Doppler ultrasonography in each arm and in the dorsalis pedis and posterior tibial arteries in each ankle. The higher of the two arm pressures is selected, as is the higher of the two pressures in each ankle to calculate a left and right ankle–brachial index. An ankle-to-arm pressure ratio of 0.91 to 1.3 is normal. A ratio of 0.41 to 0.9 indicates mild to moderate peripheral arterial disease, and less than 0.4 is severe. A ratio of greater than 1.3 indicates a calcified noncompressible vessel. Patients with claudication, defined as walking-induced pain in one or both legs (primarily in the calves) that does not go away with continued walking and is relieved by rest, typically have ankle–brachial indexes in the mild to moderate range. Patients with critical ischemia have ankle–brachial indexes in the severe range.

Increased exercise is associated with wilderness activities and this may be a limiting factor for patients with peripheral vascular disease who have claudication. Hiking or trekking, however, are reasonable activities for patients with claudication if they engage in a regular exercise training program. The benefit of exercise in improving functional capacity in patients with claudication is well proven.[121] One meta-analysis concluded that exercise training improved pain-free walking time in patients with claudication by an average of 180% and improved maximal walking time by an average of 120%.[48] Exercise-induced increases in functional capacity and lessening of claudication symptoms are primarily due to improvements in endothelial vasodilator function, skeletal muscle metabolism, blood viscosity, and biomechanics of walking. Increases in leg blood flow and oxygen delivery may also occur, but do not appear to account for the large improvements in exercise capacity that are observed. A supervised exercise program several times a week is recommended. The key elements of each session include a warm-up period followed by walking on a track or treadmill at a workload that causes claudication in about 5 minutes. Then, continue at that workload until claudication of moderate severity occurs, then rest standing or sitting for a brief period to allow symptoms to subside, and repeat the exercise–rest pattern for the duration of the session, lasting initially approximately 30 minutes but working up to about an hour. This exercise strategy might also be used while hiking, repeating periods of exercise and rest as gauged by claudication symptoms, and could potentially lead to improvements in exercise capacity over time. A walking or hiking exercise program needs to be done regularly and sustained over months or the benefits diminish.

Patients with peripheral arterial disease who wish to pursue wilderness activities or an exercise program should undergo evaluation for heart disease, hypertension, hyperlipidemia, and diabetes. Appropriate treatment and risk factor modification should be initiated (including smoking cessation). In addition, an exercise test with 12-lead electrocardiographic monitoring should be performed to evaluate for cardiac ischemia. Patients with claudication may be limited by leg pain during a maximal exercise test, thus limiting the stress on the heart, but the information gained regarding heart rate and blood pressure response, work level at which claudication occurs, and exercise capacity is useful for estimating the type of wilderness activity that may be pursued and in formulating a prescription for exercise training.

Treatment of claudication should include a formal walking-based exercise program. In addition, several options exist for drug therapy. The Seventh ACCP Consensus Conference on Antithrombotic and Thrombolytic Therapy recommends lifelong aspirin therapy in patients with peripheral arterial disease.[24] Clopidogrel is a thienopyridine drug that inhibits platelet activation and is an alternative to aspirin, and has fewer hematologic side effects than the related drug ticlopidine. Clopidogrel has U.S. Food and Drug Administration (FDA) approval for prevention of ischemic events in patients with peripheral arterial disease, but aspirin is usually recommended over clopidogrel.[24,58] Cilostazol and pentoxifylline are the two drugs approved in the United States for treating claudication. Cilostazol is a phosphodiesterase inhibitor that suppresses platelet aggregation and is a direct arterial vasodilator. Cilostazol improves both pain-free and maximal treadmill walking distance in patients with stable moderate to severe claudication,[126] and is recommended for patients with more severe disabling claudication.[24] Because other oral phosphodiesterase inhibitors used for inotropic therapy have caused increased mortality in patients with advanced heart failure, cilostazol is contraindicated in heart failure of any severity. Pentoxifylline is a methylxanthine derivative that improves deformability of red cells, lowers plasma fibrinogen concentrations, and has antiplatelet effects. It is not as effective in improving walking ability as cilostazol.[29] The Seventh ACCP Consensus Conference did not recommend pentoxifylline for treatment of peripheral arterial disease.[24]

Patients with symptomatic peripheral arterial disease can pursue activities in the wilderness that are appropriate for their functional capacity. Such activities should include walking or hiking and allow for periods of rest when necessary. An exercise program along with drug therapy can improve ability to participate in wilderness activities. Environmental extremes such as cold and high altitude may worsen limb ischemia and predispose to frostbite.

Raynaud's Phenomenon

Raynaud's phenomenon is an exaggerated vasoconstrictor response to cold or emotional stress in digital arteries, precapillary arterioles, and cutaneous arteriovenous shunts of the fingers or toes. It presents clinically as pallor, cyanosis, and often rubor of the skin[134] (Fig. 29-1). It occurs more commonly in the hands than in the feet. Pallor shows the vasospasm and loss of arterial blood flow, cyanosis shows the deoxygenation of static venous blood, and rubor shows reactive hyperemia after return of blood flow. This classic three-phase color change occurs in approximately two thirds of affected patients; the remainder

Figure 29-1. Active Raynaud's phenomenon. **A,** Sharply demarcated pallor resulting from the closure of digital arteries. **B,** Digital cyanosis of the fingertips in a patient with primary Raynaud's phenomenon. (From Wigley FM: N Engl J Med 347:1001–1008, 2002. Copyright © 2002 Massachusetts Medical Society. All rights reserved.)

have only pallor and cyanosis.[12] Numbness, tingling, and burning frequently accompany these changes, particularly after reperfusion of the vascular beds with rewarming. The ischemic phase of the attack is evidenced by demarcated pale or cyanotic skin limited to the digits. It typically starts in one or several digits after exposure to cold and then spreads symmetrically to all fingers of both hands. In most cases the attack is self-limited and resolves after rewarming the hands or feet.

In most patients, Raynaud's phenomenon is a primary syndrome and represents an exaggerated physiologic response to cold. Raynaud's phenomenon can also be secondary to collagen vascular disease such as systemic sclerosis or systemic lupus erythematosus. In severe secondary Raynaud's phenomenon, superficial ulceration or deep tissue necrosis with gangrene and amputation can occur. Primary Raynaud's phenomenon is more common in women than men, and occurs in approximately 5% of the U.S. population.[134] Vasoconstriction is triggered by cold temperatures of the ambient environment when the patient's body becomes cold, or by exposing the hands alone to cold, such as when handling cold objects. Raynaud's phenomenon can be exacerbated by the combination of cold and high altitude compared with cold exposure alone, which may be due to the higher sympathetic tone at high altitude.

There are several risks accompanying attacks of Raynaud's phenomenon in a wilderness environment. The intense vasoconstriction predisposes to cold injury, including frostbite and trench foot. The intense vasoconstriction can also make the hands nonfunctional for performing basic tasks essential for survival in a cold-weather environment, such as zipping up a coat or putting on crampons. A person with Raynaud's phenomenon must rely on the help of others when his or her hands become cold and incapacitated. This includes help with performing simple tasks such as adding layers of clothing, zippering outer garments, lacing boots, or rewarming hands.

Measures can be taken to minimize the risk of serious attacks of Raynaud's phenomenon. The location of a wilderness trip could be in a warmer climate, or could take place in the summer rather than winter. If winter activities are pursued, a location with a less severe winter environment can be chosen. For example, high-altitude mountaineering in South America is more equatorial and takes place in a warmer climate than mountaineering in North America, Asia, or Europe. Appropriate cold-weather gear and clothing are essential to keep the entire body warm and help mitigate attacks. High-quality plastic mountaineering boots or ski boots and expedition-type mittens or gloves with space for disposable chemical hand warmers help in winter environments while climbing or skiing. Adequate hydration and nutrition also help prevent attacks. Good physical condition before the climb reduces the chance of exhaustion, which would exacerbate Raynaud's phenomenon. It is especially important for a climber with Raynaud's phenomenon to use conservative judgment and retreat early, before becoming overextended and exhausted. Nicotine and other drugs with peripheral vasoconstrictive effects, such as over-the-counter decongestants, should be avoided.

Treatment of Raynaud's phenomenon is initially focused on behavioral modification. It is clear that a significant behavioral component contributes to attacks because in placebo-controlled trials of Raynaud's phenomenon, the placebo group usually significantly improves along with the active drug group. This implies that increased awareness of Raynaud's phenomenon and improving clothing insulation can reduce the frequency of attacks. Temperature-related biofeedback is used in combination with various relaxation techniques to treat Raynaud's phenomenon, with inconsistent results.[106] Behavioral modification, supplemented by pharmacologic therapy if necessary, is the mainstay of therapy for primary Raynaud's phenomenon. Secondary Raynaud's phenomenon often requires primary pharmacologic treatment.

Calcium channel blockers, specifically nifedipine, are the most widely used drugs for treatment of Raynaud's phenomenon. In one blinded, randomized clinical trial, sustained-release nifedipine treatment reduced attacks by 66% compared with placebo.[106] Only 15% of subjects discontinued therapy because of adverse effects of nifedipine. Extended-release nifedipine is favored because of the possible hypoperfusion of digits with short-acting nifedipine owing to a decrease in systolic blood pressure. Other oral drugs that have been used to treat Raynaud's phenomenon include the peripherally acting α_1-adrenergic blocker prazosin, although its efficacy may be transient; the angiotensin II receptor antagonist losartan; the

selective serotonin reuptake inhibitor fluoxetine;[134] and the vasodilator isoxsuprine.[132] *Ginkgo biloba* was effective in treating primary Raynaud's phenomenon in one study,[92] and topical therapy with nicotinates (hexyl nicotinate) is used to treat mild to moderate Raynaud's phenomenon.[12]

Osteoarthritis

Osteoarthritis is a major cause of disability in adults and most commonly affects the joints of the hands, hips, knees, and cervical and lumbar spine. Uncommonly affected joints include the shoulder, elbow, and wrist. Factors in the evolution of osteoarthritis include initiation in either previously injured or susceptible joints; development, which is biochemically mediated and biomechanically driven; and clinical expression, which may be modified by factors such as weight and sex.[27] The primary symptom of osteoarthritis is pain that is typically exacerbated by activity and relieved by rest. With more advanced disease, pain may occur with progressively less activity. Osteoarthritis can be inflammatory or noninflammatory. Patients with noninflammatory osteoarthritis complain primarily of joint pain and disability. Physical findings in affected joints include tenderness, bony prominence, and crepitus. Patients with inflammatory osteoarthritis complain of articular swelling, morning stiffness, and night pain. Signs of inflammation include joint effusion on examination or radiography, warmth on palpation of the joint, and synovitis on arthroscopic examination.

The degree of disability caused by hip or knee osteoarthritis is an important consideration for wilderness activities. Patients should be guided toward activities that are within their functional capability. Wilderness activities that cause increased weight bearing on lower extremity joints should be avoided. For example, hiking or trekking with a light pack or bicycling are recommended over activities such as a multiday backpacking trip or mountaineering where carrying heavy loads might be required. Risk of development of hip or knee osteoarthritis is also relevant to wilderness activities. There is increased risk of lower limb osteoarthritis associated with repetitive, high-impact sports and that risk is increased with joint injury.[27] When assessing risk for osteoarthritis from wilderness activities, the nature and intensity of the activity, presence of previous injury, and body mass index should all be taken into account. Recreational running does not appear to increase the risk of knee osteoarthritis,[27,72] but how this applies to trail running is not known. The evidence that obesity is strongly associated with development of knee, and probably hip, osteoarthritis and that weight loss improves joint pain and function[94] are relevant to wilderness activities where carrying heavy loads is required. Wilderness activities that repeatedly require carrying a heavy pack, or squatting and kneeling maneuvers, may cause progression of knee or hip osteoarthritis, especially if there has been previous injury to a joint or associated joint muscles, ligaments, or tendons.[118]

Very little information is available on development of hand arthritis with wilderness activities such as climbing. One study suggests that rock climbing at a high standard for over 10 years may increase risk of osteoarthritis in certain joints of the hands.[110]

Measures that can be taken to improve functional capacity for wilderness activities include nonpharmacologic and pharmacologic treatments. The goal of management of osteoarthritis is to control pain and improve function and health-related quality of life with avoidance of therapeutic toxicity. Potential treatments include exercise, biomechanical techniques, pharmacologic therapy, and surgery.

Exercise is the primary nonpharmacologic intervention for lower limb osteoarthritis that is directly related to wilderness activities. There is good evidence that strengthening and aerobic exercise can reduce pain and improve function and health status in patients with knee osteoarthritis; the evidence for hip osteoarthritis is not as compelling, but exercise is still recommended.[41,109] Well-conditioned muscle and muscular balance are needed to attenuate impact loads and provide joint stability. Muscular conditioning may prevent exercise-related osteoarthritis.[118] Muscular conditioning is achieved through well-designed exercise programs performed with supervision or as home exercise routines that include range-of-motion and flexibility exercise, muscle conditioning, and aerobic cardiovascular exercise.[43,130]

Biomechanical treatments for knee osteoarthritis are relevant to wilderness activities such as hiking and trekking and are helpful at reducing symptoms. For appropriate application, consultation with a physiatrist or sports medicine physician may be required. Shock-absorbing footwear reduces impact loading, heel wedges reduce loading of the medial knee joint surface, neoprene support sleeves increase proprioception and reduce feelings of instability, dynamic bracing controls lateral instability, and taping allows repositioning of the patella.[43] Hiking poles are an additional method to help unload lower extremity joints while hiking or trekking, and may be helpful for patients with osteoarthritis of the hip and knee, especially during downhill walking.[66,113]

The major pharmacologic treatments for osteoarthritis include analgesics, nonsteroidal anti-inflammatory drugs (NSAIDs), and intra-articular corticosteroids. The major goal of treatment with these agents is relief of pain, which usually is achieved with nonopioid analgesics. The nonprescription analgesic acetaminophen at doses up to 4 g/day is the recommended primary treatment.[13,135] Patients with osteoarthritis who have mild to moderate pain will obtain a similar degree of pain relief with acetaminophen as with NSAIDs.[43] Although it is one of the safest analgesics, acetaminophen can prolong the half-life of warfarin and can cause hepatic toxicity at therapeutic doses in patients with chronic alcohol abuse.

For patients who do not obtain adequate symptom relief with nonopioid analgesics, self-limited use of nonselective NSAIDs is an alternative after considering the risk of upper gastrointestinal and renal toxicity. Cyclooxygenase-2–selective NSAIDs are an alternative with less potential risk of gastrointestinal toxicity, but with an increased risk of serious cardiovascular events. The risk of adverse cardiovascular events may also apply to the nonselective NSAIDs, and they are recommended only for short-term use in the relief of pain[11] (updated information is available at the FDA website, www.fda.gov/cder/index.html).

An alternative to NSAIDs for osteoarthritis pain not relieved by acetaminophen is the centrally acting oral analgesic tramadol. It is a synthetic opioid agonist that inhibits reuptake of norepinephrine and serotonin and has been approved by the FDA for treatment of moderate to severe pain. The efficacy of tramadol has been found to be comparable with that of ibuprofen in patients with hip and knee osteoarthritis.[43]

Another alternative therapy for osteoarthritis is glucosamine and chondroitin. These are compounds extracted from animal products that are absorbed by the gastrointestinal tract and

appear to be capable of increasing proteoglycan synthesis in articular cartilage. One meta-analysis concluded that, despite methodologic flaws in many studies, these compounds are probably efficacious for treatment of osteoarthritis and have no significant side effects.[87] Relevant to wilderness activities, glucosamine and chondroitin were effective in relieving symptoms of knee osteoarthritis in active members of the U.S. Navy diving and special warfare community.[77]

In persons with osteoarthritis of the hand or knee who have mild to moderate pain, use of topical analgesics, such as capsaicin cream, is appropriate as adjunctive treatment or monotherapy.

Evidence supports short-term (up to 2 weeks) improvement in symptoms of osteoarthritis of the knee after intra-articular corticosteroid injection. Significant improvement may also occur for up to 16 to 24 weeks with a dose equivalent to 50 mg of prednisone.[4] There is concern that multiple injections of intra-articular corticosteroids may promote disease progression; further study is required. Corticosteroid injection for knee osteoarthritis to provide benefit for the duration of a wilderness trip for up to 2 weeks seems reasonable,[81] and may provide enough pain relief and increase in function to make wilderness activity safer and more enjoyable.

Surgical treatment of osteoarthritis is usually considered after failure of nonsurgical treatments. Four categories of surgical treatment are available: osteotomy, arthroscopy, arthrodesis, and arthroplasty. Osteotomies are performed in persons with early osteoarthritis and may relieve symptoms and slow the rate of progression. Arthroscopic debridement and lavage can also successfully alleviate symptoms, particularly in the case of degenerative meniscal tears in the presence of mechanical symptoms. When there is substantial joint space narrowing, however, arthroscopic surgery has limited benefit. Arthrodesis, or joint fusion, successfully alleviates pain and is commonly performed in the spine and in small joints of the carpus, hand, and foot. Arthrodesis of the major proximal joints of the upper and lower extremities is not well tolerated because of the functional deficits associated with loss of motion.

Total joint arthroplasty represents the most significant advancement in the treatment of osteoarthritis in the past century. It is the mainstay of surgical treatment for advanced osteoarthritis of the hip, knee, and glenohumeral joints. For older persons, total joint replacement is a highly successful procedure that will probably last for the duration of their lives. The pain and disability of end-stage osteoarthritis can be eliminated, restoring patients to near-normal function. However, a total joint replacement may not be sufficiently durable in persons with life expectancies exceeding 20 years and those who wish to participate in high-demand activities.

After total joint arthroplasty, patients should be encouraged to remain physically active for general health and also for the quality of their bone. There is evidence that increased bone quality improves prosthesis fixation and decreases the incidence of early loosening. To recommend a certain activity after total knee or hip replacement, factors such as wear, joint load, intensity, and the type of prosthesis must be taken into account for each patient and sport. Because load will influence the amount of wear exponentially, only activities with low joint loads such as swimming, cycling, or walking should be performed regularly for exercise. If an activity is performed intermittently for recreation, then activities with higher joint loads such as skiing or hiking may be acceptable. It is unwise to start technically demanding wilderness activities after total joint replacement because the joint loads and the risk for injuries are generally higher for these activities in unskilled individuals. Activity recommendations differ after total knee and total hip replacement. During activities such as hiking or jogging, high joint loads occur between 40 and 60 degrees of knee flexion, where many knee designs are not conforming and high stress will occur. It is prudent to be more conservative after total knee arthroplasty than after total hip arthroplasty for activities that exhibit high joint loads in knee flexion. After total knee replacement, patients should alternate activities such as walking and cycling. For mountain hiking, patients are advised to avoid descents or at least use hiking poles. Jogging or sports involving running should be discouraged after total knee replacement.[69,70]

Hematology

Anemia

The most common hematologic condition encountered is anemia. Although specific anemias have certain concerns discussed in the following sections, the general effect of mild to moderate anemia is a reduction in exercise capacity. In general, for every 1% fall in hematocrit, maximum oxygen consumption ($\dot{V}O_2max$) decreases 1% and endurance decreases by 2%.[18,37,50,129] Thus, anemic patients should be counseled that their ability to perform strenuous exercise will be reduced compared with traveling companions; they may not be able to keep the same pace or hike as far.

Thalassemia Trait. Thalassemia trait is the most common inherited hemoglobin disorder and can be encountered in patients from diverse ethnic origins. β-Thalassemia trait is autosomal dominant and results in a mild microcytic anemia (mean corpuscular volume [MCV] approximately 60 to 70 femtoliters [fL]) and hematocrit in the 30% range. It is diagnosed by hemoglobin electrophoresis showing elevated hemoglobin A_2 in the presence of normal iron stores. The genetics of α-thalassemia trait are more complex, with the most common forms being autosomal dominant; the condition is electrophoretically silent. α-Thalassemia trait leads to microcytosis (MCV approximately 70 fL) with hematocrit at the lower end of the normal range. A severe recessive variant, hemoglobin H disease, predominantly affects people of southeast Asian origin. This type of α-thalassemia results in a hemolytic anemia with a hematocrit of 25% to 35%.

Presence of either α- or β-thalassemia trait does not lead to any specific problems except for anemia. Diagnosis is important to avoid inappropriate therapy with iron and provide genetic counseling for families.

Iron Deficiency. Iron deficiency is very common, affecting up to 40% of women and 1% to 5% of men. In athletes, especially runners, the incidence is increased to 50% to 80% of women and 10% to 17% of men.[93,111] This increase in iron deficiency may reflect iron loss through gastrointestinal bleeding[20,22,88] or urinary iron loss through hemolysis.[20,56] Iron deficiency has multiple impacts on exercise ability. As noted previously, a lower hematocrit decreases exercise ability. However, iron deficiency also has a negative effect on exercise beyond a decrement in hematocrit. Studies have shown that repletion of iron improves $\dot{V}O_2max$, exercise endurance, and strength, suggesting that lack of tissue stores of iron is detrimental.[15–17,47,111] Iron deficiency

has also been shown to impair cold tolerance, perhaps because of alteration in thyroid hormone metabolism.[7,14]

Going to altitude puts additional stresses on body iron stores. Although the initial increase in hematocrit at altitude is due to contraction of plasma volume, red cell production increases several days later. Studies have shown that after as little as 1 week at altitude, serum ferritin falls as iron is consumed to make more red cells.[8,57,107] For example, Berglund and colleagues[8] showed that after 10 days of breathing 14% oxygen, a 10% increase in hemoglobin was associated with a 46% decrease in ferritin.

Although serum ferritin is the best test for iron deficiency, the range of "normal" listed on the laboratory report may not be the most appropriate level for athletes. Improvement can be seen in exercise ability and fatigue with ferritin above 50 ng/dL.[131] Climbers with low measured ferritin may not be able to mount an adequate hematocrit response to altitude and may have impaired exercise ability. In one study, climbers with ferritin measured above 50 to 100 ng/dL were the ones who performed best.[107] In theory, 250 mg of storage iron are required for every 1 g/dL increase in hemoglobin; that amount of iron is equivalent to 25 to 32 ng/dL of serum ferritin. It has been suggested that an "ideal" hemoglobin for altitude should be 2.5 g/dL higher than the usual range; increasing the hemoglobin by that amount would require a serum ferritin of 62 to 80 ng/dL.[8]

Iron replacement therapy should be prescribed for any person with ferritin under 50 ng/dL who is planning a high- or extreme-performance expedition, or if the ferritin is under 100 mg/dL in a patient planning a prolonged trip to altitude. The best method of iron replacement is still controversial. One approach is to start with 1 pill that contains at least 60 mg of elemental iron every day, and if that is tolerated after 1 week, moving up to 2 pills per day. Taking the pills with vitamin C can aid absorption. The subject should avoid certain foods, such as fiber and tea, within several hours of iron ingestion. People who cannot tolerate or absorb oral iron can have intravenous iron therapy. Currently, either iron sucrose or iron glucose can be used intravenously; these preparations are safer than iron dextran.[44]

Hemolytic Anemias. Many patients have well-compensated congenital hemolytic anemias. The laboratory findings are mild to moderate anemia with elevated reticulocyte count, indirect bilirubin, and lactate dehydrogenase. Red cell membrane defects, such as hereditary spherocytosis, are the most common causes of inherited hemolytic anemia.

Any patient with hemolysis is prone to folate deficiency, so adequate intake (400 to 800 µg/day) should be part of nutrition planning. Patients with hemolytic anemia may have increased hemolysis with fevers. The incidence of gallstones is also increased, and consideration should be given to obtaining screening ultrasonography before prolonged expeditions away from medical care.

Glucose-6-Phosphate Dehydrogenase Deficiency. Glucose-6-phosphate dehydrogenase (G6PD) deficiency is an important cause of hereditary hemolytic syndromes with wilderness implications.[9] G6PD deficiency is sex linked and thus most commonly affects men. The defect is in the hexose monophosphate shunt and renders the red cell unable to withstand oxidative stress. Most people with this disease have hemolysis only with such stressors as infections and intake of oxidative drugs. There are two main subtypes—African and Mediterranean. In the African type of G6PD deficiency, the enzyme is unstable and older cells have diminished activity. Therefore, when these patients have hemolysis, it is self-limited because as the reticulocyte count increases, G6PD activity returns to normal. The Mediterranean type is caused by a defective enzyme and tends to be more severe, because with oxidative stress the G6PD activity does not increase as the reticulocyte count increases, and fatal hemolysis may result. G6PD deficiency is of concern because many drugs used in travel medicine can cause sudden and severe hemolysis. A list of such drugs is given in Box 29-1. Many patients are unaware that they are G6PD deficient; their first symptom may be fulminant hemolysis with antimalarial drugs. In the field, acute hemolysis presents as back pain and dark urine. Management consists of stopping the suspect drug and hydrating the patient. Ingestion of fava beans (an oxidative stress) in patients with the Mediterranean type of G6PD deficiency may cause severe, potentially fatal hemolysis.

Box 29-1. Drugs That May Precipitate a Hemolytic Crisis with G6PD Deficiency

- Acetanilide
- Dapsone
- Furazolidone
- Isobutyl nitrate
- Methylene blue
- Nalidixic acid
- Naphthalene
- Nitrofurantoin
- Pamaquine
- Phenazopyridine
- Phenylhydrazine
- Primaquine
- Quinolones
- Sulfacetamide
- Sulfamethoxazole
- Sulfanilamide
- Sulfapyridine

Sickle Cell Anemia and Trait. Eight percent of African Americans and 0.08% to 0.5% of whites have sickle cell trait.[45,84,117] It is mostly clinically silent, although there is a potential for problems in the wilderness. Sickling can occur in patients with the trait under moderately hypoxic conditions at altitudes of greater than approximately 1830 m (6000 feet). Multiple case reports describe splenic crisis occurring at altitude or during airplane travel.[45,84] Patients present with acute onset of severe left upper quadrant pain that may have a pleuritic component, nausea, and vomiting. Therapy is descent, oxygen, and pain control. In rare cases of large splenic infarction, splenectomy may be required.

Patients with sickle trait are more prone to dehydration because of a renal concentration defect. Maintenance of adequate fluid intake is very important. Because patients cannot maximally concentrate their urine, the color of the urine cannot be used as a guide to hydration. If diarrhea or vomiting develops in patients with sickle trait, close attention should be paid to hydration status.

Concern about the risk of "sudden death" in people with sickle cell trait with strenuous exercise is valid. Data collected in the 1960s from army recruits in boot camp showed a 28-fold increased risk of death (absolute risk, 1:3200).[116] Review of these cases revealed that most patients had heatstroke, with resulting severe rhabdomyolysis causing death. Practical advice

TABLE 29-5. Guidelines for Factor Replacement

SITE OF BLEED	DESIRED PLASMA FACTOR LEVEL	HEMOPHILIA A: DOSE OF FACTOR VIII CONCENTRATE	HEMOPHILIA B: DOSE OF FACTOR IX CONCENTRATE
Joint	80% acutely, then 40% every other day until resolved	40 U/kg initially, then 20 U/kg every other day until healed	80 U/kg initially, then 40 U/kg every other day or third day as needed
Muscle	40%–50%	20–40 U/kg per day until healed	40–60 U/kg, then 20–30 every other day as needed
Oral	100%*	50 U/kg*	100 U/kg*
Nose	Initially 80%–100%, then 30% until healing	40–50 U/kg, then 30–40 U/day	80–100 U/kg, then 35–40 U/day
Gastrointestinal	Initially 100%, then 50% until healing	50 U/kg, then 30–40 U/kg per day	100 U/kg, then 30–40 U/day
Genitourinary	Initially 100%, then 30% until healing	50 U/kg, then 30–40 U/kg per day	100 U/kg, then 30–40 U/day
Central nervous system	Initially 100%, then 50%–100% for 14 days	50 U/kg, then 25 U/kg every 12 hr	100 U/kg, then 50 U/kg every day
Surgery/trauma	Initially 100%, then 80%–100% until wound healing begins, then 30% until suture removed.	50 U/kg, then 40–50 units every 12 hr, adjusted according to healing	100 U/kg, then 50 U/day, adjusted according to healing

*Antifibrinolytic agents are useful for oral bleeding.
Note: For severe or persistent minor bleeding, factor levels should be followed.
Modified from DiMichele D, Neufeld EJ: Hematol Oncol Clin North Am 12:1315–1344, 1998, with permission.

for patients with sickle cell trait is to be cautious about dehydration and avoid strenuous exercise in the heat, especially if they are deconditioned.

Patients with sickle cell anemia often have end-organ damage, which may complicate wilderness travel. Older patients may have chronic pain syndromes due to multiple bony infarcts. The leading cause of overall mortality in adults is complications of pulmonary hypertension, which result in hypoxia and impaired lung function. Pain crises are unpredictable but can be provoked by heat extremes, dehydration, or hypoxia—all features found in wilderness travel.

Patients with sickle cell anemia who are going on limited trips to the wilderness need to be reminded about the importance of adequate fluid intake and to bring sufficient pain medicine to manage both chronic pain and any crisis. When considering longer-term or adventure travel, patients should be screened for pulmonary hypertension. Patients should be reminded to be compliant with folic acid supplementation. Because most adults with sickle cell anemia are functionally asplenic, they should take the same precautions as outlined later for asplenic patients.

Hemophilia

Deficiency of factor VIII or IX occurs in 1 of 10,000 males. Patients with severe hemophilia are at risk for severe bleeding even with minor trauma. Bleeding most often occurs in muscles and joints, but intracranial hemorrhage is the leading cause of fatal hemorrhage. Patients can have severe arthritis due to repeated bleeding. A rough rule is that patients with less than 1% of normal levels of factor VIII or IX can have spontaneous bleeding, those with 1% to 15% of normal levels may have bleeding with minor trauma, and those with greater than 15% of normal factor levels may have bleeding with major trauma.

Factor concentrates to correct the coagulation defects are factor VIII for hemophilia A and factor IX for hemophilia B. A suggested dosing scheme is given in Table 29-5. The World Hemophilia Foundation website is an invaluable guide to resources available for travelers (www.wfh.org).

Box 29-2. Factors That Do Not Require Refrigeration

Factor VIII	Alphanate
Recombinant	Factor IX
ReFacto	BeneFix
Hemophil M	Mononine
Monarci-M	Alphanine
Koate DVI	Profilnine SD

Hemophilia does not preclude travel or expedition travel. However, patients with hemophilia who are planning travel should take factor replacement with them, especially if they will be away from health care facilities. Only certain factors listed in Box 29-2 can be stored without refrigeration. The patient should also have the supplies necessary to inject the factor, such as alcohol wipes and needles. At least one member of the traveling party should also be trained to infuse factor in case the patient is incapacitated.

The biggest functional limitation to the patient with hemophilia is joint disease. Arthritis due to joint bleeds is very common and can be disabling. Joint bleeds can occur with minor injuries or ankle twists. Before travel, patients should work to strengthen their muscles to provide joint protection. Simple measures to prevent ankle sprains, such as wearing high-top boots, should also be used. Damaged joints should be splinted to prevent reinjury.

von Willebrand's Disease

The most common inherited bleeding disorder is von Willebrand's disease, which may affect up to 1 of 1000 people.

It is characterized by easy bruising and mucocutaneous bleeding. Joint bleeding is unusual for most patients. Patients with von Willebrand's disease can have significant bleeding with trauma. There are multiple types of von Willebrand's disease, but the 80% of patients with type 1 respond to desmopressin, which is available as a nasal spray (Stimate; ZLB Behring, King of Prussia, PA). The dose of Stimate is one squirt in each nostril, with the effect lasting up to 24 hours. The physician needs to be specific when prescribing Stimate because generic nasal desmopressin that is used for enuresis has too little desmopressin per dose to be effective for bleeding disorders. Estrogen can also raise levels of von Willebrand factor; the use of oral contraceptives can normalize levels in mildly affected women. The patient with rarer types of von Willebrand's disease may require specific replacement therapy with Humate-P, a factor concentrate that contains von Willebrand factor. Unfortunately, this concentrate must be refrigerated.

Thrombocytopenia

Immune thrombocytopenia is the most common autoimmune hematologic disease; it occurs in 1 in 50,000 people. Patients with modest (>30,000 platelets/μL) thrombocytopenia are at no greater risk for bleeding except with extreme trauma, and no specific precautions are needed. Patients with chronic immune thrombocytopenia and stable platelet counts can be cleared for wilderness travel. For the patient with a history of recurrent severe thrombocytopenia who is planning prolonged travel, the onset of petechiae and bleeding can be treated with a pulse dose of dexamethasone (40 mg orally × 4 days).

There are multiple causes of congenital thrombocytopenia and platelet function defects. Some patients have mild thrombocytopenia with no bleeding defects, whereas others may have both platelet number and function defects. One such group of patients has mildly decreased platelets (75,000 to 150,000/μL) but no bleeding defects. These patients require no special precautions. Most patients with congenital platelet defects have a mild bleeding diathesis. Many of these patients respond to desmopressin and can carry the nasal form (Stimate) with them.

Anticoagulation

The indications for chronic anticoagulation are increasing, especially because of recommendations for lifelong anticoagulation for idiopathic and recurrent deep venous thrombosis. The patient on warfarin and other oral anticoagulants poses special challenges. Fluctuating amounts of vitamin K in the diet, increased exertion, and potential travel-related illness can dramatically alter the level of anticoagulation. Unless the patient has access to a point-of-care international normalized ratio (INR) monitor, there may be no way to monitor the level of anticoagulation.

Patients with a stable INR can safely go several weeks without an INR check. However, this assumes that the diet is the same as their regular diet and there are no concurrent illnesses. For patients who plan to travel longer or for those who will be away from health care, the use of point-of-care INR monitors should be strongly considered. The patient should be very familiar with the machine's operation and be able (or have a nomogram) to adjust warfarin dose. A suggested nomogram is given in Table 29-6. Vitamin K tablets should also be carried to allow treatment of very high INR. Another option for long-term therapy is to consider the use of low–molecular-weight (LMW) heparin injections. Dosing is weight based and does not need monitoring. The tradeoff is the extra space required to pack the syringes and inconvenience of proper needle disposal. For the patient who has an absolute requirement for stable anticoagulation, the use of LMW heparin is the safest alternative.

TABLE 29-6. Maintenance Warfarin Adjustment Nomogram

INTERNATIONAL NORMALIZED RATIO (INR)	DOSE CHANGE
1.1–1.4	Day 1: Add 10–20% TWD Weekly: Increase TWD by 10–20% Return: 1 Week
1.5–1.9	Day 1: Add 5–10% of TWD Weekly: Increase TWD by 5–10% Return: 2 Weeks
2.0–3.0	No change Return: 4 wk
3.1–3.9	Day 1: subtract 5%–10% TWD Weekly: reduce TWD by 10%–20% Return: 2 wk
4.0–5.0	Day 1: no warfarin Weekly: reduce TWD by 10%–20% Return: 1 wk
>5.0	Stop warfarin until INR <3.0 Decrease TWD by 20%–50% Return daily

TWD, total weekly dose.

From DeLoughery TG: Oral anticoagulants. In Goodnight SH, Hathaway WE (eds): Disorders of Hemostasis and Thrombosis: A Clinical Guide. New York, McGraw-Hill Professional, 2001, pp 533–566, with permission.

Travel Thrombosis

Airplane travel has been considered a risk factor for DVT for over 40 years, but has only recently been rigorously studied. Case-controlled studies suggest a relative risk of thrombosis of three- to fourfold with prolonged (>4 to 6 hours) travel, with a higher risk for travel times over 8 hours.[1,64,124] The absolute risk for thrombosis is uncertain. For example, the overall risk for symptomatic pulmonary embolism is estimated to be 0.4 per million passengers, rising to 4 per million in the highest-risk group.[73,103] In contrast, small prospective trials showed a calf vein thrombosis rate of 2% to 10%.[114,115] The presence of risk factors, such as history of deep venous thrombosis or underlying hypercoagulable state, is important. Up to 70% to 90% of persons with thrombosis had other risk factors for thrombosis.[62,85]

The pathogenesis of "traveler's thrombosis" is controversial. Venous stasis appears to be the primary risk factor. Prolonged sitting is a risk factor for thrombosis, perhaps through increased venous stasis. Although mild hypoxia due to the low cabin air pressure is often blamed in the popular press, there are no data showing activation of coagulation with mild hypoxic exposure.[31] Preexisting risk factors for thrombosis are also important. As noted previously, most studies indicate that people who experience travel-related thrombosis have other risk factors, such as history of thrombosis or estrogen use. The combination of thrombotic risk factors and long travel duration raises the risk of thrombosis by 16-fold.[85]

The best method of prophylaxis is controversial. Ideally, people should try to be up and exercising their legs at least once an hour. However, given the crowded cabins and today's secu-

Box 29-3. Risk Factors for Traveler's Thrombosis

Age > 65 years
Cancer
Estrogen use
History of lower extremity deep venous thrombosis
Hypercoagulable state
Lower leg cast
Obesity (>2× ideal body weight)
Recent surgery (previous 6 weeks)

rity climate, this is unrealistic. Aspirin remains a popular recommendation but has been demonstrated in a randomized trial not to be effective for preventing traveler's thrombosis.[21,49] Elastic stockings provided protection in trials, but have the side effects of discomfort and superficial thrombosis.[115] A single prophylactic dose of LMW heparin is effective, but is inconvenient for most people.

A reasonable approach is first to assess a patient's risk of thrombosis when traveling by plane for over 6 hours (Box 29-3). For most low-risk people, one could encourage foot movement and avoiding dehydration. For medium- and high-risk patients, stockings should be recommended. The feasibility of adding LMW heparin on a case-by-case basis for high-risk patients also should be considered.

The Asplenic Patient

Asplenic patients pose unique travel risks. Asplenic patients are at risk for overwhelming infections from a diverse group of infectious agents. The risk of overwhelming sepsis varies by indications for splenectomy, but the lifetime risk appears to be approximately 1%.[10,71] The use of pneumococcal vaccine and recognition of this syndrome have helped lessen the risk, but travel can expose the asplenic person to novel infectious organisms.

The classic bacterial pathogen is the pneumococcus, but in older patients gram-negative bacteria are a common cause of infections.[36] Unusual organisms causing overwhelming infections, such as *Capnocytophaga* from dog bites or *Babesia* from ticks, have been reported.[32,133]

Asplenic patients need to be counseled about the risk of infections and should be vaccinated for pneumococcus, meningococcus, and *Haemophilus influenzae*.[53,91] Patients previously vaccinated for pneumococcus should be revaccinated every 3 to 5 years.[71] In addition, because of concerns about greater severity of malarial infection, they should be scrupulous with antimalarial prophylaxis.[83,96] The role of prophylactic antibiotics is controversial for adults, but patients younger than 18 years of age should always take penicillin VK 250 mg twice daily, especially when traveling. Patients should be advised to start antibiotics and seek medical care if they develop a fever, shaking chills, or lower respiratory track infection. A reasonable oral antibiotic for self-medicating is amoxicillin/clavulanic acid 875/125 mg twice a day. Patients who are bitten by dogs should also take antibiotics. Asplenic patients should wear an identification bracelet to alert health care providers.[40]

Oncology

The numbers of cancer survivors is increasing, and many of these patients pursue wilderness activities. Chemotherapy can lead to short-term side effects such as nausea and neutropenia, but many side effects can last months or years after chemotherapy has ended.

Box 29-4. Chemotherapy Agents: Long-Term Side Effects

CARDIAC DYSFUNCTION
Anthracyclines (doxorubicin, daunorubicin, epirubicin, mitoxantrone, idarubicin)*

PULMONARY TOXICITY
Bleomycin*
Nitrosoureas*
Cyclophosphamide
Methotrexate

NEUROPATHY
Cisplatin*
Oxaliplatin*
Vincristine*
Taxol*

RAYNAUD'S PHENOMENON
Bleomycin
Cisplatin

*Common.

Chemotherapy

Common side effects of most chemotherapeutic agents are nausea and bone marrow suppression. The nausea is short term and managed with 5-hydroxytryptamine ($5\text{-}HT_3$) blockers such as ondansetron 8 mg orally (PO) or granisetron 1 mg PO. The highest-risk time for neutropenia is usually 10 to 21 days after chemotherapy. Patients may want to hike or go camping in between chemotherapy sessions. Neutropenic patients can be cleared for this type of activity as long as they can seek medical care if they become febrile. A good rule of thumb is that patients should not be more than 2 hours away from an emergency department. The vast majority of infections are due to endogenous organisms and not from the environment, but some precautions include wearing a mask when in crowded areas and avoiding ingestion of fresh fruits or vegetables.

Thrombocytopenia is rare with most chemotherapy regimens. Platelet transfusion should be performed only for platelet counts under 10,000/μL.

Chemotherapy can cause a variety of long-term side effects (Box 29-4). Anthracyclines such as Adriamycin can lead to cardiac damage that may be well compensated until another stressor, such as altitude, is added. The risk for chronic heart failure is highest with doses over 300 mg/m^2, but can be seen with any dose. Agents such as vincristine can lead to neuropathy that causes loss of dexterity.

Of particular concern is bleomycin, an antineoplastic agent that is part of a curative regimen for both germ cell tumors and Hodgkin's disease. Bleomycin can lead to subtle lung damage that results in loss of diffusing capacity. For very competitive athletes with germ cell tumors, effective non–bleomycin-containing regimens can be used.[97] Bleomycin can also interact with high concentrations of oxygen, causing a fulminant and fatal lung toxicity. Fatal cases have been reported many months after cessation of therapy. Controversy remains as to whether

TABLE 29-7. Post–Bone Marrow Transplantation Vaccine Recommendations

VACCINE	ALLOGENEIC TRANSPLANT	AUTOLOGOUS TRANSPLANT	TIMING	LIVE?
Tetanus toxoid	Recommended	Recommended	6–12 mo	
Diphtheria	Recommended	Recommended	6–12 mo	
Inactivated polio virus	Recommended	Recommended	6–12 mo	
Pneumococcal	Recommended	Recommended	6–12 mo	
H. influenzae	Recommended	Recommended		
Influenza	Recommended	Recommended		
Measles	No earlier than 24 mo			Yes
Rubella	No earlier than 24 mo			Yes
Hepatitis B			6–12 mo	
Hepatitis A			6–12 mo	
Inactivated polio			6–12 mo	
Oral polio	No earlier than 24 mo			Yes
MMR	No earlier than 24 mo			Yes
Typhoid (tY21a)	No earlier than 24 mo			Yes
Varicella	No earlier than 24 mo			Yes
Yellow fever	No earlier than 24 mo			Yes
Japanese encephalitis V			6–12 mo	
Meningococcal vaccine			6–12 mo	

Information from references 65, 79, 82, 117, 119.

patients who have had bleomycin can safely go scuba diving because of the high pressures of oxygen in tank mixtures. For example, during a dive to 20 m (66 feet), the partial pressure of inhaled oxygen is 0.63 atm; at 30 m (98 feet), it is 0.84 atm.[63] Patients who have received bleomycin should wear an identification bracelet in the event that emergency surgery should be required, so that the lowest possible oxygen concentration may be used.

The cancer survivor who plans to travel on a high-altitude or extreme-performance expedition should undergo careful screening. Patients who have received anthracyclines should have cardiac function screened; pulmonary function testing should be done in patients who have received bleomycin or other potential pulmonary toxic agents. Any patient who has received upper chest or neck radiation should have thyroid function screened.

Certain agents used for hematologic malignancies, especially chronic lymphocytic leukemia, can lead to profound and prolonged T-cell immunosuppression that persists months to years afterward and puts these patients at risk for unusual infections. These agents are fludarabine, cladribine, and alemtuzumab. The degrees of immunosuppression can be monitored by measuring CD4 counts. Patients with counts under 200/μL are severely immunocompromised and travel is not recommended.[23,112] Patients with higher CD4 counts still may have residual immunosuppression and be prone to herpes zoster outbreaks for years after chemotherapy.

The Stem Cell ("Bone Marrow") Transplant Recipient

Stem cell transplants are increasingly common therapy. Currently, three different types of stem cell transplantations are performed. Autologous transplants involve harvesting and preserving the patient's stem cells and then administering radiation or chemotherapy (conditioning) to ablate the marrow. This is followed by reinfusion of the stored stem cells. Allogeneic transplantation involves infusion of another person's marrow. Increasingly, nonmyeloablative allogeneic transplants ("mini-transplants") are being used that involve only modest doses of chemotherapy and radiation to prepare the patient for transplantation. The major long-term complication of allogeneic transplantation is graft-versus-host disease (GVHD) due to the donor immune system attacking the recipient. Chronic GVHD can be a debilitating problem leading to chronic skin disease, restrictive pulmonary disease, and increased propensity for infection.

When counseling the stem cell transplant recipient about travel, the physician needs to know what type of transplant the patient has received, time after transplantation, and whether he or she has GVHD. In general, a patient who has received an autologous transplant and has recovered his or her blood counts can be cleared for travel 6 months after transplantation. For the patient who has received an allogeneic transplant, the main concerns are restoration of immunity and presence of GVHD. A reasonable rule of thumb is that patients will be immunosuppressed for 12 to 24 months after they stop their immunosuppressants and should always be treated as functionally asplenic. The transplant (especially allogeneic) recipient who is considering a high- or extreme-performance trip should undergo pulmonary and cardiac screening.

Timing of travel immunizations for transplant recipients is an uncertain area.[35] If given too soon, the immature immune system will not mount a response. Live vaccines are also a concern if immune function is not normal. An approach is given in Table 29-7.

Patients with stem cell transplants who remain on immunosuppression have special concerns. Their family members and close contacts also should not receive live vaccines for fear of disease transmission to these patients. New medications can interfere with immunosuppression. For example, erythromycin can raise cyclosporin levels to the toxic range, whereas rifampin can lower cyclosporin levels.

The references for this chapter can be found on the accompanying DVD-ROM.

Mental Health in the Wilderness

30

Barry Morenz

Remote wilderness environments can have a salutary effect on a person's overall emotional health. Wilderness settings are sometimes used as intensive treatment settings for adolescents with emotional and behavioral problems.[8] Those who choose to pursue wilderness activities are often seeking relief from the stress of demanding and hectic urban lives. Traffic, phones, computers, deadlines, and endless chores can make the routine of daily life in an urban environment an objectionable ordeal. However, remote wilderness places can present unique stresses of their own in addition to being a refreshing change from the psychological stresses of urban life. Mental health and mental health problems might improve in the wilderness, but they can also become worse. New psychiatric problems can emerge in response to the demands of wilderness experiences. This chapter discusses the diagnosis and management (including the use of psychotropic medications) of emotional problems in the wilderness, both in people who have preexisting psychological difficulties and in those who develop new mental health problems in the wilderness.

The *Diagnostic and Statistical Manual of Mental Disorders, 4th edition, Text Revision*[1] (DSM-IV-TR) is the most recent, exhaustive, and widely used compendium of psychiatric disorders. However, most mental health problems can be thought of as falling into six broad disorders: anxiety, mood, psychotic, organic mental, personality, and substance abuse. Specific diagnostic assessment using the DSM-IV-TR and psychological testing in a wilderness setting is not crucial or practical. However, appropriate triage and management in a wilderness setting depends on being able at least to classify emotional and behavioral problems as falling into one or more of these broad categories. The first stage of diagnosis in the wilderness setting is to separate the normal from the abnormal, a task that may not be simple in many cases. It is not straightforward to differentiate what may be considered a normal adaptive response to the challenge of a wilderness experience from responses that are maladaptive and may signal the onset or exacerbation of a preexisting psychiatric problem. For example, at the end of a physically challenging day of backpacking with a group, one member of the group may break down in tears. Such crying may be a healthy and adaptive response. The process of crying might relieve stress and result in the member receiving needed emotional support. Alternatively, such tears may suggest the person feels mentally and physically overwhelmed. The person might be approaching a panic state, and if appropriate interventions are not undertaken the person's mental state will deteriorate. The challenge is to determine whether a pat on the back, a hug, and some encouraging words are all that is needed, or if the individual has to be assisted out of the wilderness as soon as possible to prevent emotional decompensation or prevent the person from becoming unsafe for the group or themselves.

In the following sections, five important topical areas for the assessment and management of emotional problems in the wilderness are discussed: (1) the common characteristics of the six major categories of psychiatric disorders most likely to be seen in the wilderness; (2) suicide and violence potential; (3) common complaints, such as insomnia, fatigue, headache, and other somatic complaints; (4) response to crisis (disasters and survival psychology); and (5) leadership and group process.

PSYCHIATRIC DISORDERS

Anxiety Disorders

Anxiety is a human emotion that is often experienced and is usually a normal and adaptive response to everyday life. Anxiety is an emotion that motivates us in myriad ways—for example, to complete tasks, study for tests, have a reliable belay partner when rock climbing, and work to earn a living. The experience of anxiety is physical as well as psychological. Increased heart rate, blood pressure, and respirations, in addition to sweaty palms and muscular tension, are common physical components of anxiety that are present along with a feeling of psychological tension. Rarely is anyone totally free of anxiety, but for most people anxiety is mild. There are many ritualized, socially condoned means of diminishing this everyday form of anxiety, including activities such as jogging, athletics, hot tubbing, drinking alcohol, and sexual activity.

In some circumstances, an extreme form of normal anxiety is triggered in normal people and is sometimes referred to as "the fight-or-flight response." This response occurs automatically without conscious control in some forms of competition and in response to immediate danger. Athletes, extreme sports enthusiasts, military combatants, crime victims, or people in any context confronting potential serious injury or death will have their fight-or-flight system triggered. This system is mediated through the autonomic nervous system and is a survival adaptation. When our autonomic nervous system is triggered, a variety of physical and mental changes take place. Blood is diverted from internal organs to the skeletal musculature, and heart rate, blood pressure, and respirations dramatically increase. A person in a fight-or-flight state often acts with little or no reflective thinking to preserve his or her life or the lives of others. Memories may be unreliable during these episodes because an individual is often on "automatic pilot." These

episodes of autonomic arousal in response to danger or competition are normal. However, some people develop poorly modulated anxiety that interferes with their ability to enjoy life, complete everyday tasks, and respond appropriately to danger. As anxiety begins to interfere with an individual's functioning, then the anxiety is more likely to be pathologic and might reflect an anxiety disorder.

There are several types of anxiety disorders, including generalized anxiety disorder, specific phobias, panic disorder, obsessive-compulsive disorder, and acute and post-traumatic stress disorder (discussed in the section on Response to Crisis, later). The essential feature of each is troublesome manifestations of anxiety. People with generalized anxiety disorder worry a lot. Their anxiety is far out of proportion to what most people would experience in similar circumstances. The individual afflicted may find it difficult to keep worrisome thoughts from interfering with attention to current activities. The focus of the worries in people with generalized anxiety disorders are everyday circumstances, such as getting up on time for work, making appointments, and doing chores. The focus of the worry often migrates from one topic to another. Troubling anxiety plagues these individuals most of the time. People with this type of disorder experience somatic symptoms such as sweating, nausea, and diarrhea. The prevalence of generalized anxiety disorders is approximately 2% to 7%, and the condition is more common in women.[9]

Individuals with a generalized anxiety disorder in a wilderness setting will probably find a new set of preoccupying concerns: bear attacks, getting lost, not keeping up, flash floods, and so forth. These anxieties will likely diminish the person's enjoyment of a wilderness experience, affect their focus, and drain their energies. Another deleterious effect of someone with abundant anxiety is a negative impact on a group's ability to work together and enjoy their wilderness experience. In most cases, these types of anxiety problems do not represent emergencies. They are chronic problems that can be managed in the field. Good leadership can prove extremely valuable in this type of circumstance.[4] A person with a generalized anxiety disorder needs ample reassurance. A good leader can provide such reassurance and appoint another supportive member of the wilderness team to assist. Modeling constructive behaviors with an anxiety-ridden participant can help other members learn how to deal with him or her. Building rapport and trust with the afflicted person is a good first step to helping them. A bit of humor, uncritical listening, and simple reassurance may be sufficient. It is not worth trying to talk someone out of his or her worries because it is unlikely to be productive and could consume a group's energies. In the field, a benzodiazepine such as lorazepam (Ativan) may be useful.[11] Side effects include sedation, memory difficulties, and impairment in motor coordination. Sustained use can lead to physical dependence and withdrawal symptoms on abrupt discontinuation. Thus, such medications must be used cautiously in the field. However, they can be very helpful for short-term relief of anxiety-related symptoms.

Probably one of the more common specific phobias encountered on a wilderness outing is fear of heights or exposure (to an edge with a drop-off). Other common phobias are to snakes, spiders, and water. A specific phobia is an unreasonable fear in anticipation of or exposure to a particular object or situation.[1] The intensity of anxiety with the exposure may vary from relatively mild to extreme panic. In some instances, a phobia to heights may not be possible to overcome in the wilderness. For instance, a trail that crosses a bridge over a chasm or has a section of precipitous exposure might be more than an individual with a specific phobia to heights can manage. In this instance, it may be necessary to find another route. Sometimes, gentle reassurance or distraction may be all that is required. The author has sometimes been successful in engaging height-phobic people in distracting conversations while backpacking in areas with dizzying exposure. However, if an individual was likely to respond with extreme anxiety, then it would not be safe to use such psychotherapeutic measures. As with a generalized anxiety disorder, benzodiazepines can be useful in the field for these individuals,[11] but because they have side effects of sedation and may disturb motor coordination, they should not be used when an individual must be alert and maintain full physical abilities.

People with reasonably well-controlled panic disorders may venture out into the wilderness and find their symptoms becoming worse. Individuals with panic disorders experience recurrent, unexpected panic attacks. A panic attack occurs during a discrete period of time lasting approximately 10 to 30 minutes and is manifested by several of the following symptoms: a pounding heart, sweating, trembling, chest pain, nausea, dizziness, numbness, chills, hot flushes, and shortness of breath. During a panic attack, a person may fear that he or she is dying, having a heart attack, going crazy, or losing control. Such individuals often visit emergency departments fearing they have had a heart attack or that something is seriously wrong with them physically. Any person with one of the anxiety disorders described in this section may experience a panic attack, but the person with a panic disorder experiences recurrent, unexpected panic attacks. These attacks occur without warning and seem to come "out of the blue," in contrast to being in response to a specific trigger as with specific phobias described previously. As a result of recurrent attacks, people may develop avoidant behaviors because they do not want to be in situations where escape might be difficult or embarrassing, or where help might not be available (agoraphobia). When these people are caught in these situations, they have a desperate desire to flee. Agoraphobia may lead people with panic disorders to avoid leaving their homes, or they may leave only in the company of trusted companions.

Panic disorders occur in approximately 1% to 2% of the population.[1] Treatment with selective serotonin reuptake inhibitors (SSRIs), such as fluoxetine or sertraline, or benzodiazepines, such as alprazolam (Xanax) or clonazepam, or a combination of the two with psychotherapeutic intervention can be successful in controlling panic disorder and agoraphobia.[10] People with panic disorder whose symptoms are not problematic may safely enjoy a wilderness adventure without difficulty. However, remote locations, bad weather, or physical challenges may increase the stress on such individuals, causing them to experience a resurgence of symptoms. Having panic attacks in the wilderness may precipitate agoraphobia. It is also possible for people to begin to have panic attacks and agoraphobia symptoms for the first time on a wilderness adventure. The afflicted person may want to escape from the wilderness and may be completely intolerant of being left alone, even for brief periods.

People having a panic attack in the wilderness may present a diagnostic dilemma. They may look like they are having a heart attack or acute respiratory distress caused by pulmonary edema. A careful history indicating the person has a history of panic disorder and does not have a history of heart disease may help.

However, for an older person, who may have risk factors for heart disease such as smoking, diabetes, or obesity, the distinction between panic disorder and symptoms of a myocardial infarction may be nominal. People who have a panic attack usually begin to calm down within 30 minutes to an hour. Symptoms often respond to benzodiazepines such as lorazepam, reassurance, and support. It may be important to avoid leaving such people alone, but they should not be crowded either. Providing regular doses of benzodiazepines to an individual with a panic disorder or increasing the dosage of currently prescribed benzodiazepines may be enough to allow completion of the trip. However, people with intense and recurrent panic attacks with agoraphobia may not be able to function on a wilderness trip and may have to be assisted out of the wilderness or evacuated if judicious use of lorazepam, reassurance, and emotional support does not help. It is also important to keep in mind that benzodiazepines can be sedating and adversely affect motor coordination. This is also true of SSRIs, but to a lesser extent.

A person on a wilderness trip is observed constantly stopping to wash his or her hands in a stream or lake, or with water they are carrying. The person's hands are observed to be raw from frequent washing, and the time consumed in this activity is slowing the group's progress to a crawl. When confronted, the individual apologizes and acknowledges that the behavior is irrational, but explains that he or she is deathly afraid of germs and cannot cease the behavior. The obsessions and compulsions of a person with obsessive-compulsive disorder (OCD) are usually seen as irrational by the person experiencing them, but at the same time he or she feels helpless to stop them. If a person with OCD is prevented from performing ritualistic compulsions, he or she may experience full panic attacks or near panic. The obsession of the compulsive hand washer is preoccupation with contamination by germs, and the compulsion is hand washing. Another person on a wilderness trip is frequently overheard counting in a whispered voice. When asked about the counting, the individual (with considerable embarrassment) explains that they are afraid they will attack someone, so every time they have a violent thought they count to 100.

A person with OCD may not be able to adhere to the requirements of a wilderness trip. For instance, the chronic hand washer may slow a group so profoundly that it cannot make reasonable progress. The example of the person who tries to control his or her anxiety about acting violently by counting may present less of a problem. People with OCD are not typically violent, but a person who is observed to be constantly counting to ward off violent behaviors may have difficulty comfortably fitting in with a group. In the general population, the prevalence of some manifestation of OCD is greater than 1%, but the symptoms often vary in intensity.[9] People with OCD are often successfully treated with SSRIs, such as fluoxetine or sertraline.[10] The symptoms may not be totally eliminated, but they are diminished in intensity with treatment. It is unlikely that OCD symptoms will suddenly erupt on a wilderness trip. It is more likely that preexisting symptoms will increase under the stress of a wilderness experience. Frank disclosure to a wilderness group about the OCD symptoms of a member of the group can diminish the group's anxieties about the person's odd behaviors and make the afflicted person less likely to be isolated by the group.

Mood Disorders

Everyone experiences sadness, moodiness, happiness, and elation at different points during the course of their lives. Such moods are a normal part of human experience. Periodically, such emotions become extreme and prolonged and interfere with normal functioning. It is unlikely that people with severe depression or unstable bipolar disorder (manic-depressive illness) will venture into the wilderness. However, people who are being successfully treated or who have relatively mild symptoms may participate in wilderness adventures. The wilderness experience may improve or worsen mood disorder symptoms. People who are depressed feel sad, useless, and bad about themselves and the world. Their view of the world is dark and they have difficulty believing things will get better. Depressed people have difficulty sleeping, lose appetite, have poor energy, lose concentration, withdraw socially, and have difficulty enjoying anything. They may cry for no apparent reason. The most common diagnostic term for this type of depression is *major depression,* and the prevalence of this disorder is approximately 5%.[9] At their worst, people with severe depression may become psychotic and suicidal (see sections on Suicide and Violence Potential and Psychotic Disorders).[1] However, people who are having relatively mild problems are unlikely to develop severe symptoms rapidly; severe symptoms gradually appear over a period of weeks or months. The more likely problem on a wilderness adventure is someone who started with mild symptoms that are gradually growing worse. Such individuals may already be taking antidepressant medications (typically, SSRIs like escitalopram [Lexapro] or paroxetine [Paxil]). These medications work slowly over a period of weeks, so a change in dose in the midst of a wilderness trip probably would not provide significant benefit. The most common problem the wilderness group may face with a depressed person is overall impact on group morale. Emotional outbursts, crying for no apparent reason, or offhand comments about suicide will likely concern and distress others in the group. If group members provide encouragement and emotional support, this may help the depressed person get through the trip safely without too negative an impact on morale. However, severe symptoms may not respond to ordinary support. Lack of response can anger others, who may begin to feel conflicted because they recognize that they are punishing someone who is already suffering. If this is the case, it becomes necessary to hasten the depressed person's departure from the wilderness, especially if there is suicidal ideation.

Persons with bipolar disorder experience major depression alternating with mania over a period of months, often with periods of normal functioning between abnormal episodes. The prevalence of bipolar disorder is approximately 1%.[9] During periods of normal mood or in the early stages of a manic phase (hypomania), bipolar persons may participate in wilderness adventures with no difficulty. During a period of hypomania, a person is very positive, productive, hard working, energetic, and expansive. But as the person becomes manic, problems become readily apparent. There is often rapid, pressured speech that is difficult to interrupt. Sufferers might not sleep at all, be excessively gregarious, and begin to believe they have superhuman powers. An individual may try to awaken a wilderness group in the middle of the night to hike up a nearby peak. When rebuffed, the manic person might take off alone with no water or protection from the weather because of the belief that he is superhuman. An individual who has become manic in the wilderness may present an extreme hazard to himself and possibly the group. Once manic, a person no longer listens to reason or will not listen for long. An individual with mania in

the wilderness should be considered a medical emergency and be evacuated as rapidly as possible.

Careful questioning may reveal the person was taking psychiatric medications, such as mood stabilizers (lithium or divalproex [Depakote]) or antipsychotic medications (risperidone [Risperdal] or olanzapine [Zyprexa]), or both. People who take lithium must avoid dehydration, which results in lithium toxicity. Lithium toxicity begins with tremulousness and can proceed to seizures and death. If lithium toxicity is suspected, the drug should be stopped and the person should be well hydrated. The patient may need dialysis to resolve the toxicity, so evacuation should be strongly considered. People with bipolar disorders sometimes stop their medications because they enjoy how they feel when they are hypomanic. If the person is willing, use of benzodiazepines like lorazepam or antipsychotic medications like risperidone can begin to control some of the symptoms. Risperdal 2 to 6 mg or lorazepam 4 to 8 mg spread over the course of a day may be needed to keep such an individual calm.[10] There is a likelihood that the person will not cooperate in taking medications because he or she enjoys the high of being manic. It may be possible to coax a manic person out of the wilderness through encouragement and enticements. Confrontation is unlikely to produce beneficial results because people with mania tend to be extremely irritable and sometimes aggressive. If they have any weapons, these should be removed. People in a manic episode can go for days without sleep. If they will not voluntarily leave the wilderness, then help should be obtained as soon as possible. An evacuation may require the individual to be forcibly restrained and medicated. While waiting for help to arrive, members of the group should do whatever they can to contain the manic individual's excesses in order to keep the person and the group safe. Such an individual should be monitored constantly.

Psychotic Disorders

Individuals with severe mental illnesses, such as schizophrenia or schizoaffective disorder, do not typically venture into the wilderness, especially if they are in a deteriorated state. Their overall functioning is too low for them to organize themselves to the point that they could join a wilderness group. Yet some have been able to benefit from treatment, usually in the form of antipsychotic medications.

The 1% prevalence of schizophrenia worldwide is significant.[9] Tragically, the illness often presents in young adulthood. The hallmark of these disorders is a substantial period, at least 1 month, during which the individual becomes psychotic. An individual who has lost the ability rationally to perceive reality is psychotic. This individual experiences delusions, which are false beliefs not based in reality, and hallucinations, which are sensory perceptions without a sensory stimulus. A person who believes a sinister group worshiping the devil has implanted a transmitter in his mouth (delusion) and hears mysterious people talking about him saying he is pathetic and should kill himself (auditory hallucination) is considered psychotic. There are many types of delusions and hallucinations, but the essential feature is a disturbance in a person's perception of reality.

People with psychotic symptoms usually cannot be talked out of their beliefs and perceptions. People who are successfully treated with medications may still have some unusual or even delusional beliefs, but these have receded into the background and are not an active concern for the individual. In addition to psychotic symptoms, people with schizophrenia or a schizoaffective disorder often seem awkward or distant interpersonally. They might express very little emotion and have a wooden personality. Their thoughts may seem a little jumbled, disorganized, and concrete. Their motivation may be very low. If receiving adequate treatment, such individuals might enjoy a wilderness adventure with no difficulty, although their personal contribution to the group may be nominal.

Antipsychotic medications cause a wide range of side effects, especially older antipsychotic medications such as haloperidol (Haldol). Side effects are many, including extrapyramidal symptoms (Parkinson's-like symptoms that can cause considerable difficulty in moving fluidly), acute dystonias (painful involuntary muscle contractions), insomnia, sedation, and orthostatic hypotension.[10] Side effects alone may preclude a person from participating in a wilderness adventure, but people who have been on their medication for weeks or months may have adapted to the side effects, and newer antipsychotic medications, such as aripiprazole (Abilify), have fewer side effects.

Problems arise for the wilderness group if a member of the group becomes acutely or floridly psychotic. Symptoms may range from the mild, such as a person asking the group why they are always talking about him, to paranoid beliefs that snipers are in the woods trying to kill the psychotic individual. Very mild symptoms may be tolerable or the individual might respond to periodic gentle reassurance (reality orientation). An increase in the dosage of the individual's already-prescribed antipsychotic medication may help, or a benzodiazepine may have a calming effect, at least temporarily. If psychotic symptoms become more severe, including progressive paranoia or agitation, or if the victim begins making bizarre accusations toward others in the group, the situation should be treated as a medical emergency and the person should be evacuated as soon as possible. Psychotic individuals' behavior may be unpredictable and violent toward themselves or others. There are many causes of psychotic symptoms, including schizophrenia, schizoaffective disorder, mania, illicit drugs such as methamphetamine, and organic causes such as brain tumor or metabolic disturbance. In the field, it may be extremely difficult to determine the cause of psychosis, but a good history can sometimes narrow the differential considerations considerably.

Organic Mental Disorders

There is a wide range of medical problems in the wilderness that can cause behavioral symptoms, ranging from, for example, mild confusion associated with high-altitude hypoxemia, to delirium associated with high-altitude cerebral edema. A person in the wilderness may have fallen and hit his or her head on a rock, initially resulting in a minor scrape and a headache, but progressing to a subdural hematoma that may become life-threatening. It is crucial to recognize signs of delirium in the wilderness. Delirium usually signals a medical emergency. A person with delirium often has a fluctuating level of consciousness, such that he or she may be awake and alert one minute and sleepy the next. Delirious persons may be hard to arouse, but then seem to come around on their own. People with delirium are often confused and disoriented to date, time, and place. Concentration, memory, and calculation ability may be impaired. Visual, tactile ("There are bugs crawling on me"), or other types of hallucinations may appear. Paranoid delusions might be reported.[9] The key to differentiating delirium from psychosis without delirium is that with manic or schizophrenic psychosis, attention, alertness, and level of

consciousness are preserved, and there is no fluctuation of level of consciousness.

If delirium is suspected, a cause must be determined as rapidly as possible. Dehydration, hypoglycemia, high-altitude cerebral edema, head injury, meningitis, encephalitis, drug or alcohol withdrawal, and heat illness, to name a few, can cause delirium. In their confusion, people with delirium can easily injure themselves, so they must be watched constantly. If an easily treatable cause cannot be determined and treated, evacuation may become necessary. The author was involved in reviewing a Grand Canyon backpack trip down Kanab Canyon in which a member of the group became increasingly unsteady and confused as he progressed down the canyon. He would be fine for a few hours and then he would become confused. He fainted and fell a couple of times, but was not seriously injured. Other members of the group carried most of his equipment. Shortly after the group made it to the beach lining the Colorado River, the man became stuporous. Soon he was barely awake. The leader of the group raced for help and one person was left with the delirious man. As hours passed, he became semicomatose. After 24 to 36 hours, a helicopter evacuated the victim to a nearby hospital, where he was treated for pulmonary edema. Had the helicopter arrived any later, the man probably would have died.

Personality Disorders

Personality disorders are psychiatric disturbances that can cause considerable difficulty in the wilderness, even though they rarely present as an emergency. Personality disorders are common, with an approximately 6% prevalence in some studies.[9] There are 10 different categories of personality disorder described in the DSM-IV-TR. People with personality disorders have enduring maladaptive patterns of behavior, usually dating to adolescence. Using a narrow set of adaptive strategies inflexibly leads these individuals to have chronic problems at school, at work, and in interpersonal relationships. Ironically, people with personality disorders do not tend to see themselves as having problems but rather blame some aspect of their environment for their difficulties. They tend to blame others for their failings. As a result, others see these people as troubled and troubling. Individuals with personality disorders unwittingly create their own special circle of misery. Depression and substance abuse are common additional problems in this population.

People with schizoid personality disorders are loners and tend to avoid relating to others except on a shallow and superficial level. People who are schizotypal have odd and eccentric preoccupations and manners. They may be enamored of astrology, spiritual vortices, or numerology, and they tend to relate in an awkward or uncomfortable manner. These people may not cause too many problems on a wilderness journey, but they may be annoying or aloof and unable to join in comfortably with a group. People with a paranoid personality disorder are constantly suspicious of others, believing that others have malevolent intentions toward them. The suspicions are not totally absurd, as they are with someone who is psychotic. For instance, they may believe that fellow travelers do not like them or will steal from them, but they do not believe that transmitters have been implanted in their brains or that the FBI is conspiring to kill them. A person with persistent suspicions of others can be problematic for a wilderness group, whose successful functioning depends on trust between members. Someone who is chronically suspicious may be incapable of establishing trusting bonds with others. Group leaders need to work doubly hard to draw such individuals into a group and avoid having the group isolate the aberrant group member. Such isolation serves only to amplify the paranoia and discomfort between the affected person and the group.

Another group of personality disorders is sometimes referred to as the "dramatic and erratic" personality disorders. The narcissistic personality-disordered person is one who considers himself special and unusually talented, and therefore deserving of special praise or considerations. These are people who want to be first in line, or do not want to carry a heavy pack like everyone else. The person with a histrionic personality disorder is colorful and dramatic and demands to be the center of attention. He may be perceived as vain and shallow and often exaggerates physical complaints. A small blister may be declared to be causing excruciating pain and discomfort. The person with a borderline personality disorder is unstable and has a fragile sense of identity. Relations with a borderline person tend to be stormy because one minute she is begging forgiveness, and the next shouting expletives. The person with an antisocial personality disorder is the "confidence man" who may manipulate, intimidate, or steal to obtain what he wants. He can be superficially charming, but is irresponsible and unreliable.

Sometimes an unusually talented and psychologically intuitive leader or group member is able to run interference between the afflicted person and the rest of the group. At other times, the maladjusted individual can be so disruptive that the leader or the rest of the group must insist that the person leave the group. If this is the case, the person should be assisted from the wilderness. These individuals may not willingly accept being dismissed from a group and may be combative physically or verbally. They may even become enraged and report that they are going to kill themselves, sue, or seek some other form of retaliation. Nevertheless, if the individual cannot safely function as part of a wilderness group, the leader must remain firm in dismissing the person and assisting him or her safely out of the wilderness.

A third group of personality disorders that may be less problematic includes the avoidant, dependent, and obsessive-compulsive personality disorders. The person with an avoidant personality disorder is constitutionally shy and fears rejection, but desires relationships with others. The person with a dependent personality disorder is someone who looks to others to make decisions and care for them. The person with an obsessive-compulsive personality disorder is task oriented and hard working, but is also perfectionist, moralistic, and humorless. Note that the obsessive-compulsive personality disorder is to be contrasted to OCD, described previously. A combination of social support, reassurance, and humor may be all that is needed to help people with these disorders function reasonably well with a wilderness group. The person with an obsessive-compulsive personality disorder may become tiresome and tedious because of controlling and detail-oriented behavior, but may also make valuable contributions because of hard work and orientation to task.

A normal person uses a range of strategies to adapt to the different challenges presented at work, at school, and in social relationships. People with personality disorders use a very limited range of strategies in an inflexible manner in all situations. It is normal and constructive to be suspicious when buying a used car, detail oriented when designing a new aircraft, and colorful at a party. But personality-disordered people have

just one song to sing. They sing it repeatedly, even when it is not adaptive and contributes to their own unhappiness.

Substance Use Disorders

People who abuse illicit drugs or alcohol should not be on wilderness adventures; they are a hazard to themselves and others. It is not uncommon for the wilderness traveler to enjoy a small quantity of alcohol after a long day, and this may be considered acceptable. However, if it is discovered that someone is using methamphetamines, cocaine, heroin, LSD, or other illicit drugs on a wilderness trip, they should be told to stop and the drugs should be confiscated and destroyed. If someone is unwilling or unable to stop using these drugs, then he or she should be escorted out of the wilderness. More problematic is a person who is prescribed narcotic pain medication (e.g., oxycodone). Narcotics are central nervous system depressants that can impair concentration and coordination. In modest doses, such medications may be reasonable, but at higher doses, or if the individual is abusing prescription narcotics, then it is not safe to have him continue a wilderness journey and he must be escorted out of the wilderness.

Some people may be unaware that they have a substance abuse problem when they start a wilderness trip, until they start to experience physical or psychological withdrawal. Alcohol withdrawal is considered a medical emergency because of seizures and delirium tremens.[2] A careful history should establish if alcohol or other drugs may be the cause of the problematic symptoms. Withdrawal symptoms usually start with shakiness and increased heart rate and blood pressure. If withdrawal is suspected, a benzodiazepine (e.g., lorazepam) can be given. Alcohol can be used if necessary and available. Withdrawal from barbiturates or benzodiazepines can present similarly to alcohol withdrawal and can be life-threatening. Giving a benzodiazepine can diminish symptoms of alcohol, benzodiazepine, and barbiturate withdrawal. Anyone withdrawing from these substances should be taken to a hospital as soon as possible and should not be allowed to remain in the wilderness, even if enough benzodiazepine is available to diminish the withdrawal symptoms. There are too many potential complications to try to manage withdrawal in the wilderness. People who abuse cocaine, methamphetamines, or narcotics will not experience life-threatening withdrawal, but their symptoms can be extremely uncomfortable and disabling. Extreme irritability and fatigue are common, as are aches and pains typical of influenza. The withdrawal symptoms may last several days. The wilderness is no place to undergo withdrawal from cocaine, methamphetamines, or narcotics, so the person should be assisted out of the wilderness as soon as possible.

On a wilderness trip someone may experiment with natural plants hoping to achieve an induced "high," or someone may ingest, inject, smoke, or inhale a psychedelic drug. Tea made with jimson weed (sacred *Datura*), a common plant in the southwest United States with large, white, tubular flowers, may induce florid psychosis and delirium. Delirious persons may shed clothes, experience severe sunburn, become dehydrated, and expose themselves to all manner of risks (e.g., cliffs, whitewater). Plants that can cause psychotic symptoms and delirium (see Chapter 58) should be avoided.[2] People who are experiencing drug-induced psychotic symptoms or delirium must be watched constantly until evacuation to a hospital can be arranged.

SUICIDE AND VIOLENCE POTENTIAL

Suicide is a major public health problem. The suicide rate in the United States is approximately 12.5 per 100,000, with a recent increase in suicides among people 15 to 24 years of age.[9] Depression and alcoholism are frequently associated with suicide. People who are depressed, lonely, or physically ill might commit suicide. Rejection, unemployment, and legal problems are often associated with suicide. Many factors indicate a greater potential for suicide. The factors vary with age. In the context of a wilderness trip, factors to consider are depression and recent loss. For example, a participant recently divorced, widowed, or fired from his or her job shortly before the trip may be at higher risk for suicide. A person who is isolated and lacks social support will be at greater risk for suicide. A family history of suicide or past history of suicidal behavior increases suicide risk. If there is a concern that someone might be suicidal, then they should be asked about suicidal thinking in a straightforward, concerned manner. Asking about suicide does not increase someone's suicide risk. People who kill themselves often think about or even become preoccupied with ending their lives. They plan how they will kill themselves, so they should be asked about plans for suicide. If someone has become suicidal, it may be useful to obtain an agreement from them that they will not try to kill themselves in the wilderness. If the suicide potential is felt to be great, the person must be watched closely. People who are suicidal should be escorted out of the wilderness and brought to psychiatric help. People who are delirious or psychotic can suddenly become impulsively suicidal in the midst of their confusion and frenzy, so they must be evacuated from the wilderness as soon as possible.

United States culture is much more violent than that of other industrialized nations. Each year, 4 to 5 million assaults are reported in the United States.[9] Rape, child abuse, homicide, and other violent crimes are far too common. Men are much more likely to be violent than women; young men are particularly prone to violence. Substance abuse is highly correlated with violence. People who have a history of acting violently in the past are more likely to do so in the future. People who are going to be violent often signal their intentions. They act aggressively and clench their fists and jaw, or speak loudly or shout. They may have thoughts of acting violently, so they should be asked if they are thinking of hurting anyone. People who have a personality disorder, abuse drugs or alcohol, or are psychotic are at an elevated risk for violence. If a concern exists about someone being violent in the wilderness, weapons should be removed if possible and everyone should be vigilant. The psychotic or delirious person who is prone to violence should be evacuated. If the issue is substance abuse or a personality disorder, or both, the person should be asked to leave the group and assisted out of the wilderness. If the person refuses to leave the group, then help should be sought and the entire group should leave the wilderness setting.

SOMATIC COMPLAINTS

Some people are more sensitive to physical irritations and discomfort than are others. For most people, a headache, scrape, or sore muscle is not cause for alarm. But for some individuals, a headache is a sure sign of a brain tumor, the scrape will

undoubtedly become gangrenous, and sore muscles are evidence of a smoldering systemic illness. Simple reassurance that there is no major pathologic process may be enough for many people, but others continue to worry despite solid medical evidence to the contrary. These people suffer from hypochondriasis.[1] Insomnia, gastric distress, and fatigue are common complaints associated with travel and are usually not cause for concern. However, persons who tend to magnify the significance of these types of common complaints worry excessively and may voice their concerns to the entire group. Occasionally, this can have the effect of creating a kind of "mass hysteria," where other members of the group begin to experience the same symptoms. For a person who is very worried about his or her health, repeated reassurance might be successful in ameliorating the problem. The group leader can periodically inquire about the status of the problematic symptoms. The difficulty lies in discriminating symptoms of little consequence from those that might be of greater importance. The key is to take a good history and consider a reasonable differential diagnosis leading to an organic explanation for the symptoms before concluding that the complaints are primarily of psychological origin.

RESPONSE TO CRISIS

Disasters

Avalanches, storms, rockfalls, climbing and whitewater accidents, and many other untoward events can lead to the death or serious injury of one or more group members. People who venture into the wilderness usually do not anticipate someone getting seriously injured or killed. Even persons who have taken wilderness first-aid courses do not enter the wilderness expecting the worst. Otherwise, no one would participate in these types of activities. There are many descriptions of outdoor trips that encountered crises.[7]

Humans wrap a cloak of denial around themselves. We know that bad things happen, and we may even be prepared, but no one ever expects it will happen to them or on their watch. Thus, when a crisis or tragedy occurs, it is almost always an emotional shock. When you watch your climbing partner fall and die or your buddy drown when his kayak flips in whitewater, there is a reaction of horror, dread, and disbelief. The situation is overwhelming, and people often describe a sense of unreality associated with the tragic event. A period of grief and preoccupation regarding the event is common. People who are involved in tragedies often need to replay the event in order to put it into some type of meaningful personal context. People who are relatively intact psychologically and have good social support can usually mentally process a trauma successfully without long-term mental health problems. As the size of a disaster grows and its impact on a person or a community's support networks increases, the likelihood of long-term mental health problems grows. People who handle trauma by psychological avoidance and casting blame may have poorer outcomes.[12]

In response to natural disasters, some people develop acute stress disorder (ASD) or post-traumatic stress disorder (PTSD). Estimates of the frequency of these disorders after disasters (man-made or natural) vary from 2% to over 50%.[12] The symptoms typical of these disorders include efforts to avoid reminders of the traumatic event and markedly increased anxiety when exposed to such reminders. The affected person may be flooded with uncontrollable memories, including bad dreams and flashbacks (feeling and behaving as if the trauma was happening again). The person may feel a sense of emotional detachment from others. Finally, people often have difficulty concentrating, have difficulty sleeping, feel restless, and become easily startled. The symptoms begin to interfere with daily interpersonal and occupational functioning. The symptoms can begin immediately after a disaster. If they persist for more than 2 days but less than 1 month, the disorder is called ASD. If the symptoms are present for more than 1 month, the disorder is called PTSD.[1] For most people, symptoms diminish as time passes, although there is a common intensification of symptoms around the time of the 1-year anniversary of the tragedy. Symptoms may become chronic and require ongoing psychiatric care. The author has evaluated men who were held as prisoners of war and were subjected to cruel deprivation or torture during the First or Second World Wars. Some did not experience symptoms of PTSD until they retired, many years after they were traumatized (delayed PTSD).[1]

The immediate response to a wilderness disaster should emphasize ensuring the safety and security of the survivors. This includes provision of food, water, warmth, and shelter. People who have been involved in the disaster should be clearly informed of what has happened and what rescue efforts are underway. Communication with loved ones should be facilitated. People should be provided as calm a setting as possible to decrease arousal as much as possible. Medication use is controversial, with SSRIs showing some clear benefits during the weeks and months after the disaster. However, use of psychotropic medications in the immediate aftermath may not be necessary and may even be harmful, particularly if the sedative properties interfere with survival behavior or motivation. Individualized support should be provided as needed. The concept of critical incident debriefing is controversial, with some studies indicating worse outcomes after such interventions.[12] People providing support should approach survivors with an expectation of normal recovery. Efforts to force the trauma victim to discuss the trauma should be avoided. Discussion is appropriate if the person initiates it. Connecting survivors with continuing information about the aftermath of the disaster and the means to contact other survivors can be helpful. Clinics for disaster survivors to obtain assistance in emotionally processing recent events should be provided. Such support should not focus on details of the incident unless the person initiates such discussion, because premature discussion may lead to unnecessary arousal and possibly long-term problems.

Survival Psychology

It is not uncommon for people to become lost in the wilderness. Human spatial orientation abilities are limited; we often function under the illusion that we know exactly where we are.[3] People rationalize disorientation in the wilderness by explaining away inconsistencies between their topographic map and the actual landmarks before them. The author experienced this phenomenon on a recent backpacking trip in the Wind River Range of Wyoming. Although the landmarks did not match the map, I rationalized the inconsistencies and believed a trail would become obvious at the top of the next pass. There was no trail. After an hour of staring at the landmarks and the topographic map, I finally determined our location. Some people are not so lucky and find themselves completely disoriented in the wilderness. As a person becomes convinced that he is completely lost, he might experience intense fear, panic, and disorientation

unique to this type of dilemma. People with considerable experience in the wilderness may demonstrate inexplicable behaviors, such as abandoning provisioned backpacks or other potentially valuable items. Some have even removed clothing, which increased their exposure to the elements.[3]

After becoming lost, some persons develop a strategy and carry it through, but when it fails, they abandon hope and resign themselves to fate. Ironically, children younger than 6 years of age fare better when lost than do older children and adults. The explanation for this paradox might be that younger children act on instinct and seek shelter when cold and water when thirsty, but older children and adults panic and sometimes overlook obvious and simple means to help them survive.[3] In groups that become lost, good leadership is often a key component to survival of the group. Perhaps the most famous survival story is that of Ernest Shackleton's ill-fated voyage to reach the South Pole in 1914. His ship, the *Endurance,* broke up in the ice. Under his leadership, the group survived months of incredible hardship. Against all odds, all 27 men survived.[5]

LEADERSHIP

Wilderness activities are usually not solo affairs. Outdoor adventures can range from a 5-mile day hike with a social group to an expedition involving dozens of people to climb to the summit of Mt. Everest. For adventures to be successful and safe, some level of planning and preparation is required, along with leadership or guidance. People who share wilderness activity together experience the same types of group interactions that occur among coworkers, families, and myriad other groups. A safe and rewarding wilderness adventure often begins long before the actual trip. Careful planning includes attention to numerous issues: food, equipment, maps, screening of participants, impact on environment, and permits, among others. The planning process itself may involve a group working together. Subsequent to the adventure, some type of communication may be essential to bring spiritual, emotional, or logistical closure to the experience.

The manner in which groups interact can result in a safe, challenging, and rewarding adventure or a conflicted, dangerous, and disorganized affair. Trips in the wilderness sometimes involve people who may not know one another very well, yet are thrown into close quarters and frequent close contact for an extended period. Regular close contact inevitably leads to annoyances and conflicts, but can also result in pleasurable camaraderie. Leadership is one key ingredient to a successful wilderness experience. A group in the wilderness without a leader and a plan can be a recipe for disaster. Groups without direction and guidance tend to deteriorate. They often break into factions or drift aimlessly. Morale suffers and tempers flare, as arguments and fights erupt. Such situations tend to bring out the worst in individuals. However, an effective leader can bring out the best in everyone. The type of leadership needed for a day hike differs from that needed for a difficult expedition. But even the short hike needs coordination. What if someone breaks his or her leg on the hike? Who will go for help, administer first aid, and reassure upset members of the group?

Leadership skills should be taught and developed; otherwise, the leader will make the same mistakes repeatedly.[4] Good leaders have a vision for the outings, but are flexible enough to adapt according to the exigencies of evolving circumstances. Successful leaders care about their people and build teams that use the strengths of each member of the group. Communication abilities are an essential ingredient in a competent leader. Sometimes, leaders emerge during crisis or disaster and are critical for survival of a group.[6] Leadership and interactions among group members evolve during the course of a trip. Often, leadership is fluid, as people with different skills take leadership roles in different functional areas, such as building fires, reading maps, or maintaining good humor and morale. People can be remarkably courageous, kind, and ingenious but they can also be petty, mean, and destructive. A group on a wilderness journey is a microcosm of society, where the full range of human strengths and weaknesses can be witnessed. The leader sets the tone.

The references for this chapter can be found on the accompanying DVD-ROM.

31 Wilderness Emergency Medical Services and Response Systems

Franklin R. Hubbell

Today, medicine is replete with improving communications, computer-assisted diagnosis, futuristic imaging technology, faster and luxurious emergency medical vehicles, and highly skilled trainees. However, the physical world that we explore is still rugged and enduring: majestic mountains, spectacular shorelines, wild rivers, dense tropical rain forests, horizonless polar ice sheets, arid deserts, vast oceans, and hazards beyond imagination. We enter and must sometimes be rescued, treated, and saved. First response is critical.

The wilderness will always have the ability to place humans in situations in which immediate help is simply not available. When in the wilderness, preexisting or created by a disaster, people must know how to survive and be able to perform emergency medicine barehanded, without all the technical wizardry that exists in a modern ambulance or emergency department.

The anatomy of disaster response and its limitations are very similar to those found on a mountain or backcountry rescue: time and distance work against the rescuers, where notification of the emergency can be delayed and initial information inaccurate. The weather conditions at the site of the emergency may be poor, hostile, and rapidly changing. Terrain can make travel very difficult, slow, or even impossible. Manpower, equipment, and resources will be very limited and hard to come by. Persons responding need specialized training, skills, and knowledge of everything from self-preservation skills to emergency care skills in an extended care environment.

What has changed relatively recently is the scope of practice. The principles of wilderness emergency medicine are now being applied to the expansive realm of providing care outside the "golden hour." These same techniques, methodology, and experience learned in providing extended care in the wilderness environment apply equally to providing long-term patient care in natural and human-made disasters. Earthquakes, hurricanes, blizzards, tornados, and tsunamis not only can cause thousands of deaths, but also leave many more injured, exposed to disease, and without access to emergency, long-term, or definitive care. The principles discussed in this chapter apply equally to any extended care environment outside the golden hour, from a short 1-hour delay to extended multiday management at a disaster site.

Whether an accident or medical crisis occurs in the wilderness, in the backcountry, on the high seas, or at a disaster site away from access to immediate assistance, the chain of events set in motion hopefully leads to a successful rescue and lives saved. However, how it unfolds varies tremendously, depending on the part of the world in which the critical events occur. Currently, no national or international standards for wilderness emergency medical service (EMS) and response exist. Instead, the configurations of personnel and policies reflect local, national, and international influences. In the United States, wilderness EMS is the most diverse because it is provided by a broad range of agencies and individuals with a wide variety of training and certification levels, ranging from first aid to paramedic. Canadian wilderness EMS is generally provided through the military, whereas in European wilderness situations, civilian physicians have a prominent role.

The skills developed in providing long-term patient care in the wilderness are proving to be beneficial in medical and rescue assistance in the disaster response and relief setting. Although there is not a great effort to produce national or international standards for wilderness or disaster relief, there are national and international organizations that have their own command structures, response systems, and response criteria. It may be that organizations or structures such as the American Red Cross, Federal Emergency Management Agency (FEMA), or Department of Homeland Security will address these issues and develop standards in communications, response, and medical and rescue techniques. The art form will be to create a system-wide or nationwide set of standards that can be used when diverse entities come together during an emergency to provide relief, so that they will be able to function well together.

In the world of mountain rescue, the American Alpine Club's Safety Committee gathers, reviews, and analyzes mountaineering accidents that have occurred throughout North America and publishes the annual report, *Accidents in North American Mountaineering*. The data collected illustrate both the necessary diversity of wilderness and mountain rescues and the current limitations (Box 31-1).

Several states have established (or are in the process of establishing) working protocols for providing care in the wilderness or "extended care" environment. With increased natural and human-made disasters, EMS systems worldwide have suddenly found themselves essentially operating in a "wilderness" setting because of prolonged exposure to a hostile environment, delayed evacuation and transport time, and lack of medical resources and direction. Prehospital personnel in extended care situations find themselves providing care for much longer than

Box 31-1. Mountain Search and Rescue Factors in the United States

1. Search and rescue is the responsibility of national parks, state parks, county sheriffs, or state conservation officers, depending on the state or park.
2. Most backcountry and technical rescues are carried out by volunteer rescue groups.
3. Ninety percent of all rescues are carry-outs on foot rather than with airlifts by helicopter or fixed-wing aircraft.
4. At least 95% of rescues are performed without physicians present, instead using the skills of first responders, emergency medical technicians, and paramedics, who may or may not be trained in wilderness medicine and rescue techniques.
5. Only two of the major climbing areas, Yosemite and Grand Teton National Parks, use helicopters extensively.
6. Only Denali National Park uses fixed-wing aircraft extensively and helicopters occasionally.
7. Only three national parks have rangers who are trained specifically for technical rescue, advanced medical support, and helicopter operations: Yosemite, Grand Teton, and Mt. Rainier National Parks.
8. National and state parks are not mandated with a "duty to rescue." However, virtually all parks provide rescue service. Most parks have a budget for these activities.
9. Many roadside climbing areas and popular backcountry areas are not within the jurisdictions of parks. Technical and backcountry rescues carried out at these locations are often performed by local rescue squads, fire departments, and ambulance units, usually without the benefit of specialized training or technical backcountry skills.

the golden hour and discover that their street-oriented skills are often very inadequate.

Internationally, the Union Internationale des Associations d'Alpinisme (UIAA) (International Mountaineering and Climbing Federation, www.uiaa.ch), headquartered in Bern, Switzerland, has established criteria and courses for postgraduate training for physicians in mountain medicine. After fulfilling these requirements, physicians in the European Union become certified in wilderness medicine and can practice the relevant skills in an appropriate arena. There is now a similar program for physicians being offered in the United States by the Wilderness Medical Society (www.wms.org). The Wilderness Medical Society has produced a curriculum on various wilderness medical topics for a total of about 100 contact hours. Upon completion and qualification, the individual becomes a Fellow of the Academy of Wilderness Medicine.

In the United States, the foundation has been set for national standards in providing emergency care and rescue by adoption and implementation of the Incident Command System (ICS). This system of command and communication was developed by the U.S. Forest Service for coordinating forest firefighting tactics in the western United States, where many different agencies had to work together while fighting large-scale forest fires. The ICS command system has been improved and adopted by many state and local agencies and has quickly become the command system standard for all emergency response organizations, especially when it becomes necessary to orchestrate several agencies working together.

The assimilation of ICS into police, fire, rescue, and EMS has offered a solution to the single biggest problem facing these services: how to coordinate and interface a variety of teams working on the same rescue effort. When each team follows its own set of operating procedures, standing orders, leadership protocols, terminology, and egos, it is often virtually impossible to effectively and safely coordinate a major rescue effort.

The ICS works well, and organizations such as the National Fire Academy and various state police, fire, rescue, and EMS offices nationwide have adopted and implemented this system at all levels of emergency response. ICS is now the standard for responding to any emergency situation, ranging from a single department answering the call to a minor motor vehicle crash to a complex search and rescue effort involving many agencies and rescue teams. ICS is the foundation of emergency response because it establishes a common language and command structure that allows everyone involved to anticipate how the effort is going to proceed, what their individual and team jobs are, who is in charge, and how to communicate with each other. As the ICS becomes widely adopted and utilized, it will help establish a national standard upon which national prehospital standards, including wilderness or extended care protocols, can be built.

Most states have now produced a set of working protocols for providing prehospital emergency care. However, few have attempted to write protocols for providing emergency care in the wilderness or extended care environment. At the time of this writing, Maine, New Hampshire, and Alaska have tackled some of the issues associated with providing wilderness care. New Hampshire is the only state that has written a complete set of emergency medical protocols for the wilderness, extended, or long-term patient care environment.

Wilderness emergency medicine is a combination of emergency medical training and outdoor wilderness skills. Blending these elements, although essential, is not necessarily natural or easy. The art and science of prehospital emergency medicine began over 35 years ago in the United States and has evolved into a highly regimented and well-defined subspecialty of emergency medicine. Today, there are highly trained first responders, including emergency medical technicians (EMTs) and paramedics, as well as organized EMS systems. Each state has an independent EMS system and its own regulations; a truly national standard does not yet exist. The only national prehospital EMS standard that currently exists is the testing and evaluation and certification standards developed by the National Registry of EMTs.

The National Registry of EMTs offers individual state EMS systems standardized written and practical examinations for first responders, EMT-Basics, EMT-Intermediates, and EMT-Paramedics. This guarantees that regardless of where someone was trained, he or she will be tested using the same standard. Compiling the statistics from each of the 50 U.S. states, there are about 84,000 first responders, 500,000 EMT-Basics, 140,000 EMT-Intermediates, and 140,000 EMT-Paramedics who are currently certified and licensed to practice prehospital care. Because a central agency to monitor persons trained in providing wilderness care does not exist, there is no registry of

trained individuals. Organizations have been training people in wilderness medicine for the past 20 years. Currently, there are more than 100 different organizations, schools, and individuals offering various forms of wilderness medical training. It is not unreasonable to estimate that more than 500,000 persons have some sort of certificate training in prehospital wilderness EMS.

Training programs that focus on rapid response, rapid intervention, and rapid transport to advanced care facilities exist nationwide. Prehospital personnel are prepared to work within the framework of the golden hour, when time is precious and critical actions save lives. This is a nationally accepted urban standard to which all EMS personnel are currently trained. Although this standard is appropriate for evaluating and training urban EMS personnel and response systems, it is often not adequate for rural, wilderness, mountain, or extended EMS personnel and response systems. In these situations, patient care is measured in hours and days rather than minutes.

Traditional EMS recognizes rapid notification (the 911 system), dispatch, response, assessment, thorough prehospital care, transport, evaluation, and critical care in a hospital emergency department. Rapidity is the most critical factor that distinguishes urban emergency medical care from wilderness emergency medical care. However, time is not the only difference. Wilderness emergency medicine is governed by a complex set of medical skills and protocols, equipment requirements, and other specialized skills, including different attitudes or psychological requirements, each of which combine premeditated action with improvisation. A productive mental attitude comes largely from the individual's training, expertise, and experience in the outdoors.

In mountain and wilderness outdoor activities, including mountain and wilderness rescue, haste truly makes waste, which may, in certain circumstances, cost lives. As a result, wilderness and mountain rescue teams must achieve a balance between the urgency of the situation and the necessity for adequate preparation. This is not an easy or natural blend of emotions and skills. On one hand, trained EMS professionals are always primed and ready to go, feel comfortable moving rapidly, acting quickly, and thinking on their feet. On the other hand, skilled outdoors people are always eager and willing to travel into the backcountry but understand the necessity of thorough preparedness. This attitude ensures not only that each team is prepared but also that each individual is prepared. The team must be organized from a leadership perspective and know where it is headed, what injuries to anticipate, and how weather will affect the rescue. The team counts on each individual member being physically and mentally prepared. This difficult task requires recognition of the differences between short-term and long-term care during a rescue so that a safe and successful extended care rescue can be achieved. Box 31-2 outlines key skills that training needs to include.

Box 31-2. Rescue Personnel and Training in the United States

1. Most technical rescue personnel in the United States are climbers or skiers who have added wilderness rescue techniques and medical training to their skills.
2. Currently there are about 800,000 certified and licensed EMT-Basics, EMT-Intermediates, and EMT-Paramedics in the United States.
3. A growing number of wilderness EMTs are trained in the skills of extended victim care in the backcountry environment. There are a growing number of courses available for training.
4. Key skill elements of medical training for wilderness medical and rescue training include the following:
   a. Thorough victim assessment skills and monitoring
   b. Technical skills and the authority to perform the following:
      (1) Airway management, to include endotracheal intubation, LMA, Combitube, and other advanced airway skills
      (2) Shock management to include intravenous therapy
      (3) Use of the pneumatic antishock garment (MAST and PASG; no longer recommended for general treatment of shock, so use in special circumstances only)
      (4) Oxygen administration and its limitations
      (5) Use of appropriate medications:
         (a) Epinephrine for anaphylactic reactions
         (b) Antibiotics for major open or penetrating wounds, protruding intestines, compound fractures
         (c) Acetazolamide, steroids, nifedipine, and furosemide for acute high-altitude problems
         (d) Pain medications for musculoskeletal trauma
         (e) Albuterol for bronchodilation
         (f) Aspirin and nitroglycerin for chest pain
      (6) Field rewarming techniques
      (7) Field reduction of angulated fractures and dislocations
   c. Victim packaging and transportation skills
5. Key skill elements of technical training for rescue personnel in the United States include the following:
   a. Appropriate climbing skills for terrain (rock, ice, snow, glacier)
   b. Radio communication skills and protocols
   c. Helicopter and fixed-wing protocols
   d. Training and expertise in using the Incident Command System in field protocols

SEQUENCE OF EVENTS IN BACKCOUNTRY RESCUE

The principles and standards of a wilderness or mountain rescue (extended care rescue), including organization, specialized skills and knowledge, and essential components of the team, can best be illustrated by reviewing the sequence of events during a typical backcountry rescue in North America (Box 31-3).

Occurrence of the Critical Event

The critical event occurs when an individual participating in an activity away from immediate help is suddenly stricken by injury or illness. The key factor is immobilization. The fact that the injured or ill person cannot self-evacuate or move to seek shelter or stay warm results in the need for a rescue. Once the victim or others in the party realize this, the need to seek help becomes obvious.

Box 31-3. Sequence of Events in Backcountry Rescue

1. The critical event occurs: an injury or illness that requires assistance and evacuation.
2. A decision is made to "get help," and someone goes for help.
3. The emergency medical system is notified of the emergency.
4. The emergency medical system is activated, or "dispatched."
5. Eventually, the "extended rescue team" is notified and mobilized.
6. The rescue team assembles and organizes, then leaves the trailhead (may be preceded by a "hasty team").
7. The team locates the victim.
8. The team provides appropriate "extended emergency care."
9. The team organizes and evacuates the victim to the appropriate facility.
10. The team returns to base, is debriefed, and prepares for the next rescue.

Making the Decision to Get Help

Before anyone leaves to seek assistance, the victim's companions should perform a physical examination, record vital signs, determine the level of consciousness, and provide appropriate emergency care, which may entail moving the victim into protective shelter. Victim information should be summarized in a note that accompanies the individuals going for help. A map depicting the victim's exact location and a list of the other party members, noting their level of preparedness to endure the environmental conditions, should be included. The individuals going for help should carry appropriate provisions. To prepare information adequately generally takes 30 minutes to 1 hour. However, thorough preparation rarely occurs. Often, someone suddenly yells, "I'll get help" and disappears, running down the trail with sparse vital knowledge.

With the improvements and availability of communications technology, such as cellular phones and global positioning systems, backcountry adventurers may have more rapid access to the EMS system from the mountains. Whether this will increase the number of inappropriate callouts remains to be determined. Recent reports indicate that reliance on fallible technology may be responsible for outdoors people using poor judgment in terms of trip planning. Assuming that help is just a phone call away, hikers are taking more risks.

Notifying the Emergency Medical System

Eventually, the messenger notifies someone in authority that an emergency has occurred and help is needed. Usually, the request is made to a central 911 system. If no central service is available, a local dispatch agency is notified. The agency contacts the closest emergency medical service, which can be a rescue squad, ambulance corps, fire department, or first response team.

Activating the Emergency Medical System

Notification of an emergency usually occurs by means of pagers worn by individual members. An alert tone is followed by an oral message describing the emergency, its location, and type of response required.

At this point, a wide variety of events can occur, involving agencies within and outside the EMS system. Even in areas of the United States with well-organized extended care rescue teams, the team may be notified last. Ideally, it should be notified immediately, but all too often this is not the case. Instead, local agency responders are notified and rush around trying to determine how quickly they can reach the victim.

Notifying and Mobilizing the Extended Rescue Team

The first step is to notify team members. In many parts of the United States, organized and coordinated extended rescue teams do not exist, so a "team" is created of relatively untrained volunteers willing to hike in and assist. The task of further organizing and coordinating the rescue effort generally falls on the shoulders of a local rescue squad, fire service, or police department, which may or may not be willing and prepared to manage and execute an extended or technical rescue.

In the parts of the United States where backcountry use is common and backcountry accidents occur regularly, extended care rescue teams have generally evolved from local EMS squads with skilled outdoor enthusiasts. Some teams offering local search and rescue capabilities may be coordinated locally (such as the Appalachian Mountain Club [AMC], Stonehearth Open Learning Opportunities [SOLO], and Mountain Rescue Service in the White Mountains of New Hampshire); other teams may be part of a nationwide system responding to incidents throughout the country and be coordinated on a regional or national level (such as the National Cave Rescue Commission), or the future may involve FEMA, or even the Department of Homeland Security. Coordination of extended care rescue teams may also come under the jurisdiction of a law enforcement body, such as state conservation officers (e.g., New Hampshire Fish and Game), sheriff's department (e.g., the Los Angeles County Sheriff in California), or a statewide coordinating system (e.g., the Pennsylvania Search and Rescue Council). Organized teams can be quite sophisticated in their dispatching function so that all members can be notified simultaneously, or they may use a more "low-tech" telephone tree to call out members.

Assembling and Organizing the Rescue Team

Once members are notified, they assemble at a common location (rescue station) to organize the rescue effort. The first task is to define the type of rescue to establish equipment needs. Estimating the time it will take to effect the rescue and assessing the need for other agency involvement and assistance are also primary tasks. The questions to be answered and the variables to be considered may include the following:

1. Time of day: will this be a night rescue?
2. Weather: what are the current weather conditions at the rescue location and what is the forecast?
3. When did the accident occur?
4. What are the supposed injuries?
5. How many victims are there?
6. How many people are in the party?
7. How well prepared are they?
8. Does anyone in the party have medical expertise?
9. Do we know the exact location, or is this a search and rescue?

10. Is a "hasty team" needed? Has it left for the scene yet?
11. Is each of the team members prepared? Does each have personal equipment, a bivouac kit, head lamp, food, and water?
12. Is each member trained and skilled in this particular type of rescue?
13. Who is on the medical team?
14. Who is on the evacuation team?
15. Is the team equipment organized and divided up?
16. How urgent is the situation? Is a helicopter required? Is one available?
17. Are the weather conditions appropriate for an air rescue?
18. Will multiple agencies be involved? If so, are radio frequencies coordinated?

Once the team is assembled and all pertinent issues have been addressed satisfactorily, the team is transported to the trailhead (launch point) to begin the search.

Commonly, a hasty team starts out ahead of the main team. Once the hasty team has enough information to locate the victim, team members travel as lightly as possible, with only enough gear to ensure their own safety and equip them to manage the victim's primary injuries. The goal is to reach the victim as quickly as is reasonably possible and to deliver primary care, then apprise the rest of the team of the victim's condition, equipment needs, and environmental concerns.

Locating the Victim

How long it takes to locate the victim varies tremendously, depending on distance, terrain, weather conditions, mode of transportation, and whether the victim's exact location is known. A general rule of thumb for a team responding on foot is that it will take 1 hour for each 1 mile through the backcountry. If a search is involved, all bets are off. A search and rescue effort can involve many agencies, individuals, and days (see Chapter 32).

Providing Appropriate Extended Emergency Care

Once the victim is located, appropriate medical care can be provided. The rescue team should ensure its own safety; wet clothes should be replaced with warm, dry clothing, and members should check for emerging problems within their group. While the medical team cares for the victim, the evacuation team secures shelter, prepares warm drinks, establishes and maintains communications, and plans and organizes the evacuation.

Companions with the victim may have been affected by the environment while waiting for the rescue team to arrive and require assistance. They may need to be assessed and treated for hypothermia, frostbite, heatstroke, heat exhaustion, or dehydration.

Regardless of what transpired before the medical team arrived, a complete victim assessment is essential. Do not assume that all the injuries have been found or that all medical conditions have been managed properly (Box 31-4).

In the extended care environment, the victim must be monitored for changing conditions that indicate an underlying problem. Awareness of environmental emergencies is particularly important, with constant care to prevent hypothermia, frostbite, heatstroke, heat exhaustion, and dehydration. To do this, it is necessary to monitor the victim and write a new SOAP note or "reSOAP" the patient every 15 minutes:

Box 31-4. Victim Assessment

PRIMARY SURVEY: LOCATING AND TREATING LIFE-THREATENING PROBLEMS

A—Airway Management
- Is the airway open?
- Is the airway going to stay open?

B—Breathing
- Is air moving in and out?
- Is the airway quiet or silent?
- Is breathing effortless?
- Is the respiratory system intact?
- Is breathing adequate to support life?

C—Circulation
- Is there a pulse?
- Is bleeding well controlled?
- Is capillary refill normal (less than 2 seconds)?
- Is circulation adequate to support life?

D—Disability
- Conscious vs. unconscious
- Level of consciousness—awake, verbal, painful, unconscious (AVPU) or Glasgow Coma Scale
- Cervical spine stabilization

E—Environment
- Internal vs. external?
- Is the victim warm and dry?
- Protected from the cold ground?
- Protected from the elements?

SECONDARY SURVEY: WHAT IS WRONG AND HOW SERIOUS IS IT?
- Vital signs: Indicate the condition of the victim
  - Respiratory rate (RR) and effort
  - Pulse rate (PR) and character
  - Blood pressure (BP): systolic/diastolic
  - Level of consciousness (LOC): AVPU or Glasgow Coma Scale
  - Tissue perfusion (TP): skin color, temperature, and moisture
  - Capillary refill (less than 2 sec)
- Victim examination: Head-to-toe examination to locate injuries
- AMPLE history:
  - Allergies
  - Medicines
  - Past medical history
  - Last in and out: food/drink & voiding
  - Events leading up
- SOAP Note: To record and organize victim data
  - Subjective: age, gender, mechanism of injury, chief complaint
  - Objective: vital signs, victim examination, AMPLE history
  - Assessment: problem list
  - Plan: plan for each problem

"ReSOAPing" the patient:

Subjective: Is the victim comfortable, too hot, too cold, hungry, thirsty, or in need of urination or defecation?
Objective:
Vital signs—are they stable? Record these.
Victim examination—recheck all dressings, bandages, and splints; are they still controlling bleeding? Are they too tight or too loose? (Swelling limbs can cause bandages or splints to impede circulation, resulting in ischemic injuries or worsening frostbite or snakebite.)
Assessment: Has the initial assessment changed?
Plan: Is the rate of evacuation still the same?

Evacuating the Victim to the Appropriate Facility

While providing emergency care, part of the team is designated as the evacuation team. This group has evaluated the various options for evacuation. To properly evaluate the situation, the first information they need is provided by the medical team leader, who needs to know the status of the victim to establish the pace. If the victim's condition is stable, time is less important; if the victim's condition is critical, time is critical.

The evacuation team must explore different options. If speed is a consideration, weather conditions are reviewed, and the availability of a helicopter-assisted rescue is determined. If a helicopter is not an option, the fastest route out is established. If time or speed is not critical, the safest means of evacuation that is easiest on the victim and rescuers is defined. A general rule for the duration of an evacuation is that it will take 1 to 2 hours for every 1 mile to be covered, requiring six well-rested litter bearers for every 1 mile. Thus, a 4-mile carryout will require a 24-member litter team and can take 4 to 8 hours to complete. Eventually, the team reaches a trailhead, and the victim is transferred to an ambulance for transport to a hospital emergency facility.

Returning to Base

The team returns to base to reorganize equipment in preparation for the next extended rescue, and to debrief. Because people are exhausted and hungry, the debriefing session is often canceled. However, establishing a mechanism to debrief the rescue effort is imperative so that team members can learn from the shared experience, discuss victim care, and work through problems. Whenever several different emergency organizations with disparate rescue and emergency personnel combine to perform a complex rescue, there may be tension, conflicting egos, and concerns about the medical care provided or evacuation plan used. These problems deserve to be discussed and managed in real time as expediently as possible so that teams will cooperate successfully in the future, improve their performance, and provide the best possible patient care on the next rescue. This process minimizes the burnout syndrome that can occur with volunteer teams.

TEAM ORGANIZATION AND FUNCTION

The organization of an extended care rescue team is based both on training of individuals and on type of rescue. The structures of teams can vary from loosely knit groups of friends with no leadership hierarchy to paramilitary organizations with rigid leadership roles.

Box 31-5. The Ten Essentials

1. Attitude
   Positive belief that you can make things better; the will to survive
2. Fuel to burn: food
   High-carbohydrate foods that require no preparation
   High-carbohydrate foods that can be made into a drink
3. Quench your thirst: water
   A minimum of 2 L/day to survive, 4 L/day to thrive
   Up to 3 L/hr if active
   Ability to make more disinfected water
4. Stay warm and dry: clothing
   Warm clothing that retains heat even if wet
   Waterproof raingear, top and bottom
5. Get dry: shelter
   Ability to improvise shelter or bivouac
   A bivouac ("bivy") kit (see Box 31-6)
6. Get warm: fire
   Ability to warm water (stove, candle, fire)
   Ability to build a fire (waterproof matches and tinder)
   Ability to make kindling or tinder (folding knife)
7. Know where you are going: navigation
   Map and compass skills and route-finding skills
   Ability to move about at night (headlamp)
8. Know the environment: weather
   Basic understanding of weather patterns
   Knowledge of how to react in severe weather; lightning
9. Getting help: signaling
   Whistle, preferably plastic
10. Providing help: first aid kit
    Basic small personal trauma kit

Team members require personal knowledge, experience, and expertise in the particular aspect of extended care and rescue in which they will participate, as well as knowledge and expertise in the principles of extended emergency care, extended rescue techniques, and technical rescue skills.

Personal Knowledge, Experience, and Expertise

Individuals who want to be part of an extended rescue team need to acquire outdoor skills before they become part of a rescue team. Every member must have extensive knowledge of likely environmental emergencies: hypothermia, frostbite, heat syndromes, snakebite, dehydration, lightning strike, and so forth. Each must understand general principles of weather behavior. Rescuers need to be comfortable with route finding, map and compass, personal preparedness, and bivouac and survival skills. The knowledge, skills, and equipment that a skilled outdoors person should possess are often referred to as "the ten essentials" (Boxes 31-5 and 31-6). The same skills, knowledge, and equipment commonly used by the outdoor enthusiast are essential on a mountain rescue.

Extended Rescue Techniques and Skills

Specific skills and techniques applicable to a particular situation include those of search and rescue, vertical and technical rock

Box 31-6. Bivouac Kit

Two large garbage bags (emergency shelter or raingear)
10- × 10-foot sheet of plastic and 100 feet of parachute cord (shelter)
Emergency space blanket (shelter, ground cloth)
Stocking cap (warmth)
Spare socks (warmth; can act as spare mittens)
Metal cup (to warm liquids)
Gelatin (to make a drink)
Two plumber's candles (to warm water or start fire)
Waterproof matches or lighter
Knife
Compass
Whistle
All these items fit neatly into a small stuff sack that is 6 × 6 inches (15.2 × 15.2 cm) and weighs less than 1 pound (2.2 kg) when filled.

climbing, and white-water navigation. Snow or winter camping or avalanche rescue may be required, depending on the environment. Extended rescue teams should require their members to have, at a minimum, the working knowledge and equipment in Box 31-7.

Knowledge is acquired over time. Specific medical, rescue, and technical skills are obtained and retained through courses, continuous training, and refresher programs. The Appendix at the conclusion of this chapter provides a list of schools, institutes, and organizations that are involved in wilderness medicine and mountaineering research, standards development, and training programs.

Wilderness and Mountain Rescue Team Organization

Organization of wilderness and mountain rescue teams is where the greatest diversity exists because no universal standard has been established. Teams vary from local mountain rescue teams with extreme skills and qualifications for providing mountain rescue care to informal collections of friends without leadership. Other, more "professional," teams are operated under the jurisdiction of law enforcement agencies with paramilitary hierarchy and leadership. This diversity is particularly noticeable in the United States because most teams are composed of volunteers who are not reimbursed for their rescue efforts.

In Europe, mountain rescue teams are professional and employ full-time personnel. They charge for rescue efforts, with the fees providing money for personnel, equipment, helicopters, technical gear, and ongoing training. As with any "profession," standards have evolved. As a result, there are more standards in Europe than in the United States. Still, there is variation across Europe from country to country, especially in leadership and organization.

In many parts of the world, especially remote and wild areas, organized and available rescue teams do not exist. If someone is in need of help, the expedition team necessarily becomes the rescue team.

TRAINING OF WILDERNESS EMERGENCY MEDICAL TECHNICIANS

The best way to develop an appreciation for the vast difference between what is required of the traditional (urban) EMT and what is required of the extended care or wilderness emergency medical technician (WEMT) is to compare their respective course curriculums.

The Department of Transportation (DOT) is responsible for developing and updating the EMT-Basic, EMT-Intermediate, and EMT-Paramedic curricula in the United States. These curricula are considered the minimum national standard for EMT students to qualify for the National Registry or an individual state practical and written examination. Passage of such an examination enables a student to become certified as a National Registry or state EMT-Basic, EMT-Intermediate, or EMT-Paramedic.

A national standard for WEMT curricula does not exist. Despite the lack of a DOT-like standard, there are several similar curricula for wilderness emergency care at the EMT level. Based on the recommendations of the Wilderness Medical Society and other groups that address the issues of wilderness prehospital emergency medicine, these curricula adhere to the same principles of long-term patient care, which can be used for comparison with the standard DOT curriculum.

A WEMT course typically contains all of the material in the DOT EMT course curriculum plus what is necessary to acquire the skills attendant to long-term wilderness emergency care. Typical EMT courses are about 100 hours, with 10 additional hours of emergency department observation time. The WEMT module carries an additional 48 to 80 hours of training. There are EMT Wilderness Modules available that are postgraduate courses designed to train EMTs in long-term patient care.

A typical WEMT course outline appears in Box 31-8. The topics in boldface are unique to WEMT programs; the other topics are those required in a DOT EMT course. Hours per topic illustrate the time required for both EMT and WEMT training. This outline is arranged in the current DOT EMT recommended format, with the WEMT material added on a per topic basis; topics are not necessarily listed in the order that they would be taught for a particular course. An explanation follows of the extended emergency medical care material that must be learned by WEMTs.

Introduction to Emergency Care

"Wilderness versus urban emergency care" is an introductory presentation to illustrate the differences between urban (golden hour) emergency care and extended, or wilderness, emergency care. For WEMTs, it will be necessary in certain instances to learn two different modalities of therapy, one for short-term (less than 1 hour) care and one for long-term or extended (several hours to days) care.

"Backcountry rescue gear inspection" is a hands-on review of gear for the outdoor practice sessions and backcountry mock rescues. The course staff must inspect the participants' boots, clothing, raingear, and rescue equipment to determine their adequacy for the particular environment in which they will be deployed. Inspecting equipment not only ensures the safety of each individual in the course but also teaches a standard for

Box 31-7. Knowledge, Skills, and Equipment for Extended Rescue Teams

MOUNTAINEERING SKILLS

- Understanding fabrics and clothing systems and their seasonal variations (see Chapter 70)
- Fabrics and fibers
- Layering techniques
- Vapor barrier systems
- Waterproof fabrics, raingear systems
- Footgear
- Personal protection equipment:
  - Helmets
  - Harnesses
  - Gloves
  - Goggles, sunglasses
  - Hearing protection
- Backcountry equipment
- Internal or external frame packs and soft packs
- Shelter (natural and human-made)
- Specialty equipment: snow shoes, crampons, ice axes, stoves, skis
- Backcountry travel
- Route finding
- Map and compass: map reading, dead reckoning, types of maps; compass reading, bearings, magnetic vs. true bearing, triangulation, global positioning systems
- Survival skills: the ten essentials
- Shelter and warmth; emergency bivouac ("bivy") kits
- Food, water
- Understanding how backcountry travel and rescue vary with the seasons
- Understanding how backcountry travel and rescue vary with different environments:
  - Alpine
  - Desert
  - Forest
  - Water (swamp, river, lake, ocean)
  - Tropics
  - High altitude
- Low-impact camping and rescue work
- Basics of weather and weather forecasting
- Principles of barometric pressure
- Clouds and their significance in weather forecasting
- Prevailing weather patterns in the rescue area
- Personal fitness
- Physical conditioning
- Nutrition and hydration requirements for different activities

MOUNTAIN AND EXTENDED EMERGENCY MEDICAL SKILLS

- Emergency medical training should be at a minimal level of first responder or higher (emergency medical technician, paramedic, registered nurse, nurse practitioner, physician's assistant, or physician). Regardless of the level, training must include specific information on wilderness and extended emergency care procedures.

TOPICS OF EXTENDED CARE TRAINING AND PRINCIPLES SHOULD INCLUDE THE FOLLOWING:

- Patient assessment system
- Cardiopulmonary resuscitation
- Airway management, including endotracheal intubation and needle decompression for tension pneumothorax
- Shock and control of bleeding, including the use of intravenous (IV) therapy for fluid resuscitation
- Long-term wound care and prevention of infection
- Musculoskeletal injury management, including specific information on diagnosis and long-term management of the following:
  - Sprains and strains
  - Fractures, including how to reduce or realign angulated fractures
  - Diagnosis and reduction of dislocations
  - Management of compound fractures
- Management of chest injuries, including decompression of a tension pneumothorax with a needle thoracostomy
- Spinal cord injury diagnosis and management
- Head injury, including recognition and management of increasing intracranial pressure
- Management of environmental emergencies
- Hypothermia and frostbite, including the use of IV fluids
- Heatstroke and heat exhaustion, including the use of IV fluids
- Dehydration and nutrition, acute and during evacuation
- Lightning injuries
- Animal attacks, insect bites, and reptile and marine envenomations, including anaphylactic reactions and the use of epinephrine and antihistamines
- Contact dermatitis, such as poison ivy, oak, and sumac
- Sunburn and snowblindness
- High-attitude injuries, including acute mountain sickness, pulmonary edema, cerebral edema
- Near drowning
- Diagnosis and management of acute medical emergencies:
  - Chest pain (myocardial infarction, angina, costochondritis)
  - Shortness of breath (asthma, anaphylaxis, pneumothorax)
  - Seizures and cerebrovascular accidents
  - Acute abdomen (peritonitis, constipation, diarrhea)
  - Pyelonephritis
  - Septic shock
- Victim lifting and handling techniques
- Improvising techniques: "emergency medicine barehanded" (see Chapter 19)
- Training in the use of the Incident Command System
- Blood-borne pathogens and infectious disease prevention
- Monitoring of bodily functions (hunger, thirst, and bodily waste management)
- General understanding and appreciation for the difference between urban (short-term) and wilderness (long-term) emergency care

Continued

Box 31-7. Knowledge, Skills, and Equipment for Extended Rescue Teams—cont'd

MOUNTAIN AND EXTENDED RESCUE SKILLS
- Understanding equipment used in wilderness search and rescue operations, including maintenance and care
  - Ropes, slings, carabiners, harnesses, helmets
  - Litters, litter harnesses, haul systems
  - Litter patient packaging equipment
- Basic radio communications
- Care and maintenance of communications equipment
- Procedures and protocols
- Basic helicopter operations and procedures
  - Approach to a helicopter
  - Safety considerations
  - Landing zones
  - Haul techniques
- Interagency relations
- Basic understanding of search procedures
- Basic understanding of rescue procedures
- Basic understanding of Incident Command System and its use in search and rescue management
- Basic rope-handling and knot-tying skills
- How to care for and handle ropes
- Rappelling, belaying, and braking techniques
- Knots
  - Figure-8
  - Figure-8 follow-through
  - Figure-8 on a bight
  - Double figure-8
  - Double fisherman's Prusik
  - Tensionless hitch (round turn and two half hitches)
  - Water knot
  - Half hitch and full hitch
  - Bowline
  - Alpine butterfly
  - Specific rescue training
- Water search
- White-water
- Avalanche
- Technical or vertical (rock)
- Cave

LEADERSHIP
- Leadership and "followship" training
- Ability to use the Incident Command System

preparedness, awareness, and attention to detail that is critical for wilderness travel and emergency care.

"Medical-legal issues" is usually offered early in a course so that the participants are aware of the legal concerns surrounding practicing medicine as EMTs and WEMTs. WEMTs need to be aware of protocols that exist where they will become licensed.

"The human animal—our natural physiologic limits" course is an overview of how humans fit into the natural environment, including their daily nutritional and fluid requirements and natural limitations in different settings. The WEMT must understand physiologic limits, such as those of endurance, temperature, and altitude, and the consequences when these limits are exceeded.

Patient Assessment Systems

"Patient assessment in the wilderness and practice" takes the newly learned skills of patient assessment and adapts them to the backcountry. The WEMT must be knowledgeable and skillful in wilderness patient assessment, a step-by-step approach to the first 5 minutes of scene safety and patient care. The WEMT will develop an awareness of potential life-threatening dangers in the environment, how to ensure personal safety and the safety of others, how to approach a victim safely, how to perform primary and secondary surveys to determine the extent and severity of injuries, and what impact the environment might have on the victim.

Airways, Oxygen, and Mechanical Aids to Breathing

"Airways, oxygen, cardiopulmonary resuscitation, and mechanical aids to breathing in the wilderness environment—uses and limitations" addresses one of the most important lifesaving and life-maintaining skills in emergency medicine: the ability to establish and maintain a patent airway. Unfortunately, most EMTs are not provided with the training and tools they need to properly maintain an open airway in an unconscious victim. Failure to be able to maintain an open airway by the recovery position, endotracheal intubation, use of laryngeal mask airway (LMA), or Combitube can be disastrous.

Endotracheal intubation is commonly used by EMT-Intermediates and EMT-Paramedics and other advanced life support (ALS) personnel in cardiac arrest settings and for unconscious, unresponsive victims. In the extended care environment, the use of intubation by endotracheal tube, LMA, or Combitube in a cardiac arrest situation is not nearly as common as it is for the normothermic, unconscious, and unresponsive person, who has probably suffered head trauma. In this situation, without the ability to perform intubation, the only way to maintain a patent airway while lifting, moving, and transporting a victim in a litter is to place the victim on his or her side in the recovery position. In the recovery position, gravity pulls the tongue forward and allows secretions to drain from the mouth.

Oropharyngeal, nasopharyngeal, and tongue-pinning techniques may temporarily keep the tongue from occluding the airway, but they are ineffective in preventing vomitus, blood, or saliva from entering the airway. Also, during evacuation in a litter, constant monitoring of a victim's airway is virtually impossible, which makes proper airway management and monitoring of paramount importance. The WEMT should know how to establish and maintain a patent airway, including the use of positioning and advanced airways.

"Oxygen administration" presents the use of supplemental oxygen, for which both EMTs and WEMTs follow the same general guidelines. Even though oxygen is important to prehospital care, its use has significant logistic limitations in the backcountry. The WEMT must realize that carrying large quantities of oxygen into the backcountry is impossible. Small D and E cylinders can be carried, but each provides high-flow oxygen

Box 31-8. EMT and WEMT Course Curricula and Hours per Topic*

1. Introduction to emergency care
   Wilderness vs. urban emergency care (1 hour)
   Backcountry rescue gear inspection (1 hour)
   Medical-legal issues (1 hour)
   Blood-borne pathogens
   Overview of human systems—anatomy and physiology (2 hours)
   The human animal—our natural physiologic limits (2 hours)
2. Patient assessment systems
   Primary survey—ABCs (1 hour)
   Secondary survey (1 hour)
   Patient assessment practice (2 hours)
   Patient assessment in the wilderness and practice (3 hours)
3. Cardiopulmonary resuscitation (8 hours)
   Mannequin practice and certification (8 hours)
   Cardiopulmonary resuscitation (CPR) teaching, practice, and testing to American Heart Association standards
4. Airways, oxygen, and mechanical aids to breathing (3 hours)
   Airways, oxygen, CPR, and mechanical aids to breathing in the wilderness environment—uses and limitations (6 hours)
   Airways: oropharyngeal, nasopharyngeal, endotracheal intubation, LMA
   Oxygen administration
   Suction techniques
5. Bleeding and shock (3 hours)
   Shock, intravenous (IV) fluids, and long-term patient care (4 hours)
   Practice starting IV infusions and fluid administration (4 hours)
   Use of pneumatic antishock garment (PASG) (3 hours)
   Use of PASG in the wilderness (1 hour)
6. Soft tissue injuries (3 hours)
   Long-term wound care (1 hour)
7. Principles of musculoskeletal care
   Fractures of the upper extremities (3 hours)
   Fractures of the pelvis, hip, and lower extremities (3 hours)
   Fracture laboratory—practice in assessment and management (3 hours)
   Musculoskeletal trauma management in the wilderness (3 hours)
8. Injuries of the head, face, eye, neck, and spine (3 hours)
   Practical laboratory: spinal cord injury management (SCIM) (3 hours)
   Head trauma, increasing intracranial pressure (1 hour)
   SCIM: Long-term care and improvising (l hour)
9. Injuries to the chest, abdomen, and genitalia (3 hours)
   Chest trauma in the wilderness (3 hours)
10. Medical emergencies I (3 hours)
    Poisoning, bites and stings, heart attack, stroke, dyspnea
    Medical emergencies II (3 hours)
    Diabetes, acute abdomen, communicable disease, seizure, substance abuse, and pediatric emergencies
    Medical emergencies in the wilderness (3 hours)
11. Emergency childbirth (3 hours)
12. Burns and hazardous materials (3 hours)
    Long-term care of burns (1 hour)
13. Environmental emergencies (3 hours)
    Hypothermia, frostbite, immersion foot (4 hours)
    Heatstroke, heat exhaustion, dehydration (4 hours)
    Drowning (2 hours)
    High-altitude emergencies (2 hours)
    Barotrauma (2 hours)
    Animals that bite and sting (2 hours)
    Plants—contact dermatitis (2 hours)
    Marine animals that bite and sting (2 hours)
14. Psychological aspects of emergency care (3 hours)
15. Lifting and moving patients (3 hours)
    Use of Stokes litters and improvising litters (3 hours)
    Principles of backcountry evacuation (4 hours)
    Search and rescue organization and execution (4 hours)
    Wilderness mock rescue with or without overnight (8 to 12 hours)
16. Principles of vehicle extrication (4 hours)
    Practice laboratory (3–8 hours)
17. Leadership and "followship" skills
    The Incident Command System
18. Ambulance operations I (3 hours)
    Ambulance operations II (3 hours)
    Helicopter-assisted rescues (3 hours)
19. Review (3–6 hours)
    Testing—written and practical examinations (16-20 hours)
20. Emergency department observation time (10 hours)

*Topics in boldface are peculiar to WEMT programs, whereas the other topics are required topics covered in a DOT EMT course.

for only 20 to 30 minutes. Oxygen is a compressed gas in a tank, so as it expands, cools dramatically, and may contribute to hypothermia. To prevent this, the gas should be preheated by wrapping the oxygen tubing around a chemical heat pack during administration.

"Suction techniques" presents the use of suction devices to clear the airway, which is similar for EMTs and WEMTs. Hand-operated, as distinct from battery-operated, suction devices are usually used in extended care scenarios.

Bleeding and Shock

"Shock, intravenous (IV) fluids, and long-term patient care" and "Practice starting IV infusions and fluid administration" provide information about the care of victims in shock or suffering from environmental emergencies that cause volume depletion, such as heatstroke or hypothermia.

For urban management of shock, the essential component is recognition. Once shock is recognized, the victim can be rapidly transported to an emergency department or intercepted by

paramedics for definitive care, namely, fluid resuscitation. In the extended care environment, WEMTs must be able to manage the volume-contracted patient by providing appropriate definitive care and fluid resuscitation. During extended evacuations, WEMTs should know how to administer IV fluids to stabilize hypovolemia. This includes starting a peripheral IV line, maintaining catheter placement, using proper fluids, and prewarming and maintaining warm solutions before and during administration.

"Use of pneumatic antishock garments (PASG) in the wilderness" discusses the use of these garments for victims of a pelvic fracture. The practice of treating shock using PASG is no longer recommended. As long as an IV line can be established and maintained, fluid administration is the definitive method for managing shock. The WEMT must be aware that the PASG has other uses. In the extended care situation, the PASG may be invaluable as a splint to stabilize a fractured pelvis, and thus contribute to control of internal bleeding. It may also be used as an improvised air splint for a fractured femur. An added benefit is that the PASG may facilitate a more comfortable and well-padded ride for the victim in the litter during evacuation. However, WEMTs must recognize the limitations of a PASG. The primary drawback in the backcountry is the potential for cold injury. Once the apparatus is inflated, the decrease in peripheral circulation greatly increases the risk for cold injuries or frostbite to the lower extremities. This can be prevented by properly packing the feet with chemical heat packs and adequately insulating the lower extremities in the litter. Careful monitoring of the lower extremities every 15 minutes is essential.

Soft Tissue Injuries

"Long-term wound care" covers proper wound management once bleeding has been controlled, and further care if more than 12 hours will be required to bring the victim to definitive care. The principles of long-term wound care are to stabilize the wound and prevent and control infection.

To prevent infection, the WEMT must know how to sterilize or disinfect fluid and how to properly débride and rinse out a contaminated wound. Once the wound is cleaned and débrided, the edges can be approximated but not tightly closed because this may increase the risk of abscess formation and a life-threatening infection. Training in suturing techniques to close wounds is not currently recommended because suturing is usually for cosmetic reasons and can almost always be done later, lessening the risk for infection by tightly closing the wound.

Even the most fastidiously cleaned wound can still become infected, particularly in a remote setting, because of constant exposure to microbes. Recognition of wound infections and appropriate management are important. The WEMT must learn to use specific antibiotics in extended care settings of greater than 3 days and for prophylaxis with grossly contaminated wounds and compound fractures. Antibiotic therapy is not controversial because various safe broad-spectrum antibiotics can cover most wound infections with minimal risk for a severe allergic reaction. In certain circumstances, the benefits of antibiotic administration clearly outweigh the risks.

Principles of Musculoskeletal Care

"Musculoskeletal trauma management in the wilderness" presents the treatment of injuries. In an urban setting, the primary concern with fracture and dislocation care is that the injury site be splinted properly to prevent further injury. In the extended care environment, the primary concern is to maintain proper circulation distal to the site of the injury. This may require straightening an angulated fracture or reducing a dislocation.

When an angulated fracture occurs, distal circulation can be impaired, putting the soft tissues at considerable risk for ischemic injury or frostbite. Under normal circumstances, it would take hours for moderate ischemia to cause irreparable soft tissue injury, but in the backcountry, prolonged time under hostile weather conditions frequently occurs, which decreases the amount of heat and oxygen being transferred to the extremity.

Knowing how to properly straighten out an angulated fracture significantly decreases the risk for secondary ischemic injury and frostbite, controls bleeding at the fracture site, and diminishes pain. It is much easier to splint and stabilize a fracture in proper position if it is in anatomic position than if it is angulated.

About 3 additional hours of training are needed to teach a WEMT how to straighten an angulated fracture and reduce dislocations. Without an x-ray, it is impossible to see the exact positioning of bone fragments or disarticulated joints, making it difficult to know exactly how to manipulate the bone. The concern is that if a jagged bone end is moved improperly, secondary injury might occur because part of a neurovascular bundle might be severed, a fascial sheath surrounding a muscle might be cut, or the bone ends might erupt through the skin. Fortunately, all these structures are richly endowed with pain receptors. If the sharp end of a bone fragment begins to impinge, it causes a dramatic increase in pain at the site. A commonly used technique is to straighten the angulated site slowly while maintaining constant gentle traction. With each 1 to 2 cm of movement, the victim is asked if the new position is better or worse (causes less or more pain). If the pain diminishes with movement, the reduction is proceeding properly; if pain increases, all movement is stopped, and the extremity is returned to the previous position of improvement. While still under gentle traction, the extremity is repositioned, and another attempt at reduction is made.

As long as nothing is forced and movement is achieved slowly under gentle traction, angulated fractures can be easily realigned and dislocations reduced without the need for pain medication or any risk for further injury.

Musculoskeletal injuries in the long-term care setting must be carefully monitored. It is essential to re-inspect the injury site at reasonable intervals for circulation, sensation, and motion. Fracture sites swell; as a result, even the best splint can act as an inadvertent tourniquet. Immobilized extremities cool because of lack of activity and impaired circulation, also increasing the risk for ischemic injury or frostbite.

Injuries of the Head, Face, Eye, Neck, and Spine

"Head trauma, increasing intracranial pressure" addresses one of the leading causes of death from backcountry accidents. Many who die of head trauma in the wilderness would have survived in an urban setting because of rapid access to definitive care. The WEMT must be able to recognize a potentially serious head injury long before the victim is at risk for brainstem herniation.

In the extended care environment, there are few situations when the team should hurry. One such situation is the presence

of significant head trauma, for which the only appropriate care may be rapid evacuation to a facility where the victim can be treated by a neurosurgeon.

It is important to establish and monitor the level of consciousness. The AVPU (awake, verbal, pain, unresponsive) scale is used. Within the primary survey, an initial evaluation of disability or neurologic status is made. After that, level of consciousness is reevaluated every 15 minutes to observe in particular for any evidence of increasing intracranial pressure.

Injuries to the Chest, Abdomen, and Genitalia

Chest trauma can result in a pneumothorax that can evolve into a tension pneumothorax. WEMTs need to be taught how to inspect, palpate, percuss, and auscultate the chest to identify significant injuries. It is not difficult to train an individual to detect breath sounds, determine the presence of a pneumothorax, and monitor a pneumothorax for its development into a tension pneumothorax. Unlike increasing intracranial pressure, for which there is little to do but evacuate the victim, a tension pneumothorax can be relieved, increasing the chance of survival. The easiest and most effective technique a WEMT may learn is needle thoracostomy in the fifth intercostal space in the midaxillary line.

Medical Emergencies

Diagnosing medical emergencies in the wilderness requires the WEMTs to be aware of essential signs and symptoms.

Environmental Emergencies

The typical EMT course includes 3 to 6 hours of training in management of environmental emergencies. A WEMT course will have a minimum of 22 hours of additional training in environmental emergencies.

"Hypothermia, frostbite, and nonfreezing cold injuries" covers cold injuries, which are among the most common environmental injuries seen in the backcountry. The WEMT must understand principles of thermoregulation; heat production and heat loss; recognition of hypothermia, frostbite, and immersion foot; and appropriate care.

"Heatstroke, heat exhaustion, and dehydration" provides necessary information about the balance of heat production and heat loss in a hot environment and the fluid requirements necessary to support physiologic cooling. WEMTs need to know how to recognize and provide long-term care for victims of heat exhaustion, heatstroke, and dehydration.

Lifting and Moving Patients

"Use of Stokes litters and improvising litters" discusses the primary device for evacuation from the backcountry. Even when a helicopter is used, the victim is usually "packaged" in a litter before being loaded. WEMTs must know the specific techniques for victim packaging in a litter to protect and support injuries. Proper carrying techniques and methods of belaying a litter up or down a steep slope are critical to the safety of everyone involved.

Ambulance Operations

"Helicopter-assisted rescues" describes the use of helicopters in backcountry rescue efforts and evacuation. WEMT training should address the dangers, hazards, and limitations of helicopters.

Self-Preservation

Whether incidents or disasters are caused by nature or man, rescuers responding to remote places must know how to take care of themselves, from the moment they arrive to the time they return to safety.

The classic mistake is when responders assume that there will be someone waiting to make sure that they have potable water, safe food, adequate shelter, and basic protection from the elements and from violence. This is never a safe assumption. When responding to a mountain rescue, each member is responsible for carrying a bivouac kit that contains all the necessities to sustain life in the wild for at least 24 hours. When responding to a disaster, each individual needs to be prepared to survive on his or her own for at least 3 days. Each member has to be potentially self-sufficient, carrying the essentials for survival in a disaster team bivouac kit. The kit must provide shelter and protection from elements, allowing the ability to produce potable water, calorie-rich foods, navigation, and light in the dark.

Self-preservation is not an easy task. In the case of wilderness rescue, participants are often "outdoors people," who before getting involved in search and rescue activities have already obtained wilderness skills. On the other hand, as teams form to respond to a larger disaster, they may be highly skilled medically but without essential survival skills, such as how to make potable water, choose safe foods, or establish a bivouac.

The essentials of personal survival:

Water: Humans need at least 2 liters of water per day to survive, 4 liters to maintain health, and much more if sweating heavily. Water can be a source of infectious disease, such as giardiasis, hepatitis, amebiasis, and cholera, any of which can cause major diarrhea and abdominal pain. It is absolutely essential to be able to produce potable, safe water.

Three ways to produce potable water are: boiling, chemical disinfection, or filtering. Drinking potentially unsafe water puts the entire team at risk.

Food: One must know what is safe to eat and how to properly prepare meals and maintain a clean "kitchen." If refrigeration is not available, meat has to be kept alive until it is ready to be cooked. Safe food rules include:

Eat only vegetables or fruits that can be peeled, once they are peeled.

Eat vegetables that have been cooked and are served hot.

Never eat raw, lightly cooked, or cold meats.

Meat should be cooked until "well done" and served hot.

Shelter: Shelter is both a roof over your head and clothing on your back. It is not enough to carry a tent; one also must have the skill to improvise or bivouac (build an emergency camp).

Navigation: Know how to use a compass and read a map. One should be able to walk a straight line using a compass and determine the distance traveled.

Other important items in the survival kit include sources of light, fire building materials, signaling devices, knife, parachute cord, spare socks, hat, and metal cup to make warm water. See Box 31-5.

Attitude: The most important aspect of survival is attitude and confidence. Practice and experience build confidence. Maintaining a positive attitude will help ensure that you not only survive, but also thrive.

APPENDIX

The following is a list of organizations and committees dedicated to some aspect of extended medical, rescue, and technical training. Many are also active in mountain, wilderness, marine, or disaster rescue and management efforts.

Advanced Wilderness Life Support (AWLS)
University of Utah School of Medicine
358 South 700 East B509
Salt Lake City, UT 84102
888-521-2957
www.awls.org
Resource: Teaches AWLS certification programs and practical approaches to prevention and treatment of injuries and illnesses related to the wilderness environment.

Aerie Backcountry Medicine
240 North Higgins Suite 16
Missoula, MT 59807
406-542-9972
E-mail: info@aeriemed.com
www.aeriemed.com
Resource: Emphasizes prevention, treatment, and improvisation; offers wilderness EMT, wilderness first responder, wilderness first aid, avalanche, and swift-water rescue courses.

American Alpine Club
710 Tenth Street, Suite 100
Golden, CO 80401
303-384-0110
www.americanalpineclub.org
Resource: Publishes the *American Alpine Journal* and annual *Accidents in North American Mountaineering*. Has committees dedicated to establishing and promoting standards in safety and education in mountaineering.

American Mountain Guides Association (AMGA)
Physical address:
1209 Pearl St. Suites 12 and 14
Boulder, CO 80302
Mailing address:
P.O. Box 1739
Boulder, CO 80306
Phone: 303-271-0984
Fax: 303-271-1377
www.amga.com
Resource: Dedicated to establishing and maintaining standards for mountaineering and professional mountain guides. Publishes quarterly *Mountain Bulletin*.

Appalachian Mountain Club
5 Joy Street
Boston, MA 02108
617-523-0655
E-mail: information@outdoors.org
www.outdoors.org
Resource: Active mountain rescue team that offers a variety of workshops on outdoor skills, environmental issues, and wilderness medical and rescue skills. Publishes quarterly *Appalachia*.

International Society for Mountain Medicine (ISMM)
PO Box 31142
Colorado Springs, CO 80931-1142
Phone: 719-572-1372
Fax: 719-572-1514 or 800-967-7494 (in the United States)
E-mail: membership@ismmed.org
www.ismmed.org
Resource: An international organization dedicated to research and education in mountaineering. Publishes the quarterly *Newsletter of the ISMM*.

Mountain Rescue Association
P.O. Box 880868
San Diego, CA 92168-0868
Fax: 619-374-7072
www.mra.org
Resource: National wilderness rescue organization dedicated to the development of standards and certification of mountain rescue teams.

Nantahala Outdoor Center
13077 Highway 19 W
Bryson City, NC 28713-9165
Phone: 888-905-7238 or 828-488-2176
www.noc.com
Resource: Offers a variety of courses on white-water rescue and wilderness medical and rescue training.

National Association for Search and Rescue
P.O. Box 232020
Centreville, VA 20120-2020
703-222-6277
Toll Free: 877-893-0702
E-mail: info@nasar.org
www.nasar.org
Resource: National information resource for search and rescue, as well as certifications in various search functions. Publishes quarterly journal *Response*.

National Cave Rescue Commission (NCRC)
c/o National Speleological Society
Cave Avenue
Huntsville, AL 35810
205-852-1300
EMERGENCY: National Rescue Coordination: 800-851-3051
www.caves.org/io/ncrc
Resource: Active national cave rescue team.

National Ski Patrol System, Inc. (NSP)
Ski Patrol Building, Suite 100
133 South Van Gordon Street
Lakewood, CO 80228
Phone: 303-988-1111
Fax: 800-222-4754
E-mail: nsp@nsp.org
www.nsp.org
Resource: Active rescue teams and ski patrols. Offers an outdoor emergency care course, various ski patrol certifications, avalanche training, and introductory mountaineering training.

Stonehearth Open Learning Opportunities (SOLO)
P.O. Box 3150
Conway, NH 03818
603-447-6711
888-SOLO-MED
E-mail: info@soloschools.com
www.soloschools.com
Resource: An international organization dedicated to developing and offering a variety of courses and certifications in wilderness and marine medicine, rescue, leadership, and outdoor skills. An active mountain rescue team. Publishes bimonthly *Wilderness Medicine Newsletter.*

Union Internationale des Associations d'Alpinisme (UIAA)
(International Mountaineering and Climbing Federation)
Monbijoustrasse 61
Postfach
CH-3000 Bern 23/Switzerland
Tel: +41 (0)31 370 1828
Fax: +41 (0)31 370 1838
E-mail: office@uiaa.ch
www.uiaa.ch
Resource: An international organization dedicated to the promotion of standards, safety, awareness, and education in mountaineering worldwide. Produces multiple publications on mountain safety and medicine.

U.S. Coast Guard Headquarters
2100 Second Street, SW
Washington, DC 20593-0001
202-267-1012 (Boating Operations)
www.uscg.mil
Resource: Active national marine rescue military organization. Source of information and various boating-related certifications.

Wilderness Medical Associates
400 Riverside Street, Suite A-6
Portland, ME 04103
Phone: 207-797-6005 or 888-WILDMED (945-3633 within US and Canada)
Fax: 207-797-6007
E-mail: office@wildmed.com
www.wildmed.com
Resource: Offers a variety of courses and certifications in wilderness medical and rescue courses.

Wilderness Medical Society
810 E. 10th Street, P.O. Box 1897
Lawrence, KS 66044
Phone: 800-627-0629
Fax: 785-843-1853
E-mail: wms@wms.org
www.wms.org
Resource: A physician-based national wilderness medical organization with various committees dedicated to education in wilderness emergency medicine. Particular attention to education for physicians. Publishes quarterly newsletter and peer-reviewed journal *Wilderness and Environmental Medicine* (formerly *Journal of Wilderness Medicine*).

Wilderness Medicine Institute of the National Outdoor Leadership School (NOLS)
284 Lincoln Street
Lander, WY 82520-2848
Phone: 800-710-NOLS
Fax: 307-332-1220
www.nols.edu/wmi
Resource: Offers a variety of courses and certifications in wilderness medicine and rescue.

SUGGESTED READINGS

American Academy of Orthopedic Surgeons: Emergency Care and Transportation of the Sick and Injured, 7th ed. Sudbury, MA, Jones & Bartlett, 1999.
Auerbach P: Medicine for the Outdoors. New York, The Lyons Press, 1999.
Bowman W: Outdoor Emergency Care, 3rd ed. Lakewood, CO, National Ski Patrol System, 1998.
Forgey W (ed): Wilderness Medical Society: Practice Guidelines for Wilderness Emergency Care. Merrillville, IN, ICS Books, 1995.
Henry M, Stapleton E: EMT Prehospital Care, 2nd ed. Philadelphia, WB Saunders, 1997.
Houston C: Going Higher, 4th ed. Seattle, The Mountaineers Books, 1998.
Iverson KV (ed): Position Statements of the Wilderness Medical Society. Point Reyes Station, CA, Wilderness Medical Society, 1989.
Lindsay L, et al: Wilderness First Responder, Wilderness and Environmental Medicine. Lawrence, KS, Alliance Communications Group, 1999.
McSwain N, et al: The Basic EMT: Comprehensive Prehospital Care, 1st ed. St Louis, Mosby, 1997.
Mistovich, J et al: Prehospital Emergency Care and Crisis Intervention, 6th ed. Upper Saddle River, NJ, Brady, 2000.
Schimelpfenig T, Lindsey L: NOLS Wilderness First Aid. Lander, WY, National Outdoor Leadership School, 1991.
U.S. Department of Transportation, National Highway Traffic Safety Administration: Emergency Medical Technician-Basic: National Standard Curriculum, 4th ed. Washington, DC, 1994, U.S. Government Printing Office.
Wilkerson J (ed): Medicine for Mountaineering, 4th ed. Seattle, The Mountaineers Books, 1993.
Williamson J (ed): Accidents in North American Mountaineering, Golden, CO, American Alpine Club, published yearly.

32 Search and Rescue

Donald C. Cooper, Patrick H. LaValla, and Robert C. Stoffel

As ever-increasing numbers of outdoor users turn to the wilderness for recreation, the medical community and search and rescue (SAR) organizations are contending with a growing number of lost, sick, and injured persons. Wilderness search, rescue, and medical intervention are unique in several ways. All aspects of SAR are enormously time consuming. Simply raising the alarm for someone lost or injured in an isolated area may take hours, days, or even weeks. Organizing a response, including obtaining equipment and transportation for responders, requires a variable amount of time, depending on the level of preparedness of the response organization. Locating, gaining access to, stabilizing, and transporting a victim to definitive care can be a lengthy process.

Because it takes many persons to perform a wilderness rescue (six or eight persons are required to carry a litter 1 mile), logistic considerations such as food, shelter, and transportation for responders quickly create their own problems. SAR personnel are subjected to the same risks and environmental stresses that compromise victims. To obviate further tragedy, they must have a heightened awareness of potential danger, adverse conditions, and personal limitations. In addition to basic and advanced life support training, rescuers must have extensive wilderness experience—or experience in the particular environment in which they will be operating—that combines practicality with creativity and resourcefulness. SAR personnel must have training in survival, improvisation, communications, leadership, navigation (e.g., map, compass, and global positioning system [GPS]), first aid, and specific SAR techniques. Many interventions, such as cardiopulmonary resuscitation, defibrillation, tube thoracostomy, tracheal intubation, and intravenous therapy, are difficult—if not impossible—in the wilderness setting. Examinations may be hampered by the bulky clothing necessary to keep the victim warm and dry. Medications and equipment are subject to rough handling and extremes of temperature, which may render them ineffective, unsterile, or inoperative (see Appendix: Drug Storage and Stability).

Finally, decision making that optimizes patient care while not unduly risking the well-being of SAR personnel requires experienced leadership grounded in both common sense and technical skill. Perhaps the demands of SAR were best summarized by the wise rescuer who said that climbers, divers, hikers, and other outdoor enthusiasts get to choose where they practice their skills, but SAR personnel have no such choice. The situation, usually urgent, dictates where and when rescuers practice their art. The same situation that already compromised at least one person's health or well-being subsequently endangers the SAR participants.

This chapter introduces medical professionals to the unique search, rescue, and medical problems encountered in wilderness, remote (including urban disaster environments), and backcountry situations. The rudiments of SAR coordination, resources, and specialized problems are discussed. This information will help medical personnel understand how the SAR community works and provide an educational foundation to help prevent situations requiring undue risk, or SAR personnel themselves from having to be rescued.

SEARCH AND RESCUE: AN OVERVIEW

SAR systems provide the response for overdue, lost, injured, or stranded persons, usually in connection with outdoor activities and environments. In the context of SAR, "wilderness" can take on several meanings. For instance, most consider wilderness to be regions that are uninhabited and uncultivated. Personnel may be called out to search a natural area such as a large park or desert, but it is equally likely that a search will be urban, in an area devastated by a natural disaster such as an earthquake or hurricane. Because most of the population in the United States resides in urban areas, emergency responders and SAR personnel are far more likely to encounter urban wilderness than a natural one. However, this chapter focuses on the nonurban setting.

Types of SAR emergencies vary nationally and internationally, as do the responders. Programs, equipment, and personnel differ geographically in accordance with local needs and available resources. SAR can generally be defined as "finding and aiding people in distress-relieving pain and suffering."[5] However, contemporary references find it most useful to define "search" and "rescue" separately (Box 32-1).[16]

SAR often involves a great many volunteers and entails a multitude of skills. The eruption of Mt. St. Helens (1980), terrorist attacks on the World Trade Centers (2001), multiple hurricanes that struck the southeastern United States in 2004 and 2005, including Katrina, and the Indonesian earthquake and subsequent tsunamis of 2004, are all examples of significant, yet vastly different, threats and hazards requiring widely diverse sets of SAR skills.

SAR operations benefit comprehensive emergency management, providing a training ground and experience builder for disaster response capability at the most elementary level. The management concepts used in SAR operations establish foundation principles for providing response capability to large-

Box 32-1. Search and Rescue Defined

Search—An operation using available personnel and facilities to locate persons in distress.
Rescue—An operation to retrieve persons in distress, provide for their initial medical or other needs, and deliver them to a place of safety.

From the International Maritime Organization and International Civil Aviation Organization (IMO/ICAO): International Aeronautical and Maritime Search and Rescue Manual: Vol. I, Organization and Management; Vol. II, Mission Co-ordination; and Vol. III, Mobile Facilities. London/Montreal, The International Maritime Organization (IMO) and the International Civil Aviation Organization (ICAO), 1999.

scale emergencies and disasters. Nearly every type of hazard mentioned in comprehensive emergency management plans (local and state disaster coordination plans, mandatory in all states) and international treaties require planning for search and rescue.[24] Management of these SAR operations can range from directing the actions of a few responders in a small community hit by minor flooding to managing an effort involving thousands of searchers in a large urban calamity. Often, large situations involve several political subdivisions (e.g., counties, states, countries) and coordination of air and ground resources. Local governments and other agencies that participate in SAR response must coordinate diverse multiskilled responders. In addition, many agencies that collectively support multiorganizational SAR responses operate under their own specific statutory authority.[40]

SAR operations entail a motivating time factor that focuses on a successful conclusion: finding or rescuing a missing, lost, or injured subject before he or she succumbs to the effects of the environment, injuries, or a specific hazard. To be effective, extremely diverse organizations must be drawn together in a life-threatening situation with a commonality of purpose; this is even more true during a large-scale disaster.

International Agreements

International conventions are often what countries use to establish rules between them. The Convention on International Aviation, the International Convention on Maritime Search and Rescue, and the International Convention for the Safety of Life at Sea (SOLAS) include rules requiring countries that are parties to these agreements to provide aeronautical and maritime SAR coordination and services for their territories and territorial seas. Coverage of the high seas is apportioned among the member countries by these instruments as well. To carry out its SAR responsibilities, a country usually establishes a national SAR organization, or joins one or more other countries to form a regional SAR organization. In the United States, the national SAR organization is called the National Search and Rescue Committee (NSARC).

While airborne, virtually all commercial aircraft on international routes are under positive control (e.g., followed by radar and in direct communication with air traffic controllers) by air traffic services (ATS) units. The International Civil Aviation Organization (ICAO) has linked ATS units into a worldwide system. Consequently, SAR agencies are usually notified very quickly when an international commercial flight has an emergency. Commercial aircraft on domestic routes and general aviation aircraft may not be under positive control, which can result in delayed reporting of their emergencies. 121.5 MHz is the international aeronautical distress frequency and is monitored by ATS, some commercial airliners, and other aeronautical facilities where needed to ensure immediate reception of distress calls. Emergency locator transmitters (ELTs) are carried in most aircraft and are required in most aircraft flown in the United States (see COSPAS-SARSAT).

Additional information on international SAR may be found at the International Maritime Organization (IMO) website (www.imo.org) and ICAO website (www.icao.int).

International Stages of SAR Operations

The international SAR community has developed an approach to organizing operations that has evolved over the past several decades. This system is documented in the *International Aeronautical and Maritime SAR (IAMSAR) Manual* that was developed jointly by the ICAO and the IMO. Each of the three *IAMSAR Manual* volumes is written with specific SAR system duties in mind and can be used as a stand-alone document or in conjunction with the other two volumes as a means to attain a full view of the SAR system. Volume 1, *Organization and Management,* discusses the global SAR system concept, establishment and improvement of national and regional SAR systems, and cooperation with neighboring countries to provide effective and economical SAR services. Volume II, *Mission Coordination,* assists personnel who plan and coordinate SAR operations and exercises. Volume III, *Mobile Facilities,* is intended to be carried aboard rescue units, aircraft, and vessels to help with performance of a search, rescue, or on-scene coordinator functions and with aspects of SAR that pertain to their own emergencies.

The *IAMSAR Manual* describes a series of five stages of a SAR operation through which most SAR events pass: Awareness, Initial Action, Planning, Operations, and Conclusion. The manual suggests that, "These stages should be interpreted with flexibility, as many of the actions described may be performed simultaneously or in a different order to suit specific circumstances."[16] The reason the *IAMSAR Manual* does not have a "Preplanning" stage is twofold. First, the five stages of a SAR operation are about what happens in a specific incident. Preplanning can address types of incidents, but not specific incidents. Second, the preplanning function is addressed in what are called plans of operations or operations plans (OPLANs); see Appendix C of the *IAMSAR Manual,* volume II. These are "standing" plans for how to deal with various kinds of situations.

Awareness Stage

A SAR organization cannot respond to an incident until it becomes aware that someone is in need of assistance. At this stage, information is received that someone (or an aircraft or vessel) is, or will be, in distress. There may not be enough information to initiate action, or action may not be necessary yet.

Initial Action Stage

When enough information is available, immediate action may be necessary. The first action is always to evaluate the information available, attempt to gain more, and determine the degree of the emergency. The "phase" of the emergency is used

by the SAR authority to describe the level of confidence in the available information and the level of concern it raises about the safety of an aircraft or vessel and the persons on board. The *IAMSAR Manual* describes three emergency phases: uncertainty, alert, and distress.

Uncertainty phase: a situation wherein doubt exists as to the safety of an aircraft or vessel and the persons on board

Alert phase: a situation wherein apprehension exists as to the safety of an aircraft or vessel and the persons on board

Distress phase: a situation wherein there is reasonable certainty that a vessel or other craft, including an aircraft or person, is threatened by grave and imminent danger and requires immediate assistance

An overdue aircraft, as an example, may be late for a wide range of reasons. The phases of the emergency allow persons in charge to establish and communicate a summary of the available information that matches its validity, the urgency of action, and the extent of the response required. The *IAMSAR Manual* requires specific initial actions based on the established emergency phase.

Planning Stage

Planning operations is essential, especially when the location of the distress situation is unknown. "Proper and accurate planning is critical to SAR mission success; if the wrong area is searched, there is no hope that search personnel will find the survivors, regardless of the quality of their search techniques or the amount of their search effort."[16] The *IAMSAR Manual* suggests the use of computers for operational planning, but also includes basic information on manual methods of planning searches.

Operations Stage

The operations stage encompasses all the physical activities involved in finding, providing assistance to, and rescuing people in distress. In short, this phase is where the plans are carried out.

Conclusion Stage

The conclusion stage is entered when it is determined that no one is in distress, when the search or rescue is concluded, or when nothing was found, and the search is called off. This phase is when all SAR resources are notified that the mission is concluded.

COSPAS-SARSAT

The SARSAT system (search and rescue satellite-aided tracking) was developed in a joint effort by the United States, Canada, and France. The COSPAS system (*cosmicheskaya sistyema poiska avariynich sudov*; in English, "space system for the search of vessels in distress") was developed by the Soviet Union. These four nations banded together in 1979 to form COSPAS-SARSAT. In 1982, the first satellite was launched and the first life saved using the system. By 1984, the system was declared fully operational. As of this writing, the four original member nations have been joined by 31 other nations that operate 47 ground stations and 25 mission control centers worldwide or serve as search and rescue points of contact (SPOCs). Visit www.cospas-sarsat.org or www.sarsat.noaa.gov for more details.

The system uses satellites to detect and locate emergency beacons carried by ships, aircraft, and individuals. The system consists of a network of satellites, ground stations, mission control centers, and rescue coordination centers. When an emergency beacon is activated, the signal is received by a satellite and relayed to the nearest available ground station. The ground station, called a *local user terminal* (LUT), processes the signal and calculates the position from which it originated. This position is transmitted to a mission control center (MCC), where it is joined with identification data and other information on that beacon. The MCC then transmits an alert message to the appropriate rescue coordination center (RCC) based on the geographic location of the beacon. If the location of the beacon is in another country's service area, then the alert is transmitted to that country's MCC (see Fig. 2-2 in Chapter 2).

Distress Radio Beacons

The most recognizable component of the SARSAT system is the distress radio beacon, also known as a "beacon." There are generally three types of beacons used to transmit distress: emergency position indicating radio beacons (EPIRBs) designed for maritime use, ELTs designed for aviation use, and personal locator beacons (PLBs) designed for use by individuals and land-based applications. Although the three types of devices are physically different owing to the differing environments in which they must operate, they all work on the same principle and in the same way. When turned on, each transmits alert signals on specific frequencies intended to be received by COSPAS-SARSAT satellites.

Emergency Position Indicating Radio Beacons. EPIRBS in use for maritime applications are of two types. One type transmits an analog signal on 121.5 MHz. The other type transmits a digital identification code on 406 MHz and a low-power "homing" signal on 121.5 MHz.

The U.S. Coast Guard (USCG) maintains an outstanding website with more information on EPIRBs: www.navcen.uscg.gov/marcomms/gmdss/epirb.htm.

Emergency Locator Transmitters. ELTs were the first emergency beacons developed, and most aircraft are required to carry them. ELTs were intended for use on the 121.5-MHz frequency to alert aircraft flying overhead. Obviously, a major limitation to these is that another aircraft must be within range and listening to 121.5 MHz to receive the signal. One of the reasons the COSPAS-SARSAT system was developed was to provide a better receiving source for these signals. Another reason was to provide location data for each activation (something that overflying aircraft were unable to do).

Different types of ELTs are currently in use. There are about 170,000 of the older-generation 121.5-MHz ELTs in service in the United States. Unfortunately, these have proved to be highly ineffective. They have a 99% false alert rate, activate properly in only 12% of crashes, and provide no identification data. In order to fix this problem, 406-MHz ELTs were developed to work specifically with the COSPAS-SARSAT system. These ELTs dramatically reduce the false alert impact on SAR resources, have a higher accident survivability success rate, and decrease the time required to reach accident victims by an average of 6 hours.

Presently, most aircraft operators are mandated to carry an ELT and have the option to choose either a 121.5-MHz or a 406-MHz version. A 1996 U.S. Government study concluded that 134 extra lives and millions of dollars in SAR resources

could be saved per year if only the 406-MHz type ELTs were used.[27] Unfortunately, 406-MHz ELTs cost nearly three times as much as 121.5-MHz ELTs, and convincing aircraft owners to upgrade has been a slow process.

On the recommendation of ICAO and IMO, the COSPAS-SARSAT Council announced in 2000 that it will be phasing out 121.5/243-MHz satellite alerting from the system. Users of 121.5/243-MHz beacons will have until February 1, 2009 to switch over to the generally superior 406-MHz models.

Personal Locator Beacons. PLBs are portable units that operate much the same as EPIRBs or ELTs. These beacons are designed to be carried by an individual person instead of on a boat or aircraft. Unlike ELTs and some EPIRBs, they can only be activated manually and operate exclusively on 406 MHz in the United States. Similar to EPIRBs and ELTs, all PLBs also have a built-in, low-power homing beacon that transmits on 121.5 MHz. This allows rescue forces to home in on a beacon once the 406-MHz satellite system has put them within 2 to 3 miles (3.2–4.8 km). Some newer PLBs also allow GPS units to be integrated into the distress signal. This GPS-encoded position dramatically improves the location accuracy down to the 100-meter level.

In the United States, PLBs were in limited use until July 1, 2003, after which they were fully authorized for nationwide use. The National Oceanic and Atmospheric Administration encourages all PLB users to be acutely aware of the responsibility that comes with owning one of these devices. PLBs are a distress alerting tool and work exceptionally well. PLB users should familiarize themselves with proper testing and operating procedures to prevent false activation and be careful to avoid their use in nonemergency situations.[27]

Search and Rescue in the United States

SAR involves many agencies and volunteers. The federal government assumes some responsibility for overall coordination, especially of federal or military resources requested by local or state agencies.

U.S. National Search and Rescue Plan

The U.S. National Search and Rescue Plan (NSP) was first published in 1956. It provides guidance to signatory federal agencies (Departments of Transportation, Defense, and Commerce, as well as the Federal Communications Commission [FCC], the National Aeronautics and Space Administration [NASA], and the land management components of the Department of the Interior) for coordinating civil SAR services to meet domestic needs and international commitments. The federal government assists with coordination of certain SAR services, including any federal or military resources that are requested by local or state agencies. Guidance for implementing the NSP is provided in the *IAMSAR Manual,* the National Search and Rescue Supplement (NSS, a domestic interagency supplement to the *IAMSAR Manual;* discussed later), and other relevant directives of the plan participants.[28]

NSARC is responsible for coordinating and improving federal involvement in civil SAR for aeronautical, maritime, and land communities within the United States. NSARC is also the federal-level committee formed to sponsor and oversee the NSP. Member agencies of NSARC are the signatories of the NSP. More information about NSARC may be obtained from the Committee's web site at www.uscg.mil.

The *IAMSAR Manual* and the NSP define a SAR region (SRR) as "an area of defined dimensions, associated with a rescue coordination center (RCC), within which SAR services are provided."[16,28] An RCC is a unit responsible for promoting efficient organization of SAR services and for coordinating the conduct of SAR operations within an SRR. For every SRR, there is one RCC, and the goal is to have no overlaps or gaps between SRRs around the world.

In the United States, there are two types of SRRs: maritime and aeronautical. Although only the ocean areas surrounding the United States and its territories fall within the maritime SRRs (marine vessels cannot sail on land), both the ocean and land areas of the United States fall within aeronautical SRRs (aircraft can fly over both water and land). The maritime SRRs surrounding the United States include, in the Pacific: Juneau, Honolulu, Seattle, and Alameda; and, in the Atlantic: Boston, Norfolk, Miami, New Orleans, and San Juan. The oceans surrounding the United States and its territories also fall within aeronautical SRRs whose names and limits coincide with their maritime counterparts. However, with the exception of U.S. islands (Hawaiian, Puerto Rico, etc.) that are contained entirely within maritime SRRs and the Great Lakes (Cleveland Maritime SRR), all U.S. land falls within either the Elmendorff Aeronautical SRR (Alaska) or the Langley Aeronautical SRR (continental United States).

According to the NSP, an *SAR coordinator* has overall responsibility for establishing RCCs as necessary and for providing or arranging for SAR services with U.S. SRRs.[16] The SAR Coordinators for the United States are as follows:

- The U.S. Air Force for the recognized U.S. aeronautical SRR corresponding to the continental United States other than Alaska;
- The U.S. Pacific Command for the recognized U.S. aeronautical SRR corresponding to Alaska; and
- The USCG for the recognized U.S. aeronautical and maritime SRRs that coincide with the ocean environments, including Hawaii.

Although not considered a national SAR coordinator, the National Park Service is the lead agency that provides SAR and other emergency services within national parks. In small parks, this is often achieved through agreements with surrounding emergency service providers. Outside national parks, state and local authorities or SAR units often accept responsibility for providing domestic land SAR services.

Because of the unique scale and SAR challenges in Alaska, the USCG is the lead federal agency for inland SAR in certain areas of the state, including the Alaska Peninsula (south of 58° N), the Aleutian Islands, and all other coastal islands. The reason for this is simple: USCG assets can respond much quicker than can the U.S. Air Force Anchorage RCC to these areas. The Alaska State Troopers are the state agency responsible for land SAR in Alaska. Because of the unforgiving environment, all federal, state, and local agencies work together closely in response to SAR missions in Alaska.

In addition to the specific assignments of SAR coordinators, the NSP allows ". . . local and state authorities to designate a person to be a SAR Coordinator within their respective jurisdictions."[28] These local and state SAR coordinators, if established, become important contacts for national SAR coordinators.

Many U.S. states have chosen to retain established SAR responsibilities within their boundaries for incidents primarily

local or intrastate in character. In such cases, agreements have been made between SAR coordinators and relevant state organizations.

The National SAR Supplement

NSARC also directs preparation of the NSS, which provides guidance to federal agencies concerning implementation of the NSP. The NSS provides specific additional national standards and guidance that build on the baseline established by the *IAMSAR Manual*. It provides guidance to all federal forces, military and civilian, that support civil SAR operations.[29]

Specifically, the NSS is designed to serve as both a training and operational tool for civil SAR operations. SAR planning is both an art and a science, relying greatly on the creativity and experience of the personnel involved. Because of the many variables encountered during SAR operations and the individuality of each SAR case, the guidance provided in the NSS must be tempered with sound judgment, having due regard for the individual situation. Very little in the NSS is mandatory because it is not intended to relieve SAR personnel of the need for initiative and sound judgment.[29]

Each of the signatory agencies of the NSARC (and NSP) may also develop an "addendum" to the NSS for their agency. Such documents could include policies, information, procedures, and so forth, on civil SAR matters applicable to the agency concerned, and consistent with the *IAMSAR Manual* and NSS.

The NSS is available on the USCG website (www.uscg.mil).

U.S. Rescue Coordination Centers

The USCG and Air Force both operate RCCs in the United States, but each service takes a slightly different approach. The Air Force RCC (AFRCC) coordinates inland SAR activities in the continental United States but does not directly conduct SAR operations. In most situations, the Civil Air Patrol (CAP), state police, or local rescue services carry out the actual SAR operations. In contrast, the USCG not only coordinates but also conducts maritime SAR missions.

U.S. Air Force Rescue Coordination Center. Established in 1947 to meet the growing demand for SAR and its legislated responsibility, the original three AFRCCs have evolved into a single RCC located at Langley Air Force Base in Hampton, Virginia, under the Air Combat Command. The peacetime mission of the AFRCC is to build a coordinated SAR network, ensuring timely, effective lifesaving operations whenever and wherever needed. As of December 2003, the AFRCC recorded the prosecution of more than 58,000 SAR missions, resulting in more than 13,500 lives saved.[36]

The AFRCC functions around the clock and is staffed by people trained and experienced in the coordination of SAR operations. The center is equipped with extensive audio and digital communications equipment and maintains a comprehensive resource file listing federal, state, local, and volunteer organizations that conduct or assist SAR efforts in the United States, Canada, and Mexico.

There are four types of authorized AFRCC missions: search, rescue, MEDIVAC, and mercy.

Search. Once a distress situation is determined to exist but a location is unknown, federal SAR forces may be activated to search for, locate, and relieve the distress situation. The object of these searches may take the form of overdue aircraft, ELTs, hunters, hikers, or children.

Rescue. A rescue mission entails the use of federal SAR forces to recover persons in distress whose location in a remote area is known, but who need assistance. This may be in the form of transportation to safety or to an adequate medical facility. These requests are normally received by the AFRCC from park service personnel or the local law enforcement authority.

MEDIVAC. The transportation by federal assets of persons from one medical facility to another is defined as aeromedical evacuation, or MEDIVAC. Requests are normally received from a local hospital when no commercial transportation is available, the person's life is in jeopardy, and time is critical. Each request is evaluated, and the decision to use federal resources is weighted heavily by the attending physician's medical opinions.

Mercy. A mission to transport blood, organs, serum, medical equipment, or personnel to relieve a specific time-critical, life-threatening situation is referred to as a *mercy mission*. Requests are normally referred from a local hospital authority or, in some cases, the American Red Cross when commercial transportation is not available.

Although the AFRCC will accept and act on initial notification from any person or agency, it will attempt to determine the urgency and the facts pertaining to the situation before obliging itself. Several aspects of the situation are considered before a mission is opened, including the following:

1. Medical evaluation and urgency
2. State agreement requirements
3. Posse Comitatus Act
4. Conflict of interest with commercial resources
5. Resource availability

The medical condition of the victim or victims is the most important aspect of mission consideration. The AFRCC will only consider a request valid when there is an immediate threat to life, limb, or sight. A mission will only be started to prevent death or the aggravation of a serious injury or illness. The observations and opinions of a physician at the incident site weigh heavily on the decision to open a mission, and a flight surgeon is on call at the AFRCC when a local physician is unavailable.

Each state has an agreement on file in the AFRCC describing the responsible agency and coordinating requirements for the various types of SAR missions. Each request for federal assistance is evaluated to ensure the requirements stipulated in the relevant agreement are met. Title 18 USC 1385 (the Posse Comitatus Act) prohibits military participation in civil law enforcement activities. Although there are some exceptions to the prohibition, as a general rule, Department of Defense (DOD) forces, including the CAP, will be restricted from participating in searches in which the person being sought is evading searchers, or is a fugitive, or when foul play is considered.

On MEDIVAC or mercy missions in which the patient is not eligible for DOD medical benefits, federal assets cannot be used when commercial resources are available. Even when a patient is unable to pay or is destitute, commercial resources will be checked for availability and provided the opportunity to accept the mission before allocating federal resources.

Although any SAR-capable asset belonging to the federal government may be requested, each resource is evaluated for distance from the distress location, special equipment requirements, urgency of the situation, and which resource can best accomplish the mission. Military forces may be called on to assist in civilian SAR missions. However, their participation in these activities must not interfere with their primary military mission. Once the decision has been made to use federal resources, a mission number is assigned and SAR forces are selected based on the geographic location and mission requirements. The Air Force coordinator then works closely with the responsible agency in an attempt to provide the resources best suited to accomplish the mission.[36]

Additional information on the AFRCC can be found at www.acc.af.mil/afrcc.

U.S. Coast Guard Rescue Coordination Centers. The USCG, now a division of the U.S. Department of Homeland Security (DHS), is designated as the federal SAR coordinator for the maritime SRRs surrounding the United States and large portions of the high seas. The USCG is responsible for providing and coordinating SAR services over 28 million square miles of the world's oceans, mostly in the Northern Hemisphere. This responsibility is divided into two Areas: Atlantic and Pacific. These Areas are divided into several Districts, which are further divided into Groups (several per District) and Stations. A 24-hour alert status is maintained year-round at all levels; Coast Guard resources can be underway or airborne within minutes of notification of a SAR incident. At its headquarters, each Area and District maintains a fully staffed operations/command center responsible for coordinating operations within the Area or District on a 24-hour basis. When coordinating SAR missions, these operations centers are called *rescue coordination centers* (RCCs). Although minor SAR incidents are often resolved at the Station or Group level, the District or Area assumes the duties of the SAR mission coordinator in more complex or large-scale missions.[37]

The USCG arguably prosecutes more SAR missions than does any other organization or agency. It is also notable that the USCG, although considered an important part of the United States military forces, is a separate federal agency now under the DHS, not under the Department of Defense, as is the Air Force.

An important global SAR-related service of the USCG is the automated mutual-assistance vessel rescue (AMVER) system. AMVER involves ships, regardless of flag, voluntarily providing information about their capabilities (i.e., medical personnel on board, rescue equipment) and regularly reporting their location to a global computer system that tracks their whereabouts. When a situation arises that requires SAR capabilities, a surface picture (SURPIC) is produced that graphically shows the location of all AMVER participants in the vicinity. The RCC can use this information to select the best one or several ships to respond to the emergency, and allow all others to continue their voyages. Today, about 12,000 ships from more than 140 countries participate in AMVER. On average, 2800 ships are on the AMVER plot each day, with more than 100,000 voyages tracked annually. The AMVER system has saved more than 2000 lives since 1990.[38]

A "preventive SAR" service provided by the USCG as a direct result of the Titanic disaster is the International Ice Patrol, with operations funded by SOLAS Convention signatories.[17] Since 1913, the Ice Patrol has amassed an enviable safety record, with not a single reported loss of life or property caused by collision with an iceberg outside the advertised limits of all known ice in the vicinity of the Grand Banks. However, the potential for a catastrophe still exists, and the Ice Patrol continues its mission using high-tech sensors and computer models.[37]

The USCG also performs or coordinates the medical evacuation of seriously ill or injured persons from vessels at sea if the patient's condition warrants and USCG assets are within range. For less serious situations, USCG flight surgeons offer medical advice by radio. On rare occasion, the RCC may coordinate with a U.S. Navy ship to allow a USCG MEDIVAC helicopter to refuel to extend its range. Also on rare occasion, the RCC may coordinate with the Air Force to dispatch pararescue personnel to parachute to the vessel and stabilize the patient. In either case, these actions are taken only in the most serious situations when one or more lives depend on such drastic actions.

If a vessel is reported overdue or unreported (i.e., failed to check in when expected), USCG assets may or may not launch immediately, depending on whether the overdue craft is thought to be in immediate danger. Regardless, an extensive investigative effort is initiated immediately. During this investigation, a preliminary communications check (PRECOM) and extended communications check (EXCOM) will likely take place. These actually include more than just contacting intended destinations. They also include interviewing persons who may be knowledgeable about the craft, as well as dispatching USCG vehicles or small boats to physically check harbors, marinas, launching ramps, and the like. In addition, an urgent all-ships broadcast is initiated requesting information on any recent or future sightings that might be the missing vessel, and EXCOMs are repeated on a regular basis.[16]

If none of these communications and investigation efforts produces positive results (i.e., locating the vessel or indications that the persons on board are not in immediate danger), a search is undertaken. Search planning is conducted by the RCC staff, but additional assets can be requested from other agencies (i.e., U.S. Air Force and Navy) or foreign governments in a position to assist. With assistance from the USCG's computer-assisted search planning (CASP) system, the RCC develops scenarios based on the available information. These scenarios are then weighted according to a subjective estimate of how likely each one is to represent the true situation. The further analysis of available information leads to development of probability maps (using CASP), after which a search is planned and orders are issued to all participating units. The search continues until either the survivors are found and rescued, or it is deemed that further searching would be fruitless.[11]

Because SAR regions are not construed as boundaries to effective SAR action, and because the aeronautical SRRs are surrounded by the maritime SRRs, coordination between the AFRCC and the Coast Guard Rescue Coordination Centers is a daily occurrence. Missions that involve portions of both regions will be coordinated through the AFRCC or the appropriate Coast Guard RCC. It is not unusual for the Coast Guard to call upon the AFRCC for a particular resource needed to prosecute a mission in the maritime region or, conversely, for the AFRCC to utilize a Coast Guard resource in the aeronautical region.

Additional information about the USCG can be found at http://www.uscg.mil/USCG.shtm.

Federal Aviation Administration. The Federal Aviation Administration (FAA), through its Air Route Traffic Control Centers (ARTCCs) and Flight Service Stations (FSSs), monitors and flight-follows aircraft filing flight plans in the aeronautical SAR regions. In some cases, individual citizens contact an FAA facility when they have knowledge of a probable SAR situation involving aircraft. Therefore, the FAA is usually the first agency to alert the AFRCC of a distressed or overdue aircraft. The AFRCC is tied directly into the FAA's computer network, and FAA facilities use this system to alert the AFRCC.

Once the AFRCC is alerted, the FAA and AFRCC work together to determine the urgency of the situation and locate the aircraft. Initially, radio communications are reviewed to determine the last known location of the distressed aircraft. Concurrently, other FAA facilities begin a check of all possible airports where the aircraft might have landed. In the meantime, the AFRCC contacts relatives, friends, and business associates of the pilot or passengers aboard the missing aircraft, with the hope of establishing the whereabouts of the aircraft, or to gather information about the personnel aboard. Through these contacts, the AFRCC determines the pilot's intentions, flying capabilities, emergency equipment aboard, and other pertinent information that would assist if a search were to become necessary. Through experience, the FAA and AFRCC have learned that most alerts for missing aircraft are due to the pilot failing to either close the flight plan or inform some person or agency of his or her intentions. For this reason, only a small percentage of alerts issued by the FAA result in an actual airborne search for a missing aircraft.

All ARTCCs have the capability to recall recorded radar data. The National Track Analysis Program (NTAP) can identify and track targets that are at a sufficient altitude to be tracked by radar regardless of whether they are being controlled by the ARTCC. NTAPs requested by the AFRCC have been proved to be a key ingredient in aircraft searches, providing the route of flight and last radar position.

With the congressional mandate requiring most aircraft to be equipped with an ELT, the FAA works very closely with the USMCC and the AFRCC to readily locate the source of ELT signals. All ELT signals reported to FAA facilities are immediately forwarded to the AFRCC and jointly investigated as probable distress signals.[41]

Civil Air Patrol. In 1948, the CAP was permanently chartered by the U.S. Congress as the official auxiliary of the U.S. Air Force. As such, this nonprofit organization of volunteers was charged with three primary missions: the development of aviation through aerospace education, a cadet youth program, and emergency services. As of this writing, the CAP boasts more than 64,000 members, including more than 27,000 cadets between the ages of 12 and 20 years; the world's largest fleet of single-engine, piston aircraft; and access to 1000 emergency service vehicles. It is proud of the fact that it saves 100 lives per year.[4]

Under their emergency services mission, the CAP provides SAR mission coordinators, search aircraft, ground teams, personnel on alert status, and an extensive communications network to emergency response efforts. Further, they provide services to national relief organizations during a disaster; transportation of time-sensitive medical materials (e.g., blood and human tissue); and aerial reconnaissance, airborne communications support, and airlift of law enforcement personnel in the national counter-drug effort. When CAP resources are engaged in a SAR mission, they are reimbursed by the U.S. Air Force for communications expenses, fuel and oil, and a share of aircraft maintenance expenses. In addition, CAP members are covered by the Federal Worker's Compensation Act in the event of an injury while participating in a SAR mission.

The CAP is the AFRCC's prime air resource for the inland area. The AFRCC maintains an alert roster provided by CAP wings in each of the 48 contiguous states and is the central point of contact for CAP participation in SAR missions. The AFRCC also works closely with CAP national headquarters and directly provides input for CAP training in emergency services.

U.S. Coast Guard Auxiliary. The USCG Auxiliary is to the USCG as the CAP is to the U.S. Air Force. The auxiliary is made up of citizens who volunteer their time and boats or aircraft to enhance and maintain the safety of boaters. Passage of the Auxiliary and Reserve Act of 1941 designated that civilian volunteers of the USCG be referred to as auxiliary. When America entered World War II, some 50,000 auxiliary members joined the war effort. After the war, their attention returned to recreational boating safety duties in compliance with the auxiliary's four cornerstones: vessel examination, education, operations, and fellowship. Today, as in 1941, auxiliarists are civilian volunteers whose activities are directed by policies established by the commandant of the USCG. Although under the authority of the commandant, the auxiliary is internally autonomous, operating on four organizational levels (smallest to largest): flotilla, division, district regions, and national.

When auxiliary resources are engaged under USCG "orders," they are reimbursed by the USCG for communications expenses, fuel and oil, and a share of vessel and aircraft maintenance expenses. In addition, auxiliary members are covered by the Federal Worker's Compensation Act in the event of an injury while participating in an authorized mission.

Many members of the auxiliary spend their weekends providing free boating safety courses to the public and free courtesy safety inspections to boaters. However, members also respond to minor SAR incidents, and the local USCG Station, Group, or District RCC coordinates their activities. Some auxiliarists have also become qualified to work in the RCCs or assist regular USCG facilities with regulating and patrolling regattas and other maritime events.[39]

With its 36,600 members, the auxiliary saves hundreds of lives each year, in addition to assisting thousands of boaters, performing courtesy marine exams, teaching public and youth classes, and assisting the USCG in both administrative and operational missions.[39]

The State's Role in SAR: Coordination and Support

All states have passed legislation that provides for direct support to local government entities during emergencies or life-threatening situations, and most states have a specific agency responsible for overall coordination and support for local SAR problems. This support can take many forms, but most often it is in the area of coordination and "one-stop shopping" for resources. Each state must establish an agency or central location that is familiar with all aspects of emergency management

and the resources available to aid in life-threatening situations. Many of these resources belong to the state and can be used to aid local jurisdictions.

A number of states, especially in the Northwest, have designated a state agency to be responsible for directing and coordinating air SAR activities. These state departments, or divisions of aeronautics, develop and maintain aviation SAR response programs with cooperation and support from local and federal agencies. Experience shows that this system usually works better than those in other areas of the country that rely on the federal government to initiate and carry out aircraft SAR activities.

If a local emergency manager, sheriff, or fire chief requests outside assistance in the form of specialized teams, search dogs, air support, or enhanced communications, the state agency for emergency services or emergency management can in most cases locate the nearest resources available and coordinate the response. If any federal resources are needed, such as air support or military personnel, the state agency provides a direct link to that resource. For instance, the AFRCC at Langley Air Force Base in Virginia has working agreements with all states that are updated annually. Technically, the resources of local and state governments must have been exhausted or be unable to perform a task before federal support can be rendered. However, policy provides for immediate aid when time is critical and in life-or-death situations. Much discretion is given to military installation commanders regarding aid to civilian authorities, as long as the primary (military) mission of the resource is not impaired. In fact, most commanders appreciate the opportunity to fly actual missions. Access to these resources must be gained through the state and AFRCC.

Every state's emergency management agency is responsible for providing support, guidance, training, and coordination to local political subdivisions within that state. As such, it produces a vital behind-the-scenes effort to help local jurisdictions prepare for emergencies, including SAR. The state also initiates the laws necessary to enhance effective actions for SAR response. Such legislation often indemnifies volunteer SAR teams, provides their medical coverage and insurance, and in some cases replaces personal property lost during SAR work. Although most volunteers work willingly until the job is done, this recognition and coverage by the state often provide additional incentives for volunteer participation.

Local SAR Response

The official response to the call for a wilderness SAR situation is usually delegated to a political subdivision within the state. The legal responsibility for SAR is generally vested with the county sheriff or chief law-enforcement officer at the local level, but this varies by region and state. In some cases, it is the responsibility of state police agencies, and in others, it belongs to land management agencies. The SAR response for one jurisdiction may differ greatly from that of another. For instance, many national parks in some areas of the country handle all of their own SAR incidents. Others jointly manage the function, whereas some rely entirely on outside resources. National forest land is managed solely by forest service personnel, but when it comes to SAR, the forest service usually only supports the functions of the local responders.

In urban and suburban areas, police officers, firefighters, emergency medical technicians, and emergency management organizations maintain some degree of disaster and emergency readiness through daily missions that involve SAR work. Fire departments have historically been responsible for rescue and response to emergencies within certain geographic or political areas, and volunteers augment many departments. Law enforcement agencies also maintain full-time, efficient response systems designed for their particular SAR requirements. Ambulance and rescue vehicles operated by a variety of private enterprises and volunteer organizations augment existing local government services. Through local emergency response planning and coordination, these services respond to a spectrum of everyday emergencies, including fires, collapsed buildings, hazardous material spills, vehicle extrications, and home medical emergencies.

County sheriffs, reserve law enforcement, volunteer fire departments, and a variety of volunteer and rescue units have been established to address local SAR problems. Delivery of SAR aid to rural and wilderness areas often presents many special logistic problems, which may be compounded by distance, terrain, and weather. The demand for wilderness SAR is often seasonal and unpredictable. Volunteer mountain rescue units, Explorer SAR groups, SAR dog teams, CAP squadrons, motorized units, and many types of volunteer composite teams (i.e., teams that have a variety of capabilities) are usually formed locally in response to the type and nature of recurring SAR problems. Regardless of what type of SAR emergencies occur, local resources and effort must be developed because they are closest to the problem. State and federal resources are subject to problems with time lag, distance, weather, logistics, and bureaucracy. The same storm or disaster that incapacitates a local area may also prohibit outside (and sometimes inside) emergency response and resupply.

Although official agency responses differ greatly around the country, one major factor remains constant—the dedicated and unfailing willingness of volunteers to respond and work until the job is done. The volunteer effort in SAR nationwide is the backbone of aid to people in distress, as is stated in the rescue service motto: "These Things We Do That Others May Live." The volunteer response has proved crucial to wilderness-type situations. Volunteer organizations, communications, and special skills cannot be replaced by any "official agency" resources.

ORGANIZATION OF A SEARCH AND RESCUE EVENT

SAR requires people who take action and meet objectives to achieve a common goal, often with one or more lives in the balance. For any combination of actions to be effective in a particular situation, the enterprise must be systematically coordinated and organized. All participants must know their responsibilities, what is expected of them, who is in charge, and to whom they answer. If this knowledge is lacking at any level, the effort can quickly become chaotic, ineffective, and, very probably, dangerous. Nowhere are these issues more important than in an emergency, when time is a crucial factor.

Emergency response research is clear and specific. The four operational problems that continue to arise during emergency responses in the United States are ambiguity of authority, inability to communicate between agencies, poor use (or no use) of specialized resources, and unplanned negative interactions with the news media. Accordingly, the key elements for success in SAR operations continue to be good coordination of resources

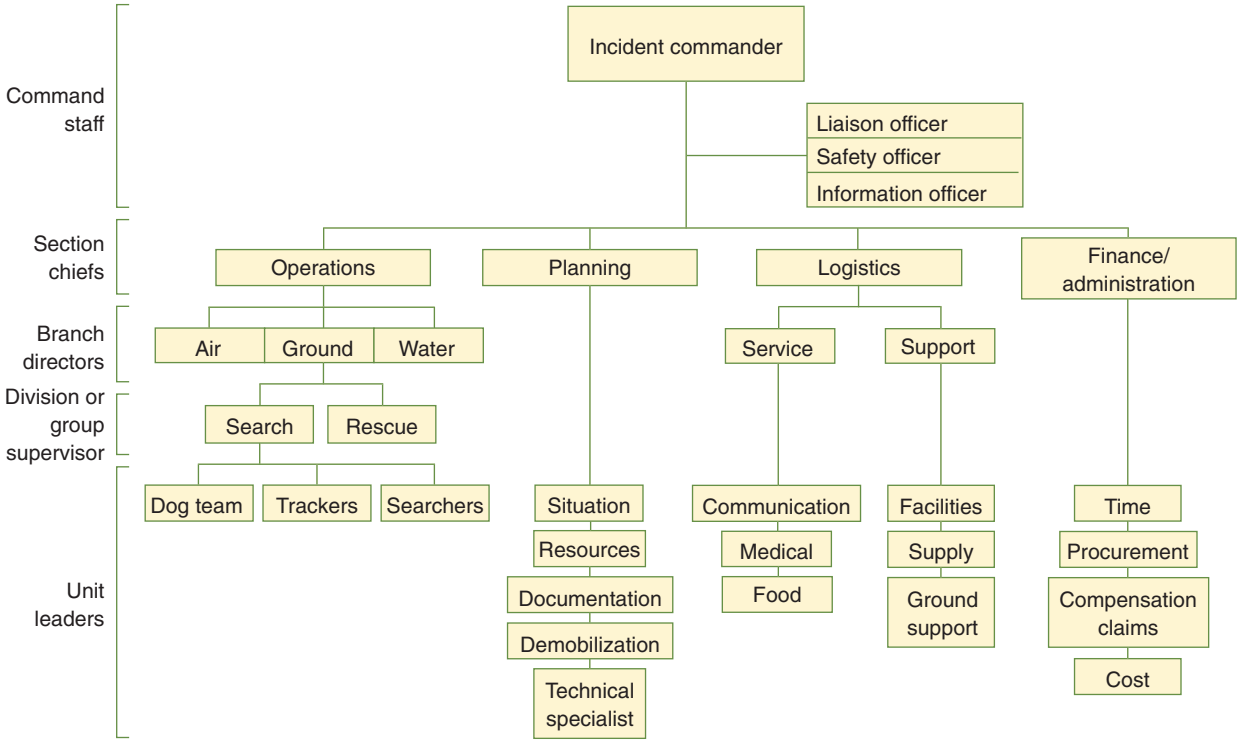


Figure 32-1. Functional hierarchy of the Incident Command System commonly used in SAR in the United States. (From Cooper DC [ed]: FUNSAR: Fundamentals of Search and Rescue. Chantilly, VA, Jones & Bartlett, 2005.)

(the right people and equipment in the right place at the right time), effective communications, and good management practices with trained leaders.[24]

Incident Command System

The system designed to address the challenges of managing emergency incidents in the United States, including SAR, is called the Incident Command System (ICS). It has been in use in the United States for many years.[31] This function-based system was designed to be adaptable to various types and sizes of incidents in a proactive, rather than a reactive, manner. The system groups similar tasks into five functional areas: command, operations, planning, logistics, and finance/administration. Each of these functions is performed at every incident to one degree or another, and all can easily be expanded as the size and complexity of the situation dictate. This expansion, however, is based on the premise that the span of control (the ratio of the subordinates to each supervisor) should never exceed seven to one and should more commonly be five to one. When this is exceeded, another level is added to the hierarchy to maintain an acceptable span of control (Fig. 32-1).

The command section is led by the incident commander and provides overall management of the organization. Within ICS, the command section is responsible for dealing with other agencies (liaison officer), the news media, and other external influences (information officer) and is responsible for the overall safety of the operation and its participants (safety officer). If the incident is too small for these functions to be performed by separate individuals, the incident commander performs them.

The operations section is led by the operations section chief, who is responsible for coordinating and performing all tactical operations. This role is commonly performed by the incident commander until the incident becomes large and complex enough that the function must be performed by another individual. When multiple casualties are involved in an incident, their triage, treatment, and transport fall under the purview of the operations section. In such an incident, the operations section is divided into functional groups, often including at least triage, treatment, and transport groups. The person in charge of managing and coordinating the efforts of each group is called the *group supervisor.* If the operations section is better divided using geography, a division rather than a group is formed. For instance, injured persons at an auto accident might be found on two sides of a road. An east division and a west division might be established to deal with the geographic separation of the resources. In a small organization, the supervisor of each division would answer directly to the operations chief.

To respond to specific challenges within an incident, a task force or strike team might be formed. A task force is any combination of single resources assembled for a particular tactical need, with common communications and a leader. For instance, the Federal Emergency Management Agency combines search, rescue, medical, and technical resources to form an urban search and rescue task force. A strike team, on the other hand, is a combination of a designated number of the same kind and type

of resources with common communications and a leader. The number of resources used in the team is based on what is needed to perform the function. For instance, four three-person hasty search teams may be combined to form a strike team. These two combinations of resources permit the necessary flexibility when allocating resources.

The planning section is led by the planning section chief, who is responsible for collecting, evaluating, and distributing all incident information. As with the other sections, the incident commander performs this function unless the size and complexity of the incident dictate otherwise. In SAR, the planning section is particularly important because it evaluates search evidence and determines, based on what has been learned, what future actions should be taken or how current actions should be modified. Because such interpretation and evaluation often require great technical knowledge, personnel such as hazardous materials specialists, physicians, structural engineers, and other technical specialists may be required to help the planning section develop and revise the incident action plan.

The logistics section is led by the logistics section chief, who is responsible for providing personnel, equipment, and supplies for the entire incident. This awesome task involves ensuring that personnel are available, rested, and fed; that all equipment, including communications equipment, is available and operable; that vehicles are fueled and repaired; and that medical care is provided for all incident personnel. Basically, logistics is charged with seeing that the physical tools required to meet the overall objectives are available, operable, and maintained.

If the size and complexity of the incident prevent the incident commander from monitoring finance and administrative issues, the finance/administration section is led by the finance/administration section chief. This section is responsible for tracking all financial data for the incident, such as personnel hours, resource costs, costs for damage survey, and injury claims and compensation.

Because most agencies involved in SAR can usually handle financial issues on their own, and most incidents are small and of short duration, the incident commander usually performs the functions of this section. Only in the largest or most complex incidents is it necessary for the incident commander to assign an individual or staff to perform finance section duties.

National Incident Management Systems

In Homeland Security Presidential Directive-5 (HSPD-5), the President of the United States called on the DHS to develop a national incident management system (NIMS) to provide a consistent nationwide approach for federal, state, tribal, and local governments to work together to prepare for, prevent, respond to, and recover from domestic incidents, regardless of cause, size, or complexity. On March 1, 2004, after collaboration with state and local government officials and representatives from a wide range of public safety organizations, DHS issued the NIMS. It incorporates many existing best practices into a comprehensive national approach to domestic incident management, applicable at all jurisdictional levels and across all functional disciplines. Among these best practices, and a key component of the NIMS, is the incident command system, which has been established by the NIMS as the standard organizational structure for the management of all incidents.[5]

The NIMS represents a core set of doctrine, principles, terminology, and organizational processes to enable effective, efficient, and collaborative incident management at all levels. To provide the framework for interoperability and compatibility, the NIMS is based on a balance between flexibility and standardization. The recommendations of the National Commission on Terrorist Attacks upon the United States (the "9/11 Commission") further highlight the need for the NIMS and the importance of ICS. The Commission's 2004 report recommends national adoption of the ICS to enhance command, control, and communications capabilities.

FOUR PHASES OF A SEARCH AND RESCUE EVENT: THE INCIDENT CYCLE

Every SAR event goes through four consecutive phases: locate, access, stabilize, and transport (LAST).[5] This sequence, however, could more accurately be described as a continuum that begins with planning or preplanning for the incident. Because planning for the next incident should be affected by what happened during the last, the incident cycle is actually continuous and only pauses between incidents. Once first notice of an incident has been received and the locate phase begins, the goal is to progress through the access, stabilize, and transport phases as quickly, safely, and efficiently as possible. Planning between incidents allows decisions to be made in a calm environment without the urgency that often accompanies a SAR operation. Such plans identify who will be in charge, the organization of the operation, specific procedures, viable alternatives, and other decisions that are best made before an incident occurs (Fig. 32-2).

Locate Phase

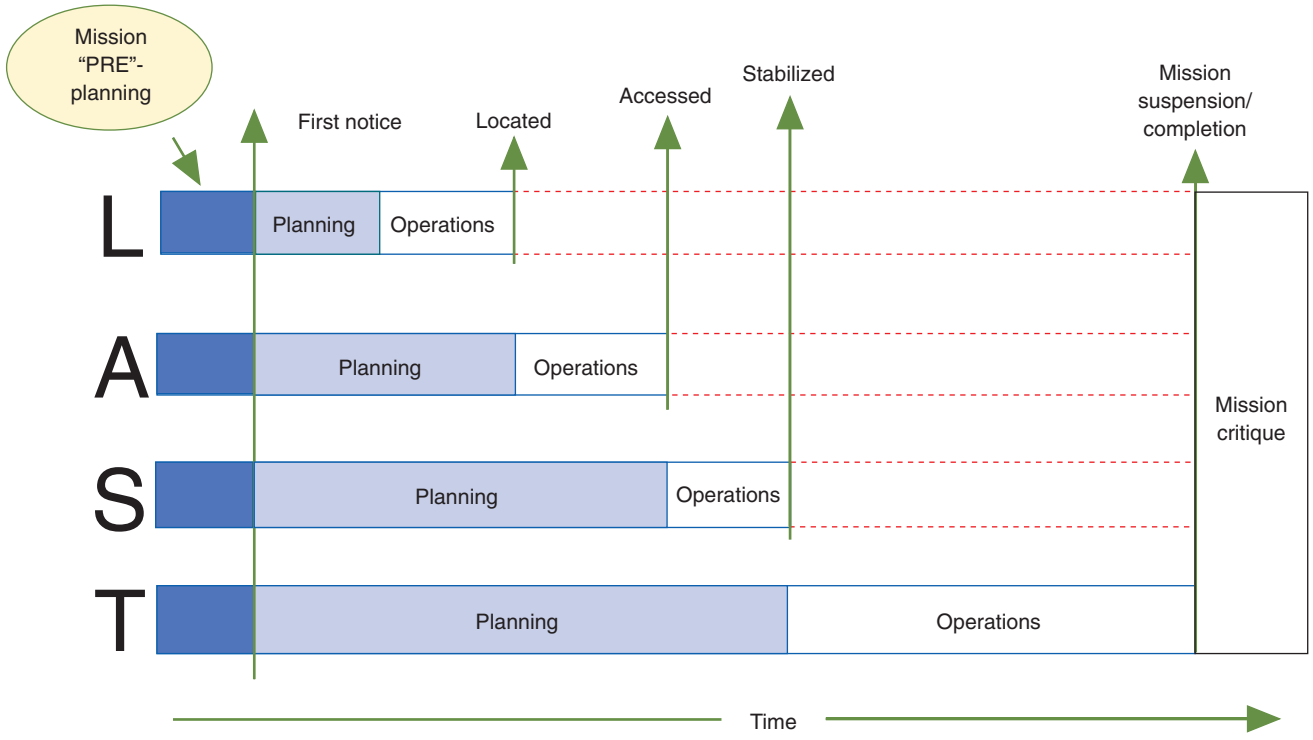
The first step in addressing any emergency situation is locating the subject or subjects in need of assistance. This may be as simple as asking for an address or as complex as conducting an extended search for a lost person or persons. If the subject is easily found, rescuers can quickly move into the access phase. However, if locating the subject is difficult, this phase may turn into the crux of the SAR problem.

First Notice

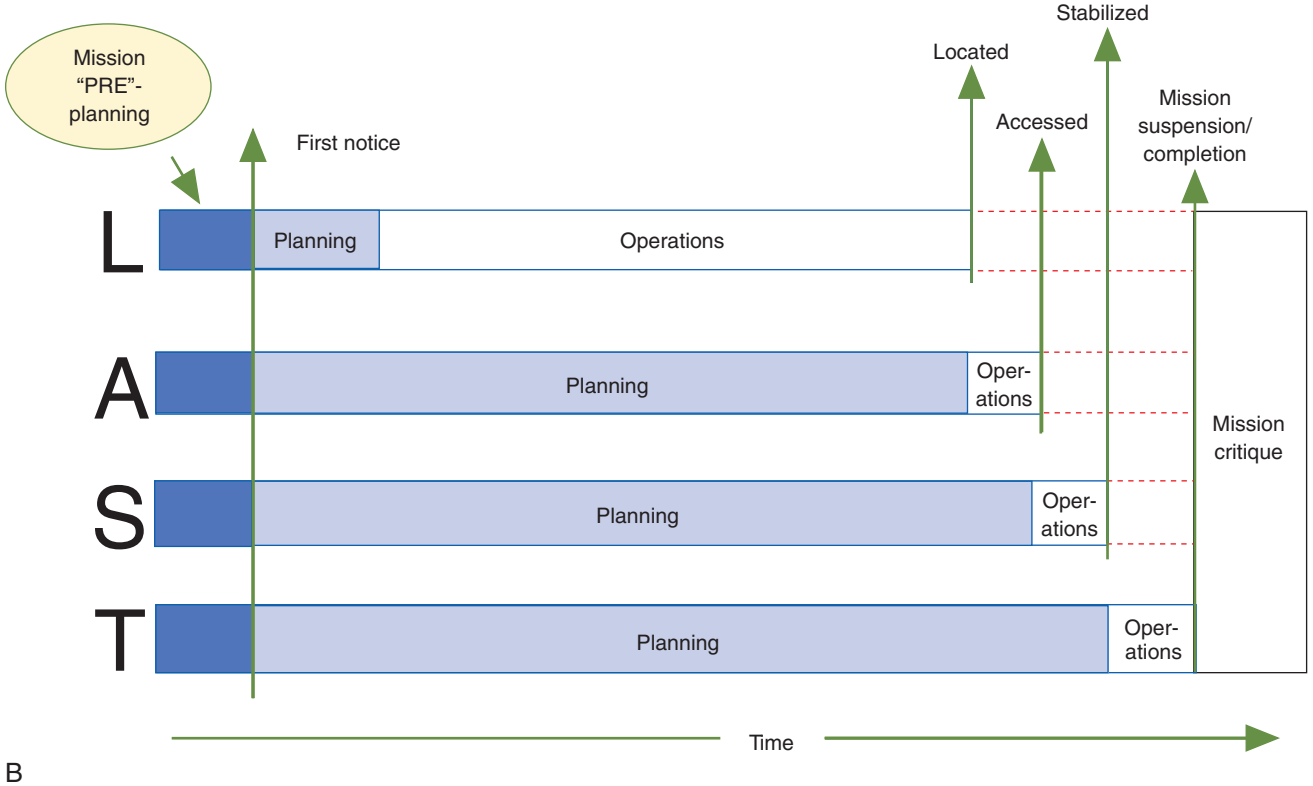
The first notice of an incident is often conveyed by relatives who report an injury or missing person, by a witness to an incident, by a government agency reporting distress signals (such as an ELT), by bystanders who perceive a problem, or by a 911 call. Once the initial notice is received, the individual taking the information must know what to do and whom to call next.

Planning Data and Its Uses

Information gathered at the onset of an incident begins an ongoing investigation. It is used to determine the appropriate response and to help predict how the subject or subjects might react to the situation. This information is called *planning data* and includes any information that might affect what should be done to resolve the situation. Examples of planning data include name of the subject, situation that caused the problem, last known location of the subject, subject's physical and mental condition, subject's plans (where was he or she going?), available resources, weather information (present and predicted), geographic information, and history of similar incidents in the area. The purpose of collecting all this information is to help decide what to do next while predicting what the subject might do to help or hinder the situation.



A



B

Figure 32-2. The time-specific components of a SAR event vary with the type of incident. Note that all components take place in both incidents but require different amounts of time. **A,** Typical rescue operation. **B,** Typical search operation.

The investigation and gathering of information continue throughout the incident and are used to modify initial plans. As new information is acquired, an action plan is developed and revised until the end of the incident cycle, when planning for the next incident commences.

Once information is gathered, the urgency of the situation is assessed. This assessment ultimately determines the speed, level, and nature of any response and may indicate whether a nonurgent or an emergency response is needed. The specific information used in urgency determination includes the age and condition of the subject, current and predicted weather, and relevant hazards. Figure 32-3 is an Urgency Determination Form, which can be used by SAR personnel to determine how urgent their response should be.[5] Urgency also contributes to allowable risks and thus influences searcher safety—a primary consideration for search managers.

Search Tactics

During the initial locate phase of the incident, emphasis is on searching for the subject. Exactly how to accomplish this is a priority, especially if this part of the incident cycle is expected to be a problem. SAR managers first initiate techniques that increase the chances of locating the subject in the shortest time. These techniques are generally termed *tactics* and involve some action performed to find the subject. These actions can be indirect (e.g., not requiring actual field searching) or direct (e.g., requiring deployment of searchers to the field). Examples of indirect tactics include confining the search area to limit movement of the subject and others into and out of the area, identifying and protecting the point last seen (PLS) or the last known position (LKP), and attracting the subject, if he or she is expected to be responsive.

Generally, indirect techniques are quicker and easier to apply, so they are started first. As the incident progresses, direct tactics are initiated. In managing an incident, efforts are almost universally made to apply quick response resources in areas likely to offer early success. The best resources are put in the most likely areas as early as possible. In addition, identifying and protecting the PLS or LKP are crucial indirect techniques that can mean the difference between success and failure of the entire effort.[5]

Direct techniques include sending teams of searchers into an area to search for clues or the subject. They are categorized by level of thoroughness. For instance, a fast, relatively nonthorough search of high-probability areas is called a *hasty search*. This type of search would be conducted at camp sites, buildings, and other very likely locations where the subject might be found. Loose grid techniques—widely spaced searches of large areas—can be applied when relatively rapid searches of large areas are desired. When using these techniques, thoroughness is greater than with hasty searches, but less than with other slower, more resource-intensive techniques. Loose grid techniques allow some level of effort to be applied to large areas fairly quickly. Alternately, tight grid techniques—slow, highly systematic area searches—are applied only when the absolute highest level of thoroughness is required. Unfortunately, this is almost always at the expense of time and resources. In short, the greater the thoroughness, the more resource intensive and time consuming the technique.

Clues and Their Value

Clues are discovered during the investigative and tactical phases of a search. Their importance cannot be overemphasized. They may take the form of physical evidence, such as a footprint or discarded item, account by a witness, or information gleaned from the investigation. Clues serve as the rudder that steers the overall search operation. Relevant clues are the basis for all search strategies and can determine or modify nearly all operational actions. Their powerful influence should be obvious; this is why searchers are taught to be "clue conscious" and to seek clues, not just subjects. There are many more clues than there are subjects.

People generate clues. A person exudes scent in the form of dead skin cells, crushes or disturbs vegetation, and, when traveling, leaves marks on the ground or other physical evidence of passing. This evidence is often discoverable if the appropriate resource is applied in a coordinated, organized search effort. Searchers must be sophisticated enough to discover this evidence and interpret its meaning before it is destroyed or decays. Because evidence important to a search effort is often easily destroyed once it is discovered, it is important to protect it from damage until it is completely analyzed.

Search Resources

Resources are defined as all personnel and equipment available, or potentially available, for assignment to support the search effort. Specific types of active tactics are categorized by the resource that performs them, such as dog teams, human trackers, ground search teams, and aircraft. Other common resources include management teams (e.g., overhead teams, public information officers), water-trained responders (e.g., river rescue, divers), cold weather responders (e.g., ice climbers, avalanche experts, ski patrollers), specialized vehicle responders (e.g., snowmobiles, four-wheel-drive trucks, all-terrain vehicles, mountain bikes, horses), and technical experts (e.g., communications experts, interviewers, chemists, rock climbers, physicians, cavers). In addition to these, other less common resources might also be available. These could include attraction devices (such as horns, flags, lights, sirens), mine detectors (military), noise-sensitive equipment (super microphones), infrared devices (forward-looking infrared [FLIR] on aircraft, night-vision equipment, thermal imagers), thermistors, and even witches, seers, prophets, and diviners.

Just about any person or thing imaginable may be available for use in a SAR incident. Their use is limited only by the creativity of those in charge. Here we discuss a few of the most common and useful.

Dogs. Dog teams are a common type of active search resource in the wilderness and are composed of a dog (occasionally more than one) and a human handler. The dog uses scent to search for and follow a subject while the handler interprets signals from the dog and searches visually for evidence. Three common categories into which dogs fit are tracking, trailing, and air-scenting.

Tracking dogs follow scent on the ground from a person's footsteps and usually very closely follow the trail where a person traveled, regardless of the wind. Trailing dogs follow scent that has fallen onto the ground from the subject along the route of travel. Unlike the tracking dog, the trailing dog may follow the scent at some distance from the actual tracks of the subject, and may therefore be more affected by wind. Tracking and trailing dogs are most effective when used in areas that have not been contaminated by humans other than the subject. Also, weather and time tend to destroy scent available to these types

Search Urgency

Remember: the lower the number, the more urgent the response!!!

Date Completed: _____
Time Completed: _____
Initials: _____

A. SUBJECT PROFILE ..... _____

Age

Very Young	1
Very Old	1
Other	2-3

Medical Condition

Known or suspected injury or illness	1-2
Healthy	3
Known fatality	3

Number of Subjects

One alone	1
More than one (unless separation suspected)	2-3

B. WEATHER PROFILE ..... _____

Existing hazardous weather	1
Predicted hazardous weather (8 hours or less)	1-2
Predicted hazardous weather (more than 8 hours)	2
No hazardous weather predicted	3

C. EQUIPMENT PROFILE ..... _____

Inadequate for environment	1
Questionable for environment	1-2
Adequate for environment	3

D. SUBJECT EXPERIENCE PROFILE ..... _____

Not experienced, not familiar with the area	1
Not experienced, knows the area	1-2
Experienced, not familiar with the area	2
Experienced, knows the area	3

E. TERRAIN AND HAZARDS PROFILE ..... _____

Known hazardous terrain or other hazards	1
Few or no hazards	2-3

TOTAL ..... _____

If any of the seven categories above are rated as a one (1), regardless of the total, the search could require an emergency response.

•••THE TOTAL SHOULD RANGE FROM 7 TO 21 WITH 7 BEING THE MOST URGENT.•••

8-11 Emergency Respone *12-16 Measured Response* *17-21 Evaluate and Investigate*

Figure 32-3. Urgency Determination Form. (From Cooper DC [ed]: FUNSAR: Fundamentals of Search and Rescue. Chantilly, VA, Jones & Bartlett, 2005.)

of dogs, so the earlier they are used in a search, the better their chances of finding something.

Air-scenting dogs work off-lead to follow a subject's scent to its source. Specifically bred and trained air-scenting dogs can even discriminate between individual humans. They may detect scent from articles of clothing and can often follow it to discover a person buried in rubble or snow or even submerged under water. Wind is very important to this type of dog, as are other environmental forces, such as sun and rain. But as long as the source exists, an air-scenting dog can usually detect the scent carried in air currents and follow it to the source.

Human Trackers. Human trackers use their visual senses to search for evidence left by a person's passing. Human trackers "cut" or look for "sign" or discoverable evidence by examining the area where the subject probably would have passed. This process of looking for the first piece of evidence from which to track is called "sign cutting." Following the subsequent chain or chronology of sign is called "tracking."[9]

In SAR, most trackers use a stride-based approach called the step-by-step method. This simple, methodical approach emphasizes finding every piece of possible evidence left by a subject. However, its most important role is undoubtedly the ability to quickly determine the direction of travel of the subject and thus limit the search area.

Ground Search Teams

Hasty Teams. A hasty team is an initial response team of well-trained, self-sufficient, highly mobile searchers whose primary responsibility is to check out the areas most likely to produce the subject or clues first (e.g., trails, roads, road heads, campsites, lakes, clearings). Their efficiency and usefulness are based on how quickly they can respond and the accuracy of initial information.

Ideally, hasty teams should include two or three individuals who are knowledgeable about tracking. They should be clue oriented, familiar with the local terrain and dangers in the area, and completely self-sufficient. Also necessary are the ability to skillfully interview witnesses and to use navigational skills with pinpoint accuracy. Team members should at least be trained in advanced first aid. Hasty teams usually operate under standard operating procedures, so they do not have to wait for specific instructions. They carry all the equipment they might need to help themselves and the lost subject for at least 24 hours.

Grid Teams. Grid searchers use a more systematic approach to searching. They usually examine a well-defined, usually small segment to discover evidence. The classic approach to grid searching involves several individuals (almost always too many) standing in a line, shoulder to shoulder, walking through an area in search of either evidence or subjects. The distance between searchers can be varied to change thoroughness and efficiency (wide spacing is less thorough and more efficient). However, such resource-intensive approaches to searching are generally less preferred than those that use fewer personnel in a more efficient manner (such as tracking, dogs, or aircraft). In addition, close-spaced grid searching tends to damage evidence and is generally difficult to coordinate.

Although grid searching may be an acceptable approach in certain limited circumstances, experience has shown that when the subject of a search is a person, searching in this thorough manner should be used only as a last resort. Experiments involving grid searching have suggested that it is better to place searchers farther apart. This is usually a more efficient use of resources.

Aircraft. Aircraft serve the same purpose as grid searchers, only from a greater distance, at a greater speed, over a larger area, and usually with a lower level of thoroughness. Within a search effort, aircraft can serve both as a tactical tool to look for clues and as transportation for personnel and equipment. They also provide an excellent attraction device for the missing subject as well as witnesses who may have information about the incident. Both fixed- and rotor-wing aircraft have their place in SAR and, like other resources, have their advantages and limitations. Among the most obvious limitations are the expense and complex use requirements. Aircraft not only require specialized personnel and cost a great deal to operate, they also have very strict weather and environmental restrictions. For instance, it would be difficult to search from an aircraft in a snowstorm, and terrain may prevent searching certain areas from the air. However, most of these difficulties can be adequately addressed and minimized in a well-developed preplan.

Search Planning Considerations

State-of-the-art searching for lost persons has come a long way from the familiar lining up of volunteers shoulder to shoulder and walking in a straight line to search an area. Many new life-saving concepts have been developed by the national SAR community. By borrowing from psychology, mathematics, business, and the analysis of past incidents, search planning and management have evolved into sophisticated sciences. Search effectiveness has also been improved through the study of human behavior, statistics, probabilities, leadership, and the use of good planning and management principles (Fig. 32-4).

Search planning is guided by two general considerations: where am I going to look for the lost or missing person (strategy), and how am I going to find this missing or lost person (tactics)? To be effective, modern searchers follow several basic principles and techniques that include the following:

Figure 32-4. Map used to brief ground team. (Photo courtesy of D. Maynard.)

1. Respond urgently—a search is an emergency.
2. Confine the search area.
3. Search for clues.
4. Search at night.
5. Search with a plan and in an organized manner.
6. Grid search (tight grid) as a last resort.

Every day, emergency responders receive calls to perform their duties, and they never know what to expect once they arrive. Life-threatening or time-critical situations have engendered what is known today as the "firehouse response." This means that emergency responders have to assume the worst until proved otherwise. They respond with "lights and siren" to most calls just in case the situation is serious. Unfortunately, people who report incidents are often wrong in their initial assessments, and because of this, every call must get the same urgent response. Essentially, the situation is considered an emergency until proved otherwise.

For years, searching has been considered less urgent than other emergencies. Although responders were running with lights and sirens to situations reported as "women not feeling well" or "dumpster on fire," reports of a missing/lost child or an overdue hiker were relegated to the "let's wait and see" category. Through years of experience, search managers now know that a search is as much an emergency as any other call for help. Experience also indicates that an urgent response to a search situation often leads to a faster resolution with less impact on family, and to the expenditure of much less effort overall. Any search situation for a lost or missing person should be considered an emergency that justifies an urgent response, high priority, thorough assessment, and immediate action.

Search Theory. The mathematical basis for searching and study of search theory had its beginnings during World War II in the work of the U.S. Navy's Anti-Submarine Warfare Operations Research Group (ASWORG) and was originally based on searching for the wakes of warships as seen by aircraft flying over the ocean.[10] The results of this work were collected in a seminal report by B. O. Koopman in 1946,[22] but the report was not declassified and generally available until 1958.[2] In 1980, Koopman developed a somewhat expanded version of this work.[23] Although Koopman's work is clearly aimed at naval interests, the general theory of search he established is applicable to virtually any type of search problem. Since this early work, search theory has undergone continuous research and development by agencies such as the USCG and U.S. Navy in both the maritime and aeronautical environments, mining and oil businesses in search of mineral and petroleum deposits,[13] and even archeologists in search of lost cities, such as Troy.[33] The fundamental usefulness of search theory lies in its ability to help determine where and how to search. It accomplishes this by (1) quantifying the likelihood of a missing subject being in a particular area, as well as the likelihood of searchers finding the subject; and (2) offering tools with which one can estimate the chances of success of a particular search.

The application of search theory requires the appropriate use of probability theory, a branch of mathematics that is used for estimating the likelihood of uncertain events, in planning a search. The chance that the missing person is in the search area is called *probability of area* (POA). The probability that a search resource will find the missing person or clue if it indeed is in the area being searched is called *probability of detection* (POD). The product of these two important variables produces a result called *probability of success* (POS = POA × POD).[33] The foremost objective and major challenge for search planners is to deploy the most capable search resources (POD) into segments in which the subject is most likely to be (POA) to produce the most POS in the least amount of time. On the surface this seems to be a straightforward proposition. However, myriad critical factors, such as resource detection capability, environmental influences, search object characteristics, probability density, probability distribution, coverage, and sweep width, conspire to make the most correct search action a complex, unintuitive series of difficult choices.

Several initiatives have been undertaken to simplify the process of applying search theory to land search, including standardizing terms[6] and developing simplified, yet mathematically correct, approaches to quantifying the variables in less subjective ways.[7] Advanced methods have also been developed to optimize the increase in POS over time[3,34] and integrate motion and other models into land search problems.[42] However, user-friendly computer software will be required for such advances to enjoy widespread use by land search planners.

During the past several years, much has been done to improve how scientific search theory is applied to land search. Before 2001, much more research on the topic of search and detection had been conducted in the maritime environment than on land. In 2001, the NSARC Research and Development Working Group convened a meeting of land search experts.[30] After this meeting, several research projects sponsored by NSARC, and supported by several national and international organizations, were developed and funded. The first project produced a model for conducting detection experiments on land.[32] In 2004, using the model developed, five detection experiments were conducted across the United States in five different ecoregions.[1,21] The results should provide tremendous insight into methods of quantifying visual detection on land.

In 2004, NSARC, with funding from the Departments of Defense and Homeland Security, published a report that compared published land search procedures with the science of search theory. The full report identified several significant opportunities for improvement in multiple, long-practiced land search procedures and concepts.[8] Several of these reports are available on the NSARC website at www.uscg.mil.

Although software is available to assist in the mathematical decisions, less than optimum but historically acceptable results have been achieved when a search planner applies the principles of search theory as an intuitive combination of hard science and sage experience. In the final analysis, search success is based on more than just science. At its finest, it involves the artistic application of science and a high degree of organizational and management skill, sprinkled with intuition and punctuated with a bit of luck.

Lost Subject Behavior. Modern search management is also based on the use of what is called a complete "subject profile." Such a profile identifies as much as is known about the missing subject, including general state of health, past experiences, and state of mind, through the use of a form called the *Missing Person Questionnaire*. This information is collected and used by search planners to predict how an individual would react in various situations. Analysis of this information from past incidents and understanding how the involved individuals behaved in given circumstances have offered great insight for search managers.

It is important to note that most data on abductions, of both children and adults, have been purposefully excluded from SAR research. Although some important work has been done in this area (e.g., by Hanfland and colleagues[12]), the study of abductions and related criminal activity is often more about the perpetrator than the subject of the crime. This is in stark contrast to the study of lost subject behavior, for which the actions of the subject are the focus of the research.

Although it is not difficult to appreciate the importance of predicting how a subject might react when lost, the scientific approach to the topic began with Syrotuck's seminal paper in 1977.[35] This paper was based on the premise that individuals have similar travel habits when compared with others in the same "category." The six categories are small children (1 to 6 years), children (6 to 12 years), hunters, hikers, older adults (>65 years), and "miscellaneous adults," such as nature photographers, fruit gatherers, bird watchers, and other outdoor enthusiasts. There are two "special categories" (mentally retarded persons and despondents), for which there were very little data. In his study, Syrotuck described the behavioral characteristics of a representative member of each category and computed "probability zones" for each, based on distances traveled. This distance was measured "as the crow flies," or as a straight-line distance, between the point where the subject was last seen and the location where the subject was eventually found. Realizing that there was likely a substantial difference between how far a lost subject actually traveled and the crow's-flight distance, the author argued that, "it is more important to realize that a known percentage of all lost persons are found within a one- or two-mile radius than it is to know how they got there." Syrotuck studied 229 cases, most from wooded areas of Washington and New York states, and all involved subjects traveling on foot.[35]

Beyond identifying categories, Syrotuck[35] also documented and described six other factors that may affect the search plan. He suggested that search personnel in possession of the following information could more accurately predict the subject's location:

1. Circumstances under which the person became lost
2. Terrain
3. Personality
4. Weather
5. Physical condition at time of loss
6. Medical problems

He went on to describe how one's general state of health, past experiences, and physical situation (e.g., hot, cold, altitude) contribute to predicting behavior patterns. How one reacts to being lost, he also suggested, can affect the type and quantity of clues (i.e., disrobing, discarding equipment), survivability (i.e., failure to build a fire), detectability (i.e., bright clothing, bad weather), and tendency to follow travel aids such as rivers, roads, and trails. What Syrotuck produced was the first relatively scientific description of how people might react to being lost and how searchers could use this information to improve operations.

Following the theme of lost person behavior and using the crow's-flight distance, Koester and Stooksbury[18] performed a retrospective study of persons who suffered from what they termed "dementia of Alzheimer's type" (DAT) and who became the subjects of organized SAR efforts in Virginia. They studied 82 cases (initially) from the Virginia Department of Emergency Services' (DES) lost-subject database and compared the DAT patients' behavior to that of lost older adults who possessed normal cognitive abilities. Their findings were of great interest to search managers and planners in that this was the first time research of this type had been conducted for the inland SAR community. The authors also described a "subject profile summary" and suggested specific search techniques for lost DAT patients. Notable in their findings were the facts that none of the subjects in the cases they studied yelled for help, and they were usually found 0.5 miles (0.8 km) from the PLS. Since this initial research, Koester[19,20] has extended his analysis of the Virginia DES data (Tables 32-1 and 32-2).

TABLE 32-1. Summary of Findings for Lost Persons with Alzheimer's and Related Disorders

	TYPE OF SUBJECT				
STATISTIC	**ALZHEIMER'S**	**ELDERLY**	**DESPONDENTS**	**RETARDATION**	**PSYCHOTIC**
n	87	33	65	29	25
Age (standard deviation)	76 (9.2)	70 (4.3)	37 (15.7)	30 (3.3)	43 (15.9)
Males	67%	67%	76%	60%	63%
Females	33%	33%	24%	40%	37%
Uninjured	51%	48%	34%	85%	72%
Injured	27%	15%	11%	11%	5%
Deceased	22%	37%	55%	4%	22%
Distance from Point Last Seen (km/mile)					
Mean	1.0/0.62	2.9/1.8	2.2/1.36	1.4/0.87	2.2/1.36
Standard deviation	0.8/0.5	0.8/0.5	5.3/3.29	1.9/1.18	3.7/2.3
Median	0.8/0.5	0.8/0.5	0.3/0.19	0.8/0.5	0.8/0.5
Range	0–4.8/0–3.0	0–8.0/0–5.0	0–32.2/0–20.0	7.7/4.7	12.9/8.0
25%	0.3/0.19	0.2/0.124	0.2/0.124	0.2/0.124	0.2/0.124
50%	0.8/0.5	0.8/0.5	0.3/0.19	0.8/0.5	0.8/0.5
75%	1.1/0.68	4.0/2.48	2.6/1.61	1.6/1.0	2.0/1.24
Max. zone	2.4 (94%)/1.49	7.7 (95%)/4.7	8.0 (96%)/5.0	4.0 (95%)/2.48	7.7 (92%)/4.7

Data from Koester RJ: The lost Alzheimer's and related disorders search subject: New research and perspectives. Conference Proceedings, RESPONSE '98, Annual Conference of the National Association for Search and Rescue, Chantilly, VA, June, 1998, NASAR, pp. 165-181; and Koester RJ: Behavioral profiles of lost subjects in Virginia. Retrieved March 10, 2004, from http://www.dbs-sar.com.

TABLE 32-2. Subject Found Location Data

	TYPE OF SUBJECT			
	ALZHEIMER'S (AND RELATED DISORDERS)	DESPONDENTS	MENTAL RETARDATION	PSYCHOTICS
Structure	15%	8%	21%	23%
Yard (open field)	18%	4%	16%	—
Drainage	18%	8%	21%	7%
Woods	7%	33%	16%	30%
Brush/briars	29%	—	11%	7%
Road	7%	—	11%	23%
Power line/linear	—	13%	5%	—
Other	4%	8% (Cliff bottom)		

Data from Koester RJ: Conference Proceedings, RESPONSE '98, Annual Conference of the National Association for Search and Rescue, Chantilly, VA, June, 1998, NASAR, pp. 165-181; and Koester RJ: Retrieved March 10, 2004, from http://www.dbs-sar.com.

TABLE 32-3. Formal Estimates of Crow's-Flight Distance (in km) Between the Point Last Seen and the Point Found for Persons Lost During Different Wilderness Activities

	PERCENTILE			
ACTIVITY GROUP (N)	25	50	75	90
Campers (18)	0.722	1.559	3.001	4.931
Cross-country skiers: break trail (5)	4.537	9.795	18.860	30.988
Cross-country skiers: groomed trail (18)	0.842	1.819	3.501	5.753
Despondents (6)	0.229	0.656	1.793	4.664
Hikers (38)	1.691	3.650	7.028	11.548
Hunters (5)	1.222	2.638	5.079	8.345
Mountain bikers (18)	3.759	8.116	15.626	25.675
Scramblers (7)	1.165	2.515	4.843	7.958
Walkaways (14)	0.701	2.007	5.486	14.274
Other (13)	1.765	3.812	7.339	12.058

Note: Estimates for despondents and walkaways were based on a different Wakeby distribution than that used to estimate the percentiles of the other user categories.
From Heth DC, Cornell EH: J Environ Psychol 18:223-235, 1998.

Also using the crow's-flight distance, Hill[15] described distances traveled and probability zones for lost persons in Nova Scotia. However, Hill found it useful to modify and add to Syrotuck's categories of lost persons. For instance, Hill broke young people into four categories: children 1 to 3 years, children 4 to 6 years, children 7 to 12 years, and youths 13 to 15 years of age. He described characteristics for fishermen, skiers, and walkaways (i.e., people who walk away from a constant-care situation), and additional characteristics for those who are despondent.

Taking a slightly different approach, Heth and Cornell[14] published a study of 162 incidents of persons lost in wilderness areas in southwestern Alberta, Canada. They tabulated crow's-flight distance traveled and angular dispersion of travel (the angle from a line that connects the PLS with the intended destination) by different categories of wilderness users. They formed 10 categories of outdoor user (Table 32-3) and included only subjects propelled by muscle (no machinery). Interestingly, the authors found a behavioral distinction between "front country" users (i.e., front referring to parking lots, groomed trails, frequent signage, along with good, available maps; these areas attracted users with a large range of outdoor experience and skill) and "backcountry" users (i.e., remote, undeveloped areas; these areas attracted prepared and experienced users). Not unlike Syrotuck and Hill, Heth and Cornell discovered that, with the exception of despondents, there is a similar distribution of distance traveled by persons lost outdoors. However, they went further and suggested that there might be a linear relationship between certain data sets. For instance, their analysis indicated that hikers travel about 2.3 times farther than campers, and cross-country skiers breaking trail travel about 5.4 times farther than cross-country skiers using groomed trails. The implication is that if archival data are possessed for one category in one region and are compared with categories of lost subjects similar to those described by Heth and Cornell, a scalar parameter could be applied to extrapolate crow's-flight

distances for other subject categories. Such a possibility is exciting to search managers, who only rarely have access to relevant and reliable archival data.[14]

Search planners have used these and other behavioral studies in a number of valuable ways. By direct analysis and limited extrapolation, search planners have been able to find answers to important planning questions that are helpful in determining where and how to search. Such efforts have also taught search managers the importance of collecting behavioral data on lost persons, and the predictive value of such data.

Access Phase

After the subject is located, the search is over. Rescuers must now gain access to the subject to assess and treat injuries, evaluate the situation, and mitigate the problem. Accomplishing these objectives may be as simple as walking up a trail to reach the subject or as complex as reaching a climber on the face of El Capitan in Yosemite. Regardless, planning for this eventuality should be complete and ready to be carried out at the conclusion of the locate phase.

Once rescuers reach a subject, the situation and scene must be assessed. In emergency services terminology, this is called the *size-up*. The size-up consists of identifying hazards to the subject and rescuers, then developing a strategy to deal with the problems. For instance, a subject might be trapped by a winter storm in a high alpine environment. Safety considerations for rescuers entering such a hostile and dangerous environment would certainly influence further actions and may well take precedence over the entire rescue effort.

Specialized skills may be required for rescuers to safely gain access to the scene. For instance, rescuers may need to rappel to a patient who has fallen onto a ledge in the Grand Canyon, or they may need to climb sheer rock faces to reach an injured mountaineer on Half Dome in Yosemite National Park. These are examples of how complex the access phase of a rescue may be and point to the importance of thorough and proper planning.

If the size-up indicates that the situation or environment is so hazardous that remaining on scene poses an immediate threat to the subject, accelerated rescue techniques may be required. Accelerated rescue techniques are immediate actions required to remove a subject from a dangerous environment without stabilization. They often entail deviations from local standard operating procedures and protocols. Examples of such situations include poisonous gas environments (e.g., in caves), fires, unstable terrain (such as avalanches and rock slides), adverse weather (hurricanes, thunderstorms, severe snowstorms), or any hostile environment that threatens the subject, rescuers, or both.

Stabilize Phase

The stabilization phase has three primary components: physical, medical, and emotional. Once rescuers have access to the subject, the scene must be quickly evaluated, or sized up, for immediate physical hazards and threats from the environment. Scene safety is an initial priority in the size-up, and risks to everyone must be weighed against the benefits to be gained. An example of physical stabilization would include an occupied automobile teetering on the edge of a cliff. Before the occupant can be medically assessed, the situation (i.e., the automobile) must be stabilized to best protect the rescuers and the patient. Other examples of physical stabilization might include protecting the patient from further injury (e.g., removing him from the hazardous environment or applying a helmet) or removing the hazard (e.g., extinguishing the fire, securing the teetering auto).

Once the physical environment is stabilized and free from immediate hazards, medical management and stabilization can begin. This process usually follows accepted procedures, starting with primary and secondary physical examinations and progressing through basic and advanced life support. The process should include full-body immobilization, usually in a litter; specific site immobilization of fractures and related injuries; treatment of shock and other hemodynamic compromise; and without exception, some type of protection from the environment. The goal of medical stabilization is usually to prepare the subject for transportation to a definitive care facility. If medical care is not required, confirming this fact may be all that is necessary at this stage before moving into the transport phase.

Emotional stabilization is necessary because an anxious victim is a hazard to rescuers and himself or herself. Again, the goal is to best protect both the rescuers and the victim. Simple, calm communication with the patient, slowly describing what happened and what rescuers are doing, is often enough to calm a nervous subject.

Stabilization, like assessment, should continue throughout the transport phase. The overall objective is to prepare the victim for transport to definitive care while maintaining his or her comfort and safety.

Transport Phase

In the fourth phase of SAR, the subject is moved to definitive care. For this to occur, the stabilized subject must be "packaged" so that he or she can be moved safely and efficiently while stabilization and assessment continue. Transportation will range from foot travel, with the subject walking on his or her own, to evacuation by aircraft. If helicopter extraction is going to be used, the injured person must be briefed thoroughly on what to expect. This should include everything from the effects of the "rotor wash" to movement and noise. The appropriate mode of transportation can be determined by weather, type and severity of injuries, overall urgency, terrain, and available resources, to name just a few.

Chapter 34, Litter and Carries, addresses the numerous considerations that exist when a patient will be packaged and transported from an isolated wilderness area to definitive care.

Rescue Equipment

Today's rescues occur in many remote and unusual environments and often require extremely technical rescue equipment and skills. Responders trained in the appropriate techniques and technologies should be the only personnel to apply them. Much of the gear and many of the techniques have been derived from those first developed by mountaineers, climbers, cavers, and, more recently, white-water enthusiasts.

Rescue equipment is generally broken down into three broad categories: personal gear, rescue software, and rescue hardware. Personal equipment includes such items as footwear, gloves, helmets, articles of clothing, eye protection, and other protective apparel. Software is equipment such as rope, webbing, slings, and harnesses that are made of soft, strong synthetic materials specifically designed and manufactured for rescue. Hardware includes equipment such as carabiners, cams, friction devices, pulleys, and litters made of steel and alloys specifically designed and manufactured to endure the rigors of rescue.

Figure 32-5. Even though the personal equipment necessary is dictated by both the rescue environment and the needs of the specific situation, it includes head, hand, foot, and eye protection as a minimum.

Figure 32-6. Rescuer wearing a personal flotation device (PFD) and helmet often used for rescue in and around moving water.

Personal Equipment. Rescuers must often wear special equipment to protect them from accidents and hazards. Head, eye, and hand protection is considered mandatory in virtually all rescue environments. Additional personal equipment requirements are dictated by the rescue environment and the specific needs of the situation (Fig. 32-5).

Special Gear. In addition to the usual challenges of the rescue environment, certain hazards require specialized equipment. Examples of such equipment include fire-resistant clothing worn by structural and wildland firefighters, personal flotation devices (PFDs) used by rescuers in and around water (Fig. 32-6), netting used in outdoor settings when insects become a problem, bulletproof garments used by law-enforcement and military rescue personnel, and chemical protective suits worn when exposure to hazardous materials is possible.

Figure 32-7. Example of $^{1}/_{2}$-inch, nylon, static, kernmantle rope of the type commonly used in rescue. Rescue rope should be checked over its entire length for damage before use.

No clothing or protective gear meets all of the requirements for involvement in or around a rescue scene. Rescuers study situations so that they understand all hazards before anyone becomes involved. Their conclusions help them identify protective equipment requirements. Gear that may be necessary for one environment can be dangerous in another. A firefighter's turnout gear may be required in a structure fire but can be deadly in a river rescue situation. Every rescuer is responsible for understanding the rescue environment and how to best prepare for it.

Software

Rope. Rope is by far the most versatile piece of rescue equipment and serves as the universal link in most rescue environments. The material from which the rope is made (e.g., nylon, polyester, or polyolefin) and the design (laid, kernmantle, flat) are important in the consideration of the use for which a rope is intended.

In most rescue environments, nylon is preferred because of its overall strength, resistance to abrasion, and ability to stretch and absorb energy. Natural fiber ropes such as hemp are no longer considered for use in rescue; synthetic materials are far better. Although design and amount of materials used influence strength, new $^{1}/_{2}$-inch diameter nylon rescue rope usually has a tensile strength in excess of 9000 pounds (40 kN) (Fig. 32-7).

Figure 32-8. **Left,** Tubular webbing with cross section visible. **Right,** Flat webbing used in anchor sling.

The most common design of rescue rope is *kernmantle*, a term derived from German, meaning "core in sheath." With this design, a core of material (often parallel fibers) is surrounded by a braided sheath. The sheath protects the inner core, which supplies much of the strength of the overall rope. Other designs, such as laid (twisted) and braided, are also used in rescue rope.

Kernmantle rope is either dynamic or static. Dynamic kernmantle stretches more than 4% of its length to absorb the impact of a fall, and it is used primarily in lead climbing. Static kernmantle stretches less (no more than 4% of its length); it is used in rescues in which a great deal of stretch would be a nuisance or even dangerous.

Because of the importance of rope in the rescue chain, frequent inspection, care, and maintenance are important. Rope used in rescue is kept clean, inspected often, and protected from sharp edges, high temperatures, sunlight, chemicals, and abrasion. In addition, a detailed history of rescue ropes is kept so that an educated decision can eventually be made regarding each rope's removal from rescue service (see Chapter 84).

Webbing. Flat rope or webbing is another common link in rescue systems. It comes in two common configurations: flat and tubular. Tubular webbing is manufactured as a tube in such a way as to seem flat when in use. In cross section, however, it is obviously tubular and a bit less stiff than true flat webbing. One-inch diameter tubular webbing can be used in rescues to tie anchor slings and harnesses. It has a tensile strength of about 4000 pounds (17.8 kN) when new.

Flat webbing is flat in cross section. Its strength is directly proportional to the amount of material used in its manufacture. Automobile seat belts are an example of the material used in rescue harnesses and anchor slings, and where strong, flat software is beneficial (Fig. 32-8).

Harnesses. Harnesses come in many sizes and shapes; they are used to attach something (usually a rope) to a person's body. They may be "full body," encompassing the thorax and the pelvis (Fig. 32-9); "seat," encompassing only the pelvis (see Fig. 32-5); or "chest," encompassing only the thorax. Each type of harness has its use and associated advantages and disadvantages. Classically, the most common harness for climbing has been the seat harness. However, rescue practitioners have been trying to standardize the full-body harness for rescuer use, with the separate seat and chest harnesses having only limited special use by trained individuals.

Figure 32-9. An example of a full-body harness. Shown is a CMC ProSeries Harness Combo. Note that this harness encompasses both the pelvis and the thorax. (Photo © CMC Rescue.)

Webbing can be tied into a large loop (runner) and applied to a person in such a way as to serve as an improvised harness. Although this is not a preferred method of attachment to a rope, it can work if other harnesses are not available.

Hardware

Carabiners. Carabiners are large, safety pin–type mechanisms used to connect various elements of a rescue system, such as a rope and anchor. They are occasionally called "biners," "snap links," or "crabs" and consist of a spring-loaded gate that pivots open, a spine that supports most of the load and lies opposite the gate, a latch, and depending on the specific style, a locking mechanism.

Carabiners are most commonly made from either steel or aluminum. Size for size, steel is stronger and heavier, but aluminum is lighter and stronger pound for pound. In rescue, steel is almost always preferred unless weight is a factor, as it is in remote alpine situations.

Common shapes of carabiners include oval, D, offset D, pear, and large offset D. The design best suited for any situation is dictated by the specific use. No matter what the shape, carabiners used in rescue usually have a mechanism for locking the gate closed so that opening it takes a specific effort. This design feature not only improves the strength of the device but also reduces the chances that a carabiner will open accidentally at an unexpected time (Figs. 32-10 and 32-11).

Figure 32-10. Various types of carabiners. Top to bottom: (1) RSI Big hook, steel, screw locking hinged gate; (2) alloy, offset D, screw locking hinged gate; (3) RSI Twist Link, steel, screw locking hinged gate; (4) SMC extra large, rescue, steel, screw locking hinged gate; (5) Tri-link, steel, triangular, screw lock; (6) Auto-lock, swivel-mount, steel, quarter twist locking hinged gate (NFPA 1983 certified). Note that locking carabiners should always be locked when in use.

Figure 32-11. Close-up view of CMC Rescue aluminum carabiner. Note the locking gate and offset *D* shape. (Photo © CMC Rescue.)

Descending (Friction) Devices. Many different descending devices exist today, but they all do primarily the same thing: apply friction to the rope to allow controlled lowering of a person or load. The most common descending devices in rescue are the figure-8 plate and the brake bar rack.

The figure-8 plate gets the name from its general shape. It has two rings of different sizes. The larger ring produces friction on the rope, whereas the smaller ring is used primarily as an attachment for the load (e.g., the rescuer during rappel). Friction is produced by passing a bight of rope through the large ring and around the small ring, then attaching the small ring to either an anchor (for a lowering system) or a rescuer's harness (for a rappel or abseil) with a locking carabiner (Fig. 32-12).

The brake bar rack, or simply "the rack," uses either steel or aluminum bars on a steel rack to produce friction on a rope. When the rope is threaded alternately around the bars and the load or rescuer is attached to the "eye" in the rack, friction is applied. The number of bars applied to the rope and the distance between them can be varied to change the friction. This variable friction allows versatility not available with the figure-8 plate; however, the rack takes a bit more training to use safely (Figs. 32-13 and 32-14).

Ascenders. Ascenders are devices that grip or hold the rope. They have been adapted from climbing and caving equipment, with which they are used to ascend or climb a fixed rope. In rescue, they are used to climb fixed lines when necessary, but they can also be used in hauling systems to grip the rope. In this way, they hold fast when the rope is pulled in one direction and allow the rope to slide easily when it is pulled in the other direction (Figs. 32-15A–D and 32-16).

When ascenders are used to climb a rope, one is fixed to the rope and supports the load while the other is moved into position ahead. When this action is alternately repeated, a skilled climber can move up a rope with relative speed and ease. Selected rope hitches (e.g., a Prusik hitch) can be used in lieu of an ascender (see Fig. 32-15D).

Pulleys. Pulleys are simple machines that apply a turning wheel to reduce friction on a rope as it rounds a turn. In rescue, these metal devices serve primarily to change the direction of a rope, such as within a mechanical advantage system. The "sheave" is the wheel or pulley, and there may be more than one. The "side plate" or "cheek" is the side of the device that makes contact with the anchor at the "hook," which is usually the weakest part. The axle or "sheave pin" is what the wheel turns on; it is supported by the side plates. In rescue pulleys, the side plates are movable so that the pulley can be attached to a rope anywhere along its length (Fig. 32-17).

The larger the diameter of the pulley, the more efficient the device. That is, the bigger the pulley, the more friction (theoretically) is reduced. A rule of thumb often used by rescuers is

Figure 32-12. Example of figure-8 plate descending device (with "ears") commonly used in rescue. Shown is a CMC Rescue 8 with Ears; aluminum *(left)* and steel *(right)*. (Photo © CMC Rescue.)

Figure 32-14. SMC NFPA Brake Bar Rack. Straight frame shown; frame also available with eye twisted 90 degrees. The top bar has been modified to allow for a secure tie-off. (Photo © CMC Rescue.)

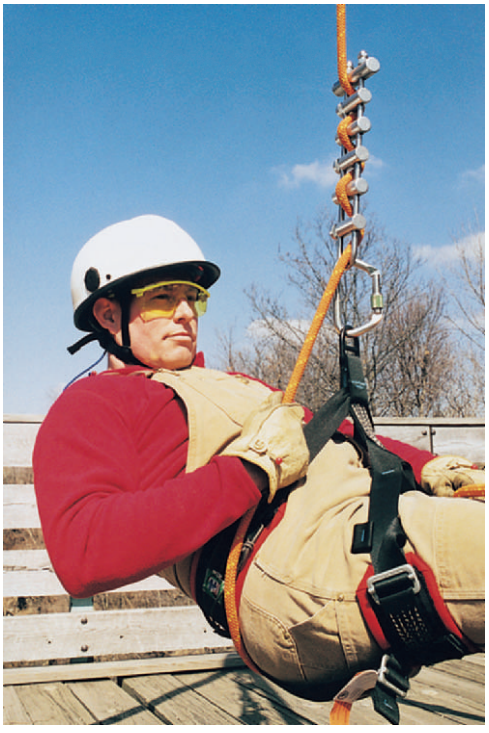


Figure 32-13. RSI Super Rack brake bar rack in use.

that a pulley with the largest diameter possible should be used, but never less than four times the diameter of the rope. Therefore, because $^1/_2$-inch (11 mm) rope is commonly used in rescue, a pulley diameter of at least 2 inches should be used.

A variation of the pulley is the edge roller. This device uses 4- to 6-inch open-face pulleys to reduce both the friction of a rope passing over an edge and any damage to the rope by protecting it from excess abrasion. Single units can protect the rope from 90-degree angles, and multiple units tied together can provide protection for complex projections.

Litters. Litters or stretchers are the conveyances in which victims are transported when they cannot travel under their own power. New high-technology materials and designs have greatly improved the choices available. In past years, rescuers were forced to choose between wooden backboards, old military stretchers, the wire Navy Stokes basket, or the "scoop" stretcher. Today, strong, lightweight synthetic materials and inventive designs have improved the strength, weight, durability, and comfort of litters.

The goals have not changed during evolution of the perfect wilderness transportation device. Rescuers still want a device that is comfortable for a person in pain, serves well as a platform for assessment and medical care during transport, allows for full-body immobilization, and provides complete security and protection of the occupant from the rescue environment. See Chapter 34 for additional information regarding specific litters, packaging, handling, and evacuation techniques.

Figure 32-15. **A,** Gibbs ascender applied to rope. When the eye of the cam is pulled, the cam squeezes the rope and holds fast. When the cam is released, the device can be moved on the rope. **B,** Gibbs ascender dismantled with shell around rope. Note cam *(upper left)* and pin *(bottom)*. **C,** Clog handled ascender. Although used where climbing a fixed rope is required, handled ascenders are rarely used in rescue. **D,** A 3-wrap Prusik hitch can often be used in lieu of a mechanical ascender.

ANATOMY OF A SEARCH AND RESCUE INCIDENT

To summarize how all of the previously discussed information fits together, it is convenient to dissect a SAR incident into its component parts and then analyze how all of the parts fit together (Fig. 32-18).

From the SAR operative's perspective, an actual callout is merely an interruption of planning for an incident. That is, people involved in SAR are constantly in a state of readiness and are prepared to respond. When a situation occurs, this constant planning stage is suddenly interrupted by the report of an incident. The individual taking the information is charged with conveying it to the appropriate authority. The authority determines the urgency, continues the investigation process, begins to develop an operational strategy, and generates an incident action plan. At the same time, those in charge begin to muster appropriate resources to carry out the action plan. In SAR, this is termed *resource callout,* or just callout.

Once notified of an incident, individual resources are gathered at a collection point and signed in. The sign-in process enhances safety and allows tracking of the types and quantity of resources that are available on-scene. Once signed in, resources are given assignments designed to meet the goals of the action plan within a specific operational time frame. This physical implementation of the incident action plan is referred to as *tactics*.

Figure 32-16. Alternate type of cam ascender specifically designed for rescue. Shown is a Petzl Rescucender for use with $^{7}/_{16}$ or $^{1}/_{2}$-inch rope. (Photo © CMC Rescue.)

Figure 32-17. Two types of pulleys commonly used for rescue. **Top,** 2-inch double pulley. **Bottom,** 2-inch,"Prusik-minding." Note that rescue pulleys are applied to rope by removing the carabiner and swiveling the side plate to allow the introduction of the rope.

Deployment of resources to the field continues until there is reason to suspend this phase of the operation. If the subject is found, the search is suspended, and the access phase can commence. Once rescuers have access to the subject, the focus turns to stabilization and transportation. If at any point the operation cannot be continued (e.g., the subject was never found, access cannot be gained, transportation is impossible), suspension and demobilization may occur without completion of the entire cycle. The decision to discontinue active search efforts is difficult and involves complex management issues, almost always of the "no-win" variety.

When a situation is resolved, mission suspension and demobilization begin. In larger incidents, this may involve structured deactivation of multiple resources, pulling teams out of the field, dismantling facilities, completing documentation, and returning resources to service. Basically, everyone finishes what he or she was doing at this incident and gets ready to do it again. All of this takes planning and preparation and should be addressed in the overall preplan long before it is required.

After every incident, participants realize that if they had it all to do again, they would do some things differently. If these thoughts and ideas are not documented, they can be lost, and future responses may be cursed to repeat past mistakes. This is one reason that every incident should contain some type of evaluation of the entire mission, known as the *postincident critique*. The critique can be formal, involving every participant at a sit-down meeting, or informal, involving just a brief discussion of recent events. The critique documents lessons learned and

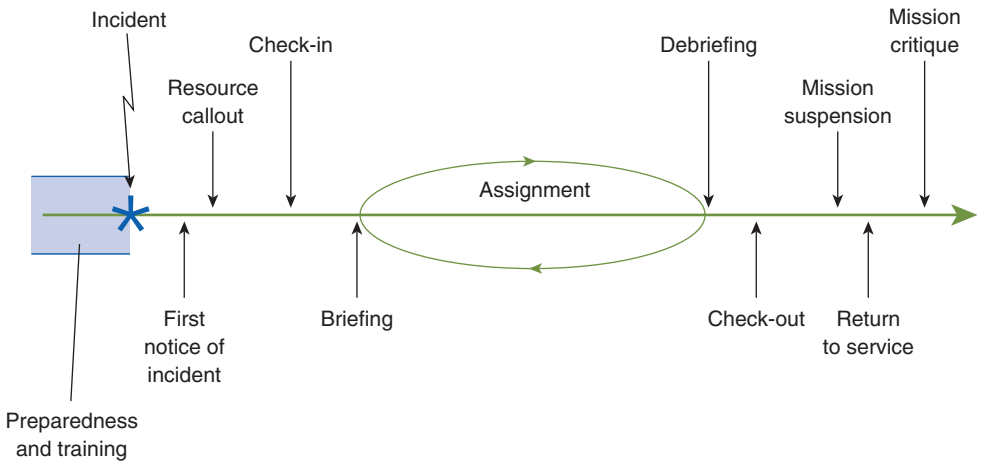


Figure 32-18. Time progression or the "anatomy" of a search from a searcher's (operational) perspective. The process is actually a continuous cycle that pauses in the planning and preparation phase until an incident occurs.

should provide a basis for revising the preplan. Thus the cycle continues, and lessons learned from one mission influence the next.

SEARCH AND RESCUE ENVIRONMENTS WITHIN THE WILDERNESS SETTING

SAR teams throughout the world are frequently called on to solve complex problems in a wide spectrum of environments. Even within the environments addressed in this text, widely diversified subenvironments exist that present unique sets of problems and hazards to SAR personnel. When confronted with the numerous and dangerous environmental conditions found in the wilderness setting, SAR personnel must be prepared to work where others have been unable to cope. An old military motto becomes the SAR credo: "Adapt, improvise, overcome."

It is beyond the scope of this text to discuss in detail how SAR personnel adapt to each of these environments, but it is important to note that adaptation and improvisation are required in nearly all wilderness situations. The particular improvisation depends on the situation, as well as the skill and experience of the individuals involved.

Regardless of the type of rescue environment encountered by rescuers, the following general rules should be followed:

1. Use technical personnel for technical rescue.
2. If a missing person is found dead, evacuate only when there is no risk to team members, or at least when the hazard has been assessed and the risk justified.
3. Stabilize any injured person before evacuating and continue stabilization during transport.
4. Find and use the easiest route for evacuation.
5. If a carry-out is required, appoint someone to serve as route finder who has a radio and markers to report potential hazards and problems.
6. Litter teams of six to eight persons per team should be used, with three teams minimum. Normally, there should be no more than 20 minutes per shift. Additional personnel may also be required to carry equipment.
7. Use accepted procedures to care for and protect the victim.
8. A radio carrier brings up the rear.
9. If using a helicopter for evacuation, make sure:
   a. That the patient is thoroughly briefed.
   b. That the patient is protected.
   c. That someone goes with the patient who knows what has been done medically. In some cases, because of agency or military service protocols, assisting medical personnel will not be allowed to go with the patient. In these cases, it is imperative that a written log specifying assessment and treatment up to that point accompany the patient.

Special Environments in Search and Rescue

Specialized SAR environments produce diverse problems and potential complications. Each environment presents its own obstacles to increase the complexity and difficulty of particular rescues.

Technical Rock

Mountaineering, rock climbing, and casual scrambling have created a need for specialized SAR expertise. Individuals and groups involved in rock rescue have refined and developed techniques for most situations. The hallmark of a technical rock rescuer is the ability to improvise and modify tools or techniques to meet any crisis. He or she must be comfortable using climbing gear and being exposed to heights.

Once an individual is located in a rock environment and the situation is surveyed, access to the site is the next step. Often, local groups familiar with well-known areas will have already solved this problem. The solution will involve either climbing up or dropping down to the victim. Because any accident during a rescue is almost always catastrophic, safety for all persons involved is of paramount importance. Ascent up rock always requires knowledge of rock-climbing techniques, proper equipment, and familiarity with its designed use and limitations. Local outing clubs or mountaineering stores can be contacted for more detailed assistance. Specialized technical rock rescue

teams, such as those sanctioned by the Mountain Rescue Association, routinely practice climbing techniques and solving vertical rescue problems.

Caves and Mines

Standard obstacles in this environment include poor communications, extreme darkness, difficulty in lighting, small and wet spaces, and often the issue of questionable atmosphere. The various environments included here are collectively termed *confined spaces*.

The levels of moisture in water-containing, or "live," caves can vary over a considerable range. Some are merely muddy; others have flowing rivers. Caves in the western United States are generally drier than eastern caves; however, humidity, wetness, and cold temperatures create potential for hypothermia in both areas. This is a fact that is greatly underestimated. Flooding can be a lethal problem, and many cavers have died because of inattention to the weather on the outside. During heavy rains, the caves become natural drains for streams. Wind and temperature are other underestimated problems associated with cave and mine emergencies. It is not unusual for strong winds to develop along subterranean passages, which intensifies convective air chilling.

Confined passages, low crawls, and squeezes pose unique problems for the rescue of injured cavers. The use of standard items, such as litters, backboards, and splints, may be impossible in such places. Confined passages with varying, or even toxic, constituent gases can lead to difficulties for victims and rescuers alike. Occasionally, a self-contained breathing apparatus or surface-supplied air is required. The potential for toxic gases justifies extensive atmospheric monitoring while operating in the underground environment.

An essential part of any cave or mine rescue operation is thorough orientation to the hazards associated with a particular underground area. This involves pinpointing the locations of pits, waterfalls, siphons, canyons, and other difficult formations that may pose problems in extrication, search, or safety. Many caves have been mapped by the National Speleological Society and the National Park Service.

The real difficulties may begin only after a victim is located. The goal is to move the person rapidly, safely, and comfortably to the surface. Without practice underground, that task will be virtually impossible. Neoprene exposure bags similar to body bags have been used for this purpose and can keep an injured person dry and protected during what may be a very long and slow evacuation.

Medical care procedures must be performed under dark, cold, and muddy conditions. Experienced cave rescuers agree that repackaging supplies and equipment for underground use is essential. Streamlining kits, packs, and containers is imperative for unobstructed passage through tight spaces in cold, damp conditions.

Team members must carry very specialized equipment, often including a minimum of 24 hours of light in a helmet-mounted lamp; two additional sources of light, with spare bulbs and batteries; and waterproof matches and candles. Depending on the situation, other equipment is also required and is discussed in Chapter 40, Caving and Cave Rescue.

Essential caving skills include all of the capabilities for rock climbing, including vertical rope technique, ascending, rappelling, belaying, and being comfortable working at the end of a rope. All these skills must be practiced until they can be done in the cold and wet without the benefit of light. Team practices are conducted both on the surface and underground, with participants being forced to work in mud, suffocatingly tight squeezes, soaking waterfalls, and complete darkness. This may be a difficult evolution for even the most experienced rescuer to endure, but just another "hang in the hole" for a seasoned caver.

Figure 32-19. Common personal river rescue equipment. In the CMC Water Rescue Package shown (*clockwise from upper left*) is a gear bag, type III/IV personal flotation device, emergency strobe light, glow-in-the-dark Fox 40 whistle, McNett Satur knife, NRS neoprene paddler's gloves, Cascade Swiftwater helmet, and a CMC ProSeries EZ-Stuf throwline bag. (Photo © CMC Rescue.)

White-Water River

There are dozens of potentially dangerous problems in the river SAR (white-water) situation (see Chapter 39). Log and debris piles at various bends in the river can function as "strainers" for the recreational victim, but they may be death traps for the would-be rescuer. The banks of the stream may be deeply undercut, with treacherous overhanging debris and snags that can catch on clothing, equipment, and skin. Combined with muddy and rapidly rising water, these factors render river rescue difficult and very unpredictable.

In fast-moving water, the single greatest problem is that responders underestimate the power and threat of moving water. Foolhardy heroics and excessive enthusiasm frequently lead to further tragedy. Cold-water immersion, coupled with wind and cold temperatures, predisposes everyone to hypothermia. Wet clothing, darkness, and injury add to the insult. The noise of moving water may obviate clear communications, and result in poor contact between the victim and rescuers. These same factors could also affect communications between rescuers and lead to additional confusion and danger.

All potential responders in this environment must be properly equipped (Fig. 32-19) and know how to read the water for capsize points and other dangerous phenomena. The hydraulics of low-head dams, collapsed bridges, and other submerged structures can produce a drowning machine for unsuspecting individuals. Rescue team members must know how to protect themselves in fast-moving water at all times. Mandatory in this environment are good judgment, strong swimming ability, knowledge of all types of technical systems, equipment used in

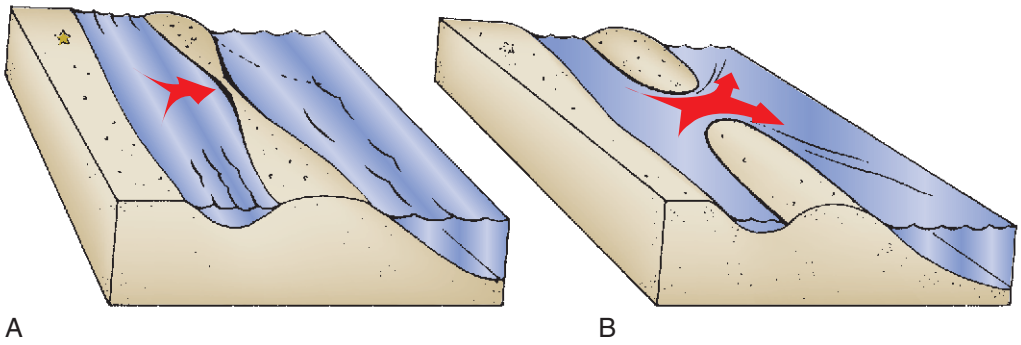


Figure 32-20. Runout. **A,** This phenomenon begins with an offshore sandbar. As waves roll in, the water level builds up behind the bar until a section gives way. **B,** As the sandbar "dam" gives way, the water develops a very rapid current running seaward. The recommended action is to swim across the current until out of the pull.

climbing, and a thorough understanding of river dynamics and hydraulic influences.

White-Water Surf

Like river white-water, ocean surf can present some very different problems in rescue because there is no "average" beach. There are recurring rescue situations that pose unique problems in the white-water surf environment.

Along with the potential for immersion hypothermia, lacerations and contusions can result from being dashed against barnacle-encrusted rocks in the wild and unpredictable ocean surf. Contact with venomous sea life is always a possibility. However, the greatest threat to ocean beach users is the action of the water itself and the possibility of drowning through inattention or unfamiliarity with ocean surf hazards in the form of runouts, undertows, and rips.

Runout. A runout occurs when an offshore sandbar or ledge is built up over a long period. Millions of tons of water flow over the bar during daily tidal changes. Eventually, the water may equal or exceed the level outside the bar. Any weak spot in the bar usually gives way, causing a funnel effect (Fig. 32-20). Water rushes toward the bar at a terrific rate, sweeping everything with it. This common phenomenon can be easily spotted from the beach. Usually 15 to 50 yards wide (14 to 46 m), it is characterized by choppy, jumbled-up, little waves. The water often has a dirty, foamy, or debris-laden surface moving seaward. If a bar is visible offshore, definite breaks can be seen where the water pours through. Surfers often seek runout currents for fast transportation out beyond shoreline waves.

Swimmers caught in a runout have two options. They may swim parallel to the shoreline out of the strip of current, or if the bar is visible (usually characterized by breaking waves), they may relax and let the current complete its runout. About 25 yards (23 m) beyond the bar, the current dissipates. This is an offshore phenomenon—current force increases near the bar but is often negligible near shore.

Rip. A far worse problem close to the beach is a rip, which can knock children and even adults off their feet and carry them to deep water in seconds. Rips are caused by a slight depression on the beach where wave water rushes after breaking on shore.

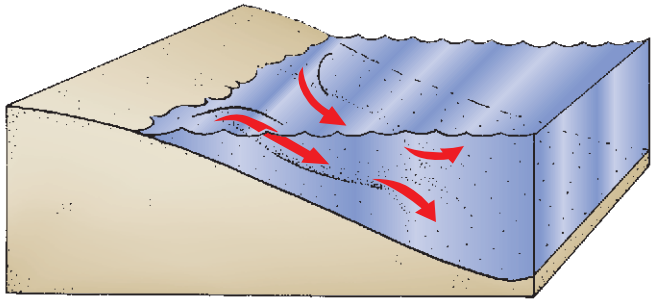


Figure 32-21. Figure rip. A depression in the beach floor concentrates returning water into a strong current. To escape, a person should ride with the current or swim to the side and out of the pull.

Water rushing to the depression soon becomes an irresistible seaward flow (Fig. 32-21). It may be as narrow as 15 yards (14 m) at its source and usually does not travel as far as a runout. Rips generally dissipate a few yards beyond the breakers. A rip looks like a runout, with a streak of turbulent discolored water or a line of foam leading directly out from shore. A swimmer has the same options as in a runout, either to swim parallel to the beach or to relax and ride the current until it ebbs. A person who swims straight toward the beach will never make it. A beach with several rips moving up and down in unpredictable patterns is very dangerous. An unwary swimmer could panic and drown.

Undertow. On narrow, steep beaches, a type of current known as undertow can be found. It is caused by gravity acting on water thrown up on the beach by wave action. Water retreating back down the steep shore continues under oncoming waves (Fig. 32-22). Undertow is usually of very short duration and is ended by the next breaking wave. Wading near shore on a steep beach, an individual could be pulled under in this current and find himself or herself quickly in deep water. If the person resists the current, the next wave may break directly on the person's back. In some circumstances, this could cause traumatic injury, especially to the neck and back. A person caught in an undertow should let the current pull until it ceases, then swim to the surface and ride the next wave into shore.

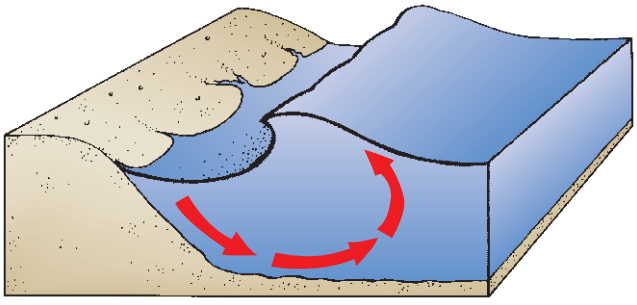


Figure 32-22. Undertow. This hazard develops on a steep beach where the water returns rapidly seaward after being tossed up by the wave action. A person should never fight this action but should relax and rise on the next wave.

Box 32-2. COSPAS-SARSAT Abbreviations

COSPAS—*cosmicheskaya sistyema poiska avariynich sudov* (Russian; in English, "space system for the search of vessels in distress")
ELT—emergency locator transmitter
EPIRB—emergency position indicating radio beacon
PLB—personal locator beacon.
SARSAT—search and rescue satellite-aided tracking

From the National Oceanic and Atmospheric Administration: National Environmental Satellite, Data and Information Service, Search and Rescue Satellite-Aided Tracking (SARSAT) website. Available at: http://www.sarsat.noaa.gov.

Cold, Snow, and Ice

Perhaps no other type of SAR environment requires a more broadly based foundation of personal and team skills than winter snow and ice. These skills include downhill and cross-country skiing, snowshoeing, technical climbing, winter survival, and a good understanding of snow and ice physics. Unlike rock, snow and ice conditions change on a daily and even minute-to-minute basis. The effects of gravity, wind, temperature, slope, heat exchange, load factors, and avalanche continually impose problems for missions under these conditions. Technical and nontechnical SAR problems in snow and ice environments take longer to address and are more taxing, technical, and complex. Combined with shorter days, extremes of weather, and the ever-present threat of hypothermia and localized cold injuries, technical missions of this type are unacceptable for all but the most experienced SAR personnel. Several chapters in this book address very important issues related to this type of environment: Chapter 5 (Accidental Hypothermia), Chapter 6 (Immersion in Cold Water), and Chapter 9 (Polar Medicine).

Versatility and improvisation are essential components of the overall strategy that must be used in snow and ice. Transportation of the victim is often one of the most difficult problems, but it can usually be resolved through detailed preplanning. Innovations such as covering a litter with a canvas cover or improvising an attachment to cross-country skis are clever solutions to common winter problems. Commercial products such as the Hegg sled and the SKED litter (see Chapter 34) have streamlined the laborious task of transporting injured people through snow and ice.

The references for this chapter can be found on the accompanying DVD-ROM.

Technical Rescue in the Wilderness Environment

33

Loui H. McCurley and Steve E. Hudson

Redcloud Peak is the 46th-highest of Colorado's 52 "14ers" (i.e., peaks whose height is at least 14,000 ft [4267 m]). Located in the beautiful San Juan Range, the peak gets its name from an intense red coloring, making the mountain visually distinctive. A saddle connects Redcloud with its nearest neighbor, Sunshine Peak, making it possible to achieve this second summit with an hour's hike from the top of Redcloud.

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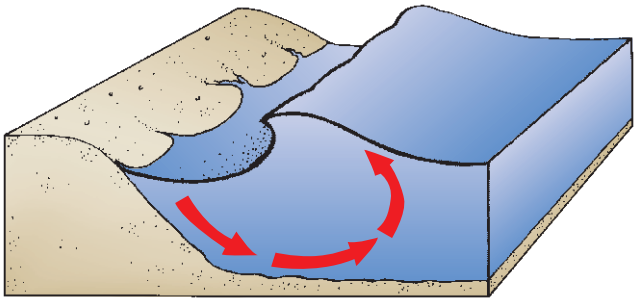

Figure 32-22. Undertow. This hazard develops on a steep beach where the water returns rapidly seaward after being tossed up by the wave action. A person should never fight this action but should relax and rise on the next wave.

Box 32-2. COSPAS-SARSAT Abbreviations

COSPAS—*cosmicheskaya sistyema poiska avariynich sudov* (Russian; in English, "space system for the search of vessels in distress")
ELT—emergency locator transmitter
EPIRB—emergency position indicating radio beacon
PLB—personal locator beacon.
SARSAT—search and rescue satellite-aided tracking

From the National Oceanic and Atmospheric Administration: National Environmental Satellite, Data and Information Service, Search and Rescue Satellite-Aided Tracking (SARSAT) website. Available at: http://www.sarsat.noaa.gov.

Cold, Snow, and Ice

Perhaps no other type of SAR environment requires a more broadly based foundation of personal and team skills than winter snow and ice. These skills include downhill and cross-country skiing, snowshoeing, technical climbing, winter survival, and a good understanding of snow and ice physics. Unlike rock, snow and ice conditions change on a daily and even minute-to-minute basis. The effects of gravity, wind, temperature, slope, heat exchange, load factors, and avalanche continually impose problems for missions under these conditions. Technical and nontechnical SAR problems in snow and ice environments take longer to address and are more taxing, technical, and complex. Combined with shorter days, extremes of weather, and the ever-present threat of hypothermia and localized cold injuries, technical missions of this type are unacceptable for all but the most experienced SAR personnel. Several chapters in this book address very important issues related to this type of environment: Chapter 5 (Accidental Hypothermia), Chapter 6 (Immersion in Cold Water), and Chapter 9 (Polar Medicine).

Versatility and improvisation are essential components of the overall strategy that must be used in snow and ice. Transportation of the victim is often one of the most difficult problems, but it can usually be resolved through detailed preplanning. Innovations such as covering a litter with a canvas cover or improvising an attachment to cross-country skis are clever solutions to common winter problems. Commercial products such as the Hegg sled and the SKED litter (see Chapter 34) have streamlined the laborious task of transporting injured people through snow and ice.

The references for this chapter can be found on the accompanying DVD-ROM.

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visitor to the area, waved goodbye to his companion at the Silver Creek trailhead at 9:30 AM one July morning and set off to do just that.

The Silver Creek trail taken by Daniel travels into a basin northeast of Redcloud Peak. From here, the trail leads high into the basin and onto the peak's northeast ridge. Traversing this ridge a half mile or so, the climber reaches the summit of Redcloud, and it is just another hour's hike to the summit of Sunshine. The best egress from Sunshine is to retrace one's steps back up Redcloud, down the northeast ridge into the basin, and back down Silver Creek Trail. Off-trail, Redcloud and Sunshine are rugged, marked by numerous steep to vertical drainages, steep scree fields, and talus the size of small houses.

At 8:15 PM on the day of Daniel's summit hike, J. Hunter Holloway, State Mission Coordinator for the Colorado Search and Rescue Board, received a call from the Lake County Sheriff's office announcing that Daniel had been reported missing. Hunter mobilized several teams from around the state and commenced a search at daybreak with 70 well-qualified responders, several dogs, and an army Chinook helicopter.

Seldom is wilderness rescue a nice walk in the woods followed by evacuation off a convenient cliff with a helicopter ride to the hospital, and the search involved to locate Daniel typifies the worst type of wilderness technical rescue for the medical professional. Daniel had undertaken a difficult route at breathtaking altitude, and when stressed, he behaved in an unpredictable way, further increasing search difficulty. Rescuers spent hundreds of hours combing the trails and surrounding terrain day and night. The physical demands of hiking miles of trail, then venturing off trail to negotiate scree fields, scramble over and search under huge talus, and rappel hundreds of feet of steep-to-vertical gullies are challenging even for the most fit of rescuer-athletes and require extraordinary levels of physical and mental agility.

When Daniel's body was eventually located, it was in a dangerous and compromising location that would require special rigging skills to evacuate. Finding Daniel alive likely would have imbued rescuers with the physical and psychological reserves to effect the evacuation. Instead, the exhausted team had to dig deep into already depleted reserves to remain attentive and energized to fulfill a discouraging operation. The search for Daniel was not unique. Lest the reader think wilderness rescue is a glorious task, let it be known that it frequently involves arduous hikes and climbs followed by less-than-ideal outcomes.

Operations such as this, involving search of technically demanding terrain and the potential for a rope rescue evacuation, require personnel with special training and preparation. A medical professional may be exposed to a wilderness incident intentionally, while working in the capacity of a responder, or spontaneously, when witnessing an incident while part of an expedition or recreational outing. Preparing for these possibilities in advance, and resolving to keep the response consistent with the level of training, is the first step toward a successful outcome.

Technical evacuations in the wilderness requiring ropes and special techniques range from steep slope to vertical, and even overhanging. Climbing skills do not prepare a person for the rigors of performing a technical evacuation of an injured person from rugged terrain. Experience with ropes and rigging in the urban environment is a useful first step, but preparation for wilderness rescue of the technical kind requires training and rescue in a genuine wilderness environment.

Figure 33-1. High-angle wilderness rescue. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

Wilderness settings are usually relatively inaccessible or not serviced by maintained roads, and they encompass a wide range of terrain and environmental conditions. High-angle rescuers find themselves working in hot, dry, desert conditions, or in snowy, thin-air, or alpine environments, and in anything between (Fig. 33-1). It is a fortunate rescuer who finds himself faced with a nice, clean cliff rather than steep and unstable rock, ice, thin gullies, or loose scree.

This chapter addresses broad concepts of wilderness search and rescue; responders should evaluate their particular circumstances and seek specific training for the types of incidents that they may encounter.

Not all rescuers need to be trained to the most advanced levels of wilderness operations, and individuals should always operate within the limits of their experience and training. Before attempting to respond to a high-angle rescue incident, responsible persons should ensure that every field team has a breadth and depth of experience to enable personnel to operate safely and make sound decisions.

High-angle rescue operations in the wilderness (Fig. 33-2) are often complicated by circumstances unique to the specific environment. Examples include difficult terrain, hoisting (raising) operations, significant obstacle negotiation, navigational difficulties, descending or ascending operations from

Figure 33-2. A haul team uses a mechanical advantage system during a high-angle rescue. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

extreme heights, and severe environmental conditions (i.e., snow, rain, altitude).

In the United States, legal responsibility for wilderness rescue varies by state. In many of the eastern states, search-and-rescue (SAR) duties fall under the state's Emergency Management Agency (EMA), which covers several services often grouped under the general umbrella of fire emergencies: firefighting, rescue, and emergency medical services (EMS). In most of the western states, law enforcement authorities are charged with SAR. On the oceans and for military personnel, U.S. Coast Guard and other military organizations handle SAR duties. In National Parks, rescues are handled by a mix of staff personnel and volunteer rescuers.

Because of unique demands in terms of equipment, training, and techniques, organizations responsible for high-angle wilderness rescue typically have relationships with teams of specially trained volunteers or paid professionals.

Rescue squads that perform technical rescue in the wilderness receive training from more experienced members of their own local teams, paid trainers from outside companies, and participation in national conferences and events. Paid trainers may provide a certificate to the individuals they train, based either on their own criteria or on nationally recognized criteria such as found in the National Fire Protection Association's (NFPA's) code, NFPA 1006.[4] On a more formal level, the National Association for Search and Rescue has developed and oversees a system of training individuals in basic SAR, and the National Cave Rescue Commission maintains a system of training individuals at various levels in cave rescue techniques. Both organizations offer certificates in their respective courses, and all are aimed at the individual.

Certification/accreditation at a team level is less common. Some paid trainers provide certificates of conformity to teams that they train, based on national team performance criteria, such as found in NFPA 1670.[5] For teams that actively engage in a variety of backcountry rescue operations, the Mountain Rescue Association (MRA) offers team accreditation. However, this accreditation requires teams to meet strict performance requirements and to demonstrate their skills in a rigorous peer evaluation in rugged terrain search, technical rope rescue, and alpine (snow-and-ice and high-altitude) rescue. It is relatively

Figure 33-3. A high-angle rescue technician. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

impractical for teams that perform technical rescue in only one or two of these areas to seek accreditation, so MRA-accredited teams tend to be limited to areas where the full mix of rescue types are found.

Persons who engage in wilderness technical rescue (Fig. 33-3) should be skilled in rope rescue techniques for mountain environments and should combine those skills with (1) an ability to operate effectively in weather extremes, (2) excellent navigational skills, (3) night operations skills, and (4) self-sufficiency for extended insertions. Medical skills and certifications are also useful.

EXPEDITIONS AND SPECIAL EVENTS

Medical and SAR personnel are often called on to assist with medical incidents and rescue needs for expeditions and at adventure-type races that involve rock climbing, rappelling, ascending, and other challenges with high exposure to adverse conditions. Sometimes, adventure race and outdoor experience events occur when there has been little or no preplanning for emergencies, relying completely on available resources, volunteer or paid, to respond in the event of an emergency. Other times, planners contract in advance with local resources, or hire individuals from local rescue agencies.

Committing to high-angle rescue responsibility for a special event is a significant undertaking in our litigious society, so the decision should not be taken lightly by an individual or an organization. Often, event managers simply want to shift responsibility and are not willing to provide the support, funding, and resources necessary to effectively develop a complete risk management program. Many such events do not

even have personnel dedicated to safety and rescue. Even Eco-Challenge, a well-known adventure race, did not hire its first dedicated safety director until several years into its existence and until that time did not have a written safety plan.

One study[6] focused on a 10-day adventure race involving 197 competitors and recorded 761 cases of injury and illness in competitors and staff. Skin and musculoskeletal injuries accounted for 86.9% of complaints. Other interesting clinical problems included an unusual pattern of tenosynovitis, sleep deprivation, hallucinations, and hypothermia occurring in the tropics. More than 80 incidents required rescue, including rope rescue, technical carry-out, and helicopter evacuation.

Provision for technical rescue services is but a small part of an overall safety plan for an event, and it should follow a comprehensive program that addresses and mitigates potential hazards to all personnel. Persons responsible for the rescue and medical components should be given sufficient authority and resources to ensure safety as well as effective rescue. Other considerations include questions of jurisdictional authority, medical authority, permits and access, and response protocols.

The Preplan

Before engaging in any high-angle rope training or rescue, responsible individuals should perform a risk assessment to identify necessary skills and capabilities. The preplan should consider the types of terrain in the response area, people exposed to that terrain, types of accidents likely to occur, and available resources.

Exposed personnel must be specifically trained for terrain and environmental considerations commonly encountered in the areas in which they may work. In addition, responders should be trained for the type of incident to which they are responding. For example, a rescuer responding to a fallen ice climber incident in the wilderness must be trained in both high-angle ice rescue and in wilderness SAR. In addition to helping prevent rescuer accidents, this also ensures an appropriate response to the incident.

Having wilderness skills also enables rescuers to work independently of external support and resources in nonwilderness incidents. For example, self-sufficiency and an ability to function with minimal external resources are beneficial when working in the aftermath of an earthquake, or in responding to a transmission tower incident far from a road.

Sometimes an accident demands a combination of traditional fire or EMS response and a wilderness response, and the personnel trained in all of the required disciplines are not available within one agency. It is not unusual for organizations or departments to have mutual aid agreements to maximize training and preparation of personnel. If personnel trained in only one of the necessary disciplines must go into the field on a wilderness operation, it should be only after a thorough risk assessment and after a conscious affirmative decision is made. Even then, rescuers not familiar with a particular environment or type of response should operate only under the direct supervision and care of appropriately trained personnel. Placing an untrained person in a high-angle rope rescue situation to perform patient care, for example, endangers that person, the patient, and others involved in the operation.

Team Training

Responders who are part of a team should train together frequently to ensure good rapport and knowledge of one another's skills. Individual skills are often acquired simply by training with a local team. NFPA 1670[5] outlines some requisite skills for responding to wilderness incidents, but teams responding frequently or to technically demanding wilderness incidents should consider the benefits of technical rescue accreditation by a wilderness-focused organization such as the MRA.

Whereas fire department teams that respond to wilderness rescue incidents may receive only 8 to 16 hours of wilderness-specific technical rescue training per year, Linda Finco, an MRA statistician, estimates that personnel from accredited MRA teams train an average of 140 hours per year. Some teams, particularly those frequently involved in highly technical or complex rescue scenarios, train as frequently as once or twice a week. Team training should take into account the types of incidents likely to occur in the local response area and should prepare team members to deal with general hazards associated with high-angle rescue operations in the wilderness. Considerations include the following:

- Personal psychological hazards: The time to learn about psychological limitations, such as fear of heights or tight spaces, or a psychological dependence on external resources, is *before* an incident occurs.
- Personal physical hazards: High-angle rescuers should be prepared for personal injury and physiologic hazards. Topics include harness fit and comfort, personal protective gear selection, care and maintenance of equipment, rappelling and ascending skills, ropes and knots, proper use of team gear, and specific rescue techniques.
- Environmental hazards: On cliff faces and steep slopes, cracks and crevices in rocks may harbor stinging insects, venomous creatures, and poisonous plants. Rescuers on a steep or vertical face are susceptible to exposure injuries (cold and heat), lightning, and sunburn.
- Terrain hazards: Terrains encountered in high-angle training and operations include rockfalls, avalanches, unstable footing, and high winds.
- Operational hazards: Effecting a rescue has inherent hazards. Personnel moving around the site can increase the likelihood of rockfall or other falling objects. The rigging can pose increased hazards. A safety officer should be appointed at every rescue site to watch for and mitigate any unsafe conditions.

High-angle rope technicians should be trained in appropriate self-care as well as in applicable rescue techniques and appropriate medical protocol for vertical rescue. Training and retraining should occur frequently enough to keep rescuers' skills honed to an appropriate level. Several hours per month is not uncommon.

BASIC SKILLS

Self-care is a critical skill for a wilderness technical rescuer. Rescuers must respect and be prepared for the wilderness environment, including being adept at functioning in it for extended periods—up to 72 hours—even in the most accessible of circumstances. They must be capable of caring for themselves as well as the victim during a rescue operation. Simple survival skills are a poor substitute for the ability to operate comfortably and effectively as a wilderness rescuer. All rescuers must be able to meet reasonable fitness requirements.

TABLE 33-1. Terrain Classification

Class 1	Flat, even—easily traveled by foot
Class 2	Uneven—traveled by foot with some balance required
Class 3	Steep—feet primary means of support, hands may be used to assist; rope a good idea
Class 4	Scramble—feet and hands required; rope necessary for safety
Class 5	Vertical—roped protection required for safety (This category is further divided by the various classification systems compared in Table 33-2.)

TABLE 33-2. Class 5 Climbing Grades: International Equivalents

UNITED STATES	UNITED KINGDOM	FRANCE	AUSTRALIA
5.2		1	
5.3		2	11
5.4		3	12
5.5	4a	4	
			13
5.6	4b	5	
5.7			14
	4c		
		5+	15
5.8			
	5a		16
5.9			17
	5b	6a	18
5.10a		6a+	19
5.10b			
	5c		20
5.10c		6b	
5.10d		6b+	21
5.11a		6c	22
	6a		
5.11b		6c+	23
5.11c		7a	24
5.11d		7a+	25
	6b		
5.12a		7b	
5.12b		7b+	26
5.12c			
		7c	27
5.12d	6c		
5.13a		7c+	28
		8a	29
5.13b	7a	8a+	30
5.13c		8b	
5.13d		8b+	31
5.14a	7b	8c	32
5.14b		8c+	
5.14c	7c	9a	33
	8a		

Wilderness is seldom flat, so field personnel involved in any wilderness rescue should understand terrain and navigation, as well as technical and rope rescue systems. In mountaineering terminology, terrain is generally classified in numeric terms (Table 33-1). When the terrain is vertical, it is subclassified into further levels of difficulty, the most common being the Yosemite decimal system (originated and primarily used in the United States), the U.K. system, and the French system (Table 33-2). A climb may be rated in accordance with the system used by the country it is in or by the nationality of the climber who established the route.

Technical rescuers for wilderness operations should be able to determine, document, and communicate their position; perform point-to-point navigation with a global positioning system (GPS) as well as map and compass; and be adept at route finding using point-of-reference, dead-reckoning, and map skills.

At the most basic level, sometimes referred to as the *awareness* level, the priorities for a responder's individual skills are to be able to recognize that a wilderness technical rescue situation is developing, to know how to activate appropriate resources, and to support the ensuing rescue. This involves being able to perform a realistic analysis of terrain hazards, weather conditions, and limitations of available resources. Would-be rescuers have been injured or killed because their initial reaction to an incident was inconsistent with their level of training or equipment.

In any emergency, it is difficult to wait for the arrival of additional resources. Yet, responders at the awareness level can tip the scales in favor of the victim by focusing their energies on effectively securing the scene and initiating the collection of key information that will ultimately help resolve the incident. This sizing up of the scope and magnitude of the situation sets the stage for the rescue, and a corresponding response is initiated. In the critical first moments, a risk-to-benefit analysis is initiated, and the safety of the rescuers and of the victim is provided for. An effective initial response can enhance the probability of success.

More advanced skills for wilderness rope rescue expand on the basic principles. NFPA 1670[5] identifies a *response* level of training, which corresponds with the MRA *support* level. At this level, responders are capable of functioning effectively on a technical rescue (one that requires rope and special equipment and skills) in the wilderness, and of working under the direction of more experienced personnel. The next NFPA level is the *technician* level (corresponding with the MRA *rescue* level). Here, technical requirements are expanded and honed. The MRA adds yet another level of expertise, the *qualified* level, at which an individual's breadth and depth of training, as well as experience, results in unsurpassed expertise. Experience is the key when it comes to making subjective decisions about technical evacuations in the mountains.

Personnel operating in mountainous terrain where steep and vertical slopes are common should be comfortable functioning in that terrain before engaging in mountain rescue. Familiarity with basic climbing techniques is essential, as is the ability to rappel and ascend rope. Techniques similar to those used recreationally are sufficient, so long as adequate safety is provided. Other useful skills to develop and practice include leadership under stressful situations, interfacing with aircraft and watercraft, provision of specialized medical care, patient care during extended transport, and equipment transport over terrain.

Rope rescue can be intense and demanding and may require more than just one or two experienced individuals to accomplish an effective response. Most teams should have several people trained to the NFPA response (or MRA support) level. Even with several people trained to this level, it is important to have at least a few with a strong depth of experience in a variety of terrains, or at the technician (or rescue) level. A skill achieved

on a steep slope, such as a lower or raise, can change dramatically as the angle increases. As the geometric influences change, opportunity for and consequences of failure increase as well.

Finally, active teams should have several people who meet the MRA requirements for *qualified* rank. True skill in this arena requires personnel who are honed in every way and backed with a depth of experience and decision-making ability. In addition, enhanced rigging skills and concepts are required—specifically, load-distributing anchors, highlines, and knot passes. Training in these concepts is stimulating and even enjoyable, but proficiency requires much repetition and numerous hours of training.

A reasonable path of education begins with the selection, care, and maintenance of appropriate rope rescue equipment. It expands to personal rope skills such as ascending and descending a rope. Finally, it extends to proficiency in high-angle systems, including raising and lowering systems, highlines, track lines, anchor systems, and belays.

Training alone, however, is not sufficient. High-angle wilderness rescue personnel should have the appropriate attitude and aptitude for working on steep terrain, sometimes for extended periods. Appropriate preparation depends on the environment in which the rescuer will function—hot, cold, dry, wet, snowy—and the length of time it will be necessary to stay in that environment. Gear must be specific for the environment.

PERSONAL PROTECTIVE GEAR

The well-prepared high-angle rescuer has equipment that is appropriate for and designed to function effectively in the wilderness environment (Fig. 33-4). The following list is an example of equipment that might be needed:

- Helmet: well fitting, with ventilation holes and a secure three-point suspension (Fig. 33-5)
- Eye protection: close fitting to protect against sun, wind, debris
- Clothing: appropriate moisture transport (wicking or non-wicking), appropriate insulation, wind- and water-repellent
- Gloves: appropriate for temperature and protection
- Footwear: well fitting, lug sole, appropriate to the environment
- Backpack: appropriate capacity; protection against injury in the vertical environment

There are several versions of the "10 essentials" list for mountaineers and wilderness travelers (including such items as water, matches, and compass), and this is a good starting point for the rescuer as well. However, the purpose of the equipment is not just survival but should instead focus on function. The high-angle rescue technician is a professional, focused on operating effectively, not just surviving!

PATIENT CARE AND TRANSPORT

The level of patient care that a responder can provide depends on circumstances, training, and resources. A spontaneous response provided when an incident happens in front of the responder is different from a planned response by a trained rescuer, and the expectations differ also.

In a spontaneous incident, it is reasonable and prudent for a bystander to render basic emergency care while notifying

Figure 33-4. Rescuer's personal protective equipment: helmet, gloves, footwear, backpack. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

Figure 33-5. Three-point suspension on a helmet. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

Figure 33-6. A Hasty pickoff seat. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

Figure 33-7. Spec Pak. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

authorities, and to continue until trained rescuers arrive. Those with some level of medical training have a slight advantage in understanding what needs to be done to evaluate the scene, protect the patient (and others) from further harm, and perform basic life support until help arrives.

Those who expect to respond intentionally to outdoor emergencies should be trained at least to an emergency medical technician basic or first-responder level, or the outdoor equivalent of one of these, and they should be able to organize and participate in a progressive litter carry on moderate to rugged terrain. If steep or vertical terrain is likely to be encountered, they should have a good working knowledge of rope rescue systems.

Extrication and evacuation of a patient from steep or vertical terrain requires special consideration in terms of practicality and patient protection. Some patients may already be wearing a harness, and the rescuer needs to determine whether it is appropriate to use the patient's harness for extrication or to use an adjunct. Using the patient's own gear is often discouraged, because the rescuer cannot know its history or condition. However, sometimes this is the best alternative available. When it is reasonable and advantageous to use the responder's gear for the evacuation, and when the patient is not injured, the best choice is usually an evacuation seat (Fig. 33-6), which is easy to put on the patient and usually feels more secure than a typical harness.

When the patient is injured and must be moved from complex terrain, the combination of a harness and an evacuation device is an excellent tool. Some common examples of these are the Spec Pak and LSP Half Back (Fig. 33-7). This type of equipment requires special medical and technical training and expertise to avoid further injuring the patient.

Depending on the patient's medical condition and injuries, it may be prudent to place him in a litter before moving him. Chapter 34 discusses litters and carries in greater detail. For vertical operations, rigid-basket-style litters offer greater patient protection and comfort than do flexible litters and are the preferred choice for rope rescue evacuations.

Rigid Basket Litter

The first priority of a high-angle rescue is the safety and protection of the patient. Numerous accessories for rigid litters are available that are specific for this use (Fig. 33-8). Immobilization can be accomplished with a narrow-profile backboard, but beanbags are by far the preferred immobilization device for litter evacuations. The patient's head and face should be protected either with a helmet and sunglasses or with a litter shield (Fig. 33-9).

Special attention should be given to securing the patient into the litter for a high-angle evacuation. This can be improvised using long strands of webbing, or special tie-in straps may be used (Fig. 33-10). For steep or vertical terrain, seat belt straps are not sufficient. The system chosen should thoroughly secure the patient into the litter and protect against further injury.

Although inversion or tipping of the litter may seem unlikely, steps should be taken to protect against the consequences if it should happen. The patient should be secured not only with straps over the body but also from slipping out either end of the litter. Foot loops, shoulder loops, and waist attachments are all excellent for this purpose. In addition, if the patient is wearing a harness, a security strap from it to the main lowering or belay line is a good idea.

Litter wheels, usually preferred for trail use, can also be useful in slope evacuation, particularly when there are a limited number of rescuers. Short, fat tires such as those on all-terrain vehicles are stable on a horizontal plane and absorb trail shocks well (Fig. 33-11). Larger, dirt-bike tires are more efficient and maneuverable (Fig. 33-12).

Figure 33-8. Cascade Professional Series litter. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

Figure 33-9. Patient protection: helmet and glasses or goggles. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

Figure 33-10. FAST patient tie-in. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

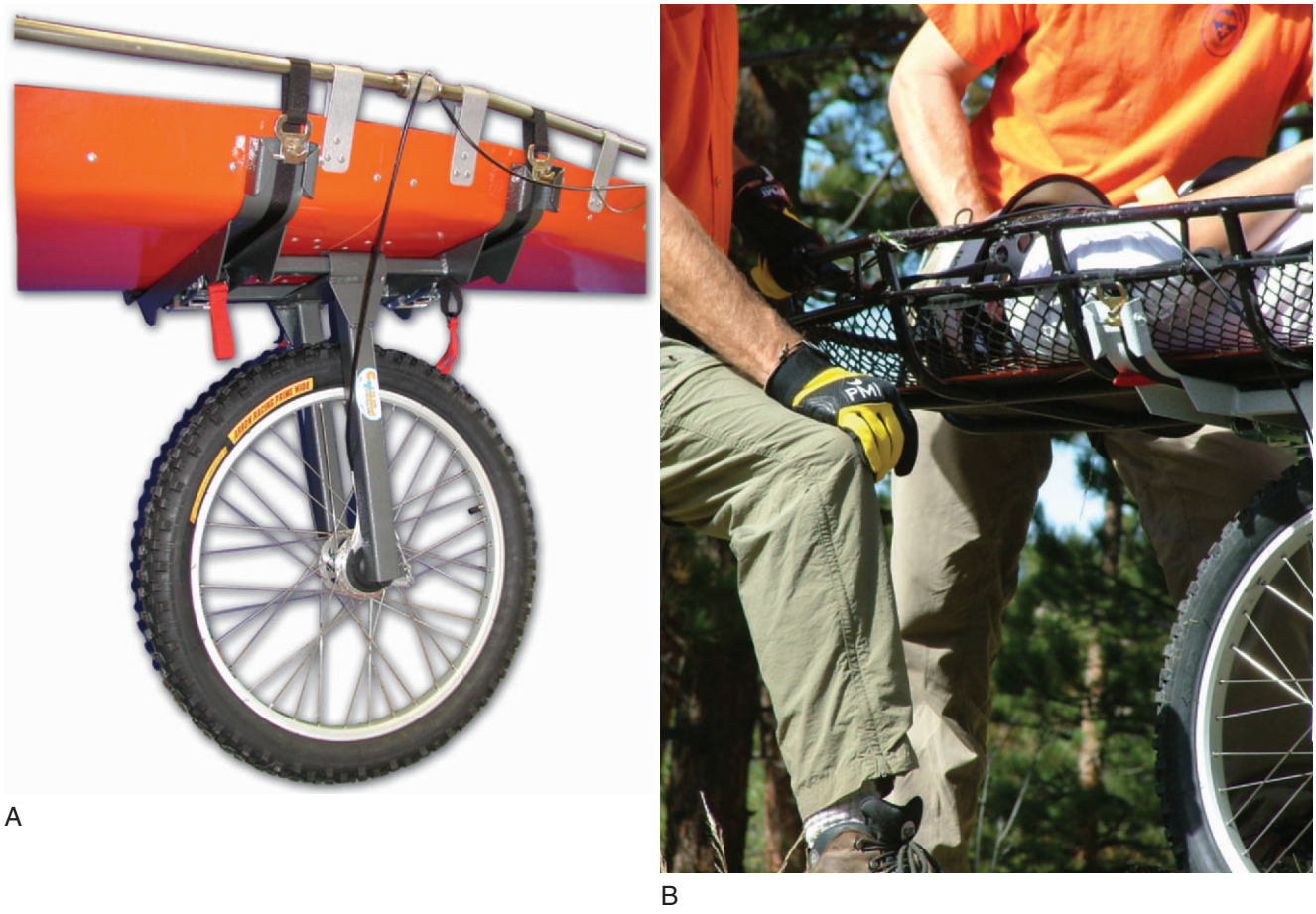
Figure 33-11. All-terrain vehicle (ATV) tire litter wheel. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

Litters can be rigged for high-angle evacuation in either a horizontal (Fig. 33-13) or a vertical (Fig. 33-14) configuration. Vertical configurations offer a lower profile for rockfall, but they can be quite disconcerting for the patient. In the United States, high-angle rescue is usually done with the litter in a horizontal configuration.

The litter is supported and connected to the end of the lowering line by a device commonly called a litter spider or bridle. These are available in different forms, ranging from four litter attachment points and a single mainline connection (Fig. 33-15) to six litter attachment points and dual mainlines (Fig. 33-16). Each system offers advantages and disadvantages. Persons wanting to begin a high-angle rescue program, or to update their current technology or systems, should seek specialized training from a qualified professional.

Flexible Litter

The light weight and compact size of flexible litters make them attractive choices for expeditions or other small-team situations. They can also be convenient solutions in caves, mines, or other confined spaces. Flexible litters are best used with some sort of spinal immobilization, and they are not well suited to high-angle rope rescue without additional equipment. They can be placed inside a larger rigid-frame litter once the patient is removed from the confined space, for quicker lowering and raising operations without a complete repackaging of the patient.



A B

Figure 33-12. **A,** Motocross-tire litter wheel uses a secure universal saddle to attach to any litter. **B,** Motocross-tire litter wheel provides clearance and maneuverability for difficult terrain. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

Figure 33-13. Litter rigged in horizontal configuration with basic single rope attachment. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

It may be feasible to use a flexible litter (Fig. 33-17) for high-angle rescue, but each brand and model requires specific training. The rescue team must be well trained in the specific high-angle rigging recommendations from the manufacturer of the chosen flexible litter.

RESPONDING TO THE WILDERNESS INCIDENT

Although different situations require different responses, every responding group or agency can be expected to establish protocols and training criteria that are appropriate to the training and experience levels of its members. Military organizations provide a good model, refined over the years, for managing varying numbers of people in constantly changing field circumstances, and many rescue teams find it convenient and effective to function in a paramilitary manner. Teams may establish personnel ranks to help quantify levels of capability. At the top of the rank is a command structure that helps to streamline

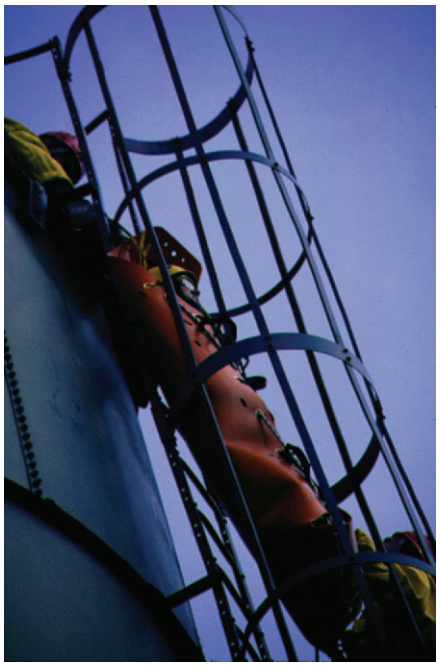


Figure 33-14. Litter rigged in vertical configuration. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

Figure 33-15. Four-point litter spider. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

operations and ensure that qualified personnel manage the incident.

The Incident Command System (ICS) or Incident Management System (IMS), a framework developed initially to assist in managing wild-land firefighting operations, has been adapted by many for wilderness rescue operations. Most professional organizations, paid or volunteer, use some variation of the ICS to manage operations.

Figure 33-16. Six-point litter spider. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

Figure 33-17. Sked flexible litter. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

In the ICS, an incident commander assumes operational control of an entire incident and has several section chiefs to oversee different aspects of the incident. The sections typically include operations, planning, logistics, and finance or administration. The ICS-100 class, available through many municipal governments, is a good introduction to this system.

Field personnel typically operate primarily under the operations section of the ICS system. Most wilderness technical rescuers have a combination of technical and medical skills. For wilderness rope rescue operations, the need for technical skills generally outweighs the need for medical skills. Most rescues involve a finite number of patients (usually only one or two), but every responder will probably be faced with the technical aspects of the evacuation.

When neighboring teams often find themselves working together on an incident, it is advisable to synchronize personnel training and team structure, at least to some degree. Having similar ranking systems and operational criteria facilitates working together. Some teams become accredited under national criteria, such as those of the MRA, for just this reason.

The Callout

Members of technical mountain rescue teams who respond at the request of local authorities often carry a pager so that they may be contacted quickly in the event of an emergency. When the pager goes off, members rendezvous at team headquarters or at a staging area (the command post or mission base) from which the event is initially coordinated.

As soon as a command post or mission base is established, it is given a unique name to identify it, in case another command post must be established for the same mission at a later time, or in case another mission occurs within radio range while the original mission is still going on. Often the name reflects the search area (e.g., Chester Valley Command).

Scene Size-Up

Some rescue organizations classify types of rescue according to the degree of slope involved in the evacuation. According to NFPA 1670,[5] *low-angle* refers to an environment in which the load is predominantly supported by itself and not the rope rescue system (e.g., flat land or mild sloping surface), and *high-angle* refers to an environment in which the load is predominantly supported by the rope rescue system. The first person on the scene immediately sizes up the situation and activates appropriate resources. Reevaluation of size-up criteria is then a continuing process throughout the duration of the incident. In addition to collecting information specific to the incident, a wilderness environment size-up should consider the following factors.

Scope and Magnitude of Wilderness Influence. Is the incident 100 yards from a vehicle access point, or several miles into the backcountry? Is the best evacuation route at the top or at the bottom of the slope or cliff? The first person on scene should ask these kinds of questions to determine necessary resources.

Scope and Magnitude of High-Angle Influence. Is the high-angle obstacle to be negotiated a steep slope, or is it a cliff? How large or accessible is the slope or cliff? Are decent anchors available? The scope and magnitude of the incident will affect the type and number of resources requested.

Assessment of Time Required. How long will the evacuation take? Will there be darkness and lighting considerations? How physically demanding will the operation be, and how often will rescuers need to be rotated for rest? The answers will influence resource requirements and may indicate that additional resources must be called in from farther away.

Assessment of Manpower Needs. Given the time and work to be done, will changing shifts be a consideration? Is it necessary to keep responders available for a second incident in the area? Are there sufficient resources within the organization, or will external resources be required? Dedicating all resources in a given area to one incident requires consideration in advance. Prearranged agreements with nearby agencies can be very useful in staffing a large incident or for backup in case of another call.

Specific Environmental Factors Involved. Are available personnel suitably trained and equipped for the terrain that will be encountered? Is there a river or lake in the area that will require additional personnel? What is the time of year? Is snow or ice a consideration? The steepness of terrain as well as groundcover must be considered when estimating whether personnel are adequately prepared to function.

Integrity and Stability of the Environment Involved. Is a storm coming? Will night fall before the operation is done? Planning to accommodate for changes in weather, ground instability, and other environmental factors must be done several hours in advance.

Number of Known and Potential Victims. When the potential of additional victims becomes a reality, the number of rescuers needed increases, and other stresses emerge. A new sense of urgency arises, and there is a need for triage, more equipment, more time, and more resources for evacuation. Additional potential victims must be anticipated.

EVACUATION TECHNIQUES

It is not possible to predict the rope rescue systems and techniques that would be most appropriate for every response. The choice is subjective and depends, for example, on environmental conditions, the nature of the incident, height considerations, and the number of rescuers available. Responders should be led by rescuers with enough experience and training to make good decisions.

Low-Angle Litter Lowers

Low-angle evacuations are typically performed in terrain that allows the majority of the rescuer's weight to be supported by the ground, but a rope is used in addition for the safety and security of the rescuers. Examples of such terrain are snow slopes, inclined land near bridges, river embankments, scree or talus fields, and roadsides.

A typical low-angle litter lower is a personnel-intensive rescue. A site commander should be assigned to manage the operation. This role requires depth of experience, knowledge of personnel and techniques, and familiarity with the terrain. Many teams assign the most experienced person on site to be in charge of site safety, responsible for checking and verifying all aspects of site safety. This person must be free from any other responsibilities or distractions. Other roles and responsibilities include brakeman, rope handlers, and medical personnel. The medical person may serve also as a litter bearer or stay separate from the system. Usually, four litter-bearers carry the patient, but the number may be as few as three or as many as six.

Either a plastic shell litter or a metal basket litter may be used for a low-angle evacuation. A plastic shell litter provides more protection to the patient from underbrush and ground cover, but it is not as rigid as the metal basket and offers fewer options for rescuers to tie into or hold onto the litter.

A litter bridle (Fig. 33-18) may be used, or the rope may be tied directly into the litter by wrapping it several times around the head of the litter (Fig. 33-19) and then tying off with a

Figure 33-18. Litter tied in with bridle for low-angle evacuation. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

Figure 33-19. Litter tied in directly for low-angle evacuation. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

bowline or figure-8, so that the litter is in line with the slope. Wrapping the litter several times like this helps to distribute the force across a wider area, gives some lateral stability to the litter, and helps to avoid undue pressure on the weld that is often found at the center-head of a wire basket litter.

The main line of the system supports the load as it is being lowered down the slope (Fig. 33-20). A secondary, or belay, line can be incorporated into the system. This decision should be made in accordance with an appropriate risk-to-benefit assessment.

The number of rescuers required for a low-angle evacuation depends on several factors, including the degree of slope, the weight and medical condition of the patient, and the stability of the environment. In flatter terrain, less of the patient's weight is taken by the rope and more must be carried by litter bearers. A litter wheel may be used in a low-angle evacuation to help support the weight of the litter.

On steep slopes, rescuers can gain a certain amount of leverage by wearing a harness and connecting to the litter with cordage or webbing (Fig. 33-21). This adds stability and helps support the litter. Tie-ins may be made of cordage or webbing and are hitched to the litter rail before being clipped to the litter bearer's harness with a carabiner. If a split litter is used, tie-ins should be connected to the head-end of the litter to avoid undue stress on the connections.

Other techniques, including the use of hauls and counterbalances, hand passes, and litter wheels, may be combined with low-angle evacuation techniques or, occasionally, used in their stead. Further direction and instruction regarding low-angle rescue may be found in specialized textbooks.[7]

Vertical Litter Lowers

When vertical or near-vertical terrain dictates a high-angle rescue system, personnel with specific experience in these techniques should be deployed. Proficiency in high-angle rescue techniques takes a great deal of training and experience.

The most common type of high-angle rescue system is known as a single-point vertical fixed-brake lower (Fig. 33-22). This type of system requires at least six well-trained, skilled rescuers. For this, as with the low-angle operation, a site commander should be assigned to oversee all aspects of the operation. Other roles involved include site safety, brakeman, and rope handlers. A belay system is almost always utilized in high-angle rescue operations, in which case a belayer and separate rope handler also become necessary.

High-angle rescue operations generally utilize only one or two litter bearers, although one or two additional persons may be required to assist the litter in getting over the edge. These "edge tenders" are also responsible for ensuring that ropes and equipment are well protected as they go over the edge.

Occasionally, a separate rappel or lowering line is arranged for a medical attendant or for an extra person to assist with the technical aspects of a mid-face loading procedure. This "third man" is especially valuable as because he or she is not linked directly to the lowering system.

Variations on the basic single-point fixed-brake litter lower include the single-point traveling-brake (Fig. 33-23) and the two-rope (scaffold) vertical lowering system (Fig. 33-24). These systems are even more complex and require special training and skills beyond those required for the single-point fixed-brake lower.

Raising and Haul Systems

Although the knowledge of lowering systems is essential for competent rope rescue personnel, many rescues involve raising a victim from a low point to a high point. This may occur as part of a lowering system or separately—as in the case of a victim who has fallen into a gorge, or where the carryout trail is higher than the victim's location.

Mechanically, the simplest means of raising a load is via a direct pull. Physically, however, this is not always feasible. The force required to move a load can be greatly reduced by using pulleys and rope to create a mechanical advantage. With a mechanical advantage system, a relatively heavy load can be

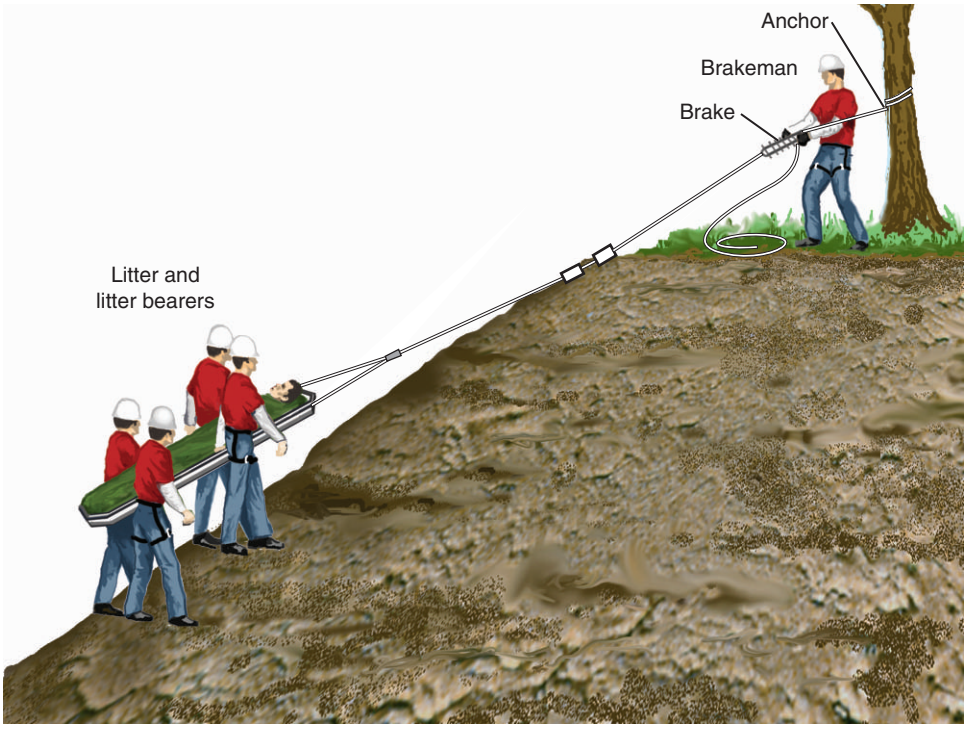


Figure 33-20. Example of the rigging and teamwork in a low-angle rescue. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

Figure 33-21. Low-angle litter bearer tie-in, in use. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

moved with minimal force. Depending on the configuration of pulleys and rope, varying amounts of mechanical advantage can be produced, and the amount of the advantage can be calculated. This haul capability is generally expressed as a ratio. For example, a system wherein a 100-pound load could be moved with a calculated 50 pounds of force is a 2:1 (two-to-one) mechanical advantage, known as a theoretical mechanical advantage.

The theoretical mechanical advantage is always greater than the actual mechanical advantage. The actual mechanical advantage produced by a hauling system depends in great part on how efficiently the system is rigged and on the efficiency of the components used to build it. Designing haul systems and optimizing mechanical advantage are advanced skills and require special training and expertise.

Alternative Techniques

Occasionally, a terrain is too complex to allow the use of a basic low-angle or high-angle raising and lowering system, in which case a number of techniques are available. One group of techniques that is particularly useful involves a *highline*, which is a rope suspended between two points and across which personnel and equipment can be moved. Highlines can be simple or very complex to rig. Even at their simplest, however, highlines are a time-, personnel-, teamwork-, and communications-intensive technique. At the simplest level, a highline may consist of a single line, or "main line," across a span. Adding a second main line results in a dual main-line system.

One of the most difficult parts in rigging a horizontal highline is getting personnel from the near side of the operation to the far side of the operation. Because a highline often spans difficult terrain such as a river or gorge, the process can be time consuming. Once personnel are at the far side of the operation, the rope can be sent across using a lighter-weight leader-line launched by a giant slingshot or line gun.

Perhaps more than any other rope rescue technique, highlines have the potential for overstressing rope, equipment, and anchors, thus causing failure of the system. Rigging and use of highlines require thorough knowledge of the potential forces involved.

A highline system may include one or two main lines, also known as track lines. These should be of a low-stretch design, such as static kernmantle (see Chapter 84), to avoid excessive

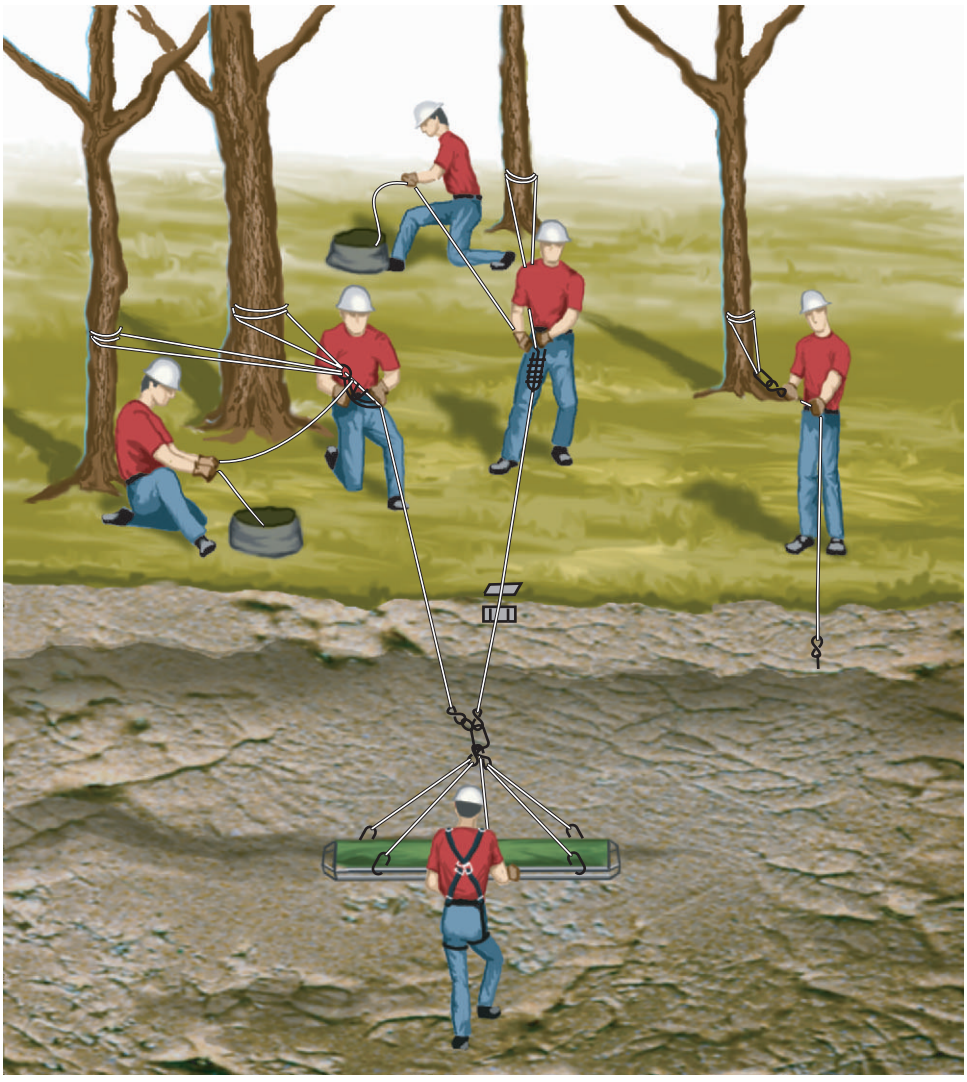


Figure 33-22. Example of the rigging and teamwork in a high-angle rescue. (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

stretch in the system. Track lines should be rigged with sufficient sag (up to 10%) to prevent overstressing the system.

The main lines should be attached to very trustworthy anchors at each end. At least one of these anchors should be rigged with a tensioning system to allow the sag in the highline to be adjusted. Pulleys are used to facilitate movement of the load across the track line or lines. Nearest the point of origin of the operation, a lowering system is helpful for controlling the load as it starts across the highline. At the other end, a haul system may be required to bring the load back up the other side of the sag. These additional systems may also be incorporated into a safety (or backup) for the highline.

A complex highline is a good test of many different rope rescue skills, particularly those incorporating special rigging known as a reeve. Such systems can make it possible to raise and lower loads from mid-span.

The personnel requirements for operating a highline are quite similar to those for a low-angle or vertical lowering operation, but multiplied by the complexity of the system. There should be a site commander at each end of the system, plus sufficient personnel to operate whatever lowering or raising systems are incorporated.

A highline may be rigged at a steep angle to provide a "track" for lowering or raising personnel or equipment above obstacles. This type of highline, sometimes called a guide line, can be very useful in negotiating difficult terrain.

Such was the case in an area known as the Wonderland of Rocks at Joshua Tree National Monument several years ago, when a 250-lb (113-kg) Boy Scout sustained a tibia-fibula fracture and ended up stuck in a crack between house-size boulders. Wonderland of Rocks is a unique formation involving more than 6 miles of huge boulders jumbled across the desert floor. The boulders range from the size of a car to the size of a large house. They are stacked and strewn upward and outward so that the area is largely impassable—making it a challenging and enjoyable area for scrambling, hiking, and technical climbing. One miscalculation, however, can have severe consequences.

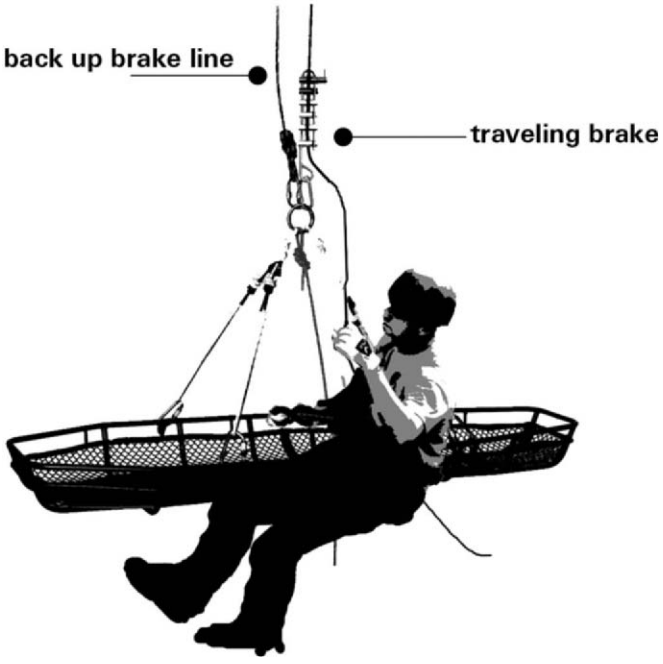


Figure 33-23. Traveling-brake system (vertical). (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

Figure 33-24. Scaffold system (vertical). (Courtesy Pigeon Mountain Industries, Lafayette, GA.)

This particular accident scene was visible from an adjacent access road, but accessing it required an hour of strenuous scrambling and climbing. At outset, it seemed that the 20 or so rescuers who responded would need to rig a vertical raise to extricate the patient, followed by a series of high- and low-angle lowers and raises in series to get the patient to the desert floor. From there, the carry-out would involve crossing a sandy dry riverbed and then a short haul up a slope to reach the road. The operation would take many hours, and additional resources would be required. The patient, who was in great pain, would be subjected to a grueling evacuation.

The alternative evacuation method involved a track line system with a short raise at the top to extricate the patient. Two 600-foot ropes were located, and the track line that was established allowed the patient to soar over the difficult terrain in a fraction of the time that would have been required for the original evacuation plan.

SUMMARY

Wilderness can be defined as "an uncultivated, uninhabited, and natural area usually, but not necessarily, far from human civilization and trappings."[5] A wilderness rescue, then, is one that occurs in this environment, and a technical wilderness rescue is a wilderness rescue that requires rope and special equipment and skills. Depending on the terrain and on environmental factors, a wilderness incident may be as little as a few minutes into the backcountry or a few feet off the roadway.

Specialized mountain or wilderness rescue teams are common in only a fraction of the United States, but this does not preclude the rest of the country from susceptibility to an occasional wilderness type of incident. Advance planning, preparation, and training are the best way to mitigate the hazards associated with this type of incident.

The critical thing, according to MRA legal advisor David Thorson (www.mra.org), is not that one particular standard or another be followed, but that an organization acknowledge the existence of a given standard and make a conscious decision to either abide by or deviate from it.

There are several resources for information on accepted practice and standards for technical wilderness rescue. The ASTM (formerly the American Society for Testing and Materials) F32 Committee on Search and Rescue is a national standards body that has authored several in-depth national standards on search-and-rescue equipment, training, and resources.[1] The MRA[2] provides resources and accredits teams in skills and capabilities for technical rescue operations in the mountains. The National Association for Search and Rescue (NASAR)[3] is an excellent resource but does not yet provide certification for technical rescue skills. It has developed the SAR-TECH program for individuals.

In addition, some states, including Maine and Pennsylvania, have developed standards for SAR, and individual teams standardize everything from techniques and equipment to the color of their webbing.

High-angle technical rescue in the wilderness environment is a very specialized field. Medical personnel and rescuers interested in engaging in high-angle rescue work should receive thorough training and complete an apprenticeship. Further direction and instruction regarding high-angle rescue may be found in specialized textbooks.[7]

The references for this chapter can be found on the accompanying DVD-ROM.

34 Litters and Carries

Donald C. Cooper, James Messenger, and Timothy P. Mier

Every search and rescue event goes through a series of four consecutive phases. These phases are illustrated by the acronym LAST (locate, access, stabilize, and transport). This process ends with the movement of the patient (or patients) from the scene to either a medical facility or an area of comfort and safety (transport)[6] (see Chapter 32, Search and Rescue).

In the United States, the term *stretcher* suggests a flat, unsophisticated frame covered with canvas and used for carrying the sick, injured, and deceased short distances. The term *litter* can mean the same thing, but usually suggests an apparatus specifically designed to immobilize and carry a patient longer distances. Over the years, the subtle differences in the terms have been lost, and users have gravitated to one or the other. In the United States, the term litter is used to describe all manner of rescue conveyance. In Great Britain, however, the preference is to use the term *stretcher* to describe the same devices. In this chapter, the two terms are used interchangeably.

SIZE-UP

To select the best method for getting a patient to definitive care, the rescuer must make a realistic assessment of several factors. Scene safety is the initial priority. The necessary evaluation, called the *size-up* (Box 34-1), involves a (usually hasty) determination of whether the victim, rescuer, or both are immediately threatened by either the environment or the situation. Proper immobilization and patient packaging are always preferable, but sometimes the risk of aggravating existing injuries is outweighed by the immediate danger presented by the physical environment. In such a situation, the rescuer has little choice but to immediately move the patient to a place of safety before definitive care is provided or packaging is completed.

Evacuation options are limited by three rescuer-related variables: (1) the number of rescuers, (2) their level of fitness, and (3) their technical ability. Carrying a victim, even over level ground, is an arduous task. At an altitude where just walking requires great effort, carrying a victim may be impossible. The specific rescue situation or environment encountered also may present challenges beyond the capability of the available rescuers. Complex rescue scenarios requiring specially trained personnel and special equipment are called *technical rescues* and often involve dangerous environments, such as severe terrain, crevasses, avalanche chutes, caves, or swift water. To avoid becoming victims themselves, rescuers must be realistic when evaluating their ability to perform these types of rescues.

DRAGS AND CARRIES

The most fundamental and expedient method of transporting an ill or injured person is by dragging or carrying him or her. Although these methods of transportation are far less than ideal and may not meet standard care criteria, the urgency of the situation may outweigh the risks involved. In addition, the process can be physically demanding, and rescuers can quickly become fatigued to the point of hazard. Therefore, other options often should be considered before a victim is moved, especially a long distance. A drag or carry may be the best option when a person cannot move under his or her own power, injuries will not be aggravated by the transport, resources and time are limited, the need for immediate transport outweighs the desire to apply standard care criteria, the travel distance is short, or the terrain makes the use of multiple rescuers or bulky equipment impractical.

A *blanket drag* (Fig. 34-1A) can be performed on relatively smooth terrain by one or more rescuers rolling the victim onto a blanket, a tarp, or even a large coat and pulling it along the ground. This simple technique is especially effective for rapidly moving a person with a spinal injury to safety because the victim is pulled along the long axis of the body. In extreme circumstances, the *fireman's drag* (see Fig. 34-1B) can be used. In this type of drag, the rescuer places the bound wrists of the victim around his or her neck, shoulders, or both and crawls to safety.

A carry should be considered only after it is confirmed that the victim cannot assist rescuers or travel on his or her own. Beyond simply lifting a person over one's shoulder in a *fireman's carry* (Fig. 34-2) or acting as a human crutch, a more efficient one-person carry can be accomplished by using equipment, such as webbing, backpacks, coils of rope, or commercial harnesses. Equipment-assisted carries are particularly effective when an injured climber or hiker must be evacuated across a short distance over rough terrain or when a person must be quickly removed from a hazardous environment. In the simplest equipment-assisted carry, 4.5 to 6 m (15 to 20 feet) of webbing is wrapped around the victim, who is "worn" like a backpack by the rescuer (Fig. 34-3).

Similarly, a backpack or split coil of climbing or rescue rope can be fashioned into a seat around the victim and hoisted by the rescuer. A rescuer can modify a large backpack by cutting holes in the bottom for the victim's legs, who then sits in it like a child would sit in an infant carrier (Figs. 34-4 and 34-5).

Box 34-1. Evacuation Size-Up Factors

What are the scope and the magnitude of the overall situation?
Are there immediate life-threatening hazards?
What is the location, and how many victims are there?
What is the patient's condition? Is the subject able to assist rescuers?
a. No injury (able to walk unassisted)
b. Slight injury (able to walk unassisted)
c. Slight injury (assistance required to walk)
d. Major injury (requires considerable attention and assistance)
e. Deceased
Is there a need for technical rescue?
Is the scene readily accessible?
What rescue resources (including rescuers and equipment) are available?
How far must the patient (or patients) be transported?
Are ground or air transport assets available?

Figure 34-1. **A,** Blanket drag. **B,** Fireman's drag. Both techniques are intended to be used when expeditious transport over a short distance is required. (From Auerbach PS: Medicine for the Outdoors: The Essential Guide to Emergency Medical Procedures and First Aid, 4th ed. New York, Lyons Press, 2003.)

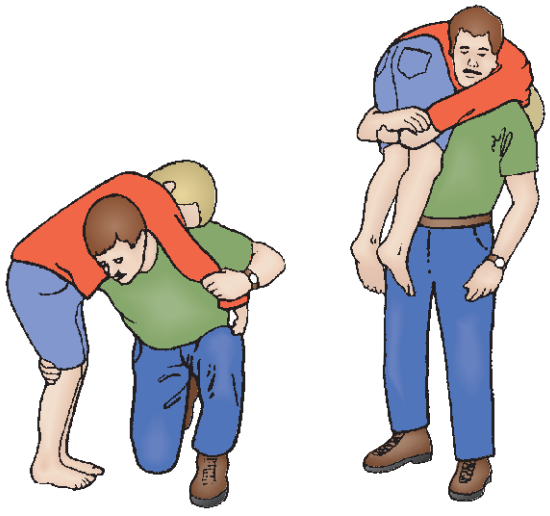


Figure 34-2. Classic fireman's carry: a single rescuer technique for short distance transport only. The rescuer must use his or her legs for lifting. (From Auerbach PS: Medicine for the Outdoors: The Essential Guide to Emergency Medical Procedures and First Aid, 4th ed. New York, Lyons Press, 2003.)

A few commercial harnesses allow a lone rescuer and single patient to be raised or lowered together by a technical rescue system. A Tragsitz is one example (Fig. 34-6).

For carrying infants and small children, a papoose-style sling works well and can easily be constructed by the rescuer tying a rectangular piece of material around his or her waist and neck to form a pouch. The infant or child is then placed inside the pouch, which can be worn on the front or back of the rescuer's body.

If two rescuers are available, additional and often superior options for carrying a victim are possible. One option consists of two rescuers forming a seat by joining their hands or arms together. The victim sits on the "platform" and holds on to the rescuers for support. It is difficult to cover a long distance or rough terrain when using this technique (Fig. 34-7).

A coil of climbing or rescue rope can be used to form a *two-rescuer split coil seat,* with each rescuer slipping a side of the rope coil around his or her outside shoulder (Fig. 34-8). The patient then sits on the "seat" formed by the rope. A similar approach involves using padded ski poles or stout limbs tied together and supported by backpacks worn by rescuers. The victim sits on the supported poles with his or her arms around the rescuers' shoulders. If the poles are properly padded and securely attached to sturdy rescuers, this technique can be quite comfortable for both rescuer and victim. This approach requires gentle terrain without narrow trails.

Spine injuries generally prohibit the use of drags or carries because the victim cannot be properly immobilized, but drags or carries may be acceptable when immediate danger outweighs the risk of aggravating existing injuries. Drags are particularly useful for victims who are unconscious or incapacitated and unable to assist their rescuer (or rescuers), but may be uncomfortable for conscious victims. When a drag is used, padding should be placed beneath the victim, especially when long

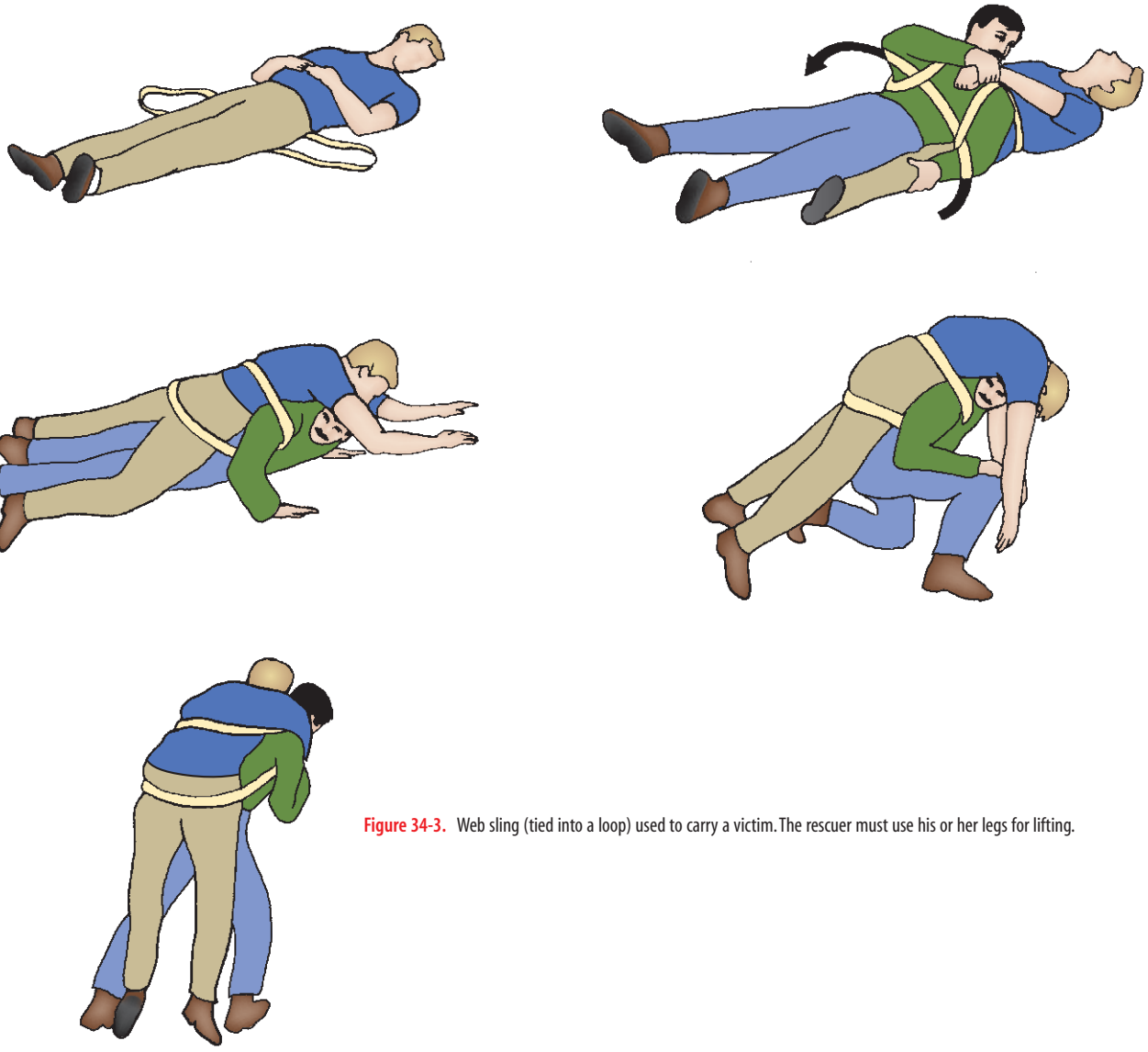


Figure 34-3. Web sling (tied into a loop) used to carry a victim. The rescuer must use his or her legs for lifting.

Figure 34-4. Backpack carry.

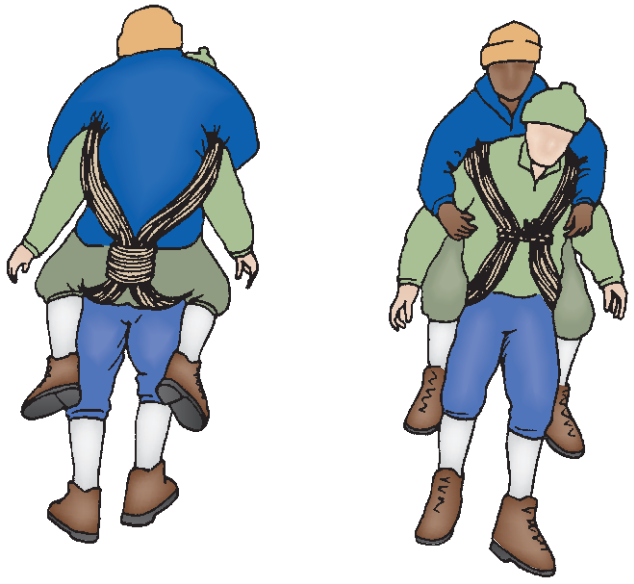


Figure 34-5. Single-rescuer split coil carry. Note that the coil can be tied in front of the rescuer, and the wrists of the victim can be bound and wrapped around the rescuer's neck for more stability.

distances are involved. The high fatigue rate of rescuers makes carries less attractive options when long distances are involved.

LITTER IMPROVISATION

The simplest improvised litter is made from a heavy plastic tarpaulin, tent material, or large polyethylene bag (Fig. 34-9). By wrapping the material around a rock, wadded sock, or glove and securing it with rope or twine, the rescuer can fashion handles in the corners and sides to facilitate carrying. The beauty of this device is its simplicity, but it can be fragile, so care must be taken not to exceed the capability of the materials used. As an additional precaution, all improvised litters should be tested with an uninjured person before being loaded with a victim. This type of nonrigid, "soft" litter can be dragged over snow, mud, or flat terrain but should be generously padded, with extra clothing or blankets placed beneath the victim.

A coil of rope also can be fashioned into a litter, called a *rope litter* or *clove hitch stretcher,* but a 46- to 61-m (150- to 200-foot) climbing or rescue rope is required (Fig. 34-10). The rescuer constructs the litter by laying out 16 180-degree loops of rope (8 on each side of center) across an area the desired width of the finished litter. The running ends of the rope are used to tie a clove hitch around each of the loops, and then the unused portion of rope can be passed through the loops on the other side and tied off. The litter is then padded with clothing, sleeping pads, or a similar material. Lateral stability can be added by tying skis or poles to the finished product. Because of its nonrigid construction, this litter offers little back support and is best suited for victims with injuries that do not require immobilization.

Figure 34-6. Tragsitz harness in use.

A sturdy blanket or tarp can be used in combination with ski poles or stout tree branches. The blanket or tarp is stretched over the top of two poles, which are held about 1 m (3 ft) apart; tucked around the far pole; and folded back around the other pole. The remaining material lies over the first layer to complete the litter (Fig. 34-11). The weight of the victim holds the blanket in place. A similar device can be improvised by passing the poles through the sleeves of two heavy, zipped (closed) parkas.

It may be necessary to transport victims with certain injuries (i.e., spine injuries; unstable pelvis, knee, or hip dislocations) on a more rigid litter. Ski poles, stout tree limbs, or pack frames can provide a rigid support framework for such a device. For example, three curved backpack frames can be lashed together to form a platform (Fig. 34-12). Ski poles or sturdy branches then can be fastened to the frames for use as carrying handles,

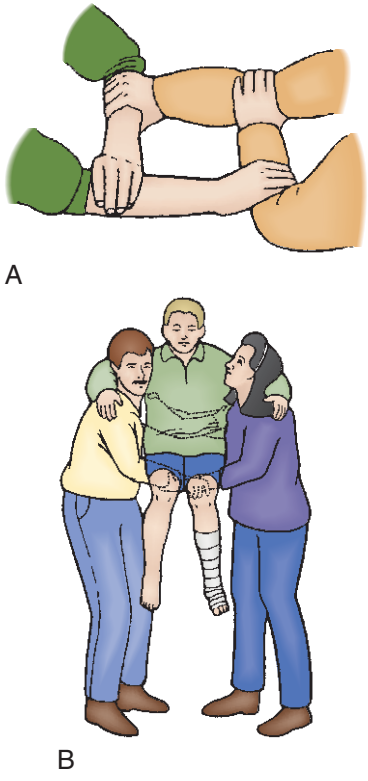


Figure 34-7. **A,** Four-handed seat used to carry a person. In this technique, the upper body is not supported. **B,** Alternative four-handed seat that helps support the victim's back. (From Auerbach PS: Medicine for the Outdoors: The Essential Guide to Emergency Medical Procedures and First Aid, 4th ed. New York, Lyons Press, 2003.)

and the platform can be padded with ground pads, sleeping bags, or a similar material.

Combining a rope litter with a rigid litter can provide more strength and versatility. The rescuer fashions this type of litter by first building a platform of poles or limbs, using a blanket as in a rigid litter, and placing the victim in a sleeping bag on the platform. The patient and platform are wrapped and secured with a length of rope. Because a mummy sleeping bag is used to encapsulate the victim, this device is sometimes called a *mummy litter.* Although this type of litter offers improved support, strength, and thermal protection, careful thought must be given to the physical and psychological effects such a restrictive enclosure may have on the victim (Fig. 34-13).

If long distances must be traveled or if pack animals are available, a litter may be constructed so that it can be dragged or slid along the ground like a sled. One such device is known as a *sledge* (Fig. 34-14). This litter is fashioned out of two forked tree limbs, with one side of each fork broken off. The limbs form a pair of sledlike runners that are lashed together with cross members to form a patient platform. The sledge offers a solid platform for victim support and stabilization. If sufficient effort is put into fashioning a smooth, curved, leading edge to the runners, a sledge can be dragged easily over smooth ground, mud, ice, or snow. Ropes also can be attached to the front of the platform for hauling and to the rear for use as a brake when traveling downhill.

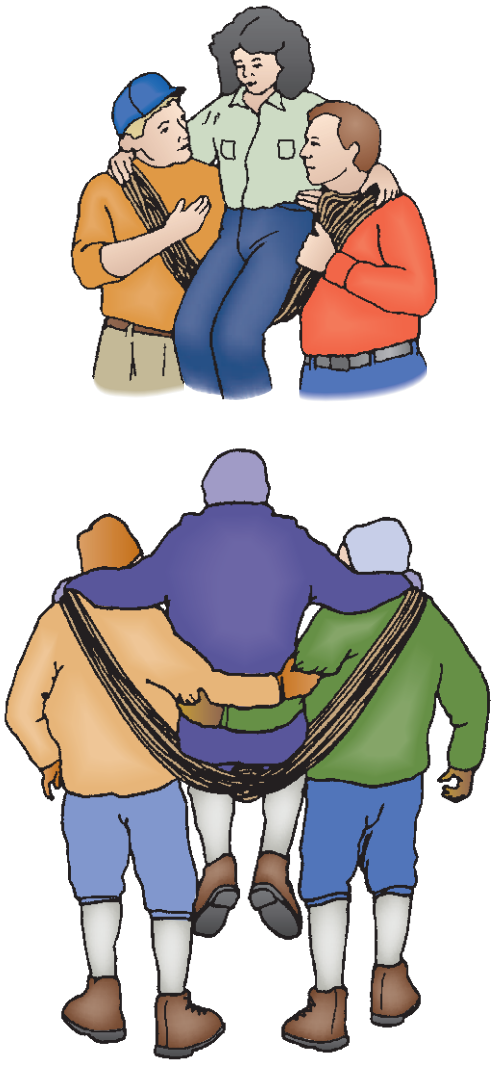


Figure 34-8. Two-rescuer split coil seat.

A *travois* is a similar device that is less like a sled and more like a travel trailer (without wheels). A travois is a V-shaped platform constructed out of sturdy limbs or poles that are lashed together with cross members or connected with rope or netting. The open end of the V is dragged along the ground, with the apex lashed to a pack animal or pulled by rescuers. Although the travois can be dragged over rough terrain, the less smooth the ground, the more padding and support necessary for comfort and stabilization. A long pole can be passed through the middle of the platform and used for lifting and stabilization by rescuers when rough terrain is encountered.

When victims are transported in improvised litters, especially over rough terrain, they should be kept in a comfortable position, with injured limbs elevated to limit pressure and movement. To splint the chest wall and allow full expansion of the unaffected lung, victims with chest injuries generally should be

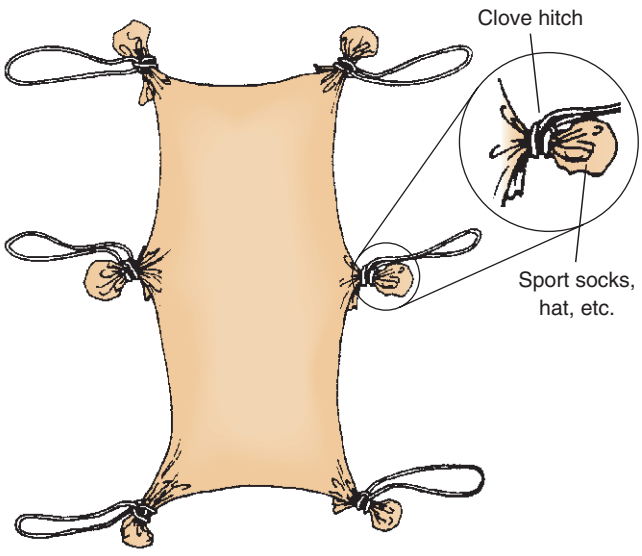


Figure 34-9. Improvised handled soft stretcher.

Figure 34-11. Improvising a stretcher from two rigid poles and a blanket or tarp.

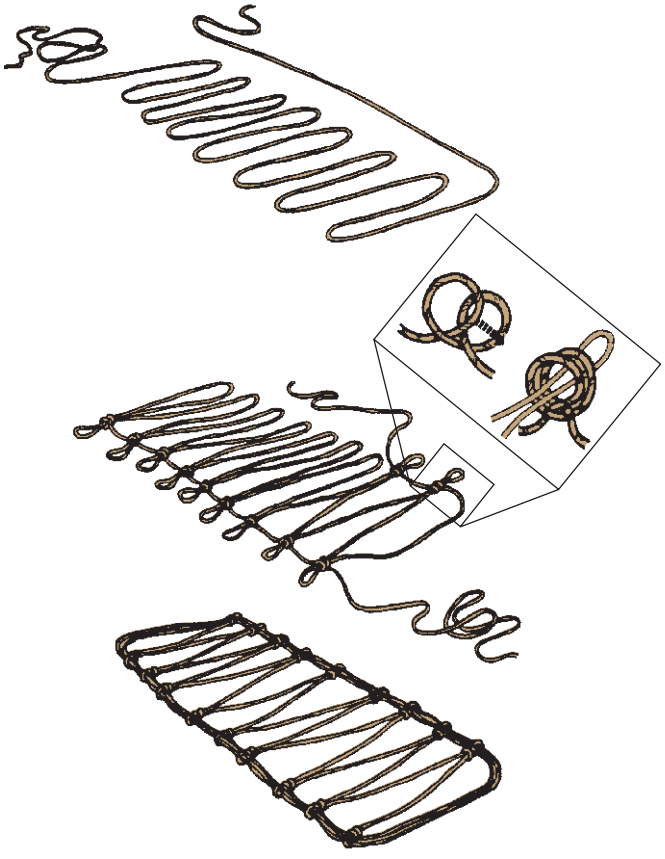


Figure 34-10. Tying an improvised rope (nonrigid) stretcher.

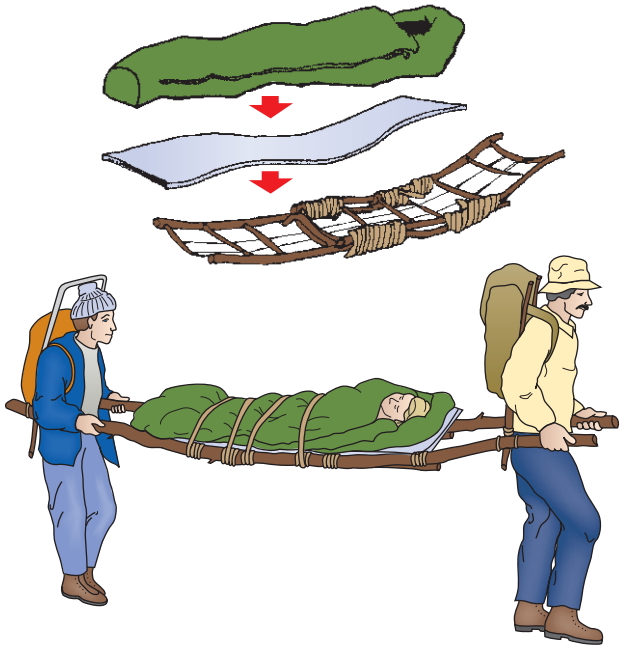


Figure 34-12. Pack frame litter. Note that the sapling poles on the litter can be attached to the rescuer's pack frames to help support the victim's weight.

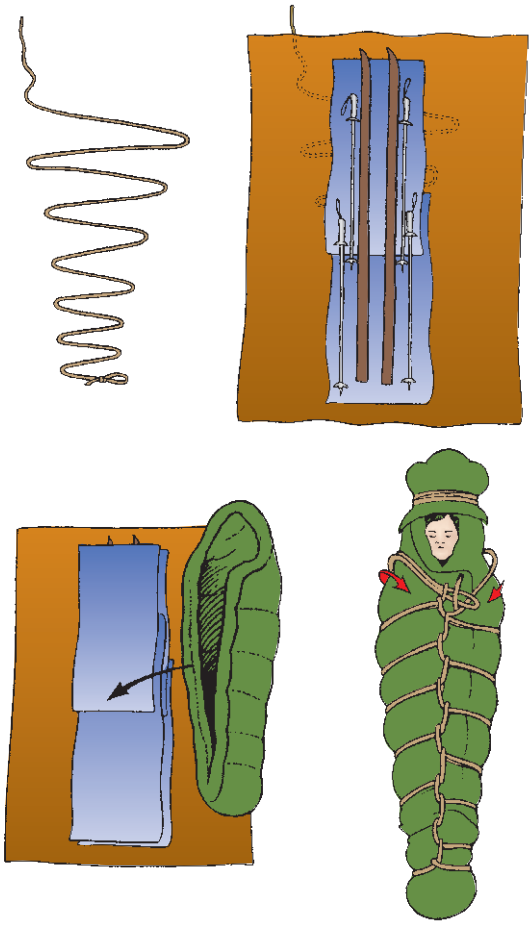


Figure 34-13. Mummy litter. (From Auerbach PS: Medicine for the Outdoors: The Essential Guide to Emergency Medical Procedures and First Aid, 4th ed. New York, Lyons Press, 2003.)

positioned so that they are lying on the injured side during transport. For a person with a head injury, the head should be elevated slightly, and for persons with dyspnea, pulmonary edema, or myocardial infarction, the upper body should be elevated. Conversely, when the victim is in shock, the legs should be elevated and the knees slightly flexed. Whenever possible, unconscious patients with unprotected airways should be positioned so that they are lying on their side during transport to prevent aspiration.[1]

RESCUE LITTERS AND STRETCHERS

The image most often associated with rescue immobilization and transportation devices in the United States is that of a traditional tubular steel and chicken wire–netted basket, which came to be known as the *Stokes basket*. Although this apparatus was and still is ubiquitous, many may recall the Thomas, Duff, Mariner, Brancard Piguillem, Perche Barnarde, Neil Robertson, MacInnes, and Bell stretchers for their evolutionary and robust designs. Today there are a variety of devices that meet the following two primary wilderness medical needs:

1. Immobilization and protection of a patient during transportation
2. Safe, comfortable, and stabilized transportation of a patient to definitive care

DESIRABLE CHARACTERISTICS OF A WILDERNESS STRETCHER

Peter Bell,[2] rescue equipment historian and developer of Bell stretchers, has described several specific characteristics of a high-quality, useful rescue stretcher. Bell claims that a good stretcher should:

1. Be as strong and robust as possible, with materials compatible with the rescue environment
2. Be as lightweight as possible

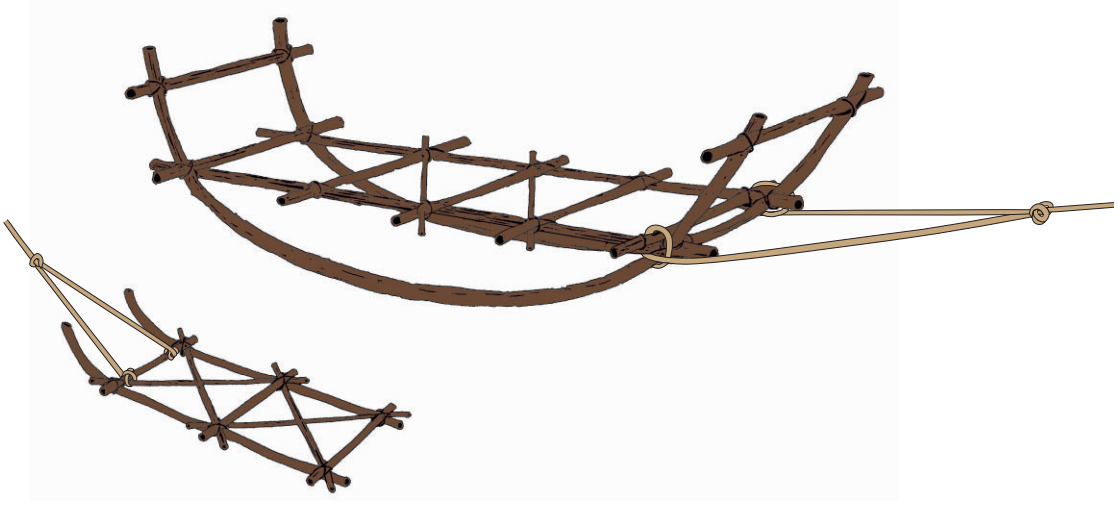


Figure 34-14. A "sledge."

3. Have smooth edges that will not snag
4. Be devoid of small spaces that will trap or pinch fingers (rescuers' and patients')
5. Be large enough to provide strength, security, and comfort for the largest of persons when the device is in any position (horizontal, vertical, on its side, upside down, etc.)
6. Prevent worsening of injuries during use
7. Provide security for the victim regardless of his or her condition (e.g., slippery, wet, muddy)
8. Be comforting to the conscious victim
9. Be easy to use in the dark and in temperature extremes (very hot or cold)
10. Protect the victim from the environment (heat, cold, brush, rocks, etc.)
11. Be reliable for many years after many uses in extreme conditions
12. Be easy to carry and use when carrying a heavy, large person
13. Be portable (can be carried in a car, boat, plane, helicopter, etc.)
14. Be impossible to use improperly
15. Be easy to clean and sterilize

STRETCHERS

In the interest of brevity and with some technical latitude, this discussion describes stretchers in four categories: basket-style, flat, wrap-around, and mountain rescue.

Basket-Style Stretchers

The basket-style stretcher derives its name from its shape. The sides curve upward to protect the victim's sides and to prevent the victim from rolling out. Most basket-style stretchers combine a steel frame (solid, tubular, or both) with a shell of either steel wire netting ("chicken wire") or plastic. Many include wooden slats in the bottom to provide additional protection and support.

Most likely, basket-style stretchers initially were adopted by wilderness rescue organizations because they met fundamental needs and were sufficiently robust to endure great abuse in severe terrain. The seminal basket-style stretcher, called the Stokes, first appeared in the late 1800s and likely got its name from the fact that it was designed to be used on naval and commercial ships to remove casualties from the "stokehold," a room in which the boilers were stoked (Fig. 34-15). However, at least one source makes an unsubstantiated claim that the device was invented in 1895 by Charles Stokes.[8] The significant influence of this device is reflected in the fact that the Stokes was the first commercially available basket-style stretcher in the United States.

The original Stokes design included a leg divider meant to separate and support each leg individually. Many came to consider this configuration counterproductive when the use of long (16 × 72-inch [0.40 × 1.83-m]) backboards and other spinal, full-body immobilization devices became widespread, especially for early treatment of trauma. Most current designs of the Stokes basket-style stretcher have eliminated the leg divider to allow full immobilization of a patient on a long backboard, which can be inserted into the basket. The Junkin Safety Appliance Company manufactures several Stokes basket-style stretchers, including models that break into two pieces and models with and without wooden slats or leg dividers, that meet the more robust military specifications (MIL-L-37957 and RR-L-1997) (Fig. 34-16).

Although the traditional materials and design (i.e., tubular and flat, welded steel with a steel chicken wire covering) are still in use today, basket-style stretchers are more often constructed from tubular stainless steel or aluminum because of the added corrosion resistance, increased strength, and reduced weight. Manufacturers, such as Junkin and Ferno, offer basket-style devices in full rectangular and tapered rectangular shapes. Both are also available in break-apart versions for easy carrying. Narrower versions (usually 19 inches [0.48 m] wide, instead of 24 inches [0.60 m]) are available for use in confined spaces or caves, although cave rescuers rarely prefer any type of chicken wire litter.

Because of the importance of portability in wilderness areas, the break-apart capability is an adaptation to nearly all styles and types of litters. Junkin manufactures a version of the Stokes stretcher completely coated with a plastic material called Plastisol, which provides nonsparking, nonconductive, and antistatic properties. Junkin suggests that this coating allows improved purchase (handgrip) on the litter and offers insulation from the temperature of the metal. A collateral benefit of the coating is that it extends the life and integrity of the steel chicken wire netting.

Figure 34-15. Traditional tapered Stokes litter with leg divider. (Courtesy of Junkin Safety Appliance, Inc.)

Figure 34-16. Break-apart Stokes litter with wooden slats. Many manufacturers also offer accessory straps or backpack devices that allow the rescuer to carry the litter halves on his or her back. (Courtesy of Junkin Safety Appliance, Inc.)

Figure 34-17. Lifesaving Systems Corporation's Model 404-F Medevac II stainless steel basket litter with optional flotation. (Courtesy of Lifesaving Systems Corporation.)

Figure 34-18. Ferno Model 71 Stretcher. (Courtesy of Ferno.)

Lifesaving Systems Corporation of Florida markets the Medevac II series of Stokes-style litters used widely by the U.S. Coast Guard. These devices are made from either a powder-coated stainless steel or titanium, which is 40% lighter (9 kg [20 lb] versus 14.5 kg [32 lb]). It also comes in a break-apart version (Medevac IIA) and is designed to include flotation for use in and around the marine environment (Fig. 34-17). In this device, the wood slats used on the original Stokes litter have been replaced with stainless steel reinforcements that run the full length of the litter; and smooth, plastic net mesh (with $^1/_2$-inch [13-mm] openings) replaces the chicken wire liner.

Taking advantage of substantial improvements in polymer research, some manufacturers began producing a stretcher shell composed of rigid plastic instead of steel mesh. Ferno's model 71 stretcher has an orange plastic shell wrapped around an aluminum frame and secured with aluminum rivets. Brass grommets in the plastic serve as attachment points for a lifting harness (Fig. 34-18). At half the weight of a traditional steel Stokes, this device offers protection from snags and obstacle penetration that cannot be provided by the wire netting of the Stokes. In addition, the plastic used is chemical resistant, and the molded underside runners make it slide smoothly over flat

Figure 34-19. International Stretcher Systems' Yellow Jacket Basket Litter with flotation added. (Courtesy of International Stretcher Systems.)

ground, ice, and snow. Ferno offers a version with tow handles and a chain brake that is designed specifically for ski patrol applications so that a packaged victim can be "skied" down a slope or pulled along a snow-covered trail. The orange Ferno litter has a load limit of 270 kg (600 lb), but its usefulness in a vertical raise configuration depends on the integrity of the aluminum frame and the plastic shell; if one is compromised, both may fail. Because of this limitation and because of the lightweight materials used, bending or twisting the device should be avoided.

International Stretcher Systems also builds a basket-style stretcher called the *Yellow Jacket*. It has a lightweight aluminum frame but uses a different approach for combining the frame with the shell. Its high-density polyethylene shell is similar to the shell in the Ferno model 71, but it is placed outside a full aluminum skeleton to facilitate sliding over the ground. Inside the stretcher, a spring-suspended victim "bed" that doubles as the victim retention system has been added to protect the victim from the internal frame members. This bed minimizes transport shock and features built-in shoulder straps, pelvic padding, a head and chin immobilizing harness, foot and ankle straps, and a large "double-security" Velcro body restraint flap that wraps around the victim. The stretcher weighs 40 pounds (18.2 kg) and has an engineered load rating of 2500 pounds (1136 kg) (Fig. 34-19).

The Junkin model SAF-200-B includes parallel stainless steel top rails. The top tube is larger to allow comfortable hand-gripping and to provide an attachment for lifting bridles, and the smaller solid steel lower rail allows attachment of patient retention straps. The twin rail configuration keeps patient straps and lifting systems from interfering with each other and helps protect the attached materials from abrasion during use. Unlike the International Stretcher Systems device, the stainless steel frame in the Junkin stretcher wraps around the exterior of the basket, which is lined with a smooth-surfaced, permanently padded plastic shell. This design offers comfort for the victim but makes it difficult to slide on the ground because of the external, exposed steel frame members. The unit is heavy (weighing 14.5 kg, or 32 lb) but breaks apart for packing and marries well with a litter wheel to allow easier handling.

Cascade Toboggan manufacturers the Model 200 Advanced Series of rescue litters (Fig. 34-20). These robust devices—

Figure 34-20. Cascade Toboggan's Model 200 Advanced Series Rescue Litter with optional snow handles. (Courtesy of Cascade Toboggan Rescue Equipment Company.)

Figure 34-21. Cascade Toboggan's Professional Series Litter Skin that can be attached to many types of rescue litters for easy sliding across any rough or abusive surface. (Courtesy of Cascade Toboggan Rescue Equipment Company.)

common sights at many recreational ski areas—have a $^{5}/_{8}$-inch (16-mm) stainless steel top rail that attaches to a composite basket. It is available in a break-apart version and integrates four 1-inch (25-mm) patient restraint straps into the basket. The device is compatible with both the CMC rescue Litter Shield and Litter Wheel, and weighs less than 18 pounds (8.1 kg) for the one piece version. The two-piece version is just a bit heavier. Cascade Toboggan also developed and manufactures the Litter Skin, an abrasion-resistant shell that can be quickly attached to many types of rescue litters to make sliding across any surface easier (Fig. 34-21).

Bell Rescue Stretchers offers the Series 2 Ludlow stretcher, which is simply their strongest flat stretcher with deep basket sides added. This strong, stainless steel–framed design has fold-down sides, which simplify access with a backboard. The sides can be completely removed to revert the device back to a flat stretcher. Other available variations on this theme from Bell include versions with shorter sides (the Otterburn), an open foot end (the Newark), and a steel plate welded into the bottom of the stretcher to help protect the victim (the Manchester). The Manchester weighs nearly 22.5 kg (50 lb) by itself. The manufacturer claims that the Series 2 models can all accommodate a long spine board and have been "proof tested" to between 500 and 720 kg (1102 and 1587 lb). The "bed" of Bell's Series 2 stretchers is made of 14 polypropylene web straps that cross between the steel frame members. This webbing also passes through two movable stainless steel spinal supports (flat steel frame members that run the length of the caudal two thirds of the litter). Four patient retention straps attached to the outermost rails, a patient shoulder strap, and integral lifting rings are supplied with the device. A slightly smaller, lighter, and less robust version, called the Bell Emergency Stretcher (discussed in the next section), also is available.

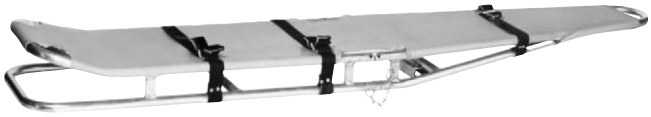


Figure 34-22. The Junkin Air Rescue Stretcher (SAF-350) was designed for use in Bell Jet Ranger helicopters. This lightweight, flat stretcher folds for easy storage but is not designed for long carries. (Courtesy of Junkin Safety Appliance, Inc.)

Flat Stretchers

Flat stretchers are generally flat and have very short or no sides. Restraint straps or built-in tie downs serve as the physical means by which the victim is secured in the litter. Although the specific characteristics of these types of stretchers vary greatly (from extremely lightweight to high strength), generally they are used when specific benefit is derived from their low-profile shape. Although this style of stretcher has been modified to allow dragging or sliding (e.g., mountain rescue stretchers), the primary purpose of the flat design is to reduce weight and profile for carrying or for specific applications, such as loading into an aircraft.

Although a simple, two-pole canvas litter is fine for use over short distances, uncomplicated terrain, or the battlefield (where haste is paramount), the lack of patient protection and immobilization capability limits its usefulness outside of the hospital or battlefield setting. A more modern version of this simple device is made of aluminum, folds for easy storage, and doubles as a long backboard. Another version is hinged at one end so that it can be spread along its long axis and slid under a patient on the ground with little movement or rolling. This "scoop" stretcher was commonly used by emergency medical service providers but has been supplanted by the use of long backboards. In the final analysis, both the military and aluminum iterations of the flat stretcher are intended for carrying a person short distances in an environment with few terrain obstacles or where complete security and immobilization are not required.

Several successful varieties of flat stretchers have evolved over the years, including the Brancard Piguillem, the Junkin Air Rescue Stretcher (Fig. 34-22), and a few of the Bell stretchers that are categorized as flat for the purposes of this discussion.

The Brancard Piguillem (Brancard is French for "stretcher" and Peguillem is a proper name) is a flat, old-style stretcher consisting of a canvas patient bed lashed to a steel and aluminum frame (weighing 14 kg, or 30 lb), which folds in half for easy carrying by one person. The design, which has evolved over the years in the European and British mountain rescue communities, includes a patient bed with a permanently attached, integral casualty bag lashed to the frame to protect and secure the victim. Full-length runners raise the stretcher a few inches above obstacles and allow for easy barehanded gripping. This folding, portable design with integral patient protection made the Brancard Piguillem popular with mountain rescuers and served as the impetus for the evolution of several of the current styles of mountain rescue stretchers.

Figure 34-23. Life Support International's Rescue Sled (manufactured by Switlik) is a unique, durable, and inflatable device that provides 225 lb (101 kg) of buoyancy and weighs under 20 lb (9 kg). Shown is the Rescue Sled with integral flotation devices inflated (one tube each side). (Courtesy of Life Support International, Inc.)

An interesting variation on the flat theme is the Rescue Sled Inflatable Rescue Litter manufactured by Switlik and distributed by Life Support International (Fig. 34-23). The Rescue Sled is an inflatable, flat litter made of coated ballistic nylon that offers 225 pounds (101 kg) of buoyancy and weighs less than 20 pounds (9 kg). The device can be lifted, paddled, towed, or slid, both in and out of the water. It may be inflated either manually or by a small carbon dioxide canister that comes with it. Uninflated, it packs easily into a medium-sized backpack or duffle bag. The integrated head restraints and body straps allow for securing a patient, and towing and lifting attachments simplify the process of moving the device when loaded. Although this device may not be best for all situations, its unique buoyancy and design characteristics make it attractive for many ice- and water-related rescues.

The Kendall Stretcher, from Bell Rescue Stretchers, has the same features as their Ludlow Stretcher without the basket sides (see the previous section). The Kendall has a stainless steel frame, integral lifting rings, a bed made of polypropylene web straps that cross between the frame members, color-coded patient retention (38-mm web) straps, and a detachable foot loop. The Kendall Stretcher can be used either side up; there is no top or bottom. A slightly less robust version of Bell's flat stretcher is their Basic Emergency Stretcher. This device is smaller and lighter (weighing 5.2 kg, or 11 lb, 8 oz) and works well in commercial and industrial settings where severe terrain is rarely encountered.

Although the manufacturer considers this stretcher to be a mountain rescue device, the MacInnes Mark 7 Rescue Stretcher is the latest in a long line of strong, user friendly stretchers from Hamish MacInnes, the founder of MacInnes Rescue Stretchers and member of the Glencoe (Scotland) Mountain Rescue Team. The designer's aim was to develop a compact, versatile stretcher made from light, "space age" materials. The Mark 7 is 22 pounds (11 kg) complete, less than 5 cm (2 in) high (unfolded), constructed of abrasion-resistant titanium and composite materials, and can be folded for easy packing into isolated areas. It also includes a folding foot rest and head protector that double as handles.

Mountain Rescue Stretchers

Mountain rescue stretchers are essentially stronger, more robust flat litters with runners or skis attached to the bottom for easy movement over rugged terrain. Over the years, engineers and litter designers with mountaineering and rescue backgrounds have adapted rescue litter designs to meet specific practical needs of their environment. The result is the strongest and most robust platform available for patient treatment and transportation, but these benefits come at the cost of weight and size.

The Thomas Stretcher is an early and beautifully simple example of the mountain rescue device. Invented in the United Kingdom in the 1930s by Eustace Thomas (no relation to Hugh Owen Thomas, who invented the Thomas "half-ring" splint), it consisted of wood (ash) runners, an aluminum frame, a canvas bed, and six or seven patient straps attached to the rails. It also had locking, tubular, retractable handles that stowed in the tubular rails.[5] The Thomas Stretcher is still manufactured, with some modifications, by Bell Rescue Stretchers in the United Kingdom.

In the late 1950s, Donald Duff, a pioneer of mountain rescue in Scotland, designed a stretcher with a steel tubular frame and no handles. Channeled steel runners extended along two thirds of the caudal end of the stretcher. It weighed about 13.5 kg (30 lb) and could be fitted with a wheel and undercarriage for easier movement over rocky terrain. Its profile was low and sleek, and the runners could be detached and the remainder folded in two for backpacking.[4]

Although they have almost completely been replaced by more modern designs, two basket-style mountain rescue stretchers that evolved in Britain and Europe over the first half of this century deserve mention. The Perche Barnarde (Perche is French for "perch" or "pole," and Barnarde is a proper name) consists of a 2-m (7-foot) square section of steel tube from which a canvas casualty bag is suspended. The tube breaks into three pieces for easy carrying. The bag is attached to the tube at each end. Where the patient's shoulders would fall, a spreader bar is placed to keep the bag open. From each end of the steel tube extend two removable, bicycle-type handlebars fitted with pads so that the handlebars can rest on rescuers' shoulders. Although this device has been used successfully in many difficult mountain evacuations over the years, its limitations regarding patient comfort, protection, and immobilization are obvious.

The Mariner consists of a canvas bed attached to a steel sled-like frame. The bed functionally resembles a reclining chair in that the patient sits flexed at the hips and waist with the lower legs supported by a canvas platform. The frame is rounded from end to end and includes two steel runners to deflect obstacles. Two adjustable handles extend from each end of the frame for carrying. Today, the Mariner is used by several U.K. mountain rescue teams and is noted to have contributed significantly to the evolution of the mountain rescue stretcher.

The current British standard for mountain rescue stretchers includes two devices that are incredibly strong, durable, and, unfortunately, heavy. The Mark III Bell Rescue Stretcher (which weighs 24 kg, or 53 lb) and the model 6 MacInnes Rescue Stretcher (which weighs 22 kg, or 48 lb) (Fig. 34-24) are intended to survive years of use in extreme mountainous environments. Both have break-apart versions for easy packing into isolated areas, and both incorporate lifting rings, skids, and head guards.

Flexible, Wrap-around Stretchers

A focus on improving stretchers for particular environments or situations has led to major developments in a number of litter design areas. It is difficult for a single device to excel in every

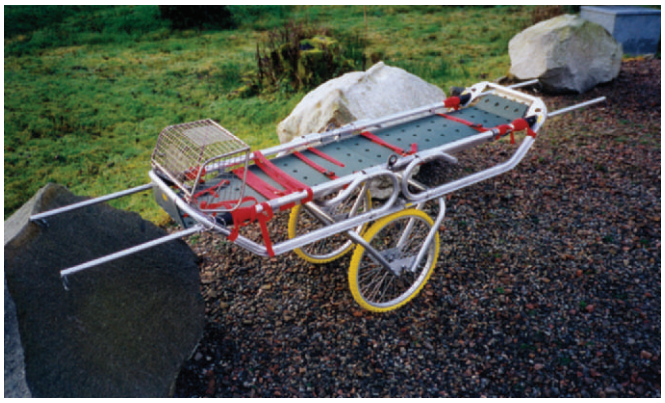


Figure 34-24. MacInnes MK 6 Mountain Rescue Stretcher. Note the extending handles, folding head guard, and optional twin (solid) tires. (Courtesy of MacInnes Rescue Stretchers.)

Figure 34-25. Quick extrication, ultralight, nylon litters; six handle *(left)* and four handle *(right)*. Designed for rapid movement of casualty by dragging (one person) or lifting and carrying. Note torso straps to secure patient. (Courtesy of London Bridge Trading Company, Ltd.)

situation because enhancing one capability or characteristic can be detrimental to another. For instance, it can be difficult to achieve a substantial increase in strength while decreasing overall weight. However, new innovative stretcher designs and materials allow structural flexibility to meet specific needs.

Flexible, wrap-around stretchers can be folded, rolled, or otherwise compacted for storage and "wrap around" in that they contain the victim to provide protection, immobilization, and often sufficient support for vertical lifting. The Neil Robertson Stretcher was the impetus for this entire category of device. Adapted from a Japanese design by John Neil Robertson and first produced between 1906 and 1912, this wooden and canvas stretcher was originally made of bamboo and sewn by hand. The "Neil Rob" supplanted the Mansfield military stretcher in the United Kingdom and was first given the name "Hammock for Hoisting Wounded Men from Stokeholds and for Use in Ships whose Hoists are 2 feet, 6 inches in Diameter."[3]

The Neil Rob consists of wooden slats that are covered with semirigid canvas and sewn the length of the stretcher. These slats wrap around the patient in mummy fashion, with arms in or out, providing protection without bulk. Full-body immobilization, protection, and a small cross section combine to produce a device well suited for use in small spaces or for situations in which the victim must be moved through a small opening.[9]

The most basic type of flexible, wrap-around stretcher has long been used by the military under combat conditions to quickly move casualties. London Bridge Trading Company manufactures several versions of a Quick Extrication Litter that integrates web handles and torso straps on an ultralight, coated nylon panel (Fig. 34-25). While in use by one person, it may look more like a drag sheet than a litter. But, by passing litter poles through the web handles, the device, which weighs less than 2 pounds (0.9 kg) and can almost be packed in a pocket, works well when spinal immobilization is unnecessary or can be provided separately.

The Ferno LifeSaver Stretcher is a modern version of the traditional Neil Robertson Stretcher. It is 153 cm (60 inches) long and uses full-length 5-mm (0.2-in) ribs interwoven by 1000 denier nylon fabric to provide full-body splinting in a very narrow package. Five built-in restraints with forged steel buckles are integrated into the device, and stainless steel rings are used at the foot and head for vertical attachment points.

Figure 34-26. Ferno Traverse Rescue Stretcher configured for horizontal lift. (Courtesy of Ferno.)

For situations requiring full-body protection for the victim without complex restraint systems, the Reeves Sleeve and Ferno Traverse Rescue Stretcher almost totally encapsulate the patient. When used in conjunction with a cervical collar and spinal immobilization, these devices provide environmental and mechanical protection while allowing the victim to be carried through narrow passages.

The Ferno Traverse Rescue Stretcher (model UT2000) uses heavyweight Cordura nylon to cover a high-density polyethylene sheet that gives the device rigidity when wrapped around the patient (Fig. 34-26). This stretcher has an integrated adjustable, full-body harness to secure the patient in the device,

Figure 34-27. Reeves Sleeve in use. (Courtesy of Reeves Manufacturing, Inc.)

padded straps in the shoulder and groin areas to make for a comfortable fit, and a foot strap that stops the patient from sliding down during vertical lifts.

The Reeves Sleeve is a compact immobilizing stretcher suitable for hand-carry situations or vertical environments, but is not intended for vertical lifting (Fig. 34-27). The device uses a vinyl laminated nylon shell, slides over a full backboard, and depends on the backboard or short spinal immobilizer to provide rigidity. The company now makes a Reeves Sleeve Model 122 that was specifically designed to be lifted both vertically and horizontally.

Skedco's Sked stretcher provides wrap-around protection similar to the Neil Robinson, but a combination of shape and material, rather than integral slats, provides longitudinal rigidity (Fig. 34-28). Light and compact when stored in its packable case, the flexible, low-density polyethylene plastic litter wraps around the victim to form a rigid sleeve that is superbly compact for maneuvering in tight quarters. A half-length version is available for moving persons from areas that are too confined for a full-length device when flexing the victim at the hips might facilitate extrication. The hard and smooth plastic material can be easily dragged over a variety of surfaces and offers substantial protection from penetrating obstacles. Though the Sked stretcher provides spinal protection, the manufacturer recommends using an Oregon Spine Splint (Skedco) or similar device when cervical spine immobilization is necessary. External lift slings are included with the Sked to allow vertical or horizontal lifting, and flotation is available for use in a marine environment.

Figure 34-28. Sked Rescue Stretcher system. (Courtesy of Skedco.)

Figure 34-29. The Medical Devices International Immobile-Vac Full Body Mattress is a full-body vacuum splint that can be inserted into other litters. (Courtesy of Medical Devices International.)

Medical Devices International (MDI) makes the Immobile-Vac Full Body Mattress, which is a full-body vacuum splint on which the victim is carried (Fig. 34-29). The vinyl-coated, fabric patient bed contains loose polystyrene (Styrofoam) beads similar to those in a beanbag chair. Once the victim is positioned on the mattress, a small hand pump is used to expel air from within it. This process creates a rigid, full-body splint that conforms to the victim's shape. This "cocoon" immobilizes the spinal column and extremities while providing a comfortable platform. The mattress has integrated web carrying straps that can be used to carry the patient directly, or the mattress can be inserted into a basket-style stretcher for added versatility and strength. The use of a basket stretcher will be necessary for high-angle rescue because the MDI device is not designed for vertical rescue by itself.

Figure 34-30. CMC Rescue Mule II Litter Wheel with handles (manufactured by Traverse Rescue). This versatile device allows the wheel to be removed and stored inside the frame and is available without the handles. (Courtesy of CMC Rescue, Inc.)

Figure 34-31. The CMC Rescue Litter Shield protects the victim from falling debris while allowing access to the head and face. It can also be inverted and placed in the litter to save space during storage. (Courtesy of CMC Rescue, Inc.)

TRANSPORTATION HARDWARE ACCESSORIES

A number of wheeled devices can be attached to most basket-style stretchers. These devices take the carrying burden off rescuers in nontechnical evacuations and reduce the load on low-angle haul systems. One example is the CMC Rescue Mule II Litter Wheel (Fig. 34-30), which incorporates a large, under-inflated all-terrain vehicle tire into a lightweight frame that clamps to the underside of the litter. This single wheel is positioned under the center of gravity, and the rescuers walk alongside, or at each end of, the litter to steady and guide it, with the wheel carrying most of the load. When they encounter large obstructions, such as logs or trenches, rescuers simply lift the litter and continue rolling. One advantage of this device is that it reduces the number of rescuers required to move a litter safely over a long distance. When using this device, only two rescuers (one at each end) are required to control the litter, but more may be used as necessary.

To meet a similar need, International Stretcher Systems employs a collapsible steel frame trailer (called the *Anchor Man Trailer*) with two all-terrain vehicle (ATV) wheels. With no axle between the wheels, the tires can be set to the outside of the frame for greater side-to-side stability or to the inside and under the frame to negotiate narrow trails or doorways. The drawbar end of the trailer is set up to receive either the T-bar handle, for towing by rescuers on foot, or a hitch adapter, for towing behind motorized vehicles. Skis can also be substituted for the wheels.

Junkin markets several useful stretcher accessories, but at least one is notable. Junkin's Comfo-Pad is designed to fit in all Junkin stretchers and is simply a pad on which the patient lies in the litter. It is made of $^1/_2$-inch (13-mm) foam rubber with a durable, orange nylon cover and is held in place with hook-and-loop–type fasteners. Because comfort is always an issue, this simple accessory can be quite helpful.

MacInnes and Bell integrate steel wire head protectors in their mountain rescue stretcher designs. This feature is important where falling rock is a hazard. CMC Rescue markets a similar aftermarket device made of clear polycarbonate under the trademark CMC Rescue Litter Shield (Fig. 34-31). It protects the victim's face from falling debris; allows for easy, rapid airway access; and can be moved out of the way because it hinges on the end of the litter. The shield stores compactly in the litter when not in use.

Flotation systems are available for some litters. Ferno, International Stretcher Systems, and Skedco each offer this option to make their devices safer and more versatile in swift or open water rescue situations.

Most litter manufacturers offer specific packaging or methods for carrying their devices into isolated areas. For instance, London Bridge Trading Company's Quick Extrication Litter, the Sked, and Ferno's Traverse Rescue Stretcher can all be compacted and carried in by a single rescuer. Other manufacturers sell special backpacks or integral carrying harnesses for carrying half of their break-apart litters because of the greater weight.

Carrying a Loaded Litter

An evacuation is defined as high angle or vertical when the weight of the stretcher and tenders (stretcher attendants) is primarily supported by a rope and the angle of the rope is 60 degrees or greater.[7] This type of situation is often encountered when a rescue is performed on a cliff or overhang or over the side of a structure and usually requires only one or two tenders. In high-angle rescues, most often the stretcher is used in the horizontal position to allow only one tender and to keep the victim supine and comfortable. However, when the packaged victim and stretcher must be moved through a narrow passage or when falling rock is a danger, the stretcher may be positioned vertically.

In a scree or low-angle evacuation, the slope is not as steep (less than 60 degrees), the tenders support more of the weight of the stretcher, and a rope system is still needed to help move the load. In this type of rescue, more tenders (usually four to six) are required, and the rope is attached to the head of the stretcher. The head of the litter is kept uphill during a low-angle rescue.

In a nontechnical evacuation, tenders completely support the weight of the stretcher during a carry out. Generally, the terrain dictates the type of evacuation. If the stretcher can be carried without the support of a rope, it is a nontechnical evacuation. If rope is needed to support the load or to move the stretcher, it is either a low- or high-angle evacuation, depending on the angle of the slope.

Carrying a litter in the wilderness is difficult and requires many resources. It takes at least six rescuers to carry a person in a litter a short distance (0.4 km, or $^1/_4$ mile, or less) over relatively flat terrain. With six rescuers, four can carry the litter

while the other two clear the area in the direction of travel and assist in any difficult spots. However, depending on the terrain and the weight of the victim, all six rescuers may be needed to safely carry the litter any distance. If the travel distance is longer, many more rescuers are required (Fig. 34-32).

PATIENT PACKAGING

Patients (victims) on stretchers must be secured, or "packaged," before transport. Packaging consists of stabilization, immobilization, and preparation of a victim for transport. Physically strapping a person into a litter is relatively easy, but making it comfortable and effective in terms of splinting can be a challenge. The needs of a person secured and transported in a litter are great and should not be overlooked or underestimated. The rescuer's goals are as follows:

1. Package the person to avoid causing additional injury.
2. Ensure the victim's comfort and warmth.
3. Immobilize the victim's entire body in such a way as to allow continued assessment during transport.
4. Package the victim neatly so that the litter can be moved easily and safely.
5. Ensure that the victim is safe during transport by securing him or her within the litter and belaying the litter as necessary.

Generally, proper patient packaging must provide for physical protection and psychological comfort. Once packaged in a carrying device, a person feels virtually helpless; thus, transport preparation must focus on alleviating anxiety and providing rock-solid security. With this in mind, rescuers must provide for the victim's ongoing safety, protection, comfort, medical stabilization, and psychological support.[6]

Splinting and spinal immobilization are usually achieved by using a full or short backboard. The victim is secured to the board, and then the victim (on the board) is placed into the litter. When the immobilized patient is finally placed into the litter, adequate padding (e.g., blankets, towels, bulky clothing, sleeping bags) placed under and around him or her contributes to comfort and stability. During transport, victims like to have something in their hands to grasp, to have pressure applied to the bottom of their feet by a footplate or webbing, and to be able to see what is happening around them.[6]

Because persons are so vulnerable to falling debris when packaged in a litter, especially in a horizontal high-angle configuration, a cover of some type should always be used to protect the victim. A blanket or tarpaulin works well as a cover to protect most of the body, but a helmet (in the absence of cervical injury) and face shield or goggles are also options to protect the head and face from projectiles. Alternatively, a commercially available litter shield can be used and allows easy access to the airway, head, and neck (see Fig. 34-31). Because the head and neck usually require immobilization, the technique and equipment used to protect them should allow this. Remember also that the conscious victim desires an unobstructed view of his or her surroundings.

Carrying a person in the wilderness often requires that the litter be tilted, angled, placed on end, or even inverted. In all these situations, the victim must remain effectively immobilized and securely attached to the litter, the immobilizing device within the litter, and any supporting rope be easily accessible and securely attached. Poor attachment can cause patient shift-

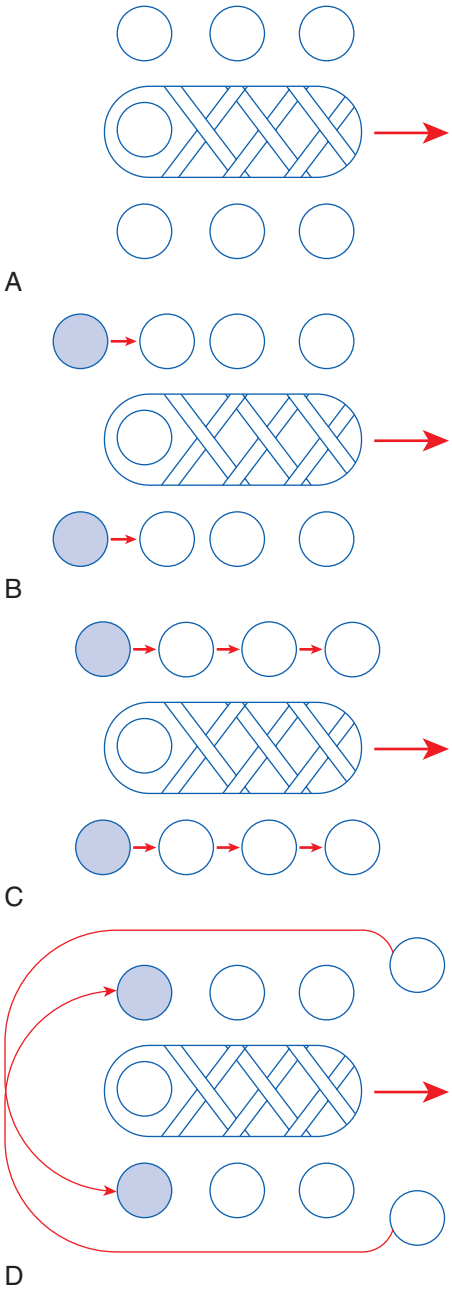


Figure 34-32. Litter-carrying sequence. **A,** Six rescuers are usually required to carry a litter, but rescuers may need relief over long distances (greater than 0.4 km, or $^1/_4$ mile). **B,** Relief rescuers can rotate into position while the litter is in motion by approaching from the rear. **C,** As relief rescuers move forward, others progressively move forward. **D,** Eventually the rescuers who are furthest forward can release the litter (peel out) and move to the rear. Rescuers in the rear can rotate sides so that they alternate carrying arms. Carrying straps (webbing) also can be used to distribute the load over the rescuers' shoulders. In most cases, the litter is carried feet first, with a medical attendant at the head monitoring airway, breathing, level of consciousness, and so on.

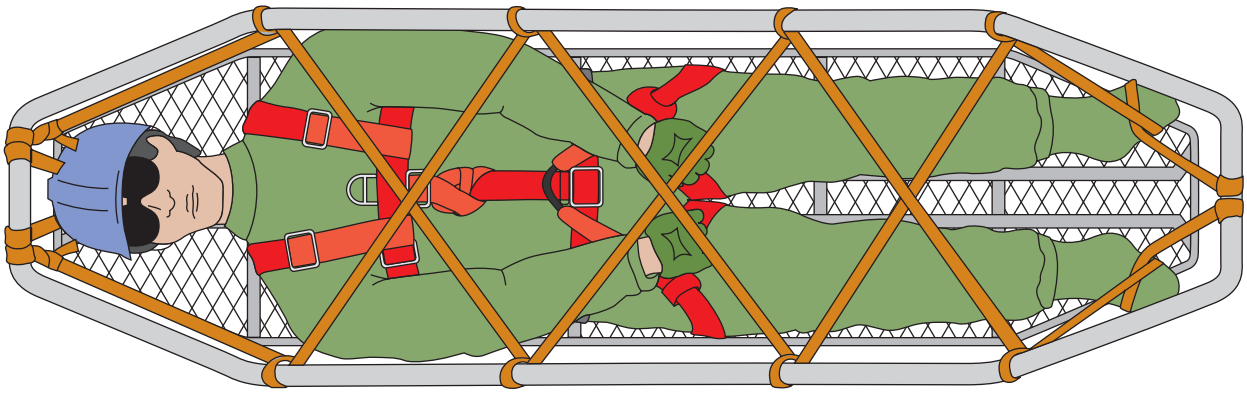


Figure 34-33. One 10-m (30-foot) web or rope can be used to secure a person into a litter.

ing, exacerbation of injuries, or even complete failure of the rescue system.

Manufacturers have taken several approaches to securing a person within the litter. Most integrate a retention or harness system directly into the litter. However, a few require external straps to secure the victim to the device. Many users suggest that an independent harness be attached directly to the victim to provide a secondary attachment point in case any link in the attachment chain fails.

When a harness is not available, tubular webbing, strips of sturdy material, or even rope can be used to secure the victim. One approach uses tubular webbing slings in a figure-8 at the pelvis and shoulders to prevent the victim from sliding lengthwise in the litter.

A 10-m (30-foot) piece of 5-cm (2-inch) webbing or rescue rope can be used to achieve the same goal (Fig. 34-33). The rope or web is laced back and forth between the rails of the litter in a diamond pattern until the victim is entirely covered and secure. Such a technique also easily incorporates a protective cover and support of the victim's feet.

Regardless of the techniques and equipment used, frequently checking vital signs (i.e., distal pulse and capillary refill) during transport can help ensure that strapping does not obstruct circulation.

The references for this chapter can be found on the accompanying DVD-ROM.

35 Aeromedical Transport

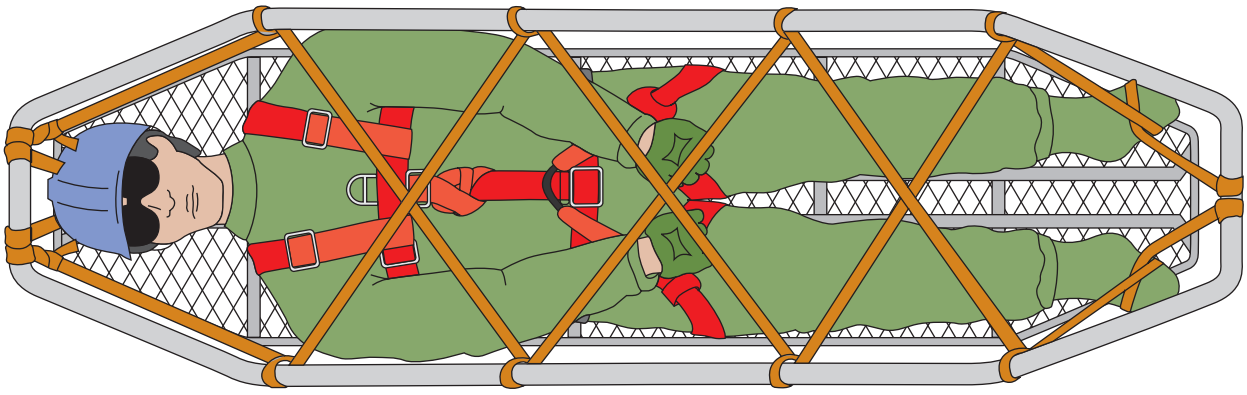
Robert C. Allen

Rapid provision of appropriate definitive care to acutely ill and injured patients is a major goal of all emergency medical services (EMS) systems. The ability to rapidly transport and initiate treatment of severely ill or traumatized patients is important in decreasing morbidity and mortality. This is particularly germane to wilderness and environmental emergencies, for which medical resources are scarce, transport times to definitive care facilities are often prolonged, and terrain and weather conditions are inherently difficult. Aeromedical transport crews can deliver emergency medical care at the scene, and the time to definitive care can be greatly decreased. This maximizes the patient's chance for a successful recovery.

AEROMEDICAL EVOLUTION

Rapid evacuation of trauma victims from an injury scene to the location of definitive care is a modern concept with roots in antiquity. The New Testament documented an early instance of prehospital care and transport: "A certain Samaritan . . . went to him and bound up his wounds, pouring oil and wine, and set him on his own beast and brought him to an inn, and took care of him."[91]

The greatest impetuses to the advancement of emergency care and transportation have been epidemics and wars.[69] Before the



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classical Greco-Roman period, injured soldiers were often left on the battlefield to die. Later, Homer described the use of chariots to evacuate fallen warriors during the Trojan War.[52] Napoleon's forces devised horse-drawn carriages, or ambulance volantes, for the same purpose.[59] North American Indians devised the travois, a litter that could be pulled by a person or animal to transport ill or injured persons.[67] The U.S. Army began a similar practice during the Seminole War of 1835–1842 and used it again in the Civil War. Major Jonathan Letterman established the process of rapidly clearing wounded soldiers to a point behind the battle line where they could be further triaged to an expectant area for persons with mortal wounds, a local treatment area for the "walking wounded," or a hospital if definitive care was feasible. The central concept was efficient access to surgery for victims of trauma.

These developments were soon followed by invention of flying machines. In France, Richet had prophesied the potentials of air transport in 1869.[69] This was before the first balloon airlift. The prophesy was validated the following year during the Franco-Prussian War when the first documented aeromedical evacuations took place. During the Prussian siege of Paris, 160 wounded soldiers were evacuated and transported by hot air balloon over enemy lines.[93]

In the United States, air evacuation took place soon after the Wright brothers flew in 1903.[42] Grossman and Rhoades presented their idea of air transport of patients to the War Department in 1910, but the government refused to fund them. It was not until World War I that the U.S. military began to utilize aircraft to carry injured soldiers, and this occurred only rarely. However, the French transported patients as early as 1912 aboard Dorland ARII fighters converted to carry litters, despite the government's objection to the concept of aeromedical transport: "Are there not enough dead in France today without killing the wounded in airplanes?"[42]

The United States began utilizing its first dedicated air ambulances in 1920, using the deHavilland DH-4A, followed by the Cox-Klemin XA-I. World War II saw widespread application of fixed-wing aircraft for evacuation. More than 1.4 million patients were transported from front-line hospitals to tertiary care facilities, with only 46 deaths en route.[99] During this time, the concept of medical care during transport was implemented. In November 1942, the War Department began to train flight surgeons, flight nurses, and enlisted medical personnel for aeromedical transport.[42] Also during 1942, Igor Sikorsky produced a rotor-wing aircraft, called a "helicopter," which the army configured with external litters. It was used in an air evacuation for the first time in 1944 in Burma.[39]

Helicopters did not enjoy widespread use until more reliable and powerful machines became available. The Sikorsky S-51 and later the Bell 47-B were deployed over the rugged terrain and uncertain roads of Korea with great success to provide wide-scale evacuation of wounded soldiers to Mobile Army Surgical Hospital (MASH) units. Although only 11 dedicated "Medevac" helicopters were used, more than 17,700 casualties were evacuated. For the first time, injury victims could travel directly from the point of injury to definitive surgical care.

This set the stage for Army helicopter evacuation ("Dust Off") operations in Vietnam in 1962. With the Bell UH-1A Iroquois ("Huey") under the leadership of Major Charles Kelly, the Army's 57th Medical Detachment became known for the courage and hard work of flight crews, who flew despite darkness, adverse weather, and enemy fire. Later, the Bell model UH-1H was used to evacuate up to nine patients at a time by hoist from above a dense jungle canopy. By 1967, about 94,000 injured men had been evacuated.[77]

TABLE 35-1. Mortality Rates and Evacuation Times During Major Wars

CONFLICT	EVACUATION TIME (hr)	MORTALITY RATE (%)
World War I	18–30	18.3
World War II	4–6	3.3
Korea	2–4	2.4
Vietnam	1–2	1.8

From Stewart RD: Trauma Q. 1985; May: 1.

As air evacuation matured, the time from wounding to definitive care declined from 18 hours in World War I to between 1 and 2 hours in Vietnam.[101] Although medical advances have contributed to improved survival, battlefield mortality has steadily declined from 18% in World War I to 1.8% in Vietnam, perhaps more because of rapid aeromedical transport to definitive care (Table 35-1).

Unfortunately, emergency medical care for civilians greatly lagged behind the developments in the military. In the late 1960s, rescue efforts were more organized, skilled, and rapidly performed for a man shot in the Vietnam conflict than for a civilian injured on U.S. highways.[69] Civilian ambulances were said to be no faster than taxis.[90]

Civilian transport began to change dramatically in the United States in 1966 when the National Academy of Sciences–National Research Council put forth the white paper "Accidental Death and Disability: The Neglected Disease of Modern Society" (U.S. Department of Health, Education, and Welfare). This document was the impetus for improving EMS systems through the country, and soon the civilian sector began to emulate the military model.

Outside the United States, Germany and Switzerland had developed a network of helicopter and fixed-wing air evacuation and transport services that continue to provide rapid access to care from even the most remote areas.[41] The first U.S. civilian aeromedical program was begun in 1969 as a joint effort between the Maryland State Police and the University of Maryland Center for the Study of Trauma (now the Maryland Institute for Emergency Medical Service Systems). Certain hospitals were designated as trauma centers, and victims of highway and other trauma were flown by police pilot–paramedic teams in a primary response role at the accident scene. Since 1970, the service has flown more than 199,000 missions.[95]

The modern-day U.S. Air Force (USAF) aeromedical evacuation system has its root in the Vietnam conflict, during which more than 406,000 wounded troops were aeromedically evacuated. Current USAF aeromedical evacuation crews are composed of flight nurses and aeromedical evacuation technicians. Experienced nurses and medical technicians are trained to be aeromedical crews at the USAF School of Aerospace Medicine, Brooks City-Base, Texas. This 5-week course includes aircraft configuration, air and ground safety, patient flight physiology,

aeromedical nursing considerations, and a 9-day survival course. Aeromedical evacuation crews are trained to move stable patients by fixed-wing aircraft distances ranging from a few miles to transoceanic flights. Physicians do not normally accompany aeromedical evacuation flights; patient care and medication are provided by prior physician orders and written protocols.

U.S. military operations in Afghanistan and Iraq have led to significant changes in the USAF aeromedical evacuation system. Changes in military doctrine have resulted in the need to move seriously ill but stabilized patients over great distances by air. The requirement has led to development of dedicated critical care air transport (CCAT) teams, who fly with aeromedical evacuation crews to provide care for critical patients. CCAT teams include a critical care physician, critical care nurse, and cardiopulmonary technician. A formal training course in CCAT is given at the USAF School of Aerospace Medicine, Brooks City-Base, Texas.[84] The trend toward formal CCAT teams has been mirrored throughout the world, with countries such as Great Britain, Germany, Japan, Australia, and Colombia developing and fielding CCAT capabilities.[53,58]

With development of faster and more powerful helicopters, reconfiguration of fixed-wing aircraft for aeromedical needs, enhanced knowledge of aeromedical physiology, and experience accumulated through more than 50 years of transport experience, the acceptance, utilization, and success of aeromedical transport are universal. The role of aeromedical transport in the wilderness setting continues to evolve as its importance in providing rapid emergency medical care and evacuation to sick and injured patients is recognized.

TYPES OF AEROMEDICAL TRANSPORT PROGRAMS

Hospital-based Programs

The most ubiquitous type of program is hospital based. Helicopter service is often provided in primary (to the accident scene) and secondary (to the community hospital emergency department) response roles. In addition, many hospitals provide fixed-wing transport in a secondary response role for long-distance transports or when transport by helicopter is impractical.

According to the Association of Air Medical Services (AAMS), in early 1994, there were more than 175 hospital- or health care provider–affiliated and 20 freestanding rotor-wing transport programs in the United States. These services transported more than 172,000 patients in 1993. Nationally, about 70% of all flights are interfacility transports, and 30% are flights from the scene.[71]

In a hospital-based transport program, the hospital frequently leases the helicopter from a vendor, who also supplies the pilots, maintenance, and fuel. The hospital has the responsibility for providing the medical crew and determining the configuration of the crew. In addition, the program directors are responsible for medical control and quality improvement.

The hospital may choose to own the aircraft and contract with a vendor for operations or employ its own pilots and mechanics. In most cases, the helicopter resides on a helipad atop or near the hospital, and the crew, which may consist of a specially trained flight nurse, flight paramedic, and physician, is quartered in the hospital ready for immediate launch (see "Flight Crew").

Non–Hospital-based Programs

Non–hospital-based service is provided by an entity that may be supported by a consortium of hospitals, or it may be an independent corporation, ambulance service, or aviation fixed-base operator (FBO). The aircraft may be owned or leased by the entity or by an aviation contractor. Although this is not a common model for helicopter services in the United States, many fixed-wing services operate in this manner. A corporate airplane may be provided on demand for use in an air ambulance mode with its interior reconfigured, or a dedicated airplane may be provided with a custom-made air ambulance interior configuration, usually under a Supplemental Type Certificate (STC).

Public Safety, Police, or State Services

The aircraft (usually a helicopter) may be owned and operated by a governmental agency such as the state highway patrol and operated under part 135 of the Federal Aviation Regulations (FAR). As in the Maryland model, flight personnel typically include police pilots and emergency medical technician (EMT)-paramedics.

Military Assistance to Safety and Traffic Program

The Military Assistance to Safety and Traffic (MAST) program was established to supplement the civilian EMS systems. Under this program, air medical evacuation services are supplied by active-duty military medical units to the extent that their training budgets allow, provided they can use actual patient transports instead of training exercises. The MAST mission is "secondary"; it is available only when its personnel and equipment are not being used in support of the unit's primary mission. MAST may be requested by the local EMS or disaster management agency. Typical aircraft include the Bell UH-1 and Sikorsky UH-60 (Blackhawk). The medical crew usually consists of medical corpsmen. The MAST program may not compete with similar civilian services.

Other Military Resources

The USAF provides aeromedical transport in support of U.S. military operations and some disaster situations. The formal aeromedical evacuation system run by the USAF is the largest in the world, able to move large numbers of stable and critical-stabilized patients over intercontinental distances. In fiscal year 2004, the USAF aeromedical evacuation system moved 28,496 patients (4543 of whom had battle injuries); about 5% of these patients were critically ill and required CCAT team support during flight.

Other available resources include the Air National Guard and the U.S. Coast Guard. Many states and some counties have organizations (e.g., California Department of Forestry, Los Angeles County Sheriffs Department) that may be called on to assist in search and rescue (SAR) operations in preparation for aeromedical transport. Many other countries have analogous units; the Israeli Air Force, for example, operates a squadron that provides civilian and military rescue and evacuation services.

PATIENT MISSION TYPES

Primary Response

In a primary response role, the aeromedical transport service responds to an accident scene or field location, usually at the request of police, fire, or local EMS personnel, and serves as the initial and sole mechanism of transport to the hospital. In this instance, the aeromedical crew may function as "first responders." Helicopters are most suited to a primary response role. The required response times must be short (less than 10 minutes from call to takeoff); thus, the flight crew must be stationed at or near the launch site 24 hours a day. The service radius ("stage length") is short (typically less than 50 miles), and crews need to be experienced in techniques for landing in proximity to obstacles, under poor conditions, and on uncertain surfaces. In prehospital situations, patients' conditions vary widely, and often, little or no assessment or stabilization is performed before arrival of the flight crew. Medical personnel must possess a high degree of training and experience and should possess at a minimum EMT skills required for patient extrication and stabilization at the scene. It must be kept in mind that the availability of helicopter transport, particularly for on-scene response, may not affect the outcome of the patient. Several studies have found that helicopter transport does not significantly improve outcomes in areas where ground ambulance transport is readily available.[10,15,81,83] The possible benefit to the patient must be measured against the risk to the helicopter crew, especially in poor weather conditions or during the night.

Secondary Response

In the secondary response role, a patient has already been transported by other means to a hospital where some degree of stabilization may have occurred. The aeromedical service transports the patient in the early stages of care from the emergency department of a hospital to a facility better equipped to offer definitive care. Response times required for this type of mission must be competitive with one-way ground transport times. Stage lengths are short to intermediate (150 miles). The transport vehicle is typically a helicopter, although in some remote and wilderness areas, fixed-wing services are also suited to this role. Flight crews used in a secondary response vary depending on the needs of the patient (see "Medical Mission Types"). The responding aeromedical service typically consists of flight nurses, paramedics, and in some cases flight physicians.

Tertiary Response

In a tertiary response, an inpatient who requires specialized services unavailable at the current facility or who requests relocation is transported to a new facility. Tertiary transports may involve helicopters or fixed-wing aircraft, depending on the level of urgency, stage length, and cost of transport. Commercial entities throughout the world specialize in this type of service.

MEDICAL MISSION TYPES

The needs of different patient types may be categorized by medical problem; this in turn dictates the requirements of the aeromedical transport service. In most hospital-based helicopter programs, most patients transported are categorized as adult trauma, cardiac, or medical noncardiac. A number of programs offer or specialize in pediatric, neonatal, perinatal, and organ transplantation services, for which specialized crews and equipment may be required. In addition, aeromedical transport programs that provide SAR operations require specialized equipment and training.

Trauma Patients

Trauma patients transported in the primary or secondary response modes may account for 20% to 60% of a hospital-based helicopter service's transport activity, depending on the hospital's function and capability as a trauma center and the relationship between the aeromedical service and the community EMS and public safety network. A study of one urban setting noted that 20% of helicopter missions were to injury scenes, which were located at a mean distance of 14.4 miles from the hospital. Of patients transported, 19% had penetrating trauma, and 81% blunt trauma (66% from motor vehicle crashes). The most common organ system injuries involved the head (65%), extremities (39%), chest (31%), and abdomen (27%). The overall mortality of transported patients was 24%. The most common procedures required at the scene were endotracheal (ET) intubation (41%) and cardiopulmonary resuscitation (CPR) (18.7%). The most common life-threatening conditions were cardiac arrest (18.7%), airway obstruction (5.1%), cardiac tamponade (3.2%), and tension pneumothorax (1.7%).[36]

A multicenter study of blunt trauma victims transported by helicopter aeromedical services from both urban and rural environments found a mean trauma score of 13 (of 16), mean age of 29 years, and overall mortality rate of 15%.[10]

These and other studies indicate the need for skilled crews in the transport of trauma patients.[105] Medical personnel must have the ability to assess the patient adequately to detect frequent in-flight complications and to intervene with appropriate procedures, including intravenous (IV) cannulation, ET intubation, CPR, chest decompression, and at times a surgical airway (Box 35-1).

In wilderness areas, the flight crew must be skilled at victim extrication and operating in rugged terrain. They must be familiar with standard trauma care and the range of clinical entities most frequently seen in the wilderness setting. In addition, because resources may be limited and backup unavailable, they may be required to function semi-autonomously. For this reason, protocols and standing orders are valuable. Most important are training, skill, and judgment.

Patients with Cardiac Disease

Patients with cardiac disease most often are transported in a secondary or tertiary response role, by either helicopter or fixed-wing aircraft. They typically account for 20% to 50% of an aeromedical service's transport activity. The condition of these patients is often medically complex. Technologically sophisticated treatment modalities may include antiarrhythmics, vasopressors, inotropes, vasodilators, thrombolytic agents, cardiac monitoring, arterial and central venous pressure monitoring, pacemakers, implantable defibrillators, and intraaortic balloon counterpulsation devices.[25,33,40,55] The flight crew must have sophisticated knowledge, expertise, and experience and may include a cardiac critical care nurse and a physician.

Patients with Medical, Noncardiac Conditions

Patients with medical, noncardiac conditions, including those with cardiac disease, are most often transported in the second-

Box 35-1. Trauma Care Aboard Emergency Medical Services Helicopters

MECHANISM OF INJURY
- Motor vehicle crash
- Fall
- Industrial or agricultural accident
- Gunshot or stab wound
- Burn
- Sporting accident
- Drowning
- Hypothermia

PROCEDURES PERFORMED BY FLIGHT CREW
- Endotracheal intubation
- Cardiopulmonary resuscitation
- Intravenous lines
- Central venous access
- Extrication and splinting
- Bladder catheterization
- Nasogastric tube insertion
- Venous cutdown
- Tube thoracostomy
- Cricothyrotomy
- Pericardiocentesis
- Antishock garment application

ary or tertiary response mode by either helicopter or fixed-wing aircraft. This group consists largely of patients with acute neurologic disease or shock or those who require assisted ventilation.[45] The spectrum of potential in-flight challenges includes cardiovascular problems, arrhythmias, hypotension, respiratory difficulties requiring acute airway management, seizures, and alterations in level of consciousness. The flight team must be able to manage an airway and operate a ventilator. Additional considerations relate to the cabin environment and need for pressurization if hypoxemia is present, if barotrauma is likely, or if trapped gas exists, as well as the need to predict the requirement for and manage finite oxygen resources in flight.

Pediatric Patients

Pediatric patients may have traumatic or medical conditions.[11,48] In a study of 636 pediatric patients transported by air in the Salt Lake City area, 57.5% were transported by helicopter and 37.5% by fixed-wing aircraft, with a mean stage length of 207 miles (helicopter, 82 miles; fixed-wing, 452 miles). Less than 1% of flights were from the scene. The patient ages ranged from 3 weeks to 16 years, with 45% younger than 1 year. Trauma was the most common diagnosis (15.3% had head injury, 9.3% multiple injuries), followed by neurologic illness (24.2%), respiratory failure or infection (20.1%), gastrointestinal or genitourinary problems (10.2%), metabolic disease (9.2%), cardiovascular disease (6%), and general pediatric surgical problems (5.7%). The overall mortality rate was 7%.[68] Many of the considerations for pediatric transport are similar to those for adults, especially with older children. Infants may require an incubator, however, and flight crews must be experienced in caring for infants and children. Specifically, knowledge of pediatric advanced cardiac life support skills, including pediatric drug dosages, airway sizes, and fluid management, is essential.

Perinatal Patients

The need for expedient evaluation, preparation, and transport of the obstetric-gynecologic patient is increasing. Types of problems include ectopic pregnancy, pelvic inflammatory disease, toxic shock syndrome, abnormal fetal presentation, multiple gestation, diabetes in pregnancy, placenta previa, abruptio placentae, disseminated intravascular coagulation, preeclampsia-eclampsia, and preterm labor. The decision to transport patients in advanced preterm labor should be based on such factors as distance between hospitals, time required to cover the distance, personnel available for the transport, gestational age, and speed with which labor has progressed. The flight crew must be knowledgeable about these problems and comfortable with their treatment in order to ensure a favorable outcome for both mother and child.

Neonates

Neonates have unique anatomy and physiology, and the diseases that affect them require specific knowledge and skills by those involved in their transport. Specific issues include newborn assessment, including assignment of an Apgar score, airway clearance, temperature, homeostasis, and familiarity with neonatal resuscitation.[3] Access to references concerning neonatal emergency drug dosages should be available.[2]

The ability to perform umbilical vein catheterization is an important skill for any member of the transport team involved in neonatal care. In addition, knowledge of fluid, electrolyte, and glucose requirements is essential.[53,60] The flight crew involved in the transport of a neonate often includes a neonatal nurse and a neonatologist.

Search and Rescue

Wilderness SAR is a unique aspect of aeromedical care and transport that requires significant training and expertise. Most dedicated aeromedical aircraft in the United States are not well suited for SAR operations (see "Aircraft for Search and Rescue"). Most standard aeromedical crews are not trained in SAR techniques. Many aeromedical helicopters and some fixed-wing aircraft become involved in SAR activities, however, and it is important to be familiar with SAR techniques. In addition, outside the United States, persons providing aeromedical transport are frequently involved in SAR activities (see Chapter 32).

The keys to a successful SAR operation include proper communications, transport, evacuation, and medical treatment, in the setting of favorable weather conditions and topography. The helicopter, equipped with a hoist and winch, is one of the most effective means of providing SAR in the wilderness setting and is essential in mountainous regions. A long delay between the time of the accident and the call for assistance, combined with a serous injury, adversely affects patient outcome.

Helicopters are helpful in various SAR activities, including low-altitude search activity, search area evaluation, and movement of supplies and equipment. They may be the only means of extrication and rescue from the scene. Fixed-wing aircraft are also useful for search and can provide secondary transport, especially when long distances are involved.

The U.S. Air Force routinely uses helicopters and fixed-wing aircraft for long-distance SAR operations. Fixed-wing aircraft often arrive at the scene first and may deploy pararescue specialists ("PJs") by parachute. If the rescue site is over water, the aircraft may also deploy an inflatable boat with motor and rescue equipment. The PJs and their survivors are then

Figure 35-1. MH-60G Pave Hawk hoisting a para-rescuer during a search and rescue exercise. A variant of the UH-60 Blackhawk, the Pave Hawk is flown by U.S. Air Force rescue squadrons. Modifications include forward-looking infrared equipment, night vision–compatible cockpit lighting, terrain and navigation radar, air-refueling probe, auxiliary fuel tanks, and hoist. (Courtesy U.S. Air Force.)

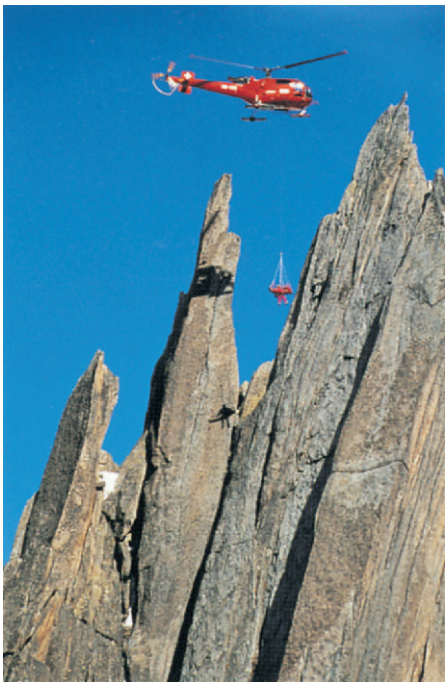


Figure 35-2. Helicopter rescue in extremely difficult terrain. (Courtesy P. Bärtsch, MD, and the Swiss Alpine Club.)

recovered by surface vessels or by rescue helicopters that have been refueled aerially in order to reach the rescue site (Fig. 35-1). This capability permits the rapid deployment of rescuers while allowing the most expeditious recovery of survivors and their delivery to definitive care. These services are on alert for all space shuttle launches to provide SAR support in the event of a mishap.

In the United Kingdom, the Royal Air Force operates a helicopter SAR service that flew 1490 missions from 1980 to 1989, almost all of which involved vacationers along the coasts or in the mountains.[63] The Danish helicopter rescue service was founded in 1966 and uses a Sikorsky (S-61) helicopter. Since 1973, its crew has included a physician trained in aerospace medicine and helicopter transport. From 1973 to 1989, it flew 5733 missions, 2075 of which involved direct medical intervention. The most frequent problems were abdominal trauma and cardiopulmonary diseases.[112]

In the high Alps, more than 90% of all rescues are performed using helicopters (3000 per year).[6] Of these, 5% are combined rescues; that is, the helicopter carries the rescuers below cloud level, near the site of the accident. Only 5% of mountain rescues are purely ground rescues, mainly necessitated by visibility.[89] Currently, a network of SAR systems extends throughout the Alps. In some countries (France, Italy, Germany, Austria, and Spain), air rescues are managed partially or totally by the army or the state. The aircraft most often used for this purpose are the Alouette III, Lama, Ecureil (French), Bolkow 105, 117 (German), Augusta AK 117 (Italian), and Bell (United States). In Switzerland, the rescue system in remote terrain is managed by the Swiss Alpine Club and three air rescue companies, Swiss Air Rescue (REGA), Air Glaciers, and Air Zermatt. Switzerland may be unique in that its 18 strategically placed helicopter rescue bases allow an aircraft to reach any accident scene within 15 minutes of takeoff. Since the foundation of REGA in 1952, more than 150,000 patients have been transported by either fixed-wing aircraft or helicopter.

Up to 8000 patients (5500 from accident scenes) are transported by helicopter every year. Twenty percent of these rescues require a winch, with one third of all winch operations occurring in accident sites that are difficult to reach.[31] More than 75% of all persons rescued by winch were thought to have injuries requiring physician assistance at the scene. Eighty percent of all Swiss air rescue missions are physician assisted, and 20% have a paramedic in charge. All the physicians and rescue crews are physically fit and trained in alpine techniques because two thirds of all rescue missions performed from 1990 to 1993 were in topographically remote and difficult terrain (Fig. 35-2).

Difficult helicopter SAR operations are those that involve low visibility, strong winds, night missions, high-angle rescues, and long-line hoist operations (extension of the hoist cable up to 120 m [394 feet]). In addition, in mountainous regions, power cables and transport cables present considerable risks. In all cases, the risks to the flight crew (as well as to the patient) must be weighed against the degree of injury and risk for further morbidity. The U.S. Air Force, Army, Navy, and Coast Guard equip and train groups to operate in these hostile rescue environments. Helicopters are frequently equipped with precision navigation systems and night vision, forward-looking infrared (FLIR), and thermal imaging equipment. The intense training and specialized equipment permit rescue operations under much more demanding conditions than those encountered by civilian services. Of the military services, only the U.S. Coast Guard has a primary mission of civilian SAR; Army, Navy, or Air Force groups may be requested to assist in civilian rescues in areas where they are available.

An increasing number of people participate in alpine sports, including mountain climbing, downhill skiing, mountain biking, and paragliding.[33] A typical representation of the type of mountaineering accidents experienced in the Swiss Alps is shown in Table 35-2. In addition to SAR in mountainous regions, aeromedical rescue presents great challenges to the medical and flight crews involved in rescues from sea and white water, floods, vertical rock faces, and avalanches.

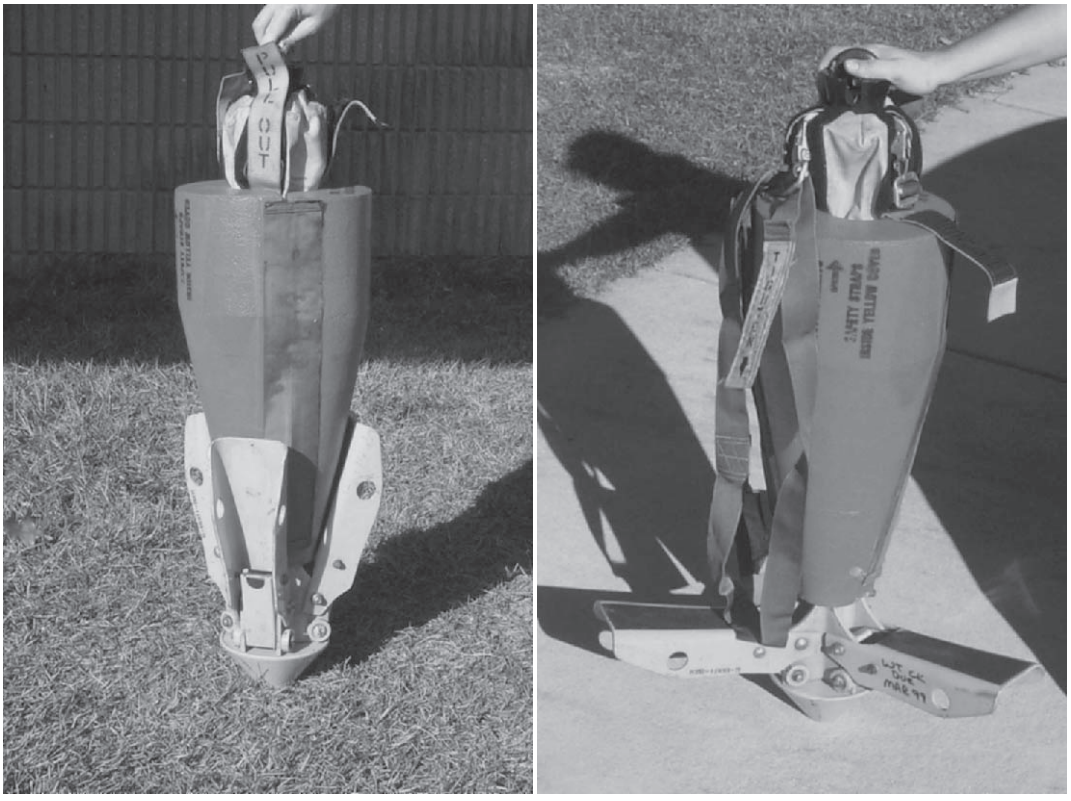
TABLE 35-2. Mountaineering Accidents in the Swiss Alps, 1992 (N = 1845)

ACTIVITY	PATIENTS RESCUED (n)
Delta gliding	18
Paragliding	196
Off-slope skiing	35
Ski touring	238
Mixed climbing	456
Rock climbing	178
Hiking	723

From Dürrer B, Hassler R, Mosimann U: Mountaineering accidents in the Swiss Alps and rescue activities of the Swiss Alpine Club, 1992.

Medical treatment of the survivors should begin immediately at the site of the accident unless weather conditions are deteriorating or the scene is inherently unsafe. If a hoist extraction is needed, the patient with potential multisystem trauma should be evacuated by a rescue net or basket (Stokes) litter with careful spinal immobilization. A tag line attached to the litter prevents spinning during hoist operations. The tag line should be attached with a weak link so that the tag line will break away if the litter becomes uncontrollable. Persons with minimal or isolated injuries may be hoisted by a jungle penetrator (Fig. 35-3), rescue basket, or other dedicated hoist device. If a hoist device is not available, the victim may be hoisted by climbing harnesses or rescue belts.[30] The climbing harness or belt should be carefully inspected to make sure that it has not been damaged in the mishap, that it will withstand the strain of the hoist operation, and that it can be safely attached to the hoist cable.

The extent of medical treatment rendered on site before extraction depends on many factors, including the victim's condition, scene safety, medical supplies available, medical skill of the rescuers, weather conditions, aircraft loiter time, and flight time to definitive care. Good communication between the flight



A B

Figure 35-3. **A,** Jungle penetrator used as a hoist device on most military rescue helicopters. The streamlined shape allows it to slip through dense tree canopies to reach the ground. A foam flotation collar can be attached, making the penetrator buoyant. **B,** Jungle penetrator rigged for hoist. The seats are flipped down, and the safety straps are pulled out from their stowed position and passed over the head and under the arms of the victim, who then straddles one of the seat paddles. Although the penetrator has three seats, usually only one or two persons are hoisted at a time. (Courtesy Robert C. Allen.)

Figure 35-4. Stanford LifeFlight MBB BK-117. (Courtesy Geralyn Martinez.)

Figure 35-5. U.S. Coast Guard HH-60J Jayhawk helicopter in low hover over water, preparing to lower hoist cable to the rescue swimmer. Note the extensive spray under the aircraft. The Jayhawk, a variant of the UH-60 Blackhawk, is a medium-range helicopter used for search and rescue, drug interdiction, and maritime law enforcement. (Courtesy U.S. Coast Guard.)

crew and the rescue team is essential to the decision-making process. Aeromedical crews involved in mountain rescue need to have a thorough understanding of the unique medical problems frequently found in high-altitude rescue situations.

AEROMEDICAL AIRCRAFT

Many different types of aircraft can be adapted to the air ambulance role. Each type has its strengths and weaknesses. On the other hand, not all aircraft are well suited for SAR in wilderness areas. Matching the physical and flight characteristics of the aircraft to the needs of the mission is vital. In many circumstances, compromises must be made because of aircraft availability. Rescuers must consider the physical characteristics of the aircraft when caring for a victim in flight.

Cabin Space

Cabin space should be considered not only in terms of total interior volume in cubic feet, but also with regard to floor space, headroom, and the ergonomics of cabin layout. Ample headroom should be available for the patient to lie comfortably on a secured stretcher and for access by two crew members to all parts of the body. Specifically, access to the head is needed for intubation, the chest for CPR, and the extremities for monitoring perfusion. Some helicopters, such as the Aerospatiale AStar/TwinStar and the MBB BO-105, provide ample upper body access but only limited lower body access while in flight. The relationship of flight crew members when seated (and secured by seat belts) in proximity to the patient is important. The ideal configuration places one medical crew member at the patient's head for airway management and verbal interaction and one at the patient's side to monitor vital signs and perform necessary non–airway-related procedures. This arrangement is typified by the MBB BK-117 helicopter (Fig. 35-4).

Although some rotor-wing aircraft, such as the BK, are theoretically capable of transporting two patients, this greatly increases demands on the flight crew and the aircraft and diminishes access to both patients. Policies and procedures regarding two-patient transport in these aircraft should be carefully considered. In some large helicopters, such as the CH-46, CH-47, and MH-53, there is enough room to work on multiple patients. Even on relatively large (by civilian standards) military helicopters, however, such as the Coast Guard HH-60J Jayhawk (Fig. 35-5), space for patient care can be at a premium.

Cabin space can be more generous in fixed-wing aircraft. Cabin-class airplanes, such as the Beech King Air and Piper Cheyenne III, provide an aisle and capability to carry more than one patient and additional crew or family members. However, even in large military aircraft such as the C-130, carrying multiple patients with equipment and supplies can still result in a remarkably crowded cabin.

Access for Patient Loading

The cargo door should be wide enough that the patient's stretcher can be maneuvered into the aircraft without undue tilting, and it should be positioned comfortably near stretcher height to obviate the need for strenuous lifting during ground loading. Standard door configurations on many aircraft do not meet these needs. The "clamshell" doors on an MBB BK-117 helicopter and the oversized cargo door on a Gulfstream Commander 1000 work well for patient loading (Fig. 35-6).

Useful Load

One of the most important considerations for a given patient transport is the aircraft's useful load. This difference between the maximum takeoff weight and the basic empty weight is a reflection of the load-carrying capability. In most EMS helicopters, the useful load ranges between 1500 and 2800 pounds (680 and 1270 kg). On-board avionics, medical equipment, fuel, and crew weights must be subtracted from this value to yield the maximum allowable patient weight (Fig. 35-7). Fuel weighs 6 pounds (2.7 kg) per gallon; a twin-engine helicopter may burn 70 gallons (265 L) per hour (420 pounds [190.5 kg] per hour), requiring it to carry 600 pounds (272.1 kg) or more fuel for a 30-minute-radius flight (with 30-minute reserve). Thus, it becomes evident that a flight crew of three weighing a total of

Figure 35-6. MBB BK-117 with rear clamshell doors open. (Courtesy Susan Lockman, Stanford Lifeflight.)

500 pounds (226.8 kg) with a full load of fuel, oxygen, and medical gear may not have the capability to carry even a small patient, especially on a hot day when the helicopter's performance (lift) is reduced. This consideration can become critical on flights from the accident scene, where terrain obstacles may require vertical takeoff and climb-out, demanding maximum helicopter performance. Density altitude (a factor of the air temperature and the pressure altitude) is critical in the performance of helicopters in mountain regions. High altitude combined with hot weather can seriously degrade the performance of even the most powerful helicopters.

Weight and Balance

Not only must the weight of the loaded aircraft remain at or below the maximum allowed takeoff weight for that aircraft, but also the center of gravity (CG) must lie within fore-and-aft limits established by the manufacturer. Each loading configuration places the CG in a unique position, which must be calculated by the pilot before flight, or the flight characteristics may be adversely affected, compromising safety. This consideration may dictate where certain pieces of medical equipment, such as oxygen bottles, may be placed or where heavier crew members must sit.

On flights from the accident scene, the patient's weight is approximated before departure. With pressure to hasten departure, accurate weight and balance calculations are difficult. The aircraft used must have enough margin in the CG envelope that CG limits are not easily exceeded for the given mission profile.

Cruise Speed

One of the most basic reasons for transporting a patient by air is to take advantage of the greater speed of aircraft compared with ground vehicles. This allows the patient earlier arrival at the destination and minimizes time spent out of the hospital.

Maximum take-off weight[a]		_____
Less basic empty weight[b]	–	_____
Less fuel on board[c]	–	_____
Less pilot and crew	–	_____
Less medical gear[d]	–	_____
Maximum "patient payload"	=	_____

[a]Maximum take-off weight is certificated for each aircraft type and can be found in the operating manual.

[b]Basic empty weight includes added avionics, permanent equipment, fluids, and unusable fuel. It is different for each aircraft and is recorded in the aircraft's operating manual.

[c]The quantity of fuel on board depends on the mission needs. Divide round-trip distance by cruise speed to yield time en route. Add time for warm-up, climb, approach, and a 30-minute reserve (VFR) to yield total engine time. Multiply total engine time by rate of fuel consumption to yield total fuel consumed. Fuel weight 6 lb/gal.

[d]Medical gear includes carry-on and nonpermanent items.

Figure 35-7. Weight calculation aboard aircraft.

TABLE 35-3. Helicopters Frequently Used for Aeromedical Transport

HELICOPTER	CRUISE SPEED (mph)	ENGINE	SHP	USEFUL LOAD (lb)	SERVICE CEILING (ft)	RANGE (mi)*
Bell 206L-3	130	SE-T	650	1,950	20,000	325
AStar 350D	140	SE-T	615	1,868	15,000	379
TwinStar 355F1	147	TE-T	420 each	2,391	13,120	368
MBB BO-105 CBS	145	TE-T	420 each	2,732	17,000	334
Agusata 109A II	163	TE-T	420 each	2,605	15,000	359
Bell 222UT	152	TE-T	684 each	3,376	15,800	380
MBB BK-117	160	TE-T	650 each	2,645	17,000	368
Sikorsky S-76	167	TE-T	650 each	4,700	15,000	550
Dauphin 2	161	TE-T	700 each	4,118	15,000	564

*Range includes fuel for warm-up, taxi, climb, and 30-minute reserve.
SE-T, single engine, turbine; SHP, shaft horsepower; TE-T, twin engine, turbine.
Data from Collins RL, et al: Flying 1985 Annual and Buyer's Guide. New York, Ziff-Davis, 1985; and 1987 Hospital Aviation Directory. Hosp Aviat 1987;6(4):8.

Not only do aircraft have a speed advantage, but they can also travel in a straight line from origin to destination without the curves and deviations present in surface travel. For an aircraft to compete with a ground vehicle in speed in a primary or secondary response mode, it must be at least twice as fast as an ambulance because the helicopter must fly round trip (outbound to destination and inbound with patient) in the time that the ambulance would travel one way. This is possible with most EMS helicopters, unless (1) the referral location has no suitable landing area (necessitating a time-consuming transit of crew and stretcher to and from the location), (2) ground "packaging" times for the flight crew with the patient are excessive, or (3) an ambulance has a clear, straight highway as a means of alternative transport.

Most EMS helicopters are capable of attaining 120 to 150 mph (193 to 241 km/hr) over the ground, although a headwind or tailwind may hinder or improve these figures (Table 35-3). Piston twin-engine aircraft have a cruise speed range of 220 to 275 mph (354 to 443 km/hr), turboprop aircraft of 300 to 385 mph (483 to 620 km/hr), and jets of 400 to 535 mph (644 to 861 km/hr) or more.[24,78]

Range

Aircraft range is limited by the amount of fuel, which is a function of fuel tank capacity and useful load. In most cases, a tradeoff is made between payload and fuel; the more weight in fuel, the less weight in passengers (or patients). The maximum time aloft can be calculated by dividing usable fuel on board by rate of fuel burn per hour at cruise speed. Multiplying maximum time aloft by cruise speed yields the maximum range. The Federal Aviation Administration (FAA), under FAR part 91.23, requires that a 45-minute fuel reserve remain at the conclusion of all flights conducted under instrument flight rules (IFRs), and a 30-minute reserve under visual flight rules (VFRs). Most EMS helicopters operate under VFRs, whereas most fixed-wing operations are IFRs. Because helicopters typically fly to and from a point at which refueling is not available, round-trip fuel must be carried; this effectively limits the customary radius of operation to about half the range (less required reserves). Although it is possible to refuel en route to an airport, this adds to the flight time. Therefore, helicopters typically operate within a radius of 150 miles (241 km) or less, unless the transport is one-way outbound or unless refueling at the destination is feasible. Fixed-wing aircraft operate from airport to airport; thus, the radius of operation is closer to the maximum range with reserves. Many fixed-wing aircraft are capable of ranges in excess of 1000 miles, with some jets able to travel more than 2000 miles without refueling.

Pressurization

The partial pressure of oxygen in the atmosphere declines with increasing altitude so that at 5486 m (18,000 feet) it is one half that at sea level. Part 91.32 of FAR requires the use of supplemental oxygen for the pilot at flight altitudes above 3810 m (12,500 feet) for longer than 30 minutes.[34] Above 4267 m (14,000 feet), supplemental oxygen must be used by the pilot and all minimum required flight crew at all times. Technically, a medical flight crew member is not a required minimum crew member for the operation of the aircraft; neither is the copilot of an aircraft operated under FAR part 135 and certified for single-pilot operations (as with most aeromedical aircraft). Thus, the medical crew member is not required to wear supplemental oxygen, although doing so would be prudent, especially for smokers. At altitudes greater than 4572 m (15,000 feet), in addition to the above requirements, each occupant must be provided with supplemental oxygen (although there is no legal requirement to use it).

The effects of hypoxia with increases in altitude are more pronounced in patients with lung disease and preexisting hypoxia; this necessitates supplemental oxygen at much lower altitudes. Supplemental oxygen at night will enhance night vision even at altitudes below 3810 m and should be considered for the flight crew, based on operational requirements.

To eliminate the need to provide supplemental oxygen, pressurization is available in many larger, fixed-wing aircraft (Table 35-4). A pressurized aircraft is able to pump air into the cabin to maintain a pressure differential between the cabin and outside air, generally 4 to 8 pounds per square inch (PSI). This allows the cabin atmosphere to be maintained at or below the equivalent of a 2438-m (8000-foot) altitude, despite actual altitudes of 9144 m (30,000 feet) or higher.[53,70] Pressurization obviates the need for supplemental oxygen for crew members and nonpatient passengers, but passengers with lung disease may still require it. Also, by limiting the drop in cabin pressure that occurs with altitude, changes in trapped gas volumes, such as in ET tube cuffs, air splints, and the gastrointestinal tract, can

TABLE 35-4. Fixed-Wing Aircraft Used for Aeromedical Transport

AIRCRAFT	CRUISE SPEED (mph)	CABIN	ENGINE LOAD (lb)*	USEFUL CEILING (ft)	SERVICE (ft)	RANGE (mi)†	TAKEOFF (ft)‡
Seneca III	221	NP	TE-P	1,921	25,000	721	1,250
Baron 58TC	277	NP	TE-P	2,447	25,000	1,150	2,700
Cessna 402C	245	NP	TE-P	2,774	26,900	1,164	2,200
Navajo 350	250	NP	TE-P	2,533	24,000	1,200	2,200
Cessna 414	258	P	TE-P	2,386	30,800	1,300	2,600
Cessna 421	277	P	TE-P	2,807	30,200	1,522	—
Cessna 441	330	P	TE-T	4,124	35,000	2,195	—
Cheyenne II	293	P	TE-T	4,053	31,000	1,275	2,500
Cheyenne III	347	P	TE-T	4,448	35,000	1,789	3,200
MU-2	317	P	TE-T	3,975	27,300	1,412	—
King Air F90	309	P	TE-T	4,383	31,000	1,315	2,900
Commander 1000	323	P	TE-T	3,965	30,750	2,149	
Citation I	410	P	TE-J	5,222	41,000	1,500	3,000
Lear 25D	509	P	TE-J	7,150	51,000	1,600	4,000

*Useful load excluding avionics, fuel, passengers.
†Range estimated at cruise speed, less 45-minute reserve.
‡Approximate nonbalance-field takeoff length.
NP, nonpressurized; P, pressurized; TE-P, twin engine, piston; TE-T, twin engine, turboprop; TE-J, twin-engine, turbojet/turbofan.
Data from Collins RL, et al: Flying 1985 Annual and Buyer's Guide. New York, Ziff-Davis, 1985; 1987 Hospital Aviation Directory. Hosp Aviat 1987;6(4):8; and McNeil EL: Airborne Care of the Ill and Injured. New York, Springer-Verlag, 1983.

be decreased or eliminated. Special categories of patients include those with dysbarism. Exposure to increased altitude, with its concomitant decrease in ambient pressure, should be avoided. If possible, sea-level ambient pressure should be maintained when transporting these patients.

On the other hand, most helicopters are nonpressurized and generally fly at lower altitudes, where altitude-related hypoxia is unlikely. One exception occurs in mountainous regions where altitudes required to rescue victims or cross mountain passes may exceed 3658 m (12,000 feet). Reasons for transporting patients at higher altitudes include terrain avoidance, the need to surmount adverse weather (which usually occurs within 6096 m [20,000 feet] above ground), and greater speed and fuel efficiency at higher altitudes.

Service Ceiling

The service ceiling is the maximum altitude at which an aircraft can still maintain a rate of climb of 30.5 m (100 feet) per minute. This ceiling is important in predicting an aircraft's ability to climb above adverse terrain and weather and in taking advantage of favorable winds aloft to maximize ground speed. In the western United States, mountainous areas require flight at least 610 m (2000 feet) above the highest terrain along the route of flight, which means a 3658- to 4877-m (12,000- to 16,000-foot) service ceiling. These altitudes restrict most helicopters and require use of supplemental oxygen in nonpressurized airplanes. Flight operations that typically require flight at these altitudes should have access to aircraft with sufficiently high service ceilings and pressurization.

Runway Length

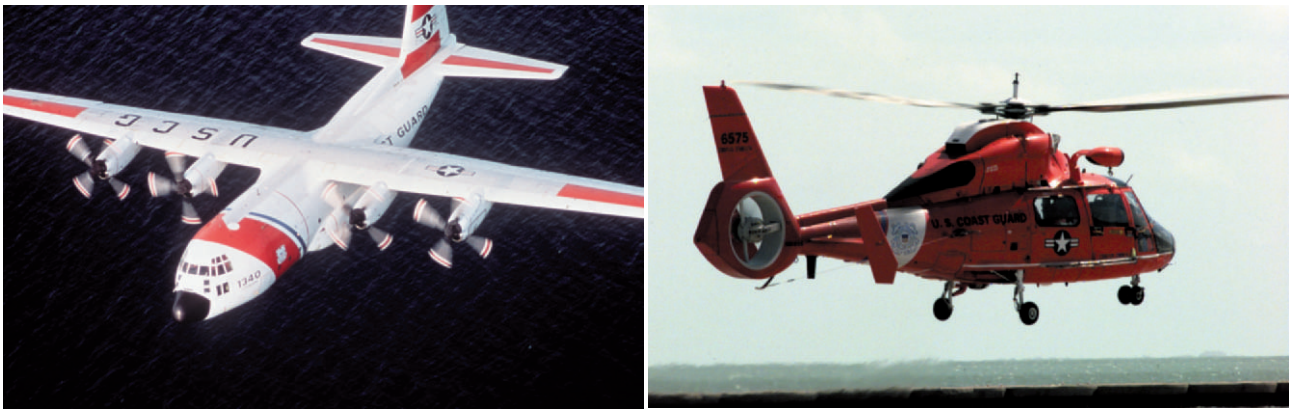
Although not a factor in helicopter operations, runway length restricts certain fixed-wing aircraft from landing. Most airports in rural areas have runway lengths between 610 and 1220 m (2000 and 4000 feet). Higher-performance airplanes usually have progressively longer runway requirements and may be unable to land and take off safely on these strips. Thus, when transport from a rural location with a short runway is requested, it is important to determine the capability of the aircraft being used. Piston twin-engine aircraft can usually operate safely from a 762- to 914-m (2500- to 3000-foot) strip but may have difficulty with 610 m (2000 feet); turboprop airplanes require 762 to 1067 m (2500 to 3500 feet); and jets usually require runway lengths of 1220 m (4000 feet) or more.[70] The takeoff roll for airplanes increases with increasing temperature and airport altitude; on a hot day, many airplanes may be incapable of taking off from a short runway if heavily loaded.

In winter conditions, operating on icy runways may pose a safety hazard for braking. Turboprop airplanes and jets have a reverse thrust mode that can slow the aircraft on rollout without braking.

Weather Operations

Adverse weather conditions that may affect a given flight include restrictions in visibility resulting from precipitation, fog, haze, or clouds, as well as airframe icing, turbulence, and wind shear. Flight during instrument meteorologic conditions (IMCs) requires adherence to IFR, whereas visual meteorologic conditions (VMCs) allow alternative use of VFR. VMCs for airplanes are defined as visibility of at least 3 miles and ceiling of at least 305 m (1000 feet) (departing from an airport in controlled airspace).[34] The ability to fly IFR not only improves the likelihood that the mission can be undertaken and completed safely should clouds or adverse weather be present but also enhances the ability of the air traffic control center to follow the flight and properly separate aircraft.

IFR capability has drawbacks. Sophisticated and expensive equipment and training are required. Virtually all fixed-wing aircraft are capable of IFR operations, but most EMS helicopters are not. IFR operations are usually conducted from airport to airport (where an instrument approach is available), but most helicopters travel to and from nonairport points without an instrument approach. The percentage of actual



A B

Figure 35-8. **A,** U.S. Coast Guard C-130 Hercules aircraft. This long-range aircraft is used for ice patrol, fisheries management, and search and rescue. **B,** Coast Guard HH-65 Dolphin helicopter. This short-range light-lift helicopter is used for near-shore search and rescue, maritime fisheries enforcement, and law enforcement. (Courtesy U.S. Coast Guard.)

missions canceled or aborted because of IMCs is small in most rotor-wing programs. One study determined that inadvertent excursions into IMCs occurred about 1.3 times per pilot per year, and the anticipated percentage of operations that would be conducted IFR, if it were available, was 9.4%.[76] For most hospital-based programs, the cost of upgrading to a more expensive IFR-equipped helicopter (especially if a copilot is necessary for IFR certification), plus the added expense of maintaining pilot IFR proficiency, would be prohibitive. As a rule, military helicopters fly with a pilot and copilot and are usually capable of flying in IFR conditions, although IMCs are far from ideal for SAR operations.

Performance

Closely related to aircraft speed is its ability to climb, expressed in feet per minute (fpm). Known as performance, this ability dictates the type of aircraft used for a given aeromedical transport mission. The greater the performance (a complex function of power, weight, wing, propeller, and air density characteristics), the better is the aircraft's ability to out-climb adverse weather or to avoid rising terrain or obstacles. Helicopters are unique in their ability to hover above the ground effect, that is, to climb vertically out of the supporting cushion of air produced by the rotor wash. Helicopters perform better when they have a running start, building up forward speed while still in the cushion of ground effect until translational lift is developed. Translational lift results from the forward-to-backward flow of air over the rotor blades. A helicopter' s ability to climb vertically out of ground effect is limited by horsepower and weight. On a hot day at high altitude, performance may be insufficient to take off vertically.[50] This must be considered when selecting a landing site away from an airport. If a confined space surrounded by obstacles is selected, a vertical takeoff may be required.[19]

Fixed-wing aeromedical aircraft are virtually all twin engine not only for enhanced speed, performance, and cabin space but also for the necessary redundancy of systems required for IFR operations under FAR part 135. If one engine fails, a second is available to allow flight to be maintained; however, if failure occurs during takeoff, single-engine climb performance may not be adequate to provide lift. This fact may be critical if insufficient altitude has been gained to allow a return to the airport before obstructions are encountered. Therefore, single-engine climb performance for various types of aircraft must be considered, especially if operating out of high-altitude airports, in hot weather, or in mountainous regions (see Table 35-4). In general, single-engine climb performance is about 200 to 290 fpm in piston twins, 600 to 900 fpm for turboprops, and 1000 to 2000 fpm for jets.[70] The airplane with the best single-engine climb performance will provide the greatest margin of safety, but the cost of equipment, pilot training, and adequate runways will be high.

Aircraft for Search and Rescue

SAR is a special type of aeromedical transport that demands aircraft uniquely suited to this role. The aircraft should have good visibility to the sides and below, the ability to fly slowly and to hover, the ability to land away from an airport, and adequate performance in high-density altitude conditions. In addition, certain extrication situations require the capability to hoist victims from rugged or hostile terrain.

Helicopters are the aircraft of choice for many SAR missions (Fig. 35-8). Few hospital-based EMS helicopters are configured for hoist operations, and hospital flight crews are typically not trained in SAR techniques. The SAR mission differs from other types of medical missions in its requirement for low-level flight over potentially hostile terrain, its use of flight crews for visual surveillance for survivors or wreckage, the need for a prolonged hover if hoist operations occur, and the need for flight crew training in wilderness survival principles if a mishap occurs. Experience and training in these activities are essential for safety. In general, military helicopters and their crews are better equipped and trained to carry out SAR operations. For example, U.S. Coast Guard and Air Force helicopters have radio locating equipment to pinpoint emergency location transponders (ELTs), also known as emergency position-reporting beacons; possess night vision and FLIR cameras to maximize the probability of visually locating a survivor; and have hoists that enable them to extract survivors from areas where landing is not an option.

Flight crews on these missions must be specially trained in the use of rescue equipment and must possess the appropriate medical qualifications and experience to deal properly with atypical EMS situations. U.S. Navy and Coast Guard rescue swimmers and Air Force PJs are trained to enter the water or proceed on land to aid in the recovery of survivors. Navy and Coast Guard rescue swimmers receive basic medical training; USAF PJs are trained to the EMT-paramedic level and are given additional training in long-term care of trauma victims.

Special hazards exist in mountainous areas. High-density altitudes may limit an aircraft's performance, and local weather patterns may be erratic. On the leeward side of mountains or ridges, severe downdrafts may prevent a helicopter from hovering out of ground effect. The landing site selected should be free of terrain obstacles and should allow for a long, shallow approach and departure. Open areas away from the leeward side of mountains or ridges are preferable.

SAR aircraft may not routinely carry the same medical equipment (e.g., ventilators, pacemakers, and defibrillators) as the typical EMS helicopter. Care should be taken to verify that the medical equipment carried on a SAR aircraft is adequate for the intended mission (see Chapters 32 and 33).

Pilot Requirements

Helicopter EMS are usually VFR operations, and the FAA has established minimum requirements for pilot experience. FAR part 135.243 specifies that the pilot in command of a helicopter carrying passengers for hire must have at least 500 hours of flight time, including at least 100 hours of cross-country time with 25 hours at night. Fixed-wing services are typically IFR operations, and pilots must have at least 1200 hours of flight time, including 500 cross-country, 100 night, and 75 hours of actual or simulated instrument time. They must also be instrument rated and possess a commercial certificate.

Most EMS helicopter pilots have much more experience than the minimum requirements; one survey found 59% had more than 4000 hours, and none fewer than 2000 hours.[35] The pilot in command is solely responsible for the safety of all passengers and must decide whether to accept or decline a mission. For this reason, the pilot is often not told the nature of the medical mission until a decision to go is made. This decision should be based on the destination, weather conditions, environmental circumstances, and estimated time at the scene, airport, or destination facility. No mention of patient type or severity should be made to the pilot before the launch decision is made so that this decision is objective. The pilot has the final say on all decisions related to safety of flight.

Communications

Helicopter EMS units must have the capability to communicate on very-high-frequency (VHF) air bands assigned for air traffic control, flight service, and local airport Unicom. In addition, the ability to communicate with ground EMS and public safety through VHF and ultrahigh-frequency (UHF) air bands is essential. Air use frequencies are accessible through standard aircraft communications transceivers, but EMS communications require additional radio equipment designed for this purpose. Additional needs include communication with the helicopter's base station, either on a locally assigned public-use frequency or a Federal Communications Commission (FCC)-assigned discrete frequency in the VHF air band. Another means of communication is aircraft 800-MHz radiotelephones that can access the surface telephone network. Communication over air band frequencies requires strict adherence to FAA communications guidelines and a radiotelephone operator permit from the FCC.[29]

Medical Equipment and In-Flight Monitoring

On-board medical supplies and equipment are typically tailored to the needs of a specific transport program and include medications, airway and ventilation supplies, dressings and bandages, IV fluids, immobilization devices, military antishock trousers, and stretchers.[86] The U.S. Department of Transportation (DOT), in conjunction with the American Medical Association (AMA), has published guidelines for onboard equipment for air ambulance operation[92] (Box 35-2).

Power

Most aircraft systems operate from 14 or 28 volts of direct-current (DC) power supplied by an engine-driven alternator or generator. This is not adequate to operate most medical devices, which require 110 to 120 volts of alternate-current (AC) power. Such devices cannot be used without an internal battery of sufficient charge to provide power for the duration of the mission, or unless a 110- to 120-volt AC power source is available from a power inverter, which must be installed under STC. Power inverters are common components of EMS helicopters and dedicated fixed-wing aircraft that have been retrofit with a custom-made air ambulance cabin configuration, but they may not be a standard component in fixed-wing aircraft that support a dual role and use an interchangeable corporate configuration.

Stretcher

The patient stretcher must be secured to the aircraft according to the requirements of FAR part 23.561 or 25.561 for seats: 3.0 g upwards (2.0 part 25), 9.0 g forward, and 1.5 g sideways (in this context g is a measure of force, 1.0 g being equal to the force generated when an object is subjected to an acceleration equal to the acceleration of gravity, e.g., 9.8 meters per second per second). For helicopters, the requirements are 1.5 g upward, 4.0 g forward, and 2.0 g sideways. Special configurations, especially those incorporating oxygen bottles and metal framework, may require an STC. Other guidelines (recommendations only) for stretcher configurations are for clear view and access to the patients with at least 30 inches (76.2 cm) of headroom and at least 12 inches (30.5 cm) of aisle beside the head. The stretcher should be at least 19 inches wide (48.2 cm) by 73 inches long (185.4 cm).[108] If the patient is positioned with head forward, the acceleration that occurs during takeoff of a fixed-wing aircraft may cause venous pooling in the lower extremities and transient hypotension. To prevent this, the patient can be positioned with feet forward.

Climate Control

The aircraft must be capable of maintaining a comfortable interior environment; about 24° C (75° F) is recommended. During summer months, the extensive glass area on a helicopter can produce a greenhouse effect, which may necessitate air conditioning for the comfort of both crew and patient.

Lighting

Lighting should be available to enable the crew to attend to the patient's needs but not interfere with cockpit operations. Curtains or other physical barriers may satisfy this need.

Box 35-2. U.S. Department of Transportation–American Medical Association Guidelines for On-Board Medical Equipment for Aeromedical Transport

BASIC MEDICAL EQUIPMENT RECOMMENDED FOR EACH FLIGHT

1/patient	Litter or stretcher with approved restraints
2/patient	Sheets
2/patient	Blankets
1/patient	Pillow with cover impervious to moisture
1/patient	Pillowcase
1 set	Spare sheets and pillowcase (if weight and space allow)
1 unit	Medical oxygen with manual control; adjustable flowmeter with gauge (0–15 L/min); attachment for humidification (NOTE: The oxygen unit must be attached to the aircraft in an approved manner. The amount of oxygen to be carried is determined by multiplying the prescribed flow rate by the length of time the patient must be on oxygen and adding a 45-minute reserve. The minimum amount of oxygen carried should be enough to supply one patient for 1 hour at 10 L/min. It may be necessary to carry a portable oxygen unit if oxygen is not available for patient transfer at some point in the flight.)
2 each	Oxygen masks in adult, child, and infant sizes
6	Connecting tubes
1	Oxygen key
1 unit	Portable suction with connecting tubes
2 each	Suction catheters (various sizes)
2	Tonsil suction tips
1 unit	Squeeze bag-valve-mask unit capable of receiving oxygen through an inlet, and delivering 80% to 100% oxygen through the mask; with masks in adult, child, and infant sizes (bags in adult and small child and infant sizes)
1 unit	Oxygen-powered, manually triggered breathing device (100-L/min flow rate)
1	Blood pressure cuff, sphygmomanometer
1	Stethoscope (NOTE: To record blood pressure readings, a Doppler or electronic stethoscope may be required if noise or vibration levels are high. An electronic unit must not cause electromagnetic interference on aircraft equipment.)
2 each	Oropharyngeal airways in adult, child, and infant sizes
1	Emesis basin
1	Urinal or bedpan or both
1/patient	Sound suppressors
1	Pneumatic antishock trousers with pressure relief valve
2	Cervical collars
2	20-gallon trash bags
1 box	Zipper-lock plastic bags or similar product
1	Flashlight, 2 D batteries or equivalent with spare batteries and bulb
2	Locking hooks (or other positive locking device for intravenous fluid containers)
1 qt	Drinking water
12	Paper cups

DRESSINGS AND SUPPLIES KIT, DESIGNED TO BE CARRIED ON EACH FLIGHT

4	Cardboard or air splints or equivalent in arm and leg sizes
12	Tongue depressors
2	Mouth gags or padded tongue depressors
1	Bandage scissors
4	Tourniquets
1 each	Rolls of adhesive tape, $^1/_2$, 1, 2, 3 inch
1 each	Rolls of paper tape, various sizes
4	Kling bandages or equivalent
1	3-inch elastic bandage
1	4-inch elastic bandage
4	Kerlix rolls or equivalent
2 pair	Sterile gloves
3	Petrolatum gauze
1 box	Adhesive bandages
6	Disposable surgical face masks
2 each	Syringes, 3, 5, and 10 mL (TB and insulin)
3 each	Needles, 18, 20, and 22 gauge
3 each	Needles, 19, 21 gauge, scalp/vein
2	Surgical dressings
24	Sterile gauze pads
6	Nonsterile gauze pads
2	Triangle bandages
2	Wrist restraints
2	Eye covers
1 roll	Aluminum foil, sterilized and wrapped
1	Large safety pin
2	Clinical thermometers
4	Airsick bags
12	Waterless towelettes
1 box	Tissues

MEDICATION AND INTRAVENOUS KIT, DESIGNED TO BE CARRIED ON EACH FLIGHT

2	Epinephrine HCl, 1:1000, 1 mL, prefilled syringe
2	Epinephrine HCl, 1:10,000, 10 mL, prefilled syringe with intracardiac needle
2	Aminophylline inj., 500 mg in 2-mL ampules
4	Atropine sulfate, 0.5 mg in 5-mL prefilled syringe
2	Diphenhydramine HCl, 50 mg/mL, 1-mL prefilled syringe
2	Dextrose, 25 g/50 mL, prefilled syringe
2	Intravenous injection sets with microdripper
2	Lidocaine HCl, 2 g/10 mL, prefilled syringe
3	Lidocaine HCl, 20 mg/mL, 5-mL prefilled syringe
6	Naloxone HCl, 0.4 mg/mL, 1-mL ampules
1	Nitroglycerin, 0.4 mg, sublingual tablets, 100
2	Digoxin inj., 0.5 mg/2 mL, ampules
4	Furosemide, 10 mg/mL, 2-mL ampules
2	Chlorpromazine HCl, 25 mg/mL, 1-mL ampules
6	Sodium bicarbonate inj., 3.75 mg/50 mL, prefilled syringe
2	Morphine sulfate, 15 mg/mL, prefilled syringe
1	Hydrocortisone sodium succinate, 100 mg/vial
1	Methylprednisolone sodium succinate, 1000 mg/vial

Box 35-2. Department of Transportation–American Medical Association Guidelines for On-Board Medical Equipment for Aeromedical Transport—cont'd

1	Plasma protein fraction, 250 mL with infusion set
2	Sterile water for injection, 20 mL
3	Diazepam, 5 mg/mL, 2-mL prefilled syringe
6	Alcohol swabs
1	Phenylephrine HCl, 0.25%, nasal spray
2	Ammonia inhalant solution, 0.5-mL ampule
2	Isoproterenol HCl, 1:5000, 1-mL ampules
1	Tourniquet
1	0.9% sodium chloride inj., 500-mL bag
1	0.9% sodium chloride inj., 250-mL bag
1	Lactated Ringer's inj., 250-mL bag
1	Lactated Ringer's inj., 500-mL bag
2	Lactated Ringer's inj., 1000-mL bag
3	Needles, 15 gauge, 1½ inch
1	Dextrose, 5% in water, 250 mL
1	Dextrose, 5% in water, 500 mL
1	Dextrose, 5% in normal saline, 250 mL
1	Dextrose, 5% in normal saline, 500 mL
1	Pressure pack or infusion pump
1 each	Drip tubing, regular and pediatric
2	Armboards
6	Alcohol wipes
1	Clean hemostat
1 each	Sterile hemostat, curved and straight
1	Nasogastric tube, 14 gauge
2	Sterile normal saline for injection, 20 mL
2 pair	Sterile gloves
1	Knife handle
1	Subclavian set
1	No. 15 blade
1	Intravenous infusion cuff
1 each	Rolls of tape, 1 and 2 inch

AIRWAY MANAGEMENT KIT, DESIGNED TO BE CARRIED ON EACH FLIGHT

1	Laryngoscope with curved and straight blades in various sizes; spare batteries and bulb
As required	Adapters for attaching endotracheal tubes to oxygen, etc.
1	Rubber-shod forceps
1	Magill forceps
1	Esophageal obturator airway with gastric suction capability
1	McSwain dart or Heimlich valve
1	Syringe, 60 mL
1	Needle, 14 gauge
1	Syringe, 10 mL
1 each	Rolls of adhesive tape, 1 and 2 inch
1	Viscous lidocaine HCl, 2%
1 tube	Surgical lubricant

BURN KIT, TO BE CARRIED WHEN REQUIRED

3	Normal saline, 1 mL in plastic container
1	Sterile burn sheet, 57 × 80 inch
5 packs	Xeroform gauze, 5 × 9 inch
1	Irrigating syringe, 50 mL
2 pairs	Sterile gloves
4	Kerlix rolls
2 packs	Fluffy gauze

POISON DRUG OVERDOSE KIT, TO BE CARRIED WHEN REQUIRED

1	Irrigation tray
1	Surgical stomach tube for lavage
1 each	Specimen bottles for urine, gastric, and miscellaneous
2 each	Stomach tubes, 14, 16, and 18 fr
1	Rubber stomach tube, no. 20
1 tube	Lubricant
1 box	Glucagon, 1 unit
2	Ipecac syrup, 30 mL
1	Physostigmine salicylate, 1 mg/mL, 2-mL ampules
1	Pralidoxime chloride, 1-g kit
1	Activated charcoal, 10 g

OBSTETRIC KIT, TO BE CARRIED WHEN REQUIRED

1	Disposable obstetric pack with sheets, cord clamps, DeLee suction, plastic bag, silver swaddler, sterile gloves
2	Oxytocin, 10 units/mL, 1-mL ampule
1	Episiotomy scissors
1	Ring forceps

PEDIATRIC KIT, TO BE CARRIED WHEN REQUIRED AND ALWAYS WITH OBSTETRIC KIT

1	Pediatric laryngoscope handle with blades
1 each	Pediatric endotracheal tubes with stylette, 2.5, 3, 3.5, and 4 fr
1	Pediatric Magill forceps
2	Bulb syringes
2	DeLee suction
2	Pediatric drip intravenous tubing
1 each	Feeding tubes, 3.5, 5, and 8 fr
1	Pediatric blood pressure cuff, sphygmomanometer

ADDITIONAL EQUIPMENT FOR TRAUMA PATIENTS, TO BE CARRIED WHEN REQUIRED

1	Scoop stretcher
1	Long backboard
1	Foley catheter set
1	Femur traction splint
1	Suture kit

ADDITIONAL EQUIPMENT FOR CARDIAC PATIENT, TO BE CARRIED WHEN REQUIRED

1 unit	Cardiac monitor with strip chart recorder
1 each	Spare ECG electrode for each lead
1	Spare roll of ECG recording paper
1 unit	Defibrillator with four pads and conductive gel (defibrillator may come as a unit with the cardiac monitor)
1	Rubber mat or other means of electrically isolating the patient from the aircraft
1	Cardiac board

ADDITIONAL EQUIPMENT FOR SPECIFIC PATIENTS, TO BE CARRIED WHEN REQUIRED

1 unit	Respirator capable of continuous ventilation, with ventilator, tubing, exhaled volume measuring device, set of tracheostomy endotracheal adaptors
1 unit	Incubator, with all equipment suitable for neonatal care

Modified from U.S. Department of Transportation, National Highway Traffic Safety Administration; American Medical Association Commission on Emergency Medical Services: Air Ambulance Guidelines. Washington, DC, DOT, 1981.

ECG, electrocardiogram; fr, French; inj., intramuscular injection.

TABLE 35-5. Oxygen Tank Specifications Endurance† (hr)

CYLINDER SIZE	WEIGHT (lb)	CAPACITY* (L)	ENDURANCE (hr) AT 2 L/min	ENDURANCE (hr) AT 10 L/min
D	11	356	2.0	1.0
G	32	1200	10.0	2.0
Q	70	2320	9.3	3.8
H and Q	150	6900	57.5	11.5

*At 21° C (70° F), 14.7 PSI. Capacity varies with ambient conditions.
†Estimated endurance; actual values may vary.
From National EMS Pilot's Association: Hosp Aviat 1986;5(6):17.

Suction

Suction is a requirement for ambulance operations in most states and should be available at all times during aeromedical transport. Integral suction as a custom retrofit system or a portable battery-powered device can be used.

Oxygen

In general, enough oxygen should be provided for the flight, plus a 45-minute reserve (IFR; 30 minutes VFR). In addition, oxygen should be carried to allow for ground handling time at either end. The amount of oxygen required can be obtained by multiplying the desired flow rate in liters per minute (L/min) by the total duration of transport and patient loading and unloading. Table 35-5 lists the capacities of various types of oxygen tanks and their respective weights. Some portable ventilators have a gas-driven logic circuit that requires additional air or oxygen. Electrically powered ventilators have a lower requirement for oxygen but carry the additional need for a power inverter. Most patients are transported with oxygen supplied by nasal cannulae (1 to 6 L/min). A single E-sized oxygen cylinder is adequate for short flights, although backup cylinders are usually carried. Patients intubated and maintained on 100% oxygen, as well as those ventilated on long flights, will exceed the capacity of an E cylinder quickly; several E cylinders or an H cylinder will be required.

Ventilators

On short flights, most patients can be bag ventilated manually, with the addition of a positive end-expiratory pressure (PEEP) valve as needed. Manual ventilation has drawbacks. Minute ventilation can rarely be precisely controlled, leading either to respiratory acidosis or more often to alkalosis. The patient's tidal volume limits may be exceeded, with resultant pulmonary barotrauma. More important, the medical attendant will be completely occupied with ventilation and is thus unavailable to perform other tasks. This takes on added importance if complex infusions are being administered or in-flight complications occur. The likelihood that manual ventilation will be unsatisfactory increases with the duration of transport, medical complexity of the patient, and severity of underlying lung disease.

Compact ventilators are available for use in the aeromedical transport environment.[14,53] The simplest are pressure ventilators with a timing valve mechanism that will deliver a predicted minute ventilation at a given rate and tidal volume adjustable to patient size. These require that the patient have normal airway compliance. If airway resistance increases, smaller tidal volumes will result, and tidal volumes usually cannot be varied independently from rate. Volume-cycled ventilators are superior and available in configurations in which tidal volume and rate can be varied independently. Oxygen bottles, a 50-PSI regulator, high-pressure gas lines, a patient breathing circuit, and source of humidification need to be present.

As mentioned, ET tube cuffs need to be appropriately monitored during flight to prevent overinflation resulting from decreased ambient pressure.

Infusion Devices

Several methods of IV infusion delivery are available in the aeromedical setting: gravity-feed microdrip or macrodrip tubing with the drip rate manually adjusted, gravity-feed automatic infusion regulators with a closed-loop drip-monitoring feedback mechanism controlling drip rate, and infusion pumps. If a pump is used over moderate or long transport distances, the internal battery power may be inadequate, necessitating an external source of power, usually an AC power inverter. With infusions that must be carefully maintained, an infusion pump is preferable. With frequent patient movement and manipulation, tubing can bend and kink, altering resistance to fluid flow. Air trapped in tubing (or in glass IV bottles) can expand with changes in altitude and increase or decrease the infusion pressure. Thus a reliable servo-controlled infusion system provides a margin of safety.

Monitor-Defibrillator and External Pacemaker

Combination monitor-defibrillators operate from internal batteries when 110 to 120 volts AC is not available. Other monitors capable of pressure monitoring from arterial lines or pulmonary artery catheters may be used, but these may have limited usefulness on flights of short duration, during which vibration and motion (turbulence) can introduce artifact and erroneous readings, as may occur aboard a helicopter. These devices may find a greater role with dedicated fixed-wing aircraft that frequently transport critically ill patients over long distances. Noninvasive blood pressure measurement is reasonably accurate in most patients, although these devices may have insufficient sensitivity.[65]

Potential hazards of defibrillation while airborne remain a concern, although trials support its safety.[25,47] Caution is still advised, and care should be taken to ensure that crew and aircraft systems are isolated from potential electrical contact (see "Common Aeromedical Transport Problems"). The pilot should be notified before defibrillation. It is unlikely that defibrillation will cause a problem with the aircraft, but if in a critical phase of flight (e.g., takeoff, landing), the pilot may delay the shock until the critical phase of flight is accomplished. Some external pacemakers have been approved for use in flight; however, care must be taken to ensure that the pacemaker and pads are properly shielded so that it will not interfere with the aircraft systems.

Oximetry

Pulse oximetry is often indicated for optimal patient care. Pulse oximeters use a colorimetric method, with placement of a soft probe over a fingertip, in a thin skinfold such as an earlobe, or against the conjunctiva.[1,53,96] They may be extremely useful in aeromedical transport, during which other methods to detect changes in respiratory status are difficult.

TABLE 35-6. Medical Attendants in Aeromedical Transport, 1988–1993

	PERCENTAGES				
	1988	1990	1991	1992	1993
Helicopter					
Medical Crew					
One attendant	8	3	3	2	2
Two attendants	92	97	97	98	98
Crew Configuration					
RN/paramedic	44	54	53	57	71
RN/RN	15	11	11	19	21
RN/physician	10	12	11	10	3
RN/other	17	15	20	7	2
Other	14	8	5	7	3
Regular-Duty Shift Length					
8 h	2	2	4	6	3
12 h	62	68	70	82	63
24 h	18	16	15	11	10
12 and 24 h	9	9	5	0	19
Other	9	5	6	1	5
Fixed-Wing Aircraft					
Medical Crew					
One attendant	17	17	10	4	5
Two attendants	83	83	90	96	95
Crew Configuration					
RN/paramedic	41	38	36	54	59
RN/RN	17	15	25	18	24
RN/physician	5	3	2	0	6
RN/other	22	35	32	18	9
Other	15	9	5	10	2
Regular-Duty Shift Length					
8 h	—	3	2	2	8
12 h	46	43	50	47	46
24 h	14	14	14	19	8
Other	40	40	34	32	38

RN, registered nurse.
From Cady G: Air Med J 1993;12:308.

Mechanical Resuscitators

Cardiac arrest resuscitation while airborne in a small cabin is difficult and physically demanding. In most instances, a standard medical crew of two will be completely occupied in performing chest compressions and ensuring ventilation. Additional tasks may be impossible. Therefore, a mechanical resuscitator may be used to prevent fatigue and free crew members for other tasks.[75] Mechanical resuscitators are gas-powered devices capable of providing ventilation and chest compressions automatically. Some models provide only chest compressions.

FLIGHT CREW

Crew Configuration

One of the continuing controversies in aeromedical transport involves crew composition (Table 35-6). The ideal crew composition varies considerably with the mission profile. When the aircraft is involved in a primary response to the accident scene, inclusion of an EMT may be beneficial. Transport of patients whose illness or injuries are complex or whose clinical conditions are extremely unstable may benefit from the presence of a physician. All aeromedical transport programs include one or more of the following providers in the transport medical crew.

Emergency Medical Technician–Paramedic

EMTs are increasingly a part of the aeromedical flight team. In 1993, 71% of rotor-wing transport programs reported using an EMT-paramedic as a member of the flight team, compared with 44% in 1988.[17] Paramedics vary in their level of training depending on the state in which they work but usually follow DOT guidelines, which include three levels of certification. EMT-basic provides basic ambulance, rescue, and first-aid skills. EMT-intermediate may include IV and intubation skills. The EMT-paramedic level involves such skills as intubation, IV techniques, medication administration, defibrillation, and arrhythmia recognition and treatment. For an aeromedical flight team member, additional training relating to the aeromedical environment is desirable.[53,94,108]

EMTs can be of particular value in operations that necessitate frequent interaction with ground EMS. In some regions, helicopter EMS service is integrated into the regional primary response network so that they arrive at the accident scene before ground units, and flight team members experienced in scene assessment and victim extrication are essential.

Flight Nurse

At least one flight nurse is part of almost all aeromedical transport programs; in 1993, 21% of rotor-wing programs reported using two flight nurses as the sole team members.[17] Critical care or emergency nursing experience is usually a prerequisite, with additional training that includes patient assessment, advanced cardiac life support, a trauma life support course, prehospital care skills, certain procedures such as ET intubation, advanced IV cannulation techniques, and in some cases, needle thoracostomy, venous cutdown, cricothyrotomy, and other specialized patient care activities. The flight nurse often is also a certified EMT.

Flight Physician

The experience and training of physicians involved in aeromedical transport depend on their role. Those who function as the on-line medical control physician communicate via radio or 800-MHz radiotelephone with the flight crew, monitor care, and give necessary orders. In the United States, physicians fly as a component of the flight team in a minority of aeromedical transport programs; in 1993, only 3% of all rotor-wing transport programs reported the routine use of a physician, compared with 10% in 1988.[17] In helicopter EMS operations, an emergency physician or trauma surgeon may be appropriate, whereas with fixed-wing transport of intensive care unit patients, an intensivist may be of value.

Physicians functioning in this role must have a current level of skill and expertise sufficient to address a wide range of clinical problems. They must also possess additional training relative to the airborne environment, including flight physiology, aircraft operations, and prehospital care (Box 35-3). Most important, they must function in this role with sufficient frequency so as to maintain their skills and remain safe and comfortable within the aeromedical setting. In doing so, they become an asset to the flight team rather than a distraction or liability.

Box 35-3. Specialized Training for Aeromedical Transport

Aviation physiology
Atmospheric pressure changes with altitude
Gas expansion with altitude
Changes in partial pressure of oxygen with altitude
Effects of motion and acceleration
Effects of noise and vibration
Changes in temperature and humidity
Aircraft safety
Aircraft systems and equipment operations
In-flight emergency procedures
Survival techniques
Patient extrication and immobilization
Patient loading and handling aboard an aircraft
Patient care techniques in the aeromedical environment
Respiratory support and ventilation aboard the aircraft
Pertinent Federal Aviation Administration regulations and procedures
Familiarity with the local EMS system
Radio communications skills and techniques
Hazardous materials response procedures
Record keeping and documentation in aeromedical transport
Preflight, in-flight, and postflight procedures
Clinical procedures
Cardiopulmonary resuscitation aboard the aircraft
Defibrillation aboard the aircraft
Intravenous cannulation
Endotracheal intubation
Tube thoracostomy
Needle thoracotomy
Cricothyrotomy
Central vein catheterization
Pericardiocentesis
Nasogastric tube insertion
Bladder catheterization
Antishock trouser application
Interosseous line placement
Umbilical vein catheterization

Studies during the late 1980s and early 1990s attempted to determine whether a physician crew member has an effect on the outcome of patients transported by helicopter.[7,9] Some studies concluded that a physician crew member had a positive impact on patient outcome, whereas others found no difference in outcome between similar cohorts of patients transported by flight crews with two nurses or a nurse and a paramedic.[43,88] The cost of using a physician crew member is substantially higher than that of a nurse–nurse or nurse–paramedic crew configuration. Some argue that this higher cost would be offset by the decrease in hospital stay or lost person-years that would occur if a physician were a standard member of the flight crew. With advanced training in critical procedures and treatment protocols, combined with on-line medical direction, a nonphysician flight crew usually functions as well as a crew that includes a physician. No objective evidence supports the benefit of a physician as a standard flight crew member in helicopter transports.

USAF CCAT teams, U.S. Army burn transport teams, and other military transport teams designed to move critically ill or injured patients over long distances will almost invariably have a specially trained physician (intensivist or burn specialist) as part of the team. Given the long duration of intercontinental missions and the difficulty of diverting transoceanic flights, the presence of a specialist physician onboard is vital to safe transport.[53,84]

Crew Member Stress

By its nature, aeromedical transport involves moving a gravely ill patient into an adverse environment with limited resources. Under these conditions, a medical crew of only two or three persons must perform complex tasks, solve difficult problems, and make life-or-death decisions. They must perform in a physically confining space that may be uncomfortable, and they must do so under time pressure and with little or no physical assistance. In some cases, rescuers' lives may be at risk. This scenario occurs in few other arenas of civilian medical care.

The term *stress* describes an array of adverse physiologic and psychological reactions that occur when a person perceives a threat to existence. Although stress may not diminish performance, it may be responsible for errors, faulty judgment, and uneven manual skill performance. It may also affect the physical and psychological health and satisfaction of the flight crew member.[18]

When measured during patient flights, the level of anxiety among aeromedical crew members was significantly higher than during a baseline period on the ground.[100] Factors that correlate with high in-flight anxiety levels include adverse weather conditions (e.g., low ceilings, high winds), severity of the patient's medical condition, complexity of illness or injuries, and the crew member's fatigue.

Efforts should be made to minimize stress among crew members. This includes frequent and adequate training; continuing education and feedback; adequate medical backup, including on-line medical direction, written protocols, and treatment guidelines that can aid in difficult decisions; a supportive rather than an intimidating or critical quality assurance program; adequate rest; and safe weather minimums. Both routine mission debriefing and critical incident stress debriefing should be an integral part of all transport programs.[71,72]

FLIGHT PHYSIOLOGY

Aeromedical care is different from ground-based care not only because of special equipment and the space-limited environment but also because of the hostile physical milieu.

Hypoxia and Altitude

The earth is blanketed by a sea of air. The troposphere lies in the first 9144 to 18,288 m (30,000 to 60,000 feet) and contains atmospheric moisture. Vertical convection currents, as well as a temperature decline with increasing altitude at a lapse rate of 2° C per 305 m (3.6° F per 1000 feet), occur here. Virtually all atmospheric weather occurs in this layer. Above this level lies the stratosphere, extending from 18,288 to 30,480 m (60,000 to 100,000 feet), where temperature remains relatively constant and no moisture or vertical convection currents exist.

Air exerts pressure on everything it contacts in an amount equal to the weight of the column of air above the point of reference. At sea level, the atmospheric pressure is 14.7 PSI or

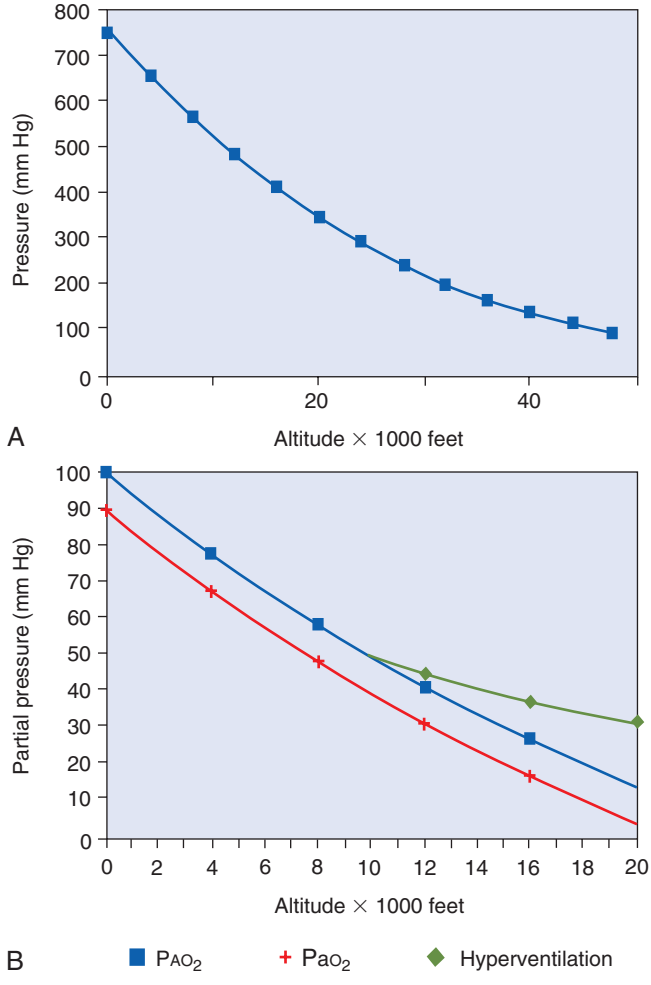


Figure 35-9. **A,** Atmospheric pressure vs. altitude. **B,** Alveolar (P_{AO_2}) and arterial (P_{aO_2}) oxygen tensions versus altitude.

760 mm Hg. As the person ascends, a progressively smaller air mass remains to exert weight, and the pressure diminishes. At 5486 m (18,000 feet), the atmospheric pressure is one half of that at sea level, and at 8534 m (28,000 feet), it is one third as great (Fig. 35-9). Similarly, under the weight of air, individual molecules tend to compact, so the density of air is also greatest at the surface and diminishes with increasing altitude.

These phenomena underlie most of the important physiologic consequences of flight. Air is composed of several gases, of which oxygen makes up about 21%, an amount that is relatively constant despite increasing altitude[28,53,76] (Table 35-7). Henry's law states that the quantity of gas that goes into solution depends on the partial pressure of that gas (and its solubility characteristics) as exerted at the air–water interface. The partial pressure of oxygen in alveolar air (P_{AO_2}) is determined by multiplying the fractional composition of oxygen in inspired air (F_{iO_2}) by the atmospheric pressure (barometric pressure, P_B) after the opposing vapor pressure of water (P_W, 47 mm Hg) has been subtracted. This is the basis of the alveolar air equation, as follows:

TABLE 35-7. Composition of Air

GAS	PERCENTAGE
Nitrogen	78.09
Oxygen	20.95
Carbon dioxide	0.03
Other gases	0.07
Water vapor	1–5 at sea level

From Del Vecchio RJ: Physiologic Aspects of Flight. Oakdale, NY, Dowling College Press, 1977.

$$P_{AO_2} = F_{iO_2} \times (P_B - P_W) - P_{CO_2}/R$$

where P_{CO_2} is the arterial carbon dioxide tension and R is the respiratory quotient (about 0.8). Arterial oxygen tension (P_{aO_2}) in normal individuals is within 10 to 15 mm Hg of the P_{AO_2}. With lung disease characterized by ventilation–perfusion mismatch, intrapulmonic shunting, or severe diffusion defects, the alveolar-arterial (A-a) oxygen gradient is much larger, and higher amounts of inspired oxygen are required to produce sufficient oxygenation of arterial blood. The atmospheric P_{O_2}, P_{AO_2}, and P_{aO_2} all decline with altitude (see Fig. 35-9). To some extent, P_{aO_2} can be maintained through hyperventilation as P_{CO_2} is reduced, but eventually P_{aO_2} will decline below 60% and hemoglobin will begin to desaturate greatly.

It is important during aeromedical transport to maintain hemoglobin saturation at or above 90%. Knowledge of the patient's preflight P_{aO_2} will enable a calculation of the patient's A-a oxygen gradient, which can then be subtracted from the P_{AO_2} calculated for the anticipated en route altitude (or cabin altitude in a pressurized craft) to yield the expected en route P_{AO_2}. Nomograms can be devised for this purpose (Fig. 35-10). If the en route expected P_{aO_2} is unacceptably low, supplemental oxygen is required. The F_{iO_2} required to maintain the P_{AO_2} at a given level can be calculated from the alveolar air equation as follows:

$$F_{iO_2} = (P_{AO_2} + P_{CO_2}/R)/(P_B - P_W)$$

Or, if $P_{CO_2} = 40$, R = 0.8, and $P_W = 47$ mm Hg, then:

$$F_{iO_2} = (P_{AO_2} + 50)/(P_B - 47)$$

This allows P_{AO_2} to be determined by adding the P_{aO_2} desired (the minimum acceptable is 60 mm Hg) to the known A-a oxygen gradient (calculated from the preflight blood gas). P_B can be estimated over the first 15,000 feet (4572 m) of ambient or cabin altitude as follows:

$$P_B = 760 - (23 \times \text{Alt})$$

where Alt is the altitude above sea level in thousands of feet. Transport cabin environments rarely exceed these altitudes because pressurized craft are usually capable of maintaining cabin pressure equal to 8000 feet (2438 m) or below at normal cruising altitudes. Nonpressurized craft must provide supplemental oxygen above 15,000 feet. Even a modest increase in F_{iO_2} is usually enough to maintain oxygenation under these

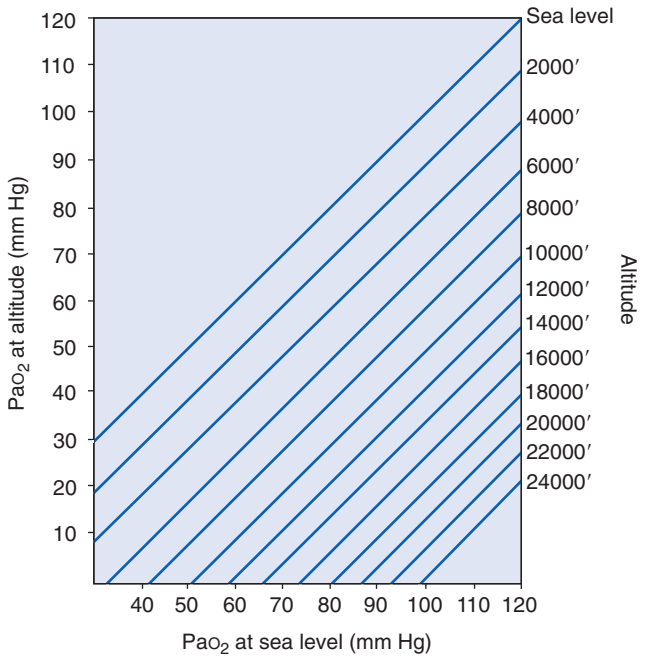


Figure 35-10. Arterial oxygen tension (Pa_{O_2}) at altitude versus Pa_{O_2} at sea level. Locate the sea-level Pa_{O_2} on the x-axis and intersect with the cruise altitude (on the diagonal). Read across to find the Pa_{O_2} at altitude (y-axis).

circumstances, unless a severe A-a oxygen gradient exists, in which case the addition of CPAP or PEEP may be necessary. The previous equation would predict that a Pa_{O_2} of 80 mm Hg at sea level on 40% oxygen would require that an Fi_{O_2} of 50% at 8000 feet be maintained. An oximeter can simplify monitoring of oxygenation; as altitude increases, a fall in oxygen saturation can be treated with increases in Fi_{O_2}.

Effects of Pressure Changes

Trapped Gas

Boyle's law states that the volume of a gas varies inversely with pressure. This means that trapped gases expand as an aircraft ascends to higher altitude (and lower pressure) and contract as it descends. The volume change can be determined from the following equation:

$$P_1 \times V_1 = P_2 \times V_2$$

Clinically, this is manifested by such alterations as expansion or contraction of air splints or military antishock trousers, changes in ET tube cuff size, expansion of bowel gas in cases of intestinal obstruction or ileus, expansion of pneumothorax air space, and expansion of air trapped in IV lines (or glass IV bottles) (Table 35-8). Certain precautions must be taken, such as the use of plastic IV bags and frequent monitoring of pressure cuffs.

Dysbarism

Decompression sickness occurs mainly in scuba divers that ascend too soon after a dive. Too rapid a decrease in ambient pressure allows nitrogen bubbles to form in the microcirculation, which may lead to ischemia and tissue damage (see Chapter 57). Care must be taken in the transport of an ill or injured diver to allow a surface interval of at least 12 hours before transport. An alternative is to attain at least a level D (PADI) dive stage, with a cabin altitude not to exceed 2438 m (8000 feet). If an individual must be transported abruptly after submersion, the transport must be conducted at the lowest possible safe altitude in a pressurized aircraft, to reduce the risk for decompression illness. Portable hyperbaric chambers have been developed, and several have been approved for flight in U.S. military aircraft. With one of these chambers, it is possible to transport a diving casualty by air while providing recompression therapy during flight (Fig. 35-11). This will usually require a large aircraft and several crew members to monitor the chamber during the flight. Sufficient pressurized breathing gas must be brought along to supply such chambers. Careful planning to ensure sufficient supply is critical.

TABLE 35-8. Clinical Manifestations of Trapped Gas Expansion

TRAPPED GAS LOCATION	CLINICAL MANIFESTATIONS
Intestinal lumen	Abdominal pain, distention
Pleural space	Pneumothorax, tension pneumothorax
Subcutaneous	Subcutaneous emphysema
Paranasal sinuses	Facial pain
Middle ear	Ear pain
Dental root	Tooth pain
Blood	Air embolism, decompression sickness
Air splints, antishock trousers	Compartment syndrome, ischemia
IV bottle and tubing	Flow rate increase
Blood pressure cuff	Tourniquet effect
Endotracheal tube cuff	Air leak, hypoventilation

Motion and Acceleration

Aircraft not only move through space in a rectilinear fashion, which cannot be detected by human senses in the absence of visual cues, but also rotate around three axes: longitudinal (roll), vertical (yaw), and horizontal (pitch) (Fig. 35-12). Motions around these axes are sensed by the semicircular canals located in the inner ear. Sensations from these organs are useful as an adjunct to visual cues. However, they may quickly lead to spatial disorientation, a phenomenon often experienced by individuals traveling in a turbulent environment without a visual frame of reference. For example, when an aircraft enters a bank to the right, the sensation may be initially correctly interpreted. However, after rollout of the stationary bank to a neutral position, a sensation of rolling into a left bank may be sensed. If uncorrected, apprehension may follow. The best remedy for this effect is to maintain a visual reference to the correct position.

Vestibular stimuli, especially in turbulence with limited or no visual frame of reference, may result in motion sickness, manifested most often as nausea or vomiting. Affecting both patients and flight crews, motion sickness can be counteracted with antiemetics such as antihistamines (e.g., dimenhydrinate, 50 mg orally) or phenothiazines (e.g., prochlorperazine, 10 mg orally); however, these may produce sedation and are potentially haz-

A

B

Figure 35-11. **A,** Hyperlyte portable hyperbaric chamber being inflated and rigged for a dive on board a U.S. Air Force C-17 aircraft. Note the gas lines feeding into the near end of the chamber. At the time this photograph was taken, the aircraft was flying at 15,000 feet. B. Hyperlyte portable hyperbaric chamber being secured to the deck of a U.S. Air Force C-17 aircraft. (**A,** Photo courtesy of Robert C. Allen.)

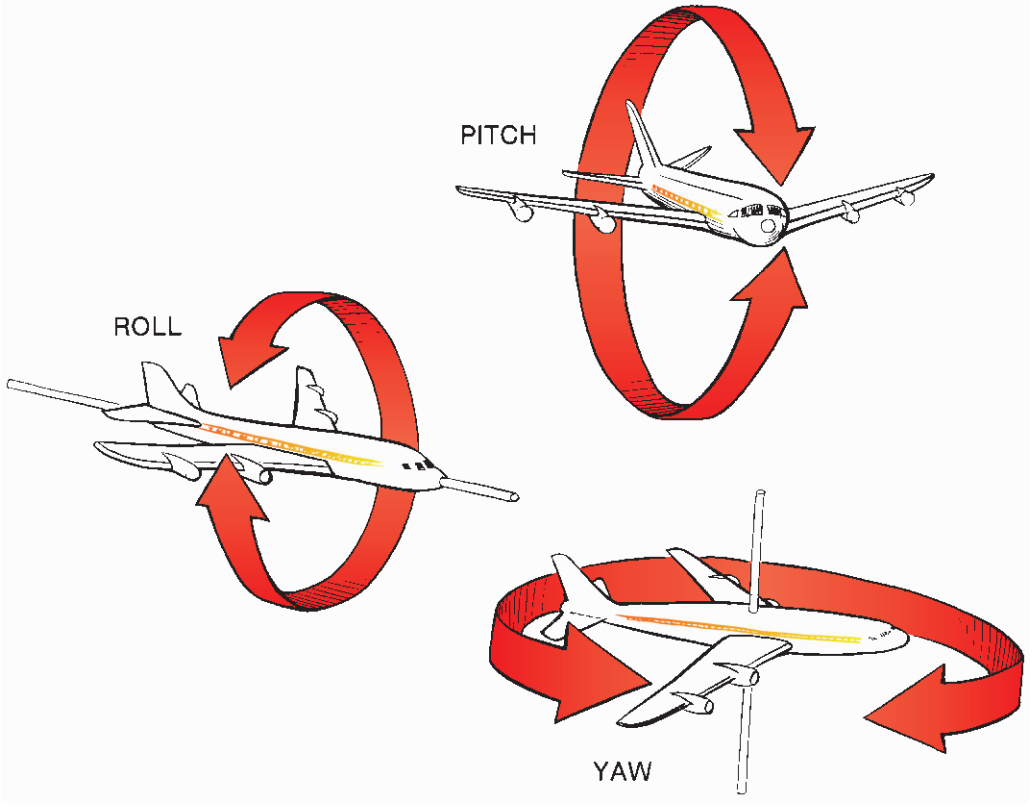


Figure 35-12. Axes of movement in aviation.

ardous in flight. Transdermal scopolamine patches applied behind the ear have been used as effective prophylaxis for nausea and vomiting during flight.

Noise and Vibration

Noise and vibration are components of all aircraft environments, especially helicopters. The most obvious impact of noise is on communications within the cabin, particularly with the patient, who is least likely to have a headset and access to the aircraft's intercom. In addition, the patient's breath sounds are difficult if not impossible to hear, and thus other means to identify changes in respiratory status, such as pulse oximetry, must be employed.

Headsets are essential for effective communication among flight crew members aboard a helicopter, although they may not be necessary aboard larger fixed-wing aircraft. Noise may lead to permanent defects in auditory acuity if exposure is prolonged or recurrent. Veteran pilots frequently demonstrate 10- to

Box 35-4. Pretransport Preparations

SCENE RESPONSE
Airway secured
Stabilization on a rigid spine board with cervical immobilization device, neck rolls, and tape
Two large-bore intravenous lines
Antishock garment applied
Landing zone selected and secured

INTERHOSPITAL TRANSPORT
Airway secured
Stabilization on a rigid spine board with cervical immobilization device, neck rolls, and tape
Two large-bore intravenous lines
Tube thoracostomy for pneumothorax or hemothorax
Bladder catheterization (if not contraindicated)
Nasogastric catheterization (if not contraindicated)
Lactated Ringer's solution hanging
Typed and cross-matched blood if available
Extremity fractures splinted (traction splinting for femur fractures)
Copies of all available emergency department records and laboratory results, including a description of the mechanism of injury

20-dB reductions in high-frequency auditory acuity. Noise and vibration may also contribute to stress and fatigue.

COMMON AEROMEDICAL TRANSPORT PROBLEMS

Pretransport Preparation

Once the decision is made to transport a patient by air and the appropriate aeromedical service is contacted, preparations must be made to ensure patient safety and comfort and to aid the flight crew in patient care. A study of the causes of ground delays in a rural interhospital helicopter transport program found that on arrival of the flight team, 31% of patients required minor interventions (insertion of IV line or nasogastric tube, blood transfusion, bladder catheterization, military antishock trousers application) before takeoff, and 33% required major interventions (ET intubation, tube thoracostomy, central venous access).[62] When no intervention was required, the mean ground time was 31.2 minutes, compared with 57.4 minutes when one or more major interventions were required.

To minimize delays, pretransport preparations should be made for victims of acute trauma (Box 35-4).

Patient Comfort

Motion, vibration, noise, temperature variations, dry air, changes in atmospheric pressure, confinement to a limited position or backboard, and fear of flying may cause patient discomfort.

Patient Movement

Patient handling and movement can contribute to morbidity and mortality in unstable patients.[110] All transported patients should be adequately secured to the stretcher with safety straps to prevent sudden shifting of position or movement of a secured fracture. During transport from the ground to the aircraft cabin, attempts should be made to limit sudden pitching of the stretcher. DOT guidelines recommend design of cabin access such that no more than 30 degrees of roll and 45 degrees of pitch may occur to the patient-occupied stretcher during loading.[70] The stretcher, in turn, should be adequately attached to the floor.

Motion sickness in the patient may be treated with an antiemetic such as promethazine (25 mg orally, intravenously, or intramuscularly). Scopolamine disks are useful for prolonged flight and do not require parenteral or oral administration, although their antiemetic effects are not always uniform and may not occur until 4 to 6 hours after application. They may be best used to decrease motion sickness in the flight crew.

Noise

Noise can be avoided with hearing protectors, which are devices similar to headphones but without internal speakers. Inexpensive hearing protectors are available as deformable foam ear plugs. In some cases, headphones may be used in an awake patient if the crew wants the patient to communicate on the intercom system.

Eye Protection

When a patient is loaded on or off a helicopter with the rotors turning, the patient's eyes must be protected. Serious eye injuries can result from debris blown into the air. Lightweight sky diver goggles ("boogie goggles") are effective and inexpensive. The eyes must be protected even if the patient is unconscious. Taping temporary patches over the eyes is also effective.

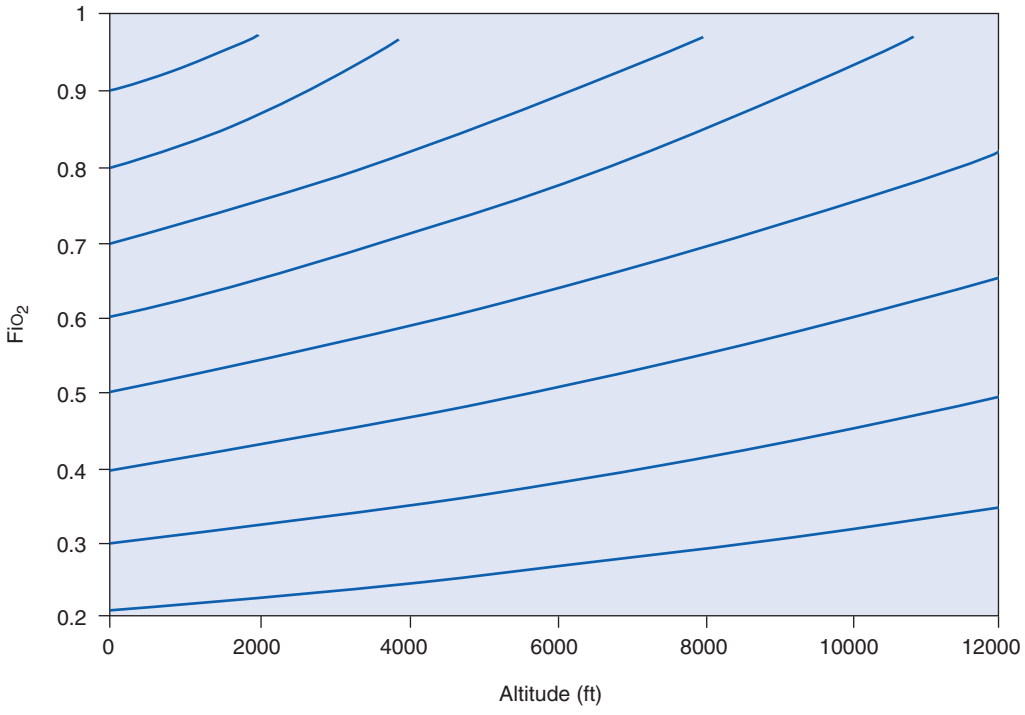
Respiratory Distress

Patients with respiratory disease or distress should have immediately treatable conditions addressed before takeoff. ET intubation is essential if airway patency is threatened or if adequate oxygenation cannot be maintained with supplemental oxygen. It is better to err on the side of caution when making a decision about a patient's airway. During flight, it is easier to treat restlessness in an intubated patient than airway obstruction or apnea in a nonintubated patient.

Nearly all seriously ill patients should receive supplemental oxygen. FiO_2 should be increased with increasing cabin altitude to maintain a stable PO_2 (Fig. 35-13). Small, portable pulse oximeters are readily available and should be used to monitor oxygen saturation while in flight. When oxygen saturation monitoring is unavailable and pretransport arterial oxygen content unknown, 100% oxygen may be administered throughout the flight to ensure adequate oxygenation. Patients with chronic lung disease who are prone to hypercapnia may experience deterioration in condition if the hypoxic drive is eliminated. In these patients, the least oxygen necessary to maintain saturation above 90% is advisable; this amount may be estimated in advance or calculated from the alveolar air equation. Close in-flight monitoring is essential, preferably by continuous pulse oximetry. Finally, altitude changes may affect ET cuff volume; thus, cuff pressure must be checked frequently throughout the flight.

Cardiopulmonary Resuscitation and Cardiac Defibrillation

CPR in an aircraft is difficult. The rescuers must perform several tasks simultaneously while ventilating the lungs or compressing



P_B	Altitude (ft)	Fio_2								
	0	0.21	0.30	0.40	0.50	0.60	0.70	0.80	0.90	1.00
760	2000	0.23	0.32	0.43	0.54	0.65	0.76	0.86	0.97	PP
707	4000	0.25	0.35	0.47	0.59	0.70	0.82	0.94	PP	PP
656	6000	0.27	0.38	0.51	0.63	0.76	0.89	PP	PP	PP
609	8000	0.29	0.41	0.55	0.69	0.83	0.97	PP	PP	PP
564	10000	0.31	0.45	0.60	0.75	0.90	PP	PP	PP	PP
523	12000	0.34	0.49	0.65	0.82	0.98	PP	PP	PP	PP
483	A-a:	15	79	150	222	293	364	435	507	578

Calculated from the alveolar air equation under standard temperature and pressure conditions. *PP*, Positive pressure required; P_B, barometric pressure (mm Hg); *A-a*, alveolar–arterial oxygen gradient (mm Hg).
X axis: en route altitude
Y axis: Fio_2 required to maintain Po_2 = 90 mm Hg
To calculate the Fio_2 necessary to maintain Po_2 = 90 mm Hg at a specific altitude, choose the value on the Y axis closest to that necessary to maintain Po_2 = 90 mm Hg at sea level (altitude = 0 ft) and follow the corresponding line across to the new altitude (X axis). The proper Fio_2 is the Y value corresponding to the new X value.

Figure 35-13. Fraction of inspired oxygen required to maintain oxygen tension at 90 mm Hg (varying with altitude).

the chest, all in a physically confining space. The crew must be familiar with modifications in technique.[49] As previously mentioned, there should be no concern with airborne defibrillation if all electronic navigational equipment on the aircraft has a common ground, as mandated by the FAA standards. Despite cramped quarters and sensitive electrical equipment, defibrillation can be safely performed in all types of aircraft currently used for emergency transport utilizing standard precautions routinely used during defibrillation on the ground.[25,111] In the interest of safety, however, it is best to notify the pilot before performing defibrillation.

Patient Combativeness

Patients may be combative to the point that they pose a threat to the safety of the flight and crew. An uncontrollable patient may cause sudden shifts in aircraft balance or may strike a crew member or important flight instruments or equipment. Such patients should be properly restrained in advance. If sedation is necessary, a careful neurologic examination documented beforehand is essential. Useful agents include diazepam or shorter-acting agents, such as midazolam. Paralyzing agents, such as pancuronium, vecuronium, and succinylcholine, have the advantage of not altering the sensorium, but they require airway control with ET intubation.[103] In addition, it is necessary to sedate a patient who is paralyzed to facilitate intubation and transport.

Endotracheal Intubation

ET intubation may be difficult to perform while airborne, especially in a confining cabin, and should be done before departure if possible. This is especially true in trauma victims with head injuries and in burn victims who have carbonaceous sputum or hoarseness. Special techniques are available to supplement standard methods of intubation, including a lighted stylet, ET tubes with controllable tips, intubating laryngeal mask airways, and digital intubation. Sedation or pharmacologic paralysis may be necessary. Of 106 aeromedical transport programs in the United States that reported using neuromuscular blocking agents, 39 use them to facilitate intubations, and 67 use them once a patient is intubated to manage combativeness and ensure airway patency.[92] Induction of paralysis before intubation in the aeromedical setting is controversial. One study reported a 96.6% success rate using succinylcholine to facilitate intubation, with 3.4% of patients requiring an emergency surgical airway.[74] Besides the need for surgical airway if intubation is unsuccessful, concerns exist about cervical spine manipulation during intubation in the paralyzed patient, unrecognized esophageal intubation in a nonbreathing patient, and the relative contraindications to the use of succinylcholine in certain patients. As shorter-acting nondepolarizing paralytic agents (e.g., mivacurium) are developed, this adjunct to airway and combativeness control in the aeromedical setting will be studied further.[57]

In some flight programs, nonphysician crew members are taught to perform emergency cricothyrotomy. Although occasionally lifesaving, this procedure can be difficult to perform and should be undertaken only as a final method to secure an emergency airway.

Thrombolysis

The air transport of patients with acute myocardial infarction often involves thrombolytic therapy. Because bleeding is a major adverse effect of thrombolytic agents, one study investigated whether air transport resulted in a higher incidence of bleeding complications compared with a similar cohort of patients given thrombolytic drugs who were transported by ground ambulance. The study concluded that helicopter transport of patients with acute myocardial infarction after initiation of thrombolysis is comparatively safe and without a clinically significant increase in bleeding complications.[37]

Flight Safety

Because aeromedical transport involves medical care delivered in a hostile environment, the patient and crew are at risk for injury or death in the event of a mishap. Flight crew training must emphasize safety. The pilot is ultimately responsible for the safety of the aircraft's occupants and is trained not only to operate the aircraft skillfully and safely but also to provide necessary safety instructions and guidance to crew members and passengers. Safety practices vary depending on the type of aircraft but include common guidelines.

Approaching the Aircraft

Helicopters with turning rotor blades must be approached only from the front and sides and only while under pilot observation (Fig. 35-14). The tail rotor must be given wide berth, especially on helicopters with rear doors. It is advisable to station a crew member in a safe position to direct approaching individuals away from the tail rotor. Shutting down the helicopter's engines completely, when the situation allows, is prudent before patient loading and unloading. Approaching in a crouched position minimizes the risk for contact with the rotor blades should a sudden gust of wind or movement of the aircraft cause them to dip. Loose clothing and debris should be secured (Box 35-5).

Fixed-wing aircraft should be approached with similar precautions regarding propellers. This is especially important in aircraft with access doors in front of the wing and engine nacelles. Engine shutdown on the side of entry enhances safety of loading and unloading.

Safety Belt Use

The use of safety belts (preferably with shoulder harnesses, especially in helicopters) is an important safety measure. Certain patient care activities (e.g., ET intubation, CPR), however, may be impossible to perform with safety belts secured. The design and selection of aircraft and interior configurations should allow maximal access to the patient with the crew members properly restrained. Throughout the flight, the crew members and patient should remain restrained as much as possible in smooth air and at all times in rough air. Movement inside the cabin affects aircraft balance. An aircraft loaded near its aft CG limit may exceed its limits if a crew member moves to a new position within the cabin. Changes in position should be preceded by consultation with the pilot. Light aircraft are sensitive to turbulent air, and appropriate precautions must be taken to avoid being injured from sudden motion.

Proper Use of Aircraft Equipment

Crew members must be familiar with all aircraft equipment they may be required to operate in flight or in an emergency. This includes all aircraft doors, fire extinguisher, communications equipment, oxygen equipment, and electrical outlets. In addition, the crew must be familiar with emergency shutdown pro-

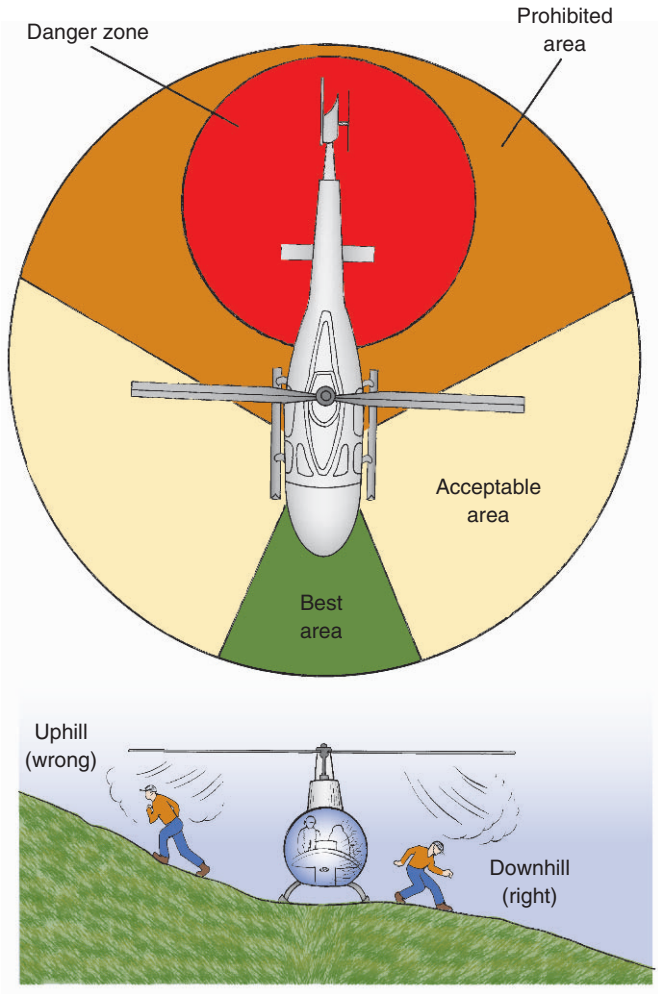


Figure 35-14. Helicopter safety. **A,** Safe approach zones. **B,** The proper way to approach or depart a helicopter.

Box 35-5. Helicopter Safety

DO:
Approach and depart downhill.
Use crouched position.
Approach after visual contact and approval from pilot.
Await direction of flight crew.
Approach from the front or the sides.
Secure area first of people and then of loose debris.

DON'T:
Approach or depart uphill.
Use tall intravenous poles or other objects.
Use loose sheets or clothing.
Smoke tobacco within 50 feet.
Run near the aircraft.
Drive a vehicle within 30 feet.
Shine headlights or flashlights toward the aircraft.

cedures. Finally, before takeoff, door security must be confirmed by a crew member familiar with operation of the door.

In-Flight Obstacle Reporting

An extra pair of eyes can be invaluable to a pilot in a busy airspace or on a scene approach complicated by trees and electrical or phone wires. Primarily important in VFR conditions, assistance with obstacle identification can enhance the safety of the mission; however, flight should not occur under conditions in which obstacle reporting by a crew member is essential to safety because the person must then divide attention between patient care and obstacle reporting.

Ground Coordination and Control

Enthusiastic rescue personnel or curious onlookers may approach the aircraft in a hazardous manner. Flight crew members must be able to communicate with ground units during the landing phase to ensure adequate scene preparation; they may be required to perform crowd control while on the ground. This requires directing individuals away from the rotor blades, propellers, or other hazardous equipment at the scene. If loading or unloading the patient while the rotors or propellers are still turning ("hot loading" or "hot off-loading") is necessary, special precautions must be undertaken for ground crews, the flight crew, and the patient.

Emergency Procedures

All crew members should memorize and routinely practice emergency procedures. These procedures should address in-flight fires, electrical failures, loss of pressurization, engine failure, emergency landing with and without power, precautionary landing away from an airport, and other in-flight emergencies.

Survival

An emergency or precautionary landing away from an airport necessitates survival before rescue arrives. Under adverse environmental conditions and with injured victims, survival may depend on specific actions by the crew. The crew should be proficient in emergency egress from the aircraft, including escape after crashes and water landings, especially in helicopters. After water landings, helicopters usually roll inverted and sink rapidly. Helicopter "dunker" training is required for all military helicopter crews. The crews are strapped into a simulated helicopter fuselage and lowered rapidly into a pool, simulating a semicontrolled water landing. The fuselage then rolls inverted, and the crew must open emergency exits and egress the simulator. This type of training has been shown to save lives in helicopter water crashes. All crew members should be trained in the use of emergency signaling devices, such as emergency locator transceivers (ELTs), flares, signal fires, and ground emergency signals. Survival skills should be taught to all crew members, including advanced first aid, water survival, building emergency shelters, fire starting, and obtaining water and food from the environment.

Aeromedical Accidents

Attention continues to focus on EMS helicopter accidents. Statistics from 1986 estimated a rotor-wing accident rate of 17.65, with 5.88 fatalities per 100,000 transports.[38] Aviation accident rates are typically reported in relation to flight hours. EMS fatalities, therefore, amounted to 6.0 per 100,000 flight hours, compared with 3.3 for the helicopter industry in general. The EMS

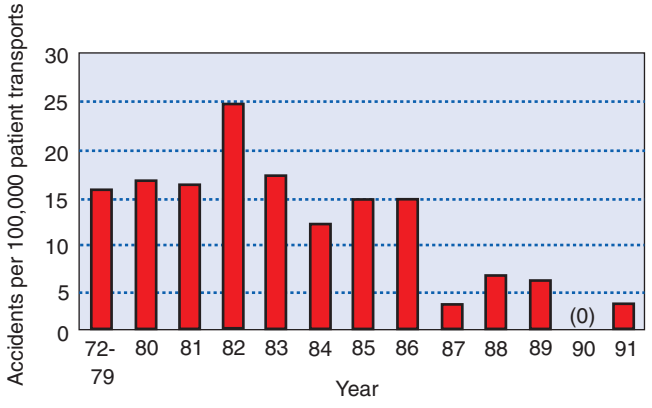


Figure 35-15. Aeromedical helicopter accident rate, 1972 to 1991. (From Preston N: J Air Med Transport 1992;11:14.)

TABLE 35-9. Major and Primary Causes and Severity of Aeromedical Accidents, 1972–1989

	PERCENTAGES		
	1972–1985	1987–1988	1989
Major Cause			
Weather	30	30	17
Engine failure	18	9	0
Obstacle strike	18	10	33
Control loss	9	20	17
Other	25	40	33
Primary Cause			
Pilot error	64	60	83
Mechanical failure	30	30	0
Unknown	6	10	17
Accident Severity			
Fatal	36	50	67
Injury	28	30	17
Damage only	36	20	17

From Collett HM: J Air Med Transport 1990;9(2):12.

TABLE 35-10. Effect of Weather on Accident Seriousness

SEVERITY OF ACCIDENT	WEATHER RELATED	PERCENTAGE
Fatal	14/21	67
Injury	3/17	18
Damage only	1/32	3

From Collett HM: Hosp Aviat 1986;5(11):15.

TABLE 35-11. Environment of Fatal Accidents

VISIBILITY	ACCIDENTS	PERCENTAGE
Day visual meteorological conditions	3	14
Night visual meteorological conditions	4	19
Day marginal	4	19
Night marginal	10	48
TOTAL	21	100

From Collett HM: Hosp Aviat 1986;5(11):15.

rate subsequently declined, however, with 3.0 accidents per 100,000 transports reported in 1991[87,101] (Fig. 35-15).

Sixty-four to 84% of EMS accidents result from pilot error, about 23% from mechanical causes, and 3% from unknown causes.[21] Of accidents caused by pilot error, adverse weather was a contributing factor in 67%[19] (Table 35-9). In all, two thirds of fatal weather-related accidents occur at night, and 86% of all fatal accidents occur at night or in marginal weather conditions[22] (Table 35-10). Only 35% to 40% of all EMS helicopter flights occur at night. The most common phase of flight for weather-related accidents was en route (86%), with 14% occurring on departure and none on approach (Table 35-11).

Only 5% of fatal EMS helicopter accidents occur in flights to or from the scene, although such flights account for 24% of EMS helicopter missions.[23] When scene-related accidents occur, they result in fatalities 9% and injuries 35% of the time, suggesting a low-energy impact versus an en route crash. Causal factors related to scene accidents include wire and obstacle strikes (70%), loss of control (18%), and mechanical failure (12%). The landing phase is involved in scene-related accidents in 41% of cases, whereas the takeoff phase is involved in 59%. Because 40% of scene-related accidents occur at night and about 40% of helicopter EMS flights occur at night, scene accidents do not appear more hazardous at night than in the day. Single-engine helicopters have more often been involved in engine failure accidents (77%) than have twin-engine craft (the two types are about equal in number in the EMS industry).

Recommendations for enhancing safety include instituting stringent guidelines to limit flights at night or in adverse weather, increasing pilot proficiency training, and reducing pilot fatigue and workload factors. Decisions on the appropriateness of air transport regarding utilization and weather should be based on general protocols that are not subject to the emotional turmoil of a medical crisis.[12] The trend in the industry has been toward twin-engine helicopters for an increased margin of safety from engine failure accidents.

In addition, for dedicated EMS helicopter flights, a statistically strong relationship exists between the ability to fly under IFR and a lower accident rate.[64] The AAMS considered the question of whether all helicopters should be required to have IFR capability when it published its voluntary standards for rotor- and fixed-wing aircraft. This was not mandated because of the tremendous expense and because many programs were unlikely to comply.[66]

Ground to Air Signaling

It is best to have radio communication between the ground party and the helicopter crew. This may not be possible, however, and hand signals may be needed to communicate. Standard hand signals are used by military rescue personnel for communication between a deployed rescue swimmer and the helicopter (Table 35-12). These same signals can be used while

TABLE 35-12. Swimmer to Helicopter and Ground to Air Signals

INTENTION	ACTION
Deploy medical kit	Arms above head, wrists crossed
Situation okay	Thumbs up
Lower rescue cable	Arm extended over head, rescue device attached with fist clenched
Lower rescue cable	Climbing-rope motion without rescue device with hands attached
Helicopter move in/out	Wave in/out with both hands
Cease operations	Slashing motion across throat
Deploy litter	Hands cupped, then arms outstretched
Personnel secured, raise	Vigorously shake hoist cable or thumb up; vigorous up motion with arm
Team recall	Circle arm over head with fingers skyward

on land. To acknowledge the signals, the hoist operator gives a "thumbs-up" sign or the pilot flashes the rotating beacon.

Landing Zone Operations

The ideal helicopter landing zone is a wide, flat, clear area with no obstacles in the approach or departure end. Vertical landings and take-offs can be done, but it is safer for the helicopter to make a gradual descent while flying forward. Higher altitudes and higher temperatures require larger landing zones. The center of the landing zone can be marked with a V, with the apex pointing into the prevailing wind. Any obstacles can be marked with brightly colored, properly secured clothing.

The size of the landing zone depends on the weather conditions, type of helicopter involved, altitude, temperature, and types of obstacles in the area. Small helicopters such as the Jet Ranger can usually land safely in a 60- × 60-foot landing zone. Larger helicopters such as the Bell 412 require a 120- × 120-foot zone.[112] Large military helicopters may require even greater landing zones. In general, pilots prefer the largest, flattest piece of ground they can find. The condition of the ground (e.g., loose snow, dust, gravel) should be communicated to the pilot before the final approach. Before the helicopter lands, all loose clothing and equipment should be secured. During approach, no personnel or vehicles should move on or near the landing zone.

Once the helicopter is on the ground, it must be approached only from the front and side, and then only while under direct observation of the pilot. The aft portion of the aircraft and areas around the tail rotor must be avoided at all times. Some helicopters (e.g., BK-117) have rear doors for loading and unloading patients, and ground personnel should wait for directions from the crew before approaching the rear area.

If the ground is uneven or slopes, all personnel should approach and depart from the helicopter on the down-slope side. It is safest to load the patient into the helicopter with the engines off and the rotors stopped ("cold load"). If the patient must be loaded with the engines on and blades turning ("hot load"), eye and ear protection should be worn by all personnel approaching the helicopter, including the patient. A safety person should be assigned to prevent anyone from inadvertently walking toward the tail rotor.

Once the patient is loaded, all personnel should leave the landing zone, take cover, and stay in place until the helicopter has departed. It is best to be off to the side, not directly in the takeoff path.

Hoist Operations

If the helicopter is not able to land and has a rescue hoist installed, hoist operations may be the only means of evacuating the patient. In most circumstances, a helicopter crew member rides the hoist down to the site to rig the survivor into the rescue device and to oversee the hoist operation, using the following guidelines:

- It is critical that personnel on the ground not touch the hoist, rescue device, or cable until after it has touched the ground (or water). Helicopters can build up a very powerful static electricity charge that will be grounded through whatever the hoist touches first. This has been known to knock rescuers and survivors off their feet.
- Once the rescue device and cable have touched the ground, put the patient into the rescue device, taking care to keep the hoist cable clear of all personnel. Do not allow the hoist cable to loop around any personnel or around the rescue device because serious injury is possible when the cable slack is taken up.
- Make sure that the patient is properly secured in the rescue device, with all safety straps tightened.
- When the patient is secured, move away from the rescue device and signal "up cable."
- If the rescue device is a basket (Stokes) litter, use a tag line with a properly installed weak link to prevent the litter from spinning during the hoist.

Night Operations

Night helicopter rescue operations are considerably more dangerous than daylight operations. In most cases, it is best to delay helicopter insertion or extraction operations until daylight. If this is not possible, however, night missions can be done safely with careful planning and coordination between the helicopter and ground party. The same rules of daylight helicopter operations apply to night operations, but with extra care taken to ensure that all personnel understand their roles.

In night operations, it is virtually mandatory to have radio communications between the ground and the helicopter. "No-comm" night operations should be attempted only by personnel specially trained and experienced in these techniques. The landing zone should be clearly marked and the pilot allowed to make the approach. All personnel should stay clear of the landing zone until the pilot has made a safe landing.

Personnel approaching the landing zone should have a small light or reflective material attached to their outer clothing so they can be clearly seen. The minimum number of people should approach the helicopter, and a safety observer is mandatory to keep the ground team together and clear of the tail rotor and rotor blades.

The landing zone should be as large as possible, preferably at least 50% larger than a daylight landing zone. Any obstacles should be clearly marked with light-colored streamers, small lights, or even light-colored clothing. The landing zone can be illuminated with flashlights at the corners, with another flashlight at the center point. These flashlights should be pointed at the ground, not into the air; flashlights pointed at the helicopter during landing and takeoff may distract or momentarily blind the crew. If flashlights are not available, small fires can be used to illuminate the edges of the landing zone, although the helicopter can scatter burning embers for many meters.

If crew members are using night vision equipment, lights must never be flashed at the helicopter. Even the amount of white

light from a small flashlight may be sufficient to overload the night vision equipment, functionally blinding the crew.

Dispatch and Communications

The dispatch center is the focal point for communications during aeromedical transport operations. Dispatchers receive incoming requests for service; obtain necessary information relative to the launch decision; coordinate the interaction between essential parties; "scramble" the flight crew; assemble and maintain necessary information regarding destination, weather, local telephone numbers, and frequencies; follow the progress of the flight; input data into the system database; and communicate with ground EMS units and hospitals. Communication may occur through a combination of methods: land telephone lines into a dispatch switchboard, hospital–EMS net transceiver, discrete frequency transceiver (communications with aircraft), or walkie-talkie radios. Familiarity with the EMS system and EMS communications is essential for successful dispatch.

Flight following is an important part of aeromedical safety and involves tracking the position of the aircraft during a mission by plotting the location according to reports from the pilot at 10 to 15 minute intervals. If an accident or in-flight emergency occurs, the dispatcher is soon aware and can initiate SAR to a precise location, which enhances the chances of survival.

APPROPRIATE USE OF AEROMEDICAL SERVICES

Aeromedical transport combines skilled treatment and stabilization capability with rapid access to definitive care, but not without risk, and at high cost ($1 to $2 million per year for a program, or about $2000 per transported patient, which is about 400% higher than ground transport).[15] However, the comparative risk of aeromedical transport must be placed in perspective against the risk for patient death from nonreferral or from less timely ground transport with limited medical capability en route.

Although not proved, advanced provider skill levels during prehospital care are considered beneficial, especially in severely ill or injured patients.[54] In rural and wilderness environments, advanced life support services may be made more readily available by EMS helicopters. This is especially true in areas that are difficult or impossible to reach by ground.

The speed of access to definitive care is another consideration in choosing the mode of transport. In isolated rural or wilderness locations, a helicopter may be the only means of expedient access. Prolonged victim extrication allows time for a helicopter to arrive at the scene, decreasing total transport time and thereby increasing the advantage of helicopter transport.

Patient comfort also must be considered, especially on long transports over rough roads. Although a helicopter moves in three dimensions, fore-and-aft acceleration is usually steady, without the starting and stopping motions present during ground transport. However, helicopters typically travel within 914 m (3000 feet) of the ground's surface and are more subject to turbulence than are high-flying fixed-wing aircraft.

Whether aeromedical transport reduces mortality when compared with ground transport has not been determined definitively. An uncontrolled national multicenter study of trauma patients transported by helicopter showed a 21% reduction in mortality from that expected based on predictions from the Trauma Score–Injury Severity Score (TRISS) methodology and national normative trauma outcome data.[10]

A similar study using the TRISS methodology compared actual mortality with helicopter versus ground transport and showed a 52% reduction from expected mortality when patients were transported by air, compared with no reduction in expected mortality when transport occurred by ground.[7] Another study using TRISS methodology found a benefit of aeromedical transport only in patients with severe trauma (a probability of survival less than 90%).[13]

In 1990, the AAMS issued a position paper on the appropriate use of emergency air medical services. In 1992, these recommendations were accepted by the California Medicaid provider as reasonable criteria for the use of air medical transport. In a review of 558 consecutive patient transports, 98% had met at least one of the AAMS criteria.[81]

The risk of aeromedical transport can be placed in perspective if the overall risk for death using ground transport, estimated from the trauma score, is compared with the risk when patients are transported by air. Assuming a reduction in risk of between 21% and 52% when transport is by air, the additional risk for death from crashes (6 per 100,000 transports, or 0.006 per transport) is negligible in comparison to the benefits. This is probably true, however, only for patients with moderate to severe, but nonmortal, injuries (i.e., trauma scores between 5 and 14). Those having minor injuries, with near 100% likelihood of survival, are unlikely to gain additional benefit; those having mortal injuries, with little hope of survival, are unlikely to be saved by any means attempted or employed.

The decision to transport a patient by air requires judgment and a realistic appraisal of the risks. A patient should be transported by air only if he or she is so ill that transport is necessary; if ground transport is unavailable, delayed, or unable to reach the patient; or if aeromedical transport would reduce the risk for death by permitting more rapid access to definitive care, providing greater medical skill en route, or both.[83]

SPECIAL PROBLEMS IN AEROMEDICAL TRANSPORT

Trauma

Aeromedical evacuation experience during operations in Afghanistan and Iraq have shown that large numbers of even severely injured patients can be moved long distances by air if proper equipment and personnel are available to carry out the transport. In July 2004, 2121 patients were moved by the USAF: 39 (1.8%) required transport by CCAT teams. In many instances, critical trauma patients are moved after "salvage surgery," in which initial surgical care is limited to stopping bleeding and limiting contamination, with expectation of further surgery for definitive repair. Patients have been moved with open abdomens and with intracranial pressure monitors, ventriculostomies, and wound vacuum drainage devices in place. In one case, a patient was successfully flown from Hawaii to California while on cardiopulmonary bypass. Transport of severe trauma victims requires careful attention to proper resuscitation, pain control and sedation, fracture stabilization, and temperature control. Abdominal and chest injury patients are frequently flown while on ventilator support; proper attention to sedation, analgesia, antibiotic administration, and neuro-

muscular paralysis is required. Patients with even a minor pneumothorax must have a chest tube in place before flight.[53,107] Blood and blood products can be given in flight; however, careful attention must be given to proper refrigeration of blood and blood products. Blood administration protocols for use in flight should mirror standard practices for administration of blood and blood products, and medications to deal with transfusion reactions must be immediately available.[107]

Burns

Burn critical care and transport are both very similar and very different from standard critical care and transport. As in any aeromedical evacuation, careful planning is the key to successful transport.[44] Transport of significant burn victims is best accomplished as early as possible in the course of the burn injury, preferably in the first 24 to 36 hours after injury. If there is any evidence of significant smoke inhalation or thermal injury to the orotracheal area (i.e., carbonaceous sputum, hoarse voice, singed nasal hair), the patient should be intubated before flight. Circumferential third-degree burns may require escharotomy before flight. Burn victims require careful temperature monitoring during flight to avoid hypothermia. Fluid resuscitation algorithms may need to be modified to compensate for increased insensible water losses due to low cabin humidity. Monitoring urine flow hourly is mandatory in burn patients.[53] The U.S. Army Institute for Surgical Research (ISR) at Brooke Army Medical Center in San Antonio, Texas houses the only dedicated burn unit in the U.S. military. The ISR has several burn transport teams that specialize in short- and long-range transport of critical burn patients (Fig. 35-16). Teams generally include four personnel and about 800 pounds of medical equipment. These teams train alongside, and work closely with, USAF CCAT teams and aeromedical evacuation crews.

A

B

Figure 35-16. **A,** Two "burn victims" (actually manikins) being transported by the U.S. Army Burn Transport Team during a training mission on board a U.S. Air Force C-17 aircraft (**B**). (Photos courtesy of Robert C. Allen.)

Infectious Disease Patients

Transport of infectious patients may be a hazard to the flight crew, medical crew, and other passengers because everyone on board the aircraft is sealed in an aluminum tube for hours, sometimes breathing recirculated air. Airflow patterns inside the aircraft cabin may contribute to the problem, wafting fomites downwind of a patient. Several cases of severe acute respiratory syndrome (SARS), influenza, and tuberculosis have been attributed to passenger-to-passenger transmission on commercial aircraft.[56,61,80]

Standard infection control practices, including wearing gloves, masks, and gowns (as required), should be followed with all patients, not just those suspected of being contagious. Handwashing facilities on aircraft are likely to be nonexistent;

Box 35-6. Communicable Disease Checklist

RULE-OUT CHECKLIST (HIGHLY TRANSMISSIBLE INFECTIOUS DISEASE AGENTS)

- Any history of exposure to sewage, body fluids, animals prior to illness?
- Any report of insect bites?
- Did progression to severe symptoms and illness occur rapidly, over a period of less than 3 days, once the patient began to feel ill?
- Fever (temperature greater than 102.0°F [38.9°C])?
- Any bleeding from gums?
- Any nose bleeds?
- Any report of petechiae on the palate, throat, or on exam of the mouth?
- Are the patient's eyes "bloodshot" in appearance, typically described as "conjunctival injection" in medical terminology?
- Any report of tender or painful lymph nodes?
- Any report of lymph nodes with a "bruised" or darkened appearance?
- Any report of "matted lymph nodes," "goose-egg" lymph nodes, or lymph nodes draining pus?
- Any report of petechiae or purpura on the skin?
- Any report of bloody stools, melena, hematochezia, hematemesis?
- Any report of a positive "tourniquet test"? (NOTE: This test is performed by inflating a BP cuff on an extremity. It is considered a positive test if petechiae appear on the skin of the extremity distal to the cuff and is a very good indicator of vasculitis associated with hemorrhagic fever agents.)
- Any report of icterus (yellow eyes, skin, or tongue)?
- Any report of cough *and* fever in conjunction with any of the following:
  - rapid disease progression?
  - petechiae, purpura?
  - lymphadenopathy?
- Any report of "pox" or "poxlike" skin rash or lesion?

NOTE: Answering "yes" to any one question requires immediate telephone consultation with the theater validating flight surgeon.
(Data from references 5, 6, 14, 40, 42, 75, 83, 85.)

use of waterless hand cleaners such as alcohol gel is the next best thing.[114]

Patients in the early stages of some highly infectious diseases may not present with obvious symptoms, or these symptoms may not be recognized by personnel inexperienced in infectious disease or tropical medicine. To help aeromedical evacuation crews evaluate patients before flight, USAF aeromedical evacuation crews are supplied with a checklist of symptoms (Box 35-6). The patients' clinical signs and symptoms are compared with the list, and if any "yes" answers are found, the aeromedical evacuation crew is required to seek additional guidance from a higher medical authority before allowing the patient on the aircraft.

In most cases, patients with contagious diseases can be safely moved by air simply by applying appropriate infection control procedures. Precautions required for transporting an infectious disease patient depend on the pathophysiology and means of contagion. For example, although malaria is an infectious disease, it is not a significant contagion hazard onboard an aircraft, whereas pulmonary tuberculosis is a significant contagion hazard. However, this hazard can be mitigated by the patient and medical crew wearing N95 masks, proper handwashing techniques, and making sure the patient is seated downwind and well away from other patients or passengers.[114]

Patients suspected of having highly contagious diseases can be moved by specialized transport teams. The U.S. Army Medical Research Institute for Infectious Disease at Ft. Detrick, Maryland has a portable Biological Level IV patient transport system capable of moving two patients with highly contagious disease.[5] The British Royal Air Force has a similar system, which it has used to transport patients suspected of having diseases such as Lassa fever. However, in most cases, such units are not necessary; standard infection control practices modified for the flying environment suffice.

An outstanding paper on aeromedical transport of infectious patients was authored by Withers and Christopher.[114]

SUMMARY

Aeromedical transport, like most other endeavors in medicine, requires teamwork and good communication. Cooperation between the sending and receiving medical facility, the medical aircrew, and the cockpit crew is vital to a successful mission. It has been estimated that emergency aircraft transport of sick and injured patients has allowed more than 1 million patients to be transported over 100 million miles. With a conservative estimate of mortality reduction of even 10%, close to 100,000 patients may owe their lives to the speed and skill provided by aeromedical transport teams.[51]

DISCLAIMER

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The references for this chapter can be found on the accompanying DVD-ROM.

36 Essentials of Wilderness Survival

Warren D. Bowman and Peter Kummerfeldt

This chapter is concerned with the *general principles* of wilderness survival, particularly in temperate and moderately cold (nonpolar) environments such as the mountains of the American West. For additional information, especially on specific environments, please refer to Chapter 9, which covers polar medicine, Chapter 37 on survival in the jungle, Chapter 38 on survival in the desert, Chapter 85 on wilderness navigation techniques, Chapter 1 on high-altitude medicine, and Chapter 65 on living off the land.

The term *survival* means to "continue to live or exist" and implies the presence of adverse conditions that make this more difficult. These conditions may, and frequently do, include lack of oxygen, food, or water; the presence of rain, snow, high winds, or temperature extremes without shelter; the complicating presence of illnesses or injuries; and the necessity to rely completely on the physical, mental, and material resources at hand. As we learned in February 2002, even as powerful and vigorous an individual as 2000 Olympic Greco-Roman heavyweight wrestling champion Rulon Gardner can be significantly injured when marooned alone in subzero weather without proper equipment or shelter. Physicians who participate in wilderness recreation or who treat adventurers need to be aware of the physical, physiologic, and psychological hazards of environmental stress and how related deleterious effects can be prevented and treated in themselves and their patients.

Increased leisure time and growing interest in outdoor activities place more people into settings where survival situations may develop. Increasing use of all-terrain vehicles and snow-

use of waterless hand cleaners such as alcohol gel is the next best thing.[114]

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Increased leisure time and growing interest in outdoor activities place more people into settings where survival situations may develop. Increasing use of all-terrain vehicles and snow-

mobiles has made it easier for novices to get lost or stranded far from help. Along with this, the rise in sedentary lifestyles with inattention to healthy diets, decreased emphasis on physical conditioning, and the apparently increasing incidence of the "metabolic syndrome" (central adiposity, dyslipidemia, insulin resistance, and hypertension) mean a decrease in fitness worldwide. With modern communications and tactics, such as the use of combined helicopter and ground teams, search and rescue operations have become more efficient. At the same time, electronic gadgets, such as cellular and satellite phones, Global Positioning System (GPS) locators, emergency locater transmitters (ELTs) for aircraft, and personal locator beacons (PLBs), have made it easier to get assistance, but have led to an increase in risk taking and a decrease in healthy respect for wilderness, neglect of survival training, decreased provision of survival equipment, and less effective use of simple tools such as maps and compasses. Societal affluence, then, has made it easier both to get into trouble and to get assistance in case of trouble. This chapter's main thrust is the prevention and successful initial definitive management of such trouble. By paying heed to the recommendations discussed here, the reader will be more apt not only to live through a survival emergency but also perhaps even to do so in relative comfort.

The exact type of environmental stress depends on the type, location, and duration of the wilderness experience. Cross-country skiers, winter mountaineers, and winter campers may be exposed to extremes of cold and storm. Expeditionary mountaineers may explore regions where winter exists year-round and where ambient oxygen is low. Desert or tropical travelers may be exposed to extremes of heat and humidity. Passengers in aircraft, sea craft, or land vehicles may be stranded in almost any type of environment.

Requirements for survival (discussed in detail later) are similar whether the subject becomes lost with few resources during a simple day hike or whether injury occurs or severe environmental conditions develop during a well-planned wilderness expedition. Although traveling alone in the wilderness is universally condemned, one must always assume that he or she may be alone and possibly injured when in a survival scenario. Therefore, emergency equipment must be familiar, sturdy, uncomplicated, easy to assemble, and—most important—available immediately. Necessary skills cannot be learned on the job but must be acquired *and practiced beforehand* so that essential tasks can be accomplished with minimal effort should an emergency arise. Therefore, although improvisation and living off the land are important, *anticipation, prevention,* and especially *preplanning* and *carrying emergency equipment* are vital. As examples taken from polar exploration, Roald Amundsen's style of thorough preparation and the use of familiar equipment should be emulated, rather than Robert Scott's careless, arbitrary, and stubborn approach.[10] Both of these explorers had extensive polar experience, but Scott elected to use European clothing instead of Eskimo furs and unproven motor drawn sledges and ponies instead of Eskimo dogs. He relied excessively on sled hauling by men on foot, rejected the use of skis, failed to establish enough supply depots for his return, and died of cold, exhaustion, and starvation on the way back from the South Pole. Amundsen, who used Eskimo clothing, well-tested Greenland sled dogs, and experienced skiers, won the race to the South Pole and made it back alive.

The outcome of an encounter with severe environmental stress varies with both the stressed person's resources and the type, magnitude, and duration of the stress. These resources include the state of acclimatization; physical integrity—particularly conditioning and the presence of illness or injury; experience; equipment on hand plus the ability to improvise intelligently; and such intangibles as good judgment and "backcountry common sense." A common judgment error is to insist on traveling during a storm or other stressful environmental condition when more prudent persons would stay put in a comfortable bivouac. Excuses for this include desiring to reach a predetermined but not essential goal on time, so that others "will not worry." Recall the old adage (attributed to Will Rogers) that "good judgment comes from experience; experience comes from bad judgment." To this we should add ". . . provided you survive the bad judgment." The most important resource, however, may well be the *will to survive*. This may be inbred in some persons, can be established through training and experience in many, and is impossible to acquire in a few. Without a will to survive, the best training and resources may be useless.

With a strong will to survive, however, persons have survived incredible hardships. A recent example is Aron Ralston, an expert climber who at the time of his accident had done successful winter solo climbs of 45 of the 59 Colorado peaks of more than 14,000 feet (4267 m) elevation (see Ralston [2004] in "Suggested Readings"). On Saturday, April 26, 2003, he parked his truck at a trailhead and entered deep and narrow Horseshoe Canyon in Utah's Canyonlands National Park. After about 8 miles (12.8 km) down the canyon, he reached a narrow, 12-foot (3.7-m) high chimney-like dropoff that narrowed to about 3 feet (0.09 m) wide at its bottom. He stepped down onto a large boulder wedged a foot below its top and started to lower himself over the boulder's edge. The boulder stirred and, before he could get out of its way, it fell, struck his left hand, then jammed into the lower end of the chimney, pinning his right hand and wrist against the floor of the chimney.

For tools, he had carabiners, rope, slings, and a Leatherman multitool; for sustenance, he had a liter of water and two bean burritos. He was expected to be at work on Monday in Aspen, Colorado, but had told no one where he was going. Over the next 5 days, he attempted unsuccessfully to move the boulder with a rigged pulley system of rope and carabiners, and to scrape away enough of the boulder with the large knife blade of his multitool to free his hand. He became weaker and more dehydrated, starting to hallucinate intermittently. After he ran out of water he was reduced to drinking his own urine. By Thursday, severely starved and dehydrated and almost ready to give up all hope, he decided that his only choice was to free himself by amputating his trapped hand and wrist. His knife was too dull to cut the forearm bones but he was able to break both of them just above the wrist by forcefully bending the wrist over the edge of the bounder with his free hand. He then applied a tourniquet and cut himself free using the small knife and wire-cutting pliers from the multitool on the remaining tissues. Following this, he was able to reach water and refresh himself enough to hike 6 miles down the canyon toward his truck. At this point he met a party of hikers, who were able to alert the Park Rangers and get a helicopter evacuation. After multiple surgeries and a bout with osteomyelitis, Aron is climbing again with a specially designed right forearm prosthesis.

Although based on science, the study and practice of survival is more a craft or learned skill than an exact science. The recommendations in this chapter are based on the opinions of

survival experts, research, analysis of actual survival situations, and personal experience. General principles are emphasized, but "tricks of the trade," which are usually acquired only through experience, may hold the key to life or death. Unfortunately, much of the lay literature emphasizes tales of misfortune, hazardous adventures, and mindless bravado in the face of unnecessary hardships brought on by errors of the participants, whereas great deeds go unrecorded or forgotten because the experience and competence of the adventurers kept catastrophic "newsworthy" experiences to a minimum. In the words of Corneille, "To vanquish without risk is to triumph without glory."[5]

Travelers should always plan for the unusual and unexpected. Tools include familiarity with weather forecasts, strategizing worse-case scenarios, carrying emergency items, avoiding solo travel, and leaving notice of the projected route and expected time of return. With good planning, deteriorating weather or an injury-forced bivouac becomes more of an inconvenience than a life-threatening ordeal. However, chance always plays a part in survival. Serious but unforeseen or unavoidable hazards can occur, or environmental stresses can become so severe that survival is impossible regardless of preparations. Anyone venturing into wilderness must accept the possibility, however remote, of death or serious injury.

For survival, the body requires a constant supply of oxygen; a core temperature regulated within relatively narrow limits (about 24°–42° C [75°–107° F]); water and food; and self-confidence, faith, and the will to live. For comfort and optimum performance, however, body temperature must be close to normal, and the body must be rested, well nourished, in top physical condition, and free from disease and injury. The most immediate of these latter requirements are maintenance of body integrity (through accident prevention) and regulation of body temperature. Dehydration, starvation, and exhaustion make temperature maintenance more difficult and interfere with the rational thought and agility required to prevent accidents. Insufficient oxygen becomes a contributing factor at extreme altitude or in such mishaps as suffocation caused by avalanche burial or carbon monoxide (CO) poisoning from cooking in an unventilated shelter. Abundant food and water are of little value to a hypothermic person with insufficient clothing and shelter or to the victim of heat stroke, even though lack of food and water will eventually weaken and kill an otherwise healthy individual. Lack of self-confidence, faith, and the will to live may cause an attitude of panic and defeatism that prevents a person from taking timely survival actions, such as conserving energy, preparing shelter, or lighting a fire. Poor physical conditioning or the presence of illness or injury may interfere with the body's ability to produce heat by shivering or to lose heat by sweating and increasing skin perfusion, and can hamper wood gathering, shelter building, and other necessary tasks.[3]

The most important human organ for survival is the *brain* because voluntary actions such as preparedness, regulation of energy expenditure, adjustment of clothing, and providing shelter are more important than involuntary mechanisms of adaptation to environmental stress.

OXYGEN

As a human ascends from sea level, the body is subjected to increasing cold, decreasing oxygen, increasing solar radiation, and decreasing atmospheric pressure. For every 305 m (1000 feet) of altitude gain, the ambient temperature drops by about 2.2° C (4° F), the barometric pressure drops by about 20 mm Hg (i.e., 27 millibars [mb], or roughly 0.1 mb drop per meter of altitude gain), and the amount of ultraviolet (UV) radiation increases by about 5%. The percentage of oxygen in the atmosphere remains constant, but the partial pressure of oxygen diminishes with altitude, so that at 3077 m (10,000 feet), it is only two-thirds that at sea level, and at 5488 m (18,000 feet), only half.[3]

During acute exposure to high altitude, the effects of hypoxia initially can cause fatigue, weakness, headache, anorexia, nausea, vomiting, dyspnea on exertion, insomnia, and Cheyne-Stokes respirations. These symptoms are probably present to some degree in everyone who goes rapidly from sea level to 2462 m (8000 feet) or above. The clinical effects of hypoxia are often difficult to distinguish from those of cold, high winds, dehydration, and exhaustion. Serious degrees of acute mountain sickness (AMS) are unusual below 3692 to 4308 m (12,000–14,000 feet) but have been reported in trekkers as low as 2308 m (7500 feet). In Yellowstone National Park, mild AMS is not infrequently seen in visitors at just over 1829 m (6000 feet). At any height, oxygen in ambient air may be prevented from reaching the cellular level because of interruption of normal transport pathways, generally by illness or injury. CO poisoning is probably a greater hazard than is generally appreciated. Many famous polar explorers, including Byrd, Andree, and Stefannson, were killed by or had narrow escapes from the effects of stoves operated in tightly enclosed spaces.[12,17]

REGULATION OF BODY TEMPERATURE

Humans are called *homeotherms* because as warm-blooded animals they maintain a body temperature that varies within very narrow limits despite changes in environmental temperature.[2,3] In *poikilotherms,* or cold-blooded animals, body temperature varies with that of the environment. Homeothermy is necessary to support the enzyme systems of the human body, which function best at 37° to 37.5° C (98.6°–100° F). The human body can be viewed as a heat-generating and heat-dissipating machine, where internal temperature is the net result of opposing mechanisms that tend to increase or decrease body heat production, increase or decrease body heat loss, and increase or decrease addition of heat from the outside. Through these mechanisms, the internal body temperature usually can be regulated successfully, despite ambient temperatures that vary more than 55° C (100° F) from the coldest to the hottest seasons in temperate climates.

Basal body heat production is about 50 kcal/m^2/h. This can be increased by muscular activity (involuntary [shivering] and voluntary), eating, inflammation and infection (fever), and in response to cold exposure. Shivering can increase heat production up to five times the basal rate and vigorous exercise up to 10 times. Cold exposure increases hunger, the secretion of epinephrine, norepinephrine, and thyroxin, and semiconscious activity such as foot stamping and dancing in place. Eating provides not only needed calories but also the temporary increase in basal metabolic rate occurring during digestion alone (specific dynamic action, or SDA). The SDA of protein is five to seven times higher than that of fat and carbohydrate and lasts

Wind mph	Temperature (°F)																	
Calm	40	35	30	25	20	15	10	5	0	−5	−10	−15	−20	−25	−30	−35	−40	−45
5	36	31	25	19	13	7	1	−5	−11	−16	−22	−28	−34	−40	−46	−52	−57	−63
10	34	27	21	15	9	3	−4	−10	−16	−22	−28	−35	−41	−47	−53	−59	−66	−72
15	32	25	19	13	6	0	−7	−13	−19	−26	−32	−39	−45	−51	−58	−64	−71	−77
20	30	24	17	11	4	−2	−9	−15	−22	−29	−35	−42	−48	−55	−61	−68	−74	−81
25	29	23	16	9	3	−4	−11	−17	−24	−31	−37	−44	−51	−58	−64	−71	−78	−84
30	28	22	15	8	1	−5	−12	−19	−26	−33	−39	−46	−53	−60	−67	−73	−80	−87
35	28	21	14	7	0	−7	−14	−21	−27	−34	−41	−48	−55	−62	−69	−76	−82	−89
40	27	20	13	6	−1	−8	−15	−22	−29	−36	−43	−50	−57	−64	−71	−78	−84	−91
45	26	19	12	5	−2	−9	−16	−23	−30	−37	−44	−51	−58	−65	−72	−79	−86	−93
50	26	19	12	4	−3	−10	−17	−24	−31	−38	−45	−52	−60	−67	−74	−81	−88	−95
55	25	18	11	4	−3	−11	−18	−25	−32	−39	−46	−54	−61	−68	−75	−82	−89	−97
60	25	17	10	3	−4	−11	−19	−26	−33	−40	−48	−55	−62	−69	−76	−84	−91	−98
						Frost bite in >>	30 min		10 min		5 min							

Figure 36-1. Windchill chart. (From Bowman WD, Johe DH, American Academy of Orthopaedic Surgeons, National Ski Patrol System [U.S.]: Outdoor Emergency Care: Comprehensive Prehospital Care for Nonurban Settings, 4th ed. Boston: Jones & Bartlett, 2003, p 32, Figure 2-5.)

longer. However, the onset of the SDA is much faster with carbohydrate than with protein or fat. Therefore, the person who is cold inside a sleeping bag at bedtime should eat carbohydrate for quicker warming, and protein to stay warm all night. In hot weather, body heat production can be decreased by slowing muscular activity and avoiding foods with a high SDA.

In cold weather, heat can be added to the body by close exposure to a fire or other heat source, such as sunlight, and by ingesting hot food and drink. In hot weather, external heat addition can be decreased by staying in the shade, wearing clothing that blocks the sun's rays, and avoiding hot objects and hot food and drink.

The body loses heat to the environment by conduction, convection, evaporation, radiation, and respiration. It may gain heat from the environment by the same mechanisms (except for evaporation). The relative importance of these mechanisms depends on temperature, humidity, wind velocity, cloud cover, insulation, contact with hot or cold objects, sweating, and muscular exercise. With a resting body in still air at 21°C (70°F), radiation, conduction, and convection account for about 70% of total heat loss, evaporation for about 27%, and urination, defecation, and respiration for only 3%. During work, however, evaporation may account for up to 85% of heat loss.[3]

It is useful to think of the body as composed of a core (heart, lungs, liver, adrenal glands, central nervous system, and other vital organs) and a shell (skin, muscles and extremities). Most of the physiologic adjustments in response to cold or heat exposure occur in the shell. They are designed to maintain a relatively constant core temperature; in below-freezing weather, these adjustments may predispose parts of the shell to frostbite and other types of localized cold injury.

The importance of avoiding travel and seeking shelter during storms and extreme cold cannot be overemphasized. The additive chilling effect of wind when added to cold is impressive. Windchill charts (Fig. 36-1) show the relationship between actual temperature, wind velocity, and "effective" temperature at the body surface. *Windchill* refers to the *rate* of cooling; the actual temperature reached is no lower than it would be if wind were absent (unless evaporation of liquid is occurring at the body surface). The increase in heat loss as the wind rises is not linear but is more proportional to the *square root* of the wind speed.

At moderate ambient temperatures, the body's core temperature is kept stable by constant small adjustments in metabolic rate, muscular activity, sweating, and skin circulation. When the body is chilled, automatic and semiautomatic mechanisms increase internal heat production by slightly increasing the metabolic rate, by shivering, and by semiconscious activities (e.g., foot stamping), and reduce heat loss by diminishing sweat production and shell circulation. The person has a strong urge to curl up into a ball, thereby reducing the body's surface area. At the same time, the brain tells the body to decrease heat loss by adding insulation and wind protection, to seek shelter, and to increase heat gain by increasing muscular activity, building a fire, seeking sunlight, and eating.[3]

When the body overheats, these actions are reversed. The body increases heat loss by increasing circulation to the skin and extremities and increasing sweating. These mechanisms require more water, which stimulates the thirst response. Heat production is decreased because of a feeling of sluggishness and languor, leading to a reduction in physical activity and the amount of heat produced by muscles. The brain tells the body to decrease heat gain and increase heat loss by providing shelter from the sun, removing clothing, and fanning itself.[2]

COLD WEATHER SURVIVAL

Body temperature in a cold environment is maintained by decreasing body heat loss, increasing internal body heat production, or adding heat from the outside. The most efficient of these methods is conservation of body heat by decreasing heat loss, generally by using clothing and shelter.

DECREASING BODY HEAT LOSS

Heat loss from conduction and convection can be prevented by interposing substances of low thermal conductivity, such as clothing made of insulating materials, between the body and the outside air. Clothing creates a microclimate of warmed, still air

TABLE 36-1. Fiber Characteristics of Natural and Synthetic Fibers

FABRIC	SPECIFIC GRAVITY* (RATIO TO WATER)	THERMAL CONDUCTANCE† (cal/m^2)	EVAPORATIVE ABILITY‡	WICKING ABILITY	MOISTURE REGAIN§
Wool	1.32	2.1	Low	Moderate	17
Cotton	1.54	6.1	Low	High	7.9
Nylon	1.14	2.4	High	Low	4
Polyester	1.38	2.4	High	Low	1
Acrylic	1.15	2.4	High	High	1
Polypropylene	0.91	1.2	High	High	5

*The lower the specific gravity, the better the insulating ability.
†The lower the thermal conductance, the slower the flow of heat from the body.
‡The higher the evaporative ability, the shorter the amount of time a fiber will be wet, that is, in a reduced insulative state.
§Moisture regain is the amount of moisture a fiber can absorb before feeling wet.
Modified from Davis AK: Nordic Skiing: A Scientific Approach. Minneapolis, University of Minnesota, 1980.

next to the skin surface. Clothing's value depends on how well it traps air, the thickness of the air layer, and whether these qualities are reduced by wetting (Table 36-1). Traditional insulating materials are wool, down, foam, and older synthetics such as Orlon, Dacron, and polyester. Wool retains warmth when wet because of a moderately low wicking action and a unique ability to suspend water vapor within its fibers without affecting its low thermal conductance. It can absorb a considerable amount of water without feeling wet but is heavier than synthetics, itchy, and more difficult to dry. Its toughness and durability, however, make it a good choice for garments subject to hard wear, such as trousers, mittens, and socks. Cotton, particularly denim and corduroy, is a poor insulator. It dries slowly because of low evaporative ability; high thermal conductance is further increased by wetting. Cotton has no place in the backcountry in cold weather.

Orlon, acrylic, and polyester were developed to duplicate wool's properties without wool's higher cost and other perceived drawbacks. They traditionally have been used in hats, shirts, sweaters, and long underwear. They are almost as warm and not as itchy as wool, and they evaporate moisture better. A number of newer fabrics are woven from fibers that have lower thermal conductance, greater insulating ability, and better wicking action than traditional fibers. Examples include polypropylene and treated polyesters, such as Capilene, Thermax, and Thermastat. Polyester is also made into pile and fleece, which are light, dry easily, trap air well, and stay warm when wet because the fibers do not absorb water. Examples are Polartec, Borglite, Polarplus, and Synchilla. Fibers used as fillers in quilted garments, such as parkas, include hollow synthetic fibers designed on the principle of reindeer hair, such as Hollofil II and Quallofil. Microfibers that provide good insulating ability with less bulk include Thinsulate, Thermoloft, and Thermolite. One of the newer fibers, Microloft, is supposed to be warmer than down at the same weight. New synthetics come on the market frequently, so one should consult trade journals and "gear" issues of outdoor magazines, such as *Backpacker*.[2,3]

The "layer principle" of clothing is effective in preventing both chilling and overheating. Multiple layers of clothing provide multiple layers of microclimate. Layers are added as necessary to prevent chilling or subtracted to prevent overheating that would lead to perspiring. Because water conducts heat 25 to 32 times faster than air at the same temperature, clothing wetted by perspiration or water may cause rapid heat loss from conduction and evaporation. The need to add or subtract layers should be anticipated before either chilling or heavy perspiring occurs.

Clothing should be easily adjustable, sweaters should be of the zipper or cardigan type, and outer layers should be cut full enough to allow expansion of inner layers to their full thicknesses. Zippers in the axillary, lateral chest, and lateral thigh areas are useful for ventilation.

Loss of heat from convection can be prevented by wearing windproof outer garments of nylon, tightly woven cotton-nylon blends, or water-resistant laminates such as Gore-Tex. Typical examples include a parka with hood and a pair of windproof pants (regular or bib style) or ski warm-up pants.

The loss of heat from infrared radiation can also be prevented by insulation, emphasizing proper covering for body parts with a large surface area to volume ratio. The uncovered head can dissipate up to 70% of body heat production at an ambient temperature of −16°C (5°F), partly because the body does not reduce blood supply to the head and neck as it does to the extremities in cold weather. High heat loss through radiation during cold nights can be decreased by sleeping in a tent or under a tarpaulin (tarp) instead of in the open. Coverage for the head, ears, hands, and feet should not restrict circulation. Developed initially for skiers, the "neck warmer," or "neck gaiter," can be pulled up over the back of the head to form a hood or up over the lower face to form a mask. In addition to head and neck protection, this device also blocks some of the heat that would otherwise be lost by the bellows action of clothing caused by body motion.

Heat loss from the respiratory tract can be diminished by avoiding overexertion and overheating that occur with excessively heavy breathing. When it is extremely cold, inspired air can be warmed by pulling the parka hood out in front of the face to form a "frost tunnel."

Heat loss from conduction occurs by direct contact with a colder object. Sitting on a pack, foam pad, log, or other object of lower heat conductivity is preferable to sitting in the snow or on a cold rock. At low temperatures, bare skin freezes to metal. This can be avoided by wearing light gloves when handling metal objects. Gasoline or other liquids with freezing points lower than that of water can cause frostbite if accidentally poured on the skin at low temperatures. During bivouacs in snow shelters, contact with the snow can be avoided by sitting on a foam pad or backpack or improvising a mattress of

evergreen boughs, grass, or dry leaves. In cold or windy weather, an injured person needs windproof insulating material under as well as over and around the body.

Heat loss from conduction and evaporation can be lessened by avoiding wetting and by changing to dry clothes or drying out quickly when wet. Ideally, outer clothing should be windproof, should not collect snow, and should shed water but not be waterproof because waterproof garments prevent evaporation of sweat. Laminated fabrics, such as Gore-Tex and its relatives, are suited for this and for outermost layers.

Dressing for Cold Weather

Anyone who ventures outdoors in cold weather should have enough clothing of the proper kind, either on the body or in the backpack, for the most extreme environmental conditions likely to be experienced. Even though fire-building, carrying emergency shelters, and improvising survival shelters are discussed at length later in this chapter, the process of dressing for cold weather should be approached with the idea that clothing may become the only "shelter" and endogenous heat production the only heat you have available. Clothing and other types of insulating materials should be selected with the idea that they need to keep one warm and dry even during periods of inactivity.

First Layer

Long Underwear. Wool is an excellent choice for long underwear but is expensive and may be difficult to find. Merino wool is less itchy. Polypropylene, acrylic, and the newer polyesters may be preferable because of their lower cost, good insulating ability, and outstanding evaporative ability (Table 36-1). Avoid fabrics containing cotton. Synthetics tend to retain body odor more than wool after washing.

Socks. One or two pair of moderate to heavy wool or wool-nylon blend socks are excellent, preferably with an additional pair of light polypropylene socks worn underneath next to the skin. Consider carrying at least one spare pair of wool socks.

Thin Gloves (Glove Liners). Light polypropylene, wool, silk, or fingerless wool or pile gloves are useful for moderately cold conditions or when finger dexterity is required, as in adjusting ski bindings. Polyester/Lycra gloves provide a tighter but more stretchable fit, which enhances fine finger movements.

Second Layer

Shirt. Shirts should be made of light, soft wool or a durable synthetic such as acrylic, and should have long sleeves. Large breast pockets with buttons or Velcro fasteners are handy to carry such items as sunglasses and a compass. For ventilation, the front of the shirt should open completely or at least halfway (buttons or zipper closure). A turtleneck feature protects the neck, as do neck warmers and mufflers that can be pulled up to protect the lower face.

Pants. Wool or pile pants are best and should have pockets that are easily accessible for hand warming. Pile pants should have reinforcements at the knees and buttocks and a zipper or Velcro fly for males. Full or partial lateral leg zippers are convenient.

Foot Gear. The type of boot chosen depends on the type of activity and the expected environmental temperatures. For moderate temperatures, sturdy leather climbing boots made of full-thickness leather, 6 to 8 inches in height with rubber lug soles and roomy enough to accommodate the desired numbers of socks, are ideal. Boots made of leather and fabric, such as Gore-Tex, are lighter and suited for trail hiking, but may not be as durable for rough terrain.

Boots must be long enough so that the toes do not strike the front of the boot during downhill walking. They should be laced firmly enough that the heel does not move up and down, but not so tightly that circulation is restricted and the toes cannot be wiggled easily.

For colder temperatures, double boots are preferable. These can be all-leather boots or can have outer shells of plastic or nylon with inner boots of felt or foam. All-leather versions may be difficult to obtain. The Canadian type of shoe-pack with a removable inner felt liner is a good choice for light snowshoeing and other nontechnical outdoor activities in the cold. Special double ski boots are available for ski touring, Telemark skiing, and ski mountaineering, depending on whether three-pin or rondonnee bindings are used.

Hat. Hats should be of the stocking variety, made of wool, pile, Orlon, polypropylene, or wool-polypropylene, and large enough to cover the ears. A small bill feature is desirable to shade the eyes. "Bomber" caps with bills and pull-down earflaps and "Andean" caps with ear coverings are popular. Some arrangement should be provided to protect the face from cold wind, as with a balaclava configuration or a separate face mask. A useful combination for very cold, windy weather is a ski hat together with a neck warmer that can be pulled up to cover most of the lower face, plus using the parka hood.

Third Layer

Parka. The parka can be a standard ski or mountain parka filled with down, Dacron, Quallofil, Thinsulate, or other lofting material. A more versatile combination is two separate garments: a pile jacket plus a water-resistant shell. For snow-camping, a pile jacket with a thin outer cover of nylon (three-season, squall, or warm-up jacket) may be preferred because, unlike an uncovered pile jacket, it does not collect snow when worn without the shell. The shell should have a hood with a drawstring, a two-way zipper with an overlying weather flap closed with full-length Velcro (in case of zipper failure, very cold hands, or upper extremity injury), a cloth flap to protect the chin from the metal zipper pull, armpit or lateral chest zippers for ventilation, and at least four outer pockets plus one or two inside pockets to contain frequently needed items (e.g., gloves, compass, map, sunglasses, neck warmer). Outer pockets should be located where they can be reached while wearing a backpack with a fastened waist belt. The shell should be fingertip length unless bibs are worn. Zippers should have extra-long tabs added to facilitate closure with cold or mittened hands.

Pockets with horizontal openings may close with Velcro, but those with vertical openings should close with zippers. Because the parka is anchored by the shoulders, when using one hand, it is generally easier to pull a vertical zipper down than up. In some brands of parkas (e.g., Columbia, Marmot, Moonstone, and Patagonia), vertical zippers are pulled *down* to close the pockets; in other brands, they are pulled *up*. The senior author prefers the former type because the danger of losing pocket

contents from difficulty closing a zipper is worse than any perceived delay from difficulty opening a zipper.

For ventilation, there should be zippered openings at the armpits. These should be large enough so that the parka can be converted into a vestlike garment during warm conditions by inserting the wearer's arms through the openings and tucking the sleeves inside the parka. Because these zippers usually perform more easily when pulled from the distal to the proximal direction, this direction should close them; increasing wind protection is usually more urgent than decreasing it (freezing is more dangerous than sweating).

Wind Pants. These should be light and water repellent; a laminated garment such as Gore-Tex is a good choice. Long, zippered side openings are useful to permit donning pants without removing boots, as well as for ventilation and access to inner pants' pockets.

Hand Gear. Mittens and gloves provide hand protection from trauma and cold. Because survival depends to some extent on normal hand dexterity, gloves or mittens should be available to protect hands from cuts, bruises, blisters, and resulting infections. In temperate weather, these can be light, unlined leather gloves.

One of the more serious and still unsolved cold weather problems is how to keep fingers warm while leaving them sufficiently unhampered to do work. Mittens are warmer than gloves because fingers that touch each other warm each other, but even thin mittens do not allow delicate finger movements. An important part of the cold finger solution is to prevent core cooling and compensatory extremity vasoconstriction by addressing core temperature stabilization through exercise, eating, and wearing enough layers on the trunk.

A common strategy is to wear a pair of thin gloves of polypropylene, silk, thin wool, or polyester-Lycra inside a wool or pile mitten covered with a windproof and water-resistant glove shell. For delicate finger work, the gloved hand is removed from the mitten, the work done as fast as possible, and the hand returned to the mitten. However, because insulating materials insulate in both directions, when inside a warm mitten, cold fingers wearing gloves will not rewarm as fast as cold, bare fingers. Therefore, another solution is to use bare fingers, which will perform faster than gloved fingers, work as fast as possible, and return them to warm mittens periodically until the task is done. This is not practical, however, when working with metal in very cold weather. Another approach is to keep a pair of gloves warm in a pocket and put them on after removing the hands from mittens. Polyester-Lycra gloves are easier to don than are many other types of thin gloves.

Excellent three-layer mitten sets include windproof shells with leather palms and two sets of removable pile mittens, at least one of which is fastened to the inner shell with Velcro. Another good system is a thin glove liner inside a heavy wool (Dachstein, rag wool, or wool-polypropylene) mitten inside a Gore-Tex shell. An option that gives more finger dexterity in moderately cold conditions is a polypropylene glove liner inside a fingerless wool glove inside a shell. A new combination is a fingerless wool glove inside a wool mitten that has a horizontal slit in the distal palm (Cordova's rag wool convertible gloves). The distal tip of the mitten can be folded backward dorsally and secured with a Velcro patch, allowing the hand covered with the fingerless wool glove to be exposed through the slit. However, more layers and increasing complexity mean more difficulty doing delicate hand tasks.

Shells should have easily accessible "nose warmers" of pile or mouton on the backs, should be long enough to cover the wrists, and should have palms of soft leather or sticky fabric for securely holding ice axes and ski poles.

Gaiters and Over-boots. Gaiters, which are long, nylon tubes that cover the lower leg and upper part of the boot, are designed to keep snow, sand, and gravel out of boots and socks. They extend upward to just below the knee, open at the side or in front with a zipper or Velcro, and have a strap that fits under the boot sole to keep them snug on the boots and a drawstring at the top to hold them up. Gaiters with a front opening closed by a wide Velcro strap are easiest to get on and off. Shorter versions that extend to just above the ankle are adequate for summer mountaineering and may be preferable for cross-country skiers who need access for tightening boot buckles before descents.

High altitude mountaineering requires special insulated overboots or lined gaiters.

Fourth Layer

The previous three layers are usually worn on the body. A fourth layer should be easily available in the pack. This should include quilted or pile pants and jacket (or vest).

Rain Gear

In moderate climates or in spring conditions when rain and wet snow may be encountered, outer garments of Gore-Tex or similar material should be used. For maritime climates and during seasons of heavy rain, it may be better to have two separate sets of outer garments: a light, thin windproof nylon jacket and pants and a waterproof (coated nylon) jacket and pants.

Vapor Barrier Systems

Waterproof garments and sleeping bag liners worn close to the skin are favored by many because they can prevent wetting of outer clothing by sweat that might result in decreased insulating capacity. There also is claimed to be some reduction in water consumption requirements. Such "vapor barriers" seem to work better in very cold weather than at moderate temperatures. A light garment of polypropylene or similar material should be worn next to the skin, with the waterproof garment over this. Vapor barrier systems should probably be tried out in the field in nonsurvival conditions before being relied on in severe weather. Persons with hyperhidrosis and those who dislike clammy skin may not feel comfortable with them.

SHELTER

Summer Emergency Shelters

When faced with an unplanned night out, one's first step should be to locate a place out of the wind. Getting behind a rock, moving to the lee side of a ridge, going down into the treeline, or even turning one's back to the wind can make a difference. Eliminating windchill allows one to concentrate on dealing with ambient air temperature by using natural insulation and starting a fire. Look for a space into which you can crawl, such as a rocky overhang or a space under dense evergreens or fallen logs. Experienced and prepared individuals will have some type

of waterproof material (plastic bag, tube tent, or tarp) into or under which they can crawl as well. Bright-colored (orange or red) materials are easier for searchers to spot. Getting out of the wind, rain, or snow quickly and being able to stay as warm and dry as possible are vital. Once one is wet and cold, it may be very difficult, sometimes impossible, to rewarm and dry out.

Mylar Space Blankets and Bags

These are lightweight, inexpensive, and compact, but they are of limited value in an emergency. Consider a typical emergency scenario, when it is late in the day, cold, rainy, and windy, and the survivor is injured, hypothermic, or both. A "space blanket" is frequently difficult to get out of the package, to unfold, and to manage in windy conditions. Depending on the brand, space blankets are usually too small to fully encase an adult. When wrapped up, the survivor will find that a space blanket makes a shelter that is so noisy than even an approaching aircraft or ground search party may not be heard. Space blankets also tear very easily when nicked or punctured.

The "space bag" has the same flaws as the blanket except that it is easier to encase yourself and stay enclosed.

Thermal Blankets

These are similar to space blankets, but are made from much heavier material reinforced with fiberglass threads and with a grommet in each corner. They can be used as a body wrap, but again, depending on the size of the person, they are often too small to encase an adult. Some survivors have attempted to use a thermal blanket as a shelter roof by tying lines to each corner and then stretching the blanket between various anchor points. In benign conditions, this may work, but with any wind or snow loading, the grommets quickly tear or pull out.

Tube Tents

These are usually about 8 feet (2.4 m) long and provide a tubular shelter 3 to 5 feet high, depending on the brand. They can be pulled over the body to provide a quick shelter or pitched as a "pup tent." To do this, find two anchors (rocks or trees) the proper distance apart and tie a line to one of them, spread the tent out along the length of the line, run the line through it, and tie off the other end of the line. The height of the line should be such that the tent can be spread out to accommodate the occupant. To avoid ripping, the tent plastic should be 3 to 4 mil thick (1 mil = .001 inch or .0254 mm). Tube tents can be improvised from two plastic 55-gallon drum liners (3 to 4 mil thick) or large, heavy-grade household trash bags by opening up the closed end of one bag, sliding it into the open end of the second bag, and then duct-taping the bags together (Fig. 36-2).

Tarpaulins

Sheets of Visqueen plastic, painters' drop cloths, large pieces of canvas, or other similar materials can be used to erect a wide variety of effective survival shelters. "Blue Crinkly" plastic tarps made of a laminated polyethylene weave are readily available and inexpensive. They can be purchased from most hardware stores, come in a variety of sizes, and are usually blue on both sides with grommets in each corner and at intervals along the sides. An 8- × 10-foot (2.4- × 3-m) tarp is needed to protect an adult. Tarps of this size weigh about 26 oz and roll up into a tube 6 inches in diameter by 12 inches long, which is convenient to carry tied to the outside of a daypack or fanny pack. To save valuable time in a survival situation, tie 10 feet of parachute line to each corner grommet ahead of time.

Figure 36-2. Two large plastic bags can be taped together in tandem and used with a line to form a tube tent. (Photo courtesy of Peter Kummerfeldt.)

Tarps can be erected in a number of shelter styles, depending on weather conditions (Fig. 36-3). To erect a lean-to shelter (see Fig. 36-3A), first select a line long enough to stretch between two trees far enough apart for the tarp to be stretched tight. Using a timber hitch, tie off one end of a line to one of the trees about chest height. Then, rather than passing the line itself through the grommet eyes, insert a small loop of the line through the first grommet eye, and secure the loop with a short stick thrust through it on the opposite side (see Fig. 36-3B). Repeat this process for each grommet, stretching the tarp tight each time. After the tarp is attached to the line, tie off the other end of the line to the second tree, with the line stretched as tightly as possible. The lower edge of the tarp is then pegged to the ground or anchored with large stones or a length of log. When making pegs, select a length of wood $1^1/_2$ to 2 inches (3.8–5 cm) in diameter and twice as long as needed. Using a saw, make one 45-degree cut at the midpoint of the stick. In this way, one cut produces two pegs, both of which are sharp enough to be driven into the ground by pounding their blunt ends with the back of an ax-head or a large rock. Fill in the sides of the lean-to with vegetation.

If a fire will be built in front of the lean-to, the front opening should be parallel to the prevailing wind in order to carry the smoke away from the shelter. If not, the back edge of the lean-to should point into the prevailing wind.

To erect a pup-tent type of shelter (see Fig. 36-3C), tie a line between two trees, drape the tarp over it, and peg down the sides. Block the ends with vegetation or personal equipment.

A lean-to with an eave (see Fig. 36-3D) gives more protection from rain and snow than one without. Instead of attaching the long edge of the tarp to the line between the two trees, drape the tarp over the line so that several feet are on the other side and tie the two corners to pegs to form a down-sloping eave.

A triangular tarp shelter can be erected rapidly and gives good protection (see Fig. 36-3E). It requires three pegs and an anchor point on a tree.

Figure 36-3. Four types of shelters that can be made from a tarp. **A,** A simple lean-to. **B,** Illustration of a method for attaching a line to a tarp. **C,** A pup-tent type of shelter. **D,** A lean-to shelter with an eave.

When using a tarp without grommets, use improvised buttons instead of cutting a hole in the tarp to attach a line. A small, smooth pebble, pine cone, or similar object is placed under the material, which is then gathered around the object to form a "button" (see Fig. 36-3F, G). Using a girth or clove hitch, attach a line to the base of the button. This method is stronger than tying a line through a hole cut in the fabric and will tear out less easily.

Plastic Bag Shelters

Large, heavy-grade (3 to 4 mil) orange plastic 55-gallon drum liners make good short-term emergency shelters. Having one to crawl into quickly will speed warming and drying as well as shelter the survivor from further wetness and chilling. However, total enclosure in a plastic bag is both uncomfortable and dangerous, because of increased wetting from condensation of water vapor in exhaled air and perspiration plus poor ventilation, with lack of oxygen and buildup of carbon dioxide. To minimize these problems, cut an opening in the bottom end of the bag just large enough for your head. Then, pass the bag over your head so that your face is at the opening (Fig. 36-4). If you get too warm, you can push your head through the hole. When creating the hole, cut the plastic at 90 degrees to the fold to reduce the likelihood of the bag tearing along the seam.

Winter and Cold Weather Emergency Shelters

Everyone who spends time in the winter wilderness should practice the construction of several types of emergency survival shelters before they may be needed. The functions of a shelter are to provide an extension of the microclimate of still, warm air furnished by clothing; to contain heat generated by the body, a fire, or other heat source; and to protect from snow, rain, and wind. A properly designed shelter should permit easy and rapid construction with simple tools and should give good protection from wind, rain, and snowfall. The type and size of shelter depend on the presence or absence of snow and its depth, on natural features of the landscape, including the availability of natural building and insulating materials, and on whether fire-

E

F

G

Figure 36-3, cont'd. **E,** A triangular tarp shelter. **F,** Creating a button for attaching a line to a grommetless tarp. **G,** Attaching a line to the neck of the button using a girth or clove hitch. (Photo courtesy of Peter Kummerfeldt.)

wood or a stove and fuel are available. If external heat cannot be provided, a shelter must be small and windproof to preserve body heat.

If possible, a shelter should be constructed in the timber to provide protection from the wind and access to firewood. Generally, shelters partway up the side of a ridge are warmer than those in a valley because cold air tends to collect in valleys and basins during the night. Exposed, windy ridges above the timberline are cold. Areas exposed to flooding (drainages, dry riverbeds), rock-falls, cornice falls, or avalanches or under dead trees or limbs should be avoided. If open water is available, the camp may be located nearby, although in nonsurvival conditions, camps should be at least 200 feet from bodies of water. To avoid drifting snow, tents and shelters should be located with the entrance parallel to the prevailing wind.

Snow is a good insulator (Table 36-2). Its heat conductivity is 1/10,000 that of copper, and its insulating ability superior to wool felt, so snow shelters may be warmer than other types of constructed shelters, as long as the inhabitants remain dry. Contact with the snow or cold ground is avoided by sitting on a foam pad, dry leaves, grass, a backpack, or (in survival conditions only) a bed of evergreen boughs.

Natural Shelters

Caves and alcoves under overhangs are good shelters and can be improved by building wind walls with rocks, snow blocks, or brush. A fire should be built in such as way that heat reflects onto the occupant. The fire should be 5 to 6 feet from the back of the shelter, with a reflector wall of logs or stones on the opposite side of the fire; the occupant should sit between the fire and

the back of the shelter (Fig. 36-5). Adjustments may have to be made to prevent too much smoke from reaching the occupant.

In deep snow, large fallen logs and bent-over evergreens frequently have hollows under them that can be used as small caves. Cone-shaped depressions around the trunks of evergreens ("tree wells") can be improved by digging them out and roofing them over with evergreen branches or a tarp. A fire built to one side of such a shelter will reflect its heat off the snow toward the occupant. Ventilation must be adequate, and the fire should not be positioned under snow-laden branches that can extinguish the fire by dumping snow on it.

Figure 36-4. Example of two large plastic bags used to form a one-person survival shelter. (Photo courtesy of Peter Kummerfeldt.)

TABLE 36-2. Thermal Conductivity of Various Substances

SUBSTANCE	CONDUCTIVITY*	TEMPERATURE MEASURED (°C)
Air	0.006	0
Down	0.01	20
Polyester (hollow)	0.016	–
Polyester (solid)	0.019	–
Snow (old)	0.115	0
Cork	0.128	30
Sawdust	0.14	30
Wool felt	0.149	40
Cardboard	0.5	20
Wood	0.8	20
Dry sand	0.93	20
Water	1.4	12
Brick	1.5	20
Concrete	2.2	20
Ice	5.7	0

*Conductivity is the quantity of heat in gram calories transmitted per second through a plate of material 1 cm thick and 1 cm^2 in area when the temperature difference between the sides of the plate is 1° C.

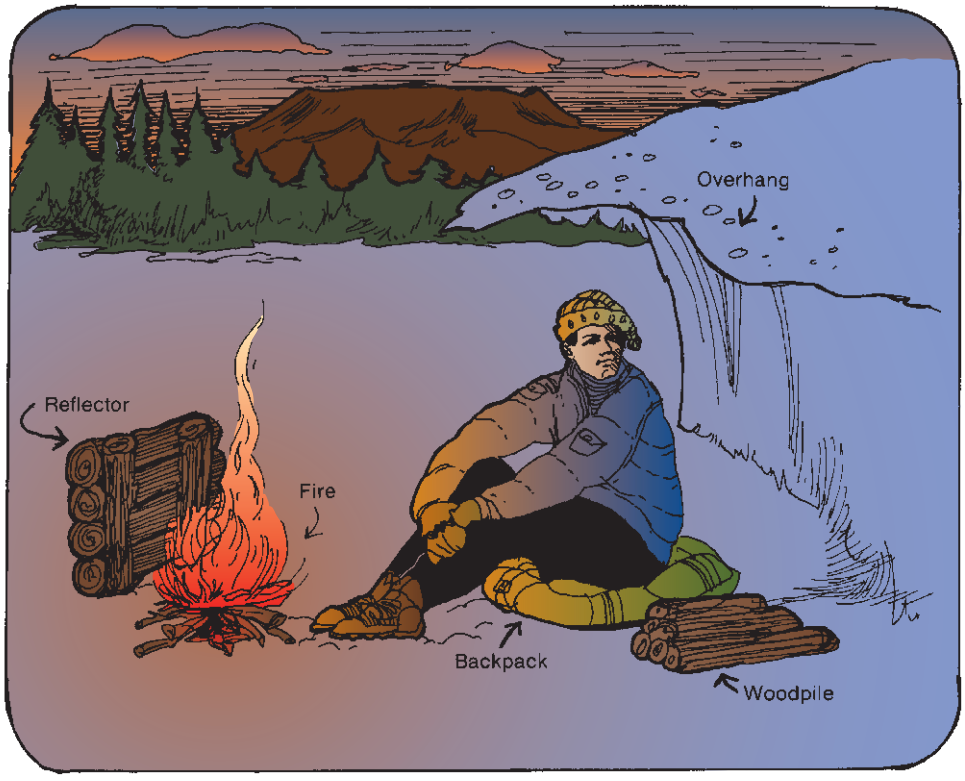


Figure 36-5. Natural shelter.

Figure 36-6. Lean-to shelter. Sides should be closed with brush or snow and a fire built in front.

Constructed Shelters

When no snow is available, shelters can be built of small trees, branches, brush, and boughs. In cold weather with minimum or absent snow cover, the most satisfactory form is a lean-to with two sides closed with brush or piled snow, a fire at the open front, and a wall of logs or stones on the far side of the fire to reflect heat into the interior of the lean-to (Fig. 36-6). Walls or a roof of brush, branches, or broad leaves should be thatched (i.e., each layer should overlap the one below it).

Snow Shelters

If snow shelters become too warm, the walls will be wet, and the roof will drip. A useful rule of thumb is that persons inside a snow shelter should be able to see his or her breath at all times.[19] Snow shelter building should be practiced and perfected beforehand under nonsurvival conditions.

A

B

Figure 36-7. Emergency snow trench. **A,** Pit is dug and overlaid with skis and poles. **B,** Tarp is placed over the skis and secured with snow and heavy objects.

Snow Trenches

A snow trench is the easiest and quickest survival snow shelter and the one least likely to make the diggers wet. It can be dug in most areas that are flat or on slight to moderate inclines as long as the snow is 3 feet deep or deeper or can be piled to that depth. A 4- × 6-foot (1.2- × 1.8-m) trench can be dug in 20 minutes, one end roofed over with a tarp or boughs, and a fire built at the opposite end (Fig. 36-7). Again, adjustments may need to be made to avoid excessive smoke exposure, which can be prevented to some extent by situating the long axis of the trench at a right angle to the apparent wind direction.

If a large (8- × 10-foot [2.4- × 3-m]) tarp and a stove are available, a trench can be dug that is as comfortable as a snow cave and will hold two or three people. The object is to keep the maximal amount of snow around and over the trench. The trench is dug as narrow as possible at the surface while still providing sufficient room to shovel; a suitable size for the top is 4 feet wide by 8 feet long. It is undercut at the back and sides so that the bottom is 6 to 7 feet (1.8 × 2.1 m) wide by 9 to 10 feet (2.7 × 3 m) long (Fig. 36-8). A narrow entrance helps contain heat and can be closed with a small plastic sheet or a pack. Four or more skis or thick limbs are laid from side to side over the top of the trench, with ski poles or branches interwoven at right angles. A tarp is then laid on top of these and the snow piled around its edges to hold it down. In very cold weather, the entire tarp can be covered by a layer of snow; at least 8 inches (20 cm) is needed for proper insulation. When the entrance is closed, a small stove and the occupants' body heat will raise the interior temperature to –4° to –1° C (25°–30° F). Higher temperatures should be avoided so that clothing and bedding will not become wet from melting snow.

Above the timberline in deep, wind-packed snow, a similar trench can be roofed with snow blocks that are laid horizontally, set as an A-frame, or laid on skis (Fig. 36-9). Chinks between the blocks are caulked with snow.

Snow Caves

Although a small snow cave large enough for one person can be dug with a ski or cooking pot, it is much better to have a shovel. Two shovels are best: a medium-sized general-purpose aluminum scoop shovel and a small, straight shovel (French type) to use while excavating the interior of the cave. The best site is a large snow drift, such as is found on the lee side of a

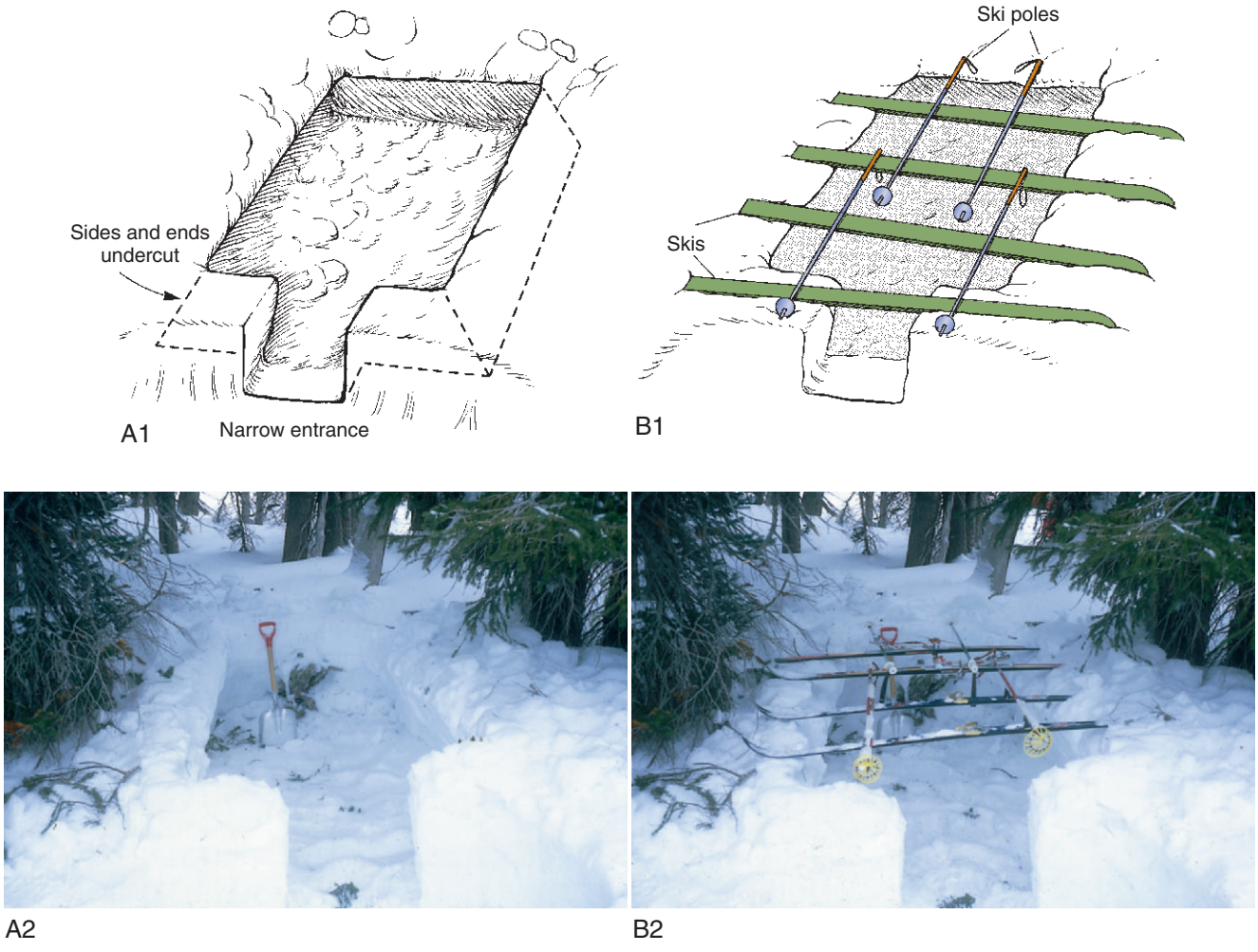


Figure 36-8. **A1** and **A2,** Three-person snow trench. A narrow entrance and narrow top with undercut sides are advisable to trap as much warm air as possible. **B1** and **B2,** Skis and ski poles or stout limbs form the roof support.

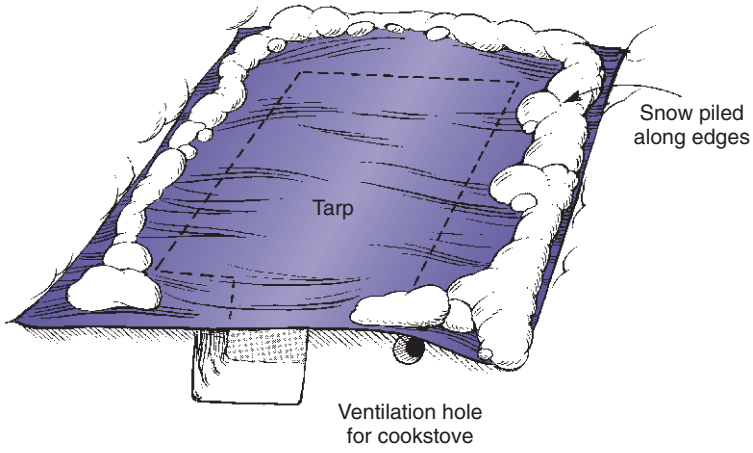
small hill. Areas in avalanche zones or under cornices should be avoided. Because the diggers tend to become wet, water-resistant or waterproof jackets and pants should be worn. In the traditional snow cave, the entrance is dug just large enough to crawl through and is angled upward toward the sleeping chamber (Fig. 36-10A), an arrangement that tends to trap warm air inside. It should be large enough for a stove and two occupants lying side by side. After the entrance is dug with the scoop shovel, the digger crawls inside, lies supine, and uses the straight shovel to excavate the chamber until the space is large enough to allow room to use the larger scoop shovel. A long, level bench about a foot higher than the floor of the cave is constructed and used for a sleeping area. A ventilation hole as large as a ski pole basket is cut in the roof over the cooking area.

Because the traditional cave takes at least 2 hours to dig and gets the diggers quite wet, the T method has found favor.[18] This involves excavating a much larger entrance hole, shaped like a T, so that the digger can stand erect, have plenty of room to dig, and stay drier (see Fig. 36-10B). The crossbar of the T is at the level of the sleeping bench; the digger stands in the foot of the T. After the cave is finished, the top of the T is closed with snow blocks, leaving the foot of the T as a small access hole.

Pine branches or other natural materials are used to cover the floor if a sleeping pad is not available.

Snow Domes

When the ground is flat or the snow cover is shallow, snow can be piled into a large dome 6 to 7 feet (1.8–2.1 m) high and left to harden for a few hours (Fig. 36-11). A low entrance is dug on one side, and from there, the interior is carved out to make a dome-shaped room large enough to sleep three people. A ventilation hole is cut in the roof over the stove. Another method is to make a form (pile of vegetation or equipment), cover this pile with snow, allow the snow to set, and then open one end and remove the form.



C1

D

C2

Figure 36-8, cont'd. **C1** and **C2,** Finished trench with snow piled along edges of tarp to hold it down. Note ventilation hole for cookstove. **D,** Completed trench after a heavy snowfall.

Igloos

Igloos are the most comfortable arctic shelters but require time, experience, and some engineering skill. They are not recommended for the novice, but may be worth the effort if the party will be stranded for any length of time. Igloos require one or ideally two snow saws and snow of the proper consistency. Wind-blown snow in a treeless area is best; otherwise, a large area of snow can be stamped well and left to harden over several hours. To mark the diameter of the igloo, a ski pole is held by the handle, pointed to the side, and the body turned so that the pole basket makes a large circle in the snow that outlines the base of an igloo suitable for three people. Cutting some of the snow blocks from inside this circle will lower the floor so that fewer blocks are required for the dome.

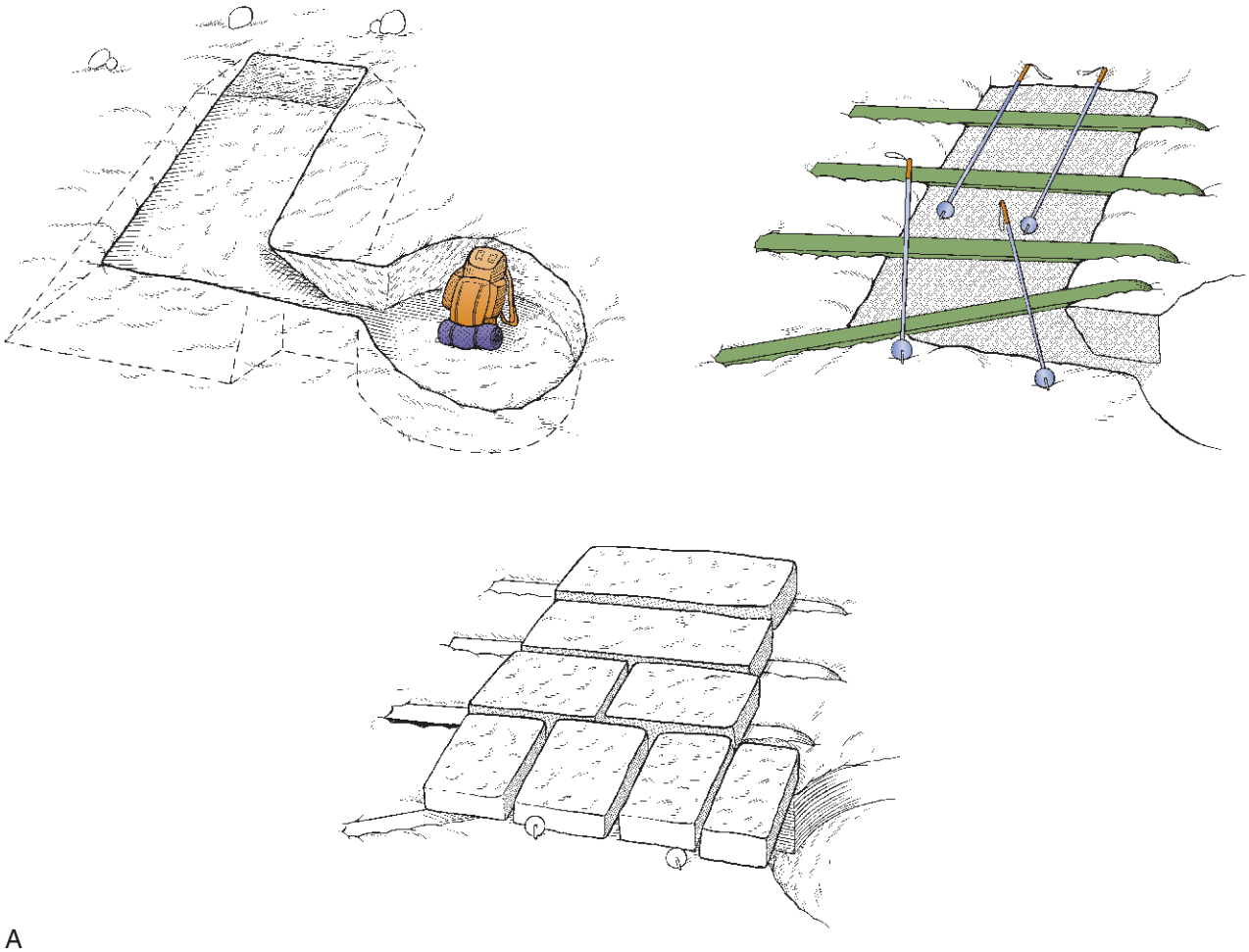
At least two persons are needed: one to cut and carry the blocks and the other inside the igloo to lay the blocks. The blocks should be about 18 inches wide, 30 inches long, and 8 inches (45.7 cm × 76.2 cm) thick. They are laid in a circle leaning in about 20 to 30 degrees toward the center of the igloo, with the sides trimmed for a snug fit. The tops of the first few blocks in the first circle are beveled so that a continuous line of blocks is laid, with the first few blocks of each succeeding circle cocked upward (Fig. 36-12). A common error is to not lean the blocks inward enough, resulting in an open tower instead of a dome. Gaps are caulked with snow. The dome should be 5 to 6 feet (1.5 to 1.8 m) high inside and can be closed with a single capstone of snow. The entrance is dug as a tunnel under rather than through the edge of the igloo, to prevent warm air from escaping.

Tents

Tents are generally comfortable and dry, but in very cold weather are not as warm as snow shelters. They are preferable to snow shelters at mild temperatures, during damp snow conditions at temperatures above freezing, or when the snow cover is minimal. Because of their weight, they are not usually a component of emergency equipment.

Bivouac Sacks and Other Small, Portable Emergency Shelters

These shelters are usually made of thin, waterproof or water-resistant fabric or plastic, do not include an insulating layer, and

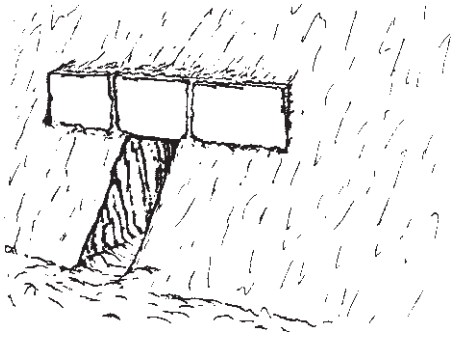


A

B

Figure 36-9. **A,** Above-timberline snow trench. **B,** Completed snow trench the morning after a heavy snowfall.

A



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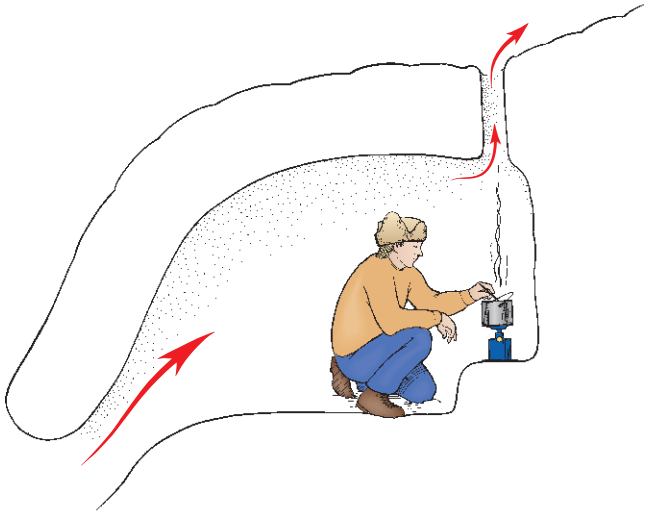
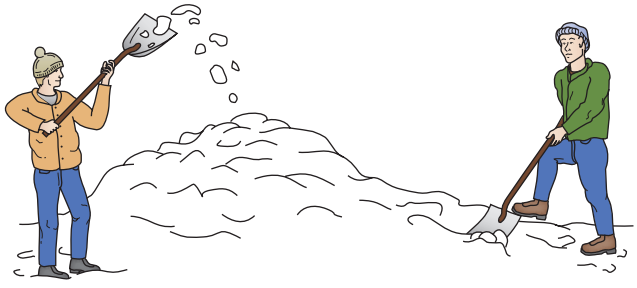


C

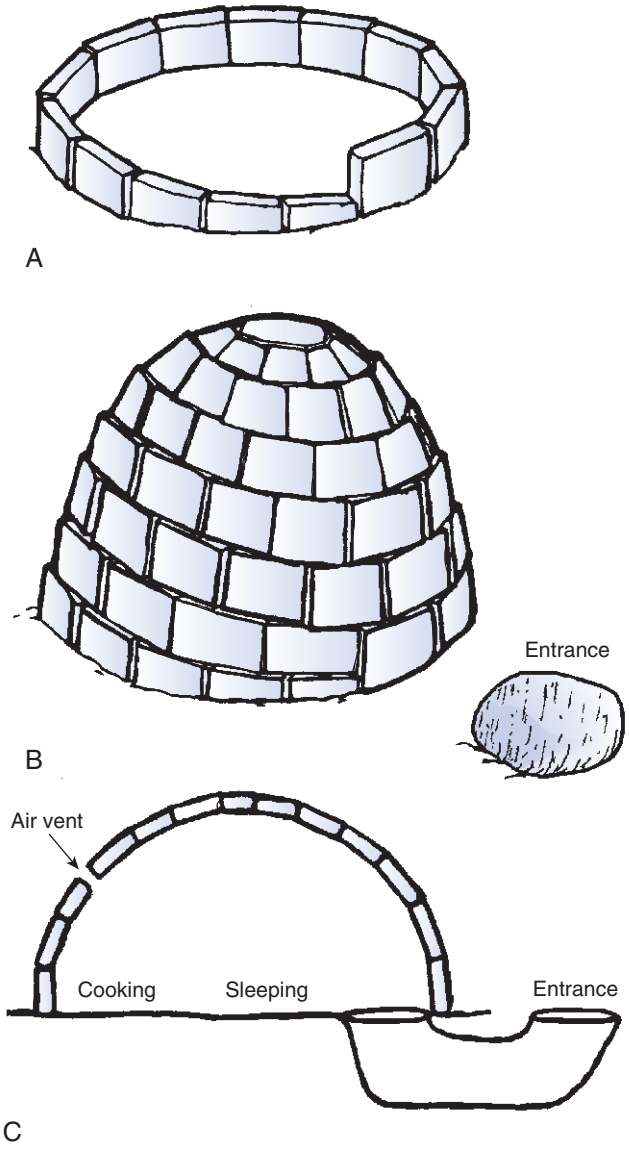
Figure 36-10. **A,** Snow cave entrance. **B,** Snow cave partly closed with snow blocks. **C,** Interior of snow cave.



A

B

Figure 36-11. **A,** Preparing a snow dome. **B,** Completed snow dome.



D

E

Figure 36-12. **A–C,** Stages of igloo construction. **D,** Building an igloo, southeast ridge of Mt. Foraker. **E,** Double igloo for a party of five.

hold one or occasionally two persons. Bivouac sacks are carried for emergencies or for sleeping purposes by climbers on long, alpine-style climbs. Their main value is to protect from wind and rain. Many modern packs have extensions so that when used with a cagoule or anorak (roomy, knee-length, hooded pullover garment), they form an acceptable bivouac sack. Tube tents and plastic bag shelters were discussed in the previous section.

Caution: When spending the night in a snow shelter (e.g., trench, cave, igloo), always have a shovel and flashlight *inside* the shelter within easy reach.

INCREASING BODY HEAT PRODUCTION

Internal body heat production can be increased voluntarily by raising the level of muscular activity and by eating. To obtain maximal heat production from exercise, the body should be well fed and in peak physical condition. This is particularly important for persons with sedentary jobs who participate in vigorous outdoor sports, and for rescue personnel who may be subject to severe, unplanned, and prolonged physical stress. A

suitable physical conditioning program should develop both aerobic and motor fitness. The goal of aerobic exercise is efficient extraction of oxygen from alveolar air. This is best developed by rhythmic endurance exercises such as running, cross-country skiing, cycling, swimming, and using exercise bicycles and Nordic skiing simulators. The most effective activities are those that exercise lower and upper extremities simultaneously. Exercise should be vigorous enough to produce a heart rate of 75% of the age-related maximum (0.75 × [220 minus the participant's age]) for at least 15 minutes 4 days a week. Motor fitness, which includes strength, power, balance, agility, and flexibility, is developed by vigorous competitive team sports, selected calisthenics, and weight-lifting exercises.

ADDING HEAT FROM THE OUTSIDE

Fire Building

The ability to build a fire under adverse conditions is an essential skill that should be practiced by all people who engage in outdoor activities (Fig. 36-13). Not only does a fire provide warmth that may mean the difference between life and death, but it also cheers the spirit, improves morale, serves as a signal to rescuers, and reinforces hopes of rescue and survival. Necessary equipment includes a knife, fire starter, and dry matches. A metal match and a candle should be carried as backup. Matches should be of the "strike anywhere" type stored in a screw-top waterproof container that can be opened with one hand and that has a large, rough striking side or bottom. If this area is too small, a large piece of rough sandpaper should be glued to the bottom or side of the container.[14] The fire starter, which can be purchased commercially or homemade of cotton pledgets impregnated with Vaseline (Fig. 36-14), is stored in a secure container such as an extra match container or plastic film can and is highly recommended, especially for wet climates. A small saw makes the task of gathering firewood much easier. This can be collapsible for easier storage in the pack. However, a saw that does not require assembly, such as the Dandy Saw, is easier to make useable when one is cold, tired, or injured.

To burn, a fire needs air, but not too much air. The fire site should be out of the wind (behind a rock or log or in a snow pit) on rocks or rocky soil. If the fire is built on bare ground, all flammable material such as moss and grass should be cleaned off by scraping the ground surface down to mineral soil over an area at least 4 feet (1.2 m) in diameter. Remember that every year there are reports of signal fires started by lost people that become uncontrolled and burn thousands of acres of forest.

Building a fire requires three types of combustible material: tinder, kindling, and fuel (Fig. 36-15). Tinder is any type of finely divided, highly flammable material, and must be dry. Examples include grass and leaves, inner bark of birch trees, shavings from dry sticks, and small dead sticks. The most readily available natural materials are the small dry twigs found on the lower, dead branches of evergreens. If the outer wood of small branches is wet, it can be shaved off, or the branches can be split lengthwise into several thinner lengths with a knife to expose the dry core. If there is a question about flammability, use fire starter if you have it. The tinder is arranged in lean-to form by placing it against a larger branch, smallest sticks on the bottom and larger ones on top, separated just enough so that air can reach each piece. To conserve matches, one match should be used to light a candle or segment of fire starter, which in turn lights the tinder. The initial flame should be placed under the middle of the lean-to of tinder so that lower, smaller pieces will set fire to higher, larger pieces.

Kindling is larger material, usually pieces of dead branches and branches that have been split lengthwise with a knife. Once the tinder is burning well, these larger pieces of dry wood are added.

Fuel is the largest material, usually huge branches and sections of dead tree trunks several inches or more in diameter. These should be split if an ax is available. Standing dead wood is preferable to wood lying on the ground, and wood that has lost its bark to wood with bark, because both will be drier and less rotten than their alternatives. Fuel is added after the kindling is burning well.

Several times more fuel than the predicted need should be collected. When dead branches are gathered, only those that snap loudly when broken off should be selected. Long sections of dead trees can be shortened by laying them across a fire so that when they burn through, two shorter sections result. Fires generally should be kept small, both to conserve wood and allow them to be approached more closely. The dry wood supply should be protected from wind, rain, and snow as much as possible.

Although the importance of carrying matches in the survival kit cannot be overemphasized, one should also know several ways of starting a fire without matches. A commercial "metal match" is a good backup device. When scraped repeatedly with any sharp, metal edge, it produces showers of sparks that will ignite very dry tinder or fire starter. On a sunny day, a small magnifying glass can focus the sun's rays enough to ignite dry tinder. If you are stranded in an automobile, the cigarette lighter and battery can be used. If a length of wire is available, its midportion can be stripped of insulation and wrapped 7 to 10 times around a dry stick. Touching the wire ends to each of the two battery terminals will cause the wire to become red hot, igniting the stick. The wire should be long enough that the flame is not close to the battery, where it could ignite hydrogen gas produced by the battery. Because fire bows and fire ploughs require considerable practice and several types of dry wood, they have little use under survival conditions.

Carbon Monoxide Poisoning

Open flames in tents, snow caves, and other confined spaces may be dangerous, especially at high altitude and in winter conditions.[12] The first caution is to always keep the possibility of CO poisoning in mind. Provide a shelter with adequate internal space, ventilated as well as possible. Caves and igloos should have ventilation ports at least as big as a ski pole basket, located as high as possible. Avoid prolonged simmering, keep stoves highly pressurized, and use a maximally blue flame. If the flame becomes yellow, turn the stove off and repressurize. For warmth, rely on clothing and bedding as much as possible, rather than keeping a stove going. When cooking, make regular trips outside for fresh air so that early signs and symptoms will be easier to notice. Be aware of the early symptoms of CO poisoning, which include headache, tachycardia, and alteration in mental status.

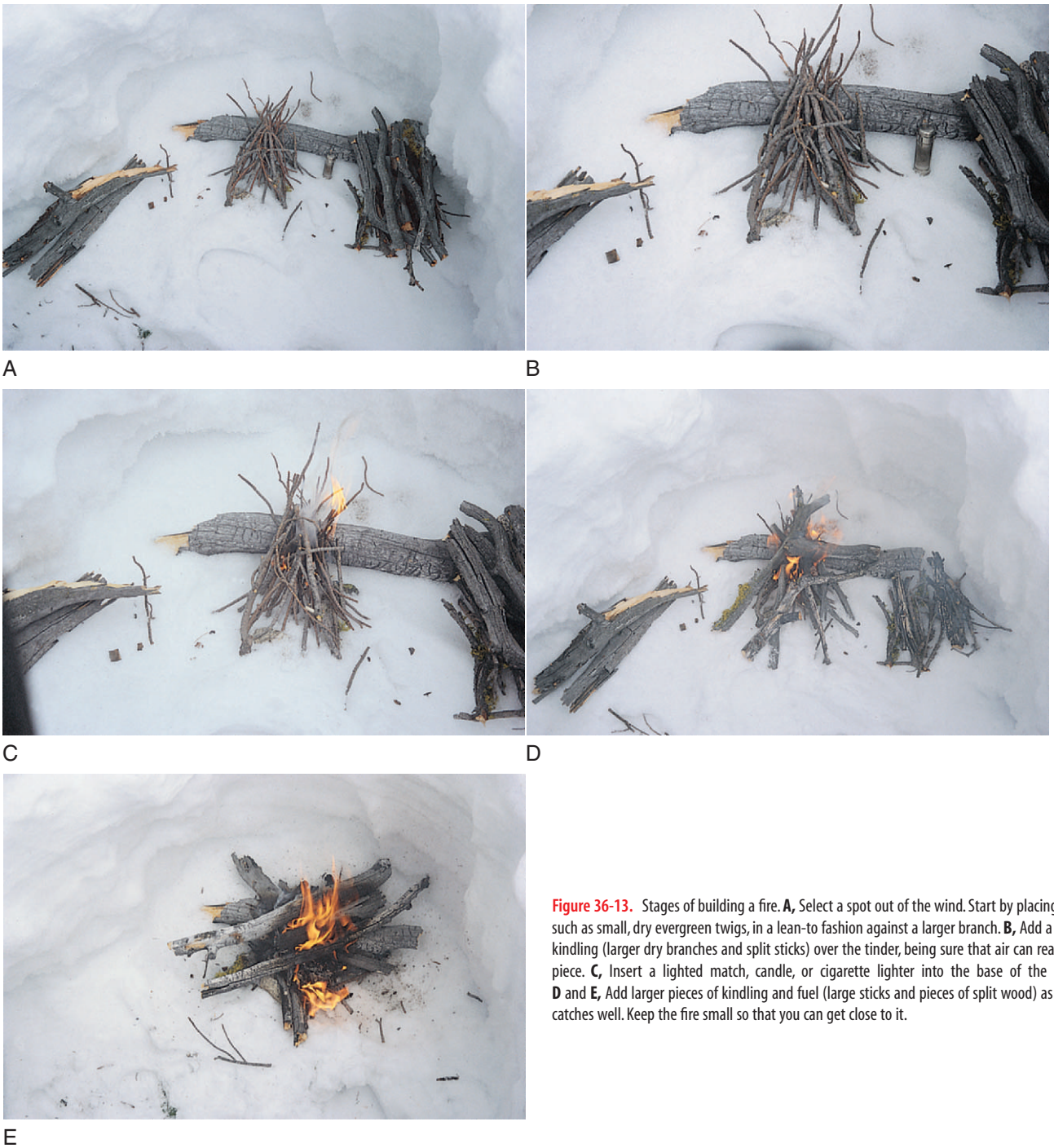


Figure 36-13. Stages of building a fire. **A,** Select a spot out of the wind. Start by placing tinder, such as small, dry evergreen twigs, in a lean-to fashion against a larger branch. **B,** Add a layer of kindling (larger dry branches and split sticks) over the tinder, being sure that air can reach each piece. **C,** Insert a lighted match, candle, or cigarette lighter into the base of the lean-to. **D** and **E,** Add larger pieces of kindling and fuel (large sticks and pieces of split wood) as the fire catches well. Keep the fire small so that you can get close to it.

FOOD

Although most persons faced with a survival emergency worry more about food than anything else, food is usually less important than shelter or water because a person can survive for weeks without food, even in cold weather. Enough water must be available, however, and energy expenditure must be kept to a minimum. Most wilderness parties carry adequate supplies of food; problems arise if food is exhausted, lost, or contaminated. Bare ridges, high mountains above timberline, and dense evergreen forests are difficult places to find wild food, especially in winter. Success is more likely on river and stream banks, on lake shores, in the margins of forests, and in natural clearings.

Figure 36-14. Use of Vaseline-impregnated cotton as a fire-starter. (Photo courtesy of Peter Kummerfeldt.)

Figure 36-15. The three sizes of combustible material, from left to right: fuel, kindling, and tinder. Metal match with striker shown at bottom left. (Photo courtesy of Peter Kummerfeldt.)

Because in most cases the amount of wild food found by an untrained individual will not provide enough calories to replenish the energy expended in searching for it, it is important to always carry extra food for emergencies. This is true even on a short afternoon hike.

Readers interested in the details of obtaining wild food should consult Chapter 65, Living Off the Land.

WATER

Water constitutes about 60% of the body weight of an average young adult male; the value for a female is slightly lower. The percentage of water tends to decrease with age. In a sedentary adult, normal daily water loss includes about 1400 mL of urine, 800 mL through the skin and lungs, and 100 mL in the stool, for a total of 2300 mL daily. Because about 800 mL of water per day is contained in food and 300 mL produced by metabolism, a minimum daily intake of 1200 mL is necessary in a temperate climate at sea level to avoid dehydration.[7] In a hot dry climate, at high altitude, or with exertion, insensible losses and sweating increase considerably, so fluid intake should be increased proportionally. Monitoring urine output determines whether intake is adequate; 1 to 1.5 L of light-colored urine should be excreted per day. Adding fruit flavors and making hot drinks improve the palatability of water. Electrolyte drinks and salt tablets are generally unnecessary in cold or temperate weather because the electrolytes lost in sweat are easily replaced by a normal diet. When water supplies are limited, overexertion is avoided and sweat "rationed."

Almost all surface water should be considered contaminated by animal or human wastes, with the possible exception of small streams descending from untracked snowfields or springs in high, uninhabited areas. At altitudes below 5488 m (18,000 feet), simply bringing water to a boil will kill *Giardia* cysts and most harmful bacteria and viruses. Water can also be disinfected by filtration or addition of chemicals. (See Chapter 61 for details.)

At subfreezing temperatures and in locations above the snow line where liquid water is difficult to find, snow or ice must be melted to obtain water. This requires a metal pot (which should be included in every survival kit), fire-starting equipment, and wood for fuel. The time and effort required to obtain water and decreased thirst in cold weather favor the development of dehydration in cold weather travelers.

In the winter, whenever open water is encountered, individuals should drink their fill of disinfected water, then top off all canteens. Each evening, enough snow is melted to provide water for supper plus a full canteen, which is placed in the bottom of the sleeping bag to keep it from freezing, and is ready for use during the night or for making breakfast in the morning. Before leaving camp in the morning, enough snow is melted to provide everyone with at least a full canteen for the day. Melting ice or hard snow is more efficient than melting light, powdery snow. To avoid scorching the pot, the snow is melted slowly, or water is heated in the bottom of the pot before adding snow. On warmer, sunny days, snow can be spread on a dark plastic sheet to melt.

EMERGENCY SNOW TRAVEL

Travel in deep snow is almost impossible without skis or snowshoes. Snowmobilers, pilots, and other mechanized oversnow travelers should carry snowshoes for emergencies. Even though travel may be unwise for other reasons, wilderness foot travelers in both subarctic and temperate latitudes should know how to improvise snowshoes from natural materials in the event they are stranded by a late- or early-season snowstorm. Emergency snowshoes (Fig. 36-16A) can be made from poles that are 6 feet long, $^3/_4$ to 1 inch (1.9 to 2.5 cm) thick at the base, and $^1/_4$ inch thick at the tip, and sticks $^3/_4$ inch thick and 10 inches long.[1] Twelve long poles and 12 short sticks are needed. For each snowshoe, six long poles are placed side by side on the ground, and the middle point of the poles is marked. One short stick is lashed crosswise to the tail (base) of the poles, and three short sticks are lashed side by side just forward of the midpoint of the poles where the toe of the boot will rest. Two sticks are lashed where the heel of the boot will strike the snowshoe. The tips of the six poles are tied together. Each binding (see Fig. 36-16B) is made of a continuous length (about 6 feet

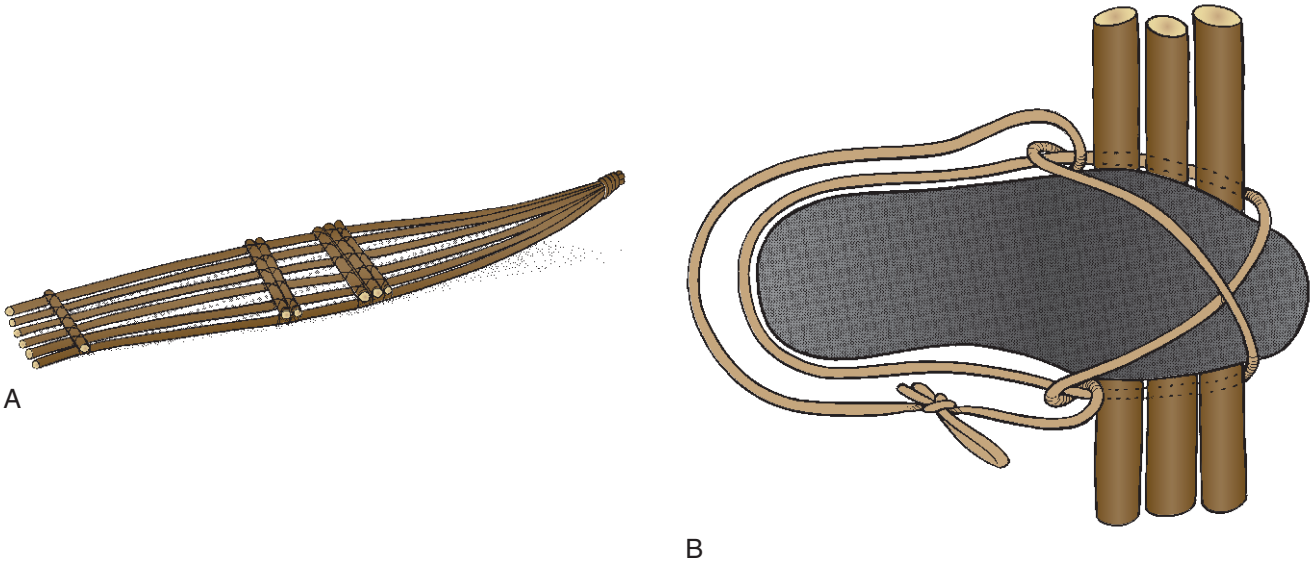


Figure 36-16. **A,** Emergency snowshoe. **B,** Detail of snowshoe binding.

[1.8 m]) of nylon cord (preferably braided because it will eventually fray). The midpoint of the cord is positioned at the back of the boot above the bulge of the heel. Each end of the cord is run under the three side-by-side short sticks at the side of the boot, then up and across the boot toe so that it crosses the other end on top of the toe, forming an X. Then, each end is looped around the cord running along the opposite side of the boot, and the ends are brought around the back of the boot heel. The cord is pulled tight around the boot, and the ends are tied together at the lateral side of the heel.

On walking, the tip of the snowshoe should rise, the boot heel should rise, and the boot sole should remain on the snowshoe.

Snow travelers should avoid stepping close to trees (because of funnel-shaped tree wells around tree trunks), large rocks (because of weak snow or moats around them), and overhanging stream banks. In snow country, the person who falls into a stream or lake should roll repeatedly in powdery snow to wick water from clothing, brushing off the snow after each roll. A fire completes the drying process.[1]

SURVIVAL IN SPECIAL INSTANCES, INCLUDING NATURAL CATASTROPHES

The general principles of survival in all catastrophes are the same, particularly the need for forethought, planning, and keeping emergency equipment, water, and food on hand.[1,18,21] Nevertheless, different types of catastrophes require different approaches, particularly at first. Some common types are discussed next.

Stalled or Wrecked Automobiles

Anyone who drives faces the possibility of spending an unplanned night out in a vehicle.[20] Causes include bad weather, breakdown, running out of fuel, and getting stuck. Winter driving is especially hazardous because of the dangers of driving on snow or ice and the threats of frostbite and hypothermia. Accepting the possibility of trouble, carrying a vehicle survival kit, and giving some thought to survival strategies will help prevent a night out in a car from deteriorating into a life-threatening experience.

Most travelers dress to arrive at a destination and not to survive a night out—"to arrive, not to survive." A vehicle survival kit (see Appendix C) should include extra clothing, blankets or sleeping bags, food, water, signaling equipment, and communications equipment (e.g., citizen's band radio, cell phone). It is also generally better to stay with the vehicle, which provides significant protection and is more visible to rescuers than is a person on foot.

In cold weather, and especially for long-distance travel, drivers should keep their vehicles in the best possible mechanical condition, using winter-grade oil, the proper amount of radiator antifreeze, deicer fluid for the fuel tank, and antifreeze in the windshield-cleaning fluid. Windshield wiper blades that are becoming worn should be replaced. A combination snow brush and ice scraper should be carried. A can of deicer is useful for frozen door locks and wiper blades. Snow tires, preferably studded (illegal in some states), are desirable, but chains should be carried as well. All-wheel drive or four-wheel drive is optimal, and front-wheel drive is superior to rear-wheel drive. The battery should be kept charged, the exhaust system free of leaks, and the gas tank full ("drive on the upper half of your tank!"). The marooned driver should tie a brightly colored piece of cloth (such as a length of surveyor's tape) to the antenna and at night should leave the inside dome light on to be seen by snowplow drivers and rescuers (headlights use too much current). If necessary for heat, the standard recommendation is that the motor and heater can be run for 2 minutes each hour (after checking to see if the exhaust pipe is free of snow). However, a recent newspaper column[15] notes that because it takes more gasoline to start a cold engine than a warm one, one should initially turn the heat up all the way and run the engine

until the inside is comfortable. Then, shut off the engine and wait until it becomes uncomfortably cold (which could be 10 to 30 minutes depending on outside temperature). The engine, however, will still be "warm." Start the engine again and run the heater until the occupants feel warm. Keep repeating this.

CO poisoning is a real threat, so do not go to sleep with the engine running, and keep a downwind window cracked 1 to 2 inches. A reusable CO detector is a wise addition to the survival kit. One or two large candles ("fat Christmas candle" size) should be carried to provide heat and light if the gasoline supply runs out; two lit candles can raise the interior temperature well above freezing. However, resources should be used sparingly because you are never sure how long you will be marooned. Foresight enough to include heavy clothing and blankets or sleeping bags in the cold-weather vehicle survival kit is better than relying excessively on external heat generation. Do not smoke or drink alcohol. If you have to get out of the vehicle, put on additional windproof clothing and snow goggles, and tie a lifeline to yourself and the door handle before moving away from the vehicle. In a blizzard, visibility can be as low as 12 inches.

Aircraft Accidents

Passengers on aircraft are routinely lectured on locations of exits and procedures for emergencies. The safest place to sit is as far back in the tail as possible.[21] In a crash, this frequently breaks off, and most survivors of crashes were sitting in this area. When a crash is imminent, tighten the seat belt, link arms with people on either side, bend your head so that your chin is firmly on your chest, and lean forward over a folded coat or blanket. After the aircraft finally stops moving, leave it as instructed. Assist injured companions to leave, then get everyone away from the immediate area to avoid the hazard of fire. Do not stop to gather personal belongings or luggage. After the fire risk is over, revisit the wreckage to salvage food, clothing, water, and other useful items.

Remember that aircraft fuselages are poorly insulated. Unless a stove is available, survivors are usually better off constructing a shelter that can be heated with a fire (as described earlier), outside but near the craft. Batteries can be used as fire starters. Oil and gasoline can be used as fuel if poured into a container full of dirt or sand.

Unless there is no chance that a search will be mounted to find the aircraft, you should remain close to it rather than trying to go for help. It is much easier for searchers to spot a crash site than survivors on foot in the wilderness.

Floods

Flooding in wilderness areas may be caused by thunderstorms, unusual storms such as hurricanes, and rapid melting of ice and snow during heat waves. Flash floods in canyons can occur both during and after rains and can be caused by rains many miles upstream. Submarine earthquakes can cause huge tidal waves (tsunamis) that may drown hundreds of thousands of shore dwellers. Whenever traveling or camping near bodies of water in the wilderness, one should keep in mind what might happen if overflow or rising water should occur. When traveling or camping in box canyons, below dams, close to banks of water channels, and on low ground such as valley bottoms, one should have a well-thought-out exit strategy in case flooding occurs. If along the shore and you receive a radio report of an impending tsunami, or if you see the sea recede and expose a large expanse of sea bed, flee immediately and seek high ground as far from the shore as possible.

Persons caught in moving water have no recourse other than trying to swim to the side or hang on to floating debris, trying to hang onto emergency supplies in the process. Never try to cross an area of moving water unless you are sure it is no more than knee-deep.

Thunderstorms

Dangers from thunderstorms include flooding (discussed earlier), lightning strike (see Chapter 3), and exposure, including wetting and hypothermia. Severe thunderstorms may produce hailstones of various sizes, including stones large enough to harm a struck individual. During a thunderstorm, it is wise to seek shelter. This requires getting into raingear and avoiding heavy rain and hailstones by getting into a metal vehicle or dense grove of trees. If you are on water, head for shore as soon as threatening weather approaches. If in an area where you are the highest object around, leave immediately or crouch down on your haunches. Avoid small buildings, electric wires, metal objects, rocky overhangs where you may be hit by side flashes from ground currents, solitary trees, and trees taller than surrounding trees.

Tornados

These funnel-shaped clouds typically form on a hot spring or summer afternoon in the midwestern United States. They are usually preceded by large cumulonimbus clouds, from the bottoms of which a careful observer occasionally can see the formation of the tornado as a dimple that elongates into a typical conical funnel cloud. Persons who witness this phenomenon should leave immediately by vehicle in a direction opposite to the tornado's likely path, or seek shelter on the spot. In houses, the best shelters are small, interior rooms with non–weight-bearing walls, preferably in the basement but on the ground floor if no basement exists. Ditches and small buildings are not safe. Caves and outdoor root cellars are fairly safe, but vehicles are not.

Hurricanes

These severe tropical storms are extreme cases of the vortices of wind and rain that frequently form over tropical seas. By definition, they contain winds of 119 kph (74 mph) or stronger. They require ocean temperatures of 25.5°C (78°F) and a spawning area at least 10 degrees latitude from the equator so that the earth's rotation is strong enough to start them in motion.

Deaths due to hurricanes are usually due to high winds, flooding from heavy rain, or coastal flooding due to storm surges. With modern technology, the formation of a tropical cyclone (tropical low) and its change into a tropical storm and then a hurricane is predictable and reported in detail by the National Oceanic and Atmospheric Administration. Persons at risk should keep the radio or television on so they will be aware when there is a need for evacuation. Most casualties are persons who remained against advice or were caught by winds and flooding before they could get far enough inland. Persons who live in hurricane-prone areas should have emergency caches of water and nonperishable food, clothing, bedding, a first-aid kit with supplies of any regular medications, emergency lights (candles, matches, flashlights, spare batteries), cell phone, and battery-operated radio, should keep the car's gas tank full, and

should be ready to head inland with these supplies as soon as advised to do so. Before they leave, they should board or shutter all windows, take inside any yard objects that might be blown away, and shut off the gas and electric power.

Earthquakes

Earthquakes can be anticipated on geologic grounds but their time of occurrence cannot be predicted with much accuracy. They are most common along known fault lines, allowing decisions about the strength of building construction to be based on risk to some extent. If you are warned of a possible earthquake because of early tremors, keep your radio on and be sure you have supplies as listed earlier under "Hurricanes." Turn off electricity, water, and gas if advised to do so. Remove heavy and fragile objects from high shelves, ceilings, and other places from where they might fall. If an earthquake strikes while you are at home, stay away from mirrors and windows and seek shelter in a corner or under a table, preferably in a basement.

Volcanoes

Almost all volcanoes near inhabited areas are monitored by seismograph (see also Chapter 15). Because increasing steam releases, small eruptions, increases in seismographic activity such as small to major tremors, and other signs of impending volcanic activity frequently precede major eruptions, residents, workers, tourists, hikers, and campers usually have time to evacuate. Danger to humans comes from earthquakes, explosions, lava flows, mud flows, ash, and flooding. Because many fatalities in eruptions such as that of Mt. St. Helens were in persons who were warned of the danger but refused to evacuate, the obvious advice is to leave the area quickly when advised. The forces released during an eruption are too great to permit survival of unprotected humans.

NAVIGATION

Even if in a familiar area, backcountry travelers should always carry a compass, map, and altimeter. Prior training and experience in map reading and compass use are necessary.[11] An excellent type of compass for the layperson is the Swedish Silva compass, designed to be used in the sport of orienteering. The compass reading is always believed, even if at odds with "gut feelings" about direction and location. Topographic maps are available at most outdoor stores in both the 7.5- and 15-minute series and can be ordered from the U.S. Geographic Survey (1-888-ASK-USGS or on the Internet at www.USGS.gov).

Travelers without a compass should still be able to find rough directions. At night, north can be found by identifying the Big Dipper (Northern Hemisphere only) and following the "pointers" (farthest stars on the bowl of the dipper) to the North Star (Polaris), the most distal star in the handle of the Little Dipper, which is located about halfway between the Big Dipper and the W-shaped constellation Cassiopeia. On a sunny day, a non-digital watch set to standard time can be used to find direction. When the hour hand is pointed toward the sun, south will be one-half of the shorter of the two distances between the hour hand and 12 o'clock.

The availability of small, hand-held GPS units has revolutionized wilderness navigation but not replaced the need for good map and compass skills. Detailed information on wilderness navigation is found in Chapter 85.

WEATHER FORECASTING

Travelers should check expected weather conditions before entering the backcountry.[4,9,13,22] With radar, satellite technology, and other advanced techniques, modern weather forecasting is accurate but not infallible. Local and national radio and television networks broadcast local and regional reports hourly. The Weather Channel (on television) has frequent forecasts and is also available on the Internet at www.weather.com. The best source of up-to-date local weather information is the National Weather Service, which broadcasts 24 hours a day at frequencies from 162.400 to 162.550 MHz on very-high-frequency (VHF) FM. Multichannel radios with a weather band receive these frequencies, and inexpensive, lightweight radios receive only these frequencies.

When evaluating avalanche conditions, travelers must know the weather conditions over the previous few days as well. The U.S. Forest Service provides information on avalanche conditions in many mountainous areas, especially in western states, available online at www.avalanche.org. (See Appendix in Chapter 2.)

Because weather information from outside sources may be impossible to access in a true wilderness environment, the wilderness traveler should be able to predict weather to some extent. This requires knowledge of both basic meteorology and local weather patterns, particularly the significance of cloud patterns, wind directions, barometric pressure changes, and temperature changes. Backcountry weather forecasting is an inexact science, however. In Michael Hodgson's words, "Predictions relative to weather are only educated guesses, never statements of fact. Always be prepared for the worst."[9]

The major factors that influence weather are solar radiation, the components of the atmosphere (especially water vapor), topography, physical properties of large water bodies and land masses, and the effects of the earth's daily and yearly rotations on the amount of solar radiation that reaches each part of the earth.

The earth's atmosphere is constantly in motion. The primary motion is basically circular, involving vertical upward motion as air is heated at the equator, then horizontal motion of this warm air toward the poles, where it cools, descends, and moves back toward the equator to replace heated, upward-moving air. If the earth did not rotate, this circular motion would be in a north-to-south direction. The earth's rotation, however, causes the direction of movement to be deflected to the right (in the Northern Hemisphere), so the movement becomes more west to east (Coriolis effect) in the northern temperate zone (area between the Tropic of Cancer and the Arctic Circle). Because the earth's surface is covered unevenly by land masses, water bodies, and polar ice, and because these regions are heated irregularly as the earth rotates on its axis, systems of moving cold and warm air masses are formed. Cold, moist air masses tend to form over cold (polar) seas and warm, moist air masses over warm seas. Cold, dry air masses tend to form over cold (polar) land and warm, dry air masses over warm land. The polar regions have large, stable areas of cold, high-pressure air (polar highs), and the equatorial regions (between 10 degrees south and north of the equator) have a large area of stable, warm, moist, and low-pressure air (doldrums).

Atmospheric pressure in air masses depends on their temperature: higher in cold air masses and lower in warm air masses.

Winds are caused by air moving from a high-pressure to a low-pressure area; the greater the pressure difference, the higher the wind speed.

Each air mass, which may cover hundreds to thousands of square miles of the earth's surface, is nearly homogenous for temperature and humidity. In the northern temperate zone, air masses generally move from west to east in North America. Exceptions include cold, relatively dry air masses that move south from northern Canada in the winter and warm, moist air masses ("monsoons") that move north from the Gulf of Mexico in the summer.

Cold air masses move faster than warm air masses (25 to 35 mph versus 10 to 20 mph). The boundaries between air masses are called *fronts*. Frontal air is generally unstable and frequently an area of violent weather. A cold front is an area where heavy, cold air is displacing lighter, warm air, frequently by coursing under it. A warm front is an area where lighter, warm air is replacing a retreating mass of heavier, cold air.

Warm, low-pressure air masses are called *lows,* or *cyclones;* cold, high-pressure ones are called *highs* or *anticyclones*. Because air flows from areas of high pressure to areas of low pressure, lows are characterized by winds blowing from their edges toward their centers and highs by winds blowing from their centers toward their edges. The Coriolis effect causes winds in the Northern Hemisphere to move in a clockwise direction from the center to the periphery in a high and in a counterclockwise direction from the periphery to the center in a low. Understanding these principles can help the traveler interpret shifting wind directions as highs and lows pass.

Weather within an air mass is controlled by its moisture content, the relationship between land surface and air mass temperatures, and terrain features such as up- or down-slopes. Precipitation can occur in either a high or a low, but is more common in a low. The amount of moisture in an air mass can be described as the air's *relative humidity,* which is the amount of water vapor in the air compared with the amount it could hold at its current temperature without condensation occurring. Warm air can hold more water vapor than cold air. The term *dew point* refers to the temperature at which the relative humidity becomes 100% and the water vapor in air starts to condense. Because the temperature drops about 2.2°C (4°F) for every 305 m (1000 feet) of ascent (1.6°C [3°F] if the air is moist; 3°C [5.5°F] if dry), rising air cools, and descending air warms. Water vapor in rising air will condense when the air cools to its dew point, producing fogs, clouds, and precipitation.

The wilderness traveler should be able to identify the different types of common clouds and know their significance. Clouds are divided into four types based on level, form, and association with precipitation (Figs. 36-17 and 36-18). The highest are *cirrus* clouds, which develop above 6000 m (20,000 feet). These ice-crystal clouds frequently appear thin, veil-like, and delicate. The feathery ones are called "mare's tails." The sun shines brightly through cirrus clouds. Middle-level clouds (2000 to 6000 m [6500 to 20,000 feet]) have the prefix *alto-* (e.g., altocumulus). Low-level clouds (2000 m or below) have the prefix *strato-* or suffix *-stratus* (e.g., stratocumulus, nimbostratus.) These terms are also used to indicate clouds in sheets or layers at higher altitudes (e.g., altostratus, cirrostratus).

Clouds of high vertical development (500 to 18,200 m [1600 to 60,000 feet]) are the larger types of cumulus clouds frequently associated with heavy precipitation. Developing ones that ascend to 9100 m (30,000 feet) and have billowing tops

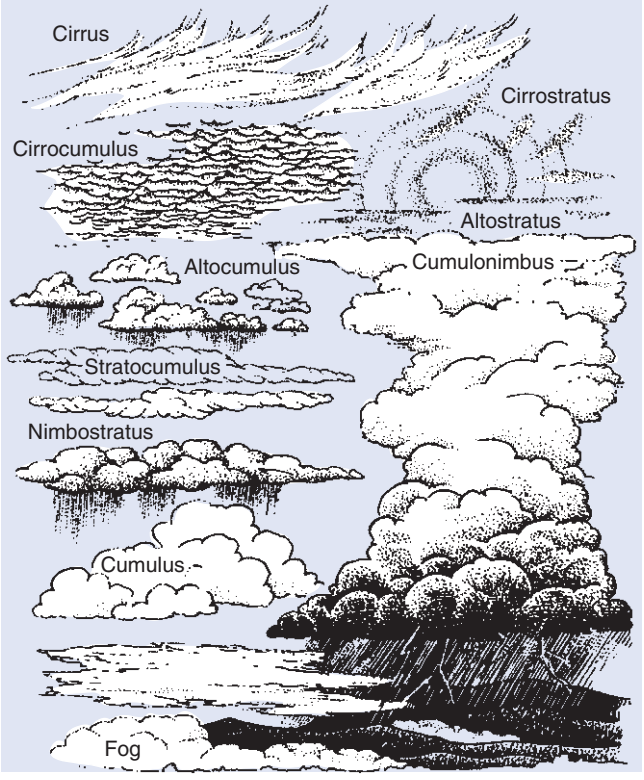


Figure 36-17. Different types of clouds. (Modified from Woodmencey J: Reading Weather: Where Will You Be When the Storm Hits? Helena, MT, Falcon, 1998.)

resembling cauliflowers are called cumulus congestus. The largest ones rise to 18,200 m (60,000 feet) or above, have anvil-shaped tops, and are called cumulonimbus. Both types have darkened bases. Because of these clouds' height, precipitation falls long distances through areas of super-cooled water droplets. Depending on conditions, soft hail (graupel) or huge snowflakes may result.

The prefix *nimbo-* or suffix *-nimbus* indicates that a cloud is associated with precipitation. The term *cumulus* refers to any clouds that present as large or small groups of separate fluffy masses (e.g., cirrocumulus, altocumulus, cumulus fractus, depending on their altitude). Cumulus humilis refers to the middle-level, white, cottony clouds with white bases seen in fair weather.

In the inland parts of North America, the worst winter weather (blizzards) is associated with moving masses of warm air (lows) and the worst summer weather (severe thunderstorms) with either rising masses of warm air or moving masses of cold air.

There are two types of thunderstorms, both associated with cumulonimbus clouds (thunderheads). *Frontal* thunderstorms result when an arriving cold front slides under a warm air mass. *Air mass* thunderstorms consist of two subtypes: *orographic* thunderstorms, which result when moist air is forced up over a mountain range causing thunderstorms on the windward side, and *convective* thunderstorms, which result from rising vertical currents of air caused by heating of ground or water by solar

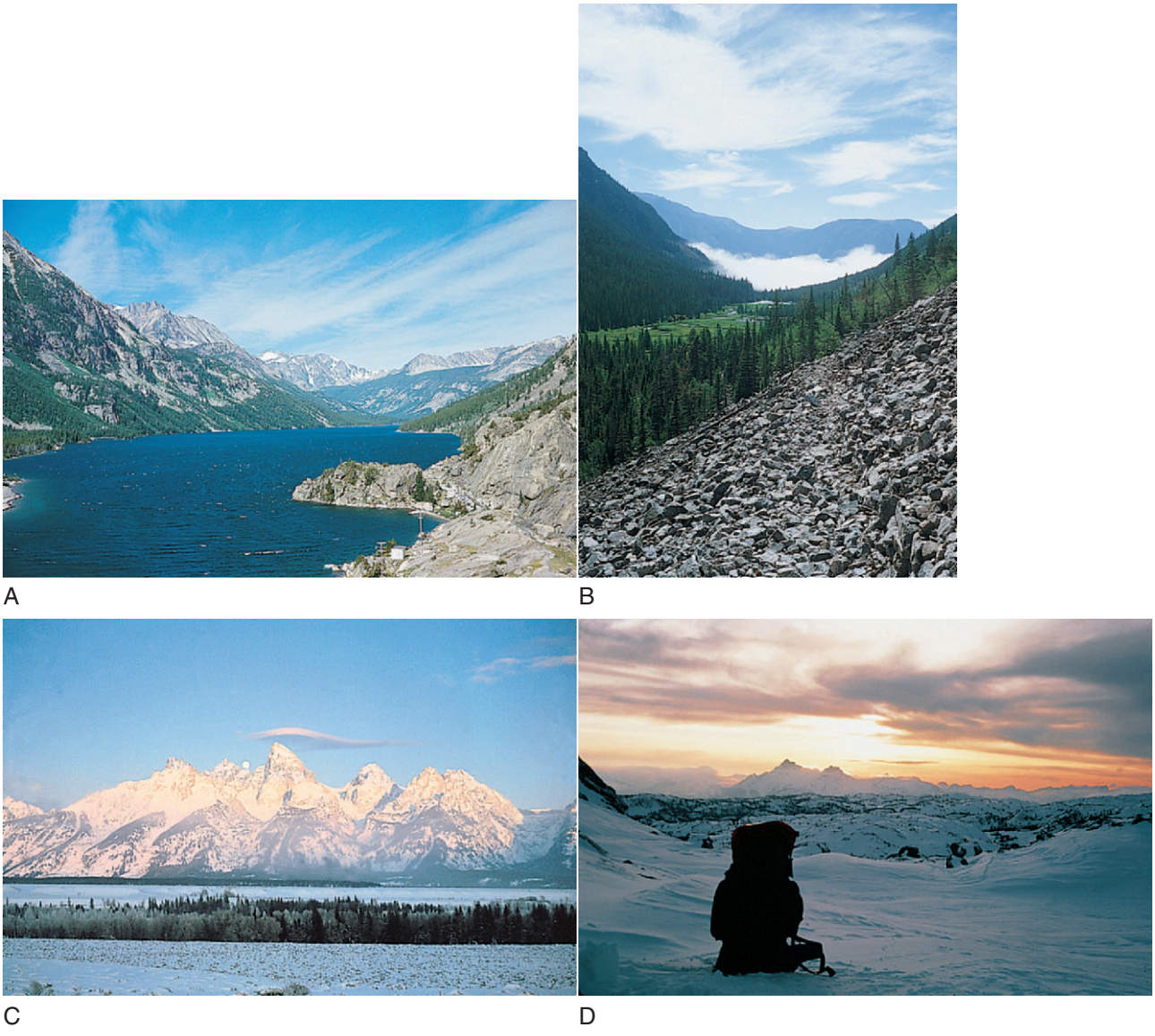


Figure 36-18. **A** and **B,** Cirrus. **C,** Altocumulus lenticularis. **D** and **E,** Altostratus.

radiation. These latter are the typical afternoon or early-evening thunderstorms, which may spawn tornadoes when severe.

An advancing warm front bringing precipitation has a predictable series of lowering, darkening cloud formations: scattered cirrus, sheets of cirrostratus, sheets of altostratus, then nimbostratus. Precipitation can begin with the appearance of either altostratus or nimbostratus clouds. The combination of cirrus followed by cirrostratus and altostratus clouds usually predicts precipitation within 24 to 48 hours. Cumulus congestus and especially cumulonimbus clouds indicate thunderstorms.

The wilderness traveler can anticipate weather to a limited extent by using a thermometer and barometer (or altimeter), noting wind direction, and identifying clouds. Remember, however, that even with the best modern forecasting equipment, the National Weather Service itself is not accurate 100% of the time, especially in forecasting conditions in a small area of wilderness. Nevertheless, we can say that if the signs that predict a severe storm are present, the storm is not guaranteed, but if the signs are absent, a severe storm is unlikely.

Some digital watches have altimeter and barometer features. The senior author prefers to carry an aneroid altimeter (which does not require batteries) and that serves as both altimeter and barometer. Both instruments measure air pressure, but are geared oppositely, so that the altimeter registers higher as altitude increases (pressure drops), whereas the barometer registers higher as pressure rises. During mountain travel, barometer and altimeter readings change as altitude changes, regardless of the

Figure 36-18, cont'd. **F,** Altocumulus. **G,** Nimbostratus. **H,** Cumulus humilis. **I,** Cumulus congestus. **J,** Cumulonimbus.

presence of low- or high-pressure areas. In the evening, however, the moveable arrow on the rotating face of the aneroid altimeter can be set at the present altitude or the altitude (or barometric pressure readout) recorded. The next morning, the evening reading is compared with the morning reading. Most severe winter storms in the temperate zone are accompanied by an altimeter rise of 150 to 240 m (500–800 feet), equivalent to a barometer drop of 0.5 to 0.8 inch of mercury (in Hg) or 15 to 24 mb. A rapid altimeter rise (barometer drop) may signify high winds or a short, severe storm, and a slow, steady altimeter rise (barometer drop) a long storm. A rapid altimeter drop (barometer rise) may also mean high winds (Table 36-3).

TABLE 36-3. Barometer and Altimeter Forecaster

BAROMETER CHANGE	ALTIMETER CHANGE	TIME (hr)	WEATHER CHANGE EXPECTED
.04–.07 in Hg (1–2 mb)	40–70 ft (12–20 m)	3	Some changes should be expected. May be slow to develop.
.08+ in Hg (2.5+ mb)	80+ ft (24+ m)	3	Significant changes should be expected. May be developing rapidly.
.20–.40 in Hg (6–12 mb)	200–400 ft (60–120 m)	12–24	Some changes should be expected. May be slow to develop.
.40+ in Hg (12+ mb)	400+ ft (120+ m)	12–24	Significant changes should be expected. May be developing rapidly.

Hg, mercury; mb, millibar.
From Woodmencey J: Reading Weather: Where Will You Be When the Storm Hits? Helena, MT: Falcon, 1998, Table 15, p 113.

Because of their different meteorologic features (as described previously), the marked altimeter rise (barometer drop) seen in a severe winter storm typically does not occur in a severe summer thunderstorm. Because cloud patterns are always changing, they must be observed at regular intervals throughout the day, to develop the ability to predict their significance with any confidence.

Measuring and recording the air temperature several times daily (remembering that temperature normally drops with increasing altitude) and noting wind directions can confirm an advancing low or high when coordinated with other observations. If a low is moving directly toward you, the wind will shift so that it is blowing from the southeast or south (Northern Hemisphere). If the wind comes from the southwest or west, the low is passing north of you; if from the northeast or east, it is passing south of you. An easy way to check this is to stand with your back to the wind. The low-pressure area will be in front of you and to your left.[4] As a front passes, the wind will gradually shift 180 degrees.

The shores of large bodies of water have characteristic wind patterns because of the difference in specific heats of water and land leading to different warming and cooling rates. In the morning, because land warms and cools faster than water, breezes begin to blow from water to shore as the air over land rises. As the land cools in the evening, breezes begin to blow from land to water.

Mountain Weather

Mountain weather is more unpredictable than weather in lower, flatter country. Winds frequently blow up and down mountain valleys regardless of their orientation because of the funnel effect of the valley and temperature differentials caused by solar radiation. The funnel effect may also cause heavy snowfalls at passes or the higher ends of valleys.

On a sunny day, the sun warms mountaintops and high slopes first, the warm air rises, and winds blow up the slope. In the evening, the tops and high slopes cool first, the cool air descends, and winds blow down the slope. Glaciers and large snowfields produce significant cool, down-slope winds. Except on the clearest days, mountaintops may have clouds over them or nearby because of the up-slope winds that carry moist air high enough to reach its dew point. In the summer, mountains warm up during the morning and early afternoon, creating cumulus congestus and cumulonimbus clouds that produce thunderstorms, lightning, and hail. Therefore, the standard caution for summer climbers is to arise early and reach the summit well before noon.

Lightning usually precedes rain in a thunderstorm and can strike up to 5 miles (8 km) away from the storm. Distance is estimated by counting the seconds between the lightning flash and the first noise of thunder: 5 seconds equals 1 mile (count: "one-and-two-and, etc.," at a speaking pace slightly faster than normal).

Precipitation is frequently heavy on the windward side of large mountain ranges; the leeward sides are usually much drier and in low areas may actually be desert. Stationary lens-shaped clouds (altocumulus lenticularis) are frequently seen near mountaintops in windy weather and signify high winds at the summits. High winds also produce long snow plumes from summits and ridges. The summit wind speed will be about twice the valley wind speed.[22]

Winds blowing at right angles to a mountain range tend to concentrate at any gaps or passes in the range, creating high winds due to the Venturi effect. Warm down-slope winds in the winter (Chinook or foehn winds) are produced when cool, dense air blowing over mountaintops loses its moisture as precipitation on the windward side; the drier air then warms rapidly as it descends on the lee side. These warm winds can melt snow rapidly.

Weather is more stable at some times of the year than at others, but the times vary by geographic area. For example, in the northern Rocky Mountains, the best time to hike or climb in the summer are the last week of July, all of August, and the first two weeks in September. The best time to ski-tour or climb in the winter is February. In Alaska, the best winter climbing weather is during February and in spring and summer from April through June. In the Himalaya and Karakorum, the best climbing weather is immediately before and after the summer monsoon (a seasonal, northward flow of warm, moist air from the Indian Ocean).

The principal value of understanding weather signs is in predicting severe, life-threatening storms and providing adequate time to seek shelter or the option to stay put rather than try for a summit. Travel in severe weather, especially above timberline, should never be undertaken casually.

Summary of Backcountry Weather Forecasting

1. Blue sky, a few cirrus or cumulus humilis clouds, cool temperatures, low to medium winds, and a steady or dropping altimeter (steady or rising barometer) are predictors of good weather.
2. A lowering cloud pattern (cirrus followed by cirrostratus, altostratus, and nimbostratus), rising temperatures, wind freshening and shifting to blow from the southeast or south, and an altimeter rise of 152 to 244 m (500–800 feet) or barometer drop of 0.5 to 0.8 in Hg (15–24 mb) indicate a possibly severe winter storm.
3. Building cumulus congestus clouds changing to cumulonimbus clouds indicate probable thunderstorms with lightning and possibly hail. A thunderstorm is frequently preceded by a rush of cold air (cold front).
4. Signs that a severe winter storm is abating include clouds thinning, cloud bases rising, temperature falling, altimeter dropping (barometer rising), and the winds shifting to blow from the north or northwest.

SANITATION

Adherence to proper habits of cleanliness and sanitation is as important in the wilderness as at home.[2] Hands should be washed with biodegradable soap after urinating or defecating, before cooking and eating, and before dressing open wounds. If soap is unavailable, snow or the cleanest available plain water is used. Dishes and pots are washed with hot water and biodegradable soap and rinsed well to remove all soap. Waste water is scattered over a wide area of ground and never dumped into a body of water. Bathing and washing clothes should be done at least 60 m (200 feet) from bodies of water, using water in a container. Gloves, preferably rubber or vinyl (impermeable) ones, are worn when handling moist animal or human tissues (be aware of possible latex allergy).

For defecation, campers should dig a "cat-hole" at least 18 inches (46 cm) deep, 6 inches (15 cm) wide, downhill, and at least 60 m (66 yds) from camp, water sources, or snow to be melted for water. Cover feces completely with dirt or snow. No camp should be within 60 m (66 yds) of a lake or stream, which represents 70 to 80 steps for most adults.

Travelers should urinate on rocks or dirt, not on green plants. When sleeping in a tent or snow shelter, a 500-mL, wide-mouth polyethylene bottle can be used as a urinal to avoid having to go outside, especially at night. Funnels designed for use with the bottle are available for women.

PSYCHOLOGICAL AND ORGANIZATIONAL ASPECTS OF SURVIVAL

Dealing with the psychological aspects of survival is as important as confronting physical and environmental factors. As mentioned previously, the dangerous mindset that keeps a person continuing on a hazardous course, such as completing a climb or reaching a distant campsite despite conditions that would make a more prudent person bivouac or turn back, is a recurring scenario.[6] In a survival emergency, a person with survival training, adequate oxygen, a stable body temperature, shelter, water, and food may still die if unable to withstand the psychological stress. Conversely, persons have survived amazing hardships with little more than a strong determination to live. Individual reactions, however, cannot be predicted in advance. Groups faced with emergencies testify that courage and leadership appear in unexpected places.

Still, if persons possess the necessary skills, have at least a minimum of survival equipment (see appendices to this chapter), and have given some forethought to techniques of wilderness survival, the odds are strongly in their favor. Medical personnel have the advantage of being trained to suppress panic. Fear and surrender are normal reactions that must be opposed by whatever psychological tools are available. In some cases, religious faith or the desire to rejoin loved ones have been credited with survival.

Anxieties that paralyze action include fear (of the unknown, being alone, wild animals, darkness, weakness, personal failure, discomfort, suffering, and death) and panic. Panic, the uncontrolled urge to run away, interferes with good judgment, resulting in inappropriate actions such as abandoning weaker companions, dividing the party, and discarding vital survival equipment. Useless flight saps available energy, leads to exhaustion, and hastens death.

Other psychological reactions include apathy and the normal desires to be comfortable and to avoid pain.[18] Apathy is giving up, a state of indifference, mental numbness, surrender, and unwillingness to perform necessary tasks. The victim shows resignation, quietness, lack of communication, loss of appetite, fatigue, drowsiness, and withdrawal. Apathy in one's self is overcome by faith in abilities and equipment and belief in survival and the possibility of rescue. Apathy in others is combated by communicating plans and positive feelings about resources and outcomes to them, and including all group members in planning and survival activities.

Comfort is not essential to survival. Marked discomfort (from injuries, illnesses, thirst, hunger, excessive heat or cold, sleep deprivation and exhaustion) is inevitable in a survival situation and must be tolerated in order to live. As illustrated by the scenario described at the start of this chapter, there are many accounts of adventurers who have survived many days with severe injuries such as open fractures because of the will to live, or who, despite multiple injuries, have dragged themselves for miles to find help.

Providing an ill or injured party member with psychological support is important. This includes appearing calm, unhurried, and deliberate yourself while trying to encourage optimism, patience, and cooperation. A person with a minor injury or illness should be encouraged to self-evacuate, accompanied by at least one healthy party member. When a person with a severe injury or illness needs to be evacuated, the party must decide whether to use the resources at hand or to send for help. The decision will depend on the weather, party size, training, available equipment, distance, type of terrain involved, type of injury or illness, victim's condition, and availability of local search and rescue groups, helicopters, and other assistance. Unless the weather is excellent, party strong and well equipped, route short and easy, and victim comfortable and stable, the best course of action generally is to make a comfortable camp and send the strongest party members for help. A written note should include each victim's name, gender, age, type of injury or illness, current condition, and emergency care given; the party's resources and location (preferably map or GPS coordinates); and names, addresses, and telephone numbers of relatives. The victim who

must be left alone (e.g., because of small party size) should have an adequate supply of food, fuel, and water.

As soon as you realize that you are lost, stop, sit down in a sheltered place, calmly go over the situation, and make an inventory of your survival equipment and other resources. If it is cold or becoming dark, start a fire and eat if you have food. Take out your map or draw a sketch of your route and location based on natural features. Then, unless you know your location and can positively reach safety before dark, immediately start to prepare a shelter for the night. Do not allow yourself to be influenced by a desire to keep others from worrying or the need to be at work or keep an appointment. Your life is more important than someone else's peace of mind.[8]

If you are alone and unquestionably lost, and especially if injured, you must decide whether to wait for rescue or attempt to walk out under your own power. If rescue is possible, it is almost always better to use the time to prepare a snug shelter (before dark) and conserve your strength. If you decide to leave, mark the site with a cairn or bright-colored material such as surveyor's tape, leave a note at the site with information about your condition, equipment, and direction of travel, and then mark your trail. These actions will aid rescuers and enable you to return to the site if necessary. Travel should never be attempted in severe weather, desert daytime heat, or deep snow without snowshoes or skis. If no chance of rescue exists, prepare as best possible, wait for good weather, and then travel in the most logical direction.

The best way for a lost or stranded person to aid potential rescuers is to do everything possible to draw attention to his or her location. Most modern rescues utilize ground parties, helicopters, and fixed-wing aircraft. When the latter locate victims from the air, they can drop supplies and vector helicopters or ground parties to the site. Besides radios, cell phones, and other electronic equipment, signaling devices are either auditory or visual. Three of anything is a universal distress signal: three whistle blasts, three gunshots, three fires. The most effective auditory device is a whistle. Blowing a whistle is less tiring than shouting, and the distinctive sound can be heard farther than a human voice. An effective visual ground-to-air signal device is a glass signal mirror with a sighting device, which can be seen up to 10 miles away but requires sunlight. Special rescue beacons are available and can be carried as emergency equipment. These include strobe lights, laser signal lights, special beacons with both signaling and GPS capability, and PLBs.

Smoke is easily seen by day and a fire or flashlight by night. On a cloudy day, black smoke is more visible than white; the reverse is true on a sunny day. White smoke stands out well against a green forest background but not against snow. Black smoke can be produced by burning parts of a vehicle, such as rubber or oil, and white smoke by adding green vegetation to a fire. The lost person who anticipates an air search should keep a fire going with large supplies of dry, burnable material (wood and brush) and have a large pile of cut green vegetation close by. When an aircraft is heard, the dry materials are placed on the fire, allowed to flare, and then armloads of the green vegetation are piled on top. This produces lots of smoke and a hot thermal updraft to carry it aloft.

Ground signals (e.g., SOS, HELP) should be as large as possible—at least 3 feet wide and 18 feet long—and should contain straight lines and square corners, which are not found in nature. They can be tramped out in dirt or on grass or can be made from brush or logs. In snow, the depressions can be filled with vegetation to increase contrast.

Many pilots do not know the traditional 18 international ground-to-air emergency signals. These have been replaced by the following five simple signals adopted by the International Convention on Civil Aviation.[18] Although in the age of helicopters these are not as important as they once were, they are worth remembering:

V: I require assistance.
X: I require medical assistance.
N: No
Y: Yes
→: I am proceeding in this direction.
Air-to-ground signals include the following:
Message received and understood: rock plane from side to side.
Message received but not understood: make a complete right-hand circle.

When using cell phones, radios, and other electronic devices, persons should move out of valleys and gullies to higher elevations if possible. Operational pay phones in campgrounds closed for the season or other facilities can be used to call for help. Most will allow 911 or another emergency number to be dialed without payment, but carrying sufficient coins and memorizing your telephone credit card number are recommended.

Protection from Wild Animals

Although persons in a survival situation often worry about wild animal attacks, these are rare. Many wild animals flee when confronted with a shouting, moving human. Exceptions include polar bears, grizzly bears, moose, bison, cougars, jaguars, wild pigs, elephants, lions, tigers, water buffalo, leopards, wolverines, females with young (particularly bears, moose, elk, and deer), rabid mammals, and feral dogs and cats (see Chapters 51 and 52). Polar bears, some grizzly and back bears, the great cats, and crocodiles may hunt humans as food. Venomous snakes, insects, arachnids (spiders, scorpions, and ticks), and marine animals are also a concern (see Chapters 44 to 50).

The only effective weapon against large mammals and reptiles is a high-powered firearm, although pepper spray may discourage attacking bears and ungulates. Improvised weapons, such as a spear tipped with a hunting knife, are useless.

Food should not be kept in a shelter or backpack during the night. All food should be placed in a nylon bag and hung between two trees on a high line. Above timberline, small rodents such as mice may gnaw holes in expensive tents to reach food inside, so all food should be bagged and hung on a line between two high boulders.

In warm weather, insect repellent should be carried and used liberally.

Summary of Preparations for a Possible Survival Situation

1. Keep in good physical condition by frequent exercise and healthful habits. Avoid tobacco and "recreational" drugs; keep alcohol to a minimum. Maintain current immunizations.

2. Develop the ability to swim well.
3. Learn how to use a map and compass and find directions without a compass.
4. Be able to build a fire under adverse conditions.
5. Have a working knowledge of local weather patterns and be able to use an altimeter (or barometer), thermometer, wind directions, and knowledge of cloud forms to predict storms. Avoid exposure to dangerous meteorologic conditions, such as blizzards and lightning strikes.
6. Be familiar with the special medical problems of the type of wilderness involved. For example, in cold weather and at high altitude, be familiar with the prevention, diagnosis, and treatment of hypothermia, frostbite, and altitude illnesses. Safe travel in the desert and tropics requires familiarity with tropical infections, snakebites, tropical skin diseases, and heat illnesses. Understand basic principles of prehospital emergency care and the improvisation of splints and bandages.
7. Carry a survival kit containing equipment appropriate for the topography, climate, and season (see appendices to this chapter). At a minimum, the kit should include such things as fire-starting, shelter-building, and signaling/navigating equipment plus emergency food, water, spare clothing, and a first-aid kit.
8. Be able to construct appropriate types of survival shelters.
9. Acquire a working knowledge of the characteristics of natural hazards and how to avoid them. These include forest fire, lightning strike, avalanche, rock-fall, cornice fall, flash flood, whitewater, dead-falls, storms of various kinds, and the hazardous animals and plants of the area of travel.
10. Read and analyze accounts of survival experiences (see "Suggested Readings" at the end of this chapter). Remember that more people are killed on simple day hikes than on long wilderness expeditions.[14]
11. Be aware of the psychological aspects of a survival situation and of errors in judgment that can lead to a survival emergency.
12. Know the edible plants and animals of the area of projected travel, as well as the poisonous or venomous species. Know how to obtain water. Basic hunting, trapping, and fishing skills are valuable.
13. Be intimately familiar with the contents of your survival kit and how to use them. Practice and perfect survival activities such as fire building and shelter building before you need them.
14. Never travel alone. Hunters and other group members who may separate should have means of communicating with each other, such as small radios with a 5-mile (8 km) range. Products are available that combine a GPS locator with a small radio.
15. Always let at least two responsible persons know your destination, the number of persons in the party, their names, the type of vehicles and license plate numbers, and expected time of return. Add several hours to the latter for "normal" mishaps and miscalculations.[14] To avoid unnecessary rescue attempts, do not fail to notify these persons of your return. Failure to follow these guidelines has led to many unsuccessful searches.

APPENDIX A: SUGGESTED BASIC CONTENTS OF TEMPERATE TO COLD WEATHER SURVIVAL KIT

The kit is divided loosely into fire-building, shelter-building, signaling/navigating, and miscellaneous items, all of which will fit into a small backpack (capacity, 3200 to 4000 cubic inches [52.4 to 65.5 L]). The small items can be carried together in a small net bag for convenience and easy identification. Frequently used items, such as a knife, map, dark glasses, and compass, should be in a pocket or on the belt.

Always remember to replace consumed items, especially first aid items, repair kit items, and emergency food, as soon as you return from a trip. Replace stored matches at least every year and first aid kit medications before they expire.

ITEM	APPROXIMATE WEIGHT (ounces [grams])
Fire-Building Equipment	
Two waterproof screw-top match containers filled with "strike-anywhere" matches or Recreational Equipment, Inc. "storm-proof" matches. If match container doesn't include a large, rough match-striking area, a strip of heavy sandpaper should be glued to the top or side of each container.	2 (57)
Candle	1.5 (43)
Fire-starter, e.g., cotton impregnated with Vaseline, in waterproof container such as film can or additional match container	1 (28)
Metal match	0.5 (14)
Knife: Swiss Army (consider "camping" model with saw, file, scissors, etc., made by Victorinox)	5 (142)
Alternate: Leatherman tool with wire-cutting pliers, file, one serrated and two regular blades, saw and scissors	8 (227)
Shelter-Building Equipment	
$^1/_8$-in braided nylon cord or parachute cord, 100 ft (30.48 m)	4 (113)
Rip-stop waterproof nylon tarp, approx. 8 × 10 ft (2.4 × 3 m) with grommets around the edges. Campmor is a good brand. (See online at www.campmor.com.)	19.5 (55.3)
Alternatives:	
"blue, crinkly" tarp (laminated polyethylene weave)	26 (737)
or	
1 to 2 large, heavy-duty (3- to 4-mil) orange plastic bags, 3 × $5^1/_2$ ft (0.9 × 1.7 m)	9–18 (255–510)
Folding saw or small rigid saw, such as 18-in Dandy Saw	12 (340)
Signaling/Navigating Equipment	
Headlamp (or flashlight) with spare bulb and batteries. A good choice is a headlamp with both LED and bulb options. The LED light uses up much less electricity but is more diffuse and doesn't cast a long beam. The bulb is brighter and casts a longer beam but uses much more electricity.	7.5 (212)

Plastic whistle (pea-less) on lanyard	0.5 (142)
Small notebook and pencil	1.5 (43)
Small roll of orange surveyor's tape	2 (57)
Glass signaling mirror with sighting device	2 (57)
Miscellaneous	
Metal pot with bale, containing emergency food of choice (e.g., tea, soup mix, power bars, small can of mixed nuts, trail-mix)	34 (964)
Metal cup with handle (can heat liquid by putting cup at edge of fire)	3 (85)
Plastic or Lexan spoon	0.5 (14)
Toilet paper	1.5 (43)
Sunburn cream with SPF 30 or greater	4 (113)
Lip balm with SPF 30 or greater	0.5 (14)
Insect repellent such as DEET (in season)	4 (113)
First aid kit, one per party (see below for contents)	25 (701)
Canteen (1 to 1.5 L), full; metal or plastic	38.5–51.0 (1091–1446)
Sunglasses, preferably polarized with side shields	2 (57)
Light raingear, e.g., laminated (Gore-Tex, etc.) jacket with hood and pants	31 (879)
Repair kit, adapted to type of travel (e.g., ski, snowshoe, kayak), including:	
Small needle-nosed pliers with wire-cutting feature (if Leatherman tool not carried)	3 (85)
Small crescent wrench	2 (57)
Small screwdriver with multiple tips	5.5 (156)
Picture wire	1 (28)
Fiberglass tape—standard roll	3 (85)
Duct tape, small roll	1 (28)
Steel wool for shimming (ski binding repair, etc.)	1 (28)
Assorted nuts, bolts, and screws	1.5 (43)
Total weight of repair kit	18 oz (510)
Total weight of basic survival equipment (not including shovel, snow saw, backpack, or cold weather fourth layer—see below under Winter Survival List)	approx. 230 to 250 oz = 14 to 16 lb (6.4 to 7.3 kg)

Other Useful Equipment for Consideration

Nondigital watch
Altimeter
Magnifying glass
Two sets of correct change for pay phone
Light pair of leather gloves (hand protection)
Water disinfection equipment: chemicals (Potable Aqua, etc.) or filter
Thermometer (e.g., plastic alcohol type clipped to outside of pack)
Spare eyeglasses
Electronic communication/navigation equipment:
  Cellular telephone (if service available)
  Global Positioning System locator
Personal locator beacon (personal emergency locator transmitter)
Pepper spray (to repel bears, moose, etc.)

Usual Day Trip Items to Be Added

Small thermos of hot or cold drink
Lunch
Binoculars
Camera

APPENDIX B: SUGGESTED ADDITIONS FOR WINTER SURVIVAL KIT (COLD WEATHER OR SNOW PRESENT OR EXPECTED)

Basic survival items from Appendix A
Spare clothing for severe weather, to provide at least four layers total, including spare mittens
Small snow shovel, e.g., small collapsible grain-scoop type with Kevlar blade and detachable handle (24 oz)
Snow saw (consider for above-timberline travel/igloo building) (8 oz)

Optional Items

$^3/_4$-length piece of open or closed cell foam mattress or Therm-a-Rest mattress
Sleeping bag
Bivouac sac
Small stove and fuel
Light ax (Hudson's Bay type)
Small piece of closed-cell foam (2 × 2 feet) for sitting in snow

Mandatory for Avalanche Country

Avalanche probe (folding) or ski poles that join together to form probe for each party member
Avalanche transceiver for each party member
Shovels—preferably one for each party member
Inclinometer for measuring slope angles (included in some compasses)

APPENDIX C: VEHICLE COLD WEATHER SURVIVAL KIT

Sleeping bag or two blankets for each occupant
Extra winter clothing, including gloves, boots, and snow goggles, for each occupant
Emergency food
Metal cup
Waterproof matches
Long-burning candles, at least two
First-aid kit (see Appendix D)
Spare doses of personal medications, if any
Swiss Army knife or Leatherman multitool
Three 3-lb empty coffee cans with lids, for melting snow or sanitary purposes
Toilet paper
Citizen's band radio, cell phone, etc., with chargers
Portable radio receiver, with spare batteries
Flashlight with extra batteries and bulb
Battery booster cables
Extra quart of oil (place some in hubcap and burn for emergency smoke signal)
Tire chains
Jack and spare tire
Road flares
Snow shovel
Windshield scraper and brush

Tow strap or chain
Small sack of sand or cat litter
Two plastic gallon water jugs, full
Tool kit
Gas line deicer
Flagging, such as surveyor's tape (tie to top of radio antenna for signal)
Duct tape
Notebook and pencil
Book to read
Long rope (e.g., clothesline) to act as safety rope if you leave car in blizzard
Carbon monoxide detector
Ax
Saw
Full tank of gas

Data from Winter Survival Handbook. Available at: Montana State Department of Transportation website, http://www.mdt.state.mt.us.

APPENDIX D: MINIMAL EQUIPMENT FOR SURVIVAL FIRST-AID KIT

Recognizing that the most common significant medical problems will be injuries, each item is selected based on likelihood of need, possibility of multiple use, urgency of need, weight to usefulness ratio, "biggest bang for the buck," and whether it can be improvised from natural materials. As with the survival kit, the small items can be carried together in a mesh bag for faster location.

Basic Items

Small Bag Items

CPR mouth shield
Surgical gloves, preferably nonlatex
20-mL syringe with needle, catheter, or splash shield for wound irrigation
Steel sewing needle (can be part of small sewing kit)
Clinical thermometer (consider low-reading thermometer for cold environment)
Small pill boxes containing:
Nonprescription analgesic of choice (acetaminophen, ibuprofen, etc.). Consider ibuprofen if frostbite risk.
Prescription analgesic of choice (acetaminophen with codeine, etc.)
Diphenhydramine, 25 or 50 mg caps
Small tube of biodegradable soap
Splinter forceps
Seam ripper
Small needle holder (useful for retrieving small things out of tight quarters)
Small magnifying glass
Four large safety pins

Other Items

1 or 2 cravats
Roll of 3-inch Ace or self-adhering roller bandage
Roll of 2-inch adhesive tape (waterproof preferred)
Small roll of $^1/_2$-inch 3M Transpore tape
Small, prepackaged bandage strips, 1 inch (can cut in half length-wise for smaller sizes)
Nonadhering sterile gauze pads (Telfa or equivalent), 3 × 4 inch
Sterile compresses, 4- × 4-inch
Alcohol pledgets for skin cleansing
Duct tape, fiberglass strapping tape (for improvising litters and splints; carried in repair kit)

Additional Items for Consideration

Acetazolamide if acute mountain sickness risk (125–250 mg twice daily to speed acclimatization)
Plastic oral airways (three sizes)
Pocket mask (instead of mouth shield). This device is easier to use, works better than a mouth shield, and protects the user from the victim's saliva.
Povidone-iodine swabs for skin preparation
14-gauge IV catheter-over-needle (emergency chest decompression). A flutter valve can be improvised by using the finger cut from a rubber glove with its base tied around the base of the catheter and a hole cut in its tip.
Small bandage scissors or paramedic shears (substitute: scissors on Swiss Army knife or Leatherman tool)
Small, light-weight stethoscope
SAM splint
Persons who are taking regular medications should carry emergency supplies of their medicines in addition to regular supplies; anyone who has had an anaphylactic reaction should carry an emergency epinephrine kit (EpiPen)

SUGGESTED READINGS

Accidents in North American Mountaineering: Golden, CO, American Alpine Club, and Banff, Alpine Club of Canada, 1999 (published yearly by the Lyons Press, Guilford, CT).
Anderson K, Tavernier A (eds): Wilderness Basics, 3rd ed. Seattle, Sierra Club, Mountaineer's Books, 2004.
Auerbach PS: Medicine for the Outdoors, 4th ed. Guilford, CT, The Lyons Press, 2003.
Clifford H: The Falling Season. New York, HarperCollins, 1995.
Craighead FC Jr., Craighead JJ (revised by Smith RE, Jarvis DS): How to Survive on Land and Sea, 4th ed. Annapolis, MD, U.S. Naval Institute, 1984.
Kochanski M: Bushcraft: Outdoor Skills and Wilderness Survival. Vancouver, Lone Pine, 1987.
Krakauer J: Into Thin Air. New York, Villard, 1997.
Landsing A: Endurance. New York, Avon, 1976.
Logan N, Atkins D: The Snowy Torrents: Avalanche Accidents in the United States, 1980–1986. Denver, Colorado Geological Survey, 1996.
MacInnes H: The Price of Adventure: Mountain Rescue Stories from Four Continents. Seattle, The Mountaineers, 1978.
MacInnes H: High Drama: Mountain Rescue Stories from Four Continents. Seattle, The Mountaineers, 1980.
Nesbitt PH, Pond AW, Allen WH: The Survival Book. New York, Funk & Wagnall, 1959.
Olson LD: Outdoor Survival Skills, 6th ed. Chicago, Chicago Review Press, 1997.
Parr P: Mountain High Mountain Rescue. Golden, CO, Fulcrum, 1987.
Patterson CI: Surviving in the Wilds. Toronto, Personal Library Publishers, 1979.
Ralston A: Between a Rock and a Hard Place. New York, Atria Books, 2004.

Randall G: Cold Comfort: Keeping Warm in the Outdoors. New York, Lyons Books, 1987.
Riles MJ: Don't Get Snowed: A Guide to Mountain Travel. Matteson, IL, Greatlakes Living Press, 1977.
Rutstrum C: Paradise Below Zero. New York, Collier Books, 1974.
Simpson J: Touching the Void. New York, HarperCollins, 1988.
U.S. Army Survival Handbook. Department of the Army. Guilford, CT, The Lyon's Press, 2002.
Waterman J: Surviving Denali: A Study of Accidents on Mount McKinley, 1903–1990, 2nd ed. New York, AAC Press, 1991.
Whittlesey LH: Death in Yellowstone: Accidents and Foolhardiness in the First National Park. Boulder, CO, Rinehart, 1995.

The references for this chapter can be found on the accompanying DVD-ROM.

37 Jungle Travel and Survival

John Walden

TROPICAL ENVIRONMENT

In these forests lies a virtually limitless supply of excitement, joy, and wonder to be encountered in new illuminations on the constructs and workings of life on earth.[31]

Tropical rainforests, located between the Tropic of Cancer (23° 27′ N latitude) and the Tropic of Capricorn (23° 27′ S latitude), are regions with at least 4 inches of precipitation per month and a mean annual monthly temperature exceeding 24°C (75°F) without any occurrence of frost. Contrasted to temperate regions, the hours of day and night are about equal throughout the year. Seasons are characterized as "wet" or "dry." These seasonal designations refer to historical trends and should not be taken as gospel: in some years, the dry season extends for months beyond the usual range; in other years, the rains may persist for months as a daily drizzle, making it difficult to trek over the spongy terrain. In the Pacific coastal region of the Colombian Chocó and the Cayapa River Basin of Ecuador, where annual rainfall can exceed 400 inches, the seasons are said to be "wet" and "wetter."

As the most biologically diverse community of living things on Earth, the tropical rainforest is a realm of superlatives: a single acre of tropical forest may contain more than half as many species of trees as occur in all the land mass of temperate North America; one square mile of Amazonian forest may be home to double the variety of butterflies that exist in all of the United States and Canada; a single tree in Peru yields as many species of ants as in all of the British Isles. Facts and figures fail to capture the essence of tropical rainforests and their extraordinary biologic diversity. Seen from the air, the forest stretches from horizon to horizon in a vast green carpet. In season, the crowns of trees in full blossom dot the landscape with vivid splashes of red, orange, and yellow. Sizable streams may be hidden beneath the emerald canopy. Rivers, usually muddy yellow or black, snake through the forest; early-morning or late-afternoon sun transforms these braided rivers into glistening, mirrorlike strands of liquid silver.

Observed from the forest floor, the jungle is entrancing. In virgin, deep forest, all is muted and shadowy, save for random shafts of light that spotlight labyrinths of oddly shaped branches and spectacularly colored flowers. Shrubs and herbaceous plants are scarce in forest away from the flood plain, so it is relatively easy to walk undisturbed. The dimness is occasionally disrupted by areas bathed in bright light from larger holes in the canopy caused by a recently fallen tree, sandy beach, or cutting and burning by humans. It is in these sunlit areas that the traveler encounters the lush and nearly impenetrable wall of foliage portrayed in adventure films. The tidy textbook division of vegetation into distinct tiers is somewhat arbitrary and not easily confirmed, even by experts.[36]

In addition to drier upland terra firme forests, lowland forests remain submerged for several months each year. Such forests, or várzeas, make up only a small percentage of forested land, but are infinitely more fertile than their nonflooding and nutrient-poor counterparts.

Despite environmental differences within the jungle, the basics of travel remain the same.

TRIP PREPARATION

Reading

Back issues of *National Geographic* magazine and the writings of Wilson provide an excellent introduction to people, places, and biodiversity issues.[4,51,52] The *Smithsonian Atlas of the Amazon,*[18] the definitive illustrated atlas (150 color maps and 289 photographs) of the region, is highly recommended, as is the *Conservation Atlas of Tropical Forests* series (Asia and the Pacific,[9] Africa,[38] the Americas[22]). The references at the end of this chapter offer insights into the complex inner workings of the moist tropical forest.[1,35,36] The books by Kritcher[26] and Forsyth and colleagues[17] are especially helpful.

Randall G: Cold Comfort: Keeping Warm in the Outdoors. New York, Lyons Books, 1987.
Riles MJ: Don't Get Snowed: A Guide to Mountain Travel. Matteson, IL, Greatlakes Living Press, 1977.
Rutstrum C: Paradise Below Zero. New York, Collier Books, 1974.
Simpson J: Touching the Void. New York, HarperCollins, 1988.
U.S. Army Survival Handbook. Department of the Army. Guilford, CT, The Lyon's Press, 2002.
Waterman J: Surviving Denali: A Study of Accidents on Mount McKinley, 1903–1990, 2nd ed. New York, AAC Press, 1991.
Whittlesey LH: Death in Yellowstone: Accidents and Foolhardiness in the First National Park. Boulder, CO, Rinehart, 1995.

The references for this chapter can be found on the accompanying DVD-ROM.

37 Jungle Travel and Survival

John Walden

TROPICAL ENVIRONMENT

In these forests lies a virtually limitless supply of excitement, joy, and wonder to be encountered in new illuminations on the constructs and workings of life on earth.[31]

Tropical rainforests, located between the Tropic of Cancer (23° 27′ N latitude) and the Tropic of Capricorn (23° 27′ S latitude), are regions with at least 4 inches of precipitation per month and a mean annual monthly temperature exceeding 24°C (75°F) without any occurrence of frost. Contrasted to temperate regions, the hours of day and night are about equal throughout the year. Seasons are characterized as "wet" or "dry." These seasonal designations refer to historical trends and should not be taken as gospel: in some years, the dry season extends for months beyond the usual range; in other years, the rains may persist for months as a daily drizzle, making it difficult to trek over the spongy terrain. In the Pacific coastal region of the Colombian Chocó and the Cayapa River Basin of Ecuador, where annual rainfall can exceed 400 inches, the seasons are said to be "wet" and "wetter."

As the most biologically diverse community of living things on Earth, the tropical rainforest is a realm of superlatives: a single acre of tropical forest may contain more than half as many species of trees as occur in all the land mass of temperate North America; one square mile of Amazonian forest may be home to double the variety of butterflies that exist in all of the United States and Canada; a single tree in Peru yields as many species of ants as in all of the British Isles. Facts and figures fail to capture the essence of tropical rainforests and their extraordinary biologic diversity. Seen from the air, the forest stretches from horizon to horizon in a vast green carpet. In season, the crowns of trees in full blossom dot the landscape with vivid splashes of red, orange, and yellow. Sizable streams may be hidden beneath the emerald canopy. Rivers, usually muddy yellow or black, snake through the forest; early-morning or late-afternoon sun transforms these braided rivers into glistening, mirrorlike strands of liquid silver.

Observed from the forest floor, the jungle is entrancing. In virgin, deep forest, all is muted and shadowy, save for random shafts of light that spotlight labyrinths of oddly shaped branches and spectacularly colored flowers. Shrubs and herbaceous plants are scarce in forest away from the flood plain, so it is relatively easy to walk undisturbed. The dimness is occasionally disrupted by areas bathed in bright light from larger holes in the canopy caused by a recently fallen tree, sandy beach, or cutting and burning by humans. It is in these sunlit areas that the traveler encounters the lush and nearly impenetrable wall of foliage portrayed in adventure films. The tidy textbook division of vegetation into distinct tiers is somewhat arbitrary and not easily confirmed, even by experts.[36]

In addition to drier upland terra firme forests, lowland forests remain submerged for several months each year. Such forests, or várzeas, make up only a small percentage of forested land, but are infinitely more fertile than their nonflooding and nutrient-poor counterparts.

Despite environmental differences within the jungle, the basics of travel remain the same.

TRIP PREPARATION

Reading

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Trips into the rainforest should be scheduled for the dry season because trails are more serviceable for trekking at that time. Information on weather patterns can be obtained from agencies of national governments, anthropologists, missionaries, and the series entitled *World Survey of Climatology,* which is available in large reference libraries.[43] The *Times Books World Weather Guide* by Pearce[34] is a comprehensive guide to the weather of every country for every month of the year. Although out of print, this guide is easily obtained from any major online marketplace for used, rare, and out-of-print books. The National Climatic Data Center (www.ncdc.noaa.gov) is an excellent source for weather patterns and trends worldwide. Weather Underground (www.wunderground.com) provides current, as well as seasonal, weather averages for many cities in tropical regions.

Attitude

In selecting participants, experienced expedition leaders look for a sense of humor. The ability to see the bright side in difficult times may be an asset more valuable than physical conditioning. Houston[24] and others have discussed the role of humor as a predictor of success. Erb[13,14] noted that successful or failed participation in wilderness ventures also is a significant predictor.

Relationship Considerations

Couples routinely report benefits from the shared experience of wilderness adventure travel, ecotourism, and academic field research. However, in the setting of high-risk expeditions where safety and prompt rescue cannot be assured, a number of group leaders privately note that two individuals who have a sexual relationship may form a team within a team, to the detriment of the expedition as a whole.[49]

Conditioning

Indigenous peoples in jungle regions are almost always slender. After trekking with large numbers of nonindigenous men and women in equatorial regions, I have observed that overweight or powerfully built individuals, particularly men, seem to fare the worst, especially with heat-related illness. Achieving an ideal body weight and becoming aerobically fit by walking or jogging for extended periods of time is beneficial before jungle trekking.

Although being in good shape is sensible, a person need not be an elite athlete to trek through the jungle and enjoy the experience. Good leg strength, acquired by training 20 to 30 minutes on alternate days with stair-climbing exercise machines, offers appropriate preparation.

To keep up with native porters and guides, the prospective expedition member should practice hiking at a fast pace. Once in the jungle, travelers should imitate the energy-saving, fluid rhythm of local inhabitants.

Because trekkers frequently encounter single-log bridges, a well-developed sense of balance is desirable (Fig. 37-1). Walking on the rails of untrafficked train tracks or on roadway curbs may help in preparation. To adapt to specific situations, trekkers should go to the woodlands and practice walking on logs. Head stability is important. Equilibrium can be enhanced by avoiding brisk head movements and by employing the "gaze-anchoring" technique of tightrope walkers. The person fixes the gaze on a spot near the end of the log and does not stare down at the spot just ahead of the feet.[3,8] Special cleats (Covell Ice Walker Quick Clip cleats) (Fig. 37-2) should be considered for

Figure 37-1. A well-developed sense of balance is desirable. (Photo courtesy of John Walden.)

Figure 37-2. Covell Ice Walker Quick Clip cleat. (Photo courtesy of Jon Willis.)

crossing log bridges that are high off the ground, long, and slippery. The cleats can be snapped on quickly before crossing a log bridge and promptly snapped off at the other end.

IMMUNIZATIONS

Travelers to rainforest regions should protect against the following diseases by vaccination or with prophylactic medications (see Chapter 77):

1. Diphtheria, tetanus
2. Hepatitis A, hepatitis B
3. Influenza (see later)
4. Measles, mumps, rubella
5. Polio (where appropriate)
6. Rabies (see Chapter 54)
7. Typhoid
8. Yellow fever (in certain regions of tropical Africa and South America)
9. Malaria (see Chapter 43)

Malaria is prevalent throughout the tropics. Before travel to malarious areas, appropriate prophylaxis is needed. Updated information on the risk for malaria in various regions may be obtained through the Centers for Disease Control and Prevention (www.cdc.gov/travel). The *Medical Letter on Drugs and Therapeutics* (www.medicalletter.com) is an excellent source for current recommendations on preventing and treating malaria.

Persons traveling into remote regions of Amazonia where Indian groups live in isolation should receive yearly influenza vaccinations to reduce the likelihood of inadvertently transmitting disease to these high-risk native inhabitants. Protection against meningococcal disease should be considered where circumstances warrant.

Medical Kit

The Wilderness Medical Society points out that it is inappropriate to pack medications and equipment when no team member has the knowledge or experience to use them safely.[25] The following items for a basic medical kit (Box 37-1) are adequate for personal use in the rainforest setting:

Box 37-1. Medical Kit for Jungle Travel

Bismuth subsalicylate tablets (48)
Diphenhydramine hydrochloride, 25- or 50-mg capsules (15)
Ciprofloxacin hydrochloride 500-mg tablets (20)
Clotrimazole and betamethasone dipropionate cream (60 g)
Epinephrine autoinjector (2 adult, 2 child)
Ibuprofen, 200- or 600-mg tablets (30)
Ketorolac, 60-mg single-dose syringe (2)
Lidocaine hydrochloride carpules (3)
Metronidazole, 250-mg tablets (21)
Mupirocin ointment 2% (30 g)
Permethrin, 5% cream (60 g)
Permethrin, 1% shampoo (2 oz)
SAM splint (1)
Sulfacetamide sodium ophthalmic solution 10% (15 mL)
Sunscreen (4 oz) (2)
Tramadol hydrochloride, 50-mg tablets (20)

1. Bismuth subsalicylate (Pepto-Bismol tablets) is an effective over-the-counter preparation for preventing and treating common traveler's diarrhea. It also is useful for heartburn and indigestion. Pepto-Bismol tends to turn the tongue and stools black.
2. Diphenhydramine hydrochloride (Benadryl, 50-mg capsules) is safe and effective as an antihistamine, for motion sickness, and as a nighttime sleep aid.
3. Ciprofloxacin hydrochloride (Cipro, 500-mg tablets) is highly active against the important bacterial causes of enteritis, including diarrheogenic *Escherichia coli, Vibrio cholerae, Salmonella* species, *Shigella* species, *Campylobacter jejuni, Aeromonas* species, and *Yersinia enterocolitica*.
4. Clotrimazole and betamethasone dipropionate (Lotrisone) cream combines an antifungal with a steroid for rashes.
5. Epinephrine autoinjector (EpiPen/EpiPen Jr.) provides for emergency treatment of severe allergic reactions to insect stings, foods, or drugs.
6. Ibuprofen (600-mg tablets) is a good choice for mild to moderate pain from such problems as menstrual cramps, rheumatoid arthritis, and osteoarthritis. It also lowers elevated body temperature caused by common infectious diseases.
7. Ketorolac (Toradol, 60 mg for injection) provides good short-term relief for moderate to severe pain. It is preferred over narcotics only because it is less likely to cause problems with customs officers and police.
8. Lidocaine hydrochloride may be required as a local anesthetic agent for relief of the excruciating pain resulting from stingray envenomation or conga ant and caterpillar stings. It should be infiltrated into and around the wound area using a dental syringe and 25-gauge needle. Lidocaine carpules (used by dentists) are protected and easy to carry and use in the rainforest.[19]
9. Metronidazole (250-mg tablets) is excellent for treating giardiasis, acute amebic dysentery, and *Trichomonas* species vaginitis.
10. Mupirocin (Bactroban) ointment 2% should be immediately applied to burns, abrasions, lacerations, and ruptured blisters, which can rapidly become infected in the tropics.
11. Permethrin 5% cream and 1% shampoo should be applied to treat scabies and head lice before returning home by travelers who have been in close contact with heavily infested tribal peoples. Many natives, especially in the tropics of Central and South American, are infested with these ectoparasites. Oral ivermectin (200 µg/kg) can also effectively treat scabies and head lice.[16] Marmosets and tamarins are often kept by indigenous peoples of the Americas as pets to aid in grooming for head lice (Fig. 37-3).
12. The SAM splint is lightweight, waterproof, reusable, and not affected by temperature extremes.
13. Sulfacetamide sodium (Sodium Sulamyd/Bleph-10) ophthalmic solution 10% is excellent for treating conjunctivitis, corneal ulcers, or other superficial ocular infections.
14. Sunscreen is essential in open areas such as rivers or jungle clearings. Sunscreens designated "waterproof" retain their full sun protection factor (SPF) rating for

Figure 37-3. Tamarins and marmosets are kept as pets to aid in grooming for head lice. (Photo courtesy of John Walden.)

longer periods during sweating or water immersion than do products designated "water resistant." Opaque formulations are excellent for the nose, lips, and ears. Visitors to the tropics should wear lightweight, long-sleeved shirts and a wide-brimmed hat when exposed to the sun for prolonged periods.

15. Tramadol hydrochloride (Ultram, 50-mg tablets) is used for moderate to severe pain.

Common sense dictates supplementary items. Women on long trips might add miconazole vaginal suppositories or fluconazole (Diflucan, 150 mg as a single oral dose) to treat yeast infections; older men might take a 16-French catheter and sterile lubricating jelly for dealing with urinary retention from prostatic hypertrophy. The fingers may swell rapidly during vigorous activity in the rainforest. To eliminate the possible need for emergency removal, all rings (including toe rings) should be removed before jungle trekking. Body piercings should be removed to eliminate the risk for infection.

Gear

The goal is to travel as "light" as possible. The more gear that is packed, the greater is the likelihood of breakdowns, complications, and misery. The items mentioned in Box 37-2 have withstood the test of time over years of long-distance tropical trekking.

Gear must hold up under difficult jungle travel conditions that include heat, wetness, and mud. No line of advertised gear is ideally suited for the traveler in the tropics. In the United States, L. L. Bean, Inc. *(www. llbean.com)* and Recreational Equipment, Inc. (REI) *(www.rei.com)* are good sources for equipment, especially clothing and footwear, that is usually satisfactory for the tropics.

Footwear

Because feet absorb more punishment than any other part of the body, suitable footwear is the most important item of gear. This is one area in which a person absolutely must not carry inferior equipment. If the feet cannot go, nothing can go.

Military "Vietnam-style" jungle boots with leather uppers, steel insole plates, and speed lacing are unsuitable for serious, long-distance trekking. After an hour of hard walking through streams and muddy trails, blisters can form on every surface of the foot, and the skin will peel off in sheets, bringing a jungle trip to a premature end. Furthermore, safely crossing log bridges and mossy, slime-covered river rocks is almost impossible in these boots.

Two pairs of shoes are needed: one suitable for the wet, slippery conditions imposed by the trail and another that meets the need for dryness and comfort in camp.

Trail Shoes

The following features are desirable in trail shoes:

1. Uppers that hit just above or just below the ankles. Some people choose a high-cut design, reasoning that the extra height gives some added snake protection.
2. Extra protection over the big toe. Rubber or leather toecaps prevent the big toe from being severely battered and bruised.
3. Moderately deep-tread outsoles. Traction on rugged and muddy terrain is important. Running shoes with hard, "high-impact" soles should not be worn because they become slippery on wet logs or river rocks.
4. Quick drying time. Uppers of Cordura nylon and split leather, in addition to resisting abrasion and being aerated, dry rapidly in the sun. Even though hiking shoes usually become soaked within minutes on the trail, it is a psychological boost to start each day with dry shoes. Because jungle travelers can be in waist-high water while on the trail, waterproof shoes with Gore-Tex liners are not essential.
5. Snag-proof design. Shoes or boots with "quick-lace" steel hooks should be avoided; vines and weeds become tangled around the metal hooks, causing the wearer to stumble and pulling the laces untied. Shoelaces should always be double knotted.
6. Lightweight
7. Well broken in

Camp Boots

Footwear needs are different in camp, where the trekker wants dry feet. Shoes, although excellent for the trail, are not suited

Box 37-2. Gear for Jungle Travel

Trail shoes (1 pair)
Camp boots (1 pair)
Covell cleats
Socks, lightweight cotton or thin nylon (3 pairs)
Hat (1)
Pullover garment, polyester (1)
Shirts, cotton
  Long sleeved (2)
  Short sleeved (2)
Pants, lightweight cotton or Supplex or Taslan (2 pairs)
Undergarments
  Underpants, lightweight polyester mesh (3)
  Sports bra, cotton or cotton-Lycra blend mesh (2)
Poncho, nylon (1)
Flannel sheet
Hammock or Therm-a-Rest
Mosquito net
Backpack for porter
Personal backpack
Antifogging solution for eyeglasses
Batteries
Binoculars
Camera equipment and film
Campsuds
Candles, dripless
Cup (Lexan polycarbonate)/plate (melamine)
Duct tape, 1 small roll
Ear plugs
Fishing supplies
Garbage bags
  30-gallon size (4)
  13-gallon size (4)
Headlamp
Inflatable cushion
Insect repellent
Laminated maps
Machete (Collins style)
Waterproof matches or butane piezo ignition lighter
Pen
Toilet paper
Leatherman pocket survival tool
Polycarbonate wide-mouth bottles (2)
Razor/battery-operated shaver
Spoon
Sport sponge
Sunglasses
Umbrella
Whistle, plastic
Zipper-lock bags
  Gallon size (5)
  Quart size (5)
  Pint size (5)

for camp. A noninsulated, pull-on (laceless), open-top boot that comes to midcalf keeps mud off the feet and pants and, when worn with thin nylon socks, allows enough air to circulate to keep the feet cool and dry.

Rubber remains an excellent material for keeping water away from the feet. Rubber lug soles provide traction. When rubber-soled boots are worn at an encampment, however, extreme caution is needed when crossing bridges and walking on wet rocks. Camp boots should be lightweight because they must be carried in a pack on the trail. Discount stores usually carry lightweight, lug-soled rubber boots that meet the criteria for jungle camp boots.

Other Options

The lightweight, comfortable, mesh/neoprene fabric "water" shoes popular for beach and sailboarding activities may have a place on river trips when substantial time will be spent in dugout canoes or rubber rafts.

Thongs and open-toe sandals are fine for most towns and cities in the tropics, but in certain jungle regions, such as the Amazon Basin, exposed feet invite hordes of biting insects.

The jungle traveler must never go barefoot. Plant spines and glass can puncture the feet, and larvae of ubiquitous parasites, such as *Ancylostoma duodenale* and *Necator americanus* (the hookworms) and *Strongyloides* species can enter through the skin. The burrowing jigger flea, *Tunga penetrans,* is a serious pest and can be avoided by wearing shoes.

Socks

Cotton or thin synthetic socks should be worn in the jungle to decrease the risk for blisters from wet trail shoes, to reduce insect bites, particularly from no-see-ums (family Ceratopogonidae), and to lessen the risk for lacerations from sawgrass (Fig. 37-4).

Figure 37-4. Sawgrass. (Photo courtesy of V. Ramey, University of Florida, Center for Aquatic and Invasive Plants.)

Clothing

In many countries, military green or camouflage-style clothing is strictly contraindicated. This is particularly true in military dictatorships or in remote border regions. To be mistaken for a guerilla or foreign infiltrator by the military, police, or security (undercover) forces can lead to harassment, detention, or worse.

Hat
For protection from radiant heat and objects falling in the forest, the traveler should wear a lightweight, light-colored hat that has a medium or wide brim. It need not be waterproof, but should be made of material that can be wadded up. A useful feature is a fastener on each side to snap the brim up for traveling on the trail. A pith helmet, widely regarded as an affectation, is fine for open savanna and river trips, but on the trail, branches make it impractical.

Pullover
Drenching rain may leave a person feeling chilled and uncomfortable, particularly when traveling mainly by canoe or raft. Chilling generally is not a problem when hiking on the trail as long as the person keeps moving. A Dacron polyester fleece pullover, such as L.L. Bean's Polartec pullover, REI's polyester lightweight MTS long-sleeve crew, or Patagonia's polyester pullover, will keep a person warm. Wet garments should be wrung out so that they continue to offer thermal protection. Professional white-water boatmen working in tropical regions generally pack a polyester outerwear garment.

Shirts
Two light-colored, ultra-lightweight, long-sleeved cotton shirts should be taken. At the end of the day, the trail shirt should be washed and rinsed so that it will be ready, although perhaps still damp, the next morning. The second shirt can be used in camp or as a spare for the trail. Expensive synthetic shirts guaranteed to wick away moisture are poor jungle trail shirts and make the person sweaty and sticky.

In camp, if no-see-ums and mosquitoes are few, a lightweight, short-sleeved cotton shirt is practical. Two should be packed. A four-pocket style called the guayabera, favored by men throughout Latin America and the Caribbean, is ideal. La Casa de Las Guayaberas (Naroca Plaza, 5840 SW 8th St., Miami, FL 33144; 305-266-9683; fax 305-267-1687) has an exceptional selection of short- and long-sleeved guayaberas; be sure to specify 100% cotton.

Pants
Two pairs of ultra lightweight, light-colored cotton pants are needed. One pair is worn on the trail during the entire trip. Trail pants should be washed often. The other pair is worn around camp and in towns along the way. Jeans become waterlogged as soon as they become wet and are totally unsuitable for tropical trekking. Although synthetic shirts are unsuitable, nylon Supplex or Taslan pants with a built-in mesh brief can substitute for cotton. Pants made of these materials hold up well, are quick drying, and meet criteria for comfort on the trail. Pants with zip-off legs to create instant shorts should be avoided; they will not hold up to strenuous trail conditions.

Undergarments
Underpants and sport bras should be made of cotton or polyester mesh.

Poncho
An ultrathin waterproof poncho is useful on rafting or canoe trips and in villages, but is worthless for use on the trail because it will become snagged on twigs and tree spines.

Bedding

Flannel Sheet
Tropical rainforests become uncomfortably cold between midnight and sunrise. A cotton sheet does not provide enough warmth, a blanket is too heavy, and a summer-weight sleeping bag retains too much body heat. A flannel sheet sewn together like a mummy bag (40 × 90 inches), but without a taper, provides suitable warmth placed either in a hammock or on a pad.

Many inhabitants of the tropical forests sleep with their feet near a fire that is tended throughout the night. They have learned that the chill of damp, cool jungle nights can be lessened as long as the feet stay warm. Disposable "warm packs" (Heat Treat Toe Warmers) can be attached by the adhesive backing to the outside of your sock, under your toes.

Hammock
Soft cloth hammocks are too bulky and heavy for trips and begin to smell after a few days. Fishnet cotton hammocks tend to fall apart within hours or days. So-called camping tent-hammocks or military tent-hammocks are usually bulky, heavy, impossible to sling properly, extremely uncomfortable, hot, unstable, and never able to keep the rain out in a heavy tropical downpour.

The nylon Marina Double Hammock (Model EZ-199 by E-Z Sales Manufacturing, 1432 West 166 St., Gardena, CA 90247) sold by Wal-Mart and REI has proved nearly ideal for jungle travel. It is compact, lightweight, durable, and reasonably comfortable. It cannot rot or absorb odors. For easier handling, the ski rope tie-end lines that are sold with the Double Hammock should be replaced with 3/8-inch nylon double-braided rope available from home improvement retailers or a boating supply source such as West Marine *(www.westmarine.com)*.

Therm-a-Rest
The Therm-a-Rest foam pad is the choice of expedition organizers in temperate and cold climates throughout the world. It combines the insulating qualities of foam and the cushioning of an air mattress, rolls up to a compact size, and inflates on its own when the valve is opened.

The traveler who will be sleeping on a pad should pack a $1^1/_2$- × $2^1/_2$-yard (1.4- × 2.3-m) plastic ground sheet. The sheet should not be placed directly on the jungle floor, where stinging insects and snakes abound. It should be used only in a hut or on an elevated platform. The ground sheet may also be beneficial for temporary rain protection and for keeping bow spray off a person or gear during water travel. For jungle travel, I prefer a hammock over a foam pad.

Mosquito Netting
A mosquito net designed for use with a hammock is basically a rectangular box that is open at the bottom with sleeves at each end panel for the passage of the ropes by which the hammock is slung. Such nets are difficult to find outside the tropics. Fortunately, a serviceable mosquito net can easily be made from "no-see-um netting" (REI No-See-Um Mosquito Net: available by request). See later in this chapter for details of mosquito net construction.

Figure 37-5. Indigenous peoples are accustomed to hauling loads using a tumpline. (Photo courtesy of John Walden.)

Backpacks
A sturdy, well-designed backpack should be used to carry gear. Reflective material should be sewn onto the back of each backpack. Iron Horse Safety Specialties (800-323-5889, *http://fabric.ironhorsesafety.com*) sells red-orange reflective material for daytime visibility and reflective silver material for nighttime reflectivity. On serious jungle treks, porters are often present. This frees expedition members to carry much lighter loads.

Backpack for Porter
An internal-frame backpack of 3000 to 4000 cubic inch capacity is a good size. It should have external pockets for quick access to liter-sized water bottles.

Indigenous peoples are accustomed to carrying packs and hauling loads with a strap, known as a *tumpline,* slung over the forehead or chest. Many natives, including Amazonian Indians, dislike using the shoulder straps that come as standard equipment on backpacks. Given enough straps, almost any native porter can quickly rig a satisfactory tumpline on a backpack. If you don't have extra straps, tribesmen reared in the tropical forest will strip bark from saplings and fashion an adequate tumpline in minutes (Fig. 37-5).

Personal Pack
A daypack of 1200 to 2000 cubic inch capacity is useful for carrying a camera, snack food, and other gear that must be kept handy. A waterproof liner will keep perspiration from wicking into the bag and wetting everything inside. The pack should have two outside pockets for quick access to liter-sized water bottles.

Pack for River Trips
A durable, waterproof "dry" bag, used by river runners, is worth considering, especially if the trip will involve spending days or weeks at a time in dugout canoes or rubber rafts. Most of these packs, however, cannot stand up to the demands of long-distance overland trekking. The straps tend to be uncomfortable and frequently rip out on the trail.

Other Useful Items

Antifogging Solution for Eyeglasses
Antifog solution, available from dive shops, reduces humidity-induced fogging of glasses.

Batteries
Alkaline batteries should be brought from home. Batteries purchased in Third World nations do not last long and often leak.

Binoculars
The traveler who is an avid bird watcher or enjoys watching butterflies or seeking out orchids high on distant limbs will want to pack a pair of binoculars that are lightweight, compact, shockproof, and waterproof or water resistant.

Camera Equipment
Older-style cameras with mechanical shutters are reliable in regions of high humidity. Film with an ISO of 200 is ideal for use in low-light conditions of the jungle and much preferred over slower film. Water-resistant point-and-shoot and high-performance, professional digital cameras are now available.

Camera Case or Bag
Hard-bodied Pelican cases are waterproof and virtually indestructible. The silver-gray color cuts down on heat absorption and is preferred in hot climates. The cases are ideal for rafting or canoe trips but bulky for trekking. On the trail, waterproof "dry" bags protect equipment.

Camp Soap
A biodegradable soap should be used. The soap Campsuds works in hot, cold, fresh, or salt water and cleans dishes, clothing, hair, and skin.

Candles
Electricity tends to fail at unpredictable times in small towns and even in larger cities in Third World countries. Travelers should carry dripless candles. Spring-loaded candle lanterns should be avoided because they give off an anemic light, gum up, get crushed or broken, and basically waste space in the pack.

Cup and Plate
A large Lexan polycarbonate cup is unbreakable, does not retain taste or odor, and serves the role of cup, bowl, and plate. Travelers who want an actual plate should buy one made of indestructible Melamine.

Duct Tape
High-quality duct tape, such as Duck brand, is excellent for protecting existing blisters and for preventing blisters from developing on areas prone to blister formation when applied before trekking. Although a number of products are marketed to prevent blister formation and protect skin once blisters have formed, in my experience nothing beats duct tape.

Ear Plugs
Travel in the tropics often involves flying in incredibly loud helicopters, cargo planes, or short takeoff and landing (STOL) aircraft. Sponge ear plugs that roll up and fit in the ear canal offer inexpensive, effective protection against hearing damage.

Fishing Supplies
For additional "food insurance" when traveling in extremely isolated regions, the jungle traveler should carry 75 feet of 20-pound-test fishing line, a 12-inch steel leader with swivel, and a few size #4 hooks. Travel rods that break down for compact carrying and spin-cast reels should be considered for sport fishing or adding fresh meat to the daily provisions. Throughout the tropics, most species of fish find Rat-L-Trap lures, particularly the chrome and blue combination, irresistible. The hooks that come standard with Rat-L-Trap lures are not sturdy enough to withstand the hard mouth and powerful bite of tropical freshwater fish. Replace these hooks with 3× or 4× strong, size #4, treble hooks.

Garbage Bags
Four 30-gallon capacity and four 13-gallon capacity large plastic garbage bags can hold clothes, bedding, and other items that must stay absolutely dry and can keep dirty boots isolated from clean items in the backpack.

Headlamp
Battery-operated headlamps offer hands-free convenience at night for reading or going to the latrine. The Petzl Tikka LED Headlamp weighs a mere 2.7 ounces (with batteries) and has a battery life of 24 hours at 21°C (70°F).

Inflatable Cushion or Pillow
A small, self-inflating cushion made with a low-slip polyester fabric top and durable nylon bottom is recommended for sitting in a dugout canoe or aluminum boat.

Insect Repellent
To repel mosquitoes, flies, ticks, chiggers, fleas, and gnats (but not no-see-ums), insect repellent should contain 15% to 30% N,N-diethyl-meta-toluamide (DEET). Formulations (often called "jungle juice") should not contain higher than 30% DEET, because they may pose health hazards (see Chapter 41).

Technique is critical when applying insect repellent. Before dressing, the person should spray the ankles, lower legs, and waist. If clothing has not been pretreated with permethrin (see later), then after the socks are put on, a band of repellent should be sprayed around the top; a band should also be sprayed around both pant legs to midcalf. A light spray to the shirt, front and back, may also help. The hands should be sprayed, rubbed vigorously, and run through the hair. Some repellent should be dabbed on the face, neck, and ears, carefully avoiding the eyes; contact lens wearers should be especially vigilant when applying insect repellent.

No-see-ums, which are tiny gnats that abound throughout the tropics of the Americas, are the most common source of insect annoyance in many regions. They are active at sunset and attack humans emerging from jungle streams. No-see-ums cannot bite through even the thinnest cloth and are usually inhibited by Skin So Soft (SSS, Avon), which appears to have a slight chemical repellent effect, but more likely works by drowning the tiny gnats in oil. SSS is not effective against ticks, fleas, flies, and chiggers and offers little protection against mosquitoes. SSS should be applied liberally and often to the wrists, knuckles, bare ankles, face, ears, and scalp. Men with full beards seem to be especially troubled by tiny gnats and may benefit by applying small amounts of SSS to the beard area.

Permethrin kills or stuns insects that land on clothing that has been impregnated with this product. Permethrin is safe, is highly effective, and persists even after extensive washing of garments (see Chapter 77). It is effective against insects when used on mosquito netting, even when the netting has sizable holes and tears. Permethrin can be purchased as a solution for application from a pump spray.

Laminated Map
Accurate maps exist for most regions on Earth. From the best map available, travelers should laminate photocopied portions that are relevant to a particular itinerary (see "Rescue Strategies").

Machete
A Collins-style machete (Fig. 37-6) is the single essential tool for jungle survival and for the many tasks in camp and on the trail that require steel with a sharp edge. Do not purchase a model with a hand guard. A hand guard serves only for protection against an opponent's blade when used as a weapon in combat; it does not offer added protection when the machete is used as a cutting implement.[23] It is hazardous to use a machete in the rain or when cutting wet grass because the weapon may fly out of the hand. Also, when cutting brush, the person often encounters sawgrass. The resulting skin lacerations, which are not noticed at first because sawgrass is razor sharp, may take a week or two to heal. Because of the risks involved, an experienced individual should be in charge of transporting and using the machete. For added safety, a machete should be carried in a sheath.

Figure 37-6. Collins style machete with sheath. (Photo courtesy of John Walden.)

Matches or Cigarette Lighter
Waterproof, windproof Hurricane Matches light when damp and stay lit for several seconds, even in the strongest wind. Many jungle travelers prefer a butane cigarette lighter with a piezo ignition system.

Organizer Bags
See-through organizer bags help reduce clutter and minimize the risk for misplacing small items.

Pen
J. L. Darling Corp. (2614 Pacific Hwy. E., Tacoma, WA, 98424-1017, phone 253-922-5000, *www.riteintherain.com*) sells outdoor writing products, including "Rite in the Rain" shirt-pocket field notebooks, travel journals, and all-weather pens that write upside down without pumping, underwater, over grease, and in hot and cold temperature extremes. These pens have an estimated shelf life of more than 100 years. The Fisher Space Pen Co. (711 Yucca St., Boulder City, NV 89005, phone 905-713-1163) sells all-weather pens.

Pocket Tool
The Leatherman Super Tool is recommended for jungle travel and survival and features needle-nosed pliers and 12 locking implements.

Poly Bottles
Essential gear includes two quart- or liter-sized wide-mouth water bottles made of high-density polyethylene or Lexan polycarbonate. A 2-ounce, heavy-duty poly bottle comes in handy for carrying a salt and pepper mixture to add flavor to boiled plantains and yucca. Nalgene products are legendary; whatever you put in a Nalgene bottle will stay there and not leak into your pack.

Razor or Battery-Operated Shaver
Both men and women should carry lightweight disposable razors. Most men find that lightweight, AA battery-operated shavers give two shaves a day for up to 2 weeks before requiring a change of batteries.

Spoon
Knife-spoon-fork sets are unnecessary. A knife blade and a good tablespoon made of either Lexan polycarbonate or stainless steel are sufficient for eating.

Sport Sponge
A camp towel, made of microporous material, is lightweight, compact, and superabsorbent; it replaces the cotton towel. With the Cascade Designs Pack Towel or similar brand, the body and even hair can be dried much more quickly than with a traditional towel.

Sunglasses
Sunglasses should be polarized with full ultraviolet light protection. Many travelers prefer sunglasses with red-tinted lenses. Because red is the complement of green, these lenses make the jungle foliage stand out intensely and sharply, with enhanced contrast and depth of field. Retainers hold eyeglasses securely during vigorous activity.

Toilet Paper
American toilet paper is much softer than that purchased in Third World countries. The traveler should never wipe with jungle leaves.

Umbrella
A collapsible umbrella is useful in tropical cities and in remote villages when walking from hut to hut. It also offers excellent protection from the sun on canoe or raft trips. The umbrella should be reflective silver, not heat-absorbing black.

Whistle
A high-quality plastic whistle can be used to signal in case someone strays off the path.

Zipper-Lock Bags
Heavy-duty zipper-lock freezer bags are excellent for organizing medicines, toiletries, and other small objects. Bring five each of the gallon, quart, and pint sizes.

COPING WITH THE JUNGLE ENVIRONMENT

A visit to the rainforests of the New World tropics can be either a sublime experience or a hellish ordeal.[17]

Wetness
The superhumid lowland rainforest receives up to 400 inches of rain a year. In contrast, West Virginia averages about 40 inches per year. In the higher-elevation cloud forest, dense cloud cover throughout the year is accompanied by constant mist or drizzle. In such heat and high humidity, people become mentally fatigued as a result of being constantly wet. Fortunately, travelers can use basic strategies for coping with the physical and psychological burden of wetness.

Dryness while trekking or working during the day is not a requisite for physical or mental health. Wetness does not equate with illness, significant discomfort, or dampened spirits. People can tolerate being wet throughout much of the day if they know that they have a dry change of clothes to wear in camp and that they will be dry at night. In addition to the psychological ben-

efits, being dry at night means that maceration is less likely to develop in intertriginous areas.

Bedding and clothing can be protected from moisture by careful wrapping in plastic garbage bags. Despite all efforts, however, certain "dry" items eventually become damp or accidentally soaked. Wet articles should be spread out on shrubs and bushes. They will dry within 2 hours in full sun. Myiasis caused by the tumbu fly, *Cordylobia anthropophaga,* of sub-Saharan Africa can be avoided by hanging clothing to dry in bright sunlight, never on the ground. Clothing dried over a wood fire absorbs odors that do not wash out.

Health Issues

Health Risks

The subject of the tropics causes many people to think about tropical diseases, such as filariasis, and animals, such as the candirú (see later discussion). Malaria, hepatitis, and motor vehicle accidents are the three leading health problems in most tropical regions. Tropical travelers who venture off the path may be exposed to bodily harm and serious diseases, such as leishmaniasis, onchocerciasis, Chagas' disease (in the Americas), and sleeping sickness (in sub-Saharan Africa). Close contact with many indigenous populations of Amazonia increases the likelihood of infestation with scabies and head lice. Bouts of diarrhea or other annoyances will likely occur, regardless of the extent of precautions taken, but death is unlikely.

Duration of Travel and Emotional Response

Cashel and colleagues[6] examined the mood pattern of participants in eight separate 9-day wilderness expeditions conducted over 4 years and noted a high level of confusion, fatigue, anger, depression, and tension on day 4.

After 2 to 3 weeks of travel in remote areas, the general health of expedition participants deteriorates as a result of insect bites, falls, and noxious plants. Inexperienced trekkers may quickly tire of unfamiliar food and miss usual comforts. Experienced leaders therefore prefer to limit expeditions to not more than 3 weeks.

Preventing Heat-Related Illness

The following guidelines may help prevent heat-related illness:

1. Before undertaking long-distance trekking in the tropics, acclimatize by spending at least 5 days in a hot, humid environment and engaging in moderate daily exercise. This acclimatization will be lost within a week if not maintained.
2. Avoid alcohol and certain drugs. Medications, such as β-blockers, anticholinergics, and diuretics, increase the likelihood of heat-related illness and should be avoided if possible.
3. Wear ultra-lightweight, light-colored, and loose-fitting cotton clothing and a wide-brimmed hat. As previously noted in this chapter, shirts should always be made of cotton. Pants, however, may be made of cotton or synthetic fabrics such as nylon Supplex or Taslan.
4. Whenever possible, have a native porter carry all gear.
5. Maintain adequate hydration. Before setting off on the trail, drink a liter of disinfected water. A half hour later, drink a second liter. One hour after the second liter, drink a third, then consume about 1 L every 2 to 4 hours during strenuous trekking.

Heat cramps, often severe, tend to occur when large amounts of water are ingested without adequate salt replacement. Oral rehydration solution (ORS) products, available in premeasured powder form, added to a liter of disinfected water, provide an ideal balance for replacing lost electrolytes. The first liter of disinfected water (noted previously) consumed before trekking should include a packet of ORS. After especially strenuous days, trekkers should drink an additional $^1/_2$ packet of ORS. The rice-based ORS product CeraLyte has a high absorption rate and reduces ongoing fluid loss in diarrheal illness (see Chapter 62). In the United States, rice-based oral rehydration therapy packets may be obtained from Cera Products *(www.ceraproductsinc.com)* and glucose-based ORS from Jianas Brothers (816-241-2880). To eliminate or reduce the chances of intensely painful leg cramps rousing you from sleep, always perform calf-stretching exercises before falling asleep.

Sport beverages do not contain the ideal concentration of electrolytes found in ORS and are not recommended. Salt tablets are not recommended because they are gastric irritants.

Unexpected Isolation

Various factors contribute to unexpected isolation in the jungle. These include inclement weather, mechanical problems, or political turmoil that shuts down public transportation. Many people respond with anger and irritability, which can be devastating to group dynamics. Travelers should accept the situation and use the additional time to appreciate the tropical forest, take photographs, or read paperback books.

It helps to shift out of gear mentally and allow the intellectual machinery to idle. Nearly everyone has the experience of driving for hours and arriving at a destination with virtually no recollection of the trip. The same can be accomplished in the village setting, lying around on a hammock. The hours and days pass surprisingly quickly, akin to cruising in a sailboat with no engine. The person learns patience and develops an appreciation that the rhythms of nature are not governed by the ticking of a clock.

Camp Life

Shelter

Natives rarely spend the night in makeshift shelters. It is usually best to use existing dwellings for a hammock or sleeping pad. Common courtesy governs placement inside the hut of a native. Travelers should ask about taboo spots before bedding down.

When huts are not available for use, a tarpaulin provides satisfactory shelter from the rain. Rip-stop polyethylene tarps (8 × 10 feet) are lightweight and waterproof. Coated nylon tarps are also acceptable but must be sealed with a product such as Seam Grip.

Figure 37-7 illustrates a typical method for erecting a tarp. First, a thick line is run 7 to 8 feet (2.1 to 2.4 m) off the ground between two trees and cinched tight. The long axis of the tarp is centered over the rope, and a rope attached to the middle grommet on each end is tied to the tree. The corner grommets are tied to available trees, bushes, or strong clumps of grass; a tie-down in the middle on each side is also helpful. The sides of the roof should be made high enough to enter and exit conveniently but not so high that driving rain can come in at an angle.

Once the tarp is up, the hammock ropes are run through the sleeves of the mosquito net. Then the hammock is slung. The hammock should be suspended high enough that it will not sag

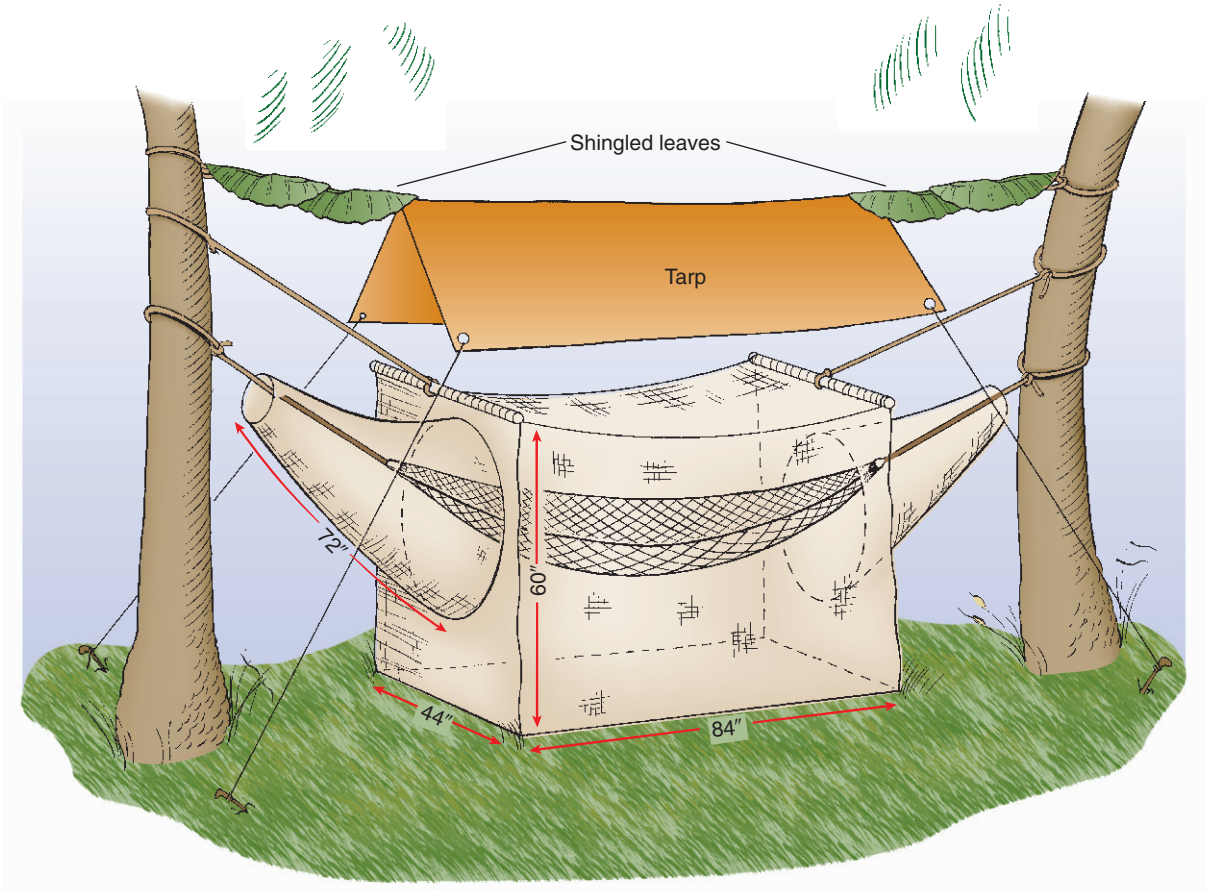


Figure 37-7. Construction of mosquito netting for use with a hammock: sleeve hole is 88 inches (2.2 m) in circumference; small hole is 18 inches (0.46 m) in circumference; smallest holes (for supporting sticks) are 4 inches (10 cm) in circumference.

to the ground during the night because it naturally sags under an adult's weight. Next, the mosquito net is suspended. The ropes running from tree to tarp, from tree to mosquito net, and from tree to hammock should be sprayed with DEET-containing insect repellent (or permethrin insecticide) to keep ants and other pests from using the ropes as trails. Finally, a few broad leaves (banana leaves or heliconia) folded at the spine are draped over the bare rope extending from the tree to the tarp to keep rain from running down the tarp and hammock ropes.

Knowledge of two knots is needed for slinging a hammock. These knots always hold and always come undone quickly without jamming. The half hitch is used to tie the hammock to a horizontal beam, as follows (Fig. 37-8):

1. Pass the working end of the rope around the object to which it is to be secured.
2. Pass the working end of the rope around again without crossing over itself.
3. Bring the end over and around the standing part and through the loop that has just been created. You have completed a half hitch.
4. Make a second half hitch below the first half hitch.
5. Pull tight.

The camel hitch is used to tie the hammock to a vertical post, as follows (Fig. 37-9):

1. Make three turns around the vertical pole.
2. Bring the working end up and over the turns.
3. Make a turn at the top and pass the end back under itself.
4. Make a second turn at the top and pass the end back under itself.

Weather conditions can change in minutes, and travelers must be prepared with adequate shelter. The use of a tent as shelter in the tropical rainforest is not recommended. Clearing a tent space is time consuming, and the stumps remaining from cutting saplings and bushes invariably perforate the floor. Air does not circulate; after a restless night sweltering in the tent, the traveler emerges tired and irritable.

Food

Solitary travelers or small groups usually do not need to pack large amounts of food. Edibles are always available in areas inhabited by friendly natives. As a general rule, food is safe to eat if it is peeled, cooked, or boiled.

Travelers in the tropics must be open to eating local food. Most creatures are edible, such as boiled caiman (alligator), cooked capybara (a 50-kg rodent), or roasted palm grubs

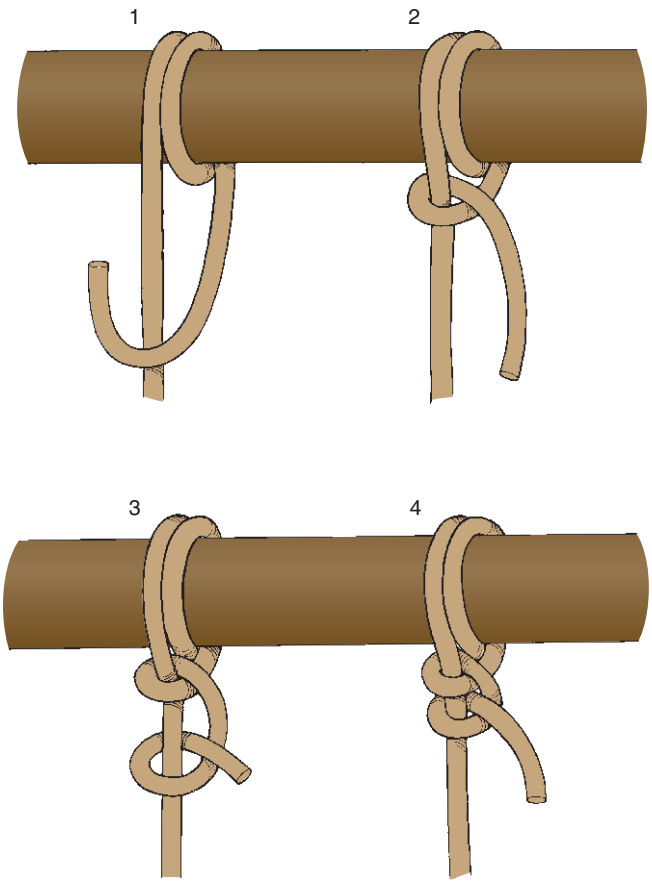


Figure 37-8. Half hitch.

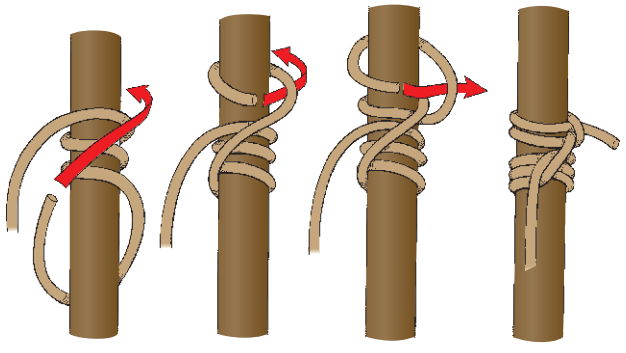


Figure 37-9. Camel hitch.

(larvae of *Rhynchophorus*). Raw palm grubs, up to 5 inches long, are tasty and a favorite of Amazonian Indians (Fig. 37-10). They are eaten by slashing open the thin integument with the thumbnail, extracting and discarding the intestinal tract, placing the opened skin to the mouth, and sucking out the turgid contents.

In addition to palm grubs, more than 20 species of edible insects, including ants and termites, are collected year-round by

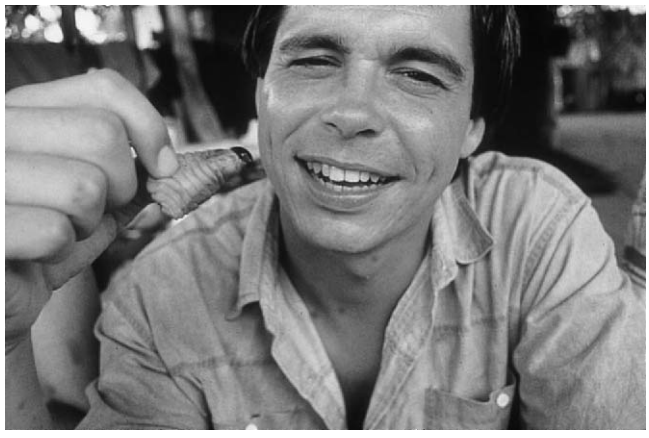


Figure 37-10. The palm grub is a favorite delicacy. (Photo courtesy of John Walden.)

the people of Amazonia.[12] Large hairy spiders, *Theraphosa leblondi,* 10 inches in diameter, are often roasted on an open fire. After the barbed hairs are singed off, the spiders are placed in the embers. They have a shrimplike taste.

Indians of the Americas have perfected the art of smoking fish and meat so that they remain safe to eat for long periods. It is common to see huge hunks of tapir meat or slabs of 100-pound catfish resting on racks, coal black from the smoking process.

Table 37-1 illustrates the diversity of wild game from 867 day hunts by members of the Waorani tribe of Ecuador.[54] The Aguaruna and Huambisa of Peru conceptually recognize and classify as food nearly 500 species of animals.[2]

The tropics have an abundance of flora as food. The yard-long heart of palm is cool and delicious when eaten in its raw state or may be included in a soup spiced with tropical herbs. Familiar tropical fruits include papaya, mango, pineapple, and passion fruit (genus *Passiflora*). Many New World fruits have no name in English and generally have not found their way into the world market. These include chirimoya, guanabana, pitahayas, naranjilla, uchuva, tamarillo, zapote, sapotilla, and badea.[30,33,50] The boiled fruit of the peach palm, *Bactris gasipaes,* is nutritious and flavorful.

The banana and its cousin, the plantain, provide a large percentage of the total caloric intake of natives in the American and African tropics. Curiously, in many native villages, it is difficult to find the sweet, finger-length bananas and the common yellow bananas exported from tropical countries. The green plantain features prominently in the daily fare of inhabitants of the tropics. The plantain has little taste and is exceptionally dry.

Yucca (manioc or cassava), *Manihot esculenta,* is a staple source of carbohydrate nutrition throughout the Americas and much of tropical Africa. The two kinds of yucca, "sweet" and "bitter," are the same species but differ in their distribution and amount of a poisonous constituent, a cyanogenic glycoside, in the root.[42] Sweet and bitter yucca cannot be easily distinguished; one must know which variety was planted. Sweet yucca, common in the eastern lowlands of the Andean countries of Colombia, Ecuador, and Peru, is eaten after the bark containing the toxic substance is peeled off and the root boiled. With bitter yucca, the poison is more concentrated and distributed throughout the root, so it must be extracted before consump-

TABLE 37-1. Rank Order of Species by Total Weight

RANK	COMMON NAME	SPECIES NAME	TOTAL WEIGHT (kg)*	PERCENTAGE OF GROSS WEIGHT
1	White-lipped peccary	*Tayassu pecan*	4,940.1	26
2	Wooly monkey	*Lagothrix lagotricha*	3,873.5	21
3	Collared peccary	*Tayassu tajacu*	2,740.6	15
4	Howler monkey	*Alouatta seniculus*	2,197.4	12
5	Tapir	*Tapirus terrestris*	1,314.4	7
6	Salvin's curassow	*Mitu salvini*	610.9	3
7	Cuvier's toucan	*Ramphastos cuvieri*	414.4	2
8	Capuchin monkey	*Cebus albifrons*	400.8	2
9	Spix's guan	*Penelope jacquacu*	301.7	2
10	Blue-throated piping guan	*Pipile pipile*	261.8	1
11	Brocket deer	*Mazama americana*	253.4	1
12	Capybara	*Hydrochoerus hydrochaeris*	226.5	1
13	Paca	*Agouti paca*	194.8	1
14	Coati	*Nasua nasua*	130.6	1
15	Great tinamou	*Tinamus major*	116.5	
16	Spider monkey	*Ateles belzebuth*	110.1	
17	Saki	*Pithecia monachus*	100.0	
18	Ivory-billed aracari	*Pteroglossus flavirostris*	86.1	
19	Red squirrel	*Sciurus igniventris*	82.9	
20	Gray-winged trumpeter	*Psophia crepitans*	66.5	
21	Agouti	*Dasyprocta fulignosa*	47.5	
22	Golden-collared toucanet	*Selenidera reinwardtii*	30.4	
23	Tayra	*Tayra barbara*	27.3	
24	Squirrel monkey	*Saimiri sciurea*	23.7	
25	Speckled chachalaca	*Ortalis guttata*	21.6	
26	Nocturnal curassow	*Nothocrax urumutum*	20.4	5
27	Cayman	*Caiman sclerops*	20.0	
28	Mealy parrot	*Amazona farinosa*	19.6	
29	Red-mantled tamarin	*Saguinus fascicollis*	18.7	
30	Scarlet macaw	*Ara macao*	18.2	
31	Douracouli	*Aotus tirvirgatus*	17.6	
32	Titi	*Callicebus moloch*	17.2	
33	Squirrel	*Sciurus cocalis*	15.4	
34	Acushi	*Myoprocta pratti*	11.1	
35	Yellow-ridged toucan	*Ramphastos culminatus*	11.0	
36		Miscellaneous small birds (numerous species)	29.3	
Gross weight			18,781.1	100

*Includes only those species whose total weight was more than 10 kg.

tion. Amerindians use an apparatus called the tipití (Fig. 37-11) to express the poisonous juice from the peeled and grated flour of manioc roots.

Travelers in a large group should carry dried, packaged foods because the host village might not be able to provide sufficient foodstuffs or travelers might pass through isolated and uninhabited regions. Packaged foods should also be carried by travelers in regions where natives are unfriendly.

Dried instant food needs only water to make a meal. A few selections should be tried before a large supply for field use is ordered. It is not necessary to add hot water to all packaged foods; adding disinfected, ambient-temperature water produces acceptable results for most foods. Drawbacks to prepackaged foods include expense, space, and disposing of the empty foil packages.

I carry the following supplemental food items for 2- to 3-week treks into remote but inhabited jungle regions: one 2-ounce heavy-duty poly bottle filled with salt and pepper mixed half and half, a few pounds of rice, a tin of long-keeping butter (or oil) for cooking the rice, a few tins of tuna or sardines packed in tropical hot sauce, and several PowerGel energy packets for trail snacks.

Potable Water

Potable Aqua (tetraglycine hydroperiodide 16.7%) tablets are recommended for disinfecting water because they are easy to use and have proved effective in killing bacteria, viruses, and many parasite cysts (see Chapter 61). Potable Aqua is ineffective against *Cryptosporidium*. (Diarrheal illness due to *Cryptosporidium* species usually resolves without therapy in 10 to 14 days in immunologically healthy people.) Water filters are not recommended for purifying jungle water; they clog with silt and must be cleaned frequently. If a water filter is used, it should be fitted with a good prefilter to catch the excess silt.

Jungle Hazards

The following hazards are common in the wilderness jungle setting or thought to be common. Other chapters provide

Figure 37-11. The tipití expresses poisonous juice. (Photo courtesy William H. Crocker, Department of Anthropology, Smithsonian Institution.)

additional insights and viewpoints, particularly with respect to treatment.

Arthropods

Ants. The conga ant, *Paraponera clavata*, 1 to $1^1/_2$ inches long, is the terror of the American tropics. The bite of these large black ants can produce intense pain and fever for up to 24 hours, which accounts for the Spanish name veinte-cuatro (twenty-four). Fortunately, they are conspicuous because their large, shiny black bodies tend to stand out against foliage. Special caution is needed when ducking under or climbing over trees, where ants are often found. A conga bite requires strong pain medication and perhaps the injection of lidocaine at the bite site.

Travelers should avoid touching trees and bushes. Many plants in the tropics provide a home and food for ants, which provide aggressive defense of the plants.

Fire ants are common throughout the tropics and subtropics. Their bite causes discomfort but not excruciating pain. Characteristic sterile pustular lesions in crops often result from fire ant stings.

Chiggers. Chiggers, a form of mite, are a problem throughout equatorial regions. Whereas temperate-climate chiggers may cause mild discomfort for a few days, the tropical chigger sets up an inflammatory and allergic reaction that often persists for weeks.

In the South American tropics, chiggers are found in grassy fields, such as jungle airstrips and yards around mission compounds. Walking through chigger-infested areas without protection could leave a person covered with chigger bites. After a few days, the victim begins to itch mildly. As the days pass, the itching intensifies and seems to come in waves.

Prevention is the best treatment. Areas known to be infested with chiggers should be avoided when possible. Spraying shoes or boots and lower pant legs with repellent containing DEET is highly effective. Pretreatment of clothing with permethrin is recommended. Travelers in the American tropics should never walk through grassy areas in shorts.

Jigger Flea. *Tunga penetrans*, the jigger flea or chigoe, originally found in South and Central America, has now spread to

East and West Africa and India. The fertilized female flea enters the feet through cracks in the soles, between the toes, and around the toenails. The female swells to the size of a pea and may be readily identified as a white papule with a central pit, through which the female extrudes excrement and eggs. When eggs are ripe for release, intense itching causes scratching that helps release large numbers of flea eggs. Incomplete removal of the jigger frequently results in complications caused by secondary infection. A simple extraction technique virtually eliminates complications. Open the skin over the nest of eggs with a surgical blade. Fold back the flaps, remove the easily identified egg sac, and with tweezers, remove the head of the female flea, which can be seen once the egg sac has been removed. Wash the area with hydrogen peroxide.[19]

Myiasis. Myiasis (skin infestation by fly larvae) is common in many regions of sub-Saharan Africa (the tumbu fly, *Cordylobia anthropophaga*) and Central and South America (the human botfly, *Dermatobia hominis*). The victim finds an itchy swelling that slowly enlarges into a lesion with a single breathing pore from which bubbles emerge or from which drains slightly bloody fluid. Later, movement is felt under the skin as the developing larva wriggles around.

Removing the larvae before they emerge on their own is generally advised. Surgical excision, however, should be undertaken with caution because accidental rupture of the larval tegument can lead to secondary infections. Various methods to close off the breathing pore so the larva will emerge on its own include application of bacon fat, meat, chewing gum, or petroleum jelly.

Scorpions and Spiders. Stinging scorpions and venomous spiders are common throughout the tropics and provide another reason to exercise caution before sitting down or placing a hand on logs, bushes, or the ground. The large, aggressive banana spider, *Phoneutria nigriventer,*[49] causes excruciating pain, which may require local anesthetic infiltration of the bite site for relief.

Venomous Moths, Butterflies, and Caterpillars. The larvae and adults of a number of moths (genus *Hylesia*) and butterflies bear venomous hairs that may cause skin eruptions. A rash may result from direct contact with the adults or larvae or by windblown hairs. Direct contact with certain Amazonian caterpillars can cause disabling pain.

In the Amazon tropics, noxious smoke from burning garbage (e.g., plastic wrappers) may cause tree-dwelling caterpillars to loosen their hold on overhead branches and rain down on unwary campers.

Treatment of Lepidoptera envenomation may require injection of lidocaine at the site of intense pain and administration of analgesics, antihistamines, and corticosteroids. Moth hairs may be removed with sticky lint removers used upon clothing.

Wasp and Bee Stings. Sudden, intense pain from the sting of certain species of tropical wasps and bees can be so severe that it knocks the victim to the ground as though hit with an electric shock. Perfumes and brightly colored or flower-patterned clothing should be avoided.[25] Bird watchers should not venture too close to the hanging nests of yellow-rumped caciques and oropendolas because wasps are invariably associated with these nests.

Fish

Stingray. The stingray, a flattened, cartilaginous cousin of the shark, may be encountered buried just beneath the surface of the bottom ooze in tropical rivers and streams throughout the Amazon Basin, Africa, and Indo-China. Rays inflict injury by lashing upward with the caudal appendage, driving one or more retroserrated venomous spines deep into the victim's foot, ankle, or lower leg. This produces agonizing pain, often accompanied by headache, vomiting, and shortness of breath. After the initial phase of envenomation, tissue necrosis may develop. Wearing shoes or boots when wading in water does not always prevent a stingray from jabbing its barb into the foot or leg. Prevention lies in shuffling the feet along the bottom so that the ray will have enough warning to glide away safely.

Electric Eel. The so-called electric eel (actually an eel-shaped fish) is encountered from Guatemala to the La Plata River in South America and is especially common in the Amazon region. A person can drown after being stunned by a jolt from this fish.

Electric eels are said to prefer deep water. Inhabitants of regions heavily infested with eels report a slight tingling sensation when one is close. No practical way exists to prevent these shocks.

Candirú. The candirú is a toothpick-sized parasitic catfish that inhabits Amazonian waters and may invade the urethra of urinating humans. Orifice penetration by the wily candirú can be prevented by wearing a tight bathing suit and not urinating underwater. Native methods of dislodging these fish from the urethra include drinking a tea made from the green fruit of the jagua tree, *Genipa americana L.* Oral vitamin C (2 to 5 g) may serve the same purpose.[5] In his exhaustively referenced book entitled *Candiru: Life and Legend of the Bloodsucking Catfishes,* author Stephen Spotte downplays much of the lore regarding this little fish.[44]

Piranha. Although no human deaths have been documented, piranha have nipped off the fingertips of canoeists dangling their hands in the water. Attacks on humans by the speckled or dark-banded piranha, *Serrasalmus maculatus,* in dammed waters in Brazil have been linked to the fish's behavior of defending its brood from perceived predators.[20]

Mammals

Bats. Vampire bats are found throughout Mexico, Central America, and South America, especially in areas that have large cattle ranches. Sleeping humans are unaware of the presence of a feeding bat; the phlebotomy is painless. Both vampire and fruit bats carry rabies. Sleeping under mosquito netting prevents bat bites. The risk for rabies can be reduced by prophylactic human diploid cell rabies vaccine.

Dogs. Most native groups keep dogs for hunting. Populations with a history of recent tribal warfare often keep packs of dogs close by as an early warning system. These semi-wild dogs should be treated with caution; threatening them may cause immediate attack because they are not easily intimidated. When approaching huts or villages, the traveler should allow porters to deal with the dogs.

Dogs intent on biting often adopt particular behavior patterns. A dog protecting its territory crouches low, straightens its

back and tail, emits a deep guttural growl, and stares fixedly at a specific part of the person's anatomy. Such behavior indicates imminent attack and a sharp blow to the nose may be necessary. Freezing in place may prevent an attack, and direct eye contact should be avoided.

Jaguars. Jaguar attacks are rare. Recommendations are based on advice for avoiding a cougar attack. Increase your apparent size by raising your arms above the head and waving an object, such as a backpack or stick, or opening a jacket. Yell, shout, whistle, or speak loudly and forcefully in a low, deep tone of voice. Back away slowly; do not turn your back and run.[11]

Reptiles

Snakes. Snakebites are rare; 450,000 hours of field work at sites in Costa Rican rainforests were documented without a single snakebite[10] (see Chapters 48 and 49).

Most poisonous snakes tend to blend into their surroundings, and non-natives rarely see them. The most effective protection is putting a jungle-reared guide in front on the trail. Natives almost always spot a poisonous snake and can quickly dispatch it.

Snakes are often encountered along the shorelines of rivers and small streams. Particular caution is needed when hiking in such areas or when disembarking from a canoe or rubber raft. In the forest, the hiker should always step onto a log and then step away from it. The log should not be straddled; snakes often are encountered where the log makes contact with the jungle floor. Because many venomous snakes in the tropics are heat seeking and hunt at night, caution is needed.

Anacondas (water boas) feature in the folklore of all native cultures in the regions of Amazonia where these enormous snakes (up to 9 m [30 feet] long) live. These nonpoisonous snakes kill by looping coils around prey and then tightening the coils, suffocating the victim. Anecdotal reports of anacondas attacking and swallowing humans, particularly children and women bathing at the edge of jungle streams, are unconfirmed.

Alligators and Crocodiles. Although they appear torpid lying in the sun, alligators and especially crocodiles can move amazingly fast. Humans cannot outswim or outrun a charging crocodile.

Bites should be treated with thorough cleaning of the wound, surgical débridement if necessary, tetanus prophylaxis, and an appropriate antibiotic. A study of the oral flora of 10 alligators captured in Louisiana revealed various aerobic and anaerobic organisms responsive to trimethoprim-sulfamethoxazole.[15]

Plants

Armed or Spine-Bearing Plants. Spine-bearing trees abound in forested areas of the tropics. The peach palm *(Bactris gasipaes),* a tall, slender palm whose heart and fruit mesocarp are prized by natives, is found from Nicaragua to Bolivia. The trunk of this tree is ringed with needle-sharp spines (Fig. 37-12). Peach palms often grow alongside trails. Contact with this palm can result in penetration of spines deep into the flesh. Spines that enter a joint space may require surgical extraction. Secondary infection and inflammation often occur.

Figure 37-12. Needle-sharp spines ring the peach palm. (Photo courtesy of John Walden.)

Chicha. Throughout much of Latin America, particularly in the Andean countries, a beerlike beverage known as chicha is consumed. Fermentation is initiated by masticating maize, cassava, plantain, or the fruit of the peach palm. Salivary enzymes hydrolyze the starch to sugar, resulting in a beer of 2% to 4% alcohol content. Some traditional populations consume copious amounts of chicha: the average daily intake among men of the Shuar and Achuar tribes of Ecuador and Peru is 2 to 3 gallons; for women, 1 to 2 gallons.

From a health and safety standpoint, and in the social context, there are several aspects to chicha consumption that give pause. In addition to aesthetic considerations arising from its origin by way of human spit, the starchy substrate used for preparing chicha is often kneaded by unwashed hands—a ready source of pathogens transmitted by the fecal–oral route. Although natives accustomed to daily chicha consumption seem to be unaffected with alcohol-related instability on the trail, the non-native jungle traveler will almost certainly experience problems maintaining balance, particularly when crossing single-log bridges, if excess chicha is consumed before or during trekking.

Because chicha consumption is such a significant element in the social fabric of many tribes, to refuse to partake of the beverage can be a social gaffe. I have found it acceptable to inform my hosts, in a joking manner, I cannot trek and drink chicha throughout the day because "I stagger and fall off logs," but that I will join in the evening rounds of ritual drinking. Eye contact with a woman who is serving chicha is a cultural signal to initiate a sexual encounter among the Jivaroan tribes of Ecuador and Peru. To avoid misunderstanding, hold the chichi bowl in the outstretched hand with the head turned to face slightly to one side or downward.

Sawgrass. In many regions of the tropics, sawgrass is an ever-present nuisance. The scalpel-like blades of this grass can slice into exposed skin. Even when treated with antibiotic ointment, the lacerations often take 1 to 2 weeks to heal. Hikers should avoid sawgrass; special care is needed when working with a machete.

Hallucinogenic Plants

Hallucinogens permeate nearly every aspect of life in primitive societies. They play roles in health and sickness, peace and war, home life and travel, hunting and

agriculture; they affect relations among individuals, villages and tribes. They are believed to influence life before birth and after death.[39]

To ignore the ubiquitous use of psychoactive drugs among jungle dwelling tribesmen throughout Central and South America is to deny a key element in understanding the rich and complex weave of Amerindian life. Powerful drugs, such as ayahuasca *(Banisteriopsis),* brugmansia, the virola snuffs, and yopo *(Anadenanthera peregrina),* are used by shamans and individuals seeking the truth through visions and a supernatural experience.

Ayahuasca. Also known as caapi, natema, pinde, and yaje, this woody vine *(Banisteriopsis caapi)* is found throughout western Amazonia in Colombia, Ecuador, Peru, Bolivia, and Brazil. The term *ayahuasca* derives from the Quechua language and has been translated as "vine of the soul." The hallucinogenic activity derives primarily from harmine, the major β-carboline alkaloid in the plant. Indigenous peoples use additives (leaves of the shrub *Psychotria* and a forest liana *Diplopteris cabrerana*) to prolong and strengthen the intoxication. In addition to experiencing vivid visual hallucinations, users often report a distinct sense of clairvoyance. Adverse effects of ayahuasca ingestion include nausea, dizziness, and vomiting. For some users, the visual hallucinations are intense and disturbing.

Brugmansia. Commonly known as borrachero, floripondio, huacacachu, or toá, the lovely, large bell-shaped flowers of brugmansia are encountered throughout the Andean foothills of western Amazonia. Despite their beauty, these are among the most powerful and dangerous of plant hallucinogens. The leaves and seeds of these bushy plants contain tropane alkaloids, including scopolamine, hyoscyamine, atropine, and secondary alkaloids.[41] The effects on the user tend to be highly unpleasant to the degree that those who partake of brugmansia often must be restrained.

Virolas. Known as epena, nyakwana, and yakee among indigenous populations of northwestern Amazonia, these hallucinogenic snuffs are made from the blood-red resin of the inner bark of trees (*Virola* species). The narcotic effect is due to tryptamine alkaloids. The drug acts rapidly. The excitement phase is followed by lack of coordination, copious nasal discharge, and vomiting. A curious effect of ingesting virola snuff is macropsia, in which objects, including hallucinatory spirits (known as hekulas among the Yanomami Indians of Venezuela and Brazil), appear greatly enlarged.

Yopo. Seeds of *Anadenanthera peregrine,* a South American tree of the bean family Leguminosae, are used to prepare this tryptamine-containing hallucinogenic snuff. Employed by Indians in the Orinoco Basin region of Amazonas State, Venezuela, and adjacent regions of northernmost Brazil, yopo is inhaled to communicate with spirits during the phase of intoxication. In preparation, seeds are removed from the distinctive dangling pods and, after being moistened, rolled into a paste that is roasted over a fire and hardened for later use. The dried paste is pulverized and the resulting powder mixed with lime from snail shells or ashes from certain plants. The narcotic is blasted into the nostril through a long tube with the help of an assistant (Fig. 37-13). The user experiences immediate effects, including muscular twitching, nausea, profuse nasal secretions, and visual hallucinations.[32] As in the case of virola snuffs, an abnormal exaggeration of the size of objects is common.[39]

Detailed discussions of additional New World hallucinogens, as well as African and Asian hallucinogens used by jungle dwelling tribesmen, are found in the references listed on the accompanying CD-ROM.[39–42]

Figure 37-13. Hallucinogen is administered through a long tube. (Photo courtesy of John Walden.)

Should travelers partake of hallucinogens used by tribesmen? Some argue, with a certain validity, that outsiders cannot possibly acquire insight into the Amerindian's sense of the cosmos without ingesting their mind-altering drugs, and so make the case for limited use by anthropologists and others who plan to live and work closely with tribal populations for prolonged periods. Although none of the hallucinogens discussed in this chapter are known to be addictive, "recreational" use could, in the author's opinion, have significant adverse consequences for certain individuals, including a lingering blurring of the sense of reality. So, I recommend that these powerful intoxicants be avoided.[49]

Miscellaneous Hazards

Poison-Dart Frogs

Poison-dart frogs are tiny, brilliantly colored species of the genus *Dendrobates*. They are encountered in Central America and Northern South America (Fig. 37-14). *Phyllobates terribilis* secretes a toxin from its skin so powerful that a lethal dose could be absorbed if enough secretion entered an open wound. It is wise to avoid all contact with brilliantly colored frogs, caterpillars, and snakes.

Falling Trees

Tropical trees do not have deep roots and often fall in relatively modest winds. In many regions of the world, risk for snakebite is significantly lower than the risk for injury or death from falling trees. In the forest, hammocks should be slung away from large trees. Travelers setting up camp should always look up at the branches of any trees near camp; although the base of the tree may appear sound, higher areas may be rotted.

Fording Rivers

The hiker should never attempt to cross a fast-flowing or deep river with a pack on his back. Regaining footing in a rapidly moving current can be difficult. Unless experienced in crossing such streams, the traveler should take the hand of a native guide or porter.

Figure 37-14. Poison-dart frog. (Photo courtesy of John Walden.)

Log Bridges

On frequently used trails, natives generally place a single log across creeks, ravines, and swampy areas. These log bridges may be up to 20 feet high and 75 feet long. Good balance is essential. Because a backpack impairs balance, a porter should carry it across.

Mercury Contamination

Travelers to the Amazon Basin should be aware of the serious, widespread contamination of waterways by mercury that gold miners (garimpeiros) use to process their ore. Although most manufacturers of potable water treatment and filtration systems do not specifically claim to remove mercury, any activated carbon system should reduce the risk. Pres 2 Pure makes a line of filters claimed to remove mercury. Travelers should exercise caution in choosing rivulets as a source of potable water in areas where mercury contamination is known or suspected.

Rising Rivers

Streams, particularly narrow ones bounded by vertical banks, can rise 20 feet in a few hours as a result of intense rains. Camp should not be set up on an island or beach in a small canyon during the rainy season. A cloudburst in the headwaters can send a wall of water rushing downstream, even though it may be a clear, moonlit night at the campsite.

Traveling with Children in the Tropics

The following guidelines should be considered when trekking with children in the tropical forest:

1. Do not attempt a daylong hike. Unlike indigenous children, visitors cannot hike all day in the humid tropical forest. Preadolescents should hike only 1 to 2 hours; children aged 12 to 16 years can hike 2 to 3 hours. Do not subject a child to jungle trail conditions unless the child has had extensive experience hiking in temperate climates.
2. Do not attempt difficult or dangerous trails.
3. Avoid trekking during the rainy season.
4. Keep the child well hydrated.
5. Provide proper footwear (running or hiking sneaker-type shoes or boots with an adequate tread). Avoid leather boots.
6. Keep the child ahead of you and behind a native guide. Children should not be out of sight on the trail.
7. When wading across rivers, have an adult native guide hold the child's hand.
8. Always have children wear a properly sized life vest while rafting, taking canoe trips, or crossing deep, swift, or wide rivers.
9. Do not allow a child to carry any equipment in a daypack other than two liter-sized bottles of drinking water.
10. Ensure that routine vaccinations are up to date. Special vaccinations, such as yellow fever and typhoid, should be considered for certain jungle areas. Hepatitis A vaccine is recommended. Antimalarials are indicated.

Any child who plans to visit the tropics should be a strong swimmer. Many natives begin swimming in infancy and are accustomed to deep or rapidly flowing water that would be extremely hazardous to visiting children. Swimming holes are often located in the swift-flowing outer loop of jungle rivers, where depths may reach 6 feet or more within a yard of the shoreline.

SURVIVAL

Every year, inexperienced people enter the jungle and become lost. After a person ventures only a mere few yards into the forest, especially jungle that has been cleared and is now a tangle of secondary growth, everything begins to look the same. To avoid becoming lost, travelers should always have an experienced guide when traversing unfamiliar territory.

Tribal peoples of the world's tropical forests have an uncanny ability to find their way and arrive at the desired destination, even after days of travel. They can always find food and water and, if necessary, rapidly construct a shelter or weapon.

Occasionally, travelers are left behind on the trail by indigenous guides. Unintentional desertion occurs when trekkers hire natives who have had no experience with neophytes. Realizing that their charges cannot keep up on the trail, the guides run ahead and sit down to rest, not knowing that others cannot navigate the trail alone. Travelers who want to avoid being left behind on the trail should hire a guide who is experienced in traveling with non-natives. Suitable guides and porters can usually be identified with the help of a village leader, local school teacher, village health worker, missionary, or anthropologist.

Rescue Strategies

For individuals in a jungle survival situation, lifesaving items include a large-scale map, Global Positioning System (GPS) unit, some form of electronic voice communication, and machete. Topographic maps are available from numerous international and national mapping agencies. Satellite images with extraordinary resolution are available from Space Imaging (phone, 800-232-9037; *www.spaceimaging.com*) or the U.S. Geological Survey (Eros Data Center 47914, 252nd St., Sioux Falls, SD 57198-0001; phone, 800-252-4547; *http://edcwww.cr.usgs.gov*).

Small, lightweight GPS units display precise latitude, longitude, and altitude. Such information is extremely useful for navigation and for communicating one's location to rescue aircraft. Newer units quickly lock onto satellites and are more likely to work under the jungle canopy.

Trekkers contemplating an expedition into largely uninhabited and unexplored regions should consider buying a compact personal locator beacon (PLB), such as those made by McMurdo Pains Wessex Inc. *(www.mcmpw.com)*. These 406-MHz EPIRB units offer a reliable method of alerting various rescue services through a global satellite system. These units should be activated only in a true emergency when lives are at risk.

Hand-held satellite phones are available for worldwide communication. Although currently expensive to purchase and operate, their potential to provide rescuers with precise GPS location makes these lightweight phones worthy of serious consideration for inclusion for wilderness travel, especially on expeditions into areas of extreme isolation. The Brunton SOLARPORT portable solar panel is lightweight and allows the expeditioner to charge electronics, such as GPS units or digital cameras.

Lightweight, hand-held, very-high-frequency (VHF) aircraft transceivers are excellent for emergency communications. Visitors to remote areas should know the radio frequencies used by rescue aircraft. VHF transceivers are line-of-sight instruments and thus are most useful when aircraft are overhead without objects, such as trees or mountains, between the hand-held unit and the aircraft. In many regions of the world, the Mission Aviation Fellowship (MAF) provides air service to remote airstrips in small villages. If assistance is needed, a hand-held radio transmitter can be used to call an MAF STOL aircraft.

Bush pilots appreciate having information on the condition of seldom-used airstrips. A crude but acceptable device can be constructed to measure airstrip hardness (Fig. 37-15). Cut a pole exactly 2 inches in diameter and about 6 feet long. Starting exactly 6 inches from one end, taper that end to a point. Lash a cross-member on the pole, and have a person weighing about 170 pounds stand with assistance on the cross-member. Make a map of the strip, noting the depth to which the pointed end of the pole sinks into the ground at several dozen sites. Communicate this information to the pilot by radio. If the pole goes in only 2 inches in most areas, the strip is considered ideal; 2 to 4 inches is marginal; penetration beyond 4 inches indicates that the airstrip is unsuitable for landing and takeoff.

If rescue is not feasible, the traveler should continually move downstream at a fast pace. Inhabited areas usually have a trail running alongside a stream. The trail may veer away from the stream where natives have cut a path to connect two villages. Marking the trail every 10 yards with a machete makes it easier to return to the starting point. To avoid confusion, the traveler should mark trees only on one side of the trail. Recall will be increased by audibly describing the surrounding topography as you hike down the trail.

Where human paths are in frequent use, identifying a trail is fairly easy. Seldom-used trails or any trail traversed during times of optimal plant growth may be extremely difficult for the nonnative to identify and follow. Even under adverse circumstances, however, there are clues to trail identification. Paradoxically, concentrating only on the actual foot path will almost certainly cause you to lose sight of the trail. Think of the jungle trail not as a track on the ground but as the intestinal lumen of "some gigantic leafy creature,"[21] with vertical margins, often an overhead horizontal boundary, and sometimes a visible path beneath the feet. Diagonally sliced saplings or neatly severed branches indicate someone has used a machete. There is a particular reflectivity off the ground where humans have trod; this reflectivity is the best way to follow a trail at night. These trail-finding clues are often so subtle that you may sense the trail rather than see it. Game trails meander and are narrower than human trails.

In the jungle setting, navigation with a compass for a distance of more than 200 yards is fraught with hazard. Travelers should not attempt to cut overland if lost, inexperienced, or on their own, unless a significant landmark is visible or sounds of humans or domesticated animals, indicating a settlement, are clearly heard.

A raft may be constructed by lashing logs together with rope or tough, pliable jungle vines. Balsa trees *(Ochroma pyramidale)*, encountered throughout much of Amazonia, make the best rafts. Balsa is often found growing alongside rivers and has the following characteristics: tall, columnar trunk with branches and leaves bunched at the top, which gives the tree a "skinny" look; beige or gray-beige trunk; bark that is smooth but tends to flake, giving it a mottled appearance; and broadly heart-shaped, more or less three-lobed leaves. The key feature of balsa wood is its remarkably light weight. Bamboo also can be used to construct a first-class raft.

Figure 37-15. **A,** Young man sharpening a stick to a point. **B,** Lashing a cross-member to a pole. **C,** Standing on the pole to take measurements of the depth of penetration into the airstrip. (Photo courtesy of John Walden.)

A log flotation device may be constructed by tying together two balsa logs or other lightweight wood placed 2 feet apart[45] (Fig. 37-16).

A "brush" raft may be made by placing buoyant vegetation within clothing or a poncho. Dry leaf litter ("duff") or plants such as water hyacinth may be used.[45]

Food

Food is readily available in inhabited regions. Even abandoned villages yield enough fruit and vegetables on which to survive. Throughout the tropical world, bananas and the large plantain "cooking banana" are ubiquitous. Root crops, such as taro, yams, and yucca, should be sought. Yucca roots should be shredded or pounded and then boiled to release their toxic compounds. As an extra precaution, the wet pulp should be flattened into a "pancake" and cooked on a grate to eliminate any remaining volatile hydrogen cyanide gas.

All land crabs, mammals, birds, freshwater fish, turtles, snakes, and lizards are edible, but should be cooked first to eliminate parasites. It is virtually impossible to kill game without firearms. In inexperienced hands, traps and snares are not effective. Much better results are obtained from fishing.

Edibility Test

In a jungle survival situation, the goal is to get out as quickly as possible. In the rare circumstance in which the victim cannot recognize any familiar edible plant; has been unable to capture minnows, crayfish, and insects; and, for whatever reason, is unable to walk out of the jungle or expect prompt rescue, the following abbreviated version of the Universal Edibility Test recommended by the U.S. Army[46] and the SAS Survival Guide[53] may be used to test an unknown plant:

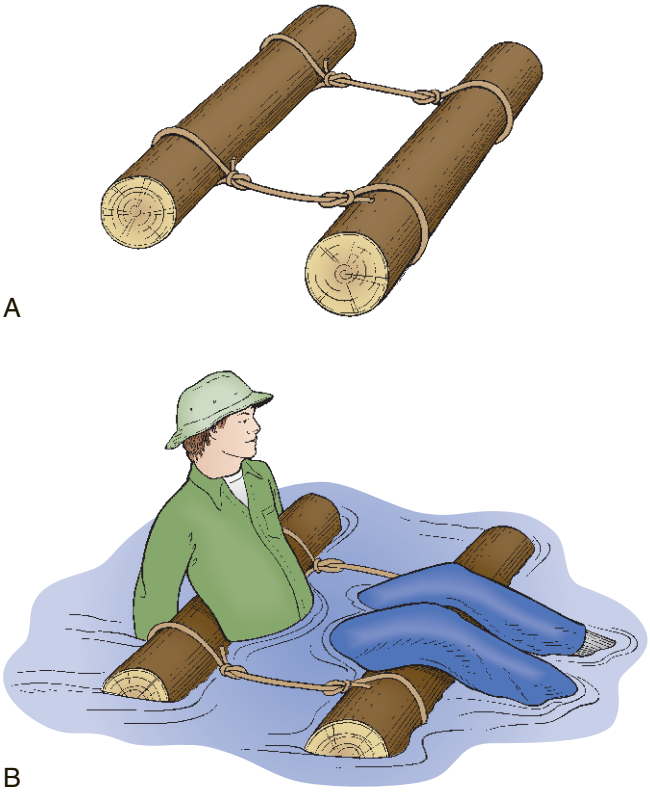


Figure 37-16. Log flotation device. **A,** Two lightweight logs are tied together. **B,** Device in action.

Caveats:
1. Never eat mushrooms.
2. Avoid unknown plants that have:
   - Milky or discolored sap
   - Beans or seeds inside pods hanging from trees
   - Bitter or soapy taste
   - Peach or "bitter almond" scent
   - Tiny barbs on the stems and leaves that could irritate the mouth and digestive tract

Choose a plant that is abundant in the area. First, inspect the new plant, then crush a small portion; if it smells of bitter almonds or peaches, discard it. Check for skin irritation by squeezing some juice into the inner portion of the upper arm; if discomfort, a rash, or swelling occurs, discard the plant. Next, proceed through the following steps, waiting 15 seconds between each to see if there is any reaction and discard if discomfort is felt:

- Place a small portion on the lips
- Place a small portion in the corner of the mouth
- Place a small portion on the tip of the tongue
- Place a small portion under the tongue
- Chew a small portion

Swallow a small amount and wait 5 hours. Drink or eat nothing else during this period of waiting. If, after 5 hours have passed, there is no soreness of the mouth, excess belching, nausea, vomiting, or abdominal pains, the plant may be considered safe.

Water

Water may be made safer by boiling or using chemical disinfectants, such as Potable Aqua tablets. Drinkable water may be found in lianas, often called "water vines," throughout jungle regions. Vines that contain water are fairly easy to identify because they tend to resemble the "grapevines" of North American forests and have rough, scaly bark. These vines may be several inches thick and contain surprising amounts of clear water. Vines that do not contain drinkable water tend to have smoother bark and, when cut, exude sticky, milky liquid. Travelers should not drink from vines that contain milky, latex-like sap; this substance is poisonous. Maximal amounts of water are collected from water-bearing vines if the first cut is high on the vine and the second cut is lower on the vine near the ground (Fig. 37-17). When the water stops flowing from the cut section, cutting about 6 inches from the opposite end will start the flow again.

Water may be trapped within sections of certain types of green bamboo. Bamboo that contains water makes a sloshing sound when shaken. Water also may be obtained from green bamboo stalks by bending a stalk over, tying it down, and cutting off the top. Water dripping from the severed tip can be collected in a container during the night[45] (Fig. 37-18).

Large amounts of water can be found in the voluminous natural cisterns formed by the cuplike interiors of epiphytes (air plants), such as bromeliads (Fig. 37-19). The water should be strained through a cloth.[45]

Water may be collected from a banana or plantain plant by cutting the plant about 1 foot above the ground and scooping out the center of the stump into a bowl shape. The hollow thus formed fills immediately with water. The first two fillings have a bitter taste and should be dipped out. The third and subsequent fillings are drinkable. A banana plant can furnish water in this fashion for several days[45] (Fig. 37-20).

In coastal regions, unripe (green) coconuts provide adequate supplies of refreshing milk. The milk of mature coconuts has a laxative effect and should be avoided.

Shelter

Abandoned, temporary shelters previously constructed by natives on hunting expeditions seem to attract particularly aggressive, large biting spiders and stinging ants. Also, venomous snakes may be attracted to rodents residing in these abandoned shelters. It is often preferable to take the extra time to set up a new camp than to risk encountering venomous insects, arachnids, and snakes.

In an emergency, a proper shelter can be constructed using only plant materials. Figure 37-21 illustrates the basics of constructing a sleeping platform and lean-to. A shingled covering can be made quickly and easily from long, broad banana or heliconia leaves. Tropical palms provide a more substantial roof, but require more time and skill in construction. After selecting a suitable ground-hugging species or chopping down a slender tall palm (palm trees with spines often provide the best fronds), each frond is separated into halves by grasping it at the distal end, separating the leaves as though parting hair down the middle, and splitting the frond in two with a quick jerk (Fig. 37-22). The halves should be overlapped like shingles and secured to the roof framework.

It is much easier to construct an adequate shelter using a tarpaulin (see "Camp Life").

Fire

In addition to boiling water for drinking and cooking food, fire lifts the spirits, warms the body on uncomfortably cool jungle nights, and can be used to signal rescue aircraft.

Tinder and Kindling

Small strips (1/8 × 2 × 4 inches) of rubber tire carried in a survival kit and a butane lighter are an excellent combination for starting a fire even in wet conditions. Tampons make a good fire-lighting aid. The silk-cotton or ceiba tree *(Ceiba pentandra)*, found throughout the American, West African, and Southeast Asian tropics, produces balls of cottonlike fibers, known as *kapok*, which immediately catch fire and make an ideal tinder. The clothlike fibrous material at the base of palm fronds makes excellent tinder.

Bamboo Fire Saw

If you don't have matches or a butane lighter handy but do have access to bamboo, you can make a bamboo fire saw[45,49] (Fig. 37-23).

1. With your machete, cut a 3- to 4-foot long section of bamboo.
2. Split the section the long way with the machete. One of the resulting long sections will be the "baseboard."
3. Shorten one of the split sections to about 1 foot in length. This section will be the "running board."
4. If kapok, palm fiber, or other suitable kindling is not available, with the machete blade, prepare tinder by scraping the outer sheath of a piece of bamboo. You need one large handful of scrapings.
5. Cut a narrow notch at 90 degrees on the outer (convex) side of the running board so that it just breaks through

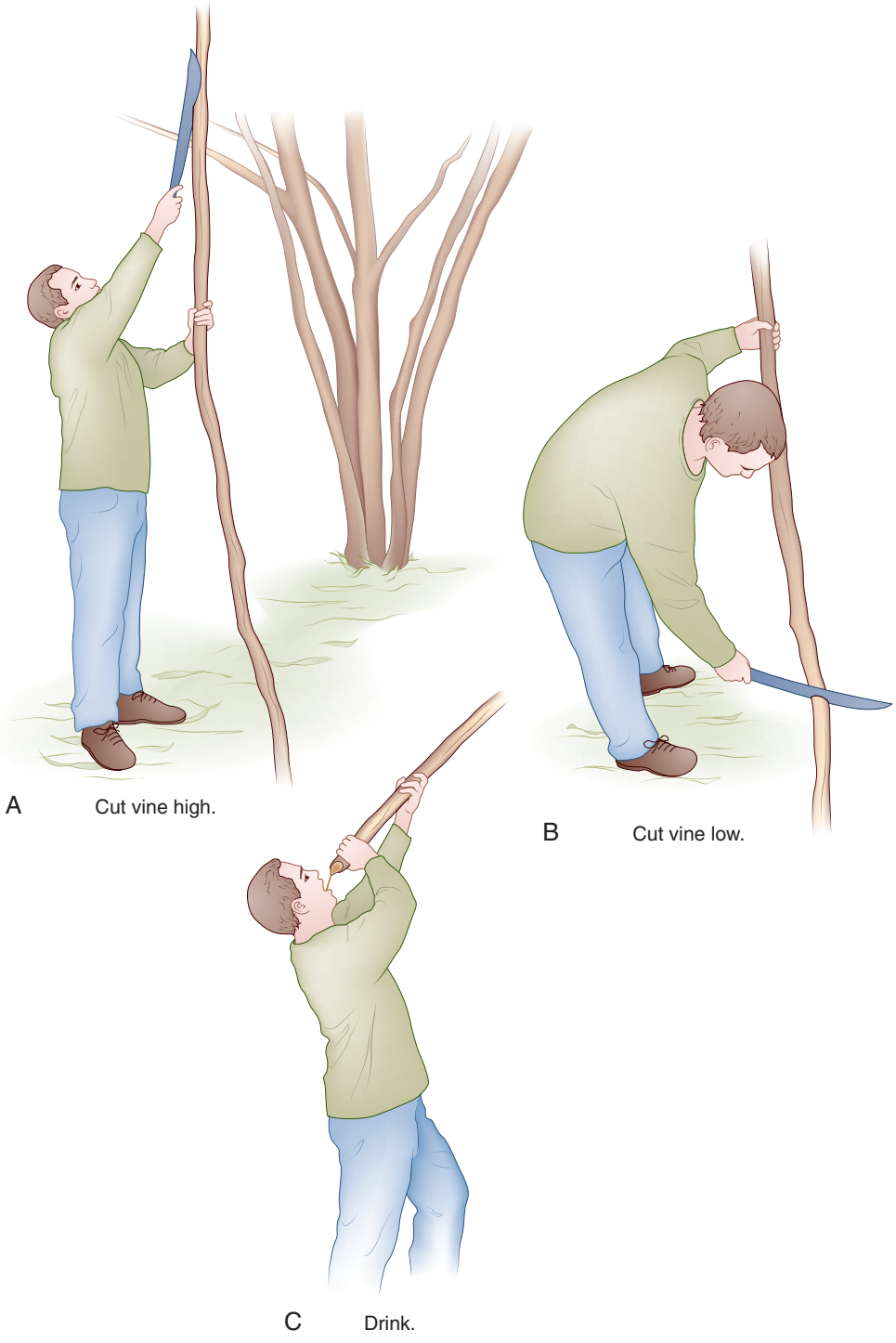


Figure 37-17. Water vine: first cut is high, second cut is low.

to the inner wall. This will serve as a guide to slide the running board rapidly over the baseboard.

6. Fill the running board with a fluff ball of tinder.
7. Place a thin strip of wood or a strip of bamboo over the ball of tinder to hold it in place.
8. Anchor the long section of bamboo (baseboard) with one end in the ground or against a rock or solid log and the other end wedged firmly against your abdomen. The sharp edges of the baseboard should be facing upward.

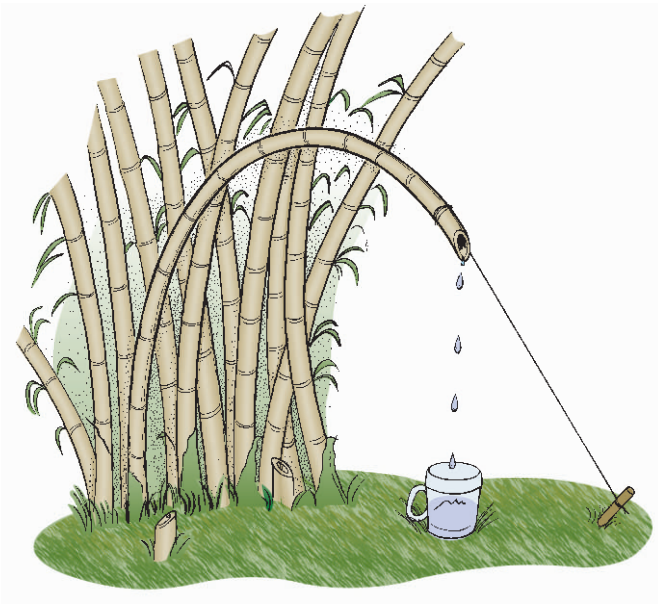
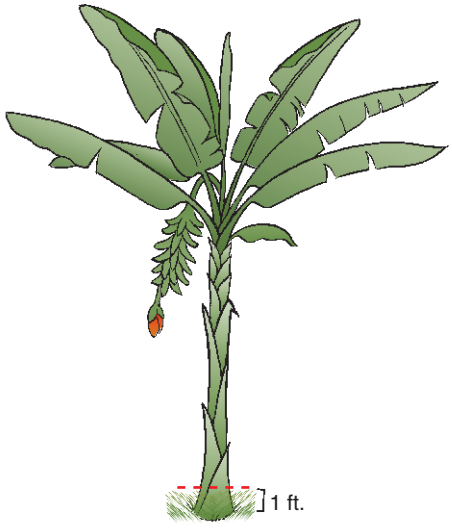


Figure 37-18. Bamboo can be a source of water.

Figure 37-19. Water can be found in the natural cisterns in bromeliads. (Main photo courtesy John Walden; inset photo courtesy Erich Lehenbauer.)



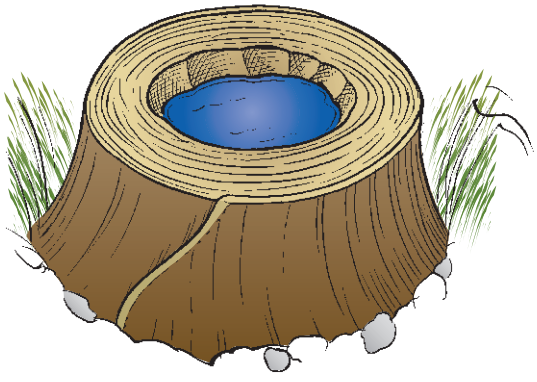


Figure 37-20. Water collected from a banana plant.

Figure 37-21. Sleeping platform.

A B

C

Figure 37-22. Indian splitting a frond to make a covering for a lean-to (see text). (Photos courtesy of John Walden.)

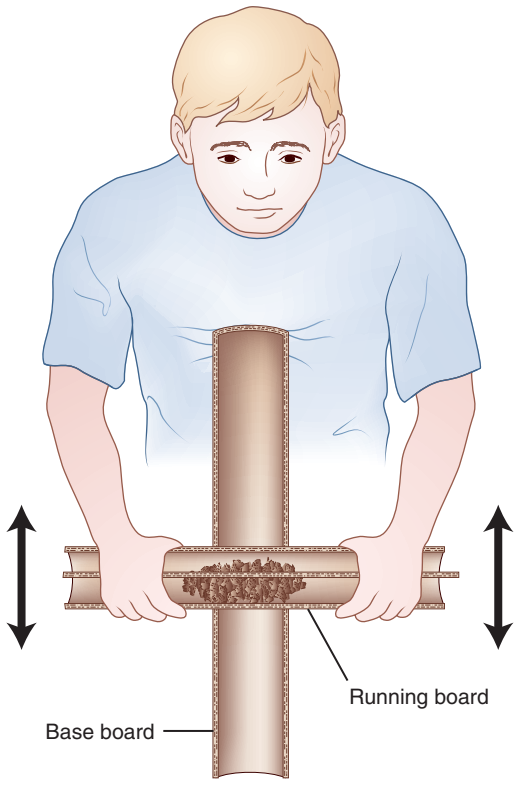


Figure 37-23. Bamboo fire saw. (Redrawn from Walden JB: Jungle Travel and Survival. Guilford, CT, Lyons Press, 2001, p. 175.)

9. Holding carefully on to each end of the strip of wood that is keeping the tinder trapped inside the running board, rapidly and vigorously slide the running board up and down the baseboard with the groove against the sharp edge of the baseboard.
10. You will know that you are exerting effort sufficient to generate enough friction to ignite the tinder when you feel nearly exhausted, have worked up a sweat, and begin to entertain thoughts such as, "This just isn't worth it."
11. As soon as wisps of smoke begin to billow up from the tinder, gently blow upon the tinder until it bursts into flame.
12. Add small pieces of kindling and avoid smothering the fire.

Using this or any other friction method for making fire is hard work, but it *can* be done.

Bamboo Container

Large diameter bamboo makes a great "pot" for heating water to a sufficient degree to cook items such as crawfish and minnows. To prepare a bamboo cooking pot, take a section of bamboo cut just past each end joint. Notch out an opening by making cuts at 45 degrees near each end, then run the machete blade between the notches and pop out the "plug." Support each end of the bamboo pot on stakes or sturdy Y-branched sticks that have been driven into the ground (Fig. 37-24).

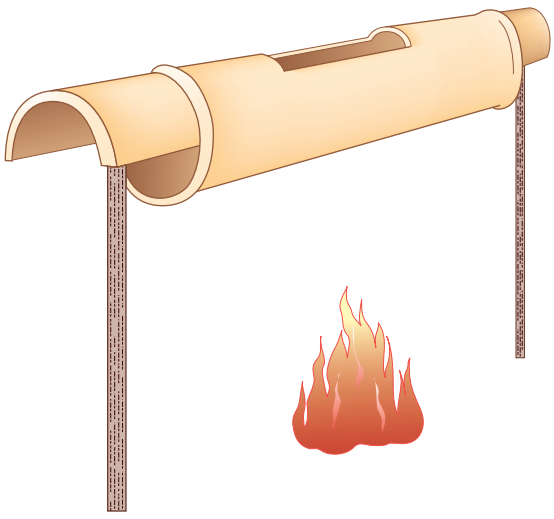


Figure 37-24. Bamboo container for heating water. (Redrawn from Walden JB: Jungle Travel and Survival. Guilford, CT, Lyons Press, 2001, p. 178.)

Figure 37-25. Palm spathe container. (Photo courtesy of Jon Willis.)

Palm Spathe Container

If bamboo is not available to construct a container, a woody spathe (the durable, canoe-shaped structure that encloses flowers and fruits of palms) can be substituted. Support the spathe at each end over a low fire and fill the vessel with water. A palm spathe container will stay intact long enough to cook crawfish and minnows (Fig. 37-25).

Psychology of Survival

Travelers reared on movies and novels depicting the horrors of the Amazon may have irrational fears of being lost or stranded in the jungle. Visible daytime threats worsen with the onset of darkness, when perception becomes distorted. Travelers incapacitated by fear may throw away survival items or may flee from rescuers.

Strategies that can increase travelers' confidence in survival include the following:

1. *Previous jungle experience.* It is helpful to begin tropical excursions in the structured setting of small-group travel. Ecotours, particularly in Costa Rica and Ecuador, offer a combination of rainforest trekking and cross-cultural experience.
2. *Survival manuals.* Military experts and others provide insights from decades of experience.[45,46]
3. *Information on the tropical rainforest.* Familiarity with exotic plants and animals lessens the likelihood of fear while increasing awareness of potential utility in a survival situation. The anthropologic literature is replete with first-person accounts by anthropologists who have lived under trying circumstances with minimally contacted tribal populations throughout the tropics.
4. *Classic accounts of survival against all odds,* such as Alfred Lansing's *Endurance: Shackleton's Incredible Voyage.*[27]
5. *Courses in wilderness-oriented skills.* The National Outdoor Leadership School (NOLS, 307-332-5300, *www.nols.edu*) teaches wilderness-oriented skills and leadership in a core curriculum stressing safety and judgment, leadership and team work, outdoor skills, and environmental studies. Wilderness and outdoor safety courses *(www.outdoorsafe.com)* teach both the psychological and physiologic aspects of surviving a wilderness emergency.
6. *Traveling with a Collins style machete, the one indispensable tool.* A map, compass, GPS unit, and windproof lighter are other recommended items.
7. *Taking stock.* The traveler facing a wilderness crisis assesses a situation analytically and rationally before planning the course of action. Having survival skills is important; having the will to survive is essential.[46]

Cultural Factors

Travelers who have the privilege of spending time with jungle-dwelling populations will find much to admire in the lives of indigenous peoples throughout the world. In some cultures, however, various facets of tribal life lend a discordant note and remind us that all cultures have their unpleasant aspects.

Infanticide

In a few tribal societies, infanticide is still practiced. Often, the first or second of twins (depending on the customs of a particular tribe) and deformed newborns are killed. In some tribes, when the firstborn is a female instead of the more prized male, the decision may be made to kill the child at birth.

Intertribal Warfare, Revenge Killings, Homicide

Although the rates may be significantly lower or slightly higher on a regional basis, the literature shows that those who observed certain indigenous populations during their first sustained contact with the outside world found a high background level of intertribal warfare, revenge killings, and homicide. Chagnon reported warfare-associated male mortality between 20% and 30% among the Yanomamö of Venezuela.[7] Summarizing data for five generations among the Waorani, a linguistic isolate inhabiting the lowlands of eastern Ecuador, Larrick and associates[28] concluded 42% of deaths were attributable to internal spearing raids and 16% were attributable to conflicts with the outside world.

Various hypotheses have been advanced by anthropologists and others to explain the high death rates due to violence among Amerindians. If you disregard the theories of squabbling academicians and ask the Indians themselves, you will find preoccupation with warfare and homicide is attributed to revenge killings (vendettas), sexual disputes, and shamanism.

Interestingly, after contact with the outside world, most groups rapidly abandon generations of warfare and violence. Influences, including Christian missions, school, and sports, in particular intervillage soccer tournaments, have been credited with transformation to more pacific lifestyles.[37]

SURVIVAL IN HOSTAGE SITUATIONS

The following overview of survival in hostage situations is based on material developed and used by the 2nd Battalion, U.S. Army John F. Kennedy Special Warfare Center and School, and draws heavily from an outline prepared by James Liffrig, MD, U.S. Army.[29]

Some areas of otherwise pleasant tropical rainforest are in regions of risk for hostage taking by terrorists, guerillas, or criminals whose motivation may be purely mercenary. Individuals traveling to such areas should have insight into the fundamentals of survival in hostage situations.

Prevention

Since the late 1960s, hostage taking has been on the increase worldwide as a way of setting up a bargaining position to achieve an otherwise unattainable objective. At times, the individual taken is an innocent victim of circumstances who happened to be in the wrong place at the wrong time. Often, however, the individual has been chosen because he or she is an easy target.

The Consular Affairs home page *(http://travel.state.gov)* of the U.S. State Department provides current travel warnings worldwide and detailed country-by-country profiles on safety and security matters in Consular Information Sheets.[47] The likelihood of becoming a victim of hostage taking or kidnapping can be diminished by avoiding travel to areas of known high risk, such as Colombia and the frontier jungle regions of the five countries that share a border with Colombia. If one must travel to an area with a history of recent terrorist attacks or kidnapping, follow common sense precautions:

1. Have your affairs at home in order (current will, insurance documents, power of attorney, guardianship arrangements for minor children).
2. Register with the nearest U.S. embassy or consulate through the State Department's travel registration website: *(https://travelregistration.state.gov/ibrs/)*.[48]
3. Avoid obvious terrorist targets, such as places where Americans are known to congregate.

4. Be sensitive to what you discuss with strangers, and that you may be overheard.
5. Vary routines, including appointments, travel times, and routes.
6. In a city, walk purposefully as if you know where you are going, even if you are lost. Indecision, hesitancy, and directionless movement attract thieves and kidnappers in the same way that thrashing surface movements attract sharks.
7. As much as possible, blend in.

Categories of Hostage Takers

Terrorist organizations are often stratified, with educated and dedicated idealists at the top and disillusioned, young, impressionable recruits from colleges or universities lower down in hierarchy. In some organizations, mentally ill persons and sociopaths recruited from prisons make up a nucleus of the more violent members of the group. Political and religious extremist hostage takers may represent the most danger to Americans because of the value of Americans as symbolic targets.

Paramilitary/guerilla combatants often operate out of remote jungle regions and may cross in and out of one country to another along an unguarded border. Those who trek in such areas may become "targets of opportunity" by chance encounter with these individuals. Guerilla organizations in certain parts of the world engage in drug trafficking in order to obtain money, weapons, and equipment. Such organizations have increasingly turned to hostage taking for ransom as a source of fund raising.

Behavior at the Moment of Capture

The initial moment of capture is dangerous because the captors are tense and may commit unintentional violence with the slightest provocation.

1. Do not reach into a coat or purse to produce a document unless so instructed.
2. Reassure your captors that you are not trying to escape.
3. If the terrorists use blindfolds, gags, or drugs at the time of abduction, keep in mind the fact that the terrorists want you alive.
4. If blindfolded, do not remove the blindfold because this could leave the terrorists no alternative but to kill you.

Hostage Rules of Behavior

> *Always smile: I had learned that lesson years ago in the Congo, where the penalty for dropping your grin, even for an instant, in the face of nervous soldiers or tribesmen, was slow death with both legs hacked off above the knee with pangas.*[55]

Hostage taking forces the terrorist or criminal into stereotyped responses. The more knowledge and understanding a hostage can have about his captors, the better he or she will be able to predict the hostage taker's behavior and feel some degree of control. The conduct of the hostage can increase or decrease his chances for survival.

1. Smile. Be polite and respectful.
2. Be aware of the common "Good Guy/Bad Guy" interrogation technique.
3. Determine the area of particular sensitivity, such as politics or religion, of the hostage taker and avoid conversation in these subjects. However, if terrorists want to talk about their cause, show an interest. Explain although you might not agree with your captors, you are interested in their point of view, emphasizing the latter point.
4. Become an active listener.
5. Small talk is better than no talk.
6. Do not argue with captors.
7. Make brief, casual eye contact during conversation; avoid prolonged eye contact.
8. Do not refuse to eat unfamiliar local dishes just because they don't look or smell appetizing. It is disrespectful to refuse to eat the food that the captors may themselves be eating.
9. Adapt to, but do not adopt, the hostage taker's value system (see "Stockholm Syndrome," later.)

Stress Management in Captivity

Long-term captivity often leads to exhaustion, lethargy, and depression. Oppose these feelings and try to maintain confidence.

1. Exercise.
2. Maintain sleep discipline and avoid sleeping too much.
3. Plan a schedule for the day and attempt to stick to it.
4. Keep a sense of humor.

Adjusting to Captivity

The hostage must make every effort to adjust to captivity.

1. Do not focus on mistakes made up to the point of captivity. Focus on the positive.
2. A positive attitude will help sustain dignity.
3. Smiles are contagious; the hostage should wear one.
4. Fight boredom. Engage in creative mental and physical activities.
5. Avoid setting anticipated release dates or allowing captors to establish these types of milestones.
6. Remember that the longer a hostage is held in captivity, the greater his or her chances of release or rescue.

Stockholm Syndrome

The Stockholm syndrome refers to stress-induced alteration of the hostage's behavior such that the hostage aligns with the hostage taker. This phenomenon is thought to be an automatic, unconscious emotional response to the trauma of becoming a victim. It is not uncommon for hostages to transfer anger from the hostage takers to the society or situation that created the dilemma in which they are now victims. Individuals held in the grip of the Stockholm syndrome have been known to actively participate with the captor group in terrorist activities.

Escape

Escape planning should begin at the onset of detention because escape may eventually prove to be a viable option.

1. Escape is considered a last resort but should be considered if conditions deteriorate to the point that the risks associated with escape seem less than the risks of remaining captive: torture, death of fellow detainees, or a credible threat of death.
2. In group hostage situations, detainees should organize and make every effort to establish and sustain communications with other hostages.
3. Terrorists often hold hostages in areas where the local population supports their cause. Do not assume locals are friendly, because they may return escapees to their captors.

Rescue

Statistically, most hostages who die are killed during rescue attempts. Hostages should, therefore, be mentally prepared for rescue attempts.

1. The first hostage execution, if known by the rescuers, increases the likelihood of a rescue attempt.
2. The hostage must be especially alert and cautious should he suspect that a rescue attempt is imminent or occurring.
3. At the first sign of an active rescue attempt, the hostage should drop immediately to the floor and avoid any sudden movement, especially with his hands.
4. From the rescuer's point of view, everyone on the scene is a terrorist until proven otherwise. Therefore, do not attempt to assist the rescue forces because you may be mistaken for a terrorist.
5. Expect to be tied up or otherwise restrained by rescue forces until you can be positively identified. If you argue, you may be subdued.

Release

Statistically, the odds favor the hostage being released.

1. The U.S. Government will not negotiate with terrorists. Our government will, however, work closely with the host government from the outset of a hostage-taking incident to encourage that government to exercise its responsibility under international law to protect all persons within its territories and bring about the safe release of hostages.
2. A number of factors can come into play (including ransom demands met by private efforts) so that the hostage is released by his or her captors. If an actual release has been arranged, pay close attention to the instructions the captors are giving when the release is taking place. Do not panic or attempt to run.

The references for this chapter can be found on the accompanying DVD-ROM.

Desert Travel and Survival

38

Edward J. Otten

This desert landscape is the indifference to our presence, our absence, our coming, our staying or our going. Whether we live or die is a matter of absolutely no concern to the desert. Edward Abbey, Desert Solitaire

THE DESERT ENVIRONMENT

Deserts are land areas that receive less than 10 inches (25 cm) of rain, unevenly distributed throughout the year. A number of climatic processes produce desert areas. The most influential are the six cells of cold air currents that descend at the poles and near the Tropic of Cancer and Tropic of Capricorn. These air currents, driven by the sun and rotation of the earth, create areas of relatively warm, dry conditions. Many of the world's deserts live in a "rain shadow," an area to the leeward side of a mountain range that prevents the small amount of moisture that is present in the air to move over the mountains. As the air rises, the moisture cools and precipitates in the higher elevations. Therefore, the area in the "shadow" of the mountain range receives little moisture. The air that does descend is quite dry and adds to the evaporative effect. The Atlas Mountains shadow the Sahara, the Andes the Patagonian, the Great Dividing Range the Australian, and the Sierra Nevada and Cascades the Great Basin deserts. The amount of rainfall is not an absolute indicator of "dryness" because the rate of evaporation and timing of the rainfall must also be taken into consideration. The amount and type of vegetation, soil composition, altitude, average temperature, wind speed, and solar radiation all contribute to "dryness" and desert formation. Antarctica would be the world's largest desert by the definition of less than 10 inches of rainfall annually, some areas of that continent having had no recorded rain in 30 years. There is a large amount of water present in the form of ice, but it is not available for use by plants. Antarctica has its own special survival problems not associated with precipitation and, for the purposes of desert survival, will not be considered here. In contrast to Antarctica stands the northern coast of Alaska, which receives less than 4 inches of rain annually yet nevertheless is quite wet because evaporation is so low there.

Deserts are one type of environment on Earth that is increasing in total area, likely because of human as well as geologic factors. Overgrazing, destruction of forests, global warming, and other aspects of increased human population contribute to desertification. Currently about 15% of the land area of the earth is desert (30% if Antarctica is included) (Fig. 38-1; Table 38-1). Most of the earth's deserts can be found between 30 degrees South and 30 degrees North latitude, making them hot

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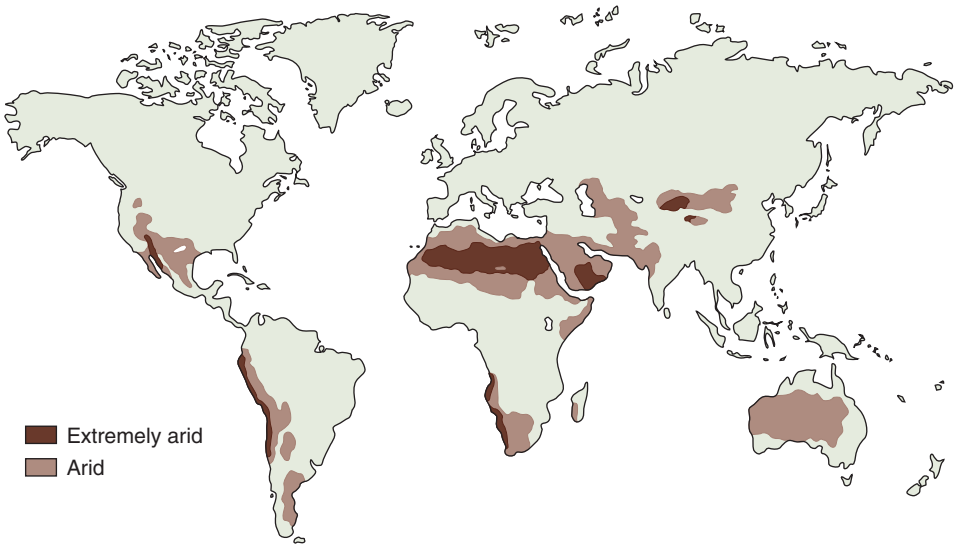


Figure 38-1. Desert areas of the world. Many of these areas are growing.

TABLE 38-1. Deserts of the World

NAME	CONTINENT	AREA (mi^2)	FEATURES
Western Sahara	Africa	103,000	Dunes, mountains
Great Erg	Africa	151,460	Dunes
Tanezrouft	Africa	57,900	Rocky, mountains
Libyan	Africa	463,320	Rocky, dunes, oases
Fezzan	Africa	212,806	Rocky, mountains
Egyptian	Africa	254,828	Basin, plateau, oases
Sinai	Africa	23,550	Mountains
Niger	Africa	179,270	Dunes, mountains, oases
Darfur	Africa	170,000	Mountains
Nubian	Africa	162,800	Rocky, dunes
Sahel	Africa	1,930,500	Steppe
Mauritania	Africa	278,000	Dunes, plateaus
Somalia	Africa	226,700	Mountains, plateaus
Kalahari	Africa	100,390	Dunes, savannah
Namib	Africa	36,300	Dunes, plateaus
Andalusian	Europe	102,700	Rocky, dunes
Arabian	Asia	888,030	Dunes, mountains
Syrian	Asia	200,000	Rocky, plateaus
Negev	Asia	4,630	Plateaus, rocky
Taklimakan	Asia	125,000	Basin, dunes
Thar	Asia	82,625	Dunes, cold
Gobi	Asia	501,930	Plateaus, rocky, cold
Karakum	Asia	266,410	Basin, plateaus, cold
Sonoran	N. America	120,000	Mountains, alluvial
Death Valley	N. America	5,700	Basin, mountains
Gran Desierto	N. America	119,690	Basin, mountains
Mojave	N. America	25,096	Basin, mountains
Great Basin	N. America	189,190	Basin, mountains
Chihuahua	N. America	200,000	Basin, mountains
Atacama	S. America	77,220	Mountains, rocky, cold
Great Sandy	Australia	139,000	Basin, dunes
Simpson	Australia	115,830	Dunes, rocky
Great Victoria	Australia	223,660	Dunes, alluvial

as well as dry. These deserts include the Sahara, Arabian, Kalahari, Australian, Atacama, Thar, Namib, and southwest United States. About 50% of Africa is desert; the Sahara by itself is almost as large as the United States. About 8% of the United States, or 300,000 square miles, is desert. Most of the U.S. desert areas are adjacent to National Parks and Forests and are frequently visited, for example, the Grand Canyon, Big Bend, Arches, Zion, Organ Pipe, Joshua Tree, Great Basin, Saguaro, and Capital Reef. Beyond 40 degrees South and North latitude and at elevations over 10,000 feet are the "cold" deserts, which have wide swings in temperature, for example, the Patagonian, Turkestan, Gobi, and Takla Makan. The large temperature variations in desert regions are greater at higher elevations and latitudes, but are present in all deserts. Lack of vegetation, cloud cover, and ground-water surface allows 90% of solar radiation to reach the desert surface. By contrast, a forest may reflect 50% to 60% of the solar radiation, and its vegetation disperses the rest. At night, lack of cloud cover and vegetation allows almost 100% of the accumulated heat to escape, as opposed to only 50% from a humid climate. This explains why the desert temperature may reach 120°F (49°C) during the day and drop to 40°F (5°C) at night. Tropical rainforests may only reach 95°F (35°C) during the day, but at night the temperature only drops to 85°F (30°C).

It might seem that the extreme desert climate would only allow for sparse life, but that is not the case (Fig. 38-2). Death Valley, one of the harshest environments in North America, where air temperatures have been recorded at 134° F (57° C), has 600 species of plants, 30 species of mammals, 25 species of reptiles, and 2 species of fishes. Oases are found in most deserts. They are isolated depressions usually fed by a constant source of water. Often, underground springs and wells supply moisture for plants and animals. Often one must dig to find water at the lowest point of the depression. Many named oases have supported camel caravans, allowing them to move from oasis to oasis and thus cross an otherwise impenetrable desert. Many ancient oases have wells hundreds of feet deep and because of overuse are gradually drying up. When the water is used up, the oasis disappears, along with its desert life.

All desert flora and fauna have one guiding principle for survival, which is to *conserve water*. The ground surface of the desert has the highest temperature because of the direct effect of solar heat and wind. Therefore, during the hottest times of the day, most animals are either below the surface, in underground burrows, or above the surface in available vegetation, cacti, trees, or shrubs. Most animals forage from dusk till dawn when temperatures are cooler. Some mammals, such as kangaroo rats, never drink but obtain necessary water through plant seeds. Plants have evolved a number of survival skills to maintain water, including stomata that are closed during the day, and crassulacean acid metabolism (CAM) photosynthesis. The latter allows for accumulation of carbon compounds at night through the dark reaction. These compounds are converted to carbon dioxide during the day when the stomata are closed. Other adaptations include stem photosynthesis in plants without leaves, thick cuticles, water storage tissues, and widespread shallow root systems. Desert plants also have evolved a variety of defense mechanisms, such as production of toxic compounds that act as herbicides to other plants, and the formation of needles, spines, and thorns that dissuade browsing animals. Obviously, humans are not able to evolve these physiologic changes, but must rely on behavior, technology, and other adaptations to mimic the methods used by indigenous desert dwellers.

Figure 38-2. Desert landscape. Note the physical features and abundant flora.

PREPARATION

All things being equal, preparation improves the likelihood of survival. However, things are never equal, so luck is probably the most important, albeit the most uncontrollable, factor. The controllable factors are mental and physical conditioning, clothing, survival kit adequacy, and survival skills. These may allow one to survive even in the most extreme conditions.

Mental preparation is key to any survival situation. The "will to survive" has been shown to be the most important factor in the outcome of a number of situations. Knowledge of the terrain features, weather, animal and plant life, and potential hazards should all be studied before travel to a desert area. Not only does this increase one's chances for survival, but it also enhances enjoyment of the desert environment. A number of recommended readings are included in the Suggested Readings section at the end of the chapter. Practical experience in finding water and food, navigation, and constructing shelters is more valuable

than reading about it. Time spent in attending a course on survival in general or desert survival in particular may be invaluable if one is later in a true survival situation.

Physical conditioning and acclimatization are as important for desert travel as for mountaineering. Desert travel is difficult under most circumstances. The terrain is rough and may include sand dunes, sharp loose rock, flash floods, steep grades, and hot surfaces. Lower-body conditioning helps prevent the ankle and knee injuries that can force a survival situation in a harsh climate. Acclimatization involves three well-described physiologic adaptations and may take 10 to 14 days. These are increase in the volume of sweat and number of active sweat glands, decrease in concentration of electrolytes in sweat, and sweating at lower body temperature. These can be induced before arriving in a hot climate by the use of a sauna or vigorous exercise to raise body temperature.

Clothing

Clothing selection for desert travel is somewhat different than for most other wilderness activities. The less exposed skin, the better. Although cotton is not good for most cold, wet climates, it is useful in the desert. Light-colored clothing reflects sunlight and lowers skin temperature. Rip-stop cotton (cotton material with nylon threads latticed within it) is best because it resists rips that are common in the desert. It is light enough to allow heat to escape, does not have a clammy feeling in low humidity, and protects against some ultraviolet rays and blowing sand. If rip-stop cotton is not available, any tight weave cotton is adequate. Long sleeves and long pants are a must to protect against spines, thorns, splinters, and insects. More important, they protect against solar radiation that causes sunburn and increased body temperature, and they trap more cool air next to the skin. Sweat that is trapped decreases water loss through evaporation. Trousers can be tucked into the tops of socks to protect from insects; sleeves should be not be rolled up in order to minimize risk for sunburn and heat gain. Gaiters can be worn to protect lower legs and the inside of footwear from sand, rocks, and dust. In a survival situation, puttees (wraps that extend from the tops of the shoes to the knees, either over trousers or bare legs) can be made of strips of cloth, elastic bandages, or stockings to protect the lower legs. They can be incorporated into the socks and wrapped to above the knees in a fashion similar to gaiters. Because of the wide temperature swings, a pile jacket or sweater is necessary at night. Layering, just as in cold climates, is the best means of preserving body warmth. In a survival situation, any insulating material, such as seat cushions, newspapers, or dry grass, can be used to insulate whatever clothing is available. Although wind is more of a problem than rain, a Gore-Tex jacket is also recommended, especially for "cold" deserts. A wide-brim hat or kepi (a cap with a cloth extending from the back protecting the neck) is necessary to protect the head, face, neck, and ears. In a survival situation, an expedient head covering can be made from whatever material is at hand (Fig. 38-3). A cotton cravat, bandanna, or handkerchief can be used to keep the head and neck cool by soaking the material in water (if plenty of water is available; do not use precious drinking water) and then placing it on the head, followed by a hat. Alternatively, it can be wrapped around the neck and shoulders underneath a shirt. Commercially produced (Cooldanna, Climatech, Stacool) neck wraps and vests, which contain crystals that can be soaked in water and then become cool through a chemical reaction, are also available, but

Figure 38-3. Expedient head coverings.

probably do not add much in the way of total-body cooling in extreme environments. The cravat can also be used during dust storms to protect the nose and mouth, as a towel, or to absorb moisture from plants when obtaining water.

High-top (6 inches [15 cm]) boots composed of leather or synthetic materials are necessary to prevent sand, rocks, and burrs from entering the boot, support the ankle on rough terrain, insulate the foot from hot surfaces, and prevent the boot being pulled off in soft ground. Boots should be well broken in before hiking. Military-issue boots with metal spike protection can become extremely hot in desert conditions and should be avoided. Running shoes do not insulate the feet well and may become extremely hot. Socks should not be cotton, because of the risk for blisters and lack of wicking and insulation. Polypropylene or a combination of polypropylene and wool is best for socks because of less friction and thicker material. Foot care is extremely important, especially in a survival situation when walking is the only means of transportation. Feet should be inspected for blisters, foreign bodies, and abrasions on a regular basis while hiking. Socks should be changed frequently, at least twice a day, to allow them to dry out and to remove accumulated dust and sand. Leather gloves are desirable to

protect hands from hot objects, plant spines, thorns and splinters, insects, and blisters. Abrasions and lacerations to the hands can quickly become infected in conditions where hand washing is difficult. Eye protection becomes very important in the desert, especially when traveling. Solar radiation, both direct and reflected, can cause keratitis similar to snow blindness. More commonly, blowing sand, dust, and insects may cause corneal abrasions and conjunctivitis. Contact lenses are difficult to manage in the dry, dusty environment. Tinted goggles are best, just as with mountaineering, but glacier glasses or standard sunglasses can be used. Duct tape, adhesive bandages, or other material can be used to fashion side shields for regular glasses to prevent sand and dust from entering through the sides. Insect head nets can be lifesaving, especially in African, Arabian, and Australian deserts, where insects and insect-borne diseases are a particular problem.

Survival Kit

The survival kit must be carried at all times, have quality equipment and supplies, and be protected from the elements. Each item should have multiple uses if possible. There must be items that can be used for shelter, signaling, fire building, first aid, and other uses (Box 38-1). Many items will routinely be carried by most hikers and backpackers, but some specialized items should be added. One's clothing is the basic survival shelter and if properly selected will be the first step in protection from the climate. General-use items include a Swiss Army knife with as many blades as possible. The "Swiss Champ" includes, among many other features, a magnifying glass, which is quite useful for removing cactus spines or starting fires. Parachute cord (eight-strand) has multiple uses, including shelter construction, creation of snares, and fishing nets and lines. A signal mirror; whistle; matches, metal match, or lighter; first-aid supplies; compass; safety pins; water disinfection tablets; and plastic bags can be stored in a relatively small container that should be kept on one's person at all times. The first-aid kit should contain bandages, insect repellent, sunscreen, lip balm, antiseptic ointment, aspirin, antihistamines, tape, and any prescription medications. The desert survival kit should include a 5- by 5-foot (1.5- by 1.5-m) piece of plastic, 4-foot (1.2 m) length of plastic tubing, and metal cup (all for making a solar still), emergency blanket (silver on one side, red on the other), extra sunglasses or eye protection, nylon canteen (minimum 5-quart [4.7 L] size), and extra sunblock.

Box 38-1. Desert Survival Kit

Swiss Army knife
Parachute cord
Compass
Topographic map
First-aid kit
Goggles
Lip balm
Leather gloves
Safety pins
Mosquito head net
Waterproof container
Rescue blanket
Plastic sheet
Plastic tube
Metal cup
Water container (5 qt [4.7 L])
Hat
Sweater or pile jacket
Gore-Tex jacket
Duct tape
Hard candy
Plastic bags
Matches
Metal match
Metal mirror
Whistle
Water filter or iodine
Peanut butter
Flashlight
Sunscreen
Insect repellent
Cravat

PRIORITIES

The "rule of 3s" gives us a priority list for survival. One can live 3 minutes without oxygen, 3 hours without warmth, 3 days without water, and 3 weeks without food.

Water

The key to desert survival is water. Unfortunately, water weighs 8 pounds (3.6 kg) per gallon (3.8 L), and individual needs may be up to 2 gallons (7.6 L) per day. Always carry as much water as possible. Collapsible canteens can carry several quarts of water and keep it cooler (because the surface heat absorption to volume is less) than can hard plastic or metal (Fig. 38-4). Drink at every stop and while hiking if using a drinking tube. Flavoring and cooling water increase palatability and thus consumption. Most hikers do not carry enough water for more than 1 day, so having a method for acquiring water is mandatory. Solar stills, vegetable stills, digging along arroyos and in dry lake beds, wiping dew from plants in the early morning, and extracting water from succulent plants are all methods for obtaining water in a desert survival environment.

Although there are a number of methods for obtaining water in the desert, minimizing water loss might be a better strategy

Figure 38-4. Collapsible 5-quart canteen.

Figure 38-5. Desert water hole—currently occupied.

Figure 38-6. Desert arroyo or wadi.

for a human with limited water resources. All desert plants and animals have developed adaptive mechanisms for conserving water. Humans, if they are to survive, must develop strategies for conserving their sweat, not their water. Adolph measured water intake, activity, and ambient temperature of World War II soldiers during desert operations. Soldiers who rested in the shade had significantly less water losses than those who exerted themselves in the heat of the day, and could extend the length of time they could survive, despite similar water intake. Although this may be intuitive, most desert travelers limit neither their travel to cooler times of the day nor their work activities to the shade. Employing a strategy of resting in the shade from 1000 hours until 1500 hours, drinking adequate water based on the appearance of the urine, and keeping as much skin covered as possible have decreased the number of heat casualties in the military during training exercises. Although thirst and amount of sweat on clothing are poor indicators of hydration status, the color and amount of urine produced can roughly estimate it. The darker the urine, the more concentrated it is, indicating a greater degree of dehydration. If potable water is available, it should be consumed; it is better to conserve sweat rather than water. If there is a limited amount of water, food should not be eaten unless the food contains a large amount of water. The metabolism of food, especially protein and fat, and excretion of waste products require potentially unnecessary consumption of water. Only potable water should be drunk because vomiting and diarrhea caused by contaminated water could quickly become fatal in the desert. Water obtained from lakes, streams, wells, or springs should be considered contaminated and must be made potable before drinking. Water holes may attract dangerous game as well as potential food (Fig. 38-5).

Desert water may be found by looking for animal signs, such as trails or spore. Some plants, such as cottonwood, sycamore, willow, and cattails, may be indicators of water. Water may sometimes be found by digging at the outside bend of a dry riverbed or stream (*arroyo* and *wadi* are terms used to indicate rain run-off channels that may contain water during the rainy season) (Fig. 38-6). Rainwater, dew, and water obtained from a solar still or vegetable are relatively potable. Urine, seawater, alkaline pools, and brackish water should never be drunk. They contain large amounts of solutes that would require more water to excrete than they provide, thus hastening dehydration and

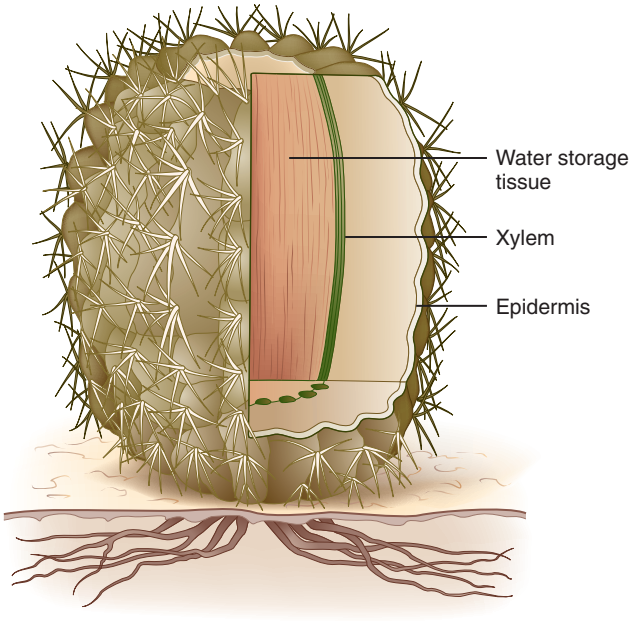


Figure 38-7. Barrel cactus showing pulp that can be a source of water.

renal failure. Liquid from radiators is contaminated with glycols and should not be drunk or used in solar stills. Many plants, such as barrel cacti and traveler's tree, and animals, such as the desert tortoise, contain water that can be used in an emergency (Fig. 38-7).

A solar still (Figs. 38-8 to 38-10) can be constructed by placing a 5- by 5-foot (1.5- by 1.5-m) piece of clear plastic over a hole 3 to 4 feet (0.9 to 1.2 m) in diameter and dug 3 to 4 feet deep in the ground into which vegetation, urine, or brackish water has been placed. Solar energy causes water to evaporate within the hole and collect on the underside of the plastic. Because it cannot escape, it will drip back into a container placed at the bottom of the hole. A tube can be attached to the inside of the container so that the water can be drunk without

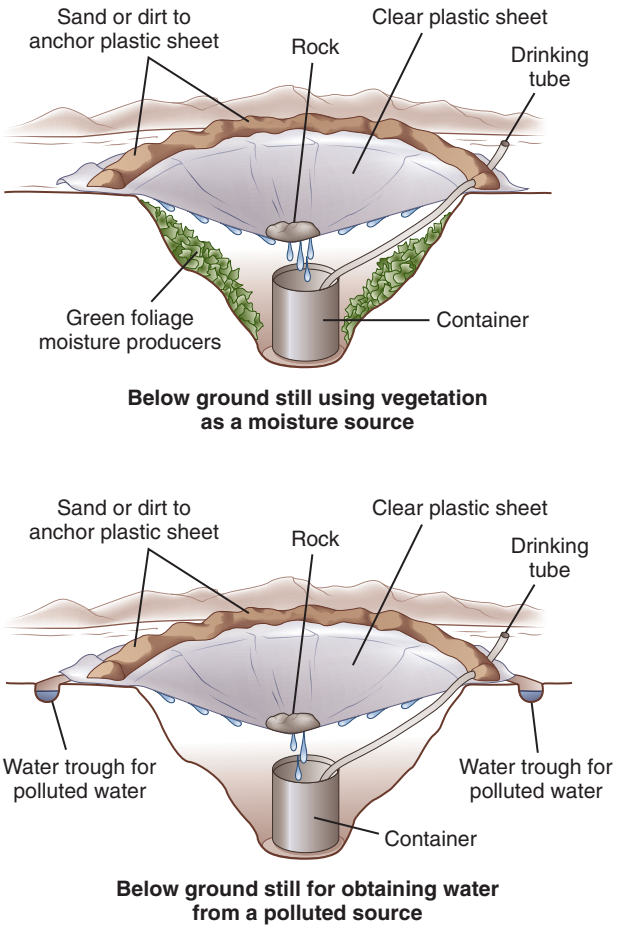


Figure 38-8. Diagram of solar still.

Figure 38-9. Photo of solar still. Note the moisture on the plastic.

dismantling the still. A plug should be put in the drinking end of the tube to prevent clogging. The amount of water produced depends on the amount of moisture in the hole, the amount of sunshine directed at the still, and the size of the plastic sheet. A still dug into a dune or dry sand will not produce much water. Dew, rainwater, and edible animals may fall into the still as a bonus. Moisture can be wiped off plants during the early morning and can be squeezed from the pulp of certain plants, such as yucca or stool. Milky, bitter, or sour pulp should not be used, and the pulp should not be eaten. Water can be made potable by boiling, or using filters or chemicals (see Chapter 61).

Shelter

Desert shelters are of three main types: natural, well-prepared human-made, and improvised human-made. Natural shelters are caves and rock overhangs. When using natural shelters, one must remember that there may already be other inhabitants that need eviction. Rattlesnakes, tortoises, ground squirrels, lizards, and skunks often use caves and other cool areas during the heat of the day. They may be sources of food, but are also potentially dangerous to humans. Some reptiles are venomous, and many mammals carry rabies or plague. They can usually be removed with a stick or dispatched with a rock. Well-prepared human-made shelters include tents, vehicles, and buildings. Most vehicles become very hot inside, which may hasten heat illness and dehydration. It is better to sit in the shade of an automobile than to be inside it during the heat of the day. Tents and buildings are best if there is adequate ventilation. Improvised shelters are typified by the desert trench shelter, shade shelter, or lean-to. Any structure that provides shade, protects from wind and blowing sand, and decreases heat gain or loss is minimally adequate. The hottest area in a desert climate is at the surface of the ground, so getting above the ground or below the ground decreases exposure to the highest temperatures. Most desert animals take advantage of this fact by their behavior. Birds perch high in trees in the shade of a branch or in some cases inside of a cactus. Most mammals and reptiles burrow into the ground, where just 3 feet below the surface the temperature may be 30% cooler. Humans can imitate this behavior by constructing a desert trench shelter (Fig. 38-11). This is a trench dug in the ground 2 to 4 feet (0.6 to 1.2 m) wide, 2 to

Figure 38-10. Photo of solar still.

3 feet (0.6 to 0.9 m) deep, and about 6 feet long. A barrier can be made of any cloth material at hand, such as a blanket, sheet, poncho, tarp, or space blanket. This barrier is placed over the trench, about 18 inches (45.7 cm) above the bottom, and its edges are weighed down with rocks or sand. Another similar barrier (the first one can be doubled back on itself if it is large enough) is placed about 12 to 18 inches (30.4 to 45.7 cm) above the first barrier and also weighed down. This construction creates an insulating barrier that traps cooler air in the bottom of the trench and reflects solar radiation from the top. Ideally, a reflective space-type blanket is used for the outer layer, which will increase the amount of heat reflected.

Removing the seat from a vehicle and sitting above the ground in the shade of the vehicle or constructing shade with a blanket or tarp and available materials can provide some shelter in the heat of the day until a more adequate shelter can be built (Fig. 38-12). At night, when temperatures drop, the shade trench remains warmer because of the heat produced by the inhabitant. The top barrier, with its reflective surface, can be reversed to reflect heat back into the trench. Any insulating material can be added to the floor of the shelter to decrease heat loss to the ground. Given enough time, a shelter such as a tepee, tent, or lean-to can be constructed using tarps, blankets, parachute panels, or other materials (Fig. 38-13). Construction should take place in the cooler times of the day to conserve sweat and water. If one has a reliable source of water, such as a desert water hole or "tank" (natural collection of water in nonporous rock), then it is best to stay in that place and try to be rescued rather than attempting to travel large distances without adequate water.

Food

Food is not as immediate a problem as water in the desert (see Chapter 65). Most humans have an extra 50,000 to 70,000 calories that they store in the form of fat and can exist for weeks without eating. Food in the form of animals or plants is usually available if water is available. Cactus fruit, such as prickly pear (Fig. 38-14), can be eaten when peeled. Legumes, such as acacia, mesquite, and palo verde, produce beans that can be crushed and mixed with water to form tortillas (Fig. 38-15). Yucca stems, cattails, agave stems, and prickly pear pads can be cooked and eaten. Avoid plants with milky saps or red berries and other plants that cannot be positively identified as edible. Mushrooms should not be eaten. It is essential to become familiar with the common poisonous and edible plants in any area through which you will travel. Insects, birds' eggs, grubs, tortoises, and other slow movers can be captured by hand. Most small animals, such as snakes, lizards, and birds, can be killed with a rock or stick. Jackrabbits, ground squirrels, and rats may be snared or trapped. Large mammals should be avoided because the likelihood of killing one of these without a firearm is small, whereas the likelihood of becoming a casualty is high. All meat should be cooked or dried before ingestion. Animals to avoid as food or in general include caterpillars, spiders, scorpions, centipedes, millipedes, toads, and bats, as well as some mammals that carry plague, tularemia, Hanta virus, and rabies.

Hazards

In addition to the hazards of heat and dehydration, there are other hazards that one may encounter in the desert. Blowing sand and dust created by vehicle traffic may cause eye, nose, mouth, and skin irritation. Desert winds up to 100 mph (161 km/hr) can blow for days, creating immense sandstorms and dust storms that can cover large areas, reducing visibility to near zero. In addition to the sand and dust, other objects can be transported with the ferocity of a hurricane. Vehicles, tents, and buildings can be demolished and roads obliterated. Sandstorms blowing for days have caused destruction in large cities, such as Melbourne and Baghdad. Exposed skin must be protected, and shelter should be sought within a hard-sided building if possible. The possibility of an individual being buried is remote, but drifting sand can block doors and strand vehicles.

Quicksand is another hazard. There are two types of quicksand. The first is a mixture of sand and water found along the shores of lakes, seas, and estuaries of rivers. This type may look solid but is a suspension of fine sand and water that cannot compact. The second type is fine sand deposited by wind in hollow depressions. Depending on the depth of the depression, one could sink 15 to 20 feet (4.6 to 6 m). A person caught in quicksand can usually escape by flattening out and swimming to firm ground because most quicksand areas are small. Animals may panic and struggle, hastening their submersion. A rescuer can usually pull the victim to firm ground using a rope, belt, or

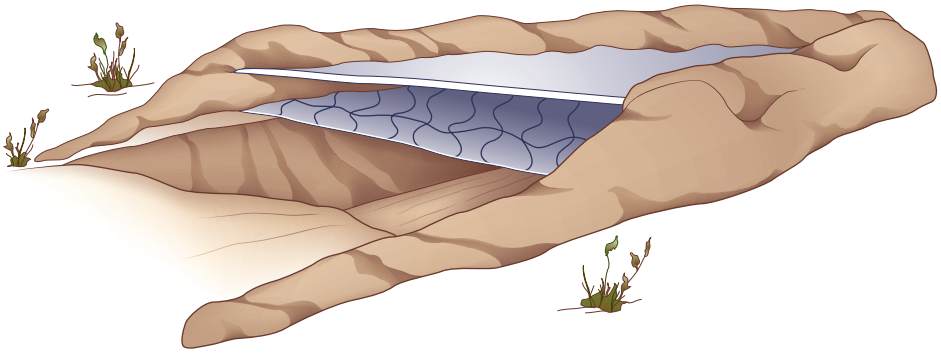
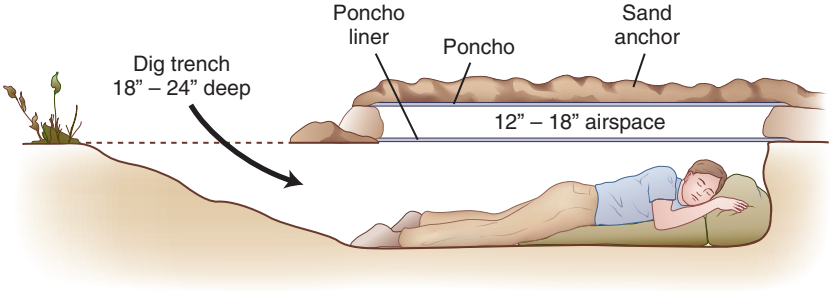


Figure 38-11. Desert shade shelter or trench shelter.



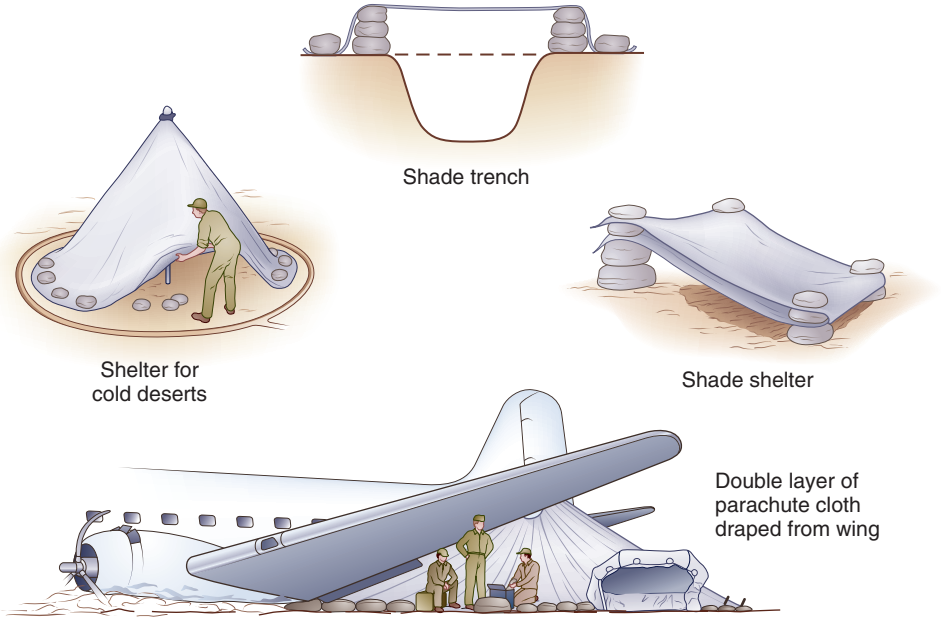


Figure 38-12. Expedient shelters.

Figure 38-13. Desert tarp shelter.

Figure 38-14. Prickly pear fruit.

Figure 38-15. Mesquite seed pods.

strap. Removing one's pack and throwing it to firm ground allows for easier escape.

Animal hazards include venomous snakes and arthropods, carriers of zoonotic illnesses, and large mammals (Fig. 38-16; Box 38-2).

Plant hazards include poisonous varieties that may cause dermatitis or systemic symptoms if ingested. Many types of plants produce spines or thorns, which commonly cause infections if they break the skin.

TRAVEL

Even in the best of circumstances, hiking in the desert can be difficult. Most modern travel is performed with high-clearance, 4-wheel-drive vehicles. These vehicles should have sand mats (for added traction on soft surfaces) and a winch and cable for self-extracting the vehicle or rescuing another vehicle. Extra water, spare parts (fan belts, hoses, spark plugs, fuses, bulbs), additional fuel and oil, shovel, two jacks, spare battery, spare tires and tire patching materials, radio, and tool kit are manda-

Figure 38-16. Western diamondback rattlesnake.

Figure 38-17. Travelers on camel.

Box 38-2. Dangerous Desert Animals

Arthropods

Venomous

Spiders, scorpions, assassin bugs, ants, bees, wasps, centipedes, millipedes

Zoonotic Disease Vectors

Fleas, sandflies, ticks, mites, mosquitoes, kissing bugs, flies

Reptiles

Venomous

Lizards, rattlesnakes, coral snakes, desert vipers, desert black snake, cobras, Australian elapids

Nonvenomous but Risk for Attack

Crocodiles, monitor lizards

Mammals

Zoonotic Disease Vectors

Dogs, rodents, rabbits, bats, squirrels, cats, primates

Mechanical Injuries

Camels, carnivores, donkeys, antelope, deer, elephants

See also Part Six.

tory for desert travel. Traveling in convoys is a must, both for safety and, in some places, for security. Salt flats, mud flats, and soft sand can bog down even 4-wheel-drive vehicles. Never attempt to cross arroyos or wadis when they are flooding.

Many areas offer no access to wheeled vehicles, so travel by camel, donkey, or horse is still common. Camels remain the most efficient desert transportation and can travel for long periods without food or water (Fig. 38-17). They also are quite resistant to blowing sand, heat, and temperature changes. Expert assistance is needed for travel by camel. Horses and donkeys are limited to short distances and require more food and water.

Current topographic maps and a good compass are essential, whether traveling by vehicle, by animal, or on foot. Local authorities should be consulted concerning water sources; these should be marked on the maps. A copy of the map should be left with someone who knows the travel plans. The route and campsites should be indicated on the map. In that way, if one becomes lost or injured, search and rescue teams will know where to initiate the search. If one becomes lost or injured, it is usually best to stay in one place and signal for help, using a mirror or other reflective object during the day and a fire at night. Cellular telephone, radio, or emergency locator transmitter may aid searchers. Flares and whistles should be used only when searchers are nearby or a search aircraft is moving in your direction. Geometric patterns drawn on the ground, such as "SOS," "HELP," or a large "X," may aid aircraft in identifying the position of a distressed person. Rocks, contrasting soil, plants, or clothing can be used to produce the patterns. It is difficult to see a single human on the ground; therefore, it is always best to stay near a vehicle if possible. An automobile can be a good source of signaling equipment. The windscreen and windows reflect light, and the mirrors can be removed and used to signal. Oil can be drained, placed in a hubcap, and mixed with sand. When set on fire, it produces dense black smoke. The hubcaps can be used to dig a desert trench shelter, the battery to start a fire, and the seat covers and floor mats for shelter and footwear. If one has adequate water, knows the direction of travel, has good footgear, and decides to try to walk to safety, it is imperative to leave a note outlining plans in a conspicuous place or to draw an arrow on the ground with rocks pointing in the direction of travel. Direction finding can be accomplished without a compass using the sun or stars (see Chapter 85). Before traveling in the desert, it is best to know the location of key terrain features, such as rivers, highways, mountain ranges, and cities. Power lines, stream beds, and pipelines may also lead to civilization, but it may require days to arrive. It is dangerous to travel through arroyos or wadis because of the danger of flash flooding. Rainfall in mountains several miles away can wash down these formerly dry stream beds, pushing tons of mud, rocks, and water in a lethal torrent. Sand dunes should be avoided because of the amount of energy

required to traverse them. Salt marshes may contain soft ground that traps the hiker. Travel in the cool of the night to conserve water, and use the stars for direction. The dangers at night are that many desert animals are more active, and the chances of falling from a cliff, stepping into a hole, or stumbling into a cactus are greater.

SUGGESTED READINGS

Adolph EF: Physiology of Man in the Desert. New York, Hafner Press, 1969.
Bender GL: Reference Handbook on the Deserts of North America, London, Greenwood Press, 1982.
Benson L: The Cacti of the United States and Canada. Stanford, CA, Stanford University Press, 1981.
Brown S: World of the Desert. New York, Bobbs-Merrill, 1963.
Craighead FC, Craighead JJ: How to Survive on Land and Sea, 4th ed. Annapolis, MD, Naval Institute Press, 1984.
Foster L: Adventuring in the California Desert. San Francisco, Sierra Club Books, 1987.
George U: In the Deserts of this Earth. New York, Harcourt Brace Jovanovich, 1976.
Larson P: The Deserts of the Southwest. San Francisco, Sierra Club Books, 1977.
MacMahon JA: Deserts. New York, Alfred A. Knopf, 1990.
Marriott BM (ed): Nutritional Needs in Hot Environments. Washington, DC, National Academy Press, 1993.
Stoppato MC, Bini A: Deserts. Buffalo, NY, Firefly Books, 2003.
U.S. Air Force: Search and Rescue Survival Training, AF 64-4. Washington, DC, Department of the Air Force, 1985.
U.S. Army Research Institute of Environmental Medicine: Sustaining Health and Performance in the Desert. Alexandria, VA, Defense Tactical Information Center, 1990.
Wagner FH: Wildlife in the Deserts. New York, Harry Adams, 1984.

39 White-Water Medicine and Rescue

Eric A. Weiss

Rivers have what man most respects and longs for in his own life and thought—a capacity for renewal and replenishment, continual energy, creativity, cleansing.
John M. Kauffman, Flow East

Rafting, canoeing, and kayaking have become the third largest outdoor recreation industry in the United States. Recent estimates place the number of rafting participants at 9.8 million. Participation in canoeing and kayaking (including flat-water paddlers) rose from 16.7 million in 1994–95 to 22.6 million in 1999. According to the Outdoor Industry Association, in 2002, there were 6.5 million white-water kayakers, with participation in river sports growing at a rate of 15% annually. Kayakers are almost 70% male, whereas the male-to-female ratio of rafters is somewhat more reflective of the U.S. population at 55% male and 45% female.

Kayaks and rafts are also used by law enforcement officers, park rangers, and game wardens to patrol and manage their territories. New equipment designs have opened up more difficult rivers for exploration and commercial recreation.

It is not surprising that the number of river-related accidents and deaths continues to increase. The American Canoe Association reports that about 130 white-water fatalities occur each year. This chapter examines the unique and dynamic hazards associated with rivers and white-water paddling. Safety equipment, accident prevention, common injuries, environmental hazards, medical management, and swift-water rescue are also reviewed.

DEMOGRAPHICS

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HISTORICAL PERSPECTIVE

White-water boating as a recreational activity began in the United States in earnest during the late 19th century when adventurers attempted to emulate Major Wesley Powell's Colorado River expedition by rowing boats down many of the West's large rivers.[25] These heavy wooden boats were replaced by inflatable rafts after World War II, when surplus neoprene assault boats and life rafts became available for civilian use.[3] In 1966, fewer than 500 people boated the Colorado River through the Grand Canyon in an entire year. Recently, the figure exceeded 500 per day.[25]

Rafting did not become popular in the eastern United States until the early 1960s. In 1968, commercially guided raft trips were offered for the first time on the New River in West Virginia.[51] The Chattooga River in Georgia attracted many rafters after the movie Deliverance was filmed there in 1971. The Youghiogheny River in Pennsylvania and the South Fork of the American River in northern California have become the two most heavily rafted rivers in the country.

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It is not surprising that the number of river-related accidents and deaths continues to increase. The American Canoe Association reports that about 130 white-water fatalities occur each year.[49] This chapter examines the unique and dynamic hazards associated with rivers and white-water paddling. Safety equipment, accident prevention, common injuries, environmental hazards, medical management, and swift-water rescue are also reviewed.

HISTORICAL PERSPECTIVE

White-water boating as a recreational activity began in the United States in earnest during the late 19th century when adventurers attempted to emulate Major Wesley Powell's Colorado River expedition by rowing boats down many of the West's large rivers.[25] These heavy wooden boats were replaced by inflatable rafts after World War II, when surplus neoprene assault boats and life rafts became available for civilian use.[3] In 1966, fewer than 500 people boated the Colorado River through the Grand Canyon in an entire year. Recently, the figure exceeded 500 per day.[25]

Rafting did not become popular in the eastern United States until the early 1960s. In 1968, commercially guided raft trips were offered for the first time on the New River in West Virginia.[51] The Chattooga River in Georgia attracted many rafters after the movie Deliverance was filmed there in 1971. The Youghiogheny River in Pennsylvania and the South Fork of the American River in northern California have become the two most heavily rafted rivers in the country.

Figure 39-1. River rescue. The plastic kayak has revolutionized white-water sports. (Courtesy of Paul Auerbach, MD.)

Technologic advances have revolutionized river running. Electronically welded plastic has largely replaced rubber as the primary material used in raft construction, making the vessels lighter, stronger, and easier to repair. Self-bailing rafts, introduced in 1983, are now ubiquitous and provide greater maneuverability, allowing rafters to run rivers previously considered too difficult and dangerous. Unfortunately, greater mobility has been paralleled by an increase in the number of accidents occurring far from medical care.

A major innovation in kayaking was the development of the plastic kayak, first manufactured in 1972 by the Holloform Company[51] (Fig. 39-1). Kayaks had been previously constructed from resinous materials, such as fiberglass and Kevlar, which were more fragile and less likely to "broach," or wrap around rocks. Paddlers were reluctant to run steep, rocky rivers for fear of breaking their boats. Most recreational white-water kayaks are now made of molded polyethylene plastic, which does not break apart and has the potential to fold when broached or pinned, trapping the paddler.

The hulls or bottoms of kayaks have undergone significant changes during the past 5 years. Older-model kayaks had rounded bottoms or displacement hulls. Most white-water kayaks today have flat bottoms, or planing hulls. The flat bottom makes the kayak more stable (both on its bottom and its sides). A rounded bottom rocks in the water from side to side, whereas a flat bottom does not. Newer boat technology, combined with shorter kayaks and more advanced paddling skills, allows better maneuverability in tight, steep rapids and is pushing the limits of navigable rivers. Even Niagara Falls has been successfully run by a kayaker!

The enormous popularity of rafting and kayaking has led to exponential growth of professional guide services. In 1990, 35 million people were taken down U.S. rivers by commercial companies.[49] Faced with increased competition, guide services have been leading inexperienced clients with little formal training and few practical skills into difficult and dangerous rivers (Fig. 39-2). In the summer of 1988, five U.S. executives died after their raft flipped on the Chilco River in British Columbia. One of the survivors was reported to have said, "We looked at white water as sort of a roller coaster ride."[47]

Figure 39-2. Class V commercial rafting on the Chattooga River, Georgia. (Courtesy of Robert Harrison, Whetstone Photography.)

MORBIDITY AND MORTALITY

Deaths are relatively rare in white-water sports. In Colorado, fewer people die while engaged in rafting, canoeing, and kayaking than in climbing, bicycling, and skiing. Figures compiled by the Colorado Department of Public Health and Environment showed that 69 people died in climbing or hiking accidents, 36 while bicycling, and 32 while snow skiing during a 3-year period ending in 1995. Rafting, canoeing, and kayaking incidents, by comparison, resulted in 19 fatalities (Table 39-1). A report from the American Whitewater Association in 2000, based on data from 30 managed rivers from 1994 to 1998, placed the fatality rate of rafters, canoeists, and kayakers at 0.87 per 100,000 user-days.[53] The fatality rate for kayakers alone was calculated to be 2.9 per 100,000 participants. The fatality rates of other outdoor sports are listed in Table 39-2.

A retrospective analysis of injury reports submitted by commercial rafting outfitters to the West Virginia Division of Natural Resources from 1995 to 1997 revealed a total of 200 injuries with a resulting overall injury incidence rate of 0.263 per 1000 rafters. The average age of injured persons was 33.14 years; 53.3% were male, and 59.8% had previous rafting experience.[52]

TABLE 39-1. Recreational Fatalities in Colorado, 1993–1995*

ACTIVITY	RANK	FATALITIES
Climbing/hiking	1	69
Bicycling	2	36
Snow skiing	3	32
Swimming	4	25
Canoeing/kayaking/rafting	5	19
Horseback riding	6	18
Boating/water skiing	7	13
Fishing	8	11
Hunting	9	6

*Two hundred fifty-two deaths were the result of recreational activities.
Data from Colorado Department of Public Health and Environment.

TABLE 39-2. Fatality Rates of Different Activities

ACTIVITY	FATALITY RATE PER 1 MILLION PARTICIPANT DAYS
Kayaking	8.7–2.2
Rafting	4.5–8.7
Trekking	5–15
Skydiving	8.7
Scuba diving	3.1
Alpine skiing	0.57
Driving	152 (1 day of driving is 100 miles)

Adapted from Fiore DC: Injuries associated with whitewater rafting and kayaking. Wilderness Environ Med 14:25–26, 2003.

The body parts most frequently injured during rafting mishaps are the face (33.3%), including the eye (12.1%), mouth (6.6%), other facial parts (5.1%), nose (4.5%), and teeth (4.0%); followed by the knee (15.3%); arm, wrist, or hand (11.6%); and other parts of the leg, hip, or foot (10.5%). The most common injury types are lacerations (32.5%), sprains or strains (23.2%), fractures (14.9%), contusions or bruises (9.8%), and dislocations (8.2%). Most injuries occur in the raft as a result of collisions among passengers, being struck by a paddle or other equipment, or entanglement of extremities in parts of the raft.[52] Because most injuries occur in the raft and involve the face, accident-preventive measures include attaching face protection to paddling helmets and carrying fewer passengers per raft.

In the summer and fall of 2000, a survey was distributed to white-water canoe and kayak paddlers at riverside and through paddle club bulletins, and was posted on the Internet. Three hundred nineteen surveys were returned, reporting 388 acute and chronic injuries. The shoulder, wrist, hand, and elbow were the most common sites of injury. Sprain or strain injuries were the most common (26%), followed by laceration and contusion (17% each). Tendinitis was the most common chronic injury (44%), followed by sprain or strain (27%). *Giardia* infection was reported in 14%.[42] Most injuries occurred while the kayaker was still in the boat (87%). Striking an object was the most common mechanism of injury (44%), followed by traumatic stress and overuse (25% each).[14]

EQUIPMENT

The dynamic and unpredictable nature of rivers can turn any mishap into a tragedy. For this reason, the initial mission of white-water medicine is to emphasize safety and accident prevention.

According to the U.S. Coast Guard's boating accident statistics, the most common factor contributing to white-water-related deaths is failure to wear a personal flotation device (PFD, or life jacket).[49] Exposure to cold river water can stimulate respiratory and cardiovascular reflexes, making it difficult for a swimmer to keep his or her head above water (maintain freeboard).[26] The Coast Guard is charged with regulating and testing life jackets and classifies PFDs into five types. Of these, only two types are commonly used in white-water sports.

The type III PFD, a vest-type jacket favored by most paddlers, permits greater mobility and comfort. The Coast Guard requires that type III PFDs have a minimum of 15.5 lbs (7 kg) of flotation (lift). Because most adults effectively weigh between 10 and 12 lbs (4.5 and 5.4 kg) in the water, this allows at least 3.5 lbs (1.6 kg) of effective required buoyancy.

Type V PFDs are used by commercial outfitters because they provide greater flotation and are constructed asymmetrically, with more than half of the jacket's flotation distributed in the front. This is supposed to turn an unconscious wearer face up. Although this may be true in calm water, it does not work reliably in swift water.

A PFD should fit snugly and not ride up over the head when a person is in the water. Because even a well-fitting life jacket can be pulled off by turbulent water, some manufacturers now include crotch straps as an added safety feature. Testifying before a congressional subcommittee, the president of the National Transportation Safety Association cited the Chilco River accident to support his contention that crotch straps be made mandatory on all white-water use life jackets. Several survivors reported that their life jackets rode up over their heads and did not keep their faces above water.

Life jackets with built-in rescue harnesses, pioneered by the Europeans, are now widely available in the United States. A typical harness system uses seatbelt webbing threaded through a metal retainer, then run into a plastic cam-lock buckle with a toggle. The toggle allows the user to find the buckle in white water. To release the system, the user pulls the toggle, opening the buckle and allowing the webbing to slip through the retainer and release. A D-ring mounted on the back of the jacket provides a point for clipping in a rope (Fig. 39-3). This quick-release belt allows the wearer to attempt a strong swimmer rescue but also to get free of the tethering line quickly in an emergency.

Beyond flotation, life jackets have other benefits that make them highly useful in wilderness settings. Their insulating properties help prevent hypothermia. The closed-cell foam flotation material acts as thoracic padding during falls on slippery rocks or when swimming rapids after exiting the craft. Life jackets also make excellent improvised splints; they can be fashioned into cervical collars, cylindrical knee braces (Fig. 39-4), or padded ankle stirrups.

The American Whitewater Affiliation (AWA) safety code recommends the use of helmets at all times in kayaks and canoes, and in rafts and other craft when attempting rapids of class IV or greater difficulty. Surveys have shown that head

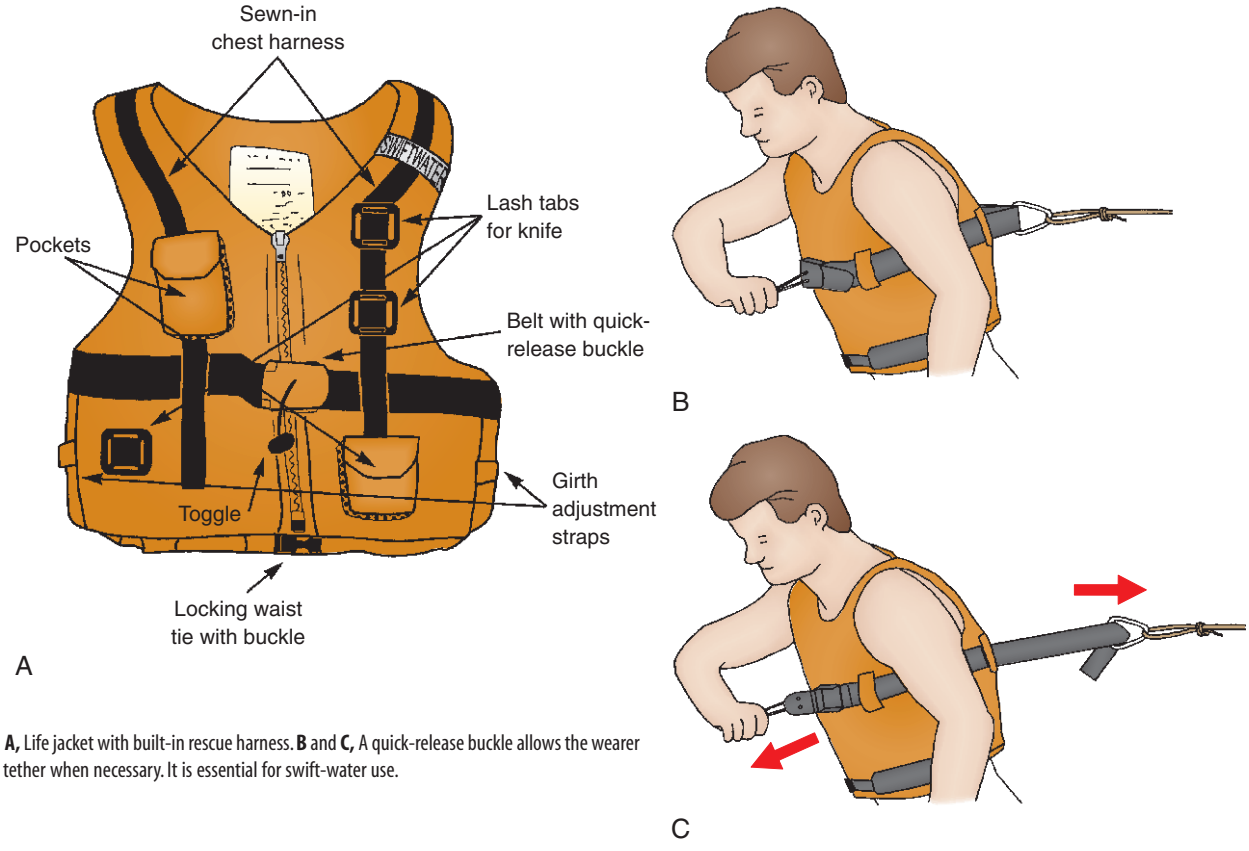


Figure 39-3. **A,** Life jacket with built-in rescue harness. **B** and **C,** A quick-release buckle allows the wearer to release the tether when necessary. It is essential for swift-water use.

Figure 39-4. A paddler wearing a type III life jacket around his knee as an improvised knee immobilizer to help stabilize a sprained knee.

trauma after capsizing constitutes 10% to 17% of all kayaking accidents.[30,50]

Another vital piece of safety equipment is a rope, which should be readily accessible and secured in a manner that facilitates rapid deployment and prevents entanglement. Throw ropes for river use should float, have a certain amount of dynamic stretch, and not absorb water. Self-contained throw bags have virtually replaced coiled ropes for river use and generally hold about 50 to 75 feet (15.2 to 22.9 m) of $^{3}/_{8}$-inch polypropylene rope inside a nylon stuff sack. Newer styles can be attached to life jackets for rapid access. They can also be thrown to rescuers by a paddler who is pinned or broached. Commercial outfitters and large groups of rafters should carry at least one 300-foot (91.4-m) long static rope to be used for Telfar lowers, Tyroleans, and other rescue situations in which mechanical advantages are used.

Knives should be readily accessible. Fixed blades are preferable to folding ones unless the folded blade can be opened easily with one hand. Double-edged blades can cut in two directions and thus require minimal handling in precarious situations. Some modern knives designed for kayakers feature serrated edges that can cut through plastic boats during entrapment.

Whistles should be worn so paddlers can alert others that an accident has occurred. Paddlers are often spread out over the course of a rapid, and yelling over the roar of the water is usually a frustrating and fruitless endeavor.

Placing adequate barriers between the human body and the environment is of paramount importance in aquatic sports. Functional, insulated clothing should be considered a mandatory safety item to prevent hypothermia. Cotton is a poor choice for river wear; it loses all of its insulating properties when wet and dries slowly. Newer synthetics such as polypropylene and polyester pile absorb no more than 1% of their weight in water and maintain thermal insulating qualities when wet.[28] When

Box 39-1. American Version of the International Scale of River Difficulty

CLASS I: EASY
Fast-moving water with riffles and small waves. Few obstructions, all obvious and easily avoided with little training. Risk to swimmers is slight; self-rescue is easy.

CLASS II: NOVICE
Straightforward rapids with wide, clear channels evident without scouting. Occasional maneuvering may be required, but rocks and medium-sized waves are easily avoided by trained paddlers. Swimmers are seldom injured, and group assistance, although helpful, is seldom needed.

CLASS III: INTERMEDIATE
Rapids with moderate, irregular waves that may be difficult to avoid and can swamp an open canoe. Complex maneuvers in fast current and good boat control in tight passages or around ledges are often required; large waves or strainers may be present but are easily avoided. Strong eddies and powerful current effects can be found, particularly on a large-volume river. Scouting is advisable for inexperienced parties. Injuries while swimming are rare; self-rescue is usually easy, but group assistance may be required to avoid long swims.

CLASS IV: ADVANCED
Intense and powerful but predictable rapids requiring precise boat handling in turbulent water. The advanced river may feature large, unavoidable waves and holes or constricted passages that demand fast maneuvers under pressure. A fast, reliable eddy turn may be needed to initiate maneuvers, scout rapids, or rest. Rapids may require "must" moves above dangerous hazards. Scouting is necessary the first time down. Risk for injury to swimmers is moderate to high, and water conditions may make self-rescue difficult. Group assistance for rescue is often essential but requires practiced skills. A strong Eskimo roll is highly recommended.

CLASS V: EXPERT
Extremely long, obstructed, or violent rapids that expose a paddler to above-average danger. Drops may contain large, unavoidable waves and holes or steep, congested chutes with complex, demanding routes. Rapids may continue for long distances between pools, demanding a high level of fitness. Eddies may be small, turbulent, or difficult to reach. At the high end of the scale, several of these factors may be combined. Scouting is mandatory but often difficult. Swims are dangerous and rescue is difficult, even for experts. A very reliable Eskimo roll, proper equipment, extensive experience, and practiced rescue skills are essential for survival.

CLASS VI: EXTREME
Class VI runs exemplify the extremes of difficulty, unpredictability, and danger. The consequences of errors are severe, and rescue may be impossible. For teams of experts only, at favorable water levels, after close inspection and taking all precautions. This class does not represent drops believed to be unrunnable but may include rapids that are only occasionally run.

From Safety Code of the American Whitewater Affiliation, Phoenicia, NY, American Whitewater Affiliation, 1989.

combined with a nylon or Gore-Tex paddling jacket, a synthetic underlayer provides effective protection from cold and wind.

Wet suits, previously considered to be optimal garments for paddlers in extreme conditions, are stiff and somewhat constricting.[1] The dry suit, with tight-fitting latex seals at the wrist, ankle, and neck, is the new gold standard for cold-water boating. By sealing water out and preventing evaporative heat loss, the dry suit can keep a paddler warm even during winter conditions.[18]

Overheating is occasionally a problem with dry suits. Recently, a dry suit contributed to profound and unexpected hyperthermia in a kayaker who had suffered a submersion injury in cold water.[4]

RIVER HAZARDS

The International Scale of River Difficulty grades rivers and rapids into classes I to VI. An American version of this rating has been adopted by the AWA for most U.S. rivers[48] (Box 39-1). Some western rivers use the Grand Canyon System, which rates rapids on a scale from 1 to 10. Neither scale is a truly objective standard; individual and regional variations are common, and the margin of difficulty for a particular rapid may differ significantly for kayaks and rafts. Unfortunately, important safety parameters, such as water temperature, remoteness, and evacuation potential, are not taken into consideration.

The difficulty of a river generally increases with the volume of flow and the average gradient. The volume of water in a river is usually expressed as a measure of cubic feet per second (cfs). This is the amount of water moving past a certain point during a given period of time. The volume of a river can be determined by multiplying the width by the depth by the speed of the current. For example, a channel 10 feet deep and 20 feet wide moving at a velocity of 5 feet per second (fps) equals a volume of 1000 cfs. As the water level rises, its speed and power increase exponentially, raising the difficulty of most rapids.[3] When the speed of the current is doubled, the force of the water against an object in the current is quadrupled; that is, the force of the current increases as the square of its speed. Occasionally, however, a rapid becomes easier as the added water submerges hazardous obstacles. Gradient is the amount of drop between two points and is expressed as feet per mile. The steeper the gradient, the faster the water moves.

Not all water flows downstream. The most common upstream flow is an eddy, which is created when water flows around an obstacle. The water piles up higher than the river level on the upstream side of the obstacle, whereas the water on the downstream side is lower. Water flows around the obstacle then back toward it to fill in the low spot. The line between

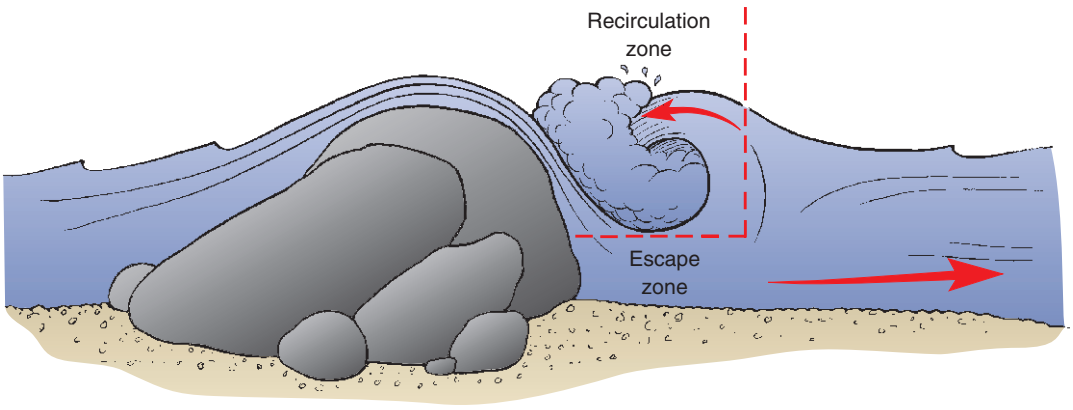


Figure 39-5. Recirculating currents created by a hydraulic. Water and "swimmers" are released downstream beneath the surface.

the upstream and downstream current is the eddy line. Eddies are one of the most important features of the river for boat maneuvering and rescue. Exiting the main current by pulling into an eddy allows the paddler to stop the descent and safely scout the next rapid. It also provides a location for a paddler to set up rescue for his or her companion upstream.

Hydraulics, also known as holes, reversals, rollers, suckholes, and pour-overs, are the most common hazards in rivers. A hydraulic is created when water flows over an obstacle, causing a depression that produces a relative vacuum within which the downstream water recirculates (Fig. 39-5). The water below a hydraulic is typically very aerated and presents a white, foamy appearance. Rafts and kayaks can be turned upside down by the force of a hydraulic, and if the reversal currents are strong enough, crafts and people can become trapped in the recirculating flow. When proceeding into a rapid that contains a hazardous hydraulic, one of the group should preset a rope below the hole to facilitate rescue.

Hydraulics release water downstream from beneath the surface. This may be the only avenue of escape for a swimmer. Escape from a strong hydraulic may require a person to stay submerged and to resist the urge to return immediately to the surface. Surfacing too early can result in recirculation. Fortunately, most hydraulics eventually release people regardless of what action they take.

Novice paddlers often misjudge the force of hydraulics. It is not the height of the drop that generates the recirculating power but rather the shape and angle of the obstruction, combined with water volume and adjacent eddy currents. A "smiling" hydraulic has its outer edges curving downstream, so that the recirculating water feeds out into the main current and is thus easier to escape. In a "frowning" hydraulic, the outer edges curve back upstream into the center of the hydraulic, making escape much more difficult.

Low-head dams or weirs form massive hydraulics with enormous recirculating potential. Unlike natural hydraulics, these human-made structures form hydraulics all the way across the river, leaving no escape routes. In the Binghamton Dam disaster of 1975, a 13.5-foot Boston whaler with a 20-horsepower engine was pulled into a hydraulic while attempting a rescue, resulting in the deaths of three firefighters.[45]

Undercut rocks are boulders or ledges that have been eroded just beneath the water surface. These usually occur on geologically older rivers. They can be difficult to recognize and pose significant risks for entrapment and drowning, even in class II rapids.

The potential for entrapment can also occur when swimmers attempt to stand up and walk in swift-moving currents. A foot can become wedged in an undercut rock or between rocks beneath the surface, causing the victim to lose his or her balance and fall face down into the river (Fig. 39-6A). With the foot entrapped, the victim cannot regain an upright or even face-up position. This type of mishap has caused drownings in water less than 3 feet deep.

A swimmer in a rapid should assume a supine position, with feet at the surface and pointed downstream to serve as shock absorbers. This position minimizes the potential for both foot entrapment and head and neck trauma (see Fig. 39-6B).

Strainers are obstacles, such as fallen trees, bridge debris, or driftwood, lodged between rocks or jutting out from the shore, that allow water to pass through (sieve effect) while trapping the swimmer or boater. Flooded rivers, a favorite of expert boaters, often develop many new strainers as riverbank debris is washed into the flow. In the summer of 1987, five paddlers drowned when their raft struck a large strainer on Canada's Ellaho River.[47]

Negotiating a strainer requires special tactics. The safest option for the swimmer is to swim aggressively into the strainer head first rather than feet first, and then attempt to climb over the debris (Fig. 39-7A). Approaching a strainer feet first may lead to underwater entrapment (see Fig. 39-7B).

Human-made hazards can also pose a threat to river runners. Bridge pilings, submerged automobiles, dams, and low-hanging power lines can pin or injure boaters.

A broach occurs when a boat wraps sideways around an obstacle or when both bow and stern become stuck on separate obstacles simultaneously. Common obstacles include boulders, trees, bridge pilings, and ledges protruding from canyon walls. Drowning can occur if the paddler leans upstream away from the obstacle and flips upside down while still broached or if the boat collapses and entraps the victim (Fig. 39-8).

A vertical pin happens when a kayaker plunges over a drop and the end of the boat becomes trapped between rocks beneath the surface. The force of the water can fold a plastic kayak over on itself, trapping the occupant upside down beneath the surface (Fig. 39-9).

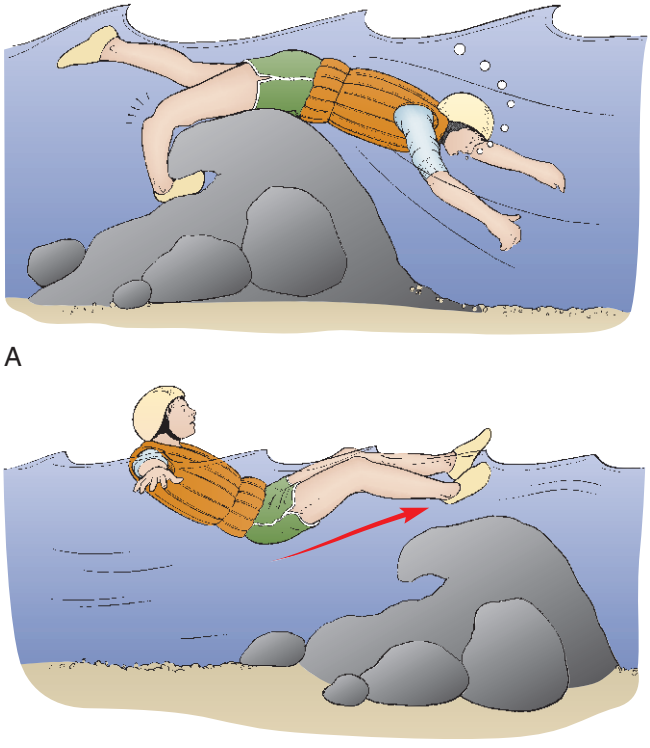


Figure 39-6. **A,** Attempting to stand up in shallow water can produce foot entrapment in an undercut rock. **B,** Proper way to swim while in a rapid.

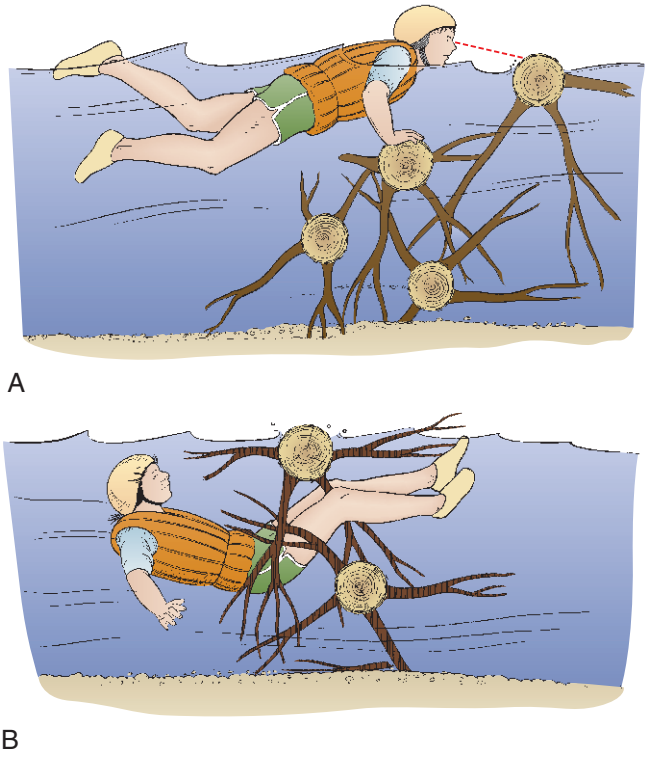


Figure 39-7. **A,** Proper approach to a strainer. **B,** Incorrect approach to a strainer.

A survey of 365 members of the AWA revealed that 33% of serious kayaking incidents and 41% of open canoeing mishaps involved either pinning or broaching[50] (Table 39-3). In a separate survey of 500 paddlers between 1989 and 1993, 42% of kayaking fatalities resulted from vertical pins, broaches, or entrapments in strainers.

Kayak construction can have important safety implications in both broach and pin situations. The force of the current against the deck of the boat or back of the paddler can make it impossible for the victim to extract his or her legs and escape. Boat makers have developed kayaks with larger cockpits that make it easier to raise the knees out and escape the craft. Transverse bulkhead-type foot braces have replaced pedal-type braces to prevent the kayaker from being shoved forward in the boat. This feature ensures the escape potential offered by larger cockpits. One of the compromises of the larger cockpit, however, is that the spray skirt is more likely to come off in turbulent water.

SUBMERSION ACCIDENTS

Almost all fatalities on rivers result from submersion. Each year, the River Safety Task Force of the American Canoe Association compiles accounts of drownings and other accidents. Every 3 years, it publishes the River Safety Report, which chronicles and analyzes these accidents.[45–47,49]

Most submersion fatalities occur after paddlers unexpectedly swim from their boats or become trapped in them underwater.

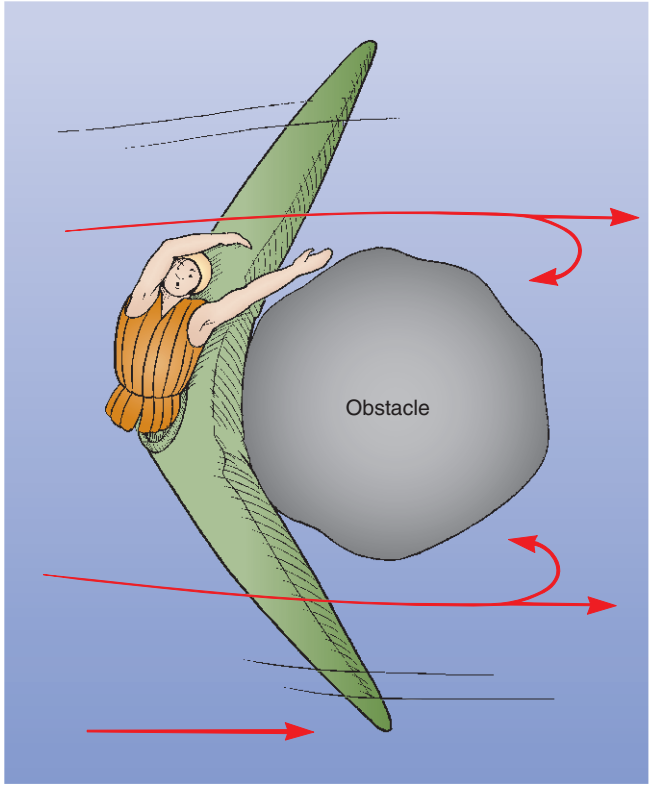


Figure 39-8. Broach.

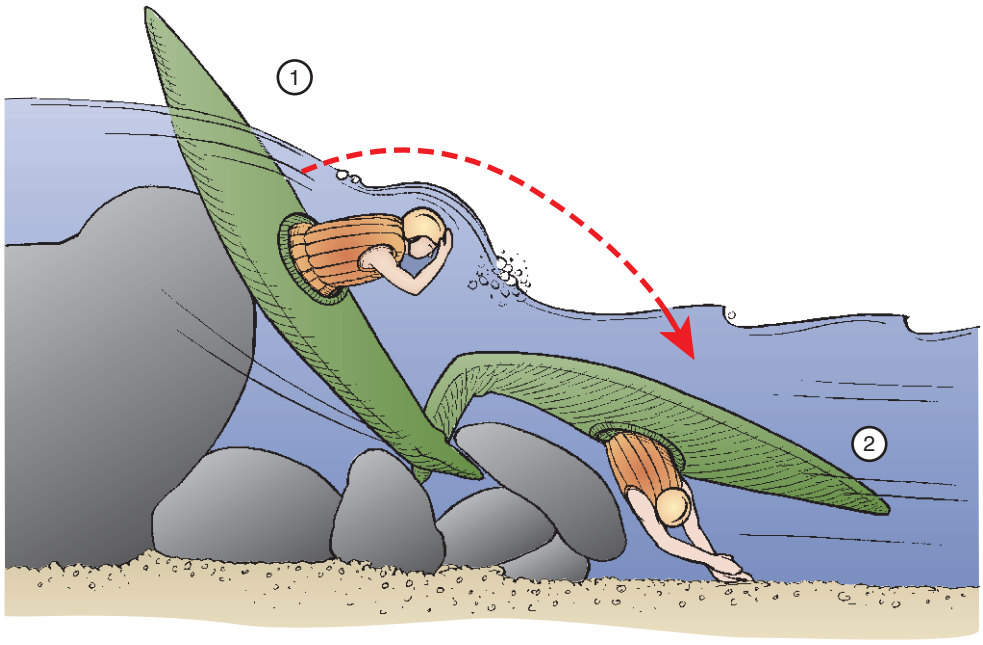


Figure 39-9. **1,** Vertical pin. **2,** Pitchpole pin.

TABLE 39-3. Serious White-Water–Related Incidents

INCIDENT TYPE	NO. OF INCIDENTS	PERCENTAGE OF ACCIDENTS
Vertical pin entrapment	18	8
Broach entrapment	46	21
Rock sieve entrapment	16	7
Undercut rock entrapment	23	10
Recirculation in hydraulic	47	21
Long swim	42	19

From Wallace D: Scary numbers and statistics—results of AWA close calls and serious injuries survey. American Whitewater: Journal of the American Whitewater Affiliation 37:27–29, 1992.

The exact cause of drowning often remains unclear and is inexplicably blamed on immersion hypothermia. Although hypothermia induces impaired judgment and coordination and may be an important contributing factor, immersion hypothermia is probably never the sole cause of death.[52] Studies by Hayward and others have shown that seminude subjects are able to maintain normal core temperatures for 15 to 20 minutes in 10° C (50° F) water.[20,21] Continuous immersion for up to 1 hour would be required to produce profound hypothermia.[21]

Cold-water immersion precipitates drowning by three other mechanisms. Sudden cold-water immersion produces profound cardiovascular and respiratory responses. Reflex sympathetic output can markedly increase blood pressure and heart rate, resulting in lethal arrhythmias.[17,26,27]

An immediate and involuntary gasp occurs after cold-water immersion. This is followed by hyperventilation.[8] Pulmonary ventilation increases up to fivefold because of increased tidal volume and respiratory rate.[44] The initial gasp can result in aspiration of water and laryngospasm. Hyperventilation produces respiratory alkalosis with resultant muscle tetany and cerebral hypoperfusion.[8] This response can increase the risk for drowning in a person struggling to maintain an airway freeboard in rough water.

The respiratory stimulation produced by cold-water immersion significantly decreases breath-holding duration.[22] This fact has enormous implications for kayakers, who must hold their breath while attempting to roll up a boat after flipping upside down. This probably accounts for the unexplained swims by expert kayakers who sometimes fail to right themselves after flipping in cold water. I have found that the respiratory response to cold water can be mitigated somewhat by prior acclimatization. This is accomplished by repeatedly splashing cold water on your face for about 1 minute before entering the rapid.

Peripheral cold-water–induced vasoconstriction exacerbates rapid cooling of muscles and nerves in the extremities, resulting in loss of strength and coordination.[44] The ability to swim, maintain freeboard, avoid obstacles, and climb from the river may be greatly impaired.[29] Even when the air temperature is warm, paddlers running cold water rivers should wear sufficiently insulated clothing.

The combination of hyperventilation and muscle dysfunction can be lethal for a swimmer in rough water. A PFD helps, but does not prevent even small waves from submerging a swimmer's head.[16] These dangers make imperative the need to preset safety systems in significant rapids and to rescue swimmers first. Unfortunately, paddlers have drowned when their companions chased after equipment, assuming that the swimmer could climb out of the river without assistance.[46,48]

Safety kayaks with enhanced buoyancy are recommended on commercial raft trips because they provide additional flotation for clients who fall overboard.

Although some maintain that the respiratory and cardiovascular reflexes can be abolished by repeated exposure of the face to cold water, there are currently no scientific data to support this theory of acclimatization.

TRAUMA

A survey of commercial raft clients revealed that the most common significant injury was a sprained or fractured ankle.[51] Ironically, these injuries usually occur out of the water when walking on loose, wet, and slippery rocks during scouting and portaging or when entering or leaving the river. Ankle and other lower-extremity injuries also occur on the river when rafters are tossed onto each other in rapids.

Kayakers are prone to ankle injuries from forced dorsiflexion or inversion when the bow of the boat hits an obstruction. The feet are held against the narrow horizontal braces while the heels are pushed underneath, or the entire ankle is inverted. European- and some American-designed kayaks with bulkhead foot braces have reduced this problem.

Management of foot and ankle injuries should begin with ice, elevation, and compression to reduce swelling. Cold river water is usually substituted for ice. Compression wrapping is important after icing to prevent swelling from reflex vasodilation. Splinting is important to reduce pain and edema and to limit exacerbation of the injury during evacuation. Pneumatic splints, still carried by many raft companies, provide adequate support and compression but are prone to overinflation when heated by the sun. Zippers often malfunction when they rust or jam with sand. Neurovascular integrity must be checked frequently with an air splint. Ankle splints can be improvised from life jackets, kayak float bags, articles of clothing, or a SAM splint (SAM Medical Products, Newport, OR).

Strains are common in white-water sports. Researchers at Dalhousie University in Halifax, Nova Scotia analyzed dynamic electromyographic potentials of the various muscle groups used in kayaking and then correlated them with videotaped sequences.[34] Muscles used most often in kayaking that are prone to strain injury are shoulder extensors (latissimus dorsi, teres major, pectoralis major), medial scapula rotators (rhomboideus major and minor, pectoralis minor), lateral scapula rotators (pectoralis minor, serratus anterior), shoulder flexors and horizontal adductors (anterior deltoideus, pectoralis major, coracobrachialis), elbow extensors (triceps), and spine erector muscles. Any training program for kayakers needs to emphasize conditioning of these muscle groups.

Back strain afflicts rafters, kayakers, and canoeists. Rafters are prone to back injuries while portaging, pushing stuck rafts off rocks, and carrying the crafts to and from the river. Raft guides are notorious for suffering back strain when pulling capsized customers, who often weigh more than they do, back into the rafts. Kayakers and canoeists injure their backs lifting water-laden boats and loading their crafts onto automobile roofs. Sitting for prolonged periods with legs extended and minimal back support leads to muscle fatigue in kayakers, compounding the potential for injury.

Repetitive dorsiflexion of the wrist required to operate an offset (feathered) kayak paddle produces tendonitis and synovitis.[32] A paddle constructed with a 75- to 80-degree offset instead of the traditional 90 degrees can reduce wrist stress. Aspirin or nonsteroidal anti-inflammatory agents ingested 30 minutes before paddling, combined with ice application afterward, may be beneficial. Wrist supports provide limited relief.

The injury most often associated with kayaking is anterior shoulder dislocation. Various surveys have placed its incidence in kayakers at 10% to 16%, making it the second most common white-water–related injury[5,31,50,51] (Table 39-4). The maneuver most notorious for precipitating this injury is the high brace. Often used while supporting the kayaker in a hydraulic, surfing on a wave, or rolling the kayak upright after a flip, the high brace entails abduction of the humerus, with external rotation of the glenohumeral joint (Fig. 39-10). If the arm becomes extended behind the midline plane of the body by the force of the current, the triad of abduction, external rotation, and extension of the shoulder can stretch or rupture the glenoid labrum and capsule, resulting in anterior subluxation or dislocation.[43] The paddle acts as a lever to increase the force on the glenohumeral joint.

To minimize the risk for shoulder dislocation, the preferred method of bracing is the "low brace," in which the arm is held in internal rotation and close to the body (adduction). Although initially awkward for the novice paddler, this bracing maneuver is inherently stronger and more versatile because it allows backpaddling out of a hydraulic. Exercises that strengthen the rotator cuff and deltoideus, triceps, and pectoralis muscles reinforce the glenohumeral joint.

Figure 39-10. High brace maneuver.

TABLE 39-4. Common White-Water–Related Injuries, 1980–1991 (N = 85)

INJURY TYPE	NO. OF INJURIES	PERCENTAGE OF INJURIES
Shoulder dislocation	14	16.5
Near drowning	11	12.9
Fractures	15	17.6
Head and neck	6	7.0
Hypothermia	4	4.7
Leg injuries	11	12.9
Lacerations	9	10.5
Fatalities	7	8.2

From Wallace D: Scary numbers and statistics—results of AWA close calls and serious injuries survey. American Whitewater: Journal of the American Whitewater Affiliation 37:27–29, 1992.

The paddler with a dislocation is usually aware that something has gone wrong and holds the extremity away from the body, unable to bring the arm across the chest.[43] The shoulder may appear square because of anterior, medial, and inferior displacement of the humeral head into a subcoracoid position.

Although on-scene reduction of shoulder dislocations is controversial, immediate relief of pain, curtailment of ongoing injury, and subsequent ability to function more actively in evacuation are strong reasons to do it. Several techniques have been advocated for reduction.[40] The key element is rapid initiation because the longer a shoulder remains dislocated, the more difficult the eventual reduction becomes. Relocation is often delayed because river corridors rarely afford rapid access to a flat and comfortable area upon which to place a victim in the supine or prone position, a requirement for most techniques.

For river and other wilderness settings, reduction is facilitated by using a technique in which the victim is standing or sitting (Fig. 39-11). As soon as the diagnosis is made, the victim bends forward at the waist while the rescuer supports the chest with one hand. With the other hand, the rescuer grabs the victim's wrist and applies steady downward traction and external rotation. While maintaining traction, the rescuer can slowly flex the shoulder by moving it in a cephalad direction until reduction is obtained. If two rescuers are available, one should support the victim at the chest while the other pulls countertraction and flexion at the arm. Scapular manipulation by adducting the inferior tip using thumb pressure and stabilizing the superior aspect of the scapula with the cephalad hand may augment reduction[35,40] (Fig. 39-12).

Shoulder reduction can also be done while the victim is sitting. Grab the victim's forearm close to his or her elbow with both hands, and with the elbow bent at 90 degrees, pull steady downward traction on the arm. After about a minute of sustained traction, slowly raise the entire arm upward until reduction is complete. Gingerly rotating the forearm outward while pulling traction may facilitate reduction. If a second rescuer is present, scapular manipulation can be performed simultaneously as described earlier.

Another relocation technique uses the victim's life jacket to allow one rescuer to apply both controlled traction and countertraction.[11] This technique requires that the victim be supine, with room for the rescuer to sit adjacent to the dislocated shoulder. The rescuer then slides his or her foot and leg through the life jacket's arm opening, under the neck, and out through the jacket's head opening. The rescuer's leg functions as a head rest, whereas the foot braced against the opposite shoulder strap of the life jacket provides countertraction. Holding the forearm of the affected side with the elbow bent at 90 degrees, the rescuer slowly leans back to apply traction while the leg exerts countertraction. The life jacket allows countertraction force to be distributed across the victim's chest. External and internal rotation can be applied to the humerus during traction to facilitate reduction (Fig. 39-13).

One should always monitor circulation and motor-sensory function to the wrist and hand before and after attempting a shoulder reduction. To prevent a recurrent dislocation, the kayaker's arm should be splinted across the chest with a sling or swath or by safety pinning the sleeve of the arm across the chest. If circumstances preclude exiting the river without further kayaking, the shoulder can be partially stabilized by wrapping an elastic or neoprene wrap around the torso and involved arm to limit abduction and external rotation (Fig. 39-14).

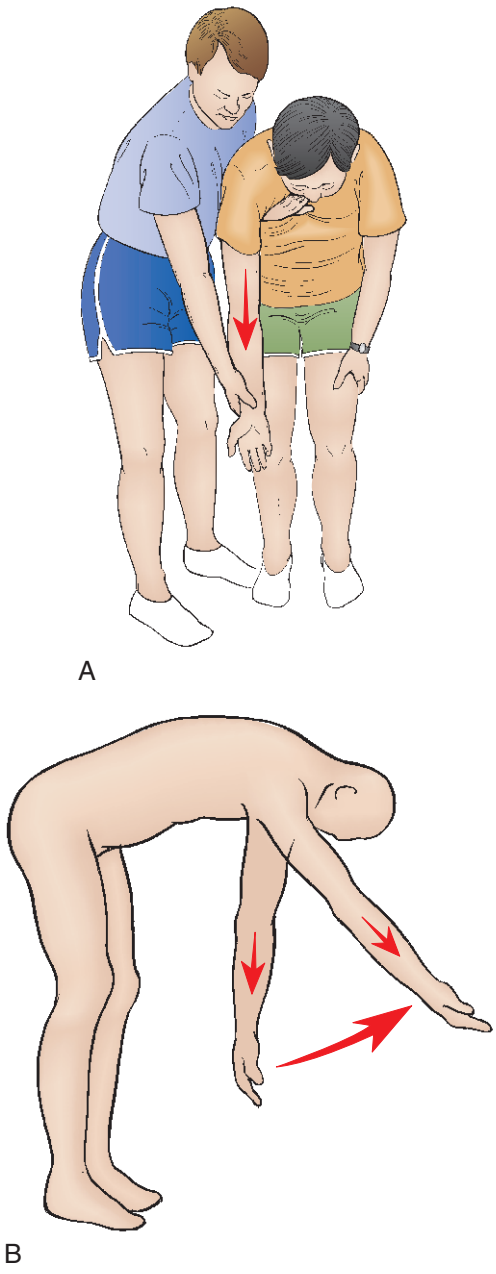


Figure 39-11. Weiss technique for shoulder relocation with the victim standing. **A,** The rescuer supports the victim's chest with one hand and pulls down and forward (**B**) with the other hand.

Head, facial, and dental trauma are more common in kayakers and decked canoeists than in rafters because of the potential for flipping upside down while still in the craft. Minor abrasions, lacerations, and contusions are common; serious head injury with loss of consciousness is rare. Head and facial trauma can be minimized by wearing a protective helmet and tucking forward, instead of leaning backward, while rolling.

Spine fractures have been reported in kayakers and canoers.[46,47,49] Cervical spine injuries have occurred in kayakers

Figure 39-12. If two rescuers are available, scapular rotation to assist shoulder relocation can be performed while the second rescuer pulls the arm down and forward. The inferior top of the scapula is pushed medially.

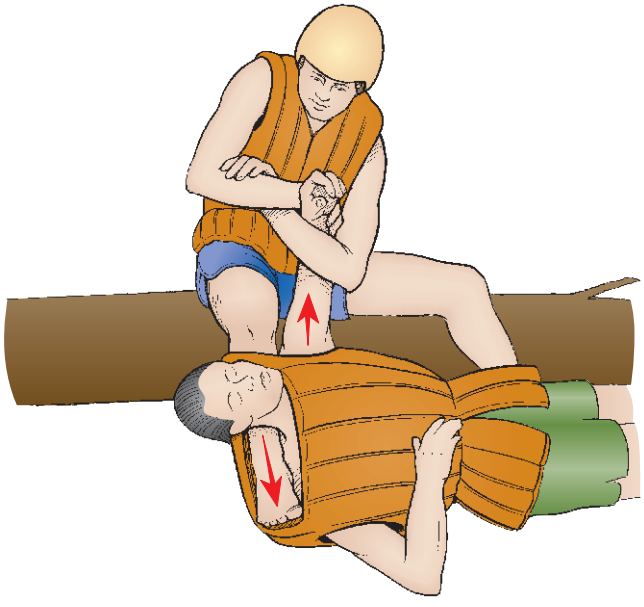


Figure 39-13. Using a life jacket to assist in countertraction for shoulder relocation.

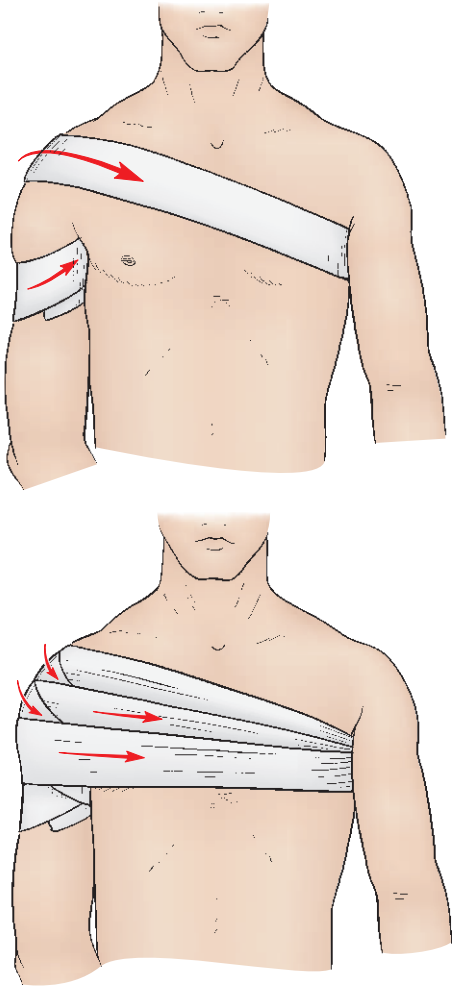


Figure 39-14. Shoulder harness for support after shoulder dislocation.

in conjunction with head trauma sustained after flipping upside down. Vertical compression fractures of the thoracolumbar spine have occurred from axial loading when a kayaker landed flat after paddling over a waterfall. One kayaker was rendered paraplegic after landing on his back on rocks while attempting to negotiate a waterfall.[49] Fortunately, his companions recognized the injury and kept him supported on minicell blocks from their kayaks until a backboard could be obtained.

Significant visceral and musculoskeletal injury can occur when a swimmer is sandwiched between a downstream boulder or obstruction and the upstream craft that has been exited. Swimmers should always stay upstream of their craft.

Many kayakers suffer abrasions and contusions to the fingers and knuckles while hanging upside down after flipping. Oar frames, oars, paddles, and the metal ammunition boxes used to keep supplies dry can all inflict injury when rafts are capsized or paddlers are tossed about in turbulent water.

Blisters on the hands are a frequently reported problem in paddling surveys.[51] Kayakers develop them at the metacarpophalangeal (MCP) joint of the thumb along the ulnar aspect.

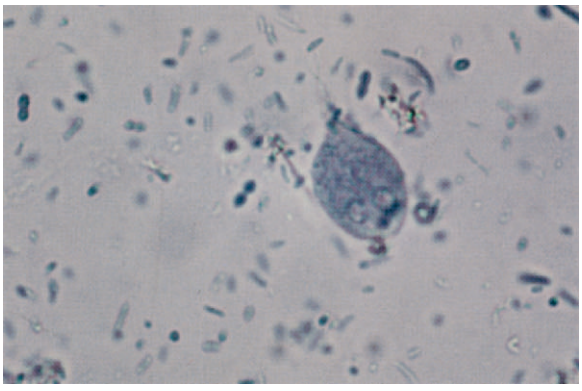


Figure 39-15. *Giardia lamblia* trophozoite seen by methylene blue wet mount staining under oil (1000×). The finding of cysts or trophozoites in a patient with diarrhea is sufficient to make a tentative diagnosis of giardiasis.

Common sites of blister formation in rafters and canoeists are the proximal palmar surfaces of the MCP joints. Taping and moleskin application reduce the incidence of this potentially incapacitating problem.

INFECTIONS

Blisters, abrasions, and lacerations are always at increased risk for infection in an aquatic environment. Maceration from prolonged immersion in water and exposure to atypical pathogens are contributing factors.

An outbreak of *Staphylococcus aureus* skin infections among raft guides in Georgia and South Carolina nearly led to the demise of two rafting companies.[10] Sharp grommets on the thwarts of the rafts had caused repeated lower extremity abrasions. The causative organism was cultured from rafts up to 48 hours after use. Daily raft disinfection enabled the companies to remain in operation.

Otitis externa (swimmer's ear) is a common problem among paddlers. Water exposure to the ear canal macerates the epithelium and elevates the normally acidic pH of the canal, predisposing the ear to infection.[12] The bacteria most commonly cultured are *Pseudomonas aeruginosa, Proteus vulgaris,* and *Staphylococcus* species.[12,24] Antibiotic eardrops with or without hydrocortisone are widely available and very useful. Irrigation of the canal with commercially available solutions containing acetic acid and alcohol helps prevent infection by lowering the pH and drying the canal.[24] The drops should be applied after each outing (see Chapter 75).

Recent publicity given to water contamination by *Giardia lamblia* has been reinforced by statistics from the Centers for Disease Control and Prevention, which report *Giardia* organisms to be the most common pathogenic intestinal parasite in the U.S. (see Chapters 61 and 62). *Giardia* cysts abound in mountain streams and rivers once considered to be sources of pristine water (Fig. 39-15). They persist in very cold water and have no detectable taste or smell. Rivers are contaminated by animals that defecate in or near the water. Studies by the Wild Animal Disease Center at Colorado State University, Ft. Collins, Colorado have identified more than 30 animal species as *Giardia* carriers.

Paddlers who travel to foreign countries should seek information on local endemic diseases and relevant prophylactic measures. White-water rafting and kayaking in Third World countries subject paddlers to unusual aquatic-related infections. This is exemplified by a report of schistosomiasis in rafters returning from Ethiopia.[23] Schistosomiasis is endemic in large areas of Africa, South America, and the Caribbean and is transmitted to humans who swim or come into contact with fresh water containing the larval stage. Paddlers who return from endemic regions should be screened with serologic testing because up to 50% of infections are asymptomatic.[32]

Malaria has been reported in rafters returning from New Guinea, and both leptospirosis and hepatitis have stricken kayakers venturing to Costa Rica.[2] In the United States, pulmonary blastomycosis was reported among canoeists in Wisconsin.[7]

ENVIRONMENTAL HAZARDS

Although hypothermia is rarely the cause of death among white-water paddlers, hypothermia-induced impairment of judgment and coordination is a significant contributing factor in many fatalities and accidents.[19,29,45,46,47,54] The paddling season usually begins in early spring when air temperatures are cool and snow melt–swollen rivers run extremely cold. Paddlers with rusty skills are more prone to frequent swims and the effects of cold-water immersion. Many rivers, especially in the western United States, are controlled by dams that release water from far beneath the surface and thus remain cold year round. Placing adequate barriers between the human body and the environment and carrying adequate food and waterproof matches are of paramount importance.

Another common environmental affliction suffered by paddlers is rhus dermatitis from poison oak or poison ivy. Most cases occur during spring paddling when the vines are potent but the characteristic leaves have not yet appeared. Barrier creams such as StokoGard Outdoor Cream and Tecnu Ivy Shield can be used by individuals highly sensitive to the plants. After plant contact occurs, the oil may be removed from the skin by washing within 30 minutes.[13] A commercial product, Tecnu Oak and Ivy Cleanser, can remove oil from the skin for up to 8 hours after exposure. Any solvent may help remove some of the urushiol oil from the skin. Gasoline, paint thinner, acetone, and rubbing alcohol have all been reported by paddlers to be effective. Unfortunately, these products can also be irritating to the skin or frankly toxic.

Treatment of rhus dermatitis consists of oral antihistamines and systemic corticosteroids. A 2-week treatment course is needed to prevent recurrence of the rash[13] (see Chapter 57).

Sunburn and the effects of chronic exposure to solar radiation are compounded by water's ability to reflect up to 100% of ultraviolet (UV) radiation, depending on the time of day. Sand can reflect up to 17% of harmful UV rays. Most rivers are situated in mountains, where UV rays increase 4% to 5% with each additional 305 meters (1000 feet) of altitude.[9] Sunscreens must be applied frequently because they are prone to wash off in the water. Zinc oxide and other barrier creams are more resistant to water and are preferable on areas of intense exposure, such as the nose and lips. Paddlers with fair skin should consider using gloves to protect the hands from UV exposure.

Eye protection from UV radiation is often overlooked or avoided by paddlers because sunglasses frequently fog while on the river. Application of Dawn dishwashing soap to the lenses prevents fogging for up to 30 minutes. Polarizing lenses reduce glare off the water, but the polarizing feature does not in and of itself filter UV and infrared radiation.

Venomous snakes, especially pit vipers, along with scorpions, spiders, and fire ants, are frequently encountered by river enthusiasts and should be considered potential hazards. Paddlers should know appropriate first-aid measures for envenomations.

Paddlers commonly consume wild foliage, which may produce severe illness. In one published report, six rafters were poisoned and one of them died after eating water hemlock, *Cicuta douglasii*.[32]

SWIFT-WATER RESCUE

Time is the most important factor in river rescue and often precludes the use of technical rope-based systems. Experience and an understanding of river dynamics are essential.

The most common rescue scenario involves a swimmer who has exited the craft. The victim may be moving downstream at 5 to 10 mph in the middle of a large river. Because the dynamic nature of swift water does not often allow time for a shore-based rescue system to be established, many white-water rescues are made from a raft, canoe, or kayak. Rafts should stay close together in rapids to render mutual aid. Throw bags can be used directly from the raft to rescue swimmers, or the victim may often be reached with an outstretched arm and a paddle. A swimmer should be pulled back into the raft by grabbing the shoulder straps on his or her life jacket and then leaning backward into the raft to pull the person in. The swimmer can assist by pulling up on the frame, D-ring, or hand line as he or she is being pulled in.

Kayaks can be used to rescue swimmers in midcurrent. The kayak is also an excellent platform to provide additional flotation for a swimmer who is trying to maintain freeboard in rough water. The most common method of rescuing swimmers with kayaks is to have them grab the bow or stern "grab loop" of the boat and then tow them to safety. The loop is usually sized so that it is easy to grab, yet will not admit an adult-sized hand. The swimmer can also grab onto the back of the cockpit rim and pull his or her torso onto the back deck. This gets the swimmer out of the cold water and reduces the likelihood of injury from rocks.

"Boogie boards" originally developed for use in the ocean surf have been modified for rescue use on rivers. Rescue boards are larger and come with two sets of handles—one for the rescuer and one for the victim. The boards add a substantial amount of flotation to a rescue swimmer and, when used with swim fins, can provide a maneuverable platform for reaching and picking up a victim.

The latest craft to be adapted to swift-water rescue is the personal watercraft, or Jet Ski. Introduced in 1987, these machines have become increasingly popular with professional rescue agencies. Because they lack an exposed propeller, personal watercraft are safe for swimmers and can negotiate shallow rivers. They can be maneuvered upstream in rapids, and turn within a short radius. Newer versions, adapted for rescue, can tow a backboard or litter device behind them and are quite stable.

Rescue from entrapment requires a higher level of skill and often presents greater potential risk to the rescuer. The method used depends on whether the victim can maintain adequate freeboard. If the entrapment site is accessible, direct contact with the victim is quickest and most effective. A rescuer may wade to the entrapment site or reach it by boat if there is a stable site to exit the craft.

Wading Rescues

There are two significant risks involved in wading through swift water: foot entrapment and losing balance and thus swimming through a downstream rapid. Before wading, the river downstream should be scouted for hazards, and if possible, a rope thrower should be stationed downstream in the event the rescuer loses his footing.

There are several techniques that help to stabilize balance and prevent foot entrapment or the need to swim during wading: (1) Using a paddle for support affords three-point stability and allows rescuers to wade into deeper water. Start by facing upstream, with legs slightly wider than shoulder width. Reach out, turn the paddle blade parallel to the current, and plunge it into the water. Just before the blade hits bottom, turn it sideways to the current. The force of the on-rushing water will pin it to the bottom. The paddle and your two legs form a tripod, which is more stable than your legs alone. Move slowly across the current, facing upstream, moving only one of these three points at a time. As the water deepens, you will have to lean more and more into the current. Although a pole, stick, or tree branch will work, paddle blades are better because they stay against the bottom better when used correctly. (2) A group working together as a team can wade into water far deeper and more securely than can a single individual. Group members support one another, and by standing together, also create eddies that provide protection from the onrushing current for each other. If there are two rescuers, they should face and grab each other by the shoulders of their life jackets, then wade across the current, facing either up and downstream or sideways to the current. One person moves at a time; the upstream person goes first, then the downstream person steps into the eddy created by the upstream individual. The two-person method can also be expanded for more people, with a resultant increase in stability.

A strong swimmer rescue is the next quickest method, but entails significant risk to the rescuer (Fig. 39-16). The rescuer is tethered to a rope that provides added stability against the force of the current. If a quick-release harness is not available, a loose loop of rope can be passed under the rescuer's armpits.

A tag line rescue should be considered if the victim cannot be reached directly. A tag line is a rope stretched across the river downstream that is then brought upstream to the victim (Fig. 39-17). Getting the line across the river sometimes constitutes an insurmountable obstacle. If the river is narrow, it may be possible to throw the line across. Otherwise, it can be ferried across by a boat or team of swimmers. During a ferry, as much of the rope as possible should be kept out of the water to avoid drag.

There are two types of tag lines (Fig. 39-18). A floating tag line has a life jacket or some other flotation device attached to the middle to keep the rope on the surface, which helps support the victim. A snag tag is a weighted line submerged and walked upstream to snare a foot or other body part that has been

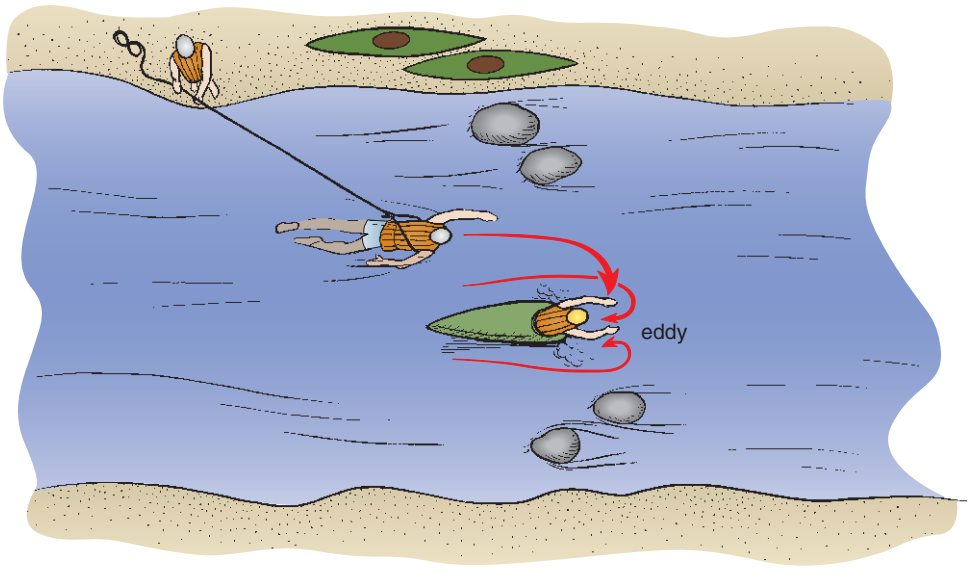


Figure 39-16. Strong swimmer rescue.

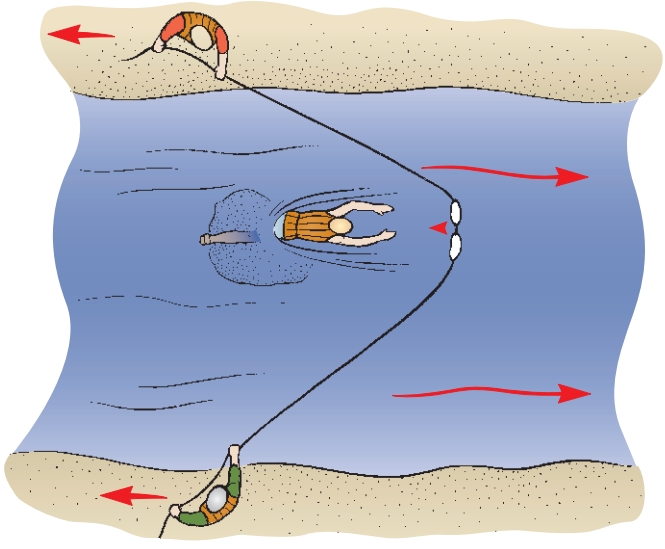


Figure 39-17. Tag line.

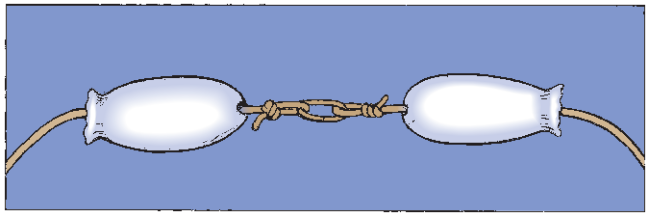


Figure 39-18. Two throw bags connected with a carabiner to make a tag line.

trapped under the surface. A snag tag can be made by joining together two throw bags filled with rocks (Fig. 39-19).

APPENDIX A: WHITE-WATER FIRST-AID KITS

The following variables should be considered when designing a white-water first-aid kit: remoteness and accessibility of the river, difficulty of travel conditions, number of people the kit will need to support, preexisting medical conditions, and space and weight restrictions. When assembling a kit, the following components are generally recommended for rafting and kayaking:

Rafting Kit

- Waterproof dry bag or Pelican box
- Cardiopulmonary resuscitation mouth shield (CPR Microshield)
- Hypothermia/hyperthermia thermometer
- Bandage scissors
- Fine-point tweezers or forceps
- Temporary dental filling (Cavit)
- Glutose paste
- Irrigation syringe with 18-gauge catheter
- Povidone-iodine solution
- 3M surgical stapler (1 stapler holds 25 staples)
- Dermabond tissue glue
- Wound closure strips (Steri-Strips)
- Tincture of benzoin
- Polysporin ointment
- Moleskin
- Latex or nonlatex (hypoallergenic) gloves
- Antiseptic towelettes
- Safety pins
- Waterproof matches
- Accident report form and pencil
- Large garbage bag
- 4- × 4-inch sterile dressings
- 8- × 10-inch trauma pad or Bloodstopper dressing
- Eye pads

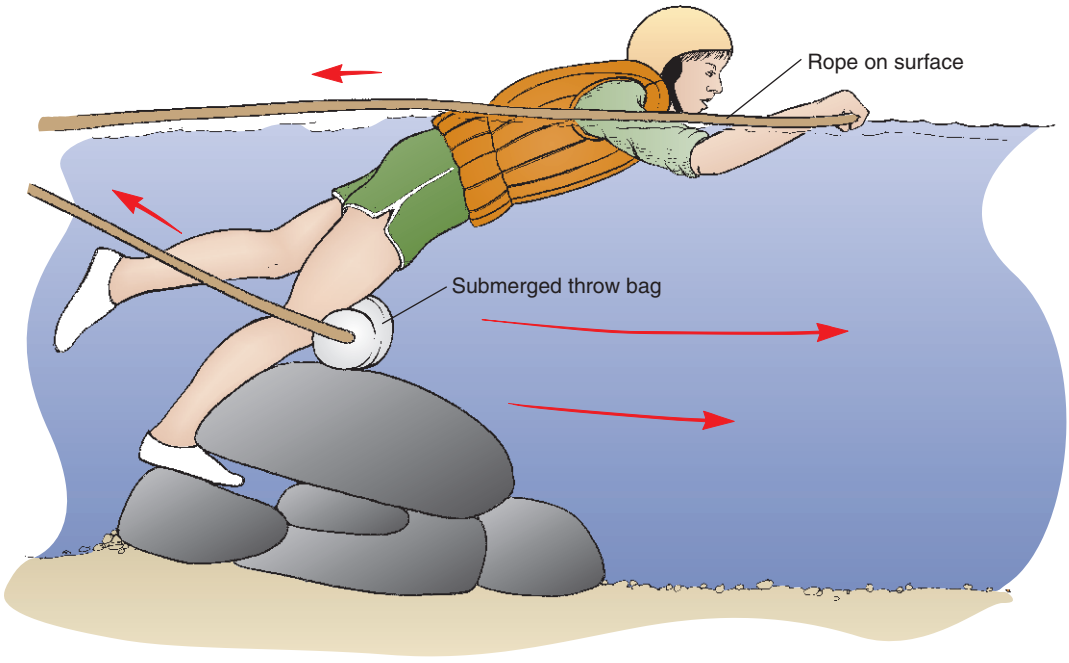


Figure 39-19. Submerged snag tag.

Nonadherent dressing (Xeroform or Aquaphor)
Triangular bandage
3-inch conforming gauze bandage
3-inch elastic bandage with Velcro closure
1-inch × 10-yard surgical tape
Duct tape (can be wrapped around the paddle shaft)
Strip and knuckle bandages
Cotton-tipped applicators
Aloe vera gel
Diphenhydramine capsules
Cortisone cream
Acetaminophen tablets
Ibuprofen tablets
Eardrops
Prophylactic eardrops (mixture of rubbing alcohol and white vinegar)
Treatment eardrops (Cortisporin Otic Suspension)
Epinephrine (injectable) or EpiPen
Prochlorperazine suppository
Diazepam or midazolam
Oxycodone
Oxymetazoline (Afrin) nasal spray
Antibiotics (trimethoprim-sulfamethoxazole, ciprofloxacin, cephalexin)
Sunscreen (sun protection factor [SPF] 15 or higher)
Insect repellent
Iodine tablets
Tampons
Tea bags

Kayaking Kit

Waterproof dry bag or small Pelican box
Cardiopulmonary resuscitation mouth shield
Hypothermia/hyperthermia thermometer
Scissors
Fine-point tweezers or forceps
Small surgical stapler (3M) or Dermabond Glue
Wound closure strips
Tincture of benzoin
Polysporin ointment
Latex or nonlatex (hypoallergenic) gloves
Antiseptic towelettes
Safety pins
Waterproof matches
Accident report form and pencil
3- × 3-inch sterile dressings
Nonadherent dressings
2-inch conforming gauze bandage
Duct tape
Strip and knuckle bandages
Cotton-tipped applicators
Diphenhydramine
Acetaminophen
Ibuprofen
Prophylactic eardrops (to protect against otitis externa)
Epinephrine
Prochlorperazine suppository
Diazepam or midazolam
Oxycodone
Sunscreen
Insect repellent
Iodine tablets

APPENDIX B: UNIVERSAL RIVER SIGNALS

Stop: Potential hazard ahead. Wait for "all clear" signal before proceeding. Form a horizontal bar with your

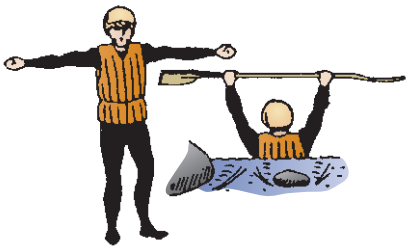


Figure 39-20. Stop signal.

Figure 39-21. Help/emergency signal.

outstretched arms. Those seeing the signal should pass it back to others in the party (Fig. 39-20).

Help/Emergency: Assist the signaler as quickly as possible. Give three long blasts on a whistle while waving a paddle over your head (Fig. 39-21).

All Clear: Come ahead (in the absence of other directions, proceed down the center). Form a vertical bar with your paddle or one arm held high above your head. Paddle blade should be turned flat for maximum visibility. To signal direction or a preferred course through a rapid around an obstruction, lower the previously vertical "all clear" by 45 degrees toward the side of the river with the preferred route. Never point toward the obstacle you wish to avoid (Fig. 39-22).

Figure 39-22. All clear signal.

APPENDIX C: ORGANIZATIONS

American Canoe Association (ACA)
7432 Alban Station Boulevard Suite B-232
Springfield, VA 22150-2311
Internet: www.acanet.org
Phone: 703-451-0141
Fax: 703-451-2245
E-mail: aca@americancanoe.org

American Whitewater (AW)
P.O. Box 1540
Cullowhee, NC 28723
Internet: www.americanwhitewater.org
Phone: 1-866-BOAT-4-AW
E-mail: info@amwhitewater.org

Chinook Medical Gear, Inc.
120 Rock Point Drive, Unit C
Durango, CO 81301
Internet: www.chinookmed.com
Phone: 970-375-1241
Toll Free: 800-766-1365
Fax: 970-375-6343
E-mail: admin@chinookmed.com

Special Rescue Services
P.O. Box 4686
Sonora, CA 95370
Internet: www.specialrescue.com
Voice: 209-743-9451
Fax: 503-210-8179
E-mail: Jim@specialrescue.com

National Organization for Rivers (NORS)
212 West Cheyenne Mountain Blvd.
Colorado Springs, CO 80906
Internet: www.nationalrivers.org
Phone: 719-579-8759
Fax: 719-576-6238
E-mail: nationalrivers@email.msn.com

The references for this chapter can be found on the accompanying DVD-ROM.

40 Caving and Cave Rescue

Steve E. Hudson and Loui H. McCurley

Caves are among the most awe-inspiring features remaining on Earth. The idea of finding virgin territory within a few miles of major cities worldwide is breathtaking. Caves are a world all their own. Unlike the far reaches of space or deep trenches of the oceans, uncharted caves can be explored by any reasonably fit person, without a plethora of fancy equipment.

Caves are found on every continent in a wide array of forms. There are limestone caves, lava tubes, ice caves, marble caves, shelter caves, and water-filled caves—the variations are endless. Full of beautiful formations, strikingly odd structures, and with mystical allure, caves have intrigued man since prehistoric times. From the practical use of caves as shelter to the sheer exhilaration of sport, it is the physical challenges of caving and the rare environment in caves that set them apart.

Cavers are a rare breed—people who thrive on the prospect of exploring deep canyons and pits within the earth. Fascination with the unseen, the thrill of the frontier, the personal contest of confronting the unknown, and the very darkness itself offer a distinctive aroma of adventure to which respond both exceptional cavers and the merely curious.

Caves can contain hostile environments. Medical and rescue professionals in particular should be thoroughly familiar with the harsh realities of the cave environment well in advance of engaging in medical or rescue duties underground (Fig. 40-1).

Even experienced cavers can quickly find themselves in unexpected trouble. Such was the case for Frank and Jim, two experienced cavers who explored a seldom-visited side shaft within an extensive cave in Georgia. It took Frank and Jim about 3 hours to walk up the mountain, negotiate the entrance, climb down into the cave, traverse several hundred feet of passage, rig and rappel a 125-foot (38.1-m) pit, travel across several hundred feet of additional passage, and then rig a side shaft they wanted to explore that ran parallel to the cave's main 586-foot (178.6-m) pit. Once down the shaft, Frank walked across a large rock slab. It shifted from his weight, and his lower leg was quickly pinned between the slab and the shaft's wall. Frank's lower leg was crushed, with pieces of bone sticking through an open wound. Over the next hour, Jim helped free Frank's leg and did his best to stabilize the injury and make Frank as comfortable as possible. Realizing there was no way Frank could climb the more than 550 feet (167.6 m) of rope back to the surface, much less walk and climb through the hundreds of feet of horizontal passage, Jim went out for help, leaving Frank alone, injured, and in great pain.

With the grim realization that he was now solo-caving and in extreme peril himself, Jim carefully climbed the pits and exited the cave as quickly as possible. Once down the mountain, he drove to town and notified local authorities.

Fortunately, the incident had occurred in a region of the country where an experienced cave rescue team was available. The team was called out and a response initiated. The first rescuer reached Frank about 7 hours after the accident. It took another 9 hours to get Frank up and across the pits and out the entrance passages using hauls, highlines, and team hand-carries. Next came a 30-minute four-wheel-drive trip off the mountain to the waiting ambulance. A short 15-minute ride had Frank in the nearest hospital. The rescue involved about 50 people, both inside the cave and as above-ground command and support. Total time was about 17 hours from accident to emergency room.

Approaching such a cave rescue situation with the mindset of it being merely a dark version of a typical wilderness environment is at best naïve and at worst a recipe for disaster. As for any environment, potential rescuers should be trained, experienced, and adept in functioning in the cave environment individually before assuming any sort of rescue role.

The first thought that enters the minds of many newcomers to caving is "claustrophobia." In fact, claustrophobia is usually less of a problem for cavers—even new cavers—than such things as physical agility, route finding, endurance, and maintaining optimum performance levels in cold, wet, and confined surroundings. Learning to cope, indeed to thrive, under such circumstances is a prerequisite for the challenges of cave rescue.

Not only must a cave rescuer be adept at managing the unique trials of functioning in the cave environment, he or she must also manage a unique set of rescue problems relating to safety, equipment, logistics, access and extrication, and mission support.

A cave rescue should be initiated only with the direct and intimate involvement of qualified cave rescuers. Rescuers without cave experience, or cavers without rescue experience, are not sufficient resources with whom to launch a cave rescue. The demands of each of these vocations are unique enough in themselves; the two together create demands that are humbling.

In addition to experienced ground personnel, the incident commander must be familiar with or seek the advice of someone who is familiar with the unique challenges of the cave environment.

The time to learn about caving is not as a medic on a cave rescue. If there is any chance that one will encounter a cave rescue as part of his or her profession or avocation, time should be spent becoming familiar with caves and caving techniques;

Figure 40-1. Opening header shot of litter in tight spot. (Photo courtesy Kris H. Green.)

the effort will at least be rewarding, and ultimately may be lifesaving.

ENVIRONMENT

Any natural opening in the earth large enough to enter is considered a cave. Caves are similar to human-made mines and tunnels only in that they share a subterranean setting. On a practical level, mines and tunnels must be approached with skills, equipment, and training that differ from those for caves. This chapter addresses only natural caves such as may be found in a wilderness environment, and not their dissimilar human-made counterparts.

Caves take many forms, including sinkholes, cracks, sumps, siphons, springs, pits, and caverns. The precise geology of cave formation is a more complex and diverse topic than can be adequately addressed within the confines of this chapter. Some caves are simply topographic in nature—cracks and fissures that are a natural result of typical geologic features. In this number are counted lava tubes, which form when a volcano erupts and lava flows away from its center. Gases create bubbles in the molten rock, leaving voids as it hardens. Lava tube caves form as flowing lava cools hard on the outside and continues to flow on the inside, leaving a tube of passage. Like most caves that are formed as a result of earthen upheaval and movement, lava tubes are unpredictable and may run for long distances or be limited to short passage and isolated rooms.

More interesting, perhaps, is the geology of caves that form over long periods of time. An oversimplification of this formation process is that caves—particularly limestone caves, but in truth any cave found in carbonic rock—are most often formed as a result of solution reaction between water, carbon dioxide, and the rock. Known as *karst*, such topography is formed when carbon dioxide and water combine to form weak "carbonic acid," which in turn works to dissolve the carbonic rock. This process is most commonly found in limestone areas.

As the limestone dissolves, the acid solution follows the path of least resistance through the earth, and eventually pits are formed, then fissures, and eventually passage. When the calcium carbonate–infused water reaches a large enough opening, the carbon dioxide dissipates and the calcium carbonate is deposited as *stalagmites* (icicle-like columns protruding from the ground) and *stalactites* (icicle-like columns hanging from cave ceiling).

Sumps, siphons, and springs are all names that describe specific features in water-based cave formations. *Springs*, an outflow of water from the earth, are an obvious access point between underground passage and the surface, but they can be called a cave only when they are large enough for a person to enter.

Springs differ from siphons in that where springs outflow water, siphons take in water. Air-filled cave passages that terminate in water-filled passages are known as *sumps*. Some sumps are only a few feet long before returning to air-filled sections, whereas others can be thousands of feet in length and may never resurface.

Sinkholes, cracks, pits, and *caverns* are terms that describe passage. These may be wet or dry and are differentiated by their shape and attributes. Sinkholes, also called *sinks*, are formed when bedrock above a void collapses. Sinks may present as a sheer vertical opening into a cave with a well-like or open-air pit entrance, a steep sloping depression with a cave entrance, or a shallow depression of many acres that may not have more than a small, impassable sump to the cave below.

Cracks are narrow vertical passages formed as the carbonic acid solution finds its way through the ground. These are usually, but not always, developed along a joint in the rock.

The term *pit* refers to a vertical cave passage. Pits can be found both inside caves and at the surface. Most open-air pits form when the roof of a sinkhole collapses. Pits can also be formed by solution or erosion of passage by flowing water.

Solution caves formed in soluble rock are the most common type found on Earth. Depending on local weather conditions and length of time, caves can take up to tens of thousands of years to form to a point where humans can enter. They are always getting larger through solution and erosion, or smaller by filling in with sand, mud, and fallen rock. These changes often happen so slowly that they are seldom noticeable.

Most cave ceilings and walls are relatively stable. Large breakdown blocks of fallen ceiling and wall rock are often found in caves, but the chances of one falling precipitously are slim. Breakdown usually occurs suddenly as a result of a major environmental event, such as an earthquake, or very slowly over years as the supporting rock below slowly degrades. Smaller rocks and rock slopes, often the result of ceiling and wall breakdown, are actually the greatest dangers in caves. Because they have not been stabilized by frequent travel, these slopes can shift and slide underfoot.

The continuous conduit leading through a cave is known as *passage*. Passage can be huge, with room dimensions so large it is difficult to see distant walls (Fig. 40-2) even with a light, while just around the next corner, passage can change to tight impenetrable cracks or a dead end (Fig. 40-3). Some caves have passages that wander around in a maze that may total many miles in as little as a few acres of ground. Others may go for miles in the same direction and involve dozens of miles of passage. Many caves are only a few hundred feet long and contain only low, tight, belly-crawl passage. Passage that opens into a wide area

Figure 40-2. Typical large "room." (Photo courtesy Kris H. Green.)

Figure 40-3. Typical tight crack passage. (Photo courtesy Kris H. Green.)

is called a *room*, whereas tight, narrow passage may be further described as a *squeeze* or *crawl*.

Caves are formidable, dark, and often dangerous. Considering that running, seeping, or standing water originally formed most caves, it is not surprising that water is a major part of many cave environments. Cavers and rescuers must be prepared to negotiate anything from crawls in water-filled tubes (Fig. 40-4) to underground rivers so large that a boat is required.

Caves that are no longer hydrologically active are called *dry caves* or *dead caves* because they are no longer in their formative state. Some caves are so dry that dust induces respiratory problems in visitors. Visitors to dusty caves should wear appropriate filter masks as a minimum level of protection from dust stirred up by the act of moving through a dry passage.

Figure 40-4. Wet passages require appropriate clothing. (Photo courtesy Kris H. Green.)

Figure 40-5. Close contact. (Photo courtesy Kris H. Green.)

Temperature extremes are another common characteristic of caves. Caves tend to be at the mean ground temperature of the area. For the most part, U.S. continental caves run from cool to cold. Very warm climates, such as in Puerto Rico, sport warm caves, whereas alpine mountain caves in places like Montana measure close to freezing temperatures and may even contain ice. Caves found in tropical and desert environments can be so hot that cavers must wear lightweight garments to explore them, and even then run the risk of heat illness.

A more common scenario in cave rescue, however, is the concern of hypothermia from sitting around underground waiting for the next assignment or struggling in cold and wet passages. Temperature differentials can also exist within a single cave, as a result of exposure, orientation, and water flow. These temperature differentials, as well as pressure differences, can result in winds flowing through the passages. It is not uncommon to be supine or prone in 4-inch deep, 13°C (55°F) water with one's back pressed against cold rock, facing a stiff breeze (Fig. 40-5).

Caves can be fragile, often heavily decorated with mineral formations that have formed over thousands of years. Cavers try to protect these formations whenever possible by avoiding walking on or touching delicate areas or otherwise altering the cave. Even the natural skin oils deposited by human hands can alter growth of an active formation. The caver's motto, "leave nothing but footprints, take nothing but pictures, and kill nothing but time," extends to rescue operations. Everything brought in must be packed out at the end of the operation. An abandoned flashlight battery can leach its chemicals and poison the cave-adapted life forms found in a cave passage, ultimately destroying the cave and its environment.

PERSONAL SAFETY

Whether entering a cave for exploration or for rescue purposes, personal gear requirements are tailored to caving. Clothing should be appropriate to the environment. Caves can be wet, dry, dusty, cold, warm, or a combination of these. The wind that can exist in passages makes chill factor an often unpredicted yet significant consideration. Undergarments should provide the necessary thermal layers and be of a fabric that remains warm when wet. Layering undergarments and thermal layers provides the most versatile system of clothing.

Many cavers wear protective suits with a rubberized, vinyl-coated, or Cordura outer layer, and in very wet caves, it is not unusual to find cavers in wetsuits. Coveralls or one-piece garments with no exterior straps or accessories help prevent snagging in crawls or tight passage. Ventilation is an important consideration in accommodating the various degrees of exertion required in caving.

A mountaineering type of helmet, with a nonelastic "three-point" chin strap that keeps it planted properly on the head, is a must. The helmet protects against impact with the hard and often sharp rock of cave ceilings and walls in tight or low passages and offers rock fall protection. It is also a convenient mounting platform for the required light source (Fig. 40-6).

It takes only one episode of trying to navigate in complete darkness underground to understand why no fewer than three light sources should be carried by each person in a cave. Rescuers underground without functioning lights become other subjects to be rescued. Electric lights are preferred, but carbide lamps may also be used. At least two of these lights should be helmet mountable for hands-free operation, each with sufficient "burn time" capacity or spare batteries to travel in and out of the cave. If carbide lamps are used, care should be taken when working close to victims because it is easy in tight spots to forget that the light on your head is an open flame that can quickly burn anything with which it comes in contact. For this reason, most cave rescuers prefer electric lights.

Figure 40-6. Typical helmet-mounted light. (Photo courtesy Kris H. Green.)

Many cavers find gloves useful for both thermal insulation and protection against sharp rocks and sticky mud. Neoprene or rubberized gloves are popular choices, and scuba or sailing gloves offer durable protection. If the cave has vertical components, leather-palmed gloves are necessary for rope work. Ideally, a rescuer should carry a different pair of gloves for rope work than for negotiating muddy passage.

Cave mud is slippery and adheres to everything. It makes walking and scrambling through a cave dangerous. Lug-sole boots provide the best traction, and stiff leather uppers help protect feet against sharp rocks. For small passages, or "crawlways," a set of durable kneepads is a necessity, and many cavers use elbow pads as well.

As in any remote environment, the caver should be self-sufficient and at least able to care for himself or herself for an extended period. This requires replacement batteries or carbide, fresh water for drinking, food for energy, a basic first-aid kit, and extra insulation, such as extra thermal layers and a hat that can be worn under the helmet. Cavers often store a folded trash bag in the suspension of their helmets to be used as an emergency shelter from wind and water, among other possibilities.

For the cave rescuer, the concept of self-reliance extends to being capable of caring for oneself as well as a patient for an extended period of time. Although the responsibility of patient care may be shared among several people, it takes planning to ensure that enough extra gear exists within the group so that the patient can be appropriately equipped and cared for.

An additional challenge for the extended cave visit is the requirement to pack out whatever is packed in, including human waste. Strong, leak-proof plastic containers are useful for that purpose.

Use a small, durable pack to carry extra gear. Because the pack will be alternately carried, pulled, pushed, and dragged through cave passages of different sizes, excessive size, straps, and external attachments should be avoided because they can impede maneuverability.

Not all caves have vertical drops, but for those that do—or when in doubt—a lightweight seat and chest harness, brake bar rack or figure-8 descender, and ascending system are essential. One or two 20-foot sections of 1-inch tubular webbing come in handy for an extra step-up or to construct a quick belay or hand line.

Vertical caving is a highly specialized sport, and vertical cave rescue even more so. The National Cave Rescue Commission offers courses in cave rescue, recommended for experienced vertical cavers.

CAVE NAVIGATION

Navigating through the cave environment can be disconcerting, particularly because of the three-dimensional nature of travel.

Cave passage moves upward, downward, and side to side through the layers of the earth. It may overlap, cross, and twist, all without the benefit of sun, stars, or moss on the north sides of trees to provide directional clues. It is always best for several cavers (or cave rescuers) in a given party to be familiar with the cave and be responsible for navigation. If this is not possible, several options exist. The first, and best, is to use a cave map. Reading a cave map is an acquired skill, and one that every cave rescuer should possess. In a pinch, cavers use navigational skills such as remembering landmarks, leaving temporary route markers (be sure to pick them up on the way out!) and using one wall of the cave as a source of consistency. Route markers are especially useful in cave rescue so that the rescuers can focus on the job at hand.

In an unfamiliar cave, keep track of prominent features so that you can find your way out. Passage often looks different on the way in than it does on the way out. A small crawl that enters into a large room can be hard to find hours later when returning across that same room. One trick for finding one's way out later is to get in the habit of turning around and looking at the passage as you enter into a different room, take a significant turn, or negotiate a climb. Rock cairns, light sticks, and flagging tape can be used as reminders, as can leaving pieces of reflective tape to mark your way and removing them again as you leave the cave.

CAVE SEARCH

When the location of the rescue subject is unknown, a search will be required. Searching a cave is a potentially hazardous task and must be done meticulously and under good planning and management.[1] A manager of a cave search uses the same basic skills as does an above-ground rescuer. Once the decision is made to begin an in-cave search, teams of cavers will be formed into task forces. Each Search Task Force's role is to look for, preserve, record, and recover clues that will lead to finding a missing person. The task force should consist of two or three people with one or more members familiar with basic search techniques, the cave being searched, and cave map reading. They should be able to perform basic first aid, including patient assessment. Required equipment in addition to personal caving gear discussed earlier would include basic medical supplies, materials to mark passages, pencil and paper, food and liquid for the subject, hypothermia protection for the subject, a map, and a compass. A marking system using colored flagging tape or other means should be established to mark side passage that has already been searched and to differentiate the best route in and out once a subject has been located.

Each Search Task Force should obtain a briefing from command and then follow instructions as to where to search, how long to search, probability of detection, how to preserve and record clues, how to report if a clue is found, and the debriefing procedures on return. If the search subject is found, the task force will assume the functions of an initial response task force. These are to determine whether the scene is safe, access the subject, treat the subject, consider possible courses of action, including walking the subject out if conditions allow, and then immediately notify command.

The report to command should include a full subject assessment, the task force's recommendation for course of action, additional medical equipment or personnel needed, type of litter needed, obstacles to evacuation, any rigging that will be needed, and suggestions for action to be taken.

BASIC EVACUATION

On any cave rescue, each person entering the cave must have at least the minimum complement of personal gear, as described in the previous section. Beyond this, the amount of equipment used during a cave rescue is prodigious, particularly in a cave with complex terrain. Equipment and personnel are used nearly as quickly as they can be produced; dry clothing, food, and water become valuable commodities; and extra supplies not normally used in cave exploration become necessities.

The latter group, items not normally used in cave exploration, should be sourced, tested, and stored at the ready well in advance of being needed. Communications equipment, such as hard-wired field phones or cave radio systems, can be particularly difficult to find—as can experienced personnel to operate them. Other needs may include generators, warming tents, food services, and a command post.

Although most cave evacuations require litter transport, each individual situation should be evaluated carefully to determine whether a litter is really necessary. A properly stabilized "walking wounded" caver can be helped out of a cave in short time and with little manpower. Put that same person into a litter, and the number of rescuers and hours to the hospital goes up exponentially.

One crisp October day, cave rescuers in Georgia were dispatched to Pettijohn's Cave on Pigeon Mountain to evacuate a fall victim some 30 minutes from the entrance. Samuel, a fit, experienced caver who was quite familiar with the cave, had taken a fall, experiencing a dislocated shoulder in the process.

Pettijohn's Cave is normally considered a horizontal caving experience, but it also involves scrambling over breakdown block and negotiation of several short, vertical drops. Although Samuel and his friends were quite familiar with the cave and he could easily negotiate the passage under normal circumstances, loss of the use of his arm would make it impossible for him to balance or climb on the difficult terrain. The pain of such a dislocation complicated matters greatly, and even a litter evacuation would prove to be a trying ordeal.

Terrain in the cave dictated that litter evacuation would require several roped systems, including an initial raise of 15 to 20 feet, a 100-foot highline, and one to two more raises to lift the patient through and out the cave entrance. Although only an estimated 30-minute walk into the cave, the evacuation would easily encompass 6 hours and require numerous riggers and scads of equipment.

The arrival of an experienced paramedic resolved the evacuation in short order by reducing the dislocation and immobilizing the limb securely to the victim's body. Instead of litter lowers and highlines, the rescuers were able to rig handlines, and Samuel walked out of the cave under his own power in a fraction of the time that the technical evacuation would have taken.

Not all cave rescue scenarios benefit from the fitness and ability of such a caver or the good fortune of a medical situation that can be resolved by a skilled paramedic on scene. If litter transport is deemed necessary, the next difficult decision is which litter (or litters) to use. On any given evacuation, this

Figure 40-7. Plastic litter offers patient protection and slides easily through passage. (Photo courtesy Kris H. Green.)

selection requires a fine balance of requirements. Maneuvering a bulky litter through narrow cave passages can be a challenging proposition. In larger caves, or where a vertical raise is required, basket litters are the best choice. Plastic-bottomed versions are preferable over steel and mesh versions because these allow the option of dragging the litter where necessary (Fig. 40-7). In tighter caves, drag-sheet types of litters, such as the wraparound Sked, provide low-profile and relatively flexible advantages, but are less comfortable or protective for the victim.

Occasionally, a cave is so restrictive that even the length of a flexible litter is problematic, and a short board, such as a KED or OSS, is the only alternative. It is not unheard of to begin the carry in a tight section of a cave using an OSS, add a Sked once the passage opens up a bit, drop the Sked into a full basket litter for ease of carrying in a large walking passage, then drop all the way back to the OSS to negotiate a tight entrance passage.

When choosing a litter and victim packaging, duration of transport and patient comfort should be prime considerations. Natural positioning, pressure points, and temperature are all key concerns. Although rescuers are working up a sweat, the patient lying in the litter is stationary, unable to regulate his own comfort, and can be extremely cold. Waste elimination may well need to be provided for. Thermal layers are a necessity and include a sleeping bag, vapor barrier, and moisture barrier to keep the patient dry. A full-body vacuum mattress works well as padding, immobilizer, and insulator. The patient should also have adequate head and face protection and, if the environment will become vertical at any point, a harness.

A reasonable first-aid kit for a cave rescue operation should contain writing materials for recording victim condition, basic medications, airway management tools, bandages, cervical collars, and splints. Sealing each item in plastic helps keep out cave grit, and the entire kit can be packaged in a large-mouthed bottle or other watertight, durable container.

Because of extended times involved in reporting caving accidents and responding to and accessing injured cavers, most victims are either very stable or dead by the time medical help arrives. Advanced life support (ALS) skills and equipment are generally not required. This is fortunate because the effects of cave mud and water are not particularly friendly to medical electronics.

Sometimes a cave accident victim is so badly injured that physicians or paramedics on scene choose serious drug intervention. At these times, electronic tools for monitoring vitals can be extraordinarily valuable, but great care should be taken. The cave environment is neither kind to such devices, nor is it friendly to even simple interventions such as intravenous (IV) infusions. In most cases, hanging a gravity-operated IV line is not an option, and positive-pressure infusion methods must be used. Furthermore, consideration must be given to methods for keeping the insertion point clean, and care taken not to interrupt flow while negotiating tight passageways.

Equipment—Vertical Evacuation

Ropes used for high-angle underground rescue are usually of the static kernmantle variety. Low stretch, abrasion resistance, and consistency of quality are considered key ingredients. Caves can be harsh and unforgiving, so for this purpose, the tougher the sheath, the better the rope. The toughest-sheathed rope is usually less pliable and forgiving than those more prone to abrasion, so skilled riggers should be used for the task. Rope length may vary, depending on the particular cave and the resources at hand. Ideally, rope lengths should be sufficient to negotiate each drop without tying knots to join ropes. Knot passes can add undesired complexity to the operation.

Most cave rescues involve negotiation of difficult passage as well as raising the patient from below ground to the surface. This may be accomplished in any number of ways, but pulleys and haul systems are generally basic necessities.

Techniques used in high-angle rescue in the cave environment are quite similar to those used above ground, but may be complicated by tight spaces, darkness, environmental conditions, and lack of alternative routes.

Brake bar racks are most often preferred for lowering operations because of their variable friction advantages. Aside from these, adequate carabiners, anchor materials, and other hardware should be available for rigging. If multiple locations must be rigged for raising, lowering, or traversing, the best possible scenario is to have enough gear to rig each site individually. Having to de-rig a system, sort the gear, carry it past the proceeding litter, and re-rig another system can be time consuming and derail the entire operation. Even a small cave might require multiple sites rigged for safe patient extrication.

Prebagged packages of gear for specific common rigging tasks are often useful. These include anchor webbing, ample carabiners, pulleys, prusiks, belay devices, lowering devices, and rope grabs as required to set up one site per bag.

Logistics

Lack of easy communication, limited access, extended time, and difficulty in obtaining rest and nutrition for teams all contribute to complexity.

Communication is essential to keep rescuers from becoming lost, to issue instructions, and to maintain the tempo of the

operation. Various hard-wired field-phone systems are available for use in cave rescue, but generally only well-established teams have access to these. A relatively new development is availability of special low-frequency cave radios that can transmit voice through dense rock and soil. Without such systems, message runners are indispensable. A group of swift, agile, safe, and well-trained cavers with waterproof notepads—and a method for keeping them rested and nourished—is invaluable.

A team of rescuers sent into a known location can take hours or most of a day to reach the victim. It is logistically impractical for these rescuers to carry sleeping bags and food to allow rest and recovery before starting their work assignment. Keeping rescuers rested, fed, watered, and warm many hours away through a challenging cave rescue requires an incident management team that can predict the needs of the underground workers hours before they themselves realize the need.

Whether or not a communication system is available, establishment of a control point at or near the cave entrance is critical. Entrance control should be established as soon as rescuers arrive at the cave. All personnel and equipment entering or leaving the cave should be recorded into a log kept by people assigned to maintain entrance control. This log becomes invaluable hours later to determine when teams should be replaced, if someone is still in the cave, and who carried in what piece of missing vital gear.

The Incident Management System (see Chapter 20), or a modified version of it, provides the best framework for managing cave rescue personnel by performing required functions while maintaining a reasonable span of control. Generally, the functions required on a cave rescue are similar to those required on any other rescue, although the specific means of accomplishing the functions will vary.

There should always be one person in command of an operation. This is the foundation of creating accountability and organization, which are the keys to efficiency and safety. The incident commander assesses the incident, activates resources, determines the strategy, and approves the plan for the operation. This may be particularly challenging in jurisdictions where the cave rescue experts are not the same individuals as the emergency response experts. Preplanning and relationships developed in advance are the best solution for preventing problems in this area.

Other functions vital to success are planning, operations, logistics, and finance. The incident commander may have one or more people to assist, or he or she may be responsible for several of these functions. Someone must plan strategies, supervise the operation, take care of the physical needs of the rescuers and required equipment, and track the resources used.

Cave Access

Gaining access to the victim is a matter of overcoming an array of obstructions inherent in the cave. Merely to move a few hundred feet through a cave might require rappelling, crawling on one's belly, squeezing through cracks in the rocks, climbing over large rocks, swimming, and slithering through mud—all of which are quite different experiences when embarked on during a rescue while dragging a vast array of equipment.

The total darkness of a cave is limiting to any operation and can be confining to some. Even this simple matter can quickly become a major obstacle, particularly if noncavers are called on to assist in medical or other aspects of the operation. Psychological inhibitions, such as fear of the dark or confined spaces, can cause panic and severe dysfunctional behavior. In no case should a would-be rescuer ever be pushed beyond his or her comfort level.

Other factors inherent to the cave rescue operation include temperature variables, wetness, and restrictive cave passages. Certain large or weak people or bulky and heavy equipment might be physically incapable of getting through these tight spaces. These considerations exemplify why it is best for any medical or other rescue person who may eventually become involved in a cave rescue to gain knowledge and experience in advance.

If the cave rescue requires raising or lowering a victim, or traversing the victim over horizontal rope lines, people skilled in cave rigging should be responsible for building the systems. Rigging in caves is an art because of anchoring difficulties, directional changes, tight squeezes, and minimal working surface. Details on cave rigging and professional training can be acquired through the National Cave Rescue Commission, a nonprofit organization that teaches courses in cave rescue techniques and management.

Many vertical drops in caves are overhung at the top, preventing the rescuer access to a wall while descending and ascending the rope or moving the patient. In cases in which the roped drop has the rope running against a wall, it can be advantageous to place anchors at points throughout the length of the drop. This "re-belay" method allows multiple people to ascend or descend simultaneously, lessens rope wear points, and provides the added safety of having a shorter rope length to protect for each anchor. Practice at crossing re-belays is essential before attempting to enter a cave thus rigged. The caver must be able to effectively transfer from one free-hanging rope to another while hanging in midair. This is easy to do with the correct equipment setup and practice, but not so easy when the technique is tried for the first time underground on the way to a medical emergency.

Usually, single-rope techniques are used, especially where a long free-hanging drop is involved. This means that just one rope is put over the side for the rescuer to ascend or descend. The use of an additional belay line not only requires additional people but also might prove more hazardous if the two lines become entangled.

In the United States, the most common cave ascending systems are the frog system, Mitchell system, and various renditions of the ropewalker system. These are all efficient means of ascending and can easily be mastered with practice. The frog system requires more climbing effort but is easier to use when ascending past re-belays in the system.

It is imperative that an ascending system be fitted to the user. Some rigs work better for tall, lean frames, and others work best for heavier body types. All rescue personnel entering a vertical cave should have their own personally fitted rope climbing system and must have practiced climbing in that system in a safe practice area.

Large holes or boulder slopes inside the cave may best be negotiated by using a highline traverse. Highlines can be time and equipment intensive to set up properly but can shave away hours of litter movement time by passing above difficult cave terrain that otherwise would present many challenges for a litter carried by hand. Use of a highline is most practical when it is known in advance that there will be sufficient time for rigging. The decision to take the time to rig a highline traverse should

be made based on three factors: the time it will take to move the victim to the obstacle you want to be traversed, the time necessary to rig the highline, and the time that will be saved by using the highline to move the victim over the obstacle.

Environmental Hazards

One often-overlooked hazard to cavers is the ambient atmosphere. Most caves in the United States breathe naturally either from changes in barometric pressure or from the chimney effect of multiple entrances. When this airflow is interrupted, limited, or polluted in some way, the resulting air can become hazardous to entrants.

The most basic type of atmospheric hazard is lack of oxygen. Some caves have small rooms with so little airflow that a few cavers can quickly consume most of the oxygen. While frightening, this type of atmospheric hazard is usually quickly recognized as open flame headlamps sputter or cavers become short of breath. Once recognized, the hazard can be mitigated by avoiding panic, controlling air consumption, and relocating to a friendlier part of the cave.

Another type of atmospheric hazard involves buildup of gases such as carbon monoxide, carbon dioxide, methane, and hydrogen sulfide. This can happen as a result of natural metabolism of organics or through external influences. Instances of gasoline seeping into caves from underground storage tanks have been recorded. If poor air quality is suspected, use of an air-monitoring device is essential. It is possible to enter a cave containing high levels of unhealthy gases, but only with appropriate caution. In such cases, it is advisable to solicit participation of the local hazardous materials emergency response team.

With the assistance of a hazardous materials or confined space rescue team, "bad air" in caves can be mitigated in several ways. One way is to release compressed air into the cave, forcing good air in and bad air out. Success of this method is limited, and because of the massive amounts of air in a cave, this method is slow at best. If this method is used, entrants should carry an air monitor because pockets of bad air may remain trapped in parts of the cave. The concept of simply releasing oxygen into the cave is a nice idea, but impractical in all but the smallest of spaces. Exhaust fans offer a reasonable method, although care must be taken to prevent generator exhaust from entering the cave.

If necessary, rescuers can be equipped for entry with surface-supplied air with bail-out bottles, self-contained breathing apparatus, or rebreathers. Each of these has advantages and disadvantages, but all are difficult to manage in the cave environment and thus should be avoided if possible. Pre-event training and practice in the safe use of any of this equipment is imperative before entering an atmosphere that is hazardous for breathing.

Other airborne hazards, such as histoplasmosis and rabies, are not detectable by air monitor. Histoplasmosis is a human fungal disease that may be contracted by inhalation of organisms found in bird droppings and bat guano. The fungus is a potential inhalation hazard in any cave, but particularly in caves largely populated by bats. Frequent, mild exposure can actually result in strengthening of the immune system against the disease, but a severe case can be deadly. Pulmonary histoplasmosis generally presents with flu-like symptoms within 2 to 4 weeks of exposure and may require hospitalization. Left untreated, histoplasmosis can destroy the lungs and result in death within years or even months.

Figure 40-8. Recreational caver in wet pit. (Photo courtesy Kris H. Green.)

Rabies is also a recognized hazard for cavers who enter caves with large bat colonies. Although actual risk for infection is considered minimal unless the caver is frequently handling bats (which is generally frowned on anyway), immunization against rabies is available and advisable for cave biologists and other scientists who run a high risk for being bitten.

In any rescue situation, atmospheric hazards should be considered before entering a cave. If suspected, no rescuer should enter the environment without an appropriate filter mask and other personal protective equipment.

Other environmental hazards posed by caves may be more readily recognized and should also be considered before any rescue. As previously discussed, water and caves are usually closely associated. Created by water, caves are a natural deposit for overflow or drainage from a variety of sources. This makes caves particularly prone to flooding with little or no warning. A recreational caver (Fig. 40-8) or rescuer caught in a flooding cave is in mortal danger.

Flooding is usually associated with heavy rains, which can cause diffuse seepage over a large area of the cave or a high flow into sinkholes. Occasionally, sinking streams can carry floodwater. In some parts of the world, entire rivers disappear underground, flow through caves, and resurface miles away. A flood crest from many miles upstream can pass through these caves without warning.

Flood-prone caves are generally identifiable by their makeup. Cave walls coated with thick mud can be an indication that flooding is not unusual in that section of the cave, and extra caution is warranted. Bedrock cave walls with gravel deposits at key points in the cave and debris lodged in the ceiling can also be warning signs.

Becoming trapped in a flooding cave is not desirable, but it can be survivable. If possible, find a high point in a wide passage, downstream of any major constriction, and wait out the flood. It is also possible, with enough advance warning, to remove a sediment dam downstream that may otherwise cause water to back up into your "safety zone." It is seldom wise to attempt to swim, either upstream or down.

For a rescuer called to assist cavers trapped in a flooded cave, entering the torrent is not wise. If the location of the trapped victims is known, it may be possible to use (or make) another

entrance from which to evacuate them. Knowledgable local cavers can be a valuable asset in this instance.

If entry into a flooded cave through the main entrance is absolutely necessary, it is imperative that the water level be controlled before entry. Methods of accomplishing this goal vary depending on the situation. Often, it may be enough to simply wait out the flood and let the water level subside naturally. If the water level is still on the rise, however, or if the source remains constant, additional measures may be warranted. Keep in mind that water is a powerful force, and any plan should be engineered by professionals.

One of the simplest diversion methods is to broaden the flood crest so that less of it flows into the cave. Water can be diverted using sandbags, hay bales, or dirt or by digging channels. If this is not possible or feasible, one may be able to lower the water level by digging through debris downstream, thereby expediting the exit. Pumping is also an option, although the hazards inherent in this method should be evaluated closely beforehand. Pumps run on fuel or electricity. Fumes from fuel-driven pumps and generators have entered caves and poisoned the air of the cavers waiting for rescue. Electrical power in a flooded cave situation adds a high risk for electrocution to the operation. Failure of the pumps after rescuers enter a cave can trap additional people if the water can rise again.

Entering a flooded cave with scuba equipment is a dangerous, last-resort method that should only be attempted by certified cave divers. A scuba entry may be justified if cavers are known to have been entrapped for an extended period of time, if there is a known medical emergency, or if the cave is completely flooded. In these cases, it may be advisable for certified cave divers to enter and assess the condition of entrapped people, transport survival supplies, or provide medical assistance. Even certified cave divers require additional skills and equipment to enter a recently flooded or sumped cave passage, compared with the typical sport cave dive. Flooded cave passage may have little to no visibility, currents, and debris blocking the passage. Only in the most dire circumstance is it justified to attempt to transport a victim through a flooded passage.

At a minimum, a scuba entry requires two to three divers, as well as a backup diver. Diving is gear intensive and requires an air compressor, extra tanks, 110-volt electricity to charge dive lights, underwater communications equipment, waterproof bags, full face masks for subjects, water rescue suits, underwater lights, transport cases, and surface personnel to assist with transporting equipment.

Victim Care

As with many remote accidents, the time it takes to report, respond to, and access a caving accident usually means that the victim, if alive, is relatively stable. Although this generalization has exceptions, the treatment issues faced by most rescuers are related to extended transportation times.

Data compiled for American Caving Accidents indicate that the leading cause of caving injury is falls, and that hypothermia, fractures, and head injuries top the list of complaints. Unfortunately, spinal injury is invariably present, and this can compound the transportation challenge. The approach to medical care should be similar to any other medical situation, with one notable difference: the victim has suffered an acute injury but will be confined for transport for an extended period. Care, then, will be a combination of acute emergency responses adapted for a victim who is, for all practical purposes, bedridden.

Figure 40-9. Monitoring patient as litter moves. (Photo courtesy Kris H. Green.)

Once the victim has been stabilized and packaged, the assessment process should continue throughout the evacuation. It is best if one medical person stays with and monitors the victim throughout the evacuation (Fig. 40-9).

Hypovolemia is a common complaint, so establishing an IV line early in the intervention can be useful. Take measures to ensure that rescuers will be able to maintain IV access and manage the supplies throughout the evacuation, and infuse only fluids not contraindicated for head or other injury. If the victim is alert and oriented, fluid administration will increase the need to urinate, so take this into consideration. As best as possible, maintain communication with the victim, encouraging him or her to flex muscles to maintain good circulation. Allow the victim to assist in care as much as possible.

Availability of ALS and drug therapy is useful on extended transports, so it is helpful to have strong rapport with the local medical authorities should complex treatment become necessary.

CONCLUSION

Never underestimate a cave rescue. Caves are unique environments, and entry should not be attempted without appropriate technical training and preparation.

The advantage to any rescue group of establishing a good working relationship with local cave rescue resources, as well as local recreational cavers, cannot be overemphasized. These are the people with knowledge of the caves; with the training, equipment, and experience to handle the obstacles and the environment; and who already feel at ease underground. They could prove to be your most valuable resource for a successful cave rescue. Many cavers have taken extensive training in cave rescue techniques and are members of organized cave and cliff rescue

teams. The first step to finding cavers is to contact a local chapter, or "grotto," of the National Speleological Society, the largest cave exploration, education, and science-oriented organization in the United States. Contact them at:

National Speleological Society
2813 Cave Avenue
Huntsville, AL 35810
nss@caves.org
http://www.caves.org/

Given the unique underground environmental and topographic conditions, nothing can replace formal training in cave rescue techniques and specific cave rescue problems and solutions. People interested in enhancing their training should get in touch with the National Cave Rescue Commission (NCRC). The NCRC conducts week-long cave rescue seminars and weekend orientations across the United States. Contact them at the address above as the National Cave Rescue Commission of the National Speleological Society or at www.caves.org/io/ncrc/.

SUGGESTED READINGS

Hudson S (ed): Manual of U.S. cave rescue techniques, 2nd ed. Huntsville, AL, National Speleological Society, 1988.

Putnam W (ed): American caving accidents. Huntsville, AL, National Speleological Society, published annually.

The references for this chapter can be found on the accompanying DVD-ROM.

41 Protection from Blood-Feeding Arthropods

Mark S. Fradin

Of all the hazards, large and small, that may befall the outdoor enthusiast, perhaps the most vexatious comes from the smallest perils—blood-feeding arthropods. Mosquitoes, flies, fleas, mites, midges, chiggers, and ticks all readily bite humans. The bites are, at best, only a minor annoyance; at worst, arthropod bites may transmit to humans multiple bacterial, viral, protozoal, parasitic, and rickettsial infections (Box 41-1). Mosquito-transmitted diseases alone will be responsible for the deaths of one out of every 17 people currently alive.[129] This chapter reviews the arthropod species that bite humans and discusses various options for personal protection against nefarious insects.

Mosquitoes (Family Culicidae)

Mosquitoes are responsible for more arthropod bites than any other blood-sucking organism. They can be found all over the world, except in Antarctica. These two-winged insects belong to the order Diptera. There are 170 species of mosquitoes in North America, and over 3000 species worldwide. Anopheline, or malaria-transmitting, mosquitoes can be distinguished by their resting position on the skin—their bodies are characteristically raised high, as if they are standing on their heads. Most other species, in contrast, rest with their bodies parallel to the skin surface (Fig. 41-1A).

Mosquitoes vector more diseases to humans than any other blood-feeding arthropod. They transmit malaria to 300 to 500 million people each year, resulting in as many as 3 million deaths per year.[119] They vector multiple arboviruses to humans, including several forms of encephalitis, epidemic polyarthritis, yellow fever, and dengue fever (see Chapter 42). Mosquitoes also transmit the larval form of the nematode that causes lymphatic filariasis.

Only female mosquitoes bite, requiring a blood-protein meal for egg production. Male mosquitoes feed solely on plant juices and flower nectar. Mosquitoes feed every 3 to 4 days, consuming up to their own weight in blood with each feeding. Certain species of mosquitoes prefer to feed at twilight or nighttime; others (such as the aggressive Asian tiger mosquito, *Aedes albopictus*) bite mostly during the day. Some mosquito species are zoophilic (preferring to feed on animals, including birds, reptiles, mammals, and amphibians), whereas others are anthropophilic (preferring human blood). Members of the genera *Anopheles*, *Culex*, and *Aedes* are the most common biters of humans. In some mosquitoes, seasonal switching of hosts provides a mechanism for transmitting disease from animal to human.

Mosquitoes rely on visual, thermal, and olfactory stimuli to help them locate a bloodmeal.[10,11,19,25,45,61] For mosquitoes that feed during the daytime, host movement and dark-colored clothing may initiate orientation toward an individual. Visual stimuli appear to be important for in-flight orientation, particularly over long ranges, whereas olfactory stimuli become more important as a mosquito nears its host. Carbon dioxide and lactic acid are the best-studied attractants. Carbon dioxide serves as a long-range attractant, luring mosquitoes at distances of up to 36 meters (118 feet).[43,44,122] At close range, skin warmth and moisture serve as attractants.[10,19] Volatile compounds, derived from sebum, eccrine and apocrine sweat, and/or the cutaneous microflora bacterial action on these secretions, may also act as chemoattractants.[51,68,84,115] Floral fragrances found in perfumes, lotions, soaps, and hair-care products can also lure mosquitoes.[36] One study has shown that alcohol ingestion increases the likelihood of being bitten by mosquitoes.[120]

There can be significant variability in the attractiveness of different individuals to the same or different species of mosquitoes.[22,62] Men tend to be bitten more readily than women, and adults are more likely to be bitten than children.[62,90] Heavyset people are more likely to attract mosquitoes, perhaps because of their greater relative heat or carbon dioxide output.[103]

During the day, mosquitoes tend to rest in cool, dark areas, such as on dense vegetation, or in hollow tree stumps, animal burrows, and caves. To complete their life cycle, mosquitoes also require standing water, which may be found in tree holes, woodland pools, marshes, or puddles. To minimize the chance of being bitten by mosquitoes, campsites should be situated as far away from these sites as possible.

Blackflies (Family Simuliidae)

At 2 to 5 mm in length, blackflies[7,14,29,46,60] (see Figure 41-1B) are smaller than mosquitoes. They have short antennae, stout humpbacked bodies, and broad wings. Blackflies are found worldwide. The adults are most prevalent in late spring and early summer and are most likely encountered near fast-running, clear rivers or streams, which they require to complete their life cycle. Unlike most mosquitoes, blackflies tend to bite during the daytime. They primarily use visual cues to locate a host. Dark moving objects are particularly attractive, but carbon dioxide and body warmth also serve as attractants. Only the female bites, taking up to 5 minutes to feed. Blackflies may be present in swarms, inflicting numerous bites on their victims.

Blackflies are attracted to the eyes, nostrils, and ears of their hosts. They often crawl under clothing or into the hair to feed.

Box 41-1. Diseases Transmitted to Humans by Biting Arthropods

MOSQUITOES
Eastern equine encephalitis*
Western equine encephalitis*
St. Louis encephalitis*
La Cross encephalitis*
West Nile virus*
Japanese encephalitis
Venezuelan equine encephalitis
Malaria
Yellow fever
Dengue fever
Bancroftian filariasis
Epidemic polyarthritis (Ross River virus)
Chikungunya fever
Rift Valley fever

TICKS
Lyme disease*
Rocky Mountain spotted fever*
Colorado tick fever*
Relapsing fever*
Ehrlichiosis*
Babesiosis*
Tularemia*
Tick paralysis*
Rickettsial pox*
Tick typhus
Southern tick-associated rash illness (STARI)

FLIES
Tularemia*
Leishmaniasis*
African trypanosomiasis (sleeping sickness)
Onchocerciasis
Bartonellosis
Loiasis

CHIGGER MITES
Scrub typhus (tsutsugamushi fever)
Rickettsial pox*

FLEAS
Plague*
Murine (endemic) typhus

LICE
Epidemic typhus
Relapsing fever

KISSING BUGS
American trypanosomiasis (Chagas' disease)

*May be found in the United States.

The insect's mouthparts are used to tear the skin surface, producing a pool of blood from which the fly feeds. Blood loss from the bite site often persists after the blackfly has departed. The resulting intensely pruritic, painful, and edematous papules are typically slow to heal. Rare systemic reactions, including fever, urticaria, anaphylaxis, and even death, have been reported following blackfly bites. Although these flies are not known to transmit disease to humans in North America, blackflies in the tropics are vectors of the parasite *Onchocerca volvulus*, which causes river blindness.

Midges (Family Ceratopogonidae)

Also known as no-see-ums, sand gnats, sand fleas, and "flying teeth," biting midges[7,14,29,46,60] are small, slender flies (less than 2 mm) with narrow wings (see Figure 41-1C). Their small size makes them difficult to see, and they can pass readily through common window screens. Biting midges are found worldwide. They breed most commonly in salt marshes but may also be found in freshwater wetlands. Despite their inconspicuous size, female midges are aggressive biters, frequently attacking in swarms and inflicting multiple painful and pruritic bites within minutes. Midges often crawl into the hair before biting. Depending on the species, midges may bite during the day or at night. Their activity is greatest during calm weather, declining as wind speed increases. Biting midges are not known to transmit disease in North America.

Tabanids (Family Tabanidae)

The family Tabanidae (see Figure 41-1D) includes horseflies, deerflies, greenheads, and yellow flies.[7,14,29,46,60] These insects are relatively large (10 to 20 mm) robust fliers, with numerous species worldwide. Tabanids breed in aquatic or semi-aquatic environments and have a life cycle of over a year. They can fly for miles and rely primarily on vision to locate a host by movement. These flies are most active on warm, overcast days. Only the females bite, using scissorlike mouthparts to create a bleeding slash in the skin that is slow to heal. Despite their size, these flies usually bite painlessly, but the resulting reaction can include intense itching, secondary infection, and, rarely, systemic reactions such as urticaria or anaphylaxis. Because the adult fly usually lives only about a month, and only one generation emerges per year, the potential season for being bitten is fortunately relatively short. In the United States, deerflies have been shown to be capable of transmitting tularemia to humans; in Africa, the deerfly may vector the filarial parasitic worm *Loa loa*.

Sand Flies (Family Psychodidae)

Sand flies[7,14,29,32,46,60] are tiny (2 to 3 mm), hairy, and long-legged flies, with multisegmented antennae and a characteristic V shape to the wings when at rest (see Figure 41-1E). Only female sand flies are blood feeders, and they feed mostly during calm, windless nights, and rest during the day in animal burrows, tree holes, or caves. Most sand fly bites tend to occur on the face and neck.

In tropical and subtropical climates, sand flies have been shown to vector multiple cutaneous, mucocutaneous, and systemic diseases, including bartonellosis and three forms of leishmaniasis. The only sand fly-transmitted disease in the United States has been cutaneous leishmaniasis, reported in Texas.

Tsetse Flies (Family Glossinidae)

Tsetse flies[7,14,29,46,60] are found only in tropical Africa. They are 7 to 14 mm long, yellowish brown, and with wings that fold over their backs, giving them the appearance of honeybees at rest (see Figure 41-1F). Both sexes bite, feeding in daytime on a wide variety of mammals, including humans. Tsetse flies seem

Figure 41-1. Blood-feeding arthropods. **A,** Mosquito: *Culex* and *Anopheles*. **B,** Blackfly. **C,** Biting midge.

to rely primarily on vision and movement to identify their hosts. Their bites may cause petechiae or pruritic wheals. Tsetse flies vector African trypanosomiasis (sleeping sickness).

Stable Flies (Family Muscidae)

Stable flies[46,60] resemble common houseflies and are most often encountered in coastal areas. Unlike a housefly, which rests with its body parallel to the surface, a stable fly rests with its head held higher than its posterior (see Figure 41-1G). Both male and female stable flies are vicious daytime biters, requiring a blood meal every 48 hours to survive. If disturbed, they will attempt to feed multiple times, preferring to bite the lower extremities. Horses and cattle are the preferred hosts, but hungry stable flies will readily bite humans. These flies have knifelike mouthparts that they use to puncture flesh before pumping up the blood. Stable flies breed in decaying vegetation and in herbivore manure and are frequently found congregated on sunny walls. Their bites are generally self-limited. They are not known to transmit disease to humans.

Kissing Bugs (Family Reduviidae)

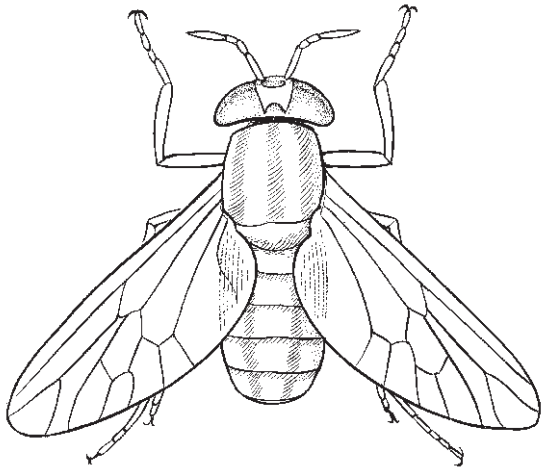
Kissing bugs[7,14,29,33,46,60] (assassin bugs) are large (10 to 30 mm in length) insects with cone-shaped heads, overlapping wings, and an alternating pattern of orange and dark brown stripes on the lateral abdomen (see Figure 41-1H). They get their name from a tendency to bite around the human mouth, but they may also bite other parts of the body. Both male and female reduviids bite, requiring a blood meal to mature through five nymphal growth stages. Reduviids are nocturnal feeders, attracted to their hosts by warmth, carbon dioxide, and odor. During the day, they rest in trees or indoors in crevices of house walls and ceilings. Their bites are initially painless, but frequent exposure to the bites can produce erythema, edema, and pruritus at the bite sites. More importantly, kissing bugs are the vector for *Trypanosoma cruzi*, the causative agent of Chagas' disease, which has been reported in Central and South America, as well as in the southwestern United States.

Fleas (Family Pulicidae)

Adult fleas[7,14,29,46,60] are small (2 to 6 mm), wingless insects, with 3 pairs of powerful legs that enable them to jump distances of up to 30 cm (see Figure 41-1I). Hungry fleas of both sexes feed on the nearest warm-blooded animal, without clear host preference. Fleas usually move around, probing and biting several times, resulting in grouped lesions of pruritic papules. Fleas are capable of transmitting sylvatic plague and murine typhus.

Chigger Mites (Family Trombiculidae)

Trombiculid mites[7,14,29,46,60] (see Figure 41-1J) are found worldwide. Commonly known as chiggers, redbugs, or harvest mites, these reddish yellow insects are readily encountered in damp, grassy, and wooded areas, especially along the margins of forests, where they may number in the thousands. Only the tiny (less than 0.2 mm) larval stages are parasitic, feeding on mammals, birds, reptiles, and amphibians. Chiggers are most active in the summer and early autumn. They usually infest humans by crawling up the shoes and legs, preferring to attach to skin at areas where the clothing fits tightly, such as at the top of socks or around the elastic edges of underwear. Chiggers do not burrow into the skin or actively suck blood. Rather, they pierce skin with their mouthparts and secrete a proteolytic salivary fluid that dissolves host tissue, which they then suck up. If undisturbed, chiggers may feed for several days before dropping off. In humans, this rarely occurs, because the larvae usually cause enough irritation that they are dislodged by scratching. The host response to chigger bites is brisk, often leading to intensely pruritic, bright red, 1- to 2-cm nodules. In Asia,



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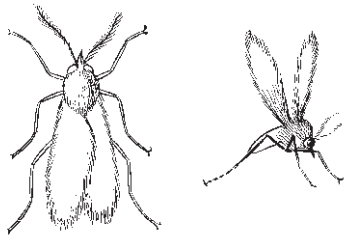

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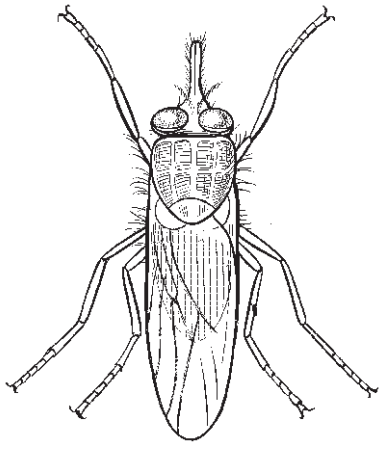

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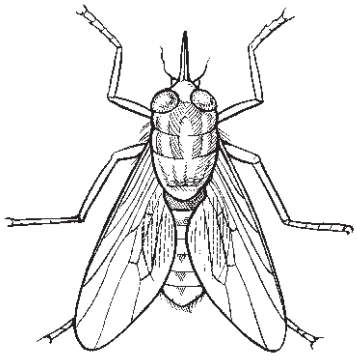

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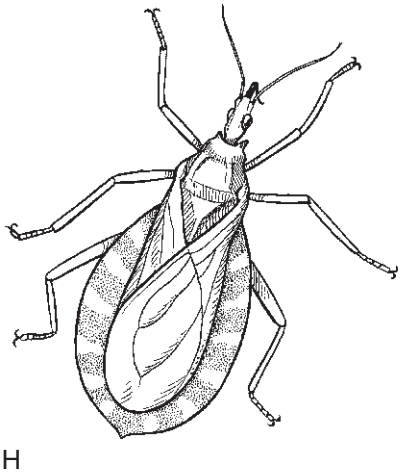

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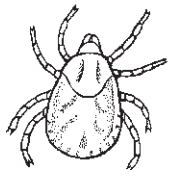

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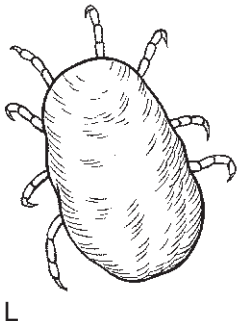

L

Figure 41-1, cont'd. **D,** Tabanid fly. **E,** Sand fly. **F,** Tsetse fly. **G,** Stable fly. **H,** Kissing bug. **I,** Flea. **J,** Chigger mite. **K,** Hard tick. **L,** Soft tick.

chiggers may serve as vectors of scrub typhus. Rickettsial pox is also transmitted by a mite bite.

Ticks (Families Ixodidae and Argasidae)

(See Chapter 33)

Ticks[7,14,29,46,60,125] are classified as hard ticks (family Ixodidae) and soft ticks (family Argasidae) (see Figure 41-1K and L). Hard ticks are so named because of the presence of a sclerotized plate, or scutum, that covers part of the body. Both types of ticks are found worldwide, but hard ticks are more commonly encountered in North America. Hard ticks are usually found in weedy or shrubby areas, along trails, and at forest boundaries, where mammalian hosts, such as deer, are plentiful. Soft ticks are more resistant to desiccation than are hard ticks. Soft ticks thrive in hot and dry climates and are commonly found in animal burrows or caves.

Both sexes are bloodsuckers. Soft ticks are nocturnal and feed rapidly, finishing in just a few minutes. Hard ticks most commonly feed during the day and may feed on a single host for days. Ticks are unable to fly or jump. Hard ticks climb vegetation and "quest," waiting passively for hours or days, forelegs outstretched, until they detect the vibration or carbon dioxide plume of a passing host. When they encounter fur or skin, they climb onto the host and then crawl around in search of an appropriate location on which to attach and feed. The attachment bite is usually painless.

People in suspected tick habitats should check clothing frequently for the presence of ticks. If multiple ticks are seen on clothing, they are most easily removed by trapping them on a piece of cellophane tape, or by rolling a sticky tape–type lint remover across them; hundreds of small ticks can be easily removed by this method. Laundering infested garments cannot be relied on to kill nymphs unless the clothing is subjected to the hot cycle of the dryer.[15] Attached ticks are more difficult to remove. Tick mouthparts are barbed, and some species of tick also secrete a cement that firmly anchors the tick into the skin. Erythema, pruritus, and edema are commonly seen at the site of a tick bite. Improper partial removal of the mouthparts may initiate a long-lasting foreign-body reaction, leading to secondarily infected lesions that are slow to heal, or to granuloma formation that may persist for months. (For a discussion of the best method for tick removal, see Chapter 45.)

After the tick is removed, the bite site should be cleansed with soap and water, or with an antiseptic, and hands should be washed. It may be prudent to save the tick, in case later identification becomes necessary. Laboratory studies of ticks infected with *Borrelia burgdorferi* (the cause of Lyme disease) showed that duration of attachment was directly correlated with the risk of transmission of the spirochete.[98,99,101,102,124,125] Prompt removal of attached ticks (ideally within 24 hours of attachment) greatly reduces the likelihood of disease transmission.[100]

In the United States, soft ticks of the single genus *Ornithodoros* are capable of transmitting to humans the *Borrelia* spirochete that causes relapsing fever. Three genera of the hard ticks Ixodidae transmit disease to man: *Ixodes* (vectors of Lyme disease, babesiosis, and tick paralysis), *Dermacentor* (vectors of tularemia, Rocky Mountain spotted fever, ehrlichiosis, Colorado tick fever, and tick paralysis), and *Amblyomma* (vectors of tularemia, ehrlichiosis, southern tick-associated rash illness (STARI) and tick paralysis).[87,125] Larval, nymph, and adult ticks can all transmit disease during feeding. Transovarial transmission also enables female ticks to directly infect their offspring.

PERSONAL PROTECTION

Personal protection against insect bites can be achieved in three ways: by avoiding infested habitats, by using protective clothing and shelters, and by applying insect repellents.

Habitat Avoidance

Avoiding infested habitats reduces the risk of being bitten. Mosquitoes and other nocturnal bloodsuckers are particularly active at dusk, making this a good time to be indoors. To avoid the usual resting places of biting arthropods, campgrounds should be situated in areas that are high, dry, and open, and as free from vegetation as possible. Areas with standing or stagnant water should be avoided, as these are ideal breeding grounds for mosquitoes. Attempts should be made to avoid unnecessary use of lights, which attract many insects.

Physical Protection

Physical barriers can be extremely effective in preventing insect bites, by blocking arthropods' access to the skin. Long-sleeved shirts, socks, long pants, and a hat will protect all but the face, neck, and hands. Tucking pants into the socks or boots makes it much more difficult for ticks or chigger mites to gain access to the skin. Light-colored clothing is preferable, because it makes it easier to spot ticks, and it is less attractive to mosquitoes and biting flies. Ticks will find it more difficult to cling to smooth, tightly woven fabrics (e.g., nylon).[118] Loose-fitting clothing, made out of tightly woven fabric, with a tucked-in T-shirt undergarment is particularly effective at reducing bites on the upper body. A light-colored, full-brimmed hat will protect the head and neck. Deerflies tend to land on the hat instead of the head; blackflies and biting midges are less likely to crawl to the shaded skin beneath the brim.

Mesh garments or garments made of tightly woven material are available to protect against insect bites. Head nets, hooded jackets, pants, and mittens are available from a number of manufacturers in a wide range of sizes and styles (Box 41-2). Mesh garments are usually made of either polyester or nylon and, depending on the manufacturer, are available in either white or dark colors. With a mesh size of less than 0.3 mm, many of these garments are woven tightly enough to exclude even biting midges and ticks. As with any clothing, bending or crouching may still pull the garments close enough to the skin surface to enable insects to bite through. One manufacturer (Shannon Outdoors, Louisville, GA) addresses this potential problem with a double-layered mesh that reportedly prevents mosquito penetration. Although mesh garments are effective barriers against insects, some people may find them uncomfortable during vigorous activity or in hot weather.

Lightweight insect nets and mesh shelters are available to protect travelers sleeping indoors or in the wilderness (Fig. 41-2). Their effectiveness may be enhanced by treating them with a permethrin-based contact insecticide, which can provide weeks of efficacy after a single application.

Repellents

For many people, applying an insect repellent may be the most effective and easiest way to prevent arthropod bites. Development of the perfect insect repellent has been a scientific goal for

Box 41-2. Manufacturers of Protective Clothing, Protective Shelters, and Insect Nets

PROTECTIVE CLOTHING*

Bug Baffler, Inc.
P.O. Box 444
Goffstown, NH 03045
(800) 662-8411
www.bugbaffler.com

Insect Out
P.O. Box 49643
Colorado Springs, CO 80949
(888) 488-0285
www.insectout.com

BugOut Outdoor Wear, Inc.
P.O. Box 185
Centerville, IA 52544
(877) 928-4688
www.bug-out-outdoorwear.com

Buzz Off Insect Repellent Apparel
(Nonmesh clothing impregnated with permethrin)
701 Green Valley Rd, Suite 302
Greensboro, NC 27408
(336) 272-4157
www.buzzoff.com

The Original Bug Shirt Company
908 Niagara Falls Blvd, #467
North Tonawanda, NY 14120
(800) 998-9096
www.bugshirt.com

Shannon Outdoor's Bug Tamer
1210-A Peachtree St
Louisville, GA 30434
(800) 852-8058
www.bugtamer.com

PROTECTIVE SHELTERS AND INSECT NETS

Long Road Travel Supplies
111 Avenida Dr
Berkeley, CA 94708
(800) 359-6040
www.longroad.com

Travel Medicine, Inc.
369 Pleasant St
Northampton, MA 01060
(800) 872-8633
www.travmed.com

Wisconsin Pharmacal Co.
1 Repel Rd
Jackson, WI 53037
(800) 558-6614
www.pharmacalway.com

*Clothing includes hooded jackets, pants, head nets, ankle guards, gaiters, and mittens.

years and has yet to be achieved. The ideal agent would repel multiple species of biting arthropods, remain effective for at least 8 hours, cause no irritation to skin or mucous membranes, and possess no systemic toxicity, and it would be resistant to abrasion, greaseless, odorless, and not easily washed off. No presently available insect repellent meets all of these criteria. Efforts to find such a compound have been hampered by the many variables that affect the inherent repellency of any chemical. Repellents do not all share a single mode of action, and different species of insects may react differently to the same repellent.[108]

To be effective as an insect repellent, a chemical must be volatile enough to maintain an effective repellent vapor concentration at the skin surface, but it must not evaporate so rapidly that it quickly loses its effectiveness. Multiple factors play a role in effectiveness, including concentration, frequency and uniformity of application, the user's activity level and overall attractiveness to blood-sucking arthropods, and the number and species of the organisms trying to bite. The effectiveness of any repellent is reduced by abrasion from clothing; by evaporation from and absorption into the skin surface; by its tendency to be washed off via sweat, rain, or water; and by a windy environment.[42,62,64,82,83,106] Each 10°C (18°F) increase in ambient temperature can lead to as much as a 50% reduction in protection time.[64] Insect repellents do not cloak the user in a chemical veil of protection; any untreated exposed skin can be readily bitten by hungry arthropods.[83]

Chemical Repellents

DEET. *N*,*N*-diethyl-3-methylbenzamide (previously called *N*,*N*-diethyl-*m*-toluamide), or DEET, remains the gold standard of presently available insect repellents. DEET has been registered for use by the general public since 1957. It is a broad-spectrum repellent, effective against many species of crawling and flying insects, including mosquitoes, biting flies, midges, chiggers, fleas, and ticks. The United States Environmental Protection Agency (EPA) estimates that about 30% of the U.S. population uses a DEET-based product every year; worldwide use exceeds 200 million people annually.[132,134] Decades of empirical testing of more than 20,000 other compounds has not yet led to the release of a superior repellent.[24,58,66,105,111,131]

DEET may be applied directly to skin, clothing, mesh insect nets or shelters, window screens, tents, or sleeping bags. Care should be taken to avoid inadvertent contact with plastics (such as watch crystals and glasses frames), rayon, spandex, leather, or painted and varnished surfaces, as DEET may damage these. It does not damage natural fibers like wool and cotton.

In the United States, DEET is sold in concentrations from 5% to 100%, in multiple formulations, including lotions, solutions, gels, sprays, roll-ons, and impregnated towelettes (Table 41-1). As a general rule, higher concentrations of DEET provide longer-lasting protection. For most uses, however, there is no need to use the highest concentrations of DEET. Products with 10% to 35% DEET provide adequate protection under most conditions. In fact, most manufacturers, responding to consumer demand, have recently begun to offer a greater variety of low-concentration DEET products, and the vast majority of products now contain DEET concentrations of 40% or less. Persons averse to applying DEET directly to their skin may get long-lasting repellency by applying it only to their clothing. DEET-treated garments, stored in a plastic bag between wearings, maintain their repellency for several weeks.[22]

Figure 41-2. **A** to **C,** Protective shelters. **D,** Bed net. (**A, C,** and **D** courtesy Wisconsin Pharmacal Co.; **B** courtesy Long Road Travel Supplies.)

Products with a DEET concentration over 35% are probably best reserved for circumstances in which the wearer will be in an environment with a very high density of insects (e.g., a rain forest), where there is a high risk of disease transmission from insect bites, or when there may be rapid loss of repellent from the skin surface, such as under conditions of high temperature and humidity or rain. Under these circumstances, reapplication of the repellent will most likely be necessary to maintain its effectiveness.

Sequential application of a DEET-based repellent and a sunscreen can reduce the efficacy of the sunscreen. In a study of 14 patients who applied a 33% DEET repellent followed by a sunscreen with a sun protection factor (SPF) of 15, the sunscreen's SPF was decreased by a mean of 33%, although the repellent maintained its potency.[91] Some products contain a combination of sunscreen and DEET and will deliver the SPF stated on the label. However, these products are generally not the best choice, as it is rare that the need for reapplication of sunscreen and repellent is exactly the same.

Two companies (3M, Minneapolis, MN, and Sawyer Products, Tampa, FL) currently manufacture extended-release formulations of DEET that make it possible to deliver long-lasting protection without relying on high concentrations. The 3M product Ultrathon was originally developed for the U.S. military but is also available to the general public. This acrylate polymer formulation containing 35% DEET, when tested under many different environmental and climatic field conditions, was as effective as 75% DEET, providing up to 12 hours of greater than 95% protection against mosquito bites.[2,49,67,88,114,116] Sawyer Products' controlled-release 20% DEET lotion traps the chemical in a protein particle that slowly releases it to the skin surface, providing repellency equivalent to a standard 50%

TABLE 41-1. DEET-Containing Insect Repellents

MANUFACTURER	PRODUCT NAME	FORM	DEET (%)
Sawyer Products Tampa, FL (800) 940-4664	Sawyer Controlled Release	Lotion	20
	Sawyer Broad Spectrum Insect Repellent (with R-326 fly and gnat repellent)	Aerosol and pump spray	25
	Sawyer Maxi DEET	Pump spray	100
S.C. Johnson Wax Racine, WI (800) 558-5566	OFF! Skintastic	Pump spray	5
	OFF! Skintastic	Pump spray	7
	OFF! Skintastic with Sunscreen (SPF 30)	Pump spray	10
	OFF! Fresh Scent	Pump spray	25
	Deep Woods OFF!	Aerosol and pump spray	25
	Deep Woods OFF! Sportsman	Aerosol spray	30
	DEEP Woods OFF! Sportsman	Pump spray	100
Tender Corp. Littleton, NH (800) 258-4696	Ben's Tick and Insect Repellent	Aerosol, pump spray, roll-on	30
	Ben's 100 Tick and Insect Repellent	Pump spray	95
Spectrum Brands St. Louis, MO (800) 874-8892	Cutter All Family Insect Repellent	Aerosol, pump spray, wipes	7
	Cutter Skinsations Insect Repellent	Pump spray	7
	Cutter Unscented	Aerosol spray	10
	Cutter Backwoods	Aerosol and pump spray	23
	Cutter Backwoods	Wipes	30
	Cutter Outdoorsman	Pump	23
	Cutter Outdoorsman	Aerosol	28.5
	Cutter Oudoorsman	Stick and lotion	30
	Cutter Tick Defense (with MGK 264 and 326)	Aerosol spray	25
	Cutter Max	Pump	100
	Repel Camp Lotion for Families	Lotion	10
	Repel Sun and Bug Stuff (SPF 15)	Lotion	20
	Repel Family Formula	Aerosol spray	23
	Repel Sportsman Formula	Lotion	20
	Repel Sportsman Formula	Pump spray	25
	Repel Sportsman Formula	Aerosol spray	29
	Repel Sportsman Formula	Wipes	30
	Repel Sportsman Max Formula	Aerosol spray	40
	Repel Hunter's Repellent with Earth Scent	Pump spray	55
	Repel 100% Insect Repellent	Pump spray	100
3M St. Paul, MN (888) 364-3577	Ultrathon	Aerosol	25
	Ultrathon	Lotion	35

DEET preparation and lasting about 5 hours.[34] Compared with a 20% ethanol-based preparation of DEET, 60% less of this encapsulated DEET is absorbed.[20]

DEET has been used by millions of people worldwide for 50 years, and it continues to show a remarkable safety profile. In 1980, as part of the U.S. EPA Registration Standard for DEET,[132] over 30 additional animal studies were conducted to assess acute, chronic, and subchronic toxicity; mutagenicity; oncogenicity; and developmental, reproductive, and neurologic toxicity. The results of these studies neither led to any product changes to comply with current EPA safety standards nor indicated any new toxicities under normal usage. The EPA's Reregistration Eligibility Decision (RED),[134] released in 1998, confirmed that the Agency believes that "normal use of DEET does not present a health concern to the general U.S. population."

Case reports of potential DEET toxicity exist in the medical literature and have been extensively reviewed.[8,38,127,136] Fewer than 50 cases of significant toxicity from DEET exposure have been documented in the medical literature over the last 4 decades; over three quarters of these resolved without sequelae. Many of these cases involved long-term, excessive, or inappropriate use of DEET repellents; the details of exposure were frequently poorly documented, making causal relationships difficult to establish. These cases have not shown a correlation between the concentration of the DEET product used and the risk of toxicity.

The reports of DEET toxicity that raise the greatest concern involve 18 cases of encephalopathy, 14 in children under age 8 years.* Three of these children died; one of them had ornithine carbamoyl transferase deficiency, which might have predisposed her to DEET-induced toxicity.[55,56] The other children recovered without sequelae. The EPA's analysis of these cases concluded that they "do not support a direct link between exposure to

*See references 12, 28, 31, 38, 52, 55, 56, 71, 79, 93, 94, 142.

DEET and seizure incidence."[134] Animal studies in rats and mice show that DEET is not a selective neurotoxin.[93,109,132] Even if a link between DEET use and seizures does exist, the observed risk, based on DEET usage patterns, would be less than one per 100 million users.[134] Other studies have confirmed that children are not at greater risk for developing adverse effects from DEET when compared with older individuals.[8,69,136]

Very limited studies have been done investigating the safety of DEET use during pregnancy. One published study followed 450 Thai women who used 20% DEET daily during the second and third trimesters of pregnancy to reduce the risk of contracting malaria.[86] Of these women, 4% had detectable levels of DEET in umbilical cord blood at the time of delivery. However, no differences in survival, growth, or neurologic development could be detected in the infants born to mothers who used DEET when compared with an equal number of mothers treated with a daily placebo cream during their pregnancies.

The EPA has issued guidelines to ensure safe use of DEET-based repellents[134] (Box 41-3). Careful product choice and common-sense application greatly reduce the possibility of toxicity. The current recommendation of the American Academy of Pediatrics is that children over the age of 2 months can safely use up to 30% DEET.[138] When required, reapplication of a low-strength repellent can compensate for the inherent shorter duration of protection.

Questions about the safety of DEET may be addressed to the EPA-sponsored National Pesticide Information Center, available every day from 6:30 AM to 4:30 PM PST at 800-858-7378, or via their website at http://npic.orst.edu.

Box 41-3. Guidelines for Safe and Effective Use of Insect Repellents

- For casual use, choose a repellent with 10% to 35% DEET. Repellents with 10% DEET or less are most appropriate for use on children.
- Use just enough repellent to lightly cover the exposed skin; do not saturate the skin.
- Repellents should be applied only to exposed skin and clothing. Do not use under clothing.
- To apply to the face, dispense into palms, rub hands together, and then apply thin layer to face.
- Young children should not apply repellents themselves.
- Avoid contact with eyes and mouth. Do not apply to children's hands, to prevent possible subsequent contact with mucous membranes.
- After applying, wipe repellent from the palmar surfaces to prevent inadvertent contact with eyes, mouth, and genitals.
- Never use repellents over cuts, wounds, or inflamed, irritated, or eczematous skin.
- Do not inhale aerosol formulations or get them in eyes. Do not apply when near food.
- Frequent reapplication is rarely necessary, unless the repellent seems to have lost its effectiveness. Reapplication may be necessary in very hot, wet environments because of rapid loss of repellent from the skin surface.
- Once inside, wash treated areas with soap and water. Washing the repellent from the skin surface is particularly important when a repellent is likely to be applied for several consecutive days.
- If you suspect you are having a reaction to an insect repellent, discontinue its use, wash the treated skin, and consult a physician.

Adapted from United States Environmental Protection Agency, Office of Pesticide Programs, Prevention, Pesticides and Toxic Substances Division: Reregistration Eligibility Decision (RED): DEET (EPA-738-F-95-010), Washington, DC, 1998, EPA.

IR3535 (Ethyl-Butylacetylaminoproprionate). IR3535 is an analog of the amino acid β-alanine and has been sold in Europe as an insect repellent for 20 years. In the United States, this compound is classified by the EPA as a biopesticide, effective against mosquitoes, ticks, and flies. IR3535 was brought to the U.S. market in 1999, sold exclusively by the Avon Corporation as Skin-So-Soft Bug Guard Plus, with 7.5% IR3535. Depending on the species of mosquito and the testing method, this repellent has demonstrated widely variable effectiveness, with complete protection times ranging from 23 to 360 minutes.[5,6,39] In general, IR3535 provides longer-lasting repellency than the botanical citronella-based repellents, but it does not match the overall efficacy of DEET.[5,6,39] In 2006, Avon also released a 15% IR3535 spray to the market.

Avon's Skin-So-Soft Bath Oil (Avon, New York, NY) received considerable media attention several years ago when it was reported by some consumers to be effective as a mosquito repellent. When tested under laboratory conditions against *Aedes aegypti* mosquitoes, Skin-So-Soft Bath Oil's effective half-life was found to be 0.51 hours.[107] In one study against *Aedes albopictus*, Skin-So-Soft oil provided 0.64 hours of protection from bites, and it was 10 times less effective than was 12.5% DEET.[116] Skin-So-Soft oil has been found to be somewhat effective against biting midges, but this effect is felt to be a result of its trapping the insects in an oily film on the skin surface.[81] It has been proposed that the limited mosquito repellent effect of Skin-So-Soft oil could result from its fragrance, or from the presence of diisopropyl adipate and benzophenone in the formulation, both of which have some repellent activity.[17,66]

Picaridin. The piperidine derivative picaridin (also known as KBR2023) is the newest insect repellent to become available in the United States. Picaridin-based insect repellents have been sold in Europe since 1998 under the brand name Bayrepel. In 2005, the first picaridin-based repellent was brought to the market in the United States, as Cutter Advanced, containing 7% of the active ingredient. This nearly odorless, nongreasy repellent is effective against mosquitoes, biting flies, and ticks. The 7% repellent should provide protection for up to 4 hours. Studies have shown that, when used at higher concentrations of up to 20%, picaridin repellents can offer an efficacy comparable to that of DEET,[3,26,27,40,123] giving up to 8 hours of protection. The chemical is aesthetically pleasant and, unlike DEET, shows no detrimental effects on contact with plastics. The EPA found picaridin to have a very low toxicity risk. In April 2005, the Centers for Disease Control and Prevention (CDC) released a statement adding picaridin to the list of approved repellents that could be used effectively to prevent mosquito-borne diseases.

TABLE 41-2. Botanical Insect Repellents

MANUFACTURER	PRODUCT NAME	FORM	ACTIVE INGREDIENTS
HOMS, LLC Clayton, NC (800) 805-2483	Bite Blocker	Lotion, pump spray	Soybean oil 2%
Tender Corp. Littleton, NH (800) 258-4696	Natrapel	Lotion, pump spray, roll-on, wipes	Citronella oil 10%
All Terrain Co. Encinitas, CA (800) 246-7328	Herbal Armor	Pump spray and lotion	Citronella oil 12%, peppermint oil 2.5%, cedar oil 2%, lemongrass oil 1%, geranium oil 0.05%, in a slow-release encapsulated formula
	Herbal Armor Bug & Sun SPF15	Lotion	
	Kids Herbal Armor	Pump spray	
	Kids Herbal Armor SPF 15	Lotion	
Green Ban Norway, IA (319) 446-7495	Green Ban for People:		
	Regular	Oil	Citronella oil 5%, peppermint oil 1%
	Double Strength	Oil	Citronella oil 10%, peppermint oil 2%
Quantum Inc. Eugene, OR (800) 448-1448	Buzz Away	Towelettes, pump spray	Citronella oil 5%; oils of cedarwood, peppermint, eucalyptus, lemongrass
	Buzz Away, SPF 15	Lotion	
Kiss My Face Gardiner, NY (800) 262-5477	SunSwat SPF 15	Lotion	Citronella oil, bay, cedarwood, lavender, vetivert, patchouli, juniper, tea tree, lemon peel, pennyroyal, pansy, goldenseal oils
Lakon Herbals, Inc. Montpelier, VT (800) 865-2566	Bygone Bugzz	Lotion	Eucalyptus, rosemary, birch, peppermint and geranium oils

Botanical Repellents

Thousands of plants have been tested as sources of insect repellents. Although none of the plant-derived chemicals tested to date demonstrates the broad effectiveness and duration of DEET, a few show repellent activity. Plants with essential oils that have been reported to possess repellent activity include citronella, neem, cedar, verbena, pennyroyal, geranium, catnip, lavender, pine, cajeput, cinnamon, vanilla, rosemary, basil, thyme, allspice, garlic, and peppermint.* Unlike DEET-based repellents, botanical repellents have been relatively poorly studied. When tested, most of these essential oils tended to show short-lasting protection, lasting minutes to 2 hours.

A summary of readily available plant-derived insect repellents is shown in Table 41-2.

Citronella. Oil of citronella was initially registered as an insect repellent by the EPA in 1948. It is the most common active ingredient found in "natural" or "herbal" insect repellents presently marketed in the United States. Originally extracted from the grass plant *Cymbopogon nardus*, oil of citronella has a lemony scent.

Conflicting data exist on the efficacy of citronella-based products, varying greatly depending on the study methodology, location, and species of biting insect tested. One citronella-based repellent was found to provide no repellency when tested in the laboratory against *Aedes aegypti* mosquitoes.[17] However, another study of the same product conducted in the field showed an average of 88% repellency over a 2-hour exposure. The product's effectiveness was greatest within the first 40 minutes after application and then decreased with time over the remainder of the test period.[128] All Terrain Company (Encinitas, CA; 800-246-7328) produces a citronella-based lotion in which the essential oil has been encapsulated into a beeswax matrix, which slowly releases it to the skin surface, prolonging its efficacy. In laboratory testing against *Aedes aegypti*, this product provided complete protection for the first 2 hours, and 77% protection 4 hours after application.[53]

In general, studies show that citronella-based repellents are less effective than are DEET repellents. Citronella provides a shorter protection time, which may be partially overcome by frequent reapplication of the repellent. In 1997, after analyzing available data on the repellent effect of citronella, the EPA concluded that citronella-based insect repellents must contain the following statement on their labels: "For maximum repellent effectiveness of this product, repeat applications at one hour intervals."[133]

Citronella candles have been promoted as an effective way to repel mosquitoes from one's local environment. One study compared the efficacy of commercially available 3% citronella candles, 5% citronella incense, and plain candles to prevent bites by *Aedes* species mosquitoes under field conditions.[77] Subjects near the citronella candles had 42% fewer bites than controls who had no protection (a statistically significant difference). However, burning ordinary candles reduced the number of bites by 23%. There was no difference in efficacy between citronella incense and plain candles. The ability of

*See references 4, 13, 22, 30, 48, 63, 66, 97, 104, 130.

plain candles to decrease biting may be due to their serving as a decoy source of warmth, moisture, and carbon dioxide.

The citrosa plant (*Pelargonium citrosum* "Van Leenii") has been marketed as being able to repel mosquitoes through the continuous release of citronella oils. Unfortunately, when tested, these plants offer no protection against bites.[18,85]

Bite Blocker. Bite Blocker is a "natural" repellent that was released to the United States market in 1997. It combines soybean oil, geranium oil, and coconut oil in a formulation that has been available in Europe for several years.[135] Studies conducted at the University of Guelph showed that this product was capable of providing over 97% protection against *Aedes* species mosquitoes under field conditions, even after 3.5 hours of application. During the same period, a 6.65% DEET-based spray afforded 86% protection, whereas Avon's Skin-So-Soft citronella-based repellent gave only 40% protection.[74] A second study showed that Bite Blocker provided a mean of 200 ± 30 (SD) minutes of complete protection from mosquito bites.[75] A laboratory study using three different species of mosquitoes showed that Bite Blocker provided an average protection time of about 7 hours.[6] Another study showed that it could give about 10 hours of protection against biting black flies; in the same test, 20% DEET protected for only about 6.5 hours.[76]

Eucalyptus. A derivative (*p*-menthane-3,8-diol, or PMD) isolated from oil of the lemon eucalyptus plant has a strong lemony scent and has shown promise as an effective "natural" repellent.[23] This repellent has been very popular in China for years and is currently sold in Europe as Mosi-Guard and in the United States as Repel Lemon Eucalyptus Repellent (Wisconsin Pharmacal Co., Inc., Jackson, WI) and as FiteBite Plant-Based Insect Repellent (Travel Medicine, Inc., Northampton, MA). Field tests of this repellent have shown mean complete protection times ranging from 4 to 7.5 hours, depending on the mosquito species.[6,47,89] PMD-based repellents can cause significant ocular irritation, so care must be taken to keep them away from the eyes. In 2005, the CDC added this repellent to the approved list of products that can be effectively used to prevent mosquito-borne diseases.

Efficacy of DEET versus Botanical Repellents

Limited data are available from studies that directly compare plant-derived repellents to DEET-based products. Available data proving the efficacy of "natural" repellents are often sparse, and there is no uniformly accepted standard for testing these products. As a result, different studies often yield varied results, depending on how and where the tests were conducted.

Studies comparing "natural" repellents to low-strength DEET products, conducted under carefully controlled laboratory conditions with caged mosquitoes, typically demonstrate dramatic differences in effectiveness between currently marketed insect repellents.[39] Citronella-based insect repellents usually provide the shortest-duration protection, often lasting only a few minutes. Low-concentration DEET lotions (under 7%) typically prove to be more effective than citronella-based repellents in their ability to prevent mosquito bites and can generally be expected to provide about 1.5 to 2 hours of complete protection.[39,54] Reapplication of these low-concentration DEET products can compensate for their shorter duration of action. Because DEET repellents show a clear dose–response relationship, higher concentrations of DEET can be used to provide proportionately longer complete protection times—up to 6 to 8 hours after a single application. Bite Blocker and oil of eucalyptus repellents appear to be the best of the botanical repellents. Because of its pleasant aesthetic qualities, picaridin repellents may eventually replace DEET as the consumer's preferred repellent.

Alternative Repellents

There has always been great interest in finding an oral insect repellent. Oral repellents would be convenient and would eliminate the need to apply creams to the skin or put on protective clothing. Unfortunately, no effective oral repellent has been discovered. For decades, lay literature has made the claim that vitamin B_1 (thiamine) works as a systemic mosquito repellent. When subjected to scientific scrutiny, however, thiamine has not been found to have any repellent effect on mosquitoes.[65,139] The U.S. Food and Drug Administration (FDA), prompted by misleading consumer advertising, issued the following statement about thiamine in 1983: "There is a lack of adequate data to establish the effectiveness of this, or any other ingredient for OTC [over the counter] oral use as an insect repellent. Labeling claims for OTC orally administered insect repellent drug products are either false, misleading, or unsupported by scientific data."[35] Tests of over 100 ingested drugs, including other vitamins, failed to reveal any that worked well against mosquitoes.[126] Ingested garlic has also never proven to be an effective deterrent.

Insecticides

Permethrin

Pyrethrum is a powerful, rapidly acting insecticide, originally derived from crushed dried flowers of the daisy *Chrysanthemum cinerariifolium*.[16] Permethrin is a synthetic pyrethroid. It does not repel insects but works as a contact insecticide, causing nervous system toxicity, leading to death, or "knockdown," of the insect. The chemical is effective against mosquitoes, flies, ticks, fleas, lice, and chiggers. Permethrin has low mammalian toxicity, is poorly absorbed by the skin, and is rapidly metabolized by skin and blood esterases.[57,141]

Permethrin should be applied directly to clothing or to other fabrics (tent walls[110] or mosquito nets[78]), not to skin. Permethrins are nonstaining, are nearly odorless, are resistant to degradation by heat or sun, and maintain their effectiveness for at least 2 weeks, and through several launderings.[112,117]

The combination of permethrin-treated clothing and skin application of a DEET-based repellent creates a formidable barrier against biting insects.[50,67,121] In an Alaskan field trial against mosquitoes, subjects wearing permethrin-treated uniforms and a polymer-based 35% DEET product had greater than 99.9% protection (one bite per hour) over 8 hours; unprotected subjects received an average of 1188 bites per hour.[73]

Permethrin-sprayed clothing also proved very effective against ticks. One hundred percent of *Dermacentor occidentalis* ticks (which carry Rocky Mountain spotted fever) died within 3 hours of touching permethrin-treated cloth.[70] Permethrin-sprayed pants and jackets also provided 100% protection from all three life stages of *Ixodes dammini* ticks, the vector of Lyme disease.[118] In contrast, DEET alone (applied to the skin), provided 85% repellency at the time of application; this protection deteriorated to 55% repellency at 6 hours, when tested against the lone star tick *Amblyomma americanum*.[123] *Ixodes scapu-*

TABLE 41-3. Permethrin Insecticides

MANUFACTURER	PRODUCT NAME	FORM	ACTIVE INGREDIENT
Coulston Products Easton, PA (610) 253-0167	Duranon Odorless Perma-Kill	Aerosol spray Liquid concentrate	Permethrin 0.5% Permethrin 13.3%
Sawyer Products Tampa, FL (800) 940-4464	Permethrin Clothing Insect Repellent	Aerosol and pump sprays	Permethrin 0.5%
Spectrum Brands St. Louis, MO (800) 874-8892	Repel Permanone Clothing and Gear Insect Repellent	Aerosol spray	Permethrin 0.5%
3M St. Paul, MN (888) 364-3577	Clothing and Gear Insect Repellent	Aerosol spray	Permethrin 0.5%

laris ticks, which may transmit Lyme disease, also seem to be less sensitive to the repellent effect of DEET.[113]

Permethrin-based insecticides available in the United States are listed in Table 41-3. To apply to clothing, spray each side of the fabric (outdoors) for 30 to 45 seconds, just enough to moisten it. Allow it to dry for 2 to 4 hours before wearing it. Permethrin solution is also available for soak-treating large items, such as mesh bed nets, or for treating multiple garments simultaneously. Permethrin-pretreated shirts, pants, socks, and hats can also be purchased. The manufacturer (Buzz Off Insect Shield, Greensboro, NC) claims that they will maintain their insecticidal effect through 25 machine washings.

Reducing Local Mosquito Populations

Consumers may still find advertisements for small ultrasonic electronic devices that are meant to be carried on the body and claim to repulse mosquitoes by emitting "repellent" sounds, such as that of a dragonfly (claimed to be the natural enemy of the mosquito), male mosquito, or bat. Multiple studies, conducted in the field and in the laboratory, show that these devices do not work.[9,21,37,59,72] Mass-marketed backyard bug zappers, which use ultraviolet light to lure and electrocute insects, are also ineffective: mosquitoes continue to be more attracted to humans than to the devices.[92] One backyard study showed that of the insects killed by these devices, only 0.13% were female (biting) mosquitoes.[41] An estimated 71 to 350 billion beneficial insects may be killed annually in the United States by these devices.[41] Newer technology, utilizing more specific bait, such as a warm, moist plume of carbon dioxide, as well as other known chemical attractants (e.g., octenol), may prove to be a more successful way to lure and selectively kill biting insects. Their manufacturers accurately claim that these machines can lure and kill thousands of mosquitoes, but it remains to be proven that an individual unit can actually kill enough mosquitoes to reduce the local biting pressures. Pyrethrin-containing yard foggers set off prior to an outdoor event can temporarily reduce the number of biting arthropods in a local environment. These products should be applied before any food is brought outside and should be kept away from animals or fish ponds. Burning coils that contain natural pyrethrins or synthetic pyrethroids (such as D-allethrin or D-*trans*-allethrin) can also temporarily reduce local populations of biting insects.[59,80,140] Some concerns have been raised about the cumulative safety of long-term use of these coils in an indoor environment.[1,96] Wood smoke from campfires can also reduce the likelihood of being bitten by mosquitoes. The smoke's ability to repel insects may vary depending on the type of wood or vegetation burned.[95,137]

Integrated Approach to Personal Protection

An integrated approach to personal protection is the most effective way to prevent arthropod bites, regardless of where one is in the world and which species of insects may be attacking. Maximal protection is best achieved through avoiding infested habitats and using protective clothing, topical insect repellents and permethrin-treated garments. When appropriate, mesh bed nets or tents should be used to prevent nocturnal insect bites.

DEET-containing insect repellents are the most effective products currently on the U.S. market, providing broad-spectrum, long-lasting repellency against multiple arthropod species. Insect repellents alone, however, should not be relied on to provide complete protection. Mosquitoes, for example, can find and bite any untreated skin and may even bite through thin clothing. Deerflies, biting midges, and some blackflies prefer to bite around the head and readily crawl into the hair to bite where there is no protection. Wearing protective clothing, including a hat, reduces the chances of being bitten. Treating clothes, including the hat, with permethrin maximizes their effectiveness by causing knockdown of any insect that crawls or lands on the treated clothing. To prevent chiggers or ticks from crawling up the legs, pants should be tucked into the boots or stockings.

The U.S. military relies on this integrated approach to protect troops deployed in areas where arthropods constitute either a significant nuisance or medical risk. The Department of Defense's insect repellent system consists of DEET applied to exposed areas of skin, and permethrin-treated uniforms, worn with the pant legs tucked into boots and the undershirt tucked into the pant's waistband. This system has been proven to dramatically reduce the likelihood of being bitten by insects.

Persons traveling to parts of the world where insect-borne disease is a potential threat can protect themselves best if they learn about indigenous insects and the diseases they might transmit. Protective clothing, mesh insect tents or bedding, insect repellent, and permethrin spray should be carried. Travelers would be wise to check the most current CDC recommenda-

tions about traveling to countries where immunizations (e.g., against yellow fever) or antibiotic prophylaxis (e.g., against malaria) should be undertaken before departure. The CDC maintains these recommendations on its website at www.cdc.gov/travel/index.htm, or by telephone at 888-232-3228. An excellent summary of information on issues related to travel health can also be found at www.tripprep.com. This website culls information daily from the CDC, the *Morbidity and Mortality Weekly Report*, the World Health Organization, and the U.S. State Department.

The references for this chapter can be found on the accompanying DVD-ROM.

42 Mosquitoes and Mosquito-Borne Diseases

Renee Y. Hsia

Mosquitoes are in the biologic phylum Arthropoda, class Insecta, order Diptera, and family Culicidae. There are approximately 35 genera, which include *Anopheles, Culex, Psorophora, Ochlerotatus, Aedes, Sabethes, Wyeomyia, Culiseta,* and *Haemagogus*. Worldwide, there are over 3000 species and subspecies, and in the United States alone, there are an estimated 200 different species of mosquitoes that differ in their habitats and behaviors.[81] In addition to transmitting the parasitic disease of malaria to 300 to 500 million people per year and thereby killing up to 2.7 million people per year,[1] mosquitoes also serve as vectors for arboviruses that cause significant morbidity and mortality to humans worldwide. In addition, mosquitoes are responsible for transmission of the larval nematode that leads to lymphatic filariasis.

MOSQUITOES

Mosquitoes (the word is derived from the 16th century Spanish or Portuguese word for *little fly*) have been in existence for an estimated 170 million years. They are characterized by scaled wings, long legs, and a slender body. The size varies but rarely exceeds 15 mm in length. They weigh approximately 2 to 2.5 mg and can fly at about 0.9 to 1.6 mph.

Mosquito Anatomy

Mosquito anatomy (Fig. 42-1) includes a slender body with two wings (rather than the four possessed by flies) and six long, delicate legs. The wings are covered with scales. The body is divided into three parts: head, thorax, and abdomen. The head has two large compound eyes with many lenses angled in different directions, and two antennae. The mouth, or proboscis, looks like a funnel extending downward, and it is used to pierce skin and suck blood (for females) or sip nectar (for males). The thin and short neck connects the head to the thorax, which is triangular and holds the wings in place. The thorax also has two pairs of spiracles, which are tubes through which the mosquito can breathe. The shape of the abdomen can be pointed or rounded, depending on the species, and it has eight pairs of spiracles. The legs are divided into the coxa, femur, tibia, and tarsus.

The spiracles form the basis of the mosquito's tracheal system, which is finely branched so that the cells of the body are directly oxygenated. Mosquitoes have a dorsal blood vessel that extends directly from the eighth abdominal segment into the head. The heart is the portion of the blood vessel located within the thorax and although it is not innervated, it pulses automatically at a rate of 85 beats per minute.[74]

The mouthparts of the mosquito include the labrum and the labium, which can be understood as the upper and lower lips of the mosquito, respectively, and the mandible and maxilla are anterior and posterior structures that compose a jawlike configuration. The maxillary palps are small, antenna-like sensory structures that detect chemicals from animals, and the sensory hairs (setae) on the antennae sense vibration and motion from potential prey and other organisms.

Mosquito Life Cycle

The life cycle of most species of mosquitoes is defined by four stages: egg, larva, pupa, and adult. The time a mosquito spends in each stage depends on the species and the temperature. *Culex tarsalis,* for example, has a 14-day life cycle at 20° C (68° F), and a 10-day life cycle at 25° C (77° F). The life cycle varies from 4 days to 1 month, but it averages around 2 weeks, which means that several generations can arise within a single year.[81] Most mosquitoes that do not have habitats in the tropics lie dormant as eggs, although some, such as the genus *Culex,* overwinter as larvae or adults.

The female mosquito usually mates once in her lifetime and stores sperm in her body. She fertilizes her eggs when she lays them, which is generally on the surface of the water—often in the form of egg rafts, as shown in Figure 42-2, or sometimes in groups in empty containers or jars, as shown in Figure 42-3. Eggs can survive winter and hatch in the spring. They hatch into larvae (also known as wrigglers), about 1 to 2 cm long, that feed on microscopic animal, plant life, and, in some cases, other

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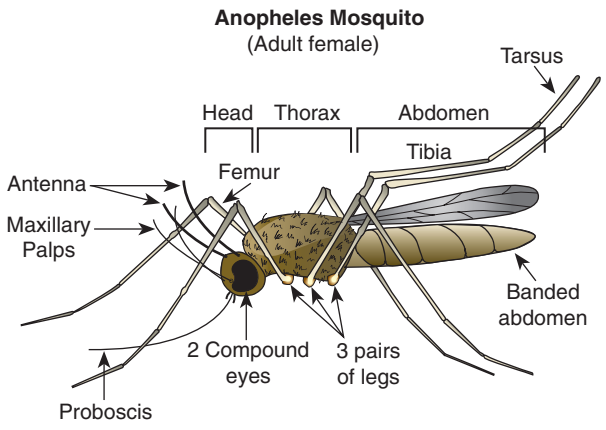

Figure 42-1. The anatomy of a mosquito.

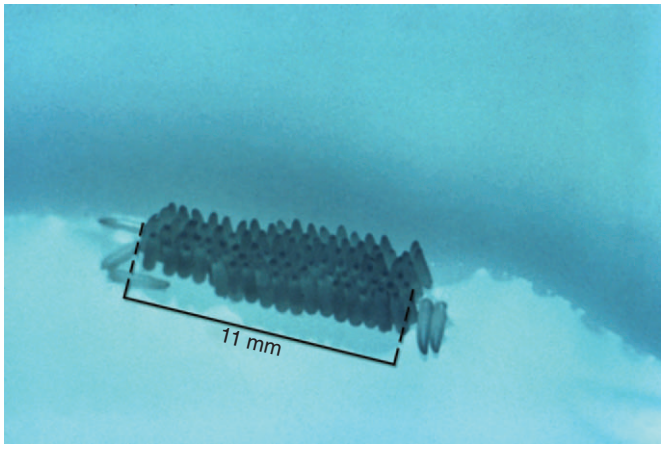

Figure 42-2. A *Culex* mosquito "egg raft," measuring 11 mm in length and composed of 200 to 300 eggs.

Figure 42-3. Eggs of *Aedes aegypti* mosquito, which transmits dengue virus.

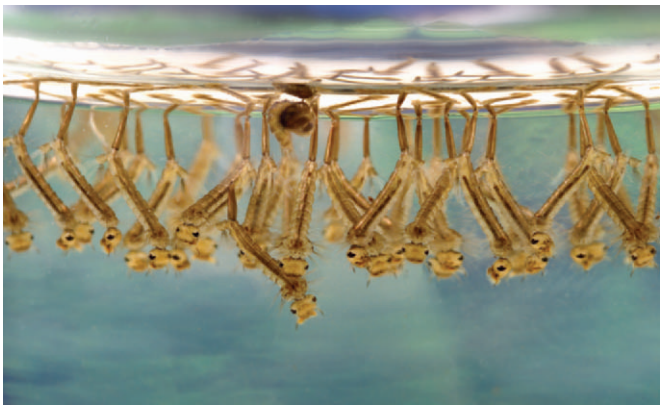

Figure 42-4. *Culex* larvae suspended diagonally, with siphons at the surface of the water.

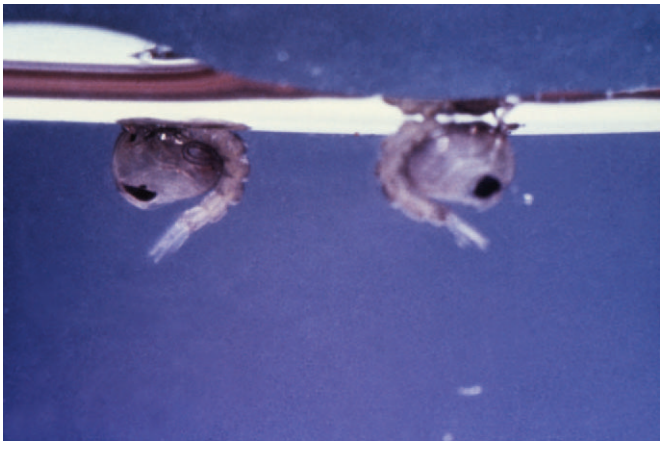

Figure 42-5. Pupae, where the eyes, legs, and wings are visible.

mosquito larvae. Except for the genus *Anopheles*, the larvae must come to the surface to employ their air tubes, or siphons, which supplement their gills (Fig. 42-4). After molting several times, they develop into pupae (Fig. 42-5), floating on the surface of the water and breathing through an air tube, now called a trumpet. While encased in the hard pupal case, they transform into adult mosquitoes. Enough pressure is created to burst the case, and when their wings harden, they can fly and live on land.[74]

Mechanism of Mosquito Bites

Mosquitoes technically do not sting, because there is no stinger; they simply pierce the skin and suck blood. Only the females of most species have sucking mouth parts that can pierce skin and thus transmit disease (Fig. 42-6). The blood protein is required because the normal mosquito diet of nectar and fruit juices does not contain enough protein for egg development. (One genus, *Toxorhynchites*, does not suck blood, and its larvae prey on other mosquito larvae.) Mosquitoes generally identify victims by scent, as well as by the carbon dioxide of exhaled breath and some chemicals found in sweat. Mosquitoes have been shown to have odorant-binding proteins on their antennae that bind

Figure 42-6. A female *Aedes albopictus* mosquito (a vector of the West Nile virus) becoming engorged with blood while feeding on a human.

Figure 42-7. Urticarial plaque.

Figure 42-8. Mosquito bite demonstrating papular urticaria.

human-specific odorants, stimulating their olfactory neurons and allowing them to locate their human prey.[71] Other risk factors for getting bitten include male sex, heavier weight, and type O blood.[30]

When mosquitoes pierce human skin, all six legs are usually positioned on the skin surface, along with the labella (part of the proboscis). The female proboscis is made up of six shafts, four of which cut and pierce skin. One serves as a conduit for blood into the mosquito, and another transports mosquito saliva into the skin. The mosquito lays the labella on the skin, which allows the maxillae of the fascicle (a bundle of feeding stylets, contained within the proboscis) to saw into the skin in an oscillatory cutting fashion. Blood is drawn up into the fascicle, which collects into the labrum held between the mandibles of the mosquito. The female mosquito usually requires about 50 seconds to insert the fascicle into the skin, and approximately 2 minutes to finish feeding. She can withdraw her fascicle in 5 seconds.[29,35]

The saliva of mosquitoes contains a wide array of antihemostatic molecules that allow feeding of blood. These molecules include vasodilators, such as amines and prostaglandins; platelet aggregation inhibitors, such as nitric oxide, prostaglandins, apyrase, molecules that sequester ADP-sequestering molecules, and peptides and proteins specifically targeted to integrin receptors; and anticoagulants, such as thrombin and factor Xa inhibitors. The saliva and its accompanying proteins (specifically, the 33-kilodalton F-1 protein found in mosquito saliva) that remain in human skin after feeding serve as antigens that evoke an immune response.[25]

Pathophysiology and Clinical Manifestations of Mosquito Bites

Individuals vary in their response to a mosquito bite. Almost all experience local irritation caused by a type I immunoglobulin E (IgE)-mediated response of a soft, pale, pruritic wheal or plaque (Fig. 42-7). The subsequent effects reveal which immune response a person has to the bite. Type I IgE-responses can also create immediate hivelike skin lesions (rare), whereas type IV cell-mediated immunity causes delayed pruritic papules or vesicles within 48 hours of the bite. Other type IV reactions caused by mosquito bites include blisters, bullae, and erythema multiforme or purpura, but these are rare.

One common type IV hypersensitivity response is papular urticaria, which describes an eruption of pruritic papules measuring 3 to 10 mm in diameter that may be surrounded by vesicles grouped in irregular clusters (Fig. 42-8). Because repeated exposures may lead to desensitization in adults, these lesions are more common in children. They begin as an erythematous wheal, lasting from 2 to 10 days, and may lead to temporary hyperpigmentation. Children who suffer this hypersensitivity

may develop a firm, light brown papule, whereas most adults have only a transient wheal without formation of a persistent papule. The dermatopathology of the epidermis in papular urticaria shows spongiosis with exocytosis and vesicle formation. The upper dermal layer reveals a localized perivascular infiltrate with lymphocytes, histiocytes, eosinophils, and mast cells, and a spattering of eosinophils and mast cells in the mid-dermis.[77]

The pathophysiology behind the phenomenon of itching is complex. There are abundant theories on the neurophysiologic basis of itch, and the most traditional is that local reaction induces the production of histamine, which stimulates C nerve fibers that travel to the brain and induce a sense of itching.[90] Histamine, however, is only one of many pruritogenic substances that stimulate a subset of specialized skin C-fibers. Other mediators of itch include proteases, opioids, lipid peroxidation metabolites (such as leukotrienes and prostaglandins), neuropeptides (e.g., substance P), cytokines, and growth factors (e.g., nerve growth factor). Pruritus is now seen as an interdisciplinary issue that reaches into the fields of neurophysiology, neuroimmunology, neuropharmacology, protease research, internal medicine, and dermatology.

In general, mosquitoes confer disease on humans by carrying and harboring viruses that have animal reservoirs. Different viruses have different transmission patterns, some with avian reservoirs and others with nonhuman primate reservoirs (and sometimes even humans). The geographic distribution of mosquitoes and changes in their habitats affect the distribution of disease as well. Although mosquitoes are often thought of as living only in temperate and tropical areas, their geographic distribution extends to climatic extremes and ranges north of the Arctic Circle, where they have adapted to conditions by spending their larval stage frozen in the ice. Disease patterns differ depending on the geographic distribution of the species. Malaria, for example, is mostly found in sub-Saharan Africa, India, Bangladesh, Southeast Asia, Central and South America, and some parts of the Middle East; dengue fever is in Southeast Asia, Central America, the Caribbean, the southwestern Pacific, and recently in the United States; Japanese encephalitis spans the bottom half of India in a northeastern direction up to Japan, covering eastern China and Southeast Asia. Geographic distributions and transmission patterns are discussed in the remainder of this chapter.

DISEASES

The effects of mosquitoes on human life are far reaching. Every year, they are responsible for transmitting a wide spectrum of diseases, such as malaria, dengue fever, yellow fever, and encephalitis to millions of people. This chapter discusses the main diseases transmitted by mosquitoes, other than malaria (see Chapter 43). Table 42-1 summarizes the viruses and the clinical presentations of the severe forms of the diseases.

Dengue

Dengue was first identified as being caused by a virus in the 1940s, although its existence was probably present much earlier, as reports of dengue fever epidemics were reported as early as 1779. Like West Nile virus, dengue viruses belong to the family Flaviviridae, genus *Flavivirus,* and have serotypes 1 through 4 (DEN-1, DEN-2, DEN-3, and DEN-4), all of which cause infection. Currently, it is estimated that 50 to 100 million people in more than 100 countries are infected yearly with dengue viruses.[42]

Epidemiology and Transmission

Aedes mosquitoes are responsible for transmission of dengue viruses via a human-mosquito-human cycle: having picked up the virus, the female mosquito carries it for the rest of her life and can transmit the disease to many susceptible individuals. These mosquitoes breed either inside or close to houses and often bite humans unnoticed. Because they feed many times in a breeding cycle, numerous people living in one household can be infected by the same mosquito.

Several species of *Aedes* can transmit dengue, but the most important is *Aedes aegypti,* which is found in temperate zones from latitude 45° north to 35° south. *Aedes albopictus,* another species that can transmit the virus, has a larger geographic distribution than *A. aegypti* but is less often the culprit responsible for dengue transmission because these mosquitoes breed farther from households and bite humans less frequently.[42]

Dengue virus transmission is classified as epidemic dengue when a single virus strain is introduced into a region with a large number of hosts and mosquitoes, or as hyperendemic when there is uninterrupted circulation of numerous dengue serotypes in one area. Epidemic dengue is the usual transmission pattern for many smaller islands in South America, Africa, and Asia, where several years pass between epidemics and then the disease reemerges. For travelers, this means that the risk of acquiring the infection is high during epidemics, but it is very low between those periods. Generally, the frequency of dengue hemorrhagic fever (DHF) during epidemics seems to be lower than in areas where the transmission pattern is hyperendemic. In hyperendemic areas, competent mosquito vectors are present throughout the year and the population of uninfected individuals is sufficiently large to sustain the disease. This latter pattern of transmission accounts for the preponderance of global dengue virus infections.[40]

As shown in Figure 42-9, dengue virus has been found in Southeast Asia and the western Pacific islands (with hyperendemic transmission of all four serotypes present in Thailand, Vietnam, Indonesia, Malaysia, and the Philippines). Hyperendemic dengue fever (DF) is also found in India, Pakistan, and Sri Lanka. The *A. aegypti* mosquitoes are also distributed across much of sub-Saharan Africa and the Middle East. In the Americas, *A. aegypti* exists in the southeastern United States, Mexico, Central America, South America, and the Caribbean.[42]

Reports of DF and DHF are increasing, and the most recent global epidemic was in 1998. In the past, DF was considered a relatively benign and nonfatal disease, but since World War II there has been expansion of the disease not only in frequency and geographic distribution but also in morbidity and mortality. The 1980s and 1990s saw a rise in DHF (specifically, serotype DEN-3) in southern Asia, and in the 1980s, there was a rise in epidemic DF/DHF (specifically serotype DEN-2) in Taiwan and the People's Republic of China. Africa has seen large increases in DF incidence since 1980.[38] Dengue has also been emerging much more rapidly in the Americas, with the first major outbreak in Cuba in 1981 and continuing outbreaks in other Central and South American countries since then.[64,68]

Demographic changes, such as population growth and increased human density, contribute to transmission and also to emergence of DHF, and unplanned urbanization that results in

TABLE 42-1. Characteristics of Mosquito-Borne Viral Diseases

VIRUS	FAMILY	GEOGRAPHIC DISTRIBUTION	MAIN AGE GROUP AFFECTED	MORTALITY (%)	SPECIFIC TREATMENT?	HUMAN VACCINE AVAILABLE?	ADDITIONAL NOTES
West Nile virus	Flavivirus	Africa, West Asia, Middle East, Europe, United States	Adults	3–15	No	No	—
St. Louis encephalitis virus	Flavivirus	Central, west, southern United States	Adults	3–30	No	No	<5000 cases (1964–now)
Japanese encephalitis virus	Flavivirus	Japan, China, Southeast Asia, India	Children and adults	30	No	Yes	50,000 cases reported yearly
Murray Valley encephalitis virus	Flavivirus	Australia, Papua New Guinea	Children and adults	30	No	No	Australian
Eastern equine encephalitis virus	Togavirus	Eastern and Gulf coasts of United States, southern USA, South America	Children and adults	35	No	No	200 cases (1964–now)
Canyon virus	Bunyaviridae	United States (including Alaska)	Children and adults	Unknown	No	No	—
La Crosse virus	Bunyaviridae	Central and eastern United States	Children	<1	No	No	California; 60–100 cases yearly
Dengue fever virus	Flavivirus	Central and South America, Africa, Asia, southeastern United States	Adults	DHF, 5 DSS, 40	No	No	50–100 million infected yearly
Yellow fever virus	Flavivirus	Africa and South America	Men ages 15–45	20	No	Yes	200,000 infections yearly
Ross River virus	Togavirus	Australia, Papua New Guinea, South Pacific	Children and adults	Rare	No	No	—

DHF, dengue hemorrhagic fever; DSS, dengue shock syndrome.

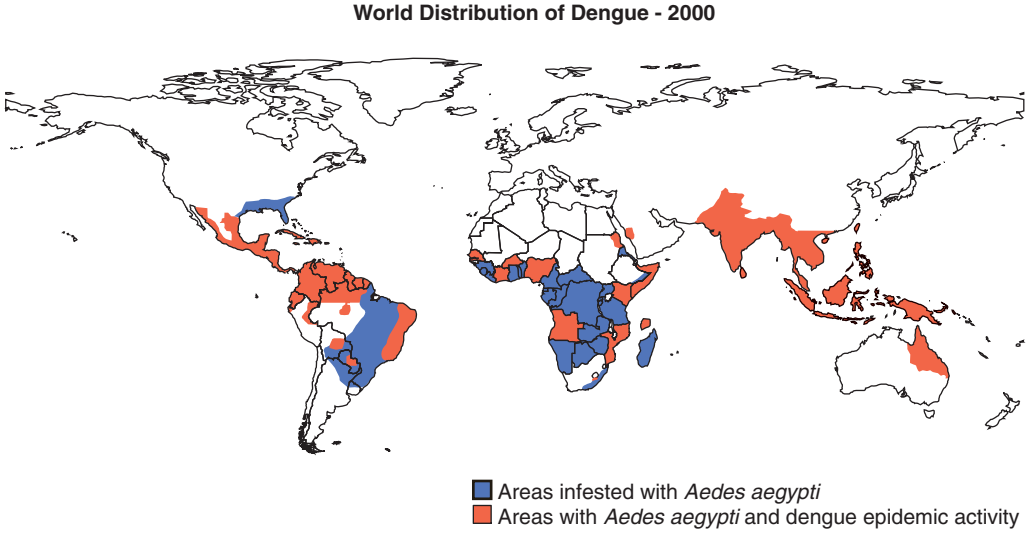

Figure 42-9. World distribution of dengue and mosquito vector *Aedes aegypti*, 2000.[14]

substandard housing situations and poor water and sanitation systems has the same effect. The situation seems to be potentiated by the deterioration of mosquito control programs and of health systems in general in dengue-endemic countries, where, therefore, the disease cannot be well controlled.[52] Climate change alters the interaction between infective mosquitoes and humans. Increased rainfall during certain seasons in tropical areas causes an increased density of mosquitoes. Not only do warmer temperatures decrease the incubation period of the virus inside the mosquito so that the virus can be transmitted earlier but also they may allow greater mosquito population growth and raise the number of susceptible humans to perpetuate the disease.[43]

Clinical Presentation

The four serotypes do not provide cross-protective immunity, so it is possible for one individual to sustain four separate dengue infections over time from the four serotypes.

Risk factors influence disease severity and the development of DHF or dengue shock syndrome (DSS) as opposed to simply dengue fever. These factors include serotype, prior exposure, age, malnutrition, and genetic factors such as human leukocyte antigen (HLA) type. All four serotypes can cause DHF, but DEN-2 appears most virulent. Although previous infection provides homologous immunity to one serotype, persons who experience secondary infection by another serotype are at higher risk of more severe forms of disease. This theory supports the observation that infants less than 1 year of age with passively acquired maternal antibodies have a higher rate of DHF/DSS. This phenomenon, known as immune enhancement or antibody-dependent enhancement, is thought to occur because the Fc receptors of monocytes by dengue viruses complexed to IgG antibodies are more accessible to infectious immune complexes and result in a higher number of cells being infected with this different serotype.[56,62] In terms of age distribution, DHF in the past was seen as a disease of children (especially in Asia, where the disease was previously more predominant). This distribution, however, is slightly different in the Americas, and epidemics have shown increasing numbers of adult patients.[41] Malnourished children, compared with well-nourished children, seem less inclined to develop DHF/DSS than other diseases, perhaps secondary to immunosuppression. Whites have a higher frequency of developing severe disease than do blacks.[56]

The incubation period for the virus in the mosquito is 8 to 12 days before it is transmissible, and in humans there is a 4- to 6-day incubation from bite to clinical infection. Pathogenesis of the virus is hypothesized to be a result of endothelial cell dysfunction, activation of cytokines and complement, and suppression of the reticuloendothelial system.[42] At autopsy, the virus is commonly found in the liver, spleen, thymus, lymph nodes, and lung cells.[87]

The majority of children infected with dengue viruses are asymptomatic or present with mild fever. Typically, older children and adults with DF present with high fever (greater than 39°C [102.2°F]), myalgias, headache, arthralgias, and rash.[87] The tourniquet test (Fig. 42-10) for capillary fragility, which describes the appearance of 20 or more petechiae over a square-inch patch on the forearm after deflation of the blood pressure cuff (held for 5 minutes between systolic and diastolic pressures), may also be positive. Although the World Health Organization (WHO) typically uses a positive tourniquet test to help distinguish dengue hemorrhagic fever from dengue fever, the test has been shown to be positive in both clinical phenomena.[85] A particular characteristic of dengue infection is CNS involvement in both DF and DHF. Patients present with symptoms that range from irritability and depression to encephalitis with seizures and death, with the more severe symptoms noted in DHF. Despite these complications, the prognosis for patients with DF is generally good, with an acute phase of 1 week and up to 2 weeks of convalescence with general malaise and anorexia. Case fatality rates for dengue fever are less than 1%.[19]

Figure 42-10. Positive tourniquet test.

The main characteristic that differentiates DHF from DF is development of plasma leakage. After 2 to 7 days of high fever, patients with DHF have bleeding (ranging from petechiae, ecchymoses, epistaxis, and mucosal bleeding, to more severe gastrointestinal bleeding and hematuria), thrombocytopenia (<100,000/mm^3), and, paradoxically, hemoconcentration and hepatomegaly in some settings.[56] Symptoms include severe abdominal pain, nausea and vomiting, and restlessness or lethargy. Plasma leakage may cause pleural effusion, ascites, and hypoproteinemia. This may lead to circulatory failure, resulting in DSS with hypotension, cool extremities, and altered mental status. Encephalopathy is possible in DHF and can result from cerebral anoxia, edema, or intracranial hemorrhage. It is still currently unknown whether dengue viruses infiltrate the CNS directly or if neurologic manifestations are nonspecific complications. Case fatality rates for DHF are approximately 5%;[14] in DSS, they can be as high as 44%.[19]

Diagnosis

Laboratory findings for patients with DF may show neutropenia with subsequent lymphocytosis marked by atypical lymphocytes, hyponatremia, hypoproteinemia, and mild elevation of liver enzymes. Other findings include albuminuria and occult blood in the stool. In patients with DHF, either thrombocytopenia (defined by the WHO as less than 100,000/mm^3) or hemoconcentration (elevation of hematocrit 20% or greater than the average for age, sex, and population) is used by the WHO to reach a provisional diagnosis of DHF if certain clinical observations (e.g., acute onset of high fever, hemorrhagic manifestations, hepatomegaly, shock) are made. In cases with extensive liver involvement, vitamin K–dependent prothrombin factors, such as II, VII, IX, and X, may be reduced, and assays of coagulation factors may reveal reduced fibrinogen, pro-

thrombin, factors VIII and XII, or antithrombin III. About one third to one half of patients with DHF have increased partial thromboplastin and prothrombin times. Chest films may show a unilateral (usually right-sided) pleural effusion in more severe disease.[87]

Definitive diagnosis of dengue is most likely to be made when the virus is circulating in the periphery, from 2 days prior to onset of symptoms to 6 days after onset. Virus identification can be confirmed by inoculation of serum into certain mosquito cell lines (most commonly *A. albopictus* [C6-36] and *Aedes pseudoscutellaris* [AP61], but with better sensitivity when mosquitoes in the genus *Toxorhynchites* are used), but these methods are not commonly available. More commonly, diagnosis can be made by immunofluorescence assay of serum or cerebrospinal fluid (CSF) with serotype-specific monoclonal antibodies (IgM enzyme-linked immunosorbent assay [ELISA]), which has a sensitivity of 90% to 97%.[42] Unfortunately, the IgM-capture ELISA is negative early in the disease, only becoming positive 4 to 5 days after onset of symptoms.[85] Other methods of laboratory diagnosis of DF include demonstration of a fourfold increase in IgM or IgG antibody titer between acute-phase and convalescent-phase serum samples, or detection of DF genomic sequences from autopsy tissue, serum, or CSF with polymerase chain reaction (PCR).[87] However, although several protocols have been written for PCR detection of DF, this method is not widely used clinically and has been utilized more often in the setting of detecting dengue viruses in pathologic tissue, cell cultures, and mosquito specimens.[42]

The recommendation of the Centers for Disease Control and Prevention (CDC) is that, for testing and reporting, both acute-phase and convalescent-phase (6 days after the onset of symptoms) serum samples be sent to the state health department (which will forward them to the CDC for testing). The acute-phase specimen should be stored on dry ice (or unfrozen at 4° C [39.2°F] if sent within a week), and the convalescent-phase specimen should be shipped on ice (or in a rigid container without ice if next-day delivery is possible).[19]

Despite all this, however, dengue infections are often diagnosed clinically because laboratory-based diagnosis is often unavailable, and they should be considered only after other common tropical infections such as malaria are ruled out.[85] History is of utmost importance: symptoms in a traveler or immigrant that begin 2 weeks after departure from an endemic area cannot be attributed to dengue, as the incubation period is less than 2 weeks.

Treatment and Prevention

There is no specific treatment for dengue. Treatment includes supportive therapy, such as aggressive fluid and electrolyte resuscitation. Early recognition is important, as the acute increase in vascular permeability may cause extreme amounts of plasma to be lost from vascular compartments for 24 to 48 hours. Fever should be treated with antipyretics (especially in children to avoid febrile seizures), and the WHO recommends treatment with acetaminophen (paracetamol) if the temperature is greater than 39° C (102.2° F), with dosing as follows: for infants less than 1 year of age, 60 mg per dose; 1 to 3 years of age, 60 to 120 mg per dose; 3 to 6 years of age, 120 mg per dose; and 6 to 12 years of age, 240 mg per dose. The WHO recommends no more than six doses in a 24-hour time period, and advises against salicylates to prevent Reye-like syndrome.[87]

Volume expanders can include normal saline, Ringer's lactate, or 5% glucose diluted in half-normal or normal saline administered in 20 mL/kg boluses initially, and then continued with maintenance therapy as needed. One recent study suggests that Ringer's lactate at 25 mg/kg over 2 hours should be given for initial resuscitation in children with moderately severe DSS, and that 6% hydroxyethyl starch (also in the amount of 25 mg/kg) may be preferable in children with severe shock.[86] Because of plasma loss, the hematocrit may initially increase, but blood transfusion should be given if there is a clinical indication of internal bleeding. The WHO recommends fresh-frozen plasma and concentrated platelets only if there is massive risk of bleeding from coagulopathy. At the same time, because of the volume resuscitation, a drop in hematocrit does not definitely suggest internal hemorrhage, as reabsorption of extravasated plasma may occur when shock ceases. Continued overzealous administration of fluids at this point can lead to pulmonary edema or heart failure. All these measures must be based on clinical judgment, in turn based on indicators such as vital signs, urine output, and hematocrit.[87]

Despite ongoing vaccine initiatives, there is not yet a vaccine against dengue viruses. Vector control is the mainstay of prevention.

Yellow Fever

The first epidemic caused by the yellow fever virus is thought have been in the Yucatan Peninsula in 1648. This single-stranded RNA flavivirus causes infection in up to 200,000 persons per year, with an estimated 30,000 deaths annually, mainly in Africa (90% of cases) and South America (10% of cases).[84]

Epidemiology and Transmission

It is thought that the yellow fever virus evolved about 3000 years ago from other arthropod viruses in Africa and was carried to the Americas by Dutch slave traders in the 17th century. During the late 17th century, epidemics and outbreaks occurred in the Yucatan Peninsula, several places on the east coast of the United States, and the Caribbean islands. While the English, Spanish, and French were colonizing the Americas, yellow fever played a prominent role by decimating populations of sailors, troops, and citizens, especially near port cities. Figures 42-11 and 42-12 show the most recent endemic areas in Africa and the Americas.[88]

The mosquito species *A. aegypti* was hypothesized as early as 1881 to be the vector, and it is indeed one of the main mosquitoes that transmit this disease in urban settings. There are three patterns of yellow fever transmission: urban, intermediate, and sylvatic (jungle). In urban yellow fever, no monkeys are involved in transmission, and domestic mosquitoes, such as *A. aegypti,* carry the virus from person to person. In intermediate yellow fever, mosquitoes of the genus *Haemagogus,* or other forest-canopy species, are known to transmit this disease to both monkeys and humans; this is the most common type of transmission in recent outbreaks in Africa. Finally, in sylvatic yellow fever, nonhuman primates, such as monkeys, are the zoonotic focus, and wild mosquitoes can become infected by the monkeys and pass the disease to humans.[89]

Interestingly, there is also vertical transmission of the virus within mosquitoes: a female mosquito can pass the virus to her offspring. That mosquitoes serve as additional reservoirs and vectors of the virus ensures endemicity of the disease. Peak

Figure 42-11. Endemic zones for yellow fever in Africa.

transmission in Central and South America is between January and March, and it is highest during rainy season and early dry season (July through October) in Africa.[84]

The female mosquito inoculates about 1000 virus particles (the 50% lethal dose for monkeys is less than one particle) intradermally during one human feeding, and viral replication occurs at that site. It is believed that the virus spreads through lymphatic channels and has preference for replication in lymphoid cells, and then it spreads hematogenously. During this viremia, uninfected mosquitoes can become infected if they bite infected humans.

Clinical Presentation

The main risk factor for disease is exposure, as evidenced by the fact that a large portion of cases are in men of ages 15 to 45 years who have occupational exposure in mosquito-infested areas. Unvaccinated individuals (such as persons too young to have been immunized) in epidemics are also at higher risk,

Figure 42-12. Endemic zones for yellow fever in the Americas.

and areas with poor health care have the highest rates of cases.[84]

There are three phases in the classic description of yellow fever: infection, remission, and intoxication. After an incubation period of 3 to 6 days, the infection stage consists of sudden onset of fever, headache, photophobia, back and epigastric pain, anorexia, and vomiting. Helpful clinical signs include Faget's sign (bradycardia related to fever), conjunctival injection, and a coated tongue with pink edges. The infection stage may resolve rapidly within 3 to 4 days and be followed by up to 48 hours of remission.[84]

The intoxication period occurs in an estimated 15% or more of individuals infected with this virus, manifesting with liver and renal failure (jaundice, azotemia, and oliguria), myocardial

involvement, encephalopathy, and shock. Viral-induced thrombocytopenia and acquired coagulopathy can cause severe hemorrhage, ranging from epistaxis, metrorrhagia, and gastrointestinal bleeding (the reason Spanish-speaking peoples called the disease *vomito negro*, or black vomit, before the disease was officially named).[4] Severe illness has a case fatality rate of about 20%, which may approach 50% in some outbreaks.[59]

Diagnosis

Laboratory findings may include leukopenia, thrombocytopenia, elevation of liver enzymes (but usually normal alkaline phosphatase), elevated blood urea nitrogen (BUN) and creatinine, and albuminuria. Definitive diagnosis is obtained by IgM ELISA and is best done with paired acute and convalescent sera. The acute sample should be drawn within several days of symptom onset, and the convalescent sample should be drawn about 14 days later. A neutralization test, which is done by showing loss of infectivity through reaction of the virus with a specific antibody, can also be done to confirm the diagnosis of yellow fever.[84]

The virus can be isolated from serum specimens or liver tissue after death, via inoculation of mice, mosquitoes, or cell cultures, but these techniques are generally not used for diagnosis.[84]

Treatment and Prevention

Although there is no treatment besides supportive care, there is a vaccine against yellow fever (17D-204 strain YF-VAX [Aventis, Swiftwater, PA]), and 0.5 mL of the reconstituted vaccine is administered subcutaneously.[24] This vaccine is a live, attenuated vaccine that is generally considered very safe: usually, the only reactions are mild headaches, myalgias, or low-grade fevers for several days. As more travelers enter endemic areas without receiving the vaccine, the number of yellow fever cases imported into the United States and Europe has increased.[59] The CDC has estimated the risk of going to endemic zones to be 0.4 to 4.3 cases per million travelers.[12] The CDC and the Advisory Committee on Immunization Practices recommend that travelers (older than 9 months) to certain areas in Africa and South America be vaccinated. Some countries (not the United States) require vaccination before allowing travelers to enter from endemic areas. Vaccination guidelines (and authorized centers for vaccination) can be found on the CDC Yellow Book website at www2.ncid.cdc.gov/travel/yb/utils/ybGet.asp?section=dis&obj=yellowfever.htm.

International travelers must receive yellow fever vaccines approved by the WHO and administered at an approved yellow fever vaccination center, and then obtain an international certificate of vaccination. Although the International Health Regulations require boosters every 10 years, many studies suggest that vaccine immunity endures for over 30 years and possibly for life.[15]

Immediate hypersensitivity reactions in people with allergies to eggs or other substances are thought to occur in 1 out of 131,000 vaccinations. Before 1945, the vaccine was associated with encephalitis in children, but standardization of the virus vaccine has decreased the incidence to one case per 8 million doses administered. Yellow fever vaccine–associated encephalitis has been reported in four adults in the United States since 2001, without fatality.[65] Other cases of vaccine-associated viscerotropic disease and hepatic failure may have resulted from host factors; this is under investigation. Caution is advised in babies less than 6 months old, and in persons who are pregnant, nursing, immunocompromised, sensitive to eggs, or receiving other vaccines simultaneously.[24] The CDC encourages health care providers to report febrile illness thought to be caused by the yellow fever vaccination to the CDC/FDA Vaccine Adverse Events Reporting System (VAERS), either online at https://secure.vaers.org/VaersDataEntryintro.htm, by printing a VAERS form at www.vaers.org/contact.htm, or by calling 800-822-7967.

Eradication of *A. aegypti* via well-executed control campaigns in the 1970s has been successful in many Central and South American countries, with much credit owed to the Pan American Health Organization. However, vigilance must be maintained, as *A. aegypti* has reestablished itself in many of these areas. Much work must be done in Africa, where the mosquito has never been eradicated.[59]

Japanese B Encephalitis

The Japanese encephalitis (JE) virus, a member of the *Flavivirus* genus, is the major cause of viral encephalitis in Asia, with as many as 50,000 cases reported every year. Although the JE virus is a leading cause of viral encephalitis in Asia, there is less than one case per year in U.S. civilians and military personnel living in or traveling to Asia, with rare outbreaks in U.S. territories in the western Pacific. There has been a recent expansion of JE into northern Australia (Fig. 42-13).[44]

Epidemiology and Transmission

The *Culex* mosquitoes (usually *Culex tritaeniorhynchus*, *Culex vishnui*, and *Culex pseudovishnui*) are the vectors for this virus, and vertebrate hosts such as pigs and birds sustain the virus. Because humans are not necessary for transmission, the virus can still be maintained in an enzootic cycle even where JE immunity is high (either in endemic regions or through vaccinations) and human cases do not occur. The mosquitoes breed in flooded fields associated with rice production. Because these settings contain factors that enhance the JE transmission cycle (e.g., large numbers of pigs and birds to serve as reservoirs for infection), JE infection rates may be as high as 3% in these areas.[59]

JE has two transmission patterns in Asia. Seasonal transmission occurs in the more temperate northern regions (e.g., Japan, People's Republic of China, Taiwan, northern India) and can cause large epidemics. The second pattern of year-round transmission presents in tropical southern regions (e.g., southern Thailand, southern Vietnam, southern India, Indonesia, Malaysia, Philippines, Sri Lanka) and is usually more endemic or sporadic.

Clinical Presentation

Persons at increased risk include individuals (residents, active duty military, or expatriates) living in endemic areas. Travelers have a low risk of acquiring the disease, estimated to be less than one in a million American travelers going to endemic areas.[59] Most residents of endemic areas are infected during childhood or early adulthood,[47] with about 10% of the susceptible population infected yearly. The disease appears to affect mainly children less than 15 years of age, although there also seems to be a secondary increase of disease in older adults, posited to be caused by waning immunity.[6]

The incubation period is from 5 to 15 days. Most infections with JE are subclinical. Early symptoms include sudden onset

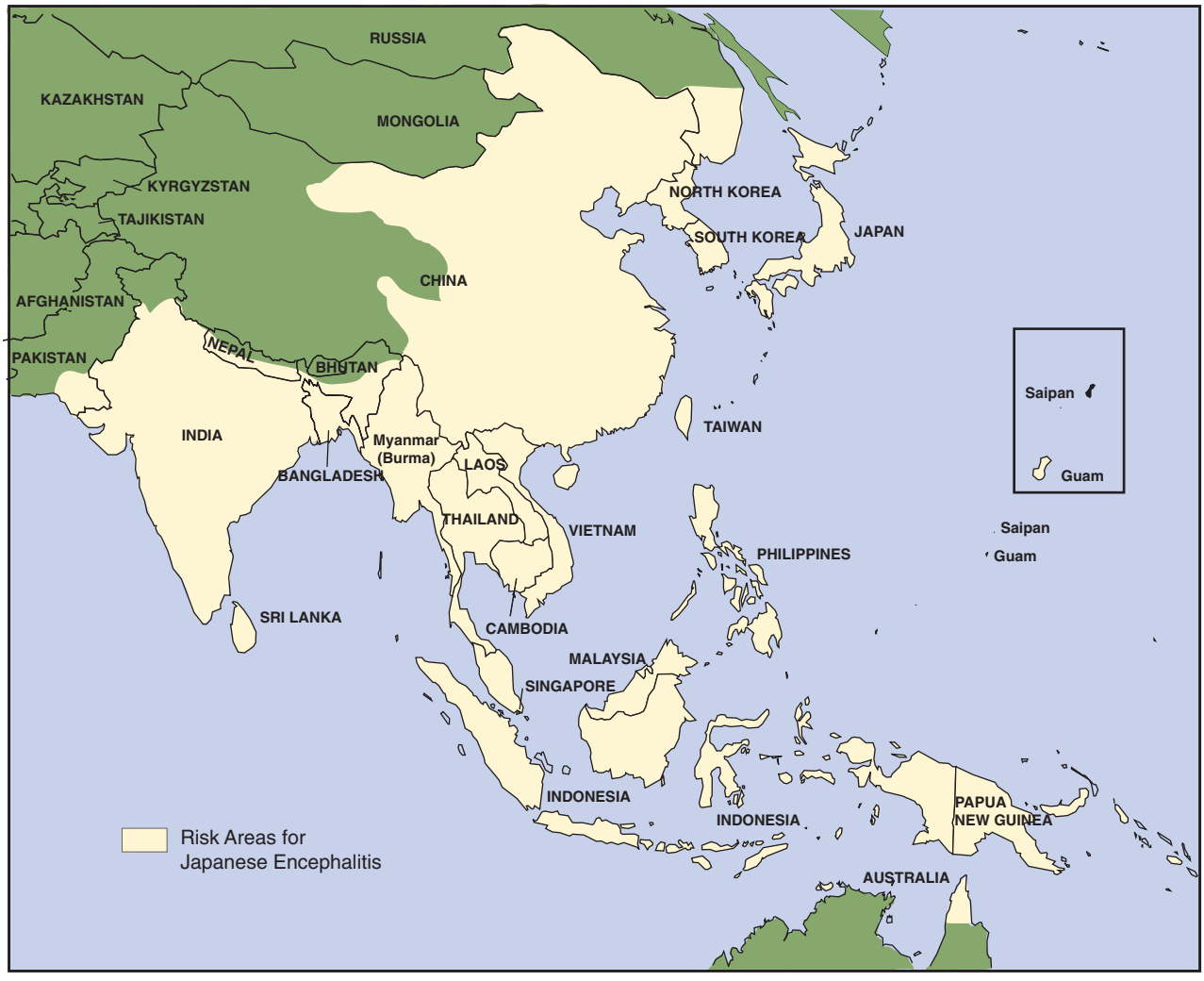

Figure 42-13. Geographic distribution of Japanese encephalitis.

of fever, headache, nausea and vomiting, and abdominal pain. JE may also present with coryza, diarrhea, and rigors. In some individuals, abnormal behavior is the only clinical manifestation, so JE has been misidentified as mental illness.[75] In the Korean War, for example, American military personnel with JE were sometimes diagnosed with "war neurosis."[55] After several days, JE may develop into aseptic meningitis with or without encephalopathy, or into encephalitis (less than 1% of infected individuals) presenting with delirium and abnormal motor movement. Children in particular seem to have more convulsions than do adults infected with the virus (85%[51] compared with 10%[27]). Seizures are more often generalized tonic-clonic than focal. Abrupt onset of flaccid paralysis in one or more limbs has been reported.[76]

The case fatality rate of JE is approximately 30%, and almost 50% of survivors have severe neurologic sequelae, including motor deficits, upper and lower motor neuron weakness, and cerebellar or extrapyramidal signs.[75]

Diagnosis

Most patients have peripheral leukocytosis; hyponatremia is secondary to the syndrome of inappropriate antidiuretic hormone secretion (SIADH). Fifty percent of patients have high CSF opening pressure on lumbar puncture, and analysis of CSF may show pleocytosis (predominantly lymphocytic) of 10 to 100 cells/mm^3, with normal glucose and slightly elevated protein. In the early disease, however, there may be no CSF pleocytosis, and leukocytes are predominantly neutrophilic.

It is difficult to isolate JE virus from blood because of the low viral burden. The most common method of diagnosing JE in the past was with a serologic hemagglutination inhibition test. Since the 1980s, however, IgM-capture ELISA has been used, and confirmation is recommended with another assay for IgG.

Both noncontrast and contrast computed tomography (CT) and magnetic resonance imaging (MRI) may show nonenhancing low-density areas in the thalamus, basal ganglia, midbrain, pons, or medulla,[73] and an electroencephalogram may show

theta and delta coma, burst suppression, and epileptiform activity.[64]

Treatment and Prevention

There is no specific treatment for JE beyond supportive care. Other modalities, such as reducing intracerebral pressure with mannitol and providing seizure prophylaxis, have been suggested by some experts, but there have been no studies on agents to use and whether these interventions are beneficial. Steroids and interferon-alpha in other trials have not been shown to be beneficial.[59]

Vaccination is an important method of disease control and has nearly eliminated the disease in some parts of the world. In Asia (e.g., the People's Republic of China, Japan, Korea, Taiwan), more than 500 million doses of JE vaccine have been administered since the 1970s.

The three types of JE vaccine currently used are (1) inactivated mouse brain–derived vaccine, (2) inactivated primary hamster kidney cell–derived vaccine, and (3) live, attenuated vaccine (so far used only in China, but it appears to be effective). The first, known as the Biken vaccine (produced by the Research Institute of Osaka University, distributed in the United States by Connaught Laboratories, Inc.), is the only vaccine available to international travelers from nonendemic countries. For adult travelers, the recommendation for primary immunization is a series of three subcutaneous vaccinations of 1.0 mL each (0.5 mL for children 1 to 3 years of age), on days 0, 7, and 30 (an abbreviated schedule of days 0, 7, and 14 is used if the longer schedule is not feasible). The last dose should be given at least 10 days before the actual date of travel to allow a sufficient immune response. There have been no studies on administering this vaccine to infants less than 1 year of age.[6]

This killed vaccine has a high efficacy (91%) and a low rate of side effects.[6] It is associated with a moderate frequency of local adverse effects, as well as mild systemic effects (e.g., fever, headache, malaise, rash), in 10% to 20% of vaccinated individuals. More serious events, including angioedema, can appear up to 2 weeks after administration, and sometimes after the second or third dose when the first was received without complications. There have also been concerns about vaccine-related neurologic events, including encephalitis, seizures, and even death. Since the 1990s, the reported rate for all adverse events for the killed vaccine is 2.8 per 100,000 in Japan and 15 per 100,000 in the United States.[59]

It is difficult to assess which travelers should receive the vaccine. The low rate of human disease must be balanced against the severe effects of JE infection. Furthermore, the JE infection rate in travelers is low, and there are legitimate concerns regarding the side effects of vaccination. The vaccine should not be given to all travelers to Asia; according to the CDC, it should be offered to those persons older than 9 months staying a prolonged amount of time (>1 month) in endemic areas during transmission season, and if travel includes rural areas. Persons staying less than 1 month but with specific risk factors for exposure (e.g., extensive amount of time spent outdoors during seasons of peak transmission) may also consider vaccination.[6]

West Nile Virus

First identified in 1937 in a febrile patient in Uganda, the West Nile virus (WNV) is a flavivirus that, prior to 1999, was found only in Africa, West Asia, and the Middle East. WNV has received much recent press in North America because of its intrusion into the United States, with the first report in the summer of 1999 in New York. In 2004, the CDC received 2535 reports of WNV disease, including 98 deaths, down from 264 deaths the previous year.[13] Since 1999, WNV has been found in all 50 of the United States except Hawaii, Alaska, and Oregon.[16]

Epidemiology and Transmission

WNV is a single-stranded RNA virus that is part of the family Flaviviridae, genus *Flavivirus*. All flaviviruses have a size of approximately 40 to 60 nm (Fig. 42-14), are enveloped with icosahedral nucleocapsids, and have single-stranded RNA of about 10,000 to 11,000 bases.

Mosquitoes are the vectors for this virus, and birds are the most common reservoir. The most commonly named vector is the *Culex* mosquito species, but WNV has recently been identified in more than 40 different mosquito species in North America, which contributes to its permanent establishment and wide distribution in the United States (Fig. 42-15). The different species of mosquitoes have varying host preferences and behavior, and frequent transmission to migrating birds extends the reach of the virus.[36] Newly infected birds sustain viremia for about 1 to 4 days (after which they develop lifelong immunity), and during that time, mosquitoes can contract the virus. Humans and other mammals such as horses are considered incidental or "dead-end" hosts, as they often do not sustain viremia long enough to serve as reservoirs for the virus. The effects on nonhuman life can be significant: the 2002 WNV epidemic and epizootic in the United States, for example, reported 16,471 dead birds and 14,751 equine cases. In horses, as in humans, WNV crosses the blood–brain barrier and interferes with CNS functioning, ultimately leading to possible death.[11]

Other, rarer methods of transmission include organ transplantation, blood transfusion,[10] transplacental infection,[8] and possibly breast milk.[9] Because transmission from blood transfusions reached 2.7 cases per 10,000 units during the peak of the 1999 outbreak in New York, and because there were confirmed cases of WNV transmission to organ transplant recipients, routine screening of blood donors for WNV RNA began in the summer of 2003. In 2003 and 2004, 519 donors were identified by this method, resulting in the removal of more than 1000 potentially infectious units of blood products from the Red Cross supply. All donated blood continues to be screened for WNV with investigational nucleic acid amplification tests, either on individual samples or on "minipools" of up to 16 donations.[78] A recent study of targeted nucleic acid amplification testing of individual donations in high-prevalence regions suggests that this approach may be cost effective, because units with low levels of viremia may not be detected in minipools of blood donations.[5]

Clinical Presentation

The virus has an incubation period of 3 to 14 days.[67] Disease usually occurs in temperate zones either in late summer or early fall, or year-round in milder climates. Most WNV infections in humans are asymptomatic or present with a mild influenza-like illness with malaise, headache, nausea, vomiting, anorexia, lymphadenopathy, and rash. In a review of 98 community-dwelling patients with laboratory evidence of WNV infection but no evidence of neuroinvasive disease, fatigue was present in 96% of patients, fever in 81%, headache in 71%, and difficulty concentrating in 53%.[83]

Severe infections (in less than 1% of persons infected with WNV) include encephalitis, meningitis, or meningoencephalitis, with case fatality rates of 3% to 15%.[17] So far, advanced age has been the only clearly identified risk factor for development of severe neurologic disease from WNV. Clinical criteria are listed in Table 42-2. For public health and surveillance purposes, the CDC uses more specific criteria for arboviral infections and includes disease caused by California serogroup viruses, eastern and western equine encephalitis viruses, Powassan virus, St. Louis encephalitis virus, and WNV. This was most recently revised in 2004 (Box 42-1).

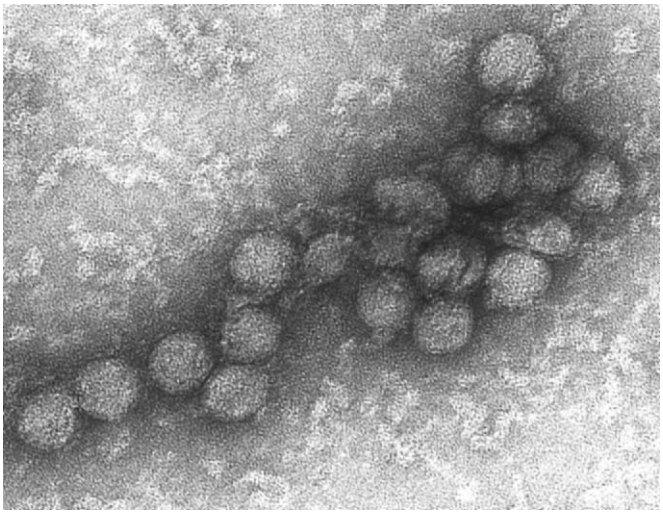

Figure 42-14. Electron microscopy of the West Nile virus.

Although the clinical presentation of meningitis and encephalitis in WNV is nonspecific, one particular feature of WNV encephalitis is that it may present with Parkinsonian features or other movement disorders, such as myoclonus or intention tremor. Another clinical syndrome associated with severe WNV disease is acute flaccid paralysis, which can rapidly progress to paralysis of one or up to all four limbs. It may also present as severe, asymmetric weakness, usually with sensation preserved. Affected limbs show decreased or completely absent deep tendon reflexes, most likely from anterior horn cell involvement similar to that seen in poliomyelitis.[36]

Other clinical findings may include ocular complications (e.g., optic neuritis, chorioretinitis, retinal hemorrhage, uveitis, conjunctivitis), bladder dysfunction,[36] myocarditis, pancreatitis, and fulminant hepatitis.[67]

Diagnosis

Laboratory findings for severe disease usually include normal or slightly elevated leukocyte counts in peripheral blood, possible hyponatremia in individuals with WNV encephalitis, pleocytosis and increased protein in the CSF, and no findings on CT scans of the brain.[67] For definitive diagnosis, serum is ideally collected within 8 to 14 days of symptom onset, and CSF is collected within 8 days to give the highest diagnostic sensitivity. Serum or CSF should show IgM antibodies to WNV by IgM-

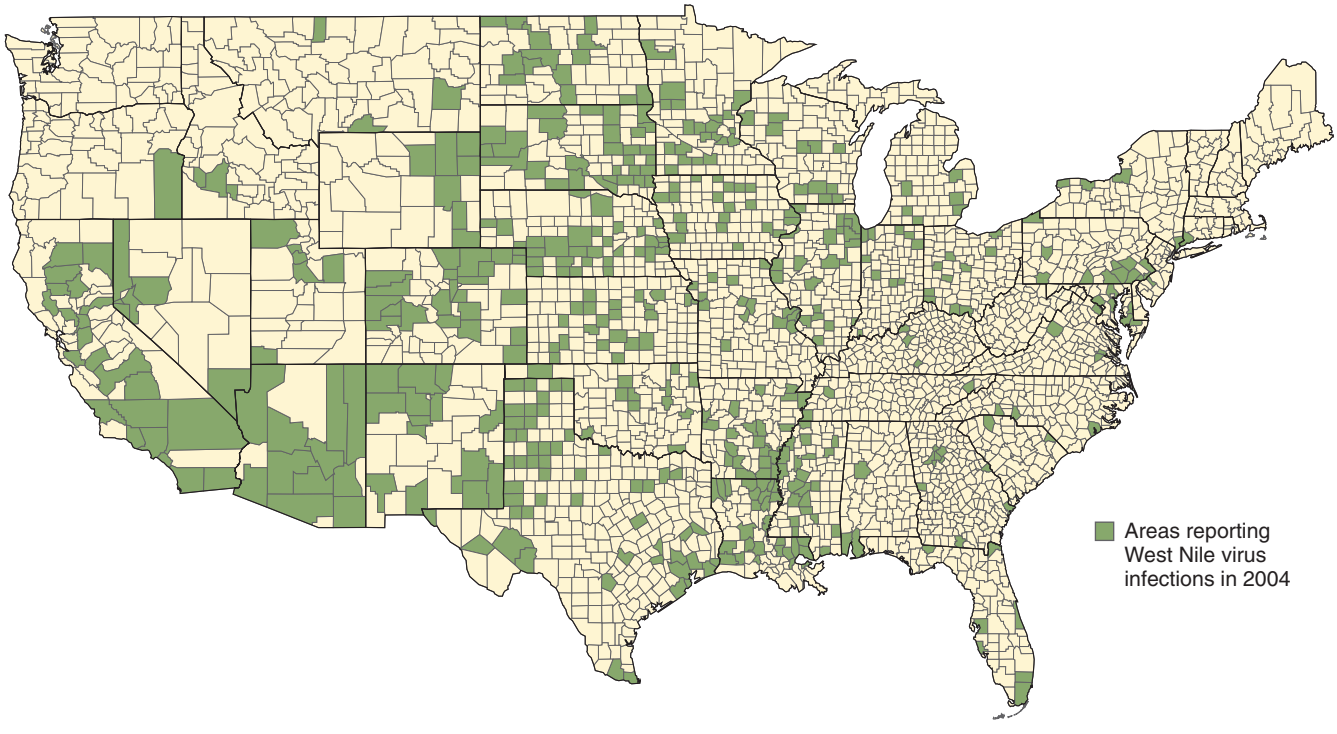

Figure 42-15. Map of 2004 West Nile virus infections in the United States.

TABLE 42-2. Clinical Criteria for West Nile Virus Infection

SYNDROME	CLINICAL CRITERIA SIGNS	ADDITIONAL EVIDENCE
West Nile meningitis	Nuchal rigidity, Kernig or Brudzinski sign, or photophobia or phonophobia	One or more of the following: Fever (>38°C [100.4°F]) or hypothermia (<35°C [95°F]) CSF pleocytosis (5 leukocytes/mm^3) Peripheral leukocyte count, >10,000/mm^3 Neuroimaging findings consistent with acute meningeal inflammation
West Nile encephalitis	Encephalopathy (depressed or altered level of consciousness, lethargy, or personality change lasting 24 hours)	CNS inflammation, with 2 or more of the following: Fever (>38°C [100.4°F]) or hypothermia (<35°C [95°F]) CSF pleocytosis (5 leukocytes/mm^3) Peripheral leukocyte count, >10,000/mm^3 Neuroimaging findings consistent with acute inflammation (with or without involvement of the meninges) or acute demyelination Presence of focal neurologic deficit Meningismus EEG findings consistent with encephalitis Seizures, either new onset or exacerbation of previously controlled
Acute flaccid paralysis	Acute onset of limb weakness with marked progression over 48 hours	At least 2 of the following: Asymmetry to weakness Areflexia/hyporeflexia of affected limb(s) Absence of pain, paresthesia, or numbness in affected limb(s) CSF pleocytosis (5 leukocytes/mm^3) and elevated protein levels (45 mg/dL) Electrodiagnostic studies consistent with an anterior horn cell process Spinal cord MRI documenting abnormal increased signal in the anterior gray matter

CSF, cerebrospinal fluid; EEG, electroencephalography; MRI, magnetic resonance image.
From Sejvar JJ, Haddad MB, Tierney GL, et al: Neurologic manifestations and outcome of West Nile virus infection. JAMA 290:511–515, 2003.

Box 42-1. 2004 CDC Case Definition for Neuroinvasive and Non-neuroinvasive Domestic Arboviral Diseases

CLINICAL CRITERIA FOR DIAGNOSIS

Neuroinvasive disease requires the presence of fever and at least one of the following, as documented by a physician and in the absence of a more likely clinical explanation:

- Acutely altered mental status (e.g., disorientation, obtundation, stupor, or coma), or
- Other acute signs of central or peripheral neurologic dysfunction (e.g., paresis or paralysis, nerve palsies, sensory deficits, abnormal reflexes, generalized convulsions, or abnormal movements), or
- Pleocytosis (increased white blood cell concentration in cerebrospinal fluid [CSF]) associated with illness clinically compatible with meningitis (e.g., headache or stiff neck).

Non-neuroinvasive disease requires, at minimum, the presence of documented fever, as measured by the patient or clinician, the absence of neuroinvasive disease (above), and the absence of a more likely clinical explanation for the illness. Involvement of non-neurological organs (e.g., heart, pancreas, liver) should be documented using standard clinical and laboratory criteria.

LABORATORY CRITERIA FOR DIAGNOSIS

Cases of arboviral disease are also classified as either confirmed or probable, according to the following laboratory criteria:

Confirmed Case

- Fourfold or greater change in virus-specific serum antibody titer, or
- Isolation of virus from or demonstration of specific viral antigen or genomic sequences in tissue, blood, CSF, or other body fluid, or
- Virus-specific immunoglobulin M (IgM) antibodies demonstrated in CSF by antibody-capture enzyme immunoassay (EIA), or
- Virus-specific IgM antibodies demonstrated in serum by antibody-capture EIA and confirmed by demonstration of virus-specific serum immunoglobulin G (IgG) antibodies in the same or a later specimen by another serologic assay (e.g., neutralization or hemagglutination inhibition).

Probable Case

- Stable (less than or equal to a twofold change) but elevated titer of virus-specific serum antibodies, or
- Virus-specific serum IgM antibodies detected by antibody-capture EIA but with no available results of a confirmatory test for virus-specific serum IgG antibodies in the same or a later specimen.

Case Definition

- A case must meet one or more of the above clinical criteria and one or more of the above laboratory criteria.

From Centers for Disease Control and Prevention, and available at www.cdc.gov/epo/dphsi/casedef/arboviral_current.htm.

capture ELISA (MAC-ELISA, available in the United States from Focus Technologies and PanBio, Inc.). There is, however, a chance that persistent IgM antibodies can be found in people years after living in places where WNV is epidemic. The diagnosis can be further confounded by the fact that these IgM ELISA tests have a cross-reactivity of up to 44% with other flaviviruses during acute infection.[36]

The most specific test for arthropod-borne flaviviruses is the plaque-reduction neutralization test (PRNT). It can be used both to negate the false-positive results sometimes seen in MAC-ELISA or other assays, and to facilitate discrimination among the flaviviruses.[22]

Efforts have been made to develop more specific and rapid serologic assays. Promising candidates include a microsphere immunoassay that discriminates among different flavivirus infections, as well as an immunofluorescence assay that has less cross-reactivity among WNV IgM antibodies and closely related flaviviruses. PCR (used for screening blood products in the United States) or viral culture can also isolate the virus, but sensitivity is not high because of the transient and low levels of viremia, as the virus quickly disappears from the periphery into the CNS in severe disease.[67]

Treatment and Prevention

Currently, there is no specific treatment for WNV, although certain antivirals, antibodies, and drugs altering the inflammation process (e.g., interferon) are being tested. Nucleoside analogs have been studied in vitro, and the most advanced testing in vivo has been with ribavirin, with no reduction in disease[48] or viremia[58] shown.

For prevention of disease, general control measures against mosquitoes can be taken, but so far there is no specific way to prevent WNV. A vaccine exists for horses, but it has not yet been studied in humans. Theoretically, there is a potential for an inactivated virus to induce long-term immunity, because such a vaccine exists for JE and other flavivirus diseases. Candidate vaccines against WNV include a chimeric virus based on the live, attenuated yellow fever 17D vaccine virus containing the pre-membrane and envelope proteins of WNV,[32] a DNA vaccine virus based on a low-virulence form of WNV found in Australia (the Kunjin virus),[53] and a recombinant envelope-based protein vaccine.

Surveillance and Reporting

Reporting requirements for suspected WNV infections vary across local and state health departments, but WNV encephalitis is now on the list of designated nationally notifiable conditions. The CDC may use results from commercial laboratories but may also request samples for diagnosis in its own laboratory. Information on how to send specimens can be found at www.cdc.gov/./ncidod/dvbid/misc/arboviral_shipping.htm.

For ecologic surveillance, the CDC and other federal agencies have established specific guidelines and protocols to track avian, equine, and mosquito epizootics.

St. Louis Encephalitis

The St. Louis encephalitis (StLE) virus is a flavivirus related to Japanese encephalitis and West Nile virus, and it is also transmitted by mosquitoes. Since 1964, less than 5000 human cases of StLE have been reported in the United States.[21]

Epidemiology and Transmission

The *Culex* mosquito is the main species that transmits StLE virus, with birds as a reservoir and humans as another host. The viremia, however, does not cause illness in either birds or mosquitoes. Around the Gulf Coast, Ohio, and Mississippi Valley, the culprit mosquitoes are *Culex pipiens* and *Culex quinquefasciatus;* in Florida, *Culex nigripalpus;* and in the western states, *Culex tarsalis*.[21]

In temperate parts of the United States, disease usually occurs in late summer or early fall, but it can occur year-round in the southern United States. Outbreaks can occur almost anywhere in the United States; the most recent epidemic was in the mid-1970s in the Midwest. Recent outbreaks of StLE have been less than 30 cases each time. The most recent outbreak in Louisiana was 20 reported cases.[18]

Clinical Presentation

Risk factors are low socioeconomic status, outdoor occupations, and older age.

StLE has an incubation period of approximately 5 to 15 days, with a 300:1 ratio of asymptomatic to symptomatic cases. Patients with symptomatic StLE present with high fever and headache (55%), aseptic meningitis (40%), and encephalitis (5%).[71] Infants, in particular, may have occasional convulsions and spastic paralysis. Of recognized cases, 95% are hospitalized for CNS involvement. The case fatality rate is 3% overall and up to 30% in older adults.[18]

Treatment and Prevention

As is the case for many viral illnesses, there are neither specific therapies nor vaccines.

Eastern Equine Encephalitis

First discovered in the brain of a horse with encephalitis in 1933 in New Jersey, eastern equine encephalitis (EEE) is a serious mosquito-borne disease with a high case fatality rate that appears in the eastern half of the United States. The virus is a member of the family Togaviridae, genus *Alphavirus,* and it is related to western and Venezuelan equine encephalitis viruses.[60]

Epidemiology and Transmission

The transmission cycle involves birds and several species of mosquitoes, predominantly *Culiseta melanura* (Fig. 42-16). Other possible mosquito vectors include species in the genera *Aedes* and *Coquillettidia*.[60] Humans and horses are considered hosts with incidental infections that can progress to severe disease.

EEE occurs mainly along the eastern and Gulf coasts of the United States, but cases have been documented as far north as southern Canada and as far south as northern parts of South America. It occurs mainly in the summertime.

From 1964 to the present, there have been 200 confirmed cases in the United States, with an average of four cases per year. The real incidence is probably higher because of underreporting and underrecognition. Most of the cases have been in Florida, Georgia, Massachusetts, and New Jersey, near coastal areas and freshwater swamps.[20]

Clinical Presentation

At risk for developing EEE are persons who live (or visit) endemic areas and those who spend significant time outdoors. Individuals older than 50 years and younger than 15 years have a greater chance of developing severe disease.

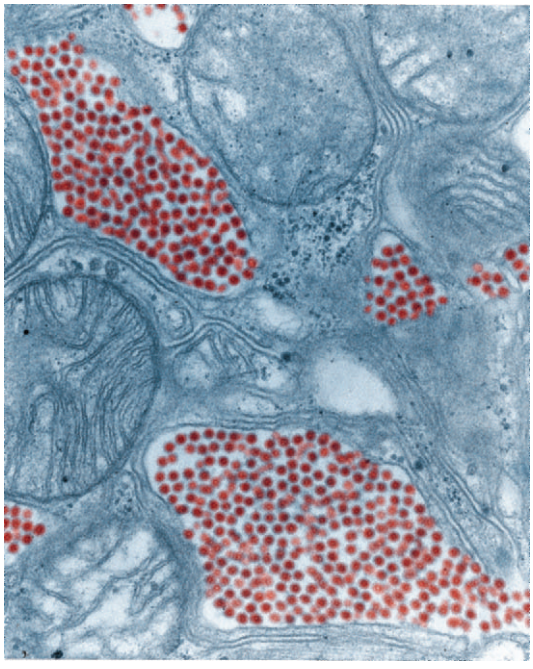

Figure 42-16. Micrograph of eastern equine encephalitis virus.

Clinical presentation can be within the spectrum from mild influenza-like illness to encephalitis, coma, and death. One study collecting the 36 cases from 1988 to 1994 in the United States showed that clinical signs included fever (83%), headache (75%), nausea and vomiting (61%), confusion (4%), myalgias and arthralgias (36%), chills (25%), seizures (25%), focal weakness (23%), abdominal pain (22%), respiratory symptoms (11%), and cranial nerve palsies (8%).[26] The case fatality rate for EEE is 35%, and 35% of persons who survive are left with mild to severe neurologic deficits.[20]

Diagnosis

Peripheral blood may show leukocytosis, and analysis of CSF may reveal elevated protein with red and white cells in the range of 600 to 2000/mm^3 (white cells being predominantly lymphocytes). Definitive diagnosis lies in serologic evidence based on paired sera (acute and convalescent samples) in hemagglutinin-inhibition assays or in neutralization assays. Acute samples should be obtained within 10 days of symptom onset, and convalescent samples about 2 or 3 weeks after the symptom onset. IgM antibodies may also be captured by ELISA. It is possible to isolate the virus directly from the serum in acute infection, but this method is less common.[60]

Other helpful diagnostic modalities include MRI, which may show focal changes, particularly in the basal ganglia and thalamus.[26,34]

Treatment and Prevention

No specific treatment is available. There has been one case report of a child being treated with intravenous immunoglobulin (IVIg),[30] but this has not been further studied. No human vaccine is available, but there are equine vaccines that should be employed in endemic areas.

Surveillance and Reporting

EEE is a nationally notifiable disease.

Australian Encephalitis (Murray Valley Encephalitis)

Epidemiology and Transmission

Occurring mainly in Australia and New Guinea, Murray Valley encephalitis (MVE) virus is from the family Flaviviridae, genus *Flavivirus*, and is transmitted via a bird-mosquito-bird cycle with *Culex annulirostris*. Because humans do not have high levels of viremia, they are seen as dead-end hosts.

Clinical Presentation

Most infections are asymptomatic, and only one in 1000 to 2000 infections actually develops into clinical illness.[57] The clinical course is similar to that of JE, with headache, fever, myalgias, nausea and vomiting, and anorexia. The neurologic involvement can involve lethargy, irritability, confusion, or meningismus, as well as seizures, spastic paresis, and coma. Persons who succumb to progressive MVE infection have a poor prognosis; more than 30% die and 50% suffer neurologic sequelae, with a bimodal risk for children and older adults.

Diagnosis

As is true for many other viral illnesses, definitive diagnosis of MVE is by identifying IgM antibody via immunoassay of the CSF. Direct viral isolation or PCR can also prove the infection in tissue, blood, or CSF. However, these tests can be obscured by cross-reactive antibodies to Kunjin, JE, Edge Hill, Alfuy, Sepik, dengue, and other flaviviruses.[79]

Two-thirds of patients have normal CT scans. If the scans are abnormal, they may show nonspecific signs of encephalitis, including mild hydrocephalus and cerebral edema.[49]

Treatment and Prevention

There is no specific treatment or vaccine for MVE.

California (La Crosse) Encephalitis

La Crosse (LAC) virus is a California serogroup bunyavirus that can cause encephalitis (usually in children) in the central and eastern United States. La Crosse encephalitis comprises 8% to 30% of all cases of encephalitis in the United States, and it is one of the major causes of arboviral illness.[70] In fact, its incidence in endemic areas (20 to 30 cases per 100,000 population per year) exceeds that of bacterial meningitis.[63]

Epidemiology and Transmission

Aedes triseriatus is the main vector (although *A. albopictus*, or Asian tiger mosquito, may be emerging as another significant species), with vertebrate hosts such as chipmunks, tree squirrels, and foxes. The annual incidence is about 60 to 130 cases.[66] Most infections are asymptomatic and occur from late summer to early fall in the northern United States.[28]

Clinical Presentation

For 80% to 90% of infected persons, the clinical course is mild, with only headache, fever, and vomiting after an incubation period of 3 to 7 days. Approximately 10% to 20% of infected individuals develop a severe form of the disease within the first 8 to 24 hours of symptom onset.[62] In one pediatric study of 127 hospitalized children, it was noted that 70% of patients presented with headache, fever, and vomiting. Of children, 46%

had seizures (generally focal seizures, with some progression to status epilepticus), and 20% had focal neurologic findings. Encephalitis does not always occur, as aseptic meningitis was also found. Although all patients survived, over 10% were left with neurologic deficits.[61] Only one case report has mentioned California encephalitis causing infarction of the basal ganglia, leaving a child with acute hemiparesis.[54] The case fatality rate is less than 1%.

Diagnosis

Laboratory findings may reveal leukocytosis with polymorphonuclear leukocyte predominance and pleocytosis in the CSF (either neutrophilic or lymphocytic predominance), although studies of laboratory values for LAC and non-LAC cases show no statistically significant differences.[45] More than half of patients have abnormal electroencephalography. A CT scan may show nonspecific cerebral edema, if there are any findings at all, and MRI may reveal gadolinium enhancement in cortical areas.[61]

Definitive diagnosis is made with IgM antibody by capture immunoassay of CSF, a fourfold rise in serum antibody titers against LAC virus, or isolation of virus from or demonstration of viral antigen or genomic sequences in tissue, blood, or CSF.[7] As in almost all other viral infections discussed in this chapter, IgM antibody capture with ELISA is the most common method, with the recommendation to confirm IgG antibody with another serologic assay such as neutralization.

Treatment and Prevention

No specific treatment is available besides general measures to control cerebral edema and seizures. Certain studies have looked at ribavirin,[62] but there is no strong evidence to recommend its use. No vaccine is available against this virus.

Epidemic Polyarthritis (Ross River Virus)

A mosquito-borne alphavirus in the family Togaviridae, Ross River virus (RRV) is a single-stranded, enveloped RNA virus in the same family as Chikungunya, Sindbis, and eastern and western equine encephalitis. The virus is endemic and enzootic in Australia (where approximately 5000 cases are reported annually) and Papua New Guinea and causes epidemic polyarthritis.[46] In the late 1970s, RRV spread to the South Pacific islands, but it disappeared after the end of an epidemic in which 500,000 people were infected; recent reports, however, show that there are new cases reappearing in Fiji.[50]

Epidemiology and Transmission

The main vectors are *Culex* and *Aedes* (particularly *Aedes vigilax*) mosquitoes, and marsupials such as kangaroos and wallabies are important as vertebrate-amplifying hosts.[69] Vertical transmission is possible from mosquito to offspring, and human-mosquito-human transmission is also thought to occur. Tropical coastal regions with salt marsh habitats are the habitat for the mosquito vector species, and infections are most common in the spring, after summer rains, or after inundation of salt marshes by rain or tides.[50]

Clinical Presentation

After inoculation, the virus is thought to replicate within macrophages. It causes local cell-mediated immune responses that appear to be the cause of arthritis. After an incubation of 3 to 9 days (or as long as 21 days), patients can present either with acute febrile illness with arthritis and rash; with acute fever, rash, and arthritis alone; with polyarthralgia; or with arthritis.[32] The joints involved are usually symmetric and include the wrists, knees, ankles, and metacarpophalangeal and interphalangeal joints of the hands, with true arthritis present in 40% of infected individuals. About 50% have fever and a sparse maculopapular rash on the limbs and trunk that may occur before or after the arthritis. Whereas fever may resolve within several days, rash may persist for several months. Joint symptoms, on the other hand, have been reported to last up to 3 years, even after fever and rash have subsided.[60]

Diagnosis

There are no specific laboratory findings for RRV infection, with normal leukocyte counts (although mild neutrophilia or atypical lymphocytes can be found) and possibly elevation in the nonspecific erythrocyte sedimentation rate. Joint aspiration may reveal viscous fluid with predominantly mononuclear cells, and points to viral arthritis.[32]

Serum can be screened for IgM antibody against Ross River virus IgM, which can then be confirmed by PRNT. These tests are available at only a few reference laboratories, including the CDC.[50] Viral culture and PCR are not commonly used for diagnosis of RRV infection.

Treatment and Prevention

There is no specific treatment. Management of arthralgias and myalgias is with standard analgesic and nonsteroidal anti-inflammatory drugs. There does not seem to be any direct association of mortality with RRV.

Jamestown Canyon Virus

Jamestown Canyon virus is a widely distributed California serogroup bunyavirus that appears to be responsible for several cases of encephalitis in the United States. It is transmitted by *Culiseta inornata* as well as several species of *Aedes* mosquitoes; the vertebrate reservoir has been identified as the white-tailed deer.[33] These infections are rare. There is a case report of Jamestown canyon virus–induced encephalitis in an 8-year-old girl in rural southwestern Michigan, who presented with symptoms of headache and fever that quickly progressed to seizures and coma.[37] The infection is difficult to distinguish from other California serogroup bunyaviruses such as California encephalitis, snowshoe hare, Keystone, and trivittatus viruses, because of cross-reactivity in the more common serologic tests and difficulty in isolating the actual virus.[80] The virus has been posited as a possible emerging infectious disease,[37] but there have been few identified cases and the literature on this subject is scant.

MOSQUITO CONTROL

General Guidelines for Individual Protection

For personal protection and prevention, individuals can take precautions such as avoiding mosquitoes at their most active time (from dusk to dawn) and wearing loose-fitting cotton clothing covering their arms and legs (see Chapter 41). Individuals may also apply repellents to exposed areas of skin. The most effective preparations contain *N,N*-diethyl-3-methylbenzamide (DEET), and generally concentrations need not exceed 20% to 35%.[31] DEET is safe in children and pregnant women,[23] but it has rare side effects of neurologic toxicity and should be used sparingly to avoid systemic absorption, and it should be kept away from mucous membranes. Studies have shown that

a single application of a product containing 24% DEET confers an average of 5 hours (and up to 12) of protection against *A. aegypti* mosquitoes. Compared with DEET, newer formulations containing the compound IR3535 fared poorly.[31] Permethrin-containing compounds and other residual insecticides that kill rather than simply repel mosquitoes may be applied to clothing and netting. Other strategies include using mosquito coils and sprays containing pyrethroids in sleeping areas. Citronella has been also shown to be mildly effective in reducing the number of bites, but it requires frequent applications if used topically (as opposed to the candle form).[30] Current research in insect repellents involves a sophisticated appreciation of the olfactory senses of mosquitoes, and creating proteins to inactivate human-specific odorants.[71]

Global Programs

In general, global eradication programs[5] for all infectious diseases, including mosquito vector surveillance and control, have declined dramatically in the past few decades. Because of the success of control programs in the 1970s and the resultant decreased public health threat, less attention has been paid and fewer resources have been allocated to maintaining good control. One result is fewer trained personnel, because training programs are less well funded. The effect has been detrimental, with a reversal of previous gains. In Central and South America, for example, dengue fever was largely eliminated with eradication in the 1950s and 1960s of the *A. aegypti* mosquito, the main vector for the disease. However, because these programs were largely dismantled in the 1970s, the mosquito species has re-infested most of these tropical regions.[39]

Vector control has three arms: environmental management, biologic control, and chemical control. Environmental management consists of monitoring and intervening to reduce the vector population, as well as transmission factors that sustain the human-vector-pathogen contact. For example, simply removing old tires, covering water storage containers with tight lids, keeping floor drains cleaned and covered, and chlorinating ornamental pools and fountains (or populating them with larvivorous fish) can help prevent breeding of mosquitoes.

Biologic control includes using organisms that naturally prey on the vector. For example, larvivorous fish and *Bacillus thuringiensis* H-14 (BTI) are commonly used against larvae. Biologic control, however, requires expense and time to raise these organisms, and the organisms may have limited success when the environment in which they are placed does not sustain their existence. Furthermore, even if they are effective in reducing the number of larvae, there is not conclusive evidence to show that this decreases transmission of disease, because a lower density of larvae in a certain area may mean healthier and stronger adult mosquitoes if food is limited.

Chemical control is the most well known vector control. It consists of using products such as pyrethrins (e.g., deltamethrin, resmethrin, permethrin) or organophosphate insecticides (e.g., fenthion, malathion, fenitrothion, temephos). These can be applied in several forms: larvicidal application, perifocal treatment, or space spraying. Larvicidal application is focal treatment to domestic water supplies that cannot be eliminated (e.g., for *A. aegypti,* either 1% temephos or methoprene to regulate larvicidal growth, or BTI can be used to safely treat drinking water). Insecticides such as fenthion, malathion, and fenitrothion can be used as perifocal treatment for nonpotable sources of water to eliminate larvae and adult mosquitoes. Finally, space spraying can be used against adult mosquitoes, often in emergency situations. The chemicals mentioned here can be used as aerosols or mists applied by portable machines, vehicle-mounted generators, or aircraft. Parameters for use (e.g., grams of active ingredient per hectare, wind conditions that may limit effectiveness) depend on the type of chemical, the equipment, and the target vector. Table 42-3 shows selected insecticides and dosages for cold-spray control of *A. aegypti.* Precautions must be taken seriously when using these products, such as following instructions on labels, using physical protection such as gloves and masks, and correctly disposing of chemicals.[87]

TABLE 42-3. Insecticides for Cold-Spray Control of *Aedes aegypti*

INSECTICIDE	DOSAGE (grams of active ingredient per hectare)
Malathion	112–693
Fenitrothion	250–300
Naled	56–280
Pirimiphos-methyl	230–330
Deltamethrin	0.5–1
Resmethrin	2–4
Bioresmethrin	5
Permethrin	5
Cypermethrin	1–3
Lambda-cyhalothrin	1

From World Health Organization: Dengue Haemorrhagic Fever: Diagnosis, Treatment, Prevention and Control, ed 2. Geneva, World Health Organization, 1997.

Integrated control programs that incorporate all three methods, along with public education and campaigns, are the most effective way to control disease. This includes initiatives such as encouraging vaccination—not only of humans, but of the animal reservoirs for which vaccines are available. This includes, for example, equine vaccinations against West Nile virus.

Recently, leading health organizations have been trying to reinstate vector and disease control, as can be seen by a short history of their efforts. In the 1950s, the WHO embarked on a mission to eradicate malaria. Despite some early success, by the mid-1970s, it was increasingly difficult to achieve the eradication goal for a number of reasons, including resistance to DDT and other insecticides. However, in 1998, the WHO, the United Nations Development Programme (UNDP), the World Bank, and UNICEF established the "Roll Back Malaria" initiative with a special focus on Africa. One arm of the campaign is to encourage widespread use of antimalarial drugs in clinics and health centers with a simple diagnosis. The other objectives are to plan and implement sustainable vector control. Experience has shown that pyrethroid-impregnated bed nets may be a better global strategy for eradicating disease, as this approach is less expensive than repeated spraying of household walls, it reduces infections (and thus the human reservoir for infection in malaria), and it is more effective in terms of horizontal implementation than top-down, or vertical, programming.[2] Although source reduction through environmental management has been shown to be effective in some places,[82] it is generally very difficult and may not be feasible everywhere.[72]

Resistance to insecticides is a very real problem. However, it is often a local phenomenon, so certain chemicals and insecticides should not be completely abandoned once reports of

resistance are raised. For example, sampling sites only a few kilometers apart in Guatemala showed a large difference in resistance for *Anopheles albimanus* mosquitoes. Similarly, in the United States, resistance of *Culex* species to organophosphates is high in areas where vector control is well implemented, but it is lower in rural areas.[39]

The WHO has developed bioassays to determine resistance and keeps a database of resistance. This database, however, can be misleading because it is based on a single dataset from a single point in time that may be several years, or even decades, old and no longer relevant.[39] There are newer diagnostic methods to test resistance, including genetic linkage and physical maps, that may elucidate factors in vector competence.[3]

A further step is to detect and contain epidemics through epidemiologic surveillance, and then to train personnel and build local capacity to sustain these efforts. Vector surveillance is of primary importance, not only to learn the geographic distribution and density of mosquito vectors and to evaluate control programs but also to predict and intervene to stop the advance of preventable diseases. For mosquitoes, indices have been created to study immature and adult populations (e.g., the "house index" (the percentage of houses infected with larvae or pupae), and an index of adult mosquitoes' landing or biting rates per person-hour). Surveillance also includes verification of control measures, which includes periodically testing the vector's susceptibility to certain insecticides. Ongoing inspection of areas free of disease and taking measures to prevent re-infestation by vectors (e.g., removing standing water sources and environmental habitats, such as tires and cemetery vases) should be instituted.[87]

Many countries are committed to the idea of vector and disease surveillance (particularly aided by the work of bodies such as the Pan American Health Organization, the CDC, and the WHO), but hundreds still fall short of pursuing these initiatives and are thus ill-prepared to effectively control disease. Concerted international efforts to collect accurate information and relay it in a timely fashion is the next herculean, but worthwhile, task.

The references for this chapter can be found on the accompanying DVD-ROM.

43 Malaria

Sheral S. Patel and James W. Kazura

Malaria is a major international health problem. As global travel increases, malaria is found with increasing frequency in areas where malaria is not endemic. Human malaria is caused by four species of parasitic protozoa, *Plasmodium falciparum, Plasmodium vivax, Plasmodium ovale,* and *Plasmodium malariae,* which are transmitted by mosquitoes. Prevention of malaria infection through education, chemoprophylaxis, and personal protective measures can significantly reduce the risk of malaria morbidity in travelers. Because malaria infection can be life threatening, awareness of rapid diagnosis and management of this disease by health care providers is essential.

EPIDEMIOLOGY

Malaria is endemic throughout tropical areas of the world and is usually transmitted to humans through the bite of a female *Anopheles* mosquito (Fig. 43-1A and B). Forty-eight percent of the world's population live in malaria-endemic areas.[14] Malaria infects at least 300 to 500 million people annually, resulting in 1 to 2 million deaths.[13,43] Most malaria-related deaths occur in infants and young children, particularly in sub-Saharan Africa. *P. falciparum* and *P. malariae* are found throughout malaria-endemic areas of the world, including Latin America, sub-Saharan Africa, Asia, and the South Pacific. *P. vivax* is endemic in Latin America, Asia, and the South Pacific but uncommon in areas of sub-Saharan Africa. Both *P. falciparum* and *P. vivax* were historically endemic in temperate areas such as North America and Europe, and transmission still occurs rarely in these areas.[14] Low frequencies of *P. ovale* infection are prevalent in most malaria-endemic areas, especially in West Africa.

The risk of acquiring malaria is highest for travelers to sub-Saharan Africa and Oceania (Papua New Guinea, Vanuatu, and the Solomon Islands), intermediate in the Indian subcontinent and Haiti, and lowest (but still significant) in Southeast Asia and the Americas (Table 43-1).[8] There can be variability in transmission within endemic areas based on season, altitude, and type of travel. For example, the risk of acquiring malaria is lower for a business traveler to a Southeast Asian city staying in an air-conditioned hotel than for a backpacker hiking and sleeping in tents in East Africa. Rates of malaria in U.S. civilians have been rising as travel to malaria-endemic areas increases.[29] In 2002, there were 1337 cases of malaria in the

resistance are raised. For example, sampling sites only a few kilometers apart in Guatemala showed a large difference in resistance for *Anopheles albimanus* mosquitoes. Similarly, in the United States, resistance of *Culex* species to organophosphates is high in areas where vector control is well implemented, but it is lower in rural areas.[39]

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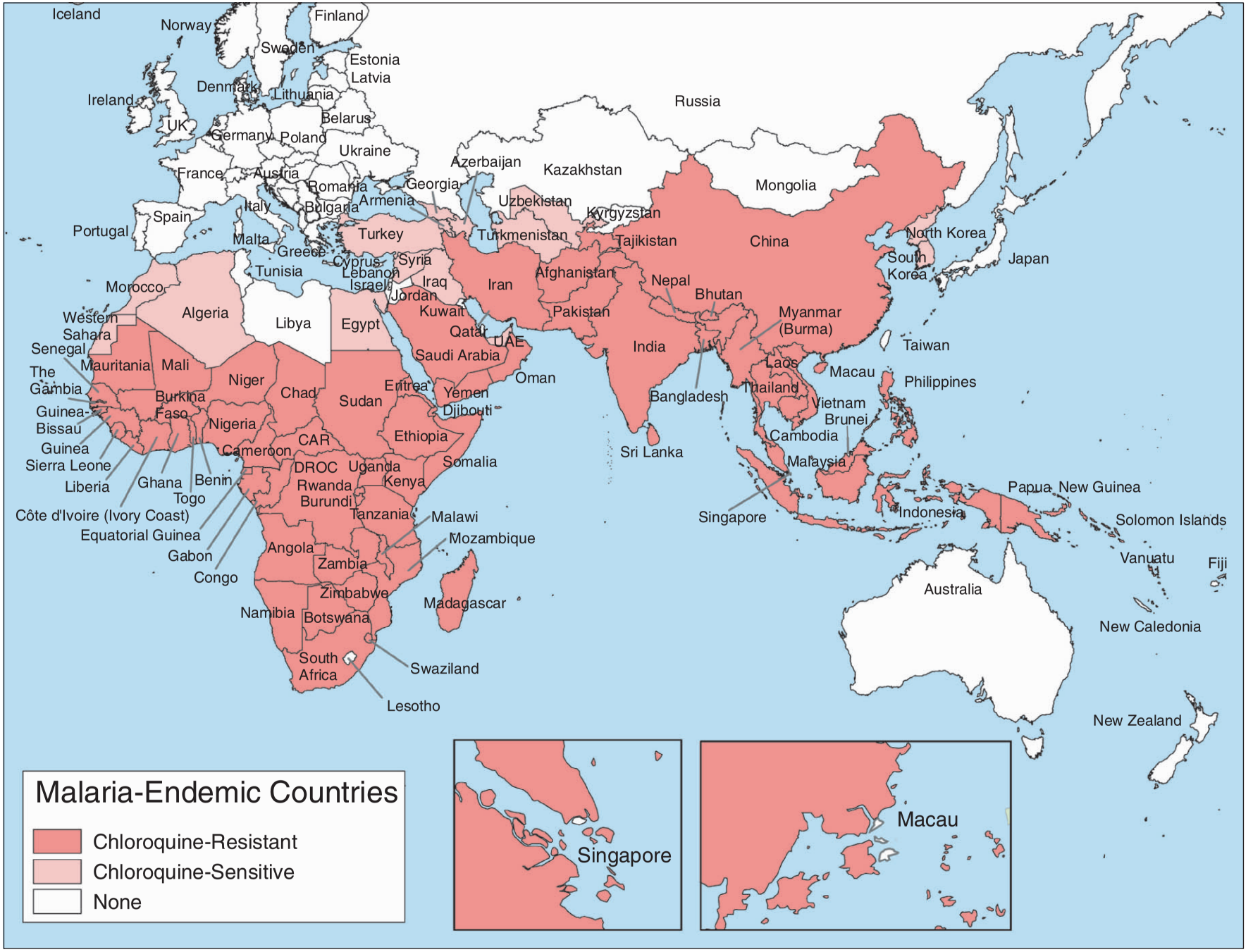
A

Figure 43-1. **A,** Distribution of chloroquine-resistant and chloroquine-sensitive malaria-endemic areas in the Americas, 2002.

United States reported to the Centers for Disease Control and Prevention.[29] The majority were imported from malaria-endemic areas of Africa (68%) and Asia (13%) and caused by *P. falciparum* (52%).[29] In addition, two cases of malaria were acquired in the United States, indicating that transmission is possible in temperate areas where *Anopheles* mosquitoes are present.

Parasite resistance to antimalarials has been increasing (Table 43-2).[41] Most parts of the world have chloroquine-resistant *P. falciparum* (see Figure 43-1A and B).[8] In addition, *P. falciparum* is becoming resistant to other antimalarials such as mefloquine, pyrimethamine-sulfadoxine, and halofantrine (Fig. 43-2).[8,27] In Southeast Asia, partial resistance of *P. falciparum* to quinine or quinidine has been reported. Areas with reports of chloroquine-resistant *P. vivax* include Southeast Asia, India, the South Pacific, Somalia, and South and Central America.

Many genetic changes in human red blood cells occur in areas where malaria is or was historically endemic, which suggests that these polymorphisms have arisen to protect individuals from malaria. Sickle cell disease, thalassemia, glucose-6-phosphate dehydrogenase (G6PD) deficiency, and Southeast Asian ovalocytosis (including Gerbich negativity) have all been



B

Figure 43-1, cont'd. **B,** Distribution of chloroquine-resistant and chloroquine-sensitive malaria-endemic areas in Africa, the Middle East, Asia, and the South Pacific, 2002. (**A** and **B** from Centers for Disease Control and Prevention (CDC): Malaria. In Health Information for International Travel, 2005–2006. Atlanta, U.S. Department of Health and Human Services, 2005. [**A** from Map 4-6, available at http://www2.ncid.cdc.gov/travel/yb/utils/ybGet.asp?section=dis&obj=index.htm#map4-6; **B** from Map 4-7, available at http://www2.ncid.cdc.gov/travel/yb/utils/ybGet.asp?section=dis&obj=index.htm#map4-7])

TABLE 43-1. Risk of Malaria by Region

RISK	REGION
High	Sub-Saharan Africa, Papua New Guinea, the Solomon Islands, Vanuatu
Intermediate	Indian subcontinent, Haiti
Low (but significant)	Southeast Asia, Central and South America

From American Academy of Pediatrics: Malaria. In Pickering LK, et al (eds): Red Book: 2006 Report of the Committee on Infectious Diseases, 27th ed. Elk Grove Village, IL, APP, 2006, pp 435–441.

TABLE 43-2. Antimalarial Drug Resistance in Malarial Parasites

PLASMODIUM SPECIES	DRUGS FOR WHICH RESISTANCE IS ESTABLISHED
P. falciparum	Chloroquine, mefloquine, pyrimethamine-sulfadoxine, halofantrine; partial resistance to quinine and quinidine; resistance to multiple drugs
P. vivax	Chloroquine, pyrimethamine-sulfadoxine, primaquine
P. ovale	None established
P. malariae	None established

suggested to confer protection from falciparum malaria mortality.[26a,39] In sub-Saharan Africa, nearly 100% of the population is negative for the Duffy blood group and has absolute protection against *P. vivax*, which depends on the Duffy antigen as a receptor for invasion.[23]

MALARIA PARASITE

Mosquito Vector

The female Anopheline mosquito is the arthropod vector for the malaria parasite (Fig. 43-3). Of almost 430 *Anopheles* species, only 30 to 40 transmit malaria (Fig. 43-4). These include *Anopheles gambiae, Anopheles funestus,* and *Anopheles arabiensis* in Africa; the *Anopheles punctulatus* group in Papua New Guinea; *Anopheles culicifacies* in India; *Anopheles darlingi* in South America; and *Anopheles quadrimaculatus* in North America.[17] The female mosquito's proboscis pierces the skin of a person to obtain the blood meal necessary to produce eggs. Distinguishing features of *Anopheles* mosquitoes include sensory palps that are as long as the proboscis, and discrete blocks of black and white scales on the wings.[25] Both male and female *Anopheles* mosquitoes rest with their abdomens sticking up in the air, as opposed to parallel to the surface on which they are resting.[25]

Life Cycle

The life cycle of the malaria parasite involves both vertebrate and arthropod hosts (Fig. 43-5). The asexual haploid form of the malaria parasite in the mosquito is the sporozoite. Transmission of 8 to 12 sporozoites usually occurs through the bite of a nocturnal-feeding female *Anopheles* mosquito (see Figure 43-3). Sporozoites then rapidly migrate through the bloodstream to the liver, where they mature and produce 10,000 to 30,000 merozoites per sporozoite. The merozoites are released into the bloodstream 8 to 25 days later to invade circulating erythrocytes (see Figure 43-5).[12] A subset of *P. vivax* and *P. ovale* parasites may remain dormant as hypnozoites in the liver and emerge as merozoites months to years after the initial inoculation to establish blood-stage infection. Merozoites enter red blood cells rapidly through specific erythrocyte receptors, many of which are yet undefined. *P. falciparum* has several invasion pathways, including glycophorin A and C, whereas *P. vivax* depends solely on the Duffy antigen for erythrocyte invasion.[44]

In the red blood cell, the asexual parasites consume hemoglobin and enlarge from the ring forms to become trophozoites and then schizonts. As the schizonts mature through multiple nuclear divisions, the red blood cell bursts, releasing 6 to 24 merozoites to invade additional circulating erythrocytes.[3] The blood-stage cycle takes place over 48 to 72 hours. Some parasites develop into gametocytes (the sexual stage), which can infect mosquitoes when taken up during a feeding. After being ingested by an *Anopheles* mosquito, diploid zygotes are formed. Zygotes mature into ookinetes in the mosquito midgut.[31] The resulting oocyst expands through meiotic reduction division within 7 to 10 days, releasing sporozoites that localize through the hemolymph to the salivary gland of the mosquito.[4] The sporozoites are subsequently transmitted to another human host at the next blood meal.

Recurrent and Persistent Infections

Recurrent malaria infection can occur in several ways. First, relapses from *P. vivax* or *P. ovale* can occur when dormant hypnozoites mature and release merozoites, thus producing blood-stage infections. Second, incomplete treatment or a partially effective host immune response may lead to recrudescence of blood-stage infections. Relapse and recrudescence are caused by the same parasite clone that was responsible for the initial infection. Although recrudescence can occur with any malarial species, it is most common with *P. falciparum* because of antimalarial resistance. Finally, in areas of intense transmission, simultaneous infection or re-infection with multiple parasite species or strains can occur. *P. malariae* is frequently associated with persistent infections that can remain in the bloodstream at undetectable levels for up to 20 to 30 years.

Transmission

Natural transmission of malaria occurs through the bite of a female *Anopheles* mosquito. Blood-stage infection can also be established by transfusions of blood or blood products, by organ transplantation, or by sharing of contaminated needles or syringes.[35] Occasionally, congenital malaria occurs when mothers of newborns are infected.[29] It may be difficult to distinguish congenital malaria from natural mosquito-borne transmission when the diagnosis is made in neonates 2 to 3 weeks after birth.

CLINICAL MANIFESTATIONS AND PATHOGENESIS

Susceptible Populations

Patients with malaria present with variable and nonspecific signs and symptoms ranging from asymptomatic parasitemia to severe disease and death. Individuals living in malaria-endemic

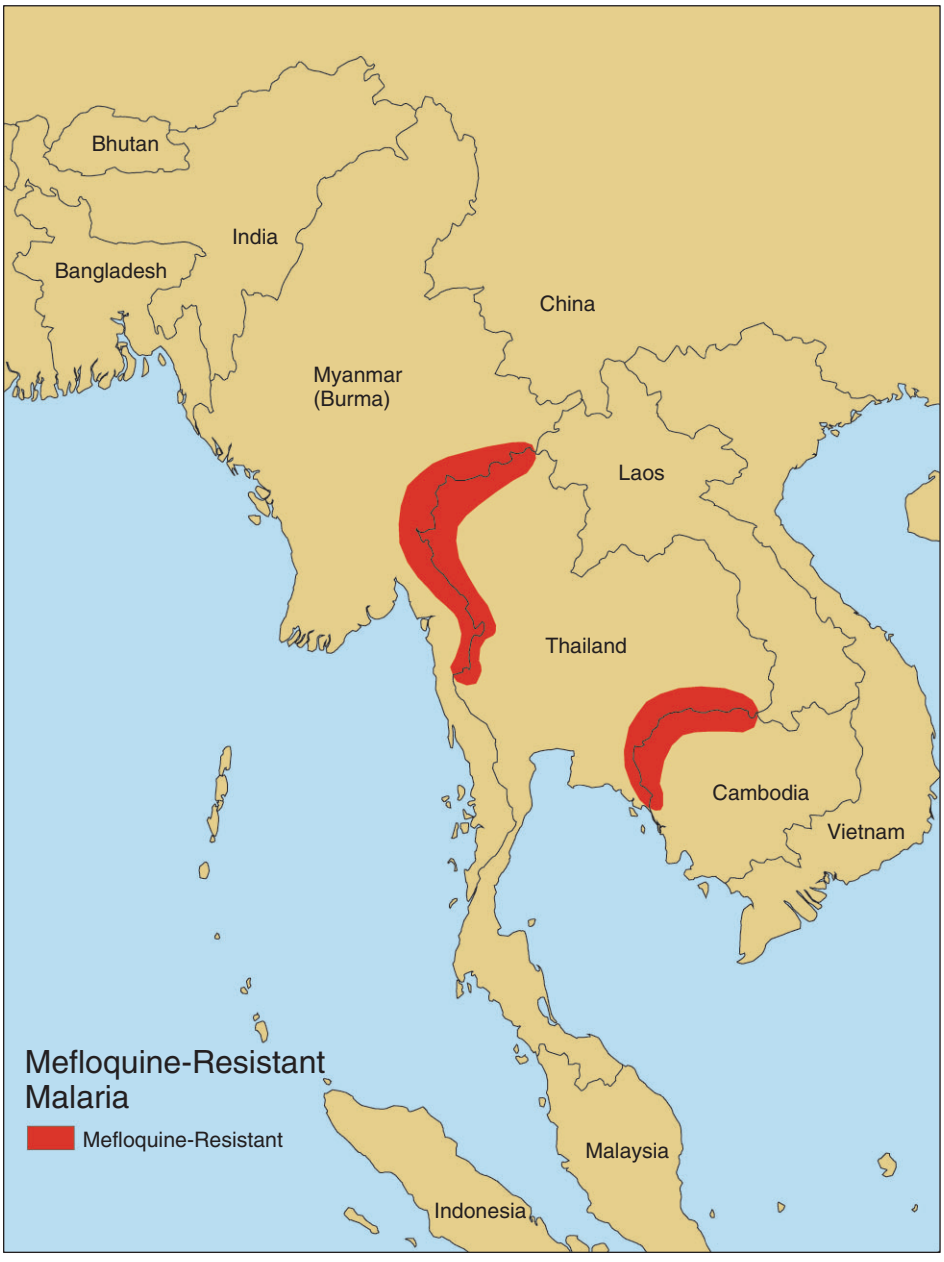


Figure 43-2. Distribution of mefloquine-resistant malaria. (From Centers for Disease Control and Prevention: Malaria. In Health Information for International Travel 2005–2006. Atlanta, U.S. Department of Health and Human Services, 2005. Map 4-8, available at http://www2.ncid.cdc.gov/travel/yb/utils/ybGet.asp?section=dis&obj=index.htm#map4-8.)

areas develop partial immunity through repeated infections with malaria parasites and rarely experience serious complications after childhood. Nonimmune individuals, such as travelers and immigrants from nonendemic areas, children (6 months to 5 years) living in endemic areas, and pregnant women, are at risk for severe disease and complications, especially with *P. falciparum* infection.[38]

Major Clinical Findings

Red blood cell lysis and the release of merozoites at the end of a period of intraerythrocytic asexual reproduction result in the classic presentation of a malarial paroxysm characterized by high fevers, chills, rigors, sweats, and headache (Table 43-3). Without appropriate therapy, paroxysms can recur in a cyclic pattern; every 48 hours with *P. vivax* and *P. ovale*, and every 72 hours with *P. malariae*. Although *P. falciparum* has a 48-hour asexual erythrocytic cycle, fever and chills typically occur without any periodicity because erythrocyte lysis is not synchronized.

Patients with malaria infection may also present with generalized weakness, backache, myalgias, vomiting, diarrhea, and pallor. If an appropriate travel history is not obtained, malaria infection can be mistaken for a viral syndrome or acute gastroenteritis. In addition, severe malaria can mimic other diseases (e.g., meningitis, typhoid fever, dengue, hepatitis) that are common in malaria-endemic countries. Partially immune indi-

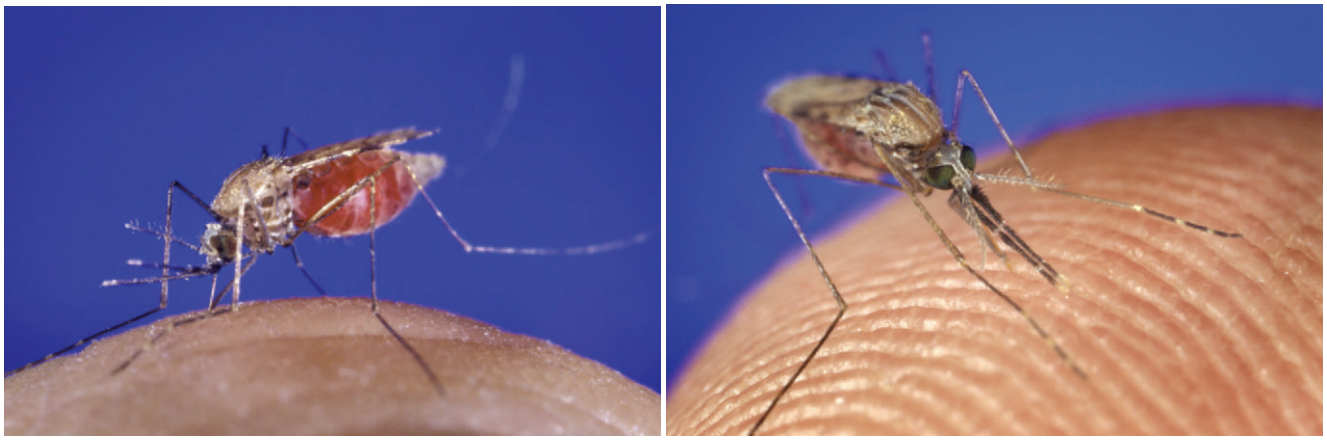


Figure 43-3. Female *Anopheles gambiae* mosquito feeding. Distinguishing features include sensory palps that are as long as the proboscis, and discrete blocks of black and white scales on the wings. Both male and female *Anopheles* mosquitoes rest with their abdomens sticking up in the air. (From Centers for Disease Control and Prevention Public Health Image Library, http://phil.cdc.gov/phil/home.asp. *Left*, image ID#1665; *right*, image ID#1664. Courtesy Dr. Jim Gathany and the Centers for Disease Control and Prevention, Public Health Information Library.)

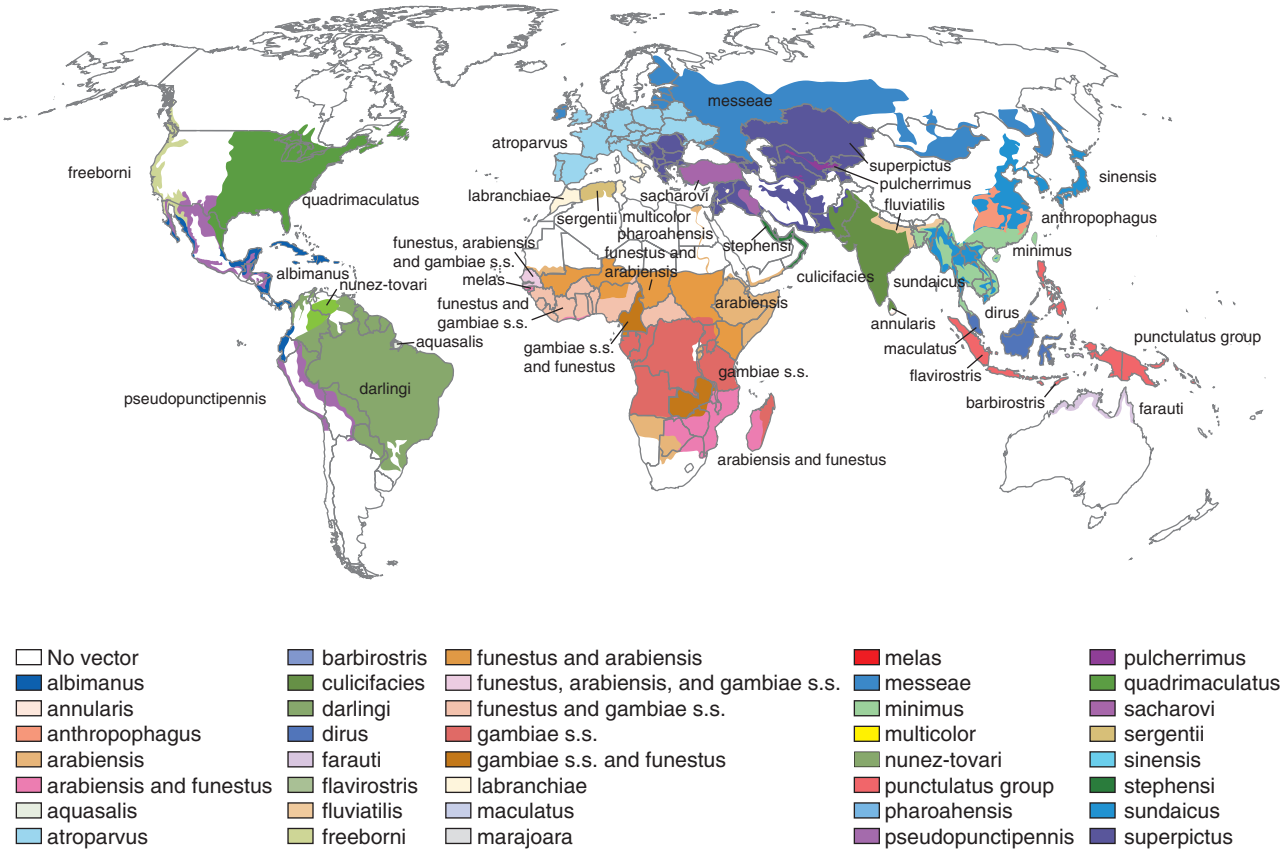


Figure 43-4. Global distribution of malaria vectors. (From Kiszewski A, Mellinger A, Spielman A, et al: Am J Trop Med Hyg 70:486–498, 2004, with permission.)

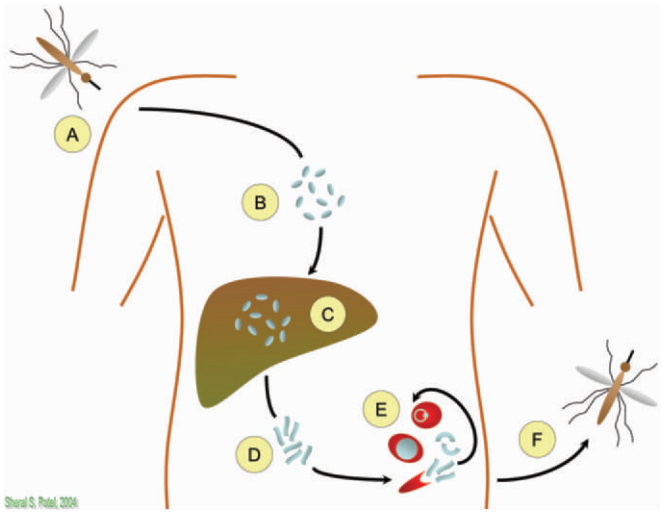


Figure 43-5. Malaria parasite life cycle. During the malarial parasite life cycle, sporozoites are transmitted through the bite of a nocturnal-feeding female *Anopheles* mosquito (**A**). Sporozoites then migrate to the liver (**B**) and mature to merozoites (**C**). A subset of *P. vivax* and *P. ovale* parasites remains dormant as hypnozoites emerging months to years after the initial infection to cause disease. Eight to 25 days after the initial infection, 10,000 to 30,000 merozoites are released to invade erythrocytes (**D**). Asexual parasites mature in 48 to 72 hours, each releasing 6 to 24 merozoites to invade more erythrocytes (**E**). Some parasites develop into gametocytes (sexual stages), which are taken up during a mosquito blood meal. Diploid zygotes form ookinetes and develop into haploid sporozoites (**F**). The sporozoites migrate to the mosquito salivary gland and continue the life cycle in humans with the next blood meal. (From Sheral S. Patel, with permission.)

TABLE 43-3. Clinical Manifestations and Complications of Human *Plasmodium* Infection

PLASMODIUM SPECIES	MANIFESTATIONS AND COMPLICATIONS
All species	Fever, chills, rigors, sweats, headache Weakness Myalgias Vomiting Diarrhea Hepatomegaly Splenomegaly Jaundice Anemia Thrombocytopenia
P. falciparum	Hyperparasitemia Cerebral malaria: seizures, obtundation, coma Severe anemia Hypoglycemia Acidosis Renal failure Pulmonary edema (noncardiogenic) Vascular collapse
P. vivax and *P. ovale*	Splenic rupture Relapse months to years after primary infection because of latent hepatic stages
P. malariae	Low-grade fever, fatigue Chronic asymptomatic parasitemia Immune complex glomerulonephritis

viduals who have recently arrived from endemic areas, such as immigrants and refugees, may be asymptomatic or have jaundice or signs of hepatosplenomegaly. Laboratory studies may reveal anemia and thrombocytopenia. Perinatal transmission of malaria is usually caused by *P. falciparum* and *P. vivax*. Clinical manifestations of congenital malaria can mimic neonatal sepsis and include fever, poor appetite, irritability, and lethargy.

Complications of *P. falciparum*

Infection with *P. falciparum* has a greater risk for complications and death than does infection with the other three human malarial species. First, *P. falciparum* can invade erythrocytes of all ages and thus produce overwhelming parasitemia. Second, *P. falciparum*–infected red blood cells adhere to endothelial cells, leading to microvascular pathology not observed with other malarial species.[22] Third, *P. falciparum* is frequently resistant to antimalarials (see Figure 43-1A and B and Figure 43-2).

Travelers from nonendemic areas, children, and pregnant women are at greatest risk for developing complications from malaria infection. A person is considered to have complicated malaria when the presenting signs include altered mental status, seizures, profound anemia, respiratory distress, gross hematuria, shock, or severe laboratory abnormalities, including hypoglycemia, acidosis, signs of disseminated intravascular coagulation, or hyperparasitemia (see Table 43-3). These are summarized as follows:

- *Cerebral malaria:* Acute neurologic events such as seizures, obtundation, and coma are associated with cerebral malaria. Cerebrospinal fluid examination is typically unremarkable, thus differentiating cerebral malaria from bacterial meningitis. Pathogenesis is a result of microvascular obstruction by parasitized erythrocytes, but neurologic impairment can result from multiple factors, including hypoglycemia, acidosis, impaired cerebral oxygenation from anemia, and pulmonary edema.[33] The mortality rate can be as high as 15% to 30% in endemic areas, and higher in nonimmune adults, such as travelers. Children surviving episodes of cerebral malaria often have subtle cognitive and motor deficits.[37]
- *Severe anemia:* Overwhelming parasitemia (>10^6 parasitized red blood cells/μL or >20% parasitized circulating red blood cells) can develop rapidly in nonimmune individuals, resulting in severe anemia (hemoglobin, <5 to 7 g/dL). The degree of parasitemia is always proportional to the degree of anemia. Moderate to low-grade parasitemia, as well as persistent or recurrent infections, in semi-immune individuals can lead to slow development of severe anemia. Factors contributing to development of anemia include red blood cell lysis and release of mature schizonts, suppression of erythropoietin by cytokines, and peripheral destruction of erythrocytes by the spleen.[22]
- *Metabolic complications:* Nonimmune individuals are at risk for hypoglycemia and acidosis. Hypoglycemia can result from decreased oral intake, depletion of liver glycogen, parasite consumption of glucose, insulin release from the pancreas by quinine or quinidine, and inhibition of gluconeogenesis.[18]
- *Renal failure:* The pathogenesis of renal failure may be multifactorial. These individuals have sequestration of parasitized erythrocytes in the cortex and findings consistent with acute tubular necrosis in the medulla. Other factors contributing to renal failure include hypotension, microvascular obstruction by *P. falciparum*–parasitized red blood cells, and free

malarial pigment or heme.[38] Renal failure is rare in children younger than 8 years. Nonimmune individuals receiving treatment with quinine or quinidine may develop renal failure from massive hemolysis, resulting from overwhelming parasitemia leading to free hemoglobin in the urine (blackwater fever).[5]

- *Pulmonary edema:* Late in the course of severe malaria, individuals can develop pulmonary edema with normal intrapulmonary and intracardiac pressures consistent with a capillary leak syndrome.[38] Sequestration of parasitized red blood cells is uncommon. Pulmonary edema is a rare complication in children.
- *Diarrhea:* Children living in malaria-endemic areas often present with watery diarrhea as a result of *P. falciparum* infection. This is thought to be secondary to sequestration of parasitized erythrocytes in the intestinal microvasculature. The precise mechanism is not known, but cytokines may also contribute to a secretory diarrhea through tumor necrosis factor, prostaglandins, and cyclic AMP.

Factors indicating a poor prognosis for persons with severe malaria include clinical, biochemical, and hematologic features (Table 43-4).[40] Poor clinical prognostic indicators include impaired consciousness, repeated convulsions, respiratory distress, bleeding, and shock. Biochemical indicators include renal impairment, acidosis, jaundice, hyperlactatemia, hypoglycemia, and elevated aminotransferases. Finally, hematologic features indicative of a poor prognosis include a hemoglobin level of less than 5 g/dL, parasitemia of greater than 500,000 parasites per milliliter, greater than 10,000 mature trophozoites and schizonts per milliliter, or malaria pigment in ≥5% of neutrophils.

TABLE 43-4. Indicators of a Poor Prognosis in Severe Malaria

Indicator	FACTOR	COMMENTS
Clinical	Age < 3	
	Impaired consciousness	
	Seizures ≥3 in 24 hr	
	Absent corneal reflexes	
	Papilledema	
	Decerebrate/ decorticate rigidity	
	Opisthotonus	
	Respiratory distress	
	Shock	
Biochemical	Hypoglycemia	Glucose <40 mg/dL
	Acidosis	Plasma bicarbonate <15 mmol/L
	Hyperlactatemia	Lactate >45 mg/dL
	Renal impairment	Serum creatinine >3 mg/dL; BUN >60 mg/dL
	Elevated aminotransferases	>3 times normal
	Hyperbilirubinemia	Serum total bilirubin >2.5 mg/dL
Hematologic	Hyperparasitemia	>500,000 parasites/mL or >10,000 mature trophozoites and schizonts/mL
	Anemia	Hemoglobin <5 g/dL; packed cell volume <15%
	Visible malarial pigment	>5% neutrophils with malarial pigment

BUN, blood urea nitrogen.

Complications of *P. vivax* and *P. ovale*

Individuals with *P. vivax* infection typically present with fevers, chills, headaches, and myalgias. Individuals with *P. ovale* infection do not usually present with a toxic appearance. Anemia can result from acute *P. vivax* or *P. ovale* parasitemia. Anemia and splenomegaly are common in individuals chronically infected with either *P. vivax* or *P. ovale,* and splenic rupture is an important late complication (see Table 43-3).[45] Because both *P. vivax* and *P. ovale* have a hepatic hypnozoite stage, symptoms may present months to years after the initial infection.

P. vivax invades reticulocytes, resulting in lower levels of parasitemia than seen with *P. falciparum* infection. Cytoadherence does not occur with *P. vivax* or *P. ovale*. Thus, all stages of the parasite circulate in the peripheral blood, and microvascular changes associated with sequestration in deep vascular beds are not observed. Individuals lacking expression of the Duffy blood group antigen, such as many Africans, are protected from *P. vivax* infection because this antigen is used as a receptor for erythrocyte invasion by the *P. vivax* parasite.[22]

Complications of *P. malariae*

Because *P. malariae* produces persistent low-level parasitemia, it usually manifests with mild symptoms such as weakness, low-grade fever, and fatigue. Individuals can have chronic asymptomatic parasitemia for years. This can be associated with an immune complex glomerulonephritis, where complement-fixing antibodies are directed against *P. malariae* antigens.[10]

DIAGNOSIS

History

The most important factor in the diagnosis of malaria, particularly in nonendemic areas, is for the health care provider to consider it in the differential diagnosis. Because of the risk of complications and death, fever in the returned traveler should be taken seriously. A thorough travel history must be obtained, including travel dates, destinations, and any chemoprophylaxis taken.[20] Because of drug resistance and the possibility of inadequate adherence to medication schedules, a history of chemoprophylaxis use in a returned traveler does not exclude a diagnosis of malaria. The patient may not have the typical malarial paroxysms of fever and chills. Nonspecific symptoms, such as fatigue, diarrhea, headache, myalgias, or sore throat, may lead to another diagnosis. Fever and persistent malaise may occur in semi-immune individuals.

Blood Smears

The thin and thick blood smear is the gold standard for the clinical diagnosis of malaria (Fig. 43-6A and B).[30] Blood smears should be interpreted by skilled microscopists trained in malaria diagnosis.

Blood Smear Preparation

Thin smears are used to determine erythrocyte size as well as parasite morphology. A drop of blood is placed at one end of a slide and drawn across the slide with the edge of another slide (see Figure 43-6B).[30] Individuals with low parasitemia may have false-negative thin blood smears because the number of red blood cells examined in the thin smear is insufficient to detect the parasites. In these cases, thick smears are useful in establishing a diagnosis of malaria. Thick smears are made by placing a drop of blood on a smear, spreading it in a circle, and allowing it to dry (see Figure 43-6B).[29] The red blood cells are lysed by staining without methanol fixation. Thus, the degree of parasitemia can be ascertained, but the size and morphology of

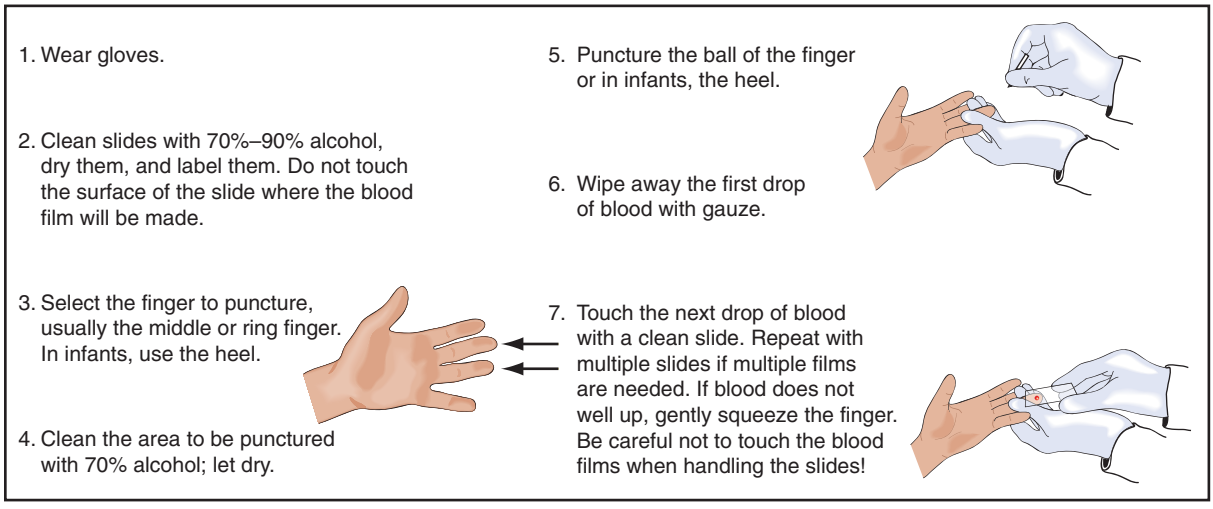


Figure 43-6. **A,** Blood collection method for blood smear preparation. **B,** Preparation of thin and thick blood smears. (From Centers for Disease Control and Prevention: MMWR 53(SS01):35–36, 2004, available at www.cdc.gov/mmwr/PDF/SS/SS5301.pdf.)

parasitized red blood cells cannot. Giemsa is the preferred stain for malaria smears because details such as Schüffner's dots are often missed with Wright's stain. However, Wright's stain is generally more available in malaria-nonendemic areas than is Giemsa, and it usually allows one to determine the presence of parasites.

Blood Smear Examination

Oil immersion (×1000) is used to examine thick and thin blood smears (Table 43-5 and Figs. 43-7 and 43-8). Examination of the thin smear should begin at the thin edge of the blood smear, farthest away from where the drop of blood was placed (see Figures 43-7 and 43-8). *P. falciparum* ring stages have a delicate cytoplasm with one to two small chromatin dots and occasional appliqué forms.[6] *P. falciparum* trophozoites and schizonts are rarely seen in peripheral blood, and gametocytes have a crescent or sausage shape. Red blood cells infected with more than one parasite are more common with *P. falciparum* than with the other species. *P. vivax* ring stages have a large cytoplasm with occasional pseudopods and a large chromatin dot.[6] Occasional

TABLE 43-5. Characteristics of *Plasmodium* Species on Human Blood Smear

	INFECTED RED BLOOD CELLS				PARASITES		
PLASMODIUM SPECIES	SIZE	SHAPE	SCHÜFFNER'S DOTS?	RINGS	TROPHOZOITES	SCHIZONTS (MATURE)	GAMETOCYTES
P. falciparum	Normal	Crescent (gametocyte)	No	Numerous delicate rings only (occasional gametocytes) Multiply infected red blood cells Numerous, appliqué forms	—	8–24 (rare)	Crescent
P. vivax	Enlarged	Ameboid	Yes	Thick	Ameboid	12–24	Round
P. ovale	Enlarged	Fimbriated, elongated, oval	Yes	Thick	Compact	6–14	Round
P. malariae	Normal	—	No	Thick	Compact, band form	6–12, rosettes	Round

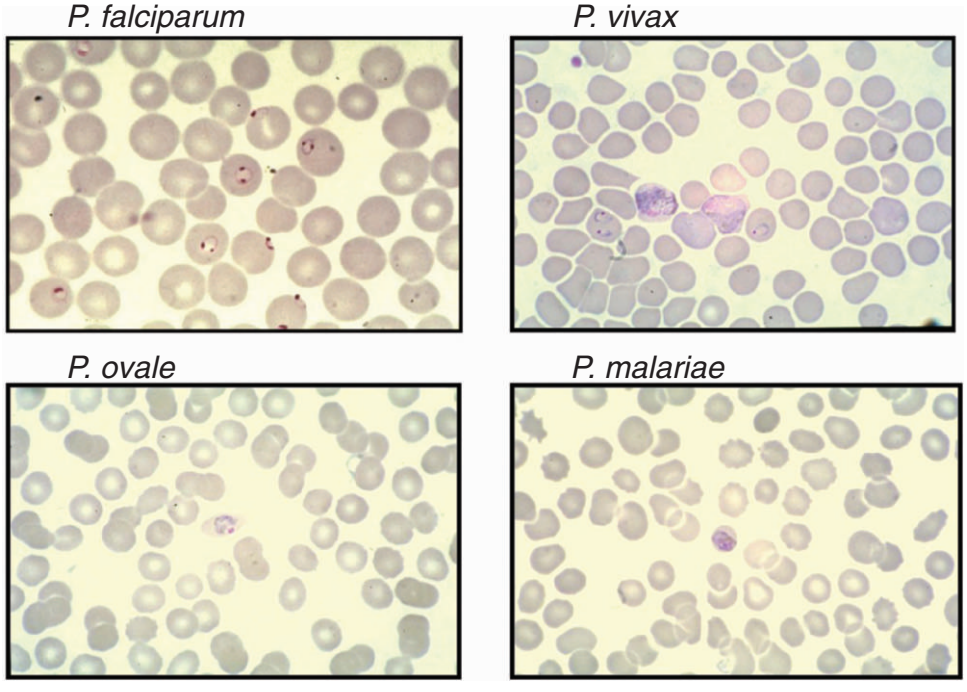
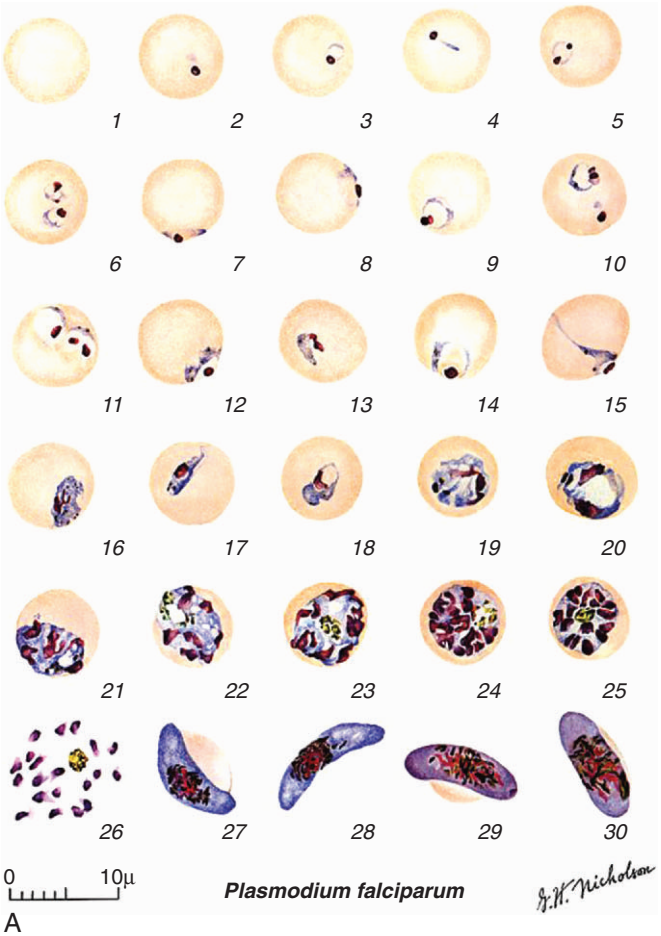


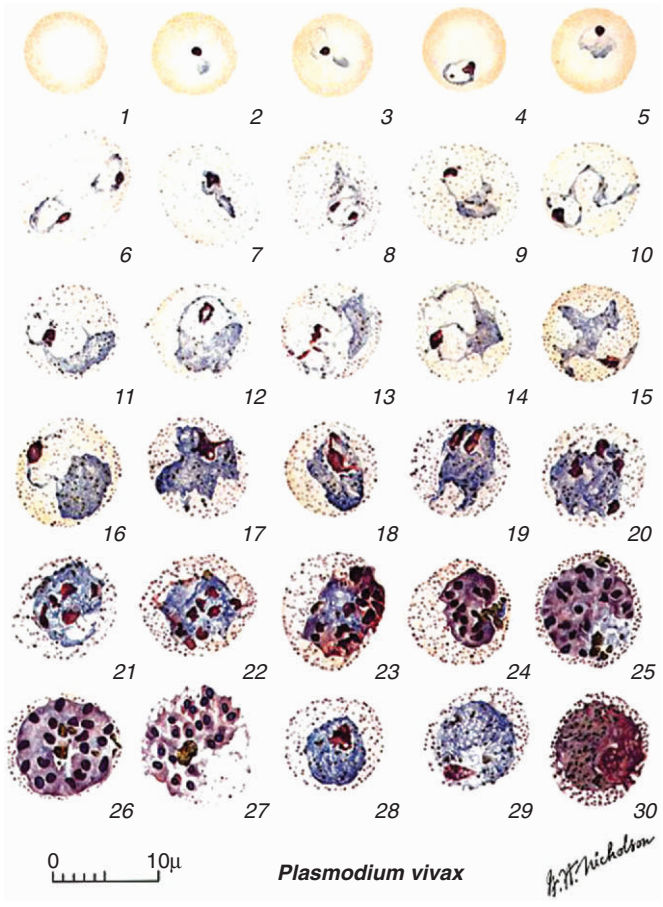
Figure 43-7. Representative malaria blood smears. *P., Plasmodium*. (Photographs courtesy Royal Perth Hospital, Perth, Western Australia, Australia; available at www.rph.wa.gov.au/malaria/teach.html.)

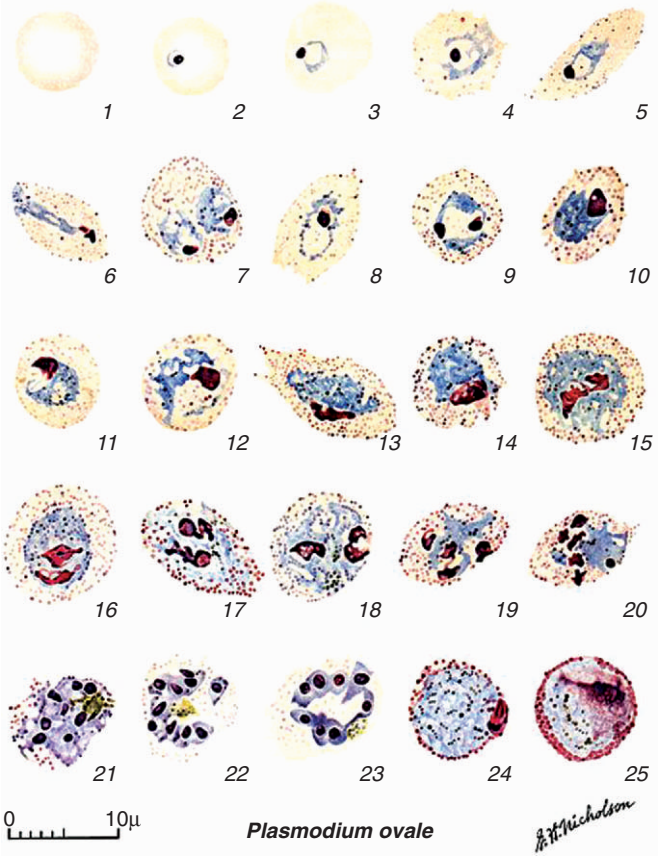
Schüffner's dots are seen in the red blood cell. *P. vivax* trophozoites are large with an amoeboid cytoplasm; schizonts contain 12 to 24 merozoites. Gametocytes of *P. vivax* are round to oval in shape. Ring stages of *P. ovale* have a large chromatin dot. *P. ovale*–infected red blood cells occasionally have Schüffner's dots and are fimbriated.[6] *P. ovale* trophozoites are compact with large chromatin, and the schizonts contain 6 to 14 merozoites with large nuclei. The gametocytes of *P. ovale* are round to oval in shape. *P. malariae* rings also have large chromatin.[6] *P. malariae* trophozoites have a compact cytoplasm with an occasional band form. Schizonts contain 6 to 12 merozoites with large nuclei, and the gametocytes are round to oval.

Parasite Density

Parasite density is usually calculated on the thick smear by counting the number of parasites per 200 white blood cells (WBC). The actual number of parasites per microliter of blood is then calculated based on an average of 8000 WBC/μL, or on the actual WBC count if it is available.[19] False-positive blood smears are common when inexperienced microscopists mistake platelets or stain debris for parasites. Most false-negative blood smears result from low parasitemia (often with the semi-immune state) or altered parasite morphology related to chemoprophylaxis, partial treatment, or drug-resistant parasites. If the initial blood smears are negative for *Plasmodium* species,







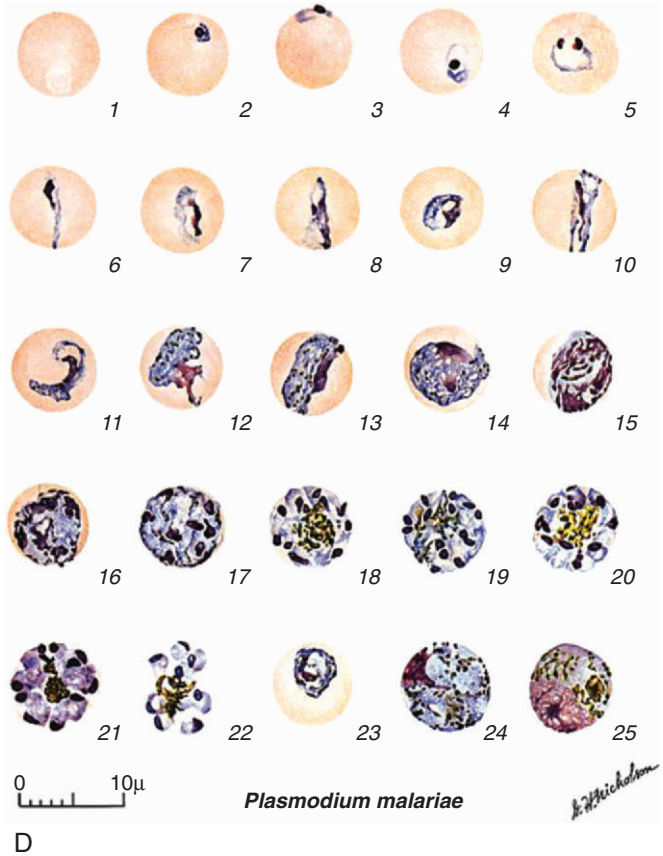
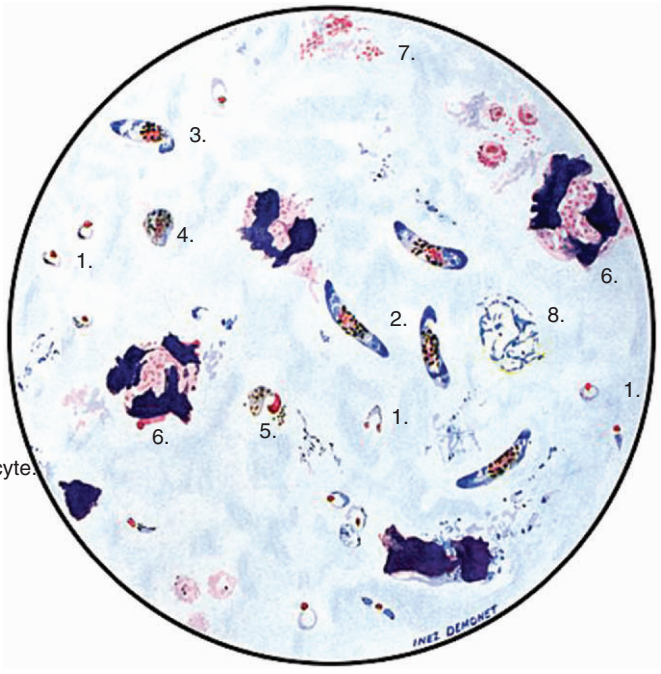


Figure 43-8. **A,** Thin smears, *Plasmodium falciparum*. **B,** Thin smears, *Plasmodium vivax.* **C,** Thin smears, *Plasmodium ovale.* **D,** Thin smears, *Plasmodium malariae.*

Plasmodium falciparum
Blood stage parasites,
thick blood smears

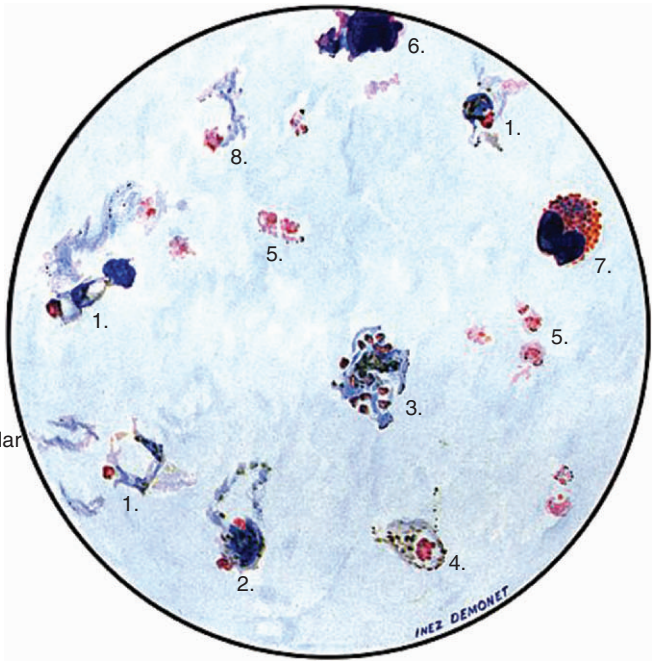
1: Small trophozoites.
2: Gametocytes – normal.
3: Slightly distorted gametocyte.
4: "Rounded-up" gametocyte.
5: Disintegrated gametocyte.
6: Nucleus of leukocyte.
7: Blood platelets.
8: Cellular remains of young erythrocyte.



E

Plasmodium vivax
Blood stage parasites,
thick blood smears

1: Ameboid trophozoites.
2: Schizont – 2 divisions of chromatin.
3: Mature schizont.
4: Microgametocyte.
5: Blood platelets.
6: Nucleus of neutrophil.
7: Eosinophil.
8: Blood platelet associated with cellular remains of young erythrocytes.



F

Figure 43-8, cont'd. **E,** Thick smears, *Plasmodium falciparum.* **F,** Thick smears, *Plasmodium vivax.*

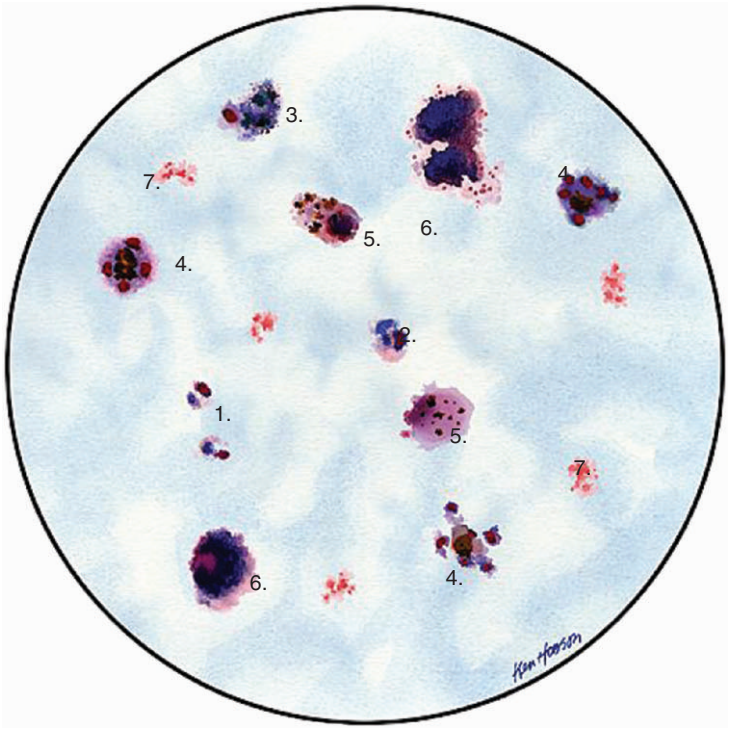
but malaria is still a leading diagnosis, the smear should be repeated every 12 to 24 hours during a 72-hour period.

Other Diagnostic Methods

Most of the alternative nonmicroscopic methods of malaria diagnosis are more applicable to malaria-nonendemic areas because their reagent costs are much greater. The sensitivity of many of these tests is equal to or greater than that of the blood smear for the diagnosis of malaria.[24] These tests include (1) fluorescence microscopy, where parasites are stained by acridine orange, (2) immunologic detection of *Plasmodium* histidine-rich protein 2 or parasite lactate dehydrogenase, and (3) DNA probes for parasite-specific sequences. Commercially available

Plasmodium ovale
Blood stage parasites, thick blood smears

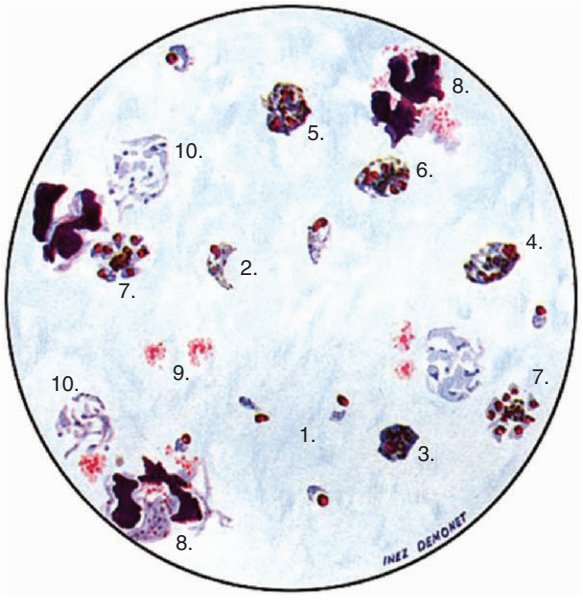
1: Small trophozoites.
2: Growing trophozoites.
3: Mature trophozoites.
4: Schizonts.
5: Gametocytes.
6: Nucleus of leukocyte.
7: Blood platelets.



G

Plasmodium malariae
Blood stage parasites, thick blood smears

1: Small trophozoites.
2: Growing trophozoites.
3: Mature trophozoites.
4, 5, 6: Immature schizonts with varying numbers of divisions of the chromatin.
7: Mature schizonts.
8: Nucleus of leukocyte.
9: Blood platelets.
10: Cellular remains of young erythrocytes.



H

Figure 43-8, cont'd. **G,** Thick smears, *Plasmodium ovale.* **H,** Thick smears, *Plasmodium malariae.* (**A** through **D** from Coatney GR, Collins WE, Warren M, Contacos PG. The Primate Malarias. Bethesda, MD, U.S. Department of Health, Education and Welfare, 1971, available at www.dpd.cdc.gov/dpdx/HTML/PDF_Files/Malaria%20Comparison%20p3-6.pdf; **E** through **H** from Wilcox A: Manual for the Microscopical Diagnosis of Malaria in Man. Washington, DC, U.S. Department of Health, Education and Welfare, 1960, available at www.dpd.cdc.gov/dpdx/HTML/PDF_Files/Malaria%20Comparison%20p7-8.pdf.)

kits are available for the enzyme linked immunosorbent assay of histidine-rich protein 2 or parasite lactate dehydrogenase and can be completed in 10 minutes. These kits have a sensitivity of 49% to 99% for the detection of *Plasmodium* infection and are useful in areas where experienced microscopists may not be available.[24]

PREVENTION

Prevention of malaria infection requires appropriate counseling, chemoprophylaxis, and personal protective measures (Table 43-6).

TABLE 43-6. Checklist for Malaria Prevention in Travelers

Counseling	Importance of chemoprophylaxis Potential adverse reactions or side effects from chemoprophylaxis Clinical manifestations of malaria Plan for urgent medical care
Chemoprophylaxis	Assess malaria risk Determine malaria resistance patterns Consider: Age Underlying medical condition Allergies Tolerability Length of stay Advise when to start medication and how long to continue after return Determine whether medication should be prescribed for presumptive self-treatment
Personal protective measures	Minimize outdoor activity at dusk, nighttime Long sleeves, long pants, and hats during dusk, nighttime Insect repellent 10% to 35% DEET (*N*,*N*-diethyl-3-methylbenzamide) for exposed skin Permethrin for clothes, shoes, mosquito nets, tents, other gear Insecticide-treated bed nets Mosquito coils and candles

Counseling

The risk of malaria and the importance of malaria chemoprophylaxis and personal protective measures must be emphasized with each traveler.[28] Potential side effects of drugs prescribed for malaria prophylaxis as well as the signs and symptoms of malaria should be discussed. Because treatment is most effective early in the course of disease, a delay in appropriate therapy can have potentially serious or fatal consequences. A plan for where to obtain urgent medical care should be in place *prior* to traveling to any malaria-endemic area.

Chemoprophylaxis

Chemoprophylaxis is a strategy for preventing malaria by administering medications. Primary prophylaxis uses medications prior to, during, and after travel to prevent disease by the initial infection (inhibiting the replication of asexual parasites that have emerged from the liver), whereas terminal prophylaxis uses medications toward the end of the exposure period to prevent relapse or delayed-onset of disease caused by the *Plasmodium* liver stages, including hypnozoites of *P. vivax* and *P. ovale*.[2] With the exception of pregnant women, routine chemoprophylaxis is not recommended in residents of malaria-endemic areas because of the emergence of parasite resistance and potential suppression of the development of immunity that protects against severe disease after 3 to 5 years of age.

Chemoprophylaxis should be prescribed for nonimmune individuals, including children, traveling to malaria-endemic areas.[1a,11,28] The health care provider must consider several factors when choosing an appropriate chemoprophylactic regimen for the traveler. These include destination-specific malaria risk and resistance patterns, age, underlying medical conditions, allergies, tolerability, and length of stay. Pediatric dosages are based on weight and should never exceed adult dosages. A summary of available medications and recommendations for malaria chemoprophylaxis can be found in Table 43-7.

The most current information regarding malaria risk, resistance patterns, and recommendations for travelers is available through the Centers for Disease Control and Prevention (CDC) (see Resources, later). Most areas of the world have chloroquine-resistant *P. falciparum* with the exception of the Dominican Republic, Haiti, Central America west of the former Panama Canal Zone, Egypt, and some countries in the Middle East. Pyrimethamine-sulfadoxine resistance is widespread and increasing in the Amazon River Basin area of South America, Southeast Asia, and other parts of Asia and Africa. In addition, mefloquine-resistant *P. falciparum* has been found on the borders of Thailand with Burma (Myanmar) and Cambodia, in the western provinces of Cambodia, and in the eastern states of Burma (see Figure 43-2).

In general, primary chemoprophylaxis should be started 1 to 2 weeks (chloroquine or mefloquine) or 1 to 2 days (doxycycline or atovaquone-proguanil) before travel to allow the antimalarials to achieve adequate serum concentrations prior to exposure. If there are concerns about tolerance or adverse effects, chemoprophylaxis can be started earlier prior to travel to allow discovery of potential adverse events. Chemoprophylaxis should be continued during travel in the malaria-endemic area and for 4 weeks (chloroquine, mefloquine, and doxycycline) or 7 days (atovaquone-proguanil) after leaving the malaria-endemic area.

Travelers to Areas with Chloroquine-Sensitive *P. falciparum*, *P. vivax*, *P. ovale*, and *P. malariae*

Chloroquine is the drug of choice for primary prophylaxis for travelers to areas with chloroquine-sensitive malaria. The drug should be started 1 to 2 weeks before travel to a malaria-endemic area, continued once a week on the same day of the week during travel, and for 4 weeks after leaving the malaria-endemic area. Hydroxychloroquine is an alternative that may be better tolerated by some people. Side effects of chloroquine and hydroxychloroquine are generally mild and include gastrointestinal disturbance, headache, dizziness, blurred vision, pruritus, and insomnia. If the traveler is unable to take chloroquine or hydroxychloroquine, other medications effective against chloroquine-sensitive *Plasmodium* include atovaquone/proguanil, doxycycline, or mefloquine.

Travelers to Areas with Chloroquine-Resistant *P. falciparum*

Three efficacious chemoprophylaxis options for travelers to areas where there is chloroquine-resistant *P. falciparum* are atovaquone-proguanil, doxycycline, and mefloquine.[1a,16] In special situations, primaquine can be used as primary prophylaxis as well. Travelers to areas with mefloquine-resistant strains of *P. falciparum* (the borders of Thailand with Burma [Myanmar] and Cambodia, in the western provinces of Cambodia, and in the eastern states of Burma [see Figure 43-2]) can use either

TABLE 43-7. Medications Used for Prevention of Malaria*

DRUG	TRADE NAME	ADULT DOSAGE	PEDIATRIC DOSAGE¶	WHEN TO START BEFORE TRAVEL	HOW LONG TO CONTINUE AFTER RETURN	ADVERSE EFFECTS	COMMENTS
Chloroquine-Sensitive Areas							
Drug of choice:							
Chloroquine phosphate	Aralen, generic	300 mg base (500 mg salt) orally, once/wk	Base: 5 mg/kg (salt: 8.3 mg/kg) orally, once/wk, up to max. adult dose of 300 mg base	1 to 2 weeks	4 weeks	Pruritus, nausea, headache, skin eruptions, dizziness, blurred vision, insomnia. May exacerbate psoriasis.	Has been used extensively and safely in pregnancy. Where *P. vivax* and *P. ovale* are found, some recommend primaquine phosphate 52.6 mg (30 mg base)/day (for children, 0.6 mg base/kg/day) during the last 2 weeks of prophylaxis. Others prefer to avoid the risk of toxicity and rely on surveillance to detect cases.
Alternative:							
Hydroxychloroquine sulfate	Plaquenil	310 mg base (400 mg salt) orally, once/wk	Base: 5 mg/kg (salt: 6.5 mg/kg) orally, once/wk up to max. adult dose of 310 mg base	1 to 2 weeks	4 weeks	Pruritus, nausea, headache, skin eruptions, dizziness, blurred vision, and insomnia. May exacerbate psoriasis.	Has been used extensively and safely in pregnancy. Where *P. vivax* and *P. ovale* are found, some recommend primaquine phosphate 52.6 mg (30 mg base)/day (for children, 0.6 mg base/kg/day) during the last 2 weeks of prophylaxis. Others prefer to avoid the risk of toxicity and rely on surveillance to detect cases.
Chloroquine-Resistant Areas							
Drug of choice:							
Atovaquone-proguanil	Malarone	1 adult tablet† orally, daily	11–20 kg: 1 pediatric tablet‡ daily 21–30 kg: 2 pediatric tablets‡ daily 31–40 kg: 3 pediatric tablets‡ daily ≥40 kg: 1 adult tablet† daily	1 to 2 days	7 days	Headaches, nausea, vomiting, abdominal pain, diarrhea, increased transaminase levels, seizures.	Approved for once-a-day dose but dose can be divided in two to reduce nausea and vomiting; take with food or milk. Contraindicated in persons with severe renal impairment (creatine clearance <30 mL/min). Not recommended for children <11 kg, pregnant

TABLE 43-7. Medications Used for Prevention of Malaria*—cont'd

DRUG	TRADE NAME	ADULT DOSAGE	PEDIATRIC DOSAGE¶	WHEN TO START BEFORE TRAVEL	HOW LONG TO CONTINUE AFTER RETURN	ADVERSE EFFECTS	COMMENTS
							women, and women breastfeeding infants <11 kg. Where *P. vivax* and *P. ovale* are found, some recommend primaquine phosphate 52.6 mg (30 mg base)/day (for children, 0.6 mg base/kg/day) during the last 2 weeks of prophylaxis. Others prefer to avoid the risk of toxicity and rely on surveillance to detect cases.
OR:							
Doxycycline	Vibramycin, Vibra-Tabs, Doryx, Periostat, and others, generic	100 mg orally, daily	>8 years: 2 mg/kg/day up to adult dosage of 100 mg/day	1 to 2 days	4 weeks	Gastrointestinal upset, vaginal candidiasis, photosensitivity, allergic reactions, blood dyscrasias, azotemia in renal diseases, hepatitis	Contraindicated in children ≤8 years and pregnant women. Where *P. vivax* and *P. ovale* are found, some recommend primaquine phosphate 52.6 mg (30 mg base)/day (for children, 0.6 mg base/kg/day) during the last 2 weeks of prophylaxis. Others prefer to avoid the risk of toxicity and rely on surveillance to detect cases.
OR:							
Mefloquine	Lariam, Mephaquine, generic	228 mg base (250 mg salt) orally, once/wk	≤9 kg: 4.6 mg/kg base (5 mg/kg salt) 10–20 kg: 1/4 tablet§ once/wk 21–30 kg: 1/2 tablet§ once/wk 31–45 kg: 3/4 tablet§ once/wk >45 kg: 1 tablet once/wk	1 to 2 weeks	4 weeks	Gastrointestinal disturbance, headache, insomnia, vivid dreams, visual disturbances, depression, anxiety disorder, dizziness	Contraindicated in persons with active depression or a previous history of depression, generalized anxiety disorder, psychosis, schizophrenia, other major psychiatric disorders, or seizures. Not recommended for persons with cardiac conduction abnormalities.

Continued

TABLE 43-7. Medications Used for Prevention of Malaria*—cont'd

DRUG	TRADE NAME	ADULT DOSAGE	PEDIATRIC DOSAGE¶	WHEN TO START BEFORE TRAVEL	HOW LONG TO CONTINUE AFTER RETURN	ADVERSE EFFECTS	COMMENTS
							Use with caution in travelers involved in tasks requiring fine motor coordination and spatial discrimination. In the US, a 250-mg tablet of mefloquine contains a 228-mg mefloquine base. Outside the US, a 275-mg mefloquine tablet contains a 250-mg mefloquine base. Where *P. vivax* and *P. ovale* are found, some experts recommend primaquine phosphate 52.6 mg (30 mg base)/day (for children, 0.6 mg base/kg/day) during the last 2 weeks of prophylaxis. Others prefer to avoid the risk of toxicity and rely on surveillance to detect cases.
OR: Primaquine	—	30 mg base (52.6 mg salt) orally, daily	0.6 mg/kg base (1 mg/kg salt) up to adult dosage, orally, daily	1 to 2 days	3–7 days	—	An option for primary prophylaxis in special circumstances and in consultation with malaria experts. Take with food. Contraindicated in persons with G6PD deficiency, during pregnancy and breastfeeding (unless the infant being breastfed has a documented normal G6PD level).

*Caused by *Plasmodium falciparum*, *P. ovale*, *P. vivax*, and *P. malariae*.
†Adult tablets contain 250 mg atovaquone and 100 mg proguanil hydrochloride.
‡Pediatric tablets contain 62.5 mg atovaquone and 25 mg proguanil hydrochloride.
§Approximate tablet fraction is based on a recommended dosage of 5 mg/kg body weight once weekly. A pharmacist should prepare exact doses for children weighing <10 kg.
¶Should never exceed adult dosage.
G6PD, glucose-6-phosphate dehydrogenase; US, United States.

atovaquone-proguanil or doxycycline for malaria prophylaxis.[21,26] The following medications are used for prophylaxis in areas with chloroquine-resistant falciparum malaria (in alphabetical order):

Atovaquone-Proguanil. Atovaquone-proguanil is a fixed combination drug. Primary prophylaxis should begin 1 to 2 days before travel to malaria-endemic areas, and continued once a day at the same time each day during travel and daily for 7 days after leaving the malaria-endemic area. Common adverse effects associated with atovaquone-proguanil prophylaxis include abdominal pain, nausea, vomiting, and headache. Atovaquone-proguanil should not be used in pregnant women, women breastfeeding infants weighing less than 11 kg, children weighing less than 11 kg, or individuals with severe renal impairment.[11]

Doxycycline. Doxycycline primary prophylaxis should be started 1 to 2 days before travel to malarious areas, continued once a day at the same time each day during travel, and continued daily for 4 weeks after the traveler leaves the malaria-endemic area. Doxycycline can cause photosensitivity and is associated with increased frequency of *Candida* vaginitis. Gastrointestinal disturbances, such as nausea or vomiting, can be decreased if the medication is taken on a full stomach. Doxycycline should not be used in children less than 8 years old and pregnant women because of the risk of dental staining.

Mefloquine. Mefloquine prophylaxis should be started 1 to 2 weeks before traveling to a malaria-endemic area, and continued once a week on the same day of the week during travel and for 4 weeks after leaving the malaria-endemic area. Rare serious adverse reactions with prophylactic doses of mefloquine that have been reported include psychoses and seizures.[9] Other side effects include gastrointestinal disturbance, headache, insomnia, vivid dreams, visual disturbances, depression, anxiety disorder, and dizziness.[9] Mefloquine is contraindicated in people with depression or a history of psychosis or seizures, and it is not recommended for use by people with cardiac conduction abnormalities. Individuals taking mefloquine should use caution in tasks requiring fine motor coordination and spatial discrimination.

Primaquine. Rarely, primaquine can be used, in consultation with malaria experts, for primary prophylaxis for travelers unable to take any of the other recommended chemoprophylaxis regimens for areas with or without chloroquine-resistant *P. falciparum*.[16] Primaquine prophylaxis should begin 1 to 2 days before travel to the malaria-endemic area, and continued once a day at the same time each day during travel and daily for 7 days after the traveler leaves the malaria-endemic area. The traveler must have a G6PD level in the normal range, because primaquine can cause oxidant-induced hemolytic anemia and methemoglobinemia in G6PD-deficient persons.

Travelers to Areas with Chloroquine-Resistant *P. vivax*

Because the prevalence of chloroquine-resistant *P. vivax* is not known, chloroquine is still recommended for chemoprophylaxis against *P. vivax*.

Prevention of Relapses with *P. vivax* and *P. ovale*

Terminal prophylaxis with primaquine decreases the risk of relapse by acting against the liver stages of *P. vivax* and *P. ovale*. Individuals, such as missionaries, Peace Corps volunteers, and military personnel, with prolonged exposure to malaria returning to nonendemic areas should generally be given terminal prophylaxis.[16] Primaquine is given for 14 days after returning from malaria-endemic areas (see Table 43-9). If chloroquine, doxycycline, or mefloquine is used for primary prophylaxis, primaquine can be given during the last 2 weeks of postexposure prophylaxis. If atovaquone-proguanil is used for primary prophylaxis, primaquine can be given during the last 7 days of atovaquone-proguanil prophylaxis plus an additional 7 days, or for 14 days after atovaquone-proguanil is completed. Individuals should have a normal G6PD level documented before primaquine is prescribed.

Chemoprophylaxis for Infants, Children and Adolescents

Because infants, children, and adolescents are at increased risk for developing complications from malaria infection, antimalarial drugs should be prescribed for them when traveling to malaria-endemic areas.[11] Drug dosages should be calculated on the basis of body weight and should never exceed adult dosages (see Table 43-7).[1a,2] Depending on *P. falciparum* susceptibility, chloroquine and mefloquine can be used for children of nearly all ages and weights. Doxycycline can be used for children 8 years old and older. Atovaquone-proguanil is available in pediatric tablets but is not yet recommended for malaria prophylaxis in children weighing less than 11 kg. Overdoses of antimalarials can be fatal, so medications should be stored in childproof containers and kept out of reach of infants and children.

Many antimalarials have a very bitter taste and are available only as tablets. Pharmacists can pulverize tablets and put them in gelatin capsules, which the parents can then break and mix with small amounts of sweetener such as applesauce, chocolate syrup, or jelly. To ensure adequate antimalarial blood levels, the caretaker should make sure the child takes the entire dose at one time. Gastrointestinal upset and vomiting can be minimized by giving the antimalarial dose on a full stomach.

Chemoprophylaxis during Pregnancy

Pregnant women are at increased risk for severe malarial disease and adverse pregnancy outcomes, including prematurity, abortion, and stillbirth. If possible, women who are pregnant or are likely to become pregnant should be advised not to travel to malaria-endemic areas. If travel cannot be avoided, an effective chemoprophylactic regimen in conjunction with personal protective measures should be recommended. Malaria prophylaxis with chloroquine phosphate or hydroxychloroquine sulfate is safe and effective in pregnant women traveling to areas with chloroquine-susceptible *P. falciparum* and *P. vivax*.[34] For pregnant women traveling to areas with chloroquine-resistant *P. falciparum*, mefloquine is the drug of choice. Limited data show that mefloquine used at prophylactic dosages is not associated with adverse fetal or pregnancy outcomes.[34] There are insufficient data to recommend the use of atovaquone-proguanil in pregnancy. Doxycycline is contraindicated in pregnant women because it may be associated with discoloration and dysplasia of teeth and inhibition of bone growth in the fetus. Primaquine

should not be prescribed to pregnant women because the fetus may have G6PD deficiency and develop hemolytic anemia in utero.

Chemoprophylaxis while Breastfeeding

Both the mother and the breastfeeding infant should receive chemoprophylaxis, as most antimalarial drugs are not secreted in sufficient amounts to protect the infant from malaria. The very small amounts of chloroquine and mefloquine excreted in breast milk are not harmful to the infant but are at insufficient levels to provide protection against malaria. There are limited data about the use of doxycycline in lactating women, but most experts believe the risk of adverse events in the breastfeeding infant to be remote. No data are available regarding the amount of primaquine excreted in breast milk. Therefore, an infant should be tested for G6PD deficiency before primaquine is given to a woman who is breastfeeding. It is not known whether atovaquone is excreted in human milk, but proguanil is excreted in quantities insufficient to provide protection to the infant. Because data are not yet available on the safety and efficacy of atovaquone-proguanil in infants less than 11 kg, this drug is not currently recommended for the prevention of malaria in women breastfeeding infants less than 11 kg.

Changing Medications during Chemoprophylaxis because of Side Effects

Travelers should consult with a health care professional if they must change medications during chemoprophylaxis as a result of side effects. There may be special considerations because medications recommended for prophylaxis against malaria have different modes of action that affect the parasite at different stages of its life cycle. For example, if a traveler starts with chloroquine, mefloquine, or doxycycline and then changes to atovaquone-proguanil, the standard atovaquone-proguanil duration of therapy would be insufficient. Atovaquone-proguanil would have to be continued for 4 weeks after the switch or 1 week after returning, whichever is longer.

Medications Acquired Overseas

Antimalarials are widely available outside the United States. Travelers should be cautioned against purchasing medications overseas because not all countries have manufacturing controls and approval procedures similar to those in the United States.[36] Medications available overseas may not have undergone rigorous testing and review to verify their identity, potency, purity, and stability. Furthermore, medications purchased overseas may be ineffective, dangerous, counterfeit, or contaminated.

Personal Protective Measures

Preventing bites from infected *Anopheles* mosquitoes (see Chapter 41) is an important adjunct to chemoprophylaxis for malaria prevention in travelers (see Table 43-6). *Anopheles* mosquitoes are nocturnal feeders, so outdoor activity should be minimized during dusk and nighttime when they are most active. If outdoor exposure is unavoidable during these times, individuals should wear dark-colored long-sleeved shirts, pants, and hats. Mosquito repellent (10% to 35% DEET [*N,N*-diethyl-3-methylbenzamide]) should be applied to exposed skin.[7,28] DEET is safe and effective when used on infants and children. Permethrin or deltamethrin is a long-acting insecticide that can be applied to clothes, shoes, mosquito nets, tents, and other gear. When applied properly, permethrin-treated clothes repel mosquitoes for up to 25 launderings. In addition, an insecticide-treated bed net tucked under the mattress provides protection during peak mosquito feeding times, particularly when accommodations are not adequately air conditioned or enclosed by screens. Finally, travelers can use mosquito coils or candles to repel mosquitoes when outdoors.

TREATMENT

Malaria treatment consists of rapid and appropriate antimalarial therapy as well as supportive care. Whereas uncomplicated malaria can be treated with antimalarials and antipyretics, severe falciparum malaria is a medical emergency and requires immediate assessment and treatment. In general, drugs used in the treatment of malaria should be different from those the patient was using for malaria chemoprophylaxis. Individuals living in areas with perennial malaria transmission develop immunity against severe malarial disease. These semi-immune individuals are often asymptomatic and have a lower risk of serious complications and death. Malaria in nonimmune and at-risk populations, such as infants, children, and pregnant women, should be considered a medical emergency, because falciparum malaria can progress rapidly and be lethal. When initiating medical therapy, the health care provider should consider the infecting *Plasmodium* species, resistance patterns, parasite density, and the patient's clinical status.[32] If the diagnosis of malaria is suspected but cannot be confirmed, antimalarial treatment should be initiated that is effective against *P. falciparum*. Health care workers requiring assistance in the diagnosis and treatment of malaria should consult physicians specializing in tropical medicine or infectious diseases or the CDC (see Resources, later).

Presumptive Self-Treatment

Travelers with signs or symptoms of malaria should be counseled to seek urgent medical evaluation, which includes obtaining thick and thin blood smears. Antimalarials for self-treatment may be prescribed for individuals who plan on living in remote areas where prompt access to medical care will be difficult (Table 43-8). Presumptive self-treatment should be taken immediately if the traveler develops an influenza-like illness with fever and chills and professional medical care is not available within 24 hours. The traveler should seek medical care promptly after taking medication for presumptive malaria.

Atovaquone-proguanil can be used for presumptive malaria self-treatment if it is not being used for malaria chemoprophylaxis. Depending on local malaria resistance patterns, pyrimethamine-sulfadoxine is another option for malaria self-treatment.

Treatment of Malaria Caused by Chloroquine-Sensitive *P. falciparum*, *P. vivax*, *P. ovale*, and *P. malariae*

Chloroquine is the drug of choice for infections with chloroquine-susceptible malarial parasites.[40] Chloroquine is as efficacious as quinine in the treatment of cerebral malaria caused by chloroquine-susceptible *P. falciparum*.[42] It has excellent oral absorption and can be administered via nasogastric tube in patients with impaired consciousness. Parenteral quinidine or quinine is recommended (Table 43-9) for patients unable to take oral chloroquine. Intramuscular quinidine or quinine should be

TABLE 43-8. Medications for Presumptive Self-Treatment* of Malaria†

DRUG	TRADE NAME	ADULT DOSAGE	PEDIATRIC DOSAGE‡	ADVERSE EFFECTS	COMMENTS
Atovaquone-proguanil	Malarone	4 adult tablets‡ (each dose contains 1000 mg atovaquone and 400 mg proguanil) orally as a single daily dose for 3 consecutive days	Single daily dose to be taken for 3 consecutive days <5 kg: not indicated 5–8 kg: 2 pediatric tablets‡ 9–10 kg: 3 pediatric tablets 11–20 kg: 1 adult tablet 21–30 kg: 2 adult tablets 31–40 kg: 3 adult tablets ≥41 kg: 4 adult tablets	Nausea, vomiting, abdominal pain, diarrhea, increased transaminase levels, seizures	Approved for once-a-day dose but dose can be divided in two to reduce nausea and vomiting; take with food or milk. Contraindicated in persons with severe renal impairment (creatine clearance <30 mL/min). Adult tablet not recommended for children <5 kg, pregnant women, women breastfeeding infants <5 kg, and persons on atovaquone-proguanil prophylaxis.
OR:					
Mefloquine		750 mg salt PO followed 12 hrs later by 500 mg salt Total dose = 1250 mg salt	15 mg/kg (13.7 mg base) followed 12 hrs later by 10 mg/kg (9.1 mg base) Total dose = 25 mg salt/kg		See mefloquine comments in Table 43-9.
OR:					
Quinine sulfate		650 mg PO q8 h × 3–7 days	30 mg/kg/day in 3 doses × 3–7 days		See quinine comments in Table 43-9.
PLUS:					
Doxycycline		100 mg PO bid	4 mg/kg/day in 2 doses × 7 days		See doxycycline comments in Table 43-9.

*Self-treatment drug is to be used for febrile illness if professional medical care is not available within 24 hours. Medical care should be sought immediately after treatment.
†Caused by *Plasmodium falciparum*, *P. ovale*, *P. vivax*, and *P. malariae*.
‡Each adult tablet contains 250 mg atovaquone and 100 mg proguanil; each pediatric tablet contains 62.5 mg atovaquone and 25 mg proguanil. Pediatric dosages should never exceed adult dosages.

given with extreme caution because of the chance of cardiac arrhythmias with inadvertent intravenous administration. In patients with *P. vivax* or *P. ovale,* treatment should be followed by a course of primaquine.

Treatment of Chloroquine-Resistant *P. falciparum*

Medications that can be used for the treatment of chloroquine-resistant *P. falciparum* infection include quinine or quinidine in conjunction with doxycycline, tetracycline, pyrimethamine-sulfadoxine, or clindamycin (see Table 43-9).[1a,2,32] Atovaquone/proguanil can also be used as first-line treatment for chloroquine-resistant *P. falciparum*. Second-line drugs include mefloquine alone or artesunate in conjunction with mefloquine.

Individuals on mefloquine prophylaxis should not be treated with mefloquine. Furthermore, mefloquine should not be used in malaria infection acquired in areas of reported mefloquine resistance. The risk of adverse events, such as myocardial conduction disturbances, is increased in individuals taking mefloquine prophylaxis and receiving treatment with quinine and quinidine. In addition, resistances to mefloquine and halofantrine have been linked; therefore, halofantrine should not be used to treat malaria in individuals taking mefloquine prophylaxis. Halofantrine has been associated with adverse cardiac advents at treatment dosages and is not approved by the U.S. Food and Drug Administration. Artesunate is not available in the United States but is used widely overseas for the treatment of malaria. Because resistance to pyrimethamine-sulfadoxine is common in areas of chloroquine-resistance, individuals treated with this drug should be watched carefully.

In the United States, parenteral quinidine is preferred over quinine for the following reasons: (1) it is more active than quinine, (2) blood levels can be followed, and (3) parenteral quinine is no longer available in the United States. If malaria treatment is being given outside the United States, artemisinin derivatives should be considered because they clear parasitemia and end coma more rapidly than quinine or quinidine, particularly when the level of parasitemia is very high. To prevent recrudescence with artemisinin derivatives, additional drugs are often given. These include doxycycline, tetracycline, pyrimethamine-sulfadoxine, clindamycin, or mefloquine.

For infections acquired in Southeast Asia, quinidine or quinine treatment should be continued for 7 days because of increased relative resistance. If mefloquine is used to treat malaria infections from areas where mefloquine resistance has been reported, such as Southeast Asia, a higher dosage should be used.

Text continued on p. 946.

TABLE 43-9. Medications for the Treatment of Malaria*

DRUG	TRADE NAME	ADULT DOSAGE	PEDIATRIC DOSAGE†	ADVERSE EFFECTS	COMMENTS
Chloroquine-Sensitive *P. falciparum*, *P. vivax*, *P. ovale*, *P. malariae*					
Drug of choice:					
Chloroquine phosphate§	Aralen	1 g salt (600 mg base) PO immediately, followed by 500 mg salt (300 mg base) PO at 6, 24, and 48 hr. Total dosage: 2500 mg salt (1500 mg base)	10 mg base/kg (max. 600 mg base) PO immediately, followed by 5 mg/kg (base) PO at 6, 24, and 48 hr. Total dosage: 25 mg/kg (base)	Pruritus, nausea, headache, skin eruptions, dizziness, blurred vision, insomnia. May exacerbate psoriasis	Has been used extensively and safely in pregnancy.
Chloroquine-Resistant *P. falciparum* (Oral)					
Drugs of choice:					
Atovaquone-proguanil	Malarone	2 adult tablets‡ (500 mg atovaquone/ 200 mg proguanil) bid × 3 days daily OR 4 adult tablets‡ (1 g atovaquone/ 400 mg proguanil) qd × 3 days	<5 kg: not indicated 5–8 kg: 2 pediatric tablets/day‡ × 3 days 9–10 kg: 3 pediatric tablets/day‡ × 3 days 11–20 kg: 1 adult tablet‡/ day × 3 days 21–30 kg: 2 adult tablets‡/day × 3 days 31–40 kg: 3 adult tablets‡/day × 3 days >40 kg: 2 adult tablets‡ bid × 3 days OR 4 adult tablets‡ qd × 3 days	Headache, nausea, vomiting, abdominal pain, diarrhea, increased transaminase levels, seizures	Approved for once-a-day dosing but dose can be divided in two to reduce nausea and vomiting. Take with food or milk. Contraindicated in persons with severe renal impairment (creatine clearance <30 mL/min). Adult tablet not recommended for children <5 kg, pregnant women, women breastfeeding infants <5 kg.
Quinine sulfate	—	650 mg salt (542 mg base) q8h × 3–7 days	30 mg salt/kg/ day in 3 doses × 3–7 days	—	In Southeast Asia, continue treatment for 7 days because of increased relative resistance to quinine.
PLUS:					
Doxycycline	Vibramycin, Vibra-Tabs, Doryx, Periostat and others, generic	100 mg bid × 7 days	>8 yr: 4 mg/kg/ day in 2 doses × 7 days	Gastrointestinal upset, vaginal candidiasis, photosensitivity, allergic reactions, blood dyscrasias, azotemia in renal diseases, hepatitis	Contraindicated in children ≤8 yr and pregnant women.

TABLE 43-9. Medications for the Treatment of Malaria*—cont'd

DRUG	TRADE NAME	ADULT DOSAGE	PEDIATRIC DOSAGE†	ADVERSE EFFECTS	COMMENTS
OR PLUS:					
Tetracycline	Achromycin, Sumycin, Panmycin, and others	250 mg qid × 7 days	25 mg/kg/day in 4 doses × 7 days	Gastrointestinal upset, vaginal candidiasis, photosensitivity, allergic reactions, blood dyscrasias, azotemia in renal diseases, hepatitis	Contraindicated in children ≤8 yr and pregnant women.
OR PLUS:					
Clindamycin	Cleocin and others	20 mg/kg/day divided in 3 doses × 7 days	20 mg/kg/day in 3 doses × 7 days	—	For use in pregnancy.
Alternatives:					
Mefloquine	Lariam, Mephaquin, generic	750 mg salt followed by 500 mg 6–12 hr later Total dose = 1250 mg salt	<45 kg: 15 mg/kg (13.7 mg base) followed by 10 mg/kg (9.1 mg base) 6–12 hr later Total dose = 25 mg salt/kg	Gastrointestinal disturbance, headache, insomnia, vivid dreams, visual disturbances, depression, anxiety disorder, dizziness	Contraindicated in persons with active depression or a previous history of depression, generalized anxiety disorder, psychosis, schizophrenia, other major psychiatric disorders, or seizures. Contraindicated for treatment in pregnancy. Not recommended for persons with cardiac conduction abnormalities. Use with caution in travelers involved in tasks requiring fine motor coordination and spatial discrimination. In the US, a 250-mg tablet of mefloquine contains a 228-mg mefloquine base. Outside the US, a 275-mg mefloquine tablet contains a 250-mg mefloquine base. Do not give with quinine, quinidine, or halofantrine. In areas of reported resistance (e.g., the Thailand-Myanmar and Thailand-Cambodia borders and the Amazon basin), 25 mg/kg should be used.
OR:					
Artesunate	—	4 mg/kg/day × 3 days	4 mg/kg/day × 3 days	—	Available in US only from the manufacturer.
PLUS:					
Mefloquine	Lariam, Mephaquin, generic	750 mg salt followed by 500 mg 6–12 hr later Total dose = 1250 mg salt	<45 kg: 15 mg/kg followed by 10 mg/kg (9.1 mg base) 6–12 hr later Total dose = 25 mg salt/kg	Gastrointestinal disturbance, headache, insomnia, vivid dreams, visual disturbances, depression, anxiety disorder, dizziness	Contraindicated in persons with active depression or a previous history of depression, generalized anxiety disorder, psychosis, schizophrenia, other major psychiatric disorders, or seizures. Contraindicated for treatment in pregnancy. Not recommended for persons with cardiac conduction abnormalities. Use with caution in travelers involved in tasks requiring fine motor coordination and spatial discrimination. In the US, a 250-mg tablet of mefloquine contains a 228-mg mefloquine base. Outside the US, a 275-mg mefloquine tablet contains a 250-mg mefloquine base. Do not give with quinine, quinidine, or halofantrine. In areas of reported resistance (e.g., the Thailand-Myanmar and Thailand-Cambodia borders and the Amazon basin), 25 mg/kg should be used.

Continued

TABLE 43-9. Medications for the Treatment of Malaria*—cont'd

DRUG	TRADE NAME	ADULT DOSAGE	PEDIATRIC DOSAGE†	ADVERSE EFFECTS	COMMENTS
Chloroquine-Resistant *P. vivax* (Oral)					
Drugs of choice:					
Quinine sulfate	—	650 mg q8h × 3–7 days	30 mg/kg/day in 3 doses × 3–7 days	—	In Southeast Asia, continue treatment for 7 days because of increased relative resistance to quinine.
PLUS:					
Doxycycline	Vibramycin, Vibra-Tabs, Doryx, Periostat and others, generic	100 mg bid × 7 days	>8 yr: 4 mg/kg/day in 2 doses × 7 days	Gastrointestinal upset, vaginal candidiasis, photosensitivity, allergic reactions, blood dyscrasias, azotemia in renal diseases, hepatitis	Contraindicated in children ≤8 yr and pregnant women.
OR:					
Mefloquine	Lariam, Mephaquin, generic	750 mg salt followed by 500 mg salt 6–12 hr later Total dose = 1250 mg salt	<45 kg: 15 mg/kg (13.7 mg base) followed by 10 mg/kg (9.1 mg base) 6–12 hr later Total dose = 25 mg/kg salt	Gastrointestinal disturbance, headache, insomnia, vivid dreams, visual disturbances, depression, anxiety disorder, dizziness	Contraindicated in persons with active depression or a previous history of depression, generalized anxiety disorder, psychosis, schizophrenia, other major psychiatric disorders, or seizures. Contraindicated for treatment in pregnancy. Not recommended for persons with cardiac conduction abnormalities. Use with caution in travelers involved in tasks requiring fine motor coordination and spatial discrimination. In the US, a 250-mg tablet of mefloquine contains a 228-mg mefloquine base. Outside the US, a 275-mg mefloquine tablet contains a 250-mg mefloquine base. Do not give with quinine, quinidine, or halofantrine. In areas of reported resistance (e.g., the Thailand-Myanmar and Thailand-Cambodia borders and the Amazon basin), 25 mg/kg should be used.
Alternatives:					
Chloroquine phosphate	Aralen	25 mg base/kg in 3 doses over 48 hr	Same as adult dosage	Pruritus, nausea, headache, skin eruptions, dizziness, blurred vision, insomnia. May exacerbate psoriasis	Has been used extensively and safely in pregnancy. If chloroquine phosphate is not available, hydroxychloroquine sulfate is effective (400 mg of hydroxychloroquine sulfate is equivalent to 500 mg of chloroquine sulfate).
PLUS:					
Primaquine phosphate	—	30 mg base/day × 14 days	0.6 mg/kg/day × 14 days	—	Take with food. Contraindicated in persons with G6PD deficiency, and during pregnancy and breastfeeding unless the infant being breastfed has a documented normal G6PD level.
All Plasmodium (Parenteral)					
Drug of choice:					
Quinidine gluconate	—	10 mg salt (6.25 mgbase)/kg loading dose (max. 600 mg salt) in normal saline slowly over 1 to 2 hr, followed by continuous	Same as adult dosage	—	In patients with severe malaria, use with one of the following: doxycycline, tetracycline, or clindamycin. Continuous EKG, blood pressure, and glucose monitoring are recommended, especially in pregnant women and children. The loading dose should be decreased or omitted in patients who have received quinine or mefloquine.

TABLE 43-9. Medications for the Treatment of Malaria*—cont'd

DRUG	TRADE NAME	ADULT DOSAGE	PEDIATRIC DOSAGE†	ADVERSE EFFECTS	COMMENTS
		infusion of 0.02 mg salt (0.0125 mg base)/kg/min until oral therapy can be started or parasitemia is <1%. Treat for 7 days in multidrug-resistant areas, 3 days in non–multidrug-resistant areas.			For problems with quinidine availability, call the manufacturer (Eli Lilly, 800-821-0538) or the CDC Malaria Hotline (770-488-7788). If >48 hr of parenteral therapy is required, the quinine or quinidine dosage should be decreased by $^1/_3$ to $^1/_2$.
OR:					
Quinine dihydrochloride (IV)	—	20 mg/kg loading dose IV in 5% dextrose over 4 hr, followed by 10 mg/kg over 2–4 hr q8h (max. 1800 mg/day) until oral therapy can be started or parasitemia is <1%. Treat for 7 days in multidrug-resistant areas, 3 days in non–multidrug-resistant areas.	Same as adult dosage	—	In patients with severe malaria, use with one of the following: doxycycline, tetracycline, or clindamycin. Not available in the US. Continuous EKG, blood pressure, and glucose monitoring are recommended, especially in pregnant women and children. The loading dose should be decreased or omitted in patients who have received quinine or mefloquine. If >48 hr of parenteral therapy is required, the quinine or quinidine dosage should be decreased by $^1/_3$ to $^1/_2$.
Alternative:					
Artemether	—	3.2 mg/kg IM, then 1.6 mg/kg daily × 5–7 days	Same as adult dosage	—	Available in the US only from the manufacturer.
Prevention of Relapses: *P. vivax* and *P. ovale* Only					
Drug of choice:					
Primaquine phosphate	—	30 mg base (52.6 mg salt)	0.6 mg base/kg/day × 14 days	—	For individuals who have had prolonged exposure to *P. vivax* or *P. ovale*. Take with food. Contraindicated in persons with G6PD deficiency, and during pregnancy and breastfeeding unless the infant being breastfed has a documented normal G6PD level. Relapses have been reported with this regimen and should be treated with a second 14-day course of 30 mg base/day. In Southeast Asia and Somalia, the higher dosage (30 mg base/day) should be used initially.

*Caused by *Plasmodium falciparum*, *Plasmodium ovale*, *Plasmodium vivax*, and *Plasmodium malariae*.
†Should never exceed adult dosage.
‡Adult tablet contains 250 mg atovaquone and 100 mg proguanil hydrochloride. Pediatric tablet contains 62.5 mg atovaquone and 25 mg proguanil.
§Hydroxychloroquine sulfate can be used if chloroquine phosphate is not available. 400 mg of hydroxychloroquine sulfate is equivalent to 500 mg of chloroquine phosphate.
EKG, electrocardiography; G6PD, glucose-6-phosphate dehydrogenase; IM, intramuscularly; IV, intravenously; US, United States.

Treatment of Chloroquine-Resistant *P. vivax*

Chloroquine-resistant *P. vivax* can be treated with quinine or quinidine in conjunction with doxycycline (see Table 43-9). Mefloquine can also be used for treatment if the individual was not taking mefloquine prophylaxis. Treatment should be followed by a course of primaquine.

Eradication of *P. vivax* and *P. ovale* Hypnozoites

To prevent relapse, primaquine can be used to eradicate the hypnozoites in individuals with *P. vivax* or *P. ovale* infection. A normal G6PD level should be documented because of the risk of hemolysis and methemoglobinemia. Given reported resistance patterns, infections acquired from Southeast Asia and Somalia should be treated initially with a higher dosage of primaquine (see Table 43-9).

Supportive Care

Nonimmune individuals diagnosed with malaria should be hospitalized until a response to treatment has been seen and complications from severe disease are unlikely (i.e., 24 hours after a negative blood smear, and when afebrile). Individuals with partial immunity, such as those residing in malaria-holoendemic areas, and uncomplicated *P. falciparum* infection, as well as some nonimmune individuals with *P. vivax, P. ovale,* or *P. malariae* infection, may be treated as outpatients if reliable follow-up is anticipated. Patients with severe complicated malaria require aggressive supportive care.[32,38]

In addition to rapid clinical assessment and initiation of antimalarial chemotherapy, dehydration, hypoglycemia, and acidosis should be corrected. Admission to the intensive care unit is often required to manage complications such as seizures, severe anemia, hypoglycemia, acidosis, renal failure, pulmonary edema, hypoxemia, and hypovolemia.[32,38,40]

A patient receiving quinine derivatives should be placed on a cardiac monitor in the intensive care unit with frequent blood pressure measurements. Quinine or quinidine derivatives are typically given with a continuous infusion of 5% to 10% dextrose. Blood glucose measurements should be monitored every 4 to 6 hours. Some drug dosages should be adjusted for patients in renal failure. For example, individuals receiving quinine derivatives should have the standard loading dose, but maintenance doses should be decreased 30% to 50% to prevent accumulation of the drug secondary to decreased renal excretion. Total plasma concentrations of 8 to 15 mg/mL for quinine and 3.5 to 8.0 mg/mL for quinidine are effective for the treatment of severe malaria without causing toxicity. Dose reductions for renal failure are not necessary with chloroquine or artemisinin derivatives.

Anticonvulsants, such as benzodiazepines, should be used for treatment of seizures. Antibiotics that treat bacterial meningitis should be initiated until a lumbar puncture can be performed to exclude the diagnosis. Corticosteroids and heparin should not be used for treatment of cerebral malaria, as there is no proven benefit and these agents may be harmful.[38] Guidelines for the use of exchange transfusions for the treatment of malaria remain controversial. Exchange transfusion may be beneficial for severely ill patients with parasitemia greater than 15% or with 5% to 15% parasitemia and signs of a poor prognosis (see Table 43-4).[40]

Therapeutic efficacy can be monitored by checking multiple sequential blood smears (at least every 6 to 12 hours for 2 days). If high levels of parasitemia persist or clinical improvement is not evident, there may be primary drug resistance and therapy should be changed immediately. Management of severe malaria should preferably be carried out in consultation with a tropical medicine or infectious diseases physician, or with the assistance of the CDC (see Resources, later).

RESEARCH AND VACCINE DEVELOPMENT

The human host responds to malaria infection by several mechanisms. After repeated exposures to different parasite strains over years, individuals develop protection from high-level parasitemia and severe disease but not from *Plasmodia* infection. The malarial parasite has developed multiple mechanisms by which to evade human immune responses. Progress toward an effective vaccine against malaria, especially *P. falciparum,* is hindered by the diversity of human populations infected with malaria as well as by the diversity of parasite strains that infect humans.

Three strategies toward vaccine development are being developed simultaneously. The first strategy is to prevent infection caused by sporozoites, thus preventing development of merozoites in the liver and the asexual parasitemia responsible for clinical disease. The second strategy of vaccine development is to limit parasite erythrocyte invasion and multiplication. The third strategy consists of preventing spread of the parasite by a transmission-blocking vaccine that inhibits sexual development of the parasite. An effective vaccine may require a combination of these strategies. Because most deaths from malaria occur in children with severe malaria disease, a vaccine may be effective if it can prevent severe disease rather than asymptomatic asexual parasitemia. There are 25 vaccines undergoing phase Ia safety and immunogenicity studies.[15] Only two vaccines are currently undergoing phase IIb trials, which test for vaccine efficacy in a malaria-endemic country.[15] These results will help guide future vaccine development efforts. Much research must be done before malaria is no longer one of the major causes of death in children.

RESOURCES

Several resources are available to aid the health care provider in prevention, diagnosis, and treatment of malaria. Comprehensive websites include the CDC (www.cdc.gov/malaria), the World Health Organization (www.who.int/topics/malaria/en), and the Malaria Foundation (www.malaria.org).[1]

Updated detailed information regarding malaria prophylaxis can be obtained through the CDC's Traveler's Health information service by phone, 877-394-8747 (877-FYI-TRIP); fax, 888-232-3299; and the internet, www.cdc.gov/travel. Information regarding the diagnosis of malaria is available through the CDC's Division of Parasitic Diseases diagnostic internet site (www.dpd.cdc.gov/dpdx). Health care professionals requiring assistance with the diagnosis and treatment of suspected or confirmed cases of malaria should call the CDC's Malaria Hotline (Malaria Branch, 770-488-7788 from 8:00 AM to 4:30 PM EST, Monday to Friday; or the Emergency Operations Center during evenings, weekends, and holidays, 770-488-7100, ask to page the person on call for the Malaria Branch).

The references for this chapter can be found on the accompanying DVD-ROM.

Arthropod Envenomation and Parasitism

44

Timothy B. Erickson

The phylum Arthropoda contains about four fifths of the known animals of the world, and insects are the largest group of arthropods. Insects are an important part of the biota of all terrestrial and freshwater environments that support life. More species of insects exist than of any other form of multicellular life, and they may well exceed all other land animals in biomass. Insects can use most animal and plant substances as food, and their feeding plays a vital role in recycling organic compounds. They compete with other organisms for the world's food supplies but are themselves a major food source for many forms of life. They are essential for the pollination of many plants. Insect life cycles are diverse and often complex, involving developmental and sexual stages that are widely different in morphology and ways of life. Although sexual reproduction is the rule, parthenogenesis (unisexual reproduction) and pedogenesis (sexual development in the larval stage) occur. Some groups, such as ants, bees, and termites, have developed a high degree of social organization. During at least part of its life cycle, an insect's body is divided into three distinct regions (head, thorax, and abdomen), with three pairs of legs attached to the thorax. Except for a few primitive or parasitic groups, most adult insects have wings.

The greatest direct medical importance of insects is associated with their feeding on human blood and tissue fluids. In doing so, they often inject salivary secretions. This is a highly effective method of transmitting pathogenic microorganisms; moreover, the secretions are often allergenic and sometimes toxic. Other insects may carry human pathogens passively on their feet or mouthparts or in their digestive tracts.

Venoms have evolved in several insect groups, and venomous insects may attack humans, sometimes with lethal results. Skin, hair, and secretions of insects may be irritant or allergenic, producing cutaneous and respiratory syndromes. Finally, insects can be highly annoying.

HYMENOPTERA (BEES, WASPS, AND ANTS)

By far the most important venomous insects are members of the order Hymenoptera, including bees, wasps, and ants (Fig. 44-1). They vary in size from minute to large (up to 60 mm in body length). The abdomen and thorax are connected by a slender pedicle that may be quite long in certain wasps and ants. Bees and most wasps are winged as adults; ants are wingless, except for sexually mature adults during part of the life cycle. Mouthparts are adapted for chewing but in some species are modified for sucking. The life cycle includes egg, larval, and pupal stages before emergence of adults. Immature stages may be protected and provided with food by the adult. Both animal and plant foods are used. Many species are parasitic on other arthropods. All ants and many species of bees and wasps are social insects. Colonies range in size from a few dozen individuals to many thousands. In cold climates, most individuals die in autumn, leaving the fertilized females to winter over and found new colonies in the spring.

Bees

The honeybee (*Apis mellifera*) is one of the few domesticated insects and is maintained in hives in many countries (Figs. 44-2 and 44-3). Numerous geographic races of the honeybee exist; the Italian bee (*Apis mellifera ligustica*), a common domestic strain of Europe, is also widely distributed in the United States. Feral honeybee colonies usually nest in hollow trees or crevices in rocks but may nest in the walls of occupied buildings.

An event of considerable health and economic significance in the Americas was the introduction of an African race of the honeybee (*Apis mellifera scutellata,* also referred to as *Apis mellifera adansoni*). This race was introduced from Africa into Brazil because it was thought to be a more efficient honey producer in the tropics. It is characterized by large populations (one queen may lay tens of thousands of eggs), frequent swarming (six to 12 swarms a year), nonstop flights of at least 20 km (12.4 miles), and a tendency toward mass attacks on humans after minimal provocation. As a result, these Africanized honeybees, also known as killer bees, are much more aggressive than the typical Western Hymenoptera. They attack in swarms of hundreds and chase their victims much greater distances from the hive than any other species.[109,126,130]

The first escapes from hives occurred in the state of São Paulo in 1957, and the "Brazilian killer bees," or "Africanized bees," have spread widely. These bees are actually hybrids between *A. scutellata* and European honeybee races. Cold climate seems to have stopped their southern spread in Argentina, but they have moved steadily northward at 200 to 300 miles (322 to 482 km) per year and in October 1990 reached the southern border of the United States.[279] By mid-1991, 103 swarms had been captured in southern Texas. Populations are established in Arizona, New Mexico, and California.[238] Future populations may eventually be distributed as far east as North Carolina.[277] Several human deaths have occurred from multiple stings. Unless the

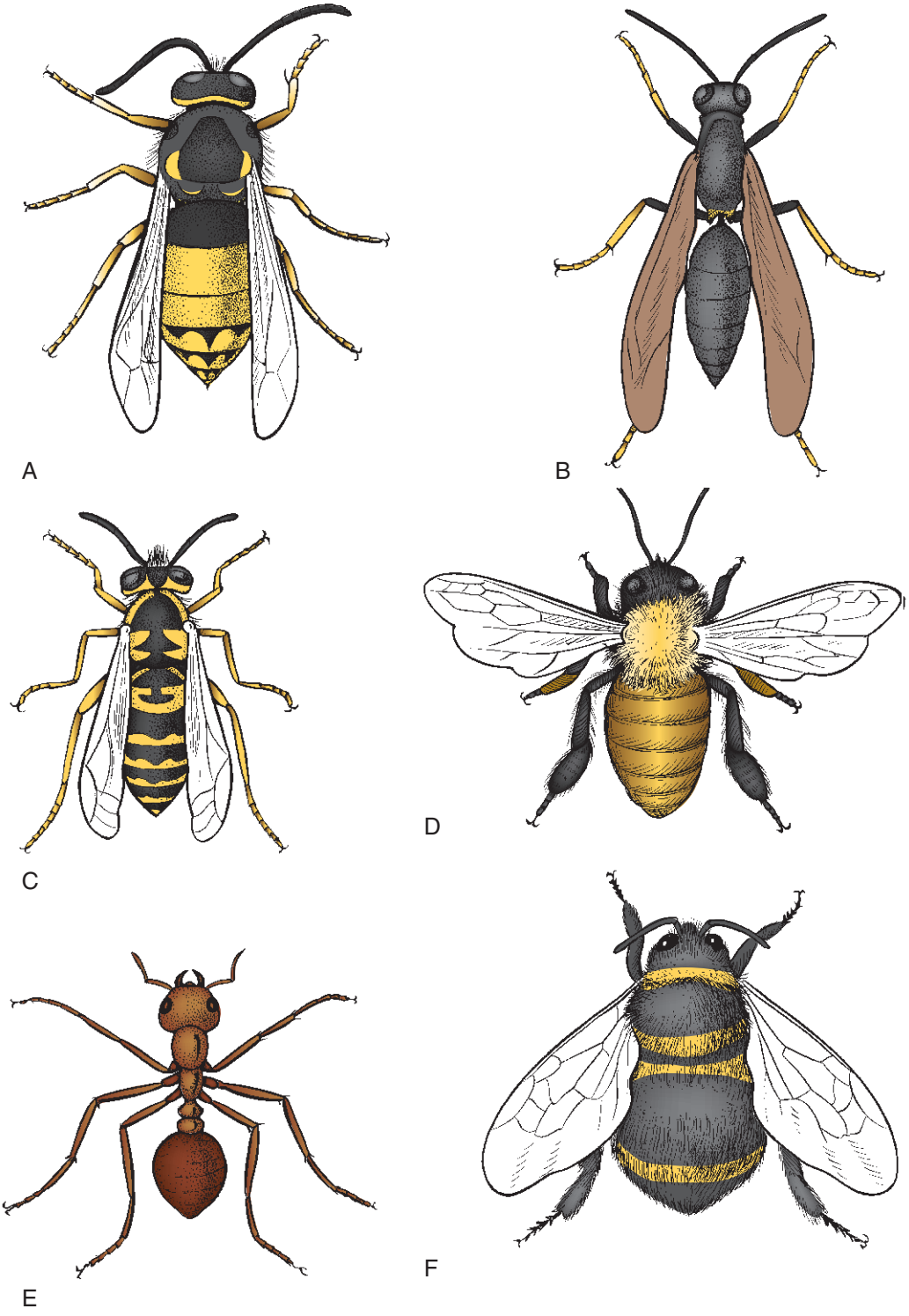


Figure 44-1. Representative venomous Hymenoptera. **A,** Hornet (*Vespula maculata*). **B,** Wasp (*Chlorion ichneumerea*). **C,** Yellowjacket (*Vespula maculiforma*). **D,** Honeybee (*Apis mellifera*). **E,** Fire ant (*Solenopsis invicta*). **F,** Bumblebee (*Bombus* species).

Figure 44-2. Worker honeybee, *Apis mellifera*.

Figure 44-4. Sweat bee, family Halictidae. (From www.encarta.msn.com.)

Figure 44-3. Stinger of a honeybee.

bees acquire greater resistance to winter conditions, their range will be confined to the southern third of the United States and may also be restricted by scarcity of suitable flowers in the arid Southwest. Those periods of the year characterized by high temperatures and low rainfall are related to the greatest activity of Africanized bees and a larger number of swarms, thus giving rise to increased contact with human populations.[59] The greatest impact of Africanized bees in the United States will probably be economic, related to decreased honey production and less effective pollination of crops. The bees also present a threat to human health. Africanized bee colonies are extremely sensitive to disturbance, respond faster in greater numbers, and are up to 10 times more active in stinging than European bees. The quantity of venom per sting is slightly less in African bees; however, there is no significant biochemical or allergenic difference between the venoms.[171,185,240] About 50 simultaneous stings can cause systemic envenomation, and an estimated 500 are necessary to cause death by direct toxicity.[125] As few as 30 to 50 stings have proven fatal in small children.[26]

About 350 fatal attacks have been documented worldwide, of which at least 70 occurred in Venezuela during 1977 and 1978. More than 300 bee attacks occurred in Mexico between 1987 and 1992, with 49 fatalities.[182] A more recent account puts the fatalities at 190, with future estimates of 60 deaths per year.[238] Because Mexico and the southern United States have many feral and domestic honeybee populations, researchers thought that the aggressiveness of the African bees could be dampened by hybridization. Large numbers of male European bees were released to facilitate this, and African queens were replaced by European stock when possible. However, recent studies indicate that European bee populations are becoming rapidly Africanized with little reciprocal gene flow, as African females take over European hives.[110,147,251,280]

Bumblebees (*Bombus* and related genera) are a largely holarctic group often found in quite cold environments. Small colonies usually nest just under the surface of the ground, often in mammal burrows. Some species are aggressive if disturbed, although most have mild dispositions.

Sweat bees (family Halictidae) are small bees of cosmopolitan distribution (Fig. 44-4). They are attracted to sweaty skin and ingest perspiration. They nest in burrows, often in clay banks. Females sting if squeezed or trapped under clothing. The sting is not very painful, but anaphylactic reactions have been reported. The allergens are immunologically unrelated to those in other bee and wasp venoms.[199]

Wasps

Social wasps occur throughout most of the world but are recognized as a medical problem chiefly in the United States and Europe. They often establish colonies close to human dwellings. Yellowjackets (*Vespula* species) may be more important than honeybees as a cause of human stings in the northeastern United States (Fig. 44-5). They make underground nests (Fig. 44-6) in rotted-out tree stumps, cavities under stones, and mammal burrows. They are strongly attracted to garbage. Paper wasps (*Polistes* species) (Fig. 44-7) suspend their nests in shaded places, often in shrubbery near houses or below eaves, gutters, or window frames. Old World hornets (*Vespa* species) and

Figure 44-5. Yellowjacket, *Vespula maculiforma*.

Figure 44-8. White-faced hornet, *Dolchiovespula maculata*, largest of the common social wasps in the United States.

Figure 44-6. Early yellowjacket nest.

Figure 44-9. Typical nest of the white-faced hornet.

Figure 44-7. Paper wasp, *Polistes* species.

white-faced hornets (*Dolchiovespula maculata*) create large paper nests that may be plastered to buildings but more typically hang from tree branches (Figs. 44-8 and 44-9).

Solitary wasps are predators, feeding largely on other insects and spiders. Adults often carry the prey alive and paralyzed to the nest as food for the larvae. Some wasps excavate burrows, whereas others make mud nests that may be plastered on shaded walls of buildings or under bridges. Although many nests may be grouped together, the adult wasps have no social organization and make little effort to defend them. The cicada killers (*Sphecius speciosus*) (Fig. 44-10) and tarantula hawks (*Pepsis* species) (Fig. 44-11) are among the largest North American wasps. Velvet ants (family Mutillidae) are female wingless wasps that are nest parasites of other Hymenoptera (Fig. 44-12). They occur in deserts and other dry and open habitats and can inflict a painful sting.

Internationally, hymenopteran insects are worldwide in distribution and often constitute a major part of a region's insect fauna. Honeybees are exploited for their honey throughout the

Figure 44-10. Cicada killer wasp, *Sphecius speciosus*. (From www.ento.okstate.edu.)

Figure 44-11. Tarantula hawk, *Pepsis* species. (From www.rsantz.com/wasps.)

Figure 44-12. Velvet ant, family Mutillidae.

Figure 44-13. Paper wasp nest.

world; even the aggressive *A. m. scutellata* is used for honey production in Africa. Honeybees in southern Asia attach huge nests to limbs of forest trees. The giant bee *Apis dorsata,* of Southeast Asia, has a reputation for savagery, and deaths from multiple stings have occurred. Yellowjackets and hornets are common in Europe and the Middle East, where their medical importance is similar to that in the United States. The genera *Vespula, Vespa,* and *Dolichovespula* are found all over Europe. The genus *Polistes,* present throughout central Europe, is not found in the United Kingdom and represents a larger clinical problem in areas surrounding the Mediterranean Sea. Amino acid sequencing of the venom allergens of the different species of *Polistes* in Europe shows them to be very similar, but the similarities to American strains are less pronounced.[82]

Two species, *Vespula orientalis* and *Vespula vulgaris,* have recently been introduced into Australia, where they have become a significant problem.[44] Paper wasps of the genus *Polistes* are plentiful in tropical America and Australia (Fig. 44-13). Another Australian paper wasp, *Ropalidia revolutionalis,* constructs nests that resemble belts of bullets and hangs them from shrubs and fences. Fire wasps (*Polybia*) are found from Mexico to northern South America. The common species are black with yellow markings. They construct globular, cylindric, or cone-shaped paper nests up to 70 cm (27.6 in) long that are hung usually from trees and sometimes under bridges. They may defend these nests with great vigor.

Ants

Ants are social insects, worldwide in distribution over a wide range of habitats. Many ants sting, and others have repugnant secretions. The ant species of greatest medical significance in the United States is the imported fire ant *Solenopsis invicta* (see Figure 44-1E).[127] It was apparently introduced from South America into Mobile, Alabama, in 1939 and has subsequently spread throughout the southern states from southeastern Virginia to central Texas and Oklahoma, largely eliminating another introduced fire ant (*Solenopsis richteri*) and two native species. Mound nests are usually found in open grass settings, often in urban areas (Fig. 44-14). Other states at risk of harboring fire ants include Arizona, California, New Mexico,

Figure 44-14. Fire ant mound.

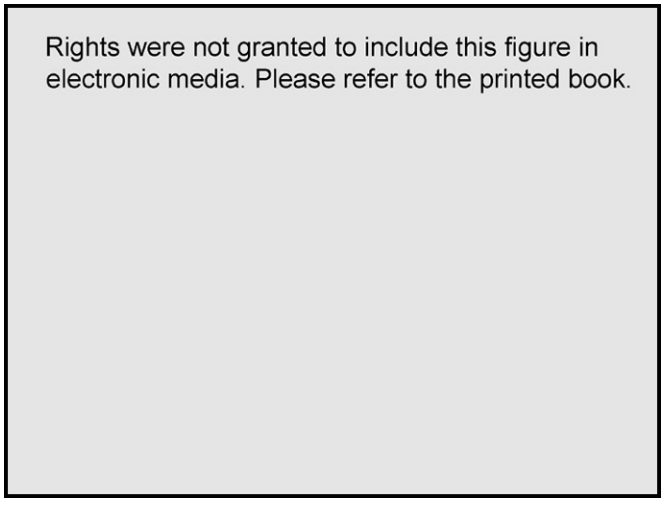
Oregon, and Washington, as well as Puerto Rico.[126] As many as 600 mounds per acre have been reported. Worker colonies may reach a maximum size of 500,000 ants in 2 years and rapidly give rise to satellite colonies.[256] *S. invicta* are extremely irritable insects. They are usually easy to identify, as they do not fly away but instead grasp their victims with their mandibles and inflict multiple painful stings.

Harvester ants (*Pogonomyrmex* species) of the southwestern United States and Mexico are of some medical importance. Entrances to the underground nests are usually surrounded by clear zones and sometimes by rings of soil. Some species react aggressively to disturbance of the nest. The stings are painful and may be accompanied by systemic symptoms; anaphylaxis has been reported.

There are numerous stinging species of ants in the tropics. Although native to South America, fire ants do not seem to be a major medical problem there, perhaps because of native competitors, predators, and parasites. Two fire ant species are important in areas outside the United States. *Solenopsis geminata* has been introduced into Okinawa and Guam and is widespread in Central America, Mexico, and some Caribbean islands. *Solenopsis xyloni* is common in Mexico and also occurs in California and Texas. Anaphylaxis caused by imported fire ants has also been described in Australia.[253] Severe human urticaria produced by the ant *Odontomachus bauri* has been reported in Venezuela.[226] Amino acid sequences of all fire ant venoms are very similar.[117] Therefore, all fire ant stings can be managed medically in the same manner.

The samsum ant, *Pachycondyla sennaarensis,* is an ecologic counterpart of the fire ant that is widely distributed in the African tropics and the Arabian Peninsula. It nests in the ground but does not make a conspicuous mound. In the United Arab Emirates, it is plentiful in urban areas and may cause multiple stings.[65] Australian bull ants (*Myrmecia* species), such as the "jumper jack," are large insects (about 20 mm or the size of a medium cockroach) with prominent jaws (Fig. 44-15).[217] They are ground dwelling and common in suburban areas in southeastern Australia. Many neotropical stinging ants live in trees. The giant black ants (16 to 22 mm) of the genus *Paraponera* are found from Nicaragua to the Amazon basin. Although they nest in the ground, workers forage in trees from almost ground level

A



B

Figure 44-15. **A,** Red bull ant. **B,** Australian jumper jack ant. (From www.richard-seaman.com).

to high in the forest canopy. They are most active at night. The green tree ant of northeastern Australia makes a leaf nest in trees. It has no true sting but ejects formic acid into wounds made by its jaws.

Hymenoptera Stinging Patterns

Multiple stings often result from disturbance of a nest, as the first insects encountered release alarm pheromones that incite aggressive behavior in other members of the colony. With large species such as the white-faced hornet, 40 to 50 stings may create a life-threatening injury.[276] The lethal dose of honeybee venom has been estimated at 500 to 1500 stings.[246] However, most of the 40 to 50 deaths per year in North America from Hymenoptera stings are the result of anaphylaxis occurring in victims with prior stings who developed specific immunoglobulin E (IgE) antibodies.[94] In the United States and other Western nations, the incidence of serious insect stings is higher in adults than in children, and higher in males than in females. Most persons are stung while engaged in outdoor work or recreation. Beekeeping is a high-risk occupation; however, many beekeepers develop considerable immunity as a result of frequent stings. Other relatively high risk occupations are farmer, house painter, carpenter, highway worker, and bulldozer operator. Wasps and

bees are sometimes swept into the interior of a moving automobile, exposing the occupants to risk of both a sting and a highway accident. Many foods, particularly meats, ripe fruit, or fruit syrups, attract yellowjackets, which often swarm around picnic areas and recycling bins. Syrups, flowers, sweat, and some perfumes attract bees. In such aggregations, the insects are not particularly aggressive but may become trapped in clothing or hair. A recent source documented that Africanized bees were more aggressive after exposure to 20% ethanol than to standard sucrose solutions placed in front of their hives.[2] In temperate zones, the incidence of hymenopteran stings is highest in late summer and early fall, when insect populations are highest.[23]

Fire ants may invade houses during periods of heavy rain and in hot, dry weather as they seek food and water.[62] There have been increasing incidents of fire ant attacks on patients in health care facilities, such as nursing homes, where the patients were incapable of fleeing for protection.[100] In one report, one patient experienced a severe anaphylactic reaction and four patients died within 1 week. The presence of fire ants around immobilized, often cognitively impaired patients seems to be a primary risk for massive fire ant attacks.[61]

Venom and Venom Apparatus

Venom is present in many hymenopteran species and is used for both defense and subjugation of prey. The venom apparatus is located at the posterior end of the abdomen and consists of venom glands, a reservoir, and structures for piercing the integument and injecting venom. Venoms of most medically important Hymenoptera are mixtures of protein or polypeptide toxins, enzymes, and pharmacologically active, low-molecular-weight compounds such as histamine, serotonin, acetylcholine, and dopamine. Melittin, a strongly basic peptide, is the principal component of honeybee venom, making up 50% of the dry venom weight. It damages cell membranes through detergent-like action, with liberation of potassium and biogenic amines.[50] Peptides with similar activity occur in bumblebee venom. Histamine release by bee venom appears to be largely mediated by mast cell degranulating (MCD) peptide. A third peptide, apamin, is a neurotoxin that acts principally on the spinal cord. Adolapin, a recently described bee venom peptide, has anti-inflammatory activity, which may explain the effectiveness of bee venom in treating some forms of arthritis. The chief enzymes of bee venom are phospholipase A and hyaluronidase. The former is believed to be one of the major venom allergens and, with melittin, to account for much of the acute lethality. Histamine makes up about 3% of the dry weight of bee venom. The intravenous (IV) median lethal dose (LD_{50}) of honeybee venom for mice is 6 mg/kg. An average sting injects about 50 µL of venom containing approximately 0.05 mg of solids.

Intense pain after stings by hornets and other social wasps is largely caused by serotonin and acetylcholine, which constitute 1% to 5% of dry venom weight. Wasp kinins (peptides) contribute to pain production and have strong, brief hypotensive effects. Mastoparans are similar in action to MCD peptide but are weaker. Phospholipase A, phospholipase B, and hyaluronidase are present in relatively large amounts. Unidentified proteins, some of which appear to be major allergens, are also present. A lethal protein in *Vespa basalis* venom releases serotonin from tissue cells and has hemolytic and phospholipase A activity.[115] The IV LD_{50} of different hornet venoms for mice ranges from 1.6 to 4.1 mg/kg.

Figure 44-16. Solitary mud dauber wasp.

Less is known of venoms of solitary wasps. The venom of *Sceliphoron caementarium,* a mud dauber (Fig. 44-16), is comparatively low in protein and contains no acetylcholine, histamine, serotonin, or kinins but does contain several unidentified low-molecular-weight compounds. Its proteins are immunologically different from those of honeybee, yellowjacket, and paper wasp venoms. Philanthotoxin (molecular weight 435) from venom of the beewolf (*Philanthus triangulum*) acts at the insect's myoneural junction and has potential value as an insecticide.

Ant venoms show great variation. In the life of the fire ant (*S. invicta*), venom plays several important roles, including prey capture, defense, and antimicrobial action. The synthesis of fire ant venom is limited to early life, and the injected venom dose appears to be carefully modulated. Older ant workers (foragers) deliver less venom per sting than mid-age workers (reserves), and the volume from nest defenders is 50% higher than from their counterparts, particularly in the spring.[108] Venoms of more primitive ants (subfamilies Ponerinae, Myrmicinae, and Dorylinae) resemble venoms of social wasps, containing kininlike peptides, enzymes, and unidentified proteins. In more highly evolved ants (subfamilies Dolichoderinae and Formicinae), a variety of low-molecular-weight compounds (terpenes, ketones, and organic acids) make up the bulk of the secretion, which may be sprayed rather than injected. Venoms of fire ants (*Solenopsis* species) are composed largely of piperidine alkaloids, which cause histamine release and necrosis in human skin. Proteins make up only 0.1% of the dry weight of fire ant venoms, but they are highly allergenic.[93,94] Hyaluronidase and phospholipase activities have been demonstrated.

Clinical Aspects

Hymenoptera stings are most often inflicted on the head and neck, followed by the foot, leg, hand, and arm. Stings in the mouth, pharynx, and esophagus may occur when bees or yellowjackets in soft drink, iced tea, or beer containers are accidentally ingested.[187,245] A single wasp, bee, or ant sting in an

Figure 44-17. Fire ant lesions.

unsensitized individual usually causes instant pain, followed by a wheal and flare reaction, with variable edema. Fire ants typically grasp the skin with their mouthparts and inflict multiple stings. These produce vesicles that subsequently become sterile pustules (Fig. 44-17). Multiple Hymenoptera stings may cause vomiting, diarrhea, generalized edema, dyspnea, hypotension, tachycardia, and collapse. Ocular stings with corneal involvement and bilateral ptosis have been described.[5,249,250] Widespread necrosis of skeletal muscle with hyperkalemia, rhabdomyolysis, acute tubular necrosis with renal failure, and hepatorenal syndrome with hemolysis have been reported.[43,131,148] Acute renal failure may result from toxic ischemic mechanisms, with hypovolemia or anaphylactic shock, myoglobinuria, or acute tubular necrosis.[26] Hemolytic anemia and acute renal failure requiring dialysis have also been described following massive attacks by 600 to 1500 Africanized bees.[52] Acute pancreatitis, disseminated intravascular coagulation, and multiorgan dysfunction are also well documented.[53,84,91,139,276] Myocardial infarction, atrial flutter, and atrial fibrillation in previously healthy individuals may follow multiple hymenopteran stings.[38,41,145,273] In one case, a 67-year-old man with acute-ST-segment-elevation myocardial infarction required fibrinolytic therapy.[155] Possible pathogenic mechanisms include severe hypotension due to hypovolemic shock, and prolonged coronary spasm with subsequent thrombosis of coronary vessels due to the release of vasoactive, inflammatory, and thrombogenic substances contained in Hymenoptera venom.[38] Cerebral infarction and descending aortic thrombosis are also reported.[48,219]

Dermatologically, large local reactions spreading more than 15 cm (6 inches) beyond the sting site and persisting more than 24 hours are relatively common. Formation of large bullae may also occur.[148] This represents a cell-mediated (type IV) immunologic reaction, although more than half these patients also have IgE antibody against venom or show a positive skin test. Later stings in these individuals usually result in another large local reaction; systemic reactions are rare.[282]

Allergy is the most serious aspect of hymenopteran stings. Anaphylaxis and related syndromes from this source are relatively common outdoor wilderness emergencies. An estimated 0.4% of the U.S. population shows some degree of clinical allergy to insect venoms, and 40 to 50 deaths are reported annually.[213] This number may be underestimated, because not all anaphylactic episodes are recognized or reported. Fatal anaphylaxis due to fire ant stings has also been reported.[206] Asymptomatic sensitization, as shown by positive venom skin test, was observed in 15% of 269 randomly selected subjects with no history of an allergic sting reaction.[102] Sensitization is transient but may persist for years. These individuals are at higher risk of systemic allergic reactions than are those with negative skin tests.[103]

Sudden death from insect sting may not always be recognized. Unexplained deaths at poolsides, golf courses, or any recreational area may be caused by unrecognized Hymenoptera envenomations.[68] Of 142 sera obtained after sudden, unexpected death, 23% contained elevated levels of IgE to at least one insect venom. In contrast, 6% of sera from 92 blood donors contained comparable IgE levels. In eight fatal cases of Hymenoptera sting anaphylaxis, IgE to the putative venom source was elevated in all, although levels were not higher than those of some healthy individuals in the same population.[242] Allergic-specific IgE antibodies to imported fire ants is nearly twice as common in adults living in endemic areas, making these individuals more susceptible to severe anaphylaxis.[39] Anti–fire ant IgE and elevated serum tryptase were detected in a case of fatal fire ant sting.[183] Elevated levels of venom-specific IgE were detected in two fatal cases of wasp sting.[267] However, the level of specific IgE antibodies against venoms is not predictive of the severity of anaphylactic reaction, as documented by recent studies conducted to determine the reliability of postmortem specific IgE antibody testing in venom-induced anaphylactic deaths.[118]

Wasp and bee venoms contain 9 to 13 antigens, some of which are potent allergens. Available evidence indicates little cross-sensitization between honeybee and wasp venoms. About 50% cross-sensitization occurs between *Polistes* and other social wasp venoms, and nearly 100% between yellowjacket and hornet venoms. Positive radioallergosorbent test (RAST) reactions to imported fire ant venom were seen in 51% of patients allergic to bee and wasp venoms but without exposure to fire ants. The allergen appears to be identical to antigen 5 of wasp venoms.[116]

Examination of sera of hypersensitive individuals for IgE and IgG antibodies against purified venom proteins indicates that phospholipase A, hyaluronidase, and acid phosphatase are important in honeybee venom, whereas phospholipase A, antigen 5, and hyaluronidase are important in wasp venoms. Antigen 5, a nontoxic protein of unknown activity, is reported to have sequence similarity to mammalian testis, human brain tumor, and certain plant leaf proteins. This may explain anaphylactic reactions to first insect stings.[133,274] Despite the small amount of protein in fire ant venoms, about 12% of persons treated for fire ant stings show systemic allergic reactions, and at least 32 anaphylactic deaths have been confirmed. Four antigens in *Solenopsis invicta* venom have been reported to be allergenic.[37]

Allergic sting reactions occur remote from the sting site and include flushing, pruritus, papular urticaria,[257] hives, and angioedema. In life-threatening reactions, marked respiratory distress with airway edema, hypotension, loss of consciousness, and cardiac arrhythmias may be seen. At least half the severe reactions occur within 10 minutes after a sting, and virtually all occur within 5 hours. Most fatalities occur within 1 hour. The

interval between the first known sting and the reaction-producing sting is usually less than 3 years but may be as long as 48 years. In a group of 3236 Hymenoptera-allergic individuals, 61.5% were male and 32.3% had a history of atopy. The mean age was 30.5 years. No correlation existed between systemic reactions and number of stings in the past or number of stings per incident and severity of a systemic reaction.[152] In a series of 138 adults with a history of insect sting anaphylactic reactions, 99 had no anaphylactic reactions to later stings, 17 had more severe reactions, and 22 had mild to moderate reactions.[270] In another series of 90 adults with previous anaphylactic reactions, 60 had similar reactions when restung, and 23 had more severe reactions.[212] In children 10 years and younger, life-threatening reactions occur less often than in adults, and repeated sting episodes usually are not increasingly severe. However, 17% of children with a history of systemic bee or wasp sting reactions developed a systemic reaction after a sting challenge test, as did 5% of children who sustained a sting in the field.[112] Children were once thought to outgrow the allergy to insect stings, but there are no reports accurately documenting this theoretical maturation process.[101,106]

Fatalities that occur within the first hour after a sting result from airway obstruction, hypotension, or both. In 69% of fatal cases, obstruction of the respiratory tree by edema or secretions was the principal finding at autopsy; in 12%, vascular pathology was the principal finding; and 7% of the victims had primary central nervous system involvement such as petechial hemorrhages, infarction, and cerebral edema.[16] Hemostatic defects, including reduction of all clotting factors and release of a thrombin inhibitor, may be seen with insect sting anaphylaxis. Severe fetal brain damage, presumably associated with hypoxia, has been reported. Delayed (3 to 14 days) atypical reactions after hymenopteran stings include serum sickness and Arthus reaction, which are caused by systemic and local effects of antigen–antibody complexes; nephrotic syndrome; thrombocytopenic purpura; grand mal and focal motor seizures; transient cerebral ischemic attacks; Guillain-Barré syndrome; and progressive demyelinating neurologic disease.[232] Most appear to be immunologically mediated. In one series, elevated IgE to bee or yellowjacket venom was observed in 6 of 13 such patients.[146,170]

Identification of the individual with potentially dangerous allergy to hymenopteran sting is not always possible. Skin testing with hymenopteran venoms is the most sensitive method; RAST for IgE antibody to venoms is less sensitive.[135] A small but significant number of individuals with no history of sting reactions have IgE antibody specific for hymenopteran venoms; prevalence of this antibody is higher in summer.[287] These methods do not identify all at risk, and antibody levels do not correlate with severity of sting reactions. In a significant number of individuals, particularly children, clinical sensitivity disappears and IgE levels fall virtually to zero 3 to 18 months after a reaction-producing sting. In about 40% of cases, sensitivity may disappear within 3 years.[144] Venom antibody (both IgE and IgG) may be found in healthy individuals (40% of beekeepers, 12% of blood donors) with no history of systemic reaction to insect stings.

No unique features distinguish Hymenoptera envenomations in other parts of the world from those in North America. Venoms of the various groups show little geographic variation. This is also true of groups at risk, with the possible exception of a few honey-gathering Asian tribes. The incidence of Hymenoptera sting allergy may be slightly higher in western Europe than in the United States, and fatal allergic reactions may be slightly more common.[46] Anaphylactic reactions to bee venom are more prevalent in the rural populations closer to the Mediterranean than in western Europe, where urban populations predominate.[174]

Paraponera ant stings are intensely painful for several hours and may be accompanied by fever and lymphadenitis. Systemic anaphylactic reactions to stings of Australian bull or jumper jack ants, as well as samsum ants, are increasing, with reports of fatalities.[30,165] Acute renal failure has also been described.[1] Patients with history of systemic reactions to samsum ant stings have IgE and positive skin test reactions to fire ant venom.[65]

Treatment and Prevention

Treatment of anaphylaxis is conventional. Aqueous epinephrine 1:1000 should be administered subcutaneously at the first indication of serious hypersensitivity. The dose for adults is 0.3 to 0.5 mL, and for children under age 12 years it is 0.01 mL/kg, not to exceed 0.3 mL. When symptoms are predominantly respiratory, epinephrine by inhalation (10 to 20 puffs for an adult; 2 to 4 puffs per 10 kg of body weight for a child) may provide more rapid relief.[179] In the presence of profound hypotension, the patient may be in shock and not adequately perfusing the skin. In this scenario, 2 to 5 mL of a 1:10,000 epinephrine solution may be given by slow IV push, or an infusion may be initiated by mixing 1 mg in 250 mL and infusing at a rate of 0.25 to 1 mL/min. If an IV line is delayed or cannot be established, epinephrine may be given intramuscularly, intralingually, or via an endotracheal tube.[11] Recently, two cases of Hymenoptera-induced anaphylactic shock with profound hypotension were successfully resuscitated using 40 IU of IV vasopressin followed by a rapid fluid bolus.[132] Selective inhaled (nebulized) β_2-adrenergic agents, such as albuterol, can also be effective in relieving bronchospasm at doses of 2.5 mg/3 mL of a 0.08% solution. Aminophylline, 5 mg/kg as a loading dose followed by 0.9 mg/kg/hr as an infusion, may relieve bronchospasm not relieved by epinephrine or albuterol.

In the presence of hypotension, IV crystalloid solutions should be infused; pressor agents such as dopamine or norepinephrine may be required. Oxygen, intubation, and mechanical ventilation may be needed to correct airway obstruction. Antihistamines can alleviate symptoms of anaphylaxis but should be used only in addition to epinephrine, not as a substitute.[11] Corticosteroids are also indicated in acute anaphylactic reactions, although the time of onset with corticosteroids is delayed. Propranolol is contraindicated because of the β-adrenergic blockade effect on the bronchioles. Persons taking β-adrenergic blockers may respond poorly to epinephrine. In these cases, administration of glucagon to counteract the beta blockade effect may prove beneficial.[221] Persons with insect sting anaphylaxis require close observation, preferably in the hospital, over a period of 24 hours.[179] For mild hymenopteran stings, ice packs often provide relief. Honeybees frequently and yellowjackets occasionally leave a stinger in the wound. Although recommendations were that stingers should be scraped or brushed off with a sharp edge and not removed with forceps, which might squeeze the attached venom sac and worsen the injury, this has been refuted.[222,272] Advice to victims on the immediate treatment of bee stings now emphasizes rapid removal of the stinger by any method.[272] Wheal size and degree of envenomation increased as the time from stinging to stinger removal increased, even for a few seconds. The response was the same

whether stings were scraped or pinched off after 2 seconds. Home remedies, such as baking soda paste or meat tenderizer applied locally to stings, are of dubious value, although the latter is often regarded as effective. Topical anesthetics in commercial "sting sticks" are also of little value. Topical aspirin paste is not effective in reducing the duration of swelling or pain in bee and wasp stings and may actually increase the duration of redness.[14] Local application of antihistamine lotions or creams, such as tripelennamine, may be helpful. An oral antihistamine, such as diphenhydramine, 25 to 50 mg for adults and 1 mg/kg for children, every 6 hours is often effective.

All fire ant stings can be managed medically in the same manner. No therapy is effective against local effects of fire ant stings, although oral antihistamines and corticosteroids may provide some relief in severe cases. Because infection is common, topical antimicrobials (e.g., mupirocin) and prophylactic oral antibiotics are recommended. Breaking fire ant blisters should be avoided.[49,60]

Corticosteroids, such as methylprednisolone, 24 mg/day initial dose tapered off over 4 to 5 days, often help resolve extensive local reactions to bee and wasp stings. This may be combined with cold packs and oral antihistamines. Chronic facial edema caused by multiple bee stings has been effectively treated with plastic surgery and liposuction.[4]

Envenomation from multiple hymenopteran stings may require more aggressive therapy. IV calcium gluconate (5 to 10 mL of 10% solution) with a parenteral antihistamine and corticosteroid may be helpful in relieving pain, swelling, nausea, and vomiting. Development of a hyperimmune bee venom antiserum is under investigation.[239] Hypovolemic shock is managed conventionally. Plasmapheresis was used successfully to treat a person who sustained about 2000 honeybee stings.[64] Patients should be observed for 12 to 24 hours for coagulopathy and evidence of renal and neurologic damage. Urine output is monitored and urine tested for hemoglobin and myoglobin. Serum potassium, creatine kinase, and lactate dehydrogenase levels should be monitored. Oliguria with myoglobinuria, azotemia, and hyperkalemia are indications for hemodialysis.

Immunotherapy

Immunotherapy should be offered to patients with a history of anaphylaxis after a sting and specific IgE antibodies to the agent confirmed by positive skin testing or in vitro assays.[94] Desensitization with purified venoms produces an excellent blocking antibody response and prevents anaphylaxis in more than 95% of patients. A protective anti-idiotypic antibody to honeybee venom has been identified.[129] Venoms for desensitization generally available in the United States are honeybee, yellowjacket, wasp (*Polistes*), and mixed vespid. More than one venom can be given concurrently, but this requires multiple injections. Commercial preparations consisting of a mixture of yellowjacket, white-faced hornet, and yellow hornet venoms are available.[94] A whole-body extract of fire ant containing at least three venom antigens has also been developed.[275] Persons receiving β-adrenergic blockers should be shifted to other appropriate medications if possible. Children under 16 years with only cutaneous or mild systemic allergic reactions and persons with a history of only large local reactions do not need immunotherapy.[269] Evaluation of anaphylaxis risk is recommended in children using wasp venom extract challenges.[237] As mentioned previously, children were once thought to "outgrow" their allergies to insect stings, but this has never been proven. In fact, a clinically important number of children do retain their allergic reactions to Hymenoptera venom. Immunotherapy in children leads to a significantly lower risk of systemic reaction to stings even 10 to 20 years after treatment is completed. This prolonged benefit is greater than that observed when the immunotherapy is given to adults.[101,106]

Regimens for desensitization attempt to achieve tolerance to venom doses of about 100 µg. It requires about 95 days to achieve a maintenance level of immunity. Rapid or "rush" programs requiring 3 to 7 days for initial immunization appear to be effective in high-risk patients.[19,73,259,277] Some programs make use of both active and passive immunotherapies.[178] In a series of 1410 patients, 12% had systemic reactions during treatment, but no fatalities were reported.[151,153] A report of 26 women with 43 pregnancies does not suggest significantly increased risk from venom immunotherapy during pregnancy.[241] Maintenance doses are required at intervals after basic immunization. Neither skin testing nor determination of IgG and IgE antibody levels against venom will reliably indicate success of immunization, although the majority of persons will be protected by a specific IgG antibody level of 400 RAST units/mL of serum. Actual sting challenge is the most reliable test for determining immunotherapy candidates and desensitization,[271] but this is not widely used in the United States. It must be done in the hospital with careful monitoring and consideration of economic, ethical, and safety factors.[229] If the skin test is negative after 3 years of immunotherapy, patients may be placed on immunologic surveillance. Few patients require more than 5 years of immunotherapy.[213] According to some authorities, if a sting challenge or field sting is tolerated during the period of immunotherapy, treatment can be terminated after 3 to 5 years.[207] For unknown reasons, desensitization to wasp venoms is achieved more quickly than to honeybee venom.[24] Although the low mortality and morbidity associated with patients who suffer subsequent stings following immunotherapy has led some to conclude that many patients are being treated unnecessarily, this conclusion is refuted by the fact that venom immunotherapy improves the quality of life and health of the vast majority of patients receiving this intervention.[194]

Like bee venom immunotherapy, purified *Myrmecia* ant venom immunotherapy has been established and shown to be highly effective for Australian bull or jumper ant anaphylaxis.[27,29,30,165]

Antivenom Therapy and Future Interventions

Recently, a group in the United Kingdom developed an ovine Fab-based antivenom as a potential treatment for mass bee stings. Sera from sheep immunized against the venom of *A. m. scutellata* contained high levels of specific antibodies, as demonstrated by enzyme-linked immunosorbent assay (ELISA) and chromatography. Although effective experimentally in a mouse model, no human administration of the antivenom has been documented.[125]

Another approach based on genetic engineering is the use of non-IgE binding peptide fragments of the insect allergen with preserved T-cell epitopes for advanced immunotherapy. Such preparations of bee venom phospholipase A2 have been used successfully in pilot studies. Additionally, DNA vaccination with phospholipase A2 sequence plasmids has been proven effective in a mouse model.[177]

Figure 44-18. EpiPen and EpiPen Jr preloaded delivery systems for injection of aqueous epinephrine. (Courtesy Dey, LP.)

Preparedness and Preventive Measures

Persons with a history of allergic reactions to insect stings (including large local reactions) should carry an emergency kit containing epinephrine autoinjectors and should wear medical identification tags.[94] Kits should be available in work and recreation areas where the risk of insect sting is high. Two kits widely available in the United States are EpiPen and Ana-Kit. EpiPen and EpiPen Jr. are autoinjectors that deliver 0.3 mg or 0.15 mg of epinephrine, respectively (Fig. 44-18). They are quick and easy to use; however, patients should be cautioned against injecting the material into fingers or buttocks or directly over veins. Ana-Kit contains two doses of 0.3 mg of epinephrine in a single conventional syringe, plus chewable antihistamine tablets and a tourniquet. It is more versatile but requires more instruction for the user.

Frequent cleaning of garbage cans and disposal of decaying fruit makes premises less attractive to bees and wasps. Hymenopterans are highly susceptible to many insecticides, and their control around dwellings and other inhabited buildings is rarely difficult. Spraying the nests after dark is safer because these insects are less active at night. Many hymenopterans are economically valuable as pollinators of plants or predators on other insects, so their control on a wide scale is rarely desirable. The fire ant in the southern United States has been the target of massive but marginally effective control campaigns that adversely affected local ecosystems. A new approach uses grain baits containing synthetic insect growth hormones that are carried into the nests, where they disrupt ant caste differentiation and inhibit egg production. Arrays of thousands of hormone-baited traps placed in selected areas of Mexico, however, failed to stop the northward spread of Africanized bees. For controlling populations of Africanized bees, items such as boxes and empty oil drum containers should not be left outdoors. Ceilings and walls should be sealed off as potential nesting sites for colonies and swarms.[57]

LEPIDOPTERA

Venomous Species and Venoms

Insects of the order Lepidoptera typically cause human envenomation, but effects are generally less serious than with hymenopterans. Injury usually follows contact with caterpillars and is less frequent with the cocoon or adult stage. The larval lepidopteran (caterpillar) is usually free living, is moderately active, and feeds on plants, although a few are parasites of insect nests or eat food of animal origin. The pupal stage may be free or encased in a silk cocoon. Wintering over in cold climates is usually in the pupal stage. Adults (butterflies and moths) have wings with microscopic chitinous scales. They feed primarily on nectar and other plant juices, but some eat semiliquid mammalian feces and urine. The adult provides no care or protection of immature stages. No social organization exists, although larvae and adults of some species assemble in large aggregations.

Figure 44-19. Puss caterpillar, *Megalopyge opercularis*.

Venomous species occur in about 16 families of Lepidoptera, with no general rules for recognition. Many venomous caterpillars are broad, flat, and sluggish. Some have the dorsal surface densely covered with long hairs. Others are spiny and may have bright, conspicuous colors and markings. Some are highly camouflaged.

Venoms in Lepidoptera are purely defensive. The venom apparatus consists of spines that are simple or branched and frequently barbed. They may be scattered widely over the surface of the insect or arranged in clumps and often are intermixed with nonveniferous hairs or spines. In the most venomous caterpillars, the spines are hollow and brittle with venom glands at the bases. Muscles surrounding the glands may help in expelling venom. In other Lepidoptera, the spines are solid and function primarily as mechanical irritants or contain surface toxicants. Little is known of the chemistry of caterpillar venoms. Some are heat labile and contain proteins. Histamine and serotonin have been found in caterpillar venoms but are not common. Hairs of the brown-tailed moth (*Euproctis chrysorrhea*) contain enzymes with esterase and phospholipase activity.

Probably the most important venomous caterpillar in the United States is the puss caterpillar[98,244] or woolly slug (*Megalopyge opercularis*) (Fig. 44-19). This caterpillar is distributed throughout the southern states west through most of Texas and north to Maryland and Missouri. The hairy, flat, and ovoid caterpillar reaches a length of 30 to 35 mm (1.2 to 1.4 inches) and feeds on shade trees, including elm, oak, and sycamore. Some years it may be plentiful enough to be a nuisance. In southeast Texas in 1958, 2130 persons were treated for stings, and eight were hospitalized. A related species, the flannel moth caterpillar (*Megalopyge crispata*) (Fig. 44-20), occurs in the

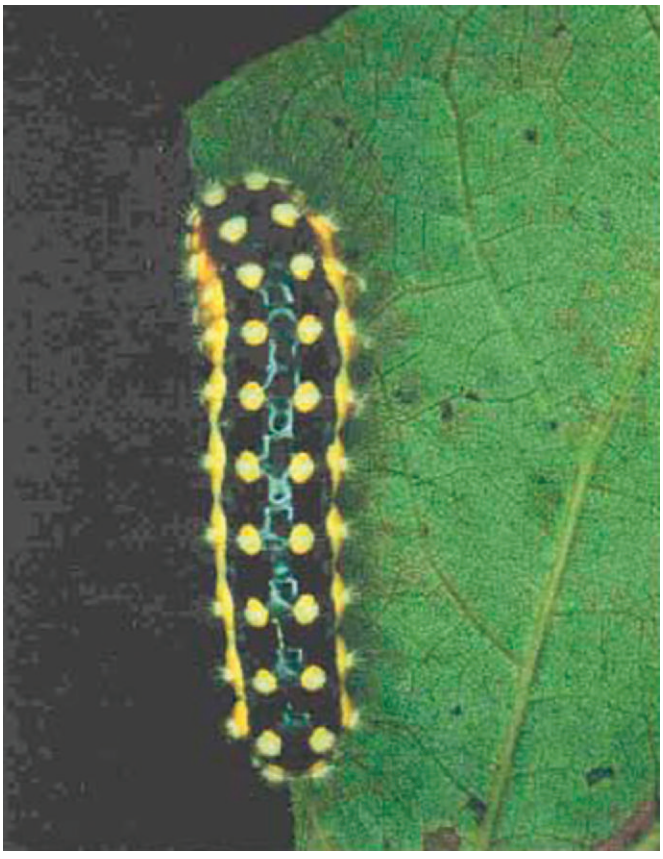


Figure 44-20. Flannel moth caterpillar, *Megalopyge crispata*.

Figure 44-21. Caterpillar of the io moth, *Automeris io*. Widespread in the eastern United States, this species can inflict a painful sting.

Figure 44-22. Saddleback caterpillar, *Sibine stimulea*.

Figure 44-23. Gypsy moth, *Lymantria dispar*. (From www.uwex.edu.)

eastern states north to New England. Its sting is less severe than that of *M. opercularis*. The large, spiny caterpillar of the io moth (*Automeris io*)[81] is pale green with red and white lateral stripes (Fig. 44-21). It is widely distributed in the eastern United States but rarely plentiful. The saddleback caterpillar (*Sibine stimulea*) (Fig. 44-22) and oak slug (*Euclea delphinii*) are flat and almost rectangular; both can deliver a painful injury. The gypsy moth (*Lymantria dispar*) (Fig. 44-23) feeds on a variety of plants and has caused thousands of cases of dermatitis in the northeastern United States. Other common nettling caterpillars are *E. chrysorrhea*, which also occurs in Europe, and the tussock or toothbrush caterpillar (*Hemerocampa leucostigma*), with its conspicuous red head and four tufts of bristles. Another tussock caterpillar, *Oryia pseudotsuga*, causes numerous cases of dermatitis and conjunctivitis among lumberworkers and foresters in the northwestern states. The hickory tussock caterpillar (*Lophocampa caryae*) can cause skin irritation and urticaria with dermal contact, or drooling if ingested (Fig. 44-24).[140,204] The eastern tent caterpillar, *Malacosoma americanum* (Fig. 44-25), is native to North America. Populations fluctuate from year to year, with outbreaks occurring every several years. Defoliation of trees, building of unsightly silken nests in trees, and wandering caterpillars crawling over plants, walkways, and roads cause this insect to be considered a pest in the late spring and early summer. Eastern tent caterpillar nests are commonly

Figure 44-24. Hickory tussock caterpillar, *Lophocampa caryae*. (From www.bnl.gov/wildlife/images. Courtesy of Brookhaven National Laboratory.)

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Figure 44-25. Eastern tent caterpillar, *Malacosoma americanum*. (From www.richard-seaman.com.)

found on wild cherry, apple, and crabapple trees but may be discovered on other various fruiting trees.

Stinging Patterns

Caterpillar envenomation usually occurs when living insects are touched as they cling to vegetation or drop onto bare skin. Persons cutting branches, picking fruit, or climbing trees are likely to be stung. However, the largest outbreaks have been associated with spines detached from live or dead caterpillars and cocoons. These may be airborne or deposited on bedding or laundry hung outdoors. In temperate regions, caterpillar stings are most common from August to early November. Heavy caterpillar infestations seem to occur during exceptionally favorable weather and with decreases in populations of parasites and predators that serve as natural controls.

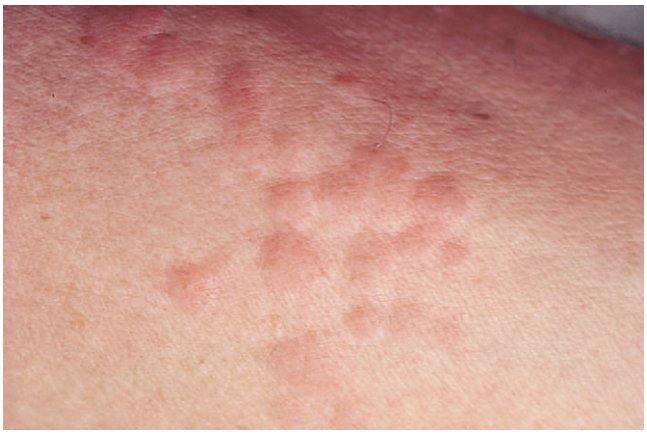


Figure 44-26. Nettle rash from unidentified caterpillar.

Clinical Aspects

Two general syndromes are associated with lepidopteran envenomations. In the case of caterpillars with hollow spines and basal venom glands (e.g., *Automeris, Megalopyge,* and *Dirphia*), direct contact with the live insect causes instant nettling pain, followed by redness and swelling (Fig. 44-26). Puss caterpillar stings show a characteristic gridiron pattern of hemorrhagic pinpoint papules. In typical cases, no systemic manifestations occur, and symptoms usually subside within 24 hours. However, pain may be intense with central radiation, accompanied by urticaria, nausea, headache, fever, vomiting, muscle spasms, paresthesias, and lymphadenopathy. Hypotension, shock, dyspnea, abdominal tenderness, and convulsions have been reported with more severe cases of puss caterpillar stings.[81,96,98,119,244]

The second syndrome is associated with caterpillars with a less highly developed venom apparatus (e.g., *Lymantria, Euproctis, Thaumetopoea*). Contact with the living insect is not necessary; detached spines are often involved. Little or no immediate discomfort is experienced. An itching, erythematous, papular, or urticarial rash develops within a few hours to 2 days and persists for up to a week (Fig. 44-27). Rarely, the lesions may be bullous. Conjunctivitis, upper respiratory tract irritation, and rare asthma-like symptoms may be seen with or without dermatitis. Ophthalmia serious enough to require enucleation may be caused by detached spines lodged in the eye. Acute anaphylactic reactions have not been reported to follow lepidopteran stings. Patch testing has demonstrated both immediate and delayed hypersensitivity. A pediatric case series reported 10 patients who ingested various caterpillar species. Adverse effects went from mild (drooling and refusal to drink) to diffuse urticaria. Five of the patients underwent direct laryngoscopy and endoscopy to assess for pharyngeal and esophageal injuries. None had adverse outcomes.[204]

Treatment and Prevention

Treatment of lepidopteran envenomations is symptomatic. Prompt application and stripping off of adhesive tape or a commercial facial peel at the site of the sting may remove many spines and serve as a diagnostic procedure, as the spines can then be identified by microscopy (Fig. 44-28). Patients with

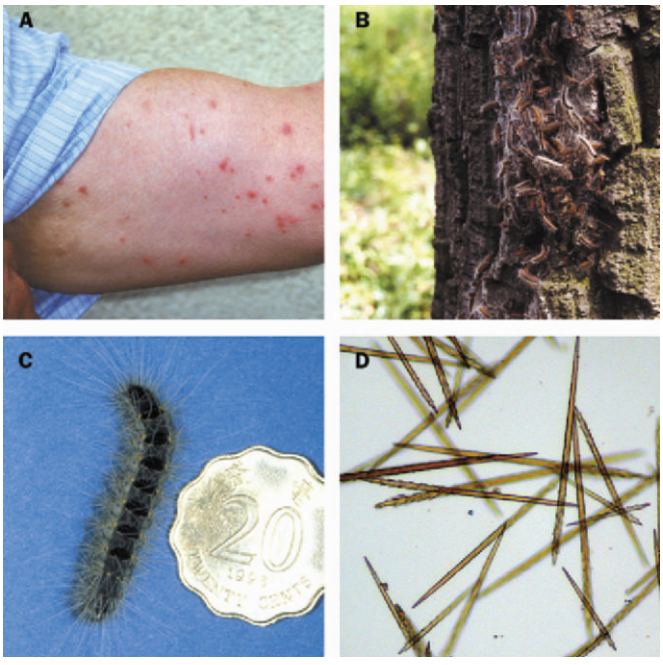


Figure 44-27. Lepidopterism. **A,** Toxic irritative caterpillar dermatitis. **B,** Silken nest on the trunk of an infested oak tree with living larvae. **C,** Sixth larval instar of *Thaumetopoea processionea* L. **D,** Setae (×200). (From Spiegel W, Maier H, Maier M: A non-infectious airborne disease. Lancet 363:1438, 2004.)

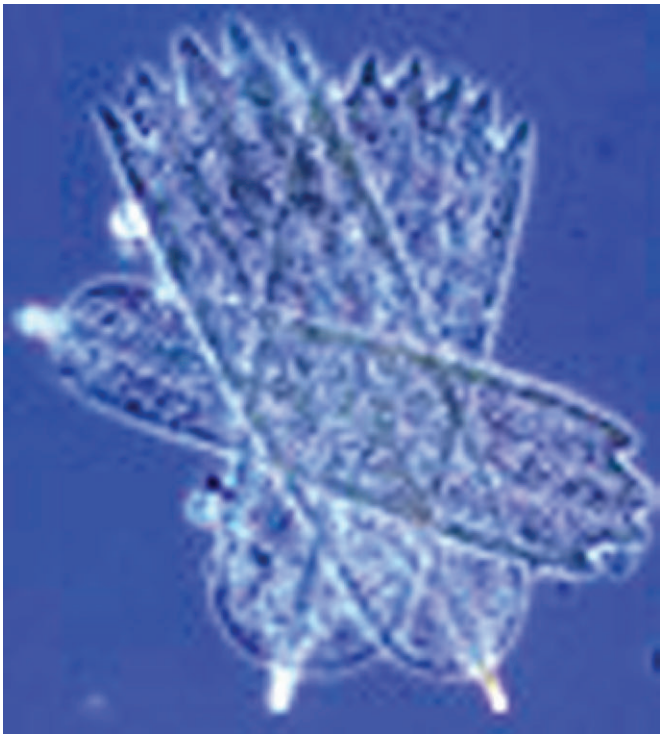


Figure 44-28. Microscopic view of Lepidoptera spines. (From www.microscope.mbl.edu.)

Figure 44-29. Caterpillar of *Lonomia achelous*, which can inflict injuries characterized by potentially fatal coagulopathy.

Figure 44-30. Moth of *Lonomia achelous*, which has no venomous spines.

local symptoms usually obtain relief from group I corticosteroid creams and ointments. Over-the-counter preparations containing corticosteroids and antihistamines are not significantly better than simpler preparations such as calamine lotion with phenol. Oral antihistamines such as fexofenadine (60 mg, twice a day) or anti-inflammatory drugs such as tolmetin sodium (400 mg, three times a day) are often effective in more severe cases. Occasionally, codeine (30 to 50 mg) or oxymorphone (1.5 mg) in combination with an antiemetic may be needed to control pain and vomiting. Intravenous calcium gluconate has been used successfully in severe puss caterpillar envenomation to control muscle spasms.[186,203]

Trees on which caterpillars feed may be sprayed with appropriate insecticides to control species such as the puss caterpillar. Screens on windows and doors protect against moths with toxic spines.

Internationally, Lepidoptera show high diversity in the tropics with correspondingly greater medical importance, particularly in Latin America. Caterpillars of the genus *Lonomia* native to northern South America can inflict life-threatening stings (Figs. 44-29 and 44-30). These caterpillars are 50 to 70 mm (2 to 3 inches) long and have numerous branched dorsal spines. They live in primary tropical forests in groups of up to about 50 indi-

viduals. Disturbance of their habitat has resulted in an increasing number of envenomations. Venom of *Lonomia* is a protein that activates prothrombin and is stimulated by factor V and calcium ions.[107] Stings cause intense pain but not much local reaction. Signs of coagulopathy, such as ecchymoses, bleeding gums, hematuria, and melena, may develop in a few hours or be delayed for several days. Fibrinogen, factor V, factor XII, and plasminogen are decreased, fibrin degradation products are increased, and platelets usually are normal. Acute renal failure,[32] cerebral hemorrhage, and pulmonary hemorrhage may occur. Coagulopathy may last 2 to 5 weeks.

In one series of 33 cases, four were fatal.[9,32,72] Treatment with prednisone, plasma, and whole blood is ineffective. An antivenom has been developed, and preliminary reports indicate possible clinical efficacy.[54,56]

Neotropical caterpillars of the genera *Dirphia, Megalopyge,* and *Automeris* are large, stout, spiny, and sometimes covered with hair. Most are forest species but can adapt to areas of cultivation. Agricultural workers are most often stung and the incidence of stings is higher in the rainy season. Intense, centrally radiating pain with local edema, erythema, and lymphadenopathy is typical. Systemic symptoms include nausea, headache, malaise, chills, and fever. Hypotension, shock, and convulsions have been reported. An *Automeris* caterpillar bite reported from French Guyana produced syncopal pain and edematous infiltration of the thigh lasting several days.[51] Symptoms usually subside within 24 hours. Treatment is symptomatic. Oral antihistamines are often effective if given within about an hour after the sting. Opioid agents are occasionally needed to control pain.

A chronic granuloma known as pararama occurs on the hands of Brazilian rubber tappers after contact with *Premolis semirufa* caterpillars. Permanent extremity disability may result.[202]

Moths of the genus *Hylesia* are found from southern Mexico to Argentina. The caterpillars have venomous spines, but the greatest problem is created by the moths, which have a coating of spines on their abdomen. The spines or setae are hollow and pointed, contain a toxin of unknown nature, and are freely shed into the air. The moths are attracted to lights in enormous numbers, and their airborne spines can cause great discomfort. Their activity has created serious problems at airports, shipping docks, and tourist resorts. Within a few minutes to a few hours after contact with the spines, victims develop a pruritic, erythematous rash that progresses to urticaria and excoriation. Any portion of exposed skin may be involved, but palms and soles are often spared. Irritation of eyes and mucous membranes is unusual. Symptoms subside in about a week if there is no further exposure. Topical and systemic treatments have had little benefit.[66]

In Korea, outbreaks of dermatitis, presumably caused by setae of the yellow moth *Euproctis flava,* are well known. In the summer of 1980, hundreds of U.S. soldiers were affected.[18] The caterpillars feed on hardwood trees, and great numbers of moths appear in summer and are attracted to lights. Dermatitis usually involves direct contact with moths or their cocoons or with clothing contaminated with setae. The lesions are similar to those described for *Hylesia* and are equally refractory to treatment. Other outbreaks of dermatitis ascribed to *Euproctis* moths and caterpillars have been reported in Japan and China. One outbreak in Shanghai in 1972 affected about 500,000 individuals. Outside Shanghai, where chemical insecticides would have been harmful to silkworm culture, *Euproctis* caterpillars (Fig. 44-31) have been controlled by spraying with an insect virus. Cases of *Euproctis* dermatitis and ophthalmia have also been reported in Australia and Great Britain. Sensitization with elevated IgE levels may occur.[202,254]

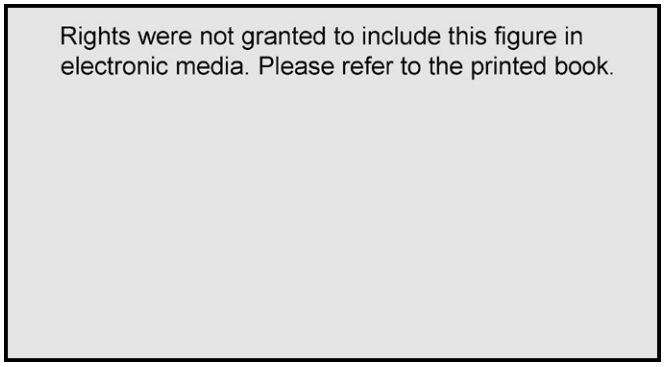


Figure 44-31. *Euproctis* caterpillar. (From www.gardensafari.net.)

In the Mediterranean region and the Middle East, the pine processionary caterpillars *Thaumetopoea pityocampa* and *Thaumetopoea wilinsoni* are plentiful and make silk nests in trees. The setae from these caterpillars can cause a maculopapular rash accompanied by urticaria, bronchitis, and conjunctivitis. A non–IgE-mediated release by *Thaumetopoea* results in a type I hypersensitivity foreign-body reaction resulting in dyspnea and bronchospasm.[255] Outbreaks typically occur when groups of tourists or military personnel camp in pine groves. The rash usually results from contact with detached setae rather than with caterpillars. The adult moth stage apparently does not have irritating spines. Systemic reactions manifesting as abdominal pain and hypertension have been reported.[137]

Moths of the genus *Calyptera,* native to Southeast Asia, have a serrate proboscis and feed on mammalian blood, including that of humans. Tropical species of several moth genera feed on human ocular secretions. Their medical importance is unknown. In Australia, five families of caterpillars that inflict lesions resulting in local pain and urticaria have been described (Arctiidae, Limacodidae, Anthelidea, Lymantriidae, and Spingidae). All dermal reactions were responsive to ice packs and antihistamines. Ingestions resulted in no adverse side effects.[12]

CENTIPEDES AND MILLIPEDES

Centipedes are elongate, flattened arthropods with one pair of legs for each of the typical body segments, which may number from 15 to more than 100. The first segment bears a pair of curved hollow fangs with venom glands at the bases. The last segment bears a pair of filamentous to forceps-like caudal appendages not associated with the venom apparatus.[35] The largest species reach lengths of about 30 cm (12 inches). Most centipedes live in crevices or beneath objects on the ground. Some are burrowers and others are climbers. Many are nocturnal. *Scutigera coleoptrata*, with body length of 25 mm (10 inches) and long thin legs, is a common house arthropod in much of the United States. *Lithobius* is a cosmopolitan ground-dwelling genus. A species common in eastern U.S. gardens is

orange and 30 to 50 mm (1 to 2 inches) long. Envenomations by imported varieties of the Vietnamese centipede have been reported in Long Island, New York.[164] Centipedes prey chiefly on invertebrates, but larger species occasionally eat small vertebrates. Female centipedes of some species curl around their egg clusters and newly hatched young and may actively defend them.

Centipedes use venom primarily to kill prey and only secondarily for defense. Venom may also have a digestive function. Enzymes, including acid and alkaline phosphatase and amino acid naphthylamidase, lipoproteins, histamine, and serotonin, are variably present.[175] Venom of *Scolopendra subspinipes* produces hypotension followed by hypertension. The major lethal toxin is an acidic protein with a molecular weight of 60,000 daltons. It produces vasoconstriction, increased capillary permeability, and cardiotoxicity.[104,105]

Like spiders, any centipede whose fangs can penetrate human skin can cause local envenomation. Centipede bites are typically pointed in shape, a feature that can help differentiate the bite of a large centipede from that of a snake.[77,149] Contrary to popular folklore, centipedes do not inject venom with their feet or caudal appendages. The jaws inject a neurotoxic venom through venom ducts. Centipede bites have been reported from numerous tropical and subtropical regions but never as a serious medical problem. Most bites have been ascribed to species of *Scolopendra,* which has a wide distribution with several species in the southern United States (Figs. 44-32 and 44-33). Fatalities are almost unknown; however, a death in the United States was recently mentioned, but the locality and other details were not given.[143]

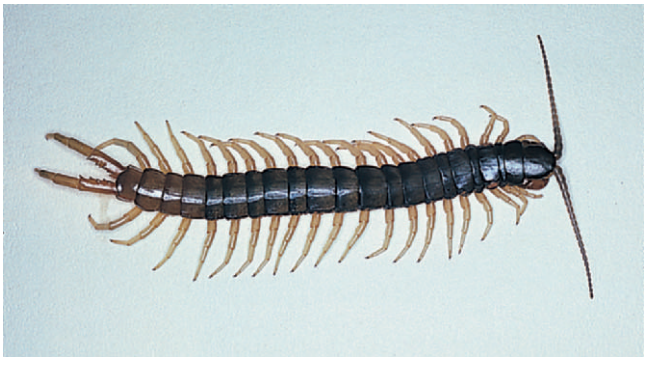


Figure 44-32. Centipede, *Scolopendra heros*.

Figure 44-33. Centipede fangs.

Burning pain, local swelling, erythema, lymphangitis, and lymphadenopathy are common manifestations of a centipede bite. Swelling and tenderness may persist for as long as 3 weeks or may disappear and recur. Superficial necrosis may occur at the site of fang punctures. Few bites with serious systemic reactions have been reported in detail. In one case ascribed to *Scolopendra heros* in the southwestern United States, a woman had massive edema of the leg, necrosis of the peroneal muscles, loss of motor function in the foot, myoglobinuria, and azotemia.[154] An Israeli patient bitten on the neck complained of inability to turn her head, probably because of muscle spasm.[175] Other cases have been characterized by dizziness, nausea, collapse, and pyrexia.[180] An infant who ingested a centipede identified as *Scutigera morpha* developed hypotonia, vomiting, and lethargy, presumably from being bitten in the mouth or pharynx. The child recovered spontaneously after about 48 hours.[17] Centipede envenomation in a newborn inflicted by *Scolopendra gigantean* was reported in Venezuela and caused crying, irritability, and localized wound edema.[225] Acute coronary ischemia with electrocardiographic changes was described after envenomation from an unidentified 12-cm (4.7-inch) centipede in Turkey.[195]

Although some centipede bites may be excruciatingly painful, they are not fatal and seldom require more than supportive care. Treatment of centipede envenomation is symptomatic. Infiltration of the bitten area with lidocaine or another anesthetic promptly relieves pain. Antihistamines and corticosteroids have been suggested for more severe reactions. Tetanus prophylaxis is advisable.[36,176] Hot water immersion has proven beneficial in Australia from bites of centipedes from the genera *Scolopendra, Cormocephalus,* and *Ethmostigmus*.[13]

Millipedes differ from centipedes in having two pairs of legs per body segment and in lacking apparatus for injecting venom. Several large species of the genus *Spirobolus* are common in the southern United States (Fig. 44-34). Some species are broad and short and roll into a ball when disturbed (Fig. 44-35). Millipedes are generally ground dwelling and secretive. Occasionally, they aggregate in enormous numbers. They generally feed on decaying vegetation.

Millipedes are exceptionally well endowed with defensive chemical secretions that include hydrogen cyanide, organic acids, phenol, cresols, benzoquinones, and hydroquinones.

Figure 44-34. *Spirobolus* millipede.

These effectively deter most predators. Some large species can eject secretions for distances up to 80 cm (32 inches).

Human injury from millipede secretions has been reported from a number of tropical regions. The most common injury is dermatitis that begins with a brown-stained area, which burns and may blister and exfoliate.[161] Millipede secretion in the eye causes immediate pain, lacrimation, and blepharospasm. This may be followed by chemosis, periorbital edema, and corneal ulceration. Blindness has been reported.[111,208] Individuals exposed to large millipede aggregations may complain of nausea and irritation of the nose and eyes. No specific treatment is available. Prompt irrigation with water or saline should be followed by analgesics, antimicrobials, and other measures appropriate for superficial chemical burns. Ophthalmologic evaluation is mandatory for eye injuries.

HEMIPTERA (SUCKING BUGS)

Hemiptera is a large order of insects characterized by sucking mouthparts, generally in the form of a beak, and a life cycle with no well-demarcated larval and pupal stages but a gradual transition from the hatchling nymph to adult. Most hemipterans are winged as adults, with the anterior wings generally divided into chitinized and membranous sections. Most feed on plant juices, but several families are predators, and two feed on the blood of humans and other vertebrates.

The assassin bugs (family Reduviidae) are generally recognized by the long and narrow head, a stout and three-jointed beak, long antennae, and typical hemipteran wings (Fig. 44-36). Most are of a dark color; a few are brightly marked or have a checkerboard pattern along the posterior edge of the abdomen. Some species attach fragments of their prey, sand grains, or other debris to their backs. Reduviidae occur on all continents. They have a variety of habitats and are often nocturnal.

The triatomids (e.g., kissing bugs, flying bedbugs, Mexican bedbugs, barberos) are a subfamily of the Reduviidae adapted for feeding on blood. They feed on a wide range of mammals and often live in the nests or burrows of their hosts. Armadillos, dogs, opossums, and pack rats are common hosts in the southern United States and Mexico. Some triatomids adapt readily to life in human dwellings, particularly those of adobe construction. Triatomids are primarily a neotropical group, with species ranging northward in the United States to Utah and southern Indiana. *Triatoma protracta* and *Triatoma sanguisuga* are among the better-known species. The family Cimicidae, or bedbugs, are flat, ovoid, and reddish brown insects whose wings are reduced to a pair of functionless pads. Lack of large terminal claws distinguishes them from lice. Bedbugs are cosmopolitan in distribution. Two species, *Cimex lectularius* and *Cimex hemipterus,* feed primarily on humans and live in dwellings, where they hide in bedding, under wallpaper, behind baseboards, and in window frames. Homes of poorer persons are more likely to be heavily infested, but the insects may be carried into well-kept residences, hospitals, and hotels. Other species of *Cimex,* normally parasitic on bats and swallows, occasionally attack humans.

Venomous aquatic hemipterans include the giant water bugs (family Belostomatidae) (Fig. 44-37), back swimmers (family Notonectidae) (Fig. 44-38A), and water scorpions (family Nepidae) (see Figure 44-38B). Water bugs are distinguished from aquatic beetles by their beak and hemipteran wings; back swimmers have greatly elongated hind legs; and water scorpions have a slender body with long, terminal breathing tubes. These insects are widely distributed in freshwater habitats.

Figure 44-35. Giant Madagascar millipede.

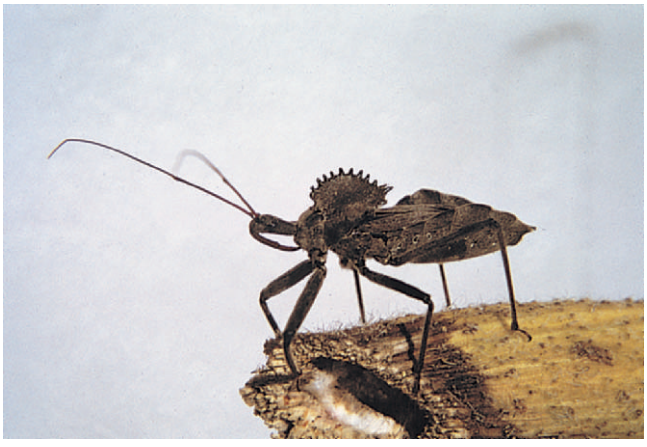


Figure 44-36. Wheel bug, *Arilus cristatus*, a large assassin bug common in the eastern United States.

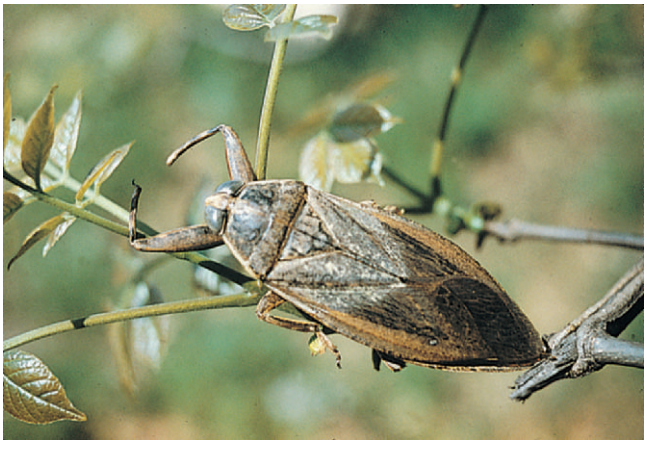


Figure 44-37. Giant water bug, *Benacus griseus*, a large insect common in aquatic habitats in the eastern United States.

Rights were not granted to include this figure in electronic media. Please refer to the printed book.

A

B

Figure 44-38. **A,** Back swimmer bugs have greatly elongated hind legs. **B,** Water scorpions have a slender body with long terminal breathing tubes (**A** photo courtesy Gari Weinraub; **B** from www.mybitoftheplanet.com.)

The hemipteran venom apparatus consists of two or three pairs of glands in the thorax. Secretions are ejected through half of a double tube formed by the interlocking of the elongated maxillae and mandibles, which have distal tips modified for piercing. Few hemipteran venoms have been studied. Venoms of two reduviids, *Platymeris rhadamanthus* of Africa and *Holotrichus innesi* of the Middle East, contain several enzymes and nonenzymatic proteins.[75,285] Sialidase, an enzyme unusual in invertebrates, is found in *Triatoma* venom; it has anticoagulant activity.[6] Venom serves primarily for subjugation and probably digestion of prey, but the insects may defend themselves by biting. Salivary secretions of blood-sucking hemipterans also contain potent allergens.

Clinical Aspects

Triatomids usually bite at night on exposed parts of the body. Feeding may last from 3 to 30 minutes. Bites are painless. On initial exposure there is usually no reaction. Repeated bites are followed by reddish, itching papules that may persist for up to a week. Bites are often grouped in a cluster or line (Fig. 44-39)

Figure 44-39. *Triatomid* feeding pattern.

and may be accompanied by giant urticarial wheals, lymphadenopathy, hemorrhagic bullae, fever, and lymphocytosis. Systemic anaphylactoid reactions with respiratory or gastrointestinal manifestations may occur.[120,158] Entomologists and small children are most frequently bitten by assassin bugs, as handling induces the insect to bite. Bites of several U.S. species, such as the wheel bug (*Arilus cristatus*), black corsair (*Melanolestes picipes*), and masked bedbug hunter (*Reduvius personatus*), are described as being as painful as the sting of a hornet and accompanied by local swelling lasting several hours. Bedbug bites usually raise a pruritic wheal with a central hemorrhagic punctum, followed by a reddish papule that persists for several days. Bullae, generalized urticaria, arthralgia, asthma, and anaphylactic shock are rare sequelae of bedbug bites. Bites by aquatic hemipterans are similar to those of assassin bugs, but few cases have been described in detail.

Treatment and Prevention

Treatment is symptomatic and not particularly effective. Various antipruritic preparations are helpful in mild cases. Topical or intralesional steroids have generally been ineffective. Immobilization, elevation, and local heat are helpful in severe limb bites. Desensitization with triatomid salivary gland extract has been used in a small series of patients with history of life-threatening anaphylactic reactions.[227] Triatomids and bedbugs are more difficult to eradicate with insecticides than many other household insects. Benzene hexachloride has been effective against triatomids in Latin America.

BEETLES AND OTHER INSECTS

Beetles (order Coleoptera) are the largest group of insects, with at least 250,000 species. The prothorax of beetles is generally very distinct, whereas the two posterior thoracic segments are more or less fused to the abdomen (Fig. 44-40). In most beetles, the anterior wings are heavily chitinized, acting as covers for the posterior membranous wings used in flight. Mouthparts are of the chewing type. The life cycle involves larval and pupal stages before emergence of the adult. Many beetles feed on plants throughout their life cycle, many are predators or scavengers, and a few are parasitic. No beetles have a bite or sting venomous to humans, but several families have toxic secretions

Figure 44-40. Beetles (order Coleoptera) represent the largest group of insects.

Figure 44-41. Spanish fly, *Cantharis vesicatoria*. (From www.ujf-grenoble.fr/JAL/imag/insectes/insect2.htm).

that may be deposited on the skin. The blister beetles (family Meloidae) are a cosmopolitan group with numerous representatives in deserts and semiarid regions. A species may suddenly appear by the thousands, especially after rains, persist for a few days, and be replaced by another. The majority are of medium size (about 15 mm, or half an inch) and have soft, leathery forewings (elytra). Some are brilliantly colored. They are plentiful on vegetation, and some species are attracted to lights. A low-molecular-weight toxin, cantharidin, is present in the hemolymph and most of the insect's tissues. It is exuded from multiple sites if the beetle is gently pressed or otherwise disturbed.

In the eastern United States, blister beetle dermatitis is usually caused by *Epicauta* species, which reside on many garden plants. Contact with the beetle is painless and seldom remembered by the victim. Blisters appear 2 to 5 hours after contact and may be single or multiple, usually 5 to 50 mm (0.2 to 2 inches) in diameter and thin walled. Although not naturally found in the United States, the most well known blister beetle is the Spanish fly (*Cantharis vesicatoria catoria*) (Fig. 44-41). Unless broken and rubbed, the blisters are not painful. Cantharidin dermatitis and nephritis have been reported after unusually heavy vesication but more frequently result from using a cantharidin preparation as an aphrodisiac.

Darkling ground beetles are moderately large, dark, and heavily chitinized insects that assume a characteristic posture with head down and tail up when disturbed. They are found worldwide in arid regions, where they live under stones and other cover and crawl about at night. Most species can spray irritant secretions, mostly benzoquinones, from the tip of the abdomen to a distance of 30 to 40 cm (12 to 16 inches). These secretions are generally harmless to humans, but blistering of the skin has been reported, and eye injury is possible.

No special treatment for beetle vesication is available. The injuries are best treated as superficial chemical burns. Local preparations containing corticosteroids or antihistamines are not particularly effective. An outbreak of a blistering disease was reported in a military unit training in the Arizona desert during an unusual heavy rainfall and flooding. Staphylinid (rove) beetles (genus *Paederus*, family Staphylinidae) (Fig. 44-42) were collected at the site. These beetles have been responsible for vesicular dermatitis in other parts of the world but were never reported in the United States before this series.[47]

Internationally, small rove beetles of the genus *Paederus* are troublesome in many tropical and subtropical regions. The whiplash beetle or Finch Hatton bug (*Paederus australis*) caused evacuation of an entire aboriginal community in northern Australia,[265] and several cases of blistering dermatitis were reported in central Queensland.[15] A large suburban hospital in Sri Lanka had 108 cases of painful dermatitis among members of its staff on night duty.[136] Over 200 cases were reported in Turkey, resulting in lesions similar to bullous impetigo or infectious herpes zoster.[268]

Pustular dermatitis epidemics from other staphylinid (rove) beetles related to the paederids have been reported in Nigeria in 1990, Egypt in 1994, Kenya in 1998 after sudden floods,[47] and most recently in Pakistan among deployed U.S. military personnel.[74] These beetles are slender with elongate abdomens and very short rectangular elytra. They frequent damp habitats and may be plentiful in irrigated crop fields. They usually fly after dark in hot, humid weather and are attracted to lights. Their vesicant secretion is an alkaloid present in greatest concentration in hemolymph; it is usually deposited on human skin when the beetles are pressed or crushed, but it may occasionally be spontaneously secreted. There is no immediate reaction to the secretion, but after 12 to 24 hours, painful erythematous lesions develop and soon become vesicular. The vesicles last 3 to 7 days and are followed by crusting and pigmentation. Conjunctivitis results if the secretion is rubbed into the eyes. This condition is known in parts of the world as "Nairobi eye" or "Christmas eye."[47] Some persons with extensive skin involvement may show generalized symptoms. Treatment is symptomatic and not very effective. Prompt soap-and-water washing after insect contact is recommended. Screening of sleeping and working quarters is the best prevention.

Lady beetles (ladybugs, family Coccinellidae) are widely distributed and highly beneficial as predators on aphids and scale insects. Species in the eastern United States often overwinter in

A

B

Figure 44-42. Rove beetles, genus *Paederus*, family Staphylinidae. (**A** from www.folderinsects/rovebeetles.html; **B** from www.kendall-bioresearch.co.uk.)

houses and do no harm. However, an Australian lady beetle, *Diomus notescens*, is reported to bite, causing papules with small necrotic centers.[205] Two cases of human ear invasion by a predaceous beetle, *Crasydactylus punctatus*, or the carabid beetle, have been reported from Oman. One victim had a severe otologic injury caused by biting and chewing on the external auditory canal and tympanic membrane.[21]

Other types of insect envenomation are sporadic. Many insects that normally feed on plant juices occasionally inflict annoying bites. This behavior may be initiated by dehydration

Figure 44-43. Stick insect, *Anisomorpha buprestoides*, ejects a noxious fluid from its thoracic region that deters birds and other predators. (From www.penvalehouse.org.uk.)

of the insect or by unknown factors. Small predatory insects, such as lacewing larvae, anthocorids, and *Sclerodermus* species, occasionally attack humans instead of their normal arthropod prey. Thrips may bite and produce itching macules.[181] A small hemipteran, *Leptodemus minutus*, caused numerous cases of dermatitis in Kuwait.[243] The stick insect, *Anisomorpha buprestoides* (Fig. 44-43), a common species in Florida and adjacent states, ejects a noxious fluid from its thoracic region that deters birds and other predators. According to regional folklore, this fluid can be directed toward human eyes with painful consequences.

DIPTERA (TWO-WINGED FLIES)

Insects of the order Diptera are characterized by one pair of wings. What would have been a second pair is usually modified to form a pair of drumstick-like structures known as halteres. A typical life cycle consists of eggs, limbless larvae, pupae, and winged adults, but numerous variations exist. Mouthparts are of the sucking type. Females of many species, although free living, take blood or other tissue fluids from vertebrates, injecting salivary secretions that are not intrinsically toxic but are potent sensitizing agents for most humans. Larvae of some Diptera are human parasites. Other adult Diptera feed indiscriminantly on feces and human foodstuffs. These habits make them by far the most important arthropod vectors of human disease (Table 44-1).

Most of these insects are cosmopolitan in distribution, except tsetse flies, which are restricted to Africa, and tropical and subtropical sand flies. Some species of mosquitoes and blackflies are adapted to cold temperate, sub-Arctic, and alpine environments, where their numbers may make areas uninhabitable during peak activity. Other mosquitoes and biting midges are equally abundant and annoying in some coastal areas and on islands. Two groups of biting flies and several species involved in human myiasis are largely confined to the tropics (see Table 44-1).

TABLE 44-1. Major Groups of Biting Dipterans

INSECT	RECOGNITION FEATURES OF ADULT	LARVAL AND PUPAL STAGES
Mosquitoes (Culicidae, subfamily Culicinae)	Prominent proboscis; wings with scales; palps of female much shorter than proboscis; usually rests with body parallel to substrate	Aquatic in great variety of habitats; both larval and pupal stages motile
Mosquitoes (subfamily Anophelinae)	Prominent proboscis; wings with scales and often with dark mottling; palps of females about as long as proboscis; usually rests with head down and body held at an angle to substrate	Same as above
Blackflies, buffalo gnats (family Simuliidae)	Stout; humpbacked; short antennae; wings broad, with most veins faint; body length >2.5 mm	Sessile in flowing water; usually attached to rocks and logs, sometimes to crustaceans
Sand flies (family Psychodidae)	Small (usually >2 mm body length); hairy; wings with straight, prominent veins	In damp crevices, animal burrows, leaf litter
Biting midges, sand flies, no-see-ums (family Ceratopogonidae)	Small (>2 mm body length); wings often mottled; most wing veins faint	In mud, wet sand, rotting vegetation; larvae very motile
Horseflies, deerflies (family Tabanidae)	Large (5 to 25 mm body length) with large eyes; usually brilliantly colored; body stout; wings with prominent veins	In mud or shallow water
Stable flies (family Muscidae)	Similar to housefly in size and general appearance; sharp-pointed proboscis projects downward and backward	In decaying vegetable matter or urine-soaked straw
Tsetse flies (family Glossinidae)	Large (6 to 14 mm); proboscis projects forward; wings fold scissorlike over back	Larvae complete most of development in female; pupate in soil a few hours after birth
Snipe flies (family Rhagionidae)	Long legs; relatively slender body; large eyes; wings with prominent veins	Aquatic; in moist soil or rotten wood

Figure 44-44. Tsetse fly, *Glossina* species. (From www.fohn.net/tsetsefly.)

Tsetse flies (*Glossina* species) are of great importance as vectors of human and animal trypanosomiasis in sub-Saharan Africa (Fig. 44-44). Although not closely related to deerflies and other tabanids, they are similar in appearance and habits. Their life history is peculiar in that a single large larva develops in the uterus of the female and is expelled shortly before pupation, which takes place in the soil. Tsetse fly bites produce comparatively little local reaction other than brief pain and itching.

Mosquitoes (see Chapter 42) are characterized by a fringe of setae along the posterior margin of the wings and delicate scales along the wing veins. Only the female feeds on blood. Her prominent beak contains a kit of piercing and sucking tools. Most mosquitoes have body lengths of 3 to 4 mm, but some large species may be about twice this size (see Table 44-1). The estimated 3000 species of mosquitoes are cosmopolitan in distribution.

Carbon dioxide, body heat, and sweat gland secretions, especially apocrine, are attractants for mosquitoes; certain skin lipids are repellent.[128] Children under 1 year of age rarely show a skin reaction to mosquito bites, but by age 5 nearly all are reactors. Both immediate and delayed types of hypersensitivity are induced. Typically, immediate pruritic wheals are followed by red, swollen, and pruritic lesions in 12 to 24 hours. These lesions are associated with both IgE and IgG antibody complexes and a lymphocyte response.[200,216]

All the classic types of immunologic injury have been reported after mosquito bites, including injury from circulating immune complexes, asthma, and Arthus reaction.[97,114,260] Seasonal bullous eruptions in a coastal area of Britain were ascribed to *Aedes detritus*. Most of those affected were women with varicose veins or deep venous thromboses. Intense skin reactions accompanied by fever, lymphadenopathy, and hepatosplenomegaly have been described and are associated with infiltration of skin lesions by natural killer lymphocytes.[166] Nodular skin lesions lasting up to a month have been reported after mosquito bites in patients with acquired immunodeficiency syndrome (AIDS) receiving zidovudine.[69] Papulovesicular lesions with eosinophil infiltration were reported following insect (including mosquito) bites in patients with lymphocytic leukemia.[49] Among 21 Japanese patients with severe local and

constitutional reactions to mosquito bites, seven died of malignant histiocytosis before age 28. Nine others retained hypersensitivity; three lost the reaction.[114]

Treatment of mosquito bites consists of local application of antipruritic lotions or creams. Antihistamines relieve the itching of early lesions but have no effect on later ones.[215] Group I corticosteroid creams and ointments may be helpful. Desensitization with insect whole-body extract is difficult but occasionally successful.[163] Prolonged heavy exposure to mosquito bites causes some individuals to lose sensitivity, occasionally in less than 1 year. Delayed hypersensitivity is lost more readily than immediate hypersensitivity, with decreases in both IgE and IgG.[201]

Biting Midges (Culicoides)

Biting midges are very small flies that have a bite out of proportion to their size. Only females feed on blood. The wormlike aquatic larvae usually develop in water-saturated soil; mangrove swamps are a common habitat. Larvae of some species use axils of banana and similar plants. The genus is cosmopolitan but presents the greatest problem in subtropical and tropical coastal regions. Activity is often seasonal. The flies bite most intensely in still air and reduced light.

Bites are immediately painful and result in raised, red, and pruritic lesions that persist from a few hours to a week or more. Some victims develop vesicles, pustules, and superficial ulcers, particularly if bitten by the genus *Leptoconops*. Hypersensitivity is involved, although some persons seem to develop intense reactions on first exposure to the insects.

Treatment of bites is symptomatic and similar to that for mosquito bites. Artificial hyposensitization has not been successful; however, spontaneous decrease in skin reactivity may occur in some individuals.

Blackflies (Simuliidae)

Blackflies are small, stocky flies that have a characteristic humpbacked appearance. Adults prefer open and sunny areas and are good fliers. Not all species are anthropophilic. The sessile larvae and pupae are found in flowing water, from large rivers to small brooks. Blackflies are cosmopolitan, but their abundance and medical significance vary widely. They range well into the Arctic and constitute a major problem for both humans and domestic animals in parts of Europe, Canada, and the northern United States. In the tropics they tend to be more localized, often remaining close to streams.

Blackflies bite more commonly on the upper half of the body. They snip the skin and suck the pooled blood, leaving relatively large punctures that may bleed, a symptom rarely seen with bites of other small flies. The local pain, swelling, and redness that follow blackfly bites are unusually intense and persistent. Vesicles and weeping, crusted lesions may last for weeks. Systemic symptoms such as malaise, fever, and leukocytosis may occur. Enlarged indurated lymphatics, particularly in the posterior cervical region, are common in Canadian children living where blackflies are abundant. Blackfly bites can infrequently cause severe hypersensitivity reactions and localized cellulitis, similar to that seen with Hymenoptera stings.[191] Hemorrhagic symptoms have been reported in Brazil. Generalized urticaria, bleeding, angioedema, cough, wheezing, toxemia, and even death may occur.[121,141,281]

No specific treatment for blackfly bites is available. Hyposensitization has been attempted with little success. Neither repellents nor ordinary clothes provide satisfactory protection against blackflies when they are present in large numbers. Avoidance of heavily infested areas during fly season is often the most practical solution. Control measures have not proved highly effective.

Blackfly population control in Africa has evolved over the years. In the 1960s, DDT was utilized. In the 1970s and 1980s, water flow manipulation was instituted for better environmental preservation. In the 1990s and currently in Africa, the organophosphate temephos and toxins produced by the bacterium *Bacillus thuringiensis* have been tested and shown to be effective.[184]

Horseflies and Deerflies (Tabanidae)

Horseflies and deerflies are medium to large (10 to 25 mm [0.4 to 1.0 inch] body length) stocky flies whose large eyes often are brightly colored. They are strong fliers and prefer open and sunny habitats. The tabanids attack a variety of large mammals, including humans. The predacious maggotlike larvae live in water-soaked soil or shallow water. Bites from these large flies, especially the deerfly (*Chrysops* species), are painful and may cause both external and subcutaneous bleeding. An itching wheal up to 1 inch in diameter develops but usually does not last long. In some victims, severe and prolonged swelling of the face or an extremity develops. About 30 cases of systemic anaphylactoid reactions have been reported. One man with a history of systemic reactions to wasp stings had a similar reaction to a horsefly bite.[95,113]

As with other fly bites, treatment is symptomatic. Hyposensitization has been attempted in a few cases, with some success.

Other Biting Diptera

Sand flies (*Lutzomyia vexator*) are very small biting flies and the vectors for leishmaniasis, a protozoal disease of humans and dogs in regions of Asia, Africa, and South America. Recently, cases of visceral leishmaniasis have been reported in upstate New York with outbreaks in dog kennels.[193] Leishmaniasis has also been described in Washington County, Texas.[166] Sand flies are widely distributed in tropical and subtropical regions. They live in damp, shaded places such as mammal burrows, rock crevices, and cracks in walls of houses and other structures. Favorite habitats in Central America are gambas, which are deep clefts between the buttresses of large forest trees. Larval and pupal stages are found in moist detritus in holes and crevices. The adults usually emerge at night when air is still and temperatures are above 13° C (56° F). They are poor fliers. Sand fly human biting activity is typically highest between midnight and 3 AM.[233] In addition to leishmaniasis, these flies are vectors of *Bartonella bacilliformis* in Peru and Brazilian Amazonia.[58,189] Sand fly populations can be controlled with pyrethroid insecticide sprayed into the mounds and burrows. Spraying of a 100-m (109-yd)-wide barrier zone around a campsite can reduce sand fly numbers for an extended period.[76] Other preventive measures include impregnated bed netting and clothing, specialized dog collars, and spraying of residential dwellings and animal shelters.[160] A recent source describes vaccine development for prevention of sand fly fever.[67]

Snipe flies (Rhagionidae) prey primarily on insects, but some species, such as *Symphoromyia,* feed on the blood of mammals. Their habits and life history are similar to those of tabanids. Reactions to snipe fly bites range from pain to anaphylaxis. A person who reacts severely may be bedridden for days.

Figure 44-45. Stable fly, *Stomoxys calcitrans.* (From the University of Nebraska Department of Entomology, www.entomology.unl.edu.)

Figure 44-46. *Dermatobia* larva.

The stable fly (*Stomoxys calcitrans*) (Fig. 44-45) is related to the housefly, which it closely resembles. It is plentiful throughout most of the United States, particularly in agricultural districts. Eggs are deposited in piles of decaying vegetation, where the larvae develop. Thunderstorms seem to stimulate fly activity, which accounts for the widespread belief that houseflies bite just before a storm. Bites cause a sharp, stinging sensation, but dermal lesions are uncommon. Itching is transient.

Louse flies (Hippoboscidae) are peculiar Diptera that may lack wings entirely or have them for only part of their adult life. The wingless forms are flat, leathery insects that resemble lice or ticks. They are ectoparasites of birds and mammals. Larvae are carried in the uterus until development is almost complete; the pupal stage may be spent in the soil or on the host. The sheep ked (*Melophagus ovinus*) is a common species in the United States and sometimes bites sheep shearers and handlers. The related deer ked (*Lipoptena cervi*) is a seasonal pest in wooded sections of northern Europe, causing hundreds of cases of dermatitis annually. The pigeon fly (*Pseudolynchia canariensis*) is an avian parasite that sometimes infests buildings and bites the occupants.

Lesions from hippoboscid bites appear 1 to 24 hours after the bites as reddish itching papules that may persist for up to 3 months. Topical corticosteroids may afford symptomatic relief and hasten resolution of the lesions. Repellents are reported to be ineffective against these insects.

Myiasis

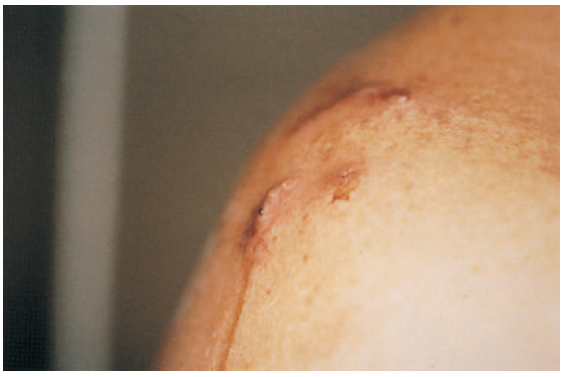
The term *myiasis* for parasitism by fly larvae was introduced into the medical literature in 1840, although the condition has been observed since antiquity. More than a hundred species of Diptera have been reported to cause human myiasis.[173] Some are obligate parasites, for which humans are one of several hosts; some are opportunistic invaders that find parasitism an alternative to feeding on decaying tissue or its products. Nevertheless, humans are not a particularly good host for most species of fly larvae, and many infections terminate prematurely. Sensitization of host tissues to fly larvae does not occur as readily as with many other arthropod and helminth parasites.

Myiasis may be classified by clinical manifestations or etiologic agents. This chapter discusses only dermal and wound myiasis. Myiases that involve primarily the gastrointestinal tract, urinary tract, eye, and nasopharynx are not covered.

Furuncular Myiasis. In furuncular myiasis, the fly larva penetrates the skin but remains sedentary, producing a boil-like lesion that usually has a central opening. Here the larva completes its development but typically emerges to pupate outside the host. As a human problem, this form of myiasis is largely confined to the tropics, although imported cases are being recognized in increasing numbers in other regions of the world, particularly in North Americans who travel to more southern tropical regions.[159] A German travel clinic reported 13 cases in travelers returning from tropical countries during a 3-year period.[123] Tourists and divers recreating in tropical regions of Mexico, South and Central America, and Trinidad are also at risk.[14]

The classic agent of furuncular myiasis is the so-called human botfly, *Dermatobia hominis* (Fig. 44-46). Humans, however, are only one of many mammals that serve as suitable hosts for the obligately parasitic larvae of this fly. It is widely distributed in the American tropics and is an important parasite of domestic cattle in many places. The adult fly resembles a bumblebee (body length, about 15 mm [0.6 inch]). It does not feed and is infrequently seen. The life cycle of this fly is unique in that the female attaches her eggs to the body of another arthropod for transfer to the host. Large mosquitoes of the genus *Psorophora* are often used (8% were found bearing *Dermatobia* eggs in one study), but about 40 other species of insects and ticks have been reported to be egg carriers. When the carrier alights on a mammal, the eggs hatch immediately, and the larvae enter the skin through the bite of the carrier or some other small trauma. Small larvae are fusiform and later become pyriform to ovoid as they reach full development at lengths of 15 to 20 mm (0.6 to 0.8 inch). They are encircled by several rings of spines. The larval stage lasts 6 to 7 weeks, after which the larva emerges from the skin and drops to the ground, where pupation occurs.

Infection is fairly common among rural people of Central America. Cases in returned tourists and visitors from Latin America have been diagnosed in many parts of the United States. Six cases occurred in one group of tourists visiting archaeologic sites in Guatemala.[70,124,138,196,261] Lesions may be on



A

B

C

Figure 44-47. **A,** Lateral view of three lesions caused by infestation with *Dermatobia hominis* larvae. The nodules were initially assumed to be furunculosis. A central breathing aperture is present in each nodule. Serosanguineous fluid is draining from two of the nodules. Larval spiracles are visible emerging from the uppermost nodule. **B,** The fatty portions of many strips of raw bacon were placed over the larval apertures to obstruct the air supply and encourage the larvae's egress from the skin. **C,** After approximately 2 hours of treatment with bacon therapy, the *D. hominis* larvae have emerged far enough from the subcutaneous tissues to be grasped with forceps. The larva is removed intact. (From Brewer TF, Wilson ME, Gonzalez E, Felsenstein D: JAMA 270:2087–2088, 1993.)

any part of the body exposed to insect bites and may be single or multiple. An initial pruritic papule becomes a furuncle, with a characteristic opening from which serosanguineous fluid exudes. Pain often accompanies movements of the older larvae, but the lesion is not particularly tender to palpation. Lymphadenopathy, fever, and secondary infection are rare.

This form of myiasis should be suspected in patients with furuncular lesions and a history of residence or travel in endemic areas. It must be differentiated from leishmaniasis and onchocerciasis, which have a different prognosis and treatment. The sensation of movement within the lesion, accompanied by pain but little tenderness or inflammation, suggests myiasis. The tip of the larva may protrude from the central opening, or bubbles produced by its respiration may be seen. Often, simple pressure will extrude the organism, particularly if it is small.[190] Occlusion of the breathing hole may cause the larva to emerge sufficiently for it to be grasped and withdrawn. An effective folk remedy is binding a piece of fat, such as bacon, over the opening (Fig. 44-47).[27] This often causes the larva to leave its burrow. Another technique is injection of about 2 mL of a local anesthetic into the base of the lesion, thus extruding the larva by fluid pressure.

If these methods are unsuccessful, surgical excision under local anesthesia is indicated. Whatever method is used, care should be taken not to break or rupture the larva. This may cause a strong inflammatory reaction, often followed by secondary infection. Repeated infections tend to confer some immunity that may abort larval development. Screens, protective clothing, and use of insect repellents are helpful in preventing infestation.

Furuncular myiasis in tropical Africa is caused by the tumbu fly, *Cordylobia anthropophaga* (Fig. 44-48). The larval stage of this fly is an obligate parasite of many mammals, but rats and dogs are the most important epidemiologically. The adult is about the size of a housefly, but stockier. It prefers shade and is most active in early morning and afternoon. Females lay eggs on dry sandy soil or on clothing. They are attracted by the odor of urine. The eggs hatch in 1 to 3 days, and hatchling larvae can survive up to 2 weeks waiting for contact with skin of a suitable host. They can penetrate unbroken skin. They become fusiform to ovoid and reach a length of 13 to 15 mm (0.5 to 0.6 inches). The larval stage is completed in 9 to 14 days.

Human infections occur in most nations of sub-Saharan Africa. Transmission increases during the rainy season. Among indigenous peoples, infection is most frequent in children, as adults evidently acquire some immunity. Infections among Americans and Europeans visiting Africa are reported regularly.[122,123,235] Lesions may be on any part of the body but are more common on the legs and buttocks. The furuncles are discrete, elevated, and nontender and have a central opening. The number of lesions, up to about 50, is greater than with *Dermatobia*. Infections in children have been mistaken for chickenpox,[188] but the course of the infection is much shorter. An exceptionally heavy infection (about 150 larvae) was caused by *Cordylobia rodhaini,* normally a parasite of forest mammals. It was accompanied by lymphadenopathy, leukocytosis, and elevated IgA. Clothing left to dry on the ground was the presumed source of the parasites.[197] Principles of diagnosis and treatment are much the same as for *Dermatobia*. Avoidance of skin contact with potentially contaminated soil and ironing of cloth-

Figure 44-48. Furuncular myiasis. (From the University of Erlangen, Department of Dermatology; accessed at www.dermis.net).

ing and bedding after open-air drying are preventive measures that often are difficult to achieve.

Hematophagous Myiasis. The sole cause of hematophagous myiasis is the Congo floor maggot, *Auchmeromyia luteola*. It is dark, distinctly segmented, ovoid, and 15 to 18 mm long, and it assumes the larval stage of a moderate-size yellowish fly. It is widely distributed in sub-Saharan Africa and is essentially a human parasite. It is unique among parasitic fly larvae in living apart from its host in the earthen floor of native dwellings. It seeks persons lying or sitting on the floor or on mats and feeds intermittently on blood, usually at night. The bites are trivial but may interfere with sleep. Sensitization appears to be uncommon.

Autochthonous furuncular myiasis may occur in the United States, usually in children. Most cases are caused by larvae of botflies of the genus *Cuterebra,* whose normal hosts are small rodents and rabbits. The fly eggs are attached to low vegetation and hatch on contact with skin of the host. Adult human skin seems impervious to them, but that of children may be penetrated. There is usually a history of outdoor play in weeds or grass or with a pet rabbit. In one case, eggs were apparently deposited directly on the skin.[99] Lesions typically develop on the head, neck, or chest in 1 to 2 weeks. Once recognized, the larvae can often be removed by simple pressure.[209,248] The syndrome may also be caused by larvae of *Wohlfahrtia vigil,* a large fly native to Canada and the northern United States. Its normal hosts are newborn mammals, particularly mink, dog, and fox. The fly deposits larvae on the skin, which penetrate in about an hour. Human infections are typically in infants under 9 months, and the furuncular lesions are usually on the face. Fever, irritability, and loss of appetite are common. Larvae can usually be expressed from the lesion; surgery rarely is necessary. Netting over the crib or pram when outdoors usually affords protection.

Migratory Myiasis. One type of migratory myiasis is caused by flies of the genus *Hypoderma*. Adult flies are large and hairy, resembling bumblebees. Normal hosts for the parasitic larvae are cattle, deer, and horses. The flies attach their eggs to hairs. Hatchling larvae penetrate the skin and wander extensively through the subcutaneous tissues, eventually locating under the skin of the back, where they produce furuncular lesions. The condition has veterinary importance. Humans are abnormal hosts in which the parasite is unable to complete its development. Human infections usually occur in rural areas where cattle and horses are raised and are more common in winter. Larvae migrate rapidly (as much as 1 cm/hr) and erratically through subcutaneous tissues, producing intermittent, painful swellings over months. The person often senses larval movement. Larvae respond negatively to gravity, so the last lesions are usually on the head or shoulders. Eosinophilia (up to 35% eosinophils on white blood cell differential) and angioedema may be seen. Larvae may emerge spontaneously from furuncles or may die in the tissues. In rare cases, larvae were seen to have invaded the pharyngeal region, orbit, and spinal canal.

Another form of migratory myiasis is caused by larvae of *Gastrophilus,* which normally are gastrointestinal or nasal parasites of horses (Fig. 44-49). In human infections, which are reported more frequently from Europe than from the United States, the young larvae burrow in the skin, producing narrow, tortuous, reddish, and linear lesions with intense itching. Lesions usually advance 1.5 cm/day, but more rapid progress has been reported. Death of the larvae terminates the infection in 1 to 2 weeks without sequelae. This infection is clinically similar to creeping eruption, an invasion of the skin by larvae of the hookworms *Ancylostoma braziliense* and *Ancylostoma caninum*. The helminthic parasitosis occurs more often in warm, moist regions, including the southern United States, and is associated with dogs and cats. The myiasis is seen more

Figure 44-49. Larvae of the botfly *Gastrophilus haemorrhoidalis* from a horse's stomach.

frequently in cooler regions and is associated with horses. Definitive diagnosis can be made only by removal of the parasite from its burrow and microscopic examination.

Removal of the larvae by surgery or expression is the usual treatment for migratory myiasis, although local freezing of cutaneous burrows is sometimes successful. Ivermectin given to a patient with *Hypoderma* myiasis resulted in expulsion of the larva.[123] The most effective prevention is control of the infections in domestic animals.

Wound Myiasis. About 85% to 90% of cases of wound myiasis are caused by larvae of flies of the family Calliphoridae, which includes both obligate parasites and opportunists. The most dangerous type of myiasis may be caused by larvae of the screw-worm flies—*Cochliomyia americana* (formerly *Callitroga hominivorax*) (Fig. 44-50A) in the Americas and *Chrysomyia bessiana* (see Figure 44-50B) in Asia and Africa. The adults are rather stocky flies, 8 to 12 mm (0.3–0.5 inches) in body length, and metallic blue-green to purplish black.

The parasitic larvae are pinkish, fusiform (providing the common name), and strongly segmented. Length at maturity is 12 to 15 mm. They are obligate parasites whose chief hosts are cattle, sheep, and goats. They are a major cause of economic loss among livestock. Enzootic areas are mostly in the tropics and subtropics; in the past, summer infections have occurred as far north as Colorado and Nebraska. Female flies deposit eggs near any break in the skin or around the nose, mouth, or ears if a discharge is present. Larvae invade healthy tissue, often causing considerable damage. The larval stage lasts 4 to 8 days, and the entire cycle is 15 to 20 days in enzootic areas. Screw-worm has been eradicated in the southern United States and some other areas primarily through the release of large numbers of laboratory-bred male flies sterilized by gamma irradiation. Females mate only once, so mating with a sterile male nullifies the female's reproductive effort, greatly reducing the population.

Flies may be dispersed by prevailing winds. Infection is often acquired while resting outside during the day or may result from trauma.[167] Lesions may appear on any exposed part of the body. Those on the scalp may be associated with pediculosis capitis (Fig. 44-51). Typical dermal lesions are ulcers or sinuses that may contain up to 200 larvae. These are surrounded by a zone of induration (Fig. 44-52). Pain is variable. Secondary bacterial

A

B

Figure 44-50. **A,** American (New World) screw-worm fly, *Cochliomyia*. **B,** Asian-African (Old World) screw-worm fly, *Chrysomyia*.

infection is common. Tissue destruction may be extensive and mortality is associated with nasopharyngeal invasion.

Irrigation of the wound and mechanical removal of larvae are generally sufficient for treatment. Topical application of 5% chloroform in olive oil followed by irrigation and manual removal of larvae is often adequate in dermal infections. Deeper nasopharyngeal and orbital infections require surgery. Antimicrobial therapy as dictated by culture and sensitivity tests is often necessary. The most effective prevention is elimination of the disease in domestic animals.

Opportunistic invasion of wounds by fly larvae is often seen during war and natural disasters, when injured persons are exposed to flies and medical facilities are inadequate to cope with the emergency. Wound myiasis may also be seen sporadically in nursing homes and hospitals and often is not reported for cultural and medicolegal reasons.[33] Six of 14 cases in one series were acquired in the hospital. Eleven patients were over 63 years of age, and nearly all had underlying problems, such as diabetes or peripheral vascular disease. Most of the infected lesions were on the feet or ankles.[156] In another series of 16 cases, most victims were debilitated and over age 65. Men were

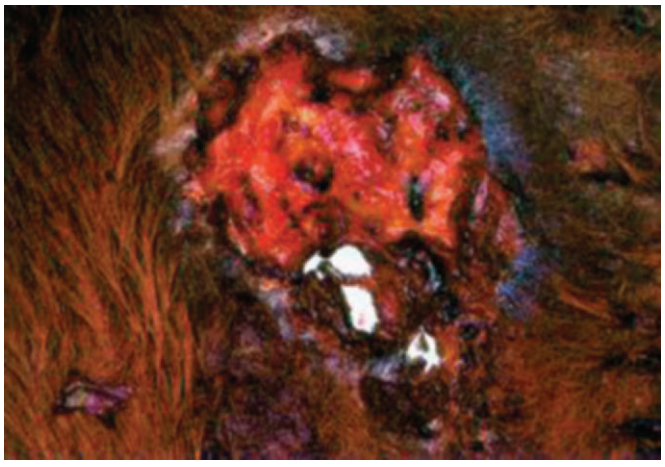
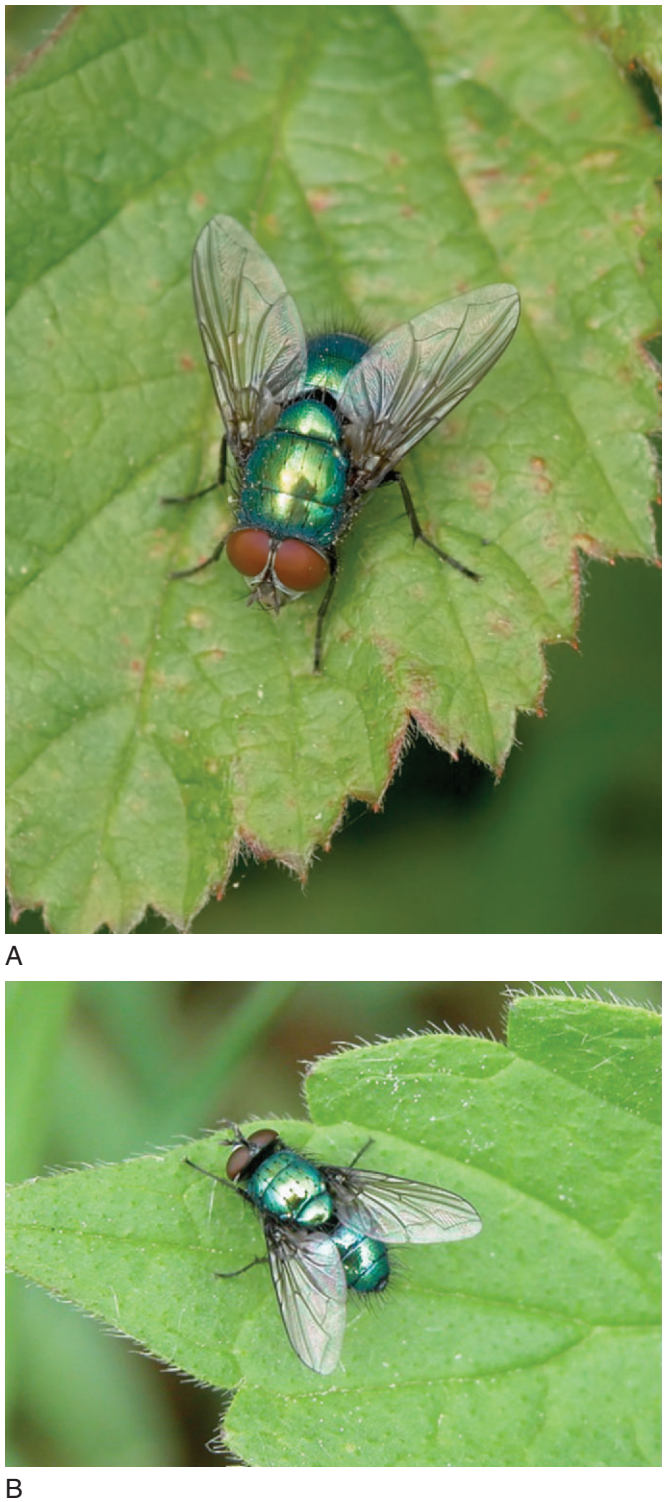


Figure 44-51. Scalp myiasis appearing as pediculosis capitis. (From www.spc.int/rash/munual/images.)

Figure 44-52. Wound myiasis. (From www.icb.usp.br.)

affected more often than women. Seven species of flies were involved.[171] Fifty *Lucilia* larvae were removed from the nose, mouth, paranasal sinuses, and enucleated eye socket of a hospitalized patient left in a room with an open window.[55] The most common fly species involved are *Lucilia* (green-bottle flies) (Fig. 44-53), *Calliphora* species (blue-bottle flies), *Phormia regina* (black blowfly), *Sarcophaga haemorrhoidalis* (flesh fly), and *Musca domestica* (housefly). The flies, whose larvae normally feed on decaying animal tissues, often deposit eggs or larvae in wounds or around body orifices if a malodorous discharge is present. The larvae feed on necrotic tissue, and damage to healthy tissues and secondary infection are uncommon. They actually may debride wounds, and "maggot therapy" with aseptically bred larvae was briefly used in the 1930s. More recently,



A

B

Figure 44-53. Green-bottle fly, *Lucilia*. (**A** from www.bollocks-designs.co.uk; **B** from www.senuah.dk/fly.)

laboratory-bred fly larvae were used to debride venous stasis ulcers and other superficial necrotic areas when antibiotic therapy and surgical debridement were unsuccessful.[247,258] Maggots serendipitously present have been used to debride lesions,[210] but this can be risky.[172] Diagnosis is usually obvious on inspection of the wound. Species identification often requires the larvae to reach maturity. If this is not feasible, examination of the spiracular plates on the last segment of the larva and the chitinized oral structures usually permits adequate identification.

LICE (ORDER ANOPLURA)

Species, Life Cycle, and Distribution

Lice are small wingless insects that are ectoparasites of mammals. They are mostly host specific, and two species are human parasites: *Pthirus pubis* (pubic louse) and *Pediculus humanus*, with two varieties, *Pediculus humanus capitis* (head louse) and *Pediculus humanus corporis* (body louse).[134] They are obligatory parasites, subsisting on blood from the host, and have mouthparts modified for piercing and sucking. The mouthparts are drawn into the head of the louse when not in use.

The number of head lice infestations occurring annually in the United States is estimated at 6 to 12 million.[278] The adult head louse is about 2 to 4 mm long with an elongated body that is flattened dorsoventrally (Fig. 44-54).[224] The head is only slightly narrower than the thorax. The three pairs of legs are about equal in length and possess delicate hooks at the distal extremities. The entire life is spent on the host's body. The eggs (nits) are deposited on hair shafts, generally one nit to a shaft (Fig. 44-55). The nits hatch in about 1 week, and the freshly hatched larvae, which must feed within 24 hours of hatching or die, mature in about 15 to 16 days. The adult female lives for approximately 1 month and may deposit more than 100 eggs during her reproductive life. Body lice are slightly larger than head lice but are similar in appearance with a similar life cycle, although the nits are deposited on fibers of clothing. Head lice and body lice interbreed.

Adult pubic lice are about 1 to 2 mm long, the head is much smaller than the thorax, and the broadly oval body is flattened dorsoventrally (Fig. 44-56). The anterior legs are much shorter than the second and third pairs, and the insect resembles a miniature crab. Nits are deposited on hair shafts, often several per shaft, and the egg-to-egg life cycle is approximately 1 month.

Lice are found wherever people are found. Able to exist only briefly away from the human body, lice are spread by close personal contact and by sharing of clothing and bedding. The various species not only have a particular host but often prefer a particular part of the host's body, so generalizing about transmission of the three varieties that parasitize humans is impossible. During biting and feeding, secretions from the louse cause a small red macule. Severe pruritus and marked inflammatory responses to bites are caused by the sensitization that occurs after repeated exposure to bites. Thus, a victim may have lice for weeks before pruritus becomes marked. Not all people are equally attractive to lice, possibly because of differences in odor and chemical composition of sweat. Lice are medically important as vectors of systemic illnesses, as well as for dermatitis and discomfort.

A

B

Figure 44-54. **A,** Full-grown human head-louse, *Pediculus humanus capitis*. **B,** Adult louse embedded in scalp skin. (Adapted from Roberts RJ: N Engl J Med 346:1646–1650, 2002.)

Clinical Aspects

The head louse localizes on the scalp and rarely on other hairy areas of the body. Children are most frequently affected, but adults, particularly women, may also be affected. Lice are particularly common in young girls, possibly because of their long hair. Infestation is uncommon in blacks, at least in the United States, probably because the shaft of African hair has an oval cross section that makes it difficult for the louse to grip while depositing eggs. However, pediculosis capitis is found in Africa, where the indigenous head louse is adapted to grip the oval hair shafts. Because nits initially attach to the hair shaft close to the skin and are carried higher as the hair grows, the presence of nits near the tips indicates a longstanding infestation.

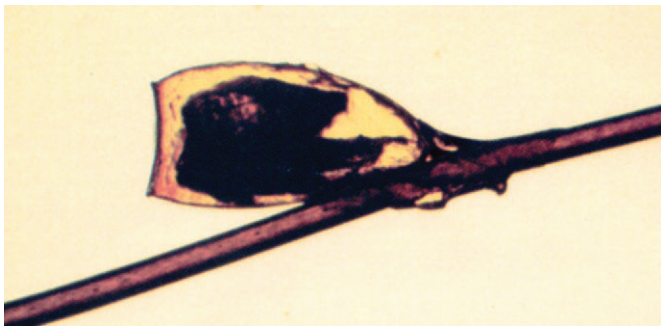


Figure 44-55. Unhatched nit of the head louse, *Pediculus capitis*. (Adapted from Roberts RJ: N Engl J Med 346:1646–1650, 2002.)

Figure 44-56. Pubic or crab louse, *Phthirus pubis*, grasping a hair.

Itching is the principal symptom, and physical findings vary with duration and extent of the infestation, cleanliness, excoriations, and degree of secondary infection. Diagnosis is established by identifying nits and lice. It is not always easy to find lice, especially in early and mild cases, when they may be few in number. Lice are very active, but nits are always present and easy to identify. Nits are whitish ovals, about 0.5 mm long, and attached firmly to one side of the hair. Flakes of dandruff, which resemble nits superficially, are not attached to the hair shafts. Occipital and posterior cervical adenopathy is common and may be present even in less severe cases. A pruritic scalp accompanied by adenopathy should prompt a thorough search for lice and nits. In severe cases, oozing and crusting may be present, sometimes with matting of the hair, and lice may be numerous.

The body louse lives chiefly in the seams of clothing and is rarely seen on the skin. These lice leave clothing to feed on the skin or remain attached to the clothing while feeding, and thus they are most abundant where clothing abuts the skin (e.g., at the beltline). The bite results in a small red macule with a characteristic central hemorrhagic punctum. Excoriations, crusts, eczematization, and other secondary lesions generally obscure the primary lesions by the time the victim seeks medical attention. Shoulders, trunk, and buttocks are favorite sites for bites, and parallel scratch marks on the shoulders are a common finding. The diagnosis is confirmed by identifying parasites or nits from the clothing. Bands of trousers, side seams, and underarm seams are sites of preference. Untreated cases may persist indefinitely, and massive infestations are sometimes seen in vagabonds who have no ready access to frequent laundering or change of clothing or who cannot bathe regularly.

Pediculosis pubis is usually acquired during sexual activity, and it may be a marker to search for other sexually transmitted diseases.[134] It may result from unchanged bedding or nonsexual activity, either from lice that live briefly away from the human body or from egg-infested pubic hairs that are shed. The lice localize principally in the pubic hair, but they are found occasionally in eyebrows, eyelashes, and axillary hairs. Adult pubic lice are not active and hug the skin at the base of the hair shafts, with their heads buried in the follicular orifice. They are not easy to find, but one or more can usually be found if suspicion of the diagnosis is strong enough to prompt a thorough search. A loupe is helpful. Nits are more easily found. Primary bite lesions are almost never seen, but the intense pruritus and pubic scratching are pathognomonic. The secondary infection, crusting, oozing, excoriations, and eczematization that often accompany head and body lice are rarely seen with pubic lice. Peculiar steel-gray macules (maculae caeruleae) may appear in association with some cases of pubic lice. These lesions do not appear until the infestation has been present for several weeks and are most common on the trunk and thighs.

Treatment and Prevention

Treatment of all types of lice strives to eradicate lice and nits and prevent re-infestation. Head lice may be treated with one application of 1% permethrin cream rinse (Nix) or 0.5% malathion lotion (Ovide).[168,278] Hair should be washed, rinsed, and dried, and the rinse is applied for 10 to 20 minutes before being washed off. A fine-toothed comb may be used to remove nits after rinsing. Failure to remove all the lice is a frequent cause of treatment failure. Combing should be repeated in 1 to 2 days to confirm treatment success. If lice are found after 7 days, retreatment is indicated. Household members and those in close contact with the patient should be screened for head lice and treated simultaneously if indicated. Extensive environmental cleanup is probably not warranted, although combs and brushes should be discarded or thoroughly washed with hot water.[224] Hats and scarves should be machine washed in a hot cycle and bed clothing dry-cleaned. A child can return to school immediately after completion of the first proper application of an effective insecticide regardless of the presence of nits. This will avoid unnecessarily missing multiple days of school (and work days for the parents) as well as relieving public overreaction and hysteria.[96,224,234] Regular weekly detection combing is recommended for up to 7 weeks after the first cure.

Other pediculicides are available if the lice are resistant. Two products, RID (Pfizer, New York) and Triple X (Young's Drug Product, Weatherfield, Conn) contain 0.3% pyrethrins and 3.0% piperonyl butoxide. One application of either preparation usually eradicates both lice and nits. A few persons may require another application 7 days after the initial treatment.[22] Alcoholic formulations or vehicles of these products are flammable until dry. Lindane 1% (hexachlorocyclohexane, Kwell) may be used on persons who fail to respond or who are intolerant of permethrin. Kwell, which was formerly the medication most often used in the United States for treatment of louse infestations, is no longer recommended in children because of its safety profile, and it has been pulled off the shelves in many states.[86] Lindane penetrates human skin and has potential for central nervous system toxicity manifesting as seizure activity. It is contraindicated in children and must be used with strict

guidelines in older patients resistant to other treatment measures.[278] Malathion (0.5% Ovide lotion) is an organophosphate and one of the only products that has retained its efficacy and safety profile over time. It is usually effective after one 20-minute application.[168,278] However, this insecticide preparation is not recommended in children less than 2 years of age.[224] Alternative treatments contain pyrethrums and piperonyl butoxide as active ingredients. Newer dry-on, suffocation-based pediculicide lotions are now being introduced that accomplish their mission without neurotoxins, the need for nit removal, or extensive household cleaning. The results are comparable or superior to the efficacy of other pediculocides.[198] Some sources warn that treatment failures may not result from improper application, noncompliance, or re-infestation, but from increasing drug resistance, particularly in underdeveloped countries.[71,78] Ivermectin, a macrocyclic lactone antibiotic highly effective against filarial worms, *Strongyloides*, and scabies (see later), has recently been demonstrated to be effective against lice both topically and orally, but it is not currently approved for this use.[34]

Body lice may be treated with the same medications, but parasites and nits are not generally found on the skin. Eradication of these from the clothing is the primary objective. Treatment includes bathing, laundering all clothing, and changing to fresh clothes free of lice and nits. Dry cleaning eradicates lice and nits, as does ordinary laundering at hot settings. Malathion preparations and γ-benzene hexachloride formulations may be used for mass delousing.

Pubic lice may be treated with the same medications used for head lice. Treatment consists of permethrin 1% cream rinse applied for 10 minutes and rinsed. Sexual partners should also be treated.[87] Crotamiton lotion rubbed into the affected area daily for several weeks to destroy hatching ova may also be used. Eyelash infestations may be managed with physostigmine ophthalmic ointment using a cotton-tipped applicator. Machine washing and drying of sheets and clothing at hot settings will kill lice and nits.

FLEAS (ORDER SIPHONAPTERA)

Species, Life Cycle, and Distribution

Fleas are small ectoparasites of mammals and birds. The wingless body, which is covered by a hard shiny integument, is compressed laterally, enabling the fleas to scurry easily among hairs and feathers of the hosts. They are active insects with legs adapted for jumping, capable of prodigious leaps. Adult fleas subsist on blood. Some species must obtain blood from one particular host, others are less host specific, and all have mouthparts adapted for piercing and sucking. The eggs are laid on or near the host and drop to the ground as the host moves about or shakes. They hatch into small wormlike larvae that feed on droppings from adult fleas, flakes of dried blood from the host, and other organic matter. The life cycle varies among species and may vary considerably within the same species, because each developmental stage is influenced by prevailing temperature and humidity. The customary larval stage of 9 to 15 days may be prolonged for months by adverse conditions, and the pupal stage varies from a week to nearly a year. Individual adult fleas may live for years when circumstances are favorable and can live for months without feeding.

Fleas exist universally, although the distribution of various species is restricted by climate and host. They are of medical importance because of the discomfort resulting from their bites, as a cause of papular urticaria, and as vectors of disease. They are more active in warm weather and cause more problems in warmer climates with a longer breeding season, such as the southwestern United States. They are a particular nuisance in California. High standards of sanitation and personal hygiene in developed countries have discouraged the human flea, *Pulex irritans,* while at the same time, the popularity of household pets has been conducive to the proliferation of dog and cat fleas, *Ctenocephalides canis* and *Ctenocephalides felis*. The incidence of other species on mammals and birds remains high. Because dog, cat, and many other fleas are only partially host specific, the fleas associated with many mammals and birds cause disease in humans. Most current flea-bite problems are caused by animal fleas. Hungry fleas are more often attracted to people from an area frequented by an animal than from the animal itself. If the family dog is absent, hordes of hungry fleas may persist for months. Consequently, anyone with pet cats or dogs or near domesticated animals is more likely to be bitten, but outbreaks in the absence of pets are common. One epidemic of flea bites among children in a day nursery was traced to dog fleas in a deserted fox nesting area beneath the building.[228] Another outbreak among poultry workers was caused by an infestation of hen fleas, *Ceratophyllus gallinae*.[264] Fleas from flying squirrels also have been documented as the source of bites.

Burrowing Flea

Flea infestations tend to be similar clinically and associated with the distribution of animal hosts. However, one flea, *Tunga penetrans,* is responsible for a distinctive infestation known as tungiasis (Fig. 44-57). The flea has a number of common names: burrowing flea, chigo, sand flea, and jigger. Infestation is common in Central and South America and in Africa, where the burrowing flea is widely distributed, but the incidence in the United States is rising as increasing numbers of tourists visit exotic places. One resident of New York City developed lesions of tungiasis on her toes after visiting several countries in East Africa, where she frequently wore sandals in rural areas.[284] The primary lesions of tungiasis are produced by the female flea. As

Figure 44-57. Tungiasis. (From Feldmeier H, Eisele M, Sabóia-Moura RC, Heukelbach J: Emerg Infect Dis 9:949–955, 2003.)

soon as it is impregnated, it burrows into the skin until only the posterior end protrudes. Sucking blood, the insect becomes as large as a small pea and deposits eggs that fall to the ground. Lodged in the skin, the gestating female produces a firm itchy nodule, with the posterior end of the flea visible as a dark plug or spot in the center of the nodule. Lesions occur most often on the feet, buttocks, or perineum of persons who wear no shoes or frequently squat, because the burrowing flea is not a good jumper and abounds in the dusty soil surrounding human habitations. If the infestation is extensive, numerous papules may aggregate into plaques with a honeycomb appearance. Secondary infections become painful or even crippling, and severe infections may lead to death. If the burrowing flea is not removed, the pustule ruptures, leaving an ulcer. Wearing shoes prevents most cases of tungiasis.

Clinical Aspects

The appearance of flea bites is not diagnostic, and the clinical features depend on the degree of sensitivity. A bite produces a small, central hemorrhagic punctum surrounded by erythema and urticaria. A small wheal at the bite site may be nonallergic because of primary urticariogenic substances in the flea saliva, but increasingly severe reactions are caused by sensitization to substances in the saliva. Bullae or even ulceration may result from flea bites in highly sensitive individuals. Flea bites are intensely pruritic, and scratching often results in crusting and impetiginization. Fleas have a habit of sampling several adjacent areas while feeding, and bites characteristically appear in irregular groups. Feet, ankles, and legs, as well as the hips and shoulder areas, where clothing fits snugly, are favorite targets. Although an individual lesion produced by a flea bite is not diagnostic, the typical clinical picture of grouped multiple bites is generally sufficient to establish a diagnosis, which is usually confirmed by locating and identifying fleas.

Treatment and Prevention

Ordinary flea bites require symptomatic treatment directed at relief of pruritus and prevention of secondary infection. Corticosteroid creams or calamine lotion with phenol, systemic antihistamines, and antibiotics are helpful when indicated, but the management of flea bites consists largely of prevention. The animals that host the fleas must be treated, as well as such places as chicken coops, rat nests, sleeping sites for dogs and cats, and often dwellings where pets live. Many effective insecticides are available. Typically, *N,N*-diethyl-meta-toluamide (DEET; also called diethyltoluamide), pyrethrins, piperonyl butoxide, and D-trans-allethrin are ingredients in sprays and foggers. An insect spray containing permethrin may be effective. Spraying or dusting must eradicate not only adult fleas but also the many larvae and pupae in grass, carpet, floorboards, furniture, and beds. Carbaryl and Malathion are the active ingredients in many sprays and dusts, and the services of professional exterminators may be necessary.

Veterinary prescriptions are available for control of fleas on dogs and cats. Preparations containing 9.1% imidacloprid (Advantage, Bayer) eliminate or reduce fleas on dogs when applied to the skin; 98% to 100% of fleas are killed within 12 hours of application, and re-infesting fleas are killed for 4 weeks after a single application. An oral preparation used for both dogs and cats contains lufenuron, an inhibitor of insect development. Lufenuron does not kill adult fleas but rather controls flea populations by interrupting the life cycle at the egg stage.

Cases of burrowing fleas should be treated promptly. One method is curettage of each nodule under local anesthesia, with concomitant use of systemic antibiotics to prevent secondary infection. Ether pledgets applied to the skin will kill the fleas before curettage is begun. Where burrowing fleas are endemic, eradication is important. Floors must be swept free of dust, and insecticides may be sprayed or dusted.

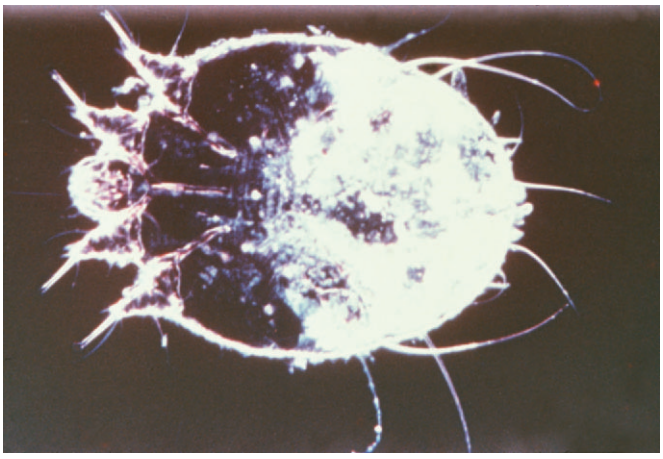
MITES (CLASS ARACHNIDA, ORDER ACARINA)

Species, Life Cycle, and Distribution

Mites make up the largest group in the class Arachnida. Most are small arthropods, and many are barely visible. Mites have two body regions, a small cephalothorax and a larger, unsegmented abdomen. The cephalothorax and abdomen are broadly joined, giving most mites an oblong to globular appearance. Newly hatched larvae have three pairs of legs, and larvae acquire a fourth pair after the first molt. Mites are highly diverse. Some are parasitic, with both vertebrates and invertebrates serving as hosts; some are scavengers, some feed on plants, and many are free living and predacious. Although most species are oviparous, some are ovoviviparous, and a few are viviparous. They occur worldwide and frequently in great numbers. Mites have been associated with disease transmission, allergies, and dermatologic manifestations. Of the approximately 35,000 species, about 50 are known to cause human skin lesions, and most of the cutaneous lesions are caused by mites feeding or burrowing in the skin. Because children and adults of all races are susceptible to these ubiquitous arthropods, they are responsible for considerable morbidity. The mites of medical importance are some of the sarcoptic mites, some of the trombiculid mites, a number of other acariform mites that infest organic substances such as grains and produce, and the gamasid mites that are vectors of several rickettsial and viral diseases. Dermatologic manifestations of mite bites may be seasonal, as with the trombiculids; individual cases or outbreaks of varying magnitude may be related to contact with mites that infest animals or various foods. Epidemics may occur, as is presently the case with scabies.

Scabies

Life Cycle. The human scabies mite is *Sarcoptes scabiei* var. *hominis,* an obligate human parasite that completes its entire life cycle in and on the epidermis of humans (Fig. 44-58). Unless treated, scabies can persist indefinitely. The adult female is responsible for the symptoms accompanying the infestation. After impregnation, she burrows into the epidermis and remains in the burrow for a life span of about 1 month. She slowly extends the burrow, feeding during travel, during which time several eggs are deposited daily. The ovoid female mite is approximately 0.3 to 0.4 mm long. Numerous transverse corrugations and dorsal spinous processes are adaptations to prevent backward movement in the burrow. The males are much smaller than the females, spend more time on the surface, and have a brief life, dying shortly after copulation. The mite is passed in the vast majority of cases by intimate contact, but adult human scabies mites can survive off the host for 24 to 36 hours at room conditions and still remain infestive.[8] Thus, scabies can be acquired from infested bedding, furniture, and clothing. Outbreaks not related to sexual activity occur fre-



A

B

Figure 44-58. Scabies mite, *Sarcoptes scabiei* var. *hominis.* (From CDC Public Health Image Library, courtesy Joe Miller/Reed and Crnrick Pharmaceuticals, 1975.)

Figure 44-59. Scabies—palm of hand. (From www.emedicine.com.)

Figure 44-60. Scabies—sole of foot. (From www.emedicine.com.)

quently among nursing home patients and personnel; epidemics in schools for small children are also common. Scabies became uncommon after World War II (during the war it was a common problem), but since 1964 the disease has increased to epidemic proportions worldwide.[192]

Clinical Aspects. Severe nocturnal pruritus is the hallmark of scabies. Itching also may be provoked by any sudden warming of the body and generally does not involve the face. A warm bath or radiant heat may cause a paroxysm of itching. Because the pruritus is caused by sensitization, 4 to 6 weeks may elapse between infestation and the onset of severe pruritus. Re-infestation is common, because eradicating the disease from all contacts simultaneously is often difficult, and re-infestation after cure results in prompt recurrence of symptoms. Cutaneous manifestations are varied. The primary lesion is the epidermal burrow, a tiny linear or serpentine track, rarely longer than 5 to 10 mm. The female mite may burrow anywhere on the body, but sites of predilection include the interdigital spaces, palms (Fig. 44-59), flexor surfaces of the wrists, elbows, feet and ankles, beltline, anterior axillary folds, lower buttocks, and penis and scrotum. The distribution of burrows in infants may be atypical, with burrows frequently found in the scalp and on the soles (Fig. 44-60). In the present epidemic, involving many people with excellent hygiene, cutaneous changes may be almost absent and burrows difficult to find. On the other hand, after the disease has been present for some time, eczematization, lichenification, impetiginization, myriad nonspecific papules and excoriations, and even urticaria may be present. The burrows are often the least conspicuous of various skin changes. The clinical picture varies with differences in personal hygiene, topical treatments used before diagnosis, and individual scratch threshold. Scabies can also result in secondary skin cellulitis, sepsis, and postinfective complications such as glomerulonephritis.[162,230]

Diagnosis is based on the combination of nocturnal pruritus and cutaneous findings and is confirmed by microscopic examination of burrow contents. The burrow and contents may be collected for examination by scraping with a scalpel blade or

Figure 44-61. Case of Norwegian scabies. (From www.dermatologylectures.com/dermnet.)

by pinching the skin to elevate it and shaving off a superficial layer. Burrows are often inflamed and no longer typical after the disease has been present for some time. The most productive sites to find burrows for examination are finger webs, sides of fingers, wrists, and elbows. Ectoparasites, ova, egg castings, feces, or pieces of mites are diagnostic.

Norwegian scabies is a term describing a particularly severe form of scabies occasionally seen in senile and mentally impaired patients, those with debilitating illnesses, and immunosuppressed patients (Fig. 44-61). Extensive crusting occurs, particularly of the hands and feet. Erythema and scaling may develop, and patients are literally "crawling with mites." This form of scabies is highly contagious, resulting from the incredible number of mites on the patient and in the immediate vicinity.[40,142]

Nodular scabies is another troublesome clinical variation. Persistent pruritic nodules develop, particularly on the male genitalia or in the groin, but usually on some covered body part. Nodules may be the only finding and may persist for months after adequate antiscabetic therapy.

Treatment and Prevention. A number of topical treatments are available. In most cases, a single overnight application of 5% permethrin cream (Elimite) is curative. Permethrin has the advantages of low mammalian toxicity and high cure rate.[262] Even after adequate therapy, symptoms may persist more than a month until the mite and mite products are shed with the epidermis. The chemical must be applied even beneath the fingernails, since ova and live mites are frequently lodged there as a result of frenzied scratching. If the itching has not abated in several weeks, the patient should be reexamined for treatment failure or re-infestation. Permethrin may be used for retreatment, or alternative scabicides may be considered. Lindane lotion is highly effective but has the potential for central nervous system toxicity, and percutaneous absorption may occur.[20] It is contraindicated in infants and pregnant women and persons known to be allergic to hexachlorocyclohexane, and it is reserved for adults with resistant strains. Sulfur in petrolatum (5% to 10%) or another suitable vehicle applied for three consecutive nights is a suitable alternative. Crotamiton 10% cream or lotion applied for two consecutive nights is also used. In the treatment of Norwegian scabies, salicylic acid ointment may be needed to soften scales and permit penetration of the scabicide. Nodular scabies can be a perplexing therapeutic problem and may necessitate intralesional injections of corticosteroids in addition to adequate antiscabetic therapy. Application of crude coal tar to the nodules has been recommended. In France, a combination of 10% benzyl benzoate and 2% sulfiram has proven useful in scabies treatment.[31]

Contacts must be treated simultaneously. Clothing and linens should be laundered the morning after treatment to kill mites that may have strayed from the skin. When many members of a household are infested, live mites may be on the furniture; γ-benzene hexachloride sprays are available.

Control of scabies outbreaks in nursing homes and similar epidemic situations can be almost insurmountable because of the number of patients and contacts that must be treated simultaneously. An uncured case of Norwegian scabies as the focus of an epidemic may be surrounded by millions of mites. Ivermectin is a cheap and effective alternative antiscabetic when taken orally and is useful in institutional epidemics.[7,28,31,85,231] A single dose of 250 μg/kg cured 10 of 11 patients with crusted Norwegian scabies as well as all otherwise healthy persons with scabies.[169] This agent is also approved in humans for strongyloidiasis and onchocerciasis.

Zoonotic Scabies. Other burrowing mites similar to the human scabies mite infest animals, such as swine, cattle, horses, mules, sheep, dogs, and wild animals. They are relatively host specific but under conditions of close contact may cause self-limited dermatitis in humans. Because of humans' close association with dogs, the most common animal scabies is canine, caused by *Sarcoptes scabiei* var. *canis*. Studies indicate that the dog scabies mites are able to survive for at least 96 hours on human skin, even burrowing and laying eggs, but whether a perpetual life cycle can be established is not yet determined.[79] Infested dogs have reddish papules, scaling, crusting, and evidence of scratching. Humans develop itchy papules, often with some urtication, and scratching may give rise to varying degrees of secondary infection. The initial lesions are most often on areas of skin that come in contact with dogs: forearms, chest, anterior abdomen, and anterior thighs. Outbreaks are frequently traced to a kennel or litter of puppies. In one case, 15 patients developed an itchy dermatitis from five puppies in one litter.[45] Human infestation with dog scabies mites subsides spontaneously when contact with dogs is discontinued or when the dogs are cured. The dogs must be treated with scabicides and the human victims with symptomatic therapy for pruritus. Cats, also closely associated with humans, have been known to harbor mites that can infest humans. *Notoedres cati* infestation is seen more often in Czechoslovakia and Japan than is dog sarcoptic scabies.[42]

Trombiculid Mites

Mites of the family Trombiculidae are distributed worldwide. In the United States the most important species is *Eutrombicula alfreddugesi* (red bug, chigger [Fig. 44-62], harvest mite). Another species, *Eutrombicula batatas*, is also indigenous to parts of the United States. Adults are free living and predacious on small arthropods and their eggs, but the larvae are ectoparasites of vertebrates. Wild and domestic mammals, as well as

Figure 44-62. Chigger mite, *Eutrombicula alfreddugesi.*

Figure 44-63. Chigger bites.

reptiles, serve as hosts. The larval bite causes human dermatitis. Adult mites lay their eggs among vegetation, and newly hatched larvae crawl up on the vegetation, waiting to attach themselves to a passing host. They attach themselves to the skin with hooked mouthparts and feed on blood, falling off when full. However, humans are not good hosts, and larvae usually do not stay long. Severity of the response depends on the species of trombiculid, the irritating qualities of the mites' saliva, and the host's allergic response. The typical bite is a maddeningly pruritic, hemorrhagic punctum that usually becomes surrounded by intense erythema within 24 hours. Bites may number in the hundreds and can be associated with an allergic reaction. Hypersensitivity causes blisters and weeping of clear fluid with crusting. The surrounding area may be purplish in color, with severe swelling, particularly of the feet and ankles (Fig. 44-63). The lesions regress in 1 to 2 weeks, but pruritus is persistent and often paroxysmal during this time, with secondary infection in excoriated skin. One pediatric case series reported a "summer penile syndrome," describing a seasonal hypersensitivity reaction caused by chigger bites to the penis in 94 male patients over a 4-month period from June to September.[252]

Treatment is symptomatic and consists of topical antipruritic agents, corticosteroids, systemic antihistamines, and, occasionally, pulse therapy with systemic corticosteroids. Superpotent topical corticosteroid creams and ointments, such as 0.05% clobetasol, applied sparingly to individual bites several times daily, are effective but must be used properly. Prolonged application can result in dermal atrophy, and absorption can be significant if excessive body surface is treated. Phenol (1% in calamine) often is effective. As in all self-limited conditions with no satisfactory cure, home remedies abound, such as meat tenderizer rubbed into the moistened skin. Application of clear nail polish to the individual lesions is a popular home remedy, even though no evidence suggests that this is effective.

Preventive measures consist of avoidance, and insect repellents used on skin and clothing. Clothing pretreated with permethrin has resulted in a 74.2% increase in protection compared with unprotected controls.[25] Other repellents suggested for treating clothing are ethylhexanediol, DEET, and flowers of sulfur. Permanent residents in infested areas may develop tolerance to repeated bites.

Miscellaneous Mites

Parasitiformes. The Parasitiformes group contains gamasid mites that are parasites of birds, mammals, snakes, insects, and, rarely, humans. In addition to being vectors of disease, gamasid mites are responsible for some cases of dermatitis. The chicken mite, *Dermanyssus gallinae,* is responsible for most of the dermatitis caused by this group. This pest of poultry is widespread and is associated with both domestic and wild birds. Poultry workers are common targets, but other persons may be infested from insidious sources, such as a pet canary or a bird nest near an intake for ventilation or air conditioning. The clinical picture is nonspecific, but the diagnosis may be made by identifying the mite. Treatment consists of symptomatic therapy and eradication of the mite source.

The tropical rat mite *Ornithonyssus bacoti* has also been reported to cause dermatitis from such diverse sources as a rat nest in the attic or a colony of laboratory mice.[44,88] Snake mites have been implicated as a cause of dermatitis. Four members of one family developed a vesicobullous eruption from *Ophionyssus natricis* harbored by a pet python.[236]

Acariniformes. The Acariniformes are a huge group that includes mites that infest foods, feathers, and furs. Individual

infestations and larger outbreaks are common, with increased exposure by occupation, resulting in such terms as grocer's itch, miller's itch, and copra itch. Dogs, cats, and rabbits are primary hosts for mites of the genus *Cheyletiella,* and domestic pets are increasingly the source of mite dermatitis. Pet house cats are often involved.[220]

Mites of the genus *Dermatophagoides* are said to be the principal inhaled allergen of house dust. *Dermatophagoides scheremetewskyi* is an unusual mite that has been found in kapok and feather pillows, in a sparrow's nest, in monkey food, and on rats and other animals. This mite has been reported as the cause of feather pillow dermatitis.[10]

The most common type of dermatitis in this group is grain itch caused by *Pyemotes ventricosus*. This tiny mite parasitizes various insects often found in and around grain and straw. It attacks humans when a large mite population has no ready access to normal hosts. Grain itch implies an occupational bias, but outbreaks not involving farmers or rural workers have been described. During a widespread epidemic of *Pyemotes* infestation of farm workers in the midwestern United States in 1950 to 1951, straw used at the Indiana State Fair was infested. During a 2-year period, 642 visitors were treated for grain itch at a dispensary maintained on the fairgrounds, and about 1100 animal attendants and fair workers were treated over the same period at a separate facility. The reservoir of infestation by *Pyemotes* may be quite obscure. Several reported cases were associated with the common furniture beetle *Anobium punctatum* in the floor joists of a house.[83] Therapy is symptomatic. Large-scale eradication measures may require the services of professional exterminators.

GENERAL TREATMENT OF INSECT BITES

Oral antihistamines can be effective in reducing the symptoms of insect bites. Cetirizine was given prophylactically in a double-blind, placebo-controlled, 2-week crossover trial to 18 individuals who had previously experienced dramatic cutaneous reactions to mosquito bites.[214] Subjects given the active drug had a 40% decrease in both the size of the wheal response at 15 minutes and the size of bite papule at 24 hours. The mean pruritus score, measured at 15 minutes and 1, 12, and 24 hours after being bitten, was 67% less than that of the untreated controls. In highly sensitized individuals, prophylactic treatment with nonsedating antihistamines, such as loratadine, may safely reduce the cutaneous reactions to insect bites.[3,226]

A 3.6% ammonium solution (After Bite) relieves the type I hypersensitivity symptoms associated with mosquito bites. In a double-blind, placebo-controlled laboratory trial, 64% of mosquito-bitten subjects experienced complete relief of symptoms after a single application of the ammonium solution, and the remaining 36% had partial relief lasting 15 to 90 minutes. No subjects treated with placebo reported complete symptom relief.[286]

PROTECTION AND PREVENTION

An integrated approach to personal protection is the most effective way to prevent arthropod bites, regardless of location and species. Protection from arthropod bites is best achieved by avoiding infested habitats, wearing protective clothing, and using insect repellents.[89] Insect repellents containing DEET are the most effective products on the market, providing broad-spectrum repellency lasting several hours.[89,223] Currently available non-DEET–containing repellents do not provide protection for as long as DEET-based repellents. DEET's peak duration of action plateaus at a concentration of 50%, with no added benefit from products containing higher concentrations.[89] Additionally, liberal use of higher-concentration products with ethanol bases can lead to increased skin absorption and neurotoxicity, particularly in young children.[223] Topical insect repellents alone do not provide complete protection. Ethyl butylacetylaminopropinate (IR3535)–containing products can provide protection for up to 3 hours and can be added to sunscreen lotions without safety concerns or potential side effects.[263] A piperidine derivative (Bayrepel) offers 2 to 8 hours of protection similar to DEET but is currently available only in Europe. Large doses of vitamin B_1 have not proven effective.[89,90]

Mosquitoes can find and bite any untreated skin and may even bite through thin clothing. Deerflies, biting midges, and some blackflies prefer to bite around the head and will crawl into the hair to bite unprotected areas. Wearing protective clothing, including a hat, reduces the chances of being bitten. Treating clothes and hats with permethrin, in addition to spraying DEET on the skin, offers the greatest protection by also repelling any insect that crawls or lands on the treated clothing.[89] To prevent chiggers or ticks from crawling up the legs, pants should be tucked into boots or stockings.

Persons traveling to a part of the world where insect-borne disease is a potential threat should be certain to learn about indigenous insects and the diseases they might transmit. Protective clothing, mesh insect tents or bedding, insect repellent, and permethrin spray should be carried.

DELUSIONS OF PARASITOSIS

Patients with delusions of parasitosis are convinced, against all evidence to the contrary, that parasites infest their skin and often their homes and clothing. No single cause is known for this condition, although some cases may be associated with proven parasitic infestation. The idea may also be suggested by infestations of relatives or acquaintances. Patients older than 50 years are most often female; patients younger than 50 are equally male and female. Most cases of delusions of parasitosis commence with pruritus, which may be accompanied by crawling, creeping, stinging, and burning sensations. The initial reaction is to scratch, replaced soon by digging to remove the "parasites." Self-mutilation and suicidal behavior may develop. Generally, the first contact with a physician is to bring in evidence of the "infestation." Evidence typically consists of scales, lint, crusts, hairs, dust, and small pieces of skin, carefully collected and stored in a small box or folded in facial tissue. Medical attention is often sought not to relieve the symptoms but to eradicate the parasites. Patients may take the evidence to a professional entomologist for identification and may employ professional exterminators for repeated fumigation. Patients may be so convincing that household members or acquaintances come to share the delusion.[63,80]

Many patients with parasitophobia know that their fear is groundless but are still unable to overcome it. Other patients with delusions of parasitosis are convinced that they have an infestation and regard as incompetent the physician who makes

the correct diagnosis of no infestation. A complete examination of the patient and the evidence is essential, and investigation of the home or workplace may be indicated. Other medical conditions that may produce cutaneous sensations include liver and renal disease, alcoholism and toxic states, diabetes mellitus, cardiovascular disease, lymphoma, anemia, sideropenia, vitamin B_{12} deficiency, pellagra, peripheral neuritis, dermatitis herpetiformis, drug reactions, and environmental irritants (e.g., arthropods, fiberglass).[157,266,283]

Psychiatric intervention is often unsatisfactory to both patient and physician. Convinced that the physician is wrong, patients often seek repeated opinions and finally become despondent. Pimozide, a neuroleptic medication used to treat other monosymptomatic hypochondriacal psychoses, has been found useful in treating this condition.[183,211,218,283] In one group of 14 patients treated with pimozide and followed for an average of 34 months, seven had complete remissions, three had relapses that responded to treatment with pimozide, and four were treatment failures.[150]

ACKNOWLEDGMENTS

Special thanks and appreciation to Sherman Minton and Bernard Bechtel for their valuable contributions to the original versions of this chapter in previous editions.

The references for this chapter can be found on the accompanying DVD-ROM.

45 Tick-Borne Diseases

Stephen J. Traub and Gregory A. Cummins

As a group, the approximately 850 species of ticks rank second only to mosquitoes in importance as insect vectors of human disease.[164] Tick-borne disease was first described when Hippocrates noted an epidemic of relapsing fever nearly 2500 years ago. In the early 18th century, European physicians described patients with signs and symptoms of Lyme disease. In 1857, David Livingstone, the British explorer of Africa, discovered that a human disease was caused by spirochetes from soft ticks. In the late 1880s, Dr. Arvid Afzelius in Europe described a ring-like lesion in association with the bite of the *Ixodes ricinus* tick. This was almost surely Lyme disease, and a causative agent of Lyme disease in Europe, *Borrelia afzelii,* is named after him. The German physician Robert Koch confirmed the role of ticks in cases of tick-borne relapsing fever, previously thought to be a variant of malaria, in Africa in 1904.

Ticks are the most common insect vectors of disease in the United States (Fig. 45-1). For those who have outdoor interests and enjoy outdoor activities, ticks are unavoidable. Ticks and the diseases they transmit may also afflict those who do not actively participate in outdoor activities, as pets may bring them into the home where they may survive for weeks before transmitting disease. Also, several tick-borne diseases are readily transmissible by blood donation.[208]

New infectious diseases continue to emerge, and our awareness of them continues to grow. Ticks have recently been discovered to transmit several diseases that were formerly unknown, that were not known in humans, or that were not formerly known to be transmitted by ticks—for example, ticks have recently been implicated in transmission of the West Nile virus.[204]

The overall warming trend noted around the globe, which is likely to continue,[210,392] has expanded both the season in which ticks are found and the window of opportunity for exposure to them.[78] The highest altitude at which tick-borne encephalitis is found in Europe has risen over the past 30 years in direct correlation with this warming.[392] As a likely consequence of warming, ticks known to transmit diseases are now found at higher altitudes than they were found in the past.[77,78]

Ticks often harbor, and may transmit, multiple infectious organisms simultaneously.[7,139,163,361,362] In one region, 24% of *I. ricinus* nymphs were infected with more than one infectious organism.[268] Different genospecies of *Borrelia* have been documented in a single patient with Lyme disease,[86] and it is likely that these multiple strains came from the same tick. In addition to the classic tick-associated illnesses described in this chapter, ticks may also harbor other bacteria. *I. ricinus* ticks may carry *Pasteurella pneumotropica/haemolytica* and other gram-negative organisms, including *Pseudomonas aeruginosa*.[346]

Ticks do not respect geographic borders. Many species of ticks feed on migratory animals, such as birds, that travel great distances.[8,189] Ticks can survive unnoticed on a traveler, or in a traveler's bag, from one continent to another. At least 29 different species of ticks have been introduced into the United States on imported reptiles.[48] Outdoor pets, especially dogs, are well documented to carry infected ticks.[25] One study indicated that dog owners are five times more likely to get spotted fever than are non–dog owners.[250] International travel is now easier, and areas not formerly accessible are easily visited. A group of American paleontologists working in Mongolia were recently

the correct diagnosis of no infestation. A complete examination of the patient and the evidence is essential, and investigation of the home or workplace may be indicated. Other medical conditions that may produce cutaneous sensations include liver and renal disease, alcoholism and toxic states, diabetes mellitus, cardiovascular disease, lymphoma, anemia, sideropenia, vitamin B_{12} deficiency, pellagra, peripheral neuritis, dermatitis herpetiformis, drug reactions, and environmental irritants (e.g., arthropods, fiberglass).[157,266,283]

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Figure 45-1. Painting by Sue Oliver depicting complex interaction among the tick vector, reservoir hosts, causative agent, and humans occurring in Lyme disease. (Courtesy Sue Oliver.)

Figure 45-2. Electron micrograph of the hypostome of an ixodid tick.

tested for North Asian tick typhus, and four of the 13 researchers were infected.[209]

Many of the agents causing diseases mentioned in this chapter are commonly researched in military biological warfare labs. Several of these agents are listed by the Centers for Disease Control and Prevention (CDC) as possible biological weapons.[34,65,180] Tularemia is listed as category A. Q fever is listed as category B, and most other tick-borne diseases are listed as category C, meaning they are of concern but not easily grown, disseminated, or spread from person to person. Although an outbreak of any of these diseases may produce only a few fatalities, such an outbreak could seriously strain any advanced healthcare system.

Most ticks are not species selective with respect to their hosts, although some exhibit species preference. At least one, which feeds only on the Galapagos giant tortoise,[166] is completely species specific. Some ticks are more anthropophilic (readily feeding on humans) than others. Different stages of a tick species may feed on different animals, even different orders of animals. For example, *Ixodes scapularis* larvae and nymphs feed readily on mammals, reptiles, and birds, with varying degrees of host preference,[173] whereas the adult ticks feed predominantly on large mammals.

Once the tick initiates feeding, the hypostome (feeding organ) enters the skin. The hypostome contains hundreds of barbs, pointed in reverse direction from the entry into the skin to serve as an anchor (Fig. 45-2). During feeding, the tick secretes a mixture of salivary proteins containing both anticoagulants and a proteinaceous cement. The combination of salivary proteins and mechanical irritation may cause a local reaction in the host, and the combination of reverse-pointing barbs and proteinaceous cement make embedded ticks difficult to remove.

Ticks belong to the order Acari (which includes mites) and the class Arachnida (which includes spiders and scorpions). Taxonomists divide ticks into two major families: Ixodidae (hard ticks) and Argasidae (soft ticks).

Ixodid Ticks

Ixodid ticks, which transmit a plethora of diseases (Table 45-1), possess a hard, shieldlike scutum. Their mouthparts are visible from the dorsal view (Fig. 45-3). The scutum covers only the anterior portion of the dorsum of the females but the entire dorsal surface of the males. The presence of the scutum limits the size of the tick during feeding. Thus, the body of the female (to a much greater extent than of the male) may expand tremendously with a blood meal, as the tick ingests up to 50 times its weight in blood and body secretions. Ixodid females generally feed and mate only once as adults, whereas males may feed and mate several times. Each female ixodid tick lays several thousand eggs in a single deposition, and then dies. These eggs hatch at approximately the same time, and if a potential host brushes by, thousands of larvae, or "seed ticks," can attach nearly simultaneously. Some species live through all three life stages (larva, nymph, adult) on a single host (and are thus called *one-host ticks*), while most species feed, drop into leaf litter, molt, and seek another host for each life stage (and are called *three-host ticks*).

TABLE 45-1. Ixodid Ticks (Ixodidae)

TICK SPECIES	TRANSMITTED DISEASES	GEOGRAPHIC DISTRIBUTION
Amblyomma americanum (lonestar tick)	Human monocytic ehrlichiosis (HME), southeastern tick-associated rash illness, tularemia	Southeastern and south-central United States, Eastern Seaboard
A. cajennense (cayenne tick)	Rocky Mountain spotted fever (RMSF)	Extreme south Texas, Mexico, Central America
A. hebraeum	Boutonneuse fever	Southern half of Africa
Dermacentor andersoni (Rocky Mountain wood tick)	RMSF, Colorado tick fever (CTF), tularemia, tick paralysis	Western half of United States, Rocky Mountain states
D. marginatum	Omsk hemorrhagic fever, Siberian tick typhus, tick-borne encephalitis (TBE)	Europe, western half of Asia
D. nuttalli	Siberian tick typhus, tularemia	Northern Asia
D. occidentalis	Tularemia, RMSF, CTF, tick paralysis	U.S. Pacific coast, Mexico
D. silvarum	TBE, Siberian tick typhus	Eastern Europe, northern Asia
D. variabilis (American dog tick)	RMSF, tularemia, tick paralysis, human granulocytic ehrlichiosis (HGE)	Entire United States except Rocky Mountain states; Mexico
Haemaphysalis concinna	Siberian tick typhus, tickborne encephalitis	Europe and temperate Asia
H. leachi	Boutonneuse fever	Africa, Southeast Asia, Indonesia
H. spinigera	Kyasanur forest virus	Southern half of India; Indonesia
Hyalomma anatolicum	Crimean-Congo hemorrhagic fever (CCHF)	Northern half Africa, southern Europe, southern Asia
H. asiaticum	Siberian tick typhus, spotted fever group	South-central Asia
H. marginatum	CCHF	Africa, southern Europe, southern Asia
Ixodes holocyclus	Tick paralysis, spotted fever group	Australia
I. pacificus (western black-legged tick)	Lyme disease, HGE	United States and Canada west of Rocky Mountains
I. persulcatus (taiga tick)	Lyme disease, Russian Spring-Summer encephalitis (RSSE)	Europe, northern half of Asia
I. ricinus (castor bean tick)	Lyme disease, TBE, babesiosis, tularemia, HGE	Northern Africa, Europe, Asia
I. scapularis (black-legged tick)	Lyme disease, RMSF, HGE, babesiosis	Eastern third of United States
Rhipicephalus sanguineus (brown dog tick)	Spotted fever group, Q fever	Worldwide

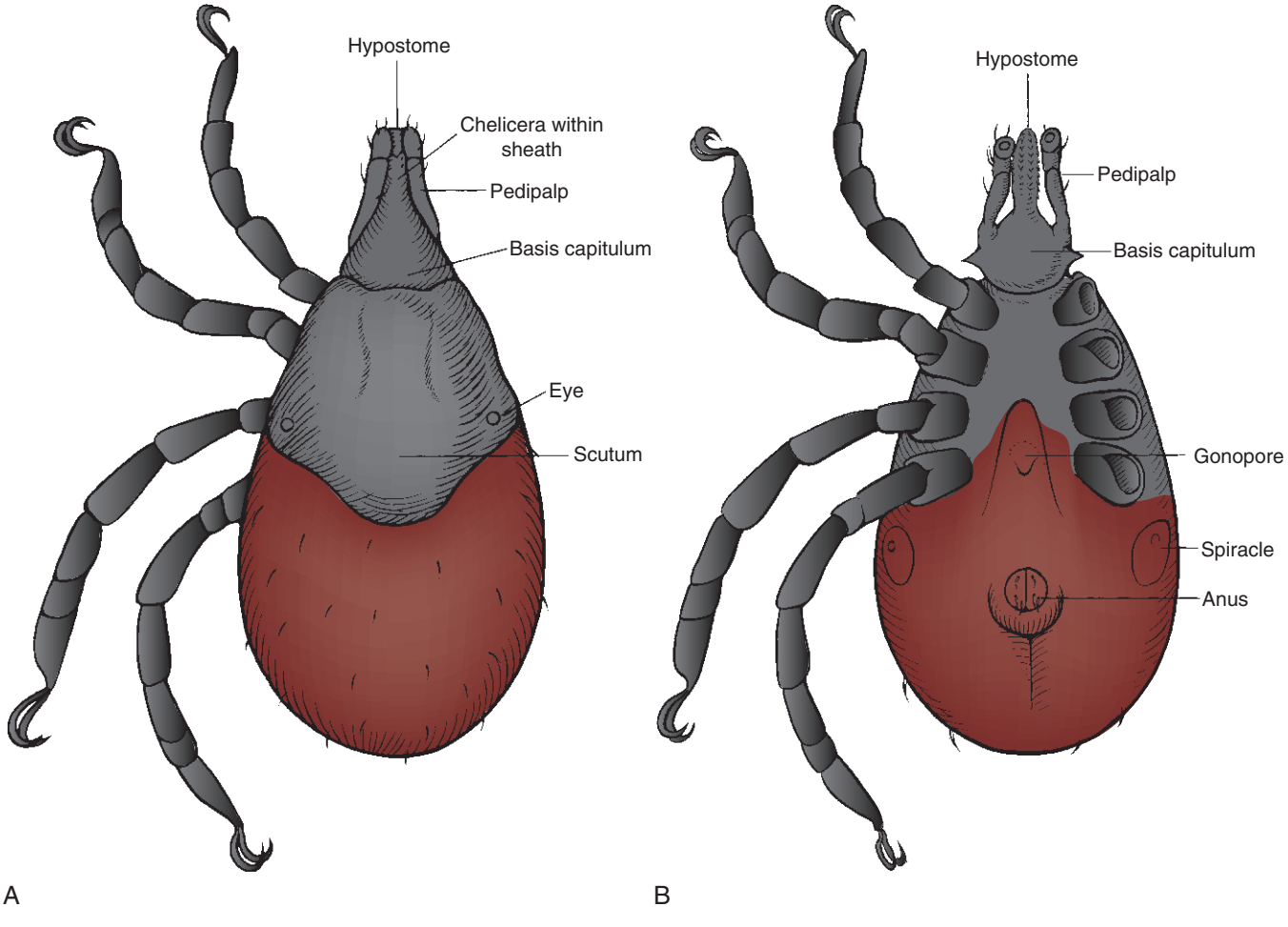

Figure 45-3. Tick anatomy. **A,** Dorsal aspect. **B,** Ventral surface.

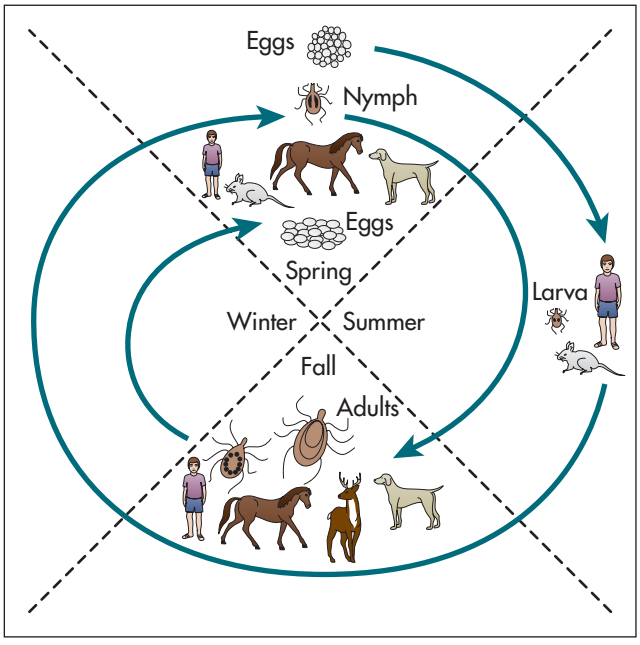

Figure 45-4. Life cycle of *Ixodes scapularis*. The cycle of ixodid ticks typically spans 2 years and includes three blood meals in the spring. The adult female tick releases her eggs, which hatch as six-legged larvae, during the summer. The larvae take blood meals that last approximately 2 days. Larval ticks then enter a dormant phase with cooler fall weather. The ticks then molt in the spring, entering the second phase of their life cycle as eight-legged nymphal ticks. In the late spring or summer, nymphal ticks take blood meals that typically last for 2 to 3 days. The nymphal ticks then molt as eight-legged adults in the fall. The adults mate on white-tailed deer. After mating, the male dies, but the female takes one more blood meal before she lays her eggs and dies. (Modified from Habicht GS, Beck G, Benach JL: Sci Am 257:78–83, 1987.)

Figure 45-5. Soft ticks (family Argasidae) possess a leathery integument rather than the tough scutum possessed by hard ticks (family Ixodidae). North American argasid ticks of the genus *Antricola* possess a tuberculated, or "knobby," roughened integument, as seen here from a close-up perspective. These ticks are parasites that use bats as hosts. (Courtesy CDC, 1975.)

A typical ixodid tick life cycle is depicted in Figure 45-4. It is typically a 2-year cycle, although in some areas it may be 1 year.[101] The tick hatches as a six-legged larva and attaches to a small mammal, often a rodent. After feeding for 3 to 5 days, it drops off and molts to the eight-legged nymph. The nymph hibernates in the soil and becomes active again in the spring. It then attaches to and feeds on a larger animal for 4 to 9 days, after which it again drops off (into leaf litter) and molts a second time to the adult stage. The mature tick attaches to a third host, on which feeding and mating may occur. A large mammal, such as a deer, may support thousands of ticks.

Ticks do not find their hosts haphazardly. The tick's front legs are equipped with chemoreceptors to detect respiratory gases (especially CO_2) and proprioceptors to detect fine vibrations. Nymphs and adults actually climb up grass blades and "seek" a host ("seeking" or "questing"), holding up the front legs that also contain small barbs to help them attach. The ticks hook onto the host's fur, feather, scale, or skin and find a place to attach, often after climbing to an area with a relatively abundant blood supply.[201] Ixodids are generally on their host for hours to a day or more before they begin to feed, and the feeding process may last from days to weeks. The duration of feeding depends more on the host than the tick; feeding on reptiles, for example, occurs more slowly than on other species.

A blood meal and mating, which usually occurs on the host, are prerequisites to laying eggs. Females, once engorged, drop off the host often in the night or early morning hours and typically into an animal's nest or bedding area, and lay eggs nearby in leaf litter or nest material. This provides a meal for the larval ticks upon hatching.

Ixodid ticks typically require dense vegetation and prefer areas of high animal traffic and humidity, such as leaf litter or moist soils. "Seeking" adults are often found on animal paths or game trails.[134] They are found in temperatures from freezing to over 100° F (38° C), although they move more slowly at cold temperatures. They can be frozen in a block of ice yet when thawed move freely and feed without difficulty.

Argasid Ticks

Argasid ticks do not exhibit sexual dimorphism or have a hard scutum. The mouthparts are not visible from above. The body typically has a wrinkled, leathery appearance (Fig. 45-5). Argasid ticks move quickly in comparison to the ixodids.

Argasid ticks usually feed completely in 10 to 30 minutes and do not cause pain. They generally feed at night, often moving unnoticed while their hosts sleep. After feeding, they drop off the host and lay dozens to a few hundred eggs and then repeat the cycle many times. The remainder of the life cycle is similar to that of the ixodids, except that nymphs may have multiple stages. Argasid ticks are generally found in animal burrows, dens, and caves, and in the floors and walls of huts and cabins. They thrive in hot, dry climates.

TICKS AS POISONOUS ARTHROPODS

Tick Paralysis

As early as 1912, Todd[355] recognized that an acute, ascending, flaccid motor paralysis followed the bite of certain ticks. Tick paralysis has been reported worldwide, although most human cases occur in North America and Australia.[325,381] Forty-three species of ticks have been reported to cause tick paralysis.[63]

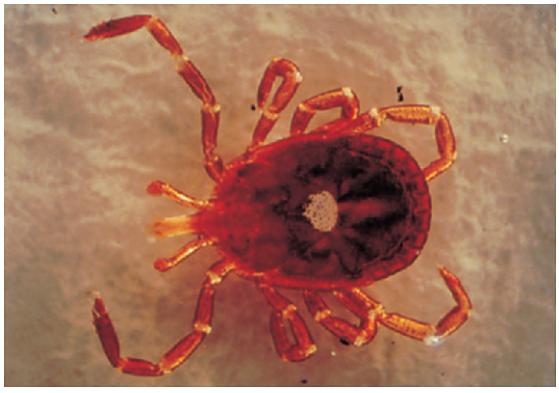

Figure 45-6. Lone-star tick (*Amblyomma americanum*), implicated in cases of tick paralysis in North America. (Courtesy Sherman Minton, MD.)

Human cases in North America are usually caused by *Dermacentor andersoni,* although *Dermacentor variabilis, Amblyomma americanum* (Fig. 45-6), *Amblyomma maculatum,* and *Ixodes scapularis* have also been implicated.[147] The Pacific Northwest and Rocky Mountain areas account for most cases. In Australia, *Ixodes holocyclus* is primarily associated with the disease, although *Ixodes cornuatus* has been implicated.[148,150]

Tick paralysis usually occurs during the spring and summer months (April to June), when nymphs and mature wood ticks are feeding. Most cases occur in areas where ticks are endemic. Patients in urban areas may be affected if they have recently traveled[144]; however, the diagnosis should not be excluded on the basis of geography alone. Children are affected more often than adults, with most cases reported among girls less than 10 years of age. Among children, girls are affected twice as often as boys, probably because ticks on the female scalp are hidden in longer hair.[1] Among adults, men account for most cases, presumably because of increased occupational and recreational exposure to tick habitats.

A neurotoxic venom secreted from tick salivary glands during a blood meal causes the paralysis. The venom appears to diminish acetylcholine release at the neuromuscular junction,[146,252] leading to a conduction block. Electrophysiologic measurements in humans consistently demonstrate slower motor conduction and reduction in muscle action potential amplitude.[93,247,349] The neurotoxin may also increase the stimulatory current potential necessary to elicit a response at the motor end plate.[190] The toxin appears to have central effects as well.

Tick paralysis in humans develops 5 to 6 days after an adult female tick attaches, usually to the head or neck. Initially, the victim may be restless and irritable and may complain of paresthesias in the hands and feet. Over the next 24 to 48 hours, an ascending, symmetrical, and flaccid paralysis develops. Deep tendon reflexes are lost. Weakness is initially greater in the lower extremities, but within 1 to 2 days, severe, generalized weakness may develop, accompanied by bulbar and respiratory paralysis. Some victims develop cerebellar dysfunction with incoordination and ataxia.[1,146] Facial paralysis as an isolated finding has occurred in patients with ticks embedded behind the ear.[263] Undiagnosed, tick paralysis may be fatal; mortality in children was 12% in a large Canadian series.[305]

Case reports of Australian tick paralysis suggest that it may be more severe than its North American counterpart. Victims often appear more acutely ill. Paralysis may continue to progress for 48 hours after tick removal, and recovery may be prolonged.[16,17,263] A recent report of six cases in children in Australia found little difference in the course between Australian and North American patients.[148]

Diagnosis is made solely on clinical grounds, as specific diagnostic laboratory tests to diagnose this condition are not available. White blood cell (WBC) count and cerebrospinal fluid (CSF) analyses are normal. If the tick is on the scalp, magnetic resonance imaging (MRI) performed as part of the diagnostic workup for other conditions may identify it incidentally as a small nodule that is hypodense to fat on T1 imaging, that is isointense to fat on T2 imaging, and that does not enhance with gadolinium.[47] The diagnosis is confirmed on resolution of paralysis after removal of the tick.

The most common misdiagnosis is Guillain-Barré syndrome; the clinical features of these diseases are nearly identical, and results of electrophysiologic studies are indistinguishable.[363] Although the CSF in patients with Guillain-Barré syndrome is classically described as having an elevated protein level, this finding may be absent in up to 30% of patients,[12] especially early in the disease. For this reason, an abnormal CSF protein analysis strongly favors a diagnosis of Guillain-Barré, but a normal analysis does not rule it out. The differential diagnosis should also include botulism, myasthenia gravis, and potassium-associated paralyses, and specific testing should readily confirm or exclude these diagnoses.

After removal of the tick, treatment is supportive; severely affected patients may require ventilatory support. Tick antivenom from hyperimmunized dogs has been developed for Australian tick paralysis and may be beneficial in victims with severe disease, although treated patients have a high incidence of acute allergy and serum sickness.[148,263]

TICKS AS VECTORS OF INFECTIOUS DISEASES

Ticks transmit a wide variety of infectious agents, and may act either as amplifiers or as reservoirs for a given infectious agent.[165] In the *agent–tick amplifier system,* an immature tick ingests the microorganism while feeding on an infected vertebrate, and the vertebrate is the reservoir for the disease. The pathogen replicates in the tick and is passed transtadially, from larval to nymphal to adult stage. The maturing tick transmits the agent to other vertebrate hosts when it feeds. A key epidemiologic feature of this system, transtadial survival of microorganisms, is common in ticks but rare in other hematophagous insects. This important difference is primarily due to the fact that tick anatomy is relatively unchanged as the tick molts.[165]

In the *agent–tick reservoir system,* the microorganism is passed transovarially from one generation of ticks to the next. The agent replicates within the tick and depends solely on the tick population for survival. The agent may also replicate within a vertebrate host, allowing for amplification and increasing the density and prevalence of the microorganism, but it need not do so.

Table 45-2 lists the major tick-borne diseases in the United States. Lyme disease is now the most common tick-borne illness

TABLE 45-2. Major Tick-Borne Diseases in the United States

DISEASE	ORGANISM	MAJOR VECTORS	GEOGRAPHIC DISTRIBUTION
Lyme disease	*Borrelia burgdorferi*	*Ixodes scapularis, I. pacificus*	Coastal mid-Atlantic, northern West Coast, Wisconsin, Minnesota
Rocky Mountain spotted fever	*Rickettsia rickettsii*	*Dermacentor andersoni, D. variabilis*	South-central states, coastal southern states
Relapsing fever	*Borrelia hermsii, B. turicatae, B. parkeri*	*Ornithodoros hermsi, O. turicata, O. parkeri*	Worldwide; most often rural and wilderness areas of western states
Colorado tick fever	*Orbivirus*	*D. andersoni*	Rocky Mountain states, California, Oregon
Ehrlichiosis	*Ehrlichia chaffeensis*	*Ixodes scapularis?*	Coastal mid-Atlantic states, northern West Coast, Wisconsin, Minnesota
		Amblyomma americanum?	South-central states, coastal southern states
Babesiosis	*Babesia microti*	*Ixodes scapularis*	Coastal southern New England and mid-Atlantic states
Tularemia	*Francisella tularensis*	*A. americanum*	South-central states, Montana, South Dakota

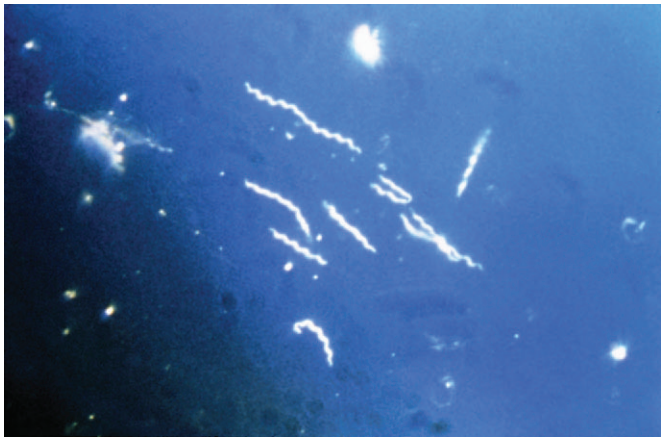

Figure 45-7. Lyme disease spirochete viewed under darkfield microscopy. (Courtesy CDC Public Health Information Library.)

in the United States and throughout the world. Tularemia and Rocky Mountain spotted fever are observed throughout the United States and continue to produce significant morbidity and mortality. Tick-borne relapsing fever and Colorado tick fever occur in the western states and are particularly likely to affect campers, hikers, hunters, and others who frequent wilderness areas.

Tick-Borne Borrelial Diseases

The genus *Borrelia* belongs, along with *Treponema* and *Leptospira,* to the order Spirochaetales, which are helical bacteria. Borreliae are usually 10 to 20 µm long, with three to ten spirals.[115] When live cultures are viewed under darkfield microscopy (Fig. 45-7), the organisms are very motile. *Borrelia* species can be stained with aniline dyes, a feature that readily distinguishes them from *Treponema* and *Leptospira* genera.[322] Borreliae can also be stained with Wright and Giemsa stains, although obtaining the organisms from blood is extremely difficult with most diseases (tick-borne relapsing fever being an exception). Culturing *Borrelia* species is technically challenging and must be done in properly formulated media incubated under specific conditions in specially equipped laboratories. In the relapsing fever borreliae, strains cannot be differentiated on the basis of morphology but are classified according to specificity of the tick–spirochete relationship, the range of animals susceptible to infection, and cross-immunity.[18]

The reservoir hosts, vectors, different species, and even different strains of *Borrelia* vary tremendously in different geographic areas.[200,211,306,360,372] The medically important diseases caused by *Borrelia* species in the United States include Lyme disease (caused by *Borrelia burgdorferi and Borrelia bissettii*), tick-borne relapsing fever (caused by *Borrelia recurrentis*), and STARI, or southern tick-associated rash illness (caused by *Borrelia lonestari*) (Fig. 45-8). Other important *Borrelia* species are being found regularly as more is learned about the spirochetes, hosts, and reservoirs.[306,361,362]

Lyme Disease

In Europe, the rash known as erythema chronicum migrans (ECM) has been associated with the bite of *I. ricinus* for nearly a hundred years.[3]

In the mid-1970s, a team from Yale University School of Medicine investigated a cluster of cases of arthritis in children near Old Lyme, Connecticut. The arthritis was initially thought to be juvenile rheumatoid arthritis. On further examination, however, the clustering, rural setting, and some response to penicillins pointed to an infectious, most likely arthropod-borne etiology. The seasonality and frequent finding of a rash pointed to a tick vector.[334]

Epidemiologic evidence implicated the deer tick, *Ixodes scapularis,* as the likely vector of what is now called Lyme disease. In 1982, Burgdorfer and associates[46] isolated a treponeme-like spirochete, *Borrelia burgdorferi,* from the midgut of *I. scapularis* ticks collected from a known endemic focus of Lyme disease.[27] Subsequently, sera from nine patients clinically diagnosed with Lyme disease yielded high antibody titers to the spirochetes by indirect immunofluorescence.[2] Isolation of *B. burgdorferi* from the blood, CSF, and skin lesions of affected patients finally confirmed the spirochetal etiology of Lyme disease.[29,30,338]

More than 12 species of *Borrelia* are now known to cause some variation of Lyme disease throughout the world. Most of these species cause disease in specific geographic regions and are transmitted almost exclusively by *Ixodes* ticks. In addition to

A

B

Figure 45-8. Presumptive southern tick-associated rash illness. **A,** Rash on patient's back. **B,** Close-up view of rash. (Courtesy Shannon B Snyder, MD.)

numerous species of *Borrelia*, there are numerous strains of each species, and ongoing research is likely to lead to discovery of more. *B. burgdorferi* is the primary agent in the United States, Europe, and Asia. U.S. isolates of *B. burgdorferi* demonstrate regional genetic heterogeneity,[233] and genetic comparison of European and North American strains indicate common ancestry.[272] *Borrelia garinii* and *B. afzelii* are causative agents of Lyme disease in Europe, and still more agents are being identified.[372] *Borrelia burgdorferi* has been isolated in Morocco from *I. ricinus* ticks.[302] The newly found *Borrelia bissettii* (formerly *Borrelia* genomic group DN127) has been found in California, Florida, and other southeastern states and in Europe, and it is responsible for Lyme disease in those areas and most likely other areas yet to be discovered.[271,306,364] Overall, *Borrelia* species are found nearly across the globe, and new species and geographic areas are being discovered regularly.

Epidemiology. Lyme disease surveillance began in 1982 at the CDC, and Lyme disease became a nationally reportable disease in 1991. In 2000, Lyme disease accounted for more than 90% of all reported cases of vector-borne illness in the United States.[159]

In the United States in 2001, 17,029 cases were reported in 43 states; in 2002, there were 23,763 cases from 48 states and the District of Columbia, or a 40% increase.[67] The national incidence of Lyme disease is 8.2 cases per 100,000 population, although 12 states reported 95% of the cases. Columbia County, New York, reported the highest incidence of Lyme disease in both 2001 and 2002.[67] Reasons for recent increases in the number of reported cases are multifactorial and most likely include public and physician awareness, increases of reservoir host populations, increased range of the vectors, efflux of people from urban cores to suburban areas (which results in more human–host–vector interfaces). Lyme disease is grossly underreported in many areas.[55,73,239]

Patients of any age may become infected, but the incidence has two peaks: among children aged 5 to 14 years, and among adults 50 to 59 years of age. Fifty-nine percent had an onset of illness in June and July, and 78% of the cases occurred in the months of May through August. Fewer than 7% of the cases began in winter months.

Lyme disease is a worldwide problem, diagnosed or found on all continents. Sporadic cases in Australia have yet to be proven as having originated in Australia and may have been imported in patients who spent time in areas endemic for Lyme disease.[168,298] Polymerase chain reaction (PCR) analysis of approximately 12,000 field-captured ticks in Australia failed to find any evidence of *Borrelia*-like infection.[299]

The primary vectors for Lyme disease worldwide are ticks of the *Ixodes ricinus* complex. In the eastern half of the United States, the primary vector is *Ixodes scapularis*, known more commonly as the black-legged tick or deer tick. The primary vector in the western United States is *Ixodes pacificus*, the western black-legged tick.[258] In Europe, Asia, and Northern Africa, *I. ricinus*, the castor bean tick, is the main vector. In Japan, China, and eastern Russia, it is *Ixodes persulcatus*, the taiga tick. Transmission of the spirochetes to humans is almost exclusively by infected nymphs or adults. Larvae are usually not infected until they feed on an infected host, pick up the spirochetes from this host, and then become infective as a seeking nymph. Unlike in many other tick-borne diseases, there is minimal transmission of the causative spirochetes from an infected female to her eggs.[256,269]

Transmission of the disease in the United States outside of the areas in which *I. ricinus* complex ticks are found, although rare, does occur, thus implicating other vectors. Several other tick species (*Amblyomma, Dermacentor, Haemaphysalis, Rhipicephalus*, and other *Ixodes* species) have been found infected with the spirochetes, but they are not successful in transmitting the spirochetes to a host.[220] Various nontick arthropods, including some biting flies, mosquitoes, and the cat flea, have been shown to harbor spirochetes, but they do not transmit spirochetes to a host or do so very rarely.[216,220,285,352] In many of the nontick arthropod cases, disease transmission is presumed to take place by a "dirty syringe" type of mechanism, in which the vector feeds partially on a spirochete-laden host, only to momentarily later inject the spirochetes into the next host.[216,221]

Reservoir host species are numerous, and they vary worldwide depending on the location and climate. At least 125 species

of mammals, birds, and reptiles are important hosts to *Ixodes* ticks throughout the world.[186] In the northeastern United States, the preferred host for *I. scapularis* larvae and nymphs is the white-footed mouse, *Peromyscus leucopus*. In Europe and Asia, various rodents are the primary hosts for the larvae and nymphs. Larger mammals usually serve as hosts for the adults. In more temperate areas of the world, there is a much wider variety of hosts, including reptiles, birds, and mammals.

In the southeastern United States, the cotton mouse (*Peromyscus gossypinus*), the cotton rat (*Sigmodon hispidus*), and other mammals, reptiles, and birds serve as primary hosts for larvae and nymphs.[101,186,257] Bird species readily harbor spirochetes, whereas lizard species do not. The reptilian hosts, almost exclusively lizards, account for a large amount of the immature (larval and nymphal) tick population.[257] Lizard sera and mule deer sera appear to deactivate spirochetes via a borreliacidal protein.[200,360] The high numbers of ticks parasitizing lizards instead of mice, and the lizards' apparent inability to harbor the spirochetes, may partially account for the differences in Lyme disease incidences between certain geographic areas.[74]

The migration of larger mammalian and avian hosts is one means by which ticks spread into new areas, often spreading various diseases with them.[8,79,189] Host populations increase when appropriate predators are absent; these fluctuations can amplify tick populations tremendously. One deer can host several thousand ticks of various stages, and deer population studies have shown that decreasing or eradicating deer populations leads to marked decreases in tick populations in subsequent years.[133,276]

The interaction between spirochete and host begins when the spirochete enters the salivary gland of the tick during the feeding process. Transmission to another vertebrate also occurs during the feeding process, which explains why disease transmission usually occurs only after a tick has been on a host for 48 to 72 hours. *B. burgdorferi* spirochetes show no transmission to mice in less than 24 hours, with maximal transmission occurring between 48 to 72 hours.[90] Another study showed that tick attachment for greater than 72 hours was more likely to transmit disease.[321]

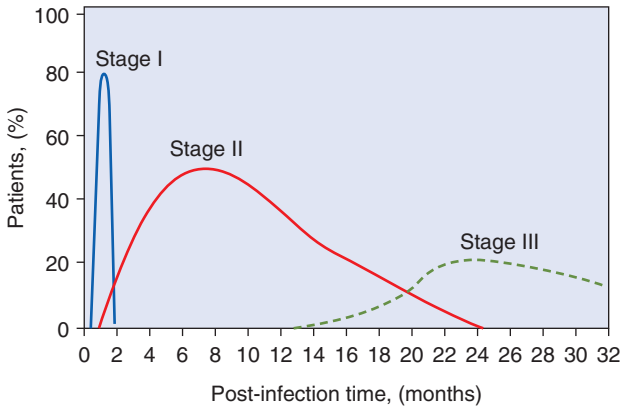

Figure 45-9. Time course and frequency of symptoms in Lyme disease. Approximately 50% of victims report recent tick bite. The initial stage I symptoms occur in approximately 70% to 80% of infected individuals. Weeks after the initial tick bite the skin, nervous system, and joints are most often affected as the spirochete spreads hematogenously. Stage II symptoms typically begin 4 to 10 weeks after the initial infection and occur in approximately 50% of untreated patients. Stage III typically starts much later, often 11/2 to 2 years and rarely more than 5 years after initial infection. (Modified from Schmid GP: Rev Infect Dis 11[Suppl 6]:S1460–1469, 1989.)

Box 45-1. Lyme Disease: Stage I

Incubation: 7–10 days (range, 3–32 days)
Duration: 28 days (range, 1–14 months)

SYMPTOMS (INCIDENCE)
Erythema migrans (60%–89%)
Mild lymphadenopathy (23%)
Low-grade fever (19%–39%)
Mild fatigue and malaise (54%)
Neck stiffness (35%)
Mild arthralgia and myalgia (44%)

Clinical Manifestations. Lyme disease has a myriad of constitutional, dermatologic, neurologic, cardiac, and musculoskeletal symptoms. The most common are vague myalgias, headache, fatigue, neck stiffness, and arthralgias. Rarely, gastrointestinal complaints or ocular complaints such as conjunctivitis, iritis, and keratitis may be reported. Collectively, these symptoms are difficult to distinguish from a benign viral syndrome, which is one of the reasons Lyme disease is easily misdiagnosed by both patients and physicians.[14]

Lyme disease is multisystemic and multiphasic. The disease is classically divided into three stages based on the chronologic relationship of symptoms to the original tick bite, with different clinical manifestations at each stage (Fig. 45-9). There is no clear delineation distinguishing one stage from another, however, and signs and symptoms of different stages often overlap.

The disorder usually begins as a localized infection with constitutional findings and associated symptoms (stage I) (Box 45-1).[129,253] Within days to weeks, spirochetes may disseminate through blood or lymph, and neurologic, cardiac, or joint abnormalities may develop (stage II). Finally, chronic, persistent infection of the joints, nervous system, skin, or eyes may occur months or years later (stage III).

Although the exact roles of the infecting spirochete, spirochetal antigens, and host immune responses are unclear, tissue invasion and persistence of infection probably cause many of the later manifestations of Lyme disease. This concept is supported by the isolation of *B. burgdorferi* from blood, skin lesions (erythema migrans), and CSF.[27,338] The organism has also been identified in the eye,[335] myocardium,[224] and synovium.[317] Patients at all stages of disease respond to appropriate antimicrobial therapy, and early treatment generally leads to an excellent long-term prognosis.

Early Localized Disease (Stage I). The ability of patients to remember a tick bite varies, frequently by species of tick, as some bites are more painful than others. Early localized disease typically begins as a localized erythema migrans rash or lesion, which occurs 7 to 10 days (range, 3 to 32 days) after a tick bite. Approximately 75% of patients with Lyme disease develop an erythema migrans lesion.[67,137,198,249] In one prospective study in New England, erythema migrans was reported in almost 90%

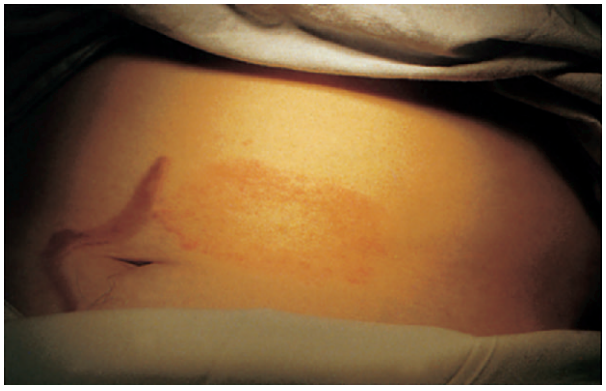

Figure 45-10. Erythema migrans rash of Lyme disease in a pregnant woman. (Courtesy Paul Auerbach, MD.)

of patients with Lyme disease. A large number of these patients were children, however, who often have a greater febrile and dermatologic response.[60,129,130,330] There are small regional variations in the development of erythema migrans, which may be due to infection with agents other than *B. burgdorferi*, such as *B. lonestari* or a similar agent.[117,231,232,329] Erythema migrans may appear anywhere on the body but usually occurs at or near the site of the tick bite. In cases with a single erythema migrans lesion, the most common sites include the head and neck region (26%), extremities (25%), back (24%), abdomen (9%), axilla (8%), groin (5%), and chest (3%)[129] In Europe, up to 18% of patients present with disseminated or multilocular erythema migrans.[198]

The erythema migrans rash is variable in size, ranging from 2 cm to over 60 cm in diameter, and is usually in a circular pattern. To meet the CDC case definition of Lyme disease, the lesion must be at least 5 cm. It usually begins as a red macule or papule, with an area of central clearing that becomes more apparent as the lesion expands in size (Fig. 45-10). The central region may become indurated and, rarely, necrotic. The borders, which are usually bright red, may expand as much as 1 cm/day. These borders are usually flat, although rarely they may be raised or indurated. Occasionally, there are multiple, alternating concentric rings of erythema and clearing, a rash pattern referred to as "bulls-eye." The rash is usually warm to the touch.

The lesions are sometimes difficult to differentiate from local immune reactions to the tick salivary proteins, and they are sometimes confused with secondary bacterial cellulitic reactions. Local immune reactions usually occur within hours of the tick bite and are very pruritic. Secondary cellulitic reactions typically occur within a few days of the tick bite and lack the central clearing and rapid expansion.

Patients often describe the lesion as burning but may also report itching or pain. Children may develop temperatures to 104°F (40°C), but low-grade fevers are more common in adults. Constitutional symptoms, such as malaise and myalgias, may also be present. Generalized lymphadenopathy is rare, but local lymphadenopathy may occur.

Erythema migrans fades after an average of 3 to 4 weeks (range, 1 to 14 weeks) without treatment; with antibiotics, the lesion resolves after several days and seldom recurs.[337] Although the erythema migrans lesions do resolve without treatment, untreated patients are at risk for developing early (stage II) or late (stage III) disseminated disease.

Early Disseminated Disease (Stage II). Untreated Lyme disease may enter the second stage, early disseminated disease, within days to weeks after the initial infection. The spirochetes spread from the skin to other organs via blood or the lymphatics. Although organisms can be visualized in peripheral blood smears, they are very difficult to culture from blood. *B. burgdorferi* has a preference for certain tissues, but it can be found in nearly any organ.[332]

Dermatologic. Disseminated erythema migrans is the most common manifestation of stage II disease. These secondary lesions are usually smaller, there are usually many of them, and they may lack the central clearing. They may appear anywhere on the body, with the exception of the palms and soles. Ten percent to 15% of patients have more than 20 such lesions; rarely, there are more than 100. Blistering and mucosal involvement do not occur—an important feature in differentiating these lesions from erythema multiforme. Other skin manifestations include a malar rash (10% to 15%) and (rarely) urticaria.[337]

Borrelial lymphocytoma, an unusual dermatoborreliosis, appears in Europe and western Asia in approximately 1% of patients. Patients typically present with a firm nodule that is red, purple, or blue and one to several centimeters in diameter. It is usually solitary, but multiple lesions may occur in approximately 25% of cases. It often occurs in areas of cooler body temperature, with approximately 80% on the nipple or areola, 10% on the earlobe, and 10% on other areas (including the scrotum).[223] It is caused by *Borrelia afzelii* in approximately 90% of cases, although it has been seen with *B. bissettii* and *B. garinii*.[223] Histologically, it consists of cutaneous lymphoid hyperplasia, usually in deep portions of the dermis. It may be confused with well-differentiated nodular lymphoma.[249] There is associated pruritus, but otherwise it is benign and resolves with appropriate antibiotic therapy.

Neurologic. Neurologic manifestations occur in 10% to 40% of untreated or improperly treated patients.[261] Meningitis, encephalitis, and meningoencephalitis are the major neurologic findings of early disseminated disease; lethargy, forgetfulness, disorientation, somnolence, dizziness, photophobia, and incoordination are the most common signs and symptoms. Mood disturbances may also be seen, especially in pediatric patients.[26] Nuchal rigidity is sometimes present, along with a severe headache. Lumbar puncture reveals CSF with pleocytosis (usually less than 1000 cells/mm^3), typically with lymphocyte predominance and high protein level.

Paralysis of the facial nerve (cranial nerve VII) may occur and persist for months, and it usually resolves spontaneously.[70,264] Bilateral facial nerve paresis or paralysis may occur in 25% of cases with cranial nerve VII involvement, usually evolving asymmetrically. Bilateral facial palsy is an unusual physical finding and should suggest Lyme disease, although this finding may also be caused by sarcoidosis, herpes zoster, or the human immunodeficiency virus (HIV).

Radiculoneuronitis may be present in various nerves, causing burning, stabbing, or shooting pain. Large nerve bundles such as the brachial plexus are more commonly involved. Mononeuritis multiplex may be present, with peripheral motor weakness and sensory pain. Other neurologic findings are rare but include pseudotumor cerebri, chorea, and dementia.[128,173a,287]

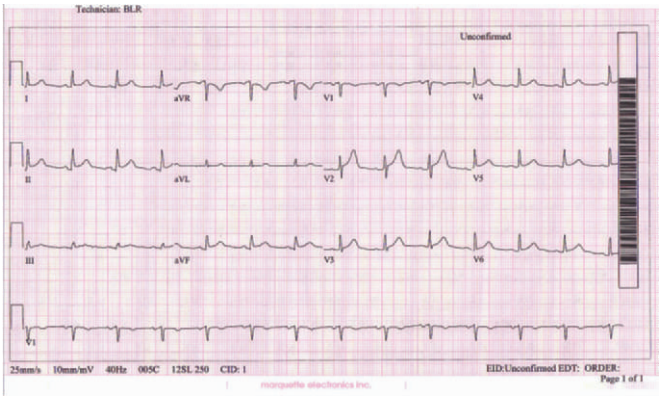

Figure 45-11. An electrocardiograph of a patient with an acute myopericarditis.

Gastrointestinal. Ten percent of patients may develop hepatitis, hepatomegaly, and generalized abdominal pain. Hepatic involvement does not progress to hepatic failure; symptoms usually resolve in less than 1 week but may persist for several weeks. Splenomegaly may also occur.[337]

Cardiac. Untreated or improperly treated Lyme disease leads to cardiac manifestations in up to 10% of cases.[333] Although cardiac involvement is usually self-limited and rarely life-threatening, temporary intervention is required in some cases.[151,185] The most common cardiac manifestation of Lyme disease is conduction delay, which may be of any degree and is found in 80% of cases with cardiac involvement.[259] Electrophysiologic studies indicate that the block is at the level of the atrioventricular node. The block does not respond to atropine, suggesting a direct effect on the node.[259,293] In cases of complete heart block, the presence of spirochetes can be confirmed in cardiac tissue by cardiac biopsy. Noninvasive imaging modalities such as cardiac MRI can be used to document improvement in response to antibiotics.[85,185]

Conduction delays usually, but not always, resolve with appropriate antibiotic therapy. Patients with first-degree atrioventricular block and a PR interval exceeding 300 msec, as well as all patients with second- and third-degree heart block, should be admitted for observation and antibiotic treatment. Patients with third-degree heart block require temporary transvenous pacing and may require permanent pacemaker implantation.

More diffuse cardiac involvement, such as myopericarditis, is found in about half of patients with cardiac involvement. The electrocardiograph (ECG) in such patients usually demonstrates diffuse, nonspecific changes (Fig. 45-11). Radionuclide angiography may demonstrate left ventricular dysfunction, but this is seldom clinically significant. Cardiomegaly due to Lyme disease rarely occurs in U.S. patients, although dilated cardiomyopathy is often reported in Europe.[320,328] Valvular involvement is uncommon.

At least one death has been linked to cardiac involvement,[224] but abnormalities usually resolve completely, often within 1 to 2 weeks.[333] Severe cardiac complications have been described in pediatric patients but are relatively rare.[129]

Rheumatologic. Monoarticular arthritis develops in 60% of patients with erythema migrans who are not treated, usually occurring between 4 weeks and 2 years after the onset of illness.[342] Although common in North America, arthritis is rarely a finding in other areas of the world.[69,332]

In adult patients, the arthritis may occur in a migratory pattern, involving many joints, usually one joint at a time. Joint involvement typically occurs for about a week (median, 8 days), but it may persist for months. Many patients have repeated episodes of arthritis, and each subsequent recurrence is less severe. Soreness often persists between episodes, never fully resolving, and persistent morning stiffness is common. The knee is most commonly affected, followed by the shoulder, elbow, temporomandibular joint, ankle, wrist, hip, metatarsals, and metacarpals.[383] Joint swelling is not common except in the knee, and the joints are typically described as more sore than painful. Chronic effusions often occur.

In pediatric patients, the arthritis appears an average of 4.3 months after initial infection, with a broad range of a few days to 20 months.[129,130] Approximately 50% of pediatric patients have multiple recurrences, although complete remission between episodes is typical and chronic arthritis is uncommon. Ninety percent of children in a large study had arthritis in at least one knee.[130]

Synovial fluid of involved joints has a median WBC count of 25,000/mm^3, with a polymorphonuclear leukocyte predominance. Synovial membrane biopsy shows cellular hypertrophy, vascular proliferation, and mononuclear cell infiltrate.[336,340] PCR testing of synovial fluid is positive in 85% of cases of Lyme arthritis.

The organism may also persist in synovial tissue. Four patients who completed antibiotic therapy for Lyme arthritis and who subsequently had negative PCR testing of synovial fluid had positive PCR testing of the synovial membrane.[273] This persistence may account for the reactivation of Lyme arthritis that has been described after autologous chondrocyte transplantation for degenerative joint disease or cartilage injury.[229]

Late Disease (Stage III). Late disease may occur a year or more after the initial presentation, and it usually has rheumatologic, neurologic, and dermatologic manifestations. Recurrent hepatitis, eosinophilic lymphadenitis, acute respiratory distress syndrome (ARDS), and other rare manifestations may also be present.

Rheumatologic. Arthralgias and oligoarthritis occur in approximately 50% of untreated patients.[339] In about 10% of patients, cartilage loss, subarticular sclerosis, osteophyte formation, cortical or marginal bone erosions, joint effusions, and other radiographic signs of degenerative arthritis may be seen.[205] The rheumatologic workup, including erythrocyte sedimentation rate, C-reactive protein (CRP), antinuclear antibodies, and rheumatoid factor, is generally negative.[339] Interestingly, late rheumatologic disease is rare in children.[389]

Neurologic. Central nervous system (CNS) involvement may develop months to years after the initial infection. Patients most typically present with progressive encephalomyelitis, with ataxia, cognitive impairment, spastic paresis, and involvement of cranial nerves VII and VIII.[2] Rarely, strokes, seizures, and dementia may occur.[111] Lumbar puncture in patients with stage III CNS involvement reveals CSF with a lymphocytic pleocytosis and anti–*B. burgdorferi* antibodies. MRI of the brain reveals white matter changes consistent with encephalopathy. In seropositive pediatric patients, the meningoencephalitic changes are often seen as behavioral changes and are noted more often in boys than girls.[26]

Peripheral nervous system involvement may manifest as peripheral paresthesias, painful radiculopathies, and motor nerve palsies. The underlying pathophysiologic mechanism appears to be mononeuritis multiplex.[119] Such patients may present with radicular pain that is frequently asymmetrical, originating in the spine and referring to the extremities. Nerve conduction and electromyographic studies show mild axonal polyneuropathy with decreased motor or sensory nerve conduction velocities, with denervation of spinal and limb muscles.[119]

Dermatologic. Patients with late-stage dermatologic involvement present with acrodermatitis chronica atrophicans, also called diffuse idiopathic cutaneous atrophy. It is found almost strictly in central and southern Europe and is caused by *B. afzelii*. It may persist for many years.[254] The dermatitis usually occurs on the distal extremities, often having started at a previous area of erythema migrans, commonly on the extensor surfaces of the extremities. It begins as inflammation, hyperpigmented with red or blue, and is followed by scarring and atrophy of the skin. The atrophy causes the skin to thin, and it later becomes hypopigmented. Hair loss and dysfunction of sweat and pilosebaceous glands also occurs.[249] Other findings, such as polyneuropathy, may accompany these skin changes. *Borrelia* has been cultured from 10-year-old lesions of acrodermatitis chronica atrophicans. Histologically, fibrosis occurs early, the glands become infiltrated, and the dermis reveals a patchy to bandlike mononuclear infiltrate composed of lymphocytes, histiocytes, and plasma cells in greater numbers than found in typical erythema migrans lesions.[249]

"Chronic Lyme Disease." There is considerable debate over whether a chronic form of Lyme disease exists. A collection of symptoms, lumped into a category sometimes referred to as postborreliosis syndrome or post–Lyme disease syndrome (PLDS), consists of signs and symptoms such as persistent fatigue, sleep disorders, depression, cognitive defects, mood swings, and other neuropsychiatric manifestations.[214] This symptom complex fails to respond to traditional antibiotics.

This collection of symptoms might be an entirely separate disease, or partially treated Lyme disease, or the disease complicated by the cystic form, which is difficult to treat. As early as the early 1900s, spirochetes were noticed to have developed into cysts, also called blebs or vesicles. Today, these cysts are called spherocytes, or the L-form of the spirochete.[45] These spherocytes are induced under adverse conditions, such as the presence of antibodies targeted against the spirochetes, adverse pH, or adverse temperatures. Thus, the spherocytes may be responsible for the spirochetes' surviving conditions that would kill or deactivate the typical spirochete form.[251] When environmental conditions again become favorable for the spirochetes, the cysts or spherocytes convert back to the traditional spirochete form.[38]

Further research is likely to yield more information about the appropriate treatment of the cystic form of the disease, such as the addition of antiprotozoal medications to the treatment regimen.[39–41] Patients afflicted with these persistent symptoms are very active in pursuing formal recognition and treatment of these disorders through political, legislative, and research means.

Lyme Disease in Pregnancy. Fetal demise may result in mothers who develop Lyme disease in pregnancy.[218,228,304,374] Transplacental transmission of Lyme disease is rare but appears to be associated with an increased risk of cardiac malformations.[382]

Lyme disease in pregnancy should be aggressively treated. A 1996 prospective study of 58 pregnant women with erythema migrans given oral or intravenous (IV) penicillin or ceftriaxone reported only seven adverse outcomes, none clearly associated with Lyme disease.[222] The American College of Obstetrics and Gynecology recommends oral penicillin or amoxicillin for 3 weeks in pregnant women with cutaneous involvement or recent tick bites in endemic areas. For patients with severe acute disease, or disseminated or late disease, 3 weeks of parenteral penicillin is recommended. If the patient is allergic to penicillin, parenteral erythromycin is recommended.[10,105] Tetracyclines should not be used in pregnancy.

Diagnosis. The CDC has established a surveillance case definition for Lyme disease. This case definition requires exposure to ticks in an endemic county *and* an erythema migrans lesion, diagnosed by a physician, that is larger than 5 cm. In this definition, exposure requires outdoor activities within 30 days of the onset of the lesion. An endemic county is one in which at least two confirmed cases of Lyme disease have been acquired, or known tick vectors are established. It is important to note that Lyme disease case definitions are designed for national reporting, not individual patient diagnosis. The clinical diagnosis of Lyme disease is based on history, clinical suspicion, and a working knowledge of the disease process and epidemiology; laboratory tests may be performed but generally serve only to confirm the presence of the disease.

Routine laboratory examinations may reveal nonspecific findings, such as elevated liver enzymes (alanine transaminase [ALT], aspartate transaminase [AST], and/or gamma-glutamyltransferase [GGTP]) decreased leukocyte count, and elevated erythrocyte sedimentation rate. Immunoglobulins A and G (IgA and IgG) are usually normal, whereas IgM is usually elevated, especially in severe disseminated disease. Culture is the gold standard for diagnosis but is often difficult to obtain. Biopsy of an erythema migrans skin lesion is positive in 60% of punch biopsies from the leading edge of the lesion, and this figure may be as high as 80% in specialty centers.[31] The production of anti–*Borrelia burgdorferi* antibody develops slowly, and serologic assays in the first 3 to 6 weeks of illness are often negative unless severe disease is present.[242,312] In some patients, treatment with antibiotics may blunt or entirely suppress the antibody response; in others, IgG and IgM antibodies may persist for years after resolution of clinical symptoms.[112] Patients with complicated Lyme disease (with neurologic, cardiac, or joint involvement) or those in remission are more likely to have elevated specific antibody titers.[242,312] Serologic testing has the highest predictive value in patients with a pretest probability of having Lyme disease between 20% and 80%.[358] Patients from endemic areas with an 80% or higher pretest probability of having the disease benefit from empiric treatment, not testing. Patients with a pretest probability of less than 20% have a high likelihood of a false-positive testing.

Indirect immunofluorescent assay (IFA), enzyme-linked immunosorbent assay (ELISA), and Western blot test are the most commonly used tools to detect antibodies to *Borrelia burgdorferi*. For diagnostic purposes, ELISA appears to be the most accurate, with 89% sensitivity and 72% specificity. Low levels of agreement are common between various laboratories

and individual assays.[308] Western blot has proven to be a poor screening tool. New tests using recombinant proteins or synthetic peptides as ELISA antigens yield detection rates for serum antibodies of 20% to 50% for early disease, 70% to 90% for early disseminated disease, and nearly 100% in late disease.[384] Although Western blot is a poor screening tool, it is useful to confirm a positive ELISA result.

Only positive serologic assays confirmed by Western blot meet the criteria for laboratory diagnosis, according to the CDC national consensus panel. Many disease states, including other treponemal diseases, autoimmune diseases, and some viral diseases, may give false-positive ELISA results.[242] Antibodies directed toward oral flora may also cause false-positive results.[9,242]

A current research tool, reverse transcriptase DNA PCR, is both highly sensitive and specific for Lyme disease. It may eventually find a place in the diagnostic armamentarium.[110]

Patients with vague nonspecific symptoms such as chronic fatigue, musculoskeletal pains, and other neuropsychiatric signs and symptoms, in the absence of true Lyme disease signs and symptoms, should, in general, not undergo serologic testing for Lyme disease. Positive results in such patients are probably falsely positive and may create an inordinate amount of confusion and conflict.

The importance of appreciating the limitations of serologic testing cannot be overstated. In one study, only 23% of patients referred to the Lyme Disease Clinic at New England Medical Center had active Lyme disease, and a majority of those without Lyme disease had been treated inappropriately with antibiotics.[340] In this study, the most common reason for lack of response to antibiotics was misdiagnosis. The limitations of laboratory testing in Lyme disease include lack of sensitivity and specificity of serologic tests and considerable interlaboratory and intralaboratory variability in test results.[308] The persistence of antibodies in patients with past or asymptomatic infection with *B. burgdorferi* also complicates serologic testing. If another illness develops, as occurred in 20% of patients referred to the New England Medical Center clinic, it may be incorrectly attributed to Lyme disease. This is particularly problematic in patients with nonspecific symptoms of chronic fatigue or fibromyalgia, in whom the predictive value of a positive ELISA is low.

Overdiagnosis of Lyme disease has significant health system and patient care implications. In another study of 209 individuals referred to a university-based Lyme disease clinic with a diagnosis of the disease, only 21% met criteria for active Lyme disease, 60% had no evidence of current or previous infection, and 19% had evidence of previous but not active disease. The 79% of patients without active Lyme disease displayed significant anxiety and stress related to their diagnosis of Lyme disease, used considerable health care resources, and had frequent adverse antibiotic reactions.[286]

Treatment. Although most manifestations of Lyme disease resolve spontaneously without treatment,[301] treatment of Lyme disease with appropriate antibiotics hastens recovery in all stages of the disease. Both in vitro and in vivo studies have confirmed that *B. burgdorferi* is highly sensitive to tetracyclines, aminopenicillins, ceftriaxone, and imipenem.[158,176–178] The macrolides, oral second- and third-generation cephalosporins, and chloramphenicol all seem to have equal efficacies.[4,176] Although fluoroquinolones have traditionally not been effective against *Borrelia* species, the fourth-generation fluoroquinolone antibiotics seem to have good efficacy in vitro.[193] Table 45-3 lists general antimicrobial therapy options and recommendations for Lyme disease.

In early localized Lyme disease (stage I), amoxicillin and doxycycline are the drugs of choice. Tetracyclines should not be used in pregnancy, in lactating women, or in children under age 9. The recommended treatment duration is 14 to 21 days, but this may be extended to 4 weeks if symptoms persist or recur. Medications that will penetrate the CSF are ideal and may lead to less disease recurrence.[215] Sequestering of spirochetes in the brain may account for some apparent antibiotic failures, as not all antibiotics cross the blood–brain barrier.

Within the first 24 hours of treatment with antibiotics, up to 15% of patients develop a Jarisch-Herxheimer reaction (JHR), which manifests as a rise in temperature, vasodilation, and hypotension.[254] Normal saline infusion around the time of initial dosing of antibiotics may help reduce the degree of hypotension. In rare cases, JHR may be severe and fatal. Symptoms rarely persist beyond 24 hours.

Disseminated disease (stages II and III) should be treated according to severity. In mild disseminated disease (such as secondary erythema migrans or cranial nerve VII palsy), 3 weeks of oral doxycycline is as effective as 2 weeks of parenteral ceftriaxone.[81] In patients initially treated with oral antibiotics who are therapeutic failures or in whom disease recurs, parenteral antibiotics are indicated. Patients with severe disseminated disease (neuroborreliosis, high-degree heart block) require parenteral antibiotics for up to 4 weeks.[80,213] Ease of once-daily administration of ceftriaxone makes it a better choice for both inpatient and outpatient administration than other typically used agents requiring administration three or four times daily.[80,81]

Neurologic symptoms begin to improve with therapy within 1 week, although full resolution may take 7 to 8 weeks.[341] Prolonged oral antibiotics are often necessary to treat stage III arthritis, sometimes for as long as 4 to 8 weeks.[341] Delayed clinical response in arthritis is not uncommon. Nonsteroidal anti-inflammatory drugs (NSAIDs) may also reduce symptoms.

No vaccines are presently available for the prevention of Lyme disease in humans. A vaccine based on recombinant outer surface protein A (OspA) was approved in 1998 but was removed from the market by the manufacturer in 2002. The vaccine had limited efficacy, required frequent boosters, was expensive, could not be used in children, and resulted in an apparent immunogenic reaction in some vaccinated individuals.[152] Future vaccines, although still in their infancy, will most likely use polyvalent outer surface protein C (OspC).

Treatment of asymptomatic tick bites in patients who are in areas endemic for Lyme disease is controversial, although this does not stop patients from requesting care. In one survey, physicians in Maryland reported seeing 11 times as many patients seeking help after "tick bites" as they did actual cases of Lyme disease.[73] Three randomized, placebo-controlled studies have addressed antibiotic prophylaxis for tick bites.[5,72,310] Of patients in the placebo groups, 1% to 3.4% developed Lyme disease or had evidence of seroconversion, even though 15% to 30% of ticks were infected; no patients in the treatment groups developed Lyme disease. A meta-analysis of the three studies concluded that there was no significant difference between the groups, and that routine prophylaxis of tick bites is not warranted, even in endemic areas.[373]

TABLE 45-3. Antimicrobial Recommendations for Lyme Disease

DRUG	DOSE[¶]	DURATION
Early Localized Disease		
Adults		
Tetracycline	250–500 mg PO qid	14–28 days
Doxycycline	100 mg PO bid	14–28 days
Amoxicillin	500 mg PO tid	14–28 days
Cefuroxime	500 mg PO tid	14–28 days
Children		
Amoxicillin	50 mg/kg/day PO divided tid	14–28 days
Doxycycline (over 8 yr)	100 mg PO bid	14–28 days
Erythromycin	30–50 mg/kg/day PO divided qid	14–28 days
Tetracycline (over 8 yr)	250 mg PO qid	14–28 days
Cefuroxime axetil	30–40 mg/kg/day PO divided bid	14–28 days
Early Disseminated and Late Diseases,* and Late Disseminated Neurologic Disease[†]		
Adults		
Ceftriaxone	2 g IV qd	14–28 days
Cefotaxime	2 g IV q8h	14–28 days
Penicillin G	20 million IU/day IV divided q4h	14–28 days
Children		
Ceftriaxone	75–100 mg/kg/day IV qd	14–28 days (max, 2 g/day)
Cefotaxime	90–180 mg/kg/day IV q8h	14–28 days
Penicillin G	300,000 U/kg/day IV q4h	14–28 days (max, 20 million U/day)
Late Disseminated Arthritis		
Adults		
Amoxicillin	500 mg PO qid	28 days
Doxycycline	100 mg PO bid	28 days
Ceftriaxone	75–100 mg/kg/day IV qd	14 days (max, 4 g/day)
Children		
Amoxicillin	50 mg/kg/day PO divided tid	28 days
Ceftriaxone	80–100 mg/kg/day IM/IV divided bid–qd	14 days (max 2 g/day)
Disseminated Disease and Carditis		
Adults		
Doxycycline[‡]	100 mg PO bid	21–28 days
Amoxicillin[‡]	500 mg PO q8h	21–28 days
Ceftriaxone[§]	2 g/day IV qd	14–28 days
Children		
Ceftriaxone	75–100 mg/kg IV/IM qd	14–21 days (max, 2 g/day)
Penicillin G	300,000 U/kg/day IV divided q4h	14–21 days (max, 20 million U/day)

*For multiple erythema migrans, use treatment for early localized disease for at least 21 days. For isolated facial palsy, use treatment for early localized disease for at least 21 to 28 days.
[†]Neurologic involvement limited to an isolated facial palsy should be treated as early disease.
[‡]For mild cardiac involvement (i.e., first-degree atrioventricular block with PR interval less than 0.30 second).
[§]For second- or third-degree heart block, although no evidence indicates that intravenous is better than oral regimens.
IM, intramuscularly; IV, intravenously; PO, orally; bid, twice a day; tid, three times a day; qid, four times a day; qd, every day; q4h or q8h, every 4 or 8 hours.
[¶]Total daily dose shown to be divided (if necessary) as indicated.

Many physicians do not adhere to this recommendation, however. Maryland physicians ordered serologic testing in two thirds of patients with asymptomatic tick bites and treated more than half with prophylactic antibiotics.[123] The concern often cited for such practices is that patients with tick bites who become infected may not develop erythema migrans (when the disease is easily treated) but go on to develop the late and more serious manifestations of Lyme disease.

The risk of serious late sequelae in untreated patients with a tick bite, however, is extremely low, for three reasons. First, less than 5% of those bitten by the *I. scapularis* tick in endemic areas become infected. Second, most patients with an identified tick bite remove the tick before it has been attached for the 36 to 48 hours required to transmit an infectious inoculum of *B. burgdorferi*. Finally, 80% to 90% of the few patients who become infected develop erythema migrans, which makes diag-

TABLE 45-4. Tick-Borne Relapsing Fever

ARTHROPOD VECTOR	BORRELIA SPECIES	GEOGRAPHIC DISTRIBUTION
Ornithodoros hermsii	*B. hermsii*	Western United States and Canada
O. turicata	*B. turicatae*	Southwestern United States and Mexico
O. parkeri	*B. parkeri*	Western United States and Mexico
O. moubata	*B. duttonii*	Tropical Africa
O. tholozani	*B. persica*	Central Asia, Middle East, Greece
O. tartakovskyi	*B. latyschevi*	Iran, central Asia
O. erraticus	*B. crocidurae*	Russia, Middle East, East Africa, Turkey
O. graingeri	*B. graingeri*	Kenya
O. talaje	*B. mozzottii*	Mexico, Central America
O. rudis	*B. venezuelensis*	Central and South America
O. asperus	*B. caucasica*	Iraq, Russia
O. marocianus	*B. hispanica*	Northern Africa, southern Europe

nosis and treatment easy. Physicians must therefore weigh a number of factors related to the likelihood of disease acquisition in deciding whether to treat asymptomatic tick bites with antibiotics. These factors include species and stage of the offending tick, duration of attachment, geography, and patient factors such as pregnancy. If a decision is made to treat, reasonable antibiotic choices include a 10-day course of either amoxicillin or doxycycline.

Overtreatment of presumed Lyme disease with antibiotics has significant healthcare ramifications. If the patient does not have Lyme disease, treatment failure is virtually ensured. Patients being treated for Lyme disease have a significant amount of anxiety regarding the diagnosis.[286] Adverse drug reactions may occur. Overuse of antibiotics has led to resistance to multiple antibiotics by many organisms, although resistance to tetracycline is not documented for *Borrelia*.[11]

Southern Tick-Associated Rash Illness (STARI)

Since the mid-1980s, erythematous rashes similar to those seen in early Lyme disease have been found in persons from the southeastern and south-central states, a geographic area in which the presence of Lyme disease is in dispute.[231] A correlation was noted between these rashes and the bite of the lone-star tick, *Amblyomma americanum*[172] (see Figure 45-6). A causative agent, *Borrelia lonestari,* has been discovered in ticks and in patients by detecting DNA from *Borrelia* in specimens[19] and by culturing the organism.[361,362] The disease has since been named southeastern tick-associated rash illness, or STARI, but it is also known as Master's disease and southern Lyme disease.[231]

A. americanum ticks are found throughout the southeastern and south-central United States, and along the eastern seaboard into Maine. All three stages of *A. americanum* feed aggressively on humans, and various stages are found in most of its distribution throughout the year.

Patients with the disease present with a rash similar to erythema migrans, although it often lacks the central clearing. Skin findings are followed by generalized fatigue, headache, myalgias, and other generalized symptoms. *A. americanum* ticks have proportionately large mouthparts (see Figure 45-6) and are very immunogenic; the rash is therefore sometimes difficult to distinguish from a local reaction.

Diagnosis at the present time is by gene amplification of *B. lonestari* using PCR. The disease is currently believed to be self-limited, although current recommendations are that it be treated with a course of an oral tetracycline. Research into STARI is in its infancy and will probably yield much more information over the next decade.

Tick-Borne Relapsing Fever

Tick-Borne relapsing fever (TBRF) is an acute borrelial disease characterized by recurrent paroxysms of fever, separated by afebrile periods. It occurs in both endemic and epidemic forms (Table 45-4). The endemic or sporadic form occurs worldwide and is caused by a group of closely related *Borrelia* species. The epidemic form of relapsing fever is transmitted by the human body louse and has not been reported in the United States in recent years.

The *Ornithodoros* ticks that transmit TBRF act as both vectors and reservoirs for *Borrelia* organisms; wild rodents also serve as reservoir hosts.[356] Ticks ingest *Borrelia* organisms while feeding on an infected vertebrate, most often a rodent. Borreliae enter the tick hemocele and then spread to other tick tissues, including the salivary glands, coxal organs, and reproductive organs. The coxal organs in argasid ticks are specialized for excretion of excess fluids and solutes accumulated during feeding. In some *Ornithodoros* species, the coxal fluid is released near the mouthparts during feeding, allowing transmission of spirochetes to vertebrate hosts. Transmission may also occur through saliva or regurgitated gut contents.[18] Borreliae remain infective within ticks for many months.[322]

Transovarial transmission allows all developmental stages to be potentially infective. The ticks generally feed at night and attach themselves to the host for a short time, usually less than 1 hour. The bite is seldom painful and frequently goes unrecognized. The ticks are extremely resilient and may survive for years between feedings.

A high degree of specificity exists between the major strains of *Borrelia* that cause relapsing fever and their associated tick vectors. For example, the three *Borrelia* species found in the United States—*Borrelia hermsii, Borrelia turicatae,* and *Borrelia parkeri*—show complete specificity for their respective tick vectors *Ornithodoros hermsi, Ornithodoros turicata,* and *Ornithodoros parkeri.* This specificity is used extensively in the classification of *Borrelia* species.[82]

Epidemiology. *Ornithodoros* ticks generally inhabit rodent burrows and nests, and cracks and crevices in human and animal habitats, caves, and similar locations. Habits and patterns of infection vary between tick species. In parts of Africa, ticks live in the dust and cracks of earthen-floored huts, and sporadic cases are seen throughout the year. In the Middle East, Mexico, and southwestern United States, ticks live in the guano on cave floors, and human infection is often associated with visiting or camping in caves.

The majority of cases of TBRF in the United States are attributed to *B. hermsii*. Its vector, *O. hermsii,* inhabits the coniferous forest biome, where it lives in remains of dead trees and burrows inhabited by mice, rats, and chipmunks. Ticks are carried by rodents into poorly maintained cabins and huts. Lodging in such shelters by hikers and hunters is a major factor in acquiring relapsing fever,[118,167] and rodent-proofing such shelters dramatically reduces the risk of acquiring TBRF.[262] Occasional cases are also caused by *O. turicata* (transmitting *B. turicata*) in Texas (associated with travel into caves) and adjacent areas of the Southwest; *O. parkeri* (transmitting *B. parkeri*) rarely bites humans.[165]

TBRF is found throughout most of the world, and endemic areas include Colorado,[104,167] California,[104] the Pacific Northwest,[118] southern British Columbia, plateau regions of Central and South America, central Asia, Mediterranean countries, and most of Africa. In California, where reporting of relapsing fever is encouraged, two to 12 cases are reported per year.[104] Large outbreaks have been reported from Spokane County in Washington,[353] Colorado,[356] and the north rim area of the Grand Canyon.[61] Between 1985 and 1996, the 285 cases of TBRF reported in the United States occurred in California, Colorado, Idaho, Texas, Washington, Arizona, New Mexico, Nevada, Oregon, Utah, and Wyoming.[356] Although the vast majority of cases occur in states where TBRF is endemic, a review of 450 cases of TBRF acquired in the United States from 1977 to 2000 noted that 7% of cases were diagnosed in states where it is not endemic.[102] TBRF is more common in men, presumably because of increased exposure to tick vectors, and occurs primarily during summer months. TBRF is rarely fatal in adults, but in infants less than 1 year of age, case fatality rates may be 20% or higher.[206,322]

Clinical Manifestations. *Ornithodoros* ticks feed for a relatively short period (1 hour), so most patients have no recollection of a tick bite. After an incubation period of about a week, patients develop the characteristic clinical features of TBRF: abrupt onset of a fever lasting about 3 days (range, 12 hours to 17 days), followed by an afebrile period of variable duration, then relapse with return of fever and other clinical manifestations. The initial febrile period terminates with rapid defervescence or "crisis," accompanied by drenching sweats and intense thirst. Febrile periods in TBRF are associated with spirochetemia. Resolution of fever occurs when the host develops an adequate antibody response to the spirochete. During afebrile periods, the spirochete remains "hidden" in organ tissue and undergoes antigenic conversion to a new serotype (see Antigenic Variation, next). Relapse occurs when the new serotype causes a new round of spirochetemia.

After being bitten by an infected tick, victims may develop a pruritic eschar at the site of the tick bite, but the lesion is usually absent by the time the clinical symptoms appear. Victims typically develop fever, frequently accompanied by shaking chills, severe headache, myalgias, arthralgias, upper abdominal pain, photophobia, cough, nausea, and vomiting. The temperature is usually greater than 102.2°F (39°C), and patients may manifest extreme muscular weakness and lethargy. Splenomegaly develops in approximately 40% and hepatomegaly in 18% of patients with TBRF. From 10% to 40% have neurologic involvement, approaching the incidence seen in Lyme disease. The most common neurologic complications are meningismus and facial nerve palsy.[51] Facial palsy, when present, typically occurs after the second febrile episode and usually resolves within 2 to 9 weeks with or without treatment. Other reported neurologic complications include neuropsychiatric disturbances, encephalitis, peripheral neuropathy, myelitis, and pathologic reflexes. Iritis or iridocyclitis occurs in up to 15% of untreated cases, typically occurring later in the fever course. Formation of adhesions between the iris and anterior lens capsule may lead to visual defects. A rash, which may consist of a macular eruption, petechiae, or erythema multiforme, develops in 25% to 30% of patients.[322]

Mortality rates have been as high as 40% in some epidemics of louse-borne relapsing fever,[313] but TBRF is generally self-limited. Only rarely are clinical features severe and prolonged. Neurologic complications generally resolve spontaneously, but severe depression may persist for months. Hemorrhagic complications, pneumonia, ARDS, hepatosplenomegaly, petechiae, or myocarditis may develop rarely.[83,118,377]

Relapsing fever in pregnancy results in a high incidence of spontaneous abortion,[322] premature birth, and perinatal morbidity.[181] Fetal death is probably caused by direct placental invasion by spirochetes, resulting in thrombocytopenia and retroplacental hemorrhage.[331] Prior studies have linked pregnancy with a more serious maternal disease course.[148,241,242] A more recent case-control study, however, found no significant difference in pregnant women's mortality or complications.[179] Infection in the neonatal period usually occurs by placental transmission and appears as overwhelming sepsis with very high mortality.[390]

Antigenic Variation. The phenomenon of relapse in TBRF is caused by the ability of borreliae to undergo antigenic variation in an infected host. The organisms are capable of spontaneous conversion to many serotypes. Clinically, defervescence occurs when the dominant serotype is eradicated by interaction with host antibody. Spirochetemia probably persists at undetectable levels during the afebrile period and consists of mixed serotypes. Relapse occurs when a variant population reaches detectable levels. Antigenic variation is under complex genetic control and does not appear to require contact of the organism with host antibody.[17,345]

Diagnosis. Clinical diagnosis of TBRF requires thorough knowledge of the epidemiology of the disease and a high index of suspicion. TBRF is uncommon and occurs only sporadically. A history of recent exposure to old cabins, caves, or any rodent-friendly environment should raise the specter of TBRF. Routine laboratory tests are of little value. The WBC count is usually normal but may be increased or decreased. A left shift is often present. Thrombocytopenia is common but nonspecific. The CSF is often abnormal, with a lymphocytic pleocytosis (typically 10 to 2000 cells/mm^3), usually with a normal glucose and elevated protein. A false-positive serologic test for syphilis (Wassermann test) occurs in about 5% of cases.[322]

The diagnosis of relapsing fever is confirmed by demonstrating spirochetes on peripheral blood smears. A routine peripheral blood smear (Wright-Giemsa stain) from a febrile patient is initially positive in 70% of cases.[322] The diagnostic yield can be increased by examining thick smears and by staining with acridine orange using fluorescence microscopy.[309] Inoculating laboratory animals (rats, mice) with blood and examining blood smears from the animals will also increase the diagnostic yield. The visual yield of spirochetes in peripheral blood smears diminishes with each successive febrile sampling. *B. hermsii* can be cultured in BSK-II medium, with the yield increasing in acutely febrile patients. Serologic tests are difficult to perform and do not yet have practical utility.[356] TBRF is probably underrecognized and underreported and has been misdiagnosed as Lyme disease.[102]

Treatment. Tetracycline and erythromycin are both effective in treating relapsing fever. A single oral dose of 500 mg of either drug is effective in louse-borne relapsing fever.[50] However, a 7- to 10-day course (500 mg orally four times a day) is generally recommended in the tick-borne disease.[165,322] For children less than 9 years of age, erythromycin (30 to 50 mg/kg/day in four divided doses) is recommended, with the first dose given intravenously. The borreliae are also sensitive to penicillin and chloramphenicol, but treatment failures have been reported with penicillin.[50] Animal studies have shown that early treatment with a β-lactam antibiotic within 24 hours of onset of spirochetemia decreases CNS involvement.[51]

JHR often occurs after the first dose of antibiotics. In one series, 54% of patients developed a JHR.[102] It is often severe and may be fatal. The reaction begins with a rise in body temperature and exacerbation of existing signs and symptoms; vasodilation and a fall in blood pressure follow. This complex reaction is mediated in part by products of mononuclear leukocytes. The leukocytes are stimulated by increased contact with antibiotic-altered spirochetes. Neither endotoxin nor complement appears to be necessary in the pathogenesis of JHR.[49] JHR typically occurs within a few hours of initial antibiotic dosing.

Waiting to begin treatment until the patient is afebrile does not prevent JHR.[167] Pretreatment with acetaminophen and hydrocortisone results in only mild reduction of hypotension and does not prevent rigors.[50] Patients who are receiving the initial dose of antibiotics for relapsing fever should receive an IV infusion of isotonic saline in anticipation of a possible JHR. This is generally sufficient to counteract hypotension. Lower initial dosages of tetracycline or erythromycin may reduce the frequency of JHR.[167]

In Israel, tick-borne relapsing fever is caused by *Borrelia persica*, transmitted by *Ornithodoros tholozani* ticks. In one study, doxycycline 200 mg PO day 1, then 100 mg PO days 2 through 5 was safe and efficacious in preventing disease following exposure to ticks in a high-risk environment.[153a]

Tick-Borne Viral Diseases

Tick-Borne Encephalitis

Tick-borne encephalitis (TBE) is known by myriad names, including bi-undulating meningoencephalitis and biphasic milk fever. It is caused by a flavivirus and is found throughout Europe and northern Asia. It is actually a spectrum of diseases, varying in severity from louping ill in the British Isles to Russian spring–summer encephalitis (RSSE) in eastern Russia. Central European TBE often has a biphasic course, with an early viremic flulike stage followed later by meningoencephalitis.[245] Symptoms range from mild viral illness to high fevers, severe headache, nausea and vomiting, delirium, coma, paralysis, and death. After an incubation period of 7 to14 days, patients usually develop viremia (2 to 4 days of flulike symptoms), followed by an asymptomatic period of 8 days. Patients then develop the second stage of the disease, which consists of 2 to 4 weeks of mainly CNS involvement.

Treatment is purely supportive. A vaccine is available in Europe and is recommended for those at high risk for exposure, such as farm workers, local residents, and travelers in peak incident seasons and areas. Transmission mainly occurs by *I. ricinus* and *I. persulcatus* ticks, as well as other *Ixodes, Haemaphysalis,* and *Dermacentor* species of ticks indigenous to northern Europe and Asia.

Variants of TBE, also caused by flaviviruses, include Kyasanur Forest disease (KFD) in India, Omsk hemorrhagic fever (OHF) in Siberia, and Alkhurma virus in Saudi Arabia.[68] Symptoms are similar to those of the rest of the TBE complex, except that CNS symptoms are less prominent and there is usually hemorrhage of mucosal surfaces and the respiratory tract. The vector for Kyasanur Forest disease is *Haemaphysalis spinigera.* Omsk hemorrhagic fever is transmitted by contact with infected animal carcasses, unpasteurized milk, and possibly ticks of the genus *Dermacentor.*

Until recently, the only TBE agent known to be endemic to North America was the Powassan (POW) virus in Canada and the northeastern United States. The affliction resembles mosquito-borne encephalitis and has been found when investigation for the West Nile virus yields negative results in clinical situations typical for West Nile virus.[66] It is also often mistaken clinically for aseptic meningitis. The fatality rate is approximately 50%, but fortunately the vector, *Ixodes cookei,* rarely bites humans.[245] The incubation period is 7 to 14 days, after which the disease starts with sudden onset of fever to 104°F (40°C), chills, and occasionally seizures. Nausea, vomiting, and respiratory distress are also common. Care is purely supportive. Reservoirs are skunks, woodchucks, hares, raccoons, and other mammals.

A newly identified TBE agent in the United States was first found in the New England states and subsequently identified in the northern Midwestern United States.[103] It has been dubbed the deer tick virus, and it is transmitted by the deer tick, *Ixodes scapularis,* in these areas.[103,351] It is presumed to be similar to the Powassan virus, or some form of the European TBE, and the resulting clinical appearance is similar. Much is yet to be learned about this new entity. It remains to be seen if *Dermacentor* species in the United States may also be vectors, as they are in Europe. Treatment is entirely supportive.

Overall, with the exception of POW, the case fatality rate for TBE is 1% to 5%. The most lethal of the tick-borne encephalitides to infect humans is RSSE, with a mortality rate of 25% to 30%.[136]

Colorado Tick Fever

Colorado tick fever (CTF) is caused by a retrovirus of the genus *Coltivirus.* Also called mountain tick fever, mountain fever, and American mountain fever, CTF is the only tick-borne virus that occurs with significant frequency in the United States.

CTF is found in the western third of the United States and Canada and is primarily transmitted by *Dermacentor andersoni,* although other vectors are implicated.[203] Larval stages of *D. andersoni* ingest the virus while feeding on a viremic rodent host and pass it transtadially from larva to nymph to adult. Hibernating nymphs and adults carry the virus through the winter. Infected ticks emerge in the spring and feed on susceptible animals, renewing the cycle. Nymphal and adult ticks may also acquire the virus by feeding on viremic hosts. The virus does not appear to cause disease in its natural hosts; it cycles between the vector ticks and many reservoirs, primarily rodents.[237]

Typically, only adult ticks transmit the disease to humans, who are incidental and dead-end hosts. The attachment time required for transmission may be very brief. In human infection, viral replication occurs in bone marrow, lymph nodes, spleen, and hematopoietic erythrocyte precursor cells, and virus is thus found in mature erythrocytes, protected from the body's immune system.[184]

Disease transmission usually occurs from March through September, with the lower elevations yielding cases at the season's extremes, and the higher elevations producing cases in the middle of the season. The incidence is 200 to 300 cases per year in the United States, usually confined to the western states, although remote cases are reported in other areas. CTF is primarily found in areas higher than 4000 feet above sea level. Blood transfusion has been documented as a means of its transmission, and the virus has been documented in blood samples up to 4 months after infection.[53]

Ninety percent of patients can recall a tick exposure. The incubation period is usually 3 to 6 days (range, 0 to 14 days),[143,324] after which the disease typically presents with acute onset of fever. The fever is classically taught as being biphasic (like dengue fever), with fevers to 104° F (40° C) for a few days to a week followed by a brief remission prior to another bout of similar duration. This "saddle-backed" temperature pattern, however, is present in only about half of patients.[143,324] In some cases, further exacerbations of the fever cycle may occur. The fever may also be monophasic, presenting with persistent fever rather than defervescence and relapse. Other symptoms suggestive of a viral illness may occur in mild cases.

Severe complications can occur, particularly in children less than 10 years old. Meningoencephalitis has been described in several children.[62,96,314,324] Two children developed a hemorrhagic diathesis and died.[143] Other unusual complications associated with CTF include pericarditis,[1,162] myocarditis,[106] hepatitis,[212] epididymo-orchitis,[145] and pneumonitis.[143]

Information on CTF contracted during pregnancy is inconclusive, but of five cases reported, one terminated in a spontaneous abortion and in another, a live-born infant suffered multiple congenital anomalies.[246] CTF virus is known to be teratogenic in mice.[153]

Laboratory abnormalities in CTF are not specific. Leukopenia involving both granulocytes and lymphocytes is common and may be helpful diagnostically, although one third of patients have normal WBC counts.[146] Thrombocytopenia may also develop; anemia associated with the virus is rare. Liver function tests may be elevated.

Diagnosis is usually made on clinical grounds. The most reliable means of laboratory diagnosis is inoculating suckling mice or cell cultures with blood or CSF from a febrile patient. PCR is predominantly used in disease research but is slowly finding use as a diagnostic tool as well. Although both highly sensitive and specific for this and many other tick-borne diseases,[266,291] PCR is generally cost prohibitive and not widely accessible. Serologic testing (neutralizing antibody, complement fixation, IFA) by immunoassay is available; however, antibody titers are slow to rise, making these tests better for following efficacy of treatment than for establishing the diagnosis.

Treatment is entirely supportive. Recovery takes several weeks, especially in persons older than 30 years,[143] and persistent malaise and weakness are common. Infection generally confers lifelong immunity.

Pajaroello Tick Bites

In the mountainous regions of coastal southern California, Mexico, and South America, the pajaroello (pajahuello) tick, *Ornithodoros coriaceus,* readily attacks cattle, deer, and humans. The bite is painful and often results in a 10- to 30-mm erythematous papule that may evolve to become erythematous and edematous and may progress to ulceration or necrosis. The lesion gradually resolves over 3 to 4 weeks. Treatment consists of appropriate local wound care and administration of tetanus toxoid (if needed). Secondary infection is not uncommon.

Though no data strongly link the bite of *O. coriaceus* to human miscarriage, it is strongly linked to epizootic bovine abortion, also called foothills abortion, a disease long known among cattle ranchers in these areas. Abortion rates in cattle run as high as 50%.

Tick-Borne Rickettsial Diseases

Species of the genus *Rickettsia* (family Rickettsiaceae) are small, fastidious intracellular parasites with gram-negative bacterium–like cell walls, a typical prokaryotic DNA arrangement, and considerable independent metabolic activity. Several major antigenic groups cause a variety of human diseases worldwide (Table 45-5). Three are potentially transmissible to humans by ticks: spotted fever group (SFG) diseases, Q fever, and ehrlichial infections. Exposure to crushed ticks, or fluid from ticks, may also transmit rickettsiae.

Rickettsiae of the SFG share intracellular growth characteristics and a group-specific antigen. They are distributed worldwide and, with the exception of *Rickettsia akari* (rickettsial pox), are transmitted by bites of ixodid ticks (Table 45-6). Ticks serve as the natural hosts, reservoirs, and vectors for the rickettsiae.[234] The organisms replicate freely within the tick host and are passed transovarially and transtadially. Amplification of the cycle occurs when uninfected ticks feed on an infected vertebrate host or concurrently with an infected tick.

In most natural vertebrate hosts, the SFG rickettsiae induce a subclinical infection with transient rickettsemia. Human infection occurs through accidental intrusion into the natural cycle of infection or when ticks are transferred into human environments. Humans are incidental, dead-end hosts not involved in sustaining the life cycle of the organism.

Rocky Mountain Spotted Fever

The disease currently known as Rocky Mountain spotted fever (RMSF) was first recognized in the Snake River Valley area of Idaho in 1896 and was originally referred to as black measles because of the typical late appearance of the rash as dark papules. At the turn of the century, Dr. Howard Ricketts and others established the epidemiology behind the disease. They realized it was transmitted by tick vectors and named the

TABLE 45-5. Human Rickettsial Diseases

DISEASE	ETIOLOGIC AGENT	ARTHROPOD VECTOR	GEOGRAPHIC DISTRIBUTION
Typhus Group			
Murine typhus	*Rickettsia mooseri* (*typhi*)	Flea	Worldwide
Epidemic typhus	*R. prowazekii*	Body louse	Worldwide
Scrub typhus	*R. tsutsugamushi*	Chigger	Asia, Australia
Spotted Fever Group			
Rocky Mountain spotted fever	*R. rickettsii*	Ticks	Western Hemisphere
Eastern spotted fevers	*R. conorii, R. sibirica, R. australis*	Ticks	Eastern Hemisphere
Rickettsial pox	*R. akari*	Mites	United States, Russia
Other			
Q fever	*Coxiella burnetii*	Ticks	Worldwide
Trench fever	*Bartonella quintana*	Body louse	Africa, Mexico
Ehrlichial infections	*Anaplasma phagocytophila, Neorickettsia sennetsu, N. risticii, Ehrlichia chaffeensis, E. ewingii, E. muris E. ruminatum*	Ticks	Worldwide

TABLE 45-6. Spotted Fever Group Diseases

DISEASE	ETIOLOGIC AGENT	MAJOR VECTOR	GEOGRAPHIC DISTRIBUTION	PRIMARY LESION	USUAL SEVERITY
Rocky Mountain spotted fever	*Rickettsia rickettsii*	*Dermacentor andersoni, D. variabilis*	Western Hemisphere	None	Moderate to severe
North Asian tick typhus	*R. sibirica*	*Dermacentor, Haemaphysalis*	Europe to Russian Far East	Often present	Mild
Mediterranean spotted fever	*R. conorii*	*Rhipicephalus sanguineus, Haemaphysalis*	Mediterranean littoral, South Africa, Kenya, India	Often present	Moderate
Queensland tick typhus	*R. australis*	*Ixodes holocyclus*	Australia	Often present	Mild

causative organism *Rickettsia rickettsii* (Ricketts died in 1910 from typhus, another rickettsial disease). RMSF is also known as tick typhus, Tobia fever (in Colombia), Sao Paulo fever and fiebre maculosa (in Brazil), and fiebre manchada (in Mexico).

Epidemiology. RMSF is found throughout the Western Hemisphere, with 600 to 1000 cases reported in the United States per year in recent years. Although the disease was first discovered in the Rocky Mountains, the name is a misnomer. Most cases are found in the eastern third of the United States, and between 1994 and 1998, less than 3% of the 2643 cases documented nationwide were reported from the Rocky Mountain states. The disease has now been reported in every state except Hawaii, Maine, Vermont, and Alaska, and it is found in southern Canada and Central and South America. Prior to the antibiotic era and in untreated cases, the mortality rate is 30%. With appropriate antibiotic therapy, the mortality rate is 3% to 5%.

Ixodid ticks of the genus *Dermacentor* are the primary vectors of RMSF in the United States: *D. andersoni,* the wood tick, in the West, and *D. variabilis,* the American dog tick, in the East. *Rhipicephalus sanguineus* has recently been implicated in the transmission of RMSF in Arizona.[88] Worldwide, ticks of the *R. sanguineus* complex have been implicated in transmission of various spotted fevers, each in a particular geographic distribution.[131,132] In Mexico, the disease is also transmitted by *R. sanguineus,* the brown dog tick.[21] In Central and South America, the main vector is *Amblyomma cajennense,* the cayenne tick. Within a vector tick species, only 1% to 5% of ticks are infected with *Rickettsia rickettsii,* although coinfection of the tick with other rickettsial species that are nonpathogenic to humans is common.[44] The duration of tick attachment required for disease transmission is as short as 6 hours. The bacterium is readily transmitted by the transovarial route, with 100% transmission in one study.[217]

Rickettsiae are well maintained in the tick population, as ticks may serve as both vectors and hosts. Large mammalian species serve as additional reservoirs. Dogs are an important host of *D. variabilis* and can develop an acute rickettsial illness consisting of rash and fever. Additionally, dogs bring infected ticks home, and many cases of RMSF have been obtained by de-ticking dogs, as the ticks get crushed.[25,145,250] Other tick species have been found to harbor rickettsiae, but they play an insignificant role in disease maintenance or transmission.

RMSF is a seasonal disease: 95% of cases in the United States occur between April and September, with a peak in May through July. Exposure to habitats for the vector ticks, such as wooded and grassy areas adjacent to woods, increases the chance of acquiring the disease. There appear to be "islands" of infection, which could be partially accounted for by transovarial transmission.[230] The disease is slightly more common in males (60% of cases) and in individuals under 20 years of age (50% of cases).[75,350,354] Children aged 5 to 9 years in the mid-Atlantic and southern states have the highest individual incidence.[75] The demographic groups most likely to contract the disease are also more likely to participate in outdoor activities in which ticks are encountered.

Pathophysiology. *Rickettsia rickettsii* are very small bacteria, 0.2 to 0.3 by 0.5 to 2.0 μm in size. Rickettsiae are usually introduced to the host via tick bite, although transmissions via blood transfusion[375] and through aerosolization in a laboratory setting[260] are reported. After inoculation, bacteria spread via the bloodstream and lymphatics and enter cells, specifically vascular endothelial and vascular smooth muscle cells.

In RMSF the rickettsiae invade, proliferate within, and ultimately destroy capillary and precapillary endothelial cells. The organisms may also spread into larger arterioles and arteries and invade medial smooth muscle cells. Medial necrosis and destruction of the vascular wall may follow. At sites of endothelial cell damage, a perivascular inflammatory response ensues, and platelet and fibrin thrombi tend to form and occlude the vessel lumen. In severe cases, vascular thrombi lead to necrosis of peripheral parts, including fingers, toes, the external ear, and the scrotum. Antibodies develop 5 to 7 days after the onset of illness but do not appear to play a significant role in the pathogenesis of the vasculitis.[387]

Clinical Manifestations and Diagnosis. The clinical appearance of RMSF ranges from very mild to fulminant, and it may be fatal within several days of onset. A history of tick exposure is present in 85% of confirmed cases.[354] The incubation period is 2 to 14 days, with a median of 7 days; fulminant disease usually has a shorter incubation period. There is usually sudden onset of chills and fevers (in 94% of patients), with 66% reporting fever within the first 3 days, 86% reporting headache, and 85% reporting myalgias.

RMSF produces a disseminated vasculitic rash[369] in 85% to 90% of patients. The rash, which usually occurs after onset of constitutional symptoms, usually begins on the wrists, hands, ankles, and feet and spreads centripetally. It initially appears as blanching pink or red macules, 2 to 5 mm in diameter, that evolve into more pronounced, red, papular, nonblanching lesions. Finally, the rash becomes petechial and dark red to black; occasionally, areas of ecchymotic coalescence are noted. End-terminal vascular areas such as fingertips, toes, nose, and genitalia may become necrotic. Involvement of the scrotum or vulva is a diagnostic clue.[95] Ten percent of patients have a delayed rash, and 10% to 15% have no rash.[154,155,157] Patients most likely to have no rash, such as the immunocompromised, are also those most likely to die from the disease.[155]

Neurologic manifestations range from minor headache to profound neurologic symptoms such as encephalopathy, seizures, ataxia, and delirium. Headache is common, occurring in approximately 90% of cases, and is of varying intensity. Lethargy and confusion are common and progress to stupor or coma in 10% of cases. Cerebral vasculitis may develop but is usually mild and transitory. CSF protein analysis may be normal or elevated, sometimes dramatically. Mild CSF pleocytosis of lymphocytes and polymorphonuclear cells (8 to 35 cells/mm^3) may occur.

Myocarditis is frequently found at autopsy in fatal RMSF; however, the clinical significance of the cardiac involvement is unclear. Pathologic study shows a patchy, interstitial, mononuclear infiltrate that appears to coincide with distribution of rickettsiae in myocardial capillaries, venules, and arterioles.[370] Abnormal left ventricular function can frequently be demonstrated echocardiographically in hospitalized patients.[116,226] Overt clinical manifestations of left ventricular dysfunction are uncommon, however. Hypotension (present in 17% of cases) and pulmonary edema are generally attributable to noncardiogenic causes such as hypovolemia and hypoalbuminemia. Cardiac enlargement rarely may be seen on chest radiographs.[207,227] ECG abnormalities include nonspecific ST-T changes, conduction abnormalities (primarily first-degree atrioventricular block), and arrhythmias (paroxysmal atrial tachycardia, nodal tachycardia, and atrial fibrillation).[227] Most patients have complete resolution of cardiac abnormalities with clinical improvement, but persistent echocardiographic abnormalities have been noted.[226]

Infection of the pulmonary microcirculation by *R. rickettsii* results in interstitial pneumonitis and increased pulmonary vascular permeability. Although pulmonary involvement is not usually a prominent aspect of RMSF, a significant number of patients complain of cough, chest pain, or dyspnea.[54] Patchy infiltrates are occasionally seen on chest radiographs, and noncardiogenic pulmonary edema may develop in severe cases, with potential progression to ARDS.[94,207,300]

Gastrointestinal symptoms are common in RMSF and are prominent complaints in some patients. On physical examination, 10% of patients have guaiac positive stool. At autopsy, rickettsial vascular lesions are frequently found throughout the gastrointestinal tract and pancreas, although actual necrosis appears to be a rare event.[277] Occasionally, patients with RMSF present with an acute abdomen suggesting appendicitis or cholecystitis.[368]

In the kidneys, focal perivascular interstitial nephritis is concentrated near the corticomedullary junction. Clinically, however, significant renal involvement is usually caused by prerenal azotemia or acute tubular necrosis after a hypotensive episode.[348]

Monarticular arthritis in the acute phase of RMSF has been reported.[348]

Major complications of RMSF result from direct vasculitic injury. In late stages of the disease, diffuse vasculitic lesions cause increased systemic capillary permeability, leading to hypovolemia and vascular collapse. Disseminated intravascular coagulation (DIC), acute renal failure, metabolic acidosis, and cardiac and respiratory dysfunction may ensue and are frequently preterminal events. Endothelial leukocyte adhesion molecules and cytokines are thought to play an important role in pathogenesis of fulminant RMSF. Long-term sequelae include paraparesis, hearing loss, peripheral neuropathy, bladder and bowel incontinence, language disorders, and disability from limb amputation, and cerebellar, vestibular, and motor dysfunction.

Untreated, RMSF has a mortality rate approaching 30%.[44] Mortality is higher in patients with elevated serum creatinine,

elevated hepatic function tests, hyponatremia, and thrombocytopenia and associated immunosuppression.[71] Acute renal failure has been linked to a 17-fold increase in mortality.[71] Patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency are more likely to develop fulminant disease,[367] and RMSF is nearly always fatal in these individuals. Persons of African, Middle Eastern, and Southeast Asian descent are most likely to have G6PD deficiency.

Diagnosis. Early diagnosis and treatment substantially reduce morbidity and virtually eliminate mortality in RMSF. For this reason, suspicion of RMSF is sufficient to begin treatment. At the onset of illness, however, signs and symptoms are frequently nonspecific, leading to diagnostic confusion with viral or other infectious diseases.[183]

Serology is the best confirmatory test, but false-negative results early in the course of infection (before the body has mounted an appropriate antibody response) are common. A fourfold increase in titers, or a single titer over 64, is required for a serologic diagnosis; this is usually not achieved by the time testing is performed. Indirect IFA of *R. rickettsii* in skin biopsy specimens yields 70% to 90% sensitivity and 100% specificity[274] and can detect rickettsiae early in the disease,[388] but this is impractical for routine use.

Routine laboratory analysis for hematology and chemistry may reveal common abnormalities that suggest RMSF. Thrombocytopenia is present in 35% to 50% of patients,[154,157,365] and hyponatremia[112] and anemia are also common.

Treatment. Antibiotics should be started empirically in patients living in or recreating in an endemic area who develop any of the constitutional symptoms (myalgias, headache, fever) of RMSF. The classic triad of rash, tick bite, and fever is present in only 18% of patients within the first 3 days of illness.[157] Antibiotic treatment should not be withheld until the patient provides a self-diagnosis; patients started promptly on antibiotics are three times less likely to die than if started on antibiotics later.[75]

Tetracyclines and chloramphenicol are the antibiotics of choice in treating RMSF. Treatment is generally for 5 to 7 days, or until the patient has been afebrile for 48 hours or more. Parenteral antibiotics are not necessary in nontoxic patients, but they should be used in patients who are systemically ill or unable to tolerate oral medications. Although tetracyclines are generally not used in young children because of their propensity to permanently stain developing teeth, tetracyclines are superior to other antibiotics in the treatment of RMSF, and typical courses of treatment for RMSF have been shown to have no adverse effects in the pediatric population.[52] Ciprofloxacin has been used in Europe to treat Mediterranean spotted fever.[297] Other antimicrobials have been used in animal studies and are as effective or nearly as effective as tetracyclines, but there are no strong data to support their use in children.[37] Even with timely and appropriate antibiotics, however, mortality is approximately 3% to 5%.[155] The high mortality associated with improper treatment of RMSF warrants treatment of most patients with doxycycline, with a warning to the parents that permanent staining of teeth may occur.

Supportive therapy, including aggressive fluid resuscitation in many cases, is a mainstay of treatment of RMSF. Corticosteroids may be of benefit in cases of severe vasculitis or encephalitis.[235,387]

Other Spotted Fever Group Diseases

Mediterranean Spotted Fever. Boutonneuse fever, also called Mediterranean spotted fever (MSF), Carducci fever, tick typhus, Indian tick typhus, and tick bite fever is caused by *Rickettsia conorii* and is endemic in the Mediterranean basin and in southern Asia and India. The major vector of MSF in Mediterranean countries is the dog tick, *Rhipicephalus sanguineus*. Several tick species have been implicated as vectors in other areas; these include *Haemaphysalis leachi* (Kenya, South Africa), *Rhipicephalus simus, Dermacentor reticulatus,* and *Ixodes hexagonus*.[114] All stages of *R. sanguineus* occasionally attach to humans, and dogs may be important in transporting the ticks close to humans.[357]

Infection with *Rickettsia conorii* usually results in mild illness, although symptoms can be severe in 10% of patients, typically older adults or immunocompromised patients. Symptoms are fever, exanthems, and an eschar or necrotic plaque at the tick bite site. Fever of 102° to 104° F (39° to 40° C) is noted in 97.2% to 100% of patients. Erythematous papules, primarily in the lower extremities, are noted in 96.1% to 100% of patients. A black eschar, appearing like a button at the site of the tick bite, is seen in 71.8% of patients (Fig. 45-12). In addition, a vasculitic rash, similar to that seen in RMSF, may occur on the palms, soles, wrists, and ankles. In some cases, the only symptom is lymphadenopathy. The incubation period is typically from 4 to 15 days but may be as long as 28 days. Although the disease is usually mild in children and young adults, a malignant form resembling severe RMSF has been described.[282,366] Older adults, alcoholics, and patients with G6PD deficiency appear particularly at risk.[171,176]

The incidence in Mediterranean rim countries and other countries varies from 51.6% of those reporting tick bites in Croatia, to 4.6% to 13.5% in a region of Spain, 13.3% of healthy patients in Turkey, and 16.7% in Zambia. Numerous cases have been imported, diagnosed, and confirmed in the United States and Canada from travelers to endemic regions.

Abnormal laboratory values and other clinical symptoms share the clinical picture often seen with other tick-borne diseases. Treatment is with tetracycline, administered orally or

Figure 45-12. Tick-bite eschar associated with African tick typhus. (From Armstrong D, Cohen J [eds]: Infectious Diseases. London, Mosby, 1999, with permission.)

parenterally. Chloramphenicol and the fluoroquinolones are reasonable second-line agents.

Siberian Tick Typhus. Siberian tick typhus, or North Asian tick typhus, caused by *Rickettsia sibirica,* is found throughout northern Russia and on islands of the Sea of Japan. It is primarily transmitted by various *Dermacentor* and *Haemaphysalis* species ticks. Symptoms are very similar to RMSF, although it is seldom fatal. Diagnosis and treatment are the same as for the other spotted fever group diseases.

African Tick Bite Fever. African tick bite fever is caused by *Rickettsia africae,* a newly discovered agent of disease. It is similar to Boutonneuse fever but typically occurs without the rash. It is transmitted by *Amblyomma hebraeum* and is endemic to sub-Saharan Africa. Although rarely reported in indigenous peoples, it is well documented in travelers to the African continent.[169,174]

Queensland Tick Typhus. Queensland tick typhus is caused by *Rickettsia australis* and is transmitted by *Ixodes holocyclus,* which is also the primary vector of tick paralysis in Australia. It is also very similar in appearance to the other spotted fever groups, and it is diagnosed and treated in the same fashion.

Q Fever

Q fever is a worldwide zoonosis affecting both wild and domestic animals. It was first described in 1937 as an occupational disease of abattoir workers and dairy farmers in Australia.[89] Aerosol spread of *Coxiella burnetii,* the causative organism, is the usual mode of transmission to humans. Sexual transmission has been implicated but not proven.[199] Although ticks may become infected with *C. burnetii* after feeding on an infected vertebrate, tick-borne transmission to humans appears to be very rare.

Fewer than 10 cases of Q fever per year are reported in the United States. Serosurveys have shown widespread prevalence, suggesting frequent asymptomatic infection or underdiagnosis and underreporting.[149,313]

The most common clinical manifestation of Q fever is an influenza-like illness with fever, headache, myalgias, and pneumonitis. Abnormal liver function tests, jaundice, and hepatomegaly may be seen, and Q fever may cause acute acalculous cholecystitis.[290] Glomerulonephritis has been reported with both acute Q fever and endocarditis-associated chronic infection.[192] *C. burnetii* is not known to be teratogenic, but infection during pregnancy may cause placental insufficiency resulting in premature delivery or even intrauterine death[127,281]; the risk of miscarriage is also increased.[279] Reported neurologic manifestations of Q fever include acute cerebellitis with tonsillar herniation,[303] meningitis/encephalitis/meningoencephalitis,[32] transverse myelitis,[32,371] and peripheral neuropathies.[32,319] Cardiac involvement in acute Q fever is rare but may include pericarditis,[23,208] endocarditis, or myocarditis.[125] In most patients, symptoms resolve spontaneously within 2 to 4 weeks of onset, with treatment hastening the resolution.

Q fever may also be a chronic infection, with or without a history of an acute episode. Granulomatous hepatitis and culture-negative endocarditis are the major manifestations of the chronic forms of the disease. Endocarditis, fatal in 25% to 60% of patients, can affect native and prosthetic valves (underlying valvular disease is almost invariably present), and it has a predilection for the aortic valve.[141] Infections of aneurysms and vascular prostheses have also been described.[124] A post–Q fever syndrome involving fatigue, myalgia, arthralgia, night sweats, sleep disturbances, and mood alterations has been described and may occur in 20% to 42% of proven cases.[13,265]

Diagnosis of Q fever depends primarily on serologic testing. Two specific complement-fixing antibodies (phase 1 and phase 2) develop after infection with *C. burnetii.* In patients with acute Q fever, phase 2 antibody is usually detectable by the second week of illness; phase 1 is not detectable. The finding of phase 1 antibody indicates chronic infection. IgA subclasses (IgA1 seen in acute and chronic disease, IgA2 seen only in chronic disease) may also help differentiate between chronic and acute infection. Only patients with endocarditis were found to have IgA2 antibodies to phase 2 antigens.[54]

For the acute phase, tetracycline (500 mg orally four times a day) will hasten resolution. Treatment of chronic Q fever is not always successful. Patients probably need to be treated with tetracycline for at least 12 months.[5] Some authorities recommend adding another drug, such as lincomycin or cotrimoxazole, but the efficacy of this strategy is unproven.[126,359] Patients with endocarditis typically receive treatment with tetracycline and a quinolone for at least 4 years, although new research supports shortened treatment with doxycycline and hydroxychloroquine.[280] A vaccine has been used to prevent infection in high-risk abattoir workers. Preventive measures such as avoidance of potentially infectious animal tissues, especially raw milk and products of conception, should be taken. Pregnant patients should be aggressively treated with cotrimoxazole for the duration of the pregnancy, as insufficient treatment is associated with a higher rate of fetal loss.[279]

Ehrlichiosis

In Texas in the 1940s, there was an epidemic of disease among World War II U.S. Army recruits. The illness was associated with bites from the lone-star tick, *Amblyomma americanum.* The location was Camp Bullis, and the disease became known as Bullis fever.[386] In retrospect, it is very likely that Bullis fever was human ehrlichiosis.[135,138]

Ehrlichiosis was "discovered" in the mid-1980s when several individuals in rural areas of central and northern Arkansas became ill with a febrile disease that had many of the symptoms of Rocky Mountain spotted fever, with which they were initially diagnosed.[122] Clusters of bacteria in intracytoplasmic inclusions were identified as an *Ehrlichia* species, similar if not identical to *Ehrlichia canis. E. canis* had previously been known to infect only dogs. The new bacterium was later isolated and given the name *Ehrlichia chaffeensis,* after Fort Chaffee, Arkansas, the area from which the original cultures came. The late 1980s and early 1990s yielded several hundred cases, including some fatalities. By the year 2000, there were over 1000 reported cases of ehrlichiosis. Most cases are sporadic; however, there are epidemics in some occupational and recreational groups, such as military personnel in field exercises and golfers with frequent errant shots.[267,326,391]

The taxonomy of the species that cause ehrlichiosis is very complicated and has undergone frequent change. This is a dynamic area of research, with new species and new vectors being found worldwide and nearly every day. The species that cause the various ehrlichioses belong to the family Anaplasmataceae and are obligatory intracellular gram-negative

bacteria. They multiply in host cell vacuoles and form large mulberry-shaped inclusion bodies called morulae.

At present, about a dozen species are known in the family Anaplasmataceae. The bacteria causing human disease are divided into three genera: *Neorickettsia* (formerly *Ehrlichia sennetsu* and *Ehrlichia risticii*), *Anaplasma* (formerly *Ehrlichia phagocytophila* and *Ehrlichia equi*), and *Ehrlichia*. The *Ehrlichia* species consist of *Ehrlichia ewingii, E. chaffeensis, E. canis*, *Ehrlichia muris,* and *E. ruminatum*.[100] More than 21 different strains of *E. chaffeensis* have been isolated from U.S. patients in the southern states. Additional strains have been isolated from animal sources, although infectivity of these strains is not well established.[98]

There are two basic forms of ehrlichiosis in humans: human monocytic ehrlichiosis (HME) and human granulocytic ehrlichiosis (HGE).

Human Monocytic Ehrlichiosis

The first of the ehrlichiae identified as a cause of human disease was *E. chaffeensis*. Although reported in 46 states, *E. chaffeensis* is found predominantly in the south-central and southeastern United States. It causes HME, and is transmitted by *Amblyomma americanum,* the lone-star tick. *E. chaffeensis* infects predominately mononuclear phagocytic cells, particularly monocytes, although other cell types may also be affected. Various organs are affected, particularly those with abundant mononuclear phagocytic cells. Cross-reactivity exists between the tests for *E. chaffeensis*, *E. ewingii*, and *Anaplasma phagocytophila,* so it is possible that not all cases attributed to *E. chaffeensis* are truly caused by this organism. This may change as tests become more specific.

Less is known about the epidemiology of HME because the vector, *A. americanum,* has not been as extensively studied as *I. scapularis* ticks, the primary vectors for HGE and Lyme disease. The five states with the highest incidence of HME, compiled from data obtained from 1986 to 1997, were Arkansas, North Carolina, Missouri, Oklahoma, and New Jersey.[238]

HME occurs worldwide. The causative agent is always *E. chaffeensis* or very similar *Ehrlichia* species. There are multiple tick vectors worldwide, however, including primarily ticks of the *Ixodes ricinus* complex in Eurasia, *Amblyomma cajennense* in South America, *Amblyomma testudinarium, Haemaphysalis yeni, Rhipicephalus sanguineus* in China, and others.[56,288,376]

Nontick transmission has been suggested by the occurrence of HME after contact with infected animal blood.[15] *E. chaffeensis* can live in refrigerated blood for longer than 11 days, so transmission by blood transfusion is possible.[236]

In the United States, HME is transmitted primarily in the months of March through November, with 70% of the cases occurring between May and July.[120,121,326] Cases are reported in the late fall and winter months from southern areas. HME has a male-to-female ratio of greater than 2:1, and it is reported more often in patients older than 40.[120] Younger patients may have less symptomatic disease and thus go unreported. History of a tick bite is present in approximately 68% to 80% of patients.[109,120] The high rate of recollection of a tick bite is probably a result of the pain associated with the relatively long mouthparts of *A. americanum* and the immunogenic local reaction that often accompanies the bite.

There are numerous reservoirs for the ehrlichiae. *A. americanum* is very nonselective in its feeding and is found on a variety of birds and mammals.[33] In the United States, the white-tailed deer is the most important host. Domestic dogs also are vital in the epizootiology of the ehrlichioses. Dogs outnumber every other large mammal in the United States and are often highly infected, with a Virginia study showing 38% of dogs from southeastern Virginia positive for *E. chaffeensis*.[84] Dogs also provide a large degree of interface between the wilderness areas and the domestic front, and they often carry ticks into the home.[25,250] A study of coyotes in Oklahoma documented infection rates of 71%.[191] Wild turkeys also host *A. americanum,* which is so abundant on some turkeys that it is referred to as the turkey tick in some areas.[87]

Human Granulocytic Ehrlichiosis

In the 1990s, *E. phagocytophila* (now *Anaplasma phagocytophila*) and *E. ewingii* were also identified as human pathogens. These bacteria primarily infect neutrophils, causing HGE. These species cause disease in the north-central and northeastern United States, and they are transmitted by *Ixodes* species ticks, primarily *I. scapularis,* and *Dermacentor variabilis*. The numbers of reported cases quickly exceeded those from the original areas of *E. chaffeensis*. The reservoir hosts are similar to those for HME.

Clinical Features. The clinical features of HME and HGE are very similar. Within 1 to 2 weeks of exposure to an infected tick, a prodrome of malaise, back pain, gastrointestinal symptoms, and fevers to 102°F (39°C) develops. HME symptoms usually manifest within 3 to 4 days of onset and include fever (>95%), headache (60% to 75%), myalgias (40% to 60%), nausea (40% to 50%), arthralgias (30% to 35%), cough or dyspnea (20% to 25%), and malaise (30% to 80%).[109,120] Respiratory complications are common; cough, pulmonary infiltrates, dyspnea, and respiratory failure have been reported. Rash is noted in both HME and HGE, although it is more common in the former (40%) than in the latter (8%).[99]

Meningoencephalitis is the most common neurologic complication. In one study of 15 patients with altered sensorium from ehrlichiosis who underwent CSF testing, eight had abnormalities, including elevated lymphocytes (up to 1000 cells/μL) and protein. In a review of 21 additional cases with CNS manifestations, 13 of 21 patients had abnormal CSF findings. Fourteen patients underwent brain computed tomography, revealing no abnormalities. Four of 21 patients with CNS symptoms died.[283]

Hematologic complications are also described. In HME, bone marrow and hepatic granulomas and multiorgan perivascular lymphohistiocytic infiltrates have been observed. In HGE, *Ehrlichia*-mediated defects in host defense and immune suppression have resulted in opportunistic fungal and viral infections.[365]

Up to 60% to 70% of patients require hospitalization.[120,327] In severe cases, patients suffer renal failure, pancytopenia, DIC, ARDS, hepatic failure, profound hypotension, and death. There is an estimated 3% case fatality rate, down slightly from 5% in the mid 1990s.[238] Death occurs in males in 70% of cases, and usually results from multisystem organ failure, usually with profound pulmonary, hematologic, and gastroenterologic manifestations. Generally, immunocompromised patients, individuals older than 60, and those not receiving appropriate antibiotics within 8 days of the onset of symptoms are at highest risk of mortality.[120]

Diagnosis. In patients with symptoms suggestive of ehrlichiosis, the diagnosis is suggested by history of tick bite in an endemic area and confirmed by laboratory testing.

Multiple abnormalities may be noted on routine blood testing. The complete blood count (CBC) shows mild to moderate thrombocytopenia in 70% to 90% of patients and mild to moderate leukopenia (principally lymphocytopenia) in 60% to 70% of patients. Hepatic transaminases are elevated in 80% to 90% of patients at some point in their illness, often early in the course. Hyponatremia is present in up to 50% of adult patients and 70% of pediatric patients.[109,170] Elevated creatinine, elevated fibrin degradation products, and prolonged coagulation times are indicative of more severe disease and carry a poor prognosis.

Serologic tests confirm the diagnosis, but the results are not immediately available. The most widely used test is an IFA, which detects IgG and IgM. A rise in titer of fourfold or greater, or a single titer of 256 or greater is sufficient to confirm the diagnosis. The serologic tests use an antibody reactive to *E. chaffeensis;* the degree of cross-reactivity with other erhlichiae is unclear. The gold standard is the IFA, which is 95% sensitive and 100% specific when properly performed at appropriate times. Early testing may yield false-negative results, however, because of the relatively slow human antibody response to ehrlichiosis. Suspicion of disease in the appropriate setting should therefore prompt treatment, and a negative test early in the presentation should not preclude antibiotics.

Western blot tests identify isolates of *E. chaffeensis* but do not play an important role in diagnosis. For *E. ewingii,* however, Western blot is useful for diagnosis, as there is no IFA test for this organism. ELISA tests are being developed but are not routinely used. PCR is beginning to complement IFA, but it is primarily a research tool; it has the advantages that any tissue can be used and the disease can be detected early. Blood smear testing has very low sensitivity and specificity, is very operator dependent, and plays little role in diagnosis. Culture is feasible but is technically difficult, requires specially equipped laboratories and special media, and takes several weeks.

Treatment. *E. chaffeensis* is susceptible to tetracyclines but resistant to many antibiotics, including aminoglycosides, fluoroquinolones, penicillins, macrolides, and sulfa drugs. Doxycycline (100 mg orally or intravenously twice daily) is the drug of choice for treatment of *E. chaffeensis* in adults and children older than 9 years of age. Treatment for 7 to 10 days (or for 3 days after the last febrile episode) is typically adequate. Failure to respond within 48 to 72 hours of initiation of antibiotics suggests a different diagnosis or coinfection. Other antibiotics may be considered when tetracyclines are contraindicated. Rifampin is very bactericidal in vitro, but it has not been used in large trials in vivo.[36,42] Chloramphenicol, which has been used in children with varying degrees of success,[22,219] has been shown to have weak inhibitory action against the ehrlichiae, but it is not bactericidal.[188] Both rifampin and chloramphenicol are safe in children but are rated as category C for use during pregnancy. Despite this rating, a pregnant patient with HGE was treated successfully with rifampin.[43]

Babesiosis

Babesia species, like malarial organisms, are pleomorphic intraerythrocytic protozoan parasites. More than 70 distinct species have been described from various vertebrate hosts.[289] Some of the most important species include *Babesia bigemina, Babesia bovis,* and *Babesia divergens* (all in cattle); *Babesia caballi* and *Babesia equi* (horses); *Babesia canis* (dogs); and *Babesia microti* (rodents). Babesiae are transmitted to vertebrates primarily through the bites of ixodid ticks.

Epidemiology. Babesiosis has long been recognized as an important veterinary disease, receiving biblical reference as the "divine murrain" infecting cattle of the pharaoh Ramses II (Exodus 9:3). Various epidemics of cattle fever have been documented throughout history.[35] Human disease was originally reported in 1957 in a splenectomized man in Yugoslavia.[315] Five cases of human infection with bovine *Babesia* (*B. bovis, B. divergens*) were reported from Europe between 1957 and 1976; two cases of babesiosis of unknown species were reported from California in 1968 and 1981; a human case of *B. gibsoni* infection was reported in California in 1993; and a single *Babesia caucasia* infection was reported from Russia in 1978.[175,289,292] These cases were widely separated geographically, and all occurred in splenectomized individuals.

Since 1970, the incidence of human babesiosis has accelerated, primarily because of an outbreak of *B. microti* infections in the northeastern United States. The first case was recognized in a patient with an intact spleen in 1969 on Nantucket Island, Massachusetts.[378] Since then, more than 450 confirmed cases have occurred in the United States.[76,240] *B. microti* is endemic to the coastal regions of southern New England, where the principal vector is the northern deer tick *Ixodes scapularis.* Most cases are contracted on Cape Cod and the offshore islands of Massachusetts (Nantucket, Martha's Vineyard), New York (Shelter Island, Long Island), Rhode Island (Block Island),[76] and New Jersey.[161] Cases have also been reported from Ontario,[182] Wisconsin, and Minnesota, a known focus of *I. scapularis.*[343] The incidence of *B. microti* infection is higher than that of other *Babesia* species because of its ability to produce disease in individuals with intact spleens.

Although most cases of babesiosis in the United States involve *B. microti,* infection with other strains occurs. A new strain of *B. microti* called WA-1, thought to be spread by *Ixodes pacificus,* has been isolated from an immunocompetent patient with an intact spleen in Washington State.[275] Another strain, termed MO1 and similar to (but distinct from) *B. divergens,* was isolated in Missouri.[160] *B. divergens* infection is reported in the United States but is rare.[24] Interestingly, *B. divergens* was found in 16% of cottontail rabbits in Nantucket, indicating an abundant reservoir in at least that area.[140] The ecology of *B. microti* parallels that of *Borrelia burgdorferi,* the agent of Lyme disease. The major vector in both diseases is *I. scapularis.* White-footed mice (*Peromyscus leucopus*) constitute the major reservoir for *B. microti.* Larval or nymphal ticks ingest the parasite during a blood meal from an infected rodent. Babesiae replicate within the tick and are passed transtadially. Amplification of the cycle occurs when the infected tick transmits the organism to a vertebrate host during the next blood meal. White-tailed deer (*Odocoileus virginianus*) are the principal hosts for adult ticks; larvae and nymphs feed on deer, mice, and other small mammals.[323]

Human babesiosis occurs when humans accidentally intrude on the natural cycle and are bitten by an infected tick. As is the case with other tick-borne diseases, the peak incidence is during the warm weather months from May to September, when ticks are actively feeding. The majority of infections with *B. microti*

are asymptomatic. Recent serologic testing shows that although seroconvergence has increased greatly over the past 30 years, it stabilized in the 1990s.

An epidemiologic survey of 136 cases in New York State showed that the most important risk factors for severe babesiosis are advanced age, asplenia, and immunodeficiency. Babesial infection appears to be as prevalent in children as in adults, although the disease tends to be more severe in those older than 40. In one study, 23% of patients with babesiosis had concurrent Lyme disease infection.[240] Case reports have also shown multiple coinfection involving borreliosis, ehrlichiosis, and babesiosis,[244] with more severe symptoms in coinfected individuals than in those infected with a single organism.[197]

Prolonged, subclinical infection creates the potential for transmission of *B. microti* through blood donation. More than 26 cases of babesiosis acquired by transfusion have been reported.[91,202,225,316,385] Seroprevalence surveys in endemic regions show donor exposure rates of 3% or greater.[92] Currently, control of transfusion-related infection is limited to identifying donors at high risk of exposure (from endemic regions, history of tick bites, or seasonal exposure to tick-favorable landscapes). This strategy may become less effective as tick-endemic areas expand, however. Transfusion has also reportedly transmitted new species of *Babesia*, causing significant disease in immunocompetent individuals.[162]

Pathogenesis. Babesiae are transmitted from wild and domestic animal reservoirs to humans by *Ixodes* ticks. The life cycle of *I. scapularis* (the vector for transmission of *B. microti* to humans from rodents) spans 2 years, beginning in spring of the first year with hatching of the larval form. In late spring and summer months, the larvae feed on a variety of hosts and acquire babesial infection. Typically, larvae become infected from their preferred host, the white-footed mouse, *P. leucopus*. Ingested babesiae reach the gut of the feeding tick, where they reproduce asexually. The newly formed zygote eventually spreads throughout the body of the tick. After reaching the salivary glands, the babesial sporoblasts remain dormant until the next spring, when the tick larva molts to a nymph. The nymph then seeks a blood meal, infecting a new host (rodent or human). In endemic areas, as many as 60% of white-footed mice are infected by late summer. The nymph form of the tick transmits most human disease, although adult ticks may also do so.

After inoculation by a tick bite, *Babesia* sporozoites enter erythrocytes, where they differentiate into merozoites. Great pleomorphism is displayed,[248] but ring-shaped and ameboid trophozoites are the predominant forms. Multiplication occurs by asexual asynchronous budding. After the parasite multiplies, the infected erythrocyte ruptures, freeing the organisms to invade other red blood cells (RBCs). Severe hemolytic anemia may ensue (Fig. 45-13).

Infection with *B. microti* reduces the malleability of erythrocytes, leading to microvascular stasis and decreased RBC life span. Electron microscopy has shown extensive RBC wall damage, including protrusions, perforations, and extrusions in the cell membrane. Asplenia or steroid therapy can worsen the disease and prolong parasitemia. An intact spleen preferentially destroys infected RBCs because of their decreased malleability, which plays an important role in resistance to *Babesia* organisms. Although the presence of a spleen is not protective against

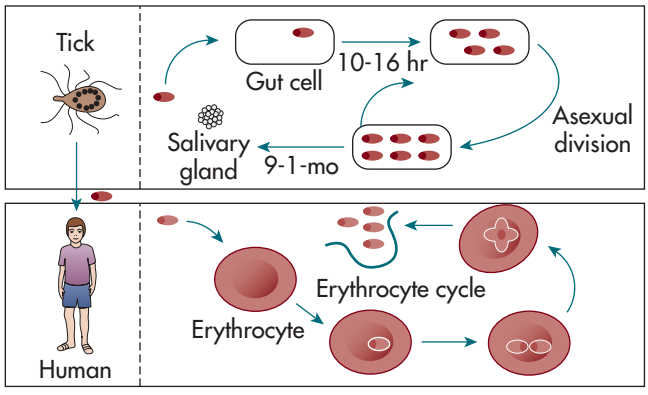

Figure 45-13. Babesia life cycle. Within a tick, babesial parasites infect gut cells, undergo asexual division, and eventually migrate to the salivary glands. The organism is introduced into a human when the infected tick takes in a blood meal. Transmission usually involves the nymphal tick, but the duration of attachment required for transmission of *Babesia* species is not known. *Babesia* species can be transmitted to humans concomitantly with *Borrelia burgdorferi*. (Modified from Boustani MR, Gelfand JA: Clin Infect Dis 22:612, 1996.)

B. microti, the disease is often more severe in splenectomized patients.

Age is also an important factor in susceptibility to babesiosis. Children and young adults usually have subclinical or mild, self-limited infections, whereas older adults and those with chronic medical problems[29] are more likely to have severe, clinically apparent disease.[28,296] Prolonged parasitemia is common in babesiosis.[294] *B. microti* may persist for as long as 4 months in an otherwise healthy patient.[378] Parasitemia may remain after clinical recovery or may develop in asymptomatic individuals.

Clinical Manifestations. Acute *B. microti* infection is characterized by gradual onset of malaise, anorexia, and fatigue, followed within several days to a week by fever, sweats, and myalgias. Other, less common symptoms include headache, nausea and vomiting, depression, abdominal pain, and dark urine. In a review of 17 patients with babesiosis, 52.9% presented with temperature greater than 38.3°C (101°F), and four of the nine had morning fever spikes; eight had relative bradycardia.[187]

Incubation is 1 to 4 weeks after a tick bite or 6 to 9 weeks after transmission by blood transfusion. Most victims of tick-transmitted disease do not recall a tick bite. Physical examination is usually normal, except for fever (steady or intermittent) and mild splenomegaly in some patients. Petechiae and ecchymosis rarely occur.

The CBC usually reveals mild to moderate hemolytic anemia, normal to slightly reduced WBC count, and (in some patients) mild to moderate thrombocytopenia. Serum lactate dehydrogenase (LDH) and bilirubin levels are mildly elevated in most patients, reflecting the hemolytic anemia.[142,294] AST and ALT may be elevated, and urinalysis may be notable for proteinuria and hemoglobinuria. In one series, 13 of 17 patients (76.5%) had lymphopenia, and five (29.4%) had rouleau formation in the peripheral blood smear.[187]

A review of 139 hospitalized patients with babesiosis in New York from 1982 to 1993 attempted to identify common signs and symptoms and the prognostic factors associated with poor

Box 45-2. Laboratory Diagnosis of Babesiosis

Peripheral blood smear (Wright or Giemsa staining) showing intraerythrocytic *Babesia*
Polymerase chain reaction
Indirect immunofluorescent assay
Intraperitoneal inoculation of splenectomized hamsters

outcome.[379] Of the 139 patients, 9 (6.5%) died, 35 (25.2%) were admitted to the intensive care unit, and 35 (25.2%) required more than 14 days of hospitalization. Among patients with severe disease, the mean age was 62.5 years, and 62% were male. The most common symptoms were fatigue, malaise, weakness (91%); fever (91%); shaking chills (77%); and diaphoresis (69%). Prognostic indicators for severe outcome included high alkaline phosphatase, male sex, and elevated WBC count. Only 12% of patients with severe disease had a history of splenectomy, and only 2% had received a prior blood transfusion.

Although most patients with normal splenic function recover without specific therapy, prolonged fatigue and malaise are common.[294] Splenectomized patients generally have more severe clinical disease, with higher levels of parasitemia and more severe hemolytic anemia.[296] Older adult patients and immunocompromised patients, such as those with HIV infection, are also at higher risk for severe infection. Pulmonary edema has been reported.[35] Typically, the level of parasitemia ranges from 1% to 10%.

In Europe, babesiosis results from infection with *B. divergens* or *B. bovis* and has been reported only in splenectomized patients. The illness is characterized by high fever, chills, headache, and severe hemolytic anemia, often resulting in hemoglobinuria, jaundice, and renal insufficiency. Major findings on physical examination include fever, hepatomegaly, jaundice, and hypotension. More than half of the reported cases have been fatal.[292]

Babesiosis during pregnancy is reported,[113,284] as is vertical transmission from mother to child in utero.[108]

Diagnosis. Babesiosis should be considered in any person with an unexplained febrile illness who has lived in or traveled to an endemic region in the midsummer months, especially with a history of a tick bite or tick exposure. The diagnosis of babesiosis can be confirmed by identifying the intraerythrocytic parasites on Giemsa-stained blood smears (Box 45-2). Persons with intact spleens usually have low levels of parasitemia (less than 5% parasites), and examination of repeated smears may be necessary.[142,294] The predominant forms of *B. microti* closely resemble the small rings of *Plasmodium* species. A later tetrad form ("Maltese cross") may be seen and is positive morphologic evidence of babesiosis. Differentiating *Babesia* from *Plasmodium* may be difficult but is possible by noting the absence of pigment deposits in erythrocytes parasitized with the older stages of *Plasmodium* species.

When organisms cannot be detected on blood smears, the diagnosis can made by intraperitoneal inoculation of the patient's blood into splenectomized hamsters. Serologic studies may also be helpful in confirming the diagnosis[295] but are performed in only a few laboratories. A titer greater than 1:64 on IFA testing is considered consistent with seropositivity, and a titer greater than 1:256 is diagnostic of acute infection. No test for circulating *Babesia* antigen is available. PCR testing for *Babesia* can be as sensitive and specific as standard testing and may be reproducible enough for routine use in diagnosis of acute babesiosis.[6,196]

Treatment and Prevention. Chemotherapy for *B. microti* infection consists of a combination of clindamycin and quinine and should reserved for patients with severe disease, those with asplenia or immunosuppression, or older adults.[64] The clindamycin dosage for adults is 1.2 g intravenously twice daily or 600 mg orally three times daily for 7 days. Children should receive oral clindamycin for 7 days (20 to 40 mg/kg/day in three divided doses). The quinine dosage for adults is 650 mg orally three times daily and for children is 25 mg/kg/day in three divided doses, each for 7 days. Parasites are eradicated from the blood with this therapy, but treatment failures have been reported.[311,316] Recently, azithromycin was used as a successful alternative to clindamycin in one treatment failure.[311] Combination therapy with atovaquone and azithromycin is also reported.[194] In seriously ill patients with high levels of parasitemia, exchange transfusion may also produce rapid clinical improvement.[171,347] Treatment of pregnant patients should (when possible) include clindamycin and quinine, both of which cross the placenta; use of quinine in the first trimester should be avoided, however, as high dosages are associated with miscarriage.

Patients with mild clinical disease typically recover without specific anti-*Babesia* chemotherapy, although few patients have been followed longitudinally. A recent prospective study showed that parasitemia lasted for a mean of 82 days in 24 asymptomatic subjects not treated with the standard regimen.[195] Even those receiving clindamycin and quinine had delayed resolution of parasitemia, however, and 9 of 22 subjects had significant side effects from standard therapy. Exchange transfusion with clindamycin–quinine therapy has proved to be effective in three cases of *B. divergens* infection.[35]

Avoidance of ticks is the only currently effective method of preventing babesiosis. Splenectomized patients should be wary of visiting areas endemic for babesiosis.

Crimean-Congo Hemorrhagic Fever

Crimean-Congo hemorrhagic fever (CCHF), also called central Asian hemorrhagic fever, is caused by a Nairovirus (ssRNA) in the Bunyaviridae family. Although these viruses are more of a problem in livestock than humans, human cases carry up to a 50% fatality rate. The incubation period is generally a week if transmitted by tick bite, and 7 to 10 days if contacted by airborne means or from bloody tissue exposure. Most cases occur between June and September. Primary symptoms are sudden onset of fever, headache, weakness, malaise, myalgias, anorexia, and mental status changes. Gastrointestinal symptoms are common, and patients present mainly with abdominal pain, hepatic enzyme elevation, and melena. A petechial rash usually occurs, sometimes leading to large areas of ecchymosis. Some cases proceed to hepatorenal syndrome, coma, and pulmonary failure within the first week. Cases with extensive multiorgan system involvement are often fatal.

Several species of ixodid ticks are vectors; two species, *Hyalomma marginatum* and *Hyalomma anatolicum,* are anthropophilic and the most important vectors with respect to human disease. In areas endemic for CCHF, these ticks are found in very high numbers. Infection may also be transmitted

via aerosols, either from crushing ticks or from the slaughter of infected animals. Nosocomial transmission is reported in hospitals in Africa.

CCHF is diagnosed clinically and confirmed by laboratory tests. Treatment is mostly supportive, although ribavirin has been shown to provide some benefit.[107,380] CCHF is listed by the CDC as a category C bioterrorism agent.

TICK-BORNE DISEASE PREVENTION AND AWARENESS

The best way to prevent tick-borne diseases is to create awareness of the ticks and the diseases they transmit. Proper clothing should consist of lighter-colored clothing, as this makes ticks easier to spot. Avoiding heavily infested areas reduces exposure, as does reducing contact with leaf litter.[59,307] Reducing time in areas frequented by ticks reduces exposure further. A study of the upper surface of downed logs, as would be found and utilized by weary hikers, yielded a large number of *Ixodes scapularis* nymphs.[59] Boots provide better protection than do tennis shoes and socks.[59] Tucking pant legs into the top of the socks reduces the ticks' ability to climb up the legs of potential hosts. A ring of masking tape or duct tape at the top of the socks further reduces exposure.

Insecticides containing diethyl toluamide (DEET) are generally safe, work well, and may be applied directly to the skin. For more protection, a light spray of a permethrin-based acaricidal spray, such as Permanone, to clothing will last up to a month, and through several washings, and it is very toxic to ticks. Permethrin should not be applied to skin, as it has been associated with neurologic sequelae in mammals.[270] A light spray around bedding may deter nighttime feedings. The use of the "four-poster" system in applying acaricides for wildlife or livestock has been shown to reduce tick populations significantly.[58] This system, developed by researchers at Kerrville, Texas, involves creating a feeding station for deer or cattle that has four corner posts on which are rollers soaked in a 10% permethrin solution. When livestock or wild game feed at the station, the permethrin is applied to their heads, an area often heavily infested with ticks. Typically, there is a 92% to 98% reduction in the tick population within 3 years.

One study in Maryland showed a 91% to 100% reduction in adult, larval, and nymphal questing ticks and 70% to 95% reduction in nymphal and larval ticks on mice the first year.[58,318] However, primarily as a result of use of acaricides for livestock on a large scale, ticks and other insects are showing resistance to even the most toxic of acaricidal treatments.[243,278]

Frequent inspection of the body should be performed when in a tick-infested areas. Although some diseases require as much as 24 to 48 hours of tick attachment, others are transmitted within an hour. The argasid ticks feed in minutes, usually while the hosts are sleeping, so avoidance or protection through habitat selection or the use of physical or chemical barriers makes good sense.

Removal of attached ticks should never be done by bare or even gloved hands. A quality pair of fine-tipped forceps is necessary to safely remove embedded nymphal and adult ticks, by gently grasping the ticks as close to the skin as possible and gradually retracting outward in a straight line (Fig. 45-14). Special tick-removal devices available from outdoor and camping supply companies provide some advantage over simple

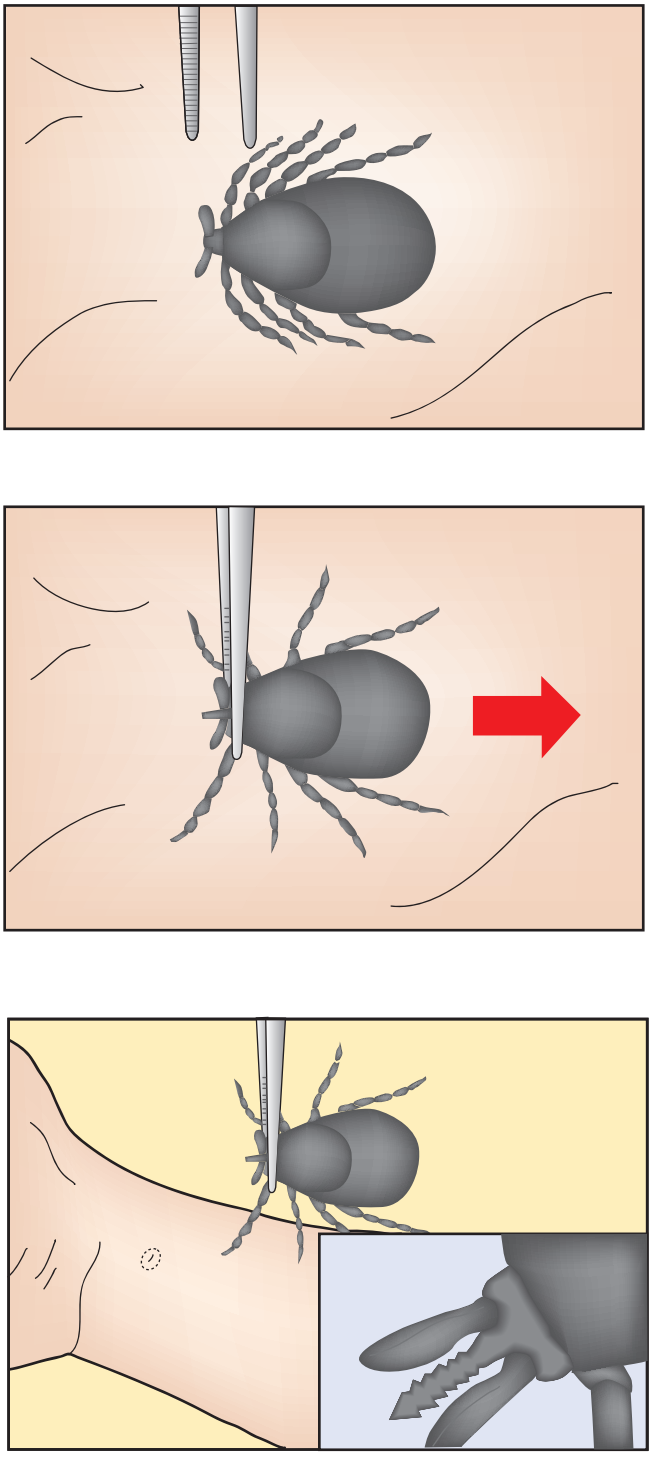

Figure 45-14. Ideal tick removal method. Grasp the tick near the surface of the skin, withdraw from the skin in a steady, constant motion. Do not turn, jerk, or twist. (From Goddard J: Infectious Diseases and Arthropods. Totowa, NJ, Humana Press, 1999, with permission.)

forceps but generally cost more.[344] The area should then be cleaned with a local antiseptic. Other methods for tick removal, such as applying petroleum jelly to the tick, or using a lighted match or cigarette, isopropyl alcohol, or fingernail polish, do not ease removal of the tick. These and other "remedies" most likely increase expression of tick saliva and foregut contents, thus increasing the chance of disease transmission.[20,255] Removal of larval ticks is easier, as their size and mouthparts are extremely tiny. A piece of tape, such as duct tape, applied to the skin, and then removed, easily removes free-crawling or attached larval ticks. Alternatively, the thin edge of a plastic object such as a credit card or driver's license scraped along the skin easily removes attached larvae. This method is appropriate only for larval ticks, not for nymphs or adults.

Laundering clothing in hot water and drying on high heat after being in tick-infested areas generally works to kill ticks and keeps them from invading residences. However, despite exposure to hot water in the wash cycle, ticks often survive. High drying temperatures for 1 hour are generally required to kill ticks.[57] Unfortunately, expensive Gore-Tex and similar outdoor clothing items do not tolerate high-heat drying. Free-crawling ticks can easily be transported from the field to a vehicle during a drive home from an endemic area, and then from the vehicle to the residence.

For homeowners or landowners, proper landscaping, with an appropriate barrier or buffer zone of wood chips or gravel between forested areas and formal lawn areas, has been shown to reduce the potential tick–human interaction.[97,156]

The references for this chapter can be found on the accompanying DVD-ROM.

46 Spider Bites

Leslie V. Boyer, Greta J. Binford, and Jude T. McNally

INTRODUCTION TO SPIDERS

The spiders number approximately 34,000 described species and are found in all habitats except the open sea.[36,57,185] They are carnivorous predators with important ecologic roles in most terrestrial ecosystems. Many are capable of wind-borne dispersal (ballooning), which has led to colonization of even the most isolated land masses on Earth.

As with ticks, mites, scorpions, and other arachnids, the spider's body consists of an abdomen and an unsegmented cephalothorax (prosoma) with chelicerate jaws, pedipalps, and four pairs of legs (Fig. 46-1). They are distinct from other arachnids in that they have no abdominal segmentation and the male pedipalpal tarsi are modified as secondary genitalia.[224] In addition, spiders have venom that is produced in a gland in the anterior prosoma and delivered through a cheliceral fang. On the abdomen, they have silk-producing glands and a set of spinnerets.

The primary function of venom in spiders is prey capture or, rarely, defense. Venoms are complex mixtures of neurotoxic and proteolytic peptides, proteins, and biogenic amines.[1,15,88,165,224] Most of these toxins are target specific, acting selectively on arthropods, vertebrates, or other groups, including some with mammalian-specific activity.[1,106,181] Venom composition varies widely across spider species, and variation may exist within species between sexes and among geographically isolated populations.[162] Venom potency also varies within individuals, both seasonally and developmentally. Despite this tremendous venom diversity, only a few dozen spider species are considered harmful to humans because (1) most others have an insufficient quantity of venom, (2) the toxins do not affect mammals, or (3) the fangs cannot penetrate human skin. In a few species, although laboratory evidence suggests potential mammalian toxicity, human envenomations have not been reported, perhaps because of the rarity of encounters between spiders and humans in some habitats. Table 46-1 lists an assortment of spider families, including those with species that have been reported to bite humans.

All spiders are carnivorous and have the challenge of capturing live prey. Prey capture is a multistep process in which spiders must find prey, ensnare it, immobilize it, and digest it externally before the liquefied meal can be consumed. This process and the role of venom are very diverse. The neurotoxins in spider venom are primarily used for prey immobilization. Venoms of a few spiders are known to have some proteolytic components that likely begin the process of external digestion. The bulk of digestion, however, results from digestive enzymes that are ejected from the mouth, a distinctly separate opening from the venom duct (Fig. 46-2). Occasionally, spider digestate may infect wounds created by spider bites, which could in theory affect clinical appearance, although no direct evidence substantiates this suspicion.

In addition to venom, some theraphosids (tarantulas) produce urticating hairs that irritate skin or mucous membranes of

forceps but generally cost more.[344] The area should then be cleaned with a local antiseptic. Other methods for tick removal, such as applying petroleum jelly to the tick, or using a lighted match or cigarette, isopropyl alcohol, or fingernail polish, do not ease removal of the tick. These and other "remedies" most likely increase expression of tick saliva and foregut contents, thus increasing the chance of disease transmission.[20,255] Removal of larval ticks is easier, as their size and mouthparts are extremely tiny. A piece of tape, such as duct tape, applied to the skin, and then removed, easily removes free-crawling or attached larval ticks. Alternatively, the thin edge of a plastic object such as a credit card or driver's license scraped along the skin easily removes attached larvae. This method is appropriate only for larval ticks, not for nymphs or adults.

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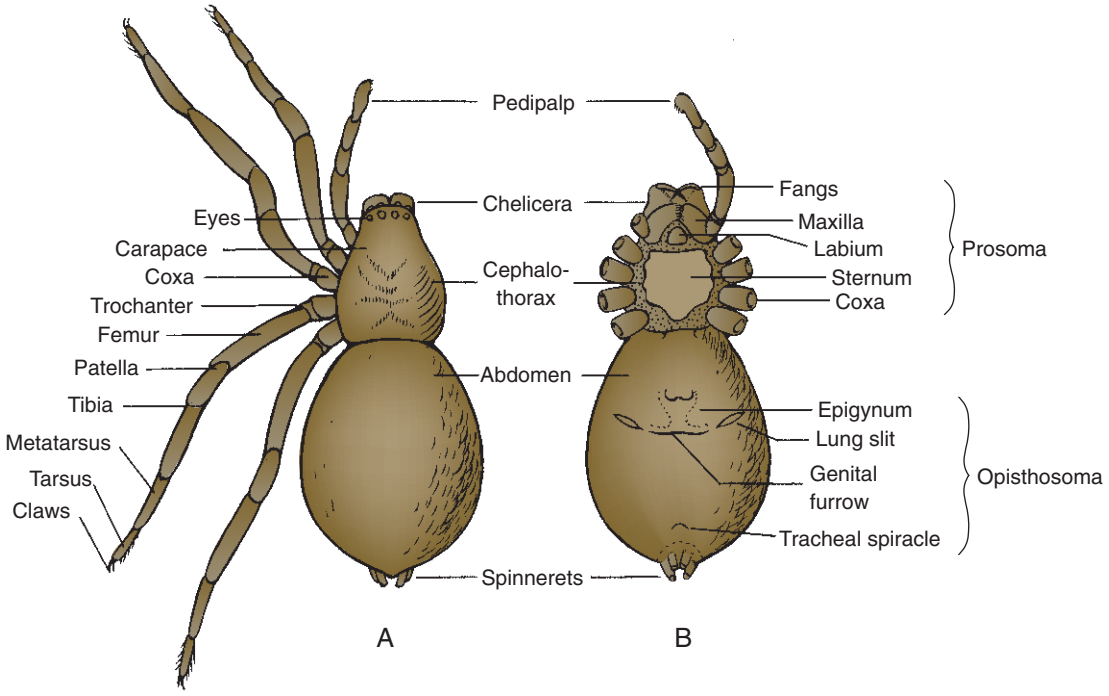

Figure 46-1. External anatomy of an araneomorph spider. **A,** Dorsal. **B,** Ventral (legs omitted).

animals or humans. Exposure may result from direct contact with a spider.

GENERAL ASSESSMENT AND TREATMENT OF SPIDER BITES

Awareness of the differential diagnosis is crucial for management of any patient presenting for evaluation of "spider bite," because the offending creature is rarely observed and identified. In general, it is outside of normal biologic activity for spiders to bite humans, except in defense. A defensive bite risks the spider's life and tends to occur only when its life is threatened by being crushed. Thus true spider bites are much less common causes of skin lesions than are insect bites or cutaneous infections. Furthermore, no pathognomonic clinical signs prove the diagnosis without retrieval and identification of a spider that was seen actually biting.[208] Diagnosis of arachnidism without direct evidence can lead to inappropriate treatment and the lack of consideration of more severe underlying medical issues.[139,142,144,203,246] Therefore the medical history, physical examination, and laboratory evaluation of putative spider bites must usually take into consideration an alternative etiology. Treatment plans should include careful follow-up and patient counseling to manage any uncertainties in the final diagnosis.

The differential diagnosis of a local lesion may include fungal, bacterial, and viral infection, especially herpes simplex and zoster; the vesiculobullous diseases; arthropod-borne infectious diseases (e.g., Lyme disease); other bites and stings; foreign body reactions; and systemic conditions that predispose to focal skin lesions (e.g., diabetes mellitus, leukemia, lupus erythematosus). First-aid interventions by the patient may mask an otherwise benign process by superimposition of trauma, burns, or chemical irritation. Systemic signs and symptoms may require differentiation from the effects of snake or scorpion neurotoxin, pesticide toxicity, sepsis, meningitis, hemolytic anemia, or acute abdomen, depending on the circumstances.

The medical history should include details of the bite circumstances to demonstrate consistency with expected spider habitat and behavior. This includes location indoors or outside, time of day or night, and human activity at the time of injury. The victim should attempt to recall the appearance of the involved arthropod. If it was believed killed through garments or bedclothes, an attempt should be made to retrieve its remains. Crushed spider parts can be examined and identified by arachnologists or entomologists at many universities and museums. Until identified, spiders may be preserved in 70% to 80% ethanol. The evolution of subsequent wound and systemic symptoms should be noted, along with modifiers, including home treatment and underlying health of the patient. If the local geography lacks species consistent with the suspected pathophysiology, the recent travel history of household contacts should be considered.

Physical examination includes particular attention to the bite site, as well as general assessment for systemic effects. Local findings of importance include anatomic location (spiders are more likely to bite defensively at sites where clothing binds tightly; thin skin is more readily envenomed than callous skin) and number of separate lesions (multiple bites suggest parasitic insect bite rather than spider bite). Central punctae, vesicles, or erosions should be noted, as well as the pattern of peripheral changes, including erythema, pallor, hemorrhage, induration,

TABLE 46-1. Spiders of Clinical Interest

SCIENTIFIC NAME (NUMBER OF GENERA AND SPECIES): COMMON NAME	GEOGRAPHIC RANGE	CLINICAL REMARKS	TAXONOMIC REFERENCES	CLINICAL REFERENCES
Phylum Arthropoda, Class Arachnida, Order Aranea (3 Suborders, 105 families, 3067 genera, approx. 34,000 species): Spiders	Worldwide		57, 186	
Suborder Mesothelae (1 family, 2 genera, 40 species)	Asia (part)	No species believed clinically relevant	187	
Suborder Mygalomorphae (15 families, 260 genera, 2200 species): Mygales	Worldwide		193	
Theraphosidae (86 genera, 800 species): tarantulas and baboon spiders	Worldwide		193	
Aphonopelma (Rheochostica): tarantula	New World	Minor local reaction; urticating hairs	193	
Rheochostica henzi (previously *Dugesiella henzi*): tarantula	New World		193	
Mygalarachne (previously *Sericopelma*)		Minor local reaction	193	
Pterinochilus: tarantula	Africa	Minor local reaction	193	
Pamphobaetus: tarantula	Brazil	Minor local reaction	193	
Avicularia: tarantula	New World	Minor local reaction	193	
Lasiodora: tarantula	Brazil	Minor local reaction	193	
Grammostola cala: Chilean rose tarantula	New World	Minor local reaction; urticating hairs	193	35, 60, 61
Acanthoscurria: tarantula	New World	Minor local reaction; urticating hairs	193	
Euathlus (previously *Brachypelma*): tarantula	New World	Minor local reaction; urticating hairs	193	
Harpactirella lightfooti: baboon spider or bobbejaan-spinnekop	Africa		193	39, 174, 175
Poecilotheria: tarantula	Australia	Minor local reaction	193	
Selenocosmia	East Indies, India, Australia		193	
Phormictopus	South America, Caribbean		193	
Lampropelma: tarantula	Asia, India	Minor local reaction	193	
Haplopelma minax: Thailand black tarantula	Asia, India	Urticating hairs	193	35
Hexathelidae (11 genera, 74 species): funnel-web mygalomorphs			193	
Atrax	Eastern Australia		193	
Atrax robustus: Sydney funnel-web spider	Eastern Australia	Local pain; severe neurotoxicity	193	77, 78 113, 234, 235
Hadronyche (Atrax)	Eastern Australia		193	
H. formidabilis: northern funnel-web spider	Eastern Australia	Local pain; severe neurotoxicity	193	11, 117
H. versutus: Australian Blue Mountains funnel-web spider	Eastern Australia	Local pain; severe neurotoxicity	193	11
H. infensus	Eastern Australia	Local pain; severe neurotoxicity	193	11, 99
H. cereberus	Southeastern Australia	Local pain; severe neurotoxicity	193	
Dipluridae (17 genera, 157 species): funnel-web mygalomorphs	Worldwide		193	
Trechona venosa	South America	Mammalian toxin; no human reports	193	
Idiopidae (18 genera, 267 species): front-eyed trapdoor spiders	Pantropical, India		193	
Aganippe subtristis	Australia	Minor local reaction	193	
Arabantis	Australia		193	
Mysgolas (previously *Dyarcyops, Hermea*)	Australia, New Zealand	Minor local reaction	193	

TABLE 46-1. Spiders of Clinical Interest—cont'd

SCIENTIFIC NAME (NUMBER OF GENERA AND SPECIES): COMMON NAME	GEOGRAPHIC RANGE	CLINICAL REMARKS	TAXONOMIC REFERENCES	CLINICAL REFERENCES
Ctenizidae (10 genera, 116 species): trapdoor spiders	Worldwide, except South America		193	
Bothriocyrtum	North America		193	
Ummidia	North, Central America		193	
Actinopididae (3 genera, 41 species): trapdoor spiders	Australia, Central and South America		193	
Missulena bradleyi: mouse spider	Australia	Mammalian toxin; one case of systemic effects	193	126, 132
Suborder Araneomorphae (90 families, 2700 genera, 32,000 species): true spiders				
Filistatidae (12 genera, 87 species)	Worldwide		148	
Filistata				
Sicariidae (2 genera, 129 species): recluse, brown, or fiddle spiders	Worldwide		187	
Loxosceles: recluse, brown, or fiddle spiders	New World, Mediterranean, Africa		91	10, 20
L. reclusa: brown recluse spider	North America (central, southeast)	Local necrosis; systemic syndrome	91	97, 190
L. arizonica: Arizona brown spider	North America (southwest)	Local necrosis; systemic syndrome	91	
L. rufescens	Worldwide		91, 148a	
L. intermedia	Brazil		39a	
L. gaucho	Brazil		39a	
L. spinulosa	South Africa			
L. laeta: corner spider or spider behind the pictures	South America, Australia, Finland	Painful local lesion; systemic syndrome	91, 148	10
L. parrami	South Africa		174a	
Sicarius: six-eyed crab spiders	Neotropics, South Africa	Local necrosis; DIC	174–176	
Dysderidae (20 genera, 371 species): giant-fanged, six-eyed spiders	Worldwide		62c	
Dysdera	Worldwide		62c	
Desidae (28 genera, 200 species): long-jawed intertidal spiders	Worldwide		204a	
Badumna insignis: black house spider	Australia, New Guinea	Local pain, rarely necrosis	103a	153, 275, 276
Zodariidae (350 species): hunting spiders	Worldwide			
Supunna picta	Australia	Minor local reaction		37
Tetragnathidae (50 genera, 900 species): long-jawed orb-weaving spiders	Worldwide		149i	
Nephila: golden-silk spiders	Pantropical		149f	
Argyronetidae (1 genus, 1 species): water spiders	Europe		204a	
Argyrontea aquatica	Europe		204a	
Gnaphosidae (111 genera, 2156 species): ground and mouse spiders	Worldwide		186	
Herpyllus ecclesiasticus: parson spider	North America	Minor local reaction; brief systemic effects	113a	154
Drassodes	Worldwide		185a	
Lamponidae (1 genus, 50 species): white-tailed spiders	Australia, Tasmania, New Zealand		186	
Lampona cylindrata: white-tailed spiders	Australia, New Zealand	Minor local reaction; rarely necrotic	186	102, 128, 275
Miturgidae (23 genera): forest floor and cave spiders	Worldwide			
Miturga	Australia, New Zealand			
Heteropodidae (82 genera, 850 species): giant crab, huntsman, and large, wandering crab spiders			79a	
Palystes natalius: lizard-eating spider	South Africa	Minor local reaction	62a	174

Continued

TABLE 46-1. Spiders of Clinical Interest—cont'd

SCIENTIFIC NAME (NUMBER OF GENERA AND SPECIES): COMMON NAME	GEOGRAPHIC RANGE	CLINICAL REMARKS	TAXONOMIC REFERENCES	CLINICAL REFERENCES
Heteropoda	Worldwide			
Isopeda	Australia, New Guinea, East Indies	Minor local reaction		
Olios	America, Australia	Local pain, brief nausea	205a	
Delena cancerides: social huntsman spider	Australia	Minor local reaction		
Oxyopidae (9 genera, 400 species): lynx spiders	Worldwide		65c	
Peucetia viridans: green lynx spider	North and Central America, West Indies	Minor local reaction	37a	40
Salticidae (475 genera, 4500 species): jumping spiders	Worldwide		68a, 190a	
Breda jovialis	Australia	Minor local reaction		
Phidippus	New World	Local pain, minor ulceration		206
Holoplatys	Australia	Minor local reaction, itch, headache		
Mopsus	Australia			
Thiodina	America			
Opisthoncus	Australia	Minor local reaction		
Thomisidae (160 genera, 1960 species): crab spiders	Worldwide		65a, 218a	
Misumenoides	Americas		218a	
Ctenidae (37 genera, 472 species): wandering spiders	Worldwide			
Phoneutria: armed spiders	South America			
P. nigriventer: Brazilian armed spider	Brazil, Paraguay	Local pain; systemic neurotoxin	69a	214, 215
Elassoctenus harpax	Australia			
Cupiennius	South and Central America, West Indies		155a	
Lycosidae (76 genera, 2196 species): wolf spiders	Worldwide		65c, 284	
Lycosa: wolf spiders	Worldwide	Local pain		45, 125, 201
L. raptorial	South America	Local pain		
L. tarentula: "tarantula"	Palearctic	Subject of folklore		
L. godeffroyi	Australia	Local pain		
L. erythrognatha				
Clubionidae (25 genera, 590 species): sac, running spiders, and two-clawed hunting spiders	Worldwide		65b	
Cheiracanthium: sac and running spiders	Worldwide			
C. mildei	Holarctic		65b	
C. punctorium	Europe	Minor local reaction; fever	228a	159, 160
C. japonicum	Japan, China	Minor local reaction; brief systemic illness	282b	182
C. longimanus	Australia	Minor local reaction; brief systemic illness		29
C. lawrencei	Africa	Local necrosis; systemic syndrome		174, 175
C. mordax	Australia	Minor local reaction, brief systemic illness		29
C. inclusum	America	Local pain, nausea	65b	29
Corinnidae (51 genera, 659 species): sac spiders	Worldwide		187a, 282a	
Trachelas		Minor local reaction	187a	184, 252
Supunna picta	Queensland	Minor local reaction	62b	
Tengellidae (5 genera, 18 species): running spiders	New World			
Liocranoides	Appalachia, California			
Agelenidae (41 genera, 600 species): grass and funnel-web spiders	Worldwide		204a	

TABLE 46-1. Spiders of Clinical Interest—cont'd

SCIENTIFIC NAME (NUMBER OF GENERA AND SPECIES): COMMON NAME	GEOGRAPHIC RANGE	CLINICAL REMARKS	TAXONOMIC REFERENCES	CLINICAL REFERENCES
Tegenaria agrestis: hobo or northwestern brown spider	Europe, America (Pacific Northwest)	Local necrosis; systemic syndrome	205	2, 258, 260, 262
Agelenopsis aperta: grass spider or funnel-web spider	Southwest United States, Mexico		50a	
Zoridae (12 genera)				
Diallomus	Australia			
Superfamily Orbicularia (13 families, 10,300 species)				
Theridiidae (62 genera, 2000 species) comb-footed spiders	Worldwide		282a	
Latrodectus: widow spiders	Worldwide	Local pain; systemic neurotoxicity	149a, 149g	
L. mactans: black widow spider	North America		149g, 149k	55
L. mactans hasselti: redbacked spider	Australia		250a	134, 274
L. hesperus: black widow spider	North America			54
L. tredecimguttatus	Mediterranean		149k	158
L. pallidus	Libya to Russia		149k	
L. indistinctus: black button spider	Africa			168
L. geometricus: brown button spider	Worldwide		149k	168
Steatoda	Worldwide		149b	
S. paykulliana: false black widow spider	Europe, Mediterranean		149j	270
S. nobilis	England	Local pain; mild systemic effects		
S. foravae: false button spider	Southern Africa	Minor local reaction		170
Achaeranea tepidariorum	Worldwide	Trivial local reaction	149c	
Araneidae (155 genera, 2600 species): orb-weaving spiders	Worldwide			
Argiope: argiopes	Worldwide			253
A. argentata	America	Local pain; vesicle	149h	
A. trifasciata	Worldwide		149h	
Araneus: cross, garden, and shamrock spiders	Worldwide		149d, 149e	
Eriophora biapicata	Australia	Minor local reaction		
Neoscona: orb-weaving spiders	Worldwide		28a	
Phonognatha graeffei	Australia	Trivial local reaction		

DIC, Disseminated intravascular coagulation.

tenderness or anesthesia, and local lymphatic involvement. Systemic findings, depending on the species involved, may include changes in vital signs, diaphoresis, generalized rash, facial edema, gastrointestinal distress, muscle fasciculations, spasm or tenderness, or altered mental status.

The laboratory evaluation for envenomation is usually simple, seldom requiring more than complete blood count and urinalysis. Assessment for other elements of the differential diagnosis, however, may be much more elaborate. Depending on circumstances, this may include viral, bacterial, or fungal culture; Lyme disease titer; radiography of the abdomen or of the injured part; stool test for occult blood; electrocardiogram; or skin biopsy.

General supportive measures are the mainstays of therapy for most spider bites. These include basic local hygiene, tetanus prophylaxis, analgesics, hydration, and surgical follow-up if indicated for debridement and management of extensive necrotic lesions. Corticosteroids are of unproven benefit and are generally not indicated. Antibiotics, although not of value for simple venom injury, are prescribed when bacterial cellulitis cannot be eliminated from the differential diagnosis. Specific measures, including antivenom, for treatment of envenomation by particular spider species are discussed later in this chapter.

GUIDE TO SPIDER DIVERSITY AND IDENTIFICATION

This chapter provides more information on spider diversity than most reviews in order to (1) inform the reader of the immense diversity of spiders that are medically insignificant and thus emphasize the rarity of spiders known to cause medical problems; (2) emphasize the need for accurate species identification, particularly in reporting of cases for publication or teaching; and (3) facilitate accurate identification of spiders that are caught in the act of biting by directing professionals to proper

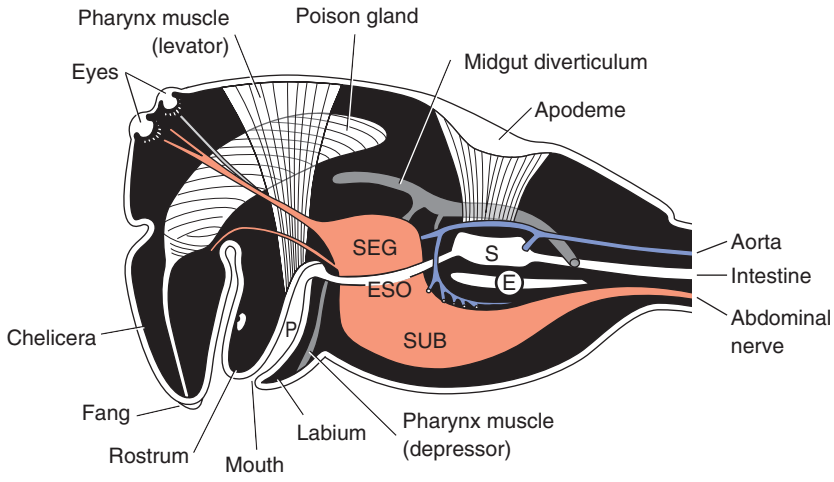

Figure 46-2. Longitudinal section of the prosoma. E, endosternite; Eso, esophagus; P, pharynx; S, sucking stomach; SEG, supraesophageal ganglion; SUB, subesophageal ganglion. (Modified from Foelix RF: Biology of Spiders, 2nd ed. Oxford, UK, Oxford University Press, 1996, with permission.)

identification keys. Table 46-1 includes species worldwide that are currently known or suspected to be medically noteworthy, even minimally, either through the effect of bites or urticating hairs. Geographic range and the most recent systematic work on each group are included. Box 46-1 lists genera of serious medical significance for each continent. For effective communication, groups of spiders must be recognized by the same name worldwide. This chapter uses the current official nomenclature.

Of the three spider suborders, two contain clinically significant species: Mygalomorphae and Araneomorphae. Mygalomorphs include the baboon spiders or tarantulas, trapdoor spiders, purse-web spiders, mygalomorph funnel-web spiders, and several other groups that lack common names. Most spiders are araneomorphs, including jumping spiders, orb-weaving spiders, widow spiders, wolf spiders, and fiddleback spiders (see Table 46-1). The most conspicuous characteristics that distinguish these groups are the orientation of the chelicerae (jaws) and the number of book lungs. Spider fangs are located on the chelicerae, which open sideways in the Araneomorphae and move diagonally in the Mygalomorphae, requiring the latter to rear back for a downward, snakelike strike (Figs. 46-3 and 46-4). Mygalomorphae have two pairs of book lungs, whereas most Araneomorphae have only one pair. Lung slits, which open into the book lungs, are easily visible in a ventral view of the anterior abdomen (see Figure 46-1). Characteristics that distinguish families, genera, and species include eye number and pattern, numbers of tarsal claws, and details of genitalic structure.

Box 46-1. Clinically Important Spider Genera by Geographic Distribution[2,107,215,244]

NORTH AMERICA
Loxosceles
Latrodectus
Tegenaria

SOUTH AMERICA
Loxosceles
Latrodectus
Phoneutria

AFRICA
Loxosceles
Latrodectus

EUROPE
Loxosceles
Latrodectus

AUSTRALIA
Atrax
Hadronyche (Atrax)
Latrodectus

ASIA
Latrodectus

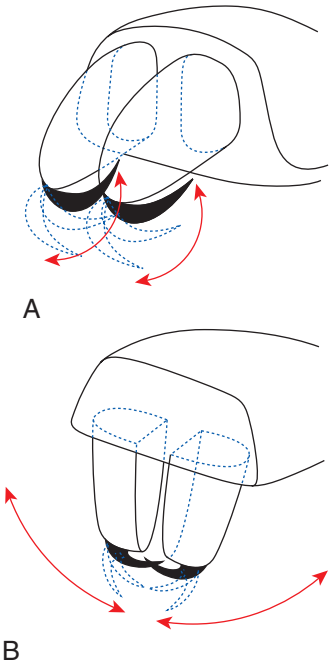

Figure 46-3. Movement of the chelicerae in mygalomorphs (**A**) and araneomorphs (**B**). (Modified from Foelix RF: Biology of Spiders, 2nd ed. Oxford, UK, Oxford University Press, 1996, with permission.)

Figure 46-4. Fangs of mature male tarantula (*Aphonopelma* species). (Courtesy Michael Cardwell & Associates, 1992.)

Although many spider species are geographically localized (e.g., *Atrax robustus* in Australia, *Phoneutria nigriventer* in Brazil), some, such as the widow spiders *(Latrodectus),* are found worldwide (see Table 46-1). Others, such as members of the *Loxosceles* genus, are widely distributed in more than one continent, and still others, such as *Tegenaria agrestis,* appear to have naturalized in specific geographic regions distant from their point of ecological origin. Because of the resulting worldwide species diversity and overlap, the remainder of this chapter is structured according to spider taxonomy rather than isolated species or geographic location.

Figure 46-5. Theraphosidae. (Courtesy Gita Bodner.)

SUBORDER MYGALOMORPHAE

Mygalomorphs make up less than 10% of all spider species. They are found worldwide, with greatest abundance and diversity in tropical regions. Tarantulas (Theraphosidae) are the most famous mygalomorphs and include the largest spiders known, reaching up to 10 cm in body length. Most mygalomorphs are smaller, some less than 1 mm in adult body length. Most live multiple years (some up to 20), and females continue to molt after reaching adulthood. They have diverse habits but typically live in silk-lined burrows or silken tubes. For the purpose of prey capture, mygalomorph silk is only slightly sticky relative to araneomorph silk and is generally used to signal the presence of prey rather than to ensnare them. Once prey are detected, spiders run out of their retreat, seize prey in their jaws, and return to the retreat to feed. Individuals wander when dispersing, and males leave their retreats in search of females during mating season.

Family Theraphosidae: Tarantulas and Baboon Spiders

Theraphosidae is the largest mygalomorph family with respect to both numbers of species and sizes of the largest individuals (Fig. 46-5). Approximately 800 described species are found on all continents, with greatest abundance and diversity in tropical regions. They mature in 3 to 9 years and can live for 15 to 25 years. Individuals live in burrows, with trip-line threads extending from the entrance. These are sometimes located in abandoned rodent burrows or hollow trees. Theraphosids have dense tufts of specialized hairs on their tarsi (feet) that enable them to climb on smooth surfaces and may aid in prey capture. They have two tarsal claws, eight closely grouped eyes, and two pairs of spinnerets. As a group, they are found mainly in tropical and subtropical areas.

Confusingly, the word *tarantula* was first applied to *Lycosa tarentula,* a species of European spider actually belonging to the wolf spider family, or Lycosidae, which are properly classified within the suborder Araneomorphae, described later in this chapter. In the United States, the term *tarantula* usually refers only to the large spiders of the family Theraphosidae, suborder Mygalomorphae.

Figure 46-6. Mature female *Aphonopelma iodium.* (Courtesy Michael Cardwell & Associates, 1997.)

Grammostola mollicoma is the largest tarantula known, with a body length of 7 to 10 cm and leg spread of 21 to 27 cm.[39,92] *Harpactirella lightfooti,* the baboon spider or bobbejaanspinnekop, is a mygalomorph spider found in South Africa. Body length is 3 cm; the cephalothorax is brown with a yellowish border.

Venom

Few tarantula venoms have been studied systematically. In the United States, *Rheochostica henzi* and members of the genus *Aphonopelma* (Fig. 46-6) have venom containing hyaluronidase, nucleotides, and polyamines.[44,51,148,212,213] Polyamines are thought to act as neurotransmitters and increase venom effectiveness, particularly with respect to paralysis of insect prey.[224] Hyaluronidase is postulated to be a spreading factor, and the nucleotide adenosine triphosphate (ATP) potentiates the major effects of the venom on mice. Both venoms cause rapid, irreversible necrosis of skeletal muscle when injected intraperitoneally into mice.[183] *Dugesiella (Rheochostica)* venom was found to have a necrotoxin with several similarities to sea snake venoms.[148] In comparison, the venom of *Scodra griseipes,* an African tarantula, includes higher molecular-weight (greater than 25,000 daltons) proteins and enzymes plus lower weight polypeptides (4000 to 9000 daltons); the second group is believed to contain polypeptide neurotoxins.[50] *S. griseipes* venom toxins have a mammalian effect, but this species is not known to be clinically relevant. Recent work suggests that venom chemotaxonomy may be a useful method of nondestructive species recognition, at least within the *Brachypelma* genus.[72]

Urticating Hairs

Several genera of tarantulas, including *Haplopelma, Lasiodora, Grammostola, Acanthoscurria,* and *Brachypelma,* possess urticating hairs irritative to skin and mucous membranes. These genera are located throughout the western hemisphere, with many species indigenous to the United States. When one of these spiders is threatened, it rubs its hind legs across the dorsal surface of its abdomen and flicks thousands of hairs toward the aggressor. These barbed hairs can penetrate human skin, causing edematous, pruritic papules. The itching may persist for weeks. There are four morphologic types of urticating hairs. Tarantulas within the United States possess only type I hairs, which do not penetrate the skin as deeply as type III hairs. Type II hairs are incorporated into the silk web retreat and are not thrown off by the spider. Type III hairs can penetrate up to 2 mm into human skin; this is the type of hair most likely to cause inflammation. They are typically found on Mexican, Caribbean, and Central and South American species. Type IV hairs, which belong to the South American spider *Grammostola,* are able to cause inflammation of the respiratory tract in small mammals. Rats and mice have been reported to die of asphyxia within 2 hours after exposure to the hairs.[60,275]

Clinical Presentation

Despite the presence in venom of components toxic to rodent nerves and skeletal muscle, most tarantula bites result in only mild to moderate local symptoms in humans. A few can cause more severe pain and swelling, numbness, or lymphangitis. In a series of nine cases in Australia, by either *Selenocosmia* species or *Phlogiellus* species, no major effects occurred. Local pain was the most common effect, followed by puncture marks or bleeding. Severe pain occurred in 4 of 7 patients, where severity of pain was documented. Mild systemic effects occurred in one case.[127] Species of tarantula implicated as causes of human envenomation include those in genera *Mygalarachne* (formerly *Sericopelma*) of Panama, *Pterinochilus* of Africa, *Aphonopelma* of Mexico and the United States, *Pamphobaeteus* of South America, *Euathlus* of Costa Rica, *Theraphosa* of French Guyana, *Grammostola* of Colombia, *Selenocosmia* and *Phlogiellus* of Australia, *Poecilotheria* of India, *Lampropelma* of Thailand, *Lasiodora* of Brazil, and *Avicularia* of Central America and the southwestern United States. Envenomation usually involves immediate pain at the bite site, occasionally followed by some redness and swelling, and usually without necrosis or serious sequelae.[39,44,51,90,213]

A paucity of reports in the peer-reviewed medical literature hinders proper characterization of occasional tarantula neurotoxicity, particularly in cases (e.g., *Poecilotheria*) that are reported anecdotally, and in the pet trade, to provoke severe neuromuscular symptoms (personal communication, Leslie Boyer, 2004) Although no fatalities have been reported, localized pain followed by emesis, weakness, and collapse has been noted after envenomation by *Harpactirella lightfooti,* the baboon spider of South Africa.[39,109,174]

Urticating hairs may cause intense inflammation, which may remain pruritic for weeks. Individuals who handle tarantulas may unwittingly transfer urticating hairs from hand to eye, causing keratoconjunctivitis or ophthalmia nodosa. Keratoconjunctivitis has been described after handling of a Thailand black tarantula, *Haplopelma minax*. Fine intracorneal hairs were noted at examination, and inflammation settled quickly with topical corticosteroid treatment; at 36-month follow-up the eye was normal.[35]

More severe ophthalmic complications occurred in two cases after handling of Chilean rose tarantulas, *Grammostola cala*. In these victims, initial findings were similar, with intracorneal hairs and keratoconjunctivitis, but progressive panuveitis followed, with corneal granulomas, iritis, cataract, hyalitis, and chorioretinitis apparently related to migration of hairs through the media of the eye. The differences in outcome with exposure to the two species may result from differences in hair morphology, which may also explain differences in other reports of

Figure 46-7. Funnel-web spider (*Atrax* species) wearing an engagement ring. (Courtesy Sherman Minton, MD.)

ophthalmic injury from tarantula or caterpillar hair exposure.[35] Similar cases of ophthalmia nodosa have been described.[24,147]

Treatment

Theraphosid bite management is symptomatic. Elevation and immobilization of the extremity and oral analgesics may help reduce pain. All bites should receive local wound care and appropriate tetanus prophylaxis.

Urticating hairs can be removed from skin with repeated application and removal of sticky tape, followed by copious irrigation if necessary; exposed eyes should be irrigated primarily. Topical or systemic corticosteroids and oral antihistamines may also be useful for urticating hair reactions.

Family Hexathelidae: Funnel-Web Mygalomorphs

The family Hexathelidae includes 11 genera and approximately 74 species, which are currently known from the Old World and Chile.[193] The funnel webs that typify members of this group are silk-lined tubular retreats that extend into a protected space, such as a burrow in the ground or a hole in a tree. Sheets of silk radiate from the retreat and signal the presence of prey. These webs superficially resemble webs of araneomorph spiders in the family Agelenidae. Distinguishing characteristics of the spiders include a shiny carapace; long, spiny sensory hairs on the legs; and paired claws lacking claw tufts on the tips of the feet, with teeth lining the medial claw.

Clinically significant hexathelid spiders are species of *Atrax* (Fig. 46-7) and *Hadronyche*. Technically, taxonomists now consider *Atrax* and *Hadronyche* species as members of only one genus.[193] To avoid confusion in the literature, species names still use either *Atrax* or *Hadronyche,* depending on their original names. We discuss these species as a cohesive taxonomic group. Among these, the species *Atrax robustus* is best known and most carefully studied. *Atrax* and *Hadronyche* species, all of which are believed to be dangerous, have been described in southern and southeastern Australia, Tasmania, Papua New Guinea, and the Solomon Islands. As a group, they prefer cool, moist coastal and mountainous regions.[103,104,220]

Genus *Atrax/Hadronyche*

Biology. Funnel-web spiders have a glossy ebony cephalothorax and velvety black abdomen. The abdominal undersurface may have brushes of red hair. The fangs reach 4 to 5 mm in length and are capable of penetrating a fingernail or a chicken's skull, sometimes making removal of the spider difficult. Females are somewhat larger than males, with a body length of 4 cm. Mature males are more delicate, with a tibial spur on the second pair of legs and pointed pedipalps.[124,215]

Atrax robustus, the Sydney funnel-web spider, is limited to a 160-km range around the center of Sydney, Australia. The spider creates a tubular or funnel-shaped, silk-lined shallow burrow under rocks, logs, fences, stumps, or thick vegetation or around foundations of houses. Colonies of up to 150 spiders have been found. Females rarely roam far from their webs; males live a vagrant life after reaching maturity. Wandering males may enter houses or other areas of human habitation, especially during the summer months after a heavy rain. Its aggressive behavior and potent venom make the male Sydney funnel-web arachnid one of the most dangerous spiders in the world. It is responsible for all known fatal *Atrax* envenomations.[2,107,215,244]

Hadronyche formidabilis, the northern funnel-web spider, is found in the central coastal region of New South Wales and the adjacent Blue Mountains. Its tree-dwelling habit was once thought to be unique, but it is now known that other species also live in trees. The webs may be camouflaged in rough-barked trees, such as melaleuca (paper bark), banksia, and eucalyptus.[104]

Venom. Although many species have venom with significant in vitro toxicity, few have been implicated in human illness. The best described of these is *A. robustus*. *Atrax* venom causes widespread release of neurotransmitters.[68,104,112,227,232] This may occur by a direct action of the venom on nerve membranes, producing spontaneous action potentials and consequently provoking a global outpouring of transmitters that accounts for the neuromotor and autonomic stimulation seen clinically.

Early efforts to purify the active component of *Atrax* venom resulted in reports of neurotoxins of various molecular weights purified from venom preparations in separate experiments.[231] These were termed *atraxotoxin* (10,000 to 25,000 daltons, from milked venom),[104] *robustoxin* (4887 daltons, also from milked venom),[221] and *atraxin* (9800 daltons, from ground venom glands).[105] Venoms of *A. robustus, H. cereberea, H. formidabilis, H. infensa, H. lanunstian, H. variela, H. versula,* and an *Atrax* species preparation and *Hadronyche* species preparation have similar high- and low-molecular-weight protein electrophoretic patterns and Western blotting, suggesting that all of these species contain similar atraxotoxins. The relationships among these toxins are not clear, but atraxotoxin or atraxin may be a precursor of robustoxin. In addition to these components, *Atrax* venom contains various lower-molecular-weight compounds, including citric acid, lactic acid, phosphoric acid, glycerol, urea, glucose, gamma-aminolevulinic acid, glycine, spermidine, spermine, tyramine, octopamine, and 5-methoxytryptamine.[67]

The best characterized of the toxins is robustoxin, whose 42-amino acid sequence was determined in 1985.[221] It is the sole lethal toxin that can be isolated by cation-exchange chromatography, and its effects in monkeys duplicate the effects of crude venom preparations. A 5 mg/kg intravenous (IV) dose of robustoxin to monkeys causes dyspnea, blood pressure fluctuations culminating in severe hypotension, lacrimation, salivation, skeletal muscle fasciculation, and death within 3 to 4 hours of administration.[171]

Isolated human intercostal muscles were studied to determine the etiology of muscle fasciculations. Muscles treated with *A. robustus* venom developed marked contractions, which were abolished by D-tubocurarine.[46] Muscles treated with venom for more than an hour stopped contracting and could be stimulated only by increasing the stimulus duration. *A. robustus* venom has been shown to lack anticholinesterase activity.[230] Contractions are not a direct venom action on the muscle fiber, so acetylcholine appears to have been released from the presynaptic terminals.[83] Muscle fasciculations are apparently caused by abnormal repetitive firing of motor neurons. It was hypothesized that the venom changes the membrane's electric field, activating sodium channels without altering the transmembrane potential or damaging the neuronal membrane ultrastructurally.[68,104,227]

The initial hypertension in *Atrax* toxicity may have several causes. Morgans and Carroll[166,167] demonstrated direct alpha-adrenergic stimulation with vasoconstriction of isolated arterial preparations exposed to *A. robustus* venom. In rabbit atria, an initial decrease followed by an increase in cardiac inotropy and chronotropy may result from vagal acetylcholine and myocardial norepinephrine releases, respectively.[47] The combination of myocardial responses and peripheral vasoconstriction may explain the initial hypertensive response.

Animal species vary in susceptibility to *Atrax* venom. Rabbits given 15 mg of crude venom intravenously and cane toads given 12 mg of female *Atrax* venom show no effects after envenomation. Primates, including humans, are among the most susceptible species. Newborn mice, also highly susceptible, have been used as an in vivo biologic assay for venom toxicity.[104,235] Sutherland[237] found that a lethal dose of venom from a male *A. robustus* could be neutralized in newborn mice by nonimmune sera from rabbits and other nonprimate vertebrates. Sheumack and coworkers[222] later demonstrated that the active fraction of nonimmune rabbit sera contained immunoglobulins G and M (IgG, IgM).

In addition, venom potency varies with time of year, recent feeding history, maturation, and gender of the individual spider.[12] Between 1956 and 1963, Wiener[277] demonstrated significant differences in the venoms of male and female *Atrax* spiders. Males had an average venom yield of 1.01 mg, less than the 1.84 mg average from females. On the other hand, guinea pig lethality was much greater after a bite by a male (75% to 90%) than by a female spider (20%). Weiner concluded that significant qualitative difference exists between the venoms of males and females.

Monkeys, which have a pattern of envenomation similar to that in humans, provide a model in which Sutherland[235,241] has described a biphasic clinical syndrome. Phase I begins minutes after venom injection, with local piloerection and muscle fasciculation. This extends proximally, becoming generalized over the next 10 to 20 minutes. After another 5 minutes, severe hypertension, tachycardia, hyperthermia, and coma with increased intracranial pressure may occur, followed by diaphoresis, salivation, lacrimation, diarrhea, sporadic apnea, borborygmi, and grotesque muscle writhing. Death may result from asphyxia caused by laryngeal spasm, combined with copious respiratory secretions, apnea, or pulmonary edema. Laboratory evaluation reveals metabolic acidosis and elevated plasma creatine phosphokinase. Phase II begins 1 to 2 hours after envenomation as the phase I symptoms subside. The victim may return to consciousness and appear to recover. In severe cases, hypotension gradually worsens over 1 to 2 hours, with periods of apnea. Pulmonary edema and death may occur despite ventilatory support.

Clinical Presentation. Up to 90% of *Atrax* bites may not result in significant envenomation.[275] Intense pain at the bite site may result from direct trauma as well as the venom's effect, but the bite does not provoke cutaneous necrosis. Wiener[277] studied the cutaneous effects of the venom on himself by injecting 0.5 mg intradermally. Local pain and a wheal surrounded by erythema lasted for 30 minutes, followed by localized sweating and piloerection. No systemic effects occurred.

The earliest systemic signs and symptoms may include perioral tingling, nausea and vomiting, diaphoresis, salivation, lacrimation, and dyspnea. Pulmonary edema follows, along with a generalized central and peripheral neurologic syndrome that includes muscle fasciculations, tremor, spasms, weakness, and impaired consciousness. Death may occur secondary to pulmonary failure, hypotension, or cardiac arrest.[232,275]

Thirteen fatalities from *A. robustus* envenomation were recorded between 1927 and 1984. Children are particularly susceptible; those younger than 12 years of age may die within 4 hours of the bite.[79,113,238] Before the development of specific antivenom in 1980, severe envenomations resulted in a minimum 8-hour critical period, followed by a 9- to 21-day hospital course.[78,238,241]

No fatalities caused by *H. formidabilis* have been recorded, although several severe envenomations have occurred.[104] Venoms of *A. robustus, A. versutus, A. infensus,* and *A. formidabilis* appear to have comparable vertebrate toxicity in vitro.[11]

Treatment. Immediate treatment after a bite is modeled after that for Australian snakebite and consists of four steps: (1) wrap the length of the bitten extremity with an elastic bandage, (2) splint to immobilize the extremity, (3) immobilize the victim, and (4) transport to the nearest hospital with the bandage in place.[180,239] A human case report has illustrated the utility of this method, with occurrence, disappearance, recurrence, and resolution of symptoms coinciding with compression wrap removal and replacement in a man bitten by a male *A. robustus*.[99] An experimental model in *Macaca fascicularis* monkeys has supported efficacy of the pressure immobilization technique.[234,235,242]

Specific antivenom has been the mainstay of treatment for *Atrax* envenomation since 1981. The antivenom is a purified IgG product developed by Sutherland and associates[236,243] at the Commonwealth Serum Laboratories by immunizing rabbits with a combination of male *Atrax* venom and Freund's adjuvant. The antivenom was demonstrated to neutralize *Atrax* venom in vitro and to reverse symptoms in monkeys before its introduction for human use. It has been used with good effect in humans bitten by *Atrax* and *Hadronyche* species.[65,241]

If a tourniquet or bandage is in place when the victim presents for hospital care, an intravenous line should be initiated before removing the tourniquet or bandage in an intensive care setting, with careful observation for development or progression of symptoms. If systemic signs or symptoms occur, victims are usually treated with antivenom administration. Two ampules of antivenom (100 mg of purified IgG per ampule) are administered intravenously every 15 minutes until symptoms improve. Dosing is the same for children as for adults, and total doses of 2 to 8 ampules have been reported. During a 10-year

period, antivenom was given to at least 40 persons, with no adverse effects or deaths reported.[241]

In addition to antivenom administration, management is symptomatic and supportive. Oxygen, mechanical ventilation, and IV fluid support may be indicated in severe cases. Atropine (0.6 mg) may be used to lessen salivation and bronchorrhea. Beta-adrenergic blockers may be indicated for severe hypertension and tachycardia.

Other than antivenom, no consistently effective agent has been found to enhance survival after *Atrax* envenomation. Diazepam, atropine, and furosemide have been found to increase survival in monkeys, but this may not be the case in humans.[77,117]

Sheumack and colleagues[223] developed a toxoid from robustoxin by polymerization with glutaraldehyde. Immunization with the toxoid conferred protection against the lethal effects of 50 mg/kg *Atrax* venom in monkeys for at least 26 weeks after toxoid injection.

Family Dipluridae: Funnel-Web Mygalomorphs

Members of the family Dipluridae are found on all continents and are concentrated in tropical areas. Individuals build funnel webs similar to those of Hexathelidae. Diplurids are distinct from hexathelids in having posterior lateral spinnerets that are very long and widely separated. Most are 5 to 25 mm in length.

Family Actinopodidae

Genus *Missulena*: Mouse Spiders

Biology. *Missulena* are large spiders (10 to 35 mm) native to Australia with one species found in Chile. Like all Actinopodidae, they are trapdoor spiders that burrow underground and seal the entrance with a lid that they open and close with their fangs. Silk lines spreading from the mouth of the burrow reveal the presence of prey that stumble across them. The spiders dash out, grab prey in their jaws and retreat back into the burrow to feed. These spiders spend most of the time underground, with the exception of males searching for females in autumn. The larger females are most often encountered when their well-camouflaged burrows are accidentally dug up.

Venom. *Missulena* venoms have recently been confirmed to have a major bioactive component, delta-missulenatoxin-Mb1a[108] that is homologous to the delta-atraxotoxins of atracine spiders (see earlier text). This toxin has strong insecticidal potency and acts by manipulation of tetrodotoxin-sensitive sodium channels. The discovery of this toxin and its homology with atraxotoxins indicates extreme conservation of this toxin family.[108] This finding also provides a mechanistic explanation for observed similarities between the toxic effects of bites of *M. bradleyi* and those of Atracines in animals.[192]

Clinical Presentation. In a series of 13 envenomations, pain was the most common complaint, severe in 10, with puncture marks, bleeding, redness, and edema being the next most common complaints. Three patients exhibited neurotoxic effects; all three exhibited paresthesias, one with accompanying diaphoresis. Nonspecific systemic effects occurred in five patients. There were no cases of severe envenomation in this series. There have been a total of 40 reported *Missulena* bites, with one case of severe envenomation. This occurred in a 19-month-old child in Queensland.[126,132]

Treatment. Treatment is largely symptomatic and supportive. In the case of severe envenomation of the 19-month-old child, administration of funnel-web spider antivenom, raised against male *Atrax robustus* venom, produced dramatic improvement in her condition, with full recovery following a second dosing.[192] Although severe envenomation is rare, it is suggested that all suspected Mygalomorphae bites be observed for 4 hours to exclude such envenomation.[126,132]

Dipluridae, Genus *Trechona*

Biology. *Trechona venosa* is a large South American funnel-web tarantula with neurotoxic venom potentially dangerous to humans.[39,90,109] As with all *Trechona* species, *T. venosa* is sedentary, living in holes or on plants in tropical forests along the Atlantic coast. The spider may be black or gray-brown with yellow stripes on the abdomen. Mature body length may be 3 to 4.5 cm, with 6- to 7-cm legs and 3- to 4-mm fangs. *T. venosa* is not found in Chile, but in this region it has been confused, particularly in venom studies, with a spider in the family Nemesiidae, *Acanthogonatus subcalpeianus,* which it resembles.[193]

Venom. The venom of *T. venosa* is extremely toxic to rats, with an apparent action similar to that of *Phoneutria* species.[151] Little is known about venom composition.

Clinical Presentation. No cases of human envenomation have been reported, although it is presumed that symptomatic envenomation by *T. venosa* is possible.

Treatment. Treatment is symptomatic and supportive.

SUBORDER ARANEOMORPHAE

Enormous diversity is found within the group Araneomorphae, which contains 36,400 of the 39,000 currently described spider species.[57,188,273] Araneomorphs, or true spiders, are found throughout the world in all terrestrial (and a few aquatic) habitats. They show tremendous variability in size, appearance, and habit; however, no araneomorphs are as large as the largest tarantulas. Characteristics that distinguish araneomorphs include features of the spinnerets that enable them to produce extremely sticky silk. Also, all but a few groups have only one pair of book lungs. Most have eight eyes, but eye number varies from two to eight. Prey capture tactics usually determine where a spider will be found and are generally consistent within particular groups. Thus, these tactics often provide conspicuous clues that help identify spiders.

Family Sicariidae: Recluse Spiders

Sicariidae includes two genera, *Loxosceles* and *Sicarius,* both of which are clinically important. The family falls within a larger group of families (Scytodoids) that all have only six eyes. In these two genera, the eyes are in dyad pairs. The chelicerae are fused at the base, and the labium is fused to the sternum. Males have more slender abdomens and more prominent pedipalps than do females. Previously, *Loxosceles* was placed in its own family, Loxoscelidae, but this was recently synonymized with Sicariidae.[187]

Genus *Loxosceles*: Brown or Fiddle Spiders

Biology. *Loxosceles*, commonly known as brown or fiddle spiders, build small, irregular, and sticky webs in small areas,

Figure 46-8. Adult female desert violin spider (*Loxosceles deserta*). (Courtesy Michael Cardwell & Associates, 1994.)

such as under rocks or wood or in human-made habitats. The genus contains more than 100 species, with centers of diversity in North or South America and Africa.[20,66,155,210,217] These spiders are 8 to 15 mm in adult body length, are light to dark brown, and have a dark, violin-shaped spot centered anterodorsally, such that the neck of the fiddle extends toward the posterior end of the cephalothorax. The shape and darkness of the fiddle, relative lengths of the first two pairs of legs, and genitalia characteristics are features that help distinguish species[41,120,217] (Fig. 46-8).

From the South American *L. laeta* to the South African *L. spinulosa,* these small arachnids have been associated with human pathologic conditions. Several species have been associated with necrotic arachnidism in the United States: *L. reclusa* (the true brown recluse spider, Fig. 46-9), *L. rufescens, L. arizonica,* and *L. laeta*. These spiders are native to all the southernmost states. In the Mississippi River Valley, their territory extends as far north as southern Wisconsin. Species native to one region or habitat may adapt successfully to new locations after transport by humans.[176,269]

Brown spiders regularly roam in search of new web sites, and males wander in search of females. They are most active at night from spring through fall, emerging from rock piles, woodpiles, and rats' nests to hunt insects and other spiders. South African savanna species have been observed under stones and logs and in the tunnels of old termite nests; spelean species are found naturally in caves, but have also appeared in homes and export warehouses.[173,175,178] Brown spiders may infest homes, generally preferring warm, undisturbed environments such as vacant buildings and storage sheds. In Chile, the tendency to inhabit human dwellings has earned *L. laeta* the names *araña de los rincones* (corner spider) and *araña de detrás de los cuadros* (spider behind the pictures).[218] *Loxosceles* distributions are patchy, but when they are present they can reach high densities. For example, 2055 individual *L. reclusa* were captured in a single-family home in Kansas in a 6-month period.[266] Molts, or shed exoskeletons, are good indicators of the presence of resident *Loxosceles*. Webs are small, flocculent structures made in crevices with a bluish tint in bright light. Egg sacs are laid flat against surfaces, typically with a layer of fluffy silk on the exposed surface. Females may live 1 to 3 years and longer in

A

B

Figure 46-9. **A,** Brown recluse spider (*Loxosceles reclusa*); **B,** *Loxosceles vonwredei,* a cave-dwelling species from Namibia. (**A** courtesy Indiana University Medical Center; **B** courtesy Greta Binford.)

captivity.[90,121] Naturally unaggressive toward humans, brown spiders are not prone to bite unless threatened or trapped against the skin. Bites typically follow retrieval of old bed sheets or jackets from storage. Thorough housecleaning with movement of boxes and stored items at least once per year highly reduces the likelihood of establishment of robust populations of *Loxosceles* in houses.[91]

A study by Vetter[266a] showed that of 1773 spiders thought to be *Loxosceles reclusa* by medical professionals, only 324 actually were *Loxosceles*. In addition, the true recluses were from regions historically known to be in their home range. Only one brown recluse spider was found outside the normal habitat region, and this case was explained by recent travel. Of the other 1449 submissions that were spiders, more than 38 families, 88 genera, and 158 species were identified. It was noted that more accurate identification occurred in areas with known historical populations. This study also suggests that the brown recluse has not migrated throughout the United States, but remains within its home regions.

Venom. Fractionated *Loxosceles* venom contains at least eight or nine major protein bands and three or four minor bands

identifiable by 1-D protein gel electrophoresis[34,198] and hundreds of proteins identifiable by 2-D gel electrophoresis (Binford GJ, personal observation). Hyaluronidase was first identified as a component by Wright and associates.[282] Hyaluronidase probably plays a facilitating role in lesion development, encouraging the spread of other venom components; however, it is not itself a cytotoxin. Hydrolytic enzyme activities include esterase,[282] alkaline phosphatase,[114] lipase,[136] and 5′-ribonucleotide phosphorylase,[89] but none of these alone appears to explain cytotoxicity.

Sphingomyelinase D, a 32- to 35-kDa venom protein family, is the sufficient causative agent for formation of dermonecrotic lesions following *Loxosceles* bites.[145,247,249] This enzyme activity is rare in nature and is currently only known elsewhere as an exotoxin of pathogenic bacteria. Sphingomyelinase D is present in *L. intermedia* spiderlings starting with the third instar, with increasing activity throughout development until adulthood.[95] Injection of expression products of cloned sphingomyelinase D–encoding genes isolated from *L. intermedia, L. laeta, L. reclusa,* and *L. boneti* produces characteristic lesions in rabbits.[145,249,251] Sphingomyelinase D activity has been detected from a sampling of worldwide representatives of *Loxosceles*.[34] Therefore, all species in this genus are suspected to be capable of bites resulting in dermonecrosis. Many of these species live in habitats that make them unlikely to encounter humans.

Sphingomyelinase D is postulated to operate by a variety of mechanisms, including cell membrane binding complement activation and polymorphonuclear leukocyte neutrophil (PMN) chemotaxis.[86,197,225,255,256] Lesions are inhibited in rabbits by pretreatment with nitrogen mustard to deplete PMNs. Histologic studies suggest similarities between venom-induced lesions and those seen with the Arthus and Shwartzmann phenomena.[225]

Some vertebrate species, such as rats and fish, are essentially unaffected by *Loxosceles* venom; others, such as rabbits, mice, and dogs, are highly susceptible to its effects.[216] Injected into humans, the venom is a hemolysin and cytotoxin, with enzymatic activities that may cause dermonecrosis and hemolysis. Pathogenesis of the human lesion is not well understood but depends on the functions of complement and PMNs.[17,217] Recent works suggest venom toxins act as proteases against constituents of plasma and basement membranes. Due to their degenerative nature, these proteases may be a plausible mechanism for the hemorrhage, delayed wound healing, renal failure, and associated spreading of other toxins seen in *Loxosceles* envenomations.[255]

Venom from *L. reclusa* has a direct hemolytic effect on human erythrocytes; this process depends on the presence of serum components that include C-reactive protein and calcium.[123,251] Platelet aggregation also is calcium dependent and is induced in vitro with sphingomyelinase D; this process may activate the prostaglandin cascade. Platelet aggregation appears to depend on serum amyloid protein, a serum glycoprotein of previously unknown significance.[85,200,215] Lysis of erythrocytes has been shown to be induced by *Loxosceles* sphingomyelinases dependent on activation of complement by the alternative pathway, caused by induction of surface glycophorin cleavage. Cleavage of glycophorins, observed after incubation of erythrocytes and *Loxosceles* toxins, was a result of activation of an endogenous membrane–bound metalloproteinase.[248,255]

Because *Loxosceles* venom provokes an immune response in experimental animals, efforts to develop diagnostic tests are based on antigen or antibody detection in human blood. In 1973, Berger and associates[28] reported an in vitro lymphocyte transformation assay for *L. reclusa* venom, which turned positive in the lymphocytes of exposed individuals within 4 to 6 weeks of initial exposure. This test may help to document prior exposure but not to diagnose envenomation at the time of the initial bite. Barrett and coworkers[23] reported a passive hemagglutination inhibition test using rabbit antibody and human erythrocytes incubated in vitro with venom from *L. reclusa*. Cardoso and associates,[45a] observing that efforts to detect antigen in human serum may fail because of insufficient antigenemia, have demonstrated the presence of *L. gaucho* venom in biopsy homogenate using enzyme-linked immunosorbent assay (ELISA). Barbaro and colleagues[21] demonstrated circulating IgG against *L. gaucho* venom detectable between 9 and 120 days after the bite in 4 of 20 patients.

Some of the variability in clinical presentation among victims of *Loxosceles* envenomation may be caused by differences in the venom of males and females. Female *L. intermedia* spiders produce a greater amount of more potent venom than do males.[62]

Clinical Presentation. Necrotic arachnidism, or loxoscelism in the case of bite by spiders of the genus *Loxosceles,* refers to the clinical syndrome that follows envenomation by a variety of spiders, for which *L. reclusa,* the brown recluse spider, is the prototype. The bites of these spiders often result in serious cutaneous injuries, with subsequent necrosis and tissue loss. Less often, severe systemic reactions may occur with hemolysis, coagulopathy, renal failure, and even death.[122,268]

Reports of severe reaction to spider bites possibly attributable to brown spiders date to 1872, in a report of a 45-year-old Texas woman with a febrile illness accompanying a large necrotic lesion of her thigh.[48] In 1896, death from renal failure accompanying another bite in Texas was reported.[189] Spider bite was reported as a cause of blackwater fever (massive hemoglobinuria) in Tennessee in 1940.[98] The first documented case of loxoscelism (from *L. rufescens*) was reported by Schmaus[219] in Kansas in 1929. *L. laeta* was identified as the cause of similar lesions in South America in 1937, and *L. reclusa* was the cause of necrotic arachnidism in the midwestern United States by 1958.[9,10] Since then, numerous cases of cutaneous and more severe reactions have been attributed to spiders of the genus *Loxosceles*.

The clinical spectrum of loxoscelism ranges from mild and transient skin irritation to severe local necrosis accompanied by dramatic hematologic and renal injury. Isolated cutaneous lesions are the most common presentation, and most bites may resolve spontaneously without the need for medical intervention.[27,28] Many authors distinguish between simple local presentation and more severe systemic, or viscerocutaneous, loxoscelism.

Local symptoms usually begin at the moment of the bite. A sharp stinging sensation is possible, although a victim may be unaware of having been bitten. Frequently, the bite site corresponds to a portal of entry or a region of constriction of clothing, such as cuff, collar, waistband, or groin area. The stinging usually subsides over 6 to 8 hours and is replaced by aching and pruritus as the lesion becomes ischemic from local vasospasm. The site then becomes edematous, with an erythematous halo surrounding an irregular violaceous center of "incipient necrosis" (actually hemorrhage and thrombosis).[42,211,251] A white ring of vasospasm and ischemia may be discernible between the

Figure 46-10. Brown recluse spider bite after 6 hours, with central hemorrhagic vesicle and gravitational spread of venom. (Courtesy Paul Auerbach, MD, and Riley Rees, MD.)

Figure 46-11. Brown recluse spider bite after 24 hours, with central ischemia and rapidly advancing cellulitis. (Courtesy Paul Auerbach, MD.)

Figure 46-12. Brown recluse spider bite after 48 hours, with incipient central necrosis.

central lesion and the halo. Often, the erythematous margin spreads irregularly in a gravitationally influenced pattern that leaves the original center eccentrically placed near the top of the lesion (Figs. 46-10 to 46-12). In more severe cases, serous or hemorrhagic bullae may arise at the center within 24 to 72 hours, and an eschar forms beneath. After 2 to 5 weeks, this eschar sloughs, leaving an ulcer of variable size and depth through skin and adipose tissue, but sparing muscle.[116] Lesions involving adipose tissue may be extensive, perhaps from lipolytic action of the venom.[136] The ulcer may persist for many months, leaving a deep scar.[164,207,271] Local sequelae depend on the anatomic location. Persistent segmental cutaneous anesthesia has been attributed to nerve injury after a recluse bite on the side of the neck.[107] Epiglottic and periepiglottic swelling severe enough to require endotracheal intubation has been reported in a recluse bite involving a child's ear.[115]

The bite of the somewhat larger South American spider *L. laeta* is reported to cause intense pain and extensive edema, with proportionately less necrosis than that caused by *L. reclusa*. The edema is notoriously prominent with facial bites and resolves over 2 to 4 weeks.[217]

Systemic involvement is less common but may occur in combination with cutaneous injury from any *Loxosceles* species; it occurs more frequently in children but may be seen in adults.[190,250] Systemic reaction may develop in cases with minor-appearing local findings, making diagnosis difficult.[251] When systemic involvement occurs, hemolytic anemia with hemoglobinuria is often the prominent feature, usually beginning within 24 hours of envenomation and resolving within 1 week.[69] During this time, measured hemoglobin may drop markedly, accompanied by jaundice and hemoglobinuria. The anemia is usually Coombs' test negative, but two cases of Coombs' positive anemia have been reported.[268] Fever, chills, maculopapular rash, weakness, leukocytosis, arthralgias, nausea, vomiting, thrombocytopenia, disseminated intravascular coagulation (DIC), hemoglobinuria, proteinuria, renal failure, and even death have been reported.[31,92,172,268]

The diagnosis of loxoscelism is based on spider observation and identification, typical history, and local and systemic signs. The differential diagnosis of the local injury includes bacterial and mycobacterial infection, herpes simplex, decubitus ulcer, burn, embolism, thrombosis, direct trauma, vasculitis, Lyme disease, and pyoderma gangrenosum.[5,139,191,194,240] There is no universally accepted or available laboratory test to aid in diagnosis of *Loxosceles* envenomation. However, in one case a venom-specific enzyme immunoassay was used to determine the cause of a suspicious skin lesion in a 57-year-old man. The assay was performed on both a lesional punch skin biopsy and a hair plucked from the lesion. In both samples the presence of *Loxosceles* venom was detected, indicating that use of a hair from the lesion negated the need for a punch skin biopsy.

A series of five proved or suspected *L. reclusa* bites to women in the second and third trimesters of pregnancy has been reported. Despite significant local injuries, rash, and microhematuria, no fetal injury was noted.[4]

Treatment. Treatment of loxoscelism depends on its severity. Cutaneous loxoscelism can usually be managed on an outpatient basis. Most mild cutaneous envenomations respond to application of local cold compresses,[141] elevation of the affected extremity, and loose immobilization of the part. Tetanus prophylaxis should be provided where indicated. Necrotic lesions may need debridement after erythema has subsided to define the margins of the central eschar. This usually involves significant debridement 1 or 2 weeks after the bite, with close follow-up for several weeks. In severe cases this can be followed with skin grafting or plastic surgery when the wound is stable. Severe necrotic or infected lesions may lead to hospitalization. Although many therapies have been used for *Loxosceles* envenomation, including dapsone, glucocorticoids, hyperbaric oxygen (HBO), electric shock, antivenom, metronidazole, diphenhydramine, phentolamine, and cyproheptadine, efficacy has not been established and clinical trials have not been done. Initial studies in small mammals have been inconclusive.[70,246]

Some have advocated the use of dapsone for prevention of lesion progression in potentially necrotic wounds seen within 48 to 72 hours of a bite.[82,87] Dapsone is a leukocyte inhibitor that in theory can minimize the local inflammatory component of cutaneous loxoscelism, thereby preventing or lessening subsequent skin necrosis. In 1983, King and Rees[140] reported the use of dapsone for envenomation in a human bitten by *L. reclusa,* based on a successful trial of dapsone pretreatment in guinea pigs injected with recluse venom. Since that time, no prospective, controlled human trial has proved dapsone efficacy, but a variety of case reports and series have supported its use in the treatment of potentially necrotic wounds treated in the first days after envenomation.[3,25,115,152,272] Typical dosage recommendations are for 50 to 100 mg orally, twice daily. Risks of dapsone therapy include hypersensitivity,[279] methemoglobinemia, and hemolysis in the presence of glucose-6-phosphate dehydrogenase (G6PD) deficiency. Due to these risks and the uncertain benefits, recent opinion has trended away from use of dapsone.[264]

Patients with systemic symptoms should be considered for admission when they have evidence of coagulopathy, hemolysis and hemoglobinuria, or rapid progression of other systemic signs. Care is mainly supportive, usually involving wound care, fluid management, presumptive treatment for bacterial superinfection, and occasionally, blood transfusion. Rarely, hemodialysis has been required for oliguric renal failure.[96] Discharge is appropriate when renal and hematologic status is stable.

For patients with significant local or systemic signs or symptoms, laboratory evaluation should include peripheral blood cell count, basic coagulation screening, and urinalysis. Liver and renal function tests are indicated in severe poisonings. When use of dapsone is considered, a screening test for G6PD deficiency may be indicated. The frequency of follow-up testing depends on the course and severity of envenomation. Hospitalized patients may need close follow-up of anemia and renal function over several days.

Corticosteroids have been injected either at the wound site or systemically, but this remains of questionable benefit.[26,196] Antihistamines may help control itching but do not change the lesion. Some advocate early surgical excision of the wound site,[8,16,75,118] but others have demonstrated that outcomes are better with early medical management in human patients,[63,198] as well as in experimental animals.[196] HBO treatment has been tried empirically in uncontrolled human trials, with reports of good outcome.[245] Comparison of HBO with no treatment in rabbits showed enhanced recovery at 24 days at the histologic level, but with no apparent clinical difference between the two groups.[229]

Loxosceles-specific antivenom has been tried in both the United States and South America. In the early 1980s, Rees and colleagues[196] reported a protective effect of treatment with antivenom in rabbits before or up to 12 hours after envenomation. Small-vessel occlusion, leukocyte infiltration, and necrosis occurred but were diminished in antivenom recipients. Pretreatment in a separate study abolished symptoms of systemic loxoscelism.[195] In one study, 17 patients were separated randomly into dapsone, antivenom, and combination therapy groups; all patients received erythromycin. Individual results suggested that the antivenom was efficacious when given early, but the overall trial was inconclusive, indicating the need for further study.[199] Gomez and colleagues[94] have studied the intradermal use of an anti-*Loxosceles* Fab-fragment in a rabbit model, suggesting therapeutic efficacy when the antivenom is injected up to 4 hours following envenomation. Currently, no *Loxosceles* antivenom is commercially available in the United States.

Figure 46-13. *Sicarius albospinosus* male from Namibia. (Courtesy of Melissa Callahan.)

In South America, an antivenom to *L. laeta* was developed in 1954 by Vellard using immune serum of the donkey. Furlanetto developed *L. laeta* antivenom using immune serum of horses. Reports of the efficacy of systemically administered *L. laeta* antivenom are mixed.[217] Recent work suggests an optimistic potential for development of a globally effective antivenom treatment of *Loxosceles* bites.[22] An ELISA assay, developed for the detection of circulating venom antigen, has the potential to develop into a diagnostic tool for clinicians and epidemiologists.[52]

Genus *Sicarius*: Six-Eyed Crab Spiders

Biology. About 25 *Sicarius* species are known. Their range is limited to dry regions of South America and South Africa. Individuals live under stones or are sand dwellers. Some hide under sand and emerge to capture passing insects. Their bodies are flattened and their legs are laterigrade, meaning the tips point anteriorly as in a crab. Adult body size ranges from 12 to 22 mm (Fig. 46-13).

Venom. The venom of the South African six-eyed sand spider *Sicarius testaceus* in a rabbit model demonstrated tissue necrosis and increased vascular permeability in the vicinity of the envenomation, as well as a dramatic decrease in platelet count. It is not clear whether this pattern is the same in humans.[254] South African *Sicarius* venoms have sphingomyelinase D activity as discussed for *Loxosceles* venoms.[34] The origin of this toxic enzyme in spider venoms is likely to have occurred in the most recent common ancestor of these two genera. South American *Sicarius* venoms have lost or reduced sphingomyelinase D activity (Binford, unpublished data, 2004). There are no records of clinically significant bites from South American *Sicarius* species.

Clinical Presentation. *Sicarius* species are occasionally implicated in human bites in South Africa. They tend to bite only when provoked and are rarely implicated in human poisonings, despite fairly high toxicity of the venom in laboratory animals.

Envenomation reportedly can cause edema, erythema, and necrosis, and is occasionally associated with DIC.[175–177]

Treatment. Treatment of *Sicarius* envenomation is symptomatic and supportive.

Family Desidae: Long-Jawed Intertidal Spiders

About 200 species of Desidae are distributed worldwide. Their chelicerae are often extended forward and have humps close to the prosomal attachment. Species range in body length from 4.5 to 12 mm. Most spiders in this family build small, irregular webs under rocks or logs. Some desids are closely associated with marine habitats and live in the intertidal zone. These spiders feed on marine animals and at high tide retreat into empty snail shells and worm tubes and seal off the entrance with silk. Others are found only on the water surface.

Genus *Badumna*: Black House Spider

Biology. *Badumna insignis* (*Ixeuticus robustus*), or the black house spider, is a common Australian spider associated with a few human bite reports. This species is a nocturnal forager that builds tangled webs with silk retreats in and around human habitations.[275]

Venom. Venom gland extract from *B. insignis* is reported to cause increased vascular permeability, as well as dose-dependent decreases in arterial pressure in rats, apparently from the presence of a serotonin-like substance in the venom.[143] Venom gland extract does not cause necrosis in cultured human or mouse skin.[14]

Venom of *B. insignis* has been found to contain fibrinogenolytic and gelatinolytic proteases. Sphingomyelinase activity has not been found in the venom of *B. insignis,* but has been identified in its abdominal contents.[283]

Clinical Presentation. In one report, *B. insignis* caused local pain, itching, and swelling followed by regional lymph node tenderness and discoloration of the area around the bite. Over 2 weeks, the lesion resolved, with some tissue necrosis and sloughing centrally.[153] In two series of *B. insignis* bites, local lesions were painful but transient and unaccompanied by necrosis.[130,276] Systemic symptoms have rarely been reported, including nausea, vomiting, abdominal pain, pruritus, and painful knees.[275]

Treatment. Treatment of *Badumna* envenomation is symptomatic and supportive.

Family Zodariidae: Hunting Spiders

Zodariids are a diverse group of hunting spiders.[131] The estimated 350 species have worldwide distribution, but are primarily found in the tropics and subtropics. They are medium sized (3 to 15 mm), with the anterior spinnerets usually longer than the posterior structures. They hide under stones or burrow in pebbles or sand. Very few reports of documented human envenomation exist, although *Supunna picta,* a common leaf litter–dwelling spider of Australia, reportedly caused a transient erythematous rash and slight itch in a woman bitten at home. The lesion resolved uneventfully.[37]

Family Gnaphosidae: Ground Spiders and Mouse Spiders

Gnaphosids are common and found worldwide, with the highest densities in temperate areas. They are small to medium sized (2 to 14 mm) and usually dark in color (black, gray, or reddish brown). They have long, slightly flattened abdomens, and two of their spinnerets are conspicuous and cylindrical. Gnaphosids are nocturnal hunters, mostly on the ground, and during the day retreat under rocks and stones or other tight quarters.

Herpyllus ecclesiasticus, the parson spider, is distributed widely throughout the United States under rocks and rubbish and in houses. One case report of a bite by *H. ecclesiasticus* described pruritus, arthralgia, malaise, and nausea beginning 1 hour after the bite. There was no necrosis.[154,281]

Treatment. Treatment of bites is symptomatic and supportive.

Family Lamponidae: White-Tailed Spiders

Until recently, the genus *Lampona* was placed in the family Gnaphosidae. The group was changed to family status because members do not share many of the characteristics that define Gnaphosidae.[186] Specialists believe that many undescribed genera in Australia belong to this group.

Genus *Lampona*

Biology. *Lampona cylindrata* is a hunting spider with a distinctive cylindrical shape and white spot at the tip of the abdomen. They are often found indoors in Australia and New Zealand.[275] Individuals are wandering foragers that enter webs of other spiders and prey on them.

Venom. The venom of *L. cylindrata* appears to increase vascular permeability in rats, perhaps from the release of endogenous bradykinin and prostaglandins.[143] Venom contains histamine and noradrenaline, both more highly concentrated in the venom of male spiders.[191]

Venom of *L. cylindrata* has also been found to contain fibrinogenolytic and gelatinolytic proteases. Sphingomyelinase activity has been identified in the abdominal contents, but not the venom, of *L. cylindrata*. This finding is similar to that for *Badumna insignis*.[283]

Clinical Presentation. In a series of 130 *Lampona* bites, predominantly of *L. cylindrata* and *L. murina,* pain or discomfort occurred in all and was severe in 27%. Puncture marks were present in 17%, and redness or red mark in 83% of bites. Systemic effects occurred in 9% of bites. Necrosis was not reported. There were three distinct clinical patterns: pain only (21%), pain and red mark for less than 24 hours (36%), and persistent painful or red lesion (44%).[128] Gray[102] reported a case of more significant illness, with nausea, lethargy, and a small zone of necrosis after a known white-tailed spider bite. Although the vast majority of cases are relatively benign, significant skin necrosis occasionally may result from envenomation.[275]

Treatment. Treatment of *Lampona* envenomation is symptomatic and supportive.

Family: Sparassidae Crab Spiders and Hunting Spiders

Biology

Sparassids, or "giant crab spiders," are distributed worldwide and are mostly tropical. They are large (10 to 40 mm) and resemble crabs, with the tips of all legs angling forward. They have flattened bodies and are capable of moving sideways. They have eight eyes in two straight rows. They are wandering hunters and typically nocturnal. In many places, they are welcome cohabitants with humans because they eat cockroaches.

Palystes natalius, the lizard-eating spider, is one of the largest true spiders in South Africa. It has a brownish gray body with bright yellow and black striped legs. The female is larger than the male, with body length up to 4 cm.

Venom. Venom from the Australian heteropodid *Isopeda montana* has direct beta-adrenoreceptor action, and *Delena cancerides,* the social huntsman spider, appears to have both alpha- and beta-adrenoreceptor activity, although neither of these has been involved in recognized human envenomation. Both have been shown to increase vascular permeability in rats.[117]

In a series of 173 spider bites, with 168 confirmed to be Sparassid spiders, 95% percent of the bites were from four genera: *Isopeda* (32%), *Isopedelia* (21%), *Neosparassus* (27%), and *Heteropoda* (14%); other genera included *Delena* and *Holconia*. The majority (95%) of bites were on limbs, with 82% occurring distally. Pain or discomfort was present in all cases, and severe in 27%. The median duration of pain was reported as 5 minutes, significantly less than with other spiders. Puncture marks (40%) or localized initial bleeding (35%) occurred in 54% of bites. Redness or a red mark occurred in 57%, swelling in 16%, and itching in 44% of bites. Systemic effects occurred in 4% and consisted of nausea and/or headache. There were no cases of necrotic ulcers or allergic reactions.[131]

Clinical Presentation. Only localized burning accompanied by slight swelling was noted after a female *P. natalius* bite to the left wrist.[174,179] An *Olios calligaster* bite was followed by mild local symptoms, transient nausea, and faintness. Six bites by *Isopeda* species, predominantly *I. pessleri,* resulted in minimal local symptoms only.[276]

One case of acute conjunctival inflammation, secondary to material from a squashed *Isopeda villosa* being accidentally rubbed in to the affected eye, has been reported. This type of injury is more commonly associated with small spiders, mainly Pholcidae (daddy longlegs).[129]

Treatment. Treatment of heteropodid bites is symptomatic and supportive.

In the case of the conjunctival inflammation, treatment consisted of a topical anesthetic followed by a normal saline flush for 1 hour. Redness and swelling began to resolve approximately 2 hours after injury. The patient experienced ongoing pain and mild photophobia and was discharged home with topical chloramphenicol ointment and an eye patch for 24 hours. Twenty-eight hours after the injury, symptoms had resolved.[129]

Figure 46-14. Green lynx spider (*Peucetia viridans*). (Courtesy Gita Bodner.)

Family Oxyopidae: Lynx Spiders

Lynx spiders are found worldwide, with the greatest abundance in the tropics. They are small to medium sized (4 to 18 mm) and have a distinctive eye pattern, with six eyes in a hexagon and two smaller eyes below. Their abdomens are pointed posteriorly. They have long leg spines (macrosetae) that help detect motion through vibrations in the air. They are diurnal hunters with good vision and actively search for prey. Most are found on vegetation, and some are arboreal.

Genus *Peucetia*

Biology. *Peucetia viridans,* the green lynx spider of the United States and Mexico, is a diurnal hunting spider. *P. viridans* is translucent green, with red eyes and joints[110] (Fig. 46-14). This species is common in the desert Southwest of the United States and adult individuals are large (approximately 20 mm) and conspicuous.

Venom. Whole venom of *P. viridans* causes total and reversible block of non-*N*-methyl-D-aspartate receptor-mediated transmission in the chick central nervous system.[133]

Clinical Presentation. A series of four cases in which the spider was identified as *Peucetia viridans* showed immediate pain, burning sensation, pruritus, erythema, and induration resolving in a couple of days. Necrosis and systemic signs have not been reported.[40]

Treatment. Treatment of *Peucetia* bites is symptomatic and supportive.

Family Salticidae: Jumping Spiders

Salticidae is the largest family of spiders, with more than 5000 currently described species and many more yet to be described. They are distributed worldwide, with the highest densities in the tropics. Some have called them the "butterflies of the spider world" because most are brightly colored and some have iridescent scales. Some species mimic ants, beetles, pseudoscorpions, and bird droppings. Jumping spiders have excellent eyesight, with large, posteromedian eyes. They visually search for prey, then stalk and ambush with catlike movements. Males

are often more brightly colored than females and perform elaborate courtship dances. They are always active during the day and are small, most less than 15 mm.

Bites from spiders of the genus *Phidippus,* such as the jumping spider *Phidippus audax* of the United States, can cause pain, erythema, pruritus, and sometimes minor ulceration. The swelling usually subsides within 2 days.[206] In Australia, local pain has been reported with the bite of spiders from genera *Mopsus, Breda, Opisthoncus,* and *Holoplatys*. One patient bitten by a *Holoplatys* reported headache and vomiting.[275]

Family Ctenidae: Wandering Spiders

Ctenidae are mostly found in subtropical and tropical areas. They can be large spiders ranging from 4 to 40 mm. They hunt on the ground or on vegetation. They resemble and are closely related to wolf spiders but are distinguished by their eye arrangement (three rows: two eyes, then four, then two, the last two being the largest). They sometimes travel as stowaways on bananas.

Family Ctenidae

Ctenid spiders are abundant and conspicuous worldwide in the tropics. They are large nocturnal wandering foragers.

Genus *Phoneutria*: Banana Spiders (Armed Spiders)

Biology. The *Phoneutria* spiders of South America are large, nocturnal creatures notorious for their aggressive behavior and painful bite. The best known representative of the genus is *Phoneutria nigriventer*. It is known in Brazil as *aranha armadeira,* meaning "spider that assumes an armed display," because of its characteristic defensive-aggressive display.[215] *P. nigriventer* is the largest, most aggressive true spider found in South America, with an average body length of 35 mm, leg length of 45 to 60 mm, and fangs 4 to 5 mm in length for females. Males are slightly smaller.[214] The body is gray to brown gray with white marks forming a longitudinal band on the dorsal abdomen. A distinguishing characteristic is the red-brown brush of hairs around the chelicerae. *P. nigriventer* is mainly found in southern Brazil, Argentina, and Uruguay. Other species have been found in Bolivia and Colombia. The spiders do not construct a web, because they are nocturnal hunters, often traveling several hundred meters in search of prey. They may enter houses during this time, hiding in clothes in the light of day. According to Bucherl,[38] 600 to 800 spider bites occur each year around the city of São Paulo alone.

Venom. *Phoneutria* venom is a complex mixture of histamine, serotonin, glutamic acid, aspartic acid, lysine, hyaluronidase, and other polypeptides. Histamine, serotonin, and incompletely characterized kallikrein-kinin activating fractions contribute to local tissue swelling from the increased vascular permeability that may occur with envenomation.[6,156] In addition, the venom contains at least six neurotoxic polypeptides, with molecular weights between 3500 and 8500.[61]

The neurotoxic components include sodium channel poisons that appear to potentiate action potentials along axons, provoking erratic or rapid uncontrolled muscle twitches in invertebrates[71] as well as vertebrates.[7,150] Microscopically, there is acute transient swelling of axons, particularly at the nodes of Ranvier, in a pattern similar to that caused by the venoms of scorpions *Centruroides sculpturatus* and *Leiurus quinquestriatus*. The axons recover within a few hours of exposure, but return of nodal width to normal takes several days.[150]

The effects of the venom have been studied in mice, rats, guinea pigs, rabbits, pigeons, and dogs. The venom has little or no effect on frogs and snakes, and it has four times greater toxicity in dogs than in mice. Rats and rabbits are very resistant to the venom's effects, but rabbit vascular smooth muscle contractions appear to be stimulated by a venom protein that acts independently of voltage-dependent sodium and calcium channels.[157] Dogs developed intense pain, manifested by yelping, followed by sneezing, lacrimation, mydriasis, hypersalivation, erection, ejaculation, and death after venom was injected subcutaneously.[215] This is well within the dose that a single spider may inject.

Clinical Presentation. *P. nigriventer* venom acts on both the peripheral and central nervous systems.[109] Although the majority of cases are clinically insignificant,[151] humans bitten by *P. nigriventer* may develop severe local pain that radiates up the extremity into the trunk, followed within 10 to 20 minutes by tachycardia, hypertension, hypothermia, profuse diaphoresis, salivation, vertigo, visual disturbances, nausea and vomiting, priapism, and occasionally death in 2 to 6 hours. Respiratory paralysis is generally the cause of death. Severe envenomation is more common among young children. Fatalities may occur in the debilitated or the young, but most people recover in 24 to 48 hours. Workers who handle bananas are frequently bitten because the spider hides in bunches of bananas. Bites have been reported in Switzerland and Argentina in produce workers inadvertently encountering these traveling spiders.[109,214,215]

Treatment. In most cases, symptomatic care is all that is necessary. Local pain control may be achieved by infiltration of local anesthetic near the bite site; this reportedly suffices for 95% of cases treated at the Hospital Vital Brasil.[151]

For more severe cases, a polyvalent antivenom (Sero Antiaracidico Polivalente, Instituto Butantan) and a monovalent antivenom (Belo Horizonte) active against *Phoneutria* species have been available in Brazil. Skin testing and antihistamine prophylaxis are recommended before their use. One to five ampules of antivenom are injected intramuscularly and/or intravenously, and clinical response is judged by the relief of pain or resolution of priapism. Opiates may potentiate the venom's effects on respiration and are generally not recommended in cases of systemic envenomation.[109,214,215]

An ELISA has been developed, using anti-*P. nigriventer* antibodies, for rapid detection of venom antigens in suspected *P. nigriventer* envenomation. It has been tested in humans and shows promise as a diagnostic agent.[53]

Family Lycosidae: Wolf Spiders

Lycosidae are among the most common spiders. They are diverse, with more than 3000 species distributed worldwide. They are found from beaches to grassy fields and pastures. The Greek name lycosa (wolf) comes from the former belief that they hunted in packs.[59,281] They range in size from 3 to 25 mm. Most species wander in active pursuit of prey, generally during the day. A few make deep burrows, and some even cover their burrows with doors. Most live on the ground, but some climb in vegetation. They have good vision, with conspicuously large, posteromedian eyes. Their eyes are arranged in three rows (four eyes, then two, then two). To attract mates, males wave their

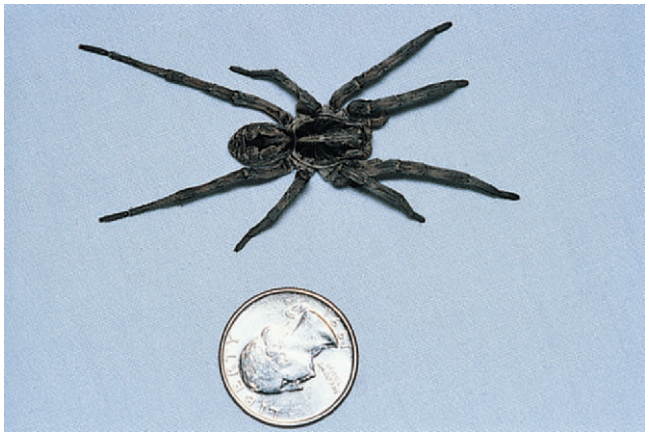

Figure 46-15. Wolf spider (*Lycosa* species). (Courtesy Arizona Poison & Drug Information Center, 1996.)

legs and sometimes stridulate to make sound. The female carries the egg sac attached to her spinnerets. When the young hatch, they climb on their mother's abdomen for transport. They have three claws on their tarsi.

Genus *Lycosa*: Wolf Spiders

Biology. *Lycosa* is a large and widespread genus of wolf spiders (Fig. 46-15). It includes various middle-sized to large spiders with mildly cytotoxic venom capable of provoking transient inflammation in humans. The most famous wolf spider species is *Lycosa tarentula*, to which "tarantula" was first applied. Its bite was once believed to cause "tarantism," a syndrome of stupor, the desire to dance, and sometimes death, but this historic syndrome is now attributed to *Latrodectus tridecimguttatus* (neither a wolf spider nor a tarantula), and the wolf spider bite is now known to cause little more than stinging pain. The South American *Lycosa raptoria* has been reputed to be more dangerous than other wolf spiders, provoking necrosis at the site of envenomation. It now appears that this was also based on a misunderstanding. Necrotic arachnidism in South America is now attributed mainly to *Loxosceles* species.

Venom. Lycosid venom is thought to be primarily cytotoxic, without hemolytic or anticoagulant activity.[281] Although scientific reports of necrosis are lacking after envenomation by the Australian wolf spider *L. godeffroyi*, media reports suggest that bites may lead to necrosis. Atkinson and Wright[13] have demonstrated that the raw venom of *L. godeffroyi* causes a strong inflammatory response and cutaneous necrosis when injected into mice. They further hypothesized that this action may result from contamination of the venom with digestive juices, in that electrically collected raw venom caused necrosis, whereas venom gland extract did not.[14]

Clinical Presentation. A series of 515 cases of confirmed *Lycosa* bites in Brazil showed that most occur between the hours of 6 AM and 6 PM, at a fairly consistent rate year-round. The most common bite sites were feet (40%) and hands (39%). The most common signs and symptoms were all local, with pain in 83%, swelling in 19%, and erythema in 14%. No local necrosis was described.[201]

Figure 46-16. *Cheiracanthium inclusum*. (Courtesy Sherman Minton, MD.)

In a series of 45 Lycosidae bites in Australia, 73% were distal (hand or foot). Pain occurred in all bites and was reported as severe in all cases. Puncture marks or localized initial bleeding occurred in 33%, swelling in 20%, redness or a red mark in 67%, and itching in 13%. Three patients reported systemic effects, two with nausea, one with headache, and one with malaise. Necrosis or ulceration was absent in all cases.[125]

In the United States, five cases of Lycosidae bites have been documented. One resulted in skin necrosis at the bite site, probably from the combined results of envenomation and infection.[45]

Treatment. Although South American antivenom active against *Lycosa* venom was available in the past, it was used in only one case of 515 reviewed by Ribeiro and associates.[201] Since 1985, the polyvalent Butantan Institute spider antivenom has not included the antilycosid fraction.[151] Most *Lycosa* cases can be managed with tetanus immunization and ice or oral analgesics; occasionally, local anesthetic block has been used for pain management.[201]

Family Clubionidae: Sac Spiders and Two-Clawed Hunting Spiders

Clubionids are common and distributed worldwide with the highest diversity of species in the neotropics. They are small to medium sized (2 to 15 mm) and are usually light brown to yellowish in color. They hunt nocturnally and make resting tubes in rolled leaves or under rocks or stones, where they retreat during the day.

Genus *Cheiracanthium*: Running Spiders and Sac Spiders

Biology. The genus *Cheiracanthium* as a whole has no distinctive marks or patterns.[98,281] Members may be pale yellow, brown, green, or olive. The dorsal abdomen may have a median longitudinal stripe. Body size ranges from 7 to 15 mm, with a total diameter of 3 cm, including long, slender legs.

In the United States, *C. inclusum* is the only indigenous species (Fig. 46-16) although several species have been introduced. *C. diversum* is widely found in the Pacific islands. It was transported into Hawaii from Australia approximately 50 years ago. *C. mildei* was introduced from Europe and is now found from New England to Alabama to Utah. It is a common biting

spider in Boston; it is most abundant in autumn, when most bites occur.[228]

The South African sac spider *C. lawrencei* is a common nocturnal house spider that forages at night and may become trapped in bedding.[175] During the day these spiders hide in the concavities of leaves, curtains, or windowsills, encased in a silk sac. They are fast moving and aggressive when threatened.[177]

Venom. Research on *Cheiracanthium* venom is limited. About 75% of guinea pigs bitten by *C. mildei* for the first time developed a wheal within 5 minutes, with 60% developing an eschar within 1 day.[228] Fractionation of dissected venom gland extracts from *C. japonicum* resulted in five fractions with lethal activity in mice. These were considered neurotoxic based on symptoms of dyspnea, flaccid paralysis, and death after intraperitoneal injection.[182]

Clinical Presentation. Spiders belonging to the genus *Cheiracanthium* have a documented history of human envenomation since the eighteenth century. Species are known for their tenacious, painful bite. A pruritic, erythematous wheal appears within 30 minutes. Nausea, abdominal cramps, headache, and local necrosis have been reported.

In 1901, Kobert described local swelling, erythema, pain, and fever after the author's third *C. punctorium* envenomation.[29] Maretic[159,160] described local redness, pain, and edema but no necrosis after *C. punctorium* envenomation. In another case, *C. inclusum* caused local pain that radiated from the forearm bite site up the arm, associated with nausea. No other signs developed.[29]

The Australian species *C. mordax* and *C. longimanus* caused local swelling, erythema, and pain associated with malaise, headache, dizziness, and nausea. Symptoms receded within 36 hours after treatment with antihistamines and local anesthetics.[29] Ori[182] found similar signs and symptoms after envenomation by *C. japonicum*.

In South Africa, most *C. lawrencei* bites occur at night during sleep.[174] Paired bite marks 6 mm apart are evident within the first few hours. Local edema and erythema may be slight. By the third day the marks may become necrotic, with more edema, erythema, and pain; headache and fever may accompany this stage. The small ulcer begins to heal 7 to 10 days after the bite.[175]

Treatment. The lesion usually heals without problems, provided secondary infections are avoided. Treatment is supportive, consisting of cool compresses, elevation, immobilization, analgesics, and tetanus prophylaxis.

Family Corinnidae: Sac Spiders

Corinnids are ground-dwelling spiders primarily found in tropical regions. They were previously placed in the family Clubionidae and have similar habits. Spiders of the *Trachelas* genus are often encountered in houses in late summer and fall. *T. volutus* and several other *Trachelas* species reportedly cause mild local reactions without necrosis. Bites are painful initially and may swell. No systemic effects have been reported.[184,252] Treatment is symptomatic and supportive.

Family Agelenidae: Hobo, Grass, and Funnel-Web Spiders

The 600 species of Agelenidae are mainly found in temperate regions of the Northern Hemisphere. They are medium-sized spiders, ranging from 8 to 15 mm in body length. Individuals build sheet webs that lead to a long funnel, which the spider uses as a retreat. When prey contacts the web, the spider runs out, bites it, and carries it back into the funnel. The large and conspicuous webs of these spiders often are long lasting, with spiders adding silk to make the sheet larger as the individual grows. Males may be found searching for females.

Genus *Tegenaria*: Hobo Spiders

Biology. *Tegenaria agrestis* is commonly called the hobo spider or Northwestern brown spider. It is a 10- to 15-mm, light-brown spider with a yellowish green tint and chevrons on the dorsal abdomen. Individuals typically build funnel webs in undisturbed habitats such as abandoned woodlots or along railroad tracks, with a hidden retreat beneath wood, rocks, or debris and small amounts of silk extending beyond the cover. Spiders mature to adulthood in midsummer and mate and lay eggs in July through September. Adult males live for 1 year, then senesce and die at the end of the mating season. Adult females live for 2 years, so adults are present throughout the year.

T. agrestis spiders are common and widespread natives of Europe and western central Asia.[111] This species was probably introduced to North America in a seaport near Seattle in the early 1900s and was first formally identified in the 1930s.[73,74] It has since expanded its range to British Columbia, Alaska, Oregon, Idaho, Montana, and Utah.[18,202,272] By the 1960s, individuals were often collected in and around human habitations in the Pacific Northwest.

Venom. The venom of *T. agrestis* has become of interest in the last 15 years because of reports suggesting that their bites result in necrotic lesions. *T. agrestis* venom chemistry is not well characterized, and no necrotoxic component has been identified. Johnson and colleagues[135] identified potent insect-specific neurotoxic peptides and mammalian-specific peptides (5000 and 9000 daltons) that were lethal to mice at high dosage.[265]

Experimental envenomation of rabbits by live male spiders has resulted in extensive cutaneous injury and clear evidence of systemic poisoning. Local erythema appeared and faded within the first day; discolored patches were visible by day 4 and sloughed by day 6. Autopsy revealed petechial hemorrhages on the surfaces of the lungs, liver, and kidneys.[258]

Since the early 1980s, medically significant bites in the Pacific Northwest have been attributed to *T. agrestis*.[2,259,261,262] In Europe, no medical problems have been associated with bites from these spiders.[30,39,56] Venom chemistry of American spiders is not different from venoms of English spiders, so the cause of the alleged difference in medical significance is currently under investigation.[33] Bites of males from the Pacific Northwest have more severe necrotic effects on mammalian tissues than bites from females.[258] Necrotic lesions have been attributed to this species all during the year, with a trend toward increased severity in the winter months.[262]

Clinical Presentation. *T. agrestis* has been implicated in several cases of necrotic arachnidism similar to that seen in *Loxosceles* envenomation. Systemic effects reported include headache, visual disturbances, hallucinations, weakness, and lethargy.

Figure 46-17. *Agelenopsis aperta*. (Courtesy Eileen Hebets.)

Hemorrhagic complications have been reported in experimental animals.

In general, direct observations are scarce of *T. agrestis* biting people, who then develop necrotic lesions.[2] According to Vest,[258,262] who studied 22 cases of "highly probable" *T. agrestis* envenomation, the local lesion followed a pattern reminiscent of loxoscelism. The initial lesion appeared as a small reddened induration, often surrounded by a large zone of erythema. Vesicles occurred within 36 hours, then burst; marked necrosis developed in 50% of cases. The most common symptoms included headache, weakness, and lethargy.[260]

If a spider has not been identified in association with a lesion, attribution of necrosis to *T. agrestis* bites is discouraged.[265]

Treatment. No studies have investigated treatment for envenomation by *Tegenaria* species. As with mild cases of loxoscelism, patients should be treated supportively, with tetanus prophylaxis, careful wound debridement as needed, and observation.[265]

Genus *Agelenopsis*: Grass Spiders and Funnel-Web Spiders

Biology. *Agelenopsis aperta* is common in and restricted to the deserts of the southwestern United States, ranging from California to East Texas (Fig. 46-17). They build large, conspicuous sheet webs, with retreats under rocks and logs or in tufts of grass. Adults are 13 mm in body length.

Venom. The venom of this species is among the best known of all spiders in terms of biochemical composition and neurophysiologic activity of the individual components. Its clinical relevance in humans, however, has only recently been recognized.

Nineteen toxins have been characterized in *A. aperta* venom, with three distinct classes that act synergistically to subdue insect prey rapidly and irreversibly.[181] The μ-agatoxins modify sodium channel kinetics, increasing neurotransmitter release generally; the ω-agatoxins block presynaptic, voltage-sensitive calcium channels; and the α-agatoxins are a family of low-molecular-weight acylpolyamines that block glutamate-sensitive receptor channels in insect muscle. The coexistence of toxins with different mechanisms of neurotoxicity appears to confer a synergistic action to the overall venom effect in insect prey. The ω-agatoxins range in target specificity from invertebrates to mammals.[257]

Clinical Presentation. Two cases of envenomation by *A. aperta* have been reported in southern California. A 9-year-old boy developed a tender but nonnecrotic bite site, followed by a 2-day systemic syndrome that included headache, nausea, disorientation, pallor, and unsteady gait. A 54-year-old man developed a painful, indurated lesion that persisted for a week; he had no systemic symptoms.[263]

Treatment. Treatment of *Agelenopsis* envenomation is symptomatic and supportive.

Family Theridiidae: Comb-Footed Spiders

The family Theridiidae, sometimes called cobweb or comb-footed spiders, is speciose, diverse, and distributed worldwide. The spiders are small to medium sized (1 to 14 mm, usually less than 8 mm) and often have globose abdomens. They make irregular, tangly webs in which the spider hangs upside down. The silk is very sticky, easily entangling prey. Spiders throw silk over ensnared prey using a tiny comb at the end of the fourth leg. They then envenom prey and suck them dry through a small hole in the exoskeleton, because they have no cheliceral teeth for chewing.

Genus *Latrodectus*: Widow Spiders

Biology. *Latrodectus* (Latin for "robber-biter") species are among the largest theridiids. Females are 12 to 16 mm in body length. Males are much smaller, with longer legs relative to their body size. Individuals build typical theridiid cobwebs with very strong strands of silk. Arthropods are the most common prey, but widows also kill and consume vertebrates (e.g., small lizards and snakes). The folkloric belief that widow females kill and consume their mates does happen, although not as a rule.

The 8- to 10-mm female black widow is shiny black with a characteristic red hourglass marking on the ventral abdomen. Species are distinguished based on hourglass shape and dorsal color patterns. Males are lighter in color, with white and gray markings and a faint hourglass. This feature becomes more prominent with maturity. The female spins an irregular web in sheltered corners of fields, gardens, and vineyards and under stones, logs, and vegetation. Uncommon in occupied dwellings, the spiders may be plentiful in barns, garages, trash heaps, and outbuildings. A few *Latrodectus* species (e.g., *L. variolus*) are arboreal. The web's tattered "cobweb" appearance may belie an ongoing state of occupation, particularly during the daytime, when the spider is out of sight. The female seldom ventures far from the web, in which she suspends an ovoid or tear-shaped, whitish egg case.

Latrodectus spiders are worldwide in distribution, most plentiful in temperate and subtropical regions, and most abundant during summer.[30] *Latrodectus mactans mactans,* the black widow, is cosmopolitan and occurs in every state except Alaska (Fig. 46-18). In North America, species include *L. geometricus* (the brown widow, Fig. 46-19), *L. bishopi* (the red-legged widow), *L. variolus,* and *L. hesperus* (Fig. 46-20). Species known to envenom humans are endemic to Australia (*L. m. hasselti,* Fig. 46-21) and to Europe and South America *(L. tredecimguttatus)*. Related species are found in Asia and the Middle East *(L. pallidus)* and in Africa *(L. indistinctus)*.

Figure 46-18. Adult female *Latrodectus mactans mactans* with fresh egg case. (Courtesy Michael Cardwell & Associates, 1999.)

Figure 46-19. Mature female brown widow spider (*Latrodectus geometricus*). (Courtesy Michael Cardwell & Associates, 1999.)

Figure 46-20. Mature female western black widow (*Latrodectus hesperus*). (Courtesy Michael Cardwell & Associates, 1993.)

Figure 46-21. Red-backed spider (*Latrodectus mactans hasselti*). (Courtesy Sherman Minton, MD.)

The species *L. tredecimguttatus* is most important in the Mediterranean region, the Middle East, and parts of Russia, where it is sometimes referred to as *L. lugubris*. It may have red or orange spots or may be pure black and is known as *kara kurt* (Russian for "black wolf"). *L. indistinctus,* the black button spider of South Africa, has a narrow or broken red dorsal band or may be black. The red-backed spider *(L. m. hasselti)* is medically important in Australia, New Zealand, and southern Asia. It has a dorsal red band similar to that of *L. indistinctus,* and the female also has a ventral red hourglass reminiscent of *L. m. mactans*. *L. geometricus* is brown with black, red, and yellow markings and is common in southern Africa and warmer parts of the Americas.

Widow spiders tend to bite defensively when accidentally crushed. In the Mediterranean basin, southern Russia, and South Africa, bites are associated with grain harvesting and threshing and with grape picking. In the United States, most bites occur in rural and suburban areas of southern and western states, with no special age, gender, or occupational predilection. In regions where outdoor privies are in common use, human envenomations are likely to involve the buttocks or genital area.[32,93,159,280] Outbreaks of latrodectism may occur locally in epidemic fashion, lasting several years, and depend on changes in spider predator and parasite balance and on occupational variations in human-spider contact. Apparent outbreaks may also result from sudden increases in publicity and reporting.[29,137]

Venom. Unlike many other arthropod venoms, that of the widow spiders appears to lack locally active toxins capable of provoking inflammation. The venom contains several toxic components, including a potent mammalian neurotoxin, α-latrotoxin, which induces neurotransmitter release from nerve terminals.

In 1964, Frontali and Grasso[80] demonstrated three electrophoretically and toxicologically distinct fractions of *Latrodectus* venom. In 1976, Frontali and associates[81] further purified and defined these fractions, encountering one major constituent (the B5 fraction, later renamed α-latrotoxin) with significant toxicity in mice and frogs (other fractions have effects more specific to insect physiology). α-Latrotoxin, a protein mix with an average molecular weight of 130,000, caused profound depletion of presynaptic vesicles with swelling of the presynaptic terminal at frog neuromuscular junctions;

complete blockade of neuromuscular transmission followed within 1 hour. The toxin binds irreversibly with the lipid bilayer of the cell membrane, producing cation-selective channels and interfering with endocytosis of vesicle membranes.[49,76] The mechanism of action is not fully understood, but multivalent cations, including calcium, may enter the presynaptic nerve terminal through these channels, interfering with calcium-dependent intracellular processes.[100,165] These effects appear to be specific to presynaptic nerves but independent of the transmitter involved. Acetylcholine, noradrenaline, dopamine, glutamate, and enkephalin systems are all susceptible to the toxin.[204]

Grishin[106] has described the venom of *L. tredecimguttatus*, which has a family of seven protein toxins of high molecular weight. These all cause massive neurotransmitter release from presynaptic endings. They differ from each other in the specificity of the target animal. α-Latrotoxin acts selectively on vertebrate nerve endings; five latroinsectotoxins act on insects, and one toxin is specific for crustaceans. These large molecules have several functional domains responsible for ionophoric and secretogenic actions. Amino acid sequence analysis of precursor toxins reveals high levels of sequence identity between the different latrotoxins. It also reveals a series of ankyrin-like repeats that might be the structural basis of the interactions between the toxins and the membranes.[106] *L. indistinctus* and *L. geometricus* also contain α-latrotoxin, the former with a greater venom yield per spider.[169]

Clinical Presentation. Latrodectism, the syndrome often resulting from *Latrodectus* envenomation, is best known for widespread, sustained muscle spasm rather than for local tissue injury. Although long-term outcomes are usually excellent, victims may have significant hypertension, autonomic and central nervous system dysfunction, and abdominal pain severe enough to be mistaken for an acute abdomen.

The initial bite may be sharply painful, but many bites are not recognized initially, so diagnosis is often presumptive and based on local and systemic signs. Local reaction is typically trivial, with only a tiny papule or punctum visible on examination. The surrounding skin may be slightly erythematous and indurated. In most cases, symptoms do not progress beyond this point. In one Australian series, 76% of victims presenting to a hospital for care had local symptoms only.[134]

In some cases, however, neuromuscular symptoms can become dramatic within 30 to 60 minutes as involuntary spasm and rigidity affect the large muscle groups of the abdomen, limbs, and lower back. Rhabdomyolysis has rarely been reported.[84] A predominantly abdominal presentation may closely mimic an acute abdomen. Associated signs include fasciculations, weakness, ptosis, priapism, thready pulse, fever, salivation, diaphoresis, vomiting, and bronchorrhea. Compartment syndrome has rarely been described after a bite by *Latrodectus* species.[58] Pulmonary edema has been described in Europe[162] and South Africa.[146,267] Respiratory muscle weakness combined with pain may lead to respiratory arrest. Hypertension with or without seizures may complicate management in elderly or previously hypertensive individuals. Isolated (normotensive) seizures do not appear to be a feature of latrodectism. Intractable crying may be the predominant feature in infants.[43] Pregnancy may be complicated by uterine contractions and premature delivery.[20,120,161,205,209] A characteristic pattern of facial swelling, known as *Latrodectus* facies, may occur hours after the bite and is sometimes mistaken for an allergy to drugs used in treatment. The usual course of an envenomation is to achieve complete recovery after a few days, although pain may last a week or more.

The clinical picture of *Latrodectus* poisoning is similar around the world. In the 1980s in California, the most common site of envenomation referred to a toxicology service was the lower extremity (48%), followed by upper extremity (28%), trunk (18%), and head or neck (5%). The most common systemic symptom was abdominal or back pain (58%), followed by extremity pain (38%), hypertension (29%), and diaphoresis (22%).[54] In Australia, a 1961 survey showed 37% of victims were bitten on the upper extremities, 27% on the lower extremity, 22% on the buttock or penis, 17% on the trunk, and 4% on the head or neck.[278] A 1978 report showed a decline in the incidence of genital and buttock involvement (9.7%), perhaps related to a decrease in the use of outdoor lavatories.[244] Australian envenomations showed a similar pattern of pain and diaphoresis, with more prominent local inflammation and lymphadenopathy and less hypertension than reported in the United States. In South Africa, envenomation by *L. geometricus* results primarily in local pain, whereas *L. indistinctus* provokes a syndrome of generalized pain, diaphoresis, and muscle rigidity similar to that seen in the United States.[168] Victims bitten by *L. tredecimguttatus* may have spasm of facial muscles, swollen eyelids, lacrimation, and photophobia, more often resulting in recognized *Latrodectus* facies. A rash may appear 2 to 11 days after envenomation.[158]

Treatment. Although the worst pain usually occurs during the first 8 to 12 hours after a bite, symptoms may remain severe for several days. All symptomatic children, pregnant women, and patients with a history of hypertension should be admitted to the hospital. Discharge is usually possible within 1 to 3 days, when hypertension and muscle spasm have subsided. A patient with a satisfactory response to antivenom may be sent home after several hours' observation.

Care of the local site includes routine cleansing, intermittent application of ice, and tetanus prophylaxis. Severe pain and muscle spasm usually respond to intravenous narcotics or benzodiazepines. Careful observation of respiratory status is vital when either or both of these are used. Calcium gluconate infusion, advocated in the past, has proved only minimally useful and is no longer recommended. Hypertension may be treated with an infusion of sodium nitroprusside or nifedipine if the patient does not respond to pain control with narcotics or antivenom.

Antivenom active against *Latrodectus* venom is available in the United States from Merck and Co.; in Australia from Commonwealth Serum Laboratories; and in South Africa from the South African Institute of Medical Research. Standards for *Latrodectus* antivenom use vary around the world, as do guidelines for its administration.[54,168,241]

In the United States, *Latrodectus* antivenom, although it has never undergone clinical trials, has been used for many years and it is believed by many clinicians to be safe and effective for reversal of systemic effects and associated pain. When used properly, these products may prevent systemic sequelae resulting from envenomation, and even the need for hospitalization in some severe cases.[55] Priapism, although refractory to treatment with opiates and benzodiazipines, has been reported to improve with antivenom.[119]

In general, antivenom should be used in cases involving respiratory arrest, seizures, uncontrolled hypertension, or pregnancy.[210] In less severe settings, its value must be weighed against the risks of acute hypersensitivity and delayed serum sickness. In Australia, 0.5% to 1% of cases result in anaphylaxis, and some patients develop serum sickness.[244] Death from anaphylaxis has been reported in the United States.[54] The production of a Fab fragment specific for *Latrodectus* venom or α-latrotoxin could eliminate much of the concern surrounding the use of whole IgG in these patients.[55] Fab fragments have reduced antigenicity, due to their lack of an Fc component present in whole IgG, prompting less of an immune response to the foreign protein by the recipient. An $F(ab)_2$ product produced in Mexico is currently undergoing clinical evaluation in the United States.

The usual therapeutic antivenom dose is one to three vials or ampules intravenously. Efficacy of antivenom is reported as satisfactory in 94% of patients in Australia,[274] with anecdotal reports of efficacy even weeks to months after envenomation.[19]

Laboratory evaluation may include complete blood cell count, electrolytes, blood glucose, and urinalysis. Common findings include leukocytosis and albuminuria. In victims with severe muscle spasm, creatine phosphokinase levels may be elevated. Abdominal films and stool examination for occult blood, both of which should be normal after widow spider envenomation, may help with the differential diagnosis of abdominal pain. A pregnancy test should be done if indicated. No specific antigen or antibody detection technique is currently available for clinical diagnosis.

Genus *Steatoda*: False Widow Spiders

Biology. *Steatoda* species are found worldwide. They are 3 to 8 mm in body length, smaller than *Lactrodectus,* and typically dark brown, often with white markings on the abdomen. They build tangly cobwebs and sit in crevices under stones, in tree bark, or in cracks of buildings near the web. *Steatoda paykulliana* of Europe, *S. foravae* of southern Africa, and *S. grossa* of the United States bear an external resemblance to members of the *Latrodectus* genus and therefore are referred to as false widow or button spiders.

Venom. The crude venom of *S. foravae* contains a major polypeptide of the same molecular weight as α-latrotoxin and can elicit a comparable neurotransmitter release syndrome in mice. The median lethal dose, however, is significantly higher, and the relative potency in mice is 10 to 20 times less than for *L. indistinctus.*[170]

Clinical Presentation. In humans, *S. nobilis* of southern England has caused brief local pain and slight swelling, followed by local sweating and piloerection, facial flushing, and feverishness.[270] One report of *S. foravae* bite in southern Africa showed minimal local inflammatory response without systemic toxicity.[170] In one Australian report, a 2-year-old child bitten by a juvenile *Steatoda* (species unknown) developed lethargy, irritability, diaphoresis, and hypertension 22 hours after the bite. He improved gradually after administration of two ampules of red-backed spider *(Latrodectus)* antivenom.[226] In another report, 30% of *Steatoda* bites (including *S. capenis* and *S. grossa*) studied in Australia presented to emergency departments and poison centers with systemic effects and persistent pain. Local and systemic symptoms were similar to those of *Latrodectus* envenomation, but overall they were less severe.[130]

Treatment. In general, care is symptomatic and supportive. Similarities between the neurotoxins in *Steatoda* and *Latrodectus* venoms, however, suggest the use of *Latrodectus* antivenom in severely symptomatic cases. Several case studies from Australia using *Latrodectus* antivenom (red-backed spider antivenom, Commonwealth Serum Laboratories, Melbourne, Australia) for *Steatoda* bites have demonstrated an improvement in and decreased duration of symptoms in victims. However, controlled studies have not been done.[101,130]

Genus *Achaearanea*: Grey House Spiders

Biology. *Achaearanea* are common spiders worldwide. They make classic theridiid cobwebs, often in corners and windows of houses. They are excellent predators of insects in homes. Individuals are mottled brown with globose abdomens and are easily distinguished from *Latrodectus geometricus,* the brown widow, by the lack of an hourglass in their ventral abdomen.

Venom. Little information is available about *Achaearanea* venom, although taxonomic proximity to other Theridiidae suggests the possibility of similar composition.

Clinical Presentation. One series of five cases in Australia indicates that redness or a red mark has been the only common local manifestation of *Achaearanea* bites. In case reports, bites to limbs produce pain that can become severe. Patients report increasing pain in the first hour that may persist up to 24 hours. Local or regional diaphoresis is absent, with few reports of systemic effects. In two cases, headaches were reported, one with and one without nausea. This suggests that *Achaearanea* bites cause similar, but less severe, pain and systemic effects as *Latrodectus* bites.[130]

Treatment. Treatment of *Achaeranea* envenomation is symptomatic and supportive.

Figure 46-22. Orb weaver (*Argiope* species). (Courtesy Eileen Hebets.)

Family Araneidae: Orb-Weaving Spiders

Members of the family Araneidae are familiar to most people because they are common and build conspicuous, mainly circular, two-dimensional orb webs in open places. This is an abundant family, with 4000 species described and distributed worldwide. Species range in adult body size from 2 to 28 mm and often have extreme size dimorphism, with males much smaller than females. They are extremely diverse in size, shape, web design, and prey capture tactics. They can be brightly colored, with typically ovoid abdomens and large chelicerae with several teeth. Although diverse and abundant, they are of minimal clinical concern.

The venoms of many araneid spiders are known to have polyamine neurotoxins that postsynaptically block glutamate receptors in vertebrates.[138] These are currently known from species of *Nephila, Argiope, Araneus,* and *Neoscona.*

Genus *Argiope*: Argiopes

Biology. *Argiope aurantia,* known as the golden orb weaver or black and yellow garden spider, is common in California, Oregon, and the eastern United States (Fig. 46-22). Other *Argiope* species are found throughout the United States, the Orient, and Australia.[90] It is a large, brightly colored spider with a large, symmetric orb web and a leg spread of up to 7.5 cm.

Venom. Although *Argiope* venom appears to be cytotoxic in vivo, research indicates the venom has neurotoxic effects in vitro. Venom gland extracts from *Argiope trifasciata* are postsynaptic blockers of neuromuscular transmission at locust glutamate receptors.[253]

Clinical Presentation. Bites may cause local pain and erythema. Bites by *Argiope argentata* reportedly cause local pain, erythema, and vesicle formation, which resolve within 24 hours except for the bite marks.[97,281]

Treatment. Treatment is symptomatic and supportive.

The references for this chapter can be found on the accompanying DVD-ROM.

Scorpion Envenomation

47

Jeffrey R. Suchard

Scorpion envenomation can result in distinct clinical syndromes. Most scorpion species' stings cause only local pain and inflammation that respond well to minimal supportive therapy and wound care. These scorpions pose no significant management issues and, with few exceptions, are not discussed here in further detail. The truly dangerous scorpions of the world, typified by *Tityus* species in the Caribbean region and in South America, *Androctonus* species and *Buthus occitanus* in North Africa, *Leiurus quinquestriatus* in the Near East, and *Mesobuthus tamulus* in India, cause an "autonomic storm" with prominent cardiopulmonary effects. A third clinical syndrome occurs from stings of *Centruroides* species in the southwestern United States and Mexico and from *Parabuthus* species in southern Africa. These produce prominent neurologic effects associated with excess cholinergic tone. Systemic effects typically affect children more severely than adults, often requiring prompt medical management to avoid morbidity and mortality. The ideal treatment of scorpion envenomation remains controversial, primarily because controlled clinical trials are lacking. Although anecdotal experience and comparisons of historic cohorts demonstrate a benefit from aggressive symptomatic and supportive care, the proper use of antivenom has not been fully resolved.

TAXONOMY AND ANATOMY

Scorpions, which are grouped in the phylum Arthropoda (Fig. 47-1), have a crablike or lobsterlike body shape with seven sets of paired appendages (Fig. 47-2): the chelicerae, the pedipalps (claws), four sets of legs, and the pectines (a pair of comblike structures on the ventral surface). The segmented tail curves upward dorsally, ending in a terminal bulbous segment called the telson, which contains paired venom glands and the stinger (Fig. 47-3). The presence and size of a subaculear tooth, a small tubercle near the base of the stinger, vary among species and with stage of maturity. In the United States, a subaculear tooth on a small, slender scorpion usually indicates *Centruroides exilicauda* (sometimes called *Centruroides sculpturatus*) (Fig. 47-4).[47,148]

Scorpions grasp prey in their pedipalps and then rapidly thrust the tail overhead to sting. The chelicerae tear the food apart. Scorpions feed primarily on ground-dwelling arthropods and small lizards. The scorpion consumes only the juices and liquefied tissues of its prey, discarding the solid parts. Scorpions envenom by stinging, not biting. Although envenomations are

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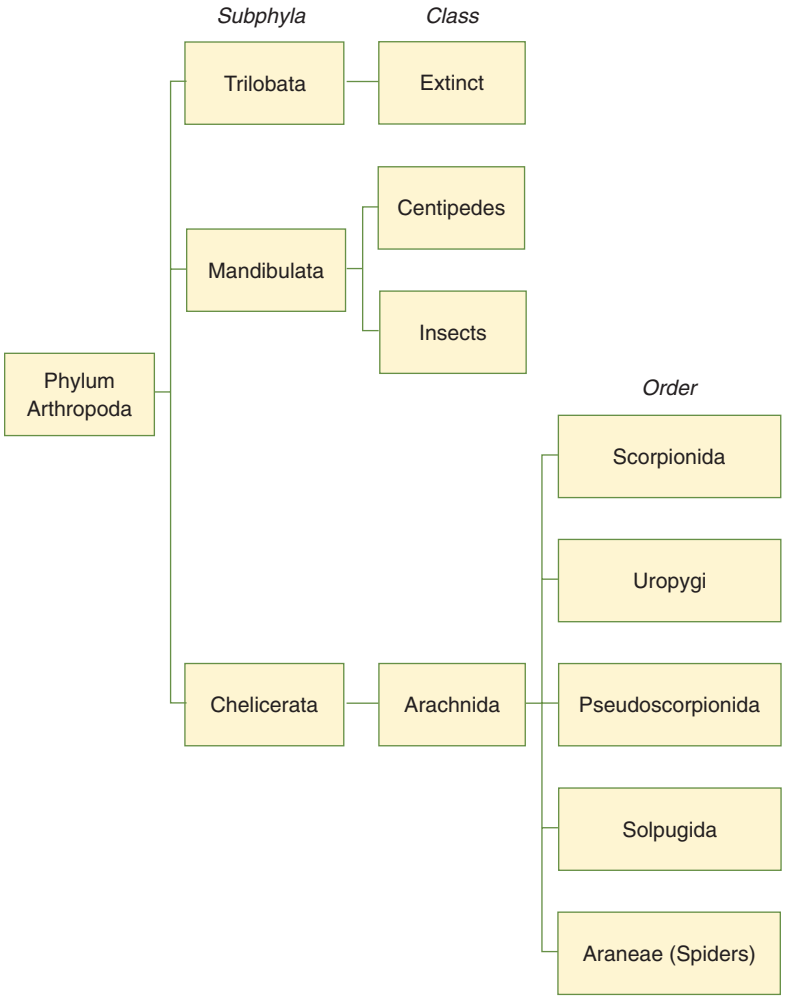


Figure 47-1. Organization of the phylum Arthropoda, showing the relationship of scorpions to spiders and more distantly related animals.

sometimes reported as bites,[15,52,142] true scorpion bites have not been documented and would be inconsequential if they did occur.

A characteristic physical property of scorpions is that they fluoresce when illuminated by ultraviolet light, as from a black light or a medical Wood's lamp (Fig. 47-5). This property is used in collecting scorpions for breeding or venom harvesting and in providing pest control. The fluorescent pigment in scorpion cuticle is probably riboflavin.[109]

Scorpions can sting multiple times; however, it appears that the first sting depletes or nearly depletes the telson of venom. A case series of three pairs of scorpion sting victims from India found that consecutive stings by *Mesobuthus tamulus* caused severe cardiovascular manifestations in the first victim but not in the second.[17] The author has anecdotally noted a similar difference in the severity of neurologic manifestations from consecutive *C. exilicauda* stings in Arizona.

GEOGRAPHIC DISTRIBUTION

Scorpions are widely distributed in regions within 50 degrees north and 50 degrees south of the equator[96] and are found on all continents[133] except Antarctica. Scorpions are characteristic of desert areas, semiarid grasslands, and the tropics, but they may also be found in temperate and subtropical regions.[85,96,109,133] An estimated 5000 deaths from scorpion stings occur annually worldwide,[72] making scorpions second only to snakes as sources of fatal envenomation.[170]

Estimates vary regarding the number of scorpion species. In 1985, Herschkovich and colleagues[95] reported the existence of 500 scorpion species divided into six families. Russell[148] reported 500 to 800 species, others reported 650,[33,41,172] and Neale[133] estimated at least 700 species. More recent reports estimate 1000 species,[72,152] and in 1998, Hutt and Houghton[96]

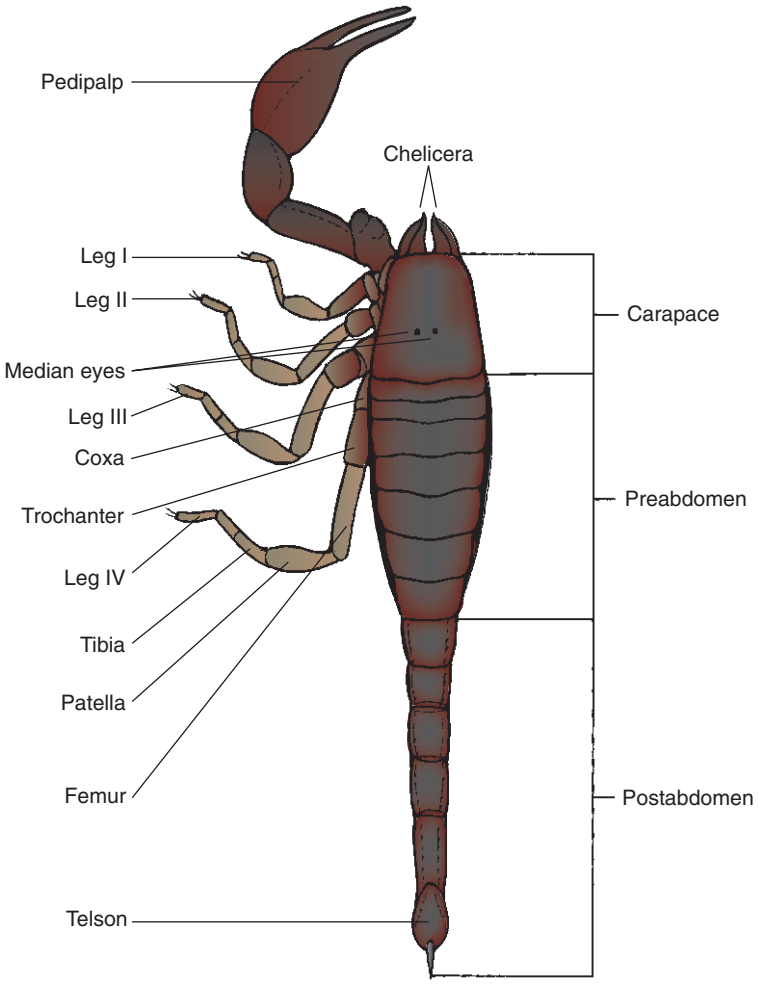
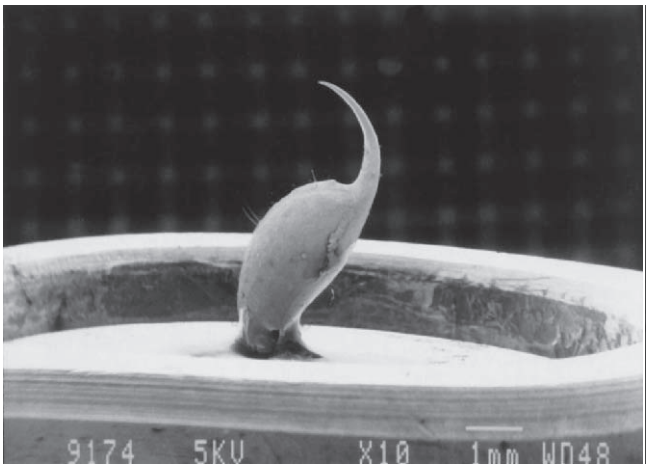


Figure 47-2. Anatomy of a scorpion. (Redrawn from Keegan HL: Scorpions of Medical Importance. Oxford, MS, University Press of Mississippi, 1980.)

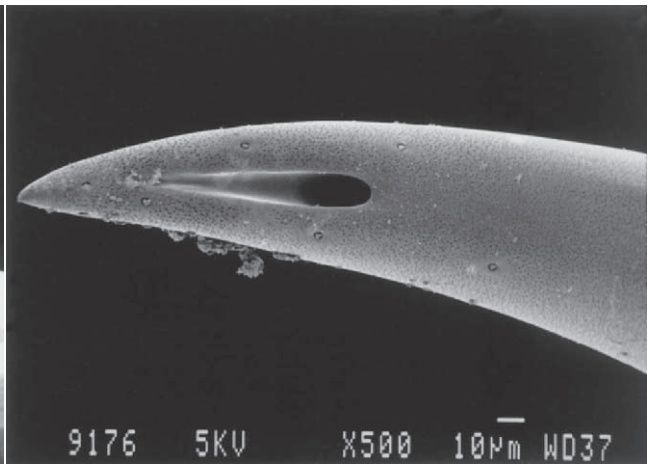
reported 1400 scorpion species divided into nine families (Fig. 47-6). Buthidae is the largest and the most dangerous family and, with few exceptions, contains the only species capable of producing clinically significant envenomations, through their neurotoxic venoms.[85,96,152] At least 30 species can inflict potentially fatal stings.[72] All of the genera commonly recognized as dangerous are buthid scorpions: *Centruroides* and *Tityus* in the Western Hemisphere, and *Androctonus, Buthus, Mesobuthus, Leiurus,* and *Parabuthus* in the Eastern Hemisphere.

VENOM

Scorpion venoms are complex mixtures containing mucopolysaccharides, hyaluronidase, phospholipase, acetylcholinesterase, serotonin, histamine, protease inhibitors, histamine releasers, and neurotoxins.[48,152] Neurotoxins are pharmacologically the most important venom constituents.[152] *C. exilicauda* venom glands contain two types of columnar cells, one secreting mucus and another producing neurotoxins. This species' venom has no enzyme that causes tissue destruction, however, so local effects are minimal or absent.[47] The neurotoxins are single-chain, basic polypeptides of 60 to 70 amino acids, reticulated by four disulfide bridges. Each scorpion species' venom contains several neurotoxins, but they all share a similar structure and homologous sequences.[79,152] In neuronal membranes, these toxins cause two effects with regard to fast sodium channels involved in action potential transmission: (1) incomplete inactivation of sodium channels during depolarization, resulting in a widening of the action potential, and (2) a slowly developing, inward sodium current after repolarization, leading to membrane hyperexcitability. The net result is repetitive firing of axons,[47] enhancing release of neurotransmitters (acetylcholine, norepinephrine, dopamine, glutamate, aspartate, γ-aminobutyric acid) at synapses and at neuromuscular junctions.[61,69,147,168] This is clinically demonstrated as excessive neuromuscular activity and autonomic dysfunction. Some scorpion neurotoxins also have effects on calcium-activated potassium channels,[73,146] chloride channels,[51] and L-type calcium channels.[12]



A



B

C

Figure 47-3. **A,** Electron micrograph of scorpion telson. **B** and **C,** Close-up images demonstrating stinger with paired venom pores.

Figure 47-4. *Centruroides exilicauda (C. sculpturatus),* the bark scorpion of Arizona.

Figure 47-5. Scorpions fluoresce in ultraviolet light.

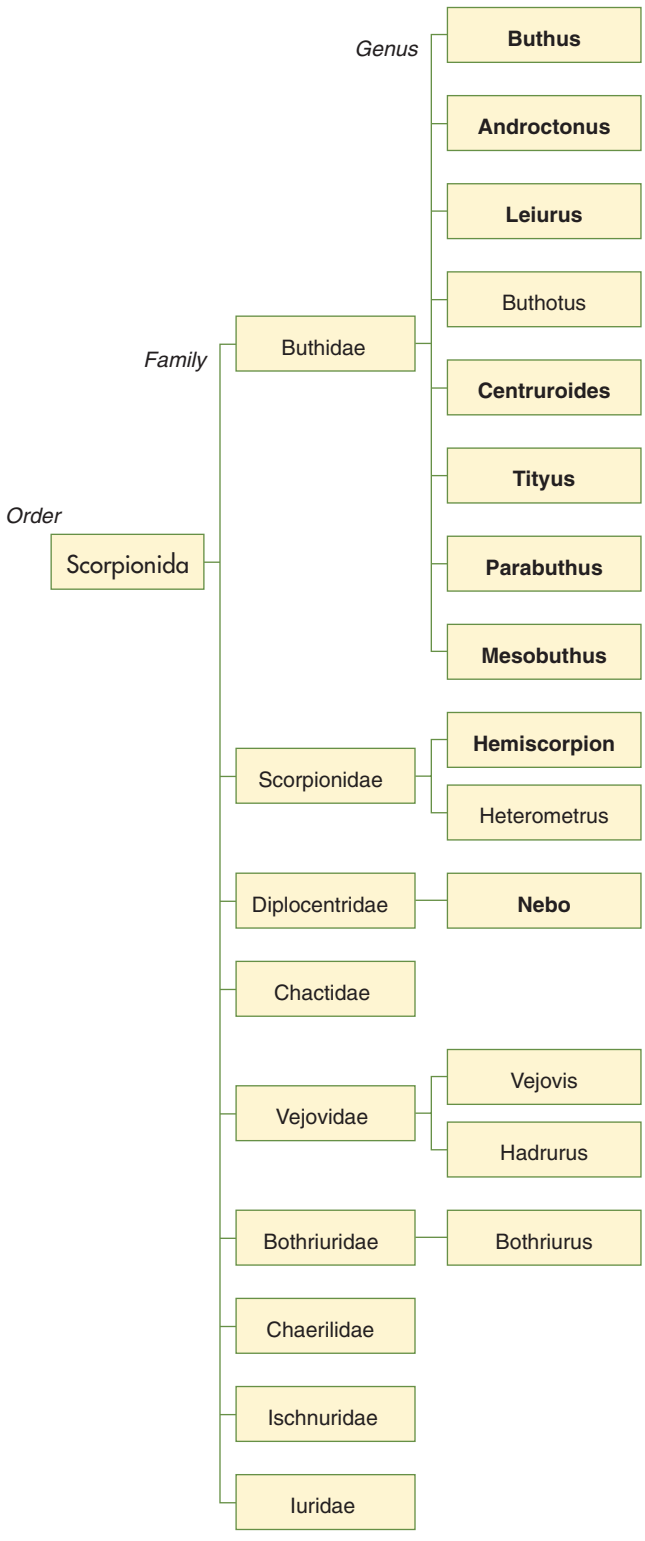


Figure 47-6. Families of the Scorpionidae order. The Buthidae family contributes the largest number of medically significant genera, as indicated by *bold type*.

REGIONAL EPIDEMIOLOGY

India

Between 86 and 99 scorpion species, with at least 45 buthid species, are found in India. Only one species is regarded as dangerous: *Mesobuthus tamulus,* formerly known as *Buthus tamulus,* and commonly called the red scorpion.[50,128] *Heterometrus* (formerly *Palamnaeus*) *gravimanus,* the black scorpion, is a larger species that does not cause systemic toxicity.[18,170] *Heterometrus bengalensis* is common in eastern India.[107] *M. tamulus* is a particular problem in southern coastal India. Stings occur predominantly in April, May, and June at night among young farmers wearing minimal clothing.[18,25] In many cases, stings occur at the tip of an extremity, with the only symptom being pain, which can be controlled with local anesthetic injections.[25,49] Systemic toxicity occurs from release of catecholamines, with major morbidity and mortality resulting from cardiopulmonary toxicity.[16,18,22,25,33,49] The 30% fatality rates reported in the 1960s and 1970s are now 2% to 3% with treatment using vasodilators and calcium channel blockers.[20,22] An 11.8% mortality rate, however, was found among 152 children admitted in Calcutta from 1985 to 1989, but treatment details were not reported.[33]

M. tamulus antivenom is produced for research purposes only. It is not commercially available and would probably not be available in the predominantly rural environment where most stings occur. Bawaskar and Bawaskar[16–25] have developed treatment protocols recognizing the limited medical resources available for the majority of victims, including the potential risks of transporting unstable patients. They recommend oral prazosin and nifedipine for victims with adrenergic toxicity and intravenous (IV) nitroprusside for severe pulmonary edema.

Iran

Radmanesh[139,140] has reported on scorpion envenomation in Khuzestan, a hot and humid province in southwest Iran. A specialized scorpion sting department was established in the provincial capital to study and treat this public health concern, because fatalities occur, especially among children in rural areas during the hot seasons. Over 6 months, 3217 patients were referred to the scorpion sting department, with 200 admitted and the remainder treated as outpatients. Three scorpion species were responsible for almost all cases: *Androctonus crassicauda* (41%) and *Mesobuthus eupeus* (45%) of the Buthidae, and *Hemiscorpion lepturus* (13%) of the Scorpionidae.[139] Systemic envenomations by *A. crassicauda,* the Khuzestan black scorpion, resulted in prominent cholinergic signs, such as exocrine gland hypersecretion, urinary frequency and incontinence, and increased gastrointestinal (GI) motility. Adrenergic effects also occurred with lesser frequencies. Neurologic toxicity manifested as delirium, confusion, coma, restlessness, convulsions, localized muscle spasm near the sting site, opisthotonus, and paralysis. A polyvalent scorpion antivenom was ineffective in this series of *A. crassicauda* envenomations.[139]

Hemiscorpion lepturus was responsible for 10% to 15% of stings during the hot season but caused almost all reported stings during winter. This scorpion has a cytotoxic venom, unlike the buthid neurotoxins. Most victims develop a 3- to 4-mm dark blue macule surrounded by a 1- to 2-cm red halo within the first hour. The skin lesion may enlarge, become indurated and inflamed, and eventually necrose and slough.

Figure 47-7. *Leiurus quinquestriatus,* the yellow scorpion of North Africa and the Middle East. (Courtesy R. David Gaban.)

Serious skin lesions are associated with hemolysis and renal failure. Central nervous system (CNS) and cardiovascular toxicity may be seen in severely envenomed patients. Ankylosis of the joints and psychologic sequelae have also been reported. Local authorities recommend surgical intervention for skin lesions, including prophylactic excision of the sting site, as well as supportive care for hemolysis and renal failure. No antivenom is available.[140] Fatal *H. lepturus* envenomation associated with renal failure has also been reported in Pakistan.[132] No prospective or controlled trials of excisional therapy for *H. lepturus* envenomation have been published, and routine or prophylactic surgical intervention is not recommended for envenomation by other scorpion species.

Israel

Leiurus quinquestriatus, known as the five-keeled gold scorpion or the yellow scorpion, is the most dangerous species found in Israel[35,64,95,146] (Fig. 47-7). Other native species include *Buthotus judaicus* (the black scorpion), *Androctonus crassicauda, Androctonus bicolor, Nebo hierochonticus, Scorpio maurus,* and *Orthochirus innesi.*[34,35,64,156] More than 90% of all scorpions encountered in neighboring Jordan are either yellow or black scorpions (*L. quinquestriatus* or *B. judaicus*),[64] with the yellow being most common.[95] Most cases occur during the warmer months of April through October.[27] In the Negev desert region, Bedouin children are stung about six times more frequently than Jewish children, probably because of more time spent outdoors and lack of protective footwear. Males are affected 2.3 times more often than females, related to differences in gender roles, such as boys herding sheep or goats.[95] The reported mortality rate in children was 18% among Palestinians living on the West Bank in 1965, 3.7% among children in the Jerusalem area in 1991,[64] and 1.2% among children in the Negev area in 1985.[95] Most scorpion stings have a mild clinical course: 13% of reported sting victims remain asymptomatic, 72% have mild illness, and 15% become moderately to seriously ill.[27]

The *L. quinquestriatus* sting initially produces intense local pain, erythema, and edema, which can be followed by an outpouring of catecholamines and acetylcholine from nerve endings. In severe cases, clinical signs of sympathetic overload predominate, with severe hypertension, tachyarrhythmias, and pulmonary edema.[1,85–87,141] Parasympathomimetic action of the venom may also cause bradyarrhythmias or atrioventricular block, usually preceding the sympathetic overload. Cardiomyopathy and myocardial damage with electrocardiographic (ECG) and serum marker (creatine kinase [CK], CK-MB, troponin) changes have been reported.[64,85,151,158] Other common findings from severe stings include agitation, convulsions, encephalopathy, hypersalivation, diaphoresis, priapism, and pancreatitis.[2,154,155] Treatment recommendations differ, but all emphasize aggressive symptomatic and supportive care for severely envenomed patients. However, some authors propose the routine use of antivenom,[64] whereas others argue that serotherapy does not significantly alter outcome based on experimental pharmacokinetic data.[85–88] Antivenom also had no demonstrable effect in one clinical series of Israeli patients.[157]

Saudi Arabia

At least 14 species of scorpions are found in Saudi Arabia; the two most common are *Androctonus crassicauda,* a black or dark brown scorpion, and *Leiurus quinquestriatus,* a yellow scorpion associated with more stings.[10,66,103,112,133] Scorpion envenomation is responsible for 3% to 4% of all pediatric hospital admissions in northwestern Saudi Arabia from May to August, with few admitted in other seasons.[65,66] One author reported the incidence of "scorpion sting syndrome" as 1.3 cases per 1000 emergency department patients,[133] whereas a 15-year retrospective analysis from another hospital in the same city (Riyadh), with over 100,000 visits annually, reported only 251 cases.[6] Seventy-six percent of cases occurred between May and October, and 73% of stings occurred at night between 6 PM and 6 AM.[133] Many victims are children playing barefooted outdoors or persons tending flocks of goats or sheep. Males are affected at least twice as often as females.[10,65,133] Mortality rates range from 2% to 5%.[10,65] Antivenom is recommended and routinely administered for scorpion envenomation in Saudi Arabia.[10,65,66,103]

L. quinquestriatus envenomations were reviewed earlier. *A. crassicauda* stings are similar to those of the yellow scorpion, causing hypertension and CNS manifestations, but differ in other ways.[103] The pain from the *A. crassicauda* sting has been reported as particularly severe. Generalized erythema was noted in 20% to 25% of children less than 5 years of age; this is not usually seen with other scorpion stings. The cause of this erythema is not clear, especially because elevated catecholamine levels after scorpion envenomation appear to be protective against allergic reaction. Cholinergic effects are seen less often with *A. crassicauda* stings.

Scorpion envenomation became an issue to U.S. soldiers deployed during the Gulf War.[81] *L. quinquestriatus* and *A. crassicauda* were implicated in 57 scorpion stings over 4 months among 7000 troops of an armored cavalry division stationed in eastern Saudi Arabia. All patients with adequate data for further study recovered fully, usually with only supportive care in the field, probably reflecting that all were healthy adults. No antivenom was available. Typical signs and symptoms included local pain, tachycardia, hypertension, sweating, apprehension, headache, epigastric pain, nausea, restlessness, and local muscle cramping and paresthesias in lower extremity stings. Presumably, victims with only local pain failed to present to battalion aid stations, resulting in an apparently high incidence of systemic effects. Only two persons had significant presentations or subsequent complications. One of these had a clinical picture consistent with anaphylaxis and required intubation for respi-

Figure 47-8. *Androctonus australis,* responsible for most severe scorpion envenomations in North Africa. (Courtesy R. David Gaban.)

Figure 47-9. *Androctonus amoreuxi,* demonstrating the thin claws and thick tail characteristic of dangerous scorpion species. (Courtesy R. David Gaban.)

ratory support. The other victim developed a cutaneous ulcer that healed in 3 weeks with oral antibiotic therapy.[81]

North Africa

Scorpions are a common problem throughout North Africa. Hundreds of scorpion sting deaths occur annually in Algeria.[114] Libya reported 900 stings with seven fatalities per 100,000 population in 1979.[96] *Buthus occitanus* and *Leiurus quinquestriatus* cause the majority of stings in Upper Egypt.[124] Most of the published North African scorpion research is from Tunisia, where 10 scorpion species are found, five of which are responsible for almost all stings.[80] Most stings are caused by *Androctonus australis garzonii, Androctonus aeneas aeneas,* and *Buthus occitanus tunetanus. A. australis,* known as the yellow scorpion, accounts for most severe envenomations (Fig. 47-8). *A. aeneas* is a dangerous black scorpion found only in the southern part of Tunisia. The next most common species are *Scorpio maurus tunetanus* and *Euscorpius carpathicus sicanus,* the latter being another black scorpion found only in the north; both are relatively harmless and have thin tails and thick claws. The more dangerous Tunisian scorpions have long, thin claws and a thick tail[80,110] (Fig. 47-9). Stings occur most often outdoors (92%) on the victim's extremities (95%) during the summer months.[110] Eighty percent of all reported stings occur from July to September, with half of these in August.[80]

Between 30,000 and 45,000 scorpion stings are reported annually in Tunisia, correlating to an incidence of 4.5 to 20 stings per thousand inhabitants, depending on the location. About 2.5% of stings (900 to 1100 per year) result in systemic manifestations requiring hospital admission. The mortality rate ranges from 0.25% to 0.4%, which is about 10% of victims with systemic envenomations, or 35 to 105 deaths per year.[5,80,109–111] Two thirds of reported stings affect adults and older adolescents, but nearly all fatalities occur in younger children; the mortality rate for children is about 1%.[80,110]

The effects of Tunisian scorpion stings have been classified into four stages[80] and three grades[111] based on clinical severity. The first three stages or grades are essentially identical, and the fourth stage is the most severe form of grade III envenomation. The stages or grades do not necessarily reflect the natural time course of envenomation and are designated to aid risk evaluation and to direct treatment.

Grade I envenomations have only local symptoms at the sting site. The most common complaint is intense burning pain. Paresthesias occur in 92% of victims. Mild systemic symptoms, such as irritability and restlessness, occur in 8% to 12% of victims. Local tissue alterations are rare. Grade I cases constitute 90% to 95% of all scorpion stings, and symptoms typically resolve in 3 to 8 hours.

Grade II envenomations have local symptoms as in grade I, sometimes with local edema, associated with moderate systemic symptoms. Irritability (52%), restlessness (48%), tachycardia (34%), and moderate hyperthermia (27%) are characteristic features. Signs of excess cholinergic tone (e.g., diaphoresis, hypersalivation, rhinorrhea, vomiting, diarrhea) are often found, as well as occasional dyspnea, gastric distention, and priapism. From 5% to 10% of stings occur with grade II symptoms. The prognosis is favorable, with return to baseline in 3 days, although 1% to 3% of patients initially classified as grade II progress to grade III.

Grade III envenomations involve serious systemic complications, including cardiocirculatory shock, respiratory failure, pulmonary edema, hyperthermia, seizures, priapism, and coma. If the grading system is divided into four stages, the stage III victims have depressed level of consciousness, arterial hypertension, and tachycardia, usually occurring within 2 to 4 hours after the sting. ECG changes consistent with ischemia, respiratory failure, hyperglycemia, and an elevated white blood cell count are also seen. Stage IV signifies worsening neurotoxicity, heralded by profuse vomiting and associated with cardiovascular collapse, pulmonary edema, hyperthermia, seizures, and coma. Even patients with stage III envenomations have a favorable prognosis with appropriate care. Patients who recover regain consciousness in several hours, and the ECG abnormalities resolve in 2 to 4 days. All fatalities progress through stage IV, although recovery from stage IV can occur.[80]

South Africa

The majority of scorpion stings in South Africa, Zimbabwe, and neighboring countries do not cause systemic effects, although

Figure 47-10. *Parabuthus transvaalicus,* a dangerous scorpion of southern Africa. (Courtesy R. David Gaban.)

Figure 47-11. *Hadogenes troglodytes,* an impressive-looking yet relatively harmless scorpion of southern Africa, has large claws and a thin tail. (Courtesy R. David Gaban.)

fatalities occasionally occur.[30,31,113] Scorpions found in southern Africa include the frequently dangerous Buthidae with thin pincers and thick tails (Fig. 47-10) and the relatively harmless Scorpionidae, Bothriuridae, and Ischnuridae with thick pincers and thin tails[31,113,127] (Fig. 47-11). *Parabuthus* species cause neuromuscular toxicity without the autonomic storm seen from the dangerous scorpions of northern Africa, the Mideast, and India.[30,31,127] At least 20 *Parabuthus* species are distributed throughout South Africa, Namibia, Botswana, Zimbabwe, and southern Mozambique.[127] Three other species can produce systemic effects but are not considered potentially fatal: *Parabuthus mossambicensis*, *Uroplectes planimanus,* and *Opistophthalmus glabrifrons*.[29,30] Certain *Parabuthus* scorpions with large venom vesicles are capable of spraying venom when alarmed.[127]

Stings typically occur in the early evening during the warmer months of October through April, with peak incidence in January and February.[30,31,127] Four children died in a series of 42 serious scorpion envenomations from South Africa.[127] No fatalities were noted among 244 patients (17 with severe systemic symptoms) in Zimbabwe.[31] In another study, however, five deaths occurred among 455 patients, with the fatalities in children less than 10 years old or adults over 55.[30] The overall case fatality rate from *Parabuthus* species ranges from 0.3% to 3%.[30,31]

Serious scorpion envenomation in southern Africa closely resembles that seen in the American Southwest from *Centruroides exilicauda*.[30,31,127] Immediate pain, local paresthesias, and hyperesthesia typically occur. Mild cases (60% of stings) are associated with only localized symptoms, moderate cases with three or fewer systemic features, and severe cases (10% to 30%) with more than three systemic signs or symptoms. The pain and paresthesias may generalize, sometimes within minutes in children and usually within 4 to 12 hours in adults. Of victims with severe envenomations, 75% have difficulty swallowing, myoclonic jerks, tongue fasciculations, hypersalivation, and profuse sweating; 50% have bilateral ptosis, slight local swelling, and difficulty urinating (33% with a palpably distended bladder). Children are typically affected more severely and are more likely to exhibit the uncontrollable jerking, writhing, and thrashing movements characteristic of neurotoxic scorpion envenomation. Respiratory failure is the most common proximate cause of death.

In untreated cases, urinary retention, ptosis, and sweating resolve in 2 days and dysphagia and hypersalivation in 4 days. After 1 week, 10% of victims still have muscle tremors and tongue fasciculations, and more than half still have localized pain at the sting site. Traditional herbal remedies are frequently used but have no apparent beneficial effect. In fact, rubbing the sting site, as commonly practiced with such herbal remedies, more than doubles the chance of developing a severe envenomation. Antivenom against *Parabuthus transvaalicus* is available commercially and recommended for victims with serious envenomations (see Figure 47-10).

Brazil

About 10,000 cases of scorpion envenomation are reported annually in Brazil,[126] with 80% occurring in southeastern regions.[134] Half the reported stings occur in the state of Minas Gerais,[126] although scorpions are also problematic in São Paulo and Bahia.[38] Most stings occur from December through February.[40,71] *Tityus serrulatus* is the most prevalent species in Brazil and accounts for most fatal stings.[45,126] *Tityus bahiensis* is the next most common species,[134] although severe envenomations are much more likely from *T. serrulatus*.[38] Equine-derived antivenom for either *T. serrulatus* or both *T. serrulatus* and *T. bahiensis* is able to neutralize venom from all Brazilian scorpions studied.[134] Children are much more likely than adults to have severe envenomations.[71] In Minas Gerais, children less than 14 years of age accounted for 27% of scorpion envenomation admissions but for all cases of significant morbidity and mortality; 16% were treated in an intensive care unit (ICU) setting. Mortality was 3.5% in 1938[40] but with current treatment now ranges from 1% to 1.1% in children and is 0.28% overall.[71,126,145] Antivenom from a few manufacturers is available in Brazil and routinely used in severe envenomations.[38,40,71,145]

Argentina

Among more than 100 species found in Argentina, only *Tityus trivittatus* has been identified as dangerous to humans. Over a 7-year period, 511 scorpion envenomations were reported to public health authorities.[57] Most cases occurred indoors and

during the warmer months. Scorpions are hyperendemic in the provinces of Córdoba and Santiago del Estero, and 85.6% of envenomations occurred there. Local pain and inflammation were most common, whereas systemic envenomation occurred in 7% of children 10 years and younger and in 2% of older victims. Antivenom therapy was provided in 84% of cases with available treatment records; only three fatalities occurred, and none of these victims had received antivenom. Until 1996, the Argentinians used a cross-reactive Brazilian antivenom against *T. serrulatus*. Since 1997, Argentina has produced a $F(ab')_2$ fragment equine antivenom.[57]

Trinidad

Tityus trinitatis accounts for almost 90% of the scorpion population on Trinidad. Fatalities are rare but occur more often in children. Stings are more frequent in summer months.[14] Systemic effects of serious *T. trinitatis* envenomations include tachypnea, restlessness, vomiting, hypersalivation, cerebral edema, pulmonary edema, hypovolemic shock, seizures, and myocarditis.[48] The most striking clinical observation is the high incidence (up to 80%) of acute pancreatitis; scorpion stings are the most common cause of acute pancreatitis in Trinidad.[14,75]

Venezuela

Tityus discrepans is the most common scorpion in Venezuela.[58,62] The states of Monagas and Sucre in eastern Venezuela are particularly endemic for scorpions.[58] A case series of 64 patients from the state of Merida in southwestern Venezuela classified scorpion envenomations by clinical criteria.[122] Whereas 27 patients had only local manifestations, the remainder had systemic effects of envenomation: 21 primarily had GI complaints, nine had neurologic symptoms and hypertension, and seven had severe envenomations with cardiac arrhythmias, pulmonary edema (five patients), and fatal cardiogenic shock (two patients). These last seven patients all received antivenom. The survivors received antivenom within 5 hours, whereas the children who died received antivenom more than 5 hours after envenomation.[122]

Mexico

An estimated 200,000 scorpion stings occur annually in Mexico.[53] Of at least 134 native species, eight members of the *Centruroides* genus are recognized as significantly dangerous (Fig. 47-12): *Centruroides elegans, Centruroides infamatus infamatus, Centruroides limpidus limpidus, Centruroides limpidus tecomanus, Centruroides noxius, Centruroides pallidiceps, C. sculpturatus (exilicauda),* and *Centruroides suffusus suffusus*.[53] *C. noxius* has the most potent venom,[39,115] but *C. suffusus* is usually cited as the most dangerous Mexican scorpion.[152,172] *Centruroides* scorpions are relatively small and described as yellow, tan, or brown. Clavigero recognized as early as 1780 that "the venom of the small and yellow scorpions is more active than that of the big grey ones."[123] The 11 Mexican states on the Pacific coast are particularly endemic places for scorpions, with Colima, Nayarit, Guerrero, and Morelos having the highest mortality rates from scorpion envenomation.[53,123] Morelos has the highest incidence of scorpion envenomation, with a total of 30,663 stings and nine deaths in 1999. Nearly 2% of the population in Morelos is stung annually, and in some hyperendemic villages this figure is over 10%.[143] In the 1940s, 7.4 to 8.9 scorpion sting deaths occurred per 100,000 population. This rate decreased to 3.4 to 4.7 per 100,000 in 1957–1958. The great majority occurred in very young children. Approximately 10 times more deaths occurred from scorpions as from all reported snakebites. Most fatal cases occur in the summer months from April through July.[123] In the 1980s, 272 to 401 scorpion fatalities per year occurred nationwide, with respiratory failure as the proximate cause of death; this figure probably underestimates the true total by a factor of 2 to 3.[53]

Figure 47-12. *Centruroides limpidus limpidus,* one of several closely related, dangerous Mexican scorpions. (Courtesy R. David Gaban.)

The following signs and symptoms have been reported with Mexican scorpion stings, although not all effects are necessarily seen in the same victim, and no apparent sequence of effects has been observed: hyperexcitability, restlessness, hyperthermia, tachypnea, dyspnea, tachycardia or bradycardia, diaphoresis, nausea, vomiting, gastric distention, diarrhea, lacrimation, nystagmus, mydriasis, photophobia, excessive salivation, nasal secretion, dysphagia with foreign body sensation, dysphonia, cough, bronchorrhea, pulmonary edema, arterial hypertension or hypotension, heart failure, shock, convulsions, ataxia, fasciculations, and coma.[53] Many of these effects appear to be caused by autonomic nervous system dysfunction, which can be seen from either neurotoxic or cardiotoxic scorpions. Unpublished verbal reports by physicians who have treated victims of scorpion envenomation in Mexico suggest that the clinical presentation is virtually identical to that caused by *C. exilicauda* in the United States. The higher mortality from Mexican scorpion stings compared with American stings most likely results from differences in the human and scorpion population densities, in the ease of access to medical care (e.g., monitoring equipment, ICUs, and antivenom), and perhaps also from cultural differences in housing and protective clothing that promote human–scorpion interactions in Mexico.

Currently, a polyvalent antivenom protective against all native *Centruroides* species is produced by injecting horses with a mixture of macerated venom glands from the most important species (*C. noxius, C. l. limpidus, C. l. tecomanus,* and *C. suffusus*). Antivenom is recommended and often used in patients with systemic symptoms.[53]

United States

About 40 species of scorpions are found in the United States.[41] Only *C. exilicauda* causes a significant number of systemic

reactions and is known to be potentially fatal.[28,41,47,148,161] Approximately 30 *Centruroides* species are found distributed throughout the New World,[148] several of which are of medical importance and are mostly found in Mexico.

C. exilicauda (the currently preferred taxonomic designation) is also known as *C. sculpturatus,* or the bark scorpion, because of its preference for residing in or near trees. These scorpions also often hide under wood (old stumps, lumber piles, firewood, loose bark, or fallen trees), in ground debris, or in crevices during the daytime. This is troublesome to humans, because the scorpions may hide in shoes, blankets, or clothing left on the floor during daylight hours, as well as under common ground covers and tents. *C. exilicauda* is found statewide in Arizona and also in some areas of Texas, New Mexico, northern Mexico, small areas of California, and near Lake Mead, Nevada.[47] The bark scorpion is relatively small, measuring up to 5 cm in length. Specimens are variously described as being a uniform yellow, brown, or tan; stripes are uncommon. The pincers (pedipalps) and tail are thin, giving the scorpion a streamlined appearance, in contrast to several of the larger but less dangerous scorpions with thick claws and tails. The presence of a subaculear tooth, a tubercle at the base of the stinger, is distinctive to *C. exilicauda* and is helpful in differentiating this neurotoxic scorpion from other species.[47,161]

The bark scorpion presents a significant public health problem in Arizona. About 10% of all calls received by the Banner Poison Control Center in Phoenix are related to scorpion stings, the vast majority of which are known or highly suspected to have been caused by *C. exilicauda*. The bark scorpion was at one time the number-one killer among Arizona's venomous animals. From 1931 to 1940, more than 40 deaths were attributed to envenomation by this scorpion, mostly in young children and infants. The fatality rate fell dramatically between 1940 and 1970, probably because of improved pest control measures and advances in medical technology and supportive care. In 1972, these scorpion stings were still considered generally fatal to infants less than 1 year of age without treatment, extremely dangerous to older children, and occasionally fatal to adults with hypertension.[161] No deaths have been reported from scorpion envenomation in Arizona since 1968.[28] However, in 2001 a scorpion-related fatality was reported, although death was ascribed to anaphylaxis rather than to direct venom toxicity, in a patient previously stung and presumably sensitized to scorpion venom.[37] Because death can apparently be prevented with currently available supportive care, prior fatalities were probably caused by loss of upper airway and respiratory muscle control with the potential for aspiration, exacerbated by metabolic acidosis, hyperthermia, and rhabdomyolysis from excessive muscular activity.[47]

Centruroides vittatus, the common striped scorpion, accounts for the most reported envenomations in the United States, after *C. exilicauda*. *C. vittatus* has a black intraocular triangle and black stripes on the thorax (Fig. 47-13). A review of 558 *C. vittatus* stings reported to Texas poison centers in 1997 found that 96% produced local symptoms of pain, bleeding, burning sensations, erythema, edema, hives, local paresthesias, and pruritus; systemic reactions occurred in 20% of victims.[162] The most common systemic features were paresthesias of the face, tongue, and perioral region, followed by dysgeusia, chills, sweating, dysphagia, fasciculations, nausea, and vomiting. *C. vittatus* is found primarily in the Southwest and Texas but also extends into southern Indiana and Illinois.[167]

Figure 47-13. *Centruroides vittatus,* the striped scorpion found in central and southwestern United States. (Photo courtesy of Dr. Richard M. Houseman, Department of Entomology, University of Missouri-Columbia.)

Figure 47-14. *Hadrurus arizonensis,* a "giant hairy scorpion" of the American Southwest. (Courtesy R. David Gaban.)

Hadrurus species are the longest and most heavy-bodied scorpions native to North America and are known as "giant hairy scorpions" because of their size and conspicuous bristles (Fig. 47-14). They are native to Arizona, California, and parts of Utah, Nevada, Idaho, and Mexico. *Vejovis* species have a wide distribution from southern Canada, south through Wyoming, Colorado, and Texas, and west to California. Hypersalivation after *Vejovis* envenomation has been reported. *Uroctonus* species are found in mountain habitats from southern California to Oregon.[148] *Isometrus maculatus* is the only scorpion found in Hawaii,[121] and it is also found in southern Florida and California.[41] Envenomations cause mild systemic effects (myalgia and nausea); no fatalities have been reported.[121] *Diplocentrus* species have been found in Florida, Texas, and California.[41] The Midwest and New England are not natural scorpion habitats, although three envenomations in Michigan were

Box 47-1. Grades of *Centruroides exilicauda* Envenomation

I. Local pain and/or paresthesias at site of envenomation
II. Pain and/or paresthesias remote from the site of the sting, in addition to local findings
III. Cranial nerve dysfunction or somatic skeletal neuromuscular dysfunction
    1. Cranial nerve dysfunction: blurred vision, wandering eye movements, hypersalivation, difficulty swallowing, tongue fasciculations, upper airway problems, slurred speech
    2. Somatic skeletal neuromuscular dysfunction: jerking of the extremity (or extremities), restlessness, severe involuntary shaking and jerking that may be mistaken for a seizure
IV. Cranial nerve dysfunction and somatic skeletal neuromuscular dysfunction

Modified from Curry S, Vance MV, Ryan PJ, et al: J Toxicol Clin Toxicol 21:417–449, 1983–1984.

caused by scorpions unintentionally transported with personal belongings or with produce, two by *Centruroides hentzi* from Florida, and one by *C. exilicauda* from Arizona.[167] The poison control center in Phoenix has also been consulted regarding stings from bark scorpion stowaways in the mail to Minnesota or personal belongings to Germany.

Centruroides exilicauda Envenomation

Stings from *C. exilicauda* often produce significant neuromuscular effects without severe cardiopulmonary toxicity. Curry and coworkers[47] reviewed clinical findings after *C. exilicauda* envenomation and proposed four clinical grades of envenomation intended to direct treatment (Box 47-1). Grade I envenomation is characterized by local pain and paresthesias at the sting site. Usually, no local inflammation occurs, and the puncture wound is too small to be observed. If no scorpion is seen, the diagnosis may require historical or epidemiologic clues or other physical signs. The "tap test" has been empirically recommended to confirm a bark scorpion sting, although its reliability has not been rigorously tested. With the patient looking away or otherwise distracted, gently tapping the sting site will greatly exacerbate the pain, a sign that does not occur with other envenomations.[47,161]

Victims with grade II envenomations have local symptoms plus pain and paresthesias remote from the sting site. The more distant symptoms often radiate proximally up the affected extremity but may occur in even more remote sites (e.g., contralateral limbs) or as generalized paresthesias. Victims may complain of a "thick tongue" and "trouble swallowing" in the absence of objective motor abnormalities. Children and adults frequently rub their nose, eyes, and ears, and infants may present with unexplained crying.[47]

Cranial nerve or somatic skeletal neuromuscular dysfunction is found in grade III envenomations. Cranial nerve dysfunction can be demonstrated as blurred vision, abnormal eye movements, slurred speech, tongue fasciculations, and hypersalivation. The combination of bulbar neuromuscular dysfunction and increased oral secretions may cause problems with airway maintenance. Abnormal eye movements most often are involuntary, conjugate, slow, and roving. Chaotic multidirectional conjugate saccades resembling opsoclonus and unsustained primary positional nystagmus may also be seen.[43] Many patients with abnormal eye movements prefer keeping their eyes closed. Somatic skeletal neuromuscular dysfunction can cause restlessness, fasciculations, alternating opisthotonos and emprosthotonos, and shaking and jerking of the extremities that can be mistaken for a seizure. The abnormal skeletal muscle activity appears more undulating and writhing, however, than the tonic-clonic movements of generalized seizures. Also, unlike victims with seizures, these victims often remain awake and alert the entire time.

Grade IV envenomation is characterized by both cranial nerve dysfunction and somatic skeletal neuromuscular dysfunction. On close examination, victims with skeletal muscle hyperactivity (at least grade III) usually also have cranial nerve dysfunction, meeting criteria for grade IV. In the most severe cases, stridor and wheezing occur, suggesting foreign body aspiration or reactive airways disease. Hyperthermia up to 40° C (104° F) probably results from excess motor activity. Respiratory failure, pulmonary edema, metabolic acidosis, sterile cerebrospinal fluid pleocytosis, rhabdomyolysis, coagulopathy, pancreatitis, and multisystem organ failure have also been reported in a few severely ill children.[28] After envenomation, symptoms may begin immediately and progress to maximum severity within 5 hours. Infants can reach grade IV in 15 to 30 minutes.[47] The symptoms abate at a rate that depends on the age of the victim and the grade of envenomation. Symptomatic improvement occurs within 9 to 30 hours without antivenom therapy.[36,43,47,161] Pain and paresthesias are exceptions and have been known to persist for days to 2 weeks.

Although adults appear to be envenomed more often, children are more likely to develop severe illness requiring intensive supportive care.[47] A review of 673 patients found that 67.8% of stings occurred in adults older than 20, with 14.9% in children younger than 11. Many more unreported envenomations probably occur in adults, placing the relative incidence for children even lower. Of the patients, 621 (92.3%) had symptoms of either grade I or II envenomations or were asymptomatic and thus required no specific therapy. Younger patients had a higher percentage of the more severe envenomations: 25.9% of children less than 6 years of age had grade III or IV envenomations, or 34% if asymptomatic patients (most likely never stung) are excluded. Only 6.1% of adults had grade III or IV envenomations.[47]

Medically reliable descriptions of the victim's perspective of neurotoxic scorpion envenomation are rare, mostly because of the young age of those most severely affected (Box 47-2).

Other Countries

In Spain from 1974 to 1978, 100 scorpion stings were reported, with most occurring in hot weather and 50% from *Buthus* species.[78] The poison control center in Marseilles, France, reported 976 scorpion stings from 1973 to 1994.[52] Local signs and symptoms predominated; only a few developed systemic toxicity (nausea, vomiting, tachycardia), and none developed neurotoxicity. Recommended treatment consisted of administering analgesics and addressing tetanus immunization status, because hospitalization and antivenom were not necessary.

Scorpions are also found throughout the eastern and tropical regions of Asia. Envenomation by *Buthus martensii* is a

Box 47-2. *Centruroides exilicauda* Envenomation, as Reported by an Intensive Care Specialist

Arriving home in the early evening, I decided to go for a run. My running shoes were in the kitchen area, where I had left them the day before. As usual, I would wear my shoes without socks. As I put my left foot into the shoe, I felt an intense burning pain on the dorsum of my first toe. I pulled my foot out of the shoe and along with it came a $1^1/_2$- to 2-inch, clear-brown scorpion.

Having no idea what to do for a scorpion envenomation, I called the poison control center. I was informed that the systemic toxicity was usually mild for someone my age, and that if the pain was too severe, I should come in and be evaluated. As the minutes went by, I began to salivate and feel perioral paresthesias. As I walked, the paresthesias became more generalized, with a very noticeable paravertebral tingling with each step. After a few more minutes, I decided to call the poison control center to ask for advice. After dialing the number, I was unable to speak clearly because of severe dysarthria and excess salivation. The toe pain seemed to abate as other neurologic symptoms developed.

Since I was unable to talk on the phone, and no neighbors were home to drive me to the hospital, I decided to drive myself. The normal 10-minute drive took 45 minutes. I had coordination difficulties with the gas pedal, clutch, and gear shifting. It was also nighttime, and I could not process the multiple visual inputs of car lights, street lights, and road lines in a way that would allow me to drive more than 5 to 10 miles per hour. I not only had to stop frequently and close my eyes for a few seconds but also had difficulty keeping the car in my driving lane.

After arriving at the emergency department, I was ataxic, dysarthric, and drooling and had difficulty giving the admitting nurse a proper history. I'm certain that I was thought to be either mentally retarded or intoxicated. Examination by the emergency department physician revealed many abnormal cerebellar findings, continued salivation, inability to swallow liquids, continued symptomatic paresthesias, but no objective motor or sensory deficits. There were no physical signs of envenomation [at the sting site], but tapping the toe produced worsening pain. As my story became clearer to the ED physician, antivenom was ordered and administered. Within 20 minutes of finishing the infusion, all neurologic signs and symptoms were gone, except for toe pain.

Personal account courtesy of Dr. Thomas Bajo, Phoenix, AZ.

common medical problem in China.[169] This scorpion is also used in traditional Chinese medicine for its reputed effects of reversing circulatory failure. The black (Asian forest) scorpion *Heterometrus longimanus* is found in Indonesia, Malaysia, and the Philippines.[94]

Australia is home to over 40 named scorpion species and is believed to have many other as yet unnamed species.[97] Nevertheless, these scorpions generally pose little threat to the human population. In a 27-month-long study, only 192 scorpion stings were reported to Australian poison centers. In 95 of these cases, the offending scorpion was collected and identified by an arachnologist. These stings generally caused only local pain lasting several hours, although 11% of the victims had systemic complaints of nausea, headache, and malaise. The majority of victims were managed without seeking additional medical attention.[97] No neurotoxins were found in *Urodacus novaehollandiae*, the only Australian species that has had its venom studied.[160]

CLINICAL MANIFESTATIONS

Cardiovascular

Detailed descriptions of myocardial damage and other cardiovascular manifestations from the scorpions of Israel, India, Trinidad, Tunisia, and Brazil have been reported since the 1960s.[85,92] The overall incidence of heart failure or pulmonary edema is 7% to 32%, with shock reported in 7% to 38% and sudden cardiac death in 7% of victims.

The cardiovascular effects of scorpion envenomation are complex and varied. Stimulation of the sympathetic and parasympathetic branches of the autonomic nervous system results in different clinical presentations that may change with time. Distinct syndromes may dominate the clinical picture in severe scorpion stings. Hypertension or hypotension can occur with or without pulmonary edema, and rhythm disturbances may consist of sinus bradycardia or tachycardia, premature depolarizations, supraventricular tachycardia, atrioventricular block, and ventricular tachycardia.[70,87] A recent Indian study postulated that the cardiovascular effects follow a predictable pattern.[107] Stage I consists of vasoconstriction and hypertension. Stage II is characterized by left ventricular failure manifested as hypotension, with or without pulmonary edema depending on the patient's volume status. Stage III combines both left and right ventricular dysfunction, resulting in cardiogenic shock. A similar progression from a hyperdynamic and hypertensive phase to a hypokinetic, hypotensive phase with left ventricular dysfunction is also reported from Tunisia.[137] Transient parasympathetic effects may occur initially, resulting in bradycardia and hypotension, and are followed by sustained adrenergic hyperactivity.[20]

Sinus tachycardia and hypertension are related to venom-induced catecholamine and angiotensin release.[20,48,89,107,112] Significant hypertension may be seen in up to 77% of patients with systemic envenomation, although a 17.5% to 30% incidence of hypertension is more common.[85] A loud protosystolic gallop, systolic parasternal lift, and transient apical murmur of papillary muscle dysfunction are associated with systemic hypertension.[20,85]

Myocarditis, with ECG changes and biochemical evidence of cardiac injury, is often reported. This myocardial damage is most likely caused by massive catecholamine discharge and sympathetic overstimulation, although direct venom cardiotoxicity has not yet been ruled out.[20,48,71,85,137,158] Many ECG changes consistent with myocardial ischemia and myocarditis, including Q waves, ST-segment elevation or depression, peaked or inverted T waves, U waves, prolonged QTc intervals, and atrioventricular and bundle branch blocks, have been found in persons stung by scorpions.[18,40,85,151] Most ECG abnormalities are transient, lasting only as long as the most severe clinical effects. Prolonged QTc intervals last for 48 to 72 hours,

however, and T-wave inversions have persisted for 4 to 6 weeks.[14] Echocardiography has demonstrated left ventricular systolic dysfunction, which usually resolves by the next day.[112] Concurrent right ventricular dysfunction supports primary global scorpionic myocarditis rather than secondary myocardial ischemia from systemic hypertension.[137]

Elevated serum levels of CK and CK-MB have been found in about one half of persons with systemic envenomation. Only about half of those with elevated CK-MB levels also have ECG changes consistent with myocardial injury.[156] Concurrent CK, CK-MB, and troponin-I elevations have also been reported in a victim with transient bradycardia and second-degree (Mobitz type I) atrioventricular block.[151] Troponin-I was found to be a more specific marker for clinical signs of myocarditis (such as heart failure, cyanosis, shock, abnormal heart sounds, and EKG changes) than total CK, CK-MB, or LDH among scorpion-envenomed children.[125] Even in patients with severe *Tityus serrulatus* envenomation, troponin-I may be normal at admission, typically peaking within 24 to 36 hours of the sting.[46] Histologic examinations of cardiac tissue in fatal human cases have shown a mixed picture of toxic myocarditis and myocytolysis, with interstitial edema and hemorrhage, inflammatory cell infiltrates, focal necrotic foci, and fatty droplet deposition.[48,85]

Respiratory Effects

Respiratory failure after scorpion envenomation has been attributed to direct CNS depression, hypertensive encephalopathy, upper airway obstruction, bronchospasm, impaired surfactant synthesis, and pulmonary edema.[9,128,153,155] Pulmonary edema is the most severe respiratory feature in severe scorpion envenomation, occurring in 7% to 35% of victims and accounting for approximately 25% of scorpion-related deaths.[1,2,141] The etiology and pathogenesis of pulmonary edema from scorpion stings are not clear, and both cardiogenic and noncardiogenic factors have been implicated. Left ventricular systolic dysfunction may cause pulmonary edema through venom-induced myocarditis and acutely increased afterload.[2,3,83,141] Increased pulmonary capillary wedge pressures,[2] abnormal radionuclide scans,[141] and left ventricular dysfunction demonstrated by Doppler ECG studies[1] support a cardiac origin of pulmonary edema.

Noncardiogenic causes for pulmonary edema are less well documented but include shock, primary venom-induced lung injury, oxygen toxicity,[83] the presence of various inflammatory mediators (interleukins, kinins, platelet activating factor),[3] and decreased lung liquid clearance.[44] Histologic and biochemical evidence of increased alveolocapillary membrane permeability has been demonstrated in animal studies and in a fatal human case of *T. serrulatus* envenomation.[7] Rabbit studies with *Tityus discrepans* venom found abundant intravascular microthrombi in lungs with pulmonary edema, and heparin prevented the development of pulmonary edema.[62] Such findings are atypical of noncardiogenic pulmonary edema, or adult respiratory distress syndrome (ARDS), and the researchers therefore suggest that scorpion venom respiratory distress syndrome (SVRDS) be recognized as a distinct clinical entity.

Neurologic Effects

Centruroides exilicauda (*sculpturatus*) stings produce significant neuromuscular effects, manifested by pain and paresthesias in lower grades of envenomation and by cranial nerve and somatic skeletal neuromuscular dysfunction in higher grades. These effects are caused by repetitive firing of neurons from venom-induced incomplete sodium channel inactivation.[47] Other scorpions may cause similar neurologic findings as part of their clinical picture. *Centruroides vittatus* causes neurotoxic symptoms in about 20% of victims.[162] Both local and diffuse paresthesias have been reported with *Leiurus quinquestriatus* stings.[35] *Tityus serrulatus* has caused unilateral facial paresthesias and fasciculations in the facial nerve distribution.[136] Transient ophthalmoplegia has been reported after *Mesobuthus eupeus* sting in Iran.[149]

Neurologic signs are fairly common in patients with severe envenomations from scorpions categorized here as cardiotoxic. For purposes of discussion, scorpions may be divided into neurotoxic and cardiotoxic categories. In reality, all scorpions that produce systemic effects are primarily neurotoxic. However, neurotoxic effects from some species can induce massive release of endogenous catecholamines, causing prominent cardiopulmonary effects. Neurologic signs reported with cardiotoxic scorpions include paresthesias, tremors, shivering, agitation, hyperirritability, apprehension, restlessness, myoclonus, oculogyric crisis (opsoclonus), convulsions, confusion, delirium, hyporeflexia, and coma.[25,38,40,64,86,111,133,140,155] In fatal human cases, a preterminal encephalopathic phase is typical.[106]

Intracranial pathology has been noted in several case reports of scorpion envenomation. *Mesobuthus tamulus* and *Heterometrus swammerdami* in India have been implicated in causing hemorrhagic strokes. In two cases, acute arterial hypertension may have ruptured intracranial blood vessels in the basal ganglia,[72,142] although *Heterometrus* species do not cause systemic toxicity related to a catecholamine surge, as does *M. tamulus*.[18,170] A third case suggests that frontoparietal hemorrhage results from venom-induced vasculitis.[15] Some cases of hemiplegia associated with scorpion stings in India are believed to result from cerebral thrombosis.[15] A 3-year-old boy stung by *Nebo hierochonticus* developed diffuse intracranial hemorrhages with cortical blindness and deafness, most likely related to disseminated intravascular coagulation (DIC).[11] Multiple watershed cerebral infarcts associated with unilateral arm ischemia were reported in a 17-year-old stung by *M. tamulus* and were believed to be caused by venom-induced autonomic stimulation.[165] A 13-year-old Israeli boy stung by *L. quinquestriatus* developed mutism and buccofacial apraxia with bilateral infarcts of the frontal opercular regions,[82] probably related to an episode of cardiogenic shock from ventricular tachycardia.

Many persons with systemic scorpion envenomation exhibit anxiety and agitation, consistent with CNS excitation. Animal experiments have shown that intracerebroventricularly administered *M. tamulus* venom produces effects similar to those caused by yohimbine, a known anxiogenic agent.[32] Although in fatal cases of *L. quinquestriatus* envenomation, CNS manifestations always precede terminal hypotension and cardiac arrest, the venom crosses the blood–brain barrier poorly if at all,[100,106,144] so any encephalopathy would be secondary to peripheral effects. Others suggest that CNS manifestations of scorpion stings are caused by hypertensive encephalopathy or excessive levels of circulating catecholamines[154,155] rather than a direct venom effect. Hypoxia and pain may also contribute to agitation.

Pancreatitis

Scorpionic pancreatitis was reported by Waterman in 1938 from *Tityus trinitatis* stings and was found in 80% of patients

studied by Bartholomew in 1970.[14] Most of these patients had epigastric abdominal pain radiating to the back, starting within 5 hours of the sting and resolving within 24 hours. Some patients with hyperamylasemia (38%) did not complain of abdominal pain, suggesting that the true incidence of pancreatitis may be significantly higher. Scorpion stings are the most common cause of acute pancreatitis in Trinidad.[14] Acute pancreatitis is the most common form of the disease, but edematous, hemorrhagic, and chronic relapsing pancreatitis may also occur.[75] Transient pancreatitis has also been reported from *C. exilicauda*[28] and in 93% of children envenomed by *L. quinquestriatus*.[159] Hyperamylasemia was found in 46% of patients envenomated by *T. discrepans* in Venezuela.[63] The systemic severity of the envenomation or amount of abdominal pain does not appear to correlate with the degree of elevation in the serum amylase level.[14,159] Scorpion venom is known to be a potent secretagogue, stimulating exocrine secretion of the stomach,[166] salivary glands,[47] and pancreas. Enhanced release of proteolytic enzymes, accompanied by spasm of the sphincter of Oddi,[75] is hypothesized to cause acute scorpionic pancreatitis.

Other Gastrointestinal Effects

Nausea, vomiting, gastric distention, abdominal cramping, and occasional diarrhea are reported in victims with severe systemic symptoms.[53,95,112,127] In Tunisia, the onset of vomiting heralded worsening neurotoxicity and marked progression from stage III to stage IV envenomation.[80] Gastric distention associated with agitation and depressed level of consciousness place scorpion sting victims at increased risk for pulmonary aspiration of gastric contents. *T. serrulatus* venom increases the volume, acid output, and pepsin output of gastric juice in rats, probably mediated by release of acetylcholine and histamine. Serum gastrin levels are also elevated.[166] Pig studies with *L. quinquestriatus* venom found that despite an increase in oxygen transport and consumption, oxygen utilization in the GI tract was impaired.[153] Such impairment in oxygen utilization may occur in other tissues as well, contributing to metabolic acidosis in severe envenomations.

Endocrine and Other Humoral Effects

Scorpion envenomation has long been known to cause an autonomic storm with increased release of endogenous catecholamines, contributing to hypertension, tachycardia, and potentially fatal cardiopulmonary dysfunction. Envenomed patients with abnormal ECG tracings excrete elevated levels of free epinephrine, norepinephrine, and vanillylmandelic acid.[91] Elevated circulating catecholamine levels have also been reported.[8] Release of catecholamines may be caused by direct stimulation of postganglionic sympathetic neurons and the adrenal glands. Hypertension may also be caused by activation of the renin-angiotensin endocrine axis. Elevated levels of renin and aldosterone were found in victims stung by *L. quinquestriatus*.[84]

Stings often induce hyperglycemia,[45,63,128] possibly related to suppression of insulin secretion, in contrast to the enhanced secretion by the exocrine pancreas. Murthy and Hase[128] propose that this results in a syndrome of fuel-energy deficits related to inability to utilize existing metabolic substrates, exacerbating the cardiopulmonary insult. Insulin therapy has been found to reverse ECG changes, reduce angiotensin levels and circulating free fatty acids in experimental animals, and reverse hemodynamic changes and pulmonary edema in children stung by scorpions.[128,129] *M. tamulus* venom lowers thyroxine and triiodothyronine levels in experimental myocarditis.[130]

Kinins and other inflammatory mediators may play a role in the cardiovascular toxicity. *T. serrulatus* and *C. exilicauda* venoms potentiate the action of bradykinin by inhibiting angiotensin-converting enzyme (ACE).[117] Animal experiments show reversal of venom-induced cardiovascular effects with aprotinin,[14,68,106] a kallikrein-kinin inhibitor, and icatibant,[68] a bradykinin antagonist, and augmentation of adverse effects with captopril,[13] an ACE inhibitor.

Serum interleukin-6 (IL-6) levels were greatly elevated in eight of 10 Israeli children, gradually decreasing toward normal over 24 hours.[156] High levels of circulating IL-1, IL-3, IL-6, IL-10, and granulocyte-macrophage colony-stimulating factor (GM-CSF) have also been found in a severely envenomed Brazilian patient.[3] The IL-8 level appeared to correlate with evenomation severity caused by *Buthus occitanus* in Egypt.[125]

Genitourinary Effects

Priapism is frequently reported among male patients with systemic envenomation. Priapism results from enhanced parasympathetic tone and is often associated with vomiting and profuse sweating.[15,19] The incidence of priapism ranges from 4% to 10%[5,10,139] to up to 78% to 96%.[95,157] In India, priapism, vomiting, and diaphoresis are considered premonitory diagnostic signs of severe *M. tamulus* envenomation. Priapism is reduced or absent within 6 hours of the sting even in severe cases, and the degree of priapism does not appear to correlate with the severity of envenomation.[18] In Tunisia, however, the incidence of priapism positively correlated with degree of severity.[111]

Urinary retention is found in 33% of victims with systemic envenomation in southern Africa.[30,31,127] This finding, however, is not consistent with increased cholinergic tone, which should produce increased urination, as seen in other series.[10] A dorsal nerve block with 1% lidocaine was successful in treating severe local pain from a *T. serrulatus* sting to the penis.[135]

Hematologic Effects

Scorpion venoms are not generally noted to produce coagulopathies or other significant hematologic effects, although DIC has been reported.[10,116,124] The occasional "defibrination syndrome" is seen more often in children, probably related to a higher relative dose, and can be experimentally reproduced in dogs. Platelet aggregation can be induced by catecholamines alone and may therefore be an indirect effect of scorpion venom. Increased osmotic fragility of red blood cells has been demonstrated in some experimental models.[147]

The only scorpion routinely implicated in dangerous hematologic effects is *Hemiscorpion lepturus,* which can cause severe hemolysis and consequent renal failure.[132,140] Case reports from Saudi Arabia associate severe *Nebo hierochonticus* stings with DIC and intracranial hemorrhage.[10,11] Neither species is from the Buthidae family, which more characteristically causes cardiopulmonary and neurologic toxicities.

Immunologic Effects

Scorpion toxins are antigenic and therefore capable of eliciting an immune response. Positive skin prick and intradermal skin tests and radioallergosorbent assays have been found with the venom from *C. vittatus*[54] and *Androctonus australis hector*[114] in previously envenomed patients. Envenomation by *C. exilicauda* has produced an anaphylactic reaction, with urticaria, wheez-

ing, and facial angioedema but without systemic neurotoxic findings, in an otherwise healthy adult patient previously stung by a scorpion.[171] Fatality from scorpion envenomation in the United States is extremely rare; however, a recently reported death from 2001 appears to have been caused by anaphylaxis to scorpion venom.[37]

Treatment with animal-derived antivenoms places a person at risk for both immediate and delayed immunologic reactions. Since the treatment for hypersensitivity reactions includes epinephrine, and because many persons envenomed by cardiotoxic scorpions have elevated circulating catecholamines, such persons should be less likely to have anaphylactic reactions to antivenom. Brazilian patients stung by *T. serrulatus* were separated into groups with or without adrenergic manifestations, and both groups received antivenom.[8] The group with adrenergic toxicity developed significantly fewer signs and symptoms of early anaphylaxis (8% compared with 42.9%).

Miscellaneous Systemic Effects

Leukocytosis with white blood cell counts as high as 44,000/mm^3 is common in persons with severe scorpion envenomation.[38,40,107] Mild to moderate hypokalemia was found in 13 of 17 patients with severe *Tityus* envenomations in Brazil[38] and in eight patients with *M. tamulus* stings in India,[107] although elevated potassium and lowered sodium levels were found previously.[172] The same venom-induced metabolic changes that inhibit insulin release favor glycogenolysis and lipolysis, leading to elevated circulating levels of free fatty acids.[128,130] Scorpion envenomation appears to increase apoptosis. Severely envenomed patients in Egypt showed dysregulation of multiple markers of apoptosis, which might be used to predict clinical outcome.[124]

DIFFERENTIAL DIAGNOSIS

Bites or stings from other arthropods should be considered in the differential diagnosis of scorpion envenomation. Pain at the site of *Centruroides exilicauda* sting may be similar to that from a black widow spider (*Latrodectus* species). However, severely ill victims of scorpion envenomation appear unable to lie still, whereas those with black widow spider bites can maintain a position for short periods before moving again to find a comfortable position. Black widow envenomation may produce hypertension, tachycardia, sweating, and other signs of adrenergic excess, but it does not produce the abnormal eye movements, fasciculations, or paresthesias, or induce a positive tap test as found with scorpion stings. Widow spider bites frequently produce a characteristic halo lesion at the site, whereas no lesion is usually visible after a *C. exilicauda* sting. Many other arthropods can produce a small puncture wound accompanied by local tissue inflammation. This may be difficult to differentiate clinically from stings by scorpion species in the absence of cardiovascular or neurologic toxicity or without tentative visual identification of the arthropod involved.

The tachycardia, respiratory distress, excessive secretions, and occasional wheezing that occur from *C. exilicauda* sting may be mistaken for asthma, airway obstruction with a foreign body, or poisoning with a cholinergic agent, such as an organophosphate insecticide. In the absence of the history of a scorpion sting, other disorders to be considered include CNS infection, tetanus, dystonic reaction, seizure, and intoxication with an anticholinergic, a sympathomimetic, phencyclidine, nicotine, or strychnine. Autonomic storm from a cardiotoxic scorpion sting may be confused with pheochromocytoma or a monoamine oxidase inhibitor–tyramine reaction. A victim of severe scorpion envenomation presenting late in the course may appear to have cardiac failure or sepsis.

Toxicity from an illegal sympathomimetic is sometimes mistaken for envenomation by *C. exilicauda*. Young children from endemic areas presenting with unusual neurologic symptoms, such as agitation, choreiform or repetitive motion of the trunk and extremities, and abnormal eye movements, may be assumed to have been envenomed even without history of a sting or scorpion.[131] Occasionally, the caregivers are aware of this potential for misdiagnosis and claim that their child was stung when they know or suspect that the child ingested methamphetamine. A case series of 18 inadvertent methamphetamine poisonings among children in central Arizona included three victims initially misdiagnosed with a scorpion sting and inappropriately treated with antivenom; one patient had an anaphylactic reaction.[108]

TREATMENT

Ismail and colleagues[106] summarized the current understanding of treating scorpion stings when noting, "It is strange that despite the long experience with scorpion envenomation, most of the treatment protocols advocated are based on isolated clinical observations, are sometimes controversial, and not instituted on rigid or strictly controlled animal or clinical studies. . . . Even in serotherapy, there are no quantitative studies regarding antivenom dosage, routes of administration, time-effectiveness relationship and titre of the antivenom used."

Some treatment recommendations can be eliminated. A so-called lytic cocktail was once considered essential in treating *Mesobuthus tamulus* stings in India.[49] This mixture of pethidine (meperidine), chlorpromazine, and promethazine (equivalent to the DPT cocktail for pediatric sedation, which has fallen into disfavor in the United States) was claimed to induce "artificial hibernation" and improve outcome. Morbidity and mortality statistics, however, have not shown a difference between cohorts historically treated with the lytic cocktail and those not treated. Furthermore, any effect of such treatment would be sedation, which would increase the risk of airway difficulties. Some traditional herbal therapies involve rubbing the area near the sting site, which doubles the chance of developing a severe envenomation.[30,31] Purported herbal therapies for scorpion stings have not been adequately tested and are probably of no benefit.[96] Other therapies no longer recommended include electric shock, barium, iodine, physostigmine, and snake or spider antivenom.[148]

Although much controversy surrounds the proper treatment of scorpion envenomation, some consensus exists. Most victims, even those stung by potentially dangerous scorpions, demonstrate only local signs and symptoms and require only symptomatic outpatient treatment. It is prudent to observe such persons for several hours after the sting to ensure that progression to severe envenomation does not occur. Experience with *C. exilicauda* envenomation in Arizona shows that progression of envenomation grade occurs rapidly, with a mean time of 14 minutes and a median time of less than 1 minute.[119]

For localized pain, many authorities recommend local anesthesia with lidocaine, bupivacaine, or dehydroemetine by infiltration or nerve block, such as a digital block for fingers or toes.[25,30,31,40,98,135] In southern Africa, Bergman[30,31] has reported successful treatment of poorly localized pain radiating up an extremity with an intracutaneous sterile water injection (ISWI), usually also with a local anesthetic infiltrated at the sting site. ISWI is performed by injecting small amounts (about 0.1 mL) of sterile water intradermally into points of maximal pain, producing up to 10 wheals. Pain relief is said to occur within 1 to 5 minutes, but pain may return in 4 to 12 hours and can be persistent. ISWI therapy cannot be generally recommended because it has not been tested in prospective or controlled trials or with scorpion stings elsewhere in the world. Scorpion stings are traumatic puncture wounds, and victims should be given appropriate wound care and tetanus prophylaxis if indicated.

Victims with significant systemic scorpion envenomations should receive supportive and symptomatic care in a monitored hospital setting.[38,65,71,86,90] Many require admission to an ICU, although treatment should begin in the emergency department or outlying facility if available. Airway control must be addressed and continuous cardiovascular and respiratory monitoring instituted. Fluid resuscitation may be indicated secondary to fluid losses from vomiting, sweating, and increased insensible losses from hyperthermia. Both hyperthermia and hypothermia have been reported from scorpion stings, and both appear to worsen toxicity.[105] Hyperthermia usually resolves with standard acetaminophen doses, and hypothermia resolves with warm blankets.

Pharmacologic Therapy

Many drugs have been recommended in the treatment of scorpion envenomations, but few have been rigorously tested. Recommendations for atropine vary. Many victims exhibit signs of excess cholinergic tone, such as bradycardia, vomiting, sweating, and hypersalivation. If venom induces prominent adrenergic effects, the rescuer should not administer atropine.[21,71,86] Parasympathetic venom effects are usually transient and not life-threatening, although atropine may be indicated for severe bradycardia. Also, in subsequent phases with more prominent adrenergic toxicity, atropine could worsen tachycardia and hypertension, leading to more severe cardiovascular effects. Atropine may be safe, however, for cholinergic effects from scorpions that do not cause prominent cardiotoxicity. Published cases from southern Africa[30] and Arizona[163] suggest that atropine can reverse hypersalivation that interferes with airway control, which may obviate the need for intubation and decrease the risk of aspiration, although caution is still advised.[127] Protocols for the use of atropine and its optimal dosage have not been prospectively determined.

Treatment with corticosteroids has been regularly recommended and is still common in many countries; however, a prospective, randomized, placebo-controlled human study of anti-inflammatory corticosteroids showed no benefit.[5] In Tunisia, 600 consecutive patients received either 50 mg/kg of hydrocortisone hemisuccinate or a placebo. No differences between the two groups were found in clinical severity (baseline and 4 hours after treatment), mortality, need for artificial ventilation, or duration of hospitalization.[5] Glucocorticoids and antihistamines are not recommended unless administered as treatment for allergic manifestations (e.g., anaphylaxis to antivenom).[31,127]

Vasodilator therapy has received much attention. Because excessive adrenergic tone appears to cause the most significant morbidity, vasodilators should block or reverse severe cardiopulmonary effects from scorpion envenomation. Prazosin is a selective α_1-adrenergic blocker, which is also thought to reverse the inhibition of insulin.[23,24] In India, an initial dose of 0.5 mg by mouth for adults and 0.25 mg for children is given to relieve hypertension and pulmonary edema. Repeat doses are given in 4 hours, then every 6 hours as needed for up to 24 hours. Sodium nitroprusside is used in cases of life-threatening pulmonary edema.[25] Nifedipine, 5 to 10 mg "sublingually" by puncturing and swallowing the gelatin capsule, is also used in India along with the initial dose of prazosin. When this protocol was compared with "conventional treatment" (digoxin, furosemide, hydrocortisone, antihistamines, and atropine), the cohort treated with prazosin had significantly reduced morbidity and shortened recovery time.[24,88] In Israel, hypertension from scorpion sting unresponsive to analgesics and sedatives has been treated with IV hydralazine or sublingual nifedipine, which are believed to reverse hypertensive encephalopathy.[154] A potential benefit of vasodilators over antivenom is the rapidity of onset.[88] Captopril has also been used to treat hypertension[107] but theoretically could worsen pulmonary edema.[13,25,100]

Insulin infusion has been used in India.[128,129] Because scorpion venoms inhibit insulin secretion, this treatment may help reverse the consequent metabolic derangements. "Standard therapy" to which insulin infusions were added consisted of furosemide for elevated central venous pressures or pulmonary edema, crystalloid for low central venous pressures, and hydrocortisone and dopamine for hypotension. Insulin was given in a glucose solution with 0.3 units of regular insulin per gram of glucose, at a rate of 0.1 g glucose per kilogram bodyweight per hour. Six patients treated with insulin showed improvement in pulmonary edema and hemodynamics, although furosemide and hydrocortisone appeared to offer little benefit.[128]

Intravenous lidocaine increased the LD_{50} (dose at which 50% of the group died) of *Centruroides limpidus limpidus* venom in a mouse model, presumably through antagonism of the sodium channel,[138] but it has not been evaluated in humans. Dobutamine infusions of 7 to 20 µg/kg/min improved cardiac function in a series of seriously envenomed Tunisian patients.[67] Digoxin, β-adrenergic blockers, dantrolene, aminophylline, quinine, and aspirin have also been suggested as potential therapeutic adjuvants in scorpion envenomation.[25,93]

Antivenom

Treatment recommendations seriously diverge regarding antivenom. Most medical researchers believe that antivenom plays a crucial role in the treatment of seriously envenomed patients.[2,36,40,47,56,70,98,101,127] Proponents believe that (1) antivenom is the only specific therapy available against the primary physiologic insult, and (2) it greatly improves outcome. Any previous disappointing experience with antivenom, they argue, probably results from inadequate dosing.[99,100] Researchers from Israel and India do not recommend antivenom.[19,86,157] Opponents believe that (1) morbidity and mortality are not caused by the venom but by autopharmacologic agent release, which should not be reversed by antivenom therapy, (2) pharmacokinetic data do not support a role for antivenom, (3) antivenom has not improved outcome in Israeli studies, and (4) antivenom is often unavailable and would be prohibitively expensive in India, so other options must be

chosen. Some suggest that commercial production of scorpion antivenom is needed in India.[170] Treatment without antivenom consists of managing serious cardiopulmonary and neurologic effects with pharmacologic agents and supportive care. Even when antivenom is administered by its proponents, adjunctive therapies to treat cardiac failure, pulmonary edema, and other physiologic treatments are also used.

A major issue in the debate regarding the utility of antivenom is the pharmacokinetics of scorpion venom. Detectable circulating levels of venom support the use of antivenom to neutralize the toxins. *Tityus serrulatus* venom injected subcutaneously in rodents rapidly distributed to various tissues, with peak serum levels in 30 to 60 minutes.[144,150] After 2 hours, the venom decreased rapidly and was no longer detectable after 8 hours.[144] IV serotherapy therefore should be initiated as soon as possible, because it would become less effective when administered many hours after envenomation.[144]

Ismail and coworkers[100,103] reported that scorpion venoms in animal experiments were rapidly absorbed and distributed to tissues but had more prolonged elimination phases. Scorpion venom has a half-life of 4.2 to 24 hours.[100] The authors concluded that although antivenom would theoretically be most efficacious if given immediately after envenomation, it is still indicated after a delay of several hours or more.[112,150] Interestingly, Gueron and colleagues[85,86] reviewed these data and concluded instead that serotherapy would be ineffective and recommended against it. They suggested that the most severe cardiopulmonary effects would be present early and would not be reversed by antivenom given later. The clinical effects of scorpion envenomation are related to tissue concentrations of *Androctonus amoreuxi* venom in experimental animals,[102] although the slower distribution of antivenom to tissues suggests that it may be less effective when given after a significant delay. A series of 56 victims stung by *T. serrulatus* in Brazil correlated clinical severity to plasma venom concentrations.[55] This correlation between venom levels and clinical severity of envenomation has been seen in other studies.[63,76] IV antivenom lowers circulating levels of venom and presumably the clinical severity of envenomation; however, the effectiveness of antivenom on venom bound to other tissues has not been well defined. The role of serotherapy depends on the time to administration, becoming less effective with increasing delay.[110,122] Because scorpion venom acts indirectly through the release of autopharmacologic substances, treatment with specific blockers may be more effective than antivenom in persons with delayed presentation.[102]

In 1999, a large (825-patient), randomized, placebo-controlled Tunisian clinical trial of scorpion antivenom in humans found no significant benefit to giving antivenom in terms of progression to systemic illness.[3] However, the majority of patients in this study had mild envenomations, possibly obscuring an effect that could be seen only in more serious cases. In 2003, one canine study showed that whereas simultaneous administration of antivenom and venom prevents clinical manifestations of envenomation, delayed administration of antivenom does not alter the clinical course.[4] A recent clinical case series from India is consistent with these findings: four children with severe envenomations by *Mesobuthus tamulus* who received scorpion antivenom within 1.5 hours of the sting were not observed to have any clinical improvement.[26] Another 2003 canine study found that the administration of antivenom 20 minutes after venom improves cardiac output, as does the administration of IV fluid; combination therapy worked better than either single treatment alone.[164] A treatment protocol utilizing high doses of IV polyvalent antivenom (i.e., doses greater than the in vitro neutralizing dose) is used in Egypt and has reduced mortality from 4.6% to 8% down to less than 0.05%.[101]

The entire literature on scorpion antivenom appears to be a muddle, with many contradictory findings. This confusion may stem from differences between benchwork and clinical studies, differences in study design, venom and antivenom dosing, patient susceptibility, and many other factors. Based on this author's own clinical experience with stings from *Centruroides* scorpions in the United States, the use of IV antivenom is favorable for severe scorpion envenomation in cases where it is available.

Antivenom research is ongoing and may produce improved products in the future. Maximal neutralizing capacity of antivenom is obtained when soluble venom, rather than telson macerates and purified toxins, is used as the antigen.[39] Antibodies have been produced against nontoxic analogs of some scorpion neurotoxins,[59] nontoxic proteins found in scorpion venom,[42,126] and neurotoxin amino acid sequences that are highly conserved among many species.[60] Fab antibody fragments have also been produced against scorpion venoms.[71,115] These fragments induce a lower incidence of hypersensitivity reactions[71] and possess pharmacokinetic characteristics that make them more suitable than whole immunoglobulin G (IgG) antibodies.[104]

Centruroides exilicauda Envenomation

Until very recently, a goat-derived scorpion antivenom was available, produced by the Antivenom Production Laboratory at Arizona State University in Phoenix.[36,47,74] This antivenom had been produced since 1965 by lyophilizing micron-filtered serum obtained from goats hypersensitized to *C. exilicauda* venom. Antivenom was produced in batches every 2 to 3 years and supplied without charge to regional hospitals. Because of the limited geographic area of its use, the antivenom had not been submitted for U.S. Food and Drug Administration approval, but it was used within the state by special action of the Arizona Board of Pharmacy. Fresh batches have not been made in the last several years, and the most recently released batch expired in 2004.[118]

No widely recognized standard of care exists in the treatment of *C. exilicauda* envenomation in the United States. Because antivenom is no longer available, treatment must of necessity be symptomatic and supportive, with the most severely envenomed patients cared for in an ICU setting. When antivenom was available, the clinical toxicology group based in Phoenix, Arizona (where this author trained), favored its use in treating severe *C. exilicauda* envenomation. Antivenom was routinely recommended and used for patients with respiratory compromise, as often seen in grade III or IV envenomation, and typically resulted in rapid resolution of symptoms, allowing the patient to be discharged home rather than requiring admission to the hospital, usually to an ICU setting. However, the presence of grade III or IV symptoms was not an absolute indication for antivenom, as some victims (usually older children or adults) who met criteria for higher grades of envenomation remained alert with no respiratory distress. In several cases, and although it was not part of standard protocol, atropine was the initial treatment given to some very young victims with hyper-

salivation significant enough to impact respiratory status. No clinically significant adverse cardiovascular effects were noted in one small case series and atropine appeared to improve airway maintenance.[163]

Historically, the recommended therapy for the neuromuscular hyperactivity seen in severe envenomations was large doses of barbiturates.[161] Sedation with barbiturates, benzodiazepines, or other drugs in a person already with tenuous airway control increases the risk for aspiration and respiratory failure. Therefore, these drugs should not be given unless close airway monitoring is available and the patient can be expeditiously intubated and mechanically ventilated if necessary.

A case series of patients treated with continuous IV infusions of midazolam was reported by physicians from Tucson, Arizona.[77] Among 33 patients treated in the ICU, the mean infusion time was 9.5 hours and the mean ICU stay was 15 hours, with 85% of patients discharged directly to their homes. Four patients with hypoxia responded to supplemental oxygen. The researchers concluded that continuous midazolam infusions were safe and effective in the ICU setting. The neuromuscular hyperactivity of *C. exilicauda* envenomations, however, is not a centrally mediated phenomenon. CNS depressants may decrease motor agitation but only indirectly, whereas antivenom directly treats the underlying cause. Regardless of the method, excessive motor activity should be controlled to avoid potential complications of hyperthermia, rhabdomyolysis, and metabolic acidosis. Antivenom typically works quickly, obviating the need for additional pharmacologic sedation to control hyperactivity.

The primary concern about using antivenom is hypersensitivity reactions. Both immediate (type I, or anaphylaxis) and delayed (type III, or serum sickness) hypersensitivity reactions may occur. In 1992, Bond[36] reported no anaphylactic reactions to antivenom in 12 patients; 58% developed a delayed rash or serum sickness that resolved with oral antihistamines and corticosteroids. In 1994, Gateau and coworkers[74] reported 145 cases of severe *C. exilicauda* envenomations treated with antivenom, with immediate hypersensitivity reactions in 8% generally characterized as mild; no follow-up for incidence of serum sickness was conducted. In 1999, LoVecchio and colleagues[120] reported a prospective study of 116 patients treated with antivenom. Three patients (2.6%) who developed a rash during the antivenom infusion were treated with hydroxyzine, methylprednisolone, and epinephrine, and the infusions were completed at slower rates. An asthmatic patient who developed wheezing responded to treatment with an epinephrine infusion and an inhaled β-agonist and was admitted for observation. Nine other patients were admitted for oversedation from medications received before antivenom, to rule out aspiration pneumonitis, or for social reasons. All were discharged within 24 hours. During a 3-week follow-up period, 60 of the 99 patients (61%) developed serum sickness that responded to oral corticosteroids and antihistamines; mean duration of symptoms was 2.8 days with medication.

Intravenous antivenom results in rapid reversal of neurologic, respiratory, and cardiovascular toxicity in persons envenomed by *C. exilicauda*. Symptoms completely resolved within 1 to 3 hours in one study[36]; 71% of patients in another study had resolution within 30 minutes.[74] The mean time to abatement of symptoms in a series of 86 patients was 31 minutes.[119] Bond[36] summarized the potential benefits of antivenom administration as decreased time to resolution of symptoms, cost savings in the emergency department (versus about $1000/day in ICU charges), and the risks of sedation, paralysis, and intubation. Bond believes the data support the safe use of IV antivenom for high-grade *C. exilicauda* neurotoxicity, and that serious adverse effects are both uncommon and treatable. Other reasonable and informed physicians have concluded that because fatalities should not occur with appropriate supportive care, patients should not be subjected to any risk of antivenom anaphylaxis. However, this debate is moot, as antivenom is not currently available in the United States.

Recommendations and Procedure

Because of the possibility that alternate domestic antivenom production sources may be developed or that foreign (e.g., Mexican) antivenoms active against *Centruroides* scorpions could become available in the future, a recommended procedure for antivenom administration is given here. Any of the following relative contraindications (in decreasing order of importance) should suggest withholding animal-derived antivenom in favor of symptomatic and supportive care only: (1) prior administration of antivenom derived from the same species, (2) current β-adrenergic blocker use, (3) history of asthma or atopy, (4) current ACE inhibitor use, and (5) history of allergy to the animal species from which the antivenom is derived, allergy to that animal's milk, or prior extensive exposure to the animal, especially its blood.

The patient is placed on continuous ECG and pulse oximetry monitors. IV access is obtained, preferably with at least two IV lines. Equipment and medications for treating anaphylaxis and respiratory arrest are available. Specifically, an epinephrine drip is prepared by adding 1 mg epinephrine to 250 mL of 5% dextrose in water (D_5W) or normal saline (NS) on a primed IV line with a pump; IV methylprednisolone and hydroxyzine or diphenhydramine are also made available in the room. Lyophilized antivenom is reconstituted with as much sterile NS or D_5W as can fill the vial, with gentle rocking to avoid foaming. The antivenom is then withdrawn from the vial and dissolved in 50 mL of crystalloid and placed on an IV pump. A skin test is performed. Up to 0.02 mL of a 1:10 dilution of reconstituted antivenom is injected intradermally with a 27- or 30-gauge needle, the site marked with a pen, and the patient observed for at least 10 minutes for development of a wheal, rash, wheezing, or hemodynamic signs of anaphylaxis, any of which constitute a positive skin test. The patient with a reaction is treated appropriately with the medications made available (as just described). For *C. exilicauda* envenomation, a positive skin test contraindicates further antivenom therapy, and the patient is admitted for supportive care. Skin testing, however, does not always predict adverse reactions. One study showed 96% sensitivity for mild immediate hypersensitivity reactions but only 68% specificity.[74]

The antivenom is initially administered at a very slow rate (5 mL/hr), which is doubled every 2 to 3 minutes as long as the patient tolerates the infusion (i.e., develops no signs of anaphylaxis), up to 150 mL/hr. Infusion of one vial by this method takes approximately 30 minutes, and most patients require only one vial of antivenom. The patient is observed for at least 1 hour after the initial antivenom infusion is complete before proceeding with additional vials, because most symptoms resolve in this period. If the symptoms have resolved or regressed to a grade I or grade II envenomation and no complications (e.g., aspiration) are suspected, the patient is discharged. The ideal amount of time to observe patients after antivenom therapy has

not been formally evaluated, but because resolution of symptoms is usually rapid,[36,74,119] 1 hour appears adequate. Symptoms from *C. exilicauda* or other scorpion envenomations have not been reported to rebound after treatment with antivenom.

Because many patients treated with antivenom develop a delayed hypersensitivity reaction, the patient and caregivers are informed about signs and symptoms of serum sickness before discharge. An urticarial rash developing within a few days to weeks is the most common sign, although malaise, myalgias, and arthralgias also may occur. More serious problems are rare. The patient may be discharged with prescriptions for antihistamines (hydroxyzine or diphenhydramine) and a tapering dose of corticosteroids to be started if serum sickness develops. Filling these prescriptions and initiating therapy immediately as prophylaxis against serum sickness is not recommended. Any persons treated with antivenom should be warned that they are now very likely to be allergic to serum products from the animal species used. Repeated use of antivenom for subsequent envenomations is relatively contraindicated but can be undertaken with extreme caution if necessary.

Consultation with toxicologists experienced in treating *C. exilicauda* envenomations can be obtained by calling the Banner Poison Control Center in Phoenix at 602-495-4884 or the Arizona Poison and Drug Information Center in Tucson at 520-626-6016.

PREVENTION

Many scorpion stings result from human practices that place persons at risk. Residences with small cracks and crevices offer many hiding places, increasing the risk of human–scorpion interactions. In several countries, playing or working outdoors with inadequate protective clothing, especially in the early evening during warm months, is associated with most scorpion stings. In scorpion-infested areas, clothing, shoes, packages, and camping gear should be shaken out and checked for scorpions. Footwear is recommended. Unnecessary ground cover and debris should be removed to reduce potential nesting places.

Certain insecticides, including organophosphates, pyrethrins, and several chlorinated hydrocarbons, are known to kill scorpions. Home spraying is often ineffective because the insecticide does not come in contact with the scorpion. Spraying insecticides around the home can work indirectly by killing other insects in the area and reducing the scorpions' food supply. A village-wide scorpion eradication program with pyrethroid insecticides in the state of Morelos, Mexico, reduced the incidence of scorpion stings by 17%.[143]

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Bites by Venomous Reptiles in the Americas

48

Robert L. Norris and Sean P. Bush

The Western Hemisphere is unique in that it is home not only to venomous snakes but also to the world's only known venomous lizards. Fortunately, bites by venomous reptiles in North America are relatively uncommon, although precise statistics are not available. The only attempt to evaluate the overall incidence of venomous snakebite in the United States was done in the late 1960s by Dr. Henry Parrish. He estimated that, on an annual basis, there were approximately 7000 bites by venomous snakes, of which approximately 15 ended in death.[140] It is unclear just how reliable these numbers are, and the incidence of venomous snakebite may have changed significantly since Parrish's study. Given that snakebite is not a reportable "disease," no mechanism exists for obtaining reliable statistics.

Data from the American Association of Poison Control Centers for 2003 included 2911 bites by indigenous venomous snakes in the United States.[182] However, the total is clearly greater, because a significant number of such bites are never reported to poison control centers. The incidence of snakebite in Canada is lower than that in the United States because fewer snake species and fewer snake numbers are found farther north in the continent. In Mexico, snakebite takes on increasing medical importance because Mexico has more venomous snake species than any other nation in the New World.[25] An estimated 3000 snakebites occur in Mexico each year (personal communication, Juan Silanes, June 2005). As many as 150 deaths may be caused by snakebite in Mexico each year.[64] Chippaux estimated that 5000 deaths in Central and South America each year are related to venomous snakebite.[29] For a global perspective, it is estimated that as many as 2.5 million venomous snakebites occur internationally per year, and estimates of deaths each year from snakebite are as high as 125,000 worldwide.[29] Again, however, the reliability of these numbers is hard to determine.

Establishing credible estimates of the incidence of venomous lizard bites is even more difficult than for snakes. The vast

not been formally evaluated, but because resolution of symptoms is usually rapid,[36,74,119] 1 hour appears adequate. Symptoms from *C. exilicauda* or other scorpion envenomations have not been reported to rebound after treatment with antivenom.

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Establishing credible estimates of the incidence of venomous lizard bites is even more difficult than for snakes. The vast

majority of victims are bitten while intentionally interacting with the lizard, and because these creatures are legally protected, many bites are probably never reported.

Many physicians called on to render acute care to snakebite victims have had limited experience with the potentially complex syndromes of snake envenomation. In addition, it is difficult to obtain funding in North America to develop antivenoms, and to evaluate prehospital care techniques and hospital management principles, because the targeted population is composed of only a few thousand individuals.

Snake venoms are highly complex mixtures of enzymes, low-molecular-weight polypeptides, glycoproteins, minerals, and other, unidentified substances. Venoms vary among species, within a single species depending on its geographic distribution, and even within an individual snake depending on factors such as age, diet, and health.[155] The effects of a particular venom can be different depending on the species of prey or research animal that has been envenomed. Such variables make it difficult to conduct meaningful research into snakebite management techniques.

Tables 48-1 and 48-2 list the species of venomous reptiles found in North, Central, and South America.[26] The medically important New World venomous snakes all fall into the families Viperidae (subfamily Crotalinae—the crotalines or pit vipers) and Elapidae (elapids—the coral snakes and sea snakes). Although there are reports of human envenoming by a handful of species of Colubridae, a family of snakes traditionally regarded as harmless, the cases from the New World have largely been relatively minor (limited to pain, soft tissue swelling, and bruising) and non–life-threatening compared with the bites of some species of this family in the Eastern Hemisphere, which have resulted in fatalities (see Chapter 49). There

TABLE 48-1. Venomous Reptiles of North America

GENUS	SPECIES	COMMON NAME	CANADA	U.S.	MEXICO
Agkistrodon		Cantils, copperheads, water moccasins			
	bilineatus	Common cantil	—	—	*
	contortrix	Copperhead	—	*	*
	piscivorus	Cottonmouth water moccasin	—	*	—
	taylori	Taylor's cantil	—	—	*
Atropoides		Jumping pit vipers			
	mexicanus	Central American jumping pit viper	—	—	*
	nummifer	Mexican jumping pit viper	—	—	*
	occiduus	Guatemalan jumping pit viper	—	—	*
	olmec	Tuxtlan jumping pit viper	—	—	*
Bothriechis		Palm pit vipers			
	aurifer	Yellow-blotched palm pit viper	—	—	*
	bicolor	Guatemalan palm pit viper	—	—	*
	rowleyi	Mexican palm pit viper	—	—	*
	schlegelii	Schlegel's viper	—	—	*
Bothrops		Lanceheads			
	asper	Terciopelo	—	—	*
Cerrophidion		Montane pit vipers			
	barbouri	Barbour's montane pit viper	—	—	*
	godmani	Godman's montane pit viper	—	—	*
	petlalcalensis	Petlalcala montane pit viper	—	—	*
	tzotzilorum	Tzotzil montane pit viper	—	—	*
Crotalus		Rattlesnakes			
	adamanteus	Eastern diamondback rattlesnake	—	*	—
	aquilus	Querétaro dusky rattlesnake	—	—	*
	atrox	Western diamondback rattlesnake	—	*	*
	basiliscus	Mexican west coast rattlesnake	—	—	*
	catalinensis	Santa Catalina rattlesnake	—	—	*
	cerastes	Sidewinder	—	*	*
	enyo	Baja California rattlesnake	—	—	*
	horridus	Timber/canebrake rattlesnake	*	*	*
	intermedius	Mexican small-headed rattlesnake	—	—	*
	lannomi	Autlán rattlesnake	—	—	*
	lepidus	Rock rattlesnake	—	*	*
	mitchelli	Speckled rattlesnake	—	*	*
	molossus	Black-tailed rattlesnake	—	*	*
	oreganus	Western rattlesnake			
	oreganus abyssus	Grand Canyon rattlesnake (formerly *C. viridis abyssus*)	—	*	—
	oreganus caliginus	South Coronado Island rattlesnake (formerly *C. viridis caliginus*)	—	—	*
	oreganus cerberus	Arizona black rattlesnake (formerly *C. viridis cerberus*)	—	*	—
	oreganus concolor	Midget faded rattlesnake (formerly *C. viridis hellea*)	—	*	—

TABLE 48-1. Venomous Reptiles of North America—cont'd

GENUS	SPECIES	COMMON NAME	CANADA	U.S.	MEXICO
	oreganus helleri	Southern Pacitic rattlesnake (formerly *C. viridis helleri*)	—	*	*
	oreganus lutosus	Great Basin rattlesnake (formerly *C. viridis lutosus*)	—	*	—
	oreganus oreganus	Northern Pacitic rattlesnake (formerly *C. viridis oreganus*)	—	*	—
	polystictus	Mexican lance-headed rattlesnake	—	—	*
	pricei	Twin-spotted rattlesnake	—	*	*
	pusillus	Tancitaran dusky rattlesnake	—	—	*
	ravus	Mexican pygmy rattlesnake	—	—	*
	ruber	Red diamond rattlesnake	—	*	*
	scutulatus	Mojave rattlesnake	—	*	*
	simus	Middle American rattlesnake	—	—	*
	stejnegeri	Long-tailed rattlesnake	—	—	*
	tigris	Tiger rattlesnake	—	*	*
	tortugensis	Tortuga Island diamond rattlesnake	—	—	*
	totonacus	Totonacan rattlesnake	—	—	*
	transversus	Cross-banded mountain rattlesnake	—	—	*
	triseriatus	Mexican dusky rattlesnake	—	—	*
	viridis	Prairie rattlesnake (formerly *C. viridis abyssus* [Grand Canyon rattlesnake], *C. viridis caliginis* [Coronado Island rattlesnake], *C. viridis cerberus* [Arizona black rattlesnake], *C. viridis concolor* [midget faded rattlesnake], *C. viridis lutosus* [Great Basin rattlesnake], *C. viridis nuntius* [Hopi rattlesnake], *C. viridis viridis* [prairie rattlesnake])	*	*	*
	willardi	Ridge-nosed rattlesnake	—	*	*
Heloderma		Venomous lizards			
	suspectum	Gila monster	—	*	*
	horridum	Beaded lizard	—	—	*
Micruroides		Coral snakes			
	euryxanthus	Sonoran (Arizona) coral snake	—	*	*
Micrurus		Coral snakes			
	bernadi	Blotched coral snake	—	—	*
	bogerti	Bogert's coral snake	—	—	*
	browni	Brown's coral snake	—	—	*
	diastema	Variable coral snake	—	—	*
	distans	West Mexican coral snake	—	—	*
	elegans	Elegant coral snake	—	—	*
	ephippifer	Oaxacan coral snake	—	—	*
	fulvius (formerly *M. fulvius fulvius*)	Eastern coral snake, Harlequin coral snake	—	*	—
	laticollaris	Balsan coral snake	—	—	*
	latifasciatus	Long-ringed coral snake	—	—	*
	limbatus	Tuxtlan coral snake	—	—	*
	nebularius	Ixtlán coral snake	—	—	*
	nigrocinctus	Central American coral snake	—	—	*
	pachecogili	Azpotitlán coral snake	—	—	*
	proximans	Nayarit coral snake	—	—	*
	tener (formerly *M. fulvius tenere*)	Texas coral snake	—	*	*
Ophryacus		Mexican horned pit vipers			
	melanurus	Black-tailed horned pit viper	—	—	*
	undulates	Mexican horned pit viper	—	—	*
Pelamis	*platurus*	Yellow bellied sea snake (see Chapter 74 for discussion of sea snake bites)	—	*	*
Porthidium		Hognose pit vipers			
	dunni	Dunn's hognose pit viper	—	—	*
	hespere	Colima hognose pit viper	—	—	*
	nasutum	Rainforest hognose pit viper	—	—	*
	yucatanicum	Yucatán hognose pit viper	—	—	*
Sistrurus		Rattlesnakes			
	catenatus	Massasauga	*	*	*
	miliarius	Pygmy rattlesnake	—	*	—

Adapted from Campbell JA, Lamar WM: The Venomous Reptiles of the Western Hemisphere. Ithaca, NY, Comstock, 2004.
*, present.

TABLE 48-2. Venomous Reptiles of Central and South America and the Caribbean

GENUS	SPECIES	COMMON NAME	COUNTRIES FOUND IN
Agkistrodon			
	bilineatus	Common cantil	Ga, Be, Ho, ES, Ni, CR
Atropoides		Jumping pit vipers	
	mexicanus	Central American jumping pit viper	Ga, Be, Ho, Ni, CR, Pn
	occiduus	Guatemalan jumping pit viper	Ga, Ho, ES
	olmec	Tuxtlan jumping pit viper	Ga
	picadoi	Picado's jumping pit viper	CR, Pn
Bothriechis		Palm pit vipers	
	aurifer	Yellow-blotched palm pit viper	Ga
	bicolor	Guatemalan palm pit viper	Ga
	lateralis	Side-striped palm pit viper	CR, Pn
	marchi	Honduran palm pit viper	Ho, Ni
	nigroviridis	Black-speckled palm pit viper	CR, Pn
	schlegelii	Eyelash palm pit viper	Ga, Be, Ho, Ni, CR, Pn, Cl, Ve, Ec, Pe
	supraciliaris	Blotched palm pit viper	CR
	thalassinus	Merendón palm pit viper	Ga, Ho
Bothriopsis		Forest pit vipers	
	bilineata	Two-striped forest pit viper	Cl, Ve, Gy, Su, FG, Br, Ec, Pe, Bo
	chloromelas	Inca forest pit viper	Pe
	medusa	Venezuelan forest pit viper	Ve
	oligolepis	Peruvian forest pit viper	Pe, Bo
	pulchra	Andean forest pit viper	Cl, Ec, Pe
	taeniata	Speckled forest pit viper	Cl, Ve, Gy, Su, FG, Br, Ec, Pe, Bo
Bothrocophias		Toadheaded pit vipers	
	campbelli	Ecuadorian toadheaded pit viper	Ec
	colombianus	Colombian toadheaded pit viper	Cl
	hyoprora	Amazonian toadheaded pit viper	Cl, Br, Ec, Pe, Bo
	microphthalmus	Small-eyed toadheaded pit viper	Cl, Br, Ec, Pe, Bo
	myersi	Chocoan toadheaded pit viper	Cl
Bothrops		Lanceheads	
	alcatraz	Alcatrazes lancehead	Br
	alternatus	Urutu	Br, Py, Ur, Ar
	ammodytoides	Patagonian lancehead	Ar
	andianus	Andean lancehead	Pe, Bo
	asper	Terciopelo	Ga, Be, Ho, Ni, CR, Pn, Cl, Ve, Ec
	atrox	Common lancehead	Cl, Ve, Gy, Su, FG, Br, Ec, Pe, Bo
	barnetti	Barnett's lancehead	Pe
	brazili	Brazil's lancehead	Cl, Ve, Gy, Su, FG, Br, Ec, Pe, Bo
	caribbaeus	Saint Lucia lancehead	SL
	cotiara	Cotiara	Br, Ar
	diporus	Chaco lancehead	Br, Py, Ar
	erythromelas	Caatinga lancehead	Br
	fonsecai	Fonseca's lancehead	Br
	insularis	Golden lancehead	Br
	itapetiningae	São Paulo lancehead	Br
	jararaca	Jararaca	Br, Py, Ar
	jararacussu	Jararacussu	Br, Bo, Py, Ar
	jonathani	Cochabamba lancehead	Bo
	lanceolatus	Martinique lancehead	Ma, LA, WI
	leucurus	Bahia lancehead	Br
	lojanus	Lojan lancehead	Ec
	lutzi	Cerrado lancehead	Br
	marajoensis	Marajó lancehead	Br
	mattogrossensis	Mato Grosso lancehead	Br, Bo, Py, Ar
	moojeni	Brazilian lancehead	Br, Bo, Py
	muriciensis	Murici lancehead	Br
	neuwiedi	Neuwied's lancehead	Br
	osbornei	Osborne's lancehead	Ec, Pe
	pauloensis	Black-faced lancehead	Br, Py
	pictus	Desert lancehead	Pe

TABLE 48-2. Venomous Reptiles of Central and South America and the Caribbean—cont'd

GENUS	SPECIES	COMMON NAME	COUNTRIES FOUND IN
	pirajai	Piraja's lancehead	Br
	pubescens	Pampas lancehead	Br, Ur, Ar
	punctatus	Chocoan lancehead	Pn, Cl, Ec
	roedingeri	Roedinger's lancehead	Pe
	sanctaecrucis	Bolivian lancehead	Bo
	venezuelensis	Venezuelan lancehead	Ve
Cerrophidion		Montane pit vipers	
	godmani	Godman's montane pit viper	Ga, Ho, ES, Ni, CR, Pn
Crotalus		Rattlesnakes	
	durissus	South American rattlesnake	Cl, Ve, Gy, Su, FG, Br, Pe, Bo, Py, Ur, Ar
	simus	Neotropical rattlesnake	Ga, Be, Ho, ES, Ni, CR
Heloderma		Venomous lizards	
	horridum	Mexican beaded lizard	Ga
Lachesis		Bushmasters	
	acrochorda	Chocoan bushmaster	Pn, Cl, Ec
	melanocephala	Black-headed bushmaster	CR
	muta	South American bushmaster	Cl, Ve, Gy, Su, FG, Br, Ec, Pe, Bo
	stenophrys	Central American bushmaster	Ni, CR, Pn
Leptomicrurus		Slender coral snakes	
	collaris	Guianan slender coral snake	Ve, Gy, Su, FG, Br
	narduccii	Andean slender coral snake	Cl, Br, Ec, Pe, Bo
	renjifoi	Ringed slender coral snake	Cl
	scutiventris	Pygmy slender coral snake	Cl, Br, Ec, Pe
Micrurus		Coral snakes	
	albicinctus	White-ringed coral snake	Cl, Br
	alleni	Allen's coral snake	Ho, Ni, CR, Pn
	altirostris	Uruguayan coral snake	Br, Ur
	ancoralis	Regal coral snake	Pn, Cl, Ec
	annellatus	Annellated coral snake	Cl, Br, Ec, Pe, Bo
	averyi	Black-headed coral snake	Gy, Su, Br
	baliocoryphus	Mesopotamian coral snake	Br, Py, Ar
	bocourti	Ecuadorian coral snake	Ec, Pe
	brasiliensis	Brazilian coral snake	Br
	browni	Brown's coral snake	Ga
	catamayensis	Catamayo coral snake	Ec
	circinalis	Trinidad coral snake	Ve
	clarki	Clark's coral snake	CR, Pn, Cl
	corallinus	Painted coral snake	Br, Py, Ur, Ar
	decoratus	Decorated coral snake	Br
	diana	Diana's coral snake	Bo
	diastema	Variable coral snake	Ga, Be, Ho
	dissoleucus	Pygmy coral snake	Pn, Cl, Ve
	dumerilii	Dumeril's coral snake	Pn, Cl, Ve, Ec
	elegans	Elegant coral snake	Ga
	filiformis	Slender coral snake	Cl, Br, Ec, Pe
	frontalis	Cerrado coral snake	Br, Py
	hemprichii	Hemprich's coral snake	Cl, Ve, Gy, Su, FG, Br, Ec, Pe, Bo
	hippocrepis	Mayan coral snake	Ga, Be
	ibiboboca	Caatinga coral snake	Su, FG, Br
	isozonus	Venezuelan coral snake	Cl, Ve, Br
	langsdorffi	Langsdorff's coral snake	Cl, Br, Ec, Pe
	latifasciatus	Long-ringed coral snake	Ga
	lemniscatus	South American coral snake	Cl, Ve, Gy, Su, FG, Br, Ec, Pe, Bo, Py, Ar
	margaritiferus	Speckled coral snake	Pe
	medemi	Medem's coral snake	Cl
	meridensis	Mérida pygmy coral snake	Ve
	mertensi	Mertens's coral snake	Ec, Pe
	mipartitus	Red-tailed coral snake	Pn, Cl, Ve, Ec, Pe
	multifasciatus	Many-banded coral snake	Ni, CR, Pn
	multiscutatus	Cauca coral snake	Cl
	nigrocinctus	Central American coral snake	Ga, Ho, ES, Ni, CR, Pn, Cl
	oligoanellatus	Tambo coral snake	Cl
	ornatissimus	Ornate coral snake	Cl, Br, Ec, Pe

Continued

TABLE 48-2. Venomous Reptiles of Central and South America and the Caribbean—cont'd

GENUS	SPECIES	COMMON NAME	COUNTRIES FOUND IN
Micrurus cont'd	*paraensis*	Pará coral snake	Br
	peruvianus	Peruvian coral snake	Ec, Pe
	petersi	Peters's coral snake	Ec
	psyches	Daudin's coral snake	Ve, Gy, Su, FG, Br
	putumayensis	Putumayo coral snake	Cl, Br, Pe
	pyrrhocryptus	Argentinian coral snake	Br, Bo, Py, Ar
	remotus	Neblina coral snake	Cl, Ve
	ruatanus	Roatán coral snake	Ho
	sangilensis	Santander coral snake	Cl
	serranus	Bolivian coral snake	Bo
	spixii	Amazonian coral snake	Cl, Ve, Br, Ec, Pe, Bo
	spurrelli	Colombian coral snake	Cl
	steindachneri	Steindachner's coral snake	Ec, Pe
	stewarti	Panamania coral snake	Pn
	stuarti	Stuart's coral snake	Ga
	surinamensis	Aquatic coral snake	Cl, Ve, Gy, Su, FG, Br, Ec, Pe, Bo
	tschudii	Desert coral snake	Ec, Pe
Pelamis	*platurus*	Yellow-bellied sea snake (see Chapter 74 for discussion of sea snake bites)	Ga, Ho, ES, Ni, CR, Pn, Cl, Ec, Pe
Porthidium		Hognose pit vipers	
	arcosae	Manabí hognose pit viper	Ec
	lansbergii	Lansberg's hognose pit viper	Pn, Cl, Ve
	nasutum	Rainforest hognose pit viper	Ga, Be, Ho, Ni, CR, Pn, Cl, Ec
	ophryomegas	Slender hognose pit viper	Ga, Ho, ES, Ni, CR
	porrasi	White-tailed hognose pit viper	CR
	volcanicum	Ujarran hognose pit viper	CR

Ar, Argentina; Be, Belize; Bo, Bolivia; Br, Brazil; Cl, Colombia; CR, Costa Rica; Ec, Ecuador; ES, El Salvador; FG, French Guiana; Ga, Guatemala; Gy, Guyana; Ho, Honduras; LA, Lesser Antilles; Ma, Martinique; Ni, Nicaragua; Pe, Peru; Pn, Panama; Py, Paraguay; SL, Saint Lucia Island; Su, Suriname; Ur, Uruguay; Ve, Venezuela; WI, West Indies.
Adapted from Campbell JA, Lamar WM: The Venomous Reptiles of the Western Hemisphere. Ithaca, NY, Comstock, 2004.

are anecdotal reports of rare fatalities by a few Latin American colubrids such as *Phalotris lemniscatus trilineatus*, *Philodryas olfersii*, and *Tachymenis peruvianus*.[181] Patients with severe colubrid envenomation may demonstrate local and systemic bleeding, intravascular hemolysis, and acute renal failure.

Pit vipers are widely dispersed throughout most of the New World south of southern Canada (i.e., south of 55° north latitude).[128] In the United States, for example, all 48 contiguous states except Maine have at least one pit viper species.[142] Because snakes are poikilothermic and rely on environmental heat energy to support locomotion, feeding, digestion, and reproduction, the number of snake species tends to increase as one moves toward the equator. More than 100 species of pit vipers are found in the New World,[26] and these can be divided into rattlesnakes (genera *Crotalus* and *Sistrurus*, approximately 31 species); copperheads, cottonmouth water moccasins, and cantils (genus *Agkistrodon*, four North American species); and the numerous Latin American crotalines (genera *Atropoides* [jumping pit vipers], *Bothriechis* [palm-pit vipers], *Bothriopsis* [forest-pit vipers], *Bothrocophias* [toad-headed pit vipers], *Bothrops* [lancehead pit vipers], *Cerrophidion* [montane pit vipers], *Lachesis* [bushmasters], *Ophryacus* [Mexican pit vipers], and *Porthidium* [hognose pit vipers]). There are more than 70 species of coral snakes in the Western Hemisphere, and one species of sea snake *(Pelamis platurus)* is found along the Pacific coast of Central and South America, reaching the southwestern coast of North America. Sea snakes are discussed in Chapter 74.

Figure 48-1. Eastern diamondback rattlesnake *(Crotalus adamanteus)* is the largest pit viper of the United States and can attain lengths of 2 m ($6^1/_2$ feet). (Courtesy Michael Cardwell/Extreme Wildlife Photography.)

Rattlesnakes are the most widespread of pit vipers, found throughout most of the Americas (Figs. 48-1 to 48-9). Copperheads *(Agkistrodon contortrix)* are found in the central and southeastern United States and westward into the Big Bend region of Texas (Fig. 48-10). Cottonmouth water moccasins *(Agkistrodon piscivorus)* are found in the southeast from

Figure 48-2. Western diamondback rattlesnake *(Crotalus atrox)* causes many serious bites in the U.S. Southwest. (Courtesy Michael Cardwell/Extreme Wildlife Photography.)

Figure 48-5. Prairie rattlesnake *(Crotalus viridis viridis)* is a widely distributed species of the western United States. (Courtesy Michael Cardwell/Extreme Wildlife Photography.)

Figure 48-3. Mojave rattlesnake *(Crotalus scutulatus)* has two geographic populations in terms of venom composition, one with predominantly neurotoxic effects and one with more local sequelae. (Courtesy Michael Cardwell/Extreme Wildlife Photography.)

Figure 48-6. Northern Pacific rattlesnake (*Crotalus oreganus oreganus,* formerly *C. viridis oreganus*) is a moderate-sized but very toxic snake of the Pacific Northwest. (Courtesy Michael Cardwell/Extreme Wildlife Photography.)

Figure 48-4. Timber rattlesnake *(Crotalus horridus)* is a large, dangerous snake of the eastern United States. (Courtesy Michael Cardwell/Extreme Wildlife Photography.)

Figure 48-7. Southern Pacific rattlesnake (*Crotalus oreganus helleri,* formerly *C. viridis helleri*) replaces *C. oreganus oreganus* in southern California and northwestern Mexico. (Courtesy Michael Cardwell/Extreme Wildlife Photography.)

Figure 48-8. Tropical rattlesnake (*Crotalus simus,* formerly *C. durissus durissus*) is a large, dangerous species found in southern Mexico. (Courtesy Michael Cardwell/Extreme Wildlife Photography.)

Figure 48-9. Western pygmy rattlesnake *(Sistrurus miliarius streckeri)* is one of the smaller rattlesnake species of North America. (Courtesy Michael Cardwell/Extreme Wildlife Photography.)

Figure 48-10. Southern copperhead *(Agkistrodon contortrix contortrix)* has markings that make it almost invisible when lying in leaf litter. (Courtesy Michael Cardwell and Carl Barden Venom Laboratory.)

Figure 48-11. Cottonmouth water moccasin *(Agkistrodon piscivorus)* exhibiting its threat display. This snake is found most often around standing water sources in the southeastern United States. (Courtesy Sherman Minton, MD.)

Figure 48-12. Cantil *(Agkistrodon bilineatus)* is a close relative of the copperheads *(A. contortrix)* and cottonmouths *(A. piscivorus)* of the United States. This pit viper is found in Mexico and Central America. (Courtesy Michael Cardwell and William W. Lamar.)

Virginia to Florida and extend westward into central Texas (Fig. 48-11). In Mexico, the copperhead and cottonmouth are replaced by the cantils, *Agkistrodon bilineatus* (Fig. 48-12) and *Agkistrodon taylori.* The pit vipers of North America come in a wide range of sizes. Among the smaller rattlesnakes are the sidewinders *(Crotalus cerastes),* ridge-nosed rattlesnakes *(Crotalus willardi),* and pygmy rattlesnakes *(Sistrurus miliarius),* whose adult sizes are routinely less than 65 cm (25.6 inches).[100] At the other extreme, the eastern diamondback rattlesnake *(Crotalus adamanteus)* can exceed 2 m ($6^1/_2$ feet).[100] The largest pit viper, however is the Central and South American bushmaster (genus *Lachesis*), with a recorded maximal length of 3.6 meters (11.8 feet).[49]

Outside of the Western Hemisphere, the family Elapidae contains a number of extremely dangerous species, such as cobras (genus *Naja*), mambas (genus *Dendroaspis*), and all the potentially lethal snakes of Australia (e.g., *Oxyuranus, Notechis, Pseudonaja*) (see Chapter 49). The elapids of North America and Latin America are all coral snakes and belong to one of three genera, *Leptomicrurus, Micrurus,* and *Micruroides* (Figs. 48-13 to 48-15). In the United States, these colorful reptiles are

Figure 48-13. Texas coral snake (*Micrurus tener,* formerly *M. fulvius tenere*) has a highly potent venom but is secretive, and bites are uncommon. (Courtesy Michael Cardwell and the Gladys Porter Zoo.)

Figure 48-15. Sonoran coral snake *(Micruroides euryxanthus)* is also known as the Arizona coral snake. No documented fatality has followed a bite by this species. (Courtesy Michael Cardwell and Jude McNally.)

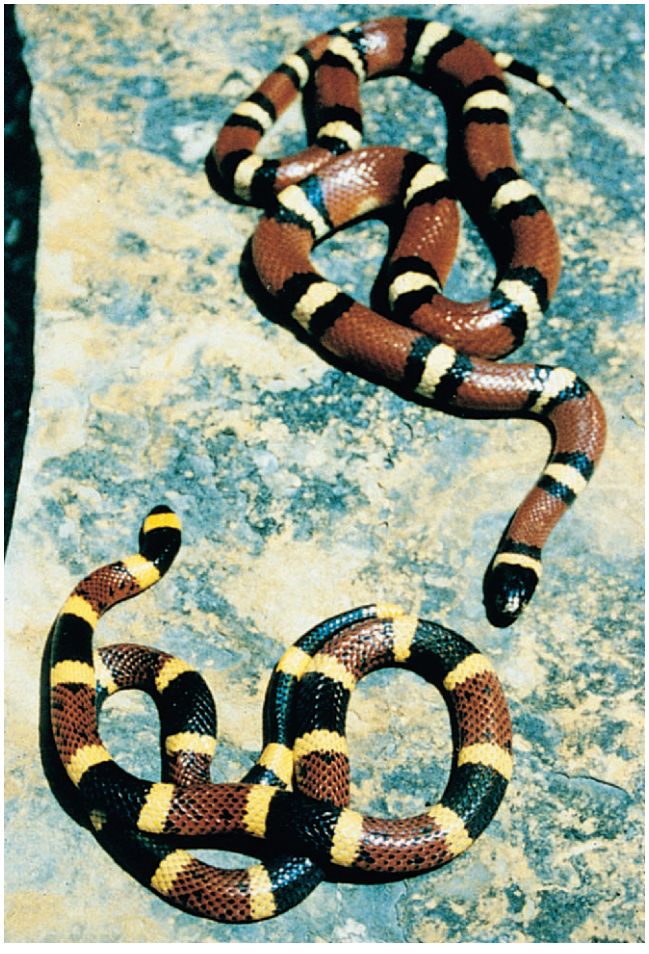


Figure 48-14. Comparison of the Texas coral snake *(Micrurus tener)* with the harmless Mexican milk snake *(Lampropeltis triangulum annulata).* Coral snake *(bottom)* has contiguous red and yellow bands, whereas red and yellow bands of the milk snake are separated by black. (Courtesy Charles Alfaro.)

found in Arizona (Sonoran coral snake, *Micruroides euryxanthus*), the southeastern United States (eastern coral snake, *Micrurus fulvius*), and Texas (Texas coral snake, *Micrurus tener* [formerly *M. fulvius tenere*]). Mexico has 16 species of coral snake, including *M. euryxanthus* and 15 *Micrurus* species. Central and South America boast more than 50 species of coral snakes (*Micrurus* and *Leptomicrurus*).[26] Because of the inoffensive habits of these fossorial (burrowing) snakes and their less efficient venom delivery device (see Anatomy), bites are relatively uncommon. Although in 1967 Parrish and Khan[143] estimated that fewer than 20 coral snake bites occurred in the United States each year, the 2003 report of the American Association of Poison Control Centers (AAPCC) Toxic Exposure Surveillance System recorded 97 cases.[182]

The only two known species of venomous lizards in the world are found in North and Central America, and they belong to the genus *Heloderma*. The Gila monster *(Heloderma suspectum)* is found in the southwestern United States (Arizona, western New Mexico, southeastern California, the southern tip of Nevada, extreme southwestern Utah) and northwestern Mexico (Fig. 48-16).[172] The range of the Mexican beaded lizard *(Heloderma horridum)* is south of that of the Gila monster, south to Guatemala (Fig. 48-17).[162] The helodermatids are discussed in more detail later (see Venomous Lizards).

VENOMOUS SNAKES

Anatomy

Pit Vipers

The term *pit viper* comes from the presence of paired, thermoreceptor organs (pits) present on the forward portion of these snakes' heads (Fig. 48-18). These structures, also known as foveal organs, serve the snake in locating prey, aiming strikes, and adjusting venom dose (Fig. 48-19). A neurologic feedback loop between the foveal organs and the venom delivery apparatus may allow the snake to adjust the volume of venom it injects into a potential meal or a perceived threat.[193]

Figure 48-16. Gila monster (*Heloderma suspectum*) is one of only two known venomous lizards and the only species found in the United States. (Courtesy Michael Cardwell/Extreme Wildlife Photography.)

Figure 48-17. Mexican beaded lizard (*Heloderma horridum*) is located south of the Gila monster's range in Mexico. (Courtesy Michael Cardwell/Extreme Wildlife Photography.)

The anatomy of the venom delivery system of crotalines is the most sophisticated of all snakes (Fig. 48-20). Bilateral glands located at the sides of the head, behind the eyes, produce and store the venom. These glands are connected through ducts to more anterior accessory glands that probably serve to activate or potentiate the venom.[155] From here, the venom is passed forward through other ducts into the proximal portion of the hollow fangs with which the snake pierces its victim in a stabbing motion. These fangs, found on the anterior surfaces of the maxillary bones, are large (up to 20 mm [0.79 inches] in large rattlesnakes[100]) and highly mobile. At rest, the snake folds the fangs against the roof of its mouth. For the strike, the fangs are brought into an upright position, perpendicular to the maxillae. The snake has voluntary control over its fangs and can open its mouth without raising the fangs or can raise each fang individually. The fangs are relatively brittle and fracture or become dull with time and use. Replacement fangs at varying stages of development behind the functional set move forward on the

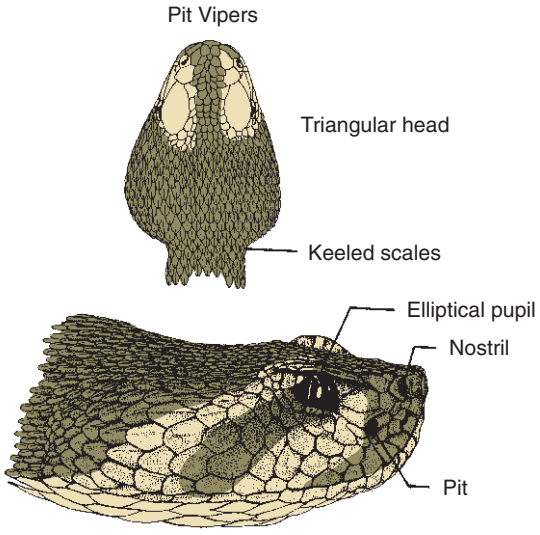


Figure 48-18. Pit viper's head. Note the elliptical pupil and the heat-sensing pit for which these reptiles are named. Viewed from above, the head has a distinctly triangular shape. Many nonvenomous snakes also possess triangular heads, but this is not a reliable means of differentiation. (Marlin Sawyer, 1994.)

maxillary bone when needed (Fig. 48-21). The speed of a pit viper's strike has been clocked at 8 feet (2.4 m) per second; the snake can reach distances of approximately one half of its body length.[193] Table 48-3 lists venom yields for various North American pit vipers.

The fastest pit viper can crawl at a maximum speed of approximately 3 miles (4.8 km) per hour, which equates to an average adult walking pace.[193] Crotaline snakes do not chase people. Accounts suggesting otherwise can be explained by snakes' poor eyesight: when threatened, they may unwittingly retreat in the direction of people.

The characteristic forked tongue of the snake is an olfactory tool and possesses none of the offensive "stinging" function ascribed to it in folklore. The snake extends its tongue to detect chemical odors in its environment. The tongue is then retracted and its tips placed into the paired Jacobson's organs, lined with olfactory epithelium, in the roof of its mouth. This sensory system is highly sensitive, allowing the snake to identify potential mates, locate food, and track down a prey item that has been struck and released to die.

Pit vipers have elliptical, or catlike, pupils (see Figure 48-18), whereas most American harmless snakes have round pupils. A few essentially harmless, rear-fanged colubrids in the United States, such as the night snake *(Hypsiglena torquata)* and lyre snake *(Trimorphodon biscutatus),* also possess elliptical pupils but lack facial pits. Although these species possess a special venom gland (Duvernoy's gland) in the rear of the mouth, they are innocuous creatures. They are reluctant to bite humans, and their salivary toxins cause few signs or symptoms, other than slight swelling, bruising, and pain. This contrasts with the truly dangerous rear-fanged colubrids of other parts of the world, including a handful of species in Latin American and in the Eastern Hemisphere (see earlier and Chapter 49). The two species of boas in North America, the rosy boa *(Lichanura*

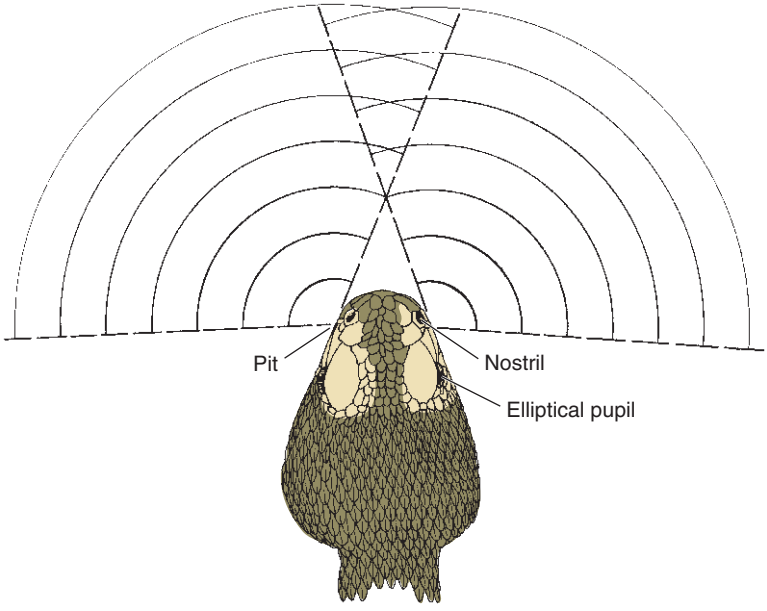


Figure 48-19. Paired heat-sensing pits of the pit vipers used to help the snake locate its prey, direct its strike, and probably determine the volume of venom to be expended. (Marlin Sawyer, 1994.)

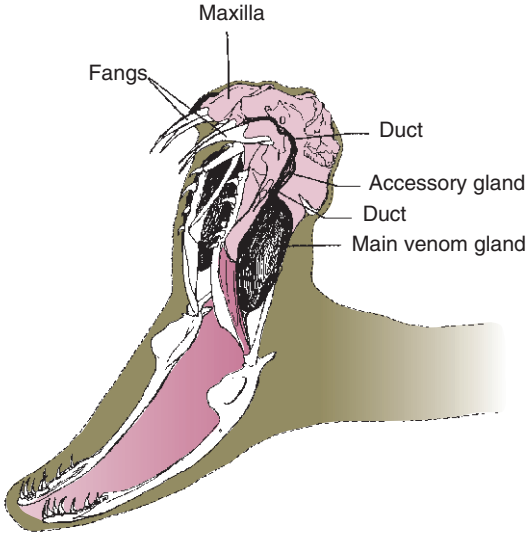


Figure 48-20. Venom delivery apparatus of a pit viper. Venom is produced in large venom glands just posterior to the eyes and is passed through a duct system into the hollow, anterior fangs when the snake bites. (Marlin Sawyer, 1994.)

Figure 48-21. Rattlesnake skull. Replacement fangs are behind the primary, functional fangs. The smaller teeth of the palatine, pterygoid, and mandibular bones are used for gripping food. (Courtesy Michael Cardwell/Extreme Wildlife Photography.)

trivirgata) and the rubber boa *(Charina bottae)*, also possess elliptical pupils, but their body form (more cigar shaped and lacking the broad, triangular head of a pit viper) and skin patterns are very different from those of any pit viper.

The caudal rattle of the rattlesnake is composed of loosely interlocked plates of keratin that emit a buzzing sound when the snake rapidly vibrates its tail. This characteristic sound serves as a warning to a perceived threat. A new segment is added to the rattle each time the snake sheds its skin, which can occur from one to several times each year, depending on its age, health, and feeding success. Newborn rattlesnakes have a single button present at birth. Not until after their first shed do they possess a true rattle that can create a sound. Rattles may be broken off during the snake's life, so their number cannot be used reliably to determine age. Although rattlesnakes are generally quick to sound out a warning when threatened, it is a misconception that they will always do so before striking.

Another helpful anatomic feature of pit vipers in the United States is the scale pattern on the underside of the tail, the subcaudal scales. The junction of the snake's body and its tail can be easily ascertained by viewing its ventral side. At this location is a large scale (sometimes divided) known as the anal plate. Just distal to this in U.S. pit vipers is a sequence of single scales that entirely cross the ventral surface. In nonvenomous snakes and in coral snakes (as well as some Latin American pit vipers) the subcaudal scales are paired (i.e., each covers approximately half the width of the tail) (Fig. 48-22). This feature becomes clinically useful when a U.S. snakebite victim brings in the body of a decapitated snake for identification and when the skin pattern is unfamiliar to the physician.

TABLE 48-3. Venom Yields of Some Medically Important Snakes of North America

SPECIES	MAXIMUM VENOM YIELD (mg DRY WEIGHT)	REFERENCE
Crotalus adamanteus	848	100
Crotalus atrox	1145	100
Crotalus cerastes	63	100
Crotalus durissus	514 (average)	100
Crotalus horridus	229	100
Crotalus molossus	540	100
Crotalus oreganus helleri (formerly *C. viridis helleri*)	390	100
Crotalus oreganus (formerly *C. viridis oreganus*)	289	100
Crotalus scutulatus	141	100
Crotalus viridis	162	100
Sistrurus catenatus	33	58
Sistrurus miliarius	18 (average)	100
Agkistrodon contortrix	45	141
Agkistrodon piscivorus	150	141
Micruroides euryxanthus	6	152
Micrurus fulvius	38	152
Micrurus nigrocinctus	20	152

Coral Snakes

Coral snakes are identified primarily by color pattern. U.S. coral snakes are banded in a red-yellow-black-yellow-red pattern (see Figure 48-14), and the bands completely encircle the snake's body. The contiguity of the red and yellow bands distinguishes U.S. coral snakes from a number of harmless mimics (e.g., several king snakes and milk snakes, genus *Lampropeltis*), which generally have red and yellow bands separated by black bands. This can best be remembered by recalling the phrase "red on yellow, kill a fellow; red on black, venom lack" or by considering that the red and yellow lights on a traffic signal are the warning lights. Contiguous red and yellow bands can be used to reliably identify coral snakes only in North America, north of Mexico City. Further south, routinely bicolor (red and black) coral snake species are found, and there are many harmless mimics that closely resemble these venomous serpents.[129] Although one harmless U.S. colubrid has a contiguous red and yellow banding pattern (the shovel-nosed snake, *Chionactis* species), this is an inoffensive reptile, and its bands do not completely encircle its body. In exceptionally rare cases, coral snakes can be all black (melanistic) or albino.[126]

The coral snake venom apparatus is much less complex than that of the pit vipers. The paired venom glands connect through ducts to slightly enlarged, hollow, maxillary fangs that are fixed in an upright position in the forward portion of the jaw (Fig. 48-23). For the coral snake to inflict a potentially serious bite, it must chew on the victim to inject a sufficient volume of venom through its relatively small fangs. These animals are not capable of striking out with the stabbing motion of the crotalines. In the vast majority of coral snakebite cases, the victim was handling the creature when bitten, and, in some cases, this occurred when the victim misidentified the snake as a harmless kingsnake.[144]

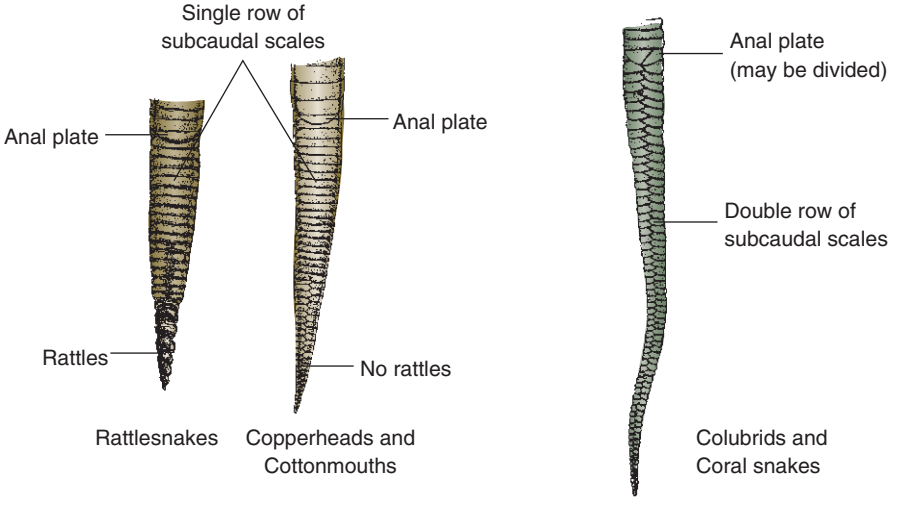


Figure 48-22. Subcaudal scale patterns of U.S. snakes. In the United States, pit vipers have a single row of scales on the ventral side of the tail, just distal to the anal plate. Most harmless colubrid snakes have double rows of subcaudal scales, as do coral snakes and some species of Latin American pit vipers. (Marlin Sawyer, 1994.)

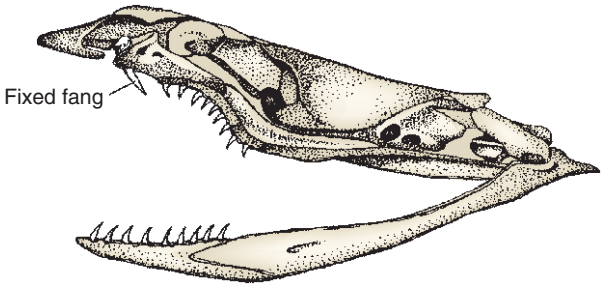


Figure 48-23. Coral snake's skull. Note the slightly enlarged anterior maxillary fang that is fixed in its upright position. (Marlin Sawyer, 1994.)

Venoms

Snake venoms are extraordinarily variable and complex chemical cocktails of approximately 100 distinct molecular moieties. A complete discussion of all known venom components is beyond the scope of this text. Some of the better characterized and more interesting of these include the phospholipase A_2 neurotoxins, metalloproteinases, and thrombinlike enzymes.[41,95] Phospholipase A_2 neurotoxins are among the most toxic of pit viper venom components and can increase the lethality of the venom 10- to almost 100-fold.[59] The first of these, crotoxin, was isolated in 1938 from the South American rattlesnake *(Crotalus durissus terrificus)*.[1] Phospholipase A_2 neurotoxins noncompetitively bind to presynaptic calcium channels, inhibiting acetylcholine release and thereby blocking neurotransmission at the neuromuscular junction, inactivating the muscle. This can cause paralysis of the muscles of respiration, for example, and lead to respiratory difficulty. Additionally, phospholipase A_2 damages muscle cell membranes, allowing calcium influx and release of creatine and creatine kinase (CK), which can progress to diffuse myonecrosis and rhabdomyolysis.[97] Metalloproteinases, the enzymes that cause much of the locally destructive effects of pit viper envenomation, activate tumor necrosis factor alpha and stimulate endogenous human metalloproteinases, intensifying inflammation. Certain metalloproteinases, the hemorrhagins, cause leakage of red blood cells out of the vasculature, leading to ecchymosis (characteristic of more severe pit viper bites) and fluid shifts.[70,189] High venom toxicity is generally associated with phospholipase A_2 neurotoxins, which are typically (but not always) inversely related in proportionate concentrations to the tissue-damaging metalloproteinases.[122] Thrombin-like enzymes cause a consumptive coagulopathy but do not directly activate coagulation factors or complex with antithrombin III (and so are not affected by heparin). Disintegrins bind to proteins on blood platelets, blocking their combination with fibrinogen to form clots.[24] Bradykinins cause hypotension, vomiting, and pain. Hyaluronidase decreases the viscosity of connective tissues, allowing venom to spread. Lysolecithin, a byproduct of the enzymatic action of phospholipase A_2, damages mast cell membranes and results in histamine release.[31]

Pit viper venom has both offensive (i.e., food gathering) and defensive functions. In predatory strikes, the venom immobilizes the prey, facilitates its retrieval by altering its scent, and accelerates digestion.[30,80,82] Defensive strikes are meant to deter predators and tormentors. The amount of venom injected differs from bite to bite. Factors such as prey size and species, duration of fang contact, and time elapsed since last meal influence the amount of venom released.[80,82] Rattlesnakes have been shown to inject significantly more venom into large mice than into small mice.[80] The mass of venom expended differs between predatory and defensive bites. In comparison studies, venom expenditure by North American pit vipers appears to be greater in defensive bites than in predatory bites.[82] In one comparison, the northern Pacific rattlesnake (*Crotalus oreganus* [formerly *C. viridis oreganus*]) expended almost 4 times more venom when biting a hand model (defensive) than a mouse (predatory).[80,82] The most important factor influencing potential venom delivery is the size of the snake.[82] A direct relationship has been demonstrated between snake length and mass of venom expended in both predatory and defensive bites.[78,82]

A popular belief is that juvenile rattlesnakes are more dangerous than adult snakes because their venom is more toxic and they are unable to control the volume they release. The venom of some juvenile rattlesnakes may be slightly more toxic, but larger rattlesnakes are capable of delivering much greater amounts of venom in a bite. Juvenile prairie rattlesnakes (*Crotalus viridis* [formerly *C. viridis viridis*]) have been shown in one small study to possess venom that is 2 to 3 times more toxic than that of adults.[50] Large adult snakes, however, deliver an average of 17 times (range, approximately 10 times to greater than 100 times) more venom than juveniles.[78] The ability to control venom expenditure has been demonstrated in juvenile rattlesnakes. In a series of first exposures to different-sized prey, "naive" juvenile rattlesnakes injected similar quantities of venom into all size classes. However, in the second series of exposures, "experienced" snakes injected significantly more venom into larger prey.[79] The clinical relevance of this is uncertain. In many species, venom composition appears to change as the snake ages. Phospholipase A_2 activity decreases with age, probably accounting for some decrease in toxicity. Proteolytic activity, however, increases with age, possibly to aid digestion of larger prey eaten by older, larger snakes.[78] Coagulopathic effects can be different between juvenile and adult western diamondback rattlesnakes *(Crotalus atrox)*, partly because of greater amounts of thrombinlike enzymes in younger snakes.[148]

Clinical studies showing that envenomations by large rattlesnakes are generally more severe than envenomations by smaller ones corroborate and complement these findings in the laboratory and the field.[76,91] Not to be underestimated, however, juvenile pit vipers can deliver a very serious, even life-threatening envenomation.[133]

Venom characteristics vary with geographic origin of the snake.[61] For example, certain populations of the Mojave rattlesnake *(Crotalus scutulatus)* cause human neurotoxicity with severe envenomation while causing minimal local tissue destruction and no hemorrhagic effects.[34,92] Neurotoxic findings may include respiratory difficulty, generalized weakness, and cranial nerve palsies.[34] The venoms of these snakes possess a presynaptic neurotoxin, Mojave toxin, and are classified as venom A populations. Venom B populations lack Mojave toxin and are less toxic. Bites by venom B snakes result in consequences more typical of most rattlesnake envenomation: soft tissue swelling, necrosis, and coagulopathy. Venom A populations are found in California, western Arizona, Nevada, Utah, New Mexico, and Texas. Venom B populations are found in more eastern parts of Arizona. A zone of intergradation between venom A and venom B populations occurs along a line between Phoenix and Tucson.[73,191] Toxins with structure and physiologic effects similar to those of Mojave toxin have been isolated from

venoms of other species of rattlesnakes, including southern Pacific rattlesnakes (*Crotalus oreganus helleri* [formerly *C. viridis helleri*]), prairie rattlesnakes (*C. viridis* [formerly *C. viridis viridis*]), midget faded rattlesnakes (*C. concolor* [formerly *C. viridis concolor*]), tropical rattlesnakes *(C. durissus)*, timber rattlesnakes *(Crotalus horridus)*, and tiger rattlesnakes *(Crotalus tigris)*.[52,60,62,81,187] Geographic differences occur in the venoms of other snakes as well. Timber rattlesnakes (sometimes referred to as canebrake rattlesnakes) *(C. horridus)* from Florida, Georgia, and South Carolina possess more neurotoxic and myotoxic "canebrake toxin" than do specimens from Alabama, Mississippi, Tennessee, and North Carolina.[62] Differences in concentration of this toxin correlate with variable clinical effects seen after bites by this species from different geographic regions.[27]

Neurotoxicity has been clinically associated with severe myotoxicity in many cases.[21,27,38,92] Severe rhabdomyolysis and myoglobinuric renal failure have been reported after Mojave rattlesnake envenomation and are thought to be related to Mojave toxin.[92] The association between neurotoxicity and myotoxicity has been confirmed in laboratory animals.[6] *C. horridus* specimens possessing significant amounts of the neurotoxin (canebrake toxin) produce a rise in serum CK levels as a biochemical signature of significant envenomation. The rise in CK level appears to parallel the severity of poisoning by these snakes.[27] Autopsy findings have demonstrated that myonecrosis in this setting is systemic and not limited to the bite site.[27,98] Concomitant rises in MB fractions of CK can occur in the absence of any clinical evidence of cardiac damage. In one such case, troponin-T was measured as negative despite abnormal total CK and CK-MB.[27] Lesser CK elevations (usually less than 500 U/L) may be seen with other rattlesnake bites, such as that of the eastern diamondback *(C. adamanteus)*. In these cases the elevations appear to more closely parallel local effects.[27]

Mojave toxin is thought to inhibit acetylcholine release at the presynaptic terminal of the neuromuscular junction.[34] Myokymia, or muscle fasciculation, may be considered by some a manifestation of neurotoxicity. This phenomenon, however, occurs through a different mechanism than Mojave toxin–induced neurotoxicity. Muscle fasciculations are believed to be caused by the interaction of certain venom components with calcium or calcium-binding sites on the nerve membrane.[34] Fasciculations may occur after envenomation by various species of rattlesnakes, including northern and southern Pacific rattlesnakes (*C. oreganus oreganus* [formerly *C. viridis oreganus*] and *C. oreganus helleri* [formerly *C. viridis helleri*], respectively), eastern diamondback rattlesnakes *(C. adamanteus)*, western diamondback rattlesnakes *(C. atrox)*, Mojave rattlesnakes *(C. scutulatus)*, and timber rattlesnakes *(C. horridus)*.[10,34,184]

Coral snake venoms are less complex than pit viper venoms and have received less research attention. *Micrurus* and *Micruroides* venoms have minimal proteolytic activity but contain the spreading enzyme hyaluronidase and some phospholipase A_2.[162] The primary lethal component is a low-molecular-weight, postsynaptic neurotoxin that blocks acetylcholine binding sites at the neuromuscular junction.[28,179] In addition, the venom contains at least one myotoxic component that may clinically produce a rise in CK levels.[69]

What coral snake venom lacks in complexity it makes up in potency. Among U.S. snakes, *Micrurus* and *Micruroides* venom potency, as determined by median lethal dose (LD_{50}) values in mice, is surpassed only by that of the Mojave rattlesnake *(C. scutulatus)*.[155] It is estimated that a full-grown coral snake carries enough venom in its delivery apparatus to kill four to five adult humans.[51] It is indeed fortunate that these reptiles are shy, inoffensive, and possess a less effective venom delivery device than the pit vipers.

Many modern medicines are derived from reptile venoms, and these are being investigated and utilized for the treatment of heart disease, stroke, cancer, diabetes, hypertension, pain, and other conditions. Some of the most promising of these venom-derived pharmaceuticals come from the disintegrins.[24]

Clinical Presentation

Pit Vipers

The clinical presentation of pit viper envenomation is quite variable and depends on the circumstances of the bite. Important factors include the species, size, and health of the snake, the age and health of the victim, the circumstances that led up to the bite, the number of bites and their anatomic locations, and the quality of the care rendered to the person, both in the field and in the hospital. Although statistics vary, most accidental bites occur to the lower extremities.[140] The next most common anatomic site is an upper extremity. Many of these bites occur while the victim is intentionally interacting with the snake (e.g., tormenting the animal, trying to catch it, or handling a captive specimen). Less often, bites occur to the head, neck, or trunk. Most bites occur around dawn or dusk, during warmer months, when snakes and people are more active outdoors.[140] A young, intoxicated male bitten on the hand while intentionally interacting with a snake is a common clinical profile in the United States.

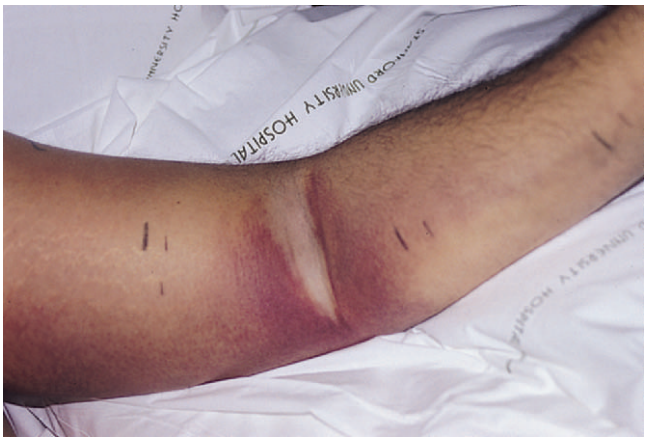
About 75% to 80% of pit viper bites result in envenoming. Approximately one in every four to five bites is "dry," meaning no venom has been injected.[142,155] The snake may voluntarily choose to save its venom for its next meal rather than waste it on a large human. Alternatively, the feedback mechanism may "short-circuit" between the pit organs and the venom apparatus, so that when faced with a huge, heat-radiating mass (a human), the system fails and no venom is expelled. Other possible causes of dry bites include glancing blows that fail to penetrate the skin and an exhausted venom supply. Approximately 35% of cases are mild envenomations, 25% are moderate, and 10% to 15% are severe.[142] Given their less efficient venom delivery system, coral snakes effectively envenom only about 40% of the time.[155]

The clinical findings found in crotaline envenomation can be divided into local and systemic signs and symptoms (Table 48-4). After most pit viper bites, severe burning pain at the site begins within minutes. Soft tissue swelling then progresses outward to a variable extent from the bite site. Over hours, a bitten extremity can swell all the way to the trunk. Bites to the face or neck may result in rapid, severe swelling that can compromise the airway.[149] Blood may persistently ooze from fang marks, marking the presence of anticoagulant substances in the venom. Ecchymosis is common, both locally and at more remote sites, as the vasculature becomes leaky and red blood cells (RBCs) escape into the soft tissues (Fig. 48-24). Fang marks are usually evident as small puncture wounds, but the precise bite pattern can be misleading.[131] Most nonvenomous snakebites result in multiple rows of tiny puncture wounds (from the maxillary, palatine, pterygoid, and mandibular teeth),

TABLE 48-4. Signs and Symptoms Seen after Rattlesnake Bites

SIGN OR SYMPTOM	FREQUENCY*
Fang marks	100/100
Swelling and edema	74/100
Pain	65/100
Ecchymosis	51/100
Vesiculations	40/100
Change in pulse rate	60/100
Weakness	72/100
Sweating and/or chill	64/100
Numbness or tingling of tongue and mouth, or scalp or feet	63/100
Faintness or dizziness	57/100
Nausea, vomiting, or both	48/100
Blood pressure changes	46/100
Change in body temperature	31/100
Swelling regional lymph nodes	40/100
Fasciculations	41/100
Increased blood clotting time	39/100
Spherical red blood cells	18/100
Tingling or numbness of affected part	42/100
Necrosis	27/100
Respiratory rate changes	40/100
Decreased hemoglobin	37/100
Abnormal electrocardiogram	26/100
Cyanosis	16/100
Hematemesis, hematuria, or melena	15/100
Glycosuria	20/100
Proteinuria	16/100
Unconsciousness	12/100
Thirst	34/100
Increased salivation	20/100
Swollen eyelids	2/100
Retinal hemorrhage	2/100
Blurring of vision	12/100
Convulsions	1/100
Muscle contractions	6/100
Increased blood platelets	4/25
Decreased blood platelets	42/100

*Times symptom or sign is reported as observed per total number of patients.
From Russell FE, Walter FG, Bey TA, Fernandez MC: Toxicon 35:1469-1522, 1997, with permission.



A

B

Figure 48-24. **A,** Mottled rock rattlesnake *(Crotalus lepidus lepidus)* bite in a young man at 24 hours. Note the exudation of red cells into the soft tissues remote from the bite site. The man was bitten on his left thumb. **B,** Northern Pacific rattlesnake *(Crotalus oreganus oreganus)* bite to the left pretibial region resulted in extensive ecchymosis of this patient's right lower extremity and extended into his flank (seen here 1 week after the bite). (**A** and **B,** Courtesy Robert Norris, MD.)

which usually coagulate quickly. Pit vipers also possess palatine, pterygoid, and mandibular teeth, which can result in more than the classic paired puncture marks. Also, a snake may make contact with only a single fang. Thus, the associated signs and symptoms should be considered more than the bite pattern in determining whether a bite was inflicted by a pit viper or another snake. Some rattlesnake bites result in little or no local pain despite envenoming; the best example is the Mojave rattlesnake *(C. scutulatus)*. Specimens containing significant amounts of Mojave toxin in their venom tend to cause few local findings. This can lead the treating physician to misjudge the severity of envenoming.[193] Over a period of hours to days, the patient may develop hemorrhagic or serous vesicles and bullae at the bite site and more proximally, especially if there is a delay in obtaining care (Fig. 48-25).

Systemic findings after pit viper bites can be extremely variable because any organ system can be affected. Nausea with or

Figure 48-25. Hemorrhagic bleb at the site of a western diamondback rattlesnake *(Crotalus atrox)* bite at 24 hours. (Courtesy Robert Norris, MD.)

without vomiting is common and may occur early in serious bites. The victim may complain of an overall sense of weakness. An odd sense of taste, such as a rubbery, minty, or metallic taste, may be present.[155] Occasionally the victim complains of a numb sensation of the mouth or tongue. The vital signs may be abnormal. The respiratory and heart rates may be increased. The victim may experience respiratory distress as a result of neurotoxic components of the venom, especially after bites by venom A–producing Mojave rattlesnakes *(C. scutulatus)*.[155] Another important cause of respiratory distress is pulmonary edema from pulmonary artery congestion and translocation of intravascular fluid into alveoli. This can be compounded by myocardial depressant factors in some venoms.[155] The victim's blood pressure may be elevated; however, hypotension, which may progress to frank shock, is more common in severe cases. In the first several hours, hypotension is usually caused by pooling of volume in the pulmonary and splanchnic beds. Later, as swelling progresses and fluids exude into remote soft tissues, intravascular volume can become significantly depleted. A rare cause of early shock is an anaphylactoid reaction to the venom.[11,22,45]

Musculoskeletal and neurologic abnormalities may be present. As mentioned, a number of rattlesnake venoms possess a component that can result in local or systemic muscle fasciculations as a sign of significant envenoming. These fasciculations can persist for many hours despite adequate treatment with antivenom. Other findings of neurologic dysfunction can include paresthesias, numbness, and frank motor weakness, especially after bites by some Mojave rattlesnakes *(C. scutulatus)* and eastern diamondback rattlesnakes *(C. adamanteus)*.

Although uncommon, hemorrhage can occur at multiple anatomic locations because of the complex procoagulant, anticoagulant, and metalloproteinase components of some venoms.[155] Bleeding can occur in the gingival membranes, renal system (microscopic or frank hematuria), gastrointestinal tract (Hemoccult-positive stools, frank blood per rectum, or hematemesis), pulmonary tree (hemoptysis) or central nervous system. The Martinique lancehead *(Bothrops lanceolatus)* and Saint Lucia lancehead *(Bothrops caribbaeus)* are unusual in that they can induce intravascular thrombosis and resultant end-organ damage via the unique thrombotic components in their venoms.[189]

Laboratory evaluation of a victim of pit viper bite may reveal significant abnormalities. The white blood cell count may be elevated, reflecting a neutrophil leukocytosis. The hematocrit may be elevated from hemoconcentration or may be depressed secondary to bleeding or hemolysis. Platelet counts can drop precipitously due to consumptive coagulopathy, sequestration at the bite site, or direct venom effects.[188] Serum chemistries may be abnormal. Blood sugar may be elevated. Muscle damage can result in elevated serum potassium and CK levels. Renal dysfunction may result from hypotension, myoglobin and hemoglobin deposition, and direct venom effects.[32] Hepatic dysfunction with elevations of serum transaminases may be seen.[125] Coagulation studies may reveal significant abnormalities. Prothrombin time (PT), International Normalized Ratio (INR), and activated partial thromboplastin time (aPTT) can be prolonged. Fibrinogen levels may be depressed, along with an elevation of fibrin degradation products and D-dimers.[8] If resources are limited, a 20-minute whole blood clotting test can be used to diagnose coagulopathy. A few milliliters of blood are drawn and placed in a clean glass tube and allowed to sit, undisturbed, for 20 minutes. After this time, the tube is tipped. If the blood is still liquid, coagulopathy is present.[181] Major abnormalities may be seen in serum coagulation studies in the absence of any clinically significant bleeding (i.e., more serious than gingival oozing or microscopic hematuria).[12] This is particularly relevant for determining when to use blood products in treating these victims (see later discussion). Recurrent coagulopathic parameters may persist or recur for as long as 2 weeks after envenomation.[34]

Urinalysis results should be obtained to identify hematuria. Proteinuria and glycosuria may also be seen.[160] Each time the victim voids, the urine should be evaluated with bedside testing strips for the presence of blood.

If the envenomation seems severe or if the victim has significant underlying medical problems (e.g., cardiovascular or respiratory disease), an electrocardiogram (ECG), arterial blood gases (ABGs), and a chest radiograph should be obtained. The ECG may reveal evidence of myocardial ischemia. ABGs give important information regarding adequacy of tissue perfusion and respiratory status. Pulmonary vascular congestion or frank pulmonary edema may be seen on the radiograph.

Coral Snakes

The findings of significant coral snake envenoming reflect the predominant neurotoxic effects of these venoms. The victim may have some early, mild, transient pain.[154] Local swelling is uncommon. Fang marks may be difficult to see and should be carefully sought.[134] Systemic signs and symptoms may be delayed as long as 12 or 13 hours after significant bites and can then progress rapidly.[99] The earliest findings may be nausea and vomiting, followed by headache, abdominal pain, diaphoresis, and pallor.[48] Victims may complain of paresthesias or numbness or may have altered mental status, such as drowsiness or euphoria.[126] The victim may develop cranial nerve dysfunction (e.g., ptosis, difficulty speaking, difficulty swallowing), followed by peripheral motor nerve dysfunction.[126] In severe cases, respiratory insufficiency and aspiration are significant risks.[99] Cardiovascular insufficiency may also be seen.[160] Unlike with many crotaline envenomations, coagulopathy is not a feature of coral snake envenomation.

Laboratory studies are of little value in the evaluation of a victim of coral snake bite. Occasionally, a rise in serum CK and myoglobinuria occur, reflecting myotoxicity.[99] ABGs may be useful in evaluating the victim's respiratory status if endotracheal intubation is considered. A chest radiograph is indicated in the setting of apparent cardiac dysfunction or after intubation.

Management

Prehospital Care

Pit Vipers. The factors that most reduce snakebite-related injury and mortality in the United States are rapid transport, intensive care, and antivenom.[15] All patients with snakebites should be transported to the hospital as expeditiously and safely as possible, preferably through activating the emergency 911 system (where available). Increasing the area of coverage for cellular phone services also provides snakebite victims with easier access to ambulance or helicopter rescue (Fig. 48-26).

Attempts to secure or kill the snake are not recommended because of the risk of additional bites to the victim or rescuer, and because precious time can be wasted. All emergency per-

A

B

Figure 48-26. **A,** The fastest and safest way out of a deep wilderness setting may entail helicopter evacuation, where available. **B,** Specialized techniques, such as hoisting, may be required to rescue a snakebite victim out of a tenuous situation. (**A,** Courtesy Jeff Grange; **B,** Courtesy Carlos Quezada.)

sonnel should be able to distinguish a venomous from a nonvenomous snake (described previously). However, if the personnel are uncertain about whether a particular snake is venomous, photographs may be taken of the snake from a safe distance (at least 6 feet away) using a digital or Polaroid camera. These images can be seen immediately and may help make clinical decisions. Although it may be helpful to identify the species of snake,[16,18] transporting it (alive or dead) is discouraged because of inherent dangers. On scene, snakes should be moved or contained only if absolutely necessary (i.e., for safety). A snake hook or long shovel may be helpful to move a snake into a large, empty trash canister where it can be recovered by a professional, such as an animal control agent. Serious morbidity and even death have been reported after envenomation by decapitated rattlesnake heads.[27,98,173] In addition, an apparently dead snake or a decapitated snake head can have a bite reflex for at least 90 minutes after death. Therefore, emergency personnel and hospital care providers should exercise extreme care if handling any specimen accompanying the victim, even if it appears dead.

Recommendations for first-aid and prehospital treatment of pit viper envenomation have historically been based on speculation and anecdotal experience, although better evidence is accumulating in the literature. In one large retrospective series, first-aid treatment had no relationship to ultimate envenomation severity.[192] Some first-aid measures recommended in the past have caused more injury than the snakebite itself, and delays in care have been shown to increase morbidity and mortality.[45,71] It is inappropriate to use any technique that could potentially injure the patient or impede immediate travel to the nearest facility where antivenom can be administered. General support of the airway, breathing, and circulation should be provided, depending on the capabilities at hand. Oxygen, cardiac monitoring, and intravenous (IV) fluids should be used in the field when available. Although it may be necessary for the victim to hike out from the scene of the incident, activity should be minimized as much as possible. Alternative methods (e.g., stretcher, helicopter, boat) of extracting the victim from a wilderness setting can be used when available and when conditions such as weather and terrain allow. Jewelry and tight-fitting clothing are removed from the involved extremity in anticipation of swelling. The border of advancing edema is marked with a pen every 15 minutes so that emergency personnel can estimate the severity of poisoning by following the rate of progression. These measures suffice as adequate prehospital care for the vast majority of cases of pit viper bites in the United States.

The following measures are not recommended for first aid: incision, suction, tourniquets, electric shock, ice, alcohol, and folk therapies. Until recently, the Sawyer Extractor Pump (Sawyer Products, Safety Harbor, FL) was recommended. However, at least three studies, done independently of each other and using different methodologies, arrived at the same conclusion—that the Extractor does not work for venomous snakebite and could make things worse.[2,14,19,20] Mouth suction is contraindicated for the additional concern of potentially contaminating the wound with oral flora.

Incising the bite site across fang marks is contraindicated. This creates additional injury and has never been shown to be effective. Because viperid fangs are curved, incisions may miss the track along which venom is actually injected. Incisions made by laypersons can cause serious injury to underlying blood vessels, nerves, or tendons. Because of venom-induced coagulopathy, bleeding from such incisions can be severe.[73] Furthermore, the lack of sterile conditions in a field setting increases the risk of infection.

Venom sequestration techniques, such as application of a lymphatic or superficial venous constriction band or pressure immobilization, inhibit the systemic spread of venom.[13,174] It is not clear, however, whether such measures improve outcome after pit viper envenomation. Some argue that restricting the spread of potentially necrotizing venom to local tissues may intensify injury.[75] Because local sequelae are the predominant complications after pit viper envenomation, and because permanent systemic injury and death are rare, especially in North

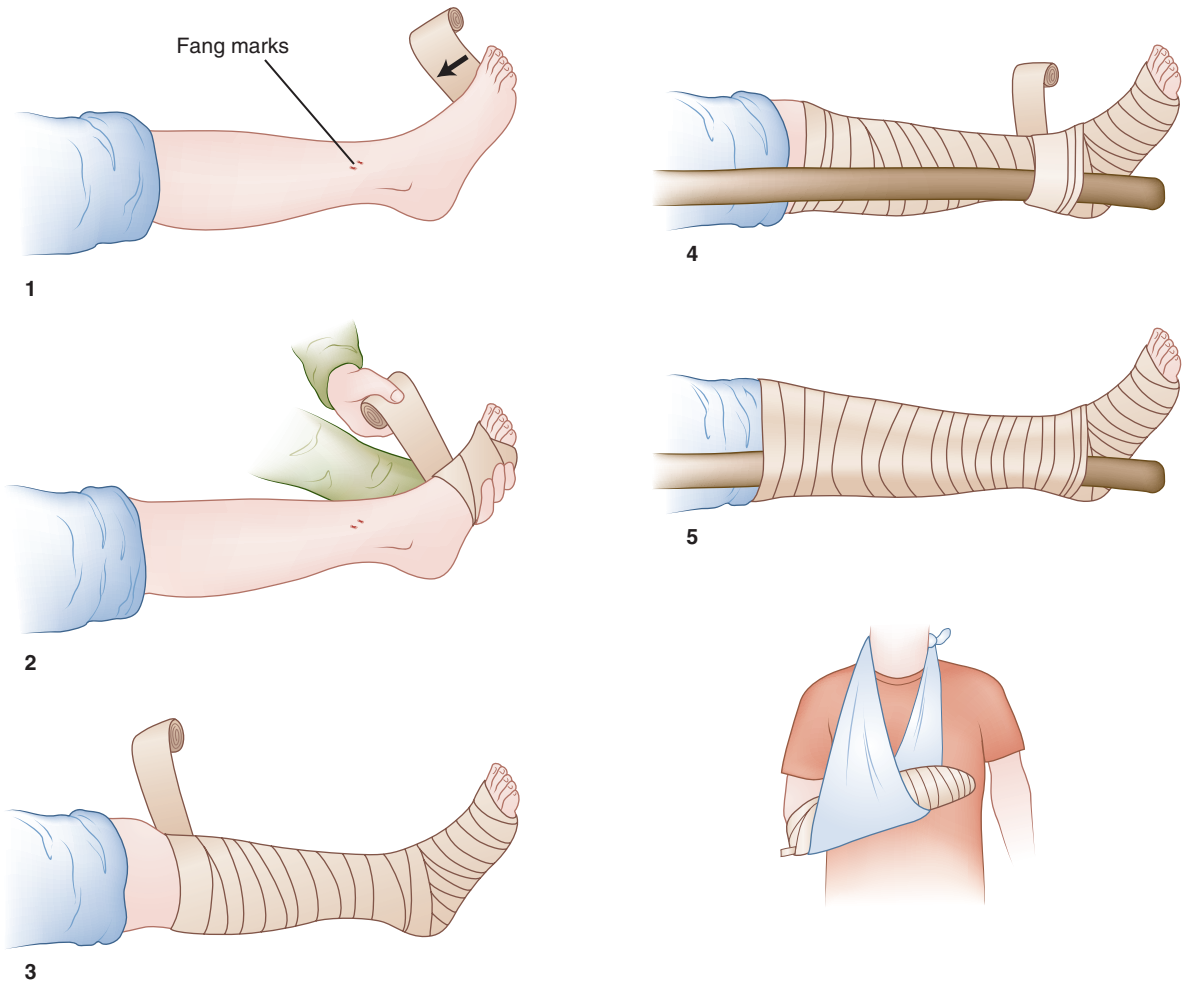


Figure 48-27. The Australian pressure-immobilization technique. This technique has proven effective in the management of elapid and sea snake envenomations. Its efficacy in viperid bites has yet to be fully evaluated clinically.

America, such attempts to limit venom to the bite site are ill-advised.[45] Tourniquets have worsened injury when used for snakebite and are contraindicated.[73]

Pressure immobilization has been used effectively in Australia for field management of elapid snakebites (see Chapter 49).[175] Pressure immobilization resulted in significantly longer survival, but higher intracompartmental pressures after artificial, intramuscular western diamondback rattlesnake *(C. atrox)* envenomation with a pig model.[17] This technique involves immediately wrapping the entire snakebitten extremity with an elastic ACE wrap or crepe bandage as tightly as would be done for a sprain, and then splinting and immobilizing the extremity (Fig. 48-27). Studies have shown that lay people as well as physicians have difficulty properly applying the bandage, generally underestimating the necessary tightness for effective application.[135] Furthermore, the victim must be carried out of the field following application of pressure immobilization, because any walking stimulates pumping of venom into the systemic circulation and negates any benefit even in upper extremity bites.[88] Although pressure immobilization is not recommended widely, certain scenarios may warrant its use. Although it is difficult to predict snakebite severity at the time of the bite, certain factors may reflect an increased likelihood of a more severe envenomation: large snake size, dangerous snake species, small patient size, prolonged fang contact, previous venomous snakebites (treated or not) or exposures to snakes, or delays to medical care and antivenom administration.[91] Individuals who consider applying pressure immobilization must assess risks versus benefits versus alternatives on a case-by-case basis. An informed decision should take into consideration the potential severity of the bite (see earlier). However, once pressure immobilization is placed after any snakebite, it should not be removed until antivenom is ready to infuse (if asymptomatic) or is infusing (if symptomatic) because of a potential bolus venom release after its removal.

With the exception of the Extractor Pump and pressure immobilization, all other field management recommendations are based primarily on theory and speculation, with very little science available with which to make an informed decision. This area is open for future research.

For the victim of a pit viper bite who is many hours or days from medical care, the best course of action may present a challenge and is highly dependent on the situation. If present, a companion may hike out for help, if conditions allow for the prompt return of a rescue team. At times, if sufficient rescuers are available, the victim can be carried out. The victim who must hike out should use a makeshift crutch (for a lower extremity bite), rest frequently, and maximize oral intake of fluids unless vomiting becomes pronounced.

Insufficient evidence exists regarding the efficacy of splinting or specific positioning (e.g., above or below the level of the heart) of the limb. Theoretically, elevation may hasten systemic spread of venom, whereas lowering of the extremity may worsen swelling. In the absence of any reliable data, it is recommended at this time that the extremity should be maintained in a neutral position of comfort until it can be ascertained whether the predominant effects of the bite will be systemic or local and therapy can be tailored appropriately.

Electrotherapy was proposed in the late 1980s for first-aid treatment of snakebites and subsequently was popularized by the lay press.[67,83] Early proponents recommended application of high-voltage, low-amperage, DC shocks to the bite site using a source such as an outboard motor or lawn mower engine.[66] A "stun gun," typically designed for self-defense, was even modified by one company and marketed for snakebite treatment.[73] This marketing was halted by the U.S. Food and Drug Administration (FDA) in 1990 because of total lack of testing or evidence of efficacy. In subsequent controlled animal studies, electric shock showed no efficacy in reducing morbidity or mortality after rattlesnake poisoning in mice, rats, or rabbits.[89,93,171] In humans, application of electric shock for snakebite has been associated with acute myocardial infarction and increased local tissue damage secondary to electrical burns.[42,44,156] Because of its lack of efficacy and inherent risks, electric shock should not be used in the treatment of snakebites.

Local application of ice to the bite wound as a first-aid measure has not been adequately studied as to its benefits or risks. This should not be confused with "cryotherapy," or packing the injured limb in ice for extended periods. This form of treatment was popularized in the 1950s and 1960s. Use of cryotherapy resulted in a significant increase in tissue loss and amputation rates after pit viper bites, and it has now been completely abandoned.[154] Whether brief (e.g., less than 1 hour) application of ice is beneficial (by reducing venom activity or decreasing pain) or harmful (by worsening local ischemia and resulting necrosis) is unknown. The American Red Cross recommends that no cooling measures should be applied in the field management of venomous snakebite.[184] In any case, ice should not be applied directly to the skin for any prolonged period.

Indigenous peoples in many parts of the world have long used plants in the treatment of snakebite, either topically or systemically, but little formal research has been done in this area. No current data support the use of any plants in the management of snakebite.[87]

Other first-aid measures lacking therapeutic value or potentially being more harmful than the snakebite itself include scarification of the bite wound, ingestion of alcohol, use of stimulants, and various folk remedies such as application of ammonia, silver nitrate, oil, potassium permanganate, or saliva. Similarly ineffective is the application of poultices made from various parts of the offending reptile (or other creatures), such as the snake's crushed head, bile, or fat and the use of snake stones.[155]

Antivenom use in the field can be recommended only when a qualified medical provider is on scene and when all equipment and drugs are available to manage a potential anaphylactoid reaction to the antiserum (including definitive airway management equipment). Studies and clinical experience show that newer, purified Fab-fragment antivenoms may be safer than whole immunoglobulins (see later). Adverse reactions still occur with these new-generation antivenoms but are less frequent and less severe than reported with previous preparations. With the safety profile of these new antivenoms, field use may be worth reconsidering. Antivenom has been shown to retain activity for at least 60 days under conditions of increased heat and motion (as might be encountered in a field setting). However, monetary costs can be prohibitive.

Coral Snakes. When a bite by a coral snake is suspected, the animal's skin color pattern should be noted if possible. Because of the potential delay in onset of signs and symptoms after coral snake envenoming, and because the recommendation is to administer antivenom to such victims even in the absence of clinical findings, distinguishing between a coral snake and a harmless mimic is important. It may be helpful if the snake can be quickly and safely captured and contained or photographed.

Recent research suggests that the Australian pressure-immobilization technique may be useful in the field management of coral snake bites. This technique has been shown to be effective in one small-animal study using *Micurus fulvius* venom.[55] Being elapids, coral snakes are related to the venomous snakes in Australia, for which this technique has been shown to be highly effective in limiting venom absorption (see Chapter 49).

Hospital Care

Pit Vipers. Emergent medical care after a pit viper bite (Box 48-1) must initially focus on the victim's airway, breathing, and circulation. Most victims should receive supplemental oxygen until it is clear that they are stable. Patients with bites to the face or neck may require early intubation to prevent loss of the airway caused by severe swelling. Pulse oximetry and cardiac monitoring should be instituted and two large-bore IV lines established. The initial management of hypotension or shock should include vigorous fluid resuscitation with crystalloids (normal saline or Ringer's lactate). If organ perfusion remains inadequate after vigorous fluid infusion (e.g., of 2 L of crystalloid in an adult, 20 to 40 mL/kg in a child), a trial infusion of albumin should be started. Evidence supports the utility of adding albumin early in this setting because of the rapid onset of increased vascular permeability after significant pit viper envenomation.[163] Vasopressors should be used to treat venom-induced shock only as a last resort, after adequate volume infusion and initiation of antivenom therapy. Prolonged, inadequately treated hypotension has been strongly implicated in fatal cases of envenomation.[45,75]

Antivenom. The mainstay of therapy for pit viper envenomation is the judicious use of antivenom (Table 48-5). Antivenom therapy is predicated on imparting passive immunity to the victim against circulating snake venom antigens. Once these deleterious antigens bind to their target tissues, antivenom is unable to pull them off or reverse their effects. Therefore, to be

Box 48-1. Management Principles in Treating a Victim of Pit Viper Bite

PRELIMINARY MEASURES
- Determine: Was the snake venomous?
- Determine: Is there evidence that venom poisoning has occurred?
- Assess and support the airway, breathing, and circulation.
- Establish two large-bore intravenous lines for crystalloid infusion.
- Obtain appropriate laboratory studies (see text).
- Measure the bitten limb's circumference at two or more sites every 15 minutes.
- If significant local findings are progressing, if systemic signs or symptoms are present, or if laboratory abnormalities are present, obtain and reconstitute an appropriate antivenom.
- Obtain informed consent for antivenom infusion if possible.
- Expand the patient's intravascular volume with crystalloid fluids unless there is a contraindication to doing so.
- Have epinephrine (an appropriate subcutaneous dose) drawn up at the patient's bedside for use in the event of an acute reaction to antivenom.

CroFab (See Table 48-5 for Severity Grading Guide)
- No skin test for possible allergy.
- No pretreatment with antihistamines required
- Starting dose:
  - Nonenvenomation: 0 vials
  - Mild envenomation: 4–6 vials
  - Moderate envenomation: 4–6 vials
  - Severe envenomation: 6+ vials
- Dilute the vials of antivenom to be given in crystalloid (250 mL; 20–40 mL/kg for a child, up to 250 mL).

Antivenom (Crotalidae) Polyvalent (ACP) (See Table 48-5 for Severity Grading Guide)
- No skin test for possible allergy (ineffective).
- Pretreat:
  - H_1- and H_2-blocking antihistamines
- Starting dose:
  - Nonenvenomation: 0 vials
  - Mild envenomation: 0 or 5 vials
  - Moderate envenomation: 10 vials
  - Severe envenomation: 15 vials
- Dilute the vials of antivenom to be given in crystalloid (1 L for an adult; 20–40 mL/kg for a child).

Other Antivenoms for Snakes of the Western Hemisphere
- Follow the directions in the package inserts, which may require translation.

First Phase of Antivenom Administration
- Begin administering antivenom by slow intravenous infusion with physician at the bedside.
- Gradually increase the rate of infusion (in the absence of an acute reaction) to accomplish the total dosage over a period of 1 hour (CroFab) or in 1–2 hours (ACP).
- If a reaction occurs, stop the antivenom and treat as necessary.
- Consider the need for further antivenom:
  - Further antivenom to be withheld: conservative care only
  - Further antivenom to be given: dilute the antiserum further, administer intravenous steroids, and restart the antivenom at a slower rate. If the reaction occurred to ACP, consider switching to CroFab (if available).

Second Phase: Continued Reaction in the Setting of Severe Poisoning
- Admit to the ICU.
- Institute maximal monitoring as needed (e.g., arterial line if no evidence of coagulopathy).
- Consult an expert in snake envenomation.
- Employ epinephrine drip titrated against the reaction.

FURTHER DOSING OF ANTIVENOM

CroFab
- If signs, symptoms, or laboratory abnormalities continue to worsen in the first hour after antivenom infusion, repeat the starting dose.
- Once stabilized, give two additional vials every 6 hours for three more doses. (Alternatively, the patient can be observed closely for recurrence of venom toxicity and further antivenom given as needed.)

ACP
- If signs, symptoms, or laboratory abnormalities progress after the starting dose, more antivenom can be infused in increments of 5 to 10 vials every 30 minutes to 2 hours as needed.

WOUND CARE (DESCRIBED IN TEXT)

DISPOSITION
- Apparent dry bite: admit or observe for a minimum of 8 hours
- Any degree of envenomation: admit
- If severe venom poisoning, admit to ICU

most effective, antivenom should be given as soon as possible after the bite, preferably within 4 to 6 hours.[101] Antivenom may be useful, however, in patients with significant coagulopathy or acute renal failure for days after envenomation.[158] Venom antigens have been detected as late as 13 days after venomous snakebite.[56] The clinical implications of prolonged, recurrent, isolated, and mild coagulopathy without evidence of bleeding and the utility of late antivenom administration remain unresolved. No clear end points for antivenom's therapeutic window have been established.

Two antivenoms are currently available in the United States for use in pit viper bites, CroFab (Savage Laboratories, Melville, NY) and Antivenom (Crotalidae) Polyvalent (ACP) (Wyeth-Ayerst Laboratories, Philadelphia, PA). Both products are

TABLE 48-5. Snake Antivenoms Available in the United States

ANTIVENOM [SOURCE]	MANUFACTURER	VENOMS USED	COMPOSITION	STARTING DOSE	FURTHER DOSES	OVERALL INCIDENCES OF REACTIONS	APPROXIMATE ACQUISITION COST
CroFab [Ovine]	Savage Laboratories, Melville, NY	*Crotalus adamanteus, Crotalus atrox, Crotalus scutulatus scutulatus, Agkistrodon piscivorus piscivorus*	Fab fragments; Fc fragments (<3%)	Dry: none Mild: 4–6 vials Moderate: 4–6 vials Severe: 6 vials	Starting dose should be repeated if signs, symptoms, or laboratory values fail to stabilize in the first hour after the initial dose. Once stabilized, the patient should receive 2 additional vials every 6 hours for 3 additional doses. Alternatively, the patient can be observed closely for the first 24 hours and given further antivenom if findings recur.	Acute allergic reaction: 14.3%* Serum sickness: 3%*	$2000 (per 2-vial box)
Antivenom (Crotalidae) Polyvalent [Equine]	Wyeth-Ayerst Laboratories, Philadelphia, PA	*Crotalus adamanteus, Crotalus atrox, Crotalus durissus terrificus, Bothrops atrox*	Whole IgG(T), equine albumin	Dry: none Mild: 0 or 5 vials Moderate: 10 vials Severe: 15–20 vials	Further antivenom is given if signs, symptoms, or laboratory values fail to stabilize or worsen following the initial dose.	Acute allergic reaction: 23%–56%† Serum sickness: 18%–86%†	$900 per vial
Antivenom (*Micrurus fulvius*) [Equine]	Wyeth-Ayerst Laboratories, Philadelphia, PA	*Micrurus fulvius*	Whole IgG(T), equine albumin	3–6 vials	Further antivenom is given if signs or symptoms progress after the initial dose—3-5 additional vials (rarely are more than 10 vials required). If the victim has been bitten by a positively identified *Micrurus* species, treatment with antivenom should be started regardless of whether evidence of venom poisoning is present.	Acute allergic reaction: 35%‡ Serum sickness: 10%‡	$1300 per vial

*From Reference 44.
†From Reference 63.
‡From Reference 99.

produced by injecting animals with venoms of four different species of pit vipers, and they provide adequate antigenic coverage to be effective in managing envenomation by any North American pit viper species (and many Central and South American species). Current experience and research indicate that CroFab is more effective for most North American crotalid species and safer to use than ACP.[86,96,167] Drawbacks to its use include its significant cost and short half-life, which requires, in many cases, repeat dosing at regular intervals during the initial stages of management (see later).[165] Other manufacturers of antivenoms effective for snakes of Latin America can be located using resources such as regional poison control centers, textbooks, or the internet.[181]

CroFab. CroFab has been available for commercial use since the year 2000. It is produced by injecting sheep with the venom of one of four species of pit viper: the eastern diamondback rattlesnake *(C. adamanteus),* the western diamondback rattlesnake *(C. atrox),* venom A–producing Mojave rattlesnakes *(C. scutulatus scutulatus),* and the eastern cottonmouth water moccasin *(Agkistrodon piscivorus piscivorus).* Serum from immunized sheep is harvested, and the IgG molecules are papain digested into Fc and Fab fragments. The immunogenic Fc fragments are precipitated and discarded. The protective Fab portions are then isolated by affinity column chromatography to the venom components against which they are active. These fragments are subsequently desorbed from the column and combined in equal parts from the four groups to yield a polyspecific Fab pit viper antivenom.[35,47]

It appears that CroFab causes fewer acute, anaphylactoid reactions than does ACP, an equine-derived product. Unlike equine IgG(T), ovine IgG is not glycosylated and therefore is less immunogenic. In addition, Fab fragments, with their single antigen binding site, are incapable of cross-linking immune complexes and stimulating the cascade of mediator release operant in anaphylaxis.[96] Immunologic response is also less likely because the immunogenic Fc portion is eliminated and the molecule is smaller.[167] Another potential advantage to CroFab, with its smaller molecular size, is that it is more rapidly cleared by the kidneys and thus may induce less serum sickness, which results from immune complex deposition in tissues. A smaller molecule may also allow improved tissue penetration and better access to injected venom.[86]

In animal models, CroFab has been shown to be 3 to 10 times more potent than ACP in protecting mice against lethal effects of venom from various North American pit vipers.[35] In clinical trials, the initial dosage of CroFab ranged from 4 to 12 vials, with repeated doses of 2 to 6 vials given for recurrence or progression of swelling or systemic effects.[34,47,165] Venom effects may recur after initial treatment because Fab fragments are cleared before all venom antigens are neutralized.[165]

According to the manufacturer, CroFab is indicated for use within 6 hours for mild and moderate North American pit viper envenomation,[36] although it is being used successfully in severe cases as well. It also appears effective in reducing local findings and systemic abnormalities when used in more delayed fashion,[5] although its benefit in limiting local necrosis remains to be established. There is evidence that this antivenom may also be effective against some South American pit viper venoms.[150] No skin test is recommended and no premedication is required, but appropriate medications (epinephrine and antihistamines) and equipment should be immediately available to treat an acute reaction if needed. The starting dose is 4 to 6 reconstituted vials, further diluted in 250 mL of normal saline.[36] Administration should be started slowly for the first several minutes while the patient is observed closely for any signs of adverse reaction. The rate is then increased to achieve the initial dose administered in 1 hour. The victim should then be observed over the course of an additional hour for evidence of control of venom effects. If there is evidence of progression of local findings or if coagulation studies and systemic signs and symptoms fail to improve, the starting dose should be repeated until such stabilization occurs. After stabilization, 2 vials should be given every 6 hours for three more doses. Further administration after that time should be based on the clinical condition of the patient. There is evidence that patients with significant coagulopathies during the initial phase of their poisoning may have recurrence of these findings during the first 2 weeks after the bite.[7,36] The clinical significance of these findings and the need for additional antivenom remain to be determined. If, after the acute phase of envenomation has been controlled, clinical coagulopathy recurs in the absence of any evidence of bleeding, it is likely that further antivenom administration may be unnecessary. Patients should be warned, however, of the potential recurrence of coagulopathy and the need to return if there is any evidence of bleeding. They should also be cautioned to avoid high-risk activities, such as contact sports or mountain climbing, and elective surgical procedures for 2 to 3 weeks following their bite.

Antivenom (Crotalidae) Polyvalent (ACP). ACP has been used for the management of pit viper bites in the United States since 1954. It is derived from the serum of horses immunized to the venoms of two North American and two South American pit vipers: the eastern diamondback rattlesnake *(C. adamanteus),* western diamondback rattlesnake *(C. atrox),* South American rattlesnake *(C. durissus terrificus),* and fer-de-lance *(Bothrops atrox).* Like CroFab, this antivenom can be used for bites by any North American pit viper. It is also effective for Central and South American crotalines and some Asian pit vipers. The degree of protection, however, varies depending on the species involved. There has been, for example, concern over its ability to protect adequately against the bite of venom A–producing Mojave rattlesnakes *(C. scutulatus scutulatus),*[34,161] because Mojave toxin is not found in appreciable quantities in the venoms used in its production.

ACP dosing is guided by the clinical severity of envenomation. No antiserum is necessary in dry bites, and it is questionable whether the risks associated with the use of ACP justify its use in mild envenomation. Some authors recommend a starting dose of 5 vials in mild cases.[155] For moderate poisoning, the initial dose should be 10 vials, and for a severe case, 15 to 20 vials. If symptoms, signs, or laboratory parameters continue to deteriorate after the initial dose of antivenom, further dosing in 5- to 10-vial increments every 30 minutes to 2 hours is indicated. A typical total dose of ACP for a serious rattlesnake envenomation is approximately 20 to 40 vials.

Severity Grading and Dosage of Antivenom. Decisions about initial dosing of antivenom in cases of venomous snakebite are based on the apparent or predicted severity of poisoning. The grading scale described in Table 48-6 is useful for determining the severity of North American pit viper bites, but it should not be applied to bites by noncrotaline or exotic species. A more

TABLE 48-6. Clinical Severity Grading Scale for Pit Viper Bites in North America

SEVERITY	LOCAL FINDINGS		SYSTEMIC SIGNS AND SYMPTOMS		LABORATORY STUDIES
Nonenvenomation (dry bite)	Puncture wounds only	*and*	None	*and*	Normal
Minimal envenomation	Puncture wounds Swelling limited to bite site Bloody ooze from bite site Local ecchymosis Local pain (may be severe)	*and*	None	*and*	Normal
Moderate envenomation	As for minimal, but swelling may involve entire extremity Severe pain	*and*	Mild (e.g., nausea, vomiting, general weakness, mildly abnormal vital signs)	*or*	Mildly abnormal (e.g., slightly decreased platelet count or fibrinogen; presence of fibrin degradation products; slightly increased PT/PTT; hemoconcentration)
Severe envenomation	May be severe (e.g., severe soft tissue swelling and pain) May be less severe than expected (especially if deep or intravenous deposition of venom)	*and*	Very abnormal vital signs Shock Respiratory distress Significant clinical bleeding	*or*	Profoundly abnormal (e.g., coagulopathy; hemolysis/severe anemia; renal dysfunction)

detailed snakebite severity score was developed as a research tool but is not intended for clinical use.[43]

In the past, decisions as to when to begin antivenom therapy for pit viper bites in North America could be challenging, especially in the early stages of the clinical course. The potential benefits of using ACP had to be weighed against the possible adverse reactions that could occur. Today, with the availability of an apparently safer antivenom, CroFab,[35,47] the decision is more easily made. CroFab is indicated for any patient bitten by a North American pit viper who has findings of progressive local envenomation or any evidence of systemic toxicity.

Antivenom should certainly be given for any serious envenomation (e.g., when there is cardiovascular insufficiency, respiratory difficulty) or when there is risk of an acute complication (e.g., life-threatening bleeding, tissue necrosis, compartment syndrome, rhabdomyolysis). Likewise, antivenom should be started or additional vials given to a victim with a worsening clinical course.

The grade of envenomation at any given time is determined by whichever parameter (local effects, systemic effects, or laboratory abnormalities) is most severely affected. Snake envenomation is, however, a dynamic process, and severity grade can increase suddenly and progressively.

In nonenvenomations, or dry bites, fang marks are present but there are no other clinical signs or symptoms and no laboratory abnormalities. The victim has only superficial punctures that require standard wound care, including tetanus prophylaxis as necessary. Antivenom is not indicated in this scenario.

Minimal envenomation results in effects confined to the immediate area around the bite site (pain, swelling, erythema, ecchymosis). Systemic effects are absent and laboratory values are normal.

In a moderate envenomation, local effects progress beyond the immediate bite area but involve less than the entire body part (arm or leg). Systemic effects may be present and may include paresthesias, strange taste sensations, numbness of the mouth and tongue, muscle fasciculations (myokymia), generalized weakness, or mild hypotension. Along with systemic signs and symptoms, laboratory abnormalities may be present. Coagulation times (e.g., PT, aPTT, INR) may be elevated, platelets may drop to less than 90,000/mm^3, fibrinogen may fall below 90 mg/dL, and CK levels may exceed 500 to 1000 U/L. Although evidence is limited that antivenom corrects venom-induced thrombocytopenia, rhabdomyolysis, or myokymia,[8,34] these abnormalities serve as markers of systemic injury and the need for antivenom therapy.

With severe envenomation, local effects may rapidly involve the entire body part and may become limb threatening. Systemic manifestations may include shock, clinically significant bleeding (e.g., hematemesis, central nervous system bleeding), respiratory difficulty, and multifactorial renal failure. Severe muscle fasciculations may contribute to respiratory difficulty. Profound laboratory abnormalities, such as thrombocytopenia less than 20,000/mm^3, coagulopathic parameters associated with potentially life-threatening bleeding, or rhabdomyolysis or myoglobinuric renal failure, also indicate severe envenomation. If venom injection has been deep intramuscularly or intravascularly, systemic effects may become severe very quickly, out of proportion to local findings.

Antivenom Administration. Most antivenoms (including both U.S. products) are supplied in lyophilized form and require reconstitution before administration. This can be an onerous task. The contents of each vial must be dissolved in 10 mL of diluent (e.g., sterile water, normal saline). Using warm diluent helps facilitate reconstitution. Gentle agitation of each vial for several minutes is required to get the proteins into solution. Care must be taken, however, to not denature the antiserum with overzealous shaking or by using diluent that is too hot. Reconstitution should begin as soon as it is apparent that antivenom therapy will be necessary, as the process can take

approximately 40 minutes for CroFab and 90 minutes or longer for ACP.[84]

The risks of adverse reactions (see later) to antivenom must be weighed against the potential benefit of improved mortality and morbidity from its administration. Informed consent should be obtained whenever possible prior to administration. Specific risk factors for reactions to antivenom (e.g., previous exposure to equine serum, history of allergy to horses [for ACP], sheep, wool, or papaya [for CroFab]) should be considered when deciding on a course of therapy. Any time antivenom is given, an acute adverse reaction should be anticipated and preparations made to manage a life-threatening situation. Airway equipment must be immediately available and two functional IV lines secured.

In a hospital setting, snake antivenoms should be administered only intravenously, preferably in an unbitten extremity. There is no benefit to administering antivenom locally.

Skin Testing and Pretreatment. Wyeth-Ayerst recommends a skin test for possible allergy to equine-derived products before administration of ACP. Such testing, however, is very unreliable. As many as 10% to 28% of persons with a negative skin test still manifest an acute reaction to ACP, and up to 30% with a positive skin test have no reaction if the product is still given.[94,169] Furthermore, people can develop an acute reaction or delayed serum sickness to the skin test alone.[169] For all these reasons, skin testing should be omitted. No skin test is required or recommended by the manufacturer of CroFab.

Before beginning ACP infusion, the patient can be premedicated with standard doses of antihistamines. Both an H_1 blocker (e.g., diphenhydramine, 1 mg/kg) and an H_2 blocker (e.g., cimetidine, 5 mg/kg up to 300 mg) may be given intravenously in hopes of preventing or mitigating an acute reaction. There is, however, no concrete evidence that such pretreatment is beneficial. Pretreatment is not recommended by the manufacturer of CroFab, but it can be considered if the patient appears to be at increased risk of developing an acute reaction (see earlier). Expanding the victim's intravascular volume with an appropriate bolus of crystalloid (e.g., 1 to 2 L in an adult, 20 to 40 mL/kg in a child) may also prevent or blunt acute reactions.[4]

If the risk of an acute reaction is exceptionally high, a prophylactic dose of epinephrine might be considered as well. Epinephrine has been shown to reduce the incidence of acute reactions to antivenom. Subcutaneous administration of 0.25 mL of epinephrine (1:1000 aqueous) reduced acute adverse reactions from 43% to 11% in one randomized, controlled, prospective series of 105 patients.[146] No patients pretreated with epinephrine had severe reactions to antivenom, whereas 8% of patients who did not receive epinephrine had reactions. Prophylactic epinephrine must be used with caution in persons at risk for ischemic heart disease or stroke. If coagulopathy is present, prophylactic epinephrine should be avoided because of the risk of elevating blood pressure and causing intracranial bleeding.[188] The short half-life of epinephrine might be expected to limit potential benefits from its prophylactic use.

Routine premedication with corticosteroids is not common practice in the United States; it is more common in Australia. Because the beneficial effects of steroids in limiting allergic phenomena would not be present for 4 to 6 hours after administration, and because steroids may worsen local effects of envenomation by North American pit vipers,[166] steroids are generally not given unless an acute allergic reaction has occurred.

Once the vials of antivenom are dissolved, each should be further diluted before administration. Some early reactions to antivenom may be related to the concentration of the product that is given intravenously[4]; therefore, antivenom should be given in a relatively dilute form whenever possible. For adults without preexisting cardiac insufficiency, the initial total dose of ACP can be placed in a liter of normal saline. For children, the total starting dose should be placed in a volume equivalent to 20 to 40 mL of crystalloid fluid per kilogram bodyweight up to 1 L. For CroFab, the starting dose can be placed in 250 mL of diluent.

The infusion should be started at a slow rate (e.g., 1 mL/min) for the first 10 to 15 minutes, with the attending physician monitoring for signs of acute reaction. If no adverse reaction occurs, the rate is increased to complete the infusion over 1 to 2 hours for ACP and 1 hour for CroFab.

If an acute reaction develops, the antivenom infusion should be stopped immediately and the reaction treated. This may require administration of epinephrine, further antihistamines (both H_1 and H_2 blockers), and steroids. Appropriate ventilatory and circulatory support should be instituted as needed. Once these interventions have stopped the reaction, the physician must determine if antivenom therapy is still indicated. In most cases, antivenom infusion can be restarted, after further dilution of the antiserum (twofold if the patient's cardiovascular reserve will tolerate the increased volume) and at a slower rate. If the reaction was serious or persists after restarting the antivenom, and if the envenomation is life-threatening, the patient should be placed in an intensive care setting with maximal cardiovascular monitoring. Invasive monitoring (e.g., arterial line access) is helpful as long as no coagulopathy would make such line placement risky. An epinephrine IV infusion should be established (starting at 0.1 µg/kg/min) and titrated to hold the anaphylactic reaction at bay as further antivenom is infused. Generally, enough antivenom can be administered using this technique to benefit the patient's outcome.[21,121] Careful reevaluation of the need for antivenom should be made before embarking on this hazardous approach. It is advisable to consult with a specialist who manages difficult envenomations. If the reaction occurred with the use of ACP, a change to CroFab should be made if it is available.

Adverse Reactions to Antivenom. As heterologous serum products, all antivenoms carry some risks with their use. Early, acute reactions to antivenom can be either anaphylactic (type I, IgE mediated) or, more commonly, anaphylactoid (due to direct complement activation).[192] These immediate reactions are clinically indistinguishable and may manifest with urticaria, pruritus, flushing, facial edema, vomiting, diarrhea, crampy abdominal pain, bronchospasm, laryngeal edema, and hypotension. Also important is the risk of delayed serum sickness after antivenom use. Serum sickness (a type III, IgM- and IgG-mediated hypersensitivity reaction) is characterized by rash, fever, chills, arthralgias, lymphadenopathy, and malaise, and it occurs anywhere from 3 days to 3 weeks after antivenom administration. In severe cases, there may be renal or peripheral nerve involvement.

Approximately 23% to 56% of patients who receive Wyeth's ACP can be expected to have an acute reaction, and about half of these are serious.[44,63,94] Deaths from allergic reactions to this

product (resulting from airway obstruction or cardiovascular collapse) have occurred but are rare.[94,157] Serum sickness occurs in as many as 18% to 86% of patients who receive ACP.[63] The likelihood of serum sickness increases with total antivenom dosage, and it occurs more often than not when 8 or more vials of ACP are required.[94]

In the first 42 patients treated with CroFab, there were seven acute reactions (five with "urticaria," one "cough," and one "allergic reaction" manifested by urticaria, dyspnea, and wheezing).[36] There were also five late serum reactions (two with "rash," one with "pruritus," one with "urticaria," and one "serum sickness" manifested by severe rash and pruritus).[36] Data currently suggest an incidence of approximately 14% for acute reactions to CroFab and approximately 3% for serum sickness.[63] No anaphylactoid deaths have been reported at the time of this writing with the use of CroFab.

Monitoring Therapy. Antivenom administration should continue until the patient is stabilized, which may be indicated by reduced swelling, subjective systemic improvement, and stabilization or normalization of vital signs and laboratory values. Muscle fasciculations should not be used as a guide for continued administration of Wyeth's ACP, because they may be refractory to this therapy. Preliminary reports, however, suggest that myokymia improves with administration of CroFab.[34] Neurotoxicity has been documented to improve with antivenom administration, even with delayed administration.[21,34]

The efficacy of antivenom in treating venom-induced coagulopathy and thrombocytopenia is not established. Although ACP binds to venom antigens responsible for these effects, abnormalities in hemostatic parameters may persist or recur.[8,165] In several reports, venom-induced thrombocytopenia improved after antivenom infusion.[12,46,151] Other reports contradict these findings.[147,177] In a series of patients envenomed by timber rattlesnakes *(C. horridus),* PT and PTT, but not thrombocytopenia, normalized after antivenom therapy.[8] In another series, rattlesnake venom–induced thrombocytopenia usually improved after antivenom infusion, although the degree of improvement was small, was frequently incomplete, and did not appear to be related to the total dose of antivenom given.[23] Seifert and colleagues[165] reported investigational use of Fab antivenom (CroFab) in a thrombocytopenic victim of western diamondback rattlesnake *(C. atrox)* envenomation. Although the platelet counts rebounded from 12,000/mm^3 to 227,000/mm^3 within 1 hour, recurrent thrombocytopenia was documented from day 5 through day 10, possibly because of more rapid clearance of Fab fragments than of venom antigens. Thus, regardless of the antivenom preparation chosen, recurrent coagulopathy and thrombocytopenia should be anticipated. Coagulation studies should be repeated every 6 hours until stable.

Special Bite Situations.

Mojave Rattlesnake Bites: Systemic findings may be delayed after bites by venom A–producing Mojave rattlesnakes *(C. scutulatus),* with deceptively minimal local tissue effects. This can lead to an underestimation of severity based on the grading scale in Table 48-6. Therefore, in geographic areas where this variant is found, some recommend that antivenom be started empirically for known Mojave bites, regardless of the initial apparent severity of the bite.[16,192] ACP provides relatively poor coverage for *C. scutulatus* venom containing Mojave toxin, which supports this recommendation.[161] To compensate for reduced overall efficacy in these cases, Wyeth's ACP is usually given early and in larger doses than for most other rattlesnake bites. CroFab would probably provide better protection against Mojave toxin and other phospholipase A_2 neurotoxins.

Copperhead Bites: Copperheads *(A. contortrix)* and cottonmouths *(A. piscivorus)* generally cause less severe clinical effects than do most rattlesnakes. Many *Agkistrodon* envenomations can be treated without ACP, especially *A. contortrix*.[43,190] Severe envenomation, however, regardless of the crotaline species involved, requires antivenom therapy. Because *A. piscivorus* venom is used in the production of CroFab, it is likely that this product is very effective for *Agkistrodon* bites.

Bites in Children: Although few studies have compared children with adults in terms of the effects of crotaline envenomation, relatively higher doses of antivenom may be needed in pediatric victims. Children may receive a higher dose of venom per kilogram of bodyweight, which may predispose them to greater toxicity. The starting dose (number of vials) in children should be the same as in adults. With adequate antivenom administration, however, children do not appear any more susceptible to systemic venom effects than are adults.[45]

Bites in Pregnant Women: As is usual for all disease states that can occur in pregnant women, the management approach that optimizes fetal outcome is the one that best supports the mother. Fortunately, snake bites are rare in pregnant women. The potential effects of snake venom on the fetus have not been well studied, although fetal malformation has been described.[123] Preterm labor and abruptio placentae have also been reported after pit viper envenomation.[137,195] The anticoagulant and proteolytic actions in most crotaline venoms are probably responsible for disrupting the integrity of the placental attachment to the uterus. To inhibit these systemic venom effects, antivenom administration is important, even though all U.S. antivenoms carry an FDA Category C designation for safety in pregnancy (i.e., "uncertain safety—animal studies show an adverse effect, no human studies"). Informed consent may be even more important to obtain, if possible, in this situation. Standard doses of antivenom should be used, although the severity rating should be liberally upgraded in pregnant patients.

If an acute allergic reaction develops during antivenom administration, antivenom should be temporarily stopped. Epinephrine should be avoided if possible because of its adverse effects on uterine blood flow. Instead, ephedrine should be given at a dose of 5 to 25 mg by slow IV push.[139] This dose can be repeated every 5 to 10 minutes as needed (maximum: 150 mg/24 hr). Other drugs (e.g., antihistamines, steroids) should be given as for the nonpregnant patient. Antivenom administration can usually be restarted in a more dilute concentration and at a slower rate. If the reaction was to ACP, a switch to CroFab, if it is available, should be considered. Additional consultation is advisable.

Other management principles for snakebitten pregnant women include early obstetric consultation, fetal monitoring, and early ultrasonography for fetal or placental assessment.

Blood Products. In the rare situation of clinically significant bleeding after pit viper envenomation (e.g., gastrointestinal bleeding, central nervous system hemorrhage), transfusion of

blood products, including platelets, coagulation factors, or packed RBCs, may be necessary. As venom effects can interfere with blood typing and cross matching, it is important that a specimen be drawn for the blood bank as soon as the patient presents.[189] Antivenom should always be started before beginning blood product replacement to avoid adding further fuel to an ongoing consumptive coagulopathy. Packed RBCs should be considered for acute, severe anemia (e.g., hemoglobin less than 7 g/dL). Solvent- or detergent-treated plasma or fresh-frozen plasma may be needed to replace depleted coagulation factors if bleeding is significant, and cryoprecipitate may be added as needed for fibrinogen levels less than 100 mg/dL.[12] Regardless of the presence or absence of bleeding, platelet transfusion should be considered for a count of less than 20,000/mm^3. Any improvement in platelet count after transfusion, however, may be temporary.[8,177] Patients with dangerously low platelet counts should be restricted to bed rest to lower the risk of bleeding until their counts rebound.

Analgesia and Wound Care. Pain control can be a significant issue after pit viper envenomation. Analgesia is best obtained using appropriate, titrated doses of opiates (e.g., IV morphine sulfate, 2 to 10 mg in adults and 0.1 mg/kg in children; repeated when needed as vital signs allow). Aspirin and nonsteroidal anti-inflammatory drugs should be avoided, as they may exacerbate coagulopathy.

Wound care for pit viper envenomation follows standard principles. Tetanus immunization (diphtheria-tetanus toxoid, 0.5 mL intramuscularly) is recommended if the patient's immunization is not up to date. Any wounds should be cleaned and the extremity placed in a well-padded splint with additional padding between the digits. The extremity is then elevated above the heart to reduce swelling. Antivenom, if indicated, should be started before elevating the limb.

Although snakes' mouths are colonized with several potentially pathogenic organisms, such as *Pseudomonas aeruginosa, Staphylococcus epidermidis, Enterobacteriaceae* species, and *Clostridium* species, wound infections from bites are uncommon in North America, and prophylactic antibiotics are unnecessary in most cases.[33,186] In South America, however, as many as 10% to 15% of patients bitten by *Bothrops* species develop secondary infection.[181] Pit viper venom has antibacterial activity, which may account in part for the low overall incidence of wound infections.[176] If misdirected first-aid efforts included incisions into the bite wound or mouth suction, an appropriate broad-spectrum antibiotic (e.g., amoxicillin/clavulanate) should be considered. If secondary infection occurs, appropriate aerobic and anaerobic wound cultures (and possibly blood cultures, depending on the clinical situation) should be obtained. Empiric therapy should then be started (e.g., ciprofloxacin, 500 mg orally twice a day in adults). If anaerobes are suspected, metronidazole or clindamycin in appropriate doses can be added. Children and pregnant women can be treated with an initial dose of ceftriaxone (50 mg/kg up to 1 g IV or intramuscularly) followed by oral amoxicillin/clavulanate (40 mg/kg divided three times a day). Changes in antibiotic choice can be made once culture and sensitivity results are available. Abscesses should be drained if they develop. An infected snakebite should prompt further examination of the wounds for potentially retained teeth or fangs. Radiographs may be helpful but are not particularly sensitive. Rare cases of necrotizing fasciitis have been reported after venomous snakebite.[3]

Intact blisters should be protected as much as possible from rupture (Figs. 48-28 and 48-29). Any debridement of clearly necrotic tissue or ruptured blebs and bullae should be delayed for 3 to 5 days and should occur only in the absence of ongoing coagulopathy. Standard wound care can follow. The patient should begin a physical therapy regimen as soon as possible to expedite return of function.

Although the fangs of many pit vipers are long enough to penetrate muscle compartments, most crotaline bites result in subcutaneous deposition of venom.[155] Compartment syndrome is an uncommon occurrence after venomous snakebite,[53,132] and muscle necrosis after pit viper envenomation is primarily caused by direct myotoxic venom effects rather than any increase in intracompartmental pressure. The diagnosis of compartment syndrome can be difficult because envenomed extremities are often very swollen, discolored, tender, and painful on attempted ranging-of-motion of the digits. These findings may closely mimic compartment syndrome. To differentiate subcutaneous swelling from intramuscular swelling, it is best to objectively measure intracompartmental pressures using a wick catheter or digital readout device. If compartmental pressures exceed 30 to 40 mm Hg, antivenom should be started or continued while the extremity is elevated above heart level. The hemodynamically stable patient should receive IV mannitol (1 to 2 g/kg) over 30 minutes. These measures may reduce the pressures to safe levels.[39] If the pressures fail to decrease within 60 minutes and ischemia within the compartment is suspected, a fasciotomy should be considered (Fig. 48-30). Animal evidence, however, suggests that myonecrosis may actually be worsened by fasciotomy.[178] Nevertheless, surgical decompression may be needed to prevent pressure necrosis of intracompartmental nerves.[74] A knowledgable surgical consultant (hand, orthopedic, plastic, or general) should be involved if compartment syndrome is a possibility. Prophylactic fasciotomy, however, is not recommended. In animal studies, prophylactic fasciotomy did not reduce the amount of muscle necrosis when rattlesnake venom was injected intramuscularly.[54] In some cases of finger bites, if the vascular supply to the digit is compromised despite antivenom and elevation of the extremity, digital dermotomy may be needed.

Occasionally, after a bite to a digit, severe necrosis results, requiring eventual amputation of the appendage. Such an extreme procedure should be delayed until a confident level of demarcation of damage can be ascertained. A digit may require amputation if the bite results in severe contracture deformity and chronic, severe pain.

Disposition. Disposition decisions for patients bitten by pit vipers are generally straightforward. Admission to an intensive care unit (ICU) is prudent for victims with severe envenomation or with progressive clinical findings despite initial antivenom administration. Persons bitten in the head, neck, or trunk should be monitored in an ICU because of the greater risks associated with these bites. Patients who develop a serious acute reaction to antivenom should also be admitted to the ICU. Victims who require a higher level of care than is available at the treating institution should be transferred to an appropriate facility. After antivenom infusion and clinical stabilization, victims of mild or moderate envenomation can be admitted to a regular hospital floor, but they should be carefully watched, particularly if repeat doses of antivenom are planned.

A

B

C

Figure 48-28. **A,** Soft tissue swelling, hemorrhagic blebs, and early necrosis after red diamond rattlesnake *(Crotalus ruber)* bite to the long finger (day 2). Victim received 10 vials of Antivenom [Crotalidae] Polyvalent 6 hours after the bite and 10 more vials for severe thrombocytopenia on day 2. **B,** Seven weeks later. Note the degree of necrosis. **C,** Five years later. Extensor function was never regained. (**A** and **B,** Courtesy Sean Bush, MD; **C,** Courtesy Kent Denmark, MD.)

A

B

Figure 48-29. **A,** This patient was bitten by a 3-foot-long Southern Pacific rattlesnake *(Crotalus oreganus helleri)* 72 hours earlier. The snake hung on for 15 seconds and the patient suffered an extraordinarily severe systemic envenomation. Prolonged fang contact is a factor associated with increased snakebite severity. These bullae were left in place and the hand ultimately healed without any surgical intervention. **B,** The patient's hand at 3-week follow-up. At 1-year follow-up, this patient had nothing more than a slightly stiff index finger. (Courtesy Sean Bush, MD.)

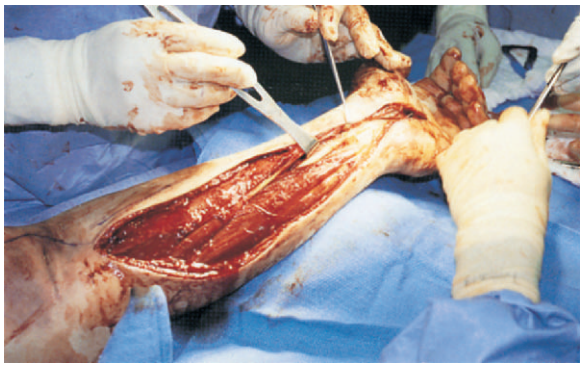


Figure 48-30. Rarely indicated fasciotomy in a victim of a severe bite from a rattlesnake *(Crotalus oreganus helleri)*. Compartment pressures were greater than 60 mm Hg despite aggressive antivenom therapy. (Courtesy Robert Norris, MD.)

Admission to the hospital should be strongly considered for all persons with apparent envenomation. In one series, more than half of persons with minimal or no signs of envenomation at presentation subsequently developed significant envenomation with moderate to severe swelling, elevated PT, or thrombocytopenia.[90] About 25% of these persons deteriorated more than 8 hours after envenomation. Even with apparent resolution of swelling, victims may later develop severe toxicity.[68] Because onset and progression of signs and symptoms after a pit viper bite vary greatly, all potential victims should be closely watched in the emergency department for a minimum of 8 hours if not admitted. Because of the delayed onset of findings with some Mojave rattlesnake bites, all persons with suspected *C. scutulatus* bites should be admitted to the hospital for 24 hours of observation. Admission is also highly recommended for children with potential venomous snakebites.

Discharge is considered after 8 hours of observation for victims who sustain an apparent dry bite. These victims have no symptoms or signs other than puncture wounds. All laboratory studies and vital signs must be normal. On discharge, patients should be instructed to return for onset of swelling, increased pain, bleeding, blood in the urine, severe headache, difficulty breathing, rash, joint pain, swollen lymph nodes, fever, or signs of wound infection. The victim should be scheduled for a follow-up reexamination in 24 to 48 hours and should be accompanied and assisted by another responsible adult.

At hospital discharge, patients who received antivenom should be reminded about the possibility of developing serum sickness and the need for prompt medical attention if they develop fever, arthralgias, myalgias, urticaria, or other findings consistent with serum sickness within the first few weeks after treatment.

Discharge instructions to patients should include information on ways to prevent venomous snakebite. More than half of bites occur during intentional interactions with snakes.[130] These usually involve attempts to handle, harass, capture, or kill the animal. Commonly, the bite is inflicted by a specimen in captivity. Avoiding intentional interaction with venomous snakes can prevent many injuries. Wearing shoes and long pants can prevent some strikes. In areas where snakes are common, young children should be closely supervised and older children educated to avoid snakes. Animal control services should be called to remove snakes found close to human habitation. If a snake is encountered in the wilderness, people should carefully move a safe distance away from it.

Assistance with managing a victim of snakebite can be obtained from regional poison control centers, including the University of Arizona Poison and Drug Information Center (520-626-6016), Rocky Mountain Poison Control (303-739-1123), and San Diego Regional Poison Control (accessed via a central number for California poison control centers, 800-411-8080). The Antivenom Index, published jointly by the American Zoo and Aquarium Association (8403 Colesville Rd., Suite 710, Silver Spring, MD 20910; 301-562-0777) and the AAPCC, lists antivenom sources within the United States.

Coral Snakes. Hospital management of coral snake bite victims (Box 48-2) can be challenging. The first priority in the stable victim is to determine that a coral snake was actually the culprit, and photographs of coral snakes and nonvenomous mimics indigenous to the area can be useful. If a coral snake is identified and appears to have inflicted an effective bite, management should proceed in anticipation of a significant envenoming, even if systemic abnormalities are currently absent. Evidence supports this approach because the progression of neurotoxicity can be extremely difficult to halt once it begins, even with antivenom administration.[154]

The patient should receive cardiac and pulse oximetry monitoring, and an IV line should be established. A history and careful physical examination should be performed; local findings are minimal, and fang marks can be difficult to see.[134] The victim typically has little or no swelling and variable local pain. The victim should be carefully assessed for any neurologic abnormalities. Evidence of respiratory dysfunction or difficulty with secretions demands aggressive airway management. Early endotracheal intubation in such cases may prevent aspiration and its complications. Bedside spirometry testing can be helpful in monitoring the respiratory status of these patients. Laboratory studies have little benefit except for ABGs if respiratory insufficiency is suspected.

If a coral snake inflicted an effective bite, preparation for antivenom administration should begin, even in the absence of systemic signs or symptoms.[99] Several antivenoms are produced for coral snakes of the Western Hemisphere. The antivenom currently used in the United States is manufactured by Wyeth-Ayerst (North American Coral Snake Antivenom *[Micrurus fulvius]*) using eastern coral snake *(M. fulvius)* venom. The current status of this antivenom as far as its continued production by Wyeth-Ayerst is uncertain, and there is no other antivenom currently available commercially in the United States. Antivenom *[Micrurus fulvius]* is effective against bites by the eastern (*M. fulvius,* formerly *M. f. fulvius*) and Texas (*M. tener,* formerly *M. f. tenere*) coral snakes. It has never been studied against Sonoran coral snake *(Micruroides euryxanthus)* venom. No antivenom is currently produced for this species, but it is a small, inoffensive creature, and no deaths have been reported after its bite.[48] Treatment of patients bitten by the Sonoran coral snake is entirely supportive. There is little information regarding efficacy of Antivenom *(Micrurus fulvius)* for other *Micrurus* species of North America. If a significant bite has occurred by a Mexican *Micrurus,* its use would probably be helpful. If Wyeth-Ayerst permanently discontinues production of Antivenom *(Micrurus fulvius),* it may be necessary for U.S. centers that treat coral snake bites to import exotic antiven-

Box 48-2. Management Principles in Treating a Victim of New World Coral Snake Bite

PRELIMINARY MEASURES

- Determine: Was the snake a coral snake?
- Determine: Is there evidence that venom poisoning has occurred (onset of signs and symptoms may be delayed)?
- Assess and support the airway, breathing, and circulation. If there is any evidence of respiratory difficulty or difficulty swallowing, secure the airway by endotracheal intubation.
- Establish one large-bore intravenous line for crystalloid infusion.
- Obtain appropriate laboratory studies (see text).
- Measure the bitten limb's circumference at two or more sites every 15 minutes (swelling is likely to be minimal).
- If local findings are progressing, if systemic signs or symptoms are present, or if it is determined that it was an effective bite by a positively identified coral snake despite absence of signs or symptoms, obtain and reconstitute an appropriate coral snake antivenom.
- Obtain informed consent for antivenom infusion, if possible.
- Expand the patient's intravascular volume with crystalloid fluids unless there is a contraindication to doing so.
- Have epinephrine (an appropriate subcutaneous dose) drawn up at the patient's bedside for use in the event of an acute reaction to antivenom.

North American Coral Snake (*Micrurus fulvius*) Antivenom

- No skin test for possible allergy to antivenom (ineffective)
- Pretreat:
  - H_1- and H_2-blocking antihistamines

First Phase of Antivenom Administration

- Starting dose:
  - All 3–6 vials
- Dilute the vials of antivenom to be given in crystalloid (1 L for an adult; 20–40 mL/kg for a child).
- Begin antivenom by slow intravenous infusion with physician at the bedside.
- Gradually increase the rate of infusion in the absence of an acute reaction to get the total dose in over 1–2 hours.
- If a reaction occurs, stop the antivenom and treat as necessary.
- Consider the need for further antivenom.
  - Further antivenom to be withheld: conservative care only (may require endotracheal intubation and prolonged respiratory support if respiratory embarrassment arises).
  - Further antivenom to be given: dilute the antiserum further, administer intravenous steroids, restart the antivenom at a slower rate.

SECOND PHASE: CONTINUED REACTION IN THE SETTING OF SEVERE POISONING

- Admit to the ICU.
- Institute maximal monitoring as needed (e.g., arterial line).
- Consult an expert in snake envenomation.
- Employ epinephrine drip titrated against the reaction.

Further Dosing of Antivenom

- Administer 3–5 more vials as needed (rarely are more than 10 vials needed).

DISPOSITION

- Admit all (even apparent nonenvenomations) to an ICU for observation and further management.

oms for this purpose. Research has demonstrated effectiveness of Mexican coral snake antivenom and Australian tiger snake *(Notechis scutatus)* antivenom in reversing the effects of *Micrurus* venom.[194]

Preparation for coral snake antivenom administration is similar to that for pit vipers. Informed consent is obtained if possible. Skin testing for potential allergy to horse serum is inaccurate and of no benefit in making therapeutic decisions. The victim's intravascular volume should be expanded with prudent crystalloid infusion and the patient premedicated with IV antihistamines (both H_1 and H_2 blockers). The starting dose for Antivenom *(Micrurus fulvius)* is 3 to 6 vials, diluted in 500 to 1000 mL of crystalloid (in 20 mL/kg bodyweight for pediatric patients).[193] IV infusion is begun at a slow rate, with the physician at the bedside, ready to intervene with epinephrine if an acute adverse response occurs. The rate is then increased to administer the entire dose over approximately 2 hours.

If a reaction occurs, the antivenom infusion should be temporarily halted and the patient treated as necessary (e.g., with epinephrine, further antihistamines, steroids). Once the reaction is treated, the antivenom can usually be restarted at a slower rate after further dilution of the dose. If a severe reaction occurs, a difficult decision must be made: whether to continue antivenom administration efforts, as outlined for pit viper antivenom reactions (see earlier), or to treat the patient conservatively (with endotracheal intubation and respiratory support as needed). Respiratory paralysis after severe bites can be prolonged and may require days to weeks of mechanical ventilation.[99]

Acute allergic reactions occur in approximately 35% of patients who receive North American Coral Snake Antivenom *(Micrurus fulvius)*, and serum sickness is seen in approximately 10%.[99] These reactions are treated in the fashion outlined earlier in the discussion of pit vipers.

The role for anticholinesterase agents in the management of bites by coral snakes remains to be determined.[181] If there is evidence of neurotoxicity, a Tensilon test can be performed. After IV pretreatment with atropine (adults, 0.6 mg; children, 0.02 mg/kg, minimum of 0.1 mg) to prevent cholinergic crisis, edrophonium chloride (10 mg for adults, 0.25 mg/kg for children) is given intravenously over 3 to 4 minutes. Patients who demonstrate definite improvement in neurologic findings can be started on a continuous infusion of neostigmine methyl-

sulfate (50 to 100 µg/kg every 4 hours) with intermittent atropine boluses (same as starting dose, every 4 hours). Anticholinesterase drugs are not, however, a substitute for antivenom administration.

Hypotension and Shock. When patients have swelling of more than half the bitten limb, and the central venous pressure is low or there is other clinical evidence of hypovolemia, IV fluids should be infused. A plasma expander, such as fresh whole blood or fresh frozen plasma, may be indicated. If this fails to improve the blood pressure, a selective vasoconstrictor can be used, such as dopamine (starting dosage, 2.5 to 5 µg/kg/min by IV infusion).

All victims who have possibly been bitten by a coral snake should be admitted to an ICU for close monitoring even if a dry bite is suspected. If signs of neurotoxicity progress after initial antivenom infusion, 3 to 5 more vials of antivenom should be administered; rarely are more than 10 vials required.[138]

Morbidity and Mortality

Pit Vipers

Reliable estimates of morbidity and mortality from snakebite in the United States are not available, but deaths are uncommon. Extensive work by Parrish in the late 1950s revealed an estimated 7000 venomous snakebites per year with approximately 15 deaths.[140] More recent assessments reveal that snake-related fatalities average fewer than half a dozen per year (range, 0 to 12) in the United States, ranking snakes far behind dogs and hymenopterans in terms of animal-related fatalities.[102,103] Most snakebite deaths occur in white males in the southern and western United States. Of interest, the proportion of snake-related fatalities to Hymenoptera-related fatalities was far less in Parrish's studies than in Langley's. In the 1950s, snakes comprised 30% and Hymenoptera 50% of the venomous animal-related fatalities. In the 1990s, the proportion of snakes fell to 7.5%, whereas Hymenoptera-related deaths rose to over 70%.

Another source of information on snakebite-associated morbidity and mortality is the AAPCC. Over a 21-year period (1983 to 2003), only 18 deaths from endemic venomous snakes were reported to the AAPCC (Table 48-7).[104–121,180,182,183] When the offending reptile was identified, almost all were rattlesnakes. These numbers are underestimates and can be used only as a rough gauge of incidence and mortality. Not all venomous snakebites, even fatal ones, are reported to the AAPCC. Snakebite is not classified as a reportable disease, and no reliable government statistics exist.

Mortality from snakebite depends largely on treatment, and although causes of death vary, delay in reaching emergency medical care is considered to be a major contributor.[72] When death results, it may occur (but rarely) within a few minutes or, more commonly, it can take several days or more. A quick death can be caused by anaphylactic or anaphylactoid reactions to venom or antivenom or by venom-induced respiratory insufficiency. Prolonged hypotension, shock, and tissue hypoxia can lead to hypoxic encephalopathy, cerebral edema, and herniation. Venom-induced coagulopathies can lead to life-threatening bleeding complications, such as intracranial or gastrointestinal bleeding. Multistroke encephalopathy has been observed to occur in patients with true disseminated intravascular coagulation, which is very rare. Renal failure may take several days to manifest and could cause death if untreated. Comorbidities, such as coronary artery disease, can affect the chances of surviving snakebite. Iatrogenic complications, such as adverse drug reactions or nosocomial infections, can also lead to death. Before the introduction of Wyeth's ACP in 1954, mortality rates were estimated to be as high as 5% to 25%.[142] After this time, mortality rates in patients treated without antivenom declined to approximately 2.6%, largely because of improvements in other aspects of care (e.g., development of ICUs and of fluid resuscitation principles). Antivenom, however, further reduces the mortality rate to 0.28%, a statistically and clinically significant tenfold reduction.[142] This difference may be even more significant with the use of CroFab, although mortality statistics are not yet available for this product. The challenge and focus of future research is likely to shift from mortality issues to measures that reduce morbidity, disability, pain, and inconvenience (i.e., issues associated with prolonged monitoring after treatment).[40]

Other than death, permanent systemic morbidity after pit viper envenomation is rare.[45] Local sequelae, such as the partial or complete loss of a limb or digit, loss of function at a joint, or permanent sensory loss, are more common.[45] The reported incidence of permanent local morbidity is less than 10%, although this does not include complications that may follow surgical interventions.[64] Most patients recover fully after rattlesnake envenomation in the United States, but the incidence of local complications is probably underestimated. Unless careful follow-up is done, including objective range-of-motion and sensory testing, permanent disabilities that impact lifestyle and occupation can be missed.[166]

Although not clearly substantiated, children are probably more susceptible to systemic morbidity and mortality from snakebites.[4,37,136,185] However, of the 104,750 snakebite exposures and 23 deaths (including endemic and exotic snakes) described by the AAPCC since its first report in 1983, only two deaths have been in pediatric patients.[104–121,180,182,183] Better pediatric supportive care and improved understanding of how antivenom should be administered have blurred any distinction between pediatric and adult prognosis.[45] Older adult patients appear to have a higher case-fatality rate, probably related to comorbid conditions.[45]

Morbidity and mortality from snakebites can also result from efforts to treat the victim. Significant wound complications can follow ill-advised incisions in and around the bite site and the application of mouth suction.[57,193] Serious burns and systemic complications, such as myocardial infarction, can follow application of electric shocks to the wound.[156] Tourniquets or ice may increase the risk of tissue damage. Antivenoms can precipitate early anaphylactic or anaphylactoid reactions that can add to morbidity and even result in death (see earlier).

Coral Snakes

Although no deaths have been reported from treated coral snake bites in the United States since antivenom *(Micrurus fulvius)* was introduced,[155] the mortality rate for an untreated bite is estimated at approximately 10%.[143] Prolonged muscle weakness is common after severe envenoming, and victims with respiratory compromise may require mechanical ventilation for many days. There are no reports of permanent sequelae in patients who survive coral snake envenomation.

TABLE 48-7. Snake-Related Deaths Reported to the AAPCC, 1983–2003

YEAR	TOTAL BITES REPORTED*	DEATHS	SPECIFICS OF DEATHS
1983	717	1	*1 rattlesnake*
1984	1347	1	*1 rattlesnake*
1985	1676	0	—
1986	2416	0	—
1987	2701	1	*1 rattlesnake*
1988	3076	0	—
1989	3851	1	*1 prairie rattlesnake*: A 27-yr-old man was bitten on two fingertips by a 2-ft-long prairie rattlesnake (*Crotalus viridis*, formerly *C. v. viridis*). He arrived in the ED 15 minutes later. Swelling rapidly progressed to the upper arm over the next $2^1/_2$ hours. He was pretreated with diphenhydramine, epinephrine, and methylprednisolone and 5 vials of antivenom in 250 mL of D5W were started. Twenty minutes later, 60% of the antivenom had been given and he developed anaphylaxis. He was given IV epinephrine, and a cricothyroidotomy was performed as he could not be intubated because of laryngospasm. He suffered cardiac arrest. Autopsy revealed bronchospasm but no swelling of the upper airway.
1990	4461	0	—
1991	5255	1	*1 unidentified rattlesnake:* A 52-yr-old man was bitten on his thumb by an unidentified rattlesnake in central Oregon at high altitude. In the ED 20 minutes later he complained of circumoral numbness, tingling, and flushing. BP was 140/94 mm Hg, P was 92 bpm, RR was 16/min. On his thumb there were "two entrance wounds and minimal swelling." He had been given 6 mL of a solution of 5 vials of antivenom in 500 mL D5W when he became diaphoretic and dyspneic with increased pulmonary secretions. He became near-syncopal. The antivenom was discontinued and he was intubated and given epinephrine and steroids. He became hypotensive and developed asystole within 15 minutes. Laboratory values showed acidosis, hemoconcentration, and mild coagulopathy. Autopsy revealed extensive coronary artery disease.
1992	1055	2	*1 northern Pacific rattlesnake:* A 20-yr-old man was handling a northern Pacific rattlesnake (*Crotalus oreganus*, formerly *C. viridis oreganus*) when it bit him on the lips. He collapsed, began vomiting, and then was driven 3 miles to the hospital. He was intubated 40 minutes later, but he suffered cardiac arrest during the procedure. He was resuscitated with epinephrine, atropine, and 5 vials of antivenom for 30 minutes before recovering a sinus rhythm and a BP of 100/60 mm Hg. He developed profound coagulopathy and was given 30 vials of antivenom. He was later determined to be brain dead. Autopsy showed brain edema and herniation. Blood alcohol level was 207 mg/dL. *1 black Indian cobra:* A 25-yr-old male snake expert was bitten on the toe by his pregnant pet black Indian cobra. He died within minutes. Autopsy revealed bloody pulmonary exudate, cerebral edema, and a fine petechial rash.
1993	5653	0	—
1994	6317	2	*1 presumptive Mojave "green" rattlesnake:* A 40-yr-old man was bitten while working in bushes. He collapsed 20 minutes later. In the ED 4.5 hours later, he was comatose and in atrial fibrillation with labile blood pressure. He was intubated for poor respiratory effort. Fasciculations and metabolic acidosis were noted. Two small puncture wounds, 1 cm apart, were noted on the forearm and lower leg, which had been initially overlooked. The presumptive diagnosis of Mojave "green" rattlesnake *(Crotalus scutulatus scutulatus)* was made, and antivenom was administered. He was cardioverted to normal sinus rhythm and dopamine was started, but he remained hypotensive. He developed disseminated intravascular coagulopathy and died on the 8th hospital day. *1 rattlesnake:* A 34-yr-old male "snake handler" was bitten on the hand when he picked up a rattlesnake on the road. He collapsed 10 minutes later and family began cardiopulmonary resuscitation. Medics arrived 30 minutes later and he was in full cardiopulmonary arrest. He was intubated and given IV fluids, dopamine, antivenom, and hydrocortisone. He died within 3 hours. Autopsy revealed hemorrhage in the myocardium, alveoli, pancreas, and kidney. The airway was patent, but there was laryngeal edema, pulmonary edema, and evidence of aspiration. His right coronary artery was 75% stenotic. Blood alcohol level was 118 mg/dL.
1995	7100	2	*1 canebrake rattlesnake:* A 35-yr-old man was bitten near the radial artery while playing with a *Crotalus horridus atricaudatus* (now classified as *C. horridus*). He was asystolic when medics arrived 30 minutes later. At autopsy, no necrosis was noted. Blood alcohol level was 250 mg/dL. *1 unknown reptile*
1996	7494	0	—
1997	7045	2	*1 rattlesnake:* A 4-yr-old boy bitten on the thigh was treated with 5 vials of antivenom and transferred. He was intubated en route and subsequently developed massive swelling. He suffered cardiac arrest, was resuscitated and given additional antivenom, and then arrested again in the PICU 7 hr after envenomation. *1 poisonous exotic snake*
1998	7194	0	—

Continued

TABLE 48-7. Snake-Related Deaths Reported to the AAPCC, 1983–2003—cont'd

YEAR	TOTAL BITES REPORTED*	DEATHS	SPECIFICS OF DEATHS
1999	6832	2	*1 rattlesnake:* A 43-yr-old man who was drinking with his friends was bitten on the hand by a rattlesnake presumed to be dead. After further ethanol consumption, he presented 1 hr later with complaints of hand pain and mild abdominal cramping. His HR was 134 bpm, RR was 28/min, and BP was 81/46 mm Hg. Tongue swelling, fang marks, and minimal edema of the hand were noted. Hypotension necessitated vasopressor therapy, and increased tongue edema was treated with diphenhydramine and steroids. Unsuccessful attempts at intubation were complicated by hypoxia, and cricothyroidotomy was performed. Further treatment included sodium bicarbonate for metabolic acidosis, 1 unit of polyvalent antivenom, and fresh frozen plasma. The course was complicated by acute renal failure secondary to hemolysis requiring hemodialysis, rhabdomyolysis, and prolonged ventilatory support. On the 13th hospital day, cardiac arrest secondary to pulmonary embolism occurred, after which the patient was declared brain dead. *1 unknown snake*
2000	7316	2	*1 eastern diamondback rattlesnake:* A 2-yr-old boy was bitten just proximal to the knee by an eastern diamondback rattlesnake *(Crotalus adamanteus).* Findings included one fang mark and minimal local signs. On arrival at the hospital, BP was 98/48 mm Hg, and HR was 130 bpm. Listlessness, progressive swelling at the site of the bite, hypotension, and bleeding from his cutdown site, nose, mouth, and gastrointestinal tract ensued. Treatment included 90 vials of polyvalent Crotalidae antivenom, vasopressors, and blood products. Within 24 hours, he had fixed, dilated pupils and no response to pain. A CT brain scan revealed bilateral cerebral infarctions and he was declared brain dead. *1 timber rattlesnake:* A 45-yr-old man was bitten under the right nipple by a timber rattlesnake (*Crotalus horridus horridus* [now classified as *C. horridus*]) during a snake-handling congregational meeting. Church members reportedly prayed over him for the next 2 days until he died. At autopsy, systemic petechiae, marked ecchymosis of his anterior chest wall, and hemorrhagic mediastinitis were noted.
2001	7463	2	*1 rattlesnake:* A 30-yr-old man was comatose after a bite from his pet rattlesnake. In the ED, he was found to have coagulopathy and a subarachnoid hemorrhage. On admission, his INR was 5.5, and his platelet count was 7000/μL. He received 10 to 20 vials of crotalid antivenom and was admitted to the ICU. His condition continued to worsen and support was withdrawn on the day after admission. *1 unknown crotaline*
2002	7829	2	*1 timber rattlesnake:* A 43-yr-old man was reportedly bitten by a timber rattlesnake (*Crotalus horridus horridus* [now classified as *C. horridus*]) while he was trying to capture it. He was unstable in the ED 1 hr after the bite, requiring endotracheal intubation and epinephrine for a BP of 60/47 mm Hg. He was treated with 10 vials of crotaline polyvalent antivenom, diphenhydramine, corticosteroids, morphine, and diazepam. He was transferred to another hospital. An additional 16 vials of antivenom were administered. Dopamine and epinephrine were administered for hypotension. Four hours after the admission there was laboratory evidence of disseminated intravascular coagulation. Six vials of Fab antivenom were given. Over the next few hours, he appeared to stabilize and vasopressors were discontinued. Several hours later epistaxis and bleeding from his mouth began. An additional 7 vials of antivenom, fresh-frozen plasma, platelets, and cryoprecipitate were given. Renal failure developed and required hemodialysis. By the 3rd day he had developed multisystem organ failure. He died on the 10th hospital day. *1 rattlesnake*
2003	7952	2	*1 rattlesnake:* A 33-yr-old man was hospitalized for a bite to his hand by a *Crotalus viridis lutosus* [now classified as *C. oreganus lutosus*]. He was treated with CroFab and discharged within 24 hours after minimal symptoms. He was given a prescription for acetaminophen/oxycodone on discharge. That night he was noted to be snoring in bed and was difficult to arouse, and in the morning he was found dead in bed. Autopsy revealed pulmonary edema, thick secretions occluding the trachea and bronchi, and cerebral edema. Postmortem heart blood had an oxycodone concentration of 200 ng/mL. The medical examiner attributed the death to respiratory arrest from an opiate intoxication. *1 poisonous exotic snake*
Totals:	104,750	23	*Of the cases where circumstances were described:* • 2 appeared to be due to anaphylaxis to antivenom. • 1 appeared to be due to self-overmedication with opiates. • All victims were male. • Ages were 2, 4, 20, 25, 27, 30, 33, 35, 40, 43 (two), 45, and 52. • 17 rattlesnakes, 3 exotics, 3 unknown • 3 were victims known to be intoxicated with alcohol. • 7 upper extremity, 4 lower extremity, 1 trunk, 1 facial • Time to death varied from minutes to 10 days. • Most reported deaths were associated with very unusual circumstances.

*Includes all bites by reptiles (including exotics, nonpoisonous, and unknowns).

AAPCC, American Association of Poison Control Centers; BP, blood pressure; bpm, beats/min; CT, computed tomography; D5W, 5% dextrose in water; ED, emergency department; HR, heart rate; ICU, intensive care unit; INR, International Normalized Ratio; P, pulse; PICU, pediatric ICU; RR, respiratory rate.

From references 104–121, 180, 182, 183.

Figure 48-31. Teeth of the helodermatid lizards are grooved to aid instillation of venom during a bite. These teeth are loosely adherent and may become dislodged in the bite wound. (Courtesy Michael Cardwell/Extreme Wildlife Photography.)

A

B

Figure 48-32. **A,** This patient sustained a bite by a captive subadult Gila monster *(Heloderma suspectum)* after he touched the lizard's tail. It took approximately 40 seconds to pry the reptile off with tongs. Immediately after, the patient complained of "10 out of 10" pain. Shortly after, he developed swelling and redness extending to his mid-forearm, as well as nausea and vomiting. **B,** A tooth was retained in the wound for over 2 months before it was spontaneously expelled. (Courtesy Sean Bush, MD.)

VENOMOUS LIZARDS

Anatomy

The two venomous lizards of the world are impressive creatures about which much misinformation has been spread for centuries. They have been thought to possess supernatural features such as poisonous breath, a stinging tail, and the ability to spit their venom.[155] The Gila monster *(Heloderma suspectum)* reaches a maximum length of approximately 50 cm (19.7 inches), whereas the beaded lizard *(Heloderma horridum)* is larger, reaching almost a meter (39.4 inches). They are both heavily built and possess massive muscles of mastication with powerful biting capacity. The venom delivery apparatus consists of a pair of anterior, multilobed glands that open through a series of ducts into the labial mucosa of the lower jaw. Their teeth are lancet shaped, grooved, and loosely attached to the jaws (Fig. 48-31). When the lizard becomes agitated, it salivates heavily, producing a flow of venom into the labial mucosa. It bites with a powerful, chewing motion, instilling venom into the wounds by capillary action along the grooves of the teeth. Teeth may be left in the wounds, especially if the animal must be forcefully removed from the victim. The tenacious creature may still be attached when help arrives. Effective envenomation occurs in about 70% of bites.[9]

Venom

Gila monster and beaded lizard venoms are similar in composition and are as potent as some rattlesnake venoms.[170] They possess enzymatic components, including hyaluronidase (spreading factor), protease, phospholipase A_2, kallikrein-like substances, and nonenzymatic substances such as serotonin. Venom kallikreins stimulate the release of vasoactive kinins, such as bradykinin, which may be responsible for occasional hypotension seen after helodermatid bites.[172] These venoms contain no neurotoxic components and no components that directly interfere with coagulation.[127] Of note, Gila monster venom inspired the production of a drug that helps diabetics get better control of their blood sugar.[168]

Clinical Presentation

The vast majority of victims bitten by a lizard are attempting to catch or handle the reptile; accidental bites are rare. Most bites involve captive animals, and many are not reported.

Bleeding usually occurs from punctured and torn tissues, but it is generally not excessive.[127] Throbbing or burning pain may radiate proximally along the bitten extremity. Local edema may be progressive (Figs. 48-32 and 48-33). Victims may complain of generalized weakness, nausea and vomiting, difficulty breathing, profuse sweating, dizziness, and paresthesias.[85,155,172]

On examination, the victim may be tachycardic, hypotensive (related to kinin release), and diaphoretic.[85] There may be significant local tissue trauma. The site may be cyanotic or ecchy-

Figure 48-33. Bite of a captive beaded lizard *(Heloderma horridum).* (Courtesy Michael Cardwell.)

motic with local vasospasm.[155] Regional lymphadenopathy may be present.[9]

Management

Prehospital Care

Data are minimal regarding prehospital care of venomous lizard bites. In some cases, the first priority is to detach the tenacious lizard from the victim. The lizard can be placed under running hot water, or the jaws can be pried apart using a stick or metal instrument.[172] Care must be taken not to injure the victim further and to avoid a second bite, perhaps to the rescuer.

Once freed from the lizard, the victim should be placed at rest and the bite site rinsed and cleaned as much as possible. Any bleeding should be stopped with direct pressure. No evidence supports the use of suction devices, ligatures, or pressure immobilization. Local application of ice should be avoided as it may increase the risk of vasospasm and tissue damage.[159] A dressing to stop bleeding and a splint to limit movement may be beneficial. Transport to a medical facility should be carried out as expeditiously as possible. Incisions and electrotherapy should be strictly avoided.

Hospital Care

Assuming the lizard has been detached before arrival at the hospital, the victim's airway, breathing, and circulation are assessed. Vital signs should be obtained while the patient is being placed on oxygen and cardiac and pulse oximetry monitoring. At least one large-bore IV line should be established with either normal saline or Ringer's lactate. If the victim is hypotensive or tachycardic, a second line should be placed, and physiologic saline should be given (1 to 2 L for an adult, 20 to 40 mL/kg for a child). Hypotension rarely persists after volume resuscitation. If necessary, vasopressors can be added after the victim's intravascular volume has been repleted.[172]

Laboratory testing should include a complete blood count, serum electrolytes, renal function studies, and coagulation studies. Total white blood cell count may be elevated, and in severe cases, platelets may be decreased.[155] Despite the fact that lizard venoms do not appear to possess anticoagulant fractions, rare hemostatic abnormalities have been reported after severe bites and are probably indirectly related to endothelial cell damage.[9,172] Urinalysis is useful to assess for microscopic hematuria or renal casts.

These bites can cause transient ECG abnormalities, such as T-wave anomalies and conduction delays.[9,85] A single case of myocardial infarction after a Gila monster bite has been reported in a patient with a past history of cocaine abuse.[9] Thus, it is reasonable to obtain an ECG if the victim shows any sign or symptom of envenomation.

Once the victim's overall status is stabilized, attention focuses on wound care. A soft tissue radiograph of the bite site may identify retained lizard teeth (see Figure 48-32B) but does not replace careful exploration of the wounds.[85] After an assessment of functional status, the wound can be anesthetized using a local or regional block. The bite site should then be carefully explored for damage to underlying vital structures and for retained teeth. Thorough cleansing and irrigation should follow exploration. The wounds are dressed and splinted with generous padding. The extremity is then elevated to reduce swelling and discomfort. Opiates may be necessary in the management of pain during the victim's initial evaluation and for any pain not controlled by local or regional anesthesia. The victim's tetanus immunization status should be updated. Prophylactic antibiotics are not required.[172] Daily wound care should include cleansing with soap and water, followed by hydrogen peroxide, application of a topical antiseptic, and redressing. Physical therapy is helpful in returning the patient more rapidly to full function.

Victims who are relatively asymptomatic, with normal vital signs and diagnostic studies, may have received a dry bite, and they can be discharged home after 8 hours of observation in the emergency department. They must have proper resources for care at home and the ability to return if they get worse. Proper wound care instructions should be given and patients should be scheduled for reevaluation in approximately 24 to 48 hours. An oral narcotic analgesic may be prescribed. Evidence of envenomation (i.e., signs or symptoms besides simple wounds; laboratory or ECG abnormalities) necessitates admission. Systemic findings (e.g., chest pain, ECG changes, hypotension, rare coagulopathy) require monitoring for at least 24 hours.[172] There is no commercially available antivenom for venomous lizard bites.

Morbidity and Mortality

No fatalities from helodermatid bites have been documented in the past 50 years,[127] but a bite might prove fatal if a large lizard hangs on to the victim and chews in venom for a matter of minutes. Bites to children are especially concerning because of the larger venom load per body mass that they may receive, compared with adults. Likewise, given their complicating comorbidities, chronically ill or older adult victims could have a serious outcome.

Pain and tenderness may be prolonged for days or weeks after these bites,[159] although necrosis is rare.[127,155] Wound infections are uncommon. If infection occurs, cultures should be obtained and the victim started on broad-spectrum antibiotic coverage for both gram-positive cocci and gram-negative rods. Culture and sensitivity results should guide further treatment.

ALLERGY TO REPTILE VENOMS

Snake and lizard venoms are highly immunogenic substances, and occasionally a bitten victim presents with an acute, ana-

phylactic reaction.[11,65,145] The risk of such reactions increases in victims previously bitten by venomous reptiles or those who work with snake or lizard venoms (e.g., in a venom-extraction or research facility). The presentation can be typical for anaphylaxis, with bronchospasm, hypotension, and urticaria, but differentiating between an acute allergic reaction to venom and severe direct venom toxicity can be difficult. If the precise etiology for hypotension or respiratory distress is unclear, treatment can be rendered for anaphylaxis while antivenom preparation (in the case of snakebite) and supportive care proceed.

BITES BY EXOTIC SNAKES IN THE UNITED STATES

With the growing popularity of herpetoculture in the United States, bites by captive venomous reptiles have increased. Many involve species indigenous to North America, but an increasing number are caused by exotic species. Russell estimated that exotic venomous snakes were responsible for 30 bites in the United States in 1984.[157] In 2003, however, 126 such cases were reported to the AAPCC Toxic Exposure Surveillance System.[182] An emergency physician anywhere in North America may see a victim who has been bitten by a king cobra, a black mamba, or another exotic species. Management principles in these cases are similar to those outlined earlier (see also Chapter 49).

THOUGHTS BEFORE GOING INTO THE FIELD

Before leaving on a trip into habitat in which venomous reptiles may be encountered, it is important to plan the steps to be taken should a bite occur. The traveler should become familiar with the local dangerous fauna, using field guides or internet resources. A plan should be developed for field management of a bite (e.g., possible use of pressure immobilization in the event of a life-threatening bite in a remote location). How will a victim be evacuated from the location? What will be the destination hospital? Does the hospital have antivenom and personnel knowledgable in the management of snakebite? Appropriate supplies should be gathered (e.g., crepe bandages or elastic wraps and splints [Sam Splints are lightweight and easily carried] for pressure immobilization). The question of whether to carry antivenom on a trip to a remote location often arises. If this is considered, an appropriate, preferably broad-spectrum, polyspecific product is preferable. This involves purchasing the chosen antiserum in the destination country, as advance importation of exotic antivenoms into the United States is time consuming and difficult. If a foreign antivenom is to be carried, its package insert should be translated if necessary before the trip begins. If traveling to Central or South America, it is likely that CroFab or ACP would provide broad enough coverage for most pit vipers that might be encountered (though further research is needed in this area). CroFab has been studied and found to be relatively stable despite prolonged exposure to high temperatures and rough handling,[44] and it should, therefore, stand up to the punishment of vigorous hiking in tropical regions. The cost of U.S. antivenoms is, however, likely to prohibit any but the best-financed expeditions from carrying antiserum. If antivenom is to be taken into the field, all of the equipment necessary for its administration must also be carried (IV supplies, a bag of normal saline, airway equipment, and the drugs needed to adequately treat an anaphylactoid reaction), and someone in the expedition must have the skills needed to give the drug and treat any reaction that might occur. Although it may be possible to administer CroFab intramuscularly,[164] further research is needed to determine if the smaller molecular size of Fab fragments allows adequate distribution by this route to effectively treat snake envenomation. The most important consideration when entering the field is, of course, prevention. Long pants and boots should be worn in snake country, and team members should be extra vigilant about visually clearing where they will place their hands and feet when hiking, climbing, or gathering firewood. If a snake or venomous lizard is encountered, it is best appreciated and photographed from a safe distance.

The references for this chapter can be found on the accompanying DVD-ROM.

49 Bites by Venomous Snakes outside the Americas

David A. Warrell

INTRODUCTION: DANGEROUS SNAKES

In the rest of the world outside the Americas, there are three families of venomous snakes: Atractaspididae, Elapidae (including true sea snakes [subfamily Hydrophiinae] and sea kraits [subfamily Laticaudinae]), and Viperidae (including pit vipers [subfamily Crotalinae]), as well as a fourth large family, Colubridae, long considered harmless but now recognized to contain a number of species in the subfamily Boiginae with venoms dangerous to humans.[67] Some species of giant python (family Boidae) have been responsible for rare fatal attacks reported from Africa (rock python, *Python sebae*), south and southeast Asia, especially Indonesia (reticulated python, *Python reticulatus,* and Indian python, *Python molurus*), and Australia, where there was a recently reported fatal attack by a 17-foot-long scrub python, *Morelia amethistina* (P. Mirtschen, personal communication, 2005). (See Chapter 48 for a discussion on attacks by South American Boidae.) The victims were asphyxiated and crushed by constriction and in some cases swallowed.[69,150] In 1974, a man attacked by *P. sebae* near Harrar, Ethiopia, died 48 hours later as a result of perinephric hematomas resulting from crush injuries.

CLASSIFICATION AND TAXONOMY OF VENOMOUS SNAKES

Snake taxonomy is based on morphologic features, such as the numbers and arrangement of scales (lepidosis), dentition, osteology, myology, sensory organs, the form of the hemipenes (paired reproductive organs), and, increasingly, by sequence analysis of DNA encoding of important mitochondrial and other enzymes.[46,79,174–176,178,180] The accelerating pace of taxonomic revision of medically important taxa often leaves clinicians and nonherpetologists confused and frustrated. To change familiar names and to split genera and species is particularly misleading, even though it may be well justified on evolutionary and taxonomic grounds. However, correct identification and designation of species responsible for bites is essential for logical design of regional antivenoms and for appropriate selection of venoms for their manufacture. It is also crucial for laboratory toxinologists who work on venoms that are sometimes of uncertain provenance. Several toxins have been misnamed because of ignorance of the correct nomenclature for the source species. Thus "ceruleotoxin" was named after *Bungarus caeruleus*, although its true origin was *Bungarus fasciatus,* and "grambin" and "graminelysin" were named after *Trimeresurus gramineus* but were obtained from *Trimeresurus (Viridovipera) stejnegeri.* In an attempt to improve the flow of information between taxonomists, toxinologists, and clinicians, a series of reviews were published in *Toxicon.*[176,177]

IDENTIFICATION OF VENOMOUS SNAKES

Distinguishing Snakes from Legless Lizards, Amphibians, and Snakelike Fish

Most lizards have eyelids, external ear orifices, thick and fleshy tongues, and very long friable tails; they lack the enlarged ventral scales of snakes. Some have only vestigial limbs. Legless lizards, such as slow worms and glass lizards (family Anguidae), wormlike geckos (family Pygopodidae) (Fig. 49-1, *Lialis jacari*), and legless skinks (family Scincidae), may be confused with snakes unless these features are detected. The fossorial Amphisbaenids have wormlike annular grooves along the length of their bodies (Fig. 49-2, *Amphisbaena fuliginosa*) and caecilians (legless amphibians) have no obvious eyes or scales. Eels and pipe-shaped fish are easily distinguished from snakes by their gills and fins.

Distinguishing Venomous from Nonvenomous Snakes

There is no quick, simple, and absolutely reliable method for distinguishing venomous from nonvenomous snakes. The fangs may be very small in elapids or they may be folded back inside a sheath in vipers (Fig. 49-3, *Bitis arietans*). The shaft of a needle passed along the maxilla from the angle of the jaw to the snout may engage upon and reveal the fangs. Some key morphologic features are subtle. Most elapid snakes lack the loreal scale (between the preocular and nasal scales) possessed by most colubrid snakes (Fig. 49-4, *Dendroaspis angusticeps*). Fortunately, the most dangerous species tend to be well known in the areas where they are medically important. The characteristic

Figure 49-1. Legless lizard: Jacar's snake-lizard (*Lialis jacari:* family Pygopodidae) (Madang, Papua New Guinea) showing external ear opening distinguishing it from snakes. (Copyright D.A. Warrell.)

Figure 49-2. Amphisbaenid lizard (*Amphisbaena fuliginosa:* family Amphisbaenidae) (Peru) showing distinctive annular rings distinguishing it from snakes. (Copyright D.A. Warrell.)

Figure 49-3. Fangs of African puff adder *(Bitis arietans)* partly folded and concealed in the dental sheath. (Copyright D.A. Warrell.)

Figure 49-4. Eastern green mamba *(Dendroaspis angusticeps)* (KwaZulu Natal, South Africa) showing the absence of a loreal scale between the preocular and nasal scales, a feature that distinguishes elapid snakes from most colubrid snakes. (Copyright D.A. Warrell.)

hood of cobras and some other elapids is visible only when the snake is rearing up in a defensive attitude (Fig. 49-5, *Naja mossambica*). Vipers are often identifiable by their repeated and sometimes colorful dorsal pattern (Fig. 49-6, *Bitis arietans*; Fig. 49-7, *Bitis rhinoceros*). Russell's vipers *(Daboia russelii)* (see Figure 49-8A), *Daboia siamensis* (Fig. 49-8B), and puff adders *(B. arietans)* (see Figures 49-3 and 49-6) hiss loudly by expelling air through their large nostrils. The saw-scaled or carpet vipers (genus *Echis*) produce a characteristic rasping sound by rubbing their coils together (Fig. 49-9, *Echis carinatus,* and Fig. 49-10, *Echis ocellatus*), and king cobras *(Ophiophagus hannah)* "growl." Asian pit vipers (Crotalinae) include the genera *Gloydius, Deinagkistrodon, Calloselasma, Trimeresurus sensu lato* (Fig. 49-11), *Hypnale* (Fig. 49-12), *Ovophis,* and *Tropidolaema.* A recent taxonomic revision of the large genus *Trimeresurus* (arboreal and terrestrial pit vipers) has created seven new genera, which might cause great confusion.[62,86] The pit of crotaline snakes is a heat-sensitive organ, situated between the eye and nostril, that detects warm-blooded prey (see Figures 49-11 and 49-12). Snakes of the subfamily Viperinae, the Old World vipers and adders, lack this pit organ.

Some harmless snakes are easily mistaken for the venomous species that they mimic: for example, *Telescopus* (cat snake) and *Dasypeltis* (egg-eating snake) are confused with *Echis* (saw-scaled viper) species in Africa; *Boiga multomaculata* with *D. siamensis* in Thailand; various species of *Dryocalamus* and *Lycodon* with the kraits *Bungarus candidus* and *B. caeruleus* in South Asia.

Fangs and Venom Apparatus[47]

In their upper jaws, venomous snakes have one or more pairs of enlarged teeth (fangs) containing grooves or venom channels

Figure 49-5. Mozambican spitting cobra (*Naja mossambica:* family Elapidae) (Harare, Zimbabwe) showing the hood spread in a threatening/defensive attitude and sensing tongue. (Copyright D.A. Warrell.)

Figure 49-6. Puff adder *(Bitis arietans)* (South Africa) showing the distinctive repeated U (chevron) dorsal pattern. (Copyright D.A. Warrell.)

through which venom is injected through the skin of their prey or human victim. One or two reserve fangs may be situated immediately behind the active fang on each side.

Colubridae

Venom from supralabial (Duvernoy's) glands tracks down grooves in the anterior surfaces of the short, posteriorly situated (opisthoglyphous) fangs (hence the term "rear-fanged

Figure 49-7. Pair of West African Gaboon vipers *(Bitis rhinoceros)* showing their striking dorsal pattern. Two species of Gaboon viper are now recognized, this West African species and *B. gabonica* from eastern and southern Africa. (Copyright D.A. Warrell.)

A

B

Figure 49-8. Russell's vipers. **A,** Western species *(Daboia russelii)* from Chennai, India. **B,** Eastern species *(Daboia siamensis)* from Tharrawaddy, Burma (Myanmar). (Copyright D.A. Warrell.)

Figure 49-9. Indian saw-scaled or carpet viper *(Echis carinatus)* (Tamil Nadu, India) showing its defensive coiling movement during which the rough lateral scales rub together producing a characteristic rasping sound. (Copyright D.A. Warrell.)

Figure 49-10. West African saw-scaled or carpet viper *(Echis ocellatus)* (Kaltungo, Nigeria). (Copyright D.A. Warrell.)

snake") (Fig. 49-13, *Rhabdophis subminiatus*). To envenom a human, the snake must seize, hold onto, and chew the finger of its victim, who is usually a herpetologist.

Atractaspididae

Atractaspididae, genus *Atractaspis* (African and Middle Eastern burrowing asps, stiletto snakes, burrowing or mole vipers or adders, false vipers, or side-stabbing snakes), have long front fangs upon which they impale their victims by a side-swiping motion of the fang protruding from the corner of the partially closed mouth (Fig. 49-14, *Atractaspis aterrima*).[47] In some species, the venom glands are very long—perhaps one-sixth the snake's total length. The Natal black snake *(Macrelaps microlepidotus)* possesses two very large grooved opisthoglyphous fangs at the posterior ends of its maxillae.

Figure 49-11. Pair of arboreal green pit vipers (*Trimeresurus (Popeia)* near *popeiorum:* family Viperidae; subfamily Crotalinae) (Chiang Mai, northeastern Thailand) showing heat-sensitive loreal pit between eye and nostril. (Copyright D.A. Warrell.)

Figure 49-12. Sri Lankan hump-nosed pit viper (*Hypnale hypnale:* family Viperidae; subfamily Crotalinae) (Anuradhapura) showing heat-sensitive loreal pit between eye and nostril and long hinged front fangs erected as when the snake strikes its prey. This species also occurs in the Western Ghats of India. (Copyright D.A. Warrell.)

Elapidae and Viperidae

The venom glands lie behind the eye. Compressor muscles squeeze venom out of the gland through the venom duct to the base of the fang. Venom is transmitted to the tip of the fang through a partially or completely closed canal.

Elapidae

Elapidae (cobras—*Naja;* kraits—*Bungarus;* mambas—*Dendroaspis;* shield-nosed and African coral snakes—*Aspidelaps;*

Figure 49-13. Rear fangs of the dangerous Southeast Asian red-necked keelback (*Rhabdophis subminiatus:* family Colubridae) (Chantaburi, Thailand). (Copyright D.A. Warrell.)

Figure 49-15. Short front fangs of a Papuan taipan (*Oxyuranus scutellatus:* family Elapidae) (Port Moresby, Papua New Guinea). (Copyright D.A. Warrell.)

Figure 49-14. Long front fang of a slender burrowing asp (*Atractaspis aterrima:* family Atractaspididae) (Zaria, Nigeria). (Copyright D.A. Warrell.)

Asian coral snakes—*Calliophis;* African garter snakes—*Elapsoidea;* terrestrial venomous Australasian snakes—e.g., *Acanthophis, Pseudechis, Pseudonaja, Oxyuranus, Notechis;* sea snakes—e.g., *Laticauda, Enhydrina, Hydrophis, Lapemis*) have relatively short, proteroglyphous (fixed) front fangs mounted on a relatively fixed maxilla so that they cannot be folded flat against the jaw (Fig. 49-15, *Oxyuranus scutellatus canni*). However, mambas can rotate their fangs in the sagittal plane because the maxilla has a posterior articulation with the prefrontal bone (Fig. 49-16, *Dendroaspis angusticeps*, and Fig. 49-17, *Dendroaspis polylepis*). This allows the snake to close its mouth despite having unusually long elapid-type fangs and to increase its gape when striking at large prey.

Spitting Elapids. The fangs of African and Asian spitting cobras and the South African ringhals or rinkals *(Hemachatus haemachatus)* have venom channels that turn forward at their point of exit near the tip of the fangs so that a spray of venom can be ejected forward for a meter or more into the eyes of an aggressor (Fig. 49-18, *Naja nigricollis*).[7,178,179] This is a defensive strategy that temporarily blinds the aggressor (e.g., a large, threatening mammal), thus allowing the snake to escape.

Viperidae

Viperidae (vipers, adders, and pit vipers) have highly evolved, long, curved front solenoglyphous (hinged, venom-conducting) fangs, mounted on a rotatable maxilla so that they can be erected when the snake strikes and folded flat against the jaw when not in use. They contain a closed venom channel and resemble hypodermic needles (see Figure 49-12, *Hypnale hypnale*, and Fig. 49-19, *B. rhinoceros*).

Figure 49-16. Eastern green mamba *(Dendroaspis angusticeps)* (coastal Kenya) showing angulation of the maxilla (bearing the fangs) on the prefrontal bone, allowing rotation of the long elapid-type fangs. (Copyright D.A. Warrell.)

Figure 49-17. Mouth of black mamba (Gede, Kenya) *(Dendroaspis polylepis),* showing long fangs and the black coloring of the buccal mucosa that gives this species of mamba its name. Note also the angulation of the maxilla, as in Figure 49-16. (Copyright D.A. Warrell.)

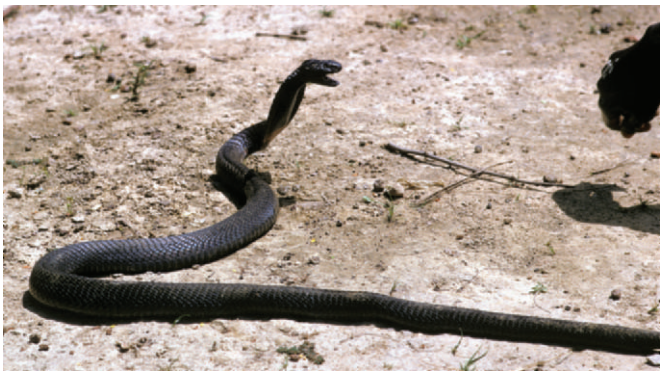


Figure 49-18. Black-necked spitting cobra *(Naja nigricollis)* (Zaria, Nigeria) in the act of "spitting" venom from its fang tips toward the snake charmer's hand. (Copyright D.A. Warrell.)

Figure 49-19. Enormous solenoglyphous front fangs of a West African Gaboon viper (*Bitis rhinoceros:* family Viperidae; subfamily Viperinae) (Cameroun) erected as when the snake strikes its prey. Gaboon vipers have longer fangs than any other snake, up to 8 cm ($3^{1}/_{7}$ in) in length. (Copyright D.A. Warrell.)

VENOM COMPOSITION[34,65]

Snakes have evolved the most complex of all venoms, each venom containing more than 100 different components. The evolutionary origins of these molecules is of great current interest.[26,27] More than 90% of the dry weight is protein, comprising a variety of enzymes, nonenzymatic polypeptide toxins, and

nontoxic proteins. Nonprotein ingredients include carbohydrates and metals (often part of glycoprotein metalloprotein enzymes), lipids, free amino acids, nucleosides, and biogenic amines such as serotonin and acetylcholine.

Enzymes

Eighty percent to 95% of viperid and 25% to 70% of elapid venoms consist of enzymes (molecular weight 13 to 15 kDa), including digestive hydrolases (proteinases, exopeptidase, endopeptidases, phosphodiesterases, and phospholipases), hyaluronidase,[77,78] and activators or inactivators of physiologic processes. Most venoms contain L-amino acid oxidase, phosphomonoesterase, diesterase, 5′-nucleotidase, DNA-ase, NAD-nucleosidase, phospholipase A_2 (PLA_2), and peptidases. Elapid venoms, in addition, contain acetylcholine esterase, phospholipase B, and glycerophosphatase, whereas viperid venoms have endopeptidase, arginine ester hydrolase, kininogenase (which releases bradykinin from bradykininogen), thrombin-like serine proteases, and factor X-activating and prothrombin-activating enzymes. PLA_2 is the most widespread and extensively studied of all venom enzymes. Under experimental conditions, they damage mitochondria, red blood cells, leukocytes, platelets, peripheral nerve endings, skeletal muscle, vascular endothelium, and other membranes, and they produce presynaptic neurotoxic activity, opiate-like sedative effects, and the autopharmacologic release of histamine. Hyaluronidase promotes the spread of venom through tissues. Proteolytic enzymes (endopeptidases or hydrolases) are responsible for local changes in vascular permeability, leading to edema, blistering, bruising, and necrosis. Metalloproteinases cause local and systemic hemorrhage and local myonecrosis, blistering, and edema through their actions on vascular endothelium, platelets,[45] muscle, and other tissues. L-amino acid oxidase may have a digestive function.

Neurotoxins

Polypeptide toxins are low–molecular-weight, nonenzymatic proteins found almost exclusively in elapid venoms. Postsynaptic (curaremimetic) or α-neurotoxins are either "short" (60 to 62 amino acids) or "long" (66 to 74 amino acids, such as α-bungarotoxin and cobrotoxin). They bind to the α_1 component of nicotinic acetylcholine receptors at the motor endplates of skeletal muscles, eventually causing generalized flaccid paralysis and death from bulbar and respiratory muscle weakness. They have a distinctive "three-finger" structure, complementary in shape to their receptor, and have also been found in some colubrid venoms.[25,58]

Presynaptic phospholipases A_2 or β-neurotoxins, such as β-bungarotoxin, crotoxin, and taipoxin, contain about 120 to 140 amino acid residues and a phospholipase A subunit. These damage nerve endings at neuromuscular junctions, targeting voltage-gated potassium channels, causing sequential suppression, enhancement, and finally complete failure of acetylcholine release. The resulting paralysis is clinically indistinguishable from that caused by postsynaptic toxins, except that the latter may be ameliorated by anticholinesterases such as edrophonium or neostigmine. Some of the neurotoxic PLA_2s and other phospholipases have myotoxic activity.

Mamba Toxins

Mamba *(Dendroaspis)* venoms contain a number of unusual neurotoxins.[34,65] Dendrotoxins (59-amino-acid proteins), which bind to voltage-gated potassium channels at nerve endings and cause acetylcholine release, and calciscludine, which blocks calcium channels, are both "pear" fold structures. Two "three-finger" neurotoxins are unique to mamba venoms. Fasciculins (61-amino-acid residues) inhibit some acetylcholinesterases, causing persistent muscle fasciculations, while calciseptine binds to calcium channels.

Krait Bungarotoxins

Krait venoms have proved to be an important source of neurotoxins for experimental neuropharmacologists. They include presynaptic PLA_2 and β-bungarotoxins, as well as postsynaptic α-bungarotoxins and κ-bungarotoxins that bind to some specific nicotinic acetylcholine receptors in the brain and various ganglia.

Biogenic amines, such as histamine and 5-hydroxytryptamine, found particularly in viper venoms, may contribute to local pain and permeability changes at the site of a snakebite.

Venom Potency and Lethality

The activities of different venoms can be compared in the laboratory using in vitro and animal in vivo assays. The only practicable index of lethality is the median lethal dose (LD_{50}) measured in mice of specified strain and size (Table 49-1). However, this value cannot easily be extrapolated to give any reliable idea of the lethal dose or even the relative lethality of different venoms in humans. Quite apart from the marked species variations in vulnerability to venoms, the mode of death in different animals may not be the same. For example, in mice, the venom of the black-necked spitting cobra *(N. nigricollis)* causes death with convulsions following paralysis (intravenous LD_{50} 0.40 to 1.37 mg/kg), but humans envenomed by this species do not show neurotoxicity.[118,155] A series of standard World Health Organization (WHO) assays for assessing different aspects of venom activity has been developed.[113] It is to be hoped that an assay in insensate chick embryos,[92] and increasing use of tissue culture assays, may reduce the need for studies in living animals.

Variations in the Quantity and Composition of Venom Injected by a Snakebite

In human victims of snakebite, the amount and composition of venom injected by the snake is the most important, but indeterminate, factor influencing evolution of envenoming and prognosis. Apart from inferring the quantity and quality of injected venom from the range and severity of clinical manifestations of envenoming, the only currently available objective method of assessment is to measure venom antigenemia when the patient is admitted to the hospital. This has been found to correlate with clinical severity in the case of several different species of snake, including European vipers,[3] Burmese Russell's viper,[129] and Martinican lancehead *(Bothrops lanceolatus)*.[117]

Box 49-1 summarizes the factors that determine severity of envenoming. The amount of venom injected at a bite seems to be very variable, so that a proportion of human victims of snakebite, more than half in some series, show negligible or no signs of envenoming, even though the snake was identified as being venomous and there were obvious puncture marks indicating that the fangs had penetrated the skin.[70,73] These so-called dry bites have led to the belief that snakes can somehow control the amount of venom injected, according to the size of their natural prey,[36] and that they can inflict a "defensive bite" without expending venom when they strike at an enemy. When

TABLE 49-1. Selected Snake Venoms: Venom Yield on Milking and Mouse Lethality in Roughly Declining Order of Potency

SPECIES	VENOM YIELD (DRY WEIGHT) (mg) RANGE	AVERAGE	MAXIMUM	MOUSE MEDIAN LETHAL DOSE (LD_{50}) (mg/kg) INTRAVENOUS (iv) OR SUBCUTANEOUS (sc)
Western/inland taipan or small-scaled snake *Oxyuranus microlepidotus*		44	110	0.025 (sc)
Beaked sea snake *Enhydrina schistosa*	7–20			0.01 (iv), 0.164 (sc)
Papuan taipan *Oxyuranus scutellatus canni*			400	0.05 (sc)
Eastern brown snake *Pseudonaja textilis*		8	67	0.053 (sc)
Russell's viper *Daboia russelii*	130–250			0.08 (iv)
Common krait *Bungarus caeruleus*	8–20			0.09 (iv)
Eastern taipan *Oxyuranus* s. *scutellatus*		40	120	0.099 (sc)
Mainland tiger snake *Notechis scutatus*		35	189	0.118 (sc)
Black mamba *Dendroaspis polylepis*				0.26–2.9 (iv)
Indian cobra *Naja naja*		169	610	0.13–0.42 (iv)
Green mamba *Dendroaspis angusticeps*	60–95			0.45 (iv)
European adder *Vipera berus*	6–18			0.55 (iv)
King cobra *Ophiophagus hannah*		421	1000	1.80 (sc)
Eastern diamond back rattlesnake *Crotalus adamanteus*	370–720	410	848	11.4 (sc), 1.68 (iv)
Saw-scaled viper *Echis carinatus*	20–35			2.30 (iv)

Sources: Kochva E: London, Academic Press, 1978, pp 99, 442–447; Sutherland SK, Tibballs J: Melbourne, Oxford University Press, 2001, pp 15–16.

Box 49-1. Determinants of Snakebite Incidence and Severity of Envenoming

INCIDENCE OF BITES

1. Frequency of contact between snakes and humans, depends on:
   - Population densities (snake, human)
   - Diurnal and seasonal variations in activity
   - Types of behavior (e.g., human agricultural)
2. Snakes' "irritability"—readiness to strike when alarmed or provoked—varies with species

SEVERITY OF ENVENOMING

1. Dose of venom injected—depends on mechanical efficiency of bite and species and size of snake
2. Composition and hence potency of venom—depends on species and, within a species, the geographic location, season, and age of the snake
3. Health, age, size, and specific immunity of human victim
4. Nature and timing of first aid and medical treatment

humans are bitten, the circumstances of the bite and hence the mechanical efficiency with which the snake is able to bring its venom apparatus into action against an unexpected and abnormal target may be important in determining how much venom is injected. The idea that a snake might exhaust its supply of venom by a recent strike and therefore be less venomous after eating or after striking another victim has been refuted. Snakes do not exhaust the contents of their venom glands at the first strike. The proportion of unexpended venom after the first strike was found to be 0% to 85% for the Australian death adder *(Acanthophis)*, 5% to 81% for Australian tiger snakes *(Notechis)*, 40% for the Indian cobra *(Naja naja)*, and 30% for the saw-scaled viper *(E. carinatus)*.[47] Palestine vipers *(Daboia palaestinae)* continue to deliver substantial quantities of venom after nine or more consecutive strikes.[47] In several reports of consecutive strikes by a single snake on several human victims, the first victim was not always the most severely envenomed. In the case of Russell's viper bites in Burma Myanmar, snakes that had recently fed (those in which a rodent was found in their stomach) inflicted bites that were no less venomous than those with empty stomachs.[129] Although the largest specimens do not always inflict the most highly venomous bites, there is a tendency, in most species that have been studied, for the severity of local and systemic envenoming to increase with the size of the individual snake responsible.[130] Venom yield has been shown to increase with ambient temperature, but no definite seasonal variation has been documented.

Intraspecies Variation in Venom Composition[145]

Age (ontologic) variations: In many species of snake, venom composition has been found to change as the snake grows older, presumably to accommodate a changing range of prey species. Most of the species that have been studied are from the Americas (see Chapter 48), but in the rest of the world, Indian cobra *(Naja naja)* venom has been shown to have decreasing lethality with increasing age, while venoms of the saw-scaled viper *(E. carinatus)* and black-necked spitting cobra *(N. nigricollis)* show decreasing lethality despite increasing protein concentration.[145] Seasonal variation in the venom composition of a particular species has been suspected but not proven. There is now abundant literature describing variations in venom lethality, composition, and activities between individuals of a

TABLE 49-2. Species of Snake Probably Responsible for Most Deaths and Morbidity Outside the Americas

GEOGRAPHIC AREA	SCIENTIFIC NAME	COMMON NAME
Europe	*Vipera* spp.	Vipers, adders
	Vipera ammodytes	Long-nosed viper
Africa	*Echis* spp.	Saw-scaled or carpet vipers
	Bitis arietans	Puff adder
	Naja nigricollis, Naja mossambica, among others	African spitting cobras
	Naja haje, Naja anullifera,	Egyptian and snouted cobras
	Naja anchietae	
	Dendroaspis spp.	Mambas
Asia, Middle East	*Echis* spp.	Saw-scaled or carpet vipers
	Macrovipera lebetina	Snub-nosed viper
	Daboia palaestinae	Palestine viper
	Naja oxiana	Oxus cobra
Asia, Indian subcontinent, Southeast Asia	*Naja naja, Naja kaouthia,*	Asian cobras
	Naja siamensis	
	Bungarus spp.	Kraits
	Daboia russelii, Daboia siamensis	Russell's vipers
	Calloselasma rhodostoma	Malayan pit viper
	Echis carinatus	Saw-scaled or carpet vipers
	Trimeresurus and related genera	Arboreal and other pit vipers
Asia, Far East	*Naja atra*	Chinese cobra
	Bungarus multicinctus	Chinese krait
	Trimeresurus (Protobothrops) flavoviridis	Japanese habu
	Trimeresurus (Protobothrops) mucrosquamatus	Chinese habu
	Gloydius blomhoffii subsp.	Mamushis
Australasia, New Guinea	*Acanthophis* spp.	Death adders
	Pseudonaja spp.	Brown snakes
	Pseudechis spp.	Black snakes
	Notechis spp.	Tiger snakes
	Oxyuranus scutellatus subspp.	Taipans
	Micropechis ikaheka	New Guinean small-eyed snake

particular species at the same geographic focus or from different places within the snake's geographic range.[17,74,145] In the case of the Malayan pit viper *(Calloselasma rhodostoma),* available prey species seemed to have had a major evolutionary influence on venom composition in different parts of its geographic range.[17]

Effect of the Site and Depth of the Bite

In experimental animals, the route of venom administration (topical, intradermal, subcutaneous, intramuscular, intraperitoneal, intravenous, intracerebral) affects the speed, pattern, and lethality of envenoming. This is virtually impossible to study clinically, but in anecdotal cases of human envenoming, in which systemic effects develop rapidly after a bite with minimal local signs, it was assumed that venom entered the bloodstream directly as a result of a fortuitous intravenous puncture.

EPIDEMIOLOGY OF SNAKEBITE

Snake Species Responsible for Most Bites

Table 49-2 lists the species responsible for most snakebite deaths and severe morbidity outside the Americas. Fortunately, some species notorious for the potency of their venom (e.g., many species of sea snakes and Australasian elapids), or their great size (e.g., the south Asian king cobra, *O. hannah,* which can grow longer than 19 feet [5.79 m], and Gaboon vipers, *Bitis gabonica* and *B. rhinoceros*) rarely bite humans. The African night adders (genus *Causus*),[162] Asian green pit vipers (genus *Trimeresurus*),[41,132] and Sri Lankan and southwest Indian humpnosed pit viper *(H. hypnale)*[19,439] are responsible for many bites, but severe envenoming is rare or uncommon.

DISTRIBUTION OF VENOMOUS SNAKES

Venomous snakes are distributed from sea level up to high altitudes. The Himalayan pit viper, *Gloydius himalayanus,* normally confined below 1500 m (4,900 feet), has been recorded at the base of the Dharmsala glacier in northern India as high as 4877 m (16,000 feet).[91] The European adder, *Vipera berus,* is very widely distributed, from England, Scotland, and Wales in the west, across Europe and Asia to North Korea and Sakhalin Island in the east, and north into Scandinavia and the Arctic Circle. No other venomous species is found in the Arctic and none occurs in the Antarctic. Crete, Ireland, Iceland, Madagascar, New Caledonia, New Zealand, and Hawaii. Most of the islands of the western Mediterranean Sea and Atlantic and Pacific Oceans are free of venomous snakes.

Sea snakes occur in the Persian Gulf and Indian and Pacific Oceans between latitudes 30°N and 30°S, as far south as New Zealand and Tasmania and as far north as Siberia (yellow-bellied or pelagic sea snake *Pelamis platurus*).[59,82,141] The beaked sea snake *(Enhydrina schistosa)* and *Hydrophis* species frequent estuaries and have been found 130 to 160 km (80 to 100 miles) up rivers. In Papua New Guinea, *E. schistosa* was taken in the Ramu River, 30 km (19 miles) from the sea, where it had caused

fatal envenoming. In freshwater lakes, *Hydrophis semperi* occurs in Lake Taal (also called Lake Bombon) in the Philippines and *E. schistosa* in Ton Ley Sap in Cambodia. There are no sea snakes in the Atlantic Ocean or the Red Sea.[141]

SNAKEBITE MORTALITY AND MORBIDITY

In the tropical countries where snakebite is most common, there are few reliable data. The only serious attempt to assess global snakebite mortality was by Swaroop and Grabb for the WHO in 1954.[105] Subsequent work has revealed gross underreporting of deaths in this study, for example, in Nigeria[151] and Thailand.[139,142,143] One reason is that records of patients treated by traditional methods are missing from official hospital-based statistics and deaths reported at the hamlet or district level may not be sent on to ministry headquarters. Population surveys[35,76,94,98,122] have given a far more accurate picture of snakebite incidence, morbidity, and mortality. The prevalence of chronic morbidity following snakebite is unknown, but it is possible that, for every person killed by snakebite, one or two may survive with a long-term physical handicap.

Asia

The highest recorded incidence was 162 deaths per 100,000 population per year, determined in the Eastern Terai area of Nepal.[94] In this study, only 20% of deaths occurred in hospitals. Increased risk of fatality was associated with being bitten inside the house while resting between midnight and 6:00 AM, which suggests bites by the common krait *(B. caeruleus)* (see later discussion). Other risk factors were an initial visit to a traditional healer and delayed transport to the hospital. An estimated 15,000 to 20,000, but possibly as many as 100,000, people die each year from snakebite in India.[105] In Barddhaman (Burdwan) District, West Bengal, a field survey in randomly selected villages suggested that among the total population of nearly 5 million people, nearly 8000 were bitten and 800 killed by snakes each year.[35] In the 1930s, the annual snakebite mortality reported in Burma exceeded 2000 (15.4 per 100,000 population).[105] Thirty years later, it was still estimated to exceed 1000 (3.3 per 100,000) per year. Russell's viper *(D. siamensis)* bite was once the fifth most important single cause of all deaths in Burma.[105] In 1984, about 900 snakebite deaths were recorded in Sri Lanka, an incidence of 6 per 100,000 population per year. In the Amami and Okinawa Islands of Japan, there were 5488 bites by the habu *(Trimeresurus [Protobothrops] flavoviridis)*, resulting in 50 deaths during the 9 years from 1962 to 1970.[88] The highest incidence of bites on one of the islands was 4.6 per 1000 population per year.

Africa

In the Benue Valley of northeastern Nigeria, the incidence of snakebites was 497 per 100,000 population per year with a mortality of 12.2%.[151] Most bites and deaths were attributed to saw-scaled vipers *(E. ocellatus)*. In Bandafassi, southeast Senegal, in a population of 10,509, snakebite mortality was 14 per 100,000 population per year. Saw-scaled vipers *(E. ocellatus)*, puff adders *(B. arietans)*, and spitting cobras *(Naja katiensis)* were implicated.[121] A community survey of snakebites by the black-necked spitting cobra *(N. nigricollis)* in Malumfashi, northern Nigeria, found that in a population of 43,500 there were 15 to 20 bites per 100,000 population per year. Only 8.5% of the victims had visited a hospital. The case-fatality rate was 5%, and 19% of survivors had persistent physical disability from the locally necrotic effects of the venom.[76] In Kenya, a preliminary survey based on Ministry of Health, hospital, clinic, and dispensary records in Kakamega and western Kenya, Lake Baringo, Laikipia, Kilifi, Malindi, and northern Kenya suggested an overall average frequency of snakebite of 14 (range 2 to 68) per 100,000 population per year with a minimum death rate of 0.45 per 100,000 population per year. Puff adders, black mambas, and spitting cobras were responsible for the fatalities.[15] However, a community-based study on the coast in Kilifi district discovered 15 adult snakebite fatalities per 100,000 population per year.[98]

Oceania

In Australia, 1000 to 2000 bites occur per year with an average of three to four deaths per year. In Papua New Guinea, the incidence of bites was 215 and of deaths was 7.9 per 100,000 population per year in Central Province, whereas in Kairuku subprovince, there were 526 bites per 100,000 population per year.[56] The number of deaths may have increased recently because of inadequate antivenom supplies.

Europe

In Britain, there are more than 200 adder *(V. berus)* bites each year, but there have been only 14 deaths during the last hundred years, the last in 1975.[81,144,147] There were 44 deaths caused by this species in Sweden between 1911 and 1978; in Finland, there were 21 deaths in 25 years with an annual incidence of almost 200 bites.

Risks to Indigenous Nomadic Hunter-Gatherers

The Hadza people in Tanzania are at great risk from snakebite as they hunt rock hyraxes, which are also the prey of black mambas *(D. polylepis)* (Fig. 49-20, *D. polylepis*). The nomadic Phi Tong Luang of northeastern Thailand and Laos also fall victim to snakebites. However, among many indigenous ethnic groups such as Congo pygmies and Australian aboriginal peoples, snakebites are remarkably uncommon.

Snakebite as an Occupational Disease

In tropical countries, farmers, plantation workers, herders, and hunters are most frequently bitten.[70] Rice farmers in Burma, Sri Lanka, and central Thailand tread on Russell's vipers or in-

Figure 49-20. Black mamba (*Dendroaspis polylepis:* family Elapidae) (Harare, Zimbabwe), a specimen 3 m (9 ft, 10 in) long. (Copyright D.A. Warrell.)

advertently pick them up in a handful of paddy during the harvest.[70] Cobras *(Naja philippinensis)* are an occupational hazard to rice farmers in the Philippines.[169] The Chinese predilection for snakes and their organs, both as food and medicines, has resulted in many bites in snake restaurants and snake farms in China and southeast Asia. In the savanna of West Africa, farmers are bitten by *Echis* species as they dig the fields at the start of the rainy season.[151] Rubber-tappers in southeast Asia tread on Malayan pit vipers *(Calloselasma rhodostoma)* in the dark and are bitten as they make their early morning rounds of the rubber trees.[158] This species is also responsible for numerous fatal bites among coffee and rubber plantation workers in Vietnam, especially in Song-be province.

Sea snakebites were an occupational hazard of fishermen in those parts of southeast Asia where hand nets were used.[84] Mechanization of fishing methods in this region has resulted in a dramatic decrease in sea snakebites, but they still occur along the coast of south Vietnam.[141] The beaked sea snake *(E. schistosa)* has caused most bites and deaths. Other common and medically important species are *Hydrophis cyanocinctus, Hydrophis spiralis,* and *Lapemis curtus.*

Bites by Snakes Kept in Captivity

Especially in the more industrialized countries, venomous snakes are increasingly popular as pets. Many are kept illegally. Most bites are inflicted on the hands when the snakes are picked up by an owner who is under the influence of alcohol or recreational drugs.[150a]

Risk to Wilderness Travelers

The risk of snakebite is a major anxiety among members of expeditions and wilderness travelers. Provided that sensible precautions are taken (see later text), the risk is small. However, for field biologists working in snake-infested areas, especially those who are interested specifically in finding and capturing snakes, the risk is far higher. The death of Joe Slowinski in 2001 illustrated the mortal danger of handling snakes in places far from medical help. He was an expert herpetologist from the California Academy of Sciences who died at Rat Baw in remote northern Burma, 32 hours after being bitten by a 30-cm juvenile krait (*Bungarus* near *multicinctus*) that he pulled out of a bag with his bare hand, believing it to be the harmless krait-mimic *Dinodon septentrionalis.*[56a] Snakebite is a rare accident for ordinary backpackers, but people have been bitten even while walking down the main track into the Grand Canyon.

Circumstances in Which Snakebites Occur

Unprovoked attacks are unusual, but snakes will bite if they are cornered or feel threatened. Some species, notably *B. caeruleus* (Fig. 49-21) in India[85] and Sri Lanka, *B. candidus* in southeast Asia,[159] *N. nigricollis* in western Africa,[155] and *N. mossambica* in South Africa,[119] enter human dwellings at night in pursuit of their prey (rodents, lizards, toads) and may strike at a person who moves in his sleep. Epidemics of snakebite can be caused by flooding, the building of new roads through jungles, and implementation of irrigation schemes.

SYMPTOMS AND SIGNS OF ENVENOMING[64,142,143,150]

Fear as well as direct and indirect actions of venom, side effects of treatment, and complications contribute to the symptomatology of snakebite. The principal clinical features of envenoming are summarized in Table 49-3.

Figure 49-21. Common Indian krait *(Bungarus caeruleus)* (Hyderabad, India). (Copyright D.A. Warrell.)

Local Swelling

In the bitten limb, increased vascular permeability leads to swelling and bruising. The factors responsible include endopeptidases, metalloproteinases, hemorrhagins, membrane-damaging polypeptide toxins, phospholipases, and endogenous autacoids, such as histamine, 5-hydroxytryptamine, and kinins, released by the venom. Venoms of some Viperidae, such as *D. russelii, D. siamensis,* and *V. berus,* can also produce a generalized increase in vascular permeability resulting in pulmonary edema, serous effusions, conjunctival and facial edema, and hemoconcentration.

Local tissue necrosis results from direct action of myotoxins and cytotoxins, ischemia caused by thrombosis, and compression of blood vessels by first aid methods such as tight tourniquets or by swollen muscle within a tight fascial compartment. Myotoxins are proteins that can damage the muscle cell plasma membrane directly. Most are phospholipases A_2, either enzymatically active (aspartate-49) or enzymatically inactive (lysine-49). Cobra cardiotoxins are low–molecular-weight polypeptides with cytotoxic action.

Hypotension and Shock

Profound hypotension is part of the autopharmacologic syndrome that may occur within minutes of bites by *Daboia palaestinae, V. berus, D. russelii,* and *Actractaspis engaddensis.* Presumably this is caused by release of vasodilating autacoids. Oligopeptides in many Viperidae venoms inhibit bradykinin-deactivating enzymes and angiotensin-converting enzymes (ACEs) and were the models for synthetic ACE inhibitors used to treat hypertension.[20] Snake handlers may become sensitized to snake venoms and can develop life-threatening anaphylactic reactions within minutes of being bitten. Leakage of plasma or blood into the bitten limb and elsewhere or massive gastroin-

TABLE 49-3. Clinical Manifestations of Snakebite Envenoming with Suggested Pathophysiology

SIGN OR SYMPTOM	PROPOSED MECHANISM
Local Envenoming	
Swelling, bruising and blistering	Increased vascular permeability (digestive enzymes, metalloproteinases, PLA_2 myotoxins)
Enlarged, painful, and tender lymph nodes draining the bitten area	Absorption and passage of large-molecular-weight venom toxins via the lymphatic system
Tissue necrosis/gangrene	Venom myotoxins, cytotoxins, metalloproteinases, for example; secondary ischemia (iatrogenic from tourniquet), thrombosis, intracompartmental syndrome
Peripheral nerve lesion	Iatrogenic from pressure of tourniquet (e.g., lateral popliteal nerve), compartment syndrome, hematoma
Systemic Envenoming	
Hypotension and shock	Hypovolemia from extravasation of blood/plasma into the swollen limb Vasodilatation by venom toxins and autacoids (ACE inhibitors and bradykinin-potentiating peptides) Myocardial damage, cardiac arrhythmias Rarely, venom hypersensitivity anaphylaxis
Intravascular hemolysis	PLA_2-mediated, complement (cobra venom C3b) activation or microangiopathic
Renal failure: acute tubular necrosis	Hypotension, disseminated intravascular coagulation, direct nephrotoxicity, pigment nephropathy (hemoglobinuria, myoglobinuria) associated with microangiopathic hemolysis and secondary to hyperkalemia.
Neurotoxicity	Presynaptic and/or postsynaptic neuromuscular block
Generalized rhabdomyolysis	Myotoxic PLA_2

testinal hemorrhage may cause hypovolemia after viper bites. Vasodilatation, especially of splanchnic vessels, and a direct effect on the myocardium may contribute to hypotension after viper bites.

Bleeding and Clotting Disturbances[42,45,172]

Hemostatic disturbances are an important feature of envenoming by vipers, pit vipers, Australasian elapids, and dangerously venomous colubrids.

Snake venoms affect hemostasis in several ways:

- **Procoagulant enzymes** activate intravascular coagulation, producing consumption coagulopathy and incoagulable blood. For example, procoagulants in the venom of Colubridae, *Echis* species and *Notechis scutatus* activate prothrombin, whereas those in venoms of *Daboia russelii* and *D. siamensis* activate factors V and X.
- **Thrombin-like enzymes** in venoms of many crotaline snakes have a direct action on fibrinogen. Some venoms cause defibrinogenation by activating the endogenous fibrinolytic (plasmin) system.
- **Anticoagulant** activity is attributable to venom phospholipases.
- **Platelet activation or inhibition and thrombocytopenia** are common accompaniments of systemic envenoming.

Activators of platelet agglutination include alboaggregin B (*T. [Cryptelytrops] albolabris*), which acts through receptors GPVI and GPIb. Aggretin (*Calloselasma rhodostoma*) activates platelets via receptors GPIb-IX-V and GPIa-IIa. Rhodocytin (or aggretin, *C. rhodostoma*) is a platelet agonist independent of GPIb, GPIa-IIa or GPVI, which activates via CLEC-2. Trimucytin (*T. [Protobothrops] mucrosquamatus*) activates platelets via GPIa-IIa, independent of the GPIa-IIa I domain (collagen binding site). Convulxin (*Crotalus durissus*), ophioluxin (*Ophiophagus hannah*), and alborhagin (*T. [Cryptelytrops] albolabris*) activate platelets via GPVI. Proatherocytin (*Proatheris superciliaris*) is a platelet receptor PAR-1 agonist.

Platelet inhibitors include mocarhagin (*Naja mossambica*) and jararhagin (*Bothrops jararaca*), which are metalloproteinases that cleave GPIbα and GPIa-IIa respectively, thus inhibiting platelet responses depending on those receptors.

Rhodocetin (*C. rhodostoma*) inhibits platelets via interaction with GPIa-IIa. Arg-Gly-Asp (RGD) tripeptide sequence–containing venom peptides such as trigramin, echistatin, contortrostatin, flavoviridin are powerful inhibitors of the fibrinogen receptor GPIIb-IIIa.

Few studies of platelet function have been attempted in envenomed humans. In patients bitten by Malayan pit vipers and green pit vipers (*T. [Cryptelytrops] albolabris*) there was initially inhibition of platelet agglutination followed by activation and the appearance of circulating clumps of platelets.[41,42]

- **Hemorrhagins:** in the absence of trauma, defibrin(ogen)ation induced by venom coagulants, such as ancrod (Arvin, Arwin) from *C. rhodostoma* venom, is a relatively benign state.[83] **Potentially lethal spontaneous systemic bleeding** is attributable to distinct venom components, haemorrhagins, which damage vascular endothelium. These metalloproteinases are zinc metalloendopeptidases (reprolysins), some of which include disintegrin-like, cysteine-rich and lectin domains.[44] The combination of incoagulable blood, thrombocytopenia and vessel wall damage will result in massive bleeding, a common cause of deaths from viper bites.

Intravascular Hemolysis

Although most snake venoms are hemolytic in vitro, this effect is rarely of clinical significance. However, envenoming by Russell's vipers (in India and Sri Lanka), some Australasian elapids, and members of the colubrid genera *Dispholidus, Thelotornis,* and *Rhabdophis* may be associated with massive intravascular hemolysis contributing to renal failure. Snake venom–induced disseminated intravascular coagulation may result in deposition of fibrin on activated vascular endothelium,

producing microangiopathic hemolysis (schistocytes, helmet cells, fragmented erythrocytes), renal failure, and a clinical picture reminiscent of hemolytic uremic syndrome, for example, after bites by the desert horned viper *(Cerastes cerastes)*[90] and Australian western brown snakes (*Pseudonaja* spp.).

Complement Activation[134,155]

Elapid and some colubrid venoms activate complement via the alternative pathway ("cobra venom factor" is the snake's C3b), whereas some viperid venoms activate the classic pathway.[134] This has been documented in human victims of snakebite.[153,155] Complement activation may in turn affect platelets, the blood coagulation system, and other humoral mediators.[64,134]

Renal Failure

Renal failure is a potential complication of severe envenoming, even by species that usually cause only mild envenoming, such as *T. (Cryptelytrops) albolabris,* the hump-nosed pit viper *(H. hypnale),*[19] *V. berus,*[81] and *Cerastes cerastes.*[90] However, it is a common event and cause of death following bites by Russell's vipers[70,96,116,120] and sea snakes.[82,96,141] Possible mechanisms of acute tubular necrosis are prolonged hypotension and hypovolemia, disseminated intravascular coagulation, a direct toxic effect of the venom on the renal tubule, hemoglobinuria, myoglobinuria, and hyperkalemia. Russell's viper venom produces hypotension, disseminated intravascular coagulation, direct nephrotoxicity,[80] and, in Sri Lanka and India, intense intravascular hemolysis.[73] In Burmese patients envenomed by Russell's vipers *(D. siamensis),* high urinary concentrations of beta-2 microglobulin, retinal binding protein, and N-acetyl glucosaminidase suggested failure of proximal tubular reabsorption and tubular damage.[120] High plasma concentrations of active renin suggested that renal ischemia with activation of the renin-angiotensin system was involved in the development of renal failure. A marked but transient capillary and glomerular leak of albumin was an early sign of oliguric renal failure.[116] A large variety of renal histologic changes has been described after snakebite, including proliferative glomerulonephritis, toxic mesangiolysis with platelet agglutination, fibrin deposition,ischemic changes, acute tubular necrosis, distal tubular damage ("lower nephron nephrosis") suggesting direct venom nephrotoxicity, and bilateral renal cortical necrosis with subsequent calcification[96] (Box 49-2). Preexisting chronic renal disease and the effects of antivenom (serum sickness) may confuse interpretation.

Box 49-2. Renal Histopathologic Changes in Human Snakebite Victims

GLOMERULAR CHANGES

- Toxic mesangiolysis, platelet aggregation, mesangial proliferation
- Proliferative glomerulonephritis; endocapillary, exocapillary, mesangiocapillary
- Fibrin deposition
- Ischemia

TUBULAR CHANGES

- Direct nephrotoxicity "lower nephron nephrosis"
- Secondary ischemic changes (fibrin deposition)
- Acute tubular necrosis (pre-renal)
- Cortical infarction, calcification

Neurotoxicity

The neurotoxic polypeptides and phospholipases A_2 of snake venoms cause paralysis by blocking transmission at the neuromuscular junction.[123] Paralytic symptoms are characteristic of envenoming by most elapids, such as kraits, coral snakes, mambas, and cobras, but not of the African spitting cobras (e.g., *N. nigricollis, N. nubiae, N. pallida, N. mossambica*).[118,155] Venoms of terrestrial Australasian snakes,[10–12,14,104] sea snakes, and a few species of Viperidae, notably the Chinese and Japanese mamushis (*Gloydius/Agkistrodon blomhoffii* subspp.), Sri Lankan and South Indian *D. russelii,* the southern African berg adder *(Bitis atropos),* and some other small *Bitis* species of southern Africa *(B. peringueyi, B. xeropaga),* are also neurotoxic in humans. Patients with paralysis of the bulbar muscles may die of upper airway obstruction or aspiration, but the most common mode of death after neurotoxic envenoming is respiratory paralysis. By prolonging the activity of acetylcholine at neuromuscular junctions, anticholinesterase drugs may improve paralytic symptoms in patients bitten by snakes with neurotoxins that are predominantly postsynaptic in their action (e.g., Asian cobras and Australasian death adders [genus *Acanthophis*]). Some patients bitten by elapids or vipers are unphysiologically drowsy in the absence of respiratory or circulatory failure. This is unlikely to be an effect of neurotoxic polypeptides, which do not cross the blood–brain barrier. It may be caused by endogenous opiates released by a venom component. Intracerebral injection of β-RTX (receptor-active protein), a potent antagonist of opiate receptors from *D. russelii* venom, produced sedation in rats.[34]

Generalized Rhabdomyolysis

Generalized rhabdomyolysis, with release into the bloodstream of myoglobin, muscle enzymes, uric acid, potassium, and other muscle constituents, is an effect in humans of PLA_2 presynaptic neurotoxins of most species of sea snakes,[82] many of the terrestrial Australasian elapids[104,171] such as tiger snake (*Notechis scutatus* and *N. ater*), king brown or mulga snake *(Pseudechis australis),* taipan *(O. scutellatus),* rough-scaled snake *(Tropidechis carinatus),* Australian small-eyed snake *(Rhinoplocephalus/Cryptophis nigrescens),*[104,171] and possibly some species of krait *(Bungarus)* and some species of Viperidae, such as Sri Lankan Russell's viper *(D. russelii).*[73] Patients may die of bulbar and respiratory muscle weakness, acute hyperkalemia, or renal failure.

Venom Ophthalmia[72,142,143,160]

Venoms of the spitting cobras and rinkhals are intensely irritant and even destructive on contact with mucous membranes such as the conjunctivae and nasal cavity. Corneal erosions, anterior uveitis, and secondary infections may result.

CLINICAL PATTERN OF ENVENOMING BY DIFFERENT TAXA OF VENOMOUS SNAKES

Colubridae (Back-Fanged Snakes)[142,143]

Species Capable of Causing Fatal Envenoming

In Africa, the boomslang *(Dispholidus typus)* (Fig. 49-22) and the bird, twig, vine, or tree snake (*Thelotornis kirtlandii* and

Figure 49-22. Boomslang *(Dispholidus typus)* (Johannesburg, South Africa), an arboreal colubrid snake capable of fatally envenoming humans. (Copyright D.A. Warrell.)

Figure 49-23. Bird, twig, vine, or tree snake *(Thelotornis capensis)* (Johannesburg, South Africa), an arboreal colubrid snake capable of fatally envenoming humans. (Copyright D.A. Warrell)

Thelotornis capensis) (Fig. 49-23) have caused fatalities, including among their victims two famous herpetologists, Karl P. Schmidt (bitten by *Dispholidus typus*) and Robert Mertens (bitten by *Thelotornis kirtlandi*). In Asia, the Japanese yamakagashi *(Rhabdophis tigrinus)* and the Southeast Asian red-necked keelback *(R. subminiatus)* (see Figure 49-13; Fig. 49-24) have caused severe or fatal envenoming.

Species Capable of Causing Systemic Envenoming

In Europe, the Montpellier snake *(Malpolon monspessulanus)* has been reported to cause neurotoxic symptoms. In Oceania, the brown tree snake *(Boiga irregularis)*, exported from Australia and New Guinea to Guam, has been known to attack and possibly envenom infants.[24]

Figure 49-24. Red-necked keelback *(Rhabdophid subminiatus)* (Chantaburi, Thailand), a dangerously venomous colubrid snake of Southeast Asia. (Copyright D.A. Warrell.)

If these colubrid snakes are able to engage their rear fangs and chew for 15 seconds or longer, severe envenoming may result. Envenoming by *D. typus*, *Thelotornis* spp., *R. tigrinus*, and *R. subminiatus* gives rise to similar symptoms with onset that may be delayed for many hours or even days after the bite.[142,143] There is nausea, vomiting, colicky abdominal pain, and headache. Bleeding develops from old and recent wounds, such as venipunctures, and there is spontaneous gingival bleeding, epistaxis, hematemesis, melena, subarachnoid or intracerebral hemorrhage, hematuria, and extensive ecchymoses. Intravascular and microangiopathic hemolysis have been described. Most fatalities followed renal failure from acute tubular necrosis, many days after the bite. Local effects of the venom are usually trivial, but several patients showed local swelling; one person bitten by *D. typus* had massive swelling with blood-filled bullae. Investigations reveal incoagulable blood, defibrination, elevated fibrin(ogen) degradation products (FDPs), severe thrombocytopenia, and anemia. These clinical features are explained by disseminated intravascular coagulation triggered by a venom prothrombin activator.

Atractaspididae, Genus *Atractaspis* (Burrowing Asps or Stiletto Snakes)

Species Capable of Causing Fatal Envenoming

Only three of the 17 species in this genus, *A. microlepidota* (see Figure 49-14; Figs. 49-25 and 49-26), *A. irregularis*, and *A. engaddensis*, have proved capable of killing humans.[3a,140,142,162] Local effects include pain, swelling, blistering, necrosis, tender

Figure 49-25. Small-scaled burrowing asp (*Atractaspis microlepidota,* family Atractaspididae) (Watamu, Kenya), a snake capable of fatally envenoming humans. (Copyright D.A. Warrell.)

Figure 49-26. Small-scaled burrowing asp (*Atractaspis microlepidota,* family Atractaspididae) (Watamu, Kenya), showing detail of colubrid-like head scales. (Copyright D.A. Warrell.)

enlargement of local lymph nodes, local numbness, and paraesthesias. The most common systemic symptom is fever. Most of the fatal cases died within 45 minutes of the bite after vomiting, producing profuse saliva, and lapsing into coma.[162] Severe envenoming by *A. engaddensis* may produce violent autonomic symptoms (nausea, vomiting, abdominal pain, diarrhea, sweating, and profuse salivation) within minutes of the bite. One patient developed severe dyspnea with acute respiratory failure; one had weakness, impaired consciousness, and transient hypertension; and in three, there were electrocardiographic changes (ST-T changes and prolonged PR interval).[48] Mild abnormalities of blood coagulation and liver function have also been described. *Atractaspis* venoms have high lethal toxicity. The venom of *A. engaddensis* contains four 21-amino-acid peptides, sarafotoxins, that have 60% sequence homology with endogenous endothelins. They can cause coronary vasoconstriction and atrioventricular block.[99] The venom also contains hemorrhagic and necrotic factors, but no true neurotoxins. Bites by the Natal black snake *(M. microlepidotus)* are said to have resulted in collapse and loss of consciousness for up to 30 minutes in two cases.

Elapidae

Venoms of Elapidae (cobras, kraits, mambas, African shield-nosed snakes, garter snakes, and coral snakes (Fig. 49-27, *Aspidelaps*), Asian coral snakes, sea kraits, and true sea snakes)

Figure 49-27. African coral snake *(Aspidelaps lubricus lubricus)* (South Africa), a mildly neurotoxic elapid. (Copyright D.A. Warrell.)

are well known for their neurotoxic effects. In the case of kraits,[112] mambas, most of the Australasian elapids (see later text), some of the cobras (e.g., Philippine cobra, *N. philippinensis*[168]; Cape cobra, *Naja nivea*) and sea snakes,[82,141] local effects are minimal.

Cytotoxic Elapids

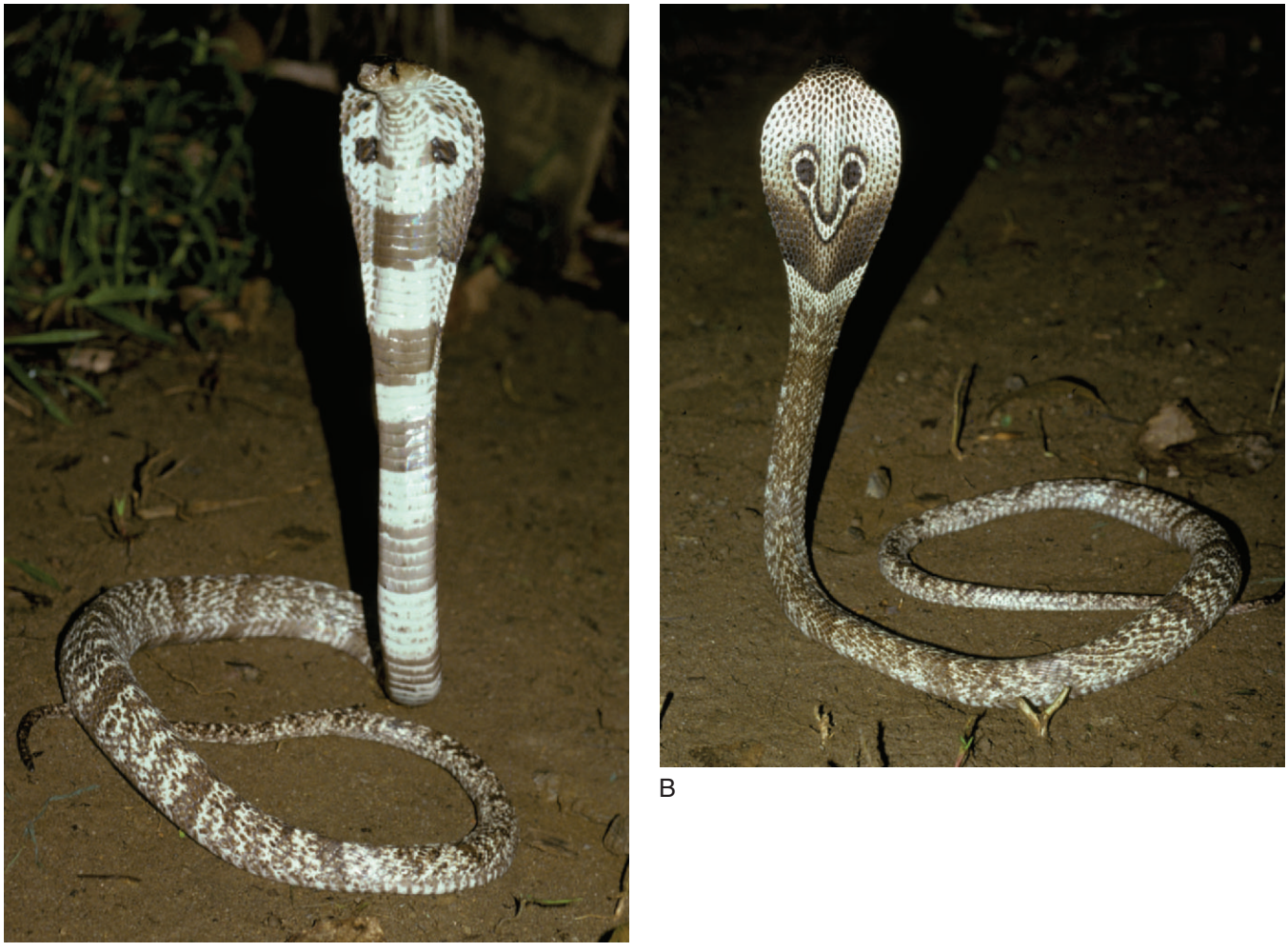
Patients bitten by African spitting cobras (e.g., *N. nigricollis*) and Asian cobras (e.g., *N. naja, N. kaouthia*) (Fig. 49-28) commonly develop tender local swelling, which may be extensive, and regional lymphadenopathy.[118,133,155] A characteristic lesion may appear within 24 to 48 hours. Blistering often surrounds a demarcated pale or blackened anesthetic area of skin (Fig. 49-29, *N. kaouthia* bite). The lesion smells putrid and eventually breaks down, with loss of skin and subcutaneous tissue that may be extensive (Fig. 49-30, *N. nigricollis* bite). Skip lesions, separated by areas of apparently normal skin, may extend proximally up the limb. Prolonged morbidity may result and some patients may lose a digit or the affected limb if there is secondary infection. Envenoming by the black desert cobra *(Walterinnesia aegyptia)* may result in marked local swelling as well as neurotoxicity.[140,146]

Envenoming by African Spitting Cobras

Bites by spitting cobras (e.g., *N. nigricollis, N. katiensis, N. pallida, N. mossambica, N. nubiae, N. nigricincta*) cause a distinctive clinical syndrome unlike that caused by any other elapid snake.[118,135,155] Most bites occur at night inside homes while the victims are asleep. There is immediate pain, followed by vomiting within 6 hours and extensive local swelling, local blistering (60%), and local tissue necrosis (70%). Tissue necrosis usually involves the skin and subcutaneous connective tissues only and there may be the appearance of skip lesions, in which areas of necrosis are separated by strips of apparently normal skin. There is leukocytosis and evidence of complement activation, principally by the alternative pathway.[135,155]

Envenoming by Asian Cobras (Genus *Naja*)

Evolution of local pain, swelling, blistering, and necrosis, as described for African spitting cobras, has been described in patients bitten by Chinese cobras *(N. atra),* monocellate cobras *(N. kaouthia),* Indian spectacled cobras *(N. naja),*[112] and south-



A B

Figure 49-28. Indian cobra *(Naja naja)* (Gampola, Sri Lanka) from the front **(A)** and from the back **(B),** showing "spectacle" marking on the hood. (Copyright D.A. Warrell.)

Figure 49-29. Characteristic local necrotic lesion produced by bites of African spitting cobras and Asian cobras. In this case, the patient was bitten by a monocellate Thai cobra (*Naja kaouthia:* family Elapidae) near Chantaburi, eastern Thailand. Two days after the bite, there was a darkened anesthetic area at the site of the bite (fang marks indicated by red arrows) surrounded by blisters. There was an odor of putrefaction. (Copyright D.A. Warrell.)

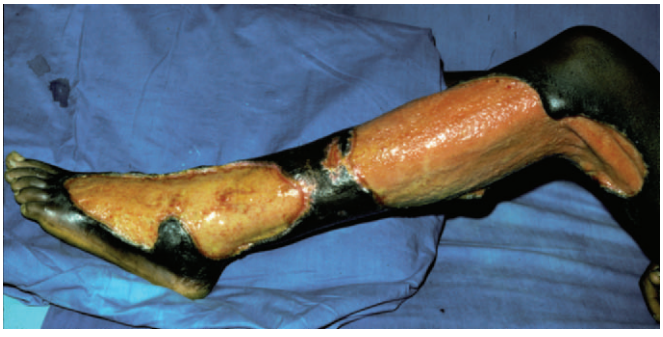


Figure 49-30. Extensive necrosis of skin and subcutaneous tissues in a Mende woman in Sierra Leone bitten 2 weeks earlier by a black-necked or spitting cobra (*Naja nigricollis:* family Elapidae). Note the "skip lesions," areas of necrosis separated by intact skin. (Copyright D.A. Warrell.)

east Asian spitting cobras (*N. siamensis*[181] and *N. sumatrana*). However, the Philippine cobra, which is responsible for about half of all snakebites in Luzon Island, rarely causes local swelling or necrosis.[168] Classic neurotoxicity has been observed in patients envenomed by Asian cobras, with the possible exception of *N. siamensis*. Neurotoxic deaths are claimed to have occurred as soon as 15 minutes after the bite in the case of *N. naja* and from 1.25 to 60 (mean 12.6) hours in the case of *N. kaouthia*.[57,133]

Neurotoxic Elapids

Severe envenoming by the king cobra *(O. hannah)* results in swelling of the whole limb and formation of bullae at the site of the bite, but local necrosis is minimal or absent.[119] Rapidly developing neurotoxicity is the dominant clinical feature of envenoming by this species. Neurotoxic effects are also seen in patients envenomed by Asian cobras (it is the main feature in victims of *N. philippinensis*) and other elapids, but have not been documented in victims of African spitting cobras. The earliest symptom of systemic envenoming is repeated vomiting, but the use of emetic herbal medicines may confuse the interpretation of this symptom. Other early preparalytic symptoms include contraction of the frontalis muscle (before there is demonstrable ptosis), blurred vision and loss of visual accommodation attributable to mydriasis, paresthesia (especially around the mouth), hyperacusis, loss of the senses of smell and taste, headache, dizziness, vertigo, and signs of autonomic nervous stimulation such as hypersalivation, congested conjunctivae, and piloerection ("gooseflesh"). Paralysis is first detectable as ptosis and external ophthalmoplegia, in that ocular muscles are most sensitive to neuromuscular blockade (Fig. 49-31, *B. caeruleus* bite). These signs may appear as early as 15 minutes after the bite (cobras or mambas), but may be delayed for 10 hours or more following krait bites. Later, the facial muscles, palate, jaws, tongue, vocal cords, neck muscles, and muscles of deglutition may become paralyzed (Fig. 49-32, *Oxyuranus s. canni* bite). The pupils are usually dilated. Many patients are unable to open their mouths, but this can be overcome by force. In a minority, the jaw is said to hang open. Respiratory arrest may be precipitated by obstruction of the upper airway by the paralyzed tongue or inhaled vomitus. Intercostal muscles are affected before the limbs, diaphragm, and superficial muscles, and the pattern of breathing becomes abdominal or paradoxical (protrusion of the abdomen during inspiration). Even in patients with generalized flaccid paralysis, slight movements of the digits may be possible, allowing the patients to signal. Loss of consciousness and generalized convulsions are usually explained by hypoxemia in patients who have respiratory paralysis. However, drowsiness, before the development of significant paralysis, has often been described but remains unexplained. Drooping eyelids from tiredness may be misconstrued as ptosis, unless the extent of lid retraction with upward gaze is formally assessed. Patients with systemic envenoming suffer from headache, malaise, and generalized myalgia. Intractable hypotension can occur in patients envenomed by Asian cobras, despite adequate respiratory support. Neurotoxic effects are completely reversible, either acutely in response to antivenom (e.g., in Asian cobra and Australasian death adder bites) or to anticholinesterases,[52,124,159,166,170] or they may slowly wear off spontaneously.[10,11,53,112] In the absence of specific antivenom, patients supported by mechanical ventilators usually recover sufficient diaphragmatic movement to breathe adequately in 1 to 4 days; ocular muscles recover in 2 to 4 days, and there is usually full recovery of motor function in 3 to 7 days.[10–12,53] However, especially in the absence of antivenom treatment, paralysis may persist. Patients have recovered after 30 days of manual ventilation (in a case of envenoming by the Chinese krait, *B. multicinctus*) and after 10 weeks of mechanical ventilation (Australian rough-scaled snake, *Tropidechis carinatus*).

A

B

Figure 49-31. A neurotoxic envenoming by a common krait (*Bungarus caeruleus:* family Elapidae) near Anuradhapura, Sri Lanka, that required mechanical ventilation. **A,** Ptosis, external ophthalmoplegia, and inability to open the mouth 40 hours after the bite. **B,** Recovery 55 hours after the bite. (Copyright D.A. Warrell.)

Figure 49-32. Ptosis and inability to open the mouth or protrude the tongue in a patient bitten by a Papuan taipan *(Oxyuranus scutellatus canni)* in Central Province, Papua New Guinea. (Copyright D.A. Warrell.)

Envenoming by Mambas (Genus *Dendroaspis*)[142,149]

These are the most dreaded snakes of Africa. All four species *(D. polylepis, D. angusticeps, D. jamesoni, and D. viridis)* are capable of causing rapidly progressive and fatal respiratory paralysis. The speed of evolution of envenoming is well illustrated by a patient seen in Harare, Zimbabwe. After a bite by a 3-m (9 ft 10 in) long black mamba *(D. polylepis)*, the victim noticed tingling of the tongue and lips within 1 minute, followed by generalized tingling, abdominal pain, and lightheadedness. Within 20 minutes, he was sweating profusely, had dilated pupils, and was too weak to stand up. He became nauseated and vomited 30 minutes after the bite, by which time he was unable to pass urine and had detectable ptosis. He became breathless and found it difficult to clear his throat of thick secretions. Forty minutes after the bite, he felt cold all over and noticed gooseflesh, his conjunctivae were congested, and he was unable to open his mouth or protrude his tongue. There was then a rapid deterioration in his breathing and level of consciousness. Generalized fasciculations were noticed. He was treated with antivenom after 75 minutes and, $4^1/_2$ hours after the bite, was intubated and mechanically ventilated for 40 hours, after which he made a complete recovery.[142]

Envenoming by African Neurotoxic Cobras (*Naja haje, N. annulifera, N. anchietae,*[8] *N. melanoleuca,* and *N. nivea*)

Bites may cause some local swelling, but no necrosis develops. Classic neurotoxic symptoms develop as early as 30 minutes after the bite and can evolve to the point of fatal respiratory paralysis 2 to 16 hours after the bite.[142,149,152]

Envenoming by the King Cobra (*Ophiophagus hannah*)[119,143]

The world's largest venomous snake can cause extensive local swelling, but neurotoxicity is the most prominent feature, reputedly capable of leading to death "soon" or in a "few minutes" after the bite. One patient, bitten by a 2-m (6 ft 7 in) long specimen in Burma, began to feel dizzy 15 minutes after the bite, had difficulties with speech and breathing after 30 minutes, showed bilateral ptosis after 40 minutes, and at 90 minutes suffered a respiratory arrest requiring 38 hours of mechanical ventilation. He made a full recovery.[119]

Envenoming by Asian Kraits (Genus *Bungarus*)[18,112]

The common Indian krait *(B. caeruleus)* is considered to be the most dangerous species of venomous snake in the Indian subcontinent, together with *N. naja*.[113] It is responsible for 22% to 44% of snakebite deaths in Anuradhapura, Sri Lanka, and for 33% of all venomous snakebites in West Bengal. The epidemiologic pattern is distinctive. Most patients are bitten while asleep on the floor of their huts, the bites occurring particularly at the time of night when sleepers are particularly restless during rapid eye movement sleep. At that time, they are more likely to touch or trap a snake that is prowling around their home in search of its natural prey. Because people are bitten while lying down, the target may be any part of the body, including the head, neck, and shoulders. In one series of 88 cases of proven *B. caeruleus* envenoming in Sri Lanka, all patients were bitten while sleeping on the ground, at night, indoors. Only 23% of bites were on the lower limbs (compared to 82% by other species). Only 9% of patients had any sign of local envenoming, but 64% developed respiratory paralysis and the case-fatality rate was 6%, twice as high as with other species (A. Ariaratnam et al., 2006, unpublished). Krait bites usually produce invisible or scarcely perceptible puncture marks, no local symptoms apart from mild tenderness, itchiness, numbness, or some other paresthesia, and negligible or no local swelling. As a result, snakebite may not be suspected and patients commonly present to the hospital after waking in the morning with descending paralysis ("early morning paralysis").[85] A virtually identical syndrome has been described following Malayan krait *(B. candidus)* bites in southeast Asia, Sri Lankan krait *(B. ceylonicus)* bites in Sri Lanka, and bites by the banded krait *(B. fasciatus)* and the Chinese krait *(B. multicinctus)*. Fasciculations were described in a victim of *Bungarus sindanus walli* bite in Bangladesh.[47a] Rare differential diagnoses

Figure 49-33. Papuan taipan *(Oxyuranus scutellatus canni)* (Port Moresby, Papua New Guinea) specimen 2.5 m (8 ft 2 in) long showing the orange dorsal stripe that distinguishes this subspecies from the smaller Australian eastern taipan *(O. s. scutellatus).* (Copyright D.A. Warrell.)

of this type of neurologic presentation would include acute idiopathic inflammatory polyneuropathy (Guillain-Barré syndrome, especially its variants, Miller-Fisher syndrome and polyneuritis cranialis), Bickerstaff brainstem encephalitis, myasthenia gravis, and botulism. Rarely, local lymph nodes may be painfully enlarged. Abdominal pain is described in the majority of cases. Paralysis may develop within 2 hours or be delayed for 7 to 12 hours. Severe generalized muscle pains were noticed by a few patients in whom moderately elevated plasma myoglobin concentrations raised the possibility of generalized rhabdomyolysis. Fatal rhabdomyolysis and renal failure has been described in a victim of greater black krait (*Bungarus niger*) bite in Bangladesh.[22a] Reported case-fatality rate in the absence of antivenom treatment and assisted ventilation was as high as 77% in India and 100% in Sri Lanka.

Envenoming by Australian and New Guinean Elapids[10–12,14,51–55,104,171]

Venoms of these snakes cause a distinctive triad of symptoms: neurotoxicity similar to that seen with other elapid bites (see Figure 49-32, *O. s. canni* bite), generalized rhabdomyolysis, and hemostatic disturbances. Local signs are usually mild, but extensive local swelling and bruising with necrosis has been reported, especially after bites by the king brown or mulga snake *(Pseudechis australis)*. Painful and tender local lymph nodes are a common feature in patients developing systemic envenoming.

Early symptoms include vomiting, headache, and syncopal attacks similar to those experienced after some viper bites. Electrocardiographic changes were common in patients envenomed by taipans *(Oxyuranus s. canni)* (Fig. 49-33) in Papua New Guinea, but only a few had raised cardiac troponin-T levels suggesting myocardial damage.[53,54] In dogs, common brown snake *(Pseudonaja textilis)* venom caused myocardial depression attributed to disseminated intravascular coagulation, and tiger snake *(Notechis scutatus)* venom caused formation of thrombi within the heart leading to pulmonary and coronary artery thromboembolism.[104]

All Australian elapid venoms are potentially neurotoxic in humans.[123] Some patients lose their sense of smell and taste, in a few cases permanently, which is reminiscent of bites by the African berg adder, *Bitis atropos* (see later text). Many of the venoms contain myolytic PLA_2s[100,104] and generalized rhabdomyolysis has been confirmed after bites by *Notechis, Pseudechis, Oxyuranus, Austrelaps, Tropidechis,*

Figure 49-34. Ptosis, external ophthalmoplegia, facial paralysis, and trismus in a fisherman bitten 24 hours previously by a beaked sea snake (*Enhydrina schistosa:* family Elapidae; subfamily Hydrophiinae) in northwestern Malaysia. (Courtesy of the late H. Alistair Reid.)

Rhinoplocephalus/Cryptophis nigrescens, and *Micropechis ikaheka.*[104,156,171]

Persistent bleeding from wounds and spontaneous systemic bleeding from gums and the gastrointestinal tract are found in association with incoagulable blood following bites by many Australasian species. Venoms of 15 out of 19 species exhibited procoagulant activity in vitro.[104] Some venoms (e.g., *Pseudechis, Acanthophis, Micropechis ikaheka,* and *Pseudonaja*) are anticoagulant and might cause incoagulable blood without evidence of activation of coagulation and fibrinolysis.[44,52,101,156] Hemostatic abnormalities are particularly frequent and serious in patients bitten by tiger snakes (*Notechis* spp.), taipans (*Oxyuranus* spp.), and brown snakes (*Pseudonaja* spp.); uncommon with bites by black snakes (*Pseudechis* spp.); and rare with bites by death adders (*Acanthophis* spp.).[53]

Envenoming by Sea Snakes (Hydrophiinae) and Sea Kraits (Laticaudinae)[82,84,104,141,171]

The bite is usually painless and may not be noticed by the wader or swimmer. Fangs and other teeth may be left in the wound. There is minimal or no local swelling and involvement of local lymph nodes is unusual. Generalized rhabdomyolysis is the dominant effect of envenoming by these snakes. Early symptoms include headache, a thick feeling of the tongue, thirst, sweating, and vomiting. Generalized aching, stiffness, and tenderness of the muscles becomes noticeable between 30 minutes and $3^1/_2$ hours after the bite. Trismus is common. Passive stretching of the muscles is painful. Later, there is progressive flaccid paralysis starting with ptosis, as in other neurotoxic envenomings (Fig. 49-34, sea snakebite). The patient remains conscious until the respiratory muscles are sufficiently affected to cause respiratory failure. Myoglobinemia and myoglobinuria develop 3 to 8 hours after the bite (Fig. 49-35, sea snakebite). These are suspected when the serum/plasma appears brownish and the urine dark reddish brown ("Coca-Cola-colored"). Bedside "stix" tests will appear positive for hemoglobin/blood

Figure 49-35. Myoglobinuria resulting from generalized rhabdomyolysis in a Malayan fisherman envenomed by a sea snake. (Courtesy of the late H. Alistair Reid.)

Figure 49-36. Intense conjunctivitis, leukorrhea, and corneal erosions in a patient "spat" at 3 hours previously by an African black-necked spitting cobra (*Naja nigricollis:* family Elapidae) near Zaria, Nigeria. (Copyright D.A. Warrell.)

in urine containing myoglobin. Myoglobin and potassium released from damaged skeletal muscles may cause renal failure, while hyperkalemia developing within 6 to 12 hours of the bite may precipitate cardiac arrest.

Snake Venom Ophthalmia[72,76,136,160]

Venom ophthalmia results when venom of the spitting elapids (African and Asian spitting cobras, genus *Naja;* southern African rinkhal, *Hemachatus haemachatus*) enters the eye. There is intense local pain, blepharospasm, palpebral edema, and leukorrhea (Fig. 49-36, *N. nigricollis* spit). Slit-lamp or fluorescein examination reveals corneal erosions in more than half the patients spat at by *N. nigricollis*.[135,160] Secondary infection of the corneal lesions may result in permanent opacities, causing blindness or panophthalmitis with destruction of the eye. Rarely, venom is absorbed into the anterior chamber, causing

Figure 49-37. Demarcated local swelling spreading up the upper arm in a patient bitten 90 minutes earlier by a saw-scaled viper *(Echis ocellatus)* in Kaltungo, Nigeria. (Copyright D.A. Warrell.)

Figure 49-38. Tense, painful swelling extending up the thigh, bruising and continuing bleeding from fang punctures on the lateral surface of the calf in a patient bitten 2 hours earlier by a Malayan pit viper (*Calloselasma rhodostoma:* family Viperidae; subfamily Crotalinae) near Trang, southern Thailand. Although arterial pulsation was detectable by Doppler ultrasound, the pressure in the anterior tibial compartment was elevated at 69 mm Hg, justifying fasciotomy once hemostasis had been restored with antivenom. (Copyright D.A. Warrell.)

hypopyon and anterior uveitis. Seventh (facial) cranial nerve paralysis is a rare complication that results from tracking of venom from the conjunctival sac through lymphatics posteriorly to the superficially situated seventh cranial nerve.

Viperidae (Old World Vipers and Adders, Asian Pit Vipers)

Local envenoming is usually more severe than that caused by other snakebites. Swelling may develop immediately, but is sometimes delayed for several hours. It may spread rapidly (Fig. 49-37, *E. ocellatus* bite), sometimes involving the whole limb and adjacent trunk (Fig. 49-38, *C. rhodostoma* bite). There is associated pain, tenderness and enlargement of regional lymph nodes. Bruising is common, especially along the path of superficial lymphatics and over regional lymph nodes (Figs. 49-39 and 49-40, *C. rhodostoma* bites). Persistent bleeding from the fang marks suggests coagulopathy (see Figure 49-38, *C. rhodostoma* bite). Swollen limbs can accommodate many liters of extravasated blood, leading to hypovolemic shock. Blistering

Figure 49-39. Swelling, bruising, bleeding, and blistering 36 hours after a bite on the lateral side of the ankle (fang punctures are circled) by a Malayan pit viper (*Calloselasma rhodostoma:* family Viperidae; subfamily Crotalinae) near Trang, southern Thailand. (Copyright D.A. Warrell.)

Figure 49-40. Extensive local swelling, bruising, and blistering 3 days after a bite on the shin by a Malayan pit viper *(Calloselasma rhodostoma)* near Chantaburi, eastern Thailand. (Copyright D.A. Warrell.)

Figure 49-41. Swelling and blistering of the hand and arm 36 hours after a bite in the first interdigital cleft by a puff adder (*Bitis arietans:* family Viperidae; subfamily Viperinae) near Zaria, Nigeria. (Copyright D.A. Warrell.)

Figure 49-42. Surgical debridement of necrotic tissue 7 days after a bite by a Malayan pit viper *(Calloselasma rhodostoma)* near Chantaburi, eastern Thailand on the dorsum of the foot. (Copyright D.A. Warrell.)

may appear at the bite site within a few hours after the bite (see Figures 49-39 and 49-40, *C. rhodostoma* bites; Fig. 49-41, *B. arietans* bite). These contain clear or bloodstained fluid. Necrosis of skin, subcutaneous tissue, and muscle (Fig. 49-42, *C. rhodostoma* bite) develops in up to 10% of hospitalized cases, especially following bites by Asian pit vipers (e.g., *Calloselasma rhodostoma, Deinagkistrodon acutus,* and *T. [Protobothrops] flavoviridis*), African vipers (genus *Bitis*), saw-scaled vipers (genus *Echis*), and the Palestine viper *(Daboia palaestinae)*. Bites on the digits and in areas draining into the tight fascial compartments, such as the anterior tibial compartment, are particularly likely to result in necrosis. High intracompartmental pressure may cause ischemia that contributes, together with direct effects of the venom, to muscle necrosis.[28] Severe pain associated with tense swelling, segmental anesthesia, and pain on stretching the intracompartmental muscles (e.g., dorsiflexion of the foot in the case of the anterior tibial compartment) should raise the possibility of raised intracompartmental pressure. Sudden severe pain, absence of arterial pulses, and a demarcated cold segment of limb suggest thrombosis of a major artery. Deep venous thrombosis has been described surprisingly rarely. The absence of detectable local swelling 2 hours after a viper bite usually means that no venom has been injected, but fatal systemic envenoming by the Burmese Russell's viper *(D. siamensis)* may occur in the absence of local signs.

Systemic Envenoming

Hemostatic Abnormalities. These are characteristic of envenoming by Viperidae, but are usually absent in patients bitten by the smaller European vipers (e.g., *V. berus, V. aspis, V. ammodytes*).[81] Persistent bleeding (>10 minutes) from the fang puncture wounds and from new injuries such as venipuncture sites and old partially healed wounds is the first clinical evidence of consumption coagulopathy. Spontaneous systemic hemorrhage is most often detected in the gingival sulci (Fig. 49-43, *C. rhodostoma* bite). Bloodstaining of saliva and sputum usually reflects bleeding gums or epistaxis. True hemoptysis is rare. Hematuria may be detected a few hours after the bite. Other types of spontaneous bleeding are ecchymoses; intracranial and subconjunctival hemorrhages; bleeding into the floor of the mouth, tympanic membrane, and gastrointestinal and genitourinary tracts; petechiae; and larger discoid and follicular hemorrhages (Fig. 49-44, *C. rhodostoma* bite). Bleeding into the iliacus muscle may result in weakness of hip flexion (Fig. 49-45, *E. ocellatus* bite). Hemorrhagic infarction of the ante-

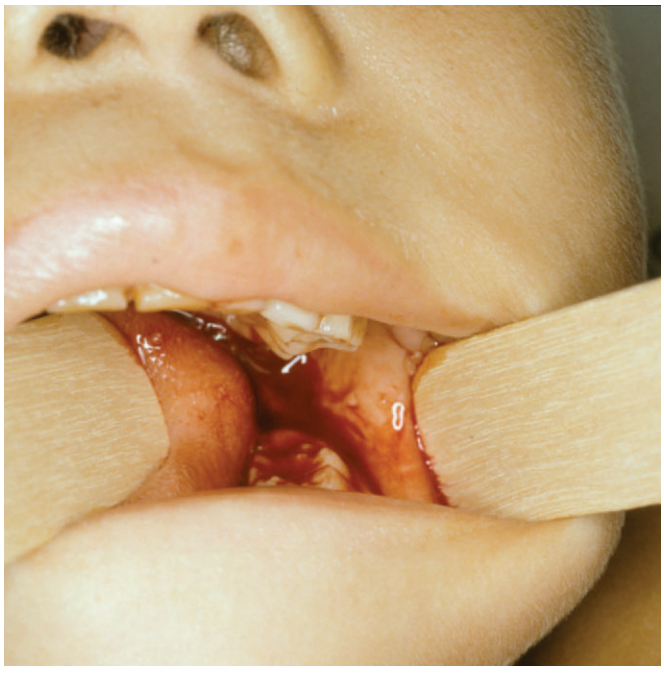


Figure 49-43. Bleeding from gingival sulci 3 hours after a bite by a Malayan pit viper *(Calloselasma rhodostoma)* near Chantaburi, eastern Thailand. (Copyright D.A. Warrell.)

rior pituitary (resembling postpartum Sheehan's syndrome) may complicate envenoming by Russell's vipers in Burma and India.[131] Menorrhagia and antepartum and postpartum hemorrhage have been described after envenoming by vipers.[5] Severe headache and meningism suggest subarachnoid hemorrhage. Evidence of a developing central nervous system lesion (e.g., hemiplegia), irritability, loss of consciousness, and convulsions suggest intracranial hemorrhage (Fig. 49-46, *C. cerastes* bite) or cerebral thrombosis. Abdominal distention, tenderness, and peritonitis with signs of hemorrhagic shock but no external blood loss (hematemesis or melena) suggest retroperitoneal or intraperitoneal hemorrhage. Incoagulable blood resulting from defibrination or disseminated intravascular coagulation is a common and important finding in patients systemically envenomed by members of the following genera: *Atheris, Daboia, Vipera, Echis, Gloydius, Calloselasma, Deinagkistrodon,* and *Trimeresurus (sensu lato)*.

Intravascular hemolysis, causing hemoglobinemia (pink plasma) and black or grayish urine (hemoglobinuria or methemoglobinuria), has been convincingly described in patients bitten by Sri Lankan Russell's viper *(Daboia russelii)*,[73] the Saharan horned viper *(Cerastes cerastes)*[90] (Figs. 49-47 and 49-48), and Australian western brown snakes (*Pseudonaja* spp.) (Mark Little, personal communication, 2006). Features of microangiopathic hemolysis that have suggested hemolytic-uremic syndrome, although associated with marked coagulopathy, may progress to severe anemia and renal failure.[90]

Circulatory Shock (Hypotensive) Syndromes

A decrease in blood pressure is a common and serious event in patients bitten by vipers, especially in the case of some of the Old World Viperinae (e.g., *D. russelii, D. siamensis, D.*

Figure 49-44. Discoid hemorrhages visible 18 hours after a bite by a Malayan pit viper *(Calloselasma rhodostoma)* in Song-be, South Vietnam. (Copyright D.A. Warrell.)

Figure 49-45. Weakness of flexion of the left hip in a patient who bled into his left iliacus muscle after envenoming by a saw-scaled viper *(Echis ocellatus)* near Kaltungo, Nigeria. (Copyright D.A. Warrell.)

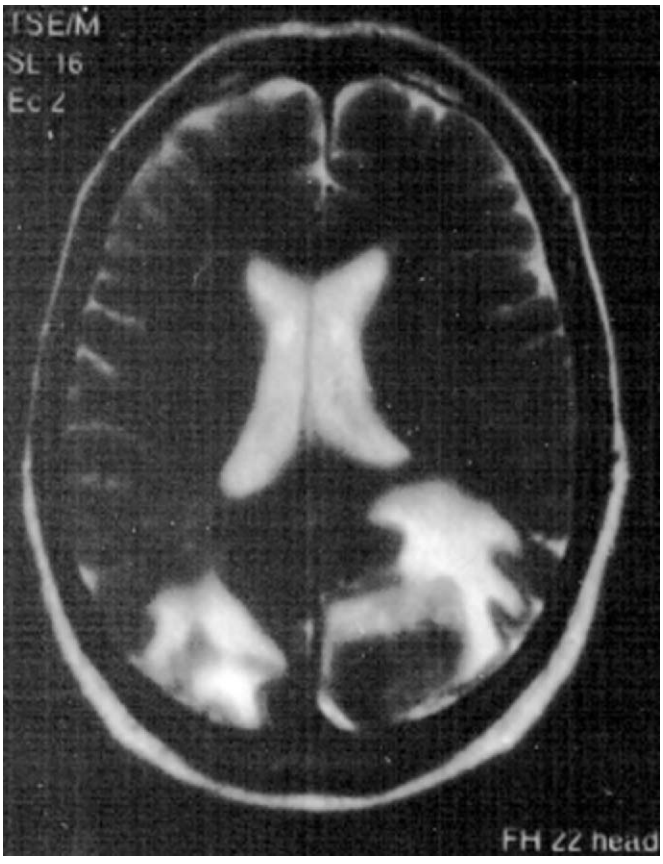
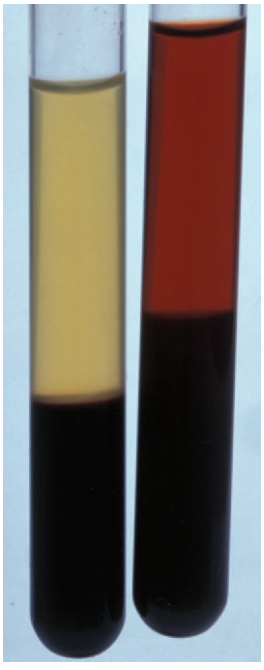
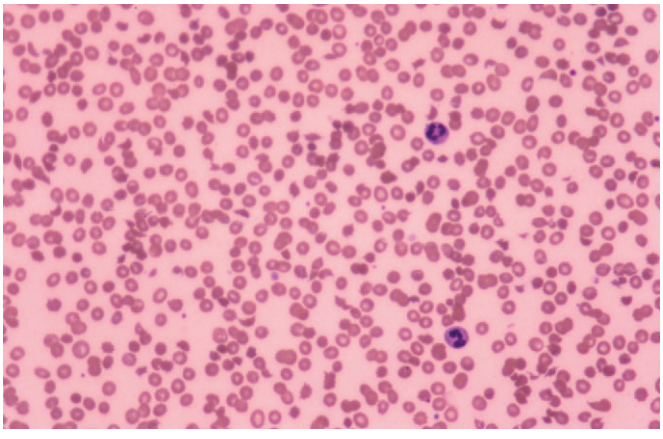


Figure 49-46. Cerebral computed tomography scan of a 63-year-old man who developed weakness of a hand and problems with vision 4 days after being bitten by a Saharan horned viper (*Cerastes cerastes:* family Viperidae; subfamily Viperinae). (Copyright D.A. Warrell.)[90]

Figure 49-47. Saharan horned viper *(Cerastes cerastes)* (Morocco). (Copyright D.A. Warrell.)



A



B

Figure 49-48. A 43-year-old man who developed evidence of intravascular hemolysis after being bitten by a Saharan horned viper *(Cerastes cerastes).* **A,** Hemoglobinemia; patient's plasma was allowed to separate without centrifugation (*right*) compared to normal control (*left*). **B,** Blood film showing evidence of microangiopathic hemolysis (numerous schistocytes/fragmented erythrocytes/helmet cells). (Copyright D.A. Warrell.)[90]

palaestinae, *V. berus*, *Bitis arietans*, *B. gabonica*, and *B. rhinoceros*).[60,70,161] Sinus tachycardia suggests hypovolemia resulting from external hemorrhage, blood loss into the tissues, or local or generalized increase in capillary permeability. Patients envenomed by Burmese Russell's viper *(D. siamensis)* may develop conjunctival edema (Fig. 49-49, *D. siamensis* bite), serous effusions, pulmonary edema, hemoconcentration, and a decrease in serum albumin concentration, all evidence of increased vascular permeability.[70,116] Pulse rate may be slow or irregular if the venom is affecting the heart directly or reflexly (e.g., *V. berus*, *Bitis arietans*, *Calloselasma rhodostoma*). Vasovagal syncope may be precipitated by fear and pain. Early, repeated, and usually transient syncopal attacks with features of anaphylaxis develop in patients bitten by some Viperidae (e.g., *D. palaestinae*, *V. berus*, *V. aspis*, *D. siamensis*, and *D.*

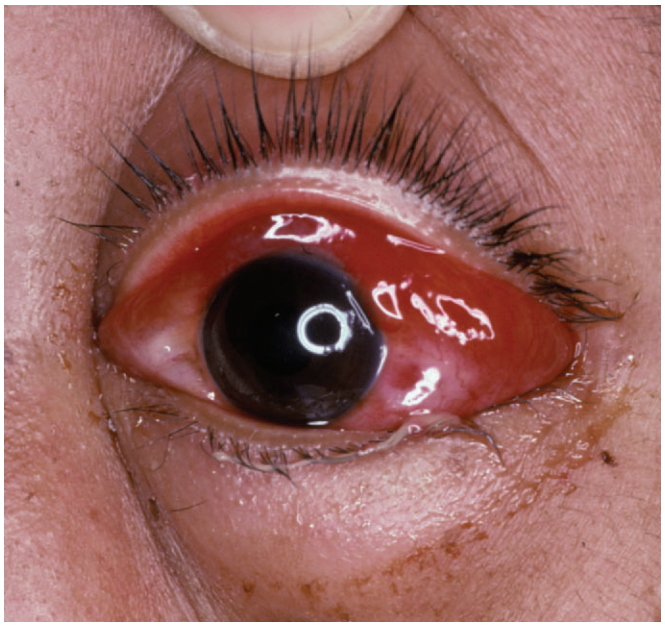


Figure 49-49. Intense bilateral conjunctival edema (chemosis), evidence of generalized increase in capillary permeability in a man bitten 24 hours previously by a Russell's viper (*Daboia siamensis:* family Viperidae; subfamily Viperinae) near Tharrawaddy, Burma. (Copyright D.A. Warrell.)

russelii). Vomiting, sweating, colic, diarrhea (with incontinence), shock, bronchospasm, urticaria, and angioedema of the face, lips, gums, tongue, and throat may appear as early as 5 minutes or as late as many hours after the bite. Hypotension is an important feature of anaphylactic reactions to antivenom (see later text). Renal failure can complicate severe envenoming by any species of snake, but it is common and is the most frequent cause of death in victims of Russell's viper. Persons bitten by Russell's viper may become oliguric within a few hours of the bite. Loin pain and tenderness may be experienced within the first 24 hours and, in 3 or 4 days, the victim may become irritable, comatose, or convulsing, with hypertension and evidence of metabolic acidosis.

Neurotoxicity attributable to venom phospholipases A_2 is a feature of envenoming by a few species of Old World Viperidae (e.g., *Gloydius/Agkistrodon blomhoffii, Vipera aspis, Bitis atropos,* and other small South African *Bitis* species and Sri Lankan and south Indian Russell's viper). The clinical features are the same as with elapid envenoming. Progression to respiratory or generalized paralysis is unusual but has been described. Associated generalized myalgia suggests the possibility of rhabdomyolysis. Pupillary dilatation, causing visual disturbance from loss of accommodation, is a feature of severe envenoming by small *Bitis* species (e.g., *B. peringueyi*) and may be a permanent neurologic sequela that can be corrected transiently by instillation of pilocarpine.

Envenoming by Viperinae

Envenoming by European Vipers[64,81,147,150a]

The common viper, or adder *(V. berus),* the only venomous snake found in Britain, occurs in England, Wales, Scotland, and

Figure 49-50. Nose-horned or sand viper *(Vipera ammodytes)* (Balkans). (Copyright D.A. Warrell.)

Figure 49-51. Milos viper *(Macrovipera schweizeri)* (Milos Island, Cyclides near Greece). (Copyright D.A. Warrell.)

northern Europe, extending into the Arctic Circle and through Asia as far east as Sakhalin Island and south to northern Korea. Four other vipers are widely distributed in mainland Europe: the nose-horned or sand viper *(V. ammodytes)* (Fig. 49-50) in the Balkans, Italy, Austria, and Romania; the asp viper *(V. aspis)* in France (south of Paris), Spain, Germany, Switzerland, and Italy; Lataste's viper *(V. latastei)* in Spain and Portugal, and Orsini's viper *(V. ursinii)* in southeastern France, central Italy, and Eastern Europe. The Ottoman viper *(Montivipera xanthina)* occurs around Istanbul and in some eastern Aegean islands, northern Greece, and Asia Minor. The Milos viper *(Macrovipera schweizeri)* (Fig. 49-51) inhabits some of the Cyclides Islands, southeast of Greece, while the Levantine viper *(Macrovipera lebetina)* occurs on Cyprus.

Clinical Features of European Adder (*Vipera berus*) Bite[150a]

Pain usually develops quickly at the site of the bite and local swelling is evident within a few minutes, but is sometimes delayed for 30 minutes or longer. Local blisters containing

blood are uncommon. Swelling and bruising may advance to involve the whole limb within 24 hours, extend to the trunk, and, in children, become generalized. A few cases of compartmental syndromes and necrosis have been described. Pain, tenderness, and enlargement of local lymph nodes is sometimes noticeable within hours. Marked lymphangitis and bruising of the affected limb appear within a day or two. Dramatic early systemic symptoms may appear within 5 minutes of the bite or be delayed for many hours. These include retching; vomiting; abdominal colic; diarrhea; incontinence of urine and feces; sweating; vasoconstriction; tachycardia; shock; angioedema of the face, lips, gums, tongue, throat, and epiglottis; urticaria; and bronchospasm. These symptoms may persist for as long as 48 hours. Hypotension is the most important sign. It usually develops within 2 hours. It may be transient, resolving spontaneously within 2 hours, or persistent and recurrent, or progressive and fatal. Electrocardiogram (ECG) changes include flattening or inversion of T waves, ST segment elevation, second-degree heart block, bradyarrhythmias or tachyarrhythmias, atrial fibrillation, and myocardial infarction. Defibrination (incoagulable blood) or milder degrees of coagulopathy and spontaneous bleeding into the gastrointestinal tract, lungs, or urinary tract are uncommon. Other clinical features include fever, drowsiness, and rarely, coma and seizures secondary to hypotension or cerebral edema, respiratory distress/pulmonary edema (in children), acute renal failure, cardiac arrest, intrauterine death, acute gastric dilatation, and paralytic ileus. Laboratory findings include neutrophil leukocytosis (more than 20,000/μL in severe cases), thrombocytopenia, initial hemoconcentration and later anemia resulting from extravasation into the bitten limb, and, rarely, hemolysis, elevation of serum creatine kinase, and metabolic acidosis. Deaths usually occur from 6 to 60 (average 34) hours after the bite. Most adder bites cause only trivial symptoms, but patients must be assessed individually. Children may be severely envenomed; in a recent French series, there were three deaths in a group of seven children aged between $2^1/_2$ and 10 years. The dangers of adder bite should not be underestimated.

Envenoming by other European and Mediterranean vipera produces similar features, most severe in the case of *M. lebetina* (Cyprus), *V. ammodytes,* and *V. aspis* bites, and least severe in *V. ursinii* and *V. latastei* bites. It is surprising that bites by *M. schweizeri* (Cyclides Islands, Greece) do not appear to result in severe envenoming. In one area of southeastern France, *V. aspis zinnikeri* and *V. a. aspis* can cause mild neurotoxic envenoming.

Envenoming by Saw-Scaled or Carpet Vipers (Genus *Echis*)[142,143,151]

This genus of vipers is of great medical importance. It is widely distributed in the northern third of Africa from Senegal in the west to the Tana River in Kenya in the south; through the Middle East and western Asia, as far north as the Aral Sea; and throughout the Indian subcontinent, including Sri Lanka, as far as the border of West Bengal. Throughout this range, it is usually the most important cause of human snakebite morbidity and mortality. In Africa, bites are most common in West Africa, where *E. ocellatus* (see Figure 49-10) is the most important species.[21,135,154,163,165] In Nigeria, only 4% of patients admitted to the hospital lacked signs of envenoming, the lowest rate of "dry bites" reported in any large case series. The remainder had both local and systemic envenoming. Twelve percent developed local blistering and 9% developed necrosis. Coagulopathy (attributable to venom prothrombin-activators and factor X-activators) was universal; 55% of victims showed spontaneous systemic bleeding, usually from the gingival sulci, but thrombocytopenia ($<103 \times 10^9$/L) was detected in only 7%. Soluble fibrin complexes and fibrin(ogen) breakdown products were detected, but heparin does not inhibit *Echis*-thrombin in vitro or in patients.[21,42,163] The case-fatality rate of 3.6% was attributable to hemorrhagic shock (three cases) and intracerebral hemorrhage (two cases). Envenoming by eastern and North African *E. pyramidum* is generally less severe, but fatalities are reported in Turkana and Wajir in northern Kenya and in Somalia. One patient bitten by a Tunisian specimen developed transient ptosis.[30] *Echis* species cause most fatal snakebites in the Middle East.[140,142,146] *E. carinatus* is prodigiously common in some areas of India and Pakistan (e.g., in Sind and Jammu), where it is the major cause of snakebite morbidity and mortality.[5,151] In Jammu in northern India, envenoming by *Echis carinatus sochureki* caused symptoms similar to those observed in Nigeria.[5]

Envenoming by Western and Eastern Russell's Vipers (*Daboia russelii* and *D. siamensis*)

These medically important species occur from Pakistan in the west through India and Sri Lanka, north into Nepal[6] and Bhutan *(D. russelii),* and as far east as West Bengal; and in Southeast Asia, southern China, Taiwan, and in parts of Indonesia[4] *(D. siamensis).* Throughout this range, there are intriguing geographic variations in the clinical manifestations of envenoming that reflect differences in venom composition.[22,74,127,136,137,145] In Sri Lanka, where *D. russelii* is a major cause of venomous snakebites, 28% of patients bitten by this species showed no clinical evidence of envenoming.[1,2,73] Apart from typical features of viperine envenoming (local envenoming, coagulopathy, bleeding, and sometimes shock), there were distinctive features of neuromyotoxicity attributable to venom PLA_2: ptosis (77%), external ophthalmoplegia (82%), inability to open the mouth (23%) or to swallow and protrude the tongue, generalized muscle tenderness (32%), and myoglobinuria (27%). Most patients showed evidence of intravascular hemolysis. Acute renal failure was a common feature of severe envenoming.

In most parts of India, *D. russelii* is an important cause of snakebite, but in Jammu in the northeast, only 4 of 310 identified viperine bites were caused by this species.[5] In Kerala, South India, neurotoxic signs, such as ptosis and ophthalmoplegia, associated with hemostatic disorders, are familiar signs of envenoming by this species. Features of panhypopituitarism, presenting between 1 month and 1 year after the bite, were observed in 7 of 1000 cases of snakebite and there was one case of diabetes insipidus. Especially in the south, Russell's viper bite is the most common cause of acute renal failure in both adults and children.

In Burma (Myanmar), Russell's viper *(D. siamensis)* is the most important cause of snakebite morbidity and mortality. However, about one third of all patients hospitalized after proven Russell's viper bites develop no clinical evidence of envenoming at any stage. In Tharrawaddy, north of Rangoon, two distinct populations of Russell's vipers were found to be responsible for bites during the November to January rice

harvest. Smaller snakes (125 to 375 mm ($4^{9}/_{10}$–$14^{3}/_{4}$ in) in total length) had probably been born that year, while the larger snakes (500 to 1125 mm ($19^{1}/_{3}$–$44^{1}/_{3}$ in) in total length) had been born in previous years.[129] Bites by larger snakes were associated with more intense local swelling and a higher risk of systemic envenoming.[129] Severe systemic envenoming can result despite there being little or no local evidence of envenoming. Spontaneous bleeding (gums, epistaxis, hemoptysis, hematemesis, hematuria) develops within a few hours. A sign, apparently unique to Russell's viper bite in Burma, is orbital and conjunctival edema with conjunctival hemorrhages (see Figure 49-49) that develops in severe cases and is evidence of generalized increase in capillary permeability. Other manifestations of this permeability syndrome are facial edema, pleural effusions, ascites, pulmonary edema, and transient proteinuria with hypoalbuminemia.[116] Other clinical features include hypotension and shock, epigastric and chest pain, and development of acute renal failure within 3 to 4 days of the bite. This is associated with bilateral loin tenderness and hypertension. In some cases, spontaneous diuresis began within 7 to 10 days, followed by complete recovery without residual renal abnormalities. Some patients developed a late hemorrhagic syndrome with recurrent shock, and at autopsy were found to have hemorrhagic infarction of the anterior pituitary and in some cases adrenal glands, intracranial hemorrhage, and swollen hemorrhagic kidneys.[130] Renal angle tenderness predicted the development of oliguria (sensitivity 0.7, specificity 0.9).[116,120] Russell's viper venom contains a number of different components that affect hemostasis, including procoagulants (activating factors X, IX, and V, and protein C), fibrinolytic agents, platelet aggregating and inhibiting factors, an anticoagulant, and a hemorrhagin.[42] In human patients, the most striking laboratory features are depletion of fibrinogen, factors V, X, and XIIIa, antithrombin III, plasminogen, antiplasmin, protein C, and platelets. There are high levels of fibrin(ogen)-related antigen, mostly cross-linked in the form of D-dimer.[107,108] At autopsy, there is widespread evidence of bleeding but also fibrin deposition, occluding small blood vessels in the pituitary, lungs, and kidneys.[106] Some patients who survive the acute pituitary adrenal crisis described presented later with more insidious symptoms of panhypopituitarism, including loss of libido, impotence, loss of secondary sexual hair, hypotension, amenorrhea, and features of hypothyroidism.[130]

Russell's viper bites are common in the central rice growing area of Thailand, but although the snake is apparently the same species *(D. siamensis)* as in adjacent Burma, clinical features are generally less severe, although there have been deaths attributed to shock, cerebral hemorrhage, and renal failure.[57]

Envenoming by African Puff Adders (*Bitis arietans*)[142,161]

This species (or species complex) is thought to be responsible for the majority of venomous bites throughout Africa. Local swelling is often extensive and associated with hypovolemic shock. Compartmental syndromes, especially involving the anterior tibial compartment after bites on the feet and ankles, may develop. Direct myocardial effects and arrhythmias, commonly sinus bradycardia, may contribute to hypotension. In West Africa, hemostatic abnormalities are confined to spontaneous bleeding and bruising, whereas in eastern Africa and South Africa, coagulopathy has been reported.

Envenoming by Gaboon Vipers (*Bitis gabonica* and *B. rhinoceros*)[60,142]

These giant rain forest species are the most common cause of snakebite in some areas of Africa. It is surprising, in view of the snakes' wide distribution, prodigious size, enormous fangs, and massive venom yield, that so few cases of envenoming have been reported. Local effects of envenoming may be less severe than those produced by puff adder bites, but swelling, bruising, blistering, and necrosis are common. Systemic symptoms may be early and dramatic. Cardiovascular abnormalities, including hypotension and shock, arrhythmias, and ECG changes, are reported. Spontaneous systemic bleeding is a common feature, while hemostatic abnormalities include thrombocytopenia and evidence of thrombin-like and fibrinolytic activities.

Envenoming by the Berg Adder (*B. atropos*)[142]

This mountain species, which has been responsible for envenoming rock climbers in Zimbabwe and South Africa, can cause unusual symptoms. After initial pain and local swelling, nausea, vomiting, paresthesia of the tongue and lips, blurring of vision, and loss of taste and smell may develop. Ptosis, external/internal ophthalmoplegia, dilated pupils, loss of visual accommodation, and anosmia were reported in 93% of cases in one series. There is respiratory paralysis in 72%, hyponatremia (attributed to a natriuretic hormone-like toxin), and dysphagia in 64%, and convulsions in 29% (GJ Mueller and JM van Zyl, personal communication). Only one fatal case has been reported.

Envenoming by Desert Vipers (Genus *Cerastes*)[142]

C. cerastes, C. gasperettii, C. vipera, and *Pseudocerastes persicus* inhabit the vast arid deserts of North Africa and the Middle East. *Cerastes* species are the most common cause of snakebite in these regions. A few fatal cases were reported in the nineteenth century French colonial military literature, but there have been no recent reports. Envenoming usually results in local pain and swelling, complicated by necrosis in some cases. Nausea, vomiting, spontaneous bleeding, and coagulopathy have been observed in some cases. Recently, disseminated intravascular coagulation, microangiopathic hemolysis, and acute tubular necrosis were described in two proven cases of envenoming by *C. cerastes*.[90]

Envenoming by Pit Vipers (Crotalinae)

Envenoming by Japanese and Chinese Mamushis (*Gloydius/Agkistrodon blomhoffii*)

In Japan, bites by the Japanese mamushi *(Gloydius/Agkistrodon b. blomhoffii)* are mainly inflicted on the hands. There is local swelling with blistering, nausea, vomiting, fever, headache, abdominal and lumbar pain, shock, acute renal failure, bleeding gums, and ecchymoses. Neurotoxic features are confined to ptosis and external ophthalmoplegia, which can develop between 1 and 48 hours after the bite.

In the oriental region of China, north of latitude 25°N, the Chinese mamushi *(Gloydius/Agkistrodon b. brevicaudus)* is the most common cause of snakebite. There is local swelling and neurotoxic symptoms such as blurring of vision, ptosis, and diplopia within 24 hours of the bite. Breathlessness, dysphagia,

and difficulty opening the mouth are associated with symptoms suggesting generalized rhabdomyolysis. Some patients require assisted ventilation.

Envenoming by the Malayan Pit Viper (*Calloselasma rhodostoma*)[83,158]

This species is an important cause of snakebite in northwestern Malaysia, Thailand, Laos, Cambodia, Vietnam, and Java. It inhabits coffee and rubber plantations in areas of cleared jungle. Bites are a major occupational hazard of plantation workers. About half the patients develop minimal or no envenoming, but in the remainder, local swelling starts within minutes and reaches its maximum after 24 to 72 hours. Local necrosis develops in 11% of victims and is always preceded by blistering. Secondary infection by bacteria peculiar to the venom and oral cavity of the snakes is common.[112] The blood becomes incoagulable as early as 30 minutes after the bite and, in untreated patients, may persist for 1 to 26 days.[83] Most patients have profound thrombocytopenia, probably resulting from sequestration. Local necrosis may be complicated by fatal tetanus. Fatal cases in Thailand were attributed to cerebral hemorrhage, shock, tetanus, septicemia, and anaphylaxis.[57]

Envenoming by Asian Arboreal Pit Vipers (Genus *Trimeresurus sensu lato*)[40,143]

In Thailand, the white-lipped green pit viper *(T. [Cryptelytrops] albolabris)* is the most widely distributed venomous snake and the second most common cause of snakebite (27% of cases).[133] Pain and swelling develop early after the bite, spreading to involve more than half the bitten limb in 46% of cases. Local bruising and tender enlargement of local lymph nodes are common, while local blistering and necrosis are rare. Coagulopathy, thrombocytopenia, and leukocytosis may develop. In Burma, *T. (Cryptelytrops) erythrurus* bites resulted in no evidence of envenoming in 65% of cases. In the rest, there was local swelling without necrosis, incoagulable blood, and thrombocytopenia. Although the Indian bamboo viper *(T. gramineus)* is the most abundant pit viper in the southern half of India, there is little reliable information about the effects of envenoming. In south India, the Malabar pit viper *(T. malabaricus)* is said commonly to cause local necrosis.[40,61] In Thailand, the dark green pit viper *(T. [Cryptelytrops] macrops)* is a common cause of mild envenoming in some areas, notably in gardens in central Bangkok, while the Kanchanaburi pit viper *(T. [Cryptelytrops] kanburiensis)* is capable of causing severe envenoming.[157] The Chinese habu *(T. [Protobothrops] mucrosquamatus)* causes about one third of venomous snakebites in Taiwan and results in an 8% case-fatality rate, whereas the Chinese green pit viper *(T. stejnegeri)*, although responsible for 53% of bites in Taiwan, carries a case-fatality rate of only 1%.

CLINICAL COURSE AND PROGNOSIS

Risk of Envenoming

Even when the fangs of a venomous snake have pierced the skin, envenoming is not inevitable. About 20% of patients bitten by *Calloselasma rhodostoma* and *Daboia russelii* show absolutely no evidence of envenoming, and as many as 80% of those bitten by sea snakes and Australasian common brown snakes *(Pseudonaja textilis)* and 50% by *C. rhodostoma* or *D. russelii* have trivial or no envenoming. These are the so-called dry bites.

Risk of Death

Untreated snakebite mortality is hard to assess, for hospital admissions include a disproportionate number of severe cases, and data for untreated snakebites are available only from the preantivenom era or from occasions when antivenom supply is limited, an antivenom of low potency is used,[151] or when antivenom is withheld by doctors who doubt its efficacy. The mortality of *E. ocellatus* bites has been reduced from about 20% to 3% with antivenom.[151,154] Prognosis appears to be the worst in infants and in the elderly, but there is no convincing evidence that older children have a worse prognosis than young adults do, despite the larger dose of venom they may receive relative to their body weight.

Interval between Bite and Death

Death after snakebite may occur as rapidly as within a few minutes (reputedly after a bite by the king cobra, *O. hannah*) or as long as 41 days after a bite by the saw-scaled or carpet viper *(E. carinatus)*. However, the speed of killing has been exaggerated. Most elapid deaths occur within hours of the bite, most sea snakebite deaths between 12 and 24 hours, and viper bite deaths within days.

Rate of Evolution and Recovery of Envenoming

Local swelling is usually evident within 2 to 4 hours of bites by vipers and cytotoxic cobras. Swelling is maximal and most extensive on the second or third day after the bite. Resolution of swelling and restoration of normal function in the bitten limb may be delayed for months, especially in older people (e.g., after bites by the European adder *V. berus*). The earliest systemic symptoms, such as vomiting and syncope, may develop within minutes of the bite, but even in the case of rapidly absorbed elapid venoms, patients rarely die less than an hour after the bite. Defibrination may be complete within 1 to 2 hours of the bite (e.g., saw-scaled or carpet viper *E. ocellatus*).[135,153] Neurotoxic signs may progress to generalized flaccid paralysis and respiratory arrest within a few hours. If the venom is not neutralized by antivenom, these effects may be prolonged. Defibrination can persist for weeks (*Echis* sp. and *C. rhodostoma*).[83,135,153] Patients with neurotoxic envenoming have recovered after being artificially ventilated for up to 10 weeks. Tissue necrosis usually declares itself within a week of the bite. Sloughing of necrotic tissue and secondary infections including osteomyelitis may occur during subsequent weeks or months. Deaths occurring from neurotoxic envenoming are caused by airway obstruction or respiratory paralysis, whereas later deaths may result from technical complications of mechanical ventilation or intractable hypotension. Late deaths, more than 5 days after the bite, are usually the result of renal failure. Delayed shock with recurrent spontaneous hemorrhage has been described in victims of Burmese Russell's viper; pituitary and other intracranial hemorrhages have been found at autopsy.

Chronic Sequelae of Snakebite

Tissue loss, amputations, contractures, arthrodeses, hypertrophic and keloid scars, tendon damage, and complications of surgery are the most common causes of persistent morbidity in survivors of snakebites. Chronic ulcers, pyogenic arthritis, and osteomyelitis are described. Malignant transformation

(Marjolin's ulcer) has been seen in patients with chronic ulceration after bites by *N. nigricollis*[155] and *C. rhodostoma*.[38,158] Chronic renal failure (bilateral cortical necrosis) and panhypopituitarism (from Sheehan like syndrome) are reported complications of Russell's viper bites.[130,137] Cerebral hemorrhage complicating viperid, colubrid, and Australasian elapid bites may result in chronic neurologic deficits, and it is possible that survivors of severe presynaptic neurotoxicity (e.g., from krait and Australasian elapid envenoming) may be susceptible to a late poliomyelitis-like syndrome. Apart from these purely objective abnormalities, snakebite victims may complain of chronic or recurrent symptoms in the bitten limb and attribute a wide variety of physical and mental problems to that unforgettable, dramatic, and traumatic event of their snakebite.

LABORATORY INVESTIGATIONS

Hematology

Systemic envenoming is usually associated with a neutrophil leukocytosis: counts above 20×10^9/L indicate severe envenoming. Initially, hematocrit may be high from hemoconcentration when there is generalized increase in capillary permeability (e.g., Burmese *D. siamensis*). Later, hematocrit falls because of bleeding into the bitten limb and elsewhere, and from intravascular hemolysis or microangiopathic hemolysis in patients with disseminated intravascular coagulation. Thrombocytopenia is common (e.g., *D. russelii, C. rhodostoma, B. arietans*).

Twenty-Minute Whole Blood Clotting Test[87,153]

Incoagulable blood is a cardinal sign of systemic envenoming by most of the Viperidae, many of the Australasian elapids, and the medically important Colubridae. For clinical purposes, a simple, bedside, all-or-nothing test of blood coagulability is adequate. A few milliliters of blood taken by venipuncture are placed in a new, clean, dry, glass vessel; left undisturbed at room temperature for 20 minutes; then tipped once to see if there is clotting or not (Fig. 49-52).

Figure 49-52. Twenty-minute whole blood clotting test. In this Papua New Guinean patient with persistent bleeding from scarifications made at the bite site after a bite by a Papuan taipan *(Oxyuranus scutellatus canni),* a few milliliters of venous blood were placed in a new, clean, dry, glass bottle and left to stand at room temperature for 20 minutes. At the end of this time the blood remained liquid, indicating defibrinogenation. (Copyright D.A. Warrell.)

Other Tests of Hemostasis

More sensitive laboratory tests that are rapid and relatively simple to perform are whole blood or plasma prothrombin times and detection of elevated concentrations of FDP by agglutination of sensitized latex particles or of D-dimer. Serum concentrations of creatine kinase, aspartate transferase, and blood urea are commonly raised in patients with severe envenoming, because of local muscle damage at the site of the bite.

Evidence of Muscle Damage

Generalized rhabdomyolysis caused by sea snake, Australasian elapid, tropical rattlesnake, and Sri Lankan Russell's viper bites causes a steep rise in serum creatine kinase and other muscle-derived enzymes, myoglobin, and potassium concentrations. Plasma is stained brownish by myoglobin and pink by hemoglobin. Heparinized blood should be allowed to sediment spontaneously (without centrifugation) to reveal these pigments.

Evidence of Intravascular Hemolysis

Patients with intravascular hemolysis have black urine (as in malarial "blackwater fever"). It is brownish, pinkish, or reddish in those with hematuria or myoglobinuria.

Evidence of Renal Dysfunction and Acid-Base Imbalance

Blood urea or serum creatinine and potassium concentrations should be measured in patients who become oliguric, especially in cases with a high risk of renal failure (e.g., *D. russelii,* terrestrial Australasian snakes, sea snakes, and Colubridae). All snake-bitten patients should be encouraged to empty their bladder on admission. Urine should be examined for blood/hemoglobin and protein (by dipstick test) and for microscopic hematuria and red cell casts. Severely sick, hypotensive, and shocked patients will develop lactic acidosis (suggested by an increased anion gap). Those with renal failure will also develop a metabolic acidosis (decreased plasma pH and bicarbonate concentration, reduced arterial P_{CO_2}), and patients with respiratory paralysis will develop respiratory acidosis (low pH, high arterial P_{CO_2}, decreased P_{O_2}) or respiratory alkalosis if they are mechanically overventilated.

Electrocardiographic Abnormalities[53,54,81,135,147,153]

These include sinus bradycardia, ST-T wave changes, various degrees of atrioventricular block, and evidence of hyperkalemia. Shock may induce myocardial ischemia or infarction in patients with diseased coronary arteries.

Chest Radiography[150]

Chest radiography is useful for detecting pulmonary edema (e.g., European *Vipera* and *D. russelii/D. siamensis*), pulmonary hemorrhages and infarcts, pleural effusions, and secondary bronchopneumonia.

Immunodiagnosis

Detection of venom antigens in body fluids of snakebite victims using enzyme immunoassays has proved a valuable research tool for confirming the species responsible for envenoming (immunodiagnosis), as a prognostic index of the severity of envenoming, and to assess the efficacy of antivenom treatment.[31,39,40,95,103,109,110,129] Of the various techniques used, enzyme immunoassay (EIA) has proved the most rapid, sensitive, and specific. However, commercial venom detection kits for clinical

diagnosis are available only in Australia (Commonwealth Serum Laboratories Venom Detection Kit).[104] They are highly sensitive, but their specificity may be inadequate to distinguish between envenoming by different species in the same genus or in closely related genera.[104,126] Envenomed patients develop persisting levels of antibodies to venom antigens, including individual toxins[93] detectable by EIA, but they have proved insufficiently specific to allow reliable retrospective diagnosis.[39,133]

MANAGEMENT OF SNAKEBITE[11,64,104,125,148,150,171]

First Aid Treatment[13,138,139]

Principles of First Aid

1. Reassure the victim, who is often terrified.
2. Do not tamper with the bite wound in any way, but immobilize the bitten limb using a splint or sling. If the patient is thought to have been bitten by a dangerously neurotoxic elapid snake (including sea snakes), consider pressure-immobilization (see later).
3. Take the patient as quickly as possible to the nearest medical care facility. The whole patient should be immobilized, especially the bitten limb, because any muscular contractions will promote spread of venom. Ideally, the patient should be transported by motor vehicle, bicycle (as a passenger), boat, or on a stretcher.
4. Avoid harmful and time-wasting treatments.
5. Because species diagnosis is critically important, the snake should be taken along to the hospital if it has already been killed. However, if the snake is still at large, do not risk further bites and waste time by searching for it. Even snakes that appear to be dead should not be touched with the bare hands, but carried in a bag or dangling across a stick. Some species (e.g., *Hemachatus haemachatus*) sham death, and even a severed head can inject venom! So be very careful.

Pressure Immobilization

The splinting and crepe bandaging method (pressure immobilization; see Figure 48-27) developed and advocated by Struan Sutherland in Australia proved effective in limiting the absorption of Australian elapid toxins in restrained monkeys.[103] Although never subjected to formal clinical trials, the method has proved successful, as judged by some anecdotal reports of delayed systemic envenoming and rapid deterioration after release of the bandage, in some cases supported by measurements of venom antigenemia. However, there have been practical difficulties in implementing this discovery and, even in Australia, only 18% to 53% of the bandages in place when patients arrived in the hospital had been correctly and effectively applied. The method is still regarded by some experienced physicians as of unproven benefit.[13] Crepe bandaging is thought to exert a pressure of about 55 mm Hg, that of a venous tourniquet. In practice, it is difficult to judge how tightly the bandage should be applied and difficult for the patient to put it on unaided.[150b] This is why so many are incorrectly applied.[13] External compression increases intracompartmental pressure and may accentuate the effects of some necrotic snake venoms, so the method may not be appropriate for all cases of snakebite.[138,139] However, if a patient is bitten by a dangerously neurotoxic elapid (such as a mamba, king cobra, or taipan) or by a sea snake, there is a risk that respiratory paralysis might develop en route to the hospital. In these cases, it is recommended that a crepe bandage and splint be applied as tightly as for a sprained ankle, but not so tightly as to obliterate the peripheral arterial pulse. Lymphoscintigraphy studies in simulated envenoming showed that excessive pressure (>70 mm Hg) and movement of the other limbs increased lymphatic flow.[40] The patient should lie down and remain as immobile as possible during the journey to hospital.

An alternative, simpler method of local compression has been developed by Tun-Pe and his colleagues in Burma. Application of a foam rubber pad directly over the bite wound delayed systemic envenoming, as assessed by measurements of venom antigenemia, and the method appeared safe and effective in a preliminary field trial.[128]

Rejected or Controversial First Aid Methods[139,150b]

Traditional treatments are widely preferred by the inhabitants of rural parts of the developing world.[71] However, even in the West, tourniquets, constriction bands, wound cauterization, incision or excision, amputation of the bitten digit, suction by mouth, vacuum pumps[9] or "venom-ex" apparatus, instillation of chemical compounds such as potassium permanganate, application of ice packs (cryotherapy), "snake stones," electric shocks, and many other outlandish first aid treatments have been advocated. These are absolutely contraindicated in that they are harmful and have no proven benefit.[33,139]

Incisions can provoke uncontrolled bleeding when the blood is incoagulable (see Figure 49-52, *O. s. canni* bite; Fig. 49-53, *E. ocellatus* bite); may damage nerves, blood vessels, or tendons; and introduce infection.[5] Suction, chemicals, and cryotherapy can cause tissue necrosis. Snake stones do not remove venom from the wound and usually require an incision to make them adhere. This can result in persistent bleeding and infection (Fig. 49-54, *E. ocellatus* bite).

Dangers of Tight Tourniquets

Tight (arterial) tourniquets have been responsible for terrible morbidity and even mortality in snakebite victims and should never be used.

Figure 49-53. Profuse and persistent bleeding from incisions, performed as an ill-advised first aid method after a bite by a saw-scaled viper *(Echis ocellatus)* near Gombe, Nigeria. On admission to the hospital 20 hours after the bite, the hematocrit was only 15%. (Copyright D.A. Warrell.)

Figure 49-54. Traditional black snake stone used by a patient bitten by a saw-scaled viper *(Echis ocellatus)* near Kaltungo, Nigeria. To make the stone stick to the bite wound, an incision is made, which, in this case, led to persistent bleeding and infection. (Copyright D.A. Warrell.)

Figure 49-55. Lateral popliteal palsy resulting from peripheral nerve damage inflicted by a tight tourniquet applied just below the right knee around the neck of the fibula following a bite by a Russell's viper *(Daboia siamensis)* near Tharrawaddy, Burma. The patient is unable to flex or evert his right foot. (Copyright D.A. Warrell.)

Dangers of tourniquets include ischemia and gangrene if they are applied for more than about 2 hours, damage to peripheral nerves (especially the lateral popliteal (common peroneal) nerve when they press against the neck of the fibula) (Fig. 49-55, *D. siamensis* bite), increased fibrinolytic activity, congestion, swelling, increased bleeding, increased local effects of venom and shock, rapid development of life-threatening systemic envenoming, or even pulmonary embolism after their release (see later).[138,139]

Treatment of Early Symptoms before the Patient Reaches the Hospital

Distressing and dangerous manifestations of envenoming may appear before the patient reaches the hospital:

- Local pain. This may be intense. Oral acetaminophen is preferable to aspirin or nonsteroidal anti-inflammatory agents, which carry the risk of gastric bleeding in patients with incoagulable blood. Severe pain should be treated with opiates.
- Vomiting. This is a common early symptom of systemic envenoming. Patients should be laid in the recovery position (on their left side) with their head down to avoid aspiration. Persistent vomiting can be treated with intravenous promethazine (12.5 to 25 mg in adults, 0.1 to 0.25 mg/kg in children older than 2 years of age).
- Syncopal attacks and anaphylactic shock. Patients who collapse within minutes of the bite may show features of either a vasovagal attack with profound bradycardia or of anaphylaxis with angioedema, urticaria, asthma, abdominal colic, and diarrhea. Anaphylaxis should be treated with aqueous epinephrine 0.1% (1:1000) (0.3 to 0.5 mL in adults, 0.01 mL/kg in children) by intramuscular injection, followed by a histamine H_1-blocker such as chlorpheniramine maleate (10 mg in adults, 0.2 mg/kg in children) given by intravenous or intramuscular injection. In patients with incoagulable blood, injections can cause hematomas. Pressure dressings should be applied to all injection sites to prevent oozing.
- Respiratory distress. This may result from upper airway obstruction if the jaw, tongue, and bulbar muscles are paralyzed or from paralysis of the respiratory muscles. Patients should be placed in the recovery position, the airway cleared if possible using a suction pump, an oral airway inserted, and the jaw elevated. If the patient is cyanotic or respiratory movements are weak, oxygen should be given by any available means. If clearing the airway does not produce immediate relief, artificial ventilation must be initiated. In the absence of any equipment, mouth-to-mouth or mouth-to-nose ventilation can be lifesaving. Manual ventilation by bag-valve-mask is rarely effective. Ideally, a cuffed endotracheal tube should be introduced using a laryngoscope, or a cuffed tracheostomy tube inserted. The patient can then be ventilated by bag. If no femoral or carotid pulse can be felt, external cardiac massage should be instituted.

Examination of Pregnant Women

Potential complications of envenoming in pregnancy include antepartum and postpartum hemorrhage, premature labor, abortion/stillbirth, and fetal distress. If possible, uterine contractions and fetal heart rate should be monitored continuously. Fetal distress may be signaled by fetal bradycardia, tachycardia, or late deceleration after each uterine contraction. If there is vaginal bleeding or the likelihood of imminent surgery, correction of antihemostatic abnormalities after antivenom treatment should be accelerated using blood products.

Medical Treatment in the Hospital

Snakebite is a medical emergency. The history, symptoms, and signs must be assessed rapidly to direct urgent appropriate treatment. Patients may arrive at the hospital soon, or in some cases many days, after being bitten. They may, therefore, show early or late signs of envenoming or its complications. It is essential that all patients with a history of snakebite be assessed rapidly; they may be moribund but still salvageable by appropriate resuscitation. Cardiopulmonary resuscitation may be needed, including clearance of the airway, oxygen administration by face mask or nasal catheter, and establishment of intravenous access to allow treatment with drugs and intravenous fluids.

Airway, respiratory movements (breathing), and arterial pulse (circulation) must be checked immediately. Vital signs must be recorded: blood pressure, pulse rate, and respiratory rate.

Need for Rapid Assessment and Resuscitation

Patients with severe envenoming may present with the following problems requiring urgent intervention:

- Profound hypotension and shock, resulting from:
  - Direct cardiovascular effects of the venom (e.g., *V. berus, D. russelii, D. siamensis, B. arietans, O. scutellatus*)
  - Hypovolemia secondary to blood loss, persistent vomiting, or other causes of dehydration
  - Autopharmacologic effects of the venom (activation/inhibition of physiologic vasomotor systems, such as the angiotensin-renin-bradykinin systems, by venom toxins)
  - Rarely, anaphylaxis provoked by antivenom given outside the hospital and, even more rarely, provoked by venom in habitual snake handlers who have been sensitized by previous exposure
- Sudden deterioration after release of a tourniquet or compression bandage, resulting in shock, bleeding, or respiratory paralysis.[148,167] These bands, bandages, or ligatures are often removed too hastily by hospital staff before antivenom treatment has been initiated and appropriate staff and equipment are on hand in the event that resuscitation is needed.
- Airway obstruction, resulting from aspirated vomitus, a foreign body, or the tongue blocking the upper airway, especially in patients with evolving bulbar paralysis who have not been transported to hospital in the left lateral (recovery) position. Vomiting can be the result of systemic envenoming or ingestion of emetic herbal remedies.
- Terminal respiratory failure from progressive neurotoxic envenoming that has led to paralysis of the respiratory muscles.
- (Hours after the bite) Cardiac arrest resulting from hyperkalemia in patients with massive generalized skeletal muscle breakdown (rhabdomyolysis) after sea snakebites.
- (Days after the bite) Acute renal failure.
- (Days after the bite) Septicemia complicating aspiration pneumonia (see earlier text) or infection of incisions made at the site of the bite.

Clinical Assessment

Four important preliminary questions are as follows: (1) Where (in which part of your body) were you bitten? (2) How long ago were you bitten? (3) Have you brought the snake or, if not, what did it look like? (4) How are you feeling now?

If the snake has been killed but not brought, someone should be dispatched to collect it. Only if the snake can be identified confidently as nonvenomous can the patient be discharged after a booster dose of tetanus toxoid. Patients should be asked whether they have taken any drugs or alcohol, whether they have vomited, fainted, or have noticed any bleeding or other ill effects of the bite, and whether they have passed urine since being bitten. Physical signs should be assessed before any compression bandage or tourniquet is removed. Fang marks are sometimes invisible and rarely help the diagnosis, although the discovery of only two or three discrete puncture marks suggests a bite by a venomous snake. Local swelling, tenderness, and lymph node involvement are early signs of envenoming. The gingival sulci are usually the earliest site of detectable spontaneous bleeding (see Figure 49-43). Bleeding from venipuncture sites, recent wounds, and skin lesions suggests incoagulable blood. If the patient is in shock (collapsed, sweating, cool, cyanotic extremities, low blood pressure, tachycardia), the foot of the bed should be raised, and an intravenous infusion of a plasma expander, such as fresh frozen plasma, dextran, Haemaccel (3.5% colloidal solution), Gelofusine (succinated gelatin), or fresh blood started immediately. The central venous pressure should be observed. The earliest symptom of neurotoxicity after elapid bites is often blurred vision, a feeling of heaviness in the eyelids, and drowsiness. The earliest sign is contraction of the frontalis muscle (raised eyebrows and puckered forehead) even before true ptosis can be demonstrated. Signs of respiratory muscle paralysis (dyspnea, "paradoxical" abdominal respiration, and cyanosis) are ominous. Patients with generalized rhabdomyolysis may have trismus and muscles that are stiff, tender, and resistant to passive stretch. Urine output may dwindle very early in the course of Russell's viper bite. Dark urine suggests myoglobinuria or hemoglobinuria.

If there is initially no evidence of envenoming, the patient should be admitted for observation, ideally for 24 hours. Every hour, symptoms, level of consciousness, degree of ptosis, pulse rate and rhythm, blood pressure, respiratory rate, extent of local swelling, and other new signs should be recorded. If there is any evidence of neurotoxicity, ventilatory capacity should be recorded every hour. Useful investigations include the 20-minute whole blood clotting test (or other tests of coagulation), peripheral leukocyte count, hematocrit, urine microscopy and dipstick testing, and electrocardiography.

If a tourniquet or tight compression bandage is in place, it may create distal edema through venous congestion even in the absence of envenoming and, if applied tightly enough to prevent arterial perfusion, might cause a cold, pulseless, and cyanotic extremity. Blockage of venous drainage from a limb into a powerfully procoagulant venom that has been injected by the snake may promote deep vein thrombosis and subsequent pulmonary embolism when the tourniquet is released. Such occlusive devices must not be released until intravenous access has been established and antivenom, plasma expanders, and adequate medical staff are available to cope with the dramatic deterioration that may follow.

Early Clues That a Patient May Be Severely Envenomed

- The snake is identified as a dangerously venomous species and was perhaps a large specimen.
- Enlarged, painful, and tender lymph nodes draining the site of the bite indicate early spread of large-molecular-weight venom components into the lymphatic system.
- Early symptoms of systemic envenoming include collapse (hypotension, shock); nausea, vomiting, and diarrhea; severe headache; "heaviness" of the eyelids; pathologic drowsiness or early ptosis/ophthalmoplegia.
- Early spontaneous systemic bleeding occurs, for example, from the gums or nose, or in vomitus, feces, or urine.
- Urine is dark brown or black.

Antivenom (Antivenom, Antivenene, Antisnakebite Serum)[115]

Antivenom is the concentrated whole gamma immunoglobulin (IgG) or enzyme-refined gamma IgG fragments ($F(ab')_2$ or Fab) of horses or sheep, which have been immunized with venom.[32,71a,115] It is the only specific treatment available and has

proved effective against many of the lethal and damaging effects of venoms. In the management of snakebite, the most important clinical decision is whether or not to give antivenom, because only a minority of snake-bitten patients need it, it may produce severe reactions, and it is expensive and often in short supply.

Indications for Antivenom

1. Systemic envenoming
   - Hemostatic abnormalities: spontaneous systemic bleeding, incoagulable blood or prolonged clotting time, severe thrombocytopenia
   - Cardiovascular abnormalities: hypotension, shock, abnormal electrocardiogram, cardiac arrhythmia
   - Neurotoxicity
   - Generalized rhabdomyolysis
   - In patients with definite signs of local envenoming, the following indicate significant systemic envenoming: neutrophil leukocytosis, elevated serum enzymes such as creatine kinase and aminotransferases, hemoconcentration, uremia, raised serum creatinine, oliguria, hypoxemia, acidosis, and vomiting in the absence of a history of ingesting emetic agents
2. Severe local envenoming: local swelling that is spreading rapidly or that extends more than halfway up the bitten limb; extensive blistering or bruising, especially in patients bitten by species known to cause local necrosis (e.g., Viperidae, Asian cobras, African spitting cobras). Bites by these species inflicted on the digits carry a high risk of necrosis and warrant antivenom treatment. In Europe, to improve the rate of recovery after bites by *V. berus,* antivenom has been recommended in adults with swelling extending beyond the wrist or ankle within 2 hours of a bite.[81,147]

Special Indications for Antivenom in Specific Areas

Australia. Antivenom should be given to any patient with proved or suspected snakebite if there are tender regional lymph nodes or any other evidence of systemic spread of venom and in anyone effectively bitten by an identified highly venomous species.[104,171]

Europe (Adder—*Vipera berus*—and other European *Vipera*). Zagreb antivenom, or Protherics ViperaTAb (see Table 49-4), is indicated to prevent morbidity and reduce the length of convalescence in patients with moderately severe envenoming, as well as to save the lives of severely envenomed patients. Indications are as follows:

- Fall in blood pressure with or without signs of shock
- Other signs of systemic envenoming (see earlier text), including spontaneous bleeding, coagulopathy, pulmonary edema or hemorrhage (shown by chest radiograph), ECG abnormalities, peripheral leukocytosis, profound anemia, or elevated serum creatine kinase
- Severe local envenoming—swelling of more than half the bitten limb developing within 48 hours of the bite—even in the absence of systemic envenoming
- In adults, early swelling extending within 4 hours of the bite beyond the wrist after bites on the hand or beyond the ankle after bites on the foot

Patients bitten by European *Vipera* who show any evidence of envenoming should be admitted to hospital for observation for at least 24 hours. Antivenom should be given whenever there is evidence of systemic envenoming (see earlier text) even if its appearance is delayed for several days after the bite.

Contraindications to Antivenom Treatment

There are no absolute contraindications to antivenom in cases of life-threatening envenoming. However, patients with atopic asthma and those who have reacted to equine antiserum on previous occasions have an increased risk of developing severe antivenom reactions. In such cases, antivenom should not be given unless there is evidence of systemic envenoming. If antivenom is given, pretreatment with adrenaline (epinephrine), antihistamine, and a corticosteroid is recommended. Rapid desensitization is not recommended.

Choice of Antivenom in a Particular Case: Monovalent and Polyvalent Antivenoms

The range of venoms neutralized by an antivenom is usually stated on the package insert and is to be found in compendia of antivenoms.[64,114,148,171] If the biting species is known or strongly suspected, the appropriate monovalent (monospecific) antivenom should be used. Monovalent antivenom is raised against the venom of a single species and is effective for treating envenoming by that species of snake alone, or, in some cases, for envenoming by a few closely related species. They are generally less expensive than are polyvalent antivenoms.[126] In parts of the world where several species produce identical signs, patients who fail to bring the dead snake (or, in Australia, who cannot be diagnosed by venom detection kits) must be treated with polyvalent (polyspecific) antivenom. Polyvalent antivenom is raised against the venoms of a selection of the most important species of snakes in a particular geographic region. This will contain a relatively lower concentration of species-specific antibody per unit volume (or total protein content) than does a monovalent antivenom. A relatively higher dose of polyvalent than monovalent antivenom will therefore be required, carrying a higher risk of reactions.

The chosen antivenom must have demonstrated ability to protect against the venom of the species thought to have been responsible. Nonspecific antivenom is generally of no benefit to the patient and may cause reactions (see later text). Some antivenom manufacturers claim a wide range of "para-specific" neutralization for their products, but mere immunologic cross-reactivity in the laboratory is inadequate grounds for relying on an antivenom to be effective clinically. Table 49-4 lists some important antivenoms for this region. In some cases, clinical trials have established an average initial dose.*

Conservation of Antivenom

Liquid antivenoms stored at below 8°C (46°F) usually retain most of their activity for 5 years or more.[173] Freeze-dried (lyophilized) antivenoms are even more stable. Under expedition conditions, liquid antivenoms can be expected to retain their activity even if exposed to environmental temperatures above 30°C (86°F).[173] Opaque solutions should not be administered because precipitation of protein indicates loss of activity and increased risk of adverse reactions.

Supply of Antivenoms

Antivenom manufacturers and suppliers change often, so availability of antivenom is uncertain. For this reason, a timely inter-

*References 53, 97, 109, 115, 122, 146, 154, 158, 161, 165, 166.

TABLE 49-4. Guide to Initial Dosage of Some Antivenoms for Treating Bites by Medically Important Snakes Outside the Americas

SPECIES LATIN NAME	ENGLISH NAME	MANUFACTURER, ANTIVENOM	APPROXIMATE AVERAGE INITIAL DOSE
Acanthophis species	Death adder	CSL[1] Death Adder or Polyvalent Antivenom	1–3 vials
Bitis arietans (Africa)	Puff adder	Sanofi-Pasteur ("Fav Afrique" or "FaviRept") polyvalent; SAVP[2] polyvalent	80 mL
Bitis arietans (Middle East)	Puff adder	NGHA[3] Polyvalent Snake Antivenom	80 mL
		Vacsera Polyvalent or Anti-Viper Venom Antiserum	80 mL
Bungarus caeruleus	Common krait	Indian manufacturers[4] polyvalent	100 mL
Bungarus candidus	Malayan krait	Thai Red Cross Malayan Krait Antivenom	50 mL
Bungarus multicinctus	Chinese krait	Shanghai Vaccine & Serum Institute Antivenom of *Bungarus multicinctus*	5 vials
		Blyth Taiwan NIPM Taipei Naja-Bungarus antivenom	5 vials
Calloselasma (Agkistrodon) rhodostoma	Malayan pit viper	Thai Red Cross, Malayan Pit Viper Antivenom monovalent	100 mL
Cerastes species	Desert (horned) vipers	NGHA[3] Polyvalent	30–50 mL
		Vacsera AntiViper or Polyvalent	30–50 mL
Daboia (Vipera) palaestinae	Palestine viper	Rogoff Medical Research Institute, Tel Aviv, Palestine viper monovalent	50–80 mL
Daboia (Vipera) russelii, D. siamensis	Russell's vipers	Myanmar Pharmaceutical Industry, monovalent	40 mL
		Indian manufacturers[4] polyvalent	100 mL
		Thai Red Cross, Russell's Viper Antivenom monovalent	50 mL
Dendroaspis species	East/South African mambas	SAVP[2] *Dendroaspis* or Polyvalent Antivenoms	50–100 mL
Dispholidus typus	African boomslang	SAVP[2] Boomslang Antivenom	1–2 vials
Echis species (Africa)	Saw-scaled or carpet vipers	SAVP[2], *Echis*, monovalent	20 mL
		Sanofi-Pasteur ("Fav Afrique")	100 mL
Echis species (Middle East)	Saw-scaled or carpet vipers	NGHA[3] Polyvalent Snake Antivenom	50 mL
		Vacsera Polyvalent ans Anti-Viper Venom Antiserum	50 mL
Echis carinatus (India)	Saw-scaled or carpet vipers	Indian manufacturers[4] polyvalent	50 mL
Gloydius (Agkistrodon) blomhoffii	Chinese mamushi	Shanghai Vaccine & Serum Institute Mamushi antivenom	5 vials
Hydrophiinae	Sea snakes	CSL[1] Sea Snake Antivenom	1–10 vials
Micropechis ikaheka	New Guinean small-eyed snake	CSL[1] Polyvalent Antivenom	2 vials
Naja kaouthia	Monocellate Thai cobra	Thai Red Cross, monovalent	100 mL
Naja naja, N. oxiana	Indian cobras	Indian manufacturers[4] polyvalent	100 mL
Naja haje, N. anchietae, N. annulifera, N. melanoleuca, N. nivea (Africa)	African neurotoxic cobras	SAVP[2] and Sanofi-Pasteur Polyvalent	100 mL
Naja haje (Middle East)	Egyptian cobra	Vacsera Polyvalent Venom Antiserum	100 mL
Naja haje arabica	Arabian cobra	NGHA[3] Bivalent Naja/Walterinnesia or Polyvalent Snake Antivenom	100 mL
Naja nigricollis, N. mossambica, etc.	African spitting cobras	SAVP[2] and Sanofi-Pasteur Polyvalent	100 mL
Naja (nigricollis) nubiae	Egyptian spitting cobra	Vacsera Polyvalent Venom Antiserum	100 mL
Notechis scutatus	Tiger snake	CSL1 Tiger Snake or Polyvalent Antivenom	1–3+ vials
Oxyuranus scutellatus	Australian/Papuan taipans	CSL[1] Taipan or Polvalent Antivenom	1–6+ vials
Paranaja species	Australian brown snakes	CSL[1] Brown Snake or Polyvalent Antivenom	1–6+ vials
Pseudechis species	Australian black snakes	CSL[1] Black Snake Antivenom	1–3 vials
Rhabdophis tigrinus, R. subminiatus	Japanese yamakagashi, Southeast Asian red-necked keelback	Japanese Snake Institute, Nitta-gun Yamakagashi antivenom	1–2 vials
Trimeresurus (Cryptelytrops) albolabris, T. (Cryptelytrops) macrops	Green pit vipers	Thai Red Cross Green Pit Viper Antivenom	100 mL
Vipera berus, and other European *Vipera*	European adder	Immunoloski Zavod-Zagreb Vipera polyvalent	10–20 mL
		Protherics Fab monovalent "ViperaTAb"	100–200 mg
Walterinnesia aegyptia	Black desert cobra	NGHA[3] Bivalent Naja/Walterinnesia or Polyvalent Snake Antivenom	50 mL

[1]Commonwealth Serum Laboratories, Parkville, Australia.
[2]South African Vaccine Producers, formerly SAIMR, Johannesburg.
[3]National Guards Hospital, Riyadh, Saudi Arabia.
[4]Indian manufacturers: Bengal Chemicals & Pharmaceuticals, Kolkata: Bharat Serums & Vaccines, Mumbai; Central Research Institute, Kasauli; Haffkine Biopharma, Mumbai; King Institute, Chennai; Serum Institute of India, Pune; Vins Bioproducts, Hyderabad.

net search (see Useful Websites) followed by confirmation of clinical efficacy and safety, as well as immediate availability, are essential, well in advance of the time when antivenom is needed. In urgent cases, some national poison centers (e.g., London, UK) and zoo networks hold stocks of antivenoms covering most exotic and local species. Europe, Asia, and Australia are reasonably well supplied with antivenoms, but there is currently a crisis in antivenom supply to the sub-Saharan countries of Africa and to New Guinea.[32a] This problem may be relieved by the production of new "pan-African" polyvalent antivenoms, raised against appropriate African venoms, by manufacturers outside of Africa.[32,49,50]

Beware, some clinically ineffective, nonspecific but highly reactogenic antivenoms raised against Indian venoms (e.g., *Naja, Echis*) are sold in African countries, where they are purported to be effective against venoms of the local African species.

Prediction of Antivenom Reactions

Hypersensitivity testing by intradermal or subcutaneous injection or intraconjunctival instillation of diluted antivenom has been widely practiced in the past. However, these tests delay the start of antivenom treatment, are not without risk, and have no predictive value for early (anaphylactic) or late (serum sickness type) antivenom reactions, which are not usually manifestations of IgE-mediated type I hypersensitivity.[16,61]

Prevention of Early Antivenom Reactions

Prophylactic antihistamines (anti-H1 and anti-H2), corticosteroids, and epinephrine have been very widely used, singly or in combination, on empirical grounds, but not without risk. A few randomized controlled trials have now been completed and several more are in progress. Intramuscular promethazine proved ineffective,[23] but in one published and two unpublished studies, subcutaneous epinephrine (adrenaline) (0.1%, adult dose 0.25 mg) reduced the incidence of early antivenom reactions.[75] A study purporting to demonstrate the efficacy of combined hydrocortisone and antihistamine infusion was underpowered.[29]

Administration of Antivenom

Antivenom should be given as soon as it is indicated, but it remains potentially effective as long as signs of systemic envenoming persist (e.g., up to 2 days after a sea snakebite and many days or even weeks for prolonged defibrination following bites by Viperidae).[82,83,153,158] In contrast, local effects of venoms are probably not reversible by antivenom delayed for more than 1 to 2 hours after the bite.[118,135,155,161]

Route of Administration

The intravenous route is most effective. Although intramuscular injection is associated with a lower rate of early anaphylactoid reactions, absorption is very slow, especially when the gluteal site is used (but see later text). An infusion over 30 to 60 minutes of antivenom diluted in isotonic fluid may be easier to control than an intravenous "push" injection of reconstituted but undiluted antivenom given over 10 to 20 minutes. There is no difference in the incidence of severity of antivenom reactions in patients treated by these two methods.[61]

Snakebite in Remote (Wilderness) Locations

If someone is bitten by a snake in a remote location and signs of envenoming develop, but there is no one in the group capable of giving an intravenous injection, antivenom may be given by deep intramuscular injection at multiple sites into the anterolateral aspect of the thighs (not into the gluteal region from which absorption is exceptionally slow) followed by massage to promote absorption. An algorithm has been developed to guide the use of antivenom in these circumstances (Fig. 49-56). However, the volumes of antivenom normally required would make this route impracticable, as would the risk of hematoma formation in patients with incoagulable blood.

Dose of Antivenom

Ideally, the initial dose of a particular antivenom should be based on results of clinical studies when available, depending upon which species of snake was responsible and the severity of envenoming. However, most manufacturers' recommendations are based on mouse assays, which may not correlate with clinical findings.[165] Clinical trials of antivenom have been carried out since 1973,[154] but are inappropriately neglected even though they are the only reliable means of obtaining efficacy and safety data. Initial doses of some important antivenoms are given in Table 49-4. The apparent serum half-lives of antivenoms in envenomed patients range from 26 to 95 hours, depending on which IgG fragment they contain.[37,109] Children must be given the same doses of antivenom as adults.

Recurrent Envenoming

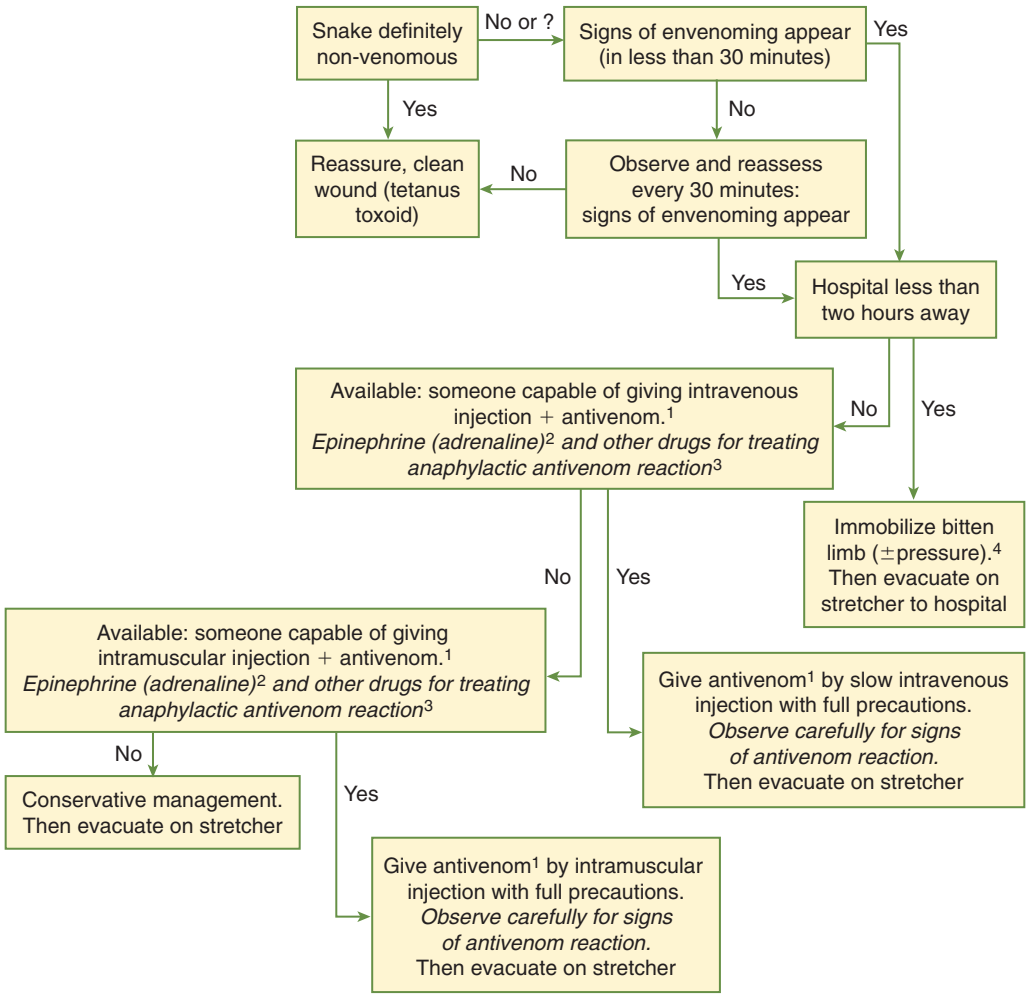
Recurrence of clinical and laboratory features of systemic envenoming, including recurrent venom antigenemia, several days after an initially good response to antivenom, was clearly documented in patients envenomed by Malayan pit vipers *(C. rhodostoma)* in Thailand in the 1980s, although this work was overlooked when the phenomenon was rediscovered after the introduction of CroFAb in the United States.[38,139,158] Recurrent envenoming is probably the result of continuing absorption of venom from the injection site after antivenom has been largely cleared from the circulation or perhaps by redistribution of venom from tissue in response to antivenom.[3,39] Paradoxically, venom absorption may increase after a hypotensive, shocked patient has been resuscitated. It is more likely to occur when a rapidly eliminated small IgG fragment antivenom, such as Fab, is used.[1,66] This suggests that an initial dose of antivenom, however large, may not prevent late or recurrent envenoming.

Repeated Dosing

The response to antivenom will determine whether further doses should be given. Neurotoxic signs may improve within 30 minutes of antivenom treatment, but this usually takes several hours. Hypotension, sinus bradycardia, and spontaneous systemic bleeding may respond within 10 to 20 minutes and blood coagulability is usually restored between 1 and 6 hours, provided sufficient antivenom has been given. A second dose of antivenom should be given if severe cardiorespiratory symptoms persist for more than about 30 minutes and when incoagulable blood persists for more than 6 hours after the start of the first dose.

The "6-Hour Rule"

Studies of envenoming by several species of snakes whose venoms cause coagulopathy have demonstrated that once an adequate neutralizing dose of antivenom has been given, blood coagulability (assessed by the 20-minute whole blood clotting test) will be restored within a median of 6 hours.[41,53,55,70,135,153,154,158,163] This reflects the ability of the liver,



Notes:
1 "Antivenom" means appropriate specific antivenom for the species of snake involved.
2 "Epinephrine (adrenaline)" means 0.1% (1:1000) adrenaline for intramuscular injection (adult dose 0.3–0.5mL).
3 "Other drugs for treating anaphylactic antivenom reaction" means antihistamine and hydrocortisone for intravenous injection.
4 "Immobilize bitten limb (±pressure)" means immobilization of the bitten limb with a splint or sling. "Pressure" means use of the pressure immobilization method using a long crepe bandage. This should only be used in the case of neurotoxic elapid snakes, *not* for viper bites.

Figure 49-56. Algorithm indicating appropriate management for snakebite in a patient while in a remote wilderness location. (From Warrell D. In Warrell D, Anderson S [eds]. New York: Fitzroy Dearborn, 2003, pp. 223–237.)

highly activated by circulating fibrin(ogen) breakdown products, to restore coagulable levels of clotting factors in patients with consumption coagulopathy. This important observation is the basis for a simple method of titrating antivenom dosage in individual patients whose blood is initially incoagulable. The 20-minute whole blood clotting test is performed at 6-hour intervals, and the initial dose of antivenom is repeated every 6 hours until blood coagulability is restored. After that, the 20-minute whole blood clotting test is checked at 12-hour intervals for at least 48 hours to detect recurrent envenoming.

Very large doses of antivenom may be required to treat patients bitten by species capable of injecting enormous amounts of venom or extremely potent venom. A patient bitten by the king cobra *(O. hannah)* was given 1150 mL of specific antivenom and prolonged artificial ventilation and survived.[119]

Antivenom Reactions

Antivenom treatment may be complicated by early (anaphylactoid), pyrogenic, or late (serum sickness–type) reactions.

Early Antivenom Reactions

These are not usually type I IgE-mediated hypersensitivity reactions to equine serum proteins and are therefore not predicted by hypersensitivity tests.[16,61] For this reason, it is inappropriate and misleading to apply the adjectives "allergic" or "hypersensitivity" to early antivenom reactions in general. Antivenoms activate complement in vitro,[102] while the clinically similar reactions to homologous serum are associated with complement activation and immune complex formation in vivo. The complement system is probably activated by aggregates of IgG or its fragments. Reactions usually develop within 10 to 180 minutes of starting antivenom. There is itching, urticaria, fever, tachycardia, palpitations, cough, nausea, and vomiting. The reported incidence, which varies from 3% to 84%,* appears to increase with the dose of antivenom and to decrease when manufacturing processes are improved[115,173] and administration is by intramuscular rather than intravenous injection. Unless patients are watched carefully for 3 hours after treatment, mild reactions may be missed and deaths misattributed to the envenoming itself. Up to 40% of patients with early reactions show features of severe systemic anaphylaxis: bronchospasm, hypotension, or angioedema—but deaths are rare. Early reactions respond readily to aqueous epinephrine given by intramuscular injection of between 0.5 and 1.0 mL of 0.1% (1 : 1000, 1 mg/mL) in adults (children, 0.01 mL/kg) at the first sign of trouble. Antihistamines such as chlorpheniramine maleate (adult dose 10 mg, children 0.2 mg/kg) should be given by intravenous injection to combat the effects of histamine released during the reaction.

Pyrogenic reactions result from contamination of the antivenom by endotoxin-like compounds. High temperature develops 1 to 2 hours after treatment and is associated with rigors, followed by vasodilatation and a decrease in blood pressure. Febrile convulsions may be precipitated in children. Patients should be physically cooled and given antipyretic drugs such as acetaminophen, by mouth, powdered and washed down a nasogastric tube (15 mg/kg), or by suppository. Aspirin and nonsteroidal anti-inflammatory agents are not safe in patients with hemostatic problems.

Late (serum sickness–type) reactions develop 5 to 24 (mean 7) days after treatment. Their incidence and speed of development increase with the dose of antivenom. Symptoms include fever, itching, urticaria, arthralgias (which may involve the temporomandibular joint), lymphadenopathy, periarticular swellings, mononeuritis multiplex, albuminuria, and (rarely) encephalopathy. This immune complex disorder responds to an antihistamine such as chlorpheniramine (adults, 2 mg four times a day; children, 0.25 mg/kg per day in divided doses), or, in more severe cases, to corticosteroids (prednisolone 5 mg four times a day for 5 days in adults; 0.7 mg/kg per day in divided doses for 5 days for children).

Supportive Treatment (Assuming That Adequate Doses of Antivenom Have Been Given)

Neurotoxic Envenoming

Artificial Ventilation. This was first suggested for neurotoxic envenoming more than 130 years ago, but patients continue to die for lack of vital respiratory support.

*References 53, 68, 97, 109, 115, 122, 146, 154, 158, 161, 165, 166.

Bulbar and Respiratory Muscle Paralysis. Inability to swallow is indicated by accumulation of secretions in the pharynx. Stridor indicates paralysis of the vocal cords; it can be accentuated by extending the neck. Early paralysis of intercostal respiratory muscles is suggested by "abdominal breathing" (or "paradoxical respiration") in which exaggerated compensatory contraction of the diaphragm causes the paralyzed abdomen (rather than the chest) to expand on inspiration. Objective measurement of ventilatory capacity is very useful to monitor respiratory paralysis. This can be achieved with a peak flow meter, spirometer, respirometer (if the patient is using a face mask or is intubated) or by asking the patient to blow into the tube of a sphygmomanometer to record the maximum expiratory pressure. If patients are adequately oxygenated, even those with profound generalized flaccid paralysis from neurotoxic envenoming are fully conscious. However, because their eyes are closed (ptosis) and they do not move or speak, they are commonly assumed to be unconscious. Tactless remarks made by medical staff can be heard by the apparently comatose patient! These patients may still be able to flex a finger or toe, allowing simple communication. It is very important not to assume that patients with snakebite neurotoxicity have irreversible brain damage, although they may be areflexic, unresponsive to painful stimuli, and have fixed, dilated pupils.

Neurotoxic effects are fully reversible with time. A patient bitten by *B. multicinctus* in Canton recovered completely after being ventilated manually for 30 days, and a patient probably envenomed by *T. carinatus* recovered after 10 weeks of mechanical ventilation in Queensland, Australia. Endotracheal intubation or tracheostomy using a cuffed tube is needed. The patient can be ventilated manually with an anesthesia bag or, preferably, with a mechanical ventilator.

Anticholinesterase Drugs

These may produce rapid, useful improvement in neuromuscular transmission in patients envenomed by some species of Asian and African cobras, mambas, death adders (*Acanthophis* species), and kraits.[52,159,166,170] It is worth trying the "Tensilon test" in all cases of severe neurotoxic envenoming, as for a patient with suspected myasthenia gravis. Atropine sulfate (adults, 0.6 mg; children, 50 µg/kg) must be given first by intravenous injection to block unpleasant muscarinic effects (increased secretions, abdominal colic) of acetylcholine. Edrophonium chloride (Tensilon), or any other available anticholinesterase, is then given by slow intravenous injection in an adult dose of 10 mg, or 0.25 mg/kg for children. Patients who respond convincingly can be maintained on neostigmine methylsulfate, 50 to 100 µg/kg, and atropine every 4 hours or by continuous infusion. Patients must be observed closely for symptoms of cholinergic crisis.

Hypotension and Shock

Hypovolemia is a common cause of hypotension and shock and should be treated by infusing a plasma expander. Central venous pressure is the safest way to control volume replacement. Hypotensive patients envenomed by Burmese Russell's viper responded to dopamine, 2.5 µg/kg per minute by intravenous infusion; however, methylprednisolone, 30 mg/kg, and naloxone were not effective.[70]

Renal Failure[96,148]

If urine output drops below 400 mL/24 hours, urethral and central venous catheters should be inserted. If urine flow fails to increase after cautious rehydration, diuretics should be tried (e.g., furosemide by slow intravenous injection, 100 mg followed by 200 mg if urine output fails to increase, and then mannitol). If these measures fail to restore urine flow, the patient should be placed on strict fluid balance. Peritoneal or hemodialysis will be required in most patients with established renal failure.

Local Infection

A booster dose of tetanus toxoid should be given. As in the case of Latin American pit vipers, local bacterial infections with formation of wound abscesses are common after bites by some species such as Malayan pit vipers *(C. rhodostoma)*.[111] Even a case of Buruli ulcer *(Mycobacterium ulcerans)* has been attributed to snakebite. Prophylactic antibiotics are not justified unless the wound has been incised or there is evidence of necrosis.[43] Penicillin, erythromycin, or chloramphenicol is appropriate, as would be an antibiotic effective against the bacterial flora of the buccal cavity and venoms of local snakes.[111] An aminoglycoside such as gentamicin should be added for 48 hours. Bullae are best left intact.

Nursing Snake-Bitten Limbs

Excessive elevation of snake-bitten limbs should be avoided in that this has been shown to increase the risk of compartmental syndromes.[63]

Surgical Management of Snake-Bitten Limbs[164]

Debridement of Necrotic Tissue

Necrotic tissue should be debrided as soon as possible and the denuded area covered with split-thickness skin grafts (see Figure 49-42). Muscle should not be excised just because it looks dark or even black, because intense hemorrhage may produce this appearance in viable muscle.

Intracompartmental Syndromes[63]

In a snake-bitten limb, swelling of muscles within tight fascial compartments, such as the anterior tibial compartment, may raise the tissue pressure to such an extent that perfusion is impaired and ischemic damage (as in Volkmann's contracture of the forearm) is added to the effects of the venom. Risk factors include pressure bandaging and excessive elevation of the limb that reduce arterial perfusion pressure in the compartment but cannot reduce local venous pressure below local tissue pressure. Reduction of the arteriovenous pressure gradient was associated with decreased muscle Po_2 and nerve conduction velocity.[63] The classic signs of an intracompartmental syndrome include excessive pain, weakness of the compartmental muscles and pain when they are passively stretched, hypoesthesia of areas of skin supplied by nerves running through the compartment, and obvious tenseness of the compartment.[63] These features have been characterized as "the 7 Ps": pain at rest, pain on possible movement, paralysis, pallor, paresthesia, poikilothermia, and pulselessness. Unfortunately, local effects of envenoming often result in a painful, immobile, pale, cyanotic, cold, tensely swollen, and apparently pulseless limb with poor capillary refill in the absence of any demonstrable compartmental syndrome. These appearances may mislead inexperienced surgeons into diagnosing compartment syndrome without objective evidence and encourage them to proceed to fasciotomy. The discovery of dark or even black-looking muscle fasciculi may reassure the surgeon that the operation was necessary, but envenomed yet viable muscle often looks black because of hemorrhage. Dangers of fasciotomy include neglect of early adequate antivenom treatment, severe persistent bleeding if the venom-induced hemostatic abnormalities have not been corrected by adequate doses of antivenom, delayed recovery of function, prolonged hospital admission, persistent morbidity from damage to sensory nerves, and contractures from keloid formation or hypertrophic scarring in some ethnic groups. Palpation of peripheral pulses or their detection by Doppler ultrasound does not exclude intracompartmental ischemia. However, direct measurement of intracompartmental pressure is reasonably simple, using a perfusion pump and saline manometer system or a commercial transducer such as the Stryker apparatus. An intracompartmental pressure of more than 45 mm Hg in an adult or 30 mm Hg in a child indicates a high risk for ischemic necrosis (see Figure 49-38). In these circumstances, fasciotomy may be justified to relieve the pressure in the compartment, but this treatment did not prove effective in saving envenomed muscle in animal experiments.[28] Necrosis occurs most frequently after digital bites. Fasciotomy must never be embarked upon until blood coagulability has been restored by adequate doses of specific antivenom, followed by the transfusion of fresh whole blood, clotting factors, or platelets as required.

Inappropriate Treatments

Corticosteroids, heparin, antifibrinolytic agents such as aprotinin (Trasylol) and ε-aminocaproic acid, antihistamines, trypsin, and a variety of traditional herbal remedies have been used and advocated for snakebite. Most are potentially harmful and none has been proved to be effective.

Treatment of Snake Venom Ophthalmia

The "spat" venom should be washed from the eye or mucous membrane as soon as possible using large volumes of water or other bland fluid. Unless a corneal abrasion can be excluded by fluorescein staining or slit-lamp examination, a topical antimicrobial agent such as tetracycline or chloramphenicol should be applied, as with any other corneal injury.[149,160] Epinephrine eyedrops (0.1%) relieve the pain.

PREVENTION OF SNAKEBITE

The risk of snakebite can be reduced by simple precautions. Snakes should never be disturbed, attacked, or handled unnecessarily even if they are thought to be harmless species or appear to be dead. Venomous species should never be kept as pets or as performing animals. Protective clothing—boots (not open sandals), socks, long trousers—should be worn when walking in undergrowth or deep sand and a flashlight should always be carried at night. Particular care should be taken while collecting firewood; moving logs, boulders, boxes, or debris likely to conceal a snake; climbing rocks and trees covered with dense foliage; or swimming in overgrown lakes and rivers. Where there are sea snakes, wading in the sea, especially in sand or near coral reefs, is a dangerous pastime and should be avoided

if possible. Shuffling is safer than a high stepping gait. Divers should keep clear of sea snakes. Fishermen who catch sea snakes in nets or on lines should return them to their element without touching them.

In the camp, sleep off the ground on a camp bed or in a tent with a sewn-in ground sheet. Remove unnecessary junk and litter that might attract rodents, because rodents attract snakes. Various toxic chemicals such as naphthalene, sulphur, insecticides (e.g., DDT, dieldrin, and pyrethrins), and fumigants (e.g., methyl bromide, formaldehyde, or tetrachloroethane) are lethal to snakes, but no true repellents have been identified.

Prophylactic immunization against snakebite using venom toxoids has been attempted, for example, against habu *(T. flavoviridis)* among farmers in the Amami and Ryuku Islands of southern Japan, where it proved ineffective.[89]

The analogy with immunization against infectious diseases is misleading, because the immunized person would not have time to develop an enhanced secondary (anamnestic) response to the venom after being bitten. Protection would depend solely on levels of neutralizing antibody circulating at the time of the bite.

USEFUL WEBSITES

Snakebite Management

South and southeast Asia: http://w3.whosea.org/bct/snake/
Global (especially Australasia): www.toxinology.com/

Antivenoms

Munich AntiVenomINdex (MAVIN): http://toxinfo.org/antivenoms/
CSL Australian antivenoms: www.toxinology.com/generic_static_files/cslavh_antivenom.html
Antivenom index: www.aza.org/AI/ (for poisons centers' prescribers)
http://globalcrisis.info/latestantivenom.htm
WHO: www.who.int/bloodproducts/animal_sera/en/

Venomous Snake Taxonomy Updates

http://sbsweb.bangor.ac.uk/%7Ebss166/update.htm

The references for this chapter can be found on the accompanying DVD-ROM.

Antivenoms and Immunobiologicals: Immunotherapeutics of Envenomation

50

Angela F. Gardner and Richard C. Dart

The ideal therapeutic agent effectively treats or prevents a specific medical condition and is free of adverse effects. Commercially available antivenoms (antivenins) meet the condition of specificity but are not free of adverse effects. The vast majority of antivenoms are equine derived and thus possess the potential adverse immunologic effects inherent in the administration of foreign proteins. Although antivenom lowers the morbidity and mortality associated with envenomation, it may have life-threatening side effects. As a result, many physicians are reluctant to administer antivenom even to patients who have a clear indication for its use. Additionally, the production of antivenoms has changed rapidly in the past 10 years, with resulting shortages and inequality of distribution of existing antivenoms.

Worldwide, commercial production of antivenom preparations for the treatment of snake, arachnid, fish, and coelenterate envenomations declined dramatically over the past decade.[28] In affluent nations, economic pressures have forced some manufacturers out of the antivenom production market. There has been a long-standing undersupply of antivenom for Africa and Asia.[43] In spite of these barriers, the growth of the internet and computer technology has made locating antivenom much easier. One source providing a list and links to international sources of antivenom is found at www.globalcrisis.info/latestantivenom.htm. Overall, there are more than 200 different formulations of antivenom available worldwide.

The available crotalid antivenom in the United States is effective against all indigenous North American pit vipers. However, zoos and amateur snake enthusiasts are likely to house non-indigenous, exotic snakes for which the anticrotalid antivenom is ineffective. Physicians must expeditiously exploit all available resources to identify and acquire the appropriate antivenom for unusual envenomations. One such resource, the *Anti-Venom*

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USEFUL WEBSITES

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Antivenoms

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CSL Australian antivenoms: www.toxinology.com/generic_static_files/cslavh_antivenom.html
Antivenom index: www.aza.org/AI/ (for poisons centers' prescribers)
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Index, is compiled by the American Zoo and Aquarium Association (www.aza.org) and the American Association of Poison Control Centers (AAPCC). The Index lists the location of foreign antivenoms stocked by zoos in the United States. Guidance for use of these antivenoms and general snakebite management may be obtained through an AAPCC-certified poison center.

HISTORICAL PERSPECTIVE

Although the French physician and bacteriologist Calmette is often credited with producing the first therapeutic antivenom in 1896,[9] scholars of ancient Greece believe that King Mithridates VI of Pontus (132 BC to 63 BC)[22] deserves recognition as the first recorded user of immunotherapeutic principles. The legendary King of Pontus in Asia Minor was known for his prodigious memory and for his paranoia. To protect himself from poisoning, he took sublethal doses of various venoms until he was able to tolerate lethal doses. Ironically, when Pompey the Great defeated Mithridates in battle and capture by Rome was imminent, Mithridates attempted suicide by poison. He was unsuccessful, and instead had to fall on his sword. The tale has spawned literature (Borges), poetry (A. E. Houseman), theater (Racine), and opera (Mozart). Even now, a universal antidote is known as a *mithridate.*

Two thousand years later, in another part of Asia, Albert Calmette produced the first snake antisera against the Indian cobra, *Naja kaouthia.*[8] In 1895, while working at the Institute de Pasteur in what is now Vietnam, he immunized horses with cobra venom and then used the equine antivenom to save a human victim of cobra bite. Although Dr. Calmette mistakenly believed that the equine serum was protective against all neurotoxic venoms, he successfully established a principle of immunotherapy, interspecies cross-reactivity, that is still operative.

Dr. Vital Brazil, another pioneer in the study and production of antivenom, demonstrated both cross-reactivity and specificity while developing the first monospecific and polyspecific antivenoms for the *Crotalus, Bothrops,* and *Elaps* genera, as well as for certain species of spiders, scorpions, and batrachia.[7] Monospecific antivenom is produced by immunizing animals with the venom of a single species. Polyspecific antivenom is produced either by inoculating animals with the venom of different species or by pooling different monospecific antivenoms to produce a final product. Brazil recognized the need for a polyspecific antivenom, because snakebite victims were often unable to differentiate between the poisonous snakes endemic to the area.

His work at the Institute Butantan in Sao Paolo, Brazil, from 1901 to 1914 established firmly that there is no natural immunity to animal venoms in humans, although there may be differences in their physiopathologic responses. Using the process of mithridatization—the administration of small and graded doses of venom—Dr. Brazil produced an acquired immunity in humans.

Dr. Brazil devised an interesting method for ensuring an adequate supply of venom for his research. He convinced the Brazilian government and railroad officials to provide free passage for snakes shipped to his institute. Anyone requesting participation in his program received free devices for snake capture, crates for transport, and a vial of antivenom for every snake shipped to him. For every six snakes sent, he provided a syringe and needle for administering the accompanying serum.[36] After a decade of operation, his exchange program produced a significant decrease in mortality from snakebites.[7,36]

The halcyon days of passive antibody therapy were the 1920s and 1930s. At that time, passive antibody therapy, known as serum therapy because the preparations were derived from horse or rabbit serum, was routinely used for pneumococcal pneumonia, diphtheria, measles, and tuberculosis. Although many of the sera, especially the pneumococcal serum, were clinically effective, they held the high risks of immediate and delayed hypersensitivity reactions. Other problems included uncertain dosing, lot-to-lot variability, high cost, and the need for intravenous (IV) administration.[10] The discovery of sulfonamide in 1935 and penicillin in 1942 led to discontinuation of serum therapy for bacterial infections.

Serum-based therapies continue today in areas where no known drugs are available. Toxin-mediated conditions such as tetanus and botulism are treated with antibody preparations. Passive immunity is conveyed by serum therapy for conditions such as rabies and hepatitis. A preparation known as *Fab* was developed with a high specificity for digitoxin, and it is used in digitalis poisoning. Antivenom remains the front line therapy for envenomations.

PRINCIPLES OF ANTIVENOM THERAPY

Neutralizing Antibody—The Immunology of Envenomation

The core of antivenom therapy is the antigen–antibody interaction. Antibodies are Y-shaped proteins generated by B lymphocytes. These proteins are present on lymphocyte surfaces and are secreted into the blood or lymph systems in response to an antigenic stimulus. Antibodies have physiologic properties that include toxin neutralization, microbial opsonization, complement activation, and antibody-directed cellular cytotoxicity (ADCC). The term *neutralizing antibody* refers to a form of antibody that reacts with an antigen via a specific, noncovalent bond, thus attenuating the effects of the antigen. For example, a neutralizing antibody to phospholipase A, a common antivenom constituent, would bind with the phospholipase A antigen and diminish the myotoxic or neurotoxic effects associated with this venom component.

Antibody molecules are immunoglobulins that have a specific amino acid sequence and tertiary structure intended to bind with a complementary structure on the antigen molecule. Although antibodies are heterogeneous, those in each immunoglobulin class have similar structures. Each molecule has four polypeptide chains, two identical heavy chains and two identical light chains. The heavy chains each have molecular weights between 50,000 and 70,000 daltons (Da). The light chains have molecular weights of about 23,000 Da. The chains are joined by disulfide bridges, forming the familiar Y configuration.

The immunoglobulin G (IgG) molecule is responsible for the therapeutic activity of antivenom. It has two functional parts, the antigen-binding site and the effector site. The antigen-binding site consists of two Fab portions. The effector site is the Fc portion. The antigen-binding site is responsible for neutralizing further activity of the antigen. The effector site is responsible for binding and activating immune system cells and triggering a host of immunologic responses. These responses, including degranulation of mast cells and activation of the

complement cascade, are responsible for the immediate, life-threatening allergic reactions to antivenom.

The large size of the IgG molecule has implications for the immunogenicity, distribution, and elimination of these neutralizing antibodies. Large–molecular-weight entities such as IgG and other antivenom components are more likely than smaller entities to trigger immunologic events. The large size of the IgG molecule confines it primarily to the intravascular compartment, and limits more extensive distribution into tissue spaces. Theoretically, this may limit the effectiveness of IgG in binding antigens in the tissue compartments.

The large molecules that make up most antivenom preparations have long distribution half-lives and may predispose recipients to further immunologic complications. Large antivenom proteins exceed the threshold for filtration and elimination by the kidney, and they are eliminated by the reticuloendothelial system (RES). Elimination by the RES is a much longer process than renal elimination, allowing the foreign proteins a much longer time to be exposed to the patient's endogenous antibodies. Immune complexes formed by the interaction of foreign (antivenom) proteins and endogenous (patient) antibodies may precipitate in susceptible tissue, resulting in the clinical disease of serum sickness.

Enzymatic cleavage of IgG with papain or pepsin produces smaller substituent fragments that are capable of neutralizing venom components. Snake antivenom composed of these fragments avoids many of the serious side effects of IgG antivenom, representing a significant advance in the treatment of envenomation.

Fab Fragments and the Treatment of Foxglove (Digitalis) Poisoning

Sheep-derived IgG antibodies to digoxin have been used successfully for years to treat patients with cardiac glycoside toxicity. Infusion of commercial polyclonal antidigoxin Fab has been used to treat plant poisonings, Asian herbal medicine or aphrodisiac overdose, and toad envenomation.[13] Common plants containing digitalis and digitalis-related compounds include *Digitalis lanata* (foxglove), *Digitalis purpurea* (foxglove), *Nerium oleander* (common oleander), *Thevetia peruviana* (yellow oleander), *Convallaria jamalis* (lily of the valley), *Urginea maritime* and *Urginea indica* (squill), and *Strophanthus gratus* (oubain). Toxicity from these plants was well recognized by 1785, when William Withering published his classic work describing the therapeutic uses and toxicity of foxglove. Toxicity may occur after consuming teas brewed from plant parts or after consuming leaves, flowers, or seeds from plants containing cardiac glycosides.

Toxic exposure to plants containing cardiac glycosides is rare in the United States. Of 2.24 million exposures to toxic substances reported by the AAPCC in 1998, only 2553 resulted from exposure to plants containing cardiac glycosides. Internationally, exposure to cardiac glycosides of plant origin is higher in countries and communities that rely heavily on folk or herbal medicines containing cardiac glycosides.[63,64] Sri Lanka reported a recent outbreak of yellow oleander (*T. peruviana*) poisonings. Its seeds are a popular suicidal ingestion in Sri Lanka, with thousands of cases reported each year.[29]

Mortality is rare, but case reports documenting fatalities from foxglove, squill, oleander, and other related plants do exist. In 1988, the AAPCC reported one death and 2553 exposures to cardiac glycoside–containing plants. The AAPCC noted major morbidity or death in only 0.1% of cardiac glycoside–containing plant exposures in 1998. Moderate morbidity occurred in only 0.9% of exposures; mild morbidity occurred in 5.4%. Most plant exposures occur in children less than 6 years old and are generally accidental and without significant toxicity.

Digoxin Fab fragments are indicated for cardiac glycoside intoxication in several situations, including hyperkalemia (>5.5) unresponsive to standard treatments, life-threatening supraventricular and ventricular dysrhythmias not quickly responsive to standard treatments, and cardiac arrest. Because serum digoxin levels do not accurately reflect the ingested amount of plant cardiac glycoside, drug levels should not be used to calculate Fab dose.[72] Elevated levels of digoxin only confirm exposure. Undetectable levels of serum cardiac glycosides do not rule out exposure. Elevated serum potassium would be a useful indicator when considering this agent.

The initial dose of Fab, 5 to 10 vials IV, is empiric. Larger doses may be needed.[27] The pediatric dose is the same as the adult dose. In the case of cardiac arrest, give 10 to 20 vials of Fab and continue to treat with standard ACLS (Advanced Cardiac Life Support) protocols. Because the onset of action of Fab fragments takes 30 to 60 minutes, prolonged efforts at resuscitation may be warranted. Consultation with a toxicologist available through a poison control center is strongly recommended if the use of Fab fragments is being considered or if the signs and symptoms of toxicity are severe.

Antivenom and Immunotherapy

After Dr. Calmette demonstrated species cross-reactivity, scientists labored to produce a "mithridate" for venoms. The therapeutic activity of antivenom is based on specific, noncovalent antigen–antibody binding. Depending on the type of neutralizing molecule, antivenoms are made of (1) whole IgG molecules, (2) $F(ab')_2$ fragments or (3) Fab fragments. The efficacy, propensity for adverse reaction, and pharmacokinetic profile are related to the structure of the neutralizing molecule of the antivenom.

The whole IgG molecule weighs approximately 150,000 Da and consists of two parts—the antigen-binding site and the effector site. The antigen-binding site consists of two Fab portions, and the effector site is the Fc region. Specific binding of the antigen to the Fab sites neutralizes further activity of the antigen. The Fc region is responsible for activating immune system cells, triggering a host of immunologic responses (Fig. 50-1). The Fab portion is found at the amino-terminal end of the IgG molecule and the Fc portion is found at the carboxyl end of the molecule. A disulfide bridge joins the parts of the molecule. Proteolytic cleavage of the IgG molecule results in the formation of predictable substituent fractions. Pepsin digestion results in an Fc portion and an $F(ab')_2$ portion (Fig. 50-2). $F(ab')_2$ consists of two Fab portions connected by a sulfide bridge, and it weighs 100,000 Da. Proteolytic cleavage with papain yields an Fc portion and two separate Fab segments per molecule of IgG (Fig. 50-3). The molecular weight of Fab is 50,000 Da.

Goals of Antivenom Therapy

The ideal antivenom would have a high affinity for toxicologically relevant venom components and a volume of distribution similar to that of the toxin being neutralized. In addition, high levels of neutralizing antibodies should remain in the blood for

a relatively prolonged time, ensuring neutralization of toxins that reach the bloodstream slowly after the initial envenomation. Finally, the ideal antivenom would promote redistribution of toxins from extravascular components to blood.

This "ideal" antivenom provides a context in which to consider the pharmacokinetics and pharmacodynamics of the IgG, $F(ab')_2$, and Fab-based antivenoms. The large size of the IgG antibody has important implications for the immunogenicity, distribution, and elimination of the neutralizing antibodies. Large-molecular-weight entities in general (IgG and other large antivenom components) are more likely than smaller ones to trigger immunologic reactions.[34] The IgG molecule has a relatively small volume of distribution, which confines it primarily to the extravascular space and may limit extensive distribution into tissue compartments. This may be of some advantage in countering the high-molecular-mass toxins of Viperidae venoms, which exhibit rapid initial absorption followed by a slow absorption from the envenomation site. Large antivenom proteins exceed the limit of filtration of the glomerular system and therefore are primarily eliminated through the RES, a much slower process. Unfortunately, the long elimination half-life of relatively large antivenom proteins creates a perfect environment for interaction with endogenous human antibodies, creating an immune complex. Precipitation of such immune complexes results in the clinical condition known as serum sickness.

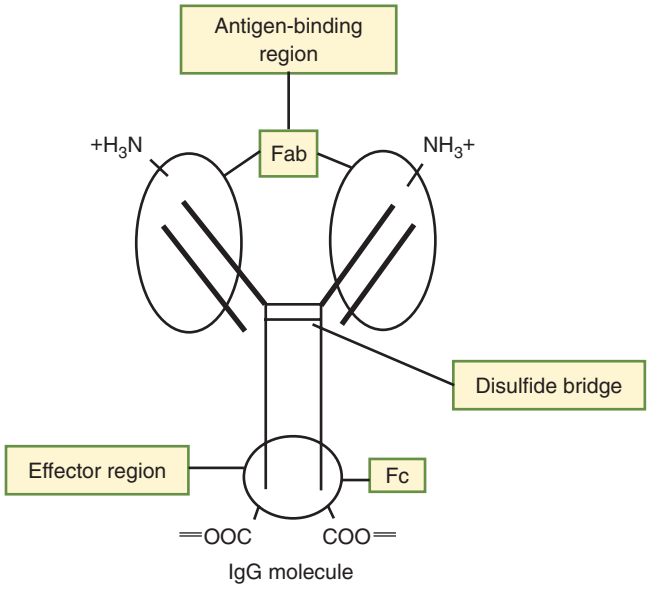

Figure 50-1. Schematic diagram of an IgG molecule. The antigen-binding region (Fab portion) is present at the amino-terminal end. The effector region (Fc portion) interacts with the immune system cells and resides at the carboxyl end of IgG. Note the disulfide bridge.

Cleavage of the Fc fragment using pepsin produces an $F(ab')_2$ portion that has less allergenicity than the IgG molecule. Because they are slightly smaller, the $F(ab')_2$ antivenoms, which are not currently in use in the United States, are cleared by the kidney. The volume of distribution is only slightly greater than that of the IgG molecule, and the half-life is slightly less. The $F(ab')_2$ antivenom produced in Mexico, known as Antivipmyn, has been shown to have about 78% efficacy against 15 North American vipers.[61]

Cleavage of the Fc fragment using papain produces two Fab portions. Removal of the Fc portion decreases antigenicity of the fragments and lowers the weight of each fragment to about 50,000 Da. Fab fragments have the largest volume of distribution of the three immunoglobulins, and they readily enter the extravascular compartments. They are eliminated easily through renal mechanisms and therefore have a more rapid clearance than do either $F(ab')_2$ fragments or IgG molecules. This may lead to the phenomenon of *recurrence*. Because the Fab fragments clear quickly and the venom of Viperidae is absorbed slowly from the site, symptoms may recur after the initial dose(s) of antivenom (see later discussion).

MODERN ANTIVENOM THERAPY

Maximizing the neutralizing potential of antivenom while minimizing its immunogenicity has become the principal goal

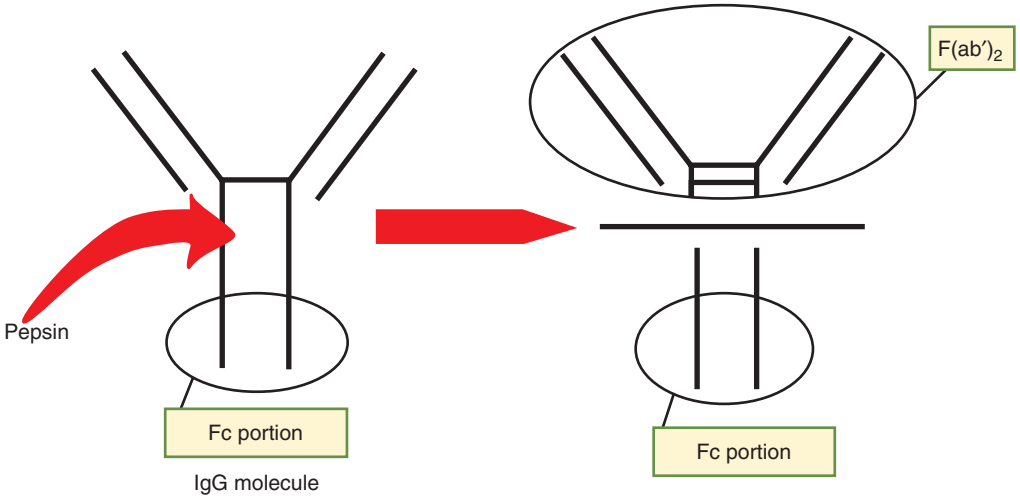

Figure 50-2. Proteolytic cleavage of IgG results in predictable substituent fractions. Pepsin digestion produces Fc and $F(ab')_2$ fractions for each IgG molecule. $F(ab')_2$ consists of two Fab portions connected by a disulfide bridge. The molecular weight of $F(ab')_2$ is 100,000 daltons.

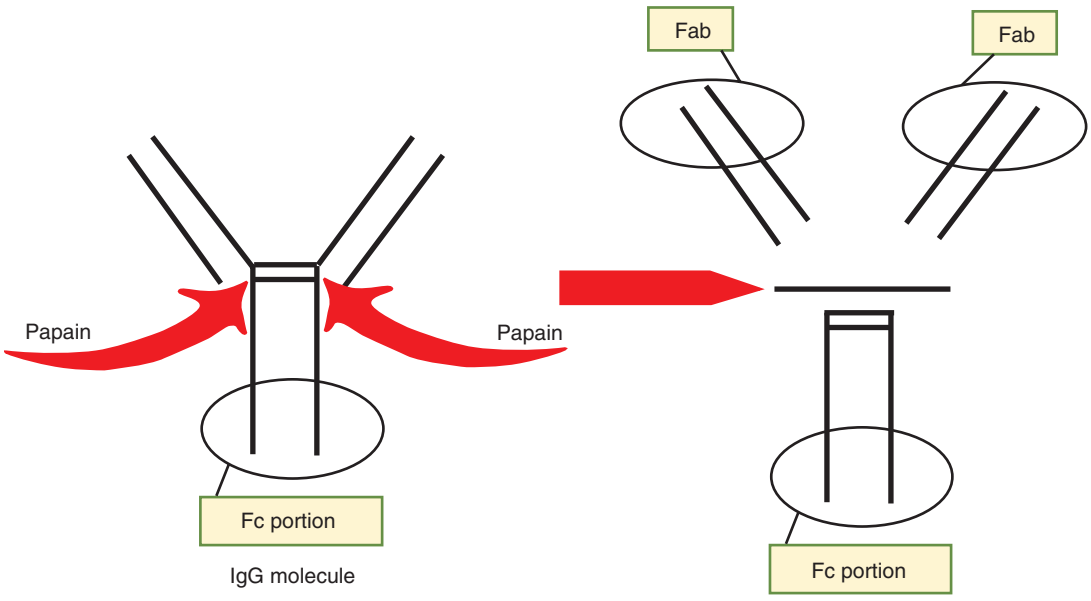

Figure 50-3. Proteolytic cleavage of IgG results in predictable substituent fractions. Papain digestion yields the Fc and two Fab segments for each IgG molecule. The molecular weight of Fab is 50,000 daltons.

of modern antivenom production. Despite major advances in molecular pharmacobiology, the commercial manufacture of antivenom relies on the same principles of host animal immunization with snake venom described by Calmette more than a century ago.

Production

All antivenoms follow a two-step production process. First, a neutralizing antibody is produced by immunizing a host animal with sublethal doses of venom. This antibody is one of many proteins present in the resulting immune serum preparation. Step two involves purification of antivenom with reduction in the fraction of nonneutralizing proteins. The preparation is then concentrated and packaged in its final lyophilized or liquid form. Two commercial antivenom preparations for North American pit viper envenomations are available in the United States.

Crotalidae Polyvalent Immune Fab (Ovine)/Cro-Fab (Savage Laboratories, Melville, NY). Fab antivenom (FabAV) was approved by the U.S. Food and Drug Administration in October 2000 for use in crotaline snakebite. It is produced by immunizing sheep with increasing doses of venom from four crotaline species: *Crotalus adamanteus* (Eastern diamondback), *Crotalus atrox* (western diamondback), *Crotalus scutulatus* (Mojave), and *Agkistrodon piscivorus* (cottonmouth). The immune serum is harvested and digested with papain to produce an Fc portion and two Fab fragments (see previous discussion). The Fc portion is eliminated during sodium sulfate precipitation and affinity purification. The two remaining individual Fab fragments are combined to produce the FabAV. The resulting product is about five times more potent in animal studies against 14 different crotalid venoms than is the ACP product (described next).[16]

Wyeth Polyvalent Antivenom. Wyeth (Madison, NJ) has produced Antivenom (Crotalidae) Polyvalent (ACP) (equine) using the same basic process since 1954. This process utilizes horses, which are immunized with increasing doses of venom from four crotalid species: *C. adamanteus* (Eastern diamondback), *C. atrox* (western diamondback), *Crotalus durrissus terrificus* (South American or tropical rattlesnake), and *Bothrops atrox* (fer-de-lance), plus adjuvant for immune system stimulation. The resultant hyperimmune serum is subjected to ammonium sulfate precipitation, which removes a variety of plasma proteins. A fraction of neutralizing antibody is also lost in this process, resulting in diminished potency. The product is then precipitated, filtered, resuspended, and lyophilized. The final concentrated antivenom contains 1.5 to 2 g of horse protein, only 15% to 20% of which is IgG. Wyeth-ESI Lederle ceased production of its antivenom products in January 2001. It currently maintains a supply of ACP for emergency use by hospitals only.[78,79]

Efficacy

FabAV has been tested in two prospective, open-labeled, multicenter trials, using patients presenting within 6 hours of minimal to moderate crotalid envenomation.[6,18,20,21] The snakebite severity score (SSS) was used to document severity of envenomation, and all patients showed evidence of worsening symptoms in the emergency department prior to inclusion in the study. Rapid onset of action was seen in both trials during the initial infusion of FabAV and during the initial period of observation. Improvement was seen in the SSS components representing coagulation, and in the central nervous, gastrointestinal, and cardiovascular systems. In contrast, the components of the SSS measuring local injury (pain, swelling, ecchymosis) improved slightly, but the improvement was not statistically significant. This may be because the local processes of hemorrhage and cell death are not as quickly reversible as are the systemic processes.

An unexpected observation during the first clinical trials was recurrence after completion of the initial FabAV treatment. *Recurrence* is defined as the recrudescence of any venom effect after that abnormality had resolved. Two patients developed recurrent swelling 15 to 18 hours after envenomation, and one patient had a recurrent coagulopathy at the 1-week follow-up

visit. Several explanations for this phenomenon are hypothesized, mainly relating to the short half-life of Fab. To address this, a new dosing schedule was devised and tested. This second trial compared a single dose of FabAV to a loading dose (four to 12 vials) followed by three additional doses (two vials each) at 6, 12, and 18 hours after initial control of symptoms. Although this was the first, albeit unintended, prospective study of the recurrence phenomenon in the United States, similar findings have been described in worldwide and subsequent United States research.[58,71]

The efficacy of ACP can be estimated by noting the decline in mortality from crotalid envenomation over the past century. Mortality in the 19th century from venomous snakebite was 5% to 15%. Mortality in the 20th century was less than 0.5% in patients treated in a health care facility.[59] A recent animal study compared the effectiveness of ACP in preventing local effects of envenomation (myonecrosis) when given by two different routes. The results showed no effect by either route in limiting local myonecrosis after envenomation in the animal model.[48]

Indications

There are no rigorous definitions for the use of antivenom in the United States; however, progression of envenomation syndrome is generally considered the basis of treatment (see Chapter 48). This may include worsening of local effects such as pain, swelling, or ecchymosis; worsening of systemic effects such as hypotension or altered mental status; or worsening of coagulopathies.

FabAV is optimally used within the first 6 hours after envenomation. However, there are reports of use after 9 hours, and in one report, coagulopathy correction occurred as late as 52 hours after envenomation.[4]

ACP is most effective when given within the first 4 hours after envenomation, showing diminishing efficacy after 12 hours. However, there are reports of reversal of coagulopathies as late as 24 hours after envenomation.

Administration and Clinical Use

FabAV is administered to achieve initial control and then maintenance. Skin testing for reactivity to the serum components is not currently recommended by the manufacturer, nor is a need for it supported by the clinical trial experience. The initial dose of FabAV antivenom is customarily 4 to 6 vials, followed by another 4 to 6 vials if symptoms continue to progress. In most cases, 8 to 12 vials sufficiently provide control. There is one case noted in the literature that required 22 vials for initial treatment.[57] After control has been established, two additional vials are given at 6, 12, and 18 hours to prevent recurrence.

The entire dose of reconstituted FabAV should be diluted in crystalloid fluid to a total volume of 250 mL. This can be administered slowly over the first 10 minutes and the delivery completed over the course of an hour. To date, no cases of anaphylaxis have been reported with FabAV. The most severe reaction reported during the initial infusion was bronchospasm, which responded promptly to therapy.

Both antivenoms, FabAV and ACP, are lyophilized antivenoms and require reconstitution prior to administration. Once the decision to treat has been made, any time lost to preparation of antivenom risks worsening envenomation progression. Each antivenom kit provides 10 mL of diluent (bacteriostatic water for injection, USP) to add to the lyophilized antivenom. *Gentle* swirling of the vials assists in solubilization. The 1-hour delay needed to reconstitute ACP could be critical in a patient with rapidly progressing envenomation. FabAV requires only 15 to 30 minutes for reconstitution, as documented in the clinical trials.[20] Studies have been conducted to determine the stability of FabAV to heat and motion. Although the FabAV appears to be less potent after agitation, it remained effective in a mouse model.[23] The same study concluded that FabAV could be safely stored for 60 days without refrigeration under most field conditions where snake envenomation occurs.

Because ACP is equine derived, intended recipients should be skin tested for sensitivity prior to its administration, as recommended by the manufacturer. However, because skin testing is not reliable in predicting sensitivity,[41] careful monitoring of the patient during subsequent ACP administration is imperative. Pretreatment with histamine-1 (H_1) and histamine-2 (H_2) blockers should precede administration of ACP in patients who have a positive skin test, and in those who have life- or limb-threatening envenomations. This should be carried out in a critical care setting. Early anaphylactic reactions frequently result from overly rapid infusion of the antivenom. Management includes stopping the infusion, administering epinephrine, administering H_1 and H_2 blockers, support with isotonic fluids, and airway support if necessary. The ACP may be further diluted and resumed at a much slower rate with careful clinical monitoring.

In general, dosing of ACP is as follows:

Level of Envenomation	Initial ACP Dose (Number of Vials)
Minimal	None
Moderate	10 to 15
Severe	15 vials
Profound circulatory collapse	20

The progression of envenomation guides the administration of additional ACP doses. Worsening local, systemic, or coagulopathic abnormalities require repetition of the initial dose every 1 to 2 hours as needed.

Reconstituted ACP antivenom should be diluted in 250 to 1000 mL of normal saline or 5% dextrose in water. The IV solution should be administered at 50 to 75 mL/hr for the first 10 minutes. If no reaction occurs, the remainder may be infused over 60 minutes. The total amount of IV fluid administration should be minimized in children and in older adults. Antivenom should never be injected into the fingers or toes.

Management of Recurrence

Very little exists in the scientific literature to support firm recommendations for the treatment of recurrent local or coagulopathic effects after antivenom administration. In fact, reports of local recurrence after envenomation had not been described separately in the United States prior to the FabAV trials.[62] As described earlier, recurrence of symptoms led to a change in the recommended dosing of FabAV.[6,20,21] Instead of the original dosing, patients received a loading dose, followed by additional doses at 6, 12, and 18 hours. None of the patients who received the scheduled doses of FabAV over 18 hours required further doses. Because only a small number of patients (31) were in this study, all patients should be monitored closely for extension of local effects, and some may require additional FabAV doses. As experience accumulates in this area, the recommended dosing

regimen and treatment suggestions for local recurrence may change.

Patients exhibiting signs of coagulopathy in the first 12 hours after FabAV treatment is completed have a 66% risk of recurrence.[6] Recurrence is expected to be of the kind and degree of the initial coagulopathy. Patients who have a normal platelet count and who do not demonstrate an elevation in the level of fibrin degradation products during the hospital phase of treatment are unlikely to exhibit coagulopathy at follow-up, but they should be reevaluated within 5 days of treatment. Patients who exhibit abnormal coagulation during the first 36 hours after envenomation should be followed closely (at least every 48 hours) until the coagulation values are stable or clearly improving for several days. If coagulation studies are significantly abnormal on follow-up or if they continue to follow a worsening trend, consideration should be given to retreatment with FabAV.

Boyer and colleagues recommend that retreatment be considered for (1) fibrinogen concentration less than 50 µg/mL, platelet count less than 25,000/mm^3, International Normalized Ratio greater than 3.0, or a partial thromboplastin time greater than 50 seconds; (2) a lesser degree of abnormality in these same laboratory values but with multiple components involved; (3) high-risk behavior for trauma; (4) comorbidity; or (5) a worsening trend in a patient with prior severe coagulopathy.[6] Although data are scarce for definitive recommendations, it is recommended that the patient requiring retreatment be given two vials of FabAV and monitored closely. Coagulation parameters should be followed daily after retreatment. Patients should be cautioned to avoid trauma. Hospitalization should be considered for patients who have severe hemorrhage or comorbid conditions. The duration of retreatment should be based on individual response to therapy.

Complications

Animal serum products can produce a wide range of adverse effects, from mild cutaneous reactions to anaphylaxis and death. Antivenom reactions may occur during the infusion (anaphylaxis) or may be delayed (serum sickness).[19] Heterologous, non-neutralizing proteins such as albumin, alpha- and beta-globulins, and IgM are produced by incomplete purification of the antivenom product. These heterologous proteins are responsible for the life-threatening anaphylactic and anaphylactoid reactions and the more common, self-limited delayed reaction known as serum sickness.

The clinical trials of FabAV reported acute reactions in six of the 42 patients, accounting for 14% of that small number of patients.[19] In addition, these cases were clustered around one lot of FabAV thought to be contaminated with excess residual Fc fragments. All the reactions to FabAV were minor except for one case of mild bronchospasm that responded well to bronchodilator therapy. Intuitively, the elimination of the Fc fragment in the production of FabAV seems to be responsible for the decreased incidence of anaphylactic and anaphylactoid reactions experienced with its use. The incidence of acute reactions following the administration of ACP ranges from 23% to 56%, based on retrospective data.[33,37,74]

True anaphylactic reactions, known as type I hypersensitivity, result from IgE-mediated degranulation of mast cells and the subsequent release of vasoactive mediators. Histamine, leukotriene, platelet activating factor, adenosine, neutrophilic chemotactic factor, and eosinophilic chemotactic factor have been identified as active components in anaphylaxis.[44] These substances cause hemodynamic instability and hypotension by inducing vasodilation and increasing capillary permeability. Bronchospasm, laryngospasm, and death may result without aggressive airway management and pharmacologic intervention.

In contrast, anaphylactoid reactions are clinically indistinguishable from anaphylaxis but are not IgE mediated. Anaphylactoid reactions are often related to the dose, concentration, and rate of infusion of the antivenom preparation.[39,55] Mast cell degranulation in anaphylactoid reactions is postulated to result from direct interaction of non-neutralizing antivenom proteins with the mast cell membranes. In ACP, the Fc fragment of the IgG molecule is theorized to interact directly with the mast cell membranes, causing degranulation of the mast cells and activation of the complement cascade.[41] The resulting release of vasoactive mediators leads to hypotension, bronchospasm, and airway compromise. Management of acute antivenom reactions should include epinephrine (IV bolus or infusion),[52] antihistamines (H_1 and H_2 blockers), administration of isotonic fluids, and aggressive airway support, if warranted.

Patients with significant envenomations and a history of allergic reaction to either equine- or ovine-derived antivenom represent a difficult challenge. There is only one case in the literature of a patient with a known sensitivity to horse serum who developed an acute allergic reaction to FabAV antivenom.[14] The patient had a positive skin test prior to the administration of ACP, the only antivenom readily available at the institution. She was premedicated with H_1 and H_2 blockers, but even so, she developed angioedema after infusion of only 1 mL of the ACP antivenom. The hypersensitivity reaction responded well to methylprednisolone and epinephrine. Unfortunately, the envenomation progressed over the next 17 hours, leading the medical team to decide to give FabAV. The FabAV was started slowly, but the patient developed erythroderma, urticaria, and facial angioedema within the first 24 minutes. She was treated with H_1 and H_2 blockers, methylprednisolone, and repeated subcutaneous doses of epinephrine. The symptoms subsided quickly and the infusion was completed without recurrent symptoms. The patient had no further sequelae, and no evidence of coagulopathy or serum sickness at follow-up 2 weeks later. Patients with previous life-threatening allergic reactions to antivenom may not necessarily have recurrent reactions when rechallenged. Even so, prudent management would involve pretreatment with epinephrine and antihistamines (H_1 and H_2 blockers), establishment of IV access, appropriate fluid therapy, availability of advanced airway support, and monitoring in a critical care environment during and following administration of antivenom.

In addition to the acute effects of antivenom administration, a delayed type III hypersensitivity reaction may occur. The syndrome of serum sickness is common[17,37,66] after administration of ACP, with a strong correlation between ACP dosage and serum sickness frequency.[25,75] In retrospective studies by Jurkovich and coworkers[37] and Downey and colleagues,[25] the incidence ranged from 18% to 86%. LoVecchio and coworkers[40] reported an incidence of 56%, with increasing frequency correlating with number of vials given. There was a 100% occurrence of serum sickness in the group of patients who received a total of four or more vials. The only prospective study of ACP reactions reported that 6 of 8 patients developed serum sickness.[65] The clinical trials of FabAV exhibited a rate of serum sickness of 16%, occurring in 6 of 38 patients. However, five of the reactions were associated with an early

lot of antivenom in which the Fc fragment had been retained inadvertently.

The syndrome of serum sickness was originally described in 1905 by two pediatricians, von Pirquet and Schick, who noted that children receiving equine-derived streptococcal antitoxin sometimes developed fever, lymphadenopathy, and rash.[47] They were the first to use the term *serum sickness* to describe this constellation of symptoms resulting from the administration of heterologous serum. Modern definitions of this syndrome have varied little from the original description. Serum sickness is a spectrum of disease, typically characterized by fever, malaise, urticaria, lymphadenopathy, and arthralgias. Infrequently, glomerulonephritis or neuritis may be seen. The onset of symptoms usually develops 7 to 21 days after exposure to foreign antigens. In this case, the foreign antigens are the heterologous proteins found in antivenom prepared from equine or ovine serum.

The pathophysiology of serum sickness results from deposition of immune complexes, usually antigen–IgG, in susceptible tissue. Under nonpathologic conditions, these complexes are cleared by the RES without obvious consequences. Under conditions such as those present during the treatment of envenomation, specifically with increased vascular permeability and antigen excess, precipitation of the immune complexes occurs. These precipitated complexes diffuse between the endothelial cells and deposit along the basement membranes. The interaction between immune complexes (trapped in the vascular wall) and vasoactive mediators results in activation of the complement cascade, further release of vasoactive mediators, and exacerbation of the inflammatory process. Immune-complex deposition tends to occur in the vasculature of tissues with a filtering function. Characteristically, the skin, synovial joints, and glomerular apparatus are involved, resulting in the clinical manifestations of serum sickness.[15,37,44,77]

Treatment of serum sickness should be tailored to the individual needs of the patient. Symptoms are variable and range from low-grade fever and urticaria to debilitating arthralgias and myalgias. The mainstays of treatment are steroids and antihistamines. Patients should be treated with steroids, usually a 60-mg oral daily dose, tapered over 7 to 10 days. Diphenhydramine or hydroxyzine is given for the antihistaminic effect. In addition, H_2 blockers have been given in some cases.[30,40] Arthralgias and myalgias should be treated with appropriate narcotic or non-narcotic analgesics. Clinical illness usually resolves within 1 to 2 weeks.

The clinical use of antivenoms remains complex. The erratic and dynamic course of snake envenomation syndrome may require assistance of a physician experienced in the use of antivenom. A regional poison control center should be contacted for assistance with the treatment of patients who have been bitten by a native or an exotic snake. The regional centers are staffed by trained individuals who maintain a list of consulting physicians throughout the United States available for assistance with the management of venomous snakebite. The national hotline number is 800-222-1222.

ELAPID ENVENOMATION

Coral Snakes

Coral snakes are members of the family Elapidae; two genera, *Micrurus* and *Micruroides*, are native to the United States. The Sonoran or Arizona coral snake (*Micruroides euryxanthus*) is a small snake, generally 15 to 20 inches long. It is found in New Mexico and Arizona. The Texas coral snake (*Micrurus fulvius tenere*) and the eastern coral snake (*Micrurus fulvius fulvius*) are slightly larger and are native from North Carolina southward to Florida, and through the Gulf Coast states to Texas.

Coral snakes in the United States can be identified by characteristic broad red-and-black bands separated by yellow (or cream) bands on their bodies, although various black and albino color phases have been identified. A number of quite harmless snakes can mimic the coral snake, which led to the familiar rhyme, "Red on yellow kill a fellow, red on black venom lack." This rhyme only applies to coral snakes native to the United States. These snakes are shy and nocturnal, and they represent a very small portion of the total number of snakebites in the United States. Coral snakes have short, fixed fangs and no facial pits. They inject their venom through a series of "chewing" movements. It is estimated that 50% of all coral snakebites in the United States lack envenomation and are thus considered "dry" bites.[30]

The AAPCC reported 97 cases of coral snakebites in 2003.[73] The majority, 85 of the 97, were treated in health care facilities. Only four were considered to have a "major" outcome, and there were no reported fatalities.

As recently as 2002, Gold and colleagues[30] recommended that strongly suspected or proven *Micrurus fulvius* envenomations be treated with an initial dose of five vials of coral snake antivenom immediately. Progression of symptoms warranted an additional 10 to 15 vials and admission to the intensive care unit. Early antivenom treatment was recommended, because cranial nerve dysfunction and respiratory paralysis are difficult to reverse once the venom components are bound to the neuronal target tissue.[38] Treatment is recommended within the first 8 hours.

However, Wyeth has discontinued production of a coral snake antivenom (*Micrurus fulvius*).[78,79] No other approved coral snake antivenom is currently available in the United States. One study has shown the efficacy of Australian *Notechis* antivenom and Mexican *Micrurus* antivenom in mice.[76] Anticoral (Elapidae) snake antivenoms produced in Costa Rica have also found to be effective in neutralizing the venom of *Micrurus fulvius*, again in a mouse model.[2] Studies in Argentina comparing the usefulness of two common *Micrurus* antivenoms in that country showed limited effectiveness against North American *Micrurus* species.[24]

Management of a coral snake bite should involve a call to the regional poison control center for referral to a physician specializing in the treatment of elapid envenomation. The hotline number is 800-222-1222.

Other Elapids

The increasing popularity of herpetoculture as a hobby or a business has led to an increase in the number and variety of exotic snakes in the United States. The AAPCC reported 273 exotic snakebites in 2003, approximately half of which were considered poisonous.[73] Of these, nine were found to have major outcomes and there was one fatality. Cobras are members of the Elapidae family. The monocellate cobra (*Naja kaouthia*) appears to be the species that is most available in the United States. The cobras have a pair of short, fixed fangs in the front of the mouth that allow them to maintain a hold on the victim as venom is injected during a series of chewing movements.

No cobra antivenoms have been approved by the U.S. Food and Drug Administration (FDA) for use in the United States. Antivenoms are available in Africa, Asia, and Europe, but there are no international standards regulating quality, purity, or effectiveness. Most of these antivenoms are polyvalent and of equine origin. The AAPCC maintains a resource of the preferred antivenoms available for most venomous snakes around the world, which is useful if the snake can be positively identified. The resource, known as the *Anti-Venom Index,* also has information about where specific antivenoms can be found, and the AAPCC can help locate them and facilitate their rapid transport.

If an appropriate antivenom can be located and transported in a timely fashion, the health care team should prepare to manage administration of the antivenom and any possible consequences. For example, because elapid antivenoms are equine derived, the patient must be skin tested to identify an obvious severe hypersensitivity to the antivenom. Manufacturers provide a protocol for the skin testing.

A significant percentage of false-negative and false-positive reactions occurs. A negative test does not rule out the possibility of anaphylaxis, and the team should be prepared to handle that eventuality. Equally important, a positive skin test does not preclude the use of the antivenom. The purpose of the skin test is to identify the patient with obvious severe hypersensitivity so that the health care team is prepared to manage an allergic reaction.

SPIDER ENVENOMATIONS

There are approximately 34,000 species of spiders worldwide. All are fanged and venomous, but less than 0.5% can penetrate the human skin, and of those, only a few are considered dangerous. Of those considered dangerous, there are specific antivenoms for three. These are the brown recluse, the black widow spider, and the funnel-web spider.

Spiders in general are shy and not aggressive, but more spiders are being kept as exotic pets, leading to more opportunities for mishap. In general, a patient with clinically significant arachnidism presents with either necrotic or neurotoxic manifestations.

Brown Recluse

Necrotic arachnidism is exemplified by the response to the genus *Loxosceles,* or recluse spider. The *Loxosceles* spider produces a characteristic macular, erythematous, halo lesion that may progress to a necrotic ulcer. Sphingomyelinase D is thought to be responsible for the majority of the myonecrosis and local hemolysis.[51,67] Arachidonic acid, prostaglandins, and chemotactic infiltration of neutrophils are thought to be responsible for the intense inflammatory response.[32] Complement activation occurs via C-reactive protein, causing amplification of the intrinsic vascular cascade. Systemic loxoscelism is characterized by fever, jaundice, morbilliform rash, and intravascular hemolysis. Rarely, patients develop renal failure, seizures, and disseminated intravascular coagulation.[67]

There is no FDA-approved antivenom available in the United States. There is a *Loxosceles* antivenom available in some countries, although results have not been as promising in necrotic arachnidism as in neurotoxic arachnidism. Antiarachnidic serum is produced in Brazil, at the Instituto Butantan. The United Kingdom has approximately five vials of this product at two locations: the Aintree Hospitals NHS Trust in Liverpool, and the National Poisons Information Service, London Centre, London. The production of *Loxosceles* antivenom is small, and the product loses its potency rapidly, so there are very few readily available supplies.

Black Widow

The only nonsnake antivenom approved by the FDA for use in the United States is indicated for envenomation by the black widow spider *Latrodectus mactans*. Antivenom Latrodectus Mactans is produced by Merck and Co., Inc. The active component of *Latrodectus* venom is alpha-latrotoxin, a potent neurotoxin.[67] It alters presynaptic cation channels, including calcium channels. The resulting increased release and then depletion of neurotransmitters affect both autonomic and somatic nerves, disrupting nerve terminals. The primary manifestations of envenomation are headache, vomiting, and abdominal muscle spasm, which may be confused with peritonitis. The well-described *facies latrodectismica* is a painful grimace caused by facial spasm and trismus. Fortunately, the venom of the black widow spider rarely results in life-threatening manifestations. In certain conditions, however, *Latrodectus* antivenom may be indicated. In general, patients with underlying medical problems and those who may not be able to tolerate the large doses of benzodiazepines and narcotics required for management may be appropriate candidates for antivenom. Some authorities advocate antivenom use for older adults and for children. Coronary artery disease and chronic obstructive pulmonary disease, conditions in which severe pain may worsen the underlying condition, are diagnoses that warrant use of *Latrodectus* antivenom. Pregnancy, extremes of age, and failure to respond to pain relief measures are also indications for use of antivenom.[46,60]

The commercially available antivenom for the black widow spider is an equine-derived IgG product from the venom of *Latrodectus mactans*. Patients requiring the antivenom should be skin tested prior to administration. As discussed previously, the skin test is not reliably predictive of sensitivity.[41] As in the use of other equine sera, if a positive reaction occurs, the patient may be pretreated with H_1 and H_2 blockers and methylprednisolone, followed by administration of antivenom in an acute care environment. One 2.5-mL vial of antivenom is mixed with 50 to 100 mL of normal saline and infused intravenously over 1 hour. Most patients experience resolution of symptoms with one vial of antivenom.

Reactions to the antivenom are the same as those to the snake antivenoms, including anaphylaxis and serum sickness. Treatment of the acute reaction is the same as for adverse reactions to other antivenoms, and it includes H_1 and H_2 blockers, steroids, crystalloid if needed, and aggressive airway management. Treatment of serum sickness that may develop 7 to 21 days after the use of the antivenom involves oral antipyretics, analgesics, prednisone, and antihistamines. The management of black widow spider bite with antivenom involves careful calculation of the risk-to-benefit ratio of the envenomation and the treatment. Consultation with a physician specialist skilled in the use of *Latrodectus* antivenom is recommended and can be achieved by contacting the national hotline at 800-222-1222.

In Australia, antivenom for *Latrodectus* envenomation is available from Commonwealth Serum Laboratories, and in South Africa from the South African Institute of Medical

Research. Indications for antivenom use and routes of administration vary around the world.

Funnel-Web Spider

Although the funnel-web spider is not native to the United States, it is found in Australia and presents a classic picture of neurotoxic arachnidism. The initial local signs are usually mild. Shortly afterward, the patient develops painful regional lymphadenopathy, then headache, nausea, vomiting, sweating, horripilation (gooseflesh), and painful muscle spasms. The muscle spasms predominantly affect the legs and abdomen, mimicking the response to *Latrodectus*. Other features include hypertension, irritability, priapism, psychosis, renal failure, and cardiorespiratory failure.[67] The massive release of neurotransmitters caused by the atraxotoxin of the funnel-web spider results in uncontrolled autonomic hyperreactivity. Approximately 2 hours later, the patient experiences neurotransmitter depletion, weakness, and cardiovascular collapse. Death from the funnel-web spider bite usually occurs in the very young and the very old, and it happens approximately 2 hours after the initial envenomation. The use of specific antivenom in Australia has dramatically reduced mortality associated with this spider.

An antivenom for the Sydney funnel-web spider was developed for clinical use in 1981 by Dr. Struan Sutherland and his team at the Commonwealth Serum Laboratories. No deaths have occurred since its introduction. Much of the venom supplied for this research was obtained through a funnel-web milking program at the Australian Reptile Park. Antivenom is held at major city and regional hospitals in Australia.

MARINE ENVENOMATIONS

Marine envenomations are covered in detail in Chapters 73 and 74.

NEW HORIZONS IN ANTIVENOM THERAPY

Scientists continue their quest to find, refine, or develop the perfect mithridate for envenomations. Research is ongoing in several areas. Specific therapies targeting cellular-level responses to antivenom are being investigated. As more is understood about the inflammatory response to envenomation, biochemical therapies may follow. In addition, the body of knowledge about targeted receptors continues to increase. It is possible that future therapies will involve specific blockade of the identified receptors, using monoclonal antibody therapy or other developing delivery systems. Adjunctive therapies are being tried in addition to the antivenom therapies, seeking an improvement in clinical outcome. Different antivenom dosing strategies are being explored, especially in countries with limited economic resources. Alternate production pathways are being investigated as well, seeking more efficient and less costly animals than horses to produce antivenom. All are addressed in the following discussion.

Cellular-Level Interventions

Venom is a chemically complex mixture of enzymes, growth factor activators, and small low-molecular-weight polypeptides. Quantity, composition, and lethality vary with the species, weight, age, and diet of the snake. Venom proteins include hyaluronidase, phospholipase, phosphodiesterase, endonuclease, and transaminase. They range in molecular weight from 6000 to 100,000 Da.

Envenomation by snakes involves activation of proinflammatory cytokines and other mediators, such as nitric oxide. Recently, a battery of cytokines produced by activated T cells or macrophages has been added to the known cytokines of envenomation. Cytokines initiate a cascade of events that lead to illness behaviors such as fever and anorexia, as well as a host of physiologic events, such as activation of vasodilation, increased nitric oxide production, and hypotension. Accumulating evidence indicates that these cytokines play important roles in mediating the cell recruitment and activation necessary for both inflammation and the repair of tissue damage.[50] Proinflammatory cytokines, nitric oxide, and prostanoids are involved in spinally mediated pain facilitation. Tumor necrosis factor-alpha (TNF-α) and interleukin-1β (IL-1β) are also involved in the production of hyperalgesia.[81] One recent animal study suggests that the systemic release of TNF is responsible for the cardiotoxicity of *Vipera* venom.[68] Identification and isolation of the various biochemical components of the body's response to envenomation will lead the way to development of specific therapies at the cellular level.

Experiments with Habu snake venom point to a model of toxin activity that may be important for future therapeutics.[69,80] Vascular endothelial growth factor (VEGF) mediates vascular permeability in humans through VEGF receptor -1 (VEGFR-1) and VEGFR-2 activation. The Habu snake venom contains a venom-specific VEGF-like molecule annotated as TfsvVEGF. The TfsvVEGF binds the human VEGF-A site, but it has a unique binding affinity for VEGFR-1. The signaling via VEGFR-1 promotes vascular permeability without the VEGFR-2 signaling for angiogenesis and subsequent tissue repair. Further research is needed, but this holds promise for future treatment of envenomations using therapies targeting blockade of the VEGFR-1 site or recruitment of the VEGFR-2 site.

Adjunctive Therapies

Adjunctive therapies are being investigated to augment current therapy and optimize treatment. Investigation of pretreatment with a combination of antivenom, verapamil, and magnesium sulfate in box-jellyfish envenomation shows promise in the laboratory setting.[54] Other adjuncts under investigation include epinephrine, glucocorticoids, and aminophylline.

Investigators in Bangkok, Thailand, suggest that the coagulation abnormality caused by the Malaysian pit viper is secondary to activation of the complement cascade at several levels, and that antivenom alone is not sufficient to reverse or prevent this phenomenon. They used antithrombin (AT)-III in a rat model to produce synergy with antivenom that exceeded antivenom alone, AT-III alone, or a combination of low-dose AT-III and antivenom.[53]

Low-Dose versus High-Dose Antivenom

In some areas of the world where venomous snakebites are frequent and economic resources are scarce, research has focused on producing the optimal dosage of antivenom to ameliorate the condition at the least possible cost.[49] A trial in India investigated the use of antivenom in two doses for patients with envenomation and hematologic or neurologic changes. One group received high dose antivenom—two vials over 1 hour, followed by two vials over 4 hours and repeated every 4 hours until clotting parameters normalized, and then continuous infu-

sion of two vials over 24 hours. The other group received low dose antivenom—two vials over 1 hour, followed by one vial over 4 hours, repeated every 4 hours until clotting parameters normalized, and then continuous infusion of one vial over 24 hours. There was no difference in the transfusion, dialysis, or ventilation requirements of the two groups, and there was no mortality. The durations of stay averaged 4.94 days for the high-dose group and 3.48 days for the low-dose group. The study concluded that low-dose therapy was just as effective, and it represented a saving in both cost of antivenom and cost of hospital stay.[70]

A retrospective analysis of neurotoxic envenomations requiring mechanical ventilatory support in an intensive care setting supports the conclusion that low-dose antivenom therapy is as effective as high-dose antivenom therapy in elapid envenomations.[1] Either high-dose therapy or low-dose therapy was selected empirically in the clinical setting, in the absence of measurements of serum venom concentrations. There were three deaths (out of 55 patients) in the high-dose group and two permanent neurologic sequelae in the low-dose group. All other patients left the hospital neurologically intact.

Avian Production

In Australia, research has begun on the use of chickens to produce a Fab antivenom product. Because chickens can be produced, fed, and housed more economically than cows, sheep, or horses, this holds some promise for less expensive antivenom. In addition, harvesting antibodies from eggs instead of via phlebotomy may introduce a new efficiency into the production process. The isolation is relatively simple, involving removal of the lipids from the diluted egg yolk, followed by gel filtration. Each egg produces 80 to 100 mg of pure immunoglobulin, and specific antibodies are present for about 100 days after immunization. The avian antivenom neutralizes the thrombinlike activity of equivalent amounts of venom on human plasma.[42]

Scientific forward thinkers believe that, much like the antiserum use of the 1930s, antivenom use will decline as science and technology present biochemical advances and developments. Until that time, antivenom remains the mainstay of therapy in envenomations.

The references for this chapter can be found on the accompanying DVD-ROM.

Bites and Injuries Inflicted by Wild and Domestic Animals

51

Luanne Freer

Wild and domestic animal bites are distinct from other injuries suffered by humans. Tearing, cutting, and crushing injuries are sometimes combined with blunt trauma caused by falls. Few traumatic lacerations are as regularly contaminated with as broad a variety of pathogens as are animal bites. Domestic animal bites are common, and their incidence is rising.[28,124,149] Wild animal attacks are often more spectacular, but in the developed world, injuries from domestic animals have a much greater health and economic impact. Humans are not a *natural* prey of any animal. Although some attacks are predatory, most are caused by fear of humans (real or perceived), territoriality, protective instinct, or accident.

Other special features are as follows:

- Many victims have been terrorized by the attacking animal.
- Animals can transmit systemic diseases, many of which induce substantial morbidity and mortality (see Chapter 53).
- In contrast to the extensive scientific literature on traumatic injuries that do not involve bites, the literature on animal bites, especially wild animal bites, is largely unscientific and often anecdotal. As a result, treatment decisions are often made without a completely satisfactory scientific basis.
- Many decisions involved in the treatment of wild animal attack victims are based on experience with domestic animal attacks—that is, dog and cat bites.
- Perhaps most important, animal attack injuries are usually preventable. When experience allows humans to understand typical behavior for a species, people can take proper precautions in the vicinity of potentially dangerous animals.

This chapter interprets the present state of knowledge to make logical, specific recommendations for all of these features.

EPIDEMIOLOGY

Domestic Animal Bites and Injuries

The 2006 National Pet Owner Survey estimated that more than 69 million households own at least one pet.[7] The great majority of bites (about 80% to 90%) are inflicted by dogs.[96]

sion of two vials over 24 hours. The other group received low dose antivenom—two vials over 1 hour, followed by one vial over 4 hours, repeated every 4 hours until clotting parameters normalized, and then continuous infusion of one vial over 24 hours. There was no difference in the transfusion, dialysis, or ventilation requirements of the two groups, and there was no mortality. The durations of stay averaged 4.94 days for the high-dose group and 3.48 days for the low-dose group. The study concluded that low-dose therapy was just as effective, and it represented a saving in both cost of antivenom and cost of hospital stay.[70]

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Domestic cats account for about 5% to 15%, although some studies report a figure as high as 25%.[96,135,218]

Each year, an estimated 1.8% of Americans are bitten by dogs, resulting in 4.7 million bite wounds. More than 750,000 of these bite victims seek medical attention.[41] Bites to children are common, with boys ages 5 to 9 years having the highest incidence.[41] These figures represent only bite victims who sought medical attention; one analysis of dog bite incidence in Pittsburgh showed that 790 bites were reported but that an estimated 1388 were unreported, yielding an annual incidence of 58.9 per 10,000.[44] In India, where stray dogs cause 96% of rabies cases, the annual dog bite rate is 25.7 per 1000, and the most common victims are males.[3,46]

The victim of a bite is often a pet owner or a member of the owner's family, and injury is frequently sustained while playing with the animal.[44] Surveys of schoolchildren have shown that 55% of boys and almost 40% of girls have been bitten, with 17% requiring medical attention.[21]

The annual incidence of cat bites is about 400,000 in the United States,[192] which is probably an underestimate. Biting cats are typically stray females, and most human victims are also female.

Of the approximately 30 million Americans who ride horses, 50,000 a year are treated for horse-related injury in an emergency department, principally because the rider is unrestrained and can fall off while traveling at speeds of up to 40 mph (64 km/hr). Horses can kick with a force of up to 1 ton, and they frequently bite. A 2-year review of animal bites in Oslo revealed that 2% of 1051 recorded bites were caused by horses; 53% of these horse bite victims were children.[62]

The American Ferret Association estimates that 6 to 8 million domesticated ferrets reside in the United States.[181] The risk of attack by a ferret is greatest in infants and small children. Bite statistics are scarce, but in Arizona, 11 ferret bites were reported over 11 months. The ferret population in Arizona was estimated at 4000, so the ratio of reported bites to total number of ferrets was about 0.003.

Wild Animal Bites and Injuries

Neither the annual number of wild animal bites nor the population at risk can be reliably estimated, especially when the human population to be considered consists only of those people exposed to a wild animal or in a wilderness setting. The world supports approximately 4600 species of mammals, 10,000 species of birds, and 6000 species of reptiles,[141] and the actual number of wild animals in the world is estimated to be in the billions. Many people who suffer relatively minor injuries from wild animals do not seek medical attention unless infection or another complication occurs, or unless they fear exposure to rabies. If the injury is minor, patients are generally treated and released, without a notation that would lead to statistical analysis.

Few studies have examined the incidence of wild animal bites (Table 51-1). In Sweden, three of 1000 inhabitants were injured by animals each year.[29] Domestic animals accounted for over 90% of the total, moose accounted for 6% (almost all involved in auto accidents), and all other animals totaled 4%. However, bites were not examined separately, and many injuries occurred during accidents caused by animals. Some officials estimate there are two additional bites for every one reported, but a survey of children 4 to 18 years old estimated an incidence of more than 36 times the reported bite rate. These figures are most likely based on domestic bites, although this was not specifically stated.[23,24] During a 7-year period in Texas, 2% of all trauma related to large animals was caused by wild animal attacks.[153]

TABLE 51-1. Incidence (%) of Animal Bites in the United States as Reported in Five Studies

	STUDY				
ANIMAL GROUP	*A*	*B*	*C*	*D*	*E*
Dog	89	91.6	78	—	75
Cat	4.6	4.5	16	—	20
Rodent	2.2	3	<1	65	2.5
Monkey	0.1*	0.2	2.2	15	0.2
Skunk	—	0.02	0.02	0.1	—
Lagomorph	0.2	0.5	—	—	1
Large mammal	0.03†	0.01‡	1.2	3§	0.5
Reptile	0.1	—	—	—	—
Bat	—	0.004	0.3	6	0.7
Raccoon	—	0.08	1	3‖	0.5

A, Marr J, Beck A, Lugo J: An epidemiologic study of the human bite. Public Health Rep 94:514, 1979.
B, Scarcella J: Management of bites. Ohio State Med J 65:25, 1969.
C, Sinclair CL, Zhou C: Descriptive epidemiology of animal bites in Indiana, 1990–1992: A rationale for intervention. Public Health Rep 110:64, 1995.
D, Kizer K: Epidemiologic and clinical aspects of animal bite injuries. JACEP 8:134, 1979.
E, Spence G: A review of animal bites in Delaware—1989 to 1990. Del Med J 62:1425, 1990.
*Includes 21 monkeys, 4 raccoons, 3 ferrets, 1 weasel, 1 coatimundi, 1 skunk, and 1 goat.
†Includes 3 lions, 1 ocelot, 1 leopard, 1 polar bear, and 1 anteater.
‡Includes 1 goat, 1 ocelot, 1 jaguar, and 1 groundhog, which inflicted a bite on Groundhog Day.
§One coyote.
‖One kinkajou.

No reported statistics exist on the typical victim of wild animal attacks. Two U.S. state health departments report that if all animal bites (including domestic) are considered, animal bites occur most often in boys aged 5 to 9 years.[191,194] However, more than 90% of animal attacks in these states are caused by domestic animals, so this group is probably not representative of wild animal attacks. In underdeveloped countries, many persons are exposed daily to bites from species considered exotic in the developed world.

Persons in certain occupations in developed countries, such as veterinary, animal control, and laboratory workers, are at greatest risk of wild animal bite. A British survey reported a 70% incidence of animal-handling injuries during the course of a typical veterinarian career; the rate was 42% in veterinary technical staff.[57] The U.S. Bureau of Labor Statistics reported that in a 5-year period, 186 occupational injury fatalities were caused by animal attack, and that the majority were caused by cattle.[142] In one study of 102 animal control officers, the overall bite rate was 2/57 per working day, 175 to 500 times the estimated rate in the general population (this study did not differentiate between wild and domestic animal bites).[23] The incidence of biting species varies with exposure. Among veterinarians, most injuries are inflicted by cattle, followed by dogs and then horses.[123]

In every statistical series of bites, small numbers of exotic animals, such as ocelots, jaguars, lions, leopards, polar bears,

TABLE 51-2. Human Deaths from Animal Attacks

ANIMAL	ANNUAL ESTIMATED DEATHS	ANNUAL ESTIMATED ATTACKS	PREDOMINANT AREA	COMMENTS
Humans	200,000	—	Worldwide	Individual murders only; excludes 1 million/yr by war in recent decades
	3,400	3 million	United States only	Deaths by firearm, both intentional and accidental; attacks include all assaults
Snake	60,000	—	Worldwide	—
Crocodile	1,000	—	Africa	Man eating, predominantly
Alligator	<1	7	Florida only	Human encroachment on alligator habitat
Tiger	800–1,600	800–1,600	India	Frequently man eaters
Lion	60–200	3–17	Africa, India	Frequently man eaters
Leopard	30–125	4–16	Africa, India	Frequently man eaters
Elephant	200–500	—	Central Africa, India	Occasionally rogue man-killers; incidence increasing because of environmental pressures
Hippopotamus	200–300	—	Africa	Unpredictable; bites and trampling common
African buffalo	20–100	—	Africa	Only if cornered or wounded
American bison	<1	3–5	United States only	Usually only when approached at close range
Moose	1–2	20	Sweden only	Almost always involves vehicle collision with animal
Hyena	10–50	Hundreds	Africa	Frequently bites off face of sleeping victim
Wolf	20–50	1–5	Worldwide, mostly	Most deaths in 1800s in Eurasia
Domestic dog	10–20	1,500,000	United States only	—
		25.7/1,000	India only	—
Gorilla	0	2–3	Africa	Only if cornered; injuries usually not severe
Baboon	0	1–2	South Africa	Usually pets
Ostrich	1–2	—	South Africa	Disembowelment, kick to head, stomping
Emu	None reported	1–2	Worldwide	Usually on farms
Black rhinoceros	<1	—	Africa	Easily provoked

wolves, anteaters, and weasels, are represented. Bites from these animals occur from exposure to wild and zoo animals, and from the increasing popularity of wild animals as pets, which are usually kept illegally and without adequate training and understanding regarding animal care and behavior.

Several thousand people per year are killed by mammalian bites, and most of the deaths are inflicted by man-eating lions and tigers in Africa and Asia (Table 51-2). The World Health Organization (WHO) estimates that roughly 60,000 people per year are killed by snakes and that additional millions are killed by insect-borne diseases.[217] An estimated 200 persons are killed by animals in the United States each year; 131 of these die in traffic accidents involving deer.[102] Bees kill approximately 43 persons; dogs, 14; and rattlesnakes, 10. Wild animals, such as bears and cougars, kill fewer people than do goats, rats, jellyfish, and a captive elephant. More than 3 million people visit the wilderness in Yellowstone National Park every year, but the incidence of serious injury by a wild animal at the park is less than the chance of being struck by lightning.[97]

PREVENTING ANIMAL BITES

Prevention of animal bites requires a thorough knowledge of the patterns of behavior and personalities of various species of animals. A person wishing to avoid the bite of a particular species is often able to gain expertise about that species' behavior only from those who work with it regularly. Detailed information on animal behavior and the attack patterns of animals is also available on internet websites listed in Box 51-1.

Box 51-1. Animal Behavior and Attack Prevention Websites

Dog and cat attack/bite prevention
www.dogexpert.com
Dog bite prevention and legal information
www.dogbitelaw.com
Domestic pet bite prevention information
www.petplace.com
Cougar attack prevention
http://wildlife.state.co.us/Education/mammalsguide/mountain_lion.asp
Moose, wolf attack avoidance
www.state.ak.us/adfg/wildlife/wildmain.htm
Wolf behavior and human encounters
www.bitterroot.net/wild
Hunter education
www.everythingalaska.com/eta.akgovx.html
Wild animal attack compilations
www.igorilla.com/gorilla/animal
Animal attacks by circus animals (PETA site)
www.circuses.com
Animal behavior information with links to seven animal behavior journals
http://cricket.unl.edu/nbbg.html
Animal behavior news groups
www.sci.bio.ethology

Box 51-2. How to Avoid Being Attacked and Bitten by Common Pets

DOGS

Do not leave a young child alone with a dog.
Never approach or try to pet an unfamiliar dog, especially if it is tied up or confined.
Always ask owners if you can pet their dog.
Do not lean over a dog or pet it directly on the head; do not kiss a dog.
Avoid quick, sudden movements that may startle a dog.
Never pet or step over a sleeping dog.
Never try to take a bone or toy from a dog (other than your own).
Know the appearance of an angry dog: barking, growling, snarling with teeth showing, ears laid flat, legs stiff, tail up, and hair on back standing up.
Never step between two fighting dogs; if you need to separate them, use a bucket of water or a hose.
Do not approach a female dog that is nursing her pups.
Teach children these tips from an early age.

CATS

Be aware that some cats do not like prolonged petting.
Know warning signs of an impending bite: twitching of the tail, restlessness, and "intention" bites (i.e., cat moves to bite but does not bite).

FERRETS

Do not sell or adopt a ferret that is known to bite.
Do not push your fingers through the wires of a ferret cage.
Reach for a ferret from the side, palm upward, rather than from above.
Do not handle food and then handle young ferrets without washing your hands first.
Do not poke a ferret or pull on its tail or ears.
Never leave a ferret alone with a child or infant.
If a ferret bites and locks on very tightly, pour cold and fast-running water over its face.

Box 51-3. What to Do When Threatened or Attacked by a Dog

Stand totally still and let the dog come to you.
Stand passively rather than in an aggressive or submissive pose.
Do not pat the dog.
Keep your eye on the dog, but do not stare at it.
To reprimand the dog, say "no" in a harsh voice; do not attempt to hit the dog.
Do not make any threatening or provocative movement.
Do not fight back, especially against a "fighting" dog.
Do not let the dog get behind you; keep turning to face it.
If you are knocked down, feign death and curl up into a ball until the dog loses interest.
If the dog punches you with its nose, ignore it.
If a dog puts one of your legs in its mouth without tearing the flesh, waiting for a reaction, stay still if you can.
If attacked by more than one dog, try and stand with your back to a wall or car.

Modified from Wilson S: Bite Busters: How to Deal with Dog Attacks. New York, Simon & Schuster, 1997.

Domestic animals of any kind rarely attack unless provoked, although unrestrained dogs may be exceptions. Physical attack is often a last resort, but an animal often fights if it perceives that it is trapped. Common sense and knowledge of animal behavior can be used to reduce the risk of injury. For example, horses kick backward and with both rear feet, whereas cattle kick forward with only one foot.

How humans react during a confrontation with an animal is also important, and following recommendations can reduce the chances of being attacked and bitten by a pet (Box 51-2). Nonpredator species such as cattle and deer are very susceptible to human intimidation, whereas a direct stare to a dog may be seen as a challenge. Dogs are guided by memory and instinct; fear and self-preservation are very strong instincts, so any perceived threat can lead to an attack. Territoriality is still ingrained in domestic dogs, even if humans provide for them. Protection of food can cause aggression, even in a docile dog. Any threat to a dog's mate, offspring, or owner may result in an attack. Personality changes may lead to aggression; causes include illness (e.g., distemper) or physiologic factors (e.g., female in heat). Specific actions can be taken when threatened or under attack by a dog (Box 51-3).

Like their domestic counterparts, wild animals rarely attack people without provocation (although the animal's perception of provocation may differ from the human's perception). Exceptions are large carnivores, which may be relatively unafraid of humans, and creatures clinically infected with rabies. However, carnivores do not commonly hunt humans as preferred prey. Patterns of behavior and attack differ by species.

People often capture or restrain wild animals, creating stress that may induce even the most benign animal to turn on its captor. Allegedly tame animals are very likely to struggle. Even shy animals that are being captured for treatment of an injury may attempt self-defense and can inflict a life-threatening injury, such as a goring. Therefore, all situations of animal restraint and capture are considered high risk, and careful study of the species' behavior, the individual animal, and the physical environment and resources should precede actual attempts at restraint (see Chapter 55).

Because people seem drawn to raising wild animals as pets,[133] a large and lucrative market exists, particularly in the United States. No matter how they are raised, these animals remain wild and will never be as predictable, trustworthy, and nonaggressive as animals that have been domesticated for centuries. Often, owners demonstrate a lack of common sense, as in the case of a pet Bengal tiger that attacked and killed its trainer, then 6 weeks later did the same to its owner.[16,69]

Most wild species have a strong sense of territoriality. Individuals, pairs, or larger groups establish a territory that ranges from square feet to square miles and aggressively prevent any intrusion into that territory, particularly by members of their own species. During mating season, this drive may stimulate even small animals to threaten or attack humans, particularly in protection of the nest and young.

A major principle of animal behavior is that physical attack is often a last resort, and they generally give ample warning of their intentions. Spectacular contests between animals that occur in the wild are governed by elaborate rituals and rules that encourage a nonviolent solution, so that the victor may successfully defend its territory and itself with little or no injury. Humans can often avoid attack and injury by successfully interpreting visual, auditory, and olfactory warning signs. If a human slowly and carefully backs off without making sudden or threatening gestures, usually no harm will be done, but the ideal reaction depends on the species. For example, mountain lions have been turned from a full charge by an angry human who acted aggressively or fought back.[25] Given a choice of victims, such a predator prefers the fleeing, panicked victim who demonstrates expected flight rather than unexpected behavior.

If an animal must be captured, detailed preparations should be undertaken. For small animals, using nets or heavy cloth and wearing extremely heavy gloves and other protective clothing are advisable. Desperate animals can bite with tremendous force; large carnivores can easily amputate a gloved digit. A wolf can tear apart a stainless steel bowl with its teeth, and a hyena can bite through a 2-inch plank.[52] Four men are needed to subdue an adult chimpanzee; an orangutan can maintain a one-fingered grip that an adult human cannot break. Larger animals generally require a team approach by animal control specialists with equipment such as nets, barriers, cages, and immobilizing drugs. Ideal immobilization techniques for various species are detailed in veterinary texts.[82]

TREATMENT

Prehospital Treatment

Attacks by domestic and farm animals are fairly predictable and preventable, unlike attacks in the wilderness. In Africa, life-threatening attacks by large animals, such as water buffalo, lions, tigers, and elephants, are common. Attacks by large animals can result in major blunt or penetrating trauma, with possible major arterial blood loss, airway damage, broken ribs, pneumothoraces, and intraperitoneal bleeding. The victim's condition and the availability of rapid evacuation determine the extent of treatment in the field. Medical personnel or supplies are often not available, so the victim must be moved to a hospital or clinic as soon as possible.

Many of the complications and serious infections from animal bites are caused by inadequate first aid and a delay to medical care. Local wound treatment should be initiated at the scene of the bite (Box 51-4), and, more than any other therapy, can determine the course of healing. Simple first-aid measures must be initiated immediately unless definitive or better treatment will be available in a short time (see Chapter 20). Pressure on the wound or on pressure points controls most bleeding; avoid tourniquets unless blood loss cannot otherwise be controlled. If the victim is more than 1 hour from a treatment facility, cleanse the wounds at the scene as soon as resuscitation efforts are complete. Early cleansing reduces the chance of bacterial infection and is extremely effective in killing rabies and other viruses. Potable water, preferably boiled or treated with germicidal agents, is adequate for wound irrigation. Ordinary hand soap adds some bactericidal, virucidal, and cleansing properties. If a 1% povidone-iodine (Betadine) solution is available, it should be used as an irrigant. Thoroughly irrigate the wound with at least a pint of soapy water and then gently debride it of dirt and foreign objects by swabbing with a soft, clean cloth or sterile gauze. Irrigation with a syringe is preferable, if available.

After cleansing, cover the wound with sterile dressings or a clean, dry cloth. Wounds of the hands or feet require immobilization. If the wounds are at high risk for infection, treatment is hours away, and an antibiotic such as amoxicillin-clavulanate, azithromycin, or ciprofloxacin is available, it is reasonable to start immediate treatment with an oral dose.[81,88] To most effectively prevent subsequent wound infection, this should be given within 1 hour of the wounding. However, for a severe wound, it is worthwhile to provide the antibiotic even many hours later. If antibiotics are not available, the wound is infection prone, and medical care is hours or days away, a simple remedy such as filling the wound with honey may be an effective antibacterial strategy.[165]

When definitive medical care cannot be obtained for a day or more, the wound should be cleansed and irrigated thoroughly. For some wounds, attempting closure is reasonable. Further discussion of bandaging and wound-repair techniques may be found in Chapters 18, 20, and 21.

Box 51-4. Treatment for Animal Bite Wounds

MANDATORY FOR ALL WOUNDS

1. Evaluate for potential blunt trauma and injury to deeper and vital structures by penetrating teeth, claws, or horns.
2. Ensure appropriate tetanus immunization.
3. Irrigate wound with copious volume (minimum, 100 to 300 mL) of normal saline or 5% povidone-iodine solution.
4. Debride obviously crushed and devitalized tissue.
5. If the animal is suspected to be rabid (atypical behavior, high-risk species), do the following:
   a. Infiltrate wound edges with 1% procaine hydrochloride.
   b. Swab wound surface vigorously with cotton swabs and 1% benzalkonium chloride (Zephiran) solution or other soap.
   c. Rinse wound with normal saline.
   d. Assess need for rabies immune globulin and vaccine.
6. Assess risk factors to decide on further (selective) treatment (see Box 51-5).
7. Do not culture fresh wounds.
8. Do not give prophylactic antibiotics for routine low-risk bite wounds.

SELECTIVE TREATMENT (DEPENDING ON RISK FACTORS)

1. Suture, staple, or adhesive-strip closure of all skin wounds in the usual fashion unless wounds are high risk (e.g., hand wounds, high-risk species, immunosuppressed patient).
2. Culture infected wounds only if they fail to respond to initial antibiotic therapy, they are very high risk (see Box 51-5), or there is evidence of systemic sepsis.
3. Consider delayed primary closure of high-risk wounds and administration of prophylactic antibiotics to victims with high-risk wounds.

In addition to treating the bite victim, try to capture the offending animal for examination, if this can be done without risk of further injury. Unusual behavior, such as an unprovoked attack by a wild animal in broad daylight or a complete absence of fear of humans, should raise the level of suspicion for rabies. Live capture is optimal, but freshly killed animals are usually satisfactory for examination for fluorescent rabies antibody (FRA). Avoid damaging the animal's head and brain, because brain tissue is needed for analysis. Availability of the animal can eliminate the need for costly and uncomfortable rabies prophylaxis. If more than 1 hour will elapse before the animal can be transported to a hospital or public health department, refrigerate the body. Do not use preservatives. Rabies is discussed in Chapter 54.

Examination of the animal is not useful for most other diseases and will not help predict local wound infections. Therefore, use good judgment in deciding how much time and energy to expend on capture.

Hospital Evaluation and Treatment

Evaluate all victims of animal bites for blunt trauma and internal injuries, which may be less obvious than the bite wound (see Box 51-4). Many animals are large, strong, and heavy, and victims should be treated like any other victim of blunt or penetrating trauma. Internal organ damage, deep arterial and nerve damage, and penetration of joints are all possible. Particularly in children, animal bites can penetrate vital structures such as joints or the cranium;[32,131] radiographs are needed whenever these injuries are suspected. A complete head-to-toe evaluation for trauma is advised in all but the most trivial and isolated bite injuries. Few laboratory tests are of use in evaluating animal bite injuries. Unless hematocrit is being assessed for evidence of blood loss from occult trauma, the complete blood cell (CBC) count is not useful because it is a nonspecific and an unreliable gauge of infection. Definitive trauma evaluation and treatment are discussed in detail in Chapter 20. Routine wound cultures obtained at the time of initial wounding do not reliably predict whether infection will develop or, if it does, the causative pathogens.[81]

The principles of wound care for injuries caused by inanimate objects apply to bite wounds as well. Many bite injuries are simple contusions that do not break the skin. The infection potential of these injuries is low; superficial wound cleansing and symptomatic treatment of pain and swelling suffice. Treatment should include prompt and liberal application of ice or other cold packs during the first 24 hours. However, this is not beneficial in snakebite and is clearly impractical in many underdeveloped locales. Snakebite is discussed in Chapters 48 and 49.

When skin is broken, the risks of local wound infection or transmission of systemic disease are incurred. Infection can be caused by organisms carried in the animal's saliva or nasal secretions, by human skin microbes carried into the wound, or by environmental organisms that enter the wound during or after the attack. Virtually any bacterium, virus, or fungus can become a bite wound contaminant. Fortunately, most wounds do not become infected.[139]

Animal bites are not clean lacerations but are instead crush injuries that usually contain devitalized tissue. Debridement removes bacteria, clots, and soil far more effectively than does irrigation.[81] In addition, debridement creates cleaner surgical wound edges that are easier to repair, that heal faster, and that produce a smaller scar. Topical antiseptic ointments, such as neomycin, bacitracin, and polymyxin, are highly effective in promoting healing in minor skin wounds.[85,128] However, although topical ointments are appropriate for abrasions produced by animal bites, they may be less effective for punctures and sutured lacerations.

A sutured wound is covered by a simple, sterile, dry dressing to protect from rubbing against clothing or repetitive minor trauma. Delayed primary closure requires that the wound be kept moist; this is usually done with a wet saline dressing.

Wound Closure and Infection Risk Factors

Three major considerations govern the decision of whether to suture a wound: cosmetics, function, and risk factors. Cosmetic appearance virtually mandates suturing all facial wounds, which are usually low risk. Similar reasons may dictate closure of wounds on other visible portions of the body. Function is of critical importance in wounds of the hand and foot, which are high-risk areas in which infection can have disastrous consequences. Thus, hand wounds should generally be left open. Risk factors are many and complex and provide a useful logical framework in making the decision to suture, administer antibiotics, or undertake other treatments. For more information on surgical procedures, see Chapter 20.

The amount of time elapsed after wounding is a critical risk factor: the longer the interval, the more likely is the chance for infection. After the first few hours, adequate wound cleansing is unlikely to be accomplished. In developed countries, many victims are seen within hours of wounding, and the results are usually very good. In remote and underdeveloped areas and countries, wounds commonly do not receive medical attention for half a day or more, putting them into a high-risk category that may eliminate the possibility of suturing. Certain species, including primates, wild cats, pigs, and large wild carnivores, seem to inflict infection-prone wounds, although evidence is incomplete. The presence of one or more of these risk factors may preclude suturing or may suggest the use of delayed primary closure (discussed later). However, most fresh bites can be safely sutured after proper wound preparation, and data suggest that carefully selected mammalian bite wounds can be sutured with approximately a 6% rate of infection.[45] Optimal conditions include prompt medical treatment, which is seldom available in remote and underdeveloped areas. In those locations, leaving bite wounds open (or with a drain, although this is also controversial) is the more prudent course.

Bites of the Hand

Hand bites are common, and infection can be disastrous.[211] Therefore, the hand is considered at risk for complications (Box 51-5). The hand contains many poorly vascularized structures and tendon sheaths that poorly resist infection. The fascial spaces and tendon sheaths of the hand communicate with one another, and movement seals off the wound from external drainage and spreads bacteria and soil internally. Because of the unique anatomy of the hand, thorough irrigation of wounds is often impossible.

Data on hand wound infection have been collected mostly from experience with domestic dog and cat bites. From a retrospective study in Oslo, nearly all hand bite wounds healed uneventfully when the wounds were left open, either without antibiotics or with penicillin after wound treatment.[62] In another European center, the total infection rate was 18.8% in hand bite wounds; this rose to 25% when the hand wound was

Box 51-5. Risk Factors for Infection from Animal Bite

HIGH RISK

Location
Hand, wrist, or foot
Scalp or face in patients with high risk of cranial perforation (computed tomographic or skull radiographic examination mandatory)
Over a major joint (possibility of perforation)
Through-and-through bite of cheek

Type of Wound
Punctures (difficult or impossible to irrigate adequately)
Tissue crushing that cannot be debrided (typical of herbivores)
Carnivore bite over vital structure (artery, nerve, joint)

Patient Specific
Older than 50 years
Asplenic
Chronic alcoholic
Altered immune status (chemotherapy, acquired immunodeficiency syndrome [AIDS], immune defect)
Diabetic
Peripheral vascular insufficiency
Chronic corticosteroid therapy
Prosthetic or diseased cardiac valve (consider systemic prophylaxis)
Prosthetic or seriously diseased joint (consider systemic prophylaxis)

Biting Species
Large cat (canine teeth produce deep punctures that can penetrate joints, cranium)
Primates
Pigs (anecdotal evidence only)
Alligators, crocodiles

LOW RISK

Location
Face, scalp, ears, and mouth (all facial wounds should be sutured)
Self-bite of buccal mucosa that does not go through to skin

Type of Wound
Large, clean lacerations that can be thoroughly cleansed (the larger the laceration, the lower the infection rate)
Partial-thickness lacerations and abrasions

Species
Rodents
Quokkas
Bats (although high risk for rabies)

closed primarily. The average time from injury to the first medical treatment was 11 hours in infected wounds and 2 hours in noninfected ones.[4]

Because of high morbidity and permanent residual impairment from hand infections, treating them aggressively is best (see Box 51-4). Hand bite wounds should be irrigated, debrided if possible, and initially left open.[81,211] The hand should be immobilized with a bulky mitten dressing in an elevated position, and the victim should usually be started promptly on intravenous antibiotics. Specialty consultation and follow-up are mandatory for persons with an established infection, and hospitalization should be considered. Persons not hospitalized should be rechecked daily until signs of infection clear. For the patient without initial evidence of infection, 5 to 7 days of splinting and oral antibiotics should suffice if no complications develop. Radiographic examination should be performed on all significantly injured extremities.

Punctures

Punctures may occur from bites, clawings, or gorings. The infection rate is related to difficulty of irrigating properly and degree of contamination, which is highest in bites. Usually, attempts to irrigate narrow punctures simply result in rapid development of tissue edema from infused irrigant solution, which does not cleanse the wound. However, if the wound is large or can be held open wide enough to permit fluid to escape, irrigation is worth the effort. Large goring-type puncture wounds from bison up to 8 to 10 inches deep have a low incidence of infection when closed primarily after irrigation and debridement.[55] For most smaller puncture wounds, one should irrigate or debride as well as possible, suture only if cosmetic or functional considerations require it, and treat as high risk for infection.[81] Delayed primary closure should be used liberally.

Facial and Scalp Wounds

Facial and scalp wounds tend to heal rapidly with little risk of infection; in general, they may be sutured primarily and do not require prophylactic antibiotics. Typical dog bites of the face and neck (including punctures) have an infection rate of only 3%, even when sutured.[4,62,63,214] In general, cosmetic closure of facial wounds is afforded by the lower incidence of infection, and standard of care in most cases is primary closure of an animal bite to the face.[214]

A major risk associated with facial and scalp wound victims of large carnivores is that the teeth can easily perforate the cranium, producing depressed skull fracture, brain laceration, intracranial abscess, or meningitis.[42,162] In young children with such wounds, or with adult victims of large carnivores, computed tomography (CT), or in the absence of CT, skull radiographs, should be routine and may provide evidence of perforation that would mandate immediate neurosurgical consultation and admission to the hospital.

Cultures

Cultures of animal-bite wound surfaces, whether judged quantitatively or qualitatively, are useless as predictors of infection. Some of the pathogens of greatest concern (such as genus *Eikenella*) can take 10 days to grow out in culture, by which time most therapeutic decisions have been made. Other organisms (such as genus *Pasteurella*) are fastidious, hard to identify, and frequently missed by the laboratory technician, who rarely encounters them.[87]

If the victim or wound is very high risk, or if animal bite sepsis is suspected (see Septic Complications, later), obtaining cultures would be prudent to guide subsequent antibiotic therapy. In certain cases, cultures should be sent to reference laboratories, such as those within state health departments or at the Centers

TABLE 51-3. Tetanus Prophylaxis: Toxoid or Immune Globulin?

HISTORY OF IMMUNIZATION (DOSES)	*CLEAN MINOR WOUNDS*		*MAJOR DIRTY WOUNDS*	
	TOXOID*	TIG†	TOXOID	TIG
Unknown	Yes	No	Yes	Yes
None to one	Yes	No	Yes	Yes
Two	Yes	No	Yes	No (unless wound older than 24 hr)
Three or more				
Last booster within 5 years	No	No	No	No
Last booster within 10 years	No	No	Yes	Yes
Last booster more than 10 years ago	Yes	No	Yes	Yes

*Toxoid: Adult, 0.5 mL diphtheria and tetanus toxoids (DT) intramuscularly (IM); child less than 5 years old, 0.5 mL diphtheria, pertussis, and tetanus toxoids (DPT) IM; child older than 5 years, 0.5 mL DT IM.
†Tetanus immune globulin (TIG): 250 to 500 units IM in limb contralateral to toxoid.

for Disease Control and Prevention (CDC) in Atlanta, because reference laboratories have been shown to successfully isolate more pathogens on identical samples sent to both reference and local laboratories.[199]

Prophylactic Antibiotics

Currently, the weight of evidence does not support the use of prophylactic antibiotics for other than high-risk wounds. A recent Cochrane Database Systems review of eight randomized controlled trials found no evidence that the use of prophylactic antibiotics is effective for cat or dog bites, except in the case of bites of the hand.[139] The use of prophylactic antibiotics is advisable for wounds of the hand, because the speed of development, the frequency, the severity, and the complications of hand wound infections can be impressive.[81,211] Persons with other risk factors, particularly prolonged time from injury to treatment, complex wounds with massive crush injury, or medical conditions such as asplenia, diabetes mellitus, vascular insufficiency, or immune deficiency, may benefit from prophylactic antibiotics (see Box 51-5).

In bite wounds, treatment can begin only after wounding and bacterial inoculation. Thus, antibiotics are never truly prophylactic. In major surgery, prophylactic antibiotics are of proven value only in carefully selected high-risk procedures and only if begun before surgery.[208] Several controlled studies of dog bite wounds found no significant benefit to using prophylactic antibiotics in low-risk facial and scalp wounds.[68,214] Other studies recommend the use of prophylactic antibiotics only for high-risk wounds or patients at high risk.[62,63,96,211,214]

If used, prophylactic antibiotics must be administered early. The offending bacteria are already present in the wound when the victim is first seen. Therefore, bite victims needing prophylactic antibiotic treatment should be identified early, preferably during triage on entry to the treatment facility. The victim should receive immediate antibiotics by protocol; the intravenous route is by far the quickest. Although previous practice led to prescription of prophylactic antibiotics for 7 to 10 days, now prophylaxis is needed for only 3 to 5 days after surgery. For animal bites, 5 days of prophylaxis should be more than sufficient for uninfected bites.

Therapy should be tailored to the largest variety of most likely pathogens for a particular type of bite (see Box 51-6). For most terrestrial mammals, the choice of antibiotic is based on experience with human, dog, and cat bites. However, for alligator or crocodile bites, or other wounds incurred in fresh water, antibiotic choice should be directed against *Aeromonas hydrophila*.[104,140]

Tetanus Prophylaxis

Recently, about twice as many cases of human tetanus from animal bites have been seen each year as cases of human rabies.[19,22] The spores of *Clostridium tetani* are ubiquitous in soil, and on the teeth and in the saliva of animals. Therefore, the risk of tetanus may be present from any animal injury that penetrates the skin. Because tetanus is preventable, and many persons still do not receive tetanus immunoprophylaxis according to CDC guidelines, proper emergency prophylaxis against tetanus remains an important, but often underaccomplished, intervention.

Tetanus prophylaxis is administered in standard fashion (Table 51-3). With a clean wound that contains little devitalized tissue and that can be easily irrigated and debrided, a previous full course of immunization plus a booster within the last 10 years is sufficient. With a deep puncture or wound with much devitalized tissue that is difficult to irrigate and debride (predisposing to anaerobic growth), a full series of previous immunizations plus a booster within the last 5 years is sufficient. If there is any uncertainty about the status of the victim's prior immunization, a high-risk wound should prompt intramuscular injection of 250 to 500 units of tetanus human immune globulin (TIG), as well as 0.5 mL of diphtheria-tetanus (DT) toxoid booster vaccine.

Follow-up Care

If the possibility of major or occult trauma has been ruled out, follow-up of animal bites depends on the risk factors present (see Box 51-5) and the patient's response to treatment. For a superficial abrasion, infection is unlikely, and no return visit is needed. For an ordinary low-risk bite wound, one follow-up visit in 2 days to assess any infection is all that is needed. If the patient is very reliable and no sutures have been placed, even one visit is not necessary. Infected wounds dictate much closer follow-up, the frequency depending on the wound's response to treatment and the patient's risk factors. In a high-risk patient, the initial follow-up should be within 24 hours if the patient is not hospitalized.

Box 51-6. Bacteria Isolated from Dog and Cat Bites

AEROBES
Pasteurella multocida
Other *Pasteurella* species
Streptococcus mitis
Streptococcus mutans
Streptococcus pyogenes
Streptococcus sanguis II
Streptococcus intermedius
β-Hemolytic streptococci, group G
β-Hemolytic streptococci, group F
Other *Streptococcus* species
Staphylococcus aureus
Staphylococcus epidermidis
Staphylococcus warneri
Staphylococcus intermedius
Other *Staphylococcus* species
Neisseria weaveri
Neisseria subflava
Other *Neisseria* species
Corynebacterium minutissimum
Other *Corynebacterium* species
Moraxella (Branhamella)
EF-4b
Enterococcus faecalis
Enterococcus avium
Other *Enterococcus* species
Bacillus
Pseudomonas aeruginosa
Other *Pseudomonas* species
Actinomyces
Brevibacterium
EF-4a
Weeksella zoohelcum
Other *Weeksella* species
Klebsiella
Lactobacillus
Citrobacter
Flavobacterium
Micrococcus
Proteus mirabilis
Stenotrophomonas maltophilia
Capnocytophaga
Eikenella corrodens
Flavimonas oryzihabitans
Acinetobacter
Actinobacillus
Alcaligenes
Enterobacter cloacae
Erysipelothrix rhusiopathiae
Riemerella anatipestifer
Rothia dentocariosa
Aeromonas hydrophilia
Pantoea agglomerans
Rhodococcus
Streptomyces

ANAEROBES
Fusobacterium
Bacteroides fragilis
Other *Bacteroides* species
Porphyromonas
Prevotella
Propionibacterium
Peptostreptococcus
Eubacterium
Clostridium sordellii
Veillonella

Data from Talan DA, Citron DM, Abrahamian FM, et al: N Engl J Med 340:85–92, 1999.

COMPLICATIONS OF BITE WOUNDS

Infection

Immense numbers of bacteria inhabit animals' mouths and can be inoculated into a bite wound. The exact pathogens vary depending on the biting species. The major aerobic and anaerobic bacteria are listed in Box 51-6. If inoculated in sufficiently large numbers, these microorganisms can cause localized cellulitis and abscess formation, the most common forms of infection. Wound infection is generally diagnosed on the basis of increasing redness, swelling, and tenderness of the wound margins, eventually progressing to production of pus, cellulitis, lymphangitis, and local lymphadenopathy. Lymphadenitis and lymphangitis, which are much less common, occur as local defenses are overwhelmed. Systemic symptoms of infection are rare and suggest bacteremia or sepsis.

Wound infection from animal bites should be treated in the same way as infection of any other traumatic wound—that is, elevate the wound, immobilize the affected part, remove sutures or staples if present, and provide antibiotic therapy (Box 51-7). Based on the 1999 Emergency Medicine Animal Bite Infection Study Group findings, recommended empiric treatment includes a combined β-lactam antibiotic/β-lactamase inhibitor, a second-generation cephalosporin with anaerobic activity, or combination therapy with either penicillin and a first-generation cephalosporin, or clindamycin and a fluoroquinolone.[199] Additional studies recommend azithromycin, trovafloxacin, or ketolide antibiotics HMR 3004 or HMR 3647 (telithromycin), which show good in vitro activity against unusual aerobic and anaerobic animal pathogens.[87–89,199] In 2002, a study showed that BMS-284756 (garenoxacin, a new des-fluoro(6) quinolone), was very active against 240 aerobic and 180 anaerobic isolates from bite victims. It inhibited 403 of 420 isolates (96%), including those of *Moraxella* species, CDC group EF-4, and *Eikenella corrodens*, all *Pasteurella* species, and *Bergeyella zoohelcum*. *Fusobacterium russii* and 6 of 11 *Fusobacterium nucleatum* isolates of animal bite origin were resistant.[91]

Extremely rare pathogens can cause infection (see Box 51-6). Culture of debrided tissue is the only reliable identification technique, and sensitivity testing may take weeks. In 2002, a 7-year-

Box 51-7. Initial Oral Antibiotics Recommended for Bite Wounds

ORGANISMS KNOWN
Treat according to specific antibiotic sensitivities of cultured organisms.

ORGANISMS UNKNOWN (DOG AND MOST OTHER BITES)
β-Lactam antibiotic and β-lactamase inhibitor
or
Second-generation cephalosporin with anaerobic activity
or
Penicillin and first-generation cephalosporin
or
Clindamycin and fluoroquinolone
or
Azithromycin
or
Trovafloxacin*
or
BMS-284756 (garenoxacin, a des-fluoro(6) quinolone)
or
Experimental ketolide antibiotics: HMR 3004 (RU64004)
or HMR 3647 (RU 66647)

*1999 FDA recommendations restrict use to seriously ill, hospitalized patients.

old girl developed a wound infection as a result of a tiger bite; DNA sequence analysis revealed that one of the causative organisms was a previously undescribed subspecies of *Pasteurella multocida,* which the authors designated *Pasteurella multocida* subsp *tigris* subsp *nov*.[37] These organisms are usually sensitive to ciprofloxacin, cefoxitin, and perhaps rifampin.[166] A diabetic patient developed tenosynovitis caused by *Mycobacterium kansasii* after an accidental bite by his pet dog,[193] and *Bergeyella zoohelcum* has been reported rarely as a fastidious species cultured with difficulty from patients with infected cat bites.[188]

Septic Complications

Bacteremia and sepsis, although theoretical risks with any animal bite pathogen, have so far been reported with only a limited number of species.[6,79,120,162] Clinical manifestations include cellulitis, endocarditis, meningitis, pneumonitis, Waterhouse-Friderichsen syndrome, renal failure, shock, and death. Purpuric lesions are seen in one third of cases and may progress to symmetric peripheral gangrene and amputation. House cats are an increasing source of human plague in the U.S. Southwest. Since the onset of the human immunodeficiency virus (HIV) epidemic, *Rochalimaea* infection (bacillary angiomatosis) has also become more prominent and is closely associated with exposure to cats. Although sepsis after animal bite is reported more often in immunosuppressed patients, as in a case of fatal *Pasteurella dagmatis* peritonitis and septicemia in a patient with cirrhosis,[11,183] there are reports of fatal cases of purpura fulminans with gangrene and sepsis and meningitis caused by *Capnocytophaga canimorsus* in previously healthy patients bitten by a dog.[65,126]

Allergic Reactions

Allergic reactions to animal bites are virtually unheard of. However, up to 11% of laboratory workers have allergic reactions to the dander, hair, and urine of laboratory animals.[215] One case of proven hypersensitivity to rat saliva after a bite has been reported.[215] The patient was subsequently proven to be allergic to the saliva (presumably because of salivary proteins) and not to other portions of the rat. The bite produced lymphangitic swelling and itching that subsided within 24 hours. Two cases of anaphylaxis after dwarf hamster bites have been reported.[151]

Transmission of Systemic Infection

Rabies

Rabies is discussed in detail in Chapter 54, so comments here are limited to brief remarks about epidemiology, assessment of risk in the bite victim, and local wound treatment.

Rabies is a rhabdovirus that occurs in wild and domestic animals. Migrating epidemics alternate with periods of endemicity. It is generally believed that no true reservoir host exists for rabies—that is, no species harbors a latent and nonfatal infection.

The epidemiology of rabies varies widely in different parts of the world. In the United States, western Europe, and Canada, wild animals are by far the main vectors of rabies, accounting for more than 88% of all reported cases in the past 2 decades,[119] but in India, 95% of rabies postexposure prophylaxis treatment is given after bites from stray dogs.[46,47] In recent years, rabies in humans has become an extremely rare disease in the United States, with only two cases occurring in 1997.[119] Since 1980, rabies-infected bats caused 58% of the human cases of rabies diagnosed in the United States.[119] Of the more than 8000 cases of animal rabies reported in the United States in 1997, the terrestrial animals most represented were woodchucks, raccoons, skunks, foxes, and coyotes.[119] Foxes are the primary offenders in Europe, although some countries have eliminated rabies in wild populations by using innovative vaccination programs.[50]

Because of local variations in animal vectors and endemics, consultation with the state or local health department is prudent before a decision is made to initiate antirabies postexposure prophylaxis.[119] Although the number of human cases has declined, about 18,000 people per year in the United States receive postexposure prophylaxis.[80] In the rest of the world, virtually all rabies occurs in dogs. Worldwide, dogs account for 91% of all human rabies cases, cats 2%, other domestic animals 3%, bats 2%, foxes 1%, and all other wild animals only 1%.[50] Each year in India, 25,000 humans die from rabies and a half million receive antirabies vaccine.[197] In Thailand, 50% of human rabies cases occur in children under the age of 15 years.[200] In Africa, Latin America, and most of Asia, dogs are the principal vector, although jackals are also a factor. In South America and Mexico, rabid vampire bats cause occasional human infection. In recent years, disruption of the natural ecology by introduction of humans and domestic animals into the rainforest has produced epidemics of rabies caused by vampire bats. In Israel, wolves and jackals are the chief vectors, and the mongoose prevails in Puerto Rico. In eastern and central Europe, the raccoon dog *(Nyctereutes procynoides)* is an increasingly common vector.[50]

Judgment of the risk of rabies exposure rests on several factors. The incidence of rabies in local species is important;

in the United States, urban dogs and cats, domestic ferrets, rodents, and lagomorphs (rabbits and hares) are at low risk. Description of the animal's behavior is sometimes helpful. This is easily evaluated in wild animals because most tend to shun humans. The urban appearance of a skunk, fox, or bat in broad daylight, showing no fear of human beings, is abnormal and should raise the index of suspicion.

In addition to situations involving animal bites, contact of mucous membranes with rabid saliva or an animal scratch should prompt consideration for rabies postexposure prophylaxis. If a person found in a room with a bat is unable to reliably report the absence of contact that could have resulted in exposure (e.g., an unattended child or a sleeping or mentally incompetent adult), then rabies postexposure prophylaxis should be administered.

Thorough and rapid early treatment of wounds from animals suspected of being rabid is mandatory. Immediately cleanse all bite wounds and scratches with soap and water and a virucidal agent, such as povidone-iodine solution.[191] Evaluate all persons exposed to a possibly rabid animal for rabies immunoprophylaxis; CDC guidelines issued in 1999 recommend that for previously unvaccinated persons, the entire dose of rabies immune globulin, 20 IU/kg body weight, should be infiltrated at the wound site if possible. Three types of rabies vaccines are currently available in the United States: human diploid cell vaccine, rabies vaccine adsorbed, and purified chick embryo cell vaccine. The chosen vaccine is given in 1-mL doses on days 0, 3, 7, 14, and 28 after exposure. Further information on rabies preexposure and postexposure prophylaxis is discussed in Chapter 54.

Other Neurotropic Infections

Although Creutzfeldt-Jakob disease is not caused by bites or wounds, its oral transmission has been reported from the regionally common practice of eating the brains of wild goats, pigs, or squirrels, even when cooked. This disease is characterized by progressive dementia, ataxia, and myoclonus, and it is untreatable. It is caused by a virus also identified in the brains of domestic sheep and mule deer.[114] Mad cow disease is discussed in Chapter 53.

Psychiatric Consequences

Victims of traumatic or life-threatening events may develop post-traumatic stress disorder (PTSD). This syndrome has been recognized anecdotally by the author as a result of wild animal attack, although it is rarely reported in the scientific literature.[64] After physical recovery from an attack, the victim may be plagued by recurrent nightmares and flashbacks of the event and may have an aversion to outdoor travel. Critical incident stress debriefing and post-trauma intervention counseling may be important aspects of care for victims of animal attack.[64] PTSD has recently been described in children who are victims of dog bite.[164]

BITES AND INJURIES FROM DOMESTIC ANIMALS

Dog Bites

Dogs are the only species whose bites have been well studied in large numbers. Although domesticated for at least 12,000 years, dogs retain many of their wild instincts, such as territoriality in a guard dog. Although many people keep dogs to repel burglars, a burglar is rarely a fatal dog-attack victim; the fatal dog-attack victim is a child in 7 of 10 cases.[41]

Dogs under 1 year of age are responsible for the highest incidence of bites.[41] The incidence of biting increases substantially during the summer months. The increased susceptibility of children results from their smaller size, relative inability to defend themselves, interest in animals, and unintentional abuse of animals.

Risk factors for increased biting propensity include male dog sex and unneutered status. Many dogs that seriously wound or kill humans have long histories of aggressive behavior. Strategies that reduce biting risk include education of owners and the public, and selection, training, care, and socialization of the dog (see Box 51-2).

Wounds from dog attack can involve a mixture of biting, clawing, and crushing forces. It is natural to focus treatment on the crushing component of the wound, but the penetrating component may cause the most morbidity. The dog may move and shake its head during the attack, further tearing tissue. Snorting, grunting, or wound manipulation by the biting animal may force air into the tissues. Besides infectious organisms, foreign debris and teeth may be deposited. In a severe attack, the dog may eat tissue and blood or scavenge on an unconscious or intoxicated victim.[95,168] Dog bites to the genitalia have been reported, resulting in rare cases in orchiectomy.[72,93]

Microbiology of Dog and Cat Wounds

A comprehensive study that evaluated 50 patients with dog bites and 57 patients with cat bites identified a median of five bacterial isolates per culture (see Box 51-6).[199] *Pasteurella* species were the most common pathogens in bites of both dogs (50%) and cats (75%).[199] The association of *Pasteurella* with infections of rapid onset was confirmed. *Streptococcus, Staphylococcus, Moraxella, Corynebacterium,* and *Neisseria* were the next most frequent aerobic isolates. *Eikenella corrodens,* usually associated with human bite infections, was found in only one cat and one dog bite wound. *Capnocytophaga* species and *Weeksella zoohelcum,* both of which can cause invasive sepsis, were uncommon in this study and may be opportunistic pathogens. *Fusobacterium, Bacteroides, Porphyromonas,* and *Prevotella* were common anaerobic isolates.[199]

Cat Bites

Domestic cats are becoming an increasing problem in the United States as the population of strays grows, increasing the opportunities for exposure to wild vectors of rabies. Cats now account for 50% of animal control calls, which has led to proposals for leashing and licensing laws for cats.

Cats have a weaker biting force than dogs, but they possess sharp, slender teeth that often produce deep puncture wounds. Their bites are associated with a 15% to 80% infection rate[199] and with a higher incidence of osteomyelitis and septic arthritis than dog bites. Cat bites frequently possess two risk factors for infection: location on the hand and increased depth of puncture. Other risk factors associated with wound infections include an older victim, longer delay to treatment in the emergency department, wound inflicted by a pet rather than a stray, wound care at home, and a more severe wound.[67]

Because the hand and lower extremities are common sites of injury and the wounds may be deep and penetrating, most cat bites are considered high risk. Such wounds should prompt administration of prophylactic antibiotics (see Box 51-7).

Figure 51-1. Pet ferret. (Photo by Luanne Freer.)

Superficial cat bites and scratches elsewhere on the body are not high risk and should receive standard wound care without antibiotic coverage.

Ferret Bites

The two species of ferret in the United States are the common ferret *Mustela putorius furo* (Fig. 51-1), sold as a domestic pet, and the wild black-footed ferret *Mustela nigripes,* which is an endangered species. The pet ferret was domesticated from the wild European polecat and was first introduced into the United States in the late 19th century. Ferrets are kept in increasing numbers as domestic pets, especially by urban apartment dwellers.

Ferrets were bred to hunt and kill small game and rodents in their burrows and are particularly attracted to suckling animals, possibly because of the scent of milk. Their mouths contain 34 teeth, and sharp claws are found on all four feet. Severe injuries caused by ferrets are not common. When they occur, an infant is often the victim and is typically sleeping or in a crib. The face, ears, and nose may be mutilated.[10] Scratches, lacerations, and puncture wounds are seen, and the ferret may chew on a victim (e.g., a baby's ear). The neck is also a common target.[204]

In a comprehensive review of 452 ferret attacks over 10 years, virtually all the unprovoked attacks were on the faces of unattended infants.[58] Most victims were less than 3 years of age and were attacked while sleeping or lying down. Several injuries were severe, and one child died. Bites were usually multiple, and sometimes the ferret's jaws had to be pried open or the ferret had to be killed to secure release.[159]

Ferrets are unusually adept at escaping from cages and enclosures, guaranteeing that they will occasionally be loose and unsupervised in the house, and also that they will escape to the wild, where they will be exposed to endemic rabies. In one study, 4% of biting ferrets were positive for rabies virus.[58] Ferrets are now classified in the same category as cats and dogs regarding rabies pathogenesis and viral shedding patterns, rather than as wild terrestrial carnivores. They may be confined and observed for 10 days rather than being routinely euthanized after biting. An effective rabies vaccine for ferrets (IMRAB-3) has been available since 1990.

Little is known of infection rates or bacteriology in ferret-inflicted wounds, although unusual species such as *Mycobacterium bovis* have been observed.[110] Initial treatment should be as for dog bites.

Domestic Herbivores

Horses and Donkeys

The horse is inclined to both bite and kick, but most horse-related injuries follow a fall during riding activities. More accidents occur per hour of horse riding than of motorcycling.[49] Young females are most often injured by falls,[35] and head injuries cause the majority of deaths.[100] Appropriate helmets and footwear help to reduce the severity of injuries. A recent report of a series of horse- and cow-related injuries found a high correlation of occult craniofacial and torso injuries when field assessment documents the presence of upper extremity injury.[153]

Horse bites are common but not severe injuries. The occlusal surfaces of both lower and upper incisors are flattened. Most male horses possess canines that can be used to grab onto a mare's neck during mounting. The soft tissue contusions inflicted by a horse bite can be severe, but in a series of 24 horse bites, 21 healed uneventfully.[73] Bites can produce significant injury, including muscle rupture and fat necrosis, with no external wound. Horses also have a propensity for biting human nipples, which are at the same height as a horse's mouth.

Horse kicks from the rear legs can be extremely powerful, causing severe blunt trauma, including cardiac rupture[31] and massive pulmonary embolism.[167]

Donkeys also bite. One rural worker was bitten twice in the same year by the same donkey. Death has been reported from fat embolism caused by fractures after a donkey bite.[30]

Cattle

Cattle are usually docile but can inflict a variety of injuries. Serious bites are infrequent, as these animals possess neither upper incisors nor canines. A cow typically weighs 1400 pounds (636 kg), and a bull can exceed 3000 pounds (1363 kg). Accidental treading on the human victim or butting can cause major crush injuries and fractures. Rams have killed farmers by repeated blunt trauma. Farm animals in Wisconsin kill about six people a year.[36] One hospital in rural Wisconsin treated an average of 22 persons a year for horse and cattle injuries, most of them inflicted by a kick or other assault. Domestic cattle and horses are fairly frequently infected with rabies, but because veterinary workers are immunized against the virus, these animals seldom account for human infection.

Cattle horn injuries (or gorings) present typical and unique damage patterns (Fig. 51-2). The horns are used in an inward hooking motion to butt and fling the victim, or the horn tip can be used for goring. Goring injuries seen in bullfighting typically involve the perineum and thigh, and they tend to be deep and sometimes fatal. By contrast, bull horn injuries from domestic cattle involve a sweeping arc at the level of the bull's head, which is at the level of the human abdomen. The semicircular motion of the horn often produces a relatively superficial laceration, leaving deeper structures of the abdomen intact. In one series of 29 cases in which the peritoneum was breached, usually producing prolapse of bowel or omentum, 27 laparotomies demonstrated no additional injuries. In only a few cases was the

Figure 51-2. Domestic bull goring victim (Photo by Luanne Freer.)

bowel itself damaged.[187] The rate of wound infection in this series was high (54%), probably because of delayed treatment.

Bulls should be approached only with a protective device (e.g., a heavy stick) and a preplanned exit. A ring in the bull's nose gives a victim something to hang onto besides the horns and a way to yank the bull's nose up, which may stop the attack. Dehorning the bull will not eliminate the potential for crushing. If struck by a bull or cow, the victim should not attempt to stand, as this will provoke being thrown to the ground again, but should try to crawl to safety. Children must be educated about the risks of large animals and kept away from them whenever possible.

Camels

In regions where camels are used for domestic or agricultural purposes, bite injuries are quite common.[125] Although herbivores are usually docile, camels are much more likely to bite in the winter rutting season, and bite fatalities have been reported. Camels have 34 teeth, including large backward-inclined upper canines, or tushes. The lower tushes are placed relatively forward, and the resultant mouth grip is very effective. Jerking movements of the head result in severe tissue damage and limb avulsion, and rarely they can break the victim's neck. The forearm is often injured, and bites to the face are well documented.[154,183,190] Virtually all bites are single.[180] Injury or death can also occur if the camel presses the victim to the ground and crushes, after gripping the person in its jaws.[125]

Microbiology of Domestic Herbivore Bites

Little is known about the incidence of wound infection after an herbivore bite, but infection after camel bites may be as common as 86%.[125] Species of *Actinobacillus lignieresii* and *Actinobacillus suis,* as well as *Pasteurella multocida,* have been isolated from infected horse and sheep bites.[26,161] All are common organisms in the mouths of herbivores. Most domestic herbivores carry *Pasteurella,* and most are given frequent and regular doses of different antibiotics, especially in their feed, leading to antibiotic resistance of bite wound organisms.[169] *Pasteurella caballi* has been isolated from a horse bite wound.[75] *Staphylococcus hyicus* subsp *hyicus,* a well-known cause of disease in many animals, has been reported as a human wound pathogen after a donkey bite.[158]

Bites from horses, donkeys, cattle, sheep, camels, deer, and most other herbivores are treated with the same antibiotics as bites from dogs, cats, and humans (see Box 51-7).

Domestic Swine

Bites from domestic swine are fairly rare, but when these animals attack they can be aggressive, inflicting deep goring or bite injuries, often on the posterior thigh as the pig approaches from behind.[20,152]

Pigs have a reputation for inflicting bites that have a high risk for infection, and care should include thorough wound exploration and debridement. The usual wide range of bacterial pathogens is reported, including *Pasteurella aerogenes, Pasteurella multocida, Bacteroides, Proteus, Actinobacillus suis,*[74] and hemolytic streptococci, including *Streptococcus milleri.*[20] Unusual gram-negative bacteria, such as *Flavobacterium* group IIb, have been isolated, as has *Mycoplasma.*[90,143] Both of these organisms are resistant to amoxicillin-clavulanate, so the addition of ciprofloxacin is recommended as prophylaxis for a serious pig bite wound.[143]

Domestic Birds

Birds may be kept as domestic pets or on farms and can be responsible for serious injuries. On farms, rooster attacks, often by male fowl defending their territory, are well documented. Children, especially infants, are particularly vulnerable to attack. Rooster injuries have included serious clawing to the face and a fractured skull.[171] In one report, a rooster spur was retained in a wound, resulting in a chronic infection.[59] Septic arthritis was reported in a child after a bite from a domestic fowl.[107]

BITES AND INJURIES FROM WILD ANIMALS

The Wild (Big) Cats

Adult cats have 30 permanent teeth, arranged in rows of 16 upper and 14 lower. The upper teeth overlap the lower, resulting in an overbite. This helps the animal lock its teeth into prey and exert twisting and tearing forces. The feline bite is much shorter and more rounded than that of a dog.

Big cats typically attack from behind, biting the neck and occiput of their prey and attempting to maneuver their canine teeth between the victim's cervical vertebrae and into the spinal cord.[53,177,210] Proprioceptors in the cat's tooth allow it to detect when it has encountered bone. The goal of rapidly paralyzing the prey is also accomplished by a violent shake of the cat's head, which fractures the cervical spine. In a study of fatalities from jaguar attack, 77% of victims were bitten on the nape of the neck and half of the bites were made to the base of the skull.[53] In 20% of cases, the killing bite was to the head, with at least one canine piercing the skull or ear canal. Cheetahs prefer to attack the throat of their prey, crushing the larynx and strangling the victim, a method also used by lions and leopards.[132] Big cats also claw their prey, producing deep parallel incised wounds. Several victims have died of exsanguination without evidence of strangulation or cervical spine injury.[53,177] Because of the growing propensity for people of developed

countries to keep exotic animals as pets (or raise them for profit for hunting purposes), injuries by big cats can occur anywhere.

Wound care is the same as for other species, with special attention to evaluation for major internal injury. In particular, observe for penetration of deep structures of the cranium and neck, and rule out injuries of the cervical spine and deep cervical vessels (see Box 51-4).[53] One victim with an apparently trivial puncture wound after a bite to the neck from a pet cougar was discharged from the emergency department.[115] Within hours her voice was hoarse, and on return, she recalled that the cougar had shaken her in its jaws when it bit her, and air was found in the prevertebral and retropharyngeal spaces on radiographic examination.

Like domestic cats, big cats usually carry *Pasteurella* as normal flora, and because of the deep penetration of their large teeth, *Pasteurella* septic arthritis, meningitis, and other serious deep infections can occur.[84]

Cat-scratch disease is an uncommon disease; 90% of cases are caused by scratches from domestic cats, and it has been reported in a big-cat attack victim.[60,115] The average incubation period is 3 to 10 days. The characteristic feature is regional lymphadenitis, usually involving lymph nodes of the arm or leg. In most cases, clinical diagnosis is based on finding three of the following four features:[60]

- Single or regional lymphadenopathy without obvious signs of cutaneous or throat infection
- Contact with a cat (usually an immature one)
- Detection of an inoculation site
- A positive skin test for cat-scratch disease

Detailed information on diagnosis and treatment of this disease is found in Chapter 53.

Tigers

Adult tigers are so powerful that the human victim is often killed instantly. It is not unusual for a limb to be severed with a single bite.[71,127] A swiping blow to the human head can cause a skull fracture.[170] Like many big cats, tigers typically strike without warning from behind, biting the head and neck and often shaking their heads violently to sever the victim's spinal cord.[117]

Big cats are a major threat to the lives of humans in the cats' native regions.[138] Although the number of tigers in the world is dwindling rapidly, they are still the number one animal killer of humans (see Table 51-2). Nonetheless, man killing almost invariably results from stress (wounds or old age) or lack of natural prey and habitat that forces the animal to prey on humans. A tiger subsisting solely on human meat would have to kill approximately 60 adults a year, and documented cases in selected regions have approached this rate over periods of up to 8 years. Man-eating tigers in India between 1906 and 1941 ate an estimated 125 persons each, and one killed 436 persons. However, unlike lions, tigers are not thought to become exclusive man eaters. Some tigers have become opportunistic man eaters in lieu of plentiful natural prey, and tiger biologists hypothesize that these animals have become unafraid of man.[138]

Over the last 5 centuries, an estimated 1 million people have been eaten by tigers. In the 19th century, the tigers' toll in India averaged 2000 victims per year. From 1930 to 1940, the annual number never dropped below 1300. In the late 1940s, this rate dropped to about 800 per year, where it remains. At the same time, approximately 17,000 people per year are killed in India by other wild beasts, which include nonmammalian species.

Lions

Despite their appearance and reputation, lions are not as greatly feared or respected by experienced hunters as are tigers. Lions are primarily scavengers, making fewer original kills than hyenas. A protected population of 250 Asiatic lions in India has attacked 193 humans, killing 28 between 1977 and 1991; biologists credit drought and lion baiting for tourist shows for this increasing carnage.[178]

A threatening gesture or shout may repel a lion, although a lioness guarding her cubs is more likely to attack. Experienced hunters find that when a charging lion is faced head on and confronted, it will often turn tail and run. An intended victim who flees is most likely to be attacked. Walking unarmed in lion country is usually safe; most hunters who succumb to lions are killed by wounded or sorely provoked animals, usually in dense brush (Fig. 51-3). Many who survive the initial mauling die later of infection. Persons who have survived attacks state that it is unwise to struggle and that the cat should be allowed to chew on an arm or other extremity. With luck, it will lose interest and leave.

A

B

Figure 51-3. **A,** African lion. **B,** Lion attack victim. This African victim was rescued by bystanders as his head was in the lion's mouth. His only injury was a degloving scalp laceration. (**A** photo by Cary Breidenthal; **B** photo by Harold P. Adolph.)

Man-eating lions exist, as noted recently in South Africa, where illegal immigrants from Mozambique attempt to cross the border at night through Kruger National Park. In direct proportion to the number of immigrants on foot, the number of humans maimed or killed by lions has increased. Rangers suggest that humans on foot are an easier food source than normal prey for old or sickened lions.[146] Lions are estimated to eat 300 to 500 Africans a year and rank second to tigers among man eaters. Conversion to man eating has been blamed on drought, famine, and human epidemics in which large numbers of corpses are abandoned in the bush. Lions that become man eaters and subsist exclusively on human flesh need approximately 40 victims a year to stay alive. They usually kill instantly with one bite to the head or neck or with a swipe of the paw, which can break an ox's neck. Lions have tremendous strength and can easily carry a human victim for a mile without rest.

Although most lions in captivity become passive and dull, circus and zoo lions periodically kill attendants. Many such deaths have occurred when keepers accidentally have backed into or trod on an animal.

Leopards

Most leopard attacks are provoked by wounds or by a dog attack. When wounded, trapped, or cornered, a leopard is unpredictable and ruthless, attacking the first person within striking distance. Unmolested and in normal health, the leopard is a shy, nervous animal with marked fear of humans. Unlike a lion or tiger, the leopard relies on fast claw work and biting. Like the jaguar, the leopard may go for the neck (in an effort to sever the spinal cord) or attempt disemboweling by raking at the victim's abdomen with the claws. The leopard seems inclined to retreat when much resistance is offered, even in encounters with baboons. There is a documented report of a man armed only with a screwdriver fighting and killing an attacking leopard.[122] Before the era of antibiotics, three fourths of people mauled by leopards died from wound infection, but modern morbidity is estimated to be less than 10%.[18,52]

Mauling by leopards is much more common than killing; estimated casualties are 400 per year, mostly in Africa. The leopard does not often turn into a man eater; when it does, it attacks mainly children or sick adults. In the state of Bihar in India, leopards ate 300 people in 1959 and 1960.[52] The man-eating leopard of Rudraprayag in India killed 150 people between 1918 and 1926. Becoming increasingly bold, it eventually took its prey by banging down doors, leaping through windows, or clawing its way through the walls of mud huts. Like man-eating lions, man-eating leopards completely change their normal hunting patterns when the prey becomes exclusively human.

Cougars

The North American cougar (mountain lion or puma) is a clever, basically shy cat. It is the most widely distributed large animal on the American continent. Recently, cougars are encroaching with increasing frequency into populated areas of the western United States, probably because of human expansion into the wilderness and an increased population of protected cougars.[116] The combined cougar population of California, Colorado, and Idaho is estimated at 9000 to 12,000, and humans live, exercise, or picnic in cougar country with increasing frequency.[196] Thus, modern suburban dwellers (who typically are ignorant of wild animal behavior) are now in regular close contact with cougars in their homes and parks—whether they know it or not.

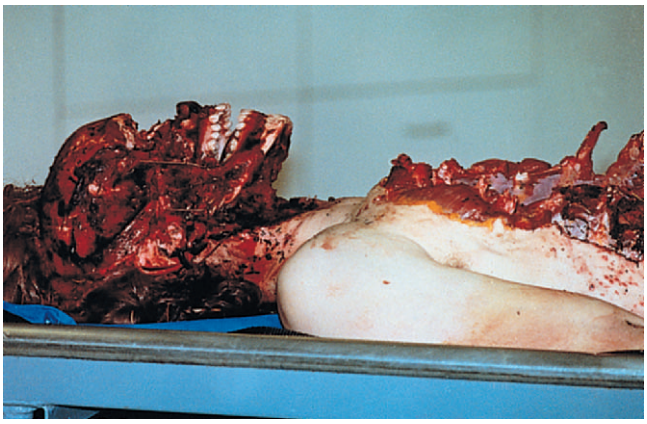

Figure 51-4. Victim of cougar predation and fatal mauling. (Photo by Ben Galloway.)

In a 2-decade period in the United States, 31 cougar attacks on humans were documented, with five fatalities.[25] More people have been attacked since 1975 than over the entire previous century.[25,132,196] Most attacks occurred in the western United States and Canada. Throughout the United States, cougars ranked 16th as an animal-related cause of death in recent years, just behind jellyfish and goats.[174] In California, no attacks occurred from 1925 until 1986, when two children were attacked in a regional park in southern California.[115] In the 1990s, several joggers were killed by cougars in Colorado and California and a biker was killed in 2004 in California. Victims jogging and biking may evoke a predatory response. Young animals that are forced out by adults and must find their own territory are the most frequent attackers of humans.[116] Children are the preferred victims (86% of fatalities are children).[109,113] There has been only one alleged report of a cougar as a primary man eater, but cougars have sometimes partially eaten victims of their attacks (Fig. 51-4).[23,54,177]

The cougar hunts like a domestic cat: crouching, slinking, sprinting, pouncing, and then breaking the prey's neck. The types of injuries to the neck are similar to those described for lions and tigers. Like many potentially dangerous wild animals, the cougar can often be scared off by the victim's aggressive behavior, even after the attack has begun.[102] In 2002, a man fought off and killed an attacking cougar with a 7.5 cm (3 inch) pocket knife.[207]

Bobcats

The North American bobcat *(Felis rufus),* found in southwestern U.S. deserts, probably got its name from its short tail, which is only 6 or 7 inches (2.75 cm) long. The bobcat has long legs and large paws, can weigh up to 30 pounds (13 kg), and is equipped to kill animals as large as a deer.

Bobcat attacks are extremely rare. A hunter who sustained injury to the eye and ocular adnexa requiring surgical repair has been reported.[103]

Rodents

Rodents do not tend to bite unless severely provoked. Their bites are usually small and do not cause much disability. The

exact number of rodents in the world is unknown, but they probably number in the hundreds of millions, with 1500 species of laboratory and pet rodents. Despite these numbers, they account for only 1.7% to 10% of animal bites brought to medical attention.[156] Such bites are infrequent, seldom cause any problems for the victims, and often occur among lower socioeconomic groups without good access to medical attention. The other human populations at risk are owners of pet rodents and laboratory workers who handle rodents used in research. Insufficient data exist to determine any difference in outcome between wild and domestic rat bites.

Rats, Mice, and Other Small Rodents

Rat bites show an infection rate of 2% to 10%, even without treatment, and the rate is usually on the low end of this spectrum.[156] Other than bites inflicted by laboratory animals, the vast majority occur in poverty areas while the victim is sleeping and involve the face and neck, usually in infants or physically or mentally disabled adults.[155,156] There were 415 reported rat bites in New York City between 1986 and 1994.[48] Sometimes these can be severe: a week-old infant was bitten around the eyes by a rat, resulting in perforations of the globe, an estimated vascular loss of 55% of red blood cell mass (with an initial hematocrit value of 20%), and eventual blindness.[147] Despite the rat's reputation for spreading disease, however, infection did not occur. Similar bites in infants have resulted in loss of more than three fourths of the eyelids.[219]

The bacteriology of a rat bite is similar to that of other animal species. The various systemic diseases transmitted by rats are discussed in Chapter 53. Sporotrichosis has been reported; this widely distributed saprophyte is found on various plants, in the soil, and on many animals.[83]

Although rodents occasionally become infected with rabies, they seldom secrete this virus in saliva; therefore, they inflict extremely low-risk bites for transmission of the disease. However, local epidemics can occur, as documented in the 1980s among rodents and lagomorphs on the U.S. East Coast.[145] In that epidemic, woodchucks constituted 80% of all rabid animals; the remainder were squirrels, beavers, rabbits (lagomorphs, not rodents), and one rat. Some of these rabid animals were very aggressive: a woodchuck attacked and knocked down an older adult woman in her garden, biting her repeatedly. Rabies was isolated from the buccal cavity of the woodchuck. A reasonable current recommendation is that a biting wild rat in the United States should be caught and examined for rabies, and rabies immunoprophylaxis should be initiated only if the rabies test is positive. Outside the United States and Canada, rabies immunoprophylaxis is probably appropriate for bites inflicted by uncaptured rodents.[156]

A case has been reported of a cowpox virus–like infection transmitted by a probable rat bite.[214] Rodents are believed to be the natural reservoir for cowpox virus.

Rat-bite fever is an acute illness caused by *Streptobacillus moniliformis* or *Spirillum minus,* which are part of the normal oral flora of rodents, including squirrels. It may also result from bites by wild and domestic carnivores, such as weasels, dogs, cats, and pigs, which may have become infected when hunting rats and mice.[185] Carrier rates among wild rats vary from 50% to 100%.[209] Fewer than 100 cases have been reported in the North American literature; rat-bite fever is not a reportable disease in the United States.[209] Although relatively rare, cases can occur in any setting and can easily be fatal, particularly when the proper diagnosis is not suspected.[61,182,209] Further information is found in Chapter 53.

Plague is caused by the bacterium *Yersinia pestis*. Wild plague is endemic in many parts of the world, chiefly among rats, mice, moles, marmots, squirrels, hares, cats, and mongooses. One of the larger areas of endemic plague is the western United States (362 cases reported in 50 years), where voles, field mice, ground squirrels, prairie dogs, and pack rats carry the infection. The infection is usually transmitted to humans by the bite of arthropods that infest infected animals. Handling infected animals allows *Yersinia pestis* to enter cuts and abrasions, as seen in veterinarians and in hunters who skin and clean infected rabbits.[39] Transmission by bite or scratch has never been reported. The disease is mentioned here because of its historical significance and the frequency of occurrence in wild animals. Further information is found in Chapter 53.

Tularemia encompasses various syndromes caused by *Francisella tularensis*. This bacterium normally parasitizes about 100 different mammals and arthropods, most commonly cottontail rabbits, rodents, hares, moles, beavers, muskrats, squirrels, rats, and mice. The primary mode of transmission to humans is via a bloodsucking arthropod, such as a tick, or by skin or eye inoculation resulting from skinning, dressing, or handling diseased animals. Other routes of infection include ingestion of water contaminated by urine or feces, and inhalation of dust. Infections after bites or scratches from dogs, cats, skunks, coyotes, foxes, and hogs have been reported, although these are rare.[173] The disease is an occupational hazard of hunters, butchers, cooks, campers, and laboratory technicians. Humans are quite susceptible, and although tularemia was removed from the national notifiable disease list in 1995, 105 cases were reported in 1997 in the United States.[40] More information is found in Chapter 53.

Porcupines

Porcupines (a species of rodent) are virtually never reported to bite, but their quills may become embedded in the skin of humans. Because of the structure of quills, they not only embed themselves and are extremely difficult to extract, but they can migrate as much as 10 inches (24 cm) under the skin. The average porcupine has 30,000 quills, which range from less than an inch (2 cm) to 4 inches (12 cm) in length. The quills are barbed and their cores are spongy, so if they are not removed immediately, they absorb body fluid and expand, causing the barbs to flare even further outward. Thus, each movement of the victim's body or muscles helps a quill work its way in deeper. Allegedly, such migration has led to injury to internal organs and death in humans. Infection seldom results because the quills have mild antiseptic properties, presumably to protect the porcupines, which sometimes impale themselves.

No medical reports are available of appropriate human treatment or complications of porcupine quill injuries, though anecdotal reports of human tetanus from porcupine quill puncture arise from Africa.[2] Most veterinarians remove quills from animals by simple extraction; the same technique is probably sufficient in humans.

Omniverous Mammals

Bears

Bears (Fig. 51-5) are among the most feared and powerful predators on Earth, yet the incidence of bear attacks on humans is

Figure 51-5. Alaskan brown bears spar for fishing territory. (Photo by Luanne Freer.)

small when compared with that of the other animals discussed in this chapter. However, the scientific literature on bear behavior and human interactions is far more plentiful than that existing on, for instance, tiger or alligator attacks, and a separate chapter in this text is devoted to bears (see Chapter 52).

Bats

Vampire bats are a vector of rabies in Central and South America; 177 cases were reported from 1980 to 1990, with 27 of those cases in Brazil.[22,157] Sometimes, small epidemics of bites occur in isolated villages in the jungle, as in one outbreak of 26 bat bites in Honoropois, Brazil; all victims were treated with rabies prophylaxis, and no clinical human rabies was reported.[22] Such clusters of attacks may be triggered by human destruction of wild or domestic hosts (such as pigs) of the bats.[136] Vampire bats feed at night on animal blood, including that of humans, by making an incision in the skin to lap up the blood from the victims' earlobes, forehead, fingers, or toes. One bat can eat a maximum of 1 ounce (30 mL) of blood per night, which is clearly not enough to cause death. However, a cave of 1000 bats needs 15 gallons (60 L) of blood each night, which amounts to more than 5750 gallons per year.[51] Protection against vampire bats is effectively provided by mosquito nets.

Insectivorous bats, such as free-tailed bats, are noteworthy for the nearly undetectable bite wounds they leave on their victims.[86] All bats should be considered a high risk for rabies, and exposure—even to sick or "pet" bats that have been in captivity—should be avoided. The known pathogenesis of rabies and available data suggest that all or nearly all cases of human rabies attributable to bats involved transmission by bat bites that were minimized or unrecognized by the patients. Nonbite transmission of rabies is very rare, and aerosol transmission has never been well documented in the natural environment.[86] Some situations mandating rabies prophylaxis are bizarre and reveal more about human nature than about rabies or bats. For example, rabies prophylaxis was initiated after a patient dunked a dead bat in his beer, chewed on the bat's ear, and then drank the beer.[8] In a similar case, prophylaxis was needed when a miner swallowed a live bat on a bet.[9] Most bats have small teeth that often cannot penetrate human skin, so the risk of bacterial wound infection is low.

Primates

Monkeys and other primates inflict vicious bites that virtually always become infected, despite use of prophylactic antibiotics.[92] Although in developed countries this is largely an issue for laboratory workers, in tropical underdeveloped countries, large apes (such as baboons) are not only common but often aggressive. Weighing up to 90 pounds (40 kg), a large baboon can be dangerous and lethal. These animals have frequent contact with humans and lose most fear of them. There have been numerous recent reports of packs of wild monkeys driven out of the jungle by hunger and attacking humans who get in the way of food sources.[176,179,195]

Monkeys often bite hands and have been known to amputate parts of fingers. A literature review revealed 132 cases of simian bites in which *Bacteroides*, *Fusobacterium* species, and *Eikenella corrodens* were isolated from some of the wounds.[92] Three victims of simian bites with infected wounds grew diverse bacteria, including β-hemolytic streptococci, enterococci, *Staphylococcus epidermidis*, and Enterobacteriaceae.[92] Simian bites should be considered relatively high risk and treated as if they were human bites.

Old World macaque monkeys (rhesus macaque, cynomolgus, and other Asiatic macaque monkeys) are often infected with simian herpes virus (B virus); transmission to humans is rare but the risk is real, especially for animal control and laboratory workers.[70,150] As with rabies, local wound treatment may be important; of 61 persons bitten by probably infectious monkeys and who received wound cleansing with cetrimide and iodine solution, none became infected. See Chapter 53 for additional information.

The wild gorilla, despite its reputation and appearance, is shy and avoids humans. Although it may charge in defense, it seldom attacks and can be easily confronted and forced to retreat. When a gorilla attacks, it takes one bite and runs. In Africa, gorillas are responsible for two or three attacks per year, none of which are fatal and few of which are severe. Chimpanzees occasionally attack humans, usually only if provoked or cornered. Rare instances of chimpanzees eating children and women have been reported. The baboon is responsible for one to two attacks per year, almost all in South Africa; these are usually by pets. Occasionally, man eating has been reported. The incidence of hunting and meat eating by these animals has increased over the last century, perhaps paralleling the evolution of humans into hunters and meat eaters.[52]

Coyotes, Dingoes, Wolves, and Hyenas

The coyote *(Canis latrans)* not only has survived the onslaught of civilization in the United States but has thrived and multiplied. Perhaps as a result, more and more coyote attacks on people have been reported, even in urban areas such as Los Angeles and Cape Cod.[15,38,106] Occasionally, these animals are rabid, but most are not. Some attacks (especially on small children) are fatal.[112] Recently, there has been an increase in reports of attacks in national parks by animals captured and found to be disease free.[38,116] The bite of a coyote should be treated like a dog bite regarding antibiotic and closure issues; if the animal cannot be captured and examined, rabies prophylaxis should be undertaken. Coyotes have been identified as the reservoir for a human pathogen *Bartonella vinsonii* subsp *berkhoffii*.[43]

In Australia in a 5-year period, dingoes habituated to people have accounted for 224 attacks on humans requiring medical treatment, and for several fatal attacks on children.[172]

Figure 51-6. Yellowstone wolves. (Photo by Rick McIntyre.)

There are an estimated 70,000 wolves in North America. Significant injuries are rare but have been reported. Five wolf attacks on humans occurred within a 12-year period in Algonquin Provincial Park in Ontario, and a kayaker was pulled from his sleeping bag by wolves in British Columbia.[130] Wildlife experts suggest that these attacking wolves were habituated to humans and human food sources, and that most unhabituated wolves are traditionally timid. However, throughout Europe and Asia, the wolf has a well-documented history of cunning behavior, pack attacks, and human killing.[130] European wolves tend to hunt in packs and attack women and children. Rabid wolves are seen; a substantial number of attacks by rabid wolves in Iran in a 10-year period provided the clinical population on which the human diploid cell vaccine for rabies was tested.[17] The reintroduction of wolves to wild habitat in the Yellowstone ecosystem and Idaho in 1995 has resulted in the successful proliferation of many new wolves (Fig. 51-6). However, there has not yet been a negative human interaction since their release.[94] A 22-year-old man was killed by a wolf-pack in Saskatchewan in November 2005.[72a]

Comparison of victims of fatal attacks by domestic dogs and wild wolf packs reveals distinct differences in bite-mark patterns; the neck and face of a domestic dog attack victim were primary sites of attack, whereas a feral wolf pack victim was spared damage to the neck but had facial tissue destroyed postmortem. Most punctures are found on the ventral aspect of the domestic victim and the dorsal aspect of the feral victim. It is speculated that most wounds are attributable to dominant animals of both packs. Differences in bite-mark patterns may well have been caused in part by differences in genetics, training, breeding, socialization, and impetus of attack between wolves and dogs.[216]

Two other canines that traditionally hunt in packs are the cape hunting dog of Africa and the Indian "devil dog." Although both are feared in their respective environments, they are not deliberate attackers of humans.

Hyenas have tremendously strong jaws and can leave teeth marks in forged steel. The hyena frequently attacks humans in Africa. In certain areas, Africans leave the dead or dying in the bush for predators (hyenas) to eat, which accustoms the animals to the taste of human meat. Hyenas forage around campsites and villages and are wary of awake people. During the summer months when Africans sleep outside their huts, many are assaulted with one clean, massive bite that removes the face or the entire head. Young children have been dragged from their huts while sleeping when a family member leaves briefly without latching the door, and it is common for the hyena to injure or amputate the thumb of a sleeping victim as it drags the victim (by the thumb) to a more convenient place for consumption.[2] Campers are also frequently bitten on the face or limbs while they sleep at night, particularly if they have left food nearby. They usually survive but are massively disfigured. In some parts of Africa, the hyena is a more consistent man eater than the leopard or lion.

Skunks, Foxes, and Raccoons

Skunks bite readily when captured and are frequent carriers of rabies. No data exist concerning the likelihood of other wound infections after skunk bite. In one series, only 21 bites were reported for the entire United States for a 10-year period.[66]

A skunk's most frequent means of defense is spraying the secretions of its anal sac. A skunk ready to spray directs its hindquarters to the enemy, feet firmly planted and tail straight in the air, often stamping the front feet in warning. The spray is accurate to 13 feet and, contrary to popular belief, can be discharged when the animal is lifted by the tail.

Skunk musk causes skin irritation, keratoconjunctivitis, temporary blindness, nausea, and occasionally convulsions and loss of consciousness.[82] The chief component of the musk is butyl mercaptan. This can be neutralized by strong oxidizing agents, such as sodium hypochlorite in a 5.25% solution (household bleach), further diluted 1:5 or 1:10 in water. The chlorine forms odorless sulfate or sulfone compounds by oxidizing the mercaptan and breaking the sulfur free from the carbon chain. This solution can be cleansed with tincture of green soap, followed by a dilute bleach rinse. Tomato-juice shampoo has been advocated for deodorizing hair, which can be washed and mildly bleached or cropped short.

Most human attacks by foxes are by rabid animals. Fox bites have caused eyelid lacerations in children sleeping in tents and leg punctures in adults.[118,198,206] One child died of rabies from fox bites despite appropriate postexposure prophylaxis.[198]

Raccoons are the definitive host of *Baylisascaris procyonis*, a zoonotic nematode parasite that may cause larva migrans syndromes in humans.[220] In the United States, large raccoon rabies epizootics in New England and the Middle Atlantic states are spreading. Most rabies-positive animals are taken from near private homes.[189,212] Raccoons account for more than 50% of all animal rabies in the United States.

River Otters

In 2004, a family of three was attacked while swimming in calm river water near Belden, California. A 12-year-old boy was suddenly attacked both above and below the water by a river otter,

Figure 51-7. Victim of river otter attack (Photo courtesy Jill Hanna.)

Figure 51-8. African water buffalo with calf. (Photo by Cary Breidenthal.)

and was bitten and scratched over much of his body. The otter then attacked the child's mother and father when they arrived to help, and the attack was described as "vicious and unrelenting" despite many attempts by the three to hit and push the animal away. State game officials were unable to locate or capture the animal after the attack. The child's injuries required plastic surgery for facial wounds, as well as suturing of numerous arm, leg, and trunk lacerations and punctures. The victims healed without incident after prophylactic treatment with amoxicillin-clavulanate and rabies postexposure prophylaxis (Fig. 51-7).[101]

Twenty-four rabies cases in river otters have been recorded at the CDC, and wildlife experts suppose that because of low population densities and semi-aquatic existence, otters probably have limited opportunities to contract diseases from other species. Otters travel along rivers while denning, allowing contact with raccoons, which carry a rabies strain that is enzootic in raccoon populations.[184]

Herbivorous Mammals

Wild Swine

Wild pigs are more likely to inflict injury than their domestic counterparts. With a population of over half a million roaming the French countryside, wild pigs cause crop loss and occasionally gore and bite humans.[134] In the United States, feral pigs, some weighing up to 400 pounds (180 kg) with 4 inch sharp tusks and prolific breeding qualities (litters of up to 19 have been reported), are experiencing explosive population growth. Texas has over 1.5 million wild swine, and motor vehicle collisions as well as human attacks are increasing.[33] Swine wounds should be treated as having a high risk for infection, warranting broad-spectrum prophylactic antibiotics (perhaps parenterally) and close follow-up if the victim is not admitted to a hospital.

African Buffalo

The African buffalo is a threat chiefly when provoked, but it accounts for many deaths and maimings. Left alone, it does not attack; however, when shot or cornered, it charges, is difficult to avoid or stop, and can hook the victim 10 feet into the air with its horns (Fig. 51-8). Buffalo that charge humans are usually old, solitary bulls that have left the safety of the large herds, most often because of wounds from poachers' snares or spears, or from lion attacks. The buffalo is also wily and intelligent; wounded buffalo may lie in wait for trackers or may double around and come up behind them on the trail, often with fatal consequences for the humans. The buffalo's hatred of hunters is legendary in Africa: one hunter was treed but could not get his feet high enough to keep them clear of the animal. The buffalo repeatedly hooked the man's feet with his horns, eventually cutting them to ribbons so that he bled to death, still hanging in the tree. In another anecdotal series, a single buffalo injured several humans in one day; all of the wounds were impaling injuries through the anus.[2]

Once the victim is prostrate, the buffalo gores into the ground with its horns and the heavy horny boss across its forehead and then whips its head from side to side, disemboweling the victim with the sharp horn tips. Such injuries are estimated to be responsible for 20 to 100 deaths per year, mostly in Africa. The horns are always covered with mud, so goring wounds may be heavily contaminated. However, victims who do not have major traumatic injuries generally do well.[186]

American Bison

Brought back from the brink of extinction, a free-ranging herd of more than 3000 North American bison (American buffalo) now lives on land in and around Yellowstone National Park and is responsible for an average of three tourist injuries per year. Fifty-six injuries and three deaths were documented from 1975 to 1993 in Yellowstone as a result of bison–human interaction.[55,56] Despite warnings to avoid approaching these animals any closer than 25 feet, most attacks involve close human approaches to obtain photographs. The mechanism of injury in bison attack is usually penetrating injury, with punctures from goring by horns and blunt injury caused by being tossed in the air and falling or being butted by the animal's massive head. Goring injuries most frequently involve the buttock or posterior thigh as the victim turns away from the bison to flee.[55,56] Gored abdomens and evisceration have been reported. Despite the inevitable contamination of these deep punctures, wound

Figure 51-9. Wound from bison goring. (Photo by Karen Hansen.)

Figure 51-10. African elephant bluff-charges the photographer. (Photo by Cary Breidenthal.)

infection is rare if careful operative irrigation and debridement with closure are combined with broad-spectrum prophylactic antibiotics. Cephalosporins were used in most cases in one series of reported injuries (Fig. 51-9).[55]

Elephant

The elephant can be one of the most dangerous wild animals and was until recently perhaps the greatest killer of hunters. The annual death toll in central Africa was probably between 200 and 500.[52] In defense of the elephant, most injuries occur when humans accidentally approach elephants too closely, which the animals interpret as a threat. However, elephants turn rogue from time to time, deliberately attacking and killing humans (Fig. 51-10). The incidence of such deaths might decrease as the world becomes progressively less wild, but as humans and elephants compete for the same space and humans despoil and devastate the forests, it is increasing.[121] In India, home of most of the world's 40,000 Indian elephants, approximately 4000 people have been gored or crushed by elephants in the past 20 years. In Sri Lanka, such encounters force some families to sleep in trees, and at least four people a year are killed by elephants in one area with a human population of only 25,000.[12]

Elephants kill by trampling, goring with the tusks, or striking or throwing with the trunk. Once victims have been run over or skewered with the tusks, the elephant kneels on them and crushes them. Elephants have been known to use weapons in their attacks; a villager who retreated safely up a baobab tree was hit by a tree branch the bull elephant picked up in its trunk and used as a club. Elephants frequently rip the victim's body apart and scatter the pieces, later covering them with grass and branches. Another elephant tactic is to toss the victim into the trees or straight over the pachyderm's back; a number of victims have survived this experience. Some hunters pursued by elephants have diverted them by throwing off items of clothing.

Many rogue elephants seem to be injured; others have become intoxicated by eating fermenting marula berries. Persistent hunting has made the elephant more wary and irritable than it was 100 years ago. Its traditional feeding and migration ground is increasingly invaded by farmers, leading inevitably to negative interactions. The elephant is incredibly destructive of plants and trees, and its future in this escalating conflict is in grave doubt. A few rare stories suggest that an elephant may actually be a man eater; one of these occurred in 1944 at the Zurich zoo. Elephants captive in zoos and circuses may cause deaths while temporarily deranged; since 1990, captive elephants have killed 39 and injured more than 100 humans worldwide.[163]

Hippopotamus

The hippopotamus is a frequent killer in Africa. Its placid and indolent appearance in zoos completely belies its activity and personality in the wild.

A hippopotamus can run at speeds of up to 45 mph on land. It is relatively unpredictable and made irritable by people.[129] It will attack boats and people in the water if it feels trapped (e.g., between a boat and deep water) or if its calf is threatened. With its large canine teeth, it can chop canoes (and the people in them) in half, and it does so several times a year on the Zambezi River in Zimbabwe. However, these attacks are usually in self-defense, and a hippopotamus will get out of the way if it can. Hippopotamuses graze on land and habitually run along established narrow tracks back to the river, mostly at night but also in the day. They will not change course for anything, and humans who make the mistake of staying in the tracks may be trampled.

Moose

Moose are large and potentially aggressive animals, especially during fall rutting and spring calving seasons. When moose are fed by humans, they become more aggressive when food is no longer available.[5] Moose are difficult to chase away, but it is easy to flee from a moose to a larger shelter. If the rump hairs are raised, the ears are laid back, or the moose is licking its lips, it is likely to charge. The subject of the attack should get behind a large solid object for protection. If knocked to the ground by a moose, the victim should roll into a ball, protecting the head with hands and arms and remaining still until the moose leaves.[5] A man was killed in 1995 by a moose in urban Alaska when he approached a cow with a calf who had been harassed by students throwing snowballs.[148] Moose consider dogs predatory and will attack and kill dogs without other obvious provocation.

Most human injuries caused by moose result from moose–motor vehicle collisions, which are quite hazardous in New England and in Sweden.[29,77] In a New England report, an

Figure 51-11. Toddler victim of moose attack. Hoof mark on chest wall overlies rib fractures and pulmonary contusion. (Photo by Luanne Freer.)

Figure 51-12. Yellowstone visitors approach bull elk. (Photo by Luanne Freer.)

average of two persons per year are seriously injured when their vehicle strikes a moose, usually after dark; 70% of those injured had head and facial injuries, and 26% had cervical spine injuries. The mortality in this series was 9%, and morbidity declined with use of seat belts and rear seat location (Fig. 51-11).[77]

Deer and Elk

The deer population is burgeoning in parts of the United States, and as a reservoir of the tick that carries Lyme disease, it may be credited with the increasing incidence of human Lyme disease.[144] Block Island, an 11-square-mile island off Rhode Island that reintroduced one buck and four does to its area in 1968 now has a herd of over 1000 animals and has recorded 45 cases of Lyme disease in residents over the past 9 years. In addition, residents have been attacked by deer in their own yards.[144]

Deer rarely attack humans, but deer–motor vehicle collisions are quite common in densely populated areas and accounted for 231 deaths in the United States and for over 21,000 collisions in Finland in 1 year, the majority of these events occurring in the first hour after sunset.[99] One deer jumped through a window of a law office, injuring an attorney,[14] and another buck in rut carried a man in its antlers for 45 minutes, then pinned the man on the ground until he was rescued.[27] A child was gored by a startled buck when he was feeding the animal; the antler penetrated the axilla, lacerated the pulmonary artery, and caused death by exsanguination.[76] Deer may bite, and like other ungulates may carry *Pasteurella* as oral flora. Female deer bite other deer when fighting. Males bite when testosterone levels are low, because at these times antlers are soft, pain sensitive, and cannot be used as weapons. Foreleg kicks are more common, because the deer can stand on its back legs. They have a dental pad instead of upper incisors, so bites are rarely serious and are usually directed at an arm and the back, which are normally well covered by clothing. These bites are usually single nips.

Like moose, bull elk may attack humans when in rut, and cows may attack by butting and stomping when protecting their young.[160] Up to 1% of elk sampled in one study carried *Brucella abortus* and *Mycobacterium bovis* (Fig. 51-12).[176]

Rhinoceros

The black rhinoceros has been represented as one of the meanest animals in Africa because it charges any moving object, including trains. The click of a camera, a gentle movement, or a scent is enough to induce a charge. Because the rhinoceros has poor eyesight but excellent hearing, it may well be running toward sounds to investigate them. Contrary to the popular belief that because of its nearsightedness it can be easily sidestepped, the rhinoceros can turn on a dime.[51] At the end of its charge, it usually hooks right and left with its horns and is generally satisfied with tossing the victim high (12 feet [4 m]) in the air. However, like so many large wild animals, it probably does not desire confrontation. Rhinoceroses often flee once they have identified a sound as originating from something dangerous (such as a human), and persons who have fallen while running from a charge have been investigated with a few typical snorts and then ignored. However, severe injuries can result if the human fails to get out of the way of a rhinoceros. As with the hippopotamus, injuries and death may have more to do with being in the path of a very large, fast-moving object than with malicious intent. The white rhinoceros is docile and has killed few, if any, people.

Tapir

The tapir *(Tapirus terrestris)* lives in rainforested areas in South America, Central America, and Mexico. The female reaches a length of up to 2 m and a height of up to 1 m, and weighs up to 200 kg. Tapirs are herbivorous and generally docile, seeking refuge in brush when threatened, but they may defend themselves by biting with powerful jaws and teeth. A tapir bit off the arm of a zookeeper who came between the usually docile animal and its calf,[108] and a man died of exsanguination from arm and neck lacerations after a tapir attack in Brazil.[99,213]

Other Wild Herbivores

Other wild species, such as the giraffe, may turn rogue, but this is exceedingly rare. The black wildebeest has killed one or two zookeepers, as have the spiral-horned kudo and bushbuck. Other antelopes have killed or wounded hunters or zookeepers with their sharp horns. Zebras are known to bite tourists who approach too closely.[202]

Marsupials

Kangaroo

The largest living marsupials in the world (weighing up to 175 pounds [80 kg]), kangaroos use their tail as a balance when they jump, and they can make jumps almost 30 feet (10 m) long and 8 feet (3 m) high. They are active in the evening and at night, when groups of kangaroos, called mobs, forage on grasses and other plants. Traffic collisions involving kangaroos in Australia are increasing in incidence and occur predominantly at night and in the summer season; 46 human injuries were reported at one hospital alone in a 6-year period.[1]

Opossum

The American opossum *(Didelphis virginiana)* inflicts bite injuries both when hunted for food and when accidentally provoked while being handled in captivity. Aerobically cultured organisms from the mouths of seven wild opossums included streptococci, coagulase-positive and coagulase-negative staphylococci, *Aeromonas* species, *Citrobacter freundii, Eikenella corrodens,* and *Escherichia coli.*[105] No literature is available on the bites themselves.

Quokka

One of the smallest of the wallaby family residing in Western Australia, the quokka typically bites only humans who attempt to feed or pet the animal. Bites tend to occur on the finger or hand. Despite the presence of mixed coliform bacteria in quokka mouth cultures, the incidence of bite-wound infection in this series was zero.[137]

Venomous Mammals

Only two types of venomous mammals are known. The short-tailed shrew *(Blarina brevicauda)* of the northeastern United States secretes a protein venom from its maxillary gland and injects it with its lower incisors. The venom may cause edema, a few days of burning sensation, and pain lasting up to 2 weeks.[42] No specific antivenom is available, and treatment is symptomatic. No bites have been reported since the 1930s. A similar venom is possessed by the European water shrew *(Neomys fidiens)* and the primitive Cuban insectivore *(Solenodon paradoxus).*[34] Documented bites are exceedingly rare.

A second type of venomous mammal is the male platypus *(Ornithorhynchus anatinus),* which injects venom from a hollow spur in its hind leg. This venom appears to resemble viperine snake venom and causes local pain, edema, and lymphangitis.[34,201] The pain can be excruciating and completely unresponsive to intravenous narcotics. Regional nerve block has been reported to be effective in combination with limb immobilization.[78] Localized edema also occurs. No specific treatment is available, and the exact pathophysiology is unknown. Functional recovery may be delayed for up to 3 months. The echidna, or spiny anteater, possesses a similar spur and venom, but envenomation has not been reported.[82]

Large Wild Birds

The ostrich is responsible for one or two deaths per year, mostly in Africa, where it is raised commercially (Fig. 51-13).[13] Most of the fatal attacks are kicks to the head and abdomen. The ostrich can kick only forward, but when it does, a sharp toenail flicks out like a switchblade and can penetrate the abdomen. Because the ostrich can easily outrun a human being, the only protection is to lie prone to protect against disembowelment and to cover the neck to protect against pecks. Eventually, the ostrich loses interest and allows the victim to escape.

Figure 51-13. African ostrich. (Photo by Cary Breidenthal.)

Figure 51-14. Alligator. (Photo by Stuart Coleman.)

The cassowary (common in Papua New Guinea) can easily disembowel a hunter with a single kick from its long, sharp toe claws.

Birds of paradise have been found to secrete a venom on their feathers, although cases of human toxicity from this have not been reported and the phenomenon is little studied.

The emu is a large flightless bird native to Australia that may weigh 100 to 150 pounds (45 to 70 kg). It is usually docile, but when cornered or frightened it may lacerate a human handler by kicking.[111]

Large Reptiles

The Nile crocodile accounts for 1000 human deaths per year in Africa.[51] Individual crocodiles have been responsible for up to

400 deaths. Most attacks take place in the water, where crocodiles are accustomed to scavenging for the dead, sick, and deformed human babies that are tossed into the water to be disposed of by these reptiles. The crocodile has tremendous grip strength and locks this grip by slotting two lower teeth into holes in the upper jaw. When a crocodile is unable to drag the victim completely underwater, it may grasp a limb and then spin over until the limb is detached.

In a 10-year period in Australia, there were 16 attacks and four fatalities caused by crocodiles; most victims were swimming or wading at night, and alcohol ingestion was present in half.[140] Wound infections with *Aeromonas hydrophila, Pseudomonas pseudomallei, Pseudomonas aeruginosa,* and *Proteus, Enterococcus,* and *Clostridium* species were reported in 6 of 11 survivors in this series.[140] In Malawi, over a period of 4 years, 60 victims were admitted to hospitals after injury by crocodiles—40% had serious injuries resulting in permanent deformity, and one person died from sepsis.[205]

The American alligator is thriving in the southern United States (Fig. 51-14), and its habitat is so greatly threatened by human expansion that incidents of alligators appearing in suburban backyards and swimming pools are now common. Attacks on children and pets are increasing, with 127 attacks and five deaths reported in Florida from 1973 to 1990.[104] The alligator causes crushing injuries to the torso and open extremity fractures, and it may roll underwater with the victim, resulting in drowning. Blunt trauma may result from a strike by the animal's massive tail. Prevention of alligator attacks includes avoiding touching or feeding the animal or swimming at feeding time (dusk) with a dog or in waters with heavy vegetation.[56]

MEDICOLEGAL CONSIDERATIONS

In some cities and regions, animal bites must be reported to public health authorities. Reporting suspected exposure to rabies is mandatory in most regions, and failure to report could become the basis for legal action. Reporting often leads to examination of the offending animal by public health authorities and sometimes to quarantine or destruction of the animal.

Although most wild animals by definition do not have an owner, some exotic species are kept as pets. An owner's failure to meet local regulations regarding licensure and vaccination can lead to legal action. In addition, the victim may seek compensation from the owner of the animal, with the result that the health care provided and the medical record will be reviewed in court. The injury may be related to the victim's employment, generating worker's compensation or other insurance claims. Therefore, injuries and their circumstances should be documented as fully as possible, with line drawings or photographs added to the medical record whenever possible. Certain types of animal bites, particularly those of the hand, are prone to infection and can lead to permanent, litigation-associated complications. A complete medical record is the best protection in these cases and will also be valuable if a malpractice claim is made against the health care provider.

The references for this chapter can be found on the accompanying DVD-ROM.

Bear Behavior and Attacks

52

Steven P. French

Bears are among the most widely distributed animals in the world. At least one of the eight bear species currently exists in Asia, in Europe, in North and in South America, and in the Arctic (Table 52-1). Bears in Africa became extinct several million years ago. Australia and Antarctica are the only continents where bears have never existed. The koala bear of Australia is a marsupial and not a true bear.

Bears occupy a wide variety of habitats, including tropical forests, polar ice sheets, swamps, barren ground tundra, bamboo jungles, alpine meadows, and coniferous and deciduous forests. Their range extends from sea level up to about 6100 m (20,000 feet).

Bears are carnivores. Although some bear species practice specialized feeding in response to their habitat, all bears are omnivores and retain the ability to feed on a variety of food types, including vegetation, insects, and meat.

Modern bears have larger brains than did their extinct ancestors,[23,34] and the relative brain size of bears is larger than that of other carnivores.[13] This greater brain size probably resulted from a need to increase sensory and perceptual capacities for locating an omnivore food base with both seasonal and annual variations in distribution and abundance.[5,8,28] The larger brain size reflected the increased intelligence required by bears to develop a complex foraging strategy. Increased intelligence also allowed them to develop individual behavior, shaped by both experience and memory. Thus they possess a wide variety of behaviors and have been described as playful, lazy, doleful, entertaining, intelligent, caring, powerful, aggressive, terrifying, and vicious.[39]

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TABLE 52-1. Distribution of Bear Species

COMMON NAME	SCIENTIFIC NAME	DISTRIBUTION
Panda bear	*Ailuropoda melanoleuca*	Eastern rim of China's Tibetan Plateau
Spectacled bear	*Tremarctos ornatus*	Andes Mountains in South America
Sloth bear	*Melursus ursinus*	Nepal, Bangladesh, Bhutan, northern India, Sri Lanka
Asiatic black bear	*Ursus thibetanus*	Southern Asia from Pakistan across northern India and into China and southeast Asia; separate populations in eastern Russia, Korea, Taiwan, and Japan
Sun bear	*Helarctos malayanus*	Borneo, Burma, Java, Malaysia, Sumatra, Thailand
American black bear	*Ursus americanus*	Alaska, Canada, most of lower 48 states
Brown bear	*Ursus arctos*	Eurasia, Alaska, Canada, northern Rocky Mountain states (including Wyoming, Montana, Idaho, and Washington)
Polar bear	*Ursus maritimus*	Arctic circle (circumpolar)

The image of bears as "man-eaters" ignites our fear of them. Human injury and deaths from natural phenomena, especially wild animal attacks, are sensationalized. Bear attacks are rare, but the psychological impact of widespread media coverage inflates the perception of their frequency and significance.[6,25] Every bear attack is traditionally referred to as a mauling, regardless of the extent of injuries. This term contributes to the emotional response regarding such attacks and leads to "bear-anoia" in many people who visit bear country. This fear of bears may affect how people use wilderness areas that have bear populations and how they view the conservation of bears and their habitat. Better understanding of bears and their behavior helps reduce bear attacks, assists physicians in treating bear attack victims, and promotes conservation of bears.

Figure 52-1. Grizzly bear. (Courtesy Marilynn G. French.)

NORTH AMERICAN BEARS

Grizzly bears (*Ursus arctos horribilis*) are larger and more heavily built than most other ursids, with adults weighing 146 to 383 kg (325 to 850 pounds) (Fig. 52-1).[1] Polar bears (*Ursus maritimus*) are similar in size and weight but are more elongated in shape (Fig. 52-2). Black bears (*Ursus americanus*) (Fig. 52-3) have the same general shape of grizzly bears but are generally smaller than both polar and grizzly bears. Weights for black bears range from 63 to 135 kg (140 to 300 pounds) for adult females and 113 to 293 kg (250 to 650 pounds) for adult males.

Dentition in these three species is bunodont and reflects their omnivorous diet, although polar bears are the most carnivorous of the three (Fig. 52-4). Their canine teeth are sturdy and can reach a length of 7 cm ($2^3/_4$ inches). Their legs are of approximately equal length and taper to large, plantigrade feet. The fore claws of a grizzly bear are heavier, longer, and straighter than those of a black bear and can reach a length of 8.75 cm ($3^1/_2$ inches) measured along the external curvature (Fig. 52-5).[40] A large, muscular hump overlies the scapulae of grizzly bears, giving additional strength to the forelimbs for digging (Fig. 52-6). The face of a brown bear tends to be more dish shaped (concaved) than that of a black bear (Fig. 52-7).

The physical strength of bears is tremendous, and they can run at speeds up to 65 kph (40 mph) over irregular terrain. They have a keen sense of hearing and an even keener sense of smell. Their eyesight has been described as poor,[19] although many field researchers believe that bears can see as well as humans and are

Figure 52-2. Polar bear. (Courtesy Steven D. Evans.)

Figure 52-3. American black bear. (Courtesy Marilynn G. French.)

Figure 52-6. Alaskan coastal brown bear, demonstrating muscular back hump. (Courtesy Luanne Freer.)

Figure 52-4. Grizzly skull, demonstrating canine teeth. (Courtesy Marilynn G. French.)

Figure 52-7. Silhouette of brown bear, demonstrating hump and "dished" face. (Courtesy Timothy Floyd.)

Figure 52-5. Grizzly paw with claws. (Courtesy Marilynn G. French.)

especially adept at detecting movement. Evidence also suggests that grizzly bears have good night vision.[11]

Grizzly and black bears hibernate for about 5 months during the winter, an evolutionary adaptation to reduced food availability. The hibernation of polar bears is slightly different, as their primary food (seals) is available during the winter.[31] Adult male polar bears tend to hibernate for short periods each winter in response to severe storms, whereas pregnant females have more extended hibernation. During the active (nondenning) season, all bear species wander throughout a general home range in search of seasonal foods.

The guard hairs on brown bears can be lighter in color and lend a "grizzled" appearance. Black bears can be many colors, ranging from white to black, cinnamon, brown, or "blue."[9]

Grizzly Bears

The grizzly bear symbolizes wilderness in North America (Fig. 52-8). In certain respects, "grizzlies" define the "wild" in wilderness. They range from Alaska down through western Canada and into the lower 48 states in remnant populations

Figure 52-8. Grizzly bear encounters have become popular events for wildlife photographers. (Photo by Timothy Floyd.)

located in relatively undeveloped federal lands, primarily in the northern Rocky Mountains.

Attacks by grizzly bears are relatively rare and sporadic. A total of 162 bear-inflicted injuries (including deaths) were reported from 1900 through 1985 in Canadian and North American national parks.[19,20] In the Canadian province of Alberta, there were 29 documented serious or fatal injuries caused by grizzly bears during the period of 1960 through 1998.[22] From 1980 to 1994, 21 grizzly bear attacks, including two deaths, occurred in Yellowstone National Park.[18] During the following decade, one or two people were injured by grizzly bears in Yellowstone each year, with no recorded deaths. In Alaska, the number of people injured by grizzly bears has increased in recent times, most likely as a result of increased recreational use of grizzly habitat.[29,36]

Calculation of an accurate injury rate remains elusive. Earlier records were incomplete, and it has always been difficult to define and quantify those at risk. Injury rates are based on total visitation days to the national parks in Canada and the northern United States.[20] The average number of grizzly bear–inflicted injuries is 1 in 2,260,276 visitors to these parks combined, with a high of 1 in 317,700 visitors in Kluane National Park and a low of 1 per 6,693,859 visitors in Banff. During this same period, the grizzly bear–inflicted injury rate for Yellowstone National Park was 1 in 1,543,287 visitors, and for Glacier National Park it was 1 in 848,180 visitors.

Not every visitor to a national park is exposed to the same risk of being attacked by a grizzly bear. To obtain an injury rate that more accurately reflects the risk for visitors with higher and more uniform exposure, the incidents among registered backcountry users have been reported. However, this method provides an inaccurate injury index because some parks do not register backcountry use and others generally underestimate it. Also, significant and perhaps most backcountry use (and therefore exposure) is by unregistered day hikers.

The number of bear attacks (both black and grizzly) increases in months when more people seek recreation in grizzly country. For national parks, the incidence of bear attacks increases during the peak tourist season, July and August. For surrounding national forests, another peak occurs during hunting season, September to November. With more people seeking recreation in bear country, greater opportunity exists for human–bear encounters.

Native peoples and grizzly bears occupied the same land for thousands of years in North America in what was probably a neutral coexistence, as neither had a profound influence on the other. However, the European expansion into the West after Lewis and Clark's expedition in the early 1800s tipped the scales heavily in favor of humans, both in sheer numbers and in technology, such as guns, traps, and poisons. Bears were killed in large numbers, out of fear and hatred and to protect life and property. Most of their original habitat was occupied by either people or livestock or was dramatically altered by ranching and agricultural development.

Selection pressures that began with European expansion into the grizzly bears' habitat have probably been altering their behavior. Since that early period and even today in protected areas such as national parks, aggressive bears have been removed at a higher rate than nonaggressive bears. Bears that were curious about humans and human developments and those that did not readily flee the presence of humans were also removed at a higher rate. Therefore, bears that avoided humans survived at a higher rate than other bears and probably passed that trait on to their offspring through genes and learning.

A built-in safety factor exists for people entering grizzly country, because the vast majority of bears now avoid a confrontation if given the opportunity, which probably explains why grizzly bear attacks on humans are so rare. Unfortunately, the available information on grizzly attacks does not always yield an accurate account of the cause-and-effect relationship. The specific sequence of events is not always known and is subjectively reconstructed, although case histories reveal certain patterns.

A sudden and close encounter with a grizzly bear is the primary event leading to human injury. From 1980 to 1994, of 21 people injured by grizzlies in Yellowstone National Park,[18] 18 resulted from people surprising a grizzly at close quarters. These attacks were often brief, and the bear generally left the area soon after the attack. Although injuries were typically described as a mauling, they were generally much less severe than the bear had the potential to inflict, and victims were rarely killed. This suggests that the bear's behavior in response to a close encounter was to remove a perceived threat.

A close encounter with a female with cubs is considered more dangerous, as she is considered to be more aggressive in defense of her young (Fig. 52-9). Evidence to support this hypothesis is strong. Females with young represent about 20% of a bear population but account for more than 80% of the bears that injure humans. Another explanation, however, is that females with young are more likely to be active during daylight hours when humans are active, whereas males are active primarily in the predawn hours and after dusk.[11]

Grizzly bear attacks sometimes occur near a carcass on which the bear has been feeding. The bears may be more aggressive under these circumstances in defense of the carcass. Grizzly bears of all ages and either sex, however, may readily exit when they sense people approaching.[11] When a grizzly bear injures someone near a carcass, the precipitating event may simply be a close encounter with a preoccupied bear.

Another class of attacks results from provocation, most often when a grizzly bear is wounded with a gun. Once the bear is

Figure 52-9. Yellowstone grizzly sow with cubs. (Courtesy Brian Ertel.)

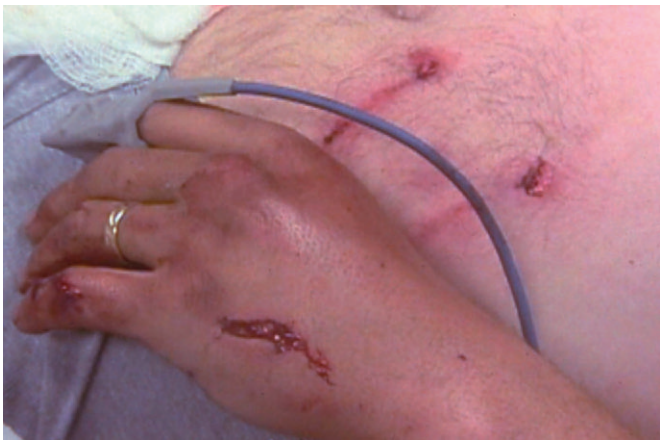

Figure 52-11. Hunter attacked by grizzly bear. (Courtesy Luanne Freer.)

Figure 52-10. Photographers dangerously close to bears in the wild. (Courtesy Luanne Freer.)

injured, its behavioral response is no longer to remove a threat but to fight for its life. These attacks tend to be more prolonged and aggressive, resulting in more severe injuries than those resulting from a close encounter. Provoked bear attacks can result from direct harassment by aggressive photographers (Fig. 52-10). Although such incidences are rare, these attacks tend to resemble the response of an injured bear rather than one responding to a close encounter. The injuries tend to be more severe, and a disproportionate number of photographers are killed. Up to 1985, at least 10 photographers were injured, one fatally, and from 1986 to 1992, at least four were injured, two fatally.[19]

Most people attacked by grizzly bears are injured but not killed; the intent of the bear is simply to remove a perceived threat, not to prey on the individual (Fig. 52-11). From 1900 to 1979, 19 human deaths resulted from grizzly attacks documented in the national parks in North America, and 22 deaths occurred in Alaska outside the parks.[19,25] Although there have been no published data for the number of human deaths from grizzly attacks for the past 25 years, that number has averaged less than one per year for these same areas. Some were victims of defensive attacks, and prior to the 1960s these victims would probably have survived if current medical techniques had been available. Some deaths, however, resulted from predatory attacks. It is important to ask why grizzlies do not prey on humans more often. As a potential prey species, humans are predictable and abundant, easy to catch and kill, and easy for a grizzly to consume.

Historic evidence suggests that grizzly bears did not routinely prey on humans except in unusual circumstances. In 1860, a smallpox epidemic struck a small band of Stonie Indians (Assiniboin tribe) camped in the Yarrow Creek drainage in Alberta, Canada.[37] Grizzlies began scavenging on the dead left on the ground as the tribe moved to the next drainage. Grizzlies followed them to their next encampment and began preying on survivors. For years, the Indians avoided this area for fear of being eaten by grizzlies that had "learned" to prey on humans.

Since about 1900, when reasonably accurate records were first kept, predatory attacks on humans by grizzly bears generally have been rare, sporadic, and isolated events.[1] However, a disturbing trend has begun in recent times. Between 1967 and 1986, 12 deaths were inflicted by grizzly bears in Banff, Glacier, and Yellowstone National Parks. In each case, the bear was conditioned to humans' food (regularly seeking out and obtaining it) or habituated to human presence (not readily fleeing). Nine of the victims were partially consumed, and eight deaths were classified as predatory events.[19] During this same period, however, many bears with these same behavioral traits did not prey on humans. Conditioned and habituated behavior may predispose some grizzlies to prey on humans under certain but still not understood circumstances. The relationship between conditioning and habituation appears strong but is not conclusive. The bear involved was not always known, and the terms *conditioned* and *habituation* are both borrowed from learning theory and have never been precisely defined by wildlife biologists. This potential relationship, however, has significantly influenced grizzly bear management. Currently, the primary thrust is to prevent bears from obtaining human foods and from routinely being around people and human developments.

Figure 52-12. Black bear. (Courtesy Timothy Floyd.)

Grizzly bears may also mistakenly perceive a person as one of their normal prey species. Five such incidents have been documented. Two victims were killed by grizzly bears while making prey calls to lure in other predators. Two victims were attacked while field-dressing a game animal, and the fifth was attacked while carrying the hide of a deer draped over his shoulder. Clearly, persons should not look, smell, or sound like a prey species when in grizzly country.

Black Bears

Black bears are the most numerous and widely distributed of all North American bears (Fig. 52-12). They occur in more than 30 of the lower 48 states, from Maine to Florida and from California to Washington. They also occur throughout Canada and Alaska, extending up to tree line below the Arctic Circle. They are well adapted to an arboreal habitat and prefer to eat vegetation, carrion, and mast (nuts, acorns), with small mammals and insects accounting for less than 5% of the diet.[9]

Between 1960 and 1980, more than 500 people were injured by black bears, but at least 90% of these episodes resulted in minor scratches or bites inflicted by bears that were either conditioned to human foods or habituated to human presence.[19] The number of victims of black bear attacks has declined significantly during the past 25 years, in large part because of the aggressive bear management in and around national parks that discourages the feeding of black bears and removes those bears that seek out human foods. Injuries as a result of close encounters are extremely rare, and in contrast to female grizzly (brown) bears, female black bears display little aggression in defense of their young and rarely cause injury. They have short, sharp radial claws better adapted for climbing trees than for attacking humans. They often retreat rather than attacking, even in defense; thus, hunters using dogs can "tree" black bears.[9]

Whereas grizzly (brown) bears sometimes prey on humans at night, black bears occasionally prey on humans during the daytime. From 1900 through 1980, 20 people were killed by black bears, with predation considered the motivation in 18 cases. All but one case occurred in remote areas outside park boundaries, an indication that neither conditioning nor habituation was a major factor.

Since 1985, black bears attempted to prey or preyed on humans in 15 episodes, with two fatalities and seven major injuries.[19] Details are scant, but at least four occurred at night while the victims were asleep. In one case, the bear broke into a camper and pulled the victim out, and in another case the bear entered a wooden teepee ("wickiup") and dragged the victim out by her foot.[33] In most attacks, the black bears were driven away by aggressive actions by the victims and their companions, such as yelling and throwing objects.

POLAR BEARS

Polar bears are distributed in a circumpolar fashion around the Arctic Circle and subsist almost exclusively on a diet of seals (see Figure 52-2). In winter, these bears feed primarily on ice-covered polar seas. Some southern populations live on land during the summer in a state of waking hibernation and starvation. Polar bear–inflicted injuries are much less frequent than those by grizzly (brown) or black bears, primarily because of their remote and harsh environment with relatively little human intrusion. From 1973 to 1987, three people were injured (one fatally) in Norway,[14] and from 1965 to 1985, 20 people were injured (six fatally) by polar bears in Canada.[20] The number of injuries would probably be much higher except that most people in polar bear habitat are armed, and in the majority of aggressive encounters the bear is killed before causing injury.

Polar bear–inflicted injuries have been classified into two general categories. The major one is predation, primarily by subadult and adult males. In these instances, five of the six victims who died were probably killed instantly. The other category is injury by adult females thought to be defending their young. These episodes are typically brief and nonfatal, which supports the theory that the bear is removing a perceived threat. In more than 90% of aggressive encounters with polar bears, an attractant, such as food, garbage, or carcasses, was considered contributory.[20]

BEARS ON OTHER CONTINENTS

The available data on attacks by bears on other continents are much less complete than those for North American bears. In Europe, the brown bear has coexisted with humans much longer than those in North America. (In some parts of North America, the brown bear is called a grizzly bear, but they are genetically the same species.) As a result, its behavior is less aggressive and more like that of black bears. Numbers of European brown bears are extremely low; the animals are highly cryptic and nocturnal and thus are rarely seen or encountered. Human injury by brown bears in Europe today is almost nonexistent.

The brown bears in the former Soviet Union live in vast, relatively undeveloped areas and appear to have aggressive responses against humans similar to those of North American brown bears. Many human injuries from brown bear attacks,

including deaths, may be related to bears injured by sport hunters.[4]

The panda bear (*Ailuropoda melanoleuca*), commonly known as the giant panda, lives in the temperate climate of the bamboo jungles distributed along the eastern rim of China's Tibetan Plateau. It is one of the most recognized bears in the world, with a distinctive white and black coloration. It is a relatively poor climber but will climb trees on occasion to avoid danger. During winter months, the panda bear migrates to lower elevations where food remains plentiful, thus avoiding the need to hibernate.

The panda bear is primarily a vegetarian. About 99% of its diet consists of stalks, leaves, and shoots of only two bamboo species.[38] The panda has an enlarged wrist bone that serves as an opposable digit, much like a thumb. This evolutionary adaptation enables the panda to efficiently hold and strip bamboo stalks. Because bamboo is a poor-quality food, the panda must compensate by eating large amounts. Each day it feeds up to 12 hours. The panda bear is shy and reclusive, representing a minimal threat to human safety in the wild.

The spectacled bear (*Tremarctos ornatus*) lives in the tropical climates of the Andes Mountains along the northwest border of South America. It is one of two bear species that live below the equator. The spectacled bear has a distinctive white coloration around its eyes. It is an excellent climber and spends most of its time in trees eating fruit. It often builds nests and rests in trees as well. Because its source of nutrition is abundant year-round, it does not hibernate. Spectacled bears are relatively small and shy. Encounters are extremely rare, and they pose very little threat to human safety.

The sloth bear (*Melursus ursinus*) lives in the subtropical forests of Nepal, Bangladesh, Bhutan, India, and Sri Lanka. It has a disheveled appearance because of its long, shaggy fur coat. In some of its range, the sloth bear coexists with elephants, wild boars, leopards, tigers, greater one-horned rhinos, and Asiatic black bears.

The sloth bear is a special type of insectivore, termed myrmecophagous, because it feeds primarily on ants and termites. It is uniquely adapted for this feeding behavior. Without the two upper incisors and with an elongated and raised hard palate, mobile lips, and nearly naked snout, the sloth bear can blow away dust to expose termites and create a strong sucking force to feed. It can dig out insect burrows with its long claws, and its coat protects it from insect stings.

Although data are limited primarily to anecdotal reports, after the Russian brown bear, the sloth bear appears to be the most dangerous bear species in Europe or Asia. Approximately one person is seriously injured or killed by a sloth bear in Chitwan National Park in Nepal each year.[12] In the remote regions of western Nepal, at least one villager is seriously injured by a sloth bear every other year. Most of these injuries are the result of a close encounter, and the victims receive wounds to the head and neck. No predatory behavior has been reported.

The aggression of the sloth bear is rated between that of the American black bear and grizzly bear. Sloth bear researchers in Nepal work exclusively while riding elephants because of their concern about attacks from rhinos, sloth bears, and tigers, in that order.[24]

Asiatic black bears (*Ursus thibetanus*) occupy the broadleafed forests throughout a large portion of south Asia, from Pakistan across northern India and into China and Southeast Asia (Fig. 52-13). Separate populations also occur in eastern Russia, Korea, Taiwan, and Japan. Some of their range overlaps with that of brown bears, sloth bears, and sun bears. The Asiatic black bear is sometimes called the "moon bear" or the "white-breasted bear" because of the crescent-shaped white coloration on its chest.

Figure 52-13. Asiatic black bear. (Courtesy Marilynn G. French.)

Figure 52-14. Sun bear. (Courtesy Marilynn G. French.)

The Asiatic black bear is a dietary generalist and feeds on a wide variety of plants, insects, and animal matter. It is a good climber and often forages and rests in trees. Unlike pandas and spectacled bears, these bears hibernate during the winter months. They are hunted extensively for illegal trade of bear parts and rarely present a threat to human safety. There are in fact one to two attacks per year that result in significant injury requiring hospitalization. Although rare, these do occur mostly as a result of a close encounter.

The sun bear (*Helarctos malayanus*) is the smallest of all bear species, rarely weighing more than 45 kg (100 pounds) (Fig. 52-14). It occupies tropical regions in Borneo, Burma, Java, Malaysia, Sumatra, and Thailand. As with the spectacled bear, the sun bear is equatorial because part of its range extends below the equator. It has a white to cream-colored, horseshoe-shaped marking on its chest, providing its common name. In

some localities it is called the honey bear. It has a long slender tongue, an adaptation for extracting honey from beehives. Its claws are long and more sharply curved than those of other bears, enabling it to be a proficient tree climber, where it can easily hang upside down from a branch. The sun bear is rarely seen and represents almost no threat to humans.

PREVENTION AND RISK REDUCTION

Much literature about safety in bear country involves attack victims.[6,19,20,25,29] Such information is gathered from victims who are generally unfamiliar with bear behavior and whose interpretations of the events reflect their cultural biases. Also, victims often become instant media celebrities. In several cases, the circumstances surrounding the attack changed significantly with each telling, usually reducing the victim's culpability. Because of potential litigation, some victims have told their stories only through an attorney. Caution must be used when compiling and analyzing such "data."

Recommendations for avoiding bear attacks have been drawn primarily from what attack victims did "wrong." Because most people who live, work, and regularly vacation in bear country are never injured, it is equally important to understand what they have done "right." Unlike bear attack victims, these people have successfully navigated grizzly country without being injured. Although this information is not as readily available as attack records, it is critical to our knowledge of grizzly–human interactions.

From 1900 to 1985, 115 human injuries were reported from black, polar, and grizzly bear attacks in Alaska, but only two victims were natives.[29] This strongly suggests that the behavior of people is important in determining how to coexist with bears safely. Safety in bear country involves four levels of interaction: (1) avoiding an encounter, (2) reducing the chances of being attacked after an encounter, (3) reducing the severity of injuries received if attacked, and (4) reducing the chances of becoming prey to a bear.

Avoid an Encounter

The following actions can significantly reduce the chances of having a close encounter with a bear:

1. Make noise so that the bear knows a person is present. This requires only casual conversation to prevent startling a bear at close range. The voice may have to be amplified somewhat while traveling along a noisy stream or a windy ridge. Foghorns have been used successfully in Alaska; "bear bells" may not be sufficiently loud (Fig. 52-15).
2. Remain alert in bear country and be aware that the terrain and environment may hamper a bear's ability to detect a human by sight, smell, or sound. The terrain and environment may also hamper *your* ability to see or hear a bear before it discovers you. An "upwind bear" is more likely to be surprised, as is one in heavy forestation or near loud rushing water, in the rain, or in fog.[9] Avoid ripened berry patches, streams with spawning fish, and elk calving grounds.[9] A flock of ravens may indicate carrion and the presence of feeding bears.
3. Always use good judgment to avoid a potentially dangerous situation. If fresh bear signs are seen, such as tracks (Fig. 52-16), droppings, tree scratchings (Fig. 52-17), or a carcass (or even scavenger activity indicating that a carcass may be nearby), consider that a bear is in the vicinity and take an alternate route. If the bear is seen first, slowly and quietly retreat to safety; consider aborting the trip or taking an alternate route. Do not approach bears, or any wild animals, too closely for a better view or photograph.

Figure 52-15. One hopes not to find bear bells within or on bear scat. (Courtesy Timothy Floyd.)

Figure 52-16. Bear prints. (Courtesy Timothy Floyd.)

Bears in bear–bear confrontations demonstrate signs of aggression and annoyance. These include standing or turning in profile to appear larger and intimidating, vocal hissing and jaw popping, "yawning" (Fig. 52-18), and head swinging (Fig. 52-19).[9]

Avoid Provoking an Attack

Again, the best way to avoid bear-inflicted injuries is simply to avoid surprising a bear in a close encounter situation. Although no set of responses is guaranteed to prevent injury in a close encounter, the following generalizations may be useful:

1. Allow the bear to know you are human and not a prey species. Step away from any visual obstruction to allow the bear to see you fully. Any attempt to hide at this point will only confuse the bear, which may approach closer to iden-

Figure 52-17. Tree scratchings from bear claws. (Courtesy Timothy Floyd.)

Figure 52-18. Yawning behavior may indicate agitation in a bear. (Courtesy Timothy Floyd.)

tify you, thus creating an encounter at an even closer distance. It is probably best to talk in a calm voice to allow the bear to identify you as a human.

2. Although remaining calm is difficult, do not make sudden movements or yell out, particularly with a grizzly bear. The bear may view this as an aggressive action and deal with it by an aggressive response.
3. Do not stare directly at the bear. Look to the side or stand sideways to the bear. Standing your ground is important in determining the bear's response.[40] This posture tells the bear you are willing to defend yourself if necessary, and it may prevent further aggressive behavior.

Figure 52-19. Head-swinging behavior in a polar bear. (Courtesy Timothy Floyd.)

4. Do not consider climbing a tree or running away. Not only is it impossible to outrun a bear, but running may prevent the bear from correctly identifying a human and may initiate a charge. Once a bear charges, you cannot locate a climbable tree and achieve a safe height fast enough. Attempting to climb a tree may also prevent the bear from correctly identifying you as a human. Therefore, the best defense during a charge is to stand quietly and nonaggressively, and allow the bear to identify you as human and not a prey species.

In most cases, the bear aborts the charge after a close encounter without making contact or causing injury. At this point you should leave the area, retreating in the direction opposite to that taken by the bear. If a bear continues to charge, however, resulting in physical contact, your actions should depend on the species, and information from bear attack victims is useful.

Reduce the Severity and Extent of Injuries

If attacked by a bear, a victim can take several important steps to minimize injury. The actions taken immediately before, during, and after an attack will most likely influence the type and severity of the injuries.

Humans are rarely killed during an attack precipitated by a surprise close encounter, even though bears could do so easily and quickly. During these attacks, grizzlies are only trying to remove what they perceive as a threat, and their intent is to use only as much force as necessary. When interacting with others of their species, grizzly bears are head oriented, and they usually direct their aggression toward humans in the same manner—toward the head and neck. Therefore, the general rules to follow during a grizzly attack are to "help" the bear remove the perceived threat and to protect vital body parts, as follows:

1. Do not run, try to climb a tree, fight, or scream.
2. Drop to the ground and protect the head and neck by interlocking the hands behind the head (ear level) and flexing the head forward, either in the fetal position or flat on the ground face down (Figs. 52-20 and 52-21). Use elbows to cover the face if the bear turns you over.
3. Do not hold out a forearm or hand to ward off the attack. Bears can readily cause significant neurovascular injuries to these structures.

Figure 52-20. Curling into the fetal position to defend against a bear attack. (Courtesy Marilynn G. French.)

Figure 52-22. Standard government-issued bear-resistant food container. (Courtesy Marilynn G. French.)

Figure 52-21. Prone position to defend against a bear attack. (Courtesy Marilynn G. French.)

4. Never try to look at the bear during an attack because it could expose you to serious facial injuries.
5. After the attack, stay down until you are sure the bear has completely left the area. This is extremely important. Victims who have gotten up before the bear has left after the first attack generally received more severe injuries during the second one.
6. When you believe the bear has left the area, peek around while moving as little as possible, try to determine which way the bear went, evaluate options, and then leave the area.

In a close encounter situation, attack victims who immediately protected themselves and did not try to resist typically received minor injuries that could be treated on an outpatient basis. Victims who tried to run or fight the bear and those who left after the initial attack but before the bear had left the area typically received more severe injuries that required multiple surgical procedures, resulting in permanent cosmetic or functional disabilities.

If the attack is by a black bear, a different set of guidelines should be followed. Black bear aggression should be countered with aggression, such as shouting, yelling, throwing rocks or sticks, or whatever means are available. The victim should never lie down in a protective, submissive position because black bears are more likely to prey on humans they encounter at close range than are grizzly bears.

The data on polar bears are less complete but suggest that attacks by females with offspring are behavioral responses similar to those of grizzly females with offspring. The attacks are defensive, brief, and result in nonlethal injuries. In addition, the bear typically leaves shortly after the incident. If a polar bear is alone, however, a person should assume it is a male, whose behavioral response is more likely predatory, and should use any aggressive response available.

Prevent Predatory Behavior

The most important way to reduce the chance of being preyed on by a bear is to avoid anything that may attract a bear to the campsite while the occupants are sleeping, as follows:

1. Avoid camping along bear travel corridors or at seasonal feeding sites.
2. Avoid campsites littered with human refuse.
3. Use proper food storage to render human food unavailable to bears. Bear-resistant food storage containers are often provided at designated campsites in bear country (Fig. 52-22).
4. Reduce food odors by cooking and eating at a site away from the sleeping area. Do not sleep in clothes worn when cooking and eating.
5. Do not leave garbage or food buried or poured into the ground at the campsite. This can cause problems for future campers at this site.

The chance of a bear entering a campsite to prey on humans is small, but everyone in the camp should be familiar with a contingency plan. Everyone should know the area, even in the dark, and be aware of potential escape options, such as climbable trees or rocky ledges. Everyone should sleep in a tent because it offers a boundary of protection and may deter an inquisitive bear from walking directly to the campers. Although no study has proved its effectiveness, some people build a brush barrier around the tent to prevent a bear from readily approaching it.

Sleeping bags should be kept at least partially unzipped to facilitate a quick exit. In several instances, a victim trapped inside a sleeping bag has been dragged away from a campsite by a bear.

Each tent should be equipped with a flashlight. Pepper spray is useful, as well as a firearm, unless prohibited in that area. Again, a bear that enters a tent or picks up a sleeping camper is trying to prey on that person, so all available defenses should be used.

The behavior of a predatory bear is different from that of a bear responding to a close encounter. During a close encounter, a bear's response is driven by a defensive reaction, which can be aggressive and injurious. In contrast, the behavior of a predatory bear is driven by the desire for food. The bear is not looking for a confrontation or fight but rather a victim to drag from camp, usually only a few hundred feet, and to consume. Predatory grizzly and black bears rarely kill their victims before consuming them. They concentrate on soft tissue or visceral consumption, and the victims frequently remain alive for an hour or more. Therefore a quick, aggressive, and unified response by companions may save the victim's life. Surprisingly, yelling, throwing rocks, or striking the bear with a stick has been effective in driving the bear away from its victim. Approaching a predatory bear in the dark while it is feeding on a human is risky, but it is probably the victim's only chance for survival.

In contrast, the victim of a predatory attack by a polar bear is typically killed instantaneously, so prevention of such attacks is the only chance for survival. In all predatory attacks by polar bears, all defensive measures must be considered, including guns where permitted.

Special Considerations

Menstruation. In August 1967, two women were killed in separate events on the same night by different grizzly bears in Glacier National Park. The postmortem examination showed that one had been menstruating. The assumption that menstruation may be a precipitating factor in bear attacks has unfortunately become solidly ingrained into popular opinion. Hysterical coverage by the media enhanced this misconception, and the scientific question was left unanswered by both scientists and government officials.[2]

A study of polar bear response to menstrual odors was published in 1983.[7] Although it was not designed to adequately test the hypothesis that menstruating women were more likely to be either attacked or preyed on by bears, the press came to this conclusion. The Interagency Grizzly Bear Committee then printed an ambivalent caution in the government's official grizzly bear pamphlet ("Bear Us in Mind") that said, "Women may choose to stay out of bear country during their menstrual period." Fortunately, this has since been removed from the pamphlet, because no scientific evidence suggests that menstrual odors precipitate grizzly bear attacks. The attack mentioned previously is the only serious attack on a menstruating woman that has been documented in North America, and even the official investigating team at that time concluded that menstruation did not appear to have played a major role.[19]

Black bear researchers in North America report no evidence of black bears attacking or being attracted to menstruating women.[35] Furthermore, no evidence links menstruation to any of the 21 grizzly bear attacks in Yellowstone National Park from 1980 to 1994.[17,18]

Sexual Activity. A common concern among backcountry users is that sexual activity may attract bears and make them more aggressive toward campers. As with menstruation, these fears are based on hysteria and folklore. No anecdotal or scientific evidence supports this hypothesis.

Pepper Spray. Because of the desire for protection against aggressive bears, several methods were investigated for backcountry users. In the late 1970s and early 1980s, several compounds were tested for effectiveness in deterring grizzly bear attacks. The most effective method was an aerosol spray containing capsicum oleoresin, a derivative of red pepper. Captive grizzly bears were sprayed in the face at close range when they charged the researchers (who were outside the cage). Under these controlled conditions, red pepper spray was found to be highly effective in deterring a charging grizzly bear.

Pepper spray (5% to 10% capsicum oleoresin) is commercially available as personal protection against aggressive animals. The effectiveness of pepper spray used in the field against aggressive bears has been difficult to assess. It has been used in too few cases to draw firm conclusions. Also, it is impossible to conduct scientific studies testing its effectiveness under these conditions.

By 1985, pepper spray was used in the field against aggressive black and grizzly bears in 66 documented cases.[21] In general, it appeared more effective in deterring bears that charged after a close encounter than against food-conditioned bears in search of food. During the 1990s, however, most professional outfitters and guides in the northern Rocky Mountains began carrying pepper spray to deter aggressive grizzly bears, preferring spray over firearms. Although data are limited to anecdotal reports, pepper spray can be an effective and nonlethal alternative to repel aggressive grizzly bears, perhaps even more effective than firearms.

Carrying pepper spray, however, is not a substitute for knowledge of bear safety and good judgment to avoid aggressive encounters. If carried, spray must always be readily available, either in a belt-mounted holster or on a chest strap. It should be test-fired, and the user should practice drawing and firing it regularly (Fig. 52-23).

Despite a manufacturer's claim that pepper spray has an effective range of 9 m (30 feet), the effective range under field con-

Figure 52-23. Discharging a canister of pepper spray. (Courtesy Marilynn G. French.)

ditions is significantly less when there are headwinds or crosswinds. Preliminary field experience indicates that the oil-based pepper spray is more effective than the water-based spray. The author recommends a pepper spray that contains 10% capsicum oleoresin, has 2 million Scoville heat units, and comes in a 15-ounce pressurized aerosol container.

Unfortunately, people have used pepper spray in the same way they use mosquito spray. Despite its obnoxious and caustic smell, some sprayed it on themselves, as well as their tent, their sleeping bags, and the ground around their campsite. Once the aerosol has been released, the capsicum begins to lose its potency, and soon the active ingredient dissipates. At this point, bears may investigate the smell of pepper. Pepper spray so used thus becomes a bear attractant rather than a deterrent. One manufacturer of pepper spray (UDAP Industries, www.udap.com) provides a detailed instructional pamphlet on its proper use.

Pepper spray should be aimed toward a charging bear and discharged when the bear is within 6 to 9 m (20 to 30 feet). The person should continue spraying until the bear has stopped its charge, keeping the sprayer aimed at the bear in case it charges again. This continues until the bear has left the area. If the pepper spray is depleted, the best defense if the bear attacks is to lie down, cover the face, and offer little or no resistance. Again, there is no guarantee that pepper spray (or anything) will prevent injury by an aggressive bear.

If pepper spray is accidentally discharged into a person's face, it stimulates facial nociceptors and causes eyelid, ocular, and facial muscle spasms, which may result in temporary blindness. The victim should not rub the eyes (to avoid corneal abrasions) and should irrigate the eyes and skin vigorously with water for at least 15 minutes. Intraoral burning may be relieved by swishing and spitting milk or another casein-containing food product.[9]

Firearms. Many people consider carrying firearms for protection when they enter bear country. Guns can be useful in some situations. However, the target area to kill a grizzly with a shot to the head is only about 30 cm^2 (12 square inches). The cranial vault is narrow and sloped caudally (Fig. 52-24). A bear initiating a charge from a distance of about 45 to 55 m (50 to 60 yards) will take only 4 to 5 seconds to reach its victim.

Figure 52-24. Cranial vault of a bear. (Courtesy Timothy Floyd.)

Unless a proficient marksman, a person is unlikely to access a weapon, release the safety, aim accurately, fire, and hit a small target in a brief time under incredible emotional stress. Also, even if a shot could be fired, it probably would only wound the bear. Wounding a charging bear changes its behavior and may make its attack even more aggressive. Because of these factors, pepper spray should be considered as a nonlethal alternative to guns, especially when traveling in places where guns are not permitted, such as national parks.

According to most U.S. and Canadian wildlife agencies, the most effective firearms against grizzly bear attacks are a 12-gauge 3-inch magnum shotgun with 1-ounce slugs and a 30-06 rifle with a 200-grain bullet. A pistol is not considered to be an effective weapon against a charging grizzly. A large-caliber pistol, such as a 44-caliber magnum, can be used as a point-blank weapon to kill a bear attempting to prey. The effective ranges of a pistol and pepper spray are about the same, but it is easier to hit a moving target with pepper spray because of its shotgunlike aerosol pattern.

Dogs. In most national parks, dogs are not permitted in backcountry settings. Unfortunately, rare and questionable accounts report a dog stirring up a grizzly, then running back to its owner with the bear in pursuit. Most outfitters, guides, and hunters, however, report positive experiences with dogs in grizzly country. Their dogs are generally well trained and have been raised in wilderness environments. Most of these dogs can effectively deter grizzly and black bears from coming into a camp. Although no study has been conducted on the use of untrained dogs to deter aggressive bears, most people who spend considerable time in grizzly country use their dogs for this purpose.

Horses. For individuals concerned about an aggressive bear encounter, another option is the use of horses. No one has been injured by a grizzly or black bear while riding a horse. Horses that frequently travel in bear country are the best to use, because they generally do not react unpredictably and endanger the rider when they encounter bears. Although horses may protect against aggressive grizzly encounters, riders may still be injured by the horse. People are seriously injured or killed in horse accidents each year in the northern Rocky Mountains.

Hunter Safety. Many people participate in sport hunting in bear country each year. Some are hunting for bears, but most hunt for other game species.

For hunters of bears, the risks are obvious. Bear hunters intentionally break bear safety rules to close in on their prey. The most dangerous situation, however, is after they shoot and injure a bear. They have an ethical obligation to track the wounded animal and kill it, but this is when most bear hunters are injured. This confirms that guns are not completely effective in preventing bear injury. An injured bear may take refuge in heavy cover, then charge when the hunter is at close range. With little time, a surprised hunter often cannot fire a lethal round, and even when shot, the bear can continue its attack and cause significant injury before it dies.

Hunters of other game species in bear country are at significant risk of close encounter and injury. Besides violating bear

Figure 52-25. Bite wound injuries typical of a bear attack. (Courtesy Luanne Freer.)

safety rules, they frequently become preoccupied with the stalk and forget they may encounter a bear. During the 1990s, more than half the people injured by grizzly bears in the Yellowstone ecosystem were elk hunters. Some injuries occurred during the stalk, but other factors contributed. Grizzly bears in this ecosystem have learned the association between gunfire and available food. After an elk or other game (moose, deer, bighorn sheep) has been killed, hunters field-dress their kills and leave edible remains (gut piles) on the ground. In several cases, bears approached the kill site before the hunters completed this process. In other cases, an elk or animal was field-dressed late in the evening and then hung in a nearby tree. When the hunters returned the next morning, they encountered a grizzly bear that had claimed the gut pile or the carcass. Hunters must assume that under these circumstances, at least one grizzly bear will be at the site, and they must approach cautiously, preferably on horseback.

Bow hunting represents another high-risk activity. In most states, bow hunters are not allowed to carry a firearm as a backup weapon. They also tend to violate bear safety rules to set up a shot. Elk hunters blow an artificial elk call (a bugle or a cow call) to lure in bull elk. This also alerts grizzly bears that prey on adult male elk (bulls) during the breeding season. For protection, bow hunters should (1) hunt from a tree-stand, which provides some protection from grizzly bears, which are poor tree climbers, and (2) carry pepper spray and be prepared to use it.

BEAR-INDUCED INJURIES

Bear-inflicted injuries range from minor, treated on an outpatient basis, to complex, requiring hospitalization and surgery, typically resulting in significant cosmetic and functional disability. In this regard, bear attacks are similar to most other animal attacks, particularly those inflicted by large animals.

The character of such injuries is determined in part by the three main sources: teeth, claws, and paws. The teeth of bears, especially the canines, are large and sturdy. Although the teeth

Figure 25-26. Claw markings from a bear attack. (Courtesy Luanne Freer.)

are not particularly sharp, the power of the jaw muscles allows the teeth to penetrate deeply into soft tissues and fracture facial bones and bones of the hand and forearm with ease. The trauma characteristically results from punctures, with shearing, tearing, and crushing forces (Fig. 52-25).

The claws are another important source of trauma (Fig. 52-26). Although the claws on the front pads can be as long as human fingers, they are not particularly sharp on grizzly and polar bears. The bear's shoulders, however, provide the force and speed that allow claws to cause significant soft tissue damage in a scraping maneuver that results in deep, parallel gashes. Because black bear claws are sharper and more curved, the cuts tend to have sharper, less ragged edges.

The bear paw is capable of delivering a powerful force, resulting in significant blunt trauma, particularly to the head and neck, rib cage, and abdominal cavity, especially solid organ

rupture. Therefore, victims of bear attacks should be evaluated for occult blunt trauma.

Several victims of a bear attack were further injured when a companion accidentally shot them while trying to kill the attacking bear.[16] Others were injured when they fell out of a tree while escaping a bear; some sustained long-bone fractures. At least two persons in North America have been killed by such falls, and in both incidents the bear did not attack the victims once they fell to the ground.

WOUND MANAGEMENT

The specifics of initial wound treatment are determined in part by the available medical equipment and the location in which the victim is first received. Stabilization of the victim remains the primary objective. All victims of bear attacks should be considered to have major trauma and should be transported to the most appropriate facility after stabilization.

By the time most bear attack victims reach medical care, their injuries are relatively old. Bear-inflicted injuries are often occult, producing greater deep structure involvement and tissue necrosis than initially expected. Internal injuries from either direct penetration (claws, teeth) or blunt trauma are common. Neurovascular injuries must be considered with trauma to the extremities, and neurosensory and cosmetic injuries are common with trauma to the face.

ANTIBIOTIC THERAPY

There are only a few published studies concerning the oral flora of black or grizzly bears.[10,15,32] The bacterial spectrum of black and grizzly bears appear similar to that of dogs, although anaerobes, common in human, dog, and cat bites, have not been reported. In the few case reports available,[26,27,36] many victims received antimicrobial administration prior to hospitalization and prior to wound cultures. Nonetheless, there is no evidence that bear attack victims develop rare or unusual septic complications from unknown pathogens, with the exception of a single reported case of an atypical mycobacterial infection after a brown bear bite in Finland.[27]

The use of antibiotics shortly after the injury but before clinical evidence of infection remains controversial. The usual risk factors should be assessed (Box 52-1).[3] However, the blunt trauma, deep punctures, and shearing-and-tearing forces that are typical of bear attacks create significant tissue ischemia and necrosis that are not apparent on initial examination. In one study, nearly 50% of bear attack victims (four out of nine) developed clinical infections.[36] Therefore, antimicrobial prophylaxis should be considered for all bear-inflicted wounds pending culture results and before clinical evidence of infection.

Before culture results are available, victims of bear attacks should be treated with broad-spectrum agents to cover *Staphylococcus aureus* and gram-negative rods in addition to anaerobes. However, adequate wound debridement and cleansing are the primary means of reducing the infection rate. Antibiotics should be administered parenterally for at least 3 days after wounding and for at least 2 days after each debridement.[9]

Box 52-1. Animal Bite Risk Factors

HIGH RISK

Location
Hand, wrist, or foot
Scalp or face in infants
Through-and-through bite of cheek
Bites over vital structures (e.g., artery, nerve)
Bites over a major joint

Type of Wound
Puncture
Tissue crushing that cannot be debrided

Victim Characteristics
Older than 50 years
Asplenic
Chronic alcoholic
Altered immune status
Diabetic
Peripheral vascular insufficiency
Chronic corticosteroid therapy
Prosthetic or diseased cardiac valve
Prosthetic or seriously diseased joint

LOW RISK

Location
Face, scalp, ears, or mouth
Self-bite of buccal mucosa

Type of Wound
Large, clean lacerations that can be cleansed
Partial-thickness lacerations and abrasions

Victim Characteristics
Younger than 50 years
Good medical health

RABIES

No case of rabies has been reported or documented in either wild or captive bears. The Centers for Disease Control and Prevention (CDC), however, recommends rabies immunization for victims attacked by wild carnivores. Therefore, all victims of bear attacks should receive the standard informed-consent discussion of the risks and benefits of rabies immunization.

The references for this chapter can be found on the accompanying DVD-ROM.

Wilderness-Acquired Zoonoses

53

Jamie Shandro and Daniel Garza

A zoonosis is an infectious disease that may be transmitted from animals to humans under natural conditions. There are more than 200 zoonotic pathogens. The risk of acquiring zoonoses increases proportionately with the frequency and intensity of contact with animals. For example, hunters and trappers who handle and are exposed to the blood, viscera, secretions, and excretions of wild animals are at much greater risk than are recreational campers. Similarly, international travelers who frequent locations with a much higher density of infected animals are at greater risk for infection. The trekker in Nepal is more likely to confront rabies than is the hiker in California. Disease severity is important in human infections.

This chapter emphasizes diseases in which wildlife plays a significant role in transmission to humans. Rabies is discussed in a separate chapter. Although glanders and melioidosis do not fit this pattern, they are also discussed. Zoonoses acquired primarily from domestic animals that also have a minor reservoir in wildlife are mentioned briefly; standard texts of veterinary public health[104,198,213] and infectious disease[94,139] provide further discussion.

ANTHRAX

Anthrax is a traditionally zoonotic disease that has come to the forefront most prominently as a result of bioterrorism. It still deserves attention as a disease potentially acquired through exposure to animals, particularly in the developing world.

Anthrax has afflicted men and beasts for centuries.[214] The word *anthrax* comes from the Greek word *anthrakites* for coal, referring to the black eschar seen in cutaneous anthrax. Anthrax is believed to have been one of the Egyptian plagues that affected cattle at the time of Moses.[67] Virgil described clinical anthrax as the murrain of Noricum in his works on agriculture, *The Georgics:* "The pelts of diseased animals were useless, and neither water or fire could cleanse the taint from their flesh. The sheepmen could not shear the fleece, which was riddled with disease and corruption, nor did they dare even to touch the rotting strands. If anyone wore garments made from tainted wool, his limbs were soon attacked by inflamed papules and a foul exudate."[231]

In the mid-19th century, anthrax was called woolsorters' and ragpickers' disease in England and Germany because workers contracted the disease from working with hides and fibers contaminated with anthrax spores. The anthrax bacillus was the model first used by Robert Koch in the 1870s in the development of his postulates on the germ theory of disease. Louis Pasteur developed the first anthrax vaccine for livestock in 1881.

Because anthrax spores (Fig. 53-1) are resistant to heat and drying and may remain dormant for decades, they have long been considered a potential biological weapon. Germ warfare programs in the Soviet Union during the 1920s and 1930s included development and stockpiling of anthrax spores. The first report of anthrax spores used as a weapon of war was in the 1940s, when the Japanese army used them in Manchuria during the Chinese-Japanese war.[100] Japan killed thousands of Chinese in widespread attacks with anthrax, typhoid, and plague in its assault on Manchurian towns and cities. *Bacillus anthracis* spores were accidentally released at a military facility in Sverdlovsk in 1979, resulting in 66 deaths.[153] In 2001, the intentional contamination of U.S. mail with *B. anthracis* spores turned attention once again to anthrax as an agent of bioterrorism as well as a zoonotic disease.

Bacteriology

Anthrax is caused by *Bacillus anthracis* bacteria (Fig. 53-2), which are nonhemolytic, nonmotile, spore-forming, gram-positive rods that grow well on blood agar. *B anthracis* has a polysaccharide cell wall antigen and an anthrax toxin. Anthrax spores are resistant to heat, drying, ultraviolet light, and disinfectants, and they may survive up to decades in soil.[236] Infections are initiated by skin, pulmonary, or gastrointestinal (GI) contact with endospores, which are phagocytosed by macrophages and carried to regional lymph nodes.[69] Inside macrophages, the spores germinate into vegetative bacteria, which can then rapidly divide and initiate further spread of infection.

Epidemiology

Most animals are susceptible to anthrax to some degree, but clinical anthrax is primarily a disease of herbivores, such as sheep, cattle, horses, and goats. The disease is rarely seen in countries where vaccination of herbivore stock is practiced. Outbreaks among herbivores are thought to occur under environmental conditions that are favorable for bacterial multiplication, such as where the pH is higher than 6.0 and the soil is rich in organic matter.[206]

Anthrax is rare in developed nations with aggressive vaccination of livestock, but it is still problematic in areas of Asia and Africa with sporadic vaccination and significant wildlife reservoirs. Most cases in industrialized nations are seen after exposure to contaminated animal products—for example, goat

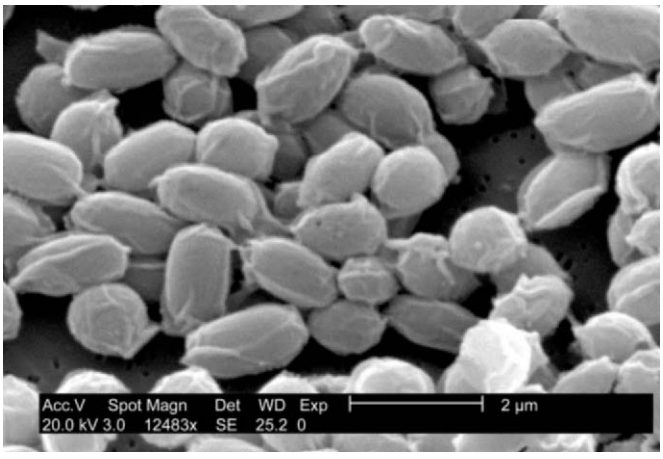

Figure 53-1. Anthrax spores. (Available at http://bepast.org/docs/photos/Anthrax/anthrax%20spores.jpg.)

Figure 53-3. Cutaneous anthrax lesion on the skin of the forearm caused by the bacterium *Bacillus anthracis.* (From CDC Public Health Image Library, available at http://phil.cdc.gov/phil/home.asp.)

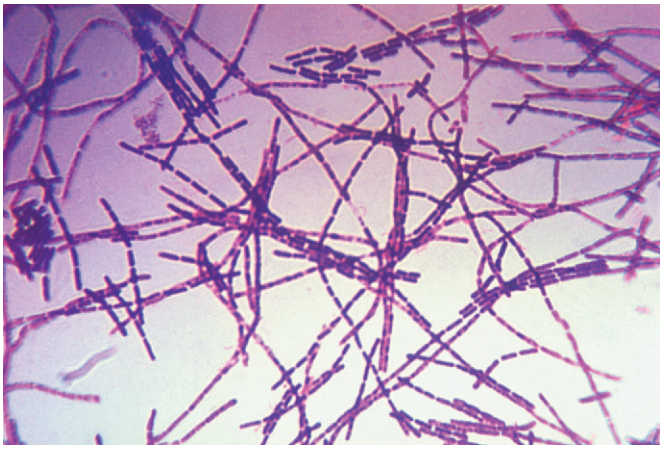

Figure 53-2. A photomicrograph of *Bacillus anthracis* bacteria using Gram stain technique. Anthrax is diagnosed by isolating *B. anthracis* from the blood, skin lesions, or respiratory secretions, or by measuring specific antibodies in the blood of persons with suspected cases. (From CDC Public Health Image Library, available at http://phil.cdc.gov/phil/home.asp.)

hair imported from Turkey and Pakistan.[256] Shepherds, farmers, and workers in industrial plants with potentially contaminated animal products are at highest risk. Most recently, anthrax has been associated with bioterrorism events.

Transmission

Humans are infected by anthrax through contact with infected animals or animal products, or through a preparation of spores in the case of bioterrorism attacks. There has been no documented human-to-human transmission of anthrax.[69] Transmission occurs through direct contact with spores, ingestion of spores, or inhalation of spores.

Presentation and Symptoms

There are three forms of anthrax infection: cutaneous, inhalational, and gastrointestinal. The forms are defined by the route of entry of spores into the human body as well as by the constellation of presenting symptoms.

Cutaneous anthrax is most often acquired by close contact with infected animals. The primary skin lesion typically begins as a nondescript, painless, and pruritic papule within 3 to 5 days of exposure. This progresses within 1 to 2 days to a vesicle that undergoes necrosis and drying to leave the characteristic black eschar surrounded by edema (Fig. 53-3).[69]

Gastrointestinal anthrax, which is more common outside of the United States, is caused by ingesting *B. anthracis* spores.[15] The two forms of gastrointestinal anthrax include oropharyngeal and intestinal, each with an incubation period of 1 to 6 days.

Symptoms of oropharyngeal anthrax include fever greater than 39°C (102.2°F), severe sore throat, dysphagia, posterior oropharyngeal ulcers, and regional lymphadenopathy. Oropharyngeal lesions begin as edematous ulcerations that variably progress to ulcerations with central necrosis and then a pseudomembranous covering. The oropharyngeal form of gastrointestinal anthrax is also associated with significant soft tissue neck swelling, which may progress to the point of upper airway obstruction.

The intestinal form of gastrointestinal anthrax is characterized by anthrax spore infection of the stomach or bowel wall. Initial symptoms include nausea, vomiting, anorexia, and fever greater than 39°C (102.2°F). Symptoms then progress to include severe abdominal pain, hematemesis, and/or watery, melanotic, or bloody diarrhea. Patients can present with symptoms of an acute abdomen or new-onset ascites. Mortality can result from bowel perforation or *B. anthracis* septicemia.

Inhalational anthrax is rare worldwide and is associated most prominently with bioterrorist attacks such as that seen in the United States in 2001. Inhalational anthrax has an incubation period of 8 to 10 days after inhalation of spores into the airways.[28] Symptoms include an insidious flulike onset of malaise, fatigue, fever, nonproductive cough, and myalgia.[206] The rapid deterioration that follows includes dyspnea, cyanosis, respiratory failure, meningismus, mediastinal hemorrhage, hypotension secondary to septic shock, and possibly death.

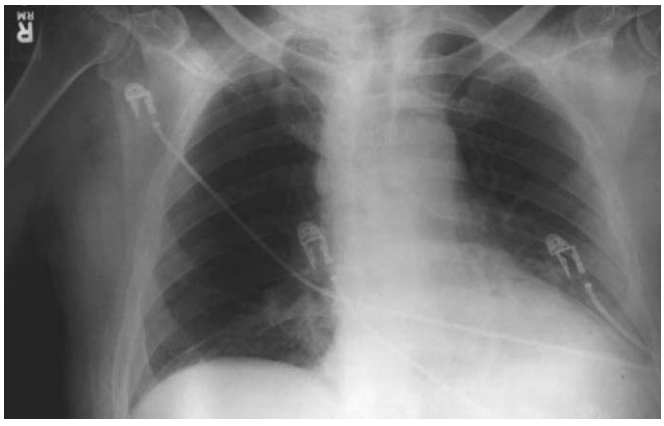

Figure 53-4. Anthrax-widened mediastinum. (From Jernigan JA, Stephens DS, Ashford DA, et al., members of the Anthrax Bioterrorism Investigation Team: Emerg Infect Dis 7:933–944, 2001.)

Of the three forms of disease, inhalational anthrax has the highest mortality rate (45% with antibiotic treatment and 97% without), followed by gastrointestinal anthrax (40% with antibiotic treatment), and cutaneous anthrax (1% with antibiotic treatment and 10% to 20% without).[109,206]

Diagnosis

Cutaneous anthrax can be diagnosed by culture of cutaneous lesions. Gastrointestinal anthrax can be diagnosed by cultures from oropharyngeal lesions, blood, and ascitic fluid. Computed tomography (CT) of the abdomen is likely to show mesenteric adenopathy. Stool culture has not been shown to be useful in aiding the diagnosis of anthrax.

Patients with inhalational anthrax typically present with a normal to elevated white blood cell count. Chest radiographs typically demonstrate mediastinal widening secondary to hilar adenopathy, and they frequently demonstrate pleural effusions (Fig. 53-4).[109] Cultures of blood, sputum, and cerebrospinal fluid (CSF) should be obtained for analysis in suspected cases of inhalational anthrax.

Treatment

Effective treatment of all forms of anthrax requires a high index of suspicion and prompt antibiotic therapy. In the 2001 outbreak of bioterrorism-related anthrax, all isolates were susceptible to ciprofloxacin and doxycycline, as well as a number of other agents.[45] Because *B. anthracis* has the potential for penicillinase and cephalosporinase activity, ciprofloxacin and doxycycline, rather than penicillin-based antibiotics, are the first line of therapy.

For active disease, treatment for cutaneous anthrax is oral ciprofloxacin or doxycycline twice daily for 60 days. In children, the dosage is ciprofloxacin 10 to 15 mg/kg/day divided every 12 hours and not to exceed the adult dosage of 500 mg every 12 hours, or doxycycline 4.4 mg/kg/day divided every 12 hours and not to exceed the adult dosage of 100 mg every 12 hours. If systemic symptoms are present, treatment should be initiated with intravenous agents. If patients are clinically improved, therapy may be changed to amoxicillin 500 mg three times daily for adults or 80 mg/kg/day divided three times daily for children.

For inhalational and gastrointestinal anthrax, treatment begins with intravenous therapy of ciprofloxacin or doxycycline as well as one or two additional agents (options include rifampin, chloramphenicol, vancomycin, penicillin, ampicillin, imipenem, clindamycin, and clarithromycin). If symptoms improve, treatment switches to an oral regimen of ciprofloxacin or doxycycline for a total course of 60 days. Treatment is the same during pregnancy; the risk of doxycycline or ciprofloxacin during pregnancy is outweighed by the potential mortality resulting from undertreated anthrax infection.

In the event of potential exposure to inhaled anthrax spores, the Centers for Disease Control and Prevention (CDC) recommends 60 days of ciprofloxacin or doxycycline in combination with a three-dose regimen (0, 2 weeks, 4 weeks) of anthrax vaccine (BioThrax, formerly known as AVA) as an emergency public health intervention. There is no recommended post-exposure prophylaxis for exposure to cutaneous anthrax alone.

Prevention

Prevention of zoonotic anthrax infection largely relies on vaccination of animals at risk. Travelers should avoid contact with infected animals and undercooked meat.

BACILLARY ANGIOMATOSIS

Bacillary angiomatosis, first described in 1983 during the early years of the acquired immunodeficiency syndrome (AIDS) epidemic, forced a reconsideration of cat-scratch disease (CSD), bartonellosis, and trench fever.[215] In the early 1990s, it was determined that organisms of the genus *Rochalimaea* caused a diverse array of clinical syndromes, including cutaneous bacillary angiomatosis, bacillary peliosis hepatitis, fever with bacteremia (*Rochalimaea* bacteremic syndrome), and CSD. Whether the different clinical syndromes result from subtle differences in the infecting organisms or in the response of the immune system remains unclear. Each of these conditions can be caused by the bacteria now known to be *Bartonella* (formerly *Rochalimaea*) *henselae* and *Bartonella quintana*, also the agent of trench fever.

Epidemiology

Although clinically different from CSD, bacillary angiomatosis is also closely associated with a recent cat scratch or bite. The vast majority of victims are human immunodeficiency virus (HIV) positive, but a few are not and appear otherwise well. In 34% of cases, this infection was the first one to establish the diagnosis of AIDS in a given patient. AIDS patients with bacillary angiomatosis can die if untreated, but erythromycin is usually effective.[19]

Symptoms

Most cases of bacillary angiomatosis involve cutaneous or subcutaneous lesions. The lesions are typically elevated, friable, red granulation tissue papules resembling pyogenic granulomas, numbering a few to thousands (Fig. 53-5). Deeper subcutaneous nodules are seen in about half of the victims. Similar lesions can occur in other body tissues.[19] Visceral lesions may be the first sign of infection; patients often have fever, weight loss, and malaise.[62] Hepatic involvement can lead to hepatic failure or even rupture (bacillary peliosis hepatitis). This usually presents with GI symptoms (nausea, vomiting, diarrhea, or abdominal distention), fever, chills, and hepatosplenomegaly. Histopatho-

Figure 53-5. Bacillary angiomatosis. (Available at www.prn.org/images/prn_nb_cntnt_images/vol2num4/v2n4a2_bacillary__angiomatosis_ir.jpg.)

logic examination of liver biopsy specimens reveals dilated capillaries or multiple blood-filled cavernous spaces. The organism can also cause bacteremia, even in immunocompetent patients. *Rochalimaea* bacteremic syndrome is characterized by a prolonged symptom complex of malaise, fatigue, anorexia, weight loss, and fevers that are recurring, with ever-increasing temperatures.[12,17,18] No site of focal infection is apparent. The symptoms are usually present for weeks to months before the diagnosis is finally made by isolation of the organism in blood cultures.[209]

Diagnosis

B. henselae and *B. quintana* can be isolated from blood using lysis-centrifugation blood cultures,[11,12,21] but both species have also been isolated with traditional blood culture systems.

Serologic diagnosis can also be made using techniques of indirect fluorescent antibody (FA) testing. Serum samples can be sent (for both *B. henselae* and *B. quintana*) to the CDC. An enzyme immunoassay for the detection of IgG antibodies to *B. henselae* is now commercially available and is reportedly 5 to 10 times more sensitive than the indirect FA test.[63] Positive results should be interpreted cautiously, taking into account the clinical context, because the meaning of positive serologic results awaits further evaluation with stricter epidemiologic methods.

Diagnosis of bacillary angiomatosis is usually made from clinical features and biopsies of lesions, with characteristic histopathologic findings in tissue sections. Blood cultures should be obtained and incubated for a prolonged period. As more is learned about the growth requirements of the causative organisms, they may become easier to culture directly from skin lesions and lymph nodes. Serologic analysis will become an important means of diagnosis.[1]

Treatment

Treatment is with erythromycin. If patients cannot tolerate this therapy, rifampin and doxycycline or trimethoprim-sulfamethoxazole should be given. Norfloxacin, gentamicin, and ciprofloxacin are also clinically effective.[199] Penicillin and first-generation cephalosporins are not beneficial. Therapy is for a minimum of 6 weeks and may have to continue indefinitely in an immunosuppressed patient.

Rights were not granted to include this figure in electronic media. Please refer to the printed book.

Figure 53-6. Electron micrograph of *Brucella abortus*. (Courtesy Dennis Kunkel Microscopy, Inc.)

BRUCELLOSIS

Brucellosis is an anthropozoonotic disease with a broad clinical spectrum caused by a number of species of *Brucella*, a small, gram-negative bacterium (Fig. 53-6). The bacterium can survive in soil for up to 10 weeks, in goat cheese for up to 180 days (at 4° to 8° C [39.2° to 46.4° F]), and in tap water for up to 60 days. It is, however, very sensitive to heat and most disinfectants, and it is entirely killed by pasteurization. Brucellosis usually results from the ingestion of contaminated milk or milk products or by direct skin contact. *Brucella* organisms are carried chiefly by swine, cattle, goats, and sheep and may be recovered from almost all tissues in a sick patient. Most animals used as livestock are susceptible to brucellosis, whereas the occurrence in wild animals is rather small.[146]

Epidemiology

Brucella in domesticated animals tends to be species specific, with *Brucella abortus* infecting cattle, *Brucella melitensis* goats, *Brucella suis* swine, *Brucella canis* dogs, and *Brucella ovis* sheep. The disease is found worldwide and has an annual attack rate of approximately 500,000; U.S. cases number less than 200. Internationally, most cases are transmitted by ingestion of fresh, nonpasteurized goat cheese and raw goat's milk. The disease caused by *B. melitensis* has a higher attack rate and is more severe, whereas brucellosis in the United States is usually caused by *B. abortus* from cattle, which produces mild clinical disease and is usually occupationally acquired. A proven case of transmission by dog bite has been reported, and dogs carry their own pathogenic species.[176]

Symptoms

Many organ systems may be involved, with a wide range of disease severity and acuity.[56] The disease can be classified into

three forms: acute, subacute, and chronic. In acute brucellosis, the victims complain of headache, weakness, diaphoresis, myalgias, and arthralgias. This is the most common presentation. Anorexia, constipation, and weight loss are often seen in the first 3 to 4 weeks. Physical examination reveals hepatomegaly and splenomegaly. Bacteremia in the early stages typically induces lesions of the viscera, bones, and joints; osteomyelitis, particularly spondylitis, is a common complication.

In subacute, or undulant, brucellosis, symptoms are milder but with more frequent arthritis and orchitis. The clinical picture is more varied, and the diagnosis is considered in any fever of undetermined origin. In the pre-antibiotic era, most patients spontaneously cleared their disease in 6 to 12 months.

In chronic brucellosis, symptoms have persisted for more than 1 year. It is rare in children but increasingly common as the patient ages. Many describe arthralgias and extra-articular rheumatism. Chronic brucellosis can mimic chronic fatigue syndrome.

Diagnosis

Brucellosis is most often diagnosed by serologic testing, including rose bengal, immunofluorescence, the complement fixation (CF) test, and the enzyme-linked immunosorbent assay (ELISA).[133] After acute infection, high titers may persist for 18 months. False-positive results may be caused by *Francisella tularensis* or *Yersinia enterocolitica* infection. The isolation of *Brucella* organisms by blood culture is also used for definitive diagnosis. Blood cultures incubated in 10% carbon dioxide is a common method, with sensitivities of 50% to 80%.[236] Bone marrow biopsy and culture can be used in patients with clinically suspected brucellosis but negative serologic tests and blood cultures.

Treatment

Oral tetracycline, 50 mg/kg/day in four divided doses for 21 days, is used to treat brucellosis. Doxycycline may also be used. In severe cases, streptomycin, 20 to 40 mg/kg intramuscularly once a day for 1 week, is added to this regimen. The next week, streptomycin is continued at a level of 15 mg/kg. Alternatively, rifampin can be added to doxycycline.[55] The role of quinolones has been investigated.[3] The prognosis is generally excellent, with only two deaths reported in several thousand cases.[82]

CAT-SCRATCH DISEASE

Cat-scratch disease is transmitted through a break in the skin (bite, scratch, or other injury) caused by a cat. First recognized by Robert Debré at the University of Paris in 1931, the disease was not officially reported until 1951. CSD is relatively rare but is probably the most common cause of unilateral lymphadenopathy in children.[211] The current cause of CSD is thought to be the gram-negative rod, *Bartonella henselae*.[25]

Epidemiology

CSD has been reported from all countries and in all races. An estimated 24,000 cases are recognized each year in the United States.[143] Compared with healthy cat-owning control subjects, patients with CSD are more likely to have at least one kitten 12 months of age or younger, to have been scratched or bitten by a kitten, and to have at least one kitten with fleas.[255]

Transmission

About 90% of cases are caused by scratches from cats, but dog and monkey bites, as well as thorns and splinters, have also been implicated.[57] The organism may be on the claws or in the oral cavity of the offending cat. Most cases occur in children, particularly boys, who tend to play more aggressively with domestic animals.

Symptoms

The average incubation period is 3 to 10 days. The characteristic feature of CSD is regional lymphadenitis, usually involving lymph nodes of the arm or leg. In one series, 54% of lymphadenopathy occurred in the axilla, with the remainder in the neck.[211] Often, only one node is involved. The nodes are often painful and tender, and about 25% suppurate.[235] Adenopathy may spread proximally; occasionally, cervical adenopathy is mistaken for Hodgkin's disease. In most cases, a characteristic raised, erythematous, slightly tender, and nonpruritic papule with a small central vesicle or eschar that resembles an insect bite is seen at the site of primary inoculation. Constitutional symptoms are mild, with approximately two thirds of patients manifesting fever, which is rarely greater than 38.8°C (102°F). Chills, malaise, anorexia, and nausea are common. Infrequent evanescent morbilliform and pleomorphic skin rashes lasting for 48 hours or less have been reported. This typical clinical course occurs in 88% of victims; the remainder seek medical treatment for complications such as encephalopathy, atypical pneumonia, and severe systemic disease. Parinaud's oculoglandular syndrome of granulomatous conjunctivitis and an ipsilateral, enlarged, tender preauricular lymph node occurs in about 6% of cases.[144]

Serious complications are rare and include encephalitis, seizures, transverse myelitis, osteolytic bone lesions, arthritis, splenic abscess, mediastinal adenopathy, optic neuritis, and thrombocytopenic purpura.[24,144,158,169] Although encephalopathy is rare, CSD is becoming a more common cause of encephalopathy as other viral infectious diseases disappear; the incidence of CSD-associated neurologic complications now ranks with those of varicella and herpes simplex infections, Lyme disease, Rocky Mountain spotted fever, and Kawasaki disease.[30] CSD encephalopathy should enter the differential diagnosis of patients (especially young ones) with unexplained coma or seizures (half of whom may be afebrile). The prognosis for encephalopathy generally is good.

Diagnosis

Results of routine laboratory studies, including urinalysis and complete blood cell count, are usually normal, although mild leukocytosis may be seen. An indirect FA test for *B. henselae* is now commercially available.

Immunity is thought to be largely cell mediated.[90] An intradermal skin test of 0.1 mL of CSD antigen is positive in approximately 95% of victims, although 10% of the population has a false-positive reaction. In confusing cases, biopsy of lymph nodes can yield characteristic findings of areas of granulomatous change and necrosis with central neutrophilic infiltration, a peripheral zone of histiocytic cells, and an outermost zone infiltrated by small lymphocytes and plasma cells.[141] This picture is not diagnostic, however, and is also seen in lymphogranuloma venereum (LGV), histoplasmosis, tularemia, brucellosis, sarcoidosis, and tuberculosis. Thus, lymph node biopsy is most useful to rule out malignancy. Warthin-Starry or

Brown-Hopps staining of the nodes or the primary skin lesion usually demonstrates the small, pleomorphic bacilli.[144]

In most cases, clinical diagnosis is based on finding three of four criteria: (1) single or regional lymphadenopathy without obvious signs of cutaneous or throat infection, (2) contact with a cat (usually an immature one), (3) detection of an inoculation site, and (4) a positive skin test.[33]

The workup should exclude other causes of regional lymphadenopathy, such as tuberculosis, tularemia, LGV, lymphoma, brucellosis, and sporotrichosis.[196] In general, only sporotrichosis and LGV demonstrate localized unilateral lymphadenopathy; LGV usually occurs in the groin. Cat scratches are normally found on the upper extremities. Skin tests, cultures, serologic tests, and biopsies are available for the differentiation of these other diseases.

Treatment

CSD usually resolves spontaneously in weeks to months, although in 2% of persons (usually adults) the course is prolonged and involves systemic complications.[144] Systemic CSD in an adult has been successfully treated with gentamicin,[131] and in a child, with cefuroxime.[91] In a retrospective series of 71 patients, trimethoprim-sulfamethoxazole was seen to have had good results; this was not the case with other antibiotics.[54] The best study, of 202 patients, is also retrospective, but found a response rate of 87% with rifampin, 84% with ciprofloxacin, 73% with intramuscular gentamicin, and 58% with trimethoprim-sulfamethoxazole.[142] Antibiotics that were of no benefit included amoxicillin-clavulanate, erythromycin, dicloxacillin, cephalexin, tetracycline, cefaclor, ceftriaxone, and cefotaxime. Antibiotics should be reserved for persons with severe or very prolonged disease.[142] Symptomatic treatment and reassurance that the prognosis is excellent are the best therapies. No sequelae of CSD other than the rare complications mentioned are known.

GLANDERS

Although little known in the Western world today, glanders is one of the classic infectious diseases. Its greatest historical impact has been through its effect on cavalry horses during military campaigns, influencing battles from before the time of Christ through the Crusades and World War I.

Theories and disputes about the origin, nature, transmission, and treatment of glanders figured prominently in the development of veterinary science in Europe in the latter half of the 18th century. In 1795, Erik Viborg published an account that is remarkably close to our understanding of the disease today. He demonstrated that equine "farcy," characterized by cutaneous lymphangitis, and the respiratory form of the disease in horses, classically referred to as glanders, were different manifestations of the same infection. He demonstrated that the disease was transmissible from one horse to the next by infectious exudates, and that the causative organism could be carried by fomites and killed by heat.

Transmission of glanders from horses to humans was documented in France and Germany during the first 3 decades of the 19th century. The causative organism was isolated by Loeffler and Schütz, as well as by Bouchard, Capiton, and Charrin in 1882. In 1891, Kalning and Helmann independently discovered mallein, derived from the glanders bacillus. Like tuberculin, mallein was thought to have therapeutic or prophylactic value. This turned out to be erroneous, but mallein provided a means of diagnosing the infection in clinically ill and carrier animals and provided a basis for test and slaughter techniques, which have largely eliminated glanders from most parts of the world.

Bacteriology

The causative organism is *Burkholderia mallei,* a member of the newly renamed *Burkholderia* genus, which includes *Burkholderia pseudomallei,* the cause of melioidosis, and *Burkholderia cepacia.* It is a gram-negative, nonsporulating, obligately aerobic, and nonmotile bacillus that requires glycerol for optimum growth in vitro.[175,212] In 1992, Yabuuchi and colleagues[250] proposed that seven species, formerly of the *Pseudomonas* RNA group II, should be transferred to a new genus, *Burkholderia*, with *B. cepacia* as the type species. The genus included *Burkholderia caryophylli, Burkholderia gladioli, B. mallei, B. pseudomallei, Ralstonia pickettii,* and *Ralstonia solanacearum;* the latter two species were transferred to the genus *Ralstonia.*[250]

Epidemiology

Glanders occurs in a few Asian and African countries, such as India, China, Mongolia, Egypt, and Mauritania. It is primarily a disease of horses and is spread most rapidly when large numbers of horses, mules, or donkeys are kept in proximity. Many carnivorous mammals are also susceptible to infection, and outbreaks have occurred when infected horse meat was fed to lions, tigers, and other wild animals in zoos. Occasionally, infections occur in dogs, cats, sheep, and goats.

Transmission

Humans are usually infected by exposure to sick horses. Infection can occur by inhalation of respiratory droplets or by contact with infected discharges. Human infections have also occurred from direct contact in the laboratory and from patients.

Symptoms in Equids

Horses may have unilateral or bilateral mucopurulent nasal discharge. There may be enlargement and induration of lymphatics, with ulceration and discharge, especially involving the legs. Nodules, pustules, and ulcers may be seen on the horse's skin. The cutaneous form of the disease is often referred to as farcy, the thickened, inflamed lymphatics as farcy pipes, and the enlarged lymph nodes as farcy buds. Horses also have pneumonia, with mild respiratory embarrassment in early stages, and more severe respiratory difficulties and cachexia in later stages. Septicemia with lesions in multiple internal organs can occur.

Glanders can run an acute and fulminant course in equids, more often in donkeys and mules, or a more chronic course, more often in horses. The case-fatality rate is high, especially with more virulent strains of the organism.

Symptoms in Humans

The incubation period of glanders in humans can be as short as 1 to 5 days. Cases with apparent incubation periods of several months may have represented smoldering, unrecognized infection. The severity of disease can vary from mild to fatal, and the course can be acute and fulminant or chronic. Relapses can occur after quiescent periods of up to 10 years. As in horses, manifestations in humans usually involve the skin and respiratory tract. There may be pustular cutaneous eruptions, thick

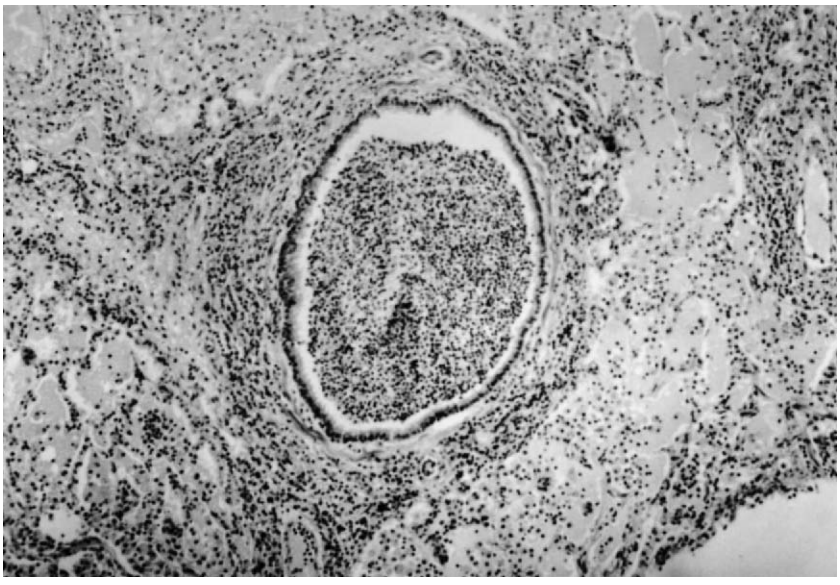

Figure 53-7. A bronchus filled with and surrounded by pus in the lung of a horse with glanders. (Hematoxylin and eosin, ×100.)

indurated lymphatics that may ulcerate, mucopurulent discharge from the eyes or nose, pneumonia, and metastatic abscesses in internal organs. Depending on the severity, the patient may have anorexia, fever, weight loss, headache, nausea, diarrhea, or septicemic shock. Lobar pneumonia, bronchopneumonia, or nodular densities may be seen on chest radiographs. Cases recently reported from Southeast Asia have been relatively mild, indicating that the local strain of the organism appears to have moderate pathogenicity for humans.

Diagnosis

Clinical diagnosis in horses based on symptoms can be confirmed by reaction to mallein with a cutaneous hypersensitivity test. Mallein, a filtrate derived from culture of the organism, is injected into the eyelid of a horse. A positive reaction, read 48 hours later, consists of marked local swelling and purulent conjunctivitis. Several serologic tests are also available. The CF test is often used. A dot-ELISA is a more sensitive test.[229]

Clinical diagnosis in humans is based on compatible symptoms in an individual exposed to horses in an endemic area. The diagnosis can be confirmed by culture of the organism from lesions or by serologic testing, using CF or agglutination tests. Agglutination titers are often detectable by the second week of infection. The CF test is less sensitive but more specific than agglutination. CF tests become positive during the third week of infection.[175]

Laboratory diagnosis can be made by injection of infected material intraperitoneally into male guinea pigs or hamsters. The animals develop peritonitis that extends into the scrotal sac with severe inflammation—the Strauss reaction.

In acute phases of the disease, abscessation occurs. Later, the inflammatory focus is surrounded by a granulomatous reaction, but central karyorrhexis remains a prominent feature of the lesion. The lungs are the internal organs most typically involved (Figs. 53-7 and 53-8), but septicemic glanders can involve liver, spleen (Fig. 53-9), bone, or brain. With chronic infection, multiple subcutaneous and intramuscular abscesses may develop.

Treatment

Early studies indicate that sulfadiazine, 100 mg/kg/day in three divided doses given for 3 weeks, is effective. Treatment with tetracyclines and streptomycin is also recommended.[175] The organism is sensitive in vitro to sulfamethizole, sulfathiazole, trimethoprim-sulfamethoxazole, gentamicin, kanamycin, streptomycin, and tetracycline.[4] Ciprofloxacin and ofloxacin, but not norfloxacin, were found to be effective in treating experimentally infected guinea pigs and hamsters.[13] Therapy should be based on culture and sensitivity testing of isolates and on clinical response to treatment.

Acute untreated septicemic cases are almost uniformly fatal within 7 to 10 days.[175] The prognosis is better in chronic forms of the disease, which can last for years, but deaths are still likely without adequate treatment.

Prevention and Control

The only significant reservoir of infection in nature is equids. If the disease were eradicated in them, it would disappear. National programs should be instituted in enzootic countries to eradicate the infection, by mallein or serologic tests of all horses, donkeys, and mules and slaughter of reactive animals. Persons who handle horses in enzootic countries, including trekkers who pack gear into the wilderness on these animals, should be advised of the signs of glanders in equids and warned to avoid contact with sick animals.

Glanders can be transmitted from one person to another, so strict infection control should be exercised with suspected infected patients. Personnel should avoid contact with all secretions and respiratory droplets. Transmission is also a risk in the laboratory, so if this organism is being cultured, all work should be done under appropriate microbiologic hoods.

LEPTOSPIROSIS

Leptospirosis is an infectious disease caused by *Leptospira interrogans*. It can be acquired by animals and humans, usually by

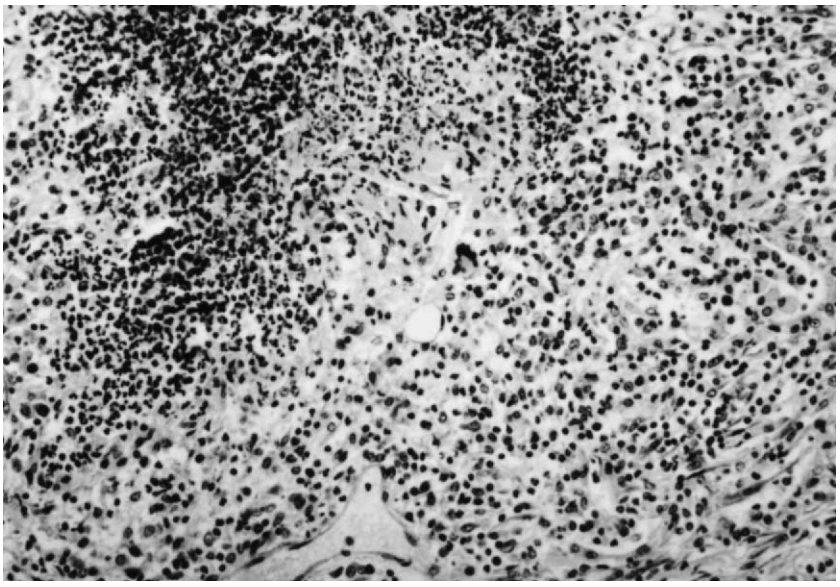

Figure 53-8. Gangrenous pneumonia with characteristic karyorrhexis in the lung of a horse with glanders. A multinucleated giant cell is in the center of the figure. (Hematoxylin and eosin, ×250.)

exposure to contaminated urine of wild or domestic animals. Adolf Weil first described the clinical picture of human leptospirosis in 1886. He described four febrile men with "particularities of an acute infectious illness with spleen tumor, jaundice, and nephritis." In addition, each had "severe nervous symptoms" and an enlarged liver. All recovered, and three had a biphasic clinical course with fever recurring after an afebrile period of 1 to 7 days. The term *Weil's disease* was coined by Goldschmidt in 1887. The carrier status was described in asymptomatic field mice by Ido and coworkers in 1915. Since then, the infection has been recognized both as a disease and as an asymptomatic carrier state in hundreds of animal species.

Bacteriology

Leptospira is a genus in the order Spirochaetales, an order of organisms characterized by thin, helical, gram-negative bacteria. More than 170 serovars, or serologically distinct strains of *Leptospira,* are known. *Leptospira* organisms are spirochetes with hooked or curved ends, 6 to 20 µm long by 0.1 µm wide (Fig. 53-10). They can grow on artificial media containing rabbit serum, such as Fletcher's semisolid and Stuart's liquid media, or on media containing albumin and fatty acids, such as Ellinghausen-McCullough-Johnson-Harris (EMJH) medium.[217]

In describing outbreaks with shared epidemiologic or clinical features, several syndromes were ascribed to different serotypes, such as Fort Bragg fever caused by *Leptospira autumnalis,* swineherd's disease caused by *Leptospira pomona,* and Weil's disease caused by *Leptospira icterohaemorrhagiae*. Such terms are no longer commonly used because of overlap in the symptoms and epidemiology associated with various *Leptospira* serotypes.

Epidemiology

Leptospirosis is found throughout tropical and temperate areas of the world. It is particularly common in Southeast Asia and parts of Latin America, including some Caribbean islands.[76] Approximately 40 to 100 cases are reported annually in the United States (Fig. 53-11). Undoubtedly, many cases are unreported. Active surveillance on Kauai and the east coast of the big island of Hawaii revealed a high incidence, accounting for a large proportion of flulike illness.[186] In 1996, five of 26 travelers returning from a white-water rafting trip in Costa Rica developed a febrile illness and were found to have leptospirosis.[42] In 1998, leptospirosis was implicated in an outbreak of acute febrile illness among athletes from 44 states and seven countries who participated in triathlons (which involve open-water swims in fresh water) in Springfield, Illinois, and Madison, Wisconsin. Eighty-four (11%) of Illinois participants and 20 (5%) of Wisconsin participants were infected, and the diagnosis was confirmed by the CDC.[43] Both these reports highlight the potential risk to those with occupational, avocational, or recreational (including travel) exposure to contaminated fresh water.

Leptospirosis is a zoonosis in which certain serovars tend to have host specificity (Table 53-1). Dogs are usually associated with *L. icterohaemorrhagiae* and *Leptospira canicola,* and swine and cattle are more frequently infected with *L. pomona* and *Leptospira grippotyphosa,* although all four of these serovars or serotypes have been isolated from each host species. The major reservoir for *Leptospira* infections for humans and domestic animals is wildlife, principally wild mammals, although the organism has also been isolated from frogs and snakes, and serologically positive fish and turtles have been found.[140]

Leptospirosis may be asymptomatic in animals, as is usually the case in wild animals, including rodents. Animals that acquire clinical disease have fever, appear depressed, lose appetite, may become jaundiced, develop hemorrhages on mucous membranes, and in late stages of the disease may have renal failure.[99] In cattle, leptospirosis can cause stillbirths, hemoglobinuria, and thickened yellowish or blood-tinged milk. Leptospires have been isolated from the milk of cattle and goats. A theoretic risk exists that leptospirosis could be transmitted by consuming such milk. Pasteurization should destroy organisms. Stillbirths or delivery of weak piglets is a common sign of leptospirosis in swine.

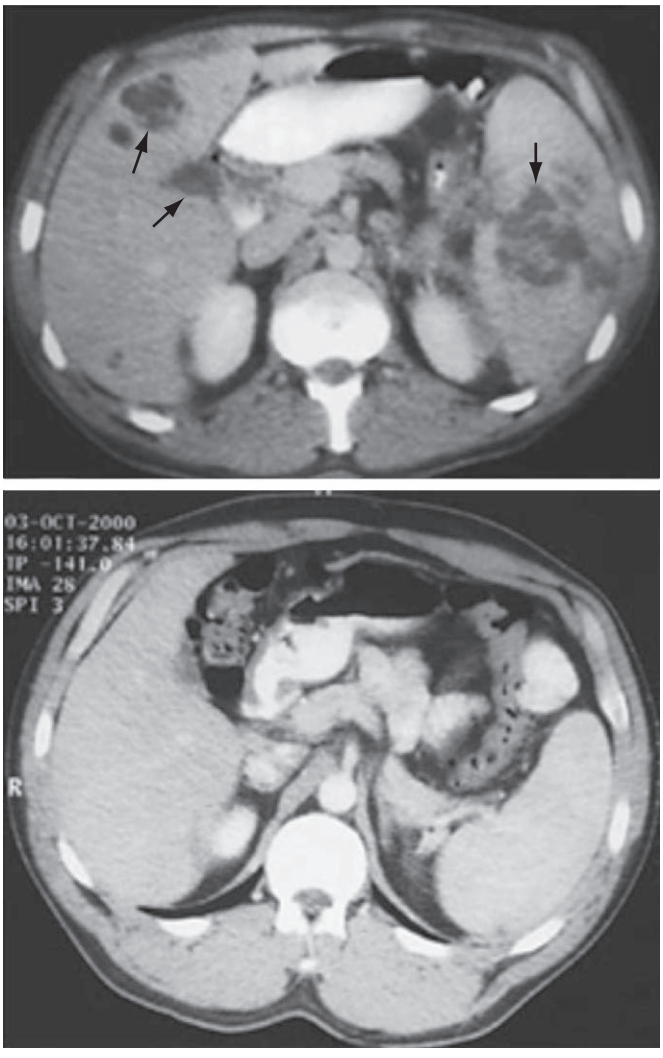

Figure 53-9. Abdominal computed tomographic scans from a patient with glanders. **A,** Before treatment, multiple hepatic and splenic abscesses *(arrows)*. **B,** After treatment, nearly complete resolution of the abscesses. (From Srinivasan A, Kraus CN, DeShazer D, et al: N Engl J Med 345:256–258, 2001.)

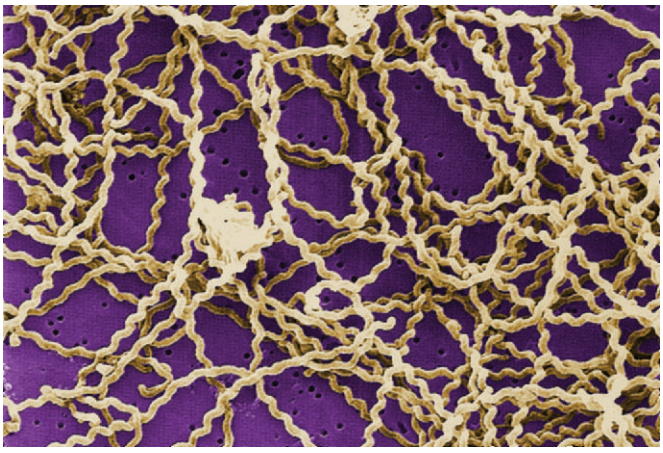

Figure 53-10. Scanning electron micrograph (SEM) of a number of *Leptospira* bacteria atop a 0.1-µm polycarbonate filter. Leptospires are long, thin, motile spirochetes that may be free-living or associated with animal hosts; they survive well in fresh water, soil, and mud in tropical areas. Organisms are antigenically complex, with over 200 known pathogenic serologic variants. Molecular taxonomic studies at the Centers for Disease Control and Prevention and elsewhere have identified 13 named and four unnamed species of pathogenic leptospires. Although some infected persons have no symptoms at all, leptospirosis can cause a wide range of symptoms, including high fever, severe headache, chills, muscle aches, and vomiting. Patients also may have jaundice (yellow skin and eyes), red eyes, abdominal pain, diarrhea, or a rash. (From Public Health Image Library, available at http://phil.cdc.gov/phil/home.asp, and courtesy Dr. Rob Weyant, 1998. Photo courtesy Janice Carr).

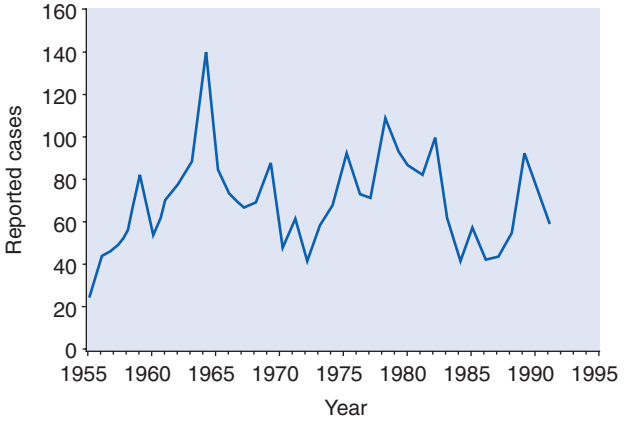

Figure 53-11. Reported annual cases of leptospirosis in the United States, 1955 through 1991. (From Centers for Disease Control and Prevention: MMWR Morb Mortal Wkly Rep 40, 1991.)

Cats are rarely affected by leptospirosis. They may be resistant to the disease because they are frequently exposed to infection through catching mice and other rodents.

Leptospirosis has been suspected as a cause of recurrent uveitis in horses. This is of more interest in comparative pathology than in public health, because horses infrequently transmit the infection to humans.

Transmission

Animals contaminate the environment by shedding organisms in their urine. Most human cases are environmentally acquired by contact with contaminated water or soil, and discovering the original animal source is difficult. A wet, alkaline environment favors survival, with tropical, unpolluted, nonsaline water with a slightly alkaline pH providing an optimum environment for infection. Heavy tropical rains increase infection risk by saturating soil, flushing leptospires into surface water, and drawing rodents and other small mammals into swampy areas. Infection can also be acquired by contact with infected animal blood and tissues. Factors strongly associated with acquiring leptospirosis in Hawaii include household use of rainwater catchment systems and the presence of skin cuts at the presumed time of exposure.[186]

Leptospirosis is an occupational problem for veterinary, agricultural, sewer, slaughterhouse, laboratory, and military

TABLE 53-1. Animal Reservoirs of Leptospires Isolated from Humans*

SEROGROUP (SEROVAR)	DOMESTIC ANIMALS	WILDLIFE
Icterohaemorrhagiae (Icterohaemorrhagiae)	Dogs, cattle, swine	Brown rat, house rat, cotton rat, Pacific rat, house mouse, muskrat, gray fox, red fox, opossum, striped skunk, woodchuck, nutria
Canicola (Canicola)	Dogs, cattle, swine	Striped skunk, raccoon, armadillo, mongoose
Pomona (Pomona)	Dogs, cattle, swine, goats, sheep, horses	Striped skunk, raccoon, wildcat, opossum, woodchuck, red fox, deer, armadillo
Grippotyphosa (Grippotyphosa)	Dogs, cattle, swine	Muskrat, fox squirrel, gray squirrel, bobcat, cottontail rabbit, swamp rabbit, raccoon, striped skunk, red fox, gray fox, vole, opossum
Hebdomidis (Hardjo)	Cattle	None

*Isolated in the United States.
Modified from Hanson LE: J Am Vet Med Assoc 181:1505, 1982.

personnel.[75,101,186] Dairy farmers are at risk in milking parlors, probably through exposure to cow's urine.[7,108] Leptospirosis poses an avocational risk for hunters, trappers, hikers, and persons who swim in fresh water, such as ponds and streams that may be contaminated with infected urine. It is acquired by ingestion or by entry through abraded skin or through the mucous membranes of the eye and mouth.

Symptoms

The incubation period for leptospirosis is usually 7 to 12 days (range, 1 to 26 days).[185] The disease characteristically is biphasic.[110,177] The primary stage lasts 4 to 7 days and is characterized by organisms in blood, CSF, and various body tissues. During the initial phase, more than half of victims have fever, chills, severe malaise, myalgias, headache, lymph node enlargement, and conjunctival injection, usually without exudate. Nausea, vomiting, and abdominal pain may occur. A nonproductive cough is common. The initial clinical differential diagnosis includes meningitis, hepatitis, influenza, nephritis, encephalitis, and viral illness. The rickettsioses, typhoid fever, brucellosis, relapsing fever, toxoplasmosis, dengue fever, malaria, yellow fever, septicemia, Kawasaki syndrome, and toxic shock syndrome are also differential diagnoses.[101]

The primary stage is usually followed by an afebrile period of 1 to 2 days. The onset of the second stage coincides with development of immunoglobulin M (IgM) antibodies. The organisms usually cannot be cultured from blood or CSF during this phase but can be isolated from urine for weeks or months. During the second stage, the victim may have fever, but the temperature is lower than in the primary stage. Headache is persistent, severe, and unresponsive to analgesics. It often heralds the onset of meningitis, one of the common complications of the secondary stage.

Myalgias, abdominal pain, nausea, and vomiting can occur in the second as well as in the primary stage. In addition to the conjunctival injection seen in the primary stage, uveitis (iridocyclitis) can be seen in the secondary stage. This can cause long-term ocular damage.[207] Occasionally, pharyngitis and a macular, purpuric, or ecchymotic rash occur (Fig. 53-12). Rarely, endocarditis or myocarditis occurs. In a clinical study of leptospirosis on Barbados, cardiac arrhythmias and myocarditis occurred in 18% and pericarditis in 6% of patients.[71]

Splenic enlargement develops in approximately 20% of patients in the second stage. Hepatomegaly is sometimes found,

Figure 53-12. Hemorrhagic macular rash in a patient with leptospirosis. (Courtesy University of Massachusetts Medical School.)

especially if the patient is icteric. Jaundice is a serious prognostic sign. Mortality in cases with jaundice exceeds 15% but is rare in anicteric cases. The overall case-fatality rate is approximately 5%. Mortality depends on the patient's prior condition and is higher in older individuals than in young adults. Death can occur from hemorrhagic manifestations as a result of vasculitis, renal or hepatic failure, cardiogenic shock, or myocarditis.

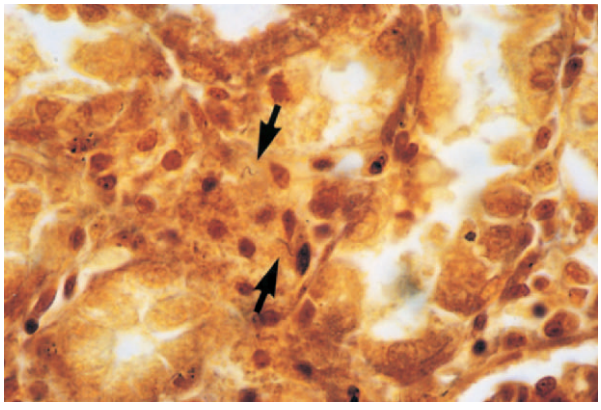

Figure 53-13. *Leptospira icterohaemorrhagiae* in the kidney of an experimentally infected dog. The organisms are indicated by *arrows*. (Warthin-Starry silver stain, ×1000.)

Diagnosis

Laboratory findings in leptospirosis include moderate leukocytosis, usually caused by an increase in neutrophils, elevated erythrocyte sedimentation rate, and thrombocytopenia. Elevated bilirubin level (up to 65 mg/dL, mainly direct bilirubin), greatly increased serum creatine kinase level (often 5 times normal), and a less-than-fivefold increase in aspartate transaminase suggest the diagnosis. An elevated blood urea nitrogen level is a common finding. Serum amylase concentration may also be elevated.

Definitive diagnosis of leptospirosis can be made by culture of the organism on Fletcher's, Stuart's, EMJH,[217] or Tween 80–albumin medium. Blood and CSF should be cultured during the first week of illness; urine should be cultured thereafter. The likelihood of obtaining a positive culture is greatly diminished once antibiotics have been given. Oxalated blood samples can be sent to the laboratory for culture because the organisms can remain viable in oxalated blood for up to 11 days.

Some physicians have relied on darkfield examination for identification of the organisms, but this method is not considered reliable. Artifacts such as fibrin are readily mistaken for leptospires. The spirochetes can be demonstrated in tissue sections with silver stains (Fig. 53-13).

Leptospirosis can be diagnosed serologically by demonstrating a fourfold rise in antibody titer.[234] The commercially available macroscopic slide agglutination test, using killed organisms of multiple serovars, or the CF test is useful for screening purposes. The macroscopic agglutination test may become positive within the first week of illness and can persist for several months. CF antibodies are detectable between days 10 and 21. Acute and convalescent sera should be tested 2 weeks apart because false-positive reactions can occur in single samples.

Specific serologic diagnosis of the serovar of infection can be made with the microscopic agglutination test (MAT), which uses live organisms but is available in relatively few reference laboratories. A genus-specific MAT employs a single, broadly reactive antigen.[237] An IgM-specific, dot-ELISA is comparable to the MAT in its ability to detect recent exposure to leptospires and is rapid and simpler.[166] Latex agglutination and indirect hemagglutination have high specificity and sensitivity and are especially useful early in the infection.[168] Although still investigational, a urine dipstick test shows promise as a rapid screening test for the diagnosis of leptospirosis.[202]

Treatment

Treatment of leptospirosis with antibiotics is most effective when begun during the first week of illness, preferably within 4 days of symptom onset. Although antibiotics were thought to have little value after this time, more recent studies indicate that they may still have some usefulness.[238] The treatment of choice is doxycycline, 100 mg orally twice a day for 7 days.[150] Alternative antibiotics include tetracycline, 2 g/day orally in three or four divided doses, or penicillin G, 3 million units/day parenterally. Amoxicillin, ampicillin, a cephalosporin, or erythromycin could also be used. Doxycycline and tetracycline should not be given to pregnant women or to children younger than 9 years old.

A Jarisch-Herxheimer reaction may occur after treatment. This is a response to release of endotoxins, usually occurring within 2 to 6 hours after initiating therapy, with sudden onset of fever, chills, malaise, headache, and tachycardia. The reaction typically resolves spontaneously within 24 hours.

Other than antibiotic treatment, therapy for leptospirosis is supportive, including fluid therapy, dialysis for renal failure,[113] and transfusion for hemorrhagic complications. Recovery from leptospirosis apparently leaves serovar-specific immunity. Individuals can become infected with other serovars. Assuming that the infection and hemorrhagic complications can be controlled, the long-term prognosis after successful treatment is good. Renal and hepatic functions usually return, but headache and ocular damage may persist.[207]

Prevention

Prevention of human leptospirosis is based on avoiding infected animal tissues and areas contaminated by animal urine, blood, or tissue. Individuals who are at particularly high risk should be educated about prevention and encouraged to wear protective clothing, such as rubber gloves, when handling infective material. Swimming in freshwater ponds and streams likely to be heavily contaminated by urine from livestock or wildlife should be discouraged.

Doxycycline, 200 mg once a week, effectively prevented infection in U.S. soldiers training in Panama.[220] Such prophylactic treatment could be given to individuals at unusually high risk.

Although *Leptospira* vaccines have been experimentally produced for human use, no product is approved and commercially available in the United States. Vaccines are available for animals. Immunization of domestic animals has primarily a veterinary benefit, in that the animals are protected from clinical disease. Immunity lasts about 6 months, but immunization does not guarantee that the animal cannot become infected. Several human cases have been traced to immunized dogs that apparently were still able to shed organisms.[77] Since then, some veterinary vaccines have been shown experimentally to reduce the renal carrier state.[115]

MELIOIDOSIS

Melioidosis is an infection caused by the bacterium *Burkholderia* (formerly *Pseudomonas*) *pseudomallei,* which lives freely in soil and water.[250] Disease is spread from the environment to both humans and other animals. The causative agent and

disease process in humans were first described by Whitmore and Krishnaswami[241] in Rangoon, Burma, in 1912.[129]

Bacteriology

B. pseudomallei is a bipolar, gram-negative, aerobic rod approximately 0.5 to 1 µm in width and 3 to 5 µm in length. It is readily grown on standard laboratory media at 37° C (98.6° F). After 48 to 72 hours of growth, distinctive wrinkled colonies with a "daisy head" appearance are formed. These give off a pungent, putrefactive odor.[128] The organism is oxidase positive and nonpyocyanogenic. It is resistant to colistin and gentamicin in vitro.[97] In 1992, Yabuuchi and coworkers proposed that seven species, formerly of *Pseudomonas* RNA group II, should be transferred to a new genus, *Burkholderia,* with *B. cepacia* as the type species.[250]

Transmission and Epidemiology

Melioidosis is a saprozoonosis transmitted to animals and humans from the environment; transmission does not generally occur between living organisms. There is a single case of presumed transmission by the venereal route.[151] Disease is reported much more often in adults than in children.

Most cases of melioidosis have been reported in areas between 20 degrees north and 20 degrees south of the equator.[96] A majority of cases have been reported from Southeast Asia and tropical Australia. The most heavily endemic areas include Myanmar (Burma), Malaya, Vietnam, Singapore, Cambodia, Thailand, Java, Borneo, New Guinea, and northern Australia. Occasional human and animal cases have been reported from Central India, Sri Lanka, Niger, Madagascar, Ecuador, Panama, Aruba, and Mexico. Cases from the Western Hemisphere have been reviewed.[10] *B. pseudomallei* survives during Australia's dry season in the clay layer of the soil, 25 to 30 cm below the surface, and can be brought to the surface and distributed by water seeping through this layer during the wet season.[97] Clinical and subclinical infections occur in a variety of animals, most often sheep, goats, and swine. Infections in animals offer no direct threat to human health but are epidemiologic indicators that the organism is in a given geographic area.[96] Transmission is thought to occur by direct percutaneous inoculation through wounds contaminated with soil or water, by ingestion, or by inhalation of infective droplets. A high incidence of pulmonary melioidosis in helicopter crew members in Vietnam was ascribed to inhalation of dust and aerosols raised by the helicopters operating in highly endemic areas.[96]

Symptoms

Most human infections are asymptomatic, as indicated by the high prevalence of seropositivity without clinical disease within endemic areas. Active disease is most likely to be seen in individuals with predisposing conditions, particularly diabetes mellitus, alcoholism, neoplasms, malnutrition, and various forms of immunodeficiency.

Clinical disease can occur in acute, subacute, or chronic forms. The lungs are the organs most frequently involved. Pulmonary melioidosis can mimic tuberculosis in that the upper lobes are most frequently involved, cough is productive, sputum often contains blood, and patients complain of chest pain and fever (Fig. 53-14). However, calcifications in the lung and hilar lymph nodes typically seen in tuberculosis are rarely seen with melioidosis. An entire lobe or major segment of a lobe may be consolidated, and multiple pulmonary abscesses may be scattered in the lung parenchyma.

Septicemia can develop in the acute form of the disease and may mimic other forms of gram-negative sepsis in its manifestations. Multiple abscesses can form in skin, lungs, liver, spleen, kidney, and bone, but the CNS is rarely involved. Without treatment, the case-fatality rate exceeds 90%.

In the subacute and chronic forms of disease, abscesses can form in internal organs and may drain through sinus tracts.

Melioidosis has been referred to as a "medical time bomb" because infection can lie dormant for months or years, only to become manifest when resistance is lowered. An incubation period as long as 26 years has been reported.[147] The infection should be suspected in anyone with a compatible clinical picture, including fever of unknown origin, who has resided in an endemic area.

Diagnosis

The only definitive diagnostic procedure is culture of the organism from blood, bone marrow, sputum, pus, or infected tissue. Several serologic tests are available but are not reliable, because many individuals from endemic areas have antibodies to the organism without any evidence of clinical disease. An indirect hemagglutination antibody titer of 1:40 or greater is considered compatible with infection, as is a CF titer of 1:10 or greater. Rising titers by IgM indirect FA are probably the best immunologic indication of infection.

No pathognomonic lesions are seen histologically. The abscesses consist of central areas of necrosis without unique or distinctive features that would permit definitive histopathologic diagnosis.

Treatment

Combinations of antibiotics have been used, almost always including chloramphenicol.[2] For adults, recommended therapy includes chloramphenicol, 40 mg/kg/day divided four times daily for 8 weeks; doxycycline, 4 mg/kg/day divided twice daily for 20 weeks; and trimethoprim-sulfamethoxazole, 10 mg/kg/day of trimethoprim divided twice daily for 20 weeks. For children younger than 9 years or pregnant women, recommended treatment is amoxicillin-clavulanate extended-release twice daily for 20 weeks. Even with compliance, relapse rate is up to 10%.

Ceftazidime has largely replaced combination antibiotic therapy and is now generally recognized as the drug of choice for initiation of treatment.[241] It has reduced mortality in disseminated septicemic melioidosis to 35% to 40%.[128] After parenteral treatment for at least 10 days with clinical improvement, a combination antibiotic oral regimen is recommended for a complete course of treatment.

In vitro sensitivity tests do not always correlate with clinical response to antibiotics. Therapy must be given at sufficient dosage and long enough to avoid recurrence of infection and emergence of resistant organisms.

Prevention and Control

In the field, prevention consists of avoiding ingestion or inhalation of potentially infective soil or water. Wounds, burns, and other injuries should be thoroughly cleaned to avoid infection through contamination. No evidence exists that transmission is likely to occur from person to person, so isolation of hospitalized patients is not necessary. Reasonable care and precautions

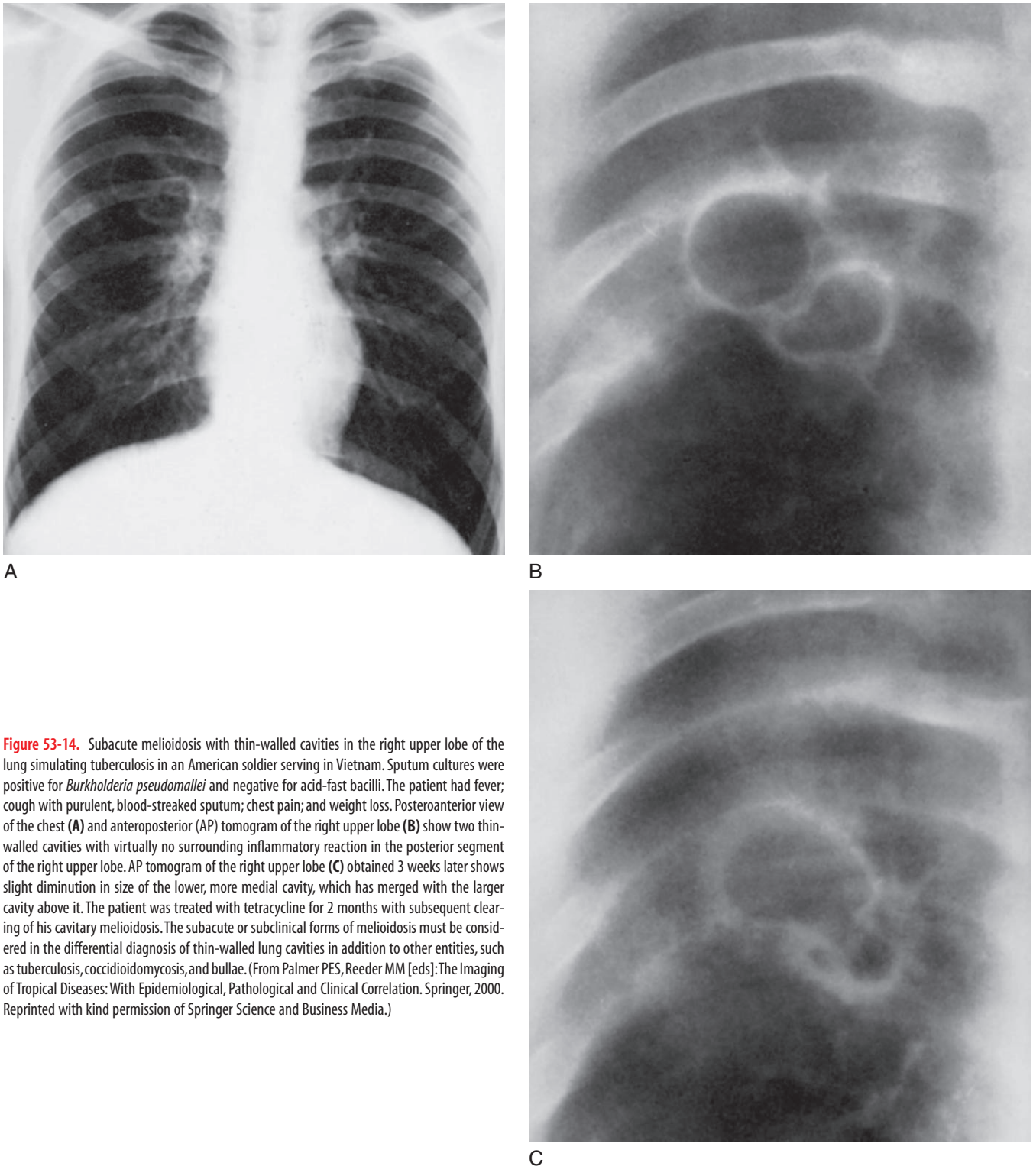

Figure 53-14. Subacute melioidosis with thin-walled cavities in the right upper lobe of the lung simulating tuberculosis in an American soldier serving in Vietnam. Sputum cultures were positive for *Burkholderia pseudomallei* and negative for acid-fast bacilli. The patient had fever; cough with purulent, blood-streaked sputum; chest pain; and weight loss. Posteroanterior view of the chest **(A)** and anteroposterior (AP) tomogram of the right upper lobe **(B)** show two thin-walled cavities with virtually no surrounding inflammatory reaction in the posterior segment of the right upper lobe. AP tomogram of the right upper lobe **(C)** obtained 3 weeks later shows slight diminution in size of the lower, more medial cavity, which has merged with the larger cavity above it. The patient was treated with tetracycline for 2 months with subsequent clearing of his cavitary melioidosis. The subacute or subclinical forms of melioidosis must be considered in the differential diagnosis of thin-walled lung cavities in addition to other entities, such as tuberculosis, coccidioidomycosis, and bullae. (From Palmer PES, Reeder MM [eds]: The Imaging of Tropical Diseases: With Epidemiological, Pathological and Clinical Correlation. Springer, 2000. Reprinted with kind permission of Springer Science and Business Media.)

should be taken in handling purulent drainage of blood, sputum, and other materials from patients with melioidosis.

RAT-BITE FEVER

Epidemiology

Rat-bite fever is an acute illness caused by either *Streptobacillus moniliformis* or *Spirillum minus,* which are part of the normal oral flora of rodents, including squirrels. It may also result from bites by wild and domestic carnivores, such as weasels, dogs, cats, and pigs, which may have been infected when hunting rats and mice.[205] Carrier rates among wild rats vary from none to 50%.[20,218] Fewer than 70 cases have been reported in North America, with at least half caused by *S. moniliformis* in laboratory workers. At least 10%, and up to 100%, of rats are nasopharyngeal carriers of *Streptobacillus.*[6,40,93] Victims of disease caused by *S. minus* are primarily children of lower socioeconomic groups with poor sanitation and heavy rodent populations.[152] Rare cases can occur in any setting and can easily be fatal, particularly when the proper diagnosis is not suspected.[152]

Streptobacillary Type

Streptobacillary rat-bite fever (Haverhill fever) is caused by *S. moniliformis,* an aerobic, nonmotile, gram-negative bacillus. The onset usually occurs within a week of the bite, but the incubation period may extend to several weeks, during which time the original wound usually heals completely. A bite need not be present, because the disease can also be transmitted by contaminated food, milk, or water.[79] It has also been transmitted by simply playing with pet rats, with no history of bite or injury.[182]

Symptoms

Initial symptoms include fever, chills, cough, malaise, headache, and, less frequently, local lymphadenitis. These are followed by a nonpruritic morbilliform or petechial rash, which frequently involves the palms and soles. Migratory polyarthritis develops in approximately 50% of victims. Generalized lymphadenitis may be present; splenomegaly and hepatomegaly are rare.[218]

Diagnosis

The Venereal Disease Research Laboratories (VDRL) test shows a false-positive result for 25% of victims. Leukocytosis with left shift is common, and agglutinating antibodies for the bacillus appear during the course of the disease. On autopsy, interstitial pneumonia, fibrinous endocarditis, mononuclear meningitis, hepatosplenomegaly, and mononuclear cell infiltrates in regional lymph nodes have been found.[204]

When a history of animal bite is lacking, the differential diagnosis must include rickettsial and viral infections. The fever and rash may suggest meningococcemia, but meningeal signs are lacking in rat-bite fever. Definitive diagnosis requires demonstration of rising antibody titers or culture of the bacillus from the blood, joint fluid, pustules, or the original bite location. This can make diagnosis difficult. Typically, identification by culture requires experienced laboratory workers who are seeking the organism.

Treatment

Untreated, the disease runs a course of several weeks; prolongation of symptoms should raise the suspicion for endocarditis. The mortality in untreated persons is 10%, with most deaths caused by endocarditis and pneumonia. Although mortality is low, the disease can be fulminant; an infant bitten by a wild rat died 4 days after the bite and 2 days after the onset of symptoms.[204] The drug of choice is procaine penicillin, 600,000 units intramuscularly twice a day for 7 to 10 days.[218] Effective alternatives for penicillin-allergic patients are tetracycline, 30 mg/kg/day orally in four divided doses, or streptomycin, 15 mg/kg/day intramuscularly in two divided doses. Erythromycin is not effective. Complications such as endocarditis should be treated with high-dose intravenous potassium penicillin G, 10 to 20 million units a day. The organism has both a bacillary and a cell wall–deficient L phase, which is thought to account for some of the antibiotic failures. The bacterial phase responds to penicillin, streptomycin, and tetracycline, whereas the L phase is resistant to penicillin.

Spirillar Type

Spirillar rat-bite fever is caused by *Spirillum minus,* a gram-negative, tightly coiled, spirillar microorganism. It is usually transmitted by infected wild rats, although cats have also been implicated. The general setting of socioeconomic deprivation in which this disease occurs is the same as in the streptobacillary form; cases in laboratory animals are unusual. The incubation period is 7 to 21 days, during which the bite lesion heals.

Symptoms

The onset of illness is heralded by chills, fever, lymphadenitis, and a dark-red macular rash. Myalgias are common, but arthritis is absent, which helps in the differentiation from streptobacillary fever. Leukocytosis and a false-positive VDRL test are often present. The disease is episodic and relapsing, with a 24- to 72-hour cycle. The differential diagnosis includes rickettsial and viral diseases when the history of animal bite is not present. In addition to the absence of arthritis, the manifestations at the bite site are more pronounced than in the streptobacillary variety (usually involving lymphadenitis, and still present when systemic illness occurs), and the fever and illness are relapsing in nature.[218]

Diagnosis and Treatment

Definitive diagnosis rests on demonstrating the presence of *S. minus* in a darkfield preparation of exudate from an infected site. The patient's blood can be inoculated into mice, which may be tested for subsequent infection. The mortality from untreated disease is considerably lower than that from streptobacillary fever. The untreated course spans several months. Antibiotic therapy is the same as for streptobacillary fever.[93,217]

PLAGUE

Plague, a bacterial disease caused by *Yersinia pestis* and transmitted by the bite of infected fleas, has occurred in explosive epidemics. Probable epidemics of plague occurred during the Peloponnesian War, as described by Thucydides, in approximately 400 BC. It ravaged the Roman Empire and western Europe during the age of Justinian and continued through the 7th century. The best-known and most devastating epidemic started in 1348. Known as the Black Death, the infection spread from Asia throughout western Europe, killing one third of the population (Fig. 53-15).[84] Plague is a reportable disease under the International Sanitary Regulations.

Figure 53-15. Burial of plague victims.

Plague is carried by various rodent reservoir hosts and transmitted by rodent fleas. Most of the outbreaks in Europe have been ascribed to importation of plague from enzootic foci in Asia, by ships bringing infected rats and people to port cities. A matter of considerable historical and epidemiologic interest is how and why the various epidemics eventually subsided. Theories include development of mutant bacteria; changes in patterns of shipping, building, and hygiene; replacement of *Rattus rattus* by *Rattus norvegicus* (in the case of the Black Death) (Fig. 53-16); and development of immunity in animals and humans.[72]

Bacteriology

Y. pestis is a gram-negative, nonmotile, nonsporulating rod with a bipolar, or "safety-pin," appearance in smears stained with Giemsa or Wayson stain (Fig. 53-17).[32] The appearance is somewhat variable by Gram technique.

Most standard bacteriologic media support the growth of the organism aerobically or under facultative anaerobic conditions. Optimal in vitro growth occurs at 28° C (82.4° F), at which temperature colonies become visible on plain agar in approximately 48 hours.

Virulence factors encoded by the bacterial chromosome include surface capsular material that is antiphagocytic, the ability to synthesize purines even if phagocytosed, and a surface component needed for iron uptake. Virulence factors mediated by a plasmid include dependence on environmental calcium for growth at 37° C (98.6° F) and production of v[73] and w[136] antigens. The relationship between calcium dependence and virulence is under investigation.[84]

Epidemiology

The epidemiology of plague is complex. Various fleas can transmit the infection between reservoir rodent hosts and humans. In major epidemics the prominent carriers were the tropical rat flea *(Xenopsylla cheopis)* (Fig. 53-18) and the black rat *(R. rattus)*.

Smoldering foci of infection are maintained in nature in wild rodents and their fleas. In the United States, enzootic (maintenance) hosts include deer mice *(Peromyscus maniculatus)* and various voles *(Microtus species)*. Epizootic (amplifying) hosts include the prairie dog (*Cynomys* species) (Fig. 53-19) and ground squirrel (*Spermophilus* species). Other rodents and lagomorphs that maintain infection include chipmunks (*Eutamias*

Figure 53-16. A Norway rat *(Rattus norvegicus)* in a corn storage bin in Kansas City, Missouri. *R. norvegicus* is known to be a reservoir of bubonic plague (transmitted to man by the bite of a flea or other insect), endemic typhus fever, ratbite fever, and a few other dreaded diseases. (From CDC Public Health Image Library, available at http://phil.cdc.gov/phil/home.asp.)

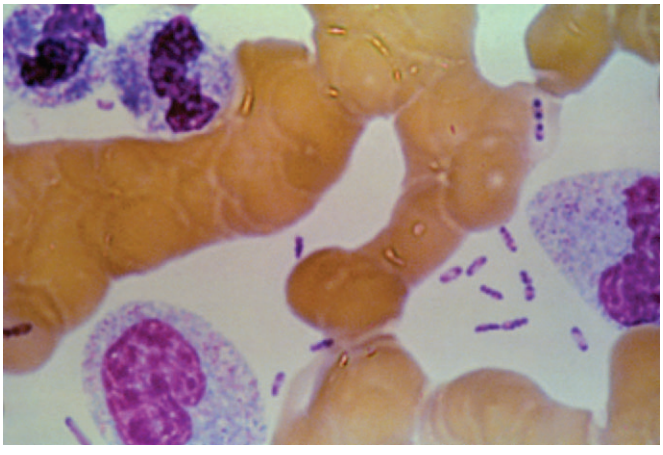

Figure 53-17. Wayson stain of *Yersinia pestis*. Note the characteristic "safety pin" appearance of the bacteria. (Available at www.cdc.gov/ncidod/dvbid/plague/wayson.htm.)

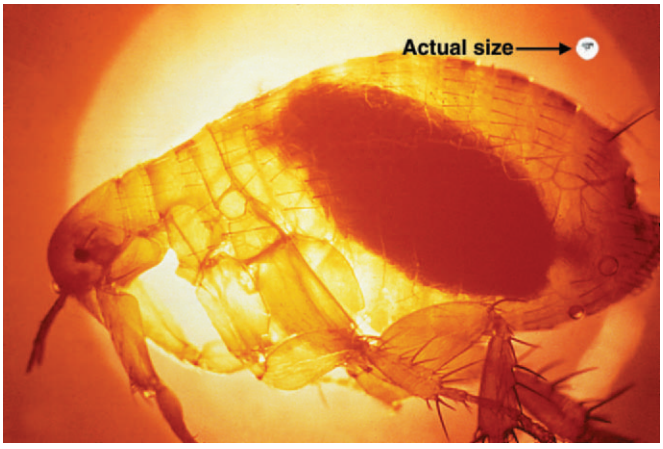

Figure 53-18. Male *Xenopsylla cheopis* (Oriental rat flea) engorged with blood. This flea is the primary vector of plague in most large plague epidemics in Asia, Africa, and South America. Both male and female fleas can transmit the infection. (Available at www.cdc.gov/ncidod/dvbid/plague/index.htm.)

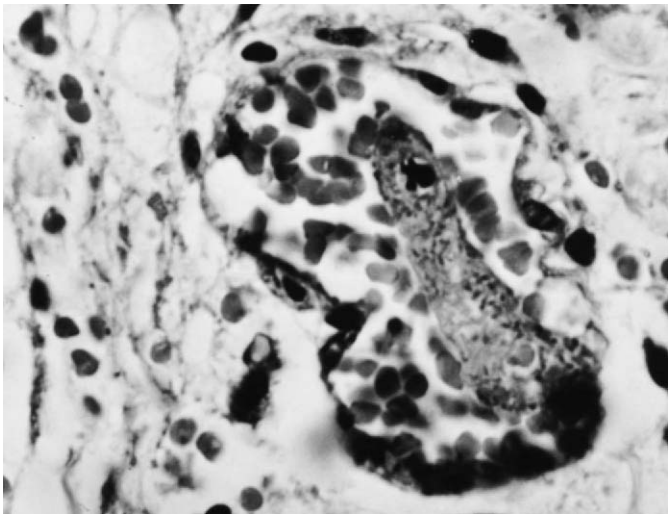

Figure 53-19. Thrombus in a vein in the subcutis of a prairie dog naturally infected with plague. Many organisms *(fine stippling)* are present in the thrombus. (Hematoxylin and eosin, ×1000.)

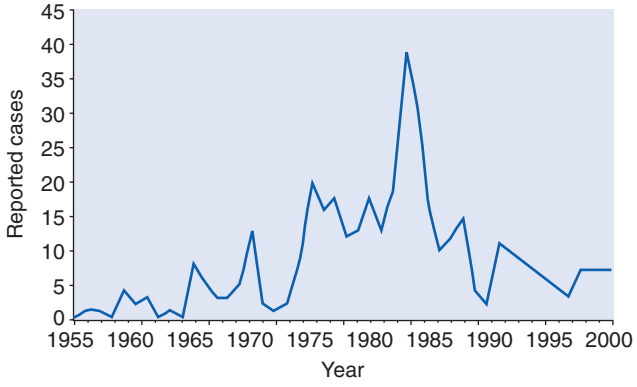

Figure 53-20. Reported cases of plague in humans in the United States, 1955 to 1999. (Modified from Centers for Disease Control and Prevention: MMWR Morb Mortal Wkly Rep 40, 1991.)

species), marmots (*Marmota* species), wood rats (*Neotoma* species), rabbits (*Sylvilagus* species), and hares (*Lepus* species).[170]

Enzootic foci of plague remain in parts of Asia, Africa, and South America,[29,170] as well as in the western United States. The major enzootic states in the United States are New Mexico, Arizona, California, Colorado, Oregon, Nevada, and Utah. From 1944 through 1993, 362 cases of human plague were reported in the United States; approximately 90% of these occurred in Arizona, California, Colorado, and New Mexico (Fig. 53-20). Seven confirmed cases of human plague were reported in 1995 (three in New Mexico, two in California, one in Arizona, and one in Oregon). Five of the seven cases were bubonic, one was septicemic, and one a fatal pneumonic case.[136] In many foreign areas, the exact species of rodents and fleas involved in transmission and maintenance are not known. Vietnam is the only country considered a threat for the international introduction of plague (Fig. 53-21).

Carnivorous mammals can acquire plague by ingesting infected rodents or by being bitten by their fleas. Dogs usually do not become very ill with plague, but cats can acquire severe, often fatal, forms of infection, resembling syndromes seen in humans. Bubonic plague in cats is usually manifested as severe submandibular lymphadenopathy. Cats can also acquire pneumonic plague, characterized by a subacute febrile course with cough and respiratory distress. Cats can transmit plague to humans by bite or by respiratory droplets, or by carrying fleas to people. Plague is an occupational risk for veterinarians who handle sick cats or their tissues.[112,179]

Although dogs are usually not as severely affected as cats, at least one human case was associated with a dog that apparently died of plague.[181] Dogs may be significantly involved in the epidemiology of plague in Tanzania, as reservoirs of vector fleas or bacterial infection.[116] In the United States, one person acquired plague while skinning an infected coyote[34] and two while skinning bobcats.[37] Other carnivores, such as skunks, badgers, and raccoons, have been found with antibody to the plague bacillus and presumably were exposed while hunting infected rodents. Exposure to infected rabbits, their fleas, or both has been associated with human plague.[32] Plague is not generally recognized as a disease of farm animals in modern times, but a recent report ascribed one outbreak of plague in a Libyan village to contact with a sick camel and another to contact with two goats.[48]

In the United States, the major epidemiologic factor associated with acquiring plague is living in a rural or suburban area where the enzootic disease occurs. People who hike, camp, or perform field studies in such areas are subject to this disease. Its diagnosis should be considered in anyone with a compatible history who has recently been in an enzootic area.

Transmission

Plague is normally a flea-borne disease of rodents, although other animals, including humans, can become infected. Humans are generally infected by the bite of an infected flea; however,

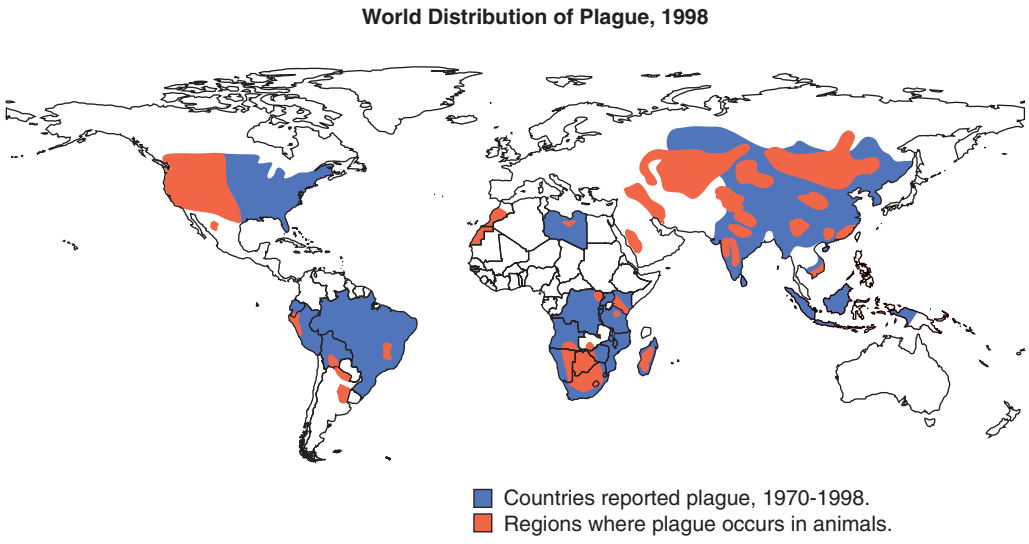

Figure 53-21. World distribution of plague, 1998. (Available at http://www.cdc.gov/ncidod/dvbid/plague/index.htm.)

direct contact with and percutaneous inoculation of *Y. pestis* organisms can lead to disease. Rarely, inhaled infective droplets cause pulmonary plague; human-to-human transmission can occur and is a threat in developing countries and in the United States.

The black rat is highly susceptible to plague and dies with severe septicemia. The concentration of organisms in the rat's blood ensures infection of the biting flea. When the rat dies, the flea leaves to seek other hosts. This cycle is unusual in that the infection kills the reservoir host and the vector, but the nature of the infection in the flea and rat guarantees further transmission and survival of the microorganism. The bacteria multiply so extensively in the *X. cheopis* flea that they block its proventriculus, or foregut. The flea cannot feed effectively, becomes ravenously hungry, and regurgitates large numbers of bacilli when it bites.

Symptoms

The three most common forms of plague are bubonic, pneumonic, and septicemic. Less common forms are meningeal, pharyngeal, and ophthalmic.

In the most common form, bubonic plague, buboes develop. These are greatly enlarged and very tender lymph nodes proximal to the point of percutaneous entry, such as a flea bite or a cut infected by handling infected tissues. Inguinal nodes are most often involved because fleas usually bite on the legs. Skinning an infected animal or handling its tissues often results in axillary buboes. Frequently, cervical, hilar, or mesenteric lymph nodes are enlarged.

The incubation period for bubonic plague is usually 2 to 6 days. In mild or early stages of infection, seeding of the blood occurs intermittently, causing a low sensitivity to blood culture testing. Later, if disease becomes severe, all blood cultures are positive. Victims usually have high fever, chills, severe malaise, headache, and myalgias. Toxicity, cardiovascular collapse with shock, and hemorrhagic phenomena may occur. Blackened hemorrhagic skin lesions gave rise to its being called the Black Death during the pandemic of the 14th century.

Victims with bacteremia and significant symptoms but no buboes are considered to have septicemic plague. Such cases may be difficult to diagnose unless the physician suspects plague on the basis of epidemiology. As with other forms of gram-negative sepsis, victims have fever, chills, malaise, headache, and GI symptoms such as nausea, vomiting, and diarrhea. The disease can result in cardiovascular collapse. Thrombosis (see Figure 53-19) and disseminated intravascular coagulopathy may be present. Untreated septicemic plague progresses to pulmonary involvement and death. However, transmissible pneumonic infection is estimated to occur in only 5% of patients with septicemic plague, because victims die before alveolar pneumonitis and the potential for droplet spread.

Pneumonic plague can result from inhalation of droplets from another pneumonic patient or by pulmonary seeding from the blood. Pneumonic plague runs an acute and fulminant course and is almost uniformly fatal if not treated. The incubation period is 2 to 3 days and disease is characterized by the sudden onset of fever, cough, bloody sputum, headache, and shaking chills. Radiographs reveal progressive consolidation of pneumonic patches in the lung, often with pleural effusion (Fig. 53-22).

Of 71 cases of plague reported in New Mexico between 1980 and 1984, 18 were septicemic.[106] The victims were significantly older and more likely to die than those with bubonic plague. The white blood cell counts in the New Mexican septicemic cases were low, normal, or elevated (range, 3000 to 68,700/mm^3), but all patients had relative or absolute neutrophilia with a left shift. Bacteria were demonstrable on direct blood smears in 3 of 17 septicemic patients. Plague is most common in persons less than 40 years old, perhaps because of their greater outdoor activity, but the risk of the septicemic form increases in individuals older than 40 years. Among patients with septicemic plague, mortality was greater in younger patients, perhaps because of greater delay in diagnosis and decreased likelihood that they would receive antibiotic therapy on an empiric basis.[106]

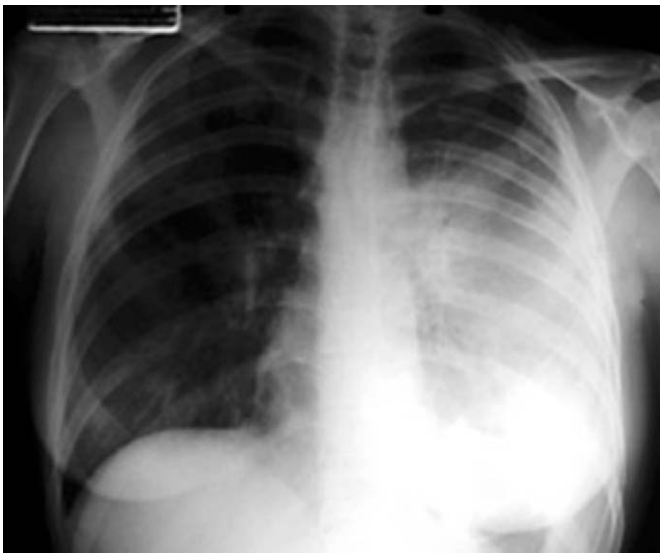

Figure 53-22. Pneumonic plague. (Available at http://er1.org/docs/photos/Plague/pneumonic%20plague.jpg.)

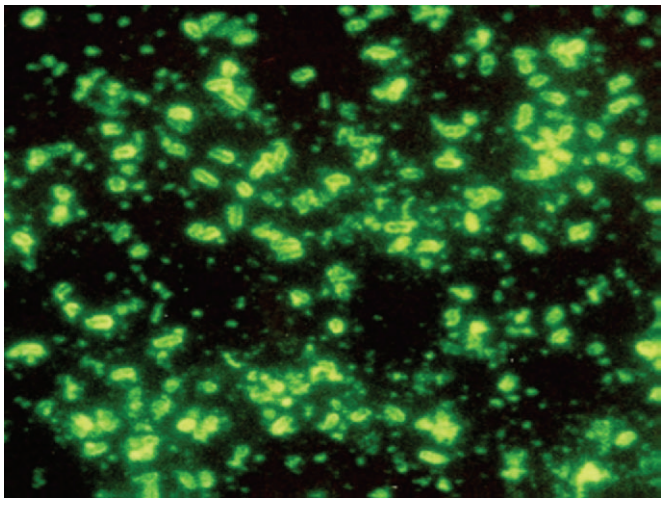

Figure 53-23. *Yersinia pestis* seen on direct fluorescent antibody testing. (Photo courtesy of the Centers for Disease Control and Prevention.)

Abdominal pain was present in nearly 40% of the patients in New Mexico. Hepatosplenomegaly occurs in plague and may be a source of abdominal pain, but it was not found in the patients reported in this series. The presence of abdominal symptoms in plague should be emphasized because the disease could be mistaken for other forms of GI illness. A review of the 71 plague cases in New Mexico from 1980 to 1984 revealed that GI symptoms occurred in 57% of patients, with vomiting (39%) the most frequent symptom. Nausea (34%), diarrhea (28%), and abdominal pain (17%) were also observed.[105]

Plague should be suspected in patients who have various combinations of lymphadenopathy, high fever, malaise, tachycardia, tachypnea, hypotension, and abdominal symptoms and who have come from an endemic area. If the suspicion is reasonable, antibiotic therapy should be instituted immediately. Treatment should not be delayed to await laboratory confirmation of the diagnosis.

Rare forms of plague include pharyngeal, meningeal, and ophthalmic. Pharyngeal and ophthalmic plague can be acquired by exposure to droplets expelled by a pneumonic patient. Endophthalmitis can also be secondary to septicemia. Meningitis is acquired by direct seeding while the patient is bacteremic.

Diagnosis

Definitive diagnosis is based on culture of the organism from sputum, transtracheal aspirate, blood, or aspirates of buboes. The best staining technique to demonstrate bipolar morphology of the organism is the Giemsa or Wayson technique.

Direct FA stain of aspirates and smears provides a reasonably rapid diagnostic technique (Fig. 53-23). Although cross-reactions with *Yersinia pseudotuberculosis* have been recorded, and occasional strains of the plague bacillus do not stain well with FA, a positive FA test in a patient with a compatible epidemiologic and clinical picture is a reasonable basis for making a diagnosis of plague and instituting therapy.

Material from buboes should be obtained by fine-needle aspiration rather than excision or incision and drainage. This reduces risks of transmission to medical personnel and of iatrogenic septicemia.

Treatment

Patients with suspected plague should be treated immediately without awaiting definitive laboratory studies. The best available drug is streptomycin. If given too rapidly, streptomycin can cause a severe reaction as a result of the rapid killing of organisms and release of endotoxin. The recommended dosage is 30 mg/kg/day intramuscularly in four divided doses every 6 hours for 5 days. A less preferred alternative to streptomycin is gentamicin, 5 mg/kg/day intravenously in four divided doses every 6 hours, reduced to 3 mg/kg/day after clinical improvement. Patients with impaired renal function should have their dosage of streptomycin or gentamicin modified appropriately.[170]

Tetracycline is often used concurrently with streptomycin. The recommended loading dose is 15 mg/kg orally, up to 1 g total dose. This is followed by 40 to 50 mg/kg in six divided doses every 4 hours on the first day. Thereafter, 30 mg/kg is administered orally in four divided doses every 6 hours for 10 to 14 days. If the patient cannot tolerate treatment by mouth, tetracycline can be given intravenously, using one third of the calculated oral dose until oral therapy is tolerated.

An alternative to tetracycline is chloramphenicol, administered in a loading dose of 25 mg/kg orally, up to 3 g total, followed by 50 to 75 mg/kg orally in four divided doses for 10 to 14 days. If the patient does not tolerate oral therapy, chloramphenicol can be given intravenously, 50 mg/kg in four divided doses every 6 hours until oral therapy is tolerated. It is preferable to use chloramphenicol in the event of meningitis or endophthalmitis because of good penetration into affected tissues. Whichever drug combination is selected, antibiotic therapy should be given for at least 10 days, or for 3 or 4 days after clinical recovery.

Sulfadiazine is a less satisfactory alternative. A loading dose of 25 mg/kg is given orally, followed by 75 mg/kg orally in four divided doses every 6 hours for 10 to 14 days. Co-trimoxazole (320 mg of trimethoprim and 1600 mg of sulfamethoxazole) twice or three times a day for 10 to 17 days was found effective in treating plague,[2] but much less experience has been accumulated with this medication than with other antibiotics.

Infants born to plague-infected mothers may have congenital infection. They should be treated with kanamycin, 15 mg/kg/day intravenously or intramuscularly in four divided doses every 6 hours, or streptomycin, 10 to 20 mg/kg/day in four divided doses every 6 hours.

The recent discovery of a multidrug-resistant strain of *Y. pestis* in Madagascar has led to clinical concern.[83] Resistance to all first-line antibiotics, to the principal treatment alternatives, and to prophylactic drugs was mediated by a transferable plasmid. However, these isolates were susceptible to cephalosporins, quinolones, and trimethoprim.

The greatest risk of contagion is by aerosol transmission from patients with the pneumonic form of plague. All persons with suspected plague should be placed in strict quarantine and isolation for a minimum of 48 hours of specific antibiotic treatment. If no respiratory signs develop within 48 hours, wound and skin precautions will suffice for the rest of the hospitalization. Patients with plague pneumonia or pharyngitis should be kept under strict respiratory quarantine for at least 4 days of antibiotic treatment, until the pharyngeal culture is negative for the organism or respiratory signs abate. Contact personnel should wear gloves, gowns, masks, and eye protection. Strong[216] dramatically illustrated effective protective clothing during a pneumonic plague epidemic in Manchuria in 1911.

The greatest risk of acquiring plague from a patient with the septicemic or bubonic form is by inoculation of blood or exudate; therefore, strict needle precautions should be taken. Buboes should not be incised and drained. In theory, fleas harbored by a septicemic patient should be capable of transmitting the disease, but the CDC recommendations for control of plague do not cover eradication of fleas from human patients.[240] No pesticides have been approved by the U.S. Food and Drug Administration (FDA) for this use. Products effective against lice may have limited effectiveness because most fleas do not remain long on a human host.

Household contacts and individuals exposed to patients with pneumonic plague should be prophylactically treated with antibiotics. Tetracycline, 500 mg orally every 6 hours for 6 days, can be given to adults. Trimethoprim-sulfamethoxazole can be given to children less than 8 years of age and to pregnant women. Streptomycin, chloramphenicol, and sulfadiazine are alternative prophylactic medications, given in therapeutic doses for 1 week to 10 days.

Household contacts of individuals with bubonic plague do not have to be treated prophylactically. They should have their temperature recorded twice daily, and if it exceeds 37.7° C (100° F) orally, they should report immediately to a physician for evaluation. Careful surveillance is indicated for persons who have had face-to-face contact with patients with pneumonic plague. Their well-being should be confirmed daily.

The incubation period for primary pneumonic plague is 1 to 3 days and for pharyngeal plague 3 to 6 days. Precautionary follow-up observation of contacts should be maintained throughout this time. All cases of suspected or confirmed plague should be reported to the state health department.

Prevention

Residents and visitors to plague-endemic areas should be advised of the risks of infection. They should avoid contact with rodents and other possible animal reservoirs of infection that are found sick or dead in the wild. Disposable plastic or rubber gloves should be worn when skinning or dressing a possibly infected animal. Cats and dogs should be kept indoors, leashed, or otherwise restrained. Owners of pets that have access to wild rodent populations must maintain flea control. Veterinary personnel working on animals that could have plague should follow strict infection control procedures.

Health departments in enzootic areas should maintain surveillance for plague in local reservoir species. At times of increased plague activity, insecticide sprays and powders can be applied to rodent burrows. Ectoparasite control is essential before any attempt is made to kill the rodents, because killing the rodents without control of their ectoparasites causes fleas to seek other hosts, including dogs, cats, and people.

A killed bacterial vaccine is available in the United States but is rarely used. Its application is limited to persons who have intensive, usually occupational, exposure to the infection. Many of these persons prefer to rely on sanitation, protective clothing, and prophylactic or therapeutic treatment rather than use the vaccine, because it has short-term (6- to 12-month) efficacy and has a significant incidence of side effects, such as fever, malaise, and pain at the site of injection.[38]

Clinical and epidemiologic assistance for problems relating to plague can be obtained from the Plague Branch, Centers for Disease Control and Prevention (PO Box 2087, Fort Collins, CO 80522; 303-221-6400). After-hours consultation can be obtained from the CDC in Atlanta (404-639-8107).

TULAREMIA

Tularemia was first described in 1837 by Homma Soken, a Japanese physician who wrote of a febrile illness with generalized lymphadenopathy in persons who had eaten infected rabbit meat. In 1911, McCoy described a disease resembling plague in California ground squirrels. In the following year, McCoy and Chapin isolated the organism from rodents in Tulare County, California; this geographic site gave rise to the name of the disease. Edward Francis did much of the landmark bacteriologic and clinical investigation, and the genus of the causative organism, *Francisella,* is named after him. The role of ticks as vectors of the disease was discovered by Parker and Spencer in 1924. In 1929, they described transovarial transmission of the bacterium in ticks.

Bacteriology

Francisella tularensis is a nonmotile, gram-negative coccobacillus measuring 0.2 by 0.3 to 0.7 µm. It must be grown aerobically on a medium containing cysteine or other sulfhydryl compounds. The organism is best grown on glucose cysteine agar with thiamine, or on cysteine glucose blood agar. The organism has also been isolated in thioglycollate broth, charcoal yeast extract, and Thayer-Martin agar.

Two varieties of the organism are recognized in North America. Type A can ferment glycerol and has citrulline ureidase activity. It generally causes more severe disease than type B, which is found in Europe and Asia as well as in North America. Type B does not ferment glycerol and does not have

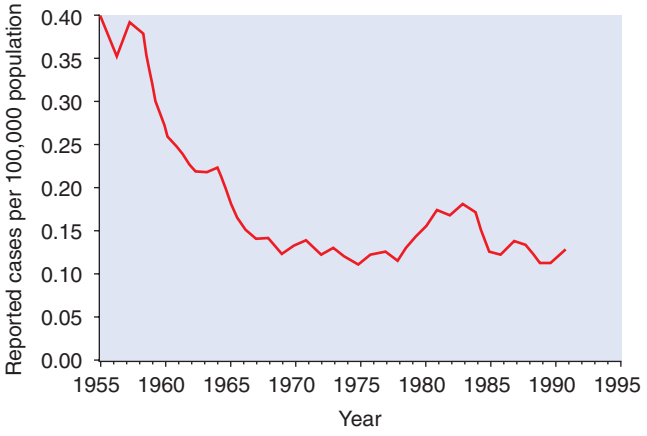

Figure 53-24. Reported cases of tularemia in the United States, 1955 through 1991. (From Centers for Disease Control and Prevention: MMWR Morb Mortal Wkly Rep 40, 1991.)

Figure 53-25. Cutaneous tularemia. Thumb with skin ulcer of tularemia. (From CDC Public Health Image Library, available at http://phil.cdc.gov/Phil/home.asp, and courtesy Dr. Thomas Sellers, Emory University.)

citrulline ureidase activity. Type A is more frequently recovered from rabbits and various bloodsucking arthropods. Type B is often recovered from water voles, muskrats, and beavers. However, the two varieties sometimes share an ecologic niche.[145]

Epidemiology

The reported incidence of tularemia has been steadily decreasing in the United States since its peak at 2291 cases in 1939 (Fig. 53-24). Other tick-borne infections, such as Rocky Mountain spotted fever and Lyme disease, have increased. Empiric use of antibiotics may have aborted undiagnosed cases.

Most cases in the United States have been reported from the South and Midwest, particularly Arkansas, Illinois, Missouri, Oklahoma, Texas, and Tennessee. The disease is also widespread in Europe, Canada, the Middle East, Russia, and Japan. Transmission by ticks and other arthropods usually occurs in the spring and summer. Transmission from rabbits most often occurs during the fall and winter hunting seasons.

Transmission

Before 1950, most reported cases of tularemia were associated with direct contact with rabbits. Tularemia is now most frequently transmitted by ticks.[23] Many different species of ticks are potential or proven vectors. A common vector in the United States is the dog tick, *Dermacentor variabilis*. The lone star tick, *Amblyomma americanum,* is the main vector in the southern United States (see Figure 45-3). Because the infection can be transmitted transovarially, ticks are an important natural reservoir. Ticks may transmit the bacteria through their feces, as the organism has not been isolated from their salivary glands. Deerflies and other biting flies may also be suitable vectors.[119] In the United States, the second most common source of human infection is rabbits. The infection can be acquired by skinning, eviscerating, or handling the tissues of infected rabbits or by eating improperly cooked infected meat. Transmission can also occur by direct contact with or ingestion of infected soil, water, or fomites. Infection can occur by inhalation of dust or water aerosol[61] or in the laboratory. Organisms remain viable in mud samples stored as long as 14 weeks, in tap water for 3 months, in dry straw for 6 months, and in salted meat for 31 days.[16]

Occasional cases of tularemia have been transmitted by cat bite[39,174] or by handling infected tissue from animals other than rabbits, such as bear,[36] deer,[35] beaver, and muskrat.[252] Person-to-person transmission is rare.

Symptoms

Classically, tularemia occurs in one of six clinical presentations: glandular, ulceroglandular, oculoglandular, oropharyngeal, pneumonic, or typhoidal.[16,145] Evans and colleagues[74] simplified this classification into two major categories: ulceroglandular and typhoidal. Patients are considered to have ulceroglandular tularemia if they have lesions of the skin or mucous membranes (Fig. 53-25), with or without associated lymphadenopathy, with affected lymph nodes at least 1 cm in diameter. Patients without lesions of the skin or mucous membranes and with lesser enlargement of lymph nodes are considered to have typhoidal tularemia. In this classification, pharyngitis or pneumonia can occur in either the ulceroglandular or the typhoidal form of the disease.

The ulceroglandular form accounts for approximately 80% of tularemia cases. The typical skin lesion begins as an erythematous papule or nodule that indurates and ulcerates. It is frequently painful and tender. Ulcers associated with handling infected animals are usually located on the hand, with associated lymphadenopathy found in the epitrochlear or axillary regions. Infections transmitted by tick bites are usually located on the lower extremities, associated with inguinal or femoral lymphadenopathy.

So-called glandular tularemia is characterized by the presence of enlarged, tender lymph nodes without an associated skin lesion. However, the skin lesion may have healed or gone unnoticed before the development of lymphadenopathy.

In the oculoglandular form, unilateral conjunctivitis occurs with concentration of inflammatory response in and around a nodular lesion on the conjunctiva and with enlargement of the ipsilateral preauricular lymph node.[98] The oculoglandular form constitutes 1% to 2% of tularemia cases.

The typhoidal form occurs in approximately 10% of tularemia cases. It is characterized by fever, chills, and debility.

As the disease progresses, weight loss may be significant. Hepatosplenomegaly can occur, especially in children.[107] Pericarditis occurs rarely.[74] Exudative pharyngitis can occur with either the typhoidal or the ulceroglandular form. There is usually associated cervical lymphadenitis.

Pneumonia is a fairly common complication of tularemia. The pulmonary infection can be acquired by inhalation of aerosol or from bacteremia. Symptoms include cough, chest pain, shortness of breath, production of sputum, and hemoptysis. Radiographic abnormalities of the chest may be found in patients without pulmonary symptoms. Chest films may reveal infiltrates, most often of the lower lobe, hilar lymphadenopathy, and pleural effusion. Tularemia patients with pneumonia are more likely to be older, less likely to have a known source of infection, and more likely to die than those without pulmonary infection.[200]

Severe complications of tularemia, such as bacteremia, pneumonia, and rhabdomyolysis, are most likely to be seen in patients with significant underlying disease, such as lymphoma, other forms of cancer, or diabetes.[167] From 1981 through 1987, the case-fatality rate of tularemia in the southwestern and central United States was 2%.[223]

Diagnosis

Ulceroglandular tularemia can be confused with cat-scratch disease, streptococcal and staphylococcal skin diseases, sporotrichosis, and plague. Typhoidal tularemia can mimic septicemic plague, brucellosis, salmonellosis, typhoid fever, other forms of gram-negative sepsis, and leptospirosis. Tularemic pneumonia can appear similar to other forms of bacterial and nonbacterial pneumonias, including Q fever, psittacosis, and legionnaires' disease.[180,200]

Oculoglandular tularemia resembles Parinaud's syndrome (granulomatous conjunctivitis with preauricular lymphadenitis) caused by other bacteria (such as *Leptothrix* species and *Mycobacterium tuberculosis*), syphilis, and cat-scratch disease.[98]

The differential diagnosis of oropharyngeal tularemia includes infectious mononucleosis, streptococcal pharyngitis, and plague pharyngitis. The disease most likely to be confused with tularemia is plague, because both diseases occur under similar epidemiologic circumstances and are characterized by certain similar clinical syndromes.[33,208] The bacteria causing plague and tularemia share morphologic and cultural features but can be differentiated serologically and with appropriate microbiologic techniques.

Definitive diagnosis of tularemia is usually based on antibody studies.[122,219] The most common test is agglutination, either tube agglutination or microagglutination.[191] ELISA is also used for diagnosis. An advantage of ELISA is the identification of IgM, IgA, and IgG antibodies.

Diagnosis is established serologically by demonstrating a fourfold or greater rise in titer between acute and convalescent sera taken 1 or more weeks apart. Titers of 80 or greater are generally considered significant in the agglutination test. Values rarely reach that level during the first week of infection but usually reach or exceed that by day 16 of infection. Agglutinating antibodies remain detectable for 10 to 30 years after infection. IgM, IgA, and IgG antibodies also remain detectable by the ELISA technique for at least 11 years after infection. Because of this long persistence of antibody, single titers cannot be used for definitive diagnosis.

Tularemia can also be definitively diagnosed by isolation and identification of the organism from blood or lesions. Samples for culture, however, are not routinely taken in suspect cases, and they are not encouraged because of the high frequency of contamination and infection of laboratory workers using this organism in vitro.

Treatment

Streptomycin, the drug of choice, should be given in a dosage of 30 to 40 mg/kg/day, in two divided portions intramuscularly every 12 hours for 3 days, followed by half that dosage for another 4 to 7 days. The rate of cure for streptomycin is 97%, with no relapses. For gentamicin and tetracycline, respectively, the rates of cure were 86% and 88%; rates of relapse, 6% and 12%; and rates of failure, 8% and 0%. The duration of therapy with gentamicin and a delay in its initiation may have affected outcome in severe cases.[167] For chloramphenicol and tobramycin, cure rates were 77% and 50%; relapse rates, 21% and 0%; and failure rates, 2% and 33%, respectively. Treatment with imipenem/cilastatin was successful in one case, and with ciprofloxacin or norfloxacin successful in six cases; in contrast, therapy with ceftriaxone was ineffective in eight cases.[73]

Prevention

Prevention of tularemia involves avoidance of ectoparasites such as ticks and appropriate hygiene in the handling of infected animal tissues. Insect repellents should be applied when going into areas where ticks, deerflies, and other possible vectors are found. Persons walking in tick-infested brush should wear long pants, with the bottoms of the trouser legs tucked into socks or boot tops. Individuals should check frequently for the presence of ticks while in the field. Ticks should be removed as quickly as possible, preferably with pointed forceps grasping the mouthparts, taking care not to break them or to squeeze the body of the tick in the removal process.

Persons handling suspect animals should wear rubber or plastic gloves. Reservoir animals such as rabbits or muskrats that appear ill should not be handled. When it is necessary to handle sick animals, infection control procedures should include the use of gloves, face masks, and disposable gowns.

Culturing the organism should be attempted only in laboratories that have appropriate containment facilities for handling such dangerous organisms. Laboratory work with *F. tularensis* should always be conducted under an appropriate microbiologic hood. Standard halogen-containing phenol or alcohol-based antiseptics can be used for disinfecting surfaces.

Although person-to-person transmission is rare, reasonable infection control measures should be taken to reduce exposure to aerosols from patients with oropharyngeal or pneumonic tularemia, and exposure to exudates should be avoided.

A live-attenuated vaccine is available as an investigational new drug from the U.S. Army Medical Research Institute for Infectious Diseases at Fort Detrick, Maryland. Physicians using this vaccine must register as collaborative investigators and follow a prescribed protocol in advising the patient, administering the vaccine, and reporting on its use. The vaccine is made available only for those who are at high risk, such as laboratory personnel who frequently work with the organism. The vaccine is effective in preventing the typhoidal form of tularemia[187–190] but will not prevent cutaneous lesions from forming at the site of an inoculation. The vaccine, when admin-

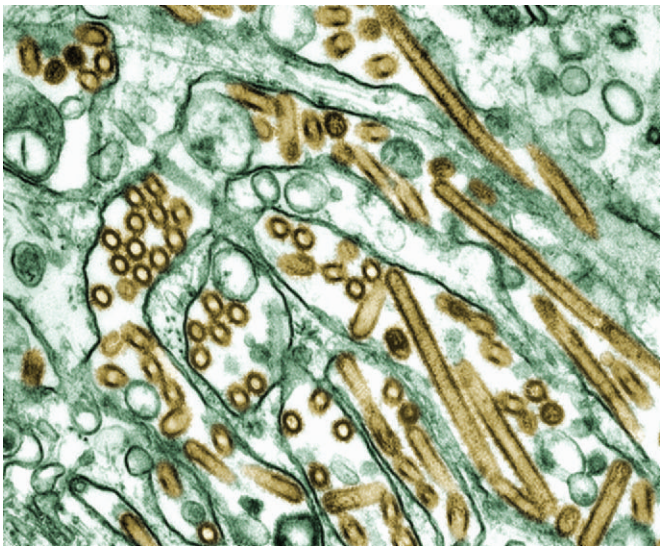

Figure 53-26. Avian influenza. Colorized transmission electron micrograph of avian influenza A H5N1 viruses (seen in *gold*) grown in MDCK cells (seen in *green*). (From CDC Public Health Image Library, available at http://phil.cdc.gov/Phil/home.asp, and courtesy of C. Goldsmith, J. Katz, and S. Zaki, 1997.)

istered by scarification, induces humoral and cell-mediated immune responses.[233]

AVIAN INFLUENZA

The first confirmed human infections by avian influenza H5N1 virus were reported in Hong Kong in 1998, after 6 of 18 patients died.[254] No additional cases were reported until February 2003, when one of two people infected with avian influenza died in Hong Kong.[160] In December 2003, several Asian countries reported an outbreak of avian influenza among poultry, but as of March 2004, transmission to humans had occurred in only Vietnam and Thailand. Although only 32 cases were laboratory confirmed, the mortality reached 70% and, given the limited ability to track disease in these countries, the probability is high that the number of cases is underreported.[224]

Virology

Avian influenza A H5N1 is a subtype belonging to the family Orthomyxoviridae, all of which are single-stranded RNA viruses (Fig. 53-26). Other subtypes include H9N2 and H7N7. Subtype identifiers refer to the specific surface proteins present on the virus. *H* refers to the type of hemagglutinin protein, and *N* refers to the type of neuraminidase protein present on the surface of the viral subtype. Hemagglutinin primarily facilitates binding to the target cell, whereas neuraminidase allows for the release of progeny virions from infected cells.[224] The genetic reassortment that occurs in influenza A results in an antigenic shift, and it is of particular concern because new strains may arise to which humans have no immunity despite previous exposure. It is believed that this antigenic shift is responsible for severe global outbreaks of influenza.

Epidemiology

As of this writing, the highly pathogenic H5N1 avian influenza has been confined to poultry outbreaks in Asia. Transmission to humans of this subtype has been limited to Hong Kong, Vietnam, Indonesia, Thailand, and eastern Europe. Subtype H7N7 occurred in humans, primarily as conjunctivitis, in the Netherlands (2003) and Canada (2004).[81,121] One of the 83 infected patients in the Netherlands died. In an effort to control the outbreak, Japan, the European Union, and the United States have banned poultry imports from infected areas.

Transmission

Most H5N1 infections in humans result from contact with infected birds or their contaminated feces. There are, however, several cases of limited human-to-human transmission reported in the literature, the most recent believed to be in Vietnam in January 2005.[26,121,247]

Symptoms and Diagnosis

Patients typically present within days of exposure with a febrile respiratory illness. GI manifestations such as diarrhea may also occur. Chest radiographs typically show infiltrates of varying patterns. Laboratory tests may demonstrate hepatic dysfunction, renal insufficiency, or coagulopathy, but leukopenia/lymphopenia is common.[224]

Laboratory testing can be performed on a nasopharyngeal aspirate or swab, preferably within 3 days of onset of symptoms.[160] Diagnostic tests available for influenza include viral culture, serology, rapid antigen testing, polymerase chain reaction (PCR), and immunofluorescence. Sensitivity and specificity of these tests vary by the laboratory that performs the test, the type of test used, and the type of specimen tested. In suspected cases of avian influenza, it is critical to collect specimens for viral culture. Only culture isolates can provide information regarding the specific influenza subtypes and strains.

Treatment and Prevention

Although, because of the small number of cases, no efficacy trials have been performed, influenza H5N1 is susceptible to oseltamivir and zanamivir in vitro. It is resistant to amantadine and rimantadine.[224] The virus is killed by heat and disinfectants such as alcohol. However, the influenza virus can survive in feces for months. Persons coming into contact with patients suspected of avian influenza should observe strict hand hygiene and contact precautions (including wearing a particulate respirator, gloves, gown, and goggles).[46]

COWPOX AND MONKEYPOX INFECTION

Poxviruses are large DNA viruses that are implicated in a variety of human diseases, including smallpox, monkeypox, cowpox, and molluscum contagiosum. Historically, immunization against smallpox was performed with an injection of vaccinia virus, which resulted in a local skin lesion. As a result, pox infections are rare, because this immunization provided cross-protection against other poxviruses. Now, however, because smallpox has been eradicated and immunization is no longer recommended, cases of monkeypox, cowpox, catpox, and others will increase.[230]

Figure 53-27. Monkeypox lesions on the arm and leg of a 4-year-old child in Bondua, Grand Gedeh County, Liberia. This infection was caused by a pox virus of the vaccinia, variola, monkeypox type. (From CDC Public Health Image Library, available at http://phil.cdc.gov/Phil/quicksearch.asp.)

Epidemiologic evidence now suggests that cowpox is not enzootic in cattle, that the cow has a minor role, that feline cowpox is important as a source of human infection, and that wildlife, principally rodents, are virus reservoirs.[14] Although poxvirus infection in the domestic cat has only recently been described, the incidence has increased steadily, and cats are now the most frequently reported hosts of poxvirus in England.[230] The infection occurs mostly among hunting cats in the late summer and early autumn; infection in humans is usually reported after close contact with or a scratch from a sick cat, although transmission from rats has also been reported.[243] The infection is manifested as an inflamed vesicular nodule with lymphadenitis, systemic symptoms (e.g., fever), and a rapid but self-limited course, similar to the orf poxvirus carried by sheep, cattle, and goats. This disease has not yet been reported in immunosuppressed persons. No effective treatment is available, but normally the disease is self-limited.

Monkeypox is endemic to central and western Africa, with the first reported case in laboratory monkeys in Copenhagen in 1958.[232] In 2003, the first community-acquired outbreak of monkeypox occurred in the central United States. There were no fatalities and the reservoir was believed to have been infected prairie dogs. Initial symptoms include fever, headache, lymphadenopathy, myalgias, and malaise. Within days, a maculopapular rash erupts, usually spreading in a centrifugal pattern and progressing through several stages before crusting over and sloughing (Fig. 53-27).[66] In the United States, the mode of transmission is believed to have been through respiratory droplets or direct contact.[66] Diagnosis is based on serum samples and scrapings from lesions. As with cowpox, there is no known effective treatment and the disease is self-limited.

HANTAVIRUS PULMONARY SYNDROME

In 1951, American troops in the Korean War were afflicted by an unfamiliar illness characterized by hemorrhagic fever and acute renal dysfunction. This illness became known as Korean hemorrhagic fever, which much later was linked to a Hantavirus infection.[126] Until 1993, hemorrhagic infections associated with Hantavirus strains were thought to be limited to Asia and Eastern Europe, and they largely lacked respiratory symptoms. In 1993, an outbreak of severe respiratory illness with high mortality occurred in the southwestern United States. This outbreak was subsequently investigated and described as the Hantavirus pulmonary syndrome (HPS), caused by a novel Hantavirus.[70]

Virology

HPS is a rodent-borne viral disease characterized by severe respiratory illness and a case-fatality ratio of over 50%. The causative agent has been identified by serologic tests, PCR to RNA, and immunohistochemistry (Fig. 53-28).[43,130] The Sin Nombre virus, also known as the Four Corners virus, is the primary Hantavirus causing HPS in the United States, with the deer mouse *(Peromyscus maniculatus)* as its predominant carrier (Fig. 53-29).[44] Other small mammals, such as piñon mice, brush mice, and western chipmunks, may also be infected.

Hantaviruses of the Bunyaviridae family cause the hemorrhagic fever with renal disease syndrome (HFRS). This is predominantly a disease of East Asia, where it has been called Korean hemorrhagic fever or epidemic hemorrhagic fever.[125] Wild rodents are the vectors of Hantaan, Puumala, Prospect Hill, and Seoul viruses. Hantaviruses have been isolated from the lung tissues of bats.[117]

Epidemiology

Although most cases of HPS have been clustered in the western United States, particularly the Four Corners area (Arizona, New Mexico, Colorado, and Utah), the virus may be present across the entire country.[159,171] The CDC had confirmed 217 cases of HPS in 30 states by mid-1999. From January through May 1999, seven cases of HPS were confirmed in Colorado, New Mexico, New York, and Washington. An additional 11 suspected cases with preliminary clinical and serologic evidence of HPS were reported in Arizona, California, Idaho, Iowa, Montana, New Mexico, and Washington. Eight of the confirmed and suspected cases were from Arizona, Colorado, and New Mexico. In the same 5-month period during each year from 1995 through 1998, this area averaged approximately two cases each year.[44]

Since 1994, the CDC has sponsored continuous monitoring studies of rodent populations at nine sites in Arizona, Colorado, and New Mexico. Spikes in the host population correlate with HPS outbreaks among humans. Hantavirus antibody prevalence in deer mouse populations surveyed during spring 1999 were 35% to 45% in some populations in New Mexico and up to 40% in Colorado. In comparison, prevalence during the population peaks of spring 1998 was less than 10% in New Mexico and approximately 20% in Colorado. These figures were comparable to antibody prevalence of 10% to 15% in deer mouse populations throughout the United States since 1993; during the 1993 outbreak, a prevalence of 30% was detected.[47]

Transmission

The hantaviruses do not cause apparent illnesses in the reservoir hosts, but the animals shed virus in saliva, urine, and feces for weeks. Human infection probably occurs when infective saliva or excreta are inhaled as aerosols, or when excreta are directly inoculated through the skin or perhaps ingested. Persons have been infected with Hantavirus via a rodent bite.

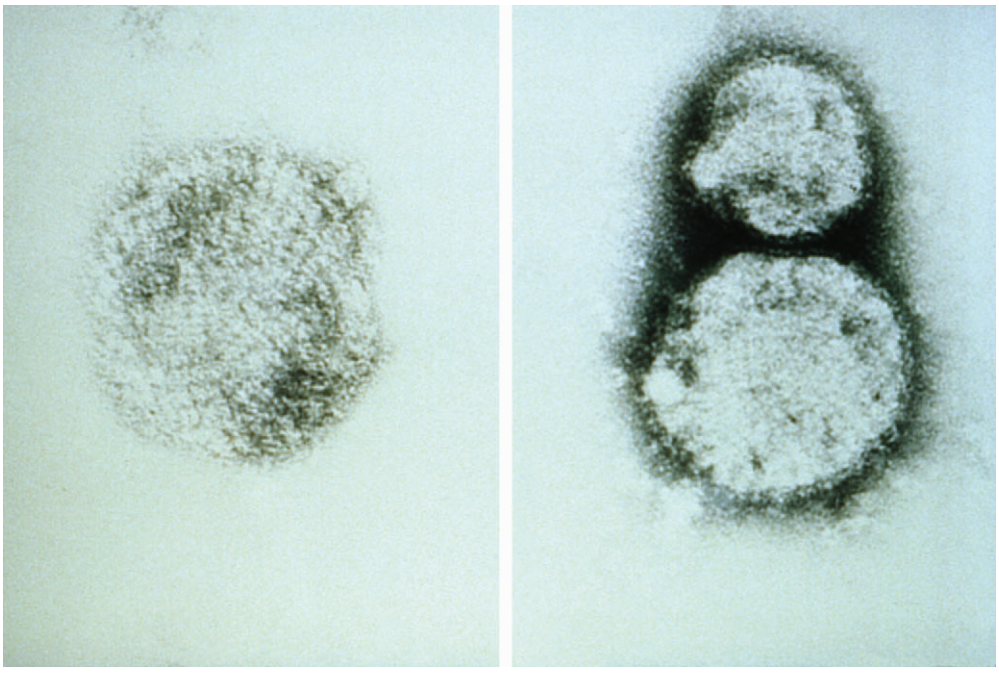

Figure 53-28. Electron micrographs of the Hantavirus virions responsible for causing Hantavirus pulmonary syndrome, or HPS. (From CDC Public Health Image Library, available at http://phil.cdc.gov/Phil/home.asp.)

Figure 53-29. The deer mouse, *Peromyscus maniculatus,* a Hantavirus carrier that becomes a threat when it enters human habitation in rural and suburban areas. All Hantaviruses known to cause Hantavirus pulmonary syndrome (HPS) are carried by New World rats and mice of the family Muridae, subfamily Sigmodontinae. This subfamily contains at least 430 species, which are widespread in North and South America. (From CDC Public Health Image Library, available at http://phil.cdc.gov/Phil/quicksearch.asp, and photo courtesy James Gathany.)

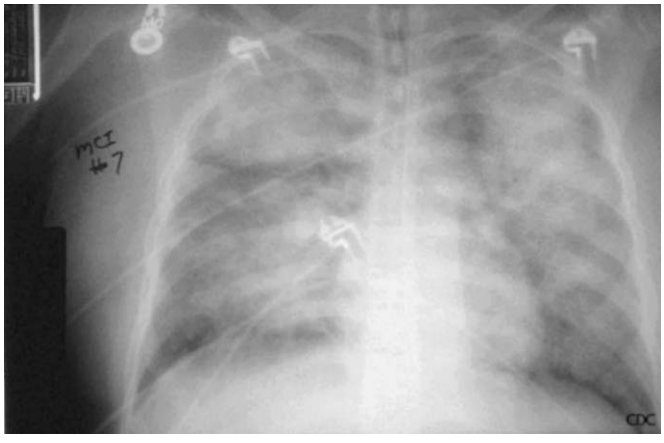

Figure 53-30. Hantavirus pulmonary syndrome (HPS) as shown on chest radiograph. (Available at www.bt.cdc.gov/agent/plague/trainingmodule/3/12hantavirus.htm.)

There is no known arthropod-to-human or human-to-human transmission.

Symptoms

A prodrome of fever, myalgia, and variable respiratory symptoms may include cough and shortness of breath with minimal bronchospasm. Acute respiratory distress rapidly follows. Other early-phase symptoms include headache, chills, abdominal pain, nausea, and vomiting. Patients have often shown hemoconcentration and thrombocytopenia, leukocytosis, hypoalbuminemia, and lactic acidosis.

Rapid deterioration occurs, coincident with marked bilateral pulmonary infiltrates identified on chest radiographic examination (Fig. 53-30). Fever, hypoxia, and hypotension may culminate in death; survivors have few or no sequelae. Autopsies have demonstrated intense pulmonary infiltration, with marked accumulations of Hantavirus antigens in the endothelial cells.

Diagnosis

Laboratory evidence of acute Hantavirus infection can be obtained by any of the following tests: IgM antibodies to Hantavirus antigens, fourfold or greater increase in antibody titers

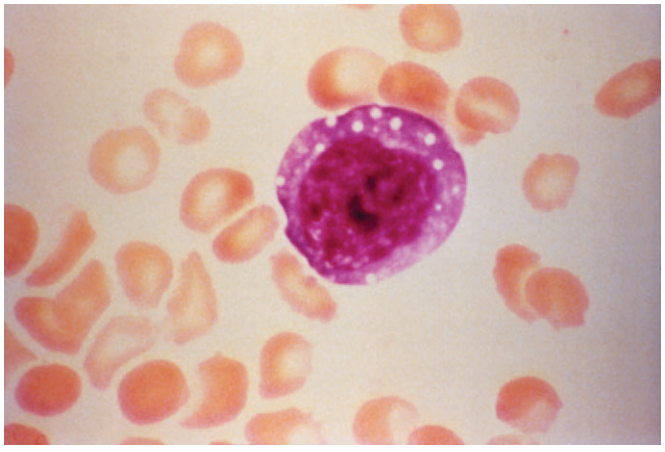

Figure 53-31. Micrograph depicting an atypical, enlarged lymphocyte found in the blood smear from a patient with Hantavirus pulmonary syndrome (HPS). The laboratory finding of this lymphocyte, combined with a bandemia and dropping platelet count, is characteristic of HPS. (From CDC Public Health Image Library, available at http://phil.cdc.gov/Phil/home.asp.)

to Hantavirus antigens in paired serum specimens (Fig. 53-31), positive immunohistochemical stain for Hantavirus antigen in formalin-fixed tissues, or positive PCR from frozen tissue specimens (usually lungs).

Any person with a severe and sudden respiratory illness should be suspected to have been infected with Hantavirus. CDC screening criteria include febrile illness in a previously healthy person characterized by unexplained adult respiratory distress syndrome or bilateral interstitial pulmonary infiltrates developing within 1 week of hospitalization, with respiratory compromise requiring supplemental oxygen.

Treatment

Treatment is supportive. Previously isolated Hantaviruses have demonstrated in vitro sensitivity to ribavirin. The CDC has made the drug available as an investigational agent through an open-label protocol for treatment of patients with HPS. The protocol calls for administration of a 2-g loading dose of intravenous ribavirin, followed by 15 mg/kg body weight every 6 hours for 4 days and then 7.5 mg/kg every 8 hours for another 4 days. Ribavirin contributed to a patient's survival in at least one case.[173]

Prevention

According to the CDC, Hantavirus transmission to humans may be epidemiologically associated with planting or harvesting field crops, occupying previously vacant dwellings, disturbing rodent-infested areas while hiking or camping, inhabiting dwellings with indoor rodent populations, or residing in an area with an increasing rodent density. Most persons with HPS who had high-risk exposures are thought to have been infected in and around their homes; limiting opportunities for domestic exposure is important. Measures to prevent HPS can be divided into four areas, as shown in Box 53-1.

Box 53-1. CDC Recommendations for Preventing Hantavirus Pulmonary Syndrome

1. Eliminate rodent harborage.
   a. Keep cooking, eating, and food storage areas clean.
   b. Cover human food and animal feed.
   c. Contain and elevate garbage.
   d. Seal holes and cracks in dwellings to prevent entrance by rodents.
   e. Clear brush and trash from around homes and outbuildings.
2. Control rodent populations by maintaining snap traps and using rodenticides; in areas where plague occurs, control fleas with insecticides.
3. Safely clean up rodent-infested areas.
   a. Air out infested spaces before cleanup.
   b. Spray areas of infestation and all excreta, nesting, and other materials with household disinfectant or 10% bleach solution, then clean up, seal in bags, and dispose.
   c. Avoid sweeping, vacuuming, or stirring dust until the area is thoroughly wet with disinfectant.
   d. Wear rubber gloves; disinfect gloves before removal, and wash hands afterward.
   e. In areas where plague occurs, spray insecticide on trapped rodents and nesting materials to prevent fleas from abandoning rodents to find new hosts.
4. Avoid rodents when outdoors.
   a. Do not disturb rodent droppings or camp or sleep near burrows or areas where trash is present.
   b. Avoid feeding or handling rodents, even if they appear friendly.

CDC, Centers for Disease Control and Prevention.

HENDRA VIRUS

Hendra virus was first described after a September 1994 outbreak of a severe respiratory illness among a group of horses and humans, in Queensland, Australia. Of 21 affected horses, 14 died of respiratory failure, and of the two humans affected—a trainer and a stable hand—one died of severe interstitial pneumonia.[156] In a second and unrelated outbreak 13 months later, a farmer died of severe meningoencephalitis after contact with a horse infected with Hendra virus.[164]

Virology

Hendra virus is a paramyxovirus that shares 70% to 78% of its nucleotide sequencing with the closely related Nipah virus.[49,134,178] Formerly called equine Morbillivirus, Hendra virus was further sequenced and classified as a paramyxovirus after the 1994 outbreak in Australia.[95]

Epidemiology and Transmission

Outbreaks of Hendra virus have occurred in Queensland, Australia, among horses and humans in close contact. The natural reservoir for Hendra virus is thought to be the fruit bat (*Pteropus* species), but the mode of transmission to horses is unknown.[252] Although the virus is found in a large portion of the fruit bat population, human bat handlers have had no reported cases of the virus.[203] The documented human cases of Hendra virus are thought to be acquired through close contact with infected horses.

Symptoms
The equine form of Hendra virus presents as a severe respiratory infection. In humans, clinical symptoms include severe respiratory symptoms as well as meningoencephalitis. Few cases have been described.

Diagnosis
Definitive diagnosis can be accomplished by viral culture or by ELISA of the CSF and serum samples.

Treatment
Supportive measures are the mainstay of treatment of Hendra virus infection.

Prevention and Control
Early identification of equine cases and quarantine of affected animals is the key to preventing human cases.

NIPAH VIRUS

The original outbreak of Nipah virus in humans occurred in September 1998 in northern Malaysia and spread to farms south of this original epicenter and into Singapore by June 1999. It is believed that the spread within Malaysia resulted from the sale of infected pigs.[154] In Malaysia, there were 265 reported cases with 105 deaths, and in Singapore there were 11 reported cases with 1 death. The outbreak was contained in Singapore once importation of pigs from Malaysia ceased; in Malaysia, more than 1 million pigs from the outbreak area were destroyed before the illness disappeared.[123] The virus derives its name from Kampung Sungai Nipah, the village whose patients supplied the first viral isolates.[244]

Virology
The Nipah virus is a paramyxovirus that is closely related to Hendra virus, a paramyxovirus that caused an outbreak of respiratory disease in horses in Australia.[250] Nipah is a single-stranded RNA virus encapsidated into a helical core surrounded by a lipid membrane containing two distinct viral glycoproteins.[250]

Transmission
Nipah virus is transmitted to humans by contact with infected pigs or fresh pig products. Although there have been isolated suggestions of animals-to-human transmission or human-to-human transmission, these modes have not been confirmed by epidemiologic studies.[51,221,222] There is evidence that fruit bats of the species *Pteropus hypomelanus* bats may serve as a host reservoir for Nipah virus, as they do for Hendra virus. Nipah virus has been isolated from the urine of *P. hypomelanus* on Tioman Island, which is just off the Malaysian coast, and neutralizing antibodies have been detected in two species of fruit bats *(Pteropus hypomelanus, Pteropus vampyrus)*.[50,251] If fruit bats serve as the reservoir, it is unclear how transmission then occurs to pigs.

Symptoms
Onset of symptoms occurs within 2 weeks, and patients present with a wide range of symptoms, ranging from nonspecific fever and headache to rapidly progressive encephalitis.[92,127] The largest population with Nipah virus that has been studied was 94 patients in 1999 at the University of Malaya Medical Center in Kuala Lumpur, Malaysia.[92] More than half of patients had decreased level of consciousness (Glasgow Coma scale [GCS], <15) at presentation and signs of brainstem dysfunction, including abnormal doll's eye reflex, segmental myoclonus, vasomotor instability, and pinpoint pupils. The fatality rate in patients with febrile encephalitis was 32%.

Diagnosis and Treatment
Nonspecific laboratory findings include thrombocytopenia, leukopenia, transaminitis, and CSF with either elevated white blood cell count or elevated protein level.[92] Definitive diagnosis can be accomplished by viral culture or by ELISA of CSF and serum samples.[244] There is no established treatment other than supportive measures.

CYSTICERCOSIS (TAENIASIS)

The encysted larvae, or cysticerci, of the tapeworm *Taenia solium* in the flesh of pigs, known as "measly pork," were well known to the ancient Greeks. These cysticerci are referred to by Aristotle (384 to 322 BC), who, in the section on diseases of pigs in his *History of Animals,* gives an account of "bladders that are like hailstones."[161] These tapeworms have been described throughout the centuries and across the globe.

Parasitology and Transmission
Taenia is a genus of Cestodes, or tapeworms, belonging to the family *Taeniidae*.[157] The two species causing infection in humans are *T. solium*, the pork tapeworm, and *Taenia saginata,* the beef tapeworm. Both cause an intestinal form of tapeworm infection. *T. solium* alone produces the more significant clinical syndrome of cysticercosis, involving neural and other tissues in the human body.

Taenia solium
The life cycle of *T. solium* involves pigs and humans. Humans are infected by ingesting insufficiently cooked pork containing larval tapeworms, or cysticerci. Ingested cysticerci attach to the small intestine with four muscular suckers and a crown of hooklets. Over the next few days, these worms produce proglottids, or segments, which elongate to form a mature tapeworm that can be several meters in length. These proglottids are gravid and release embryonated eggs.

Gravid proglottids or eggs are released in human feces into the environment, where they contaminate water or vegetation and then are ingested by pigs. Once ingested, the eggs become a six-hooked larval form called an oncosphere, which penetrates the intestinal wall of the pig and migrates via the lymphatic or venous system into tissues to complete the cycle and become a cysticercus.

Human cysticercosis develops when humans ingest the embryonated eggs or gravid proglottids that normally infect pigs. The infection is also initiated when humans ingest vegetation or water contaminated with human feces containing *T. solium* eggs. It is hypothesized that carriers of *T. solium* tapeworms can ingest eggs via reverse peristalsis and autoinfection, but this has not been proven. Once ingested, these eggs release oncospheres, which, as in the pig, invade the intestinal mucosa and migrate hematogenously into tissues. Once in the tissues, most commonly brain and muscle, the larval form develops into a cysticercus over 3 to 4 months. These cysticerci may remain viable for several years.

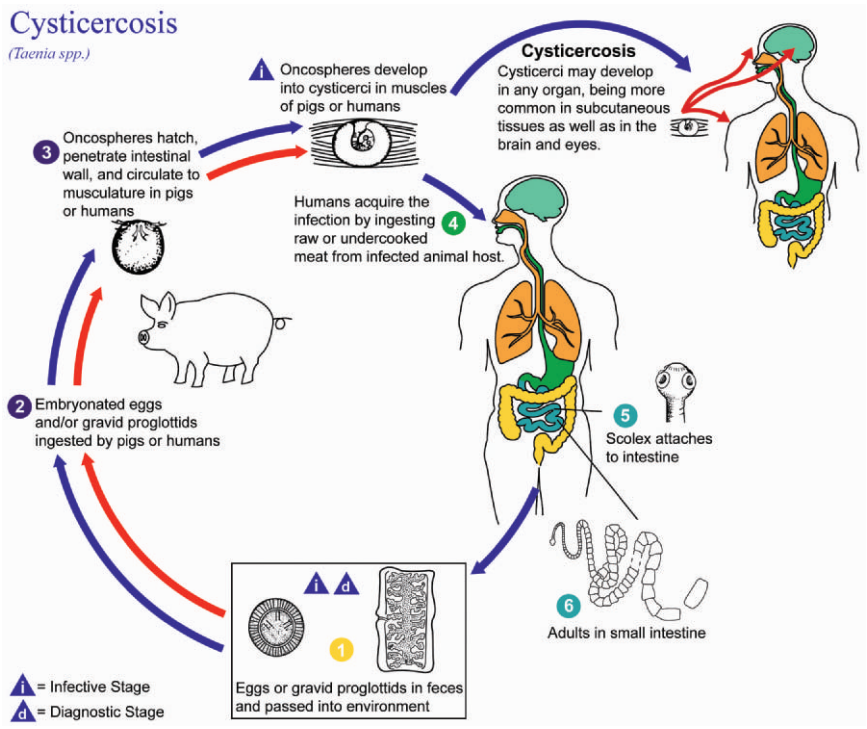

Figure 53-32. Cysticercosis is an infection of both humans and pigs with the larval stages of the parasitic cestode, *Taenia solium*. This infection is caused by ingestion of eggs shed in the feces of a human tapeworm carrier *(1)*. Pigs and humans become infected by ingesting eggs or gravid proglottids *(2)*. Humans are infected either by ingestion of food contaminated with feces, or by autoinfection. In the latter case, a human infected with adult *T. solium* can ingest eggs produced by that tapeworm, either through fecal contamination or, possibly, from proglottids carried into the stomach by reverse peristalsis. Once eggs are ingested, oncospheres hatch in the intestine *(3)*, invade the intestinal wall, and migrate to striated muscles, as well as to brain, liver, and other tissues, where they develop into cysticerci. In humans, cysts can cause serious sequelae if they localize in the brain, resulting in neurocysticercosis. The parasite life cycle is completed, resulting in human tapeworm infection, when humans ingest undercooked pork containing cysticerci *(4)*. Cysts evaginate and attach to the small intestine by their scolices *(5)*. Adult tapeworms develop (2 to 7 m in length); produce less than 1000 proglottids, each with approximately 50,000 eggs; and reside in the small intestine for years *(6)*. (From www.dpd.cdc.gov/dpdx/HTML/Cysticercosis.htm.)

Taenia saginata

The life cycle of *T. saginata* involves humans and cattle (Fig. 53-32). Humans ingest larval tapeworms, or cysticerci, from insufficiently cooked beef. Ingested cysticerci attach to the small intestine with four muscular suckers and a crown of hooklets. These worms then produce proglottids, or segments, elongating to form a mature tapeworm that can parasitize the small intestine of a human for as long as 25 years and grow up to 10 m in length (Fig. 53-33). The proglottids release eggs, which are released via human feces into the environment. These eggs are ingested by cattle, where they form oncospheres, penetrate the intestinal wall, and migrate to other tissues to form a cysticercus and complete the cycle.

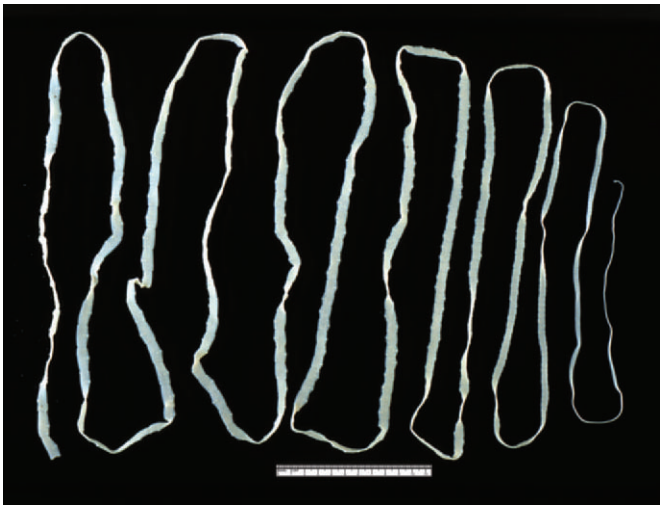

Figure 53-33. Adult *Taenia saginata* tapeworm. Humans become infected by ingesting raw or undercooked infected meat. In the human intestine, the cysticercus develops over 2 months into an adult tapeworm, which can survive for years, attaching to and residing in the small intestine. (From CDC Public Health Image Library, *http://phil.cdc.gov/Phil/home.asp*.)

Epidemiology

T. solium infection is directly associated with eating undercooked pork. It is prevalent in Africa, India, Southeast Asia, China, Mexico, Central America, and South America. It is seen infrequently in Muslim countries and in Israel.

It is important to note that cysticercosis also results from ingestion of water or vegetation contaminated by human feces containing *T. solium* eggs; therefore, human cysticercosis can occur in populations where pork is not consumed and people are not in close contact with pigs.[195] Cysticercosis is estimated to affect 50 million people worldwide, although this may be an underestimate, as many cases are undiagnosed.[194] Prevalence is higher in rural areas and areas where pigs are raised; seroprevalence in endemic regions in South America is as high as 25%.[87]

In the United States, many cases of cysticercosis are seen in immigrants from endemic areas. In one study, 10% of patients in Los Angeles presenting to emergency departments with a seizure had a diagnosis of neurocysticercosis, and in New Mexico it was 6% of patients.[163] People can be infected when traveling to endemic areas and by ingesting produce or water contaminated with *T. solium* eggs.

T. saginata occurs worldwide. Its life cycle is perpetuated by humans and cattle.

Symptoms

The presentations of intestinal infections caused by *T. solium* and *T. saginata* are similar, with mild abdominal discomfort, chronic indigestion, hunger pains, and/or diarrhea. Many patients are asymptomatic and present only after discovering a length of the tapeworm in their feces.

The symptoms of cysticercosis are related to the tissues involved. The syndrome is usually classified as either neurocysticercosis or extraneural cysticercosis. Neurocysticercosis, the more common form, is further divided into parenchymal and extraparenchymal neurocysticercosis.[31]

The more common form of neurocysticercosis is parenchymal. Cysticerci in the brain often locate to the cortex or basal ganglia, and patients most often present with either focal or generalized seizures. Cysts are typically less than 1 cm in diameter. In endemic areas, neurocysticercosis is the most common cause of epilepsy, and the prevalence of epilepsy is higher than in nonendemic areas.[85] Other symptoms of parenchymal neurocysticercosis include headaches, nausea and vomiting, and psychiatric presentations. Over time, parenchymal cysticerci calcify as they degenerate and may serve as foci for the seizures.

One complication of parenchymal neurocysticercosis is cysticercal encephalitis, which is thought to be caused by an intense immune reaction resulting from a massive number of cysts in the brain parenchyma. This reaction can be spontaneous, or it may be provoked by medical therapy that causes degeneration of a large number of cysts simultaneously. Patients present with fever, headache, vomiting, impaired visual acuity, altered mental status, and even seizures. For unknown reasons, this is more common in children and in young women.

Extraparenchymal neurocysticercosis includes subarachnoid cysts, racemose cysticercosis, ventricular cysticercosis, and spinal cysticercosis. Patients with subarachnoid cysts may present with cranial nerve palsies and hydrocephalus because of mass effect and inflammation associated with large cysticerci at the base of the brain.

Racemose cysticercosis is an infrequent but serious form of extraparenchymal neurocysticercosis. It is characterized by proliferating lobulated cysts resembling a cluster of grapes that are typically lodged in the ventricular system. The presentation consists of arachnoiditis, basilar meningitis, and hydrocephalus, and it is associated with a poor prognosis.

A more common form of extraparenchymal neurocysticercosis involves the presence of ventricular cysts, free-floating or attached to the choroid plexus. These cysts cause inflammation in the ventricles, and patients present with obstructive hydrocephalus and elevated intracranial pressure. Symptoms include focal neurologic symptoms, seizures, and/or dementia. An atypical presentation of a ventricular cyst is Bruns' syndrome, which presents with episodes of sudden loss of consciousness associated with head movements; these episodes are caused by intermittent obstruction from mobile cysts in the fourth ventricle.[184]

Spinal cysticercosis, in which cysticerci are located in the subarachnoid space or intramedullary, is less common. These cysts can lead to inflammation and demyelination. Patients present with radicular pain or paresthesias in the affected area. Thoracic lesions are most common.

Extraneural cysticercosis may involve muscles, the eye, and subcutaneous tissues. Ocular cysticercosis, which occurs in 1% to 3% of all cases of cysticercosis, can involve the anterior chamber, subretinal space, or vitreous humor.[88] These cysticerci can cause inflammation when they degenerate, so a thorough ophthalmologic examination should be performed in patients with cysticercosis prior to starting treatment.

Other sites of extraneural cysticercosis include muscles and subcutaneous tissues. Cysticerci in these locations are often asymptomatic but may be seen incidentally as nodules or calcifications on radiographs. Rare cases of cysticerci in cardiac muscle have been reported, with resulting conduction disturbances and cardiomyopathy.[22]

Diagnosis

Stool samples from humans harboring *Taenia* tapeworms may reveal proglottids and eggs. Eggs are spherical, 30 to 40 µm in diameter, and have a thick radially striated shell. *T. solium* and *T. saginata* eggs are identical. Proglottids of *T. solium*, however, are smaller than those of *T. saginata*.

Diagnostic testing for cysticercosis depends on the clinical presentation. Routine blood counts and hepatic panels are normal in typical patients with cysticercosis. The most sensitive serologic test is an enzyme-linked immunoelectrotransfer blot (EITB) assay, with a sensitivity of 98% and a specificity of 100% for patients with active cysticercosis.[89,225] Other serologic tests, including ELISA, CF, radioimmunoassay, and other immunoblots, have been used but are less sensitive and specific. The sensitivity of all tests is lower in patients with inactive cysticercosis or only isolated lesions. The EITB is more sensitive in serum than in CSF.[242] As with any antibody test, a positive test does not indicate active infection.

A lumbar puncture can be useful in the diagnosis of cysticercosis, although it is not necessary. CSF findings include normal glucose, normal to mildly elevated protein and white blood cell counts, and occasional eosinophilia. Serologic tests can be done on CSF, but they are not as sensitive as serum serologies.

Imaging modalities are useful in diagnosing cysticercosis. Plain films may demonstrate calcified cysticercal lesions (Fig. 53-34). CT and magnetic resonance imaging (MRI) are most useful in diagnosing neurocysticercosis. CT findings include nonenhancing hypodense lesions of viable cysts, and calcified lesions of old cysts. Degenerating lesions may have some surrounding inflammatory edema. CT may also demonstrate extraparenchymal cysts in the ventricles or subarachnoid space, and it may demonstrate the resultant hydrocephalus. CT is preferred over MRI for serial examinations to follow progress.

MRI is more sensitive than CT in detecting small lesions and extraparenchymal lesions. MRI is also better at characterizing the scolex and evaluating degenerative changes in the parasite. For spinal cord lesions, MRI is the preferred imaging modality.

Diagnosis of cysticercosis can also be made on pathology, either by brain biopsy (rarely), or by biopsy of muscle or subcutaneous nodule. Cysticerci are 5 to 10 mm in diameter and look like a fluid-filled bladder containing a small larval scolex (Fig. 53-35).

Treatment

For intestinal involvement of either *T. solium* or *T. saginata*, the treatment of choice is praziquantel.

For neurocysticercosis, treatment depends on presentation, and consensus guidelines have been developed.[86] Treatment options include anticonvulsant therapy, antihelminth therapy with albendazole or praziquantel along with corticosteroids, or

Figure 53-34. This x-ray reveals cysticercosis of the muscle due to the presence of the cestode *Taenia saginata,* or beef tapeworm. Humans are the only definitive hosts for *Taenia saginata* and *Taenia solium.* Eggs or gravid proglottids are passed with feces; the eggs can survive for days to months in the environment. (From CDC Public Health Image Library, *http://phil.cdc.gov/Phil/home.asp*.)

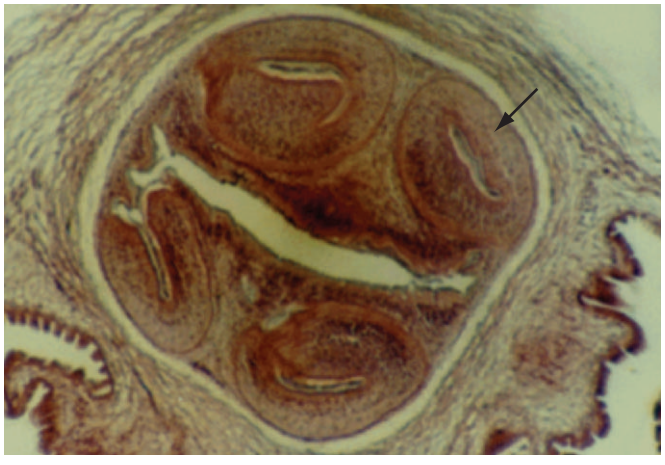

Figure 53-35. Cysticercosis in muscle section. Note sucker (arrow) of bladderworm. The fluid-filled cyst contains an inverted scolex. The worm larva is inverted and as such the rugose tegument is on the inside of the bladder. The rugose tegument will become the adult worm's body surface. (Photo courtesy Department of Pathology, University of Texas Southwestern Medical Center. Accessed at http://pathcuric1.swmed.edu/MicroBiology/LabRef/Parasites/Platyhelminth/SlideC4.html.)

surgery. Anticonvulsant therapy is indicated and extremely important for patients who present with seizures and neurocysticercosis. Drugs of choice include phenytoin or carbamazepine. Length of therapy depends on recurrence of seizures.

Antihelminth therapy is not always indicated in neurocysticercosis, and data showing any added benefit over symptomatic management alone are conflicting.[210] Calcified asymptomatic cysts do not require antihelminth therapy. When it is administered, drugs of choice include albendazole, 15 mg/kg/day for 8 days, or praziquantel, 75 to 100 mg/kg given in three divided doses 2 hours apart. Praziquantel has reduced serum levels in the presence of phenytoin, phenobarbital, or corticosteroids because of cytochrome P-450 induction, so albendazole has a more favorable profile in a patient on anticonvulsants or steroids.

Steroids are administered along with antihelminth therapy to decrease adverse effects of an inflammatory response from degenerating cysts resulting from therapy. Drugs of choice include either prednisolone, 30 to 40 mg/day, or dexamethasone, 12 to 14 mg/day starting 2 days prior to treatment and continuing for 2 to 3 days after therapy is completed. Additionally, for intraventricular cysts alone, steroids alone are indicated to decrease the inflammatory response and the chance of obstruction.

Surgery may be required in the case of obstructive neurocysticercosis. Patients with obstructive hydrocephalus require ventriculoperitoneal shunting. Rarely, patients with intraventricular or subarachnoid cysts may require surgical excision.

Extraneural cysticercosis does not require treatment if it is asymptomatic. Patients with extraneural cysticercosis should be evaluated for neurocysticercosis.

Prevention and Control

Prevention of infection with *T. solium* and *T. saginata* involves good hand washing and hygiene to avoid fecal–oral infection. Prevention of primary infection with *T. solium* requires cooking pork until the interior of the meat is not pink, or else freezing it to –20°C (–4.0°F) for at least 12 hours. Carriers of *T. solium* pose a public health risk and need to be identified and properly treated. Every effort must be made to avoid contact between human feces and the vegetation and water near pigs or livestock.

ECHINOCOCCOSIS

Echinococcosis (hydatidosis, or hydatid disease) is a parasitic disease found wherever sheep are herded and dogs are in close contact with humans. Sheep were first domesticated in the Neolithic era, and large herds of sheep were present during the Mesolithic era around the Caspian Sea in 5000 to 6000 BC.[162] Because dogs have long lived in close quarters with humans, feeding off scraps and offal from livestock and transmitting infection through close contact, echinococcosis is a disease that has very likely been present in human populations since the beginning of humanity. Hydatid disease was well known in ancient times, and there are references to cysts in slaughtered animals in the Babylonian *Talmud,* by Hippocrates in the 4th century BC, by Arataeus in the 1st century AD, and by Galen in the 2nd century AD.[80] Echinococcosis is one of the most broadly spread parasitic zoonoses, with cases occurring in nearly all countries and climates around the world.[60]

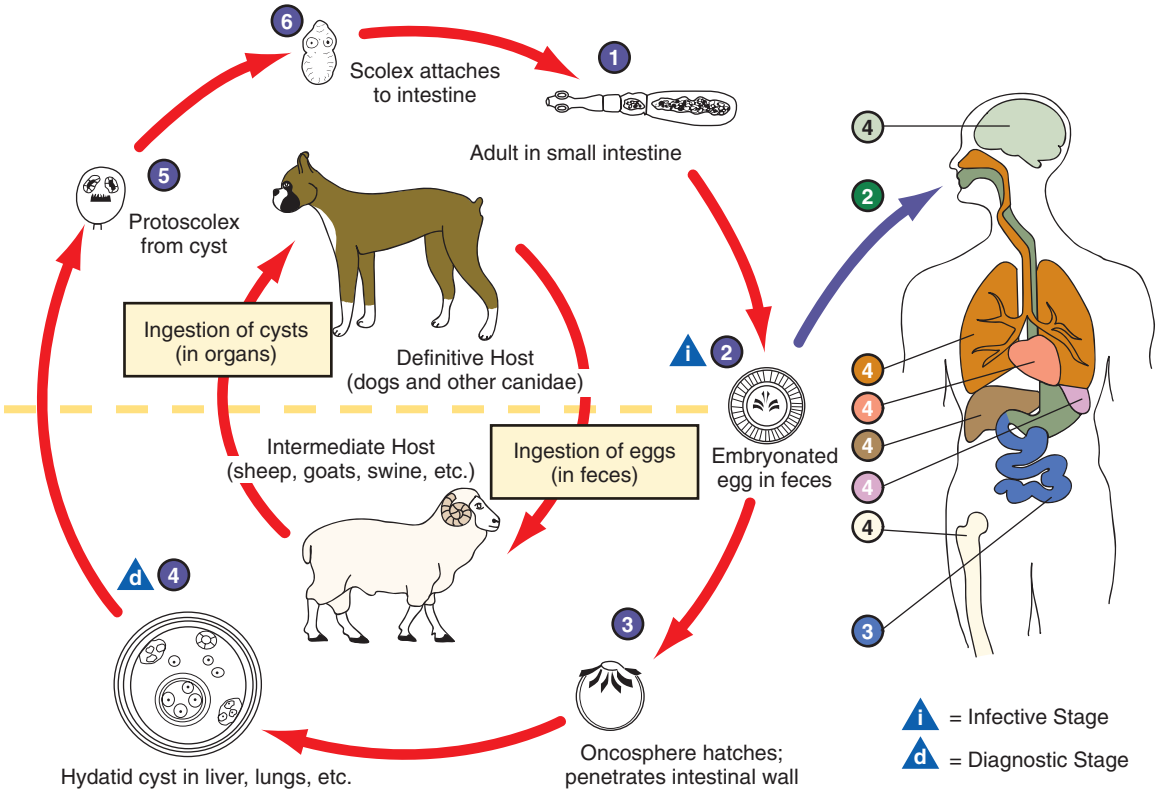

Figure 53-36. The adult *Echinococcus granulosus* (3 to 6 mm long) *(1)* resides in the small intestine of the definitive host (dogs or other canids). Gravid proglottids release eggs *(2)* that are passed in the feces. After ingestion by a suitable intermediate host (under natural conditions: sheep, goat, swine, cattle, horses, camel), the egg hatches in the small intestine and releases an oncosphere *(3)* that penetrates the intestinal wall and migrates through the circulatory system into various organs, especially the liver and lungs. In these organs, the oncosphere develops into a cyst *(4)* that enlarges gradually, producing protoscolices and daughter cysts that fill the cyst interior. The definitive host becomes infected by ingesting the cyst-containing organs of the infected intermediate host. After ingestion, the protoscolices *(5)* evaginate, attach to the intestinal mucosa *(6),* and develop into adult stages *(1)* in 32 to 80 days. The same life cycle occurs with *Echinococcus multilocularis* (1.2 to 3.7 mm), with the following differences: the definitive hosts are foxes, and to a lesser extent dogs, cats, coyotes, and wolves; the intermediate hosts are small rodents; and larval growth (in the liver) remains indefinitely in the proliferative stage, resulting in invasion of the surrounding tissues. For *Echinococcus vogeli* (up to 5.6 mm long), the definitive hosts are bush dogs and dogs, the intermediate hosts are rodents, and the larval stage (in the liver, lungs, and other organs) develops both externally and internally, resulting in multiple vesicles. *Echinococcus oligarthrus* (up to 2.9 mm long) has a life cycle that involves wild felids as definitive hosts and rodents as intermediate hosts. Humans become infected by ingesting eggs *(2),* with resulting release of oncospheres in the intestine and the development of cysts *(4), (4), (4), (4), (4), (4)* in various organs. (From www.dpd.cdc.gov/dpdx/HTML/Echinococcosis.htm.)

Parasitology and Transmission

Echinococcus is a genus of cestodes, or tapeworms, belonging to the family Taeniidae.[102] Human echinococcosis is caused by infection with the larval stages of *Echinococcus*. Several species of *Echinococcus* are known to cause disease in humans. *Echinococcus granulosus* causes cystic echinococcosis, the most common form of the disease in humans. *Echinococcus multilocularis* causes alveolar echinococcosis, accounting for 5% of hepatic hydatid disease. *Echinococcus vogeli* and *Echinococcus oligarthrus* very rarely cause echinococcosis in humans.

The life cycle of *Echinococcus* involves a definitive (e.g., a dog) and an intermediate (e.g., a sheep) host. Humans become infected as accidental hosts in this life cycle (Fig. 53-36).[68] In the definitive host, the adult tapeworm inhabits the small intestine. These adult worms differ by species. *E. granulosus* worms are 2 to 7 mm long and consist of a scolex with hooks and suckers as well as at least three proglottids. *E. multilocularis* worms are up to 4 mm long with two to six proglottids. Proglottids are segments consisting of both male and female sexual organs that produce eggs, which contain oncospheres. These eggs are 30 to 40 µm in size.

The definitive host may harbor thousands of worms, each of which can produce thousands of eggs from the proglottids each day. These eggs are expelled in the feces of the definitive host. Intermediate hosts, and incidentally humans, are infected by ingesting these eggs from the environment. The eggs hatch within the intermediate host and release oncospheres, which then penetrate the intestinal mucosa, enter the blood and lymphatic systems, and migrate to visceral organs. After reaching these organs, most commonly the liver, the oncospheres develop into fluid-filled cysts, which then develop into the metacestode or hydatid cyst. Adult forms, or protoscolices, eventually develop within these hydatid cysts.

The adult protoscolices develop into secondary daughter cysts within the intermediate host (Fig. 53-37). If infected organs from the intermediate host are ingested by the definitive host, the protoscolices develop into mature worms within the definitive host's small intestine over a period of 4 to 7 weeks to com-

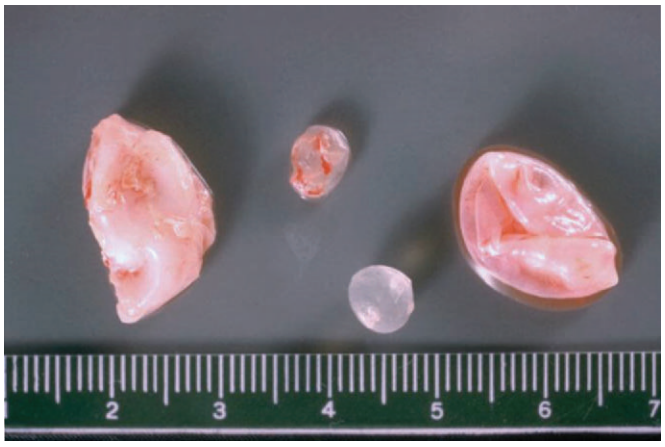

Figure 53-37. Gross pathology of membrane and hydatid daughter cysts from human lung. (From CDC Public Health Image Library, available at http://phil.cdc.gov/Phil/quicksearch.asp.)

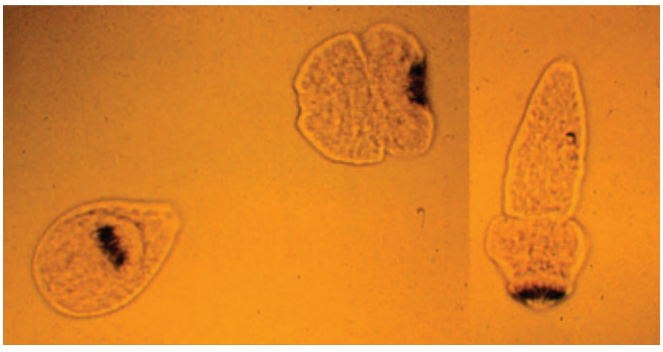

Figure 53-38. "Hydatid sand." Fluid aspirated from a hydatid cyst shows multiple protoscolices (approximately 100 µm), each of which has typical hooklets. The protoscolices are normally invaginated *(left),* and they evaginate (*middle,* then *right*) when put in saline. (Image contributed by Georgia Division of Public Health.)

plete the life cycle. A definitive host is required for development of mature worms and thus the release of infectious eggs into the environment. Adult tapeworms do not develop in the intestines of humans or intermediate hosts; therefore, direct transmission of echinococcosis from human to human does not occur.[157]

Epidemiology

The epidemiology of *Echinococcus* differs by species:

Echinococcus granulosus

E. granulosus occurs nearly worldwide, and more frequently in rural, grazing areas where dogs ingest organs from infected animals. Human cystic hydatidosis is a significant public health problem in South and Central America, the Middle East, some sub-Saharan countries, China, and the former Soviet Union. Infection rates are highest in endemic rural areas, with prevalence rates of 2% to 6% reported.[192]

The definitive hosts for *E. granulosus* are dogs and other canids. The intermediate hosts are sheep, although other livestock can occasionally act as intermediate hosts. Human infection results from ingestion of eggs from the environment. These eggs may be ingested via contaminated water or vegetables, or from direct contact with infected dogs through fecal–oral contact, which occurs more commonly in children.

Echinococcus multilocularis

E. multilocularis occurs in the northern hemisphere, including central Europe and the northern parts of Europe, Asia, and North America. *E. multilocularis* has a number of definitive hosts, including foxes, wolves, dogs, coyotes, and cats. Intermediate hosts are rodents, including voles, muskrats, and housemice.

Echinococcus vogeli and *Echinococcus oligarthrus*

E. vogeli and *E. oligarthrus* occur in Central and South America. The definitive hosts of *E. vogeli* are wild canids, and the intermediate hosts are rodents. The definitive hosts of *E. oligarthrus* are wild felids, and the intermediate hosts are rodents.

Symptoms

Echinococcus granulosus (Cystic Hydatidosis)

E. granulosus in humans develops as a unilocular hydatid cyst. This cyst is a slow-growing, space-occupying cystic lesion enclosed by a laminated germinative membrane that produces protoscolices, or tapeworm heads (Fig. 53-38). The cyst grows as it slowly accumulates fluid. Spillage of this fluid can result in severe reactions including anaphylaxis and death, and it can also lead to dissemination of infection from the release of protoscolices.[157]

Because of the slow growth of the cysts, infections remain silent for many years before causing symptoms. Symptoms depend on the organs involved. The liver is most commonly affected, seen in two thirds of patients. The lungs are involved in approximately 25% of patients, whereas other organs, including brain, bone, heart, muscle, and kidney, are rarely involved. Single-organ involvement is found in 85% to 90% of patients, and more than two thirds of patients have only a single cyst.

Hepatic involvement, the most common form of echinococcosis, can involve abdominal pain, an abdominal mass, or biliary ductal obstruction. Mass effect on the portal veins, hepatic veins, and inferior vena cava can result in portal hypertension, venous obstruction, or the Budd-Chiari syndrome.

Pulmonary involvement can present with cough, dyspnea, chest pain, and hemoptysis. Seizures or elevated intracranial pressure can indicate brain involvement. Bony cysts may present as pathologic fractures; the spine, pelvis, and long bones are most commonly involved. Spontaneous or traumatic rupture of cysts releases toxic fluid into involved areas and can produce fever, urticaria, eosinophilia, and anaphylactic shock.

Echinococcus multilocularis (Alveolar Hydatidosis)

E. multilocularis infection follows a more aggressive course than does *E. granulosus* infection. *E. multilocularis* forms cysts composed of a thin laminar layer without a limiting membrane or germinal layer. These cysts behave like a slowly growing, destructive, polycystic tumor. The lesions are made up of multiple irregular cysts, often with mixed solid and cystic compo-

nents. This polycystic mass invades and destroys adjacent tissues and can metastasize to distant sites in the body.

The primary infection in alveolar hydatidosis is hepatic in nearly all cases, but spread to other organs such as the lungs and brain occurs frequently by direct extension or hematogenous or lymphatic dissemination. Presenting symptoms include right upper quadrant discomfort, malaise, and weight loss, and they most commonly relate to hepatic involvement. Biliary obstruction, portal hypertension, and the Budd-Chiari syndrome can also occur.

Alveolar hydatidosis is a more aggressive disease than cystic hydatidosis. Untreated, alveolar hydatidosis has a 10-year survival rate of less than 10% after the onset of symptoms, and a 15-year survival rate of less than 1%.[5]

Echinococcus vogeli and *Echinococcus oligarthrus*

E. vogeli and *E. oligarthrus* cause polycystic hydatid disease affecting the liver and occasionally the lungs. Both are rare causes of hydatid disease in humans.

Diagnosis

The diagnosis of echinococcosis relies mainly on findings by ultrasonography or other imaging techniques supported by positive serologic tests. Serology testing should be used prior to invasive methods of diagnosing echinococcosis. In patients with negative serology but suspect lesions on imaging, fine-needle biopsy via ultrasonographic guidance can be useful for confirmation of the diagnosis. However, precautions must be taken with this procedure to avoid leakage of toxic cystic fluid and to control any allergic reaction that results from this leakage.

False-negative serology results occur when the location, integrity, or vitality of the cyst is such that the patient has not mounted an antibody response to the cyst. Intact cysts result in the lowest rate of antibody response. Cysts in bone and liver are more likely to elicit antibody response than those in the lungs or brain. Patients with calcified or dead cysts are generally seronegative. False-positive serology results may occur in patients with other tapeworm infections, cancer, and chronic immune disorders.

Echinococcus granulosus

Routine serum tests, such as complete blood count and hepatic panels, may demonstrate mild abnormalities but are not diagnostic. Eosinophilia is uncommon and usually present only if there has been leakage of antigenic material from cysts.[78]

Serology tests for *E. granulosus* include ELISA, indirect hemagglutination (IHA), and indirect FA tests, with sensitivity ranging from 60% to 90%. The best sensitivity is obtained by using a combination of tests. These tests have limited usefulness for following the course of disease after treatment; imaging techniques are the modality of choice for monitoring disease progress.

Imaging is extremely useful in diagnosing cystic hydatidosis. Although plain radiographs may reveal calcified cysts, ultrasound, CT scanning, and MRI are preferable for diagnosis. Ultrasound has the benefits of being easy to perform and less expensive than CT or MRI, and it is particularly useful for evaluation of intra-abdominal cysts. These cysts most commonly appear as anechoic, smooth, round cysts that look much like benign cysts. Septation can be seen when daughter cysts are present. The "hydatid sand" may be visible on ultrasound imaging.

The CT scan is useful for better defining the site, size, and composition of cysts, and is better than ultrasound at detecting extrahepatic cysts. CT is also useful to monitor for recurrence during or after therapy. The sensitivity of CT is cited as 95% to 100%.[183] MRI is better than ultrasound for mapping extrahepatic cysts, particularly in the brain.[226] However, MRI has little benefit over CT scanning for abdominal or pulmonary hydatid cysts except for better delineating venous or biliary involvement. Other techniques, such as endoscopic retrograde cholangiopancreatography, are also useful in diagnosing biliary involvement of hydatid disease.

Echinococcus multilocularis

Nonspecific leukopenia or thrombocytopenia, mild eosinophilia, and nonspecific liver function abnormalities may be detected but are not diagnostic. Hypergammaglobulinemia and elevated serum IgE levels are commonly present.

Serology tests in alveolar hydatidosis are much more sensitive than for cystic hydatidosis, with a higher proportion of patients having an antibody response to *E. multilocularis*. Immunoaffinity-purified *E. multilocularis* antigens used in ELISA have a sensitivity of 95%. In alveolar echinococcosis, serial serology is useful for following disease course given its increased sensitivity.

The preferred imaging modalities for alveolar hydatidosis are similar to those for cystic hydatidosis—ultrasound, CT, and MRI. These polycystic lesions typically have irregular borders, central necrosis, and irregular calcifications. They are often difficult to distinguish from a tumor. MRI is useful for delineating obstruction of the inferior vena cava or for brain lesions.

Treatment

Surgery is the most common form of treatment for echinococcosis and results in a 90% cure rate when the cysts are completely excised.[137] Cystic hydatidosis is more often curable with surgery than is alveolar hydatidosis, which has tends to be more invasive. After surgery, medication may be necessary to keep the cysts from recurring. The drug of choice for treatment of cystic echinococcosis *(E. granulosus)* is albendazole. Albendazole or mebendazole is the recommended treatment for *E. multilocularis* infections.[114]

Not all patients with hydatid disease are surgical candidates. The World Health Organization (WHO) recommendations for hydatid disease list the following contraindications for surgery: patients at the extremes of age, patients in very poor general condition, pregnant women, patients with multiple cysts or cysts that are difficult to access, and patients with dead or completely calcified cysts.[245] Patients who are not good candidates for surgery have two options: chemotherapy with albendazole or mebendazole, and a newer treatment, the PAIR (percutaneous aspiration, introduction of a protoscolicidal agent, and reaspiration) procedure.[111]

The PAIR procedure involves percutaneous puncture of cysts under ultrasound guidance, aspiration of cyst fluid, and injection of a protoscolicidal agent such as hypertonic saline or ethanol into the cyst cavity. The cyst is then reaspirated after 15 minutes. PAIR has been employed primarily for cysts in the liver, although cysts in other sites have been treated in this manner. The WHO currently recommends PAIR for inoperable patients and for patients who refuse surgery. In the future, as

experience with this modality grows, PAIR may be used more widely for initial treatment of echinococcosis.

PAIR is contraindicated in patients with inaccessible cysts or cysts with a high risk of spillage into the abdominal cavity. It is also contraindicated in inactive cysts and for cysts with biliary communication. Risks of the procedure include spillage of fluid and the resulting allergic reaction, bleeding, infection, chemical sclerosing cholangitis, and biliary fistulas. Treatment with albendazole or mebendazole is recommended prior to and after the procedure to decrease risks and increase success of therapy.

Prevention and Control

Prevention and control of echinococcosis involves disrupting the life cycle of the parasite.[60] Control measures include eliminating stray dogs and preventing dogs from consuming infected viscera by prohibiting home slaughter of sheep. Control programs are ongoing in Australia, Argentina, Chile, Brazil, Peru, China, Portugal, and many Mediterranean countries. Many of these programs include surveillance testing of dogs for *Echinococcus* and treating infected dogs with praziquantel. The most successful programs, in Iceland, New Zealand, and Tasmania, relied on regular repeated treatment with praziquantel of all at-risk dogs to eliminate the definitive host carriers of *E. granulosus*.[60]

Vaccination against *E. granulosus* is on the horizon. A vaccine for *E. granulosus*, the EG95 vaccine, is composed of a purified recombinant protein of the echinococcal oncosphere as well as an adjuvant. It is given as two doses 1 month apart, followed by an annual booster. Initial studies show it affords 95% immunity in sheep and other livestock.[59,132]

For humans, prevention involves avoiding close contact with dogs and careful washing of vegetables and fresh produce when in endemic areas. The future may hold vaccination for humans as well.

TRICHINOSIS

Trichinosis is an infection caused by nematodes in the genus *Trichinella*. In the past, only one species, *Trichinella spiralis*, was recognized. Isoenzyme and DNA analysis indicate that the genus is polyspecific.[172] Eight gene pools, T_1 through T_8, have been identified. T_1 is classic *T. spiralis;* the principal reservoir is in domestic swine, but some wild animals can also be infected. T_2, *Trichinella nativa*, is found primarily in terrestrial mammals such as the bear, walrus, or fox in Arctic and sub-Arctic regions. Most human infections are caused by T_1, and fewer by T_2. Relatively few data are available on how frequently the other species infect people. T_3 occurs in bears (Ursidae), and T_7 and T_8 occur in African Hyenidae and Felidae. Only T_4, *Trichinella pseudospiralis*, can infect mammals and birds.

Unless stated otherwise, the rest of this discussion relates to trichinosis generically or to *T. spiralis* specifically.

Historical Aspects

The Bible references swine as dirty animals, and some religions advocate not eating pork; it is hypothesized that part of this practice encouraged public health by preventing parasitic infections such as trichinosis and cysticercosis. *Trichinella* cysts were first noticed by Paget during an anatomic dissection in 1836, when distinct white lesions were found throughout a muscle specimen. The association of the encysted organism and ingestion of contaminated meat was not made until 1850. In 1862, Friedreich diagnosed and described the first clinical case of acute trichinosis. Outbreaks in Germany in 1849 and 1865 were associated with mortality rates of 19% and 30%, respectively. Examination of diaphragmatic muscle samples in the United States between 1936 and 1941 revealed *Trichinella* organisms in one out of every six samples tested (16.7%). National reporting of trichinellosis began in 1947. The incidence of this disease has decreased significantly since the passage of legislation (the Federal Swine Health Protection Act of 1980) prohibiting the feeding of raw sewage to swine, aided by the widespread freezing of pork and the increasing public awareness of the dangers of eating inadequately cooked pork products (Fig. 53-39).

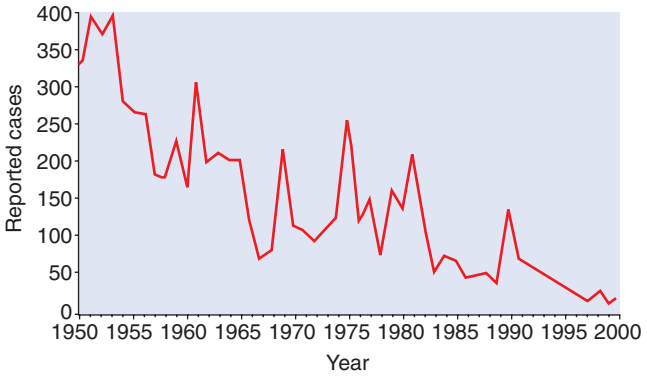

Figure 53-39. Reported cases of trichinosis in the United States, 1950 to 1999. (Modified from Centers for Disease Control and Prevention: MMWR Morb Mortal Wkly Rep 40, 1991.)

Life Cycle

The life cycle of *Trichinella* organisms is unusual in that every host is necessarily both a definitive host, harboring the adult stage of the parasite, and an intermediate host, harboring the larval stage (Fig. 53-40).

The infection is acquired by ingestion of larvae encysted in skeletal muscle. The worms mature within a few days in the small intestine. The female burrows into the mucosa and deposits larvae in the tissue, starting around the fifth day after infection. Most larvae are deposited within 4 weeks, but they can be produced for as long as 4 months. The larvae enter the circulation and invade skeletal muscle within 7 to 14 days (Fig. 53-41). They become encapsulated around day 21 after infection and are then infective for the next host that ingests them.

Epidemiology and Transmission

All carnivorous and omnivorous mammals are susceptible to trichinosis, but most human infections are acquired by eating raw or undercooked pork. Game animals can harbor the parasite. Trichinosis was found in 1.3% of black bears in New England.[103] It can infect other species of bears, raccoons, opossums, seals, walruses, peccaries, and wild swine.

Rodents, such as mice and rats, are commonly infected in nature. Except in certain cultures, these small rodents are rarely eaten by people, unlike their larger cousins: squirrels, woodchucks, muskrats, agoutis, and capybaras. These larger rodents are primarily herbivorous but are still an occasional source of human infection.

Although experimentally susceptible to trichinosis, herbivores, such as members of the deer and antelope families, are

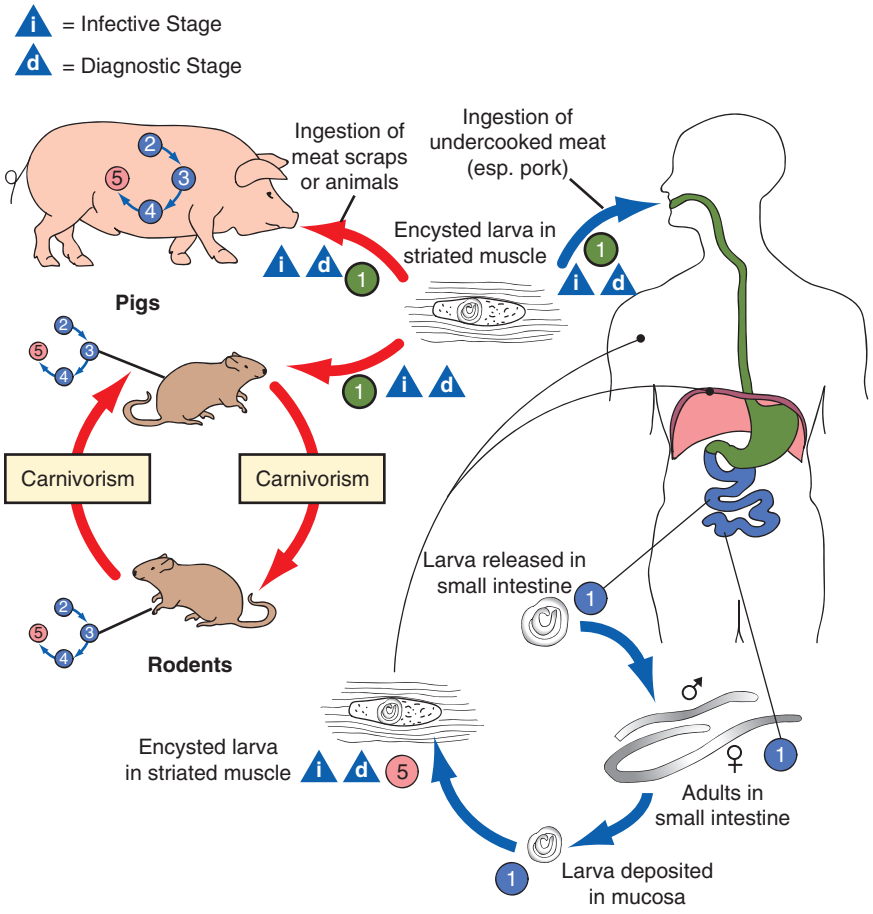

Figure 53-40. Trichinellosis is acquired by ingesting meat containing cysts (encysted larvae) *(1)* of *Trichinella.* After exposure to gastric acid and pepsin, the larvae are released *(2)* from the cysts and invade the small bowel mucosa, where they develop into adult worms *(3)* (females, 2.2 mm in length; males, 1.2 mm; lifespan in the small bowel, 4 weeks). After 1 week, the females release larvae *(4)* that migrate to the striated muscles where they encyst *(5). Trichinella pseudospiralis,* however, does not encyst. Encystment is completed in 4 to 5 weeks, and the encysted larvae may remain viable for several years. Ingestion of the encysted larvae perpetuates the cycle. Rats and rodents are primarily responsible for maintaining the endemicity of this infection. Carnivorous or omnivorous animals, such as pigs and bears, feed on infected rodents or meat from other animals. Different animal hosts are implicated in the life cycle of the different species of *Trichinella.* Humans are accidentally infected when eating improperly processed meat of these carnivorous animals (or eating food contaminated with such meat). (Available at www.dpd.cdc.gov/DPDx/HTML/Trichinellosis.htm.)

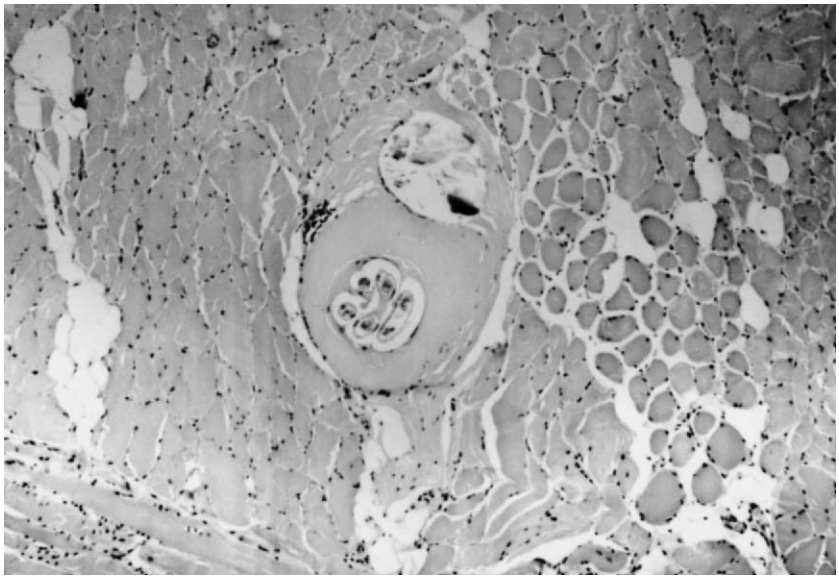

Figure 53-41. Trichinosis in a polar bear. A larva is seen within a muscle fiber in the center of the picture. The parasite found in Arctic mammals is highly resistant to freezing and has been given a separate species status, *Trichinella nativa.* (Hematoxylin and eosin, ×100.)

almost never infected naturally, and consumption of their flesh is not associated with this infection. Interestingly, some outbreaks of trichinosis have occurred in people who consumed horseflesh.[41] The horses could have become infected by consuming mice, dead or alive, in their feed. Alternatively, larvae passed in the stool of infected rodents could have been ingested in the horses' grain or hay.

Swine that are privately raised and slaughtered are a continuing source of human infections in many areas, including the northeastern United States.[9] Bears, walruses, and feral swine have been the principal nondomestic sources of trichinosis in the United States.[148] From 1991 to 1996, three deaths in 230 cases were reported to the CDC (an average of 38 cases per year), including 14 multiple case outbreaks from 31 states and Washington, D.C. Information on the suspected food item was available for 134 (58%) of the 230 reported cases. Pork was implicated in 80 (60%) cases, bear meat in 31 (23%), walrus meat in 13 (10%), and cougar meat in 10 (7%). Sausage was the most frequently implicated pork product (i.e., 57 of the 64 cases for which the form of the pork product was identified).[155] Wild boars are a source of infections in Germany.[103] Twenty-six cases of trichinosis reported in the United States from 1975 to 1989 were acquired during foreign travel. Seventeen of these persons had traveled to Mexico or Asia.[148]

Figure 54-42. Clinical appearance of the eyes of a patient with trichinosis. The patient had periorbital swelling, muscle pain, diarrhea, and 28% eosinophils. (From CDC Public Health Image Library, available at http://phil.cdc.gov/Phil/quicksearch.asp, and courtesy Dr Thomas Sellers, Emory University.)

Symptoms

The signs and symptoms of trichinosis are closely related to the activities of the parasite in its life cycle. The severity of disease is directly related to the number of adult and larval worms present. GI symptoms predominate during the first week after ingestion of infected meat. The worms mate and invade the intestinal mucosa during the first 48 hours. The female worm is capable of producing up to 1500 larvae during her lifetime. Larvae are deposited starting on approximately the fifth day after ingestion. This activity results in irritation of the bowel, with nausea, vomiting, variable diarrhea, and abdominal pain. Fever may occur. These symptoms are often mistaken for various forms of food poisoning. GI symptoms may continue until the females are cleared from the intestinal tract at approximately 4 to 6 weeks after infection.

Larval production reaches a peak during the second week after infection. The larvae start to invade skeletal muscle as early as the seventh day. During larval migration, direct capillary damage occurs, resulting in facial edema, especially involving the periorbital area, which may be accompanied by photophobia, blurred vision, diplopia, and complaints of pain on moving the eyes (Fig. 53-42). Splinter hemorrhages may appear in the nail beds, and there may be cutaneous petechiae (Fig. 53-43). Hemorrhagic lesions can also occur in the conjunctivae and retinae. The temperature may reach 40.5° C (105° F).

Eosinophils start to increase in peripheral blood during the second week, often exceeding 20% of the total white blood cell count after the third week of infection. The eosinophil count returns to normal from 6 to 12 months after infection. The GI symptoms may continue during this period, until the females are cleared from the intestinal tract approximately 4 to 6 weeks after infection. During the second phase of infection, migrating larvae can cause pulmonary damage, resulting in cough, dyspnea, and pleuritic pain. There may be hemoptysis. Myocarditis can occur and may be life-threatening. Damage to the brain or meninges by migrating larvae can cause encephalitic or meningitic symptoms. A spinal tap may reveal eosinophils in the CSF. The third phase of infection is encystment of the larvae within skeletal muscle, starting about the second or third week after infection. This can cause significant myalgias and stiffness in affected muscle groups. The final phase of infection occurs as the larvae die and become calcified. This is a period of convalescence, which is usually asymptomatic, and typically occurs between 6 and 18 months after infection.

Figure 53-43. The parasitic disease trichinosis is manifested by splinter hemorrhages under the fingernails. (From CDC Public Health Image Library, available at http://phil.cdc.gov/Phil/quicksearch.asp, and courtesy Dr Thomas Sellers, Emory University.)

In the Inuit population of northeastern Canada, trichinosis is associated with eating raw walrus, and it is characterized by prolonged diarrhea and brief muscle symptoms. High peripheral eosinophilia and high *Trichinella* antibody titers occur. The disease is probably caused by *T. nativa*, and at least some cases may be associated with re-infection.[135]

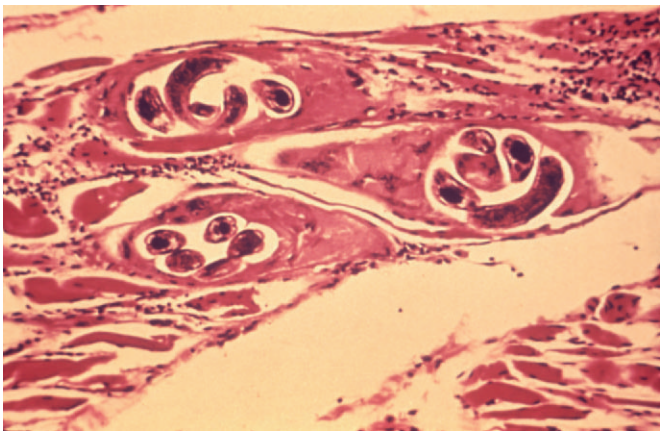

Figure 53-44. Micrograph of developing *Trichinella* cysts within human muscle tissue. (From CDC Public Health Image Library, available at http://phil.cdc.gov/Phil/quicksearch.asp.)

Diagnosis

Larvae are sometimes passed in the stool in the early stages of infection, but this occurs infrequently and inconsistently and cannot be used for diagnosis.

Trichinosis is definitively diagnosed by biopsy of the gastrocnemius muscle or of clinically affected (painful, tender) muscles (Fig. 53-44). The larvae are demonstrable in muscle beginning about the seventh day after infection. Diagnosis can also be made serologically, using the bentonite flocculation test (BFT), latex particle agglutination, or countercurrent immunoelectrophoresis. The BFT involves a suspension of aluminum silicate particles (bentonite) to which *Trichinella* antigen is bound and incubated with dilutions of serum from the patient. All these tests usually do not become positive until 3 to 4 weeks after infection. Newer ELISAs are available that measure reaction by different immunoglobulin classes. Most ELISAs offer greater specificity and sensitivity and become positive earlier than many of the other tests.[8,193,228] An ELISA for IgG, using the excretory–secretory antigen of *T. spiralis* larvae, had specificity and sensitivity of 100% at days 57 and 120 after infection, but it was negative at day 23.[138] An experimental assay, the dissociated enhanced lanthanide fluoroimmunoassay (DELFIA), can detect as little as 1 ng of antigen per milliliter of serum. Circulating antigen was detected in mice as early as 7 days after infection.[120]

Treatment

No satisfactory, safe, and effective drug is available for the elimination of larvae, which are responsible for most of the pathologic changes and symptoms of trichinosis. Thiabendazole (25 mg/kg twice a day for 5 days, maximum 3 g/day) is effective against adult worms in the intestine. Its efficacy against larvae is questionable, however, and it can cause serious side effects, such as fever, increased edema, and myocarditis.[118] Mebendazole (200 to 400 mg three times a day for 3 days, then 400 to 500 mg three times a day for 10 days) is better tolerated,[165] but poor intestinal absorption reduces its use in extraintestinal trichinellosis. Albendazole and flubendazole are well absorbed and may be more effective, but the supporting data are scarce.

Steroids (e.g., prednisone, 30 to 60 mg/day orally for 10 to 30 days) can be given for relief of severe illness, such as myocarditis caused by migrating larvae.[118] The dosage and duration of treatment are individually determined by clinical response. Steroids reduce the inflammatory response to larvae but can also interfere with rejection of adult females in the intestine, thus prolonging the period of larva deposition.

Prevention

Trichinosis is prevented by cooking meat to an internal temperature of 65.6° to 77° C (150° to 170° F). Most *Trichinella* larvae are killed by freezing. The time required depends on the thickness of the meat and the freezing temperature. Holding meat at 15° C (5° F) for 20 days, 23.3° C (10° F) for 10 days, or 28.9° C (20° F) for 6 days is recommended. Salting, drying, and smoking are not always effective. Note that *T. nativa* found in Arctic mammals (see Figure 53-41) is resistant to freezing.

BOVINE SPONGIFORM ENCEPHALOPATHY AND VARIANT CREUTZFELDT-JAKOB DISEASE

Bovine spongiform encephalopathy (BSE) is a transmissible spongiform encephalopathy (TSE), a family of fatal degenerative diseases of the nervous system that affect humans and animals. In humans, TSEs include Creutzfeldt-Jakob disease (CJD), Gerstmann-Sträussler-Scheinker (GSS) syndrome, fatal familial insomnia (FFI), kuru, and variant Creutzfeldt-Jakob disease (vCJD). In animals, TSEs include BSE, scrapie, chronic wasting disease, transmissible mink encephalopathy, and feline spongiform encephalopathy. BSE is unique in that it is the only animal TSE believed to lead to a human TSE, vCJD, through consumption of contaminated ruminant products.

Historical Aspects

The first diagnosis of BSE was reported by the Central Veterinary Laboratory in Weybridge, England, in December 1986.[239] In subsequent years, the number of reported cases rose steadily and the British government initiated a series of escalating preventive measures to combat the epidemic. Incidence peaked in 1992, with 36,680 confirmed cases that year.[58] By 2000, fewer than 1500 cases had been reported, reflecting an ongoing steady decline.[58]

In late 1995 and early 1996, CJD began appearing in Great Britain in a very unusual population—young adults. Further investigation revealed significant differences from previously documented CJD cases in histopathology, EEG waveforms, and MRI brain signal hyperintensities. These cases were named new variant CJD.

Classically, CJD has occurred sporadically without the clustering that occurred with this new variant, which led researchers to search for a new risk factor or cause. Given that the onset of symptoms of CJD after initial exposure can be delayed for up to a decade, the time between confirmation of the first case of BSE and the first case of the new vCJD raised suspicions for a bovine-to-human transmission.[197] Subsequent studies have demonstrated remarkably similar molecular characteristics, histopathology, and clinical features in BSE and vCJD patients.[53,124,201] It is now widely accepted that consumption of BSE-contaminated ruminant products can result in vCJD in humans.

Biology

The infectious agent is believed to be an abnormal isoform of the prion protein (PrP), designated PrP^{Sc}.[52] A prion is an infectious protein that lacks nucleic acids. The human PrP gene is located on the short arm of chromosome 20 and spans 16 kb.[52] PrP^{Sc} is derived, through an aberrant post-translational mechanism, from isoform PrP^{C}, which is found as a membrane-anchored glycoprotein in many cells throughout the body. If the PrP^{C} protein undergoes a conformational change to the partially protease-resistant PrP^{Sc}, accumulations in nervous tissue lead to BSE and vCJD. It is hypothesized that PrP^{Sc} can catalyze transformation of PrP^{C} to more PrP^{Sc}.[227]

Epidemiology

Since the number of BSE cases in cattle in Great Britain reached a peak of 36,680 in 1992, there has been a steady decline to a total of 611 cases in 2003.[64] A total of 778 cases of BSE were reported outside the United Kingdom in 2003, including one case in the United States from a cow that was imported from Canada.[249] To date, 129 cases of vCJD have been reported in Great Britain with an additional 6 in France, 1 in Italy, 1 in Canada, 1 in Ireland, and 1 in the United States. Three of the French cases and both North American cases are believed to have resulted from exposure during time spent in the United Kingdom.[248]

Transmission

Transmission of prions in BSE and vCJD occurs through ingestion of infected ruminant material. The only tissues outside the central nervous system that have been demonstrated to be infectious are the retina, trigeminal and paraspinal ganglia, distal ileum, and bone marrow.[27] This recovered meat was found in hot dogs, sausages, canned meats, and some luncheon meats. There are no reported cases of blood-borne transmission.

Symptoms and Diagnosis

The incubation period for BSE is 2 to 8 years. Cattle affected by BSE may display apprehension, incoordination, aggression, hind-limb ataxia, tremor, difficulty rising, and hyperesthesia to sound and touch.[65] The animal's condition gradually deteriorates and it dies in 2 weeks to 6 months.[65] Diagnosis can be made only by postmortem examination of brain tissue (Fig. 53-45).

Variant Creutzfeldt-Jakob disease is a progressive neurologic disorder, that, unlike traditional CJD, affects a younger population (average age 29 years as opposed to 65 years) and lasts relatively longer (median 14 months as opposed to 4.5 months).[246] Early illness is often accompanied by psychiatric features, including depression and a schizophrenia-like psychosis.[246] Subsequent neurologic symptoms include ataxia, involuntary movements, lack of coordination, and pain.[58] As with BSE, diagnosis can be confirmed only by postmortem histopathologic analysis of the brain.

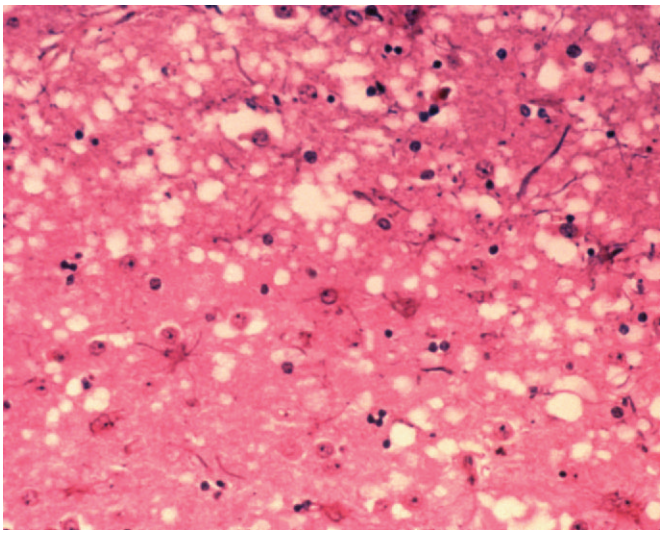

Figure 53-45. Micrograph of brain tissue showing the histopathologic changes in the cytoarchitecture found in bovine spongiform encephalopathy (BSE). The vacuoles in the gray matter give the brain of BSE-affected cows a spongelike appearance in tissue sections. (From CDC Public Health Image Library, available at http://phil.cdc.gov/Phil/quicksearch.asp, and courtesy U.S. Dept. of Agriculture, Animal and Plant Health Inspection Service. Photo by Dr. Al Jenny.)

Treatment and Prevention

There is no treatment for either BSE or vCJD. Prevention is based on public health measures, which have led to a remarkable decline in cases in Great Britain. These measures may include a ban on ruminant-to-ruminant feeding, active surveillance for early detection of BSE, and a ban on importation of cattle and bovine products from countries known to have BSE. The economic implications of an importation ban have made such a measure controversial, although it is nearly universal.

ACKNOWLEDGMENT

We thank Leonard Marcus and Eric L. Weiss for their fine work on previous versions of this chapter.

The references for this chapter can be found on the accompanying DVD-ROM.

54 Rabies

James A. Wilkerson III

Rabies has terrorized humanity since the dawn of civilization, and the menace continues. In industrialized nations where human rabies is rare, animal rabies abounds and humans are protected from infection only by vigorous animal vaccination programs and postexposure immunization. In developing countries, tens of thousands of humans die each year, and over 10 million endure agonizing anxiety after exposure to a possibly rabid animal.[179] In the United States, 15,000 to 40,000 people receive postexposure prophylaxis each year.[145] An encounter with this uniformly fatal infection, globally the most common form of viral encephalitis, leaves "a more indelible stamp of horror" than does any other disease.[113]

CURRENT STATUS

Globally, rabies is the tenth most frequent cause of death from infectious disease.[94] The actual number of deaths is unknown because reporting in developing countries where this infection is common is unreliable. Most of these countries do not have laboratory facilities capable of establishing a dependable diagnosis.[62] The World Health Organization (WHO) currently estimates the number of rabies deaths globally at 40,000 to 70,000 a year, an average of approximately one death every 10 minutes.[190]

Some—perhaps many—human rabies infections are not diagnosed, even in nations with sophisticated medical systems. This problem was vividly dramatized in 2004 by the deaths of four United States organ transplant recipients from a donor whose rabies infection had not been recognized.[57,59] A recent review has suggested that rabies may be underdiagnosed in the United States.[179]

In addition to being undiagnosed, rabies is probably incorrectly diagnosed with considerable frequency. Of 33,000 human rabies deaths reported worldwide in 1997, laboratory confirmation was available for less than 0.5%.[62]

THE RABIES VIRUS

Rabies viruses belong to the group Rhabdoviridae, genus *Lyssavirus*. At least seven genotypes are recognized (see Other Lyssaviruses, later), but genotype 1 is the only one of major significance. This genotype consists of multiple variants, or lineages, each closely linked to a single mammal species such as raccoons, skunks, foxes, mongooses, or various bat species. In the 1980s, these variants were distinguished with monoclonal antibodies. Subsequently, analysis of nucleotide substitutions in the rabies genome by reverse transcriptase–polymerase chain reaction (RT-PCR) has allowed identification of the primary reservoirs for each variant, mapping the geographic distribution of variants, and identification of virus spillover into animals and humans.[62,154] In addition, for the past 25 years, the source of many human infections has been identifiable in the absence of a recognizable exposure to rabid animals.

The rabies virus is bullet-shaped with one flat end, and it contains a single strand of RNA, made up of approximately 12,000 nucleotides, that encodes five proteins (Fig. 54-1). Nucleoprotein (N), phosphoprotein (P, M_1, or NS), and the large polymerase or transcriptase protein (L) make up the core of the virus. The matrix protein (M) and the transmembrane glycoprotein (G) form its coat.[192]

The external surface is studded with perpendicular aggregates of glycoprotein, the G protein, that recognizes cell surface receptors and facilitates virus entry into cells.[14] The 505-amino-acid G protein is composed of a 44-amino-acid internal or "cytoplasmic" portion, a 22-amino-acid hydrophobic transmembrane portion, and the large external "antigenic" portion.[129,191] The complete amino acid sequences of these proteins have been determined for several fixed rabies strains.[189]

A lipid bilayer is closely associated with the matrix protein, and the two form a clearly defined shell for the virus. The M protein is the smallest of the rabies virus structural proteins, with only 202 amino acids,[193] but it makes up approximately 25% of the virion's total protein. It is in close contact with the core and also interacts with the internal segment (cytoplasmic tail) of the surface protein.

The core of the virus forms a tightly structured helix of 30 to 35 coils that extends end to end within the virion. The RNA genome is associated with about 1800 closely packed molecules of the 55-kDa nucleoprotein, which together are known as ribonucleoprotein (RNP). The N protein protects the genome from digestion and keeps it in a suitable configuration for transcription. Some 30 to 60 copies of the large (approximately 190 kDa) transcriptase-associated L protein and about 950 copies of the smaller (approximately 38 kDa) phosphoprotein are responsible for replication of the viral RNA.[193]

RABIES IN THE UNITED STATES

Incidence in Humans

The incidence of human rabies in the United States fell dramatically from 23.5 infections per year in the late 1940s to 1.0 infection per year during the 1980s (Table 54-1). However, 27 infections were reported from 1990 through 1999, and 15 have

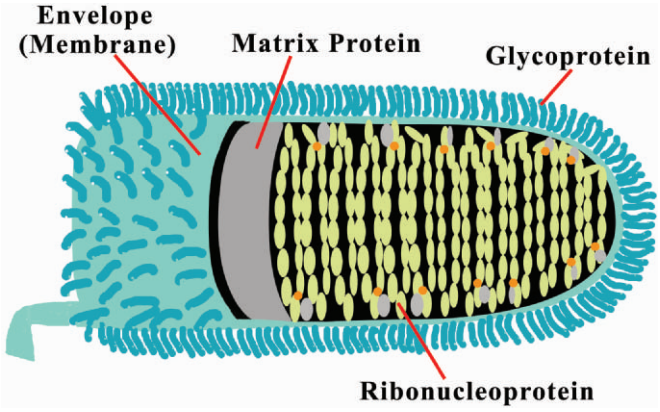

Figure 54-1. Structure of the rabies virus. (Adapted from www.cdc.gov/ncidod/dvrd/rabies/the_virus/virus.htm, accessed February 2005.)

TABLE 54-1. Human Rabies Infections in the United States, 1946 to 2004

TIME PERIOD	U.S. INFECTIONS*	CASES/YR, AVG.	NON-U.S. INFECTIONS†
1946–1949	94	23.5	0
1950–1959	136	13.6	0
1960–1969	38	3.8	3
1970–1979	17	1.7	6
1980–1989	3	0.3	7
1990–1999	22	2.2	5
2000–2004	15	3.0	2

*Rabies infections acquired within the United States.
†Rabies infections acquired outside the United States (not included in Average Cases per Year).

been reported so far in the 2000s, most acquired directly or indirectly from bats.[54–60,184]

Although reliable rabies vaccines and antisera first became available during this 58-year period, extensive vaccination of domestic animals, particularly household pets, and elimination of unrestrained and stray animals are considered primarily responsible for the decline in the human infection rate.[49,50,121] Such programs reduced the incidence of laboratory-confirmed rabies in dogs from 6949 in 1947 to 99 in 2002.[107] The annual cost for these programs is over $300 million, most of which is borne by pet owners.[155]

Rabies in Wild Terrestrial Animals

In the United States and Canada, a vast reservoir of rabies persists in wild animals (Table 54-2).[155] During 2003, 49 states and Puerto Rico reported 7170 rabies infections in animals, 6556 (91.4%) of which were in wild animals. However, some states accept only animals responsible for a human or domestic animal incident for rabies testing; others test all submitted specimens. Furthermore, the number of rabid wild animals that die without being detected is estimated to be more than 90% of the total, so that identified infections represent only a small fraction of wild animal rabies.[107]

TABLE 54-2. Animal Rabies in the United States, Annual Averages 1998–2002

SPECIES	NUMBER OF INFECTED ANIMALS	PERCENTAGE OF TOTAL INFECTED ANIMALS
Raccoons	2962	39.5
Skunks	2257	30.1
Bats	1175	15.7
Foxes	443	5.9
Cats	276	3.7
Dogs	105	1.4
Cattle	106	1.4
Horses and mules	62	0.8
Woodchucks	50	0.7
Bobcats	30	0.4
Sheep and goats	9	0.1
Other wild animals	24	0.3
Other domestic animals	3	<0.1

From Rupprecht CE, Gibbons RV: N Engl J Med 351:2626–2635, 2004.

Several major rabies epizootics are recognized. An epizootic of rabies started in arctic foxes in northern Canada in the late 1940s and early 1950s and swept southward in the middle and late 1950s to involve red foxes in Alberta, Saskatchewan, Manitoba, Ontario, and Quebec. The epizootic crossed the St. Lawrence River in 1961 and involved foxes in upper New York State, although currently it appears to be limited to the adjacent Canadian provinces, which are experiencing a lower incidence of fox rabies.[10,26,108] The epizootic, which moved westward to involve arctic foxes in Alaska and the Northwest Territories,[151] surrounds the North Pole and may cover the largest land area of any observed outbreak.[26]

An outbreak of raccoon rabies started in central Florida in the 1940s and spread at the rate of about 25 miles per year, reaching Georgia in the early 1960s and Alabama and South Carolina in the 1970s. In the late 1970s, a second outbreak appeared on the Virginia–West Virginia border. That epizootic has now spread north to all of New England and crossed into Canada in 1999. It has also spread south to join with the epizootic coming north from Florida in North Carolina.[52,62,63] The second outbreak developed when raccoons were translocated from Florida for restocking for hunters. Although the animal suppliers held legal permits and health certificates, the inclusion of some rabid animals among the more than 3500 translocated raccoons has been documented.[102,188]

More than 50,000 rabies infections in raccoons have been reported in the United States since 1975. The land mass affected by this epidemic is approximately 1 million km^2 (383,000 square miles) and includes the residences of 35% of the United States human population. The raccoon epizootic is considered particularly threatening because many raccoons live in densely populated urban and suburban areas.[62] However, the only known human rabies infection resulting from this epizootic occurred in 2003.[54] The spread of rabies from raccoons to humans appears to have been limited in part because raccoons are large animals and their bites are obvious. To some extent, vaccinated dogs and other pets form a barrier between wild animals and humans. Perhaps of greatest significance is the

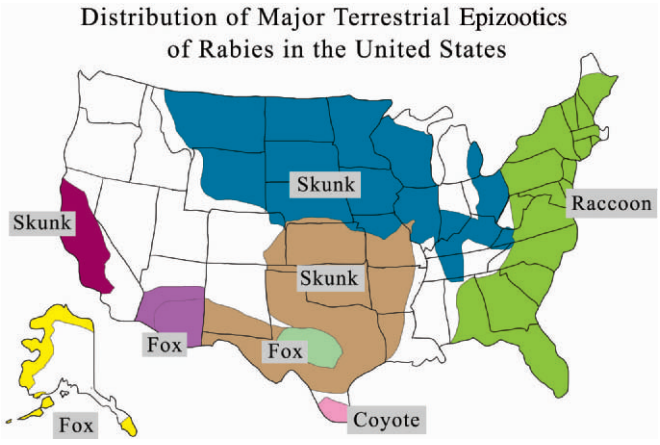

Figure 54-2. Animal rabies epizootics in the United States. (Adapted from www.cdc.gov/ncidod/dvrd/rabies/Epidemiology/Epidemiology.htm, accessed February 2005.)

nonaggressive behavior of rabid raccoons. In 38 rabid raccoon incidents in Florida over a 5-year period, bites were inflicted only when humans or dogs tried to kill or capture raccoons that seemed tame.[185]

Prior to the raccoon epizootic, most terrestrial rabies in the United States was in skunks. Human rabies resulting from exposure to a spotted skunk in California was reported in 1826.[61] Four epizootics are recognized, one of which is in Quebec and New York and is associated with the fox epizootic in that area. A larger epizootic started in Iowa in 1945. It has spread east to Ohio, west to Montana, north to the Canadian provinces of Manitoba (1959), Saskatchewan (1963), and Alberta (1971), and south to meet with a third epizootic that originated in Texas and that spread to surrounding south-central states, particularly Oklahoma and Arkansas. The fourth epizootic in skunks is located in northern California.[61]

An increase in the number of rabid skunks in East Coast states has recently occurred, but analysis of these infections indicates that they result from raccoon rabies spilling over into skunks and are not indicative of a separate skunk epizootic.[91]

Screening of rabies virus isolates from the epizootics has disclosed five distinctive patterns (Fig. 54-2). Red foxes and skunks in New York and adjacent Canada present one pattern; raccoons from the Atlantic states present a second. The skunks in the south-central states present a third, and a fourth is represented by only a small outbreak in gray foxes in Arizona. However, the fifth pattern is found in skunks from the north-central states and from California, in dogs from the Mexico border states, and in a small rabies outbreak in gray foxes in central Texas.[150]

Rabies in rodents is an intriguing problem. Rodents are the animals of choice for rabies virus isolation in the laboratory, yet rabies in small free-living rodents is rare. Rodents may usually be killed rather than just infected by the bites of rabid animals, but rodents are carrion eaters and can be infected by that source. In recent years, the largest number of rodent rabies infections has been in large rodents, such as woodchucks, that have been infected by rabid raccoons. Rabid beavers have attacked and bitten humans in North Carolina. However, no transmission of rabies to humans by rodents has been documented.[163]

Rabies in Bats

With the exception of Antarctica, rabies in bats is global. Bats are the most prominent source of human rabies in Canada and the United States, parts of South America, western Europe, and Australia where rabies in carnivores, particularly dogs, has been controlled.[119]

Rabies was diagnosed in insectivorous bats in Brazil in the 1920s and in frugivorous bats in Trinidad during the 1930s, although the principal subject of these studies was rabies in hematophagous ("vampire") bats that was being transmitted to humans. The first definitive diagnosis of rabies in non-hematophagous bats was made in a frugivorous bat that flew into a "chemist's" shop in Port of Spain, Trinidad, on September 10, 1931. However, the incident that drew widespread attention to bat rabies occurred in Tampa, Florida, on June 23, 1953. The 7-year-old son of a ranch hand was looking for a baseball near some shrubbery when a lactating female yellow bat suddenly flew out of the bushes and bit the boy on the chest, remaining firmly attached until knocked off by the boy's mother. The ranch owner had heard of rabies in vampire bats in Mexico and insisted the bat be examined for infection. Negri bodies were found in smears of the brain, and the diagnosis was confirmed by mouse inoculation of brain tissue. The boy was given postexposure treatment and did not develop an infection.[16,63,189]

The publicity given this event led to many more bats being submitted for rabies examination. Subsequently, rabies has been found in bats in every state except Hawaii, and in eight Canadian provinces.[16,133] In 1989, bat rabies was reported from all 48 of the continental states and the District of Columbia.[50] The estimated incidence of rabies in bats in the United States is 0.5% to 1.0%; the incidence in bats that appear ill or injured is much higher, 7% to 50%.[16,164]

Rabies virus variants from bats are species specific rather than geographically specific,[150] and they are distinctly different from those of terrestrial animals in the same locations, including the major terrestrial epizootics. Clearly, little exchange of infection between bats and terrestrial animals takes place, although occasional animals infected with rabies virus strains typical of bats are found. Many large areas of the United States, particularly the Pacific Northwest and New England (prior to the raccoon epizootic), report rabies in bats but in no other species. Even though cats and foxes catch and eat bats, only 3 of 136 cat and fox rabies isolates over a 2-year period were antigenically similar to bat rabies strains.[16,70,150]

Approximately 70% of human rabies infections and 75% of cryptic rabies deaths in the United States have been caused by the variant associated with silver-haired and eastern pipistrelle bats, which are reclusive animals rarely found around human habitation. (Infections by variants associated with bats that frequent human dwellings are much less common.) Infections in other animals by this variant are also disproportionately very high. These bats are small and their bites difficult to detect. However, in comparison with other rabies virus variants, the variant associated with these two bats replicates better in fibroblasts and epithelial cells, and replicates better at the low temperature of 34°C (93.2°F). These features indicate that this variant is better able to replicate in the peripheral tissues where most bites occur.[114]

Rabies in Domestic Animals

Since rabies in dogs has been controlled, rabies infections in cats have outnumbered infections in dogs (321 to 117 in 2003.)[107]

A major problem in vaccinating cats is establishing ownership. Farmers value cats for rodent control but do not recognize them as property. Cats wander from farm to farm and come in contact with wild animals with rabies. Capturing feral cats so they can be vaccinated is difficult.[37]

Rabies is not rare in other domestic animals, including cattle, horses and mules, and sheep and goats.

Sources of Human Infection

In the late 1940s and 1950s, most human rabies in the United States resulted from bites by dogs or cats. From 1946 to 1961, there were 146 infections for which a source of exposure could be identified, and dogs were responsible for 120 and cats for 9, accounting for 88.4% of the total. Foxes (7), skunks (5), and bats (5) were responsible for the rest.[49] However, when rabies in domestic animals was controlled, human rabies resulting from bites by pets disappeared. Since 1966, all but two of the 15 human rabies infections resulting from exposures to rabid dogs were acquired outside of the United States.[42,44,49,121]

Prior to 1965, the Centers for Disease Control and Prevention (CDC) had recorded no human rabies occurring within the United States that had been acquired outside the country.[121] However, since then, the number of infections acquired outside the United States has been significant: 3 of 15 (20%) between 1965 and 1970, 6 of 23 (26%) in the 1970s, 7 of 10 (70%) in the 1980s, and 7 of the 35 cases (20%) since 1980. Lack of knowledge about the risk of rabies in developing countries has led some travelers to disregard animal encounters and not obtain rabies immunoprophylaxis, but some of these infections have been in children who did not inform their parents of the animal contact.

Until the 1980s, identifying the source of a number of human rabies infections in the United States was impossible. For many infected persons, no animal exposure incident—even an opportunity for animal exposure—could be identified. An infectious source could not be found for 84 (35%) of 230 human rabies infections occurring in the United States between 1946 and 1961,[121] or for 6 (16%) of 38 human infections between 1960 and 1970.[48]

Only since the 1980s has monoclonal antibody typing or RT-PCR nucleotide analysis allowed the source of human rabies infections to be determined when no animal exposure incident could be identified.[16,70,150] However, such studies have made it unmistakably clear that bats are now the major source of human rabies in the United States (Table 54-3 and Fig. 54-3).

Thirty-six of the 40 human rabies infections acquired within the United States since 1980 are attributed to bats. Only four of the individuals had a history of a bite by a bat, and the source of infection for the others was identified by nucleotide analysis.[49,57,59]

How the infection is transmitted remains uncertain. In 1956 and 1959, two men died of rabies after exploring Frio Cave near Uvalde, Texas. The walls and ceiling of that cave hold astonishing numbers of bats—300 to 400 per square foot. Neither had been bitten, and their infections were attributed to aerosol transmission of the rabies virus. When experimental animals of various species subsequently were placed in the cave in cages that allowed the virus to be transmitted only as an aerosol, a significant percentage developed rabies.[65,66] Additionally, aerosol transmission of rabies to humans has occurred at least twice in laboratories.[22,187] The CDC recommends rabies vaccination for spelunkers.[53]

TABLE 54-3. Rabies Contracted within the United States, 1980–2004

PATIENT	AGE (yr)	SEX	ONSET (mo/yr)	LOCATION	SOURCE
1	27	M	6/81	Oklahoma	Skunk
2	5	F	2/83	Michigan	Bat, Ln/Ps
3	20	M	9/84	Pennsylvania	Bat*
4	22	M	5/90	Texas	Bat, Tb
5	Adult	F	8/91	Texas	Dog*
6	Adult	M	8/91	Arkansas	Bat, Ln/Ps
7	Adult	F	10/91	Georgia	Bat, Ln/Ps*
8	11	F	7/93	New York	Bat, Ln/Ps†
9	82	M	11/93	Texas	Bat, Ln/Ps†
10	44	M	1/94	California	Bat, Ln/Ps†
11	41	M	10/94	West Virginia	Bat, Ln/Ps†
12	24	F	11/94	Alabama	Bat, Tb†
13	42	F	11/94	Tennessee	Bat, Ln/Ps†
14	14	M	11/94	Texas	Dog†
15	4	F	3/95	Washington	Bat, Msp
16	13	F	9/95	Connecticut	Bat, Ln/Ps†
17	74	M	8/95	California	Bat, Tb†
18	27	M	9/95	California	Bat, Ln/Ps†
19	42	F	9/96	Kentucky	Bat, Ln/Ps†
20	49	M	12/96	Montana	Bat, Ln/Ps†
21	65	M	12/96	Montana	Bat, Ln/Ps†
22	64	M	12/96	Washington	Bat, Ef†
23	71	M	10/97	Texas	Bat, Ln/Ps†
24	32	M	10/97	New Jersey	Bat, Ln/Ps†
25	29	M	12/98	Virginia	Bat, Ln/Ps†
26	49	M	9/00	California	Bat, Tb†
27	26	M	10/00	Georgia	Bat, Tb†
28	47	M	10/00	Minnesota	Bat, Ln/Ps†
29	69	M	11/00	Wisconsin	Bat, Ln/Ps†
30	28	M	3/02	California	Bat, Tb†
31	13	M	8/02	Tennessee	Bat, Ln/Ps†
32	20	M	9/02	Iowa	Bat, Ln/Ps†
33	25	M	3/03	Virginia	Raccoon†
34	66	M	9/03	California	Bat, Ln/Ps†
35	Adult	M	5/04	Texas	Bat, Msp
36	Adult	M	5/04	Texas	Bat/human‡
37	Adult	F	5/04	Texas	Bat/human‡
38	Adult	M	5/04	Texas	Bat/human‡
39	Adult	?	5/04	Texas	Bat/human‡
40	15	F	11/04	Wisconsin	Bat, Msp§

*No documented exposure; infection identified by monoclonal antibodies.
†No documented exposure; infection identified by reverse transcriptase–polymerase chain reaction (RT-PCR).
‡Infection acquired through organ transplant from individual 35.
§Patient survived.
Bat species: Ln/Ps, *Lasionycteris noctivagans* or *Pipistrellus subflavus*, silver-haired bat or the eastern pipistrelle bat; Tb, *Tadarida brasiliensis*, Brazilian (Mexican) free-tailed bat; Ef, *Eptesicus fuscus*, big brown bat; Msp, species unknown.
From references 54–59, 184.

However, nursery caves such as Frio Cave contain an astounding number of bats. Saliva and urine constantly rain down on anyone entering the cave, and the blanket of guano on the floor ranges from several inches to several feet in thickness. In Frio Cave, air circulation is so poor the bats warm the cave, the air is humidified by their respiration, and the concentration of ammonia from their urine is so high that the cave usually cannot be entered without respirators.[66] Similar infections in other caves have not been reported.

Unrecognized bites appear to be the source of infection for most individuals who have had no recognized bat encounters. Bat teeth are so small and sharp that a bite may not be felt. Even recognized bites do not appear to be particularly painful, although at least one of the individuals known to have been bitten was intoxicated with ethanol at the time.[87] For centuries, South American vampire bats have been reported to bite sleeping victims without awakening them.

Limiting human rabies of bat origin is best addressed by informing the public of the risk.[133] Reducing the bat population is not an acceptable approach. Significant population reduction would be difficult, but if achieved, it would be an ecologic disaster because bats play such a major role in insect control.

The CDC and other institutions now advocate the following measures:

- Dwellings should be "bat-proofed" by tightly covering all possible entrances, particularly roof ventilation openings, with wire screens. Protection from bats in unscreened dwellings or when sleeping outdoors can be achieved with mosquito netting.
- Contact with bats must be assiduously avoided, particularly bats that are behaving unusually. Bats are nocturnal and any activity during daylight hours should be considered abnormal. Diseased bats are often unable to fly.
- Any person who has contact with a bat, regardless of whether a bite is thought to have been inflicted, should receive postexposure prophylactic therapy unless the bat has been caught, examined, and found to be free of rabies.
- Any person, particularly a child, who awakens from sleep and finds a bat in the room should receive postexposure prophylaxis unless the bat has been caught and examined. A number of the recent bat rabies victims have been children who experienced this kind of exposure.

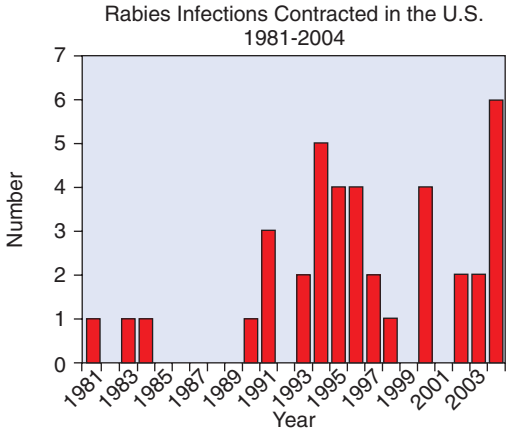

Figure 54-3. Yearly incidence of human rabies acquired within the United States from 1981 to 2004.

RABIES IN OTHER COUNTRIES

Rabies is found throughout the world, and although it is more common in tropical or temperate climates, it is by no means limited to those areas (Fig. 54-4). Arctic foxes with rabies have been found in Alaska, northern Canada, Greenland, Norway's Svalbard Islands, and much of Siberia. An epizootic in the Thule

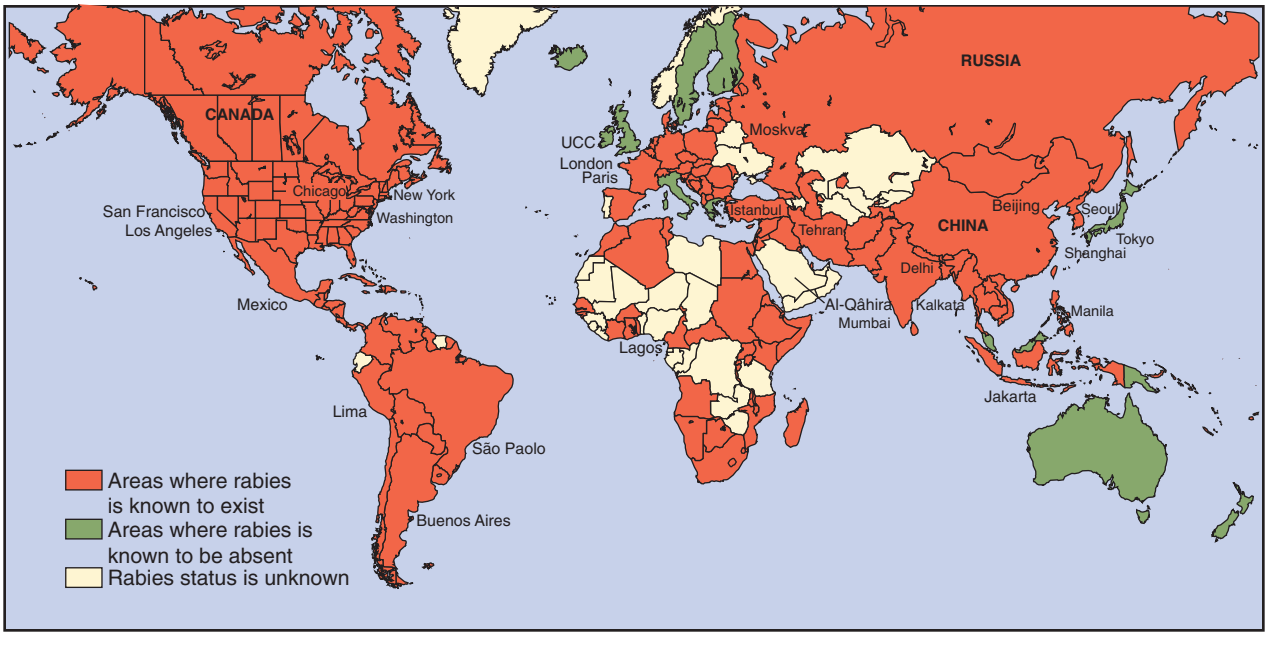

Figure 54-4. Areas of the world in which rabies is present *(orange)*, areas in which rabies is absent *(green)*, and areas for which information is not available *(yellow)*. (Adapted from www.nathnac.org/pro/factsheets/rabies.htm, and from www.ktl.fi/portal/suomi/julkaisut/oppaat_ja_kirjat/matkailijan_terveysopas_old/4._matkailijoiden_rokotukset/l415, both sites accessed August 2005.)

district of Greenland in 1958 and 1959 resulted in the death from rabies of 50% of the sled dogs in that area. Over 1000 dogs in the Egedesminde district died in another epizootic in 1959 and 1960. For reasons that are not known, transmission of rabies to humans is rare in these areas, even though exposures are common.[67] Perhaps the rabies variant is more infective for foxes and dogs.

Rabies is not found in a few areas, most of which are landmasses or peninsulas isolated by water. Rabies does not occur in Hawaii (the only state in the United States in which rabies is not found), some of the Caribbean islands, Pacific Oceania (including Australia, although Australian bat Lyssavirus is found there, and New Zealand), or Antartica.[89,118] The two human genotype 1 rabies infections that have occurred in Australia are thought to have been acquired outside that country.[22,47,89]

Great Britain had been free of rabies since an extensive dog confinement and vaccination program in 1903, although concern about reintroduction of rabies has been raised by the Channel tunnel and reduction of border controls between members of the European Community.[63,165] A 55-year-old Scotsman with a fatal infection in 2002 was the first locally acquired Lyssavirus infection on that island in 100 years, but the virus was not of genotype 1.[85]

Rabies has been endemic in Japan since the 10th century. However, after World War II, members of the U.S. Army Veterinary Corps determined that no reservoir of rabies existed in the wild animal populations of Japan, Taiwan, and the Philippines, perhaps in part because wild animals were hunted for food during the war. Extensive campaigns to eliminate stray dogs (which in some areas of Japan reduced the canine population by 70% to 80%) and to vaccinate the remaining dogs succeeded in eradicating the infection from Japan and Taiwan. Endogenously acquired rabies has not occurred in those islands since the late 1950s.[4,156] The red fox oral rabies vaccination program has succeeded in eliminating terrestrial rabies from a number of European countries not surrounded by water: Finland and the Netherlands in 1991, Italy in 1997, Switzerland in 1998, France in 2000, Belgium and Luxembourg in 2001, and the Czech Republic in 2004. (Bats in those countries are infected with lyssaviruses of genotypes 5 and 6, and those infections have not been controlled.)[180a]

The success of canine rabies eradication programs depends on the society in which the programs are initiated. Such programs achieve little success in nations that are predominantly Hindu or Buddhist, because the people do not support elimination of animals that have no apparent owner. They often put out food for stray dogs. In contrast, Malaysia, a peninsula that is predominantly Islamic, has been largely free of rabies since the early 1950s.[23]

Elimination of stray dogs must be combined with vaccination programs. Dogs that are eliminated because they cannot be associated with human ownership are quickly replaced. The annual turnover of the dog population in developing countries has been found to range between 30% and 40%.[27]

Vaccination of domestic animals for rabies is limited largely to industrialized nations. In many developing countries, vaccination of animals is considered unaffordable and rabies control resources are expended on postexposure immunoprophylaxis of humans. Even though rabies immunoprophylaxis is administered to 800 to 900 persons per million inhabitants annually in such countries, the human death rate from rabies is still high—an average of nearly five deaths for each 1 million population annually.[28] In the United States, that death rate would result in nearly 1500 rabies deaths a year.

The magnitude of the rabies threat in developing countries is illustrated by the experience in Thailand. Rabies has been of particular interest in that country since Princess Banlusirisarn was bitten by a rabid dog within the palace grounds near Bangkok in 1911 and subsequently died. No rabies vaccine was available in Thailand, then called Siam, at that time. The princess's death was instrumental in establishing the Institute Pasteur of Bangkok in 1913, which was renamed the Queen Saovabha Memorial Institute (QSMI) in 1922. This institute, a WHO Collaborating Center for Research on Rabies Pathogenesis and Prevention, has been and remains the site of many sophisticated rabies investigations.

In the 1990s, the QSMI's postexposure rabies clinic treated about 18,000 patients with new animal bites each year, an average of almost 50 new patients a day. These patients were only 28% of the estimated 64,000 Thais who receive postexposure therapy annually, many of whom are residents of rural or remote portions of the country and are treated by local physicians.[182] The number of human deaths from rabies in Thailand has declined from about 400 a year in the 1970s to 70 in 1999, even though dog rabies has not been controlled.[62]

Other developing nations have similar rabies problems. WHO agencies have estimated that 87 countries and territories with a total population of about 2.4 billion people are afflicted with endemic canine rabies.[28]

SOURCES OF HUMAN INFECTION

Although domestic animals are rarely the sources of human rabies in the United States and other developed countries, in developing countries the vast majority of human rabies—99% by some estimates—is the result of exposure to rabid dogs.[29,63,175,189] In Thailand, although rabies has been found in an array of exotic tropical animals that includes tigers and leopards, between 1979 and 1985, 90.6% of human infections resulted from dog bites and an additional 6% followed unknown events. The remaining 3.6% followed cat attacks.[182]

Other animals, particularly bats, transmit rabies. Hematophagous (vampire) bats are a major source of animal and human rabies in South and Central America, the only areas where such bats are found. Their range extends between northern Mexico and northern Argentina, basically between the tropics of Cancer and Capricorn, and fossils indicate that vampire bats have inhabited those areas for 2.5 million years.[84] These animals consume 20 to 25 mL of blood at a feeding, and although cattle are their preferred food source, a study in Colima, Mexico, found human blood in the stomachs of 15.7% of 70 vampire bats.[12]

Human rabies of vampire bat origin was first recognized in 1929 in Trinidad, when Negri bodies were found in the brains of 17 individuals, mostly school-age children, who died with acute ascending paralysis. Subsequently, small epidemics have been recognized almost every year in that country. Interestingly, almost all of the rabies transmitted by vampire bats in Latin America is the paralytic rather than furious type.[178]

Human rabies resulting from vampire bat bites has been reported almost every year from Mexico, but it was first

reported from South America in 1953 when nine of 43 diamond miners who slept outdoors died of a mysterious illness. Autopsies of five of the miners disclosed rabies. In an outbreak in two rural communities in the Amazon Jungle of Peru during the first 4 months of 1990, 29 (4.6%) of 636 residents died with a rapidly progressive illness characterized by hydrophobia, fever, and headache. Rabies virus was isolated from the brain of the only individual on whom necropsy was possible. Ninety-six percent of the victims had a history of bat bites, although bats also had bitten 22% of unaffected community members.[5]

Human infection is not the only major problem resulting from rabies transmitted by vampire bats in Central and South America. Currently, migrating epizootics of vampire bat–transmitted bovine paralytic rabies annually kill thousands of animals; the estimated cost in 1980 was $500 million.[5,62] Efforts to control these epizootics have included vaccination of cattle, and attempts to limit the vampire bat population by administering anticoagulants, usually warfarin.

Meat is often consumed from cattle slaughtered at the first, virtually pathognomonic signs of disease, paralysis of the hindquarters. Even normal-appearing animals may have infected brains. Four (0.4%) of 1000 apparently healthy cattle selected at random at the Mexico City slaughterhouse of Ferrería were found to be infected by rabies when investigated with fluorescent antibody staining and animal inoculation of brain tissue. However, no cases of human rabies from this source have been reported.[12]

Mongooses are the major source of rabies in South Africa and in some Caribbean islands.[13,76] The yellow mongoose is the main reservoir of rabies in South Africa.[189] The small Indian mongoose, imported many years ago in an effort to control rodents,[95] is an important reservoir and vector of rabies in Cuba, the Dominican Republic, Grenada, Haiti, and Puerto Rico.[189] In Grenada from 1968 to 1984, mongooses accounted for 787 (73%) of 1078 cases of animal rabies on the island. Of 208 human exposures requiring antirabies therapy, mongooses were responsible for 119 (57%). The possibility of eliminating the animals by hunting or trapping appears remote in view of the island's topography and the animal's skill at adapting to its surroundings. However, recently reported studies have found that mongooses will take oral baits, so oral vaccination appears feasible if an appropriate vaccine can be identified.[111] Interestingly, 20% to 40% of the mongooses have naturally acquired antirabies antibodies, possibly from having survived infection.[73]

Investigators in Nepal found 51 travelers who required immunoprophylaxis after rabies exposure during a 2-year period. Thirty-six of these encounters were with dogs, but 10 were with monkeys at Swayambunath, the "Monkey Temple," a Hindu shrine popular with tourists. The bites were inflicted when monkeys leapt for food carried by visitors.[148]

Human-to-human transmission of rabies is rare. Eight documented cases were in individuals who received corneal transplants (two from the same person) from individuals whose neuroparalytic disorder was not recognized as rabies.[41,83,98] In 1996, Fekadu and his colleagues reported two apparent instances of human-to-human rabies transmission in Ethiopia. A 41-year-old woman, who died of rabies 33 days after her 5-year-old son died of the same infection, had been bitten on a finger by her son. A 5-year-old boy, who developed rabies 33 days after his mother died of that infection, had been repeatedly kissed on his mouth by his mother, apparently passing infected saliva to him.[81]

In July 2004, the CDC reported four cases of human rabies transmitted by organ transplants from a single donor. The male donor was hospitalized in Texas with "severe mental status changes," low-grade fever, and neurologic imaging findings indicative of a subarachnoid hemorrhage. That lesion expanded rapidly in the 48 hours after admission and led to cerebral herniation and death. An autopsy was not performed. Only after the organ recipients' deaths from rabies was the donor's history of having been bitten by a bat discovered.

The lungs were transplanted to a man who died of intraoperative complications. The liver and one kidney were transplanted to men and one kidney was transplanted to a woman, all of whom died of rabies 27, 37, and 39 days later. The fourth victim had a liver transplant from another donor, but a segment of iliac artery from the first (rabid) donor was inserted during the procedure. This recipient died of rabies approximately a month after the transplant.[57,59] Only a few months later, in Germany, organs or tissues were transplanted into six individuals from a woman whose cause of death was not diagnosed as rabies until after three recipients died with rabies encephalitis. The other three recipients, one of whom had received a liver transplant and two of whom had received corneal transplants, were immediately given postexposure therapy and the corneal transplants were removed. None of the three developed rabies. Subsequently the woman was found to have been scratched by a dog while visiting India and the diagnosis was confirmed in tissues removed at autopsy that initially did not determine her cause of death.[85a]

One case of human rabies appears attributable to transplacental infection. However, a number of mothers dying of rabies encephalitis have given birth to healthy babies, presumably because the virus travels through nerves and cannot reach the placenta or fetus.[83,98] Viremia has never been documented.

FEATURES OF HUMAN RABIES

Mortality

Rabies in humans, once it has become clinically apparent, is uniformly fatal. No other infection is so lethal or progresses so rapidly. In the 1970s, intensive support allowed three humans with clinical rabies to survive.[40,93,128] Three rabies survivors have been reported subsequently. The first five infections were vaccination failures, and at least three of the five survivors had severe residual neurologic deficits, severe enough to be fatal 34 months later in one person.[1,112] The rabies virus was not cultured from any of these patients and at least one may have had a reaction to neurally derived vaccine rather than an actual infection.[101]

In October 2004, a 15-year-old girl in Wisconsin became the first human to survive clinical rabies without vaccination. This young lady, who developed symptoms 1 month after she failed to report a recognized bite by a bat, was admitted to the Medical College of Wisconsin, Milwaukee, 5 days later. On the second hospital day, the CDC confirmed the presence of rabies virus–specific antibody in serum and cerebrospinal fluid (CSF). Because she had evidence of an adequate immune response, because brain pathology in humans succumbing to rabies largely reflects secondary complications rather than a clear primary process, and because clinical reports have included the hypothesis that death results from "neurotransmitter imbalance" and autonomic failure, her physicians—with her parents'

approval—elected to treat her with antiexcitatory and antiviral drugs. Rabies vaccine and rabies immune globulin were not administered.

Coma was induced with ketamine and midazolam, she was intubated and maintained on a ventilator, and she received intravenous ribavirin and amantadine. (Ketamine is a dissociative anesthetic, but is also an *N*-methyl-D-aspartate (NMDA) antagonist, and the NMDA receptor has been speculated to be one of the rabies virus receptors.) Later, she also was given benzodiazepines and supplemental barbiturates. She recovered slowly, was removed from isolation on day 31, and was discharged from the hospital on day 76. Attempts to isolate the rabies virus, detect viral antigens, or identify rabies RNA in two skin biopsies and nine salivary specimens were uniformly unsuccessful. Five months after her initial hospitalization, she was alert and communicative, but she had choreoathetosis, dysarthria, and an unsteady gait.[58,186]

No other person who has not been vaccinated has survived clinically evident rabies. Subclinical human infections may occur. The clinical phase of rabies encephalitis rarely lasts more than a few days to a few weeks, and infected persons are severely incapacitated.[2,83] The catastrophe of rabies is compounded by the young age of many of its victims: 40% to 50% are 15 years old or younger.[83]

Incubation Period

Some human rabies infections have been thought to follow prolonged incubation periods. In 1987, a 13-year-old boy who had emigrated from the Philippine Islands 6 years earlier died with rabies determined by RT-PCR to be of Philippine dog origin. He had not been out of the United States since he arrived.[46] The first documented case of rabies reported in Australia, in a 10-year-old boy who died in 1987, probably resulted from a monkey bite inflicted in northern India 16 months earlier.[47] The second documented Australian patient with rabies, a 10-year-old girl of Vietnamese origin, had experienced no identifiable animal contact since she left North Vietnam 6 years and 4 months earlier, and the virus responsible for her death was of an immunotype found in Southeast Asia.[22,89] Joshi and Regmi reported an individual who had an apparent incubation period of 1100 days (3 years).[105] An 18-year-old Mexican man who died in Oregon in 1989 was infected with rabies virus of a strain to which he could not have been exposed for at least 10 months, although no history of any type of exposure could be obtained.[48] Even longer incubation periods of 10 and $19^1/_2$ years have been reported, but these occurred in areas where rabies is endemic, and a second exposure in the intervening period could not be ruled out.[153]

Confirmation of such prolonged incubation periods was achieved in three immigrants into the United States from the Philippines, Laos, and Mexico. Nucleotide analysis disclosed rabies viral amino acid compositions essentially identical to the patterns from rabies viruses isolated from dogs in their native countries, and unlike rabies viruses found in the United States. These individuals had been in the United States for 6 years, 4 years, and 11 months before the onset of clinical disease.[153,155] Such prolonged incubation periods may explain some patients' inability to recall any animal exposure.

Almost 99% of human rabies infections are clinically manifest in less than 1 year, typically 2 to 12 weeks.[14,22,83] The median incubation period in the United States for persons diagnosed between 1960 and 1990 was 24 days for those 15 years old and younger, but $43^1/_2$ days in people older than 15. Fixed (laboratory) strains of virus tend to produce a shorter incubation period than wild or "street" strains. In 1960 in Brazil, 60 people were injected with vaccine that had been inadequately inactivated. Sixteen developed rabies and the incubation periods ranged from 4 to 16 days.[83]

The size of the viral inoculum clearly influences the incubation period. Experimental animals injected with large numbers of viruses develop clinical infections significantly faster than do those receiving small inocula. However, smaller inocula result in greater CNS histologic damage and more widespread infection outside the CNS, particularly in salivary glands.[77]

Pathogenesis of Central Nervous System Infection

Immediately after a bite or investigational injection, rabies virus can be identified at the site with fluorescent antibodies, and it remains near the wound or injection site for hours to weeks, depending on the animal species. Viral antigen can be demonstrated in muscle, and viral particles budding into the sarcoplasmic reticulum and from the sarcolemma have been demonstrated by electron microscopy.[61] The virus appears to enter both motor and sensory nerves, probably through motor endplates and neuromuscular spindles.[11,143]

Passage of the virus through peripheral nerves was demonstrated in 1887, when rats[72] and rabbits[71] were protected from rabies following injections of the virus in their hind legs by sectioning the sciatic nerve. After entry into peripheral nerves, the virus travels at a rate of about 5 to 10 mm per hour to neuronal cell bodies such as dorsal root ganglia.[14,61] Replication can begin at this site, and prolonged ensconcement at this site has been suggested as one explanation for prolonged incubation periods.[143]

On reaching the CNS, the virus is widely disseminated with extreme rapidity, almost simultaneously with entry, but the manner in which the virus disseminates throughout the CNS is not known. Viremia has not been documented. Plasma membrane budding from infected to uninfected neurons, or dissemination through intercellular spaces or CSF, has been suggested. Clusters of viral particles at neuromuscular junctions, the reduction of viral infectivity by nicotinic acetylcholine receptor competitors, and other data suggest that the virus recognizes cholinergic binding sites and perhaps enters peripheral and central nerve fibers through those sites. The large numbers of muscle cholinergic binding sites in foxes, which are exquisitely sensitive to rabies, and the small number of such sites in opossums, which are highly resistant to rabies, support this hypothesis, which may explain the mechanism of sensitivity or resistance.[14] The glycoprotein that coats the viral particle is a major determinant of neuroinvasiveness, and alteration of this protein can markedly alter the kinetics of CNS viral spread.[11]

Viruses can be isolated from CSF—a significant antibody concentration in this fluid is considered diagnostic of CNS rabies infection—and spread by this route could be quite fast.[147] Additionally, rabies virions have been identified in intercellular spaces in the CNS by electron microscopy. Rabies antigen can be found in essentially all parts of the CNS and, although limited mostly to neurons, can also be found in oligodendrocytes.[99]

The rabies virus can infect a wide variety of cells in culture; no explanation for its localization to neurons in vivo has been found.[106]

After wide CNS involvement, the virus passes centrifugally through neural axoplasm to a wide variety of tissues, including salivary glands, corneas, and skin of the head and neck, sites at which identification of the virus may aid the diagnosis of clinical illnesses. The route of spread to the periphery was demonstrated over 90 years ago, when Bartarelli sectioned nerves to salivary glands and found that the glands themselves did not contain rabies virus.[21] Even within the salivary gland, the virus appears to spread by neural networks and not between adjacent epithelial cells.[61]

An element of immunopathology is produced by disseminated rabies infection. Among persons exposed to rabies, those immunized with early vaccines who subsequently developed infections did so more rapidly than unvaccinated individuals, a phenomenon termed early death. Experimental confirmation of this feature has been achieved by injecting mice with a lethal quantity of rabies virus and immunosuppressing a portion of them. The immunosuppressed animals survived 20% to 25% longer than unsuppressed animals, but their survivals were shortened to those of the control animals when they were injected with antirabies antibody. Additionally, cytolytic T cells appear to be a significant component of the protective response to rabies virus. Avirulent strains of rabies virus induce rabies-specific cytolytic T cells, but virulent strains do not.[116]

Pathologic alterations in the CNS infected by rabies are surprisingly mild, unless supportive care has kept the patient alive for several weeks, which allows much more extensive, necrotic lesions to develop.[133,143] Leptomeningeal congestion is the only grossly visible change typically found. Mild edema may be present if the patient has been hypoxic. Pressure grooves are rare. The meninges may be cloudy if severely inflamed.[124]

Typical histologic features are perivascular cuffing by mononuclear inflammatory cells, microscopic collections of reactive glial cells known as Babes nodules (following his description in 1892),[6] and areas of neuronal degeneration and neuronophagia. Some leptomeningeal inflammation is usually present. Very recently, spongiform degeneration similar to that found in prion diseases has been described in animals, particularly in skunks.[61]

Van Gehuchten and Nelis described a striking proliferation of the capsular cells surrounding ganglionic neurons that pushed these cells apart and separated them by a dense cellular layer. The neurons also contained degenerative changes. Subsequent studies have found the Van Gehuchten–Nelis changes to be present in almost everyone dying of rabies, and to be a much more consistent and reliable diagnostic feature than viral inclusions.[166,167]

Negri bodies are the best known histologic feature of rabies and were the first viral inclusions to be found (Fig. 54-5). Negri described these cytoplasmic inclusions in 1903 and considered them parasites that caused the disease.[117] Entirely independently, Bosc described identical inclusions in two separate papers published the same year, but he is rarely recognized.[30,31] Negri bodies are eosinophilic cytoplasmic inclusions that contain a small, basophilic inner body or *innerkörperchen*. Inclusions are found almost entirely within neurons and are most common in Ammon's horn and Purkinje cells of the cerebellum, but they may be seen in any part of the CNS, particularly in humans. The bodies may be seen in other tissues, such as salivary gland, skin, cornea, and pancreas, but they are not seen in the ependyma and choroid plexus.

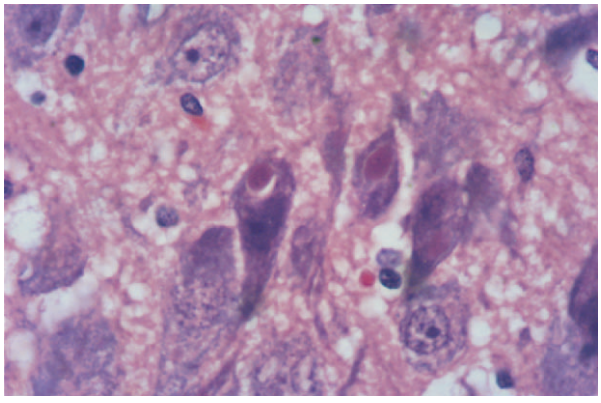

Figure 54-5. Negri bodies in the cytoplasm of neurons in the hippocampus of a dog. These inclusion bodies show some internal structure. (Hematoxylin and eosin, ×1000.) (Courtesy Merieux Institute, Inc.)

The appearance of the inclusion bodies varies in different animal species, and uninfected animals such as cats commonly contain cytoplasmic inclusions that easily could be confused with rabies bodies.[159]

By electron microscopy, three types of bodies have been identified, one composed of a granular matrix of viral protein and typical virus particles, a second composed of matrix and tubular structures, and a third composed of matrix alone. Invaginations of cytoplasm into the inclusion give rise to the *innerkörperchen*.[124]

Clinical Features

"Although the clinical features of classical rabies are said to be too well known to require description, few clinicians practicing outside the tropical endemic zone have ever seen the disease, and the few cases presenting in Europe and North America are often misdiagnosed."[178]

Pain, paresthesias, or symptoms such as burning, itching, or numbness at the site of the bite or in the limb are the most common early symptoms. Paresthesias may result from proliferation of rabies virus in the spinal cord at the level at which the nerves enter from the bite site.[83] In Thailand, this initial symptom often takes the form of severe itching that can lead to frenzied scratching and extensive excoriations.[178] This symptom is so well known among the Thai people, and animal bites are so common in that country, that any cause of itching or dysesthesia, even contact dermatitis, can lead to anxiety months to years after an animal exposure.[182]

Local symptoms may not appear at all. Systemic symptoms usually develop later and are largely nonspecific. One third or fewer of all patients initially have symptoms that suggest the etiology of their infection to physicians who do not commonly encounter the disease. Complaints may be constitutional: malaise, chills, fever, or fatigue. Symptoms that suggest an upper respiratory infection are common and include sore throat, cough, and dyspnea. Gastrointestinal symptoms include anorexia, dysphagia, nausea and vomiting, abdominal pain, and diarrhea. Headache, vertigo, irritability, or anxiety and apprehension suggest CNS involvement. However, even advanced rabies often has nonspecific features.[49,121] Hypoventilation and hypoxia are common during the prodrome and early acute

neurologic phase, and the cause is not understood. Cardiac involvement is common and is manifested by tachycardia out of proportion to the fever; hypotension, congestive failure, or cardiac arrest may ensue.[83]

Two clinical forms of human rabies are recognized. The furious, encephalitic, or agitated form that is associated with periodic episodes of hyperactivity, restlessness, or agitation is considered more typical. This form of rabies is characterized by periodic opisthotonic spasms or convulsions, particularly in response to tactile, auditory, visual, or olfactory stimuli (aerophobia and hydrophobia). Episodes of disorientation, sometimes with hallucinations or violent behavior, often alternate with periods of lucidity, which can be particularly horrifying because the patient recognizes the nature of the illness. The terror associated with hydrophobia has been labeled powerful but indescribable. Episodes of priapism, increased libido, insomnia, nightmares, and depression may suggest a psychiatric disorder. Patients maintained with supportive therapy progressively deteriorate, become comatose, lose peripheral nerve function, lose brainstem function, and die.[83,178]

Hydrophobia has been described in only 32% of recent patients in the United States,[83] although one recent U.S. rabies victim, an 11-year-old boy, was so afraid of water he would not even take a bath.[51] Experienced observers in Thailand have described hydrophobia as a violent, jerky contraction of the diaphragm and accessory muscles of inspiration that is triggered by attempts to swallow liquids and by other stimuli. Usually, it is not associated with neck or throat pain or with laryngopharyngeal spasms. It has been likened to respiratory myoclonus (Leeuwenhoek's disease). When patients lapse into coma, hydrophobia is typically converted into cluster breathing with long apneic periods.[178]

The variability of the clinical manifestations of rabies may result from heterogeneity in wild or street rabies viral populations (as contrasted with fixed viral strains maintained in laboratories), even in the viruses infecting a single animal. Rabies viruses isolated from a boy who died after being bitten by a fox and propagated by intracerebral inoculation of white mice produced three distinctly different forms of disease in the mice.[90]

The second clinical form of rabies, the paralytic, dumb, or Guillain-Barré–like form, is characterized by progressive paralysis without an initial furious phase. Even though the paralytic form of the disease does not appear to be as familiar to some health care providers, 20% to 30% of human rabies victims present in this manner.[2,182] Other animals also have furious or dumb rabies presentations.[119] Paralytic rabies is more common after rabid vampire bat bites, in persons injected with fixed virus strains, and in persons who have received postexposure vaccination.[83] Distinction from Guillain-Barré syndrome may be difficult, although individuals with that disorder usually do not have urinary incontinence, which is common in rabies-infected persons.[100]

Individual case reports make clear that every patient is different. The most common misdiagnoses are psychiatric or laryngopharyngeal disorders.[100] Physicians at QSMI, who have a vast clinical experience, consider inspiratory spasms to be the most reliable clinical sign of rabies, particularly in comatose patients, regardless of whether the disease was initially furious or paralytic. Such respirations also have been described as rapid, irregular, or jerky-apneustic.

In addition, most of the QSMI patients have myoedema, particularly in the region of the chest, deltoid muscles, and thighs.[182,189] However, this phenomenon, a brief, unpropagated, localized muscle contraction that appears in response to percussion with a tendon hammer, has not been confirmed as an important sign of paralytic rabies.[100]

Because the signs and symptoms are so nonspecific and are often rather mild at onset, many patients with rabies are not hospitalized the first time they are seen by a physician. One patient who died in the United States between 1960 and 1980 was never hospitalized. When admitted, most have a fever, which may be mild, but commonly is above 103°F (39.4°C). Of the 38 individuals who died of rabies in the United States between 1960 and 1980, 24 (63%) had difficulty swallowing, but only half of those had definite hydrophobia or aerophobia. Twenty-seven (72%) manifested excitement or agitation, 24 (63%) had paralysis or weakness, and 12 (32%) had hypersalivation. Dysesthesias at the exposure site were described by 19 (79%) of the 24 individuals who had an identifiable bite or similar exposure.[2]

Twenty-six of the 28 patients diagnosed before death, but only four of eight patients diagnosed after death, had a history of an animal exposure. All 12 patients with hydrophobia were diagnosed before death.[2]

The duration of illness for patients not given supportive care averages 7.3 days, and ranges from 2 to 23 days. For patients who are given supportive care, the average duration of illness is 25.3 days, and the range is 7 to 133 days.[2,159]

"The following [therapeutic] measures have . . . been tried in clinical rabies, but without any evidence of effectiveness: administration of vidarabine; multisite intradermal vaccination with cell-culture vaccine; administration of α-interferon and rabies immunoglobulin by intravenous as well as intrathecal routes; and administration of anti-thymocyte globulin, high doses of steroids, inosine pranobex, ribavirin and the antibody-binding fragment of immunoglobulin G."[189] In a recent review of the management of rabies in humans, a combination of some specific therapies was suggested. The authors point out that essentially no individuals with clinical rabies survive, and the best therapy often is palliative.[101]

Subclinical Rabies

Although clinical rabies in humans is a uniformly lethal infection with only six recognized exceptions, subclinical infections probably occur. Low titers of rabies virus–neutralizing antibodies have been found in Canadian Inuit hunters and their wives, and in unimmunized students and faculty members of a veterinary medical school.[100] In Nigeria, 28.6% of 158 healthy humans, who had no history of exposure to rabies or of any antirabies prophylaxis, were found to have serum neutralizing antibodies against rabies. Antibodies against Mokola virus (genotype 3) and Lagos bat virus (genotype 2) also were found in 7.5% and 2.5% of these individuals. The investigators suggested that these individuals had been infected but the infection had been halted before the virus had entered nerves. An attenuated strain of rabies virus has also been suggested as the cause of such antibodies, but an as yet unidentified virus of the Lyssa group, or even some other cross-reacting infectious agent, could be responsible.[120] Among 48 family members of Peruvians who died following rabid vampire bat bites in 1990, seven had antirabies antibody levels that ranged from 0.14 to 0.66 International Units (IU). These antibodies could not be related to exposure to bats or other animals, or to other epidemiologic events.[5]

Some animals, including dogs, have significant amounts of nonspecific virus-neutralizing antibodies in their sera. Up to 20% of raccoons in Virginia and Florida[146,188] and 20% to 40% of mongooses on Grenada have antirabies antibodies, which may be evidence of nonfatal infections.[73] Up to 80% of bats in crowded nurseries may have rabies antibodies.[16] The only finding considered diagnostic of prior rabies infection is antibodies in the CSF.[78,79]

Even clinical rabies is not always fatal in animals. Pasteur observed that some dogs recovered from early symptoms of rabies and subsequently could not be infected by rabies virus injections.[123] Recoveries from infections that produced paralysis have been described in two dogs[80]; recovery from clinical rabies has also been described in mice, donkeys, bats,[12] and pigs.[15]

Undiagnosed Rabies

Clearly, many rabies infections are not diagnosed in developing countries. The possibility that in industrialized countries a number of human rabies infections are not diagnosed and the true incidence of this infection is higher than reported is suggested by several considerations. The clinical manifestations usually are nonspecific, and many infections are diagnosed late in the course of the illness as the result of testing for any identifiable cause of encephalitis, or subsequently by autopsy examination of the CNS. Eight (21%) of the 38 human rabies infections in the United States in the 1960s and 1970s were diagnosed after death. Subsequently, the percentage of infections diagnosed postmortem has been higher.[48,179]

In recent years in the United States, most of the individuals with rabies have no recognized exposure to potentially infective animals, yet an account of animal exposure is often the only stimulus for laboratory identification of rabies as the cause of the encephalitis.[48] All but four of the individuals with rabies of bat origin had no history of bite, and approximately half had no history of any bat exposure.

The significance of undiagnosed human rabies is uncertain. When the diagnosis is made, members of the patient's family and all health care personnel who had a significant exposure to the patient are given postexposure immunoprophylaxis. However, human-to-human transmission of rabies has been reported only following tissue or organ transplantation, except for two individuals in Ethiopia.[81]

LABORATORY DIAGNOSIS OF RABIES

Conventional laboratory procedures are not helpful in establishing a diagnosis of rabies. Cerebrospinal fluid protein and leukocyte counts may be modestly elevated, but these changes are nonspecific.

Currently available techniques for a definitive laboratory diagnosis of rabies are usually nondiagnostic in the early days of infection and become useful only a week or more after the onset of illness. The diagnosis of rabies should be confirmed as quickly as possible so the number of persons exposed to the infection can be limited and therapy for those exposed can be initiated promptly. In industrialized nations, the number of persons exposed to a hospitalized rabies patient can number in the hundreds.[131,163]

If rabies is suspected, a complete set of samples should be collected for testing by all currently available diagnostic procedures. Consultation is available from the CDC 24 hours a day, 7 days a week, and should be obtained (877-554-4625; www.cdc.gov/ncidod/dvrd/rabies). However, samples taken antemortem cannot definitively rule out rabies. If infection is seriously suspected, repeated sampling is needed.[163] Samples can be transported overnight to the CDC or to state laboratories.

The rabies virus is systemically disseminated shortly after CNS infection and can often be detected with labeled antibodies or by RNA extraction. Currently, detection of rabies RNA in saliva by RT-PCR appears to be the most reliable procedure. Saliva should be collected with a sterile eyedropper or pipette and placed in a small sterile container that can be sealed securely. No preservative or other material should be added.

A 5- or 6-mm punch biopsy of hair-bearing skin with at least 10 hair follicles from the nape of the neck is commonly used; it should be placed on sterile gauze, moistened with sterile water or saline, and placed in a sealed container with no other fixative or preservative.

At least 0.5 mL of serum (not whole blood) is needed to test for antibodies by immunofluorescence and virus neutralization. If no vaccine or rabies immune serum has been administered, the presence of antibodies is diagnostic and testing for CSF antibodies is not needed. At least a similar volume of CSF is needed for antibody studies.

Rabies virus can sometimes be found in imprints from the cornea. To avoid damaging the cornea, an ophthalmologist should prepare the smears after consultation with the rabies testing laboratory. RT-PCR and immunostaining are used to identify the viral antigens.

Brain biopsies should not be routinely performed because human rabies is so rare in industrialized countries, and no effective treatment is available. If a biopsy is performed to rule out another condition, it can also be examined for rabies.[163]

Rabies virus often can be isolated from body fluids, particularly saliva and CSF. The murine neuroblastoma cells used for isolation of rabies are more susceptible to that virus than any other cell line tested, and culture on such cells can usually provide a diagnosis within 24 hours. Mouse inoculation may take 15 to 30 days, although the time can be shortened by sacrificing mice starting 5 days after inoculation and examining the brains with fluorescent antibodies.[159,189] In industrialized nations, facilities for such studies are limited almost entirely to state and national laboratories. In developing countries, they often are unavailable.

Diagnosis in Attacking Animals

Prior to 1903, the diagnosis of rabies in attacking animals was based entirely on the clinical features of the disease, or on the presence of unusual material in the animal's stomach that evidenced bizarre behavior. The dog that attacked Joseph Meister, the first recipient of Pasteur's antirabies vaccine, was diagnosed as rabid because it had hay, straw, and wood in its stomach.[7] In that year, Negri and Bosch described the typical neuronal cytoplasmic inclusions, and for many years the laboratory diagnosis of rabies in animals depended on the detection of such bodies. However, only 60% to 80% of infected animals have identifiable inclusions.[162] Typical inclusion bodies are scarce in arctic foxes, for instance.[67]

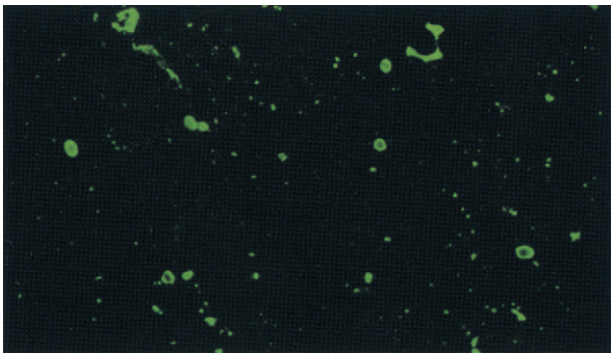

Figure 54-6. Fluorescence-labeled rabies-specific antibody staining of a smear from the brain of a rabid Canadian fox. (×400.) (Courtesy Center for Disease Control and Prevention.)

Immunofluorescent detection of rabies antigen in smears of cerebral tissues, which was introduced in 1958 and became widely used in the early 1960s,[88] is far more reliable in experienced hands. Comprehensive analyses of the performance on survey examinations established that in major U.S. public health laboratories, the sensitivity of fluorescent antibody examination is nearly 100%. Fluorescent antibodies are used to identify rabies antigen in tissue culture (see later) because the rabies virus produces few cytopathogenic changes.

The major shortcoming of the procedure is its reduced sensitivity for rabies antigen in decomposed brain tissue. Some investigators have concluded that failure to identify rabies antigen with fluorescent antibodies in partially decomposed brain tissue cannot justify withholding postexposure therapy for exposed individuals.[162]

A procedure to confirm negative diagnoses is essential. Rabies diagnosis by intracerebral mouse inoculation was introduced in 1935 and demonstrated that only 85% to 95% of rabies infections could be identified by examination for inclusion bodies alone.[157] Eventually, most laboratories, even those in developing countries, adopted adult or suckling mouse inoculation. Tissue culture inoculation began with inoculation of chick embryo cells in 1942.[159] Now, tissue culture isolation on mouse neuroblastoma cells is far faster. Only 24 hours are needed to confirm a positive diagnosis, in contrast to the 15 to 30 days needed for mouse inoculation. Tissue culture isolation is also the most sensitive technique for confirming negative immunofluorescence examination results, at least in laboratories with suitable facilities and appropriate personnel.[32,141,159] The rabies virus does not produce cytopathic changes in tissue culture, and fluorescence-labeled antibodies have to be used to diagnose the infection (Fig. 54-6).

POSTEXPOSURE RABIES THERAPY

If the effectiveness of the therapy for the 15-year-old girl in Wisconsin is confirmed[58,186] (see Mortality, earlier), it will revolutionize the treatment of rabies in humans who have developed a clinical infection. However, postexposure treatment for humans will still consist of reducing the viral inoculum by cleansing the wound as thoroughly as possible, administering antiserum to help control viral reduplication and spread (passive immunization), and administering rabies vaccine to establish immunity to the virus before signs of infection appear (active immunization). "Rabies vaccination is a race between the active immunity induced by vaccination and the natural course of infection."[170]

Identifying Exposure

Exposure to rabies is divided into bite and nonbite categories. A bite is considered a significant exposure if it penetrates the skin. Scratches that break the skin are also considered significant exposures because the claws could be contaminated by saliva. Unprovoked attacks are more likely to have been inflicted by a rabid animal than are provoked attacks, but determining whether a dog or cat bite has been provoked is frequently difficult. Bites that occur while feeding or handling apparently healthy animals are generally regarded as unprovoked, but some animals, particularly dogs, may bite anyone walking by or riding a bicycle.

Nonbite exposure consists of contamination of cutaneous wounds—including scratches, abrasions, and weeping skin rashes—with saliva, CSF, or brain tissue from a rabid animal. If the material is dry, it is considered noninfectious.[33] Contamination with urine from a rabid animal or person is not considered an exposure even though rabies viruses have been isolated from kidneys and urine. A laboratory technician cut by a broken specimen container was given postexposure therapy.[62]

Importantly, the rabies virus does pass through intact mucous membranes, and any mucous membrane contact, particularly membranes of the oral cavity or conjunctiva, with saliva or other infectious material from a possibly rabid animal is considered an exposure.

Exposure of medical personnel or family members caring for patients with rabies is a significant problem. High-risk contact is defined as a percutaneous (through needlesticks or open wounds) or mucous membrane contact with saliva, CSF, or brain tissue, which are essentially the same as bite and nonbite exposures to rabid animals. Individuals who have had a high-risk contact should receive postexposure immunoprophylaxis. However, routine infection isolation procedures, including respiratory precautions, minimize the risk for medical personnel caring for patients with rabies.[33]

Individuals who have not had a high-risk contact do not need postexposure immunoprophylaxis, although such treatment is sometimes administered to allay anxiety.

In areas where canine rabies is not enzootic, which includes all of the United States except for the area along the border with Mexico (particularly southern Texas), a healthy domestic dog or cat that bites a person should be confined and observed for 10 days, particularly if the animal has been previously vaccinated. A veterinarian should evaluate any illness during confinement. If rabies is suspected, the animal should be humanely killed, and its head should be shipped to a qualified laboratory.[53] The head must be refrigerated during shipment. Examination for rabies cannot be reliably performed on decomposed brain tissue.[163]

Such confinement and observation was judged safe for exposures to ferrets in 1998. Scientific evidence that the same quarantine period would be adequate for wolf hybrids does not exist because studies of pathogenesis and virus shedding have not been performed. Hybrids that bite humans should be euthanized immediately, and their heads should be shipped to reliable laboratories.[119]

The significance of the laboratory's qualifications was emphasized by the death from rabies of a U.S. citizen in 1981 after he was bitten by a dog in Mexico. The dog's head was shipped to a Mexican laboratory, where it was examined with Seller's stain instead of a fluorescent antibody technique. Because no evidence of rabies was found with this less-sensitive technique, he was not given postexposure therapy.[122] In a Thai investigation, 13 of 404 rabid animals diagnosed with fluorescent antibodies did not have Negri bodies identifiable with Seller's stain.[149]

Any stray or unwanted animal that has attacked should be killed immediately and its head submitted for rabies examination. Euthanasia does not have to be delayed for further development of the infection in an attacking animal for a reliable diagnosis to be made.[163]

No one in the United States has died of rabies when the attacking dog or cat has been healthy after 10 days of observation.[153] However, dogs injected with an Ethiopian strain of rabies virus excreted virus in the saliva up to 13 days before signs of disease were observed,[76] and dogs that have recovered from experimental rabies excrete virus in saliva for as long as 6 months after recovery.[82]

If an attacking dog or cat is rabid or is suspected to be rabid, postexposure therapy should be initiated at once. However, a reliable diagnosis of the presence or absence of rabies in animals can usually be completed in less than 1 day.[163] If the dog or cat escapes and is not suspected to be rabid, the Advisory Committee on Immunization Practices (ACIP) of the U.S. Department of Health and Human Services recommends that local public health officials be consulted.[53]

In a study of postexposure therapy practices in the emergency departments of a number of university hospitals from all parts of the United States, the most frequent inappropriate administration of therapy was for animals that could be observed for 10 days. The most common failure to administer postexposure therapy was for animals that could not be observed. The investigators emphasized that physicians often failed to use the well-informed consultative services available 24 hours a day from their local health departments or the CDC.[115]

In the United States, any exposure to a skunk, raccoon, bat, fox, or other carnivore should be considered a rabies exposure. Treatment should be initiated immediately, except when the exposure has occurred in a part of the continental United States known to be free of rabies, and when the results of immunofluorescent testing are to be available within 48 hours. If the animal is captured, it should be killed and its head shipped to a qualified laboratory immediately. The signs of rabies in wild animals, including wolf hybrids, are not consistent enough or sufficiently well known for a 10-day observation period to suffice in reliably determining whether the animal was rabid.[53]

Small rodents (squirrels, hamsters, guinea pigs, gerbils, chipmunks, rats, and mice) and rabbits are rarely found to be infected with rabies and have not been known to produce rabies in humans. ACIP recommends that local health officials be consulted after a bite by a rodent or rabbit.[53]

In areas where canine rabies is enzootic, which includes essentially all of Latin America and Africa and most of Asia, postexposure treatment should be initiated immediately after a bite by a dog or cat, but it can be terminated if the animal remains healthy during the 10-day observation period. The vaccination status of the biting animal must be ignored. Vaccines used for animals in developing countries are usually not as reliable as those used in the United States, and fatal rabies has been reported in U.S. citizens and in others who were bitten by "vaccinated" dogs in developing countries and did not obtain prompt postexposure therapy.[47,181]

In developing countries, except for those few areas where rabies does not occur, all attacking animals should be considered rabid if they escape. Information about rabies in developing countries is not widely disseminated. The number of rabies deaths in the United States from infections acquired outside of this country, 14 of 38 since 1980, clearly indicates the need for travelers to know about this hazard.

Human rabies immune globulin and reliable, cell-culture rabies vaccine are available for U.S citizens through United States embassies and consulates in developing countries, even though these agents may not be available in local hospitals.[51] Individuals bitten by any animal in a developing country should seek therapy at the U.S. embassy unless they are absolutely certain that some other source, such as QSMI in Bangkok or the CIWEC Clinic in Kathmandu, can provide equally reliable care.

Initial Wound Management

Immediate and thorough cleansing of bite wounds with soapy water to reduce the viral inoculum is an essential and highly effective component of postexposure therapy. Fishbein, former Chief of the Viral and Rickettsial Zoonoses Branch of the CDC, has stated, "Local treatment is perhaps the single most effective means of preventing rabies." He has also said, "In some groups exposed to a single rabid animal and many laboratory investigations, local wound care alone has been found to be as or more important than vaccine alone."[83] Vodopija has declared "Washing the wound with soap and water or with other substances that are lethal to rabies virus may be crucial for survival, irrespective of subsequent immunization."[172] Fangtao, who has had extensive experience with rabies in China, asserts, "The protective effect of each (wound care, immune globulin, and vaccine) should be considered equivalent."[74] Neglect of the wound or inadequate wound care has resulted in rabies in individuals who received ideal immunotherapy.[22,53]

Many experimental studies have shown that the duration of the incubation period following rabies exposure is inversely proportional to the size of the viral inoculum—a large inoculum produces generalized infection in a much shorter time than a small inoculum.[61] If rabies vaccine is to induce immunity before infection appears, it must have sufficient time. After a severe rabies exposure, such as a bite about the head or neck, or multiple bites, a major reduction in the number of virus organisms introduced may be essential to allow time for immunization.

In experimental studies, the best results have been obtained by thoroughly cleaning the wound with soap and water, and then irrigating it with a virucidal agent such as povidone-iodine or a 1% solution of benzalkonium chloride (Zephiran). Soap neutralizes benzalkonium chloride and must be completely rinsed from the wound before it is irrigated with the detergent. For deep wounds, only virucidal substances such as benzalkonium chloride or povidone-iodine have been found to be effective. If benzalkonium chloride is not available, 70% alcohol (ethanol) or iodine, either tincture or aqueous solution, can be instilled into the wound. However, the best cleansing agent that can be obtained immediately must be used.[33,189] The WHO Expert Consultation on Rabies strongly recommends washing

bite wounds for at least fifteen minutes, and advises obtaining professional assistance if possible.[180a] If these measures are painful, and they often are when carried out with appropriate vigor, the area can first be anesthetized with a local anesthetic, taking care not to transfer infectious fluids further into the wound.

Appropriate measures to prevent bacterial infection, including antibiotics if indicated, and tetanus prophylaxis should also be instituted.[33,140] The WHO Expert Committee and others recommend that bites not be sutured.[33,38,53]

Rabies Immune Globulin

Although proposed and investigated by Babes and his colleagues over a century ago,[8] the benefit of hyperimmune serum immediately after rabies exposure was established only about 50 years ago. The most dramatic study was initiated by Koprowski and carried out in Iran in 1954. Twenty-nine persons were bitten by a single rabid wolf. Thirteen individuals with bites about the head were treated with antiserum and neurally derived vaccine; only one died. Five individuals with similar bites received vaccine alone; three died.[18]

Similar results were demonstrated in China with a much more effective cell-culture vaccine by Lin and his associates, who suggested that immune serum might be effective because it stops virus from entering susceptible cells (i.e., nerves).[75] The need for immune serum with human diploid cell vaccine (HDCV) has been demonstrated by the 1982 death from rabies in a 29-year-old woman, a U.S. citizen in Rwanda, who received prompt therapy with HDCV but no hyperimmune serum.[68]

Measurements of the antibody response to earlier rabies vaccines demonstrated that immune serum reacted with the vaccine and limited the antibody response in the first 7 to 10 days of administration. These data were considered evidence of the passive immunity that was essential for early control of the rabies virus, particularly when the viral inoculum was large and the incubation period before the development of clinical rabies was likely to be short.[17] However, with contemporary cell-culture vaccines that have a concentration of 2.5 IU/mL, such inhibition of the serum neutralizing antibody response is not seen.[172]

The only rabies immune globulin licensed for use in the United States is human rabies immune globulin (HRIG), which is prepared by cold ethanol fractionation of plasma from hyperimmunized human donors. It is virtually free of significant side effects.[181]

In developing countries, immune serum is rarely administered, because it is expensive or unavailable. Only about one third of the immune serum required for rabies postexposure therapy is being produced. Travelers who have received postexposure therapy are usually not given immune serum unless they know enough about rabies postexposure prophylaxis to demand its administration.

When immune serum is given, it is usually of equine origin, which costs only about one-tenth as much as HRIG.[182] However, contemporary highly purified equine rabies immune globulin (ERIG) is not associated with the 15% to 46% incidence of serum sickness typical of older equine antirabies serum.[38] In 3575 individuals treated at the QSMI, ERIG from Pasteur Vaccins of France, Sclavo of Italy, and the Swiss Serum and Vaccine Institute produced allergic reactions in 0.87%, 3.58%, and 6.19% of the recipients, respectively. The reactions were uniformly mild and lasted less than a week with appropriate therapy. Only 9 of the 66 patients with reactions required steroid therapy; the remainder were satisfactorily treated with analgesics and antihistamines. ERIG is also produced in developing countries, but the purification procedures and potency criteria have not been published, and these products may be associated with a higher incidence of adverse reactions.

At QSMI, an initial intradermal injection of horse serum is administered, and individuals who manifest sensitivity are treated with HRIG. One 17-year-old boy did not react to the intradermal injection, but with subsequent injections he had an anaphylactic reaction that was successfully treated with epinephrine, hydrocortisone, and chlorpheniramine.[180,181]

DNA recombinant technology has been used to express three human rabies virus-neutralizing monoclonal antibodies in a rhabdovirus vector. Growth of the recombinant in cell culture produced high yields of three monoclonal antibodies that differed in epitope recognition. A cocktail of these antibodies neutralized several fixed and street rabies viruses. Mice and hamsters treated once with this cocktail after being infected with a lethal dose of rabies virus were protected. The protection was comparable to that provided by human rabies immune globulin, which strongly suggests that such antibody cocktails could be used for postexposure treatment of humans. Importantly, such cocktails could help compensate for the inadequate production of rabies immune globulin that now prevails in developing nations.[130]

The dose of immune globulin is 20 IU/kg for HRIG or 40 IU/kg for ERIG. Larger-than-recommended doses should not be administered because the immune globulin neutralizes vaccine given simultaneously. Immune globulin should be administered as soon after exposure as possible, but is clearly beneficial when administered within 7 days. In view of the long incubation period for some human rabies infections, immune globulin administration may be advisable regardless of the interval after exposure.

Currently, all agencies recommend injection of as much immune globulin as possible around the bite site.[181,189] For bites on the fingers and similar locations where only a limited amount of globulin can be injected locally, the remainder should be injected intramuscularly.[33] For individuals with multiple bites, which is common with rabid dog attacks in developing countries, the immune globulin may have to be diluted with saline to achieve uniform injections. The syringe used to inject immune globulin must not be used to inject vaccine, which would be inactivated by residual antiserum.

Rabies Vaccines

Pasteur revolutionized rabies therapy on July 7, 1885, when he began administering rabies vaccine to Joseph Meister, a 9-year-old boy who had been bitten 14 times by a dog that was killed and diagnosed as rabid because its stomach contained hay, straw, and fragments of wood.[171,173] Although the rabies diagnosis in this animal may be suspect, and the boy may not have developed a clinical infection, Pasteur in 1886 observed only a single failure in a series of 688 treated patients, many of whom must have been exposed to rabid animals and would have been expected to die without therapy.[7] Interestingly, Meister's only employment as an adult was as a security guard for the Pasteur Institute.[157]

Pasteur's first rabies vaccine was produced from rabid rabbit spinal cords that were allowed to dry to attenuate the virus. After 15 days, the cords were noninfectious, so the first injec-

tions were made with 14-day-old spinal cords, and subsequent injections were made with cords that had been dried for shorter times.[161,170] Soon, Roux and Calmette demonstrated that desiccated rabbit spinal cords retained appropriate virulence when preserved in glycerin, which greatly facilitated the production and storage of such vaccine.[39,138] Although other vaccines were soon developed, the Pasteur rabbit spinal cord vaccine was administered at the Pasteur Institute of Paris as recently as 1953.[170]

Subsequently, similar vaccines have been prepared in brain tissues from various animals, particularly goats and sheep, and killed with formaldehyde, phenol, or β-propiolactone.[96,161] These preparations are collectively known as Semple or neural tissue vaccines, and Semple's introduction of such vaccines in 1911 was a significant advance. In 1955, Fuenzalida and Palacios in Chile introduced the use of suckling mice less than 3 days old for the preparation of neurally derived vaccines. Such animals have little cerebral myelin and are thought to yield a product that contains less myelin basic protein. Currently, animals no older than 1 day are used. In Thailand, this vaccine has been found to be as poorly immunogenic and to be associated with as many side effects as the others,[86,182,189] but others have found the incidence of neural reactions to be lower.[171] At the present time, neural tissue vaccines are still employed for a large percentage of all postexposure rabies therapy worldwide because they are inexpensive. Of 50 million doses of rabies vaccine administered worldwide each year, 20 million are still neural tissue vaccines.[34]

Such animal brain vaccines have been described as the crudest biologic material administered to man. These vaccines have low immunogenicity, and as many as fifteen 5-mL injections of a 5% to 10% suspension of brain tissue are required to produce satisfactory antibody titers. Intense local reactions are common, and many patients abscond from treatment.[34] In Taiwan between 1952 and 1955, 55% of individuals who began rabies vaccination failed to complete treatment.[176] In one study, 20 of 681 persons bitten by dogs proven to be rabid refused therapy because they had witnessed the side effects in other villagers. Nine (45%) died of clinically diagnosed rabies. Another 62 persons received Semple vaccine even though the dogs to which they were exposed were not rabid, presumably because the results of testing the dogs were not accepted as valid.[149]

The potency of these vaccines is highly variable. Some batches are known to have been ineffective. After 15 years of observations, the Pasteur Institute of Iran, a branch of the Pasteur Institute in Paris, and a WHO Collaborating Center for Reference and Research in Rabies, concluded that rabies vaccines prepared in animal brains were totally ineffectual for preventing rabies in individuals bitten about the head or neck, who would be expected to develop encephalitis within a short time after exposure.[19] Pasteur observed that his treatment was not always successful, particularly after bites around the face.[157] In Bangkok, 11% of a group of patients who died of rabies encephalitis had received complete courses of the vaccine starting soon after they were bitten.[169] The vaccine appears to be beneficial for less severe exposures.[168]

Since the time of Pasteur, the failure of immunoprophylactically treated persons to develop clinical rabies has been attributed to the vaccine, and some of these individuals did produce antirabies antibodies. However, many individuals attacked by rabid animals do not develop clinical rabies. In fact, humans are considered to have a low susceptibility to rabies.[78] Citing the 1912 studies of Babes, which now could not be repeated, Baer has emphasized that only about 15% of individuals bitten by rabid dogs and not vaccinated develop rabies (Table 54-4).[9,143] The absence of rabies infection in many individuals treated with Semple vaccine is undoubtedly a reflection of the failure of the attacking animals to transmit the infection and not of vaccine effectiveness.

The neuroparalytic reactions associated with Semple rabies vaccines are the most serious neurologic complications that follow any vaccination,[97] and they were reported only 4 years after Pasteur introduced his vaccine.[20] Remlinger reported 26 cases between 1888 and 1905.[132] Even dogs vaccinated with this product developed anorexia, "depression, nervous symptoms, a morbid desire to bite," or even paresis of the hind limbs.[36] Of the patients receiving such vaccines, 15% develop electroencephalographic abnormalities, and 5% develop measurable antibodies against brain tissue. About one fourth of the reactions are fatal, and an additional one third cause permanent damage.[13,17,118] An incidence of neuroparalytic reactions of approximately 1 in 1200 patients seems to be generally accepted, but much higher rates of 1 in 400, 1 in 220,[97] 1 in 142,[149] and even 1 in 120 have been reported.[160] Probably the most accurate evaluation of the frequency of such complications is that the incidence is not known. The countries that have a high incidence of rabies, which are the countries that use neurally derived vaccines for postexposure therapy, also have very poor or no programs for reporting complications.[161]

Recently, the specter of transmission to humans of spongiform encephalopathies caused by prions from infected sheep by neural tissue vaccines derived from sheep brains has been raised.[34]

Because so many problems were associated with Semple vaccines, a safer vaccine was developed with viruses grown in duck embryos. This vaccine was not as immunogenic as contemporary vaccines, and 14 to 23 injections were required to produce satisfactory antibody titers. In addition, this vaccine also had a significant incidence of adverse side effects. Reactions to duck embryo vaccine in one retrospective analysis of 424,000 persons consisted of 22 cases of anaphylaxis, four cases of transverse myelitis, five cases of cranial neuropathy, two cases of nonfatal encephalopathy, and two cases of fatal encephalopathy that may actually have been fatal rabies infections.[139] Immunization with vaccine grown on duck embryos has failed to prevent rabies, but for many of the failures a prolonged interval separated the exposure and immunoprophylaxis.[2]

Human Vaccination

The postexposure prevention of human rabies by immunization is unique in the therapy of infectious diseases, and it is possible only because the moment of exposure can be vividly remembered and the incubation period is relatively long. The currently recommended procedure for producing active immunity in the United States consists of injections into the deltoid muscle of 1.0 mL of vaccine on days 0, 3, 7, 14, and 28 after animal exposure. This schedule is known as the Essen scheme in recognition of the contributions of Ernst Kuwert of the Essen Institute of Medical Virology and Immunology.[171] Immunoprophylaxis is most effective when begun within 24 hours of exposure, but the average delay between the animal exposure and vaccination in the United States and in Nepal has been 5 days.[148] Because the incubation period for rabies may be prolonged, no upper limit can be set for the time after a potential

TABLE 54-4. Babes' 1912 Estimates of Mortality among Unvaccinated Persons Exposed to Animals Assumed to Be Rabid

ATTACKING ANIMAL	LOCATION OF EXPOSURE	TYPE OF EXPOSURE	LIKELIHOOD OF MORTALITY (%)
Wolf	Face	Bites, multiple, severe	80–100
Cat	Face	Bites, multiple, severe	70
Dog	Face	Bites, multiple, severe	60
Dog or cat	Other part of head	Bites, multiple, severe	50
Wolf	Neck or arm	Bites, multiple, severe	40
Wolf	Face	Bite, single	40
Cat	Face	Bite, single	40
Dog	Face	Bite, single	30
Wolf or cat	Fingers or hand	Bites, severe	20
Dog	Fingers or hand	Bites, severe	15
Wolf	Trunk or legs	Bites, severe	15
Wolf, cat, or dog	Face	Bites, superficial	10
Wolf, cat, or dog	Hands	Bites, superficial	5
Wolf	Face	Scratches	5
Wolf, cat, or dog	Trunk or legs	Bites, severe, through clothes	3
Wolf, cat, or dog	Hands or exposed skin	Bleeding superficial wound	2
Wolf, cat, or dog	Clothing covered skin	Bleeding superficial wound	0.5
Wolf, cat, or dog	Recent wounds in contact with saliva	—	0.1
Wolf, cat, or dog	Wounds older than 24 hr in contact with saliva	—	0.0

From Traité de la Rage. Paris, Bailliere et Fils, 1921, p 81, as cited by Baer GM: Oral rabies vaccination: An overview. Rev Infect Dis 10(Suppl):S64, 1988.

rabies exposure at which postexposure vaccine therapy should be given.[22,189]

An alternative treatment schedule, termed the 2-1-1 regimen, was first developed in Yugoslavia. On day 0, two 1-mL doses of vaccine are injected into the left and right deltoid muscles. Additional 1-mL injections are given on days 7 and 21. This schedule was originally intended for use only after severe exposures, when the incubation period could be expected to be short. However, in a number of countries it is used routinely because it produces antibody levels just as high as the Essen scheme and fourth and fifth clinic visits are avoided.[172]

The only rabies vaccines licensed for use in the United States are prepared from viruses grown in cell cultures and inactivated with β-propiolactone. HDCV, the first to be licensed and the gold standard against which other vaccines are compared, was made possible by development of the WI 38 cell line of diploid human fibroblasts by Hayflick and Moorhead, and adaptation of the Pitman-Moore strain of rabies virus to growth in such cells by Wiktor, Plotkin, and Koprowski at the Wistar Institute in Philadelphia.[157,171] The virus used to prepare this vaccine was originally isolated in 1882 by Pasteur from a cow bitten by a rabid dog, and it was maintained by serial passage in rabbit brain before it was adapted to cell culture.[144,171] Imovax Rabies, the HDCV available in the United States, is produced in cultures of human diploid cells by Sanofi Pasteur. This vaccine was introduced in December 1974 and was licensed for general use in the United States on June 9, 1980.[43] A single-dose vial containing lyophilized vaccine is reconstituted in the vial to a final volume of 1.0 mL. Imovax Rabies is licensed for postexposure and pre-exposure intramuscular vaccination. The unreconstituted vial must be refrigerated; it cannot be carried into remote areas in first-aid kits.

Because HDCV is expensive and cannot be produced in sufficient quantities to supply worldwide demands, a variety of cell-culture vaccines have been developed. These vaccines, when produced according to accepted standards, are all considered equally effective.[33] These vaccines are far more immunogenic than earlier products, and protective antibody levels are routinely achieved with only four or five injections.[53,170] A protective level is defined as either an antibody concentration that will completely neutralize rabies viruses at a dilution of 1:5 as determined by the rapid fluorescent focus inhibition test, or 0.5 IU/mL. These vaccines are so effective that measurement of postvaccination antibody levels is recommended only for individuals who are immunodeficient as the result of disease or therapy.

The effectiveness of HDCV was dramatically demonstrated during 1975 and 1976 in Iran by the complete prevention of rabies in 45 persons attacked by dogs or wolves subsequently proven to be rabid.[17] Between 1980 and 1982, all 511 individuals bitten by rabid animals in the United States and treated with five doses of HDCV, and with HRIG, survived.

In the United States, two other cell-culture vaccines have been licensed. Rabies Vaccine Absorbed (RVA) is prepared from viruses grown on fetal rhesus lung diploid cell cultures, concentrated by adsorption to aluminum phosphate, and produced in a liquid form. RVA was licensed on March 19, 1988. It has been used to vaccinate humans allergic to HDCV, but it is currently (2006) unavailable.[145]

At least in part because the raccoon rabies epizootic had increased consumption of rabies vaccine so dramatically, the U.S. Food and Drug Administration (FDA) licensed purified chick embryo vaccine (PCEV) in October 1997. This vaccine (RabAvert) has been extensively used worldwide. It is thought to produce fewer allergic reactions because it does not contain human albumin altered by β-propiolactone. All three preparations are considered equally efficacious and safe for postexposure therapy and pre-exposure intramuscular vaccination.[53]

In its 1992 Eighth Report, the WHO Expert Committee on Rabies stated, "Prompt and thorough cleansing of the wound, and administration of purified equine or human rabies immunoglobulins and cell-culture rabies vaccine immediately after exposure virtually guarantee complete protection. . . . Pregnancy and infancy are never contraindications to postexposure rabies vaccination."[46] Although some antigens, particularly surface glycoprotein antigens, vary widely in different viral strains, standard vaccines have provided effective protection against all wild (or street) rabies viruses. These vaccines also appear to be effective against other lyssaviruses, except Mokola and Lagos bat virus, although no alternative vaccines are available.[179,189]

Occasional failures with cell-culture vaccines have been reported, but in essentially every instance, treatment had not been administered correctly. Many of the individuals did not receive immune globulin.[33,118,189] Some had the vaccine injected into gluteal muscles, which is thought to be the cause of these failures.[13,33,45,53,97] Hepatitis B vaccine gluteal injections have been found to be less consistently effective than are injections in the deltoid.[13] All injections of rabies vaccine must be made in the deltoid muscle, or in the quadriceps muscle for small children.

Approximately 18,000 persons receive pre-exposure vaccination, and an additional 10,000 receive postexposure immunoprophylaxis each year in the United States, but reactions of any type are very uncommon. Reactions following HDCV administration during the first 46 months of its use (and reported to the CDC) occurred in 108 individuals, a rate of 11 per 10,000 vaccinees. Few patients required hospitalization, and no fatal reactions were reported. Of these reactions, nine (1 of 10,000) were immediate hypersensitivity reactions that occurred within minutes to hours after injection and were characterized by bronchospasm, laryngeal edema, and generalized pruritic rash, urticaria, or angioedema. All nine reactions followed postexposure immunization of unvaccinated persons or pre-exposure vaccination.

Eighty-seven delayed reactions appeared 2 to 21 days after injection and were characterized by a generalized pruritic rash or urticaria. Some of the individuals also had arthralgias, arthritis, angioedema, nausea, vomiting, fever, and malaise. Twelve reactions were classified as indeterminate. Unlike the immediate reactions, 81 (93%) of the delayed reactions followed booster injections for individuals who had been vaccinated previously. Most patients improved in 2 to 3 days when treated with antihistamines, but a few required systemic corticosteroids and epinephrine.[43]

The reactions are attributed to β-propiolactone-altered albumin in the vaccine.[137] HDCV vaccine that is free of β-propiolactone has been developed but is not licensed for use in the United States.[25] PCEV and RVA are not made with albumin. Further rabies booster immunizations are not recommended for individuals who have had this type of reaction, but exposure to rabies should be treated in the usual manner.[43]

Neurologic reactions after HDCV administration have been extremely rare. After millions of vaccinations worldwide, three Guillain-Barré–type paralytic reactions have been described, and all three individuals recovered. Other neurologic disorders have occurred at the time of vaccination, but a definite causal relationship has not been established.[25,137]

PRE-EXPOSURE VACCINATION

Pre-exposure vaccination can be achieved with three deltoid or quadriceps intramuscular injections of 1.0 mL of cell-culture vaccine on days 0, 7, and 21 or 28.

Pre-exposure vaccination with 0.1 mL of HDCV injected intradermally on the same schedule was approved by the FDA in April 1987. However, intradermal vaccination does not result in antibody levels as high as those produced by intramuscular injections, and antibodies do not last as long. Some investigators have recommended that only intramuscular injections should be administered.[34] The question is moot because the raccoon epizootic on the East Coast has increased vaccine utilization so much that syringes with 0.1 mL of the vaccine are not being produced.

After a rabid animal bite, vaccinated individuals need only intramuscular injections of 1.0 mL of cell-culture vaccine on days 0 and 3. (Immune serum is not needed, a major consideration for travelers in developing countries where immune serum is often not available.) These additional two injections are essential. Fatal rabies encephalitis developed in a 23-year-old female Peace Corps Volunteer in Kenya who had previously been vaccinated for rabies, did not report a bite by a pet puppy too young to have been vaccinated, and did not receive postexposure therapy.[121]

Chloroquine interferes with the response to rabies vaccine; vaccination should be completed before malarial chemoprophylaxis with chloroquine is begun. Whether other agents used for malarial chemoprophylaxis interfere with the response to rabies vaccine has not been ascertained.[34]

ACIP divides individuals for whom pre-exposure vaccination is recommended into three groups. Group one consists of workers in rabies laboratories and others who may unknowingly be exposed to rabies, for whom vaccination followed by evaluation of antibody titers every 6 months is advised. Boosters are needed if antibody concentrations fall below a titer of 1:5 by the rapid fluorescent focus inhibition test, or 0.5 IU/mL.

Group two includes spelunkers, veterinarians in areas where rabies is endemic, animal control workers, and fish and game

wardens, for whom vaccination followed by serologic testing or a booster every 2 years is recommended.

Group three consists of veterinarians and animal control workers in areas of low rabies endemicity, travelers to foreign rabies-epizootic areas who are staying 30 days or more, and veterinary students, for whom vaccination but no subsequent serologic testing or boosters is advised. Vaccination is not recommended for the general U.S. population.[53]

Investigators in Nepal have found the incidence of animal exposure requiring immunoprophylaxis in travelers to be quite low, approximately 1 in 123,000 days (337 years). They have disagreed with the recommendation that travelers to foreign rabies-epizootic areas staying 30 days or more be vaccinated, because the cost of immunization is high and because the incidence of allergic reactions to booster immunizations is significant.[148] However, in a subsequent study in Thailand, a much higher incidence of exposure was found, and the investigators recommend intramuscular pre-exposure vaccination for travelers to such areas.[127]

Several of the recent American victims of rabies acquired outside the United States, and the only genotype 1 rabies victims in Australia, were children who, perhaps in fear of punishment, did not tell their parents about bites they had received. The problem of protecting children, who run the highest risk of animal bites, may not be widely recognized.[137] Children who cannot understand the hazard of rabies in developing countries probably should be routinely vaccinated; they must also be closely observed so that postexposure booster vaccination can be administered. The effectiveness of pre-exposure vaccination is highlighted by the fact that no one residing in the United States who has received pre-exposure vaccination with a modern cell-culture vaccine has contracted rabies.[33]

RABIES THERAPY IN DEVELOPING COUNTRIES

Rabies therapy in developing countries must be modified by financial considerations. The cost for such therapy for a typical laborer could be devastating.[149] At least four different measures have been taken to reduce the costs of postexposure rabies therapy and increase the availability of reliable cell-culture vaccines. The simplest has been the transfer of cell-culture vaccine–manufacturing technology to developing countries but, in spite of support by WHO and other agencies, progress has been slow. Local manufacturers in Latin America and Asia are producing Vero cell rabies vaccine "copies" and primary hamster kidney cell rabies vaccines. Because these vaccines do not adhere to the strict standards imposed by the FDA or the European Pharmacopeia, they are inexpensive to produce.

The substitution of ERIG for HRIG, which results in a 90% cost savings, has already been discussed.

A third approach has been the use of small-volume intradermal vaccine injections instead of larger-volume intramuscular injections. The Warrells, Phanuphak, and their coworkers at QSMI pioneered the use of intradermal vaccination for postexposure immunoprophylaxis because a smaller volume of vaccine would be required. The regimen developed by the Warrells consisted of intradermal injections of 0.1 mL of HDCV at eight different sites on postbite day 0, injections at four sites on day 7, and injections at one site on days 28 and 90, which reduced the vaccine expense to 30% of the cost of five 1.0-mL intramuscular injections.[177,182] Phanuphak intradermally injected 0.1 mL of purified Vero cell rabies vaccine (PVRV) at two sites on days 0, 3, and 14, and at one site on days 30 and 90. This regimen reduced the expense for PVRV, which is already much less expensive than HDCV, by an additional 68%.[125,126]

One hundred Thai patients, who had injuries severe enough to produce active bleeding inflicted by animals proven rabid by fluorescent antibody testing, were treated according to the schedule developed by Phanuphak and his colleagues in a prospective study. This procedure is now known as the Thai Red Cross intradermal postexposure rabies treatment schedule, or TRC-ID. All patients were uniformly protected from rabies. Since 1987, the TRC-ID regimen has been routinely used for postexposure therapy of over 1000 patients a month at QSMI, with only one failure, a 53-year-old alcoholic with cirrhosis who did not report for treatment until 6 days after his exposure.[64]

An anticipated problem with intradermal injections was the inadvertent subcutaneous injection of part or all of the vaccine. This has not been a problem at QSMI, but the staff of that institution is highly experienced.[64] Deliberate subcutaneous injections have been found to produce antibody titers just as high as intradermal injections.[125]

The fourth measure has been the development of reliable nonhuman cell-culture vaccines. Production of HDCV requires "a demanding technology, low virus yield, and enormously high production costs per unit of vaccine."[172] It is not suitable for large-scale human rabies prevention. A variety of nonhuman cell cultures have been used to produce rabies vaccines: primary cell cultures (hamster kidney, dog kidney, or chick embryo fibroblast), diploid cell lines (human or rhesus monkey), and continuous cell lines (Vero and baby hamster kidney cells). Those made according to FDA standards are considered as effective as HDCV for postexposure immunoprophylaxis and for intramuscular pre-exposure vaccination.[24,53,172]

PVRV is produced in cultures of kidney cells from African green monkeys by Sanofi Pasteur in France and by other agencies, and it is extensively used at QSMI and in other institutions.[64,92,182] This vaccine can be prepared in continuous cell lines cultured in suspension on microcarrier beads in biofermenters with a capacity of up to 1000 L, which makes possible the production of vaccine on an industrial scale at greatly reduced cost.[158,171,178]

The nonhuman cell-culture vaccines are less satisfactory than HDCV in one respect. Approximately one third of recipients reported pain at the injection site, and two thirds of vaccinees given PVRV boosters developed local erythema. About 5% to 10% of recipients had systemic reactions such as fever, headache, malaise, or urticaria.[171] In terms of safety and antigenicity, HDCV and the vaccines grown in other cell cultures are identical.

OTHER LYSSAVIRUSES

With RT-PCR, at least seven rabies virus genotypes have been recognized. The genotypes other than genotype 1 cause very few human infections. Genotype 2, Lagos bat virus, has not produced a recognized human infection. However, these genotypes are a source of considerable concern, particularly in Europe where bats infected by genotypes 5 and 6 are common. Treatment of humans infected by viruses of these genotypes with the immune serum and vaccine used for genotype 1 appears

effective, with the apparent exceptions of genotypes 2 and 3. These two genotypes are so different from the others that specific antisera can be prepared for them; identification of the other genotypes is based almost entirely on nucleotide sequence analyses. True vaccine failure occurs when animals vaccinated with rabies vaccines are exposed to Mokola and Lagos bat viruses.[62,85,100,152,179]

The following list is a brief description of the genotypes:

- Genotype 1 contains the viral strains that produce almost all recognized infections in humans and terrestrial mammals, many bat infections, and the fixed viral strains found in laboratories.
- Genotype 2 (Lagos bat virus) was first isolated from frugivorous bats on Lagos Island, Nigeria, in 1956. It is not known to infect humans, but it is found in bats and cats in Africa.
- Genotype 3 (Mokola virus) was isolated from mice in the Mokola district of Ibadan, Nigeria, in 1968. In 1971, it was recovered from a 6-year-old girl who had a nonlethal infection that was not clinically typical of rabies. Subsequently, a lethal human infection more typical of rabies has been recognized. This virus is found in shrews and cats in Africa.
- Genotype 4 (Duvenhage) was isolated in 1970 from a 31-year-old man with that name who died of the infection in Pretoria, South Africa, following a bite on the lip by a bat. It is found in insectivorous bats in Africa.
- Genotype 5 (European bat Lyssavirus, EBLV, types 1a and 1b) has produced lethal infection in two Russian girls, a 15-year-old bitten on her finger by a bat, and an 11-year-old bitten by a bat on her lip. It is found in insectivorous bats in much of Europe.
- Genotype 6 (EBLV types 2a and 2b) has produced lethal infections in two people, a 30-year-old male zoologist in Finland and a 55-year-old male bat handler in Scotland. It is also found in insectivorous bats throughout most of Europe.
- Genotype 7 (Australian bat Lyssavirus, ABLV) was isolated from a 39-year-old woman in 1996 and a 37-year-old woman in 1998 after both had died in Australia of rabieslike illnesses. It is found in insectivorous and frugivorous bats (flying foxes) in Australia. Bats with antibodies to this virus have also been found in the Philippine Islands.

Information about lyssaviruses continues to evolve, and two new genotypes have been reported: Aravan from a single bat in Kyrgyzstan, and Khujand from a bat in Takikistan.[3,109]

CURRENT RABIES DEVELOPMENTS

Rabies Eradication in Wild Animals

Attempts to destroy wild animals that provide a reservoir for rabies have been made but are expensive and, with the possible exception of a program to eliminate striped skunks in Alberta, Canada, have been ineffective. The increase in the population of animals, such as rodents, that are preyed on by the destroyed animals has also been unacceptable.[136,189] Eradication of rabies in wild animal populations by oral vaccination was first suggested by Baer, and a program using baits distributed by aircraft was initiated in Canada in the early 1980s. Foxes were the targets of the early studies because they are particularly susceptible to rabies, and an effective oral vaccine could be produced.

The most striking successes in vaccinating wild animal populations have been achieved in western Europe and in Texas. In Europe, the only significant wild reservoir for rabies is the red fox. A wave of fox rabies began on the Polish-Russian border after World War II and swept over most of central Europe.[26] The rising incidence of rabies in this species, and an economically distressing increasing number of secondary rabies infections in livestock, principally sheep and cattle, led to efforts to control that source of infection.

After extensive trials to demonstrate its safety, particularly for humans, vaccination of foxes was achieved with an oral attenuated live virus vaccine, starting in 1978 in areas of Switzerland, and later in parts of Germany. The vaccine first was placed in capsules attached to chicken heads; subsequently, a specially designed bait of fishmeal and fat, the "Tübingen fox bait," was developed. The baits included tetracycline as a marker, and fluorescence microscopy of mandible or canine tooth sections from foxes that were subsequently killed or found dead was used to determine the number of animals that had been immunized. Because dentine is deposited daily, the date of bait uptake could be calculated by counting the increment (von Ebner) lines between the pulp cavity and the tetracycline deposit.[103,104,174] Baits were uniformly distributed throughout the test areas by hand or by helicopter. Immunization campaigns were carried out in the spring and fall, the latter particularly to vaccinate young foxes that could not take the baits during the spring. Follow-up studies of tetracycline labeling indicated that 75% to 80% of the foxes had consumed the vaccine, an adequate number to interrupt the spread of infection within the fox population.

The attenuated live virus used for the initial vaccinations in Switzerland and Germany retains pathogenicity for rodents and can revert to virulence. In addition, this attenuated virus cannot be used in North America because it is pathogenic for striped skunks and ineffective for raccoons. Starting in 1990, a vaccinia-rabies recombinant vaccine that expressed the G glycoprotein was used in Belgium and France.[35,189] In the areas where the vaccine has been distributed, genotype 1 rabies has largely been eradicated in all terrestrial animals, not just foxes.[118] The incidence of rabies in livestock reported from the Belgium test area has fallen from more than 80 cases a year to none.[35,183] With subsequent modifications, this program has eradicated terrestrial rabies from eight European countries.[63]

An epizootic of rabies in raccoon dogs, which apparently had originated in the former USSR, was detected in southern Finland, and in the autumn of 1988, a field trial of oral immunization of these dogs and foxes was initiated. Rabies was eliminated from these populations within 12 months.[189]

An epizootic of rabies in coyotes in southern Texas originated in unvaccinated Mexican dogs and crossed the Rio Grande. The epizootic spread to domestic dogs and resulted in two human rabies infections and over 2000 postexposure treatments. Control by dropping baits containing oral rabies recombinant vaccine from aircraft has grown to the largest aerial vaccination program ever attempted (2.6 million vaccine–bait units over a 42,000-square-mile region during 1997). The rabies baits contain labels such as tetracycline. In 1997, 87% of coyotes manifested tetracycline fluorescence, more than enough of the population to stop the epizootic, which has disappeared. In 2001, only one infection by the dog/coyote rabies variant was found; in 2002, none were encountered.[108]

A similar program has been used in Ohio to stop the spread of the raccoon rabies epizootic from Pennsylvania on the state's eastern border. A single human vaccinia infection has been attributed to contact with the recombinant vaccine baits.[142]

Although such programs are expensive, they are considered less costly than treating humans, animal diagnosis, vaccination of domestic animals, compensation to farmers for culling infected livestock, culling wild foxes, and the salaries of the individuals conducting such programs.[35,118] Additionally, once rabies has been eliminated in a geographic area, it can be kept out by establishing buffer zones in which oral vaccination campaigns are carried out regularly. Vaccination campaigns for the entire area can be carried out less frequently or possibly eliminated entirely.[189]

In North America, control of rabies in wild animals is much more complex, because this infection is enzootic in a variety of animals: skunks, raccoons, foxes, and bats. These animals vary considerably in their sensitivity to different strains of the rabies virus and to the vaccines prepared from those strains, respond differently to the rabies virus sachet, have widely different ranges (9.5, 2.5, 1.5, 0.8, and 0.037 km^2 for coyotes, raccoons, red foxes, striped skunks, and mongooses, respectively), have widely varying population densities, and prefer different habitats.[110] Even though North America contains vast sparsely populated areas, many of the animals live in urban areas. A 60 km^2 (23.1-square-mile) area of Toronto with a perimeter of 28 km (17.4 miles) in which 252,000 humans lived, also had populations of approximately 1540 skunks, 3510 raccoons, and 70 red foxes from 1987 to 1991.[135] The entire city of Toronto is estimated to have a red fox population of approximately 1000 animals.[134] A program to trap, vaccinate by injection, and release animals in this area was effective but cost $27,000 to $69,000 (Canadian) a year. However, that sum was considerably less than the $100,000 (Canadian) spent annually to treat humans exposed to rabid animals in Toronto.[135]

Although these programs control rabies in the target species, the ecologic effects of increasing the animal population by eliminating a significant cause of mortality are almost entirely unknown.[26,174]

Vaccine Developments

Killed virus vaccines are currently used for human and most domestic animal vaccinations. Attenuated live virus vaccines have been used only to vaccinate animals. The first vaccine used to orally vaccinate wild red foxes in Europe was an attenuated live virus vaccine, and that vaccine is being used in Canada.

Subunit vaccines contain only one of the viral structural proteins. Because these vaccines contain only an antigenic portion of the virus and not the entire organism, they can induce immunity but are incapable of producing infection. The rabies-vaccinia recombinant that produces only the G surface protein of the rabies virus has proven to be noninfective, immunogenic, and very effective for vaccinating foxes in Europe.[189] However, this recombinant cannot be used for humans because much of the world's population has been vaccinated with vaccinia to prevent smallpox, and vaccinia can produce disseminated infections in humans.[35]

Some orthopoxviruses cannot completely replicate in mammalian cells, and therefore cannot produce infections, but can abortively replicate so that proteins expressed by the virus are presented to the immune system. A recombinant with canarypox virus that expresses the G glycoprotein has been developed at Sanofi Pasteur and has been found to be safe and to effectively produce a neutralizing antibody response in a variety of animals and humans. This vaccine is currently licensed for vaccinating cats in the United States.[34] Orthopox vectoring may permit the incorporation of several antigens, such as measles, mumps, rubella, rabies, and pertussis, in a single vaccine, which would greatly facilitate and reduce the cost of human immunization.[189]

Adenoviruses replicate on mucosal surfaces and could be ideal vectors for oral and intranasal vaccines. Recombinant human adenovirus type 5, into which complementary DNA for the G protein has been inserted, has been shown to elicit protective levels of neutralizing antibodies in skunks and foxes when administered orally. Deleted, replication-defective recombinants have produced high titers of neutralizing antibody in dogs.[69]

DNA vaccines based on plasmid vectors expressing the rabies G protein offer promise because they are easy to construct, manipulate, and produce. Such vaccines have produced high viral neutralizing antibody titers in mice, dogs, and nonhuman primates. They appear to be effective in much younger animals. DNA vaccines have not proven as effective for postexposure therapy, apparently because the antibody response to such vaccines is slow. Efforts to accelerate the antibody response appear to be effective.[34,69]

The references for this chapter can be found on the accompanying DVD-ROM.

55 Emergency Veterinary Medicine

Murray E. Fowler

The wilderness experience often involves animals other than humans. Such domestic animals as horses, mules, burros, llamas, elephants, camels, and yaks may be used for packing or pulling supplies, relieving hikers of this burden. Horses and mules are also ridden by trekkers. Dogs may accompany owners into the wilderness as companions, as trackers of game, or in the Arctic and Antarctic as primary draft animals. A cat may accompany a trek as a mascot or as a companion animal. Any of these animals may become injured or fatigued or may fall ill with a variety of ailments that require emergency treatment and management by trek personnel.

WILD ANIMAL ENCOUNTERS

Observation of wild animals in their natural habitat enhances the wilderness experience, but encounters between wild animals and animals accompanying the trekkers may be less than pleasant. Healthy wild animals usually try to avoid humans, but this may not carry over to domestic animals.

Medical and paramedical personnel who may be called to render emergency medical care or assist in the rescue of injured or diseased humans or support animals should recognize that wild animals may be involved. Experienced trekkers can usually recognize when they are intruding into an animal's domain. Unfortunately, some people lack a sense of courtesy for animals. When trekkers in the wilderness behave as if they are entering the home of a human friend, they show appreciation for the rights of wild animals and have less risk of injury or illness to themselves or their support animals. Understanding the biology and normal behavior of wild animals known to live in the wilderness areas to be visited enhances enjoyment and diminishes exposure to potentially dangerous situations.

Others who may be at risk are wildlife biologists, hunters, and people who choose to live in remote areas. Hunters come into intimate contact with wild animals as they pursue, dress, and eat game. Plant scientists, paleontologists, geologists, and other non–animal scientists may inadvertently encounter wild animals. Wild-animal biologists conducting field investigations may capture or immobilize wild animals to collect data. Such individuals are at potential risk for injury or disease, not only as a result of wild animal contact but also from tranquiliging drugs and the firearms used to administer the drugs.

Why and When Wild Animals Are Dangerous

Few wild animals deliberately stalk humans, but they may stalk support animals. Most wild animals fear humans and, given an opportunity, avoid human contact. Injuries to humans caused by wild animals are usually the result of judgment errors (such as approaching too closely), ill-advised handling of diseased wild animals, or unlucky exposure to highly aggressive or protective animals (see Chapter 51). Such animals may be natural predators seeking food, prey species fearful for their lives or the lives of their young, or diseased and irrational animals (such as those with rabies). Attacks on support animals usually occur at night or when humans are absent.

Animals are most likely to respond adversely if startled from sleep or if their flight distance is violated. Flight distance is that distance at which, if approached by an enemy, an animal will either fight (attack) or flee. The distance is species specific and regionally specific, and it may become greater if animals in the area have been recently hunted. Flight distance may be greater for a person walking or carrying a gun than for a person riding on horseback or in a vehicle. Animals that find themselves cornered occasionally attack or risk injury trying to escape. Any animal may attack if its offspring are in danger. With any large animal, accidental or intentional positioning between a mother and her infant invites attack.

Equal danger exists for a person who comes between a territorial male and one or more females. Many wild animals are territorial. Males and females may establish feeding, breeding, and home territories. An animal may share feeding territories with other members of the same species or with different species, but home territories may be defended against any intruder, including humans.

Injuries have been inflicted on hunters who approached a recently shot animal, believing it to be dead, only to have the animal revive explosively in a final Herculean effort to fight or flee.

Injury Prevention

Humans should not fraternize with wild animals. Food scraps should not be left to tempt wild animals closer for photographs. Any wild animal that will allow touching is a spoiled park animal or is diseased. Neither type should be hand fed. Travelers in the wild should be observant for animal signs (such as feces, tracks, trails, and resting areas) and should respect the behavioral characteristics of animals. Knowledge of the biology and habits of local animals obviates most avoidable close encounters and dangerous situations.

Dealing with an Attack

Wild animal attacks on support animals usually occur when the animal is tied on a picket line or staked out for grazing. The

animal may injure itself trying to escape the attack or may be bitten and mauled by its attacker. In one example, a frightened llama broke loose from a stake, but the lead rope suddenly caught between rocks and the animal's cervical spine was fractured by the abrupt stop. Wounds inflicted on humans may be lacerations, contusions, punctures, or abrasions. Specific therapies are addressed in Chapter 51.

SUPPORT ANIMALS

Expedition leaders and participants should consider the well-being of support animals and understand their physical capabilities with regard to maximal load, speed, and endurance. Humans should also be able to recognize signs of exertional stress, which is more likely to occur in horses that are ridden than in pack-horses that are led. A horse can generally match or exceed the physical endurance of a hiker, but adverse metabolic conditions may develop in horses pushed beyond their limits of endurance. The syndrome that arises from excessive exercise depends on intensity and duration, degree of prior conditioning, and nutritional status. Mules, donkeys, and llamas are less prone than horses to develop these problems, because they usually refuse to be pushed to extremes of exertion.

The animal best suited to the terrain, climate, and task to be performed should be selected. Horses, mules, burros (donkeys), and llamas are traditional beasts of burden in North America, wherever sufficient forage and water are available. Each animal has both admirable and undesirable qualities. Horses are usually the largest and can carry the heaviest loads. Mules and burros tend to be more difficult for novices to handle. Llamas have also become popular support animals with backpackers in North America. The llama requires less feed, can subsist better on sparse native forages, and is less damaging to the environment than is the horse. No animal should be used on a trek unless at least one member of the party has considerable experience in handling and caring for the chosen animal.

In South America, the llama is the beast of burden. Adventurers traveling into rugged and remote areas above 3000 m will probably use llamas for support.

Dogs are necessary draft animals for winter travel in the Arctic. Some hikers place small packs on dogs to carry light items or food for the dog. In other regions, dogs function more as companions. Cats must be placed in an appropriate carrying container, such as an airline shipping container, during transport, because a cat walking on its own would not be able to keep up.

Many expeditions are mounted in countries unfamiliar to both leaders and participants. In various parts of Asia, the horse is replaced by the elephant, water buffalo, yak oxen, or Bactrian camel. In North Africa, the Middle East, and Australia, the dromedary camel carries the load. In Australia, horses may be used as well. Each species has specialized requirements for handling, packing, and health care.

Expedition physicians may be asked to treat the ailments of support animals. Basic medical training provides the foundation necessary to diagnose and treat many conditions that may be encountered on the trail. This chapter provides an overview and specific anatomic and therapeutic information for some common problems. If in doubt, expedition members should handle an ailing support animal as if it were a human. When support animals carry all or part of the load, there is often capacity for a more sophisticated emergency medical kit to be carried.

Pre-Trip Animal Health Considerations

Treks involving support animals are carried out worldwide. Often, animals are supplied by a person or firm specializing in a given type of animal. The animals may be leased directly to the trek leader, or a handler may be contracted to supply and care for the animals. In either case, it is the responsibility of the trek leader to ensure that the animals meet the requirements necessary for the trek. This is particularly true in foreign countries.

The author accompanied a trek in the high Andes in Peru. The trek leader had made arrangements for a certain number of llamas to carry the gear. The requested number of llamas were present, but many were small, thin, and immature. Most of the animals had never carried a pack before and bucked and kicked every time they were turned loose with the pack on. The trek leader had failed to specify details on what was necessary to control the animals.

Any trek leader who is not animal savvy should ask for help from someone with animal handling or veterinary experience. Topics that should be covered include conditioning, foot care, and required immunoprophylaxis.

Health certificates are required for travel and entry into the state or province of destination and possibly intervening states to be traversed while in North America. Regulations are extremely variable, so inquiries should be made at least 4 weeks before departure. It is recommended that each animal receive a physical examination by a qualified veterinarian before an extended trip. Conditioning is as important for trek animals as it is for human participants. Training should be in terrain similar to that expected on the trek, with special attention given to toughening the footpads of llamas and dogs by appropriate exercise. The expected terrain to be encountered on the trek should be communicated to the animal supplier well in advance of the trek.

Immature animals should not be taken on expeditions. Dogs should be at least 1 year old. Llamas and horses should be older than 3 years. Well-conditioned and trained horses, mules, burros, and dogs can carry approximately 30% of their body weight. Llamas usually carry only 25% of their body weight (Table 55-1).

Frequently, arrangements are made to contract for support animal service from suppliers in the trek locale. Persons providing these services do not always understand the trek requirements and may supply animals that are poorly shod, unconditioned, poorly trained, or otherwise unsuitable for the needs of trek participants. Trek leaders must clearly stipulate specifications well in advance of the trek and be adamant about compliance.

Horses, Mules, and Donkeys

A tetanus booster should have been given within the past year. Vaccination against rabies is appropriate if the trek itinerary includes travel in an endemic area. Encephalomyelitis vaccine should be used in endemic areas, especially during the insect seasons of summer and fall. Internal parasite levels should be evaluated by fecal flotation and appropriate medication given to reduce the parasite burden.

The feet should be trimmed and shod properly at least 2 weeks and not more than 4 weeks before a trek begins. Sole

TABLE 55-1. Vital Statistics of Trek Animals

ANIMAL	*BODY WEIGHT* lb	*BODY WEIGHT* kg	HEART RATE (beats/min)	RESPIRATORY RATE (breaths/min)	*BODY TEMPERATURE* °C	*BODY TEMPERATURE* °F	*WEIGHT CARRIED BY WELL-CONDITIONED ANIMAL** kg	*WEIGHT CARRIED BY WELL-CONDITIONED ANIMAL** lb
Horse	800–1200	360–540	28–40	10–14	37.2–38.0	99.0–100.5	110–136	240–300
Mule	600–1200	275–540	28–40	10–14	37.2–38.0	99.0–100.5	82–136	180–300
Donkey	300–600	136–275	28–40	10–14	37.2–38.0	99.0–100.5	40–82	90–180
Llama	300–450	136–200	60–90	10–30	37.2–38.7	99.0–101.8	34–50	75–110
Dog	20–100	9–45	65–90	15–30	37.5–38.6	99.5–101.5	3–14	6–30
Camel	880–1200	400–550	40–50	5–12	36.4–42.0	97.5–107.6	225	500
Elephant	5000–8000	2300–3700	25–35	4–6	36.0–37.0	97.5–99.0	900	2000
Yak	2200	1000	55–80	10–30	37.8–39.2	100.0–102.5	235	550

*Sustained trekking for 24 to 40 km (15 to 25 miles) per day on moderately difficult trails. The weight includes tack. Animals in training should be expected to carry only one half to two thirds of this weight.

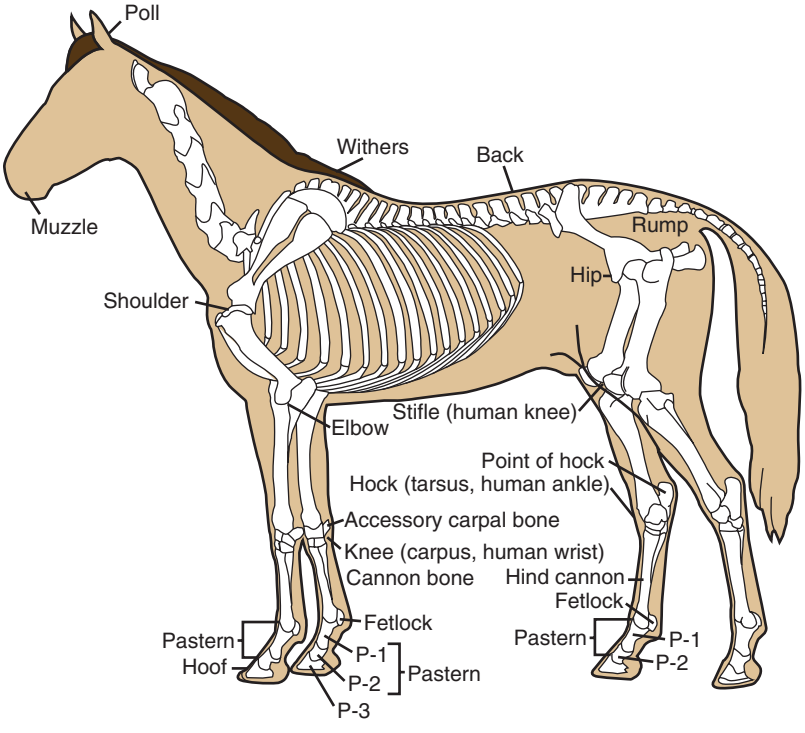

Figure 55-1. Anatomic sites of the horse.

pads should be affixed if sharp rocky terrain is expected. Extra shoes for fore and hind feet of the different-sized horses should be carried, along with the appropriate nails and hammer to reattach a shoe, should one be cast.

Selecting the breed of horse for a trek may depend on the experience and horsemanship of the trekkers. Horses for riding should be selected for gentleness, ease of mounting, and conformation that fosters a smooth gait and comfortable ride. Correct conformation includes angulation of the joints and a long, sloping pastern that provide good cushioning when a foot strikes the ground. Certain breeds are well known for this, including the Tennessee walking horse and the Missouri fox-trotter. Arabian horses are known for their stamina and endurance but may be high-strung and require more expertise on the part of the rider than do other breeds. Thoroughbred or thoroughbred crosses are excellent if they are not flighty or too tall (16 hands or 64 inches or more). Quarter horses are also good trail horses unless they have short, upright pasterns that make for a jolting ride (Fig. 55-1). Often, there are more differences within a given breed of horse than between breeds. Avoid typical riding-academy horses that have to be prodded to a gait faster than a slow walk.

The same poor conformation that produces a rough ride may also foster trauma to joints and ligaments that can lead to lameness and unsoundness. Trek leaders are not likely to have a significant choice in animals used as packers. It is important to have sound horses that are properly trained and that are conditioned to accommodate packs.

Llamas

Llamas should have been given a tetanus toxoid booster within the past 6 months. Other basic immunizations should include *Clostridium perfringens* toxoid, types C and D, within the past 6 months, and vaccinations against leptospirosis and rabies if entering an endemic area.

Toenails should have been trimmed within the previous 2 months. Internal parasitism is debilitating in llamas. Ova levels should be checked, but usually treatment with an anthelmintic (ivermectin, 0.2 mg/kg subcutaneously, or fenbendazole, 5 mg/kg orally) is desirable within the previous 2 months.

Camels

Camels are commonly the beasts of burden for baggage and people in the desert regions of Australia, the Middle East, North Africa, and parts of Asia and Southeast Asia. Camels are excellent trek animals but are considered stubborn, obstreperous, and difficult companions by persons who do not understand them. Trekkers should not use camels unless a qualified handler accompanies the trek.

Although camels are domestic animals and quite docile when trained properly, everyone on a camel excursion should be able to recognize their offensive and defensive behaviors. They can kick forward, backward, and to the side. A dromedary camel can scratch its ear with a hind foot, so be aware of the arc of the limb. Camels also bite. They have formidable canine teeth that can inflict considerable damage. Camels, like llamas, are known to spit when they are angry. The spit is composed of regurgitated, foul-smelling stomach contents.

Camels used to carry people or baggage are trained to lie down for boarding and loading. This is also the position that the cameleer should use to inspect for external parasites, administer injections, collect blood, or administer intravenous medication.

Dogs

Dogs are routinely immunized against canine distemper, canine adenovirus, leptospirosis, and rabies. Vaccinations should be current. A check should be made for internal parasites and fleas, so that appropriate action may be taken. Although dog fleas do not permanently infest humans, they bite and may cause mild to severe local dermal reactions in sensitive individuals. Dogs should be bathed with a shampoo containing a pyrethrin insecticide before a trek. Any pyrethrin-containing dusting powder may be carried on an extended trek to relieve dogs of their flea burden. It is unlikely that the fleas involved in the sylvatic plague cycle will infest a dog, so the dog is not a source of plague infections for humans in a trek situation.

Cats

Like other animals on a trek, cats should have a pre-trip physical examination, with special emphasis on fleas in the hair coat and *Toxoplasma gondii* oocysts or ascarid ova in the feces.

Vaccinations, including protection against rhinotracheitis, feline calicivirus, and feline panleukopenia, should be current. If the trip will be to or through a rabies-endemic area, rabies vaccination should be current. Although housecats may be vaccinated against other agents, these are the only diseases that are of significance to trekkers.

Rest

Support animals require less sleep than do human trekkers. Horses can sleep while standing because they have a special locking mechanism of the tendons and ligaments of the limbs. Horses may also lie in sternal or lateral recumbency. Llamas usually rest in a sternal position but may also assume lateral recumbency.

Llamas enjoy taking a dust bath by rolling in dirt soon after packs are removed. A few may even try to roll with the pack in place.

Watering and Feeding

All animals require daily access to potable water. It is not practical to filter or disinfect water for large animals, but this may be considered in extremely filthy conditions. Dogs may be susceptible to giardiasis, and, theoretically, it is desirable to provide filtered water for them. However, preventing a free-running dog from drinking from a stream or lake is virtually impossible. Heavily mineralized or silted water may be as unpalatable for animals as for humans and may cause similar gastrointestinal upsets.

The basic fluid requirement for a horse or other large trek animal is approximately 40 mL/kg/day, or 18 L (5 gal) for a 450-kg (1000-lb) horse. With work and heavy sweating, the requirement may be tripled. Horses sweat profusely and should be given the opportunity to drink along the trail. Llamas and burros may require one-third less water than a horse. However, although they tolerate dehydration better than do other species, they function better if well hydrated. Some llamas refuse to drink until evening. The camel is noted for its ability to tolerate a number of days (2 to 7) without drinking water.

Feeding Horses, Mules, and Burros

Horses consume approximately 2% of their body weight daily (or 20 lb of total feed for a 1000-lb horse). When the animals are working hard, one third of this amount should be concentrates (grains such as rolled oats, barley, or cracked corn, or mixed grains and molasses in loose or pellet form). If forage is unavailable, alfalfa pellets should be carried. Animals should become accustomed to eating pellets before the journey begins. Concentrates should be reduced on days when the animals are not working.

Feeding Llamas and Camels

Llamas require only 1.0% to 1.5% of their body weight in food daily. Given the opportunity, llamas will graze and browse to satisfy their requirements. Many packers carry a supplement mixture consisting of half alfalfa pellets and half grain mix (the same as for horses), used as required. Some llamas are reluctant and must be trained to eat pellets or grain. Training should take place at home, prior to the trek. Camels will graze or browse on any forage that is available. Grain supplementation is not necessary.

Feeding Dogs and Cats

A dog should be accustomed to high-quality dry dog food. The quantity fed should increase by half when the dog is exercising. A cat should be accustomed to eating dry cat food. Avoid feeding scraps from trekker's food.

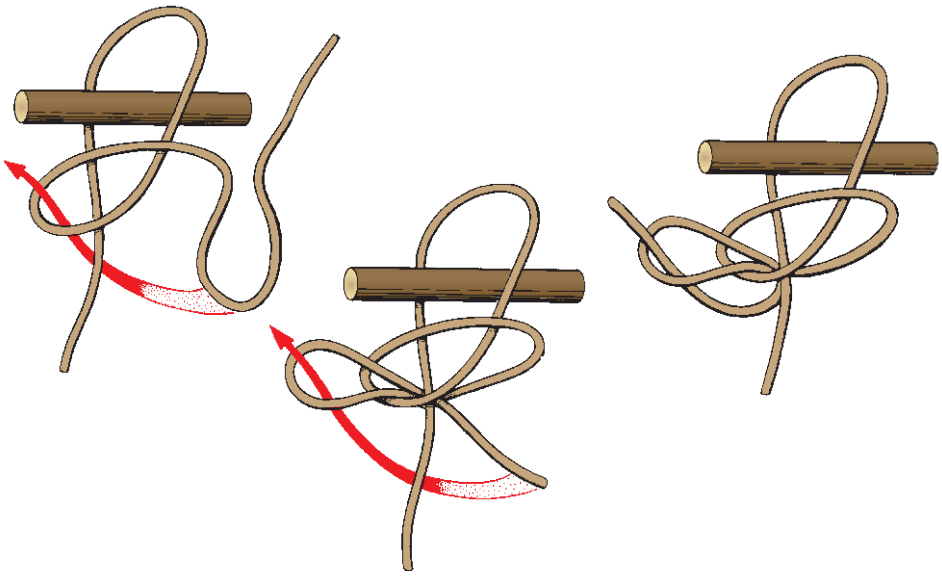

Figure 55-2. Sequence of steps to create a halter tie.

Emergency Restraint

It is assumed that one or (preferably) more persons on the trek are acquainted with the general care and handling of any domestic species involved in the expedition. Methods of haltering and leading are specific for each animal. Securing animals at night may require hobbling or tethering. Skill and experience are necessary to accomplish this without risking injury to the animal or handler. Animal handlers should be able to examine and clean the feet and hoofs of their charges. All people who deal with the animals should know how to create a halter tie (Fig. 55-2), so that a safe and secure tie can be made at rest stops or whenever an animal is to be tethered. Knowledge of temporary rope halter construction is desirable in the event of loss or breakage of halters (Fig. 55-3).[10]

Horses, Mules, and Burros

Large animals may inflict serious or lethal injuries on people. When in pain or panicked, they may not respond even to their customary handlers. If a horse is down and entangled in rope, wire, or bushes, it should be approached from its back and its head held down until it can be extricated. The handler should stay out of the reach of both fore and hind limbs.[10]

A horse's defensive and offensive actions are to strike with the forelimbs, kick forward and backward with the rear limbs, and bite. The safest place to stand is close to the left shoulder. A person examining the feet and legs of a standing horse should keep his or her head above the lower body line to avoid having the horse reach forward with a rear limb and strike it.

Additional restraint for painful procedures may be accomplished by grasping one or both ears of the horse. The method of "earing" a horse is as follows: Stand at the left shoulder and grasp the halter or lead rope with the left hand. Place the right hand palm down with the fingers together and the thumb extended, on the top of the neck. Slide the hand up the neck until the thumb and fingers surround the base of the ear (Fig. 55-4). Squeeze tightly, but do not twist the ear. The horse usually tries to pull away as the ear is grasped. Be prepared to move with the horse while maintaining a firm grip.

Llamas

Pack llamas are usually docile. Although most allow their feet to be lifted for inspection or treatment, some try to lie down. One or two people should stand on the side opposite the limb being lifted, or the llama should be placed next to a tree or large rock to prevent the animal from moving away. The limb should be firmly grasped. It may be necessary to provide semi-support to the body if the animal tries to lie down.[11]

If a llama refuses to get up, the rear limbs may be pulled out behind it. If it still refuses to rise, an injury or illness that inhibits rising should be suspected.

Only rarely does a llama "spit" against an annoyance. The spit is actually stomach contents, the foul odor of which remains until thoroughly washed off. Spitting is usually directed toward other llamas to express displeasure, but handlers may be caught in the crossfire. If a llama becomes irritated during an examination or treatment procedure, spitting can be controlled by draping a cloth over the nose and tucking the top around the nose piece of the halter. Llamas also dislike the odor of stomach contents on their noses. Llamas can be "eared" in a manner similar to that used for horses.[10]

Dogs and Cats

Dogs usually accompany their owners, who are able to handle them under difficult circumstances. If mildly painful medical procedures must be performed, the head and mouth should be secured. The dog's body can be securely held against the handler's body by reaching across the back of the dog and grasping the base of the neck while pulling the opposite shoulder with the elbow toward the handler. The other hand should tuck the dog's head under the handler's arm.

Alternatively, a muzzle can be constructed from a nylon cord or even a shoelace. A loop should be formed with an overhand knot on one side. The loop is placed over the muzzle of the dog, with the knot on top, and tightened (Fig. 55-5). The ends of the loop should be wrapped around the muzzle, crossed beneath the jaw, and tied behind the ears (Fig. 55-6).

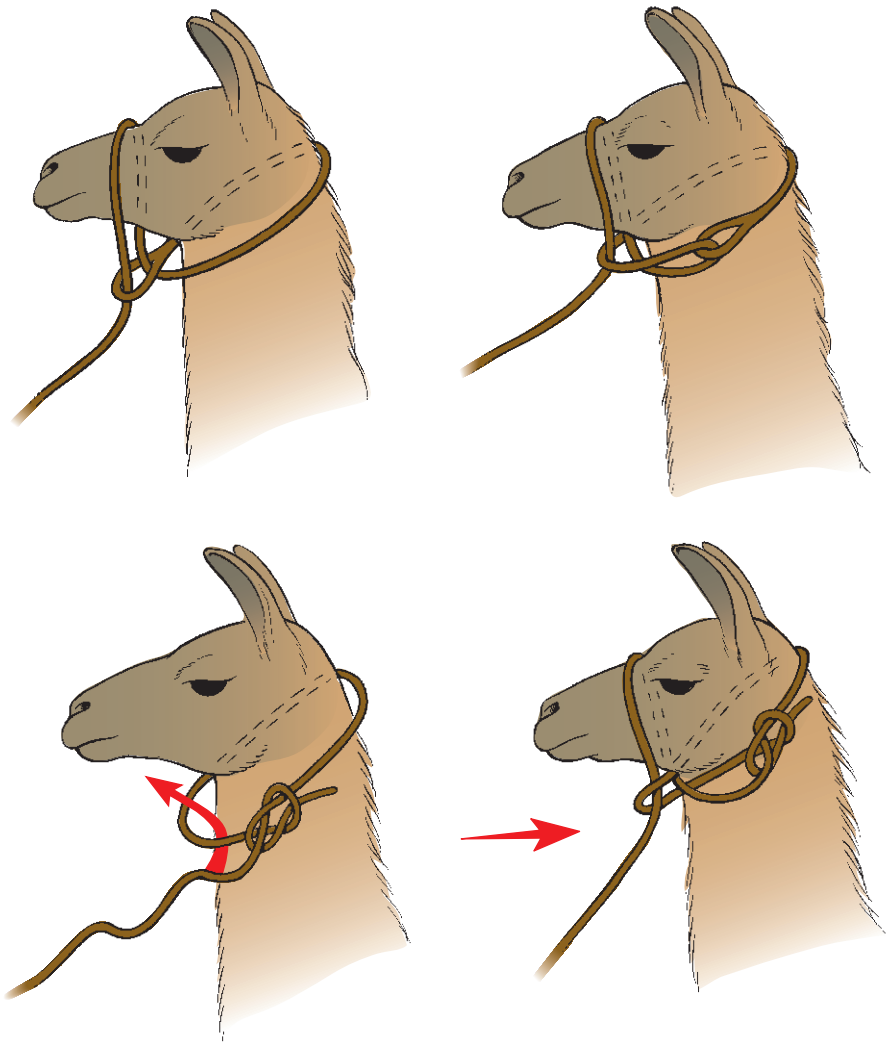

Figure 55-3. Temporary rope halters.

Figure 55-4. Earing a horse.

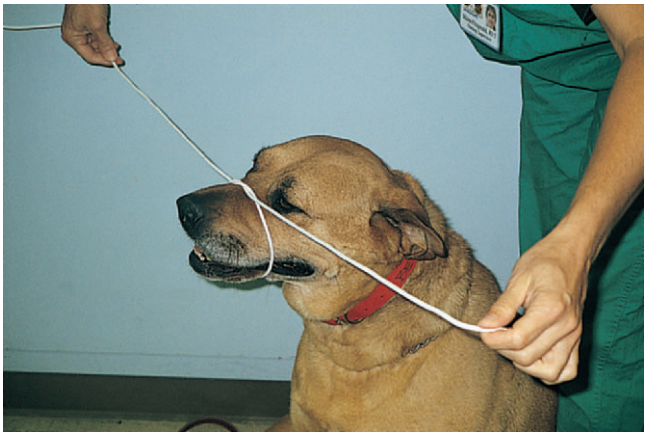

Figure 55-5. Placing a muzzle on a dog.

Figure 55-6. Completed muzzle on a dog.

Cat restraint is accomplished by wrapping the cat in a towel. Grasp the cat by the nape of the neck close to the head. Place the cat on an outstretched towel and press down on the back while another person begins to roll one end of the towel over the cat and continues to roll the cat into the towel. A limb may be withdrawn from the towel roll. Chemical immobilization of a cat is easily accomplished using ketamine HCl at a dose of 10 to 20 mg/kg administered intramuscularly or subcutaneously.

Intramuscular injections are administered in the upper rear limb. For subcutaneous injections, grasp a fold of skin on the dorsal aspect of the neck just in front of the shoulders. Lift the skin and inject beneath the tented skin.

Conditions Common to All Species

Support animals may injure each other or their human handlers, or may spread disease (such as ringworm or lice). Human injuries are discussed in Chapter 51.

Trauma

Contusions, abrasions, and lacerations are the most common ailments encountered in support animals. Falls, stumbling on sharp rocks, brushing against branches, and encounters with other animals are frequent causes of injury. Such wounds should be handled like similar human injuries. Hair or wool should be trimmed from the margins of wounds before treatment or suturing to prevent matting with exudate. The skin may be sutured with any suture material suitable for humans. Antibiotics are not necessary unless vital structures, such as synovial or serosal membranes, are exposed.

Rope burns may occur in all species, but they are more likely to occur in horses because they tend to struggle against entanglements, and their skin is quickly burned by abrasive action. Therapy is similar to that for human burns. Thermal burns are unlikely unless animals are caught in brush or forest fires.

Foot, Hoof, and Nail Problems

Foot injuries may incapacitate an animal and possibly the expedition.[6,11] The hoof of the horse covers the distal extremity and the third phalanx (P-3). The specialized horn of the hoof and nail is roughly analogous to the human nail. Horses are digitigrade, walking on the tip of P-3 (see Figure 55-1). In llamas, P-2 and P-3 lie in a horizontal plane within the foot, with only P-1 in the vertical position. The nails of the llama and dog are inconsequential in weight bearing, but if torn or contused are extremely painful.

The weight-bearing surface of the horse's hoof contains the firm sole and more flexible frog, overlying soft fibroelastic tissue called the digital cushion. Dogs have footpads similar to those of llamas.

The structures of the foot are subject to contusions, abrasions, lacerations, and penetration by foreign bodies (such as nails, stones, and sticks). Segments of the hoof wall and nail may be avulsed, exposing the sensitive laminae. Infection may invade the foot and undermine the outer layers, with abscess formation. Not all lesions are visible. Stone bruises of the sole or frog of a horse may be evident only when pressure is exerted on the site of the contusion. Foreign body penetrations and abscesses may cause reluctance to place any weight on the limb. The penetrating object may have been withdrawn or still be in place; in either case, a discolored tract leads to the depth of the wound. Erosions and ulcerations of the footpads are common in unconditioned dogs and llamas. Dogs working in ice, snow, mud, and water may suffer from maceration, cracks, erosions, lacerations, and frostbite.

Foot and limb trauma is accompanied by varying degrees of lameness (limping). It may be difficult to establish which leg is painful, but the principles are similar to evaluation of such pain in humans, with the obvious differences of two extra limbs to evaluate and the animal's inability to communicate.

Cellulitis may develop on the limbs or body. The signs include heat, swelling, and redness and are the same in all species, as is therapy.

Therapy for foot injuries includes providing drainage of infected lesions, disinfection, and protection of exposed sensitive structures. Antibiotics are not indicated for most wounds unless a joint surface is exposed. It may be necessary to bandage the foot to provide protection while in camp, and to fashion special shoes or boots to keep an animal functioning on the trail. Special booties are available commercially for dogs, but a temporary moccasin can be constructed from soft leather (such as the leather used by craftspeople to make moccasins).

Hyperthermia (Heat Stress, Heat Exhaustion)

Hyperthermia is elevation of the core body temperature above normal limits. Chapters 10 and 11 discuss hyperthermia in humans, and it is important to recognize that animals associated with a trek may become overheated as well.[11,12]

Etiology. The primary cause of hyperthermia in pack animals on treks is excessive muscular exertion, especially during periods of high environmental temperatures and high humidity. Animals that struggle for prolonged periods in swampy terrain or in snow or mud may also produce excessive heat. Although high ambient temperature is a contributing factor, excessive muscle exertion in any climate may cause elevation of body temperature. Pack animals may also develop fever as a result of infectious diseases. Dehydration from inadequate supplies of potable water exacerbates the problem.

Inadequate physical conditioning is a common problem and contributes to a cumulative heat load. Other contributing factors include lack of salt in the diet, preexisting cardiovascular insufficiency, obesity, and trauma.

Clinical Signs. Signs vary according to species and the stage of hyperthermia, but all affected animals have increased heart and respiratory rates, usually accompanied by open-mouth breathing. Rectal temperatures may vary from 106° to 110°F (41.1° to 43.3°C). Horses, mules, burros, and llamas sweat in the early stages of hyperthermia, but sweating may cease if the animal becomes severely dehydrated. Sweating is evident in horses but imperceptible in llamas, because most sweating in llamas occurs on the ventral abdomen in what is known as the thermal window, where the fibers are less dense and the fiber length is short.

Dogs cool themselves by evaporation of respiratory fluids while panting. The mouth is held open and the tongue lolls from the mouth. The respiratory rate increases from a normal of 30 breaths/min to 200 to 400 breaths/min. Moisture may be observed dripping from the tongue. As dehydration intensifies, salivation and dripping may slow or cease.

Hyperthermia causes a shift of blood volume from the viscera and muscles to the skin, resulting in hypovolemia and varying degrees of hypotension. Hypotension causes hypoxemia of the brain, resulting in dullness, restlessness, and incoordination. Hypoxemia may lead to convulsions and collapse. The shift of blood from the gastrointestinal tract may cause decreased motility and the potential for ileus and tympany. Signs of colic in horses and llamas (kicking at the belly, looking back at the side, treading, attempting to lie down and roll) may be noted.

A 1.8°F (1°C) rise in body temperature requires 10% more oxygen for proper function of the energy systems of the body. When body temperature reaches 105.8°F (41°F), the respiratory system is no longer able to supply sufficient oxygen by normal respiration. Respiratory acidosis and electrolyte imbalances associated with sweating may produce a syndrome similar to septicemia.

Hyperthermia may affect most of the organ systems of the body. The severity of the syndrome cannot be assessed in the field. It is important to recognize the conditions that lead to hyperthermia, to watch for early signs of heat stress, and to stop any activity that contributes to the problem. Mules and burros are not likely to become severely affected because they refuse to go on if stressed. Horses may be driven past their endurance and die of thermal stress.

Treatment. Cessation of excessive muscular activity may be all that is necessary if hyperthermia is mild. If streams or lakes are nearby, the animal can be walked into the water and water can be splashed on its underbelly. Contingencies for hyperthermia are part of all plans for capture operations for wildlife translocation and reintroduction projects. Water is carried for cooling, and intravenous fluids to deal with heat stress. Cold water enemas are the most effective and rapid way to cool the body of a large animal.

External Parasites

The order Diptera, class Insecta includes thousands of species that are annoying or parasitic to humans and domestic and wild animals (see Chapter 44). Trek animals suffer as do their human companions. Certain destinations are notorious for insect swarms (flies in Australia, mosquitos and gnats in the Arctic summer). Dipterans are also important disseminators of infectious microorganisms. It is sometimes difficult to differentiate venomous from parasitic species. The saliva of hematogenous dipterans may contain a toxin that aids in altering the endothelium of capillaries to induce free flow of blood, which is then ingested by the fly. Many humans are extremely sensitive to bites from certain flies and some are sensitive to the toxins in the saliva.

Animals may also be sensitive to the toxins in the saliva. Even nonbiting species, such as the housefly *(Musca domestica)* or the face fly *(Musca autumnalis)*, feeding on lacrimal secretions or open wounds, may produce a local reaction causing blepharitis, conjunctivitis, and keratitis. Biting flies (mosquitos, gnats, and midges) may produce local reactions in animals. An attack by immense numbers of simulid gnats may cause debility and even death in animals. In the Mississippi valley, horses and mules were killed by massive attacks of the buffalo gnat *(Cnephia pecuarium)*. Swarms of biting insects may cause alarming behavioral responses in horses, including crowding together, head shaking, tail switching, and running. As a youngster, the author was mowing alfalfa hay using a team of horses. A cloud of simulid gnats descended on them. It was difficult to control the horses from bolting and at the same time protect oneself from the biting insects.[8]

Bee Stings. Most of the aculeate Hymenoptera group of insects have a stinger apparatus, used to paralyze prey species or for defense. Most are small or lack the power to thrust the stinger through vertebrate skin and thus are of no consequence in envenoming people or animals. Other species—bees, wasps, yellow jackets, and hornets—inflict, at the least, a transient, painful sting and sometimes kill animals and people.

The honey bee *(Apis mellifera)* is one of two domesticated insect species (the other is the silk worm, *Bombyx mori*). Native honey bees in tropical Southeast Asia were the original source of the North American domestic honey bee. When early American colonists missed honey as a sweetener, they imported European strains of the honey bee to Jamestown, Virginia, and the bees became known as European honey bees. The aggressive African honey bee *Apis mellifera adamsonii,* a native of tropical southern Africa, was introduced into Brazil in 1956 in the hope of improving the local European strain's ability to cope with warm weather. The bees readily adapted, but swarms escaped and have been moving northward at a rate of 150 to 200 miles per year. They are now found in California. The African honey bee hybridizes with the domestic honey bee, from which it can be distinguished only by apicultural entomologists.

A sting from a single insect is not likely to cause a problem in a horse, llama, or dog. The classic anaphylactic response of humans has not been documented in livestock or companion animals. A local reaction consists of a swollen, edematous, and erythematous plaque. A tiny abscess may form at the injection site. Multiple stings may produce numerous urticarial wheals and cause a severe systemic response as a direct toxic effect of the venom.

The pain associated with multiple stings may cause excitement or frenzy in a horse, followed by tachycardia, diarrhea, hemoglobinuria, icterus, and prostration. If the stings are on the head, or near the nostrils or mouth, swelling may occlude air passages, causing dyspnea.

Dogs may yelp at the initial sting. Cats are less likely to vocalize. Most victims of single stings are presented to the veterinarian with facial, aural, or periorbital angioedema. If a dog snaps at a bee or wasp and the sting occurs in the oral or pharyngeal cavity, swelling may occlude the passageway, resulting in severe dyspnea. Multiple bee stings may result in

cardiovascular and respiratory collapse with death ensuing in minutes.

As soon as the injection site has been identified, the area should be inspected for the remains of the stinger. The stinger of the honey bee has retrograde barbs that oppose extraction of the stinger, causing the caudal insect abdomen to be torn away. A honey bee is thus unable to inflict multiple stings, as do wasps and hornets, which do not have barbed stingers. A retained stinger should be removed by the most rapid method available.

Tick Bites. Members of the order Acarina, class Arachnida (ticks, mites, spiders, and scorpions) pose minimal risk for horses and llamas but may incapacitate dogs. Envenomation should be managed as for a human. Ticks are known to transmit a variety of infectious disease agents, including *Borrelia burgdorferi* (borreliosis, Lyme disease) and *Ehrlichia risticii* (Potomac horse fever, ehrlichiosis) in horses, but the biggest hazard of tick infestation in trek animals is tick paralysis.[8] Tick paralysis occurs in horses and llamas and many species of mammals and birds in North America, Europe, Africa, Australia, and Russia. Usually, only female ticks produce the neurotoxin, which localizes in saliva and is injected into the host when the tick takes a blood meal. Numerous species of ticks are known to produce the toxin.

Signs may not appear until for 5 to 7 days after the tick bite. Initially, there is paresis of the legs, progressing to unsteady gait, knuckling, ataxia, and, ultimately, flaccid paralysis of the hind limbs. Loss of motor function ascends cranially, causing paralysis of the forelimbs. Pain perception remains. Even with paralysis of the limbs, the animal is bright and alert and able to eat and drink if feed is placed within reach. Ultimately, paralysis involves the neck, throat, and face, causing difficulties in chewing, swallowing, and breathing. Respiratory failure is the cause of death. This sequence of clinical signs is the only sure method of diagnosis, unless the tick is found. That may be quite difficult on an animal the size of a horse. The differential diagnosis includes encephalitides, head or spinal trauma, and hyperthermia.

There is no antidote for the toxin. The offending tick or ticks must be removed, which may produce a dramatic response. Look in the lightly haired areas of the axillary space, perineum, and behind the ears if tick paralysis is suspected. In a hospital situation, the animal should be endotracheally intubated and respiration supported until the effect of the toxin is dissipated. This is not an option in the field.

Skunk Odor Removal

All carnivores have scent glands contiguous to the anus, but the striped skunk *Mephitis mephitis* and several other skunks have highly developed glands that produce a powerful and pungent musk. The excretory duct of each gland leads to a papilla located at the mucocutaneous junction just inside the anus. Skunks are capable of accurate ejection of the musk for a distance as far as 10 feet (3 m). The odor may be detected 1.5 miles (2.5 km) downwind. The musk is a complex mixture of organic compounds. Those primarily responsible for the objectionable odor include *S-(E)*-2-butenyl thioacetate, *S*-3-methylbutanyl thioacetate, and *S*-2-quinolenemethyl thiocyanate, all of which are sulfur-containing thiol compounds. In addition to the obvious odor, skunk musk is nauseating to some people and may also cause retching in dogs.

If a person or a pet is sprayed in the face, the musk is an irritant that causes conjunctivitis, keratitis, lacrimation, temporary impairment of vision, glossitis, and slobbering and foaming at the mouth. Quick flushing of the face and eyes with copious quantities of cold water restores vision and minimizes persistent irritation. If conjunctivitis persists, instill contact lens solution or a drop of olive oil into the conjunctival sac.

The objective of odor removal is to wash away the offending oily liquid and neutralize the compound by oxidizing the thiols to nonodorous sulfonic acid. Simple bathing will not completely eliminate the odor, which is pungent in a remarkably dilute concentration. The product used to neutralize the thiols depends on the object to be deodorized. Colored clothing may be damaged by bleaches, and some substances should not be used directly on the coat of a dog and certainly not on the skin of a person.

One of the most effective oxidizing agents is a dilute solution of household bleach (Clorox); however, this may bleach clothing and hair and is harsh on the skin of people and animals. Dozens of substances have been recommended and testimonies abound as to their effectiveness. Skunk musk is alkaline, so mildly acidic solutions may be at least partially effective and will reduce the pungency of the odor. Tomato juice, white vinegar, and ammonia in water are all touted but may not completely eliminate the odor.

A frequently mentioned formula is a combination of hydrogen peroxide ($1^2/_5$ cup [347 mL] of 3%), water (1 cup [237 mL]), baking soda (sodium bicarbonate, $^1/_4$ cup [60 mL]), and a dog detergent (1 tbsp [15 mL]). Mix the peroxide with the water, and then add the baking soda and shampoo. Mix and pour into a squirt or spray bottle. This solution may be sprayed onto a dog or a horse, but it should not be sprayed directly into the eyes or nose. It should remain on the coat for 10 minutes while being worked in with a gloved hand.

Washable clothing should be washed with a strong soap or heavy-duty detergent. In a permanent camp, items that cannot be washed (shoes, leather goods) may be buried in sandy soil for a few days. The soil will adsorb the odorous chemicals.

The following commercial products have been formulated to neutralize the thiols and completely eliminate the skunk odor:

- Neutroleum alpha is nontoxic and may be used on clothing and pets. It may be obtained from USDA, Damage Control, P.O. Box 81886, Lincoln, NE, 68501, phone 402-434-2340.
- Skunk-Off is nontoxic and nonirritating, even to mucous membranes, and safe to use on pets and clothing.
- Odor-Mute is available in granular form and easy to transport. It is nontoxic and may be applied to pets and clothing after being dissolved in water.

Check with pet supply shops or small animal veterinary clinics for these commercial products, which are for use on pets or fabric and not recommended for use directly on people. Only the trek physician is qualified to make such a recommendation. However, washing with soap and copious amounts of cold water will wash considerable musk from human skin.

Plant Poisoning

It is unrealistic to expect to be able to identify all potentially harmful plants in every locale. However, certain highly toxic plants that grow in wilderness areas of the United States should be recognized (Table 55-2) (see Chapters 58 and 59).[2,7,11] Co-

TABLE 55-2. Poisonous Plants That May Affect Horses or Llamas on Trek

COMMON NAME	SCIENTIFIC NAME	POISONOUS PRINCIPLE	SIGNS OF POISONING	HABITAT	SPECIES	THERAPY*
False hellebore, corn lily	*Veratrum californicum*	Alkaloids	Vomiting, salivation, convulsions, fast irregular pulse	High mountains, meadows	Llama	Symptomatic
Death camas, sandcorn	*Zigadenus* species	Alkaloids	Foaming at mouth, convulsions, ataxia, vomiting, fast weak pulse	Hillsides, fields, meadows, in spring of year	Horse, llama	Symptomatic
Water hemlock	*Cicuta douglasii*	Resin	Frothing at mouth, muscle twitching, convulsions, death in 15–30 minutes	Standing or running water, obligate aquatic	Horse, llama	Symptomatic
Nightshade	*Solanum* species	Alkaloidal glycoside, solanine	Vomiting, weakness, groaning	Ubiquitous	Horse, llama	Symptomatic
Jimson weed	*Datura stramonium*	Alkaloid, atropine	Dry mucous membranes, dilated pupils, mania	Waste places	Horse, llama	Parasympathomimetics
Tobacco, tree tobacco	*Nicotiana* species	Alkaloid, nicotine	Stimulation of CNS, then depression; sweating, muscle twitching, convulsions	Waste places	Horse, llama	Symptomatic
Lupine, blue bonnet	*Lupinus* species	Alkaloid	CNS depression, dyspnea, muscle twitching, ataxia, frothing, convulsions	Ubiquitous	Horse, llama	Symptomatic
Dogbane, Indian hemp	*Apocynum cannabinum*	Cardioactive glycoside (similar to digitoxin)	Dyspnea, cardiac arrhythmias, agonal convulsions, vomiting, diarrhea	Ubiquitous	Horse, llama	Symptomatic
Oleander	*Nerium oleander*	Same as for dogbane	Same as for dogbane	Ornamental	Horse, llama	Symptomatic, gastrotomy
Castor bean	*Ricinus communis*	Ricin, water solution	Anaphylactic shock, diarrhea	Ornamental	Horse, llama	Treat for shock; fluids
Rhododendron	*Rhododendron* species	Andromedotoxin glycoside	Vomiting, colic, severe depression	Shrubs in meadows and moist places	Llama	Activated charcoal, time

*In most cases of poisoning from ingestion of poisonous plants, no specific antidote exists. Victims are treated symptomatically. The critical factor is to empty the digestive tract of the plant material with cathartics, parasympathomimetic stimulation, and enemas. Activated charcoal, given orally, may be of value.

operative extension offices at federal land grant institutions frequently publish booklets on local poisonous plants.

Horses may be less at risk than llamas because horses are primarily grazers and not likely to eat trees and shrubs. Llamas eat small quantities of almost anything within reach.

Animals are not affected by poison oak *(Rhus diversiloba)* or poison ivy *(Rhus toxicodendron)*. However, horses, dogs, and llamas may transmit the toxic oils of these shrubs to susceptible people via contact with contaminated hair.

Few specific antidotes are available for plant poisons. Symptomatic and supportive treatment includes removal of the poisonous material from the gastrointestinal tract by administration of a cathartic (see Table 55-3) or by adsorbing the toxin with a nonspecific substance such as activated charcoal.

Eye Injuries

An abrasion, laceration, or penetrating wound of the cornea is serious, especially in a field situation. Dust and other foreign bodies within the conjunctival sac require prompt removal to avoid more serious injury to the cornea. The management of eye problems in animals is the same as for humans, with the added difficulty of restraint for examination and therapy.

Lightning Strike

Lightning strike is a potential hazard in the wilderness (see Chapter 3).[9,12] Animals with high head carriage, such as horses and llamas, are especially at risk, whether they are standing or recumbent. Animals, like people, may survive a lightning strike, but frequently a strike is lethal. Unless the strike is witnessed, it may be difficult to make a diagnosis, but the following signs may be noted: depression, blindness, nystagmus, paralysis of the hind limbs, and temporary unconsciousness. Linear singe marks are definitive if present, but lightning may kill an animal without producing burns. One llama was struck while recumbent in an open pasture. When the body was moved, the green grass had been singed where the llama had been lying. A single lightning strike may kill more than one animal, especially if a group has congregated under a tree for shelter. Lightning may kill so quickly that feed is still in the mouth of grazing animals.

During an electrical storm, animals on the trek should be positioned in the safest environment possible, away from tall trees and exposed hills. Llamas can be encouraged to lie down in a small ravine or a depression, or against a rock face, with the head tied close to the ground. A picket line stake may be used for the tie-down. Avoid tying an animal to a tree; however, using a small bush for a tie-down may be safe. Horses are more difficult to deal with because it is impossible to get them to lie down unless they are specially trained to do so. Get them into a ravine, or a depressed area, or near a rock face.

If an animal is struck and still alive, only time will determine the degree of damage done to the nervous system and other vital organs. If the strike is witnessed and the heart has stopped beating, chest compression (cardiac massage) may be performed if it is determined that it is safe for a human to be in the open. With the animal in lateral recumbency, pull the forelimb as far cranially as possible and press on the chest wall just caudal to the triceps muscle. Cardiac massage may be required for a number of minutes.

Snakebite

Venomous snakes are found in most wilderness areas of the world, except the Arctic and Antarctic.[8,11] Trek leaders should be aware of the species of snakes that may be encountered on the trek and should instruct all personnel on how to deal with a bite in a human or animal. In the United States, the venomous snakes most likely to be encountered are pit vipers, the most ubiquitous of which are rattlesnakes of the species *Crotalus*. Other members of the North American pit viper group include the copperhead, *Agkistrodon contortrix*, and the water moccasin, *Agkistrodon piscivorus*.

Llamas and, to a lesser extent, young horses are curious animals and may stick their noses out to investigate strange animals in their area. Thus, it is not uncommon for an animal to be struck on the nose. Leg bites may occur in any animal.

The main clinical sign of venom injection is pronounced swelling in the area of the bite, beginning 1 to 3 hours after the bite. The most serious consequence of being bitten on the nose is swelling that occludes the nostrils, making it virtually impossible for horses and llamas to breathe. Dogs are not obligate nasal breathers, but the effects of a bite may be more severe in them than in the larger animals. A rattlesnake bite on the nose is an emergency.

The trek first-aid kit should contain a 10-cm length of a 1-cm-diameter flexible plastic tube. The first step in managing snakebite on the nose is to insert this into a nostril before any swelling occurs, to prevent occlusion of the nostril and provide a passageway for air. The tube cannot be inserted after swelling has developed. If swelling has already developed, the life-saving procedure is a tracheotomy.

DO NOT attempt to cut the skin and suck out the venom with your mouth. This procedure is not effective.

For pit viper envenomation, the only specific treatment is administration of antivenom. Freeze-dried antivenom in vials can be carried in the first-aid kit. The product used for animals is the same as that used for humans. One to three vials should be administered intravenously once signs of envenomation have appeared.

Choke

Choke in humans refers to lodging of food or other objects in the larynx or trachea, a life-threatening emergency. Choke in animals usually refers to lodging of food or other objects in the esophagus.[11,13] The signs of choke may be alarming, but an animal rarely dies from it unless feed is regurgitated and inhaled into the lungs.

As a preliminary step, animals should be accustomed to any supplemental feed to be used on the trek. Choke is most often caused by overly rapid ingestion of pellets or grain, so a preventive measure is to slow down ingestion. This can be accomplished by placing rock pebbles in the feed container. Llamas and horses are too fastidious in their eating habits to consume such objects, so they to separate the rocks from the feed and thus take longer to eat. Metallic or wooden objects are rarely swallowed.

Retching is the principal sign of choke, as the animal attempts to dislodge the mass. Choked animals are able to breathe, but they are clearly in distress. Saliva may flow from the mouth and the victim may cough up particles of the feed. It may be possible to feel a mass on the left side of the neck if the obstruction occurs in the cervical area. Peristaltic waves may be observed moving up and down the left side of the neck. The mass may lodge anywhere along the course of the esophagus, but generally it lies within the chest and is not visible externally.

Water may be offered, but feed should be withheld until the problem has corrected itself. Palpate along the lower neck to determine if a bulge is present. If so, gently massage it to determine if it can be moved. Sometimes, moderate exercise may cause the object to move toward the stomach. In some cases, passage of a stomach tube and application of gentle pressure may push the mass into the stomach. Medication (intravenous acepromazine [0.05 mg/kg] or xylazine [0.05 to 0.25 mg/kg]) may be necessary to relax esophageal spasm.

Drowning

Llamas, horses, and dogs are able to swim, but circumstances may cause submersion in water and inhalation of water into the lungs.[12] A heavy fleece would be a serious detriment to a llama in water over its head. Floods may trap and drown animals.

The primary problem is to extricate the animal from its predicament, which may be extremely difficult with a large llama or a horse. It is necessary to act quickly (in less than 5 minutes) to prevent death. If the animal is to revive, water must be drained from the lungs. Place the llama on its right side with the body slanted downward to the neck and head so that water can flow out. There is a risk of producing stomach regurgitation in this position, but the need to remove water from the lungs must take precedence, even though this is a desperation maneuver at best. If immersion lasted for more than 2 to 3 minutes, the heart may have stopped, making cardiac massage necessary. The action must be continued until the victim responds or is declared dead. It is highly unlikely that resuscitation will be successful for an animal that has been under water for more than 5 minutes, regardless of the water temperature.

Wound Dressing and Bandaging

The principles of wound dressing are basically the same as for humans.[12] Cotton or a coarse bandage should not be placed directly on the wound, if possible. A nonadherent 4×4-inch gauze compress or an absorbent hydrogel pad is the best material to place against the wound. A padding layer or absorbent layer to collect fluids should be next. Finally, a surface or protective bandage should be applied to hold the padding in place.

A variety of bandage materials are suitable. Coban or Vetrap (bandage tape) is the most satisfactory bandage material because it is porous, it sticks to itself, and it is flexible, fitting to uneven surfaces. Other products, such as Elastikon (elastic tape) or rolled gauze bandages, are excellent. Duct tape is satisfactory, but care should taken to avoid wrapping it too tightly, because the tape has no extensibility and may become a tourniquet.

The placement of a smooth bandage is an art. Once the anchor roll is in place, a firm but uniform pressure should be applied as the bandage unrolls. Joints require special attention. Bandaging should be done in a figure-8 pattern to provide uniform pressure over a variably shaped surface. Avoid pressure over the point of the hock and the accessory carpal bone (back of the carpus) (see Figure 55-1).

The foot also requires special consideration. When dressing a foot wound, make certain that the spaces between the digits of dogs or llamas are padded with cotton. The easiest bandage to apply is a Vetrap elastic bandage that conforms to the odd shape. If a severe foot wound occurs while trekking in the back country, additional protection may be necessary to allow the animal to continue on the journey. The author carries a sheet of pliable leather of the type used to construct moccasins. The dressed foot is placed in the center of the sheet and then the leather is gathered up around the pastern and held in place by duct tape or another bandage, roughly forming a boot shape. Another method is to use a rolled-gauze bandage. After dressing the foot, make a wrap around the pastern, leaving a tail of gauze approximately 6 inches (15 cm) long. The tail is used as a fulcrum to change the direction of the bandage as the digits are covered.

Cardiopulmonary Resuscitation[11,12]

Rescue breathing is rarely required for an animal on a trek, except in the case of a lightning strike or drowning. The procedure is different from that employed in humans, because mouth-to-mouth breathing cannot be performed on an adult llama or horse. (Mouth-to-mouth breathing can, however, be performed on a dog by clamping the mouth and lips shut and breathing through the nostrils.) A llama or horse should be placed in lateral recumbency, preferably on the right side. Stand at the animal's withers (top of the shoulder) and reach across the body to grasp and lift the arch of the rib cage. This maneuver flattens the diaphragm and expands the chest, producing inspiration. Do not press in this same area to force expiration, as this will put pressure on the stomach and possibly cause regurgitation. Instead, press over the heart area just above the elbow and caudal to the muscles of the upper limb. The rate for the horse or llama is 10 to 15 breaths/min. Rescue breathing in the dog is performed by compressing the chest at the widest segment of the thorax at a rate of 20 to 30 breaths/min.

Cardiac massage may be performed by placing the llama or horse on its right side. Have an assistant pull the upper foreleg forward and press the chest in the area vacated by pulling the leg forward. Kneel next to the bottom of the chest. Position the heels of both hands against the chest approximately 6 inches (15 cm) above the sternum, with the fingers directed toward the spine. Press firmly with the arms held straight and release quickly. Repeat the movement every second. After 15 compressions, check for a pulse in the saphenous artery on the medial aspect of the stifle in a llama, or listen for the heartbeat with a stethoscope. After 15 heart compressions, administer five cycles of rescue breathing, as described previously. It is futile to continue cardiac massage if no oxygen is available to the heart or the general circulation. Massage must be continued until heartbeat returns and the victim begins to breathe, or when signs indicate that the victim is dead (pupils dilated, no response to touching the cornea).

For a dog, cardiac massage is performed in lateral recumbency in the type of dogs that are likely to be on a trek. Small dogs and round-chested dogs are placed on their back, and compression is exerted over the sternum.[4]

West Nile Viral Encephalitis

Trekkers must be aware of newly emerging diseases that may affect the animals that accompany the trek. West Nile virus disease (WNVD) has become an important consideration because of its explosive spread in the United States and other countries in the New World.

WNVD is new to North America (1999) but has been present in Africa and other parts of the Old World for decades. It is a mosquito-borne viral meningomyeloencephalitis with a broad host range that includes humans, horses, camelids, and a large number of bird species. The entire host range is unknown. The virus is an arbovirus that is closely related to the viruses that cause St. Louis encephalitis, Japanese encephalitis, and Murray Valley encephalitis.[1]

Transmission is via mosquito bites (*Culex, Aedes,* and *Anopheles* species) and possibly other blood-sucking insects. The disease has become endemic and is spreading throughout the United States.

The incubation period in horses, llamas, and humans is 6 to 10 days. Most infections are subclinical in horses and humans, but 10% of affected individuals develop fever and the encephalitic signs of depression, ataxia, and paresis, particularly of the hind limbs. More advanced signs include head shaking, incessant chewing, paralysis of the lower lip or tongue, severe ataxia, ascending paralysis, and terminal recumbency.[1]

WNVD is similar to dozens of diseases affecting the central nervous system, including rabies, other encephalitides, head trauma, lead poisoning, water deprivation (dehydration, salt poisoning), brain abscess, and plant poisoning. A definitive diagnosis usually cannot be achieved while on trek, so prophylaxis is the only effective management modality.

Two vaccines (West Nile–Innovator, killed [Fort Dodge], and Recombitek, live [Merial]) are approved for use in horses in the United States and have also been used in camelids. If a trek is planned for an area where WNVD is endemic and when mosquitos are prevalent, trek horses and llamas should be vaccinated (two injections, 1 month apart). When trek managers contract for animals, they should ensure that the animals have been properly immunized.

Anthrax

Unfortunately, bioterrorism is a potential reality in many countries of the world. People and the animals on a trek may be exposed to some of these agents. Anthrax is a disease that is high on this list and could affect horses, llamas, camels, and elephants.

Anthrax is endemic in many places in North America and throughout the world. It is a highly infectious bacterial disease of most domestic mammals, many species of wild mammals, and people. It is caused by the endospore-forming, nonmotile,

gram-positive rod *Bacillus anthracis*. The spores may remain in the soil for many decades.

Horses, camels, llamas, and elephants with anthrax present with an acute septicemia of sudden onset, fever, rapidly progressive debility, respiratory distress, and disorientation. Death may occur within a few hours. In less acute cases, bloody fluid may exude from any body orifice and swellings may appear in various parts of the body, accompanied by limb and perineal edema.

The differential diagnosis includes any disease that produces acute septicemia, such as salmonellosis or plague. Oleander *(Nerium oleander)* poisoning also appears similar.[11] A live-spore vaccine (Sternovac) is used in livestock and wild animals.

Although *B. anthracis* is sensitive to many antibiotics, therapy is rarely successful because toxemia is the primary manifestation of the disease in animals.

Disaster and Emergency Management

A wilderness trek may be engulfed by a natural disaster—a sudden ecologic phenomenon that causes much suffering or loss[9] (see Chapter 79). As attitude may be the key to human survival in a disaster situation, it is no less important when dealing with animals. Advance planning and preparation are essential.

Safety and protection of people take precedence over the needs of animals. However, animals should not be neglected. The following disaster situations may involve animals.

Ice Storm. An ice storm is rain that freezes immediately on striking the ground or foliage. The unique climatic circumstances that produce this phenomenon may last only a few minutes or hours, but the consequences can be great. People and animals may have extreme difficulty walking and may fall and injure themselves. On an equine endurance trail ride, a late spring freezing rain and snow storm halted a group of horses on the summit of a small mountain peak. Lightning accompanied the storm. The horses were frightened by the thunder and lightning and milled around. Frozen rain and snow accumulated within the shoes on the bottom of the feet, producing a horse with ice skates. There was little direct danger from the lightning, as the peak was covered by radio and television repeater towers with lightning rods attached, but there were anxious moments until the storm abated and the feet could be cleaned out.

Fire. Fire is an ever-present risk in forests and shrubbed areas (see Chapter 12). Trek leaders should have a plan ready for human evacuation to safety, as well as how to deal with the trek animals.

In brush and forest fires, wind is an important factor. Crown fires (flames racing through the tops of trees in a forest) are particularly troublesome. Do not try to outrun a fire burning up a slope. The heat generated by the fire rises and preheats the area above the fire, setting up ideal conditions for rapid spread. In some instances, a hillside may seem to explode into flames because of the extensive preheating.

When llamas were caught in an advancing brush fire, they were herded into a bare ground area. In another situation, the llamas were simply turned loose to fend for themselves. Owners remarked that the animals adjusted quite well, without panic. When owners returned to their animals, they found them hungry and thirsty, but without evidence of smoke inhalation or eye damage. Llama fiber does not burn readily, but it does smolder, and in some animals burn spots were noted in the fleece. At one ranch, the only serious problem was burns on the foot pads, presumably caused by animals stepping on hot coals. No one was able to observe how the animals avoided the flames, but both adults and young animals did so. No abortions or premature births occurred as a result of stress.

Horses should be turned loose or ridden to a safe place.

Flooding. Animals accompanying trekkers in wilderness areas may become involved in flood conditions when they are tethered near streams that are known to generate flash floods, or when unexpectedly heavy storms cause overflowing banks of streams and rivers. A dam may burst, causing a sudden discharge of tremendous amounts of stored water. Animals are at the same risk as humans. Dry streambeds in many desert areas are prime candidates for flash floods that may be generated miles away, so they should be avoided as camp sites. Animals should not be tethered in such locations.

Trek leaders should be aware of dams along streams, especially earth-filled dams. Heavy storms may erode a dam or fill a reservoir behind it beyond its capacity. A llama owner in Idaho watched as a wall of water filled a gorge over 100 feet deep and nearly engulfed his home, barns, llamas, and farm from a burst dam a few mile upstream. Horses, llamas, and dogs can all swim but may not be able to cope with the currents generated during a flood.

Disorders Unique to Horses, Mules, and Donkeys

Common trail disorders encountered in horses, mules, and donkeys may be classified as traumatic, metabolic, or digestive.

Laminitis

Acute and chronic forms of laminitis (or founder) are possible, but only the acute equine form is likely to be seen in trek animals.[3,13] Horses with the chronic form should be excluded in the pre-trip physical examination. Llamas and elephants also have laminae that attach their toenails to the third phalanx, but horses, with their enclosed hoof and complex laminar anatomy, are predisposed to laminitis. Recognizing and minimizing the effects of predisposing factors is the focus of this presentation.

Etiology. Trauma is the most likely cause of inflammation of the foot in the trek horse. Horses should have been trained in a terrain similar to that expected on the trek. However, even a trained horse occasionally encounters an excessively rocky path that traumatizes its feet, especially if the rider is unwise and fails to slow the pace when negotiating the trail. Leather or synthetic pads worn under the shoe may offer some protection, but new pads may be chewed off the foot by rocky trails after less than 50 miles (80 km).

Other predisposing factors include ingestion of lush pasture or grain overload, and the metabolic stress associated with dehydration or overexertion. Endotoxemia associated with gastrointestinal disorders (severe colic) or septicemia may cause laminitis, but the primary disease is of greater concern to trek personnel. Whatever the predisposing factor, inflammation in the confines of an inflexible hoof causes swelling and malperfusion at the capillary level, and malperfusion causes arteriovenous shunting. Excessive ingestion of cold water, especially when the horse is overheated, may also precipitate laminitis.

Figure 55-7. Stance of a horse with laminitis. Front and hind legs are extended forward.

Clinical Signs. Laminitis usually develops on both forefeet, but hind feet may also be affected in severe cases. The horse shifts its center of gravity to the hind limbs to minimize pressure on the forefeet, standing with the hind limbs forward under the body and the forelimbs extended in front of the body (camped forward in front), as seen in Figure 55-7. The feet are warm or hot to palpation, and there is a pounding digital artery pulse. The horse is reluctant to move.

Management. Preventing or minimizing the causes of laminitis is vitally important. Once clinical signs occur, the objectives of treatment are to eliminate the predisposing factors, decrease inflammation, and maintain or reestablish blood flow to the laminae.

If laminitis is the result of a digestive upset, it is imperative to administer a cathartic (Epsom salts [magnesium sulfate], 1 kg in 4 L of water via nasogastric tube). Phenylbutazone (Butazolidin, 6 mg/kg daily, intravenously) should always be administered to relieve pain so the horse will move. Acepromazine maleate (0.04 mg/kg every 6 hours, intramuscularly) is used as a vasodilator to enhance blood flow to the laminae. If available, heparin (60 units/kg subcutaneously) helps to prevent platelet aggregation and thrombus formation.

In acute laminitis, the feet are warm or hot, so the inclination is to soak the feet in cold water. However, this is contraindicated, because the object is to increase circulation to the foot, not to inhibit it.

It is now thought that mild exercise is an important aid in preventing damage to the laminae. The horse should be exercised slowly on soft ground for 10 to 15 minutes every hour for 12 to 24 hours, and then exercise should be stopped. Even slow walking may be quite painful. A low volar nerve block relieves pain and inhibits vascular constriction within the foot. This is accomplished by palpating the pulsating artery on the posterior lateral aspect of the fetlock (Fig. 55-8). The nerve lies posterior to the artery. With a 20- to 22-gauge needle, 3 mL of 2% lidocaine is injected over each nerve. It may be necessary to repeat nerve blocks two or three times daily for several days. Corticosteroids are contraindicated.

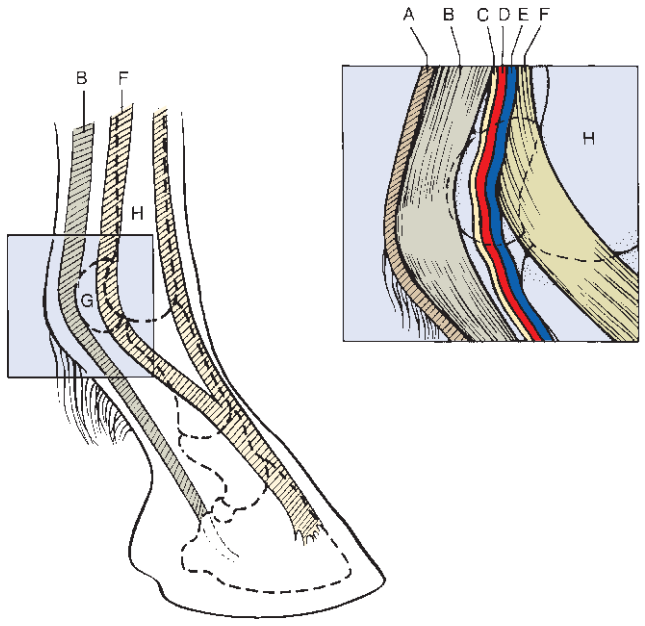

Figure 55-8. Diagram of the anatomy of the equine fetlock. **A,** Skin. **B,** Flexor tendons. **C,** Volar nerve. **D,** Palmar digital artery. **E,** Digital vein. **F,** Suspensory ligament. **G,** Sesamoid bone. **H,** Metacarpal bone (cannon).

Saddle, Cinch, and Rigging Sores

Saddle sores are contusions, abrasions, or ulcerations of the skin and subcutis caused by friction or point pressure from the saddle, cinch, or rigging.

Etiology. Improperly fitted or maintained tack causes poorly distributed pressure, which is the primary cause of saddle sores. A saddle tree may fit a horse properly at the beginning of a training period but become unsatisfactory after the horse has trimmed down. Spot pressure causes local ischemia. If pressure is prolonged, the capillary bed may be damaged. When the pressure is released rapidly, blood rushes into the blanched site, and extravasation may occur, causing swelling and pain.

If the lesion is rested and treated as inflamed tissue, complete healing may occur. However, if the saddle is reapplied, it overlies a lump that is subject to abrasion. The injury can extend through the dermis, resulting in severe ulceration. Cinch and rigging sores are usually caused by friction, leading to blister formation.

Clinical Signs. Hot and tender swellings are the primary signs of acute saddle sores. The hair or epidermis may be rubbed off. General sensitivity over the back is usually caused by muscle soreness.

Evidence of previous sores includes white hairs in spots over the withers or saddle bed, scars that may or may not be haired over, thickening of the dermis, and alopecia with or without swelling.

Treatment. Prevention is better than treatment. Toughening the backs of trail horses is a major job of the trainer. Proper

pads or blankets must be selected for each horse. On arrival at a rest area, the girth should be slowly loosened at intervals of 10 to 15 minutes to prevent rapid flow of blood into ischemic areas.

Once a sore has developed, the horse must be rested or the tack changed to eliminate pressure or friction on the lesion. Holes are often cut in pads to accommodate a saddle sore, but spot pressure at the ring edge may be as detrimental as the original cause of the saddle sore. Riders must keep tack cleaned and in good repair. When the saddle is removed, cold water poured over the back for 15 minutes may minimize swelling.

Myopathy

Exertional myopathies of horses vary from simple muscle soreness, through the tying-up syndrome (similar to a charley horse in humans), to paralytic myoglobinuria (rhabdomyolysis, azoturia).[5,13]

Etiology. Muscle integrity and function depend on proper mobilization and use of energy. Conditioned muscles may function vigorously for extended periods, but if either energy mobilization or use is accelerated to the extreme, mild to severe muscle necrosis may occur.

Clinical Signs. Mild muscle soreness is characterized by alterations in gait that indicate muscle weakness. As severity increases, the gait becomes progressively altered until the horse is in obvious pain and is reluctant to move. The horse has an anxious expression and may sweat excessively. Affected muscles are painful to palpation and may be swollen. Skin temperature over the muscle may be elevated. Muscle spasms may occur but not consistently. Myoglobinuria is observed grossly in moderate to severe cases and should be considered prima facie evidence for stopping and resting or treating the horse.

Treatment. Rest is paramount in all cases. A rider may have difficulty differentiating the pain associated with myopathy from that seen with colic until the horse is allowed to lie down, looks at its own side, or kicks at its own belly. It is disastrous to force the severely myopathic horse to walk, as is done with a suspected case of colic. If there is doubt, the horse should not be exercised.

Horses in inaccessible locations should not be walked out until all possible recovery has taken place. Horses with mild muscle soreness may improve if walked slowly, but rest from ride exertion is the primary recommended therapy.

Dehydration

Etiology. Fluid and electrolyte imbalances[5,11,12] play a major role in all metabolic medical problems that develop in the horse as a result of exertional stress. The degree of dehydration is directly related to the amount of fluid lost through evaporative cooling (sweat), which in turn is related to the amount and rate of work performed and to ambient temperatures and humidity.

The horse must have an adequate amount (10 to 15 gal [45 to 68 L]) of water each day during a trek. The more strenuous the activity, the more fluids are required. Mules, and especially donkeys, are adapted to cope with mild dehydration.

Clinical Signs. Signs of mild dehydration (3% bodyweight loss) are low urine output, dry mouth, and mild loss of skin elasticity.

Moderate dehydration (5% bodyweight loss) is characterized by marked loss of skin elasticity. The eyes become sunken. Blood pressure may fall as a result of decreased plasma volume. Weakness, fever, and weak pulse may be observed. Sweating is not possible, even with elevated body temperature.

Marked dehydration (10% bodyweight loss) may involve circulatory failure from decreased plasma volume.

Treatment. During 3 hours of hard work, a 450-kg (about 1000-lb) horse may lose as much as 45 L (10 gal) of fluid. If this degree of dehydration is not corrected quickly, death may result. The horse should be allowed to drink along the trail if water is available. Small amounts of cool, but not cold, water should be offered. If the horse refuses to drink, gastric intubation may be indicated. Fluid is also absorbed from the colon; thus, enemas (10 to 20 L [2.2 to 4.4 gal] of water) are effective in rehydration. Electrolyte replacement is encouraged, but for the usual case of dehydration is not critical. Packaged electrolytes are available in veterinary supply shops.

Heat stress usually accompanies dehydration, so cooling (such as shade or a water bath) is important. Administration of intravenous fluids, if available, is routine therapy.

Camels are uniquely adapted to desert conditions. They are able to survive a week without water. Optimally, a camel, like horses and llamas, should be watered daily. Camels do not store water but conserve it because of a diurnal fluctuation of body temperature, from a normal of 37.0°C (98.6°F) up to 42°C (107.6°F). The body acts as a heat-sink during the heat of the day, thus conserving vital water that would otherwise be lost through evaporative cooling. During the cool desert night, the heat is dissipated by conduction. Camels can concentrate urine to a syrup consistency to avoid water loss through urine. Fecal pellets are passed that are dry enough to be used for fuel immediately after defecation.

The camel can tolerate a 40% reduction in bodyweight from dehydration, and after such a severe dehydration it is able to drink sufficient water at one session to rehydrate. The shape of the camel erythrocyte is elliptical, allowing circulation despite significant hemoconcentration. This elliptical shape allows the cell volume to expand 250% without hemolysis when rehydration occurs.

Exhausted Horse Syndrome

The term *exhausted horse syndrome* (EHS) was coined to describe a complex metabolic syndrome occurring when horses are pushed beyond endurance limits.[5,13] The syndrome has no precise characterization, because the initiating mechanism varies with the prevailing conditions. The development of EHS depends on the condition and training of the horse, the pace set by the rider, the terrain traversed, and climatic factors such as temperature, wind, and humidity.

Clinical Signs. There is a composite picture, but the individual horse may show only a few signs. The severely affected horse is depressed and exhibits lethargic movements, holding its head low. The eyes are expressionless. Facial grimacing creates an anxious expression that may progress to a pained expression if colic or muscle spasms accompany the syndrome. The horse

takes no interest in its surroundings. Anorexia is typical, and frequently the horse has no inclination to drink even though dehydrated. The corneas appear glazed.

Body temperature is usually elevated and may reach 41°C (106°F). The horse does not cool properly. Usually, body temperature continues to rise. Temperature measured rectally may be inaccurate in the exhausted horse, because the anal sphincter loses tone and allows air to enter the rectum.

Cardiovascular and respiratory systems are markedly affected by endurance riding. Heart and respiratory rates are elevated. The rates depend on the prior condition of the horse, pace, length of action, and amount of work performed in climbing or walking on soft footing. Heart rates of 150 beats/min are not uncommon after a grueling climb. With 10 to 15 minutes of rest, the heart rate of a conditioned horse should drop below 60 beats/min, whereas in the exhausted horse, tachycardia and tachypnea may persist. The exhausted horse may have a respiratory rate faster than the heart rate. Respiration under these circumstances is shallow and inefficient. Additional cardiopulmonary signs may include synchronous diaphragmatic flutter, arrhythmias, murmurs, and visible jugular pulses. Auscultation of the thorax may reveal moist rales and, in extreme cases, frank pulmonary edema.

Severe dehydration is the most consistent sign of EHS. Loss of skin elasticity, sunken eyeballs, and dry mouth and mucous membranes reflect a 7% to 10% loss of bodyweight, after a loss of 30 to 40 L (6.6 to 8.8 gal) of fluid. Serious electrolyte and acid–base imbalances are associated with dehydration. Alkalosis is common.

Muscular manifestations of EHS include fatigue, trembling, spasm, stiffness, muscles painful to palpation, and, rarely, exertional rhabdomyolysis (stiff gait, excessive sweating, high respiratory rate, and firm and painful muscles of the back and rump).

Horses suffering from EHS are prone to colic, which also may occur independently. When colic accompanies EHS, it is of the spasmodic type, with diminished or absent borborygmus.

Treatment. Rest, rehydration, and electrolyte supplementation are the keys to recovery. If the horse is drinking and will take electrolytes in the water, the effect is nearly as beneficial as the administration of intravenous fluids. Packaged electrolyte powders can be carried.

If the horse is hyperthermic, shows evidence of shock, and refuses to drink, more drastic steps must be taken. The horse may require 40 to 50 L (8.8 to 11 gal) of fluid. Besides intravenous fluid (80 mL/kg/hr) administration, gastric intubation may be employed to give fluids orally. Enemas are also effective, as fluid is absorbed from the colon (it is essentially impossible to instill water into the rectum too rapidly or in too great a volume as long as the fluid is allowed to be pushed out by peristaltic activity).

Synchronous Diaphragmatic Flutter

Synchronous diaphragmatic flutter (SDF) is a clinical sign observed in endurance horses while on long-distance rides, and it may be seen on an expedition.[3,5,13] It is defined as a spasmodic contraction of the diaphragm synchronous with the heartbeat. It is not life-threatening per se, but it indicates mild to serious metabolic conditions that may be, or may become, life-threatening. Overexertion with excessive sweating produces metabolic alterations. The development of SDF at any point on a trek should be ample reason to prevent the horse from going further until the metabolic alteration is resolved.

Although acute exertional stress is characterized by acidemia, SDF is associated with alkalemia. Electrolyte imbalances may also be involved. Experimental work seems to indicate that hypocalcemia, hypokalemia, and alkalosis act in concert (or, less likely, individually) to cause equine SDF.

Clinical Signs. SDF may develop after 20 to 30 miles (32 to 48 km) of riding. There is no sex, age, or breed predilection. The primary sign is spasmodic contraction in the flank area. The "thump" is easily felt by light palpation in the flank area. Auscultation of the heart while holding a hand over the dorsocaudal rib area can reveal that diaphragmatic contraction is synchronous with the heartbeat. SDF may be the only clinical sign noted, or it may be seen as part of EHS.

The degree of thumping may vary from a barely perceptible quiver to a contraction that seems to rock the horse's body and is observable from a distance. The flutter may be continuous or intermittent, especially in degree.

Treatment. Rest and rehydration are required.

Colic[13]

Colic is the clinical manifestation of abdominal pain, usually the result of a gastrointestinal disorder. It may also arise from the urinary system or peritoneum. Types of colic are numerous because of the unique and complex anatomy of the equine digestive system (Fig. 55-9). The colon and caecum are voluminous in the horse, but there are narrow areas where impactions may form. The ileum enters the colon near the pelvic inlet.

Etiology. Colic may be produced by gastric tympany, intestinal obstruction, or hyperspasticity. The most likely inciting causes on a trek are overeating of nonregular forages, ingestion of poisonous plants, or exhaustion. One illustrative tale is of a commercial packer who hobbled a pack string of horses to graze in a meadow for a noon rest stop. Two hours later, when it was time to begin the afternoon trek, two of the horses were colicky.

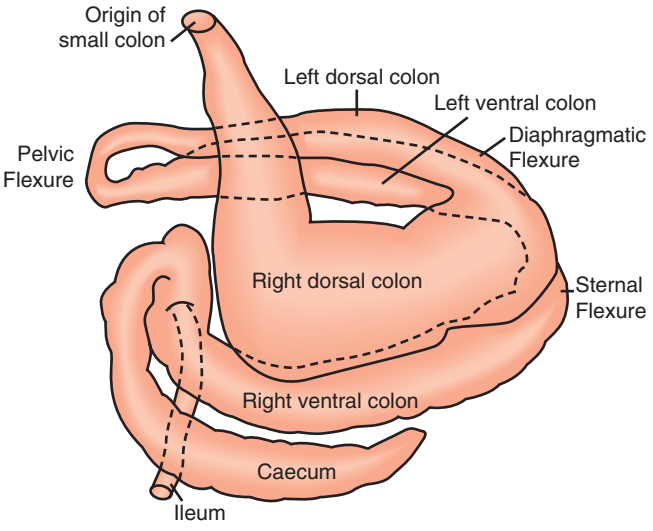

Figure 55-9. A diagram of the complex caecum and large intestine of the horse.

In checking the meadow the packer saw many plants of death camas (*Zygadenus* species), some of which had been eaten. The colic was sufficiently severe to require a layover at the meadow for the rest of the day.

Clinical Signs. Horses express colic by looking back at one side, stamping the feet, getting up and down, rolling, and pressing the head against trees or rocks. The pulse rate may exceed 100 beats/min, and the pain may be severe. The conjunctival membranes are congested and cyanotic.

Treatment. Only superficial emergency measures are discussed. Treatment is variable, depending on the anatomic location of the obstruction or spasm. Mild obstructions may be relieved by hydration and administration of a cathartic. Cold water enemas may stimulate sluggish intestinal peristalsis and relieve impaction of the terminal small colon.

Pain and spasms may be relieved by administration of flunixin meglumine (Banamine), 0.6 mg/kg intramuscularly twice a day. Walking the horse may prevent it from lying down and rolling, which may result in torsion of the intestine.

Medical Problems Unique to Pack Llamas

Most of the medical problems of llamas[11,12] have been considered under headings of disorders common to all animals. Llamas are hardy animals and unlikely to allow themselves to be pushed to exhaustion. If fatigued, they simply lie down and refuse to rise. They will not tolerate excessive loads. One hundred pounds (45 kg) is an appropriate load for a well-trained and conditioned large llama.

Plant poisoning is more likely in llamas than in horses, but such incidents are rare. Rope burns from entanglement in tethers are less likely than in horses. The llama is less excitable, and if it becomes entangled, it usually lies quietly until help arrives.

North American venomous snakes pose minimal threat to large animals. However, llamas are curious and may be bitten on the nose while investigating the strange animal. Nose bites from rattlesnakes are especially hazardous to llamas, because local swelling may occlude the nostrils (see Snakebite, earlier). Llamas are primarily obligate nasal breathers, so dyspnea and suffocation may ensue. Rattlesnake bites of the limbs cause edema and in severe cases local tissue necrosis and ulceration. Many cases are diagnosed as trauma unless the bite is observed. The signs of snakebite in llamas are essentially the same as in horses.[8,11]

Therapy for a nose bite may require tracheotomy (in the upper third of the neck; the surgical approach is similar in all species) and insertion of an improvised tube to allow breathing. Edema may persist for 2 to 3 days. If the bite is observed and progressive swelling noted, a small tube (1 cm in diameter) can be inserted 15 cm into the ventral meatus of the nasal passage and sutured to the nares. An alternative improvisation is to insert a woman's hollow plastic hair curler into the nostril. Swelling occurs around the tube or curler, but patency of the nasal passage is maintained.

Problems Unique to Dogs

Exercise Stress

Sled dogs are usually conditioned for heavy work, but the inexperienced handler may push animals beyond their endurance. Dogs without adequate conditioning are frequently taken on hikes by their owner. Muscle soreness, fatigue, dehydration, and hyperthermia are common and are dealt with as for humans. Erosions of the footpads require special care. A sheet of soft leather out of which to fashion temporary boots should be carried.

Porcupine Quills

When dogs are brought into porcupine country, the risk of an encounter is great. Some dogs fail to learn from experience and are repeatedly quilled. The dog must have physical contact with the porcupine for the tail to introduce the quills. The muzzle and face are the usual sites of penetration, and a dog can be blinded by perforation of the eyeball.

The quills must be removed physically. A pair of pliers should be included in supplies and equipment. The process is painful and sedation with diazepam is indicated. The quill should not be broken because the retrograde barbs on the quill foster migration and abscess formation.

Grass Awns

Numerous species of grass, such as foxtail grass, have awns with barbs that may become attached to the dog's hair coat or lodged in the external ear canal, nasal passage, conjunctival sac, or interdigital space. Dogs that must travel at the time of year when plants are mature and the awns are easily dislodged from the seed head should be inspected frequently for awns in these sites.

Signs depend on the location of the foreign body. When it is within the ear canal, the dog paws at its ear and shakes its head. The head may be held tilted. Exudate may flow from the ear. Awns in the nostril cause sneezing and nasal exudate. Awns in the conjunctival sac cause lacrimation, photophobia, and corneal edema and ulceration, and the dog paws at the eye. Awn penetration between the digits and at other locations through the skin is more difficult to diagnose because the awn may be at some distance from the fistula.

Awns must be removed physically. Sedation, topical anesthesia, or both may be necessary. Although topical ophthalmic anesthetics are desirable in the eye, lidocaine may be used in an emergency. A pair of small alligator forceps is most suitable for reaching into otherwise inaccessible places. An otoscope may be necessary to visualize awns in the nostril or ear canal. Instillation of an antiseptic or antibiotic ointment is desirable after removal of the awn.

Stinging Nettle Poisoning

Stinging nettle (*Urtica* species) is common along streams and lakes in wilderness areas.[12] Humans vary in sensitivity when the plants accidentally contact exposed skin. Leaves and stems are covered by harsh hairs, some of which have a tiny ball tip that breaks off just before penetration. The specialized hairs are hollow. A base gland produces histamines and acetylcholine, which are injected into the victim.

Short-haired dogs that move through patches of stinging nettle are at risk of poisoning from the cumulative effect of thousands of minute injections of acetylcholine. Weakness, dyspnea, and muscle tremors are characteristic of the action of acetylcholine on peripheral nerves. Parasympathomimetic effects include salivation, diarrhea, tachycardia, and pupillary dilation. Atropine sulfate (0.04 mg/kg) subcutaneously is a specific treatment.

Harness Sores

Improperly fitted harnesses on sled dogs may produce the same type of friction lesions that occur in horses or llamas.

Diseases Unique to Cats

Basic medical care for a cat is similar to that for a dog. Fleas are mentioned because of the usual close contact of a cat with its owner or other people on the trek. Many people are sensitive to flea bites. Cats should be de-fleaed before the trek begins, but they may acquire fleas from other cats they encounter or from rodents.

Constipation (obstipation) is a sign associated with many diseases. On a trip, a cat may respond negatively to a change in diet. If the cat is habituated to defecating in a cat-box, it may be necessary to train the cat to use soil before taking it along on a trek. Signs include anorexia, lethargy, tenesmus, and frequent unsuccessful attempts to defecate. Severe cases of constipation may be treated with cool water enemas and handled in much the same way as a constipated person.

Hairballs. The ingestion of hair during self grooming is a frequent cause of gastric irritation and obstruction. The prominent clinical sign is vomiting, commonly with hair found in the vomitus. Hairballs may exit the stomach and cause obstruction further along the gastrointestinal tract. The usual management of hairballs is to place a commercial lubricant laxative on the paw of the cat, which it will lick off and ingest. The common ingredients in such commercial products are white petrolatum, cod liver oil, corn syrup, malt syrup, and light mineral oil. Typical commercial products are Laxatone, Cat Lax, Lax-aire and Laxa Stat, which are obtained in pet shops and veterinary clinics. These medications may also be used for mild constipation in cats and dogs.

Escape of Support Animals

Support animals may escape from any type of restraint and run away or simply wander off. More commonly, animals are tied with improper knots, or poor-quality snaps and halters are used that break when the slightest pressure is applied. A trekker may tie an animal to a shrub, bush, or branch that can be pulled up or broken off with a slight tug. Animals that are frightened by predators or other wild animals wandering into an area where they are tied may bolt and exert tremendous pressure on tethers, picket lines, stakes, or branches.

Hobbling is a common technique that allows horses and mules to graze. Trek leaders must know the habits of individual animals when hobbled. One horse may accept having the front feet hobbled together and graze peacefully, taking only the short steps possible. Another horse may learn how to gallop with the front feet hobbled. The author once had to track a pair of packhorses for 5 miles to recapture them. They were heading home, and the only thing that stopped them was a stream they could not negotiate with the hobbles on. Some horses must be hobbled with three restrained legs.

Even experienced trekkers (such as commercial packers) may become overconfident and place misguided faith in an animal's willingness to stand quietly without wandering away. The trekker may leave just a lead rope or ground tie dragging on the ground. The animal may become frightened or bored and wander off. The author is aware of a llama carrying a full pack that wandered off and was missing (pack and all) for a full month.

Contrary to popular belief, not all llamas are social, so they may not return to a herd that is securely tethered. Llamas, like horses and dogs, have homing instincts and may set off toward where they presume home to be located.

Steps to Take in the Event of an Escape

If a breakaway is observed, members of the party should not further frighten the animal by rushing at it. It should be given a chance to settle down and return to the group. The handlers can move around the animal to block obvious escape routes but should not try to chase it. All of these animals can run faster than a human. If the escapee has an animal buddy in the group or a favorite person, he or she can make an approach. A favorite treat in camp can be used to entice the animal back to the fold.

If an animal is discovered missing, an analysis of the situation to determine what caused the escape is needed. Was it frightened? Did it break tack? Is it dragging a log, branch, or rope? Is there evidence that other animals were nearby? Is blood or hair strewn about? In what direction did it probably go? Generally, animals return toward the direction from which they have come. Frightened animals or those escaping at night may not adhere to any rule. A search for tracks can be made, but llamas may not leave much of a track, particularly in rocky terrain.

Scanning the surroundings may result in a sighting of the animal. It may be in the vicinity of camp resting under a tree or in a clump of bushes. The search should be conducted in a planned fashion, using all persons possible. Unless tracks direct otherwise, the trail back toward a trailhead should be searched first. From firsthand experience, the author can attest to the desirability of taking another animal, preferably a buddy of the escapee, along on the search. Even though the animal is sighted, it may not be caught unless a second animal is there to bait it. Vantage points should be used, and searchers should consider where they might have gone under the circumstances. Lush meadows or other areas where there might be desirable feed should be checked.

Fellow hikers, and ultimately the Forest Service or other regulatory agencies, should be alerted about the missing animal.

Medication Procedures

A list of medications and indications for their use is provided in Table 55-3. In the horse, intramuscular injections are given in the neck or rump. Subcutaneous injections are given by lifting a fold of skin just cranial to the scapula. Intravenous injections are given in the jugular vein, which is easily distended along the jugular groove on the ventral aspect of the neck (Fig. 55-10).

In the llama, intramuscular injections are given in the relatively hairless area at the back of the upper rear leg, by standing against the body in front of the rear limb while facing the rear and reaching around the back of the animal to give the injection. Subcutaneous injections are given in the relatively hairless area of the caudal abdomen, just in front of the rear limb, or by lifting a fold of skin just cranial to the scapula. It is difficult for an inexperienced individual to locate the jugular vein for intravenous administration in the llama. Intramuscular or subcutaneous routes are recommended.

In the dog and cat, intramuscular injections may be administered in the triceps muscles caudal to the shoulder or in the muscle masses on the upper rear limb. Subcutaneous injections are made by lifting a fold of loose skin on the neck near the withers. Intravenous administration is via the jugular vein or

TABLE 55-3. Medications for Trek Animals

GENERIC NAME	TRADE NAME (COMPANY)	CONCENTRATION IN VIAL	ROUTE OF ADMINISTRATION	*DOSAGE AND INTERVAL*			INDICATION
				HORSE	LLAMA	DOG	
Acepromazine maleate	Prom Ace (Fort Dodge)	10 mg/mL	IM or IV	0.04–0.1 mg/kg	Not indicated	0.05–0.22 mL/kg	Tranquilizer
Ampicillin sodium	Generic	—	IM	10–50 mg/kg qid	10–25 mg/kg tid	25 mg/kg q6hr	Infection
Atropine sulfate	Generic	2 mg/mL	IM, SC	0.04 mg/kg	0.04 mg/kg	0.04 mg/kg	Stinging nettle
Benzathine penicillin G	Benza Pen (Pfizer)	150,000 U/mL	IM	Not recommended	5000–15,000 U/kg q2day	40,000 U/kg q5day	Infection
Charcoal (activated)	Generic	—	PO	60–250 g	100 g	3–5 g	Toxins
Dexamethasone	Azium (Schering-Plough)	2 mg/mL	IM	2–4 mg/kg	1–2 mg/kg	4 mg/kg q8hr	Shock
Diazepam	Generic	5 mg/mL	IV, IM	0.05–0.1 mg/kg	0.2–0.4 mg/kg	2–5 mg/kg	Sedation
Epinephrine	Generic (Bayer)	1:1000, 1 mg/mL	IV, IM	0.1–0.4 mg/kg	0.1–0.5 mg/kg	0.1–0.5 mg/kg	Anaphylaxis
Fenbendazole	Panacur (Hoechst Roussel)	—	PO	5–20 mg/kg	10–15 mg/kg single dose	50 mg/kg	Parasites
Flunixen meglumine	Banamine (Schering-Plough)	—	IM	1.0 mg/kg daily	1.0 mg/kg daily	0.3 mg/kg daily	Colic pain, inflammation
Ivermectin	Ivomec (Merck AGVET)	—	PO, SC	0.2 mg/kg single dose	0.2 mg/kg single dose	Not indicated	Parasites
Ketamine	Vetelar (Pfizer)	100 mg/mL	IV, IM	2.0 mg/kg	2–5 mg/kg	Not indicated	Anesthesia
Lidocaine	Generic	2%	SC	As needed	As needed	As needed	Local anesthesia
Magnesium oxide	Carmilax (Pfizer)	361 g/lb	PO	—	10–20 g total dose	—	Cathartic
Magnesium sulfate	Generic	—	PO	20–100 g	Not indicated	8–25 g	Cathartic
Phenylbutazone	Butazolidin (Schering-Plough)	200 mg/mL 1 g tab	IV, PO	1–2 g/450 kg, 2–4 g/450 kg daily	2–4 mg/kg daily	15 mg/kg q8hr	Pain, inflammation
Trimethoprim/ sulfamethoxazole	Tribrissen (Schering-Plough)	24%	IV, SC	2 mg/kg bid	2 mg/kg bid q12hr	2.2 mg/kg	Infection
Xylazine	Rompun (Bayer)	100 mg/mL	IV, IM	0.5–1.0 mg/kg	0.25–0.5 mg/kg	1.1 mg/kg	Sedation
Euthanasia solution	T-61 (Taylor)	—	IV	40 mL total dose	25 mL total dose	0.3 mL/kg	Euthanasia

IM, intramuscular; IV, intravenous; PO, oral; SC, subcutaneous; q, every; bid, twice daily; tid, 3 times daily; qid, 4 times daily.

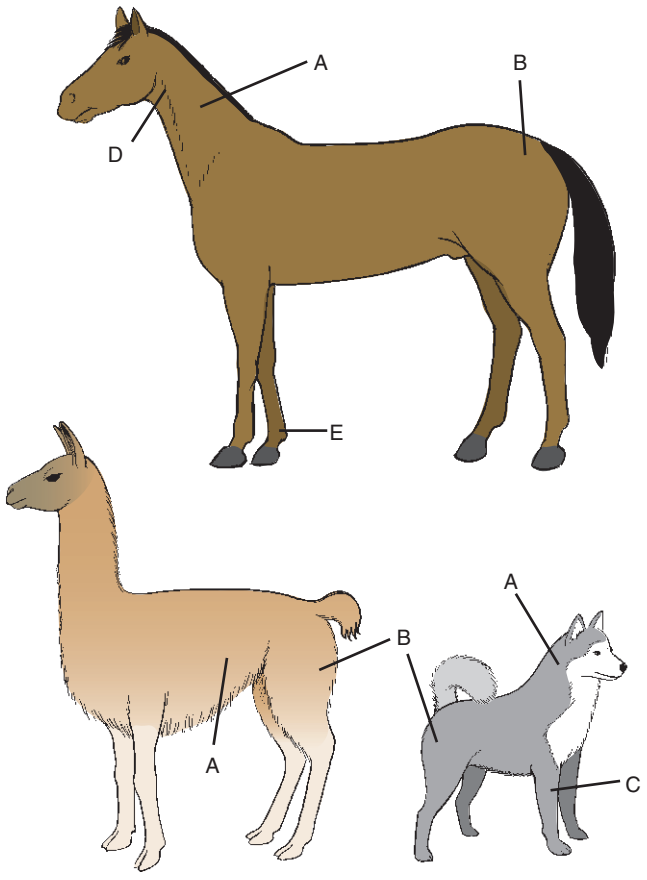

Figure 55-10. Diagrams of medication sites in the horse, llama, and dog. **A,** Subcutaneous. **B,** Intramuscular. **C,** Cephalic vein for intravenous. **D,** Jugular vein. **E,** Site for low volar nerve block.

the cephalic vein (Fig. 55-11). For the latter, an assistant grasps the limb at the elbow to occlude the vein, which courses on the dorsal aspect of the forearm. The vein is more visible if the hair is wetted down with water.

Euthanasia

Euthanasia may become necessary in the event of injury or illness that cannot be corrected or when there is little likelihood of removing the animal to a location where remedial action can be taken. Horses, mules, donkeys, and llamas cannot be carried out of a wilderness area manually but large animals may be airlifted out of the wilderness by helicopter with the animal in a special sling. However, this may be quite expensive. Dogs may be placed in an improvised stretcher and carried out.

Indications for euthanasia include compound and comminuted fractures of long bones, falling or sliding into inaccessible places from which the animal is unable to extricate itself or trek participants are unable to aid the animal, lacerations exposing abdominal or thoracic organs (such as wild animal attacks or ramming tree branches into the body), head injuries resulting in persistent convulsions or coma, and protracted colicky pain unrelieved by analgesics or mild catharsis, usually associated with a pulse rate greater than 100 beats/min, rolling, and congested conjunctival membranes.

Figure 55-11. Holding a dog for administration of intravenous medication.

The expedition may carry a bottle of euthanasia solution, which must be given intravenously or intraperitoneally (see Table 55-3). If firearms are carried, a properly placed bullet to the head produces a fast and humane death. For placement, the shooter stands in front of the animal's head and draws an imaginary line from the medial canthus of each eye to the base of the opposite ear. The shot should be aimed where those lines cross and approximately perpendicular to the contour of the forehead (Fig. 55-12). The tip of the barrel should be no more than 6 inches from the head. A heavy blow to the head at the same location is equally effective. The blow may be administered with the blunt edge of a single-bladed ax or hatchet. A large rock held in the hand may also be used. A less desirable but sometimes expedient method is to sever the jugular vein to allow exsanguination. This would probably be used on an animal that is already unconscious.

Some vital statistics for support animals are provided in Table 55-1. Equipment for support animals and the sources are listed in Box 55-1, Table 55-4, and Appendix A.

The rectal body temperature of an animal is assessed by inserting a digital clinical thermometer lubricated with water or spittle through the anus with a slight twisting motion. Heavy-duty glass veterinary clinical thermometers are available, but regular human clinical thermometers are equally useful. The use of the same thermometer for humans and animals is generally aesthetically unacceptable, even though the thermometer can be cleansed and disinfected between uses. In hot climates, clinical thermometers must be kept in insulated containers; otherwise, high temperatures may render them useless.

Transporting Animals to and from the Trailhead

Dogs

Dogs are easily transported in the fiberglass carrying cages commonly used by dog owners and for airline shipment.[11,12] Appropriate opportunities for exercise and relieving themselves should be provided. Whether food and water should be provided depends on the duration of the journey and on other conditions such as ambient temperature. A journey of longer than 12 hours would require at least water.

Horses

Horses may be transported in horse trailers or trucks.[11,12] Each animal should have been trained to enter and leave the conveyance with ease. The conveyance should be covered to prevent insects and flying debris from striking animals in the face or eyes. Legs should be wrapped to support the lower limb tendons, joints, and ligaments. Someone on the trek staff must know how to attend to these animals.

Llamas

Llamas are often moved from one location to another, and they generally travel well.[11,12] The majority lie down soon after the beginning of a journey and remain recumbent while moving. However, traveling is stressful for most llamas, whether or not they exhibit overt signs of stress. They do not need to be tied within a truck or trailer. In fact, it may be hazardous to do so. At least two fatalities have been associated with trailering tied llamas. One tied llama became entangled in a lead rope that was too long, and it suffocated. In the second accident, the llama was tied in a trailer constructed with a horizontal bar across the front, at a height corresponding to the mid-cervical region on the llama. The llama was tied with a rope over the bar. At the destination, the llama was found dead, partially hanging from the bar. At necropsy, a massive hematoma was located in the pharynx, which obstructed the glottis. Apparently, the llama had bumped against the horizontal bar at some point in the journey, rupturing a vessel in the pharynx. As the llama pulled back on the rope and halter, the mouth was forced shut so that blood in the pharynx could not flow out the mouth or nostrils.

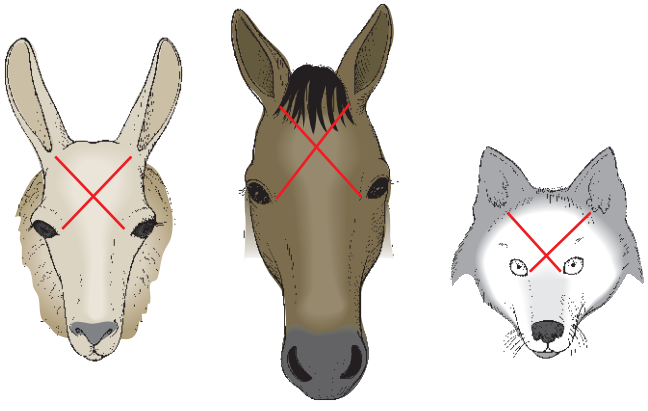

Figure 55-12. Location for euthanasia blow or shot.

Llamas are small enough and adaptable enough to be moved in numerous types of vehicles, including private vans, pickup trucks, livestock trucks, trailers, and commercial vans. Vehicle and trailer licenses must be current. If animals are insured, establish whether there is coverage for transportation; if not, explore the possibility of acquiring trip insurance. Llamas should be accustomed to the vehicle in which they are to travel. Certainly, this should include halter training and practice leading them into it or into a similar conveyance.

Vehicles and trailers should receive routine maintenance (brakes, tires, batteries, lights) before the trip. It is particularly vital to ascertain that exhaust fumes do not flow past the heads of the llamas.

Box 55-1. Equipment and Supplies for Animals*

1. Sterile pack
   Needle forceps
   Thumb forceps
   Scissors
   Scalpel handle and blades
   Hemostats (2)
2. Alligator forceps
3. Suture material (Vicryl), swaged needles, 4 packets, various sizes (0 to 4-0)
4. Gauze sponges, 4×4-inch (100)
5. Roll cotton, 1 lb
6. Vetrap bandages, 3-inch, 6 rolls
7. Syringes, plastic, 12-mL (6)
8. Needles, 22- to 18-gauge (20)
9. Tubing
   Silastic, 1 cm outside diameter, 10 cm (4 inches) long, for endotracheal tube
   Plastic flexible stomach tube, 1 cm inside diameter, 100 cm (40 inches) long
10. Funnel, plastic, to fit into stomach tube
11. Hose, garden, 20 cm (8 inches), to serve as a speculum for passage of the stomach tube
12. Hoof knife
13. Hammer
14. Pliers
15. Horseshoes and nails
16. Pliable sheet of leather
17. Heavy needle and thread
18. Otoscope
19. Stomach pump

*Table 55-4 addresses the priorities for horses, llamas, and dogs.

TABLE 55-4. Priorities of Emergency Supplies for Trek Animals*

	DRUGS		*OTHER SUPPLIES*	
	PRIMARY	SECONDARY	PRIMARY	SECONDARY
Horses	1–3,7,9,10,12	5,11,14–17	1,3–10,12–15	19
Llamas	2,4,7–12,17	6,13,15,16	1,3,4,6–12,14	19
Dogs	3,4,7–9,12,16	11,15,17	1,2,4,6–8,14,16,17	18

*Numbers refer to supplies numbered in Box 55-1.

Figure 55-13. Using a tarp to lift a llama. From top left, to top right, to bottom left, to bottom right.

Climatic conditions must be considered. More than one llama has died as a result of heat stress while being trailered. Most trek animals are not shipped by air, but it may be advisable to adopt a criterion set by most airlines: they refuse to carry live animals if daytime temperatures reach over 85°F (29.4°C) anywhere that the plane lands. Use caution when transporting in hot weather. Road conditions should be investigated, including any construction detours that may cause delays. Storms should be anticipated.

Some trailers have a small ramp leading up to the floor of the trailer for loading and unloading. Others require that the animal step up to load. Llamas prefer to step up rather than walk up a ramp. It is unwise to pull or push a llama into a step-up trailer. A leg may slip beneath the floor of the trailer and be fractured if the animal lunges forward. Appropriate training prior to loading would obviate problems of this nature.

Trailer flooring must be considered. Although llamas are surefooted and can generally walk on smooth surfaces, a bare floor in a trailer, whether it is metal or wood, is not appropriate. Rubber mats or indoor/outdoor carpeting are most desirable because they provide footing and insulation from both heat and cold and are softer to lie on. Straw is often used, but although it may provide insulation in cold weather, it does not provide footing. It does absorb urine. It is unwise to use straw for bedding on long journeys, especially if no feed is supplied. Hungry or bored animals may eat the straw, and if unaccustomed to it, may develop digestive upsets. Sand may be used, but wood shavings or sawdust become imbedded in the fiber coat and are difficult to remove.

Llamas may be trained to use different water containers, including a flowing stream or pool. If the animal is not familiar with a variety of water sources, water it with a plastic or metal bucket for a few days before beginning a trip. Animals may be reluctant to drink sufficient new-source water, which in hot weather may be disastrous. The odor or taste of new water may be disguised by pre-training the animal to drink water containing two drops of vanilla extract or oil of eucalyptus. New water can then be similarly treated.

Llamas may be unwilling to defecate or urinate in a strange environment. A periodic opportunity should be provided for them by stopping for a rest so that they may stand, or if off loading is feasible, to do so. It may be desirable to carry a container of dung, so that at a rest stop or at the destination a few pellets may be spread in an appropriate spot and the llama led to investigate them.

Moving a Recumbent Llama. Moving a recumbent llama[11,12] may be accomplished using the following technique. A plastic or canvas tarp that is longer than the length of the animal may be rolled up on one side and placed next to the animal that has been moved into the sternal position (Fig. 55-13). Allow the animal to resume lateral recumbency so that the tarp can be unrolled beneath the llama. Do not roll the animal on its back onto the tarp; rolling may cause regurgitation and possibly aspiration pneumonia. Rather, partially roll the animal from lateral to sternal recumbency and push the roll under it. Then, both sides of the tarp may be rolled up to form a hand-hold for those who will lift and carry the animal. The head and forequarters

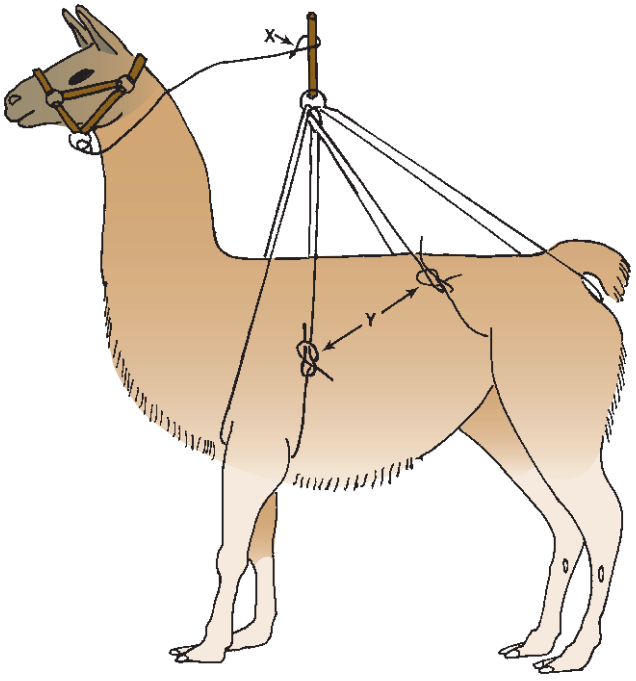

Figure 55-14. Diagram of a rope sling for a llama.

Figure 55-15. Placement of a rope sling on a horse.

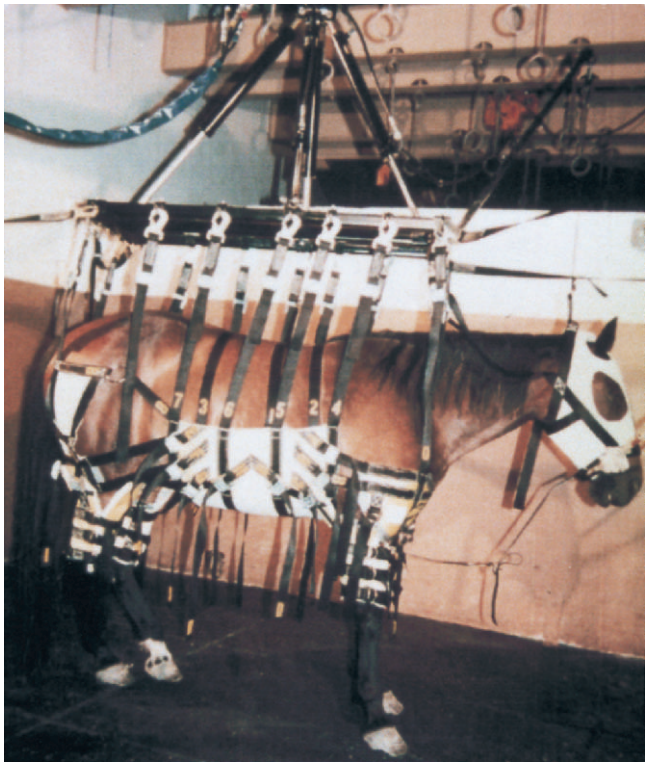

Figure 55-16. A commercial equine sling. (Photo courtesy University of California, Davis, Center for Equine Health.)

Figure 55-17. A mule being transported in a sling hung beneath a helicopter. (Photo courtesy University of California, Davis, Center for Equine Health.)

should be kept higher than the hindquarters to avoid passive regurgitation.

For llamas, it may be appropriate to rig a temporary sling inside a trailer or truck using a large bath towel or piece of canvas. Cut four leg holes, corresponding to the distance between the legs of the llama. Gently lift a forelimb and insert the foot through one hole and then repeat with the other forelimb, then repeat with the hind limbs. Fasten a small rope to each corner by first folding the corner of the towel over a small rock (1 inch [2.5 cm]), and then tie a slipknot around the cloth and the rock and pull it tight. The ropes may then be tied to the side of the trailer or partition. The feet should remain on the floor of the trailer and a person should ride beside the animal.

Field Evacuation of Injured Animals

Field evacuation of injured or seriously sick trek members by helicopter has become a viable option for all but the most remote wilderness expeditions. Cost is not an important factor in evacuating humans, but it may have to be considered in animal evacuations.

Dogs may be taken into the cabin of a helicopter. Llamas and horses must be slung beneath the helicopter. A sling for a short journey of a llama may be fashioned with four rope loops, all brought together over the back of the llama (Fig. 55-14).

A sling for a horse may be constructed from a rope (Fig. 55-15).[10] The diameter and length of the rope used depends on the size of the animal. A 50-foot, $^1/_2$-inch (15-m, 1.2-cm) nylon climbing rope is suitable to lift a 1000-lb (454-kg) horse. First, double the rope, and then form a loose neck loop using a bowline knot. Slip the loop over the head and position the loop with the knot at the base of the chest. Bring the running segments between the front legs and back through the corresponding side of the neck loop. Cross the ropes over the back, and then bring the ropes between the hind legs. Be certain not to cross ropes underneath the animal. The ropes should pass on either side of the tail, up over the back and underneath all the ropes on the back. Take all slack out of the ropes and complete the sling by doubling the running ends back and tying with a halter tie. An attempt should be made to place the lifting site over the approximate center of gravity of the animal (just behind the withers). However, even if this is not done, the animal can still be lifted, as each leg is in its own loop sling. The animal cannot slip out of the sling.

A more elaborate sling is available commercially* and has been used in numerous large animal rescue operations (Figs. 55-16 and 55-17).

*CDA Products (Care for Disabled Animals), Mr. Charles Anderson, Potter Valley, CA, phone 707-743-1300; U.S. patent number 4831967-1967.

APPENDIX A: SOURCES OF VETERINARY DRUGS FOR EXPEDITIONS

Bayer Corporation
P.O. Box 390
Shawnee Mission, KS 66201

Fort Dodge Laboratories
PO Box 518
Fort Dodge, IA 50501

Hoechst Roussel Vet
Route 202-206
Box 2500
Somerville, NJ 08876-1258

Merck AgVet
Division of Merck & Co.
PO Box 2000
Rahway, NJ 07065

Pfizer Animal Health
235 East 42nd St
New York, NY 10017

Schering Plough Animal Health Corp.
PO Box 529
Galloping Hill Rd.
Kenilworth, NJ 07033

Taylor Pharmaceutical Co.
Decatur, IL 62525

Wyeth-Ayerst Pharmaceuticals
555 Lancaster Ave.
St. Davids, PA 19087

The references for this chapter can be found on the accompanying DVD-ROM.

56 Seasonal and Acute Allergic Reactions

Naresh J. Patel and Robert K. Bush

Allergic rhinitis is the most common atopic disease, with an estimated prevalence of 15% to 20%.[59] It is the second most prevalent chronic condition in the United States, outranked only by hypertension.[69] Allergic rhinitis may be classified as seasonal (commonly referred to as hay fever) or perennial. The seasonal form is caused by allergens released from the pollens produced by trees, grasses, and weeds, whereas dust mites, furred animals, and molds are commonly implicated in the perennial form. In temperate climates, sufferers of perennial allergic rhinitis with a seasonal component often experience worsening symptoms during the warmer months because of the additional burden of pollen and outdoor mold exposure.

Although anaphylaxis is not as common as allergic rhinitis, it represents the most severe form of allergic reaction and can lead to a serious, including fatal, outcome if not treated promptly. The exact prevalence of anaphylaxis is not known. Based on extrapolation from data, the estimated risk of anaphylaxis per person in the United States is 1% to 3%.[76] Anaphylactic reactions are probably under-recognized and under-reported, particularly in cases of unexplained death. In one study, elevated tryptase levels, which is often found during acute anaphylaxis, were found in 13% of 68 patients postmortem.[57] Food and medications are the most common causes of anaphylaxis. Each year approximately 40 deaths in the United States are attributed to anaphylaxis resulting from bee stings.[2]

ALLERGIC RHINITIS

Allergic rhinitis is a chronic immunoglobulin E–mediated (IgE-mediated) inflammatory disease of the nasal mucosa. It is the most common atopic disease, and for reasons not yet clear, its prevalence is increasing worldwide. The economic impact of allergic rhinitis is impressive; the estimated total annual costs in medications and physician visits is $3.5 billion.[68] The disease usually manifests in early childhood or adolescence and peaks in the second or third decade of life. The disease does not appear to favor any sex, ethnic group, or race. The most important risk factor in the development of allergic rhinitis is a family history of atopy, especially with early onset of disease. In comparison with the general population, the risk is 30% greater if one parent or sibling is atopic, and 50% greater if both parents are affected.[44] On the other hand, lack of complete concordance for atopy in identical twins emphasizes the importance of environmental factors in disease development.[4] Allergic rhinitis, like other atopic conditions such as asthma and atopic dermatitis, tends to cluster in families.

Pathophysiology

Acute allergic reaction and late inflammatory events are important components in the pathogenesis of allergic rhinitis. Type I immediate hypersensitivity accounts for most of the acute clinical manifestations of allergic rhinitis, and cells such as eosinophils, basophils, and T lymphocytes play important roles in late inflammatory events. Production of allergen-specific IgE (sensitization) antibodies forms the underlying basis of immediate hypersensitivity; atopy is defined as the genetic predisposition to develop allergen-specific IgE antibodies. The sensitization process requires a cooperative effort between CD4 T lymphocytes and B lymphocytes (Fig. 56-1). It begins with presentation of an allergen to CD4 T lymphocytes by antigen-presenting cells (e.g., macrophages) in the context of a major histocompatibility complex. Cytokines released from CD4 T lymphocytes as a result of this interaction cause differentiation of B lymphocytes into immunoglobulin-secreting plasma cells. This differentiation leads to isotype switching (production of specific antibody types) within the plasma cells. For example, release of cytokine interleukin (IL)-4 or IL-13 from T lymphocytes promotes IgE switching.[12] Once allergen-specific IgE antibodies are produced, subsequent exposure and binding of the allergens to IgE molecules on the surface of mast cells results in cross-linking of the IgE molecules. Consequently, mast cells or basophils degranulate and release preformed and newly synthesized mediators. The prototype preformed mediator is histamine, and the newly synthesized mediators include those of the arachidonic acid pathway (leukotrienes, prostaglandins, and platelet-activating factor), neuropeptides (e.g., substance P), and cytokines (e.g., IL-4, IL-5).

The release of chemical mediators has various pathologic and clinical consequences. Sneezing and itching result from histamine stimulation of its receptors on sensory nerve endings. Rhinorrhea results from increased vascular permeability produced by all the mediators, and leukotrienes and prostaglandins are believed to play major roles in nasal congestion.[71] Our knowledge of the pathophysiology of allergic rhinitis has been greatly enhanced by nasal challenge studies.[24] It is now known that the allergic reaction consists of an early phase characterized by mast cell or basophil degranulation, and a late phase, which occurs 4 to 6 hours after the early phase (see Figure 56-1). The hallmark of the late-phase reaction is an influx of inflammatory cells, such as eosinophils, basophils, and T lymphocytes.[3] For

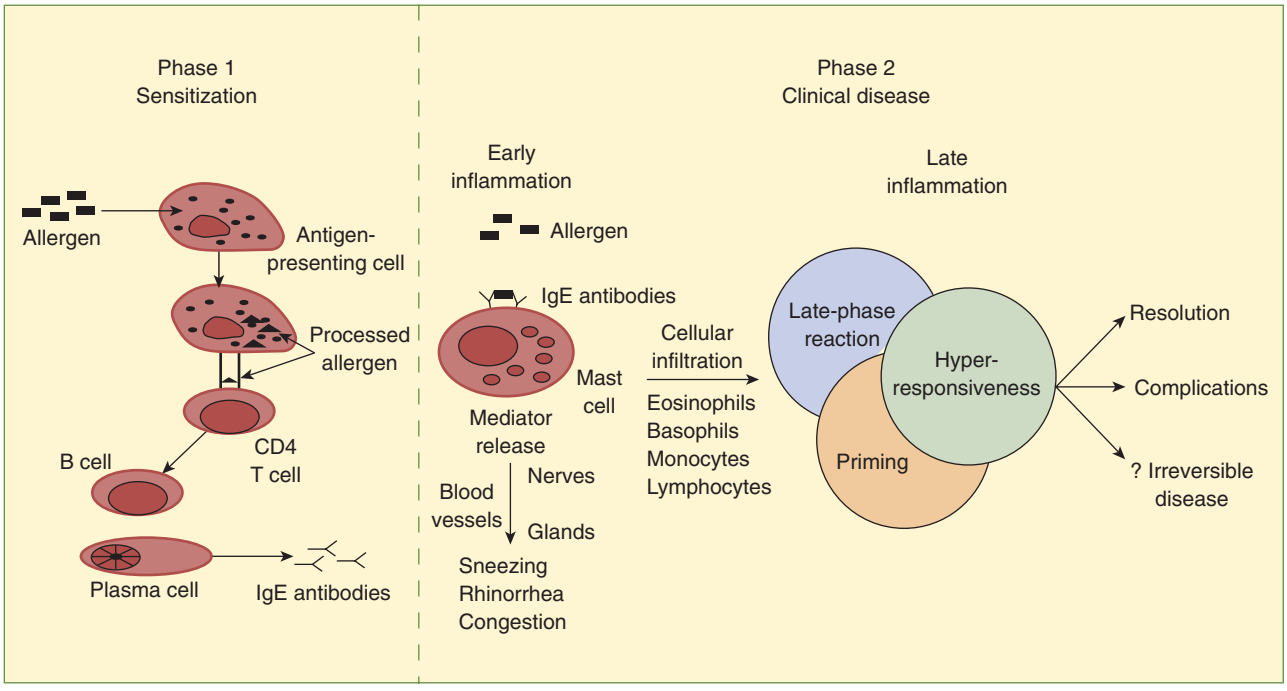

Figure 56-1. The natural history of allergic rhinitis (a simplified schematic). During phase 1, the individual becomes sensitized to an allergen. During phase 2, clinical disease develops. An overwhelming majority of individuals have an early response on reexposure to the allergen. The activation of mast cells and the release of mediators dominate the early response. After the early response, most individuals have cellular infiltration of the nasal mucosa, causing late inflammatory events, including the spontaneous recurrence of release of mediators (late-phase reaction), hyperresponsiveness to irritants, and increased responsiveness to the allergen (priming). *Circles* indicate the heterogeneity of these late inflammatory events. Inflammation can resolve spontaneously, cause a complication, or lead to an irreversible form of chronic rhinitis. (From Naclerio R: N Engl J Med 325:860, 1991.)

example, basophils cause further histamine release, and T lymphocytes release additional cytokines that enhance IgE production (via IL-4) and eosinophil activation (via IL-5). As a result of further inflammatory activity by these cells, there is recrudescence of symptoms many hours after the initial allergen exposure. Leukotrienes, prostaglandins, and cytokines released in the early-phase reaction play an important role in recruiting the late-phase cellular components to the inflammatory site. Although inhaled corticosteroids block both the early- and late-phase reactions, systemic corticosteroids block only the late-phase reaction.

Allergens

Airborne allergens are the primary etiologic agents of allergic rhinitis. Although many potential allergenic proteins exist, relatively few are clinically important and even fewer have been isolated and characterized. Allergens are low-molecular-weight proteins or glycoproteins capable of eliciting a type I immediate hypersensitivity reaction (production of IgE antibodies). Allergens can be conveniently divided into those that exist in the outdoor environment and those that exist in the indoor environment. In the outdoors, pollens of trees, grasses, and weeds, and certain fungi (e.g., *Alternaria* and *Cladosporium* species) most commonly provoke symptoms. Pollen-sensitive individuals typically experience seasonal rhinitis in predictable time intervals from one season to the next. Although fungi are more ubiquitous in nature and do not have the distinct seasons that pollens have, both outdoor and indoor fungi thrive in moist and humid environments. Therefore, in temperate climates, fungi counts start to rise in spring, with a peak in mid to late summer. Indoor allergens include fungi, furred animals, cockroaches, and dust mites. Unlike outdoor allergens, which typically cause seasonal symptoms, indoor allergens often cause perennial symptoms. However, the symptoms are less drastic in nature, often making diagnosis more difficult.

Pollens. Pollination in higher-order plants consists of the transfer of the male gametophyte to the female gametophyte. In this process, pollen grains serve as vectors for male gametophytes. Of the different types of pollen-producing plants, flowering plants (including trees, grasses, and weeds) are the most important from an allergic standpoint. Flowering plants may be divided into those that rely on animal vectors (e.g., insects) for pollination (termed entomophilous) and those that depend on the wind (termed anemophilous). In general, only anemophilous plants (including trees, grasses, and weeds) cause allergic symptoms.[63] Pollens of entomophilous plants do not achieve high airborne concentrations. In temperate climates, pollination of trees, grasses, and weeds occurs in predictable time intervals in a given region. Therefore, it is important to know the relevant local botany and respective pollination seasons (Table 56-1 and Fig. 56-2).

As shown in Figure 56-2, tree pollination marks the onset of allergy season in most parts of North America. It begins as early as mid-January in the Southwest or early April in the Northeast, and it terminates between early and late May. Trees of

TABLE 56-1. Selected Trees, Grasses, and Weeds of Allergenic Significance*

REGION (STATES)	TREES	GRASSES	WEEDS
Northeast	Birch	Orchard	Sheep sorrel
(ME, NH, VT, NY, PA, NJ, MA, RI, CT)	Elm	Timothy	Plantain
	Maple	June	Russian thistle
	Poplar	Sweet vernal	Giant ragweed
	Oak	Bluegrass	Short ragweed
Mid-Atlantic	Birch	Orchard	Plantain
(DE, MC, DC, VA, NC, SC)	Elm	Timothy	Dock
	Maple	Bluegrass	Sage
	Hickory	June	Short ragweed
	Oak	Bermuda	Giant ragweed
North Central	Ash	Orchard	Plantain
(OH, KY, WI, MI, IA, WI, northern MO, IL, IN, TN)	Elm	Timothy	Dock
	Maple	Bluegrass	Russian thistle
	Willow	June	Short ragweed
	Box		Giant ragweed
	Elder		
Pacific Northwest	Alder	Timothy	Dock
(WA, NV, OR, northern CA)	Birch	Bluegrass	Plantain
	Maple	Fescue	Russian thistle
	Oak	Rye	Nettle
	Walnut	Redtop	Sagebrush
Plains	Elm	Timothy	Marsh-elder
(NE, MN, eastern MT, Dakotas)	Oak	Orchard	Russian thistle
	Box	Bluegrass	Western hemp
	Elder	Bermuda	Short ragweed
	Willow	Redtop	Giant ragweed
	Maple		
Rocky Mountains	Cedar	Timothy	Sagebrush
(ID, WY, CO, UT, western MT)	Elm	Orchard	Russian thistle
	Ash	Fescue	Short ragweed
	Birch	Redtop	Giant ragweed
	Oak	June	
South	Cedar	Bermuda	Dock
(FL, GA, AL, TX, AR, southern MO)	Elm	Orchard	Pigweed
	Mulberry	Timothy	Russian thistle
	Poplar	Saltgrass	Giant ragweed
	Oak		Short ragweed
Southwest	Cedar	Bermuda	Sagebrush
(western TX, NM, AZ)	Ash	Johnson	Russian thistle
	Mulberry		Saltbush
	Oak		Kochia
	Olive		Short ragweed
Southern California	Ash	Bermuda	Nettle
	Walnut	Saltgrass	Bur ragweed
	Elm	Brome	Russian thistle
	Oak		Sage
	Olive		Western ragweed

*Plants are listed in the order of bloom for each region. Grasses are listed by prevalence in each region.
From Sicherer SH, Eggleston PA: Environmental allergens. In Lieberman P, Anderson JA (eds): Allergic Diseases: Diagnosis and Treatment. Totowa, NJ, Humana, 1997.

allergenic significance in various regions are listed in Table 56-1. Grass pollen, with its worldwide distribution, is a significant source of allergen exposure and a major cause of allergic rhinitis in sensitive individuals. In frost-free areas of North America, grass pollen may be present year-round, whereas in temperate climates, grass pollination peaks between mid-May and mid-July. Important grass types are rye, timothy, Kentucky bluegrass, orchard, Johnson, and Bermuda. Unlike ragweed and tree pollen allergens, grass pollen allergens show extensive cross-reactivity among different species, and allergic individuals are generally sensitive to many of these species. Weeds represent the final pollen producers of the season, typically generating pollen from mid-August through mid-October. Although a variety of weeds cause regionally significant allergies, ragweed is found in most parts of North America.

Fungi. Numerous allergenic fungi are responsible for both seasonal and perennial symptoms. The three most important classes of allergic fungi are zygomycetes (e.g., *Rhizopus*, *Mucor*), basidiomycetes (e.g., rust, smut, *Ganoderma*), and

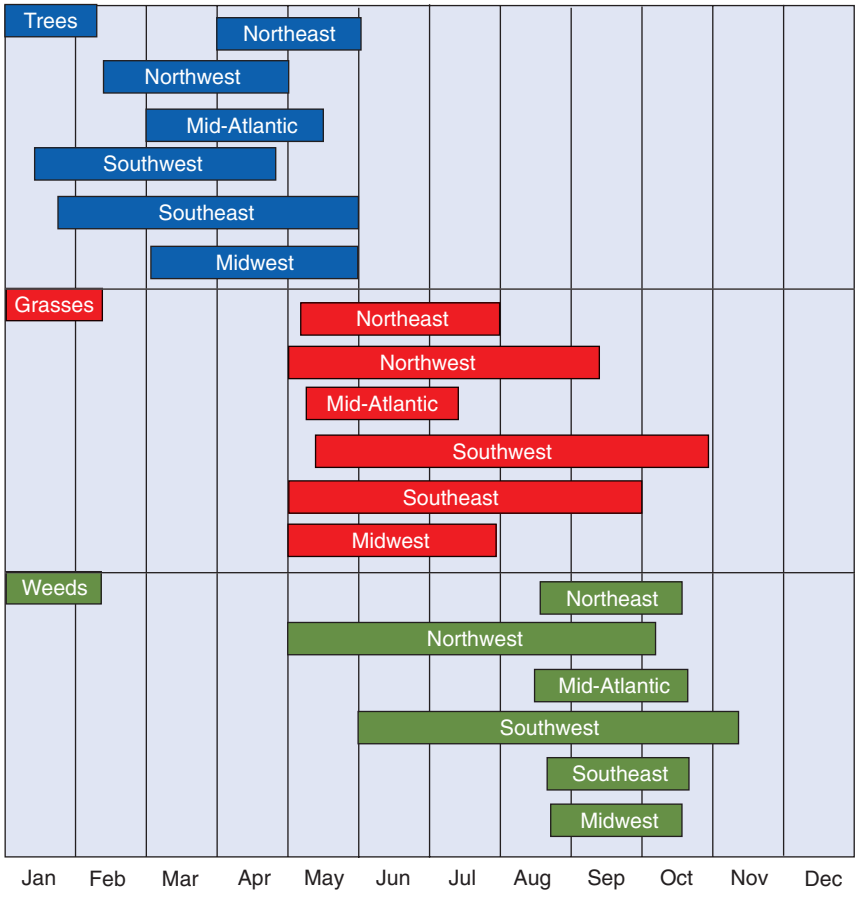

Figure 56-2. Pollen seasons by region in the continental United States. (From Sicherer SH, Eggleston PA: Environmental allergens. In Lieberman P, Anderson JA [eds]: Allergic Diseases: Diagnosis and Treatment. Totowa, NJ, Humana, 1997.)

ascomycetes (e.g., *Cladosporium, Penicillium, Aspergillus, Alternaria, Epicoccum*). Among the outdoor fungi, *Cladosporium* and *Alternaria* are the most abundant and cause significant symptoms in sensitive individuals. Although fungi can survive in various climates, they grow best in warm, humid environments. The peak fungal season in many areas is in middle to late summer and fall, with a marked reduction after the first frost in temperate climates. Fungi levels may rise in spring, when decaying vegetation is uncovered after the snow has melted. Common outdoor sources of fungal growth are leaves, moist debris, and soil; therefore, outdoor activities, such as raking, farming, and mowing, significantly increase exposure.

Dust Mites. Dust mites are microscopic arachnids that are close relatives of ticks and spiders. They feed on human epidermal scales and, like fungi, prefer a warm, humid environment. Dust mites are the most common indoor allergens and can cause significant perennial symptoms in sensitive individuals. Although there is no good correlation between exposure level and degree of rhinitis symptoms, a level of 10 µg/g of dust is considered a risk factor for development of acute asthma symptoms.[50] Common indoor sources of dust mite exposure include mattresses, pillows, blankets, upholstery, and stuffed toys. Dust mites may proliferate in sleeping bags stored in humid environments (e.g., damp basements).

Animals. Animals, especially cats, can be highly allergenic and in sensitized individuals can cause significant symptoms. Cats and dogs are the most common pets in the United States, with more than one third to one half of all homes housing at least one cat or dog.[60] Birds, rabbits, hamsters, guinea pigs, rats, and mice are some other potentially allergenic pets. The major allergens of the cat and the dog are found in their saliva and sebaceous glands. The cat allergen can remain a potential source of allergy months after the removal of the cat, because of its light, sticky qualities.

Outdoors, inhalation of the emanations of moths, locusts, beetles, and flies may cause allergic symptoms. Residents of the area around western Lake Erie may experience allergic symptoms caused by the mayfly.[73] Finally, the cockroach is potentially allergenic and is frequently encountered in heavily infested, crowded, or multifamily dwellings.

Functions of the Nose

The main functions of the nose—to warm, humidify, and filter air—are possible because of turbulent airflow resulting from increased surface area provided by the three turbinates (superior, middle, and inferior) on each side. The nose's functions are mainly controlled by the underlying neurovascular system. Blood supply to the nose is via the ophthalmic (branch of the internal carotid) and internal maxillary (branch of the external

carotid) arteries. Neural innervations include the sensory (via the trigeminal nerve, which is responsible for the sneezing reflex) and autonomic (parasympathetic and sympathetic) nervous systems. Nasal congestion is proportionally related to the amount of blood pooling in the cavernous sinusoids (located within the turbinates) and to the degree of mucosal edema. Sinusoid filling and emptying is controlled by the autonomic nervous system. Vasodilatory parasympathetic stimulation allows the sinusoid reservoir to fill via the opening and closing of capillary and postcapillary venule sphincters, respectively. Conversely, sympathetic stimulation contracts the capillary sphincter and relaxes the postcapillary venule sphincter, allowing the reservoir to empty.

In most people, cyclical swelling and shrinking of the turbinates between the two sides is inherent. This nasal cycle, which is approximately 1 to 4 hours in length, results from alternating sympathetic discharge and is responsible for the sensation of alternating unilateral nasal blockage experienced by allergic rhinitis sufferers.[20]

Another major function of the nose is olfaction. The olfactory area is located at the roof of the nasal cavity above the middle turbinate. Significant turbinate edema can lead to poor ventilation of this area and associated hyposmia, which is common among allergic rhinitis sufferers.

Clinical Evaluation

A thorough, careful history is crucial in the evaluation of allergic rhinitis and can often help determine whether an individual is allergic or not. Allergy testing is used to confirm the underlying clinical suspicion. Symptoms of allergic rhinitis can range from intermittently mild to incapacitating, such as during peak pollen season. The hallmark of allergic rhinitis is temporal correlation of symptoms with allergen exposure.[43] The most common symptoms of allergic rhinitis—sneezing, nasal congestion, rhinorrhea, and pruritus of the nose and eyes—are nonspecific. Itching and sneezing are the most distinctive complaints associated with allergic rhinitis,[44] whereas nasal congestion seems to be more prominent in perennial rhinitis than in seasonal rhinitis. In some individuals, ocular symptoms predominate over nasal symptoms. Allergic rhinitis sufferers often experience the priming effect (an increase in sensitivity to allergens after repeated exposure) and hyper-responsiveness to nonallergenic environmental stimuli, such as tobacco smoke, strong odors, pollutants, and weather changes.[9]

Because the time intervals of pollination are predictable in a given season, the timing of symptoms is often helpful in identifying the responsible pollen(s). For example, in the Midwest, ragweed sufferers characteristically start experiencing symptoms in mid-August. However, because any number of allergens may cause symptoms in individuals with perennial rhinitis, determining the specific responsible allergen(s) can be difficult. Other conditions of the upper respiratory tract may mimic allergic rhinitis and must be considered. Complications of allergic rhinitis include sinusitis, otitis media with effusion, and asthma. Additionally, numerous studies have shown that allergic rhinitis may significantly impact quality of life, with effects such as sleep loss, fatigue, poor concentration, reduced productivity, and irritability.[26] Although a decline in their quality of life may seem like an obvious clue to the severity of the disease, patients may not be aware of this decline if they have been living with their condition for years and becoming accustomed to it.[67]

As is true for the symptoms described by the patient, many of the signs noted on physical examination are not exclusive to allergic rhinitis. Classically, the nasal mucosa is pale blue and edematous, but this color change is noted in only 60% of sufferers, and many individuals have erythematous mucosa.[13] In children, the "allergic salute" (upward nasal rubbing) and "allergic shiners" (dark, puffy circles under the eyes) may be present. Other notable signs include septal deviation, polyps, and an obvious foreign body. If congestion is profound, use of a topical decongestant such as oxymetazoline (Afrin) may improve visualization.

Allergy Testing

Because of the temporal correlation of symptoms with a particular season, diagnosis of seasonal allergic rhinitis can often be made clinically without the need for testing. However, given the ambiguity of various allergen exposures in perennial rhinitis, allergy testing is generally recommended. Allergy testing may be performed either by skin testing or by the radioallergosorbent test (RAST). In skin testing, which is widely accepted and more sensitive,[28] minute quantities of specific allergens are introduced under the skin, and a positive response depends on allergen-specific release of histamine. In the RAST, evidence of allergen-specific IgE is determined using the patient's serum. Given its lesser sensitivity, greater cost, and longer turnaround time for results, RAST is typically used in special circumstances only, such as for patients with severe eczema or dermatographism, in whom skin testing might be difficult to interpret.

Testing can pinpoint specific allergen sensitivities, which can be useful in providing allergen avoidance measures. Allergy testing must be performed if immunotherapy is being considered.

Differential Diagnosis

Because of the significant and overlapping symptoms of various rhinitis conditions, the differential diagnosis of rhinitis must be considered during the initial evaluation (Box 56-1). Rhinitis can be classified as acute or chronic. The most common cause of acute rhinitis is a viral infection, and allergic rhinitis may be confused with viral rhinitis. However, unlike the symptoms of allergic rhinitis, those of viral rhinitis usually do not persist longer than 2 weeks unless superseding sinusitis develops. A foreign body or trauma also can cause acute rhinitis. Unilateral symptoms are the hallmark of foreign body obstruction.

The nonallergic rhinitis with eosinophilia syndrome (NARES) is characterized by (1) symptoms similar to allergic rhinitis, (2) eosinophilia on nasal smear, and (3) a negative skin test.[25] Chronic sinusitis, which can occur as a complication of allergic rhinitis, commonly causes nasal congestion, sinus pressure, postnasal drip, cough, and diminished senses of smell and taste. Symptoms of chronic sinusitis, unlike those of acute sinusitis, are subtle and may require radiologic evaluation, preferably computed tomography. Systemic diseases, such as vasculitis and cystic fibrosis, may cause chronic rhinitis. Rhinitis medicamentosa, which usually results from overuse of topical α-adrenergic vasoconstrictors (e.g., oxymetazoline), is associated with profound nasal congestion caused by rebound effects on withdrawal of the decongestant, usually after 5 to 7 days of topical use. Medications that have been implicated in chronic rhinitis include nonsteroidal anti-inflammatory drugs (NSAIDs), angiotensin-converting enzyme inhibitors, and β-blockers.

Box 56-1. Differential Diagnosis of Rhinitis

ACUTE RHINITIS
Upper respiratory tract infection
Foreign body
Trauma

CHRONIC RHINITIS
Allergic
Nonallergic
NARES (nonallergic rhinitis with eosinophilia syndrome)
Chronic sinusitis
Systemic diseases
Vasculitis
Wegener's granulomatosis
Churg-Strauss syndrome
Cystic fibrosis
Sarcoidosis
Hypothyroidism
Rhinitis medicamentosa
Medications
Mechanical-anatomic obstruction
Nasal polyps
Foreign body
Tumor
Septal deviation
Adenoid hypertrophy
Gustatory rhinitis
Atrophic rhinitis
Rhinitis of pregnancy
Cerebrospinal fluid leak
Vasomotor (idiopathic) rhinitis

Mechanical and anatomic abnormalities should always be sought on physical examination. Nasal polyps have a pearly, smooth, and peeled grape-like appearance and are often bilateral. Nasal polyps may be associated with chronic sinusitis, asthma, and aspirin sensitivity.[58] Unilateral symptoms should always heighten suspicion for a foreign body or tumor. Some degree of septal deviation may be found in most people. Therefore, unless symptoms are severe, treatment is unnecessary. In children, adenoid hypertrophy must be considered.

Symptoms of gustatory rhinitis are caused by a cholinergic response that occurs after such stimuli as eating, running, and exposure to cold air. Atrophic rhinitis is a rare condition that manifests in older adults as a thick, bilateral, odorous discharge with little nasal congestion.[19] Rhinitis of pregnancy should be considered in the pregnant woman with congestion, especially if there is no previous history of rhinitis. Although rare, cerebrospinal fluid (CSF) leak must be considered in anyone with clear rhinorrhea and a history of central nervous system trauma. Testing for the presence of glucose in the nasal mucus is helpful in making the diagnosis. The diagnostic test of choice to detect CSF leak is an assay for a β-2 transferrin level in the nasal mucus.[61]

Often a diagnosis of exclusion, vasomotor (idiopathic) rhinitis is a common cause of nonallergic rhinitis in adults. Although the term implies a cause relating to vascular or neurologic dysfunction, the mechanism of vasomotor rhinitis is poorly understood. The symptoms of vasomotor rhinitis are frequently obstructive rather than secretory. Therefore, nasal congestion and sinus pressure predominate over sneezing and rhinorrhea.[36] Individuals with vasomotor rhinitis typically experience perennial symptoms that may be triggered by changes in weather, spicy foods, and irritants, such as smoke, strong scents, and chemicals.

Treatment

Avoidance of the offending allergens is the first step in the treatment of allergic rhinitis. However, the ubiquitous presence of the allergens in the environment, or being unable to part from a beloved pet, make avoidance difficult, and pharmacologic intervention is often necessary. The optimal treatment plan should be individualized on the basis of symptoms, seasonal patterns, and presence of possible comorbid conditions. The ideal medication is efficacious, cost effective, compliance friendly, and has minimal side effects. Furthermore, based on the pathophysiology of allergic rhinitis, the best therapeutic agent is one that effectively addresses both early- and late-phase reactions.[64] Currently, the main classes of medications available for treatment of allergic rhinitis are antihistamines, decongestants, topical or oral glucocorticoids, anticholinergics, cromolyn, and leukotriene receptor antagonists. In addition, immunotherapy should be considered in moderately to highly sensitive individuals.

Avoidance. Although it may be difficult to avoid pollen because of its widespread distribution, certain commonsense measures can be taken to decrease exposure. Outdoor activities should be limited or completely avoided on days when pollen counts are high. On any given day, pollen counts typically peak in the late morning to mid-afternoon.[74] Limiting activities such as leaf raking, lawn mowing, and farming can reduce exposure to outdoor fungi. Because fungi thrive in moist, humid environments, important indoor control measures include dehumidification, proper ventilation, fungicide use in contaminated areas, and removal of sources of fungal growth. Dust mite exposure can be reduced by several methods, including covering mattresses and pillows with allergen-proof encasings, frequently washing bedding in hot water, and removing carpeting or treating it with denaturing chemicals. Complete removal of the offending animal from the environment is the best treatment for animal allergy. However, this option is often not exercised. If the pet stays in the home environment, then bathing the pet at regular intervals and keeping it out of the bedroom may be helpful. Use of high-efficiency particulate air (HEPA) filters has been shown to be beneficial in removing animal allergens from the environment.[10]

Antihistamines and Decongestants. Antihistamines have been used for 50 years and are considered by many to be the first line of therapy in the treatment of allergic rhinitis.[51] They are most effective against seasonal rhinitis in which sneezing, itching, rhinorrhea, and watery eyes are the most prominent symptoms. However, only 33% to 50% of seasonal allergic rhinitis sufferers obtain complete relief with antihistamine therapy alone.[6] Antihistamines have little or no effect on nasal congestion because of the minor role of histamine in the pathophysiology of congestion. For this reason, antihistamines are usually inadequate for treatment of perennial rhinitis, in which nasal congestion is often a predominant symptom.

TABLE 56-2. Newer-Generation Antihistamines

CHEMICAL NAME	TRADE NAME	DOSAGE		
		ADULT	CHILDREN (<12 YEARS)	MINIMUM AGE
Cetirizine	Zyrtec	10 mg qd	2.5 to 10 mg qd	6 mo
Desloratadine	Clarinex	5 mg qd	1.0 to 2.5 mg qd	6 mo
Loratadine	Claritin	10 mg qd	2 to 10 mg qd	2 yr
Fexofenadine	Allegra	60 mg bid to 180 mg qd	30 mg bid	6 yr
Azelastine	Astelin	2 sprays per nostril, bid	1 spray per nostril, bid	5 yr

TABLE 56-3. Nasal Corticosteroids*

GENERIC NAME	TRADE NAME	RECOMMENDED DOSE (EACH NOSTRIL)	AMOUNT PER SPRAY (μg)	MINIMUM AGE (YR)
Beclomethasone	Beconase AQ	1 to 2 sprays bid	42	6
Budesonide	Rhinocort Aqua	1 to 4 sprays qd	32	6
Flunisolide	Nasarel	2 sprays bid	29	6
Fluticasone	Flonase	1 to 2 sprays qd	50	4
Mometasone	Nasonex	1 to 2 sprays qd	50	2
Triamcinolone	Nasacort AQ	1 to 2 sprays qd	55	6

*All preparations are aqueous based.
bid, twice a day; qd, every day.

Antihistamines act by occupying H_1 receptors on cells and thereby blocking histamine binding. Older- and newer-generation antihistamines are equally effective. However, the newer-generation antihistamines have gained tremendous popularity because their use is associated with a much lower incidence of sedation and fewer anticholinergic side effects. Commonly used older-generation antihistamines, most of which are available over the counter, are chlorpheniramine, diphenhydramine, brompheniramine, clemastine, and hydroxyzine. The duration of action of brompheniramine and diphenhydramine is 6 to 10 hours, whereas that of chlorpheniramine and of hydroxyzine is 24 and 36 hours, respectively. Although sedation is a well-known side effect of the older-generation antihistamines, many patients are unaware of its effects on cognitive functions[41] and impairment in driving performance.[47]

Newer-generation antihistamines are used widely and have been shown to be safe, effective, and well tolerated.[27] Moreover, newer-generation antihistamines are more specific in their actions and do not have anticholinergic or anti–α-adrenergic activity. Five newer-generation antihistamines, including four oral and one intranasal, are currently available (Table 56-2). With the exception of azelastine, all others are given once daily. All five are approved for use in children; cetirizine and desloratadine are approved for those who are at least 6 months old. Azelastine is the only H_1 receptor antagonist available in a nasal spray formulation. Studies have shown that azelastine is safe and effective for relieving most allergic rhinitis symptoms, including nasal congestion.[32] Although all antihistamines have a relatively rapid onset of action and may be used as needed, they are most effective if taken prior to exposure (e.g., before pollen season begins) or if used on a regular basis.

In some individuals, addition of a decongestant to the antihistamine may be helpful. Decongestants act by stimulating α-adrenergic receptors and thereby reducing blood supply to the sinusoids. Most are short acting (4 to 6 hours) and available over the counter with or without antihistamine combination. Pseudoephedrine is the most commonly used decongestant. Fexofenadine, desloratadine, loratadine, and cetirizine are available in combination with a decongestant. Insomnia and irritability are the most common side effects of decongestants, although individual tolerance varies widely. Individuals with hypertension, glaucoma, or cardiac arrhythmias should use decongestants cautiously or preferably avoid their use altogether. Topical decongestants (e.g., oxymetazoline) have a fast onset of action (within minutes) and therefore provide rapid relief of symptoms. Because of the risk of rhinitis medicamentosa, topical decongestant use should be limited to no more than 3 to 5 days.

Nasal Corticosteroids. Nasal corticosteroids are the gold standard of treatment for allergic rhinitis. Numerous well-designed studies have shown that nasal corticosteroids are superior to antihistamines and cromolyn sodium.[5,7] With the exception of ocular symptoms, nasal corticosteroids are effective in controlling all symptoms of allergic rhinitis. Nasal corticosteroids are often the first line of therapy for patients with perennial rhinitis. Corticosteroids control the rate of protein synthesis by either inducing or suppressing gene transcription within the cell.[31] The inhibitory effect of all corticosteroids on the late-phase reaction is well established, but studies have shown that nasal corticosteroids, unlike their oral counterparts, also inhibit the early-phase reaction.[1]

Several preparations of nasal corticosteroids are currently available (Table 56-3). All are equally effective; hence, selection is based on convenience, cost, and delivery system. All nasal corticosteroids are in aqueous form and are administered either once or twice a day. Because of their relatively slow onset of action, the maximal benefit of nasal corticosteroids may not be

realized until 1 to 3 weeks after the initiation of therapy. For individuals with seasonal rhinitis, it is best to start nasal corticosteroid therapy shortly before the onset of pollen season. There is no role for regular use of oral corticosteroids in the treatment of allergic rhinitis. However, a short course (7 to 10 days) of oral corticosteroids may be appropriate when symptoms are severe, such as during peak pollen season. In addition to providing quicker relief, oral corticosteroids may facilitate improved delivery of nasal corticosteroids to the nasal mucosa.

The most common side effects of nasal corticosteroids are local irritation and bleeding, reported in approximately 10% and 2% of patients, respectively.[43] The incidence of side effects is higher during winter months because of drier conditions. Although rare, nasal septal perforation has been reported with use of nasal corticosteroids.[56] If significant septal ulceration and crust formation is noted, therapy should be discontinued for a few days and nasal hygiene with saline spray should be instituted. Long-term studies have shown no evidence of mucosal atrophy associated with the use of nasal corticosteroids.[42] Because of the rapid metabolism of nasal steroids, there is generally no hypothalamic-pituitary-adrenal axis suppression. Therefore, there is little risk of systemic side effects.

Leukotriene-Receptor Antagonists. Leukotriene-receptor antagonists (LTRAs), which were previously indicated only for asthma, are now approved for use in allergic rhinitis. Cysteinyl leukotrienes, which are products of the arachidonic acid pathway, are important mediators of the allergic inflammatory effects. The leukotrienes cause nasal congestion that results from vasodilation. Because leukotrienes play a major role in the pathophysiology of nasal congestion, the LTRAs may significantly reduce nasal congestion.[30] Additionally, they also cause mucus production and therefore rhinorrhea. Various studies have demonstrated that leukotriene levels are increased in nasal lavage in both seasonal and perennial allergic rhinitis.[62] LTRAs block binding of cysteinyl leukotrienes to the CYS-LT1 receptor in the respiratory tract.

Several studies have compared the effects of LTRAs with those of antihistamines and nasal corticosteroids. In one study comparing the LTRA montelukast with loratadine, both were found equally effective in alleviating nasal symptoms.[49] However, in a different study comparing montelukast with the nasal corticosteroid fluticasone propionate, montelukast was significantly less effective than fluticasone propionate in relieving nasal symptoms.[37] The effectiveness of combining LTRAs and antihistamines have also been studied. One study showed that the combination of montelukast with loratadine was more effective than either agent alone.[40] However, other studies have refuted this finding.[45] In conclusion, the studies of LTRAs in allergic rhinitis show that they perform as well as antihistamines, but not as well as nasal corticosteroids.

Montelukast and zafirlukast are two currently available oral LTRAs. The recommended dosage of zafirlukast is 20 mg twice daily for adults and children 12 years of age or older, and 10 mg twice daily in children 5 through 11 years of age. The recommended dosage of montelukast for adults and adolescents 15 years of age and older is 10 mg at bedtime. Pediatric dosing is 5 mg and 4 mg at bedtime for ages 6 to 14 years and 6 months to 5 years, respectively. In general, LTRAs appear to be well tolerated. Occasional liver toxicity has been reported with zafirlukast,[52] but not with montelukast.

Other Medications. Other classes of medications available for treatment of allergic rhinitis are cromolyn sodium and nasal anticholinergic drugs. Although much less potent than nasal corticosteroids, cromolyn is very safe. Its mechanism of action is not completely understood, but it is believed to inhibit mast cell degranulation. For optimal relief, it requires regular use and frequent dosing (three to four times daily). It is useful as a prophylactic treatment prior to known exposure, such as visiting someone with a cat. If rhinorrhea is the predominant symptom, then use of a nasal anticholinergic, such as ipratropium, may be beneficial. An additional side benefit of ipratropium may be reduction of postnasal drip. The recommended dosage of ipratropium is one to two sprays in each nostril two to four times a day. The main side effect of intranasal ipratropium is nasal dryness.

Immunotherapy. Allergen immunotherapy is a form of immunomodulation in which a state of tolerance is achieved by injecting gradually increasing amounts of allergen extracts into a sensitized person. Although the precise mechanisms of immunotherapy are unknown, several immunologic changes have been observed, including induction of allergen-specific IgG "blocking antibodies," decreases in allergen-specific IgE, modulation of mast cell or basophil function, and increases in suppressor T cells (CD8).[17]

The efficacy of immunotherapy in patients with allergic rhinitis has been well established in numerous studies.[14,39] Most individuals who receive immunotherapy for allergic rhinitis obtain a variable degree of relief, enabling better symptom control with less medication. Immunotherapy should be considered in individuals who respond poorly to medical therapy and in individuals who experience significant side effects to medications. Additionally, consideration may be given if allergen avoidance is not possible. In immunotherapy, weekly injections of allergens at escalating dosages are administered until a maintenance dosage is reached, generally within 4 to 6 months. Thereafter, one or more injections are given monthly, depending on the number of allergen sensitivities. Although there are no data regarding optimal duration of immunotherapy, the general recommendations are from 3 to 5 years.[72]

The most common adverse reactions of immunotherapy are local, consisting of erythema, edema, and pruritus at the site of injection. The risk of systemic reaction is approximately 0.1% to 1% per injection.[20] Manifestations of a systemic reaction are variable and may include urticaria, flushing, rhinitis, bronchospasm, laryngospasm, abdominal cramping, and hypotension. The risk of systemic reactions is much greater in individuals with poorly controlled asthma. Therefore, immunotherapy is generally not recommended in asthmatics with a forced expiratory volume in 1 second (FEV_1) less than 70% of predicted. Because most systemic reactions occur within 20 to 30 minutes after injection, recipients should be observed in the physician's office for this time frame. Some practitioners require patients to carry epinephrine (e.g., EpiPen, Ana-Kit) to self-treat reactions that may occur after leaving the physician's office.

ANAPHYLAXIS

Anaphylaxis is a systemic life-threatening allergic reaction resulting from IgE-mediated mast cell and basophil degranulation. It is the most severe form of type I hypersensitivity reac-

Box 56-2. Classification of Anaphylactic and Anaphylactoid Reactions

ANAPHYLAXIS (IgE ANTIBODY MEDIATED)
Food
Drugs (penicillin, cephalosporins, insulin, sometimes aspirin and other NSAIDs)
Insect stings and bites
Other (exposure to antivenom or aquatic proteins)

ANAPHYLACTOID (NON-IgE ANTIBODY MEDIATED)
Direct stimulation of mast cells
Drugs (opiates, vancomycin)
Radiocontrast material
Physical stimuli (exercise, cold)
Idiopathic
Disturbances in arachidonic acid metabolism
Aspirin and other NSAIDs
Complement activation
Transfusion reactions
Immunoglobulin

IgE, immunoglobulin E; NSAIDs, nonsteroidal inflammatory drugs.

tion, and clinical manifestations may occur in various end organs, including the skin, upper respiratory tract, lower respiratory tract, gastrointestinal tract, and cardiovascular system. An anaphylactoid reaction is non-IgE antibody mediated but may be clinically indistinguishable from anaphylaxis (Box 56-2). Mechanisms of anaphylactoid reactions include direct release of mediators from mast cells and basophils (caused by opiates, radiocontrast material, e.g.), disturbances in arachidonic acid metabolism (caused by NSAIDs, e.g.),[15] and complement activation (caused by transfusion reactions, e.g.).

Etiology

The various causes of anaphylaxis can be categorized into those that are IgE antibody mediated and those that are non-IgE antibody mediated (see Box 56-2). Foods were the most frequent causative agents of anaphylaxis in one series of 179 subjects spanning 3.5 years, accounting for 36% of episodes.[75] Egg, cow's milk, wheat, soybean, peanut, tree nuts (e.g., hazelnut, walnut, cashew, almond), fish, and shellfish (e.g., shrimp, lobster, crab) account for more than 90% of all food-related anaphylactic reactions. In children, peanuts are probably the most frequent cause of food-induced anaphylaxis.[55] Individuals may accidentally ingest the allergenic food when it is disguised in cooking preparations or hidden by misleading labeling or contamination during the preparation process.

Drugs, especially antibiotics, may cause an anaphylactic or an anaphylactoid reaction. Penicillin and its derivatives are most often implicated, with reactions occurring in between 1 and 5 of every 10,000 courses of treatment.[23] Opiates and vancomycin cause drug reactions by directly degranulating mast cells and basophils, so prior sensitization is not required.

Aspirin and other NSAIDs can provoke allergic reactions that are either IgE or non-IgE antibody mediated, depending on the presence or absence of underlying asthma or cutaneous diseases (e.g., chronic urticaria). In the presence of asthma and chronic urticaria, disturbance in arachidonic acid metabolism is the predominant mechanism. Aspirin and other NSAIDs inhibit the cyclooxygenase pathway, leading to rapid synthesis of the lipoxygenase pathway products,[34] especially leukotrienes, which are important mediators of inflammation.

Important outdoor-related causes of anaphylaxis are envenomation (e.g., bee sting), contact with aquatic proteins, and antivenom therapy (e.g., for snakebite). The estimated incidence of insect sting anaphylaxis in the general population is 0.3% to 3%.[53] Bee sting anaphylactic reactions are especially common in individuals younger than 20 years but are more likely to be fatal in older adults. In general, children's reactions are milder (e.g., urticaria only) than adults' reactions. Individuals who experience a sting-related anaphylactic reaction have a 50% to 60% risk for suffering anaphylaxis after subsequent insect stings.[53] The offending insects vary depending on geographic location. In the United States, yellow jackets cause the most allergic reactions, whereas in Europe, honeybees and wasps cause majority of insect sting–related reactions. Although rare, anaphylaxis can result from bites inflicted by certain insects (e.g., the kissing bug *[Triatoma protracta]*, the deer fly *[Chrysops discalis]*,[22] and ticks).[8]

Physical stimuli may provoke anaphylactoid reactions. In exercise-induced reactions, symptoms typically arise after 5 minutes of moderate to heavy exercise, resolving within 30 minutes to 4 hours after exercise cessation.[70] About 50% of affected individuals are atopic and most engage in regular vigorous exercise. A coinciding factor, such as the ingestion of an allergenic food (e.g., shellfish)[38,48] or NSAID, may be necessary to induce this type of reaction—that is, exercise only or ingestion of the food only does not cause the reaction. In a cholinergic reaction, symptoms occur after exercise and after passive exposure to heat (e.g., hot showers, sweating, and anxiety). In cold-induced reactions, symptoms occur after exposure to a cold stimulus, such as being outside on cold days, holding cold objects, and eating cold foods.

Recurrent anaphylaxis without identifiable cause is known as idiopathic anaphylaxis. Although all the target organs of anaphylaxis may be affected, idiopathic anaphylaxis most commonly causes urticaria or angioedema, or both. The diagnosis of idiopathic anaphylaxis is one of exclusion. Individuals who suffer frequent episodes are less likely to undergo remission. Short-term treatment is the same as that for anaphylaxis resulting from other causes. Depending on the frequency of attacks, long-term treatment may require regular use of oral corticosteroids.

Complement activation plays a key role in anaphylactoid reactions resulting from the transfusion of blood products and immunoglobulins. Transfusion of an incompatible blood type can lead to cytotoxic anaphylactoid reactions if complement-fixing antibodies to formed elements of blood, such as red cells, white cells, and platelets, are present. In γ-globulin–related anaphylactoid reactions, immune complex aggregation occurs when antigen–antibody complexes activate complement.

Pathophysiology

Unlike the local pathologic and clinical manifestations of allergic rhinitis, the clinical consequences of type I immediate hypersensitivity in anaphylaxis are systemic. Histamine is the most important mediator and is responsible for most of the clinical manifestations. Important effects of histamine include vasodilation, increased vascular permeability, smooth muscle contraction, stimulation of nerve endings, and glandular secretion. These effects lead to various clinical manifestations, including

Box 56-3. Treatment of Anaphylaxis

GENERAL MEASURES

Place the individual in Trendelenburg's position.

Establish and maintain the airway. (If necessary, perform cricothyroidotomy.)

Administer oxygen as needed.

Place a venous tourniquet above the reaction site (e.g., insect sting) to decrease systemic absorption of the antigen.

Obtain intravenous access and infuse a volume expander (colloid-containing solution or normal saline) to achieve a systolic blood pressure of 90 mm Hg in an adult. Blood pressure may be briefly augmented by the application of military antishock trousers, which increases systemic vascular resistance.

EPINEPHRINE USE AND TREATMENT OF HYPOTENSION

Administer aqueous epinephrine, 1:1000, 0.3 to 0.5 mL (0.3 to 0.5 mg) subcutaneously or intramuscularly in the deltoid region. The epinephrine dosage for children is 0.01 mL/kg. Repeat once or twice as necessary at 15 to 20 minute intervals to control the signs and symptoms.

If the reaction is life threatening and if the patient does not respond to subcutaneous epinephrine, administer epinephrine intravenously. Mix 0.1 mL (0.1 mg) of 1:1000 aqueous epinephrine in 10 mL of normal saline (final dilution, 1:100,000) and infuse over 10 minutes. If hypotension persists, a continuous epinephrine infusion may be started by adding 1 mL (1 mg) of 1:1000 epinephrine to 250 mL of normal saline, creating a concentration of 4 µg/mL. Infuse this solution at a rate of 1 µg/min (15 minidrops/min). The rate of infusion can be increased to 4 to 5 µg/min if the clinical response is inadequate. In children and infants, the starting dosage is 0.1 µg/kg/min, up to maximum of 1.5 µg/kg/min. Intravenous epinephrine should be used with caution in persons older than 35.

If epinephrine and fluids are ineffective, intravenous dopamine infusion should be initiated. Mix 400 mg of dopamine in 500 mL of D_5W (800 µg/mL) and infuse at a rate of 2 to 20 µg/kg/min. The rate should be titrated using the blood pressure as a guide. Norepinephrine, a potent vasopressor, can be used to treat severe hypotension that is unresponsive to the administration of epinephrine, fluids, and dopamine. Mix 4 mg of norepinephrine in 1000 mL of D_5W (4 µg/mL) and infuse at a rate of 0.5 to 1 µg/kg/min.

In a refractory case in which the individual is not responsive to epinephrine (e.g., an individual on a β-blocker medication), administer a glucagon bolus at a dosage of 1 to 5 mg intravenously over 2 minutes. If necessary, a continuous infusion of 1 mg in 1000 mL of D_5W (1 µg/mL) should be started at a rate of 5 to 15 µg/ minute.

TREATMENT OF BRONCHOSPASM

Administer a β_2-agonist (albuterol, metaproterenol, pirbuterol, or terbutaline) via nebulization as required for bronchospasm that is not relieved by epinephrine.

Continuous nebulization may be required if bronchospasm persists.

ANTIHISTAMINES AND CORTICOSTEROIDS

Administer diphenhydramine 25 to 50 mg (1 mg/kg in children) intravenously over 3 minutes intramuscularly, or orally when the reaction is not severe. The addition of cimetidine 300 mg intravenously over 5 minutes may reverse refractory anaphylactic shock caused by the blockade of both H_1 and H_2 receptors. Transient hypotension, bradycardia, and arrhythmias have been reported after rapid intravenous administration of cimetidine.

Most authorities advocate the use of glucocorticoids to decrease the likelihood of a late-phase reaction. The standard therapy is either hydrocortisone 200 to 300 mg intravenously, or in milder cases, prednisone 30 to 60 mg orally.

flushing, urticaria or angioedema, hypotension or shock, wheezing, laryngospasm, abdominal cramping, and diarrhea. Arachidonic acid metabolites, such as prostaglandins, thromboxane A_2, and platelet-activating factor, also are released from mast cells and basophils, causing airway smooth muscle contraction, increased vascular permeability, goblet and mucosal gland secretion, and peripheral vasodilation. Platelet-activating factor also contracts smooth muscle and enhances vascular permeability. A late-phase reaction may occur many hours after the initial event.

Clinical Features and Diagnosis

Clinical manifestations of anaphylaxis may occur in various end organs, including the skin (urticaria, angioedema, and flushing), upper respiratory tract (rhinitis, stridor, and hoarseness), lower respiratory tract (wheezing, bronchospasm, and cough), gastrointestinal tract (abdominal pain, diarrhea, and vomiting), and cardiovascular system (tachycardia, hypotension, and shock). Urticaria and angioedema are by far the most common manifestations, occurring in 83% to 90% of individuals with anaphylaxis.[29] The second most common manifestations are respiratory tract symptoms, followed by dizziness or syncope and gastrointestinal symptoms. Cardiovascular collapse with shock can occur rapidly, without any other antecedent symptoms.

Most anaphylactic reactions occur soon (within 5 minutes to 2 hours) after exposure to an inciting agent, but other patterns are possible. In bimodal anaphylaxis, symptoms begin within minutes of exposure, and after transient clinical improvement the allergic reaction returns 1 to 8 hours later.[66] Protracted anaphylaxis can begin suddenly or gradually, but the clinical manifestations are prolonged, sometimes requiring hours or even days of intense resuscitation. Generally, the more rapid the onset, the more severe the episode.[35] Fatalities usually result from airway obstruction or cardiovascular collapse, or both.

Plasma histamine and serum tryptase can be helpful in making the diagnosis of anaphylaxis, as both may be elevated during an acute episode. The best time to measure plasma histamine is between 10 minutes and 1 hour after the onset of symptoms, whereas the best time to measure serum tryptase is between 1 and 2 hours after the onset of symptoms.[33]

TABLE 56-4. Selected Kits of Self-Injecting Epinephrine

BRAND NAME	COMPANY	DOSE (mg)	DOSES PER UNIT
EpiPen	Dey Laboratories	0.3	1
EpiPen Jr.	Dey Laboratories	0.15	1

Treatment

Anaphylaxis is a medical emergency requiring prompt recognition and treatment. Treatment depends on the severity of the reaction and the organ system (or systems) involved. For mild anaphylactic or anaphylactoid reactions limited to the skin, antihistamines alone are effective. Individuals with cutaneous reactions should be monitored closely for signs of respiratory or cardiovascular compromise, and manifestations beyond the skin require more aggressive measures, such as administration of fluids, bronchodilators, and epinephrine.

Short-term management of anaphylaxis is detailed in Box 56-3.[65] Because most fatalities occur as a result of delayed treatment,[16] the importance of prompt administration of epinephrine cannot be overemphasized. Intravenous epinephrine may be administered if the reaction is life threatening and if the patient does not respond to subcutaneous epinephrine. However, intravenous epinephrine should be used with caution in persons older than 35 years. Aerosolized aqueous epinephrine can prevent upper airway edema, but is inadequate to abort systemic anaphylaxis.[11] Use of over-the-counter epinephrine inhalation aerosol bronchodilators (e.g., Primatene Mist) is generally not recommended because of its lack of adrenergic specificity (i.e., it is non-β_2 selective) and extremely short half-life. These agents should also be used with caution in individuals with a history of coronary artery disease and arrhythmias.

Repletion of intravascular volume is a mainstay of treatment for hypotension. A large volume of fluid may be necessary and should be given rapidly in either colloid or crystalloid form. Vasopressor drugs may be needed to treat hypotension if the response to subcutaneous epinephrine and fluids is inadequate. Dopamine is often the initial drug of choice. Norepinephrine, a potent vasopressor, can be used to treat severe hypotension that is unresponsive to epinephrine, dopamine, and fluids. Glucagon may be necessary to treat refractory hypotension (e.g., patients on β-blocker medications) that is resistant to standard therapeutic regimens.[77] Glucagon is the drug of choice for patients on β-blocker medications.[18] Medical antishock trousers have been used successfully to treat refractory hypotension associated with anaphylaxis.[46]

For prominent wheezing, intermittent or continuous use (depending on severity) of an aerosolized β_2-agonist is recommended. Although antihistamines are only adjunctive therapy to epinephrine, they can relieve symptoms dramatically. Combining an H_1 antihistamine and an H_2 antihistamine may be more effective than administering either alone.[54] Most authorities recommend the use of glucocorticoids to decrease the likelihood of a late-phase reaction.

Because the symptoms can recur after the initial anaphylactic event, patients should be observed for 2 to 24 hours after being stabilized, depending on the severity of the episode. Any individual with severe respiratory or cardiac compromise should be hospitalized. Persons at risk for anaphylaxis should carry a device allowing self-injection of epinephrine (Table 56-4) at all times and should wear a medical information bracelet or carry a medical information card. Finally, it is important to identify the precipitating agent for anaphylaxis so that preventive measures can be taken to reduce the risk of future reactions.

The references for this chapter can be found on the accompanying DVD-ROM.

57 Plant-Induced Dermatitis

Bryan E. Anderson and James G. Marks, Jr.

Unintended exposure to plants may cause a wide array of skin problems. Acute care providers are often the first individuals to diagnosis and treat severe plant-induced dermatitis. Plant-induced dermatitis can present in multiple fashions to the clinician. Dermatitis can present as weeping eczematous patches and plaques, vesicles and bullae, fine scaly patches, or any combination of the above. Dermatitis is often an easy diagnosis to make. Determining the type of dermatitis with which the patient is afflicted can be difficult and frustrating.

Dermatitis can be broken down into various subtypes: contact (irritant or allergic), photocontact (irritant or allergic), nummular, xerotic, stasis, seborrheic, atopic, dyshydrotic, and essential. Dermatitis can also be seen in acute and chronic forms. The acute nature of dermatitis often leads patients to seek emergency care. The history and physical examination are of the utmost importance. A morphologic approach to the physical exam is a high-yield method for attaining the differential diagnosis. Is the rash linear in nature? This often implies an

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external contactant. Is the rash in sun-exposed areas? This implies photocontact dermatitis.

The nature of the rash is important. Irritant reactions tend to be decrescendo in severity, meaning they start out at their worst and gradually improve, whereas those that are allergic tend to be crescendo, decrescendo. They gradually build over 1 to 21 days to a peak, and then slowly improve.

There are many mimics of plant-induced dermatitis. A few examples are drug reactions, connective tissue disease, tinea infections, urticaria, and cutaneous T-cell lymphoma. If a patient has persistent dermatitis that lasts longer than a month, then a biopsy is indicated to help differentiate possible mimickers. To properly differentiate these conditions, a biopsy is performed. This is best accomplished with a 4-mm punch biopsy of skin. A pathologic review will help rule out these other conditions and lead to appropriate therapy.

The subset of contact dermatitis discussed in this chapter is plant-induced dermatitis. Plant-induced dermatitis can be caused by a wide variety of plants. Thousands of species of plants have been reported to cause dermatitis. However, the effects that each of these plants has on the skin have not been fully determined or described. There are many reasons for this. Many cases are anecdotal reports that have not been confirmed by independent observers. Few studies of the effects of plants have been preformed. The exception to that is the *Toxicodendron* species of plants.

By far the most common injury to skin caused by plants is a simple scratch, laceration, or puncture wound. Plant-induced dermatitis reactions are classified into simplified groups. These include irritant phytocontact dermatitis; this irritant group can be further subdivided into mechanically or chemically induced irritant phytocontact dermatitis, allergic phytocontact dermatitis, phytophototoxic dermatitis, and phytophotoallergic dermatitis. Plants can also cause contact urticaria and foreign-body reactions.

IRRITANT PHYTOCONTACT DERMATITIS

A wide variety of plants can cause irritant phytocontact dermatitis, which is inflammation of the skin caused by an irritating plant. Most of these rashes are mild and self-limited. These mild reactions typically involve 1% to 2% body surface area and cause transient redness and pruritus of the contacted skin. The spectrum of reactions is from linear scratch marks to weeping ulcerated red scaly plaques, the latter which may be difficult to link to the originating plant.

The most common cause of phytocontact dermatitis is irritant in nature. Irritant dermatitis can be further subdivided into traumatic (mechanical) and chemical. The most common injury resulting from plants is likely to be traumatic or mechanical in nature. Most plants have the potential to cause traumatic skin injury. Whether it is a thorn from a hawthorn tree, a cut from a sharp leaf edge, or scratches from briars, human skin is ill prepared to protect itself from such insults.

A unique example of mechanical trauma is from a glochid. Glochids are modified leaves that actually appear as tufts of barbed spines or hairs found on *Opuntia* species of cacti. They have sharp tips that act to penetrate skin and cause irritation by disrupting the epidermis. They are very loosely held to the cactus, release with the slightest touch, are quite irritating to skin, and cause variable amounts of discomfort and itching.

Figure 57-1. The characteristic red nodules seen indicating lymphangitic spread of *Sporothrix schenckii.*

Glochids are present year-round and cause dermatitis in all seasons. Appropriate footwear and clothing, including gloves, are of utmost importance when encountering plants that can cause mechanical skin trauma. Treatment is to remove the glochids from the skin. This is best done mechanically with a forceps under bright illumination and magnification if necessary.

Many infections are inoculated into the skin from mechanical plant injury. The most well known is *Sporothrix schenckii* fungal infection occurring after a prick or puncture from a rose thorn. *Sporothrix schenckii* is a common dimorphic fungus found in organic material. The characteristic lymphangitic spread is easily recognized (Fig. 57-1). Patients typically develop an ulcerating nodule at the site of inoculation, and then over the next 3 weeks on average (3 days to 12 weeks) develop nodules along the draining lymphatic channels. The nodules eventually ulcerate, and the patients develop chronic lymphangitis. There are many other infections caused by traumatic implantation of bacteria, fungus, and algae into the skin or underlying subcutaneous tissues (Box 57-1).

Another common problem is direct implantation of plant organic material into the skin causing a foreign-body reaction, such as that seen with a splinter. The goals in treatment are to remove the foreign material promptly and treat infections with the appropriate antimicrobial agent.

The most common form of irritant is chemical-induced irritant phytocontact dermatitis. Acids, enzymes, isothiocyanates, phorbol esters, calcium oxalate, and alcohols cause these reactions. The chemicals are directly toxic to the skin by altering the pH balance, dissolving the protective lipids of the stratum corneum, and denaturing skin proteins. They require direct contact of the plant material with the epidermis. Many factors can modify irritant reactions. The most important variables are duration of skin contact, concentration of the irritant, and underlying skin integrity and thickness.[7] Concentration of the irritant chemical in the plant will be at different levels in the stem, petals, roots, and leaves. Levels of the irritant also fluctuate at different times of the year, so it would not be uncommon for someone to have a reaction in the summer months from contacting the leaves of an irritating plant and not have the same reaction when touching the stem in wintertime.

Box 57-1. Infections Caused by Mechanical Plant Injury

Blastomyces dermatitidis
Staphylococcus aureus
Sporothrix schenckii
Histoplasma capsulatum
Clostridium tetani

CHROMOMYCOSIS
Fonsecaea pedrosoi
Phialophora compacta
Phialophora verrucosa
Cladosporium carrionii
Rhinocladiella aquaspersa

PHAEOHYPHOMYCOSIS
Exophiala jeanselmei
Wangiella dermatitidis

MYCETOMA
Madurella mycetomatis
Actinomadura madurae
Actinomadura pelletieri
Nocardia brasiliensis
Nocardia cavae
Nocardia asteroids
Streptomyces somaliensis
Madurella grisea
Leptosphaeria senegalensis
Petriellidium boydii
Aspergillus nidulans

PROTOTHECOSIS
Prototheca wickerhamii

Data from references 102,139,199.

Treatment of irritant phytocontact dermatitis first requires strict avoidance of the offending plant. Cleansing the wound twice daily with antibacterial soap, applying cool compresses, and watching for infection are essential. Antihistamines, such as hydroxyzine, 10 mg administered orally four times daily, up to 100 mg per day, can be used, or diphenhydramine, 25 mg orally twice to four times daily, can help with itching. The sedating antihistamines tend to work better than the newer nonsedating types (fexofenadine, loratadine, and desloratadine). Topical medium-strength steroids, such as triamcinolone 0.1% cream, may be applied twice a day to the affected areas for up to 2 weeks without risk for atrophy. Clobetasol 0.05% cream or ointment (an ultrapotent topical steroid) can also be used. Typical application regimens are twice daily for 1 to 2 weeks. Care should be taken to avoid applying repetitively to the face because the risk for atrophy is ever present with these ultrapotent steroids. Cool compresses with aluminum acetate solution (Domeboro, Burow's solution) diluted 1 : 40 in water are very helpful in soothing pruritus and skin irritation.

The spurge (Euphorbiaceae) family of plants exudes a milky sap when traumatized. This sap contains a chemical mixture of irritating diterpenes and phorbol esters.[52] After skin contact, the reactions can vary from mild stinging and burning to erythema, vesiculation, and bulla formation. Blister formation occurs typically within 24 hours. Eczematous weeping plaques can also be seen in the first 24 hours. Reactions can last for 2 to 3 weeks. Vesiculations and bullae are both forms of blisters. Vesicles refer to blisters 0.5 cm or less in diameter, and bullae are those greater than 0.5 cm in diameter. The spurge family is a large family of plants with more than 7000 described members. These plants are found predominantly in tropical climates and are found in the United States, mainly in Florida and the southwestern states. Some well-known members of the Euphorbiaceae family include the croton plant, wolfsmilk, manchineel tree, and snow-on-the-mountain. Figure 57-2 demonstrates three of these commonly irritating plants. Table 57-1 is a list of the most common Euphorbiaceae. The common seasonal houseplant poinsettia (*Euphorbia pulcherrima*) is an easily recognized plant in this family.

Plants of the genus *Croton* are also members of the spurge family. These tropical plants are the source of croton oil. This oil had been used in the past as a purgative and for many medicinal remedies. The plants contain a mixture of phorbol esters and diterpenes[19] in their leaves, stems, and seeds. These esters can cause immediate blistering of the skin, as well as a weeping eczematous eruption. These plants are found mostly in Central and South America, but about 40 species live in the southern United States. They typically appear as low-lying shrubs.

Some plants, such as those listed in Table 57-2, contain proteolytic enzymes, which cause skin irritation when contacted in sufficiently high concentrations. Plants such as *Mucuna pruriens* (cowhage) contain a proteolytic enzyme, mucunain, that causes intense itching immediately upon contact. Its seed pods are covered with tiny stinging hairs called *trichomes*. Trichomes contain high concentrations of mucunain. This enzyme has been used for decades by practical jokers in its dried form as itch powder. Trichomes are specialized cells found in most plants.

Calcium oxalate is a primary skin irritant. Calcium oxalate in plants is found in a crystalline needle-like form called *raphides*. It is present in many plants, including dumb cane, rhubarb, agave, daffodils, and hyacinths, in irritating concentrations. Dumb cane (*Dieffenbachia* species) is a common houseplant well known to cause irritant skin and mucous membrane reactions.[34] The common name arises from the effect it has on those unlucky enough to chew its leaves, which release calcium oxalate. Calcium oxalate causes irritation, swelling, salivation, pain, and blistering of the mucous membranes. In an extreme case, the afflicted person is unable to speak normally owing to involvement of the mucous membranes.[203] When the sap of this plant contacts the eye, conjunctival swelling and corneal ulcers may occur.[122] Raphides are also found in *Agave* species. Irritant contact dermatitis has been reported in tequila distillery workers, who are in frequent contact with *Agave tequilana*.[167] Purpuric and irritant reactions have been reported to occur after exposure to the sap of *Agave americana*.[31,80,160] Immediate burning on exposure is characteristic of calcium oxalate toxicity.

Daffodils and hyacinths are among the many other species containing calcium oxalate, which is found in its highest concentration in the bulb.[185] Agricultural workers who gather these bulbs and flowers are at highest risk for irritant dermatitis.[87]

The family Solanaceae contains the peppers. These plant species contain several capsaicinoids, the most common of which is capsaicin. This can cause skin and mucous membrane burning, itching, and in severe cases, vesiculation and bulla

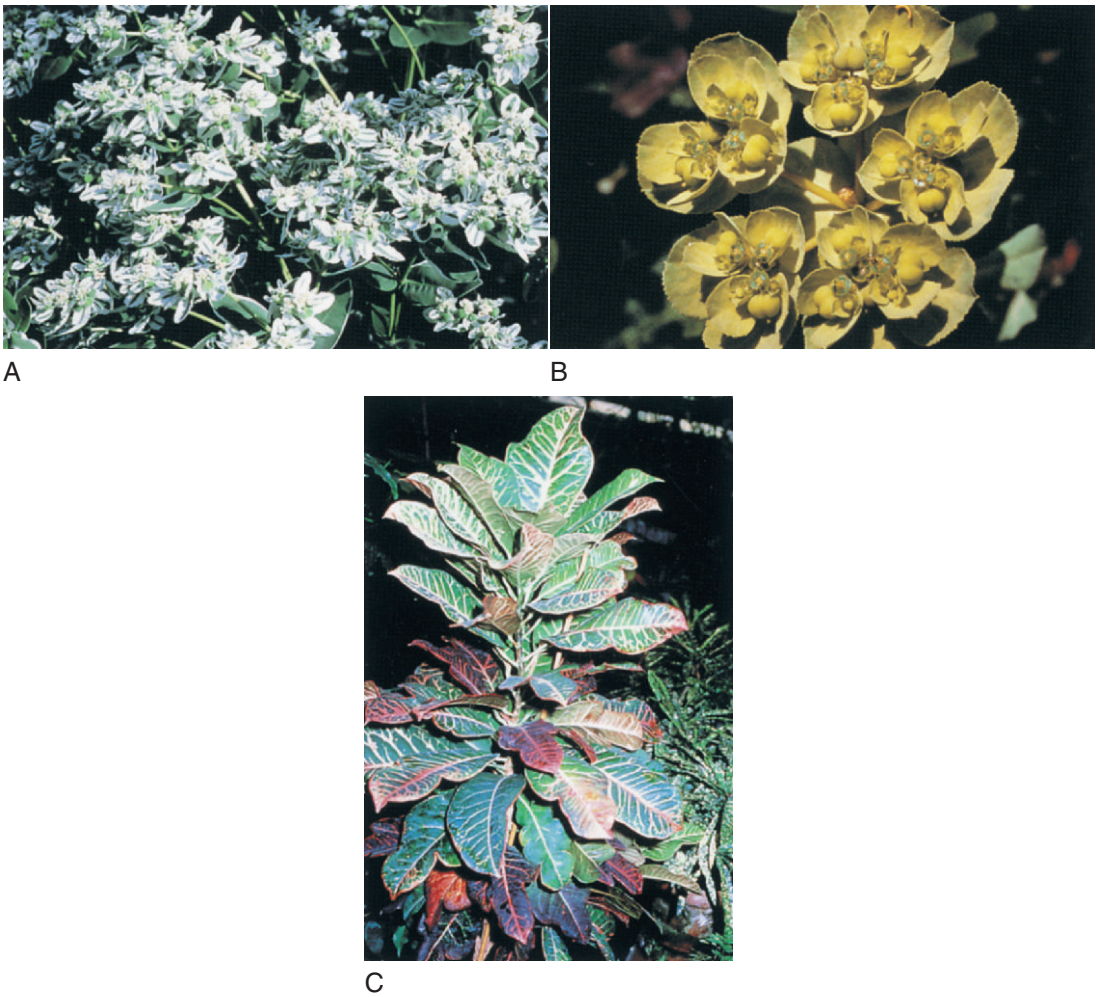

Figure 57-2. Common plants that induce irritant phytophotodermatitis. **A,** Snow-on-the-mountain. **B,** Wolfsmilk. **C,** Croton bush. (**C,** Courtesy of Yves Sell, Institute of Botany, Louis Pasteur University, Strasbourg, France.)

TABLE 57-1. Common Members of the Euphorbiaceae Family

COMMON NAME	BOTANICAL NAME
Poinsettia	*Euphorbia pulcherrima*
Candelabra cactus	*Euphorbia lactea*
Caper spurge	*Euphorbia lathyrus*
Chinese tallow	*Sapium sebiferum*
Crown-of-thorns	*Euphorbia splendens*
Cypress spurge	*Euphorbia cyparissias*
Manchineel tree	*Hippomane mancinella*
Pencil tree	*Euphorbia tirucalli*
Petty spurge	*Euphorbia peplus*
Sandbox tree	*Hura crepitans*
Snow-on-the-mountain	*Euphorbia marginata*
Sun spurge	*Euphorbia helioscopia*
Wolfsmilk	*Euphorbia purpurea*

Data from Asilian A, Faghihi G: Severe irritant contact dermatitis from cypress spurge. Contact Dermatitis 2004;51(1):37–39; and Lovell CR: Irritant plants. In: Lovell CR (ed): Plants and the Skin. Oxford: Blackwell Scientific Publications, 1993, pp 42–95.

TABLE 57-2. Common Plants with Irritant Enzymes

COMMON NAME	BOTANICAL NAME	PROTEOLYTIC ENZYME
Cowhage	*Mucuna pruriens*	Mucunain
Crownflower	*Calotropis* species	Mudarin
Pineapple	*Ananas comosus*	Bromelin
Papaya	*Carica papaya*	Papain
Fig tree	*Ficus carica*	Ficin

formation. Specialized cells of the plant's placenta produce capsaicin. Capsaicin binds to receptors on neurons and causes release of substance P from primary sensory neurons. With repeated applications, capsaicin causes depletion of substance P and desensitizes the neuron. This effect is used in clinical practice with application of capsaicin containing compounds to the skin in areas of chronic pain, such as postherpetic neuralgia.

Brassicaceae (Cruciferae) is a large family of plants that causes irritant contact dermatitis. More than 3500 species live in temperate regions of the world.[37] Its members include horse radish (*Armoracia rusticana*), black mustard (*Brassica nigra*), and white mustard (*Sinapis alba*) (Fig. 57-3). These plants contain glucosinolates, which are turned into isothiocyanate, which causes irritant dermatitis.[114] This reaction is catalyzed by the enzyme myrosinase. Substrate interacts with the enzyme after the plant is crushed, as in chewing.[37] Contact with these plants can cause a wide range of cutaneous reactions, including burning sensation, pain, red patches, and blister formation.

The Ranunculaceae family includes buttercups (*Ranunculus* species) (Fig. 57-4). Table 57-3 lists a small sampling of plants in this family reported to cause irritant contact dermatitis. Protoanemonin is considered the primary irritant toxin in this group of plants. Reactions from these plants tend to be mild and rarely cause people to seek medical care. Table 57-4 includes plants and plant families that cause irritant phytocontact dermatitis and lists their primary irritant substance.[105,185]

Figure 57-3. White mustard plant. (Courtesy of Yves Sell, Institute of Botany, Louis Pasteur University, Strasbourg, France.)

The pathogenesis of primary irritation from plants has not been fully elucidated. In the case of nonimmunologic urticaria, release of histamine from mast cells with vasodilation and leakage of fluid appears to be an early direct chemical change leading to hives.[44,175] The molecular events in mast cell degranulation are thought to be similar to those described for immunogenic urticaria, with certain discrete differences. Thus, there is no evidence for a selective membrane receptor, such as the high-affinity immunoglobulin E (IgE) receptor that is now well characterized and required for immunologic degranulation of mast cells.[175,183] Instead, current information suggests that a receptor-independent mode of action occurs for all nonimmunologic histamine liberators, acting directly on a pertussis toxin-sensitive G protein to initiate a signal through phospholipase C activation, which degranulates mast cells of their histamine content.[133] Furthermore, there does not seem to be a requirement for methylation of the membrane phospholipids. Rather, a high intracellular calcium accumulation must occur, and the reaction is rapidly terminated without delayed mediator release.[164] Arachidonic acid metabolites are not formed in any amount.[14]

It is currently believed that both irritation and contact sensitization are mediated by epidermally derived cytokines. Tumor necrosis factor-α (TNF-α), interferon-γ (IFN-γ), macrophage inflammatory protein-2 (MIP-2), and granulocyte-macrophage colony-stimulating factor (GM-CSF) are produced and secreted into tissue in response to both allergens and irritants. However, a number of other proteins are released only by allergenic stimulation.

In irritant contact dermatitis, the chemical irritant, which is usually an acid, alkali, surfactant, solvent, oxidant, enzyme or toxin, damages the keratinocyte. This damage is highly dependent on the concentration of the irritant. There is no sensitization or elicitation phase, as is seen in allergic contact dermatitis. The damaged keratinocyte activates phospholipase A_2. This in turns cleaves arachidonic acid and diacylglyceride (DAG) from the cell membrane. The arachidonic acid is converted into various prostaglandins and leukotrienes. Prostaglandins and leukotrienes cause endothelial cells to dilate and become leaky, resulting in edema. They also act on mast cells to release hista-

A B

Figure 57-4. Plants of the family Ranunculaceae. **A,** Buttercup. **B,** Old man's beard. (**B,** Courtesy of Yves Sell, Institute of Botany, Louis Pasteur University, Strasbourg, France.)

TABLE 57-3. Irritant Plants in the Family Ranunculaceae

COMMON NAME	BOTANICAL NAME
American prairie crocus	*Pulsatilla patens*
Buttercup	*Ranunculus* species
Christmas rose	*Helleborus niger*
Meadow rue	*Thalictrum foliosum*
Pasque flower	*Pulsatilla vulgaris*
Pilewort	*Ranunculus ficaria*
Staves-acre	*Delphinium* species
Traveler's joy	*Clematis vitalba*
Windflower	*Anemone nemorosa*
Wolfsbane	*Aconitum napellus*

TABLE 57-4. Plants Causing Irritant Phytocontact Dermatitis and Their Primary Irritant Chemicals

COMMON NAME	BOTANICAL NAME	IRRITANT CHEMICAL
Agave	*Agave americana*	Calcium oxalate
Black mustard	*Brassica nigra*	Isothiocyanates
Buttercup	*Ranunculus bulbosus*	Protoanemonin
Coral plant	*Jatropha*	Thioglycoside
Cowhage	*Mucuna pruriens*	Proteolytic enzymes
Croton	*Croton tiglium*	Phorbol esters
Daffodils	Amaryllidaceae family	Calcium oxalate
Dumb cane	*Dieffenbachia* species	Calcium oxalate
Hyacinths	Liliaceae family	Calcium oxalate
Manchineel tree	*Hippomane mancinella*	Phorbol esters
May apple	*Podophyllium peltatum*	Podophyllin resin
Pencil tree	*Euphorbia tirucalli*	Triterpene alcohols
Prickly pear	*Opuntia* species	Spines
Spurges	Euphorbiaceae family	Shorbol and diterpene esters
Mustard, radish, etc.	Family Cruciferae (Brassicaceae)	Isothiocyanates
Buttercups, pilewort	Family Ranunculaceae	Protoanemonin
Common caper	Family Capparidaceae e.g., *Capparis spinosa*	Isothiocyanates
Peppers	Family Solanaceae	Capsaicin
Spider plant	Family Cleomaceae e.g., *Cleome* species	Isiothiocyanates

Data from High WA: Agave contact dermatitis. Am J Contact Dermat 2003; 14(4):213–214; and Ricks MR, Vogel PS, Elston DM, Hivnor C: Purpuric agave dermatitis. J Am Acad Dermatol 1999;40:356–358.

mine and are chemoattractants for lymphocytes and neutrophils. Further recruitment of lymphocytes and neutrophils is facilitated by expression of intercellular adhesion molecule-1 (ICAM-1) by keratinocytes.[116] The DAG causes upregulation of genes for cytokines such as interleukin-1 (IL-1) and GM-CSF. These proteins then act to stimulate T cells and neutrophils. All these inflammatory cells and release of various cytokines and vasoactive substances lead to the clinical findings in irritant contact dermatitis. This is a continuing area of investigation.[162,205]

Treatment

Treatment of primary irritant dermatoses is often unsatisfactory. The victim must be removed from exposure to irritant chemicals and treated conservatively with rest, medicated soaks, and compresses, such as aluminum acetate solution (1 : 40), dilute potassium permanganate solution (1 : 16,000 in water), acetic acid solution (1 : 100), or Dalibour solution (copper and zinc sulfate and camphor, supplied commercially as Dalidane). Antihistamines usually have a small effect, but in some, they may be quite helpful in alleviating pruritus. Dermatitis generally heals in less than 7 days if no complications develop and if tissue damage is minimal. Corticosteroids are of minimal use in controlling primary irritation. If the patient is unable to be removed from the source of irritation, no medicine, cream, or soak will alleviate the problem. People are often forced to change occupations or hobbies, or at the very least modify their environment.

ALLERGIC PHYTOCONTACT DERMATITIS

Allergic contact dermatitis is a type IV delayed hypersensitivity reaction. This form of contact allergy is far more common than is contact urticaria. The most common presentation is linearly arranged eczematous, edematous patches and plaques with varying amounts of vesiculation and bulla eruption. Occasionally, the eruption is widespread. If the face is involved, there can be severe eyelid swelling. Patients are quite distressed by their appearance. They can have systemic symptoms of fever, chills, fatigue, and lethargy. The most common cause of allergic contact dermatitis in the United States is from exposure to poison ivy, oak, and sumac plants (Table 57-5). In the past, allergic contact dermatitis caused by poison ivy, oak, or sumac was referred to as *Rhus* dermatitis. Recent botanical nomenclature places the poison ivy, oak, and sumac plants in the Anacardiaceae family in the genus *Toxicodendron*. The *Rhus* genus contains plants that are not known to cause allergic contact dermatitis. Therefore the term *Rhus* dermatitis should be abandoned.

The weeds are surprisingly fastidious. They do not grow in Alaska or Hawaii and do not survive well above 1500 m (5000 feet), in deserts, or in rainforests. They grow best along cool streams and lakes and luxuriate if it is also sunny and hot. They are found in every state of the continental United States. The plants have different configurations in different regions, but generally, poison ivy grows east of the Rockies, poison oak grows west of the Rockies, and poison sumac grows best in the southeastern United States. Because avoidance is the best prevention, it is important to learn what the plants look like in a given area (Fig. 57-5). Once contaminated with the oil (resin),

TABLE 57-5. *Toxicodendron* Species

COMMON NAME	BOTANICAL NAME
Western poison oak	*Toxicodendron diversilobum*
Eastern poison oak	*Toxicodendron quercifolium*
Poison ivy	*Toxicodendron radicans*
Rydberg's poison ivy	*Toxicodendron rydbergii*
Poison sumac	*Toxicodendron vernix*

A

D

B

E

C

Figure 57-5. Plants in the *Toxicodendron* genus. **A,** Poison ivy. **B,** Poison ivy growing as a sea of vines. **C,** Poison oak. **D,** Poison oak, close up. **E,** Poison sumac. (**C** Courtesy Paul Auerbach.)

an average person has 1 to 4 hours to wash it off to prevent dermatitis.

Poison ivy (*T. radicans*) grows in moist shady regions east of the Mississippi River. The plants thrive at sea level and do very poorly above 5000 feet (1500 meters). Poison ivy is never found on the west coast of the United States. It has leaflets that are 10 to 30 cm long and found in ternate (three separate leaves). The shape of the leaves is often ovate or obtuse. The leaves can be shiny, smooth, and hairless, or they can be rough, hairy, and velvety[12,37] (Fig. 57-6). It is a climbing shrub commonly found growing up the trunks of large trees, with aerial roots that are quite prominent.

Poison oak (*T. diversilobum*) is found on the west coast of North America and is given the designation *western poison oak*.

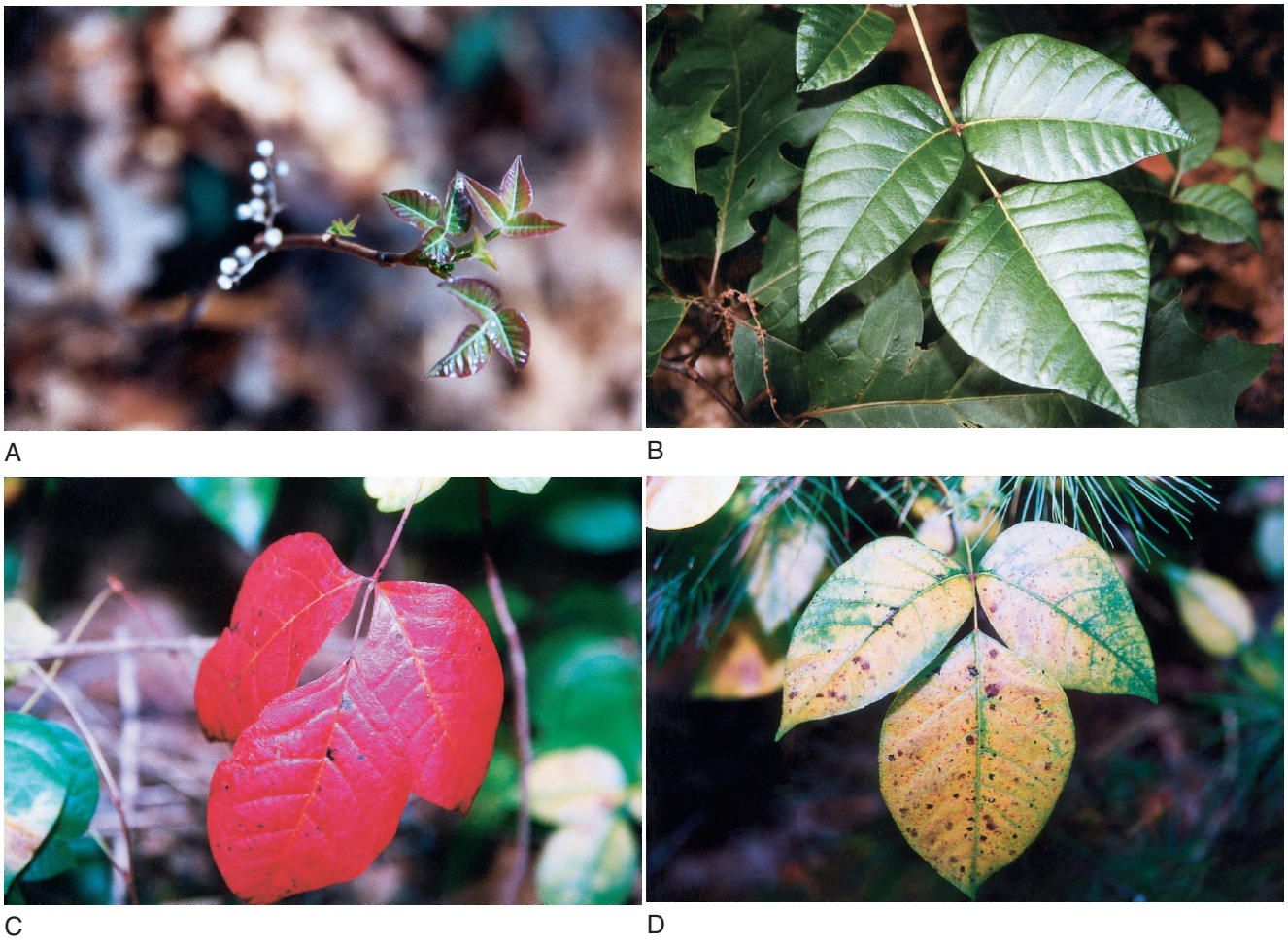

Figure 57-6. **A–D,** Poison ivy can have various appearances.

It is a common shrub that has multiple stems that form three leaflets. If it grows in shade, the leaves are larger than if it is grown in full or partial sunny conditions. It also has many brown aerial roots and has clusters of yellow flowers that bloom in the spring.[37] The flowers bear cream-colored berries. In the California hills, poison oak grows like a forest, but in cooler, dry climates, it remains isolated in small patches.

Poison sumac (*T. vernix*) is a fast-growing small tree or shrub. Some plants grow to 40 feet (12 m), but the average height is about 15 feet (5 m). The plants are found in wet marshy regions of the United States. Its leaves are unique among *Toxicodendron* species. They are configured as 7 to 13 smooth oval leaflets attached along a central stem.[37] The plant forms pale-colored fruit. Nonpoisonous sumacs can be recognized by their jagged leaf margins and red berries.

All *Toxicodendron* plants have many interconnected channels that contain the sap. When the plant is traumatized and a channel is broken open, the sap is extruded and hardens as a black resin to seal off the damage. This sap is the material that contains the urushiol.[37]

TABLE 57-6. Anacardiaceae Family

COMMON NAME	BOTANICAL NAME
Brazilian pepper	*Schinus terebinthifolius*
Cashew	*Anacardium occidentale*
El litre tree	*Lithraea caustica*
Ginkgo tree	*Ginkgo biloba*
Indian marking nut	*Semecarpus anacardium*
Japanese lacquer	*Rhus vernicifera*
Korean lacquer tree	*Rhus vernicifera stokes*
Mango	*Mangifera indica*
Pepeo tree	*Mauria puberula*
Poison ivy	*Toxicodendron radicans* and *T. rydbergii*
Poison oak	*Toxicodendron diversilobum* and *T. toxicarium*
Poison sumac	*Toxicodenderon vernix*
Poisonwood	*Metopium toxiferum*
Renges tree	*Anacardium melanorrhoea*

Data from Marks JG Jr, Elsner P, DeLeo VA: Contact and Occupational Dermatology, 3rd ed. St Louis, Mosby, 2002, pp 13–15.

TABLE 57-7. Plants Causing Allergic Contact Dermatitis

COMMON NAME	BOTANICAL NAME
(Various)	*Alstromeria* species
(Various)	*Compositae* species (about 20,000 species)
(Various)	*Grevilla* species
Lichen	*Cladonia*, *Evernia*, and *Primelia* species
Liverwort	*Frullania* species
Pine tree	*Pinus* species
Poison ivy	*Toxicodendron radicans*
Poison oak	*Toxicodendron diversilobum*
Poison sumac	*Toxicodenderon vernix*
Primrose	*Primula obconica*
Ragweed	*Ambrosia* species
Tulip	*Tulipia* species

Data from Marks JG Jr, Elsner P, DeLeo VA: Contact and Occupational Dermatology, 3rd ed. St Louis, Mosby, 2002, pp 13–15.

Figure 57-7. A 27-year-old man had had a pruritic and eczematous rash for 3 days. One week earlier, he had peeled a mango, become distracted by a telephone call, and rested his left hand on his right leg. Three days later, contact dermatitis became apparent. When much younger, the patient had been sensitized to poison oak and poison ivy; the sap of the mango rind contains oleoresins that cross-react with the oleoresins of poison ivy. The rash resolved after 1 week of treatment with topical corticosteroids. (From Tucker MO, Swan CR: Images in clinical medicine. The mango-poison ivy connection. N Engl J Med 339[4]:235, 1998.)

It is estimated that 50% to 70% of the population is sensitive to the causative antigen, urushiol. The Anacardiaceae family accounts for most cases of allergic contact dermatitis in the United States. Table 57-6 catalogs the major members of this family and the plants most likely to cause dermatitis. Most individuals who spend a great deal of time outdoors know how to recognize these plants and appropriately avoid them. Figure 57-5 shows poison ivy, oak, and sumac. However, these are not the only plants that need to be considered. Table 57-7 lists some common plants causing allergic contact dermatitis.

Mango dermatitis (Fig. 57-7) has been reported to be the leading cause of plant dermatitis in Hawaii.[185] Urushiol is found in the plant's leaves and stems and in its fruit's skin. Typically, people are exposed to the urushiol when they eat or manipulate the fruit that has yet to be peeled. Facial involvement, particularly of the lips, occurs after biting into a mango that still carries its outer peel. The other common area of involvement is the hand after individuals peel the fruit.[114] Once peeled, mangos are no longer allergenic.

TABLE 57-8. Imposters of the Poison Ivy Plant

COMMON NAME	BOTANICAL NAME
Boston ivy	*Parthenocissus tricuspidata*
English ivy	*Hedra helix*
Skunkbush sumac	*Rhus trilobata*
Virginia creeper	*Parthenocissus quinquefolia*

Data from McGovern TW, LaWarre SR, Brunette C: Is it or isn't it? Poison ivy look-a-likes. Am J Contact Dermat 2000;11(2):104–110.

Figure 57-8. Poison ivy on the left, with three leaves, adjacent to the commonly mistaken Virginia creeper with five leaves (*bottom and right*).

Multiple plants are similar in appearance to and need to be differentiated from the poison ivy plant. Table 57-8 lists some of the more common imposters for the poison ivy plant. The Virginia creeper, *Parthenocissus quinquefolia,* is commonly misidentified as poison ivy. Figure 57-8 shows poison ivy side by side with the Virginia creeper. English ivy, *Hedra helix,* is another plant that is often difficult to differentiate from poison ivy. *Hedra helix* has been reported to cause allergic contact dermatitis.[206]

The ginkgo tree, *Ginkgo biloba,* is commonly found in cities, where the trees thrive. Its characteristic split leaves are easily recognized (Fig. 57-9). It has been reported to cause allergic contact dermatitis in joggers who run through debris on sidewalks and roadways containing ginkgo leaves, flowers, and fruit.

The antigen in the Anacardiaceae family is urushiol resin (Fig. 57-10). All parts of the plant contain resin, which can be

A

B

Figure 57-9. **A** and **B,** *Ginkgo biloba.*

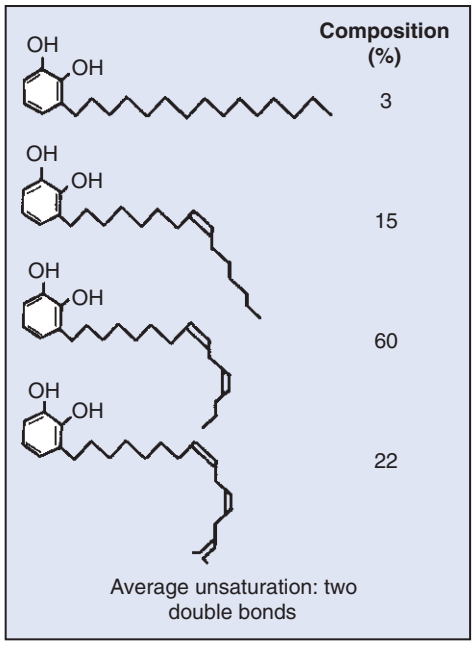

Figure 57-10. General structure and composition of poison ivy urushiol.

a heavy, nonvolatile oil. In its natural state, the oil is colorless or slightly yellow. Because the oil is virtually invisible, many people fail to understand how they acquired the rash. It is not a vapor, because at 315° C (600° F) in a fire or oven, urushiol splatters like butter. In a camp or forest fire, it attaches to smoke particles and can be carried downwind. The oil also readily coats the fur of animals, which explains why people often get the dermatitis from their outdoor pets. On exposure to air, the oil oxidizes, polymerizes, and turns black. This is a way to recognize the weeds, especially in autumn when the leaves fall off.

The amount of urushiol present in poison ivy and poison oak is roughly equal year-round, even when the plants are only sticks without leaves in the winter. As the leaves turn red and start to dry up in the fall, important nutrients, including urushiol, return to the stem and roots through subepidermal resin canals.[67] Thus, dead leaves that fall to the ground are virtually devoid of urushiol.

Urushiol is exposed to skin when a plant that has been injured or bruised releases it to the surface of the leaf, petal, stem, or root. Clinical effects are usually manifested 24 to 48 hours after contact in a previously sensitized individual, and occur 10 to 14 days after contact in a patient on first exposure. Typically, linear streaking of papulovesicles occurs; edema, weeping, and crusting are also commonly seen (Fig. 57-11). A distinct linear nature is often a clue to the diagnosis of allergic contact dermatitis. On occasion, the eruption can appear to be urticarial in nature or can mimic cellulitis. Appearance of black dots on the skin in areas of involvement can be helpful in determining the etiology.[100] The black dots represent dried urushiol that has been oxidized by air (Fig. 57-12). Severe itching leading to excoriations nearly always accompanies the rash. This can lead to secondary infection.

The antigen is found in the milky sap, which is quickly absorbed into the skin. Thus, it is nearly impossible to wash off the sap quickly enough to prevent the dermatitis. There are many unique urushiol chemicals. Collectively, they can be called *urushioids*. Each plant has a different urushioid concentration and composition. Urushioids have a structure that contains a benzene ring with a varying-length carbon side chain. The concentrations of the various urushioids of each plant depend on growing conditions and season, which also affect their antigenic properties. If the side chain is desaturated and longer, this increases the catechols' antigenicity. Conversely, if there is a

Figure 57-11. Representation of the linear nature of poison ivy–induced allergic phytocontact dermatitis. **A,** Knee. **B,** Hand. **C,** Finger.

substitution on the catechol ring, this reduces antigenicity.[116] The addition of an aliphatic side chain and presence of free phenolic groups also increase antigenicity.[185] Poison ivy contains predominantly urushiol III, poison oak contains mostly urushiol I, and poison sumac contains predominantly urushiol II.[37,116]

Urushiol resin can remain stable on inanimate objects, such as animal fur, for up to a year. It is degraded by ultraviolet light and biodegraded by bacteria and fungi. Exposed patients may not recall ever being in direct contact with plants. Severe cases of exposure from airborne aerosolized urushiol leading to dermatitis have been reported after exposure to smoke from burning plants.

If the patient avoids re-exposure, dermatitis resolves within 14 to 21 days in most cases. It is important to diagnose sec-

Figure 57-12. Poison ivy–induced allergic contact dermatitis after a young woman accidentally pinched some poison ivy leaves behind her knee. Note the central black dots, which represent the dried urushiol.

Figure 57-13. Generalized poison ivy dermatitis.

ondary impetiginization because this can lead to cellulitis if not treated with appropriate antibiotics. The most likely pathogens are *Staphylococcus aureus* and streptococcal species. Hyposensitization therapy has not proved practical or particularly effective and is rarely performed. Patients need to be informed to thoroughly clean clothing, through at least one complete automated wash and rinse cycle with detergent, and to clean any object that may have come in contact with the plant in order to avoid repeat exposure.

Delayed hypersensitivity or type IV allergy of the contact hypersensitivity variety occurs more frequently as a result of contact with these plants than does type I contact urticaria. Sensitivity to poison ivy or poison oak is undoubtedly the single most common cause of allergic skin reactions in the United States and, along with contact allergy to sesquiterpene lactones ("Compositae"), probably worldwide (see Figure 57-5). When a distraught young person arrives in the emergency department with an acute, edematous, and erythematous skin eruption and a history of outdoor activity, this is probably a serious bout of poison ivy or poison oak dermatitis.

The amount of purified urushiol required to elicit a reaction is 2 to 2.5 mg.[58] Some people (about 35%) are considered subclinically sensitive because they have negative skin test reactions to 2.5 mg urushiol, but react to higher concentrations, such as 5, 10, and 50 mg.[49,58] Clinically, this group is interesting because they invariably did not have poison ivy dermatitis as teenagers and often plucked the weeds with apparent impunity. However, usually in midlife after a bout of weed pulling, a rash spreads explosively. For unknown reasons, they have crossed the line into clinical sensitivity. If patch-tested with dilutions of urushiol, they are often exquisitely sensitive and do not appear to lose their reactivity. The flare-up may last for several weeks, probably because of prior contamination of the home and workplace with urushiol oil. Treatment must be aggressive and more prolonged than usual.

A smaller group (10% to 15%) does not react to higher concentrations and cannot be sensitized by 1000 mg. This group was first detected and studied in passive transfer experiments in the 1950s.[60] These individuals are considered to be naturally tolerant, but it remains unclear whether they achieved that state by early antigenic exposure or by genetic luck. They have no inherent resistance to contact sensitization with other chemicals[61] and otherwise appear healthy. They may hold a clue to the molecular basis for immunologic tolerance.

From a practical standpoint, only 10% to 15% of Americans (up to 40 million people) can be categorized as exquisitely sensitive. Generally, these persons seek and need emergency medical care (Fig. 57-13). They typically have had prior unpleasant experiences. Within 2 to 6 hours after exposure, swelling is accompanied by an erythematous, intensely pruritic, edematous, vesicular, and ultimately bullous eruption that can be associated with fever, malaise, and prostration (Fig. 57-14). This true dermatologic emergency should be treated immediately and vigorously.

Extreme susceptibility tends to be familial; if one parent is supersensitive, children are likely to be as well. If both parents are sensitive, the chance of sensitive offspring is about 80%.[196] The level of individual sensitivity is not determined by the severity of the initial bout of dermatitis, although nearly half of patients admit to a memorable bout of dermatitis as a teenager. However, no more than 25% to 35% of victims at age 30 to 40 were stricken within the previous year.[49] When these subjects are patch-tested with weak dilutions of urushiol, less than half react as might be expected from their history. An individual's level of reactivity does not change appreciably if he or she is tested monthly over a year[58]; testing at less frequent intervals in very sensitive subjects over 3 to 4 years has shown little or no change in the level of reactivity. Repeated mild to moderate bouts of dermatitis maintain the sensitive state, whereas a single severe bout may produce a prolonged period of anergy or refractoriness,[48,49] not unlike the clinical condition of "hardening" or unresponsiveness, which is well described in the industrial setting.[174]

A B C

Figure 57-14. Severe acute poison oak dermatitis. **A,** Facial edema. **B,** Blisters. **C,** Penile edema. (Courtesy of Axel Hoke.)

The urushiols in poison ivy, poison oak, and poison sumac differ only slightly in structure and biologically cross-react, so that a person sensitive to poison ivy may also react to poison oak and poison sumac (see Figure 57-10). Plants, shrubs, and trees with cross-reacting catechols are found throughout the world, arising either indigenously or by transplantation.[53,156] The most common cross-reacting plants are mango, cashew nut shell, India marking nut, and Japanese lacquer. Increasing numbers of cross-reacting plants are reported from South America.[85] A chemical curiosity is the cross-reaction with resorcinols from cashew nut shell oil.[93] Similar cross-reacting resorcinol-containing plants have been observed in Australia[127] and Hawaii.[98] Recently, it has been confirmed that philodendron plants contain a contact-sensitizing resorcinol, but it only reacts in poison oak– or poison ivy–sensitized patients who have traveled to the mainland.[99] Other studies have extended our information about the urushiol-like molecules that exist in many shrubs and small trees in South America, specifically the litre tree (*Lithraea caustica*).[3,113]

Immunology of Poison Ivy and Poison Oak Dermatitis

The immunologic mechanism for allergic contact dermatitis is generally conceded to be type IV cell-mediated delayed hypersensitivity.[51] There is an initial sensitization phase, followed by an elicitation phase on subsequent exposure.

Allergic contact dermatitis is caused by haptens (small chemical compounds). These haptens are nearly always 500 daltons or less,[107] and lipid soluble. Urushiol from poison ivy would be considered one such hapten. Haptens are unprocessed antigens. Langerhans cells are the main antigen-presenting cells (APCs) in the skin. They are normally in a resting state, with little to no ability to stimulate T cells. In skin, keratinocytes are also part of the immune surveillance system. They respond quickly to every chemical insult, either irritant or allergenic, in antigen-dependent fashion, to produce a variety of cytokines. These function mainly to amplify future inflammatory responses.[10,140] Early on, they secrete TNF-α and ICAM.[2] Later, they release IL-1, IL-6, IL-8, and IL-10, and still later, macrophage chemotactic factor and other cytokines.[9,70,71,140,155] These may in turn release acute-phase reactants from mast cells and endothelial cells, as seen in irritant responses.[10,140] Current research is attempting to identify which keratinocyte cytokines specify the allergic reaction, as distinct from simply injury. Nevertheless, it is known that the catechol molecules of poison oak and poison ivy enter the skin and bind through nucleophilic attack at benzene ring positions 4, 5, or 6 to surface proteins on APCs, which are primarily epidermal Langerhans cells. These cytokines from the keratinocytes cause Langerhans cells to mature and enter into an active state. Active Langerhans cells are then able to recognize and internalize antigens.

Langerhans cells are responsible for immunosurveillance. In the sensitization phase, when a hapten such as urushiol is applied to skin, it penetrates the stratum corneum and is identified by activated APCs. These cells internalize the urushiol by pinocytosis. Proteolytic enzymes in lysosomes then process the urushiol. This processing consists of proteolytic degradation of the protein into smaller antigenic peptides. The antigenic peptides that have undergone chemical degradation associate with class II major histocompatibility complex (MHC) molecules and are expressed on the surface of the APCs.

The APCs then leave the epidermis and travel to regional lymph nodes, where they present processed antigen on the cell surface in context with a class I MHC molecule to the T-cell receptor complex on a CD8+ T cell, or in the context with the class II MHC molecule to the T-cell receptor complex on a CD4+ T cell. Presence or absence of a specific T-cell receptor is likely to be genetically determined. During early fetal development, the thymus interacts with various T cells. Through gene rearrangements, thousands of unique T-cell receptors are produced that recognize unique antigens. If no T cell is present that recognizes the antigen complex on the APC, no reaction occurs, and the patient does not develop allergic contact dermatitis. To complete the sensitization signal, another surface protein (the B7 antigen) forms a costimulatory signal by binding to the CD28 ligand on the T cell. This then activates the T cell, which divides repeatedly to form a clone of urushiol-specific CD8+ and CD4+ cells. These subsequently expand into clones of circulating activated T-effector and T-memory lymphocytes.[91,113]

In the elicitation phase, upon a new challenge by urushiol, the hapten enters the epidermis and again is internalized by Langerhans cells. It is processed and expressed by the MHC

molecules, which then interact with the clone of T cells now specifically ready to interact with this MHC–antigen complex. This occurs in both skin and lymph nodes. Langerhans cells in the skin that interact with T cells secrete IL-1, which stimulates the T cells to secrete IL-2 and express IL-2 receptors, which directly leads to activation, proliferation, and expansion of the T-cell clone. Activated T cells will also secrete INF-γ, which acts on keratinocytes to express ICAM-1, which allows them to directly interact with T cells.[116] Keratinocytes also secrete many cytokines that cause expansion and proliferation of these T cells. The CD8 lymphocytes elicit a cell-mediated cytotoxic immune response characterized by erythema, edema, and vesiculation resulting from destruction of epidermal cells and activation of the dermal vasculature. Under this scenario, the clinical reaction is driven by the CD8 cytotoxic, effector T cells. This can be modified by CD4 T cells that may be either T_H1 or, more likely, T_H2 in nature.[91,113] In an individual patient, this determines the severity of dermatitis after exposure to the poisonous weeds. This logical formulation, however, does not completely explain acute poison oak or poison ivy dermatitis. An acute eruption tends to begin within hours of exposure and is primarily edematous. Some have emphasized the presence of basophils in acute poison oak and poison ivy lesions and have proposed a role for basophil mediators in the pathogenesis of an early-onset acute reaction.[45] Certainly, the clinical presentation in these cases favors a late-phase reaction[104] and a mechanism possibly involving mast cell degranulation,[95] as well as other cytokine mediators. Also possible is a CD4 T-cell–mediated induction of IgE reactivity.[90]

Suppressor pathways to decrease the extent of the above pathways also exist. These mechanisms are poorly understood, but at some time during the sensitization phase, specific suppressor T cells are generated to counterbalance or downregulate the type IV hypersensitivity reaction. It is the exposure to antigen through noncutaneous routes that leads to these suppressor pathways or, if there is a massive exposure, to antigen at the sensitization phase.[116] The final resulting skin reaction is a balance of sensitization and suppression of these antigen-specific reactions.

Treatment

Systemic corticosteroids are widely accepted as the first line of treatment, especially given early and in large, therapeutic doses. If the reaction is of less than 2 hours' duration, intravenous (IV) hydrocortisone (adult dose 100 to 200 mg) or methylprednisolone (adult dose 500 mg to 1 gm) can be curative. After a patient has suffered 4 to 6 hours with massive edema, erythema, and pruritus, IV therapy is highly effective, but it must be followed by more prolonged oral or intramuscular (IM) administration of corticosteroids. Most patients in this category seek help after 8 to 16 hours of discomfort, at which point IV therapy is less effective. Prednisone, 100 to 120 mg divided in four equal doses daily for 2 days, may achieve a salubrious effect. At the same time, patients are instructed to rest at home for 3 to 4 days. If a flare-up begins, a repeat regimen of prednisone is prescribed. Subsequent exacerbations are usually caused by re-exposure to urushiol that has contaminated the patient's local environment, so that the patient must be warned to thoroughly wash all clothing and other articles that might have been exposed during the initial contact.

If the dermatitis has been present for more than 24 hours, the aggressive regimen is less successful (see Figure 57-14). Under these circumstances, many physicians prefer a more conservative approach. Oral prednisone, 1 mg/kg per day for 3 to 4 days with a 10- to 14- every-other-day taper, helps many patients, but the danger lies in a sudden flare-up, which becomes poorly responsive to steroids, at the end of therapy. Whenever considering systemic corticosteroids for acute allergic contact dermatitis, the physician must be certain that the patient is otherwise healthy and without active infection, vascular accident, endocrinopathy, or familial history of glaucoma. Side effects from oral corticosteroids include hypertension, increased risk for infection, adrenal insufficiency, glaucoma, cataracts, increased blood sugar, mood changes, osteoporosis, osteonecrosis, edema, weight gain, hypokalemia, peptic ulcer disease, bowel perforation, myopathy, Cushing's syndrome, adrenal suppression, and stunted growth in adolescents.

When the onset of dermatitis is delayed for several days and the eruption is mild or moderate, systemic therapy offers little benefit. In the situation of a mildly to moderately sensitive patient with delayed onset, topical corticosteroids are the mainstay of therapy. Typically, an ultrapotent topical steroid, such as clobetasol (Temovate), is used for no more than 14 days. Table 57-9 lists the most common topical corticosteroids and their classification. Prolonged use of ultrapotent corticosteroids may lead to skin atrophy. The face, axilla, and groin are highly susceptible to atrophy, so use of ultrapotent products should be avoided in these regions. In most mild cases of dermatitis, a medium- to high-potency topical steroid, and 15- to 30-minute cool compresses three or four times a day with a 1 : 40 dilution of aluminum acetate (Domeboro, Burow's solution), will suffice. A bath with 1 cup of Aveeno oatmeal per tub of water is helpful for the itching. Treatment usually is required for 1 to 2 weeks.

In more severe cases with the eyelids, hands, or more than 10% body surface area involvement, a systemic corticosteroid, such as prednisone in a tapering dose starting at 1 mg/kg or IM triamcinolone (Kenalog) 40 mg IM, is employed. Antihistamines, such as hydroxyzine, 10 mg given orally four times daily up to 100 mg per day, or diphenhydramine, 25 mg orally two or three times per day, can be used to suppress itching. Calamine lotion and aluminum acetate compresses also help with the itching.

Advent of the new topical immunomodulators pimecrolimus (Elidel) 1% cream and tacrolimus (Protopic) 0.03% or 0.1% ointment offers a noncorticosteroid treatment. There is no risk for atrophy with these two agents. Preliminary studies show them to be helpful in allergic and irritant contact dermatitides.

Most patients visit a pharmacy looking for over-the-counter (OTC) preparations. Usually, they are disappointed. The eruption heals spontaneously in 7 to 10 days, so the most helpful option is an inexpensive agent, such as calamine lotion, which is comforting and helps form a crust. Aluminum acetate (Domeboro, Burow's solution) 1 : 40 diluted in plain water used as a soak, compress, or wet dressing is helpful, as is a 1% acetic acid wet dressing (white vinegar) in water. The soaks are performed for 15 to 30 minutes at a time and can be repeated as often as necessary. Regardless of the therapy chosen, time is required for healing.

Systemic antihistamines may help with pruritus. Aspirin and nonsteroidal anti-inflammatory drugs are without effect. Topical application of soothing aluminum acetate (1 : 40) soaks, calamine lotion, or tepid baths, with 1 cup Aveeno oatmeal or 2 cups linnet starch per tub, relieves pruritus and

TABLE 57-9. Steroid Classification

GENERIC NAME	TRADE NAME	PREPARATIONS AVAILABLE
Ultrapotent		
Betamethasone dipropionate	Diprolene	0.05% cream or ointment
Clobetasol propionate	Temovate, Cormax	0.05% cream or ointment
Diflorasone diacetate	Psorcon	0.05% cream or ointment
Halobetasol propionate	Ultravate	0.05% cream or ointment
High Potency		
Amcinonide	Cyclocort	0.1% cream or ointment
Desoximetasone	Topicort	0.25% cream or ointment
Fluocinonide	Lidex	0.05% cream or ointment
Halcinonide	Halog	0.1% cream or ointment
Medium Potency		
Betamethasone valerate	Luxiq	0.1% cream or ointment, 0.12% foam
Fluocinolone acetonide	Synalar	0.025% cream or ointment
Fluticasone propionate	Cutivate	0.005% ointment or 0.05% cream
Hydrocortisone butyrate	Locoid	0.1% cream
Hydrocortisone valerate	Westcort	0.2% cream or ointment
Triamcinolone acetonide	Aristocort, Kenalog	0.1% cream or ointment
Low Potency		
Alclometasone dipropionate	Aclovate	0.05% cream or ointment
Clocortolone pivalate	Cloderm	0.1% cream
Desonide	DesOwen, Tridesilon	0.05% cream or ointment
Hydrocortisone acetate	Cortaid, Corticaine	0.5% cream or ointment

allows healing to occur uneventfully. Although field workers have mentioned that *Aloe vera* latex empirically improves wound healing, a major study in contact dermatitis showed that it was ineffective.[207] Topical lotions with anesthetics or antihistamines offer no additional benefit and may induce contact sensitization to the chemical additives. Allergic contact dermatitis is a self-healing disease if iatrogenic influences are avoided. Secondary superficial infections may occur in children or during hospitalization. Cleanliness usually prevents this complication.

In the mildly to moderately sensitive person when the eruption is beginning (red and itchy but not yet blistered), potent, fluorinated corticosteroids in a gel or optimized vehicle can prevent blossoming of the rash and speed healing. Many corticosteroid preparations, such as fluocinonide 0.05% gel (Lidex) or clobetasol propionate gel 0.05% (Temovate), are available. Because topical steroids are readily absorbed and can cause adrenal suppression, they should be restricted to limited amounts (less than 15 g total dose) over a brief period (2 to 3 days). Generally, the opportunity to use this approach does not occur in an emergency department, but it can be very effective in helping people repeatedly exposed to poison ivy or poison oak in the field.

As the dermatitis heals and scales form after 10 to 14 days, the patient may note a resurgence of pruritus, which untreated can lead to a patch of subchronic lichenoid neurodermatitis (Fig. 57-15). Judicious use of almost any steroid cream or ointment, such as triamcinolone acetonide 0.025%, desonide 0.025%, or hydrocortisone 1%, helps alleviate symptoms.

Figure 57-15. Resurgence of pruritus leading to patch of subchronic lichenoid neurodermatitis.

Prevention

Prevention involves a combination of avoidance and destruction of the plants. This can be done directly by educating individuals at risk to recognize and avoid these plants, or indirectly by wearing protective clothing and gloves when coming in contact. Rubber gloves that are the thickness of surgical gloves are inadequate protection because urushiol is able to penetrate through rubber. Vinyl gloves are more protective, as are other plastic gloves. Leather gloves are acceptable. Many herbicides are available to kill the plants. Unfortunately, none is specific to *Toxicodendron* species. Proper cleaning of clothing is a must.

Washing immediately with soap (detergent) and water has been shown to destroy urushiol. Washing exposed skin with soap and water within 5 minutes after exposure may decrease the intensity of the dermatitis or even abort a reaction. A lotion containing the organoclay quaternium-18 bentonite is very effective in preventing this type of dermatitis. If applied before contact with the urushiol resin, it has been proven to decrease poison ivy, oak, and sumac dermatitis by inactivating the resin.[118]

The best approaches to prophylaxis come from an intimate understanding of the chemistry of urushiol and the biology of the weeds[53] and reside mainly in recognition and avoidance. Where this is not possible, protective clothing that is either disposable or washable should be worn. Wool is the best material to use as a protective barrier because it binds the allergen readily.[37] Clothing should be washed with detergent or, more preferably, bleach to inactivate urushiol. Tools and other inanimate objects are best cleaned with a dilute solution of bleach. Bleach rapidly inactivates urushiol, and organic solvents such as alcohol, gasoline, and acetone can extract it from contaminated surfaces. A commercially available solvent is Tecnu, sold OTC, but this is merely an inexpensive petroleum solvent sold at a high price. It should not be used for therapy. An excellent choice is rubbing (isopropyl) alcohol, which should be applied liberally for decontamination and followed by liberal use of water wash-off to avoid spreading the oil on the skin. Care should be taken to limit contact time with the skin, particularly with children, who may be susceptible to transcutaneous alcohol toxicity. Use of soap is inferior to better solvents. The newer topical agent Zanfel has been shown to experimentally decrease urushiol-induced contact dermatitis. It is a mixture of alcohol solubles and anionic surfactants that binds to the urushiol antigen and renders it unable to induce an allergic reaction. If applied soon enough after exposure, it has the potential to decrease urushiol-induced allergic contact dermatitis.[40] The idea of using barrier preparations has become popular again, even though in the past, such creams and ointments proved disappointing.[144] The current favorite, an organoclay called Ivy-Block, was developed to protect forestry workers against these weeds during national firefighting escapades.[55] This approach was confirmed in a multicenter study.[117] The lotion can be obtained readily from a pharmacist or by major marketers. Oak-N-Ivy-Armor (Tec Laboratories) is a product marketed to be used as a preventative. It actively binds urushiol and keeps it from contacting the skin. Stokogard outdoor cream, composed of a linoleic ester dimer,[145] was removed from the market for lack of U.S. Food and Drug Administration (FDA) approval but is still available in industrial supply houses that are not regulated. When the cream is first applied, it has a foul smell (like dead fish), resulting from release of the ester. The odor disappears in about 20 minutes, and the cream acts like a barrier to delay the penetration of urushiol oil. It must be washed off in 4 to 6 hours for protection.

Compositae

There are well over 20,000 species of plants in the Compositae family. A brief list of some of the more commonly recognized members appears in Box 57-2. A few highly recognizable members are pictured in Figure 57-16. Compositae species have worldwide distribution. The allergens found in these plants are sesquiterpene lactones. More than 3000 individual sesquiterpene lactones have been identified.[38,69] The lactones are made in the trichomes found on the plant's surface.

Box 57-2. Familiar Compositae Species

Artichoke	Marigold
Black-eyed Susan	Mugwort
Butterweed	Pyrethrum
Chamomile	Ragweed
Chicory	Ragwort
Chrysanthemum	Sagebrush
Cornflower	Sneezeweed
Daisy	Stinking mayweed
Dahlia	Sunflower
Dandelion	Tansy
Endive	Tarragon
Feverfew	Thistle
Goldenrod	Yarrow
Ironweed	Zinnia
Lettuce	

Data from Marks JG Jr, Elsner P, DeLeo VA: Contact and Occupational Dermatology, 3rd ed. St Louis, Mosby, 2002, pp 13–15.

The allergen is not highly sensitizing; thus, individuals typically afflicted are involved in daily handling of plants. These people include florists, nursery workers, horticulturists, and produce handlers. Rarely, home gardeners or people who spend a great deal of time outdoors are afflicted. The dermatitis is fairly characteristic across the Compositae family. The clinical scenario is often a chronic lichenified eruption resembling photodermatitis.[116] Clues that this is not photodermatitis include involvement of the upper eyelids and the submental region of the neck. Definitive diagnosis can be made by patch-testing the patient to various portions of the actual leaf, stem, and petal against appropriate controls. A screening mixture of sesquiterpene lactones is available, but this mixture will miss some relevant allergies to Compositae plants. It is prudent to patch test the patient with the actual plant that is suspected of causing the allergic reaction.

Ragweed (*Ambrosia* species) is a member of the Compositae family of plants; hence, its allergen is a sesquiterpene lactone. Figure 57-17 shows a photo of common ragweed. Ragweed pollen likely contains two individual antigens, one that can cause respiratory ailments such as asthma or allergic rhinitis and another that contains sesquiterpene lactones and causes allergic contact dermatitis. Clinically, patients show involvement of the head and neck region and other areas not covered with clothing. The dermatitis is chronic in nature and tends to mimic photocontact dermatitis. In this case, the upper eyelid and submental regions may be involved, which is not the case in photocontact dermatitis. *Ambrosia deltoidea* is a weed in the Compositae family found in the southwestern United States that has also been reported to cause airborne allergic contact dermatitis. This plant bears little resemblance to the common ragweed.[173]

Wild feverfew (*Parthenium hysterophorus*) is a weed in the Compositae family found throughout the Western hemisphere. It causes chronic lichenified dermatitis in areas exposed to the allergen. It caused epidemic outbreaks of dermatitis in India after it was accidentally transplanted there. It is not a native plant to India and has thrived in its new environment. The

A

C

B

Figure 57-16. Compositae members. **A,** Dahlia. **B,** Chrysanthemum. **C,** Daisy. (**C,** Courtesy of Yves Sell, Institute of Botany, Louis Pasteur University, Strasbourg, France.)

epidemics have become so severe that it has been nicknamed the "scourge of India."[116]

Peruvian lily (*Alstroemeria* species) (Fig. 57-18) is a very popular cut flower. Its allergen is tuliposide A or α-methylene-γ-butyrolactone. Allergic contact dermatitis to this plant is the most common cause of allergic contact dermatitis in florists. Florists who repeatedly cut the flowers and stems are chronically exposed to the allergen. Subsequently, they have the highest sensitization rate. It is a very rare cause of allergic contact dermatitis in individuals outside of the floral industry.

Liverworts (*Frullania* species) are members of the Jubulaceae family, related to mosses, that live commonly on tree bark. They are small reddish-brown plants found most often in the Pacific Northwest. There are hundreds of species of *Frullania* plants.

The dermatitis is a chronic lichenified eruption very similar in nature to Compositae dermatitis. The allergen is sesquiterpene lactone. Allergic contact dermatitis due to *Frullania* is a problem among lumberjacks and forest workers, but it may affect any individual who takes a walk in the woods.[116]

Figure 57-17. Ragweed, *Ambrosia* species.

Figure 57-18. A familiar cut flower, Peruvian lily (*Alstroemeria* species).

Grevillea banksii and *Grevillea robusta* have been implicated as causing allergic contact dermatitis. These plants are native to Australia but have been transplanted around the globe. They grow as shrubs and are used in domestic landscaping. The allergen is a resorcinol.

Allergic contact dermatitis to lichens is seen most often in forestry workers and gardeners. Lichens live worldwide and grow just about anywhere. They are composed of algae and fungi that live in a symbiotic relationship. The allergens are usnic acid, atranorin, and evernic acid.[116] The clinical picture is very similar to Compositae dermatitis.[189]

The false heliotrope plant (*Phacelia crenulata*) is found in the deserts of the southwestern United States. It causes acute allergic contact dermatitis in the direct areas of contact. A linear papulovesicular reaction, not unlike the reaction to *Toxicodendron,* is seen.

Two common, easily recognized plants that can cause allergic contact dermatitis are the tulip and primrose. The tulip (*Tulipia* species) is an early spring flowering plant. Its allergen is tuliposide A. Tuliposide A is the same allergen found in *Alstroemeria*. The allergen has its highest concentration in the epidermis of the bulb,[76] from which originate the clinical findings of "tulip fingers," a chronic fissured and scaly eczematous rash of the fingertips in people whom routinely handle the bulbs (Fig. 57-19). This is most common in commercial gardeners; occasionally, an avid home gardener is affected. People who are allergic to tulips should avoid *Alstroemeria*.

Primrose (*Primula obconica*) is a common houseplant reported to cause allergic contact dermatitis in home gardeners (Fig. 57-20). Its allergen is primin (2-methoxy-6-pentyl-1,4-benzoquinone). The allergen is found in the highest concentration in the trichomes of the stem and leaves. A genetically altered hybrid has been produced that does not make primin and thus has no potential to induce allergic contact dermatitis. This hybrid is likely responsible for the decrease noted in the incidence of primin-induced allergic contact dermatitis.[33]

Figure 57-19. Eczematous scaly patches on the fingers of a floral worker.

The creosote bush (*Larrea* species) is found throughout North and South America. It is a shrubby bush with leaves covered by a resinous substance. There are a few reports of this resin causing allergic contact dermatitis.[106]

The Hydrophyllaceae family of plants contains a class of chemicals called *phacelioids*.[158] These substances are related to urushiol and can be potent allergens. However, they do not cross-react with urushiol.

Figure 57-20. Primrose, a common houseplant.

CONTACT URTICARIA: IMMUNOLOGIC AND NONIMMUNOLOGIC

Contact urticaria can be considered to be immunologically induced or nonimmunologically induced. The immunologic subtype is due to an immediate hypersensitivity reaction requiring antibody formation to a particular substance. These type I reactions show a broad clinical spectrum, from mild skin hives to anaphylaxis. Prick testing and radioallergosorbent test (RAST) testing are of benefit in determining the cause in certain select cases. The immunologic form of urticaria depends on the individual patient's immune activity or hyperactivity. There is no predictable manner to determine to which plants a person may develop a type I reaction. Food handlers and persons otherwise constantly exposed to plant products are likely to be more at risk. Many plant species have been reported to cause contact immunologic urticaria.[101,114] Patients typically present with urticarial wheals in the areas of exposure within 1 to 2 hours of handling a particular plant. Occasionally, there is oral involvement with tongue and lip swelling. This is nearly always from eating a fruit. Box 57-3 is a partial list of some plants causing contact immunologic urticaria. Atopic individuals have been reported to be at higher risk for type I contact urticaria.[114]

Nonimmunologic plant contact dermatitis is caused by direct release of urticating substances onto or into the skin. Four main families of plants contain stinging hairs or spines that can cause contact urticaria: Euphorbiaceae, Hydrophyllaceae, Loasaceae, and Urticaceae.[114] Most of these plants are found in the tropics, with the exception of stinging nettles, which have a worldwide distribution and are found throughout the United States. Box 57-4 lists some of the nettle plants. The most common plant to cause contact urticaria is the stinging nettle *Urtica dioica*.

Box 57-3. Plants That Cause Immunogenic Urticaria

FRUITS
Apple
Carrot
Celery
Parsley
Potato
Tomato

PLANTS
Tulip

SPICES
Cinnamon
Garlic
Mustard
Rapeseed

TREES
Birch pollen
Western red cedar

VEGETABLES
Chives
Grains
Lettuce
Onion

Box 57-4. Plants in the Nettle Family

Urtica chamaedryoides
Urtica dioica
Urtica gracilis
Urtica ferox
Urtica membranacea
Urtica urens
Urtica pilulifera
Urtica parviflora

Within the *Urticaceae* family reside other plants capable of producing contact urticaria by means of the nonimmunologic route. Most skin reactions are acute. They typically resolve spontaneously, so the advice of a physician is never sought. Persistent paresthesia lasting hours has been reported.[124]

Figure 57-21A and B shows the plant and a close-up view of the spines. Contact with spines on the plant causes release of biologically active substances from the spines. These include histamine, acetylcholine, 5-hydroxytryptamine (serotonin), leukotriene B_4 (LTB_4), and leukotriene C_4 (LTC_4).[6,32,39,46] These chemicals lead directly to urticaria. There is no standardized therapy for stinging nettle dermatitis. Figure 57-21C illustrates the faint urticarial papules seen after contact with the stinging nettle plant.

Other plants, including chili pepper (*Capsicum* species) and cowhage (*Mucuna pruriens*), can release urticating substances onto the skin and cause urticaria. In the case of chili pepper, the released chemical is capsaicin; with cowhage, it is mucanain.[116]

The pathogenesis of immunogenic contact urticaria is a variation of immediate type I hypersensitivity. The central cytologic reactor in skin is the mast cell, which has high-affinity IgE receptors in its membrane. An IgE molecule binds to the receptor by its Fc portion, exposing the Fab segments as recognition sites. When a divalent protein antigen appropriately bridges two IgE molecules, a series of biochemical events transpires that leads

A

B

C

Figure 57-21. **A,** Stinging nettle. **B,** Close-up view of the stinging nettle spines. **C,** Urticarial papules induced after contact with the stinging nettle.

to mast cell degranulation. The plasma membrane is perturbed. Several lipids are phosphorylated and G proteins are activated, which in turn activate phospholipase C to hydrolyze phosphatidylinositol 4,5-bisphosphate (PIP_2) and yield two messengers: diacylglycerol (DG) and inositol triphosphate ($InsP_3$).[22] $InsP_3$ binds to its receptor on the endoplasmic reticulum, forming a calcium channel to release free calcium ions (Ca^{2+}) into the cytosol.[11,16,83,141] Simultaneously, DG activates protein kinase C in the plasma membrane, opening calcium channels and allowing entrance of extracellular Ca^{2+}, which further loads

the cytosol with free Ca^{2+}, an important "messenger" in the stimulus secretion process.[108] G proteins interact with and release the nucleotide complexed to protein α-chains,[186] some of which probably inhibit or stimulate cyclic adenosine phosphate and its actions as a second messenger.[22]

The result of the extensive alteration in intracellular milieu is activation of a serine proteinase and exocytosis of mast cell granule contents.[44,175] These come in three forms: preformed and rapidly released; preformed, bound to the granule matrix of heparin, and slowly released; and newly formed mediators. In addition, mast cells produce a variety of cytokines, including IL-1, IL-3, IL-4, IL-5, and IL-6; GM-CSF; and TNF-α.[184] These cytokines interact with cells and structures, such as endothelium in the skin, to amplify the various inflammatory responses.[44,175,184] In acute urticaria, the rapidly released preformed mediators account for most of the signs and symptoms. These mediators include histamine, some chemotactic factors, and arylsulfatase. If the lesions persist or extend, other mediators, such as newly formed leukotrienes, heparin (or heparin fragments), cytokines, and a number of proteases, may be involved in the continuing tissue damage.

The sequence of inflammatory events leading to acute urticaria is most likely to occur in people with an atopic background, especially those with pollen allergies.[101] This is the same group of people, mainly women and especially health care workers, who have been found in recent years to be exquisitely sensitive to latex rubber gloves[26,104,179,193] and sensitive to proteins in the natural latex from rubber trees in Asia.[134]

In the past decade, this has become an escalating problem. It now appears that there are more than 200 polypeptides produced in natural latex rubber. Many of these are contact sensitizers, so that a single antigen is not likely to be identified for a given patient. The major problem is IgE-mediated immediate-type hypersensitivity and the potential for anaphylaxis. In addition to these polypeptides appearing in natural rubber latex, many are also found in a large number of plants, particularly those consumed as food. These allergens are constantly being purified and characterized. One suggestion is that these cross-reacting allergens are "defense proteins" with a role to protect the plant from attack by pathogens.[103] Whether this is true requires further investigation, but it is also possible that by plant engineering, these allergens may be genetically restricted from the foods we consume.

In addition to fruit and nuts, mustard in pizza has been implicated in anaphylaxis.[146] In less severe cases, vesicular, eczematous rashes have been noted within hours of eating mustard or rapeseed, which is used widely in production of vegetable oils and margarine.[126] Erythema multiforme-like eruptions are well recognized after contact with bracelets and ornamental necklaces made of exotic woods, such as *Dalbergia nigra*.[62] Erythema multiforme has also been seen after exposure to more common plants, such as poison ivy, primula, and mugwort.[62,200] It is theorized that multiforme lesions result from vasculitis caused by deposition of immune complexes in or around the blood vessels.

Diagnosis of immunogenic contact urticaria can be confirmed by simple tests. As a use test, application of the suspected plant product to the antecubital fossa twice a day for several days may reproduce the wheal response. Open and closed patch tests, with examination of test sites in 2 to 6 hours, can be useful, as can more conventional prick, scratch, or scratch-chamber tests, in which the results are read in 15 to 20 minutes.[101] It is most important to determine whether urtication is immunologic or nonimmunologic in nature.[68,101] For complete evaluation, an allergist obtains radioallergosorbent tests to quantify specific IgE in the patient's serum. More refined serologic tests include crossed radioimmunoelectrophoresis (CRIE) and CRIE inhibition.[28]

Patients with contact urticaria visit the emergency department when an eruption is extensive or extreme, or if it is associated with stridor, wheezing, and collapse. Because mast cell degranulation is the central problem, and because epinephrine stimulates cyclic adenosine monophosphate formation that opposes degranulation, it is logical to use this drug. Other supportive treatments for anaphylactic shock may be required, such as albuterol, aminophylline, oxygen, or IV hydrocortisone. In less severe cases, antihistamines are valuable. IM or IV diphenhydramine (Benadryl), in an adult dose of 25 to 50 mg, usually stops progression of wheal formation and can be followed by oral hydroxyzine (10 to 25 mg three times a day) or cyproheptadine (4 mg three times a day) for 2 to 5 days. Pure H_1-blockers, such as fexofenadine (60 mg two times a day), are also effective and do not depress the central nervous system, although the prescriber has to be aware of any unique adverse reactions. It is important to make certain the patient has not inadvertently hidden parts of the plant on the body, in clothing, or in a towel, blanket, or knapsack. Recrudescence of the urticarial response usually can be traced to continuing unknown contact with the offending agent.

Phytophoto Reactions

Plants and their components can interact with ultraviolet rays of the sun and induce phytophotodermatitis. There are two types of phytophotodermatitis: phototoxic and photoallergic. Phototoxic reactions are analogous to irritant contact dermatitis, and photoallergic reactions are analogous to allergic contact dermatitis. Phototoxic reactions are encountered more frequently. They are often recognized by a clinician's knowledge of offending agents and the morphology of the exanthem. The reactions most commonly appear as linear red patches and plaques with or without edema, but they can be bullous in nature.[195] A characteristic finding is postinflammatory hyperpigmentation lasting for months to years. Most of these reactions are likely to go unreported or unrecognized, most often because of their mild nature, but also because it is often difficult to pinpoint the cause of the problem.

Phototoxic reactions are typically dose dependent and can occur in anyone if sufficient concentrations of ultraviolet light and the toxic plant material are achieved. In general, the wavelength of light needed to induce the most severe reactions is ultraviolet A (UVA), 320 to 400 nm.

For most phototoxic reactions, the toxin is a furocoumarin (psoralen). Furocoumarins interact with DNA and cause formation of pyrimidine dimers, which interrupt DNA synthesis. This ultimately results in cell damage and the clinical effects. Ultraviolet light interacts with the psoralen molecule (phototoxic chemical) and raises the psoralen molecule from the ground to an excited state. When the excited molecule returns to its ground state, energy is released that causes the chemical to cross-link two strands of DNA or attach itself to one strand of DNA, thus interfering with DNA synthesis and ultimately cell division. This reaction is used to our advantage in treatments such as PUVA (psoralen plus ultraviolet A) light therapy and in photopheresis. PUVA therapies are used for conditions

Figure 57-22. Phototoxic plants. **A,** Fig tree. **B,** Gas plant. (**B,** Courtesy of Yves Sell, Institute of Botany, Louis Pasteur University, Strasbourg, France.)

ranging from vitiligo and psoriasis to cutaneous T-cell lymphoma. Photopheresis is a treatment modality wherein a patient is connected by an IV line to a pheresis machine. Blood is drawn out of the patient and exposed to a photoactive drug (psoralen). Then the blood is exposed to ultraviolet light (UVA) and returned to the person. This causes cross-linking of DNA in the pathogenic T cells and death of these cells. This is used almost exclusively for cutaneous T-cell lymphoma and graft-versus-host disease.

PHYTOPHOTOTOXIC CONTACT DERMATITIS

Furocoumarins include psoralen, 5-methoxy-psoralen (bergapten), 8-methoxy-psoralen (xanthotoxin), angelicin (isopsoralen), 5-hydroxy-psoralen (bergaptol), 8-hydroxy-psoralen (xanthotoxol), and limettin.[12,195] Psoralens are modified furocoumarins. Most of these chemicals are found in four plant families: Umbelliferae, Rutaceae, Moraceae, and Leguminosae. Figure 57-22 shows a few examples of these plants. Figure 57-23 shows an example of the dermatitis that can result from exposure.

Table 57-10 lists members of the families implicated in phytophototoxic contact dermatitis. Umbelliferae is a well-recognized family that has an umbrella-like configuration to its flowers. In the United States, this family of plants is the most common cause of phytophototoxic dermatitis. The Moraceae and Leguminosae families are rare causes in the United States.[185]

So-called meadow dermatitis (dermatitis bullosa striata et pratensis) occurs after exposure of wet human skin to psoralen-containing plants during daylight hours.[12] The most common offending plants are the yellow-flowered wild meadow parsnip and wild yellow flowered herb.[161] Bizarre linear configurations, such as an imprint, are often found on the lower extremities on nonclothing-protected skin after a walk through a field of a psoralen-containing weed.

Figure 57-23. Phototoxic dermatitis induced by furocoumarins.

Agriculture field and grocery workers, in particular celery handlers, have been the most frequently reported to have problems with phytophototoxic dermatitis. Another occupation that has reports of phytophototoxic dermatitis is bartending. Lime handling while mixing drinks, if combined with ultraviolet light, may cause this dermatitis.[5,35,62,75,181,197]

Berloque (pendant- or drop-like) dermatitis is perfume-induced phytophotodermatitis. Typically, a patient seeks care for hyperpigmented macules on the head and neck, with no obvious preexisting rash or dermatitis. Unfortunately, the

TABLE 57-10. Common Members of the Umbelliferae, Rutaceae, Moraceae, and Leguminosae Families Found to Cause Phototoxic Contact Dermatitis

COMMON NAME	BOTANICAL NAME
Umbelliferae	
Angelica	*Angelica gigas*
Celery	*Apium graveolens dulce*
Carrot	*Daucus carota*
Cow parsley	*Heracleum sphondylium*
Cow parsnip	*Heracleum lanatum*
Dill	*Anethum graveolus*
False bishop's weed	*Ammi majus*
Fennel	*Foenialum vulgare*
Giant hogweed	*Heracleum mantegazzianium*
Parsley	*Petroselinum crispum*
Parsnip	*Pastinaca sativa*
Queen Anne's lace	*Ammi majus*
Spring parsley	*Cymopterus watsonii*
Moraceae	
Fig	*Ficus carica*
Rutaceae	
Bergamot lime	*Citrus bergamia*
Berry rue	*Cneoridium dumosum*
Common rue	*Ruta graveolens*
Gas plant (burning bush)	*Dictamnus albus*
Grapefruit	*Citrus paradissi*
Lemon	*Citrus limon*
Lime	*Citrus aurantifolia*
Mexican lime	*Citrus aurantifolia*
Mokihana	*Pelea anisata*
Orange	*Citrus sinensis*
Zabon	*Citrus maxima*
Leguminosae	
Bavchi (scurf-pea)	*Psoralea corylifolia*
Citrina	*Coronilla glauca*

Data from references 24, 27, 79, 86, 114, and 195.

hyperpigmentation can last for years. The original cause of this reaction was from oil of bergamot, which is derived from bergamot oranges and used in many perfumes. The oil contains a mixture of psoralens, including 5-methoxy psoralen (5-MOP).

Pseudophytophotodermatitis is a reaction that has been reported to occur after prolonged human exposure to celery that has been infected with a psoralen-producing fungus (*Sclerotinia sclerotiorum*), more commonly known as "pinkrot." This occurs primarily in field workers who are responsible for harvesting the plants.[185]

Pseudophytodermatitis has been used to describe dermatitis caused by arthropods that live on the plant or chemical agents that have been applied to the plants.[185] One common arthropod causing dermatitis is the grain itch mite *Pediculoides ventricosus*, which can infest straw or hay. Numerous chemicals that are purposefully applied to plants can also cause contact dermatitis. Most commonly these are insecticides, herbicides, or fungicides (Box 57-5). Waxes or azo-dyes applied to various fruits and vegetables can also cause pseudophytodermatitis. Azo-dyes are synthetic dyes containing the azo group (two connected nitrogen atoms) and are used in many industries, including the food industry.

Box 57-5. Some Insecticides, Fungicides, and Herbicides That Cause Irritant Dermatitis (Pseudophytodermatitis)

Captan
Captofol
Difoltan
Dithiocarbamates
Folpet
Mancozeb (manganese and zinc ethylene-*bis*-dithiocarbamate)
Maneb (manganese ethylene-*bis*-dithiocarbamate)
Organophosphates
Paraquat
Phtahan
Randox
Sulfur

Data from Craigmill AL (ed): Environmental Toxicology Newsletter. University of California, Davis. 1982;3(1):1–5; and Mark K, Brancaccio RR, Soter NA, Cohen DE: Allergic contact and photoallergic contact dermatitis to plants and pesticide allergens. Arch Dermatol 1999;135(1):67–70.

PHYTOPHOTOALLERGIC CONTACT DERMATITIS

The phytophotoallergic reaction is exceedingly rare. It is likely under-recognized and under-reported. Rare case reports are found throughout the literature. The plants reported to cause this reaction are tromso palm (*Hercacleum laciniatum*),[114] feverfew (*Parthenium hysterophorus*),[17] garlic (*Allium sativum*),[4] *Frullania*,[4,25] and some coumarin (psoralen)–containing plants.[4,89,110] The clinical importance of these reactions is unknown. In the future, better screening methods may increase recognition of these reactions. Until then, only clinicians with access to extended patch-testing series and/or with sufficient suspicion and knowledge will recognize these reactions.

WOOD DERMATITIS

Typically, wood dust causes eczematous dermatitis of the irritant or allergic type. The victims are nearly always woodworkers who have been exposed repeatedly. Allergic contact dermatitis occasionally develops. Rarely, an erythema multiforme–like configuration is noted.[62] Respiratory hypersensitivity, including asthma, to wood dusts is well documented.[182] Solid wood has very rarely been reported to cause dermatitis.[187] Table 57-11 lists the most commonly reported woods to cause contact dermatitis. Dermatitis from woods is almost entirely seen in an occupational setting.

GRANULOMATOUS AND FOREIGN-BODY REACTIONS

Any foreign body embedded in the skin can cause a granulomatous reaction pattern. Some plants are more prone to cause this type of inflammatory reaction. Plants with thorns and barbs

TABLE 57-11. Sampling of Wood Trees That Cause Contact Dermatitis

COMMON NAME	BOTANICAL NAME
African black walnut	*Mansonia altissima*
African blackwood	*Dalbergia melanoxylon*
Bolivian rosewood	*Machaerium acutifolium*
Brazilian box tree	*Aspidosperma* species
Cocobolo	*Dalbergia retusa*
Cordia	*Cordia goeldiana*
East Indian rosewood	*Dalbergia latifolia*
Ebony	*Diospyros celebica*
Honduran mahogany	*Swietenia macrophylla*
Iroko	*Chlorophora excelsa*
Litre	*Lithrea caustica*
Macassar ebony	*Diospyros celebica*
Mahogany	*Khaya* species
Milo wood	*Thespesia populnea*
Obeche	*Triplochiton scleroxylon*
Padauk	*Pterocarpus dalbergiodes*
Pao ferro	*Machaerium scleroxylum*
Perupok	*Lophopetalum dubicum*
Pine	*Pinus* species
Redwood	*Sequioa sempervirens*
Sapele wood	*Entandrophragma cylindricum*
Silky oak	*Grevillea robusta*
Sucupira	*Bowdichia nitida*
Tali wood	*Erythrophleum guineense*
Teak	*Tectona grandis*
Walnut	*Juglans nigra*
White ash	*Fraxinus americanus*
Zebrawood	*Astronium fraxinifolium*

Data from references 5, 8, 35, 37, 73, 75, 114, and 171.

A

B

Figure 57-24. Many plants (including these, from Peru) have evolved thorns and spines in self-defense. (Courtesy of Paul S. Auerbach.)

(Fig. 57-24) are the most likely culprits. Rose thorns and cactus spines are common offenders. Cacti are indigenous to the southwestern United States. They are popular as houseplants and can be grown indoors with proper care. Therefore, cactus injuries can be seen in a wide variety of locales.[180] The initial contact and acute injury may lead to chronic granulomatous inflammatory rash, which typically takes 4 to 8 weeks to develop. It is the lag time that may make the diagnosis difficult. Clinically, the rash consists of erythematous and indurated papules, plaques, and nodules. The lesions are often grouped and localized, which is a clue to the diagnosis. Cactus spines may also penetrate so deeply into the skin that they cause pseudotumors of bone.[161]

The prickly pear (*Opuntia ficus-indica*) is found in North and Central America, as well as around the Mediterranean Sea. It is a member of the Cactaceae family. The fruit is covered with glochids. Recall that glochids are modified leaves that actually appear as barbed hairs or thorns. These bristle-like structures give the prickly pear its name. The prickly pear induces dermatitis in people who contact the plant, typically after picking the fruit. Glochids are difficult to remove once embedded in the skin. They have a tendency to break off from the cactus and work their way onto the skin, causing a reaction that may resemble scabies.[161] They can also implant into the conjunctiva.[202]

When biopsies are performed of the chronic (4 weeks after initial injury) inflammatory papules that are caused by cacti glochid implantation, a granulomatous reaction pattern is found.[109] Multiple multinucleated giant cells, histiocytes, granulomas, and organic plant material can be seen histologically under polarized light sources. Occasionally, a traumatic injury may implant one or more microbes. When performing a biopsy, one should also perform a tissue culture for bacteria, mycobacterium, and fungal species. This is especially true in cases with pustular morphologies.

Initial treatment should be prompt extraction of the cactus spines. The smaller the spine, the more difficult the removal.[109] This is a particular problem when encountering injuries from the beaver tail cactus (Fig. 57-25), which has very small glochids that can be quite difficult to remove. Many methods of removal

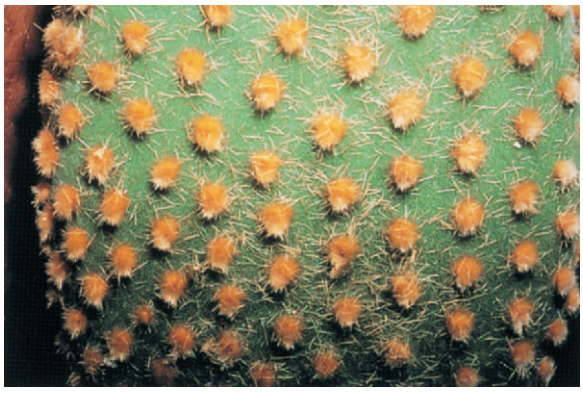

Figure 57-25. Beaver tail cactus. (Courtesy of Eric Lewis.)

have been employed to remove the spines. Tweezers are often the first line of removal, and various gels, glue, tape, and facial masks have been tried.[119] The best method is mechanical removal with a small forceps or fine needle. Once removal is accomplished, a topical corticosteroid can be employed. A mid-potency topical corticosteroid, such as triamcinolone acetonide (Aristocort, Kenalog) 0.1% cream or ointment, is often all that is needed. Most granulomatous reactions resolve within 2 to 4 months.[41] Often, the removal is unsuccessful, and the glochids work their way to the surface over a period of months to years. Supportive care with cool compresses with an aluminum acetate solution 1:40 in water used as soak, compress, or wet dressing is very helpful. Treatment with pain medication and prompt treatment of any coinfection need to be an integral part of the overall treatment plan.

The references for this chapter can be found on the accompanying DVD-ROM.

58 Toxic Plant Ingestions

Kimberlie A. Graeme

Toxic plants are ingested by curious children, by foragers mistaking poisonous plants for edible fare, by herbalists mistaking poisonous plants for nontoxic remedies, by pleasure seekers attempting to attain natural highs, and by suicidal patients attempting to harm themselves. Over 77,000 plant ingestions or exposures were reported to 64 poison control centers in 2003, representing 3.2% of all human toxic exposures reported that year. Plant exposures are more common in the pediatric population and accounted for 4.6% of poison center calls regarding children less than 6 years of age. The calls were most frequently about peace lily, poinsettia, philodendron, holly, and pokeweed. In 2003, three plant deaths were reported to these poison control centers.[210] A review of plant poisonings reported to U.S. poison control centers over an 18-year period (1983–2000) revealed 30 fatalities, with seven due to *Cicuta* species (water hemlock) and five due to *Datura stamonium* (jimsonweed).[121] Worldwide, *Atropa belladonna* (deadly nightshade), *Hyoscyamus niger* (black henbane), *Conium maculatum* (poison hemlock), *Thevetia peruviana* (yellow oleander), *Nerium oleander* (common oleander), *Cerbera manghas* (pink-eyed cerbera or sea mango), *Colchicum autumnale* (meadow saffron), *Gloriosa superba* (glory lily), *Veratrum album* (hellebore), *Ricinus communis* (castor bean), *Atractylis gummifera* (bird-lime or blue thistle), and *Blighia sapida* (ackee fruit) also cause significant morbidity and mortality.[65,72,93,112,135,208]

GENERAL CONSIDERATIONS

Supportive care takes priority over identification of plants ingested, but the history should include time of ingestion, amount and part of plants ingested, initial symptoms, and time between ingestion and onset of symptoms. Method of preparation (e.g., drying, cooking, boiling), number of persons who ate the same plant, and symptoms are important considerations.

Airway, breathing, and circulation are assessed, and then adequacy of hydration, end-organ perfusion, and urine output. Oral administration of activated charcoal (1 g/kg), up to 50 gm maximum, may aid gastrointestinal (GI) decontamination. Laboratory studies depend on clinical presentation and suspected plant exposure. Complications of poisoning include aspiration pneumonia, rhabdomyolysis, and deep venous thrombosis.

The differential diagnosis is initially kept broad so that other illnesses, such as infection and trauma, are not missed. Table 58-1 provides signs and symptoms of toxic plant ingestions.

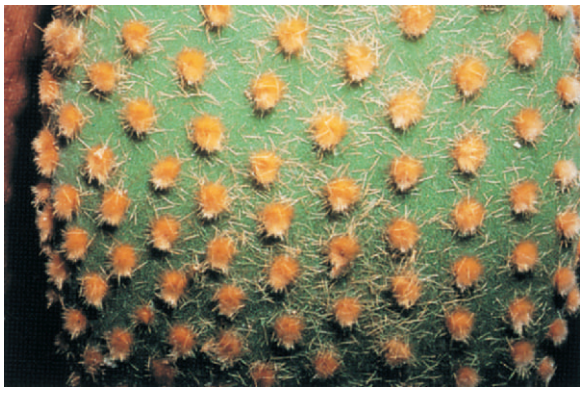

Figure 57-25. Beaver tail cactus. (Courtesy of Eric Lewis.)

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The differential diagnosis is initially kept broad so that other illnesses, such as infection and trauma, are not missed. Table 58-1 provides signs and symptoms of toxic plant ingestions.

TABLE 58-1. Signs and Symptoms of Plant Intoxications

		Dieffenbachia, Philodendron	*Colchicum, Gloriosa*	*Euphorbia, Hippomane*	*Actaea, Anemone ranunculus*	*Convallaria, Digitalis, Nerium*	*Aconitum*	*Solanum*	*Pieris, Rhododendron, Veratrum*	*Conium, Laburnum, Nicotiana, Sophora*	*Cicuta*	*Taxus*	*Gelsemium*	*Brugmansia, Datura*	*Amaryllis, Narcissus, Wisteria*	*Ilex*	*Abrus, Ricinus*	*Prunus*	*Phytolacca*	*Podophyllum*	*Karwinskia*
MOUTH & THROAT	Burning, irritation	++	++	D	++	+	+	D	+	+											
	Increased salivation	+		D	++		+		D	+	+	D									
	Dry mouth											+	+	++							
	Dysphonia, dysphagia	+		D			±						+	+							
GI TRACT	Nausea		+			+	±		D	+	+				+	+	DD				
	Vomiting		+	+	+	+	±		D	+	±	D			++	++	DD	D	++	+	
	Diarrhea		++	+	+	+		D	D	+					±	DD		D	+		
	Abdominal pain		++	++	+	+		D	D			D			+			D	+		
	Decreased bowel sounds													++						±	
CARDIOVASCULAR	Tachycardia									+				+							
	Bradycardia					+			+			D									
	Arrhythmias					±	++					D									
	Conduction defects					++															
	Hypertension													+							
	Hypotension								++			D									

Continued

PLANT TOXINS

The discussion of plant toxins in this chapter is arranged for the most part on the basis of the organ systems primarily affected by them: the central nervous, cardiovascular, GI, renal, endocrine/metabolic, hematopoietic, or reproductive system. Mention will also be made of the chemical group to which they belong, which depends on their chemical structure. Most fall into one of the following categories: alkaloids, glycosides, resins, oxalates, or phytotoxins.

Alkaloids. Alkaloids are nitrogen-containing organic compounds that act as bases and form salts with acids. Plant alkaloids are soluble organic acid–alkaloid salts that contain nitrogen in a ring structure that is heterocyclic or aromatic, or both. Alkaloids are generally distributed throughout a given plant, so all ingested parts are toxic. Further subdivision into chemical groups is based on ring structure (Table 58-2).

Glycosides. Sugars in the form of acetals are called glycosides. In glycosides, a glycosyl group replaces an alcohol or a hydroxyl

TABLE 58-1. Signs and Symptoms of Plant Intoxications—Cont'd

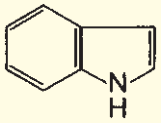
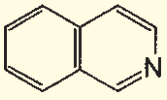
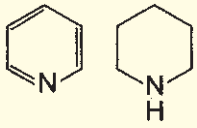
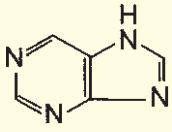
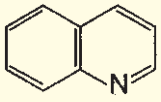
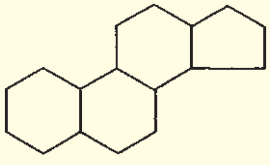
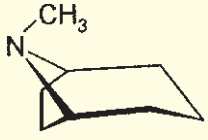
		Dieffenbachia, Philodendron	*Colchicum, Gloriosa*	*Euphorbia, Hippomane*	*Actaea, Anemone ranunculus*	*Convallaria, Digitalis, Nerium*	*Aconitum*	*Solanum*	*Pieris, Rhododendron, Veratrum*	*Conium, Laburnum, Nicotiana, Sophora*	*Cicuta*	*Taxus*	*Gelsemium*	*Brugmansia, Datura*	*Amaryllis, Narcissus, Wisteria*	*Ilex*	*Abrus, Ricinus*	*Prunus*	*Phytolacca*	*Podophyllum*	*Karwinskia*
NERVOUS & NEUROMUSCULAR	Dizziness				±		+		+	+		+	+								
	Weakness, lethargy						+			+		D						D			
	Syncope				±																
	Delirium, psychosis				±										+						
	Tremors, convulsions				±				D±		++		±					D±			
	Depression, coma						+		D	+		D						D±		±	
	Headache								+	±			+								
	Paresthesias						++		D												
	Muscle weakness, paralysis		±						D	+			+					D±		±	DD
VISUAL	Mydriasis						+			±		+		+							
	Visual disturbances						+		D				+	+							
CUTANEOUS	Increased sweating								+	+								D	++		
	Dry skin													++							
	Flushing, rash											D		+							
	Cyanosis											D						D±			
MISC.	Hyperthermia							+						+							
	Painful, bloody micturition		+		+																

+, commonly occurs; ++, pronounced or persistent; ±, occasionally reported; D, delayed onset; DD, occurrence significantly delayed.
Modified from Lampe KF, McCann MA: AMA Handbook of Poisonous and Injurious Plants. Chicago, American Medical Association, 1985.

group. On hydrolysis, glycosides yield sugars, or glycones, and aglycone compounds. The aglycone moiety accounts for most of the toxicity, although the sugar may enhance solubility and absorption (Fig. 58-1). Glycoside-producing plants include cardioactive, cyanogenic, saponin, anthraquinone, and coumarin glycoside compounds.

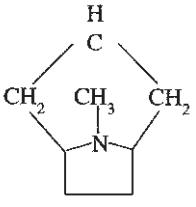
Resins. Resins are highly toxic compounds of diverse chemical and plant origin united by the physical characteristics of insolubility in water, absence of nitrogen, and solid or semisolid state on extraction at room temperature. Resins are usually mixed with other compounds, such as volatile or essential oils (oleoresins), gum (gum resins), and sugars (glycoresins).

TABLE 58-2. Plant Alkaloids and Their Structures

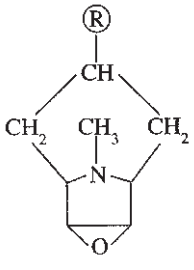
ALKALOID TYPE	ALKALOID STRUCTURE	EXAMPLES
Indole		Ergonovine (ergots) Strychnine Physostigmine (calabar beans) Rauwolfia alkaloids (reserpine)
Isoquinoline		Opium alkaloids Emetine (ipecac)
Pyridine/piperidine		Nicotine (tobacco) Arecoline (betel nut) Lobeline (Indian tobacco)
Purine		Caffeine Theobromine (cacao)
Quinoline		Cinchona alkaloids (quinidine)
Steroid		Veratrum alkaloids (false hellebore) Aconite (monkshood)
Tropane		Atropine (belladonna) Hyoscyamine Hyoscine (scopolamine)

Oxalates. Oxalates occur naturally in plants as soluble (sodium or potassium) or insoluble (calcium) oxalates or acid oxalates. Oxalates have corrosive effects and bind serum calcium, causing hypocalcemia.

Phytotoxins. Phytotoxins, or toxalbumins, are among the most toxic substances of plant origin. They are composed of large protein molecules that resemble bacterial toxins in structure and in their ability to act as antigens (Fig. 58-2).



Tropane



Scopolamine

CENTRAL NERVOUS SYSTEM TOXINS

Anticholinergic Plants (Tropane Alkaloids)

Plants containing tropane alkaloids (often called belladonna alkaloids) include atropine, hyoscyamine (the levorotatory isomer of atropine), and hyoscine (scopolamine).[29,111,113,135,139,177,199] Structures of the tropane alkaloids follow:

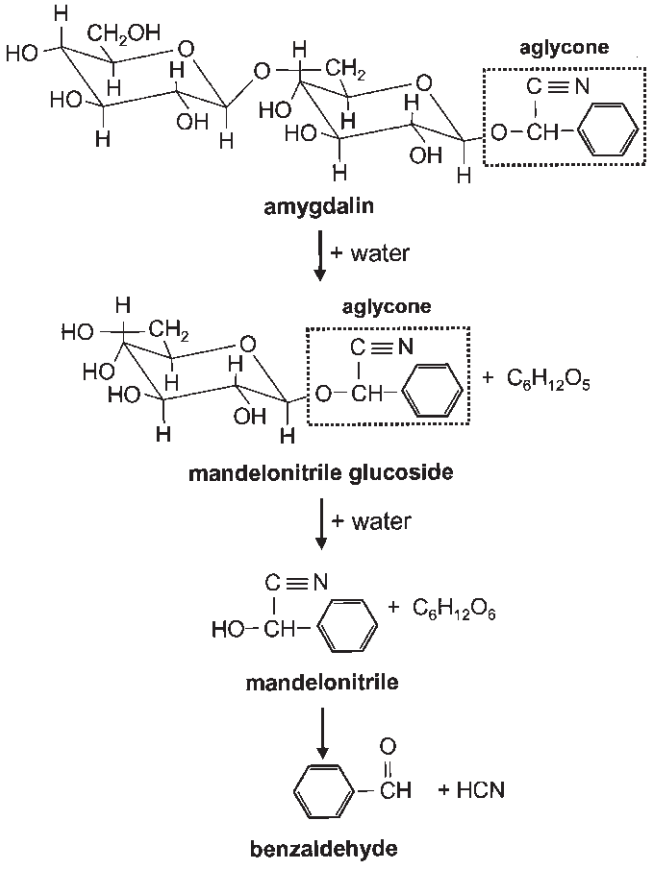
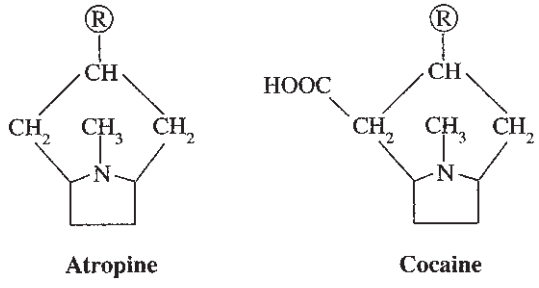


Figure 58-1. Hydrolysis of amygdalin, with its toxic aglycone group, yields hydrogen cyanide. The enzyme β-glucosidase, called emulsin, contained in plants can catalyze amygdalin hydrolysis.



Tropane alkaloids are found in about 25 genera and 2000 species of plants. Plants causing human toxicity include *A. belladonna* (deadly nightshade), *Mandragora* species (mandrake), *Hyoscyamus niger* (black henbane), *Datura* species (jimsonweed), and *Brugmansia* species (angel's trumpet).* The Solanaceae family includes *Solanum*, and *Scopolia carniolica*, the source of scopolamine.

*References 29, 87, 111, 113, 135, 139, 160, 163, 177, 188.

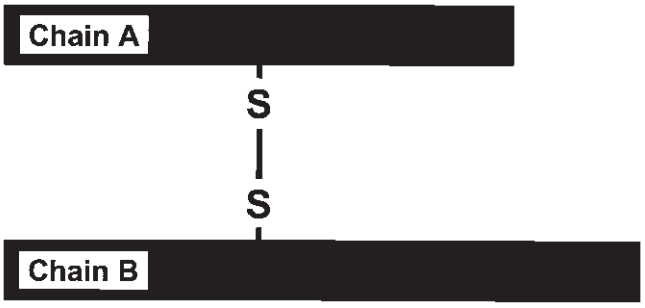


Figure 58-2. Structure of ricin and abrin, phytotoxins isolated from the castor bean plant and the jequirity bean plant, respectively, are similar in structure to biologic toxins such as botulinum. These glycoproteins are composed of two peptide chains, designated A and B, connected by a disulfide bond.

Anticholinergic Syndrome. Toxicity may ensue after ingesting or smoking plant parts.[37] The tropane alkaloids inhibit postsynaptic muscarinic receptors competitively, producing the classic anticholinergic syndrome (Fig. 58-3 and Box 58-1).[79,87,160,163,177] A useful clinical sign of anticholinergic toxicity is dry axillae (i.e., lack of perspiration). Anticholinergic findings suggesting poisoning may be remembered using the following mnemonic[163,181,187]:

Hot as a hare (or hot as Hades),
Blind as a bat,
Dry as a bone,
Red as a beet,
Mad as a hatter.

Jimsonweed. *Datura* species are generally known as jimsonweed or thorn apple, and *Brugmansia* species are generally known as angel's trumpet (Fig. 58-4).[86,87,204] Young, thin, tender stems of jimsonweed contain the highest concentration of tropane alkaloids.[139] However, the seeds also contain high concentrations of the alkaloids, and as little as half a teaspoonful of seeds may cause death from cardiopulmonary arrest. The word *jimsonweed* is thought to be derived from Jamestown, Virginia, where British troops reportedly behaved bizarrely after consuming *Datura* in 1676. *Datura stramonium* has been reportedly used in Haitian zombification rituals.[37,53,86,87] Jimsonweed has been used to treat asthma, heralding the current use of ipratropium bromide.

Reports of abuse of *Datura* and *Brugmansia* as hallucinogens have increased in the United States and Europe.[37,87,167,199] Clusters of poisonings among adolescents are typical.[36,86,111,173,199] Frequently, a tea of the plant parts is brewed.[111] A mass poisoning occurred in Botswana after consumption of porridge made from sorghum flour contaminated with seeds of *D. stramonium*.[153] Symptoms may appear within minutes and may last for days.[160,199] Tachycardia, dry mouth, agitation, nausea, vomiting, incoherence, disorientation, auditory and visual hallucinations, mydriasis, decreased bowel sounds, slurred speech, hyperthermia, flushed skin, urinary retention, and hypertension have been reported.[37,111,160,163,173,199] Blurred vision and photophobia may be secondary to mydriasis. Isolated mydriasis and cycloplegia may be seen, including anisocoria, after topical contact to the eyes.[96,103,204] The associated pupillary dilation, or atropine mydriasis, is generally not reversed by application of

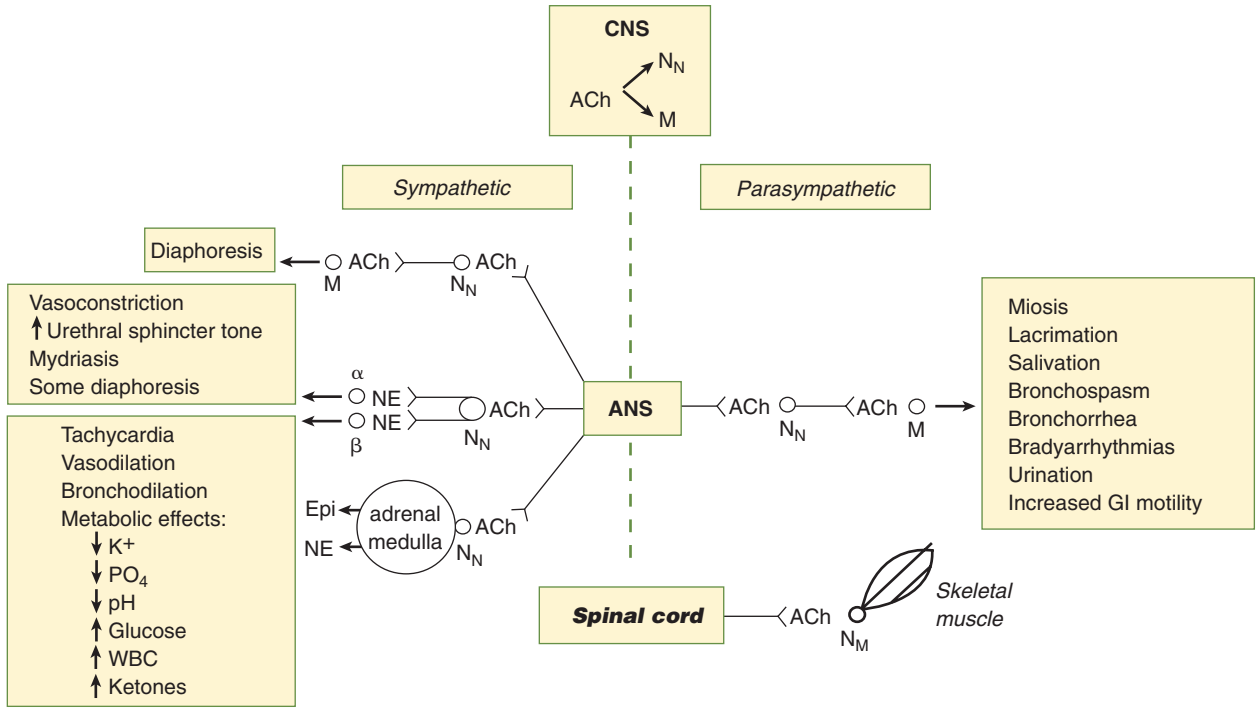


Figure 58-3. Nicotinic and muscarinic receptors of the central nervous system, autonomic nervous system, and peripheral skeletal muscles. Anticholinergic toxins (e.g., tropane alkaloids) antagonize muscarinic receptors, causing confusion, agitation, abnormal movements, hallucinations, and coma centrally, and mydriasis, anhidrosis, tachycardia, urinary retention, and ileus peripherally. Direct nicotinic agonists (e.g., arecoline, coniine, cytisine, lobeline, nicotine) stimulate nicotinic receptors; however, prolonged depolarization at the receptor causes eventual blockade of nicotinic receptors. ACh, acetylcholine; ANS, autonomic nervous system; CNS, central nervous system; Epi, epinephrine; M, muscarinic receptor; NE, norepinephrine; N_M, nicotinic receptor at skeletal muscle; N_N, nicotinic receptor in nervous system.

Box 58-1. Anticholinergic Syndrome

CENTRAL
CNS excitation
Agitation
Hallucinations
Lethargy
Coma
Respiratory depression
Mumbling speech
Muteness
Undressing behavior
Repetitive picking behavior

PERIPHERAL
Tachycardia
Mydriasis
Blurred vision
Inability to accommodate
Flushed skin
Hyperthermia
Absent bowel sounds
Urinary retention
Dry mucous membranes
Dry skin (particularly dry axillae)

1% pilocarpine.[96,103] With severe toxicity, seizures, flaccid paralysis, and coma may ensue. Focal neurologic signs and posturing, as well as death, have also been reported.[150,153,157,160,199] Death may occur as a result of behavioral changes, such as from trauma, drowning, or exposure with associated hyper- or hypothermia.[111] Autopsy has revealed edema of the brain and lungs with focal hemorrhages within the alveolar lumens, ischemic lesions and edema of the heart, and hyperemia of the sinusoidal tracts of the liver.[23,24]

Anticholinergic poisoning should be suspected when patients are observed to communicate with imaginary friends, have mumbling speech, and demonstrate repetitive picking behavior.[86,188] Symptoms generally begin 30 to 60 minutes after ingestion and continue for 24 to 48 hours, but they may begin within several minutes and persist for several days. Patients are generally amnestic of the events. Studies may show leukocytosis, mild transient elevation of liver enzymes, elevated creatine phosphokinase (CPK) levels and other evidence of rhabdomyolysis, and changes on an electrocardiogram (ECG) that are consistent with tachycardia and arrhythmias.[37,87]

Deadly Nightshade. Ingestion of *A. belladonna* is less common than *Datura* and *Brugmansia* ingestion. All parts of deadly nightshade contain tropane alkaloids, but the highest concentrations are in the ripe fruit and green leaves; each berry

A B

C D

Figure 58-4. Jimsonweed (*Datura* species) is a bush with trumpet-like flowers **(A)** and thorny seedpods that contain numerous small kidney-shaped seeds **(B).** A nickel is shown for size comparison. Angel's trumpet (*Brugmansia* species) is a tree **(C)** with trumpet-like flowers **(D).** (Courtesy Kimberlie Graeme, MD, and Phillip Saba, MD.)

may contain up to 2 mg of atropine. The berries may be mistaken for bilberries (hurtleberries). A family of eight had varying acute exposures to *A. belladonna* after eating both raw and cooked berries in a pie. The most severely poisoned patient had anticholinergic symptoms, with hypertonia, hyperthermia, respiratory failure, and coma requiring mechanical ventilation. Urine drug screens detected only atropine, not scopolamine, which is present in much smaller quantities.[177,188] A review of 49 children with acute deadly nightshade intoxication found the children most commonly demonstrated meaningless speech, tachycardia, mydriasis, and flushing, whereas lethargy and coma were seen in the more severely poisoned children.[29] A patient with seasonal chronic ingestion presented with recurrent tachycardia, mydriasis, inability to concentrate, visual hallucinations, delusions, inappropriate laughter, dizziness, and headache.[113]

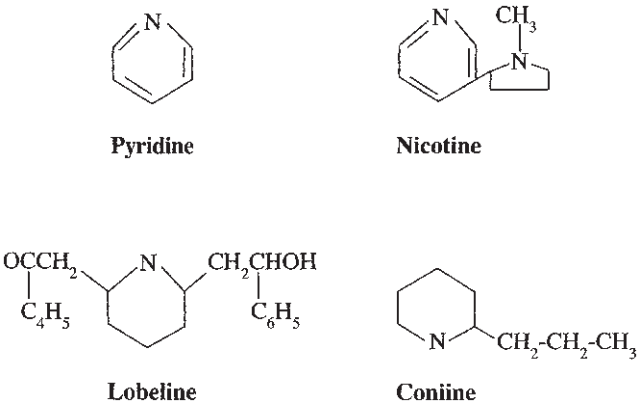
Treatment. Treatment of anticholinergic plant exposure consists primarily of decontamination and supportive care, including oral administration of activated charcoal, airway protection, intravenous (IV) fluids, and vasopressors for hypotension resistant to IV fluids. Hyperthermia should be assessed and treated if significant. Agitation can be treated with careful administration of benzodiazepines. Haloperidol and phenothiazines should not be used because these agents may enhance toxicity. Foley catheterization or nasogastric tube placement may be necessary if there is development of bladder distention or decreased gut motility, respectively.[79,111,113,157,177]

Some recommend treating severe central anticholinergic syndrome with carefully titrated physostigmine, which is derived from the calabar bean of *Physostigma venenosum*. This cholinesterase inhibitor blocks acetylcholine degradation; the resultant accumulation of acetylcholine overcomes the competitive inhibition of atropinic agents, such as those found in jimsonweed. Rapid, though perhaps transient, reversal of peripheral and central nervous system (CNS) effects can ensue. However, bradycardia, asystole, ventricular arrhythmias, hypotension, bronchospasm, bronchorrhea, and seizures have been reported after rapid IV administration of physostigmine, limiting its routine use. Persons with cardiac conduction abnormalities are particularly susceptible to the cardiac complications. Use of physostigmine generally does not shorten the hospital stay.*

Most cases of anticholinergic poisoning can be managed safely and effectively with supportive care alone.[187] Consultation with a toxicologist is recommended before using physostigmine. If physostigmine is used, the recipient should be on a cardiac monitor, with pulse oximetry, and a physician at the bedside should slowly administer graduated doses of the drug.[28] Risk-to-benefit ratio must be assessed before administration.[79,188] If the patient might have been exposed to tricyclic antidepressants, physostigmine should not be administered.[181,192]

Nicotinic Plants (Pyridine and Piperidine Alkaloids)

The pyridine–piperidine group contains the major alkaloids nicotine, coniine, lobeline, arecoline, piperine, and isopelletierine. Structures of some pyridine/piperidine alkaloids follow:



Nicotine alkaloids are found mainly in the Solanaceae family of plants. Other families containing nicotine alkaloids include Hippocastanaceae (horse chestnut) and Asclepiadaceae (milkweed).

Tobacco Plants. Tobacco contains both nicotine and related alkaloids with similar pharmacologic properties, such as the alkaloid anabasine, which is found in *Nicotiana glauca* (wild tree tobacco) (Fig. 58-5). *N. glauca* has been mistaken for *Amaranthus hybridus* (marog) and eaten with porridge in South Africa. Ingestion of *N. glauca* is generally fatal.[184,191] Anabasine, an isomer of nicotine that appears to be more toxic than nicotine, probably accounts for much of the toxicity of *N. glauca, Nicotiana debneyi,* and *Nicotiana rotundifolia.*[137,140,184,191] Other tobacco family members include *Nicotiana rustica, Nicotiana tabacum, Nicotiana trigonophylla* (desert tobacco) (Fig. 58-6), and *Nicotiana attenuata* (coyote tobacco). *N. tabacum* is the major source of commercial tobacco and contains between 0.5% and 9% nicotine. Nicotine alkaloids are rapidly absorbed from the oral and GI tracts as well as the respiratory tract and the skin.[140] The kidneys excrete nicotine fairly rapidly after biotransformation in the liver and lungs. The half-life is 1 to 2 hours. The lethal dose of ingested nicotine is not well established. Two to 5 mg may cause nausea, and 40 to 60 mg may be lethal in humans. One to two cigarettes, ingested and absorbed, could be lethal to a child.

Figure 58-5. Tree tobacco *(Nicotiana glauca).*

*References 36, 79, 111, 157, 163, 173, 181, 188, 192, 199.

Figure 58-6. *Nicotiana trigonophylla* (desert tobacco) and other *Nicotiana* species are characterized by narrow tubelike flowers. (Courtesy Kimberlie Graeme, MD.)

Box 58-2. Nicotinic Syndrome

EARLY STAGE
Hypertension
Tachycardia
Vomiting
Diarrhea
Muscle fasciculations
Convulsions

LATE STAGE
Hypotension
Bradyarrhythmias
Paralysis
Coma

Nicotinic Syndrome. Peripherally, acetylcholine is a neurotransmitter for autonomic and somatic motor fibers. It is stored in vesicles within the presynaptic neuron and is released by calcium-dependent exocytosis into the synapse, where it binds to receptors and is eventually degraded by acetylcholinesterase. Acetylcholine can bind to two receptor types, nicotinic and muscarinic. Nicotinic receptors are located on postganglionic autonomic neurons (N_N receptors) and at skeletal neuromuscular junctions (N_M receptors). Direct nicotinic agonists (e.g., arecoline, coniine, cytisine, lobeline, nicotine) prolong depolarization at these receptors and eventually cause blockade of nicotinic receptors. Clinical evidence of stimulation followed by blockade is apparent. Hypertension, tachycardia, vomiting, diarrhea, abdominal pain, salivation, bronchorrhea, muscle fasciculations, spasms, confusion, agitation, tremor, and convulsions (the stimulation) are followed by hypotension, bradyarrhythmias (occasionally asystole), paralysis, coma, and respiratory failure (the blockade). When death occurs, it is generally due to respiratory paralysis (Box 58-2, and see Figure 58-3).[35,47,137,140,184]

Green tobacco sickness is a mild form of nicotine poisoning seen in field workers with dermal exposure to leaves of green tobacco in wet environments. Nicotine, a water-soluble alkaloid, is absorbed dermally. The syndrome is characterized by weakness, salivation, nausea, vomiting, diarrhea, abdominal cramps, headache, dizziness, visual and hearing disturbances, respiratory depression, and occasionally fluctuations in blood pressure and heart rate. Neuromuscular blockade may result in death. A urinary nicotine metabolite, cotinine, may be helpful diagnostically.[12,137,191]

Lobeline, derived from the Indian tobacco plant *Lobelia inflata*, is a high-affinity nicotinic ligand. It can cause nicotine-like effects (e.g., hypertension, hypotension, bradycardia) but is generally less toxic than nicotine.[49]

Figure 58-7. Poison hemlock *(Conium maculatum).*

Poison Hemlock. *Conium maculatum* (poison hemlock) (Fig. 58-7), also known as spotted hemlock, California or Nebraska fern, stinkweed, fool's parsley, and carrot weed, is often mistaken for an edible plant, such as parsley, parsnips, or anise; however, it has a mousy odor and an unpleasant bitter taste, and it burns the mouth and throat. The stem has purplish to reddish brown spots and is hollow. Its long taproot is solid and parsnip-like. Although all plant parts are poisonous, the roots are especially toxic. Poisoning may also occur after eating birds that have consumed poison hemlock. Coniine and γ-coniceine, the principal alkaloids in *C. maculatum*, are pyridine derivatives similar to nicotine. The alkaloid is volatile and lost by drying or heating.[19,121,208]

C. maculatum was used in ancient times for capital punishment and murder. Socrates was quite likely the victim of poison hemlock, suffering nausea, paralysis, and finally death by respiratory arrest. The primary action of the toxic alkaloids is activation and then blockade of nicotinic acetylcholine receptors. Initially, stimulation causes sialorrhea, nausea, vomiting, diarrhea, abdominal cramping, tremor, and tachycardia, followed by dry mucosae, GI hypotonia, diminished cardiac contraction, and bradycardia, as with nicotinic syndrome. Muscles rapidly swell and stiffen, with multifocal necrosis of myocytes and associated muscle pain. Muscle fasciculations and flaccid paralysis may be followed by rhabdomyolysis and acute tubular necrosis with renal failure. Elevated CPK and liver function tests (LFTs) may be noted. Death is usually from respiratory failure. Autopsy may reveal congestion of the lungs and liver.[19,60,75,76,79,91,208]

Betel Nut. *Areca catechu* (areca palm) (Fig. 58-8) produces betel nut, a common masticatory drug in the Far East, Asia, India, and the South Pacific. It is also purchased elsewhere via the Internet.

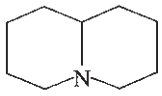
A

B

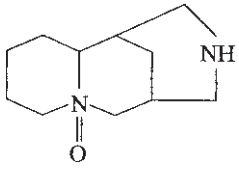
Figure 58-8. **A** and **B,** Betel nut *(Areca catechu).* (Courtesy of Daniel Brooks, MD, and Tammy Tyree, NP.)

An estimated 10% to 25% of the world's population chews "betel quid." It is generally chewed with slaked lime paste (calcium hydroxide) wrapped in leaves of betel pepper *(Piper betel),* known as "quid," "punsupari," or "pan masala." Occasionally, tobacco is added. Quid is sucked in the lateral gingival pocket. Commercially, areca nut is marketed in the form of sweetened areca nut, known as "supari." *A. catechu* contains arecoline and guvacoline, which are hydrolyzed to arecaidine and guvacine, respectively, which are strong inhibitors of gamma-aminobutyric acid (GABA) uptake. These arecal alkaloids also are nicotinic and muscarinic. Betel pepper leaves contain betel oil, which contains psychoactive phenols and cadinene. These possess cocainelike properties. Clinical effects resemble nicotinic syndrome and cholinergic toxicity, including CNS effects (dizziness, euphoria, subjective arousal, altered mental status, hallucinations, psychosis, convulsions), cardiac effects (tachycardia, hypertension, palpitations, arrhythmias, bradycardia, hypotension, chest discomfort, and acute myocardial infarction in susceptible individuals), pulmonary effects (bronchospasm, tachypnea, dyspnea), GI effects (salivation, vomiting, diarrhea), urogenic effects (urinary incontinence), and musculoskeletal effects (weakness and paralysis). Use with calcium salts may result in the development of hypercalcemia, hypokalemia, and metabolic acidosis with renal potassium wasting and renal insufficiency. Betel nut use is also associated with flushing, diaphoresis, warm sensations, red- or orange-stained oral mucosa and saliva, and teeth stained dark brown or black. Oral lesions may also occur with chronic use.[23,41,54,108,132,144,180,194]

Quinolizidine Alkaloids. Cytisine is a quinolizidine alkaloid but is often classified with the pyridine and piperidine alkaloids. Cytisine stimulates nicotinic ganglions. Common toxic plants in this group include golden chain tree *(Laburnum anagyroides),* Kentucky coffee tree *(Gymnocladus dioica),* necklace pod sophora *(Sophora tomentosa),* and mescal bean bush *(Sophora secundiflora)* (Fig. 58-9) (see Hallucinogenic Plants, later). Structures follow:



Quinolizidine

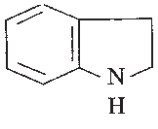


Cytisine

Treatment. Treatment of nicotinic syndromes consists of supportive care with particular attention to airway protection and ventilation. Administration of activated charcoal is generally recommended. Benzodiazepines and barbiturates are given for seizures. Adequate urine output is maintained, with consideration of urine alkalinization. Treating initial excessive adrenergic stimulation with phentolamine is ill advised because this complicates the nicotinic blockade that follows. Symptomatic bradycardia can be treated with atropine, and hypotension with IV fluids and inotropic agents (e.g., dopamine) if needed. With liver or renal failure, supportive treatment may be required.

Hallucinogenic Plants (Indoles, Phenylalkylamines)

About 90 of the 800,000 known plant species are hallucinogenic. Numerous rituals and religious ceremonies have used these plants. Many psychoactive plants are indole derivatives, which are among the most potent psychoactive compounds in nature and have the following structure:



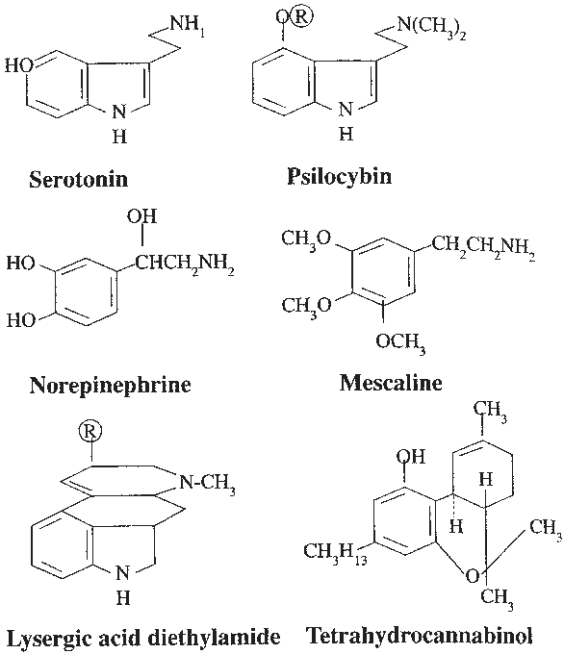
Indole nucleus

Chemical relationships exist among serotonin, psilocybin (*Psilocybe* species), and D-lysergic acid diethylamide (LSD). LSD is synthesized from ergonovine (or ergometrine), derived from *Claviceps purpurea.* Striking structural similarities exist between the most potent psychoactive plant compounds and biochemically important neurotransmitters, as follows:

A B

C

Figure 58-9. Mescal bean bush *(Sophora secundiflora)* **(A)** is characterized by bean pods that contain burnt-red seeds **(B)** and by purple blooms **(C).** (Courtesy Phillip Saba, MD, and Kimberlie Graeme, MD.)



Ergot. Commercial grains are now inspected for the presence of ergot, and inadvertent poisoning is rare. An outbreak of ergotism occurred in Ethiopia in 1977. In the Middle Ages, epidemics of ergotism resulted from repeated ingestion of ergot-contaminated breads. The ergot alkaloids ergonovine and ergotamine are indole derivatives found in the fungus *C. purpurea,* which infects rye and other grains. The ergot alkaloids are serotonin agonists.

Ergotism has two forms, gangrenous and convulsive. Both begin similarly with a vague illness that may include GI symptoms, progressing to a sensation of ants crawling on the skin, primarily of the legs. Hallucinations can occur with both forms. Gangrenous ergotism, a vasospastic disease, was the cause of the 9th-century epidemic disease known as St. Anthony's fire, which consisted of burning limb pain and eventual loss of limb sensation, followed by gangrene and autoamputation of charcoal-black limbs. People migrated to St. Anthony's Order, a society of monks, and began to heal, largely because they received a diet free of ergot-contaminated grains. Convulsive ergotism is characterized by painful flexion of the fingers and wrists, flexion or extension of the ankles, double vision, altered mental state, hallucinations, and diaphoresis. With progression, the body becomes bent in spasm, appearing to roll up into a

Figure 58-10. Morning glory *(Ipomoea violacea)*.

ball, or it becomes rigid in extension; this may be followed by seizures.[32,33,37,62,91,205]

Morning Glory. The active component of the naturally occurring hallucinogen found in the seeds of morning glory *(Ipomoea violacea)* (Fig. 58-10) is ergine, or (+)-lysergic acid amide, an indole derivative.[86] About 300 seeds, or enough to fill a cupped hand, are equivalent to 200 to 300 mg of LSD, with similar systemic and hallucinatory effects. Ingestion of Hawaiian baby woodrose seeds *(Argyrlia nervosa)* has similar effects.[80]

Nutmeg. *Myristica fragrans* (Fig. 58-11) is used to make the spices nutmeg and mace. Mature rinds of the fruit split, revealing a bright-red, fringed, fleshy coating on the outside of its seed. The coating contains mace, and the seed contains nutmeg. Nutmeg contains myristicin, which has an indolelike structure and is metabolized to amphetamine-like compounds. Other alkylbenzene derivatives, such as safrole and elemicin, are also found in nutmeg. Nutmeg has been abused for its alleged hallucinogenic effects. A person who ingested one grated nutmeg (7 g) had weakness, loss of coordination, vertigo, fainting, nausea, and paresthesias but no hallucinations.[185,206]

Cannabis. Cannabis preparations are largely derived from the female *Cannabis sativa* plant. The primary psychoactive component is δ-9-tetrahydrocannabinol (THC), which is most concentrated in the flowering tops. Marijuana generally contains 0.5% to 5% THC; however, Sinsemilla and Netherwood varieties may contain up to 20% THC, and hashish and hashish oils have higher concentrations. Cannabinoids can be smoked or ingested. A typical marijuana cigarette contains 0.5 to 1.0 g of cannabis, and the THC delivered varies from 20% to 70%, with a bioavailability of 5% to 24%. As little as 2 mg of available THC produces effects in occasional users.[92]

Cannabinoids bind to specific cannabinoid receptors in areas of the brain involved with cognition, memory reward, pain perception, and motor coordination. Cannabinoids act as neuromodulators in the release and action of neurotransmitters (e.g., acetylcholine, glutamate).[57,92] Endogenous ligands for these receptors are endocannabinoids. The first endocannabinoid identified was anandamide, after the Sanskrit word *ananda,* which means "bliss."

A

B

Figure 58-11. Nutmeg *(Myristica fragrans)*. (Courtesy of Daniel Brooks, MD, and Tammy Tyree, NP.)

Desirable effects include mild mood-altering qualities, euphoria, alteration in perceptions, time distortion, and intensification of ordinary sensory experiences (e.g., gustatory, visual, and auditory sensations). Adverse effects include impairment of short-term memory and attention, impairment of motor skills and reaction times, and anxiety. Psychotic symptoms have been reported in persons vulnerable to psychosis. Clinically, tachycardia may occur within minutes of THC exposure and may last a few hours. Minor changes in blood pressure may also occur.[92]

Peyote. The hallucinogenic peyote cactus *Lophophora williamsii* (Fig. 58-12) contains alkaloids that are phenylethylamines or isoquinolines rather than indoles. Mescaline (3,4,5-trimethoxy-β-phenylethylamine), the primary psychoactive component of peyote, is structurally similar to the neurotransmitters norepinephrine and epinephrine and to hallucinogenic amphetamines. Pharmacologically, however, mescaline is similar to hallucinogenic indoles. Mescaline may affect the action of norepinephrine and serotonin, evidenced clinically by

Figure 58-12. Peyote *(Lophophora williamsii)*.

Figure 58-13. Khat *(Catha edulis)*. (Courtesy of Daniel Brooks, MD, and Tammy Tyree, NP.)

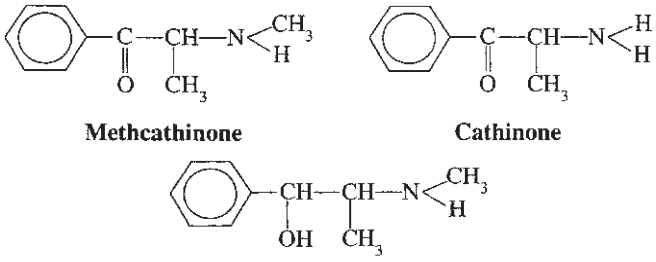
sympathomimetic effects, followed by marked visual hallucinations. Mescaline produces a slight rise in blood pressure and heart rate, tachypnea, hyperreflexia, mydriasis, ataxia, perspiration, flushing, salivation, and urination. No deaths have been reported from peyote poisoning. Type B botulism was associated with consumption of a ceremonial tea made from peyote that was stored in a jar by members of the Native American Church. Affected members had bilaterally symmetrical, flaccid weakness in all extremities, dysphagia, nasal speech, and diplopia.[8,99]

Tabernanthe iboga. *Tabernanthe iboga* contains indole alkaloids, including ibogaine. The root of this plant is used in West Africa to communicate with ancestors, reportedly producing "visions" and "waking dreams." It produces altered states of consciousness, delusions, hallucinations, mydriasis, tachycardia, tremor, and ataxia. Convulsions and lethal respiratory arrest have also been reported.[31]

Mescal Bean Bush. The mescal bean bush or Texas mountain laurel *(Sophora secundiflora)* of the pea family (Fabaceae) produces hallucinogenic dark-red beans (mescal beans; see Figure 58-9). The beans contain the toxic alkaloid cytisine, which causes nausea, numbing sensations, hallucinations, unconsciousness, convulsions, and death through respiratory failure. The beans may be boiled in water and the mixture consumed, producing a delirium or "visionary trance." The origin of mescalism in modern peyote religion is debated. Mescal beans are worn during some peyote ceremonies.[8]

Khat. The evergreen khat tree, *Catha edulis*, grows in East Africa and Arabia (Fig. 58-13). Khat is also known as chat, qat, eschat, mirra, qaad, and jaad. As early as 1237, khat was advocated in Arabic medical literature as a mood-elevating and hunger-suppressing agent. Khat leaves and bark continue to be chewed, with the juice of the masticated plant being swallowed, for stimulatory effects. Khat contains cathinone, cathine (norpseudoephedrine), and norephedrine. Cathinone (α-aminopropiophenone), a phenylalkylamine, is the major psychoactive constituent. Structurally, cathinone is similar to amphetamines, such as methcathinone synthesized from ephedrine. Like amphetamines, cathinone is an indirect sympathomimetic and induces the release of dopamine, serotonin, and norepinephrine.*

The structures of methcathinone, cathinone, and ephedrine follow:



Cathinone in fresh plant material must be extracted by masticating laboriously; this results in gradual absorption. As the leaf wilts, cathinone content decreases and the leaf loses its potency as a psychostimulant. Because only fresh leaves produce the desired stimulatory effects, much khat use in the past was limited to countries where khat was produced (e.g., North Yemen, Ethiopia, Kenya); however, khat is now air-freighted to Europe and the United States. Khat is transported damp, rolled in a banana leaf bundle called a marduff, which sells for approximately $50.[2,4,59,83,149,158]

Desirable effects of khat include increased energy and alertness, feelings of increased endurance and self-esteem, enhanced imaginative ability, higher capacity to associate ideas, and euphoria. Cathinone has both positive chronotropic and inotropic effects. Tachycardia, increased blood pressure, tachypnea, and mydriasis are seen. Adverse effects include anorexia, hypomania, insomnia, delusions, paranoid psychosis,

*References 2, 4, 6, 31, 34, 101, 146, 149, 158, 201.

aggression, depression, anxiety, hyperthermia, and endocrine disturbances. Khat is known to be habit forming and has been classified as a "substance of abuse" by the World Health Organization. A person who developed abdominal pain was found to have immature *Fasciola hepatica* (fluke infestation of the biliary tree), thought to be secondary to use of contaminated khat.[2,3,6,59,101,115,146] Khat use has also been associated with acute myocardial infarction and leukoencephalopathy.[4,141]

Anticholinergic Plants

Henbane *(Hyoscyamus niger),* jimsonweed *(Datura stramonium),* and mandrake *(Mandragora officinarum)* contain tropane alkaloids and can produce hallucinations, as discussed earlier.

Treatment. Treatment of patients exposed to hallucinogenic plants is generally supportive. First-line treatment for agitation is generally benzodiazepines.

Sedating Plants (Isoquinoline Alkaloids)

Poppy. *Papaver somniferum* flowers are large and white with purple stains at the base of each petal. They yield opium, a complex of more than 20 alkaloids, including morphine, codeine, and papaverine. Morphine was the first plant alkaloid isolated, by pharmacist Friederich Wilhelm Adam Serturner in 1806.[90] Seeds of *P. somniferum* are used in various foods and beverages, including bagels, muffins, pastries, curry sauce, rice, and teas. Ingestion of poppy seeds can result in detectable levels of morphine and codeine by urine drug screen testing; however, significant opiate-induced impairment is not apparent. Poppy dependence has been described.[116,161,203]

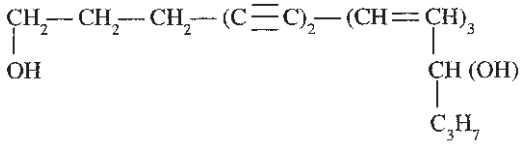
Neuromuscular Blocking Plants (Indole Alkaloids)

Yellow Jasmine. *Gelsemium sempervirens* (Carolina or yellow jessamine) is a woody perennial evergreen vine with fragrant yellow flowers. It contains multiple indole alkaloids, including gelsemine, gelseminine, and gelsemoidin. Gelsemine binds to acetylcholine receptors at the neuromuscular junction (peripheral nicotinic acetylcholine receptors) and, to a lesser extent, at muscarinic receptors. A toddler who ate the blossoms of *G. sempervirens* experienced neuromuscular blockade with ataxia, dysarthria, facial and extremity weakness, bilateral ptosis, and transient coma. The child recovered without sequelae.[21]

Convulsant Plants (Indoles, Resins)

Strychnine. Strychnine, an indole found in seeds of the tree *Strychnos nux-vomica,* is a powerful CNS stimulant. Poisoning may also occur after ingestion of rodent poisons containing strychnine, with the use of illicit drugs contaminated with strychnine, or with the use of herbal remedies contaminated with strychnine. Strychnine is a selective, competitive antagonist of glycine, a major inhibitory neurotransmitter, at its postsynaptic receptors in the spinal cord and brainstem. Poisoning produces an excitatory state, with hyperreflexia, hypersensitivity to stimuli, migratory rippling movements of the muscles, twitching, severe muscle spasm, rigidity, and spinal convulsions (generally, flexor spasm of the upper limbs, extensor spasm of the lower limbs, opisthotonic posturing, and spasms of the jaw muscles, all without loss of consciousness or postictal states). In between the spasms, which last from 30 seconds to 2 minutes, the muscles become completely relaxed. Respiratory and secondary cardiac failure may ensue during severe convulsions. Treatment consists of decontamination and support, including activated charcoal, benzodiazepines, and barbiturates. Chemical paralysis with a nondepolarizing agent, endotracheal intubation, and mechanical ventilation may be required for severely poisoned patients. Hyperthermia, rhabdomyolysis, renal failure, and acidosis may occur secondary to the convulsions; these complications often require treatment. Occasionally, death ensues despite aggressive management.[17,39,74,133,151,155,178,179,215]

Water Hemlock. Water hemlock *(Cicuta maculata)* and chinaberry *(Melia azedarach)* are two of the most toxic resin-containing plants. Chinaberry produces primarily GI symptoms (see Chinaberry Trees, later). The resin of *C. maculata,* an unsaturated aliphatic alcohol called cicutoxin, possesses convulsive properties and has the following structure:



Cicutoxin

Nine subspecies of *Cicuta* of the Umbelliferae family are poisonous (Fig. 58-14). *Cicuta virosa* is common European water hemlock, whereas *C. maculata* and *Cicuta douglasii* are found in North America. *Oenanthe crocata,* or hemlock water dropwort, also contains cicutoxin and is found in Europe and North America. Common names for *Cicuta* species include cowbane, five-finger root, snake weed, snake root, wild carrot, dead man's fingers, death-of-man, poison parsnip, wild parsnip, beaver poison, children's bane, muskrat weed, spotted hemlock, spotted cowbane, musquash root, false parsley, fever root, mock-eel root, wild dill, spotted parsley, and carotte à Moreau. Roots have a parsnip-like or carrot-like odor, and *Cicuta* species are often mistaken for edible plants, such as water parsnip. Mature roots have air-filled chambers and are found on the ends of hollow stems. All parts of the plant are toxic, but the roots contain the highest concentration of cicutoxin. Ingestion of as little as 2 to 3 cm (about 1 inch) of the root may be fatal to an adult. The plant is most toxic in the spring.[79,104,121]

Early symptoms include muscarinic effects and involve primarily the GI tract: abdominal pain, vomiting, and diarrhea. However, profuse perspiration, salivation, and respiratory distress may be seen. Nicotinic effects are less prominent (see Figure 58-3). Tachycardia and hypertension or bradycardia and hypotension may be seen. Epileptiform seizure activity or spastic and tonic movements, including opisthotonus without electroencephalographic seizure activity, may occur with severe poisoning. Pupils may be any size. Rhabdomyolysis and renal failure have been reported. Deaths may be associated with persistent seizures, cerebral edema, ventricular fibrillation, pulmonary edema, cardiopulmonary arrest, and disseminated intravascular coagulation. Laboratory abnormalities include metabolic acidosis and elevated CPK and LFTs.[79,104,121]

A B

Figure 58-14. *Cicuta* species have characteristic flowering tops, typical of the Umbelliferae family **(A),** and roots with air-filled chambers at the end of hollow stems **(B).** (Courtesy Steven Curry, MD.)

Box 58-3. Cardiovascular Toxins Found in Plants

ALKALOIDS
Purine (e.g., caffeine)
Steroid (e.g., aconite, veratrum alkaloids)
Taxane (e.g., taxines)

GLYCOSIDES
Cardiac (e.g., lanatosides, oleandrin, thevetin)

RESINS
Grayanotoxins (e.g., andromedotoxin)

Treatment is symptomatic and supportive, with particular attention to the airway. Activated charcoal is recommended, with benzodiazepine and barbiturate administration for seizure control. Adequate urine output maintenance and alkalinization of urine are initiated to treat rhabdomyolysis.[79,104]

Purine Alkaloids. The purine alkaloids caffeine and theobromine are found in various plants, but the derivative beverages and stimulants represent a toxic risk. Theobromine is especially toxic to dogs. Even cacao bean shells contain potentially toxic quantities of theobromine, and dogs that ingest large quantities of bean shells may suffer convulsions and death.

CARDIOVASCULAR SYSTEM TOXINS

Cardiovascular toxins found in plants are listed in Box 58-3.

Cardiotoxins That Inhibit Na^+,K^+ ATPase (Cardiac Glycosides)

More than 200 naturally occurring cardiac glycosides have been identified. Plant cardiac glycosides are composed of a steroid backbone, an attached five-membered unsaturated lactone ring (six-membered for *Helleborus*), and either a carbohydrate or sugar moiety in glycosidic linkage. The toxic aglycones are released by acid and enzymatic hydrolysis. The attached sugar moiety has no inherent cardiac action but may enhance solubility, absorption, and toxicity of the aglycone moiety. Cardiac glycosides bind to the membrane-bound enzyme Na^+,K^+ ATPase, increasing intracellular Na^+ and Ca^{2+} levels and automaticity (Fig. 58-15). Cardiac glycosides are found in *Digitalis purpurea* (foxglove) (Fig. 58-16), *Digitalis lanata*, *Nerium oleander* (common oleander) (Fig. 58-17), *Thevetia peruviana* (yellow oleander) (Fig. 58-18), *Convallaria majalis* (lily of the

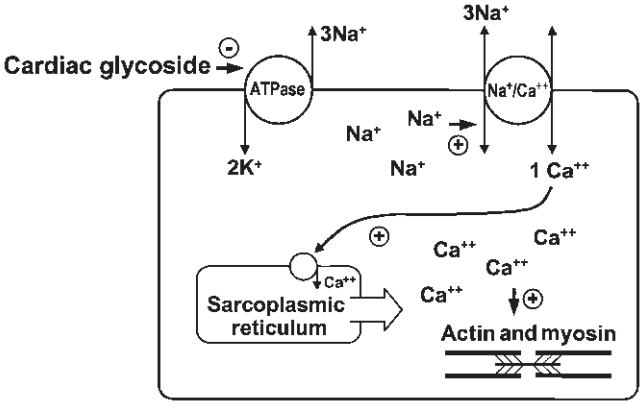


Figure 58-15. Cardiac glycosides bind to and inhibit the membrane-bound enzyme Na^+,K^+ ATPase in cardiac myocytes (shown), baroreceptor cells, and skeletal muscle cells. Inhibition of Na^+,K^+ ATPase in cardiac myocytes results in accumulation of intracellular Na^+, which results in accumulation of Ca^{2+} within myocytes via Na^+,Ca^{2+} exchangers. The resultant increase in intracellular Ca^{2+} stimulates further release of Ca^{2+} from the sarcoplasmic reticulum. The increased intracellular Ca^{2+} interacts with troponin C of the actin–myosin complex to cause increased contractions, seen as increased automaticity (e.g., premature ventricular contractions on electrocardiograms). Inhibition of membrane-bound enzyme Na^+,K^+ ATPase in baroreceptor cells and skeletal muscle cells contributes to increased vagal tone and hyperkalemia, respectively.

Figure 58-16. *Digitalis purpurea* (foxglove). (Courtesy Kimberlie Graeme, MD.)

A

B

Figure 58-17. *Nerium oleander* (common oleander) plants have white or pink flowers **(A)** and long, narrow seedpods **(B).** (Courtesy Kimberlie Graeme, MD.)

valley) (Fig. 58-19), *Urginea maritima* (squill or sea onion) (Fig. 58-20), *Urginea indica, Strophanthus gratus* (ouabain), *Asclepias* species (balloon cotton, red-headed cotton-bush milkweeds), *Calotropis procera* (king's crown), *Carissa spectabilis* (wintersweet), *Carissa acokanthera* (bushman's poison), *Cerebera manghas* (sea mango), *Plumeria rubra* (frangipani), *Cryptostegia grandifolia* (rubber vine), *Euonymus europaeus* (spindle tree), *Cheiranthus, Erysimum* (wallflower), and *Helleborus niger* (henbane).*

Foxglove. *Digitalis purpurea* grows wild in parts of the United States and is cultivated as a garden ornamental plant. Withering reported the medical use of extracts of *D. purpurea* based on a recipe for treating "dropsy."[102,148] The leaves have been consumed in a risotto when they were mistaken for borage leaves, and in a salad when mistaken for dandelion leaves.[30,145] Toxicity has also been reported from consumption of contaminated field water with *Digitalis* plants growing nearby.[145] *Digitalis lanata* was mistakenly substituted for plantain in herbal products, with resultant human cardiotoxicity.[186] *D. lanata* contains lanatosides A, B, and C, which yield digoxin and digitoxin. *D. purpurea* contains only digitoxin, not digoxin. Intentional overdoses also occur.[125,145]

*References 63, 66, 69, 79, 89, 91, 102, 127, 145, 202.

A B

Figure 58-18. *Thevetia peruviana* (yellow oleander) has yellow flowers **(A)** with smooth seedpods, known as "lucky nuts," composed of green flesh surrounding a hard brown seed **(B).** (Courtesy Kimberlie Graeme, MD.)

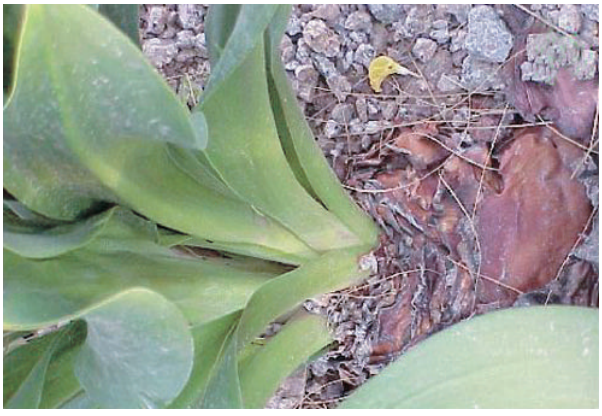
Figure 58-19. Lilly of the valley *(Convallaria majalis).* (Courtesy Donald Kunkel, MD.)

Oleander. All parts of *Nerium oleander* and *Thevetia peruviana* are toxic, but the seeds contain more glycoside than other parts of the plant.[52,79,127,129] Yellow oleander *(T. peruviana),* a native plant of tropical America, grows abundantly in the United States. Ingestion of a couple of seeds of yellow oleander, known as "lucky nuts," can result in death.[64] Yellow oleander contains the cardiac glycosides thevetin A and B, thevetoxin, neriifolin, peruvoside, ruvoside, and others. It is a popular suicidal agent in Sri Lanka. Of patients admitted with *T. peruviana* poisoning in Sri Lanka, 43% had arrhythmias, many requiring temporary pacing; 6% died shortly after admission.[63,64] Common oleander *(N. oleander),* a native plant of the Mediterranean, grows abundantly in the United States. Common oleander contains the principal cardiac glycosides oleandrin and neriine, as well as folinerin and digitoxigenin.[127,214] Severe toxicity has been reported after the consumption of unprocessed common oleander leaves and prepared teas.[85,147] Chronic toxicity has occurred with criminal intentional poisoning.[129] Topical application of homemade *N. oleander* solutions to psoriasis wounds has resulted in toxicity as well.[214]

Squill. *Urginea maritima* was used by ancient Egyptians and Romans as a diuretic, heart tonic, expectorant, emetic, and rat poison. Squill contains several cardiac glycosides, including scillaren A, glucoscillaren A, scillaridin A, and scilliroside.[66,202,211]

Clinical Presentation. The onset and duration of action is well known for certain glycoside preparations, such as digoxin, digitoxin, and ouabain, but it may vary considerably after plant ingestions. For example, oleandrin exhibits protracted binding times to cardiac myocardial tissues.[127] Generally, cardiac glycoside toxicity produces nausea, vomiting, visual changes (appearance of yellow and green colors, "halos," geometric shapes; scintillations; photophobia), mental status changes (disorientation, psychosis, lethargy, stupor, dysarthria, weakness, dizziness, seizures), cardiac disturbances (palpitations, bradycardia, atrioventricular block, sinus node block, extrasystoles, ventricular arrhythmias, syncope), hyperkalemia, and death. When death occurs, it is generally caused by cardiotoxicity. ECGs may reveal nonspecific ST-segment and T-wave changes, similar

A



B

Figure 58-20. *Urginea* species (squill or sea onion) have broad leaves **(A)** and a red, underground bulb (some varieties have a white bulb) **(B).** (Courtesy Kimberlie Graeme, MD.)

to digoxin-induced changes.[85,129,145,186,214] Serum digoxin levels (polyclonal digoxin immunoassays) may be elevated after exposure to plants containing cardiac glycosides, because antibody-based digoxin assays cross-react nonquantitatively with various cardiac glycosides. Digoxin immunoassays predict only the presence of the glycoside, not the degree of toxicity.[64,89,127,202] Laboratory studies may show hyperkalemia, which correlates with severity of toxicity.[64]

Treatment. Cardiac glycoside toxicity from plant ingestions have been successfully treated with activated charcoal (to limit enterohepatic circulation), cardiac pacing, antiarrhythmic agents, and digoxin-specific Fab fragments (e.g., Digibind). Maintenance of fluid and electrolyte balance is important. Theoretically, administration of exogenous calcium could be harmful because of high intracellular calcium concentrations that might cause the heart to perform one final contraction without relaxation, a state termed *stone heart*. In theory, digoxin-specific Fab antibody fragments could couple to circulating cardiac glycosides and limit binding of cardiac glycosides to Na^+,K^+ ATPase. The administration of digoxin-specific Fab to reverse cardiotoxicity from plant glycosides remains controversial; although it has reportedly been successful in several human cases and in animal studies.[44,64,65,85,120,127,145,168,186]

Figure 58-21. Monkshood (*Aconitum* species).

Cardiotoxins That Open Sodium Channels (Steroid Alkaloids, Resins)

Steroid alkaloids form principal toxic components of several common cardiotoxic plants: monkshood (*Aconitum* species), American hellebore *(Veratrum viride),* and death camas (*Zigadenus* species). Aconite is found in *Aconitum* species, and veratrum alkaloids are found in *V. viride* (false or American hellebore), *Veratrum californicum* (skunk cabbage), *Veratrum album* (white hellebore), *Veratrum japonicum, Zigadenus paniculatus* (death camas), *Zigadenus venenosus, Zigadenus nuttallii,* and *Zigadenus gramineus.*

Aconite. Recently, aconite poisonings have become more common, following the use of *Aconitum* species in herbal products, including *Aconitum napellus* (monkshood) (Fig. 58-21), *Aconitum carmichaeli* (chuanwa), *Aconitum brachypodium, Aconitum vulparia,* and *Aconitum kusnezoffii* (caowu). *Delphinium* species (larkspur) demonstrate similar toxicity. All parts of these plants are toxic. *Aconitum* species contain diterpenoid-ester alkaloids, particularly aconitine, but also mesaconitine and hypaconitine, which are neurotoxins and cardiotoxins. Boiling these plants in water hydrolyzes aconite alkaloids to less toxic benzoylaconine and aconine derivatives.[7,38,79]

Aconite alkaloids activate sodium channels and affect excitable membranes of cardiac, neural, and muscle tissues.[7,38] Enhancement of the transmembrane inward sodium current during the plateau phase of the action potential prolongs repolarization in cardiac myocytes and induces delayed and early afterdepolarizations. Delayed afterdepolarizations result in increased automaticity, such as premature ventricular beats. Early afterdepolarizations produce lengthening of the QT interval.

Symptoms begin within 3 minutes to 6 hours of ingestion and may persist for several days. Nonspecific GI symptoms (nausea, vomiting), salivation, diaphoresis, dyspnea, and restlessness are accompanied by cardiotoxicity and CNS toxicity. Cardiac effects are clinically similar to cardiac glycoside toxicity, with enhanced vagal tone, bradycardia, heart block, ectopic beats, supraventricular tachycardia, bundle branch block, junctional escape rhythms, ventricular tachycardia, bifascicular ventricu-

lar tachycardia, polymorphic ventricular tachycardia, torsades de pointes, ventricular fibrillation, asystole, and hypotension. Occasionally, death ensues, generally from ventricular arrhythmias, such as refractory ventricular fibrillation.[38,79]

Hypaconitine affects sodium channels of the nerve membrane more selectively than does aconitine and is more potent than aconitine and mesaconitine in producing neuromuscular block. These neurotoxins produce conduction block and paralysis through voltage-sensitive sodium channels in axons. Prolonged depolarization prevents repolarization of excitable membranes. Visual impairment, dizziness, vertigo, ataxia, paresthesias (e.g., numbness of mouth and extremities), hyporeflexia, weakness, paralysis, coma, and convulsions may occur. Elevated CPK levels, without evidence of myocardial infarction, may occur. Hypokalemia, metabolic or respiratory acidosis, respiratory alkalosis, and renal and hepatic impairment may be noted.

Veratrum Alkaloids. *Veratrum* and *Zigadenus* species belong to the lily family. *V. viride* is known as false hellebore. The whole plant is toxic, with the rhizomes being more toxic than the tops. *Veratrum* species may be found in some sneezing powders and have been accidentally ingested when mistaken for *Gentiana lutea* or *Phytolacca americana* (pokeweed). *Zigadenus* species, also known as foothill camas and death camas, have been accidentally ingested when mistaken for nontoxic wild onions and sego lilies. Veratrum alkaloids include protoveratrine, veratridine, cevadine, jervine, and zygacine, which are found in the entire plant, although the bulb and flower usually cause toxicity. These alkaloids are extremely toxic and rapidly increase the permeability of voltage-sensitive sodium channels in excitable cell membranes. This results in an initial depolarization and the subsequent loss of membrane potential. Stimulation of vagal fibers may result in bradycardias and hypotension.[73,79,82,105,162,164]

Symptoms generally occur within 30 minutes to 3 hours and resolve within 24 to 48 hours. Toxicity is characterized by diaphoresis, nausea, vomiting, diarrhea, abdominal pain, hypotension, bradycardia, and shock. Syncope, respiratory depression, scotomata, paresthesias, fasciculations, muscle spasticity, hyperreflexia, vertigo, ataxia, dizziness, coma, seizures, and death may also occur. ECG findings may mimic cardiac glycoside toxicity, including repolarization abnormalities (e.g., abnormal T waves and ST segments), sinoatrial and atrioventricular blocks, and prolonged QT intervals.[73,162]

Treatment. Treatment of cardiotoxic steroid alkaloid poisoning is supportive and includes atropine, crystalloid fluids, and vasopressors. Victims may require mechanical ventilation and cardiopulmonary resuscitation. Magnesium may suppress early afterdepolarizations and polymorphic ventricular tachycardia. Lidocaine, amiodarone, and hemoperfusion have also been utilized to treat ventricular arrhythmias secondary to aconite poisoning in various case reports. Cardiopulmonary bypass has been utilized.[7,38,61,79,82,131,152,162,217]

Grayanotoxins. Resins called grayanotoxins are found in rhododendrons, mountain laurels, and azaleas. They produce toxicity similar to that seen with the steroid alkaloids veratrum and aconite, by binding to myocardial sodium channels and increasing their permeability. Poisoning may result from the ingestion of leaves, flowers, or nectar; however, grayanotoxin-contaminated honey is the most common source.[126,209,216] The nectar of *Rhododendron* species contains the grayanotoxin *o*-acetylandromedol, formerly andromedotoxin, which is transferred to humans through consumption of honey produced by bees utilizing the plant's nectar. Poisonings still occur in the Turkish population of central Europe. Symptoms include salivation, emesis, hypotension, bradycardia, arrhythmias, hypotension, chest pain, dizziness, circumoral and extremity paresthesias, incoordination, and muscular weakness.[209]

Other Cardiotoxins

Taxine Alkaloids. *Taxus* species include *Taxus baccata* (English yew) and *Taxus brevifolia* (Western yew). The toxic alkaloids include taxine A and B. All parts of *Taxus* plants are toxic except the pulp, or aril, which contains very little taxine. The seeds, which are surrounded by the aril, may be toxic if chewed sufficiently to allow absorption of taxine. Taxine B, believed to be the primary cardiotoxic alkaloid, inhibits both calcium and sodium transport across cell membranes.[123,128,207,213]

Most *Taxus* exposures occur in children, are accidental, and are not associated with significant toxicity. However, serious toxicity and death have occurred after intentional ingestions of *Taxus* species. Although GI toxicity is most common, dizziness, pupil dilation, muscle weakness, and convulsions have also been reported. Severe toxicity is characterized by bradycardias, heart blocks, ventricular tachycardia, ventricular fibrillation, widened QRS complexes, and cardiac arrest.[123,138,143,172,207,213] Autopsy has revealed congestion of the organs with pronounced cerebral and pulmonary edema.[212]

Studies with frog hearts indicate that taxines depress atrioventricular conduction via direct action on the myocardium. Further studies indicate that taxines cause an increase in cytoplasmic calcium by antagonizing both calcium and sodium ion channel conductance.[213] Reported yew-induced arrhythmias in humans are consistent with arrhythmias induced by blockade of voltage-gated sodium channels, and these are described as bradycardic rhythms with wide QRS complexes, despite normal electrolytes. For example, a patient who ingested yew presented with severe cardiotoxicity and a wide QRS complex, which appeared to respond favorably to administration of hypertonic sodium bicarbonate.[138,172] However, controlled studies of *Taxus*-poisoned swine did not show narrowing of the widened QRS complex after sodium bicarbonate administration.[172] Lidocaine administration and cardiac pacing have also been reportedly beneficial in the treatment of humans poisoned with yew.

ORAL AND GASTROINTESTINAL SYSTEM TOXINS

Gastrointestinal Irritants (Resins, Alkaloids)

Gastrointestinal toxins found in plants are listed in Box 58-4.

Chinaberry Trees. *Melia azedarach* plants (Fig. 58-22) are found throughout the southern United States and Hawaii. The chinaberry tree, also known as China tree, Texas umbrella tree, or white cedar tree, contains tetranortriterpenes (resins), identified as meliatoxins A_1, A_2, B_1, and B_2, that are enterotoxic and neurotoxic. The exact mechanisms of these toxins have yet to be determined. Gastroenteritis can occur after ingestion of any part of the plant but typically occurs after the berries are ingested by children. Immature berries are green but turn yellow

Box 58-4. Gastrointestinal Toxins Found in Plants

ALKALOIDS
Amine (e.g., colchicine)
Isoquinoline and quinoline (e.g., emetine in ipecac)
Pyrrolizidine
Steroid (e.g., solanine)

GLYCOSIDES
Anthraquinone
Coumarin
Saponin (e.g., phytolaccatoxin)

RESINS
Cicutoxin
Meliatoxins

PHYTOTOXINS
Abrin
Curcin
Ricin

and wrinkle with age. After ingestion of as little as one berry, severe gastroenteritis ensues, often with bloody diarrhea. Symptoms may be rapid or delayed for several hours after ingestion. Death has been reported in children after the ingestion of berries of the African variety of chinaberry tree.[97] Treatment is supportive, with replacement of fluids and electrolytes and administration of activated charcoal. If hypotension ensues, it generally responds to IV fluids.

Solanum. The genus *Solanum* comprises the largest group of steroid alkaloid–containing plants, which include *Solanum tuberosum* (potato), *Solanum gracile* (wild tomato), *Solanum carolinense* (horse nettle), *Solanum pseudocapsicum* (Jerusalem cherry), *Solanum dulcamara* (woody nightshade), *Solanum americanum* (black nightshade), and other nightshade plants. *Solanum* species are used medicinally in some countries. Solanine, a glycoalkaloid with a steroidlike moiety, has been isolated from more than 1700 different *Solanum* species. It is found throughout the plants but is most concentrated in unripe fruits. Solanine generally produces gastroenteritis, but bradycardia, weakness, and CNS and respiratory depression may be seen. Treatment is supportive, with replacement of fluids and

A

B

Figure 58-22. Chinaberry *(Melia azedarach)* tree **(A)** and berries **(B).** (Courtesy Kimberlie Graeme, MD.)

electrolytes and administration of activated charcoal. If hypotension ensues, it generally responds to IV fluids. Atropine may be beneficial if bradycardia develops. Spontaneous recovery usually occurs in 1 to 3 days.[36,48,84]

Saponin Glycosides (Pokeweed). Saponin glycosides are found throughout the plant kingdom—for example, in English ivy *(Hedera helix)*, pokeweed *(Phytolacca americana)*, tung tree (*Aleurites* species), ginseng (*Panax ginseng* or *quinquefolium*), and licorice *(Glycyrrhiza glabra)*.[79] Saponins may induce lysis of erythrocytes, causing hemolytic anemia. In addition, saponins are GI irritants that facilitate their own intestinal absorption. Most saponins are found in combination with other toxins, resulting in diverse clinical syndromes.

P. americana, or *Phytolacca decandra*, is most commonly known as pokeweed but is also known as Virginia poke, inkberry, pocan, pigeonberry, American cancer-root, garget, red ink, American nightshade, scoke, jalap, and redwood. It may be mistaken for horseradish, parsnips, or Jerusalem artichoke. *P. americana* has green leaves with red stalks. Berries are green when immature and turn deep purple with maturity. Pokeberries typically leave a purple stain. The root is the most toxic part of the plant. *P. americana* is ingested intentionally in pokeweed salad and pokeberry teas. When prepared for ingestion, leaves should be parboiled, which entails boiling the leaves first in water that is discarded before boiling the leaves again and rinsing them with fresh water. However, parboiling does not necessarily offer protection against toxicity.[79]

The saponin glycosides phytolaccatoxin and phytolaccagenin account for the GI injury, which manifests as fulminant gastroenteritis, with vomiting and diarrhea 2 to 4 hours after ingestion. Diarrhea may appear foamy from the sudsing effect of saponin glycosides. Hypotension may follow significant GI fluid losses. Severe ingestions may result in weakness, loss of consciousness, seizures, and respiratory depression.[79]

P. americana also contains mitogenic, hemagglutinating, and antiviral proteins. Pokeweed mitogen may induce morphologic changes in lymphocytes and plasma cells. An increased number of circulating plasmablasts and proplasmacytes, eosinophilia, and thrombocytopenia can be seen after ingestion or after handling of *P. americana* with broken skin. Pokeweed antiviral protein is a ribosome-inactivating protein being investigated for cancer treatment.[110]

Treatment for acute ingestion includes administration of activated charcoal, and fluid replacement for dehydration secondary to GI losses. Airway support may be needed. Seizures should be treated with benzodiazepines. Hematologic changes generally resolve within weeks.[79]

Anthraquinone Glycosides. The aglycone moiety of this group is anthraquinone, which is known to have cathartic activity. Herbal teas that contain leaves, flowers, or bark of senna *(Cassia senna)*, leaves of aloe *(Aloe barbadensis)*, and bark of buckthorn *(Rhamnus frangula)* can cause severe diarrhea.[1] Treatment is supportive, emphasizing adequate volume and electrolyte replacement.

Toxins That Inhibit Protein Synthesis (Phytotoxins)

Phytotoxins (Ricin, Abrin). Phytotoxins are found in the families Fabaceae—for example, *Abrus precatorius* (jequirity bean, rosary pea, prayer bead), which contains abrin—and Euphorbiaceae—for example, *Ricinus communis* (castor bean) (Fig. 58-23A), which contains ricin, and *Jatropha curcas* (purging nut), which contains curcin. These toxins inhibit protein synthesis and cause cell death. Ricin and abrin are glycoproteins composed of two peptide chains, designated A and B, connected by a disulfide bond (see Figure 58-2). Chain B binds to the cell membrane, whereas chain A penetrates the cell by endocytosis and interferes with ribosomes and disrupts protein synthesis, leading to cell death. Much of the toxicity is the result of endothelial cell damage, which causes increased capillary permeability, fluid and protein leakage, and edema.[5,25,56,58,93,156]

Ricin poisoning results from eating or chewing on the nut or seed. Fresh, immature castor beans are encased within a soft, thorny, red seedpod. With age, the seeds and surrounding seedpod become brown, and the seeds develop hard shells that are difficult to penetrate but can be punctured by teeth (see

A

B

Figure 58-23. Castor bean plant *(Ricinus communis)* **(A)** contains ricin and is characterized by broad, serrated leaves, with fresh beans encased within a soft, thorny, red seedpod. With age, the seeds and their surrounding seedpod become brown, and the seeds develop hard shells that are difficult to penetrate **(B).** (Courtesy Phillip Saba, MD, and Kimberlie Graeme, MD.)

Figure 58-23B).[56,72,156] The oral lethal dose is estimated to be 1 mg/kg, theoretically as little as one bean in a child and eight to 10 in an adult.[25,156] After a latent period of 1 to 6 hours, nausea, vomiting, diarrhea, hemorrhagic gastritis, abdominal pain, thirst, dehydration, hypotension, and shock may occur. Liver disease, with elevated LFTs, is common.[25,58] Death may occur from dehydration and electrolyte imbalances; convulsions may precede death.[25,93,156] Death usually occurs on the third day or later due to multiorgan failure.[25] Autopsy may reveal GI tract ulcerations, necrosis of the lymph nodes and liver, nephritis, and pulmonary edema.[25]

A discrepancy between the serious toxicity of isolated ricin exposure and the milder toxicity of ingested seeds of *R. communis* has been recognized. A young adult consumed 10 to 15 seeds of *R. communis* and experienced severe crampy abdominal pain and vomiting 4 hours after ingestion, without GI bleeding or abdominal guarding. He received IV fluids. By the third day he had recovered completely. Other cases of oral ingestion of masticated seeds indicate that GI distress may be followed a few days later by elevated liver enzymes or hyperbilirubinemia, followed by complete recovery.[58,156] In contrast, a Bulgarian broadcaster who was injected with isolated ricin died from severe gastroenteritis and multiorgan failure.[5,25,58] Ingested *R. communis* is much less toxic than parenteral ricin. The degree of mastication of castor beans may determine the degree of toxicity seen with oral ingestions. Fresh seeds are likely to be more toxic than aged seeds that are enveloped by a hard coat. Although deaths have been reported with ingestions of a couple of seeds, toxicity is unlikely if mature seeds are swallowed whole.[25,156] Furthermore, some of the toxin, which is a protein, is very likely digested in the GI tract.[56]

Similarly, deaths have been reported after ingestion of fresh jequirity beans *(A. precatorius)* if the soft, immature bean is chewed or if the hard shell of the mature bean is penetrated. The estimated human fatal dose of abrin is 0.1 to 1 µg/kg. A half of a seed has been reported to cause toxicity in a child, and one chewed bean could be fatal. However, as with castor beans, the mature bean shell is often not penetrated, and therefore, significant toxicity is not often seen. For example, a 15-month-old child ingested more than 20 jequirity beans with only minor clinical toxicity, including mild hepatomegaly and mild elevation of aspartate aminotransferase (AST).[56,72]

Symptoms generally begin within a few hours. Mild symptoms involve diarrhea alone. More severe symptoms include persistent vomiting, diarrhea, and abdominal pain, with associated hypovolemia and hypokalemia.[5] Hemorrhagic gastritis and bloody diarrhea may ensue. Dehydration may be associated with shock and hepatorenal dysfunction. Severely ill patients may present with hyperthermia, mental status changes, and seizures. Systemic effects may be delayed as inhibition of protein synthesis occurs. Delayed effects may occur 1 to 5 days after ingestion. Terminally, elevated serum LFTs and CPK levels may be seen, indicating hepatorenal failure. If death occurs, it is generally 3 to 5 days after ingestion and is associated with seizures, tachycardia, GI distress, mucosal changes of the GI tract (including edema and hemorrhage of the Peyer's patches), focal necrosis and failure of the liver and kidneys, and retinal hemorrhage. Cerebral and pulmonary edema, hypertension, and pancreatitis have also been observed.[56,72,130]

Treatment. Treatment is supportive, including fluid and electrolyte replacement and activated charcoal administration. Patients who remain asymptomatic for 4 to 6 hours after ingestion of seeds may be discharged, with instructions to return if symptoms develop. Laboratory follow-up for delayed liver toxicity may be appropriate.[58,156] Studies indicate that ricin antibody administered intravenously shortly after exposure could be beneficial.[25] Animal studies indicate that vaccination with abrin and ricin toxoids may offer protection against subsequent abrin and ricin challenges, respectively; however, there does not appear to be cross immunity between abrin and ricin toxoids.[25,56] These toxoids and antibodies are not clinically available.

Toxins That Inhibit Cell Division

Colchicine. Colchicine toxicity can occur after ingestion of *Sandersonia aurantiaca* (Christmas-bells, Chinese lantern lily), *Gloriosa superba* (glory lily), and, more commonly, *Colchicum autumnale,* all of the lily family. *C. autumnale* is commonly known as autumn crocus, wild saffron, meadow saffron, naked lady, naked boy, and son-before-the-father, and it can be mistaken for edible plants such as leek or wild garlic. *C. autumnale* is found in Europe, North America, and Asia. All parts of the plant contain colchicine, an amine alkaloid, but the highest concentrations are found in the underground bulb. Colchicine binds selectively and reversibly to microtubules, disrupting cell division, cell shape, mobility, and phagocytosis. Cells with the highest turnover, such as those of the GI mucosa and bone marrow, are affected most severely. Cell arrest in metaphase and abnormal nuclear morphology are seen at autopsy; the clumps of chromatin material seen in the nuclei are called colchicine bodies.[26,27,50,65,78,81,119,174]

Acute poisoning may occur after a latent period of several hours. Initial GI effects are severe abdominal pain, nausea, vomiting, diarrhea and hemorrhagic gastroenteritis, which may result in electrolyte abnormalities, volume depletion, acidosis, shock, arrhythmias, and multiorgan failure. Muscular weakness and ascending paralysis may cause respiratory arrest, which may occur with a clear sensorium. Rhabdomyolysis and pancreatitis have also been reported. Bone marrow depression with pancytopenia may follow and may predispose to hemorrhage and infection. Autopsy reveals a hypocellular bone marrow. Autopsy may also reveal congestion of the liver, kidneys, spleen, lungs, and brain; occasionally, hemorrhagic edema is seen. Patients that survive severe toxicity may develop alopecia days to weeks after poisoning.[26,27,50,65,81,98,109,119,174]

Colchicine-specific Fab antibody fragments have been developed and utilized with success in France for the management of acute poisoning with the drug form of colchicine, but this treatment is not commercially available; therefore, treatment is symptomatic and supportive.[26,27,65,81] Pulmonary function tests can be used to monitor respiratory function, assessing for fatigue and progressive ascending paralysis. Assisted ventilation is used as needed. Parenteral analgesics are given cautiously to relieve severe abdominal pain, because colchicine sensitizes patients to CNS depressants. Fluid and electrolyte replacement and occasionally blood component replacement and granulocyte colony-stimulating factor (G-CSF) may be necessary. Maintain adequate urine output and assess for infection.[27,81,98,109]

Podophyllum. *Podophyllum peltatum* is most commonly known as the mayapple but has also been called American mandrake. Because of this common name, it has been occasionally

confused with true mandrake, *Mandragora officinarum,* an unrelated plant with anticholinergic properties. Toxicity resulting from *P. peltatum* exposure is caused by the glucoside podophyllotoxin. Acute toxicity after ingestion of *P. peltatum* may occur after a latent period of several hours, followed by GI distress and associated hypovolemia. Nervous system effects (confusion, delirium, coma, peripheral neuropathy), bone marrow depression, and multiorgan failure may ensue due to the antimitotic effect of podophyllotoxin. Treatment is similar to that for colchicine.[77]

Hepatotoxic Agents

Pyrrolizidine Alkaloids. Plants containing pyrrolizidine alkaloids include *Senecio vulgaris* (groundsel), *Senecio longilobus* (gordolobo), *Senecio jacobaea* (tansy ragwort), *Senecio latifolius* (Dan's cabbage or muti), *Symphytum officinale* (comfrey), *Gynura segetum, Ilex paraguanyensis* (mate), *Heliotropium* species, *Crotalaria* species (rattlebox), *Amsinckia intermedia* (fiddle neck or tar weed), *Baccharius pteronoides, Astragulus lentiginosus, Gnaphalium, Cynoglossum, Echium, Tussilago farfara,* and *Adenotyles alliariae* (alpendost). These plants are consumed in herbal preparations, in breads made with grains that are contaminated with pyrrolizidine-containing weeds ("bread poisoning"), and in teas. Less commonly, contaminated animal meat, milk, or honey from exposed animals or bees, respectively, may be the source of exposure. Toxicity is associated with hepatic veno-occlusive disease, hepatomegaly, cirrhosis, and Budd-Chiari syndrome, which is characterized by obstruction of the trunk or large branches of the hepatic vein. For example, *Senecio* and *Crotalaria,* which are used to make "bush tea" in Jamaica, have been associated with hepatotoxicity.[166,169,189,190,193]

Pyrrolizidine alkaloids act as alkylating agents and are activated in the liver, by cytochrome P_{450} and mixed-function oxidase systems, to form reactive pyrrolic metabolites that alkylate proteins and DNA, thereby inhibiting protein and nucleic acid synthesis. Young victims seem more susceptible than adults. Ten milligrams of pyrrolizidine alkaloid is probably enough to produce acute or chronic veno-occlusive disease. Veno-occlusive liver disease is characterized by nonthrombotic occlusion of the central veins of hepatic lobules. Histologic liver findings include central vein dilation, sinusoidal congestion, centrilobular necrosis, and fibrosis. Clinical evidence of intrahepatic portal hypertension may include painful hepatomegaly, ascites, weight gain, and jaundice. Hepatic failure and death may ensue. Neonatal death secondary to hepatotoxicity has been reported after intrauterine exposure to pyrrolizidine alkaloids from a herbal preparation; neonatal ascites, skin edema, hepatomegaly, and anemia were noted. Elevated LFTs and bilirubin, as well as hyponatremia, may be noted. Occasionally, laparoscopic liver biopsy and hepatic and portal decompression are helpful in diagnosis and treatment, respectively. Treatment, including albumin infusions, diuretics, and therapeutic paracentesis, is supportive. In theory, *N*-acetylcysteine may reduce the toxicity. Cirrhosis and portal hypertension may be persistent.[15,42,166,169,189,190,193,218]

Atractylosides. *Atractylis gummifera* (bird-lime, blue thistle) is found in North Africa and the Mediterranean. It has a sugary taste and has been confused with an edible wild artichoke. The juice of the rhizome contains two poisonous glucosides, atractyloside and carboxyatractyloside. The glucosides inhibit oxidative phosphorylation and block the conversion of ADP to ATP. The toxins inhibit the action of P_{450} and b_5 cytochromes. Clinically, headache, abdominal pain, vomiting, hematemesis, diarrhea, and dizziness are followed by liver failure, associated with jaundice and hepatitis. Seizures, coma, dysrhythmias, and renal failure may also be seen. Death is generally from multiorgan failure. Autopsy reveals lesions, necrosis, and congestion of the liver and kidneys.[93,94]

Oral Irritants (Glycosides, Oxalates)

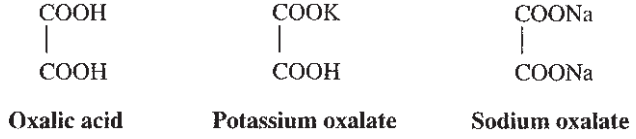
Daphne. Coumarin glycosides produce effects similar to those of irritant glycoalkaloids of the steroid alkaloid group. The plant of significance is daphne *(Daphne mezereum),* with its fragrant, succulent berries. The widely cultivated daphne represents a significant risk to curious children, for whom only a few ingested berries may be lethal.[130] The fruits contain a coumarin glycoside and a diterpene that irritate mucous membranes, with swelling of the tongue and lips. Blisters form if berries are rubbed on the skin. Severe gastroenteritis with GI bleeding may occur after ingestion.[69] In addition, progressive weakness, paralysis, seizures, and coma may develop. Treatment is supportive.

Insoluble Oxalates. Plant exposures involving *Philodendron, Dieffenbachia* (dumb cane), and *Spathiphyllum* species (peace lily) (Fig. 58-24), and *Colocasia* species (elephant's ear, common calla) are frequently reported.[122,134,159] These plants contain insoluble oxalates arranged in numerous needles of calcium oxalate (raphides) contained in specialized cells (idioblasts). When stimulated, as by mastication, idioblasts fire raphides that become embedded in exposed tissues, resulting in painful oropharyngeal edema, hypersalivation, and aphonia. These plants also contain trypsinlike proteases, histamine, and kininlike substances that may contribute to toxicity.[134,198]

Accidental ingestion of *Dieffenbachia* may result in painful edematous swelling, including angioedema. The victim may have dysphagia. *Dieffenbachia* is commonly known as dumb cane because of the victim's inability to speak after chewing the plant. Vesicle, bullae, or ulcer formation of the oral mucous membranes and esophageal erosions may be noted. Respiratory obstruction (resulting from edema) and sudden death have been reported. Treatment is supportive, with special attention to maintaining a patent airway. After massive ingestions, hypocalcemia and its effects should be anticipated.[134,159,198]

RENAL SYSTEM TOXINS

Soluble oxalates are found in rhubarb *(Rheum rhaponticum)* and sorrel (*Rumex* species) plants. Soluble oxalates are rapidly absorbed via the GI tract. Generalized disturbance of monovalent and divalent cation metabolism occurs. Serum ionized calcium levels may drop rapidly. Weakness, tetany, hypotension, and seizures may develop. Acute renal failure may occur if calcium oxalate precipitates in urine and obstructs the renal tubules.[71,117,118,175] The structures of oxalates follow:



Oxalic acid **Potassium oxalate** **Sodium oxalate**

A

B

C

Figure 58-24. Plants that are oral irritants. *Philodendron* **(A),** *Dieffenbachia* **(B),** *Spathiphyllum* **(C).** (Courtesy Kimberlie Graeme, MD.)

Sodium bicarbonate lavage should be avoided because of the risk of sodium oxalate formation. IV calcium is recommended for tetany, prolonged QT interval on ECG, or depressed serum ionized calcium. Urine output should be maintained to prevent deposition of calcium oxalate crystals in renal tubules.

ENDOCRINE AND METABOLIC SYSTEM TOXINS

Plants That Induce Hypoglycemia

Ackee Fruit. *Blighia sapida,* or ackee (akee) fruit, trees are indigenous to West Africa but are prevalent in the West Indies, Central America, and southern Florida. Unripe ackee fruit has a closed yellow aril that is toxic. Ripe ackee fruit, with a spontaneously opened red aril, is nontoxic. The seeds contain hypoglycin B and are always toxic. Unripe ackee fruit contains hypoglycin A, or L(R,S)-2-amino-3-methylenecyclopropylpropionic acid, a water-soluble amino acid that is converted to methylenecyclopropylacetyl-coenzyme A in vivo. Both compounds are hypoglycemic agents; the second is a suicide inhibitor of β-oxidation of fatty acids. Inhibition of fatty acid metabolism results in microvesicular steatosis of the liver, hyperammonemia, metabolic acidosis, and hypoglycemia. Laboratory and histopathologic findings are indistinguishable from those of Reye's syndrome, but urine shows increased concentrations of glutaric and ethylmalonic acids after hypoglycin A exposure.[136,182]

Ackee fruit constitutes a traditional Jamaican breakfast and has been associated with Jamaican vomiting sickness. Ingestion of unripe ackee fruit produced an outbreak epidemic of fatal encephalopathy in West Africa, primarily affecting children ages 2 to 6 years, who experienced vomiting, hypotonia, convulsions, and coma. All children died within 48 hours of the onset of vomiting. Necropsy revealed massive liver steatosis and severe hypoglycemia. Urine concentrations of dicarboxylic acids were elevated. Similarly, an epidemic of 80 cases occurred in Haiti, associated with vomiting, abdominal pain, loss of consciousness, convulsions, and many deaths. Fatal ackee fruit poisoning is more common in children than in adults; adults are more likely to present with self-resolving cholestatic jaundice.[136,142,182,183]

Treatment is largely supportive and consists of securing an airway and administering activated charcoal, glucose, and IV fluids. Before hypoglycemia was recognized, mortality rates of symptomatic unripe ackee fruit exposure approached 80%. Frequent glucose and electrolyte measurements with appropriate replacement are essential. Seizures are treated with benzodiazepines and barbiturates. Theoretically, riboflavin, glycine, methylene blue, and carnitine may be beneficial, although their efficacy is not established. Carnitine may facilitate transport of fatty acids into mitochondria and has been used for other toxins (e.g., valproate) that inhibit β-oxidation of fatty acids.[124,182,183]

Plants That Interfere with Steroid Metabolism

Licorice. Adverse effects with chronic ingestion of *Glycyrrhiza glabra* (natural licorice) result from altered steroid metabolism. *G. glabra* contains glycyrrhizic acid, which is converted to 18-β-glycyrrhetinic acid in the GI tract. Both acids inhibit 11-β-hydroxysteroid dehydrogenase, an enzyme essential to the in vivo conversion of cortisol to cortisone.[20,88] Excessive local cortisol binds to and activates mineralocorticoid receptors in the kidneys, producing a hypermineralocorticoid syndrome, characterized by water and sodium retention with potassium excretion. Signs and symptoms of chronic ingestion include hypertension, edema, hypokalemia, metabolic alkalosis, headache, paresthesias, weakness, paralysis, tetany, and muscle cramps.[67,70] Myopathy with reversible myoglobinuria has been reported.[16] Heart failure, arrhythmias (e.g., torsades de pointes), and cardiac arrest have been attributed to licorice root ingestion.[1]

Treatment consists of discontinuing exposure to licorice, maintaining good urine output, and alkalinizing the urine of patients with rhabdomyolysis. Occasionally, potassium-sparing diuretics (e.g., triamterene, spironolactone) are useful.[20] Torsades de pointes may respond to magnesium or potassium infusions.[67]

Plants That Inhibit Cellular Respiration

Cyanogenic Plants. Glycosides that yield hydrocyanic acid on hydrolysis are known as cyanogenic glycosides (see Figure 58-1). Cyanogenic glycosides include amygdalin, prunasin, linamarin, lotaustralin, and triglochinin.[40] Amygdalin (D-mandelonitrile-β-D-glucoside-6-β-glucoside), which is abundant in the Rosaceae family, is the cyanogenic compound found in the seeds of *Malus* species (apples) and pits of *Prunus* species, including cherries, peaches, plums, and apricots (the commercial source of laetrile). Deaths have been reported after ingestion of apricot, apple, cherry, and other fruit seeds.[130,176] Black or wild cherries *(Prunus serotina)* are considered the most dangerous. Poisonings have resulted from milkshakes that include apricot kernels and from apricot kernels sold in health food stores as snacks.[1,196] Linseeds *(Linum usitatissimum)* and cycad seeds (*Cycas* species) are also cyanogenic.[40,170] Cassava *(Manihot esculenta)* contains the cyanogenic glucoside linamarin, which is rapidly hydrolyzed to acetone cyanohydrin, which breaks down into hydrogen cyanide and acetone.[68,171] Both chronic and acute cyanide toxicity have been reported after ingestion of cassava. Chronic ingestion produces an upper motor neuron disease known as Konzo or tropical spastic paraparesis. Chronic exposure to cyanogenic glycosides occurs with cassava consumption during droughts in areas where the bitter roots of cassava are a staple food, such as in Africa. To remove cyanogens, roots should be soaked; however, when water is scarce, this soaking process is limited. Konzo outbreaks have been reported in populations exposed chronically to cassava that is not properly soaked.[13,14,68]

Cyanide combines with and inhibits many enzymes. It possesses great affinity for the ferric iron in cytochrome oxidase of the electron transport chain, accounting for most of its toxicity (Fig. 58-25). By combining with cytochrome oxidase, cyanide prevents electron transport, thereby preventing ATP production by oxidative phosphorylation. Metabolic acidosis ensues. Humans detoxify cyanide by transferring sulfane sulfur to cyanide to form thiocyanate. Numerous sulfur sources are most likely acted on by various sulfurtransferases to form the sulfane sulfur needed to convert cyanide to thiocyanate. Administration

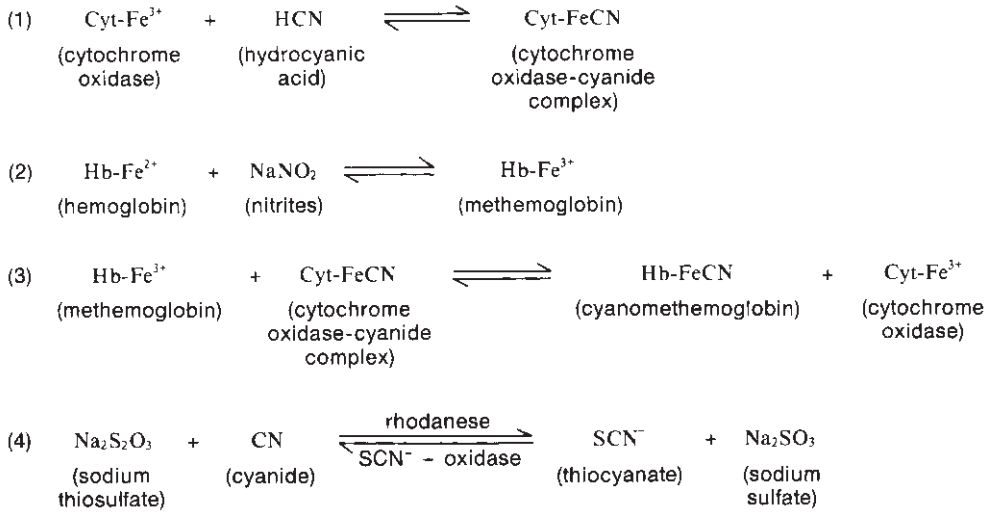


Figure 58-25. Principal steps in hydrocyanic acid poisoning and detoxification. *1,* Breakdown of cellular respiration resulting from the binding of cyanide to cytochrome oxidase. *2,* Conversion of the ferrous (Fe^{2+}) form of hemoglobin to the ferric (Fe^{3+}) form (methemoglobin) via nitrites. *3,* Preferential binding of cyanide to methemoglobin, liberating cytochrome oxidase and restoring cellular respiration. *4,* Providing exogenous thiosulfate to aid in formation of the less toxic thiocyanate via various sulfurtransferases, such as rhodanese. Thiocyanate is then excreted from the body. The reaction is slowly reversible via the enzyme thiocyanate oxidase, and rebound may occur.

of exogenous sulfane sulfur, such as sodium thiosulfate, can greatly facilitate this detoxification.[46]

Clinically, GI distress, bitter almond breath, CNS changes (agitation, anxiety, excitement, weakness, numbness, hypotonia, spasticity, coma, seizures), respiratory changes (hyperpnea, dyspnea, apnea, cyanosis), cardiovascular changes (tachycardia and hypertension followed by bradycardia and hypotension, heart block, ventricular arrhythmias, asystole), and metabolic changes (anion gap metabolic acidosis) are seen. Skin color may be pink or cyanotic, and the partial pressure of oxygen may be normal in cyanide poisoning. ECG may reveal T-on-R phenomenon as a result of progressive shortening of the ST segment. Multiorgan failure and death may occur.[46,171]

Treatment. Patients with cyanogenic glycoside poisoning may respond to treatment with 100% oxygen and the cyanide antidote kit, which includes amyl nitrite, sodium nitrite (3% solution given intravenously on the basis of hemoglobin and weight: generally 300 mg in a nonanemic adult), and sodium thiosulfate (12.5 g in an adult). Red blood cell or plasma cyanide levels can be determined; however, treatment should be initiated promptly, without confirmation of exposure, in patients with evidence of toxicity. Cyanide antidote kits are designed to induce methemoglobinemia through nitrite exposure. Cyanide binds preferentially to the ferric ion of methemoglobin rather than to that of cytochrome oxidase in the electron transport chain. Forming methemoglobin limits inhibition of electron transport by cyanide and restores cellular respiration. Sodium thiosulfate in the cyanide kit provides exogenous sulfane sulfur groups that bind to cyanide and form thiocyanate. Although not available in the United States, hydroxocobalamin is used as antidotal therapy in some countries. Cyanide combines with hydroxocobalamin to form cyanocobalamin, which is excreted in the urine and bile. Dimethylaminophenol (DMAP), a methemoglobin-forming antidote, and dicobalt edetate, a cyanide chelator, have also been suggested as antidotes. Supportive care, including mechanical ventilation, intravenous fluids, and vasopressors, may be utilized.[46,171,196]

HEMATOPOIETIC SYSTEM TOXINS

Plants with Anticoagulant Properties (Lactone Glycosides)

Dipteryx odoratum and *Coumarouna* (or *Dipteryx*) *oppositofolia* (tonka bean) were among the first historically mentioned medical sources of coumarin. *Melilotus officinalis* (melilot), *Asperula odorata*, and *Galium odoratum* (woodruff) also contain significant quantities of coumarin. Occasionally, these coumarin-containing products are consumed in teas.[169] *Melilotus* (sweet clover) poisoning is caused by dicumarol, a fungal metabolite produced from substrates in sweet clover. Fungi dimerize coumarin to produce dicumarol. Dicumarol interferes with the synthesis of vitamin K–dependent coagulation factors, inducing coagulopathy. Generally, this has been a problem in livestock but not humans.[165]

Plants That Induce Hemolysis

Fava Beans. Fava beans *(Vicia faba)* contain vicine and convicine, two metabolically inactive glycones that may be cleaved by β-glycosidase to produce divicine and isouramil. Divicine and isouramil may account for the hemolytic crisis seen in response to oxidative stress when individuals deficient in glucose-6-phosphate dehydrogenase ingest fava beans. Favism has been reported in infants who ingest breast milk from mothers who have recently ingested fava beans and in fetuses of mothers who have ingested fava beans. Clinically, favism is characterized by hemoglobinuria, anemia, and jaundice.[45,100] Patients may present with complaints of malaise, weakness, lethargy, nausea, vomiting, headache, and lumbar or abdominal pain. Renal failure may ensue.[95] Supportive care is the mainstay of treatment. Occasionally, transfusions and hemodialysis are needed.

REPRODUCTIVE SYSTEM TOXINS

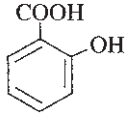
Plants used as abortifacients include *Ruta chalepensis* (ruda), *Lycopodium saururus* (cola de quirquincho), *Petroselinum hortense* (parsley), *Mentha pulegium* (pennyroyal), and *Thevetia peruviana* (yellow oleander). These plants generally cause significant morbidity and mortality to the women using the plants. Pennyroyal is discussed later, and yellow oleander was discussed earlier (see Cardiotoxins That Inhibit Na^+,K^+ ATPase).[43,65]

OTHER TOXINS

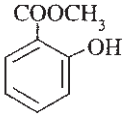
Oils

Irritant oils, including various mustards (*Brassica* species), horseradish *(Amoracia lapathifolia),* and protoanemonin from the buttercup family (Ranunculaceae), induce gastroenteritis. Essential oils, often found in combination with resins (oleoresins), are extracted commercially for use as rubefacients, salves, and liniments. Essential oils include wintergreen, eucalyptus, wormwood, and pennyroyal oils.

Wintergreen. Methyl salicylate is the methyl ester of salicylic acid, derived from *Gaultheria procumbens* (wintergreen). Widely used as an antirheumatic agent in the 19th century, it fell into disfavor because of its extreme side effects and the availability of the more effective acetylsalicylic acid. Poisoning now occurs most often in children, who are attracted to the color, smell, and flavor of wintergreen oil. As little as 4 mL may result in fatality. Oil of wintergreen is found in herbal products, such as Koong Yick Hung FA oil, and ingestion has caused significant toxicity. Oil of wintergreen may also produce laryngeal edema. Poisoning is identical to salicylism, including CNS excitation, hyperventilation, hyperthermia, and metabolic acidosis. Treatment is as for salicylism.[22,106] Structures of salicylic acid and methyl salicylate follow:



Salicylic acid



Methyl salicylate

Eucalyptus. The toxic component of eucalyptus oil is 1,8-cineole (eucalyptol), which is derived from leaves of *Eucalyptus globulus*. Ingestion of eucalyptus oil is associated with diaphoresis, CNS depression (hyporeflexia, weakness, ataxia, slurred speech, agitation, confusion, coma, respiratory depression, convulsions), GI upset (vomiting, diarrhea, abdominal pain), and respiratory effects (bronchospasm, pneumonitis, pulmonary edema). Metabolic acidosis, rhabdomyolysis, hypotension, and death may occur. Fatalities have been reported after ingestion of as little as 3.5 mL and may occur as early as 15 minutes after ingestion. Topical application may also result in significant CNS toxicity. The urine may smell of eucalyptus oil, violets, or fresh-mown hay.[10,51,200]

Wormwood. Wormwood oil, from the plants *Artemisia absinthium* and *Artemisia pontica,* is used to make the emerald green liqueur absinthe, which has been ingested for its psychoanaleptic effect. It was eventually banned because it produces neurotoxicity, but it is making a comeback. Wormwood oil contains α- and β-thujone. Alpha-thujone is an antagonist at $GABA_A$ receptors. Wormwood oil may cause vomiting, vertigo, delirium, hallucinations, convulsions, coma, respiratory arrest, and death.[18,55,107,154,195]

Pennyroyal. Pulegone, a monoterpene, is the primary toxin of pennyroyal oil, which is derived from *Hedeoma pulegioides* or *Mentha pulegium.* Pennyroyal oil also contains several other monoterpenes. Pulegone is oxidized by hepatic cytochrome P_{450} enzymes to form the toxic metabolite menthofuran and other toxins. Menthofuran induces much of the hepatic and pulmonary damage. Reactive oxidative metabolites of pulegone and menthofuran bind to target cell proteins and induce cellular damage. *M. pulegium* use has caused death in women using it as an over-the-counter abortifacient and death in infants given mint teas made with the plant. Hepatic and neurologic injuries are generally seen. Hepatic failure followed by shock, multiorgan failure, and death may occur after ingestion. Neurologic manifestations include dizziness, obtundation, hallucinations, seizures, and cerebral edema. Laboratory studies may reveal elevated LFTs, coagulopathies, and metabolic acidosis. Liver histology at autopsy reveals centrilobular hepatic necrosis or confluent hepatocellular necrosis. Treatment includes oral administration of activated charcoal and fluid and electrolyte replacement as needed. *N*-Acetylcysteine has been used for treatment because the hepatotoxicity is similar to acetaminophen-induced hepatotoxicity and is associated with glutathione depletion. Animal studies indicate that treatment with cimetidine and disulfiram may be helpful.[9,11,121,197]

Elements and Nitrates

Many plants absorb or accumulate metallic compounds (selenium, molybdenum, arsenic, lead, cadmium), nitrites, and nitrates. They are generally of little importance in humans but constitute a danger to grazing livestock. However, numerous cases of methemoglobinemia in infants ingesting vegetables high in nitrate content have been reported. Vegetables cited include spinach, beets, cabbage, and carrots grown in high-nitrate soils. Nitrates are converted to nitrites in the infant gut.[114]

ACKNOWLEDGMENTS

I thank Donald B. Kunkel, George Braitberg, Michele Adler, and Edward C. Geehr for their contributions to previous editions of this chapter.

APPENDIX A: PLANTS: TOXIC PRINCIPLES AND THERAPEUTICS

APPENDIX A: Plants: Toxic Principles and Therapeutics

COMMON NAME	GENUS	SPECIES	POISONOUS PRINCIPLE	TOXIC PARTS	DISTRIBUTION*	SECTION† THERAPEUTIC REFERENCE
Ackee (Akee)	*Blighia*	*sapida*	Hypoglycin	Fruit wall, seeds, white aril	II, S FL, Carib	Hypoglycemic agents
Anemone	*Anemone*	spp.	Protoanemonin	All parts	II	Irritant and essential oils
Angel's trumpet	*Brugmansia*	*suaveolens*	Atropine, hyoscyamine, hyoscine	Leaves, flowers	II, VI, SE US, HI	Tropane alkaloids
Apple of Peru	*Nicandra*	*physalodes*	Unknown	Leaves, berries	I	General considerations
Apple seeds	*Malus*	spp.	Cyanogenic glycosides	Seeds	III	Cyanogenic glycosides
Apricot pits	*Prunus*	spp.	Cyanogenic glycosides	Pits	II	Cyanogenic glycosides
Arnica	*Arnica*	*montana, fulgens*	Unknown	Flowers, roots	II, N US, Can	General considerations
Autumn crocus	*Colchium*	*autumnale*	Colchicine	Seeds, corms	II	Amine alkaloids
Azalea (see Rhododendron)						
Balsam pear	*Momordica*	*balsamina*	Saponic glycoside	Seeds, wall of fruit	I, coastal FL to TX	Saponin glycosides
Baneberry	*Actaea*	spp.	Protoanemonin, unknown glycosides	Berries, rootstock	I, widely except SW	Irritant and essentials oils
Beech	*Fagus*	spp.	Saponic glycoside	Nuts	III	Saponin glycoside
Belladonna (see Deadly nightshade)						
Bellyache bush	*Jatropha*	*gossypiifolia*	Curcin	Fruit, seeds, sap	I, V	Phytotoxins
Betel nut	*Areca*	*catechu*	Arecoline, arecaine	Seeds	II, FL, HI	Pyridine-piperidine group
Bird of paradise	*Poinciana, caesalpinia*	*gilliesii*	Unknown	Green seedpods	II	General considerations
Black cherry	*Prunus*	*serotina*	Cyanogenic glycoside	Bark, leaves, seeds, tea of leaves	I, E NA	Cyanogenic glycosides
Black locust	*Robinia*	*pseudoacacia*	Robin, robitin (glycoside)	Inner bark, young leaves, seeds	I, S Can, E NA, MW, roadsides	Phytotoxins
Black nightshade	*Solanum*	*nigrum*	Solanine	Unripened fruit	I, widely, roadsides	Steroid alkaloids
Black snake root (see Death camas)						
Bleeding heart (see Dicentra)						
Bloodroot	*Sanguinaria*	*canadensis*	Sanguinarine	All parts	I	Isoquinoline and quinoline group
Blister bush	*Phebalium*	*anceps*	Unknown	Leaves, fruit	I	General considerations
Blue cohosh	*Caulophyllum*	*thalictroides*	Unknown	Seeds, rootstock	I	General considerations
Boxwood	*Buxus*	*sempervirens*	Buxine, volatile oil	Leaves, twigs	II, V	Alkaloids, irritant and essential oils

Continued

APPENDIX A: Plants: Toxic Principles and Therapeutics—Cont'd

COMMON NAME	GENUS	SPECIES	POISONOUS PRINCIPLE	TOXIC PARTS	DISTRIBUTION*	SECTION† THERAPEUTIC REFERENCE
Brazilian pepper (aka pink or red peppercorn, Florida holly)	*Schinus*	*terebinthifolius*	Unknown	Berries, leaves, flowers	I, FL	General considerations
Broom (aka Scotch broom)	*Cytisus*	*scoparius*	Cytisine	Branches	VI	Quinolizidine alkaloids
Buckeye	*Aesculus*	spp.	Aesculin	Leaves, flowers, young sprouts, seeds	III, widely	Coumarin glycosides
Buckthorn	*Rhamnus*	*cathartica, frangula*	Anthraquinones	Berries, leaves, bark	I, VI	Anthraquinone glycosides
Burning bush	*Euonymus*	spp.	Unknown	Leaves, bark seeds	III	General considerations
Bushman's poison	*Acokanthera*	spp.	Oubain, G-strophanthin, acokantherin	All	I	Cardiac
Buttercup	*Ranunculus*	spp.	Protoanemonin	All parts	III	Irritant and essential oils
Caladium	*Caladium*	spp.	Oxalates	Leaves	II, IV	Oxalates
Candle nut (see Lumbang nut)						
Caper spurge	*Euphorbia*	*lathyris*	Unknown alkaloid	Milky sap throughout plant	II	General considerations
Carolina jessamine (aka yellow jessamine)	*Gelsemium*	*sempervirens*	Gelsemine, sempervirine	All parts	II	Psychoactive plants
Cassava (aka manioc, tapioca)	*Manihot*	*esculenta*	Cyanogenic glycoside	Raw root	II, VI	Cyanogenic glycosides
Castor bean	*Ricinus*	*communis*	Ricin	Seeds	III	Phytotoxins
Catnip	*Nepeta*	*cataria*	Acetic, butyric and valeric acids, nepetalic acid, limonene	Leaves	VI	Psychoactive plants
Chalice vine (see also Trumpet flower)	*Solandra*	*nitida*	Solanine-like	Leaves, flowers	II	Steroid alkaloids
Cherry	*Prunus*	spp.	Cyanogenic glycoside	Pits	II	Cyanogenic glycosides
Chinaberry tree	*Melia*	*azedarach*	Unknown resin	Fruit, tea from leaves	III	Resins
Christmas rose	*Helleborus*	*niger*	Hellebrin, helleborin, helleborein	Rootstock and leaves	II	Cardiac glycosides
Clematis	*Clematis*	spp.	Steroid alkaloids	Seeds, young plants	II, widely	Steroid alkaloids
Coca	*Erythroxylon*	*coca*	Ecogonine	Extract of leaves	VI	Psychoactive plants
Coontie	*Zamia*	*floridana*	Unknown alkaloid	Fleshy seeds	II, S US, HI	General considerations
Coral plant	*Jatropha*	*multifida*	Curcin	Fruit, seeds, sap of all parts	III, S FL to TX, HI	Phytotoxins
Corn cockle	*Agrostemma*	*githago*	Githagenin, sapogenin	All parts, especially seeds	I	Saponin glycosides
Cotoneaster	*Cotoneaster*	spp.	Unknown	Berries	II	General considerations

APPENDIX A: Plants: Toxic Principles and Therapeutics—Cont'd

COMMON NAME	GENUS	SPECIES	POISONOUS PRINCIPLE	TOXIC PARTS	DISTRIBUTION*	SECTION† THERAPEUTIC REFERENCE
Coyotillo	*Karwinskia*	*humboldtiana*	Unknown	Fruit, seeds	I, SW	General considerations
Crepe jasmine	*Ervatamia*	*coronaria*	Unknown	Leaves, flowers	II	General considerations
Crownflower	*Calotropis*	*gigantea*	Unknown	All parts	II, V	General considerations
Crown of thorns	*Euphorbia*	spp.	Unknown alkaloid	Milky sap throughout plant	II, widely	General considerations
Cup of gold	*Solandra*	*guttata*	Solanine-like alkaloids	Leaves, flowers	II	Steroid alkaloids
Cycads (see Coontie)						
Cypress spurge	*Euphorbia*	*cyparissias*	Unknown alkaloids	Milky sap throughout plant	II	General considerations
Daffodil	*Narcissus*	spp.	Lycorine	Bulb	II	General considerations
Daphne	*Daphne*	*mezereum*	Dihydroxycoumarin, diterpene mezerein	Bark, leaves, fruit	II	Coumarin glycosides
Day jessamine	*Cestrum*	*diurnum*	Tropane alkaloids	All parts	III, FL, HI	Tropane alkaloids
Deadly nightshade	*Atropa*	*belladonna*	Atropine	All parts, black berries	I	Tropane alkaloids
Death camas (aka black snakeroot)	*Zigadenus*	spp.	Zygacine, zygadenine	Bulb	I, widely	Steroid alkaloids
Delphinium	*Delphinium*	spp.	Delphinine, ajacine	Seeds, young plants	III	Steroid alkaloids
Desert potato	*Jatropha*	*macrorhiza*	Phytotoxin	Plant root	I	Phytotoxins
Devil's trumpet (aka hairy thorn apple)	*Datura*	*metel*	Atropine, hyoscyamine, hyoscine	Leaves, flowers	II, VI, coastal FL to TX	Tropane alkaloids
Dicentra (aka bleeding heart, Dutchman's breeches)	*Dicentra*	spp.	Protopine, apomorphine, protoberberine	All parts	I, widely	Isoquinoline and quinoline group
Dieffenbachia (aka dumbcane)	*Dieffenbachia*	spp.	Oxalate, asparagine	Leaves	II, IV	Oxalates
Dogbane (aka Indian hemp)	*Apocynum*	*cannabinum*	Cymarin	Flowers, seeds, leaves	I, widely, roadsides	Cardiac glycosides
Duranta (see Sky flower)						
Elderberry	*Sambucus*	spp.	Unknown alkaloids	Unripe berries, leaves, wood	III, all NA except Pac Coast	General considerations
Elephant ear	*Colocasia*	*antiquorum*	Oxalates	Leaves	IV	Oxalates
English bean (see Fava bean)						
English ivy	*Hedera*	*helix*	Hederogenin	Berries, leaves	II, IV, V	Saponin glycosides
False hellebore (aka Indian poke)	*Veratrum*	spp.	Veratrin	Leaves	I, E NA, MN	Steroid alkaloids
False sago palm	*Cycas*	*circinalis*	Alkaloids	Seeds	II, V, S, HI	General considerations
Fava bean	*Vicia*	*faba*	Hemolytic anemia in glucose-6-phosphate deficiency	Bean	II	General considerations

Continued

APPENDIX A: Plants: Toxic Principles and Therapeutics—Cont'd

COMMON NAME	GENUS	SPECIES	POISONOUS PRINCIPLE	TOXIC PARTS	DISTRIBUTION*	SECTION† THERAPEUTIC REFERENCE
Finger cherry	*Rhodomyrtus*	*macrocarpa*	Saponin	Fruit	S Pac	Saponin glycosides
Fool's parsley	*Aethusa*	*cynapium*	Unknown	Leaves	I	General considerations
Four o'clock	*Mirabilis*	*jalapa*	Unknown	Roots, seeds	II	General considerations
Foxglove	*Digitalis*	*purpurea*	Digitoxin, gitaloxin, gitoxin	Leaves	III, W	Cardiac glycosides
Ginseng	*Panax*	*quinquefolium*	Saponin glycosides	Root	VI	Saponin glycosides
Glory lily	*Gloriosa*	*superba*	Colchicine-like alkaloids	Rhizomes	II	Amine alkaloids
Golden chain (aka golden rain)	*Laburnum*	*anagyroides*	Cytisine	Flowers, seeds	II, N, S Can	Quinolizidine alkaloids
Golden seal	*Hydrastis*	*canadensis*	Steroid alkaloids	Seeds, young plants	I, NE, VI	Steroid alkaloids
Ground cherry	*Physalis*	spp.	Unknown	Leaves, unripe fruit	I, II, NA, widely	General considerations
Hill gooseberry	*Rhodomyrtus*	*tomentosa*	None—NA nontoxic (*Rhodomyrtus* spp)		II, NA	—
Holly	*Ilex*	spp.	Ilicin	Berries	III, V	General considerations
Horse bean (see Fava bean)						
Horse chestnut	*Aesculus*	spp.	Aesculin	Sprouts, mature nuts	II	Coumarin glycosides
Horse nettle (aka wild tomato)	*Solanum*	*carolinense*	Solanine	Fruit	I, widely	Steroid alkaloids
Horseradish	*Armoracia*	*rusticana*	Mustard oil	Roots	II	Irritant and essential oils
Hyacinth	*Hyacinthus*	*orientalis*	Narcissine-like alkaloids	Bulb	II, IV	General considerations
Hyacinth bean	*Dolichos*	*lablab*	Cyanogenic glycosides	Pods, seeds	II	Cyanogenic glycosides
Hydrangea	*Hydrangea*	spp.	Cyanogenic glycosides	Leaves, buds	III	Cyanogenic glycosides
Inkberry (see Pokeweed)						
Iris (blue flag)	*Iris*	*versicolor*	Irisin, irigenin, iridin	Flowers, leaves	III	Resins
Jack-in-the-pulpit	*Arisaema*	spp.	Oxalate	Rhizome	I, NE	Oxalates
Jequirity pea (aka rosary pea, precatory bean)	*Abrus*	*precatorius*	Abrin	Beans	I, V	Phytotoxins
Jerusalem cherry	*Solanum*	*pseudocapsicum*	Solanine	Fruit	II, V, widely	Steroid alkaloids
Jessamine (see Carolina jessamine)						
Jessamines	*Cestrum*	spp.	Tropane alkaloids	All parts	III	Tropane alkaloids
Jetbead	*Rhodotypos*	*tetrapetala*	Cyanogenic glycosides	Berries	II, N	Cyanogenic glycosides
Jimsonweed	*Datura*	*stramonium*	Atropine, hyoscyamine, hyoscine	Leaves, flowers	I, VI	Tropane alkaloids
Jonquil (see Daffodil)						

APPENDIX A: Plants: Toxic Principles and Therapeutics—Cont'd

COMMON NAME	GENUS	SPECIES	POISONOUS PRINCIPLE	TOXIC PARTS	DISTRIBUTION*	SECTION† THERAPEUTIC REFERENCE
Kentucky coffee tree	*Gymnocladus*	*dioica*	Cytisine	Seeds, pulp	III, E NA, OK	Quinolizidine alkaloids
Lantana	*Lantana*	*camara*	Lantanin, lantadene A	Unripe fruit	II, N, SE	General considerations
Larkspur (see Delphinium)						
Lignum vitae	*Guaiacum*	*officinale*	Unknown	Resin in wood and fruit	II, S FL, S CA, HI	General considerations
Lily of the valley	*Convallaria*	*majalis*	Convallotoxin, convallarin, convallamarin	Rhizome, leaves, flowers	II, widely	Cardiac glycosides
Lobelia (aka Indian tobacco)	*Lobelia*	spp.	Lobelamine, lobeline	All parts	III, IV, VI	Pyridine-piperidine group
Lumbang nut	*Aleurites*	*trisperma*	Saponins	Seed	I, E NA, SW	General considerations
Manchineel tree	*Hippomane*	*mancinella*	Unknown	Milky sap	I	General considerations
Mandrake (aka Satan's apple)	*Mandragora*	*officinarum*	Hyoscyamine, scopolamine	Rhizome	VI	Tropane alkaloids
Marijuana (aka grass, dope, pot, ganja, pokololo)	*Cannabis*	*sativa*	Tetrahydrocannabinol	Leaves	III, VI	Psychoactive plants
Mayapple	*Podophyllum*	*peltatum*	Podophyllotoxin	All parts except ripe fruit	I, widely	General considerations
Mescal bean	*Sophora*	*secundiflora*	Unknown alkaloids	Seeds	III, VI	General considerations
Mexican prickly poppy (see Prickly poppy)						
Milk bush (aka pencil tree)	*Euphorbia*	*tirucallii*	Unknown alkaloids	Milky sap throughout plant	I	General considerations
Mistletoe (American)	*Phoradendron*	*serotinum*	Toxalbumin	Berries	I, V	General considerations
Mistletoe (European)	*Viscum*	*album*	Viscumin	Berries	I, V	General considerations
Monkshood (aka aconite, wolfsbane)	*Aconitum*	spp.	Aconitine	All parts, especially roots	III	Steroid alkaloids
Moonseed	*Menispermum*	*canadense*	Dauricine	Berries	I	Isoquinoline and quinoline group
Morning glory	*Ipomoea*	*violacea*	(+)-Lysergic acid amide	Seeds	II, VI	Psychoactive plants
Mountain ash	*Sorbus*	spp.	Unknown	Berries	I	General considerations
Mountain laurel	*Kalmia*	*latifolia*	Andromedotoxin, arbutin, grayanotoxins	All parts	III	Steroid alkaloids
Narcissus (see Jonquil)						
Night-blooming jasmine	*Cestrum*	spp.	Tropane alkaloids	All parts	I, S	Tropane alkaloids

Continued

APPENDIX A: Plants: Toxic Principles and Therapeutics—Cont'd

COMMON NAME	GENUS	SPECIES	POISONOUS PRINCIPLE	TOXIC PARTS	DISTRIBUTION*	SECTION† THERAPEUTIC REFERENCE
Nightshade (aka woody nightshade, climbing nightshade, bittersweet)	*Solanum*	*dulcamara*	Solanine	Fruit	I, V, widely	Steroid alkaloids
Nutmeg	*Myristica*	*fragrans*	Myristicine	Nut	VI	Psychoactive plants
Oak	*Quercus*	spp.	Tannin, unknown	Acorns	III, SW	General considerations
Ochrosia plum	*Ochrosia*	*elliptica*	Unknown	Fruit	II, FL, HI	General considerations
Oleander	*Nerium*	*oleander*	Oleandrin, oleandroside, nevioside	All parts	II, S, CA, HI, roadsides	Cardiac glycosides
Peach pits	*Prunus*	spp.	Cyanogenic glycosides	Pits	II, widely	Cyanogenic glycosides
Peyote	*Lophophora*	*williamsii*	Mescaline, lophophorine	Seeds, buttons	I, S TX	General considerations, psychoactive plants
Philodendron	*Philodendron*	spp.	Oxalate	Leaves	IV	Oxalates
Physic nut (aka purging nut)	*Jatropha*	*curcas*	Curcin	Fruit, seeds	II	Phytotoxins
Pigeonberry (see Pokeweed)						
Plum pit	*Prunus*	spp.	Cyanogenic glycosides	Pit	II, widely	Cyanogenic glycosides
Poinsettia	*Euphorbia*	*pulcherrima*	Unknown alkaloid	Milky sap throughout plant	II, V	General considerations
Poison hemlock	*Conium*	*maculatum*	Coniine	Seeds, roots, young leaves	I	Pyridine-piperidine group
Pokeweed (aka pokeberry, Virginia poke, scoke, garget, inkberry, caokum, American cancer, cancer jalap)	*Phytolacca*	*americana*	Triterpene saponins	All parts	I	Saponin glycosides
Pongam	*Pongamia*	*pinnata*	Unknown	Seeds, roots	II, S FL, S CA, HI	General considerations
Potato	*Solanum*	*tuberosum*	Solanine	Unripe tubers	II, widely	Steroid alkaloids
Prickly poppy	*Argemone*	spp.	Sanguinarine, berberine, protopine	All parts, especially seeds	III, VI	Isoquinoline and quinoline group
Privet	*Ligustrum*	*vulgare, japonicum*	Unknown glycoside	Berries, leaves	II, widely	General considerations
Purging nut (see Physic nut)						
Rattlepod (aka scarlet, wisteria tree)	*Sesbania*	spp.	Pyrrolizidine alkaloids	All parts	III, V, SE coastal, S CA, HI	Pyrrolizidine alkaloids
Rayless goldenrod	*Haplopappus*	*heterophyllus*	Tremetol	Milk of cows grazing on plant	I	Irritant and essential oils
Rhododendron (aka laurel, azalea)	*Rhododendron*	spp.	Grayanotoxins	All parts	III, E NA, Pac coast	Steroid alkaloids

APPENDIX A: Plants: Toxic Principles and Therapeutics—Cont'd

COMMON NAME	GENUS	SPECIES	POISONOUS PRINCIPLE	TOXIC PARTS	DISTRIBUTION*	SECTION† THERAPEUTIC REFERENCE
Rhubarb	*Rheum*	*rhabarbarum*	Oxalates	Leaves	II	Oxalates
Rock poppy	*Chelidonium*	*majus*	Sanguinarine, berberine, protopine	Leaves, seeds	I, E NA	Isoquinoline and quinoline group
Rubber vine	*Cryptostegia*	*grandiflora*	Unknown	All parts	II, V, S, HI	General considerations
Sandbox tree	*Hura*	*crepitans*	Unknown	Milky sap	II, S	General considerations
Senecio (aka threadleaf, groundsel)	*Senecio*	*longilobus*	Pyrrolizidine alkaloids	Entire plants	I, VI	Pyrrolizidine alkaloids
Sky flower	*Duranta*	*repens*	Saponin	Berries	I	Saponin glycosides
Snow-on-the-mountain	*Euphorbia*	spp.	Unknown alkaloids	Milky sap through plant	II	General considerations
Spring adonis	*Adonis*	*vernalis*	Steroid alkaloids	Seeds, young plants	II	Steroid alkaloids
Spurge	*Euphorbia*	spp.	Unknown alkaloids	Milky sap	III	General considerations
Squill	*Urginea*	*maritima*	Cardiac glycosides	Bulbs	II, S CA, SW	Cardiac glycosides
Star of Bethlehem	*Ornithogalum*	*umbellatum*	Cardiac glycosides, amine alkaloids	All parts	III, V, E NA, MW, HI	Cardiac glycosides, amine alkaloids
Strawberry bush (see Burning bush)						
Sweet pea	*Lathyrus*	spp.	β(λ-L-glutamyl)-aminopropionitrile, α, λ-diamino-butyric acid	Peas	III	General considerations
Tobacco	*Nicotiana*	*tabacum*	Nicotine	Leaves	II, VI	Pyridine-piperidine group
Tomato	*Lycopersicon*	*esculenta*	Solanine	Leaves	II	Steroid alkaloids
Tree tobacco	*Nicotiana*	*glauca*	Anabasine	All parts	I, SW, HI, Carib	Pyridine-piperidine group
Trumpet flower	*Solandra*	spp.	Solanine-like	Leaves, flowers	II	Steroid alkaloids
Trumpet lily	*Datura*	*arborea*	Atropine, hyoscyamine, hyoscine	Flowers, leaves	II, VI	Tropane alkaloids
Tulip bulb	*Tulipa*	*gesneriana*	Unknown	Bulb	II	General considerations
Tung oil tree	*Aleurites*	*fordii*	Saponins	Seed	II	Saponin glycosides
Virginia creeper (aka woodbine, American ivy)	*Parthenocissus*	*quinquefolia*	Unknown	Berries	I, E NA, SW	General considerations
Water hemlock	*Cicuta*	*maculata*	Cicutoxin	Roots	I, widely in swamps	Resins
White snakeroot	*Eupatorium*	*rugosum*	Tremetol	Milk of cows grazing on plant	I, NC, IL, IN, OH	Irritant and essential oils
Wild balsam apple	*Momordica*	*charantia*	Saponin glycoside	Seeds and wall of fruit	I	Saponin glycosides
Wild cherry (see Black cherry)						

Continued

APPENDIX A: Plants: Toxic Principles and Therapeutics—Cont'd

COMMON NAME	GENUS	SPECIES	POISONOUS PRINCIPLE	TOXIC PARTS	DISTRIBUTION*	SECTION† THERAPEUTIC REFERENCE
Wisteria	*Wisteria*	spp.	Resin	Seeds	II	Resin, general considerations
Woody nightshade (see Nightshade)						
Yellow allamanda	*Allamanda*	*cathartica*	Unknown	Fruit	II, S, HI	General considerations
Yellow jessamine (see Carolina jessamine)						
Yellow nightshade (aka wild allamanda)	*Urechites*	spp.	Unknown	Seedpods	I, S FL	General considerations
Yellow oleander (aka lucky nut)	*Thevetia*	*peruviana*	Thevetin, thevetoxin	Flowers, seeds, leaves	II, HI	Cardiac glycosides
Yew	*Taxus*	spp.	Taxine	Berries	III, V, E NA, Pac coast	Steroid alkaloids

*I, native or naturalized; found in fields, woods, and roadsides; II, cultivated; found in gardens and yards; III, found in both I and II; IV, common houseplant; V, found in decorations or as seasonal ornamentals; VI, found in herbal or folk remedies or used for mood alteration; Can, Canada; Carib, Caribbean; Coastal FL to TX, Coastal Florida to Texas; E NA, Eastern North America; FL, Florida; HI, Hawaii; IL, Illinois; IN, Indiana; MW, Midwestern United States; MN, Minnesota; NA, North America; NC, North Carolina; NE, Northeastern United States; N, Northern United States; OH, Ohio; Pac Coast, Pacific Coast states; Roadsides, Roadsides, waste areas, and swamps; S, Southern United States; S CA, Southern California; S Can, Southern Canada; SE, Southeastern United States; SE coastal, Southeastern coastal plain; SE, Southeastern United States; S FL, Southern Florida; S Pac, South Pacific; S TX Southern Texas; SW, Southwestern United States; W, Western United States; Widely, Distributed throughout the United States.

†See appropriate section in text.

APPENDIX B: NONTOXIC PLANTS

African violet *(Saintpaulia ionantha)*
Air plant *(Kalanchoe pinnata)*
Aluminum plant *(Pilea cadierei)*
Aralia, false *(Dizygotheca elegantissima)*
Aralia, Japanese *(Fatsia japonica)*
Asparagus fern *(Asparagus plumosus)*, berry
Baby's breath *(Gypsophilia paniculata)*
Baby's tears *(Helxine* [or *Soleirolia*] *soleirolii)*
Begonia *(Begonia rex)*
Bird of paradise* *(Strelitzia reginae)*
Bird's nest fern *(Asplenium nidus)*
Boston fern *(Nephrolepsis exaltata bostoniensis)*
Bromeliad family
California poppy *(Eschscholzia californica)*
Camellia *(Camellia japonica)*
Christmas cactus *(Schlumbergera bridgesii)*
Coffee tree *(Coffea arabica)*
Coleus
Coral berry* *(Aechamea fulgens, Ardisia crispa)*
Cornstalk plant *(Dracaena fragrans)*
Crape myrtle *(Lagerstromea indica)*
Creeping Charlie* *(Pilea nummularifolia)*
Crocus* (spring-blooming only)
Croton* *(Codiaeum variegatum)*
Dahlia
Dandelion *(Taraxacum officinale)*
Dogwood *(Cornus)*
Donkey's tail *(Sedum morganianum)*
Dragon tree *(Dracaena draco, Dracaena marginata)*
Easter cactus *(Schlumbergera bridgesii)*
Easter lily *(Lilium longiflorum)*
Echeveria: Mexican snowball, painted lady, plush plant
Emerald ripple *(Peperomia caperata)*
Fiddleleaf fig *(Ficus lyrata)*
Fig tree, weeping *(Ficus benjamina)*
Forget-me-not *(Myosotis alpestris, Myosotis sylvatica)*
Forsythia
Fuchsia
Gardenia
Geranium* *(Pelargonium)*
Gloxinia *(Sinningia speciosa)*
Grape ivy *(Cissus rhombifolia)*
Hawaiian ti plant *(Cordyline terminalis)*
Hawthorne *(Crataegus)*, berry
Heavenly bamboo *(Nandina domestica)*, berry
Hibiscus
Honeysuckle berry *(Lonicera)*
Ice plant
Impatiens walleriana
Jade plant *(Crassula argentea)*
Jasmine *(Jasminum rex)*, Madagascar jasmine
Kalanchoe: maternity plant, monkey plant, panda bear plant
Lace plant, Madagascar *(Aponogeton senetralis)*
Lady, lady's slipper *(Cypripedium, Paphiopedilum)*
Lily of the Nile *(Agapanthus)*
Lipstick plant *(Aeschynanthus radicans)*
Maidenhair fern *(Adiantum)*
Marigold, African/American tall *(Tagetes)*
Moon cactus *(Gymnocalycium)*
Mother-in-law's tongue or snake plant *(Sansevieria trifasciata)*
Mother of pearls *(Grapetopetalum paraguayense)*

Nandina berry
Natal plum *(Carissa grandiflora)*
Norfolk Island pine *(Araucaria heterophylla)*
Old man cactus *(Cephalocereus senilis)*
Olive tree *(Olea europaea)*
Orchid *(Cattleya, Cymbidium, Oncidium)*
Oregon grape *(Mahonia aquifolium)*
Palm: Bamboo *(Chamaedorea erumpeus)*
Paradise *(Howea* [or *Kentia*] *forsterana)*
Parlor *(Chamaedorea elegans* or *Kentia)*
Sentry *(Howea belmoreana)*
Pansy flower *(Viola)*
Peanut cactus *(Chamaecereus sylvestri)*
Pellionia
Peony flower *(Paeonia)*
Peperomia
Petunia
Phlox
Piggyback plant *(Tolmiea menziesii)*
Pigmy date palm *(Phoenix roebelenii)*
Pocketbook *(Calceolaria herbeohybrida)*
Polka dot or freckle face plant *(Hypoestes sanguinolenta)*
Prayer plant *(Maranta leuconeura)*
Pussy willow *(Salix discolor)*
Pyracantha berry
Queen's tears *(Billbergia nutans)*
Rabbit's foot fern *(Davallia fejeensis)*
Rainbow plant *(Billbergia saundersii)*
Raphiolepsis
Rattlesnake plant *(Calathea insignis)*
Ribbon plant *(Dracaena sandriana)*
Rock rose *(Cistus)*
Rosary pearls *(Senecio rowleyanus)*
Rosary vine *(Ceropegia woodii)*
Roses *(Rosa)*
Rubber plant *(Ficus elastica)*
Schefflera plant *(Brassaia* [or *Schefflera*] *actinophylia)*
Sedum
Sensitive plant *(Mimosa pudica)*
Silver heart *(Peperomia marmorata)*
Snake plant or mother-in-law's tongue *(Sansevieria trifasciata)*
Snapdragon *(Antirrhinum majus)*
Spider plant *(Anthericum, Chlorophytum comosum)*
Staghorn fern *(Platycerium bifurcatum)*
Starfish flower *(Stapelia)*
String of beads* *(Senecio rowleyanus* and *Senecio herreianus)*
String of hearts *(Ceropegia woodii)*
Swedish ivy *(Plectranthus australis)*
Sword fern *(Nephrolepsis cordifolia, Nephrolepsis exaltala)*
Tahitian bridal veil *(Gibasis geniculata, Tripogandra multiflora)*
Umbrella tree *(Schefflera actinophylla)*
Vagabond plant *(Vriesea)*
Velvet plant, purple *(Gynura aurantiaca)*
Venus fly trap *(Dionaea muscipula)*
Violet *(Viola)*
Wandering Jew *(Tradescantia albiflora)*
Wandering Jew-Red and White *(Zebrina pendulla)*
Wax plant *(Hoya exotica)*
Yucca
Zebra plant *(Aphelandre squarrosa)*
Zinnia

*This species has not been reported to cause illness, but other species may be toxic.
Modified from "Your Guide to Plant Safety," courtesy San Francisco Bay Area Regional Poison Center.

The references for this chapter can be found on the accompanying DVD-ROM.

Toxic Mushroom Ingestions

59

Sandra M. Schneider and Mark W. Donnelly

Had nature any outcast face? Could she a son condemn?
Had nature an Iscariot? That mushroom—it is him.
—Emily Dickinson

Mushrooms are often considered the vermin of the vegetable world, likened to snakes, slugs, and worms. Some are regarded as mystical and others as delicacies. The location of tasty morels is passed from generation to generation, closely guarded from strangers. Each autumn and spring, foragers scour the woods for known delicacies and new ones untried. Some mushroom foragers search for "little brown mushrooms," not for their taste, but to evoke hallucinations.

Eating unidentified or misidentified species can be dangerous. In the vast majority of toxic ingestions (perhaps up to 95%),

Nandina berry
Natal plum *(Carissa grandiflora)*
Norfolk Island pine *(Araucaria heterophylla)*
Old man cactus *(Cephalocereus senilis)*
Olive tree *(Olea europaea)*
Orchid *(Cattleya, Cymbidium, Oncidium)*
Oregon grape *(Mahonia aquifolium)*
Palm: Bamboo *(Chamaedorea erumpeus)*
Paradise *(Howea* [or *Kentia*] *forsterana)*
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Pellionia
Peony flower *(Paeonia)*
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Eating unidentified or misidentified species can be dangerous. In the vast majority of toxic ingestions (perhaps up to 95%),

the mushroom was incorrectly identified. More than 40,000 species of fungi are currently described, with a few thousand new ones added each year. Only a few species are toxic.

The fungi kingdom contains molds, smuts, rusts, mildews, yeasts, and mushrooms, which are different from plants because they lack chlorophyll. Yeasts are single-cell organisms that divide using budding. Mold is any fungus that grows with threadlike connections and a fuzzing appearance. Mildew, rust, and smut are all fungi related to mushrooms and break down vegetable matter, leaving behind a white, red, or black powder, respectively. *Toadstool* is often used to describe toxic mushrooms; this chapter differentiates toxic mushrooms by their toxins and scientific names.

The body of a fungus is a dense network of branching filaments, or hyphae. The mushroom is the fruiting body of the fungus, containing the spores. The hyphae and mycelia generally occur in an underground network supporting the visible mushroom. Mushrooms often grow in large rings radiating from a central network of mycelia. In the past, these "fairy rings" were thought to have mythical influence. Recently a fairy ring in northeast Oregon has been found to occupy an area over 10 square kilometers (6 square miles). This ring is thought to be between 2000 and 8000 years old, making it the oldest and largest single organism on the planet. Fungi are largely saprophytic (i.e., growing on decaying vegetable matter), involved with the decomposition of rotting materials, usually wood. They can also be parasitic (i.e., living on another living organism, injuring the host) or symbiotic (i.e., living together with each benefiting), living on viable materials with or without beneficial effect to the host. Some emerge only after significant environmental changes, such as the large quantity of morels that may be found where a forest fire has recently occurred.

As a mushroom emerges from the ground, it is covered with a membrane or veil (Fig. 59-1). As the mushroom grows (Fig. 59-2), the membrane breaks, leaving residual marks known as *warts* on the cap of the mushroom. These warts may remain firmly attached to the mushroom, or may remain as only residual spots, depending on the species of mushroom and environmental conditions. The emerging cap takes on a shape consistent with the specific species, ranging from cylindrical to convex to funnel shaped.

Gills located under the cap contain the spore-producing bodies. Some gills are covered with a second membrane or partial veil, which later pulls away to form an annulus, or ring, midway down the stalk of the mushroom. Gills may be attached firmly to the stalk, even running down the stalk, or only to the cap itself (free gills) (Fig. 59-3). Attachment of the gills is an important aid to identification of some poisonous mushrooms, such as *Amanita phalloides* (Fig. 59-4).

The stalk (stipe) begins at the cap and ends either underground or in a cup (vulva). A vulva at or just below ground level often is seen in a poisonous species. The stipe is generally located in the center of the cap and may or may not be tapered. The stipes of many poisonous species enlarge below the cap, ending in a bulb. The stipe may have a ringed membrane as evidence that the partial veil formerly protected the gills. Spores are produced by spore-forming bodies on the gills and expelled into the air after they mature.

Spores vary in size, color, and shape but are usually unicellular. They average 5 to 10 µm in diameter. Spores are useful in identifying mushroom species (Box 59-1). They can be obtained by cutting the stipe of a fresh specimen close to the gills, then laying the cap gill-side down on white paper for a few hours at room temperature. The initial color seen after removal of the gills is used for identification. With drying, the color may fade or change. Additional information about spores can be acquired by staining with Melzer's reagent (a solution of iodine and chloral hydrate). Spores that stain blue are called amyloid, indicating the presence of starch (Fig. 59-5). This technique may be particularly useful in spore identification from gastric aspirates. Spores of *Amanita* species are amyloid. Thin-layer chromatography of spores available from a mycology lab, mushroom farm, or at times from a botany department is a more accurate aid to identification.

There are many species of mushrooms, including several that are hunted, that have neither caps, gills, nor stipes. They have developed alternative methods of releasing their spores. Some of the "puffball" mushrooms are well known by the cloud of spores they release through a pore on the top surface of their spherical fruiting bodies (Fig. 59-6). This spore release occurs when the spores are mature and may be initiated by a falling branch, the errant placement of a deer's hoof, or the squeezing fingers of a curious child.

Mushrooms contain approximately 90% water and 3% proteins and other nitrogen-containing compounds. The remainder is largely carbohydrate, fat, and a few vitamins. Nutritionally unimpressive, mushrooms are consumed in great quantities, primarily for their taste and texture. Wild mushrooms have the additional allure of being free. This is changing in many parts of the northwestern United States, where some species of mushroom have become so profitable that pickers have had to buy commercial permits, and gun battles have erupted over one picker infringing on another's territory.

Of the many varieties of wild mushrooms, few are deadly or cause serious pathophysiologic derangement. All mushrooms, including toxic ones, are safe to handle without gloves. Handwashing afterward is sufficient to provide protection. Many experts will even bite off and chew a small piece of mushroom to gather taste information before spitting it out. As in wine tasting, a thorough rinsing is recommended after each taste.

Many immigrants fail to realize that the nontoxic mushrooms from their native lands have toxic look-alikes in America. This has been particularly true of Southeast Asian immigrants, who are attracted to the large *Amanita* species. Entire families have been poisoned, with many fatalities. The Russian roulette played by mushroom foragers is statistically safe. Some self-proclaimed experts are simply lucky; occasionally, they are not.

NONTOXIC MUSHROOMS

The most common commercially available mushroom in the United States is *Agaricus bisporus* (Fig. 59-7). It is cultivated in abandoned mine shafts and caves. This small white mushroom with dark gills is often picked before the gills are fully exposed. Although the mushroom is considered nontoxic, hypersensitivity reactions and gastrointestinal (GI) symptoms have been reported. In some parts of the United States, close relatives of this common mushroom account for the largest percentage of toxic mushroom cases, usually causing GI disturbance. *Agaricus* species may be confused with the deadly *Amanita* species (Fig. 59-8). The popular portabello mushroom is a type of *Agaricus bisporus*. *A. bisporus* can also be found in the wild.

Nontoxic mushrooms may carry environmental toxins, such as heavy metals and pesticides. Mushrooms with high lead con-

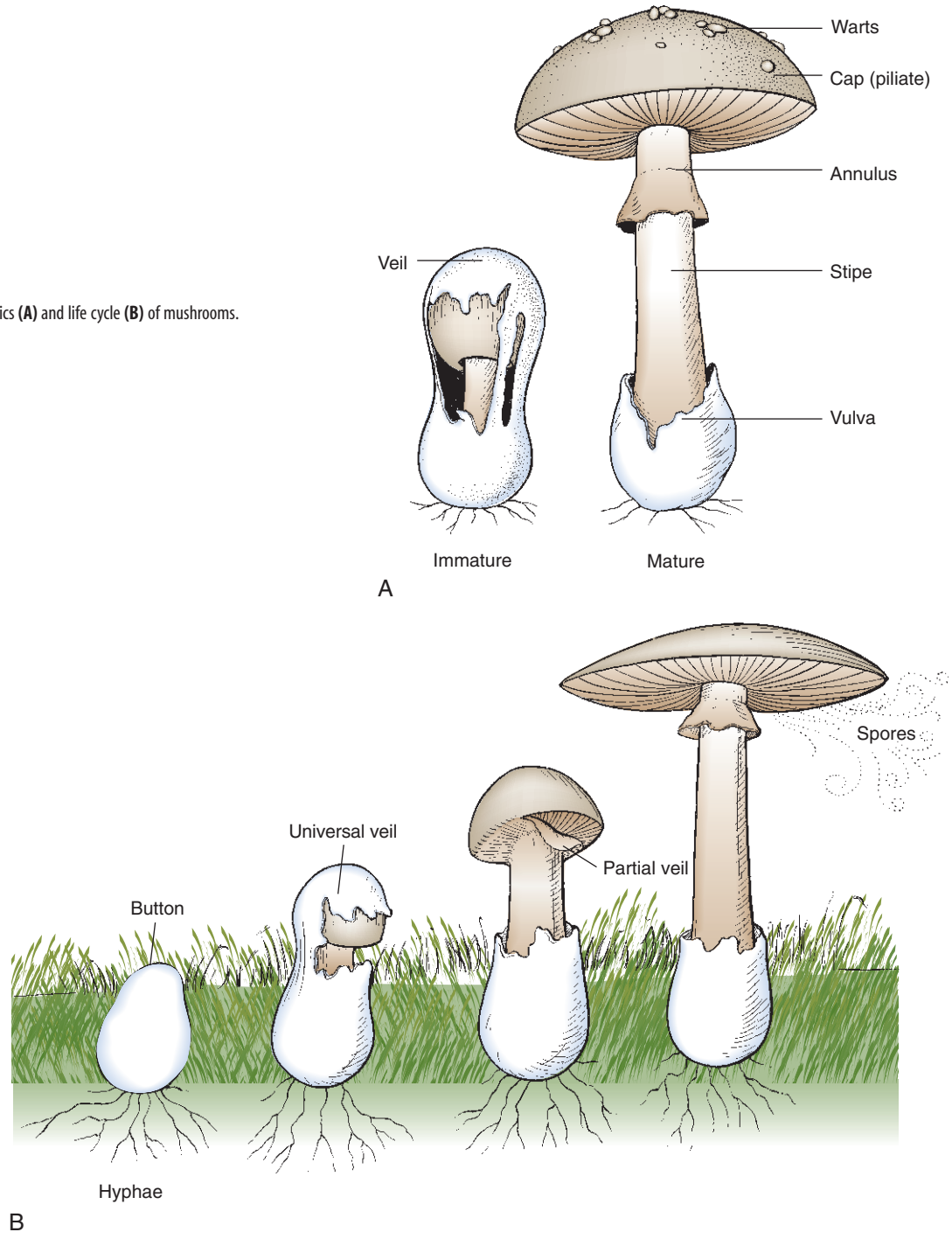

Figure 59-1. Structural characteristics **(A)** and life cycle **(B)** of mushrooms.

centrations have been gathered near highways.[35] High mercury concentrations are found in mushrooms from industrial sites.[22] Many mushrooms fruit among cultivated plants and may contain toxic levels of pesticides. Human toxicity has not been reported.

Fungi may cause allergic reactions. Molds that grow in damp locations in buildings have been suspected to cause a variety of patient complaints. They are one cause of the "sick building" syndrome that has resulted in some structures being vacated and sometimes demolished when the problem could not be remedied by conventional methods. Acute anaphylaxis from mushroom ingestion is rare, despite the presence of haptens capable of inciting an allergic response.[56] More often, symptoms develop from inhalation of spores.[70] Victims may present with anaphylaxis or, more commonly, with chronic hypersensitivity pneumonitis. Hypersensitivity reactions are described in workers

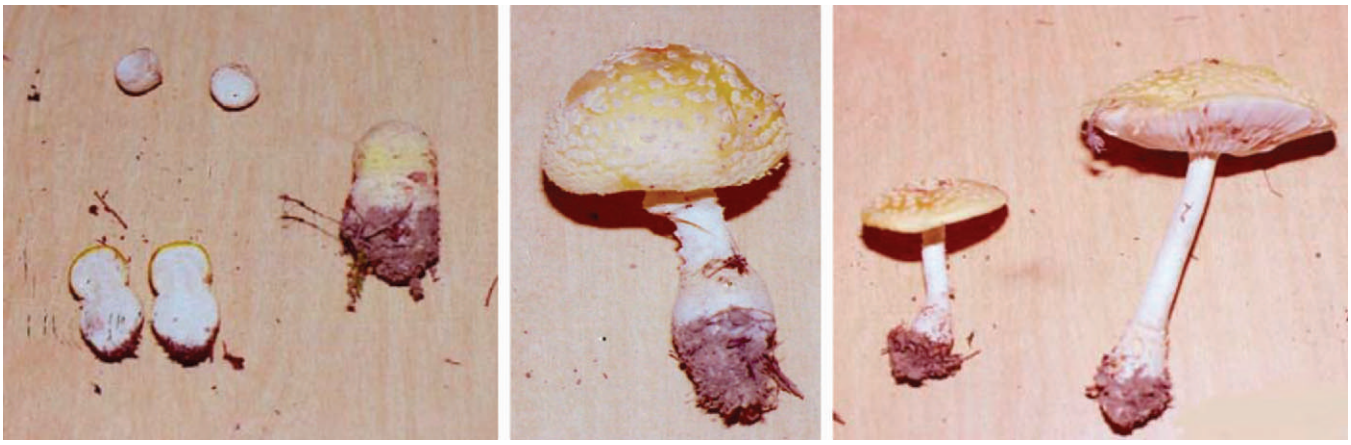

Figure 59-2. Growth of an *Amanita* species.

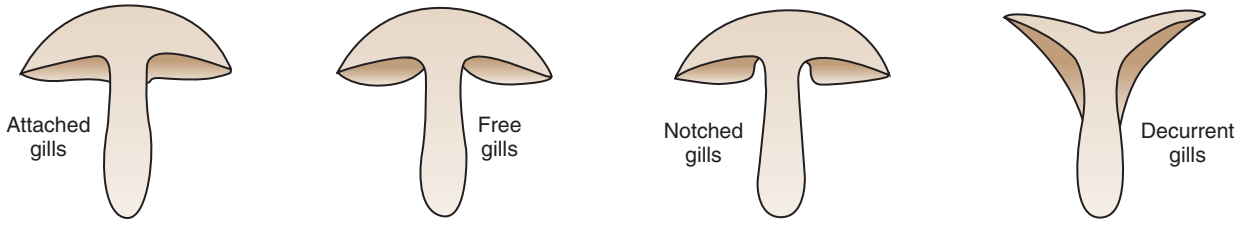

Figure 59-3. Mushroom gill types.

Figure 59-4. Death cap *(Amanita phalloides).*

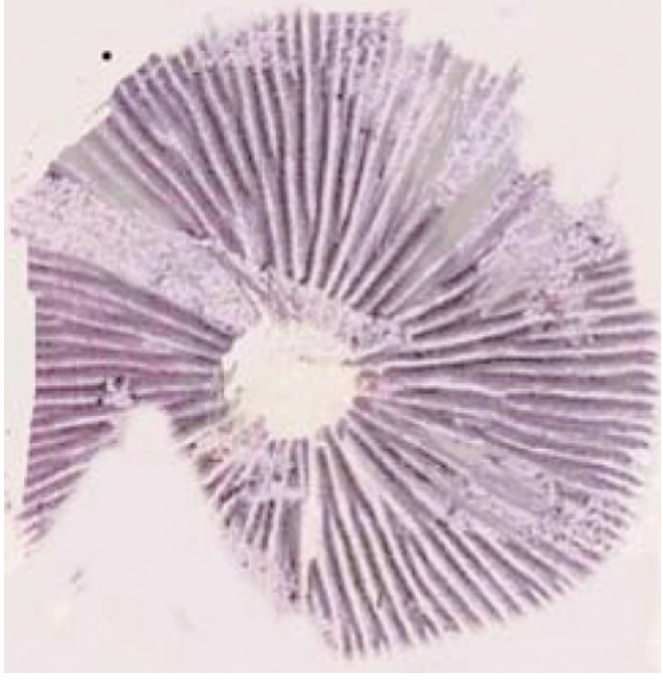

Figure 59-5. Spore print staining positive for amyloid.

exposed to cultivation of *A. bisporus* (the most popular commercially grown mushroom in America)[61] and shiitake *(Lentinus edodes),* the popular Japanese mushroom (Fig. 59-9).[84] Asthma symptoms developed in nearly 10% of shiitake-exposed workers. In one study, all workers had positive skin and inhalation challenge tests.[92] Spore counts correlate with asthma symptoms.

GI symptoms after ingestion of mushrooms may not be due to toxins. Bacterial food poisoning may occur in foods that coincidentally contain mushrooms. Small bowel obstruction occurred in a person who consumed 500 g of the edible mushroom *Cantharellus cibarius* (chanterelle) (Fig. 59-10).[36] This

Figure 59-6. Puffball mushrooms (edible).

Figure 59-8. *Amanita rubescens.*

Figure 59-7. *Agaricus bisporus*—edible.

Figure 59-9. Shiitake mushroom.

was largely a result of poor mastication, since entire mushrooms were recovered from the victim's intestines.

Most wild mushrooms are nontoxic, and many are delicious. Morels (*Morchella esculenta* or *deliciosa*) are highly prized delicacies. Chanterelles *(Cantharellus cibarius)* (see Figure 59-10) and several species of *Boletus* (Fig. 59-11) are particularly tasty. The chicken mushroom *(Laetiporus sulphureus)* (Fig. 59-12) is often used in place of chicken in Chinese dishes.

TYPES OF MUSHROOM TOXICITY

Mushroom toxicity can be classified into several types, which are summarized in Box 59-2. Detailed discussions of each type are presented in the sections that follow. Box 59-3 provides a method for identifying what kind of mushroom may have caused a patient's illness.

GASTROINTESTINAL TOXINS

Most toxic mushrooms fall into the group of GI irritants. This large, heterogeneous group of mushrooms causes GI distress, consisting of nausea, vomiting, and diarrhea, beginning 1 to 2 hours after ingestion and resolving in 6 to 12 hours. Even *A. bisporus,* the common cultivated mushroom, may cause brief gastroenteritis in some individuals.[93] The mechanism is unknown.

Causative Mushrooms

A large number of unrelated mushrooms cause GI symptoms with varying host response (Box 59-4). *Chlorophyllum molyb-*

Figure 59-10. *Cantharellus cibarius* (edible).

Figure 59-11. *Boletus edulis* (edible).

Figure 59-12. Chicken mushroom, *Laetiporus sulphureus* (edible).

Box 59-1. Obtaining Spore Prints

1. Obtain a fresh specimen
2. Cut off stalk close to the gills
3. Lay cap, gill side down, on white paper for several hours at room temperature.
4. Note color of spores on paper immediately after removal of cap. Drying may alter their appearance.

Box 59-2. Types of Mushroom Toxicity

1. Gastrointestinal irritants
2. Disulfiram-like toxins
3. Neurotoxins
   Muscarinic
   Isoxazole derivatives
   Psilocybin-hallucinogenic
4. Protoplasmic
   Gyromitrin—hepatotoxic
   Amatoxin—hepatotoxic
   Orellinine—nephrotoxic

Box 59-3. Guide to Mushroom Identification

1. Collect any specimens left at home—preferably noncooked.
2. Collect fresh specimens from gathering site(s).
3. Transport and store mushrooms in paper bags.
4. Spores can be recovered from gastrointestinal fluid.
5. Note initial toxicity and time since ingestion. Note symptoms or lack of symptoms among others ingesting mushrooms.
6. Contact a regional poison information center for assistance in locating an expert in identification.
7. When symptoms are not consistent with identified species, consider that the victim might have ingested another type of mushroom.

dites (also known as *Lepiota morganii*) (Fig. 59-13) is the most frequently ingested toxic mushroom in America. Most persons who ingest *C. molybdites* confuse it with *A. bisporus*, which it closely resembles. The common name for *C. molybdites*, green-spored parasol, describes the characteristics of this summer mushroom. The whitish cap is large (10 to 40 cm [4 to 17 inches]), initially smooth and round, and becomes convex with maturity. Tan or brown warts may be present. The gills are free from the stalk, initially white to yellow, and become green with maturity. The stalk is 5 to 25 cm (2 to 10 inches) long, smooth, and white. The ring is generally brown on the underside. Spores are green. The mushroom is common in most of eastern and southern North America and in California. In southern California, it is a common lawn mushroom.

Another common mushroom causing GI symptoms is the jack-o'-lantern (Fig. 59-14). The botanical classification is not completely settled. Most commonly, it is referred to as *Omphalotus illudens*, *O. olearius*, or *O. olivascens*. The mush-

Box 59-4. Mushrooms Reported to Cause Gastrointestinal Irritation

AGARICUS
albolutescens
hondensis
placomyces
silvaticus
silvicola
xanthodermus

AMANITA
brunnescens (see Figure 59-15)
chlorinosma
flavoconia
flavorubescens (see Figure 59-16)
frostiana
parcivolvata
spissa
spreta
volvata

***BOLETUS* (see Figure 59-11)**
luridus
pulcherrimus
satanus
sensibilis

***CHLOROPHYLLUM* (see Figure 59-13)**
molybdites (*Lepiota morganii*)

***ENTOLOMA* (*RHODOPHYLLUS*)**
lividum
nidorosum
rhodopolium
salmoneum
strictius
vernum

***CANTHARELLUS* (see Figure 59-10)**
bonari
floccosus
kauffmanii

HEBELOMA
crustuliniforme
fastibile
mesophaeum
sinapizans

LACTARIUS
chrysorrheus
glaucescens
helvus
representaneus
rufus
scrobiculatus
torminosus
uvidus

***LEPIOTA* (see Figure 59-31)**
clypeolaria
cristata
lutea
morganii
naucina

LYCOPERDON
marginatum
subincarnatum

MORCHELLA
angusticeps
crassipes
deliciosa
esculenta
semilibera

NAEMATOLOMA (HYPHOLOMA)
fasciculare

***OMPHALOTUS* (see Figure 59-14)**
olearius
illudens
olivascens

PAXILLUS
involutus

RAMARIA (CLAVARIA)
formosa
gelantinosa

RUSSULA
emetica

SCLERODERMA
aurantium
cepa

TRICHOLOMA
album
muscarium
pardinum
pessundatum
saponaceum
sejunctum
sulphureum
venenata

VERPA
bohemica

room is a bright orange-yellow mushroom with sharp-edged gills and often grows in clusters at the base of stumps or on buried roots of deciduous trees. The cap is 4 to 16 cm (1.5 to 6 inches) in diameter on a stalk that is 4 to 20 cm (1.5 to 8 inches) long. Gills are olive to orange, with white to yellow spores. The mushroom shows characteristic luminescence lasting 40 to 50 hours after collection. Members of this family are found in both eastern and western North America, generally in autumn and early spring. They may be mistaken for the edible species *Cantharellus cibarius* (see Figure 59-10). Some European reports

Figure 59-13. *Chlorophyllum molybdites,* gastrointestinal irritant. (From Phillips R: Mushrooms of North America, Boston, Little, Brown, 1991.)

Figure 59-14. Jack-o'-lantern mushroom, *Omphalotus illudens,* gastrointestinal irritant. (From Phillips R: Mushrooms of North America, Boston, Little, Brown, 1991.)

have documented hepatic impairment and muscarinic effect following ingestion.[99] It is not clear whether the mushroom and its toxins are the same on both sides of the Atlantic.

Although the genus *Amanita* is most famous for its deadly member *Amanita phalloides* (see Figure 59-4), the genus also contains tasty nontoxic mushrooms (*A. caesarea, A. calyptrata,* and *A. velosa*). Several *Amanita* species cause GI symptoms indistinguishable from those caused by jack-o-lantern mushrooms or *Chlorophyllum molybdites*. *A. brunnescens* (Fig. 59-15) and *A. flavorubescens* (Fig. 59-16) are frequently listed as containing GI toxins, although they are occasionally listed as edible. Both have broad yellowish to brown caps (3 to 15 cm [1 to 6 inches]) with loosely attached warts. The stalks are 3 to 18 cm (1 to 7 inches) long, enlarging toward the base with a superior ring. *A. brunnescens* stains reddish brown when bruised. As with their edible cousins, these mushrooms are found in summer or fall associated with hardwoods or conifers.

Several members of the genus *Agaricus,* particularly *A. albolutescens, A. silvaticus,* and *A. xanthodermas,* can cause GI symptoms. They resemble the cultivated mushrooms in grocery stores and are found in meadows and lawns in the summer and autumn. Table 59-1 lists the look-alike toxic and nontoxic mushrooms in this group.

Figure 59-15. *Amanita brunnescens.*

Figure 59-16. *Amanita flavorubescens.*

Toxins

A variety of toxins have been extracted from these mushrooms, although their structures are poorly described. Most are protein-based and heat labile, although toxicity may not be completely eliminated with cooking. In some cases the toxin

TABLE 59-1. Gastrointestinal Irritant Mushrooms Mistaken for Edible Species

GASTROINTESTINAL IRRITANT	EDIBLE SPECIES
Agaricus *albolutescens* *sylvaticus* *xanthodermas*	*Agaricus bisporus*
Amanita brunnescens	*Amanita flavorubescens* *Amanita inaurata*
Chlorophyllum molybdites	*Lepiota species* *Agaricus bisporus*
Entoloma species	*Pluteus cervinus* *Entoloma abortivum*
Hebeloma crustuliniforme	*Rozites caperata*
Naematoloma fasciculare	*Armillariella mellea* *Naematoloma sublateritium* *Naematoloma capnoides*
Paxillus involutus	*Lactarius* species
Ramaria formosa *Ramaria gelatinosa*	*Ramaria* species
Scleroderma aurantium	*Lycoperdon perlatum*
Tricholoma pessundatum *Omphalotus olearius*	*Cantharellus cibarius* *Laetiporus sulphureus* *Armillaria mellea*

Figure 59-17. *Coprinus* mushroom.

may be destroyed by heating (temperature and duration vary by species), parboiling, or even preserving in salt. Host response to a toxin varies; some persons can eat such mushrooms without harm, while others become quite ill. Some mushrooms also contain hemolysins and toxins that cause hemorrhage and hepatitis in animals.[57,96] Human hemolysis has not been reported.[57]

Clinical Presentation

Within 1 to 2 hours of ingestion of these mushrooms, nausea, vomiting, intestinal cramping, and diarrhea develop. Stools are usually watery and occasionally bloody with fecal leukocytes. Chills, headaches, and myalgias may occur. Symptoms remit spontaneously in 6 to 12 hours. Most victims require only fluid and electrolytes replacement. A few serious cases reported in the literature have been associated with severe dehydration. In a review of 106 cases, all victims responded well to fluid and electrolyte replacement and occasional antiemetic or antidiarrheal medications.[23] Admitted patients are discharged in an average of 2 days. Persons whose symptoms are delayed (beginning 4 hours or more after ingestion) probably have ingested a more toxic mushroom, possibly *Amanita, Galerina, Lepiota,* or *Gyromitra*. These persons have severe GI distress and may develop hepatic failure. Most mushroom fatalities occur from ingestion of these mushrooms.

Recent reports of ingestion of jack-o-lantern mushrooms describe mildly elevated liver transaminases.[99] Cases of metabolic acidosis and dehydration, and even death, are attributed to *Chlorophyllum molybdites*.[14,95]

Treatment

Treatment is largely supportive (Box 59-5) and does not depend on the type of mushroom ingested. Intravenous fluid and electrolytes replacement may be required. In a severe case, an antiemetic such as prochlorperazine, 5 mg intravenously (IV) or 10 mg intramuscularly (IM), may prevent further emesis though there is no evidence for its use. Activated charcoal (1 g/kg) without cathartic can be given orally or through a nasogastric tube within 1 hour of ingestion. There is no evidence however that it decreases toxicity. Once symptoms start, charcoal is likely useless. Although antimotility drugs can be administered, there is no evidence that they are effective. Most cases are self-limited.

Care should be taken not to dismiss early GI symptoms when several types of unknown mushrooms have been ingested. Individuals may ingest both GI irritant mushrooms and mushrooms containing serious toxins. Persons with prolonged gastroenteritis from unidentified mushrooms should be observed for 24 to 48 hours for development of delayed hepatic damage. Special efforts should be made to identify the ingested mushrooms.

Box 59-5. Treatment of Gastrointestinal Irritant Mushroom Ingestion

1. Note time since ingestion. If more than 2 hours, consider a more serious protoplasmic toxin ingestion.
2. Fluid and electrolyte replacement.
3. Anti-emetic and antidiarrheal medication as needed.

DISULFIRAM-LIKE TOXINS

A fascinating toxicity is caused by some members of the *Coprinus* genus, known as "inky caps" (Fig. 59-17 and Fig. 59-18). Individuals who ingest these mushrooms and subsequently ingest alcohol have symptoms similar to those of an alcohol-disulfiram (Antabuse) reaction.

Figure 59-18. *Coprinus* mushrooms.

Box 59-6. Mushrooms Suspected or Reported to Cause an Alcohol-Disulfiram Reaction

***COPRINUS* (see Figure 59-17 and Figure 59-18)**
atramentarius
comatus
insignis
micaceus

***CLITOCYBE CLAVIPES* (see Figure 59-22)**

Causative Mushrooms

Several members of the *Coprinus genus* may contain disulfiram-like toxins (Box 59-6), but symptoms are most common with *C. atramentarius*. The mushroom has a 2- to 8-cm (0.8- to 3.1-inch) cylindrical cap on a thin 4- to 5-cm (1.5- to 2-inch) stalk. The cap is white or occasionally orange or yellow at the top. The mature cap often develops cracks, which turn up at its margins. The cap blackens as it matures and then liquefies into the "inky cap." A ring may be present low on the stalk. Spores are black. *C. atramentarius* grows throughout North America in clusters of three or more in grass or wood debris. It often appears overnight after a rain. Several members of the *Coprinus* genus, including *C. atramentarius*, are edible if no alcohol is ingested for the next 72 hours.

Toxin

Mushrooms causing a disulfiram reaction contain the toxin coprine, isolated from the mushroom *C. atramentarius* in 1975.[43] Coprine is distinct from disulfiram and is most likely a derivative of glutamine. It probably is not present in the raw mushroom, but rather a hydrolyte created during cooking.[60]

Box 59-7. Treatment of Coprine Toxicity (Disulfiram-Like Toxin)

1. Generally requires only supportive care.
2. Hypotension, if present, responds to fluid or, if necessary, intravenous norepinephrine (4 to 8 µg/min or 1 to 2 µg /min in children, increasing as needed every 5 to 10 minutes).
3. Supraventricular tachycardia can be controlled with intravenous propranolol (0.5 to 3 mg or 0.01 to 0.02 mg/kg in children up to a maximum of 1 mg/dose, repeated after 5 to 10 minutes as needed) if severe.

Coprine (or its derivative L-aminocyclopropanol) inhibits acetaldehyde dehydrogenase, similar to the action of disulfiram. Acetaldehyde accumulates, leading to β-adrenergic stimulation and the typical vasomotor response of flushing, diaphoresis, headache, tachycardia, nausea, and vomiting. Some authors believe that coprine is a relatively poor inhibitor of acetaldehyde dehydrogenase and suggest that symptoms result from altered neurotransmitter levels.[72]

Clinical Presentation

A history of wild mushroom ingestion within days prior to symptoms is rarely offered. Ingestion of the mushroom imparts sensitivity to alcohol, which begins 2 to 6 hours after ingestion and may last up to 72 hours. Within 15 to 30 minutes of subsequent alcohol ingestion the victim experiences severe headache, flushing, and tachycardia. Hyperventilation, shortness of breath, and palpitations may occur. Chest pain and orthostatic hypotension occur in severe cases. Symptoms can be confused with an allergic reaction or acute myocardial infarction. Symptoms resolve spontaneously within 3 to 6 hours.

Treatment

Supportive and symptomatic treatments are suggested (Box 59-7). Baseline laboratory tests (blood urea nitrogen [BUN], creatinine, electrolytes, and glucose) should be drawn. Urine output should be monitored. Intravenous fluids should be given to keep urine output at 50 mL/hr (in children 1 mL/kg/hr). Activated charcoal is not beneficial. Charcoal does not absorb alcohol, and coprine has already been absorbed by the time the reaction occurs. Hypotension generally responds to intravenous fluid administration. Severe hypotension refractory to fluid replacement should be treated with norepinephrine (initially 4 to 8 µg/min; 1 to 2 µg/min in children and increased as necessary) rather than an indirect vasopressor, because norepinephrine stores are depleted in a true disulfiram reaction. Propranolol (0.5 to 3 mg IV; 0.01 to 0.02 mg/kg IV up to 1 mg in children) has been suggested for the treatment of supraventricular tachycardia. Propranolol may be repeated as needed after 5 to 10 minutes.

NEUROLOGIC TOXINS

Muscarine

Muscarine was first isolated from the mushroom *Amanita muscaria* (Fig. 59-19 and Fig. 59-20) over 150 years ago. A classic muscarinic reaction includes salivation, lacrimation, urination, diaphoresis, GI upset, and emesis (SLUDGE syndrome). Bud-

Figure 59-19. *Amanita muscaria.*

Figure 59-20. *Amanita muscaria.*

dhist adepts may have used *Amanita muscaria* in the 2nd to 9th centuries to achieve enlightenment.[40]

Causative Mushrooms

Amanita muscaria has a cap 5 to 30 cm (2 to 12 inches) in diameter that is scarlet red with white warts. The stalk tapers upward and is white, often hollow, and grows 15 to 20 cm (6 to 8 inches) in length. It has a prominent vulva and numerous rings. Gills are free and white, as are spores. The mushrooms grow in eastern North America and throughout much of the western United States, often near *Boletus edulis*. They grow under hardwoods and conifers from spring to autumn.

Potentially toxic amounts of muscarine are found in some *Inocybe* and *Clitocybe* mushrooms (Box 59-8 and Table 59-2). *Inocybe* mushrooms (Fig. 59-21) are small brown mushrooms with conical caps up to 6 cm (2.5 inches) in diameter. Stalks are 2 to 10 cm ($^3/_4$ to 4 inches) long, covered with fine brown to white hairs. Gills are brown and notched; spores are brown. They are found typically under hardwoods and conifers in the summer and fall. All members of this family are considered poisonous.

In contrast, many *Clitocybe* mushrooms (Fig. 59-22) are edible, but except for *C. nuda* and its close relatives, they are not worth pursuing. A few contain muscarine. All *Clitocybe* mushrooms are whitish-tan to gray mushrooms with caps 15 to 33 mm (0.6 to 1.3 inches) long on hairless stalks 1 to 5 cm ($^1/_2$ to 2 inches) long. Gills are decurrent (run down the stalk) and spores are white. They are usually single specimens (not clustered) found on lawns in summer and fall.

Many *Inocybe* and *Clitocybe* mushrooms contain larger concentrations of muscarine than does *A. muscaria*. Other toxins (e.g., ibotenic acid) are present in *A. muscaria* and contribute to its toxicity.

Box 59-8. Mushrooms Reported or Suspected to Contain Muscarine

AMANITA
gemmata
muscaria (see Figure 59-19 and Figure 59-20)
pantherina (see Figure 59-23)
parcivolvata

BOLETUS
calopus
luridus
pulcherrimus
satanus

***CLITOCYBE* (see Figure 59-22)**
aurantiaca
dealbata
nebularis

HEBELOMA CRUSTULINIFORME

INOCYBE
fastigiata
geophylla
napipes
patouillardii
pudica

MYCENA PURA

***OMPHALOTUS* (see Figure 59-14)**
olivascens
olearius
illudens

TABLE 59-2. Look-Alikes of Mushrooms Causing Muscarine Poisoning

TOXIC SPECIES	EDIBLE SPECIES
Clitocybe dealbata	*Marasmius oreades*
Inocybe species (see Figure 59-21)	*Marasmius oreades*

Figure 59-21. *Inocybe* mushrooms—some toxic.

Figure 59-22. *Clitocybe* mushrooms—some toxic.

Toxin

Muscarine is a quaternary trimethyl ammonium salt of 2-methyl-3-oxy-5-(amino)tetrahydrofuran. Muscarine stimulates postganglionic cholinergic receptors (muscarinic receptors), mimicking the action of acetylcholine. Muscarine stimulation of the GI tract leads to increased secretory activity, contraction amplitude, and peristalsis. Stimulation of the urinary tract leads to bladder contraction and increased peristalsis of the ureters. Stimulation of secretory tissue leads to salivation and lacrimation. Bronchoconstriction, flushing, and diaphoresis result from additional stimulation of bronchial and vascular tissues. Cardiac effects include reflex tachycardia, or more often bradycardia and decreased atrioventricular conduction. Central nervous system (CNS) effects include headache, ataxia, and visual disturbances. The sensorium is generally not affected (except in ingestion of *A. muscaria,* which contains other CNS toxins).

Box 59-9. Treatment of Muscarinic Toxicity

1. Supportive care with oxygen, suctioning, and endotracheal intubation as needed.
2. Fluid and electrolyte replacement.
3. Atropine (if symptoms are life threatening) 0.01 mg/kg IV every 5 to 10 minutes until secretions are controlled. Atropine may worsen central nervous system effects.

Clinical Presentation

Symptoms develop within 15 to 30 minutes after ingestion of muscarine-containing mushrooms. Typical symptoms include salivation, urination, lacrimation, diarrhea, diaphoresis, abdominal pain, nausea, and emesis. Bradycardia, bronchospasm, and constricted pupils are also noted. Copious bronchial secretions may cause respiratory failure requiring mechanical ventilation.

Symptoms remit spontaneously in 6 to 24 hours. In Europe, some deaths are reported from *Inocybe patouillardii.*[60] Although this mushroom is rarely eaten, ingestion carries a mortality rate of 6% to 12%, particularly among persons with preexisting pulmonary or cardiac disease. Most deaths occur within the first 12 hours as a result of respiratory failure or cardiovascular collapse.

Treatment

Treatment is outlined in Box 59-9. Many victims require supportive care with oxygen, suctioning, and intravenous fluid replacement. Activated charcoal and cathartics are rarely given because of the prominent emesis and diarrhea. Atropine is a muscarine antagonist but should be used only to control secretions or profound bradycardia. It should not be given prophylactically or for other symptoms because it may worsen the CNS effects induced by *A. muscaria.* Atropine (0.01 mg/kg IV for children and 1 mg IV for adults) can be repeated as needed until secretions are manageable. Symptoms resolve spontaneously within 24 hours.

Isoxazole Reactions

The isoxazole derivatives ibotenic acid and muscimol produce CNS symptoms, including excitement and alteration in visual perception.

Causative Mushrooms

Several mushrooms contain ibotenic acid (Box 59-10 and Table 59-3). *Amanita muscaria* is described previously. *Amanita pantherina* (Fig. 59-23) is 5 to 15 cm (2 to 6 inches) long with a cap 5 to 15 cm (2 to 6 inches) in diameter. The cap is generally white to pink in the young specimen, but becomes reddish brown or brown, often darker at the rim, with maturity. Fragments of the universal veil form warts on the cap but may be washed off by rain. The stalk has a distinct ring, with a vulva at the bottom. When the flesh is cut or injured (e.g., by insect larvae), it develops a pinkish tinge. Gills are free and produce white spores. The raw mushroom has little smell and tastes very much like a raw potato. It grows from June to November in woodlands throughout North America.

Figure 59-23. *Amanita pantherina,* which contains the neurotoxins ibotenic acid and isoxazole derivatives. (From Phillips R: Mushrooms of North America, Boston, Little, Brown, 1991.)

Box 59-10. Mushrooms Reported or Suspected to Contain Ibotenic Acid, Muscimol, and Related Compounds

AMANITA
cokeri
gemmata
muscaria (see Figure 59-19 and Figure 59-20)
pantherina (see Figure 59-23)

PANAEOLUS CAMPANULATUS

TRICHOLOMA MUSCARIUM

TABLE 59-3. Look-Alikes of Mushrooms Containing Isoxazole Toxins

TOXIC SPECIES	EDIBLE SPECIES
Amanita muscaria (see Figure 59-19 and Figure 59-20)	*Amanita caesarea* *Amanita rubescens* (see Figure 59-8) *Armillariella mellea*
Amanita pantherina (see Figure 59-23)	*Amanita rubescens* (see Figure 59-8)
Amanita gemmata	*Russula* species

Toxin

Ibotenic acid is found in the bright red cap of *Amanita muscaria* and undergoes decarboxylation during drying to form muscimol, the more toxic of the compounds. The potency of the cap remains high despite drying. Muscimol is a γ-aminobutyric acid (GABA) receptor agonist.[47] Muscimol increases CNS serotonin levels and decreases catecholamine levels. Ibotenic acid resembles GABA and in animals can act as it does.

Clinical Presentation

Ingestion of 10 mg of *A. muscaria* produces mild intoxication, dizziness, and ataxia. Ingestion of 15 mg leads to pronounced ataxia and visual disturbances.[37,66] Delirium or manic behavior may develop. Physical activity is accelerated, with inability to judge size. Visual hallucinations, seizures, and muscle twitching are common. Some victims complain of residual headache for up to 48 hours.

Symptoms begin within 30 minutes of ingestion and generally last two hours. Rare fatalities have been reported. Some persons had ataxia and paralysis of ocular convergence.[37] In rare cases, symptoms last as long as 48 hours, depending on the dosage and individual host effect.

Box 59-11. Treatment of Isoxazole Toxins or Psilocybin Toxin

1. Supportive care.
2. Sedation as needed with benzodiazepine (diazepam 2 to 5 mg IV every 10 minutes as needed) or phenobarbital (30 mg IV hourly).
3. If hyperpyrexia occurs (primarily in children), consider external cooling.

Treatment

Treatment consists of supportive care (Box 59-11). Emesis caused by the toxin is unusual. Gastric emptying and charcoal administration is difficult because of the CNS disturbances and have no proven effectiveness. Appropriate sedation with phenobarbital (30 mg IV hourly in adults and 0.5 mg/kg in children) or diazepam (5 mg repeated IV every 10 to 15 minutes in adults as needed, 0.1 to 0.3 mg/kg IV in children) is often necessary but requires caution. Phenobarbital or diazepam administered to treat *A. muscaria* ingestion may lead to unexpected apnea, flaccid paralysis, or both. Airway support and ventilatory assistance should be immediately available. Atropine may worsen the CNS symptoms associated with isoxazole derivatives and should therefore be withheld unless the muscarinic effects are serious.

Hallucinogenic Mushrooms

Perhaps the most sought-after mushrooms are "magic mushrooms," which are available in the wild and as spores available through mail order catalogs. These mushrooms have been used for centuries because of their hallucinogenic effects. Small stone mushroom icons believed to be 3500 years old were found in Meso-American ruins.[60] Recently, honey laced with *Psilocybe cubensis* was confiscated at the Dutch-German border.[8] Honey with *Psilocybe* can be purchased at Dutch coffee shops.

Causative Mushrooms

The most common hallucinogenic mushrooms are species of the *Psilocybe* genus (Box 59-12 and Table 59-4). This genus includes over 100 species, not all of which cause hallucinations. These are "little brown mushrooms" (LBMs) (Fig. 59-24). The cap is 0.5 to 4 cm (0.2 to 1.6 inches) in diameter (depending on the species), is usually smooth, and becomes sticky or slippery when wet. The stalk is slender and 4 to 15 cm (1.5 to 6 inches) long. Gills are gray to purple gray; spores are dark, nearly black. The flesh of these mushrooms often turns blue or greenish when bruised or cut. The mushrooms are often mistaken for more poisonous species (e.g., *Galerina* or *Inocybe* [see Figure 59-21]). These mushrooms also resemble *Agaricus bisporus*. Regular grocery store mushrooms laced with lysergic acid diethylamide

Box 59-12. Mushrooms Reported or Suspected to Contain Either Psilocybin or Psilocin or Both

AMANITA
citrina
porphyria

CONOCYBE
cyanopus
siligineoides
smithii

***GYMNOPILUS* (see Figure 59-26)**
aeruginosus
purpuratus
spectabilis
validipes

NAEMATOLOMA POPPERIANUM

***PANAEOLUS* (see Figure 59-25)**
campanulatus
castaneifolius
cyanescens
fimicola
foenisecii
phalaenarum
semiovatus
sphinctrinus
subbalteatus

PSATHYRELLA SEPULCHRALIS

***PSILOCYBE* (see Figure 59-24)**
baeocystis
caerulescens
caerulipes
cyanaescens
cubensis
pelliculosa
semilanceata
strictipes
stuntzii

STROPHARIA
aeurginosa
coronilla
hornemannii
squamosa

TABLE 59-4. Look-Alikes of Mushrooms Containing Psilocybin or Psilocin

TOXIC SPECIES	EDIBLE SPECIES
Panaeolus foenisecii	*Psathyrella candolleana* *Agrocybe pediades* *Marasmius oreades*
Panaeolus species (see Figure 59-25)	*Coprinus* species (see Figure 59-17 and Figure 59-18)

Figure 59-24. *Psilocybe caerulipes*.

Figure 59-25. *Panaeolus* mushroom—some toxic.

(LSD) or other hallucinogens are sold on the street as *Psilocybe*. Hallucinogenic mushrooms grow in a variety of habitats and are found throughout the world.

Other mushrooms, including members of the *Panaeolus* and *Gymnopilus* genera (Fig. 59-25 and Fig. 59-26) may contain psilocybin. *Panaeolus* mushrooms are also LBMs about the same size as *Psilocybe*. Gills are dark gray or black with black spores. They grow on dung throughout the tropics and subtropics of North America. Unlike *Psilocybe*, their caps do not become sticky or slippery when wet. The hallucinogenic effect and quantity of toxin varies among *Panaeolus* species.

Some *Gymnopilus* species (e.g., *G. aeruginosus*) contain hallucinogens. These medium-sized mushrooms (cap 5 to 15 cm [2 to 6 inches]; stalk 5 to 12 cm [2 to 5 inches]) are variable in color (green, yellow, salmon, and red), with yellowish gills and rusty spores. They grow on stumps or sawdust in the Pacific Northwest.

Visual hallucinations and ataxia were reported in a person ingesting *Laetiporus sulphureus* (see Figure 59-12), previously thought to be harmless.[2] It is not clear whether this mushroom contained hallucinogenic material or the individual ingested an additional mushroom as well.

Toxin

Psilocybin and its somewhat unstable metabolite psilocin are indole compounds derived from tryptamine. These two toxins

Figure 59-26. *Gymnopilus* mushroom—some toxic.

were first isolated by Albert Hofmann,[45] known as the father of LSD. Chemically, the toxins resemble 5-hydroxytryptamine and LSD and have similar effects. They maintain their potency in dried specimens.

Psilocybin, as well as LSD, inhibits the firing rate of serotonin-dependent neurons, particularly at the presynaptic receptors. It induces euphoria, hallucinations, and loss of time sensation. Many of these symptoms are similar to that seen with LSD. Some species contain phenylethylamine, which may be responsible for tachycardia and other adverse reactions.[4] In human volunteer studies, peak plasma levels were reached at 105 minutes.[42]

Clinical Presentation

Ingestion of 5 mg of psilocybin (10 mg of fresh *P. cubensis*) causes moderate euphoria. Ingestion of 10 mg leads to hallucinations and loss of time sensation. Heightened imagination occurs within 15 to 30 minutes of ingestion. Hallucinations may last for 4 to 6 hours. Serious side effects are rarely seen, but fever and seizures have been reported in children.[65] Up to 50% of victims have tachycardia and hypertension.[76] A case resulting in myocardial infarction has been reported.[9] Flashbacks have been reported to occur in some victims for up to 4 months after ingestion.[6] Multifocal cerebral demyelination has been reported after ingestion of psilocybin-containing mushrooms.[94]

Treatment

Recommendations are similar to those for isoxazole toxin (see Box 59-11). Initially, the victim should be placed in a quiet supportive environment. Gastric emptying and activated charcoal administration are often impossible and may only enhance the hallucinations. They should be considered in victims with very large ingestions who are brought for treatment early. Sedation must be used with caution as it may worsen the effects. Sedation can be accomplished when necessary with a benzodiazepine (diazepam, 0.1 mg/kg IV in children, or 5 mg IV in adults, repeated every 5 minutes as needed), phenobarbital (0.5 mg/kg IV in children, 30 to 60 mg IV in adults, repeated every 10 minutes as needed), chlorpromazine (50 to 100 mg IM), or haloperidol (5 mg IM). There is no evidence for the use of newer antipsychotic medications. Seizures can be controlled with diazepam in the doses just listed. Hyperpyrexia, seen primarily in children, is best treated with external cooling.

Figure 59-27. *Gyromitra esculenta,* which contains the hepatoxin gyromitrin. (From Phillips R: Mushrooms of North America, Boston, Little, Brown, 1991.)

PROTOPLASMIC POISONS

Gyromitra Toxin

False morels (*Gyromitra esculenta* [Fig. 59-27]) were once thought to be edible. During times of famine in Europe, ingestion of *Gyromitra* was encouraged.[41] Since 1793, they have been suspected of causing toxicity, and since World War II have been known to cause hepatic failure, neurologic symptoms, and death. Even today they are collected, sold fresh, canned, and exported ("morschels") in eastern and central Europe.[68] Symptoms appear 4 to 50 hours (usually 5 to 12 hours) after ingestion, with timing similar to that of *A. phalloides* poisoning. Since *Gyromitra* grows primarily in the spring and *A. phalloides* in the fall, the identity of the two mushrooms is rarely confused.

Causative Mushrooms

Gyromitra esculenta (see Figure 59-27) is approximately 5 to 16 cm (2 to 6 inches) in height with a reddish-brown to dark brown, irregularly shaped cap. The cap's surface is curved and folded, resembling a brain. The stalk is often as thick as the cap. The insides of the cap and stalk are hollow. This mushroom grows in the spring near pines and in sandy soil throughout North America. It is particularly common in Germany, Poland, and other eastern European countries. Mature species, particularly those with cap decay, may have increased toxicity. These mushrooms may be mistaken for morels, which are considered among the most delicious wild mushrooms (Table 59-5).

Other members of the *Gyromitra* family contain gyromitrin. None of these mushrooms has been reported to cause toxicity in humans.

Toxin

Gyromitrin (*N*-methyl-*N*-formylhydrazone) was first isolated in 1967. This toxin is moderately volatile and heat sensitive.

TABLE 59-5. Look-Alike of Mushrooms Containing Gyromitrin

TOXIC SPECIES	EDIBLE SPECIES
Gyromitra esculenta (see Figure 59-27)	*Morchella esculenta*

Box 59-13. Symptoms of Gyromitrin Toxicity

1. Onset of nausea, vomiting, and diarrhea within 5 to 12 hours.
2. Neurologic symptoms of dizziness, weakness, and loss of muscle coordination. Severe neurologic symptoms include coma, delirium, and seizures.
3. Hepatic failure begins 3 to 4 days after ingestion. Hepatic failure is often associated with hypoglycemia.

Cooking thoroughly and discarding the cooking liquid may decrease or eliminate the toxin. Symptoms have occurred despite proper cooking.

Once gyromitrin enters the stomach, hydrolysis yields *N*-methyl-*N*-formylhydrazine (MFH), which forms *N*-methylhydrazine or monomethylhydrazine (MH),[68] a component of rocket fuel. MH is a competitive inhibitor of pyridoxal phosphate, which interferes with enzyme systems (including decarboxylases, deaminases, and transaminases) requiring pyridoxine as a cofactor.[3] As a result, levels of GABA fall, interfering with neurotransmission.[55] This may lead to altered mental status, seizures, or both. Recent questions have been raised about this widely accepted theory, since MH may cause seizures without a change in brain GABA levels.[64]

MFH and MH undergo oxidation in the liver into two highly reactive intermediates: a free methyl radical and an unstable diazonium compound.[68] These substances appear to produce local hepatic necrosis by blocking the activity of hepatic cytochrome enzyme systems, glutathione, and other hepatic biomolecules. There are significant concerns about long-term toxicity associated with repeated consumption of this mushroom. Each kilogram of fresh *Gyromitra esculenta* contains 3 to 100 mg gyromitrin. Fresh mushrooms contain 50 to 300 mg of MH per kg, and dried mushrooms up to 400 mg per kg. The human LD_{50} is suspected to be 20 to 50 mg/kg for adults and 10 to 30 mg/kg for children, or 0.4 to 1 kg of fresh mushrooms for an adult (average 30 mushrooms) and 0.2 to 0.6 kg for a child.[68]

Clinical Presentation

Symptoms are summarized in Box 59-13. They are generally delayed for 4 to 50 (average 5 to 12) hours after ingestion. Initial symptoms include nausea, vomiting, and severe diarrhea. Some victims may have dizziness, weakness, muscle cramps, and loss of muscle coordination. In a severe ingestion, delirium, seizures, and coma are present. Hepatic failure develops over several days after ingestion, although hepatic damage is generally mild. Hypoglycemia, hypovolemia, severe hepatic failure, and death may occur.

Symptoms from gyromitrin toxin depend on amount consumed, toxin concentration, nature of the mushrooms, and other factors. The variability of symptoms after ingestion of gyromitrin mushrooms is much greater than with other mushroom species. Some individuals can consume large quantities of *Gyromitra* with few or no symptoms. A second meal by the same person may lead to severe toxicity. Repeated consumption may increase the risk of a severe reaction.

Box 59-14. Treatment of Gyromitrin Toxicity

1. Consider administration of activated charcoal.
2. Fluid and electrolyte replacement as needed.
3. Glucose replacement.
4. Pyridoxine 25 mg/kg up to 20 g/day intravenously to control neurologic signs.
5. If significant hepatic failure occurs, transfer to transplant facility.

Several drugs, including isoniazid, hydralazine, and probably MH,[17] are metabolized through acetylation of a hydrazine or amino group by the liver. Individuals vary markedly in the rapidity of acetylation. Two major human groups are termed "fast" and "slow" acetylators, with slow acetylation being an autosomal recessive trait. Slow acetylators in general have greater and more prolonged toxicity after ingestion of these drugs. Similar variation in fast and slow acetylators is seen when *Gyromitra* mushrooms are ingested.

Treatment

Symptoms develop several hours after ingestion of *Gyromitra*. Treatment is summarized in Box 59-14. Activated charcoal (1 g/kg orally or via gastric tube) is often given but is of little value. Pyridoxine has been useful in victims with neurologic disorders from MH toxicity and is of theoretical benefit in patients with gyromitrin toxicity. Persons who ingest *Gyromitra esculenta* and develop significant neurologic symptoms should receive pyridoxine (25 mg/kg IV initial dose, up to 20 g/day).[3,54,107] No evidence indicates that pyridoxine alters the course of hepatic disease. High-dose pyridoxine therapy may cause peripheral neuropathies.[1]

Baseline measurements of liver transaminases (alanine aminotransferase [ALT], aspartate aminotransferase [AST]), prothrombin time (PT), partial thromboplastin time (PTT), BUN, creatinine, complete blood cell count (CBC), platelet count, glucose, and electrolytes should be performed. ALT, AST, PT, PTT, BUN, and creatinine should be monitored at least daily for 3 to 4 days for the development of hepatic failure. Rapid deterioration of liver (ALT or AST >2000 IU, PTT >50 sec) mandates immediate transfer to a tertiary center with liver transplantation capability. Persons with significant hepatic failure may require monitoring of blood glucose every 2 to 4 hours and supplemental glucose administration for symptomatic hypoglycemia.

No specific antidote or treatment is available for the fulminant hepatic failure. The appropriate timing or necessity of liver transplantation is uncertain. In persons with fulminant hepatic failure from infectious causes, the presence of an abnormal PT (unresponsive to fresh-frozen plasma) and development of hepatorenal syndrome, grade II hepatic encephalopathy, hypoglycemia, and uncorrectable metabolic acidosis are used as signs that transplantation is needed on an emergent basis. Persons with fulminant hepatic failure from toxic ingestion, elevated bilirubin level, and young age are important indicators of a poor prognosis. Mortality from gyromitrin poisoning is reported to be 15% to 35%.[32]

Figure 59-28. *Cortinarius* species (United States).

Box 59-15. Mushrooms Reported as Causing Renal Failure

AMANITA
proxima
pseudoporphyria
smithiana

CORTINARIUS
gentilis
orellanus
rellanus
rubellus
speciosissimus
splendena
venenosus

Renal Toxicity

Although originally thought to be an edible mushroom, *Cortinarius orellanus* was associated with 81 cases of renal toxicity in the 1950s.[39] This led to isolation of the toxin orellanine, which is found in the mushrooms *C. orellanus*, *C. speciosissimus*, and *C. gentilis*. Most cases occur in Europe and Japan.

Causative Mushrooms

C. orellanus has a small brown to brownish-red smooth cap 30 to 80 cm (12 to 31 inches) in diameter. The stalk is somewhat yellow, often darker closer toward the soil. Gills are orange to rust with rust colored spores. It grows in deciduous woods, most commonly in sandy soil underneath oaks and birches. It is ubiquitous throughout Europe. Some other species of *Cortinarius* are found in the United States and may be toxic (Fig. 59-28; Box 59-15). Several *Amanita* species also have been reported to have renal toxicity.

Toxin

The two toxins isolated from *C. orellanus*, orellanine and orelline, are structurally related to paraquat and diquat. Their mechanism of action remains a mystery. These are heat-stable compounds, unaffected by cooking. The toxin appears to cause intense interstitial nephritis with early fibrosis.[46] The toxin can be identified with thin-layer chromotography.[49]

Amanita smithiana, which contains the nephrotoxins norleucine (aminohexadrenoic acid) and chlorocrotylglycine, grows in the Pacific Northwest and may be mistaken for *Tricholoma magnivalere*.[104] *Amanita proxima* (found in France) causes renal failure.[18]

Clinical Presentation

Persons who ingest these mushrooms are generally asymptomatic for 2 to 20 days. After this latent period, acute renal failure develops. Some persons develop neurologic changes, including paresthesias, taste impairment, and cognitive disorders.

Symptoms vary greatly. One case report described 26 soldiers who ate soup made of *C. orellanus* in nearly identical quantities.[11] Acute renal failure developed in 12 victims on or around day 11. Eight individuals later recovered normal renal function, whereas 4 required long-term dialysis or kidney transplantation. The other 14 soldiers showed no rise in BUN or creatinine levels but developed leukocyturia and hematuria that persisted for more than 1 month. Renal failure reportedly occurs in 30% to 46% of persons who ingest these mushrooms and become ill. Renal function returns in 46% to 66% of affected victims.

Renal biopsy in patients with orellanine-induced renal failure shows changes in the tubular epithelial cells and in the actin filaments within their cytoplasm.[73] These biopsy changes may persist for up to 3 months.[11]

Treatment

In most persons who ingest renally toxic mushrooms, unexplained acute renal failure develops many days later. If a person presents early after eating orellanine-containing mushrooms, gastric emptying and activated charcoal administration (1 g/kg orally or via gastric tube) might prevent some absorption, decreasing the resultant toxicity. Early presentation however, is rare.

Once acute renal failure develops, baseline and repeated monitoring of BUN, creatinine, electrolytes, CBC, differential, and urinalysis should be performed to monitor renal function. Urine output should be monitored, and if it decreases, fluid administration should be used to achieve optimal hydration. Serum potassium, calcium, and magnesium should be monitored closely. If renal failure progresses, the victim should be transferred to a facility for hemodialysis and possible renal transplantation. Renal function may return to normal after months of dialysis dependency. Steroids seem to have no effect on the course of the disease, but they may have been given too late.

Amatoxins

The mushrooms that contain amatoxins are responsible for 90% to 95% of fatalities due to mushrooms (Box 59-16 and Table 59-6). *A. phalloides* is most common in central and eastern Europe; immigrants to the United States may have carried mushroom spores in wood products from eastern Europe. *A. verna* and *A. virosa* are more common in the United States. Toxicity has occurred from cooked or frozen mushrooms and even tea made from *Amanita* mushrooms.[75]

Causative Mushrooms

Mushrooms reported or suspected of containing amatoxins are listed in Box 59-16. The common names of *A. phalloides* (Fig.

Box 59-16. Mushrooms Reported or Suspected of Containing Amatoxins

AMANITA
bisporigera
decipiens Jacquetant
hygroscopica Coker
ocreata Peck
suballiacea Murr
tenuifolia Murr
ocreata
phalloides (see Figure 59-29)
verna
virosa (see Figure 59-30)

CONCYBE FILARIS

LEPIOTA BRUNEO-INCARNATA

GALERINA
autumnalis Smith and Singer
badipes Kühn
beinrothil Brsky
fasciculata Hongo
helvoliceps Sing
marginata Kühner
Sulciceps Boedjin
Unicolor Sing
marginata
venenata AH Smith

LEPIOTA
kuehneri Huijsm. Ex Hora
langei Locq.
josserandii
josserandii Bon and Boiffard
brunneoincarnata Chodat and Martin
brunneolilacea Bon and Boiffard
castanea
castanea Quelet
citrophylla (Berk and Br.) Sacc.
clypeolaria (Bull.:Fr.) Kummer
clypeolarioides Rea
feline (Pers.:Fr) Karsten
fulvella Rea (by semiquantitative Meixner test)
fuscovinacea Moeller and Lange
griseovirens Maire
heimii Locq.
helveola
helvella Bres.
helveoloides Bon ex Bon and Andary
lilacea Bres.
locanensis Espinosa
ochraceofulva Orton
pseudohelveola Kuhner ex Hora
pseudolilacea Huijsm.
rufescens Lange
subincarnata Lange
xanthophylla Orton

Figure 59-29. *Amanita phalloides*—toxic.

TABLE 59-6. Look-Alikes of Mushrooms Containing Amatoxin

TOXIC SPECIES	EDIBLE SPECIES
Amanita phalloides (see Figure 59-29)	*Amanita fulva*
Amanita virosa (see Figure 59-30)	*Agaricus bisporus* (see Figure 59-7)
Amanita verna	*Lepiota flavovirens*

59-29) and its relatives, *A. verna* and *A. virosa* (Fig. 59-30), are death cap, death angel, and destroying angel, reflecting their association with fatal outcome. *A. phalloides* has a white to greenish cap 4 to 16 cm (1.5 to 6 inches) in diameter, often with remnants of the veil as warts. The stalk is generally thick, 5 to 18 cm (2 to 7 inches) long, with a large bulb at the base, often with a vulva. A thin ring is usually present on the stalk. Gills are generally free and white to green in color; spores are white. The mushrooms grow under deciduous trees in the autumn.

A. virosa (see Figure 59-30) is more common in the U.S. It resembles *A. phalloides*, but the cap is more yellow or white. *A. verna* is characteristically white. All grow in deciduous woods. Even mushroom experts have been tempted by the large white mushroom, which is tasty. The fatality rate is 35% in adults and 50% in children. Unfortunately, mushroom gatherers may mistake amatoxin-bearing *Amanita* species for the edible mushrooms that they were seeking (see Table 59-6).

Some *Lepiota* mushrooms (Fig. 59-31), including *L. castanea* and *L. josserandii*, contain high concentrations of amatoxin. There have been two deaths attributed to *L. josserandii* in upstate New York.

Mushrooms that contain amatoxin may have a positive Meixner test. This test was first described by Wieland in 1949[106] and popularized by Meixner.[67] A drop of liquid is expressed from a fresh mushroom onto print-free (ligand-free) newspaper and allowed to dry. A drop of concentrated (10 to 12 N) hydrochloric acid is added. A blue color develops within 1 to 2 minutes in the presence of amatoxins. Control tests on news-

Figure 59-30. *Amanita virosa*—toxic.

Figure 59-31. *Lepiota* species—some toxic.

paper without mushroom juice and paper containing ligand should be conducted. False-positive results are common and can be elicited by excessive drying temperatures (greater than 63°C [145°F]) or exposure to sunlight. The test can detect 2 μg of alpha-amatoxin.[7] False-positive tests also occur from mushrooms containing psilocybin, terpenes, bufotenin, and other tryptamine compounds.[7] Nearly 20% of gilled mushrooms that did not contain amatoxins tested positive in one study.[91] A positive test does not identify an amatoxin-containing mushroom, and further identification is necessary. Thin-layer chromatography more accurately identifies the presence of amatoxin and can be done on mushroom liquid, human serum, or urine.[80,88] Radioimmunoassay (RIA) of serum or urine can detect amatoxins in the body. These tests are available in specialized laboratories usually associated with mushroom farms or botany schools.

Toxins

The mushroom *A. phalloides* contains two groups of toxins: amatoxins and phallotoxins. Each group contains several toxins. There are now eight identified amatoxins: α-amanitin, β-amanitin, γ-amanitin, ε-amanitin, amanin, amaninamide, amanullinic acid, and amanullin. Of these, α-amanitin is thought to be primarily responsible for human disease. α-Amanitin injected into animals produces hepatic toxicity characteristic of human ingestion of *A. phalloides*. Phallotoxins include phalloidin, phalloin, phallisin, phallacidin, phallacin, phallisacin, and prophalloin. Phalloidin is the primary phallotoxin. Phallotoxins bind to F-actin, disrupting plasma membranes and causing massive efflux of calcium and potassium. Phallotoxins cause death in animals within 2 hours, but are not believed to play a role in human toxicity.[33] Humans may not even absorb these toxins, which may be responsible for local gastric irritation. *A. virosa* contains amatoxins and virotoxins. Virotoxins resemble phallotoxin biochemically and also bind F-actin and cause death within a few hours. Six different virotoxins have been isolated, but none is thought to play a role in human *Amanita* hepatotoxicity.

Amatoxins are heat stable and not destroyed by cooking, freezing, or drying. Recently, *Amanita phalloides* mushrooms stored in a freezer for 7 to 8 months resulted in death.[44]

Amatoxins are concentrated in the cap, gills, ring and stalk. Phallotoxins are more concentrated in the bulb and vulva. The concentration of toxin in a given mushroom depends on environmental factors and soil.[19] Amatoxins can be detected by high-performance liquid chromatography (HPLC) or radioimmunoassay but not gas chromatography–mass spectrometry (GC-MS). After ingestion, amatoxins are absorbed from the gut and actively transported into the liver through transport systems shared by bile acids and xenobiotics. They do not appear to cross the placenta.[101] α-Amanitin is rapidly cleared from plasma.[50] Amatoxins are not protein bound. They bind to ribonucleic acid (RNA) polymerase II and inhibit formation of messenger RNA (mRNA).[20] This in turn inhibits transcription, as the reservoir of mRNA is depleted. Amatoxins are excreted into the bile, where they are reabsorbed and once again transported into the liver.[12] Interruption of this enterohepatic circulation may be therapeutic tool.

Within the liver, α-amanitin may undergo some metabolism through the hepatic cytochrome system. Animal studies suggest that a more toxic metabolite may be produced through this metabolism, although this has never been isolated.[89,90] Nuclear fragmentation and condensation of chromosomal material have been observed within 15 hours of injection.[27] Glycogen is rapidly depleted, and fatty degeneration occurs within liver parenchymal cells. Mitochondria become swollen, and microvesicules appear throughout the cytoplasm.[27] Direct renal toxicity may occur.

Clinical Presentation

Symptoms are summarized in Box 59-17. Persons who ingest the mushroom *Amanita phalloides* feel well for 4 to 16 hours. Severe nausea, vomiting, abdominal cramps, and diarrhea follow this characteristic latent period. Early complications include fluid and electrolyte imbalance (hypoglycemia, hypokalemia, and elevated BUN). Persons in whom symptoms develop earlier (between 4 and 10 hours) are more likely to

Box 59-17. Symptoms of Amatoxin Ingestion

1. Delayed onset of nausea, vomiting, and diarrhea 4 to 16 hours after ingestion.
2. Resolution of GI symptoms 12 to 24 hours later.
3. Onset of hepatic and occasionally renal failure 48 to 72 hours after ingestion.
4. Coagulopathy and pancreatitis may be seen with the hepatotoxicity.

Box 59-18. Treatment of Amatoxin Ingestion

ESTABLISHED:
Administer activated charcoal (1 g/kg orally or via gastric tube)
Silymarin (silibinin) 20 to 40 mg/kg/day IV, 1.4 to 4.2 g/day

EXPERIMENTAL:
Consider hyperbaric oxygen if available
Cimetidine 8 to 20 g IV divided over 48 hours
If significant hepatic failure develops, transfer to transplant facility

experience severe hepatotoxicity. Over the next 12 to 24 hours, the victim's GI symptoms abate. The second latent period is followed by hepatic failure, which develops between 48 and 72 hours after ingestion in most victims. Hepatic failure may be of varying severity; it is frequently worse in children and depends minimally on the amount of mushroom ingested.

Children have greater toxicity and higher mortality, perhaps because the relative quantity of mushrooms ingested or of the varying metabolism in young children (differing levels of cytochrome enzymes). Previous experiments showed that concurrent ethanol with *A. phalloides* ingestion (whole mushroom lyophilized) decreased hepatotoxicity.[29] Therefore, decreased toxicity in adults could result from ingestion of ethanol with an *Amanita* mushroom dinner. More recently, however, ethanol failed to alter hepatotoxicity in an animal model poisoned with α-amanitin, which raises doubts about this explanation for increased toxicity in children.[88]

In addition to hepatic failure, endocrinopathies can develop, with hypocalcemia, decreased thyroid function, and elevated insulin levels in the presence of hypoglycemia.[53] Hypocalcemia may be caused in part by loss of calcium through diarrhea or by a direct effect on osteoclasts. Renal failure may contribute to hypocalcemia. The thyroid abnormalities probably result from decreased hormone synthesis caused by overwhelming illness and blocked peripheral conversion of thyroxine (T_4) to triiodothyronine (T_3). Thyroid-stimulating hormone depression may result from decreased synthesis caused by inhibition of RNA polymerase II by amatoxin. Hypothyroidism has not been clinically significant. Hypoglycemia is probably the result of several processes, including impaired hepatic gluconeogenesis, increased insulin release from the initial hyperglycemia, and tissue destruction of the pancreas.[28] Bone marrow toxicity with decreased neutrophils, lymphocytes, and platelets has been noted. Disseminated intravascular coagulation and coagulopathies secondary to hepatic dysfunction are common.[83] Pancreatitis occurs in up to 50% of patients.[28] Hypophosphatemia is particularly common in children, for unknown reasons. Myopathy has been associated with *Amanita* toxicity.[38]

Hepatic biopsy shows diffuse and severe steatosis with periportal inflammation and necrosis. Renal biopsy shows acute tubular necrosis with hyaline casts.[26] Extremely high levels of hepatic enzymes are seen. The level of liver transaminase is not helpful in predicting the victim's prognosis. A precipitous drop may occur just prior to death.

Treatment

Attempts to treat *A. phalloides* poisoning have ranged from scientific to purely empirical (Box 59-18). Noting that rabbits were able to eat the *A. phalloides* mushroom with impunity, clinicians fed ground raw rabbit to victims of *Amanita* poisoning, without success.[98] Hemodialysis was long recommended but now has been shown to be ineffective, because the toxin is rapidly cleared. Amatoxin is taken up in liver cells within 5 hours after intravenous administration.[25] In a retrospective study of 205 cases of amatoxin ingestion,[30] hemodialysis worsened the prognosis, and charcoal hemoperfusion did not provide any improvement in outcome. Plasmapheresis has been used, but this appears to be ineffective for similar reasons.[77]

Despite this information, the literature continues to report the use of dialysis, hemoperfusion, and plasmapheresis.[52] In Europe, an extracorporeal liver assistance device (molecular absorbent regenerating system) has been developed and used successfully in 4 patients.[16]

Antigen-binding fragment (FAB) monoclonal antibodies against amatoxin were developed and tested in mice. Although hepatic toxicity was markedly lessened, renal toxicity was 50 times greater than in control animals, possibly due to dissociation of the amatoxin-FAB molecule in the kidney.[24]

Previously, thioctic acid and benzylpenicillin were recommended as treatments. Both these treatments are now questioned. In a retrospective study, thioctic acid was more frequently associated in humans with fatal outcome.[30] A more recent review of treatment showed benzylpenicillin to be more effective than simple supportive care, but less effective than silymarin or antioxidant therapy.[20] The authors of that paper suggested abandoning the use of penicillin. However, since silymarin is not easily available in the U.S., some poison centers may still recommend penicillin. If so, the recommended dose is 300,000 to 1,000,000 units/kg/day IV. Side effects are significant and include allergy, hyponatremia,[21] and granulocytopenia.[71]

Silymarin is the active component of the milk thistle *Silybum marianum*. It is an antioxidant that accumulates in the liver.[58] Silymarin binds tightly to the plasma membrane, stabilizing it,[78,79] and hinders amatoxin from penetrating the cell wall.[51,102,103] It is a free radical scavenger, [31,63,105] inhibits lipid peroxidation,[10,13,31,69] and stimulates RNA polymerase I.[105] In both retrospective studies, silymarin has shown promising reductions in mortality.[20,28] Although the intravenous form is not available in the United States, the oral form can be found in health food stores. The intravenous dose of *Silybum dihemisuccinate* is 5 mg/kg over 1 hour followed by 20 mg/kg/day for 6 days or until transaminases normalize. The oral preparation is less well studied; dosage recommendation is 1.4 to 4.2 g/day (70-mg capsules).

Gastric decontamination (ipecac, gastric lavage, whole bowel irrigation) is not considered effective. The ingestion is generally

many hours old, and the intense vomiting is generally enough to clear the stomach. Although amatoxin has enterohepatic circulation, there are no data that provide support for any therapy designed to disrupt the enterohepatic circulation.[20] Likewise, there are no data to support forced diuresis.

The French have used hyperbaric oxygen as a treatment for *Amanita* toxicity.[59] Hyperbaric oxygen has been shown to have efficacy in animal models.

Recent animal work has suggested the use of antioxidants. Cimetidine has been shown to be effective in animals[89] and has been reportedly used in 21 humans[20] (dosage: 4 to 10 g/day IV for adults; pediatric dose not described). Vitamin C and *N*-acetylcysteine have been used with some success.[20,100] Other reported treatments include aucubin and kutkin, both plant derivatives.[20]

Intravenous normal saline or Ringer's lactate is needed to replace GI fluid losses. Electrolyte losses (particularly potassium) may be great. BUN, creatinine, CBC with differential, platelet count, electrolytes, glucose, calcium, phosphorus, magnesium, urinalysis, PT, PTT, fibrinogen, amylase, protein, and albumin should be initially measured and repeated at least daily to monitor liver and renal function. Hyperglycemia is common on the first day, but insulin is generally not required. Hypoglycemia that occurs after 24 hours may be severe, requiring intravenous concentrated glucose. Therefore, bedside determinations of glucose should be performed at least every 6 hours. The patient with liver failure due to *Amanita* will appear like a patient with any other type of advanced liver disease, with jaundice and asterixis. Hepatic encephalopathy can develop with elevated ammonia and intracranial pressure. Tests of liver function, including ALT, AST, alkaline phosphatase, ammonia, PT, and PTT, should be repeated at least daily and more often as indicated if findings become abnormal. Levels rapidly rising or in excess of ALT or AST >2000 IU or PTT >50 sec signal severe toxicity and the need for referral to a transplant center.

To confirm the diagnosis of amatoxin ingestion, a red-top glass tube blood sample drawn on admission can be collected for serum radioimmunoassay for amatoxins. Urine can also be studied. The nearest laboratory performing this assay is usually known to the regional poison information center.

Once liver failure begins, it is treated the same as any other fulminant hepatitis. Hypoglycemia becomes more likely. Supplemental glucose should be readily available. Dietary protein should be limited to 0.5 g/kg/d. Thiamine (100 mg IV) and multivitamin supplementation are given, though there is little rationale for their use. Oral lactulose, 30 to 45 mL every 6 to 8 hours, may reduce hepatic encephalopathy. In patients who do not tolerate lactulose, neomycin 1 g orally three times a day can be tried. PT and PTT should be measured at least twice a day, and vitamin K (100 mg IM), fresh-frozen plasma (2 to 6 U initially), or both are used to correct abnormalities.

If hepatic failure progresses, liver transplantation may be required. The timing of transplantation is highly controversial, and criteria for transplant in other forms of fulminant hepatic failure are often applied to this setting. Factors associated with poor prognosis in acetaminophen-induced hepatic damage include metabolic acidosis, PT >50 sec, and elevated serum creatinine (>2.0).[74] However, in viral hepatitis and other drug reactions, factors such as bilirubin, victim age, and the duration of jaundice prior to clinical encephalopathy are important.[62] In the few pertinent studies of *A. phalloides* ingestion, poor outcome was related to age younger than 10 years, a short latent period (time from ingestion until the onset of nausea/vomiting) of less than 8 hours, and coagulopathy that was not corrected by fresh frozen plasma.[30] The largest study suggests that a person with ALT or AST level >2000 IU, grade II hepatic encephalopathy, or PT >50 sec is at serious risk for death and should be considered for emergency liver transplantation.[23] Persons who met these criteria have survived without transplant.[62,81] A recent report suggests that increased reparative enzymes correlate with hepatic recovery.[48] These enzymes, including alpha-fetoprotein, retinal binding protein, gamma-glutamyl transferase, and des-gamma-carboxyprothrombin, are thought to be released during the healing phase of liver disease. Because hepatic failure develops rapidly, victims with significant hepatic dysfunction must be transferred early to a transplantation site. Patients undergoing liver transplantation for fulminant hepatic failure (not caused by *A. phalloides*) have a 60% to 80% survival rate.[20] Recently, a temporary liver transplant was performed on a child and sustained her while her own liver recovered.[82]

Persons who survive acute hepatic failure due to *Amanita* without needing hepatic transplant may have persistent elevation in liver transaminases. In one study of 14 persons with severe *Amanita*-induced hepatotoxicity, 8 showed persisted elevation in AST and ALT without normalization over a 1-year follow-up period.[23] All had biopsy evidence of chronic active hepatitis with positive anti–smooth muscle antibody and cryoglobulins. It is not known whether these persons will have an increased risk of hepatoma or develop more serious complications of chronic active hepatitis.

Amatoxin ingestion during pregnancy does not appear to have serious consequences. There are now several reports that suggest amatoxin does not cross the placenta.[87,97] A case study of 22 pregnant women poisoned during their pregnancy had similar outcomes to a control group; however, only 5 ingested mushrooms in the first trimester.[97]

Miscellaneous Reports of Toxicity From Mushrooms

Occasional toxicity is reported with other mushrooms. Erythromelalgia has been reported after ingestion of *Clitocybe amoenolens*.[34,85,86] The mushroom contains the toxin clitidine, which resembles nicotinic acid. *Tricholoma equestre* ingestion is associated with rhabdomyolysis, respiratory failure, and hepatic and renal dysfunction.[5,15] Deaths have been reported due to acute myocarditis and renal failure.[5]

APPROACH TO THE VICTIM OF MUSHROOM POISONING

Four types of individuals develop mushroom toxicity: foragers, children, those seeking hallucinogenic "highs," and, rarely, victims of attempted homicide. Most victims seek medical care after symptoms develop. If small children are observed chewing on lawn mushrooms, caretakers should be advised to call the nearest regional poison information center.

Persons with agitation, altered perceptions, or frank hallucinations temporally related to mushroom ingestion are probably intoxicated with isoxazole or hallucinogenic mushrooms. Whether the mushrooms are picked accidentally or ingested intentionally, the treatment and clinical course are identical.

Persons who develop muscarinic symptoms (salivation, urination, diaphoresis, GI upset, and emesis) present such a classic picture that it is rarely confused with any other presentation. Some drugs (e.g., bethanechol) may cause similar symptoms when taken in overdose. Victims of mushroom poisoning generally remain mentally clear and should be able to relate an appropriate history.

Victims with GI symptoms can be divided into those with early and those with delayed presentations. Those with early GI symptoms (within 2 hours of ingestion) generally have a benign course, except for persons with a mixed ingestion. Most guidebooks for mushroom hunters recommend eating only one variety of mushroom at a time, but more daring or foolish individuals mix multiple mushrooms and eat them frequently over the day. This makes diagnosis based on time of onset of symptoms difficult. Early onset of GI symptoms may mask more significant delayed symptoms. In these cases, identification of ingested mushrooms becomes essential to planning therapy.

Accurate botanical identification of the mushroom can be difficult. Only 800 of the 3000 species found in Europe can be identified without a microscope.[98] With the popularity of digital imaging, photos of suspicious mushrooms can be sent to poison centers via email and similarly can be forwarded to mycologists. This may save several hours of travel and may help initiate treatment if needed in a more timely fashion. When multiple mushrooms are eaten together, the residual specimens brought from home may not be those causing toxicity. Cooking and refrigeration alter identifying features. Fresh mushroom specimens should be transported in a paper bag rather than in a plastic container to limit the effects of humidity. Finally, precise identification of even a good specimen can be difficult and should be done by an expert. Mycologists can be contacted through a poison center, university, museum, or commercial mushroom grower.

In difficult cases, spores can be obtained from emesis or gastric emptying procedures. Specimens should be refrigerated while awaiting analysis. More specific diagnosis can be made through thin-layer chromatography or RIA techniques. Botanical identification may not match the victim's symptoms. The victim should be treated according to time of onset of symptoms and current condition when examined.

Victims with early-onset GI symptoms require supportive care with fluid and electrolyte replacement. For those with delayed GI symptoms or mixed ingestions containing amatoxin or gyromitrin mushrooms, treatment should begin as soon as possible. These mushrooms can be differentiated by the season (spring: *Gyromitra;* autumn: *Amanita*).

Persons who have disulfiram-like reactions to alcohol should be questioned about prior mushroom ingestion. This situation is rarely correctly diagnosed because symptoms are thought to result from panic attacks, alcohol intoxication, or even an allergic reaction. Persons rarely relate their symptoms to the dinner of mushrooms eaten days earlier.

Any person with unexplained acute renal failure should be questioned about prior wild mushroom ingestion. Although *Cortinarius orellanus* is more common in Europe and Japan, it is found with increasing frequency in the United States. Because of the long delay before the onset of renal failure (1 to 2 weeks), the history of mushroom ingestion may be missed.

Mushroom poisoning cases remain relatively rare in the U.S. (they are still more common in parts of Europe and Asia), and until recently, up-to-date information was not accessible to the general public except through poison centers. With the plethora of worldwide Internet connections, there are now mushroom experts readily available to answer questions about possible exposures within minutes to hours of contact. The active forager should utilize well-written field guides in addition to internet sources and should cross-reference several sources before consuming any unknown mushrooms.

RECOMMENDED FIELD GUIDES

Lincoff G: The Audubon Society Field Guide to North American Mushrooms. New York, Knopf, 1992.
Lincoff G: Simon and Schuster's Guide to Mushrooms. New York, Simon and Schuster, 1981 (European mushrooms).
Phillips R: Mushrooms of North America. Boston, Little, Brown and Company, 1991.
Miller OK: Mushrooms in North America. New York, E. P. Dutton, 2006.

RECOMMENDED WEBSITES

www.fungaljungal.org
www.mushroomexpert.com
www.evergreen.edu/mushrooms
www.bluewillowpages.com/mushroomexpert/sitemap2.html

The references for this chapter can be found on the accompanying DVD-ROM.

Ethnobotany: Plant-Derived Medical Therapy

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Kevin Jon Davison and Rick Marinelli

The history of ethnobotany begins before the advent of written records. In all ancient civilizations, plants served as important elements of food, shelter, dyes, ornamentation, religious rituals, and medicines. The term *ethnobotany* refers to an individual culture's use of specific plants. The medicinal use of the plant kingdom has been termed herbalism, plant medicine, and natural-based medicine, and it is called phytomedicine in its current application. The word *herb* is broadly defined as a nonwoody plant that dies down to the ground after flowering. The most commonly used interpretation, however, is any plant used for medicinal therapy, nutritional value, food seasoning, or dyeing another substance.

The history of the discovery of the medicinal uses of plants by humans remains conjectural. Many scenarios probably occurred. Perhaps, in a prehistoric jungle of South America, a pool of water containing fallen plant material leached out some of the precious medicinal constituents of leaves, flowers, stems, and bark. Tannins, glycosides, sugars, and alkaloids from the bark were infused into the waters. Because of burning fever and severe dehydration, an extremely ill native drank from the pool, and his fever miraculously disappeared. The pond became known for its magical healing powers. If the water held bark from the cinchona tree, the native may have serendipitously discovered quinine.

Archaeological evidence shows that prehistoric humans used plants extensively to treat physical ailments. Instinct and trial and error led to the realization that, for example, cinchona bark controlled intermittent fevers, animals fed ergotized grain aborted their fetuses, and the latex sap from the opium poppy could be eaten to alleviate pain. Innumerable medicinal plant traditions, some originating as far back as 2700 BC, still remain intact. Ethnobotanically, the use of plant-based medicines in a particular culture represented much more than an individual's efforts to survive. Analyzing the methods and degrees of use of indigenous medicines reveals information about cultural philosophy, ingenuity, and sophistication. The Chinese developed an extensive and elaborate system for prescribing, classifying, and processing herbs that dates back to the 3rd millennium BC. The formulas identified the specific effect of each herb and interactions with other herbs. Less tolerable herbs were blended with those that would counteract undesirable effects. Formulas were custom blended, taking into account a victim's constitution and the stage of the disease. Some of the ancient knowledge from these writings is being used in contemporary herbal preparations commercially sold as "patent" (readily available in pill form) medicines.

Many native tribes of New Guinea, Indonesia, and the Amazon use single-herb formulations to treat nearly all medical conditions, as they did thousands of years ago. In the West, written records dating to the Sumerians accurately describe the medicinal uses of specific plants.[109] In the same period of about 3000 years ago, the first Asian written record, the Ben Tsao Gan Mu, was compiled by the Chinese. It listed more than 360 medicinal plants and their classification, uses, contraindications, and methods of action as perceived at that time. Roman and Greek herbal remedies were described in the writings of Hippocrates and later in those of Galen, providing a pattern for development of the Western medical tradition. Hippocrates was an advocate of using a few simple plant preparations along with fresh air, rest, and proper diet to help the body's own "life force" eliminate problems. In contrast, Galen promoted the use of direct intervention to correct the imbalances that cause disease, employing large dosages of complicated mixtures that included animal, plant, and mineral ingredients.[123]

The earliest European compendium that listed the uses and properties of medicinal plants, *De Materia Medica,* was written by the Greek physician Dioscorides in the 1st century AD. He described about 600 plants, and his work remained the authoritative herbal medicinal resource into the 17th century.[41]

Herbalism was practiced in many different ways during and after the Middle Ages. There were learned traditional herbalists and lay practitioners, as well as wandering herbalists, who professed pagan animism or Christian superstitions that often were more influential in healing than were the herbs' properties. Little was added to the knowledge of herbalism during this period. After the Middle Ages and the invention of the printing press in the 1400s, hundreds of herbal publications were compiled. Most early works were available only in Latin or Greek; it was not until the 15th through 17th centuries that the great age of herbalism was appreciated in English.[109]

Tides changed in European herbalism when a Swiss pharmacist-physician named Theophrastus Bombastis von Hohenheim, better known as Paracelsus (1490–1541), introduced a new dimension. He advocated chemistry and chemical processing and used mineral salts, acids, and other preparations in medicinal therapies. This was a departure from the plant-based medicinal methods of the past. During the latter part of the 17th century, the predominance of plant medicines slowly eroded. In 1806, Freidrich Serturner, a small-town German pharmacist, became known for his efforts to isolate organic acids from plants in an attempt to find the active ingredient in opium. He discovered organic alkaloids, which became known as the first

set of active plant constituents.[160] Because of their physiologic activity, the search for plant alkaloids continued into the 20th century.

Discoveries quickly followed. The bronchodilator and antitussive ephedrine, from the herb *Ephedra sinica,* was often used in Chinese medicinal formulas for bronchial asthma. The discovery of morphine led to the creation of all the narcotic analgesics. The bark of the cinchona tree was found to contain quinine in 1819, which led to the development of antimalarial drugs.

The traditional herbal extract from rhubarb (*Rheum* species) has several active compounds. These compounds mediate many of the pharmacologic effects, such as its purgative action (from sennosides); antibacterial, antifungal, and antitumor activities (from anthraquinones); anti-inflammatory and analgesic activities; and improvements of lipid metabolism (from stilbenes). Treatment of leukemias from an extract of Madagascar periwinkle *(Catharanthus roseus),* known as vincristine, has been highly effective.[42]

Discoveries in the 19th and 20th centuries included atropine (from belladonna leaves, *Atropa belladonna*) in 1831, cocaine (from coca leaves, *Erythroxylum coca*) in 1860, ergotamine (from *Claviceps purpurea*) in 1918, and tubocurarine in 1935.[123]

European settlers brought herbal knowledge and their medicinal methods to the Americas. Because of the abundance and wide use of plants on the new continents, they also learned much from the indigenous peoples. The colonists found that conditions afflicting them, such as malaria and scurvy, were treated effectively with herbs by the Native Americans.[125] In the 1700s, herbal medicine continued to have popular applications in lay circles, but it was also investigated by the new medical establishment. Although the creation of a small elite group of learned professionals was thought to violate the political and constitutional concepts of the early American democratic movement, the practice of medicine was carried over from England and Scotland during pre-Revolutionary days. Before a professional medical class was established, most illness in America was treated within the family or extended family network.

Many concepts were modified in the colonies between 1765, when the first medical school opened, and 1850, when more than 42 schools of medicine had been recognized. The inquiry into *Digitalis purpurea* (foxglove) by William Withering exemplified the change in perspective from anecdotal folk medicine to a critical examination for specific uses of botanicals from a biochemical point of view. During the early 1800s, the trend was to look at the efficacy of botanicals and their intrinsic value from a more scientific perspective.

Several developments delayed the appreciation of herbalism by physicians in the colonies. For instance, Samuel Thomson promoted a system of herbal medicine by proselytizing about his patented method of herbal prescribing, which used many Native American herbs. A central theme in his approach was advocacy of self-prescribing based on the philosophies and herbal prescriptions found in his book, *New Guide to Health*. The right to sell "family franchises" for use of the Thomsonian method of healing was the basis of a widespread lay movement between 1822 and Thomson's death in 1843. Thomson adamantly believed that no professional medical class should exist and that democratic medicine was best practiced by lay persons within a Thomsonian "family unit."[39] Although his methods were considered crude and unscientific, he had over 3 million faithful followers in 1839. Founded on ignorance, prejudice, and dogma, the Thomsonian school did little to help physicians accept European and American herbal medicines. European physicians in the Thomsonian movement wished to separate themselves from lay practitioners by creating requirements and standards for the practice of Thomsonian medicine. Thomson was adamantly against this, but a decade after his death, the Thomsonian physicians formed the Eclectic School of Medicine, which attempted to unite "professional physicians," Thomsonianism, and traditional herbal medicine. Establishment of several Eclectic medical schools was a step toward validating herbal medicine, but it failed to bring herbalism into the mainstream medical establishment. The founding of the American Medical Association and the Flexner Report on medical education in 1910 thoroughly established the modern pharmaceutical industry in the medical education system.[39]

Because of the availability of pure, active constituents from plant drugs and the synthetic drugs that began to appear on the market toward the end of the 19th century, the prescribing habits of physicians began to change. The sensibility and predictability of administering exact dosages were appealing. For example, the pure alkaloid of quinine, rather than a foul-tasting extract of cinchona bark containing variable percentages of quinine and other alkaloids with different physiologic properties, could be prescribed for malaria.

Many "crude drugs" were standardized for therapeutic activity. Digitalis, which still retains its status in the United States Pharmacopeia (USP), is one example. Of the 200 plant drugs officially listed in the USP in 1936, about 19% are still official today.[160] An estimated 25% of all prescriptions dispensed in community pharmacies between 1959 and 1980 contained ingredients extracted from higher plants. For a significant number of synthetic drugs, natural drug products continue to serve as either models or starting points for synthesis.

EVOLUTION OF PHYTOPHARMACEUTICALS

The drive toward patenting and ownership in the pharmaceutical industry has been a strong incentive to research and develop plant-based products. Because a plant cannot be patented, however, little U.S. effort has gone into developing herbal medicines during the past century. The principal active constituents of botanicals are investigated for their biologic activity, but in many cases these are less effective than the whole crude extract of an herb.[123]

One problem in the development of the U.S. botanical pharmaceutical industry has been quality control. In addition, lack of standardization plagues plant-based products. Quality control and standardization of crude plant extracts for herbal medicines were virtually nonexistent until recently,[123] or we might be using more botanical medicines for common ailments. In Europe and Asia, where pharmaceutical firms have been producing standardized phytopharmaceuticals (plant-based standardized extracts) for decades, research and development have demonstrated that they make economic and medical sense. Europeans use phytopharmaceuticals as part of their mainstream medical practice. In hospitals, they are used primarily as adjuvant therapies. More than 70% of general practitioners in Germany prescribe phytopharmaceuticals, and the public health insurance system pays for most of these prescriptions. The total annual market for phytopharmaceuticals in Germany alone is

A B

Figure 60-1. **A,** *Calendula officinalis.* **B,** *Calendula* drying and dried in a jar. (Courtesy Cascade Anderson Geller.)

$1.7 billion. Beginning in 1993, the licensing procedure for German physicians required a knowledge of phytotherapy.[141]

Production and evaluation of botanical medicines have improved significantly in the past 6 decades. In crude plant evaluation, modern laboratory analysis can determine the percentage of active constituents, as well as solubility, specific gravity, melting point, optical rotation, and water content. Scientists detect resins, alkaloids, flavonoids, enzymes, essential oils, fats, carbohydrates, and protein content. They can precisely assay using liquid, high-pressure liquid, paper, and thin-layer chromatographies; spectrophotometry; atomic absorption; and magnetic resonance imaging. These methods improve the predictability and therapeutic effectiveness of standardized crude botanical medicines, which are then evaluated for their efficacy in animal studies to determine pharmacologic potency, activity, and toxicity. U.S. and European companies have set strict quality control guidelines to ensure optimal yields of pharmacoactive constituents, with acceptable levels of impurities, pesticides, residual solvents, and heavy metals, and acceptable bacterial counts.

Specific cultivation and harvesting techniques affect the therapeutic value of a given herb, which is related to the amount of active constituents in a specific medicinal plant. Methods of packaging, storage, and transport can dramatically affect the stability of active compounds. Both extracts and concentrates are obtained by adding appropriate solvents to raw herbs, which draws out the active constituents. The most common method is infusion, which is analogous to a tea bag being steeped in hot water to make tea; in this case, the water is the solvent. When the water is slowly evaporated, the concentrate contains the active constituents.

Pure ethanol is a solvent that is often used effectively to concentrate active herbal constituents. Immersing a high-quality bulk or raw herb in pure ethanol for hours or days, depending on the herb and the part used, and then pressing the solids out, yields an herbal tincture. The alcoholic tincture is diluted with water to yield a 20% alcohol tincture. In another method, a 20% alcohol mixture is the solvent. Fluid extracts can be made by vacuum-distilling some of the alcohol off; this avoids elevating the temperature, which may affect some of the active constituents. Another concentration process, solid extraction, yields a solid or semisolid product that can then be powdered or granulated for administration.

Once an extract is produced, qualitative and quantitative analyses can be performed to assist in standardization. The percentage of known active constituents is assayed, to obtain predictable clinical results.

An herbal infusion is generally a better source of active compounds than an air-dried or a sun-dried powdered herb (Fig. 60-1), but its action may not be as strong as that of concentrates such as tinctures, solid extracts, and fluid extracts. Potency of an extract can be defined by (1) percentage of active constituents or (2) concentration. Herbalists express concentration as an equivalency: a four-to-one extract is equivalent to or derived from four parts of the crude herb to yield one part extract. This is usually written as "4:1 solid extract." Longer shelf life, greater effectiveness, and higher concentration of active constituents make a more standardized (thus better) product than the raw powdered herb; however, efficacy is difficult to compare.

An example of a product that is standardized by the percentage concentration of pharmacoactive glycosides is *Ginkgo biloba* extract, marketed in Europe under the trade names Tanakan, Rokan, and Tebonin. It is typically standardized as 24% flavonoid glycoside. *G. biloba* extract has been shown to prevent metabolic and neuronal disturbances of cerebral ischemia and hypoxia in experimental models.[92,104]

Quality control is addressed for many herbal products when the known clinical effectiveness can be attributed to a specific active constituent. Improved analytic methods and use of high-quality herbs (i.e., high in active constituents) helps ensure standardization. In Europe, the dosage is expressed in milligrams of active constituents, a system that favors consistency. The main difference between the infusion or extraction method and chemical isolation or synthesis is that the extracts still contain all the synergistic cofactors that enhance the function of the active ingredient. This important aspect of herbal medicine is lost once the active constituent is removed from the whole plant.

HERBAL PREPARATIONS FOR CLINICAL AND WILDERNESS USE

Botanical preparations can be readily and accurately prescribed for travelers and wilderness enthusiasts who need medical help. Throughout the ages, botanicals have been useful adjunctive

therapeutic agents. Knowing what preparations from the natural pharmacopeia can be used and how to use them engenders a sense of integration with the natural environment. Indigenous peoples who depend on the botanical world hold a vast amount of untapped knowledge. Wilderness enthusiasts should help preserve this understanding of the natural world and should do what they can to save natural habitats. Further investigation into the plant kingdom for useful medicinal agents will aid in these efforts.

Herbal medicines can be prepared by decoction or infusion of bulk or raw herbs, or by making an extract, a concentrate, or a tincture.

Infusions are prepared like a standard tea. The soft parts of plants—flowers, stems, and leaves—are placed in a warmed pot. Boiling water is poured over the herb, and the pot is covered to prevent beneficial essential oils from evaporating. The mixture infuses for about 10 minutes and then is strained. The supernate can be used immediately or refrigerated in an airtight container for as long as 2 days. A standard adult dose of an herbal preparation is 1 ounce (28 g) of dried herb in 1 pint (500 mL) of water, or a tablespoon per cup. The amount is doubled if the herb is fresh.

Generally, it is best to take infusions hot by the cupful, three times daily for a chronic problem and up to every hour or two during an acute illness. To make infusions palatable, many herbalists add licorice, aniseed, or honey. The hard or woody parts of plants, such as bark, seeds, roots, rhizomes, and nuts, have tough cell walls that must be broken down by longer heating before they impart their constituents to water. The herbs can be first broken into small pieces by chopping, crushing, or hammering.

Traditionally, a *decoction* was prepared in an earthen crock reserved especially for making herbal preparations. In the past, herbalists believed that some quality of the medicine was affected by the type of vessel or container in which the brew was prepared. Contemporary practitioners generally recommend the use of stainless steel, ceramic, or enamel and specifically discourage the use of aluminum or other alloyed metal pots. The herb is placed in an appropriate container and covered with cold water. The mixture is brought to a boil, covered, and simmered for 10 to 45 minutes, depending on the type and part of the herb being used. A decoction can be strained, flavored, or sweetened like an infusion, and it is consumed while hot.

Modern practitioners use the most efficient and predictable forms of specific herbal medicines. *Concentrates* in capsule form are most effective and easiest to administer. The standard herbal concentrate found in the marketplace is in a ratio of 4:1. Ease of administration and dosing and the predictable clinical effects have made this the industry standard. Herbal *tinctures* are extracted into a specific percentage of alcohol and can be mixed easily to make formulas tailored to personal circumstances. Formula prescribing is an art, and a combination may be many times more effective than a single herb. Classic formulas for common ailments have been cataloged since the first herbal compendiums were recorded centuries ago. In this chapter, however, the focus is on single herbs and their specific uses, identification, and preparation.

HOMEOPATHIC USE OF BOTANICALS

Medical pioneer Samuel Hahnemann developed a radically different system of medicine nearly 200 years ago. Homeopathy is derived from the Greek words *homoios,* which means "similar," and *pathos,* which means "disease" or "suffering." The law of similars states that a substance that causes a set of symptoms in pharmacologic dosages can be used as a cure for similar symptoms (even if the etiologic agent is different), if that substance is given in a homeopathic dilution. Most homeopathic remedies are prepared from plant, mineral, and animal products. In homeopathic medicine, there is a perfectly matched simillimum (the most effective medicine) if the predominant symptoms of a disease or illness match the symptoms produced when the substance is taken in large dosages by a healthy individual. For example, the herb *Atropa belladonna,* which contains atropine, is poisonous. In excessive dosages, the herb causes death; in moderate dosages, it creates hot, feverish states; and in tiny (homeopathic) dosages, it can effectively treat certain types of fevers, viral syndromes, and inflammatory states.

A homeopathic dilution is created by taking a prepared tincture (mother tincture) of a botanical or an extract from nonplant sources and diluting it in a sequential or serial method. The difference in a homeopathic dilution is in its methodology. A homeopathic medicine must be succussed (shaken or agitated) mechanically or manually a prescribed number of times between the serial dilutions to be effective. The succussion method originally discovered by Hahnemann is said to "dynamize" the medicine. The succussion method is purported to affect the water molecules, creating a "memory" that the water molecules store in a lattice formation. This is similar to the storage of information on a magnetic disk or tape, except the signature resonance pattern is created from the interaction of the original tincture within the water's lattice structure. The dilution can range from a 1x potency, which is a decimal dilution of a given ingredient (one part mother tincture per nine parts solute), to a 1c (one part mother tincture to 99 parts solute), to an extremely dilute 200c (one part mother tincture per 99 parts solute, serially diluted 200 times). A high-potency dilution (serially diluted more than 30 times in the x potencies and more than 12 times in c potencies) would be taken much less frequently than a low-potency dilution.

To make a 30× homeopathic preparation of *Arnica montana,* one drop of the plant tincture is added to nine drops of pure water, and the mixture is succussed 50 to 100 times. Next, one drop from that solution would be added to nine drops of pure water and again succussed 50 to 100 times. This is repeated 30 times to yield the desired 30x homeopathic remedy. The number refers to the number of succussions and the letter to the ratio of the mother tincture to pure water.

The mechanisms by which homeopathy works have yet to be elucidated, even though it has been practiced effectively for several hundred years. In 1900, an estimated 15% of U.S. physicians were prescribing homeopathic remedies.[113] Recent studies have shown effective results in clinical trials using homeopathic medicines.[30,87,99] Mechanisms of action for many common pharmaceuticals also remain unknown. Many theories in medicine are still based largely on empirical observations rather than on theoretical understanding.

One herbal folk remedy for bruises, sprains, strains, and rheumatism in European and Native American medicine is topical application of the plant *Arnica montana* (leopard's bane). Consistent with the homeopathic principle, toxic quantities of the whole-plant extract of *Arnica* produce the same set of symptoms that it is intended to cure when administered internally in a homeopathic dosage or when the tincture or oil is applied topically to the affected area.

Arnica is contained in herbal and homeopathic dosages in numerous ointments, salves, and poultices for the treatment of trauma resulting from localized sprains, strains, or contusions. Controlled studies in Germany have shown that effective products for sprains from athletic activity use an ointment that contains homeopathic *Arnica*.[171]

TOPICAL APPLICATION

The earliest method of plant administration was topical application. Although many plants contain generalized moisture-enhancing properties, some were found to be particularly effective in ameliorating specific acute conditions when applied topically. Two methods are used to apply remedies to the skin. The endermatic method applies medicine on the skin without friction, as when applying a compress to the dermis and epidermis after an abrasion or laceration. The epidermatic method uses friction and is most effective with botanical oils, liniments, ointments, and medicated warm and cold friction rubs, primarily for subdermal contusions and trauma, to effect circulatory changes.[53]

Topical application of medicinal plants is useful for many conditions, including abrasions, lacerations, burns, insect bites, infections, rashes, and dermatoses. Other applications include contusions, varicosities, joint pain, inflammation, and musculotendinous aches, strains, and sprains.

Topical herbal remedies are applied with a poultice, a compress, a fomentation, or an ointment. Probably the most common, the poultice is used to apply a remedy to a skin area with moist heat. A poultice is prepared by bruising or crushing the medicinal parts of the plant to a pulpy mass, and then applying this to the affected area and covering it all with a moist heat source. If dried plants are used (or fresh plants if necessary), the materials are moistened by mixing with a hot, soft, adhesive substance such as moist flour or corn meal. A good way to apply a poultice is to spread the paste or pulp on a hot, wet cloth, which is wrapped around the affected area to help retain moisture and heat. The cloth is moistened with hot water as necessary. With irritant plants, such as those used in a mustard plaster, the paste is kept between two pieces of cloth to prevent direct contact with the skin. After the poultice is removed, the area is washed well with water to remove any residue. A poultice can be used to soothe, to irritate, or to draw impurities from the affected area, depending on which plants are applied.

A fomentation is a hot cloth soaked with an herbal infusion or decoction. Fomentations are generally less active than poultices. A cold compress is used for conditions that require an anti-inflammatory cure. A cold, infusion- or decoction-soaked cloth is applied to an area and then removed when the body's circulation has warmed the cloth to body temperature. The botanicals' active constituents determine what actions the external applications will impart. For example, a poultice with an astringent herb such as *Hamamelis* (witch hazel) has an entirely different effect from one made with a strong vasodilator and rubefacient, such as *Capsicum* (cayenne pepper).

Ointments are another method of topical administration. Most ointments are made in a base of petroleum jelly, stable vegetable oils, beeswax, or a combination of these. The extract from the desired botanical is suspended within the base to create a stable solid product. Topical botanical products have the same function as topical pharmaceutical ointments and are used to treat lacerations, abrasions, infections, and insect bites. Other uses for botanical topicals include hemostatic, anti-inflammatory, antihistamine, rubefacient, analgesic, emollient, and circulatory stimulant actions. Herbal poultices, compresses, and ointments deliver their active compounds transdermally, as do pharmaceutical topical agents.

The first uses of most medicinal plants were probably topical. In contemporary herbology, many of these plants are also used internally. Whole plants containing more than one ingredient with biologic activity generally invoke synergistic action of several components to produce the therapeutic action. Thus, most botanicals have multiple applications for therapeutic purposes. Herbalists and homeopaths treat trauma of the skin, muscles, tendons, ligaments, and joint tissue with a topical agent in ointment or poultice form and give the same medicine internally in minute (homeopathic) dosages to enhance the activity, as with concurrent use of *Arnica* ointment and homeopathic *Arnica*.

The major precaution in medical botany is to identify toxicity. Some of the most effective topical agents can be toxic if ingested. Most of these plants found in the wild could not be taken in sufficient dosages to be fatal before causing gastrointestinal (GI) upset. A tincture, herbal concentrate, or powdered version of the plant, however, could have deadly potential.

USE OF HERBAL MEDICINE IN THE WILDERNESS

Travelers in the wilderness can choose preprocessed herbal preparations or naturally available plants in the immediate vicinity. A surprisingly large number of minor medical conditions encountered in an outdoor setting can be treated with plants in that location. North American recreational areas are home to medicinal plants that have been used by Native Americans for centuries. Recreationists in desert, alpine, and river environments can find medicinal plants in abundance. Nearly all plants encountered during an alpine trek in North America have some medicinal property, as do many in tropical and subtropical regions.

Considerations for using herbal products in the wilderness are availability, ease of application, incidence of side effects, toxicity, spectrum of applicability, affordability, and effectiveness.

Availability and Application

If a condition can be improved by application of a local botanical growing in the immediate vicinity, the pharmacy is immediately available. Plants may be in season, plentiful, and easily harvested. Finding the appropriate plants can be challenging, however, depending on the location, the season, the traveler's familiarity with botanicals, and the type of medical condition. During mild seasons and at elevations conducive to plant growth in the continental United States, the chances of finding common plants are good. Otherwise, standardized commercial preparations of these herbs can be carried. These are packaged for long storage life, sanitary and convenient application, and standardization of active ingredients.

Hundreds of plants can be applied topically for a variety of conditions. Most of the readily available plants, even if properly identified, require some form of processing for the active constituents to be used fully. Furthermore, expertise in the field requires years of training by a knowledgeable botanist and herbalist. It also requires knowledge of plants' seasonal varia-

tions, ecologic niches, and precise identifications. However, a person who is neither a botanist nor a herbalist can gain a basic understanding of a few plant medicines that have a wide spectrum of applicability and a broad range of geographic distribution.

Side Effects and Toxicity

The American Association of Poison Control Centers annually reports plant ingestion as a significant category of accidental poisoning. In 1997, 5.6% of U.S. poisonings came from plants and mushrooms. Of the substances that were involved in pediatric poisonings, plants were responsible for 7.4% of exposures.

Side effects or toxic reactions from botanicals are rare. Among the botanicals covered in this chapter, toxicity is not a major consideration, although anything can be toxic when used excessively or indiscriminately. Many toxic plants produce GI distress, vomiting, or diarrhea before any severe neurologic or cardiorespiratory derangement. Often, toxic side effects are caused by one substance in a plant. When isolated, minute amounts of an alkaloid may be potentially dangerous, but when ingested in a form modified by other constituents, the altered drug effect allows tolerance of larger amounts of the toxic substance or substances.

As is true for any medication, medicinal plants should be applied appropriately, and dosages for internal use should not exceed recommendations. Pregnancy and nursing may be contraindications. Dosages for almost any herb can be found in numerous references.[123] Felter[53] stated that "as a rule, doses usually administered are far in excess of necessity and it is better to err on the side of insufficient dosage and trust to nature, than to overdose to the present or future harm or danger of the patient." In general, for the self-harvested herbs presented in this chapter, the dry, crushed, herbal adult dosage should be 1 teaspoon per pint of water; when the fresh herb is used, the amount should be twice that. Although no absolute law exists for administering medicines to children, Cowling's rule takes the child's age at the next birthday and divides by 24 to determine what fraction of the adult dosage should be given.[53]

Spectrum of Applicability

Most herbal medicines that have been cataloged and used historically are specifically indicated for one condition, although additional therapeutic effects have been noted over time. All the botanicals covered here have multiple uses. Comfrey *(Symphytum officinale)* may be used as a topical anti-inflammatory agent; it also has constituents that are effective for GI conditions when taken internally.[109] *Aloe vera* gel is an excellent topical agent for abrasions and burns; taken internally, the latex portion serves as an effective laxative.[109] *Calendula officinalis* has antimicrobial properties that make it an effective topical dressing for mild infectious conditions, whereas internally it has antipyretic effects.[109]

Affordability

If the herbalist collects plants and processes them personally, the cost is minimal. The purchase price of botanicals depends on the rarity and origin. Some exotic and rare botanicals from Asia and the Amazon rainforest demand a high price on the world market. *Panax ginseng* has long been regarded by Asian peoples as a prized herbal tonic and can cost hundreds of dollars per root, depending on the size, origin, and age. *Panax quinquefolius,* or American ginseng, can cost as much as $52 per pound, and was valued at $62 million as a cash crop in 1992.[17] Many exotic herbal and animal-derived medicines from China have prices as high as those of precious metals.

Most of the herbs produced in the continental United States and used for common ailments average 20 to 30 cents per dose (equivalent to 1 teaspoon of herbal tincture). Prices are not yet standardized. Quality control for production and supply and demand seem to dictate the cost of the mass-marketed herbal products. The best way to obtain a standardized product with a good quality-to-price ratio is to acquire the product from a botanical company that has been in business for at least 10 years and sells only to licensed health care practitioners.

NORTH AMERICAN PLANT MEDICINES

Ephedra *(Ephedra viridis)*

Description and Habitat. Common names for ephedra include Brigham Young weed, desert herb, Mormon tea, squaw tea, and teamsters' tea.

Ephedra species are shrubs with erect, strawlike branches found in desert or arid regions throughout the world and in the southwestern deserts of the United States (Fig. 60-2). The Chinese ephedra called Ma Huang, *Ephedra sinica,* is found throughout Asia; *E. distacha* is found throughout Europe; *E. trifurca* or *E. viridis* (desert tea), *E. nevadensis* (Mormon tea), and *E. americana* (American ephedra) are found in North America; and *E. gerardiana* (Pakistani ephedra) is found in Pakistan and India. The 2- to 7-foot shrubs grow on dry, rocky, or sandy soils. The broomlike shrub has many jointed green stems with two or three small scalelike leaves that grow at the joint of stems and branches.

Pharmacology. Ephedra is generally utilized for its alkaloid content, which tends to consist of ephedrine, pseudoephedrine, and norpseudoephedrine. The various species vary significantly in both alkaloid type and content. In *E. sinica,* the total alkaloid content can be from 3.3% to 20%, with 40% to 90% being ephedrine and the remainder pseudoephedrine.[47] The North

Figure 60-2. *Ephedra viridis.* (Courtesy Cascade Anderson Geller.)

American varieties, such as Mormon tea *(E. nevadensis)*, are reported to contain no ephedrine.

Ephedra's pharmacology centers on the actions of ephedrine. Ephedrine and pseudoephedrine are used widely in prescription and over-the-counter drugs to treat asthma, hay fever, and rhinitis.[61]

The central nervous system (CNS) effects of ephedrine are similar to those of epinephrine but are much milder, and the duration of action is much longer. The cardiovascular effects are increased blood pressure, cardiac output, and heart rate. In addition, ephedrine increases brain, heart, and muscle blood flow while decreasing renal and intestinal circulation.[61] Relaxation of bronchial, airway, and uterine smooth muscles also occurs.[61]

Pseudoephedrine has weaker CNS and cardiovascular system actions but has bronchial smooth muscle relaxation effects. Because it has fewer side effects, it is used more often than ephedrine for asthma.[61] Pseudoephedrine also demonstrates significant anti-inflammatory activity.[73,93] Per 100 g, the dry leaf of ephedra is reported to contain 5 g protein, 5810 mg calcium, and 500 mg potassium.[47]

Native American and European Medicinal Uses. Ephedra has been used extensively in the West and in Asia for upper respiratory conditions such as asthma, bronchitis, and hay fever. It has also been used to treat edema, arthritis, fever, hypotension, and urticaria.[34] It is said to be valuable as a diuretic, febrifuge, and tonic.[109]

The Navajo Indians applied the dried, crushed, long leaf of ephedra to syphilitic sores, and the Hopi Indians drank a tea from the branches and twigs of a related species for the same condition.[165] Other tribes used the ground and roasted root for making bread.[47]

Mormon tea is a folk remedy for colds, gonorrhea, headache, nephritis, and syphilis.[47] Mexicans mix the leaves with tobacco and smoke them for headaches.[47]

Modern Clinical and Wilderness Applications. Ephedra has proved to be an effective bronchodilator for treating mild to moderate asthma and hay fever. The common preparations include other herbs that have antitussive and expectorant effects, such as licorice *(Glycyrrhiza glabra)* and grindelia *(Grindelia camporium)*.

Ephedrine promotes weight loss.[123] Appetite suppression plays a role, but increased metabolic rate of adipose tissue is the main mechanism.[7] The weight reduction effects can be enhanced by up to 60% with the addition of methylxanthine.[48]

Clinically, standardized ephedra preparations are used because of the predictable alkaloid content. *E. sinica* extracts are available with a standardized 10% ephedrine alkaloid content. The dosage of a 10% alkaloid content extract is 125 to 250 mg three times a day.

In the wilderness, specifically the desert, the raw herb Mormon tea from *E. nevadensis* or *E. viridis* can be useful for hay fever, mild asthma, bronchitis, or upper respiratory infection (URI). These species contain minimal amounts of ephedrine and principally contain pseudoephedrine; thus, they can be used without some of the unpleasant side effects of the Asian species. They can also be used for mild fevers associated with influenza or URI.

The shrubs are typically found growing on dry, rocky, or sandy slopes. The leaves can be picked fresh or sun-dried for 6 to 8 hours and can be prepared as a steeped tea or an infusion. Generally, the dose should be the equivalent volume of 1 tablespoon of dried, crushed stems per 4 ounces of water, steeped for 10 minutes. The patient should not exceed a dosage of this amount given six times per day. Once harvested, the leaves can be kept for an indefinite period for later use if stored in an airtight container.

Toxicity. According to Duke,[47] an infusion of ephedra produced a "prompt and extensive contraction of uterine muscle when applied to smooth muscle strips of virgin guinea pig uteri." Ephedra may also elevate blood pressure. Frequent use may result in nervousness and restlessness. It should be used with caution if the patient has hypertension, heart disease, thyrotoxism, diabetes, or benign prostatic hypertrophy. Ephedra should not be used with antihypertensive or antidepressant medications.

Goldenseal *(Hydrastis canadensis)*

Description and Habitat. *Hydrastis* (Fig. 60-3) has a perennial root or rhizome that is tortuous, knotty, and creeping. The internal color is bright yellow, with numerous long fibers. The stem is erect, simple, herbaceous, and rounded, from 15 to 30 cm (6 to 12 inches) in height, becoming purplish and bearing two unequal terminal leaves. The leaves are alternately palmate with three to five lobes, hairy, dark green, and cordate at the base. The flowers, which are evident in early spring, are solitary, terminal, small, and white or rose colored.

The plant is a native of eastern North America and cultivated in Oregon and Washington. The parts used are the dried rhizome and roots.

Pharmacology. The alkaloids derived from *Hydrastis* are hydrastine (1.5% to 4%), berberine (0.5% to 6%), berberastine (2% to 3%), canadine, hydrastinine, and related compounds. Other constituents include meconin, chlorogenic acid, phytosterins, and resins.[123]

Native American and European Medicinal Uses and Folklore. Native Americans used *Hydrastis* extensively as an herbal med-

Figure 60-3. Goldenseal *(Hydrastis canadensis)*.

icine and clothing dye. The Cherokee Indians used the roots as a wash for local inflammations, as a decoction for general debility and dyspepsia, and to improve appetite. The Iroquois Indians used a decoction of the root for whooping cough, diarrhea, liver trouble, fever, sour stomach, flatulence, pneumonia, and heart trouble.[118]

Early European uses date back to 1793: in the Collections for an Essay towards a Materia Medica of the United States, Benjamin Smith Barton noted that *Hydrastis* was useful as an eyewash for conjunctival inflammation and as a bitter tonic. In the pharmacy of the 19th century (1830), goldenseal was listed among the official remedies in the first revision of the New York edition of the USP. It was listed in the USP until 1926 and recognized in the National Formulary until 1955.[74]

Modern Clinical and Wilderness Applications. Goldenseal is among the top sellers in the American herbal medicine market. It is used as an antiseptic, hemostatic, diuretic, laxative, tonic, and anti-inflammatory for inflammation of the mucous membranes. It has also been recommended for hemorrhoids, nasal congestion, sore mouth and gums, conjunctivitis, external wounds, sores, acne, and ringworm.[106]

Modern research into the active ingredients berberine and hydrastine has shown why some of the folk applications are effective. The most widely studied component is berberine. This isoquinoline alkaloid has demonstrated antibiotic, immunostimulatory, anticonvulsant, sedative, febrifugal, hypotensive, uterotonic, choleretic, and carminative (promoting the elimination of intestinal gas) activities.[123] Berberine has broad-spectrum antibiotic activity. The antimicrobial activity has been demonstrated on protozoa, fungi, and bacteria, both in vitro and in vivo. Antimicrobial action has been noted against *Staphylococcus, Streptococcus, Chlamydia, Corynebacterium diphtheriae, Escherichia coli, Salmonella typhi, Vibrio cholerae, Pseudomonas, Shigella dysenteriae, Entamoeba histolytica, Trichomonas vaginalis, Neisseria gonorrhoeae, N. meningitidis, Treponema pallidum, Giardia lamblia, Leishmania donovani,* and *Candida albicans*.[123] Berberine inhibits adherence of bacteria to host cells.[150]

Active ingredients in the crude botanical may be responsible for the wide-spectrum effectiveness of *Hydrastis*. The antifungal properties, for example, prevent the overgrowth of *Candida* that frequently occurs with use of other antibiotic therapies.

Other studies have shown the immunostimulatory activity of berberine-containing plants. Berberine increases blood flow through the spleen; improved circulation may augment the immune function of this lymphoid organ.[136] Berberine also activates macrophages.[99] Historically, berberine-containing plants have been used as febrifuges, and in rat studies they have an antipyretic effect three times as potent as that of aspirin.[123]

Plants such as goldenseal are very effective in treating acute GI infections. In several clinical studies, berberine has successfully treated acute diarrhea caused by *E. coli, S. dysenteriae, Salmonella, Klebsiella, Giardia,* and *V. cholerae*.[12,37,45,69,91,137,142] Berberine-containing plants, in addition to having antimicrobial properties, influence the enterotoxins produced by offending pathogens.[24,151,152]

GI illness is a major concern of travelers to areas with questionable sanitation. Both waterborne and food-borne bacterial and protozoal infections are concerns for persons in wilderness and Third World environments. Some experts recommend using a berberine-containing botanical source prophylactically at least 1 week before a visit to questionable areas and for 1 week after return.[123]

Various eye complaints involving the conjunctivae and surrounding mucous membranes have been effectively treated with forms of berberine extract. Recent studies point to the effectiveness of berberine in treating infection caused by *Chlamydia trachomatis*. Clinical trials found that a 2% berberine solution compared favorably to sulfacetamide. Although the symptoms resolved more slowly with the berberine extract, the rate of relapse was much lower in the berberine-treated group.[9,119]

A standardized form of *Hydrastis canadensis* is beneficial for generalized digestive disorders (acute dysentery, gastritis) and for infective, congestive, and inflammatory states (sinusitis, pharyngitis, stomatitis) of mucous membranes. A typical dosage depends on the source and method of the extract. For the previous conditions, the following dosages, three times a day, are recommended: dried root or as infusion, 2 to 4 g; tincture (1:5), 6 to 12 mL (1.5 to 3 teaspoons); or solid extract (4:1 or 10% alkaloid content), 250 to 500 mg. *Hydrastis* can also be used as a wash or rinse for conjunctivitis, sinusitis, and pharyngitis. Eye drops, nasal lavage, and gargle are applied in a 5% preparation of a 1:5 tincture, or 1 to 2 teaspoons of powdered herb in 8 ounces of water to create an infusion for application to inflamed mucous membranes. This can be repeated three times a day.

Toxicity. Berberine and berberine-containing plants are generally nontoxic. In recommended dosages, berberine-containing plants have not been shown to be toxic in clinical trials. The median lethal dose (LD_{50}) of berberine sulfate in mice is approximately 25 mg/kg, and in dogs, intravenous (IV) dosages up to 45 mg/kg do not produce lethal or gross toxic effects.[136] *Hydrastis* should not be used during pregnancy, and long-term ingestion may interfere with metabolism of B vitamins.

Arnica *(Arnica montana)*

Description and Habitat. *Arnica* is a perennial plant generally found in mountainous areas of Canada, the northern United States, and Europe. The plant reaches a height of 30 to 60 cm (1 to 2 feet) and generally contains from one to nine large daisy-like flowerheads, which bloom during summer months (Fig. 60-4).

Figure 60-4. *Arnica latifolia.*

Pharmacology. The flower is used both internally and externally for medicinal effects. The rootstock is used to make commercial preparations for tinctures and oils that are applied topically. The active constituents of the plant drug are flavonoids, volatile oils, and plant pigments (carotenoids).[166] Specific constituents include arnicine, formic acid, thymohydroquinone, lobelamine, and lobeline (piperidine alkaloid).[30]

Native American and European Medicinal Uses and Folklore. The Catawa Indians administered the tea of *Arnica* roots to treat back pain. In Europe, the flowerheads have been used since the 16th century as an application for bruises and strains.[164] European *Arnica* was included in the USP from the early 1800s until 1960 and was recognized for its effects on the healing of bruises and sprains.

Specific instructions given in the *American Dispensory* in 1922 listed *Arnica* as effective for "muscular soreness and pain from strain or overexertion; advanced stage of disease, with marked enfeeblement, weak circulation, and impaired spinal innervation; . . . tensive backache, as if bruised or strained; [and] . . . headache with tensive, bruised feeling and pain on movement."[53] *Arnica* in concentrated tincture form has been a popular but not necessarily safe medicine to treat inflammatory swellings and to relieve the soreness of myalgia and the effects of bruises and contusions. Dosages above the therapeutic range cause vagal inhibition when ingested and may cause toxicity if the concentrated tincture is applied topically. Therefore, the most common use has been fomentation of the flowers for topical applications in treatment of strains and sprains.

Modern Clinical and Wilderness Applications. Contemporary use of *Arnica montana* is generally limited to topical commercially prepared ointments and salves, in conjunction with internal homeopathic (low-dose) use for the same indications. Although its alkaloid (arnicine) and volatile oil (thymohydroquinone) are both relatively toxic, the actions of these constituents are extremely useful in resolving contusions and soft tissue injury. Most ointments are found to contain a 1× homeopathic dilution of *Arnica* tincture, which is about 4% by volume. Oral dosage is given in homeopathic potencies of 6× to 200c, depending on the severity of the condition.

For application in the wilderness, most naturopathic first-aid kits include both the ointment and the oral homeopathic forms of *Arnica*. For direct use of the plant in treating minor sprains and strains, 2 teaspoons of the dried flower tops can be steeped in 1 cup of water for 10 minutes, and the infusion can be applied in a cold compress to the affected area. This should be repeated each 2 hours in addition to standard first-aid procedures. The infusion lasts a day if refrigerated and a few hours if not; therefore, it is best to use a fresh infusion whenever possible. In addition, if available, the oral homeopathic preparation (30× to 200c) should be taken three times daily until the swelling is reduced significantly. A topical ointment can be applied every 2 to 3 hours for this condition in place of the compress.

According to Weiss,[165] *Arnica* is safe and effective for topical contusions and for stimulating granulation and epithelialization. A tablespoon of tincture is added to 500 mL of water, and the gauze compress is then placed on the wound. This stimulates local circulation and acts on the peripheral vasculature. After granulation has occurred, ointments may be applied.

Toxicity. *Arnica* tincture or infusion can be toxic if the concentration is too high. Undiluted tincture should not be used internally or in compress form over an open wound. Vagus nerve inhibition is the primary toxic effect; GI irritation is also noted. Toxic reactions include gastric burning; nausea; vomiting; headache; decreased temperature; dyspnea; cardiovascular collapse; convulsions; motor, sensory, and vagal paralysis; and death.[30]

Garlic *(Allium sativum)*

Description and Habitat. Garlic is a member of the lily family. It is a perennial plant cultivated worldwide (Fig. 60-5). The garlic bulb is composed of individual cloves enclosed in a white skin. The medicinal herb is found in the bulb and is used either fresh or dehydrated. Garlic oil, which also has medicinal value, is obtained by steamed distillation of the crushed fresh bulbs.[106]

Pharmacology. The medicinal compounds in garlic generally contain sulfur and have been the subject of most research on garlic. Two primary compounds are an odorless chemical called alliin and the enzyme allinase, which begins a cascade of chemical reactions when the garlic clove is cut, crushed, or bruised. Alliin is converted to allicin, which is responsible for the characteristic odor of garlic. Allicin is strongly antibacterial and considered to be the major source of the antimicrobial effects of garlic. Breakdown products of allicin include diallyl sulfide, disulfide, and trisulfide. Heat speeds up the reaction, so cooked garlic and steamed distilled garlic oil contain little or no allicin. About 0.1% to 0.36% of the volatile oils in garlic is composed of sulfur-containing compounds (e.g., allicin, diallyl sulfide, diallyl trisulfide). These volatile oils are considered to be responsible for most of the pharmacologic properties of garlic. Other constituents of garlic include s-methyl-L-cysteine sulfoxide, protein (16.8% by dry weight), a high concentration of trace minerals (particularly selenium and germanium), vitamins, glucosinolates, and the enzymes allinase, peroxidase, and myrosinase.[123,131]

Native American and European Medicinal Uses and Folklore. Throughout history, garlic has played an important part in medicinal herbology. Clay garlic bulbs dating back to 3750 BC were found in Egypt. Preserved garlic bulbs were discovered in the tomb of Tutankhamen. An entire basket of these bulbs from

Figure 60-5. Garlic blossom (*Allium* species). (Courtesy Cascade Anderson Geller.)

the tomb of Kha at Thebes is in the Turin Museum. The Greek historian Herodotus recorded that an enormous amount of money was spent on garlic for the builders of the great pyramids. One of the earliest Sanskrit manuscripts, the Bower manuscript, devotes its entire first section to garlic, describing its legendary origins. It says that garlic keeps in order the three fluids and can cure thinness, weakness of digestion, lassitude, coughs, inflammation of the skin, piles, glandular swellings in the abdomen and enlargement of the spleen, indigestion, constipation, excessive urination, worms, wind in the body (rheumatism), leprosy, epilepsy, and paralysis.

Within the traditional medical circles of Greece and Rome, medieval Europe, and the Far East, similar claims may be found. Galen, Dioscorides, and Aristotle extolled garlic as an excellent medicine. Hippocrates recommended garlic as a diuretic; to regulate digestion; to treat bowel pains, inflammations, and infections; and to regulate menstruation. Early Chinese and European herbalists used garlic as a heating and drying agent, and therefore to prevent and cure diseases arising from cold, poisons, excesses of diet and drink, and sluggish metabolism.

Pasteur noted garlic's antibiotic properties in 1858. Albert Schweitzer used garlic in Africa to treat amebic dysentery. Garlic was also used as an antiseptic to prevent gangrene during both world wars.

Modern Clinical and Wilderness Applications. The pharmacologic effects of garlic are based on its activity as a hypoglycemic and hypolipemic regulating agent,* anticoagulant,† antihypertensive,[111,132] antimicrobial,[3,4,32,55,100,110,125,154,157] detoxifier of heavy metals,[2] and immune system modulator.[88]

Animal and human studies have substantiated that garlic lowers serum cholesterol and triglyceride levels and increases the amount of high-density lipoproteins. Dietary atherosclerosis was significantly reduced in rabbits fed garlic consistently for weeks; also, an extract of garlic and onions was more effective than clofibrate against hyperlipidemia and subsequent lipid deposition within the aorta.[20] After 4 months of feeding rabbits a high-cholesterol diet, the average lipid content in the aorta of the control animals rose from 5.95 to 13.75 mg/100 g dry weight. Animals taking clofibrate for 4 months had 7.95 mg and garlic-fed animals 6.23 mg/100 g dry weight of lipid content in the aorta.[20] Other studies of experimental atherosclerosis in rabbits support these findings.[83,95] Decreased atheromatous lesions seem to be a consistent finding in rabbits fed high-cholesterol diets supplemented with garlic.

Of various sulfur-containing amino acids isolated from garlic, s-methylcysteine and s-allylcysteine exert the greatest antilipidemic effects.[81] Components of garlic can combine with the sulfhydryl group, the functional part of coenzyme A that is necessary for biosynthesis of fatty acids, cholesterol, triglycerides, and phospholipids. The lipid-lowering effect may best be attributed to inactivation of the sulfhydryl group.[8] In vitro and in vivo tests show reduced conversion of acetate into cholesterol by liver tissues.[35] Because sulfhydryl groups are involved at all levels of metabolic activity, the impact of garlic could be more extensive. Studies suggest that garlic may lower blood pressure by acting the same way prostaglandin E_1 does, by decreasing peripheral vascular resistance.[128]

*References 6, 8, 13, 19, 21, 25–27, 35, 36, 68, 81, 82, 85–87, 89, 90, 95, 96, 126.
†References 5, 18, 23, 24, 28, 59, 84, 110, 124, 138, 147.

As a nutritional supplement, garlic is composed of magnesium, iron, copper, zinc, selenium, calcium, potassium chloride, germanium, sulfur compounds, amino acids, and vitamins A, B_1, and C. Garlic increases the body's capacity to assimilate thiamine by enhancing its absorption. Thiamine is a key part of the co-carboxylase enzyme system, which has beneficial effects on liver cells; this may explain why garlic offers prophylaxis against liver and gallbladder damage. In one study, garlic was shown to protect hepatocytes in tissue culture from the damage of carbon tetrachloride.[128]

Antioxidant activity has been attributed to garlic and garlic derivatives. The free radical scavenger action of garlic may be explained by its germanium, glutathione, selenium, and zinc content. The last three are key components of the antioxidant enzyme superoxide dismutase and glutathione peroxidase. Animal studies show that feeding garlic oil enhanced physical endurance in normal rats and also reduced the decrease in physical endurance induced by isoproterenol, a synthetic catecholamine that induces necrosis of the myocardium.[140]

Garlic inhibits platelet aggregation in animals; similar effects can be demonstrated in vitro and in vivo in humans.[43,147] An antiplatelet extract of garlic, ajoene, was found to potentiate the antithrombotic effect of anti-inflammatory drugs. Under fasting conditions, inhibition of platelet aggregation by garlic or its extracts is dosage related.[148]

The garlic effect may be linked to inhibition of thromboxane synthesis or to altered properties of the plasma membrane. Methyl (2-propenyl) trisulfide, another component of garlic, is 10 times more potent as an inhibitor of platelet aggregation than diallyl disulfide or trisulfide.[5] Thrombocyte aggregation inhibition is enhanced by two other compounds, 2-vinyl-1,3-dithiene and allyl-1,5-hexidienyl-trisulfide.[16]

Garlic and its juice or oil also enhance fibrinolysis.[22] In a double-blind placebo-controlled trial, cycloalliin, a component of garlic, was given to volunteers and patients after myocardial infarction and significantly increased fibrinolysis $1^1/_2$ hours later.[51] Chutani and Bordia[38] observed that the increase took place $6^1/_2$ to 12 hours after garlic intake. Daily garlic ingestion for 1 month generated a 72% to 85% increase in fibrinolysis in patients with ischemic heart disease.[135]

The pharmacologic versatility of garlic is best reflected by its antiviral, antifungal, antiprotozoan, antiparasitic, and antibacterial activities.[3,4,32,55,125,153,170] Laymen are credited with being the first to describe the scientific basis for the medicinal use of garlic extract.[168] Huddleson and colleagues[77] and Cavallito and Bailey[33] demonstrated in 1944 that garlic juice and allicin at low concentrations inhibited the growth of *Staphylococcus, Streptococcus, Bacillus, Brucella,* and *Vibrio* species. Recent studies using serial dilutions and filter paper disk techniques have shown that fresh garlic, powdered garlic, and vacuum-dried preparations were effective antibiotic agents against many bacteria, including *Staphylococcus aureus,* α- and β-hemolytic *Streptococcus, E. coli, Proteus vulgaris, Salmonella enteritidis, Citrobacter,* and *Klebsiella pneumoniae.*[123] These studies compared the antimicrobial effects of antibiotics, including penicillin, streptomycin, chloramphenicol, erythromycin, and tetracycline, with those of garlic. Besides confirming garlic's well-known antibacterial effects, studies demonstrated its effectiveness in inhibiting the growth of some antibiotic-resistant bacteria.[1,50,143]

Garlic has also demonstrated significant antifungal activity against a wide range of fungi.[1,3,57,78,117,120,130,139,160] From a

wilderness perspective, inhibition of fungi that can affect the skin (*Microsporum, Trichophyton, Epidermophyton,* and *Candida albicans*) can be significant. Garlic juice applied topically is an effective alternative in treating fungal skin diseases.[3] Garlic compares well with nystatin, gentian violet, and six other reputed antifungal agents to treat *C. albicans*.[1,120,130,139]

Garlic has long been associated with prophylaxis against influenza virus. In vivo studies with mice revealed that garlic administration protected mice against intranasal inoculation with influenza viruses and enhanced reproduction of neutralizing antibodies after vaccine administration.[125] In vitro studies have shown that garlic has antiviral activity against influenza B virus and herpes hominis virus type I.[155] Preliminary studies have revealed significant enhancement of natural killer (NK) cell activity in humans administered raw or cold, aged whole-clove garlic preparations daily for 3 weeks.[88] The antiviral activity of garlic in humans may be secondary to the direct toxic effect on viruses and enhanced NK cell activity that destroys virus-infected cells.

Wilderness Medical Applications. The use of garlic in the outdoor setting can be extensive. Its use as a food should be encouraged despite its odor, particularly in people with elevated cholesterol levels, heart disease, hypertension, diabetes, asthma, fungal infections, respiratory infections, and GI disorders (intestinal parasites, dysentery). A macerated garlic poultice and garlic slices serve as topical agents for fungal infections, ulcerated wounds, pyoderma, and other skin infections. The poultice can be used directly on the dermatologic problem, and as a suppository it can be used to treat vaginitis, particularly infections caused by *C. albicans*. For this application, one to two fresh chopped cloves can be made into a poultice. This should be kept on the affected site for several hours and changed at least once every 6 hours with a fresh preparation. If the garlic causes epidermal irritation, its use is discontinued.

Prophylactic use during the flu season can reduce the incidence of infection. Within the first 48 hours of onset of a flu or URI, one or two cloves can be consumed with a carbohydrate source to prevent stomach irritation. Alternatively, two or three oil-of-garlic capsules can be taken. For persons concerned about the social segregating aspect, extracts that preserve the allicin content but remain odorless can be used.

Toxicity. For the vast majority of individuals, garlic is nontoxic at usual dosages. However, some people develop allergic contact dermatitis or irritation of the digestive tract. Apparently, they are unable to detoxify allicin and other sulfur-containing components. Prolonged consumption of large amounts of raw garlic by rats results in anemia, weight loss, and failure to grow.[127]

Ginger *(Zingiber officinale)*

Description and Habitat. Ginger is an upright perennial herb with tuberous rhizomes, from which an aerial stem grows to 1.5 m (5 feet) in height. It is native to southern Asia, although it is cultivated in the tropics. Extracts and dried ginger are produced from dried unpeeled ginger; peeled ginger loses much of its essential oil content.[159]

Pharmacology. Ginger is composed of a rich variety of nutrients and enzymes. The general composition is starch (50%); protein (9%); lipid (6% to 8%), composed of phosphatidic acid, lecithin, free fatty acids, and triglycerides; protease (up to 2.26%); volatile oils (1% to 4%), the principal components of which are three sesquiterpenes (bisabolene, zingiberene, zingiberol); vitamins, especially niacin and vitamin A; and resins.[159]

Native American and European Medicinal Use. *Zingiber officinale* is native to southern Asia and tropical Africa. Therefore, it did not have a role in the early herbal preparations of European and Native American herbal medicine.

Modern Clinical and Wilderness Applications. Clinical use of ginger for anti-inflammatory action, cholesterol-lowering effects, and relief of dizziness and motion sickness is noted in herbal texts.[53,54,65–67,97,122,144,148,149,159] A choleretic effect (the promotion of bile flow to the gallbladder and small intestine) and the conversion of cholesterol into bile acids are enhanced by ginger ingestion and may be responsible for its overall cholesterol-lowering effect.

An early eclectic medical text listed ginger as a local stimulant, sialogogue, diaphoretic, and carminative.[53] Powdered ginger in a large quantity of cold water taken before sleep frequently "breaks up" a severe cold, and a hot infusion of ginger tea is a popular remedy for similar use to mitigate the pains of dysmenorrhea.[53] Ginger may relieve painful spasmodic contractions of the stomach and intestine. The anti-inflammatory action of ginger is thought to be caused by potent inhibition of inflammatory compounds, such as prostaglandins and thromboxanes.[97] Ginger is also known to contain strong plant proteases such as bromelain, ficaine, and papain, which may explain some of its anti-inflammatory action.[159]

Ginger has been used historically for major GI complaints. It is generally regarded as an excellent carminative (promoting elimination of intestinal gas) and intestinal spasmolytic.[123] One of the most noted uses of ginger in contemporary herbal medicine that applies to wilderness medicine is its action on the symptoms of motion sickness and seasickness.[65,66,122] Ginger is also a significant antiemetic. It has long been used for treatment of nausea and vomiting associated with pregnancy. The efficacy of ginger has been confirmed in hyperemesis gravidarum, a severe form of nausea and vomiting during pregnancy. Ginger root powder at a dosage of 250 mg four times a day brought a significant reduction in both the severity of nausea and the number of attacks of vomiting during pregnancy.[54] To treat motion sickness and vertigo, two 500-mg capsules of powdered ginger root are eaten 20 to 30 minutes before the precipitating event. The same dose is used for the nausea of pregnancy during the acute attack. The raw ginger root can be grated using 1 teaspoon in 4 ounces of water, steeped for 10 minutes, and taken every 30 minutes until the symptoms of motion sickness abate.

Toxicity. There appears to be no toxicity associated with ginger root ingestion.

Comfrey *(Symphytum officinale)*

Description and Habitat. Comfrey (Figs. 60-6 and 60-7) is a perennial herb with a stout spreading root that is divisible for propagation. Comfrey grows about 1 m (3 feet) high and has coarse, bristly, oblong, lanceolate leaves. The tubular flower can be purplish, blue, white, red, or yellow (Fig. 60-8). About 25 *Symphytum* species are described; they are indigenous to coun-

Figure 60-6. Comfrey *(Symphytum officinale),* plant.

Figure 60-7. Comfrey *(Symphytum officinale),* leaf.

tries around the Mediterranean Sea and in northern Asia. Comfrey is typically found in moist meadows and other wet places in the United States and Europe.

Pharmacology. The chemical constituents of *Symphytum officinale* roots include carbohydrate, predominantly sucrose; the amino acids serine and asparagine; the phenolic acids chlorogenic acid, caffeic acid, and *p*-coumaric acid; the alkaloids choline and allantoin; and the pyrrolizidine alkaloids viridiflorine, echinatine, heliosupine, symphytine, echimidine, and lasiocarpine.[161] The most concentrated (0.88% to 1.71%) alkaloid, allantoin, is generally credited with comfrey's beneficial effects.

Figure 60-8. Comfrey *(Symphytum officinale),* flower. (Courtesy Cascade Anderson Geller.)

Native American and European Medicinal Uses and Folklore. In Europe, comfrey is a common perennial grown in the garden for animal fodder. Russian comfrey is often promoted as a medicinal herb for use as a tonic. Comfrey is also cultivated in Japan as a green vegetable, and a tonic made from it has been used in American herbal medicine for hundreds of years.[102]

Comfrey has long been known as an external agent for rehabilitation of musculoskeletal and orthopedic injuries. Its former name, bone knit, derives from the external use of poultices of leaves and roots, which were believed to help heal burns, sprains, swellings, and bruises. Comfrey has been claimed to heal gastric ulcers and hemorrhoids, suppress bleeding, and relieve bronchial congestion and inflammation.[14] The healing action of a poultice derived from the roots and leaves is probably related to the presence of allantoin, an agent that promotes cell proliferation. The underground parts contain 0.6% to 1.3% allantoin and 4% to 6.5% tannin.[31,116] Comfrey extracts applied topically have been reported to heal wounds and bones in about half of the normal time. In herbology, a general rule is that if anything is broken, use comfrey.[169] Herbalists have also found that the allantoin concentration from a fluid extract of comfrey

can increase the rate of wound healing of lacerations sufficiently to avoid the use of sutures.[167]

In European folklore, comfrey was regarded as an herb having unsurpassed ability to heal any injured or broken tissue. The mucilage (gelatinous mucopolysaccharide) of the comfrey root was named "the great cell proliferator," helping new flesh and bones to grow. Comfrey was one of the main herbs found in any poultice or fomentation. European herbalists considered comfrey exceptional for coughs and soothing inflamed tissues. Comfrey is effective for treating upper respiratory inflammation and has been used successfully to treat hemorrhagic conditions of the lungs.

Modern Clinical and Wilderness Applications. Comfrey lotions and salves containing 0.5% to 2.5% allantoin have been used for sprains, strains, and contusions. In the 1980s, comfrey became controversial because of potential hepatotoxicity. Members of the family Boraginaceae *(Heliotropium, Symphytum)* contain a variety of related pyrrolizidine alkaloids reported to cause hepatotoxicity in animals. Although no hepatotoxic episodes from ingestion of comfrey have been reported in humans, the potential exists, so caution is advised when using comfrey for internal consumption.[102] Topical use of comfrey products as yet poses no concern for toxicity.

As a topical agent after acute trauma such as musculoskeletal injuries, strains and sprains, or contusions, comfrey is an exceptional medicine.[15] A prepared gel of comfrey with a standardized allantoin concentration should be carried during travel or camping expeditions in the wilderness.

The raw herb can be used if the plant is nearby. The herb is readily identifiable, but it should not be confused with foxglove *(Digitalis purpurea)* and should be used with caution when taken internally in its raw state. For use in a poultice or compress, the leaves may be picked damp, macerated, and applied topically for up to 24 hours.

Toxicity. Comfrey is not recommended for routine internal ingestion. Animal studies indicate that hepatic damage is an eventual outcome if the herb is consumed over a long period.

Aloe *(Aloe vera)*

Description and Habitat. The aloe is a perennial plant native to South and East Africa and is also cultivated in the West Indies and other tropical and temperate areas. The leaves, which emerge from a central rosette produced by a central fibrous root, are 30 to 60 cm (1 to 2 feet) long, narrow, fleshy, and light green with spiny teeth on the margins (Fig. 60-9). Aloe is easily cultivated as a houseplant and can be grown in a sunny warm spot with good drainage.

The genus *Aloe* comprises more than 300 species, which are members of the Liliaceae (lily) family. *Aloe* species are perennial succulents native to Africa. They are not cacti and should not be confused with American aloe, the century plant.

Pharmacology. Two important products are derived from aloe: a gel and a latex. Aloe gel is a clear gelatinous material extracted from the mucilaginous cells found in the inner tissue of the leaf. It is obtained by crushing the leaves and straining the mass repeatedly to remove cellular debris. The result is a clear gel, which is the product most frequently used in the health food and cosmetic industries. It is generally devoid of anthraquinone glycosides. A variety of compounds have been identified in *Aloe* species, including polysaccharides, tannins, organic acids, enzymes, vitamins, minerals, saponins, and steroids.[102]

Figure 60-9. *Aloe claviflora.* (Courtesy Cascade Anderson Geller.)

The bitter yellow latex of aloe contains cathartic anthraquinone glycosides, mostly barbaloin, as the active constituents. The concentrations of the glycosides vary with the type of aloe, ranging from 4% to 25% of aloe in concentration. The water-soluble fraction of aloe is called aloin and is a mixture of active glycosides. Cathartics have been derived from extracts of the latex and can create strong purgative effects by stimulating the large intestine.

Native American and European Medicinal Uses and Folklore. Fresh *Aloe vera* gel is well known for its domestic medicinal values.[62,105,121,129] It has been dubbed the burn plant, the first-aid plant, and the medicine plant. When fresh, the gel relieves thermal burns and sunburns and promotes wound healing. It also has moisturizing and emollient properties. Because of these effects, aloe is widely used as a home remedy.

Aloin and other anthraquinone derivatives of aloe are extensively used as active ingredients in laxative preparations. Aloin is also used as an antiobesity preparation.[106] Aloe or aloin extracts are also used in sunscreens and other cosmetic preparations, and in drugs for moisturizing, emollient, or wound-healing purposes.

In folk medicine, aloe is used for condylomas, warts, abnormal skin growths, and cancers of the lip, anus, breast, larynx, liver, nose, stomach, and uterus.[47] Other folklore suggests that parts of the plants be chewed to purify the blood. The pulp is said to possess wound-healing hormonal activities and "biogenic stimulators" and is used for intestinal ailments, sore throat, and ulcers. In India it is used to treat piles and rectal fissures. Slukari hunters in Africa's Congo basin rubbed their bodies with the gel to eliminate the human scent, making them less likely to disturb prey. During epidemics of influenza, Lesotho natives take a public bath in an infusion of *Aloe latifolia*.[47]

Modern Clinical and Wilderness Applications. Although numerous claims have been made for aloe gel, its most common lay use is in the treatment of minor burns and skin irritations. In 1935, a report described the use of aloe in the treatment of radiation-induced dermatitis.[40] This study followed a 5-week course of topical applications of either the whole leaf or the leaf macerated into gel, resulting in complete wound healing after 4

months. In 1937, studies used a calamine- and lanolin-based aloe preparation to treat skin irritations resulting from burns, pruritus vulvae, and poison ivy. The results suggested that aloe stimulated tissue granulation and accelerated wound healing.[102]

Barnes[10] evaluated the effect of 5% aloe ointment on sandpaper-abraded fingertips and found that the wound-healing rate was two to three times that of control subjects, as measured by decreased electrical potential of the wound. Other studies measured tensile strength of the healed surgical wounds of mice. Healing occurred within 9 days, an improvement over the results in control mice.[63]

Studies of antibacterial activity of aloe extracts have been attempted several times, yielding mixed results. In 1963, studies of the antibacterial effect of macerated *Aloe vera* gel found no activity against *S. aureus* and *E. coli*.[56] Other studies have determined that *Aloe chinensis* is effective against *S. aureus, E. coli,* and *Mycobacterium tuberculosis,* although *Aloe vera* showed no inhibitory effect.[64] The latex possesses in vitro activity against several pathogenic strains of bacteria, although the whole leaf minus the latex from the leaf epidermis and mesophyll of aloe showed no activity.[107] Two commercial preparations of aloe gel were found to exert antimicrobial activity against gram-negative and gram-positive bacteria and *C. albicans* when used in concentrations greater than 90%.[71]

The moisturizing effect of aloe may be beneficial in the treatment of burns. The healing process may be related to mucopolysaccharides along with sulfur derivatives and nitrogen compounds in the gel, but this has not been well substantiated.[101] In attempts to document the anti-inflammatory effects of aloe, a 1976 study found that *Aloe vera* had bradykinase activity in vitro, but this was not confirmed in vivo.[58]

Evidence for the internal use of aloe has been limited to studies involving mucous membrane tissue repair. Corneal ulcers treated with aloe extracts had more healing, less cellular reaction, and fewer signs of irritation than did control groups.[102] Topical application of *Aloe vera* gel after periodontal flap surgery reduced postoperative pain more than the saline control did, and swelling of the treated tissue was less marked than with the control.[72]

Because of easy recognition and administration, use of the aloe plant in the wilderness environment is practical. The wild plant can yield an excellent preparation for dermal abrasions, cuts, and superficial wounds. A leaf cut from the base of a healthy plant can be conveniently carried. This allows the gel to remain intact, protected by the outer skin of the leaf. It can be squeezed from the inside through the cut portion directly onto superficial wounds with or without a gauze dressing. A standardized preparation of *Aloe vera* may be used as an antibacterial agent and an emollient for superficial wounds or dermatitis.

In the event of constipation, the mixture of aloe gel and latex can be scraped or squeezed from the leaf cortex and ingested, 1 tablespoon three times daily, or until a mild laxative effect is noted. A gel and latex mixture produces less cathartic effect than latex alone does. Because of the bitterness of the gel and latex, the mixture should be taken with food or a flavored beverage.

Toxicity. Because of its cathartic effects, oral aloe is not advised if gripping pain is associated with constipation. Aloe taken orally is contraindicated in pregnancy. Otherwise it has no reported toxicity.

Figure 60-10. Plantain *(Plantago major).*

Plantain *(Plantago major)*

Description and Habitat. The common broadleaf plantain (Fig. 60-10) is a familiar perennial "weed," found along roadsides and in meadowlands. Plantain belongs to the order Plantaginaceae, which contains more than 200 species, 25 or 30 of which have a domestic use. The plant is a small weed with a rosette of ribbed leaves and small projecting seed stalks. Its seeds, known as psyllium seeds in North America, resemble those of another species, *Plantago psyllium*. The leaves contain 84% water, 2.5% protein, 0.2% fat, and 14% carbohydrate; trace amounts of calcium, phosphorus, iron, sodium, and potassium; and β-carotene, riboflavin, niacin, and ascorbic acid. Biochemically identified compounds include allantoin, adenine, baicalein, baicalin, benzoic acid, chlorogenic acid, choline, cinnamic acid, ferulic acid, L-fructose, fumaric acid, gentisic acid, D-glucose, *p*-hydroxybenzoic acid, indicain, lignoceric acid, neochlorogenic acid, oleanolic acid, plantagonine, planteose, saccharose, salicylic acid, scutellarein, sitosterol, sorbitol, stachyose, syringic acid, tyrosol, ursolic acid, vanillic acid, and D-xylose.[47]

Native American and European Medicinal Uses and Folklore. Historically, plantain has long been used for stings, bites, and irritations from venomous insects and reptiles. The folk medicine of the eastern United States suggests using crushed plantain leaves to stop the itching of poison ivy. It has also been reported to help relieve toothache. Ancient herbalists maintained that plantain had "refrigerant" (imparting a cooling sensation to the mucosa and allaying thirst), diuretic, and astringent properties. When the leaves are applied to a bleeding surface wound, hemorrhage lessens. In the highlands of Scotland, plantain is still called *slan-lus,* or plant of healing.

In the United States, plantain has been known as snake weed, from the belief that it is effective for bites from venomous creatures. Felter[53] noted that "the crushed leaves were very effective for the distressing symptoms caused by puncture by the horny appendages of larvae of *Lepidoptera* and the irritation produced by certain caterpillars, as well as the stings of insects and bites

Figure 60-11. Chamomile *(Matricaria chamomilla)*.

of spiders." In Native American folklore, the plant was known as "white man's foot," in reference to its trait of growing in the settlements of white men. The Shoshoni Indians heated the leaves and applied them in a wet dressing for wounds.[166]

Modern Clinical and Wilderness Applications. Plantain is readily available in the recreational areas of North America. This plant is extremely useful for various superficial wounds, abrasions, stings, and bites of mildly venomous insects. The constituents in the crushed leaves have an antihistaminic effect and anesthetic quality. In the event of a tooth fracture, a compress or poultice of $^1/_2$ teaspoon of fresh leaves may be used on the tooth's exposed nerve root. The seeds of the plantago plant are useful for spastic colon, an effect that appears to be related to their mucilaginous properties. Psyllium seeds, known on the Asian continent as flea seed husk, are often used as a bulk laxative. The seeds are collected from the stalk, and 1 teaspoon of fresh seeds in 4 ounces of water is taken twice a day for mild constipation. Water should be ingested throughout the day to alleviate the condition and assist the laxative effect.

Because of its astringent quality, an infusion of the leaves is recommended to treat diarrhea. The preparer pours 1 pint of boiling water on 1 ounce of the herb and leaves it in a warm place for 20 minutes. After straining and cooling, $^1/_2$ cup is ingested three or four times a day.

Toxicity. No known toxicities are attributed to *Plantago*.

Chamomile *(Matricaria chamomilla)*

Description and Habitat. Chamomile (Fig. 60-11) is a low-growing perennial with a hairy prostrate branching stem. It blooms late in July through September and is found growing throughout North America and Europe. The name *chamomile* is derived from the Greek *chamos* (ground) and *melos* (apple), which refer to the plant's low growth and the applelike scent of its fresh blooms.[145] The flower head is about 2.5 cm (1 inch) in diameter, has a conical receptacle, and is covered by yellow disk-like flowers surrounded by 10 or 20 white, down-curving ray flowers.

Pharmacology. The most important chemicals associated with chamomile are the volatile oils containing tiglic acid esters, chamazulene, farnesene, and α-bisabolol oxide. These volatile oils are destroyed if the herb is boiled.[146]

Native American and European Medicinal Uses and Folklore. A distinction should be made between German and Roman chamomile, although they have been used interchangeably for centuries. German chamomile is preferred on the European continent, whereas Roman chamomile has been used widely in Great Britain. In the United States, German chamomile is by far the more widely consumed of the two species.[112]

German chamomile has a long tradition as a folk or domestic remedy. It has been used as an external compress or fomentation for gout, sciatica, inflammations, lumbago, rheumatism, and skin ailments. Infusions, decoctions, and tinctures have long been used internally to treat colic, convulsions, croup, diarrhea, fever, indigestion, insomnia, teething, toothaches, and bleeding or swollen gums. Historically, Roman chamomile was used similarly.[47,106] Chamomile is also a folk remedy for cancer.

Modern Clinical and Wilderness Applications. The principal biochemical constituent of chamomile is chamazulene. It is found in both species of chamomile and is reported to have antihistaminic properties.[52] Both histamine release and inhibition of histamine discharge have been considered mechanisms for the potential antiallergic action of chamazulene.

In Germany, chamomile products include tinctures, extracts, teas, and salves, widely used as anti-inflammatory, antibacterial, antispasmodic, and sedative agents.[112] Studies have shown that both chamazulene and α-bisabolol have anti-inflammatory activity. Chamazulene may constitute as much as 5% of the essential oil. Other studies have shown that α-bisabolol has a protective effect against peptic ulcer, as well as antibacterial and antifungal effects. α-Bisabolol has also reduced fever and shortened the healing time of skin burns in laboratory animals.[44] Most commercial European chamomile preparations have been standardized with regard to the chamazulene and α-bisabolol content.[160]

According to Rudolph Weiss,[165] one action of chamomile is to reduce gastric motility and secretions, which would alleviate colic and painful spasm. About 20 flavones and flavonols, such as apigenin, are found in the aqueous portion of the distillation process. These are three times as effective at spasmolytic activity as is the opium alkaloid papaverine. Chamomile also has a significant calming effect and has traditionally had an application as a mild sedative.

Chamomile is a good botanical to have on hand when traveling or camping. For infants experiencing restlessness and discomfort from teething, one third of the adult dosage may provide relief. For treatment of conditions (intestinal gas, colic, peptic ulcers) that may arise from excessive nervous tension, 2 teaspoons (or one standard teabag) of the flower tops can be added to a cup of boiling water and infused for 5 to 10 minutes; 2 to 3 cups may be taken in 30 minutes for acute intestinal colic.

Echinacea (*Echinacea* species)

Description and Habitat. Echinacea (Fig. 60-12) is a perennial herb native to the Midwestern region of North America, from Saskatchewan to Texas. Species include *Echinacea angustifolia* and *Echinacea purpurea*. The plant produces a characteristic

Figure 60-12. Echinacea *(Echinacea purpurea)*.

large, pale purple flower and thick hairy leaves and grows 60 to 90 cm (24 to 36 inches) high. The dried root is typically used for medicinal purposes.

Pharmacology. The compounds currently identified from *Echinacea* species are inulin, glucose, fructose, betaine, echinacin, echinacoside, 3-(*m*-trihydroxyphenyl) propionic acid, and nonspecific resins.[158]

Native American and European Medicinal Uses. This medicinal herb came to the attention of American herbalists in the late 1800s. Echinacea was originally used by the Indian tribes of Nebraska and the Sioux for treatment of snakebite and as an antiseptic and analgesic. Eclectic practitioners used it externally for the same purposes but used it internally to treat "bad blood," or any condition that manifested signs of local or systemic infection, whether bacterial or viral.

Modern Clinical and Wilderness Applications. Echinacea is probably the most common botanical used and known by the public, especially in relation to its immunomodulating effects. Many have empirically found that it can reduce symptoms and derail the onset of URIs and minor influenza episodes. However, an evaluation of *Echinacea angustifolia* in experimental rhinovirus infections concluded that extracts of this plant's root, either alone or in combination, did not have clinically significant effects on infection with the virus or on the clinical illness that results from it.[156]

Echinacea is also a good systemic adjunct to the treatment of any contusion or laceration. The polysaccharide component echinacin can maintain the structure and integrity of the collagen matrix in connective tissue and ground substance and can accelerate wound healing experimentally.[123] Echinacin also has a cortisone-like effect, with intermediate stabilization of inflammation reactions. Inulin, a major component of *Echinacea,* is a powerful activator of the immune system's alternative complement pathway. It may increase host defense mechanisms for the neutralization of viruses, destruction of bacteria, and increased action of white blood cells (lymphocytes, neutrophils, monocytes, eosinophils) within areas of infection. Extracts of the root have been shown to possess interferon-like properties. As an immune stimulant early in infection, and for post-trauma rehabilitation, doses are taken orally three times a day: tincture (1:5), 30 to 60 drops, or solid extract (dry powdered extract, 6:1), 250 to 500 mg.

Calendula *(Calendula officinalis)*

Description and Habitat. *Calendula,* a member of the daisy and dandelion family, is found throughout Asia, North America, and Europe. It is most often known as the pot marigold. The flower is generally used for production of a tincture.

Pharmacology. Calendula's chemical constituents include flavonoids, carotenes, saponin, resin, and volatile oils. The volatile oil content is responsible for a localized increase in blood circulation and diaphoresis. The resin content is responsible for the antimicrobial and anti-inflammatory action of the topical application.

Native American and European Medicinal Uses. Native Americans apparently did not use calendula extensively, and the early European literature mentions only its medicinal role. Calendula, however, is one of the best topical applications for treatment and prevention of infection and skin irritation. Early American surgeons highly regarded its ability to treat and prevent postsurgical infections.

Modern Clinical and Wilderness Applications. A fluid or water extraction, or an oil infusion (prepared as a tincture but using vegetable oil instead of alcohol) of calendula should be used in the initial treatment of lacerations, abrasions, and scalds; immediately after any required debridement and cleaning of a wound; and for generalized inflammation of mucous membranes. It has shown its usefulness in dermatitis and in vaginal, sinus, ophthalmic, and middle and external ear infections. The choice of application mode (ointment, tincture, or fluid extract) depends on the wound. The succus (fluid extract) of the flower should be applied for irrigation of wounds and for ophthalmic uses.

Gentian, Bitter Gentian, Yellow Gentian (*Gentiana* L. species)

Description and Habitat. Over 300 species of gentian (Fig. 60-13) are found throughout the world, but it is especially common in mountainous regions of southern and central Europe, Eurasia, and western North America. All parts are used, but it is primarily the roots and rhizomes that are medicinal. Because of concern that overharvesting may endanger this genus, partial root/rhizome collection or leaf-only preparations are recommended.

Pharmacology. The yellow gentian is native to Europe and has been used as a digestive bitter, as an anti-inflammatory, and as an aid to treating infection. Secoiridoids, γ-pyrones, and triterpenoids are among the most notable constituents that have been studied. The bitter secoiridoid gentiopicroside (2% to 4% of the root) is known to have antibacterial and smooth muscle relaxing effects consistent with gentian's traditional use as a digestive bitter, an antibacterial, and an antiarthralgic.[98,134]

Figure 60-13. Gentian *(Gentiana lutea)*.

Figure 60-14. Desert parsley or fern-leafed lomatium (*Lomatium dissectum,* Nutt.).

Native American and European Medicinal Uses. Gentian's use is ancient. The genus is named after King Gentius, the king of Illyria (180–167 BC), and it was described by Dioscorides in *De Materia Medica*. Although gentian is native to Europe, it is generally considered one of the very best stomach tonics in the world. In Germany, it is a standard medicinal and is approved by the Commission E for digestive disorders (loss of appetite, fullness, flatulence)

Traditionally gentian has been used for chronic dyspepsia, indigestion, and poor digestive function. The root, rhizome, or cut herb is steeped and drunk 15 to 30 minutes before eating, especially in the evening. It stimulates the appetite and disperses the "full" and not hungry sensation. It is very bitter.

Modern Clinical and Wilderness Applications. A tea of the leaf, root, or rhizome is most convenient and readily available in many areas at altitude at or below treeline. It can be very helpful for indigestion and digestive dysfunction brought about by poor food quality, weakness, and overexertion that impairs appetite. This tea can also be very helpful for fever or joint inflammation from infection, fatigue, and overuse. In Chinese herbal medicine, gentian is an essential component of Long dan xie gan wan, an excellent herbal formula for fever, viral infection, headache, iritis, cystitis, urethritis, genital herpes, and liver dysfunction.

Desert Parsley, Fern-Leafed Lomatium (*Lomatium dissectum,* Nutt.)

Description and Habitat. Lomatium, a perennial herb (Fig. 60-14), is native to the western United States and grows predominantly at low to mid elevation in temperate to arid regions. The genus *Lomatium* contains over 80 species, used more or less interchangeably. It is found on wooded or brushy rocky slopes, on alpine meadow steppes, or on dry hillsides. The root is drunk as an infusion, chewed, or pounded for topical application.

Pharmacology. The root extract has been shown to completely inhibit the cytopathic effects of rotaviruses in vitro.[115] The active constituent from *Lomatium suksdorfii,* suksdorfin, is related to coumarin and has been shown to strongly inhibit human immunodeficiency virus (HIV)-1.[103] This finding is consistent with its use as an anti-infective. Flavonoids and ichthyotoxic (fish killing) tetronic acids have also been identified.[163]

Native American and European Medicinal Uses. *Lomatium* was highly revered by Native Americans and was used for a wide variety of problems. It gained legendary status in the U.S. Southwest when Native Americans used it during the influenza pandemic of 1917. The Paiutes of Nevada treated sore throats with a decoction of the root. Many Native American groups considered *Lomatium* important for treatment of tuberculosis, asthma, and other lung diseases. It was also used internally and topically for venereal disease. Naturopathic physicians in the western United States have popularized the use of *Lomatium* as an antiviral when the root extract is taken internally, for vaginitis when used as a douche, or as an oral rinse to treat periodontal disease.[11]

Modern Clinical and Wilderness Applications. Because of unconfirmed toxicity, it is probably best to avoid the tops of *Lomatium,* but the root is very helpful for URIs, viral illnesses, sore throats, mouth infections, and topical local infections. In the clinic, specific root isolates (with resins removed) are used; in naturopathic medicine, these are considered specific for herpes infections. These isolates are also thought to reduce the incidence of a benign hive-like rash that appears in individuals sensitive to *Lomatium*. A pioneer in naturopathic medicine, John Bastyr, said the appearance of the rash is a sign to decrease the dose, not discontinue the medicine.[11]

In the wilderness, *Lomatium* may be a significant aid in treating and preventing a wide variety of microbial and viral infections and is easily prepared from fresh or recently dried root as a decoction.

Devil's Club *(Oplopanax horridum)*

Description and Habitat. Native to North America, *Oplopanax* (Fig. 60-15) is an erect, slightly spreading decidu-

Figure 60-15. Devil's club *(Oplopanax horridum).*

ous shrub present in moist but well-drained forested and river bank ecosystems from coastal Alaska to central Oregon, east to Idaho, Montana, northwestern Alberta, southwestern Yukon, and the Canadian Rockies, with a few populations near northern Lake Superior, the upper peninsula of Michigan, and Ontario. The stems, leaves, and petioles have a dense armor of needlelike yellow spines that can cause injury and irritation. Flowers are small, whitish, and numerous in compact terminal pyramidal clusters. Notably, *Oplopanax* is an Araliacea, which includes *Panax ginseng* (Asian ginseng), *Panax quinquefolius* (American ginseng), and *Aralia nudicauli* (sarsaparilla).

Pharmacology. The best known constituents of devil's club include saponin triterpenoid glycosides, which have been described primarily in Japanese and Russian *Oplopanax*. There has been much discussion as to whether these saponins are like the ginsenosides in *Panax* species, consistent with devil's club traditional use among Native Americans as a tonic. Sterols, sesquiterpenes, and polyenes that have been shown to have significant action against mycobacteria, fungi, and common bacteria such as *S. aureus, Bacillus subtilis, Pseudomonas aeruginosa, E. coli, and C. albicans.*[94] These findings are consistent with the primary historical use of devil's club internally and topically for all varieties of infection, including tuberculosis.

Native American and European Medicinal Uses. It is difficult to overstate the importance of this herb to the native peoples of North America. It is of extreme significance as a spiritual and shamanic herb, including purification and healing, protection against evil spirits, and gaining of supernatural powers, and as an emetic and purgative. Its primary uses as medicinal herb are for infections, fever, arthritis, respiratory ailments, post-

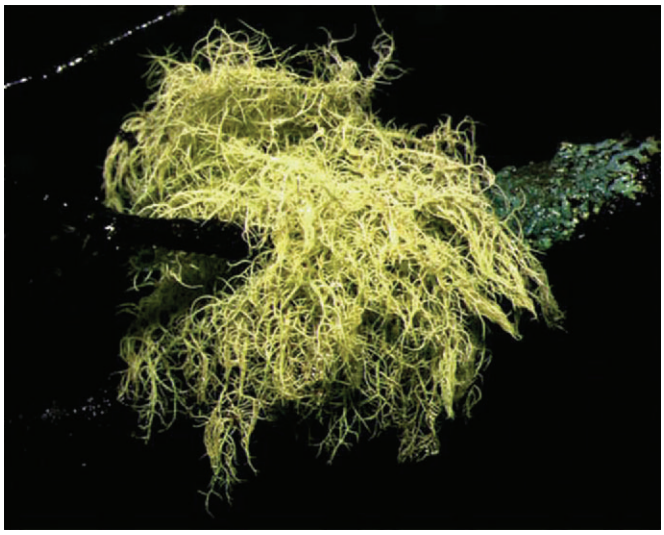

Figure 60-16. Old man's beard (*Usnea* species).

childbirth bleeding, pain, broken bones, digestive ailments, stomach complaints, and even dandruff and lice.

European medicinal use is mostly in formulas for joint pain, arthritis (especially autoimmune arthritis), and rheumatism. The root is considered to be an effective expectorant and respiratory stimulant. Recently, some marketers have played up the adaptogenic potential of *Oplopanax,* calling it "Alaskan ginseng" or "Pacific ginseng." Commercial products also emphasize the root, whereas traditional uses and much of the research have focused on the inner bark of the stem.

Modern Clinical and Wilderness Uses. Modern clinical use is focused primarily on joint pain, osteoarthritis, and inflammatory arthritides in various formulas or as a single herb. Devil's club is often found in combination with devil's claw *(Hapagophytum),* cat's claw (*Uncaria* species), or one of its western companions chaparral *(Larrea tridentata)* and Oregon grape *(Berberis acquifolium)* as an anti-infective combination. Decoction of the inner bark of the stem is the primary method of preparation, but other parts, such as the inner bark (burned, and the ash used topically), whole stems, berries, leaves, and root, have all been utilized. In the wilderness, an infusion or decoction of the stem's inner bark is easily prepared and may help allay fatigue, ease joint or headache pain, or serve as an expectorant. In the practice of traditional medicine, devil's club appears to be good for everything.

Old Man's Beard (*Usnea* species)

Description and Habitat. *Usnea* species (Fig. 60-16) are lichens (composed of fungi and algae in a stable symbiotic relationship) that are ubiquitous in old-growth forests of the Northwest. They can be seen hanging from shrubs or conifers, often lightly tethered to the tree bark, or lying on the forest floor, especially after a storm. It is difficult for any but the expert to distinguish between species of usneas, but they typically have a long, single, unbranched (or sparsely branched) central cord

colored from white to yellow with an outer portion that is gray to pale green.

Native American and European Medicinal Uses. This plant has been used since ancient times in the Americas, Europe, and China. Native Americans saw *Usnea* as representing the male sex and the northerly direction, and maintaining the lung system of the planet (namely the trees); they saw its human uses as secondary to this crucial function.[80] *Usnea* appears in the ancient Chinese herbal, the Shennong Ben Cao Jing (Divine Farmer's Materia Medica, around 200 BC), and it is presently classified as a phlegm-resolving herb. Among northwestern Native Americans, it has variously been used as bedding, as sanitary napkins, and to wipe slime when cleaning salmon, but most significantly it is used as wound dressing and bandage material.[162] This is because of not only its wispy soft form but also its antibacterial properties. In Europe, it has been used predominantly as a topical medication, employing the active constituent usnic acid, present in many lichens, as an antibiotic, anti-inflammatory, and analgesic substance.[75] Since its isolation in 1844, usnic acid has been the most studied of lichen constituents and one of the few to be commercially available. In addition to the bacterial inhibition of staphylococci, streptococci, pneumococci, and mycobacteria, usnic acid has been shown to have antiviral, antiprotozoal, and antiproliferative effects consistent with its traditional use.[79] Also present in *Usnea,* but less well studied than usnic acid, are the organic acids usnaric, thamnolic, lobaric, and sticitinic.

Modern Clinical and Wilderness Uses. Although pure usnic acid has been used for weight loss in recent times, this use cannot be considered safe and has been associated with liver failure.[49] *Usnea* has been used in several formulas in modern-day China, usually paired with the seaweed *Laminaria* or *Sargassum* (or both) for treatment of thyroid cancer,[76] and in the treatment of bronchitis with profuse sputum.[46] Other current uses include lozenges for oral inflammation and many different salves and creams with antimicrobial and anti-inflammatory action.

In the wilderness, *Usnea* is readily available in the regions mentioned earlier, and it makes excellent bandages for wounds, superficial infections, and contact dermatitis. Most herbalists recommend collection from the ground so as not to disturb growth on shrubs or trees. Soft and wispy *Usnea* is applied directly to the affected area and held in place with whatever means are available. Alternatively, dry *Usnea* may be powdered and sprinkled directly on wounds, affording antimicrobial wound protection.

FIRST-AID KIT OF NATURAL PRODUCTS

A natural products first-aid kit should contain a variety of products that are easy to obtain and replace and that have a wide spectrum of use, including herbs (Box 60-1), homeopathic preparations, and vitamin and enzyme supplements (Table 60-1).

Box 60-1. Herbal Medicines Recommended for a First-Aid Kit

Aloe gel and powder capsules
Arnica ointment
Calendula gel, ointment, and tincture
Chamomile tincture
Comfrey gel or ointment
Echinacea tincture or freeze-dried powder capsules
Ephedra freeze-dried powder capsules
Goldenseal tincture or ointment
Hypericum ointment or tincture
Plantain tincture
Witch hazel fluid extract or tincture

Homeopathic Medicines

Homeopathy can be an excellent source of relief and treatment for emergencies and general first-aid situations. Homeopathic preparations include powders, tablets, tinctures, lotions, ointments, creams, and sprays. The advantages of homeopathy are ease of administration, lack of toxicity, rapid action, and small volume of material. The disadvantages are the degree of understanding and competence required to become an effective prescriber, and the lack of readily available sources of each medicine at most North American pharmacies.

A kit made exclusively of homeopathic medicines can cover most first-aid emergency situations. For the acute, straightforward injury or malady without a complex set of symptoms, the correct simillimum and rapid amelioration of symptoms are not difficult to achieve. This section discusses a few indications for use of homeopathic medicines and the preparation most often used. Unless otherwise noted, a 6× to 12× potency in lactose pellet form should be given every 15 to 30 minutes immediately after the injury until noticeable improvement occurs. If no effect is noted after the first two doses, the medicine selection should be reconsidered.

The personal experience of one of these authors (KJD) exemplifies the relief that can be obtained from an acute injury with the appropriate homeopathic medicine:

> *I was bitten on the lip by a small centipede while sleeping. I instantly experienced swelling and intense burning pain. Local application of ice provided no relief. I chose the homeopathic medicine* Apis mellifica *in a 6c potency because the wound was shiny and felt hot, and the swelling was increasing. After sublingual ingestion of two pellets, I waited 1 to 2 minutes, still in excruciating pain with no change in symptoms. My next selection was* Cantharis *in a 6c potency, because a key symptom for this remedy is extreme red and hot burning pain of the face. Less than 30 seconds after administration, the pain was almost undetectable. Total relief was obtained within a few minutes after being bitten. As an unintended control, I have had no homeopathic kit available after other centipede bites, and the pain generally lasted for hours and the residual swelling for days. Reactions from different centipedes can cause different sensations and symptoms, however, so* Cantharis *may not work for all bites.*

Proper selection of the simillimum or indicated homeopathic medicine requires the ability to note the subtle differences in the ways the victim responds to apparently similar traumatic or

TABLE 60-1. Uses for Phytopharmaceuticals

PLANT MEDICINES	ANALGESIC	ANTIBIOTIC	ANTIFUNGAL	ANTI-INFLAMMATORY	ASTRINGENT	ANTISEPTIC	DECONGESTANT	SEDATIVE
Aconitum napellus	—	—	—	—	—	—	—	Homeopathic internal
Apis mellifica	—	—	—	Homeopathic topical, internal	—	—	—	—
Arnica montana	Homeopathic internal Botanical topical	—	—	Homeopathic topical, internal	—	—	—	—
Arsenicum album	—	—	—	—	—	—	—	Homeopathic internal
Bromelain	—	—	—	Botanical internal	—	—	—	—
Calendula	—	—	Botanical internal, topical	Botanical topical	Botanical topical	Botanical topical	—	—
Chamomile	Botanical internal, topical	Botanical internal, topical	—	Botanical internal, topical	—	Botanical topical	Botanical internal	Botanical homeopathic internal
Comfrey	—	—	—	Botanical topical	—	—	—	—
Echinacea	Botanical internal, topical	Botanical internal, topical	Botanical internal, topical	Botanical internal	—	Botanic homeopathic topical	—	—
Ephedra	—	—	—	—	—	—	Botanical internal	—
Goldenseal	—	Botanical internal, topical	Botanical internal, topical	Botanical topical	Botanical internal, topical	Botanical topical	—	—
Hypericum	—	—	—	—	—	Homeopathic topical, internal	—	Homeopathic internal
Peppermint	—	—	—	—	—	—	Botanical internal, topical	—
Plantain	—	—	—	—	Botanical topical	Botanical topical	—	—
Rhus toxicodendron	—	—	—	Homeopathic topical, internal	Botanical topical	—	—	—
Witch hazel	—	—	—	—	Botanical topical	—	—	—

toxicologic influences. An appropriate homeopathic field guide that lists specific indications and differentiations for each homeopathic remedy should accompany any first-aid kit. It is essential to understand the specific homeopathic indications for each of the remedies (simillima) on hand. Otherwise, the chance of obtaining a successful outcome is small.

Practitioners and the homeopathic industry have realized the difficulty of single-remedy prescribing, which involves understanding and memorizing indications for every homeopathic medicine. Therefore, medicines have been developed that combine remedies to cover a large number of the symptoms and symptom characteristics that typically accompany most ailments. These medicines, known as complex or combination homeopathic preparations, can be very helpful for the new user.

Single Preparations, and Their Indications

Aconite. Tincture of the whole plant with its root is derived when monkshood or wolfsbane *(Aconitum napellus)* begins to flower. Aconite is indicated for acute states of emotional disturbance, including anxiety and intense fear or pain. This is one of the key remedies that should be administered after an acute injury that has dazed, shocked, or frightened the victim. Persons who are fearful or restless, cannot tolerate being touched, and have pain followed by numbness and tingling sensations are most responsive to aconite. Those with sudden onset of fever, nausea, and vomiting and who exhibit symptoms of fear, restlessness, and anxiety may also benefit.

Apis. The original tincture is manufactured from the whole honeybee and from dilutions of its venom *(Apis mellifica)*. Apis is used for insect stings, particularly from bees and related insects, when the wound is swollen, shiny, and hot to the touch. Treatable symptoms from other conditions are histamine reactions (resulting in facial flushing; puffiness or swelling around the mouth, face, and eyes), sunburn, hives, burns, and early stages of abscesses and frostbite. If symptoms include a stinging, burning, or swelling quality and subside by applying cold rather than heat, Apis is the indicated remedy.[133]

Arnica. Tincture comes from the whole fresh plant, flowers, and dried roots of leopard's bane or Fallkraut *(Arnica montana)*. Arnica is indicated for blunt traumatic wounds (resulting in both deep and superficial hematomas), contusions, swelling, and localized tenderness. It is also effective for sore muscles, as well as sprains, fractures, dislocations, and internal bleeding. The authors recommend taking a 6× to 30× potency every 15 minutes to 3 hours for the first few days after a severe injury. The more severe the injury, the more frequently the dose is taken for the first day. As symptom severity decreases, the medicine is taken less often. Arnica can be helpful in decreasing severity of symptoms and recovery time.

Arsenicum. Derived from arsenic trioxide, arsenicum is used for skin rashes (those that feel warm but are relieved by hot applications), hay fever, asthma (especially when accompanied by notable anxiety), diarrhea, vomiting, and gastroenteritis (especially from food-borne microbes).

Hypericum. The tincture comes from the whole fresh plant and flowers of St.-John's wort *(Hypericum perforatum)*. Indications include any pain that affects the peripheral or central nervous system and exhibits shooting pains that travel in a dermatomal pattern (e.g., sciatica). Wounds that affect nerve endings, such as injuries to fingers, toes, or teeth, are improved by *Hypericum*. Pain from dental surgery, toothache, injury to the coccyx, or first- and second-degree dermal burns is another indication.

Ledum. Ledum is made from leaves and stems of the whole fresh plant of wild rosemary *(Ledum palustre)*. Homeopathic indications include puncture wounds from small, sharp objects (e.g., nails, needles) and some mosquito bites when the injured area feels cold, swollen, and numb and pain is relieved by cold application.

Rhus. The homeopathic preparation of *Rhus* comes from leaves and stems of the whole fresh plant of poison ivy. This is the remedy of choice for the urticaria caused by poison ivy exposure, and it is also helpful for some cases of poison oak. Other skin rashes that are red, weeping, blistered, and swollen with itching can be treated with *Rhus*. It is also effective for treatment of connective tissue irritations with swelling, stiffness, and tightness. *Rhus* is often used for overuse injuries (e.g., fasciitis, tendinitis) and some forms of arthritis, especially when the injured area feels better with warm applications and movement. Note that the name of this preparation may change, as *Toxicodendron* is now the correct name of the genus.

Combination Preparations for Acute Sprains and Strains

Homeopathic companies have created combination remedies for the general public that can be used without the need for in-depth understanding of homeopathic prescribing. These remedies are designed to cover a broad range of symptoms associated with acute ailments and trauma-induced medical conditions. They are sometimes touted to be effective for many disorders on the basis of empirical observations, rather than on the basis of randomized, blinded studies. Practitioners are advised to be aware of the possibility for unsubstantiated claims of clinical efficacy for these and any other medications marketed for profit.

Traumeel (Heel Biotherapeutics, Albuquerque, NM) is a combination homeopathic formula that is effective for treatment of trauma and inflammatory changes affecting skin, connective tissue, and muscle.[60] The preparation comes in liquid, tablet, and ointment form. Traumeel includes remedies indicated for traumatic injuries (sprains, strains, contusions) and resulting pain, swelling, and ecchymoses. Many German studies have demonstrated its effectiveness.[29] Traumeel may be the primary homeopathic medicine chosen for the first-aid kit because of its wide range of applications and multiple delivery systems.

Herbal Combination Formulas

In the tradition of Chinese herbal medicine, many formulas have been developed over the centuries to treat acute ailments. Many of these formulas were kept secret and reserved for the nobility and ruling class. As the field of Chinese herbology has developed and become more accessible to the general populace, some of the secret formulas have been mass produced into convenient pill form, known as patent medicines. Many are extremely useful for acute conditions.

Zheng Gu Shui ("Rectify Bones Liquid") trauma lotion is wonderful for sprains, strains, and bruises. It is also indicated for back pain and arthritis pain. The herbs of which it is com-

posed invigorate circulation, relieve pain, induce a nice warming feeling, and accelerate healing. This formula has long been a mainstay of martial artists, massage therapists, and Chinese herbalists. It should be applied to affected areas two to three times a day, keeping the liquid away from mucous membranes and open cuts. It accelerates resolution of bruises and connective tissue injuries, reducing swelling, pain, and inflammation. It is produced by Guangxi Yulin Pharmaceutical Factory, which is well known in China and has been honored with awards for quality and effectiveness. This product can be ordered through the Institute for Traditional Medicine (ITM; listed in the resources section of the Appendix) and from most Chinese herbal pharmacies.

Nutritional Supplements

For immune system support, anti-inflammatory action, and pain relief, many natural products in the nutritional supplement category have proved to be effective agents.

Bromelain. Bromelain is a naturally occurring proteolytic enzyme found in pineapple that is used to reduce pain and swelling after sprains and strains of soft tissues. Ingested on an empty stomach, the complex proteases in bromelain are absorbed intact and have significant antiedema, anti-inflammatory, and coagulation-inhibiting effects. Bromelain exhibits fibrinolytic activity and acts to inhibit fibrinogen synthesis, decreasing kininogen and bradykinins.[108] For treatment of injuries and postsurgical recovery, 125 to 400 mg is ingested three times daily at 30 minutes before or 90 minutes after a meal. Bromelain is nontoxic even at high dosages and is generally prepared as 100-mg tablets.

Papain. Like bromelain, papain is a naturally occurring plant enzyme (from papaya fruit) that exhibits proteolytic activity. Papain is generally used externally to neutralize bee, ant, or wasp venom. It is available as commercial meat tenderizer (e.g., Adolph's) or in tablet form. After removal of the stinger, a thick paste is prepared from water and tenderizer (or five or six crushed tablets) and applied to the area as soon as possible.

A convenient form that contains bromelain, papain, and pancreas-derived proteolytic enzymes is the product Wobenzym. This product has had decades of use in addition to clinical trials to back its claims as an effective proteolytic anti-inflammatory. Suggested use is three tablets twice a day or as directed by the practitioner. Wobenzym is taken on an empty stomach at least 45 minutes before meals.

Vitamin C. Ascorbic acid has both wound-healing and anti-inflammatory effects. Vitamin C is required for hydroxylation of proline and subsequently for synthesis of effective collagen. Studies have shown that the stress associated with injury and wound healing results in an increased need for vitamin C.[114] For acute trauma and acute upper respiratory allergy, vitamin C in larger dosages (2 to 5 g/day in divided doses) has been claimed to reduce anaphylactic reactions and recovery time.[70] Therefore, for any traumatic event, high-dose vitamin C should be administered as part of the treatment.

For Acute Gastroenteritis

Pill Curing (Kang Ning Wan, "Healthy Quiet Pill"), botanically called Coix Formula, consists of 16 herbal medicines that are collectively effective for relieving disturbances caused by motion sickness, food poisoning, overeating, excessive alcohol consumption (nausea, headache, vomiting), difficulty passing stool, loose stools, and GI cramping and pain. Coix Formula is currently produced in a convenient globule form (Metagenics, San Clemente, Calif). One or two capfuls of globules are swallowed with warm water every hour until symptoms improve. Relief should occur within 4 hours of administration. Pill Curing (or Culing) is also available as a Chinese patent (or prepared) medicine from ITM or from a Chinese herbal pharmacy. The package includes 10 vials; the usual dose is 1 to 2 vials, each containing multiple small pillules to be swallowed all at once. Relief is generally within hours. This is an all-purpose remedy claimed to be useful for everything from traveler's diarrhea, to hangover, to the common cold.

For Acute Hemorrhagic Conditions

The product Yunnan Bai Yao ("Yunnan White Medicine"), produced in the western Chinese province of Yunnan, has been used for centuries as a first-line approach to trauma that results in internal or external bleeding. It is prescribed in China for excessive menstrual cramps and bleeding, bleeding ulcers, trauma-induced swelling, bleeding wounds, and allergic reactions to insect bites. It comes in powder (4 g per bottle) and capsule (packets of 20) form and contains one red pellet that is to be ingested only for serious bleeding conditions. Dosage is 1 to 2 capsules four times per day. The powder can be applied externally after the wound has been properly cleaned. This product is exclusively produced in China from a proprietary formula and can be obtained from most Chinese herbal pharmacies.

For Dermatologic Conditions

Ssssting Stop Gel. The Boericke and Tafel Ssssting Stop gel is for temporary relief of itch, pain, and redness of nonpoisonous insect bites and stings from insects—mosquitoes, bees, wasps. It also soothes fever blisters and cold sores. The London School of Hygiene and Tropical Medicine conducted two double-blind clinical studies using mosquitoes that had not been fed for 24 hours and human volunteers. The tests were reported to prove that the medicines in Ssssting Stop provide dependable, effective relief.

Ssssting Stop combines three natural homeopathic medicines prepared from botanical sources and listed in the Homeopathic Pharmacopoeia of the United States (HPUS). It contains no hydrocortisone or other steroids, antihistamines, "-caine" anesthetics, or synthetic medicines of any kind. It is applied to the affected skin area, with applications repeated as needed.

Ingredients are *Echinacea angustifolia* (1×, 10%), *Ledum palustre* (1×, 10%), *Urtica dioica* (1×, 10%), and citronella oil and eucalyptus oil in a water gel base.

Ching Wang Hun (or Jing Wan Hong) Burn Ointment. This fast-acting analgesic and burn healing salve is a remarkable herbal medicine. It is effective for chemical, thermal, electrical, radiation, and solar burns. It can also be used to decrease inflammation and stimulate regeneration of the skin, and for contact dermatitis (poison oak, ivy, sumac), hemorrhoids, and infected skin.

The ointment is applied liberally to the affected areas. Ideally it is covered to prevent accumulation of dirt and grime and to reduce the red stain that occurs if the ointment gets on clothing. It should be reapplied and the dressing changed once or twice a day. This ointment is made by one of the best-known manufacturers in China, and it can be ordered through ITM (see the resource section in the Appendix) and from most Chinese herbal pharmacies.

APPENDIX

Companies

Nature's Apothecary
997 Dixon Rd
Boulder, CO 80302
800-999-7422
Small herbal kits include Dental Poultice Pac for toothaches and abscesses, Clear Eyes Eyewash Kit (including Eyebright *Euphrasia* extract), and reformulated Home Herbal Medicine Kit.

Boiron
1208 Amosland Rd
PO Box 54
Norwood, PA 19074
800-258-8823
Natural Home Health Care LeKit contains 36 single-remedy medicines in distinctive blue tubes, including the commercial flu remedy Oscillococcinum. The home kit also contains four external remedies: tinctures of *Calendula* and *Hypericum*, and ointments of *Arnica* and *Calendula*. Travel LeKit is a more compact collection of single remedies (22 multidose and 16 one-dose tubes) plus the flu remedy.

Dolisos America
3014 Rigel Ave
Las Vegas, NV 89102
702-871-7153, 800-365-4767
Single-Remedy Family Kit contains 48 single remedies, a Flu-Solution remedy, *Calendula* ointment and tincture, and *Arnica* cream. The 48-combination Energy Medicine Kit includes remedies for bruises, insect bites, and poison ivy.

Biological Homeopathic Industries (BHI)
11600 Cochiti S.E.
Albuquerque, NM 87123
800-621-7644
BHI is the U.S. distributor of the German line of complex homeopathic remedies manufactured by Heel.

Institute for Traditional Medicine (ITM)
2017 SE Hawthorne Blvd
Portland, OR 97214
503-233-4907
www.itmonline.org
A nonprofit resource for Chinese herbal medicines, patent formulas (prepared medicines), and professional herbal formulas for practitioners. Jintu is the nonprescription resource for the products listed here: www.itmonline.org/jintu.

www.pureprescriptions.com is a website to access Wobenzym-N and other products that are generally sold only to health care practitioners.

Books

Natural Health and Medicine

Lust J, Tierra M: The Natural Remedy Bible. New York, Pocket Books, 1990.
Murray M, Pizzorno J: Encyclopedia of Natural Medicine. Rocklin, CA, Prima, 1991.
Weil A: Health and Healing: Understanding Conventional and Alternative Medicine. Boston, Houghton Mifflin, 1983.

Herbs and Herbalism

Murray M: The Healing Power of Herbs. Rocklin, CA, Prima, 1991.
Weiss RF: Herbal Medicine. Beaconsfield, England, Beaconsfield, 1988.

Homeopathy Books

Boericke W, Boericke O: Boericke's Materia Medica with Repertory. New Delhi, B Jain, 1991.
Kruzel T: The Homeopathic Emergency Guide. Berkeley, CA, North Atlantic Books/Homeopathic Education Services, 1992.
Reckeweg HH: Homotoxicology: Illness and Healing through Anti-Homotoxic Therapy, 2nd ed. Albuquerque, NM, Menaco, 1984.
Subotnick S: Sports and Exercise Injuries: Conventional, Homeopathic, and Alternative Treatments. Berkeley, CA, North Atlantic Books, 1991.

Practitioners

American Association of Naturopathic Physicians
601 Valley St, Suite 105
Seattle, WA 98109
206-323-7610
www.naturopathic.org

Herbal Medicines

Herb Research Foundation
1007 Pearl St, #200F
Boulder, CO 80302
303-449-2265

Homeopathy

International Foundation for Homeopathy
2366 Eastlake Ave E, #329
Seattle, WA 98102
206-324-8230

Nutritional Products

Crane Herb Company
745 Falmouth Road
Mashpee, MA 02649
800-227-4118
www.craneherb.com (registration required)

Thorne Research
25820 Highway 2 West
PO Box 25
Dover, ID 83825
800-228-1966

Metagenics
100 Avenida La Pata
San Clemente, CA 92673
800-692-9400

Mountain Peak Nutritionals
3310 SW Vista Drive
Portland, OR 97225
877-686-7325
www.mpn8.com

PhytoPharmica
825 Challenger Dr
PO Box 1745
Green Bay, WI 54305
800-553-2370

Vital Nutrients
45 Kenneth Dooley Dr
Middletown, CT 06457
888-328-9992
www.vitalnutrients.net

The references for this chapter can be found on the accompanying DVD-ROM.

61 Field Water Disinfection

Howard D. Backer*

The text in this chapter provides supporting information and data for recommendations. The boxes and tables summarize most important data and products (Box 61-1).

Water-borne disease is a risk for international travelers who visit countries that have poor hygiene and inadequate sanitation and for wilderness users relying on surface water in any country, including the United States. Natural water may be contaminated with organic or inorganic material from land erosion, dissolution of minerals, decay of organic vegetation, biologic organisms that reside in soil and water, industrial chemical pollutants, and microorganisms from animal or human biologic wastes.[47,77] The main reason for treating drinking water is to prevent gastrointestinal illness from fecal pollution with enteric pathogens.[221] Appearance, odor, and taste are not reliable to estimate water safety. Natural organic and inorganic material may not cause illness but can impart unpleasant turbidity, color, and taste to the water.

Of the 1700 million square miles of water on Earth, less than 0.5% is potable.[244] Global warming is accelerating deterioration of the remaining potable supplies,[122] and emerging water-borne pathogens create the need to reevaluate detection and disinfection methods.[221] Chemical contamination of groundwater is increasing at an alarming rate in the United States and worldwide from industrial, agricultural, and individual sources. Virtually none of the surface water in the United States is drinkable without treatment.[122] According to the National Water Quality Inventory Report by the U.S. Environmental Protection Agency (EPA), as of 2000, about 40% of the nation's surveyed rivers, lakes, and estuaries were too polluted for basic uses such as fishing and swimming.[66] Disasters, such as floods and hurricanes, often overwhelm treatment facilities and contaminate groundwater, requiring point-of-use disinfection.[202]

BENEFITS OF WATER TREATMENT

Methods for treating water are found in Sanskrit medical lore, and pictures of apparatus to purify water appear on Egyptian walls from the 15th century BC. Boiling and filtration through porous vessels, sand, and gravel have been known for thousands of years. The Greeks and Romans also understood the importance of pure water.[141] Sanitation, including water treatment, is considered one of the 10 great public health achievements that helped conquer infectious disease as a main cause of mortality in the United States.[36] As the percentage of the U.S. urban population served by water treatment utilities increased after 1900, the annual death rate from typhoid decreased.[201]

Drinking water treatment processes provide enormous benefits with minimal risk. Without disinfection and filtration, water-borne disease would spread rapidly in most public water systems served by surface water.[51,230] The combined roles of safe water, hygiene, and adequate sanitation in reducing diarrhea and other diseases are clear and well documented. Recent studies of simple water interventions in households of developing countries clearly document improved microbiologic quality of water as well as reduced incidence of diarrhea illness independent of other measures to improve sanitation.[7,100,127,139,172,173,180,212] There is little information demonstrating the benefits of water disinfection in the U.S. wilderness. Boulware and colleagues demonstrated that drinking untreated water correlated with higher rates of diarrhea among Appalachian trail hikers.[22]

RISK AND ETIOLOGY

Infectious agents in contaminated drinking water with the potential for water-borne transmission include bacteria, viruses, protozoa, and parasites (Box 61-2).[72,221] The long list of pathogenic microorganisms capable of water-borne transmission is similar to that of potential etiologic agents of travelers' diarrhea; almost all enteric pathogens and opportunistic pathogens that are transmissible by the fecal-oral route can be transmitted through water. Separating the contribution of water-borne transmission of these pathogens from food-borne and person-to-person transmission is impossible; the latter two are probably more common. However, in developing countries, 15% to 20% of diarrhea is estimated to be water-borne. In developed countries, as much as 15% to 30% is attributed to municipal drinking water. It had been assumed that improved sanitation was essential for reducing diarrheal diseases. Even though the combination of improved water quality and sanitation have the greatest effect, improvement in water quality alone also has a beneficial effect on health.[212]

Risk of water-borne illness depends on the number of organisms consumed, which is determined by the volume of water, concentration of organisms, and treatment system efficiency.[49,99]

*The views and opinions expressed in this chapter are those of the author and do not necessarily represent those of the California Department of Health Services.

Box 61-1. Chapter Outline and Shortcuts

- For summary of each technique, see boxes in specific sections.
- For comparison of various techniques, see Tables 61-16 to 61-18.
- For details of products, see the Appendices.

Methods of Water Disinfection That Can Be Applied in the Field	Page
Heat	1376
Physical removal	1378
Sedimentation	1378
Coagulation-flocculation	1379
Granular activated charcoal	1379
Filtration	1380
Reverse osmosis	1382
Halogens	1382
Chlorine	1387
Iodine	1389
Iodine resins	1391
Other methods	
Chlorine dioxide	1394
Silver	1394
Potassium permanganate	1395
Hydrogen peroxide	1395
Ultraviolet light	1395
Copper-zinc	1396
Citrus	1396

Additional factors include virulence of the organism and defenses of the host. Infection and illness are not synonymous; the overall likelihood of illness from multiple studies for all three categories of microorganisms (bacterial, viral, protozoan) is 50% to 60%. Death is unlikely except with specific organisms, such as *Escherichia coli* O157:H7, *Vibrio cholerae*, hepatitis E in pregnant women, and *Cryptosporidium* with underlying malnutrition. Total immunity does not develop for most enteric pathogens and reinfection may occur.[99]

Water-borne outbreaks do not give a complete picture of the potential for water-borne illness. Most outbreaks of water-borne disease are not identified because not enough people become ill, providing an insensitive mechanism for detecting water contamination. When an outbreak is identified, it is very difficult to prove conclusively that the source was water-borne. The supply may have been only transiently contaminated; water samples from the time of exposure are seldom available; some organisms are difficult to detect; and almost everyone has some exposure to water.[225]

The data on concentration of microorganisms in surface water show widely varying values, but the testing is insufficient for risk assessment and dose-response models. Instead, infectious dose data and statistical techniques have been used to devise models for determining risk (Table 61-1).[99] These cannot be applied unless the microbial content of water is known. The excretion and loading of microbial contaminants are dynamic and change over time. Pathogenic microorganisms clearly exist in most raw source waters, especially in surface waters.[230] Most microbiologic testing is done on community water intake sources and sewage treatment effluent. Less information is available for more remote water sources.[50,191,249] The source of fecal contamination in water may be either human or animal. Some bacterial pathogens *(Shigella, Salmonella typhosa)* occur exclu-

Box 61-2. Water-borne Enteric Pathogens

BACTERIAL
Escherichia coli
Shigella
Campylobacter
Vibrio cholerae
Salmonella
Yersinia enterocolitica
Aeromonas

VIRAL
Hepatitis A
Hepatitis E
Norovirus
Poliovirus
Miscellaneous enterics (more than 100 types: e.g., adenovirus, enterovirus, calcivirus, ECHO viruses, astrovirus, coronavirus)

PROTOZOAL
Giardia lamblia
Entamoeba histolytica
Cryptosporidium
Blastocystis hominis
Isospora belli
Balantidium coli
Acanthamoeba
Cyclospora

PARASITIC
Ascaris lumbricoides
Ancylostoma duodenale (hookworm)
Taenia spp. (tapeworm)
Fasciola hepatica (sheep liver fluke)
Dracunculus medinensis
Strongyloides stercoralis
Trichuris trichiura (whipworm)
Clonorchis sinensis (Oriental liver fluke)
Paragonimus westermani (lung fluke)
Diphyllobothrium latum (fish tapeworm)
Echinococcus granulosus (hydatid disease)

ECHO, enteropathic cytopathogenic human orphan.
Data from references 72, 77, 201, and 230.

sively in human feces, whereas others (*Yersinia, Campylobacter,* nontyphoidal *Salmonella*) may be present in wild or domestic animals. The enteric viruses seem to occur exclusively in human feces. No enteric viruses excreted by animals have been shown to be pathogenic to humans.[177] Some organisms, for example, *Legionella pneumophila* and *V. cholerae,* exist as natural organisms in water. (However, the mode of transmission of *Legionella* is inhalation of aerosolized water.)[215]

New detection techniques using enzyme immunoassay and polymerase chain reaction may give a much more accurate picture of the specific microbes and the degree of water contamination.[221]

Recreational Contact

Inadvertent ingestion during recreational water contact is a risk for swimmers and white-water boaters. The microorganisms that cause infection require only a small dose. Recreational water activities have resulted in giardiasis, cryptosporidiosis,[33,38] typhoid fever, salmonellosis, shigellosis, *E. coli* O157 infection,[68] viral gastroenteritis, and hepatitis A, as well as in wound infections, septicemia, and aspiration pneumonia due to *Legionella*.[51] During 2001–2002, a total of 65 outbreaks associated with recreational water were reported in the United States; these involved 2536 persons and resulted in 61 hospitalizations and 8 deaths. Of the 30 outbreaks of gastroenteritis for which a pathogen could be identified, 12 were caused by protozoa (9 were in chlorinated water), of which 11 were caused by *Cryptosporidium* and 1 by *Giardia.* Six outbreaks were attributed to bacterial pathogens (three linked to freshwater sources): four from *E. coli* O157:H7 and two from *Shigella sonnei.* Five outbreaks of gastroenteritis were attributed to norovirus. Eight cases of primary amebic meningoencephalitis *(Naegleria fowleri)* were reported, with 100% fatality. The number of recreational water-borne outbreaks in fresh and treated water steadily increased from 1989 to 2000.[39]

TABLE 61-1. Estimated Infectious Dose of Enteric Organisms

ORGANISM	INFECTIOUS DOSE
Salmonella	10^5
Shigella	10^2
Vibrio	10^3
Enteric viruses	1–10
Giardia	10–100
Cryptosporidium	10–100

Data from Hurst C, Clark R, Regli S: Estimating the risk of acquiring infectious disease from ingestion of water. In Hurst C (ed): Modeling Disease Transmission and Its Prevention by Disinfection. Melbourne, Cambridge University Press, 1996.

Developing Countries

In tropical areas and developing countries, water has a complex relationship with spread of disease. Table 61-2 presents a useful classification, and Steiner and colleagues[216] proposed adding the category "water carried" for infections resulting from accidental ingestion in recreational water. The World Health Organization (WHO) estimates that worldwide, 25% of the population (1.5 billion rural and 200 million urban people) lack safe drinking water and 50% lack adequate sanitation. In Africa and Asia, nearly 20% of rural water supplies are not functioning at any one time. Only 35% of wastewater is treated in Asia and 14% in Latin America.[251]

An estimated 80% of the world's diseases are linked to inadequate water supply and sanitation. Of the four diseases that may cause the most morbidity and death in developing parts of the world—cholera, hepatitis, malaria, and typhoid—malaria is the only one that is not water-borne.[99] Between 10 and 25 million people die each year (28,000 to 68,000 persons each day) from diseases caused by contaminated water and unsanitary conditions. In developing countries, these illnesses account for 1 billion cases of diarrhea every year and 95% of deaths in children younger than 5 years of age.[72,89,242] Another estimate puts the disease burden from water-related diseases, sanitation, and hygiene at 4% of all deaths and 5.7% of the total disease burden occurring worldwide.[171]

The sanitary situation in many undeveloped countries is illustrated by statistics from Peru evaluated during a cholera epidemic in the 1990s. Only 73% of the urban populations have access to a water distribution system and only 50% to sanitation services. In rural areas, only 23% have access to a water supply and 6% to a sanitation system. In urban areas over the past 5 years, the quality of water has deteriorated because of the lack of water treatment chemicals, laboratories for monitoring, and operators to control the processes. Institutional barriers impede improvements for adequate water and sanitation systems.[52]

TABLE 61-2. Water and Spread of Disease

TYPE	MECHANISM	EXAMPLES	PREVENTION
Waterborne	Fecal contamination of drinking water by infectious organisms	Typhoid fever, cholera, campylobacteriosis, giardiasis, hepatitis A	Sanitation and disinfection of water
Water-washed	Person-to-person fecal–oral spread via direct contact, food, water (all these are also waterborne)	Shigellosis, amebiasis, ascariasis, eye and skin infections	Hand washing and personal hygiene
Water-based	Organism or agent that lives in water	Schistosomiasis, dracunculosis, parasitic worms	Prevention of exposure from bathing
Water-related	Spread by insects that breed in fresh water or collecting water	Malaria, sleep sickness, yellow fever, dengue	Insect protection and piped water

Information from Bradley D: Health aspects of water supplies in tropical countries. In Feachem R, et al (eds): Water, Wastes and Health in Hot Climates. New York, Wiley, 1977, p 3, and Steiner T, Thielman N, Guerrant R: Protozoal agents: What are the dangers for the public water supply? Annu Rev Med 48:329, 1997.

Box 61-3. Enteric Pathogens in U.S. Wilderness or Recreational Water

COMMONLY REPORTED
Giardia
Cryptosporidium

OCCASIONALLY REPORTED WITH FIRM EVIDENCE FOR WATER-BORNE
Campylobacter
Hepatitis A
Hepatitis E
Enterotoxigenic *Escherichia coli*
E. coli 0157:H7
Shigella
Enteric viruses

UNUSUAL OCCURRENCES, WATER-BORNE SUSPECTED
Yersinia enterocolitica
Aeromonas hydrophila
Cyanobacterium (blue-green algae)

In certain tropical countries, the influence of high-density population, rampant pollution, and absence of sanitation systems means that available raw water is virtually wastewater.[43] Contamination of tap water must be assumed because of antiquated and inadequately monitored disposal, disinfection, and distribution systems. Water from springs and wells and even commercial bottled water may be contaminated with pathogenic microorganisms.

United States

Water-borne pathogens account for most outbreaks of infectious diarrhea acquired in U.S. wilderness and recreation areas (Box 61-3). From 1920 to 1980, 178 water-borne outbreaks caused by use of contaminated, untreated surface water or groundwater were reported in systems serving parks, campgrounds, and recreation areas.[51] Between 1970 and 1990, gastroenteritis of undefined etiology accounted for most cases overall, while *Giardia* caused the most cases of defined etiology.[51,90]A distinct seasonal variation is seen, with most cases from recreational areas occurring during summer months.[51] This is probably a result of both increased contamination and number of persons at risk. Even treated community water supplies extensively treated by modern methods are responsible for outbreaks of gastrointestinal illness. Developed countries like the United States still face substantial challenges to assure potable water in households and to maintain quality of water sources.[72,122,201,221] Between 1993 and 1996, *Giardia* continued to be one of the most common water-borne infections, but *Cryptosporidium* epidemics were being identified with increasing frequency in both public and surface water supplies.[88,190] Enteric bacteria are still associated with 12% of water-borne outbreaks in the United States.[192] From 1993 to 1996, bacteria linked to water ingestion included *Salmonella, Campylobacter, Shigella sonnei, Plesiomonas shigelloides,* and another emerging pathogen, *Escherichia coli* O157:H7.[34,35] The general pattern and etiology of water-borne outbreaks in the United States was little changed in 1999 to 2000.[39]

Viruses

The infectious dose of enteric viruses is only a few infectious units in the most susceptible people.[132,241,249] The risk of infections from enteric viruses is estimated to be 10 to 10,000 times higher than bacteria at the same level of exposure.[221]

Hepatitis A virus (HAV), norovirus, and rotavirus are the main viruses of concern for potable water supplies. Many other viruses are capable and suspected of water-borne transmission, and more than 100 different virus types are known to be excreted in human feces.[83,191] The most frequent water-borne illness (acute infectious nonbacterial gastroenteritis of unknown etiology) in the United States may be caused by undetected viruses; estimates of viral water-borne illness are 6.5 million cases annually.[51,72,132,208] In addition to HAV, water-borne transmission of hepatitis E is suspected in outbreaks among travelers from Asia.[30,110] During 1993 and 1994, an explosive water-borne epidemic of hepatitis E virus occurred in Islamabad, Pakistan, with about 4000 cases of acute icteric hepatitis.[204]

Testing in the United States, Europe, and developing countries shows consistent, sometimes astounding, degrees of viral contamination of drinking and surface water. All surface water supplies in the United States and Canada contain naturally occurring human enteroviruses.[244] Even remote surface lakes and streams tested in California showed disturbing levels of viral contamination.[83] Widespread enteric viral contamination was found at multiple sites in a popular recreation canyon in Arizona. Viruses included poliovirus, echovirus, coxsackievirus, rotavirus, and other unidentifiable viruses, exceeding the recommended state level for recreational water use in several areas. Virus levels correlated with human activity but not with excess levels of standard coliform indicators.[191] In 2002, a series of outbreaks was reported on 17 different Colorado River raft trips involving more than 130 individuals. Laboratory evaluation of effluent from portable toilets found norovirus, which was also isolated from river water at Lee's Ferry. No specific risk factors were identified in individuals or trips, and it was concluded that risk was likely from the river water.[93]

No evidence exists of human immunodeficiency virus (HIV) transmitted via a water-borne route, and no epidemiologic evidence exists of causal transmission by fomites or by any environmentally mediated mode.[185]

Protozoa

Six protozoa cause enteric disease and may be passed via water-borne transmission: *Giardia lamblia, Cryptosporidium parvum, Entamoeba histolytica, Cyclospora cayetanesis, Isospora belli,* and the microsporidia.[72,131] The first two are the most important for wilderness travelers. *Cryptosporidium* is an emerging enteric pathogen that has overtaken *Giardia* as the most common water-borne protozoa.[31,72] Many aspects of the epidemiology and transmission appear similar to those of *Giardia;* for example, only 10 *G. lamblia* cysts may result in infection and the infectious dose of *Cryptosporidium* may be equally small.[181] Water-borne transmission of *E. histolytica* is common in developing countries. *Cyclospora* has been epidemiologically linked to water-borne transmission in the United States and in Nepal, but the reservoir and host range are not known. Unlike *Giardia* and *Cryptosporidium, Cyclospora* is not infectious when passed in feces and requires up to 2 weeks in the laboratory to sporulate.[206] Surface water is a common environmental source for microsporidia; however, the route of infection is

TABLE 61-3. *Giardia* Cysts and *Cryptosporidium* Oocysts in North American Surface Water

SOURCE	POSITIVE SAMPLES	NUMBER OF CYSTS	REFERENCE
Giardia			
Surface water from 301 municipalities in the U.S.	798 of 4423		Rice, 1982[159]; Rose, 1991[6]
Pristine surface waters		0.4–5 cysts/100 L	
Unprotected watersheds		0.33–104 cysts/100 L	
Rocky Mountain National Park, California Sierra Nevada	44% in high use areas	Average 3–10 cysts/1000 L, up to 100–600 cysts/1000 L (not adjusted for 10–30% recovery rate)	LeChevallier, 1981[117]; Merson, 1974[125]; Singh, 1990[180]; Sobsey, 1991[182]
3 pristine rivers and 12 tributaries in Pacific Northwest	94/224 (43%) samples over 9 mo	0.1–5.2 cysts/L (adjusted for 22% recovery rate)	Neumann, 1969[135]
3 high quality surface creeks in California and Washington	1.3–6 cysts/gallon		Neefe, 1947[132]
Yukon Canada pristine surface water	7/22		Regunathan, 1987[155]
Cryptosporidium			
Raw surface water entering U.S. treatment plants	87%		Steiner, 1997[8]
Surface water samples from 17 states	55% of 257 samples positive (39% from pristine sources positive)	0.43 oocysts/L	Rose, 1991[6]
Western U.S.	77% rivers 75% lakes 28% treated drinking water samples	Average 0.02–1.3 oocysts/L Pristine areas: 0.02–0.08/L Streams and rivers: 0.58–0.91/L	Neumann, 1981[134]
Yukon water	None found in pristine water but some found in water that received sewage effluent		Regunathan, 1987[155]

unknown. *Naegleria fowleri* is a water-borne protozoan that enters the body through the nasal epithelium during swimming in contaminated surface water and causes meningoencephalitis.

Giardia and *Cryptosporidium*

New methods to detect *Giardia* cysts in surface water have found widespread contamination[101,186,214] (Table 61-3). Cysts have been found as frequently in pristine water and protected sources as in unprotected waters.[91,192,193] Repeated sampling of "negative" sources invariably produced positive results.

A zoonosis with *Giardia* is known, but with at least three different species, the extent of cross-species infection is not clear.[12,247] Many of the species apparently capable of passing *Giardia* cysts to humans, including dogs, cattle, ungulates (deer), and beaver, are present in wilderness areas. Forty percent of beaver in Colorado were infected and shedding 1×10^8 cysts per animal per day. All 386 muskrats found were infected. Up to 20% of cattle examined were infected.[91] Beaver have been implicated in multiple municipal outbreaks of giardiasis. Samples from Rocky Mountain National Park[117,142] and the California Sierra Nevada[214,218] show a direct correlation between numbers of cysts and levels of human use or beaver habitation. In Yukon, Canada, 13 of 61 scat samples from various wild animals yielded *Giardia* cysts.[187]

Even with a low infectious dose, environmental cyst recovery data indicate that the risk of ingesting an infectious dose of *Giardia* cysts is small.[254,255] However, the likely model that poses a risk to campers is pulse contamination, that is, a brief period of high cyst concentration from fecal contamination. Beaver stool and human stool may contain 1×10^6 cysts/g. Stream contamination from a beaver has been calculated to reach 245 cysts/gallon.[101] In this instance, small amounts of water may cause infection, similar to an outbreak among lap swimmers from inadvertent water ingestion in a fecally contaminated pool.[164]

Cryptosporidium oocysts are found widespread in surface water, and the cyst is durable in the environment. A large zoonosis is evident. Environmental occurrence appears ubiquitous.[121,189,190] *Cryptosporidium* is now found more frequently than *Giardia* in surface water, but in smaller numbers. Large water-borne outbreaks of *Cryptosporidium* have been documented.[29,57,88] An outbreak of *Cryptosporidium* in a public water supply in Milwaukee affected more than 400,000 people.[128] A surface water outbreak of *Cryptosporidium* occurred at Lake Mead (Arizona/Nevada).

Parasitic Organisms

Parasitic organisms other than protozoa are seldom considered in discussions of disinfection. Infectious eggs or larvae of many

helminths are found in sewage, even in the United States.[179,203] The frequency of infection by water-borne transmission is unknown, because food and environmental contamination or skin penetration is more prevalent.[250]

The most obvious risk is from nematodes, with no intermediate hosts, which are infectious immediately or soon after eggs are passed in stool. *Ascaris lumbricoides* (roundworm) is transmitted by ingestion of the eggs in contaminated food or drink. In endemic areas, 85% of the population is infected; this leads to daily global environmental contamination by 9×10^{14} eggs.[250] *Ancylostoma duodenale* (hookworm) usually infects as larvae penetrating the skin of the foot, but it also may be acquired by mouth. Oral entry of the larvae causes pulmonary (Wakana) disease. *Necator americanus* (hookworm) does not appear to be infectious via the oral route.

Taenia solium (pork tapeworm) is infectious to humans in cyst or egg form. Eggs passed in stool are ingested in food or water and develop into tissue cysts, often in the brain, resulting in cysticercosis.

Echinococcus granulosus (dog tapeworm) can use humans as intermediate hosts. Eggs from the feces of an infected dog or other carnivore are ingested in food and water. Hydatid disease generates cysts in the liver, peritoneum, and other sites.

Fasciola hepatica (liver fluke of herbivores and humans) is normally acquired by ingestion of encysted metacercariae on water plants or free organisms in water.

Cercariae of schistosomiasis, which live in fresh water and normally enter through skin, can enter through the oral mucosa. The cercariae are killed by stomach acid.

Dracunculus medinensis (Guinea tapeworm) is a tissue nematode of humans and causes the only such disease transmitted exclusively through drinking water.[242] *Dracunculus* larvae are released in water from subcutaneous worms on the legs of infected bathers or water-gatherers. Larvae are ingested by a tiny crustacean (*Cyclops* species), which acts as the intermediate host and which releases infectious larvae when ingested by humans. Worldwide eradication of dracunculosis has nearly been accomplished.

Bacterial Spores

Bacterial spores can cause serious wound and gut infections but are not likely to be water-borne enteric pathogens. *Clostridium* is ubiquitous in soil, lake sediment, tropical water sources, and the stool of animals and humans.[89,199] *C. botulinum* and *C. perfringens* type A food poisoning are not water-borne because they require germination of spores in food by inadequate cooking, then production of an enterotoxin, which is ingested. *C. perfringens* type C causes enteritis necroticans, probably through in vivo production of an enterotoxin, and thus has the potential for water-borne transmission in the tropics. However, the epidemiology of these infections in the United States, as in infant botulism, is related to food-borne sources.

Chemical Hazards

Chemical hazards are also becoming an alarming source of pollution in surface water. Wilderness users must consider removal of chemical as well as microbiologic contaminants. The greatest risks to wilderness travelers are pesticides from agricultural runoff and heavy metals from old mine tailings. In the United States, chemical contamination is routinely responsible for about 30% of water-borne gastroenteritis where an etiology can be identified.[34,35] Industrialization proceeds worldwide without adequate environmental protection. A vast array of toxins are sold with little concept of safe use and no means of safe disposal. Inorganic chemicals in drinking water include common salts, asbestos, fluorides, nitrates, radionuclides, and some heavy metals (arsenic, copper, iron, lead, selenium). Natural organic chemicals predominate from soil runoff, forest canopy aquatic biota, and human and animal wastes. Synthetic organic matter includes pesticides, herbicides, and chemicals from industrial or human activities.[230] Major underground aquifers are becoming contaminated. Streams and rivers in rural areas are contaminated by individual carelessness, leaching landfills, and agricultural runoff. Numerous pesticides have been found in runoff and rivers in agricultural areas of the Midwest.[138] Atmospheric spread has resulted in pesticides being found in remote wilderness lakes and in the well-publicized acid rain.

Persistence of Enteric Pathogens in the Environment

Once environmental contamination has occurred, a natural inactivation or die-off begins. However, enteric pathogens can retain viability for long periods (Table 61-4).[193,230] Factors promoting survival of microorganisms are pH near neutral (between 6 and 8) and cold temperatures, which explain the risk of transmission in mountain regions. In temperate and warm water, survival is measured in days, with densities of infectious agents decreasing by 90% every 60 minutes. However, tropical water differs from temperate water in nutrients that create a microbiologically rich environment. Coliform bacteria can survive several months in natural tropical river water and may even proliferate. Survival of other bacteria is also much longer (about 200 hours in tropical compared with 30 hours in temperate water). *E. coli* and *V. cholerae* may occur naturally in tropical waters and are capable of surviving indefinitely.[52,89,160]

Most enteric organisms, including *Shigella,* resist freezing.[60] *Salmonella typhosa (typhi)* can survive for up to 5 months in frozen debris and ice.[244] HAV survives 6 months at below-freezing temperatures.[224] *Cryptosporidium* may be able to survive a week or more in home freezers.[216]

Natural Purification Mechanisms

It is widely believed that streams purify themselves and that certain water sources are reliably safe for drinking. These concepts have some truth but do not preclude the need for disinfection to ensure water quality (Box 61-4).

Surface water is subject to frequent, dramatic changes in microbial quality as a result of activities on a watershed. Storm water causes deterioration of source water quality by increasing suspended solids, organic materials, and microorganisms. Some of these contaminants are carried by rain from the atmosphere, but most come from ground runoff. In water sources downstream from towns or villages, storms may overload sewage facilities and cause them to discharge directly into the receiving water. However, rainwater can also flush streams clean by dilution and by washing microbe-laden bottom sediments downstream.[77,89]

Every stream, lake, or groundwater aquifer has limited capacity to assimilate waste effluents and storm water runoff entering the drainage basin. Self-purification is a complex process that involves settling of microorganisms after clumping or adherence to particles, sunlight providing ultraviolet destruction, natural die-off, predators eating bacteria, and dilution.

TABLE 61-4. Viability of Enteric Pathogens in Water

ORGANISM	CONDITIONS	SURVIVAL	REFERENCE
Vibrio cholerae	Cold	4–5 wk	Felsenfeld, 1965[69]
V. cholerae	Tropical	>1 yr	Perez-Rosas, 1989[144]
Campylobacter	Cold	3–5 wk	Blaser, 1980[19]
Campylobacter	Temperate stream	3–10 days	Singh, 1990[180]
Escherichia coli	Temperate stream	13 hr	Singh, 1990[180]
E. coli	Tropical	>1 yr	Perez-Rosas, 1989[144]
Salmonella	Temperate stream	Half-life 16 hr	Perez-Rosas, 1989[144]
Yersinia	Temperate stream	540 days	Singh, 1990[180]
Shigella	Temperate stream	Half-life 22 hr	Singh, 1990[180]
Shigella	Freeze/thaw	Yes	DuPont, 1969[61]
Enteric pathogens	Freeze/thaw	Yes	Dickens, 1985[60]
Salmonella typhosa	Ice/frozen debris	5 mo	White, 1992[210]
Viruses	Cold	17–130 days	Sattar, 1978[173]; World Health Organization, 1979[218]
Enteric viruses	15–25° C (59–77° F) water	6–10 days	Rose, 1996[287]
Enteric viruses	4° C (39.2° F) water	30 days	Rose, 1996[287]
Hepatitis A virus	Cold	1 yr	Biziagos, 1988[17]; Thraenhart, 1991[192]
Hepatitis A	Fresh, sea, wastewater	12 wk	Biziagos, 1988[17]
Hepatitis A	<0° C (32° F)	6 mo	Thraenhart, 1991[192]
Giardia	Cold	2–3 mo	DeReigner, 1989[59]; Bingham, 1979[15]
Giardia	15° C (59° F) lake, river	10–28 days	DeReigner, 1989[59]
Entamoeba histolytica	Cold	3 mo	Chang, 1953[46]
Microsporidia	4° C (39.2° F)	>1 yr	Marshall, 1997[266]
Cryptosporidium	Cold	12 mo	Current, 1985[58]
Ascaris eggs	Wet or dry	6–9 yr	World Health Organization, 1981[217]
Hookworm larvae	Wet sand	122 days	World Health Organization, 1981[217]

Box 61-4. Water Quality: Key Points

- In wilderness water, most sediment is inorganic and clarity is not an indication of microbiologic purity.
- In general, cloudiness indicates higher risk of contamination.
- A major factor determining the amount of microbe pollution in surface water is human and animal activity in the watershed.
- Streams do not purify themselves.
- Settling effect of lakes may make them safer than streams, but care should be taken not to disturb bottom sediments when obtaining water.
- Groundwater is generally cleaner than surface water because of the filtration action of overlying sediments.

Environmental factors include water volume and temperature, hydrologic effects, acid soil contact, and solar radiation. The process is time dependent and less active during wet periods and winter conditions. Hours needed in flow time downstream to achieve a 90% bacterial kill by natural self-purification vary with pollution inflow and rate of water flow. They have been measured at approximately 50 hours in the Tennessee River in summer, 47 hours in the Ohio River in summer, and 32 hours in the Sacramento River.[77]

Storage in reservoirs or lakes also improves microbiologic quality, with sedimentation as the primary process. A 100- to 1000-fold increase in fecal coliform bacteria can be found in bottom sediments compared with overlying water. This removal must be considered temporary, influenced by recirculation of organisms trapped in bottom sediments.[59,76] In optimal conditions, 10 days of reservoir storage can result in 75% to 99% removal of coliform bacteria and 30 days can produce safe drinking water. Generally, 80% to 90% of bacteria and viruses are removed by storage, depending on inflow and outflow, temperature, and no further contamination. Cysts, with a larger size and greater weight, should settle even faster than bacteria and viruses.[2]

Groundwater is generally cleaner than surface water because of the filtration action of overlying sediments, but wells and aquifers can be polluted from surface runoff. Spring water is generally of higher quality than surface water, provided that the true source is not surface water channeling underground from a short distance above the spring.

Drawing conclusions from the preceding factors is difficult. The major factor governing the amount of microbe pollution in surface water is human and animal activity in the watershed. The settling effect of lakes may make them safer than streams, but care should be taken not to disturb bottom sediments when obtaining water.

STANDARDS

Because coliforms originate primarily in the intestinal tracts of warm-blooded animals, including humans, they are used as indicators of possible fecal contamination.[89] Although compelling reasons exist for testing other organisms before determining the safety of drinking water, cost and relative difficulty in testing for viruses and protozoa are major obstacles to expanding routine water testing. Coliforms remain the worldwide standard indicator organism. Only recently (1998) have

U.S. regulations stated that testing must be done for specific organisms, mainly *Cryptosporidium* and other organisms. In the future, molecular probes should make this process much easier.[141,221]

The basic federal law pertaining to drinking water is the 1974 Safe Drinking Water Act, which was expanded and strengthened by amendments in 1977, 1986, and 1996.[141] Additional rules were added in 1989, 1996, and 1998.[39] The U.S. Public Health Service recommendations for potable water specify a mean of one coliform organism per 100 mL of water, or 10 organisms per liter. Absolute limits are three coliform bacteria per 50 mL, four per 100 mL, and 13 per 500 mL.[238] In 1989, the standards for detection of fecal coliform bacteria in drinking water were relaxed slightly in recognition that coliform bacteria occur in large numbers in many water distribution systems that have no problem with water-borne disease.[236] However, sampling was increased. Generally, the goal is to achieve a 3- to 5-log reduction in the level of microorganisms. Treatment must reduce *Giardia* by 99.9% (3 log) and enteric viruses by at least 99.99% (4 log).[176] All standards acknowledge the impracticality of trying to eliminate all microorganisms from drinking water, allowing a small risk of enteric infection.[208] Risk models are used to predict levels of illness and desired levels of reduction. For example, EPA guidelines suggest *Giardia* cyst removal with the goal of ensuring high probability that consumer risk is no more than one infection per 10,000 people per year.[177] The concept of risk is important for wilderness travelers as well, because it is impossible to know the risk of drinking the water in advance and may not be practical to eliminate all risk with treatment.

Standards for Portable Disinfection Products: EPA Registration

Mechanical Filters

Until recently, no testing criteria were mandated for EPA registration. The EPA does not endorse, test, or approve mechanical filters; it merely assigns registration numbers.[4] However, registration requirements distinguish between two types of filters: those that use mechanical means only and those that use a chemical, designated as a pesticide. Standards were developed to act as a framework for testing and evaluation of water purifiers for EPA registration, as a testing guide to manufacturers, to assist in research and development of new units, and as a guide to consumers.[236]

Filter Testing

Current registration of mechanical filters requires only that the product make reasonable claims and that the location of the manufacturer be listed; no disinfection studies are required.[24] However, many companies now use the standards as their testing guidelines. For mechanical filters, the standards should be applied only for those microorganisms against which claims are made, such as protozoa and bacteria, excluding viruses. Despite criticisms of the methodology and inconsistencies and loopholes in the reporting process, the EPA standards are currently the best means to compare filters.

The EPA standards include performance-based microbiologic reduction requirements, chemical health limits for substances that may be discharged, and stability requirements for chemicals sufficient for the shelf life of the device. The unit should signal the end of effective lifetime (e.g., by terminating discharge of treated water) or give simple instructions for servicing or replacing within measurable volume, throughput, or time frame. There are currently no national guidelines for the removal of chemicals by portable filters.

EPA standards require that challenge water seeded with specific amounts of microorganisms be pumped through the filters at given intervals during the claimed volume capacity of the filter. Between the bacteriologic challenges, different test waters without organisms are passed through the unit. Water conditions are specified to include average and worst-case conditions, which are 5° C (41° F) with high levels of pollution, turbidity, and alkaline pH. Testing must be done with bacteria *(Klebsiella),* viruses (poliovirus and rotavirus), and protozoa (*Cryptosporidium* has replaced *Giardia*). A 3-log reduction (99.9%) is required for cysts, 4-log reduction (99.99%) for viruses, and 5- to 6-log reduction for bacteria. Testing is done or contracted by the manufacturer; the EPA neither tests nor specifies laboratories.

To be called a "microbiologic water purifier," the unit must remove, kill, or inactivate all types of disease-causing microorganisms from the water, including bacteria, viruses, and protozoan cysts, so as to render the processed water safe for drinking. An exception for limited claims may be allowed for units removing specific organisms to serve a definable environmental need, for example, removal of protozoan cysts.

Chemical Methods

Products that are used for treating municipal or private water supplies for drinking are considered pesticides and must be registered by the EPA Pesticide Branch. Registration signifies the following:

1. The composition is such as to warrant the proposed claims.
2. The labeling and other material required to be submitted comply with the requirements of the act.
3. The method will perform its intended function without unreasonable adverse effects on the environment.
4. When used in accordance with widespread and commonly recognized practice, the method will not generally cause unreasonable adverse effects on the environment.

Thus, EPA registration implies only that the "pesticide" agent is not released into the water at unsafe levels.[4,28] This is less stringent than for filters that contain halogens.

DISINFECTION METHODS

Definitions (Box 61-5)

Disinfection, the desired result of field water treatment, means the removal or destruction of harmful microorganisms. Technically, it refers only to chemical means such as halogens, but the term can be applied to heat and filtration. Pasteurization is similar to disinfection but specifically refers to the use of heat, usually at temperatures below 100° C (212° F) to kill most pathogenic organisms. Disinfection and pasteurization should not be confused with sterilization, which is the destruction or removal of all life forms.[118] The goal of disinfection is to achieve potable water, indicating only that a water source, on average over a period of time, contains a "minimal microbial hazard," so that the statistical likelihood of illness is acceptable. Water sterilization is not necessary, because not all organisms are enteric human pathogens.[94] *Purification* is the removal of organic or inorganic chemicals and particulate matter to remove

Box 61-5. Definition of Terms

Clarification	Techniques that reduce turbidity of water.
Coagulation-flocculation	Removes smaller suspended particles and chemical complexes too small to settle by gravity (colloids).
Contact time	The length of time that the halogen is in contact with microorganisms in the water.
Disinfection	A process that kills or destroys nearly all disease-producing microorganisms, except bacterial spores. As applied here, the term refers to pathogenic water-borne microbes and is the desired result of water treatment.
Enteric pathogen	Microorganisms capable of causing intestinal infection after ingestion; may be transmitted through food, water, or direct fecal-oral contamination.
Halogen	Oxidant chemical that can be used for disinfection of water.
Halogen demand	The amount of halogen reacting with impurities in the water.
Potable	Implies "drinkable" water, but technically means that a water source, on average, over a period of time, contains a "minimal microbial hazard," so that the statistical likelihood of illness is acceptable.
Purification	Frequently confused with "disinfection," but is more accurately used to indicate the removal of organic or inorganic chemicals and particulate matter to improve offensive color, taste, and odor.
Residual halogen concentration	The amount of active halogen remaining after halogen demand of the water is met.
Reverse osmosis	A process of filtration that uses high pressure to force water through a semipermeable membrane that filters out dissolved ions, molecules, and solids.
Sterilization	A process by which all forms of microbial life, including bacteria, viruses, protozoa and spores are destroyed.

offensive color, taste, and odor. It is frequently used interchangeably with disinfection, but purification may not remove or kill enough microorganisms to ensure microbiologic safety.[242]

HEAT DISINFECTION

Heat is the oldest means of water disinfection. It is used worldwide by residents, travelers, and campers to provide safe drinking water. In countries with normally safe drinking water, it is often recommended as backup in emergencies or when water systems have become contaminated by floods or a lapse in water treatment plant efficacy. Fuel availability is the most important limitation to using heat. One kilogram of wood is required to boil 1 L of water.[43] For wilderness travelers without access to wood, liquid fuel is heavy (Box 61-6).

Box 61-6. Heat

Advantages	Disadvantages
Does not impart additional taste or color to water	Does not improve the taste, smell or appearance of poor-quality water
Single-step process that inactivates all enteric pathogens	Fuel sources may be scarce, expensive, or unavailable
Efficacy is *not* compromised by contaminants or particles in the water, as happens with halogenation and filtration	Does not prevent recontamination during storage
Can pasteurize water without sustained boiling	

Relative susceptibility of microorganisms to heat: protozoa > bacteria > viruses.

Heat inactivation of microorganisms is exponential and follows first-order kinetics. Time plotted against temperature yields a straight line when plotted on a logarithmic scale.[107] The thermal death point is reached in shorter time at higher temperatures, whereas lower temperatures are effective with a longer contact time. Pasteurization uses this principle to kill enteric food pathogens and spoiling organisms at temperatures between 60° C and 70° C (140° and 158° F), well below boiling.[74] Therefore, the minimum critical temperature is well below the boiling point at any terrestrial elevation.

Microorganisms have varying sensitivity to heat; however, all common enteric pathogens are readily inactivated by heat (Table 61-5). Bacterial spores (e.g., *Clostridium* spp.) are the most resistant; some can survive 100° C (212° F) for long periods but, as discussed, are not likely to be water-borne enteric pathogens. Boiling is not dependent on water quality as are filtration or chemical disinfection. Heat kills or inactivates all enteric water-borne pathogens, regardless of whether they are freely suspended or present in particles.[201] Protozoal cysts, including *Giardia* and *Entamoeba histolytica,* are the most susceptible to heat. *Cryptosporidium* is also inactivated at these lower pasteurization levels.

Parasitic eggs, larvae, and cercariae are all susceptible to heat. For most helminth eggs and larvae, which are more resistant than cercariae and *Cyclops* (the copepod that carries *Dracunculus*), the critical lethal temperature is 50° to 55° C (122° to 131° F).[203]

Common bacterial enteric pathogens *(E. coli, Salmonella, Shigella)* are killed by standard pasteurization temperatures of 55° C (131° F) for 30 minutes or 65° C (149° F) for less than 1 minute.[74,149] Recent studies confirmed safety of water contaminated with *V. cholerae* and *E. coli* after 10 minutes at 60°

TABLE 61-5. Data on Heat Inactivation of Microorganisms

ORGANISM	LETHAL TEMPERATURE/TIME	REFERENCE
Giardia	55°C (131°F) for 5 min	Jarrol, 1984[99]
	100°C (212°F) immediately	Bingham, 1979[26]
	50°C (122°F) for 10 min (95% inactivation)	Ongerth, 1989[142]
	60°C (140°F) for 10 min (98% inactivation)	
	70°C (158°F) for 10 min (100% inactivation)	
	55°C (131°F)	Aukerman, 1989[5]
Entamoeba histolytica	Similar to *Giardia*	
Nematode cysts, helminth eggs, larvae, cercariae	50–55°C (122–131°F)	Shephart, 1977[179]
Cryptosporidium	45–55°C (113–131°F) for 20 min	Anderson, 1985[15]
	55°C (131°F) warmed over 20 min	
	64.2°C (148°F) within 2 min	Fayer, 1994[240]
	72°C (162°F) heated up over 1 min	
Escherichia coli	55°C (131°F) for 30 min	Frazier, 1978[74]
	60–62°C (140–144°F) for 10 min	Neumann, 1969[135]
Salmonella and *Shigella*	65°C (149°F) for <1 min	
Vibrio cholerae	60–62°C (140–144°F) for 10 min	Rice, 1991[160]
	100°C (212°F) for 30 sec	
E. coli, Salmonella, Shigella, Campylobacter	60°C (140°F) for 3 min (3-log reduction)	Bandres, 1988[228]
	65°C (149°F) for 3 min (all but few *Campylobacter*)	
	75°C (167°F) for 3 min (100% kill)	
E. coli	50°C (122°F) for 10 min ineffective	Groh, 1996[245]
	60°C (140°F) for 5 min	
	70°C (158°F) for 1 min	
Viruses	55–60°C (131–140°F) within 20–40 min	Alder, 1992[13]
	70°C (158°F) for <1 min	
Hepatitis A	98°C (208°F) for 1 min	Krugman, 1970[116]
	85°C (185°F) for 1 min	Thraenhart, 1991[192]
	61°C (142°F) for 10 min (50% disintegrated)	
	60°C (140°F) for 19 min (in shellfish)	Peterson, 1978[147]
Hepatitis E	60°C (140°F) for 30 min	Thraenhart, 1991[192]
Bacterial spores	>100°C (212°F)	Alder, 1992[13]

to 62°C (140° to 143.6°F) or after boiling water for 30 seconds.[86,183]

Viruses are more closely related to vegetative bacteria than to spore-bearing organisms[107] and are generally inactivated at 56° to 60°C (132.8° to 140°F) in less than 20 to 40 minutes.[1,161,220] Inactivation at higher temperatures is similar to that of vegetative bacteria. Death occurs in less than 1 minute above 70°C (158°F). This has been confirmed in milk products, despite some degree of thermal protection from particles.[219]

Given its environmental stability and clinical virulence, HAV is a special concern. It should respond to heat as do other enteric viruses, but data indicate that it has greater thermal resistance. Widely varying data probably result from different models for virus infectivity and destruction and from the use of various test media.

Boiling Time

The boiling time required is important when fuel is limited. The old recommendation for treating water was to boil for 10 minutes and add 1 minute for every 1000 feet (305 m) in elevation. However, available data indicate this is not necessary for disinfection. Evidence indicates that enteric pathogens are killed within seconds by boiling water and rapidly at temperatures above 60°C (140°F). In the wilderness, the time required to heat water from 55°C (131°F) to boiling temperature works toward disinfection. Therefore, any water brought to a boil should be adequately disinfected.[209a] An extra margin of safety can be added by boiling for 1 minute or by keeping the water covered for several more minutes, which will maintain high temperature without using fuel, or allowing it to cool slowly. Although the boiling point decreases with increasing altitude, this is not significant compared with the time required for thermal death at these temperatures (Table 61-6). In recognition of the difference between pasteurizing water for drinking purposes and sterilizing to kill all microbes, including spores, for surgical purposes, many other sources, including WHO, now agree with this recommendation to simply bring water to a boil. Because of scant data for hepatitis A, the Centers for Disease Control and Prevention (CDC) and the EPA still rec-

TABLE 61-6. Boiling Temperatures at Various Altitudes

ALTITUDE (ft)	ALTITUDE (m)	BOILING POINT
5,000	1524	95°C (203°F)
10,000	3048	90°C (194°F)
14,000	4267	86°C (187°F)
19,000	5791	81°C (179°F)

ommend boiling for 1 minute to add a margin of safety.[32] Other sources still suggest 3 minutes of boiling time at high altitude to give a wide margin of safety.[37,76,190,231]

Hot Tap Water

Although attaining boiling temperature is not necessary, it is the only easily recognizable end point without using a thermometer. Other markers, such as early bubble formation on the bottom of the pot, do not occur at a consistent temperature. When no other means are available, the use of hot tap water may prevent travelers' diarrhea in developing countries. Newman[149,150] cultured samples from the hot water tap of 17 hotels in West Africa and in 15 found no coliforms; one yielded a single colony and another two colonies. Water temperature ranged from 57° to 69° C (131° to 140° F). As a rule of thumb, water too hot to touch fell within the pasteurization range. Bandres and colleagues[8] also measured hot tap water temperature in 14 hotels in four different countries outside the United States. Most temperatures were 55° to 60° C (131° to 140° F), but one was 44° C (111.2° F), one was 65° C (149° F), and several were 52° C (125.6° F). The authors concluded that hot water from taps would not be safe to drink. Groh and colleagues[86] showed that tolerance to touch is too variable to be reliable, in that some people found 55° C (131° F) too hot to touch. From these studies, one can conclude that if water has been sitting in a tank near 55° to 60° C (131° to 140° F) for a prolonged period, enteric pathogens will be significantly reduced, likely to potable levels. Neumann's suggestion is reasonable if no other method of water treatment is available.

Solar Heat

Pasteurization has been successfully achieved using solar heating. A solar cooker constructed from a foil-lined cardboard box with a glass window in the lid can be used for disinfecting large amounts of water by pasteurization. Bottom temperatures of 65° C (149° F) have been obtained for at least 1 hour in up to three 3.7-L jugs. Exposure to full sunshine in Kenya destroyed *E. coli* in 2-L clear plastic bottles within 7 hours when the maximum temperature reached 55° C (131° F). No thermal inactivation occurs below 40° C (104° F). Inactivation in this situation was a combination of thermal and ultraviolet irradiation.[108,134] This could be a low-cost method for improving water quality, especially in refugee camps and disaster areas. (See Ultraviolet Light discussion.)

PHYSICAL REMOVAL

Turbidity and Clarification

River, lake, or pond water is often cloudy and unappealing. Turbidity (cloudiness) is an optical measurement of light scattering as it passes through water. Visibility in water with turbidity of 10 nephelometric turbidity units (NTU) is about 30 inches and with 25 NTU is 10 inches. Turbidity is caused by suspended organic and inorganic matter, such as clay, silt, plankton, and other microscopic organisms. High turbidity is often associated with unpleasant odors and tastes, most often caused by organic compounds and metallic hydroxides with a much smaller particle size.[47,120] Clay-organic complexes may also carry pesticides or heavy metals. Bacteria, as well as viruses, may be adsorbed to particulate matter or be embedded in it, and in highly contaminated water, microorganisms tend to aggregate and clump.

Box 61-7. Summary of Clarification Techniques

Technique	Process	Uses, Advantages
Sedimentation	Settling by gravity of large particulates	Requires long time Greatly improves water esthetics
Coagulation-flocculation	Removes suspended particles, most microorganisms, some dissolved substances	Simple process, easily applied in field Greatly improves water quality Improves efficacy of filtration and chemical disinfection
Activated charcoal	Removes organic and some inorganic chemicals	Removes toxins such as pesticides and removes chemical disinfectants Improves taste of water
Filtration	Physical and chemical process	Removes microorganisms If charcoal stage, may improve taste and remove chemicals

In one study, 17% of turbidity particles contained attached microbes, averaging 10 to 100 bacteria per particle.[120] Organisms in the center of these conglomerates are afforded some protection from disinfectants. Even the flocculate produced by a chlorination-flocculation tablet may harbor viable organisms.[167] Thus, removing particulate matter also decreases the number of microorganisms and halogen demand.[109,146] So, removal of turbidity and particulates may be important in preventing chemical or infectious illness.

Clarification to remove turbidity is an important first initial step before using filtration or chemical disinfection that greatly improves the efficacy of these techniques[200] (Box 61-7). Even when turbidity is caused by benign inorganic particles, such as clay, removal is desirable for improving the esthetic quality of the water. Filtration can remove larger particles, but cloudy water can rapidly clog a filter. Sedimentation and coagulation-flocculation are other clarification techniques routinely used in municipal disinfection plants that can be easily applied in the wilderness for pretreatment of cloudy water, which is then disinfected by microfiltration or halogenation. Coagulation-flocculation and filtration are also used to remove *Giardia* and *Cryptosporidium* cysts that are more resistant to chlorine. Early experiments with water heavily contaminated with feces containing HAV demonstrated that filtration and sedimentation alone did not prevent infection, but reduced the severity of the illness. Water pretreated with coagulation, settling, and filtration and subsequently disinfected with 0.4 ppm of residual chlorine was noninfectious, whereas water chlorinated to 1 mg/L without pretreatment remained infectious.[147,148]

Sedimentation

Sedimentation is the separation of suspended particles large enough to settle rapidly by gravity, such as sand and silt. Water

Box 61-8. Coagulation-Flocculation

Advantages	Disadvantages
Highly effective to clarify water and remove many microorganisms	Unfamiliar technique and substances
Improves efficacy of filtration and chemical disinfection	Adds extra step unless combined flocculent–disinfectant tablet
Inexpensive and widely available	
Simple process with no toxicity	

Relative susceptibility of microorganisms to coagulation-flocculation protozoa > bacteria > viruses.

is allowed to sit without agitation. After sediment has formed on the bottom of the container, the clear water is decanted or filtered from the top. The time required depends on the size of the particle. Generally, 1 to 3 hours are adequate for large particles such as inorganic sands and silts. Microorganisms, especially protozoal cysts, eventually settle; reductions up to 90% may be achieved overnight or in 1 to 2 days.[43] However, the organisms are easily disturbed during pouring or filtering. Reservoirs take advantage of sedimentation to improve microbiologic quality. In conclusion, sedimentation is often effective in reducing water turbidity and improves microbiologic quality, but it is not recommended as the sole means of disinfection.

Coagulation-Flocculation

Smaller suspended particles and chemical complexes too small to settle by gravity are called *colloids*. Most of these can be removed by chemical precipitation, known as coagulation-flocculation, a technique that has been used to remove unpleasant color, smell, and taste in water since 2000 BC. This technique is used routinely in large municipal disinfection plants, but is simple enough to be used at the household level and in the outdoors[209] (Box 61-8).

Coagulation is achieved with addition of an appropriate chemical that alters the physical state of dissolved and suspended solids, causing particles to stick together on contact because of electrostatic and ionic forces.[47,230] Aluminum salts (alum), iron, and lime (alkaline chemicals principally containing calcium or magnesium and oxygen) are commonly used, readily available coagulants. Rapid mixing is important to obtain dispersion of the coagulant. The second stage, flocculation, is a purely physical process obtained by prolonged gentle mixing to increase interparticle collisions and promote formation of larger particles. The flocculate particles can be removed by sedimentation and filtration. Coagulation-flocculation removes most coliform bacteria (60% to 98%), viruses (65% to 99%),[55,174,208] *Giardia* (60% to 99%), helminth ova (95%),[203] heavy metals, dissolved phosphates, and minerals.[47,126,230,249] Organic and inorganic compounds may be removed by forming a precipitate or by adsorbing onto aluminum hydroxide or ferric hydroxide floc particles.[230] Despite removal of most microorganisms, a subsequent disinfection step is advised.

The sequential use of coagulation and activated carbon is often beneficial. Coagulation generally removes large molecules that absorb poorly on granular activated carbon (GAC). On the other hand, carbon has limited effectiveness for removing organic matter from water.[6]

To clarify water by coagulation-flocculation in the field, add 10 to 30 mg of alum per liter of water. The exact amount is not important, so it can be done with a pinch of alum, lime (calcium oxide), or both for each gallon of water, using more if the water is very cloudy. Next, stir or shake briskly for 1 minute to mix the coagulant, then agitate gently and frequently for at least 5 minutes to assist flocculation. Settling requires at least 30 minutes, after which the water is carefully decanted or poured through a cloth or paper filter. The process can be repeated, if necessary. Finally, filtration or halogenation should be used to ensure disinfection.

Toxicity

Questions have been raised concerning the association of aluminum with central nervous system toxicity in mammals, but these effects have been observed only after exposures other than ingestion. Most of the aluminum in alum is removed with the floc. A report from the National Academy of Sciences concluded that aluminum in drinking water does not present a significant risk.[230] Alum is a common chemical used by the food industry in baking powder and for pickling. It can be found in some food stores or at chemical supply stores.

Alternative Agents

Many inorganic and organic compounds can be used as a coagulant, including lime (calcium oxide) or potash (from wood ash). In an emergency, baking powder or even the fine white ash from a campfire can be used.[233] Other coagulation-flocculation agents used traditionally by native peoples include seed extracts from the nirmali plant in southern India, or moringa plants in Sudan, crushed almonds or dried, crushed beans, and rauwaq (a form of bentonite clay).[43]

Adsorption

Adsorbents such as charcoal, clay, and other types of organic matter have been used for water treatment since biblical times.[119] These processes are often combined with filtration or coagulation, because these substances are used as the filter media and act as coagulants.[209] Clays can decrease turbidity and microbes in water by about 90% to 95%, but the process is difficult to control and adsorption is not the main action of ceramic or clay filters. Vegetative matter, including burnt rice hulls and activated (burnt) coconut shell, also have adsorptive capacity.

Granular Activated Carbon (Charcoal)

Granular charcoal is widely used for water treatment and for medical detoxification. When activated, charcoal's regular array of carbon bonds is disrupted, yielding free valences that are highly reactive and adsorb dissolved chemicals.[78,194] GAC is the best means to remove toxic organic and inorganic chemicals from water (including disinfection byproducts) and to improve odor and taste.[146,230] Thus, it is widely used in municipal disinfection plants and in home under-sink devices. Compressed into block form that acts both as a depth filter and an adsorbent, GAC is a common component of field filters. Block carbon is more effective than granular because the passages are smaller, forcing closer contact with the carbon.

Many viral particles and bacteria adhere to GAC,[146] and some cysts are trapped in the matrix.[126] However, using a bed of GAC to filter particles and microorganisms results in more rapid saturation of binding sites and clogs the bed. Dissolved organic matter in water takes up adsorption sites and bacteria colonize GAC, forming a biofilm that can slough off into the effluent water. GAC does not kill microorganisms; in fact, bacteria attached to charcoal are resistant to chlorination because the chlorine is adsorbed by the GAC.[119,146,230] This bacterial contamination has not been found to be harmful because the usual heterophilic bacteria are not enteric pathogens. Enteric pathogens have been shown to survive on GAC, but if an active biofilm exists, the pathogens are rapidly displaced by heterophilic bacteria and fail to become established. Therefore, nonpathogenic bacterial colonization is encouraged in municipal plants.[178] However, these properties of carbon indicate that it will not reduce pathogenic microbes in water over an extended period of time, and an alternative means of disinfection should always be used.

Eventually, the binding sites on the carbon particles become saturated and no longer adsorb; some molecules are released as others preferentially bind.[146] Unfortunately, no reliable means are available to determine precisely when saturation is reached. Filters using charcoal in compressed block form as the filter element may clog before the charcoal is fully adsorbed. Presence of unpleasant taste or color in the water can be the first sign that the charcoal is spent. To test the charcoal, filter iodinated water or water tinted with food coloring. With regular use the lifetime of GAC is probably measured in months; it is substantially longer with infrequent use. GAC can be "recharged," but this is not practical for small-quantity use. Ingested particles of charcoal are harmless.

GAC can be used before or after disinfection. Before disinfection, GAC removes many organic impurities that result in bad odor and taste. GAC is best used after chemical disinfection to make water more palatable by completely removing the halogen[146,244] and other chemical impurities. With increasing industrial and agricultural contamination of distant groundwater, final treatment of drinking water with GAC may be a necessity for some wilderness users. GAC also removes radioactive contamination.

Filtration

Filters are appealing and amenable to commercial production. They have the advantages of being simple and requiring no holding time (Box 61-9). They do not add any unpleasant taste and may improve taste and appearance of water. However, they take space and add weight to baggage. All filters eventually clog from suspended particulate matter, present even in clear streams, requiring cleaning or replacement of the filter. As a filter clogs, it requires increasing pressure to drive the water through, which can force microorganisms through the filter. A crack or eroded channel allows passage of unfiltered water. Filtration is a standard step in municipal disinfection. Many different types of media, from sand to vegetable products to fabric, have been used throughout history in various parts of the world.

Filtration is both a physical and a chemical process, so many variables influence filter efficiency. The characteristics of the filter media and the water, as well as flow rate, determine the interactions. Filtration can reduce turbidity, bacteria, algae, viruses, color, oxidized iron, manganese, and radioactive particles.[55]

Box 61-9. Filtration

Advantages	Disadvantages
Simple to operate	Adds bulk and weight to baggage
Mechanical filters require no holding time for treatment (water is treated as it comes out of the filter)	Most filters not reliable for removal of viruses
Large choice of commercial products	Expensive relative to chemical treatment
Adds no unpleasant taste and often improves taste and appearance of water	Channeling of water or high pressure can force microorganisms through the filter
Rationally combined with halogens for removal or destruction of all pathogenic water-borne microbes	Eventually clogs from suspended particulate matter; may require some maintenance or repair in field

Susceptibility of microorganisms to filtration: protozoa > bacteria > viruses.

The size of a microorganism is the primary determinant of its susceptibility to filtration (Table 61-7 and Fig. 61-1). Filters are rated by their ability to retain particles of a certain size, which is described by two terms. *Absolute rating* means that 100% of a certain size of particle is retained. *Nominal rating* indicates that more than 90% of a given particle size will be retained. Filter efficiency is generally determined with hard particles (beads of known diameter), but microorganisms are soft and compressible under pressure. Water-borne pathogens often adhere to larger particles or clump together, making them easier to remove by physical processes. Therefore, observed reductions are often greater than expected based on their individual sizes.[209]

A membrane with pore size of 0.2 µm can remove enteric bacteria. *Giardia* and *E. histolytica* cysts are easily filtered, requiring a maximum filter size of 5 µm. *Cryptosporidium* cysts are somewhat smaller than *Giardia* and more flexible; 57% are able to pass through a 3-µm membrane filter, so a filter with 1- to 2-µm pores is recommended.[190] Helminth eggs and larvae, which are much larger, can be removed by a 20-µm filter. *Cyclops* (the water-dwelling copepod that ingests the larva and transmits *dracunculosis*) can be removed by passage through a fine cloth.[203]

Many filters constructed with various designs and materials are marketed for field use. Surface, membrane, and mesh filters are very thin with a single layer of fairly precise pores, whose size should be equal to or less than the smallest dimension of the organism. These filters provide little volume for holding contaminants and thus clog rapidly, but they can be cleaned easily by washing and brushing without destroying the filter. Maze or depth filters depend on long, irregular labyrinths to trap the organisms, so they may have a larger pore or passage size. Con-

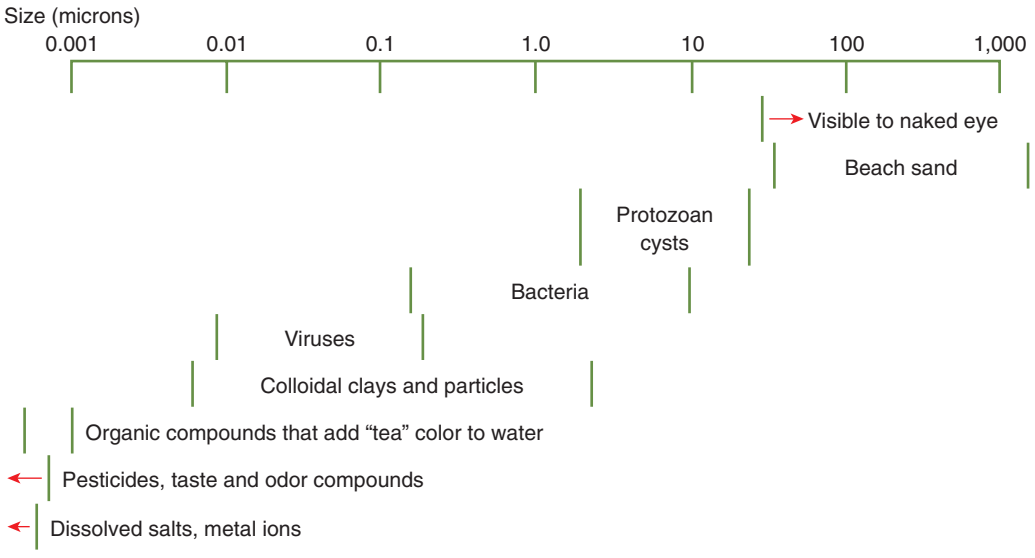

Figure 61-1. Relative size of microorganisms determines susceptibility to mechanical filtration. (Courtesy Dan Vorhis.)

TABLE 61-7. Microorganism Susceptibility to Filtration

ORGANISM	AVERAGE SIZE (μm)	MAXIMUM RECOMMENDED FILTER RATING (μm)
Viruses	0.03	N/S
Escherichia coli	0.5 × 3–8	0.2–0.4
Campylobacter	0.2–0.4 × 1.5–3.5	Same as above
Microsporidia	1–2	N/S
Cryptosporidium oocyst	2–6	1
Giardia cyst	6–10 × 8–15	3–5
Entamoeba histolytica cyst	5–30 (average 10)	Same as *Giardia*
Cyclospora	8–10	Same as *Giardia*
Nematode eggs	30–40 × 50–80	20
Schistosome cercariae	50 × 100	Coffee filter or fine cloth
Dracunculus larvae	20 × 500	Coffee filter or fine cloth

N/S, not specified.

taminants adhere to the walls of the passageway or are trapped in the numerous dead-end tunnels. Granular media, such as sand or charcoal, diatomaceous earth, or ceramic filters, function as maze filters. A depth filter has a large holding capacity for particles and lasts longer before clogging but may be difficult to clean effectively, in that many particles are trapped deep in the filter. Flow can be partially restored to a clogged filter by back flushing or surface cleaning, which removes the larger particles trapped near the surface. For ceramic filters, surface cleaning is highly effective but removes a tiny layer of the filter medium.

Portable filters can readily remove protozoan cysts and bacteria but may not remove viruses, which are another order of magnitude smaller than bacteria. Only the semipermeable membranes in reverse-osmosis filters are inherently capable of removing viruses. However, adsorption and aggregation during passage through mechanical filters reduce viruses. Virus particles may adhere to the walls of diatomite (ceramic) or charcoal filters by electrostatic chemical attraction, which can be enhanced by a coating on the filter or a positive charge.[65,79,82,178] Viruses in heavily polluted water often aggregate in large clumps and become adsorbed to particles or enmeshed in colloidal materials, making them amenable to filtration.[174,230] Thus turbidity (cloudiness from contaminants) may help remove pathogens with filtration, while it inhibits chemical disinfection. In one study, however, only 10% of total virus particles detected were recovered on 3- to 5-μm pore prefilters, suggesting that most were not associated with the suspended sediment.[153] Furthermore, adsorbed viral particles can be subsequently dislodged and eluted from a filter due to competitive binding and competing electrostatic forces.[79,170,227] Some ceramic filters now remove 99% to 99.9% of viruses, but the fourth log required by water treatment units remains a challenge. The First Need filter (General Ecology, Exton, PA) has

been able to meet the EPA standards for water purifiers, including 4-log removal of viruses, apparently through use of a charged media[82] (Appendix A). In general, however, mechanical filters should not be considered adequate for complete removal of viruses, except with special equipment.[242]

For domestic use and in pristine protected watersheds where pollution is minimal and the main concerns are bacteria and cysts, filtration can be used as the only means of disinfection. For foreign travel and for surface water with high levels of human use or sewage contamination, however, most filters should not be used as the sole means of disinfection.[65] Additional treatment with heat or halogens before or after filtration guarantees effective virus removal.[178] One rational use of filtration is to clear the water of sediment and organic debris, allowing lower doses of halogens with more predictable residual levels.[146] Filters are also useful as a first step to remove parasitic and *Cryptosporidium* organisms that have high resistance to halogens.

Filtration using simple, available products is of interest for use in developing countries and in emergency situations.[209] Sand filtration is still used widely in municipal plants. A column of fine sand 60 to 75 cm deep that permits flow of no more than 200 L/m^2/hr is capable of removing turbidity and greater than 99% of organisms.[176] These can be improvised with stacked buckets or barrels. Rice hull ash filters, crushed charcoal, sponges, and various fabrics have all been used. Typically, bacteria and viruses can be reduced by as much as 50% to 85% or more, and larger parasites by 99%, depending on the media. Fine woven cotton fabric is effective at removing larger parasites, such as schistosoma cercariae, *Fasciola* species, and guinea worm larvae. Kozlicic tested five commonly available household materials (newspaper, filter paper, cotton, four-layer gauze, and white cotton cloth) for filtration efficiency. Newspaper performed poorly, being too slow. Cotton cloth performed best for both physical and microbiologic parameters of water (although the latter was poorly studied). He incidentally noted that melted snow with the top 4 to 5 cm removed was a better quality water source than rain water from roof runoff.[114]

Reverse Osmosis

A reverse-osmosis filter uses high pressure (100 to 800 psi) to force water through a semipermeable membrane that filters out dissolved ions, molecules, and solids.[230] This process can desalinate water, as well as remove microbiologic contamination. If pressure or degradation causes breakdown of the membrane, treatment effectiveness is lost. Even *Giardia* cyst passage has been shown to occur in a compromised reverse-osmosis unit.[53]

Small hand-pump reverse-osmosis units have been developed. Their high price and slow output currently prohibit use by land-based wilderness travelers, but they are important survival items for ocean travelers. Battery- or power-operated units are standard equipment on large boats. The U.S. Department of Defense uses large-scale mobile reverse-osmosis units for water treatment. These are considered the most fuel-efficient mobile units, producing the highest quality water from the greatest variety of raw water qualities and capable of producing potable water from fresh, brackish, or salt water, as well as from water contaminated by nuclear, biologic, or chemical agents. The units use pretreatment, filtration, and desalination, then disinfection for storage.[233]

Silver Impregnation

Silver impregnation of filters neither prevents microbial contamination of the filter nor sustains its action as a bactericide in the effluent water.[11] Although silver has some antibacterial effect on coliform organisms, filter cartridges impregnated with silver typically become colonized with heterotrophic bacteria, which increase the total bacterial count in the effluent water but which have not been linked to increased illness.[11,65,78,178] In GAC filters designed to operate in line with chlorinated tap water, silver merely exerts selective pressure on the kinds of bacteria that will colonize the filter. Colonization of filters with pathogenic coliforms has not been demonstrated, but protective effect cannot be attributed to silver impregnation.[65,178]

Commercial Devices Using Mechanical Filtration

See EPA standards already described. Portable water treatment products are the third highest intended purchase of outdoor equipment after backpacks and tents.[106] Some are designed as purely mechanical filters, whereas others combine filtration with GAC. Most of the filters containing iodine resins have been withdrawn from the market. There is currently only one drink-through bottle using an iodine resin (see the discussion of halogens and Appendix A).

The ceramic filters have been tested most extensively and generally consistently perform well.[65,156,227] Results may not apply to all ceramic filters because efficacy depends on the characteristics of the ceramic, water quality, product engineering, and prior extent of filter use. Few comparable data are available on different filters. Most data are from testing organized by one filter manufacturer, so the results are not generally accepted, despite nearly all filters performing well. Schlosser tested three hand-pump filters (Katadyn Mini Ceramic, First Need Deluxe, and Sweetwater Walkabout), all of which removed 3-log (99.9%) or more of viable bacteria, leaving none in the effluent.[200] The military preventive medicine group has enumerated the requirements for individual filters for field use.[229]

HALOGENS

Worldwide, chemical disinfection is the most widely used method for improving and maintaining microbiologic quality of drinking water. Chemical disinfectants used for water disinfection are strong oxidants. Halogens, chiefly chlorine and iodine, are the most common chemical disinfectants used in the field; however, chlorine dioxide is now available in small use applications and is gaining acceptance. Germicidal activity results from oxidation of essential cellular structures and enzymes.[40,118,146,151] Halogenated amines may be synthesized by white blood cells as part of the body's natural defenses to destroy microorganisms.[243] The disinfection process is determined by characteristics of the disinfectant, the microorganism, and environmental factors.[42,96,145] Dilute solutions do not sterilize water. The relative potency of common disinfectants to inactivate water-borne microbes is as follows:

ozone > chlorine dioxide > electrochemically generated mixed species oxident > free chlorine or iodine > chloramine

Ozone and chlorine dioxide are discussed under Miscellaneous Disinfectants.

TABLE 61-8. Factors Affecting Halogen Disinfection

	EFFECT	COMPENSATION
Primary Factors		
Concentration	Measured in milligrams per liter (mg/L) or the equivalent, parts per million (ppm); higher concentration increases rate and proportion of microorganisms killed.	Higher concentration allows shorter contact time for equivalent results. Lower concentration requires increased contact time.
Contact time	Usually measured in minutes; longer contact time assures higher proportion of organisms killed.	Contact time is inversely related to concentration; longer time allows lower concentration
Secondary Factors		
Temperature	Cold slows reaction time.	Some treatment protocols recommend doubling the dose (concentration) of halogen in cold water, but if time allows, exposure time can be increased instead, or the temperature of the water can be increased.
Water contaminants, cloudy water (turbidity)	Halogen reacts with organic nitrogen compounds from decomposition of organisms and their wastes to form compounds with little or no disinfecting ability, effectively decreasing the concentration of available halogen. In general, turbidity increases halogen demand.	Doubling the dose of halogen for cloudy water is a crude means of compensation that often results in a strong halogen taste on top of the taste of the contaminants. A more rational approach is to first clarify water to reduce halogen demand.
pH	The optimal pH for halogen disinfection is 6.5 to 7.5. As water becomes more alkaline, approaching pH 8.0, much higher doses of halogens are required.	Most surface water is neutral to slightly acidic, so compensating for pH is not necessary. Tablet formulations of halogen have the advantage of some buffering capacity.

Variables with Halogenation

Understanding the principal factors of halogen disinfection allows intelligent and flexible use (Table 61-8).

Concentration and Contact Time

The major variables in the disinfection reaction with chlorine or iodine are the amount of halogen (concentration) and the exposure time of the microorganism to the halogen disinfectant (contact time). Concentration of halogen in water is measured in parts per million (ppm) or milligrams per liter (mg/L), which are equivalent. Contact time is usually measured in minutes but ranges from seconds to hours. In field disinfection, concentrations of 1 to 10 mg/L for 10 to 60 minutes are generally effective.

Theoretically, the disinfection reaction follows first-order kinetics. The rate of the reaction is determined by the initial concentration of reactants, and a given proportion of the reaction occurs in any specified interval.[96,244] This means concentration and time are inversely related, and their product results in a constant for specified disinfectant, organism, percent reduction of viable microorganisms, and given conditions of water temperature and pH: concentration × time = constant (Ct = K) (Fig. 61-2).[244] When concentration and contact time are graphed on logarithmic coordinates, a straight line results. This means that concentration and time can be varied oppositely and still achieve the same result.[9] In field disinfection, this can be used to minimize halogen dose and improve taste or to minimize the required contact time.

In reality, the disinfection reaction deviates from first-order kinetics, and Ct values do not follow the exponential rates described by the empiric equation because microorganisms do not act as chemical reagents ($C_n t = K$). An initial lag period may be seen before inactivation begins (e.g., because of penetration of the cyst wall), and inactivation declines for more resistant organisms or those protected by aggregation or association with other particulate matter (Fig. 61-3).[87,91,96]

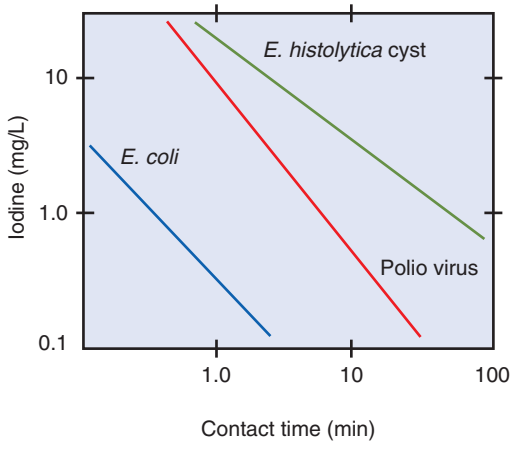

Figure 61-2. Relationship of halogen concentration and contact time for a given temperature and pH. The first-order chemical reaction results in a straight line over most values for each microorganism and halogen compound. (Data from Chang SL: J Am Pharm Assoc 47:417, 1958; and Water and Sanitation for Health [WASH] Project: Report on mobile emergency water treatment and disinfection units, WASH Field Report No 217, Arlington, VA, 1980.)

Contaminants

The quality of the water to be disinfected influences microbial inactivation by chemical disinfectants. Organic and inorganic nitrogen compounds from decomposition of organisms and their wastes, fecal matter, and urea complicate disinfection with halogens and must be considered in field water treatment.

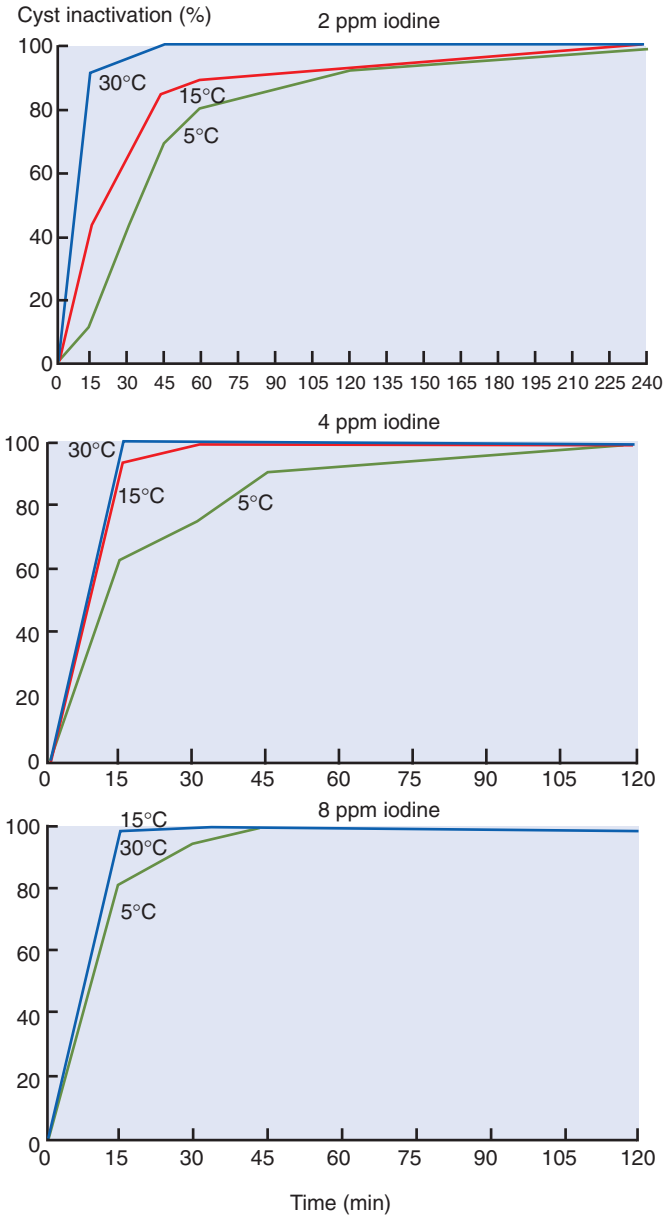

Figure 61-3. Effect of concentration and temperature on *Giardia* cyst inactivation by iodine. Low concentrations are effective at cold temperatures with prolonged contact time. ppm, parts per million. (From Fraker LD, et al: J Wilderness Med 3:351, 1992.)

Vegetable matter, ferrous ions, nitrites, sulfides, and humic substances also affect oxidizing disinfectants.[62,146,244] These contaminants react with halogens, especially chlorine, to form compounds with little or no disinfecting ability, effectively decreasing the concentration of available halogen.

Halogen Demand and Residual Concentration

Halogen demand is the amount of halogen reacting with impurities. Residual halogen concentration is the amount of active halogen remaining after halogen demand of the water is met. To achieve microbial inactivation in aqueous solution with a chemical agent, a residual concentration must be present for a specified contact time. Failure of chlorination in municipal systems to kill cysts or other microorganisms is usually caused by difficulty maintaining adequate residual halogen concentration and contact times, rather than by extreme resistance of the organism.[217]

Halogen demand and residual concentration of surface water are the greatest uncertainties in field disinfection. Nitrogen appears in most natural waters in varying amounts, which relate directly to the sanitary quality of water. Cysticidal dose of halogens is strongly affected by the level of contamination (cyst or viral density) in otherwise clean water.[42,85,217] Scant data are available on halogen demand of surface water (Table 61-9). Clear water is assumed to have minimal demand and cloudy water high demand. Surface water in the wilderness contains 10 times the organic carbon content of aquifer groundwater. The green or brown color in stagnant ponds or lakes or in tropical and lowland rivers is usually caused by organic matter with considerable halogen demand. In some cases, such as runoff after storms and snowmelt, cloudy water may be caused by inorganic sand and clay that exert little halogen demand. In general, chlorine demand rises with increased turbidity.[120] In addition, particulate turbidity can shield microorganisms and interfere with disinfection.[55,109,120] (See previous discussion of turbidity.)

The initial dose of halogen must consider halogen demand. For clear alpine waters, 1 mg/L demand can be assumed; for cloudy waters, the assumption is 3 to 5 mg/L. If a method is used that adds 4 mg/L to clear water, extra time can compensate for the lower expected residual concentration. In cloudy water, however, where the demand may be nearly 4 mg/L, an increased dose of halogen, rather than prolonging the contact time, is needed to ensure free residual. The usual field recommendation to compensate for the unknown demand of cloudy water is a double dose of halogen (to achieve 8 to 16 mg/L). This crude means of compensation often results in a strong halogen taste on top of the taste of the contaminants. If the cause of turbidity is uncertain, the water should be allowed to sit; inorganic clay and sand will sediment, clarifying the water considerably. Other means of clarification, such as coagulation-flocculation or filtration, significantly reduce halogen demand. (See clarification techniques.)

Several simple color tests (most often used to test swimming pools and spas) measure the amount of free (residual) halogen in water. Testing in the wilderness for halogen residual may be reasonable for large groups but is not practical for most. Smell of chlorine usually indicates some free residual. Color and taste of iodine can be used as indicators. Above 0.6 ppm, a yellow to brown tint is noted.[244]

pH and Temperature

Two other variables in the disinfection reaction are pH and temperature.[62,118,146]

pH. Halogen oxidizes water to form several compounds, each with different disinfection capabilities. The percentage of each halogen compound is determined by pH. The optimal pH for halogen disinfection is 6.5 to 7.5.[42,144] As water becomes more alkaline, approaching pH 8.0, much higher doses of halogens are required.

TABLE 61-9. Halogen Demand of Surface Water

SOURCE	HALOGEN DEMAND (mg/L)	REFERENCE
Cloudy river water, Portland, Oregon	3–4	Jarroll, 1980[102]
Cloudy water from clay particles	none	Chang, 1953[46]
Clear water with 10% sewage added	2	Chang, 1953[46]
Lily pond and turbid river water	5–6	Chang, 1953[46]
Colorado River; cloudy from inorganic sand, clay	0.3	Tunnicliff, 1984[228]
Unspecified surface waters	2–3	Culp, 1974[54]
Municipal wastewater	20–30	Culp, 1974[54]
High-elevation spring	0.3	Ongerth, 1989[140]
Western river	0.7	Ongerth, 1989[140]
Six watersheds in western Oregon	0.4–1.6	LeChevallier, 1981[117]
Small stream, Australia	1.3	Thomson, 1985[297]
Bolivian village (well and collected rain)	2	Quick, 1999[364]

Although pH can be measured in the field, the relationship is too complex to allow meaningful use of the information. Most surface water pH is neutral to mildly acidic, which is within the effective range of the halogens used. Granite keeps many alpine waters mildly acidic. Unfortunately, acid rain is affecting some high mountain lakes.[135] The EPA found the average pH in western alpine lakes to be no less than 5.5; other U.S. lakes are beginning to show lower pH levels. On the alkaline side, some surface water with pH 7.0 to 8.0 begins to affect the chemical species of chlorine, favoring less active forms.[96] Certain desert water is so alkaline that halogens would have little activity; however, these waters are usually not palatable. At this time, compensating for pH is not necessary. Tablet formulations of halogen have the advantage of some buffering capacity.

Temperature. Temperature influences the rate of the disinfection reaction. Cold water affects germicidal power and must be offset by longer contact time or higher concentration to achieve comparable disinfection.[84] The common rule is a twofold to threefold increase in inactivation rate per 10°C (18°F) increase in temperature. Unusual retardation of rates as temperatures approach 0°C (32°F) has not been seen.[96]

Temperature can be estimated in the field. Some treatment protocols recommend doubling the dose of halogen in cold water, but if there is no urgency, time can be increased instead of dose. Data for killing *Giardia* in very cold water (5°C [41°F]) with both chlorine and iodine indicate that contact time must be prolonged three to four times, not merely doubled, to achieve high levels of inactivation.[73,92] If feasible, raising the temperature by 10° to 20°C (18° to 36°F) allows a lower dose of halogen and more reliable disinfection at a given dose.

Susceptibility of Microorganisms

The final variable is the target microorganism. Sensitivity to halogen is determined by the diffusion barrier of the cell wall or capsule and the relative susceptibility of proteins and cellular respiration to denaturation and oxidation.[40,146] Organisms, in order of increasing resistance to halogen disinfection, are enteric (vegetative) bacteria, viruses, protozoan cysts, bacterial spores, and parasitic ova (Tables 61-10 and 61-11); for example, *E. histolytica* cysts are 160 times as resistant as *E. coli* and 9 times as resistant as hardier enteroviruses to chlorine (hypochlorous acid [HOCl]). Virucidal residuals of I_2 and HOCl are 5 to 70 times higher than bactericidal residuals.[40,146] Relative resistance between organisms is similar for iodine and chlorine. The physical state of the microbes also determines their susceptibility. Microbes that are aggregated in clumps or embedded in other matter or organisms may be shielded from disinfectants.

Bacteria

All vegetative bacteria are extremely sensitive to halogens. Inactivation involves oxidation of enzymes on the cell membrane and does not require penetration.[244] Little modern work has focused on bacterial agents because they are more sensitive than viruses and cysts, and little difference is evident between the bacterial pathogens.[96] Although halogens were first used to disinfect water during cholera epidemics in 1850, recent cholera epidemics prompted review of data that reaffirmed the susceptibility of *V. cholerae* to low levels of chlorine and iodine.[52] *Campylobacter* has susceptibility similar to that of other enteric pathogens.[20]

Bacterial spores, such as *Bacillus anthracis,* are relatively resistant to halogens, but with chlorine, spores are not much more resistant than are *Giardia* cysts.[9,244] Quantitative data are not available for iodine solutions, but iodine does kill spores. Fortunately, sporulating bacteria do not normally cause waterborne enteric disease.[94]

Viruses

Enteroviruses are more resistant than are enteric bacteria,[146] but they constitute such a large and diverse group of organisms that generalization is especially difficult.[41,118,230] Most studies have used poliovirus, a phage virus, or coxsackievirus. The mechanism of action for halogen inactivation of viruses has not been resolved. It is not clear whether the oxidant injures protein on the shell, a process similar to bacterial inactivation,[26] or penetrates the protein capsid by chemical transformation and then attacks the nucleic acid core, as in cyst inactivation.[244]

Most viruses tested against chlorine have shown resistance 10 times greater than that of enteric bacteria, but inactivation is still achieved rapidly (0.3 to 4.5 minutes) with low levels (0.5 mg/L) of chlorine.[63,234] Current data suggest that HAV is not significantly more resistant than other enteric viruses.[85,163,211,224] In one test using iodine tablets, HAV was inactivated under difficult conditions more readily than poliovirus or echovirus.[210] Norovirus may be more resistant to chlorine

TABLE 61-10. Disinfection Data for Chlorine

HALOGEN*	ORGANISM	CONCENTRATION (mg/L)	TIME (min)	pH	TEMP	DISINFECTION CONSTANT (Ct)	REFERENCE
HOCl	*Escherichia coli*	0.1	0.16	6.0	5°C (41°F)	0.16	White, 1992[210]
FAC	*Campylobacter*	0.3	0.5	6.0–8.0	25°C (77°F)	0.15	Blaser, 1986[30]
FRC	20 enteric virus	0.5	60	7.8	2°C (36°F)	30	Briton, 1980[25]
Free Cl	6 enteric viruses	0.5	4.5	6.0–8.0	5°C (41°F)	2.5	Engelbrecht, 1980[66]
FRC	Hepatitis A virus	0.5	1	6.0	25°C (77°F)	0.5	Grabow, 1983[84]
Free Cl	Hepatitis A virus	0.5	5	6.0	5°C (41°F)	2.5†	Sobsey, 1975[181]
HOCl	Amebic cysts	3.5	10		25°C (77°F)	35	Chang, 1970[44]
FRC	Amebic cysts	3.0	10	7.0	30°C (86°F)	30	Stringer, 1970[186]
Free Cl	*Giardia* cysts	2.5	60	6.0–8.0	5°C (41°F)	150	Rice, 1982[182]
Free Cl	*G. lamblia* cysts	0.85	90	8.0	2–3°C (36–37°F)	77	Wallis, 1988[240]
Free Cl	*G. muris* cysts	3.05	50	7.0	5°C (41°F)	153	Rubin, 1989[196]
Free Cl	*G. muris* cysts	5.87	25	7.0	5°C (41°F)	139	Rubin, 1989[196]
Free Cl	*Giardia*			6.0	0.5°C (32°F)	170	Hibler, 1987[89]
Free Cl	*Giardia*			6.0	5°C (41°F)	120	Hibler, 1987[89]
Free Cl	*Cryptosporidium*	80	90			7200	Korich, 1990[354]
FRC	Schistosome cercariae	1.0	30	7.0	28°C (82°F)	30	World Health Organization, 1981[217]
Free Cl	Nematodes	2–3	120			(not lethal)	National Academy of Sciences, 1980[131]
Free Cl	Nematodes	95–100	30			(95% lethal)	National Academy of Sciences, 1980[131]
FRC	*Ascaris* eggs	200	20	5.0	37°C (99°F)	2000	Krishnaswami, 1968[112]

HOCl, hypochlorous acid; FAC, free active chlorine; Free Cl, free chlorine; FRC, free residual chlorine.
*These represent nearly equivalent measurements of the residual concentration of active chlorine disinfectant compounds.
†Four log reduction. Most experiments use 2 to 3 log (99% to 99.9%) reduction as end point.

TABLE 61-11. Disinfection Data for Iodine

HALOGEN*	ORGANISM	CONCENTRATION (mg/L)	TIME (min)	pH	TEMP (°C)	DISINFECTION CONSTANT (Ct)	REFERENCE
FRI	*Escherichia coli*	1.3	1	6.0–7.0	2–5°C (36–41°F)	1.3	National Academy of Sciences, 1980[131]
I_2	Amebic cysts	3.5	10		25°C (77°F)	35	Chang, 1970[44]
I_2	Amebic cysts	6.0	5		25°C (77°F)	30	Chang, 1970[44]
I_2	Amebic cysts	12.5	2		25°C (77°F)	25	Chang, 1970[44]
FRI	Poliovirus 1	1.25	39	6.0	25°C (77°F)	49	Berg, 1964[13]
FRI	Poliovirus 1	12.7	5	6.0	25°C (77°F)	63	Berg, 1964[13]
I_2	Poliovirus 1	1	6	7.0	18°C (64°F)	6	Berg, 1964[13]
I_2	Coxsackie virus	0.5	30	7.0	5°C (41°F)	15	Berg, 1964[13]
I_2	Amebic cysts	8	10	4.0–8.0	23°C (73°F)	80	Chang, 1953[46]
I_2	Bacteria, viruses	8	20		0–5°C (32–41°F)	160	Chang, 1953[46]
FRI	*Giardia* cysts	4	15	5.0	30°C (86°F)	60†	Fraker, 1992[73]
FRI	*Giardia* cysts	4	45	5.0	15°C (59°F)	170†	Fraker, 1992[73]
FRI	*Giardia* cysts	4	120	5.0	5°C (41°F)	480†	Fraker, 1992[73]

*FRI (free residual iodine) and I_2 (elemental iodine) are nearly equivalent measurements of the residual concentration of active iodine disinfectant compounds.
†100% kill; viability tested only at 15, 30, 45, 60, and 120 minutes.

than several other viruses, which may account for its importance in water-borne outbreaks.[111] Powers and colleagues[167,169] found that poliovirus was more slowly inactivated than rotavirus and *Giardia muris* by both chlorine and iodine, but this is inconsistent with other data. Clumping and association of viruses with cells and particulate matter are thought to be significant factors affecting viral disinfection, causing departure from first-order kinetics.[63,210,234] Cell-associated HAV was 10 times more resistant than dispersed HAV.

Cysts and Parasites

Protozoal cysts are considerably more resistant than are enteric bacteria and significantly more resistant than enteric viruses, probably because of the cyst's physiologically inactive outer

shell, which the disinfectant must penetrate to be effective.[40,244] Early data exist for *E. histolytica*, but recent work on *G. lamblia* indicates similar sensitivity to both iodine and chlorine.[105] Higher pH and lower temperature decrease the effectiveness of halogens on *Giardia*.[91,101,210] Review literature frequently attributes exaggerated resistance of *Giardia* to halogens.[95] Jarrol and colleagues[103,104] tested two chlorine methods and four iodine methods for effectiveness against *Giardia* cysts. They found all methods effective in warm water, but only two methods destroyed all cysts in cold water in recommended doses. Higher doses or longer contact times would make all these methods effective. Halogens can be used in the field to inactivate *Giardia* cysts (see Figure 61-3). However, longer contact time is required in cold and dirty water.[80]

Cryptosporidium oocysts differ greatly from other protozoan cysts and are highly resistant to halogens. The Ct constant for *Cryptosporidium* in warm water with chlorine has been estimated to be 9600.[33] Other data demonstrated 90% inactivation with 80 ppm of chlorine after 90 minutes, 14 times more resistant that *Giardia* cysts.[113] The current recommendation for decontaminating chlorinated swimming pools is to raise the free available chlorine concentration to 20 mg/L for 9 hours (Ct 10,800).[27] From 65% to 80% of *Cryptosporidium* oocysts were inactivated after 4 hours by two iodine tablets in "general case" water.[80] This implies that 3-log inactivation could have been achieved after 3 to 4 more hours. Although halogens can achieve disinfection of *Cryptosporidium* in the field, this is not practical.[56,190,201,244] The resistance of *Cyclospora* and microsporidia is not well studied, but the oocysts are similar to *Cryptosporidium* and thus may resemble this protozoa more than *Giardia*. Both *Cryptosporidium* and *Giardia* are susceptible to chlorine dioxide.[46,113]

Schistosome cercariae are susceptible to low concentrations of chlorine.[245] Limited data on parasitic helminth larvae and ova indicate such high levels of resistance that chemical disinfection is not useful.[115,146,203] However, these are not common waterborne pathogens and can be readily removed or destroyed by heat, filtration, or coagulation-flocculation.

Disinfection Constant

The best comparison of disinfection power is the disinfection constant (Ct). Disparate results may be caused by lack of standardized experimental conditions of pH, temperature, chemical species of halogen, and species of microorganism or by different techniques for concentrating, counting, and determining viability of organisms.[96,146] The latter is especially a problem for cysts and viruses, which cannot be cultured easily.[198] The end point for disinfection effectiveness is now becoming standardized by the EPA guidelines, but most older studies used 99.9% for all organisms, with some using 99% or 99.99%. Differences between laboratory and field conditions also make extrapolation from data to practice inaccurate and suggest the need for a safety factor in the field. Despite variation, Ct remains a useful and widely used concept; values provide a basis for comparing the effectiveness of different disinfectants for inactivation of specific microorganisms.[96] To use halogens for disinfection, a consensus organism (the most resistant target) determines the Ct.[96,118,244] For wilderness water, this has been protozoan cysts. The resistance of *Cryptosporidium* will not raise the threshold for halogen use; rather, it will force an alternative or a combination of methods to ensure removal and inactivation of all pathogens.

CHLORINE

Chlorine has been used as a disinfectant for 200 years. Hypochlorite was first used for water disinfection in 1854 during cholera epidemics in London and was first used continuously for water treatment in Belgium in 1902. It is currently the preferred means of municipal water disinfection worldwide and the preference of the Centers for Disease Control and Prevention (CDC) and WHO for individual household disinfection of drinking water, so extensive data support its use[244] (see Table 61-10).

Chemistry

Chlorine reacts in water to form the following compounds[62,244]:

$$Cl_2 + H_2O \rightarrow HOCl + H^+ + Cl^-$$
$$HOCL \rightarrow OCl^- + H^+$$

At neutral pH, negligible amounts of diatomic chlorine are present. The major disinfectant is hypochlorous acid (HOCl), which penetrates cell and cyst walls easily. Dissociation of HOCl to the much weaker disinfectant hypochlorite (OCl^-) depends on temperature and pH. In pure water at pH 6.0, 97% of chlorine is HOCl; at pH 7.5, the HOCl/OCl^- ratio is 1:1; and above pH 7.5, OCl^- predominates.[244] The combination of these two compounds is defined as free available chlorine. Both calcium hypochlorite ($Ca[OCl]_2$) and sodium hypochlorite (NaOCl) readily dissociate in water, allowing the same equilibrium to form as when elemental chlorine is used.[118,244] Chloride ion (Cl^-, NaCl, or $CaCl_2$) is germicidally inactive. In addition, chlorine readily reacts with ammonia to form monochloramines (NH_2Cl), dichloramines, or trichloramines, referred to as *combined chlorine*. In field disinfection, these compounds are not considered, and only free residual chlorine should be measured. However, chloramines have weak disinfecting power and are calculated as a disinfectant in municipal sewage plants.[96,144,146,244] At doses of a few mg/L and contact times of about 30 minutes, free chlorine generally inactivates >99.99% of enteric bacteria and viruses.[212]

Toxicity

Acute toxicity to chlorine is limited; the main danger is irritation and corrosion of mucous membranes if concentrated solutions (e.g., household bleach) are ingested. Numerous cases have been reported of short-term ingestion of very high residuals (50 to 90 ppm) in drinking water; one military study used 32 ppm for several months without adverse effects.[244] Animal studies using long-term chlorination of drinking water at 100 to 200 ppm have not shown toxic effects.[146]

Sodium hypochlorite is not carcinogenic; however, reactions of chlorine with certain organic contaminants yield chlorinated hydrocarbons, chloroform, and other trihalomethanes, which are considered carcinogenic.[146,230] Public health regulations limit residual chlorine in public systems to decrease ingestion of trihalomethane. The concern is now fueled more by public fears than by scientific conclusion.[244] The risk of death from infectious diseases if disinfection is not used is far greater than any risk from chlorine disinfection byproducts.[184,230] These compounds are not likely to form in clean wilderness surface water, because the organic precursors are not present.

TABLE 61-12. Water Disinfection Techniques and Halogen Doses

	ADD TO 1 L OR QUART OF WATER	
IODINATION TECHNIQUES	AMOUNT FOR 4 ppm	AMOUNT FOR 8 ppm
Iodine tabs Tetraglycine hydroperiodide EDWGT Potable Aqua Globaline	1/2 tab	1 tab
2% Iodine solution (tincture)*	0.2 mL or 5 gtts	0.4 mL or 10 gtts
10% Povidone-iodine solution*†	0.35 mL or 8 gtts	0.70 mL or 16 gtts
Saturated solution: iodine crystals in water	13 mL	26 mL
Saturated solution: iodine crystals in alcohol	0.1 mL	0.2 mL
CHLORINATION TECHNIQUES	AMOUNT FOR 5 ppm	AMOUNT FOR 10 ppm
Sodium hypochlorite (household bleach 5%)†	0.1 mL or 2 gtts	0.2 mL or 4 gtts
Calcium hypochlorite (Redi Chlor [1/10 gm tab])		1/4 tab/2 quarts
Sodium dichloroisocyanurate (AquaClear)		1 tab (8.5 mg NaDCC)
Chlorine plus flocculating agent (Chlor-Floc)		1 tab

EDWGT, emergency drinking water germicidal tablet; gtts, drops.
*Measure with dropper (1drop = 0.05 mL) or tuberculin syringe.
†Povidone-iodine solutions release free iodine in levels adequate for disinfection, but scant data are available.

TABLE 61-13. Recommendations for Contact Time with Halogenations in the Field

	CONTACT TIME IN MINUTES AT VARIOUS WATER TEMPERATURES		
CONCENTRATION OF HALOGEN	5°C (41°F)	15°C (59°F)	30°C (86°F)
2 ppm	240	180	60
4 ppm	180	60	45
8 ppm	60	30	15

Note: Data indicate that very cold water requires prolonged contact time with iodine or chlorine to kill *Giardia* cysts. These contact times have been extended from the usual recommendations in cold water to account for this and for the uncertainty of residual concentration.

Products and Techniques for Chlorination

Free chlorine is the most widely available and affordable means of chemical water disinfectant.[212] For household or field water treatment, free chlorine can be obtained in liquid, granular, and tablet forms or generated from electrolysis of salt (Tables 61-12 and 61-13 and Appendix B). Tablets have the advantage of easy administration and can be salvaged if the container breaks. However, they lose effectiveness with exposure to heat, air, or moisture. Although no significant loss of potency results from opening a glass bottle intermittently over weeks, activity is rapidly lost after a few days of continuous exposure to air with high heat and humidity. To extend shelf-life, many tablets are individually wrapped in foil.

Superchlorination-Dechlorination

The process of superchlorination-dechlorination with different reagents is used in some large-scale disinfection plants to avoid long contact times and to remove tastes and smells. High doses of chlorine remove or oxidize hydrogen sulfide and some other chemical contaminants that contribute to poor taste and odor. Chlorine bleaches organic matter, making water sparkling blue, as in swimming pools.[244] This method of chlorination can be readily adapted to field use. High doses of chlorine are added to the water in the form of calcium hypochlorite crystals to achieve concentrations of 30 to 200 ppm of free chlorine. These extremely high levels are above the margin of safety for field conditions and rapidly kill all bacteria, viruses, and protozoa and could kill *Cryptosporidium* with overnight contact times. After at least 10 to 15 minutes, several drops of 30% hydrogen peroxide solution are added. This reduces hypochlorite to chloride, forming calcium chloride (a common food additive), which remains in solution, as follows:

$$Ca(OCl)_2 + 2H_2O \rightarrow 2HOCl + Ca^{++}(OH^-)_2$$
$$Ca(OCl)_2 + 2H_2O_2 \rightarrow CaCl_2 + 2H_2O + 2O_2$$

Excess hydrogen peroxide reacts with water to form oxygen and water. Chloride has no taste or smell. Hydrogen peroxide is also a weak disinfectant,[253] although not in common use.

The minor disadvantage of a two-step process is offset by excellent taste. Measurements to titrate peroxide to the esti-

mated amount of chlorine do not need to be exact, but some experience is needed to balance the two and achieve optimal results. This is a good technique for highly polluted or cloudy water and for disinfecting large quantities. It is the best technique for storing water on boats or for emergency use. A high level of chlorine prevents growth of algae or bacteria during storage; water is then dechlorinated in needed quantities when ready to use.

The two reagents must be kept tightly sealed to maintain potency of the reagents. Properly stored, calcium hypochlorite loses only 3% to 5% of available chlorine per year. Thirty-percent hydrogen peroxide is corrosive and burns skin, so it should be used cautiously.

A commercial formulation, The Sanitizer, is no longer available; however, the ingredients can be easily obtained and packaged in small Nalgene bottles.

IODINE

Iodine has been used as a topical and water disinfectant since the beginning of the 20th century.[118] Iodine is effective in low concentrations for killing bacteria, viruses, and cysts, and in higher concentration against fungi and even bacterial spores, but it is a poor algicide[42,84,146] (see Table 61-11 and Figure 61-3). Iodine has been used successfully in low concentrations for continuous water disinfection of small communities.[112] Despite several advantages over chlorine disinfection, it has not gained general acceptance because of concern for its physiologic activity.

Chemistry

Iodine is the only halogen that is a solid at room temperature. Of the halogens, it has the highest atomic weight, lowest oxidation potential, and lowest water solubility. Its disinfectant activity in water is quite complex because of formation of various chemical intermediates with variable germicidal efficiencies. Seven different ions or molecules are present in pure aqueous iodine solutions, but only elemental (diatomic) iodine (I_2) and hypoiodous acid (HOI) play major roles as germicides. Diatomic iodine reacts in water to form the following compounds[42,84]:

$$I_2 + H_2O \rightarrow HOI + I^- + H^+$$

I_2 is two to three times as cysticidal and six times as sporicidal as HOI, because it more easily diffuses through the cyst wall. Conversely, HOI is 40 times as virucidal and three to four times as bactericidal as I_2, in that inactivation of organisms depends directly on oxidation potential, without involving cell wall diffusion.[40] Their relative concentrations are determined by pH and concentration of iodine in solution.[42] At pH 7.0 and 0.5 ppm of iodine, the concentrations of I_2 and HOI are approximately equal, resulting in a broad spectrum of germicidal action. At pH 5.0 to 6.0, most of the iodine is present as I_2, whereas at pH 8.0, 12% is present as I_2 and 88% as HOI. At higher concentrations of iodine, more HOI is present. Under field conditions, I_2 is the major disinfectant for which doses are calculated.[42]

Other chemical species, including triiodide (I_3^-), iodate (HIO_3), and iodide (I^-), form under certain conditions but play no role in water disinfection.[42,55] Iodide is important because it readily forms when reducing substances are added to iodine solution. Iodide ion is without any effect for water disinfection and it has no taste or color, but it is still physiologically active.

Toxicity

The main disadvantage of iodine is its physiologic activity, with effects on thyroid function, potential toxicity, and allergenicity.[159] Acute toxic responses generally result from intentional overdoses of iodine, with corrosive effects in the gastrointestinal tract leading to hemorrhagic gastritis. Mean lethal dose is probably about 2 to 4 g of free iodine or 1 to 2 ounces of strong tincture.[71] Toxicity is limited by rapid conversion of iodine to iodide by food (especially starch) in the stomach and early reflex vomiting. Iodide is absorbed into the bloodstream but has minimal toxicity (it is used widely for radiographic imaging).

Sensitivity reactions, including rashes and acne, may occur with usual supplementation levels of iodine. Given the physiologic necessity of iodine, it is not clear why some people react to certain forms of the substance, such as iodized salt. As with other sensitivity reactions, these may occur with very low doses. Acute allergy to iodide is rare and manifests as individual hypersensitivity, such as angioneurotic and laryngeal edema.[159]

Chronic iodide poisoning, or iodism, occurs after prolonged ingestion of sufficiently high doses, but marked individual variation is seen. Symptoms simulate upper respiratory illness, with irritation of mucous membranes, mucus production, and cough.

Iodine is an essential element for normal thyroid function and health in small amounts of 100 to 300 µg/day, but excess amounts can result in thyroid dysfunction. Maximum safe level and duration of iodine ingestion are not clearly defined, making it difficult to provide recommendations for prolonged use in water treatment.

Thyroid Effects of Excess Iodine Ingestion

Most persons can tolerate high doses of iodine without development of thyroid abnormalities,[24] because the thyroid gland has an autoregulatory mechanism that effectively manages excessive iodine intake. Initially, excess iodine suppresses production of thyroid hormone, but production usually returns to normal in a few days.

Iodine-induced hyperthyroidism can result from iodine ingestion by persons with underlying thyroid disease or when iodine is given to persons with prior iodine deficiency.[24,195] During the worldwide campaign to eliminate endemic goiter and cretinism, 1% to 2% of residents developed hyperthyroidism from small amounts of dietary iodine supplementation. Groups at higher risk were elderly persons, Graves' disease patients (especially after antithyroid therapy), and patients taking pharmacologic sources of iodine. Hyperthyroidism has been reported from iodine use as a water disinfectant in two travelers. Both were from iodine-sufficient areas and had antithyroid antibodies, suggesting underlying thyroiditis; one had a mother and sister with Hashimoto's thyroiditis.[123]

Iodine-induced hypothyroidism or goiter is much more common from excessive iodine intake. Hypothyroidism is attributed to prolonged suppression of thyroid hormone production induced by excess iodine levels, but the mechanism through which iodide goiter is produced is not well understood. The incidence of goiter varies and does not correlate well with quantity of iodine or with the level of hypothyroidism. Goiters

were discovered among a group of Peace Corps volunteers in Africa and were linked epidemiologically to the use of iodine resin water filters.[102] Forty-four (46%) of the volunteers had enlarged thyroids, but 30 of these had normal thyroid function tests.

Iodine-induced hypothyroidism or goiter may occur with or without underlying thyroid disease but is more common in several groups[24,195,246]: (1) those with underlying thyroid problems, including prior treatment for Graves' disease or subtotal thyroidectomy; (2) fetuses and infants, from placental transfer of iodide from mothers treated with iodides; (3) persons with subclinical hypothyroidism, especially elderly persons, in whom the incidence is 5% to 10%; and (4) patients with excessive iodide from medications (formerly potassium iodide; currently amiodarone).

Neonatal goiter is especially worrisome because it can lead to asphyxia during birth or hypothyroidism with mental impairment. Daily intakes as small as 12 mg have been reported to produce congenital iodide goiters, but generally much higher doses are required.

Dose-Response or Threshold Level

It is unclear what percent of the population will respond adversely to excess iodine or what should be defined as excess intake. The reported incidences of goiter, hypothyroid effects, and hyperthyroid response vary so widely that they provide no clear dose limits.[159] These data and other controlled trials of high doses were reviewed by Backer. The use of iodine for decades in the military and civilian population without reports of associated clinical thyroid problems suggests that the risks are minimal and would be outweighed by the risk of enteric disease. However, biochemical assays showed that changes in thyroid function tests were common with excess iodine intake, but usually remained within the range of normal values. Even when outside the normal range, the changes in thyroid function remained subclinical. All changes reverted to normal within weeks to months without persistent thyroid disease.

Studying longer duration of ingestion, Freund[75] found minimal changes and no clinical problems when water with 1 mg/L of iodine was used to disinfect water at a prison for up to 3 years. Referring to the same project, Thomas and colleagues[222] reported that after 15 years of ongoing iodine use at 1 mg/L, iodinated water caused no decrease in serum concentrations of T_4 below normal values and no allergic reactions. Patients with prior thyroid disease had no recurrence with iodinated water; four patients with active hyperthyroidism were treated in standard fashion, and their condition remained well controlled despite the extra iodine intake. Also, 177 inmates gave birth to 181 full-term infants, and no neonatal goiters were detected.[200]

The military studied long-term toxic effects of iodine, adding sodium iodide to drinking water at a naval base for 6 months.[143] The estimated daily dose of iodine per person was 12 mg for the first 16 weeks and 19.2 mg for the next 10 weeks. No evidence of functional changes or damage in the thyroid gland, cardiovascular system, bone marrow, eyes, or kidneys was noted. No increase in skin diseases, no sensitization to iodine, and no impaired wound healing or resolution of infections was evident.

If subclinical thyroid disease or enlarged thyroid were noted with a history of excess iodine ingestion, most experts would first stop the iodine intake and follow thyroid function before treating hypothyroidism.

Recommendations

The tenth edition of the recommended dietary allowances (RDAs, 1989) set the allowable dose to 1 mg/day for children and up to 2 mg/day for adults (increased from 0.5 to 1.0 mg in the ninth edition, 1980), primarily based on the data from Freund and Thomas.[159]

Possible toxicity with intermediate- to long-term use of iodine and the question of iodide toxicity remain controversial. The EPA and the WHO, supported by the American Water Works Association (AWWA), have recommended iodine use for water disinfection only as an emergency measure for short periods of about 3 weeks.[242,256] However, this period of short use appears arbitrary.

Available data suggest the following:

1. High levels of iodine, such as those produced by recommended doses of iodine tablets, should be limited to periods of 1 month or less.
2. Iodine treatment that produces a low residual (1 mg/L or less) appears safe, even for long periods in people with normal thyroid function. This would require very low doses of iodine added to the water or an activated charcoal stage to remove residual iodine.
3. Persons planning to use iodine for a prolonged period should have the thyroid gland examined and thyroid function measured to ensure that a state of euthyroidism exists.

The following groups should not use iodine for water treatment because of their increased susceptibility to thyroid problems:

- Pregnant women
- Persons with known hypersensitivity to iodine
- Persons with a history of thyroid disease, even if controlled by medication
- Persons with a strong family history of thyroid disease (thyroiditis)
- Persons from areas with chronic dietary iodine deficiency

Products and Techniques for Iodination

Formulations

Several forms of iodine are available for field use (See Tables 61-12 to 61-15 for efficacy data and Appendix B for details on commercial products, including tablets and crystalline iodine).

Iodine Solutions

Iodine solutions commercially sold as topical disinfectants are inexpensive and can be measured accurately with a dropper but are staining and corrosive if spilled. These contain iodine, potassium or sodium iodide in water, ethyl alcohol, or glycerol (Table 61-14). Iodide improves stability and solubility but has no ger-

TABLE 61-14. Iodine Solutions

PREPARATION	IODINE (%)	IODIDE (%)	TYPE OF SOLUTION
Iodine topical solution	2.0	2.4 (sodium)	Aqueous
Lugol's solution	5.0	10.0 (potassium)	Aqueous
Iodine tincture	2.0	2.4 (sodium)	Aqueous-ethanol
Strong iodine solution	7.0	9.0 (potassium)	Ethanol (85%)

micidal activity and adds to the total amount of iodine ingested and absorbed into the body. "Decolorized" iodine solution contains only iodide and should not be used for water disinfection.

Iodophors are solutions in which diatomic iodine is bound to a neutral polymer of high molecular weight, giving the iodine greater solubility and stability with less toxicity and corrosive effect.[42,84] Povidone-iodine is a 1-vinyl-2-pyrrolidinone polymer with 9% to 12% available iodine. The iodophors are routinely used for topical disinfection, because they have less tissue toxicity than iodine solutions. Although they are not approved for water disinfection in the United States, they are used in other countries for this purpose.[10] According to the manufacturer, approval for this use in the United States was not pursued because the anticipated use did not justify the expense. Povidone is nontoxic and was used as a blood expander during World War II.

In aqueous solution, povidone-iodine provides a sustained-release reservoir of halogen; free iodine is released in water solution depending on the concentration (normally, 2 to 10 ppm is present in solution). In dilutions below 0.01%, povidone-iodine solution can be regarded as a simple aqueous solution of iodine.[84] One report found these compounds similar in germicidal efficiency to other iodine-iodide solutions.[42] Data indicate persistence of about 2 ppm of free iodine at a 1:10,000 dilution,[84] equivalent to 0.1 mL (2 drops) added to 1 L of water. Conflicting values for available iodine and free iodine in dilute solutions result from the complex chemistry of povidone-iodine.[14,97] Personal and anecdotal experience of others attest to its effectiveness in field use.[10]

Resins

Iodine resins have been widely used for water disinfection in individual or small systems and incorporated into many different filter designs available for field use. They provide advantages over chlorination systems by eliminating the need for chemical feed systems, residual monitoring, and contact time.[242] Iodine resins are considered demand disinfectants because they are minimally insoluble in water and little iodine is released into aqueous solution. However, when a microorganism comes into contact with the resin, iodine apparently transfers to the microorganism aided by electrostatic forces, binds to the wall or capsule, and penetrates and kills the organism.[130]

Iodine resins are engineered to produce low residuals in effluent water. Pentaiodide resin produces a constant 1 to 2 ppm concentration after initial use, whereas triiodide resin produces a residual iodine concentration of less than 0.20 ppm at equilibrium.[130] The concentration in the effluent of triiodide resin is temperature dependent. Concentrations less than 1.0 ppm were obtained with water at 42.2°C (108°F), but this increased to a total iodine content of 6 to 10 ppm at 71°C (160°F). After returning to room temperature, the iodine residual returned to nominal values.[130]

Measurable iodine is attached to bacteria and cysts after resin treatment, effectively exposing the organisms to high iodine concentrations. This allows reduced contact time compared with dilute iodine solutions.[70,130] Some contact time still appears necessary.[129] Fifty percent of *Giardia* cysts were viable 10 minutes after passage through a triiodine resin. Viable *Giardia* cysts could be recovered in 4°C (39.2°F) water 40 minutes after passage through an iodine resin.[129] A simple resin filter failed to pass the EPA protocol for "worst-case" water unless water was passed through the filter twice. The data implied that a holding (contact) time could have achieved the same results.[81] The Canadian Health Department, challenging an in-line triiodine resin with highly polluted water, also found that a 15-minute contact time was necessary for warm water and 30-minute contact time for cold water.[3,64] The EPA conducted tests of triiodide resin against *E. coli* but not against other organisms, for which it relied on independent testing. It concluded that the product depends on a 0.2 ppm residual and that additional testing would be necessary below this level.

Resins are chemically and physically stable during conditions of dry storage at room temperature. Aqueous suspensions or resins retain biocidal potential for 15 years. No alteration in activity was observed after dry storage for 1 month at 50°C (122°F).[130]

Resins have proved effective against bacteria, viruses, and cysts but not against *Cryptosporidium parvum* oocysts or bacterial spores.[130] When *Cryptosporidium* oocysts were passed through a triiodide resin column, most were retained in the resin column, probably by electrostatic attraction to the resin. Of those that passed through, only a small percentage were inactivated within 30 minutes by the iodine.[232] Despite the controversy regarding contact time, most of the testing done with iodine resins has shown high levels of effectiveness.

Iodine Resin Filters. Recently, several iodine resin products were withdrawn from the market when company testing showed that they failed to meet viral inactivation standards of 4-log reduction. The units had previously obtained EPA registration on the basis of successful testing contracted through a laboratory that does the majority of testing for the filter industry. It was not clear whether failure was related to inadequate contact with the resin or contact time with the iodine residual. Most designs incorporated two stages in addition to the iodine resin. A microfilter, generally 1 micron (μm), effectively removes *Cryptosporidium, Giardia,* and other halogen-resistant parasitic eggs or larva. Because iodine resins kill bacteria and viruses rapidly, no significant contact time is required for most water.[81] The addition of a third stage of activated charcoal removes dissolved residual; however, the importance of iodine residual for disinfection has not been determined.[129,226]

In conclusion, iodine resins are effective disinfectants that can be engineered into attractive field products, including use in the space shuttle and large-scale units for international disasters. They may prove useful for small communities in undeveloped and rural areas where chlorine disinfection is technically and economically unfeasible. However, companies recently have abandoned iodine resin–containing portable hand-pump filters. Only one drink-through bottle remains on the U.S. market. Other products may still be available outside the United States.

CHLORINE VS. IODINE

A large body of data proves that both iodine and chlorine are effective disinfectants with adequate concentrations and contact times (cold temperatures equate with slow disinfection time for both). Comparing effectiveness of chlorine and iodine is difficult because of the different ionic species and compounds that may exist under varying conditions.[94] Chlorine and iodine tablets have been directly compared under identical water test conditions and found to be similar in their biocidal activity in

TABLE 61-15. Data on Microcidal Efficacy of Iodine Tablets

HALOGEN	DOSE	FRC (mg/L)	TIME (min)	TEMPERATURE °C (°F)	ORGANISM	LOG REDUCTION	REFERENCE
Chlor-Floc	1 tab or 2 tabs	4–7	5	10–20 (50–68)	Bacteria	6	Powers, 1994[283]
		4–14	20	10–20 (50–68)	*Giardia muris*	3	
	1	4	5	10–20 (50–68)	Rotavirus	4	
	1	5	20	10–20 (50–68)	Poliovirus	2.5	
	1 tab	5	12	25 (77)	Poliovirus	2	
Globaline	2 tabs	12	20	Various	Bacteria	6	
	2	17	45	5 (41)	*G. muris*	3	
	2	14	20	5 (41)	Rotavirus	4	
	2	12	60	5 (41)	Poliovirus	3	
AquaPure	2 tabs	7–11	40	5 (41)			Powers, 1992[280]
	1 tab		30–40	15–25 (59–77)	Bacteria	6	
	2		20	15–25 (59–77)	Rotavirus	4	
	2		20	15–25 (59–77)	Polio virus	2	
	2		20	15–25 (59–77)	*G. muris*	2	
Globaline	2 tabs	10	60	15 (59)	*Giardia*	3	Powers, 1991[279]
	1		180	5 (41)	*Giardia*	3	
	2		120	5 (41)	*Giardia*	3	
Iodine tabs	1 or 2	8–16	60	5–25 (41–77)	Hepatitis A	4	Sobsey, 1991[213]
	1	8	60	5 (41)	Polio, echo	3	
	2	16	60	5 (41)	Polio, echo	4	

FRC, free residual chlorine.

most conditions using recommended dose and contact time[165] (see Tables 61-12, 61-13, and 61-15.) Contact times in Table 61-13 are extended from the previous recommendations for treatment in cold water to provide a margin of safety and to ensure high levels of cyst destruction. A few investigators have reported data suggesting ineffectiveness of common halogen preparations. Jarroll and colleagues[104,105] tested six methods of field disinfection and found that none achieved high levels of *Giardia* inactivation at the recommended dose and times. However, this failure simply reflected the need for longer contact times in cold water.[124] Ongerth and colleagues[156] tested seven chemical treatments for *Giardia* inactivation in clear and turbid water at 10° C (50° F). None achieved 99.9% reduction in 30 minutes. All iodine-based chemical methods were effective at 8 hours, but none of the chlorine preparations was effective, even after this extended time. Although these results after 30 minutes in cold water are to be expected, the 8-hour results do not conform with other experimental data on chlorine. Unfortunately, the authors did not test for residual halogen, although initial levels achieved should have been effective, and they did not test at regular time intervals to determine when the iodine methods had achieved the target reduction of organisms. Schlosser found that sodium hypochlorite tablets, sodium dichloroisocyanurate tablets, and iodine in ethanol used according to package instructions removed 2 to 3 log of bacteria in clear water, but less in turbid river water.[200] Again, this suggests the need for clarifying dirty water before halogen use and, if possible, providing extra contact time in any situation.

Iodine has several advantages over chlorine. Of the halogens, iodine has the lowest oxidation potential, reacts least readily with organic compounds, is least soluble, is least hydrolyzed by water, and is less affected by pH, all of which indicate that low iodine residuals should be more stable and persistent than corresponding concentrations of chlorine.[55,84,112,146]

Taste

Objectionable taste and smell are the major problems with acceptance of halogens. Most objectionable tastes in treated water are derived from dissolved minerals, such as sulfur, and from chlorine compounds, chloramines, and organic nitrogen compounds, even at extremely low levels.

People are familiar with the taste of chlorine compounds; tap water usually contains 0.2 to 0.5 ppm of chlorine, swimming pools 1.5 to 3 ppm, and hot tubs 3 to 5 ppm. Most persons note a distinct taste at 5 ppm and a strong, unpleasant taste at 10 to 15 ppm.[188] Hypochlorous acid and chloride have no taste or odor.[244]

Elemental iodine at 1 mg/L is undetectable. Most persons can detect iodine solutions at 1.5 to 2 mg/L but do not find it objectionable.[18,55,76] Eight ppm of iodine produces a distinct taste and odor; however, tablets yielding these concentrations were preferred by military personnel over tincture of iodine in equivalent doses.[42,145] Iodide ion has no color or taste.

Taste tolerance or preference for iodine over chlorine is individual. Opposite preferences have been documented when direct comparisons are done.[154,169] Informal taste tests suggest that most persons prefer the taste of iodine to chlorine at concentrations typically used in the field. In addition, iodine forms fewer organic compounds that produce highly objectionable taste and smell.

Taste can be improved by several means (Box 61-10).

Minimal Dosage

The relationship between halogen concentration and time allows use of the minimum necessary dose, with a longer contact time (see Table 61-13).

Box 61-10. Improving the Taste of Halogens

- Decreased dose; increased contact time
- Clarification of cloudy water, which decreases amount of halogen needed
- Removal of halogen
- Use of granular activated carbon (GAC)
- Chemical reduction
  - Ascorbic acid
  - Sodium thiosulfate
  - Superchlorination-dechlorination
- Use of KDF (zinc-copper) brush or media
- Alternative techniques:
  - Heat
  - Filtration
  - Chlorine dioxide or mixed species (Miox)

Theoretically, doubling the contact time allows a 50% reduction of halogen dose at any level. Although this relationship holds true at the higher field doses of halogens, as the levels drop, the reaction departs from mathematical models, and the straight-line graph has a "tail" (see Figure 61-2). This departure from strict first-order kinetics and the uncertainty of halogen demand in field disinfection mean that a margin of safety must be incorporated into contact times at lower doses.

Of all standard iodine doses, iodine tablets yield the highest dose (8 mg/L with an intended contact time of 10 minutes in warm water). The tablets cannot be broken in half but can be added to 2 quarts instead of 1 quart to yield concentrations consistent with the other preparations. In recommended doses, the liquid preparations of iodine yield 4 mg/L. Because even clear surface water has some halogen demand, this dose of 4 mg/L should generally not be reduced. The exception would be for backing up tap water in developing countries, when the dose may routinely be cut in half for an added dose of 2 ppm with a few hours of contact time. *Giardia* cysts and viruses can be killed with doses of chlorine or iodine of 2 ppm or less (see Figure 61-3).[73,92,118] For chlorination methods that add 5 mg/L, adding half the amount to clean surface water should be adequate if the contact time is tripled. Even less could be used for tap water. None of these concentrations will destroy *Cryptosporidium* oocysts.

Temperature and organic matter in the water may be manipulated. Increasing the temperature of the water, especially when initially near 5° C (41° F), decreases the Ct constant (see Table 61-13 and Figure 61-3). Filtering water before adding halogen improves the reliability of a given halogen dose by decreasing halogen demand, allowing a lower dose of halogen.[146] Sedimentation or coagulation-flocculation cleans cloudy water and lowers the required halogen dosage considerably, in addition to removing many of the contaminants that contribute to objectionable taste.

Dehalogenation

Halogen can be removed from water after the required contact time. Activated charcoal removes iodine or chlorine, allowing standard or even high doses to be used without residual taste. The relative instability of chlorine in dilute solutions can be used to decrease taste over time. Chlorine residual in an open container decreases 1 mg/L in the first hour, then 0.2 mg/L in the next 5 to 8 hours, for a total of 2 to 2.5 mg/L in 24 hours. Ultraviolet light also depletes free chlorine.[244]

Alteration of Chemical Species (Reduction)

Several chemical means are available to reduce free iodine or chlorine to iodide or chloride that have no color, smell, or taste. These "ides" have no disinfection action, so the techniques should be used only after the required contact time. In superchlorination-dechlorination, hydrogen peroxide "dechlorinates" the water by forming calcium chloride. This reaction with hydrogen peroxide works best when calcium hypochlorite is used as a disinfectant. If bleach (sodium hypochlorite) is used, hydrogen peroxide reacts with chlorine in water to form hydrochloric acid in harmless amounts.

Two other chemicals that may be safely used with any form of chlorine or iodine are ascorbic acid (vitamin C) and sodium thiosulfate. Ascorbic acid is widely available in the crystalline or powder form. Grinding up tablets that have binders may cloud the water. Ascorbic acid is a common ingredient of flavored drink mixes, which accounts for their effectiveness in covering up the taste of halogens.[154,188]

Sodium thiosulfate similarly "neutralizes" iodine and chlorine. A few granules in 1 quart of iodinated water decolorizes and removes the taste of iodine by converting it to iodide. In reaction with chlorine, it forms hydrochloric acid, which is not harmful or detectable in such dilute concentration. Thiosulfate salts are inert in vivo and poorly absorbed from the gastrointestinal tract. Sodium thiosulfate is available at chemical supply stores.

Zinc-copper alloys act as catalysts to reduce free iodine and chlorine through an electrochemical reaction (see Copper and Zinc). They also remove or reduce dissolved metals like iron, as well as heavy metals such as lead, selenium, and mercury. One product that is no longer on the market incorporated such an alloy into the bristles of a small brush to be stirred in the water after halogen disinfection. It was effective but slow, which limited its use to small volumes of water: stirring for 1 minute removed 10 mg/L of chlorine from 250 mL of water.

MISCELLANEOUS DISINFECTANTS

Mixed Species Disinfection (Electrolysis)

Passing a current through a simple brine salt solution generates free available chlorine, as well as other "mixed species" disinfectants that have been demonstrated effective against bacteria, viruses, and bacterial spores.[197] The process is well described and can be used on both large and small scale. It is practical and economical enough to be useful in developing areas of the world. The exact composition of the solution is not well delineated because many of the compounds are relatively unstable. However, the resulting solution has greater disinfectant ability than a simple solution of sodium hypochlorite. It has even been demonstrated to inactivate *Cryptosporidium*, suggesting that chlorine dioxide is among the chemicals generated.[239] A new point-of-use commercial product is now available (Miox, marketed by MSR; see Appendices for more information).

Ozone

Ozone and chlorine dioxide are highly effective disinfectants widely used in municipal water treatment plants.[244] These are

the only disinfectants that have been demonstrated effective against *Cryptosporidium* in typical concentrations.[46,113,158]

Ozone is an unstable form of oxygen, with the chemical formula O_3. In solution, it decays to O_2, producing free hydroxyl radicals. Both ozone and the hydroxyl radicals are two of the most powerful oxidants used in water treatment.[16] Ozone is a colorless gas manufactured by passing air or oxygen through a high-voltage current discharge. The resulting ozone-rich gas is then dissolved in water. Clearly, this is not conducive to small, point-of-use generation, so consumers should have a healthy skepticism of techniques claiming to rely on ozone.

Chlorine Dioxide

Chlorine dioxide (ClO_2) has been used primarily as a bleaching agent to control taste and odor in municipal disinfection, but it has gained wider use for disinfection of both community and point-of-use drinking water supplies in developed countries. Chlorine dioxide is capable of inactivating most water-borne pathogens, including *Cryptosporidium parvum* oocysts, at practical doses and contact times. It is at least as effective a bactericide as chlorine, and in many cases it is superior. It is far superior as a virucide.[244] For point-of-use treatment of water, chlorine dioxide is produced on site from the reaction of sodium chlorite with acid.[16,209] For example:

$$5NaClO_2 + 4HCl \rightarrow 4ClO_2 + 5NaCl + 2H_2O$$

Chlorine dioxide is not as unstable as ozone, but it does not produce a lasting residual. Byproducts of chlorine dioxide are chlorite (ClO_2^-) and chlorate (ClO_3^-).

Chlorine dioxide, a potent biocide, has been used for many years to disinfect municipal water and in numerous other large-scale applications. It does not form chlorinated compounds in the presence of organics and is efficacious over a wide pH range. However, chlorine dioxide is a volatile gas that must be produced on-site and, until recently, the only practical method for its production required sophisticated chemical generation equipment. Therefore, significant capital and operating costs limited the benefits of chlorine dioxide to only large-scale applications. However, recently, new technology enables cost-effective and portable chlorine dioxide generation and distribution for use in an ever widening array of small-scale applications (Box 61-11), including MicroPur MP-1, AquaMira, and Miox (see Appendices).

Silver

Silver ion has bactericidal effects in low doses. The literature on antimicrobial effects of silver is confusing and contradictory.[98,146,244,248] Concentrations in water less than 100 parts per billion (ppb) are effective against enteric bacteria. The reaction follows first-order kinetics and is temperature dependent. Calcium, phosphates, and sulfides interfere significantly with silver disinfection. Organic chemicals, amines, and particulate or colloidal matter may also interfere, but no more than with chlorine.

Silver is physiologically active. Acute toxicity does not occur from small doses used in disinfection, but argyria, which is permanent discoloration of the skin and mucous membranes, may result from prolonged use. For this reason, a maximum limit of 50 ppb of silver ion in potable water is recommended. At this concentration, disinfection requires several hours.

Box 61-11. Chlorine Dioxide

Advantages	Disadvantages
Effective against all microorganisms, including *Cryptosporidium*	Volatile, so do not expose tablets to air and use generated solutions rapidly
Low doses have no taste or color	No persistent residual, so does not prevent recontamination during storage
Portable device now available for individual and small group field use and is simple to use	Sensitive to sunlight; keep bottle shaded or in pack during treatment
More potent than equivalent doses of chlorine	
Less affected by nitrogenous wastes	

Relative susceptibility of microorganisms to chlorine dioxide: bacteria > viruses > protozoa.

Experimental results indicate 18% survival of *E. coli* at 3 hours at 40 µg/L. *Salmonella typhi* was reduced more than 5 log at 50 µg/L with a 1-hour exposure; poliovirus was not reduced at 50 µg/L with a 1-hour exposure.[11]

Water disinfection systems using silver have been devised for spacecraft, swimming pools, and other settings.[244] The advantage is absence of taste, odor, and color. Persistence of residual silver concentration allows reliable storage of disinfected water. Silver can be supplied through a silver nitrate solution, desorption from silver-coated materials, or electrolysis. When coated on surfaces, silver acts as a constant-release disinfectant that produces aqueous silver ion concentrations of 0.006 to 0.5 ppm, which are sufficient to disinfect drinking water.[130] Because of this attractive feature, silver-based devices are being designed and tested in developing countries. In Pakistan, a nylon bag with silver-coated sand was designed to be placed in earthenware pitchers that store water. Silver incorporated into alum is also being tested in India.[43]

Filters and granular charcoal media are sometimes coated with silver to prevent bacterial growth on the surface, but this does not maintain sterility. A slow, selective action against total coliform count is noted, but none against total bacterial count. Long-term use might overcome any bacteriostatic action initially shown.[78] In an EPA study, effluent populations from the silver-containing units were about as large as those from the units without silver.[11] Bacteria can develop resistance to silver ions through generation of silver reductase.

Large-scale use of silver for water disinfection has been limited by cost, difficulty controlling and measuring silver content, and physiologic effects. Short-term field use is limited by its marked tendency to adsorb onto the surface of any container (resulting in unreliable concentrations) and interference by several common substances. Data on silver for disinfection of viruses and cysts indicate limited effect, even at high doses.[40,146]

The use of silver as a drinking water disinfectant has been much more popular in Europe where silver tablets (MicroPur) are sold widely for field water disinfection. They have not been

approved by the EPA for this purpose in the United States, but they were approved as a water preservative to prevent bacterial growth in previously treated and stored water. MicroPur Forte tablets (Katadyn) release free chlorine for disinfection and silver for prolonged persistence of antimicrobial activity.

Potassium Permanganate

Potassium permanganate is a strong oxidizing agent with some disinfectant properties. It was used extensively before hypochlorites as a drinking water disinfectant.[145] It is still used for this purpose and also for washing fruits and vegetables in parts of the world. It is used in municipal disinfection to control taste and odor and is usually employed in a 1% to 5% solution for disinfection.

Bacterial inactivation can be achieved with moderate concentrations and contact times (45 minutes at 2 mg/L, 15 minutes at 8 mg/L). A 1:5000 (0.5%) solution controlled *V. cholerae* and *S. typhi* contamination of fruits and vegetables. The virucidal action has been tested, but without titrations of virus that remained after various periods of contact time, so the rate of action is not known. In most instances, however, a 1:10,000 solution destroyed the infectivity of virus suspensions in 30 minutes at room temperature; 30 mg/L was effective in inactivating HAV within 15 minutes.[224] Although potassium permanganate clearly has disinfectant action, it cannot be recommended for field use, because quantitative data are not available for viruses and no data are available for protozoan cysts, despite the chemical's frequent use in some parts of the world. Packets of 1 g to be added to 1 L of water are sold in some countries. A French military guide from 1940 instructed users: "To sterilize water, use a solution of 1 gram of $KMnO_4$ for 100 grams of water. Add this solution drop by drop to the water to sterilize until the water becomes pink. The operation is considered sufficient if the water remains pink for half an hour."[44] The solutions are deep pink to purple and stain surfaces. The chemical leaves a pink to brown color in water at concentrations above 0.05 mg/L. Small deposits of brown oxides settle to the bottom of the water container. A few drops of alcohol will cause this residual color to disappear.

Hydrogen Peroxide

Hydrogen peroxide is a strong oxidizing agent but a weak disinfectant.[21,146,253] Small doses (1 mL of 3% H_2O_2 in 1 L water) are effective for inactivating bacteria within minutes to hours, depending on the level of contamination. One million colony-forming units/mL of seven bacterial strains were killed overnight, with 80% kill in 1 hour. Viruses require extremely high doses and longer contact times. Although information is lacking on the effect of hydrogen peroxide on protozoa, it is a promising sporicidal agent in high (10% to 25%) concentrations.

Hydrogen peroxide was popular as an antiseptic and disinfectant in the late 19th century and remains popular today as a wound cleanser; for odor control in sewage, sludges, and landfill leachates; and for many other applications. It is considered safe enough for use in foods, yielding the innocuous end products oxygen and water. It is considered nature's disinfectant, because it is naturally present in milk and honey, helping to prevent spoilage. Solutions lose potency in time, but stabilizers can be added to prevent decomposition.[21]

Although hydrogen peroxide can sterilize water, lack of data for protozoal cysts and quantitative data for dilute solutions prevents it from being useful as a field water disinfectant.

Box 61-12. Ultraviolet Irradiation

Advantages	Disadvantages
Effective against all microorganisms	Requires clear water
Imparts no taste	Does not improve water aesthetics
Portable device now available for individual and small group field use and is simple to use	Does not prevent recontamination during storage
Available from sunlight	Expensive
	Require power source
	Requires direct sunlight, prolonged exposure; dose low and uncontrolled

Relative susceptibility of microorganisms to ultraviolet: protozoa > bacteria > viruses.

Ultraviolet Light

The germicidal effect of ultraviolet (UV) light is the result of action on the nucleic acids of bacteria and depends on light intensity and exposure time. UV lamp disinfection systems are widely used to disinfect drinking water at the community and household level (Box 61-12). In sufficient doses, all water-borne enteric pathogens are inactivated by UV radiation. Bacteria and protozoan parasites require lower doses than enteric viruses and bacterial spores. *Giardia* and *Cryptosporidium* are susceptible to practical doses of UV and may be more sensitive because of their relatively large size.[125] UV treatment does not require chemicals and does not affect the taste of the water. It works rapidly, and an overdose to the water presents no danger; in fact, it is a safety factor. UV irradiation with lamps requires a power source and is costly. UV light has no residual disinfection power; water may become recontaminated, or regrowth of bacteria may occur.[65] Particulate matter can shield microorganisms from UV rays. A portable field unit called Hydro Photon is now available (see Appendix A).

Solar Irradiation

There is now strong evidence that UV irradiation by sunlight in UV-A range can substantially improve the microbiologic quality of water and reduce diarrheal illness in developing countries. The technique was practiced in India before 2000 BC. Recent work has confirmed efficacy and optimal procedures of the solar disinfection (SODIS) technique.[48,152,209] Transparent bottles (e.g., clear plastic beverage bottles), preferably lying on a dark surface, are exposed to sunlight for a minimum of 4 hours, but some investigations demonstrate improved benefit from several sequential days. Multiple studies demonstrate reduction of enteric bacteria and viruses, and some data exist for reduction of bacterial spores.[134] Oxygenation induces greater reductions of bacteria, so agitation is recommended before solar treatment in bottles.

UV and thermal inactivation were strongly synergistic for solar disinfection of drinking water in transparent plastic bottles that was heavily contaminated with *E. coli* for temperatures above 45°C (113°F). Above 55°C (131°F), thermal inactivation is of primary importance.[133] Whereas thermal inactivation

TABLE 61-16. Summary of Field Water Disinfection Techniques

	BACTERIA	VIRUSES	*GIARDIA*/AMEBAE	*CRYPTOSPORIDIUM*	NEMATODES/CERCARIAE
Heat	+	+	+	+	+
Filtration	+	+/– *	+	+	+
Halogens	+	+	+	–	+/–†
Chlorine dioxide	+	+	+	+	+/–†

*Most filters make no claims for viruses. Reverse osmosis is effective. The General Ecology filtration system claims virus removal.
†Eggs are not very susceptible to halogens but have very low risk of waterborne transmission.

is effective in turbid water, UV effects are inhibited.[108] Use of a simple reflector or solar cooker can achieve temperatures of 65°C (149°F), which will pasteurize the water (see Heat Disinfection).

Where strong sunshine is available, solar disinfection of drinking water is an effective, low-cost method for improving water quality and may be of particular use in refugee camps and disaster areas.

Copper and Zinc

A copper and zinc alloy (KDF [kinetic degradation fluxion]) has electrochemical properties that can aid in water treatment. Its main actions are through its strong oxidation-reduction (redox) potential of 500 millivolts due to its propensity to exchange electrons with other substances. It is bacteriostatic with some bactericidal activity. Microorganisms are killed by the electrolytic field, and by formation of hydroxyl radicals and peroxide water molecules. Although KDF has been ruled a "pesticidal device" by the EPA and is used in industry to decrease bacteria levels and control bacterial growth, it should not be used as the sole means of water treatment. KDF is most often used to reduce or remove chlorine, hydrogen sulfide, and heavy metals from water. The redox reactions change contaminants into harmless components: chlorine into chloride, soluble ferrous cations into insoluble ferric hydroxide, and hydrogen sulfide into insoluble copper sulfide. Up to 98% of lead, mercury, nickel, chromium, and other dissolved metals are removed by KDF simply by bonding to the media. KDF controls the buildup of bacteria, algae, and fungi in GAC beds and carbon block filters, extending the life of carbon and improving its effectiveness. KDF can remove the taste of chlorine or iodine from treated water. KDF media can be manufactured as brushes with wire bristles, fine steel wool-like media, or granules.

KDF has been incorporated into a few portable field filters but has not yet gained widespread use and no portable products are currently targeting the outdoor market. Products that claim to be purifiers, with KDF destroying all microorganisms, should demonstrate rigorous testing to prove the claims.

Citrus

Citrus juice has biocidal properties. Lemon or lime juice has been shown to destroy *V. cholerae* at a concentration of 2% (equivalent of 2 tablespoons per liter of water) with a contact time of 30 minutes. A pH less than 3.9 is essential, which depends on the concentration of lemon juice and the initial pH of the water. Its activity is greatly reduced in alkaline water.[58] Lime juice also killed 99.9% of cholerae on cabbage and lettuce and inhibited growth of *V. cholera* in rice foods, suggesting that adding lime juice to water, beverages, and other foods can reduce disease risks.[209] More research is needed before this can be recommended as more than an ancillary or emergency measure. Commercial products using citrus cannot be recommended as primary means of water disinfection.

Traveler's Friend is a product marketed for water disinfection that contains citrus extract. Company-sponsored data are convincing for antibacterial and antiviral activity. However, the product was not tested against *Giardia* cysts. The active chemical disinfectant has not been identified, and a time-dose response has not been generated. Without better data, this product cannot be recommended.

PREFERRED TECHNIQUE

Field disinfection techniques and their effects on microorganisms are summarized in Table 61-16. The optimal technique for an individual or group depends on the number of persons to be served, space and weight available, quality of source water, personal taste preferences, and availability of fuel (Tables 61-17 and 61-18). The most effective technique may not always be available, but all methods will greatly reduce the load of microorganisms and reduce the risk of illness. For alpine camping with a high-quality source water, any of the primary techniques is adequate. The only limitation for halogens is *Cryptosporidium* cysts, but in high-quality pristine surface water the cysts are generally found in insufficient numbers to pose significant risk. Chlorine dioxide or mixed species techniques are currently the only one-step chemical processes available. Surface water, even if clear, in undeveloped countries where there is human and animal activity should be considered highly contaminated with enteric pathogens. Optimal protection requires either heat or a two-stage process of filtration and halogens. Currently, First Need and the Exstream iodine resin–containing water bottle are the only filters with demonstrated ability to meet requirements of a water purifier.

Water from cloudy low-elevation rivers, ponds, and lakes in developed or undeveloped countries that does not clear with sedimentation should be pretreated with coagulation-flocculation, then disinfected with heat or halogens. Filters can be used but will clog rapidly with silted or cloudy water. Even in the United States, water with agricultural runoff or sewage plant discharge from upstream towns or cities must be treated to remove *Cryptosporidium* and viruses. In addition, water receiving agricultural, industrial, or mining runoff may contain chemical contamination from pesticides, other chemicals, and heavy metals. A filter containing a charcoal element is the best method to remove most chemicals. Coagulation-flocculation or KDF resin will also remove some chemical contamination.

TABLE 61-17. Advantages and Disadvantages of Disinfection Techniques

	HEAT	FILTRATION	HALOGENS	CHLORINE DIOXIDE	TWO-STEP PROCESS	UV
Availability	Wood can be scarce	Many commercial choices	Many common and specific products	Several new products generate ClO_2	Filtration plus halogen, or clarification plus second stage	New portable commercial device; sunlight
Cost	Fuel and stove costs	Moderate expense	Cheap	Depends on method, generally inexpensive	Depends of choice of stages	Commercial device relatively expensive
Effectiveness	Can sterilize or pasteurize	Most filters not reliable for viruses	*Cryptosporidium* and some parasitic eggs are resistant	All organisms	Highly effective, should cover all organisms	All organisms
Optimal application	Clear water	Clear or slightly cloudy; turbid water clogs filters rapidly	Clear; need increased dose if cloudy	Clear water, but ClO_2 less affected by nitrogenous compounds	May be adapted to any source water	Requires clear water, small volumes
Taste	Does not change taste	Can improve taste, especially if charcoal stage	Tastes worse unless halogen is removed or "neutralized"	Unchanged, may leave some chlorine taste	Depends on sequence and choice of stages; generally improves	Unchanged
Time	Boiling time (minutes)	Filtration time (minutes)	Contact time (minutes to hours)	Prolonged, if need to ensure *Cryptosporidium* disinfection	Combination of time for each stage	Minutes
Other considerations	Fuel is heavy and bulky	Adds weight and space; requires maintenance to keep adequate flow	Works well for large quantities and for water storage. Some understanding of principles is optimal; damaging if spills or container breaks	More experience and testing would be reassuring; likely to replace iodine for field use.	More rational to use halogens first if filter has charcoal stage; C-F is best means of cleaning very turbid water, then followed by halogen, filtration or heat	Sunlight currently for emergency situations or no other methods available; commercial product good for high-quality source water, small group use

C-F, coagulation-flocculation.

TABLE 61-18. Choice of Method for Various Types of Source Water

			DEVELOPED OR DEVELOPING COUNTRY	
SOURCE WATER	"PRISTINE" WILDERNESS WATER WITH LITTLE HUMAN OR DOMESTIC ANIMAL ACTIVITY	TAP WATER IN DEVELOPING COUNTRY	CLEAR SURFACE WATER NEAR HUMAN AND ANIMAL ACTIVITY*	CLOUDY WATER
Primary concern	*Giardia*, enteric bacteria	Bacteria, *Giardia*, small numbers of viruses	All enteric pathogens, including *Cryptosporidium*	All enteric pathogens plus microorganisms
Effective methods	Any single step method[†]	Any single step method[b]	1) Heat 2) Filtration plus halogen (can be done in either order); iodine resin filters (see text) 3) Chlorine dioxide 4) Ultraviolet (commercial product, not sunlight)	CF followed by second step (heat, filtration or halogen)

CF, coagulation-flocculation.
*Includes agricultural run-off with cattle grazing or sewage treatment effluent from upstream villages or towns.
[†]Includes heat, filtration, halogens and chlorine dioxide, ultraviolet.

Halogens need to be used when water will be stored, such as on a boat, in a large camp, or for disaster relief. When only heat or filtration is used before storage, recontamination and bacterial growth can occur. Hypochlorite still has many advantages, including cost, ease of handling, and minimal volatilization in tightly covered containers.[140] A minimum residual of 3 to 5 mg/L should be maintained in the water. Superchlorination-dechlorination is especially useful in this situation because high levels of chlorination can be maintained for long periods, and when ready for use, the water can be poured into a smaller container and dechlorinated. Iodine works for short-term but not prolonged storage, because it is a poor algicide. Silver has been approved by the EPA for preservation of stored water.

Extensive research in the developing world has demonstrated that water is often recontaminated before use and that proper storage techniques can decrease this risk of contamination.[212,252] For prolonged storage, a tightly sealed container is best. For water access, narrow-mouth jars or containers with water spigots prevent contamination from repeated contact with hands or utensils.[207]

On oceangoing vessels where water must be desalinated during the voyage, only reverse-osmosis membrane filters are adequate. Halogen should then be added to the water in the storage tanks.

The preferred method of treatment for the military, when large-scale equipment can be brought to the site, is a reverse osmosis water purification unit (ROWPU), because it can produce high-quality water from a low-quality source. For smaller groups, the military relies mainly on monitored chlorine. Individual means include iodine tablets, chlor-floc tabs, and chlorine liquid bleach.[233]

PREVENTION AND SANITATION

Studies in developing countries have demonstrated a clear benefit in the reduction of diarrheal illness and other infections from safe drinking water, hygiene, and adequate sanitation.[100,127,137,172,173,180,212] While a benefit can be demonstrated for these interventions independently, the benefit is greater when all are applied together, especially with appropriate education.[43] Wilderness travelers essentially live in conditions similar to those in the developing world, without running water or sanitation. Unfortunately, many wilderness travelers confuse the importance of hygiene with the need to relax their concern about living on the ground.

A study of Appalachian trail hikers showed that water disinfection, routine hand washing, and proper cookware cleaning were all associated with decreased diarrhea.[22] A *Shigella* outbreak among river rafters on the Colorado River was investigated and assumed to be water-borne from adjacent Native American communities, but no source was found in the tributaries. It was finally traced to infected guides who were shedding organisms in the stool and contaminating food through poor hygiene.[136] Personal hygiene, mainly hand washing, prevents spread of infection from food contamination during preparation of meals.[136] A widely publicized study in the United States demonstrated that only 67% of Americans wash hands after using a public toilet. Simple hand washing with soap and water purified with hypochlorite (bleach) significantly reduced fecal contamination of market-vended beverages in Guatemala.[207] No one with a diarrheal illness should prepare food.

Sanitation should extend to the kitchen or food preparation area.[127] In addition to hand washing, dishes and utensils should be disinfected by rinsing in chlorinated water, prepared by adding enough household bleach to achieve a distinct chlorine odor.

Prevention of food-borne contamination is also important in preventing enteric illness (see Chapter 62). Washing fruits and vegetables in purified water is a common practice, but little data support its effectiveness. Washing has a mechanical action of removing dirt and microorganisms while the disinfectant kills microorganisms on the surface. However, neither reaches the organisms that are embedded in surface crevices or protected by other particulate matter. When lettuce was seeded with oocysts, then washed and the supernatant examined for cysts, only 25% to 36% of *Cryptosporidium parvum* and 13% to 15% of *C. cayetanensis* oocysts were recovered in the washes. Scanning electron microscopy detected oocysts on the surface of the vegetables after washing.[157] Chlorine, iodine, or potassium permanganate is often used for this purpose. Higher concentrations can be used than would normally be palatable for drinking water. With superchlorination-dechlorination, high-chlorine concentrations are used to rinse vegetables because the chlorine can be removed with the second step. Aquaclear (NaDCC) chlorine tablet instructions suggest 20 mg/L for washing vegetables. Although effective against most microorganisms, these levels would not be effective against *Cryptosporidium* or *Cyclospora*.

The ultimate responsibility is proper sanitation to prevent contamination of water supplies from human waste. UV rays in sunlight eventually inactivate most microorganisms, but rain may first wash pathogens into a water source.

Some suggest that campers smear feces on rocks. Although desiccation occurs, UV disinfection is not reliable, and feces may wash into the watershed with rain runoff.[45] Moreover, it would be repulsive to other campers. In the Sierras, feces left on the ground generally disappeared within 1 month, but it was not known whether disinfection occurred before decomposition or whether the feces washed away, dried, or were blown in the wind.[175] Despite more rapid decomposition in sunlight rather than underground, burying feces is still preferable in areas that receive regular use.

In the soil, microorganisms can survive for months.[235] A Sierra Club study found more prolonged survival in alpine environments.[175] The investigator marked group latrines in alpine terrain and returned 1 to 2 years later to dig test trenches. He found a thin crust of decomposition covering unaltered raw waste with high coliform bacteria counts. Microorganisms may percolate through the soil. Most bacteria are retained within 20 inches of the surface, but in sandy soil this increases to 75 to 100 feet[233]; viruses can move laterally 75 to 302 feet.[198] When organisms reach groundwater, their survival is prolonged, and they often appear in surface water or wells.[235]

The U.S. military and U.S. Forest Service recommend burial of human waste 8 to 12 inches deep and a minimum of 100 feet from any water.[233,237] Decomposition is hastened by mixing in some dirt before burial. Shallow burying is not recommended because animals are more likely to find and overturn the feces. Judgment should be used to determine a location that is not likely to allow water runoff to wash organisms into nearby water sources. Groups larger than three persons should dig a

common latrine to avoid numerous individual potholes and inadequate disposal. To minimize latrine odor and improve its function, it should not be used for disposal of wastewater.

In some areas the number of individual and group latrines is so great that the entire area becomes contaminated. Therefore sanitary facilities (outhouses) are becoming common in high-use wilderness areas. Popular river canyons require camp toilets, and all waste must be carried out in sealed containers.

APPENDIX A: DISINFECTION DEVICES

Product lines are continuously evolving, and prices change frequently and vary widely. There has been a major consolidation of filter product producers in the United States. Katadyn has acquired PUR and Exstream, while MSR has acquired Sweetwater and Miox. PentaPure (formerly WTC) was purchased by CUNO, which has completely dropped the line of iodine resin–containing portable outdoor filters. They now sell only filters for appliances.

For most of these products, claims are substantiated only by company sponsored and designed testing. Some results have been extrapolated to similar products. All new products must be tested using a standardized EPA protocol. Depending on claims, filters must demonstrate removal or inactivation of 10^3 cysts (99.9%), 10^4 viruses (99.99%), and 10^6 bacteria under varying water conditions of temperature and turbidity. Objective, comparable test results for these products are not available.

Filter capacity is especially variable, depending on clarity of water. Numbers provided for capacity are usually with use of clear water. Comparative testing for filter capacity using slightly turbid river water and following manufacturer instructions for cleaning revealed markedly different values for some filters. All field water has some sediment that clogs filters and reduces flow and capacity. For all filters, it is recommended to pump dilute bleach solution through the unit after each trip and dry thoroughly before storage to decrease bacterial growth in the filter.

APPENDIX A

Katadyn Endurance Series **www.katadyn.com**
(800) 755-6701

PRODUCT	PRICE	STRUCTURE/FUNCTION
		All filters contain a 0.2-micron ceramic candle filter, silver impregnated to decrease bacterial growth. Large units also contain silver quartz in center of filter.
Katadyn Pocket Filter (Fig. 61-4)	$189	Hand-pump; 40-inch intake hose and strainer, zipper case; In-line carbon cartridge available; size: 10 × 2 inches; weight: 23 oz; flow: 0.75 to 1 L/min; capacity 13,000 L.
Katadyn Combi (Fig. 61-5)	$160	Small hand-pump with ceramic filter and activated charcoal stage; with the optional "PLUS" package, the Combi can be connected to a water faucet for multifunctional use in campers, cottages or boats; can brush ceramic to clean or can separately replace elements; size: 2.4 × 10.4 inches; weight: 19 oz; flow: 1.0 L/min; capacity up to 50,000 L, 200 L for charcoal.
Katadyn Mini (Fig. 61-6)	$90	Smaller, lighter hand-pump; 31-inch intake hose and strainer, hard plastic enclosure and pump; size: 7 × 2.75 × 1.75 inches; weight: 9 oz; flow: 0.5 L/min; capacity: approx 7000 L.
Katadyn Expedition (Fig. 61-7)	$890	Large hand pump with steel stand for large groups; size (packed in case): 23 × 6 × 8 inches; weight: 12 lb; flow: 4 L/min; capacity (per filter element) to 100,000 L.
Katadyn Ceradyn and Gravidyn (Fig. 61-8)	$160 to $190	Gravity drip from one plastic bucket to another with three ceramic candle filter elements. Ceradyn filter candles are ceramic only, whereas Gravidyn filter candles combine ceramic and activated carbon elements. Size: 18 × 11 inches (26-inches high when assembled); weight: 9 lb 4 oz; flow: 1 pt/hr (10 gal/day); capacity to 100,000 L.
Katadyn Camp	$100	Gravity siphon filter element available alone (Syphon) or with a 10-L water bag (Camp); size: 12 × 2 inches;
S Syphon filter	$60	weight: 2 lb; flow: 2 gal/hr; capacity 5000 to 20,000 L.
Base Camp (Fig. 61-9)	$60	Camp gravity siphon filter will be phased out and replaced with Base Camp, which will have a pleated glass (see Katadyn Hiker under Katadyn Backcountry Series) cartridge, rather than ceramic, with a higher flow rate and lower price.

Katadyn Endurance Series
Claims
Removes bacterial pathogens, protozoan cysts, parasites, nuclear debris. Clarifies cloudy water. If filter clogs, brushing the filter element (which can be done hundreds of times before needing to replace filter element) can restore flow. Claims for removal of viruses by ceramic filters not made in the United States, although testimonials offered imply effectiveness in all polluted waters. Pocket Filter has a lifetime warranty.

Comments
Well-designed, durable products that are effective for claims. However, high filter volume capacity is optimistic and not likely to be achieved filtering average surface water. *Backpacker* magazine field tests found the flow comparatively slow, requiring more energy to pump and needing frequent cleaning. Abrading the outer surface can effectively clean ceramic filters, but it is necessary to use the gauge to indicate when filter becomes too thin.

Pocket Filter is the original, individual or small group filter design. Metal parts make it durable but the heaviest for its size. Minifilter was designed to be lighter and more cost competitive. Expedition filter is popular for larger groups, especially river trips where weight is not a factor. Complete virus removal cannot be expected, although most viruses clump or adhere to larger particles and bacteria that can be filtered. Silver impregnation does not prevent bacterial growth in filters. Bottle filter with iodine resin allows no contact time and may not provide complete viral protection in all situations.

Figure 61-4. Katadyn Pocket Filter (Courtesy Katadyn.)

Figure 61-5. Katadyn Combi. (Courtesy Katadyn.)

Figure 61-6. Katadyn Mini. (Courtesy Katadyn.)

Figure 61-7. Katadyn Expedition. (Courtesy Katadyn.)

Figure 61-8. Katadyn Drip Ceradyn. (Courtesy Katadyn.)

Figure 61-9. Katadyn Base Camp. (Courtesy Katadyn.)

APPENDIX A

Katadyn Backcountry Series (Formerly PUR Water Filters)

PRODUCT	PRICE	STRUCTURE/FUNCTION
Katadyn Guide (Fig. 61-10)	$80	Hand pump with 150-micron intake filter, 0.3-micron pleated filter with 143-square-inch surface area; includes prefilter and bottle adaptor; size: 9.5- × 2.25-inch; weight: 14 oz; max flow: 1 L/min (36 strokes/L); capacity: 100 gal/cartridge.
Katadyn Hiker (Fig. 61-11)	$60	Hand-pump with 0.3- micron pleated glass fiber with 107-square-inch surface; microfilter; size: 6.5 × 2.5 × 3.5 inches; weight: 11 oz; flow: 1 L/min (48 strokes/L); capacity: 200 gal.

Katadyn Backcountry series
Claims
Guide and Hiker are microfilters designed for high-quality surface water. They will eliminate *Giardia*, *Cryptosporidium*, and most bacteria; activated carbon core "reduces chemicals and pesticides, plus improves taste of water." Filters with large surface area are "guaranteed not to clog for 1 year."

Comments
None of these products contain iodine resin. The former PUR Explorer is no longer in the product line.
Guide and Hiker were designed for the domestic backpacking market with higher water quality, where cysts and bacteria are a threat but viruses are less of a problem. The Hiker received top ratings by *Backpacker* magazine for field tests evaluating user-friendliness. These filters may be used with a halogen disinfectant for international travel or conditions where high levels of contamination are possible.

Figure 61-10. Katadyn Guide. (Courtesy Katadyn.)

Figure 61-11. Katadyn Hiker. (Courtesy Katadyn.)

APPENDIX A

Katadyn Ultralight Series

PRODUCT	PRICE	STRUCTURE/FUNCTION
Mini (see earlier)		
Exstream Exstream XR (Fig. 61-12)		Drink-through bottle with three-stage water filter: 1-micron cyst filter for protozoa, ViruStat iodine resin, activated carbon; Exstream weight: 7 oz; capacity: 21 oz (bike bottle size). Exstream XR weight: 8 oz; capacity: 28 oz; cartridge capacity: 26 gal.

Katadyn Exstream

Claims

Passed EPA tests to remove 3-log cysts, 4-log viruses, and 6-log bacteria. Patented ion-release technology and carbon scrubber dramatically reduce residual iodine. I_5 is 1000 times more effective than I_3 resin.

Comments

Currently the only water bottle or filter product available with iodine resin. Drink-through with iodine resin and charcoal allows no contact time and may not provide complete viral protection in all situations.

Drink-through design limits Exstream to day use.

Figure 61-12. Katadyn Exstream. (Courtesy Katadyn.)

APPENDIX A

Katadyn Marine Products (Formerly PUR Products)
Reverse Osmosis Filters

PRODUCT	PRICE	STRUCTURE/FUNCTION
Desalinator Survivor 06 (Fig. 61-13)	$550	Hand-operated pump, reverse osmosis membrane filter with prefilter on intake line; size: 2.5 × 5 × 8 inches; weight: 2.5 lb; flow: 40 strokes/min; yield: 1 L/hr.
Survivor 35 (Fig. 61-14)	$1425	Hand-operated pump, reverse osmosis membrane filter with prefilter on intake line; size: 3.5 × 5.5 × 22 inches; weight: 7 lb; flow: 1.2 gal/hr (75 mL/min).
Powersurvivor 40E	$2499	Smallest power-operated model in this line of reverse osmosis filters; uses only 4 amps, so it can run for extended periods on alternative power alone; converts to manual operation in emergencies; contains 40% fewer parts than its predecessor. Pump size: 6.75 × 16.5 × 15.5 inches; prefilter 12 × 6 inches; weight: 25 lb (11.33 kg); flow: 1.5 gal/hr.

Reverse Osmosis Filters
Claims
Reverse osmosis units desalinate, removing 98% salt from seawater by forcing water through a semipermeable membrane at 800 psi. In the process, microorganisms are filtered out. The manual operation of these units makes them unique and useful for survival at sea or for use in small craft without power source. Larger, power-operated units are also available.

Comments
Reverse osmosis units are included here for sea kayaking and small boat journeys in open water. Most large ocean-going boats use reverse osmosis filters. These units can obviate the need to rely solely on stored water or can be carried for emergency survival. The U.S. military uses truck-mounted reverse osmosis filters on land for their ability to handle brackish water and remove all types of microorganisms. Reverse osmosis filters are not practical for land travel because of cost, weight, and flow rates. Desalination units will remove microorganisms, including viruses, which are larger than sodium molecules. Note that the company does not make claims for viral removal because they assume that the membrane is imperfect and some pores will be imprecise, perhaps allowing viral passage. Higher-flow power models are available from the company.

Figure 61-13. Survivor 06. (Courtesy Katadyn.)

Figure 61-14. Survivor 35. (Courtesy Katadyn.)

APPENDIX A

British Berkfeld **U.S. distributor: James Filter**
(800) 350-4170
www.jamesfilter.com

PRODUCT	PRICE	STRUCTURE/FUNCTION
SS-4 (Big Berkey)	$249	Stacked stainless steel containers with four 9-inch ceramic filters with activated carbon core; gravity flow; weight: 6.0 lb; assembled size: 19.5 × 8.5 inches; flow: 1.3 gal/hr; 2.4-gal capacity of lower container.
Arctic Travel Pure	$165 to $229	Smaller stainless steel containers with variable numbers of one to three 7-inch filter elements; lower container capacity: 1 gal.
LP-2	$145	Food-grade high-density polypropylene with two ceramic elements, gravity flow; weight: 4 lb; size: 24 × 10 inches; flow: 1/2 gal/hr; lower container capacity: 2.4 gal.

British Berkfeld Claims and Comments
Filter design is very similar to that of Katadyn Drip Filter. These filters are excellent for stationary base camps or expatriate homes. Ceramic filter is 0.9-micron absolute, but filters more than 99.99% of particles larger than 0.5 micron. Removes 100% cysts and 4- to 5-log bacteria. For complete protection, requires chlorine treatment as first step.

APPENDIX A

AquaRain Filter Systems (800) 572-2051
www.aquarain.com

PRODUCT	PRICE	STRUCTURE/FUNCTION
AquaRain 200 AquaRain 400	$149 $199	Stainless steel containers (3 gal each) with two or four silver-impregnated ceramic filters with carbon core; gravity flow; weight: 10 lb; size 22 × 10.25 inches; flow: 32 gal/day with four elements, 16 gal/ day with two elements; capacity: 30,000 to 60,000 gal.

AquaRain Claims and Comments
Similar design as Berkfeld and Katadyn, although ceramic elements differ slightly (see earlier comments); 0.2-micron absolute pore size, removes 100% cysts and 4-log reduction of bacteria. No claims for viruses. Clean ceramic elements 200 times before replacement.

APPENDIX A

General Ecology, Inc. (610) 363-7900
www.general-ecology.com

PRODUCT	PRICE	STRUCTURE/FUNCTION
		All filters (except Microlite) contain 0.1-micron (0.4-micron absolute) "structured matrix" filter in removable canister.
First Need Deluxe Water Filter (Fig. 61-15)	$93	Hand pump with intake strainer; outflow end connects directly to common water bottle and adaptors available for platypus bottles; self-cleaning prefilter float; size: 6 × 6 inches; weight: 15 oz; flow: 1.6 L/min; capacity: 100 to 400 L.
Trav-L-Pure (Fig. 61-16)	$144	Filter and hand pump in rectangular housing (1.5 pt capacity); pour water into housing, then pump through prefilter and microfilter; size: 4.5 × 3.5 × 6.75 inches; weight: 22 oz; flow: 1 to 2 pt/min; capacity: 100 to 400 L (carrying case included).
Trav-L-Pure Camper (Fig. 61-17)	$65	Trav-L-Pur canister with attachment to hose bibb for RVs and trailers; requires water pressure of 20 psi; flow: 0.5 gpm (1.9 L/min); capacity: 570 L.
First Need Base Camp (Fig. 61-18)	$500	Stainless steel casing and hand pump connected with tubing; capacity 1000 gal; size: canister, 4.8 × 5.4 inches; pump, 1.5 × 10.5 inches; weight: 3 lb; flow: 2 L/min (carrying case included). Also available with electric pump and can be hooked up in series to provide higher capacity and flow.
First Need gravity feed bag attachment (Fig. 61-19)		Allows the user to let gravity push water through the filter.

General Ecology Filters

General Ecology also has an extensive product line of larger-capacity filters for use on cars, boats, and aircraft, and for other situations requiring high-volume output where power is available. These utilize the Seagull IV purifier cartridge with a pump that pushes water through the system and a prefilter that can be cleaned. They can be operated off regular current or vehicle battery.

Claims

First Need Filter is a proprietary blend of materials including activated charcoal. "Microfiltration" with 0.1-micron retention (0.4 absolute) "removes bacteria and larger pathogens" (cysts, parasites). "Adsorption and molecular sieving" carbon absorbers remove chemicals and organic pollutants that cause color and taste; cavities in surface of adsorption material draw particles in deeper. Does not remove all dissolved minerals or desalinate. Proprietary process also creates ionic surface charge that removes colloids and ultrasmall particles through "electrokinetic attraction." Has passed laboratory tests as a purifier under single-pass conditions without added chemicals or hold time, reducing test virus by 10^4 as well as bacteria by 10^6 and *Cryptosporidium* by 10^3. Although it has not been tested with hepatitis virus, testing with rotavirus and poliovirus indicates effectiveness against viruses.

Microlite removes sediment, protozoan cysts, algae, and chemicals (including iodine), and improves color and taste of water. Iodine tablets are included to kill bacteria and viruses when these organisms are a concern.

Comments

Reasonable design, cost, and effectiveness. All units (except Microlite) use the same basic filter design. Most testing with *E. coli* and *Giardia* cysts show excellent removal. Charcoal matrix will remove chemical pollutants. This is the only company that has met EPA standards for 4-log reduction of viruses through filtration without halogens. Despite viral claims, recommend caution in highly polluted water; prior disinfection with halogen or clarification with coagulation-flocculation would provide additional security and coagulation-flocculation would extend filter life, whereas filter carbon would subsequently remove halogen. The filter cannot be cleaned, although it can potentially be back-flushed; so it must be replaced when clogged.

The Microlite is designed primarily for day use or light backpacking. Used alone, it makes microbiologic claims for protozoan cysts (*Giardia* and *Cryptosporidium*) only. Iodine or chlorine should be used as pretreatment with this filter for all water except pristine alpine water in North America. This filter is compact, lightweight, and designed for low-volume use with inexpensive, easily changed filter cartridges.

Figure 61-15. First Need Deluxe. (Courtesy General Ecology, Inc.)

Figure 61-16. Trav-L-Pure. (Courtesy General Ecology, Inc.)

Figure 61-17. Trav-L-Pure Camper. (Courtesy General Ecology, Inc.)

Figure 61-18. First Need Base Camp. (Courtesy of General Ecology, Inc.)

Figure 61-19. First Need Gravity feed. (Courtesy General Ecology, Inc.)

Figure 61-20. Waterworks EX. (Courtesy MSR.)

APPENDIX A

MSR Longevity Series (206) 624-7948
www.msrcorp.com/filters

PRODUCT	PRICE	STRUCTURE/FUNCTION
Waterworks EX Total Filtration System (Fig. 61-20)	$130	Four filter elements of decreasing pore size: porous foam intake filter, 10-micron stainless steel wire mesh screen, cylindrical ceramic filter with block carbon core, then 0.2-micron pharmacologic-grade membrane filter; pressure relief valve releases at 90 to 95 psi; hand pump with intake tubing; storage bag (2 or 4 L) attaches directly to outlet of pump. Size: 9 × 4 inches; weight: 19 oz; flow: 1 L/90 sec (1 L/min max); capacity: up to 2000 L (400 L under average conditions).
Miniworks EX (Fig. 61-21)	$80	Similar external design to Waterworks, but uses a slightly different ceramic filter and lacks the final membrane filter; weight: 16 oz; flow rate: 1 L/min; capacity: 400 to 2000 L.
Field maintenance kit	$10	Silicone, replacement O-rings

MSR Filter
Claims
Fully field-maintainable, meaning all elements can be removed, cleaned, and replaced. Removes protozoa (including *Giardia* and *Cryptosporidium*), bacteria, pesticides, herbicides, chlorine, and discoloration. Both filters meet EPA standards for removal of cysts and bacteria. Ceramic filters reduced turbidity from 68.8 NTU (nephelometric turbidity units) to 0.01 NTU. Carbon has been shown to reduce levels of iodine from 16 mg/L to less than 0.01 mg/L for at least 150 L.

Comments
Excellent filter design and function. Prefilters protect more expensive inner, fine-pore filters. Effective for claims; high quality control and extensive testing. No claims are made for viruses, although clumping and adherence remove the majority (currently 2- to 3-log removal, but not 4-log required for purifiers). The company is working on a microfilter that will effectively remove viruses. Until they succeed, the filter should not be considered reliable for complete viral removal from highly polluted waters in developing countries. Reservoir bag that attaches to outflow for filtered water storage is convenient. Design and ease of use are distinct advantages. Filter can be easily maintained in the field; maintenance kit and all replacement parts available. Ceramic filters can be effectively cleaned by abrading outer surface many times without compromising the filter. A simple caliper gauge indicates when filter has become too thin for reliable function. Miniworks was rated very highly in *Backpacker* magazine field tests.

Figure 61-21. Miniworks EX. (Courtesy MSR.)

Figure 61-22. Sweetwater Microfilter and Purifier Solution. (Courtesy MSR.)

APPENDIX A

MSR Simplicity Series **www.msrcorp.com/filters**

PRODUCT	PRICE	STRUCTURE/FUNCTION
Sweetwater Microfilter (Fig. 61-22)	$60	Lexan body and pump handle; 100-micron metal prefilter; in-line 4-micron secondary filter; labyrinth filter cylinder of borosilicate fibers removes pathogens to 0.2 micron; granular activated carbon; safety pressure relief valve; end-of-life indicator; outflow tubing has universal adapter that fits all water bottles; optional input adapter that attaches to sink faucet while traveling; size: 7.75 × 3.5 inches; weight: 11 oz; flow: 1.25 L/min (new filter); capacity: 200 gal.
Purifier Kit with Microfilter, purifier, and platypus water bag	$75	
Purifier solution	$9	A chlorine-based purifier solution containing 3.5% sodium hypochlorite; add 5 drops to each liter of filtered water, mix for at least 10 seconds, and wait 5 minutes.

Sweetwater Filter

Claims

Eliminates *Giardia, Cryptosporidium,* and other critical bacterial and protozoan pathogens, pollutants, heavy metals, pesticides, and flavors. Lighter, more compact, and more durable than comparable models; easiest to clean or replace. The company recycles filter cartridges.

Comments

Well-designed filter at a reasonable price. Practical design features like universal bottle adapter. Pressure-release valve indicates when filter needs cleaning, but this can be a problem as the filter clogs. A brush is provided for cleaning and cartridges are replaceable. Rated highly in *Backpacker* magazine field tests.

The iodine resin–containing Viral Guard was recently removed from the market. New in-house testing of the Guardian filter with iodine resin attachment failed to inactivate poliovirus at levels required by EPA standards under certain conditions. The failure of the viral testing was a surprise to the company, because a reliable laboratory had originally tested the product with the iodine resin and verified adequate levels of viral inactivation. It is unclear whether this indicates a problem in general with the iodine resins or with the specific design of their iodine resin filter attachment. Because they were unable to solve the problem, the company has developed a specially formulated chlorine solution (Purifier Solution) for use before filtration when high levels of contamination are possible, including viruses.

APPENDIX A

MSR pump free series **www.msrcorp.com/filters/pump_free**

PRODUCT	PRICE	STRUCTURE/FUNCTION
Miox Purifier (Fig. 61-23)	$130	New technology created by military, uses salt, water, and electrical current generated from camera batteries to produce mixed-species disinfectant solution that is added to water. Kit comes with pen, batteries, salt packet, indicator strips, instructions, storage sack. Refills of salt cartridge are available. Pen 3.5 oz; kit 8 oz.

Claims

Just add water, shake, and press a button. Solution generated is then added to drinking water. Inactivates all bacteria, viruses, and protozoa. Viruses and bacteria require dwell time of 15 min, *Giardia* 30 min, *Cryptosporidium* 4 hours. Can be used for individuals or groups.

LED (light-emitting diode) lights indicate low battery or inadequate reagents (salt and water). Test strips are used to test water for adequate chemical residual.

Comments

Truly new technology. The science has been known for some time and is used in large commercial processes (www.miox.com), but previously was not available in portable design. Although difficult to measure various disinfectant species generated, testing has confirmed that disinfectant activity is greater than with comparable concentrations of sodium hypochlorite. The active oxidant disinfectant that kills *Cryptosporidium* is assumed to be chlorine dioxide. The extended dwell time indicates low concentrations of this disinfectant and the need to plan for prolonged contact time, if *Cryptosporidium* is a strong concern.

High technology approach is appealing to some, but intimidating to others. The indicator lights and test strips add reassurance that the process is proceeding effectively. Although this technique can be used for large volumes, this instrument generates enough for only 1 L at a time and may be difficult to use for larger volume containers.

Figure 61-23. Miox Purifier. (Courtesy MSR.)

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Sawyer Products **http://www.sawyerproducts.com/**

PRODUCT	PRICE	STRUCTURE/FUNCTION
Biologic Water Filter (Fig. 61-24)	$60	0.2-micron (absolute) hollow fiber membrane filter, which is a cluster of microtubules. Viral filter has 0.02 micron fiber filter. Water is drawn through the walls of the tubules either by suction or by suction, gravity or pressure applied in squeezing the bottle; Both filter cartridges available as inline cartridge for backpack water systems, gravity drip, faucet in-line systems, or polycarbonate drinking bottle with filter. Activated charcoal prefilter cartridge included for optional use; Capacity more than 500 gallons, depending on clarity of source water; rate depends on method used, 5 gallons/30min via gravity feed system; 30 mL/sec from drink through.
Viral water Purifier (includes faucet adaptor)	$120	
Viral water Purifier Kit (in-line filter (Fig. 61-24), water bottle (Fig. 61-25), faucet adapter, gravity bag)	$150	

Claims

Microtubules are new technology that allow unprecedented flow rates and 0.2 or 0.02 micron absolute filtration. Prefilter removes lead, chlorine, odors, taste sediment, while the microfilter removes bacteria, protozoa, and cysts. Biologic filter removes 7-log (99.9999%) bacteria and 6-log protozoa but makes no claims for viruses; add chlorine to kill viruses. Viral filter claims >5 log removal through mechanical filtration. Filter is freeze-resistant, which is not true of many solid filters. Sawyer will supply liquid and tablet form of chlorine to use in combination with the filter to purify highly questionable water.

Comments

This filter was carefully designed and tested. The potential advantage over other water bottle filters is the hollow fiber technology that may provide faster filtration with less pressure. The charcoal cartridge compromises some of this low-pressure flow, so it is supplied separately for use only when needed. Highly versatile filter product that can be used as water bottle or with camel-back for individuals, gravity drip for small groups, or in-line for added microbiologic protection in households. This is the first low-pressure filter to claim viral removal through mechanical filtration alone.

Figure 61-24. Sawyer Water Filter Bottle. (Courtesy Sawyer Products.)

Figure 61-25. Sawyer filter. (Courtesy Sawyer Products.)

APPENDIX A

Hydro-Photon, Inc. (888) 826-6234
www.hydro-photon.com

PRODUCT	PRICE	STRUCTURE/FUNCTION
Steri-Pen (Figs. 61-26 and 61-27) Replacement element available Water bottle prefilter	$150 $10	Portable, battery-operated ultraviolet water disinfection system. Disinfects up to 16 oz of clear water in less than 1 min and 32 oz in 90 seconds by stirring UV element in water. Uses four AA batteries (disposable or rechargeable): alkaline batteries provide 20 to 40 treatments, lithium batteries 130 to 140 treatments; weight: 8 oz, with batteries; length, 7.6 inches, width 1.5 inches; lamp lasts 5000 treatments (625 gal). Comes with a thermoformed nylon carrying case. Prefilter available for removing particulate matter; fits on top of Nalgene water bottle.

Steri-Pen

Claims

Highly effective against bacteria, viruses, and protozoa, including *Cryptosporidium* oocysts. Dosage selector for 16- or 32-oz water treatment. Alkaline batteries not recommended for cold water treatment. Unit automatically turns off lamp after UV dose is delivered. Dose counter indicates when lamp replacement is necessary.

Microbiologic testing conducted at the University of Arizona, the University of Maine, the Oregon Health Sciences University, and Hydro-Photon against multiple types of bacteria, viruses, *Giardia,* and *Cryptosporidium* shows that Steri-Pen meets the standard as set forth in the U.S. EPA Guide Standard and Protocol for Testing Microbiological Water Purifiers, destroying in excess of 99.9999% of bacteria, 99.99% of viruses, and 99.9% of protozoa.

Comments

In general, UV light for water disinfection is well established and widely used for water treatment in many large and varied applications. Until now, these have required a larger, fixed power and light source. The use of this portable technology is currently limited to small volumes of clear water; however, the potential is great for further advances that will increase its uses in the field and make it less expensive. The testing for this device can be found on Hydro-Photon's website. Testing was only successful in clear water, not in EPA "worse case scenario" water unless prefiltered with microfiltration. The simplicity and rapidity of this technique is appealing.

Figure 61-26. Steri-Pen. (Courtesy Hydro-Photon, Inc.)

Figure 61-27. Steri-Pen in use. (Courtesy Hydro-Photon, Inc.)

APPENDIX A

Stearns Outdoors, Inc. (800) 697-5801
(Formerly Basic Designs Filters) **www.stearnsinc.com**

PRODUCT	PRICE	STRUCTURE/FUNCTION
High-Flow Ceramic Water Filter (Fig. 61-28)	$72	Ceramic filter with 0.5- micron absolute retention size and carbon center; gravity filtration with element placed near the end of a 6-foot outflow tube connected to a 7.5-L heavy plastic collection bag, providing 2 to 3 lb of hydrostatic pressure through the in-line filter; packing size: 4 × 4 × 8 inches; weight: 1 lb 2 oz; flow rate: 15 L/hr; capacity: 500 to 1000 L.
Ceramic Filter Pump (Fig. 61-29)	$26	Hand pump with ceramic cartridge at end of intake tubing and polyurethane prefilter; size: pump, 8 × 1 inch; filter, 4 × 3 inches; 18-inch tubing; weight: 7 oz; flow: 0.4 L/min; capacity: 500 gal.

Stearns/Basic Designs Filters
Claims
Ceramic filter removes *Giardia, Cryptosporidium,* cysts, tapeworm, flukes, other harmful pathogens larger than 1 micron. Carbon removes color, tastes, and odors. Filter can be cleaned with an abrasive pad. Filter pump makes claims for cysts only. Pump is easily serviced in the field; ceramic cartridge is replaceable.

Comments
Ceramic candle filters are effective filtering elements, and charcoal is an effective adsorbent. No claims for virus removal. Although the pore size is larger than that of most filters, the low-pressure depth filter increases retention of bacteria. The simple gravity design decreases cost and moving parts. Filtration rate will be slow, and this filter could clog rapidly, in that there is no prefilter for larger particles. Gravity drip can be convenient after making camp, if no time restraints. The filter pump is the most practical hand-pump unit and is reasonably priced, but the ceramic filter can break. The intake is close to the pump, which can be awkward, and the foam sleeve makes it float, requiring an extra hand to hold it underwater. This filter rated poorly on field user tests by *Backpacker* magazine.

Figure 61-28. Stearns High-Flow Ceramic Water Filter. (Courtesy Stearns, Inc.)

Figure 61-29. Stearns Ceramic Filter Pump. (Courtesy Stearns, Inc.)

APPENDIX A

Timberline Filter **www.timberlinefilters.com**

PRODUCT	PRICE	STRUCTURE/FUNCTION
Timberline Eagle	$26	1-micron fiberglass and polyethylene matrix; hand pump; size: 9 × 1 to 3 inches; weight: 6 oz; flow: 1 qt in 1.5 min.
Replacement filter	$16	
Base Camp Filter	$66	Gravity drip 2-micron filter (same filter element as the original Timberline filter) with 2-gal reservoir coated nylon bag; flow 1.5 pt/min with 5-ft of elevation; weight of reservoir, tubing, and filter element: 11 oz.; capacity: 200 qt depending on clarity of water.
Replacement filter	$26	

Timberline Filters
Claims
Removes *Giardia* and *Cryptosporidium* cysts. No claims for bacteria or viruses.

Comments
Lightweight, inexpensive filters with limited applications. Should be used in conjunction with chemical treatment.

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McNett Aquamira Filters **www.Mcnett.com**

PRODUCT	PRICE	STRUCTURE/FUNCTION
Water Filter Bottle (Fig. 61-30)	$19	Nalgene squeeze bottle with 2-micron carbon block filter; bottle size: 22 oz; weight: 5 oz; capacity: 200 refills.
Frontier (Fig. 61-31)	$10	Drink-through straw with 2-micron charcoal filter. Capacity: up to 20 gal depending on clarity of water.

Aquamira Filters
Claims
Both filters remove *Giardia* and *Cryptosporidium* cysts. "If viruses or smaller bacteria are suspected, use a certified chemical treatment or boil water before filtering. Removes chlorine and other chemical flavors. Chemical treatments are recommended when viruses may be present in the water, as when traveling in developing countries or popular natural areas. Used in conjunction with chemical treatment, the Aquamira filter provides the benefit of removing organisms that may be too large to be inactivated by chemical treatment."
Frontier is "just in case" filter for emergency use backup to drink from any bottle or cup or directly from water sources. The Frontier Filter can also be used in conjunction with chemical treatments for maximum protection.

Comments
Water bottle is reasonable for day trips, but the nominal pore size is not adequate for bacteria, so the filter must be used with chemical treatment if viral or bacterial contamination is a concern. Straw filters are not very useful. In general, they do not perform well on testing. A better means of emergency backup is to carry chemical treatment such as halogen or chlorine dioxide.

APPENDIX B: CHEMICAL DISINFECTION PRODUCTS

See text for further discussion of halogens, chlorine dioxide, and silver.

Iodination

Crystals (Saturated Solution). Because of limited solubility in water, iodine crystals may be used for disinfection. In one technique for field use, 4 to 8 g of crystalline iodine is put in a 1- to 2-ounce bottle, which is then filled with water.[99] A small amount of elemental iodine goes into solution (no significant iodide is present); the saturated solution is used to disinfect drinking water. Water can be added to the crystals hundreds of times before they are completely dissolved.

An alternative technique is to add 8 g of iodine crystals to 100 mL of 95% ethanol.[222] Increased solubility of iodine in alcohol makes the solution less temperature dependent and allows much smaller volumes to be used (8 mg/0.1 mL), which can be measured with a 1-mL syringe or dropper (2 drops).

The stability and simplicity of iodine crystals have led to their testing for in-line systems that provide continuous water disinfection for remote households and small communities. In these designs, residual iodine is removed with GAC.[57,205]

Product
Polar Pure
www.polarequipment.com
Price: $10.50
Widely available through suppliers of outdoor products (e.g., Campmor)
Formulation
8 gm of iodine crystals in a 3-oz glass bottle filled with water; 30- to 50-micron fabric prefilter provided; "trap" in bottle to catch crystals when pouring off water; capacity: 2000 quarts; weight: 5 oz.

Figure 61-30. McNett Aquamira Water Filter Bottle. (Courtesy McNett Corp.)

Instructions

The bottle cap is used to measure iodine solution. One capful is approximately 6.5 mL. Directions and color dot thermometer are printed on the bottle. Recommended dose (2 capfuls if iodine solution is 20° C [68° F]) yields 4 ppm iodine when added to 1 quart of clean water. Contact time depends on temperature of the water to be consumed; warm water to 20° C (68° F) before adding iodine to shorten contact time.

Comments

Saturated aqueous solution of crystalline iodine is an excellent and stable source of iodine. Recommendations are adequate for clear, warm water, but because it is not feasible to warm all water, extend contact time to 1 to 2 hours for very cold water. Temperature of the bottle affects the concentration of iodine in the saturated solution (200 ppm at 10° C [50° F], 300 ppm at 20° C [68° F], 400 ppm at 30° C [86° F]),[42,84] which is the reason for the color-dot thermometer on the bottle. In the field, it may be easier to warm the bottle in an inner pocket than to estimate temperature and adjust the dose. The supernatant should be carefully decanted or filtered to avoid ingestion of the crystals[255]; this is aided by the weight of the crystals, which causes them to sink. Many people prefer crystalline iodine because of its large disinfectant capacity, small size, and light weight. The glass bottle can break.

Figure 61-31. McNett Aquamira Frontier. (Courtesy McNett Corp.)

For many years, Polar Pure Carbonics has been trying to get FDA approval for iodine crystals in alcohol. Its failure to receive approval to market this new product has no relation to effectiveness or safety. At this time, iodine in alcohol is a viable option that allows for much smaller doses due to higher solubility of iodine in alcohol.

Iodine Tablets

The tablets used by the U.S. military and sold in the United States for water disinfection contain tetraglycine hydroperiodide, which is 40% I_2 and 20% iodide.[42,145] Tetraglycine hydroperiodide was originally developed and chosen as a preferred technique by the military for individual field use because of its broad-spectrum disinfection effect, ease of handling, rapid dissolution, stability, and acceptable taste.[109,154,166,188]

Each tablet releases 8 mg/L of elemental iodine into water. An acidic buffer provides a pH of 6.5, which supports better cysticidal than virucidal capacity but should be adequate for both. Tablets have the advantages of easy handling and no danger of staining or corroding if spilled. They are stable for 4 to 5 years under sealed storage conditions and for 2 weeks with frequent opening under field conditions, but they lose 30% of the active iodine if bottles are left open for 4 days in high heat or humidity.

Products

Potable Aqua (Wisconsin Pharmaceuticals)

Price: 50 tablets, $5; with P.A. Plus Neutralizing tablets, $8
Weight: 2 oz
Widely available through suppliers of outdoor products
Also sold as Globaline and EDWGT (emergency drinking water germicidal tablets)

Instructions

One tablet is added to 1 quart of water. In cloudy or cold water, add two tablets. Contact time is only 10 to 15 minutes in clear, warm water, and much longer in cold, cloudy water (see Table 61-13).

Potable Aqua is now sold with "neutralizing" tablets made of ascorbic acid. Ascorbic acid converts iodine to iodide, removing the taste and color.

Comments

This method was developed by the military for troops in the field. Advantages are unit dose and short contact time, but these concentrations create strong taste that is not acceptable to many wilderness users. The military requirements dictated a short contact time (10 minutes in clear, warm water), thus the relatively high concentration of iodine (8 to 16 ppm). With adequate contact time and moderate temperatures, one tablet can be added to 2 quarts of water to yield 4 ppm of free iodine (see Table 61-15). Rather than use two tablets in cloudy water, clarify the water first.

Ascorbic acid (vitamin C) neutralizer reduces iodine to iodide, which has no color or taste, but also has no disinfecting action. Esthetically, iodine is "removed" from the water. However, iodide is physiologically active, so concerns about toxicity or physiologic activity remain. For short-term use, iodine is safe and removing the taste is a major benefit.

Chlorination

See text for chemistry and effectiveness of free chlorine.

Sodium Hypochlorite

Household Bleach

Liquid household bleach is a hypochlorite solution that comes in various concentrations, usually 5.25%. This has the convenience of easy availability, low cost, high stability, and administration with a dropper.

Sodium hypochlorite solutions are vulnerable to significant loss of available chlorine over time. Stability is greatly affected by heat and light. Five percent solution loses about 10% of available chlorine over 6 months at 21° C (70° F) and freezes at –4.4° C (24° F). If bleach containers break or leak in a pack, the liquid is corrosive and stains clothing.

Products

Drinkwell from Katadyn

Available from FirstWater www.firstwater.info/noflash/pdct_prsnel_chem.html

10-mL bottle can treat 100 L using 3 drops/L. Stable for 3 years if stored under 25° C (77° F).

SweetWater Viral Stop

2-oz squeeze dropper bottle

Price: $9

Calcium Hypochlorite (dry chlorine)

Calcium hypochlorite is a stable, concentrated, dry source of hypochlorite that is commonly used for chlorination of swimming pools.

Redi-Chlor

www.redi-chlor.com

50 tablets in blister packs

Price: $9.95

Calcium hypochlorite is widely available in tablets or tubs of granules through chemical supply or swimming pool supply stores.

Formulations/Instructions

Redi-Chlor tablets come in different sizes and can be broken in half or fourths to treat different quantities of water. Recommended dose results in 2 to 5 mg/L residual chlorine. Add more for very cold water or if faint chlorine smell is not detected after contact time. Available in blister packs of 50 one-tenth of a gram tablets to treat 1 gal per tablet, or blister packs of 50 one-fourth of a gram tablets to treat 5 gal per tablet.

Comments

This is a convenient source of hypochlorite, which can also be used for superchlorination (see text).

Halazone Tablets

Tablets contain a mixture of monochloraminobenzoic and dichloraminobenzoic acids.[62] Each tablet releases 2.3 to 2.5 ppm of titratable chlorine.[154] These tablets have been criticized because the alkaline buffer necessary to improve halazone dissolution decreases disinfectant efficiency, requiring unacceptably high concentrations and contact times (six tablets yield 15 mg/L with recommended contact time of 60 minutes) for reliable disinfection under all conditions.[188] The shelf life is 6 months; potency decreases 50% when stored at 40° to 50° C (104° to 122° F). A new bottle should be taken on each major trip or changed every 3 to 6 months. Halazone has been replaced by newer tablet formulations of chlorine.

Chlorination-Flocculation

Tablets contain alum and 1.4% available chlorine in the form of dichloroisocyanurate (sodium dichloro-*s*-triazinetrione) with proprietary flocculating agents. Bicarbonate in the tablets promotes rapid dissolution and acts as a buffer. One 600-mg tablet yields 8 mg/L of free chlorine.

Testing by the U.S. military demonstrated biocidal effectiveness similar to iodine tablets under most conditions.[165,167,169] Extended contact time was necessary for complete viral removal in some of the tests. Because of the ability to flocculate turbid water, the action was superior to that of iodine in some poor-quality water.

Chlor-floc (Deatrick & Associates)

30 tablets individually sealed in foil packets; weight 1.6 oz.; capacity: 30 L (8 gal)

Price: $7

Chlor-Floc is widely available through military surplus and survival websites

AquaPure and AquaCure are other brands that may be available.

Proctor and Gamble has developed a packet with chlorination and flocculation powder called PUR Purifier of Water that is now available to large relief organizations for use outside the United States in disaster and conflict situations. They will also begin distributing for individual users in the developing counties.

Formulations/Instructions

One tablet for the clarification and disinfection of 1 L of water from polluted sources at temperatures of 25° C (77° F). At 5° C (41° F), use two 600-mg tablets to provide 2.8% available chlorine. To strain the sediment, pour the water through the cloth provided. The tablets are stable for 3 years if stored in their packaging out of the heat.

Comments

This is one of the individual field methods for U.S. military troops and suggested for potential use in developing countries by the WHO. It is an excellent one-step technique for cloudy and highly polluted water (see Table 61-15).

Alum is a widely used flocculant that causes suspended sediment, colloids, and many microorganisms to clump, settle to the bottom, and readily be filtered or strained. Most *Cryptosporidium* oocysts would be removed by the flocculation. Some chlorine reacts with contaminants and is inactivated. It is important to confirm some chlorine taste and smell at the end of the contact time. For added safety, prolong the contact times up to 1 hour of contact time in cold, polluted, and dirty water.

In clear water without enough impurities to flocculate, the alum causes some cloudiness and leaves a strong chlorine residual. After treatment, water should be poured through a special cloth to remove floc and decrease turbidity.

Sodium Dichloroisocyanurate (NaDCC)

NaDCC is a stable, nontoxic chlorine compound that releases free active chlorine and forms a mildly acidic solution, which is optimal for hypochlorous acid, the most active disinfectant of the free chlorine compounds. Free chlorine is in equilibrium with available chlorine that remains in compound, providing greater biocidal capacity. NaDCC is more stable and provides more free, active chlorine than other available chlorine products for water disinfection.

Aquatabs

www.firstwater.info/noflash/pdct_prsnel_chem.html

Similar product may be available under the brand Aquaclear, Puritabs, and Kintab (Bioman Products)

Formulation/Instructions

Each tablet contains sodium dichloroisocyanurate (NaDCC) in paper/foil laminate. When dissolved in 1 L of water, each effervescent tablet releases 10 mg of free chlorine, with 50% of the available chlorine remaining in compound and released as free chlorine to be used up by halogen demand. Tablets are available in multiple strengths and can be broken to treat smaller quantities of water. Larger quantities are available in tubs. Aquatab also makes slow-dissolving tablets for larger quantities of water that contain trichloroisocynauric acid (TCCA), which acts similarly to NaDCC.

Surface water disinfection of clear water is accomplished at 10 mg/L in 10 min, 1 mg/L for tap water, and 2 to 5 mg/L for well water. NaDCC can also be used to wash fruits and vegetables in concentrations of 20 mg/L.

The tablets have a 3-year shelf life; cost depends on size and quantity of tablets.

Comments

This is a good source of chlorine available in multiple dosage forms, including individually wrapped tablet form; larger concentration tablets allow for disinfection of large quantities of water for shock chlorination of tanks and other storage systems.

Chlorine Dioxide

Until recently, chlorine dioxide could be used only in large-scale water treatment applications, because it is a volatile gas that must be generated on-site. Several new chemical methods for generating chlorine dioxide on-site can now be applied in the field for small quantity water treatment. Advantages of chlorine dioxide are greater effectiveness than chlorine at equivalent doses and the ability to inactivate *Cryptosporidium* oocysts with reasonable doses and contact times.

Katadyn MicroPur MP-1

www.katadyn.ch/site/us/home/outdoor_products

Micropur is also available through Black Mountain, Campmor, and others at www.katadyn.net/micropur.html (Black Mountain commercial site markets a range of Katadyn products)

Price: 30 tablets, $13.95

Engelhard (www.engelhard.com) produces a similar product called Aseptrol, a chlorine dioxide release agent in various strength tablets and powders that are EPA registered for a wide range of disinfection tasks

Formulation/Instructions

The primary chemistry reaction that produces chlorine dioxide (ClO_2) in MP-1 tablets is the acid-chlorite reaction using sodium acid sulfate as the acid, a well-known reaction for ClO_2:

$$5NaClO_2 + 4NaHSO_4 \rightarrow 4ClO_2 + NaCl + 2H_2O + 4Na_2SO_4$$

A small amount of chlorine in the tablet also catalyzes the otherwise sluggish reaction.

These tablets generate chlorine dioxide only when coming into contact with water. Shortly after a tablet is immersed in water, a saturated solution of the soluble solid constituents forms within the matrix of the tablet. ClO_2 is rapidly formed within the pores and then carried into the bulk solution by CO_2 effervescence, which ensures that the resultant solution is well mixed without the user having to agitate the container. After the chlorine dioxide gas is released, the material reduces into common salts.

Company testing shows killing of bacteria and viruses within 15 minutes in any water conditions, and inactivation of *Giardia* and *Cryptosporidium* within 30 minutes in clear, warm water and 4 hours in cold and dirty water. One tablet is used for treating 1 quart of water. Instructions are to insert rapidly into water after removing from package and avoid exposure to sunlight during disinfection contact time.

Comments

This is a major advance in portable and point-of-use water disinfection. The product is EPA registered as an antimicrobial water purifier. The extended contact time in cold, dirty water ensures that sufficient chlorine dioxide is generated and adequate residual remains for sufficient time to treat water in all conditions. It is more reasonable to clarify the water to improve taste and esthetics and warm the water to reduce contact time. Company testing is well designed with multiple controls. They also have documentation that residual concentrations of chlorine dioxide were well maintained during the recommended contact times. Chlorine dioxide does not have extended persistence in water, so it should not be used to maintain microbiologic purity of stored water. Sunlight breaks down chlorine dioxide, so for optimal effect, keep the water bottle in a dark location, such as inside a pack or bag, during disinfection time.

aqua mira (McNett Corp)

www.mcnett.com

Pristine (Canada)

www.pristine.ca

A stabilized solution of chlorine dioxide is mixed with phosphoric acid, which activates the chemical and is then mixed with water for disinfection. Contact times for inactivation of *Cryptosporidium* by Pristine range from 15 minutes in warm water using a triple dose to 7 hours using a single dose in very cold water. The two solutions are mixed together in a mixing cap

and added to the water for treatment. Advantages include effectiveness across broad range of water, temperature, and pH; no aftertaste or odor; unaffected by freezing.

Personal size: two 1 oz plastic bottles; capacity: up to 120 liters (30 gal) of water; weight: 2.8 oz; $10.99.

Pristine also makes 2-oz bottles, and larger packages for relief agencies to disinfect large quantities.

Comments

The chemistry of generating chlorine dioxide through a similar method is well described. The company is responding to concerns raised by the EPA, so product is still pending EPA approval as a purifier in the United States. It does have limited approval now for bacteria. The Canadian product makes full claims, including claims for *Cryptosporidium*. Testing data will be available from the company when EPA approval is obtained. It will be important to note the persistence of the active disinfectant. Given the volatility of chlorine dioxide and slow reaction times, concentrations may be variable due to the mixing process and time delay. Likely the EPA delay is related to performance in cold and dirty "worst case" test water, which may be an issue in disaster situations, but it is not often encountered by wilderness users.

McNett is exploring other formulations of chlorine dioxide. They are currently developing large-scale systems for relief organizations.

Chlorine Dioxide and Mixed Species Disinfection

Miox Purifier, marketed by MSR

www.miox.com

www.msrcorp.com/filters/pump_free.asp

See Appendix A for more information on this product, which uses salt and an electric current from camera batteries to generate chlorine dioxide, free chlorine species, and perhaps other disinfectants such as ozone.

Passing a current through a simple brine salt solution generates free available chlorine, as well as other "mixed species" disinfectants that have been demonstrated effective against bacteria, viruses, and bacterial spores.[197] The process is well described and can be used on both large and small scale. Chlorine dioxide and ozone have not been detected in finished water treated with the Miox mixed-oxidant solutions using classic analytical methods. This is likely due to rapid transformation of the generated oxidant constituents to other forms of oxidants. However, the resulting solution has greater disinfectant ability than a simple solution of sodium hypochlorite and has even been demonstrated to inactivate *Cryptosporidium,* suggesting that chlorine dioxide is among the chemicals generated.[239] Additional testing information is available on the Miox website.

Silver

MicroPur Forte Tablets from Katadyn

Available from FirstWater www.firstwater.info/noflash/pdct_prsnel_chem.html

Widely available in Europe but not marketed in the United States, these tablets contain silver and sodium hypochlorite. The chlorine kills viruses, bacteria, and *Giardia*. The silver adds to the disinfection capacity, and it prevents recontamination if water is stored for up to 6 months. Contact time is 20 to 120 minutes, depending on temperature of water. Shelf life is 2 years when stored in cool, dry conditions. Available as tablets or liquid.

MicroPur from Katadyn

This product releases silver ions.

Available in two sizes of tablets (for 1 quart or 5 quarts), liquid (10 drops/gal), or crystals for treating larger quantities of water.

Comments

Although it has proven antibacterial effects, silver tablets are not licensed as a water purifier in the United States; however, they are widely used in Europe for this purpose. In addition to poorly documented effects on some types of microorganisms, there is difficulty controlling the residual concentration and concern over chronic effects. This product makes no claims for viruses and protozoa, because concentrations may not be adequate to kill these organisms. Silver has the advantage of having no taste, color, or smell. It has been approved by the EPA to be marketed in the United States as a "water preservative" to maintain bacteria-free water for up to 6 months.

Miscellaneous Products

The following products *cannot* be recommended due to insufficient effectiveness data:

Traveler's Friend (Nutrabiotic)

Description

Extract from citrus seeds in 10-mL plastic dropper bottle; size: 1 × 2 inches; weight: 1 oz; capacity: approximately 20 quarts.

Price: $6

Claims

"All natural treatment for drinking water." Nontoxic, noncorrosive, proven effective as disinfectant for bacteria, viruses, protozoa. Recommended dose (drops/qt): 5 to 10 for filtered water, 10 to 15 for ice water, 10 to 20 for tap water, 15 to 25 for untreated water. Allow 30 minutes contact time.

Comments

Citrus extract is known to have some bacteriostatic effect. This product was introduced in the health food market and is now looking for a broader market. Company data from independent laboratories support bactericidal and virucidal effects. However, protozoal tests were done with trophozoites, not cysts. The data have gaps, and too much of the marketing is testimonial to give a recommendation now. The chemical species or compound responsible for disinfection needs to be identified, then dose-time disinfection values and need to be experimentally established for various microorganisms.

Aerobic Oxygen

Aerobic Oxygen was initially introduced into the health food market but is now being offered to the general travel market. It is advertised not only as a water disinfectant but also for nearly miraculous qualities ranging from strengthening the immune system and energizing the body to curing headaches and tropical fish diseases. Company literature implies that the active disinfectant is chlorine dioxide, ozone, and free oxygen radicals, but this is not chemically feasible. Company-sponsored testing demonstrates activity against bacteria and viruses but not against cysts. No dose-time response has been developed to compare the product against other disinfectants. It cannot be recommended without further data.

The references for this chapter can be found on the accompanying DVD-ROM.

62 Infectious Diarrhea from Wilderness and Foreign Travel

Javier A. Adachi, Howard D. Backer, and Herbert L. DuPont

Acute infectious diarrhea is one of the most common and significant medical problems in any population, second only to acute upper respiratory diseases.[132,327] Worldwide, diarrheal diseases were reported to cause nearly 1 billion episodes of illness in 1996,[76,95,96,287] with at least 20 million annual deaths.[220,327] The rates of illness among children in developing areas of the world range from 5 to 15 bouts per child per year, with diarrhea being the most important cause of morbidity and mortality in many regions. Readily available oral rehydration solutions prevent great numbers of dehydration-associated deaths related to acute diarrhea, especially in developing areas, but invasive bacterial enterocolitis (caused by *Shigella* species and *Campylobacter jejuni/coli*), persistent diarrhea (defined as illness lasting 14 days or longer), malnutrition, and increased susceptibility to other infections still cause significant morbidity and mortality.[76,131,220,287] In the United States, specific groups with diarrhea rates similar to those in the developing world include travelers, homosexual men, non–toilet-trained toddlers in daycare centers, and mentally impaired residents in custodial institutions.[95,115,131,132]

This chapter provides information to help decrease exposure to risk factors and enteric pathogens, thus helping to reduce the chance of acquiring diarrheal illness. The clinical features of acute diarrheal illnesses often do not permit differentiation of the specific etiologic agent, but fortunately, the majority of these infections do not require specific treatment.[7,76,95,96,132] A clinical approach to self-therapy is formulated that is likely to minimize the complications and suffering caused by these illnesses. For the purpose of this discussion, *traveler* includes business and pleasure travelers as well as wilderness venturers.

GENERAL PRINCIPLES OF ENTERIC DISEASE

Epidemiology

Transmission. Fecal–oral contamination, through ingestion of contaminated water and food, is the usual route of transmission of the enteric pathogens causing acute infectious diarrhea. Whether food or water is the more important origin depends mainly on the location and the precautions taken. The majority of pathogens that cause travelers' diarrhea or wilderness-acquired diarrhea can be either food-borne or water-borne; however, waterborne pathogens from drinking untreated surface water or from an inadvertent ingestion during water recreational activity probably account for most infectious diarrhea acquired in the U.S. wilderness.[131,196,199,209] Avoidance of all these pathogens requires proper sanitation and water disinfection. Person-to-person transmission is seen with pathogens that do not require a large infectious dose, such as *Shigella* species, the hepatitis A virus, *Giardia,* and noroviruses. These infections are most common in populations exposed to high levels of pathogens, so prevention includes adequate hand-washing and personal hygiene[76,95,96] (Table 62-1). Other, less common routes of fecal–oral transmission are through aerosols (some viruses), contaminated hands or surfaces, and sexual activity.

Location. In several areas of Africa, Asia, and Latin America where satisfactory sanitation is lacking, diarrhea is still the leading cause of infant morbidity and mortality. Good sanitation is related to a much lower incidence of infectious diarrhea in industrialized areas of the world. Travelers to foreign countries and wilderness areas often leave behind sanitation in the form of flush toilets and safe tap water, as well as proximity to advanced medical care. Similarly inadequate hygiene conditions are also found in other settings.

Outbreaks of infectious diarrhea in daycare centers among non–toilet-trained toddlers are associated with *Giardia lamblia, Shigella, Campylobacter jejuni/coli,* and *Cryptosporidium,* which have a small infectious dose. Hospitals, especially intensive care units and pediatric wards, institutions for mentally handicapped patients, and nursing homes are also locations with high incidences of diarrheal diseases. *Clostridium difficile, Salmonella* species, rotavirus, and enteropathogenic *Escherichia coli* (EPEC) are the most common etiologic agents reported[96,131,169,249,317] (see Table 62-1).

Antimicrobial Therapy. *C. difficile*–associated diarrhea is frequently related to recent use of an antimicrobial agent (or a cytotoxic agent), usually during the 2 to 4 weeks before the beginning of the diarrheal illness.[51,106,171]

Age. In developing areas of the world, children younger than 5 years have higher morbidity and mortality rates related to dehydration superimposed on malnutrition; they may experi-

TABLE 62-1. Epidemiologic Associations with Enteropathogens

AGENTS	WATERBORNE	CHILDREN*	HOSPITALIZED/ INSTITUTIONALIZED	HOMOSEXUALITY	IMMUNOCOMPROMISED	ZOONOTIC
Bacteria						
Enteropathogenic *Escherichia coli*	–	+	+	~–	–	–
Enterotoxigenic *E. coli*	+	–	–	–	–	–
Enteroinvasive *E. coli*	–	–	–	–	–	–
Shiga-toxin producing *E. coli*	+	–	–	–	–	–
Enteroaggregative *E. coli*	–	+	–	–	+	–
Nontyphoid *Salmonella*	–	–	+	–	–	+
Salmonella typhi	+	–	–	–	–	–
Shigella species	–	+	–	+	–	–
Campylobacter species	+	+	–	+	–	+
Vibrio cholerae	+	–	–	–	–	–
Yersinia enterocolitica	–	–	–	–	–	+
Aeromonas species	+	–	–	–	–	–
Plesiomonas shigelloides	–	–	–	–	–	–
Clostridium difficile	–	–	+	–	–	–
Viruses						
Norovirus	+	–	–	–	–	–
Rotavirus	+	+	+	–	–	–
Hepatitis A	+	–	–	–	–	–
Protozoa						
Giardia lamblia	+	+	–	–	+	+
Entamoeba histolytica	+	–	–	–	–	+
Cryptosporidium parvum	+	–	–	+	+	+
Isospora belli	–	–	–	–	+	–
Cyclospora cayetanensis	+	–	–	–	+	–
Microsporidia	–	–	–	–	+	–
Balantidium coli	–	–	–	–	–	+
Sarcocystis	–	–	–	–	–	+
Blastocystis hominis	–	–	–	–	–	–

*In industrialized areas or daycare centers.
+, association; –, no association or unknown association.

ence 5 to 15 episodes of diarrhea per year. The enteropathogens more common in infectious diarrhea during childhood are rotavirus, EPEC, enterotoxic *E. coli* (ETEC), enteroadherent *E. coli* (EAEC), *C. jejuni/coli,* and *G. lamblia* (see Table 62-1). Residents in industrialized countries, such as the United States, have only one to two bouts of diarrhea per person per year, with no differences between age groups, although complications, including death, are more common in older adults.[76,95,115]

Reservoirs of Infection. Organisms are shed in the stools during asymptomatic and symptomatic infection and for a period after the illness. Long-term shedding or chronic carrier states are reported only with typhoid fever, amebiasis, giardiasis, cryptosporidiosis, and EAEC infection. These cases may act as reservoirs for spreading infection, even in areas with low risk for infection from contaminated water. A few enteric pathogens that are zoonotic (having animal reservoirs) can increase the risk for certain persons (e.g., veterinarians, field biologists) and account for wilderness-acquired infections. These zoonotic organisms include *Salmonella, Yersinia, Campylobacter, Giardia, Balantidium coli, Entamoeba, Sarcocystis,* and *Cryptosporidium*[76,95] (see Table 62-1).

Incubation Period. Food intoxication, caused by ingestion of preformed toxins from *Staphylococcus aureus* or *Bacillus cereus,* usually has a short incubation period (2 to 7 hours), and often a common source is found to have affected multiple persons.[14,93,260] An outbreak by any enteropathogen that must first infect the intestine usually has an incubation period of 8 or more hours.

Immunocompromised Status. Immunocompromised patients, including those infected with human immunodeficiency virus (HIV), are prone to acquire infection by a wide variety of enteropathogens, to develop infectious diarrhea, and to experience relapses or reinfections. Patients with HIV and advanced acquired immunodeficiency syndrome (AIDS) often experience malabsorption and chronic diarrhea because of changes in intestinal function secondary to HIV or because of reduced immunity that allows coinfection with other enteropathogens. The agents responsible for diarrheal diseases in patients with HIV are common enteric agents, *Mycobacterium avium-intracellulare* complex, *Cryptosporidium, Giardia, Isospora, Cyclospora,* microsporidia, cytomegalovirus, herpes simplex virus, and HIV. Treatment of patients with HIV with highly active antiretroviral therapy and managing the enteric infection are associated with improved symptomatology and decreased rates of infection.

TABLE 62-2. Enteropathogens Found in Tropical and Wilderness Travel

AGENTS	TRAVEL TO DEVELOPING TROPICAL REGIONS	WILDERNESS TRAVEL IN INDUSTRIALIZED REGIONS
Bacteria		
Enteropathogenic *Escherichia coli*	Rarely	Rarely
Enterotoxigenic *E. coli*	Yes	Rarely
Enteroinvasive *E. coli*	Rarely	Rarely
Shiga toxin–producing *E. coli*	Rarely	Rarely
Enteroaggregative *E. coli*	Yes	Rarely
Salmonella species	Yes	Yes
Shigella species	Yes	Yes
Campylobacter species	Yes	Yes
Vibrio cholerae	Limited	Not currently
Yersinia enterocolitica	Rare	Limited
Aeromonas species	Yes	Yes
Plesiomonas shigelloides	Yes	Rarely
Viruses		
Norovirus	Yes	Yes
Rotavirus	Yes	Rarely
Hepatitis A virus	Yes	Yes
Protozoa		
Giardia lamblia	Yes	Yes
Entamoeba histolytica	Yes	Rarely
Cryptosporidium parvum	Yes	Yes
Isospora belli	Limited	Rarely
Cyclospora cayetanensis	Limited	Rarely
Microsporidia	Limited	Rarely
Balantidium coli	Limited	Rarely
Sarcocystis	Limited	Rarely
Blastocystis hominis	Limited	Rarely

Etiology

Enteropathogens, including bacteria, viruses, and protozoa, are the most common etiologic agents of infectious diarrhea. Fungal agents have been reported rarely. Table 62-2 lists the etiologic agents often associated with travel to developing tropical areas, or with wilderness travel in an industrialized region. Food-borne illness may consist of food "poisoning" or food "infection." In food poisoning, an intoxication results when toxins produced by bacteria are found in food in sufficient concentrations to produce symptoms. The major forms of intoxication result from *S. aureus* and *B. cereus*. A rare cause of food poisoning that results in paralysis is botulism, caused when the neurotoxin of *Clostridium botulinum* is ingested. Other food-borne pathogens are viruses, including rotavirus and small round viruses (norovirus, astrovirus), and intestinal protozoal agents, including *Giardia, Entamoeba histolytica,* and *Cryptosporidium*.

Pathophysiology

Three intestinal mechanisms lead to acute diarrhea. The most common pathophysiologic mechanism in acute infectious diarrhea is alteration of fluid and electrolyte movement from the serosal to the mucosal surface of the gut (secretory diarrhea). This alteration may occur as a result of cyclic nucleotide stimulation (as a second messenger) or by an inflammatory process that releases cytokines. The second mechanism, malabsorption or the presence of nonabsorbed substances in the lumen of the bowel, and the third, acceleration of intestinal motility, are more important in chronic forms of infectious and noninfectious diarrhea, such as tropical and nontropical sprue, Whipple's disease, scleroderma, malabsorption, irritable bowel syndrome, and inflammatory bowel disease. Table 62-3 shows the virulence factors of the most important enteric pathogens related to infectious diarrhea.[72,95]

In general, enteropathogens cause diarrhea by the first mechanism and can be subdivided into noninvasive and invasive groups. Noninvasive microorganisms primarily colonize the proximal small bowel and cause secretory diarrhea without disruption of the mucosal surface. The unformed stools are usually voluminous and rarely bloody, and high fever is unusual. The common pathogens in this group include *Vibrio cholerae,* ETEC, preformed enterotoxins, noroviruses, rotavirus, *Giardia,* and *Cryptosporidium*. Dehydration is the major complication, especially in the extremes of age, and without adequate therapy it can be followed by renal insufficiency.

Invasive pathogens, on the other hand, involve the distal ileum and colon, damaging the mucosa and eliciting an inflammatory response. Stools are typically liquid, the volume is typically small, and they may contain blood and many leukocytes. The common microorganisms in this group are *Shigella, Salmonella,* enteroinvasive *E. coli* (EIEC), Shiga-toxin–producing *E. coli* (STEC), *Yersinia enterocolitica, C. jejuni/coli, Aeromonas, Vibrio parahaemolyticus,* and *E. histolytica*. Complications include dehydration and systemic involvement, especially in children with malnutrition.[72,73]

TABLE 62-3. Bacterial Enteropathogens: Virulence Properties and Distribution

PATHOGEN	VIRULENCE PROPERTIES	DISTRIBUTION
Vibrio cholerae	Heat-labile enterotoxin	Endemic areas in Asia, Africa, and Latin America
Vibrio parahemolyticus	Invasiveness (?), enterotoxin, hemolytic toxin	Endemic areas in Asia and Latin America
Enteropathogenic *Escherichia coli*	Enteroadherence, type III secretory system	Infants, worldwide
Enterotoxigenic *E. coli*	Heat-stable and heat-labile enterotoxins, colonization factor antigens	Developing countries, tropical areas, infants, travelers
Enteroinvasive *E. coli*	Shigella-like invasiveness	Worldwide, endemic in South America and eastern Europe
Shiga toxin–producing *E. coli*	Shiga-like toxin	Beef, other vehicles in industrialized areas
Enteroaggregative *E. coli*	Enteroadherence	Infants, worldwide
Salmonella species	Cholera-like toxin, invasiveness	Worldwide
Shigella species	Shiga-like toxin, invasiveness	Worldwide
Campylobacter jejuni	Cholera-like toxin, invasiveness	Worldwide
Aeromonas species	Hemolysin, cytotoxin, enterotoxin	Worldwide, especially Thailand, Australia, Canada
Yersinia enterocolitica	Heat-stable enterotoxin, invasiveness	Worldwide, especially Canada, Scandinavia, South Africa
Clostridium difficile	Cytotoxins A and B	Worldwide
Clostridium perfringens	Preformed toxin	Worldwide
Bacillus cereus	Preformed toxin	Worldwide
Staphylococcus aureus	Preformed toxins	Worldwide

TRAVELERS' DIARRHEA

Travelers' diarrhea (TD) is the most important travel-related illness in terms of frequency and economic impact. Point of origin, destination, and host factors are the main risk determinants.[95,249] International travel is more often associated with enteric infection and diarrhea, particularly when the destination is a developing tropical region, but the same infections can be contracted domestically. The 2% to 4% rate of diarrhea for people who take short-term trips to low-endemic areas (e.g., United States, Canada, northwestern Europe, Australia, Japan) may be related to more frequent consumption of food in public restaurants, increased intake of alcohol, or stress. This rate of diarrhea increases to about 10% for travelers from these low-endemic areas to northern Mediterranean areas, China, Russia, and some Caribbean islands. This incidence increases to as high as 40% to 50% for short-term travelers from low-risk countries to high-risk countries (developing tropical and subtropical regions of Latin America, Southeast Asia, or Africa). More than 25 million persons travel each year from these industrialized countries to high-risk areas, resulting in over 7 million travelers with diarrhea.[76,95,96] Multiple episodes of diarrhea may occur on the same trip.[249] Attack rates remain high for up to 1 year,[76,95] then decrease, but not to the levels of local inhabitants. Immunity to ETEC infection, either asymptomatic or symptomatic, occurs after repeated or chronic exposure,[221] which supports the feasibility of developing a vaccine.

TD is a syndrome, not a specific disease.[76,95,249] Although any water-borne or food-borne enteropathogen may cause TD, bacteria are the most common etiologic agents among persons traveling to high-risk areas. The bacterial flora of the bowel changes rapidly after arrival in a country with high rates of TD.[3] At least 15% of travelers remain asymptomatic despite the occurrence of infection by pathogenic organisms, including ETEC and *Shigella*. However, most infected patients become ill.

Definition

TD refers to an illness contracted while traveling, although in 15% of sufferers symptoms begin after the return home.[45] Most clinical studies define TD as the passage of three or more unformed stools in a 24-hour period in association with one or more enteric symptoms, such as abdominal cramps; fever; fecal urgency; tenesmus; passage of bloody, mucoid stools; nausea; and vomiting.[76,95,249]

Etiology

Because the incidence of TD reflects in part the extent of environmental contamination with feces, the etiologic agents are pathogens causing illness in local children. The list of etiologic agents changes as laboratory techniques identify new enteropathogens (Table 62-4). Twenty years ago, specific pathogens were found in only 20% of cases.[181,182] Currently, etiologic agents can be identified in up to 80% of TD episodes.[95,249] In most studies, however, causative pathogens are not identified in 20% to 40% of cases. In most of these cases, antimicrobial therapy shortens illness, suggesting that this subset of diarrhea is caused by undetected bacterial pathogens.[37,79,99,119] Overall, the major etiologic agents and their frequency of isolation are remarkably similar when one region of the world is compared with another.

ETEC has proved to be the most common cause of TD worldwide,[10,61,221,249,284] accounting for about one third to one half of cases. Since the previous edition of this book, EAEC has been identified as the second most common bacterial cause of TD, causing up to 30% of TD cases in some areas of the world.[5,156,169,243,317] One study has shown that the source of both types of *E. coli* is food. Viable ETEC and EAEC were found in hot sauces on the table in popular restaurants in Guadalajara, Mexico.[6] *Shigella* and *Campylobacter* species cause around 20% of illness. Other causes of TD include *Salmonella* (4% to 5% of cases), *Vibrio*, *Aeromonas*, *Plesiomonas*, viruses

TABLE 62-4. Major Pathogens in Travelers' Diarrhea (Travel to Developing Tropical Regions)

AGENT	FREQUENCY (%)
Bacteria	50–80
Enterotoxigenic *Escherichia coli*	5–50
Enteroaggregative *E. coli*	5–30
Salmonella species	1–15
Shigella species	1–15
Campylobacter jejuni	1–30
Aeromonas species	0–10
Plesiomonas shigelloides	0–5
Other	0–5
Viruses	0–20
Rotavirus	0–20
Norovirus	1–20
Protozoa	1–5
Giardia lamblia	0–5
Entamoeba histolytica	0–5
Cryptosporidium parvum	0–5
Unknown	10–40

TABLE 62-5. Pathophysiologic Syndromes in Diarrheal Disease

SYNDROME	AGENT
Acute watery diarrhea	Any agent, especially with toxin-mediated diseases (e.g., enterotoxigenic *Escherichia coli, Vibrio cholerae*)
Febrile dysentery	*Shigella, Campylobacter jejuni, Salmonella,* enteroinvasive *E. coli, Aeromonas* species, *Vibrio* species, *Yersinia enterocolitica, Entamoeba histolytica,* inflammatory bowel disease
Vomiting (as predominant symptom)	Viral agents, preformed toxins of *Staphylococcus aureus* or *Bacillus cereus*
Persistent diarrhea (>14 days)	Protozoa, small bowel bacterial overgrowth, inflammatory or invasive enteropathogens (*Shigella,* enteroaggregative *E. coli*)
Chronic diarrhea (>30 days)	Small bowel injury, inflammatory bowel disease, irritable bowel syndrome (postinfectious), Brainerd diarrhea

(10%),[44] and parasites (2% to 4%). Specific pathogens may predominate at a particular time or location. ETEC and EAEC are more common in semitropical countries, including Mexico, Guatemala, Goa, Jamaica, Kenya, and Morocco, and during rainy summer seasons, and they occur less often in drier winters.[5,211,243,317]

Clinical Syndromes

Table 62-5 outlines the major syndromes in patients with enteric infection. The typical clinical syndrome experienced by travelers with diarrhea secondary to the major infectious causes (e.g., ETEC) begins abruptly with watery diarrhea and abdominal cramping. Most cases are mild, consisting of passage of one to two unformed stools per day and associated with symptoms that are tolerable and do not interfere with normal activities. Approximately 30% of affected persons experience moderately severe illness, with three to five unformed stools per day and distressing symptoms that force a change in activities or itinerary. Only 10% to 20% of persons with TD experience severe illness with more than five unformed stools passed per day, incapacitating symptoms that force confinement to bed, or any number of unformed stools with concomitant fever and dysentery.[95,249] Only 4% of persons with TD consult a local physician, and less than 1% are admitted to a local hospital while traveling. Approximately one third of travelers are confined to bed or need to alter their travel plans when a diarrheal illness develops. Although the average duration of diarrhea is 3 to 4 days, 50% of cases resolve within 48 hours, 8% to 15% last longer than 1 week, and 1% to 3% last 1 month or longer. Recently, several studies have shown that infectious diarrhea might trigger irritable bowel syndrome. Up to 10% of subjects with TD developed persistent or chronic gastrointestinal (GI) symptoms.[159,236,244,318] TD is rarely life-threatening.

Clinical Examination

The etiologic organism of TD cannot be reliably identified on the basis of clinical manifestations alone, because illnesses caused by different microorganisms share similar clinical features.[65,95,225,249,295] Although noninvasive organisms rarely cause dysentery, invasive organisms often cause watery diarrhea without dysentery, or a sequential illness beginning with watery diarrhea and progressing to bloody dysentery. If multiple people acquire the illness shortly after eating a shared meal, food poisoning caused by ingestion of preformed toxins in food should be suspected, especially if the illness has a short incubation period (8 hours or less), predominant vomiting, and resolution within 24 hours.

Investigators have studied whether clinical factors can reliably be used to predict which persons will have a positive stool culture.[65,225,295] Bacterial pathogens are suspected when the sufferer has a large number (more than six) of stools per day, has a fever, and has had the ailment for more than 24 hours but less than 1 week. Regardless of the clinical similarities of enteropathogens causing TD, certain differences exist, with distinct clinical findings. When symptoms last more than 1 to 2 weeks, a protozoal etiology, such as *Giardia,* an amoeba, or *Cryptosporidium,* is suggested.

Dehydration. An important part of the initial assessment is to measure the level of hydration, which includes a determination of vital signs, orthostatic pulse and blood pressure, mental status, skin turgor, hydration of mucous membranes, and urine output. Dehydration is most common in pediatric and elder populations.

Fever. Fever is a reaction to an intestinal inflammatory process. High fever suggests a pathogen has invaded the intestinal mucosa, and classically this has meant bacterial enteropathogens such as *Shigella, Salmonella,* and *C. jejuni/coli.* Fever can also be produced by strains of EIEC, *V. parahaemolyticus, Aeromonas, C. difficile,* and viral pathogens.

Vomiting. Vomiting as the predominant symptom suggests food intoxication secondary to enterotoxin produced by *S. aureus, B. cereus,* or *Clostridium perfringens,* or gastroenteritis

TABLE 62-6. Indications for Laboratory Testing in Diarrheal Diseases, and Possible Diagnosis

LABORATORY TEST	INDICATION	DIAGNOSIS/AGENT
Fecal leukocytes or fecal lactoferrin	Moderate to severe cases	Diffuse colonic inflammation, invasive enteropathogen
Stool culture	Moderate to severe diarrhea, fever, persistent diarrhea, fecal leukocytes or lactoferrin (+), gay men	Any bacterial enteric pathogen
Blood culture	Enteric fever, sepsis	*Salmonella;* less likely *Campylobacter, Shigella, Yersinia*
Parasite examination	Persistent diarrhea, travel to specific areas, daycare centers, gay men	Any protozoan parasite
Parasite enzyme immunoassay	Persistent diarrhea, travel to specific areas, daycare centers, gay men	*Giardia, Entamoeba histolytica, Cryptosporidium*
Amebic serology	Persistent diarrhea, liver abscess	*E. histolytica*
Rotavirus antigen	Hospitalized infants (<3 years old)	Rotavirus
Clostridium difficile toxin	Antibiotic-associated diarrhea	*C. difficile*

secondary to viruses, such as rotavirus in infants or noroviruses in any age group.

Dysentery. Dysentery is defined as the passage of small-volume stools with gross blood and mucus. Common causes include *Shigella, C. jejuni, Salmonella, Aeromonas, V. parahaemolyticus, Y. enterocolitica,* EIEC, STEC, *E. histolytica,* and inflammatory bowel disease. From 30% to 50% of cases of shigellosis or campylobacteriosis are reported to involve dysenteric diarrhea in the United States. Other enteric symptoms are tenesmus (straining without passing stools) and fecal urgency (voluntary inability to delay stool evacuation by 15 minutes), which are more common with dysentery.

Abdominal Findings. An abdominal examination of persons with TD often shows mild tenderness, but there should not be signs of peritoneal irritation. A rectal examination may reveal tenderness in enterocolitis, and the victim may have painful external hemorrhoids as a result of the excess stooling.

Systemic Involvement. Some of the enteric pathogens produce both diarrheal and systemic disease, such as hemolytic-uremic syndrome related to infection with shigellosis or STEC, Reiter's syndrome or glomerulonephritis related to *Y. enterocolitica,* and typhoid fever secondary to *Salmonella typhi* and *Salmonella paratyphi.*

Laboratory Findings

Several laboratory tests are useful in evaluating patients with diarrheal disease (Table 62-6). In clinical practice, laboratory testing is reserved for illness that continues after the patient returns home. Persons with mild acute diarrhea usually need only clinical evaluation. An etiologic assessment is unnecessary, and treatment can be given empirically. Laboratory tests are reserved for persons with moderate to severe diarrhea and those with persistent diarrhea.

Fecal Leukocyte Test. The presence of fecal leukocytes is a reliable indicator or screening test for invasive and inflammatory distal GI infection. For all moderate to severe illness, this is the most rapid and useful test and the ideal screening procedure. The fecal leukocyte test should be performed on a fresh sample. A mucus strand, if available, or liquid stool is stained with a drop of dilute methylene blue and observed under a microscope. The stool can be heat-fixed and examined under oil immersion or viewed as a wet-mount preparation under a coverslip with the high-dry objective of the microscope.[138] Leukocytes are easily seen, although they can be confused with protozoal cysts. A large number of polymorphonuclear leukocytes (PMNs) per high-power field (hpf) indicates diffuse colonic inflammation (Fig. 62-1) rather than a specific etiology, but this correlates most significantly with invasive bacterial infection caused by *Shigella, Salmonella,* or *C. jejuni/coli.* Other organisms and conditions that may lead to the finding of fecal leukocytes are *C. difficile*–associated diarrhea, *Aeromonas, Y. enterocolitica, V. parahaemolyticus,* EIEC, idiopathic ulcerative colitis, and allergic colitis. Fecal leukocytes are less likely to be seen in noninvasive infections, such as diarrhea caused by ETEC, EAEC, *G. lamblia,* and viral pathogens, but they are often observed in

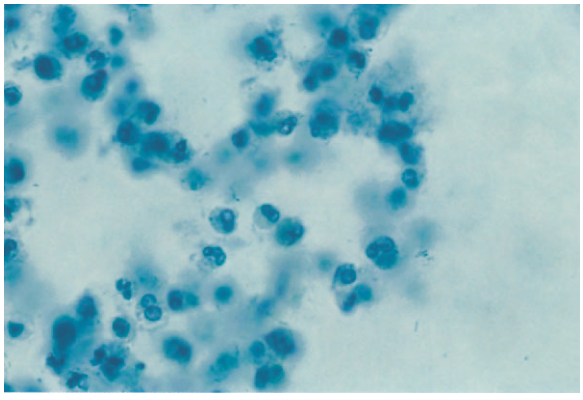


Figure 62-1. Methylene blue stain of a fecal smear from a patient with bacillary dysentery (×400). Numerous polymorphonuclear leukocytes are present, which indicates the presence of diffuse colonic inflammation.

culture-negative stools.[138] Not all patients with invasive infectious diarrhea will have leukocyte-positive stools.

Fecal Lactoferrin Test. Lactoferrin is made by neutrophils, and it can be identified by a simple immunoassay method (Leukotest, TechLab). This test does not have the limitation of requiring fresh-cup samples or an experienced technician, and apparently it is more sensitive than microscopic examination of fecal leukocytes.[47,143,216] However, it does have the limitations of cost and false-positive results in breastfeeding infants.

Stool Culture. Bacterial infection is specifically diagnosed by stool culture, but routine stool testing identifies few pathogens. A routine laboratory should be able to recover *Shigella, Salmonella,* and *Campylobacter* from a stool culture and, if specifically requested, *V. cholerae* and *V. parahaemolyticus, Aeromonas, Y. enterocolitica,* and *C. difficile*. In the United States, only about 10% of stool cultures are positive. The percentage is higher (12% for adults, about 50% for children and travelers) among patients in developing countries when research laboratories look for all the important agents, including ETEC.[248,294] The major indications for performing a stool culture are moderate to severe diarrhea, febrile and dysenteric disease, persistent diarrhea, and presence of fecal leukocytes in fecal smears.

Blood Culture. Blood cultures should be performed for all patients who are hospitalized with gastrointestinal illness or those who have enteric symptoms and high fever. Systemic infections by *S. typhi* and nontyphoid *Salmonella, Shigella, Campylobacter fetus,* and *Y. enterocolitica* may be diagnosed by blood culture.

Parasite Examination. In cases of TD, direct examination of stool samples looking for a parasite infection is less useful as a routine test than the stool culture. When microscopy is used to search for parasites, multiple samples may have to be examined to identify the causative agent. Immunologic techniques to detect antigens of protozoal parasites are more efficient and in common use for parasites that inhabit the duodenum (e.g., *Giardia, Cryptosporidium, E. histolytica,* microsporidia).[131,209] At times, intestinal parasites are better detected using a sample from duodenal aspiration or intestinal biopsy.[131,209] Performing parasitic examination is indicated for persistent diarrhea; diarrhea during or shortly after travel in mountainous areas of the United States or in Russia, Nepal, and Peru; diarrhea in someone who has regular contact with an infant daycare center; or diarrhea in a gay man or an immunocompromised person.

Identification of Enteropathogens Using Molecular Biology Tests. For almost 2 decades, there have been ongoing efforts to develop and test different nucleic acid–based techniques for the purposes of diagnosis, molecular epidemiology, and identification of virulence factors and intrinsic characteristics of almost all enteric pathogens. Polymerase chain reaction (PCR) and pulsed-field gel electrophoresis (PFGE) are the most successfully applied, but most of the techniques are still under investigation and are not available for clinical microbiology laboratories. PCR tests for direct detection of pathogens in stool samples have been developed for most of the bacteria (ETEC, EAEC, EIEC, STEC, *Shigella, Campylobacter*), viruses (norovirus, rotavirus) and parasites (*Cryptosporidium, Giardia, Cyclospora, Entamoeba,* and microsporidia).[10,20,25,109,111,202,226,304,329]

Rotavirus antigen testing of stool is sensitive and easy to perform. It is used to screen infants less than 3 years of age to help guide therapy (fluids without antimicrobials are given when the test is positive).[242,325]

Special Tests. For the Enterotest, a gelatin capsule is affixed to a nylon string. The free end of the string is taped to the cheek and then the capsule is swallowed. After the patient consumes a meal or after the capsule has been in place overnight, it is removed so that mucus and other intestinal secretions can be scraped off and studied for enteropathogens. It may also be useful to sample small bowel mucus to diagnose cases of typhoid fever, giardiasis, and strongyloidiasis.

A serologic diagnostic test for typhoid fever (Widal's reaction) is useful only in endemic areas, because exposure to cross-reacting gram-negative rods other than *S. typhi* can lead to false-positive serologic results in areas where typhoid fever is not common.[214] In a patient with a typhoid-like systemic illness who has taken one or more doses of an antimicrobial, culture of bone marrow aspirate may help to identify the bacteria. Antibody-specific serologic tests are now widely used for the diagnosis of invasive amebiasis.[112]

An assay for *C. difficile* toxins in stool samples, by tissue culture or by enzyme immunoassay (EIA), is indicated for diagnosing antibiotic-associated colitis. Many of the commercial serologic kit tests are easier to perform, but they detect mainly toxin A and are less sensitive than the tissue culture procedure. Infants and children may normally carry *C. difficile* toxin in the stools, negating the value of this test.[171]

Sigmoidoscopy and Colonoscopy. In selected cases, particularly cases of clinical colitis and diarrhea persisting for 14 days or longer, sigmoidoscopy or colonoscopy is used to study colonic lesions and collect samples for culture and microscopy. Mucosal changes may not be specific, except when pseudomembranes are sought. For gay men with acute diarrhea, examination of the distal colon may show evidence of proctitis (mucosal inflammation in the distal 15 cm of the colon), proctocolitis (inflammation beyond 15 cm), or enteritis.

Acute Diarrhea

Where routine laboratory evaluation is available, logical approaches to patients with acute diarrhea depend on the clinical syndrome. The illness, not the infection, should be managed, so most persons can be treated on the basis of symptoms and stool appearance. In certain situations, empiric therapy may be given without establishing an etiologic agent; in other cases, specific therapy follows laboratory confirmation of an etiologic agent (Tables 62-7 and 62-8). In patients with watery diarrhea and mild symptoms, only clinical evaluation is needed. An etiologic assessment is unnecessary, and symptomatic treatment can be given empirically. Persons with moderate to severe diarrhea, dysentery, fever, or presence of fecal leukocytes should have their stool cultured, if laboratory assessment is feasible, and should start empiric antimicrobial therapy.

Persistent and Chronic Diarrhea

Diarrhea may persist after the traveler returns home. Up to 3% of persons with TD in high-risk areas will develop persistent

TABLE 62-7. Empiric Treatment and Evaluation of Diarrhea in Adults

CLINICAL MANIFESTATIONS	RECOMMENDATIONS*
Watery diarrhea with mild symptoms (no change in itinerary)	Administer oral fluids and crackers (e.g., Saltines)
Watery diarrhea with moderate symptoms (change in itinerary but able to function)	Administer symptomatic treatment with loperamide or bismuth subsalicylate; or antimicrobial drugs† plus loperamide after passage of first unformed stool
Watery diarrhea with severe symptoms (incapacitating)	Perform stool culture and fecal leukocyte or lactoferrin test; administer antimicrobial drugs† plus loperamide after passage of first unformed stool
Dysentery or fever	Perform stool culture and fecal leukocyte or lactoferrin test; administer antimicrobial drugs†; no loperamide
Persistent diarrhea (>14 days)	Perform stool culture and parasite examination; consider empiric trial with metronidazole
Vomiting, minimal diarrhea	Administer bismuth subsalicylate
Diarrhea in pregnant women	Evaluation as above; administer fluids and electrolytes; fluoroquinolones not recommended; consider attapulgite; consider azithromycin or rifaximin; no fluoroquinolones

*Treatment may be started during travel in absence of facilities for evaluation.
†Fluoroquinolones (norfloxacin, ciprofloxacin, or levofloxacin), azithromycin, or rifaximin recommended in adults.

TABLE 62-8. Nonspecific Drugs for Therapy in Adults

AGENT	THERAPEUTIC DOSAGE
Attapulgite	Initially 3 g, then 3 g after each loose stool or every 2 hours (not to exceed 9 g/day); should be safe during pregnancy and childhood. (Available in 600-mg tabs, or liquid 600 mg/tsp)
Loperamide	Initially 4 mg, then 2 mg after each loose stool (not to exceed 8 to 16 mg/day); do not use in dysenteric diarrhea.
Bismuth subsalicylate	30 mL or two 262-mg tablets every 30 min for 8 doses; may repeat on day 2.

diarrhea. *Persistent* diarrhea is defined as illness lasting 14 days or longer, whereas diarrhea is considered *chronic* when the illness has lasted 30 days or longer.[74,95,249] The etiology of persistent or chronic diarrhea often differs from that of acute diarrhea. Important causes of persistent diarrhea include (1) protozoal parasitic agents (*Giardia, Cryptosporidium, Cyclospora,* and *E. histolytica*), (2) bacterial infection (*Salmonella, Shigella, Campylobacter,* and *Y. enterocolitica*), (3) lactase deficiency induced by a small bowel pathogen (*Giardia,* rotavirus, or norovirus), and (4) a small bowel bacterial overgrowth syndrome secondary to small bowel motility inhibition (as a result of enteric infection) or secondary to antimicrobial use. Occasionally, other parasitic enteric infections can cause more persistent illness. These include *Strongyloides stercoralis, Trichuris trichiura,* and severe infection by *Necator americanus* or *Ancylostoma duodenale*. In rare cases, more protracted diarrhea may be a prominent symptom in persons with schistosomiasis, *Plasmodium falciparum* malaria, leishmaniasis, or African trypanosomiasis.[74,95,231,249]

When chronic diarrhea occurs, the following possibilities should also be considered:

- Postinfectious irritable bowel syndrome has recently been noted by a number of authors.[159,236,244,291,318] Approximately 10% of travelers who develop TD could develop chronic gastrointestinal symptoms, either completely new or a reactivation of a previous disorder. An underlying condition such as inflammatory bowel disease, irritable bowel syndrome, or celiac sprue may worsen after an episode of acute enteritis.
- After eradication of microbial pathogens, bowel habits may not return to normal for several weeks. Postdysenteric colitis resembling ulcerative colitis occasionally follows infection with invasive pathogens, especially infection caused by *E. histolytica*. This could represent slow repair of damage to the intestinal mucosa.
- Chronic parasitic infection with *Giardia, Cryptosporidium, Isospora, Cyclospora,* microsporidia, or *Entamoeba* may produce a chronic intestinal process and produce persistent diarrhea. Postinfective malabsorption can persist for weeks to months after acute diarrhea; it is especially common after giardiasis.[74,96,249]
- A poorly defined condition, tropical sprue, may explain prolonged diarrhea in a traveler. Onset usually follows an episode of acute enteritis and is associated with substandard hygiene and longer stays. The cause may involve small bowel bacterial overgrowth, because small bowel incubation may yield a heavy growth of bacteria, and patients often respond to antimicrobial therapy.
- Brainerd diarrhea, named after a community outbreak in Brainerd, Minnesota, may follow consumption of raw (unpasteurized) milk[239] or untreated water.[246] There is no diagnostic test or therapy, and the diagnosis is suspected on the basis of the epidemiologic history (exposure to unpasteurized milk or untreated water just before onset of illness).

Evaluation of persistent or chronic diarrhea in travelers should begin with diagnostic tests for conventional bacterial pathogens in stools and at least three sequential evaluations for parasites in stools. Dietary modification in all cases should include avoidance of lactose. Treatment should be specific, following the results of the microbiologic tests. Because most of these chronic forms of diarrhea are self-limiting, it is unwise to keep treating these patients with multiple antibiotics, which only alters the gut ecology and encourages diarrhea. An empiric trial with metronidazole is an option if all tests are negative (see Table 62-7). If stools contain leukocytes, sigmoidoscopy or colonoscopy should be performed, along with empiric treatment for *Shigella* or *Campylobacter* infection. If there are no leukocytes, duodenal mucus should be examined for *G. lamblia,* followed by empirical treatment for *Giardia,* if metronidazole has

not already been given. The next steps are tests for malabsorption and biopsy of the small bowel mucosa.

Treatment

In all cases of diarrhea, fluid and electrolyte replacement should be the primary therapy. Outpatient treatment with instructions for oral rehydration can be used in the vast majority of adults and children. Significant dehydration from diarrhea in travelers is unusual. Treatment with intravenous (IV) fluids is indicated for the following:

- Patients with hemodynamic decompensation (hypotension)
- Inability to retain oral fluids
- Systemic compromise (high fever and toxicity)
- Moderate toxicity or dehydration and a severe underlying disease
- Patients at extremes of age

Some patients may benefit from symptomatic therapy, and others may receive empiric antimicrobial therapy (see Table 62-7). The main goal of the therapy, such as an antimotility drug or an antimicrobial agent, is to attenuate the severity and duration of the diarrhea and concomitant symptoms.

Diet and Lifestyle. Supplemental nutrition is beneficial (essential in undernourished populations) and can be given as soon as fluid deficit losses are replaced, usually after the first 4 hours. During acute diarrheal disease, the intestinal tract cannot process complex dietary products, so patients are often told to avoid solid foods. As stooling decreases and appetite improves, staple foods, such as cereals, bananas, crackers, toasts, lentils, potatoes, and other cooked vegetables, are well tolerated and can be gradually added to the diet to facilitate enterocyte renewal, with progression to white meats, fruits, and vegetables. Dairy products and red meats are recommended only after diarrhea has resolved, usually after 2 to 3 days. Only foods and drinks that prolong diarrhea or increase intestinal motility, such as those that contain lactose, caffeine, alcohol, high fiber, and fats, should be avoided. Breastfeeding of infants should be continued or should be resumed as soon as possible.[71,76,95,96,296] Patients with TD should avoid excessive physical therapy to reduce the risk of dehydration.

Fluid Treatment. The major cause of morbidity and mortality from acute diarrheal disease is depletion of body water and electrolytes. Rehydration is an essential part of therapy, especially in the extremes of age and during pregnancy. Most patients with TD do not become dehydrated, and hydration can be maintained by ingesting fluids, such as sodas, juices, soup, and potable water, in conjunction with a source of electrolytes (e.g., salted crackers).[46,76,95,96]

The most significant advance in the therapy of diarrhea in the past 50 years has been development of the oral rehydration concept. Oral rehydration solution (ORS) was first developed for treatment of cholera and has saved countless lives, primarily those of children. ORS precludes extensive use of scarce and expensive IV fluids in developing countries; its use is the cornerstone of the World Health Organization (WHO) program to combat diarrheal diseases.[46,76,95,96,132] The discovery that glucose-enhanced intestinal absorption of sodium remains intact despite active diarrhea or vomiting was the key to development of ORS.[211,273] Other electrolytes are also absorbed nonselectively when ORS is administered.

Watery diarrhea, often caused by release of an enterotoxin, has an electrolyte composition similar to that of plasma, varying somewhat with the type of infection and age of the patient. The formula packaged and promoted by the WHO and the United Nations International Children's Emergency Fund (UNICEF) contains powder to be mixed with 1 L of disinfected water, with the following resultant concentrations: sodium, 90 mEq; potassium, 20 mEq; chloride, 80 mEq; bicarbonate, 30 mEq; and glucose, 111 mmol. Newer formulations use trisodium citrate instead of sodium bicarbonate, and complex carbohydrates instead of glucose. Cereal-based products are also available. Although this concentration of electrolytes is ideal for managing the purging diarrhea associated with cholera and other dehydrating forms of diarrhea, most TD can be adequately managed with readily available soft and sports drinks, fruit juices, or salt solutions, taken with salted crackers and the foods listed earlier.[46,76,96]

Fluid status in the field must be guided by physical signs related to hydration, including pulse, mucous membranes, skin turgor, and urine output. Urine color and volume are excellent measures. For travelers in the wilderness or tropics, fluid replacement must equal basic needs plus volume of diarrhea plus estimated sweat loss.

Nonspecific Therapy. Symptomatic medications are useful for treatment of mild to moderate diarrhea, as they decrease symptoms and allow patients to return more quickly to normal activities (see Tables 62-7 and 62-8). Nonantibiotic therapies that may be used in addition to fluids are best classified by their effects on pathophysiologic mechanisms.

Probiotics. *Lactobacillus* preparations and yogurt are safe, but evidence is insufficient to establish their value in the therapy of acute diarrhea.[46,76,96,142,309]

Adsorbents. Adsorbent agents bind nonspecifically to water and other intraluminal material, including bacteria and toxins, and potentially to other medications such as antibiotics. The most common medication in this group is attapulgite (see Table 62-8 for dosages), a nonabsorbable magnesium aluminum silicate that is more active than the combination of kaolin and pectin.[37] By adsorbing water, these agents give stools more form or consistency but do not decrease stool frequency, cramps, or duration of illness. They are reliable and should be safe in all persons, although adsorbents are not approved for use in young infants and pregnant women.[270]

Antimotility Drugs. Narcotic analogs related to opiates are the major antimotility drugs. In addition to slowing intestinal motility, these drugs alter water and electrolyte transport, probably affecting both secretion and absorption.[46,76,95,96] Compared with placebo, antimotility drugs reduce the number of stools passed and the duration of illness by about 80% during their administration.[77,83] The most frequently used product is loperamide (Imodium), 4 mg initially, followed by 2 mg after each unformed stool, not to exceed 8 to 16 mg/day. Loperamide also has a weak antisecretory effect through inhibition of intestinal calmodulin. Diphenoxylate with atropine (Lomotil) is less expensive than loperamide but has greater central opiate effects, a danger in case of accidental overdose by a child, and more side effects without antidiarrheal benefits because of the atropine, which is

added only to prevent overdoses. Tincture of opium or paregoric opium preparations are rapidly and equally effective and offer a modest relief of symptoms.

Antimotility drugs should never be used alone in patients who have dysenteric or febrile diarrhea, because inhibition of gut motility may facilitate intestinal infection by invasive bacterial enteropathogens.[85] However, this theoretically deleterious effect does not appear to be an issue when loperamide is used concurrently with an effective antimicrobial agent.[97,99,300] Antimotility drugs should not be given to children younger than 3 years because of the danger of central nervous system (CNS) depression. They are recommended for only up to 48 hours in acute diarrhea.

Antisecretory Drugs. Because increased secretion of water and electrolytes is the major physiologic derangement in acute watery diarrhea, therapy aimed at this effect is appealing. Although aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs) inhibit secretion, their usefulness is limited, primarily because of mucosal toxicity.[37,95] The salicylate moiety of bismuth subsalicylate reduces the number of stools passed and the duration of diarrhea by about 50%, primarily by blocking the effect of the enterotoxin on the intestinal mucosa.[83] Bismuth subsalicylate also has antimicrobial and anti-inflammatory properties. New compounds are being developed that have antisecretory properties without motility effects.[80]

Antimicrobial Therapy. Although most enteric infections do not require antibiotics, empiric antimicrobial therapy is indicated in acute TD and febrile, dysenteric illness because of the high frequency of bacteria as etiologic agents.[96] Travelers with acute diarrhea and mild symptomatology usually do not need empiric antimicrobial therapy and could be treated with oral fluids and saltine crackers. Those travelers with acute diarrhea and moderate symptoms (serious enough to change their itinerary) could be treated with antimicrobial empiric therapy or symptomatic therapy with loperamide or bismuth subsalicylate. Finally, those travelers with severe and incapacitating symptoms, or with dysentery, should be treated with empiric antimicrobial therapy immediately after the first passage of unformed stool (together with loperamide only if there is no evidence of dysentery) (see Table 62-7).[7,37,76,96]

Therapies for specific infections are discussed later in the relevant sections (Table 62-9). At times, treatment is indicated regardless of symptoms to prevent person-to-person spread (e.g., for food handlers, river guides, daycare workers) or to eradicate pathogenic strains and prevent conversion from asymptomatic to symptomatic illness (e.g., *E. histolytica*).[36,258,292]

Fluoroquinolones, azithromycin, and the newly approved rifaximin have adequate activity against most of the bacterial enteric pathogens to be considered useful for empiric therapy.[7,81,88,293] Fluoroquinolones,[7,95] including those evaluated in TD (norfloxacin, ciprofloxacin, ofloxacin, levofloxacin), represent the treatments of choice for TD when individuals are traveling to areas where resistance in *Campylobacter* strains is not prevalent or has not been determined. The main advantage of the quinolones is the high degree of in vitro activity against virtually all bacterial etiologic agents.[79,99] Ciprofloxacin (500 mg twice a day) was as effective for treating TD as the combination of trimethoprim and sulfamethoxazole (TMP/SMX) in an area where trimethoprim resistance was unusual.[99]

TABLE 62-9. Antimicrobial Therapy for Diarrhea in Adults

DIAGNOSIS	RECOMMENDATION
Empiric Therapy in Bacteriologically Unconfirmed Disease	
Travelers' diarrhea	Rifaximin 200 mg tid or 400 mg bid for 3 days; norfloxacin 400 mg bid, ciprofloxacin 500 mg bid or levofloxacin 500 mg qd for 1 to 3 days; or azithromycin 500–1000 mg single dose
Febrile and/or dysenteric disease	Norfloxacin 400 mg bid, ciprofloxacin 500 mg bid, levofloxacin 500 mg qd for 3 days; or azithromycin 500–1000 mg qd for 3 days
Persistent diarrhea	Consider a trial with metronidazole 250 mg qid for 7 days
Oraganism-Specific Therapy in Laboratory-Confirmed Diarrhea	
Enterotoxigenic and enteroaggregative *Escherichia coli* diarrhea	Rifaximin 200 mg tid or 400 mg bid for 3 days; ciprofloxacin 1000 mg single dose or 500 mg bid for 1 to 3 days; norfloxacin 400 mg bid or levofloxacin 500 mg qd for 1 to 3 days; or azithromycin 500–1000 mg single dose
Cholera	Ciprofloxacin 1000 mg single dose or 500 mg bid for 3 days; norfloxacin 400 mg bid or levofloxacin 500 mg qd for 3 days or doxycycline 300 mg single dose
Salmonellosis (typhoid fever or systemic infection)	Norfloxacin 400 mg bid, ciprofloxacin 500 mg bid, or levofloxacin 500 mg qd for 7 to 10 days; in patients with underlying disease or immunocompromised persons
Salmonellosis (intestinal nontyphoid salmonellosis without systemic infection)	Antimicrobial therapy controversial (see text)
Shigellosis	Norfloxacin 400 mg bid, ciprofloxacin 500 mg bid, levofloxacin 500 mg qd for 3 days
Campylobacteriosis	Erythromycin 500 mg qid for 5 days; azithromycin 500–1000 mg qd, norfloxacin 400 mg bid, ciprofloxacin 500 mg bid or levofloxacin 500 mg qd for 3 days
Enteropathogenic *Escherichia coli* diarrhea	Unclear if antimicrobial therapy is necessary
Clostridium difficile colitis	Metronidazole 250 mg qid to 500 mg tid; or vancomycin 125 mg qid for 7 to 14 days

bid, twice daily; qd, daily; qid, four times daily; tid, three times daily.

TMP/SMX (160/800 mg) and trimethoprim (200 mg) twice a day for 5 days were equally effective in reducing the number of unformed stools, duration of illness, and abdominal symptoms (compared with placebo).[84] Reduced duration of illness was reported in infections caused by ETEC or *Shigella* and also in the group without identifiable pathogens. Recently, TMP/SMX is less recommended because of increasing in vitro resistance to this antimicrobial agent in several areas of the world.[7,122]

Azithromycin has been found as a good alternative for the treatment of acute TD. In a randomized, double-blind study, azithromycin (1000 mg, single dose) has been found to be as safe and effective as levofloxacin (500 mg, single dose) in the treatment of TD in Mexico.[4] Although it remains to be tested in adequate clinical trials, 500 mg of azithromycin daily for 1 to 3 days should also be safe and effective.

Rifaximin (200 mg three times a day) was approved in 2004 by the U.S. Food and Drug Administration (FDA) for the treatment of TD caused by noninvasive *E. coli* in patients older than 12 years. This approval was based on three randomized, double-blind clinical trials of rifaximin in travelers with diarrhea visiting Guatemala, Mexico, Jamaica, or Kenya[81,88,293] that showed reduction in the duration of postenrollment diarrhea and a good safety profile, mainly because it is virtually nonabsorbed after oral administration.[129,153,168] In the first study, rifaximin was shown to be more active than TMP/SMX,[81] although not with statistical significance. In the second study, rifaximin was comparable to ciprofloxacin.[88] In the last multicenter study, two different dosages of rifaximin were significantly better than placebo, with similar rates of nonserious adverse events.[293]

Azithromycin alone in pediatric dosages (10 mg/kg/day for first dose, follow by 5 mg/kg/day for second and third doses) is the treatment of choice in children. TMP/SMX plus a macrolide or nalidixic acid was previously recommended, but currently there is reported resistance to all these antimicrobial agents. Although fluoroquinolones are not yet approved to be used in children, ciprofloxacin has been used in children with cystic fibrosis without permanent cartilage damage to the joints. A short course of a fluoroquinolone or rifaximin may be considered to be safe and efficacious in children.[189,269]

Travelers to high-risk regions should carry with them an antibacterial drug for treatment of bacterial diarrhea. A drug such as loperamide may also be included for immediate relief of symptoms. If both drugs are employed in acute cases of TD, persons should be instructed to take the loperamide only if they have no fever and are not passing grossly bloody stools.[97,98,300] Loperamide induces more rapid relief of symptoms, whereas the antimicrobial effects the cure. The duration of antimicrobials needed in TD appears to be short. Many patients respond to single-dose treatment, and no person needs more than 3 days of treatment.[4,79,97]

In cases of dysenteric diarrhea, the same antimicrobial regimen is given for 3 full days. Empiric therapy should be with fluoroquinolones in adults, and with TMP/SMX plus a macrolide or nalidixic acid in children. Azithromycin is currently being studied as an alternative antimicrobial agent. Rifaximin, because of its lack of absorption and systemic activity, has not been approved when TD is caused by invasive pathogens.[153]

Campylobacter strains are more resistant to regularly used antibiotics, including ciprofloxacin and rifaximin. Azithromycin is the preferred agent for this pathogen, which is found more commonly in Asia but seen in all high-risk areas. Viruses and parasites are also not successfully treated with antibacterial drugs. Antibiotics should not be continued in the face of persistent or worsening diarrhea, because these agents may be responsible for the illness.

Prevention and Prophylaxis

Dietary Precautions. Food and water transmit the pathogens that cause infectious diarrhea and TD.[6,7,76,95,96,100] When diarrhea occurs, however, the exact source cannot be determined. It is clear that education can play an important role in prevention of TD,[16] but dietary habits usually cannot be rigidly controlled. Food in developing countries is often contaminated with fecal coliforms and enteropathogens.[6] *V. cholerae* remains viable for 1 to 3 weeks in food,[107] and *Salmonella* can survive 2 to 14 days in water or in the environment in a desiccated state.[100]

Risk of illness is lowest when most of the meals are self-prepared and eaten in a private home, it is intermediate when food is consumed at public restaurants, and it is highest when food is obtained from street vendors.[27,100] The following standard dietary recommendations for prevention are based more on known potential vehicles for transmission of illness than on strong evidence, because most of the studies evaluating risk have found little correlation between routine precautions and the presence of diarrhea[95,100]:

- Avoid tap water, ice made from untreated water, and suspect bottled water. Bottled and carbonated drinks, beer, and wine are probably safe. Boiled or otherwise disinfected water is safe. Tap water in high-risk countries is difficult to implicate in TD, but it has been shown to contain enteric bacteria and pathogenic viruses and parasites.[100,209] Bottled carbonated beverages are considered safe because of the antibacterial effects of the low acidity. Alcohol in mixed drinks does not disinfect contaminated ice cubes, but bottled beer and wine are safe to consume. Most enteric organisms can survive freezing and melting in common drinks, so ice is not considered safe unless made from treated or previously boiled water. Ice in block form is often handled with unsanitary methods.[100]
- Avoid unpasteurized dairy products. These may be the source of infection with *Salmonella, Campylobacter, Brucella, Listeria monocytogenes, Mycobacterium tuberculosis,* and others.[253]
- Avoid raw meat and vegetables. Raw vegetables in salads may be contaminated by human waste that was used as fertilization, or by washing in contaminated water.[100] Anything that can be peeled or have the surface removed is safe. Fruits and leafy vegetables can be disinfected by immersion and washing in iodinated water or by exposure to boiling water for 30 seconds. Raw seafood, including that in traditional dishes such as ceviche and sashimi, has been associated with increased risk of TD. Shellfish concentrate enteric organisms from contaminated water and can carry hepatitis A, noroviruses, *Aeromonas hydrophila, Y. enterocolitica, V. cholerae,* and *V. parahaemolyticus*. Raw fish can carry parasites such as *Anisakis simplex, Clonorchis sinensis,* and *Metagonimus yokogawai*. Raw meat is a source of *Salmonella* and *Campylobacter* and the vehicle for *Trichinella, Taenia saginata,* and *Taenia solium* (beef or pork tapeworm), and *Sarcocystis*. Although adequate cooking kills all microorganisms and parasites, if food is left at room temperature and recontaminated before serving, it can incubate *Salmonella, Shigella,* or ETEC and EAEC. Food served on an airplane, train, boat, or bus has probably been catered in the country of origin. Problems of food hygiene pertaining to these forms of public transportation may be related to employee handling of food even in the United States.

Safe foods are those served steaming hot, dry foods such as bread, freshly cooked food, foods that have a high sugar content (e.g., syrups, jellies), and fruits that can be peeled.[100]

TABLE 62-10. Prophylactic Medications for Prevention of Traveler's Diarrhea*

AGENT	PROTECTIVE EFFICACY	PROPHYLACTIC DOSE	COMMENT
Bismuth subsalicylate	65%	Two 262-mg tablets before meals and at bedtime	Safe; temporary darkening of stools and tongue
Fluoroquinolones	90%	Norfloxacin 400 mg, ciprofloxacin 500 mg, or levofloxacin 500 mg once a day	Side effects, increased resistance
Rifaximin	70%–80%	Rifaximin 200 mg once or twice a day with meals	Safe, nonabsorbable, no increased resistance; should be considered the standard agent for prophylaxis during high-risk travel

*Not generally recommended for travelers; used only in special situations (see text) and for no longer than 3 weeks.

Prophylactic Medication. Traditionally, chemoprophylaxis was an alternative for certain people making critical trips or for travelers with underlying medical conditions. Preventive drugs are generally recommended to be used for 3 weeks or less and will require a prescription from a physician. In past studies, 10% to 25% of European travelers to high-risk areas and up to one third of U.S. travelers to Mexico used to take prophylactic medication to prevent TD.[100,182] Compared with empiric therapy with a single dose of an antimicrobial agent and loperamide, chemoprophylaxis is cost effective only when its use does not exceed a few days[100] (Table 62-10).

Several nonantimicrobial agents have been studied for prevention of TD, with some found to be minimally effective. Lactobacilli have been tested on the assumption that they are safe and favorably modify intestinal flora, but they did not invariably reduce the incidence of TD and provided protective efficacy only up to 47%.[142] Antimotility drugs, such as loperamide, have adverse effects when used for prophylaxis.[100] Of the nonantibiotic drugs, only bismuth subsalicylate (BSS), the active ingredient of Pepto-Bismol, has been shown by controlled studies to offer reasonable protection and safety. Several studies with volunteers and in the field have demonstrated that the use of BSS gives a protection rate from 40% to 77%,[78,100] with fewer abdominal symptoms. Because the volume of the liquid preparation is quite large, BSS in tablet form was also evaluated. The currently recommended dosage of BSS is two tablets four times a day (2.1 g/day).[78,96,100] Mild side effects include constipation, nausea, tinnitus, and temporarily blackened tongue or stools. In areas where doxycycline is used for malaria prevention, concurrent BSS should be avoided because it may bind to the antimicrobial and prevent absorption.[100] Ninety percent of the salicylate in the liquid form of BSS is absorbed and excreted in the urine of children.[78] Whether this salicylate cross-reacts with aspirin is unknown. However, BSS should not be used by someone with a history of aspirin allergy. Caution is recommended in small children, children with chickenpox or influenza (because of the potential risk of developing Reye's syndrome), patients with gout or renal insufficiency, and persons taking anticoagulants, probenecid, methotrexate, or other aspirin-containing products. BSS is not approved for children younger than 2 years, and it is not recommended as prophylaxis for more than 3 weeks. The precise mechanism by which BSS prevents diarrhea is still unknown. Salicylate released during dissociation in the stomach exhibits antisecretory activity after exposure to bacterial enterotoxin on intestinal mucosa, and bismuth salts have antimicrobial activity.[78] Adherence of bacteria to intestinal mucosa may be affected.

Since the first studies in the 1950s, a protective effect of antimicrobials in TD has been demonstrated. Several antimicrobial agents are highly effective in preventing TD when given over short periods to people at risk. Protection levels of 80% to 90% have been found with antimicrobial prophylaxis, provided that enteropathogens in the area were susceptible to the agent under investigation.[96,100] The most experience has been obtained with doxycycline, TMP/SMX, and the fluoroquinolones. Other antimicrobials (streptomycin and sulfonamides, erythromycin, mecillinam) have shown significant protection but have not been well studied.[96,221] Studies of U.S. students in Mexico taking trimethoprim (160 mg) and sulfamethoxazole (800 mg) twice daily for 3 weeks or once daily for 2 weeks demonstrated 71% and 95% protection, respectively.[82,84] The fluoroquinolones (e.g., ciprofloxacin, ofloxacin, norfloxacin, pefloxacin, fleroxacin, levofloxacin) have been shown to be highly protective when employed as prophylactic agents. Because of the emergence of resistance among enteropathogens to tetracyclines, doxycycline can no longer be recommended for prophylaxis. Similarly, TMP/SMX resistance has been reported in many regions of the developing world.[100] The effect of the antibiotics that were evaluated lasted only as long as the drug was continued. Subjects who remained in a high-risk area experienced an increased incidence of diarrhea during the week after cessation of prophylaxis.[74,100]

Despite dramatic protection against diarrhea, investigators in general do not recommend widespread use of these absorbed medications for prophylaxis by travelers for the following reasons:

- *Side effects.* These include GI symptoms, photosensitivity, and other cutaneous eruptions and reactions. Furthermore, fluoroquinolones are not licensed for use in pregnant women and children. With larger numbers of people using these drugs, more serious side effects (e.g., Stevens-Johnson syndrome, hemolytic or aplastic anemia, antibiotic-associated colitis, anaphylaxis) will undoubtedly result.
- *Alteration of normal bacterial flora.* Broad-spectrum antimicrobials may increase risk of infection with other antibiotic-resistant bacteria. Severe pseudomembranous colitis caused by colonic overgrowth with *Clostridium difficile* has occurred after therapy with most antibiotics. Vaginal candidiasis and GI side effects, including diarrhea, are common with antibiotic therapy. Changes in anaerobic flora can cause long-term alterations in the metabolism of bile acids and pancreatic enzymes, although the clinical effects are unknown.

- *Development of antimicrobial resistance.* Overuse of antimicrobial agents increases the prevalence of resistant strains.[91,100,125,282]
- *False sense of security.* Travelers taking antibiotics may relax their vigilance of dietary precautions and increase their risk of acquiring enteric infections.
- *High cost of antimicrobials.*
- *Rapid effectiveness of presumptive therapy.* The result is that the illness is often limited to 12 to 24 hours.

Although the consensus is most travelers should not employ absorbed drugs for prophylaxis, this approach may be appropriate for some travelers.[80,96,100] Potential candidates would be residents of a low-risk country going to a high-risk area for short stays, who have one or more of the following conditions or requirements:

- An underlying illness that increases the risk of enteric infection or morbidity, such as gastric achlorhydria (from surgery or taking proton pump inhibitors), AIDS, inflammatory bowel disease, diabetes (involving insulin treatment), or a cardiac, renal, or CNS disorder.
- An itinerary that is so rigid and critical to the overall mission that travelers would not tolerate even minor schedule changes caused by illness.
- Travelers who prefer prophylaxis after hearing the pros and cons of the approach.

These recommendations were currently challenged by the new nonabsorbed rifaximin. This antimicrobial would be ideal for prophylaxis because of its activity against most enteric pathogens, its safety profile, its lack of absorption and systemic effects, and its limited medical indications. In one recent randomized, double-blind, placebo-controlled study, data support the idea of using rifaximin for the prevention of TD. Within the first 72 hours of arrival in Mexico, 210 travelers were randomized to receive either placebo or one of the following three dosages of rifaximin for 2 weeks: 200 mg once a day, 200 mg twice a day, or 200 mg three times a day. Between 72% and 77% of persons who took rifaximin were free of disease during 3 weeks of follow-up, significantly better than the rate for people on placebo alone (46%).[89,168] Because of its minimal absorption and excellent safety profile, rifaximin is more likely to be used by high-risk travelers as antimicrobial chemoprophylaxis than absorbed drugs. No studies have evaluated prophylaxis of TD in young children, although they may be at higher risk for infectious diarrhea.

Immunoprophylaxis. Because of the emergence of in vitro resistance to antimicrobial agents (including the fluoroquinolones) among enteropathogens, prophylaxis with vaccines is being developed to control bacterial diarrhea. Several vaccines are under investigation for protection against rotavirus, *Shigella*, *V. cholerae*, and ETEC.[32,63,64,148,164,220,223,266] Although these vaccines would be most beneficial to residents of developing countries, they will be heavily marketed to travelers. The ideal travel vaccine would provide immunity to multiple organisms.

BACTERIAL ENTEROPATHOGENS

Escherichia coli

E. coli is the most prevalent facultative gram-negative rod in human feces. Diarrheogenic *E. coli* is a heterogeneous group of organisms that belong to one taxonomic species but have different virulence properties, epidemiologic characteristics, and clinical features. At least six groups have been characterized, based on either genotypic or phenotypic markers.[221]

Enteropathogenic *E. coli*. EPEC strains, the first of the diarrheogenic *E. coli* strains, were described between the 1920s and 1940s as causes of hospital nursery outbreaks.[221] Usually identified by serotype, they are also characterized by a localized adherence pattern to a specialized cell line (HEp-2 cells).[313] EPEC strains have worldwide distribution, and their most accepted virulence property is enterocyte attachment with selective damage to the surface without cell invasion. They induce production of a receptor (type III secretory system) that intimately interacts with the host cells, and this interaction leads to intracellular changes in the enterocyte.[50,62,124,184,191,221,307] Lactoferrin, a component of breast milk, apparently protects against EPEC infection, with loss and degradation of the type III secretory system of this bacterium.[230]

Enterotoxigenic *E. coli*. ETEC strains, first identified in the 1970s, produce one or two enterotoxins that act on the small intestine by different mechanisms and with different time responses.[221] One of these toxins is a heat-labile cholera-like toxin (LT), a high-molecular-weight protein immunologically and physiologically similar to cholera toxin. Human ETEC strains also have a low-molecular-weight, poorly antigenic toxin (ST) that is heat stable.[263] Both enterotoxins inhibit sodium reabsorption and increase secretion of anions and fluid into the intestinal lumen, resulting in secretory diarrhea without inflammatory exudate.[221] One common method for the diagnosis of ETEC is identification of specific DNA plasmid sequences using a hybridization technique.[221] Recently, PCR has been used to improve the level of detection.[10,24,111,305,314,330] ETEC has worldwide distribution and is the major cause of TD, accounting for 20% to 50% of cases in series from all parts of the world.[155,221,249] It also accounts for a large percentage and frequently the majority of cases of enteritis in local pediatric populations of developing countries, where contaminated food and water are the primary sources of infection.[221] Most outbreaks of ETEC in the United States have been waterborne.[221,263] Person-to-person spread is infrequent because of the large infectious dose (10^6 to 10^{10} organisms).[221] Contamination of different types of food with these strains has been reported.[6,221]

Enteroinvasive *E. coli*. EIEC strains, like strains of *Shigella*, possess the property of bowel mucosa invasion, resulting in microabscesses and ulcer formation. Because of the presence of the same invasive plasmid and some antigens of *Shigella*,[191,264] EIEC must be considered (along with *Shigella*, *Salmonella*, *Y. enterocolitica*, *E. histolytica*, and *V. parahaemolyticus*) in the differential diagnoses of febrile dysenteric diarrhea and inflammatory bowel disease. EIEC strains are found worldwide and have been associated with food-borne outbreaks, especially in areas of South America and eastern Europe.[191,264,319]

Shiga Toxin–Producing *E. coli*. Shiga toxin–producing *E. coli* used to be classified as enterohemorrhagic *E. coli* (EHEC), and the organisms are also known as verotoxin-producing *E. coli*. They have caused outbreaks of diarrhea associated with consumption of contaminated beef, often fast-food hamburgers,

unpasteurized apple juice, and more recently with contaminated vegetables. Contact with contaminated swimming pools and exposure to farm animals have also been associated with this infection.[53,213,221,245,255,285,303] STEC produces copious bloody diarrhea with fecal mucus (hemorrhagic colitis), but fever is either low grade or absent.[303] The most important STEC strain thus far identified is O157:H7. The production of Shiga or similar toxins by these strains may be related to the hemolytic-uremic syndrome (HUS), a common complication in children infected with STEC O157:H7 or Shiga toxin–producing *Shigella*. HUS may be life-threatening, and it is possible that antimicrobial therapy of STEC colitis, rather than preventing this complication, is related to more frequent development of HUS.*[,326a]

Enteroaggregative *E. coli*. EAEC strains are the most recent addition to the group of diarrheogenic *E. coli*. In recent years, they have been identified as important and prevalent enteric pathogens, especially in TD. They are non-EPEC and do not produce ETEC LT or ST, and they adhere to human laryngeal epithelial (HEp-2) cells in a typical aggregative pattern. Several bacterial markers have been studied as possible diagnostic aids, but no one is present in all strains.[155,156,167] Some studies suggest that EAEC strains should be considered a phenotypically and genotypically heterogeneous group.[36,60,155,193,221,222] Although the pathophysiology of EAEC is not completely understood, multiple possible virulence factors, as well as association with inflammatory cytokines, have been described, especially with interleukin (IL)-8.[34,127,154–156,167,170,222,332] EAEC has been associated with acute and persistent illness and malnutrition in children with diarrhea, especially in the developing world,[54,152,231,275,332] and recent studies have demonstrated that EAEC is also an important cause of TD, second only to ETEC in some areas of the world.[3,5,6,116,167]

Diffusely Adherent *E. coli*. Also known as enteroadherent *E. coli*, these non-EPEC strains show a diffuse adherence pattern to HEp-2 cells. Although associated with cases of diarrhea, the pathogenicity, pathophysiology, and importance of these strains are still not completely understood, and some authors propose that they be categorized as a subtype of EAEC.[60,221,222]

Diagnosis

Laboratory culture cannot differentiate the various diarrheogenic strains of *E. coli* from normal bowel flora or from one another. Specialized assays, such as DNA probing and HEp-2 adherence technique, are specifically used for research purposes.[221] New serologic and molecular diagnostic techniques are under investigation and may become available in the future to differentiate between these organisms.[10,20,25,111,202,224,256,290,314]

Treatment

Most cases of *E. coli* diarrhea are brief and self-limited, and therapy should be primarily supportive with oral fluid replacement and maintenance, empirically based on the clinical manifestations. Dysenteric illness, however, should be treated with antibacterial drugs whether in a developing country or an industrialized region. The exception here is colitis caused by STEC, which should not be treated with antibacterial drugs because of the possibility of further development of HUS (see Shiga Toxin–Producing *E. coli*, earlier). In developing tropical countries, TD associated with the passage of numerous watery stools is often caused by both ETEC and EAEC, and antibiotics may shorten the duration of illness, especially when started within 48 to 72 hours of symptom onset. Because of the increasing resistance of ETEC strains to antimicrobial agents (including fluoroquinolones), new therapeutic agents, such as rifaximin and azithromycin, are actively being sought.[†,4,81,88,293] Because resistance patterns vary with geographic area and season, it is necessary to monitor the susceptibility of bacterial isolates in various regions of the world. Susceptibility testing is required when treating diarrhea caused by EPEC, because the strains are invariably resistant to a broad range of drugs.

Immunoprophylaxis

Several studies looking for possible ETEC vaccines have been unsuccessful,[32,192,266,276,277] apparently because of the presence of multiple and different colonization factor antigens that may not confer protection against all strains. A live vaccine for ETEC could offer advantages over a killed preparation in terms of duration and protection. Vaccines for EPEC and STEC are still under investigation.[148]

Salmonella

Salmonella infections may result in four different clinical syndromes: gastroenterocolitis, enteric (typhoid) fever, bacteremia with focal extraintestinal infection, and asymptomatic carriage,[30,274] depending on the type of organism and the host characteristics. Gastroenterocolitis is usually a mild to moderately severe, self-limited illness, with preferential involvement of the lower intestine. Enteric fever is characterized by septicemia with a prolonged toxic course if not treated. In patients infected with *Salmonella choleraesuis* strains, or with sickle cell disease or immunosuppression (e.g., those who have had a splenectomy; those who have HIV infection or a malignancy; those on immunosuppressive therapy; and newborns and older adults), nontyphoid salmonellae may disseminate and produce localized infection, including osteomyelitis or meningitis. A person with an abdominal aortic aneurysm is prone to develop *Salmonella* infection, leading to aneurysm perforation. Between 1% and 3% of patients who have recovered from typhoid fever may become chronic carriers who continue to shed the organism in the intestinal tract for 1 year or longer. Characteristically, the chronic typhoid carrier is an adult woman with cholelithiasis.[30]

Microbiology

The following discussion pertains to nontyphoid *Salmonella*, unless otherwise stated. Salmonellae are nonsporulating, facultative, gram-negative rods. The genus *Salmonella* is composed of more than 2000 serotypes that infect humans and animals. Enteric fever results from infection by *S. typhi* or by *S. paratyphi* A, B, and C, which usually cause milder disease. *S. typhi* and *S. paratyphi* are further distinguished by their adaptation to humans as the only host. Although numerous other serotypes are capable of causing enteric fever, illness is usually limited to gastroenteritis.[214,249,274] New serotypes occasionally become prominent, but most human infections are caused by only 10 serotypes, with *S. typhimurium* the most common one.

*References 26, 46, 52, 108, 213, 224, 247, 299, 303, 307.

†References 4, 81, 88, 125, 160, 214, 221, 282, 293, 320.

Epidemiology

Nontyphoid *Salmonella* organisms infect nearly all animal species and cause zoonotic infections. They can persist in fresh water for 2 to 14 days but may remain dormant in a desiccated nonsporulating state.[30] Human salmonellosis is a worldwide problem, remaining endemic in large areas of the developing world, where it is passed primarily through contaminated food and water. The Centers for Disease Control and Prevention (CDC) estimates that the 25,000 human cases of nontyphoid salmonellosis reported annually in the United States represent less than 1% of the actual number of clinical cases.[43,212,214] A recent report of typhoid fever in the United States from 1985 to 1994 showed that travel to underdeveloped countries is still a risk factor for this disease.[212]

Salmonella is the most common identifiable cause of foodborne illness. Contamination may occur from the animal feed, at slaughter, or, most often, during food preparation. Because the infectious dose is relatively high, averaging 10^3 to 10^6 organisms (but lower in water),[30] the bacteria must multiply on or in food. This accounts for the high summer case incidence, when refrigeration may not be adequate.[212] The foods most commonly implicated are meat, dairy products (especially unpasteurized), poultry, and eggs. Recent outbreaks of salmonellosis have been related to different foods, ranging from toasted oat cereal to alfalfa sprouts to infant formula.[42,212,308] Person-to-person spread accounts for 10% of cases, but 20% to 35% of household contacts may become infected.[212] *Salmonella* is an occasional cause of TD, accounting for up to 15% of cases, with marked geographic differences in serotype distribution.[43,94,249]

Elements that affect host resistance include normal gastric acid, gut motility, bacterial flora, and poorly understood immune factors. Bacterial virulence factors, the vehicle of transmission, and infectious dose are the major determinants of infection.[30,212] Salmonellosis primarily affects children and older adults. Fifty-five percent of reported isolates in the United States are from persons under 5 years of age. The organism has an unexplained propensity to infect infants less than 1 year of age, who may experience serious systemic infection, including sepsis and meningitis. Greater susceptibility has also been observed in patients with gastrectomy-induced hypochlorhydria, hemolytic disorders (e.g., sickle cell anemia), parasitic infections (e.g., malaria, schistosomiasis), and chronic illness (e.g., malignancies, liver disease).[212,214]

Pathophysiology

Salmonellosis involves mucosal invasion and possibly enterotoxin production.[172,274] After surviving the gastric acid barrier, the organisms reproduce in the gut, where they attach to the walls of the ileum and colon, inducing local degeneration of the microvilli. Invasion occurs through vacuolization, with resultant discharge of the bacteria into the lamina propria, from where they gain entry into the bloodstream. At this point, only the strains that cause enteric fever enter and multiply within lymphatic tissue and phagocytic cells. The mechanism of diarrhea in enterocolitis is not clear. A heat-stable enterotoxin has been identified. In most cases, local inflammation of the bowel wall is not severe enough to cause mucosal sloughing and dysentery.

Recent studies of pathogenesis demonstrated that IL-18 and gamma-interferon contribute to host resistance and that deficiency of IL-12 or nitric oxide is related to severity of infection.[204,210] Protection against typhoid fever is associated with the cystic fibrosis gene, called cystic fibrosis transmembrane conductance regulator (CFTR), and it resembles the protection of sickle cells against *Plasmodium* infestation. Apparently, *S. typhi* uses this gene to invade intestinal epithelial cells.[173,252]

Clinical Syndromes

Although the incubation period for typhoid fever is usually 1 to 2 weeks, it is only 8 to 48 hours for intestinal infections with nontyphoid *Salmonella*.[252] Nausea, vomiting, malaise, headache, and low-grade fever may precede abdominal cramps and diarrhea. Stools are usually foul and green-brown to watery, with variable amounts of mucus, blood, and leukocytes. Cholera-like fluid loss, or dysentery with grossly bloody and mucoid stools occurs less often. The acute phase lasts only a few days. Asymptomatic excretion of organisms in the stool continues for 4 to 8 weeks, and chronic carriers are rare. Infants less than 3 months of age experience longer illnesses (average, 8 days) with more complications. Among all ages, transient bacteremia is common, accounting for significant isolation of *Salmonella* types from blood. Fever and malaise occurring more than 1 week after resolution of diarrhea suggests a complication or another diagnosis.[173,252] In healthy adults, *Salmonella* bacteremia occurs in 5% to 10% of infections and is not distinguishable from other causes of sepsis. Focal infections may be seen in any organ, but sites adjacent to the bowel are most common. Mortality is highest at the extremes of age, but deaths occur in all age groups.[214]

Diagnosis

Diagnosis of enterocolitis can be made by clinical manifestations and isolation of *Salmonella* organisms from stool or rectal swabs cultured onto selective media (MacConkey's or salmonella-shigella agar). Blood cultures are useful to identify a systemic nontyphoid salmonellosis. Blood cultures (or culture of bone marrow aspirates) for *S. typhi* or *S. paratyphi* are also used to diagnose enteric fever. Stool cultures are often negative early in the disease. Widal's serum test is useful for diagnosing typhoid fever in areas with high prevalence, but not in industrialized areas, because of the more frequent occurrence of cross-reaction with other gram-negative organisms. Several PCR-based diagnostic techniques are currently being studied.

Treatment

Supportive treatment with fluids is sufficient therapy for most cases of uncomplicated *Salmonella* enterocolitis. Antibiotics are not indicated because they do not shorten the illness, and they slightly prolong the carrier state and increase the risk of developing resistant strains.[13,214] Antimicrobial therapy is indicated for persons who have symptomatic *Salmonella* infection with fever, systemic toxicity, or bloody stools. Patients with underlying debility (e.g., immunosuppression) that may predispose to septicemia or localized infection, young infants (less than 3 months), older adults (more than 65 years), and patients with sickle cell disease should be treated with antimicrobial agents. Fluoroquinolones are the treatment of choice because they shorten the duration of fever and diarrhea in salmonellosis. Dosages are the same as those recommended to treat shigellosis, although treatment is continued for 7 days (or 14 days if the patient is immunosuppressed).

In cases of enteric (typhoid) fever, septicemic salmonellosis, or local tissue suppuration, antibiotic therapy is indicated. The drugs of choice for enteric fever in the United States are fluo-

roquinolones. These drugs can be given for a shorter duration (10 instead of 14 days), resistance is still low, and post-treatment carriage of *S. typhi* is reduced.[13,102] In many developing countries, the drug of choice is still chloramphenicol (25 to 50 mg/kg/day in divided doses every 6 hours) because of its low price and predictable activity. Alternative empiric therapy in the United States is a third-generation cephalosporin. Other traditional options, such as ampicillin or TMP/SMX,[38] have low in vitro activity in many areas. Local suppuration may require 2 to 6 weeks of antibiotics, depending on the adequacy of surgical drainage.

Salmonella species, like *Shigella* species, are showing increasing resistance to many antimicrobial agents worldwide.[120,129,214,283]

Immunoprophylaxis

Immunity to *Salmonella* is serotype specific. Vaccines have not been successful for nontyphoid *Salmonella* because of the number of serotypes. For typhoid fever, immunoprophylaxis is possible, and three protective vaccines are now commercially available. The traditional killed vaccine is associated with a high reaction rate and has limited use in young children traveling in highly endemic areas. More frequently used for antityphoidal vaccination is a live-attenuated strain (Ty21a) that is given as one oral dose every other day for four doses,[12,117] or an inactivated Vi polysaccharide preparation given as a single parenteral immunization.[1] These two preparations are of approximately equal cost and effectiveness. New vaccines are under evaluation. One new live-attenuated *S. typhimurium* mutant is highly immunogenic and protective in animal models and induces cross-reactive antibodies to other enteric pathogens.[306]

Shigella

Microbiology

Dysentery has been described since the beginning of recorded history. At the end of the 19th century, Shiga first identified *Shigella dysenteriae* as the cause of an outbreak of dysenteric diarrhea in Japan, and since then, shigellosis has become synonymous with bacterial dysentery. As described before, other bacteria and protozoa are also capable of producing the bloody and mucoid stools that define this syndrome.

Shigellae are thin, nonmotile, nonsporulating, gram-negative rods in the Enterobacteriaceae family. There are four species or groups: A *(S. dysenteriae),* B *(Shigella flexneri),* C *(Shigella boydii),* and D *(Shigella sonnei).* A, B, and C contain numerous serotypes.

Epidemiology

Shigellosis occurs worldwide. *S. dysenteriae* 1 (Shiga bacillus), which causes severe disease, is most common in developing countries. In the United States and many other areas, particularly more industrialized regions, *Shigella* remains endemic, with *S. sonnei* replacing *S. flexneri* as the most common isolate.[133]

Humans and certain primates are the only hosts for *Shigella.* Fecal–oral contamination is the mode of spread. Common source infections occur through water or food prepared by contaminated hands.[185] *Shigella* can survive freezing and thawing in ice cubes. With an infectious dose as low as 10 to 200 organisms, person-to-person spread is common.[90] Even in countries with good sanitation, *Shigella* accounts for persistent endemic foci and high rates of transmission, especially among groups in close physical contact (e.g., gay men, children in daycare centers), groups with poor hygiene (e.g., mentally impaired patients), and those who lack sanitary facilities and water (e.g., populations in developing countries, Native Americans on reservations). Long-term carriage of *Shigella* is less common than for *Salmonella. Shigella* is a potential pathogen in the American wilderness. Environmental persistence averages 3 to 4 weeks, with best survival in cool fresh water.

Pathophysiology

The essential virulence factor of *Shigella* is invasiveness associated with a large (120- to 140-megadalton) plasmid. *Shigella* organisms invade and proliferate within the epithelium of the large bowel, producing well-demarcated ulcers with cellular infiltrates (chiefly PMNs and proinflammatory cytokines) and overlying suppurative exudates.[164,251,257] These organisms interact with the epithelial cells initially through a type III secretory system, with invasion of these cells and reorganization of their cytoskeleton.[70,124,227,272] Organisms have also been demonstrated in the small bowel, with reduced potential for invasion or changes in the mucosa, causing instead a profuse watery diarrhea, possibly mediated by an enterotoxin.[70]

Despite similarities in pathogenesis between EIEC and *Shigella* strains, different molecular biology techniques have shown controversial results.[19,264] However, one recent study supports the idea that EIEC and *Shigella* species are part of a pathovar of *E. coli.*[191]

Clinical Syndromes

As with most enteric pathogens, infection with *Shigella* may be asymptomatic, mild, or severe. Rarely, a chronic carrier state may develop, depending on a combination of host and organism factors. Two distinct diarrheal syndromes may occur separately or sequentially in shigellosis. After a short incubation period of 1 to 3 days, illness begins with malaise, headache, nausea, fever, abdominal cramps, and watery diarrhea, representing small bowel infection. Children may present with fever, with diarrhea developing later. In the second and classic form of shigellosis, after 1 to 3 days of small bowel disease, colonic involvement causes progression to clinical dysentery. In this dysenteric form, the volume of stools decreases and the frequency increases, with up to 20 to 30 bowel movements a day, containing gross blood and associated with fecal urgency and often tenesmus. Fever is common in dysenteric cases, found in up to one half of cases of shigellosis overall. Mild abdominal tenderness is also common but without peritoneal signs.

The natural history of shigellosis is varied, with most cases resolving spontaneously within 7 days but with others persisting for weeks.[73,86] The mortality rate is as high as 25% in developing countries when *S. dysenteriae* 1 (Shiga bacillus) diarrhea is untreated, but it decreases to less than 1% with adequate antimicrobial therapy.

Complications

Several potential complications of shigellosis may occur. Severe anemia and hypoalbuminemia may result from blood and protein losses. Febrile convulsions are seen in young children with shigellosis. *Shigella* has been described as a sexually transmitted disease and also as an unusual cause of urinary tract infection and pneumonitis. A severe leukemoid reaction with a white blood cell count of up to 50,000 may result after appar-

ent clinical improvement. In some patients infected by strains that produce Shiga toxin, HUS syndrome develops, probably induced by formation of immune complexes.[201] Reactive arthritis syndrome (or other reactive musculoskeletal symptoms) has also been reported in patients with *S. sonnei* and *S. flexneri* infection, sometimes associated with human leukocyte antigen (HLA) B27 positivity.[136] Septicemia was found in less than 5% of *Shigella* infections, with fewer cases of metastatic abscesses.[86,90]

Diagnosis

Laboratory tests often show mild leukocytosis with left shift (i.e., an increase in the number of immature granulocytes). If colitis is present, microscopic examination of the stool shows countless white (PMNs) and red blood cells, but this is not specific to shigellosis. Diagnosis is made by stool culture on selective media (MacConkey's or salmonella-shigella agar), which is positive in most infected patients.[86] Fresh stool or sigmoidoscopic biopsy is the best source of culture material, but rectal cotton swabs, although not as reliable, can be used if plated rapidly or placed in a holding medium. In hospitalized patients, blood cultures should be obtained. Few studies have tested the use of PCR for diagnosis of shigellosis.[10,111]

Treatment

Therapy first involves fluid replacement. Although large-volume diarrhea is unusual, significant dehydration may occur, especially in children. Antimotility drugs are controversial in patients with signs of toxicity[85]; however, these medications are unlikely to be detrimental if antibiotics are used concurrently.[7] Patients with fever and dysentery should be treated with antimicrobial agents, as these drugs decrease duration of fever, diarrhea, and excretion of *Shigella* in stool. Antibiotic-resistant strains are emerging worldwide, with recent reports in Asia, Oceania, and Latin America[38,150,289] showing that most of the strains are resistant to ampicillin and TMP/SMX, whereas the fluoroquinolones remain active. The current recommendation for treatment is with a fluoroquinolone (norfloxacin, 400 mg; ciprofloxacin, 500 mg; or levofloxacin, 500 mg daily) for a total of 3 to 5 days. Single-dose therapy is probably effective in milder forms of illness.[79,98,99,300] Fluoroquinolones are contraindicated in infants and children because of the possible effects on articular cartilage. However, short-course fluoroquinolone therapy appears to be safe. Alternative treatments for children in areas where TMP resistance occurs are nalidixic acid and furazolidone.[269] Another option is azithromycin, but it needs further testing.[4]

Immunoprophylaxis

Temporary immunity to homologous *Shigella* strains follows natural infection.[87,229] A vaccine composed of specific polysaccharide conjugates of *S. flexneri* and *S. sonnei* has been shown to be safe and immunogenic in children.[15] Other attenuated or killed strains or specific synthetic polysaccharides have shown promise in animal studies.[56,164,254,310]

Campylobacter

Microbiology

The *Campylobacter* organism is a small, curved, gram-negative rod, initially classified as *Vibrio*. *C. jejuni/coli* strains are widespread in the environment. The major reservoir is animals, including dogs, cattle, birds, horses, goats, pigs, cats, and sheep.[28] The strains of *Campylobacter jejuni* share up to 87% of their nucleotide sequence, and it is very difficult to differentiate them at the species level.[69] A reemergent species, *Campylobacter upsaliensis*, has recently been associated with diarrheal disease, persistent diarrhea in patients with HIV infection, and a few cases of HUS.[35]

Epidemiology

Most epidemics of gastroenteritis have been caused by contaminated food. The most important source for human illness is poultry, but epidemics have also been associated with ingestion of raw milk.[28,29] *C. jejuni* has been isolated from surface water and can survive up to 5 weeks in cold water, ensuring its potential for wilderness waterborne spread. Person-to-person spread occurs but is uncommon.

The prevalence of *C. jejuni* as a cause of TD varies with geographic area and time of year. TD secondary to *C. jejuni* is more prevalent in Southeast Asia and accounts for about 3% of cases in rainy summertime and up to 15% of cases during drier wintertime.[28,211,271] One recent study proposed flies as an agent transmitting *Campylobacter* and as a possible explanation of the seasonal distribution.[228] Studies in the United States and abroad have demonstrated that *C. jejuni* accounts for up to 25% of patients with infectious diarrhea and is often more common than *Salmonella* or *Shigella* species.[28] *C. jejuni* is now the most common cause of bacterial gastroenteritis in developed countries. Rates are highest among children and young adults.

Pathophysiology

The complete pathogenic mechanisms are unclear. All segments of the small and large intestine may be affected, accounting for the variety of diarrheal symptoms. Evidence of invasiveness includes recovery of bacteria from blood and presence of colitis, with cellular infiltration on intestinal biopsy. An unusual microtubule-dependent mechanism and a possible heat-labile enterotoxin may play a role in disease pathogenesis.[186]

Clinical Syndromes

The incubation period of *C. jejuni* enteritis is 2 to 7 days. Clinical symptoms are extremely variable and nonspecific. Victims often have a 1-day prodrome of general malaise and fever, followed by abdominal cramps and pain that herald the onset of diarrhea, with up to eight bowel movements a day. The diarrhea is initially watery, followed by passage of stools that are bile stained or bloody. The reported symptoms (and their frequencies) are diarrhea (75% to 95%), cramps and abdominal pain (80% to 90%), nausea (20% to 50%), headache (50%), fever (50% to 80%), vomiting (20%), and bloody diarrhea (10% to 50%).[28] Tenesmus is unusual. Physical examination is nonspecific, with variable degrees of fever (averaging 40° C [104° F]), abdominal tenderness, and dehydration. Microscopic evaluation of stool shows blood and PMNs in 60% to 75% of samples. The enteric symptoms subside in 2 to 4 days, and the entire illness resolves spontaneously within 1 week. Some patients developed postinfectious arthralgia, usually associated with more severe cases, apparently without any association with the use of antimicrobials.[200] *Campylobacter* organisms are shed in the stool for 3 to 5 weeks after resolution of symptoms, but chronic carrier states have not been described. Up to 20% of victims may show clinical relapse, which is usually less severe than the original symptoms.[28] Chronic diarrhea caused by *C.*

jejuni has been reported in children and adults but is usually associated with significant underlying disease.

Complications

C. jejuni infection has been associated with Guillain-Barré syndrome.[28,157,218,259] Apparently, the mechanism of cross-reactivity could be explained by induction of antiganglioside antibodies by specific strains of *Campylobacter.*[121] One study in Japan reported the possible association of specific strains and the development of two different syndromes: Guillain-Barré syndrome or Miller-Fischer syndrome, a variant of Guillain-Barré.[298]

Diagnosis

Definitive diagnosis is made by stool culture on a selective medium (e.g., Skirrow, Butzler, Campy-BAP), with isolation rates directly related to severity of disease. Extraintestinal sources account for 0.4% of positive *Campylobacter* cultures in the United States and are usually preceded by GI infection. Blood is the most common site in extraintestinal disease, followed by the gallbladder and cerebrospinal fluid (in children), but because blood cultures are rarely drawn in the evaluation of gastroenteritis, the real frequency of bacteremia is unknown. The serologic tests available are still not well standardized and need further evaluation.

Treatment

Treatment is primarily supportive, with oral fluids; dehydration is usually mild. Most patients have improved by the time that culture results return, and do well without antibiotics. Antibiotic treatment does not conclusively improve *C. jejuni* gastroenteritis, but earlier therapy appears to be effective[31] and eradicates the organism from the stool within 48 hours. The antibiotic of choice is erythromycin or a fluoroquinolone (unless there is a high reported prevalence of resistant strains, as in Southeast Asia). TMP/SMX is not active against *Campylobacter;* the new rifaximin is active in vitro[261] but still needs to be evaluated clinically. Fluoroquinolones are given in the same dosages as for shigellosis; they are active against all the major causes of dysentery *(C. jejuni, Shigella, Salmonella)*. In children, because fluoroquinolones are contraindicated, erythromycin (20 to 50 mg/kg every 6 hours for 5 days) is an option. Another alternative, in view of increased resistance to fluoroquinolones by *Campylobacter* strains,[118,282] is azithromycin, a newer macrolide that can be used in children and is active against all major bacterial enteric pathogens.[125,189]

Vibrio

Microbiology

Cholera is a severe form of watery diarrhea often associated with dehydration. The disease is caused by *Vibrio cholerae* O group 1 (O1), a motile, curved, gram-negative rod. These microorganisms have two major biotypes, classic and El Tor, which produce similar clinical illness. Each biotype contains two main serotypes, Ogawa and Inaba.[267] Non-O1 *V. cholerae* strains also produce diarrheal illness, but they show less potential for epidemic disease.[175] Nine other species have been associated with human disease. *V. parahaemolyticus, Vibrio fluvialis, Vibrio mimicus, Vibrio hollisae,* and *Vibrio furnissii* are associated with GI disease. Others, mainly *Vibrio vulnificus,* are associated with wound infections and septicemia. All are halophilic, gram-negative rods that reside in seawater and on marine organisms, and infection is acquired by ingesting infected and undercooked seafood or by contamination of a wound with infected water.[175,267]

Epidemiology

V. cholerae is endemic in areas of Asia, Africa, and the Middle East. It has accounted for seven deadly worldwide pandemics since the early 1800s. The last began in 1961 in Indonesia and spread throughout Southeast Asia, the Middle East, and Africa; to parts of the Pacific and Europe; and, in the 1990s, to Latin America. In 1973, cholera resurfaced in the United States after being absent since 1911. Since then, a small number of cases have occurred along the Gulf Coast of Louisiana and Texas. Only 10 cases of cholera were reported in travelers returning from endemic areas between 1961 and 1981. In January 1991, an outbreak of cholera started in Latin America along the coast of Peru. Since then, this disease has become endemic in most regions of Latin America, moving as close to the United States as northern Mexico.[175,267] The infection was associated with consumption of uncooked or poorly handled seafood, and it spread rapidly because of a highly susceptible population that has not been exposed to cholera for almost a century and because of inadequate water supply and sewage service. Cholera continues to be a disease of poor and lower socioeconomic groups; the Indian subcontinent and southwestern Asia are still the areas with the highest prevalence.[104,175,268] The risk to travelers has been estimated at 1:500,000 during a journey to an endemic area,[249] which should be further reduced with dietary discretion.

Nonhuman reservoirs for *V. cholerae* O1 include marine or brackish waters.[175,268] As with other strains (*V. parahaemolyticus,* non-O1 *V. cholerae*), shellfish ingest and carry these organisms. Fecal–oral spread is the major mechanism of transmission, and water is the most common vehicle, followed by food.[104] The organism remains viable for days to weeks in various foods. Because of the large infective dose (10^6 to 10^{10} organisms),[149] person-to-person spread is uncommon.

Most cases of gastroenteritis caused by noncholera vibrios have been associated with ingestion of raw seafood. Cases have been reported from travelers, particularly after visits to coastal areas of Southeast Asia and Latin America. *V. parahaemolyticus* causes 70% of cases of food-borne gastroenteritis in Japan (where large amounts of raw seafood are eaten), leads to sporadic outbreaks in the United States, and is a common cause of TD in Thailand.[33]

Pathophysiology

After passing through the stomach, the organism multiplies and colonizes the small bowel. The local effects of enterotoxin account for the pathophysiology of cholera. No pathologic changes are noted in the intestinal wall. The binding subunits of toxin attach to the membrane of the mucosa, after which the adenylate cyclase–activating B subunit enters the cell. The enzyme acts inside the serosal cell, enhancing production of cyclic adenosine monophosphate (cAMP). This molecule produces a 70% reduction in influx of water, saline, and a wide range of other substances into the gut mucosal cells, resulting in watery diarrhea. Glucose, potassium, bicarbonate, and most significantly, absorption of sodium and water linked to glucose remain intact. Thus, although plain water worsens cholera diarrhea, the addition of glucose renders the water and essential electrolytes absorbable, forming the basis for oral rehydration

therapy.[175,215,250,273] *V. cholerae* has the bacteriophage VPIphi, which encodes a receptor used by enterocytes for the phage CTXphi, which encodes the cholera toxin.[178]

Details of the pathogenesis of infection by the noncholera vibrios remain unclear. Some strains produce an enterotoxin, but generally it is not a cholera-like toxin. In the case of *V. parahaemolyticus*, a hemolytic toxin was thought to explain its effects, but the dysenteric illness that typically develops implies invasion. Another enterotoxin has been found in some strains.[33]

Clinical Syndromes

Some cholera infections are asymptomatic, and 60% to 80% of clinical cases are presented as mild diarrhea, which never raises suspicion for cholera.[149] After an incubation period of 2 days (range, 1 to 5 days), fluid accumulates in the gut, causing intestinal distention and diarrhea. Diarrhea may begin as passage of brown stools but soon assumes the translucent gray watery appearance known as "rice-water" stools. In serious cases, stool volume may reach 1 L/hr, leading to severe dehydration, acidosis, shock, and death. Vomiting may occur as a result of gut distention or acidosis.[175,267]

The clinical syndrome caused by noncholera vibrios is not characteristic. Intestinal illness is associated with diarrhea, abdominal cramps, and fever, with nausea and vomiting in about 20% of cases. Diarrhea may be severe, with up to 20 to 30 watery stools per day. In outbreaks of *V. parahaemolyticus* infection, explosive diarrhea associated with abdominal cramps and nausea is often described, with vomiting in about 50% and fever in about 30% of cases. In Asia, a dysentery-like syndrome with mucoid bloody diarrhea is often seen.[33] Infections are usually brief, lasting an average of 3 days, with spontaneous resolution.

Diagnosis

Diagnosis for any of the *Vibrio* strains can be made by stool culture on suitable media (e.g., thiosulfate-citrate-bile salts-sucrose [TCBS] agar). Vibrios can survive for 1 week on a stool-saturated piece of filter paper sealed in a plastic bag, before placing it in the culture medium.[175] In the case of *V. cholerae*, another way to diagnose infection is using a darkfield microscopic examination of fresh stools, which may reveal the characteristic helical vibrio motion.

Treatment

Aggressive replacement of fluid and electrolytes is the cornerstone of therapy for cholera, especially in severe cases. Severe untreated cholera has a 50% mortality, which may be reduced to 1% with appropriate treatment. Children are at higher risk for complications and death.

With fluid replacement, most cases of cholera last 3 to 5 days, with the peak fluid losses 24 hours after the onset of illness. When hypotension or persistent vomiting is present, IV fluids are necessary, but as soon as initial rehydration is complete, ORS is used for maintenance. Less than 5% of patients require IV maintenance after initial rehydration, and ORS alone is successful in 90% of cholera cases without shock. With voluminous losses, ORS can be given by nasogastric feeding tube to continue fluids during the night. A normal or light diet should be resumed early in the course of treatment, after initial rehydration. Success of fluid replacement therapy was clearly demonstrated by the low mortality rate seen during the cholera outbreak in Peru, where the principles of rehydration were applied.[175,267]

Antibiotics shorten the duration of diarrhea and excretion of organisms in severe cholera and reduce fluid losses, but they are not as important as fluid therapy. Oral antibiotics can be started within a few hours of initial rehydration. The drug of choice is doxycycline, a 300-mg single dose in adults or 50 mg/kg/day in four divided doses for children. This is perhaps the only indication for the use of tetracycline in children, because a short course (2 to 4 days) is unlikely to stain teeth. Furazolidone (100 mg every 6 hours for adults and 5 mg/kg/day in four divided doses for children) for 2 days is an alternative. *Vibrio* strains are also susceptible to fluoroquinolones, but these medications are more expensive.[175,267] In vitro activity of rifaximin against vibrios has already been established.[281]

Treatment of patients infected with noncholera vibrios should also focus on fluid replacement. Little information exists on the benefit of antibiotic therapy for GI disease, but antimicrobials may be reasonable in dysentery-like cases or prolonged illness. The same antimicrobial agents used in cholera could be used against this infection.

New developments in drug therapy of cholera, still under investigation, are aiming toward inhibiting the binding of cholera toxin to receptors in the enterocytes.[302]

Immunoprophylaxis

Temporary immunity to homologous, but not to heterologous, strains of cholera develop after infection.[175] The current parenteral vaccine has no antitoxin activity and is only about 50% effective in reducing attack rates over a 3- to 6-month period for those living in endemic areas. It is recommended for persons who live and work under poor sanitary conditions in highly endemic areas and for those with known achlorhydria. It is not recommended for travelers to endemic areas.[175]

Outside the United States, two additional vaccines are already available: an oral killed whole cell–cholera toxin recombinant B subunit (WC-rBS) and an oral live attenuated *V. cholerae* vaccine (CVD 103-HgR), both with a 60% to 100% rate of protection against *V. cholerae* O1 for at least 6 months. They are not active against *V. cholerae* O139.[266] Finally, a bivalent (CVD103-HgR plus CVD 111) oral vaccine has been shown to be more effective than the monovalent one.[13,22,134,301]

A recent advance was the development of transgenic potatoes that synthesized cholera toxin subunit B without requiring a cold chain. This is a promising option for an inexpensive, effective vaccine for the developing world.[9a]

Yersinia enterocolitica

Microbiology

Y. enterocolitica is an intracellular, facultative anaerobic, gram-negative rod in the Enterobacteriaceae family, and different serogroups have been found to cause human infection.

Epidemiology

The major natural reservoirs of the organism are wild, farm, and domestic animals. In the United States and Europe, the organism resides in surface and unchlorinated well waters. Evidence indicates that persistence in warm water ranges from days to weeks, with longer survival at colder temperatures. Human isolates of *Y. enterocolitica* are found worldwide, but with preference for colder regions such as Canada and Northern

European countries, with an incidence equal to or greater than those of *Salmonella* and *Shigella*.[208] Transmission occurs from fecal–oral contamination, through food and water, and probably through person-to-person or animal-to-person contact.[208,249,311] Raw milk and oysters have also been implicated as vehicles of transmission. The infectious dose and attack rate are not well studied, but yersiniosis is suspected to be caused by ingestion of a large infectious dose based on a common source of transmission. The incubation period averages 3 to 7 days. Patients with β-thalassemia show a greater risk for acquisition of yersiniosis.[8]

Pathophysiology

Illness caused by *Y. enterocolitica* may involve three pathogenic mechanisms: bowel mucosal invasion, release of a heat-stable enterotoxin similar to that produced by ETEC, and elaboration of a cytotoxin.[73,208] The organism multiplies in the small bowel and characteristically invades the mucosa in the region of the terminal ileum and colon. The mucosa may be diffusely inflamed with small and shallow ulcerations. Also, some bacteria migrate through lymphatics to mesenteric lymph nodes, producing adenitis with focal areas of necrosis.

Clinical Syndromes

The most common clinical presentation in yersiniosis is gastroenteritis, characterized by diarrhea, fever, and abdominal pain, with nausea and vomiting in 20% to 40% of cases and dysentery (passage of bloody stools) in 10% to 25%.[208,311] Fever or abdominal pain without significant diarrhea may be the most prominent sign, mimicking appendicitis in up to 20% of patients with positive stool cultures.[18] Although acute appendicitis has been associated with serologic evidence of *Y. enterocolitica* infection, the usual surgical findings are mesenteric adenitis or terminal ileitis. Rarely, severe colitis results in septicemia, extensive necrosis, or perforation. Numerous extraintestinal manifestations of *Y. enterocolitica* infection include skin rash (erythema nodosum or maculopapular) and arthritis, probably related to an immune reaction. Extraintestinal infection involving lung, joints, lymph nodes, wounds, or septicemia may occur with or without enteritis. In the majority of intestinal infections, illness is mild and self-limited, with duration averaging 1 week, but some patients experience prolonged symptoms.[208,311] Excretion of the organism in stool continues for a few weeks to months. Complications may be related to particularly severe disease and a misdiagnosis of Crohn's disease or appendicitis and development of Reiter's disease or collagenous colitis.[205]

Diagnosis

The diagnosis of yersiniosis is usually made by stool culture, but it can grow also from blood or surgical samples. The organism grows better at lower temperatures (22° to 25° C [71.6° to 77° F]), which inhibit most other enteric bacteria. Abnormalities related to ileitis or colitis and seen on contrast radiography and colonoscopy may be mistaken for other causes of colitis.[17,208] Serologic tests are also diagnostic and especially helpful to diagnose *Yersinia* arthritis.

Treatment

Tetracyclines have been suggested as the drug of first choice for chronic or fulminant infections,[17] but *Yersinia* is also susceptible in vitro to streptomycin, chloramphenicol, aminoglycosides, fluoroquinolones, TMP/SMX, and the new rifaximin. Most strains are resistant to penicillins and cephalosporins and variably resistant to erythromycin and sulfonamides.[122,203]

Aeromonas Species and *Plesiomonas shigelloides*

Aeromonas species and *Plesiomonas shigelloides* are gram-negative, facultative anaerobic, nonsporulating rods. Their normal habitats are water and soil. These bacteria have been implicated in a variety of human illnesses, most often gastroenteritis.[146,147,163,249,316]

Aeromonas was part of the Vibrionaceae family until the family Aeromonadaceae was established to include the 14 species so far identified. Only five species (*Aeromonas hydrophila, Aeromonas caviae, Aeromonas veronii, Aeromonas jandaei,* and *Aeromonas schubertti*) have been associated with a variety of human diseases, including gastroenteritis, soft tissue infections, HUS, burn-associated sepsis, and respiratory infections.[163,316] *A. hydrophila* is the most commonly isolated species, but its real prevalence is still uncertain. *A. hydrophila* has been associated with diarrheal illness in the United States, Australia, India, Latin America, and southwestern Asia.[163,315,316] An association between the illness and drinking untreated spring or well water was demonstrated in the United States.[163] Pathogenicity includes production of cytotoxin, enterotoxin, and proteases, as well as the capacity of adhesion and invasion, with participation of a type III secretory system.[331] The pathogenicity of *A. hydrophila* remains controversial.[163,278]

Clinical illness associated with enteric infection by *A. hydrophila* varies from acute to chronic diarrhea and from passage of watery stools to dysentery with colitis.[163,278,315] Median duration of diarrhea is 2 weeks, but occasional cases persist a month or longer. Asymptomatic carriers have been identified. Non-GI infections, such as soft tissue infections and septicemia, have been associated with exposure of wounds to water. *Aeromonas* strains are susceptible to chloramphenicol, tetracycline, TMP/SMX, fluoroquinolones, rifaximin, and aminoglycosides but are resistant to ampicillin and erythromycin.[122,163]

P. shigelloides has been isolated from patients with gastroenteritis, both in sporadic cases and in outbreaks.[147,249] Infection has been associated with recent travel and ingestion of raw or inadequately cooked shellfish. *Plesiomonas* may cause dysenteric illness suggestive of an invasive organism, but its pathogenic mechanisms remain poorly defined.[147] *Plesiomonas* is susceptible in vitro to tetracyclines, aminoglycosides, quinolones, TMP/SMX, azithromycin, and rifaximin.[122,294]

Miscellaneous Bacterial Agents

Klebsiella pneumoniae and *Klebsiella oxytoca* have been reported to occasionally cause diarrhea, but they are usually commensals of the GI flora.[130] Another cause of severe diarrhea in hospitalized (usually postoperative) patients receiving antibiotics is *Staphylococcus aureus*. The causative organism of this antibiotic-associated diarrhea illness may be methicillin-resistant *S. aureus*.[260,279]

VIRAL ENTERIC PATHOGENS

Studies have identified viruses as major causes of acute nonbacterial GI infections.[27,48,49,139] The most important defined agents are noroviruses and other caliciviruses, rotavirus, enteric

adenoviruses, and astroviruses. Usually they cause vomiting with or without mild and self-limiting watery diarrhea. Transmission occurs through fecal–oral contamination or person-to-person transmission. Respiratory symptoms are common in patients with viral gastroenteritis.

Caliciviruses and astroviruses are similar to noroviruses in structure and clinical appearance. They generally infect in early childhood and apparently provide lifetime immunity.[240,241]

Noroviruses

Noroviruses are the leading cause of viral gastroenteritis of all ages. Norwalk virus is the prototype strain that belongs to the genogroup I, and it was the first well-described etiologic agent in nonbacterial gastroenteritis outbreaks, in an elementary school outbreak that occurred in Norwalk, Ohio.[176] Soon after, several other small round viruses were identified as causes of nonbacterial gastroenteritis.[240,241]

Noroviruses are nonsymmetrical, single-stranded ribonucleic acid (RNA) viruses, recently classified as being in the family Caliciviridiae.[166,190] They are the main cause of outbreaks of epidemic nonbacterial GI illness worldwide. They also cause "winter vomiting disease" because of their wintertime predisposition and common association with vomiting. In the United States, the proportion of norovirus outbreaks increased from 1% in 1991 to 12% in 2000, mainly related to food-borne outbreaks.[326]

These viruses are highly infective (requiring only 10 to 100 organisms per inoculum), and the infection is spread by common-source vehicles with a propensity for secondary person-to-person spread (high secondary attack rate).[139] Humans are the only known carriers of these viruses. Outbreaks have been recognized in family settings, health care facilities, nursing homes, schools, cruise ships, and travel settings, characteristically affecting both children and adults in the United States. They are found less often in neonates and toddlers. Contaminated water supplies, drinking water in cruise ships, recreational swimming pools, and commercial ice cubes have been implicated in outbreaks.[139,190] Vehicles identified for food-borne outbreaks include contaminated shellfish, salads, bakery products, cold foods, cooked meat, and fresh fruits.[241] Between 20% to 67% of outbreaks of noroviruses have been associated with food.[40,233] After a cruise ship has experienced a norovirus outbreak, this viral infection can continue to be a problem in future trips, despite extensive sanitization.[161]

After invasion of the enterocytes, the viral particles replicate inside, resulting in damage to the villi and in crypt cell hyperplasia.[240] Malabsorption of fat, lactose, and xylose occurs with these histologic changes. The exact mechanisms of diarrhea production in viral gastroenteritis are not completely understood. Small numbers of viral particles are shed in stool during the acute illness, but prolonged carrier states are not seen.

In the United States, antibodies in stools typically appear during late adolescence, but in tropical, developing countries, children acquire antibodies at a young age. Although antibodies persist in most people, they do not provide protection from clinical illness.[240] A recent study has found that people with histo-blood group antigens type B may be protected against infection with Norwalk virus or norovirus genogroup I.[262]

Transmission is followed by an incubation period of 24 to 48 hours, and illness begins abruptly with vomiting, abdominal cramps, and diarrhea. Stools are watery and usually do not contain blood or leukocytes. Other common symptoms include low-grade fever, malaise, myalgias, respiratory symptoms, and headache. Illness is almost always mild and self-limited, lasting 1 to 2 days. Complications and mortality are extremely rare and usually involve older adult and debilitated patients. Some malabsorption of fats and disaccharides persists after the acute illness. Supportive treatment with oral fluids and electrolytes is sufficient in the vast majority of cases.[240]

Historically, because of the difficulty or impossibility of growing these viruses in cell culture, electron microscopy was the initial means of detecting these viruses. Now, immunoassays and molecular techniques (reverse-transcription PCR) are available for detection of these small round RNA viruses in stool.[126,165,219,240]

Vaccine development is being explored for the noroviruses but the studies are in a very early stage.

Rotavirus

Rotaviruses are 70-nm, double-stranded RNA viruses that are classified by the capsid antigens, with group A and serotype G1 being the most common in human infections worldwide. Most severe infections in children are caused by serotypes G1 to G4.[25,242] Rotavirus is the most common enteric pathogen in children, causing diarrhea worldwide and resulting in around 800,000 deaths per year.[48,139,174,242,325] Infection tends to be endemic, with peak incidence during winter months in temperate climates. Transmission is by person-to-person contact or as a result of common-source outbreaks. Viral shedding occurs in stools, and particles can retain infectivity for months. Rotaviruses have been found in almost every animal species, and in general, animal strains have reduced virulence for humans.[25,48]

Rotavirus infects humans repeatedly, at any age. It is the most frequently isolated pathogen in infantile diarrhea and is responsible for a disproportionate amount of hospitalization for dehydration.[25,242] The majority of symptomatic infections occur in children less than 3 years old, with peak incidence in children 6 months to 2 years of age. Rotavirus can also cause illness in adults, apparently with the same strains that infect the children and usually associated with secondary spread within a family.[25,61]

Nonspecific pathologic changes are seen in small bowel epithelial cells, with particles identified intracellularly. The exact mechanism of diarrhea is unknown, but a net secretion of water, sodium, and chloride occurs during the illness. Diarrheal losses contain 30 to 40 mEq/L each of sodium and potassium. Lactose malabsorption may persist for 1 to 2 weeks and is associated with continued viral excretion in stool. Large numbers of viral particles are shed in the stool of ill patients, but prolonged excretion is unusual.[242]

After an incubation period of 24 to 72 hours, illness begins with vomiting, followed by diarrhea associated with abdominal cramps, low-grade fever, and malaise. Vomiting usually resolves within 2 days (range, 1 to 5), but the diarrhea may last 3 to 8 days or longer. Natural infection reduces the incidence and severity of subsequent episodes. Serum antibody levels are demonstrated within the first few years of life and in almost all adults but appear to be nonprotective. Severity of illness usually decreases with age. Adults are more likely to have asymptomatic or mild illness with less vomiting. Dehydration frequently occurs in children, accounting for appreciable mortality in developing countries. Fortunately, oral fluid and electrolytes can be successfully used in most cases.[242]

The first rotavirus vaccine approved by the FDA in 1998 was a tetravalent rhesus-human strain that provides coverage against the four common G serotypes of human rotavirus.[232,242,312] In clinical trials in industrialized countries, this vaccine provided 50% to 70% protection (up to 60% to 100% protection against severe cases). The recommendation was to give this vaccine orally to all children at 2, 4, and 6 months of age,[232] but the vaccine was withdrawn because of associated intussusception (calculated risk of 1/10,000 to 1/32,000 vaccine recipients).[23] Several new safe and effective rotavirus vaccines are being developed, with one already licensed in Mexico in 2004 and another in the last stages for approval in the United States at the time of this writing.[63,325]

INTESTINAL PROTOZOA

Protozoal infections may be pathogenic or commensal (which have little or no effect) to the human host. Most protozoal infections are suspected on the basis of subacute or chronic GI symptoms, which may fluctuate over time. Although acute self-limited diarrheal illness may occur, only a small proportion of cases of acute TD are caused by parasites. The symptoms are nonspecific, and the diagnosis is often made on stool examination.

Several factors have increased the prevalence of intestinal parasites in the United States and worldwide: an increase in immunocompromised patients, who frequently become infected by these organisms; improvement in diagnostic techniques; changes in social behaviors (increased use of daycare and nursing homes, more frequent international travel); and in the United States, increased immigration of people from Asia, Africa, and Latin America.[131,177,179]

As with enteric bacteria, symptoms from infection by intestinal protozoa depend on the level of bowel colonized. Those colonizing the small intestine, such as *Giardia* and coccidia (see *Cryptosporidium,* later), cause a wide spectrum of GI complaints, including malabsorption (resulting in foul stools and flatulence) and weight loss in persistent infections. Although many protozoa are capable of superficial mucosal invasion, only *Entamoeba histolytica* and *Balantidium coli,* which colonize the colon, can ulcerate the bowel wall, cause dysentery, and spread to other tissues.[131]

All intestinal protozoa are transmitted by the fecal–oral route, so infection rates are highest in areas and groups with poor sanitation, close contact, or particular customs favoring transmission. These reemerging infections have been related to large outbreaks of communicable diseases in the United States, often secondary to water contamination. Protozoal parasites were the most frequent etiologic agents detected in waterborne outbreaks from 1991 to 1994.[187,209,217] In addition to spread by food, water, and person-to-person contact, mechanical vectors such as flies may spread these organisms. Transmission of intestinal protozoa is favored by a hardy cyst, which is passed in the feces of an infected host. In addition to an infective cyst, the life cycle for most intestinal protozoa includes a trophozoite, which is responsible for reproduction and pathogenicity. Only a single host is required, except for *Sarcocystis,* which requires ingestion of raw meat from an intermediate host. Zoonotic spread to humans has been documented for *Giardia, Cryptosporidium, Entamoeba polecki,* and *B. coli.* Treatment of intestinal protozoal infections is summarized in Table 62-11.

TABLE 62-11. Antiparasitic Therapy for Infectious Diarrhea in Adults

DIAGNOSIS	RECOMMENDATION
Giardiasis	Metronidazole 250 mg tid (15 mg/kg/day for children), albendazole* 400 mg qd, or quinacrine† 100 mg tid for 7 days; or tinidazole* 2000 mg single dose; or nitazoxanide 500 mg bid for 3 days (100 mg bid for 3 days for children 1 to 4 years of age; 200 mg bid for 3 days for children 4 to 11 years of age)
Entamoeba histolytica excretion (asymptomatic)	Iodoquinol 650 mg tid for 20 days or paromomycin 500 mg tid for 7 days
Entamoeba histolytica diarrhea	Metronidazole 750 mg tid for 5 to10 days or tinidazole* 1000 mg bid for 3 days, followed by iodoquinol 650 mg tid for 20 days or paromomycin 500 mg tid for 7 days
Cryptosporidiosis	Nitazoxanide 500 mg bid for 3 days; in severe cases or patients with AIDS, consider nitazoxanide 500 mg bid for 2 weeks, paromomycin 500–750 mg tid or qid for 2 weeks; or azithromycin 1200 mg qd for 4 weeks
Cyclosporidiosis	TMP/SMX 160 mg/800 mg bid for 7 days, followed by 160 mg/800 mg 3 times per week in patients with AIDS
Isosporiasis	TMP/SMX 160 mg/800 mg qid for 10 days, followed by 160 mg/800 mg bid for 3 weeks, or pyrimethamine 75 mg qd with folinic acid 10 mg qd for 2 weeks
Microsporidiosis	Albendazole* 400 mg bid for 2 to 4 weeks, followed by chronic suppression in patients with AIDS

*Albendazole and tinidazole were recently approved and are available in the United States.
†Quinacrine is not commercially available in the United States.
bid, twice daily; qd, daily; qid, four times daily; tid, three times daily; TMP/SMX, trimethoprim and sulfamethoxazole.

Giardia lamblia

G. lamblia (also known as *Giardia intestinalis* or *Giardia duodenalis*) is a flagellate protozoan that was first observed in 1681 by Leeuwenhoek. In the last 40 years, it has gained recognition as an important human pathogen.[103,197] The classification of *Giardia* species remains controversial.

Giardia is the most common protozoal intestinal parasite isolated worldwide. All age groups are affected.[103,177,237] Giardiasis usually represents a zoonosis, with cross-infectivity from animals to humans, and vice versa. *Giardia* has been found in stools of beavers, cattle, dogs, cats, rodents, and sheep.[237]

The infective dose of *Giardia* for humans is low: 10 to 25 cysts caused infection in 8 of 25 subjects; more than 25 cysts caused infection in 100%. Person-to-person spread may be the most common means of transmission for humans. Twenty-five percent of family members with infected children become infected.[103] Areas and populations with poor hygiene and close physical contact have higher rates of infection. Venereal trans-

mission occurs among homosexuals through direct fecal–oral contamination. Epidemics and carrier rates of 30% to 60% have been found among children in daycare centers and in Native American reservations. Water is a major vehicle of infection in community outbreaks.[209] Cysts retain viability in cold water for as long as 2 to 3 months. In the United States from 1964 to 1984, 90 outbreaks (24,000 cases) of giardiasis were linked epidemiologically to water, and it is still the most frequently identified cause of waterborne diarrhea outbreaks. Most of these occurred in small water systems that used untreated or inadequately treated surface water.[194,195,209] Clear and cool mountain water has been so often associated with giardiasis that the illness has been called backpacker's diarrhea or beaver fever (although fever is not usually seen). An outbreak in Aspen, Colorado, in 1964 was the first well-documented waterborne outbreak in the United States, and recent outbreaks around the same area indicate that this area remains endemically infected with *Giardia*. In the northeastern states, large outbreaks have occurred in the mountain communities of Rome, New York, and Berlin, New Hampshire. Every U.S. region has experienced waterborne outbreaks, but the western mountain regions (Rocky Mountains, Cascades, Sierra Nevada) have reported the majority, and giardiasis must be considered endemic there.[103,144,209,237]

Giardia accounts for a small percentage of TD.[249] It has been identified in a large percentage of cases among travelers to St. Petersburg, Russia, where tap water is the usual source. Because of the relatively long incubation period and persistent symptoms, *Giardia* is more likely to be found as the cause of diarrhea that occurs or persists after returning home from travel to any developing region.[90]

The pathophysiologic mechanisms of diarrhea and malabsorption in giardiasis are poorly understood.[237] Reversible malabsorption of fats, vitamins A and B, folate, and disaccharides has been demonstrated in some patients with diarrhea. Malabsorption may result from (1) physical blockade by large numbers of trophozoites blanketing the intestinal mucosa, (2) deconjugation of bile acids, (3) bacterial or fungal overgrowth in the small intestine, (4) increased turnover of cells on the mucosa of the villi, which do not absorb normally, and (5) epithelial damage. Altered gut motility and hypersecretion of fluids, perhaps through increased adenylate cyclase activity, may play a role. Histologic changes of villous atrophy and inflammatory infiltrates with epithelial cell destruction have been observed. In some series, these changes correlated with degree of malabsorption and reverted to normal after treatment. However, most small bowel biopsies in human patients demonstrate minimal or no changes, with only occasional mucosal invasion (with trophozoites found intracellularly and extracellularly) and no local inflammatory response.[103] Enterotoxins have not been found. More than one mechanism is probably involved.

Most infections are asymptomatic, and in endemic areas, *Giardia* is found in healthy people. The attack rate for symptomatic infection in the natural setting varies from 5% to 70%.[237] Asymptomatic carrier states with high numbers of cysts excreted in stools are common. Correlation between inoculum size and infection rates has been noted, but not with numbers of cysts passed or severity of symptoms. Infectivity apparently depends on both host and parasite factors.[237] Hypochlorhydria, certain immunodeficiencies, blood group A, and malnutrition apparently predispose to symptomatic infection.[103,237]

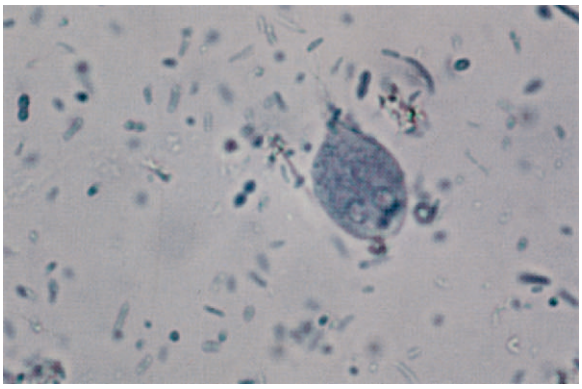


Figure 62-2. *Giardia lamblia* trophozoite seen by methylene blue wet mount staining under oil (×1000). The finding of cysts or trophozoites in a patient with diarrhea is sufficient to make a tentative diagnosis of giardiasis.

The incubation period averages 1 to 2 weeks, with a mean of 9 days and a wide clinical presentation. A few people experience abrupt onset of explosive watery diarrhea accompanied by abdominal cramps, foul flatus, vomiting, low-grade fever, and malaise. This typically lasts 3 to 4 days before transition into the more common subacute syndrome. In most patients the onset is more insidious and symptoms are persistent or recurrent. Stools become mushy, greasy, and malodorous. Watery diarrhea may alternate with soft stools and even constipation. Upper GI symptoms, typically exacerbated postprandially, accompany stool changes, but they may be present in the absence of soft stool. These include mid-abdominal and upper abdominal cramping, substernal burning, acid indigestion, sulfurous belching, nausea, distention, early satiety, and foul flatus. Constitutional symptoms of anorexia, fatigue, and weight loss are common. Unusual presentations include allergic manifestations, such as urticaria, erythema multiforme, and bronchospasm. Some *Giardia* infections are associated with a chronic illness. Adults may have a longstanding malabsorption syndrome and marked weight loss, and children may have a failure-to-thrive syndrome.[103,237]

Laboratory confirmation of giardiasis can be difficult. Stool examination used to be the primary means of diagnosis (Fig. 62-2) but is being replaced by newer immunodiagnostic tests. Trophozoites may be found in fresh, watery stools but disintegrate rapidly. Although trophozoites remain in fresh stools for at least 24 hours, stools should be preserved in a fixative such as polyvinyl alcohol or a formalin preparation if not immediately examined. Cyst passage is extremely variable and not related to clinical symptoms.[237] In the office, fresh stool can be mixed with an iodine solution (e.g., Gram's iodine) or methylene blue and examined for cysts on a wet mount. Many antibiotics, enemas, laxatives, and barium studies mask or eliminate parasites from the stools, so examinations should be delayed for 5 to 10 days after these interventions. Trichrome stain is better than the formalin–ether concentration technique for identification of protozoal cysts and trophozoites. The current recommendation is to examine three samples taken at intervals of 2 days. Another noninvasive office test is duodenal mucus sampling, using a string test (Enterotest), which has a reported sensitivity of 10% to 80%.[112,237] Duodenal biopsy is rarely necessary but may be the most sensitive test.[112]

A commercial enzyme immunoassay (EIA) has shown the same sensitivity as microscopic examination, but its specificity is 100%, making it a convenient screening method. EIA is much easier and requires less experience than microscopy, but it cannot be used to differentiate between cysts and trophozoites.[9] Immunofluorescent techniques using monoclonal antibodies could detect low numbers of organisms in a short time but require centrifugation of the sample,[113] and molecular techniques need further development.[226,237,322]

Immunologic responses to *Giardia* infection are complex. Epidemiologic studies show acquired resistance, with lower rates of infection and illness (1) among residents of endemically infected areas compared with visitors and (2) among adults compared with children. However, reinfection does occur. Levels of IgG antitrophozoite antibodies rise with both symptomatic and asymptomatic infections, helping to clear the infection. Hypogammaglobulinemic patients have a higher incidence of symptomatic giardiasis, implying an important protective function of immunoglobulins.[237] Effects of mucosal secretory antibodies in humans have not been clearly demonstrated, although mouse studies show a protective effect of IgA secretory antibodies. Both cellular and humoral responses to *Giardia* have been demonstrated. Immunologic responses are effective in the majority of infections because spontaneous clinical recovery is common with or without the disappearance of organisms. The average duration of symptoms in all ages ranges from 3 to 10 weeks.[237]

Because of the difficulty and expense of confirming the diagnosis in some patients, a therapeutic trial of drugs may be attempted when suspicion is high. Imidazole derivatives (e.g., metronidazole) affect bacterial flora as well, so they are less specific but still better for empiric (unproven diagnosis) therapy because of their wide activity.

Symptomatic patients should be treated for comfort and to prevent the development of chronic illness. Asymptomatic carriers in nonendemic areas should be treated when identified because they may transmit the infection or develop symptomatic illness. No drug is effective in all cases. In resistant cases, a longer course of two drugs taken concurrently has been suggested. Relapses occur up to several weeks after treatment, necessitating a second course of the same medication or an alternative drug. Malabsorption usually resolves with treatment, but persistent diarrhea may result from lactose intolerance or a syndrome resembling celiac disease rather than from treatment failure.[237]

Three groups of drugs are currently being used: nitroimidazoles (metronidazole, tinidazole, albendazole, ornidazole, nimorazole), nitrofuran derivatives (furazolidone), and acridine compounds (mepacrine, quinacrine). Metronidazole (Flagyl, 250 mg three times a day for 5 days for adults) is often used in the United States. Cure rates of 85% to 90% are comparable to those with quinacrine but with better tolerance. Tinidazole (Fasigyn, 2000 mg in a single dose) has the same success rate with better compliance but was not approved for this use in the United States in 2004. Nitazoxanide (Alinia) has comparable efficacy to metronidazole and is available in suspension for children as young as 1 year of age (100 mg twice a day for 3 days for younger than 4 years, 200 mg twice a day for 3 days for ages 4 to 11 years, 500 mg twice a day for adults).[108a] Quinacrine (Atabrine, 100 mg three times a day for 5 days for adults and 7 mg/kg/day in three divided doses for 5 days for children), achieves cure rates of about 95%. Unfortunately, it is no longer available in the United States because it produces more frequent side effects, especially in children. No pediatric liquid form is always available. For use in severely symptomatic individuals or pregnant women, the nonabsorbable drug paromomycin (Humatin, 25 to 30 mg/kg in three divided doses for 5 to 10 days) has been effective.[103,114,135,197,234,237]

Entamoeba

Lösch described the trophozoite form of *Entamoeba* in 1875 and Quincke and Ross the cyst form in 1893. Recently, molecular biologic studies confirmed the existence of an invasive parasite *(E. histolytica)* and a noninvasive, commensal organism *(Entamoeba dispar)*.[158,162,198,292] Isoenzyme analysis has recognized 22 different zymodemes of *E. histolytica,* which may explain the pathogenic and commensal strains and the geographic differences in rates of invasive disease.[36,162,258]

E. histolytica is found worldwide. Approximately 12% of the world's population is infected.[258] The higher prevalence (30% to 50%) in tropical countries (Mexico, India, Africa, Central and South America) is related to increased risk of fecal–oral contamination, which depends on sanitation, cultural habits, crowding, and socioeconomic status.[36,162,292] It is the second most important cause of death by parasitic infection worldwide. Similar conditions create pockets of endemic infection in the United States among institutional inmates, Native Americans on reservations, and homosexuals. Importation of infections by travelers and immigrants accounts for most cases in the United States and other temperate countries.[117] Attack rate and prevalence are difficult to determine because the majority of infections are asymptomatic, and screening with single stool samples is likely to identify only 20% to 50% of cases.[36,162] The 10% to 15% of the U.S. population once infected with *E. histolytica* has decreased to 1% to 5% overall, primarily because of adequate water and sewage treatment.[177,188] Between 1946 and 1980, six waterborne outbreaks of amebiasis were reported in the United States.[199,209] Amebiasis accounts for less than 1% of TD.[162,292]

The pathogenicity of *E. histolytica* is not well understood.[162,292,333] Invasion may be a function of motility, soluble toxins, cysteine protease, or lytic enzymes. The cecum and ascending colon are most frequently involved, followed by the rectum and sigmoid colon. Five different lesions of increasing severity can be distinguished in the colon: (1) diffuse inflammation with cellular infiltrate and an intact epithelium, (2) superficial erosions, (3) early invasion followed by shallow ulceration, (4) late invasive lesions forming the classic flask ulcers with skip lesions, and (5) loss of mucosa and muscularis, resulting in exposure of underlying granulation tissue. Extraintestinal spread is hematogenous. Abscesses containing acellular debris develop primarily in the liver but may involve the brain and lung.[101,162,292]

Although 80% to 99% of infections result in asymptomatic carriers, a spectrum of GI diseases may result. The incubation period ranges from 1 to 4 months, depending on the area of endemicity. Most often, colonic inflammation without dysentery causes lower abdominal cramping and altered stools, sometimes containing mucus and blood.[162,258] Weight loss, anorexia, and nausea may be present. Symptoms commonly fluctuate and continue for months. The subacute infection may evolve into a chronic, nondysenteric bowel syndrome, with intermittent diarrhea, abdominal pain, weight loss, and flatulence. Dysentery may develop suddenly after an incubation period of 8 to 10 days

or after a period of mild symptoms. Affected persons may have frequent passage of bloody stools, tenesmus, moderate to severe abdominal pain and tenderness, and fever. There is considerable variation in severity.[292]

Humoral antibodies increase with invasive disease and persist for long periods. Although they do not protect against reinfection or bowel invasion, they show antiamebic action in vitro and may prevent recurrent liver infection, which is uncommon. Once the infection is cleared, recurrence is unusual, but asymptomatic cyst shedding and active GI illness may persist for years, indicating lack of consistent immune response in the intestinal lumen.[162,292]

The fatality rate for amebic dysentery and its complications is about 2%. Complications of intestinal involvement develop in 2% to 20% of cases and include perforation, toxic megacolon, and ameboma. An ameboma is an annular inflammatory lesion of the ascending colon containing live trophozoites. It may be improperly diagnosed as a pyogenic abscess or a carcinoma. A postdysenteric syndrome can occur in patients with acute amebic dysentery and can be confused with ulcerative colitis.

The diagnosis of intestinal amebiasis is made by identification of cysts or trophozoites in stool. Mucus from fresh stools or sigmoidoscopic scrapings and aspirates mixed with a drop of saline may show trophozoites if examined within an hour. For delayed examination, stool must be preserved in polyvinyl alcohol or other fixative and may be examined later with trichrome stain.[162,292] The same limitations and problems discussed with *Giardia* apply to *E. histolytica*. Fecal shedding of cysts is irregular. Three stools on alternate days identify most infections. Overdiagnosis may result from misidentification of leukocytes. Sigmoidoscopy or colonoscopy is useful for viewing the pathologic lesions and obtaining selective samples of mucus and biopsies of mucosal ulcers, which usually contain organisms.[112] Finding cysts does not confirm the diagnosis of symptomatic intestinal amebiasis. The key to establishing the diagnosis is finding motile trophozoites with ingested red blood cells. Culture techniques have been developed that identify infection in some cases when small numbers of cysts are missed in stool examinations, but these techniques are expensive and time consuming.[112] Serologic tests are not useful for identifying asymptomatic carriers but are positive in 85% to 95% of patients with dysentery and 90% to 100% of patients with liver abscess.[112,137,248] New antigen detection techniques can differentiate between *E. histolytica* and *E. dispar*.[158,162,180,292] Recently, PCR techniques have been developed and show greater than 95% sensitivity and specificity.[2,109,322]

Treatment of amebiasis is based on the location of infection and the degree of symptoms. Medications are divided into tissue amebicides, which are well-absorbed drugs (e.g., metronidazole, tinidazole, emetine, dehydroemetine, chloroquine) that combat invasive amebiasis in the bowel and liver, and poorly absorbed drugs (e.g., iodoquinol, paromomycin, diloxanide furoate) for luminal infections. In general, treatment is effective for invasive infections but disappointing for intestinal colonization. U.S. guidelines suggest that asymptomatic carriers should be treated, because a cyst passer represents a potential health hazard to others, and reinfection in the United States is uncommon. Routine screening of asymptomatic persons of high-risk groups is not cost effective, except perhaps for food handlers and persons returning from extended stay in endemic countries.[292]

The current drug of choice for asymptomatic carriers is iodoquinol (650 mg three times a day for adults and 40 mg/kg/day in three divided doses for 20 days for children). Side effects are mild and consist of abdominal pain, diarrhea, and rash. Diloxanide furoate (Furamide) is another drug of choice (500 mg three times a day for adults and 20 mg/kg/day in three divided doses for 10 days for children), but in the United States it is classified as an investigational drug, available only through the CDC. Side effects are limited to flatulence and other mild GI symptoms.[124] Paromomycin is also effective (500 mg three times a day for adults and 30 mg/kg/day in three divided doses for 7 days for children). Although metronidazole has been used in asymptomatic carriers with 90% success, most reserve this drug for invasive and symptomatic infections.

Invasive disease is treated with a tissue-active drug, followed by a luminal agent (in the same dosages as just listed). For oral therapy, metronidazole is the drug of choice (750 mg three times a day for adults and 50 mg/kg/day in three divided doses for 5 to 10 days for children), followed by iodoquinol. Tinidazole (1000 mg twice daily for 3 days) appears to be effective and is well tolerated for intestinal and hepatic amebiasis. Emetine and dehydroemetine (1 mg/kg/day, maximum 90 mg/day) are used parenterally in severe cases of amebiasis, primarily extraintestinal, followed by iodoquinol for 20 days. These two drugs have frequent systemic side effects, including the development of cardiac arrhythmias requiring hospitalization for cardiac monitoring. Because this class of drugs is related to ipecac, the drugs also cause vomiting.[198,292]

Another species, *Entamoeba polecki,* although usually nonpathogenic, has been suspected of causing lower intestinal symptoms in sporadic cases involving heavy infection.[268] Cysts are passed in stool and may be confused with *E. histolytica,* which they closely resemble. Successful resolution of symptoms has been reported with metronidazole followed by diloxanide furoate in the same dosages as for amebiasis and balantidiasis.

Cryptosporidium

Cryptosporidium is a coccidian parasite that belongs to the phylum Sporozoa. It is a reemergent enteric pathogen in humans. *Cryptosporidium parvum,* the only human pathogen of this genus, was originally described in 1912 but was first identified in humans in 1976.[45,58,59,128,151,234]

C. parvum causes a ubiquitous zoonosis with a worldwide distribution. *Cryptosporidium* infects a wide variety of young animals, including domestic calves, birds, piglets, horses, pigs, kittens, puppies, and wild mammals, such as raccoons, beavers, squirrels, and coyotes.[45,128] The prevalence of infection in human populations varies from 0.1% to 3% in cooler, developed countries (in Europe, North America) to 0.5% to 10% in warmer areas (Africa, Asia). The infection has been described in those who have contact with animals, such as veterinarians and farmers; infants in daycare centers; travelers to endemic areas; and patients who have AIDS or who are otherwise immunocompromised. It may infect large numbers of individuals in community-wide waterborne outbreaks.[45,59,128] The infective dose of *Cryptosporidium* for humans is low, similar to that seen with *Giardia* species. Sporulated oocysts are infective when passed in the stool, so fecal–oral contamination is the mode of transmission.[128] The different routes of transmission are waterborne, especially in large community outbreaks; person to person, especially in daycare centers, custodial institutions, and

hospitals; food-borne, through apple cider, uncooked sausage, and raw milk; sexual, with no association with specific behavior; and zoonotic.[45,110,128,194,195] In 1993 in Milwaukee, *Cryptosporidium* caused the largest waterborne outbreak of protozoal parasites in the United States.[203] The pathophysiologic mechanisms of diarrhea and malabsorption are not completely understood. The initial invasion of parasites may activate cellular and humoral immune and inflammatory responses, leading to cell damage with villi atrophy and crypt hyperplasia, ultimately producing malabsorption and osmotic diarrhea.[45,75,123,128,235]

The clinical manifestations depend on the patient's immune status, but asymptomatic infection occurs in both normal and immunocompromised hosts.[45,128] In immunocompetent persons, the usual incubation period of *Cryptosporidium* is from 5 to 28 days. Symptoms consist of watery diarrhea associated with cramps, nausea, flatulence, and, at times, vomiting and low-grade fever. The syndrome is generally mild and self-limited, with a duration of 5 to 6 days in some groups (range, 2 to 26 days). In contrast, immunocompromised hosts experience more frequent and prolonged infections, with profuse chronic watery diarrhea, malabsorption, and weight loss lasting months to years. Fluid losses can be overwhelming in a fulminant cholera-like illness, with high mortality. Cyst passage in stool usually ends within 1 week of resolution of symptoms but may persist for up to 2 months after recovery. Reinfection of an immunocompetent person has been documented.

Rarely, *Cryptosporidium* can infect the respiratory system, which may be fatal in the immunocompromised host. The other extraintestinal manifestations relate to involvement of the liver and biliary system, particularly in immunocompromised persons. Cholangitis may not respond to common luminal agents used to treat intestinal cryptosporidiosis, requiring sphincterotomy for therapy.[45,128]

Diagnosis in initial case descriptions was made by small bowel biopsy, but oocysts can be found in the stools routinely in intestinal infections, even though shedding may be intermittent. Concentration techniques, such as formalin-ether or sucrose flotation, and subsequent staining with modified acid-fast, Giemsa, or Ziehl-Neelsen techniques facilitate identification of *Cryptosporidium* oocysts. The Enterotest is also useful in the diagnosis of cryptosporidiosis. Newer immunologic techniques are faster and have adequate sensitivity and excellent specificity. Several other methods (flow cytometry using monoclonal antibodies, PCR, restriction fragment length polymorphism [RFLP] analysis) have been developed, but their efficacy in the clinical setting is not yet known.[39,45,113,128,183,329]

No clearly effective treatment has been found for cryptosporidiosis. Because this disease is usually mild and self-limited in immunocompetent hosts, only supportive care is needed. Anticryptosporidial agents, such as paromomycin (500 to 750 mg three or four times a day for 2 weeks), azithromycin (1200 mg daily for 4 weeks), and nitazoxanide (500 mg two times a day for 3 to 14 days)[108a] may be used in immunocompetent persons with persistent infection and in immunocompromised patients. Paromomycin, azithromycin, roxithromycin, ionophores, sulfonamides, and mefloquine have been tested against cryptosporidiosis, especially in patients with AIDS and chronic diarrheal disease, with variable but generally positive effects.[11,45,128,265,286,324] Further studies with these and other new agents, including clinical trials using immunotherapy options, are in progress.

Isospora belli

Isospora belli is a coccidian protozoal parasite. The first description of *Isospora* was in 1915. More recently, *I. belli* was identified as the pathogenic species for humans. It is an uncommon cause of diarrhea in humans, but its prevalence, like that of *Cryptosporidium*, has been increasing in immunocompromised patients.[57,123,206,209,323]

Humans are the only host (although there are a few reports in dogs), and infections are transmitted by fecal–oral contamination through direct contact of food and water. *I. belli* is endemic in areas of South America, Africa, and Asia. The prevalence is not precisely known but ranges from 0.2% to 3% in United States patients with AIDS and from 8% to 20% in Haitian and African patients with AIDS. This parasite has also been associated with outbreaks in custodial institutions, in daycare centers, and among immigrants. Infection rates in otherwise healthy persons with diarrhea are usually low. Most cases have been identified in tropical regions among natives, travelers, and the military.[123,209]

The life cycle and pathogenesis of *I. belli* are similar to those of *Cryptosporidium*. The organism invades mucosal cells of the small intestine, causing an inflammatory response in the submucosa and variable destruction of the brush border.[123]

In immunocompetent persons, the *I. belli* infection may be asymptomatic or cause mild transient diarrhea and abdominal cramps. Other symptoms include profuse watery diarrhea, flatulence, anorexia, weight loss, low-grade fever, and malabsorption.[123] Generally, infection is self-limited, ending in 2 to 3 weeks, but some persons have symptoms lasting months to years, clinically similar to giardiasis. Recurrences are common. Infections in immunocompromised patients tend to be more severe and follow a more protracted course.[206] Rarely, acalculous cholecystitis or reactive arthritis has been reported in isosporiasis.[21]

Diagnosis can be made by identification of immature oocysts in fresh stool. However, excretion may occur sporadically and in small numbers, so concentration techniques are usually required. Staining with modified Ziehl-Neelsen and auramine-rhodamine can also be useful. When stools are negative, the organism can be recovered from the jejunum through a biopsy or string test. Unlike the other intestinal protozoa, *I. belli* may cause eosinophilia.[112]

Successful treatment has been reported with TMP/SMX (160/800 mg four times a day for 10 days, then twice a day for 3 weeks in normal hosts). Other options are pyrimethamine (75 mg daily for 14 days) with folinic acid, metronidazole, and nitazoxanide (for patients allergic to sulfonamides). In patients with HIV infection, chronic lifetime suppression therapy is indicated with either TMP/SMX (160/800 mg three times a week) or pyrimethamine (25 mg) plus folinic acid (5 mg) daily.[108a,123,206]

Cyclospora cayetanensis

Cyclospora species were first discovered in moles in 1870 and were identified as human pathogens in 1979. They were initially thought to be blue-green algae (cyanobacteria-like organisms).[92,114,207,238,288,328] The life cycle and pathogenesis of *Cyclospora cayetanensis* are not completely understood.

The organism has been shown to be an important cause of acute and protracted diarrhea. *C. cayetanensis* is endemic in many developing countries in all continents, with the highest rates occurring in Nepal, Haiti, and Peru. In the United States, most of the native outbreaks have been from areas east of the

Rocky Mountains, usually associated with ingestion of contaminated imported raspberries.[27,41,92,123,140,141] Fecal–oral transmission also occurs through water and soil.[288] *Cyclospora* infection is closely associated with diarrhea in travelers to endemic areas.[92,114,145,207,238,288,328]

The onset of diarrhea is usually abrupt, with symptoms lasting 7 weeks or even longer.[92,123] In patients with AIDS, the duration may be longer and the severity greater.[206] Small spherical organisms can be detected in fresh or concentrated stool, and they show variable staining with acid-fast methods. *C. cayetanensis* stains best with carbolfuchsin.[328] Phase-contrast microscopy and autofluorescence are also useful in the diagnosis.[112] A PCR method is still used only for research.[209]

The treatment of choice is TMP/SMX (160/800 mg four times a day for 10 days). This treatment provides more rapid clinical and parasitologic cure, with fewer recurrences.[92,114,207,238,288] In patients with AIDS, chronic suppression with TMP/SMX may be required.[206]

Miscellaneous Parasitic Agents

Microsporidia. More than 100 genera and 1000 species of microsporidia exist in the phylum Microspora. Most species infect insects, birds, and fish. Since the first description in humans in 1985, only 12 species have been reported to infect humans: *Enterocytozoon bieneusi,* three *Encephalitozoon* species, three *Nosema* species, two *Trachipleistophora* species, *Pleistophora, Vittaforma corneae,* and microsporidial species. Microsporidia cause a wide spectrum of disease, but only two, *E. bieneusi* and *Encephalitozoon intestinalis,* have been found to cause diarrhea in humans.[66,123,321,323]

Transmission is thought to be fecal–oral or urinary–oral[66,67] and the infection zoonotic. Waterborne transmission also occurs.[209] Prevalence of microsporidiosis in patients who have AIDS and chronic diarrhea is 7% to 50%.[17,67,321]

The clinical manifestations of intestinal microsporidiosis are chronic diarrhea, loss of appetite, weight loss, malabsorption, and fever.[17,321] Acute self-limited diarrhea has been reported in immunocompetent hosts. Other infections include keratoconjunctivitis, hepatitis, peritonitis, myositis, CNS infection, urinary tract infections, sinusitis, and disseminated disease. Diagnosis involves trichrome staining of concentrated stools or intestinal biopsy sampling, but electron microscopy is considered the gold standard. Immunologic and molecular biologic techniques are still under evaluation.[105,321]

The most effective drug is albendazole (400 mg twice a day for 2 to 4 weeks). It is effective against most species, but results are variable with diarrhea from *E. bieneusi.*[68] Other drugs show different efficacy and include thalidomide, fumagillin, atovaquone, metronidazole, furazolidone, azithromycin, itraconazole, and sulfonamides.[55,66]

Sarcocystis. Few human infections with *Sarcocystis* have been reported. Infection may be asymptomatic or associated with diarrhea, abdominal pain, nausea, and bloating. Symptoms typically improve within 48 hours of onset of illness. Diagnosis is based on identification of cysts in concentrated feces. No specific treatment has been established, but TMP/SMX and furazolidone have had variable efficacy.[280,323]

Balantidium. *Balantidium coli* is a rare pathogen in humans.[297,323] Although many aspects of the epidemiology are unclear, pigs appear to be the primary reservoir and source of human infection. Clinical features also resemble amebiasis, with a spectrum including asymptomatic infection, chronic intermittent diarrhea of variable intensity, acute dysentery with mucosal invasion, and, rarely, metastatic abscesses. Diagnosis is made by observing the organism in stool. Trophozoites are seen much more often than cysts. Recommended treatment is tetracycline (500 mg four times a day for 10 days) or metronidazole (750 mg three times a day for 10 days).[280,323]

Blastocystis. The role of *Blastocystis hominis* in diarrheal disease is still controversial, although it is often identified in stool samples. *B. hominis* has not been directly correlated with symptoms,[280,323] which could be caused by other undetected pathogens. When found in large numbers as the sole pathogen, *B. hominis* is suspected as the potential etiologic agent of diarrheal illness.

Dientamoeba. *Dientamoeba fragilis* occasionally causes diarrhea, occurring characteristically in residents of or visitors to developing tropical regions. It may be found in stools of persons without enteric symptoms. Because cyst forms have not been identified, the mode of transmission remains unknown. Illness caused by the parasite typically resembles giardiasis, but treatment of these two parasitic infections is different. Iodoquinol and tetracyclines are effective against *D. fragilis.*[280,323]

The references for this chapter can be found on the accompanying DVD-ROM.

Nutrition, Malnutrition, and Starvation

63

E. Wayne Askew

How does it feel to starve?

> *I am hungry. I am always hungry. . . . At times I can almost forget about it but there is nothing that can hold my interest for long. . . . I am cold. . . . My body flame is burning as low as possible to conserve precious fuel and still maintain my life processes. . . . I am weak. I can walk miles at my own pace in order to satisfy laboratory requirements, but often I trip on cracks in the sidewalk. To open a heavy door it is necessary to brace myself and push or pull with all my might. I wouldn't think of throwing a baseball and I couldn't jump over a twelve inch railing if I tried. This lack of strength is a great frustration. It is often a greater frustration than the hunger . . . and now I have edema. When I wake up in the morning my face is puffy. . . . Sometimes my ankles swell and my knees are puffy. . . . Social graces, interests, spontaneous activity and responsibility take second place to concerns about food. . . . I lick my plate unashamedly at each meal even when guests are present. . . . I can talk intellectually, my mental ability has not decreased, but my will to use my ability has.*
>
> Observations by a test subject, Minnesota Starvation Study, after 24 weeks of semi-starvation (1570 kcal/day, 24% weight loss) (In *The Biology of Human Starvation,* vol. II[43])

IMPORTANCE OF NUTRITION IN STRESSFUL ENVIRONMENTS

Nutrition has a profound underlying importance to proper human physiology and functioning on a day-to-day basis in everyday life; it becomes even more important when humans work or recreate in environmental extremes.[10] The central role of nutrition is often unappreciated in wilderness expedition planning and may even be thought by some to be of minor importance. Many enthusiasts do not consider food as critical as gear and equipment, medical supplies, physical fitness, and other logistical considerations. In temperate environments, where food and water are plentiful and re-supply is feasible, the importance of nutrition may seem to diminish compared with other aspects of wilderness medicine. However, when a stressful physical environment is superimposed on the physically demanding tasks associated with wilderness activities, the role of nutrition rapidly becomes of prime importance for maintenance of performance and for prevention of disease and injury, as evidenced from the description of Napoleon's disastrous 1812 winter retreat from Moscow:

> *The ice and deep snow with which the plains of Russia were covered, impeded . . . calorification in the capillaries and pulmonary organs. The snow and cold water, which the soldiers swallowed for the purpose of allaying their hunger or satisfying their thirst . . . contributed greatly to the destruction of these individuals by absorbing the small portion of heat remaining in the viscera. The agents produced the death of those particularly who had been deprived of nutriment.*
>
> Baron D. J. Larrey, Inspector General, Napoleon's Military Medical Staff (In *Hypothermia and Warfare, Napoleon's Retreat from Moscow, 1812*[61])

Fortunately, situations encountered in wilderness activities are usually less grim than those faced by Napoleon's army. There is usually some food available, and few enter these environments unprepared. However, misfortune, combined with suboptimal or haphazard nutritional planning, can spell disaster for even the best-prepared adventurer. A wrong turn on the trail, an injury, unanticipated terrain, an unexpected storm, or a downed airplane can deplete or isolate a victim from anticipated food sources. Food becomes an overriding consideration in a survival situation, particularly as the supply is exhausted. Although a shortage of food is certainly of concern, it does not necessarily imply impending disaster. Humans are remarkably adaptable and can subsist on non-ideal dietary patterns for prolonged periods without disastrous effects on health and performance. As long as some baseline level of energy intake is present, at least a minimal intake of vitamins and minerals will be ensured, forestalling the eventual onset of malnutrition and clinical nutrient deficiency states. Hunger is not comfortable; weight loss results, and performance may suffer, but a food-deprived individual can still function for an extended period of time. One purpose of this chapter is to review some of the consequences of suboptimal nutrition that might be anticipated under various degrees of food restriction. The medical planner might use this information to anticipate limitations in expedition progress or capabilities and to recognize the state of health of victims of starvation or food restriction. Equally important is the proper way to re-aliment a victim after a prolonged period of unintentional starvation. This can be critically important in a rescue situation, where transport of a victim to a medical facility requires several days of refeeding en route.

Although the goal of expedition food planning is to meet daily energy and micronutrient requirements, it is not always possible to provide optimal nutrition because of logistical or situational constraints. Three nutritional states or situations may be encountered in the wilderness: (1) optimal nutrition for effective functioning in environmental extremes, (2) malnutrition or suboptimal nutrition, and (3) starvation, or lack of nutrition. Dietary planning for both wilderness expeditions and emergencies will be discussed.

Environmental Stress and Nutrient Requirements

The physical and physiological condition of an individual (e.g., bodyweight, strength, coordination, fluid and electrolyte balance, and core temperature) plays a significant role in determining nutritional requirements to maintain homeostasis. It also directly influences survival time in the absence of food or water. In the short run, the most important nutrient is water.[9] If an adequate supply of water is not available, all discourse on the physiology of starvation and malnutrition is pointless, because death from dehydration will occur prior to depletion of energy stores. Humans can survive food deprivation for extended periods of time, weeks or even months, depending on their level of body fat. A non-obese adult may live as long as 60 to 70 days while fasting in a clinical setting.[37] At the end of this time, almost all body fat and a third of the lean body mass would be lost.[36] James Scott, a victim of unintentional situational starvation, was marooned in a snow cave in the Himalayas with water but no food. He survived 43 days without food while losing one third of his bodyweight, although he was near death at the time of rescue.[67] Death from starvation in non-obese individuals is imminent if approximately 50% of bodyweight has been lost. This usually corresponds to a body mass index (BMI) of 12 kg/m^2, although under some circumstances, a BMI of as low as 11 may be encountered prior to death.[25]

Time to death after complete water deprivation is measured in days—estimates run from 6 to 14 days, depending on the rate of water loss.[36] Water is critical because it maintains the homeostasis of the internal environment.[9] It provides an aqueous medium to transport heat from cells to blood, to solvate and pass nutrients between blood and cells, to serve as a medium for intracellular reactions, and to transfer metabolic products for redistribution or excretion via urine. Both the quantity of reactants and the volume of fluid in which they are dissolved influence cellular chemical reaction rates; hence, imbalances in hydration status can alter cellular and tissue function, such as the body's ability to regulate temperature. Muscular contraction is dependent on the transformation of chemical energy (ATP) to mechanical energy. Nearly three fourths of the energy used for muscular contraction is released as heat. Unless localized heat production from metabolism and muscular contraction is dissipated, the heat burden can be structurally damaging to enzymes or other proteins. Water absorbs heat produced at the cellular level and transfers it to the surface of the skin, where it can be dissipated to the external environment. The importance of water is discussed in Chapter 64. The focus here is on energy restriction and assumes an adequate supply of water. For planning purposes, most wilderness expeditions require 3 to 5 L (about 3 to 5 qt) of potable water per person per day.

As a result of advances in food processing, preservation, and nutrient fortification, modern camping foods and the military equivalent, field rations, can support health and performance in a variety of temperate environments, even if not consumed to complete caloric adequacy.[50] However, nutrition that was marginally adequate in a temperate environment may rapidly become inadequate in wilderness environments characterized by extreme temperatures and terrain.[49,51] Rodahl and Issekutz[65] state, "While short-term nutritional deficiencies in men at room temperature appear to have little or no detrimental effect on the capacity to do short-term, heavy work, there is a marked reduction in physical work capacity when a nutritional deficiency or nutritional stress is superimposed on a cold stress." Superimposed stressors, such as extreme heat, cold, altitude, sleep deprivation, physical exertion, and food restriction, influence nutrient requirements[7] and can jeopardize performance.[10,29,46,49,51,53] The complex interrelationship between environment and nutrition, and its effect on human physiology and performance, is depicted in Figure 63-1. Stressors in the form of environmental extremes can have serious consequences on health and performance.

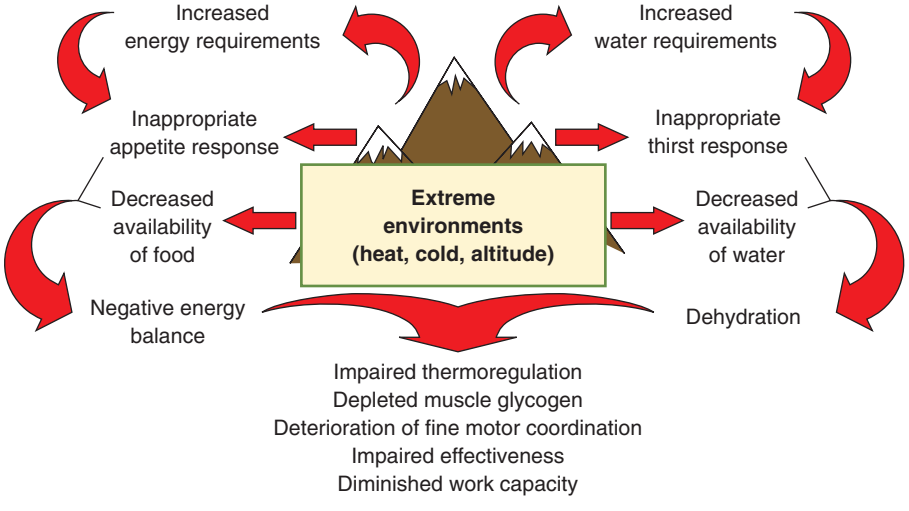


Figure 63-1. The influence of extreme wilderness environments on food and fluid intake and on physical and mental performance. (Redrawn from Askew EW: In Hickson JF Jr, Wolinski I [eds]: Nutrition in Exercise and Sport. Boca Raton, FL, CRC Press, 1989, pp 367–384.)

Energy and fluid deficits arising from the interaction of environment and nutrition can negatively impact both physical[29] and mental[53] performance. Volitional physical activity suffers under caloric deprivation. Motivation may be more acutely influenced by undernutrition than actual physical performance is.[53] Nutritional deficits may have a greater effect on what individuals are *willing to do* (i.e., on their perceived mood, symptoms, and self-motivation) than on what they *can do* (psychomotor performance).[71]

Nutritional Considerations in Planning for Wilderness Activities

Cold and altitude stressors and their influence on macronutrient and vitamin requirements have been a focus of considerable military and civilian research.[8] Research conducted primarily in the era since World War II (WWII) and the Korean conflict has established that vitamin and mineral requirements are not significantly increased by cold exposure, although caloric requirements for thermogenesis may be elevated.[51] Work in cold environments can be adequately supported by various combinations of fat, carbohydrate, and protein, although certain combinations of macronutrients may be more beneficial than others in helping a person withstand cold exposure.[4] As an example, Mitchell and colleagues[55] demonstrated that cold tolerance (the length of time body core temperature could be defended during a controlled cold challenge) was favored by previous diets high in fat as opposed to diets higher in carbohydrate or protein. Subsequent research has indicated that the macronutrient source is less important in the cold than consuming enough total calories to support activity and thermogenesis.[4,33] However, when wilderness activities shift from a sea-level, cold-weather environment to a moderate- or high-altitude, cold-weather environment, the macronutrient diet should be reconsidered. Fat is an efficient and well-tolerated energy source during relatively low-power-output, cold-weather activities at sea level; however, it is not as well tolerated at high altitude.[5] Substituting carbohydrate for fat and, to a certain degree, for protein can theoretically provide metabolic advantages to oxygen economy when an individual is working at altitude.[3] Carbohydrate is a more efficient fuel at altitude than fat, because it is already partially oxidized (i.e., it contains a higher ratio of oxygen atoms to carbon atoms) and therefore requires less oxygen to combust its carbon skeleton to CO_2, H_2O, and energy. Metabolizing carbohydrate for energy requires approximately 8% to 10% less inspired oxygen than is required to obtain a similar amount of calories from fat. A high-carbohydrate diet can reduce the symptoms of acute mountain sickness, can enhance short-term high-intensity work as well as long-term submaximal efforts, and can lower the effective "felt" elevation by as much as 300 to 600 m (984 to 1969 ft) by requiring less oxygen for metabolism.

Initial altitude exposure frequently results in anorexia and subsequently reduces energy and carbohydrate intake.[20] Anorexia (and thus food intake) usually improves with time and acclimatization (3 to 7 days at altitude), but, depending on the altitude, may never match that at sea level. Weight loss and performance decrements are quite common under these conditions. Carbohydrate supplementation of the diet at elevations exceeding 2200 m (7218 ft) is usually an effective method to increase carbohydrate and total energy intakes.[3,20,27] Some,[26] but not all,[77] studies of carbohydrate supplementation at altitude have demonstrated a decrease in the adverse symptoms resulting from acute altitude exposure. Enhancement of short-term, high-intensity performance,[26] as well as long-term, submaximal performance,[3] by carbohydrate supplementation has also been noted in some studies involving altitude exposure. The beneficial effects of carbohydrate at altitude most likely depend on the type of exercise performed (intensity and duration) and the degree of prior muscle glycogen depletion experienced by the test subject.

Muscle glycogen is related to the caloric adequacy of an individual's prior diet; carbohydrate intake usually parallels the dietary intake of the antecedent diet.[83] It is reasonable to consume a mixed diet with snacks high in carbohydrate. The most effective form of carbohydrate supplementation is usually liquid beverages; people will drink even when they are reluctant to eat.[20,27] Increasing fluid intake along with carbohydrate intake is also beneficial at altitude, where increased fluid losses occur as the result of diuresis and of respiration in the dry (low-relative-humidity) atmosphere.[9]

National nutritional recommendations have been established. The dietary reference intakes (DRIs) are reference values for nutrient intakes that can be used to assess and plan diets for healthy people. Publications that list DRIs can be obtained through the National Academy website (http://newton.nap.edu/category.html?id=fn) and the National Institutes of Health website (http://ods.od.nih.gov/Health_Information/Dietary_Reference_Intakes.aspx). The DRIs include four categories: estimated average requirement (EAR), recommended dietary allowance (RDA), adequate intake (AI), and tolerable upper limits (UL). EAR is the nutrient intake level estimated to meet the needs of 50% of the individuals in a population group. RDA is the nutrient intake level sufficient to meet the needs of 97% to 98% of the individuals in a population group. AI is an estimate of adequate intake when an EAR cannot be established. The AI and the RDA are similar but not identical. The UL is the highest daily consumption level of a nutrient; when the UL is exceeded, the nutrient poses a risk of adverse health effects.

Special Nutritional Requirements for Female Wilderness Travelers

Although our understanding of the differences between male and female nutritional requirements is incomplete, sex differences do exist for certain nutrients, such as iron, calcium, folate, and vitamin B_6 under normal environmental living conditions.[56] Differences are particularly notable for women using oral contraceptives[52] and those who are pregnant or lactating.[23] The health implications for pregnant travelers planning a trip to altitude are beyond the scope of this chapter; these individuals should consult a knowledgable pediatrician or obstetrician.

Investigations of nutritional requirements at environmental extremes have been conducted on men, but comparatively little research has been directed toward women. The few studies that have been done suggest that dietary nutrient intakes of women at moderate altitude are similar to those at sea level,[44] implying that specific sex requirements that exist at sea level may be even more important at altitude, particularly if there is scarcity of nutrient-dense foods or if appetite is blunted. Some sex-specific nutrient requirements are known. For example, research conducted in the late 1960s identified that female service members deployed to places at moderate to high altitude required supplemental dietary iron to optimally support the hematopoietic response to hypoxia.[34] Subsequently, research on iron require-

ments and the thermogenic response to cold have also identified iron as a key micronutrient for women in a cold environment.[17,48] Women, because of their reduced body size, usually consume fewer total food calories than men. They are therefore at an increased risk for reduced vitamin and mineral intake. Fortunately, the need for these vitamins and minerals (with the exception of iron) is related to lean body mass, and women usually have a lower lean body mass than men.

Because the choice of available foods may be limited during expeditions into the wilderness, female travelers should include a multivitamin supplement in their provisions. It should contain at least 50% of the female RDA for iron, zinc, folate, vitamin B_6, and calcium. Antioxidant nutrients (see Vitamins and Their Relationship to Health and Physical Performance, later), including extra vitamin C and E and perhaps certain carotenoids, such as lutein and zeaxanthan, provide additional insurance against oxidative stress. The compositions of some typical multivitamin and mineral supplements are shown in Figure 63-2. The values shown reflect the daily value (DV) of the nutrients—that is, the recommended daily amount for most adults needing 2000 to 2500 kcal/day.

Multivitamins often contain relatively low amounts of calcium compared with the female RDA of 1000 to 1300 mg/day, so it may be beneficial for female travelers to include an additional bioavailable calcium supplement containing 250 to 500 mg of calcium per dose. Commercially available chocolate- or caramel-flavored calcium "chews" are convenient and contain a small amount of carbohydrate, as well as "bone-friendly" nutrients such as vitamins D and K. The U.S. RDA for calcium intake for adults under age 50 is 1000 mg daily, and people over 50 should ingest 1200 mg daily. Calcium supplements are best taken in small (no more than 500 mg) divided doses throughout the day. If an iron supplement is being taken in a multivitamin or by itself, it is best not to take it at the same time as the calcium supplement, because the absorption of each is unpredictable and may be less than optimal in the presence of the other.[64]

The nutrients in a vitamin–mineral supplement should be present at close to the DV level (100% or less). This ensures an adequate intake of major micronutrients with minimal risk of adverse nutrient interactions. To avoid unpredictable absorption conflicts, such as between iron and calcium, the recommended dose should not be exceeded. Synthetic vitamins are structurally the same as, and cost less than, so-called natural vitamins. Generic brands are also generally less expensive and equally effective. The addition of herbs, enzymes, or amino acids accomplishes little but adds cost. Some supplements containing phytonutrients extracted from plants may be appropriate when the logistics of the trip offer little or no chance to secure fresh fruits and vegetables. A source of information on vitamin and mineral supplements is ConsumerLab, which provides independent test results and information to help consumers and health care professionals evaluate health, wellness, and nutrition products. The results of its tests, including brands that have passed testing, are available at www.consumerlab.com.

Supplements can lose potency over time, so check the expiration date on the label. Figure 63-3 provides guidance for reading and interpreting supplement labels. The initials USP (for the testing organization U.S. Pharmacopeia) or words such as "release assured" or "proven release" indicate that the supplement is easily dissolved and absorbed.

Energy: How Critical Is It?

When planning for a prolonged wilderness outing, the following question should be asked: "If we run short on food, will we suffer severe consequences in our progress along our route and experience difficulty carrying our heavy packs?" For optimal performance, it is total energy intake, especially carbohydrate intake, that is the key for sustaining high-level work capacity for extended periods. However, performance across a broad spectrum of back-country tasks, including load-bearing work, is not always severely degraded by *short* periods of suboptimal energy and carbohydrate intake. A review of the effect of energy restriction on military work performance indicated that soldiers can maintain relatively normal work capacities for short time periods (<10 days) of food restriction.[29] The Minnesota starvation studies conducted during WWII demonstrated that energy deficits resulting in a loss of less than 10% of bodyweight did not greatly impair physical performance; however, underconsumption of calories for longer periods producing continued loss of bodyweight created significant deficits in physical performance,[78] as evidenced by the observations of one test subject who participated in the 1570 kcal/day, 24-week semistarvation study, resulting in 24% bodyweight loss: "Then came February twelfth, the starting date of semi-starvation . . . only two meals a day from now on. For the first two weeks the new life was fun. I was losing weight, of course, but I still had lots of energy. Then came the day when I lost my 'will to activity'—I no longer cared to do anything that required energy."

The degree of reduction of work capacity depends on the degree of caloric restriction, the carbohydrate content of the food available, and the power output of the work demanded. Studies of food restriction in military scenarios have revealed that restricted energy and dietary carbohydrate content over a 30-day period supported light to moderate activity level without evidence of greatly impaired physical performance capabilities.[12] On the other hand, longer periods (8 weeks) of caloric restriction coupled with higher levels of energy expenditure (U.S. Army Ranger training) have been associated with significantly reduced physical performance capacity.[59] Friedl[29] reviewed the influence of reduction in bodyweight resulting from reduced food intake on muscle strength and aerobic capacity.

Because studies have varied with regard to experimental conditions and performance measures, it is difficult to draw conclusions about the relationship between energy deficit and performance. Some indicators of performance, such as grip strength, appear to be well preserved until nutritional status is severely compromised. Other measures, such as maximal lift test, maximal jump height, isometric leg extension, and maximal oxygen uptake, appear to be more sensitive predictors of impaired performance.[41] In general, in non-obese individuals, strength seems to be rather well maintained at a bodyweight loss of up to 5%. This may be good news for wilderness travelers who are concurrently on a gradual weight loss program. Shedding fat actually "lightens the load" without diminishing strength. However, when dieting or restricting food results in loss of *lean* body mass, performance decrements result. Aerobic capacity and strength are reduced when loss of bodyweight exceeds 10%. Friedl[29] concluded that changes in maximum oxygen consumption (VO_{2max}) in response to modest caloric restriction generally influence performance to a lesser degree than do reductions in muscle strength in response to weight loss. The primary concern of weight loss due to inadequate energy

Supplement Facts

Serving Size 1 tablet

A

Amount Per Tablet	% Daily Value
Vitamin A 3,000 I.U.	**60%**
Vitamin C 60 mg	**100%**
Vitamin D 400 I.U.	**100%**
Vitamin E 30 I.U.	**100%**
Thiamin 1.5 mg	**100%**
Riboflavin 1.7 mg	**100%**
Niacin 20 mg	**100%**
Vitamin B6 2 mg	**100%**
Folic Acid 400 mcg	**100%**
Vitamin B12 6 mcg	**100%**
Pantothenic Acid 10 mg	**100%**
Calcium 450 mg	**45%**
Iron 18 mg	**100%**
Zinc 15 mg	**100%**

Supplement Facts

Serving Size 1 tablet

B

Amount Per Tablet	% DV	Amount Per Tablet	% DV
Vitamin A 3,000 I.U.	60%	Iodine 150 mcg	100%
Vitamin C 120 mg	200%	Magnesium 100 mg	25%
Vitamin D 400 I.U.	100%	Zinc 15 mg	100%
Vitamin E 50 I.U.	167%	Selenium 25 mcg	36%
Vitamin K 100 mcg	125%	Copper 2 mg	100%
Thiamin 1.5 mg	100%	Manganese 2 mg	100%
Riboflavin 1.7 mg	100%	Chromium 120 mcg	100%
Niacin 20 mg	100%	Molybdenum 25 mcg	33%
Vitamin B6 2 mg	100%	Chloride 36 mg	1%
Folic Acid 400 mcg	100%	Potassium 40 mg	1%
Vitamin B12 6 mcg	100%	Boron 150 mcg	*
Biotin 30 mcg	10%	Nickel 5 mcg	*
Pantothenic Acid 10 mg	100%	Silicon 2 mg	*
Calcium 250 mg	25%	Tin 10 mcg	*
Iron 18 mg	100%	Vanadium 10 mcg	*
Phosphorus 77 mg	8%	Lutein 250 mcg	*

*Daily Value (DV) not established

Supplement Facts

Serving Size 1 tablet

C

Amount Per Tablet	% DV	Amount Per Tablet	% DV
Vitamin A 3,000 I.U.	60%	Iodine 150 mcg	100%
Vitamin C 120 mg	200%	Magnesium 100 mg	25%
Vitamin D 400 I.U.	100%	Zinc 15 mg	100%
Vitamin E 50 I.U.	167%	Selenium 25 mcg	36%
Vitamin K 25 mcg	31%	Copper 2 mg	100%
Thiamin 1.5 mg	100%	Manganese 2 mg	100%
Riboflavin 1.7 mg	100%	Chromium 120 mcg	100%
Niacin 20 mg	100%	Molybdenum 25 mcg	33%
Vitamin B6 2 mg	100%	Chloride 36 mg	1%
Folic Acid 400 mcg	100%	Potassium 40 mg	1%
Vitamin B12 6 mcg	100%	Boron 150 mcg	*
Biotin 30 mcg	10%	Nickel 5 mcg	*
Pantothenic Acid 10 mg	100%	Silicon 2 mg	*
Calcium 100 mg	10%	Tin 10 mcg	*
Iron 9 mg	50%	Vanadium 10 mcg	*
Phosphorus 77 mg	8%	Lutein 250 mcg	*

*Daily Value (DV) not established

Figure 63-2. Representative labels for multivitamin and mineral supplements. Supplement A is a typical multivitamin formulation, with most of the major nutrients of concern supplied at 100% of the daily value (DV—the amount of a nutrient recommended for daily consumption by most adults needing 2000 to 2500 kcal/day). Supplement B is a slightly more complex supplement containing greater than 100% of the DV for the antioxidant nutrients. It also contains additional minerals and carotenoids believed to be essential, but for which a DV has not been established. Supplement C is similar to Supplement B, but it contains less iron and calcium, so it would be an appropriate choice for a man. Supplements A and B would be better choices for a woman.

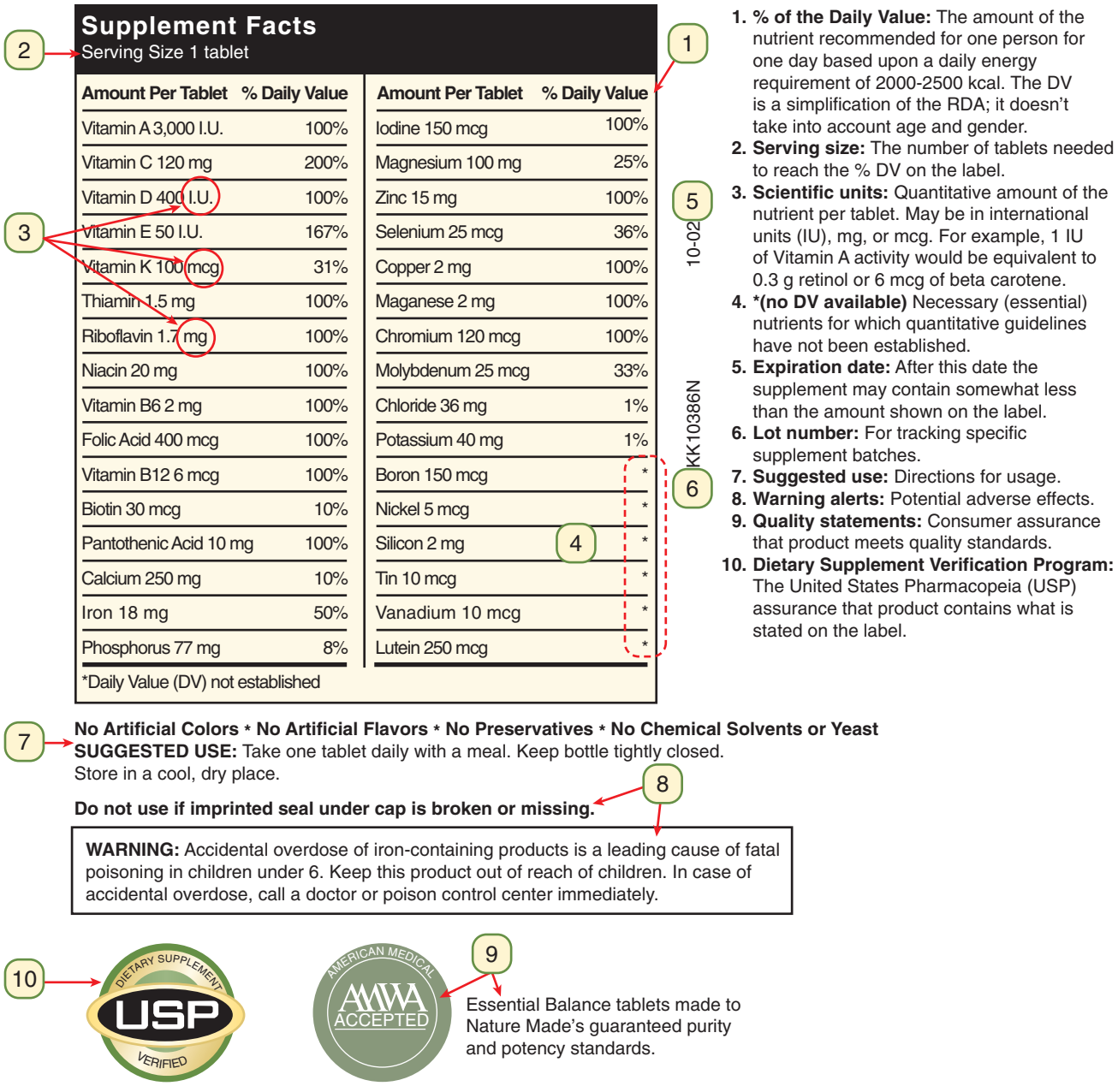


Figure 63-3. An annotated multivitamin and mineral supplement label showing some key components and pertinent information.

consumption occurring during extended wilderness activities appears to be loss of muscle strength. Significant loss of muscle strength can be expected to occur following 5% to 10% bodyweight loss.[29] Significant declines in aerobic capacity can also occur following weight losses of this magnitude, but the decline in aerobic capacity appears to have relatively little effect on individual performance at moderate (<50% VO_{2max}) sustainable workload levels.[29] In practical terms, this may mean that a gradual trek to the summit may not be precluded by a prior food restriction accompanied by significant loss of bodyweight, but that a short-term, all-out push for the summit to avoid impending bad weather would most likely be compromised.

Factors other than strength and aerobic capacity should also be considered when evaluating the effects of energy restriction on performance. Weight losses of 6% or less over periods of 10 to 45 days generally do not produce significant degradations in cognitive performance.[71] However, habitual or forced consumption resulting in 50% loss of energy requirements may significantly degrade cognitive performance.[53] Reduced food intake, when coupled with other stressors such as high rates of

energy expenditure and sleep deprivation, can also impair immune function.[39,45,46]

Carbohydrate: Critical for Performance

Both the length of time provided for dietary adaptation to carbohydrate restriction and the level of carbohydrate in the diet can influence the level of aerobic endurance performance.[2] Aerobic endurance performance can be reduced by 40% after only 4 days on a total-calorie-adequate but low-carbohydrate diet (i.e., with carbohydrates providing 10% of the kilocalories).[32] When a diet that was similarly adequate in total calories, but with carbohydrates providing only 5% of the kilocalories, was fed for 2 weeks, performance was also reduced, but only by approximately 15%, presumably because of metabolic adaptations to the shift in energy sources that occur with time.[63] Carbohydrate is important to performance, but the impact of any reduction in dietary carbohydrate intake depends on (1) the period of time over which it is reduced, (2) the absolute level of carbohydrate in the diet, and (3) the power output needed to accomplish the task at hand.

Reduced carbohydrate intake, more than the reduction of any other macronutrient (with the exception of water), has the potential to negatively influence muscle glycogen levels and endurance.[2] There is abundant evidence in the sports medicine literature to permit extrapolation to similar wilderness activities and to conclude that certain types of performance, such as backpacking, cross-country skiing, and climbing, may be influenced by an acute shortage of carbohydrate in the diet, depending on the intensity of the workload (% VO_{2max}) in which the individual is engaged. Inadequate carbohydrate in the diet, coupled with successive days of intense prolonged exercise, results in a gradual reduction of glycogen stores, deterioration of performance, and increased perception of fatigue. Perceived or "felt" exertion for certain wilderness activities, such as load-bearing work, may reasonably be assumed to be a function of the dietary carbohydrate intake and its subsequent effect on muscle glycogen levels. To avoid fatigue and extend or enhance performance, carbohydrates may be ingested before, during, and after moderate to heavy intensity aerobic exercise.[40] This requires daily consumption of approximately 500 to 600 g of carbohydrate (about 60% of the kilocalories in a day's food). The actual amount of carbohydrate required depends on the individual's body size and energy needs. To put this into perspective, a review of typical dietary carbohydrate intakes of male soldiers fed a variety of rations during 18 field studies in temperate, hot, and cold environments revealed intakes ranging from 244 to 467 g/day.[15] Similar data are not as well documented for nonmilitary wilderness activities, but it is probable that daily carbohydrate intakes would be similar and considerably less than the 500 to 600 g/day recommended for optimal physical performance, because total caloric intake during work in the outdoor environments is often less than that required to maintain energy balance.[50] Most people do not selectively consume low-carbohydrate diets during wilderness activities, but total carbohydrate intake is often low because of its relationship to total energy intake and because of limited high-carbohydrate food choices. Some backpackers seek to maximize food and caloric density because of the weight of, and space available in, their packs. Calorie-dense food item choices that achieve this goal are often high in fat and relatively low in carbohydrate. Inadequate food consumption in military field exercises has been ascribed to poor ration palatability and variety, menu boredom, not enough time to eat or prepare meals, anxiety, and intentional dieting to lose weight.[50] Similar factors may be operative in wilderness expeditions. Countermeasures to reduce the effect of these factors should be taken into consideration during ration planning. To sustain short-term performance, a shortfall of energy (calories) is not as significant a concern as the lack of carbohydrate.[29]

Field studies of carbohydrate intake and performance are difficult to conduct because of the many uncontrolled variables encountered outside the laboratory. Therefore, definitive field studies demonstrating a positive effect of dietary carbohydrate supplements on wilderness performance are lacking.[6] However, when field conditions can be imitated in well-controlled laboratory settings, results suggest that carbohydrate supplementation benefits performance. Such a study was conducted using 18 physically fit U.S. Army Special Operation Forces soldiers as test subjects.[58] It was designed to test the concept that soldier performance would benefit from carbohydrate supplementation under conditions simulating field operations. The subjects were fed a diet controlled to simulate a "typical" daily dietary intake of carbohydrate and protein encountered during field operations (327 g carbohydrate per day, 201 g fat per day, and 118 g protein per day, furnishing 3657 kcal/day). The soldiers exercised daily (11 days total) under conditions designed to simulate field energy expenditure patterns (intermittent- as well as sustained-activity patterns of varying intensity levels). Each test subject received one of three beverage supplements during the program: placebo (0 g carbohydrate), carbohydrate once a day immediately after exercise (180 g carbohydrate), or carbohydrate divided into several doses taken after the morning exercise session and at intervals during the afternoon exercise session (180 g carbohydrate in divided doses). A drop in the respiratory exchange ratio in all treatments was observed, indicating that the soldiers were experiencing a carbohydrate shortage in response to the field simulation and were switching to fat energy sources to sustain energy expenditure. However, the provision of supplemental carbohydrate permitted a higher level of physical performance or aerobic power to be attained. Run times to exhaustion were increased approximately 6% with the single carbohydrate feeding and 17% with the divided-dose administration. The ingestion pattern of the carbohydrate supplement as well as the carbohydrate itself appeared to influence performance, indicating that a supply of easily consumed carbohydrate (supplement or food) ingested before, during, and after field activities is an effective method to sustain or boost physical performance.

Fat: A Special Place in Wilderness Exploration?

Historically, wilderness explorers and expeditions have relied heavily on foods higher in fat content than current sports nutrition and healthy dietary guidelines recommend. One early 20th-century explorer, Vilhjalmur Stefansson, became such a strong advocate of meat-based high-fat diets that he volunteered himself for an unusual experiment in 1928.[73] Stefansson consumed an all-meat diet for a year with no detectable adverse medical consequences. Stefansson's championing of high-fat diets for work in cold regions led to a WWII-era U.S. Army and Canadian test of a high-protein and high-fat pemmican diet (low in carbohydrate) for work in the cold, with less-than-encouraging results.[42] Predictable fatigue occurred when the high-fat, low-carbohydrate diet was abruptly thrust on the

soldiers. Notwithstanding the military's lack of enthusiasm for high-fat food, recent arctic explorers such as Will Steger have relied on pemmican for nutritional support during cold-weather polar expeditions.[74]

The dispute concerning the suitability of high-fat diets continues. A review of numerous studies and the composition of the diets and experimental conditions under which they were conducted indicates that two factors are critical to the satisfactory utilization of high-fat diets: prior adaptation to fat in the diet and the power level (%VO_{2max}) at which the subjects were required to work.[62] Humans permitted to adapt gradually to increasing levels of fat in the diet for approximately 3 weeks can function much better than if the dietary change is abrupt. High power outputs require the capacity for repeated bursts of anaerobic metabolism, which is best fueled by carbohydrate (i.e., via glucose and the glycolytic cycle). Fat metabolism depends for the most part on the ready availability of oxygen. The ability to take up oxygen becomes limiting at high power outputs. A more recent military study of fat content of the diet confirms that a subcaloric, relatively high fat diet (1976 kcal/day; 46% fat) can maintain moderate physical performance (including load-bearing work) for periods of up to 30 days.[12,46] Caloric density, weight-per-volume considerations, prior experience, work level intensity, and local availability of food seem to affect reliance on relatively high-fat provisions.[3,28]

Protein and Maintenance of Lean Body Mass and Performance

Considerable discussion of the proper amount of protein to maintain muscle mass, prevent wasting, and sustain performance under conditions of physical stress exists in the literature. However, despite all the controversies, recommendations concerning the amount of protein in the diet have changed little since WWI, as evidenced by the 1919 report by Murlin and Miller[57]: "The amount of protein . . . sufficient to repair all of the wastes of the body and to supply an adequate reserve is 13% of the total energy intake. It seems a matter of indifference to the muscles whether they receive their energy from carbohydrate or from fat. . . . Hard muscular work therefore can be done on a diet high in carbohydrate or on a high fat diet. It is of general experience, however, that muscular work is done with less effort if there is a plentiful supply of carbohydrate."

Thirteen percent of the energy intake translates to an intake of 65 g of protein per day on an energy-restricted intake of 2000 kcal/day, or 130 g of protein per day on a more adequate 4000 kcal/day energy intake. This quantity of dietary protein is rather easy to obtain even for the most casual ration planner (e.g., one stick of beef jerky or one serving of peanut butter contains 6 to 8 g of protein). Although dietary protein is important, the quantity of carbohydrate, rather than the amount of protein, in a food-restricted diet is more closely related to maintenance of nitrogen balance and preservation of lean body mass.[22] This is believed to result from carbohydrate's amino-acid-sparing action (i.e., amino acids from dietary protein are spared from subsequent deamination, gluconeogenesis, and oxidation for energy). Approximately 40 g of dietary protein per day seems to be the minimum required to prevent excessive nitrogen loss under food restriction.[22] Although this amount is easily achieved even with a suboptimal intake of 1500 kcal/day of a mixed diet containing 15% protein, ration planners should strive to achieve 100 to 150 g protein per day (15% of the daily kilocalories) for most individuals consuming food in proportion to their energy expenditure.

Vitamins—Their Relationship to Health and Physical Performance

Vitamins are complex essential organic micronutrients that function in growth, maintenance, and metabolism. Vitamins act as coenzymes in metabolic reactions, and vitamins E and C and betacarotene (the precursor of vitamin A) act as protective antioxidants. Considerable oxidative stress may be experienced while working in environmental extremes.[7] High rates of energy expenditure, ultraviolet light exposure, and reduced dietary availability of foods containing antioxidants can cause excessive oxidative stress. Supplementing the diet with a vitamin and mineral mixture containing antioxidants to combat these stresses is perhaps more important than supplementation to prevent vitamin deficiencies (e.g., scurvy) that take a relatively long time to develop.

Prevention of vitamin deficiencies is among the least understood aspect of short- and long-range nutrition planning. Body stores of some vitamins (primarily the water-soluble vitamins) are limited, and under prolonged periods (i.e., weeks, not days) of dietary restriction, vitamin deficiencies can be manifested. This is rarely the case, however, for individuals consuming a mixed diet supplying close to the daily energy requirement. Although it is not routinely encountered, it is possible to develop a state of tissue depletion of thiamin, riboflavin, and pyridoxine in as short a time as 11 weeks by consuming a calorie-adequate, but vitamin-deficient, experimental diet composed of common food products.[81] Deficiencies of this nature do not develop over the short term; they are not caused by a few days of suboptimal vitamin intake. Van der Beek and colleagues[80–82] have studied the maintenance of human physical performance with varying degrees of vitamin restriction. Dietary vitamin deficiencies in subjects consuming restricted vitamin intakes significantly less than the RDAs were manifested slowly, particularly in terms of physical performance impairments. Diets containing restricted intakes of thiamin (28% RDA), riboflavin (31% RDA), pyridoxal phosphate (16% RDA), and ascorbate (10% RDA) resulted in less than a 20% decrement in cycle ergometer performance (maximal work load) after 8 weeks at this level of restriction (Fig. 63-4).

The relatively small change in performance decrements in response to vitamin restriction can be contrasted with the more immediate effects of acute or long-term dietary carbohydrate restriction on physical performance. The manifestation of physical performance impairment is much more sensitive to the amount of carbohydrate in the diet in the short term (1 to 3 days) than it is to the vitamin, protein, or fat content of the diet (6 to 8 weeks).[2]

It can be generalized that the development of a vitamin deficiency has four stages (Box 63-1), which involve a continuum of physiologic manifestations.

The possibility that certain nutrients (in particular, vitamins) might help people adapt to, or function more efficiently in, stressful environments has intrigued explorers and scientists for years. Perhaps the most thorough study of the interaction between nutrients and the environment was conducted in 1953 in a field study entitled the "Medical Nutrition Laboratory Army Winter Project: Vitamin Supplementation of Army Rations under Stress Conditions in a Cold Environment—The

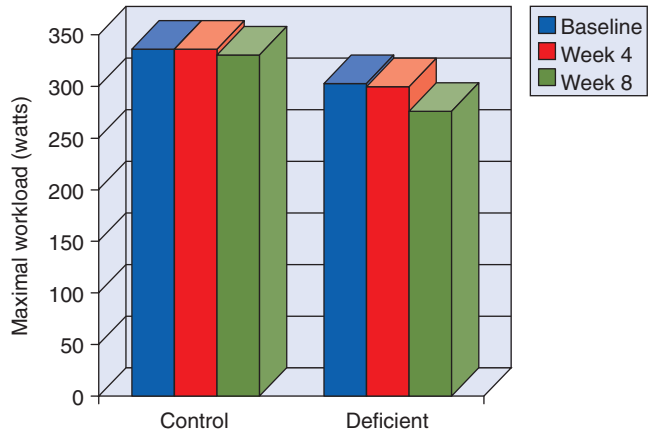


Figure 63-4. Impact of restricted vitamin intake on functional performance in humans. Experimental conditions: diet, 3070 kcal; thiamin, 28% RDA; riboflavin, 31% RDA; vitamin B_6, 16% RDA; vitamin C, 10% RDA. Performance test is workload achieved during incremental cycle ergometer testing. (Data from van der Beek EJ, van Dokkum W, Schrijver J, et al: Thiamin, riboflavin, and vitamins B-6 and C: Impact of combined restricted intake on functional performance in man. Am J Clin Nutr 48:1451–1462, 1988; and van der Beek EJ, van Dokkum W, Wedel M, et al: Thiamin, riboflavin and vitamin B-6: Impact of restricted intake on physical performance in man. J Am Coll Nutr 13:629–640, 1994.)

Box 63-1. Four Stages in the Development of a Vitamin Deficiency

1. PRELIMINARY STAGE
- Inadequate amount because of poor dietary patterns or altered availability in the diet
- Commonly encountered after short-duration (<30 days) wilderness activities with poor nutritional planning
  Consequence: None except danger of progressing to stage 2

2. BIOCHEMICAL DEFICIENCY STAGE
- The body's pool of the vitamin is decreased
- May be encountered after long-term (>30 days) wilderness activities accompanied by suboptimal daily nutrient intakes
  Consequence: Rates of enzyme catalyzed reactions may be slightly altered

3. PHYSIOLOGIC OR SUBCLINICAL DEFICIENCY STAGE
- Can be detected by functional tests
- May be encountered after extended periods of consuming foods low in vitamins or periods of food restriction
  Consequence: Performance may be impaired slightly

4. CLINICAL DEFICIENCY STAGE
- Specific symptoms manifested, physical signs clinically detectable
- May be encountered after starvation or extended periods of food deprivation
  Consequence: Possible impairment of both health and performance

Box 63-2. Conditions That Might Warrant Vitamin Supplementation

- When energy intake is below 1200 to 1600 kcal/day
- When meals are routinely missed
- When extremely poor or bizarre eating habits are practiced
- When oxidative stressors (ultraviolet light exposure, high rates of energy expenditure, lack of fruits and vegetables in the diet) are high
- When unusual physiologic conditions, such as pregnancy or lactation, are present

Pole Mountain Wyoming Study." The objective of this study was to determine if supplementation with large quantities of ascorbic acid and B-complex vitamins would influence the physical performance of soldiers engaged in high levels of physical activity in a cold environment, both with and without caloric restriction.[8] The investigators concluded that supplementing the mixed diet of men engaged in high levels of physical activity in the cold, with or without caloric restriction, did not result in significantly better physical performance.

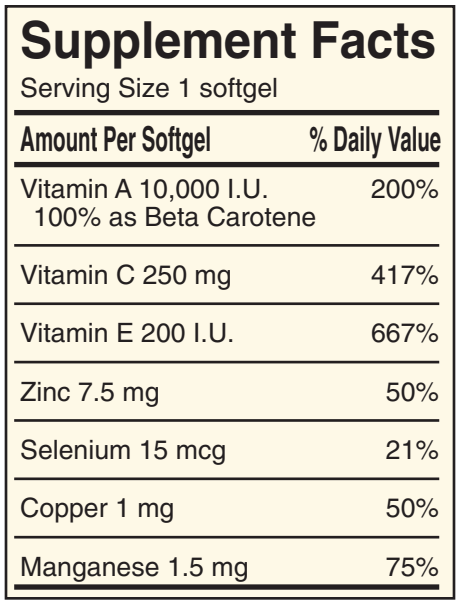
Thus, it appears reasonable to conclude (at least from a performance standpoint) that vitamin supplementation in the wilderness environment is not nearly as critical as total energy and carbohydrate provision. This does not mean, however, that dietary vitamin intakes should be ignored. Including a multivitamin and mineral supplement is a practical preventive measure. Conditions under which vitamin supplementation may be recommended are summarized in Box 63-2.

Certain vitamins, such as vitamins E and C, may have important functions beyond their conventional essential roles, such as preventing degradation of the immune response and maintaining red blood cell flexibility and oxygen delivery under conditions of increased oxidative stress. Because oxidative stressors may be encountered during wilderness activities, and because the quantity and quality of food is often uncertain, it seems prudent to include a multivitamin supplement with the food supplies. This nutritional insurance is inexpensive and takes up little space. The composition of a typical antioxidant supplement is shown in Figure 63-5.

Mineral Supplements—Electrolytes, Hematopoiesis, and Bone Health

The mineral content of the diet is usually not a primary concern, provided that a mixed diet that provides energy requirements is consumed daily. Calcium and iron are considerations for female travelers, as discussed previously. Sodium in hot environments is an exception, because it is lost during activities producing excessive sweating (see Chapter 64). With appropriate acclimatization to the heat (and resultant sodium conservation by the kidney), the amount of sodium required in the diet for work in the heat is reduced. Laboratory studies have shown that heat acclimatization can occur when as little as 4 to 8 g of salt (NaCl) is ingested per day.[1] The amount of NaCl required for safe work in the heat depends on the degree of prior heat acclimatization and the amount of sweat loss.

Altered dietary patterns or excessive sweating can influence sodium balance. Starvation can lead to plasma volume



Supplement Facts

Serving Size 1 softgel

Amount Per Softgel	% Daily Value
Vitamin A 10,000 I.U. 100% as Beta Carotene	200%
Vitamin C 250 mg	417%
Vitamin E 200 I.U.	667%
Zinc 7.5 mg	50%
Selenium 15 mcg	21%
Copper 1 mg	50%
Manganese 1.5 mg	75%

Figure 63-5. Antioxidant supplements can contain a variety of components designed to act as sacrificial antioxidants (e.g., vitamin C) or as cofactors in antioxidant enzyme systems (e.g., selenium). The supplement shown is typical of many basic antioxidant formulations.

depletion when sodium in the circulation is insufficient to allow osmotic forces to retain water.[36] When nonacclimatized humans are exposed simultaneously to acute food restriction and high sweat rates, the resulting excessive loss of sodium can lead to dizziness, syncope, and even collapse. These problems can be avoided by ensuring that extra salt is included in the food provisions for wilderness expeditions and that salt is available to vigorously season food during and after high sweat losses.

Another form of sodium deficit is dilutional hyponatremia caused by overhydration with fluids in conjunction with lower-than-normal sodium intake or excessive sodium loss in the sweat. Hyponatremia caused by overconsumption of fluids is extremely rare when regular meals are consumed. Hyponatremia as a complication of overhydration in wilderness environments where food is neglected has been described for hot[13,14] and cold[84] environments. Calorie deprivation can alter sodium intake. In addition to reduced intake of sodium via food, severe caloric restriction leads to marked natriuresis.[62] Increased loss of sodium in turn leads to depletion of fluid volume, impaired cardiovascular function, and reduced work capacity, in addition to energy restriction. Accompanying increased aldosterone levels in response to reduced plasma volume can also lead to accelerated potassium wasting.[62]

For insurance and safety considerations, include 5 g (about two restaurant packets) of table salt for every day in the field packed with the food supplies. If necessary for fluid replacement after excessive sweating, this extra salt can be used to make a dilute electrolyte beverage by adding $^1/_2$ tsp salt (2 to 3 g) to a quart of water.

NUTRITIONAL DEPRIVATION: MALNUTRITION AND STARVATION

Definitions

There is little uniformity in the terminology used to describe the physiology of human starvation. Hoffer[36] has suggested the following definitions:

Fasting: The total absence of food intake

Starvation: The physiologic condition that develops when macronutrient content is inadequate for a prolonged period

Semistarvation or food restriction: The more commonly encountered condition of some food, but of insufficient energy and protein provision

Malnutrition: A general term for the condition resulting from longstanding inadequate consumption of nutrients, abnormal absorption of nutrients, or unusual demands on certain nutrients; usually involves suboptimal food intake or the consumption of food of poor nutrient density, resulting in micronutrient and macronutrient deficiencies

Protein–energy malnutrition (PEM): The result of inadequate intake of energy, protein, or both for prolonged periods. Two related types of PEM are marasmus (primarily an energy deficiency) and kwashiorkor (primarily a protein deficiency). Both forms occur across a spectrum of situational and environmental conditions and exhibit many of the same symptoms. PEM is frequently a consequence of starvation, but all starvation does not necessarily lead to PEM. Development of PEM depends on body energy reserves, length of the fast, age, and presence or absence of disease. PEM is unlikely to be encountered by most wilderness travelers—a possible exception being within indigenous populations in certain third-world locations. When malnourished indigenous people are encountered in wilderness travels, caution should be exercised about sharing food. These individuals may not be used to ingesting large quantities of food replete with sodium, and there may be consequences to overeating (see Feeding Victims of Starvation, later).

Cachexia: This more specific term refers to the wasting that results from metabolic stress and loss of appetite. It is sometimes called cytokine-induced malnutrition to distinguish it from simple food deprivation in the absence of stress. The inanition of advanced-stage cancer patients is an example of cachexia.

Malnutrition

This discussion will be limited to the metabolic and physiologic consequences of starvation or energy restriction that might be encountered in wilderness settings, including unplanned emergencies that result in shortage of food, and wilderness rescue operations of victims of unintentional starvation. In these settings, the duration of food restriction is usually, but not always,[67] of shorter duration than that associated with the hunger encountered during famine, war, crop failure, and disease.

Solomons[72] has stated that malnutrition simply means "bad" nutrition and has listed six possible causes of nutrient deficiencies leading to malnutrition: reduced intake, decreased absorption, decreased utilization, increased destruction, increased wastage, and increased requirement. Primary malnutrition is caused by reduced intake of food. This is the most commonly

encountered cause of malnutrition in wilderness settings. Reduced intake, along with an increased requirement, can contribute to development of nutrient deficiencies during expeditions under extreme environmental conditions. Secondary causes of nutrient deficiencies are less commonly encountered but can contribute significantly if disease or illness strikes the wilderness traveler.

Malnutrition develops in stages that usually require a considerable length of time to manifest. As seen in cases of vitamin deficiency, the first changes reflect diminished dietary intake, with resultant reduced blood and tissue levels of nutrients, followed by intracellular changes in biochemical functions. Eventually, if the malnutrition is unrelieved, physical symptoms develop. These effects can include oral lesions, rashes, petechial hemorrhages, ecchymoses, pigmentation changes, and edema. People who undergo prolonged semistarvation experience hunger, weakness, lack of drive, mood changes, osteoporosis, hypoalbuminemia, edema, decreased muscle mass, alopecia, hypotension, and poor wound healing. Impaired immune function, decreased resistance to infection, and prolonged recovery from injury are all consequences of long-term food restriction.[24,30,45,69,70]

Starvation

Starvation is the physical condition brought about by inadequate consumption, absorption, or retention of protein and dietary energy from carbohydrate and fat.[37] Starvation can be acute or chronic as well as total or partial. Different forms of starvation are similar but not identical. Starvation can result from a disease or simply from a lack of food. It can ultimately manifest as a disease (PEM) or, if not prolonged, as nonpathologic weight reduction. The metabolic adaptations that occur in the progression of starvation depend on whether there is acute energy restriction or chronic undernourishment as a result of long-term low-energy intake.[68]

Acute Energy Restriction. The rapid progression of events that occur in response to acute energy inadequacy (e.g., a 50% shortfall of food during the second week of a 2-week backpacking trip) is shown in Figure 63-6. The body reacts quickly to an energy shortage by utilizing readily available muscle and liver glycogen stores. When the body's carbohydrate stores have been depleted, glucagon level rises, insulin level falls, and the process of gluconeogenesis accelerates, converting noncarbohydrate precursors such as lactate, glycerol, and amino acids to glucose to maintain the blood glucose level and prevent hypoglycemia (Fig. 63-7).

These principles are expressed in more detail in Figures 63-8 and 63-9. Data used to prepare this classic scheme of carbon flux during starvation is based largely on the work of Cahill,[21] who studied metabolic aspects of starvation in humans. Reduction in carbohydrate intake and resultant reduced glucose provision during food restriction is in large part compensated for by accelerated gluconeogenesis. The carbon skeletons of 100 g of protein from endogenous tissue sources can ultimately provide approximately 55 g of carbohydrate after metabolic transformations. The glycerol from 100 g of mobilized fat can yield 10 to 15 g of carbohydrate via gluconeogenesis.[19] As glycogen is depleted, catecholamine production rises simultaneously, facilitating fatty acid mobilization from adipose depots. Once mobilized, fatty acids are taken up by muscle in proportion to their concentration in the blood and oxidized for energy. The metabolic events of a short-term fast are shown in Figure 63-8. The major difference between short- and long-term fasts is in the shift of carbon source for energy production. As the length of the fast increases, the amount of glucose oxidized decreases and the amount of ketone bodies oxidized increases.

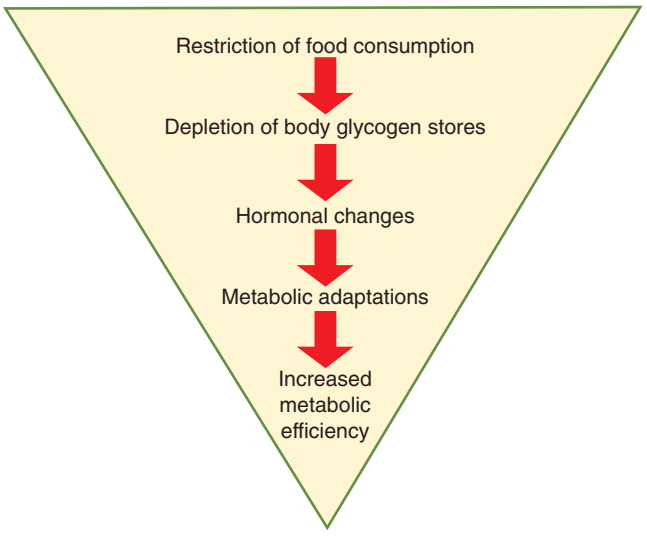


Figure 63-6. Sequence of metabolic events during short-term energy restriction or starvation. In the short term, metabolic efficiency increases to compensate for an energy deficit.

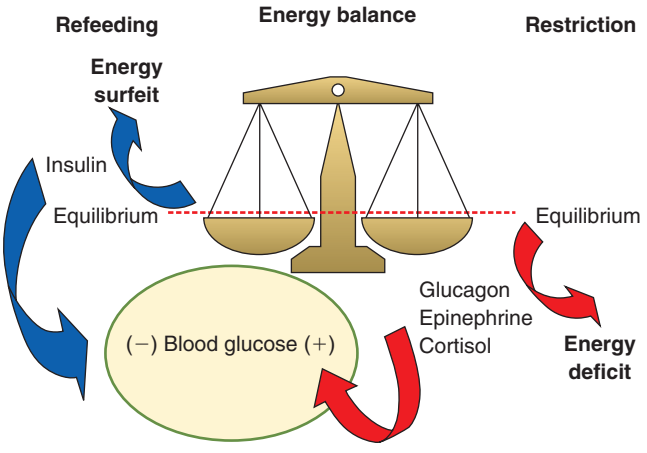


Figure 63-7. Influence of energy balance on hormonal control of blood glucose. When food is consumed after ingestion, insulin is released, lowering blood glucose. When no food is consumed and blood glucose drops, gluconeogenic hormones are released, and endogenous sources are used to raise blood glucose levels.

Long-term Energy Restriction. Prolonged fasting is characterized initially by increased protein catabolism for gluconeogenesis, followed by increased production of ketone bodies. A fast longer than 2 to 3 days exhausts liver glycogen and uses up about half the muscle glycogen stores.[36] Thereafter, glucose utilized by the body (in the absence of food consumption) must be synthesized from endogenous precursors via gluconeogenesis.

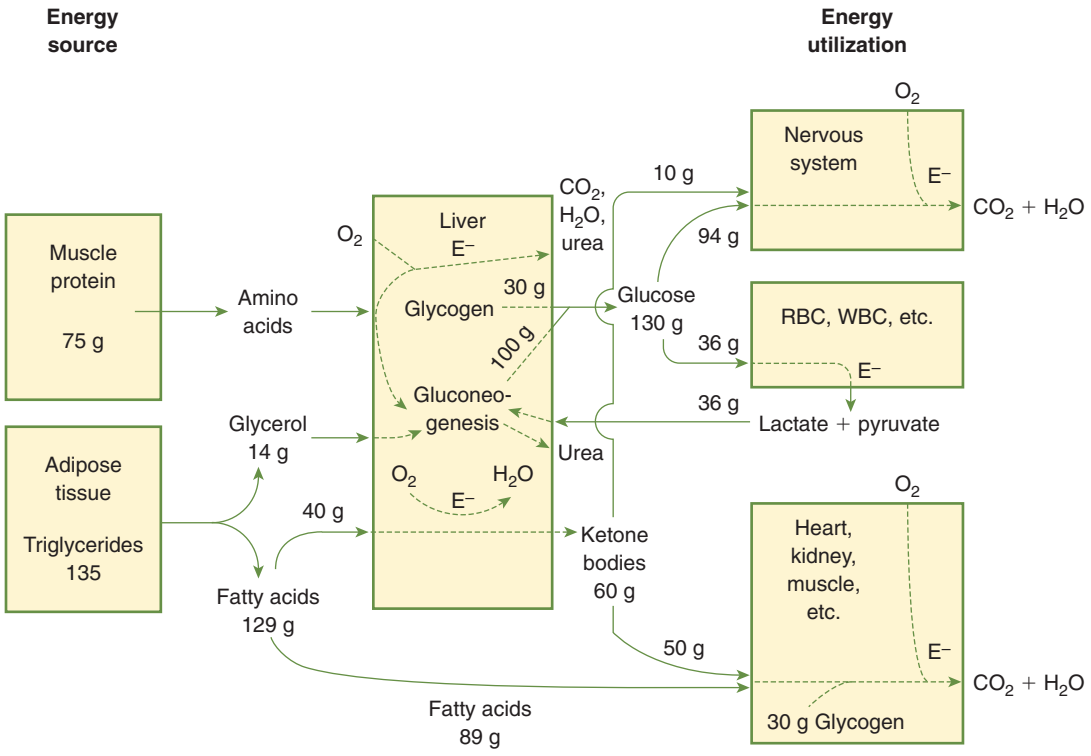


Figure 63-8. Fuel utilization in short-term starvation. Metabolic rates in g/day after a 24-hour fast. Energy expenditure, 1800 kcal/day (7.53 MJ/day); respiratory quotient, 0.76. (Original data from Cahill GF Jr: N Engl J Med 282:668–675, 1970. Algorithm redrawn from Bursztein S, Elwyn DH, Askanazi J, Kinney JM: Energy Metabolism, Indirect Calorimetry, and Nutrition. Baltimore, Williams and Wilkins, 1989.)

The appearance of large amounts of ketone bodies in the blood, breath, and urine is the result of the low insulin-to-glucagon ratio accompanying a prolonged fast, and of massive fatty acid mobilization from adipose tissue.

Ketone bodies are a metabolic consequence of vigorous fatty acid oxidation engendered by starvation in the absence of significant carbohydrate intake. Production of acetoacetate and β-hydroxybutyrate increases significantly during the first 7 to 10 days of fasting and stabilizes after 2 to 3 weeks.[18] Ketone bodies can be increasingly used for energy by muscle and brain as energy restriction becomes prolonged. Even a short-term fast elicits a significant production of ketone bodies (see Figure 63-8); however, ketone body production may be virtually abolished if a minimum of 150 g of carbohydrate is ingested daily to supply the brain with glucose for energy.[19,36] Normally, in the fed state, ketone body oxidation accounts for the provision of less than 3% of the total energy requirement. Longer periods (7 to 10 days) of fasting are accompanied by a large elevation of the level of circulating ketone bodies, which can provide as much as 40% of the total energy expenditure and greater than 50% of the brain's energy requirement.[19,36] The switch to using ketone bodies for energy in the brain is believed to be controlled by the concentration of ketone bodies in the blood rather than being a direct hormonal effect on the brain.[18] Metabolic changes during a long-term (e.g., a 30-day) fast are depicted in Figure 63-9.

With increasing duration of the fast and depletion of muscle and liver glycogen, ketones and glucose derived from gluconeogenesis from amino acid carbon skeletons contribute to energy requirements of the brain. As starvation is prolonged and ketone bodies become the predominant fuel, less and less glucose is used, thereby reducing the amount of protein that must be catabolized to support gluconeogenesis. Blood levels of branched-chain amino acids (leucine, isoleucine, valine) are preferred amino acid substrates for muscle energy metabolism and double by 3 to 5 days of fasting, but they fall during prolonged fasting. These branched-chain amino acids are believed to augment gluconeogenesis until fat metabolism has adapted to fasting.[18] The amino acid glutamine has special importance during fasting, assuming several roles—as an energy source as a precursor of glucose, and as a transporter of amino acid nitrogen in the form of ammonia (NH_3) from tissues to the kidney for excretion. Urea is the major nitrogen excretory product in the urine during the fed state, but it becomes much reduced as the fast progresses and ammonia nitrogen increases. The increase in ammonia nitrogen serves to buffer ketoacids during their excretion via the urine, as well as to provide an excretory route for nitrogen. Glutamine is released from muscle during fasting (NH_3 formed from amino acid deamination by muscle is transaminated to glutamate to form glutamine) and serves as both a special energy source for the gut and a gluconeogenic substrate for the kidney. With increasing ketone body produc-

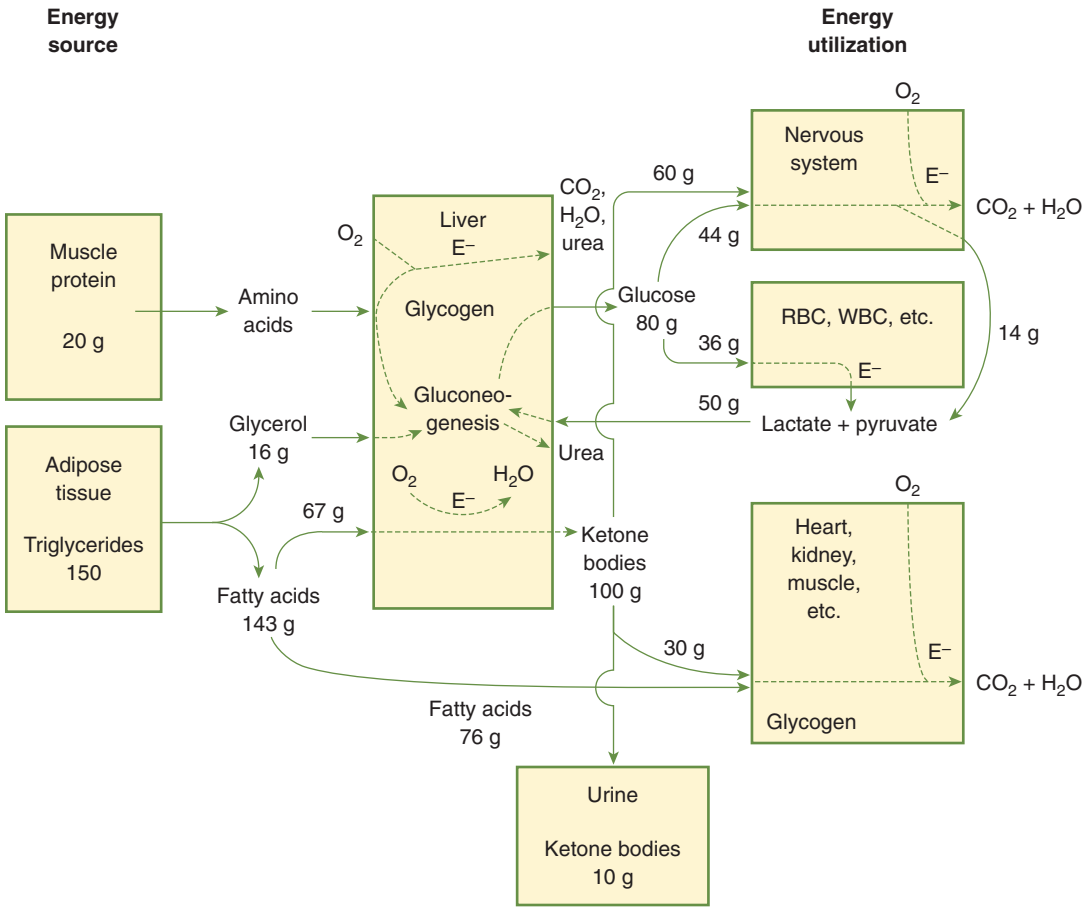


Figure 63-9. Fuel utilization in long-term starvation. Metabolic rates in g/day after a prolonged fast of 5 to 6 weeks. Energy expenditure, 1450 kcal/day (6.07 MJ/day); respiratory quotient, 0.74. (Original data from Cahill GF Jr: N Engl J Med 282:668–675, 1970. Algorithm redrawn from Bursztein S, Elwyn DH, Askanazi J, Kinney JM: Energy Metabolism, Indirect Calorimetry, and Nutrition. Baltimore, Williams and Wilkins, 1989.)

tion, the liver reduces its rate of gluconeogenesis and the kidney becomes the major organ of gluconeogenesis, producing over half the body's glucose requirement.[18] Glutamine is the predominant substrate for kidney gluconeogenesis and provides the NH_3 required to buffer ketoacid excretion produced by ketogenesis from fat oxidation.[18]

Fortunately, muscle proteolysis does not continue at the typically high initial rate of negative nitrogen balances (10 to 12 g/day during the first 7 to 10 days of fasting). After 7 to 10 days of fasting, adaptation in the nitrogen economy of the body reduces nitrogen loss in the urine to less than half the initial rate.[36] Although the signal that causes muscle to reduce its catabolic rate is not well understood, it is probably related to the shift to ketone body utilization in the brain and the shift to fatty acid oxidation in muscle during this same period. When adaptation to food restriction is not successful or food restriction is too severe, nitrogen can be lost from both central (visceral) and peripheral (skeletal muscle) sites. Development of central protein deficiency can lead to anergy and hypoalbuminemia. Reduced plasma albumin lowers plasma oncotic pressure, which permits fluid to migrate out of the vessels into the extracellular space, resulting in edema of the extremities. Edema is not present in all instances of starvation, but the presence of edema indicates severe metabolic stress and central protein deficiency and is a potentially dangerous condition.[36]

The net result of short- and long-term metabolic adaptations in energy restriction is increased efficiency of the body's metabolism. When bodyweight decreases 8% to 10% over a 14-day period, the basal metabolic rate (BMR) can decrease about 21%. This short-term decrease in BMR is greater than would be predicted if it were caused solely by the loss of metabolically active lean tissue mass. Reduction in BMR during energy restriction occurs in two different phases.[18] Initially, there is a decrease in BMR that is not attributable to changes in bodyweight or body composition. This is presumably an attempt by the body to conserve energy by increasing its metabolic efficiency despite reduced energy intake. Then, with continued energy restriction, further lowering of BMR occurs because of the loss of metabolically active tissue. Several physiologic mechanisms operate to downregulate metabolic activity in the active tissue mass and to increase its metabolic efficiency. The decreased energy flux reduces the activity of the sympathetic nervous system

and lowers secretion and activity of three thermogenic hormones: catecholamines, T_3 (triiodothyronine), and insulin. Other energy-requiring activities, such as the sodium–potassium pump and futile metabolic cycling (e.g., phosphorylating and dephosphorylating metabolic intermediates), may also be reduced during starvation, further conserving energy.

Hierarchy of Tissue Utilization during Starvation

The author offers the following whimsical "Internal 'Conversation' on Starvation" between the liver and the brain to illustrate the hierarchy of tissue utilization during starvation:

"The feast was grand," the liver said,
"exactly what I needed!"
"Glad you liked it," the brain replied,
"the muscles just seceded."
"Is that a problem for us, Boss?"
(The liver was trepidatious.)
"Not if we have you, my friend."
(The brain was quite loquacious.)
"But what about our chubby friend?"
volunteered the liver.
"Fat is always hanging 'round;
he wants to be a giver!"
"Oh, fat was most cooperative,"
(The brain was now ketotic.)
"We've sorely used our greasy friend,
and now we're quite neurotic!"

There is a hierarchy of fuel sources during starvation, as illustrated by this poem depicting a conversation between the brain and the liver. During an extended period of fasting, the mass of muscle and adipose tissue is more likely to be reduced than that of the viscera.[68] Important internal organs, such as the liver, show no evidence of dysfunction after 7 days of fasting,[66] whereas muscle cell mass decreases linearly with the severity and duration of a fast.[68] Adipose mass decreases along with muscle, but not as rapidly initially, because of the caloric density of the energy of the fat depot and the low amount of associated water. Hoffer[37] has estimated that during a typical 3-week fast that elicits a weight loss of 350 g/day, the shed tissues are composed of approximately 125 g of lean tissue and 200 g of adipose tissue. The body defends a certain amount of body fat as essential for nerve sheath insulation, brain neurolipids, cell membrane integrity, and hormone synthesis. The general hierarchy of energy-source utilization is shown in Figure 63-10.

The largest energy reserves are found in the largest organs of the body—muscle (about 28 kg) and adipose tissue (about 15 kg). Critical internal organs, such as the liver, brain, heart, and kidneys, have a collective mass of less than 9 kg and are not good fuel source candidates, because they begin to lose critical functions when they become energy sources during starvation (decompensation). The body reserves that can be drawn on for energy during fasting are shown in Table 63-1 for a sedentary 70-kg man.

Sequence of Events during Starvation

Sex hormone synthesis is depressed during extended periods of food restriction,[31,30] because reproduction is not a high priority during starvation. As the fast progresses, eventually even visceral organs begin to be used for energy as the body decompensates or "feeds on itself." During a prolonged fast, the body undergoes the series of events depicted in Figure 63-11.[18] The

TABLE 63-1. Utilizable Energy Stores in a Sedentary 70-kg Man

ENERGY SOURCE	MASS (kg)	ENERGY (kJ)
Fat	15	590
Protein (muscle)	6	100
Glycogen (muscle)	0.15	2.51
Glycogen (liver)	0.075	1.25
Plasma glucose	0.020	0.33
Plasma free fatty acids	0.0003	0.012
Plasma triglycerides	0.003	0.125

Data from Cahill GF Jr: N Engl J Med 282:668–675, 1970.

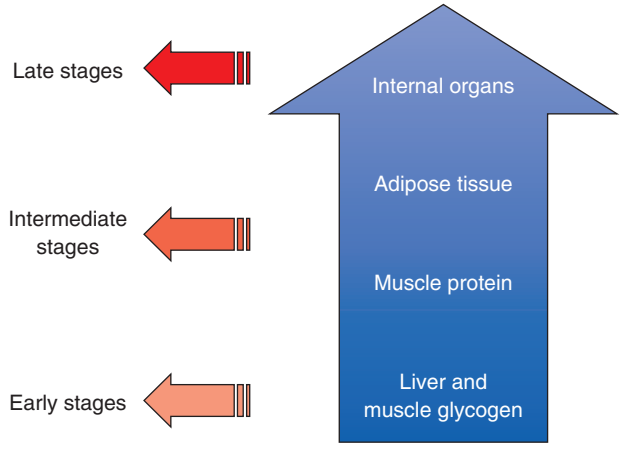


Figure 63-10. Energy sources during progressive stages of starvation. Note the hierarchy of the progression of utilization of energy sources.

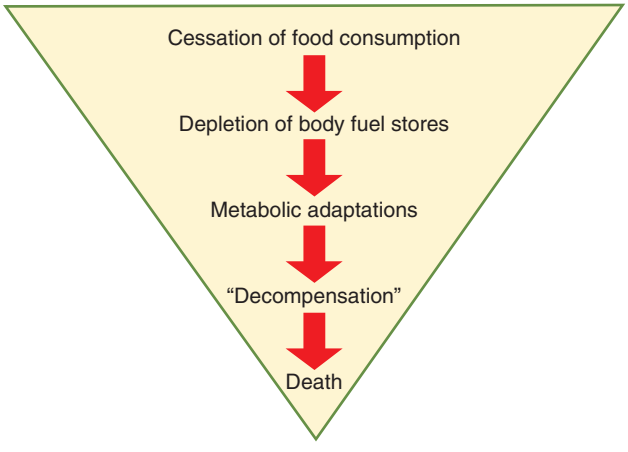


Figure 63-11. Sequence of events during prolonged starvation. If the fast is not terminated and energy reserves are depleted, internal organ systems ultimately used for energy will fail (decompensation), resulting in death.

initial steps are similar to those of short-term energy deficiency (see Figure 63-6), but they lead to a grimmer conclusion: the body has expended its reserves and enters the decomposition or final stages of metabolic self-destruction. Organ failure and death are the ultimate outcomes of starvation unless nutritional intervention occurs prior to decompensation (internal organ catabolism, loss of integration of function of bodily systems, and deterioration of homeostasis).

The Limits of Human Starvation, and Factors Influencing Survival

> "*There were horrific changes in my body. My buttocks were bones jutting against skin. I could no longer warm my hands by nestling them between my thighs. Now I could push a clenched fist between my legs without touching either thigh. I continually felt nauseated. The nausea became so severe I retched uncontrollably. This went on night after night. Death was closing in. My body was conceding defeat.*"
>
> James Scott's observations near the end of a 43-day period of starvation while lost in the Himalayas.[67]

Loss of Fat and Lean Body Mass

The body fat of humans can be considered beneficial when viewed in the context of surviving starvation.[60] A decreasing level of body fat approaching 6% loss is a harbinger of impending lean body mass deterioration in the fasting individual.[3,60] Essential fat stores (in bone marrow, heart, lungs, liver, kidneys, intestine, muscle, cell membranes, and the central nervous system) are necessary for maintenance of life and prevention of decompensation. Essential fat stores constitute 3% to 4% of bodyweight.[60] Total body fat depletion in previously non-obese individuals occurs at approximately 50% bodyweight loss. In fasting uncomplicated by disease, the time until death is largely determined by the size of fat stores and the time to reach the 3% level of essential fat. Fat stores protect function.[60] Low fat stores per se are not the cause of death, but their diminution contributes to breakdown of homeostasis and impairment of physiologic function. Typical fat stores in humans vary but are typically between 10 and 15 kg (including 2.1 kg of essential fat), or approximately 27% of bodyweight. In a well-nourished adult, there are sufficient fat energy stores to sustain life for 60 to 70 days. Death has been reported sooner in voluntary hunger strikers and may be related to simultaneous fluid restriction and the intangible lack of the will to live. A loss of greater than 50% of the lean body mass is also predictive of death. From a practical standpoint, the remaining body fat and protein reserves in a starving individual are difficult to measure accurately. A more practical measurement is the BMI—weight in kilograms divided by height in meters squared. BMI is easy to obtain and a convenient method to assess risk of mortality in severely starved individuals. Prior to the war and famine in Somalia, the lowest BMI compatible with life was thought to be 13 in males and 11 in females.[35] Collins[25] collected data at Baidoa, Somalia, in 1992 at the Concern Worldwide Adult Therapeutic Centre, where many victims of starvation were treated. He found that a BMI of 10 could be compatible with life under conditions of specialized hospital care, possibly explained by somatotype and the warm climate; however, other races in colder climates may face death prior to reaching the low BMI level noted in Somalia.[25]

TABLE 63-2. Demographics and Mortality Rates in the Donner Party, 1846–1847

	FREQUENCY *N* (%)	MORTALITY RATE *n/N* (%)
Total party	90 (100)	42/90 (47)
Age (yr)		
<5	19 (21)	11/19 (58)
6–14	21 (23)	2/21 (10)
15–34	34 (38)	16/34 (47)
>35	14 (16)	11/14 (79)
Sex		
Male	55 (61)	32/55 (58)
Female	35 (39)	10/35 (29)

Data from McCurdy SA: West J Med 160:338–342, 1994.

Age and Sex Differences in Survival from Starvation

Persons possessing the most limited body reserves, such as older adults and the very young, are at increased risk for early mortality during extended periods of starvation. Children have increased nutrient requirements for growth and develop deficiency symptoms rapidly when faced with severe food restriction.

Starving women have not been studied in the same controlled manner as have men. However, observational evidence of unintentional situational starvation involving both men and women seems to indicate that women may possess certain metabolic or cultural advantages over men, which may lead to a reduced incidence of mortality from severe starvation. Such evidence comes from wartime and famine, when there has been a disproportionate survival of women over men.[29,60] Although situational circumstances have been suggested to account for these differences, studies of the Mormon handcart trek[16] and the Donner party[75] support the conclusion that women are at lower risk for mortality under nutritionally stressful situations. McCurdy[54] examined the mortality pattern in the Donner party, which became trapped with inadequate food supplies in the Sierra Nevada Mountains during the winter of 1846–1847. The demographic mortality data derived by McCurdy (Table 63-2) indicates that very young children and males, in general, had the highest risk for mortality during starvation.

The influence of sex on starvation may be attributable, at least in part, to certain metabolic advantages possessed by women, such as a higher initial level of body fat and subsequent reduced loss of protein and lean body mass during fasting. Lowell and Goodman[47] proposed that protein sparing in skeletal muscle during prolonged starvation depends on availability of lipid fuels that may provide energy and attenuate the rise in catabolic hormones during starvation. They also suggest that fatty acids may specifically modulate the breakdown of myofibrillar protein, independent of their oxidation as a fuel, thus causing a direct muscle-sparing effect during prolonged starvation. Other factors may also contribute. Women generally have lower body mass and less lean body mass to maintain than men, as well as more subcutaneous fat, which may have insulation value during cold exposure. Sex-related cultural behavior, such as the performance of strenuous and high-risk tasks (e.g., the

TABLE 63-3. Composition of WHO Oral Rehydration Solution*

SOLUTION	GLUCOSE (g/L)	NaCl (g/L)	KCl (g/L)	TRISODIUM CITRATE† (g/L)	OSMOLALITY
World Health Organization (WHO) oral rehydration	20.0	3.5	1.5	2.9	310

*Preparation information available at http://rehydrate.org/ors/the_salts_of_life.htm#ORS%20Formula (commercially available from Jianas Brothers Packaging Co., 2533 SW Blvd, Kansas City, MO 64108).
†If trisodium citrate is not available, 2.5 g of common baking soda can be substituted.[13]

men usually pulled the handcarts during the Mormon pioneer trek west from Missouri to Utah), may also play an important role.

Feeding Victims of Starvation

With modern sophisticated means of communication, highly trained search-and-rescue teams, and air evacuation resources, rescue of a lost or injured person in a wilderness environment is usually relatively rapid. Whereas the rescue of the starving Donner party from the Sierra Mountains in 1847[54] took 4 months, the evacuation of recent polar explorers, such as the nutritionally depleted Mike Stroud and Ranulph Fiennes[76] from Antarctica in 1993, took 1 day. Although Stroud and Fiennes voluntarily prolonged their period of starvation before requesting evacuation, most individuals do not have to spend longer than a weekend or, at most, a few days longer than anticipated in uncomfortable circumstances. It is likely that some food will be available to the stranded person, as most people do not enter the wilderness totally unprepared. The individual may have consumed the initial food supply and then fasted for several days before rescue or, if disciplined, may have restricted or rationed food intake for several days. Helicopter evacuation can place the rescued individual in a hospital within hours of rescue. The worst-case scenario might be the rescue of a severely injured or ill person who could not or would not eat for an extended period and could not be evacuated by helicopter. Refeeding victims of short-term starvation is less complicated and dangerous than refeeding victims of prolonged starvation (e.g., James Scott, who was stranded in the Himalayas for 43 days without food[67]). Victims of prolonged starvation have dramatically altered blood mineral and protein levels, which can be rapidly perturbed by rapid refeeding large quantities of a normal mixed diet. The flood of nutrients with osmotic properties can lead to dangerous fluid compartment shifts. Cardiovascular and pulmonary overload caused by a rapid increase in plasma volume after fluid and nutrient ingestion can result in pulmonary edema.

The specific physiologic and metabolic effects induced by refeeding (the refeeding syndrome) depend on the individual's existing metabolic state and body composition, as well as on the composition of the refeeding diet.[36] Refeeding syndrome must be avoided in severely wasted individuals, particularly during the first week of nutritional repletion.[37] The rescuer need not be reluctant to offer normal food to most victims of short-term (3- to 5-day) starvation. The most commonly encountered problem may be that the individual wants to eat too much too soon. This problem is usually self-correcting. The individual may be very hungry, weak, and dehydrated. The victim should first be reassured and then checked for injuries, illness, and dehydration. Juices, soups, instant oatmeal, granola bars, and small pieces of jerky slowly chewed along with the fruit juice are all good choices to get the digestive system back to processing food while simultaneously supplying fluid, sodium, potassium, protein, and carbohydrate. Frequent small feedings are best. Sports drinks (if available) are a good choice for simultaneous rehydration, because they provide carbohydrates for energy and electrolytes that may be needed for plasma volume expansion.

The priority for re-alimentation of individuals lost in the wilderness for an extended time is to first correct fluid and electrolyte imbalance and arrest ongoing protein catabolism. It is not uncommon to encounter water-borne or food-borne illness in a backcountry scenario.[13] Rescued victims may be suffering from diarrheal disease as well as starvation. Oral rehydration solutions similar to those used to treat diarrheal disease can be used in wilderness rescue for extremely dehydrated individuals.[13] Intravenous saline and dextrose may be needed for those unable to eat because of shock, injury, or vomiting. The composition of the World Health Organization's oral rehydration solution is shown in Table 63-3.

It is important to ensure that the kidneys are capable of normal functioning. Fluids should continue to be offered until the victim is able to urinate every 2 to 3 hours, and then a more aggressive re-alimentation program can be followed. If the individual does not seem to be especially dehydrated, dilute fruit juices or sports drinks containing 5% to 10% carbohydrate and about 20 mEq (about 1.2 g) Na^+ per liter are reasonable rehydration fluids. Water is always appropriate, and additional sodium can be provided by liberally salting the solid food offered. A simple rehydration solution can be made by adding 1 teaspoon of table salt and 8 teaspoons of sugar to a liter of boiled water (http://rehydrate.org/solutions/homemade.htm). Sports drinks usually contain 10 to 25 mEq of Na^+ and 2 to 5 mEq K^+/L. Bouillon cubes are a good source of sodium and convenient to carry. One bouillon cube contains about 1000 mg of Na^+ (about 44 mEq). While providing sodium to a moderately depleted individual is beneficial and desirable for restoring plasma volume, this should be approached much more cautiously in victims of prolonged and severe malnutrition to avoid an overly rapid expansion of plasma volume that could lead to congestive heart failure.[36] Bananas are excellent sources of K^+ (450 mg per banana), but because they are perishable, they are seldom available to a rescue party. Dried banana chips are

Box 63-3. Refeeding Severely Malnourished Individuals

1. Normalize and maintain fluid and electrolyte parameters.
2. Provide a mixed diet at the maintenance level to establish tolerance and avoid the refeeding syndrome.
3. Slowly increase energy intake to achieve a positive energy balance and promote fat and protein gain.
4. Provide protein intakes of 1.5 to 2.0 g/kg to promote rapid repletion of body protein at any refeeding energy level.

From Hoffer LJ: In Shils ME, Ross AC, Shike M, et al (eds): Modern Nutrition in Health and Disease, 10th ed. Philadelphia, Lippincott Williams and Wilkins, 2006, pp 730–748.

a good alternative (152 mg K^+/oz), easily included in the rescue provisions.

After being corrected for hydration status and electrolyte balance, the victim should be placed on a high-protein, high-energy diet as soon as ingesting solid food is comfortable. Some diets are more appropriate than others for re-alimenting starvation victims.[37] For example, a diet high in sodium and carbohydrate fed to a severely malnourished individual may cause a rapid and large increase in extracellular volume, resulting in peripheral edema as well as fluid accumulation in the heart and lungs. A low-protein, high-energy diet may make sense initially, but, if continued, may bring about an increase in fat mass without the desired increase in lean tissue mass. A high-protein diet may arrest nitrogen loss, but it will not lead to simultaneous replenishment of fat stores. High-protein diets are also not appropriate (at least initially) for severely starved individuals encountered during relief work in famine-stricken areas. Collins[25] has reported that under the famine circumstances in Somalia, a protein-to-energy (P:E) ratio of 5 g protein per MJ of energy in the diet reduced refeeding complications compared to a P:E ratio of 9.5/MJ. Torun and Chew[79] have provided the following easy recipe for a high-protein, high-energy liquid food for initial refeeding of severely malnourished and starving individuals: powdered cow's milk, 140 g; sucrose, 100 g; cooking oil, 40 g; water, 900 mL. This mixture provides 3 to 4 g of protein and 0.6 MJ (135 to 145 kcal)/100 mL.

Victims of prolonged starvation may suffer from hyponatremia, hypocalcemia, hypomagnesemia, severe anemia, and impaired membrane and cardiovascular function. These victims should be re-alimented cautiously to avoid sudden expansion of intravascular fluid volume. Cardiac failure may develop if high-protein or high-energy feedings are instituted too vigorously too soon.[37] Increased sodium and albumin resulting from refeeding can cause a large expansion of the circulatory volume leading to pulmonary edema, congestive heart failure, and possibly secondary pulmonary infection. Hoffer[37] listed four steps to follow in refeeding severely malnourished persons (Box 63-3).

Further guidance regarding the homeostatic restoration of nutritional status in severely malnourished individuals (Box 63-4), along with appropriate diets, is discussed in detail by Torun and Chew.[79]

Box 63-4. Homeostatic Restoration of Nutritional Status in the Severely Malnourished Individual

1. Resolve life-threatening conditions first.
2. Begin restoration of nutrition as soon as possible.
3. Replace nutrient tissue deficits as rapidly and as safely as possible, *but*
4. Begin slowly, to avoid deleterious metabolic disruptions.
5. Schedule frequent feedings of small amounts (e.g., every 4 hours).
6. Diets that derive 60% to 75% of their energy from fat are usually well tolerated.
7. The protein source should be of high biologic value (i.e., high protein quality: milk, eggs, meat, fish, soy isolates).
8. Supplement the diet to provide K^+, Mg^{2+}, Zn^+, and Cu^{2+}. Fe^{2+} supplementation begins 1 week after the start of diet therapy. Sodium should be kept low until peripheral edema has disappeared.
9. Introduce a normal diet gradually when edema has disappeared, skin lesions have improved, and appetite is restored.

From Torun B, Chew F: In Shils ME, Olson JA, Shike M, Ross AC (eds): Modern Nutrition in Health and Disease, 9th ed. Baltimore, Williams and Wilkins, 1999, pp 963–988.

TABLE 63-4. Daily Calorie (kcal) Guidelines for Hikers*

BODYWEIGHT (lb/kg)	LIGHT HIKING	*BACKPACKING* LIGHT	*BACKPACKING* HEAVY
100–120/45–59	2000	2500	3500
130–150/59–68	2500	3000	4000
160–180/73–82	3000	3500	4500
190–200/86–91	3500	4000	5000

*Modify according to work level and prior experience.

NUTRITIONAL PLANNING FOR WILDERNESS ACTIVITIES

Practical Considerations

Nutritional planning is an important aspect of wilderness logistics, and one difficulty is that the amount of food carried is often constrained by the space it occupies and its weight. A daily food ration of 4000 kcal for men and 3500 kcal for women will adequately encompass most energy expenditure situations. If it is known that the task at hand will be hard physical work in the cold, 4000 to 6000 kcal/day may be needed. Generally an energy allowance of 45 to 55 kcal/kg bodyweight/day will cover energy needs for most moderate to moderately heavy levels of exertion (e.g., for a 70-kg individual anticipating moderate work levels, 50 kcal/kg/day × 70 kg bodyweight = 3500 kcal/day).[10] Some typical energy targets for food planning appropriate for temperate-weather backpacking activities are listed in Table 63-4.

TABLE 63-5. Nutritional Planning Guide for Field Rations

NUTRIENT	UNIT OF MEASURE*	DAILY RATION
Macronutrients		
Energy	kcal	3600
Protein	g	91
Carbohydrate	g	494
Fat	g	140
Vitamins		
Vitamin A	µg RE	1000
Vitamin D	µg	5
Vitamin E	mg	15
Vitamin K	µg	80
Vitamin C	mg	90
Thiamin (B_1)	mg	1.2
Riboflavin (B_2)	mg	1.3
Niacin	mg NE	16
Vitamin B_6	mg	1.3
Folic acid	µg DFE	400
Vitamin B_{12}	µg	2.4
Minerals		
Calcium	mg	1000
Phosphorus	mg	700
Magnesium	mg	420
Iron	mg	15
Zinc	mg	15
Sodium	mg	5000–7000
Potassium	mg	3200
Selenium	µg	55

*Units: 1 µg RE = 1 µg retinol (3.33 IU) or 6 µg (10 IU) betacarotene; 1 µg cholecalciferol = 40 IU vitamin D; 1 mg vit E = 1 mg RRR-α-tocopherol; 1 mg NE = 1 mg niacin or 60 mg dietary tryptophan; 1 µg DFE = 1 µg food folate or 0.5 µg synthetic folate.

Based on reference dietary intakes for active military personnel (AR 40-25 Nutrition Standards and Education, Headquarters, Department of the Army, Washington, DC, 2001).

These are only approximate guidelines, however, because it is impossible to establish energy requirements that cover all sexes, body sizes, work loads, and environments. More specific nutrient guidelines for food planning can be adapted from the U.S. military target nutrient content for operational rations, which can be viewed in its entirety at www.apd.army.mil/pdffiles/r40_25.pdf. Military field ration nutrient content has been established to meet the needs of a relatively young, active population and is applicable in most circumstances to nutritional support for many wilderness outdoor activities. The standards for daily ration content shown in Table 63-5 are based on current RDAs and can be used by wilderness planners to ensure a reasonably balanced and nutrient-adequate diet to support high levels of physical activity on any particular trek.

The macronutrient composition of the food is usually less important than the total energy content, except when heavy efforts are required over short (2- to 3-day) periods, when carbohydrate content may be the most important nutritional consideration. If a high level of performance (sustained or repeated high levels of power generation, such as working at greater than 60% VO_{2max} for prolonged periods) is anticipated, carbohydrate should be emphasized over fat. Approximately 50% to 60% of daily calories should come from carbohydrate. If weight of the provisions is an overriding consideration, maximal caloric density may be important and fat may be emphasized over carbohydrate, particularly in cold environments. Lower levels of power generation (working at a slow and steady sustained rate at less than 50% VO_{2max}) can usually be adequately supported by higher levels of dietary fat (about 50% of the daily calories). Individuals who intend to subsist on diets containing greater than 50% of the energy from fat usually require a metabolic adaptation period of approximately 2 weeks to adjust to the higher level of dietary fat.[62] Most trail rations consisting of a typical mixed macronutrient content (50% carbohydrate, 35% fat, 15% protein) will weigh approximately 1.4 to 2.7 kg (3 to 6 lb) (food for 1 day), depending on the degree of dehydration of the food. Thus, a 7-day backpacking trip may require 9.5 to 19 kg (21 to 42 lb) of food (excluding potable water). An acceptable level of protein content of the diet should be 12% to 15% of the calories, providing approximately 60 to 100 g protein per day. Higher levels of protein are feasible, but protein beyond the required amount may be an inefficient source of energy, requiring more water for urea excretion as the amino acids are deaminated, the nitrogen is excreted as urea, and the carbon skeletons are used for energy. The water burden of excess protein is not an overriding consideration if water is abundantly available, but this is an important factor in arid environments.

Palatable and nutritious backpacking foods are available at most outdoor stores specializing in camping gear. Some thought (and label reading) must be put into selecting and combining these individual food items. A simple approach is to purchase packaged military food such as the Meal, Ready-to-Eat (MRE), which contain a balanced macronutrient, vitamin, and mineral profile. A typical MRE meal packet supplies approximately 1250 kcal (13% protein, 36% fat, and 51% carbohydrate) and weighs about 0.6 kg (1.25 lb). MREs do not require any special handling or preservation and can be eaten cold or hot; their shelf life is 3 years at 80° F (27° C). Information about military rations such as the MRE can be found at: www.dscp.dla.mil/subs/rations/handbook.pdf.

Food Bars

Some outdoor minimalists choose to ignore nutritional planning and travel with their favorite easily consumed snack foods. This haphazard approach to nutrient requirements may work for a while, but it could dispose the individual to some potentially important nutrient deficiencies over time, or, at the very least, to palate fatigue. Commercial food (sports) bars are generally a better choice than candy bars and other junk food items, as they are usually fortified and less prone to melt in the pack.

Food bars are the modern equivalent of the early explorer's staple, pemmican (dried meat pulverized with fat and sometimes berries). They are not as energy dense as pemmican, but they are certainly more palatable and more nutritious. Food manufacturers have combined the camper's old standby of candy bars and trail mix into conveniently packaged and easily carried bars that usually have a good nutrient profile and may be fortified with selected vitamins, minerals, and protein. Most commercial food bars designed for sports nutrition or meal replacement are suitable for wilderness activities, although the low-carbohydrate bars may not be an appropriate choice. For unanticipated wilderness rescue operations, food bars are a good no-preparation, eat-on-the-move energy source for both the rescuer and the rescued. Some companies target outdoor activities in their marketing, but the composition of their products is similar to that of others, so their claims may not be meaningful. The main considerations in food bar selection should be stability in the pack or pocket, palatability, and the protein and carbohydrate content. If they are fortified with vitamins and minerals, they can supplement other foods in the diet. The advantages of food bars

TABLE 63-6. Food Bar Standards for Wilderness Activities

	AMOUNT	COMMENTS
Nutritional Content		
Carbohydrate	30 to 40 g/bar	To replenish glycogen stores
Fiber	3 to 5 g/bar	To prevent constipation
Protein	5 to 30 g/bar	High-quality protein such as casein or soy may be useful in emergencies when other protein sources are not available.
Fat	Variable	Usually not critical; higher-fat bars are more calorie dense.
Vitamins	~30% RDA	Not normally needed, but can provide insurance; antioxidant vitamins E and C most important
Minerals	~30% RDA	Ca^{2+}, Mg^{2+}, and Zn^{2+} are the most important, although women may benefit from supplemental Fe^{2+}, especially at altitude.
Other		
Stability	—	Low melting point in heat determined by trial and error
Palatability	—	Provide variety to avoid taste fatigue and increase food intake.

RDA, recommended daily allowance.

as nutrition supplements are that they consume little pack space, and they are at hand when energy levels slip, there is not time for a meal on the trail, or bad weather precludes food preparation.

Most bars contain significant amounts of carbohydrate, protein, and fat (Table 63-6). A bar containing 25 to 30 g of carbohydrate, a similar amount of protein, and 10 to 15 g of fat provides approximately 300 to 375 kcal of energy. Most are rather low in fat, because they are aimed at replenishing glucose and glycogen supplies during and after sports performance. The food bar market is very competitive, so some manufacturers include "unique" nutrients or herbal compounds to appeal to the buyer. The nutritional merit of some of these additions is questionable. Additions include vitamins, minerals, and phytochemicals (antioxidant nutrients); medium-chain triglycerides (as an alternate energy source); branched-chain amino acids (as fuel for muscle); soy protein (an anticancer agent); ginseng (to combat stress); and ginkgo biloba (to improve circulation) and choline (to improve concentration).

Selecting Food Bars

A typical energy or nutrition bar weighs about 60 g and contains 25 g carbohydrate (usually half from starch and half from sugar), 15 g protein, and about 5 g fat. Food bars are not dehydrated but usually have low water content, about 25% water by weight. Check labels for the calorie and nutrient content as these do vary from manufacturer to manufacturer.

Food bars claiming to be energy bars tend to have more carbohydrates, whereas "low-carb" and diet bars contain fewer carbohydrates. This reduced calorie claim is usually based on net carbohydrate content, such as fiber and sugar alcohols, which may not contain caloric value. Generally, these carbohydrates do not provide as much energy as do normal food carbohydrates, and they elicit a smaller insulin response. Sugar alcohols (i.e., polyols, such as maltitol and lactitol) that impart a sweet taste are technically carbohydrates, and they are common in low-carb bars because, compared with sugars such as glucose, they have fewer calories and less impact on blood sugar and insulin levels. These elements of low-carb dieting do not necessarily support high-level performance on a backpacking trip. Unlike other sugars, sugar alcohols are digested primarily in the large intestine by bacterial fermentation and, consequently, may promote gas or have a laxative effect, not a desirable attribute on the trail or in the tent! If the wrapper of a bar states that net carbs are only 2 g but the Nutrition Facts panel shows 20 g of carbohydrates, then there are 18 g of sugar alcohols, glycerin, or fiber in the bar. High-protein bars are designed to be higher in protein. Meal-replacement bars have a balance of carbohydrates, proteins, and fats similar to that of a typical mixed meal.

All of these bars may have the vitamins and minerals or other ingredients found in a dietary supplement. Nutrition bars can provide many nutrients needed on a daily basis, but they are not recommended as a total substitute for food except in certain situations, such as a rainy day on the trail or a late camp. They are best utilized as a quick-energy snack along the trail. If they are fortified with the vitamins and minerals found in a multivitamin pill, they can also be considered a multivitamin supplement. However, it is advisable to keep track of the amounts being ingesting from the bars in addition to the amounts from other foods and supplements to avoid exceeding recommended upper limits. Some products contain ingredients a consumer may not want in a food bar. A product that claims to be a dietary supplement, or that provides a Supplement Facts panel instead of a Nutrition Facts panel, may contain ingredients not normally expected in food. Supplementary ingredients are not necessarily undesirable, but the consumer should be aware of vitamins, minerals, herbs, or other special ingredients. For example, some products include caffeine in the form of coffee extract, guarana, or green tea. Even cocoa and chocolate contain some caffeine. Caffeine may be desirable in some circumstances, but it can enhance the action and increase the side effects of other stimulants, such as bitter orange *(Citrus aurantium)*. An independent evaluation of the nutritional properties of many current commercially available food bars can be found at www.consumerlab.com/results/nutbars.asp (subscription required for full report).

Emergency Food Supplies

Emergency food supplies of 250 to 350 kcal per person per day should be included in addition to the calculated caloric needs. These should be packed or carried separately from the main food items in case the main food supply is lost or destroyed. A stash of individually packaged food bars, jerky, dehydrated soup mixes, and hard candy provides an energy buffer that is reassuring even if it is not used. A bottle of multivitamins, extra salt packets, and water disinfection supplies are also advised. These small items may not seem important at the time of preparation, but that perception changes rapidly in an emergency. Finally, the most carefully planned food supply does little good unless it is eaten. Include foods that are palatable and comforting to the traveler. Do not overlook the morale-boosting aspects of a hot meal prepared on the trail.

The references for this chapter can be found on the accompanying DVD-ROM.

64 Dehydration, Rehydration, and Hyperhydration

Daniel S. Moran, Liran Mendel, and Stephen L. Gaffin

HYDRATION

Water is the most abundant constituent of the body and accounts for 50% to 70% of the body's weight. It is essential for supporting the cardiovascular and thermoregulatory systems and cellular homeostasis. Total body water (TBW) is distributed into intracellular fluid (ICF) and extracellular fluid (ECF) compartments, which contain 65% and 35% of TBW, respectively.[67] The ECF is further divided into interstitial and plasma spaces. The average 75-kg (165 lb) man has approximately 45 L of TBW, so the ICF contains approximately 30 L of water and the ECF contains approximately 15 L, with about 3.4 L in plasma and about 11.6 L in the interstitium.[67]

The terms used to describe body water status are *euhydration* (the idealized or "normal" body water content), *hypohydration* (body fluid deficit), *dehydration* (the dynamic process of body water loss, or the transition from euhydration to hypohydration), *rehydration* (replacement of body fluids), and *hyperhydration* (body fluid excess).

Traditionally, studies of physical performance in the hypohydrated state were performed on volunteers (college students or soldiers) who were rendered hypohydrated to various percentages of bodyweight while sitting at rest in a heated chamber or performing light to moderate exercise in the heat, and who abstained from drinking until their bodyweight fell. In a few studies, subjects were rendered hypohydrated by administration of loop diuretics (named for the kidney's loop of Henle). The extent of an individual's dehydration in the wilderness can rarely be known without adequate instruments and unless measurements were taken before beginning the activity. For closer applicability to wilderness conditions, an individual's sweat rate can be determined beforehand under controlled conditions.

Hydration Status Assessment

Because sweating involves loss of body mass, measuring changes in bodyweight (BW) is the simplest way to rapidly assess hydration status.[26] Daily BW tends to be stable, so changes can be correctly attributed to water loss. Theoretically, a fluctuation in body mass of ±1% (±700 mL) is to be expected in a euhydrated normal subject.[24] To accurately measure body mass and eliminate the potentially large underestimation of losses that results when sweat is retained in clothing, it is important that the subject be nude or seminude.[26] Measurement of BW in the early morning (after urination, before breakfast) as an index of euhydration assumes that 1 mL of sweat loss is equivalent to 1 g of lost mass. Measurement of BW is limited as a tool for long-term assessment of hydration status, because weight changes caused by changes in body composition (e.g., a change in percentage of fat) are also reflected. Thus, BW is most useful to detect acute changes in hydration status, although when used in combination with another hydration assessment technique, it can complement monitoring of long-term hydration status.

Measurement of TBW is the gold standard for assessment of hydration status. TBW is measured by dilution of infused or ingested isotopes (most commonly, ^2H_2O). A known quantity of the isotope is administered, it is allowed to mix with and be diluted by the body water, and then the new concentration of the isotope is determined in an aliquot of body fluid. The unknown volume (TBW) can then be calculated. The coefficient of variation (total error) for TBW by this method is approximately 1%.[4] For a 75-kg man with 45 L of TBW, a change of less than 0.65 L in TBW, or less than a 1% change in body mass, can be detected. (For water, 0.65 L weighs 650 g, which is less than 1% of the 75-kg man's body mass, or 750 g.)

Before assessing changes in fluid balance by determining TBW, an independent hydration assessment method must be used to establish that the subject is euhydrated at the outset and that the TBW is normal. TBW is often used in combination with body mass measurement to ascertain acute (e.g., hourly) fluctuation in fluid balance, but it is more valid for assessing chronic (e.g., in days) hydration changes in the field, when alterations in body composition are expected.[71] Nevertheless, although the choice of biomarker (e.g., TBW) for assessing hydration status should be sensitive and accurate enough to detect relatively small fluctuations in body water, its practicality (in terms of time, cost, and technical expertise) is also important.[23]

Plasma Markers. In euhydration, plasma osmolality is tightly controlled around a set point of approximately 285 mOsm/kg. This value increases by 5 mOsm/kg for every 1% to 2% loss of body mass caused by exercise-induced hypohydration.[126,141] Plasma osmolality is regulated primarily by two hormones, antidiuretic hormone (ADH) and aldosterone. Change in plasma osmolality is sensed in the hypothalamus by osmo-

receptors, and those neurons, in turn, stimulate production of ADH. When plasma osmolarity is below a certain threshold, the osmoreceptors are not activated and ADH secretion is suppressed. When osmolarity increases above that threshold, osmoreceptors recognize this cue and stimulate the neurons that secrete ADH.[23] ADH concentrations rise steeply and linearly with increasing plasma osmolality,[130] although they are sometimes confounded by exercise, hyperthermia, nausea, and fluid volume changes.[119] Aldosterone, the second hormone that regulates electrolyte balance, is secreted by the adrenal cortex. It acts directly on the kidneys to decrease the rate of sodium ion excretion, with accompanying retention of water, and to increase the rate of potassium ion excretion.

In exercise–induced hypohydration, plasma osmolality increases because the sweat lost is hypotonic relative to plasma.[30] Most laboratory osmometers have a precision of ±2 mOsm/kg, so they can measure the 5-mOsm/kg increase in plasma osmolality that occurs, as described previously, for every 1% to 2% loss of body mass.[126] Popowski and colleagues[126] demonstrated that plasma osmolality is sensitive to rehydration.

Measuring plasma sodium is an alternative to measuring osmolality, as most osmolality changes reflect changes in sodium concentration,[30] but because this relationship is variable,[141] plasma osmolality is still the simplest, most accurate and reliable plasma marker for tracking hydration changes over hours or days. However, if an iso-osmotic (rather than a hyperosmotic) dehydration occurs, such as with altitude or cold exposure,[120] then plasma osmolality changes will not follow TBW changes, and much larger volume reductions than actual calculated plasma volume (PV) will occur.

Advances in biosensor technology, including microminiaturization, have reduced commercial devices for measuring blood values to 2 × 6 × 1 inch (i-STAT Corporation, Princeton, NJ), with results as accurate as those obtained from full-sized automated blood chemistry analyzers. The i-STAT analyzer is portable enough to be useful in field scenarios for emergency diagnoses.[50]

Bioelectrical Impedance. Recently, bioelectrical impedance (BIA) has gained attention because it is simple to use and allows rapid, inexpensive, and noninvasive estimates of TBW.[121] In practice, a small, constant current is passed between electrodes spanning the body. The voltage drop between these electrodes provides a measure of bioimpedance. Prediction equations, previously generated by correlating impedance against an independent estimate of TBW, may then be used to convert a measured impedance value to a corresponding estimate of TBW.[157] Absolute BIA values are well correlated with TBW dilution techniques.[157]

BIA is not sufficiently accurate to assess dehydration (approximately 7% TBW), however, and it loses resolution with isotonic fluid loss.[121] In addition, because fluid and electrolyte concentrations can have independent effects on the BIA signal, it can provide grossly misleading values regarding hydration status.[121] Therefore, BIA has little application for the field assessment of hydration status.[23]

Urinary Markers. Urine specific gravity (USG) is the most widely used urine index. Measured against water as a standard (1.00 g/mL), USG represents the concentration of particles dissolved in urine and is a reflection of the kidneys' ability to concentrate or dilute urine in relationship to plasma.[23] Normal USG values range from 1.010 to 1.030.[7,126] It has been suggested that a USG of less than 1.020 indicates a state of hypohydration.[7] As a measure of chronic hydration status, USG appears to accurately reflect a hypohydrated state when in excess of 1.030.[7,126] However, considerable variability exists, and no single value can be used to determine a specific hydration level. Urine osmolality (U_{osm}) can also provide an approximation of hydration status,[149] as it is highly correlated with (but more variable than) USG.[7,126] However, these parameters are of limited use in evaluating acute episodes of hypohydration and rehydration, because urine flow rates are highly variable.[79,126]

The color of urine is a way to monitor hydration status. This method depends on individuals' paying attention to the color of their urine: darker urine indicates a higher level of hypohydration. Thus, a person with strongly yellow-colored urine is hypohydrated and needs to drink water. If urine is paler, the individual is adequately hydrated. This method, although enabling only gross evaluation of hydration status, excels in its practicality for determining moderate degrees of hypohydration and is more sensitive than methods based on blood measurements.[7]

Clinical Signs and Symptoms. Assessment of hypohydration via signs and symptoms is fast and easy; however, these estimates are too imprecise for accurate determination of hydration status.[14] Thirst, dizziness, headaches, and tachycardia, for example, are too common and nonspecific to be predictive. On the other hand, more severe symptoms, such as delirium or deafness, occur at hypohydration levels outside the functional range for training athletes.

Emerging Technologies. Recently, Moran and coworkers[105] presented a new method for noninvasive measurement of human state of hydration. This method is based on absorptiometry of radio waves passing through tissues at different frequencies. Absorption of radio waves through the wrist at various frequencies allows calculation of hydration state based on loss of TBW from sweating.

Recommendations and Summary

Table 64-1 is a comparison of methods for hydration assessment. Under most conditions of daily monitoring, ample sensitivity for detecting deviations from diurnal euhydration is obtained by early morning body mass measurements in combination with a measure of urine concentration (i.e., USG, osmolality, or color). Thus, bodyweight measurement, before and after endurance activity, corrected for fluid intake and urine output during the activity, is a simple, practical, and reliable method of measuring sweat rate and thus hydration status. Acute changes in hydration are resolved with better precision by determining plasma osmolality, by isotope dilution, and by measuring body mass changes. Plasma osmolality and isotope dilution, although less practical on a daily basis, can track hydration changes accurately over time. Plasma sodium assessment is easily confounded. In the future, technologic advances may allow for noninvasive assessment of body water as an index of human hydration status. Table 64-2 presents a summary of hydration assessment techniques.

TABLE 64-1. Usefulness of Various Hydration Assessment Techniques

ASSESSMENT TECHNIQUE	PRACTICALITY	VALIDITY FOR ACUTE (A) AND LONG-TERM (C) ASSESSMENT	ACCEPTABLE EUHYDRATION CUTOFF
Total body water (L)	Low	A and C	<2%
Osmotic pressure (mOsm/kg)	Medium	A and C	<290
Urine specific gravity (g/mL)	High	C	<1.020
Urine osmolality (mOsm/kg)	High	C	<700
Urine color*	High	C	<4
Body mass (kg)	High	A and C*	<1%

*Potentially confounded by changes in body composition.

TABLE 64-2. Comparison of Techniques for the Assessment of Hydration

TECHNIQUE	ADVANTAGES	DISADVANTAGES
Total body water, dilution	Valid, reliable	Premeasurement; invasive, complex, expensive
Plasma volume	—	Premeasurement; invasive, easily confounded
Plasma osmolality	Valid, reliable	Invasive, complex
Plasma sodium	Hyponatremia detection	Invasive, complex
Urine concentration	Easy, rapid, screen	Imprecise, confounded (for acute assessment)
Body mass	Easy, rapid	Confounded (for long-term assessment)
Bioelectrical impedance	Easy, rapid	Premeasurement; imprecise
Sign and symptoms	Easy, rapid	Imprecise

DEHYDRATION

Under neutral ambient temperature (T_{amb}), a person at rest dissipates heat by transitory increases in skin blood flow (BF_{sk}) to accelerate heat losses by radiation, conduction, and convection. When T_{amb} is greater than skin temperature (T_{sk}), exercise generates the need for additional mechanisms to dissipate heat. Evaporation of sweat is the only effective cooling mechanism, but this involves a loss of body fluid. Dripping sweat, especially prominent in a hot and wet climate, has no cooling effect. Under those conditions, the mean sweat rate (m_{sw}) will be higher but will not be able to adequately dissipate body heat and lower the core temperature (T_c).[38] Fluid losses through sweating may exceed 2 L/hr and may reach 15 L/day.[135] As a consequence, insufficient drinking for sweat loss can rapidly lead to dehydration, compounding the cardiovascular and metabolic stresses already present.[32]

Blood flow to exercising muscles declines in parallel with dehydration.[60] The combined stresses of dehydration and hyperthermia during exercise in the heat may exceed the body's ability to maintain cardiac output (CO) and blood pressure. This renders the dehydrated sportsman less able to cope with hyperthermia.[62]

Studies have shown that 1.9% to 6% dehydration decremented performance in various tasks by 6% to 57% in athletes (Table 64-3). Among elite athletes, the mean time difference between first and second place in international competitions was 0.32% (range, 0% to 0.83%) (Table 64-4). Thus, a small degree of hypohydration may lead to a large decrement in performance. A dehydrated top-rated athlete can be defeated by a euhydrated but inferior opponent, particularly in endurance events.

Physical and cognitive performance is decremented by 1% dehydration (600 to 800 mL body fluid loss), and the decrement becomes more severe with increasing fluid loss.[63,81] At 7% dehydration, there is evidence of exhaustion, and collapse is imminent.

Women were thought to be at higher risk for dehydration than men because they have less TBW, comparatively less lean body mass, and a higher proportion of adipose tissue than men of similar mass.[17] No sex differences were observed in m_{sw}, but among men and women with similar m_{sw}, women had a higher rectal temperature (T_{re}), suggesting that women have a less efficient process of evaporating sweat.[8] A higher ratio of surface area to mass should promote more effective thermoregulation, but nonetheless, if fluid replacement is not sufficient, women may be at higher risk for heat illness.

Recommendations. Individuals who are physically active in wilderness and other endurance activities should be familiar with their sweat rates and sweat volumes during the activity and should use this familiarity to guide fluid replacement and avoid dehydration. General guidelines for fluid replacement are sometimes useful but are often of limited value because of the large variability in sweat rate between individuals. For those spending time in a desert environment, it would be prudent to learn how to find water in the desert during emergencies (Box 64-1).[101]

Exertional Heat Illness

Hypohydration leads to a reduced ability to dissipate metabolic heat, which in turn leads to a rapid elevation in T_c and to exhaustion. Heat exhaustion occurs at a relatively low T_c

TABLE 64-3. Decrement in Performance by Dehydration

TASK	DEHYDRATION	PERFORMANCE DECREMENT	REFERENCE
Time to run 1.5, 5, and 10 km (.9, 3, 2.6 mi)	1.9%, 1.6%, 2.1%	3.4%, 7.19%, 6.74%	4a
Walk in exhaustion in heat	1.9%, 4.3%	22%, 48%	36a
Cycling, CV variables	2.5–3.2%	23.9%	27a
Treadmill during heat at 25–65% $A\dot{V}O_2max$	3%, 5%	0.3°C/3%; 0.7°C/5%; HR 2–18 bpm; CO 0.2–2.3 L/min	102a
Submaximal work	3.9%	6.7%	154a
Maximal exercise work time	3.8%, 3.6%, 4%	20%, 45%, 57%	130a
Submaximal work		T_c: 0.1°C	65
Submaximal work		T_c: 0.5°C	56
Maximal isometric strength, jumping height	3.4%	7.67–1.9%, 1.51%	157a
Isometric and isotonic exercise	3.95%	31%, 29%	156a
Maximal exercise work time	4%	12%	131a
Various strength, CV measures, wrestlers	4.9%	13.18%, 11.76%, 21.5%, 9.72%, 12.38%	157b
Treadmill, wrestlers	4.6%	9.32%, 15.6%	2a
Walking, running cross-country	5.7%	21% maximal oxygen uptake	22
Marksmanship	6%	20%	154a

TABLE 64-4. Percentage Difference between Winning and Second-Place Scores in Selected Championship Sports

EVENT	PLACE	ATHLETES	TIME (sec)	% DIFFERENCE
50 m freestyle, men's swimming, 1996 Olympics	1	Alexander Popov	22.13	0.59
	2	Gary Hall	22.26	
200 m freestyle, men's swimming, 1996 Olympics	1	Danyon Loader	107.6	0.46
	2	Gustavo Borges	108.1	
400 m freestyle, men's swimming, 1996 Olympics	1	Danyon Loader	228	0.44
	2	Paul Palmer	229	
200 m individual medley, men's swimming, 1996 Olympics	1	Attila Czene	119.9	0.17
	2	Jani Sievinen	120.1	
400 m individual medley, men's swimming, 1996 Olympics	1	Tom Dolan	254.9	0.12
	2	Eric Namesnik	255.2	
Rowing, 1996 Olympics	1	Marnie McBean, Kathleen Heddle	416.84	0.36
	2	Mianying Cao, Xiuyun Zhang	418.35	
Men's cycling, 1996 Olympics	1	Pascal Richard	17,636	0.00
	2	Rolf Sorensen	17,636	
Four-man bobsled, 1996 Altenberg World Cup	1	Czudaj, Voss, Szelig, Lehmann	113.70	0.001
	2	Langen, Kohlert, Ruhr, Hampel	113.83	
Men's marathon, 1997 World Championships	1	Abel Anton	7996	0.06
	2	Martin Fiz	8001	
Men's 5000 m, 1997 World Championships	1	Daniel Komen	787.4	0.24
	2	Khalid Boulami	789.3	
Men's discus, 1997 World Championships	1	Lars Riedel		0.23
	2	Virgilijus Alekna		
Men's pole vault, 1997 World Championships	1	Sergei Bubka		0.83
	2	Maxsim Tarasov		
Men's free rifle, 1996 Olympics	1	Ennio Falco		0.83
	2	Miroslav Rzepkowski		

Mean difference in performance between and losing: 0.32%

because blood volume displacement to the skin causes marked cardiovascular strain and instability.[98,138]

The impact of different levels of hypohydration on heat tolerance while wearing clothing was studied under various heat loads.[137,138] Under severe heat stress and wearing impermeable clothing, tolerance time was shortened when the subjects were hypohydrated before exercising, and the rate of rise in T_c during exercise increased with the degree of hypohydration.[93] The main contributor to excessive T_c rise is believed to be decreased sweat rate.[65] In warm environments, increased levels of hypohydration result in increased threshold temperature for the onset of sweating and decreased sweat rate at each work rate during exercise.[96]

Hydration status may affect thermoregulatory or cardiovascular responses to exercise via alterations of body fluid volumes or osmolality. Hypovolemia and hyperosmolality, individually or acting synergistically, impair heat loss by elevating threshold T_c for the onset of both peripheral vasodilation and sweating,

Box 64-1. Finding Water in the Desert

Persons traveling in hot climates should be prepared for strandings that might lead to death resulting from heat and dehydration. Some dehydration deaths could have been prevented if the victims were armed with proper knowledge. A well–known case of preventable dehydration is the death of large numbers of Egyptian soldiers in the Sinai, whose deaths could have been avoided had the soldiers known about subsurface "sweet" water. Another preventable death was the man in the desert who did not think to drink his car's cooling water and also did not lie in the shade below his car; instead he wandered off into the desert, where it took a long time to find his body. A woman alone for 2 days in the desert knew enough to stay in the shadow of a cliff. She benefited from the fact that maximal physical activity in the desert may cause a loss of 4 L of water per hour, whereas minimal activity in shade loses approximately 2.5 L per day.

Sources of water in the desert:

1. Look for green plants.
2. Near the seashore, drinkable water, usually originating from rain, may be found below a sandy/gravel surface and above any seawater because of its lower density. Particular plants may use this water and show greenery against a gray or brown substrate.
3. Water for drinking may be found at the foot of cliffs that become waterfalls only during a flood. Below these cliffs, the pounding water and gravel may have carved a depression in the rocky floor that becomes filled in with gravel saturated with water. At the top of the saturated gravel, evaporation may render the surface dry and hot, but much of the water often remains in the pools or below the soil surface. Green plants often inhabit these areas and are markers for water. Such natural reservoirs retain water for a long time.
4. In wadis, after heavy rain, depressions filled with gravel and rocks become filled with water, with plants surrounding them.

and possibly by decreasing the gain of these responses.[48,49,114] Hypohydration may also directly impair sweat gland function.[156] Hypohydration of 1.9% (by 24 hours of fluid restriction) increases T_c and serum osmolality during cycling exercise, and it decreases blood flow to nonexercising muscles (e.g., in the forearm) in a normothermic environment.[155] Increasing levels of hypohydration and plasma osmolality, and decreases in PV, cardiac filling, and stroke volume lead to progressively decremented CO and thermoregulation.[137] During exercise in the heat, each 1% increase in hypohydration level raised T_c 0.1° to 0.5°C (0.18° to 0.9°F) and increased heart rate (HR) by 4 beats per minute.[56,65] HR is elevated to compensate for decreased stroke volume, presumably caused by reduced central venous pressure and end-diastolic ventricular volume, both consequences of decreased blood volume.[61]

When evaporative cooling is required, and it is less than the maximal evaporative cooling capacity of the environment ($E_{req} < E_{max}$), then the heat stress is compensable; if, however, E_{req} is greater than E_{max}, the heat stress is uncompensable. At an uncompensable heat load, minor levels of hypohydration impair exercise tolerance, even at lower metabolic rates. The impairment becomes evident as the duration of the exercise increases at lower metabolic rates. Hypohydration decreases PV, increases plasma osmolality, and inhibits peripheral blood flow and the sweating response, resulting in increased rate of rise in T_c.

The effects of hypohydration on heat strain are well documented. The new physiologic strain index showed high correlation between strain and loss of body mass through sweating.[104,107,108] However, the effects on skeletal muscle performance and metabolism are less well understood.[96,137,138] Some studies indicated that hypohydration reduces muscle endurance,[18,45,106] whereas others showed no difference.[2,46,70] Montain and colleagues[101] reported that moderate hypohydration (4% of body mass) decreased skeletal muscle performance by 15%. They suggested that there had been inadequate control of prior exercise, heat exposure, and caloric intake in the previous studies.

Physiologic Consequences of Hypohydration

Hyperosmolality, even without a fall in blood volume, increases the threshold temperature for onset of skin vasodilation and sweating during exercise in the heat.[34] These effects may be neurally mediated, because preoptic anterior hypothalamic neurons are osmosensitive.[151] Moreover, hypovolemia reduces the rate of sweating during exercise in the heat.[47,137] Decreased PV as a result of lost water and salt, concomitant with increased distribution of blood volume to skin vascular beds, can reduce venous return, central venous pressure, cardiac filling pressure, stroke volume, and CO.[77,113] This would be followed by compensatory increases in HR.[99] In addition, increases in hematocrit and plasma viscosity further reduce cardiac filling pressure as a result of increased resistance. In one series of patients with exertional heatstroke,[145] 35% were hypotensive with a systolic blood pressure of less than 90 mm Hg. Similar findings have been reported for classic heatstroke.[64]

Sinus tachycardia in response to excessive circulatory requirements is consistently present in heatstroke victims.[143] CO and diastolic blood pressure are low, whereas pulse pressure is high.[146] Depending on the degree of hypoperfusion of vital organs, shock may ensue.[78] Hypohydration compromises thermoregulation,[146] with linear increases in T_c of about 0.15°C (0.27°F) for each 1% decrease in body mass during exercise in the heat.[137] Additionally, hypohydration negates most of the thermoregulatory advantages conferred by high aerobic fitness and heat acclimatization,[22] increasing the risk of heat illness.[146] Sawka and coworkers[138] reported that exhaustion from heat strain occurred at higher T_{re} for euhydrated than for hypohydrated subjects during uncompensable exercise heat stress.

Some athletes use diuretics to reduce bodyweight to compete in a lower weight category. Diuretics increase the rate of urine formation and generally result in loss of solute and volume.[158] Less intracellular water (about 65% of total body fluid) is lost after using diuretics because there is no excess of extracellular solute to stimulate redistribution of body water. Diuretic-induced hypohydration, unlike either exercise-induced or heat-induced hypohydration, generally results in iso-osmotic hypovolemia, because there is a higher ratio of plasma loss to total body water loss.[72] Common diuretics include thiazide, furosemide, and carbonic anhydrase inhibitors.

Dehydration predisposes to fatigue and collapse. Because hyperthermia and dehydration are synergistic in causing fatigue,

people who are dehydrated are less tolerant of hyperthermia and usually collapse or are fatigued at a core temperature of 38.5° to 39.5°C (101.3° to 103.1°F).[27,95,133] Furthermore, in addition to dehydration, muscle activity and sodium depletion also contribute to increasing core temperature. These factors put at risk individuals engaged in endurance exercise who seem to be "heavy-set and salty sweating."[42]

Treatment

In general, oral rehydration fluids are preferred for dehydrated patients who are conscious, coherent, and losing fluids through vomiting or diarrhea. Intravenous (IV) fluid administration is needed for those unable to ingest oral fluids, or if dehydration has caused the heat injury to progress to the point of heat stroke. The decision to utilize IV replacement of fluid in dehydrated patients also depends on the orthostatic pulse and blood pressure. Normal saline solution is the IV fluid of choice initially, as it provides excellent expansion of intravascular volume without risking too rapid a correction of undiagnosed hyponatremia. The rate of fluid administration depends on preexisting or coexisting medical conditions. An initial rate of 1200 mL administered over 4 hours has been proposed for adults.[123] Supplemental potassium should be withheld until serum electrolyte levels are known, because the victim may have previously ingested a commercial beverage containing additional K^+. Fluids given subsequently should reflect the individual's electrolyte status and cardiac and renal function.

Recommendations. Dehydration is common in hot environments during prolonged endurance activity because of the large loss of sweat volume and insufficient voluntary fluid replacement. To prevent net fluid loss, active individuals, especially in hot environments, should be aware of their sweat rates during physical activity (see "Hydration Status Assessment," earlier). Oral rehydration is preferred for the conscious dehydrated individual. However, if necessary, IV fluids may be used, most commonly normal saline or 5% dextrose in normal saline.

Dehydration (Hypohydration) and Cold Strain

Cold exposure elicits physiologic responses that minimize heat loss (vasoconstriction) and increase heat production (shivering). These thermoregulatory effector responses are modulated by the hypothalamus, and they are based on afferent input from both T_c and T_{sk},[19] weighted approximately 9:1, respectively. Despite the apparently low proportion of skin temperature input, its impact can be appreciable, because the range of possible skin temperatures is great (0° to 40°C [32° to 104°F]), whereas the range of core temperatures is small (36° to 43°C [96.8° to 109.4°F]).

During cold-weather outdoor activities, individuals may become hypohydrated by 3% to 8% of their bodyweight.[52] The reasons for this cold-induced dehydration include high sweat losses during physical activity when well insulated, blunted thirst, cold-induced diuresis, increased respiratory water losses from humidification of cold dry air during respiration, intentional reduction of drinking, and lack of availability of water.[52] Most of these factors cause greater losses of water than of solute, leading to a hypertonic hypohydration.[30] On the other hand, cold-induced diuresis causes proportional losses of water and solute, resulting in an iso-osmotic hypohydration.

O'Brien and colleagues[120] suggest that the presence of hypertonicity and hypovolemia alter whole-body cold thermoregulation by two different mechanisms. Hypertonicity appears to impair the vasoconstrictor response to cold. This is indicated by the presence of stable T_{sk} and core–skin temperature gradients throughout cold exposure in euhydrated subjects compared with those rendered hypohydrated and hypertonic. Furthermore, in this study, insulation (an index of core-to-skin heat transfer) was lower during cold exposure in the hypertonic hypohydrated state than in the isotonic hypohydrated state.

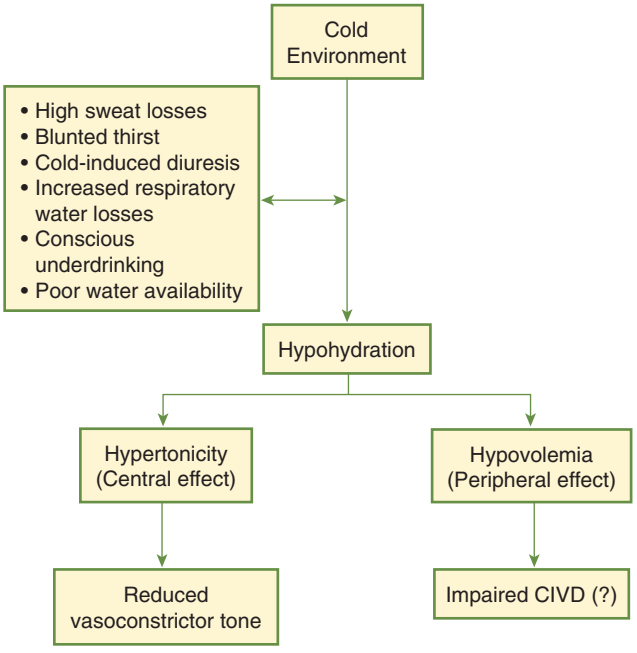


Figure 64-1. Proposed pathophysiologic effects of hypohydration in cold environment.

A hypertonic hypohydration state was suspect in impairing cold-induced vasodilation (CIVD), also known as the "hunting" response.[129] CIVD is a periodic increase in extremity blood flow that is thought to prevent or reduce excessive tissue cooling and frostbite.[87] However, a normal CIVD was not observed in the cold study of O'Brien and colleagues, which compared isotonic hypohydrated subjects with hypertonic hypohydrated subjects.[120] Nevertheless, there was some evidence in the hypertonic hypohydrated subjects of "blunted" CIVDs, which could be expected to increase susceptibility to peripheral cold injury.[85,129]

Hypertonicity appears to impair the vasoconstrictor response to cold. Concurrent hypohydration may have subtle effects on vasoconstriction that become even more important during a more severe cold exposure.[120] Figure 64-1 presents proposed pathophysiologic effects of hypohydration in cold environments.

Recommendations. In a cold environment, attention to the replacement of fluid losses is often neglected, resulting in dehydration and impaired thermoregulation. Athletes should drink adequately during endurance activity, even in the cold.

Dehydration Effects on Physical Performance

Dehydration impairs performance. Studies[25,92] that measured performance in different durations (especially longer than

90 minutes) of exercise and in different environmental conditions (cold, temperate, hot) found that the longer the duration of exercise and the heavier the heat load, the greater was the impairment in performance, even at dehydration levels as low as 2% of bodyweight.

Fluid Requirements

As described, a healthy 75-kg man has a TBW of approximately 45 L. The turnover rate of water exceeds that of most other body components, and body water content must be maintained within narrow limits. For a sedentary individual living in a temperate climate, daily water turnover is typically about 2 to 2.5 L, or 5% of the TBW pool.[90] The body is much less able to cope with restriction of water intake than with restriction of food intake. Cessation of water intake in a person who has a high sweat rate can result in serious debilitation after only an hour or two, although when sweat rate is low, this can in-crease to a few days.

Several factors influence the rate and extent of the body's water loss (Box 64-2) and thus determine the requirement for fluid intake.[90] Two important factors are ambient climatic conditions and level of physical activity. Among the interindividual differences are body composition and body volume (closely related to body mass), which define the amount of metabolically active tissue and the amount of body surface area available for both heat exchange with the environment and water loss. There is a large variability in habitual water intake and water loss between individuals, and the reasons for this have not been thoroughly studied. Along with water, essential electrolytes (particularly sodium, potassium, and magnesium) are lost. Water balance is closely associated with electrolyte (especially sodium) balance. All of these factors combine to determine the physiologic requirement for water. Superimposed on these physiologic control mechanisms are various social and other factors (e.g., voluntary dehydration, water temperature, flavor) that also affect fluid intake.

The Geigy Scientific Tables[3] suggest that the minimum of daily water intake for adults to sustain physiologic function is approximately 1.5 L, including water present in food and obtained from the oxidation of nutrients, or about 1 mL/kcal of energy expenditure. Major factors affecting basal fluid balance are presented in Table 64-5. Body size has a major influence on the turnover of water, but TBW content also depends markedly on body composition. For example, in lean tissue, water content is about 72% to 75%, but the body fat compartment contains very little water. This results in a difference between men and women, because women have a greater proportion of fat tissue. Unless otherwise specified, the values given here are for an average 75-kg man with a moderate body fat content.

Environmental conditions also affect basal water requirements. Water requirements for sedentary individuals living in the heat may be two or three times greater than those for individuals living in a temperate climate, even if there is no pronounced sweating.[1] Because the respiratory system humidifies inspired air, low relative humidity increases respiratory water losses from 200 mL/day in a warm moist environment to values as high as 1500 mL/day in a dry climate (although the average difference is usually around twofold).[82] Vapor pressure influences the amount of respiratory water loss much more than ambient temperature does. To these losses must be added insensible (or barely perceivable) loss through the skin (about 600 mL/day) and loss in urine (usually about 800 mL/day).

The water content of food ingested contributes to the total fluid intake. Furthermore, water is produced when nutrients are oxidized, the amounts varying from 0.4 mL/g for protein to 1.17 mL/g for alcohol. An energy intake of 3000 kcal/day, composed of 50% carbohydrate, 35% fat, and 15% protein, would result in about 400 mL of water produced per day by oxidation. This contribution to water requirements is rather insignificant when water losses are high, but it may be appreciable when they are low.

Another effect of the food ingested relates to the need for renal excretion of metabolic products and excess electrolytes. An intake of electrolytes in excess of the amount lost (primarily in sweat and feces) must be corrected by excretion of the electrolytes in urine, with a corresponding increase in the volume or osmolality (or both) of urine formed. The maximum concentration of electrolytes in urine varies between individuals and declines with age, but is typically about 1000 to 1200 mOsm/kg. Furthermore, this concentration depends on diet and varies between individuals (with differences seen between geographic regions, ages, and sexes), and there is a strong correlation with sweat rate and sweat volume.

A high-protein diet requires a greater urine output to allow excretion of water-soluble nitrogenous waste.[90] This effect is relatively small compared with other water losses, but it becomes meaningful when water availability is limited or when renal function is compromised. If daily protein intake is about 113 g (corresponding to 15% of the energy content of a 3000 kcal diet), and if all of this is oxidized, about 40 g of urea must be excreted. Excretion of this at a urine osmolality of 800 mOsm/kg would require urine output of about 830 mL.

Box 64-2. Factors Affecting Water Loss

- High ambient temperature
- High humidity
- Exercise intensity and duration
- Individual characteristics

TABLE 64-5. Mechanisms of Water Loss

PATHWAYS OF WATER LOSS	MECHANISM	RELEVANT ENVIRONMENTAL CONDITIONS	DAILY WATER LOSS
Respiratory	Inspired air humidified by body fluid	Dry environments (low relative humidity)	200 to 1500 mL
Skin (insensible)	Sweating	All conditions	600 mL
Urinary	Physiologic water, renal loss, electrolyte balance	All conditions	800 mL

Hydration Strategies

The American College of Sports Medicine (ACSM) guidelines of 1996 are the standard for hydration strategies.[29]

Hydration Prior to Exercise

It is recommended that 2 hours before exercise, 400 to 600 mL of water be ingested. This strategy should allow renal mechanisms sufficient time to regulate total body fluid volume and osmolality at optimal preexercise levels, and help delay or avoid the detrimental effects of dehydration during exercise. In addition, meals should be consumed regularly to return normal electrolyte losses. Normal electrolyte balance is essential to maintain euhydration and optimal physical and mental performance.

Fluid Replacement during Exercise

Because dehydration can impair thermoregulation and performance, rise in T_c and risk for thermal injury can be reduced by drinking fluid to offset sweat losses.[72] Some authors advocate replacing water losses due to sweating at a rate equal to the sweat rate.[15,36,116,125] Sweat rate is a function of the individual's metabolic rate, clothing, and climatic conditions. When a person did mild work while wearing cotton clothes, m_{sw} ranged between 0.3 and 1.2 L/hr. When the same person performed the same activity wearing nonpermeable clothing, m_{sw} ranged from 1 to 2 L/hr. During high exercise intensity in a hot climate, an athlete may have an m_{sw} of 1 to 2.5 L/hr.[134] A high m_{sw} of 3.7 L/hr was reported in a marathon runner in the 1984 Olympics.[6]

The subjective perception of thirst provides a poor index of body water needs.[132] As a result, individuals can dehydrate by 2% to 8% of their body mass during a prolonged high sweat rate, even though water may be readily available.[65] Because about 60% of body mass is water, dehydration of 2% to 8% is equivalent to a loss of 1 to 4 L of body fluids for a person with a body mass of 75 kg.[132] Increasing the palatability of the fluid is one way to induce a sweating athlete to drink. In general, fluid-replacement beverages that are sweetened (with carbohydrates or artificial sweeteners), flavored, and cooled (to between 15° and 21°C [59° and 69.8°F]) stimulate ingestion of more fluid.[20,44,73,131]

The rate at which fluid balance is restored is partly determined by the rate at which ingested fluid empties from the stomach into the intestine and then is absorbed into the blood. The most important factor influencing gastric emptying rate is the volume of fluid in the stomach—the more filled with fluid the stomach is, the faster it empties.[94,118,127] However, the rate of gastric emptying of fluid is slowed with increasing osmolarity of the beverage. For example, fluid with a glucose concentration greater than 8% leaves the stomach more slowly than a 5% glucose concentration or plain water.[33,74] Laboratory and field studies suggest that during prolonged exercise, frequent (every 15 to 20 min) consumption of moderate (150 mL) to large (350 mL) volumes of low-osmolarity fluid is possible and may improve the gastric emptying rate, provided that the individual is tolerant to these volumes.[29] Once fluid is in the intestines, the maximal rate of fluid absorption is generally adequate to cope with even extreme rates of gastric emptying.

Effects of Exercise on Water Balance

Only about 20% to 25% of the energy made available by metabolic pathways is used to perform external work, and the remainder is dissipated as heat. Exercise can increase heat production about 20-fold.[91] The extra heat generated by exercise must be removed from the body to maintain thermal homeostasis. A high skin temperature facilitates heat loss by radiation and convection, but these mechanisms are effective only when ambient temperature is relatively low and the rate of air movement over the skin is high.[86] At high ambient temperatures (>35°C [95°F]), the T_{sk} is lower than air temperature, so heat from the environment *adds* to the metabolic heat. In this situation, the only way heat can be dissipated from the body is by skin surface evaporation—that is, by sweating. Evaporation of 1 L of water from the skin removes 2.4 MJ (580 kcal) of heat from the body. Therefore, for a 70-kg marathoner running a $2^1/_2$-hour race, sweat would have to evaporate from the skin at a rate of no less than about 1.6 L/hr to balance metabolic heat with evaporative heat loss. At such high sweat rates, an appreciable fraction drips from the skin without evaporating, and a sweat secretion rate of about 2 L/hr could be necessary to achieve this rate of evaporative heat loss. This high sweat rate is possible in elite runners, but it would result in the loss of 5 L of body water over the course of the whole race, corresponding to a loss of more than 7% of bodyweight for this 70-kg runner. Water is also lost by evaporation from the respiratory tract, although in humans, this is not generally considered to be a major heat loss mechanism (Table 64-6).

In most activities, energy demand varies continuously. Average sweat losses during sporting activities can be substantial, even under temperate conditions, and not only when ambient temperature and humidity are high.[89] Large interindividual variability in sweating is normally observed. For example, in a group of professional soccer players training in a warm environment, rate of sweating varied from 1.1 to 2.2 L/hr, even though all completed the same training session.[148]

Prediction Models for Sweat Loss and Fluid Replacement

Sweating is a mechanism for losing heat during physical exercise and under heavy heat load conditions. The ability to predict

TABLE 64-6. Factors Affecting Fluid Balance during Exercise

MECHANISM	EFFECT ON FLUID BALANCE	RELEVANT ENVIRONMENTAL CONDITIONS	FLUID LOSS*
Conduction and convection	Small	Low ambient temperature (<35°C [95°F])	—
Sweating	Large	High ambient temperature (>35°C [95°F])	2 L/hr
Respiratory loss	Small	—	—

*For an average 70-kg man.

TABLE 64-7. Fluid Replacement Guidelines for Warm-Weather Training (Applies to Average Acclimated Soldier Wearing BDU in Hot Weather)

		EASY WORK		MODERATE WORK		HARD WORK	
HEAT CATEGORY	WBGT INDEX (°F)	WORK/REST CYCLE	WATER INTAKE (q/hr)	WORK/REST CYCLE	WATER INTAKE (q/hr)	WORK/REST CYCLE	WATER INTAKE (q/hr)
1	78–81.9 (26–28°C)	NL	1/2	NL	3/4	40/20 min	3/4
2 (green)	82–84.9 (28–29.4°C)	NL	1/2	50/10 min	3/4	30/30 min	1
3 (yellow)	85–87.9 (29.4–31°C)	NL	3/4	40/20 min	3/4	30/30 min	1
4 (red)	88–89.9 (31–32.2°C)	NL	3/4	30/30 min	3/4	20/40 min	1
5 (black)	>90 (32°C)	50/10 min	1	20/40 min	1	10/50 min	1

From Montain SJ, Latzka WA, Sawka MN: Mil Med 164:502, 1999.
BDU, battle dress uniform; NL, no limit to work time per hour.
The work/rest times and fluid replacement volumes will sustain performance and hydration for at least 4 hours of work in the specified heat category. Individual water needs will vary ± 1/4 quart per hour.
Rest means minimal physical activity (sitting or standing), accomplished in shade if possible.
CAUTION: Hourly fluid intake should not exceed 1 1/2 quarts.
Daily fluid intake should not exceed 12 quarts.
Wearing body armor: add 5°F to WBGT index. Wearing mission-oriented protective posture (MOPP, chemical protection) overgarment, add 10°F to WBGT index.
EASY WORK: Weapon maintenance; walking hard surface at 2.5 mph, ≤30-lb load; manual handling of arms; marksmanship training; drill and ceremony.
MODERATE WORK: Walking loose sand at 2.5 mph, no load; walking hard surface at 3.5 mph, ≤40-lb load; calisthenics; patrolling; individual movement techniques (e.g., low crawl, high crawl); defensive position construction; field assaults.
HARD WORK: Walking hard surface at 3.5 mph, ≥40-lb load; walking loose sand at 2.5 mph with load.
1 quart = 946 mL.

sweat rates under different environmental conditions and intensities of exercise would be useful, and a few models have been developed.[16,69] However, their applicability is limited because physiologic measurements under actual exposures are needed to evaluate the predicted sweat loss. Shapiro and colleagues[142] proposed a prediction model valid for a wide range of work, climate, and clothing ensembles. The model is based on easily obtained data and does not require invasive procedures. Sweat rate is calculated as follows:

$$m_{sw} = 18.7 \times E_{req} \times (E_{max})^{-0.455}$$

where E_{req} is required evaporative cooling (in W/m^2), and E_{max} is maximal evaporative cooling capacity.

First, required evaporative cooling (E_{req}) is calculated from metabolic heat production, clothing heat transfer characteristics, and the environment. Second, the maximal evaporative capacity (E_{max}), as dictated by vapor transfer properties of the clothing and the environment, is calculated. The predicted values in the preceding equation appear accurate compared with earlier models, but at very high sweat rates, they overestimate the rate of sweat loss.[68,142] One explanation might be that at very high sweat rates, sweating mechanisms are saturated[37] and the subjects could not reach the values required to achieve steady-state thermal equilibrium.[57,58] Furthermore, when other models were tested under field conditions,[103] actual sweat rates deviated appreciably from predictions.

Therefore, Moran and coworkers[103] proposed an adjusted prediction model, mainly for outdoor activities, which included two additional radiative heat exchange components: short-wave radiation (solar radiation) and long-wave emission from the body to the atmosphere. The model allows improved and more accurate sweat rate prediction for both indoor and, mainly, outdoor activities. In 1999, Montain and colleagues[97] published fluid replacement recommendations for military training in hot environments, which are based on the sweat rate prediction models (Table 64-7).

Recommendations. The sweat rate prediction model is a good and useful method to predict fluid replacement. However, the calculations are not trivial and should be carried out by an experienced environmental physiologist. The models are used as guidelines under various scenarios but do not account for individual variability. Therefore, each individual should validate the prediction model for himself or herself, at least once, and compare it to actual measurement of sweat loss.

Fluid-Replacement Beverages

The questions involved in choosing an appropriate fluid replacement during physical activity in the heat include whether there is a need for replacing lost electrolytes, and whether there is a need for carbohydrate supplementation. The benefits of fluid replacement with beverages containing electrolytes and carbohydrates, as opposed to plain water, were studied in athletes with 1.9% and 3.5% electrolyte losses.[160] No significant difference was found in the maintenance of PV between trials with water and the experimental formulations.

Early investigators promoted consumption of Na^+-containing fluid to prevent development of hyponatremia from excessive water-drinking during exercise. The rehydration effectiveness of

three commercial sports drinks (containing different minerals and glucose) was compared during 4 hours of physical activity.[75] All three solutions proved equally effective in maintaining water and electrolyte balance during moderate physical performance. A benefit of commercial sports drinks appears to be their enhanced palatability, which increases voluntary fluid consumption.[111]

The rate of rehydrating various body compartments depends on the rate of gastric emptying of ingested fluid. Gastric emptying depends principally on the osmolality and the caloric content of the fluid,[43] and these depend on the activity and on the temperature and volume of the beverage.[24a] Earlier studies suggested that there was a delay in gastric emptying and an increased rehydration time when the beverage contained more than 2.5% glucose, sucrose, or fructose. However, more recent studies have reported that there is little or no difference in gastric emptying rates between water and solutions with carbohydrate concentrations as high as 7%.[24a,39,140] Other investigators have confirmed that under exercise conditions, 5% glucose-polymer solutions show a gastric emptying rate similar to that of water.

A frequent argument for including Na^+ in fluid replacement beverages is to enhance intestinal water absorption, because Na^+ transport is the major determinant of water absorption in the proximal small bowel. Coupled active transport of Na^+ and glucose creates an osmotic gradient that pulls water from the lumen into the intestinal epithelial cells. However, the rate of intestinal absorption of carbohydrate-and-electrolyte solutions is equal to, but not faster than, the absorption of water.[111,159]

Cold fluids increase the motility of smooth muscles in the gastric wall, thereby speeding gastric emptying more rapidly than warm drinks. The commonly held belief that consumption of cold water results in stomach cramps has not been confirmed. Such a phenomenon, if it occurs, is more likely related to the volume of the beverage than to its temperature.[31]

Recommended Method of Drinking. Gastric emptying is speeded by stretching the gastric wall. Therefore, large volumes of liquid empty from the stomach more rapidly than small quantities. Athletes, however, are uncomfortable exercising with a nearly full stomach. Indeed, although there may be a benefit to rapid oral rehydration, if the nausea threshold is reached, a secondary increase in ADH secretion may impair the kidneys' ability to excrete excess fluid volume and result in hyponatremia and water intoxication.[5] By drinking smaller volumes (150 to 250 mL) at 15- to 20-minute intervals, individuals involved in endurance activities can maintain adequate hydration while minimizing gastric distention.

Glucose Composition of Sports Drinks

Coyle and Coggan suggested that a sports drink should contain 5% to 10% carbohydrate in the form of glucose or sucrose to enhance endurance.[35] However, many athletes suffer cramps, nausea, and diarrhea after drinking a 10% glucose solution. An isocaloric glucose-polymer solution has only one-fifth the osmolality of a 10% glucose solution, which allows an increased carbohydrate content without the gastrointestinal side effects of a high-osmolar drink. Several commercial polymer solutions are available.

Carbohydrate consumption during prolonged exercise enhances performance, whether assessed by time to exhaustion or by time to complete a predetermined exercise task.[28,111] Glucose-polymer solutions given before and during a soccer game sustained blood glucose concentration and improved performance, although no difference in perceived exertion was found.[144] However, dehydrated soldiers who ingested fluids during exercise in the heat reduced their strain and the intensity of their perceived exertion.[110] Whether use of these beverages spares muscle glycogen is a matter of debate.

A potential problem in using carbohydrate-rich beverages is that they may attract stinging Hymenoptera (e.g., bees) into the vicinity of the athletes, placing them at risk for sting-induced allergic or anaphylactic reactions.

Recommendations. Under normal conditions, beverages containing electrolytes and carbohydrates offer little advantage over water in maintaining PV or electrolyte concentration or in improving intestinal absorption. Consumption of an electrolyte-containing beverage may be indicated under conditions of caloric restriction, prolonged exercise, or repeated days of sustained sweat losses. Drinking carbohydrate solutions during prolonged exercise may enhance performance. For this purpose, use of glucose-polymer solutions may be considered. Carbohydrate-containing beverages should never be placed into canteens, because microbial contamination would be unavoidable and subsequent sterilization required.

For the vast majority of individuals, however, the primary advantage of using electrolyte- or carbohydrate-containing drinks appears to be enhanced voluntary consumption. This factor is important if regulated intake is impossible. However, during intense exercise lasting longer than 1 hour, the ACSM guidelines for exercise and fluid replacement recommend that carbohydrates be ingested at the rate of 30 to 60 g/hr to maintain oxidation of carbohydrates and delay fatigue.[29] In addition, the guidelines maintain that there is little physiologic basis for adding sodium to an oral rehydration solution, as long as sodium is sufficiently available in the stomach from the previous meal or in pancreatic secretions.[139]

Rehydration after Physical Activity

The 1996 ACSM position[29] on exercise and fluid replacement had little to say on the issue of rehydration strategies *after* physical activity, and none of the seven recommendations directly relate to fluid replacement *after* exercise. Two of the general recommendations for strategies after exercise are: "1. It is recommended that individuals consume a nutritionally balanced diet and drink adequate fluids during the 24-h period before an event, especially during the period that includes the meal prior to exercise, to promote proper hydration before exercise or competition. 2. It is recommended that individuals drink about 500 mL of fluid about 2 h before exercise to promote adequate hydration and allow time for excretion of excess ingested water."[29]

However, sometimes specific postexercise recommendations are warranted.[148] Complete rehydration after exercise can be achieved only if a fluid volume is consumed that is greater than the volume lost in sweat, because there is an obligatory loss of urine to eliminate metabolic waste products during physical activity that persists even in the dehydrated state. If effective rehydration is to be achieved, the volume of fluid consumed after exercise-induced or thermal sweating must therefore be greater than the volume of sweat lost.[150]

Optimal performance is attainable only with sufficient drinking during exercise to minimize dehydration. Even low levels of

dehydration (1% loss of body mass) impair cardiovascular and thermoregulatory responses and reduce capacity for exercise.[112] Fluid replacement reduces the risk of heat illness and improves exercise performance by preventing or reducing dehydration. Consuming fluids in amounts directly proportional to sweat loss maintains optimal physiologic functions and significantly improves exercise performance, even with exercise lasting 1 hour. However, it is recommended that ingested fluid be cooler than T_{amb} (15° to 25° C [59° to 72° F]) and flavored to enhance palatability and promote fluid replacement.[29]

The thirst mechanism cannot be relied on to prevent dehydration. Simply providing fluids at the sideline during athletic practices and competitions and military training scenarios is not sufficient. Scheduled breaks in activity (every 20 to 30 minutes, depending on the degree of heat stress) for required consumption of fluids should be routine.

Rehydration with and without Acclimatization

Sweating and normal thermoregulation have another impact on the predisposition to clinical states that is widely unappreciated by trainers, coaches, and team physicians. In unacclimatized individuals, sweat salt losses can theoretically produce solute deficits that have a profound impact on thirst and therefore on potential rehydration rates.

On average, 60% of body mass is water, so a 70-kg adult contains approximately 42 L of TBW. Hypothetically, a 70-kg individual who loses 6% of body mass by sweating *pure water* would lose a volume of 4.2 L (0.06 × 70 kg), or 10% of TBW, and TBW would now be 37.8 L. In this example, all salts were retained within the body, so plasma Na^+ rose in proportion to the ratio of the euhydrated volume to the new dehydrated volume (i.e., [42 L ÷ 37.8 L] × 140 mEq/L). Thus, Na^+ rose to 155 mEq/L (assuming two osmotically active particles per NaCl molecule) and 310 mOsm/L.

If, however, that same sweat volume contained NaCl at a concentration of half the normal euhydrated value (0.5 × 140 mEq/L = 70 mEq/L), then there was a sweat loss of 294 mEq Na^+ (70 mEq/L × 4.2 L), as well as the 4.2 L of water. Because the normal euhydrated plasma Na^+ concentration is 140 mEq/L, the total amount of Na^+ present in the body is 5880 mEq (42 L × 140 mEq). The final Na^+ concentration is the original amount of Na^+ (5880 mEq) reduced by the 294 mEq lost in sweat, or 5586 mEq. Finally, 5586 mEq of Na^+ divided by the TBW volume of 37.8 L (see preceding paragraph) becomes 148 mEq/L, or 296 mOsm/L.

This osmolality is high enough to reach the thirst threshold (295 mOsm/L), so the person feels moderately thirsty and is motivated to consume only 80 mL of water (see later discussion) before osmolality is reduced to the thirst threshold. However, this represents only 1.9% (80 mL/4200 mL × 100) of the water deficit. Consumption of a solute-rich meal or a fluid-replacement beverage about 2 hours before exercise potentially counteracts the salt loss in sweat of a nonacclimatized individual. This probably also explains why water balance is often not fully restored until mealtime in sweating, nonacclimatized individuals. Another clinically and physiologically relevant aspect of this calculation is that fully acclimated subjects sweat sooner (at lower T_c and T_{sk}) and with lower sweat salt concentration and in larger volume than nonacclimatized subjects. Thus, acclimated persons sweat more and require larger volumes of replacement fluids than nonacclimatized ones performing the same activity.

If this sweat were to contain a Na^+ concentration as low as 0.17% NaCl (29 mM), the solute loss in 4.2 L of sweat would be reduced to 244 mOsmoles (4.2 L × 29 mM × 2) compared with the previously calculated 294 mOsmoles. The dehydrated plasma osmolality would be 305 mOsm/kg, and the thirsty subject would have to consume 1270 mL (a 16-fold increase over the 80 mL seen in the earlier discussion) to lower the osmolality to the thirst threshold (30.2% of the water deficit versus 1.9% seen earlier). Although this theoretically important effect of acclimation on thirst and rehydration volumes has not been experimentally verified, it could significantly improve resistance to heat illness by maintaining more adequate TBW stores.

Recommendations. Complete restoration of fluid balance after exercise is an important part of the recovery process, and it becomes even more important in hot, humid conditions.[147] Rehydration after exercise requires not only replacement of fluid volume losses but also replacement of electrolytes, primarily sodium, lost in sweat. Drinks intended specifically for rehydration should therefore probably have higher electrolyte content than drinks formulated for consumption during exercise.

To restore hydration status, a person should consume 1 L (4 cups) of fluid for every kilogram of weight lost during activity. Fluid loss should be replaced before returning to activity, and if the loss is greater than 2% of body mass, activity should not be resumed. Prehydration (drinking immediately prior to exercise) slows development of dehydration and temporarily enhances performance.

HYPERHYDRATION

It had been hypothesized that consuming excess water before activity would improve performance during heat stress by expanding blood volume and reducing blood osmolality, thereby reducing cardiovascular and thermal strain during exercise.[51,136] Earlier studies[66,88,109,115] suggested that hyperhydration lowered HR and T_c compared with euhydration. Hyperhydration is usually achieved by drinking water in excess, but it can also be achieved by ingesting glycerol, which reduces free water clearance through the kidneys and thereby increases retention of fluid.[51] In one study, ingestion of glycerol plus water improved thermoregulation during exercise heat stress.[88] Rise in T_{re} was attenuated by 0.7° C (1.26° F) and sweat rate was elevated by approximately 300 to 400 mL/hr above control levels.

More recently, Latzka and coworkers[83,84] studied hyperhydration induced by drinking either water or a water-plus-glycerol mixture during compensable and uncompensable exercise heat stress. Surprisingly, compared with euhydration, hyperhydration did not alter T_c, T_{sk}, whole body sweating rate, local sweating rate, sweating threshold temperature, sweating sensitivity, or HR. Furthermore, there were no differences in physiologic variables between those achieving hyperhydration with water or by water plus glycerol. These data demonstrate that hyperhydration provides no thermoregulatory or cardiovascular advantages over maintenance of euhydration during compensable or uncompensable exercise heat stress.

Both exercise and heat stress reduce renal blood flow and free-water clearance. Therefore, the effectiveness of glycerol relative to water as a hyperhydrating agent may be masked. Latzka and colleagues[83] concluded that hyperhydration per se only provides an advantage (over euhydration) in delaying the onset of

hypohydration during either compensable or uncompensable exercise heat stress. Furthermore, glycerol ingestion caused nausea and headaches on several occasions. Why these results conflicted with previous studies is uncertain, but it may be because of differences in experimental design. For example, Latzka and coworkers[83,84] carefully controlled for the initial baseline hydration status for both treatments, whereas earlier studies did not address this important independent variable.

Recommendations. It is recommended that endurance athletes drink 500 mL of fluid about 2 hours before the activity to promote adequate hydration and allow time for excretion of excess water.

EXERTIONAL HYPONATREMIA

Although *hyponatremia* is defined as a serum sodium level below 135 mEq/L, it is also a term used clinically to refer to a syndrome occurring when there is rapid lowering of blood sodium concentration, usually to levels below 130 mEq/L. This may occur during prolonged exercise. Symptomatic exertional hyponatremia arises after prolonged work (typically longer than 6 hours), where sweating is the primary means of dissipating heat. The condition occurs primarily when individuals consume low-sodium or sodium-free water in excess of sweat loss ("overdrinking") during and/or shortly after completing exercise.[102] Electrolyte losses in sweat also contribute to development of the syndrome, particularly if sweat sodium losses are high. The consequent low osmolality of the extracellular fluid (ECF) promotes movement of water from the ECF into cells. If that fluid shift is of sufficient magnitude and occurs rapidly, it can congest the lungs, cause muscle weakness, swell the brain, alter central nervous system function, and eventually lead to cardiorespiratory arrest. Signs and symptoms of hyponatremia include confusion, disorientation, mental obtundation, headache, nausea, vomiting, aphasia, incoordination, and muscle weakness (Fig. 64-2). It can also cause a paradoxical symptom of thirst that cannot be quenched by ingesting more water. Complications of severe and rapidly evolving hyponatremia include seizures, coma, pulmonary edema, and cardiorespiratory arrest. The condition is generally treatable without long-term sequelae, but deaths have occurred.

This condition is becoming more common.[54,122] Symptomatic hyponatremia occurrence is especially common in distance (>42 km [26.1 mi]) running and triathlon events.[153] In the U.S. Army, 125 cases of documented hypo-osmolality or hyponatremia were reported over a 7-year epidemiologic study.[41] In addition, low serum sodium levels (<130 mEq/L) after exercise can occur even without symptoms.[153] These observations suggest that individual susceptibility may play a role in the etiology, but more importantly they document that a fair number of people performing prolonged work develop low serum sodium levels.[117]

The main contributing factor is overdrinking relative to sweat loss. In addition, loss of solute contributes to symptomatic hyponatremia by decreasing the safety margin before excessive water intake sufficiently lowers ECF sodium levels.[100,153] Therefore, individuals who secrete a relatively salty sweat, such as unacclimatized individuals, are at greater risk for developing hyponatremia, as less overdrinking is necessary to produce dilution of the ECF.

Impaired renal function was excluded as a possible explanation for hyponatremia.[153] A more tenable explanation is that the rate of urine excretion is limited by changes in control factors that determine water and sodium reabsorption.[163] Exercise can decrease urine flow rate 20% to 60%. This effect can persist even in an overhydrated person who drinks more during exercise.

Treatment and Differential Diagnosis

Water intoxication must be differentiated from heat exhaustion or heatstroke because the treatments are very different. In the field, this can be difficult because symptoms overlap.[55] However, in heat illnesses, T_c is generally greater than 39°C (102.2°F), whereas in hyponatremia, T_c is usually normal or close to normal.[9,55] Another possible difference is that in heat illness, symptoms of dehydration, such as the prolonged elevation of forearm skin after it is pinched, may be present.

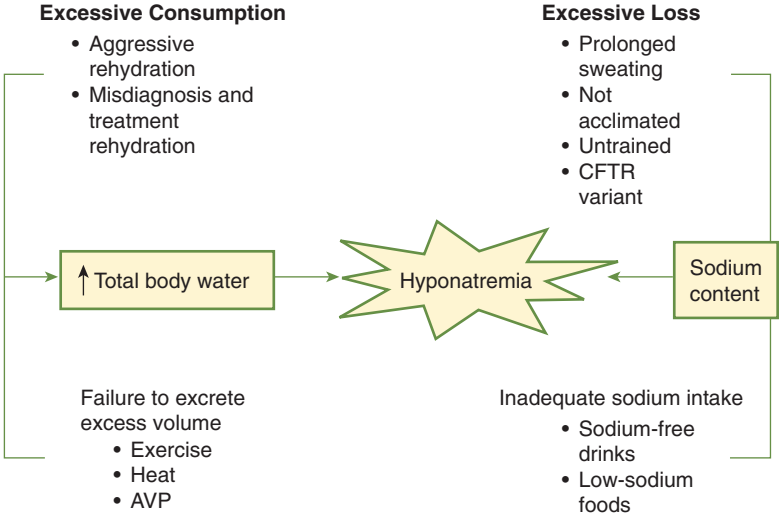


Figure 64-2. Exertional hyponatremia.

Heat illnesses are treated by cooling and sufficient rehydration, whereas hyponatremia is treated by restricting fluids and gradually correcting Na^+ concentration by infusion of hypertonic NaCl solution. Too-rapid correction of hyponatremia can cause pontine myelinolysis, a demyelinating syndrome.

Prevention

In the recently revised U.S. Army fluid-replacement guidelines, Montain and colleagues[97] emphasized both the need for sufficient fluid replacement during heat stress and concern for the dangers of overhydration and water intoxication (see Table 64-7). To achieve an accurate guideline for fluid replacement during heat stress, a two-phase study was carried out by Shapiro and coworkers,[142] who developed a computer model to predict sweat loss (m_{sw}) during different exercise intensities, followed by validation in a laboratory study. The fluid-replacement guidelines in Table 64-7 were designed to be a simple and practical tool. These recommendations specify an upper limit for hourly and daily water intake, which safeguards against overdrinking and water intoxication. However, it is recommended that individuals performing endurance activities validate their sweat rates at least once with respect to the guidelines, which do not account for individual variability.

Summary

Hyponatremia associated with prolonged exercise arises primarily from fluid overload from overdrinking. Insufficient replacement of sodium losses also contributes to sodium dilution. Lowering of plasma solute concentrations lowers osmolality of the ECF and causes movement of water into hypertonic cells. If the cells swell sufficiently, symptoms of central nervous system dysfunction occur. Those whose occupations require prolonged work in the heat, and competitive and recreational athletes, should be taught that fluid intake should not exceed sweat losses and that persistent excessive fluid intake can be harmful. During prolonged exercise lasting longer than 3 to 4 hours, salt-containing snacks or fluids should be ingested to offset sweat loss. This is especially prudent for individuals who are aware that they lose excessive salt in their sweat.

In addition, hyponatremia is suspected to be a principal cause of some exertional heat illnesses (see Exertional Heat Illness, earlier).

HYDRATION REQUIREMENTS FOR SPECIAL POPULATIONS

Hydration requirements are specific to the activity and the individual.[21] A variety of factors influence an individual's fluid intake during physical activity. Rather than taking a generic approach to fluid requirements based on the particular activity, sport, or event, each person needs tools to monitor the sweat losses experienced in a particular situation and should periodically take measurements to address these needs (see "Hydration Status Assessment," earlier).

Several groups are particularly prone to dehydration: infants and children, older adults, and special patient groups such as those with cystic fibrosis (CF).[11]

Infants and Children

The daily water needs per unit of body mass in infants are greater than in older children,[59] mainly because of the larger surface-to-mass ratio in infants. During growth and maturation, mean water turnover per kilogram of body mass decreases from 140 mL/kg/24 hr at ages 1 to 3 months, to 85 mL/kg/24 hr at ages 10 to 12 years, and to 35 mL/kg/24 hr at ages 13 to 15 years.[53] Their higher daily water needs, combined with their inability to express thirst, may explain why infants are particularly sensitive to water deprivation.

In addition, the increase in T_{co} at any given level of hypohydration is greater in 10- to 12-year-old boys than in non-obese young men, although the two groups were similar in their rates of involuntary dehydration (measured as percentage bodyweight).[13] This may reflect the lower ability of children to dissipate body heat when air temperature is similar to, or higher than, skin temperature.[10]

The best way to prevent or ameliorate involuntary dehydration in active children who are exercising and performing endurance activities is probably to provide, ad libitum, flavored drinks with the addition of 18 mmol/L NaCl and 6% carbohydrate content.[128,161] This recommendation holds for sedentary[161] as well as for exercising[128] children, and for repeated exposures to heat regardless of acclimation status.[162]

Older Adults

Because *old age* is variably defined, a definitive age threshold should not be set beyond which middle-aged people become older adults.[11] During normal daily life, without acute challenges such as exercise in the heat or water deprivation, older adults ordinarily consume enough water (in drinks and food) to prevent dehydration.[40,152] However, when their body fluid homeostasis is challenged by a decrease in extracellular fluid volume or by an increase in osmolality, older adults do not respond with an adequate thirst and do not drink sufficient fluids.[154] The pathophysiologic mechanism is more likely to be their diminished sensitivity to volume depletion than an increased osmolality of their body fluids.[76]

Cystic Fibrosis

CF patients lose exceptionally large amounts of NaCl in their sweat—so much so that their plasma osmolality is lower than that of healthy subjects.[124] Data suggest that this lower ECF tonicity deprives CF patients of a major stimulus for thirst,[12,80] therefore placing them at risk for dehydration. Dehydration in CF patients may be prevented by administering a relatively high concentration of NaCl (50 mM) in beverages, high enough to initiate the thirst reflex.[80]

Recommendations. Special populations at higher risk for dehydration should monitor their sweat rate during physical activity, especially during endurance and wilderness activity. The self-recognition of fluid loss provides people with the tool to calculate their needs and achieve fluid replacement.

DISCLAIMER

The opinions or assertions contained herein are the private views of the authors and should not be construed as official or reflecting the views of the Israel Defense Forces. Citations of commercial organizations and trade names in this report do not constitute an official U.S. Department of the Army endorsement or approval of the products or services of these organizations.

The references for this chapter can be found on the accompanying DVD-ROM.

65 Living off the Land

Peter Kummerfeldt and Denise Martinez

The ability to be completely self-sufficient in the wilderness and live off the land for an indefinite length of time is a fantasy of many. This fantasy can turn into an intentional or unintentional reality for some. The success of this reality is in large part determined by a person's capacity to procure, prepare, and store food and water, as well as have a basic understanding of human nutritional needs.

Meeting a person's nutritional needs is significant in the context of wilderness survival and living off the land. Human survival has always been held hostage to its own nutritional needs, and the success of the human species is directly related to a continued emphasis on achieving a balanced diet.[3,34]

ACHIEVING A BALANCED DIET

The Dietary Reference Intake for energy for moderately active men aged 19 to 50 years is 2900 kilocalories per day. For moderately active women of the same age group, it is 2200 kilocalories per day[17] (Table 65-1). Energy needs increase with greater physical activity and decrease in ambient temperature. Current dietary recommendations set forth by the U.S. Food and Drug Administration call for a daily intake of energy-yielding macronutrients to be composed of 60% carbohydrates, 30% fat, and 10% protein.[22] The U.S. Department of Agriculture also suggests that food choices be based on variety and balance, with an emphasis on plant foods, primarily grains.[38] However, these recommendations may not have a realistic application to wilderness survival and living off the land. To get a better idea of the probable composition of daily energy-yielding nutrients under these conditions, it may be best to look at the macronutrient intakes of hunter-gatherers.

Anthropologic analyses of hunter-gatherer diets show that 45% to 65% of their macronutrients were derived from animal food, both hunted and fished. The remaining 35% to 55% was made up of wild plants. It is also estimated that the average macronutrient composition of hunter-gatherers was 38% to 49% fat, 20% to 31% protein, and 31% carbohydrate.[6] For hunter-gatherer societies living at more than 40 degrees latitude north or south, there was an increasing latitudinal dependence on fished animal foods and decreased dependence on plant foods. Although hunter-gatherer diets were composed primarily of animal foods with a high intake of fat, field studies of 20th-century hunter-gatherers showed them to be generally free of the signs and symptoms of cardiovascular disease.[5]

Whatever the dietary intake comes to be in wilderness survival and living off the land, there are common-sense considerations to achieving a balanced diet. Of these considerations, variety is probably the most important. Variety can help ensure that the requirements for energy-yielding macronutrients (fat, protein, and carbohydrates) and micronutrients (vitamins and minerals) are met. Variety essentially translates to a balance between the intake of animal and plant foods. Although this balance depends on factors of climate, competition, and food-type availability, it is nevertheless worth every effort to attain.

ESSENCE OF SURVIVAL

Historically, many people who foraged for food from the environment were the victims of aircraft accidents isolated in far-off regions of the world. They needed to supplement meager rations—the food in their pockets and possibly the food contained in a survival kit (if they had one). Castaways who washed up onshore after their boats sank were forced to collect food from the coastal environment. Others who have scavenged for food included people who became lost, although most of these were seldom lost long enough for the lack of food to become a real life-or-death issue. For those who were not found within the typical 72-hour window, the ability to recognize and gather locally available wild food may have enhanced their ability to fend off the assaults of the environment and prolonged their lives. Anecdotal reports of these survivors describe how many of them scavenged for berries, roots, and various forms of animal life to satisfy their hunger. Gathering food from the environment becomes important when people find themselves unable to obtain food from conventional sources and a long-term survival situation is anticipated.

There are other circumstances in which being able to gather wild foods could prove very valuable. For example, during periods of civil unrest, particularly when traveling overseas, it is possible to find oneself in a situation in which grocery stores, supermarkets, and other sources of food are not accessible. Being able to forage for food could provide a viable alternative to jeopardizing safety by coming into contact with unfriendly locals. Knowing how to procure food could be a valuable skill in areas where natural disasters have caused extensive damage to the affected community's infrastructure and food supply. Being able to live off the land and consume unusual foods could also become important if one is taken hostage by a terrorist or criminal group. It may therefore be particularly relevant in parts of the world where hostage-taking is prevalent. Self-sufficiency in the gathering of food and water could be particularly important to an escaped hostage attempting to return to friendly hands, as opposed to depending on hostile locals or terrorist sympathizers. In each of these situations, one may need to live

TABLE 65-1. Recommended Dietary Allowances for Energy

GENDER	AGE (yr)	KILOCALORIES PER DAY*
Male	11–14	2500
	15–18	3000
	19–24	2900
	25–50	2900
	50+	2300
Female	11–14	2200
	15–18	2200
	19–24	2200
	25–50	2200
	50+	1900

*Based on light to moderate activity levels.

off the land to augment the provisions one has or, in a worst-case scenario, to replace food that is no longer available.

Although being able to gather food can be important, a person should not believe that he or she will definitely be able to live off the land. Believing that foraging for one's own food is possible in all situations is not realistic. The time of year, the environment, inadequate knowledge, lack of skill, and injury may severely limit a person's capability to forage for food. In fact, it may be more beneficial to limit activity by not aggressively searching for food rather than expending the limited amount of energy stored in the body in trying to procure food that may provide only meager energy in return.

Most survival manuals currently available contain numerous pages devoted to identification of edible and toxic plants, techniques used to trap animals for food, and primitive fishing methods used to procure aquatic animal life. The techniques commonly described are usually those used by indigenous people who still have to live off the land for their sustenance. The techniques depicted are presented as though they are methods that the average person could utilize when in an emergency and needing food. The fallacy lies in the belief that, based on a diagram or two accompanied by a paragraph of narrative, the techniques can be learned, remembered, and then used by the reader at some later date. More often than not, the techniques described *cannot* be learned by reading a book. A lifetime of training (or certainly months of practice) is needed for the techniques to become truly effective for the individual. Few people are willing to devote the time needed to develop expertise in the use of primitive food-gathering methods to a level of proficiency that they could depend on in a survival situation.

During certain periods of the year in many parts of the world, very little, if any, natural food is available. The local people in these areas stockpile sufficient food to last through the lean times until natural sources of food are once again available. Even a well-trained and equipped survivor who arrives during these "lean times" is hard pressed to live off the land and can only hope that rescuers arrive promptly.

Living off the land is usually thought of as a task required to maintain life during a long-term survival experience in a remote corner of the world while awaiting rescue. Although this scenario does occasionally happen, most of the survival experiences in North America are short-term, during which the need to gather food from within the environment is not a survivor's primary focus. However, even in a short-term experience, knowing how to live off the land could prove beneficial to the survivor's physiologic need for food and to his or her psychological peace of mind. In some instances, being able to live off the land could make the difference between surviving and dying.

The methods presented in this chapter are simple, are practical, require a minimum of equipment, and, most importantly, are relatively easy to use in the field. Some practice will be required, but these procedures and techniques do not require a huge investment in time to develop basic skills.

WATER PROCUREMENT AND PREPARATION

Besides the need for energy-yielding macronutrients and micronutrients, the requirement for water must not be overlooked because water is the most essential nutrient. Current recommendations on water intake recently released by the Institute of Medicine suggest daily intake of about 11 cups of total water for women and 16 cups for men.[16] These recommendations are based on the assumption that about 80% of a person's daily water intake comes from drinking beverages and the other 20% comes from water contained in food. Prolonged physical activity, heat exposure, extreme cold, and increased altitude increase water losses and therefore may raise daily fluid needs.

Many survivors begin their emergency already dehydrated (hypohydrated) and continue to dehydrate further when water supplies are limited and the quality of the available water is suspect. There have also been cases in which individuals needed water but, because of their fear that the water source was contaminated with *Giardia, Cryptosporidium,* or other harmful pathogens, delayed drinking or chose not to use the water at all. As a general rule, because dehydration can very quickly reduce a survivor's ability to function efficiently and safely, it is usually better to drink the impure water. If the water was contaminated, the onset of symptoms will be days away. Hopefully, by then the individual will have access to medical care for treatment. Remember, doctors can cure giardiasis or cryptosporidiosis, but they can't cure "dead"! It should also be noted that gastrointestinal problems usually associated with drinking contaminated water are more commonly a result of poor personal hygiene habits.

The need to maintain body water levels and the physiologic impact of dehydration have both been well documented in the medical literature (see Chapters 10 and 11). In priority order after shelter and defending body temperature, the need to locate, procure, treat, and preserve water is the survivor's next most important need. Although water is important in every environment, lack of water becomes critical very quickly in hot, dry environments. Dehydration is also a critical problem at high altitude and at high latitudes where ice and snow must be melted to produce water. People who travel in these areas frequently have great difficulty obtaining water because of the hassles involved in collecting snow and ice, often under extreme weather conditions, and then having to produce the heat needed to melt it. In hot arid regions, where no surface water exists and only infrequent precipitation occurs, depending on tanks, springs, or other natural sources for water is a very questionable practice. Experienced desert travelers abide by the adage, "If you don't have it with you, you won't have it!" Lack of water in extreme conditions can lead to incapacitation within

hours and death within days. Locating sources of water should be an important priority for the survivor.

Finding and Collecting Water

Throughout much of North America, water is usually available from open sources of water, such as lakes, ponds, rivers, and streams, and it can usually be obtained fairly easily. Despite this abundance of water, there have been cases in which survivors were unable to reach a water source because of their injuries.

Fundamental to finding water is the recognition that it will always seek the lowest level possible and that, if present, some form of vegetation most likely indicates its presence. A good strategy to locate water is to first find a vantage point from which it is possible to scan the surrounding countryside. A person should slowly and methodically search for any indicators of water, such as green vegetation, birds flocking to specific areas, trails left by domestic or wild animals, and even large rock formations from which springs may originate or where water can become trapped. Human-made sources of water, such as windmills, tanks, dams, and irrigation canals, might also be observed. Look for water in low-lying areas, such as depressions, sinks, or tanks, where rainfall or melting snow is likely to collect. Water can often be found in these areas long after the last precipitation, especially if the areas are shaded. In arid areas where there is little vegetation to obstruct a person's view, a pair of binoculars can save a lot of walking.

Green Vegetation

Although the presence of any living vegetation is an indicator of subsurface water, the amounts of water are usually miniscule, and it is not available in sufficient quantity to justify any expenditure of energy in an attempt to dig for it. Most arid-area plants survive because their deep roots extend well below the earth's surface to gather small quantities of water available in the soil. Although it may not be appropriate to dig for this water, the process of plant transpiration can be capitalized on to collect a significant amount of water in a relatively short period of time. This process is described later in the chapter.

Animal Trails

Most animals require water at regular intervals, usually once a day. By observing the movement of animals, it may be possible to determine the specific location of nearby water sources. Distinct trails are developed over time by both wild and domestic animals that travel to and from water sources on a regular schedule. Pay attention to forks in animal trails because they often indicate the direction of a water source. If an animal trail forks into two trails, then it is possibly leading a person away from a source of water. Conversely, if trails come together, an individual may be walking toward a source of water or food. Even though the direction to a water source can be determined, the distance to that source may be too far for the survivor to realistically reach on foot. Birds and animals have a far greater capability to travel long distances without water than a human does. Consequently, it is usually better to stay in one place as opposed to wandering around the desert looking for uncertain water sources.

Bird Movement

Most birds require water at least once per day. By watching their movement, especially in morning and evening hours of the day, a general direction of a water source may be determined. Once again, the urge to travel must be weighed against unknown distances and the questionable quality of water sources.

Figure 65-1. Attach a lanyard loop to your water bottle. (Courtesy Peter Kummerfeldt.)

Open Water Sources

Collecting water from open sources is usually the easiest method available to the survivor. However, caution should be exercised. Lakeshores and the edges of rivers can be hazardous. Crashing waves, swift moving water, undercut river banks, and unstable footing can all create problems. Swampy shorelines, heavy vegetation, lakes, streams, or ponds surrounded by cliffs, other difficult terrain, or unsafe ice conditions may preclude a person from getting close enough to a water source. Don't risk life and limb trying to climb or reach the water's edge when safer strategies can be used. Tie a line to a water bottle or container of some type and throw it or lower it into a water source from a safe location or vantage point. Because most water containers do not come with a reliable attachment point when the container is uncapped, make one by duct taping a loop of parachute cord or nylon line to the side of the water container (Fig. 65-1). Do not use the retaining strap that connects the cap to the water bottle for this purpose. In many cases, this strap will break or pull free from the full bottle as it is being retrieved from the water source, resulting in the loss of the bottle.

In some instances, water sources may be very muddy or silty and need filtering or settling before they can be used (Fig. 65-2). Rivers originating from glaciers carry large amounts of "glacial flour" that should be removed before consumption (Fig. 65-3). This is best accomplished by allowing the water to settle overnight and then filtering it through fabric before drinking.

The water in some lakes, particularly those found in the western United States, contains high concentrations of calcium carbonate and calcium bicarbonate in solution, which make the

Figure 65-2. Muddy water should be allowed to settle and then be filtered. (Courtesy Peter Kummerfeldt.)

Figure 65-3. Rivers originating from glaciers are discolored by the glacial "flour" they are carrying. This water should be allowed to settle before filtering. (Courtesy Peter Kummerfeldt.)

water nonpotable. Lakes of this nature are usually easy to identify because the calcium salts that are leached from the ground are deposited in the form of white powder around the perimeter of the lake as the water evaporates (Fig. 65-4). Water containing high concentrations of calcium carbonate and bicarbonate tastes terrible and should not be consumed.

Figure 65-4. Calcium salts leached from the soil are a common sight around many western lakes. (Courtesy Peter Kummerfeldt.)

Figure 65-5. All water should be purified if possible before it is drunk. (Courtesy Peter Kummerfeldt.)

Water collected from rivers and streams should not be considered "pure" (Fig. 65-5). *Giardia*, *Cryptosporidium*, and other harmful pathogens found in water sources are not deactivated by aeration or exposure to ultraviolet. All water should be disinfected and purified using methods described in Chapter 61.

Seeps and Springs

The quantity of water produced by seeps and springs varies tremendously. In some cases, the amount will be only a few teaspoons per hour (Fig. 65-6). In other cases, gallons of water can flow from the ground in minutes (Fig. 65-7). Where the quantities are small, the flat edge of the opening of a plastic bag can be used to scoop up the water from a shallow source, or if it is flowing, to collect the water as it runs into the bag. A short piece of vinyl aquarium hose also works well for sucking up water from shallow collections or to recover water from narrow cracks in the rocks.

Figure 65-6. Desert seep. (Courtesy Peter Kummerfeldt.)

Figure 65-7. Desert spring. (Courtesy Peter Kummerfeldt.)

Tanks

After a rain, water collects in low-lying areas and may be found long after the last storms have passed through the area (Fig. 65-8). Check out any depressions, sinks, or other low places where water could gather. Remember, the presence of vegetation and animals could provide a clue to the presence of a water source. Water sources like these should be checked carefully because they are frequently contaminated with debris that has been washed into the drainage. Finding the remains of animals that have died nearby or in the water, animal droppings, or other similar contaminants necessitates boiling, the use of halogens, or a proper filtration system for disinfection.

Wells

It may be possible to locate abandoned open wells from which water may be obtained. Commonly, the rope and bucket used to lift water from these wells will be missing and a person will have to improvise a means to lower a container down into the well to retrieve the water. With a closed well, where the pump handle is present but secured, or where the water is piped to another location, it may be necessary to dismantle or damage the plumbing to access the water. This may not be possible without tools.

Windmills

Windmills that could provide a ready source of water are a common sight across North America, especially where little surface water exists (Fig. 65-9). Commonly, the water pumped to the surface is collected in a nearby tank or pumped directly into a trough from which livestock can drink (Fig. 65-10). If this is not the case, then it may be necessary to dismantle or damage the pipes associated with the windmill to gain access to the water. Without tools, this may not be possible.

Guzzlers

In arid areas, particularly in the western and southwestern United States, state wildlife agencies and conservation organizations have installed rainwater collectors called *guzzlers*. These water tanks can hold hundreds of gallons of water long after seasonal rains have passed (Fig. 65-11). A guzzler consists of a concrete, metal, or fiberglass apron designed to gather precipitation and feed it into a holding tank, where it remains until either it is consumed by thirsty animals or it eventually evaporates.

Dew

Dew forms on clear nights when the air temperature decreases and the water held in vapor or air suspension condenses on cool

A

B

Figure 65-8. After rainfall, water can be found in desert tanks **(A)** and other depressions **(B).** (Courtesy Peter Kummerfeldt.)

metal surfaces or on vegetation. Dew can be collected as it drains from inclined surfaces on which it has formed, or it can be sponged up using an absorbent material. Campers' towels (Fig. 65-12) are one of the best materials for collecting dew. These highly absorbent towels quickly absorb moisture and can then be wrung out into a container or squeezed directly into a person's mouth. A sponge is also very useful for collecting dew. Dew must be collected early in the morning before the sun's heat evaporates it.

Rain

Rain water can be easily collected by erecting a flat surface (Fig. 65-13). Water collects on the upper surfaces of any material (it doesn't have to be waterproof) and drains to the lowest point, where it is collected.

Snow

For a normothermic person, there is no reason not to "eat" snow as an auxiliary source of water. However, for a person close to being hypothermic or who is already hypothermic, eating snow may increase loss of body heat and exacerbate the medical condition. Survival case studies reflect that individuals who chose to eat snow frequently experienced cuts and abrasions to the mucosa as a result. If snow is the only source of water on hand, and there are no alternative methods available to melt it, the snow should be collected, compacted by hand, and consumed in small enough quantities for heat within the mouth to melt the snow.

When snow falls and settles on the ground, it undergoes constant metamorphosis. During very cold periods, there may be very little moisture in the dry, fine, or wind-blown snow that accumulates on the ground. Over time, as snow accumulates, the weight of the upper layers of snow and the earth's latent heat cause the snow closer to the ground to change. It becomes more granular in nature and more like ice than snow. When comparing equal volumes of snow, snow collected from lower levels near the ground produces more water than snow collected near the surface (Fig. 65-14). Also, less heat will be needed to convert this snow to water.

Water Machine. The most efficient technique to convert snow into water is with what military survival schools call "a water machine" (Fig. 65-15). A bag made from any available porous fabric (a T-shirt with the neck and armholes sewn shut has been used) is filled with snow and ice and hung near, but not directly over, a fire. The fire's radiant heat melts the snow in the bag and the water runs down to the lowest point of the bag, where it drains into a container. Continually refilling the bag with snow prevents it from burning. Gallons of water can be

Figure 65-9. Windmills are a common sight across the arid regions of the western United States and may provide a survivor a source of water. (Courtesy Peter Kummerfeldt.)

A

B

Figure 65-11. **A** and **B,** Guzzlers collect and store rain water for wildlife and others in need of water. (Courtesy Peter Kummerfeldt.)

Figure 65-10. Water pumped from the ground by windmills may be readily available to a survivor. (Courtesy Peter Kummerfeldt.)

produced quickly and safely using this method. The following are the major advantages of using the water machine method:

- Once the water machine is loaded with snow and positioned, no further action is needed until the bag needs to be filled with additional snow.
- A water machine works while a person is busy with other tasks. This saves energy.
- All the debris (e.g., leaves, grass, twigs, insects) commonly found in snow is filtered out by the cloth as the water drains into a container.
- A metal container, normally needed to melt snow over a fire, is not required when using this method.

Traditionally, snow is melted by placing it in a metal container and then applying heat (Fig. 65-16). Several problems soon become apparent when using this method:

- The size of container is usually quite small, which limits the amount of snow that can be melted at one time.
- When heat is applied to the container, the snow directly in contact with the heat at the bottom of the container melts and is converted to steam. This steam is absorbed by the remaining snow, leaving a space in the bottom of the

Figure 65-12. Campers' towels (e.g., Packtowls) are very useful for soaking up dew or water from shallow sources. (Courtesy Peter Kummerfeldt.)

Figure 65-13. A plastic bag, sheet of plastic, or other material erected as shown is a very efficient method to collect large amounts of rainwater quickly. (Courtesy Peter Kummerfeldt.)

A

B

Figure 65-14. Melting granular snow contained in a 1-gallon zipper-lock bag **(A)** produced the water shown in the second picture **(B).** (Courtesy Peter Kummerfeldt.)

Figure 65-15. Water machines are the best method to use to convert snow into water quickly. (Courtesy Peter Kummerfeldt.)

Figure 65-16. Melting snow in a pot is a very inefficient, time-consuming method of obtaining water from snow. (Courtesy Peter Kummerfeldt.)

Figure 65-17. The sun's heat and a sheet of black plastic can be used to melt snow. (Courtesy Peter Kummerfeldt.)

container. If the snow is not constantly stirred and pressed into the container, it is possible to scorch the bottom of the container and even to melt the solder used to seal the seams.
- Using this method requires constant attention.
- The water produced using this process tastes unpleasant (smoky) and often contains ash and other debris from the fire.
- The system is inefficient, increases the likelihood of being burned, and often results in damaged clothing.

Using Body Heat to Melt Snow. This is a very slow, inefficient method of procuring water. If this process is the only one available, a small quantity of snow (several cups) is placed in any available waterproof container. Preferably, this should be a soft plastic water bag, zipper-type bag, or other similar container that is then placed between layers of clothing. Because the amount of heat needed to convert snow to water is large and the amount of body heat available is finite, only small quantities can be melted at a time. Large quantities of snow will not melt fast enough to provide the survivor any benefit. Large quantities of snow may also cool the body too much.

Using the Sun to Melt Snow. Another method, frequently used in winter recreation, involves the use of a sheet of black plastic. A thin layer of snow placed on a piece of black plastic (or other dark-colored waterproof fabric) positioned in the sun will melt. The waterproof material should be positioned on an incline so that the melt water runs to the lower edge of the fabric and drains into a container (Fig. 65-17).

Digging Holes to Collect Subsurface Water

Even though water is not visible on the surface of the ground, it may still be present in the soil in sufficient quantity to be collected. Locate low-lying areas where water is most likely to have accumulated and dig down until damp layers of soil are located (Fig. 65-18). Over time, water may seep into the hole, where it can be collected. If no indicators of the presence of subsurface water are present, dig a hole in the outside bend of a dry river

Figure 65-18. Damp, low-lying areas are ideal sites to dig for water. (Courtesy Peter Kummerfeldt.)

bed. Look for a location where the centrifugal force of water flowing down river has eroded the outer bend of a curve and created a depression, where the last remnants of water flowing down the river will have accumulated.

Beach Wells

In a coastal, saltwater environment, it is possible to locate water sources near a beach that are fresh and potable. A hole dug behind the first line of sand dunes adjacent to the high-water mark will often fill with fresh water. Fresh water, which is less dense than sea water, will collect in the hole. Holes dug in sandy soils are very tenuous and tend to cave in constantly, which may make it necessary to shore up the sides of any hole dug in sandy areas with driftwood to prevent the sides from collapsing (Fig. 65-19).

Solar Stills

Solar stills use a sheet of plastic and the sun's heat to capture evaporation from soil or plants or to distill nonpotable water. The water evaporates from its source (e.g., soil, plants, or urine), condenses on the plastic sheet, and runs down the sheet into a collector, from where it is retrieved (Fig. 65-20). Solar stills are *not* a reliable method of obtaining water in arid areas. The quantity of water produced by a solar still depends on the amount of water contained in the ground. Because desert soils tend to hold little or no water, the amount that a survivor is likely to obtain must be balanced against the amount lost in the sweating process while constructing the device. In most cases, a person will likely lose more water than can be recovered from the still. Even if a solar still is constructed in ground that is saturated with water, its productivity in relation to the amount of effort expended is still questionable. If the ground is saturated, the other methods of water procurement described here will most likely work.

Water from Vegetation

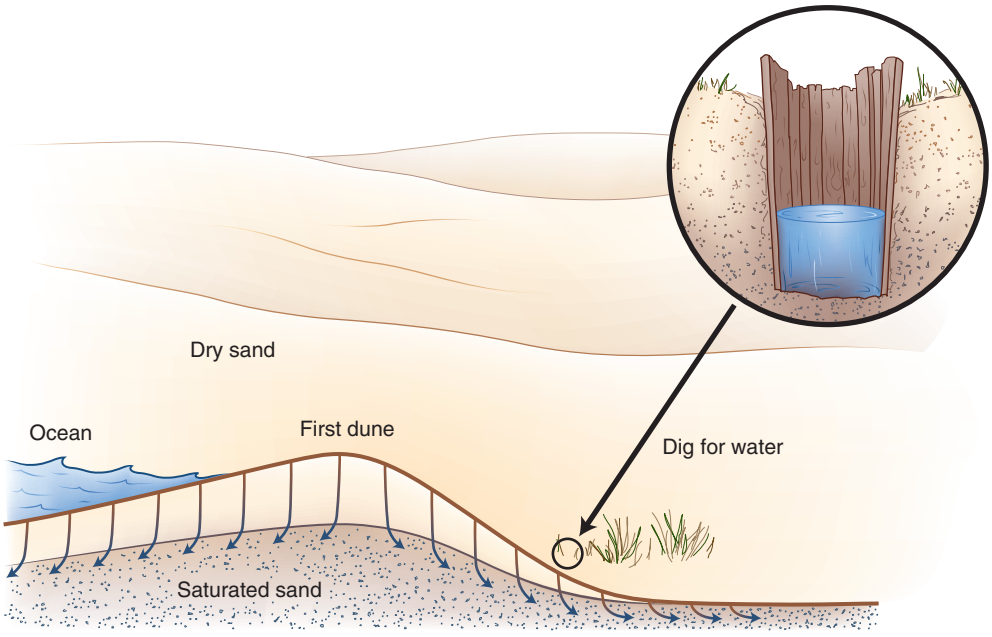
A person's ability to collect water trapped by plants or contained within them can be a valuable aid to combating dehydration. Once again, a line must be drawn between methods that are practical and those that are more myth based. Extracting water from a barrel cactus is a classic example of a survival "myth." Barrel cacti have been long featured in survival literature as a reliable source of water in arid regions (Fig. 65-21). Several issues make this practice very questionable. First and most importantly, the quantity of fluid that can be extracted from a barrel cactus is very limited. Second, the fluid that is removed is not beneficial and may in fact be detrimental to an individual's health. Third, accessing the interior of a barrel cactus requires a substantial knife or other cutting tool. The outer skin of the cactus is very tough and covered with long spines. Barrel cactus should *not* be considered a source of water.

Water Vines

Throughout tropical and subtropical regions of the world, vines can be found that can provide a reliable supply of pure water when other sources are not available. Water-producing vines varying in size from pencil thickness up to the thickness of an adult man's forearm can even be found throughout much of the southeastern United States. Select vines with a larger diameter. The greater the thickness of the vine, the more water it is capable of producing. Because water vines are woody and tough, they can be difficult to cut. A sharp knife or, better still, a machete, will be needed to sever the vine.

To determine whether a vine is a suitable water source, the outer layers of the vine should be deeply scored with a knife. Vines that exude a white latex sap or a colored or foul-smelling sap should be avoided. If no sap is observed, or if the sap observed is clear and without aroma, a section of the vine can be cut as a water source. A 24-inch-long piece of vine should be cut by severing the higher end first and then cutting the lower end. If the lower end is cut first, the water contained within the vine is drawn up by capillary action, and far less water will drain out by the time that the upper end is severed. This becomes apparent when the available knife is not large enough to cut through the tough vine quickly. In one test conducted in Florida by the first author, a 24- by 4-inch section of water vine produced more than 1 cup of water.

Once detached, the section of vine is held vertically, and the water contained within the vine is allowed to drain into a container (perhaps a cupped hand), where it should be further evaluated. Liquid that is colored should not be consumed. Liquid that has an unpleasant aroma, other than a faint "woody" smell, also should be discarded. A small amount of the water should be tasted. Water that has a disagreeable flavor, other than a slightly "earthy" or "woody" taste, should not be utilized for drinking. This source could be used to satisfy external hygiene needs. If the water is still being considered for con-



A

B

Figure 65-19. **A** and **B,** Beach wells dug behind the first sand dunes adjacent to the shore can produce potable water. (**B,** courtesy Peter Kummerfeldt.)

sumption, a small amount should then be held in the mouth for a few moments to determine if there is any burning or other disagreeable sensation. If any irritating sensation occurs, the water should be discarded. Ultimately, plant liquid that looks like water, smells like water, and tastes like water can be safely consumed in large quantities without further purification (Fig. 65-22).

Transpiration Bags

The use of clear plastic bags to enclose living vegetation and capture the moisture transpired by the leaves can be an effective method of collecting water (Fig. 65-23). A plant's survival depends on its ability to gather water from the soil. This water is passed up through the plant's roots, stems, and branches and is finally released back to the atmosphere through pores in the

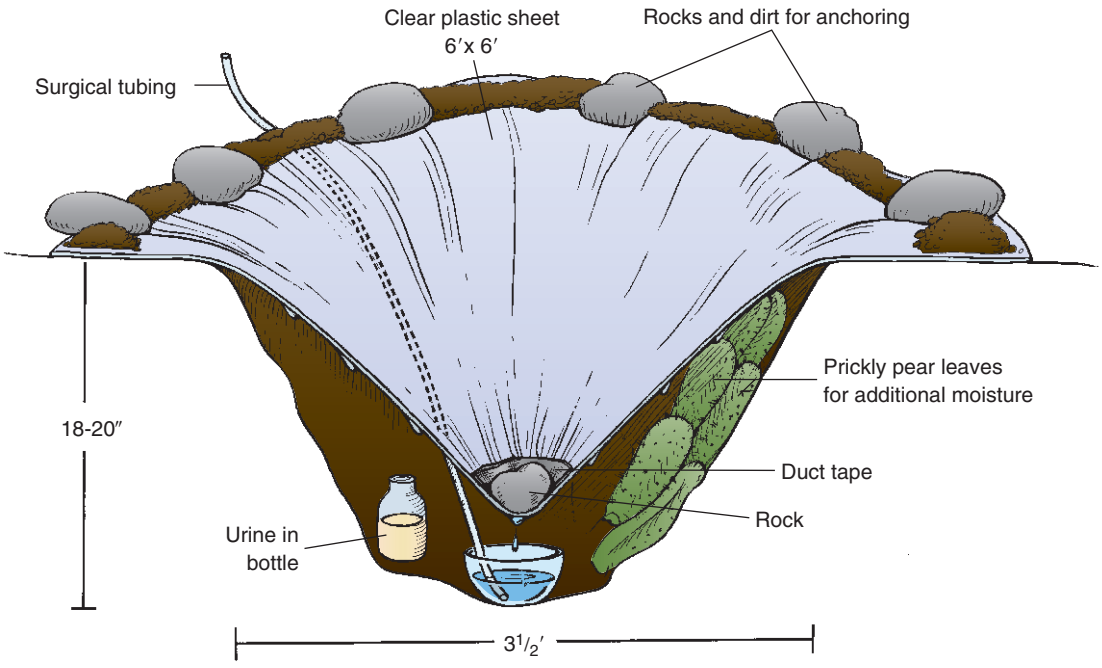


Figure 65-20. Solar stills are not a reliable procedure for obtaining water from the ground.

Figure 65-21. Barrel cacti do not contain water or water substitutes. (Courtesy Peter Kummerfeldt.)

A

B

C

D

Figure 65-22. A–E, Water vines contain large quantities of water that can be recovered by cutting out a 1-inch section and draining the water into a container. (Courtesy Peter Kummerfeldt.)

Continued

E

Figure 65-22, cont'd. Water vines contain large quantities of water that can be recovered by cutting out a 1-inch section and draining the water into a container.

leaves as water vapor. This process is called *transpiration*. Water vapor is captured by enclosing as much living vegetation as possible within a clear plastic bag and sealing the opening shut with a cord or duct tape. The vegetation should be given a vigorous shake before placing it in the plastic bag to remove any insects, bird droppings, or other materials that might contaminate the water. Within a short period of time, water will begin to condense on the inner surface of the bag and collect into water droplets.

Over a period of hours, the droplets will accumulate and drain to the lowest point. The quantity of water obtained in this manner depends on the amount of water in the ground and the type of vegetation used. Other factors that determine water production include the amount of sunlight available, the clarity of the plastic bag, and the length of time the process is allowed to work. It is not uncommon to find that 2 or 3 cups of water, and sometimes much more, have accumulated over a 6- to 8-hour daylight period (Fig. 65-24). This water is contamination free and does not require further purification.

Transpiration bags do not work at night, and they do not work well when opaque or colored bags are used. The process of transpiration slows down when it becomes dark. Depending on the temperature, the plastic bag can be left in place for 2 to 3 days, at which point the leaves become damaged by the heat that develops inside the bag, causing the process to stop. The water that collects can be removed from the bag by disassembling the apparatus and pouring out the water, or by punching a small hole at the lowest point in the bag and allowing the water to drain out. Neither of these procedures is optimal because one requires that the bag be reassembled and the other necessitates repairing the hole in the plastic bag. The best way to remove the water without disturbing the bag is to insert a length of vinyl aquarium hose through the neck of the bag down to the lowest point where water will collect. The water can then be sucked out or possibly siphoned into a container. When enclosing vegetation in the plastic bag, it is advisable to place a chicken-egg–sized stone in the lower corner where the water will collect. The weight of the stone creates a separation between the enclosed plant life and the water and precludes the plant saps from leaching into the water.

Figure 65-23. Transpiration bag collecting the water transpired by desert willow. (Courtesy Peter Kummerfeldt.)

Using the transpiration process is a practical method of collecting water in desert areas, but it is also useful in other areas where surface water is not available. Unlike in deserts in other parts of the world, a considerable amount of vegetation is found in North American deserts. This is vegetation that can be used in the transpiration bag process. Most vegetation in desert areas is thorny, so considerable care should be taken to keep these thorns from tearing the plastic bag. Desert willow, a nonthorny shrub commonly found throughout the American southwest, is an excellent source of water. The poplar family of trees, including aspen and cottonwood, found in more temperate areas, is another good source when using this method of water procurement. Because the trees and shrubs that produce the most water are deciduous (lose their leaves in winter), the transpiration bag process is generally limited to the time of year when vegetation is in leaf.

How Long a Person Can Live without Food

Other than external factors such as wind, precipitation, and temperature extremes, individual physiology and tenacity to live remain the primary determinants for survival without food or with limited caloric intake. An obese person carrying a large amount of body fat who arrives in a survival situation will have more fuel to burn than a thin person. Other factors notwithstanding, this individual should live longer. Skinny but determined survivors might live longer than expected because of their tenacity.

A distinction must be made between having no food available and having limited supplies. If the consumption of food is lower than the rate at which energy is expended, the body uses its reserves of fat, carbohydrates, and proteins at a pace directly related to the rate at which the energy is depleted. Without any food, most people die within 40 to 60 days. Self-imposed food

A

B

C

Figure 65-24. Enclose a large amount of leafy vegetation in a clear plastic bag and seal the neck **(A).** Water transpired by the plant condenses on the inner surfaces of the bag and drains to the lowest point **(B),** where it can be sucked out using a piece of vinyl hose **(C).** (Courtesy Peter Kummerfeldt.)

fasts, such those endured in Irish prisons by IRA militants, ranged from 50 to 60 days with no caloric intake before death. When sleep deprivation and danger are combined with little or no food, a person's awareness, judgment, and ability to concentrate decrease rapidly. Individuals attempting to survive with little or no food become apathetic, lethargic, confused, and indifferent. Consequently, they are not likely to stay alive for very long in a physiologically challenging environment.

How long a person can live without any food varies tremendously. In adult volunteers who fasted for 30 to 40 days, weight loss was marked (25% of initial body weight). In more prolonged starvation, weight loss may reach 50% in adults and possibly more in children. Loss of organ weight is greatest in the liver and intestine, moderate in the heart and kidneys, and least in the nervous system. Emaciation is most obvious in areas where prominent fat deposits normally exist. Muscle mass shrinks, and bones protrude. The skin becomes thin, dry, inelastic, pale, and cold. The hair is dry and sparse and falls out easily.

Most body systems are affected. Achlorhydria and diarrhea are common. Heart size and cardiac output are reduced, the pulse slows, and blood pressure falls. Respiratory rate and vital capacity decrease. The main endocrine disturbances are gonadal atrophy, loss of libido in men and women, and amenorrhea in women. Intellect remains clear, but apathy and irritability are common. The victim feels weak. Work capacity is

diminished because of muscle destruction and eventually is worsened by cardiorespiratory failure. Anemia is usually mild, normochromic, and normocytic. Reduction in body temperature frequently contributes to death. In famine edema, serum proteins are usually normal, but loss of fat and muscle results in increased extracellular water, low tissue tensile strength, and inelastic skin. Cell-mediated immunity is compromised, and wound healing is impaired. Total starvation is fatal in 8 to 12 weeks.

For more information on starvation, see Chapter 63.

The season and weather establish additional constraints on a survivor's longevity. In a benign setting, with warm temperatures both day and night, no precipitation, plenty of water available, and wood for a fire, the physiologic need for food is not as great. On the other hand, a survivor in a cold, wet, and windy environment, with inadequate clothing and no shelter, fire, or food to generate heat, will quickly succumb to hypothermia. With food, the survival outcome for an individual is still not guaranteed. However, provided that other priorities are met, life may continue long enough for a survivor to be found alive.

In a cold environment, generating heat to preserve body core temperature can be very difficult without adequate food intake. Shivering, which is a primary mechanism by which the body generates heat, rapidly depletes glycogen stored in the liver and muscles unless adequate food is available. Lacking sufficient clothing and food to protect against environmental insult, survivors must rely on their ability to build a shelter and start a fire to maintain core temperature.

Survival situations demonstrate the law of diminishing returns. During the initial hours of an emergency situation, survivors are in the best condition they will enjoy during their survival experience. The impact of the environment and the lack of food, water, and sleep result in continuous degradation of the body's ability to function normally and eventually take a toll on the survivor's ability to accomplish the tasks needed to survive. Any "heavy work" should be accomplished early. Strength, mobility, balance, and dexterity diminish as each day passes. The survivor's objective is to delay this degradation for as long as possible through the intelligent use of practical food- and water-gathering techniques and in so doing increase the chances of being rescued alive.

The ability to select, gather, and prepare natural foods, even in short-term experiences, is valuable physiologically, but perhaps even more valuable psychologically. When gathering food, survivors are actively involved in surviving, contributing to their own well-being. Although the amount of food gathered may be small, the satisfaction derived from catching a fish, snaring a squirrel, or gathering a hatful of edible berries can be a great morale builder.

Some people who are used to having all their food come from packages, cans, or other containers need to overcome their reluctance to eat nontraditional foods that come packaged in skin, hair, scales, or feathers. These aversions cause people to avoid consuming available wild foods that would enhance their chances of survival. Reluctance to eat insects, for example, is based in large part on the Western cultural belief that insects are "dirty" and that eating insects is done only by those who cannot afford better food. Additionally, the vast quantities of processed food available in developed countries have produced feeding habits in which people only consume the very best of the available foods and discard the rest. Survivors will not be so fortunate that they can pick and choose what they eat. They may have to capitalize on *any* potential food that comes their way.

Procuring wild foods involves procedures and techniques that some might view as cruel, unethical, or even illegal. For example, with the exception of trapping fur-bearing animals during legal seasons and the use of traps by licensed animal control personnel to remove "problem" animals, the use of snares to obtain food is against the law. Trapping and snaring techniques have been determined to be illegal because they are so effective and because they are virtually impossible for wildlife managers to monitor. Their effectiveness is the very reason they are highly recommended for survival. Under normal circumstances, procuring food from the environment is governed by federal, state, and local laws and regulations. Killing animals for food outside of prescribed hunting seasons is forbidden. Gathering plant foods and killing animals is specifically prohibited in national parks and other similar sanctuaries. Accepted hunting conventions also disfavor killing females with babies and younger animals. When faced with starvation and the choice between killing an animal and digging up enough plant bulbs to make a meal, it becomes a question of common sense. The survivor should gather necessary food and leave legal issues to be sorted out after the fact. It is extremely unlikely that a survivor who could clearly demonstrate a legitimate need would be charged with an infraction of the law. This does not mean that, in a short-term survival experience, it is appropriate to disobey the law, particularly if there are reasonable alternatives.

Feeding oneself in a long-term survival experience may necessitate harvesting and butchering wild game animals. These skills were once common in Western culture but nowadays are unusual. To some people, the act of taking any life is reprehensible, so even in a survival situation, when faced with killing an animal for food, they will have great difficulty or even may be unable to accomplish the task. But most commonly, hunger drives survivors to carry out what they need to do to stay alive.

When considering what should be eaten in a survival situation, some people focus all their efforts on plant foods. This misguided approach may be the result of advice given in out-of-date survival manuals and from well-intentioned, but ill-informed, writers of contemporary "how-to-survive" articles. Many of these articles devote numerous pages to the identification and use of plants to sustain life. Except for the short term, it is almost impossible to survive indefinitely on gathered plant life alone, if it is even available. Fats and proteins provided by eating meat are far more sustaining than the carbohydrates provided by eating vegetable matter. If plant and animal foods are both available, survivors should attempt to balance their diets by consuming fat, proteins, and carbohydrates in order to maintain health for as long as possible.

SUCCESS STORIES

Despite the difficulties of living off the land, survival for long periods is still possible when little or no food is available. Helen Klaben and Ralph Flores[21] survived 49 days in the Yukon in a wrecked aircraft on four $3^3/_4$-ounce cans of sardines, two 7-ounce cans of tuna fish, two 1-pound cans of mixed fruit salad, one 1-pound box of saltines, five small pieces of chocolate, and 2 tablespoons of Tang. Bob Gotchen survived for 58 days during the winter following a forced landing in Northwest Ter-

ritory, eating only a few mouthfuls of frozen fish and losing 74 pounds in the process. James Scott survived under a rocky ledge in the Himalayas for 43 days during the winter without food before being rescued. In 1972, a Uruguayan soccer team stranded in the Andes by a crash-landing survived for the next 72 days with little food. In 1979, Brent Dyer and Donna Johnson walked out of the Idaho wilderness 19 days after their plane crashed. In the last two instances, the survivors, lacking other foods, consumed the flesh of humans who had died in the accidents. Despite their psychological disinclination to eat the flesh of another person's body, it was something that they did in order to survive. Based on the accounts of these incidents, published in *Alive: The Story of the Andes Survivors* by Piers Paul Read[30] and *The Sacrament: A True Story of Survival* by Peter Gzowski,[13] it could be argued that the small amounts of human flesh they ate were of minimal physiologic benefit, but of significant psychological value. Although there is no physiologic reason why the flesh of human beings should not be consumed in an emergency, we are unwilling to do so as a result of our cultural beliefs and the longstanding prohibition against cannibalism. Eating the flesh of another human to survive after that person has died accidentally is not necessarily the wicked act of a demented person. Rather, it is a last choice, a means to an end, for a starving survivor.

WILD ANIMAL FOODS

Food available to a survivor may come from many sources, including snacks or other incidental food that survivors may have placed in their pockets, emergency food contained in survival kits, and last but not least, food that might be obtained from the surrounding environment. The ability to procure natural foods from the land depends on the following:

- Knowledge of natural foods available for each season in the relevant geographic region
- Some degree of proficiency in food-gathering methods and techniques
- Physical ability to gather food
- Availability of equipment needed to gather food or ability to improvise the equipment
- Skills needed to prepare food for consumption

Knowing the environment and the techniques to procure and prepare animal life for food could prove crucial to the successful conclusion of any survival situation. This section discusses the techniques a survivor might use to harvest and prepare commonly available animal life found throughout much of North America.

Mammals

All mammals are edible, but because of larger mammals' size, their ability to injure or kill the survivor, and the survivor's lack of a means to kill them, it is unrealistic to count on them all as a food source. Historically, survivors have targeted the rodent family (squirrels, muskrats, porcupines, marmots, nutria, rats, and mice) and other smaller mammals, such as rabbits and hares, for food. These animals may be captured or killed with the simple equipment and basic food-gathering skills possessed by any prepared survivor.

Rabbits and Hares

Members of this animal family are found in almost every environment, from the Arctic to the tropics, and have served as the main course for many survivors' dinners. Rabbit and hare populations are cyclic; at the peak of their reproductive cycle, they can be extremely numerous and easy to catch. This is especially true during the winter months, when they pack defined trails through the snow. This is a well-known characteristic of snowshoe hares, which are found throughout northern portions of the United States, Canada, and Alaska.

Some people are reluctant to eat rabbit. This preference is sometimes based on cultural traditions. In the West especially, rabbits are thought of as "Easter Bunnies" or as "pets" and not as a food source. Additionally, because rabbit meat is not a common product for sale in the supermarket, people conclude that it is not a "good" food item. In other parts of the world, rabbits and hares are an accepted meat source. They certainly should be welcomed as a food item by a survivor. As with most wild animals, they contain very lean, high-protein, low-fat meat.

Simple loop snares described later in this chapter are an effective means to procure rabbits and hares. Properly set out in large numbers (at least 15 snares for every animal you expect to catch), loop snares work constantly for the survivor while other tasks are being accomplished. Rabbits and other small animals usually die quickly when caught in a snare. However, if the animal is found to still be alive when the snare is checked, it can be killed quickly by striking its cranium with a stout stick or by dislocating the neck vertebrae. Dislocating the vertebrae can be quickly accomplished by lifting the animal by its hind legs and then forcefully striking its neck directly behind the ears with the stick. Unconsciousness and death result instantaneously.

Once dead, the animal is suspended head-down by tying a line to one of its rear legs and then tying this line to an anchor (Fig. 65-25). To remove the hide, begin tearing the skin away from legs and tail and then proceed to separate it from the body by working the pelt over the forelegs and tearing it free from the front feet. Peel the hide free from the body until the only remaining attachment point is around the animal's neck. Use a knife to sever any remaining neck tissue and cut between the vertebrae if necessary. The hide can also be quickly removed from the body by cutting or tearing the skin at the mid-body point and then grasping each side and pulling in opposite directions. Discard the head, hide, and feet. Lacking a knife, one can separate the head from the remainder of the carcass by grasping the head in one hand and the carcass in the other and pulling in opposite directions.

Once the skin is removed, the rabbit's entrails need to be removed. Open the abdomen by cutting through the belly muscles along a midline incision extending from pelvis to sternum. Without a knife, the skin and belly muscle tissue can be opened by tearing it with your fingers. Once this is done, locate and carefully remove the bladder intact, especially if it contains urine. The remainder of the internal organs, including the lungs and heart, can then be lifted out of the abdominal cavity and from within the chest walls. A minimum of cutting is necessary with a knife, but if one is not available, these organs can also be torn out. The carcass should then be washed with water (if it is available) and wiped free of any residual blood or other body fluids using a cloth or available vegetation.

Rabbits, hares, and other small animals are usually cut into pieces before cooking. However, in a survival situation, the animal can be left intact and cooked whole using the cooking methods described later in this chapter. Cooking the meat preserves it and provides the survivor with the option that portions

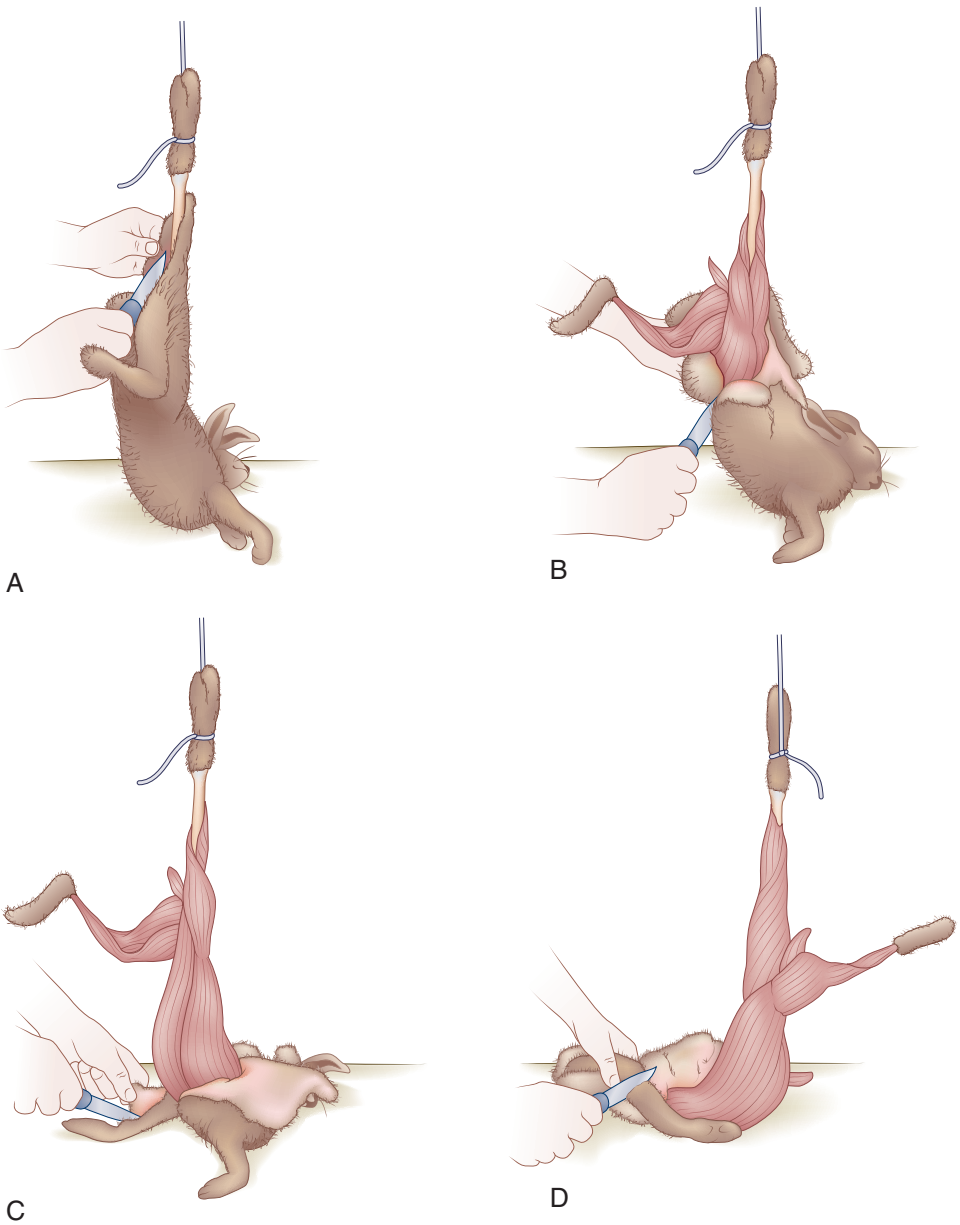


Figure 65-25. A–D, Anchoring one leg of the animal being skinned facilitates the quick removal of its skin (see text).

not immediately consumed can be saved for later meals. Generally, the heart, liver, and kidneys are not eaten. However, there is no reason not to consume these organs, especially when food is limited. These organs and the head should be retained and added to any other available ingredients for a "survival stew."

Rabbits and hares are susceptible to tularemia and other parasites, so if any snared animal appears to be in poor health, sluggish, or behaving unnaturally, the animal should not be handled or consumed. Rabbits and hares are also well known for serving as hosts to fleas and ticks, both of which will leave the host's body soon after it dies. It may be a good idea not to handle a freshly killed animal extensively for several hours. If you allow the animal to cool, pests will vacate the body.

Rodents

Despite the common aversion to eating rodents such as rats and mice, rodents can be an easily attainable and a valuable food source in a survival situation. They are widespread in distribution, usually found in large numbers, and can often be procured without the need for complex trapping procedures. Rodents commonly available to survivors in North America include squirrels (both ground and tree squirrels), porcupines, beavers, nutria, muskrats, groundhogs, prairie dogs, marmots, and a wide variety of rats and mice (Fig. 65-26). Of this list, the only rodents that North Americans generally accept as food items are members of the tree squirrel family, which are commonly hunted and eaten, particularly in eastern parts of the United

A B

Figure 65-26. Rodents. **A,** Red squirrel. **B,** Uintah ground squirrel. (Courtesy Peter Kummerfeldt.)

States. Groundhogs in the southeastern United States and prairie dogs and marmots in the West are still considered appropriate food. In other parts of the world, particularly developing nations, other types of rodents, including rats and mice, are used as a good source of protein. Other than our prejudices against eating rats and mice, there are no differences between types of rodents as to their edibility. They can all be used as a food source.

Rodents are prepared using the same procedures as those used to prepare rabbits and hares. With larger rodents, especially beavers and porcupines, the skin is tough and firmly attached, so removing it requires a sharp knife. When skinning a porcupine, considerable care should be taken to ensure that the sharp quills do not cause injuries. Puncture wounds caused by porcupine quills are painful and tend to become infected.

Larger Mammals

Large mammals may be available to a survivor, but lack of a rifle or shotgun usually limits sources to the smaller animals. It might be possible to salvage meat from the remains of larger animals that have been killed by predators or that died from natural causes. However, great care should be taken to ensure that predators are not in the vicinity when approaching a carcass. Most predators, particularly bears and mountain lions, will attempt to cover the remains of their kills or claimed animal carcasses with vegetation and soil. Covering the remains hides it from other predators and scavengers. A disturbed area surrounding a mound of earth and vegetation should serve as clear warning to the human survivor that a predator is nearby, and the area should be avoided.

Birds

Birds are found in every environment and vary in weight from ounces to many pounds. Although all birds are edible, there are a number of species that are not particularly appetizing. Birds that feed on fish and other aquatic animal life, as well as those that feed on carrion, are generally not considered desirable to eat. They can be eaten if no other food is available and if the survivor can tolerate the unpleasant odor and disagreeable taste of the meat. Skinning, rather than plucking the feathers, on these birds can reduce some of the unpleasant taste.

Because domestic versions (chickens and ducks) of wild birds make up a considerable portion of the meat eaten in a typical American diet, survivors are less likely to experience food aversion if they use birds as a food source. Wild game birds (turkeys, grouse, ducks, and geese) are similar to the domestic versions. Species of birds belonging to the gallinaceous (chicken-like) family (e.g., grouse, pheasant, chukar, turkey, and partridge [Fig. 65-27]), are often included in the diets of survivors. In remote areas where there is little contact with humans, ruffed grouse, spruce grouse, and ptarmigan in particular are very trusting in their behavior and may allow a person to approach close enough to catch them by hand. In less remote regions where they are more wary, these birds can still be killed or captured relatively easily using techniques described later in this chapter. Waterfowl, such as ducks, geese, and swans, are another group of birds that have helped to sustain survivors. Waterfowl are much more cautious and generally will not allow a person to approach. However, in the spring, when waterfowl molt and are unable to fly, they can be chased down and caught by hand or driven into nets erected close to the ground. They also can be killed or captured using the equipment and survival

Figure 65-27. Grouse, ptarmigan, pheasant, and other gallinaceous birds often allow a person to approach closely. (Courtesy Melissa Anderson.)

Figure 65-28. Wild turkey nest. All birds' eggs are edible and are best eaten before the embryos have developed. (Courtesy Peter Kummerfeldt.)

methods of gathering food described later in this chapter. The families of birds collectively referred to as "game birds" provide far more meat by weight than do the nongame species. This is primarily because they have developed large breast muscles, or pectorals, which account for about 15% to 20% of the bird's weight. Smaller birds of the songbird varieties are edible, but when the feathers and internal organs are removed, there's not much left.

All birds' eggs are edible (Fig. 65-28) and if collected early in the incubation process can be consumed in the same manner as a chicken egg. If incubation has progressed to the point where a "chick" has formed, the egg may still be eaten. However, to avoid aversion to this food source, the contents of the egg may have to be added to a "survival stew." Determining the state of maturation of the eggs in a nest is a difficult process. If a nest is located, leave the existing eggs in place, marking each egg if possible with a pencil mark or mud smear. Upon returning the following day, it is easy to determine which eggs are the newly laid ones. Any unmarked eggs should be "fresh" and can be cooked in the same manner as any domestic chicken egg. Always leave a few eggs in the nest to encourage the parent birds to return.

Preparation of birds for consumption is straightforward. Most methods used to catch a bird also result in its death. However, in the event that a bird is captured alive, the simplest method to kill it is to cut off the head after stunning it with a solid stick. Birds can be either skinned or plucked. Grouse, turkeys, pheasants, and other "chicken-like" birds are easy to skin, whereas ducks and geese should be plucked. Once waterfowl are plucked, the remaining pin feathers can be singed off by holding the carcass over the flames of a fire.

Having skinned or plucked the bird, cut off the head, neck, feet, and wings. To remove the internal organs, create an opening from the vent to the middle of the breast cartilage. All the viscera can be extracted through this opening by inserting a hand or fingers, depending on the size of the bird, into the cavity. Tear loose the organs from their attachment points in the abdomen and thorax. Some birds utilize a crop to store food until it is transferred to the gizzard, where it is ground into pieces small enough to be digested. The crop is located in a V-shaped cleft in front of the breast and is easily removed by separating this food-filled pouch from the carcass by either pulling it or cutting it free with a knife. Once the feathers and the internal organs are removed, the outer body and interior should be thoroughly washed to remove any remaining blood or other body fluids that contribute to putrefaction.

An alternative method to quickly separate the "meatier" portions of the bird from the carcass without a knife is demonstrated in Figure 65-29. The bird's breast is first exposed by tearing through the skin. The bird is then placed on its back on the ground. Place a foot on each wing and pull the breast free from the rest of the carcass by grasping it with both hands and pulling firmly straight away from the back.

Birds can be cooked whole or in pieces using any one of the cooking methods described later in this chapter.

Insects

The practice of entomophagy, the eating of insects, is well established in many parts of world, especially the developing countries in Africa, Asia, and Central and South America. In these regions, insects have long been considered either a staple part of the diet or a delicacy. Although insects are not a component of a typical American diet, they can provide a valuable food source for the survivor. More than 1400 varieties of insects found around the world are edible.[29] Among those more commonly eaten by humans are grasshoppers, crickets, ants, beetles, butterflies, moths, dragonflies, and termites (Fig. 65-30).

Not all insects are edible. Some contain powerful pharmacologic toxins that would cause significant illness if consumed; some insects (and other arthropods) can cause physical harm to the survivor. Spiders, scorpions, centipedes, millipedes, bees, hornets, and hairy caterpillars should be avoided. Because some insects harbor internal parasites, it is best to cook all of them before eating.

Advantages of eating insects include the following:

Figure 65-29. **A–E,** Removing the breast and leg meat from a grouse without using a knife. (Courtesy Melissa Anderson.)

- Insects are usually very easy to find.
- No sophisticated equipment or special skills are required to gather insects.
- Insects are often found in large quantities.
- Insects are nutritious and provide fats and proteins.
- Insects do not require extensive preparation before consumption.

Grasshoppers, crickets, and locusts (Fig. 65-31) are found throughout the world in a wide variety of environments. More than 1000 species of grasshoppers are found in North America.

Figure 65-30. Salmon fly. More than 1400 species of insects found around the world are edible. (Courtesy Peter Kummerfeldt.)

Figure 65-31. Around the world, grasshoppers are among the most commonly eaten insects. (Courtesy Peter Kummerfeldt.)

Grasshoppers contain 40% to 50% protein[32] and are best collected in the early morning when cold temperatures make them lethargic and easy to catch. Once captured, they should be left in a container overnight to purge. Then the head and any attached viscera, the smaller forelegs, the distal section of the hind legs, and the wings are removed before cooking. The insects can be placed in the coals of a fire and roasted until crisp or baked in hot sand beside or below a fire. Grasshoppers can also be roasted by skewering several on a stick and holding them over the flames of a fire. A stew can be produced by adding grasshoppers and edible vegetation to a pot of boiling water and cooking them until the vegetation is tender. Crickets are prepared and cooked in the same way.

Termites can often be collected in large quantities, drowned in water, sun-dried, and then roasted over low heat. Prepared in this manner, they will keep for many months.

Butterfly and moth larvae are rich in protein (37%) and fat (13.7%) and are edible. The larvae can be roasted over a fire or boiled and then roasted.

Earthworms should be placed in water and allowed to purge before they are consumed. Once purged, they should be dried in the sun or over low heat and then added to other ingredients of a survival stew.

Beetles and beetle larvae are edible and are eaten around the world nearly as often as grasshoppers and locusts. The insects should be roasted in the coals of a fire and then consumed. The indigestible head, legs, wings, and wing cases are best removed before the insect is cooked.

Snails are edible, easy to collect, and simple to prepare. Dropping snails into boiling water and allowing them to blanch for about 5 minutes will facilitate the removal of the meat from the shell. It can be eaten "as is" or added to a survival stew.

Reptiles

Reptiles include turtles, terrapins, snakes, alligators, crocodiles, and lizards. Because they are cold blooded, some of these animals hibernate and consequently are not available year-round.

Turtles have long been used as a food source. With the exception of a few species, all forms of sea turtles, freshwater turtles, terrapins, and land tortoises are edible. Musk and yellow mud turtles both have glands that produce a strong-smelling scent and because of this are unsuitable for food. Turtles found in North America, including the alligator snapping turtle and common snapping turtle, are all considered good to eat. The alligator snapping turtle can weigh up to 200 lbs (90.7 kg), and the common snapping turtle can weigh as much as 50 lbs (22.7 kg).

The only practical way to kill a turtle is to cut off its head. The next step is to cut off the feet. This involves three steps. With a sharp knife, cut the skin free from the shell around each leg, the neck, and the tail. Then peel the skin free from each leg and the neck. The final step is to sever each leg and the neck free from the body. The remainder of the edible portions of the turtle contained within the shell can then be withdrawn through one of the openings. With soft-shell turtles, the ventral shell can be separated from the dorsal shell by cutting through the tough cartilage that holds the two shells together. This exposes the internal organs, which can then be removed. Using a knife, separate the meat from the bones and trim off any fat as you do so. The meat should be washed in cool water and allowed to dry before cooking.

Lizards, which vary in size from minute geckos to crocodiles more than 20 feet long, are all edible. From a practical point of view, the smaller varieties provide little nourishment, so survivors are well advised to focus their attention on larger, more common species. Some lizards, such as the Gila monster (Fig. 65-32), found in a few southwestern U.S. states, and the Mexican beaded lizard found in Mexico, have a venomous bite and should not be pursued as a food source. When catching other species of lizard, no special care is required. However, although they may not be venomous, they still can bite, and some have claws. Preparing lizards for a meal requires a sharp, strong knife. If it is not already dead, kill the lizard by cutting off its head. Open the abdomen and thorax to remove the internal organs. The feet should be removed and the carcass rinsed with water. A lizard's skin is very firmly attached to its body and can be difficult to remove unless pieces of the body are boiled for at least an hour. Once cooked in this manner, the skin

Figure 65-32. Because Gila monsters are venomous, they should not be considered a food source. Other nonvenomous species can be eaten. (Courtesy Peter Kummerfeldt.)

Figure 65-33. Small crocodiles may be easy to catch and can be eaten. (Courtesy Peter Kummerfeldt.)

separates from the body without difficulty. The lizard can be consumed after this or, to improve the flavor, it can be roasted over the coals of a fire.

Alligators, crocodiles, and caimans, the largest members of the reptile family, are dangerous to humans and should not be considered as food unless they are small and can be handled safely. Small alligators and crocodiles (Fig. 65-33) can be caught by hand. They should be prepared in the same manner as other lizards. Most of the edible meat available from alligators and crocodiles is located in the tail and along either side of the spine. Because female crocodiles guard their nests and are very protective, caution should be exercised when attempting to catch small crocodiles or when attempting to gather crocodile eggs from the nest.

Snakes, both venomous and nonvenomous, are edible. One should proceed very cautiously when attempting to capture and kill a snake for food. A snake is best killed by pinning it to the ground with a long stick and then cutting off its head with a sharp knife. Take care not to be bitten and envenomed by a severed head. To remove the animal's skin, make an incision beginning at the vent and extending the full length of the body. By tying a piece of string to the snake's body at the head end and pulling against this anchor, the skin can be easily and quickly removed. A second slit is then made to access the snake's internal organs. These should be removed. Cook 1- to 2-inch sections of the snake using any of the techniques described later in this chapter.

Amphibians

Frogs found around water throughout the United States are all edible. However, as a general rule, only the larger species provide sufficient meat to justify the effort expended in pursuing them. Brightly colored frogs found in Central and South America are highly toxic and should be avoided. The most efficient method of catching frogs is to "gig" or spear them using a two- or three-pronged spear point attached to the end of a 6-foot or longer pole. Frogs can also be caught using fishing line and a hook baited with an insect. Catching frogs by hand should be avoided because this method expends far more energy than is returned by the relatively small amount of flesh provided. To kill a frog, sever the spinal cord behind the head with a sharp knife and then remove the skin. Cut off the feet and discard them. The frog's meaty legs are the parts commonly eaten, but the entire carcass, once skinned, has good food value. Frogs are best soaked in cold water for several hours and then cooked.

Toads secrete a toxin through their skin and are considered inedible. Unlike frogs that are most commonly found around riparian areas and waterways, toads are dry-land animals. Use this habitat preference as a differentiator when collecting frog-like animals for food.

Fish

Of all the animals available to a person trying to live off the land, fish are likely to be the most abundant and easiest to catch. They are also one of the few relatively available sources of fat—a food component not present in large quantities in any of the other smaller land animals that a survivor is likely to procure. Fish are often available year-round and can be caught using less sophisticated equipment than that needed to procure land forms of animal life. Fish of many species can be found in almost every water system throughout the world. Few bodies of water are completely barren.

All freshwater fish are edible, even varieties that are not usually used for food in North America, such as carp, suckers, and gar. Because fish tend to decompose quickly, they are best eaten soon after removing them from the water. All fish should be cooked before eating because some species are known to carry parasites, such as the *Anisakis* worm. This includes both freshwater and saltwater varieties. Depending on the species, scaling a fish is usually the first step in preparing it for a meal. Scaling is best done using a knife and scraping the skin from the tail toward the head of the fish. In some species, trout for example, the scales are very small and do not need to be removed before cooking. Other species of fish, such as catfish, are better skinned. A cut is made through the skin behind the gills completely around the body of the fish. The skin is then pulled from the body until only the white flesh remains. A Leatherman multi-tool or a similar tool with pliers jaws is particularly helpful in accomplishing this task.

With all the scales or skin removed, the viscera are removed by opening the abdominal cavity and removing all the internal organs. Once they are removed, a dark band of material is visible lying just below the vertebrae. This material, the posterior kidney, must be removed, which is usually accomplished by slitting the membrane that covers the kidney and scraping it out

Figure 65-34. **A–D,** Scaling and cleaning a fish. (Courtesy Peter Kummerfeldt.)

using a thumbnail or a sharpened end of a stick (Fig. 65-34). Rinse the cleaned fish with water to remove all remaining blood, slime, and other materials to enhance flavor and delay putrefaction. Be especially careful when cleaning all spiny rayed fish (e.g., bass, perch, catfish) because the spines in their fins can cause puncture injuries that may be painful or easily become infected. Do not eat the head, spinal cord, or liver of tropical fishes, and become familiar with unusual forms of seafood poisoning, such as occurs from eating puffer fish (see Chapter 66).

Crustaceans

Crayfish inhabit most water systems, both still and moving, throughout much of North America. They can be gathered by turning over rocks in a river or by dangling bait, such as a piece of meat, into the lower levels of a waterway. Crayfish are attracted to the scent given off by the meat and will grab it with a powerful claw and hang on until lifted from the water. Other than pulling off the head after it has cooked, no additional preparation is needed for this food source. They can be dropped into boiling water and left until they turn red. When no container is available, crayfish can be cooked in the coals of a fire. Most edible meat will be found in the tail, with small portions also found in the large claw.

Mollusks

Freshwater mollusks, commonly called mussels or "freshwater clams," live in many freshwater systems and are easily collected by hand. All varieties are edible. Although mollusks are very "chewy," they are nutritious, filling, and easy to gather. They must be thoroughly boiled to destroy harmful organic life absorbed by the animal. Certain shellfish should be avoided during toxic seasons and conditions (see Chapter 66).

Methods of Procuring Animals for Food

Snaring is one of the most productive methods available for procuring animal food. Snaring techniques for animals, birds, and fish are fairly easy to learn, require a minimum of equipment, and, most importantly, once set up, continue to work while the survivor is busy doing other things. Although there are traps that will kill or hold large animals, these devices usually require a good deal of energy and expertise to assemble. Survivors should concentrate their efforts on catching small

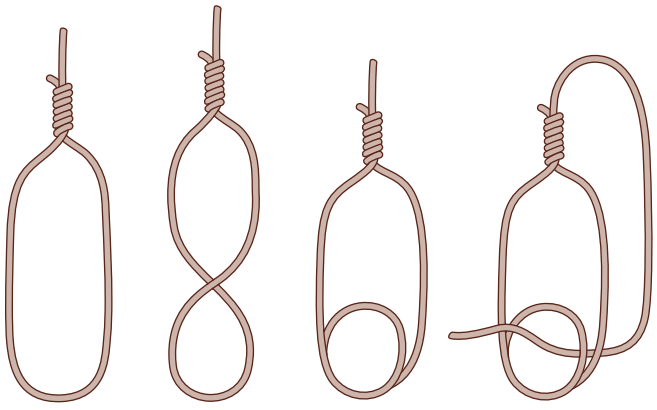


Figure 65-35. Snare wire "eye." Twist the loop into a figure-8 and then bend the upper loop into the lower loop as shown.

mammals, birds, and fish, which usually are more abundant, are less wary, and require less complicated techniques for capture.

Simple Noose Snares

The simplest form of snare is a noose made from wire, fishing line, cable, parachute line, or other similar material placed over the entrance to an animal's burrow, along its trail, or near a watering or feeding area. The snare must be securely anchored so that when the animal is snared around the neck, foot, or body, it either dies from strangulation or is held in place until the device is checked. In many parts of the world, a simple loop snare is a wildlife poacher's choice of method for illegally procuring animals. This effectiveness makes the use of a simple loop snare the food procurement method of choice for survivors trying to live off the land. Successful snaring depends on a person's knowledge of the animals' habits, ability to read the signs that the animals leave, ability to construct snares from available resources, and knowledge about where one is most likely to catch an animal. Snaring efforts should be focused on larger members of the rodent family, such as tree squirrels, ground squirrels, marmots, muskrats, woodchucks, porcupines, and prairie dogs. This family of animals, deer mice in particular, is known as a host for fleas that transmit a variety of infectious agents (see Chapter 41).

For the inexperienced survivor, setting out many simple noose snares provides the best opportunity to procure food. First, construct an "eye" in the end of a length of wire by twisting the wire back around itself several times. Twist the loop that is created into a figure-8 and then bend the upper loop into the lower loop as shown in Figure 65-35. The opposite end of the wire is then passed back through the eye, forming a loop that is adjusted according to the size of the animal being sought. Although simple loop snares can be made from many materials, soft brass wire, aircraft safety wire (0.020 inches, 0.51 mm), or braided picture-hanging wire are best (Fig. 65-36). Picture-hanging wire, floral stem wire, and bead wire are available from many hobby shops and are sold either by the pound or by the spool in a variety of gauges. Fifteen yards of 24-gauge wire should be included in a survival kit. This wire is strong, flexible, and difficult for an animal to gnaw through when trapped. The wire should be supple enough to shape into a loop, yet stiff

Figure 65-36. Suitable wire for snares can be found in hobby shops, automobile parts stores, and picture frame galleries. (Courtesy Peter Kummerfeldt.)

enough to retain the loop shape when it is positioned. Simple loop snares made from string, parachute line, shoelaces, and other similar materials may work, but more often than not, the animal is able to free itself and escape before the snares are checked. When materials other than wire are used, noose snares must be checked at least three times a day (morning, noon, and evening) to kill any snared animals that are still alive before they are able to chew through the noose and escape. When wire is used, the trap should be checked in the morning and in the evening. Commercial snares, which could be included in a survival kit, are available. Because they utilize a locking mechanism that only slides one way (tighter), they are even more effective than those that might be improvised (Fig. 65-37).

It is important to study animals in the area. Look for their signs, such as dung, footprints, food remnants, den sites, hair, or any other indicators of their movement or presence. Try to determine their size, what time of day they move, how far above the ground they carry their head, how big their heads are, and any other useful information that might assist in their capture. When constructing the snare, the size of the loop should be large enough to pass over the animal's head, and yet not so large that the animal can pass right through the loop. For a rabbit or hare, the noose should be fist-size and the lower edge of the loop should be placed three to four finger-widths above ground (Fig. 65-38). The wire should be secured to a nearby branch or stick, which prevents the animal from leaving the area once held by the snare. As the animal moves down the trail or emerges from or enters a den, its head passes through the noose, which tightens around its neck (Fig. 65-39). The animal usually will lunge forward in an effort to free itself, and thus be strangled. During the summer months, it may be difficult to clearly identify where animals are moving. Because of this, it may be more

Figure 65-37. Unlike an improvised wire snare, Thompson Lock Snares have a locking device that prevents an animal from escaping once it is held. (Courtesy Peter Kummerfeldt.)

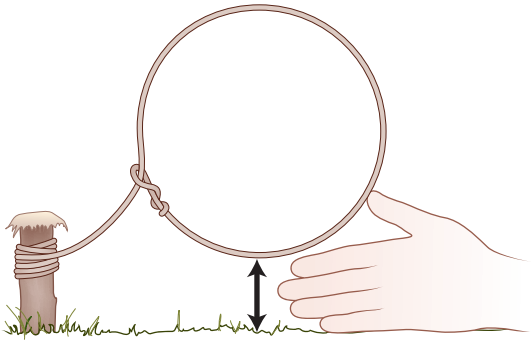


Figure 65-38. A rabbit snare loop should be about 4 inches in diameter and positioned 3 to 4 finger widths above the ground.

appropriate to identify burrows (Fig. 65-40) or feeding areas in which to place snares. Using vegetation to hide the snare and funnel animals into it increases the catch rate. During the winter months, squirrels, rabbits, and hares often use the same trail though the snow as they move from one location to another. These are ideal sites to position loop snares (Fig. 65-41). The packed trail guides a rabbit or squirrel directly into the loop. Once held, depending on the temperature, animals usually cool quickly and die. It may be necessary to thaw out a carcass before it can be skinned.

It is wise to situate snares in a pattern, known as a *trap line*, so that they can be easily found when checking for snared animals. Tying a flag to a nearby branch also expedites locating a snare, especially after snow has fallen. Knowing where each snare is located saves energy otherwise wasted in hunting for difficult-to-find snares, and it also reduces the likelihood of contaminating the area with human scent, which may discourage animals from moving through the locale.

Figure 65-39. A snare should be situated on a game trail, preferably in a location that is naturally confined.

Figure 65-40. In the summer, it may be difficult to tell where animals are moving. Locate their burrows or dens and place snares in the entryway. (Courtesy Peter Kummerfeldt.)

Figure 65-41. During the winter, snowshoe hares pack very distinct trails in the snow. These are ideal places to locate one or more snares. (Courtesy Peter Kummerfeldt.)

It is possible that a captured animal will be found still alive and only restrained by the noose. In this situation, it must be dispatched as quickly and humanely as possible. The expedient way to accomplish this is to use a stout club and strike the animal's head forward of the ears on the upper surface of the skull. Once is usually sufficient, but check for signs of life before handling the carcass. A second blow may be necessary. Be careful, because any animal, regardless of size, can inflict a nasty bite if handled.

For larger animals, a stronger noose is necessary. This can be made by twisting two strands of wire together and then forming a loop from the doubled wire, or by using nooses made from braided parachute line or other similar materials. Tie a secure fixed loop in the end of the line using either an overhand loop knot or a bowline and then position the loop over a game trail (Fig. 65-42). Tie the opposite end to the trunk of a nearby tree or stout overhead branch. It may be necessary to use vegetation to hold the noose open when using very supple materials. A good rule of thumb is that it will take at least 15 snares to successfully catch one animal.

It is entirely possible that an animal will escape after being snared. Sometimes it may be caught by one or both hind feet and manage to escape by twisting the wire until it breaks. Predators, both four-legged and winged, will quickly find and eat a snared animal, leaving little for the survivor. This is another reason why it pays to check traps frequently. It is also possible that the loop will be disturbed or moved out of place without catching an animal. This usually happens because the snare has been set too close to the ground or the loop is too small. As with virtually all skill development, practice builds proficiency.

Baited Snare

A simple loop snare with a long line attached to the end can be positioned where animals or birds are feeding. Bait is placed within the circumference of the snare. When the animal (or bird) steps into the noose, the watching survivor pulls the loop closed. Many nooses attached to pegs can be placed throughout a feeding area where birds or animals congregate. As they feed, their legs become entangled in the snares. A snare could also be placed where an animal would have to reach through a noose to retrieve bait and become snared as it did so.

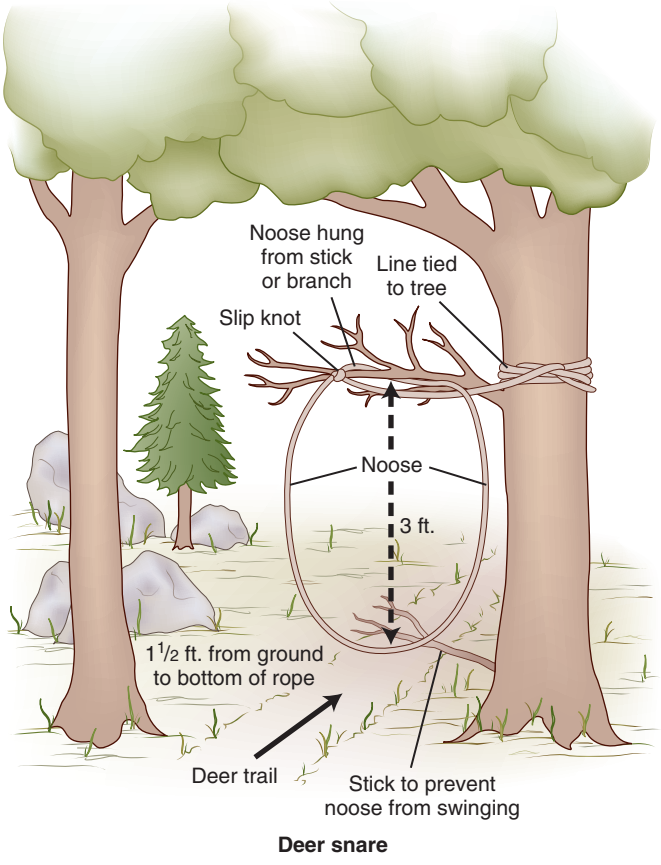


Figure 65-42. Parachute cord or similar material can be braided to make a snare that is strong enough to hold larger game.

Noose Stick

A noose made from wire or nylon monofilament can be attached to the end of a pole, then passed over a bird's head and pulled tight to capture it. This method of snaring birds is most useful when trying to catch any of the grouse family (ruffed grouse, sage grouse, blue grouse, spruce grouse, and ptarmigan). These birds can often be approached very closely and will allow a noose to be passed over their head. The noose stick can also be used to catch lizards and other reptiles (Fig. 65-43).

The noose stick can be made in two ways. Construct a noose about 6 inches in diameter and attach the opposite end of the wire or monofilament directly to the end of a pole by wrapping the wire around one end of the pole several times and then around the main body of the snare to secure it (Fig. 65-44). The distance between the pole and the noose should be about 10 inches. To catch a bird, the noose is passed over its head, and when the pole is lifted, the bird strangles. The second method requires a longer piece of wire, or a combination of a wire noose and other line. The end of a piece of wire or line is wrapped around the end of the pole and secured (Fig. 65-45). The opposite end of the wire is then passed back under the wraps, and a noose is created. To use this method, the noose is passed over a bird's or lizard's head, or even around a fish, and then closed by pulling on the end of the wire.

Figure 65-43. A wire noose attached to the end of a long stick is an effective device for catching birds, lizards, fish, and snakes.

Figure 65-44. Noose stick with slip loop.

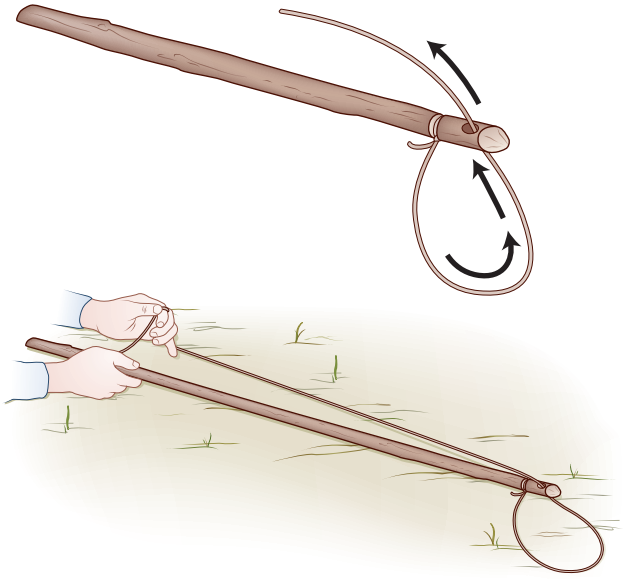
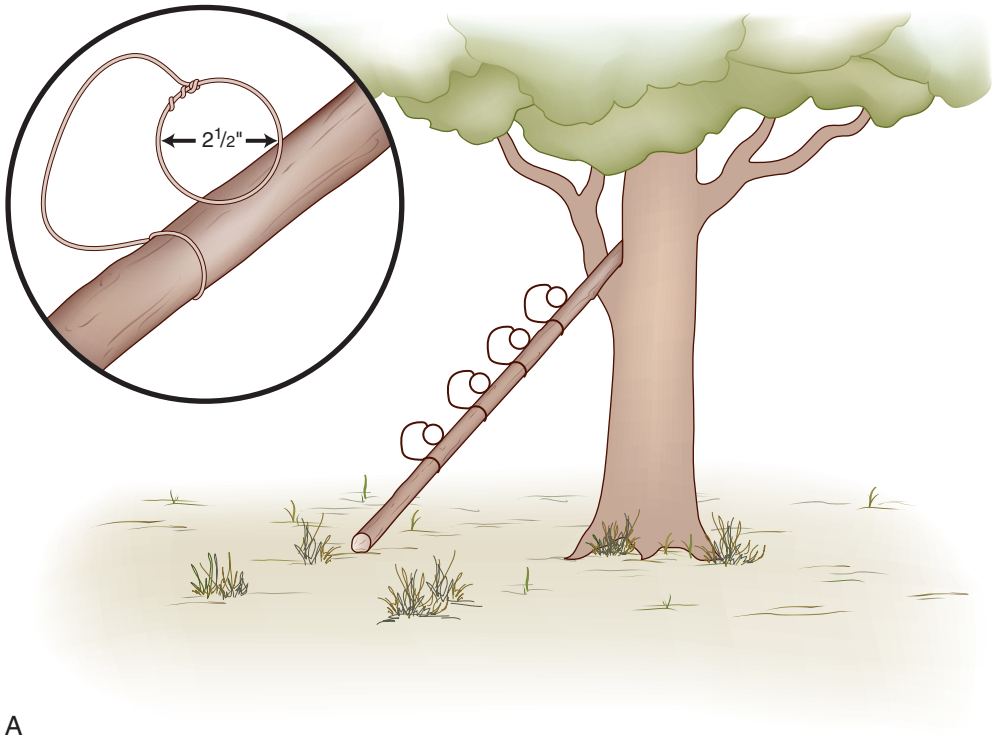


Figure 65-45. An alternative method of assembling a slip loop.

Squirrel Pole

Using a single strand of brass or steel wire, make a small loop about $2^{1}/_{2}$ inches in diameter in one end of the wire and attach the opposite end to the lower side of a 6-foot-long pole (Fig. 65-46). Snares are situated every 8 inches along the length of the pole, with the lower edge of the loop positioned about 2 inches above the limb. The pole with the attached loops is then leaned against a tree or used to bridge a space between two trees where squirrel activity has been observed. Squirrels are inclined to take the line of least resistance and will run up, down, or across the pole. Once they become snared in a loop, they usually fall off and are strangled. To condition the squirrels to use the



A

B

Figure 65-46. **A** and **B,** Squirrel pole. (**B,** courtesy Peter Kummerfeldt.)

pole, it can be pre-positioned before attaching the snares for several days. Once the animals become accustomed to taking the shortcut, the snares can be attached. The wire snares should be long enough so that when snared, the squirrel hangs well below the pole and is unable to reach up and grasp the underside of the pole or to touch the ground with its hind legs when hanging. It is quite common to snare several squirrels at a time by using this method. They are inquisitive animals and will investigate another squirrel that has become snared and become trapped themselves in one of the remaining snares. A series of loop snares placed along a tree limb will work in the same fashion as the squirrel pole.

Catapult (Slingshot or Flip) Hunting

In its simplest form, a catapult, or "flip" as it is known in the southeastern United States, consists of a Y frame made from wood or metal with two pieces of surgical tubing tied to each end of the fork and a soft leather pouch attached to the other end of the surgical tubing pieces (Fig. 65-47). Although catapults are often called by the misnomer "sling-shot" in some regions of the United States, a catapult should not be confused with a sling, which consists of two long thongs attached to a leather pouch. Placing a golf-ball-sized stone in the pouch, the pouch and thongs are twirled overhead, and when one of the thongs is released, the stone is launched toward a target. With

Figure 65-47. Sim Lovejoy "flip" or catapult. (Courtesy Mr. Sim Lovejoy.)

Figure 65-48. Commercial "wrist rocket" style of catapult. (Courtesy Peter Kummerfeldt.)

a catapult, which is much easier to use and much more accurate than a sling, the projectile is launched by stretching and then releasing the surgical tubing, which propels the projectile toward the target.

A properly constructed catapult is one of the best weapons available with which to gather animal food. Catapults are relatively small devices; are powerful, accurate, and inexpensive to make or buy; and use projectiles that can be picked up from the ground. With practice, a person with a catapult is quite capable of killing any of the smaller animals and birds that he or she is likely to encounter.

Commercially available catapults come in many varieties, including the simple forked-stick type and hi-tech versions that are even equipped with laser sights (Fig. 65-48). Catapults that come with a folding handle are especially suitable for inclusion in a survival kit. Perhaps the best option available is to preassemble the surgical tubing and leather pouch or to buy commercially available catapult replacement parts and include those in an emergency kit. A forked stick is then cut on-site to assemble the catapult (Fig. 65-49).

The catapult user will quickly realize that should the surgical tubing snap when under tension, there is a possibility of facial or eye injuries occurring. The use of protective eye equipment is highly recommended. Inspect the condition of the tubing frequently when using the catapult, and replace it immediately if any signs of deterioration are visible.

Although it is possible to kill larger animals with a catapult, more suitable-sized targets include rabbits, hares, squirrels, and

Figure 65-49. Catapult parts can be placed in a survival kit and then assembled onsite. (Courtesy Mr. Sim Lovejoy.)

Figure 65-50. Firing a catapult. (Courtesy Peter Kummerfeldt.)

Figure 65-51. Commercial survival fishing kit. (Courtesy Peter Kummerfeldt.)

most birds. With animals of this size, clean, humane kills can be made out to ranges of about 25 meters (about 25 yards). For the inexperienced catapult hunter, closer is always better. Many animals, especially those that are not traditionally hunted, will allow a person to get close if the approach is quiet and slow. Rabbits, grouse, and pheasants in particular are inclined to stay put, giving the hunter an excellent opportunity to get within range before taking a shot. This is especially true in remote parts of the country where animals that are not commonly pursued allow much closer approaches than they might in an area where hunting is frequent.

For a right-handed person, the catapult is grasped in the left hand. A projectile is slipped into the pouch and held in place using the thumb and index finger of the right hand. When the target is within range, the pouch is held at the anchor point, which is usually the base of the ear, the bend of the jaw, or the corner of the mouth. The left arm is extended, stretching the rubber between the pouch and the frame (Fig. 65-50). Stretching the rubber produces the energy needed to launch the projectile toward the target and hopefully to kill the animal on impact. The target's head is centered in the V of the catapult's frame, and when it is properly aligned, the pouch is released. As the pouch is released, the left wrist is rotated forward, or "flipped," to avoid injury to the hand holding the catapult frame. If hit, the animal will usually be killed outright, but if only stunned, it can then be dispatched by striking it on top of the head with a stout stick.

Suitable projectiles, rocks about the size of an average marble, can be hard to come by in the field, but may be found along river banks or along the shorelines of lakes where the erosive action of water produces smooth round stones.

Freshwater Fishing

As a general rule, the survivor should focus efforts on catching smaller fish rather than the larger ones. Smaller fish are not as wary or conditioned and are more likely to be caught using improvised fishing equipment. They are also less likely to break improvised fishing equipment.

When the rules that limit how fish can be caught in a recreational setting are pre-empted by the priority of surviving, a person can take advantage of otherwise-illegal techniques such as gill netting, spearing, and snaring. These are methods that require uncomplicated, simple equipment. They are techniques that can be used with little practice and that offer the best chance of procuring food. In a survival situation, the simplest fishing techniques are usually best.

Although it is possible to improvise fishhooks from many materials, the results are often crude and too large for most freshwater fishing and are not strong enough to withstand the struggles of a fish trying to escape. It is possible to improvise fishing line by unraveling clothing, twisting fibers from various plants together, or using other available line. However, the effectiveness of improvised fishing line is far inferior to the quality and strength of monofilament line. For these reasons, commercial fishing equipment, including line, hooks, split shot, flies, lures, and a commercial fish spear point or a trident frog gig, should be part of every survival kit. Prepackaged, commercially assembled, emergency fishing kits are available (Fig. 65-51). These come in two varieties: suited for saltwater fishing or suited for use in lakes and rivers.

Pole fishing allows the user to reach out from the bank and place a baited hook exactly where it is most likely to attract a fish. For trout and similar-sized fish, cut a pole 6 feet long with a 1-inch-thick butt from willow, alder, or other similar shrub. The size of the targeted fish determines the thickness of the pole needed, whereas the springiness of the material selected reduces the chance of the line breaking as the fish struggles to free itself. The line, which should be one and a half times the rod length or slightly longer, is attached at two points: The first attachment point should be the midpoint of the pole, and the second should be at the tip. In this way, if the tip of the pole should break as a fish is being landed, the line is still attached at the lower point, and the fish, along with the hook and line, is not lost (Fig. 65-52). When tying on a hook or lure, use a clinch knot (Fig. 65-53).

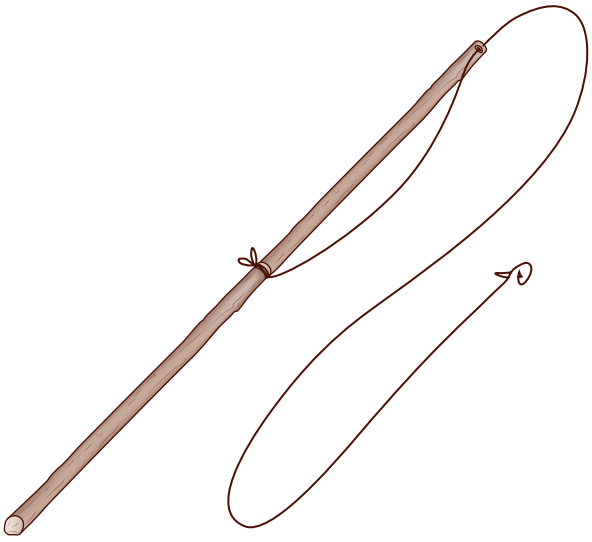


Figure 65-52. Improvised fishing pole.

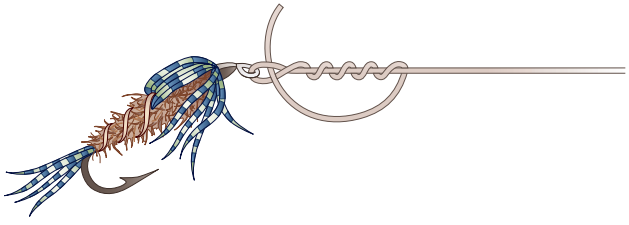


Figure 65-53. Improved clinch knot.

Figure 65-54. Seine net improvised from a shirt.

Bait can usually be found either in the water or in the surrounding area. Try to determine the types of natural foods that the fish are eating by examining the stomach contents of the first fish caught and, if possible, use these same foods for bait. Examine the underside of rocks lifted from the river for any insect life. Most of the insects observed will be very small, but some are large enough to be gathered and then impaled on a small hook. Aquatic insect life can also be gathered by seining the water with an improvised net, such as one made from a shirt (Fig. 65-54). Stand on the upstream side of the net, and disturb the streambed with your feet. Insects washed free from the gravel will be flushed into the waiting net. This method usually produces larger aquatic life that can be used for bait. Other insect life suitable for bait, such as grasshoppers, crickets, and worms, can usually be found along the lakeshore or river bank.

In moving water, it is usually best if the bait is cast upstream and allowed to drift with the current along the streambed. This simulates a free-floating insect or one that has fallen into the water and drowned. At the end of the drift, the bait is removed from the water and recast to a new drift line. This cast-and-retrieve process is repeated until a fish is caught. In still or very slow-moving water, a bobber made from a piece of wood or other buoyant material should be used with the bait suspended below it. The distance between the bobber and the bait is adjusted until a bite is detected. Because 98% of the feeding that fish do is below the surface, position the bait close to or right on the bottom of the river. A fish taking the baited hook will disturb the bobber, which is the primary indicator that there is a bite or hooked fish. The line is lifted quickly and the hooked fish removed from the water. To ensure that the fish is not lost, place an improvised net below it or use an improvised gaff to assist in the recovery of the fish. Sliding the fish up onto the bank rather than lifting it from the water will also reduce the chances of the line breaking and the fish escaping with the fishhook. In most situations, flies or bait fished below the surface will catch more fish than those presented on or closer to the water surface.

An improvised **dip net** is a very useful food-gathering tool. The net can be used to scoop up speared fish, to land those caught on a hook and line, or even to catch fish at a constricting point in a waterway. Many small fish can often be caught using a dip net improvised from a shirt. Two poles attached to the sides of a shirt can be used to scoop out minnows and other small fish (see Figure 65-54).

Hand fishing works well in smaller, shallower rivers where fish take shelter beneath undercut stream banks, under sunken logs, and around other river debris. When hand fishing, the person enters the water some distance above the spot where undercut banks are likely to shelter fish. Moving downriver, the person muddies the water and drives the fish downstream. Con-

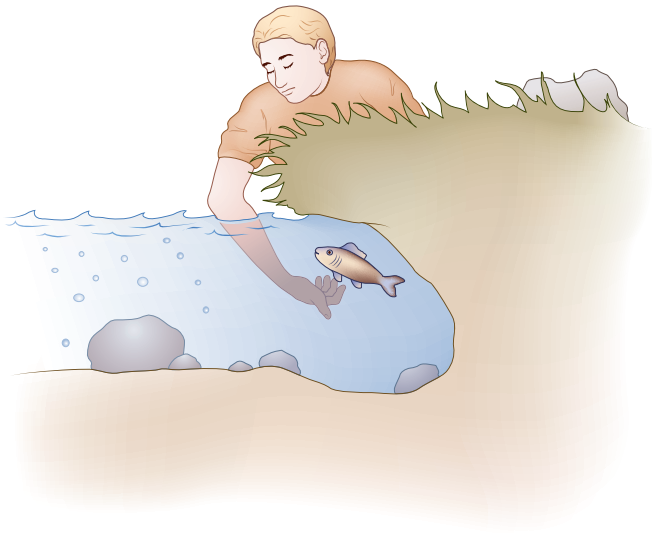


Figure 65-55. Hand fishing.

Figure 65-56. Fish or frog trident points, also called "gigs." (Courtesy Peter Kummerfeldt.)

centrating the fish in this way increases the odds of catching one. The individual then lies on the bank (Fig. 65-55) above a likely hideout and, moving slowly so as not to scare the fish, reaches into the water and carefully feels for any fish that may be present. Most fish, trout in particular, will tolerate being lightly handled. When the person touches the smooth skin of the fish, it is quickly pinned to the underside of the bank or to the bottom of the river, and then, when the body is firmly grasped, the fish is flipped out onto the bank. Trying to grip the fish by the gills or some other part of its anatomy underwater is usually not successful.

Other hand fishing methods include "noodling," whereby the person reaches into underwater holes in the riverbank or under sunken logs for catfish. Once a fish is detected, the person grasps its lower lip or gills and hauls it from the water. This process is not without risk because the resting areas favored by catfish are also those favored by turtles. This can result in a nasty bite. In the United States, catfish habitat is also water moccasin (cottonmouth snake) habitat.

Spear fishing is an effective technique available when fish are found in large schools or dense concentrations that make hitting a target much easier. Spear fishing is also a useful technique where fish can be seen, but the water is too deep for hand fishing, or where fish are too spooked to be approached closely. In addition to fish, spears can also be used to procure a variety of other animals, such as frogs, lizards, and snakes. Although spear points can be improvised, the success of spear fishing largely depends on the availability of a commercial spear point. A trident point is one of the most versatile food gathering devices available to the survivor and should be included in a survival kit (Fig. 65-56). The spear point is easily attached to one end of an 8-foot-long pole using a small nut and bolt (which usually come with the trident when purchased) inserted through the holes in the spear point and through the shaft of the spear.

Even though a commercial spear point may not be available, it may be possible to improvise one from available pieces of hardwood. Sharpen the end of the shaft with a knife and then harden it by turning the now pointed end in the flames of a fire. An improvised spear should be used to impale a fish, by pinning it to the river bottom and then lifting it out by hand. Although many fish will escape this method, some will die from their injuries and may float to the surface, where they can be recovered.

To use a fish spear effectively, hold the spear point just below the surface of the water rather than standing poised with the spear held over the shoulder while waiting for a fish to come by. Placing the spear point just below the water surface reduces the aiming problems caused by water refraction and greatly increases the odds of striking the fish.

The fisherman moves slowly from a downstream position toward a resting fish or waits patiently for a fish to come within range. When within reach, the fish is impaled with a quick strike and pinned to the river bottom. Rather than lifting the fish directly from the water using the spear point, thereby risking not only the loss of the fish but also possibly the spear point, reach down and grasp the fish with your free hand and lift it on the spear point onto the bank.

Hand line fishing greatly extends the reach of the fisherman. Many yards of line can be wrapped around a line holder, which is then unwrapped before the fisherman casts a baited hook into the water. Hand lining requires a substantial weight attached to the line to pull the baited hook out into deeper water, where it is allowed to remain until located and taken by a fish. When hand line fishing, the line is held in the fingertips awaiting the feel of a fish taking the bait, whereupon the line is withdrawn quickly and the fish hooked. With a fish on, the line is quickly retrieved hand-over-hand until the fish is landed. When casting, line control as the bait is thrown can become a problem, so tangles are common. This problem can be resolved by winding the line around the base of a soda pop can or similarly shaped smooth object. The base of the pop can is then pointed toward the open water, and the line is cast. The weight pulls the line

Figure 65-57. Pop-can hand line. (Courtesy Peter Kummerfeldt.)

Figure 65-58. Improvised gaff made from a large fishhook. (Courtesy Peter Kummerfeldt.)

off of the can, and the baited hook flies out free of tangles (Fig. 65-57). As the line is retrieved, it is once again wound onto the can until the next cast is made.

With **snaring,** as with snaring land animals, a wire loop attached to the end of a stout pole can be used to catch fish. Select wire that is strong enough to overcome the struggles of the snared fish (or, if limited in choice of wire, select smaller fish). The wire must also be supple enough to close quickly and yet stiff enough to retain the loop as it is passes through the water and over the fish's body. Attach one end of the wire to a 6-foot-long pole and create a loop in the other end large enough to pass over the head of the fish. The distance between the end of the pole and the loop should be about 12 inches. The fisherman then approaches a resting fish from directly behind (downstream) and slowly passes the noose over its head, moving the noose toward the fish's midsection. With some fish, it may work better to pass the noose from behind and over the fish's tail. When positioned properly, the pole is then lifted, tightening the noose, and the fish is removed from the water.

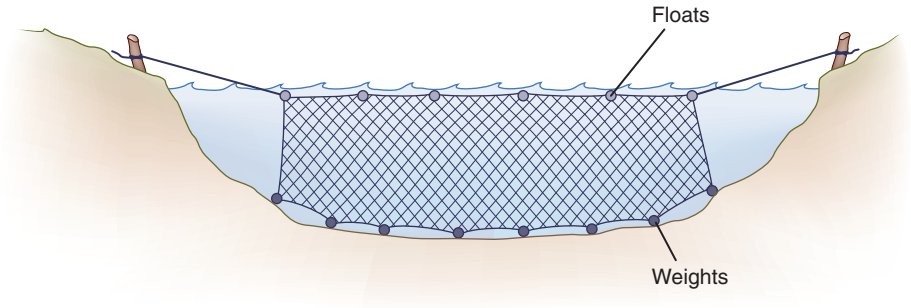
Gaffing involves a large fish hook lashed securely to a long pole, which can be used to snag a resting fish and lift it from the water. To prevent the loss of the hook should the pole break when landing larger fish, attach a length of line to the hook eye and tie it off to the stronger midpoint of the pole (Fig. 65-58). Gaffing is particularly effective when fish are confined and can be approached carefully from the bank or by wading. Approach the fish slowly from downstream and when within reach of the gaff, place the hook close to a "thick" part of the fish and strike upward quickly. Expect the fish to struggle vigorously and attempt to free itself from the hook. As quickly as possible, remove the fish from the water and strike its head above the eyes with a stout limb to kill it.

The methods discussed to this point require the active participation of a person. Sometimes, considerable amounts of energy can be expended using these techniques. These methods also expose a survivor to the existing weather conditions, contact with cold water, and, depending on the fishing methods used, risk for becoming hypothermic. From a survival perspective, better fishing techniques would reduce the amount of energy expended by the survivor, reduce exposure to inclement

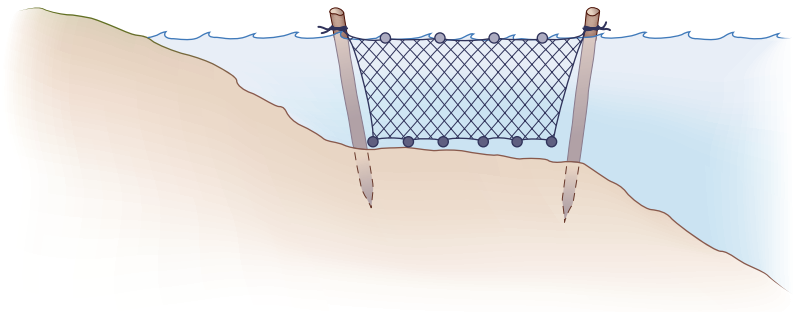
Figure 65-59. Commercial survival gill net. (Courtesy Peter Kummerfeldt.)

weather conditions, and limit contact with cold water while at the same time increasing the chances of catching a fish. The following are examples of passive fishing techniques that, once constructed, work for the survivor with little further expenditure of energy.

Given the time, materials, and ability, it is possible to improvise a **gill net.** However, it is much wiser to include a small (4 ft × 12 ft × $2^1/_2$ in) commercially available gill net in a survival kit (Fig. 65-59). The net can be used to trap fish and can also be erected on land to trap small animals and birds. Floats, made from pieces of wood or other buoyant material, need to be attached along the length of the upper edge of the net, and weights, made from stones, attached to the lower edge. Some experimentation will be necessary to find a balance where the floats remain on the surface while the stone weights fully extend the net into the depths of the water. It is better to use many smaller stones as weights than it is to use a few larger ones. The net is then placed in still or very slow moving water, anchored,



Block the whole of a smaller stream with a net.



Gill net set between the beach and the main drop-off in a lake.

Figure 65-60. Setting a gill net to catch fish in slow-moving or still water.

Fishing with nets

and then left to trap passing fish (Fig. 65-60). The net can also be placed in a naturally constricted location or in a narrow point in the waterway created by the survivor from logs and rocks. To expedite catching fish, a person would enter the water upstream of the net and move noisily downstream, scaring fish into the trap. Gill nets used in moving water must not be left in the river unattended. Debris floating downstream will collect in the net and soon destroy it. The net should be checked at least once a day and more often if the floats are seen bobbing or being drawn underwater, indicating trapped fish or river debris entangled in the net.

Set lines are usually a single hook attached to a length of line. One end is tied to an anchor onshore, and a baited hook is tied to the other end, which is cast into the water (Fig. 65-61). The line can also be tied to a branch or other similar anchor point overhanging the water. The branch serves as a "fishing rod" and plays the fish until it is retrieved by the fisherman. Multiple set lines placed along a riverbank or along a shore line provide the best opportunity to catch fish with the least amount of energy expended. Lines with multiple hooks attached along the length of the fishing line can also be assembled and used to catch fish. Commercial "trigger" devices, such as Speed Hooks (Fig. 65-62), greatly improve the fisherman's chances of hooking a fish. These devices are spring-loaded and automatically set the hook when a fish nibbles the bait. Set lines should be checked several times a day to rebait any empty hooks and to remove fish that have been hooked before other predators get them.

WILD PLANT FOODS

Plants are, and always have been, a valuable source of food and nutrients. They are readily available throughout most of the world and are often easily procured. The foraging of wild plants, usually considered weeds, has seen people through many a war, famine, and drought.[19,23] Studies demonstrate that wild plants are a good source of food in survival situations, allowing a survivor to perform hard and prolonged physical work during periods of restricted food intake.[23] They can increase daily food mass by five-fold.[26] Wild plants are certainly a critical component for wilderness survival and living off the land.

Wild plants are packed with a plethora of nutrients, particularly B vitamins, ascorbic acid (vitamin C), and antioxidants. Studies show that wild plants have significantly higher nutrient ratios than cultivated varieties.[11,25,28] It is believed that the longevity factor in the Mediterranean diet may indeed be the consistent and continued dietary integration of wild plants.[37] Sadly, of the 80,000 known edible plant species, only about 200 are cultivated regularly and only 3 (corn, wheat, and rice) function as staples in modern Western societies.[33]

Procurement of Wild Plant Foods

When one is harvesting wild plants, careful thought must be given to identification, procurement, preparation, toxicity, and plant abundance (Fig. 65-63). When considering plant

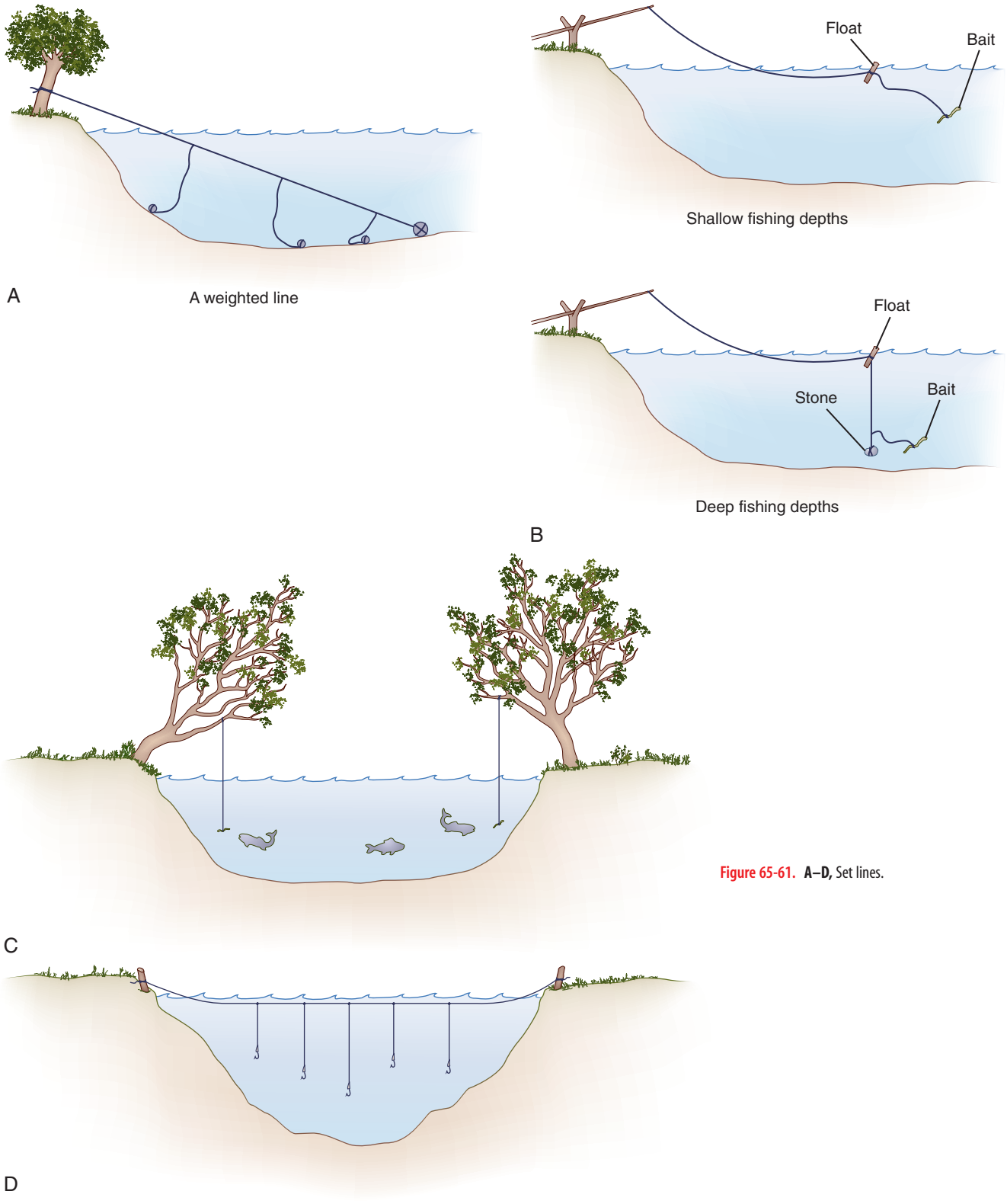
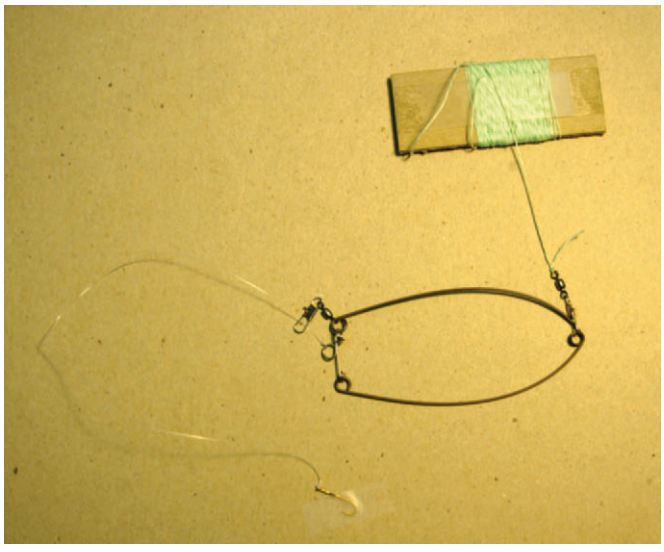
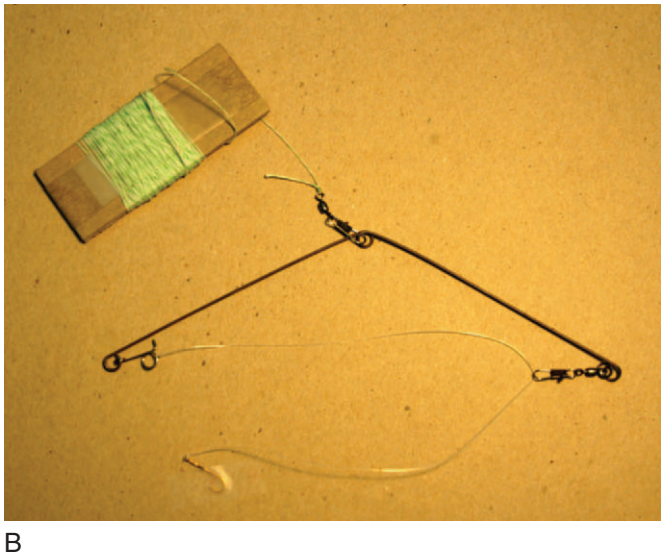


Figure 65-61. **A–D,** Set lines.



A



B

Figure 65-62. **A** and **B,** Commercial speed hooks. Set position and sprung position. (Courtesy Peter Kummerfeldt.)

abundance, it may once again be best to look at the practices of hunter-gatherers. Scientific analysis has shown that hunter-gatherers likely prioritized the collection of wild plants to provide the greatest ratio of energy capture to energy expenditure, in what has been called "optimal foraging."[6] Optimal foraging would clearly be advantageous to any wilderness survival situation and living off the land.

Before heading into the wilderness, it is wise to familiarize yourself with the types and abundance of edible wild plants that grow in the particular region in which you will be traveling. Because it would be impossible to list the tens of thousands of known edible plants throughout the world, this chapter concentrates only on the most common. Tables 65-2 through 65-6 list some of the most common edible wild plants that can be found in specific regions of the world and how these plants are prepared. Many of these plants are easy to identify; others will take practice. Take every opportunity to practice identifying edible plants in the wilderness and in the city. You'll be surprised at how omnipresent edible wild plants are. Because of the challenges associated with properly identifying mushrooms and other fungi, they have been purposely left out of the plant list. It is highly recommended that one avoid consuming any fungi in a wilderness survival situation.

Figure 65-63. Gathering blue camas bulbs for a meal. (Courtesy Peter Kummerfeldt.)

A clear understanding of plant identification and indications of toxicity is requisite for the consumption of any wild plant. Many plants throughout the world are poisonous (see Chapter 58). Tasting or swallowing even a small portion can result in severe cramping, diarrhea, rash, intestinal and metabolic disorders, and even death. When in doubt of the potential edibility of a wild plant, look for the common indicators of toxicity listed in Box 65-1.

Box 65-1. Seven Common Indicators of Plant Toxicity

Many poisonous plants exist throughout the world. Tasting or swallowing even a small portion can result in severe cramping, diarrhea, rash, intestinal and metabolic disorders, and even death. It is important to be aware of these seven common indicators of plant toxicity.

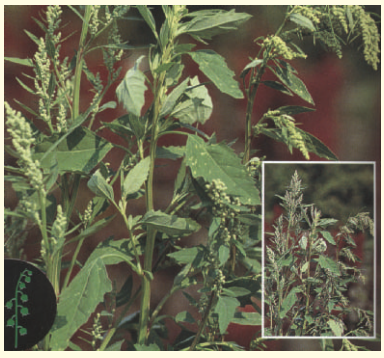
1. Milky or discolored sap
2. Beans, bulbs, or seeds inside pods
3. Spines, fine hairs, or thorns
4. Dill-, carrot-, parsnip-, or parsley-like foliage
5. Almond scent in woody parts and leaves (likely contains cyanide)
6. Grain heads with pink, purple, or black spurs
7. Three-leaved growth pattern

Text continued on p. 1529.

TABLE 65-2. Temperate Region Wild Plants

SCIENTIFIC NAME	COMMON NAME	PHOTO	EDIBLE PART(S)	SPECIAL PREPARATION
Acer saccharinum, A. saccharum	Maple	(Courtesy Denise Martinez.)	Sap, young leaves, inner bark	Boil sap to concentrate sugars. Young leaves can be eaten raw or cooked. Inner bark should be boiled. Can also be dried, ground, and added to flour to make bread.
Allium sp.	Wild onion, wild garlic, wild chive	(Courtesy Denise Martinez.)	Young leaves, bulbs	All can be eaten raw or cooked.
Amaranthus sp.	Amaranth/ pigweed		Seeds, young shoots, leaves	Seeds can be eaten raw, cooked, or roasted. Can also be ground into flour. Shoots and leaves can be eaten raw or cooked. Will concentrate toxic levels of nitrates if grown in contaminated soils under drought conditions.

TABLE 65-2. Temperate Region Wild Plants—cont'd

SCIENTIFIC NAME	COMMON NAME	PHOTO	EDIBLE PART(S)	SPECIAL PREPARATION
		(Photo from National Audubon Society: Field Guide to North American Wildflowers: Eastern Region, Revised Edition. New York, Alfred A. Knopf, 2001, Color plate 22.)		
Chenopodium sp.	Lamb's quarter, goosefoot, quinoa	 (Photo from National Audubon Society: Field Guide to North American Wildflowers: Eastern Region, Revised Edition. New York, Alfred A. Knopf, 2001, Color plate 21.)	Leaves, seeds	Leaves can be eaten raw or cooked. Seeds must be boiled to remove saponins.
Cichorium intybus	Chicory	(Photo from National Audubon Society: Field Guide to North American Wildflowers: Eastern Region, Revised Edition. New York, Alfred A. Knopf, 2001, Color plate 624.)	Roots, young leaves, flowers, flower buds	Leaves, flowers, and flower buds can be eaten raw or cooked. Roots taste best when cooked in several changes of water.
Cirsium vulgare	Thistle		Young shoots, stems, leaves, receptacle	All can be eaten raw or cooked.

Continued

TABLE 65-2. Temperate Region Wild Plants—cont'd

SCIENTIFIC NAME	COMMON NAME	PHOTO	EDIBLE PART(S)	SPECIAL PREPARATION
		(Photo from Peterson LA: A Field Guide to Edible Wild Plants of Eastern and Central North America. Peterson Field Guide Series No. 23. Boston, Houghton Mifflin, 1977, Color plate 10. Copyright © 1977 by Lee Peterson. Reprinted by permission of Houghton Mifflin Conpany. All rights reserved.)		
Hemerocallis sp.	Day lily	(Courtesy Denise Martinez.)	Roots, young shoots, flower buds, flowers	Young roots can be eaten raw. Older roots must be cooked. Young shoots, flower buds, and flowers can be eaten raw or cooked.
Malva neglecta	Mallow/ cheeseweed	(Courtesy Denise Martinez.)	Young shoots, leaves, flower buds, flowers, unripe fruit	All can be eaten raw or cooked.
Medicago sativa	Alfalfa	(Courtesy Denise Martinez.)	Leaves, flowering tops	All can be eaten raw or cooked.

TABLE 65-2. Temperate Region Wild Plants—cont'd

SCIENTIFIC NAME	COMMON NAME	PHOTO	EDIBLE PART(S)	SPECIAL PREPARATION
Pinus sp.	Pine	(Courtesy Denise Martinez.)	Young shoots, inner bark, male inflorescences, pine nuts, sap	All can be eaten raw or cooked. Inner bark best when gathered early in the year. Pine nuts best when roasted. Sizes differ depending on species. Sap can be used as cough drops.
Polygonum bistorta	Bistort	(Photo from Foster S, Hobbs C: A Field Guide to Western Medicinal Plants and Herbs. Peterson Field Guide Series. Boston, Houghton Mifflin, 2002, p 50.)	Roots, young shoots, leaves	Roots are best when cooked. Young shoots and leaves can be eaten raw or cooked.

Continued

TABLE 65-2. Temperate Region Wild Plants—cont'd

SCIENTIFIC NAME	COMMON NAME	PHOTO	EDIBLE PART(S)	SPECIAL PREPARATION
Portulaca oleracea	Purslane	(Courtesy Denise Martinez.)	Roots, leaves, stems	All can be eaten raw or cooked. Best when cooked.
Pteridium aquilinum	Bracken fern		Very young fronds	Cook in several changes of water. Fronds contain ptaquiloside, the high consumption of which has been linked to esophageal cancer.[18] Fronds also contain thiaminase, which destroys thiamine (vitamin B_1) in the body.
Quercus sp.	Oak	(Courtesy Denise Martinez.)	Acorns	Some species have sweet acorns, which can be roasted or eaten raw. Most other species have bitter acorns, which are best when chopped and boiled in several changes of water.
Rosa sp.	Wild rose	(Courtesy Denise Martinez.)	Petals, rosehips	All can be eaten raw or cooked.

TABLE 65-2. Temperate Region Wild Plants—cont'd

SCIENTIFIC NAME	COMMON NAME	PHOTO	EDIBLE PART(S)	SPECIAL PREPARATION
Rubus sp.	Blackberry, raspberry, dewberry	(Photo from Letcher Lyle K: The Wild Berry Book: Romance, Recipes, and Remedies. Minocqua, Wisc, NorthWord Press, 1994, p 43. Photo © Larry West.)	Berries, leaves	Berries can be eaten raw or cooked. Leaves can be used for tea, though excessive consumption can induce labor.
Rumex crispus	Sorrel/curly dock	(Courtesy Denise Martinez.)	Leaves, seeds	Boil leaves in several changes of water. Seeds must be removed from their astringent hulls.

Continued

TABLE 65-2. Temperate Region Wild Plants—cont'd

SCIENTIFIC NAME	COMMON NAME	PHOTO	EDIBLE PART(S)	SPECIAL PREPARATION
Sagittaria latifolia	Arrowhead/ Wapato	(Courtesy Denise Martinez.)	Tubers at end of rhizomes	All can be eaten raw or cooked.
Taraxacum officinale	Dandelion	(Courtesy Denise Martinez.)	Roots, leaves, flower buds, flowers	All can be eaten raw or cooked.
Trifolium repens	Clover	(Courtesy Denise Martinez.)	Leaves, roots, inflorescences, seed	All can be eaten raw or cooked. Seeds can also be used for sprouting.

TABLE 65-2. Temperate Region Wild Plants—cont'd

SCIENTIFIC NAME	COMMON NAME	PHOTO	EDIBLE PART(S)	SPECIAL PREPARATION
Typha latifolia	Cattail	(Courtesy Denise Martinez.)	Roots, base of leaves, young shoots, unripe inflorescences, pollen, seeds	All can be eaten raw or cooked. Remove green outer leaves to find white, tender base. Unripe inflorescences can be roasted like corn on the cob. Seeds can be obtained by removing down.
Urtica dioica	Nettle	(Courtesy Denise Martinez.)	Young shoots and leaves	All can be eaten raw or cooked. Protect hands and arms while harvesting to avoid contact dermatitis. Stinging is reduced when nettle is wet. Avoid eating excessive amounts of old leaves because they can lead to kidney lesions.
Vaccinium sp.	Blueberry, huckleberry, bilberry, cranberry	(Photo from Letcher Lyle K: The Wild Berry Book: Romance, Recipes, and Remedies. Minocqua, WI, NorthWord Press, 1994, p 58. Photo © Brett Baunton.)	Fruit	Can be eaten raw or cooked.

TABLE 65-3. Desert Region Wild Plants

SCIENTIFIC NAME	COMMON NAME	PHOTO	EDIBLE PART(S)	SPECIAL PREPARATION
Agave parryi	Agave	(Courtesy Denise Martinez.)	Trunk	Dig up trunk before flower stalk appears. Chop off leaves at base and cook for several days. Flower stalk, flower buds, and flowers are also edible. They taste best when cooked.
Cereus giganteus	Saguaro	(Photo from Huey GHH, Houk R: Wild Cactus. New York, Artisan, 1996, p 64.)	Fruit	Can be eaten raw or cooked. Fruit falls to ground when ripe. Use stick to dislodge fruit if still attached.
Echinocactus horizonthalonius	Barrel cactus/ visnaga	(Photo from Huey GHH, Houk R: Wild Cactus. New York, Artisan, 1996, p 23.)	Pulp (hydration and food), fruit	All can be eaten raw or cooked. To obtain fluid: chop off top, mash pulp inside stem, and then strain fluid through cloth.

TABLE 65-3. Desert Region Wild Plants—cont'd

SCIENTIFIC NAME	COMMON NAME	PHOTO	EDIBLE PART(S)	SPECIAL PREPARATION
Opuntia ficus-indica	Prickly pear/ cholla	(Courtesy Denise Martinez.)	Fruit, young joints	All can be eaten raw or cooked. Plant is best gathered with a three-pronged stick. Glochids (bristles) are best removed by rubbing in sand or scrubbing in running water.
Yucca baccata	Yucca	(Courtesy Denise Martinez.)	Flower stalk, flower buds, flowers, fruit	Flower stalk tastes best when cooked. The remainder can be eaten raw or cooked.

TABLE 65-4. Arctic Region Wild Plants

SCIENTIFIC NAME	COMMON NAME	PHOTO	EDIBLE PART(S)	SPECIAL PREPARATION
Arctostaphylos uvaursi	Bearberry/ kinnikinnick	(Courtesy Denise Martinez.)	Fruit, leaves	Fruit can be eaten raw or cooked. Leaves can be used to make tea.
Cetraria islandica	Iceland moss	(Photo from Department of the Army: The Illustrated Guide to Edible Wild Plants. Guilford, CT, Lyons Press, 2003, p 53.)	All parts	Boil in several changes of water.
Empetrum nigrum	Crowberry	(Photo from Letcher Lyle K: The Wild Berry Book: Romance, Recipes, and Remedies. Minocqua, WI, NorthWord Press, 1994, p 141. Photo © Joy Spurr.)	Fruit	Can be eaten raw or cooked.
Papaver nudicaule	Arctic poppy	(Photo from Department of the Army: The Illustrated Guide to Edible Wild Plants. Guilford, CT, Lyons Press, 2003, p 12.)	Leaves, petals, flower buds, seeds	Can be eaten raw or cooked.
Salix arctica	Arctic willow		Young shoots	Can be eaten raw or cooked.

TABLE 65-5. Tropical Region Plants

SCIENTIFIC NAME	COMMON NAME	PHOTO	EDIBLE PART(S)	SPECIAL PREPARATION
Bambusa sp.	Bamboo	(Photo from Department of the Army: The Illustrated Guide to Edible Wild Plants. Guilford, CT, Lyons Press, 2003, p 16.)	Young shoots	Can be eaten raw or cooked.
Colocasia esculenta	Taro	(Courtesy Denise Martinez.)	Root	Must be thoroughly cooked.

TABLE 65-5. Tropical Region Plants—cont'd

SCIENTIFIC NAME	COMMON NAME	PHOTO	EDIBLE PART(S)	SPECIAL PREPARATION
Maranta arundinacea	Arrowroot	(Photo from Department of the Army: The Illustrated Guide to Edible Wild Plants. Guilford, CT, Lyons Press, 2003, p 13.)	Root	Remove outer skin and cook thoroughly.
Psophocarpus tetragonolobus	Goa bean	(Photo from Department of the Army: The Illustrated Guide to Edible Wild Plants. Guilford, CT, Lyons Press, 2003, p 49.)	Young pods, mature seeds, root	Seeds should be parched and roasted. Other parts can be eaten raw or cooked.

TABLE 65-5. Tropical Region Plants—cont'd

SCIENTIFIC NAME	COMMON NAME	PHOTO	EDIBLE PART(S)	SPECIAL PREPARATION
Ziziphus jujuba	Jujube/Chinese date	(Photo from Department of the Army: The Illustrated Guide to Edible Wild Plants. Guilford, CT, Lyons Press, 2003, p 37.)	Fruit	Can be eaten raw or cooked. Tastes best when dried.

TABLE 65-6. Sea Plants

SCIENTIFIC NAME	COMMON NAME	PHOTO	EDIBLE PART(S)	SPECIAL PREPARATION
Alaria esculenta	Dabberlocks	(Photo from http://www.surialink.com.)	Leaves	Dry in sun or over fire. Crush and make into soup.

Continued

TABLE 65-6. Sea Plants—cont'd

SCIENTIFIC NAME	COMMON NAME	PHOTO	EDIBLE PART(S)	SPECIAL PREPARATION
Chondrus crispus	Irish moss/ carrageen moss	(Photo from http://www.sb-roscoff.fr.)	Leaves	Dry in sun or roast over fire. Crush and make into soup.
Laminaria japonica	Kelp/kombu		Leaves	Dry in sun or roast over fire. Crush and make into soup.
Palmaria palmata	Dulse	(Photo from http://omp.gso.uri.edu.)	Leaves	Dry in sun or roast over fire. Crush and make into soup.
Porphyra sp.	Nori/laver	(Photo from http://www.solpugid.com.)	Leaves	Dry in sun or roast over fire.

If you have come across an unknown plant that does not exhibit any of the potentially poisonous characteristics, you can then apply the Universal Edibility Test (Box 65-2). Before testing a wild plant for edibility, make sure there is enough of the plant around to make the testing worthwhile. Test all parts of the plant separately for edibility because some plants have both edible and inedible parts. Do not assume that a part of a plant that proved edible when cooked is edible when raw. Also, the same plant may produce different reactions in different people. In addition, bees that feed primarily on the nectar of plants that are toxic to humans can produce poisonous honey. Although this is extremely rare, it is worth noting if you come across a beehive in the wild.

Preparation of Wild Plant Foods

Some wild plant foods can be eaten raw or with minimal preparation, but most others will need to be boiled in several changes of water to render them safe and palatable. Unfortunately, boiling often results in a reduction of soluble vitamins and a plant's nutritive value. If you are unable to boil a wild plant, crush the edible plant part, put it in a sock, and securely place it in a running river for a day or two. This will help leach the astringent and bitter phytochemicals from the plant. You can do this in conjunction with boiling to help reduce preparation time.

COOKING METHODS

When possible, all wild animal foods should be thoroughly cooked before they are eaten to kill any parasites and to make the meat more palatable.

Roasting is probably the easiest way to cook a piece of meat but is the least desirable of methods because much of the meat's nutritional value is destroyed or drips away when it is cooked in this manner. Despite this, it may be the only method that can be used when no container is available. Skewering the meat on a hardwood stick and then holding it over the flames is an expedient method to quickly cook animals, fish, or insects (Fig. 65-64). Choose the stick carefully. Wood that is nonresinous is best because coniferous wood (pines and firs) may impart an unpleasant taste to the meat while it is being cooked. When using green wood, such as willow or alder, remove the bark before inserting it into the meat. By using wooden pegs, meat can also be pegged onto a slab of wood and placed near a fire, where the radiant heat will slowly cook the meat.

If a metal container is available, **boiling** is an excellent method to prepare food for consumption, especially if the broth that is created is also consumed. Alternatively, a bowl can be hollowed out of a piece of wood, which is then filled with water and the food to be cooked. Stones heated in a fire are then placed into the bowl with the food. The hot stones heat the water, and the food is cooked. As the stones cool, they are replaced with more hot ones taken from the fire. Do not use stones taken from a stream bank or other wet areas because they may explode when heated. Water contained in the rocks is converted to steam; the steam expands and explosively fractures the rock.

Baking is another effective way of cooking wild foods because the process is slow and much of the nutritive value is retained.

In *clay baking*, birds (with the feathers on) and fish (with the skin and scales on) can be packed in mud or clay and then placed in the coals of a fire to bake. When using this method to cook a bird, the clay must be massaged into the feathers and the entire animal covered with a layer of clay several inches thick. Cooking time depends on the thickness of the clay and the size of the animal being cooked. Sixty minutes is usually sufficient. After this time, the hard clay shell can be broken away, exposing the cooked flesh. The skin, scales, or feathers will come away with the clay, leaving only the cooked flesh beneath.

For an *earth oven,* a hole about 2 feet deep and 2 feet square (or larger, depending the quantity of food to be cooked) is dug, and a large hot fire is built in the hole. The fire is allowed to burn down until only coals remain and then covered with a layer of soil. The food to be cooked is wrapped in a layer of cloth or packed in vegetation and placed in the hole. The hole

Box 65-2. Universal Edibility Test

Before testing a plant for edibility, make sure there is enough of the plant around to make the testing worthwhile. Test all parts of the plant separately for edibility because some plants have both edible and inedible parts. Do not assume that a part of a plant that is edible when cooked is edible when raw. Also, remember that the same plant may produce different reactions in different people.

1. Test only one part of a potential food plant at a time.
2. Separate the plant into its basic components—leaves, stems, roots, buds, and flowers.
3. Smell the food for strong or acrid odors. Remember that smell alone does not indicate whether or not a plant is edible or inedible.
4. Do not eat for 8 hours before starting the test.
5. During the 8 hours you abstain from eating, test for contact poisoning by placing a piece of the plant part you are testing on the inside of your elbow or wrist. Usually 15 minutes is enough time to allow for a reaction. If there is a reaction, eliminate the plant part as a food option.
6. During the test period, take nothing by mouth but the plant part you are testing and purified water.
7. Select a small portion of a single part and prepare it the way you plan to eat it.
8. Before placing the prepared plant part in your mouth, touch a small portion to the outer surface of your lip to test for burning or itching.
9. If after 3 minutes there is no reaction on your lip, place the plant part on your tongue, holding it there for 15 minutes.
10. If there is no reaction, thoroughly chew a pinch and hold it in your mouth for 15 minutes. DO NOT SWALLOW.
11. If no burning, itching, numbing, stinging or other irritation occurs during the 15 minutes, swallow the food.
12. Wait 8 hours. If any ill effects occur during this period, induce vomiting and drink copious amounts of water.
13. If no ill effects occur, eat $^1/_4$ cup of the same plant part prepared the same way. Wait another 8 hours. If no ill effects occur, the plant part as prepared can be assumed to be safe to eat.

Figure 65-64. **A–D,** Roasting a fish on a stick. (Courtesy Peter Kummerfeldt.)

is then filled in with the remaining soil and the oven left for 2 to 3 hours. After the prescribed period of time has elapsed, the soil is removed and the package containing the food is carefully lifted out. Be careful when opening the oven because hot soil and steam can cause serious burns. The cooked bundles of meat and vegetation can be very fragile. Be watchful that the food is not contaminated with soil as it is taken out of the oven. Placing the wrapped food in the earth under a burning fire is a variation of this method.

Where the quantity of food is large, or an animal's carcass is to be cooked intact, a bigger hole and more heat are required. For a *rock oven,* the hole is lined with rocks, and a fire is built within it. When the fire subsides, a layer of soil is placed over the hot rocks, the wrapped food is inserted, and the hole is covered with soil. Allow at least 2 hours to elapse before opening the oven to determine whether the food is cooked.

The process described above can also be used in a seashore environment. For a *sand oven,* a hole is dug in the sand, lined with rocks, and heated. The food, fish, crustaceans, shellfish, and so forth are wrapped in seaweed, placed in the hole, and left for 2 to 3 hours before removal.

Broiling foods can be achieved by placing a thin layer of flat stones over hot coals and then laying the food to be broiled on top of the stones. Meat can also be broiled by placing it on a flat rock and propping it close to a fire. Radiant heat will cook the meat. It may be necessary to turn the meat over at least once to ensure that both sides are cooked.

BASIC FOOD PRESERVATION

Most foods cannot be stored for any length of time in the wilderness without deterioration in freshness, palatability, and nutritive value. The main causes of this deterioration are microbial growth, enzyme action, and insect damage.[10] Techniques such as drying and freezing can help reduce the causes of deterioration and prolong the time that a food can be saved and eaten.

Of these techniques, drying is probably the most feasible method used in wilderness survival and living off the land. To dry meats, thinly slice them and place them across a stick or on a plank. Place them in a warm area with low humidity. You can

also place them in direct sunlight. Turn meats regularly to ensure that the pieces dry throughout. Herbs (leafy plants) can be gathered and bound by their stems and hung upside down. Herbs are best dried away from direct sunlight. Fruits and vegetables can be cut into small pieces and dried like meats. A screen can be made of any thin fabric, like a light colored T-shirt, to reduce the possibility of pest infestation. The screen should be placed so it thoroughly protects, but does not touch, the drying food. All food should be dried in low-traffic areas free of dust.

If freezing food is a possibility, it is important to wrap the food well before freezing to avoid dehydration and cellular damage.[10] Ideally, the wrapping material should not crack or become brittle at low temperatures, nor should it absorb water, blood, or oil. If it can be spared, wrap the food with the inside of an animal hide. Foods to be frozen should be buried deep to avoid the effects of light, changes in ambient temperature, and animal predation.

CONCLUSION

In the final analysis, the survivor must weigh the energy expended in the process of procuring animals and plants for food against the energy obtained from the plants and animal life that are gathered. The amount of water that will be lost when traveling to look for water must be balanced against the chances of actually finding sources of water. It might be a better choice, albeit a difficult one, to limit activity levels, conserve energy, conserve water, and wait for rescuers to arrive. Living off the land is thus fundamentally a cost-versus-benefit situation.

Developing survival skills, to include the methods and techniques of procuring food and water, will become valuable in the unlikely event that a person ends up stranded in some remote location and unable to return to family and friends. When an emergency happens, the practice and preparation accomplished ahead of time will pay great dividends.

The references for this chapter can be found on the accompanying DVD-ROM.

Seafood Toxidromes

66

Michael J. Matteucci, Binh T. Ly, and Richard F. Clark

At least three quarters of the world's population lives within 10 miles of the coast. One of many reasons why populations congregate near the sea is the abundance of food beneath the ocean's surface. Seafood provides a significant percentage of the protein in the diets of many cultures. Presently, 200 to 240 million tons of fish are harvested each year, with 50% of the total coming from coastal regions. Americans consume 16.3 pounds of fish per person per year.[338] It is increasingly clear that the ocean is one of our last great food resources.

Throughout time, humans have recognized that toxic seafood is associated with seasons of the year, phases of the moon, water temperature, weather conditions, waterfowl mortality, and the color of the waves that wash onto shore, along with many other things. Unfortunately, none of these methods has proven entirely successful at predicting when seafood poisoning will occur.

Marine creatures whose consumption can lead to poisoning include dinoflagellates, coelenterates, mollusks, echinoderms, crustaceans, fishes, turtles, and mammals. Most marine biotoxins are naturally occurring poisons derived directly from marine organisms, including phytotoxins (plant poisons) or zootoxins (animal poisons). Ingestible toxins may be classified by specific toxin or by the donor organ of origin ingested by the victim. *Ichthyosarcotoxin* is a general term for poison derived from the fresh flesh (muscle, viscera, skin, or slime) of any fish. Geographic location, dietary and clinical histories, and appropriate index of suspicion figure prominently in the diagnosis and treatment.

Data on food-borne disease outbreaks in the United States demonstrate that seafood is the third most reported category according to vehicle of transmission, surpassed only by "unknown vehicles" ranking first and "multiple vehicles" ranking second.[75] Some 90% of the outbreaks of seafood-related illnesses and 75% of individual cases come from contaminated raw molluscan seafood (e.g., oysters, clams), histamine poisoning (scombroid), and ciguatoxin found in reef fish species.[312]

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MONITORING PHYTOTOXIN-PRODUCING MARINE ALGAE AND SEAFOOD POISONINGS

Despite the increasing risk of human intoxication from contaminated seafood, standards and methods of screening and law enforcement vary throughout the world.[451] According to the

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Despite the increasing risk of human intoxication from contaminated seafood, standards and methods of screening and law enforcement vary throughout the world.[451] According to the

U.S. Department of Agriculture, imports account for more than 55% of total U.S. seafood consumption. The largest sources of seafood imports are Canada, Asia, and Latin America. The U.S. Food and Drug Administration (FDA) has been criticized for inadequate inspection of all food imports.[312] In 1995, the FDA switched to a new program for seafood safety known as the Hazard Analysis and Critical Control Point (HACCP) system. This program became mandatory for the seafood industry on December 18, 1997.[142] HACCP focuses on the identification of sources and points of contamination; levels, transmission rate, and transport of microorganisms; and the possibility of exposure of the consumer to the contaminant. HACCP focuses on preventing hazards rather than relying on spot checks and random sampling of products. The most effective control strategies can then be implemented. For shellfish- and virus-associated diseases, the data suggest that harvesting from unapproved sources is associated with more than 30% of outbreaks.[267] Among imports, the biggest risks relate to histamines and scombroid poisoning, mainly from tuna and mahimahi that is imported from Argentina, Taiwan, and Ecuador. For foods traveling great distances, refrigeration is the most critical aspect of controlling illness.

The United States is the second largest importer of shrimp in the world. Shrimp aquaculture currently accounts for approximately 30% of the world's supply. Irradiation of seafood products is still being considered for approval by the FDA, although it is currently used in Asian and European markets, especially for shrimp.[12]

Molluscan poisoning is mainly a problem with domestic seafood. In 1991, California was the first state to require restaurants that serve or sell Gulf Coast oysters to warn prospective customers about possible deleterious effects from *Vibrio* contamination, particularly *Vibrio vulnificus*.[346] Other States have since adopted these warning regulations. Additionally, fishermen are now required to refrigerate oysters within 6 hours after harvesting from the Gulf of Mexico. Regulations require oyster lot tagging, labeling, and record retention to facilitate traceback investigations of outbreaks. The United States and Canada allow the sale of oysters if there are less than 10,000 colony-forming units per gram (CFU/g) of *Vibrio parahaemolyticus*. However, in outbreaks in the Pacific Northwest in 1997 and New York in 1998, oysters had less than 200 *V. parahaemolyticus* CFU/g of oyster meat, suggesting that human illness can occur at lower levels.[78]

Approximately a third of U.S. shellfish beds carry bans or limitations on harvesting because of high levels of fecal coliform bacteria. The fecal indicator system for shellfish-harvesting waters has been effective in protecting consumers against general types of bacteria in fecal contamination. However, several pathogenic bacteria are not predicted by the system. The efficacy of methods for virus recovery may range from 2% to 47%.[469] The most promising of the new detection methods are based on molecular techniques. DNA hybridization and the polymerase chain reaction (PCR) have the advantages of specificity for particular pathogens, sensitivity, and speed (most assays are completed within a few hours). PCR has been used in shellfish to detect *Salmonella, Vibrio* species, and viruses, including hepatitis A virus and Norwalk virus. High-performance liquid chromatography (HPLC) has also been used to detect and quantify many shellfish toxins.[20,25,100,254,332,373] Phytotoxin-producing marine algae are responsible for the syndromes of paralytic, neurotoxic, and diarrhetic shellfish poisoning. The closure of fisheries (product harvest areas) depends on the density of algae. In some cases the decision to close a fishery is based on the toxicity level in shellfish; in others, algae in the water and toxin in shellfish must both be found. In Florida, more than 5000 cells/L of *Ptychodiscus brevis* must be detected before fisheries are closed. The quarantine level of saxitoxin (a neurotoxin found in marine dinoflagellates) varies between countries and ranges from 40 to 80 mg of toxin per 100 g of seafood, as determined through mouse bioassay.[23] The higher number is used in the United States, as monitored by the Interstate Shellfish Sanitation Conference and the FDA.

The maximal acceptable concentration of diarrhetic shellfish toxin, okadaic acid, also varies between countries because of the lack of precise analytic methods for quantification. Countries with established regulations apply 4 to 5 mouse units or 20- to 25-μg equivalents of okadaic acid as an acceptance limit. In the United Kingdom, the Ministry of Agriculture, Fisheries, and Food (MAFF) shellfish surveillance program tests harvested shellfish weekly from April to October and sporadically during the winter for the presence of toxins.[377] The United States, Canada, and Portugal monitor for domoic acid (the cause of amnesic shellfish poisoning) and use 2 mg/100 g of seafood as the threshold. Ciguatoxins are monitored infrequently because of difficulties associated with the assay. In French Polynesia, ciguatoxin at 0.06 ng/g of seafood as determined by mosquito bioassay is considered toxic; in the United States (Florida, Hawaii), detection of the toxin at any level by immunoassay renders the fish unmarketable. Two primary features render toxin surveillance difficult: the performance problems of the assays and the impracticality of surveying every fish.

ICHTHYOSARCOTOXISM

The term *ichthyosarcotoxism* describes a variety of conditions arising as the result of poisoning by fish flesh. Many toxins are generally not destroyed by heat or gastric acid. Various toxins are found in the musculature, viscera, blood, skin, or mucous secretions of the fish. Further classification is based on the specific organ system poisoned—for example, ichthyocrinotoxins (glandular secretions), ichthyohemotoxins (blood), ichthyohepatotoxins (liver), ichthyootoxins (gonads), ichthyoallyeinotoxins (hallucinatory), and gempylotoxins (purgative).

Ichthyocrinotoxication

Ichthyocrinotoxic fish poisoning is induced by the ingestion of glandular secretions not associated with a specific venom apparatus; this usually involves skin secretions, poisonous foams, or slimes. Examples of these toxic fish are certain filefish, puffer fish, porcupinefish, trunkfish, boxfish, cowfish, lampreys, moray eels, and toadfish (Box 66-1). Cyclostome poisoning results from ingestion of the slime and flesh of certain lampreys and hagfishes. Pahutoxin and homopahutoxin have been isolated from the secretion of the Japanese boxfish *Ostracion immaculatus*.[150]

Ichthyotoxic skin secretions may cause a bitter taste.[167] Ingestion of ichthyocrinotoxins causes gastrointestinal symptoms within a few hours of ingestion, characterized by nausea, vomiting, dysenteric diarrhea, tenesmus, abdominal pain, and weakness. Most victims recover within 24 hours; however, some individuals have symptoms for up to 3 days. Therapy is supportive and based on symptoms. Additionally, some slime, such

Box 66-1. Representative Ichthyocrinotoxic Fish Hazardous to Humans

Phylum Chordata
- Class Agnatha
  - Order Myxiniformes: hagfishes, lampreys
    - Family Myxinidae
      - *Myxine glutinosa:* Atlantic hagfish
      - *Petromyzon marinus:* sea lamprey, large nine-eyes
- Class Osteichthyes
  - Order Anguilliformes: eels
    - Family Muraenidae
      - *Muraena helena:* moray eel
  - Order Perciformes: perchlike fishes
    - Family Serranidae
      - *Grammistes sexlineatus:* golden striped bass
      - *Rypticus saponaceus:* soapfish
  - Order Tetrodontiformes: triggerfishes, puffers, trunkfishes
    - Family Canthigasteridae
      - *Canthigaster jactator:* sharp-nosed puffer
    - Family Diodontidae
      - *Diodon hystrix:* porcupinefish
    - Family Ostraciontidae
      - *Lactoria diaphana:* trunkfish
      - *Lactoria fornasini:* trunkfish, boxfish
    - Family Tetraodontidae
      - *Arothron hispidus:* puffer, toadfish, blowfish, rabbitfish
      - *Fugu xanthopterus:* puffer
    - Order Batrachoidiformes: toadfishes
      - Family Batrachoididae
        - *Opsanus tau:* oyster toadfish
        - *Thalassophyrne maculosa:* toadfish

Box 66-2. Representative Poisonous Sharks (Elasmobranchs) Hazardous to Humans

Phylum Chordata
- Class Chondrichthyes
  - Order Squaliformes: sharks
    - Family Carcharhinidae
      - *Carcharhinus melanopterus:* blacktip reef shark
      - *Carcharhinus menisorrah:* gray reef shark
      - *Galeocerdo cuvieri:* tiger shark
      - *Prionace glauca:* blue shark
    - Family Dalatiidae
      - *Somniosus microcephalus:* Greenland shark, sleeper shark, nurse shark
    - Family Hexanchidae
      - *Hexanchus griseus:* cow shark, gray shark, mud shark
    - Family Isuridae
      - *Carcharodon carcharias:* white shark
    - Family Scyliorhinidae
      - *Scyliorhinus caniculus:* dogfish, lesser-spotted cat shark
    - Family Sphyrnidae
      - *Sphyrna diplana:* hammerhead shark
    - Family Squatinidae
      - *Squatina dumeril:* monkfish, angel shark
    - Family Triakidae
      - *Triaenodon obesus:* white-tip houndshark

as "grammistin" from the soapfish (*Rypticus saponaceus* of the family Grammistidae), can cause a contact irritant dermatitis.[193] This dermatitis is managed with cool compresses of aluminum sulfate and calcium acetate (Domeboro). All suspect fish should be washed carefully with water or brine solution and skinned before being eaten.

Ichthyohemotoxication

Ichthyohemotoxic fish are perfused with "poisonous blood," the toxicity of which is usually inactivated by heat and gastric juice. Examples are various eels, such as morays, anguilliforms, and congers. The syndrome is predominantly gastrointestinal and should be treated according to symptoms. Hematologic complications are rare. The risk of intoxication is increased by ingestion of raw or undercooked fish.

Ichthyohepatotoxication

Ichthyohepatotoxic fish carry the toxin predominantly in the liver. The remainder of the fish may be nontoxic. Fish that are always toxic fall into two basic groups: (1) Japanese perchlike fish (e.g., mackerel, seabass, porgy, sandfish), and (2) tropical sharks (e.g., requiem fish, sleeperfish, cowfish, great white shark, catfish, hammerhead, angelfish, Greenland fish, dogfish).[328] In addition, some skates and rays, whose phylogeny is similar to that of sharks, harbor ichthyohepatotoxins.

Ingestion of the Japanese perchlike fish group causes onset of symptoms within the first hour, with maximal intensity over the ensuing 6 hours.[402] Symptoms include nausea, vomiting, headache, flushing, rash, fever, and tachycardia. No fatalities have been reported. Delayed (24 to 48 hours) necrodermolysis is rare.

Ingestion of tropical shark liver (and occasionally of the musculature), such as that of the Greenland shark *(Somniosus microcephalus),* results in "elasmobranch poisoning" (Box 66-2).[30] Symptoms are noted within 30 minutes of ingestion and include nausea, vomiting, diarrhea, abdominal pain, malaise, diaphoresis, headache, stomatitis, esophagitis, muscle cramps, arthralgias, paresthesias, hiccups, trismus, hyporeflexia, ataxia, incontinence, blurred vision, blepharospasm, delirium, respiratory distress, coma, and death. Recovery varies from several days to weeks. If only the flesh is eaten, the symptoms are mild and gastroenteric, with spontaneous resolution.

In 1993, 200 people in Madagascar were poisoned after ingesting a single shark identified as *Carcharhinus leucas*. They all experienced symptoms, and 30% died. Two liposoluble toxins were isolated from the shark liver and named carchatoxin-A and carchatoxin-B.[49] Trimethylamine oxide, found in shark liver and flesh, has also been implicated in shark poisoning.[15] A similar syndrome has occurred in sled dogs that ingest large quantities of shark flesh.

Therapy is supportive and based on symptoms. If the victim is treated within 60 minutes of ingestion of shark liver or other viscera, gastrointestinal decontamination with activated charcoal (50 to 100 g) may be of value. Fish liver or any shark viscera should not be eaten. However, drying the flesh properly may minimize the toxicity.

Ichthyootoxication

Ichthyootoxic fish possess toxic gonads that may vary in toxicity with the reproductive cycle. The musculature is generally nontoxic. Examples are the sturgeon, alligator gar, salmon, pike, minnow, carp, catfish, killifish, perch, and sculpin. Sea urchins may be toxic during the reproductive period.[30] This toxicity is exemplified by *Paracentrotus lividus* (Europe), *Tripneustes ventricosus* (West Africa), and *Diadema antillarum* (West Indies). Heat does not inactivate the toxin.

Symptoms begin within an hour of ingestion and include nausea, vomiting, diarrhea, headache, dizziness, fever, thirst, xerostomia, bitter taste, tachycardia, seizures, paralysis, hypotension, and occasionally death. Treatment is supportive and based on symptoms. The roe of any fish should not be eaten during the reproductive season.

Ichthyoallyeinotoxication

Ichthyoallyeinotoxic fish induce hallucinatory fish poisoning. These fish are predominantly reef fish of the tropical Pacific and Indian reefs, and they carry these heat-stable toxins mainly in the head, brain, and spinal cord, and in lesser amounts in the musculature. Typical species include surgeonfish, chub, mullet, unicornfish, goatfish, sergeant major, grouper, rabbitfish, rock cod, drumfish, rudderfish, and damselfish. Hallucinatory mullet poisoning has been described as a seasonal condition that occurs only during the summer months in restricted areas on the Hawaiian islands of Kauai and Molokai.[195] Symptoms can develop within 5 to 90 minutes of ingestion and include dizziness, circumoral paresthesias, diaphoresis, weakness, incoordination, auditory and visual hallucinations, nightmares, depression, dyspnea, bronchospasm, brief paralysis, and pharyngitis.[30] No fatalities have been reported. Various toxins including indoles akin to lysergic acid diethylamide (LSD) have been implicated, the sources being in algae and plankton eaten by the fish.[394] Heating the fish does not appear to lessen the severity of the poisoning.

Therapy for ichthyoallyeinotoxic fish poisoning is supportive and based on symptoms. Haloperidol or benzodiazepines may be titrated if the victim is agitated, psychotic, or violent. The victim should be observed until normal mental status is regained. The head, brain, or spinal cord of any tropical fish should not be eaten.

Gempylotoxication

Gempylotoxic fishes are the pelagic mackerels that produce an oil with a pronounced purgative effect. The "toxin" is contained in both musculature and bones. No particular characteristic distinguishes a gempylotoxic fish from a nontoxic fish of the same species. The castor oil fish *(Ruvettus pretiosus)* is named for its purgative properties.

The victim suffers from abdominal cramping, bloating, mild nausea, and diarrhea, usually within 30 to 60 minutes of ingestion. The disorder is self-limited and resolves in 12 to 18 hours. Diarrhea often occurs without concomitant systemic effects. Fever, bloody or foul-smelling stools, or protracted vomiting suggest infectious gastroenteritis. No specific antidote is available. If the victim cannot tolerate oral fluids because of nausea or severe abdominal cramping, administration of intravenous fluid and antiemetics may be indicated. Antimotility agents are not recommended unless the diarrhea is debilitating because inhibition of peristalsis prolongs the transit time of the toxin through the gut and may increase the duration of the disorder.

SPECIFIC FISH-RELATED TOXIC SYNDROMES

Three specific toxic syndromes related to fish consumption are scombroid, tetrodotoxin (puffer fish) poisoning (both described in Table 66-1), and grass carp gallbladder poisoning.

Scombroid

Scombroid, the most commonly reported seafood poisoning in the United States, occurs when certain species of fish are improperly handled and stored after being caught. These fish are most often in the family Scombridae and include albacore, bluefin and yellowfin tuna, mackerel, saury, needlefish, wahoo, skipjack, and bonito. Non-Scombridae fish that produce scombroid include mahimahi (dolphin), kahawai, sardine, black marlin, pilchard, anchovy, herring, amberjack (yellowtail or kahala), and the Australian ocean salmon *Arripis trutaceus*.[285,384,399,428] In Hawaii, the most commonly implicated fish is the dolphin *Coryphaena hippurus*. In the northeastern United States, bluefish *(Pomatomus saltratix)* has frequently been linked to scombrotoxism.[336] North Carolina has reported clusters of cases after ingestion of ground tuna burgers.[35] Scombroid poisoning accounts for 5% of food-related outbreaks reported to the Centers for Disease Control and Prevention (CDC) in Atlanta.[430] Because greater numbers of previously considered nonscombroid fish are now recognized as "scombrotoxic," Prescott[336] has suggested that the syndrome be more appropriately called *pseudoallergic fish poisoning*.

Pathophysiology. During conditions of inadequate preservation or refrigeration (optimal temperatures for bacterial growth are 37° to 43°C [98.6° to 109.4°F]), the musculature of dark-fleshed or red-muscled fish undergoes bacterial decomposition.[36,328] The normal piscine surface bacteria *Proteus morganii, Klebsiella pneumoniae, Aerobacter aerogenes, Escherichia coli, Alcaligenes metalcaligenes,* and others have been implicated in the putrefactive process, which includes the decarboxylation of the amino acid L-histidine to histamine and saurine (a phosphate salt of histamine).[428] The term *saurine* originated because of the association of scombrotoxism with saury, a Japanese dried fish delicacy.[203] Because of this process, "scombrotoxin" was initially thought to be histamine, which is commonly found in large amounts in the flesh of the fish usually implicated. Evidence initially suggesting that histamine may be the causative toxin of scombroid fish poisoning was presented in an investigation of a small outbreak.[299] The urinary excretion of histamine and its metabolite, *N*-methylhistamine, was measured in three persons in this series who had scombrotoxism after ingestion of marlin. There was no increase in the principal metabolite of prostaglandin D_2 (a mast cell secretory product considered to indicate release of histamine from mast cells), supporting the hypothesis that the excess histamine was from the fish rather than endogenously produced in the victims. Histamine levels greater than 20 to 50 mg/100 g are frequently noted in scombrotoxic fish, and it is not unusual to record levels in excess of 400 mg/100 g.[384] However, it is possible that some other compound may be responsible for scombroid symptoms, because the syndrome cannot be reproduced solely by the administration of equal or even massive doses of histamine by the oral route. Histamine is rapidly inactivated by enzymes in the gastrointestinal tract and on first pass through the liver, with

TABLE 66-1. Seafood Toxidromes

TOXIDROME	SEAFOODS	REGIONS	CAUSATIVE ORGANISMS	TOXIN PRODUCED	MECHANISMS OF ACTION	CLINICAL MANIFESTATIONS	SYMPTOM ONSET AFTER INGESTION	DURATION OF ILLNESS	TREATMENT
Fish-Related Toxic Syndromes									
Scromboid	Albacore, tuna, wahoo, mackerel, skipjack, bonito, mahimahi	Worldwide	Presumably, bacteria within the fish transform histidine to histamine	Histamine, saurine	Histamine response	Diffuse erythema, flushing, nausea, vomiting, pruritus, headache, urticaria, bronchospasm	Rapid, minutes	Resolves 8–12 hr	Histamine-1 and -2 blockers, antiemetics
Tetrodotoxin	Puffer fish (fugu), porcupinefish, sunfish	Tropical and subtropical	? *Pseudomonas* species	Tetrodotoxin	Na^+ channel blocker, blocks axonal transmission	Paresthesias of lip and tongue, hypersalivation, weakness, ataxia, tremor, dysphagia, seizure, bronchospasm, hypotension, nausea, vomiting, diarrhea, death	10 min to 4 hr	Hours to days	Supportive, aggressive airway management, IVF, inotropic agents, ? anticholinesterase
Algae Bloom–Related Toxic Syndromes									
Ciguatera	Tropical and semitropical reef fish such as barracuda, grouper, snapper, jack	Worldwide, most common in Indian Ocean, South Pacific, Caribbean	*Gambierdiscus toxicus* and other species	Ciguatoxin, maitotoxin, GT1-4, palytoxin	Na^+ channel blocker	Gastroenteritis followed by neurologic symptoms: dysesthesias, hot/cold reversal, weakness, respiratory paralysis	2–6 hr	Days to months	Supportive, ? IV mannitol
Clupeotoxin	Herring, sardines, anchovies, tarpons, bonefish	Caribbean, Indo-Pacific, Africa	*Ostreopis siamensis*	Palytoxin	Inhibits Na^+,K^+-ATPase	Metallic taste, nausea, vomiting, diarrhea, paresthesias, hypotension, death	Rapid onset, 30–60 min	Days	Supportive, ? early gastric emptying
PSP	Shellfish	NE and NW coast of U.S., Philippines, Alaska, North Sea	*Protogonyaulax, Alexandrium catarella, Pyrodinium, Saxidomus, Gonyaulax*	Saxitoxin, neosaxitoxins, gonyautoxins	Na^+ channel blocker, may suppress AV nodal conduction	Paresthesias of face and extremities, numbness, dysphonia, dysphagia, ataxia, weakness, paralysis, death from respiratory failure	30–60 min	Weeks	Supportive, charcoal, ventilatory support
NSP	Shellfish	Gulf of Mexico, Florida, Texas, North Carolina, New Zealand	*Ptychodiscus brevis*	Brevetoxins	Modulate Na^+ channel	Circumoral paresthesias, ataxia, GI symptoms; if aerosolized, may cause conjunctivitis, bronchospasm	Minutes to hours	Several hours to a few days	Supportive
DSP	Shellfish	Japan, Spain, Netherlands, Chile	*Dinophysis, Prorocentrum*	Okadaic acid and others	Phosphatase A_1 and A_2 inhibitor	Acute gastroenteritis	Rapid, 30 min to 2 hr	2–3 days	Supportive
ASP	Shellfish	Canada, Japan, NE and NW U.S.	*Nitzschia pungens, Pseudonitzschia australis*	Domoic acid	Glutamate antagonist	Gastroenteritis, seizures, coma, anterograde memory disorder	1–24 hr	24 hr to 12 wk	Supportive, benzodiazepines for seizures
PEAS	Estuarine fish	Coastal waterways in eastern USA and Gulf Coast	*Pfiesteria piscicida*	Unidentified	Unknown	Headache, skin lesions, eye irritation, respiratory irritation, learning and memory deficits, cognitive impairment	Within 2 wk of exposure	Improve within 3–6 mo	No treatment, cholestyramine for persistent symptoms
Haff disease	Buffalo fish	U.S., Russia, Sweden	? Blue-green algae	Unknown	Unknown	Severe muscle pain, rhabdomyolysis, weakness, tachycardia, hypotension	6–12 hr	Days	Supportive, IVF, ? diuretics

ASP, amnestic shellfish poisoning; ATPase, adenosine triphosphatase; AV, atrioventricular; DSP, diarrhetic shellfish poisoning; GI, gastrointestinal; IVF, intravenous fluid; NE, northeast; NSP, neurotoxic shellfish poisoning; NW, northwest, PEAS, possible estuary associated syndrome; PSP, paralytic shellfish poisoning.

very little reaching systemic circulation. Other compounds, such as cadaverine or putrescine, may be present in the decomposed fish flesh and may either facilitate the absorption or inhibit the gastrointestinal or hepatic degradation of histamine.[362,428] Whatever the causative toxin is, it is heat stable and not destroyed by cooking. Affected fish typically have a sharply metallic or peppery taste but may be normal in appearance and color. Not all persons who eat a scombrotoxin- or histamine-contaminated fish become ill, possibly because of uneven distribution of decay within the fish.

Clinical Presentation. The effects of scombroid fish poisoning occur within minutes after consumption of the fish. The symptoms are similar to an allergic reaction (which it is not) and typically include headache, diffuse erythema, a sense of warmth without elevation in core temperature, nausea, vomiting, diarrhea, abdominal cramps, conjunctival injection, pruritus, dizziness, and a burning sensation in the mouth and oropharynx.[231,285] Flushing of the head, neck, and upper torso is characteristic. Severe effects, such as bronchospasm, generalized urticaria, hypotension, palpitations, and dysrhythmias, have been reported but are not frequent.[163,203] In most healthy victims the syndrome is self-limited, resolving within 6 to 12 hours. In patients with preexisting respiratory or cardiac disease, the effects of the poisoning can precipitate more severe illness.[51,285] Scombroid reactions may be markedly more severe in patients taking isoniazid (INH) because of this compound's blockade of gastrointestinal tract histaminase.[449] Death has never been reported after scombroid poisoning.

Treatment. Gastric decontamination for scombroid poisoning is not indicated because symptoms occur rapidly and vomiting can be a primary effect of the toxin. Symptoms can be lessened or controlled with the administration of histamine-1 (H_1) receptor antagonists, such as diphenhydramine or hydroxyzine, administered initially in doses of 25 to 50 mg orally or intravenously. The use of histamine-2 (H_2) receptor antagonists (e.g., cimetidine, famotidine) has also been shown to relieve most of the symptoms, and perhaps a combination of H_1 and H_2 receptor antagonists would be most effective.[47,177] Vomiting is usually controlled by an antihistamine but occasionally requires the addition of a specific antiemetic, such as prochlorperazine. The persistent headache of scombroid poisoning may respond to cimetidine or a similar drug if standard analgesics are not effective.[21] Intravenous fluids and inhaled bronchodilators should be used as needed. Vasopressors are rarely necessary because hypotension is usually mild and responds to intravenous fluid administration.

Prevention. Scombroid is a preventable illness and can be avoided if the offending fish species are promptly refrigerated below 15° C (59° F) or iced after catch and maintained until the fish is cooked or processed for storage. Recreational fishermen must pay particular attention to their coolers. No fish should be consumed if it has been handled improperly or has the smell of ammonia. Fresh fish generally has a sheen or oily rainbow appearance; "dull" packaged fish should be avoided. If an episode of scombroid poisoning is recognized, it is important to report it promptly to local public health authorities to prevent additional exposures, particularly if the food was served in a public eating establishment.[204]

Tetrodotoxin Poisoning

Tetrodotoxin (TTX) is a potent neurotoxin found in a variety of creatures and has been isolated from animals of four different phyla, including puffer fish, the California newt, the blue-ringed octopus, poison dart frogs, the ivory shell, and the trumpet shell. The puffer fish, also known as the blowfish or globefish, is one of the better-recognized species that contains TTX. These fish can be found in both fresh and salt water.[182] In humans, the most common exposure to TTX is through the ingestion of fugu, a special preparation of puffer fish. Human TTX poisonings have also occurred after consumption of gastropod mollusks.[485] Envenomation from the blue-ringed octopus is rare.[141]

Puffer fish poisoning has been recognized for millennia. Ancient Asian literature documents the dangers of eating puffer fish.[182] There are references to puffer fish in hieroglyphics of the ancient Egyptian dynasty of 2700 BC. Scholars suggest this fish was known to be poisonous during Egyptian times. Mosaic sanitary laws against eating fish without fins and scales may have been derived to avoid fish containing TTX; the TTX-containing fish in the region inhabited by the Israelites were scaleless.[182]

Captain Cook recorded in 1774 his experience after eating a piece of liver from a puffer fish purchased from a native fisherman.[459] Before preparing the fish for eating, it was described and drawn. Cook tasted the liver and wrote of a vivid feeling of extraordinary weakness and numbness.[182] There has been some contention that TTX (also known as puffer powder) was used as a component of Haitian voodoo potion in the zombie ritual.[442] This has been challenged on grounds, among others, that under the usual conditions of extreme alkaline storage, any TTX in a "zombie potion" would be decomposed irreversibly into pharmacologically inactive products.[228,488]

Tetrodotoxin was named around 1911 after searching for the active ingredient in fugu ovaries.[146] Isolation of the chemical was achieved in the 1950s. In 1964, tarichatoxin, a nonprotein neurotoxin, was confirmed to be the same molecule as TTX.[300] In the 1970s, the major toxin in certain poison dart frogs was identified as TTX. Crystalline TTX was isolated in 1978.

Fugu. TTX is characteristic of the order Tetraodontiformes.[415] The suborder Tetrodontoidei contains three families of fish (Tetraodontidae, Diodontidae, and Canthigasteridae), including puffer fish (toadfish, blowfish, globefish, swellfish, balloonfish, toado) and porcupinefish. Sunfish (*Mola* species) are members of the suborder Moloidei. Puffer fish are tropical and subtropical, and some are prepared as a delicacy called fugu.[74] Puffer fish can inflate their bodies to a nearly spherical shape using air or seawater. At least 50 of the more than 100 species of these fish have been involved in poisonings of humans or may be intermittently toxic.[361] Many nonfish species also contain TTX (Box 66-3).

In Japan, chefs must undergo a rigorous certification process before they are allowed to prepare fugu. The filet of the puffer fish contains very minute concentrations of TTX. Fugu is served raw with paper-thin slices placed into an ornate configuration. The presence of small quantities of TTX gives the desired effect of slight oral tingling. Importation of fugu into the United States is illegal, but smuggling has resulted in cases of poisoning.

TTX may be produced by *Pseudomonas* species that live on the skin of the puffer fish.[495] This would explain the trans-

Box 66-3. Non-Tetraodontiformes Containing Tetrodotoxin

- Phylum Chordata
  - Order Caudata
    - Family Salamandridae
      - *Taricha granulose:* rough-skinned newt
      - *Notophthalmus viridescens:* eastern newt
      - *Triturus:* European newts
      - *Pleurodeles:* ribbed newts
      - *Cynops:* fire belly newts
      - *Paramesotriton:* warty newts
      - *Tylototriton:* crocodile newts
  - Order Anura
    - Family Bufonidae
      - *Atelopus:* stubfoot toad
- Phylum Mollusca
  - Order Caenogastropoda
    - Family Buccinidae
      - *Babylonia japonica:* ivory shell
    - Family Naticidae
      - *Natica lineata:* lined moon shell
      - *Natica vitellus:* calf moon shell
      - *Polinices didyma:* bladder moon shell
    - Family Ranellidae
      - *Charonia sauliae:* trumpet shell
  - Order Octopoda
    - Family Octopodidae
      - *Hapalochlaena maculosa:* Australian blue-ringed octopus
- Phylum Echinodermata
  - Order Paxillosida
    - Family Astropectinidae
      - *Astropecten polyacanthus:* starfish
- Phylum Nemertea
  - Order Paleonemertea
    - Family Cephalothricidae
      - *Cephalohrix linearis:* ribbon worm
- Phylum Platyhelminthes
  - Order Polycladida
    - Family Planoceridae
      - *Planocera multitentaculata:* flat worm
- Phylum Arthropoda
  - Order Decapoda
    - Family Xanthidae
      - *Atergatis floridus:* xanthith crab
  - Order Xiphosura
    - Family Limulidae
      - *Carcinoscorpius rotundicauda:* mangrove horseshoe crab

From references 141, 146, 182, 415, 485.

mittal of toxicity between toxic and nontoxic fish through skin contact. Other investigators have found that *Vibrio* and other species isolated from the intestines of puffer fish produce tetrodotoxin.[465] Tetrodotoxin is concentrated in the liver, viscera, gonads, and skin of the fish. Female fish are considered more toxic than males because there are especially high concentrations of TTX in ovaries. Musculature is less toxic but still may contain a significant amount of TTX. The toxin is heat stable and not inactivated by freezing. Seasonal variation of TTX concentration occurs, with peak levels during spawning season.

Pathophysiology. Tetrodotoxin has a unique nonprotein structure and is widely used as a research tool to study sodium channels. Mouse bioassays demonstrate that the minimal lethal dose of TTX by intraperitoneal injection is 8 to 20 μg/kg.[300] Tetrodotoxin blocks sodium channels during the rising phase of the action potential.[227] The interaction of TTX with the sodium channel is thought to be stoichiometric, with each TTX molecule interfering with one channel. Tetrodotoxin affects the spike-generating process of sodium channels, not the resting or steady-state voltage.[227]

TTX interferes with both central and peripheral neuromuscular transmission. Although it is not a depolarizing agent, in animals it causes depression of the medullary respiratory mechanism, intracardiac conduction, and myocardial and skeletal muscle contractility. At the microcellular level, the mechanism of action of TTX is linked to the axon rather than to the nerve endplate. TTX blocks axonal transmission by interfering with sodium conductance within the depolarized regions of the cell membrane, perhaps by acting at a metal cation binding site in the sodium channel, without affecting the presynaptic release of acetylcholine or its effects on the neuromuscular junction.[6,197] Saxitoxin, implicated in paralytic shellfish poisoning, has essentially the same action as tetrodotoxin on the nerve membrane, although it is believed to have a discrete receptor.[230] There is no apparent effect on potassium permeability.[359] The LD_{50} (dose at which 50% die) for mice is 10 mg/kg when TTX is administered by intraperitoneal, intravenous, or subcutaneous routes.[440]

Animal studies suggest that TTX has a peripheral effect that results in vasodilation independent of α- or β-adrenergic receptors.[213,230,267] Further studies suggest a dose-dependent action. At low doses, systemic blood pressure is lowered, although perfusion pressure is initially maintained. Higher doses of TTX result in a profound fall in blood pressure.[227] Experiments with animal models using TTX from blue-ringed octopi demonstrate a similar profound hypotension. α-Agonists (norepinephrine or phenylephrine) have been the most effective agents in raising blood pressure in models of TTX poisoning.[141]

Clinical Presentation. The onset of symptoms after TTX administration can be as rapid as 10 minutes or delayed up to 4 hours. During a 2002 outbreak in Bangladesh of 37 people (from eight families) who were poisoned from inadequately prepared puffer fish, 31 of the victims developed symptoms within 2 hours and eight died.[3] Death has been recorded within 17 minutes of exposure. The extent and type of symptoms vary according to the individual and the amount of TTX ingested. Paresthesias of the lips and tongue (in 65%) occur first and may be rapidly followed by hypersalivation, diaphoresis, lethargy, headache (in 41%), weakness (in 59%), ataxia, incoordination, tremor, paralysis, cyanosis, aphonia, dysphagia, seizures, dyspnea, bronchorrhea, bronchospasm, respiratory failure (in 22%), coma, and hypotension. Gastrointestinal symptoms can be severe and include nausea, vomiting, diarrhea, and abdominal pain (found in 22% of the Bangladeshi victims).[3] Paresthesias advance to the extremities, often with severe numbness that

has been described as a "floating" sensation. Hypersalivation with diaphoresis is common. Motor paralysis may occur, with the onset of weakness, incoordination, hyporeflexia, and cranial nerve dysfunction. Weakness and paralysis may appear in the upper extremities and descend to the lower extremities.[361] Early miosis may progress to mydriasis with poor pupillary light reflex.[440] A disseminated intravascular coagulation-like syndrome may occur and is heralded by petechial skin hemorrhages that can progress to bullous desquamation and diffuse stigmata of prolonged coagulation. Hypotension can be profound and may be refractory to treatment. Respiratory distress and insufficiency can occur. Bradycardia and atrioventricular node conduction abnormalities may be present. Complete cardiovascular collapse with respiratory paralysis precedes death. Normal consciousness may be maintained until shortly before death.[146,440] In some older reports, 60% of victims died, most within the first 6 hours. Survival past 24 hours is a good prognostic sign.

Treatment. Treatment of TTX is primarily supportive with aggressive airway management and assisted ventilation.[395] Decontamination should be considered with a dose of activated charcoal given in early presentations. Atropine may be used to treat bradycardia in conjunction with adequate oxygenation. Intravenous fluid resuscitation should be initiated for hypotension; however, use of vasopressors may be required to maintain perfusion. α-Agonists such as phenylephrine or norepinephrine are more likely to be effective.

Anticholinesterase agents such as edrophonium and neostigmine have been used to treat victims of TTX poisoning with mixed success. Some case reports have suggested subjective improvement in the neurologic symptoms after administration of anticholinesterase agents.[87,440] Other case reports noted no improvement after infusion of these compounds.[3,275,437] A review of earlier studies suggests that TTX is not antagonized by anticholinesterase agents, because the toxin interrupts neuromuscular transmission on motor neurons and muscle membranes and not the motor endplate.[226] Antihistamines and steroids have also been utilized with no clear benefit.[275]

No antidote is currently available to treat TTX poisoning; however, studies are ongoing. TTX has been conjugated to keyhole limpet hemocyanin and the conjugate used as an immunogen in rabbits and mice. Mice immunized with the conjugate were protected from a lethal dose of toxin, and passive protection experiments using rabbit antiserum exhibited a dose-related therapeutic activity after toxin challenge.[148] The shore crab *Hemigrapsus sanguineus* is highly resistant to TTX by virtue of a toxin-binding substance.[388] Whether this natural phenomenon can be applied to the human situation remains to be seen.

A minor intoxication with TTX may be limited to paresthesias and mild dysphagia. In such a case, the victim should be observed in the emergency department or intensive care unit for at least 8 hours to detect deterioration, particularly in respiratory function. The victim should not be discharged until the symptoms are clearly improving. Although water soluble, TTX is very difficult to remove from fish, even by cooking. It is wisest to avoid all puffers, even when prepared by an expert.

Grass Carp Gallbladder Poisoning

Ingestion of the Asian freshwater grass carp *(Ctenopharyngodon idellus)* or similar fish *(Cyprinus carpio)* can lead to serious illness, attributed to the nephrotoxic and hepatotoxic properties of a toxin found in the bile.[82] The structure of the toxin in *C. carpio* has been characterized as 5α-cholestane-3α, 7α, 12α, 26,27-pentol 26-sulfate.[19] Most cases have occurred in Hong Kong, Taiwan, and South Korea. Two cases were reported in the United States in immigrants who ate raw gallbladders from carp caught in Maryland.[72] One of the patients required hemodialysis for acute renal failure.

People eat the raw gallbladder for its putative antirheumatologic properties. Several hours after ingestion, abdominal pain, nausea, vomiting, and watery diarrhea develop, which can be accompanied by marked elevations in concentrations of liver enzymes (aspartate and alanine aminotransferases).[112] The hepatitis is transient and to date has not led to hepatic failure. Nephrotoxicity may be profound, leading to oliguric or nonoliguric renal failure within 48 to 72 hours after ingestion.[364,484] Renal biopsy demonstrates acute tubular necrosis without evidence of rhabdomyolysis. Therapy is supportive and based on symptoms. Hemodialysis may be necessary until renal function improves.

POISONINGS ASSOCIATED WITH ALGAL BLOOMS

Although there are thousands of species of microalgae that form the base of the food chain, less than 60 species are toxic or harmful. These toxic species can cause significant kills of fish and shellfish, mortality among seabirds and marine mammals, and human illnesses and death. In the United States, harmful algal blooms now threaten virtually every coastal state, and the number of toxic species is increasing. Several distinct clinical syndromes exist: ciguatera fish poisoning, clupeotoxic fish poisoning, paralytic shellfish poisoning, neurotoxic shellfish poisoning, diarrhetic shellfish poisoning, amnestic shellfish poisoning, possible estuary-associated syndrome, and Haff disease (see Table 66-1).

Ciguatera

Worldwide, ciguatera accounts for more cases of fish-borne illness than any other type of ichthyosarcotoxicosis.[63] Ciguatera is most commonly seen in the Indian Ocean, South Pacific, and Caribbean. In the United States, ciguatera poisoning is the most common nonbacterial food poisoning associated with fish, with cases having been reported in many states.[156,199,297,461] Outbreaks of ciguatera are greatest between the months of April and August. In endemic areas, the incidence is estimated to be between 500 and 600 cases per 10,000 people.[248] Worldwide, ciguatera may affect over 50,000 persons each year. Most cases in the United States occur in Hawaii and Florida, with the incidence in Florida estimated to be five cases per 10,000 people.[131] Outbreaks of ciguatera have been associated with the ingestion of warm-water, reef-dwelling fish caught in the zone between the latitudes of approximately 30 and 35 degrees.[32,183] In addition, the advent of flash freezing and shipping of fish around the world has accounted for several cases of ciguatera in nonendemic areas.[199]

More than 400 species of benthic fish have been implicated in ciguatera, with the greatest concentration in the Caribbean Sea, in the Pacific Ocean around the Indo-Pacific islands, and along the continental tropical reefs. The most frequently implicated reef fishes are listed in Box 66-4. Of reported cases, 75% (except in Hawaii) involve the barracuda, snapper, jack, or

Box 66-4. Frequently Implicated Reef Fish in Ciguatera Poisoning

Phylum Chordata
- Order Anguilliformes
  - Family Muraenidae: moray eels
- Order Mugiliformes
  - Family Mugilidae: mullets
- Order Perciformes
  - Family Acanthuridae: surgeonfishes
  - Family Carangidae: jacks
  - Family Labridae: wrasses
  - Family Lethrinidae: emperor fish
  - Family Lutjanidae: snappers
  - Family Scaridae: parrotfishes
  - Family Serranidae: groupers
  - Family Sparidae: porgies
  - Family Sphyraenidae: barracuda
- Order Tetraodontiformes
  - Family Balistidae: triggerfishes

From reference 165.

Box 66-5. Some Other Fish Known to Be Ciguatoxic

Albulidae (ladyfishes)
Chanidae (milkfishes)
Clupeidae (herrings)
Elopidae (tarpon)
Engraulidae (anchovies)
Synodontidae (lizardfishes)
Congridae (true eels)
Ophichthyidae (snake eels)
Belonidae (needlefishes)
Exocoetidae (flying fishes)
Hemiramphidae (halfbeaks)
Aulostomidae (trumpetfishes)
Syngnathidae (seahorses)
Holocentridae (squirrelfishes)
Apogonidae (cardinalfishes)
Arripidae (sea perches)
Chaetodontidae (butterfly fishes)
Cirrhitidae (hawkfishes)
Coryphaenidae (dolphins)
Gempylidae (oilfishes)
Gerridae (silverfishes)
Gobidae (gobies)
Istiophoridae (sailfishes)
Kuhliidae (bass)
Kyphosidae (rudderfishes)
Mullidae (goatfishes)
Pempheridae (sweeperfishes)
Pomacentridae (damselfishes)
Pomadasyiidae (grunts)
Priacanthidae (snapper)
Scatophagidae (spade fishes)
Sciaenidae (croakers)
Scombridae (tunas)
Scorpaenidae (scorpionfish)
Siganidae (rabbitfishes)
Xiphiidae (swordfishes)
Zanclidae (moorish idol)
Bothidae (flounders)
Aluteridae and Monacanthidae (filefishes)
Ostraciontidae (trunkfishes)
Batrachoididae (toadfishes)
Antennariidae (sargassumfish)
Lophiidae (goosefish)
Ogcocephalidae (longnose batfish)

grouper. Hawaiian carriers of the toxin include the parrot-beaked bottom feeders and surgeonfishes, particularly those inhabiting waters with high dinoflagellate populations, such as those with disturbed coral reefs.[212] Other fish that have been reported as ciguatoxic are listed in Box 66-5. Ciguatera has also been reported after ingestion of farm-raised salmon.[119] There is one report of ciguatera from consumption of jellyfish.[499]

The name ciguatera is derived from the Spanish name *cigua* for the sea snail *Turbo pica* found in the Caribbean Spanish Antilles.[29,435] This neurotoxic syndrome has been recognized throughout history, with one of the earliest cases having probably been reported in the 4th century when Alexander the Great refused to allow his soldiers to eat fish, and another during the Tang Dynasty in China.[408] One of the earliest written records of suspected ciguatera poisoning is from the journal of Captain William Bligh, who described symptoms consistent with ciguatera in 1789 after eating mahimahi.[408] In addition, it was also quite possibly ciguatera that was illustrated by Captain James Cook while sailing on the *Resolution* in the South Pacific in 1774.[325] In this episode, several of his officers were poisoned after eating fish from the island Malicolo in the Pacific nation of Vanuatu.

Pathophysiology. The blue-green and free algal dinoflagellate *Gambierdiscus toxicus* is thought to be responsible for producing ciguatoxins.[408] *G. toxicus* adheres to dead coral surfaces and marine algae that are consumed by smaller herbivorous fish.[165,248] Although *G. toxicus* is very likely responsible for the majority of ciguatoxins encountered in fish, the cyanobacterium *Trichodesmium erythraeum* can produce water- and lipid-soluble precursors to the toxins that may generate ciguatera syndrome.[130] Other dinoflagellates, such as *Prorocentrum concavum, Prorocentrum mexicanum, Prorocentrum rhathymum, Gymnodinium sanguineum,* and *Gonyaulax polyedra,* may generate toxins that play a role in ciguatera syndrome.[341,438]

Larger reef fish eat the contaminated smaller fish, thereby becoming vectors as ciguatoxin is bioconcentrated up the food chain. As the fish within the food chain become larger and older, the toxin is accumulated.[98,193,203,328] Experimental induction of ciguatera toxicity in yellowfin surgeonfish *(Acanthurus xanthopterus)* has been achieved by a diet of ciguatoxic red snapper *(Lutjanus bohar)*.[196] Although the entire fish is toxic, the viscera (particularly the liver) and roe are considered to carry the highest concentrations of toxin.[31] No plankton feeders have so far been reported to be ciguatoxic.

It has been suggested that the proliferation of toxic algae may be triggered by contamination of the water from a number of

sources, including industrial wastes, golf course runoff, metallic compounds, ship wreckage, or other pollutants.[183] In the Marshall Islands (Micronesia), consequent to nuclear testing, the incidence of toxin-producing plankton has tripled.[350] Similar observations have been made with respect to various military activities (dumping and explosives) in the Line Islands and Gilbert Islands (Kiribati, Central Pacific), Hao Atoll (Tuamotu Archipelago, French Polynesia), Gambier Islands (French Polynesia), and others.[358] Yet another cause of toxic dinoflagellate proliferation may be the transfer and dumping of ballast water from large oceangoing vessels.

Ciguatera is associated with more than five toxins, including fat-soluble quaternary ammonium compounds (ciguatoxins), a water-soluble component (maitotoxin, from the Tahitian vernacular name *maito* for the striated surgeonfish *Ctenochaetus striatus*), a maitotoxin-associated hemolysin (lysophosphatidylcholine, or lysolecithin), and a ciguatoxin-associated adenine triphosphatase (ATPase) inhibitor.[186,256,262,368] Scaritoxin (isolated from *Scarus gibbus*) is similar to the fat-soluble component and is specific to parrotfishes.[91] Lipid-extracted toxins from *Gambierdiscus toxicus* have been designated GT-1, GT-2, and GT-3; a water-soluble toxin is designated GT-4.[115,291] Chemical analysis of ciguatoxins demonstrates that they closely resemble brevetoxin C (from *P. brevis*) and okadaic acid, isolated from marine sponges and the dinoflagellate *Prorocentrum lima*.[139,303] The identification of okadaic acid from the Caribbean dinoflagellate *Prorocentrum concavum* lends support to the notion that this toxin may be more significant in ciguatera poisoning than previously thought. Another compound, named prorocentrolide, has also been found in reef-dwelling fish with okadaic acid and has been implicated in diarrhetic shellfish poisoning, another common fish-borne illness.[139,202]

Three major ciguatoxins (CTX-1, CTX-2, and CTX-3) are usually found in the flesh and viscera of ciguateric fishes. Each is found in variable concentrations, which may account for the inconsistency of reported clinical signs and symptoms.[262] CTX-2 is a diastereomer of CTX-3.[265] Ciguatoxins may result from the oxidation of gambiertoxins, possibly through the cytochrome system in the liver of fish.[264] The lipid components have been characterized as crystalline, colorless, heat-stable compounds of approximate 1100 daltons molecular weight, with functional hydroxyl and quaternary nitrogen groups. CTX is excreted in bodily fluids, and toxicity can be transmitted by both semen and breast milk.[61,296]

Ciguatoxins activate voltage-dependent Na^+ channels.[261] One mechanism of their action may be that they falsely occupy calcium receptor sites that modulate sodium pore permeability in neural, muscle, and myocardial membranes.[38] This effect could allow increased membrane permeability to sodium and cause sustained depolarization. Electrophysiologic studies of the sural and common peroneal nerves in humans with ciguatera, demonstrating reduced light touch, pain, and vibratory sensation in the extremities, showed prolongation of the absolute refractory, relative refractory, and supernormal periods. These findings indirectly suggest that CTX may abnormally prolong sodium channel opening in nerve membranes.[64] This influx of sodium is antagonized by the presence of tetrodotoxin.[43]

In vitro studies have also shown that scaritoxin causes the release of norepinephrine and acetylcholine and increases sodium channel permeability.[426] Maitotoxin as well may trigger the release of norepinephrine and stimulate cellular uptake of calcium and has been hypothesized to stimulate cholinergic receptors by inhibiting acetylcholinesterase.[43,394] However, evidence suggests that highly purified ciguatoxin preparations may not have anticholinesterase effects in vivo.[255]

Palytoxin is an extremely poisonous nonprotein agent of low molecular weight that has been isolated from various zoanthid soft corals of the genus *Palythoa*, although its true origin is unknown.[178] In animal models, it causes a fast potassium outflow from erythrocytes, increases the permeability of cell membranes to sodium and potassium but not calcium, and inhibits sodium–potassium ATPase. Although palytoxin is presumed to originate in coelenterates or even bacteria, it has been suggested that it may be a causative agent for ciguatera.[241,287,317] Ingestion in humans reportedly causes abdominal cramps, nausea, diarrhea, paresthesias of the extremities, muscle spasm, and respiratory distress. Of this cluster of symptoms, the predominant physical findings appear to be respiratory distress and extreme tonic muscle contractions with massive accumulation of serum creatine kinase and myoglobinuria. Palytoxin has been found in mackerel *(Decapterus macrosoma)*, triggerfish *(Melichtys vidua)*, and crab *(Demania reynaudii)*.[9,149,241] Because a rapid and sensitive hemolysis neutralization assay for palytoxin is now available, its presence in toxic seafood, and perhaps its role in ciguatera, should become easier to determine.[44]

Hypertension occurring with ciguatera can be suppressed in animal models with phentolamine (an α-antagonist), suggesting α-adrenergic receptor activity. Although purified ciguatoxin appears to have cardiac stimulatory effects (increasing heart rate and output), maitotoxin is a myocardial depressant in vitro, which may explain the variation in clinical presentation. Isolated human atrial trabeculae show concentration-dependent positive inotropy with CTX-1 that is not reversed with mannitol.[261] Cardiac calcium conduction effects have been implicated in the activity of maitotoxin, because its action is inhibited in the presence of verapamil, magnesium ions, or low-calcium-concentration solutions. In mice, injection of maitotoxin can induce a marked increase in total calcium content of the adrenal glands and a rise in plasma cortisol concentration.[433] When injected into mice, ciguatoxin targets the heart, adrenal glands, and autonomic nervous system.[435] Ciguatoxin and CTX-4c (a derivative), administered in repeated doses, cause the mouse heart to suffer septal and ventricular interstitial fibrosis, accompanied by bilateral ventricular hypertrophy.[431] Ciguatoxin is a potent substance, with an LD_{50} in mice of 0.45 mg/kg in purified form. In most toxic fish flesh, the concentration is 0.5 to 10 parts per billion. Maitotoxin is even more potent, with an LD_{50} of 0.13 mg/kg in mice. It is interesting to note that ciguatoxins can become toxic to fish in higher concentrations, thus potentially limiting the levels of these compounds carried by a fish.[260] However, the toxin or toxins may reside in the skeletal muscle or other tissues of the fish in association with proteins that may be protective of the carrier.[179]

All identified toxins associated with ciguatera are unaffected by freeze-drying, heat, cold, and gastric acid and do not affect the odor, color, or taste of the fish. There is some evidence that cooking methods can alter the relative concentrations of the various toxins. For example, boiling fish flesh will remove water-soluble toxins, but frying or grilling the flesh may increase toxicity of lipid-soluble toxins as a result of releasing lipid-soluble components from the cellular compounds to which they are normally bound.[129]

Box 66-6. Signs and Symptoms Associated with Ciguatera Poisoning

Abdominal pain
Nausea
Vomiting
Diarrhea
Chills
Paresthesias (particularly of the extremities and circumoral region)
Pruritus (particularly of the palms and soles)
Tongue and throat numbness or burning
A sensation of "carbonation" during swallowing
Odontalgia or dental dysesthesias
Dysphagia
Dysuria
Dyspnea
Weakness
Fatigue
Tremor
Fasciculations
Athetosis
Meningismus
Aphonia
Ataxia
Vertigo
Pain and weakness in the lower extremities
Visual blurring
Transient blindness
Hyporeflexia
Seizures
Nasal congestion and dryness
Conjunctivitis
Maculopapular rash (erythematous, with occasional desquamation)
Skin vesiculations
Dermatographia
Sialorrhea
Diaphoresis
Headache
Arthralgias
Myalgias (particularly in the lower back and thighs)
Insomnia
Bradycardia
Hypotension
Central respiratory failure
Coma

Clinical Presentation. The meal containing toxins of ciguatera is generally unremarkable in taste and smell. Symptoms may develop within minutes of ingestion, although they generally occur within 2 to 6 hours after the meal. Almost all victims develop symptoms by 24 hours, and the toxicity can generally be ruled out if the onset is delayed longer than this time.[29,131] The severity of symptoms seems to follow a dose-dependent pattern, with victims who eat larger portions of ciguatoxic fish experiencing more severe symptoms (Box 66-6).

The most common initial symptoms reported in cases of ciguatera include acute gastroenteritis, with abdominal cramps, nausea, vomiting, and diarrhea.[29] These symptoms rarely persist for longer than 24 hours but may require fluid resuscitation.[131] A myriad of other symptoms have been reported in ciguatoxic patients and are listed in Box 66-6. Headache is a common symptom, and victims often complain of experiencing a metallic taste. An overall death rate of 0.1% to 12% has been reported with ciguatera, but the lower percentage seems more likely with modern supportive care. Death is usually attributed to respiratory paralysis.[222] In a well-described clinical outbreak affecting a group of scuba divers who consumed coral trout *(Cephalopholis miniatus),* the most common symptoms were weakness, cold sensitivity, paresthesias, a taste sensation of carbonation, and myalgias.[3] Two men suffering from ciguatera poisoning had painful ejaculation with urethritis, which in turn may have induced dyspareunia (pelvic and vaginal burning) in their female partners after intercourse.[249] The variations in symptoms of ciguatera poisoning may result from different concentrations of specific toxins in different fish.[240] Characteristics associated with a severe attack include ingestion of a carnivorous fish and a history of previous ciguatoxism.[166] Neurologic symptoms seem to develop after initial gastrointestinal symptoms. Paresthesias and myalgias are typically seen within the first 24 hours and usually resolve by 48 to 72 hours after ingestion of ciguatoxins, although there have been reports of neurologic symptoms persisting for weeks to months.[28,253,329]

Many case reports of ciguatera describe symptoms of a sensory perception of "hot and cold reversal," and loose, painful teeth. Although the presence of these symptoms is suggestive of ciguatera, their absence does not exclude the possibility of the disease.[28] There have also been reports of a paradoxical reversal of temperature perception, only resulting in cold feeling hot rather than hot feeling cold.[28] However, other reports demonstrated that gross temperature discernment remains intact and the description of paradoxical heat perception may be misleading.[63] These authors describe the symptoms as intense, painful tingling or "electric shock" rather than true reversal of hot and cold perception.[63] This peculiar symptom may have a delay in onset of 2 to 5 days, may last for months after ingestion, and is otherwise seen only with neurotoxic shellfish poisoning (brevetoxins), caulerpicin (from the green alga *Caulerpa*) toxicity, or turban shell poisoning.[121,487] Pruritus is another vague but often described sensation in victims of ciguatera. The onset of pruritus may be delayed for more than 24 hours but is rarely, if ever, seen in the absence of other symptoms.[139,253] Pruritus may persist for weeks and be exacerbated by any activity that increases skin temperature (blood flow), such as exercise or alcohol consumption.[253] Ciguatera-associated pruritus may occasionally become severe and may improve after treatment with histamine receptor antagonists. Delayed symptoms may also include hiccups.

Tachycardia and hypertension are often described in ciguatera poisoning, in some cases after transient bradycardia and hypotension, which can be severe.[84] Hallucinations, flushing, flaccid paralysis, and fever occur but are uncommon. More severe reactions tend to occur in persons previously stricken with the disease. Severely affected persons may report intermittent symptoms for up to 6 months, with a gradual diminution in frequency and intensity. There may be some regional variability to the symptoms of presentation.[28,296] Persons who have ingested parrotfish (scaritoxin) have been reported to suffer from classic ciguatera poisoning, as well as a second phase of toxicity 5 to 10 days after the initial onset, consisting of ataxia, dysmetria, and a resting or kinetic tremor.[90] Whether ciguatoxin

crosses the placenta is not known, but exposures during pregnancy have resulted in normal fetal outcomes.[380]

In small children, the symptoms of ciguatera poisoning may be no more specific than irritability, sleep disturbance, nausea, and vomiting.[470] Other reported symptoms have included carpopedal spasm, ptosis, and inconsolability.

Diagnosis. The diagnosis of ciguatera poisoning is based on clinical symptoms. The differential diagnosis includes paralytic shellfish poisoning, eosinophilic meningitis, type E botulism, organophosphate insecticide poisoning, tetrodotoxin poisoning, and psychogenic hyperventilation.[28,357] Unreliable folklore used in the past to aid in predicting ciguateric seafood includes the advice that a lone fish (separated from the school) should not be eaten; that ants and turtles refuse to eat ciguatoxic fish; that a thin slice of ciguatoxic fish does not show a rainbow effect when held up to the sun; and that a silver spoon tarnishes in a cooking pot with ciguatoxic fish.[96] Ciguatoxin may be detected in the flesh of fish by two immunoassay techniques, a mouse bioassay where a sample of the fish is injected intraperitoneally into a mouse, and a rapid IgG assay.[199] Rapid immunoassays have largely replaced using mice and traditional tests (e.g., feeding fish to a mongoose or cat to observe for neurologic symptoms or death). HPLC is also available for ciguatoxins and okadaic acid. Unfortunately, tests for ciguatoxin are still of limited clinical benefit because most institutions do not have the equipment needed for their performance.

Treatment. If possible, a piece of the implicated fish should be obtained in the event that analysis for ciguatoxins can be performed. The treatment of ciguatera poisoning is primarily supportive. Intravenous hydration with crystalloid and electrolyte replacement may be necessary for dehydration. Severe or refractory hypotension may require a vasopressor such as dopamine. Antiemetics such as droperidol, prochlorperazine, and metoclopramide may be beneficial. Atropine has been shown to be effective in patients with symptomatic bradycardia or excess cholinergic stimulation.[139] Gastric decontamination is rarely indicated, because presentation is usually delayed and gastroenteritis has already occurred. Activated charcoal may bind some of the toxin in the gastrointestinal tract, but this is not useful when presentation is delayed.

Many traditional remedies have been used for centuries to treat ciguatera. Edrophonium, neostigmine, corticosteroids, pralidoxime, ascorbic acid, pyridoxine (vitamin B_6), salicylic acid, colchicine, and vitamin B complex have all been tried with variable success; however, there is no current clinical support for these modalities.[296] Local anesthetics (e.g., lidocaine, tocainide) have also been administered for treatment of ciguatera.[65,250] These agents are effective blockers of sodium influx and may antagonize the sodium channel effects of ciguatoxin. In addition, amitriptyline has been used for its sodium channel blocking effects, as well as its potent antimuscarinic effects.[56,61,108] Nifedipine has been used to counteract the cellular uptake of calcium caused by maitotoxin and to relieve headache.[61] Although there is limited experience with most of these therapies, they may be beneficial in cases refractory to supportive care alone.

Mannitol has become the most widely applied therapy in severe cases of ciguatera poisoning.[54,409] Most reports of its success are based on limited data with small numbers of patients.[124,322,326,471] One series described successful treatment with mannitol in 24 victims of ciguatera poisoning. Each was infused with up to 1g/kg of a 20% mannitol solution intravenously over 30 minutes. None of the victims received more than 250 mL.[322] The mechanism by which mannitol might be effective in abating the neurologic symptoms from ciguatera poisoning is unknown, but suggested theories have included acting as a free radical scavenger, acting as a competitive inhibitor of ciguatoxin at the cell membrane, and promoting a decrease in Schwann cell edema.[326,471] It is also possible that the osmotic action of mannitol may render ciguatoxin inert.[322,326] Curiously, mannitol therapy seems to have no beneficial effect on mice administered a sublethal intraperitoneal dose of ciguatoxin (CTX-1).[263] A more recent double-blinded, randomized study on mannitol therapy found no difference in resolution of symptoms when compared with saline.[369] Of note, therapy was not begun until an average of 19 hours after exposure in the mannitol group, and 40 hours after exposure in the saline group. In humans, the empiric observation is that mannitol has greater benefit if administered early in the course of the illness, so the delay may have diminished the effect in this study. One concern with the administration of mannitol in the setting of ciguatera is that patients may present dehydrated. In these cases, patients should be adequately rehydrated before the administration of mannitol. During recovery from ciguatera, it is recommended that victims exclude fish, shellfish, alcoholic beverages, and nuts and nut oils from their diet, as these could result in an exacerbation of the syndrome.[393] Gabapentin has been used successfully in the treatment of chronic symptoms after ciguatera poisoning, but the symptoms seem to recur after cession of therapy in some patients.[329]

Prevention. For travelers, common sense dictates avoiding any fish the local fisherman and residents do not eat, or fish caught in areas known to be endemic for ciguatera. Because of the accumulation of toxin, all oversized fish of any predacious reef species (such as jack, snapper, barracuda, grouper, or parrot-beaked bottom feeder) should be suspected to be toxic. Moray eels should never be consumed. Internal organs of implicated fish seem to concentrate the toxin and should therefore be avoided. Natural events such as hurricanes and earthquakes have been associated with an increased incidence of ciguatera, presumably because of reef disturbance. El Niño storms may also affect the incidence of ciguatera in the Pacific.[32]

CLUPEOTOXIC FISH POISONING

Clupeotoxic fish poisoning involves plankton-feeding fish that ingest blue-green algae and dinoflagellates. This poisoning is distinguished from ciguatera on the basis of the severity and high fatality rate of clupeotoxic fish poisoning and identification of the implicated clupeoid fish. These fish of the order Clupeiformes are found in tropical Caribbean, Indo-Pacific, and African coastal waters. Examples include the families *Clupeidae* (herrings and sardines), *Engraulidae* (anchovies), *Elopidae* (tarpons), *Albulidae* (bonefishes), and *Pterothrissidae* (deep-sea slickheads).[30,289] Toxicity is reported to increase during the warm summer months. Viscera are considered to be highly toxic.

Previously, the toxin was poorly characterized as a result of the infrequency of the syndrome and rare access to toxic

animals. The first case to shed light on clupeotoxism was reported in a Madagascar woman who died after eating a sardine, *Herklotsichthys quadrimaculatus*.[320] This same sardine has been implicated in clupeotoxism in Fiji and the Philippines.[489,491] The causative toxin was identified as palytoxin or its analog, which distinctly differed from ciguatoxin. The benthic dinoflagellate *Ostreopsis siamensis* was presumed to be the probable toxin source.[320,486] As with ciguatoxin, the poison typically does not impart any unusual appearance, odor, or flavor to the fish.

Clinical Presentation. The onset of symptoms is rapid and characterized as violent, often within 30 to 60 minutes of ingestion and rarely prolonged beyond 2 hours. Initial signs and symptoms include a marked metallic taste in the mouth, xerostomia, nausea, vomiting, diarrhea, and abdominal pain. These symptoms are followed by chills, headache, diaphoresis, severe paresthesias, muscle cramps, vertigo, malaise, tachycardia, peripheral cyanosis, hypotension, delirium, and death, with a mortality of up to 45%. Severe debility leading to death may occur within 15 minutes of the onset of symptoms.[183] A postmortem examination in one case after ingestion of *Sardinella marquesensis* (Marquesan sardine) flesh and viscera demonstrated enterocolitis and the sequelae of hypotension and acute heart failure.[289]

Treatment. Therapy is supportive and based on symptoms. Because of the severe nature of the intoxication, early gastric emptying is desirable; however, the disease is so unusual and so rarely suspected (because of a lack of history) that gastric emptying is not often considered. Aggressive management and early intensive care are essential.

Prevention. Clupeotoxic fish should be avoided, especially during summer months. These fish are indigenous to the Caribbean, African coastal, and Indo-Pacific waters. The viscera of suspicious fish can be fed to experimental animals to see if an illness is generated.

PARALYTIC SHELLFISH POISONING

Shellfish have been implicated in poisonings for centuries, if not millennia. Epidemics of shellfish toxicity have been linked to the proliferation of dinoflagellates and other small marine organisms that are responsible for red tides or blooms in oceans around the world. The Bible refers to red tides in Exodus 7:20–21, where "the waters that were in the rivers were turned into blood, and the fish that was in the rivers died; and the river stank." The Red Sea was so named by ancient Greeks for its red appearance in certain seasons when red tides occurred. Red tides are described in the *Iliad* and were first recognized by North American Indians as luminescence or "flickering" of ocean waves.[69]

The first published description in the Western world of a patient with clinical findings suggestive of paralytic shellfish toxicity probably dates back to 1689. An article from a French journal named *Ephemeredes des Curieux de la Nature* described a young woman who had ingested mussels.[86,181] The description notes that her symptoms included fever, chest pain, respiratory insufficiency, nausea, seizures, and tachycardia. She had emesis induced, bringing up the mussels, and eventually recovered. For years after this report, the incidence and cause of paralytic shellfish toxicity were undocumented throughout the world, but epidemics were known to occur in certain seasons and under certain conditions. Improvements in monitoring and public health reporting have demonstrated patterns of occurrence. Gessner and Middaugh[157] described 54 outbreaks of paralytic shellfish poisoning (PSP) in Alaska occurring in 117 individuals between 1973 and 1992. The California Paralytic Shellfish Poisoning Prevention Program has been so successful that it has been a model of surveillance for many other countries.[337] PSP has been a reportable condition in California since 1927, with more than 500 cases and 30 deaths reported since that time. In California there is an annual 6-month quarantine (May through October) on locally harvested mussels, clams, and oysters.

Of the several types of neurologic diseases occurring after ingestion of shellfish, PSP is one of the most common. This syndrome is most frequently reported during the summer months when water temperature is highest, but it has been recorded from May to November.[168,181] Some authors suggest that the toxin responsible for PSP may be present in significant concentration in some shellfish, such as the Alaskan butter clam, in some areas year round, and that shellfish harvested from untested waters of these regions never be consumed.[157] The most commonly implicated varieties of shellfish include mussels, clams, oysters, and scallops.[157,181,229] Although almost all outbreaks have been described from shellfish consumption, 13 cases of PSP were diagnosed in Florida in 2002 after ingestion of puffer fish containing saxitoxin, rather than TTX, that were caught in the waters off Titusville.[80]

Pathophysiology. The unicellular phytoplankton *Protogonyaulax* (phylum Protozoa, class Mastigophora, order Dinoflagellata), measuring 0.03 mm in diameter, is the foundation of the ocean food chain, and in warm summer months these organisms are noted to bloom rapidly in nutrient-rich coastal temperate and semitropical waters. They rank second in abundance to diatoms and are extremely important producers of marine carbohydrates, fats, and proteins. Typical other species include *Alexandrium* species and *Gymnodinium catenatum*.

Dinoflagellates produce a number of toxins, the most commonly identified of which is saxitoxin. If a single organism predominates, it can discolor the water, creating a black, blue, pink, red, yellow, brown, or luminescent "tide."[92] Organisms can multiply rapidly from a concentration of 20,000/L to over 20 million/L. These plankton can release massive amounts of toxic metabolites into the water, at times leading rapidly to enormous mortality in various bird and marine populations, including large mammals such as dolphins and even whales. Large numbers of dead animals on the beach suggest a colored tide. The trend to increased numbers and magnitude of blooms is empirically attributable to many factors, including coastal development, dumping of sewage, fertilizer runoff, and ocean warming. Kills by the dinoflagellate *Ptychodiscus* (formerly *Gymnodinium*) *brevis* are estimated at 100 tons of fish per day. The problem is markedly increasing in Europe.[452]

A limited number out of the approximately 1200 species of dinoflagellates have been implicated in human toxic syndromes.[367] PSP has been linked to the dinoflagellate *Protogonyaulax*, species *catanella* (U.S. Pacific coast) and species *tamarensis* var. *excavata* (U.S. Atlantic coast and Europe).[284,427] In northwestern Spain, the organism that generates the toxins

causing this syndrome is *Gymnodinium catenatum*. These creatures are relatively fastidious and prefer to bloom in warm, sunlit water of low salinity. Some algal organisms may release their toxin in the form of microscopic cysts, which can overwinter at the sediment–water interface. In mollusks, the greatest concentration of toxin is found in the digestive organs (e.g., the dark hepatopancreas), gills, and siphon.[382] Toxic benthic dinoflagellate cysts may be transported by dredging operations, potentially introducing a dinoflagellate population into a new region.[492]

Although the origin of PSP toxins is assumed to be dinoflagellates, the toxins have been isolated in both marine and freshwater bivalves that are not associated with dinoflagellates. It has not been determined how this has occurred.[314] The bacterium *Moraxella* isolated from *Protogonyaulax tamarensis* has been shown to produce PSP toxins in culture. Toxin production can increase in nutritionally deficient environments.[242]

The paralytic shellfish toxins identified to date are 18 related tetrahydropurine compounds produced mainly by the dinoflagellates of the genus *Alexandrium*. These include saxitoxin, neosaxitoxin, and the gonyautoxins (GTX1, GTX2, GTX3, GTX4, GTX5), with the best characterized being saxitoxin.[154] Saxitoxin ($C_{10}H_{17}N_7O_4$) takes its name from *Saxidomus giganteus*, the Alaskan butter clam. *P. brevis* is a toxic dinoflagellate that produces a milder toxin. Other dinoflagellates considered poisonous to animals or humans include *Gonyaulax acatenella, Pyrodinium phoneus, Pyrodinium bahamense* var. compressa, *Gonyaulax monilata, Gonyaulax polyhedra, Gymnodinium veneficum,* and *Exuviaella ariae-lebouriae*.[321] *S. giganteus* and the Washington clam *(Saxidomus nuttalli)* may carry the toxin in their neck parts for up to 2 years; however, no physical characteristic distinguishes a carrier animal.

Unfortunately, a direct human serum assay to identify the toxin responsible for PSP is not readily available to the clinician. PSP is assessed in foodstuff using the mouse bioassay, in which a 20-g mouse is injected with 1 mL of an acid extract of the shellfish, and the time taken for the animal to die is recorded. One mouse unit (mu), or 0.18 mg, is the amount of injected saxitoxin that kills a test mouse in 15 minutes.[452] In most countries, the action level for closure of a fishery is 400 mu/100 g shellfish. Polyclonal enzyme-linked immunosorbent assays (ELISAs) that measure saxitoxin, neosaxitoxin, and gonyautoxins 1 and 3 may be refined soon as reasonable screening techniques. Other testing methods under investigation include a sodium channel blocking assay, spectrometry, thin-layer chromatography, and fluorometric HPLC.[154,274] An automated tissue culture (neuroblastoma cell) bioassay may become a valid alternative to live animal testing.[218]

Saxitoxin and related compounds are water soluble and heat and acid stable. Twelve derivatives of saxitoxin (such as neosaxitoxin) that originate from dinoflagellates of the genus *Protogonyaulax* are formed by the addition to saxitoxin of *N*-1-hydroxyl, 11-hydroxysulfate, and 21-sulfo groups.[180] Like tetrodotoxin, they can be destroyed to a certain extent in an alkaline medium but not by ordinary cooking. Saxitoxin acts by blocking sodium channels in nerve and muscle cells.[133,227] Inhibition of sodium flow in these cells arrests impulse conduction along neurons. In addition, saxitoxin may suppress atrioventricular nodal conduction, directly depress the medullary respiration center, and progressively reduce peripheral nerve excitability. Because of the similarity of the clinical syndrome to tetrodotoxin poisoning, it has been inferred that the same receptors may be blocked. Although the threshold levels for causing illness in humans are not definitively known, it has been suggested that ingestion of 200 to 500 µg would cause at least mild symptoms; 500 to 2000 µg, moderate illness; and more than 2000 µg, serious or fatal illness. However, serious symptoms have been reported after ingestion of less than 100 µg of saxitoxin in adults. During peak red tide seasons, each mussel may accumulate up to 50,000 mu of saxitoxin. Mussel concentrations of saxitoxin have been determined to be too high for consumption when seawater dinoflagellate counts are as few as 200/mL.[382] A saxitoxin concentration of greater than 75 to 80 µg/100 g foodstuff is considered hazardous to humans. In the 1972 New England red tide, the concentration of saxitoxin in blue mussels exceeded 9000 µg/100 g foodstuff. After cases of PSP in Massachusetts, saxitoxin concentrations of 24,400 µg/100 g were recorded in raw mussels. With oral ingestion of saxitoxin, the LD_{50} for mice is 263 µg/kg. It has been estimated that as little as 0.5 to 1 mg of saxitoxin can be fatal in humans.[382]

Neither steaming nor cooking affects the potency of the toxin. Commercial processing of shellfish does not eliminate the toxin or the potential for toxicity; therefore, public health agencies in the United States and Canada strictly monitor these canning industries.

Clinical Presentation. The onset of symptoms of paralytic shellfish poisoning is rapid. Within 30 to 60 minutes of ingesting toxic shellfish, victims complain of paresthesias, numbness, vertigo, and tingling of the face, tongue, and lips. Cranial nerve dysfunction, including dysarthria, dysphonia, dysphagia, and even blindness, can occur.[157,181,203,284] Other early symptoms include lightheadedness, a floating sensation, ataxia, weakness, hyperreflexia, incoherence, sialorrhea, thirst, abdominal pain, nystagmus, dysmetria, headache, diaphoresis, a sensation of loose teeth, chest pain, and tachycardia. Neurologic symptoms progress to involve the extremities and trunk over the first 1 to 2 hours. Weakness of the limbs may begin anytime after the sensory changes, and they gradually progresses to ataxia, inability to use the extremities, and finally paralysis. Reflexes are frequently normal throughout progression of the disease, and patients remain awake and alert. Death results from respiratory failure with diaphragmatic and chest wall muscle paralysis.

Although some victims have nausea, vomiting, or diarrhea, the lack of gastroenteritis and thus early self-decontamination may in part explain why mortality from PSP approaches 25% in some older series.[29,468] More recent reports cite a lower incidence of fatalities, probably because of improvements in supportive care. Hypotension can result from direct action of the toxin on vascular smooth muscle.[227] Toxicity is never delayed more than 10 to 12 hours, with a median onset of 3 hours. Prognosis is good for individuals surviving past 12 hours, but weakness can persist for weeks after recovery. Children seem to be more sensitive to saxitoxin than adults. In milder cases, alcohol ingestion appears to increase toxicity.

Treatment. No antidotes are currently available for saxitoxin or paralytic shellfish poisoning. The victim should be closely observed in the hospital for at least 24 hours for respiratory insufficiency. Airway patency and respiratory support are of utmost importance, and even patients with severe symptoms of

PSP often do well if expeditiously supported with mechanical ventilation. Although gastric emptying has been advocated by some authors when shellfish suspected of containing saxitoxin are ingested, airway collapse can be rapid and induction of emesis should not be attempted.[203] These toxins bind well to charcoal, and an oral dose of charcoal should be administered if this can be done safely.[345] Some authors suggest that atropine administration may worsen symptoms of PSP and should be avoided because saxitoxin and its derivatives may have some antimuscarinic effects.[381] Several studies have also suggested that acidity may enhance the potency of saxitoxin, leading some authors to speculate that serum alkalinization might be of benefit to victims, although the efficacy of this practice has yet to be established.[10,192,280,324]

At least one human case report and some animal data have implied that dialysis or hemoperfusion may benefit some victims of severe PSP.[29,345] Other reports are less optimistic, as in vitro trials have demonstrated that dialysis is not effective in removing saxitoxin.[192,125] Some authors have suggested enhancing renal clearance with diuresis, but no study supports this practice. Maintaining normal urine output should suffice in most cases.

Prevention. The most important aspect of managing PSP is prevention. Although leeching of shellfish in freshwater for several weeks followed by vigorous cooking may remove up to 70% of the toxin, such procedures are recommended only for persons stranded on desert islands. It has been said that one should not eat shellfish in the northern hemisphere in months that contain the letter *r*. It has become more apparent with changing ocean conditions that shellfish in many parts of the world may be contaminated throughout the year because of high water concentrations of *Gonyaulax*. Most coastal agencies monitor dinoflagellate concentrations off the shores of developed countries and restrict shellfish harvesting during high-risk periods. Many outbreaks of this illness have occurred on isolated islands where public health monitoring is infrequent and intensive care medicine resources scarce.

NEUROTOXIC SHELLFISH POISONING

Ptychodiscus brevis is a toxic dinoflagellate that creates a colorful tide when it blooms. Ingestion of shellfish contaminated with *P. brevis* can induce a milder version of PSP known as neurotoxic shellfish poisoning (NSP). The condition resembles ciguatera toxin poisoning in symptoms and does not have a major paralytic component. Death has not been reported in humans. Symptoms include circumoral paresthesias and paresthesias of the extremities, dizziness and ataxia, muscle aches, and gastrointestinal symptoms. The median incubation time for this illness is 3 hours, and it lasts several hours to a few days.[298] Most NSP outbreaks have occurred on the Gulf of Mexico, the west coast of Florida, coastal Texas, North Carolina, and New Zealand.[298,400]

Unlike other shellfish poisons, NSP can cause a respiratory irritation syndrome. When large blooms of *P. brevis* occur near the shoreline, wind and wave action can aerosolize the toxin; if sea breezes blow the aerosolized toxin onshore, rapidly reversible conjunctivitis, rhinorrhea, and bronchospasm with nonproductive cough can occur in sensitive individuals.[203] Severe respiratory distress is uncommon. The effects are similar to those of muscarinic stimulation.

P. brevis produces at least 10 toxins, known as brevetoxins.[26,27] Brevetoxins (designated PbTx-1 to PbTx-10) are potent, lipid-soluble, cyclic polyether compounds that bind to and modulate voltage-gated sodium channel activity. Brevetoxins produce acute neuronal injury and death in rat cerebellar neurons.[41] In a canine model, brevetoxins produce depolarization of tracheal and bronchial smooth muscle.[351] Intratracheal brevetoxin instillation in rats resulted in systemic distribution of brevetoxin, which suggests that the initial respiratory irritation and bronchoconstriction may be only part of the toxicologic syndrome with brevetoxin inhalation.[37]

The brevetoxins are potent ichthyotoxins and are associated with large numbers of dead birds, fish, and mammals. In 1996, 149 manatees died along the southwest Florida coast; brevetoxin was implicated as the primary cause of the epizootic.[52] Signs and symptoms of intoxication in fish include violent twisting and corkscrew swimming, defecation and regurgitation, pectoral fin paralysis, caudal fin curvature, loss of equilibrium, quiescence, vasodilation, convulsions, and fatal respiratory failure.[27] An enzyme immunoassay, based on antibodies to PbTx-3, has been developed to detect these brevetoxins.[443,477] A micellar electrokinetic capillary chromatography (MEKC) detection method allows measurement of brevetoxin at trace levels and is 100-fold better than previous chromatographic or electrophoretic methods.[385]

Diarrhetic Shellfish Illness

Ingestion of shellfish contaminated with the dinoflagellates belonging to the genus *Dinophysis* (*Dinophysis fortii, Dinophysis acuminata, Dinophysis norvegica,* and *Dinophysis acuta*) or *Prorocentrum (Prorocentrum lima and Prorocentrum minimum)* causes diarrhetic shellfish poisoning (DSP). The lipid-soluble toxins accumulate in shellfish fatty tissues and the hepatopancreas of mussels. They exert their effects mainly on the small intestine, leading to diarrhea and degenerative changes of the absorptive epithelium.[282,452] Symptoms include rapid onset (30 minutes to 2 hours) of diarrhea, nausea, vomiting, abdominal pain, and chills. Rarely, symptoms are delayed up to 12 hours. The syndrome is self-limited and resolves after 2 to 3 days. From 1976 to 1982, DSP was diagnosed in at least 1300 persons in Japan. The period of greatest toxicity appears to be May to August. In 1981, more than 5000 cases were reported in Spain.[490] Other outbreaks have occurred in the Netherlands and Chile.[185] In 1993, a particularly bad episode occurred in Spain with unusual symptoms; analyses revealed a complex toxin profile with both PSP and DSP toxins present.[152]

DSP toxins include okadaic acid (OA), OA diolester, dinophysistoxins (DTX-1 to DTX-4), pectenotoxins, and yessotoxin.[23,279,450,473,490] OA was first isolated from a sponge *(Halichondria okadai)* in the Pacific.[418] It is a specific and potent inhibitor of protein synthesis and inhibits phosphatases A_1 and A_2 in vitro. OA induces diarrhea because it increases phosphorylation of proteins, which either controls the sodium secretion of intestinal cells or influences the permeability of cell membranes.[95,114] It is a potent tumor promoter in mouse cells and can act as a genotoxin.[137] Other DSP toxins exert various effects in experimental animals: pectenotoxins induce liver necrosis, and yessotoxins (from the Japanese scallop *Patinopecten yessoensis*) induce intracytoplasmic edema in cardiac muscle.[432,434] Minimal doses of OA and DTX-1 neces-

sary to cause DSP symptoms are 40 µg and 36 µg, respectively.[187] Metals (e.g., aluminum, copper, lead, mercury, cadmium) in concentration at or below acceptable levels in mussels synergistically increase the cytotoxicity of low concentrations of OA in cultured cells.[444]

Increasing incidences of phytoplankton blooms with a danger of toxin release have necessitated the search for new diagnostic methods that can detect toxin quickly and reliably. A variety of techniques, including a radioimmunoassay using antibodies raised in rabbits, a competitive ELISA, idiotypic anti-idiotypic competitive immunoassay, rapid tissue culture assays, and cytotoxicity assays, can identify the presence of OA.[88,99,259,386,448] A unified bioscreen for the detection of diarrhetic shellfish toxins and microcystins (as from blooms of the cyanobacteria *Microcystis aeruginosa*) uses capillary electrophoresis coupled with a liquid chromatography-linked protein phosphatase bioassay.[50] A protein phosphatase A_2 inhibition assay has been shown to be rapid, accurate, and reproducible; it can detect concentrations as low as 0.063 ng/mL in aqueous solutions and 2 ng/g in mussel digestive glands.[447] The Japanese quarantine standard is 200 ng of OA per gram of shellfish tissue. Four times this amount of toxin has been identified in northeastern Pacific Ocean mussels.

Amnestic Shellfish Poisoning

Domoic acid, the toxin responsible for amnestic shellfish poisoning (ASP), is an excitatory neurotransmitter first described in Japan in 1958 and isolated from the red algae *Chondria armata*.[424] The first documented human outbreak of poisoning with this compound was in 1987 from Prince Edward Island, Canada, when more than 150 people became ill after ingesting cultured blue mussels, *Mytilus edulis,* later found to be contaminated with domoic acid.[217,330,429,483] Four of these individuals died, and the clinical description of persistent memory impairment in many survivors prompted the nickname of amnestic shellfish poisoning.[140] The source of the toxin in these cases was found to be *Nitzschia pungens,* a pennate diatom that had been ingested by the mussels before humans ate them.[33,412] The mussels concentrate the toxin in the hepatopancreas.

Epidemics of domoic acid poisoning have been more prominent in other marine life, especially sea birds.[467,480,481] A large number of dead and distressed pelicans and cormorants were noted in Monterey Bay, California, in September 1991.[480,481] Autopsies performed on the dead birds demonstrated they had consumed large quantities of anchovies from the bay. Subsequent testing showed the anchovies contained high levels of domoic acid. This was the first report documenting the presence of domoic acid in the United States. Further studies of mussels taken during the same period from Monterey Bay also revealed the presence of domoic acid.[467] Water samples taken in the area identified significant quantities of the diatom *Pseudonitzschia australis,* which when grown in a laboratory environment were able to produce domoic acid.[155,480] Three species of *Pseudonitzschia* are now known to produce domoic acid.

Domoic acid has also been detected in Gulf shellfish (Gulf Coast oyster, *Crassostrea virginica*) and phytoplankton in the Gulf of Mexico, although no outbreaks of amnestic shellfish poisoning have been recorded in this region. The toxic *N. pungens* forma *multiseries* has also been confirmed in Korea; Japan; Oslofjord, Scandinavia; the northeastern and northwestern United States; eastern and western Canada; and eastern South America.[116]

In the fall of 1991, the latest reported epidemic of domoic acid poisoning occurred in Washington State.[235] More than 20 people who consumed razor clams were affected. Subsequent testing confirmed the presence of domoic acid in razor clams along the coasts of both Washington and Oregon, although mussels tested in these areas were virtually free of toxin. Dungeness crabs collected from these waters were also found contaminated with domoic acid.

Pathophysiology. After the 1987 epidemic of domoic acid poisoning, significant evaluation of the surviving victims was undertaken. Chemical analysis at various laboratories ruled out all other known toxic causes of the symptoms displayed by the patients.[483] Intraperitoneal injection of extracts of the implicated mussels into mice produced a syndrome characterized by reproducible scratching followed eventually by death.[330,429] The toxin was finally identified as domoic acid.

Domoic acid is a glutamate receptor agonist. After its discovery in 1958, it was investigated for potential use as an antihelminthic in children in Japan at doses of about 0.6 mg/kg, significantly lower than that ingested by affected individuals in Canada.[483] Domoic acid has also been tested for its insecticidal properties, with disappointing results.

Domoic acid is structurally related to kainic acid, a potent neurotoxic amino acid.[319,374,429,483] This group of compounds is excitatory and acts on three types of receptors in the central nervous system (CNS), with those in the hippocampus being the most sensitive. Domoic acid seems to work by activating kainate receptors in the brain more potently than kainic acid itself. The result of this stimulation is extensive damage to the hippocampus in victims of the poisoning, as well as less severe injury to portions of the thalamic and forebrain regions.[376,398,429]

It was estimated that the mussels implicated in the Canadian outbreak of ASP contained a total amount of domoic acid in excess of 6 kg, with most being concentrated in the digestive glands.[168,483] When the same toxic blue mussels were placed into tanks of clean water, they purged themselves of the toxin over several weeks. Other organisms known to produce domoic acid include the phytoplankton *Alsidium corallinum* and *Chondria armata*. Subsequent research suggests that other phytoplankton, such as *Amphora coffeaeformis,* can also produce domoic acid. Scientists continue to monitor shellfish and marine microorganisms to determine the presence of other sources.

Clinical Presentation. Rats injected with high concentrations of domoic acid develop hippocampal seizures and die within several days.[330] Surviving rats exhibit difficulty in remembering maze pathways they had previously encountered, signifying retrograde amnesia. In these same rats injected with domoic acid, prior maze learning skills are also not retained, suggesting an additional effect on anterograde memory.[330,429] These effects are similar to that of intraperitoneally injected kainic acid. Interestingly, pelicans reportedly poisoned by domoic acid containing anchovies in 1991 exhibited a scratching behavior similar to that demonstrated in laboratory rats after domoic acid injection.[307]

Humans involved in the Canadian epidemic of amnestic shellfish poisoning had initial symptoms of nausea, vomiting, abdominal cramps, and diarrhea 1 to 24 hours after ingestion.[330,429] Neurologic symptoms initiated by memory loss began within 48 hours after ingestion and progressed in some victims

to seizures, hemiparesis, ophthalmoplegia, and coma. Some victims displayed purposeless grimacing and chewing. Follow-up neuropsychological testing on affected patients displayed predominantly an anterograde memory disorder, with most other cognitive functions preserved.[330] The most severely affected individuals also had some retrograde amnesia. A labile blood pressure and cardiac arrhythmias were recorded in a few individuals. Elevations in blood urea nitrogen (BUN) and creatine phosphokinase were also noted in many victims and have been recorded in animals suffering domoic acid poisoning, possibly resulting from exertional myopathies or tremors.[330,481]

The onset of symptoms in victims of the Prince Edward Island epidemic ranged from 15 minutes to 38 hours, with the average approaching 5 hours.[330,429] Most fatalities occurred in the oldest victims, with postmortem findings suggesting neuronal loss or necrosis, accompanied by astrocytosis.[330] The most severe damage was to the hippocampus and amygdala, which are areas of the brain participating in memory functions. Lesions were also noted in the claustrum and the septal and olfactory regions. No lesions were found in the motor nuclei of the brainstem. Hippocampal lesions in victims at autopsy resembled those seen in the brains of animals injected with kainic acid.[330,376,398] A follow-up study done on patients from the Montreal area suggested that bronchial secretions became so profuse in the hours after mussel ingestion that half of the severely affected individuals required endotracheal intubation.[330] Pupillary dilation or constriction and piloerection were also common findings. Approximately 10% of the involved patients demonstrated either persistent memory loss or other neuropathies. Of those patients exhibiting neurologic toxicity, maximal effects were noted within the first 3 days after mussel ingestion, and maximal improvement in the neurotoxicity occurred in 24 hours to 12 weeks after ingestion.

Treatment. As with most other shellfish toxins, no antidote exists for amnestic shellfish poisoning. Based on the alleged mechanism of action of both domoic and kainic acid, it is possible that benzodiazepines may be beneficial in controlling some of the excessive hippocampal activity and seizures.[319,481] Animal studies have suggested a lowered mortality in groups in which benzodiazepines are used after domoic acid exposure. Government agencies continue to observe coastal areas for outbreaks of domoic acid toxicity and for the presence of phytoplankton known to produce the compound. Blooms of organisms such as *Nitzschia pungens* and *Pseudonitzschia australis* are closely monitored and reported.

POSSIBLE ESTUARY-ASSOCIATED SYNDROME

Pfiesteria piscicida is a toxic dinoflagellate that inhabits estuarine and coastal waters of the eastern United States and has been associated with fish kills and a human illness that has been labeled possible estuary-associated syndrome (PEAS). Since 1991, *P. piscicida* and other *Pfiesteria*-like species have been implicated in massive fish kills in estuaries of North Carolina, Maryland, and the Chesapeake Bay.[164,170,390] *P. piscicida* is responsible for a fish disease formally known as ulcerative mycosis. *Pfiesteria* is primarily a benthic organism but can exist in at least 24 different life stages. Fish swimming into an area with *Pfiesteria* maybe exposed to a toxin that is produced by the dinoflagellate. These fish develop characteristic ulcerative lesions and erratic swimming behavior. *Pfiesteria* have now been found in coastal waterways extending from Delaware to the Gulf Coast of Alabama.

Although it is not associated with seafood ingestion, PEAS is associated with seafood contact. The first report of adverse health effects in humans was described after an accidental laboratory exposure; investigators working with *Pfiesteria* developed respiratory and eye irritation, skin rashes, and cognitive and personality changes.[164] During the 1990s, commercial fishermen who were exposed to waterways with *Pfiesteria* species reported similar symptoms.[170,172,173,390] The route of exposure is unknown, although it is thought to be either by prolonged direct skin contact with toxin-laden water or via aerosols after breathing the air over areas where fish are dying from toxic *Pfiesteria*.

Individuals with high exposure complain of headache, skin lesions, skin burning on contact with water, eye irritation, upper respiratory irritation, muscle cramps, and neuropsychological symptoms, including increased forgetfulness and difficulties with learning and higher cognitive function.[172] No consistent physical findings or laboratory abnormalities have been found. When skin lesions appear, they are erythematous, edematous papules on the trunk or extremities and resolve within a few days to a week after exposure. Thorough neuropsychological testing has documented deficits in higher cognitive function and learning and functional memory.[172] The severity of the cognitive dysfunction was directly related to the degree of exposure. The exact nature of the neurocognitive deficit is unknown; however, rats exposed to water containing *Pfiesteria* toxins have shown significant learning impairments.[257,258] Deficits may be expected to improve significantly within 3 to 6 months after cessation of exposure to affected waters.[417] The natural history of PEAS is improvement in most symptoms without treatment; however, cholestyramine has been successfully used in five patients with persistent symptoms.[391]

Diagnosis of PEAS is difficult because the specific causal toxins have not yet been identified, and a biomarker of exposure has not been developed. Current recommendations for diagnosis include (1) development of symptoms within 2 weeks after exposure to estuarine water, (2) memory loss or confusion of any duration, or three or more symptoms from the complex as described in the preceding paragraph, and (3) no other cause for symptoms identified.[79] A multiplex PCR is being developed for rapid identification of *P. piscicida* and other toxic *Pfiesteria* species.[295] PEAS is not infectious and has not been associated with eating fish or shellfish caught in waters where *P. piscicida* has been found. Brief, direct water contact, including swimming, has not been associated with symptoms. No deaths have been associated with exposure to *Pfiesteria* species. People should avoid areas with large numbers of diseased, dying, or dead fish.

Haff Disease

Haff disease is a syndrome characterized by severe muscle pain and rhabdomyolysis after consuming fish. It was first described in 1924 around the shores of Königsberg Haff, a bay on the Baltic Sea.[500] Further outbreaks have occurred in Sweden and Russia.[40,392,401] Twelve cases have been reported in the United States, all associated with eating buffalo fish *(Ictiobus cyprinellus)*, a bottom-feeding species found in the Mississippi River and its tributaries. Haff disease is most likely the result of a heat-

stable toxin in blue-green algae that is eaten by fish; however, the toxin is currently unidentified.[77]

Haff disease presents as generalized muscle pain and tenderness, rigidity, weakness, and rhabdomyolysis. Tachycardia, hypertension, tachypnea, and a drop in temperature can also occur. Elevated serum creatine kinase occurs with leukocytosis, myoglobinuria, and elevation of lactate dehydrogenase and other muscle enzymes. Symptoms generally appear approximately 18 hours after eating fish (range, 6 to 21 hours).[77] Pathologically, there is neuromyodystrophy with necrosis in motor neurons of the brain and spine, coagulation necrosis of muscle, and myoglobinuric nephrosis. Treatment includes large volumes of intravenous fluids and diuretics to prevent renal failure from myoglobin toxicity. Diagnosis is based on clinical presentation, laboratory data, and food history.

Blue-Green Algae

Blue-green algae are worldwide freshwater cyanobacteria that may proliferate rapidly in a bloom, discoloring the surface of the water and spoiling its odor and taste. Terrestrial, fresh, brackish, and sea-water cyanobacteria produce toxins that are acute and chronic hazards to human and animal health and are responsible for isolated, sporadic animal fatalities each year. Typical algal species include *Microcystis aeruginosa, Anabaena flos-aquae, Nodularia spumigena, Nostoc, Oscillatoria agardhii,* and *Aphanizomenon flos-aquae.*[94,132,308,360]

During conditions of a bloom (warm stagnant water, frequently enhanced by phosphorus and nitrogen fertilizers), the toxins are concentrated enough to become a significant hazard to wild and domestic animals and have been responsible for the death of livestock and dogs.[94,128,191,310,453] In most species of toxic cyanobacteria, the toxins are cyclic heptapeptides called microcystins, or cyanoginosins. More than 60 cyanobacterial toxins have been isolated from blue-green algae.[94,396] The toxins are of multiple configurations and include alkaloids, polypeptides, and lipopolysaccharides (endotoxins).[406]

Anatoxin-a and homoanatoxin-a are potent nicotinic agonists that act as postsynaptic, depolarizing neuromuscular blocking agents. These toxins along with saxitoxin cause animals to collapse quickly from neuromuscular paralysis, with features of staggering, muscle fasciculations, gasping, and convulsions.[190] Anatoxin-a(s) (for second anatoxin-a) is an anticholinesterase that causes demonstrable cholinergic toxicity in animals.[68,190] Anatoxin-a and anatoxin-a(s) are both derived from *Anabaena flos-aquae*. Nodularins and microcystins cause hepatotoxicosis. Cylindrospermopsin is a protein synthesis inhibitor and induces necrotic tissue injury of multiple organs. Cyanobacterial lipopolysaccharide endotoxins are responsible for gastroenteritis and skin irritations.[94] In mice, administration of microcystin-LR causes rapid hepatocellular necrosis with hemorrhagic shock.[103,436]

Human exposure to blue-green algae blooms has resulted in allergic reactions, skin irritations, gastroenteritis, pulmonary consolidation, and liver damage.[94,331] A person who swims through a bloom may suffer local effects, such as conjunctivitis, facial swelling, or papulovesicular dermatitis. Ingestion of contaminated water causes a dysenteric diarrhea, with green slimy stools. This may be associated with elevation of γ-glutamyl-transpeptidase and alanine aminotransferase. Inhalation of toxins is a probable exposure route; microcystin-LR and anatoxin-a cause significant toxicity in mice via intranasal aerosol exposure.[140] In 1996, more than 50 people with associated liver damage died at a hemodialysis clinic in Brazil. Microcystins are thought to have been present in the water used for hemodialysis.[94,123]

Treatment is supportive in humans and animals. Cyclosporine A has been shown to inhibit the fatal effects of microcystins administered to mice. In humans, no specific treatment is recommended other than fluid and electrolyte supplementation as needed, as all related afflictions appear to be self-limited.

OTHER SHELLFISH AND INVERTEBRATE POISONINGS

Callistin Shellfish Poisoning

The Japanese *Callista* clam *(Callista brevisiphonata)* is toxic during the spawning months of May to September, at which time cholinergic compounds in the ovaries are increased. The intoxication resembles cholinergic crisis, with both muscarinic and nicotinic components. Within an hour of ingestion of the heat-stable toxin, generalized pruritus, urticaria, erythema, facial numbness and paralysis, hypersalivation, diaphoresis, fever, chills, nausea, vomiting, diarrhea, bronchorrhea, bronchospasm, and constrictive dyspnea develop.[30] Therapy is supportive, and recovery is usually complete within 2 days. In severe cases of cholinergic crisis, particularly with marked bradycardia, atropine (0.5 mg or more intravenously every 5 minutes, titrated to dry secretions, with adequate ventilation) may be administered.

Venerupin Shellfish Poisoning

The Japanese lake-harvested oyster *(Crassostrea gigas)* and clam *(Tapes semidecussata)* occasionally feed on toxic dinoflagellate species of the genus *Prorocentrum,* posing the greatest risk during the months of December through April.[30,184] The heat-stable toxin induces the rapid onset of gastrointestinal distress, headache, and nervousness, which are followed over 48 hours by hepatic dysfunction, manifested by elevation of liver enzymes, leukocytosis, jaundice, and profound coagulation defects. Delirium and coma may ensue, and death occurs in 33% of victims. Therapy is supportive and based on symptoms. Any victim who shows the early symptoms of gastroenteritis should be placed on a low-protein diet and observed for 48 to 72 hours for signs of liver failure. There is not yet clinical experience with exchange transfusion, chemotherapy, hemoperfusion, or liver transplantation in the management of profound liver failure associated with this disorder.

Tridacna Clam Poisoning

Giant clams of the species *Tridacna maxima* are eaten in French Polynesia.[30] This species can cause a poisoning characterized by nausea, vomiting, diarrhea, paresthesias, tremor, and ataxia. Severe cases can be fatal. The toxin appears to be concentrated in the mantle and viscera of the clam. Therapy is supportive.

Whelk Poisoning

In Japan, poisoning has followed ingestion of mollusks of the genera *Neptunea, Buccinum,* and *Fusitriton* (whelks, or ivory shells). The toxin is located in the salivary glands and has been characterized as tetramine.[14] Tetramine (trimethylammonium) is a naturally occurring quaternary ammonium compound that has been identified in anemones, gorgonians, jellyfishes, and mollusks.[13] Symptoms include headache, dizziness, nausea,

vomiting, blurred vision, and dry mouth. No fatalities have been reported. Therapy is supportive.[30]

Ivory Shell Poisoning

Human intoxications have followed consumption of the ivory shell, *Babylonia japonica,* which is widely distributed along the coastline of Japan. The toxin, surugatoxin, located in the midgut of the animal, is reputed to be produced by a gram-negative bacterium on which the snail feeds. Surugatoxin and IS (ivory shell) toxins appear to have autonomic ganglionic blocking action. Symptoms include abdominal pain, diarrhea, nausea, vomiting, oral paresthesias, syncope, and seizures.[185] Tetrodotoxin has also been identified in *B. japonica.*

Abalone Poisoning

Abalone poisoning follows ingestion of the viscera of certain Japanese abalone (tsunowata, or tochiri), particularly from the Island of Hokkaido, where *Haliotis discus* and *Haliotis sieboldi* are found. Symptoms include severe urticaria, erythema, pruritus, edema, and skin ulceration. The reaction appears to be of a photosensitive nature, as the lesions are confined to areas of ultraviolet exposure. The toxin may be derived from chlorophyll contained in the seaweeds on which the abalone feed.[30] Therapy is supportive.

Cephalopod Poisoning

In certain areas of Japan, intoxications have resulted from ingestion of squid and octopus. Symptoms develop within 10 to 20 hours and consist of nausea, vomiting, diarrhea, abdominal pain, headache, weakness, paralysis, and seizures. Although most victims recover within 48 hours, deaths have occurred.[30] Therapy is supportive.

Sea Cucumber Poisoning

Sea cucumbers are eaten throughout Asia and in some Pacific islands, where they are known as trepang, sea slugs, cucumbers, erico, or hai shen. Gastroenteritis is induced by saponins of the triterpinoid variety, such as holothurin. The disorder consists chiefly of abdominal pain, nausea, and diarrhea and is self-limited.

Sea Hare Poisoning

Sea hares are marine gastropod mollusks prevalent in certain South Pacific waters, including Fiji. *Aplysia* species have been considered to be toxic since Roman times. *Aplysia juliana* secretes an antibacterial and antineoplastic protein found in the water-soluble fraction of a fetid secretion lethal to crabs. Human poisoning has been reported after ingestion of *Dolabella auricularia* ("veata").[365,404] The symptoms begin approximately 30 minutes after eating and include prickling skin sensation, vomiting, diarrhea, shaking, tremors, fasciculations, arthralgias, dyspnea, visual disorientation, altered sensorium, and fever. The course of illness may exceed a week. It has been suggested that sea hare poisoning in humans might be a form of subacute organobromine intoxication.

Ingestion of the sea hare *Aplysia kurodai* was associated with acute liver damage with sustained elevations of aminotransferases. Microscopic findings in a liver biopsy specimen revealed characteristic apoptotic hepatocytes accompanied by mitotic hepatocytes. Bioactive substances in the sea hare might induce such apoptosis of hepatocytes in the liver.[366]

Anemone Poisoning

In the South Pacific, ingestion of the green or brown anemones *Radianthus paumotensis* or *Rhodactis howesii* (mata-malu samasama) has been associated with severe illness and death. Accidental deaths generally involve small children, whereas adults may be the unfortunate recipients of improperly cooked anemone or even may be intentionally stricken in acts of suicide. The toxic substances are found in the nematocysts and the tentacular tissues. Anemones have been used for criminal purposes in the South Pacific.[30] *Physiobranchia douglasi* is poisonous eaten raw but is reputedly safe if cooked.[185]

Ingestion of the raw anemone induces altered mental status within 30 minutes, often immediately after the ingestion. The victim becomes agitated or confused, delirious, and then comatose. Other symptoms reported include fever, seizures, myalgias, abdominal pain, respiratory failure, hypotension, and death. Contact with the skin, particularly mucous membranes, is extremely painful, with rapid inflammation and vesiculation.

Treatment is symptomatic and supportive. Because of the rapid onset in symptoms, the rescuer must be prepared to provide advanced life support within the first hour after the ingestion.

A toxic protein has been isolated from the sea anemone *Urticina piscivora.* It is a potent cardiac stimulatory protein and a potent hemolysin on erythrocytes of the rat, guinea pig, dog, pig, and human, causing toxicity at concentrations as low as 10^{-10} M.[93]

Crab Poisoning

Human intoxications have followed ingestion of crabs in many Indo-Pacific islands. Most of the toxic crab species are members of the family Xanthidae and include the genera *Demania, Carpilius, Atergatis, Platypodia, Zosimus, Lophozozymus,* and *Eriphia.* Clinical symptoms develop 15 minutes to several hours after ingestion and include nausea, vomiting, diarrhea, perioral and extremity paresthesias, ataxia, aphasia, respiratory distress, altered mental status, coma, and rapid death.

A number of toxins have been isolated from crab species, and there is a marked similarity to PSP and TTX poisonings. Saxitoxin, neosaxitoxin, and gonyautoxins have been isolated from crab species in Okinawa and from *Eriphia sebana* and *Atergatis floridus* off Australian coral reefs.[268,269,270,271] Tetrodotoxin and palytoxin have also been characterized from poisonous crabs.[9] In Thailand, tetrodotoxin was responsible for an epidemic involving 71 persons (two died) who ate the toxic eggs of the horseshoe crab *Carcinoscorpius rotundicauda.*[225] The poisonous mosaic crab *Lophozozymus pictor* from the Indo-West Pacific region has caused several fatalities in the Philippines and Singapore. The toxins were concentrated in the gut and hepatopancreas, whereas the muscle was less toxic. Captive crabs lose toxicity almost completely by 24 days.

Coconut crab *(Birgus latro)* poisoning is manifested as nausea, vomiting, headache, chills, myalgias, and exhaustion, with occasional deaths. Asiatic horseshoe crabs *(C. rotundicauda)* are eaten in Thailand, where they cause mimi poisoning. Symptoms include nausea, vomiting, diarrhea, abdominal cramps, dizziness, palpitations, weakness, lower extremity paresthesias, aphonia, perioral burning, pharyngitis, sialorrhea, syncope, paralysis, and death. Again, the toxin appears highly similar to saxitoxin.[85]

Crab lung has followed the aspiration of tiny fragments of North American blue crab shells into the lung, necessitating

removal with fiberoptic bronchoscopy. The diagnosis of occult aspiration should be considered in anyone with an unexplained cough who has recently consumed cracked crab, particularly while intoxicated.

Freshwater crabs are a potential source of human paragonimiasis, a parasitic disease that was prevalent in Asia until the 1960s.[89] Paragonimiasis usually causes pulmonary disease with a productive cough and bloody sputum. CNS involvement has also been reported.[340] The disease is contracted by eating raw crab infected with the metacercariae of *Paragonimus* species. Areas known to be endemic are Vietnam, China, Japan, Korea, Ecuador, and Liberia.[7,89,101,309,340,363,458]

BACTERIAL AND VIRAL PATHOGENS IN SEAFOOD

Shellfish, particularly bivalve mollusks, contaminated with bacteria or viruses are implicated more than any other marine animal in seafood-related human illness.[267,333] As filter feeders, bivalve mollusks filter large quantities of water unselectively to gather plankton and extract oxygen, which allows concentration of bacteria and viruses (along with biologic toxins, pesticides, industrial chemicals, radioactive wastes, toxic metals, and hydrocarbons). They are sessile invertebrates that generally inhabit shallow waters close to shore and pollution sources. Standard depuration in purified (with ultraviolet light or ozone) water for 48 to 72 hours may not significantly reduce these contaminants, nor does it appear to remove viruses.[220,373] Viruses and naturally occurring bacteria that cause disease and death are of great concern because they are so common. The greatest risk of death from consumption of raw shellfish is among people with underlying health conditions.

Bacteria Associated with Fecal Contamination

Bacterial pathogens associated with fecal contamination have accounted for only 4% of the shellfish-associated gastroenteritis outbreaks in the United States.[353] Important bacteria include *Salmonella, Shigella, Campylobacter, Yersinia, Listeria, Clostridium, Staphylococcus,* and *Escherichia coli.*[210] The incidence of enteric bacteria in seafood has been greatly reduced with the development of surveillance programs and standards to minimize fecal contamination.[353] However, infections continue to occur in other parts of the world.

Salmonella typhi was the most common cause of bacteria-associated shellfish disease until the 1950s. The incidence of *S. typhi* in shellfish has dramatically declined and the risk of infection in the United States is very low.[353,472] However, *S. typhi* is still responsible for outbreaks of illness in other countries.[410,411] Nontyphoidal *Salmonella* species, including *Salmonella paratyphi* and *Salmonella enteritidis,* have been detected in shrimp and bivalves. Eight *Salmonella* shellfish infections were reported in the United States between 1984 and 1993, and *S. enteritidis* phage type 19 was responsible for an outbreak of infections from cockles in the United Kingdom.[174,474] *Shigella* was responsible for 111 shellfish cases and four outbreaks in the United States.[353] *Shigella* has a low infectious dose and a long survival time in clams and oysters.

Campylobacter species have been isolated from shellfish, but their role in seafood infections is not known.[472] *Listeria monocytogenes* has been identified in high rates in isolates from fresh and processed fish and shellfish.[209] *Listeria* seafood-borne infections are probably underreported in the United States. *Yersinia enterocolitica* has also been identified in fish and shellfish; however, most *Yersinia* infections are not associated with seafood.[210,353] *Escherichia coli* has not been an important source of seafood-related illness, although *E. coli* is found in shellfish.[60]

Viruses Associated with Fecal Contamination

Most infections associated with consumption of shellfish are viral in origin. Over 120 enteric viruses can be found in human sewage. These viruses can produce a variety of symptoms, including gastroenteritis, meningitis, paralysis, myocarditis, and hepatitis. Compared with other food-borne illnesses, those caused by viruses are less severe, and they are seldom fatal. Viruses that have been isolated from seafood include hepatitis (A, non-A, and non-B), enteroviruses (echovirus, poliovirus, coxsackie A and B), adenoviruses, rotaviruses, and, most commonly, small round viruses (Norwalk, calicivirus, Snow Mountain agent, and small rounded structured viruses [SRSVs]).[333]

Harvest areas are surveyed and closed for fecal contamination. However, the relative absence of fecal coliform bacteria in areas of shellfish harvesting does not indicate freedom from viral contamination. Outbreaks of Norwalk virus and calicivirus have been caused by oyster harvesters discharging their sewage overboard.[76] In addition, shellfish depuration processes that eliminate bacteria do not necessarily remove viral contaminants.[373] Steamed clams probably pose a significant risk because household cooking techniques are often insufficient to kill viruses. Although it takes 4 to 6 minutes of pressure-cooker steaming for the internal temperature of soft-shell clams ("steamers") to reach 100° to 106°C (212° to 222.8°F), it requires only 60 seconds for the shells to open, at which point they may appear cooked.[234] Poliovirus can survive (7% to 13%) in oysters that are steamed, fried, baked, or stewed.[118]

Methods using PCR amplification of target viral genomes provide a rapid, specific, and sensitive test for detection of viruses.[17,18] The amplification of viral ribonucleic acid (RNA), DNA, and complementary DNA (cDNA) has shown a high prevalence of human viruses that would not be detected by the use of classical techniques.[332] PCR has been used to detect the presence of hepatitis A virus in oysters and scallops during an outbreak, and SRSV, adenovirus, enterovirus, and Norwalk virus in shellfish.[17,18,20,100,254,332,373]

Hepatitis Viruses. Oysters, mussels, and clams harvested from waters contaminated with raw sewage are the most frequent cause of food-borne viral hepatitis A. Often, there is a long incubation period of 2 to 8 weeks, so it is common for hepatitis A to occur 3 to 4 weeks after gastroenteritis following consumption of shellfish.[353] Symptoms include fever, jaundice, nausea, and abdominal pain, whereas diarrhea is rare. Treatment is supportive.[45]

Enteroviruses. Enteroviruses are commonly isolated from marine water and shellfish. In the United States, up to 63% of shellfish in areas closed for harvesting, and up to 40% of shellfish in areas open for harvesting were positive for enteroviruses.[356] In contaminated waters in Venezuela, 40% of harvested shrimp contained enteroviruses.[53] Enterovirus outbreaks have not been characterized, and the impact of enteroviruses on public health is not fully appreciated.[267]

Small Round Viruses. Small round viruses include Norwalk virus, calicivirus, Snow Mountain agent, and SRSV. These viruses are a major cause of shellfish-associated gastroenteritis.[76,81,122,244,407,412] Caliciviruses are small, single-stranded RNA viruses and have been responsible for a number of oyster-related gastroenteritis outbreaks in Louisiana.[76,81] Norwalk virus has been located both in and outside the shellfish alimentary tract and is poorly depurated using conditions favorable for *E. coli* depuration. Reverse transcription PCR (RT-PCR) assay can easily detect Norwalk virus in contaminated water, shellfish, and stool from infected people.[244,373] The virus may be excreted in the feces of food handlers and harvesters for 48 hours after recovery from the infection.[46] Symptoms including nausea, vomiting, fever, abdominal cramps, and nonbloody diarrhea appear 24 to 48 hours after ingesting contaminated shellfish and resolve over 1 to 2 days. Antibodies to Norwalk virus have been measured in serum of patients with gastroenteritis, and electron microscopy or RT-PCR can detect virus in stool.[244] Treatment is supportive, and complications are rare.

Vibrio Poisoning and Septicemia

Over the last few decades, naturally occurring bacteria, particularly those belonging to the family Vibrionaceae, are becoming a more important cause of shellfish illness.[214] *Vibrio* organisms can cause gastroenteric disease and soft tissue infections after the consumption of raw shellfish that can lead to bacteremia and death, particularly in immunocompromised hosts. Between 1988 and 1996, 422 infections from *Vibrio vulnificus* were reported to the CDC; 61% of patients with primary septicemia died.[383] In 2002, 452 patients were reported to the CDC with noncholera *Vibrio* infections. Of these, 45% were hospitalized and 11% died.[463] *Vibrio parahaemolyticus* was found in 35% of the victims, but *V. vulnificus* was found in 73% of the patients who died.[463]

Vibrio species may be the most virulent halophilic organisms that flourish in the marine environment. In general, they are not associated with fecal contamination, so surveillance methods mentioned earlier for bacteria and viruses do not correlate with the presence of *Vibrio*. *Vibrio* species proliferate in warmer water; infections seem to cluster during the summer months, which may be related to increased numbers of people at the seashore.[293] *Vibrio* species are mesophilic organisms and grow best at temperatures of 24° to 40°C (75.2° to 104°F), with essentially no growth below 8° to 10°C (46.4° to 50°F).[60] They can grow in brackish waters, and they require less sodium for maximal growth than other, more fastidious marine organisms, a factor that allows explosive reproduction in the 0.9% saline environment of the human body. *V. parahaemolyticus* has also been identified in freshwater habitats.[22]

Gastrointestinal illness has been associated with toxigenic O group 1 *Vibrio cholerae,* non-O1 *V. cholerae, V. parahaemolyticus, Vibrio fluvialis, Vibrio mimicus, Vibrio hollisae, Vibrio furnissii, Vibrio alginolyticus,* and *V. vulnificus.*[63,171,343,347,383,421,474] Septicemia, with or without an obvious source, has been attributed to infections with non-O1 *V. cholerae, V. parahaemolyticus, V. alginolyticus, V. vulnificus, V. hollisae,* and *Vibrio metschnikovii.**

Whenever a *Vibrio* species is suspected, the microbiology laboratory must be alerted to use an appropriate selective culture medium for stool cultures, such as thiosulfate-citrate-bile salts-sucrose (TCBS) agar or Monsur taurocholate-tellurite-gelatin agar.[273,294] Pathogenic *Vibrio* species generally grow on MacConkey agar. The stool specimen should be collected if possible within the first 24 hours of illness and before the administration of antibiotics; specimens should not be allowed to dry. The specimen may be transported in the semisolid transport medium of Cary and Blair; buffered glycerol-saline is not satisfactory because glycerol is toxic to vibrios. Tellurite-taurocholate-peptone broth is adequate. All *Vibrio* species grow in routine blood culture mediums and on nonselective mediums, such as blood agar. New, rapid PCR tests are available for detection of *V. vulnificus* at the point of harvest.[62,323]

Key characteristics that aid in the separation of *Vibrio* species from other medically significant bacteria *(Enterobacteriaceae, Pseudomonas, Aeromonas, Plesiomonas)* include the production of oxidase, fermentative metabolism, requirement of sodium chloride for growth, and susceptibility to the 0/129 vibriostatic compound.[224] Species that cannot be identified in the hospital microbiology laboratory may be referred to a state laboratory or the CDC. Many sensitive and reliable PCR methods are now used to detect various strains of *Vibrio* species in oyster tissue and water samples.[37,257,457]

Vibrio Vulnificus. *V. vulnificus* is a free-living, motile, halophilic, gram-negative bacillus. It causes septicemia, gastroenteritis, and wound infections that occur 12 hours to 7 days after ingestion of contaminated raw or undercooked seafood, particularly raw oysters. Persons with compromised immune systems and liver disease are at a significantly higher risk for fatal septicemia. Necrotizing fasciitis and myositis have also been reported after *V. vulnificus*–contaminated seafood ingestion.[147] *V. vulnificus* has a rapid onset and the highest mortality rate of any of the *Vibrio* species. It is naturally present in marine environments, has a worldwide distribution, and is found throughout the United States.[353,383]

The growth of *V. vulnificus* is favored in waters with an intermediate salinity of 5 to 25 parts per thousand. The optimal temperature for its growth (doubling time, 15 minutes) is 35°C (95°F).[301] Below 10°C (50°F), it enters a nonculturable state and is viable but ceases to replicate.[318] The *V. vulnificus* count in the marine environment and in shellfish increases and peaks during the summer months, as does the incidence of *V. vulnificus* infections.[200]

V. vulnificus may appear as one of two colonial morphotypes: opaque and virulent, or translucent and less virulent. The opacity of the colony of the virulent morphotype is caused by an acidic mucopolysaccharide capsule. This capsule increases the resistance of the organism to the bactericidal activity of human serum and to phagocytosis and thus renders the organism more virulent. At extremely low frequency, some strains can shift between unencapsulated and capsulated serotypes.[493]

Growth of encapsulated isolates is improved in the presence of iron, but they are unable to use transferrin-bound iron. In patients with iron overload and transferrin saturation greater than 75%, free iron is available for use. Additionally, virulent isolates can use the iron in hemoglobin and hemoglobin-haptoglobin complexes.[57,59,498] *V. vulnificus* can bind specifically to human intestinal cells and quickly induce cytotoxic effects.[246] In vivo studies show that 4 hours after inoculation into the duodenum, the organism is found in the systemic circulation via bacterial translocation.[200] The severity of *V. vulnificus* infections is related to both bacterial characteristics (as just described) and

*References 1, 48, 83, 171, 216, 238, 239, 247, 272, 383.

host factors. In patients with liver disease, chronic cirrhosis, and alcoholism, portal hypertension allows shunting of the organism around the liver. These patients also have reduced phagocytic activity, chemotaxis, opsonization, and complement levels, thus promoting the virulence of *V. vulnificus*.[342] Persons with high serum iron levels (from chronic cirrhosis, hepatitis, thalassemia major, or hemochromatosis) are at increased risk for infection.[59,247,462] Any individual with impaired immunity (e.g., malignancy, human immunodeficiency virus [HIV] infection, diabetes, long-term corticosteroids use) is at greater risk for fulminant bacteremia.[419] Based on the prevalence of predisposing diseases in the general population, 3.8% of the general population is at risk of having *V. vulnificus* septicemia if they acquire this organism.[109]

Clinical Presentation. There are two clinical syndromes of *V. vulnificus* infection: primary septicemia and wound infection. The wound infection syndrome consists of flulike malaise, fever, vomiting, diarrhea, chills, hypotension, and early skin vesiculation that evolves into necrotizing dermatitis, with vasculitis and myositis (see Chapter 75). Primary septicemia occurs when *V. vulnificus* is acquired through the gastrointestinal tract. Blood cultures are positive for the organism in 97% of patients. Septic shock, disseminated intravascular coagulation, and death may occur.[462] The mortality of patients with primary septicemia is 56% and increases to 92% when septic shock occurs.[198]

Gastroenteritis was previously thought to exist as an isolated entity in 10% of cases. However, it is more likely that other enteric pathogens are the causal agents and that *V. vulnificus* illness has been erroneously attributed to the asymptomatically carried organism.[213]

V. vulnificus is also implicated in other infectious presentations, including meningitis, spontaneous bacterial peritonitis, corneal ulcers, epiglottitis, osteomyelitis, rhabdomyolysis, endocarditis, and infections of the testes and spleen.[117,136,232,288,445,455,462,479] *V. vulnificus* endometritis has been reported after an episode of intercourse in the water of Galveston Bay, Texas.[439]

Treatment. Early recognition of *V. vulnificus* infection is essential for effective treatment. Blood and wound cultures should precede immediate and aggressive antibiotic and supportive treatment. Current recommendations include doxycycline (100 mg intravenously every 12 hours) combined with ceftazidime (2 g intravenously every 8 hours) or ciprofloxacin (400 mg intravenously every 12 hours).[160] Other antibiotics that have been suggested include imipenem/cilastatin, trimethoprim/sulfamethoxazole, carbenicillin, tobramycin, gentamicin, and many third-generation cephalosporins. Supportive care includes crystalloid and pressor agents for hypotension.

Vibrio Parahaemolyticus. *V. parahaemolyticus* is a halophilic gram-negative rod that can cause gastroenteritis when consumed in raw or partially cooked seafood. It has been reported in temperate, subtropical, and tropical coastal regions.[34,78,189,283] The organisms are found in marine and estuarine waters along the entire coastline of the United States. Their concentration in seawater increases with rising water temperature and corresponds with an increased incidence of clinical disease in warm summer months when the organism is commonly found in zooplankton.[198] The optimal growth temperature of *V. parahaemolyticus* is 35° to 37° C (95° to 98.6° F), but it has also been isolated from colder waters. In the largest reported outbreak in North America of culture-confirmed *V. parahaemolyticus* infections, during July and August 1997 in the Pacific Northwest, 209 persons became ill and one person died after eating raw or undercooked oysters.[78] *V. parahaemolyticus* has been recovered at frequencies up to 25% in frozen peeled shrimp.[478]

Ingestion of raw or partially cooked *V. parahaemolyticus*–contaminated shrimp, oysters, crab, or fish is followed in 6 to 76 hours by explosive diarrhea, nausea, vomiting, headache, abdominal pain, fever, chills, and prostration. In immunocompetent persons, *V. parahaemolyticus* causes mild to moderate gastroenteritis with a mean duration of illness of 3 days. Serious illness and death can occur in persons with underlying disease (preexisting liver disease, diabetes, iron overload states, or a compromised immune system).[238] The stools may contain blood and classically demonstrate leukocytes on methylene blue staining. The syndrome generally resolves spontaneously in 24 to 72 hours but may be of severity sufficient to cause significant fluid and electrolyte depletion. Stool cultures should be obtained before the initiation of antibiotic therapy. Panophthalmitis requiring enucleation occurred in a man who suffered a corneal laceration.[420] A course of oral ciprofloxacin, trimethoprim/sulfamethoxazole, or tetracycline may shorten the course of severe gastroenteritis.

Vibrio Mimicus. *V. mimicus* is a motile, nonhalophilic, gram-negative, oxidase-positive rod with a single flagellum. It can be distinguished from *V. cholerae* by its inability to ferment sucrose, inability to metabolize acetylmethyl carbonyl, sensitivity to polymyxin, and negative lipase test.[107] Multiple toxins are produced by *V. mimicus*, including cholera-like toxin, enterotoxin, and hemolysin.[343,387,475] *V. mimicus* causes a syndrome of gastroenteritis (diarrhea, nausea, vomiting, abdominal cramps, fever, and headache) after the ingestion of raw oysters, crawfish, crab, or shrimp. It was identified by PCR in 11 individuals with gastroenteritis from eating raw turtle eggs.[66] Nonfatal bacteremia resulting from *V. mimicus* has been reported.[239] The median incubation period is 24 hours, with delayed diarrhea noted up to 3 days after ingestion of contaminated seafood. Isolates are sensitive to tetracycline, ciprofloxacin, and norfloxacin.[293]

Vibrio Alginolyticus. *V. alginolyticus* can cause gastroenteritis in immunocompetent individuals and bacteremia in immunosuppressed patients.[272,347] More commonly, it is implicated in soft tissue infections (such as those caused by coral cuts or surfing scrapes), sinusitis, and otitis media and externa.[153,272,348,446]

Vibrio Cholerae. In developing countries, cholera caused by toxigenic *V. cholerae* is a major public health problem. The last several cholera pandemics were caused by the consumption of fecally contaminated water.[482] *V. cholerae* is commonly linked to ingestion of raw or inadequately cooked mollusks and crustaceans and is responsible for the third highest number of shellfish-related illnesses behind other noncholera *Vibrio* species and Norwalk virus.[224,474] Toxigenic O group 1 *V. cholerae* infections are associated with a secretory, profuse watery diarrhea, nausea, and vomiting. Because the stool is virtually isotonic,

large amounts of fluid and electrolytes are lost, leading to rapid dehydration, shock, acidosis, and renal failure. The treatment consists of aggressive intravenous and oral fluid replacement. In an outbreak in Italy, all strains were resistant to tetracycline but the patients responded to ciprofloxacin.[276] Untreated, the disease remits in 3 to 8 days.

Nontoxigenic, non-O1 *V. cholerae* strains cause gastroenteritis and septicemia.[200] Self-limited (24 to 48 hours) nausea, vomiting, abdominal cramping, and invasive diarrhea with blood and fecal leukocytes are typical. Spontaneous non-O1 *V. cholerae* bacteremia and peritonitis have been reported in patients with cirrhosis after eating raw oysters.[266,334,355] Meningitis and death have also been associated with non-O1 *V. cholerae*.[78,238]

Other Vibrios. *V. metschnikovii* has caused bacteremia in a patient with cholecystitis; the authors postulated that it may have been associated with long-term carriage after seafood ingestion.[216] *Vibrio cincinnatiensis* has caused meningitis; a relationship to foreign travel, seawater exposure, or seafood ingestion has not been established.[48] *V. fluvialis,* previously designated as enteric group EF-6 or group F, is common in the marine environment. It causes diarrheal disease associated with vomiting, abdominal pain, dehydration, and fever.[421] Fatal gastroenteritis has been reported.[239] This bacterium can be mistaken in the microbiology laboratory for *Aeromonas hydrophila,* from which it can be distinguished by growth in 60 to 70 parts per thousand sodium chloride solution. *V. hollisae* and *V. furnissii* have both been linked to gastroenteritis after seafood ingestion, and *V. hollisae* has been associated with septicemia.[1,164]

Prevention

The Interstate Shellfish Sanitation Conference, a shellfish-industry group, has sought to decrease the number of contamination-related illnesses by education of the public, limiting harvesting to certain periods of the year, facilitating rapid refrigeration of the shellfish after harvest, and studying postharvest treatments to prevent bacterial growth.[97] Recently, postharvest high-pressure processing of oysters has demonstrated significant reductions in *V. vulnificus* levels but variable results in other *Vibrio* species; further studies are ongoing.[97] Persons who are immunosuppressed or chronically ill, particularly with hepatic insufficiency, should not eat raw or partially cooked shellfish. All seafood should be cooked thoroughly, protected from cross-contamination after cooking, and eaten promptly or stored at temperatures above 60°C (140°F) or below 4°C (39.2°F) to prevent proliferation of *Vibrio* species.

Botulism

Botulism is a paralytic disease caused by the potent natural toxin of *Clostridium botulinum*. Seven toxins (A to G) have been identified, but only A, B, E, F, and G cause human illness.[403] Seafood-related botulism can be caused by raw, parboiled, salt-cured, or fermented meats from marine mammals (seal, walrus, whale) or fish products (particularly salmon and salmon roe).[194] Toxin type E spores are found in mud and sediment in northern coastal areas and inland lakes, accounting for the prevalence of type E toxin in fish-borne botulism (although types A and B may also be involved). Improperly preserved (smoked, dried, or canned) foods are at high risk for *C. botulinum* toxin proliferation. The technique of hanging meat for decomposition (flavor and texture improvement) supports growth of the nonproteolytic, psychrotolerant forms of *C. botulinum,* which may grow at temperatures as low as 4°C (39.2°F).[194]

In the last 4 decades in the United States, over 10% of outbreaks of food-borne botulism have been related to the consumption of fish. Using quantitative PCR analysis, the prevalence of *C. botulinum* type E gene was 10% to 40% in raw fish samples and 4% to 14% in fish roe samples in Finland.[206] In 1991, 91 patients were hospitalized in Cairo with botulism intoxication associated with eating *faseikh* (uneviscerated salted mullet fish); *C. botulinum* type E was isolated.[466] In 2002, eight individuals from an Alaskan village on the Bering Sea contracted botulism type E from eating whale fluke from a Beluga whale that had washed up on shore several weeks before.[290]

C. botulinum spores germinate in an environment of appropriate pH (higher than 4.6), warm temperature (higher than 10°C [50°F]), sufficient moisture, and an anaerobic environment. The toxins are proteins of approximate molecular weight 150,000 Da and are absorbed in the proximal gastrointestinal tract.

Clinical Presentation. The toxin affects the presynaptic cholinergic neuromuscular junction, where it blocks the release of acetylcholine and causes flaccid paralysis.[31] Signs and symptoms develop within 12 to 36 hours of ingestion and include nausea, vomiting, abdominal pain, and diarrhea, followed by dry mouth, dysphonia (hoarseness), difficulty swallowing, facial weakness, ptosis, nonreactive or sluggishly reactive pupils (third cranial nerve), mydriasis, blurred or double vision (sixth cranial nerve), descending symmetrical muscular weakness leading to paralysis, and bulbar and respiratory paralysis. With adequate ventilatory support, mentation frequently remains normal. Death occurs in 10% to 50% of cases, depending on the availability of antitoxin.

If botulism is suspected, a careful food history should be obtained and the suspected food items collected. Laboratory confirmation of botulism is achieved when botulinum toxin or viable *C. botulinum* is detected in the food, toxin is demonstrated in the victim's serum or stool, or the organism is cultured from the stool. Toxin types are distinguished using type-specific antitoxin.[31] To determine the clinical need for botulism antitoxin, a number of tests may be helpful. Electromyography should be performed using repetitive stimulation at 40 Hz or greater; a positive test shows diminished amplitude of the muscle action potential with a single supramaximal stimulus, and facilitation of action potentials using paired or repetitive stimuli.[31] Cerebrospinal fluid (CSF) may be examined for white blood cells and protein (to rule out infectious etiologies), and an edrophonium (Tensilon) challenge test may be performed to rule out myasthenia gravis. Vital capacity should be monitored as a sensitive indicator of clinical deterioration.

Treatment. Ventilatory support should be provided at the first sign of respiratory inadequacy. Cathartics are recommended to eliminate residual toxin in the gastrointestinal tract. If available, equine trivalent antitoxin A, B, E should be administered as soon as possible to any person with symptoms, before irreversible neural dysfunction.[422] Each 10-mL vial of trivalent antitoxin contains 7500 IU type A, 5500 IU type B, and 8500 IU type E antitoxin.[31] Bivalent A (7500 IU) and B (5500 IU) antitoxin is

the most commonly used in the United States. Monovalent type E (5000 IU), pentavalent (A, B, C, D, and E), and heptavalent (A, B, C, D, E, F, and G) antitoxins are also available in specific circumstances. All the antitoxins are available from the CDC except for the heptavalent formulation, which is currently investigational. A physician who seeks antitoxin should first contact the state health department. If this is unsatisfactory, the CDC may be telephoned at 770-488-7100 (24 hours). The trivalent antitoxin is administered as an initial dose of 10 mL (one vial) every 2 to 4 hours for three to five doses or longer if symptoms persist.[31] Before administration, the victim should be skin tested for hypersensitivity to horse serum. If horse serum test material is not available, 0.1 mL of a 1:10 dilution of antitoxin in saline may be used. The antitoxin should not be stored at a temperature greater than 37°C (98.6°F).

An adjunct to therapy may be administration of guanidine, which increases the release of acetylcholine from nerve endings. The dosage is 15 to 50 mg/kg/day orally in four divided doses.

Prevention. Prophylaxis with antitoxin is not currently recommended; neither is general pentavalent (A to E) toxoid immunization.[11,339] The best prevention is public health education with respect to food preparation and avoidance of improperly stored food products. Because the spores are frequently detected in fish intestines, it is important to clean fish properly and to avoid consumption of the viscera, even in salt-cured products. To eliminate spores in food, heat or irradiation may be used. Types A and B may survive boiling for several hours (particularly at the lower temperatures associated with higher altitude) and generally require pressure heating at 120°C (248°F) for 30 minutes; type E spores are killed at 80°C (176°F) after 30 minutes. Preformed toxin is inactivated after heating for 20 minutes at 80°C or 10 minutes at 90°C (194°F). Germination is inhibited by acidification, refrigeration, freezing, drying, or the addition of salt, sugar, or sodium nitrate; however, heating remains the most reliable technique.[31]

PARASITES IN SEAFOOD

Most parasites of marine animals are of little public health concern to humans. However, there are 50 species of helminths found worldwide in fishes, crabs, crayfishes, and bivalves that can cause human infections. With the increasing consumption of raw seafood such as sushi and sashimi, the number of documented human infections is increasing. The overall risk of human infection is small.

Fish Tapeworm

In the United States, the consumption of raw fish (sushi) is increasingly popular, which has led to more frequent recognition of infestation with the fish tapeworm, *Diphyllobothrium latum*. Salmon appears to be a popular culprit.[102,127,205,441] Diphyllobothriasis is also reported from eating raw flesh of redlip mullet.[90] The fish tapeworm has a complex life cycle, in which a gravid egg released into freshwater releases a ciliated coracidium, which is eaten by a crustacean intermediate host. The coracidium penetrates the intestinal wall of the crustacean and then develops into a procercoid larva. A fish eats the small crustacean, and the procercoid larva migrates through the intestinal wall of the fish into fish muscle, where it changes into a plerocercoid larva. It is this final larval stage that is ingested by a human and that subsequently attaches to the intestine, where it grows into a mature tapeworm.

Classic symptoms include subacute abdominal pain, nausea, vomiting, diarrhea, and weight loss. Proglottids may be passed in the stool. Chronic *D. latum* infestation may induce megaloblastic anemia, as the tapeworm splits the vitamin B_{12} intrinsic factor complex and prevents absorption of the vitamin.[169] The diagnosis can be made by examination of the stool for typical proglottids or operculate egg forms, which measure 60 to 75 µm in length. Proper identification of the eggs is important to differentiatie them from the ova of trematodes endemic in southeast Asia, such as *Paragonimus westermani,* which may be carried by immigrants to the United States.[120] For documented *D. latum* infestation, praziquantel (5 to 10 mg/kg in one dose for adults or children) is the recommended treatment.[162,315,316] Magnesium sulfate as a purgative has been used to help expel the worm.[441] Niclosamide can also be used for treatment.[327] Because a worm may not be identifiable if expulsion is delayed or follows a purge, stool analysis should be repeated at 3 months to confirm successful therapy.

Fish tapeworm infection can be avoided by cooking fish until the parts for consumption reach a temperature of at least 56°C (133°F) for 5 minutes, or by freezing the fish to –18°C (0°F) for 24 hours or –10°C (14°F) for 72 hours.[476]

Trematodes

Humans can acquire an intestinal infection from the trematode *Nanophyetus salmincola,* which infests salmonid fishes, such as steelhead trout or salmon.[126] Canine infection with this fluke is a well-known phenomenon in the Pacific Northwest of the United States. Humans ingest the flesh of fish infested with the metacercariae, which excyst in the host and attach to the upper small bowel. The worms release eggs that are detected in the stool approximately 1 week after ingestion of infected fish.

Symptoms of nanophyetiasis include diarrhea, peripheral blood eosinophilia, abdominal discomfort, bloating, nausea and vomiting, weight loss, and fatigue. Although symptoms may resolve spontaneously over a period of months, antihelminthic treatment is recommended. Praziquantel (25 mg/kg orally three times a day for 1 day) is the first-line treatment.[2] Other regimens have included bithionol (50 mg/kg orally for two doses), niclosamide (2 g orally for three doses), or mebendazole (100 mg orally twice a day for 3 days). A similar syndrome has been caused by intestinal infection with *Heterophyes heterophyes,* which was successfully treated with praziquantel.[2]

Numerous other trematode infections are the cause of enormous morbidity worldwide as the liver and intestinal flukes cause distinct medical syndromes. For instance, in Southeast Asia, opisthorchosis caused by the liver fluke *Opisthorchis viverrini* is quite serious. The cercariae are ubiquitous in cyprinid fish.[158] Clonorchiasis occurs when humans eat raw or undercooked freshwater fish harboring the metacercariae of *Clonorchis sinensis*.[378]

Nematodes

Anisakiasis. Thousands of restaurants serve sushi in the United States, and many do so without specific knowledge of the various parasites that can infest their fare. For instance, many serve raw salmon, squid, shrimp, and mackerel.

The first report of acute gastric anisakiasis caused by penetration of the *Anisakis* larvae through the gastric mucosa was by Van Thiel in 1960.[454] It is a rare problem in the United States

but increasingly noted in Japan, where raw fish is more commonly eaten.[236,414] In a Japanese series, the fish consumed included predominantly mackerel; less common perpetrators included horse mackerel, bream, squid, sardines, and bonito.[414] In the United States, anisakine nematodes are present in many commercial fish intended for raw or semiraw consumption, such as Pacific herring (thus herring worm disease), sablefish, Pacific cod (thus codworm disease), arrowtooth flounder, petrale sole, coho salmon, Pacific ocean perch, silvergray rockfish, yellowtail rockfish, and bocaccio.[308] In rare cases, the anisakine worm can be present in tuna or yellowtail. The preservation of marine mammals along the western coast of the United States has been linked to greater worm burdens in fishes associated with these mammals, such as Pacific rockfish, red snapper, and salmon.[286]

Life and Habits. Anisakine nematodes, members of the order Ascarida (suborder Ascaridae), are found in great numbers in the viscera and muscles of fish.[304] There are 30 genera in the family Anisakidae, including *Anisakis* and *Pseudoterranova* (or *Phocanema*). Adult worms infest the stomachs of marine mammals, burrowing in clusters into the mucosal surface. Eggs passed in the stool embryonate and hatch in seawater to produce second-stage larvae, which are ingested by crustaceans, which are in turn eaten by squid or fish. In these hosts, the larvae migrate through the gut wall and encyst in the viscera or musculature.[366] The fish may then pass the parasite to other fish, to humans, or back to another marine mammal. The coiled *Anisakis* larva grows to approximately 2.5 to 3 cm in length and 0.5 to 1 mm in diameter. Fish are usually the intermediate (transport) host for larval anisakines.

The definitive host for *Phocanema decipiens* is the seal; the *Anisakis* larvae grow to maturity in the whale. Shellfish are not infested. Only four genera of anisakine nematodes have been implicated in human anisakiasis: *Anisakis, Phocanema, Porrocaecum,* and *Contracaecum*. In the United States, all cases are related to the larval stages of *Anisakis simplex* and *Phocanema decipiens*.[286]

Clinical Presentation. Symptoms from ingestion of *Anisakis* may begin within 1 hour of ingestion of raw fish and include severe epigastric pain, nausea, and vomiting. The presentation may mimic an acute abdomen. Asymptomatic gastroduodenal anisakiasis has also been a cause of acute urticaria and severe anaphylaxis in sensitized patients.[105,145,151] If the anisakine worms (such as *Phocanema*) do not implant and the infection is luminal without tissue penetration, the worms may be coughed up, vomited, or defecated, generally within 48 hours of the meal.[104] If the worm is felt in the oropharynx or proximal esophagus, the "tingling throat syndrome" is described.[286] An anisakine worm was documented in the tonsil of a 6-year-old girl with recurrent tonsillitis.[42]

Intestinal anisakiasis is more often delayed in onset (up to 7 days after ingestion) and marked by abdominal pain, nausea, vomiting, diarrhea, fever, eosinophilia (particularly with gastric anisakiasis), and occult blood in the stool.[104] This may be easily confused with appendicitis, regional enteritis, gastric ulcer, colonic or other gastrointestinal carcinoma, or, most commonly, other causes of small bowel inflammation with partial obstruction.[55,221,389,423] In one study, 29% of patients with Crohn's disease had detectable specific total immunoglobulin (IgG), IgM, and IgA antibodies against *A. simplex*.[176] Anisakiasis has also presented as small bowel obstruction requiring surgical resection.[372]

Diagnosis and Treatment. The definitive diagnosis of anisakiasis is usually made on the basis of morphologic characteristics of the whole worm when the creature is expelled by the patient or removed from the stomach after endoscopic examination.[366] Contrast-enhanced radiographs of the gastrointestinal tract may reveal threadlike gastric filling defects approximately 30 mm in length, which are typical, with a circular or ringlike shape.[55,414] Mucosal edema and pseudotumor formation are also seen. Ultrasonography can be useful in identifying intestinal anisakiasis.[207] However, once the worms have migrated to extragastric sites, the diagnosis can be difficult.

Early fiberoptic gastroscopy is recommended for patients in whom acute gastric anisakiasis is suspected and for those who have eaten raw fish within 6 to 12 hours before the onset of gastric symptoms. The *Anisakis* worm is usually found in the greater curvature of the stomach, often associated with severe mucosal edema.[223] Worms may also penetrate the intestinal wall.

The larvae of *Anisakis* can be visualized on endoscopy and removed with biopsy forceps. Fourth-stage larvae of *A. simplex* and *Pseudoterranova (Phocanema) decipiens* are found in the intestine and stomach of humans.[237] The larva is visible in the mucosa or buried within the submucosa, surrounded by an intense inflammatory granulomatous response.

When laparotomy is performed for presumed appendicitis, the diagnosis is based on identification of the worm in an inflamed segment of appendix, cecum, small intestine, mesentery, or omentum.[67,110] The only effective therapy for inflamed bowel is resection.

Antibodies to the ileal worm have been detected by radioallergosorbent test (RAST), ELISA, and immunofluorescent antibody assay, but these laboratory methods are not widely available.[176] Physical removal by endoscopy or surgery is the treatment of choice. The use of albendazole (200 mg orally, twice a day for 3 days) is of questionable efficacy.[162]

Prevention. The larvae are extremely difficult to identify in fish flesh, as they are colorless and normally tightly coiled in a spiral of approximately 3 mm. Only cooked (above 60° C [140° F]) or previously frozen (to –20° C [–4° F] for 24 hours) fish should be eaten. Smoking (kippering), marinating, pickling, brining, and salting may not kill the worms.[104] Candling is an inadequate method of surveillance, particularly in dark-fleshed fish infested with *Anisakis* larvae. Fish should be gutted as soon as possible after they are caught to limit the migration of worms from the viscera into the muscle.

The irradiation of fish to limit their infectivity is controversial because of potential generation of long-lived free radicals within the fish, as well as germination of spores of *Clostridium botulinum*.[475] To date, this practice is not legal for seafood in the United States, although it is used in other countries.

Eustrongylides. *Eustrongylides* is a genus of roundworms that can invade fish in its larval form and thus be consumed by humans in their quest for sushi and sashimi. *Eustrongylides* may also parasitize bait minnows, which are sometimes swallowed whole by fishermen. The worms are released into the human gastrointestinal tract, where they attain lengths of 15 to 30 cm

and penetrate the intestinal wall to enter the peritoneal cavity. Symptoms include unexplained abdominal pain, peritonitis, and fever in a live-bait fisherman. Surgical intervention may be required in pursuit of the acute abdomen, at which time the characteristically bright red worm is identified.[476]

Gnathostoma. Approximately 12 *Gnathostoma* species are responsible for gnathostomiasis, also known as larva migrans profundus or nodular migratory eosinophilic panniculitis. This systemic infection is caused by tissue destruction by migrating larvae.

Gnathostomiasis was first described in humans in 1889 in Thailand and has been endemic in Southeast Asia.[135A] However, with the advent of increased international travel, reports of gnathostomiasis are becoming more common in other regions of the world. Often, this is the result of travelers becoming infected in Southeast Asia and returning to other countries, as with 16 cases reported in London in 2003.[293A] There also appear to be newly endemic regions such as Central and South America. Mexico's first case was reported in 1970, but gnathostomiasis is now endemic in many regions of that country.[61A,265A] *Gnathostoma* larvae are acquired by the ingestion of raw or undercooked freshwater snakes and fish, particularly *Monopterus alba* (swamp eel), *Fluta alba* (eel), *Charias batrachus* (catfish), or *Channa striatus* (snake-headed fish).[265A] In Mexico, gnathostomiasis is particularly related to ingestion of ceviche (a dish made of raw fish marinated in lime juice).[313B,355A]

Life and Habits. There are 12 known species of *Gnathostoma,* all having similar life cycles. The definitive hosts are dogs, felines, and wild mammals.[265A] The first intermediate hosts are crustaceans which are ingested by the second intermediate hosts, fish. Humans then ingest the larvae found in these second intermediate hosts, leading to gnathostomiasis.

Clinical Presentation. Symptoms, which begin within 24 to 48 hours after ingestion of the larvae, are nonspecific: fever, arthralgias, myalgias, malaise, anorexia, vomiting diarrhea, and abdominal pain.[265A] Cutaneous gnathostomiasis presents as migratory swelling and inflammation, most often affecting the trunk, that appears 1 to 20 weeks after ingestion.[80A,265A] Visceral gnathostomiasis occurs when larvae migrate through internal organ systems such as the lungs, gastrointestinal tract, genitourinary tract, or CNS. CNS infections may present with radiculomyelitis with severe radicular pain followed by paralysis.[50A] Eosinophilia is a common but nonspecific finding.

Diagnosis and Treatment. Definitive diagnosis is by isolation of the larvae from lesion biopsies.[80A] ELISA has been used with a sensitivity of 93% and specificity of 96.7% in one study.[80A] A clinical diagnosis may be made in a patient with a history of ingesting raw or partially cooked fish, migratory swelling, and eosinophilia.[313A] The standard treatment for gnathostomiasis is albendazole (400 mg twice daily for 21 days). This results in a cure rate of 92%, and a single dose of ivermectin (200 µg/kg) results in a 76% cure rate.[245A]

Prevention. Adequate cooking of food is preventive, as is freezing to –20° C (–4° F) for 3 to 5 days. Lime juice is not effective at killing *Gnathostoma.*[265A]

OTHER SEAFOOD-RELATED POISONINGS

Poisoning by Environmental Contamination

In the process of concentrating fish proteins as a food source, a variety of protein-bound, non–water-soluble, or non–alcohol-soluble toxic compounds may be preserved. These include organic mercurials, hydrocarbons, dioxins, polychlorinated dibenzofurans, chlorinated pesticides, and heavy metals (e.g., antimony, arsenic, cadmium, chromium, cobalt, lead, phosphorus, mercury, nickel, zinc).[25,96,416] The overall public health risk for environmental contamination is concerning; however, the true risk of exposure is unknown.

Higher concentrations of polychlorinated biphenyls (PCBs) and dioxin-like compounds are found in Inuit people in the Arctic because of their traditional diet, which includes large quantities of sea mammal fat.[25] Data suggest that there may be an elevated risk of multiple myeloma in groups with high consumption of dioxin-contaminated fish from the Baltic Sea and Alaska, and accidental exposure in Italy.[375] Dioxin has been found in Dungeness crabs in Humboldt Bay, California.

In Taiwan, high levels of copper, zinc, and arsenic were found in oysters. The long-term exposure to metals from seafood consumption is potentially dangerous, although the real risk is unknown.[188] Urine arsenic levels have been shown to increase two- to sevenfold after consumption of certain types of seafood (mackerel, herring, crab, and tuna).[16] Consumption of fish rich in amines has been shown to increase the excretion of *N*-nitrosodimethylamine in the urine, demonstrating increased formation of carcinogenic *N*-nitrosamines.[456]

Mercury is found in marine organisms in the form of methylmercury (MeHg) and is concentrated in the food chain. Increased fish consumption is associated with higher blood levels of mercury.[277] MeHg is neurotoxic and crosses the placenta and blood–brain barrier, and prenatal poisoning causes mental retardation and cerebral palsy. The risk of this from seafood is unclear. High blood and hair concentrations of mercury have been found in fishermen of coastal villages, and adverse effects have been reported.[305,349] Controversy exists over the fetal risk from exposure to low-dose MeHg from maternal consumption of fish.[24] A study of children exposed to MeHg from seafood in a Madeira fishing community did not show any mercury-associated deficits.[302] A longitudinal cohort study of children showed no adverse outcome with either prenatal or postnatal MeHg exposure.[106]

In the past several years, the benefits of seafood containing long-chain omega-3 fatty acids have become popularized. This is partially responsible for an increase in seafood consumption. These fatty acids have been shown to decrease the risk of cardiac sudden death and coronary artery disease in both men and women.[8,201] However, small amounts of mercury are commonly found in the same fish, and studies have shown increased mercury levels in those with increased seafood ingestion.[175,494] These same studies have shown conflicting data on whether the elevated levels of mercury diminish the cardioprotective effect of omega-3 fatty acids, and more study is needed. Omega-3 fish oil supplements, however, have not been shown to contain elevated levels of mercury.[143]

Spills of toxic chemicals and petroleum byproducts will certainly continue to expand the list of carcinogens to which humans are becoming exposed through the marine environ-

ment. Although radiation exposure is not known to induce production of new marine poisons, the ingestion of radioactive fish poses a potential radiation hazard. Divers are exposed to a variety of environmental contaminants while exploring polluted waters. These hazards include solvents, nuclear wastes, herbicides, chemical effluents, and sewage.

Red Seaweed

Seaweed is a common component in the diet of individuals living in the Pacific Islands and the Pacific Rim. It can be eaten raw or cooked. Most *Gracilaria* species are nontoxic and edible, but a number of poisonings and deaths have been reported in Japan, Guam, and Hawaii. Ingestion of the red seaweeds *Gracilaria verrucosa* (ogonuri) and *Gracilaria chordai* (tsurushiramo) is associated with a toxic syndrome, including gastroenteritis and death.[313] It is commonly referred to as Japanese ogonuri poisoning.

In 1991 in Guam, 13 individuals became ill and three died after ingesting the red algae *Polycavernosa tsudae* (formerly *Gracilaria tsudae* or *edulisi*).[186] Symptoms consisted of diarrhea, abdominal cramping, vomiting, generalized numbness, perioral and extremity paresthesias, numbness of the fingertips, diaphoresis, jaw aching, muscle spasms, tremors, and hypotension. *Gracilaria lemanaeformis* may have been responsible for three illnesses in California in 1992.[73] In Japan, two people became ill with nausea, vomiting, and hypotension and one died after ingestion of *G. verrucosa*. Prostaglandin E_2 is suggested as the toxic component of *G. verrucosa,* and polycavernosides, which are glycosidic macrolides, are the probable toxins in *G. tsudae*.[313,496,497]

An outbreak of acute gastroenteritis from the ingestion of the red alga *Gracilaria coronopifolia* occurred in Hawaii in 1994, in which seven individuals reported symptoms of diarrhea, nausea, vomiting, and a burning sensation in the mouth and throat.[73] Aplysiatoxin and debromoaplysiatoxin have been isolated as the causative agents.[306] These toxins are probably produced by blue-green algae that are found on the surface of *G. coronopifolia* and are known to cause contact dermatitis in swimmers in Hawaii. Aplysiatoxin and debromoaplysiatoxin experimentally cause edema and bleeding of the small intestine, leading to hemorrhagic shock and diarrhea in the large intestine.[211]

Sea Turtle Poisoning (Chelonintoxication)

A variety of tropical Pacific, particularly Japanese, marine turtles is toxic when ingested (Box 66-7).[405] The term *chelonintoxication* comes from the order Chelonia. All portions of the turtle are toxic and the freshness of the meat is irrelevant. In Madagascar, 60 people became ill after eating sea turtle in 1994. The mortality rate was 7.7%.[344] Lyngbyatoxin A has been isolated from the meat of a green turtle, *Chelonia mydas,* that was involved in a fatal intoxication.[486] The source of the toxin was suspected to be blue-green algae belonging to the genus *Lyngbya*. The sea turtle may feed on sea grass contaminated with this alga.

Symptoms develop from 1 to 48 hours after ingestion and include ulcerative glossitis and stomatitis, pharyngitis, diaphoresis, hypersalivation, nausea, vomiting, diarrhea, abdominal pain, vertigo, icterus, desquamative dermatitis, hepatosplenomegaly, centrilobular hepatic necrosis with fatty degeneration, renal failure, somnolence, and hypotension. The mortality rate can be as high as 28% to 44%. Therapy is supportive and based on symptoms.

A variety of *Salmonella* serotypes has been isolated from pet turtles (*Pseudemys* [or *Chrysemys*] *scripta elegans*) imported into and from the United States.[113,243,278] Pet-associated salmonellosis was a significant problem in the 1970s. In 1975, Canada banned the importation of turtles, and in the same year, the FDA prohibited the sale of small turtles in the United States. However, the popularity of iguanas and other reptiles is increasing; these reptiles can also transmit *Salmonella* to humans. Reptile-associated salmonellosis causes febrile gastroenteritis, septicemia, and meningitis; one death has been reported due to myocarditis from *Salmonella virchow* in a small child.[71,113,311]

Box 66-7. Representative Marine Turtles Hazardous to Humans

Phylum Chordata
- Class Reptilia
  - Order Chelonia: turtles
    - Family Cheloniidae
      - *Caretta caretta gigas:* Pacific loggerhead turtle
      - *Chelonia mydas:* green turtle
      - *Eretmochelys imbricata:* hawksbill turtle
    - Family Dermochelidae
      - *Dermochelys coriacea:* leathery turtle
    - Family Thioncychidae
      - *Pelochelys bibroni:* soft shell turtle

Liver Poisoning: Hypervitaminosis A

Hypervitaminosis A can occur with the ingestion of the livers of certain polar bears, seals, sea lions, whales, dolphins, walruses, husky dogs, and Pacific sharks. The vitamin A content of shark liver can reach 100,000 IU/g. A typical ingestion involves the administration of more than 1 million (and occasionally 3 to 8 million) IU of vitamin A. The recommended daily allowance is 4000 to 5000 IU. Symptoms of hypervitaminosis A include formication, headache, apathy, drowsiness, giddiness, irritability, photophobia, nausea, vomiting, diarrhea, polyarthralgia, seizures, desquamative dermatitis, ophthalmoplegia, and raised CSF pressure with a pseudotumor cerebri type of presentation (acute or chronic, the latter with headache, lip fissuring, papilledema, decreased visual acuity, and tinnitus).[134,292] Elevated levels of serum glutamic oxaloacetic transaminase and serum vitamin A (markedly in excess of 70 mg%) may be measured. A normal serum β-carotene level excludes the possibility of a plant source (for example carrots or mangoes) for the vitamin.[292] The syndrome is rarely fatal and resolves in 2 to 8 weeks.

AMEBIC INFECTIONS

Free-living, amphizoic amebas belonging to the genera *Naegleria, Acanthamoeba,* and *Balamuthia* can cause significant CNS pathology in human beings. Approximately 350 cases of human infection have been reported to date.[281,353] These amebas are ubiquitous in nature; they are found in soil, lakes, ponds, swimming pools, hot springs, and warm water around the world. Human infection caused by amebas has significantly increased over the past 10 years.[281]

Free-living amebas are responsible for three disease entities: (1) primary amebic meningoencephalitis (PAM) produced by *Naegleria fowleri,* (2) granulomatous amebic encephalitis (GAE) caused by *Acanthamoeba* species and *Balamuthia mandrillaris,* and (3) *Acanthamoeba* keratitis (AK) caused by *Acanthamoeba* species.

Primary Amebic Meningoencephalitis

PAM is a fulminant, rapidly progressive CNS infection produced by *N. fowleri.* It was first described in 1965 by Malcolm Fowler and Rodney Carter in four human cases of meningoencephalitis from *N. fowleri.*[144] Worldwide, approximately 180 cases of PAM have been reported, with over 80 cases reported in the United States alone.[70,111,251,270,354,460] *N. fowleri* multiplies and grows between 40° and 45°C (104° and 113°F). In response to adverse environmental conditions, such as cold temperature, the ameba encysts and remains in the sediment in the bottom of lakes, rivers, and pools.

Infections occur in healthy children and adults who contact the ameba while swimming in polluted water in manmade lakes, ponds, and swimming pools, or the ameba may be inhaled from the dust and air.[281] Infection is more common during summer months. The amebas enter the CNS through the nasal mucosa and olfactory neuroepithelium. The amebic trophozoites travel up the unmyelinated fila olfactoria of the olfactory nerves and through the cribriform plate to the subarachnoid space.[219] They proliferate and penetrate into the CNS, causing edema and necrosis. The incubation period is from 1 to 15 days. Symptoms include severe headache, fever, nausea, vomiting, and stiff neck. Rapid neurologic deterioration accompanied by signs of fulminant meningitis with seizures, coma, and death follows within 2 to 3 days.

Diagnosis is made by the direct visualization of trophozoites in the CSF along with polymorphonuclear pleocytosis, elevated protein, and low glucose. *Naegleria* trophozoites typically measure 8 to 12 µm in diameter with indistinct cytoplasm, a round nucleus, and a perinucleolar halo.[281] *N. fowleri* causes acute leptomeningitis and hemorrhagic necrosis of the orbitofrontal cortex, olfactory bulbs, and base of the brain, with edema of the cerebral hemispheres and cerebellum. Computed tomography (CT) scan of the brain shows nonspecific cerebral edema.[233,370] Early detection and treatment are essential, as this disease carries a very poor prognosis with a mortality rate of 98%. To date, there are six cases of successful treatment of PAM in individuals who were treated very early in the clinical course.[58,335,379,464] Treatment includes high-dose intravenous and intrathecal amphotericin B.[161] Oral ketoconazole and rifampicin have been used in addition to amphotericin B.[335]

PAM should be suspected in any previously healthy individual who has been exposed to fresh warm water within 7 days of onset of illness and who has clinical findings of bacterial meningitis with a basilar distribution of exudate by head CT.[70]

Granulomatous Amebic Encephalitis

Several species of *Acanthamoeba* and *B. mandrillaris* are pathogenic opportunistic amebas that cause GAE, mainly in victims who are immunocompromised, debilitated, diabetic, or alcoholic. GAE has been reported in patients with systemic lupus erythematosus, acquired immunodeficiency syndrome (AIDS), and bone marrow transplantation.[5,135,245,425] However, two cases of GAE caused by *B. mandrillaris* occurred in apparently immunocompetent individuals.[354] Approximately 170 cases of GAE have been reported worldwide.[281]

Acanthamoeba species are ubiquitous in nature; they have been found in ocean water, ponds, sewage, rivers, air-conditioner filters, cooling towers, eye-wash stations, and dust. Some of the *Acanthamoeba* opportunistic species include *A. castellanii, A. hatchetti, A. culbertsoni, A. astronyxis, A. polyphaga, A. rhysodes,* and *A. mauritaniensis.*[281] *B. mandrillaris* has not been isolated from the environment, although, like *Acanthamoeba,* it probably exists in cyst form. The trophozoites and cysts can enter through the lungs and ulcerations in the skin. The olfactory neuroepithelium may also act as a portal of entry.[215,281] The incubation period is unknown but is probably weeks.

Both *Acanthamoeba* species and *B. mandrillaris* produce a chronic granulomatous encephalitis. The clinical presentation may mimic tuberculous meningitis or viral encephalitis. Symptoms include headache, fever, seizures, personality changes, cranial nerve palsies, hemiparesis, and coma. There may be ulcerations of the skin. The amebas cause hemorrhagic necrosis and foci of encephalomalacia in occipital, parietal, temporal, and frontal lobes. The lesions are multifocal and most numerous in the basal ganglia, midbrain, brainstem, and cerebral hemispheres.[281] Angiitis can occur, and trophozoites are often found invading the vascular walls.[352] The amebas multiply and can disseminate throughout the body. Other organs involved (at the time of autopsy) include liver, lungs, kidneys, adrenals, pancreas, lymph nodes, and heart.[5,425]

Magnetic resonance imaging (MRI) and CT scans have shown multiple enhancing lesions in the cerebral hemispheres and cerebellum, but the scans are nondiagnostic.[233,352,370] Diagnosis is difficult, as amebas are rarely observed in the CSF. Examination of the CSF shows a moderate mononuclear pleocytosis, elevated protein, and low glucose. Definitive diagnosis is made by direct visualization of amebic trophozoites and cysts within brain tissue. Unfortunately, there is no effective treatment for GAE, and mortality is 100% in immunocompromised patients.[281] Although pentamidine isethionate, propamidine, sulfadiazine, and ketoconazole are effective in vitro, these drugs do not appear to be useful because of the underlying immunosuppression of most of these patients.[281] Based on tissue-culture studies, pentamidine isethionate appears to be the best choice for treatment of *B. mandrillaris* encephalitis.[371] One case of widespread granulomatous skin lesions in an immunocompromised patient resulting from *A. rhysodes* was successfully treated with intravenous pentamidine isethionate for 4 weeks, topical chlorhexidine gluconate, and ketoconazole cream followed by oral itraconazole.[397]

Acanthamoeba Keratitis

AK is caused by *Acanthamoeba* species; over 700 cases have been reported worldwide.[281] *Acanthamoeba* enters the corneal stroma through minor trauma or abrasion, causing chronic inflammation of the cornea, which can impair vision and lead to vascularized corneal scarring, perforation, and loss of the eye. The use of soft contact lenses and homemade saline solutions are the greatest risk factors for AK. Symptoms include severe eye pain, photophobia, conjunctival inflammation, and blurred vision. Diagnosis is made by identification of the trophozoites or cysts by corneal scrapings or biopsies. The treatment of choice for AK is 0.02% polyhexamethylene biguanide (PHMB)

or propamidine (0.1%) with topical polymyxin B, gramicidin, or neomycin.[159,252] Penetrating keratoplasty and corneal grafting have been performed.[138] Contact lens wearers should use sterile solutions for lenses and should consider not wearing contacts while engaged in water sports.[208]

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67 Seafood Allergies

Sue L. Hefle,* Robert K. Bush, and Steve L. Taylor

True seafood allergies are immunoglobulin E (IgE)-mediated reactions. Although allergic reactions can occur to all classes of seafood, accurate epidemiologic data are difficult to obtain. Crustacean allergies are one of the most common types of food allergy in the U.S. adult population[69]; a recent report found that 2.3% of the U.S. population has seafood allergy, with 2.0% attributed to shellfish.[111] On the other hand, codfish allergy is probably the most common form of food allergy in Scandinavian countries.[2]

BIOLOGIC CLASSIFICATION

An understanding of the taxonomic relationships among different species of marine animals is important. Many seafood species may be allergenic, but most allergic individuals are not sensitive to all species. The degree of allergic cross-reactivity among the different fish species varies widely between patients. Thus, selective avoidance diets are possible. The biologic classification of the edible species of fishes is given in Table 67-1. Most edible fishes belong to class Osteichthyes, although sharks (including dogfish), rays, and skates are in a different class (Chondrichthyes). The most commonly consumed fishes belong to only a few orders: Salmoniformes (salmons, trout, whitefishes, smelts, and pikes), Perciformes (basses, perches, dolphin fishes, snappers, groupers, redfishes, mackerels, and tunas), Gadiformes (codfishes, pollocks, haddocks, and hakes), Pleuronectiformes (flounders, halibuts, and soles), Clupeiformes (herrings, sardines, anchovies, shad, menhadens, and alewives), Cypriniformes (carps and catfishes), and Scorpaeniformes (rockfishes). Because many of the edible fish species belong to the orders Salmoniformes, Perciformes, and Gadiformes, a more detailed breakdown of the relationships for those orders is provided in Tables 67-2, 67-3, and 67-4.

*Deceased. The contributions of Dr. Hefle to our knowledge of food science and to wilderness medicine are very significant and appreciated. It was my great privilege to have known and worked with her. (PSA)

The physician should be alert to the confusing common names of fishes. For instance, king mackerel may be sold as cero, silver cero, black salmon, cavalla, or kingfish. Kingfish can also mean king whiting, which itself is sold as ground mullet, whiting, gulf whiting, surf whiting, southern whiting, sand whiting, or silver whiting. Whiting may be a name used to describe hake. Unusual types of fish, such as croakers or threadfins, are sometimes used to make some food ingredients, and many consumers may not understand that these are a type of fish. In addition, identification of fish is often erroneous, particularly when performed by sport fishermen.

The biologic classification of mollusks and crustacea is listed in Table 67-5. Crustacea are classified as arthropods, together with spiders, centipedes, and insects. At least 30 edible species of crustacea are commonly consumed in the United States. The Mollusca include clams, mussels, oysters, cockles, snails, cuttlefish, limpets, abalone, squid, and octopus. More detailed lists of the edible species of mollusks and crustacea are given in Tables 67-6 and 67-7. Over the years, classification of the shrimp, in particular, has changed, even to the point of changing genus names.

CLINICAL MANIFESTATIONS

Allergy to fish is a common cause of IgE-mediated food-allergic reactions, especially in geographic regions where fish is an important dietary component. Symptoms of fish allergy are similar to other IgE-mediated food allergies, with symptoms usually appearing immediately (minutes to an hour) after exposure and ranging from mild to life-threatening. Fatal reactions have occurred as a result of allergic reactions to fish.[16,128] Although the exact prevalence of fish-allergic subjects is not firmly established, fish is listed among the most common of all allergenic foods. Although not specifically confirmed by clinical investigations, fish-allergic individuals are thought to be potentially reactive to all species of fish. General caution must be used

or propamidine (0.1%) with topical polymyxin B, gramicidin, or neomycin.[159,252] Penetrating keratoplasty and corneal grafting have been performed.[138] Contact lens wearers should use sterile solutions for lenses and should consider not wearing contacts while engaged in water sports.[208]

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TABLE 67-1. Taxonomic Relationships among the Edible Fishes

TAXONOMIC CLASSIFICATION		
CLASS	SUBCLASS OR ORDER	COMMON NAME
Chondrichthyes	Elasmobranchii	Sharks
Osteichthyes	Acipenseriformes	Sturgeons, paddlefishes
	Elopiformes	Tarpons, ten-pounders, bonefishes
	Anguilliformes	Common eels, morays
	Clupeiformes	Herrings, sardines, alewives, shad, menhaden, anchovies
	Salmoniformes	Trouts, salmons, whitefishes, graylings, discoes, pikes, lake herrings, pickerels, muskelunges, euchalons, capelins, smelts, saugers
	Gonorynchiformes	Milkfishes, awa
	Cypriniformes	Minnows, carps, suckers, catfishes
	Beloniformes	Sauries, needlefishes, flying fishes
	Mugiliformes	Mullets, barracudas, silversides, threadfishes
	Lambridiformes	Opah, mariposas
	Tetraodontiformes	Pufferfishes, boxfishes, trunkfishes
	Pleuronectiformes	Flounders, halibuts, soles, dabs, turbots
	Perciformes, suborder Percodei	Basses, crappies, bluegills, sea basses, sunfishes, perches, bluefishes, jacks, pompanos, dolphin fishes, snappers, groupers, scups, grunts, porgies, pomfrets, sheepsheads, snooks, robalos, bigeyes, catalugas, croakers, spots, redfishes, tautogs, butterfly fishes, wrasses, spade fishes, goatfishes, mojarras, rudderfishes, surf-fishes, weakfishes, roaches, drums, cichlids
	Perciformes, suborder Scombroidei	Mackerels, tunas, cutlassfishes, albacores, bonitos, kingfishes
	Perciformes, suborder Xiphoidei	Swordfishes, marlins, sailfishes, spearfishes
	Perciformes, suborder Ammodytoidei	Sand lances
	Perciformes, suborder Stromateoidei	Butterfishes
	Scorpaeniformes	Rockfishes, scorpionfishes, greenlings
	Gadiformes	Codfishes, ling cods, pollocks, haddocks, tomcods, hakes, codlings
	Percopsoidei	Trout-perches, sand rollers

TABLE 67-2. Taxonomic Relationships among Salmoniformes

TAXONOMIC CLASSIFICATION			
SUBORDER	FAMILY	GENUS	COMMON NAME
Salmonoidei	Salmonidae	*Coregonus*	Whitefishes, ciscoes, bloaters, lake herrings
		Onchorhynchus	Pacific salmons
		Salmo	Trouts, Atlantic salmon
		Salvelinus	Chars, brook trout, Dolly Varden trout, lake trout
		Thymallus	Grayling
	Osmeridae	*Osmerus*	Smelts
		Mallotus	Capelins
		Thaleichthys	Euchalon
Esocoidei	Esocidae	*Esox*	Pikes, saugers, pickerels, muskelunges

for these individuals if they choose to ingest varying fish species or fish products.

Symptoms of seafood allergy are not different from those caused by other allergenic foods. Symptoms are often, but not always, related to the method of exposure: ingestion, inhalation, or physical contact. If ingestion occurs, symptoms may be first noted in the mouth and face, and can include localized or general pruritus, urticaria, laryngeal edema, or angioedema.[95] Atopic dermatitis may be exacerbated by seafood allergies.[106] Gastrointestinal symptoms, such as nausea, vomiting, diarrhea, and cramps, may occur as a result of ingestion. Some reports indicate severe asthma/bronchospasm as primary symptoms after consumption of certain crustacea, mollusks,[20,31] and fish.[94,104] In some individuals, seafood can cause anaphylaxis. Symptoms of anaphylaxis may include some or all of these symptoms in addition to hypotension, electrocardiographic changes, and cardiovascular collapse. Symptoms of food allergy usually occur within an hour after ingestion.[1,18,19,27] However, delayed reactions (3 to 24 hours after ingestion) can occur, and have been noted with, among others, dogfish,[89,105] cuttlefish,[108] abalone,[79] and limpets.[89] Patients allergic to fish can present with skin reactions after handling raw fish.[90,103] Also, both acute and delayed asthma reactions can occur in fish-allergic children when they are provoked with aerosolized fish.[104] In one report, a 2-year-old fish-allergic child had facial urticaria and angioedema after her grandfather, who had eaten bream 2 hours earlier, kissed her.[87] Food-dependent exercise-induced anaphylaxis is a condition wherein patients in the resting state can eat

TABLE 67-3. Taxonomic Relationships among Perciformes

TAXONOMIC CLASSIFICATION		COMMON NAME
SUBORDER	FAMILY	
Percoidei	Centraranidae	Largemouth bass, black basses, breams, bluegill, sunfishes, crappie, Sacramento perch
	Percichthyidae	Striped bass, white bass, white perch, yellow bass
	Serranidae	Groupers, sea basses, jawfishes
	Percidae	Pikes, saugers, yellow perch, river perch
	Centropomidae	Snooks, robalos
	Priacanthidae	Cataluras, bigeyes
	Pomatomidae	Bluefish
	Coryphaenidae	Dolphin fish (mahimahi)
	Carangidae	Jacks, pompanos, cavallas, moonfishes, scads, jack mackerel
	Bramidae	Pomfret
	Pomadasyidae	Grunts
	Lutjanidae	Snappers, mutton fish, rabirubias
	Chaetodontidae	Butterflyfishes
	Ephippidae	Spade fishes
	Labridae	Wrasses, tautog
	Cichlidae	Cichlids
	Sciaenidae	Drums, redfish, croakers, weakfishes, kingfishes
	Mullidae	Goatfishes
	Sparidae	Porgies, scups, spots, sheepsheads
	Gerridae	Mojarras
	Kyphosidae	Rudderfishes, sea chubs
	Embiotocidae	Surf-fishes
Scombroidei	Scombridae	Mackerels, tunas, bonitos, Spanish mackerels, sierra, kingfish, cero, cavalla, petos
	Trichiuridae	Cutlassfishes
Xiphioidei	Xiphiidae	Swordfishes
	Istiophoridae	Marlins, sailfishes, spearfishes
Ammodytoidei	Ammodytidae	Sand lances
Stromateoidei	Stromateidae	Butterfishes

TABLE 67-4. Taxonomic Relationships among Gadiformes

TAXONOMIC CLASSIFICATION			COMMON NAME
SUBORDER	FAMILY	GENUS	
Gadoidei	Merlucciidae	*Merluccius*	Pacific hake
	Gadidae	*Pollachius*	Pollock
		Theragra	Walleye pollock
		Gadus	Codfishes
		Microgadus	Tomcods
		Lota	Turbot
		Melanogrammus	Haddock
		Urophycis	Atlantic hake, red hake, Gulf hake

TABLE 67-5. Taxonomic Relationships among Edible Mollusks and Crustacea

TAXONOMIC CLASSIFICATION			COMMON NAME
PHYLUM	CLASS	SUBCLASS	
Mollusca	Gastropoda	Prosobranchi	Marine snails, abalone, periwinkles, whelks
		Pulmonata	Freshwater snails
	Bivalvia	Lamellibranchii	Mussels, cockles, oysters, scallops, clams
	Cephalopoda	Coleoidea	Squid, octopus
Arthropoda	Crustacea	Malacostraca	Shrimp, crabs, lobsters, crayfish

TABLE 67-6. Edible Bivalves and Cephalopods

GROUP	COMMON NAME	SCIENTIFIC NAME
Clams	Surf or bar clam	*Mactra solidissima*
	Soft clam	*Mya arenaria*
	Hard clam or quahog	*Venus mercenaria*
	Atlantic razor clam	*Ensis directus*
	Pacific razor clam	*Siliqua patula*
	Stout razor clam	*Tagelus gibbus*
	Butter clam	*Saxidomus nuttali*
	Little neck clam	*Tapes staminea*
	Geoduck clam	*Panope generosa*
	Freshwater clam	*Corbicula leana*
	Pismo clam	*Tivela stultorum*
	Short-necked clam	*Venerupis japonica*
	Jackknife clam	*Tagelus californianus*
Mussels	European or Atlantic mussel	*Mytilus edulis*
	Pacific mussel	*Mytilus californianus*
	South Asian green mussel	*Mytilus smaragdinus*
Cockles	Common cockle	*Cardium corbis*
	Red cockle	*Cardium echinatum*
	Spiny cockle	*Cardium aculateum*
Scallops	Common scallop	*Pecten gibbus borealis*
	Deep water scallop	*Placopecten megallanicus*
	Japanese scallop	*Pecten yessoensis*
Oysters	Pacific oyster	*Ostrea gigas*
	Atlantic oyster	*Crassostrea virginica*
	Japanese oyster	*Crassostrea laperousei*
	Coon oyster	*Ostrea frons*
Squid	North American squid	*Loligo paeleii*
	North American squid	*Loligo opalescens*
	Japanese squid	*Ommastrephes sloani pacificus*
Octopus	Common octopus	*Octopus vulgaris*

TABLE 67-7. Edible Crustacea

GROUP	COMMON NAME	SCIENTIFIC NAME
Lobsters	Atlantic lobster	*Homarus vulgaris*
	European lobster	*Homarus gammarus*
	Northern lobster	*Homarus americanus*
	Spiny lobster	*Pancilirus argus*
Crabs	Blue crab	*Callinectis sapidus*
	Deep sea blue crab	*Portunus pelagicus*
	Dungeness crab	*Cancer magister*
	Jonah crab	*Cancer borealis*
	King crab	*Paralithodes camtschatica*
	Snow crab	*Chionoectes bairdi*
	Tanner crab	*Chionoectes tanneri*
	Spider crab	*Talvia maticum*
	Stone crab	*Menippe mercenaria*
Prawns and shrimp	Common prawn	*Palaemon serratus*
	Deep water prawn	*Pandalus bortalis*
	Pink Maine shrimp	*Pandalus borealis*
	Coon stripe shrimp	*Pandalus hypsinotus*
	Brown shrimp	*Panaeus aztecus*
	Pink shrimp	*Panaeus duorarum*
	White shrimp	*Panaeus indicus*
	Gamba shrimp	*Panaeus longirostris*
	Karuma prawn	*Panaeus japonicus*
	Giant tiger prawn	*Panaeus monodon*
	Common tiger prawn	*Panaeus esculentus*
	Indian prawn	*Panaeus indicus*
	Eastern king prawn	*Panaeus plebejus*
	Banana prawn	*Panaeus merguiensis*
	Brine shrimp	*Artemia salina*
	Rock shrimp	*Silyomia brevirostris*
	Sea bob shrimp	*Xiphophenaeus kroyeri*
	Royal red shrimp	*Hymenopenaeus robustus*
	Freshwater prawn	*Macrobrachium carcinus*

food without difficulty, but exercise within 2 to 4 hours after ingestion can bring on symptoms of anaphylaxis. Shrimp, mollusks, and squid are among the implicated foods.[58,84,85]

Occupational reactions can include dermatitis, urticaria, angioedema, rhinitis, conjunctivitis, asthma, and hypersensitivity pneumonitis. Employees in the fishing and seafood processing industries are at risk of developing occupational allergy to the seafoods with which they work. Although asthma is the most prominent symptom, anaphylaxis has been reported with occupational exposure.[82] Physical contact with seafood can elicit allergic symptoms in sensitive individuals, and urticaria, contact dermatitis, angioedema, and asthma have been reported after contact with shellfish,[26,41,115,126,130] fish,[10,26,34] and even fish bait.[112] Most of these contact reactions occur as part of occupational exposure, but consumers can also be affected.[95] Inhalation reactions are most often associated with occupational exposure, such as in cleaning and cooking seafood, but they can also occur when seafood is cooked at home or in restaurants. Seafood implicated in occupational asthma include oysters,[93] clams,[33] shrimp,[33,70] prawns,[42] fish,[24,36] snow and king crabs,[21,96] lobsters,[70,102] sea squirts,[59] abalone,[23] powdered marine sponges,[9] cuttlefish,[113] and clam liver extract.[60] Mollusk shell dust has been found to cause hypersensitivity pneumonitis,[97,98] and shark cartilage powder caused a fatal occupational asthma attack.[99]

DIAGNOSIS

Medical History

Diagnosis of food allergy may be simple or extremely complex. With seafood, it is particularly important to obtain correct identification of the offending food to determine if cross-reaction patterns can be established. An estimate of the quantity of the food needed to elicit the reaction may help to distinguish intolerance from a true seafood allergy.

Skin Testing and CAP Tests

Although the skin prick test is extremely useful, commercial extracts are not available for every seafood species individually. Often, mixed extracts are used. There is some risk to patients who have experienced severe anaphylactic reactions. As with other food allergen extracts, fish skin-prick tests have superior sensitivity and negative predictive accuracy but poor positive predictive value and specificity.[11] In one study, some commercial fish skin-test extracts were found to contain relatively high levels of histamine,[122] which could be a contributing factor; however, the patient often fails to correctly distinguish the species causing the allergy. Allergists generally recommend removing all edible fish, including both Osteichthyes (bony fish) and Chondrichthyes (shark), from the diet when the patient has a demonstrated history of allergic reaction to any fish or if there is a positive skin test or CAP (an immunoassay) to a fish extract. However, research using double-blind placebo-controlled fish challenges[11] and other tests[2,32] in fish-allergic children has shown that they are not uniformly sensitive to all species; hence, hypersensitivity to one species does not automatically warrant dietary elimination of all fish. Fish challenges in children with negative skin tests were negative; therefore, skin testing may be advisable before recommending radical dietary restrictions.[11] The CAP test is reserved for situations in which skin testing is inappropriate by virtue of previous severe reactions.

Double-Blind Food Challenges

The ultimate test to verify that a particular food causes a reaction is the double-blind food challenge.[13,15] This should not be performed in persons who have experienced life-threatening reactions and should be undertaken only under close physician supervision. Dried or freeze-dried foods can be encapsulated in opaque, dye-free capsules; alternatively, the food of interest can be hidden in a food vehicle. Appropriate identical placebo-controls are prepared. Although such testing is time consuming and somewhat labor intensive, it permits precise diagnosis.

Elimination Diets

When a certain type of seafood is suspected of producing symptoms, it may be eliminated from the diet. After the initial complaints have resolved, the sufferer reintroduces the food to see if the reaction is re-elicited. Elimination diets should be used only in persons who have not experienced life-threatening symptoms.

Controversial Techniques

A number of clinical and laboratory tests are unreliable predictors of the presence of seafood allergy. These include pulse index, in vitro cytotoxic food tests, intracutaneous provocation, sublingual provocation, basophil degranulation, and sublingual neutralization.[5]

Diagnosis of Occupational Asthma

Although the measures described previously are useful in diagnosing cutaneous, gastrointestinal, or systemic manifestations of food allergy, diagnosis of occupational asthma requires a different approach. If the person notes the onset of asthma symptoms related to work exposure, and there is improvement on weekends or while on vacation, occupational asthma should be suspected. Asthma per se is verified by appropriate pulmonary function tests, such as spirometry with and without bronchodilators. If the history is suggestive but not corroborated by physical examination or simple spirometry, it may be necessary to perform a provocation test with inhaled methacholine or histamine to document airway hyperreactivity. The diagnosis depends ultimately on the provocation of asthma by a bronchial inhalation challenge with the suspected allergen. Such evaluation should occur under close observation in a hospital setting.

TREATMENT

Treatment for seafood allergies is the same as for any other allergic reaction (see Chapter 56). Avoidance should encompass both consumption of seafood and inhalation of cooking vapors from the appropriate offending species. To avoid unnecessary dietary restrictions, proper diagnosis is extremely important.

IDENTIFICATION AND CHARACTERIZATION OF SEAFOOD ALLERGENS

Fish

Of the seafood allergens that have been isolated and purified, the best characterized is the major allergen of codfish, Gad c 1, which belongs to a group of muscle tissue proteins known as parvalbumins.[38] Parvalbumins are responsible for mediating the concentration of calcium in muscle. Gad c 1 is an acidic protein of molecular weight 12,328 Da, is composed of 113 amino acids and 1 glucose molecule, and has an isoelectric point of 4.75. It comprises three domains: AB, CD, and EF, consisting of three helices interspaced by one loop.[39] Each of the loops of the CD and EF domains coordinates one Ca^{+2}-binding site; these sites correspond to IgE-binding sites. It is stable to heat, extremes of pH, and mild proteolysis. The allergenicity of Gad c I is decreased by acetylation or polymerization of the tyr-30 residue,[6] modification of the arg-75 residue, or unchelating the two calcium ions,[37] but these methods cannot be used commercially. Gad c 1 is also comparatively resistant to pepsin digestion.[14] Gad c 1 shares amino acid sequence homologies in the 34% range with parvalbumins from carp, hake, and whiting.[37,78] The allergens from other fish species, including salmon (Sal s 1), horse mackerel (Tra j 1), and bigeye tuna (Thu o 1), are also parvalbumins.[109,110,116]

Another codfish allergen was identified in an in vitro study with cod-allergic patient sera to have a molecular size of 41 kDa, in addition to five other bands at 13, 21, 27, 47, and 58 kDa.[43] The 41-kDa allergen had an isoelectric point estimated at 5.8. The 13-kDa band was a major allergen for all subjects in the study. The 13- and 41-kDa bands were recognized by anti-parvalbumin monoclonal antibodies specific for the first calcium-binding site, as were the bands at 28 and 49 kDa. There is no correlation between the amino acid composition of the 41-kDa allergen and other cod allergens previously described in the literature.[43] In another study investigating IgE-binding in sera of fish-allergic patients, pre-rigor cod muscle was found to contain IgE binding bands of 12, 22, 30, 45, 60, 67, 104, and 130 kDa.[35] A major IgE-binding band for all sera was at 12 kDa; the 30- and 67-kDa bands also possessed good IgE binding activity. The bands at 104 and 130 kDa were thought to be aggregates of smaller bands. It was interesting to note that when the cod had been dead for several days, new IgE-binding bands of 18, 41, and 80 kDa appeared, and the relative content of IgE-binding proteins was increased. The action of naturally occurring proteases could have opened up sections of the peptides and made them more accessible to IgE binding during this period. The anti-codfish parvalbumin monoclonal antibody was also found to bind to all IgE binding bands identified in this study, indicating that similar structures were shared in all peptides.

Protamine sulfate, a sperm protein of salmon, herring, trout, and other species belonging to the families Salmonidae and Clupeidae, has been identified as an allergen in some patients with sensitivities to salmon, herring, and related fish.[63] It is a low-molecular-weight protein widely used as a heparin antagonist. In one study, surimi, a collection of one or many different varieties of small fish that are minced and washed extensively, gave a single band at 63.5 kDa that bound IgE from all sera of fish-allergic patients.[83] Although many fish-allergic patients are allergic to multiple fish species, monospecificity has been reported to tuna, cod, and swordfish.[57,61] In these studies, subjects with multiple allergies to fish species showed IgE binding to 12- to 13-kDa bands, whereas the monospecifically sensitive subjects showed IgE binding to unique bands at 40 kDa in tuna[57] and 25 kDa in swordfish.[61]

Hamada and colleagues[48] identified a high-molecular-weight allergen recognized by one fish-allergic serum sample in surimi made from walleye pollock. IgE-immunoblotting and amino acid analysis identified the allergen as collagen. In a separate study, a high-molecular-weight allergen was detected after five protein fractions were prepared from bigeye tuna muscle. Five of eight sera samples obtained from individuals known to be fish-allergic reacted to bigeye tuna collagen.[49]

The existence of structurally related parvalbumins in divergent fish species may offer an explanation of cross-reactivity to fish species in certain allergic individuals. Hansen and coworkers[50] noted that in adults with clinical sensitivity to cod, reactions were reported to mackerel, herring, and plaice, and in vitro results showed IgE binding to a single band in the 11- to 14-kDa region of mackerel, herring, and plaice extracts. However, in one case report, an individual had contact urticaria while cleaning dogfish but experienced anaphylaxis after ingesting dogfish. However, consumption of hake, horse mackerel, swordfish, anglerfish, and mollusks, as well as small pieces of ray (Rajidae family, but same class as sharks), was tolerated without issue.[105]

An IgE-binding protein from salmon, named Sal s 1, was identified by in vitro analyses and was shown to be salmon parvalbumin.[78] In one study, almost 50% of cod-allergic patients had clinical sensitivity to salmon.[3] Sal s 1 is described as having two bands at 12 and 14 kDa, and therefore it has at least two isotypic variants.[78] Although tuna-allergic individuals have been noted to have IgE binding to the 12-kDa Gad c 1 codfish allergen,[17] one study noted that raw tuna extracts seemed to lack IgE binding bands in the parvalbumin size range

that were present in extracts of catfish, cod, and snapper.[59] However, Park and colleagues[100] described IgE binding in sera from fish-allergic persons to tuna proteins at 12 to 13 kDa. Sera from fish-allergic patients possessed IgE to bands at 12 to 13 kDa in mackerel, pollock, and cod, and showed unique binding to a band at 19 kDa from saury and 37 kDa from tuna. In one study, canned tuna did not present problems for five fish-allergic children[12]; canning may decrease the allergenicity of tuna for some individuals. However, a case report told of a patient allergic to both canned and fresh tuna, and also to cod[62]; the patient's IgE bound only to proteins greater than 40 kDa in size. Amino acid sequencing has shown that the 12- to 13-kDa band from tuna does not have the same sequence as does cod; this may explain why tuna does not cross-react extensively with other species.[11,53,101]

Although many studies have shown skin prick test cross-reactivity and in vitro cross-reactivity between many species of fish, Glazzaz and coworkers[44] found that amounts of parvalbumin in different fish extracts did not correlate with skin prick tests in all fish-allergic subjects. An important verification tool is the double-blind placebo-controlled oral challenge (DBPCFC). Bernhisel-Broadbent and colleagues[11] found that in 11 subjects demonstrating in vitro allergy to fish, positive DBPCFC occurred to one species in seven subjects, to two species in one subject, and to three in two others. This observation has been also shared in other studies using DBPCFC.[57] Therefore, studies have shown not only that different species of fish have shared allergenic determinants but also that some fish-allergic patients are sensitive to multiple species of fish. Aas[1] found that of 61 fish-allergic children challenged with fish, 34 reacted to all species tested, but 27 could tolerate one of more types of fish. Other studies have found that subjects clinically react to multiple fish.[51,57,77] A negative skin prick test is an excellent predictor of lack of allergy, so skin prick testing is an important tool. However, careful oral challenges should follow to determine what species would be tolerated in fish-allergic patients.

Allergic reactions to fish roe have been reported.[81,114] In one case report, two persons who were not allergic to fish had been sensitized to roe, having negative skin tests to whitefish and rainbow trout but positive skin tests to the roe from the respective fish.[81] Serum was taken from both patients, and IgE-immunoblotting showed a heavy IgE-binding band for the roes, and it was not inhibited by fish extract, which made it a roe-specific allergenic protein. The authors of the study suggested that roe allergy should be explored in patients who test negative to fish but are suspected of having a seafood-related allergy.[81]

Crustacea

The crustacean family includes shrimp, prawns, crabs, lobsters, and crayfish[129] and is a common reported cause of food hypersensitivity. Research indicates that there are shared allergenic and antigenic determinants among the various members of the crustacea[47,68]; however, some reactions are species-specific.[66,88] Also, qualitative differences in the allergenic determinants of different shrimp extracts have been reported[88]; therefore, use of extracts from more than one species in skin testing and CAP tests is recommended. Presence of IgE to either unique or shared allergens may explain an individual's clinical sensitivity to one or more members of this taxonomic class.

Shrimp is the most thoroughly studied of the crustacea allergens. Hoffman and coworkers[54] were the first to partially characterize allergens from shrimp; antigen I was isolated from raw shrimp and found to be an acidic, heat-labile protein composed of two noncovalently bound polypeptide chains with a molecular weight of 21 kDa. Antigen II, isolated from cooked shrimp, was found to be an acidic, heat-stable glycoprotein, composed of 341 amino acid residues and 4% carbohydrate, with a molecular weight of 38 kDa and an isoelectric point of 4.5. Antigen II appeared to be a major allergen for the subjects in this study.

An allergenic tRNA moiety from cooked prawns has been described[97]; however, the researchers were not able to isolate a preparation completely devoid of protein, so it is possible that the allergenicity was caused by RNA-associated proteins. Further work by this group[92] yielded the description of two allergenic polypeptides from cooked shrimp. SA-I had a molecular weight of 8.2 kDa and was not analyzed further. The second allergen, SA-II, was composed of 301 amino acid residues, had a molecular weight of 34 kDa, and appeared similar to antigen I isolated by Hoffman and colleagues.

Daul and coworkers[28] have determined that the major brown shrimp allergen, readily isolated from the boiling water and meat of cooked shrimp, is tropomyosin, a muscle protein. It is composed of 312 amino acid residues and 2.4% carbohydrate. Monoclonal antibodies directed against this 36-kDa allergen (named Pen a 1) reacted with a 36-kDa protein in crayfish, crab, and lobster extracts.[28] Antigen I, SA-II, and Pen a 1 appear to be the same protein. Further work has determined that tropomyosin is major allergen of two additional shrimp species, Par f 1 *(Parapenaeus fissurus)*[76] and Met e 1 *(Metapenaeus ensis),*[71,107] and has been identified as SA-II.[107] Lin and colleagues[76] identified a 74-kDa component in shrimp for which IgE was possessed by 40% of subjects reporting a history of allergy to shrimp and positive skin prick tests. Minor components of 41, 47, 50, and 86 kDa bound to IgE from 10% to 20% of the sera. A monoclonal antibody made against Par f 1 also reacted to a 39-kDa component in crab, and the two bands had similar amino acid compositions. Par f 1 is 39 kDa in size and has an isoelectric point of 5.1 to 5.6 kDa. Ayuso and coworkers[8] described the continuous, allergenic regions of Pen a 1: serum from 18 shrimp-allergic patients was incubated with 46 overlapping peptides representing the length of the tropomyosin molecule. Five major IgE-binding areas, 15 to 38 amino acids long, were identified. The major IgE-binding areas were residues 43–57, 85–105, 133–148, 187–202, and 247–284. These five regions were positioned at regular intervals of approximately 42 amino acids, suggesting a relationship with the repetitive coiled-coil structure of the tropomyosin molecule.

Another shrimp allergen, designated Pen m 2, was reported by Yu and colleagues[127] and was deduced by cDNA sequence. The cloned cDNA encoded a 356-amino acid protein that showed homology to arginine kinase from crustaceans. Pen m 2 was found to react with serum IgE from shrimp-allergic patients.

Crab

Leung and coworkers[73] have described the identification of a crab allergen, Cha f 1, from the crab species *Charybdis feriatus,* from a constructed cDNA library screened with crab-allergic sera. Recombinant Cha f 1 is 34 kDa in size and was identified as a tropomyosin. Snow crab has also been shown to cause allergic sensitization in occupational settings.[21,52] Heat-labile and heat-stable allergens have been found in snow crab extract; snow crab–specific IgE bound more to boiled crab than to raw

crab.[52] The most prominent bands in sodium dodecyl sulfate polyacrylamide gel electrophoresis (SDS-PAGE) were at 37 to 42 kDa in crab cooking water and extracts of cooked crab meat; some patients were processors, and two were seafood restaurant workers. However, in contrast to findings by Leung and colleagues,[73] a 14-kDa protein seemed to be the most prominent IgE binding protein, indicating that in cases of sensitization by the inhalation route, the responsible allergens might be different from those by the ingestion route.

Lobster

Leung and coworkers[74] screened a library made from *Panulirus stimpsoni,* the spiny lobster, with sera from crustacea-allergic subjects. A recombinant IgE-binding protein was designated Pan s 1 and had a molecular weight of 34 kDa. The researchers also found a similar protein in *Homarus americanus,* the American lobster, which was designated Hom a 1. Hom a 1 possesses cross-reactive IgE epitopes in lobster and shrimp and has significant homology to Pen a 1 and Met e 1, the major shrimp allergen. There are similar, if not identical, IgE binding sites in Met e 1, Pan s 1, and Hom a 1. Although the size of Hom a 1 was not directly reported, the work described a 60-kDa fusion protein, which would indicate that Hom a 1 was approximately 34 kDa in size. Wiley and Griffin[121] demonstrated two IgE-binding proteins of 35 to 37 kDa and 97 kDa in extracts of lobster in pooled sera from subject with respiratory symptoms to Norwegian lobster *(Nephrops norvegicus)*. The allergens appeared in native form to be greater than 800 kDa in size and were recognized by greater than 80% of the serum samples involved, making them major respiratory allergens. The 35-to-37-kDa allergens made up between 0.02% and 1% of the total protein, and the 97-kDa allergen made up 7% to 15% of the total protein of Norwegian lobster extracts.

Mollusks

The mollusks are a very diverse group. Squid and octopus are cephalopods; clam, cockles, scallops, mussels, and oysters are bivalves; and abalones, conches, limpets, snails, and whelks are gastropods.

Squid

Allergic reactions have been documented in sensitive subjects after ingestion of squid or inhalation of vapors from cooking squid.[19] Almost all of these victims also experienced symptoms after ingesting shrimp and demonstrated strong positive skin test reactions to boiled squid extracts and various commercial crustacea extracts. Immunoassay studies showed cross-reactivity between shrimp and squid allergens, although cross-reactivity was not demonstrated between squid and octopus or squid and other mollusks. Miyazawa and colleagues[86] have described the major squid allergen as a 38-kDa heat-stable protein, designated Tod p 1. Cross-reactivity between Tod p 1 and shrimp allergens was demonstrated by in vitro IgE binding studies and with monoclonal antibodies. The amino acid sequence of Tod p 1 showed marked homology with tropomyosin from the blood fluke (a snail) and has been determined to be squid tropomyosin.

Oyster

Two allergens, Cra g 1 and 2, were isolated from the oyster *Crassostrea gigas*.[55] They are both 86-kDa glycoproteins with a carbohydrate content of about 5%, and there are 35-kDa subunits. The allergic reactivity of the two allergens was equivalent in oyster-allergic patients. Studies on molecular characteristics and amino acid composition suggested that they are oyster tropomyosin. The allergenicity is apparently heat stable, as is that of Pen a 1, shrimp tropomyosin. Partial digestion with a peptidase gave a protein sequence different from that for IgE-binding characteristics of Pen a 1. Cra g 1 has 76% protein sequence homology with mussel tropomyosin, 74% with the gastropod tropomyosin *Haliotis rufescens,* and 58% with *Metapenaeus ensis* (shrimp) tropomyosin.[55]

Limpet and Abalone

Limpet and abalone can cause moderate to severe anaphylactic reactions, and occasionally, fatalities.[7,18,22,80,89] Sensitive subjects usually have positive skin tests and CAP tests to extracts of the offending shellfish. In one study, five patients were monospecifically allergic to limpet and presented with symptoms of severe asthma after ingestion; 43 other patients in the study were cross-reactive to several species of shellfish and showed a range of allergic symptoms after ingestion.[22] In another study, it was noted that all six limpet-allergic subjects developed severe bronchospasm as their primary symptom after ingestion.[20] The major IgE binding proteins of grand keyhole limpet appear to have molecular weights of 38 and 80 kDa.[80] Another research group[79] found a unique 49-kDa major IgE binding protein (Hal m 1) and another component of 38 kDa that bound IgE in abalone. Although most subjects in the study (38) had immediate reactions to abalone, 13 had delayed reactions (up to 7 hours) after ingestion. Only 17 of these sera were positive by radioallergosorbent test (RAST) to abalone. The abalone-allergic subjects had primarily bronchial symptoms, and those reporting delayed reactions showed predominantly respiratory and cutaneous symptoms. One report[7] indicated that five limpet-allergic individuals were also allergic to dust mites (see Seafood–Insect Cross-Reactions, later).

Snail

In a study of 10 subjects allergic to snails, eight experienced bronchial symptoms, and six reported no skin or gastrointestinal symptoms. All subjects could ingest cephalopods and bivalves without adverse reaction.[30] In another study, asthma symptoms were reported after ingestion of snails by 15% of the subjects, and six different IgE-binding protein bands were found in boiled snail extract, ranging in molecular weight from 12 kDa to greater than 66 kDa. Keyhole limpet hemocyanin did not provoke cross-reactive responses in these subjects. Specific IgE binding was exhibited to bands at 12, 15, 24, and 66 kDa, but these peptides were not characterized further.[4] Many reports of severe anaphylactic reactions to snails involve bronchial symptoms and previous sensitization to dust mites (see Seafood–Insect Cross-Reactions, later).

Horned Turban

One study has reported the major allergen of the gastropod *Turbo cornutus,* a horned turban mollusk and a popular food item in Japan. The major allergen, named Tur c 1, was found to be 35 kDa in size and identified as tropomyosin. However Tur c 1 has an IgE-binding epitope in the c-terminal region that is dissimilar to those in Cra g 1 (oyster) and Pen a 1 (shrimp).[55] *Turbo cornutus* has also been described as causing exercise-induced anaphylaxis.[58]

Shellfish Cross-Reactions

Shrimp-sensitive patients often report clinical histories of allergic reactions to several species of crustacea.[120] Clinical and in vitro evidence for cross-reactions has been shown between mollusks and crustacea.[65,66,67,120] Leung and coworkers[72] found that IgE from subjects who were anaphylactic to shrimp recognized proteins from other crustaceans and also mollusks; tropomyosin was determined to be the cross-reactive allergen. In a study of crustacea-sensitive subjects, specific IgE binding was observed to six crayfish components and four spiny lobster components.[47] One study found clinical association between clam and mussel, but not between clam and mussel and crustacea.[22] There was no association between in vitro IgE binding and clinical history for shellfish. Squid-allergic IgE is cross-reactive with other mollusks.[19] Clinical association can occur within the same and between different phyla, reflecting common allergenic epitopes,[22] as evidenced by one report of cross-reactivity in a seafood restaurant worker allergic to both shrimp and scallops[45]; this patient's IgE bound to proteins in the 35- to 39-kDa region, thought to be cross-reacting tropomyosins. The presence of IgE to unique and shared class allergens may explain an individual's clinical sensitivity to one or more members of a class.

Seafood–Insect Cross-Reactions

Both insects and the crustacea belong to the phylum Arthropoda. Patients allergic to chironomids (nonbiting midges) often demonstrate positive skin tests to crustacea also. Chironomid extracts have been found to inhibit RAST with shrimp, and vice versa,[40] although other researchers report low cross-reactivity.[125] Allergic reactions were reported among aquarists and fish-food factory workers when exposed to chironomids.[75] Symptoms reported most often were conjunctivitis, rhinitis, asthma, and urticaria, and other reports indicate atopic dermatitis on exposure to chironomids in pet foods.[124] The invertebrate hemoglobin (erythrocruorin) molecule might be involved in the reported cross-reactivity of caddis fly and mollusk allergy. Erythrocruorin is a potent allergen for chironomid-allergic individuals, and serum from caddis-sensitive persons in one study reacted with a component of similar molecular weight in mollusk and bee venom extracts.[64] This raises the possibility that individuals exposed to caddis fly antigens could develop allergic reactions during their first exposure to shellfish or their first bee sting.

Tropomyosins may be the major cross-reactive allergens between insects and shellfish. Shared antigenic and allergenic determinants between shrimp Pen a 1 and fruitfly extract have been found, with Pen a 1 sharing 87% homology with fruitfly tropomyosin.[29] In vitro IgE cross-reactivity has been found between boiled Atlantic shrimp and the German cockroach,[35] showing binding in both types of extracts between 30 and 43 kDa. Leung and colleagues[72] found that IgE from shrimp-allergic subjects bound to a 38-kDa band in grasshopper, cockroach, and fruitfly preparations.

Castillo and coworkers[22] found an unexpectedly high prevalence of cockroach sensitivity among limpet-allergic subjects to crustacea and cephalopods. Crustacea and cockroaches are arthropods, but limpets are cephalopods and are less closely related, so this observation is most likely not the result of cross-reactive IgE but of concomitant allergies. In six individuals with anaphylactic reactions to limpet, five experienced severe systemic reactions during dust mite immunotherapy. The investigators theorized that because the limpet filter feeds on sea mites among other small animals, the limpets could be contaminated with mites and thus cause reaction.[20] However, no mite allergen was found in the limpet extracts used. There is no significant cross-reaction between abalone and dust mites.[79]

Cross-reactions are apparently serious between mites and snails or limpet, and there are many reports of severe reactions to snail ingestion in persons allergic to dust mites.[7,31,46,117,119] The sensitizing agent appears to be dust mite for most snail-allergic patients.[118] Allergy to snails was possibly induced in one study on individuals receiving immunotherapy for treatment of dust mite allergy; IgE from the subjects was found to be cross-reactive for snail. Strong increases in in vitro IgE reactivity to shrimp and snail were observed in some patients, and two patients developed oral symptoms after eating shrimp. The IgE response to mite allergens did not increase. The average IgE response to snail did increase, and there were two conversions among 17 patients from negative to positive for snail.[117] Mite-allergic patients had no IgE reactivity to tropomyosin, but shrimp-allergic patients possessed IgE against mite extract,[123] and tropomyosin appeared to be a major allergen for this latter group.[118] However, tropomyosin was not found to be the major cross-reactive allergen in snail-allergic subjects.

The references for this chapter can be found on the accompanying DVD-ROM.

68 Submersion Incidents

Drowning and Near-Drowning

Robert B. Schoene, Arian Nachat, Andrea R. Gravatt, and Andrew B. Newman

One of Nature's greatest ironies is that man should spend the first nine months of his existence continuously surrounded by water, but the rest of his life with the inherent fear of submersion.

—B.A. Gooden

In 18th-century Europe, sudden death by drowning sparked the interest of humanitarians who thought actions could be taken to restore life. The incidence of drowning on commercial waterways was probably high, since roads were nearly nonexistent and swimming as a recreation was unknown among the general populace. Drowning became a substantial public health issue. In response to the fashionable humanitarian cause, the Society for the Recovery of Persons Apparently Drowned was formed in 1774 in England. Their activities included research, public education, and treatment of victims. The Society survives today as the Royal Humane Society.[42] Techniques for resuscitating drowning victims developed after groups with a special interest in the problem of drowning were established.

This chapter reviews the clinical presentation, pathophysiology, prevention, and treatment of near-drowning and drowning and emphasizes the importance of water-related injury and deaths in all forms of water environments.

INCIDENCE

The Global Burden of Disease 2000 estimated that 449,000 people drown worldwide each year, which ranks drowning second to motor vehicle accidents among causes of unintentional deaths.[96] It is the leading cause of death among infants and toddlers.[167] Statistical estimates may be lower than reality due to underreporting, particularly in middle and low-income countries where data are not collected or where collection is inconsistent.[153] Collection of data includes only accidents and does not include cataclysmic events. For instance, in recent years there have been multiple natural disasters including the great tsunami of December 2004, which alone resulted in over 100,000 deaths by drowning and would not be included in accidental drowning statistics. Despite technological advances in warning systems and emergency management, hurricanes, floods and other natural disasters are likely underrepresented in drowning statistics.

In 2000 the National Center for Injury Prevention and Control (USA) reported 3,482 deaths due to drowning. This report did not include boat-related accidents, which are coded differently and in the same year accounted for 701 additional deaths.[133,179] In 2002, the National Safety Council Report on Injuries in America attributed 3000 deaths to drowning, which ranked as the fifth leading cause of accidental deaths.[134] Survivors of submersion events outnumber drowning victims by a factor of 500 to 600, which gives some insight into the cost of medical care for the survivors, many of whom may require prolonged intensive care and suffer lifelong neurologic disability. Although the incidence of submersion accidents is greater in recreational waterways, swimming pools, irrigation ditches, and even bathtubs and toilets are sites of drownings.

Drowning kills mainly the young; 64% of victims are younger than 30 years of age and 26% are younger than 5 years. Drowning ranks fourth in causes of child death in the United States and ranks second only to motor vehicle accidents as a cause of injury death.[7] In Alaska, Arizona, Florida, and California, drowning is the leading cause of pediatric injury death.[134,162] Fortunately, the rates of drowning worldwide have decreased. Trends toward more indoor leisure activities for children, improved socioeconomic conditions, safety measures, and public health education are a few of the reasons for such change.[51,176]

The economic cost of submersion injuries to society is one of the highest for any injury group, since many victims require prolonged hospitalizations and chronic care. Four hundred fifty to $650 million is spent yearly in the United States as a result of drowning.[200] An Australian report estimated that for every child who drowns, four are hospitalized and sixteen receive emergency department care.[129]

CLASSIFICATION OF SUBMERSION INJURIES

Water rescue is used when a person rescued from the water has experienced distress while swimming but is alert and requires minimal intervention that does not require transport to a medical facility. Persons who survive for at least 24 hours following submersion, regardless of the duration and extent of recovery, have been termed *near-drowning victims,* but the term near-drowning has now been replaced by *submersion injury* with all of its clinical consequences regardless of outcome. *Drowning* occurs when submersion results in asphyxia and death at the time or shortly thereafter. If death occurs within 24 hours after submersion, the term *drowning-related death* is used. When victims die quickly at the time of submersion, it is often difficult to determine the cause of death; however, the term

TABLE 68-1. Near-Drowning and Drowning Classification

GRADE	MORTALITY	CLINICAL EXAM	MANAGEMENT
0	0%	No cough or difficulty breathing	Release at scene
1	0%	Normal auscultation with cough	Rest, warm, reassure, and release
2	0.60%	Rales, small amount of foam, radial pulses	Oxygen via nasal canula, observe for 6 to 24 hr
3	5.20%	Acute pulmonary edema with radial pulses	Oxygen via NRB, ACLS, admit
4	19%	Acute pulmonary edema with hypotension	Oxygen via NRB, prepare for ETT/pressors, ICU
5	44%	Respiratory arrest	ACLS on scene, respiratory support, ICU
6	93%	Cardiopulmonary arrest	ACLS on scene, CPR/intubation, ICU

ACLS, advanced cardiac life support; CPR, cardiopulmonary resuscitation; ETT, endotracheal tube; ICU, intensive care unit; NRB, nonrebreather mask.
Adapted from Anonymous: Submersion or near-drowning. Circulation 102(Suppl I):I233-I236, 2000; and Szpilman D: Near-drowning and drowning classification: a proposal to stratify mortality based on the analysis of 1,831 cases. Chest 112(3):660–665, 1997.

immersion syndrome is often used when death occurs shortly after immersion in water at least 41°F (5°C), much colder than body temperature, which is generally attributed to cardiac arrest from cold-induced bradycardia, tachycardia, or ventricular fibrillation.[47,104,145]

Although submersion is often classified by the pathophysiology, it is more useful and practical to characterize submersion injuries by the setting and clinical outcome because this approach better prepares medical personnel in treatment of the victim. For clinical purposes, classifying the presentation of the illness by severity provides a sound base from which to institute therapy. A classification scheme that focuses on the cardiopulmonary status, and goes from the best to the worst outcomes, is presented in Table 68-1.[14,15,18,47,145,184] This classification shows that one can predict an increase in mortality from 0% to 93% from grade 1 to grade 6. Some of the pathophysiologic conditions associated with submersion are discussed later.

THE HUMAN BODY AND WATER

The human body has little natural buoyancy. The degree to which the body floats depends on air in the lungs, body fat, and clothing. For instance, when the lungs are maximally filled with air (i.e., close to total lung capacity), the body has about 2.5 kg (5.5 lb) of flotation, little more than the weight of the head. Thus, the body goes through rapid phases of greater buoyancy during inspiration and lesser buoyancy during expiration. The percentage of body fat influences buoyancy to a lesser degree than do lung air volumes but is an important factor in prolonged immersion in water related to the ability to conserve body heat. Clothing worn by the victim at the time of submersion has a profound effect on both buoyancy and heat conservation and is often the factor that dictates survival or death, particularly in the wilderness environment.

The water environment to which the human body is not naturally adapted exerts deleterious influences on respiratory physiology. For example, atmospheric pressure increases as one is further submerged. The increase in pressure on the chest wall greatly increases the work that the respiratory muscles must exert to inhale. Maximal inspiratory pressure that is routinely measured in clinical settings is no more than negative 80 to 100 cm H_2O in a healthy adult; thus, submersion in water as shallow as 100 cm prevents normal inspiration, which affects both normal alveolar ventilation and buoyancy. Therefore, staying as close to the surface of the water for as long as possible by any means of flotation may be critical to survival.

Depending on the depth of submersion, pressure exerted on the limbs prevents blood from pooling in the peripheral veins, which causes an increase in intrathoracic blood volume of up to 500 mL; the resulting increase in cardiac output and central venous pressure may not be tolerated by subjects with underlying heart disease. Healthy subjects who experience this increase in intrathoracic blood volume often are afflicted with a diuresis from release of natriuretic hormones, which explains in part why so many immersion victims have a seemingly paradoxical decrease in intravascular volume.

THE INITIAL EVENT

The typical sequence of events after submersion is an initial period of panic and struggle with breath holding. Laryngospasm to protect the airway occurs while frantic attempts to hold one's breath are interspersed with strong attempts to inspire, consuming a great deal of energy. The result of this struggle is hypoventilation with hypercapnia and hypoxemia. When asphyxia occurs, the victim becomes unconscious; laryngospasm abates while inspiratory attempts are ongoing, and water passively enters the airways. Cardiopulmonary arrest follows. Struggling does not always follow submersion, especially in trauma-associated submersion, where the victim may have been rendered unconscious before or upon entry into the water.[47,104,145]

RISK FACTORS

The problem of submersion incidents can often be related to risk factors (Box 68-1) or to certain behaviors or groups. This is particularly true of children.[150] Understanding these factors can help in planning and instituting preventive measures and practices.

Box 68-1. Factors Common to Drowning Incidents

Age: toddlers, teenage boys
Location: home swimming pools
Gender: males predominate in every age group
Race: black children most at risk
Drugs: particularly alcohol
Trauma: secondary to diving, falls, horseplay

Age

Young people have the highest likelihood of drowning. A bimodal age distribution characterizes drowning in children, with children under 1 year of age and adolescent males accounting for the peaks. Children between the ages of 5 to 14 years had the lowest risk of drowning.[163] Ten percent of children in South Carolina under 5 years of age experienced a serious threat of near drowning.

Young children are most vulnerable to drowning. This is due to their lack of awareness of danger, curiosity, poor swimming techniques, and the size of their heads in relation to body size, making them susceptible to falling into containers such as pails and buckets. Poor supervision has been attributed to childhood drowning involving bathtubs and pools, especially in toddlers. Despite the American Academy of Pediatrics recommendation never to leave a child unsupervised in the bathtub, one study reported that nearly 33% of parents left their child unattended in this setting.[178] Shared bathing with young children and a sibling younger than 2 years with brief unsupervised time was reported to account for 22% to 58% of bathtub drownings.[37]

Drowning in male teenagers is due to risk-taking behaviors, lack of supervision, and drug and alcohol intake. The scene of drowning in this age group is most commonly natural bodies of water, such as lakes, ponds, and rivers. Incidents often occur far from medical assistance and may take place where rescue is challenging.[6,7,34,164]

In people older than 65 years, deaths due to bathtub-related drowning often result from falls[36] and exacerbations of concomitant illnesses, such as cardiac arrhythmias, which may not be recognized at autopsy.[163] Otherwise, drowning in older adults is rare.

Gender

In 2000, males accounted for 79% of all drownings in the United States.[133] This fact is thought to be largely due to risk-taking behaviors characteristic of adolescent males. Beyond the toddler age, boys have a three times greater risk of drowning.[104] Females in all age groups, in comparison to boys, have a more constant and lower rate of drowning.[173] Drowning in females peaks at age one year and declines throughout the rest of life.

Race

Race plays an important role in the prevalence of drowning. In Alaska, drowning rates are twice as high in Native American children as in white and black children.[11] In the lower states, Native Americans between 15 and 19 years of age also had the highest rate of drowning.[179] In the 10- to 14-year-old age group, drowning rates are highest among African Americans and lowest in whites.[34,130] In another study, black males of the same age group had nearly three times the drowning death rate compared to white males.[173] This race-related statistic is reflected in the military, where black soldiers drown with a frequency 62% greater than that of white soldiers.[19] The reasons why there are such great differences in drowning rates between races are unclear, but socioeconomic factors that afford safety factors, such as education and swimming ability, are implicated.

Location

Any body of water can be the site of drowning. Oceans and raging rivers, in fact, account for a minority of submersion events while backyard pools, recreational lakes, bathtubs, and pails of water claim the vast majority of lives. In rural areas, having a home well increases the risk of a child's drowning by sevenfold. In the United States, submersion injuries involving children occur primarily in fresh water. Seventy-five percent of drowning occurs in bodies of water at the child's home and 50% of freshwater drowning occurs in pools, a tragic statistic that has prompted much attention and educational efforts. In spite of these efforts, a small proportion of home pools are properly protected, so they can still easily be accessed by adventuresome youngsters and teenagers. In Australia, an overwhelming majority of drowning accidents occurred in unfenced or unlocked pools. Only 2% of childhood drowning occurs in salt water.[202]

Children under 1 year of age and elders older than 65 years drown most frequently in bathtubs.[34,163] Women choose a variety of settings for childbirth, and immersion in water for a portion or all of labor has gained popularity.[136] These "water births" have been associated with drowning.[64] Toilets and buckets can be drowning vessels for young children. Children ages 1 to 4 years drown most commonly in pools or were boating when they drowned.[6,7,18,25,34,130,163,164] On the other hand, natural bodies of water were the sites of drowning in 63% of older children and the elderly.[34,163] This finding is even more common in cold-water, coastal communities, where livelihoods are more commonly made near or on the water.

Travel away from home to an environment where water is accessible for work or play places one at a higher risk for drowning. A number of fatalities have been reported during adventure travel trips. Foreigners and tourists experienced a fourfold greater risk in drowning; speculation was that familiarity for the scene was lacking.[108] Ironically, scarcity of water does not preclude drowning. For example, the highest drowning rate in California in a 10-year study was a rural desert county.[3]

Ability to Swim

Although swimming programs for very young children have proliferated, there are no studies showing that they prevent drowning.[5,100] Such programs may offer a false sense of security and encourage exposure to more hazardous or unguarded bodies of water.[31,33,164] For this reason, the American Academy of Pediatrics does not endorse swimming instruction for infants and toddlers.[5,6,7]

Good swimmers are not immune from drowning. Even competitive swimmers and divers can drown as a result of hyperventilation prior to competition. The initial respiratory alkalosis and hypocapnia blunts respiratory drives, and in certain individuals with inherently low hypoxic and hypercapnic drives, failure to breathe may result in severe hypoxemia. The subsequent hypoxic state results in unconsciousness and aspiration.[57,121]

Parental example is an important determinant of whether children can swim. Children whose parents can swim are most likely to be strong swimmers; those with parents who can not swim are likely to be nonswimmers.

Drugs

Alcohol has been implicated as a contributing factor in 25 to 50% of recreational water-related deaths.[35,106,108,129] One study demonstrated blood alcohol levels of 100 mg/dL or more as a factor in 25% of boating-related fatalities,[35] in 25% of teen drownings,[164] and in 33% of drowning deaths of young adults ages 20 to 34 years. In Australia, 64% of males who drowned had measurable levels of alcohol in their blood. Despite laws

that prohibit alcohol use during recreation on the open water, a survey in Massachusetts revealed that 36% of men and 11% of women had consumed alcohol while on the water.

Loss of judgment from the use of intoxicants can lead to accidents as well as contribute to increased loss of body heat, decreased laryngeal reflexes, and higher risk of aspiration.[163] Illicit recreational drug use with and without alcohol has also been reported as a factor in water-related deaths.[35] All types of drugs that affect judgment can result in over-crowding in the water craft, speeding, failure to wear life jackets, and reckless handling of boats.

Preexisting Disease

Risk in water accelerates rapidly when medical conditions decrease the chance for self-rescue and survival. Situations are particularly dangerous when the medical condition occurs unpredictably. Cardiac and neurologic conditions are classic examples of impending catastrophes. Individuals with preexisting conditions who wish to venture into water must take special care never to be alone and to optimize treatment for the known condition.

Seizure disorders have been implicated in drowning in natural bodies of water and bathtub submersions in children younger than 5 years.[22,30,164] Children with therapeutic and subtherapeutic anticonvulsant drug levels are at risk.[54] Other central nervous system abnormalities, such as arteriovenous malformations presenting as seizures, have also been reported with drowning.[66]

Any underlying disorder of cardiac rhythm may become manifest in water[1,27] although the mechanism for the degeneration of rhythm disturbances remains unclear.[1] The long QT syndrome is a conduction abnormality that is associated with sudden death and has been linked by forensic molecular screening with etiology of some drowning deaths.[2,111] Adverse events from other more common cardiac diseases, such as coronary artery disease, may lead to drowning.

Drowning by suicide is a rare event.[38,195] In some countries such as Denmark, however, it is a major cause of death.[108] Depression and other psychiatric disease are predisposing factors. Sober white men older than 40 years comprise a large percentage of these suicide victims, who often choose lakes, rivers, and ditches in which to drown themselves.[38,163,195] Suicidal deaths in young women, on the other hand, are uncommon, but when they do occur, the site is often the ocean or bathtub.[38]

Rescuers and medical personnel must be aware of medical factors that will affect the resuscitation. Trauma, especially in diving or boating accidents, must be suspected. Head or cervical spine injuries are particularly common. Metabolic disorders, such as hyper- or hypoglycemia, must be anticipated so that the appropriate therapeutic interventions may be instituted.

Child Abuse and Homicide

Submersion incidents as a manifestation of child abuse have drawn attention such that all suspicious events must have a thorough social service and legal investigation within the laws of the community. Unfortunately, a minority of drownings are ever reported to social services. Abuse or neglect accounted for 19% of bathtub submersion victims younger than 5 years,[164] and must be suspected in most cases, especially in children who have been previously abused. Drowning deaths due to teen homicide are rare.[179]

Boating-Related Drowning

Worldwide, recreational and commercial boating are leading causes of unintentional drowning.[100,108] Over 50% of boat-related drowning occur as a result of capsizing or falling overboard, and factors affecting the risk for drowning in these settings include water depth, current, including hydraulic currents at the base of dams and spillways, distance from shore, and water temperature. In the U.S. in the year 2000, 86% of boat-related drowning involved persons who were not wearing a protective flotation device.[134] Other factors, including swimming skills, safe-boating practices, and alcohol, also contribute to the incidence of drowning in boat-related accidents.[35,106]

The commercial fishing industry contributes a substantial portion of deaths due to drowning.[165] In the Alaskan commercial fishing industry, over 90% of deaths were from drowning, primarily as a result of vessels capsizing or workers going overboard into rough waters.[107] Refugees seeking asylum drown as a result of poor weather conditions and overcrowded boats, and from exposure and poor navigation aids or charts.[10,160] In a recent report, up to 4000 asylum seekers are estimated to drown at sea yearly.[160]

CLASSIFICATION AND TYPES OF DROWNING

Although the pathophysiologic and clinical endpoints of submersion accidents are often similar, it is helpful to understand the presumed physiologic mechanisms of different circumstances. The following classification provides guidelines for different settings.

Wet drowning occurs in 80% to 90% of submersions and refers to events when victims aspirate fluid. After initial laryngospasm abates and the glottis opens, the unconscious victim aspirates water and subsequently develops pulmonary edema.[87,121,149,202]

Dry drowning occurs 10% to 15% of the time.[198] Laryngospasm is the first reflex that initially protects the airway and thus the victim from aspirating water, thereby preventing pulmonary edema. Development of pulmonary edema in this setting is presumably secondary to fluid extravasated from the intra- to extravascular space in the lung parenchyma secondary to high negative intrathoracic pressures from attempts to inspire on a closed glottis. This high negative pressure is subsequently exerted in the interstitial spaces and presumably leads to fluid leak into the extravascular space.

Although one might assume that victims who do not aspirate will respond better to resuscitation measures, no evidence supports this notion. The victim's response depends more on rapid reversal of hypoxia than on the type of submersion injury. Acute and chronic lung responses to various amounts and types of fluids are discussed later. Persons with preexisting pulmonary disease are at greater risk for decompensation if they have a submersion incident.

Shallow water syncope or *blackout* occurs in individuals who hyperventilate prior to a prolonged underwater submersion or exertional swim, usually in a competitive setting. Hyperventilation leads to marked respiratory alkalosis with only modest increase in lung oxygen content. Hypercarbia is a more potent stimulus than hypoxia to breathe. By hyperventilating, the victim resets his hypercarbic drive, which is exacerbated by strenuous activity that depletes oxygen stores. The subject may

become profoundly hypoxic and syncopize underwater. Individuals with inherently low ventilatory responses to both hypoxia and hypercapnia may be particularly susceptible to this type of accident.

Immersion syndrome is defined as sudden death from bradycardia, tachycardia, or vagally induced ventricular fibrillation and cardiac arrest from cold-water contact (at least 5°C less than body temperature) before body cooling has occurred. Vagal stimulation from cold receptors may lead to cardiac arrest and death.[70] The mechanism of this response may be attributed to the vestigial mammalian diving reflex, which is an important survival mechanism in diving mammals that results in the most bioenergetic allocation of energy needs while the animals dive for food. The reflex involves economical distribution of blood flow from little-used tissues to muscles of locomotion that permit the animal to perform its tasks. Cutaneous receptors trigger shunting of blood to the brain and heart from the skin, splanchnic circulation, and extremities. There is accompanying bradycardia. Peripheral oxygen utilization is minimized, and brain metabolism is preserved.

In humans, there is a wide spectrum of this response that permits some individuals a higher chance of survival. Certain persons have less arterial oxygen saturation during nonexertional breath-holding in water than while not immersed. This response may be modified by training, as reflected in Korean and Japanese pearl divers, as well as in other trained breath-holding divers who exhibit more diving-related bradycardia than do their nondiving counterparts.[9,172,181]

Immersion hypothermia occurs as the core temperature falls through surface cooling, which is discussed later in the chapter.

Cold-shock response is the most common cause of drowning in cold water. This response affects both respiratory and cardiac systems. Upon exposure to cold water, people experience uncontrollable gasping that lasts approximately 1 minute. This phenomenon may result in aspiration of water unless the head is kept above water level. Sudden skin cooling results in increased peripheral vascular resistance of superficial blood vessels. Heart rate and cardiac output increase. Outpouring of catecholamines may predispose the victim to fatal arrhythmias. Cooling the body periphery decreases nerve conduction. Muscle control becomes difficult, making any attempt at self-rescue virtually impossible.

TYPES OF WATER

Victims may drown in a variety of fluids ranging from fresh pool water to sewage water. They may aspirate impurities and foreign materials, including algae, bacteria, sewage, chemicals, and sand. Much has been written to distinguish the pathophysiologies of fresh and salt water submersion, but the amount of water aspirated, not the type, is of prime importance. Aspiration of more than 11 mL/kg of body weight and more than 22 mL/kg is required to alter blood volume and electrolytes, respectively. It is unusual for near-drowning victims to aspirate more than 3 to 4 mL/kg of fluid. Therefore, most extreme derangements were noted postmortem and are of indeterminate clinical significance.[17,121,124,127,128,145] Because the final common pathophysiologic pathway is similar, the theoretical differences have little clinical relevance, and some recommend discontinuation of this classification so that the focus can be on the common pathway of hypoxemia, pulmonary injury, and resultant multiorgan system failure.[14,15,66,119,145,149] Discussion of the differences, on the other hand, stimulates consideration of disease mechanism and leads to speculation as to why there seems to be a higher mortality in salt than in fresh water.

To look into potential pathophysiologic differences of salt water versus fresh water drowning, Orlowski and colleagues[144] studied the ability to resuscitate dogs after instilling fluids of various tonicities into the lungs. The six fluids were sterile water and 0.225%, 0.34%, 0.9%, 2%, and 3% sodium chloride solutions. Each of these solutions was tested with and without chlorine. The dogs were anesthetized, intubated, and ventilated with room air. Twenty mL/kg of each fluid was instilled into the endotracheal tube, which was then clamped for 5 minutes before resumption of ventilation with 100% oxygen and 10 cm water of positive end-expiratory pressure (PEEP). Three control dogs were made anoxic for 5 minutes by occlusion of the endotracheal tubes without fluid instillation.

The results showed that chlorine made no difference in any group but that 0.225% saline solution had the least deleterious effect on gas exchange or Pa_{O_2}/FI_{O_2} ratio. Sterile water was the most damaging to the lung. Dogs who had cardiac arrest showed no difference with any of the solutions with respect to their salvageability. The results suggest that cardiovascular changes are a result of anoxia and not of the salinity of the aspirated water. The authors further suggested that pools should have 0.225% salinity. To our knowledge, this recommendation has never been widely adopted.

Fresh Water Drowning

Fresh water is hypotonic to plasma and passes readily out of the alveolus into the circulation and causes disruption and washout of surfactant. These events can cause theoretic hemolysis if enough water is aspirated, but certainly cause changes in lung compliance, alveolar instability, and alveolar collapse. Microatelectasis causes ventilation/perfusion (V/Q) mismatch and shunt, resulting in hypoxemia. The alveolar membrane damage further results in impaired alveolar fluid clearance. Capillary damage results in violation of integrity of the endothelial lining and leakage of high protein fluid from the intra- to extravascular space. Disruption of lung parenchymal architecture leads to an inflammatory response and worsening edema. As little as 1 to 3 mL/kg body weight of aspirated water have been associated with a significant fall in Pa_{O_2} and decrease in pulmonary compliance.

Fresh water aspirated into the lungs is rapidly taken up into the circulation; however, except for experimental animal models of drowning, the volumes are seldom large enough to cause significant changes in electrolyte concentration or hemodilution. Hyperkalemia and high concentrations of free hemoglobin from hemolysis[29] and hemodilution have been reported. Increases in central venous pressure (CVP) are transitory.[18,57,104] In the clinical setting, such changes sufficient to merit therapy are rarely seen.

Saltwater Drowning

Salt water is hypertonic and three- to fourfold greater in osmolality than is plasma.[43,57,104] Hypertonicity (approximately 3.5% sodium chloride) causes inflammation of the terminal bronchioles and alveoli and produces an osmotic gradient between plasma and the alveolar fluid. Intra-alveolar fluid accumulation causes pulmonary edema of highly proteinaceous fluid, V/Q mismatch and shunting.[18] Pulmonary edema through aspiration

TABLE 68-2. The Dead Sea's High Salinity

	Na	Cl	K	Ca	Mg
Dead Sea	1493	6092	970	1709	6987
Mediterranean	531	630	11	48	273

Data from Yagil Y, Stalnikowicz R, Michaeli J, Mogle P: Near drowning in the Dead Sea: electrolyte imbalance and therapeutic implications. Arch Intern Med 145:50–53, 1985.

is compounded by direct injury of the alveolar-capillary membrane by the hypertonic saline.[62,87,127] Some believe that alveolar epithelial damage is minimal, as there is rapid clearance of edema fluid by reabsorption.[43] Theoretically, if the fluid shift from the intravascular space to the alveolus is great enough, intravascular volume could decrease. As in fresh water drowning, electrolyte derangements and volume shifts are rarely large enough to be of any clinical significance.

A unique situation occurs with drowning in the Dead Sea, where there is an unusually large solute load. Dead Sea sodium, chloride, potassium, calcium, and magnesium levels (mmol/L) are exponentially greater than in the Mediterranean Sea[197] (Table 68-2). A mortality rate of 50% was reported in one study of Dead Sea drowning victims. These specific deaths resulted from pulmonary complications as well as from arrhythmias induced by severe imbalances of sodium, calcium, and magnesium.[119,149,197]

Contaminants

Contamination by debris from petroleum products, sewage, sand, and organic matter is more common in salt water and brackish waters. Such debris merely accentuates the possibility of further inflammation and lung injury, which increase mortality. Calcium salts, frequently used near oil drilling equipment, may render near-drowned divers hypercalcemic. Water contaminated by chemical wastes, cleaning solvents, detergents, or disinfectants may induce further fluid accumulation in the already damaged lung. Inhalation of mud, sand, and other particulates (Figs. 68-1, 68-2, and 68-3) may require fiberoptic bronchoalveolar lavage to cleanse the airways. Aspiration of microbes and related potential clinical manifestations are discussed later.

PATHOPHYSIOLOGY

The effect of submersion on mammals was first reported in the scientific literature in the late 1800s. In his classic monograph, Greene[76] noted that "inundation of the upper airway, the bronchial tree and segments of the alveolar spaces blocks gas exchange in the lung and produces asphyxia. Thus drowning involves the rapid development of hypoxia, hypercapnia, and acidosis with the associated sequence of hypertension, bradycardia, hyperpnea, apnea, and terminal gasping." Findings in animals were consistent with drowning episodes later observed in humans.[87,110,137]

The initial response in humans to submersion, particularly in cold water, includes a period of panic, accompanied by hyperventilation caused by stimulation of thermal receptors in the skin. Breath-holding may be attempted, but eventually there is a breaking point, mediated by progressive hypercapnia, hypoxemia, or both, where no voluntary efforts can prevent respiration. At the breaking point, deep inspiration results in aspiration of large amounts of water. If the water is particularly cold, hypothermia may be facilitated. Aspiration of large amounts of water can lead to vomiting and further aspiration of gastric contents. In most victims of submersion, violent struggling occurs before loss of consciousness; exercise-induced acidosis leads to greater hyperventilation and risk of aspiration of water (Fig. 68-4).

The initial injury is often survivable, but the cascade of ensuing organ failures from hypoxemia and malperfusion characterizes the inexorable course of many victims. The duration of hypoxemia and tissue dysoxia are significant factors in the

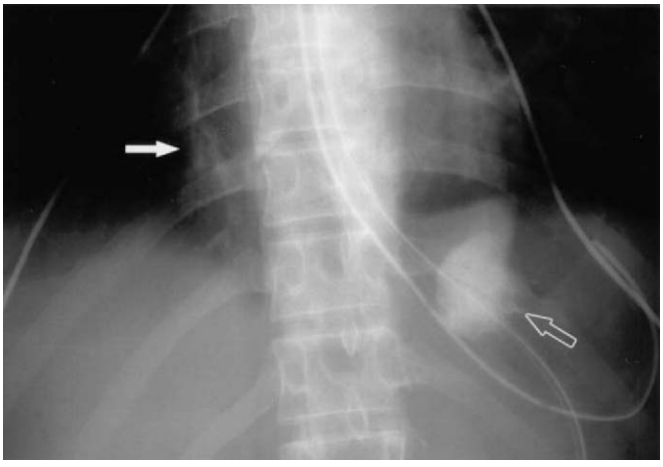

Figure 68-1. Chest radiograph taken within the emergency department, showing sand bronchograms in the right lower lobe *(solid arrow)*. Note sand within the gastric fundus *(open arrow)*. (From Dunagan DP, Cox JE, Chang MC, Haponik EF: Sand aspiration with near-drowning: Radiographic and bronchoscopic findings. Am J Respir Crit Care Med 1997; 156(1):292–295.)

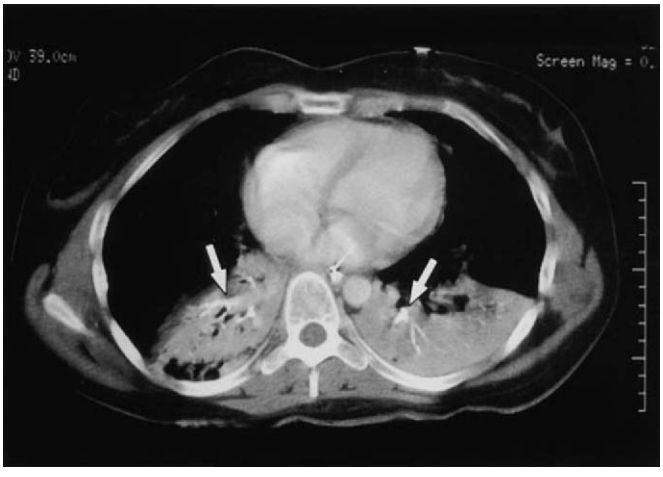

Figure 68-2. Chest CT scan showing bilateral sand bronchograms within the lower lobes *(solid arrows)* as well as significant air space opacification. (From Dunagan DP, Cox JE, Chang MC, Haponik EF: Sand aspiration with near-drowning: Radiographic and bronchoscopic findings. Am J Respir Crit Care Med 1997; 156(1):292–295.)

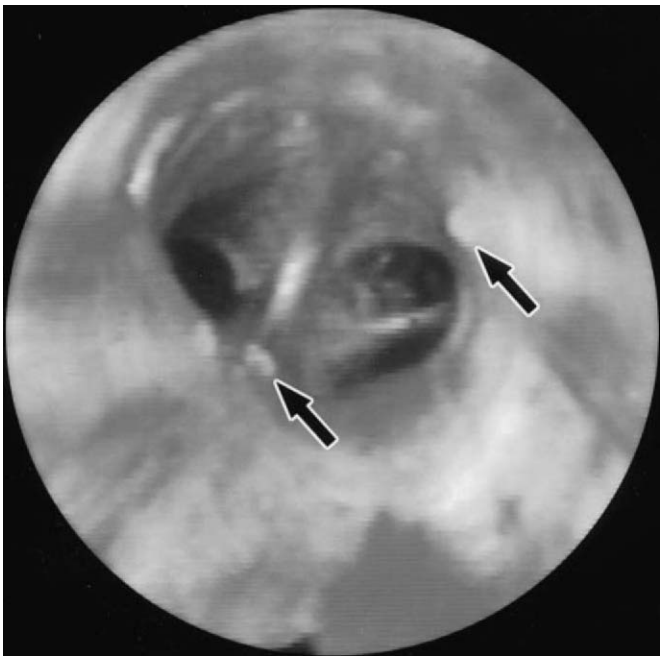

Figure 68-3. Fiberoptic bronchoscopy, demonstrating pieces of sand *(black arrows),* significant airway erythema, and inflammation following near-drowning and sand aspiration. (From Dunagan DP, Cox JE, Chang MC, Haponik EF: Sand aspiration with near-drowning: Radiographic and bronchoscopic findings. Am J Respir Crit Care Med 1997; 156(1):292–295.)

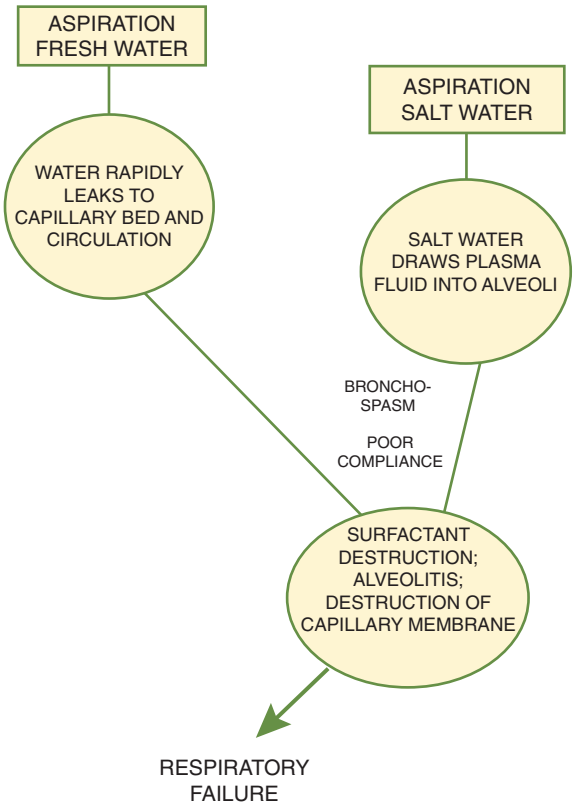

Figure 68-5. Pulmonary effects of water aspiration.

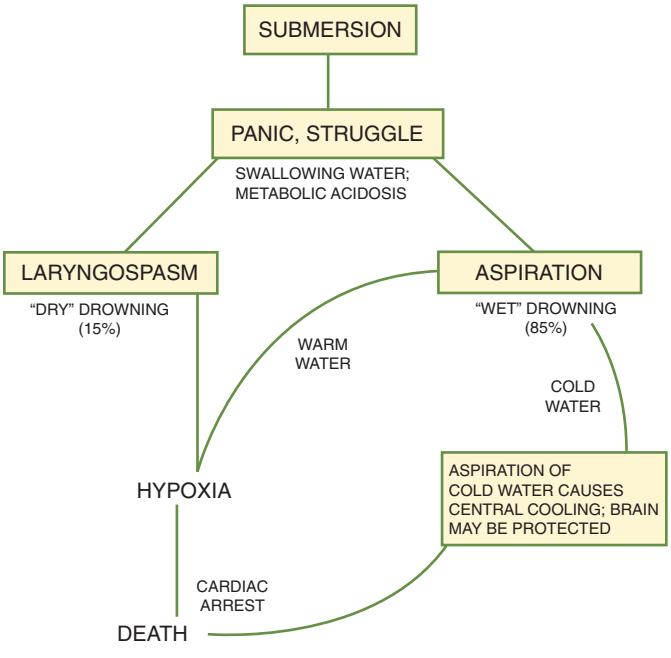

Figure 68-4. Progression of the drowning incident.

ultimate outcome. History of the submersion event can be crucial for prognostication, but estimates of submersion time are notoriously inaccurate. In one case, rescuers assumed a prolonged submersion and delayed instituting CPR in a submersion victim recovered from 11°C (52°F) water until a rhythm was detected on electrocardiogram. The victim was hypothermic and responded almost immediately to resuscitative efforts, ultimately surviving to leave the hospital.[157] With the knowledge of this grim scenario, it is the first job of rescuers to optimize life-saving techniques at the scene and in transit. Critical care physicians then must minimize organ failures that increase mortality. The following section describes the effects of submersion on the body's organs.

Pulmonary

In submersion, the lung is the first interface between the environment and the body. Aspiration of water of any type immediately affects alveolar ventilation, gas exchange, and mechanical characteristics of the lung (Fig. 68-5).

Hypoxemia is the hallmark of pulmonary pathophysiology in submersion. It is a result of both hypoventilation and gas exchange abnormalities. If apnea occurs, the victim becomes hypercapnic, which decreases alveolar oxygen concentration and arterial partial pressure of oxygen. From a gas exchange perspective, inhalation of fresh, salt, and brackish water causes washout of surfactant with subsequent alveolar collapse, leading to areas of low ventilation-perfusion relationships and

shunt, both of which compound hypoxemia. Surfactant is produced by type-2 pneumocytes, epithelial lining cells that ensure maintenance of surface tension and patency of the alveoli, which maintain ventilation/perfusion relationships. Salt or brackish water or inspired debris is more prone than fresh water to result in an inflammatory response and damage to the alveolar epithelial lining with extravasation of fluid into the alveolar space. Aspiration of gastric contents may exacerbate the pulmonary injury.[23,145]

If further inflammation or infection ensues, interstitial and microvascular damage occur with fluid spilling out of the intravascular space and contributing to worsening gas exchange. Hypoxic pulmonary vasoconstriction may decrease the quantity of blood flow to the shunted areas, but the amount of aspiration usually overwhelms the lungs' attempt to preserve gas exchange.

Extravascular lung fluid decreases lung compliance and increases the work of breathing. In more severe cases of submersion, the victims present with all the markers of adult respiratory distress syndrome (ARDS): noncardiogenic pulmonary edema, bilateral fluffy infiltrates, poor lung compliance, and hypoxemia. If infection or inflammation persists, the lungs become stiffer and gas exchange worsens. This condition may be the seminal event for the progression of inflammation, which, in and of itself, has a reasonable prognosis (~70% survival). But comorbid events or infection can occur, in which case multiorgan failure may ensue, and the prognosis is gravely worsened.

If the victim survives respiratory failure, the lung may return to normal function. If the clinical course is prolonged, collagen deposition occurs with resultant fibrosis and decreased lung compliance, restrictive lung disease, and decreased diffusion capacity for carbon monoxide on pulmonary function testing.

Cardiovascular

The effect of submersion on cardiac function runs the spectrum from no effect to complete cardiovascular collapse. The primary metabolic end-products of submersion, hypoxemia, acidosis, electrolyte disturbances, and hypothermia, individually and together, can cause profound abnormalities in cardiac rate and rhythm that cause sudden death or complicate an already grim and complicated clinical situation. These events can occur in healthy individuals and can occur earlier in the clinical course of patients with preexisting cardiovascular disease.

The effect of submersion on heart rate can be unpredictable and quite variable. Initially, an increase in catecholamines from stress can cause sinus tachycardia. Individuals with underlying cardiac disease, hypoxemia, mixed metabolic and respiratory acidoses, or electrolyte derangements are predisposed to more lethal alterations in heart rate and rhythms, such as ventricular tachycardia or fibrillation.[57]

On the other hand, cold-water immersion can result in bradycardia in some victims, secondary to a vestigial diving reflex that provides little survival benefit in the acute situation. If submersion is sufficiently prolonged and hypothermia ensues, bradycardia naturally occurs. The treatment of bradycardia during hypothermia is discussed later and in Chapter 6. Acute cold exposure can also cause increased pulmonary artery pressure,[152] which in the face of depressed myocardial function further diminishes cardiac output and peripheral perfusion. This in turn accentuates the accumulating metabolic acidosis. In an otherwise healthy individual, however, cardiac dysfunction is generally reversible, depending on the length of anoxia, duration of hypothermia, and availability of effective rescue techniques.

Central Nervous System

The brain is the most susceptible organ to the negative effects of submersion events. The focus of all resuscitative measures must be toward restoration and preservation of neurologic function. Basic life support and intensive care measures are aimed primarily at this goal. Without early intervention, all further attempts at therapy, no matter how sophisticated, will be futile to restore meaningful neurologic function. In this light, the most important step is to ensure brain blood flow, for without perfusion to the brain no degree of blood oxygenation is helpful. Other pharmacologic or metabolic (induced hypothermia) interventions aimed at preservation of the central nervous system must be considered experimental in comparison to maintenance of hemodynamic function.

As can be surmised, death from neurologic asphyxia is the most common cause of mortality in acute drowning. Neurologic dysfunction both functionally and electroencephalographically occurs within the first 20 to 30 seconds of sudden cessation of blood flow. Global hypoxic ischemic damage results in both cell death and demyelination.[90,115] Immunohistochemical examination of the brain at autopsy of submersion deaths indicates that the hypoglossal nucleus of the brain stem is severely damaged and thus may be the most vulnerable tissue to asphyxia,[73] while patchy or more global damage throughout the brain is not uncommon. After initial ischemia, reinstitution of blood flow can lead to reperfusion injury, which is characterized by cell lysis and brain edema.

It must not be forgotten that brain injury at the time of a submersion accident may also be secondary to trauma. If trauma occurs at the time of the accident, one or more of many types of brain injury can occur, so these must be suspected at the time of hospitalization (see Management). Neck and spinal injury, skull fracture, subdural hematoma, and intracerebral contusion or hemorrhage are examples of such pathology.

Renal

Acute renal failure is reported uncommonly in the literature as a result of near-drowning. The nature of the accident and time to resuscitation both play roles in the susceptibility to and extent of renal injury. Acute muscle damage and subsequent rhabdomyolysis can result in acute renal insufficiency and failure and may present with myalgias, stiffness, weakness, malaise, low-grade fever, and dark brown urine.[26,103] Excessive shivering, seizures, and muscle ischemia as a result of cold exposure and hypoxemia have been implicated as etiologic mechanisms from rhabdomyolysis.[26] Serial tests for creatine kinase levels, renal function, and urine myoglobin measurements should be done. Management protocols for hyperkalemia, rhabdomyolysis, and renal dialysis, if necessary, should be instituted. Renal failure can also occur without rhabdomyolysis in patients who have had hemodynamic compromise during resuscitation in the form of acute tubular necrosis which, if perfusion is restored, has a good prognosis.

Hematologic and Electrolyte Disturbances

Much has been made of hematologic and electrolyte abnormalities in near-drowning, but from a practical clinical standpoint, such derangements are rarely seen. Most of the data have

come from experimental animal models that, from a theoretical perspective, provide provocative insights.

Some of the data for animal experiments have been discussed earlier. Massive instillation of fresh water in the trachea of animal models induces hemodilution and hemolysis. Hemoconcentration and disturbances of magnesium, calcium, and sodium have been documented with large quantities of experimental salt-water aspiration.[43,197,201] The presumed mechanisms in each case are a result of osmotic gradients across the alveolar epithelium that lead either to dilution or concentration of the blood. It is, however, unusual for humans to aspirate more than 3 to 4 mL/kg of body weight.[80] As noted previously, fluid shifts can cause blood volume changes after 11 mL/kg of fluid aspiration directly into the lung, while 22 mL/kg of fluid aspiration must take place before electrolyte changes occur.[124,127,128] In submersion victims, however, such volume shifts are rare and, if they happen, are transient. They are now thought to have little clinical relevance. Routine observation and treatment, if necessary, are all that are indicated in the hospital setting.

MANAGEMENT

The Element of Time

Rarely does time play as significant a role in determining the ultimate outcome of a rescue as it does in submersion accidents. The length of time of submersion may be unknown or unreliable for a variety of reasons, including an unwitnessed event or an unrecognized call for help. Victims may be unable to call for help; thus, the waving of hands or thrashing about may be confused with play activity. Witnesses may exhibit panic and frantic behaviors, further impairing effective rescue. Time is of the essence. Warm-water submersions of 4 to 5 minutes often result in irreversible neurologic deficits,[47] whereas cold-water submersion up to 40 minutes with subsequent protective hypothermia, especially in children, may result in complete or near complete recovery.[177,187,199] Factors that predict outcome are based on simple clinical presentations with focus on the neurologic status and are discussed later. The initial evaluation helps the clinician determine the aggressiveness and duration of the resuscitative efforts.

Clinical Presentations

Case History

The author (RBS) took care of this patient in the intensive care unit (ICU) when he was a senior resident. The case demonstrates many of the salient features of submersion victims.

The patient was a 52-year-old, previously healthy male who worked the night shift on a barge in the brackish waters of the Duwamish slough near Seattle and Puget Sound. One night about 2300 hours, he tripped on an uncoiled rope on the deck and fell 20 feet into the water. In spite of struggling to swim, he lost his orientation and drifted under the barge. Another worker saw the patient fall and saw him drift under the boat, and he astutely threw his flashlight into the water away from the side of the boat. The patient saw the light and desperately swam to it, at which time he was rescued by his co-workers. His full time of submersion was probably 2 minutes.

Emergency personnel had been called, and the victim was transported to Harborview Medical Center. In the emergency department, he was anxious but alert and oriented, and mildly short of breath with a cough. His vital signs were respiratory rate 24 breaths per minute, pulse 115 beats per minute, blood pressure 145/100 mm Hg, and temperature 96.5° F (36° C). The rest of his exam was normal except for diffuse rhonchi throughout both lung fields. Arterial blood gas was pH 7.48, $Paco_2$ 32 mm Hg, and Pao_2 58 mm Hg on a facemask with high flow oxygen. Chest radiograph showed a normal cardiac silhouette with diffuse bilateral patchy opacities. He was admitted to the ICU for observation.

Within the first hour, gas exchange worsened in spite of adequate alveolar ventilation. He was intubated, mildly sedated, and placed on mechanical ventilation with volume ventilation mode. In spite of high concentrations of oxygen, gas exchange deteriorated, so he was given a positive end-expiratory pressure (PEEP) trial. At 15 cm H_2O pressure, his blood pressure fell to 76/52 mm Hg. Volume resuscitation was increased. A pulmonary artery catheter had been placed to monitor cardiac function. During the initial PEEP trial, pulmonary capillary wedge pressures were low (<10 mm Hg), and cardiac index had fallen from 2.8 to 1.8 L/min/m^2 of body surface area when the PEEP was increased from 10 to 15 cm H_2O. PEEP was reduced to 10 cm H_2O, and more vigorous volume resuscitation was instituted, after which cardiac index and blood pressure improved such that his PEEP could safely be increased, which was necessary to maintain adequate oxygenation.

Over the next 6 days, lung compliance decreased and then increased; his chest radiograph worsened and then slowly improved; and gas exchange improved such that he was extubated on day 10 after admission. His recovery took another 2 months before he was able to be physically active, and he had dyspnea on exertion for approximately 6 months after his accident. Upon discharge, pulmonary function tests showed a moderate decrease in both vital capacity (VC) and total lung capacity (TLC), mild obstruction to airflow, and severe decrease in diffusion capacity for carbon monoxide. Over the next 6 months, VC and TLC returned to normal, but diffusion capacity remained mildly decreased.

This case demonstrates a number of classic features for submersion injury with aspiration and respiratory failure. First, the victim was extremely lucky to be witnessed and rescued. He arrived at the hospital conscious and alert, one of the best prognostic signs, and had no comorbid conditions. The history and clinical course suggest that he aspirated brackish water, which led to his lung injury and subsequent course in the ICU. The hemodynamic course suggested that he was intravascularly volume depleted and needed volume resuscitation, also a common occurrence in submersion victims. The course of his lung disease was consistent with acute lung injury from aspiration, and the worsening and improvement of his lung mechanics and gas exchange are common in ARDS. Fortunately, he had no other organ failure, and his recovery was unremarkable. The post-hospital course was also consistent with healing ARDS with improving lung mechanics, but with subtle, persistent parenchymal injury, as suggested by low diffusion capacity.

There is a spectrum of presentations for submersion events. Drowning victims may present without symptoms or may be unresponsive, apneic, and pulseless. Systems of classification based on observations from first-aid providers[184] exist to aid in the determination of which victims require hospitalization. These guidelines are vague and are discussed in the next few sections.

As mentioned before, associated injuries should be assessed once the victim is out of the water. The presence of traumatic

injuries, particularly head and neck trauma, should be determined. Findings or circumstances consistent with child abuse, seizures, cardiac arrhythmias, or other medical issues should be ascertained.

On Scene

The scenes at submersion incidents are often chaotic. Victims are not usually found flailing but more commonly found floating on the surface of the water or motionless under the water. Rescue attempts must always take the safety of rescuers into consideration so that multiple drownings do not occur. Safety devices should be used to tow the victim, or life preservers should be tossed to people in trouble before a human lifesaver actually enters the water. Well-intentioned but ill-advised heroic efforts can create additional victims and compound the tragedy.

At the scene, the estimation of time and circumstances are often inaccurate, but attempts at accurate timing and documentation of the course of events, including vital signs, clinical state, and environmental conditions, should begin upon arrival at the scene.[23] The documented description of the scene, estimation of time of submersion and temperature of the water and air, and events at the scene and during transport to the hospital are critical evidence for the physician to make decisions about further resuscitation. If several people are around, the accident scene is usually disorganized, and it is mandatory that someone take charge. It is critical that rescuers coordinate efforts while assessing the site so that no one is put in inordinate danger. Rescue of a submerged victim is often quite difficult and requires training in the technique. Once the victim is out of the water, basic life support should be instituted and continued until emergency physicians at the hospital can determine the next steps. At this time in the rescue, evaluation for concomitant trauma is important. If there is a history of trauma to the body, spinal injuries and injuries to the organs of the chest and abdomen must be considered.

Hypoxemia is the most significant consequence of submersion. Immediate attention to ensure an airway and oxygenation is of paramount importance in the victim's outcome. Assessment by bystanders should determine the presence of breathing, and appropriate resuscitative measures should be instituted immediately and continued by rescuers at the scene. Transport to an emergency facility with ongoing CPR should take place unless resuscitation is determined futile or successful.[14] Concomitant hypothermia makes some of these clinical determinations difficult at best.

The victims of submersion incidents may be classified into four groups: (1) the asymptomatic patient, (2) the symptomatic patient, (3) the patient in cardiopulmonary arrest, and (4) the obviously dead or still-submerged patient.

The Asymptomatic Patient (Box 68-2)

Victims with no other comorbid conditions who have been rescued from the water and who are alert, have no cough or respiratory distress, and with chest exam clear to auscultation may not need further medical care, but still present a dilemma. It is in these situations that documentation by the rescuers about the factors mentioned above is critical. Victims often flee the scene, only to suffer complications later. In a survey of 52 swimmers who had submersion incidents on California beaches, 31 declined medical attention and left the beach. Telephone follow-up of 26 indicated that none had sought medical care subsequently without adverse sequelae. Of the remaining 21 who were evaluated in the hospital, respiratory distress manifested within 4 hours.[159] Any victim with shortness of breath may have mild hypoxemia. Oxygen by facemask should be administered. Hypothermia is often difficult to ascertain at the scene, so it is prudent to have the victim evaluated, if only briefly, at a medical facility.[14] Even in conscious and cooperative victims, protection against hypothermia with passive warming techniques, protection from wind, application of dry clothes and blankets, and administration of supplemental oxygen, if available, should be instituted. If subjects remain asymptomatic with normal vital signs and stable arterial oxygen saturation on ambient air for 4 hours, then it is highly likely that the victim will not require further medical care.

Box 68-2. On-Scene Management of the Asymptomatic Patient*

HISTORY
Time submerged
Description of incident
Complaints
Past health

EXAMINATION
Appearance
Vital signs
Chest examination
Electrocardiographic monitor

INTERVENTION
Oxygen by face mask, 8–10 L/min
Intravenous line at "keep-open" rate
Reexamine patient as needed
Transport patient

*Patients refusing treatment should have their telephone numbers and addresses taken and arrangement for follow-up made.

The Symptomatic Patient (Box 68-3)

All victims of submersion requiring any intervention or resuscitation or showing signs of distress (anxiety, tachypnea, dyspnea, syncope, persistent cough, or change of vital signs) should be transported to a hospital and evaluated. Initial management should focus on simultaneously preserving the safety of the victim and rescue team as constant assessment of the pulmonary, cardiovascular (CPR), and central nervous systems (Glasgow Coma Scale [GCS]) proceed. These tasks are even more difficult in the wilderness setting, and success depends on the severity of the accident, comorbid conditions, the judgment of the rescuers, and the distance from a medical facility, if one is available at all.

The two primary problems after submersion are hypoxemia and acidosis. Protection of the airway to insure oxygenation and ventilation is the first priority. Maintaining perfusion to reverse the metabolic component of the acidosis is a close second. Many victims will be vomiting. The airway must be protected such that aspiration of gastric contents, which will greatly worsen the lung injury, is prevented.

Approximately half of drowning resuscitations include bystander CPR.[23,163] Initiation of immediate ventilatory support

Box 68-3. Field Approach to the Symptomatic Patient*

HISTORY
Description of incident
Time submerged, water temperature, water contamination, vomiting, type of rescue
Symptoms

EXAMINATION
General appearance
Level of consciousness
Vital signs, electrocardiographic monitor

INTERVENTION
Oxygen via mask (8–10 L/min) by demand valve
Intravenous line at "keep-open" rate; consider intubation if patient is tachypneic or dyspneic with cyanosis or depressed mental status
Transport patient

*Be prepared for vomiting. Take measures to prevent hypothermia and shivering. Treat cardiac arrhythmias as they arise, keeping in mind that hypoxia may be the underlying cause.

Box 68-4. Field Intervention in Victims in Cardiopulmonary Arrest*

BEGIN VENTILATION AND COMPRESSION
- Use mouth-to-mouth, mouth-to-mask (preferably with oxygen supplementation), or bag-valve-mask (preferably with balloon mask or two-rescuer) ventilation
- Use supplemental oxygen

ENDOTRACHEAL INTUBATION
- Positive end-expiratory pressure at 5 to 10 cm may be useful
- Intravenous line at "keep-open" rate

IF COPIOUS DRAINAGE FROM LUNGS OR STOMACH
- Suction through endotracheal (ET) tube
- Pass nasogastric tube
- Apply abdominal thrusts between ventilations if ET tube is in place and copious drainage persists
- Sodium bicarbonate ordered on advice of physician or according to protocol of the system. Treat dysrhythmias in standard fashion. If cervical injury is suspected, use a collar and sandbags.

and early CPR, if indicated, result in a better prognosis with better outcomes.[15,23,47,57,66,80,147,154,179] Initiation of chest compressions while the victim is still in the water has been recommended by some, but this maneuver is often ineffective in the water, delays extrication of the victim from the water, and may further endanger the victim and rescuer. Effective CPR realistically cannot be started until the victim is out of the water and on a solid surface. The actions listed in Box 68-4 can then be taken.

Figure 68-6. Mouth-to-mouth ventilation in the water is difficult in the best of circumstances. (Courtesy Alan Steinman, MD.)

On the other hand, rescue breathing should be initiated as soon as the victim's airway can be opened, even if in the water, although the success of this intervention often depends on a well-trained and skilled rescuer. Even in the best of circumstances, these techniques are very difficult to effect; however, either mouth-to-mouth or mouth-to-nose ventilation can be used (Fig. 68-6).[14] Mouth-to-mouth ventilation should be performed with the head in a neutral position (jaw thrust). A higher concentration of oxygen can be delivered (from 21% to approximately 40%) if nasal prongs at 6 to 8 L/min flow are applied to the rescuer. A bag-valve mask, laryngeal mask airway, or Combitube provides a safe mode of ventilating patients. If the patient is spontaneously breathing, a facemask with a reservoir bag of about 2.5 L can be used.

Draining water from the lungs dates back to the 17th and 18th centuries. The Dutch method consisted of rolling the victim over a barrel; another method was flinging the victim over a horse, which was then made to trot. In 1975, the abdominal compression maneuver was introduced for victims of choking, which was suggested for submersion victims as well. Available data on draining techniques did not specify whether these techniques were intended to promote lung drainage or gastric drainage. In victims who have swallowed large amounts of water, gastric distention can interfere with ventilation by increasing intra-abdominal pressures. In such instances, gastric decompression by nasogastric tube is recommended.[8,161,171] In spite of centuries of controversy about "draining the lungs" of the submersion victim utilizing many different techniques, none of these is very effective. The resultant pulmonary edema is cleared over time by alveolar fluid clearance that is a result of an active Na^+-K^+ ATPase pump that operates at the level of the alveolar epithelial lining cells. This process is impaired by alveolar hypoxia.

Digital or visual examination for foreign bodies should be done and if present, removed with a swipe or grasp of the fingers. Foreign bodies, such as sand, beach flotsam, and seaweed, have been reported in up to 54% of cases in one report of surf-related submersion.[112] If foreign bodies are suspected, chest compressions should be performed, because they have been shown to be superior to the Heimlich maneuver for the expulsion of foreign material.[99] The Heimlich maneuver has no role in the resuscitation of submersion victims, in spite of the

fact that some papers suggest that the Heimlich maneuver should be performed to remove water from the airways.[121] These papers do not support this weak physiologic hypothesis. Further evidence does not support routine performance of the maneuver in submersion victims[15,146,163,171] because it delays ventilation and places the patient at risk for pulmonary aspiration of gastric contents that could be induced by the maneuver itself. Suctioning is the primary action to optimize the clearance of secretions, vomitus, or foreign debris.

Vomiting occurs during resuscitation and has been reported in 86% of victims receiving CPR.[112] Should vomiting occur, roll the victim onto his or her side or turn the victim's head to the side and sweep the vomitus with a cloth or finger-sweep maneuver.[15] If spinal injury is of concern, the victim should be log-rolled, maintaining linear integrity of the head, neck, and torso.[14] The risk of aspiration from vomiting may be reduced with the application of cricoid pressure during ventilation.[145] A nasogastric tube, if available, should be inserted to remove gastric contents, including water swallowed during submersion.

Supplemental oxygen, if available from emergency personnel at the scene, should be instituted as soon as possible. If an oxygen-delivery device such as a nonrebreathing mask or continual positive airway pressure (CPAP) mask is available, oxygen should be delivered at high-flow rates (10 to 15 L/min). CPAP of approximately 10 cm H_2O should be provided to a breathing patient, if a CPAP mask and compressed air are available.[18,55,69,163] Higher levels may result in deleterious hemodynamic effects, which should be monitored in the hospital setting.

Early intubation to avoid the risk of aspiration of gastric contents is indicated in patients whose mental status is impaired such that the airway cannot reliably be protected.[79] Further ventilator support should be continued with frequent suctioning. CPAP or even PEEP can be applied more efficaciously and reliably in the intubated patient, but here again caution must be taken in the field setting where personnel may not be familiar with ventilatory support and compressed air may not be available. Regardless of the degree of CPAP or PEEP, it is most important to ventilate the patient in volume ventilation mode, because one must be assured that a certain minute ventilation (approximately 10 L/min in an adult) is delivered, which may be difficult with pressure ventilation in lungs that may already have reduced compliance.

Once the victim is out of the water, presence or absence of adequate circulation should be ascertained. In cases of hypothermia or hypotension, the pulse may be difficult to identify. If ventilation or cardiac function is impaired, chest compressions should be initiated as soon as the victim is removed from the water. For victims older than 8 years, an automated external defibrillator (AED) may be used to evaluate rhythm,[14] although some state that in this setting, there is no major role for the AED.[23,163] In an otherwise healthy victim, ventricular fibrillation (VF) is rare after submersion,[23] but it may triggered by unnecessary compressions, especially if the patient is moderately hypothermic. In the presence of VF and profound hypothermia (30°C/86°F) defibrillation attempts should be limited to three,[15] because defibrillation is ineffective if the myocardium is colder than 28°C (82°F).[79] If the rescue team is capable of advanced life support, then endotracheal intubation, cardiac monitoring, and intravenous (IV) or intraosseous (IO) fluids and medications should be used appropriately. The continuation of BLS or ACLS should continue until the core body temperature is above 30°C or 86°F.[14] Special thermometers that read such low temperatures should be available as part of advanced rescue equipment.

See Table 68-1 for a classification scheme for near-drowning and drowning experiences.

Hypothermia

Drowning may result directly from hypothermia.[45,87] Experiences with persons shipwrecked, even in relatively warm water, have shown that body temperature of victims can be lowered to the point at which hypothermia threatens survival. Thermal conductivity of cold water is 25 to 30 times that of air.[87,104,105] Water at 91.4°F (33°C) is thermally neutral, the point at which heat loss equals heat production for a swimmer without clothes. Water colder than this leads to ongoing heat loss. Thus, in very cold water, hypothermia may ensue rapidly and lead to drowning. This is particularly true in children who have less subcutaneous fat and relatively greater body surface area than do adults.[104] Cooling may occur by a combination of conductive heat loss directly into the water and circulatory cooling from chilled venous blood returning to the circulation.

See Chapter 6 for more details.

Direct Hypothermia. At or below a core temperature of 82°F (28°C), ventricular fibrillation can occur.[83,131,180] At a water temperature of 32°F (0°C), the victim of submersion cannot survive for more than an hour; at a water temperature of 59°F (15°C), survival is not common after 6 hours.[44,67] However, several Canadian cases defied this dogma. In one incident, a pilot and passenger ditched their plane into a lake on final approach to an island airport.[114] Air temperature was 36°F (2°C), and water temperature was 39.2°F (4°C). Both men were similarly clothed and in the water for 60 minutes before rescue. The passenger was a 48-year-old man, 101 kg in weight and 190 cm in height, with a core temperature of 89.6°F (32°C) upon admission to the hospital. He was alert, complained only of being cold, was actively rewarmed, and left the hospital the next day. The pilot, a previously healthy 38-year-old man, was of medium body habitus with little subcutaneous fat. He was admitted to the same hospital in ventricular fibrillation with a core temperature of 84.2°F (29°C). Despite core rewarming and vigorous resuscitation efforts over 4 hours, he could not be revived. In view of the identical environmental conditions, clothing, and time in the water, the authors concluded than skinfold thickness was the most important discriminating factor in the outcome.[120]

A second case illustrates that preparation and protective clothing may lengthen survival time in cold water.[113] A married couple, both experienced sailors, began to take on water in their boat about 11 km from shore in a Canadian lake.[190] The boat sank after 40 minutes, during which time they were able to prepare for abandoning ship by lashing together life preservers and donning heavy parkas and special survival gear carried in the boat. The month was August, but a storm had developed with gale-force winds of 60 km/hr and high waves. Water temperature was estimated to be 64°F (18°C). Both persons spent more than 18 hours in the water before rescue and helicopter transport to the regional trauma center. Both were moderately hypothermic on admission; the man's temperature was 86.9°F (31°C), and the woman's was 97.5°F (36°C). Both were discharged the next day without complications, with survival attributed to preparedness and protective clothing.

In victims kept afloat by life preservers or other means, death may result from hypothermia. Although some people may

appear dead after rescue, efforts to resuscitate and rewarm them may result in recovery. Still, the absolute effect of hypothermia on survival remains in doubt. In certain cases, hypothermia appears to be protective, as in the case of a Norwegian child who survived neurologically intact after being rescued from an ice-laden stream in which he had been submerged for 40 minutes.[177] His temperature on admission was 75°F (24°C), and after 1 hour of resuscitation, spontaneous circulation returned. He was discharged from the hospital 8 days later. There are a number of anecdotal cases like this one, but there are just as many cases in young children in whom severe hypothermia had poor outcomes.[199] The "diving reflex," which is operant in diving mammals, may be a vestigial but inducible reflex in humans that contributes to survival.[46,71,113] The reflex is marked by efficiency of heat and energy distribution and utilization. Heart rate slows, and blood flow is regionally diverted in part to the brain and muscles of locomotion while sparing other vascular beds. The reflex may be inducible in only 15% to 30% of humans and may be a contributing factor in those who survive.[72,81]

The other explanation for survival after prolonged submersion in cold water is that rapidly induced hypothermia reduces tissue oxygen demand and protects the brain. Catecholamine release may be blunted in victims who become rapidly hypothermic in very cold water, lessening the propensity to ventricular fibrillation.[143] Muscle movement is further impaired, which reduces oxygen demand.

In contradistinction to the protective effect of hypothermia in submerged victims, hypothermia can create serious problems for the victim struggling to survive in cold water. Specific patterns of heat loss can be demonstrated in subjects placed in water at temperatures as low as 40.1°F (5°C). In these studies, thermograms have graphically recorded heat loss from various parts of the body, demonstrating preferential loss from the head and neck (up to 50%), thoracic cage, and inguinal area. Movement further enhances heat loss.

An anecdotal situation from an eco-tour boat in Antarctica describes a Zodiac boat that capsized in the surf during a beach landing. Three people were thrown overboard, and each adapted a strategy for survival. One tried to swim toward the ship; another treaded water; the other curled in a ball and floated until rescuers arrived. All three men were rescued, but each had a different clinical outcome. The one who tried to swim was hypothermic such that he had to be rewarmed for 8 hours. The one who treaded water was taken to the infirmary and required 4 hours of observation and rewarming before he was allowed to retire to his cabin. The one who had curled up, a position which presumably conserves body heat, was rescued to the ship, changed his clothes, and retired to the bar for a beer.

This anecdote has been confirmed by data in which subjects were exposed to water of 50°F (10°C). Survival time was doubled in subjects who assumed the heat escape–lessening posture (HELP), which was similar to the individual in Antarctica whose position, that of a curled-up diver, lessened the exposure of body surface area to water, where heat may be lost. Depending on how much buoyancy the subject gets from his life jacket, the subject may need to turn his head intermittently to breathe, which is not an easy task without inhaling water, particularly if the water is rough. Data that show survival time based on body position are shown in Fig. 68-7. The HELP posture is shown in Figure 68-8. If two or more persons are in the water, the huddle position (Fig. 68-9) is recommended to lessen total body heat loss. Because children become hypothermic much more quickly than adults, they should be placed in the middle of the huddle. In this position, life jackets are tied behind the back to press together the groin and lower body regions.[44]

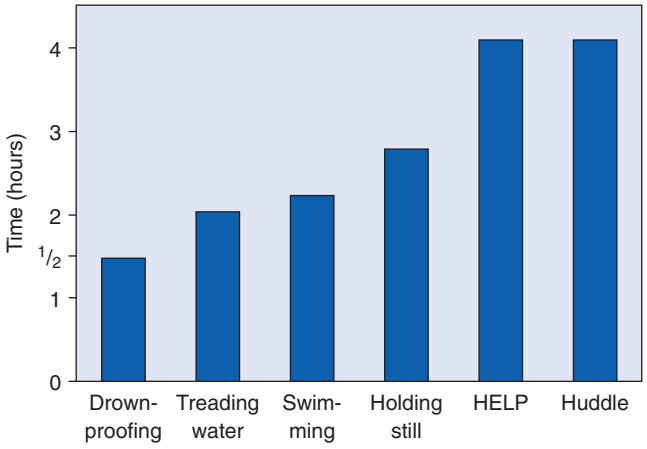

Figure 68-7. Survival times in cool (10°C [50°F]) water using various techniques in several situations. HELP, heat escape lessening posture. (Data from Collis ML: Survival behaviour in cold water immersion. In Proceedings of the Cold Water Symposium, Toronto, Royal Life-Saving Society of Canada, 1976.)

Figure 68-8. Heat escape lessening posture (HELP). (Courtesy Alan Steinman, MD.)

Hypotension. Hypotension may be a feature of submersion. Victims may be hypovolemic and require fluid administration as a result of venous pooling.[79] Surrounding water to the depth of 6 feet (the height of an adult body) exerts pressure on the extremities and trunk, which increases central blood volume and produces 32% to 66% increase in cardiac output when a victim is in an upright immersion position. Victims removed from the water suddenly lose this support. Venous pooling combined with impaired systemic resistance can result in circulatory collapse. Keeping the victim prone and horizontal as he is lifted from the water may minimize a hypotensive event.[79]

Figure 68-9. Huddle technique. (Courtesy Alan Steinman, MD.)

Once out of the water, chest compressions may be initiated if pulselessness is present. The presence of hypothermia may make the determination of pulselessness challenging, as the rate may be slow and the quality of the pulse may be weak. Many hypothermic patients also tend to be hypovolemic, which accentuates this problem.

Cervical Spine Injury. Cervical spine injury is rare in drowning except in high-impact accidents, such as high-speed boating accidents, surfing injuries, and diving mishaps.[89,192] Evaluation of the breathing pattern and breath sounds is critical. For instance, if there is an acute mid-cervical spine injury, subjects have an intact phrenic nerve and diaphragm, but flaccid intercostal muscles. During inspiration, the examiner would see diaphragmatic contraction with outward motion of the abdomen while the muscles of the thoracic cage remained flaccid, resulting in retractions of the rib cage during inspiration. Unless this physical finding is present, routine spine immobilization is unnecessary and may deter life-saving resuscitation measures.[23,89,192] On the other hand, in cases where neck injury may present as a precipitating factor in drowning or in high impact submersion, attention to head and neck trauma with appropriate immobilization is essential.[192] Hands are used to stabilize the victim's head and neck in a neutral position (without flexion or extension) as the victim is floated onto a horizontal board. Although rescue breathing may be initiated, chest compressions usually have to wait until the victim is on shore. Evaluation for additional fractures or internal injuries should commence.[15,57,79]

En Route

The outcome of the victim's course depends in large part on the preparedness and skill of the team accompanying the victim to the hospital. During transport, the basic principles of ventilation and circulation must be followed. If the victim is breathing spontaneously, supplemental oxygen should be delivered by facemask or nasal prongs. If the victim is not breathing spontaneously or cannot protect his airway, endotracheal intubation should be performed and the patient ventilated with an Ambu-Bag or portable ventilator with volume mode ventilation. A pulse oximeter should be used to estimate and monitor arterial oxygen saturation.

Ongoing hemodynamic assessment with blood pressure measurements should be undertaken while fluid administration with dextrose in normal saline is used to optimize hemodynamic stability. Body heat loss should be minimized by ensuring that the patient is dry, out of the wind, and covered with blankets; however, hyperthermia from overzealous warming should be avoided.[18,57] Shivering, although a good prognostic sign, increases tissue oxygen demands and caloric expenditure. Thus, nutritional intake with warm glucose drinks is important in an awake and communicative patient.[79,121] If active rewarming techniques for patients with deep hypothermia utilizing cardiopulmonary bypass or extracorporeal membrane oxygenation (ECMO) are anticipated, choosing a medical facility with these options is recommended when possible. Other active rewarming techniques, such as continuous arteriovenous rewarming, continuous venovenous hemofiltration, and body cavity lavage with warmed normal saline (peritoneal, pleural, gastric, bladder), are sometimes available in medical centers. Assessment of neurologic status with attention to changes in GCS should be meticulously recorded. It is crucial for all field data to be communicated to the receiving facility so that the medical team can anticipate the level of care and team that will be necessary.

The use of drugs in the field setting should be avoided. Body temperature and acid-base status of the victim may not be known in the field setting, making metabolism and the effects of drugs unpredictable. Thus, there may be more risk than benefit in giving drugs or infusions, such as sodium bicarbonate, which may have unintended deleterious effects. Drugs should therefore be reserved for use whenever possible by the hospital team, which is guided by precise monitoring.

Some victims may appear dead when retrieved from the water. They are unresponsive with a normal temperature, demonstrate postmortem lividity, and may be asystolic and apneic. If rescue operations have been in progress for more than 30 minutes with no return of vital signs, victims who are retrieved from warm water may be considered dead on the scene. However, because cold water submersion for more than 40 minutes has been associated with neurologic survival,[177] rescue crews must start and continue life support measures in all persons who are not obviously dead.

When persons are obviously dead or still submerged, the rescue team must comfort the family and friends and participate in body recovery. Once retrieved, the victim should be covered immediately and treated with proper dignity. Family members and friends should be escorted from the scene, if possible, before recovery. Most drownings are medical examiner cases, because the possibility of foul play must be investigated.

At the Medical Facility

The emergency department ideally should be notified ahead of time from the field and be prepared to greet the patient with the appropriate team. Meticulous records of the rescue and clinical course en route are essential data to prepare the receiving team for optimal hospital care.

Classification of Patients

Clinical evaluation of patients on arrival at the hospital provides useful information to the team about anticipated level of care and prognosis. Modell and Conn[123] suggested an ABC classification of patients based on the presenting neurological examination. This system has undergone revision (Table 68-3), with

TABLE 68-3. Classification of Near Drowning Victims*

CATEGORY	DESCRIPTION
A	Awake—fully oriented
B	Blunted—arousable; purposeful response to pain
C	Comatose—not arousable; abnormal response to pain
C_1	Flexor response to pain
C_2	Extensor response to pain
C_3	Flaccid
C_4	Arrested

*Assessment is made in emergency department within 1 hour of rescue.
Modified from Conn AW, Barker GA: Fresh water drowning and near-drowning—an update. Can Anaesth Soc J 31:S38–S44, 1984.

the time of initial examination currently being in the ED after initial resuscitation, usually within 1 hour after rescue. More aggressive resuscitation led to the introduction of a fourth subgroup under C. Under this system, patients were A, *a*lert; B, *b*lunted in consciousness; or C, *c*omatose. Comatose patients were further categorized according to abnormal neurologic responses: C_1, flexor response; C_2, extensor response; or C_3, flaccid. A C_4 category was added to include patients in whom vigorous CPR was being applied that might result in a change in condition and thus prognosis.[47]

In their series on children, Conn and coworkers[48,49] reported that all patients in category A survived and were normal, and all but one patient survived in category B. The C group showed various responses, with increasingly poor outcomes. Similar studies in adults have not been done.

Graf and colleagues[74] studied 194 submersion victims and found that four variables could predict unfavorable outcome: comatose state, lack of pupillary light reflex, male gender, and initial blood glucose greater than 200 mg/dL on presentation. Some studies suggest the use of the pediatric risk of mortality (PRISM) score to predict outcome. Victims with PRISM scores greater than 20 died or survived with severe neurologic deficits[60,77] (Table 68-4).

Asymptomatic Patients

These patients may be deceiving, in that depending on the history of submersion, the clinical manifestations of pulmonary dysfunction may be delayed for 12 to 72 hours. Thus, it is imperative that a careful history is taken to assess the possibility of aspiration. Most acute care clinicians, faced with a reliable history of submersion, elect to perform a physical examination, obtain arterial blood gas (ABG) measurements to look for a widened alveolar-arterial difference, and acquire a chest x-ray on even asymptomatic or minimally symptomatic victims of near drowning.[94,120,143,150] Persons who show abnormalities or who deteriorate within 4 hours of admission to the ED should be admitted for observation and further management. In-hospital management is outlined in Box 68-5.

Symptomatic Patients

Patients complain of symptoms of coughing, sore throat, burning in the chest, and dyspnea (Box 68-6). Hypoxemia may accompany dyspnea, and if symptoms are severe and hypoxemia profound, acidemia may be present. Intervention must be aggressive. Initial serum chemistries, ABG, and a chest radiograph should be performed.

TABLE 68-4. Pediatric Risk of Mortality (PRISM) Score

	AGE RESTRICTIONS AND RANGES		
VARIABLE	INFANTS	CHILDREN	SCORE
Systolic BP (mm Hg)	130–160	150–200	2
	55–65	65–75	
	>160	>200	6
	40–54	50–64	
	<40	<50	7
Diastolic BP (mm Hg)	>110	>110	6
HR (beats/min)	>160	>150	4
	<90	<80	
Respiratory rate (breaths/min)	61–90	51–70	1
	>90	>70	5
$Pa_{O_2}/F_{I_{O_2}}$*		200–300	2
		<200	3
Pa_{CO_2}† (mm Hg)		51–65	1
		>65	5
Glasgow Coma Scale‡		<8	6
Pupillary reactions		Unequal or dilated	4
		Fixed and dilated	10
PT/PTT		1.5 × control	2
Total bilirubin (mg/dL)		>3.5 (>1 mo)	6
Potassium (mEq/L)		3–3.5	1
		6.5–7.5	
		<3	5
		>7.5	
Calcium (mg/dL)		7–8	2
		12–15	
		<7	6
		>15	
Glucose (mg/dL)		40–60	4
		250–400	
		<40	8
		>400	
Bicarbonate§ (mEq/L)		<16	3
		>32	

BP, blood pressure; HR, heart rate; Pa_{O_2}/Pa_{CO_2}, arterial oxygen/carbon dioxide pressure; $F_{I_{O_2}}$, fraction of inspired air; PT/PTT, prothrombin/partial thromboplastin time.
*Cannot be assessed in patients with intracardiac shunts or chronic respiratory insufficiency; requires arterial blood sampling.
†May be assessed with capillary blood gases.
‡Assessed only if there is known or suspected CNS dysfunction; cannot be assessed in patients during iatrogenic sedation, paralysis, anesthesia, etc. Scores <8 correspond to coma or deep stupor.
§Use measured values.
Modified from Pollack MM: Quality of care: national or international standards? Crit Care Med 26:1313–1314, 1998.

Comatose Patients or Patients in Cardiac Arrest

Guidelines on predicting outcomes offer a uniform approach to management, allow for realistic expectations based on measurable data, and propose common terminology (Box 68-7).[47,143] Despite advances, patients who require resuscitation may face serious CNS deficits and a poor prognosis. In some pediatric studies, 21% of children who survived resuscitation had severe encephalopathy.[156] Others report that the outlook, particularly in children, is good, with morbidity ranging from 3.5 to 5%.[48,126,151,193]

Box 68-5. In-Hospital Management of Asymptomatic Victims of Submersion Incidents

CHECK AIRWAY
- Supplemental oxygen: nonrebreathing mask at 12 to 15 L/min or via demand valve pending oximetry or arterial blood gas determinations
- Obtain history and estimate severity of incident; look for possible underlying causes, e.g., cerebrovascular accident, epilepsy, drugs, myocardial infarction, arrhythmia

TAKE VITAL SIGNS AND EXAMINE PATIENT
- Draw and hold blood samples for complete blood count, electrolytes, blood urea nitrogen, platelets, prothrombin time, partial thromboplastin time; send if indicated
- Arterial blood gases; consider pulse oximetry; consider blood alcohol level and toxicology screen
- Urinalysis
- Chest radiograph

DISPOSITION
- Observe all patients for 4 to 6 hours; discharge home if completely asymptomatic and adequate telephone or medical follow-up can be guaranteed.

Box 68-6. In-Hospital Approach to Symptomatic Victims of Submersion Incidents

CHECK AIRWAY
- Supplemental oxygen: nonrebreathing mask at 12 to 15 L/min or via demand valve

CONSIDER ENDOTRACHEAL INTUBATION
- For comatose patients
- For patients unable to maintain a PaO_2 above 90 mm Hg on high-flow oxygen (nonrebreathing mask at 12 to 15 L/min), or unable to maintain a $PaCO_2$ below 45 mm Hg

CHECK VITAL SIGNS
- Start intravenous line: draw blood for CBC, electrolytes, BUN, platelets, prothrombin time, partial thromboplastin time
- Arterial blood gas studies
- Urinalysis
- Administer bicarbonate according to blood gas results if acidosis is severe and cannot otherwise be corrected
- Chest radiograph
- Cervical spine radiograph if any doubt about circumstances of incident
- Nasogastric tube
- Indwelling urinary catheter if necessary

DISPOSITION
- Admit all patients with abnormal vital signs, abnormal radiologic signs, or abnormal findings on blood gas measurements
- Completely asymptomatic patients with no findings may be discharged after 4 to 6 hours observation if adequate telephone or other medical follow-up can be guaranteed

In the absence of profound hypothermia, the neurologic status of a victim on admission to the ED is crucial in determining the person's course. Persons who are alert when admitted should seldom die.[48,52,91,126,142] Prognostic indicators, particularly in pediatric patients, include the Glasgow Coma Scale[52] and the presence of spontaneous breathing.[56,74,91,95] Duration of submersion, because of its unreliability, is not a helpful gauge of outcome of resuscitation attempts. Full recovery of some patients with initial fixed and dilated pupils tempers the tacit acceptance of all factors historically outlined as prognostic signs.

Pulmonary Status

Management of the patient's respiratory status from the beginning of the clinical course to the end is the single most important priority and may make the difference between life and death. In that light, respiration is defined as both pulmonary and tissue respiration—that is, obtaining oxygen from the air and transporting it successfully by perfusion to the tissues where it is utilized.

Oxygen (100%) by facemask should be administered to all drowning victims who are breathing spontaneously until arterial blood gas analysis more precisely directs the course of oxygen therapy.

The patient may already have severe pulmonary edema with diffuse infiltrates on chest radiograph, stiff lungs, and poor oxygenation and thus fit the definition for ARDS. A low tidal-volume strategy (tidal volume of 6 mL/kg) should be initiated, and PEEP started at low levels (5 to 10 cm H_2O) only if a Pa_{O_2} of >60 mm Hg cannot be maintained on an FIO_2 of ≤0.6. PEEP should be used carefully, because the higher intrathoracic pressure may impair venous return and further impair cardiac output and oxygen delivery. On the other hand, because the pulmonary edema from aspiration may not initially cause a marked decrease in lung compliance or depletion in surfactant, PEEP can be remarkably effective by expanding edematous or collapsed alveoli and thus improve gas exchange and oxygenation.

CPAP in the field remains an area of controversy. Multiple EMS systems and the military have utilized this modality in the setting of acute pulmonary edema and congestive heart failure (CHF) successfully. Following stringent guidelines, which include strict blood pressure parameters and maximum PEEP settings, minimal to no adverse outcomes have been reported to date. Furthermore, the incidence of patients requiring intubation in this setting has decreased.[55] Field providers report intense satisfaction in being able to provide a modality of therapy that appears to improve patient outcome rapidly and may prevent their deterioration. To date, there are no randomized, controlled studies in the application of CPAP in submersion victims and only preliminary case series on field CPAP application. Therefore, extrapolating from the currently available information, it appears that there may be a role for CPAP use in the conscious submersion victim who demonstrates signs of pulmonary edema, in the hands of experienced operators and with clear guidelines.[135]

Upon arrival at the hospital, it is vital to make serial assessments of pulmonary, cardiovascular, and metabolic status,

Box 68-7. In-Hospital Management of Patients in Coma or Cardiac Arrest*

AIRWAY CONTROL
- Endotracheal tube; nasotracheal preferred
- Supplemental oxygen or mechanical ventilation with intermittent mandatory ventilation (IMV), continuous positive airway pressure, or IMV with positive end-expiratory pressure
- Bicarbonate therapy guided by arterial blood gas values
- Treatment of cardiac arrhythmias according to protocol

DIAGNOSTIC
- Arterial blood gases
- Blood for CBC, electrolytes, coagulation baseline
- Urinalysis
- Chest x-ray examination
- Cervical spine films (if indicated)
- Electrocardiographic monitoring
- CT or MRI suggested in patients who remain comatose
- No antibiotics
- No steroids

FURTHER INTENSIVE CARE MEASURES
- Arterial line
- Swan-Ganz catheterization when fluid status in doubt
- Diuretics
- H_2 antagonists
- Paralysis (pancuronium, 0.1 mg/kg IV prn)
- Maintain blood pressure: fluids if needed and vasopressors: dopamine, 1–20 µg/kg/min; dobutamine, 2–15 µg/kg/min

*Routine hypothermia is not recommended. Barbiturates after cardiac arrest are not proven effective. Intracranial pressure monitoring may be considered.

because pulmonary deterioration may be delayed as late as 24 hours after the insult.[66] A nasogastric tube may be placed to avoid gastric distention and avoid vomiting and aspiration.

By the time the victim has reached the hospital, assessment of whether he or she should be in the intensive care unit under the care of a pulmonologist should have begun. ABG measurements will determine (1) alveolar ventilation (i.e., acceptable $Paco_2$ and pH); (2) gas exchange (i.e., Pao_2 >60 mm Hg on supplemental oxygen); and (3) perfusion (i.e., no metabolic acidosis). If these parameters cannot be maintained, and the patient is clinically in respiratory distress, mechanical ventilation should be started with volume-ventilation mode and high levels of supplemental oxygen. It is then important to address the metabolic contribution to the acidemia, which may result from the victim's struggle before rescue or cardiovascular collapse. Other criteria for intubation include any patients who are unable to handle their secretions, obtundation, failure to maintain Pao_2 >90 mm Hg on high flow O_2, rising $Paco_2$ over 45 mm Hg, or failure on CPAP.[84] Intubation using a rapid sequence intubation protocol (RSI) and maintaining compliance with sedatives and paralytics are needed. The selection of RSI medications is important and should be tailored to each victim. Table 68-5 is a brief summary of RSI medication choices and dosages.

TABLE 68-5. Medication Choices and Dosages for Rapid Sequence Intubation (RSI)

PURPOSE	DRUG	DOSE
Pretreatment	Lidocaine	1.5 mg/kg
	Fentanyl	3 µg/kg
	Atropine	0.02 mg/kg
	Defasiculating vecuronium	0.01 mg/kg
Induction	Etomidate	0.3 mg/kg
	Midazolam	0.3 mg/kg
	Ketamine	1–2 mg/kg
	Thiopental	3 mg/kg
	Propafol	1.5 mg/kg
Paralytic	Succinylcholine	1.5 mg/kg
	Rocuronium	1 mg/kg
	Vecuronium	0.15 mg/kg
Sedation	Lorazapam	0.05 mg/kg
	Vecuronium	0.1 mg/kg

It is recommended that intubated patients requiring CPAP and PEEP be maintained with such support for at least 24 hours to allow for regeneration of surfactant. Many patients require 2 to 4 days of CPAP and PEEP to allow for adequate surfactant production.[104,146]

Intravascular volume must be assessed and optimized. If levels of PEEP ≥15 cm H_2O are anticipated to maintain oxygenation, a pulmonary artery catheter or serial echocardiograms may be necessary to ensure that cardiac function is not impaired. Depending on the clinical course, accepted ventilator management and weaning should be followed.

Irritation of the tracheobronchial tree by inhaled water and particles may evoke cough and bronchospasm, which may worsen hypoxia. Aerosolized albuterol, a relatively selective $beta_2$ adrenergic agonist, is rapid acting, has minimal side effects, and is the initial drug of choice in the initial treatment of bronchospasm. The average dose is 1.25 to 2.5 mg diluted in 2 to 5 mL of sterile water or saline. It can be administered to spontaneously breathing patients via facemask or to intubated patients using a T-piece adaptor. Ipratropium (Atrovent) 500 µg via nebulizer may be added to decrease mucus production and spasm. In refractory cases, common in patients already on methylxanthines for preexisting COPD or asthma, aminophylline IV 0.5 mg/kg/hr can be given to raise levels to therapeutic levels.[21,139,166]

Regardless of clinical appearance, a baseline chest radiograph should be obtained.[94] Chest radiographs that show bilateral airspace abnormalities on admission typically resolve in 7 to 10 days.[93] On the other hand, a normal admission chest radiograph may be followed by one that is abnormal in 4 hours.[115] Also, the chest radiograph should be examined to look for pneumothoraces, which are not uncommon in patients who have suffered a number of pulmonary insults during the accident as well as during transport and resuscitation. Chest CT scan offers little advantage over plain films at the initial time of hospitalization. When they are obtained, 50% of the patients in one study had fine intralobular reticular opacities superimposed upon areas of ground glass attenuation.[93] Children may be safely discharged from the ED if the GCS is normal and they have normal respiratory efforts and O_2 saturation on room air of more that 95% after 4 to 6 hours of stable observation.[39]

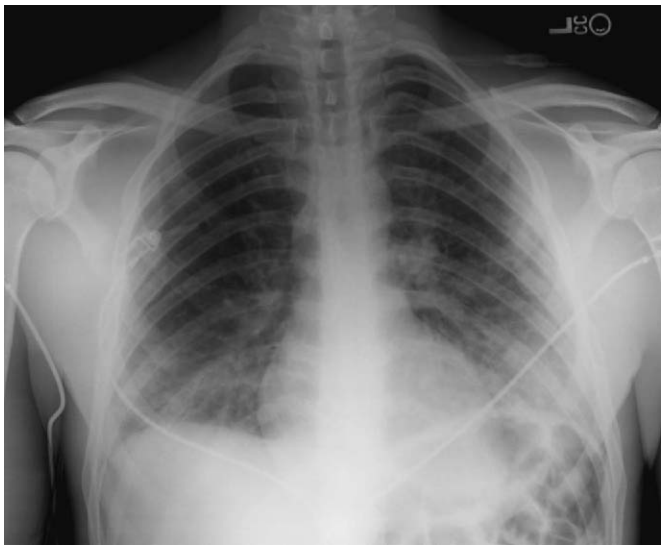

Figure 68-10. Submersion-related pulmonary edema. (Courtesy U.S. Navy.)

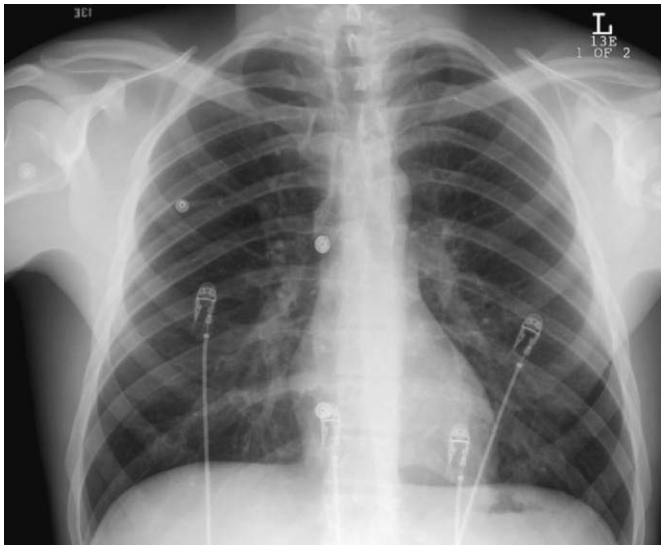

Figure 68-11. Resolution of the edema seen in Figure 68-10. (Courtesy U.S. Navy.)

Figures 68-10 and 68-11 show the progression of a case of submersion-related pulmonary edema.

Hypothermia in the Hospital

In cases of hypothermia, extraordinary cases of survival have been described. Therefore, resuscitation should be continued until body temperature reaches 33° to 35°C (91° to 95°F).[163] Resuscitation can be terminated if, at that temperature, there is still no cardiac or neurologic activity.[23] The management of hypothermia should be addressed immediately and is discussed more thoroughly in Chapters 5 and 6.

Individualization of rewarming techniques is based on the hemodynamic stability of the patient. Conductive heat warmers are recommended for patients with moderate to severe hypothermia who are hemodynamically stable. Patients with unstable hemodynamics need correction of their intravascular volume first, and if still unstable, other more active techniques of rewarming may be considered. Active core rewarming with peritoneal dialysis, hemodialysis, or continuous venovenous hemofiltration has been accomplished without excessive risk. Cardiac bypass has been used in patients who have suffered cardiac arrest.[174] These measures, along with extracorporeal membrane oxygenation (ECMO), are extraordinary and heroic but have been successful in resuscitation of deeply hypothermic drowning victims with severe pulmonary failure.[187]

Guidelines of the American Heart Association and the European Resuscitation Council do not recommend administration of vasopressin to improve hemodynamics in the hypothermic submersion victim.[12,13] Others report its use to be successful in the restoration of circulation in hypothermic drowning victims prior to rewarming.[183] Drowning victims who remain comatose after successful cardiac resuscitation may be treated by inducing mild hypothermia (90° to 93°F; 32° to 34°C) and sustaining that level for 12 to 24 hours. This maneuver may result in better neurologic outcome.[23]

Shivering occurs naturally with moderate hypothermia, as well as with body surface cooling. With active shivering, metabolic rate increases up to five times from rest, which generates heat. In survival situations, shivering serves an important function transiently. In the patient with hypothermia and potential intravascular volume depletion, shivering is accompanied by a cardiac output that is suboptimal for the metabolic rate, thus potentiating the possibility of inadequate perfusion and an accumulating metabolic acidosis. To terminate shivering once the patient is in a controlled medical environment, a low dose of meperidine, 25 to 50 mg, is effective and safe.[23] The use of meperidine in controlling shivering has been studied in the setting of hypothermia induction in the hospital in acute stroke and cardiac arrest, where hypothermia induction has been shown to decrease metabolic demands and cerebral edema.

Cardiovascular System and Hemodynamics

While ventilation and gas exchange are being established, the clinician must also address tissue respiration by ensuring that cardiac output and peripheral perfusion are maintained. Cardiac dysrhythmias and arrest before and during the course of resuscitation are often corrected by improvements in oxygenation and acid-base status, but if either of these conditions exists, treatment of dysrhythmias and cardiopulmonary resuscitation must be instituted.

Before the patient's intravascular volume status can be accurately determined, it should be assumed that this volume is decreased. Intravenous access with two large-bore venous catheters or central lines must be established, and infusion of normal saline at a brisk rate should be started. Re-establishment of systemic pressure is critical to success of the resuscitation, and any theoretical disadvantage of worsening pulmonary edema is far outweighed by hemodynamic and acid-base integrity. If cardiac output and ventilator support are sufficient, pulmonary edema will clear more readily, and gas exchange can be maintained.

After these initial steps are taken, an arterial cannula can be placed to monitor blood pressure and arterial blood gases. The use of a pulmonary artery catheter is advisable in patients who develop hemodynamic instability or, as mentioned in the

pulmonary section, require levels of PEEP approaching or exceeding 15 cm H_2O pressure to maintain oxygenation. When hypotension is present, pulmonary capillary wedge pressure and cardiac output terminations allow a rational approach to fluid administration. The goal is to maintain oxygen delivery that is a product of cardiac output and arterial oxygen content. Measurement of oxygen extraction (the difference between arterial and mixed venous oxygen content) can also help the intensivist judge adequacy of perfusion and stress on the tissues. Some clinicians advocate the use of bicarbonate when the pH remains below 7.25 despite aggressive respiratory management. If ABGs are available to guide management, the following formula can be used for calculating the dose: sodium bicarbonate (in milliequivalents [mEq]) = patient weight (kg) × base deficit × 0.2. If no ABG is available, an approximation of 1 mEq/kg may be used.[120] One half the dose is given initially and the effects on the ABG observed.

Hypotension during the initial resuscitation of submersion patients suggests intravascular volume loss or cardiac failure, rather than septic shock, which may occur later in the clinical course. Volume resuscitation and re-establishment of cardiac function from rhythm disturbances utilizing antiarrhythmic agents, and the issue of asystole managed with CPR, were mentioned previously. Acute cardiac injury must also be considered as a contributing factor and treated accordingly. Serial electrocardiograms and serum cardiac markers of ischemia may be deemed necessary. Pressor agents, such as dopamine (2 to 20 µg/kg/min), dobutamine (2 to 15 µg/kg/min), or norepinephrine (0.5 to 30 µg/kg/min), may be required to maintain a mean arterial pressure adequate for tissue perfusion, but these should be used only as intravascular volume is being restored. Unless there is ongoing fluid loss, such as bleeding, or cardiac failure, the pressors can usually be discontinued fairly quickly.

Central Nervous System

The goal of the medical team is to achieve the best neurologic outcome, which requires optimal management of respiration and hemodynamics. Hypoxemia and hypoperfusion result in cerebral dysoxia, which leads to neuronal damage. Resultant loss of cell membrane integrity results in extracellular fluid leak and cerebral edema. This may produce elevated intracranial pressure (ICP) and possible herniation. Sequencing of the effects of neurologic insult has been attempted; however, studies are few, and certain attempts at therapy have been futile,[69] while others have shown promise.[47,48,49,53]

The best indicator of outcome is serial neurologic examinations, brain imaging, and physiologic testing in the initial 24 to 72 hours of therapy.[90] When there is evidence of neurologic impairment, consultation from a neurologic specialist is essential. Measurements of ICP and cerebral oxygenation have been used to monitor the central nervous system. ICP monitors have been advocated by some and found to be not helpful by others.[23,24] Critical decreases in cerebral oxygenation have been reported in the first hours after drowning. Monitoring cerebral oxygen saturation with blood analysis from jugular bulb catheters has been reported to aid in the management of cerebral hypoxia.[85] Another study reported no differences in global cerebral blood flow, ICP, or cerebral perfusion pressure among normal survivors, those who became neurologically compromised, and those who died.[50] The electroencephalogram (EEG) has limited value except for detection of seizures.[23] Severe brain injury may be detected by CT scan; however, in most instances of drowning, early evaluation using this modality is usually normal and therefore has limited utility.[170,186] Magnetic resonance imaging (MRI) and magnetic resonance spectroscopy (MRS) provide earlier indications of brain tissue damage and metabolism than does CT and have shown promise in the prognostication of pediatric victims of drowning.[56] Brainstem auditory-evoked potential response and somatosensory-evoked potentials remain either controversial or without sufficient data to support their use.[90]

Inappropriate secretion of antidiuretic hormone (SIADH) in patients with CNS damage leading to retention of free water is not uncommon. Diuretics and mannitol have been recommended if there is evidence by examination and imaging studies of cerebral edema, but no studies have documented that osmotic diuretics improve the neurologic outcome of drowning victims. Free oxygen radicals are generated during reperfusion, although the benefits of free radical scavengers have not been established for therapy.[23] Because glucose is a vital metabolite for the nervous system, fluid management for resuscitation of the CNS should also focus on preventing hyperglycemia.[18,23] Glucose level should be maintained in the 80 to140 mg/dL range. In the rare case of CNS-mediated hyperthermia, temperature should be maintained near normal with the use of cooling modalities.

A number of "neuroprotective" therapies have been used to minimize excess metabolic activity in the injured brain. Thus, seizures in submersion victims should be controlled. Diphenylhydantoin has been used for its neuroprotective effects as well as for reduction of neurogenic pulmonary edema.[4] The attempt to slow brain metabolism in a pediatric age group with barbiturates did not demonstrate beneficial results.[140] Secondary brain damage after ischemia occurs in part due to increase in intracellular calcium and release of excitatory amino acids. Calcium channel blockers, which are protective in experimental ischemic brain damage, may be helpful in improving neurologic outcome. Nimodipine and lidoflazine have achieved some popularity in clinical use.[28,168] Specific studies on the use of calcium channel blockers in submersion patients with neurologic injury have not been conducted. The data on nimodipine that showed great promise were found in the setting of subarachnoid hemorrhage. In the setting of ischemic stroke, there was no benefit shown, and in fact, a meta-analysis of the data resulted in the premature termination of the Very Early Nimodipine Use in Stroke (VENUS) trial. Therefore, use of a calcium channel blocker in the submersion patient with neurologic injury should be done in consultation with a neurologist. The primary detrimental side effect seen is hypotension.[98] Despite its role in blocking both ligand and voltage-dependent calcium entry into the cell, magnesium has no confirmed role in drowning management.[188]

In the past, controlled hyperventilation to a $PaCO_2$ of 30 mm Hg was advocated as a neuroprotective measure for elevated ICP in the setting of trauma. It was believed that by decreasing cerebral $PaCO_2$, the resultant cerebral vasoconstriction would decrease intracranial pressure. This theory has not been validated scientifically and has even been shown to worsen certain outcomes. It is vital to consider elevation in intracranial pressure when selecting RSI medications. One recommended combination would include premedication with lidocaine, vecuronium and fentanyl, followed by etomidate and succinylcholine for sedation and paralysis.[191]

Corticosteroids for lung or cerebral injury were at one time advocated in the treatment of drowning. The role of corticos-

teroids in the management of near-drowning was reviewed by Foex. His review of the literature found little evidence to support the use of intravenous steroids in this setting.[61] Steroids are not recommended due to side effects that may complicate the clinical picture.[18,47,69,115,125] Immunosuppression, suppression of fever, and hyperglycemia are all potential deleterious side effects of corticosteroid use.

Renal and Metabolic

Hemolysis and myoglobulinuria due to muscle trauma, hypoperfusion, acidosis, and hypoxia have been implicated in the etiology of acute tubular necrosis and renal failure in drowning victims.[87,94,104] An indwelling urinary catheter should be placed to monitor urine output, and blood markers of renal function and tissue damage (creatine phosphokinase, CPK, and urine myoglobin) should be followed. Dialysis may be necessary to correct volume overload from oliguria, hyperkalemia, or profound acidosis. Osmotic diuresis with mannitol and infusion of sodium bicarbonate are often recommended, although there are few data to support the efficacy of their use.[79] A favorable prognosis usually follows the predicted course for rhabdomyolysis and acute tubular necrosis.[87] The clinician should be mindful of hypoglycemia secondary to alcohol ingestion and subsequent electrolyte derangements.[57]

Decompression Illness

Post-hypoxic encephalopathy and hypothermia may cloud the diagnosis of decompression sickness in a near-drowned scuba diver. If the clinical scenario suggests that decompression sickness and the existence of intravascular gas are likely, use of hyperbaric oxygen therapy should be entertained. In an unconscious diver, administration of critical care management should be instituted prior to and during hyperbaric oxygen therapy.[129]

See Chapter 70 for more information on decompression illness.

Infections

Pulmonary infections that develop after near-drowning are infrequent and often difficult to diagnose. When they occur, mortality may be as high as 60%.[58] Both bacterial and fungal pathogens have been implicated. Aspirated lake, pond, or canal water is much more likely to be contaminated than swimming pool or hot tub water is. Infections from *Pseudomonas* and *Vibrio* species are more likely from ocean-water aspiration. Leptospirosis may result from aspiration of water containing rat urine.[79] Water may be swallowed and later vomited, with subsequent aspiration of gastric contents and oropharyngeal microorganisms. *Streptococcus pneumoniae* and *Staphylococcus aureus,* as well as aerobic gram-positive bacteria from the patient's oropharynx, may contribute to other pulmonary infections.[23] Gram-negative bacteria have also been reported to be virulent pathogens following submersion.[23] *Aeromonas hydrophila* is a facultative anaerobic gram-negative bacterium found in both fresh and saltwater that has been reported to cause fatal pneumonia.[118] Fungi are rarely a serious consideration in drowning victims. Infections from *Pseudallescheria boydii* have been reported weeks to months after submersion incidents.[23] CNS invasion (brain abscess) has been reported with this organism.[116]

Despite the inherent risks of pneumonia and associated infectious complications, prophylactic antibiotics are not widely recommended.[69,141] Daily monitoring of temperature, tracheal aspirates with Gram stain and culture, and chest radiographs should guide the clinician's use of antibiotics. If infection seems likely, then the history of the setting of submersion, duration of hospital stay, and immunocompetence of the patient should dictate the broad nature of initial antibiotic coverage until the regimen can be narrowed to a specific drug (Table 68-6).

TABLE 68-6. Type of Environmental Exposure Related to Organisms Causing Near-Drowning-Related Pneumonia

ORGANISM	FRESH WATER	SALT WATER	CONTAMINATED WATER
Anerobic Gram-Negative Bacteria			
Aeromonas species	***	*	*
Burkholderia pseudomallei	**		*
Chromobacterium violaceum	**		**
Francisella philomirgia	Iso	**	
Klebsiella pnuemoniae		*	
Legionella species	*		
Neisseria mucosa		*	
Pseudomonas aeruginosa	*	Iso	**
Shewanella putrefaciens		*	
Vibrio species	Iso	*	
Aerobic Gram-Positive Bacteria			
Streptococcus pneumoniae	**	*	
Staphylococcus aureus	Iso	Iso	
Fungi			
Aspergillus species	Iso	*	*
Pseudallescheria boydii	Iso	Iso	***

***Frequently reported.
**Occasionally reported.
*Rarely reported.
Iso, isolate.
From Ender PT, Dolan MJ: Pneumonia associated with near-drowning. Clin Infect Dis 25:896–907, 1997.

PREDICTORS OF OUTCOME

The poor neurologic outcome of up to one fourth of near-drowning victims has raised the question about the justification for prolonged and vigorous resuscitation in hospital settings. Investigators have sought features to predict which child victims of submersion injuries will have normal outcomes. Functional recovery occurs in approximately 17% of those who still require resuscitation in the ED.[164] Hospital assessment criteria have been developed to predict the outcomes of injured children, and various factors have been used to prognosticate a victim's course.[41,47,91,97,158] Others have indicated that these tools may miss potential survivors.[68] Despite the attempts to find the combination of variables, no prognostic system can predict outcome with complete reliability. It is therefore recommended that drowning victims be treated aggressively for 48 hours and reassessed periodically to determine if meaningful recovery is possible.[163] Variables known to influence outcome are listed in Box 68-8.

On the other hand, as high as 6.3% of the patients predicted to have poor outcome on admission actually survived intact. One report concluded that individual outcomes cannot be reli-

Box 68-8. Prognostic Signs in Submerged Victims

GOOD
Alert on admission
Hypothermic
Older child or adult
Brief submersion time
On-scene basic or advanced life support (probably most important)
Good response to initial resuscitation measures

POOR
Age younger than 3 years
Fixed, dilated pupils in emergency department
Submerged longer than 5 minutes
No resuscitation attempts for more than 10 minutes
Preexisting chronic disease
Arterial pH ≤7.10
Coma on admission to emergency department

ably predicted in the ED, and aggressive resuscitation should continue for 48 hours. Withdrawing therapy from survivors who show no clinical improvement after such time should be considered.[41]

PREVENTION AND SUBMERSION SURVIVAL

Preventing submersion accidents and optimizing survival by preparation for water entry ahead of time are two measures that are more successful in achieving positive outcomes than any post-accident medical care.

Ideally, optimizing the likelihood for survival requires a prerehearsed response by individuals anticipating being near or in the water. Unfortunately, such preparation is rarely done. Additionally, a victim's response at the time of an unanticipated event often results in panic. For persons going into a wilderness environment where water might be encountered, preparation must be both practical and mental. Astounding stories of survival of victims submerged in cold water most often involve professionals or military personnel who had spent a great deal of time around cold water, wore proper survival clothing, and had been trained in responses that optimize chances for rescue and survival.

The victim who suddenly finds himself thrown into water must maintain adequate respirations and minimize heat loss. Although it is a response that may seem counterintuitive at the time of sudden immersion, the victim must stay relaxed, remember to turn his head to breathe, and remain in a curled position. Positioning one's body into a "cannonball" results in efficient flotation and minimal body heat loss. Breathing can be achieved by rhythmically turning the head to breathe as the face-down body bobs to the surface. Achieving adequate respiration with this maneuver becomes very difficult in rough water, currents, and tides or when there is concurrent injury, but the chances for successful survival are increased with practice. Trying to swim a great distance in hope for rescue increases exposure of the body's surface area to cold water and thus heat loss by convection.

Individuals anticipating recreation in a water environment must have a premeditated plan, which should include excellent equipment, practiced swimming, rescue, and life-saving and resuscitation skills. Boats, canoes, kayaks, and rafts must be in top condition, meet accepted standards, and be equipped with U.S. Coast Guard-approved life vests or other flotation devices. Courses on boating safety, rescue, and navigation are offered by the Coast Guard in most waterways.

Survival after Prolonged Submersion: Who Drowns and Who Doesn't

Dramatic recoveries after prolonged hypothermia have been well documented in children and adults.[155,177,187,199] Such cases drive heroic attempts to save lives. Two theories have been proposed to account for these outcomes: hypothermia and the diving reflex.

Hypothermia has a protective benefit if it is coincident with the submersion event. Immersion in cold water (<41°F [5°C]) produces a rapid fall in temperature. Heat loss through convection and conduction is further compounded by increased muscle activity during swimming and struggling. The relative greater surface area in children compared to adults may also help to contribute to the rapid cooling.[24,47,72,81] Some speculate that the protective effect of cold water is enhanced by the absorption of cold water into the circulation of a small child.[47] Others postulate that enough water cannot possibly be absorbed to account for this difference.[72] The subsequent brain cooling, together with decreasing metabolic demands and oxygen requirements, may protect the brain as the body recovers.

The diving reflex consists of breath-holding, bradycardia, and intensive vasoconstriction in all vascular beds except the heart and brain, triggered by facial immersion in cold water or by fear. This triad results in increased mean arterial pressure (MAP), decreased cardiac output, and preservation of perfusion of these two most important organs. This reflex, most noted in water animals such as seals, can be induced in some humans. In children, this reflex may be pronounced and may be a contributing factor to some successful outcomes.[18,57,72,94] Others discount its role.[81]

The Definition of Survival

Medicine has made huge strides in refining both techniques and management protocols for a multitude of disease states. Management of submersion reflects this trend. As we continue to save such victims, assessment of neurologic function becomes of paramount importance. Despite normal MRI and CT scan studies, longitudinal psychological monitoring may uncover deficits not apparent by initial imaging, because sequelae develop many years later. The importance of understanding these parameters to allow more complete understanding regarding improvements for quality of life cannot be understated.[88]

The decision to end CPR in drowning cases is an emotionally charged issue. Stress, and at times anger, may affect health care providers. Factoring all information contributed by prognostic indicators and utilizing the best techniques and judgment should set the foundation for a sound decision. To maintain and preserve cardiac function at the expense of an intact survivor is a challenging medical and ethical dilemma. In such cases, professional counseling for the victim's family and medical team debriefing conferences should be considered.[59] Organ donation is an issue that may become part of the discussion.

Prevention

In wilderness medicine, preparation and prevention are of paramount importance. Individuals venturing into remote settings should assume risk and be mentally trained and prepared to act rationally if an accident occurs.

The majority of submersion cases are preventable.[23,112] Although the preventive measures are multifactorial, the following strategies appear to be the most successful in preventing submersion injuries.[66,82,179] Many countries have realized reductions in deaths as a result of prevention programming.[132]

Pools

The largest impact on prevention has occurred with swimming pools, in particular, legislation regarding pools and fences. This type of protection in and of itself has the potential to reduce the incidence of drowning by approximately 50%.[164,175] The inadequacies of three-sided pool fencing and the importance of four-sided pool fencing (5 ft [1.5 m] high minimum) with appropriate locking gate has been presented.[35,108,182] Proper gate closure is emphasized, as a fence alone does not preclude drowning.[25] In-ground pools should be filled so that the water level is as close as possible to the surrounding external ground. In this setting, a struggling person who reaches the side of the pool can simply roll or flop out and does not have to pull up to gain exit from the water.

Pool alarms and rigid pool covers may provide protection but should not be used in place of fencing.[32] Fish and garden ponds should be eliminated.[108] Unfortunately, passive strategies tend to have little effect on adolescents. Primary strategies that require thought and judgment have also usually not been effective in adolescents.[179]

Legislation

Inadequate enforcements of ordinances and inadequate operation or maintenance of poor barrier equipment was a contributing factor in childhood drowning[130]; however, active legislation has shown dramatic reductions in drowning (58%) when such local ordinances are in place.[175] Community-wide drowning prevention campaigns also demonstrated a modest increase in the use and ownership of life vests among children.[20]

Supervision

Inadequate supervision has an impact on the incidence of childhood drowning. The quality and proximity of the supervising individual are both significant factors. The case fatality rate for victims supervised by adults was 30%, by lifeguards 42%, and by peers 71%.[164] Young children should never be left alone, even momentarily, where there is standing water.[7,37,82,178]

Prehospital Personnel

On-scene resuscitation has been associated with improved outcomes. Persons designated to attend the scene of victims of submersion should have appropriate training and triage ability to deliver the victim to the appropriate medical center.[82]

Lifeguards

The presence of a lifeguard positively affects the outcome of submersion.[66,179] Scanning by pool lifeguards is associated with decreased rules violations.[78] CPR training and competency is not mandated for lifeguards in all localities. Upgrading lifeguard effectiveness has been recommended.[164]

Box 68-9. Recommendations for Water Safety from the American Academy of Pediatrics

INFANTS AND CHILDREN
- Careful supervision
- Emptying all water containers, such as buckets and child pools
- Not allowing swimming lessons to provide a false sense of security
- Four-sided fences around swimming pools
- CPR instruction and 911 phone access
- Flotation devices

CHILDREN 5 TO 12 YEARS
- Swimming instruction
- Buddy swimming with supervision/lifeguard
- Personal flotation devices
- Knowing the depth of the water
- Recognizing drowning risks, such as skating on thin ice

ADOLESCENTS
- Avoidance of alcohol and drug use
- CPR instruction
- Prohibiting alcohol use during boat operation
- Proper training in SCUBA diving and water-sporting activities
- Recognize rip currents and other dangerous water situations

Swimming Techniques and Boating Instruction

There are no data that indicate swimming lessons decrease the risk of drowning.[32] Smith reviewed studies of the impact of swimming lessons in the prevention of drowning and recommended education of individuals in high-risk environments and who exhibit risky behaviors, but found no evidence for swimming training as a public heath measure to reduce drowning.[179] In fact, swimming ability may lead to overconfidence and swimming in hazardous situations.[5] However, others debate this statement.[65] Although swimming lessons are recommended for ages 4 years and up, educational efforts to explain high-risk situations and avoidance activities may be more beneficial.[5,82,179] Formalized training recertification is suggested as a method to positively impact boating-related submersion deaths. Wetting the head and face before entering the water may reduce the incidence of immersion syndrome.[145]

Personal Flotation Devices

Coast Guard statistics in 2000 indicated that more than 85% of boating-related drowning could have been prevented by personal flotation devices. Flotation devices are therefore widely recommended for both recreational and commercial boat related activities.[35,92,134,163,165] Air-filled swimming aids, such as water wings and floaties, should not replace life preservers.[32]

Alcohol and Drugs

Legal and societal acceptance of alcohol combined with recreational activities is greater than acceptance with driving, and advertisements widely promote its appeal. Any drinking or use of recreational drugs in an aquatic environment is risky and should be discouraged.[18,35,82,163,179] Development of strategies

similar to those used to curtail the use of alcohol and driving may have a similar effect on the use of alcohol while boating.[109]

Medical Concerns

Preexisting health conditions may influence recommendations for recreational water activity and bathing. Product design for water-related items should be analyzed for specific settings. Bath seats for children, for example, should not offer a false sense of safety to leave a child who otherwise would not be left unattended.[138] Children, as well as adults, with epilepsy should consider showering rather than bathing.[163]

The American Academy of Pediatrics has issued water safety recommendations for various age groups (Box 68-9).[7] Similar recommendations have been made by other agencies.[40]

The references for this chapter can be found on the accompanying DVD-ROM.

69 Emergency Oxygen Administration

Eric Douglas and Richard E. Moon

Emergency medical oxygen (O_2) administration can be a critical part of emergency medical care of the ill and injured. Every provider of emergency medical care must be familiar with O_2, its therapeutic value, indications, hazards and techniques for its administration.

O_2 is a colorless, odorless, and tasteless gas that is necessary for cellular metabolism. It makes up 21% of the earth's atmosphere, and although it is not flammable by itself, it is necessary to support combustion. O_2 used for respiration is diatomic and is obtained commercially by fractional distillation of air.[4,5] In the case of an ill or injured person suffering from hypoxia, convenient treatment devices can provide supplemental O_2 as first aid (before emergency medical service personnel arrive), prehospital therapy, or in-hospital emergency medical treatment.

INDICATIONS

Indications for the use of supplemental O_2 include but are not limited to[3,6]:

- Shock, tissue hypoxia
- Hypoxemia (low blood oxygen)
- Pulmonary gas exchange impairment due to trauma, edema, asthma, infection, embolism
- Acute myocardial infarction, cerebrovascular accident
- Decompression illness (DCI), including both decompression sickness (DCS) and arterial gas embolism (AGE)
- Acute mountain sickness (AMS)
- High-altitude pulmonary edema (HAPE)
- High-altitude cerebral edema (HACE)
- Carbon monoxide (CO) poisoning
- Respiratory or cardiopulmonary arrest

CONTRAINDICATIONS

In an acutely hypoxic patient there is no absolute contraindication to the administration of high concentrations of supplemental O_2 for a limited time. However, there is no evidence that routine administration of oxygen is of any benefit to patients with minor or moderate strokes who are not hypoxic.[14] Also, a person with a history of chronic obstructive pulmonary disease (COPD) who is not severely hypoxic or in need of emergency prehospital care and transport to an emergency department should not receive more than prescribed amounts of supplemental O_2.

For such patients, treatment guidelines are optimally based on achieving target arterial oxygen tensions (or saturations) rather than on predetermined concentrations or flow rates of inspired oxygen. Evidence suggests that in patients with impending respiratory failure due to an acute exacerbation of COPD, the arterial oxygen saturation should be maintained no higher than 85% to 92% to minimize the rise in P_{CO_2} that can accompany oxygen administration.[13]

Pulmonary Oxygen Toxicity

In situations in which a high concentration of supplemental O_2 is administered for many hours, pulmonary O_2 toxicity is possible, particularly if a diver with decompression illness subsequently requires hyperbaric oxygen. The first symptoms of pulmonary O_2 toxicity are due to tracheobronchitis: substernal burning, chest tightness, and cough. Continued exposure may result in dyspnea and adult respiratory distress syndrome (ARDS). Pulmonary O_2 toxicity is usually reversible with cessation of O_2 therapy or reduction in the inspired concentration.[10]

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- Shock, tissue hypoxia
- Hypoxemia (low blood oxygen)
- Pulmonary gas exchange impairment due to trauma, edema, asthma, infection, embolism
- Acute myocardial infarction, cerebrovascular accident
- Decompression illness (DCI), including both decompression sickness (DCS) and arterial gas embolism (AGE)
- Acute mountain sickness (AMS)
- High-altitude pulmonary edema (HAPE)
- High-altitude cerebral edema (HACE)
- Carbon monoxide (CO) poisoning
- Respiratory or cardiopulmonary arrest

CONTRAINDICATIONS

In an acutely hypoxic patient there is no absolute contraindication to the administration of high concentrations of supplemental O_2 for a limited time. However, there is no evidence that routine administration of oxygen is of any benefit to patients with minor or moderate strokes who are not hypoxic.[14] Also, a person with a history of chronic obstructive pulmonary disease (COPD) who is not severely hypoxic or in need of emergency prehospital care and transport to an emergency department should not receive more than prescribed amounts of supplemental O_2.

For such patients, treatment guidelines are optimally based on achieving target arterial oxygen tensions (or saturations) rather than on predetermined concentrations or flow rates of inspired oxygen. Evidence suggests that in patients with impending respiratory failure due to an acute exacerbation of COPD, the arterial oxygen saturation should be maintained no higher than 85% to 92% to minimize the rise in P_{CO_2} that can accompany oxygen administration.[13]

Pulmonary Oxygen Toxicity

In situations in which a high concentration of supplemental O_2 is administered for many hours, pulmonary O_2 toxicity is possible, particularly if a diver with decompression illness subsequently requires hyperbaric oxygen. The first symptoms of pulmonary O_2 toxicity are due to tracheobronchitis: substernal burning, chest tightness, and cough. Continued exposure may result in dyspnea and adult respiratory distress syndrome (ARDS). Pulmonary O_2 toxicity is usually reversible with cessation of O_2 therapy or reduction in the inspired concentration.[10]

TABLE 69-1. Common Portable Medical Oxygen Cylinder Specifications

CYLINDER SIZE	ALLOY	WORKING PRESSURE (psi)	VOLUME (L, cu ft)	LENGTH (in, cm)	DIAMETER (in, cm)	WEIGHT (lb, kg)
M9	Aluminum	2015	246.3, 8.7	10.9, 27.7	4.4, 11.1	3.9, 1.8
D	Aluminum	2015	424.7, 15	16.5, 41.9	4.4, 11.1	5.5, 2.5
D*	Steel	2015	410.4, 14.5	16.75, 42.5	4.4, 11.1	7.5, 3.4
Jumbo D	Aluminum	2216	648.3, 22.9	17, 43.2	5.3, 13.3	9.0, 4.1
E	Aluminum	2015	679.4, 24	25.6, 65.0	4.4, 11.1	8.0, 3.6
E*	Steel	2015	682.0, 24.1	25.75, 65.4	4.4, 11.1	10.5, 4.8

Note: Aluminum cylinder specifications provided by Luxfer Inc.
*Steel cylinder specifications provided by Pressed Steel Tank Co.

The rate at which O_2 toxicity develops depends on the inspired O_2 partial pressure and the individual's susceptibility. In some individuals, continuously breathing 100% O_2 at normal atmospheric pressure (1 ATA) may cause symptoms to appear in as little as 6 hours; however most people can safely breathe O_2 at a fractional inspired concentration (FIO_2) of 1.0 for 12 hours. The rate of onset of symptoms may be reduced by the use of periodic "air breaks," during which the patient breathes air for 5 to 10 minutes.

Central Nervous System Oxygen Toxicity

Central nervous system (CNS) O_2 toxicity can only occur when a person is exposed to O_2 at ambient pressures greater than 1 ATA. The inspired partial pressure of O_2 (PIO_2) is calculated by multiplying FIO_2 by ambient pressure in ATA ($PIO_2 = FIO_2 \times$ ATA). Signs and symptoms are most likely to appear at PIO_2 greater than 1.6 and include (but are not limited to) sweating, bradycardia, mood changes, visual field constriction, twitching, syncope, and seizures. During hyperbaric O_2 therapy the likelihood of CNS O_2 toxicity is reduced by the use of periodic air breaks.

EQUIPMENT

Cylinders

Medical O_2 cylinders or tanks are made of aluminum or steel and come in a variety of sizes (Table 69-1; Figs. 69-1 and 69-2). The working pressure of steel medical O_2 cylinders is 2015 psi. The working pressure of aluminum O_2 cylinders is either 2015 psi or 2216 psi, depending on the type.

Oxygen cylinders in the United States are usually painted green or have distinctive green shoulders. They come in two practical field sizes: D (20 inches [50.8 cm] in length; carries 360 L of oxygen) and E (30 inches [76.2 cm] in length; carries 625 L of oxygen). The length of time that oxygen can be delivered is calculated by dividing the tank capacity by the flow rate.

In the United States, any pressure vessel that is transported on public roads is subject to U.S. Department of Transportation (U.S. DOT) regulations. The U.S. DOT requires that cylinders be visually and hydrostatically tested every 5 years and either be destroyed if they fail or be stamped and labeled appropriately if they pass.[16] Gas suppliers will not fill cylinders that have not been appropriately tested and stamped.

Figure 69-1. Oxygen cylinders come in a variety of sizes and capacities. Cylinder decisions should be based on the amount of time you will likely need to provide care until you can get the injured person to emergency care. (Courtesy of Divers Alert Network [DAN].)

Valves

Valves for medical O_2 cylinders sold in the U.S. are designed to accept only medical O_2 regulators to avoid the possibility of using a medical O_2 regulator with an incompatible gas such as acetylene. The two types of valves available in the United States

Figure 69-2. The components of a portable oxygen cylinder include a cylinder, cylinder valve, and pressure regulator, along with various delivery masks including both constant flow and demand systems. (Courtesy of Divers Alert Network [DAN].)

Figure 69-3. Multifunction regulators allow one to provide oxygen using a constant flow device, by demand-type device, or both at the same time. (Courtesy of Divers Alert Network [DAN].)

are the CGA-870 and the CGA-540. The CGA-870 is also known as the pin-index valve and is used on smaller portable cylinders (e.g., D, E). The CGA-540 is used primarily on larger, nonportable cylinders, such as those mounted in ambulances (e.g., H, M).

There are a number of other valve types manufactured and used with medical O_2 throughout the world. For example, there are adapters available to make a U.S. pin-index regulator fit on an Australian bull-nose valve, but it must be noted that the use of adapters is discouraged by the U.S. Compressed Gas Association (CGA).

Regulators

The device that mounts directly to the cylinder is the regulator. Its function is to regulate the flow rate of the O_2 by reducing the pressure of the O_2 from either 2015 psi or 2216 psi to a usable flow rate. Regulators are primarily of three types: constant flow only, demand/flow-restricted oxygen-powered ventilator (FROPV) only, or multifunction, which has both constant flow and demand/FROPV capability (Fig. 69-3).

The regulator mounts to the cylinder with a matching-type valve. A pressure gauge allows the user to monitor the amount of O_2 in the cylinder. The pressure gauge reading indicates how much oxygen remains in the cylinder. Thus, when the gauge indicates a pressure of 500 psi, one-quarter of the tank's capacity for oxygen remains. At a reading of 200 psi, a tank is near empty.

The pressure gauge, pressure-reducing valve, and flowmeter combine to create the regulator, which reduces the pressure of the oxygen from that inside the tank to approximately 50 psi. This allows delivery to the victim at flow rates between 1 and 15 L per minute.

Other features may include a diameter index safety system (DISS) fitting for an FROPV, a constant flow controller device (either knob or gauge), or both, as in the case of the multifunction regulator.

Devices for Ventilation of Nonbreathing Patients

All of the following devices keep direct patient contact at a minimum to reduce the risk of disease transmission. Other body substance isolation equipment (e.g., gloves, goggles) and practices should be observed as well.

In addition, when used on a nonintubated patient, all of the devices discussed below depend on adequate mask seal to be able to deliver adequate ventilations and ensure adequate respiration. The single most common cause of inadequate ventilation and respiration is poor mask seal.

Bag-Valve Mask

The bag-valve mask (BVM) consists of a mask, bag, and valves that control or direct the flow of air and O_2. Like the FROPV, the mask can be changed to different sizes to accommodate different faces or can be used directly with an endotracheal tube. The volume of the bag is 1600 mL in most commercially available models (Fig. 69-4).

An adult bag-mask device should have the following features: (1) a nonjam inlet valve system allowing a maximum oxygen inlet flow of 30 L/min; (2) either no pressure relief valve or, if a pressure relief valve is present, a pressure relief valve capable of being closed; (3) standard 15-mm/22-mm fittings; (4) an oxygen reservoir to allow delivery of high concentrations of oxygen; (5) a nonrebreathing outlet valve that cannot be obstructed by foreign material; and (6) ability to function satisfactorily under common environmental conditions and extremes of temperature.[1]

An advantage of the BVM is that although it works best with supplemental O_2, it will function on room air if the O_2 supply is depleted. In addition, in intubated patients, experienced health care providers may be able to "feel" decreased lung compliance.

Figure 69-4. Bag-valve masks are commonly used to resuscitate an injured person using 100% oxygen. They are relatively inexpensive and very effective, although they can be tiring for the user and require specific training. (Courtesy of Divers Alert Network [DAN].)

Figure 69-5. Resuscitation mask allows rescuers to resuscitate an injured person either with their exhaled breath or with supplemental oxygen. (Courtesy of Divers Alert Network [DAN].)

The primary disadvantage is that it requires training and practice to effectively use a BVM, and even with much practice, many find it is difficult to maintain adequate mask seal and ventilate sufficient volumes when only one rescuer is available to use it. Even with proper training, few individuals can maintain adequate mask seal and a patent airway with one hand while squeezing the bag fully to achieve the 700 to 1000 mL standard volume. The U.S. DOT National Standard Curricula for First Responder to Paramedic recommends the BVM be used first with two rescuers (one maintaining mask seal and patency of the airway, the other squeezing the bag). The NSC recommends that a BVM with one rescuer be the last choice (after all other devices and techniques) in ventilating a patient.[16]

In addition, there is no overpressurization relief valve. This is rarely a concern in unintubated patients because of the aforementioned difficulties in achieving even minimally acceptable ventilatory volumes, but it is of concern in intubated patients.

When using a BVM:

- Attach the oxygen tubing from the BVM to the constant flow barbed outlet on the oxygen regulator.
- Set the constant flow controller to 10 to 15 L/min.
- Position one rescuer at the victim's head to maintain the victim's airway and ensure a mask seal.
- The second rescuer should ventilate the victim by squeezing the bag with both hands.
- Ventilations should last 1 second as part of CPR.
- Alternate two ventilations with 30 chest compressions.
  - If the ventilations do not go in, have the first rescuer reposition the victim's airway.
  - If the ventilation still does not go in, check for airway obstruction and initiate airway obstruction protocols.
- Ventilate with enough speed and force to make the victim's lower chest and upper abdomen rise.
- It is not necessary to empty the bag with each ventilation.

Resuscitation Mask

The pocket-type resuscitation mask consists of a clear, flexible plastic mask designed to fit over the mouth and nose of the victim while the health care provider ventilates by exhaling forcefully through the "chimney" (Fig. 69-5). There is usually a one-way valve that directs the rescuer's breath into the victim while at the same time directing the exhaled breath of the victim away from the rescuer. It is a relatively simple device that requires minimal training, is lightweight, and is more likely to be available when equipment is at a minimum. It is available both with and without an outlet for supplemental O_2; however, Guidelines 2005 stress the need for an oxygen inlet.[1]

Mouth-to-mask ventilation has been shown to be superior to that with bag-mask devices in delivering adequate tidal volumes to manikins.[16]

An adequate seal is best achieved with a mouth-to-mask device when the rescuer is positioned at the top of the patient's head. The rescuer ventilates the victim by sealing his or her lips around the coupling adapter of the mask, using both hands to hold the mask securely in position and maintain airway patency with head tilt.[17]

When using the pocket-style resuscitation mask:

- Remove the oxygen tubing from a nonrebreather mask.
- Stretch the hose out to make sure there are no kinks.
- Attach the oxygen tubing to the constant flow barbed outlet on the oxygen regulator.
- Connect the other end of the tubing to the oxygen inlet on the resuscitation mask.
- Set the constant flow controller to 10 to 15 L/min.
- The rescuer should ensure a proper mask seal by positioning the mask over the victim's mouth and nose, lifting the jaw up into the mask.
- The rescuer should take a breath away from the mask and then breathe into the one-way valve on the mask to make the victim's lower chest and upper abdomen rise.
- Ventilations should last about 1 second as part of CPR.
- Alternate two ventilations with 30 chest compressions.
  - If the ventilations do not go in, reposition the victim's airway.
  - If the ventilations still do not go in, check for airway obstruction and initiate airway obstruction protocols.

FROPV/Positive Pressure Demand Valve

Older style positive-pressure demand valves (PPDVs), such as the LSP 063-05 or Elder CPR Demand valve (both manufac-

tured by Life Support Products/Allied Healthcare Inc., St. Louis, Missouri), function both in positive-pressure mode (pushing the button to ventilate a nonbreathing patient) and in demand mode.

There has been a misconception that a PPDV will easily cause pulmonary overpressurization injury, and thus this valve has fallen out of favor with some health care providers. In fact, in positive-pressure mode, all PPDVs manufactured in the United States are required to have an overpressure relief valve that stops the flow of gas at a pressure of 55 to 65 cm H_2O (a pressure at which lung overpressurization is unlikely). The most recent model introduced in 1993, the MTV-100 FROPV (LSP/Allied), has two overpressure relief valves, the first set at 60 cm H_2O and the second at 65 to 80 cm H_2O.*

With respect to the positive-pressure mode, earlier PPDVs were originally designed to meet the Emergency Cardiac Care Committee (ECC) cardiopulmonary resuscitation (CPR) guidelines before 1986, which called for "four quick initial breaths and then two quick breaths after every 15 compressions."[8] This faster rate of ventilation was equivalent to 160 L/min.

In 1986, CPR standards were changed to "two slow breaths, each one and one-half seconds in duration."[8] The standard changed again in 1992 to "two slow, full breaths, with a duration of $1^1/_2$ to 2 seconds each" (equivalent to 40 L/min).[8]

In 1993 a new style PPDV called a flow-restricted oxygen-powered ventilator (FROPV) (MTV-100, manufactured by LSP/Allied) was introduced. Its specifications include a flow rate of 40 L/min while being used in positive-pressure mode and 115 L/min in demand mode, eliminating difficulties associated with earlier models (Fig. 69-6).

The mask adapter is a standard 15-mm fitting that fits a variety of masks and can also be used directly with an endotracheal tube. The disadvantages of the FROPV are that a supply of O_2 is required for its use and that in intubated patients the health care provider will not be able to "feel" decreased lung compliance.

When using an FROPV:

- Connect the FROPV to the DISS threaded port on the oxygen regulator.
- It is not necessary to adjust the oxygen flow rate on the regulator.
- Position one rescuer at the victim's head to maintain the victim's airway and ensure a mask seal.
- The second rescuer should ventilate the victim by depressing the button on the FROPV.
- The rescuer should place a second hand on the victim's upper abdomen and lower chest to monitor chest rise.
- Ventilations should last about 1 second as part of CPR.
- Alternate two ventilations with 30 chest compressions.
  - If the ventilations do not go in, have the first rescuer reposition the victim's airway.
  - If the ventilation still does not go in, check for airway obstruction and initiate airway obstruction protocols.

A

B

Figure 69-6. **A,** Flow-restricted oxygen-powered ventilators allow the rescuer to resuscitate an injured person using 100% oxygen. These devices have pressure restrictions to reduce the likelihood of harm to the injured person. The ventilators are more expensive and require additional training, although they are less tiring for rescuers than a bag-valve mask **(B)**. (Courtesy of Divers Alert Network [DAN].)

Demand-Only, or FROPVs in Demand Mode

When used in demand mode, the recipient simply holds the mask to his or her face. When he or she inhales, negative pressure in the mask and demand valve opens the valve, and gas flows (Fig. 69-7). The flow of gas stops when the person stops inhaling or exhales, similar to other demand regulators, such as scuba and aviation regulators.

When using a demand-only valve:

- Connect the demand valve to the DISS threaded port on the oxygen regulator.
- It is not necessary to adjust the oxygen flow rate on the regulator.

*Specifications from Allied Healthcare, Inc./Life Support Products.

A

B

Figure 69-7. **A,** Demand valves deliver 100% oxygen to an injured person when he or she inhales—much like a scuba diving regulator. This device can only be used by conscious, breathing persons **(B)**. It has the advantage of delivering high concentrations of gas and limiting waste compared to a constant flow device. (Courtesy of Divers Alert Network [DAN].)

A

B

Figure 69-8. The nonrebreather mask **(A)** is the simplest oxygen delivery device. It can deliver high concentrations of oxygen, although rescuers must be careful to ensure a good mask seal or the concentration of inspired oxygen drops **(B)**. It also wastes gas as the oxygen flows even when the injured person is not breathing or is exhaling. (Courtesy of Divers Alert Network [DAN].)

- Ask the victim to breathe normally from the mask.
- Monitor the victim to ensure breathing.
- Watch the clear mask to make sure it fogs with each exhalation.
- If the victim is not conscious or breathing adequately to activate the demand-only valve, it is necessary to switch to a constant flow delivery device such as the nonrebreather mask.

Constant Flow Devices for Adequately Breathing Patients

The Nonrebreather Mask

This is the first choice when considering constant flow supplemental O_2 in an acute medical emergency. It consists of a mask, reservoir bag, and two or three one-way valves, one separating the reservoir from the mask and the other one or two on the sides of the mask. Oxygen flows into the reservoir bag so that when the patient inhales, he or she inhales O_2 from the reservoir. The one-way valves on the sides of the mask keep air from coming into the mask and diluting the O_2. When the patient exhales, expired air goes out of the mask through the one or two valves on the face and is prevented from entering the reservoir (Fig. 69-8).

The efficiency of this mask depends on the mask fit and seal and proper functioning of the valves. Under ideal conditions this mask (when fitted with all three valves) may deliver an FIO_2

of up to 0.95. Field studies show it may deliver FIO_2 as low as 0.60, but it is still the most effective constant flow device available (except for demand regulators and O_2 rebreathers).[15]

To use the mask, it is attached to the O_2 supply at a flow rate of 10 to 15 L/min. The reservoir bag must be inflated or "primed" before placing it on the patient; some new models are designed to be self-priming and will fill automatically when connected to the oxygen flow. Priming can be accomplished by placing a thumb or fingers on the valve between the reservoir and mask while the reservoir inflates.

The mask is available with either two one-way valves on the sides or with only one (labeled as "with safety outlet") and referred to as a partial rebreather. If the mask has only one valve on the side of the mask, it will deliver reduced FIO_2.

The advantage of the nonrebreather mask is that it provides the highest FIO_2 of the constant flow devices. However, it also wastes O_2 and may not deliver a high FIO_2 under less than ideal conditions. Care must be taken to monitor the patient and O_2 supply closely to avoid allowing the tank to empty while the mask is still on the patient's face. Because of the one-way valves, if there is no O_2 supply, suffocation may result. A patient breathing from a nonrebreather mask must never be left alone.

When using a nonrebreather mask:

- Open the nonrebreather mask packaging and stretch out the tubing to make sure there are no kinks.
- Connect the tubing on the nonrebreather mask to the constant flow barbed outlet on the oxygen regulator.
- Set the constant flow controller to 10 to 15 L/min.
- Place a thumb over the one-way valve between the mask and the reservoir bag.
- Allow the bag to fill completely before placing the mask on the victim.
- Position the mask over the victim's mouth and nose.
- Pinch the nose clip over the victim's nose.
- Pull on the elastic straps to tighten the mask to the victim's face and pull the skin into the mask.
- Ask the victim to breathe normally from the mask.
- Monitor the victim to ensure breathing.
- Watch the clear mask to make sure it fogs with each exhalation.

Nasal Cannula

The only other recommended constant flow device for prehospital emergency O_2 administration is the nasal cannula.[16] This is recommended when the patient will not tolerate any kind of mask, such as a person with a long history of COPD, or when high inspired O_2 concentrations are not required. In this case it is believed that some supplemental O_2 is better than none. It must be understood that a nasal cannula is capable of delivering FIO_2 of only 0.24 to 0.29.[14]

Flow rates for a nasal cannula are limited to 1 to 6 L/min. Flow rates exceeding 6 L/min are extremely uncomfortable for the patient and may result in drying of the nasal mucosa. To use the nasal cannula, place the prongs in the patient's nares and loop the tubing over the top of the ears to hold it in place. Adjust the tightness at the neck to a comfortable level.

Other constant flow masks, such as the partial rebreather mask, simple face mask, and Venturi mask, are not recommended for use in prehospital emergency medicine because of low levels of delivered FIO_2.[15,16] Devices such as automatic transport ventilators are not practical for wilderness or remote emergency medicine.

Figure 69-9. A typical oxygen kit created for the marine/aquatic environment includes a multifunction regulator to care for more than one injured person along with delivery masks and a water-tight case to protect the equipment. (Courtesy of Divers Alert Network [DAN].)

When using a nasal cannula:

- Open the cannula packaging and stretch out the tubing to make sure there are no kinks.
- Connect the tubing to the constant flow barbed outlet on the oxygen regulator.
- Set the constant flow controller to 4 to 6 L/min.
- Position the prongs in the victim's nostrils.
- Position the hose behind the victim's head and tighten the straps.
- Ask the victim to breathe normally through the nose.
- Monitor the victim.

Oxygen Rebreathers

One of the problems with long transports commonly seen in the case of wilderness or remote emergency medical care is that all of the previously discussed O_2 delivery devices waste O_2 and require multiple portable or large nonportable cylinders if the transport time exceeds 1 hour (Fig. 69-9). Breathing room air, a person inhales 21% O_2 and exhales 16% O_2. If a person inhales (under ideal conditions) 100% O_2, the exhaled gas will contain 95% O_2 and 5% CO_2. The theory of the design of a rebreather is to remove CO_2 from the exhaled gas, supplement for the 5% O_2 that was metabolized, and reuse the exhaled O_2.

There are several manufacturers of rebreathers for emergency medical O_2 administration. The devices all have the same basic components: a mask, breathing circuit (similar to anesthesia equipment), and canister with an absorbent chemical, usually soda lime.

Soda lime chemically removes CO_2 from the exhaled gas, allowing O_2 to be rebreathed.* Supplemental O_2 is added at flow rates of slightly more than 1 L/min to replace metabolized O_2. The cylinder that can last 45 minutes with a nonrebreather

*$2NaOH + CO_2 = Na_2CO_3 + H_2O$ + heat, from WR Grace, Inc.

mask or a little more than 1 hour on demand now lasts more than 8 hours, and the patient (with proper technique) will still receive FIO_2 of 0.85 to 0.99. In a situation in which equipment is limited because of size and weight, this device may prove invaluable.

Different manufacturers recommend beginning the patient on O_2 during assembly, setting up the unit, flushing the system of air, applying it to the patient, and considering air breaks to minimize the risk of pulmonary oxygen toxicity.

Thermal considerations are important because of the chemical reaction that takes place with soda lime. The reaction produces heat and water, so it provides warmed and humidified O_2. In cold climates, this is an advantage, but in hot climates it may be a disadvantage. If one is in a hot climate, it is recommended to pass the breathing circuit hoses through cold or ice water to cool the gas.

Disadvantages are the training requirement and the fact that the breathing circuit and absorbent canister containing the soda lime are typically single-patient use. Like other O_2 delivery devices, the rebreather also depends on an adequate mask seal to function effectively. Poor mask seal results in dilution of inhaled gas with air and lower FIO_2. There may also be an increase in breathing resistance when compared with a constant-flow mask.

The most common types of resuscitators available on the market today are the American DAN $REMO_2$ system (Fig. 69-10); two German systems, Wenoll and Circulox; and an Australian system, OXI-Saver Resuscitator.[9,11]

To administer oxygen in general:

Figure 69-10. Devices such as the Remote Emergency Medical Oxygen ($REMO_2$) unit recycle an injured person's exhaled breath and add in small quantities of oxygen. A $REMO_2$ unit can extend the useful life of a portable oxygen cylinder from 1 hour to 8 hours. However, these devices require constant supervision and cannot be used for resuscitation. (Courtesy of Divers Alert Network [DAN].)

1. Place the cylinder upright. Open and close the tank valve slowly ("crack the tank") with a wrench to clean debris from the outlet.
2. Close the tank valve and attach the regulator to the tank. Tighten securely by hand. Never use a regulator without the proper oxygen washer. *Never use tape to hold a loose regulator in place.*
   a. It is best and safest, however, to keep the regulator attached to the tank and depressurized at all times. This ensures that the equipment is ready to use and free of debris.
3. Open the tank valve slowly one full turn.
4. Attach the oxygen delivery device to the regulator, either to the DISS threaded port or to the constant flow nipple on the end of the regulator. Attach the breathing mask or nasal cannula to the other end of the hose or tubing, if it is not already attached.
5. Adjust the constant flow controller to the desired flow rate in liters per minute (L/min) when using a constant flow mask.
   a. When using a demand-style mask connected to the DISS threaded port, it is not necessary to adjust the flow rate.
   b. A regulator marking of "low" indicates 2 to 4 L/min, "medium" is 4 to 8 L/min, and "high" is 10 to 15 L/min. The flow rate for a nonrebreather mask should not be less than 6 L/min; the flow rate for a nasal cannula should not be more than 6 L/min.
   c. To ensure proper oxygenation, victims should receive high-flow oxygen (10 to 15 L/min) whenever feasible.
6. Position the mask or cannula on the victim's face. Adjust for comfort. Observe the victim to be certain that the device is tolerated, and that the reservoir bag fills properly.

Precautions:

- Never allow an open flame near an oxygen-delivery system.
- Do not expose an oxygen tank to excessive heat (125°F [52°C]) or freezing cold.
- Do not position any part of a person directly over a tank valve—a loose regulator can be blown off the top of the cylinder with tremendous force.
- Do not drop a cylinder; do not roll a cylinder.
- Close all valves when the cylinder is not in use.

For more detailed information on airway management, see Chapter 19.

SPECIAL CONSIDERATIONS IN NONBREATHING OR INADEQUATELY BREATHING PATIENTS

Considerations for ventilating nonbreathing patients include rate (breaths per minute), volume, flow rate or speed, pressure, and oxygenation. The rate of ventilations per minute is 12 per minute for an adult (over 8 years old) and 20 per minute for children and infants.[8]

The recommended volume for ventilations for an adult is 700 to 1000 mL. If a ventilation device or technique does not have an overpressure relief valve and greater volumes are administered, pulmonary barotrauma (pulmonary overpressurization injury) could result. Ventilatory volumes less than 800 mL may not be sufficient to inflate the alveoli, and thus gas exchange will be inadequate. Each ventilation should be about 1 second

in duration (equivalent to 40 L/min). Faster ventilation rates or speeds force open the esophagus and then force air into the stomach rather than the lungs. Increased gastric insufflation greatly increases the risk of regurgitation and aspiration of gastric contents.[8]

It has been demonstrated that a differential pressure of as little as 90 to 110 cm H_2O has been sufficient to rupture alveolar septa and to allow gas to escape into interstitial spaces.[2,3,9] Care must be taken not to exceed these pressures when ventilating a patient. Humans can easily generate pressures exceeding 120 cm H_2O by exhaling forcefully, and thus according to ECC CPR guidelines, one should "blow until the chest rises" to accommodate various sizes of patients. The only device for ventilating adult patients that has an overpressure relief valve is the PPDV/FROPV.

The primary goal of ventilation is oxygenation of the patient. With mouth-to-mouth or mouth-to-mask breathing without supplemental O_2, FIO_2 will be the same as exhaled gas, which is 0.16, or 16% O_2. Adding O_2 at a flow rate of 15 L/min may increase the FIO_2 with a pocket mask to up to 50%. A BVM on room air is 0.21, and with O_2 at 15 L/min, up to 0.9, depending on equipment and the skill of the ventilator. An FROPV delivers close to 1.0, or 100% O_2.[3,8]

Both volume and oxygenation achieved by ventilations depend on quality of the mask seal and airway patency. The single most common cause of inadequate ventilation in a nonintubated patient is poor mask seal. Great care must be taken to ensure that the airway is fully patent and that there is a good mask seal with each ventilation. If the patient is not intubated, an oropharyngeal, nasopharyngeal, or combination airway should be used if available.

The current American Heart Association guidelines, published in 2005, state that if supplemental oxygen (minimum flow rate of 8 to 12 L/min with oxygen concentration 40%) is available, the rescuer skilled in bag-mask ventilation should attempt to deliver a tidal volume of 6 to 7 mL/kg or approximately 400 to 600 mL over about 1 second. Because the actual tidal volume delivered is impossible to determine, tidal volume can be titrated to provide sufficient ventilation to maintain oxygen saturation and produce visible chest expansion. Tidal volume should be sufficient to make the chest rise. It is important to note that this smaller tidal volume may be associated with development of hypercarbia. If oxygen is *not* available, the rescuer should attempt to deliver the same tidal volume recommended for mouth-to-mouth ventilation (10 mL/kg, 700 to 1000 mL). This tidal volume should result in obvious chest rise.[1]

Because an FROPV delivers the highest FIO_2, it is the only device that is limited to 40 L/min flow rate (1 second in duration). Because it has an overpressure relief valve, it may be the best choice for ventilating a person in respiratory arrest, whether or not they are intubated. A BVM unit used by rescuers (one to maintain the mask seal, and the other to squeeze the bag) is the best alternative. This method of ventilation is the first choice for ventilating a person in respiratory arrest, according to the U.S. DOT National Standard Curricula for EMTs (1994), First Responders (1995), and Paramedics (1998).[7,16] According to these same curricula, the following is the order of preference in ventilating a person in respiratory arrest:

1. BVM unit with two rescuers and supplemental O_2
2. Pocket mask with supplemental O_2
3. FROPV
4. BVM unit with one rescuer and supplemental O_2
5. Last choice is mouth-to-mouth breathing. This is not an option for professional rescuers because of the risk of disease transmission.

HAZARDS

Oxygen alone or in a vacuum is not flammable. However, in the presence of flammable substances, combustion can be very vigorous. High concentrations of O_2 can also facilitate conversion of sparks or embers (e.g., lit cigarettes) into full-blown fires. It is imperative to use O_2 only in open, well-ventilated areas and not in the presence of burning materials. Care must be taken when handling O_2 equipment to avoid allowing contaminants such as petroleum products to come into contact with the regulator, particularly in or around the orifices on the cylinder or regulator through which O_2 flows. Cylinders should not be exposed to temperatures above 52° C (125° F).

LEGAL ISSUES

The United States Food and Drug Administration (FDA) regulations regarding O_2 administration equipment state that to qualify as "emergency medical oxygen administration equipment" the device must "be capable of administering a flow rate of at least 6 L/min for a period of at least 15 minutes."[17] Equipment not meeting this minimum standard may not be sold in the United States as emergency medical O_2 administration equipment.

To fill a medical O_2 cylinder, the FDA states, "For emergency use only when administered by properly trained personnel for O_2 deficiency and resuscitation." For all other medical applications, "Caution: Federal law prohibits dispensing without prescription."[18]

The references for this chapter can be found on the accompanying DVD-ROM.

70 Diving Medicine

Kenneth W. Kizer and Karen B. Van Hoesen

Water covers more than 70% of the earth's surface, mostly in the form of oceans. Life on Earth began in the oceans, and the oceans have been central to its sustenance ever since. The oceans moderate Earth's climate, regulate the composition of the atmosphere, produce food for creatures of all types, and are otherwise essential to the habitability of the planet. However, very little of the underwater environment has ever been seen by humans; only recently has this wilderness begun to be explored in a systematic way. Much of this exploration has been possible because of the development of scuba diving.

Scuba diving is an exhilarating and generally safe activity for people who are healthy, well trained, physically conditioned, properly equipped, and "water wise." It can, however, be a demanding and dangerous activity because of the intrinsic hazards of the aquatic environment.

The hazards of diving include the generic problems found in other aquatic activities, such as near drowning, hypothermia, immersion-related skin disorders, water-borne infectious diseases, and hazardous marine life, as well as several relatively unique problems related to the increased atmospheric pressure found underwater and the effects of breathing gases at elevated pressure.

There are many millions of recreational, commercial, and military divers worldwide; hundreds of thousands of new divers are trained each year. More than 9 million certified divers reside in the United States alone.[45] Diving is conducted in every imaginable aquatic setting, including oceans, lakes, reservoirs, rivers, quarries, ponds, and aquariums. Despite the extent of diving activity, diving-related fatalities and serious injuries are relatively infrequent. For recreational divers, the death rate is about 15 to 20 deaths per 100,000 divers per year,[65] and the chance of suffering decompression sickness on any single dive is about 3 in 10,000.[44]

Currently, the best readily available information about diving accidents comes from the Divers Alert Network (DAN), a private, nonprofit dive safety organization associated with Duke University Health Systems in Durham, North Carolina. Since being established in 1980 to provide emergency medical assistance to recreational divers, DAN has grown to be a worldwide organization having well over 200,000 members and a broad portfolio of activities (www.DiversAlertNetwork.org).

Since 1987, DAN has reported annually on the occurrence of diving-related injuries and deaths from its worldwide network of hyperbaric chambers and associated organizations. In the 2005 edition of DAN's *Annual Report on Decompression Sickness, Diving Fatalities and Project Dive Exploration,* a total of 78 dive-related fatalities and 296 nonfatal injuries from the United States and Canada are reviewed for 2003, the latest year having complete information.[45] Reporting to DAN from other parts of the world is incomplete and variable, so these data are not included in the report. The number of dive-related accidents in North America reported by DAN has been relatively stable over the past two decades, with perhaps a small downward trend, although one has to be careful not to overinterpret this observational data because of the lack of complete information about the frequency and type of diving performed by recreational divers. Notwithstanding the apparent stable or downward trend in diving-related fatalities, concern about the safety of diving continues because of the changing character of the sport-diving population and the increasing complexity and technical nature of recreational diving.

Since scuba diving made its debut in the United States in 1951, the nature of the diving population has substantially changed. Scuba divers of the 1950s and 1960s were generally "water people"—well-trained, vigorous athletes experienced in breath-hold diving and competitive swimming. To don a scuba tank and regulator was a natural extension of familiar activity. These early scuba divers seldom encountered problems that required the attention of the general medical community. However, as scuba diving equipment became more available, adventure-minded persons from all walks of life became attracted to the sport. The popularization of scuba diving in recent years has attracted participants who are poorly conditioned, have little or no experience in aquatic or other sports, are of advanced age, have significant underlying medical conditions, or are even severely disabled. Because of the hostile and unforgiving nature of the aquatic environment, such persons may be at increased risk of a diving-related injury or illness. Certain medical conditions are an absolute contraindication to diving.

In the last decade, recreational divers have increasingly sought more technically complicated diving in efforts to increase the amount of time spent underwater or the depth dived. Although these diving techniques have long been used in commercial and military diving under highly controlled conditions, there are significant concerns about the safety of "technical" diving in the typically less-controlled recreational setting.

All primary care physicians should be prepared to answer basic questions about fitness for diving and to initially manage diving-related medical emergencies. Likewise, every emergency medical treatment facility should be prepared to evaluate, provide urgent care, and, if needed, arrange appropriate transport to a hyperbaric treatment facility (recompression chamber) if a diver is suspected to suffer decompression sickness or arterial gas embolism.

This chapter focuses primarily on the pressure-related diving syndromes collectively known as dysbarism; other conditions relevant to diving are discussed in other chapters (e.g., immersion hypothermia in Chapter 6, hazardous marine life in Chapters 72 to 74, hyperbaric oxygen therapy in Chapter 71, and submersion incidents in Chapter 68).

HISTORICAL PERSPECTIVE

Human beings did not evolve for an aquatic existence and are not well adapted for function in the aquatic environment. Nonetheless, man has been breath-hold diving to gather food and other natural resources from the oceans for thousands of years. There is archaeologic evidence that Neanderthal man breath-hold dived for shellfish 40,000 years ago. The Ama of the Izu Peninsula of Japan and the Hae Nyeo of the South Korean Island of Cheju (in both cases women) have been breath-hold diving to collect shellfish, lobster, sea urchins, octopus, and seaweed for at least 6000 years. Women of the Yahgan, Alakaluf, and other nomadic sea-going female Fuegian Indians of southern Patagonia engaged in similar diving practices for probably 5000 years before these primitive native Americans became extinct in the late 19th and early 20th century. The fires that these divers built to warm themselves along the shores of what is now known as the Straits of Magellan inspired Ferdinand Magellan to name the area "Tierra del Fuego" (Land of Fire).

Written records of diving for salvage and military purposes date back to around 500 B.C., when the Greek historian Herodotus recorded the feats of Scyllis and his daughter Cyana as they dived in the Mediterranean Sea for the Persian king Xerxes during the 50-year war between Greece and Persia. Many early cultures around the Mediterranean Sea made use of divers in military operations—usually to cut the anchor cables of ships, bore holes in the hulls of enemy vessels, and build harbor defenses. Aristotle described the use of diving bells (basically, upside down buckets) to supply air to sponge fishermen in 360 B.C., and Alexander the Great is reported to have gone underwater in a specially constructed glass diving bell to observe his divers removing defensive obstructions from the besieged harbor of Tyre in 332 B.C.

Colorful accounts of military and salvage divers dot the history of Roman and other early cultures. By 100 B.C., diving operations around the major shipping ports of the eastern Mediterranean were so well organized that there were legally binding payment schedules, which recognized that the risk to the diver increased with depth underwater.

While written records of diving in the Americas are not known until after European explorers arrived in the New World in the 16th century, Peruvian artifacts dating to A.D. 200 show divers wearing goggles and holding fish, so it is reasonable to assume that breath-hold diving had been practiced long before the Europeans arrived. Spanish explorers are reported to have enslaved native divers and made them dive for pearls in the Caribbean. These explorers also made extensive use of divers to salvage galleons wrecked in the Caribbean and along the coast of Florida. In one such instance, in 1680, Sir William Phipps is believed to have recovered more than £200,000 in sterling silver from a Spanish galleon.

Human underwater exploits remained limited to breath-hold diving until about 300 years ago, when a series of technologic developments began to expand human underwater activity. These developments principally involved the use of different types of external air supply to prolong submergence.

In the seventeenth century, primitive bells containing air were carried from the surface, allowing Swedish divers to stay underwater longer than a single breath and to salvage cannons from Stockholm's harbor.[144] In 1690, Sir Edmund Halley devised a leather tube to carry surface air to barrels, which resupplied air to manned bells at a depth of 60 feet (18 m). These barrels were submerged, and the air they contained was compressed.[51]

The first practical diving suit was fabricated by Augustus Siebe in 1837.[3,51,144] Atmospheric air was supplied to the diver as compressed air from a manually powered pump on the surface. By 1841, French engineers had developed the technique of using compressed air to keep water and mud out of caissons sunk to the bottom of riverbeds for bridge footings and tunnels. Soon thereafter it was noted that people working in a compressed-air environment sometimes suffered joint pains, paralysis, and other medical problems soon after leaving the caisson. This poorly understood condition was called "caisson's disease" and was the first recognition of what is now known as decompression sickness.[147]

Underwater diving remained an esoteric activity having limited commercial and military utility until the 1930s. By that time, and increasingly during World War II, the military importance of submarines and other undersea activities became evident to navies throughout the world. With the development of submarine forces came the need to train men to escape from submarines that became disabled at depth (an all too frequent occurrence in the early days of submarines). Given the shallow operational depths of these early boats, it was usually possible to escape by simply exiting the vessel and ascending to the surface. It was noted early that failure to exhale while ascending through the water led to pulmonary overpressurization accidents and a new and dramatic syndrome that we now know to be arterial gas embolism.

In 1865, the French engineers Rouquayrol and Denayrouse developed a device that could supply air on demand at ambient pressures different from the 1 atmosphere of pressure found at sea level. These inventors were able to supply air on demand at appropriate breathing pressure to persons underwater with a "demand regulator," as it subsequently became known. This device originally required a surface air supply connection.[3] The demand valve regulator was later modified to supply auxiliary oxygen for pilots operating at high altitude. In 1943, while working with the French resistance against Nazi Germany, Jacques-Yves Cousteau and Emile Gagnon combined a demand valve regulator with a compressed air tank, giving rise to what they called "self-contained underwater breathing apparatus," or scuba.

The potential military usefulness of scuba was immediately recognized and led to a considerable amount of investigation during World War II. As initially configured, scuba was used in an open-circuit mode in which exhaled air was just vented into the water. This was wasteful of the compressed air supply and had other disadvantages for military uses. Further work led to refinement of rebreather scuba devices (both closed and semiclosed circuit systems), such as Lambertsen's amphibious respiratory unit.[9] These rebreather systems conserved the breathing gas by using a carbon dioxide scrubber and recirculating all or part of each exhalation (see Rebreather Diving below). These specialized scuba systems were useful for military purposes

Box 70-1. Types of Commercial Diving

- Harvest of natural resources
  - Oil and natural gas
  - Minerals (e.g., gold)
  - Fish and shellfish
  - Pearls, corals, and shells
  - Algae
  - Wood (e.g., underwater logging)
  - Aquaculture
- Salvage and recovery operations
- Maintenance and construction
  - Ship hulls
  - Nuclear power plants
  - Bridges and tunnels
  - Piers and jetties
  - Aquariums
  - Water treatment plants
  - Sewers
  - Dams
- Underwater photography and motion picture productions
- Marine studies
  - Biology
  - Geology
  - Archeology
  - Other sciences
- Rescue operations
- Sport-diving instructors and tour guides

because they allowed longer submergence times and could be used in clandestine operations or when disarming pressure-sensitive explosive devices. However, they had a greater frequency of mishap, so rebreather systems were not widely used until the 1990s when there began a resurgence of interest in them by "technical" recreational divers.

After World War II, development and marketing of open-circuit scuba equipment to the general public made the underwater world accessible to growing numbers of people. In the last four decades, scuba diving has opened the underwater world to millions of divers and hundreds of millions of cinema observers. Scuba is now used as a basic tool with myriad commercial, military, scientific, and recreational applications (Box 70-1).[6]

TYPES OF DIVING AND DIVING EQUIPMENT

There are five general types of diving, each using different equipment and having different logistical support needs. From the least to the most sophisticated equipment used, the types of diving are breath-hold diving, scuba diving, surface-supplied or tethered diving, saturation diving, and one atmosphere diving.

Breath-Hold Diving

Breath-hold diving is the simplest and oldest form of underwater activity, going back many thousands of years. In breath-hold diving, no supplemental air source or underwater breathing device is used, so submergence is limited to the length of time the diver can hold his or her breath.

There are several types of breath-hold diving that are characterized by activity and equipment.

Snorkeling is the most common form of breath-hold diving. Snorkelers typically use a face mask to facilitate underwater vision, fins for propulsion, a snorkel to breathe air while swimming face down on the water's surface, some sort of attire for environmental protection (e.g., a neoprene wetsuit or full-body spandex [Lycra] suit), and sometimes lead weights to counterbalance the positive buoyancy of a wetsuit or one's innate positive buoyancy. Snorkelers often remain exclusively on the surface, breathing through their snorkel with their face submerged, but never actually diving under the water. Snorkeling is widely used at tropical resorts today to introduce people to the beauty of coral reefs.

Freediving generally refers to one of several types of competitive breath-hold diving. Freediving is classified as an "extreme sport," and many consider it to be the original extreme sport.

Competitive freediving dates back to at least the early 1900s, with perhaps the best recorded account involving the Greek sponge diver Haggi Statti Giorgios. In 1913, Giorgios was offered a few dollars to dive more than 200 ft (61 m) underwater to retrieve the anchor of the Italian ship *Regina Margherita,* which had become stuck in the Aegean Sea at a depth of 230 ft (70 m). He freed the anchor after three consecutive dives of between 1.5 and 3.5 minutes' duration, going as deep as 263 ft (80 m). He did not consider this an especially taxing feat, saying that he had dived as deep as 361 ft (110 m) and stayed underwater for as long as 7 minutes on other occasions. Some consider Giorgios to be the "father of freediving." However, it was not until Jacques Mayol of France dived to 331 ft (101 m) in 1976—which was then considered a stunning feat—that freediving really began to grow in popularity as an extreme sport. In 1988, the popular film *The Big Blue* portrayed the lives of Jacques Mayol and Italian Enzo Majorca as competitive freedivers and further popularized the sport.

Today, competitive freediving attracts athletes from all over the world and is regularly featured on sports television channels. The sport is governed by the Association Internationale pour le Developpement de l'Apnée—also known as the International Association for the Development of Apnea (AIDA) or the International Association for the Development of Freediving. AIDA has been the officiating body for freediving, setting standards and recognizing records since 1992. While AIDA recognizes eight types of freediving (described below), a number of "demonstration disciplines" or other, unofficial categories of freediving are practiced in lakes, at high altitude, and under ice, among other sites.

The freediver typically wears a facemask and a single two-foot monofin, or a pair of fins, and some sort of body suit for environmental protection.

Static apnea freediving is the simplest type of freediving and consists of immersed, resting breath-holding in a pool. The diver just holds his breath for as long as he can. Competitive times in this activity are generally longer than 5 minutes, with the world record for men of 9 minutes held by Tom Seitas of Germany, and for women of 7 minutes and 30 seconds held by Natalia Molchanova of Russia. There is considerable concern about continuing to push this record, in view of the likely harm from such prolonged periods of apnea.

Dynamic apnea freediving requires swimming horizontally underwater (usually in a pool) as far as possible on one breath, with and without fins. The world record for men using fins in a pool is 732 ft (223 m), and for women 656 ft (200 m). The

record swimming underwater in a pool without fins on a single breath for men is 600 ft (183 m) and for women 430 ft (131 m).

Constant ballast freediving (aka *constant weight freediving*) requires swimming as deep as one can without use of weights or lines and without use of any devices to aid ascent to the surface (e.g., balloons). This is also divided into categories of with and without fins. The latter is considered the premier freediving event because it requires a perfect coordination of equalization, propulsing movement, technique and buoyancy.

The current world records for constant weight freediving with fins are 282 ft (86 m) for women and 358 ft (109 m) for men. The records in the no fins category are 180 ft (55 m) for women and 262 ft (80 m) for men.

In *free immersion freediving* the freediver dives underwater by pulling on a rope during descent and ascent; no assistive propulsive equipment is used. The current world record for women is 246 ft (75 m) and for men 335 ft (102 m).

In *variable ballast freediving* (aka *variable weight freediving*) the freediver descends with the assistance of a weighted sled but must ascend on his own power (using fins or a pull rope). No lift balloons are allowed. The current world record for men is 459 ft (140 m), and for women is 400 ft (122 m).

"No limits" or freestyle freediving involves descent with as much weight as the diver wants, usually in the form of a weighted sled, and ascent using whatever assistive device the diver wishes (most often a lift bag or balloons). The AIDA-recognized men's world record of 564 ft (172 m) was set in October 2005 by Herbert Nitsch of Austria, and the women's record of 525 ft (160 m) is held by Tanya Streeter of the United States. Of note, on June 30, 2005, Patrick Musimu of Belgium made a well-documented dive to 688 feet (209.6 m) using a controversial technique of flooding his sinuses and eustachian tubes during descent. This technique allows further descent without needing to equalize ear pressure once the lungs are so compressed that they cannot provide any air for exhalation and further equalization. Since AIDA was not involved with this dive, they cannot officially recognize it, but there is no question that Musimu reached this remarkable depth.

Gamefishing is breath-hold diving that incorporates underwater hunting for food into the exercise. Breath-hold diving for fish, mollusks, seaweed, or other underwater "game" is the original form of breath-hold diving and is typified by the Ama of Japan and Hae-Nyu of Korea, the Greek sponge divers, and the pearl divers of the South Pacific.

Spearfishing is a popular type of gamefishing limited to hunting for free swimming fish, which are harvested by some type of spear. While diving to depths of over 100 feet (30.5 m) of seawater (fsw) is not uncommon in spearfishing, maximizing depth is not a primary motivation in this activity. Competitive spearfishing is governed by the International Bluewater Spearfishing Records Committee (IBSRC). The current IBSRC recognized world record has been held since 1998 by Greg Pickering of Australia, who speared a 307-lb (139 kg) yellowfin tuna off Socorro Island in Mexico.

Medical Problems of Breath-Hold Diving

The major medical concern of breath-hold diving is development of hypoxia leading to loss of consciousness and drowning, especially if submergence is preceded by hyperventilation (see Hyperventilation and Shallow Water Blackout). Breath-hold divers also may become hypothermic, get entangled in underwater debris (e.g., fishing line, ropes, and cables) or vegetation, and be harmed by marine animals or by being struck or run over by boats or other watercraft. Although very rare, breath-hold divers also can suffer from decompression sickness (see below under Decompression Sickness).

Scuba Diving

Scuba diving uses a tank fitted with a pressure regulator that supplies compressed air to the diver at a pressure equal to ambient water pressure. In recreational or sport diving, the diver's tank usually contains 90 cubic feet of filtered, oil-free compressed air pressurized to about 3000 pounds per square inch gauge (psig); tanks of 72 and 50 cubic feet are also used. Originally, tanks were made of steel, but most tanks in use today are made of aluminum.

The regulator employs two stages. The first stage is attached to the tank and makes an initial reduction in pressure to the lower-pressure second stage, which is attached to the diver's mouthpiece and from which the diver actually breathes.

Like the snorkeler, the scuba diver wears a face mask covering either just the eyes and nose (most often) or the entire face to allow underwater vision and equalization of pressure in the air space over the eyes, fins for propulsion, snorkel for breathing during surface swimming, weight belt, buoyancy-compensating vest to adjust buoyancy underwater and for flotation in case of an emergency, diving watch and depth gauge and/or a diving computer to track time and depth underwater, compass, and tank pressure gauge to monitor air consumption. Because of the higher thermal conductivity of water, divers typically wear neoprene wetsuits to stay warm (as well as to provide protection from jellyfish and other stinging marine life), even in the relatively warm tropical oceans. These suits maintain a layer of water warmed by body heat between the skin and suit. The suits are typically 7 mm in thickness when diving in temperate waters and 2 to 3 mm when diving in warm tropical waters. In tropical waters, some scuba divers wear a body suit made of spandex (Lycra) instead of a wetsuit to protect themselves from jellyfish, hydroids, and other marine stingers. An impermeable drysuit and warm undergarments are usually worn when diving in colder water (e.g., colder than 10° C [50° F]).

In addition to the preceding basic equipment, additional equipment may be needed for safety, navigation, communication, or other purposes (e.g., diving knife, camera, spear gun, or game bags). All of these objects increase the diver's resistance to movement underwater, decrease efficiency of movement, and increase the work of diving. Extra drag may become a life and death matter in the case of poorly conditioned individuals who have to respond to underwater exigencies.

Although the majority of recreational scuba diving is done using compressed air in an open-circuit mode, specially adapted scuba gear may be used for mixed-gas diving (see below) or for semiclosed or closed-circuit diving. Use of enriched air nitrox by sport divers has markedly increased in recent years.

Surface-Supplied or Tethered Diving

Surface-supplied or tethered diving includes several different diving technologies, all of which share the common characteristic of the diver's breathing gas (compressed air or mixed gas) being supplied to the diver by hoses from a surface source or from a diving bell at a pressure equal to ambient water pressure.

The best known form of surface-supplied diving is classic hardhat diving, which is sometimes called "mud diving" or

"blackwater diving" because it is often done in harbors having very muddy bottoms and dirty water with typically zero visibility. In this type of diving, which was portrayed in the 2000 movie *Men of Honor,* the diver wears a large bronze helmet with glass faceplates, a canvas suit, weight belt and weighted shoes, and other gear. Altogether, the traditional hardhat diver's gear weighs 192 lb (87.5 kg). While traditional hardhat gear is still used in a variety of settings, most surface-supplied diving uses more modern gear that is not as heavy or cumbersome as the traditional diver's dress and that has communication capabilities and the ability to keep the diver warm.

Surface-supplied diving is most often used in commercial or military settings. It is frequently performed in arduous circumstances. The diver operates in total darkness in cold water, often against a current or surge, performing tasks primarily by feel.

The diving techniques of surface-supplied diving are quite different than those of scuba diving, and they are not further discussed here; however, most of the physiologic and medical problems of surface-supplied divers are identical to those encountered in scuba diving.

Saturation Diving

Once underwater, the diver begins to absorb increased amounts of nitrogen or other inert gas, depending on the breathing medium, until a new equilibrium is established according to the pressure of the depth of submergence. In most deep-diving scenarios, the time needed to off-gas, or *decompress,* this inert gas on returning to normal atmospheric pressure may be much greater than the time spent at depth.

The need to minimize prolonged decompression after deep diving led to development of saturation diving. In the late 1950s, experiments by United States Navy (USN) diving medical officers George Bond and Robert Workman coincided with those of Jacques-Yves Cousteau and Edward Link in the commercial sector, all of whom were working on ways to stay underwater at great depths long enough to perform useful work.[3]

The basic concept of saturation diving is that after approximately 24 hours at any given depth, the diver's tissues establish equilibrium with the gases in the breathing mixture. From that point, the decompression obligation remains essentially the same no matter how long the diver remains at that depth. If the diver can be kept "at pressure" for a prolonged period, the decompression is the same as if he or she were there for only a short time.

Modern saturation complexes allow divers to live for days in large chambers at the pressure of a given work site and to be transported to the underwater site by locking into a personal transfer capsule (PTC) or sealed diving bell. When depth is reached, the water pressure equals the gas pressure within the capsule and divers may exit the PTC into the water while breathing gas supplied by umbilical hoses. To maintain the diver's thermal balance in the very cold water found at great depths, heated water is circulated in special hot-water suits.

Another application of saturation diving, used primarily for scientific purposes, utilizes underwater habitats, which are steel chambers situated at a given depth of water and pressurized with a compressed gas atmosphere at the same pressure as the surrounding water. Divers may live for days or weeks in the habitat, leaving the chamber with scuba equipment to perform studies or observe marine life in its natural state. In this specialized type of diving, rigorous precautions are taken to avoid inadvertent surfacing, thermal stress, and skin and ear infections during prolonged stays in the continuously moist environment of the habitat. Prolonged saturation decompression schedules, often taking several days, are required to return divers safely to sea level pressure on completion of the underwater mission.

One-Atmosphere Diving

In recent years it has become clear that humans cannot reliably or safely function at the greatly elevated pressure of deep water, and the human factor aspects of diving have become the limiting factor in manned exploration of the ocean depths. This has led to numerous developments in one-atmosphere diving systems. In the 15th century, Leonardo da Vinci drafted schematic drawings that look similar to modern one-atmosphere diving systems, but these systems had to await late 20th century advances in metallurgy, engineering, and communications before they could become functional.

One-atmosphere absolute (ATA) diving systems are, in essence, small submarines with various types of propulsion systems and manipulators that allow the operator to work at great depth. The interior of the unit is maintained with environmental control systems to retain safe physiologic parameters. These systems range from one-person ATA suits in which a diver can "walk" (Fig. 70-1) to submersibles that accommodate two or more occupants.

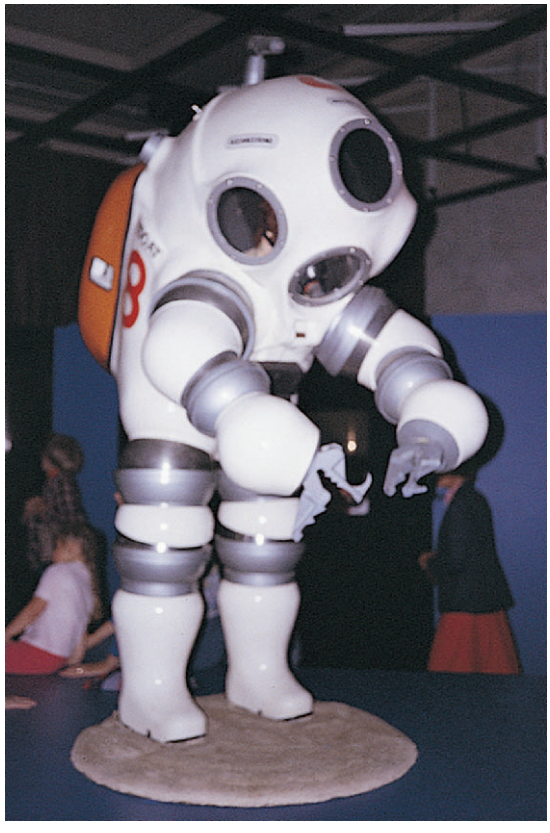


Figure 70-1. JIM diving suit. The diver remains at sea level pressure inside the suit and can work for prolonged periods of time at extreme depths underwater. (Courtesy Kenneth W. Kizer, MD.)

Mixed-Gas Diving

Diving can be done using either compressed air or mixed gas. Compressed air is most commonly used, especially with scuba, but there are a number of settings where mixed gas is needed or preferred.

Mixed-gas diving refers to diving using a breathing mixture other than compressed air (e.g., a gas mixture in which the concentrations of nitrogen and oxygen have been changed or in which a different inert gas [e.g., helium] is substituted for nitrogen). Mixed-gas diving can be used in surface-supplied, saturation, or scuba diving modes, although historically it has been used most often in surface-supplied or saturation diving operations.

Mixed-gas diving has been used for many decades, but because of the greater logistical support required and associated greater expense and hazards, it has been used primarily in commercial and military diving operations. This has changed in the past 15 years as "technical" sport divers have sought to go deeper and stay down longer. Increasing numbers of recreational divers are now using mixed gas, especially nitrox.

Enriched Air Nitrox

Nitrox is a breathing-gas mixture containing oxygen and nitrogen in concentrations different from those found in air. More than a dozen different such mixtures have been used by recreational divers, all of which are lumped under the heading nitrox, but the two most commonly used mixtures are the ones labeled by the U.S. National Oceanographic and Atmospheric Administration (NOAA) as nitrox I (a gas mixture containing 32% oxygen and 68% nitrogen) and nitrox II (a gas mixture containing 36% oxygen and 64% nitrogen). Each nitrox mixture, or blend, requires its own set of decompression tables and has its own bottom time limits. Only nitrox I is approved for recreational diving use, and subsequent reference to enriched air nitrox in this chapter is to this mixture.

Of note, the term *nitrox* historically was used to refer to gas mixtures having less than 21% oxygen. These mixtures were used in diving habitats or other saturation diving situations in which one wanted to avoid, or at least lesson, the risk of oxygen toxicity. Technically, if the oxygen percentage is adjusted to greater than 21%, the mixture is called *enriched air nitrox* (EAN), or *oxygen enriched air* (OEA), although *EAN* and *nitrox* are used interchangeably in common diver's parlance.

Beginning in the 1980s, an increasing number of recreational divers began using EAN to extend bottom time (compared with what was possible using compressed air) and reduce the risk of decompression sickness. Many thousands of recreational EAN divers have been certified in recent years, and EAN has even become the norm on many live-aboard charter dive boats.

EAN diving enthusiasts typically claim that nitrox is safer than compressed air because it has less risk of decompression sickness for equivalent bottom times. At relatively shallow depths, EAN allows considerably increased bottom time before decompression is required when compared with compressed air. For instance, at 60 fsw (18 m), EAN allows an extra 45 minutes (60 minutes on air, 105 minutes on EAN). However, this advantage diminishes greatly at depths over 100 fsw. At 100 fsw EAN provides for only 8 extra minutes.

Although diving with nitrox may lessen the risk of decompression sickness compared with diving with compressed air, it definitely does not eliminate the risk, and EAN has risks of its own. The main concern is with central nervous system (CNS) oxygen toxicity, which usually manifests suddenly (with few, if any, prodromal symptoms) by loss of consciousness and onset of convulsions. Because of the risk of CNS oxygen toxicity, EAN should not be used below 130 fsw.

A diver can still suffer decompression sickness diving with EAN if he or she stays down too long, surfaces too fast, bypasses a required decompression stop, or uses the wrong nitrox decompression table for the particular breathing medium (if not breathing nitrox I). Further, although nitrox may increase a diver's allowable no-decompression bottom time, this is often irrelevant because a diver's bottom time is as much a function of gas supply as the decompression limit. That is, most scuba divers will exhaust their gas supply before reaching the no-decompression limit.

Overall, use of nitrox for recreational diving purposes is still evolving. Clearly, it has advantages and disadvantages (Box 70-2), with the advantages most likely realized in a setting that ensures adherence to safety.

Heliox

Other than nitrox, the most commonly used mixed gas is heliox, or oxy-helium, a mixture of helium and oxygen. Helium is used as the inert gas, replacing nitrogen (and, therefore, eliminating the risk of nitrogen narcosis). With heliox, the oxygen level is reduced to prevent oxygen toxicity. As with nitrox, *heliox* is a generic term that applies to a number of different blends or mixtures of helium and oxygen.

Because it causes negligible, if any, narcosis and is easier to breathe at greater depths (because of lesser density), heliox is the preferred gas used in commercial diving at depths over

Box 70-2. Advantages and Disadvantages of Enriched Oxygen Nitrox Diving Compared with Compressed Air at Depths between 50 and 130 fsw (15 and 40 m)

ADVANTAGES

- Decreased risk of decompression sickness
- Decreased occurrence of nitrogen narcosis
- Reduced residual nitrogen time
- Shorter surface interval times
- Reduced decompression times if maximum bottom time limits are exceeded
- Reduced surface intervals between diving and flying

DISADVANTAGES

- Requires special training
- Requires equipment dedicated for use with nitrox only
- Increased oxidation of scuba cylinders
- Possible increased rate of deterioration of equipment
- Increased fire hazards
- Inability to use air-based dive computers
- Potential for nitrox mixing and filling problems
- Risk of CNS oxygen toxicity

130 fsw. The major problems with helium are its expense, which precludes its widespread use in recreational diving, greater thermal conductivity, and hindrance of speech. In commercial and military settings, helium speech unscramblers are typically used. However, at depths over 600 fsw, and especially with rapid descent, helium causes a poorly understood condition known as the high pressure nervous syndrome (HPNS). First noted in the 1960s, HPNS is characterized by dizziness, nausea, vomiting, postural and intention tremors, fatigue and somnolence, myoclonic jerking, stomach cramps, numbness, and sleep disturbances.[14] HPNS is a major barrier to manned undersea activity deeper than 600 fsw.

Trimix

Trimix is a generic term referring to mixtures of helium, nitrogen, and oxygen. This breathing medium was pioneered by military and commercial diving interests for operations at depths greater than those possible by diving with compressed air. Helium replaces some of both the nitrogen and oxygen in an effort to eliminate or minimize nitrogen narcosis and to prevent CNS oxygen toxicity. The precise concentrations of helium, nitrogen, and oxygen used in trimix vary according to the specific depth profile of the dive. Obviously, in deep-diving operations, the percentages of both nitrogen and oxygen will be much less than those present in air, which means that a "travel" gas mixture is needed for breathing in shallower depths that must be traversed to get to the depth at which the trimix will be used. Currently, the USN specifies the use of trimix for diving at depths greater than 190 fsw, and trimix is typically used in extreme-depth (greater than 600 fsw) commercial diving because the addition of small amounts of nitrogen partially mitigates the occurrence of HPNS.

A spin-off of tri-mix that has begun to be used in recreational diving is oxygen-enriched tri-mix, or *helitrox*. A tri-mix blend commonly used in this setting is 26% oxygen, 17% helium, and 57% nitrogen (TX 26/17). Helitrox advocates promote its use for diving to depths of up to 150 fsw, either using helitrox to decompress or switching to pure oxygen at the 20-fsw decompression stop.

A new world record for open-circuit scuba diving using trimix was set by the South African Nuno Gomez in June 2005 when he dived to 1044 fsw (318 m) in the Red Sea. It took 20 minutes to descend to depth, but he spent over 12 hours decompressing on ascent. A total of 9 different gas mixes were used for the dive (4 trimix, 3 nitrox, 1 air, and 1 oxygen). This record was not held long, however, because in July 2005, Pascal Bernabe of France dived to 1100 ft (330 m) off Corsica. His descent time was only 10 minutes, requiring decompression of 9 hours.

A high incidence of fatalities among sport divers using trimix has limited its use in recreational diving, but interest in its use remains high, and the recent emergence of tri-mix dive computers for rebreathers portends much greater use of trimix in the future.

Rebreather Diving

Although the first self-contained underwater diving is usually associated with scuba, it was actually first done with the assistance of a rebreather in the 1880s. Rebreathing devices for diving were perfected over the years, but with the advent of scuba in the 1940s, rebreathers were relegated to use in only commercial and military situations. Beginning in the 1990s, however, use of rebreathers started to increase in recreational diving (especially for underwater photography and cave diving).

Conceptually, rebreathers are devices that capture and recirculate a diver's exhaled breath, removing the carbon dioxide added by the body's metabolism and replacing the oxygen extracted by the body before giving the air back to the diver to once again breathe. As a diving technology, rebreathers are much more efficient than open-circuit scuba, and they allow one to dive to greater depths and stay underwater longer than is possible with conventional open-circuit scuba. They are also very useful when exhaled bubbles may be problematic (e.g., when studying or photographing marine life or when disarming pressure-sensitive mines). However, they can be quite hazardous if not maintained and operated properly.

All rebreathers have certain elements in common, and fall into one of four general categories of devices[7]:

1. Closed-circuit oxygen rebreather
2. Closed-circuit mixed-gas rebreather
3. Semiclosed-circuit rebreather using premixed gas
4. Semiclosed-circuit rebreather that mixes the breathing gas while diving

The advantages and disadvantages of the different types of rebreathers are beyond the scope of this discussion. There are many different rebreather designs available, and new systems continue to be developed. Therefore, a diver must be individually trained on the use of each rebreather.

Technical Diving

Beginning in the late 1980s, a small but rapidly growing number of recreational divers began to use mixed-gas, rebreathers, and other technology previously used by military and commercial divers to dive deeper and stay down longer than possible with conventional scuba. "Technical diving," as it was dubbed in 1990 by *AquaCorps* (a diving periodical devoted to this then new endeavor), has gained a large following since then. It is employed by wreck and cave divers.

Technical diving represents the leading edge of underwater discovery and challenge for the nonprofessional diver. It is at the forefront of dive technology and technique and is inherently more hazardous than typical sport scuba diving because of the intrinsically greater hazard and diminished margin of error allowed when diving deep and being so reliant on technology. For example, rebreathers using trimix have been used for very deep diving, and about half a dozen people have dived deeper than 800 fsw using this technology. Unfortunately, most of these divers are now dead secondary to diving mishaps.

Despite its hazards, technical diving has markedly increased in popularity in recent years, as witnessed by the establishment of the International Association of Nitrox and Technical Divers (IANTD) and other technical diving associations and by the convening of "tek" conferences drawing thousands of divers. This movement has been supported by former navy diving suppliers looking for new buyers and commercial diving vendors trying to cross over to the much larger recreational diver market. Technical divers tend to be well-educated professionals with substantial disposable incomes, which allows for the purchase of the required technology.

SPECIAL DIVING SETTINGS

Although most people usually think of diving as something that is done in warm, clear tropical oceans on beautiful coral reefs

teeming with colorful fish, diving has become a tool that allows humans to engage in a wide array of underwater activities (see Box 70-1). Scuba and surface supplied diving is undertaken for numerous special purposes in settings that often pose unique hazards. Examples of these special diving settings are shipwrecks, caves, rivers, hydroelectric power–generating dams, nuclear power plants, harbors, mines, water treatment facilities, and under ice. These settings generally require special diving techniques, health monitoring, and specialized safety precautions. Some of these settings are briefly described below.

Wreck Diving

Wreck diving—exploring sunken ships, airplanes, automobiles, and other man-made objects—is one of sport diving's most popular activities. Wrecks often provide a window on the past, capturing and preserving a single moment in history. The mixture of history and drama that surrounds many wrecks captures the imagination and sense of adventure of many divers, making wreck diving an exhilarating pastime. In addition, shipwrecks function as artificial reefs, attracting great quantities of aquatic life and so are sought by underwater photographers and fish watchers.

Wreck diving is generally classified as either recreational or technical, with each being further classified as either external survey or penetration.

Recreational wreck diving is defined as any dive on a man-made object to a maximum depth of 130 fsw and staying within no-decompression diving limits. Most recreational wreck dives involve external surveys only, without penetration into the interior of the vessel beyond the natural light zone. *Technical wreck diving* involves diving deeper than 130 fsw, typically using mixed gas and often requiring staged decompression.

The two main problems with wrecks are their depth (many are deeper than can be safely dived with compressed air scuba) and loss of direct access to the surface when the diver penetrates the interior of the submerged vessel. In addition, the hulls of shipwrecks are usually very rusty, and the interior is filled with silt, drastically reducing visibility when stirred up and possibly causing the diver to become disoriented and lost, resulting in the diver running out of air and drowning. As a result, specialized techniques are required for diving inside wrecks or down to deep wrecks.

In recent years, the lure of exploring shipwrecks has been a major stimulus for technical diving, and mixed gas and rebreathers have been increasingly used. In May 2005, a new deep wreck open-circuit scuba diving world record of 633 fsw (193 m) was set by Rob Lalumiere when diving to place a memorial plaque on the *USS Cooper,* an American destroyer sunk with 191 men on board in 1944 by a Japanese torpedo in the Battle of Ormoc Bay, Philippines.

Cave and Cavern Diving

Underwater caves and caverns are frontiers within the largely unexplored underwater wilderness. They are perhaps the only places left on Earth that have not been walked on, sonar sounded, or satellite photographed. The advent of scuba has allowed exploration of many underwater caverns and cave systems, leading to important archeologic and geologic discoveries. However, cave diving is fraught with hazard and associated with hundreds of diving deaths. Cave diving is the most dangerous form of diving.[82] The IANTD characterizes cave diving as "ultrahazardous."

To trained, experienced, and properly equipped cave divers, the number of cave-diving fatalities (over 300 in Florida alone since 1960) is distressing because so many were preventable. Most fatalities have involved recreational scuba divers who were not trained in cave diving and ran out of air after becoming lost in a cave. Many of these fatal incidents involved highly experienced open-water divers.

Because cave divers are separated from the outside world and cannot simply surface in case of an emergency, special equipment and procedures are required. These include double tanks, meticulous planning of air consumption, backup lights, and guide cables to avoid becoming lost. No one should attempt cave diving without completing formal training approved by either the National Association of Cave Divers or the National Speleological Society—Cave Diving Section.

In recent years, use of rebreathers and mixed gas in cave diving has been increasing because of the reduced potential to stir up silt (which reduces visibility and increases the chance of becoming lost) and because of the potential to achieve greater depths and bottom times. In January 2005, a rebreather and trimix were used to attempt the deepest ever body recovery from Boesmansgat (aka Bushman's Hole), a sinkhole in South Africa. This attempted recovery of the body from a depth of 927 feet of fresh water (ffw; 296 m) ended in disaster when the recovery diver died (probably of hypercarbia) and one of the support divers suffered serious decompression sickness. Ironically, the body was of a diver who had died in this cavern 10 years earlier. Both bodies were recovered when they floated to shallow water on their own as the dive operation was concluding.

Under-Ice and Cold-Water Diving

Diving under the ice in extremely cold water is done for exploration, search and rescue, salvage, cinematography, inspections, research, and other work. This most often takes place in lakes, rivers, and the polar oceans. Besides meticulous preparation and provision of thermal protection for the diver, careful planning for the diver's topside support personnel is also necessary. Extremely low temperatures, wind, and snow above water at the dive site are among the typical hazards that must be addressed. For diving in such cold water, drysuits or hot-water suits are used.

Drysuits are made of closed-cell foam neoprene, rubber, or rubberized canvas and fit loosely on the body, with seals at the wrists, ankles, and neck to keep the diver and insulating underwear dry and to allow egress of air to maintain equal suit volume during the dive. Free-flooding hot-water suits are loosely fitted, closed-cell foam neoprene suits with hoses inside to allow warm water to flow continuously over the diver's body and exit at the wrist and neck.[177]

In this specialized type of diving, other considerations are use of breathing regulators that are not likely to freeze, a plan for immediate rescue and rapid rewarming of anyone who might fall into the frigid water without thermal protection, maintenance of adequate nutrition and hydration, and absolute prohibition of alcoholic beverages or mind-altering drugs at the dive operation, because cold water diving permits little margin for error.

River Diving

Diving in rivers is often done by public safety personnel to search for evidence or to retrieve bodies (usually drowning victims or murder victims that have been dumped into the river).

In addition, some individuals have made a career out of recovering automobiles, lost possessions, and other materials from rivers. Because of the typically poor visibility, strong currents, and high potential for entanglement in vegetation or debris, this type of diving should be done only by persons who have been trained in the special techniques of river diving and who have proper surface support.

Public Safety Diving

In the last 20 years there has been a phenomenal growth and change in public safety or emergency service diving. Most law enforcement bodies and fire departments having any notable body of water in their jurisdiction either have an emergency service dive team themselves or have a mutual aid agreement with another agency for such services. Government agencies such as the Federal Bureau of Investigation (FBI) and the Bureau of Alcohol, Tobacco, Firearms and Explosives (ATF) have highly trained special diving units, such as the FBI's New York Office Scuba Team and the ATF's Underwater Explosives Recovery Team.

Although public safety diving originally focused mostly on rescuing or recovering drowning victims and used basic scuba equipment, it rapidly evolved to conduct highly complicated and dangerous underwater evidence searches and recovery missions, such as the search and recovery of TWA Flight 800 in Long Island Sound, New York, in 1996; to disarm explosives; and to carry out preventive surveillance to prevent terrorist attacks at special events, including the 1996 Olympics in Atlanta. As a result, most public safety divers today require special equipment and training.

Public safety diving is often done in very hostile and unpredictable circumstances and under extreme time pressures, all of which combine to make it a highly hazardous activity. Diving is often done in stagnant bodies of water having very muddy and silty bottoms, producing "blackwater" in which the diver can barely see his hand on his facemask; in rivers or bays where currents can be treacherous; or in waters polluted with toxic chemicals and fecal or other organic material. Entanglement can occur in vegetation, floating deck lines, and buoy lines or cables. Myriad types of debris, which are often razor sharp, are typical hazards. As a result, most public safety divers wear vulcanized rubber drysuits connected to full face masks or helmets that cover the entire head and neck and that provide a water tight seal.

The medical hazards of public safety diving vary depending on the water and other environmental conditions and the mission's objective and urgency. Drowning and near-drowning from entanglement is an ever-present hazard, as are mechanical trauma, dysbaric conditions, infectious diseases, toxic exposures, and psychological stress. The hazards of this type of diving have been underscored by a number of fatal training dive incidents in recent years.

Miscellaneous Other Settings

Commercial golf ball diving is part of the $200 million annual golf ball retrieval and recovery industry. Golf ball divers usually contract with golf courses to periodically recover balls from ponds and water hazards, and sometimes from adjacent lakes and rivers. In 2004, it was reported that a hard-working golf ball diver could recover 3,000 to 6,000 balls on an average day (some divers have reported recovering as many as 21,000 balls in a day), earning more than $100 an hour and between $50,000 to $100,000 a year. The diving is typically less than 10 ft (3 m) deep, but the water is extremely silty and often highly contaminated with fertilizer and pesticides. There is usually zero visibility, and all work is done by feel.

Hazards of golf ball diving include bites by venomous snakes, alligators, snapping turtles, and blue crabs; leech infestations; water-borne infectious diseases; toxic (fertilizer and pesticides) dermatitis; penetrating trauma from broken glass and barbed wire; drowning from entanglement in weeds or fishing line; and attack by golf ball poachers. In 2001, two golf ball divers drowned while working.

Underwater logging involves using scuba or surface-supplied diving gear to retrieve submerged logs from rivers and lakes or harvesting dead trees from land submerged by dams.[31] Lack of oxygen, reduced light, and cold temperatures typically found underwater protect the submerged trees from decay.

In recent years, considerable attention has focused on harvesting trees submerged by dams in Canada (British Columbia and New Brunswick), Malaysia, Russia, and North and South America. In the U.S., most of the interest has been in Washington, Oregon, Lake Superior, and the Southeast. There are approximately 45,000 dams worldwide, and about half of them contain harvestable timber. This equates to about 200 million trees worth an estimated $40 billion.

A good example of underwater logging is occurring in Tucurui Lake in northern Brazil. When the Tucurui Dam, the fifth largest dam in the world, was built in 1985, it submerged 1,110 square miles (1786 km) of tropical forest. There are believed to be some 1.5 million submerged tropical hardwood trees (mahogany, Brazil nut, angelim, jatoba and massaranduba) available for harvesting here at an estimated value of $100 million. A typical hardworking underwater logger can harvest 8 to 10 trees a day, frequently working at depths of 80 to 150 ffw (24.5–46 m).

Divers who harvest trees from Kenyir Lake, the result of another hydroelectric dam built in the 1980s, in Indonesia are reported to regularly work as deep as 300 ffw (91 m), which explains the relatively frequent occurrence of decompression sickness among these workers.

Reported hazards of underwater logging include hypothermia, drowning from entanglement in tree branches or other vegetation, mechanical trauma from the hydraulic tools that are used, and decompression sickness. Of note, when a tree is cut underwater, it generally "falls" up toward the surface. An increasing number of the larger logging companies involved with underwater logging are turning to remote-controlled underwater submarines equipped with hydraulic saws, instead of human divers, to harvest the trees.

Sewer diving is another very special form of diving. Large cities generally employ divers or contract with commercial diving companies to keep wastewater pipes and tunnels free of sewage solids buildups, and debris (bottles, plastic containers, waste wood or vegetation, clothes, and dead animals) and to repair leaks in the pipes or outfalls. Hundreds of miles of sewer pipes and tunnels underlie most large cities.

Sewer divers—also known as "deep slime divers"—are tethered and breathe surface-supplied air while wearing airtight heavy rubber wetsuits and leather over-suits, gloves, and sealed helmets to protect themselves from sharp objects and the fetid water. The gear is similar to that worn by divers who service the water intakes at nuclear power plants. Visibility in sewer pipes and tunnels ranges from normally zero to a few feet,

Box 70-3. Medical Problems of Scuba Divers

- Environmental exposure problems
  - Motion sickness
  - Near drowning
  - Hypothermia
  - Heat illness
  - Sunburn
  - Phototoxic and photoallergic reactions
  - Irritant and other dermatitides
  - Infectious diseases
  - Mechanical trauma
- Dysbarism
  - Barotrauma
  - Dysbaric air embolism
  - Decompression sickness
  - Dysbaric osteonecrosis
  - Dysbaric retinopathy
  - Hyperbaric cephalgia
- Breathing gas-related problems
  - Inert gas narcosis
  - Hypoxia
  - Oxygen toxicity
  - Hypercapnia
  - Carbon monoxide poisoning
  - Lipoid pneumonitis
- Hazardous marine life
- Miscellaneous
  - Hyperventilation
  - Hearing loss
  - Carotid-related blackout
  - Panic and other psychological problems

depending on the amount of run-off and other waste water diluting the solid material. Work is done by feel.

Hazards of sewage diving include penetrating trauma from discarded hypodermic needles, broken glass, wire and other sharp objects; hepatitis and other infectious diseases; hypothermia; entanglement in debris; blunt trauma from blow-ups (uncontrolled over-inflation of the diving suit); and being swept away and drowned by unexpected rushes of wastewater. The divers who maintain the sewers of Mexico City regularly report encountering corpses in the sewers. Reportedly, the sewers are a favorite place to dispose of murder victims in this city, and the bodies found by the sewage divers are rarely identified or even reported to the police because of the zero visibility conditions and the wastewater currents that quickly carry the bodies away.

After each dive, the sewer diver is repeatedly hosed down and cleaned with a bleach-based solution in a multistep decontamination process.

DYSBARISM

Divers encounter many adverse environmental conditions underwater. These include cold, changes in light transmission and sound conduction, lack of air to breathe, increased density of the surrounding environment, and increased atmospheric pressure. Not surprisingly, diverse medical problems are related to diving (Box 70-3).

Of the various environmental factors affecting divers, pressure is by far the most important because it contributes either directly or indirectly to the majority of serious diving-related medical problems. Therefore, knowing the basic physics and physiologic effects of pressure is essential to understanding and treating the pressure-related maladies that fall under the general term dysbarism.

Definitions and Terminology

Dysbarism is a term that encompasses all the pathologic conditions caused by altered environmental pressure. These disorders primarily affect divers and persons who work in compressed-air environments who are also exposed to increased atmospheric pressure. However, some of the conditions may also occur in aviators, astronauts, and certain types of industrial workers as a result of abrupt exposure to the reduced pressure found at actual or simulated high altitude.

Dysbarism most often develops acutely because of problems caused by the mechanical effects of pressure on closed air spaces (barotrauma) or problems, such as nitrogen narcosis or decompression sickness, caused by breathing gases at elevated partial pressure. Less often, clinical effects are delayed for months or years, as in the case of dysbaric osteonecrosis. The pathophysiology of these delayed conditions is not well understood.

Pressure is defined as force per unit area. Atmospheric pressure is the pressure exerted by the air above the earth's surface. Atmospheric pressure varies with altitude. At sea level, atmospheric pressure is 760 millimeters of mercury (mm Hg) or 14.7 pounds per square inch (PSI). The barometric pressure at sea level is generally referred to as 1 atmosphere (atm). Absolute pressure is the total barometric pressure at any point. With pressure gauges calibrated to read zero at sea level, gauge pressure is the amount of pressure greater than atmospheric pressure. In general, gauge pressure is 1 atm less than absolute atmospheric pressure. It is necessary to specify whether pressure is expressed in terms of gauge or absolute pressure. Except in situations requiring laboratory precision, the following units are commonly used to express water pressure:

- Feet of seawater (fsw)
- Feet of fresh water (ffw)
- Meters of seawater (msw)
- Atmospheres absolute (ATA)
- Pounds per square inch gauge (psig)
- Pounds per square inch absolute (psia)

As a diver descends under water, absolute pressure increases much faster than in air. Each foot (.3048 m) of seawater exerts a force of 0.445 psig. Therefore, if the 14.7 psi pressure of 1 atm is divided by 0.445 psi per foot of seawater, the absolute pressure will have doubled at 33 fsw. In the ocean, each 33 ft of depth adds one additional atmosphere of pressure. The gauge pressure at 33 fsw is 14.7 psig (in excess of atmospheric pressure), and the absolute pressure is 29.4 psia.

Because of the weight of solutes in seawater, it is slightly heavier than fresh water. In fresh water, 34 ft (10.4 m) equals one additional atmosphere of pressure.

Pressure change with increasing depth is linear, although the greatest relative change in pressure per unit of depth change occurs nearest the surface. Table 70-1 lists commonly used units of pressure measurement in seawater.

When a diver submerges, the force of the tremendous weight of the water above is exerted over the entire body. Except for air-containing spaces such as the lungs, paranasal sinuses, intes-

TABLE 70-1. Commonly Used Units of Pressure in the Underwater Environment

Depth (fsw)	DEPTH (m)	psig	psia	ATA	mm Hg (ABSOLUTE)
Sea level		0.0	14.7	1	760
33	10	14.7	29.4	2	1520
66	20	29.4	44.1	3	2280
99	30	44.1	58.8	4	3040
132	40	58.8	73.5	5	3800
165	50	73.5	88.2	6	4560
198	60	88.2	102.9	7	5320
231	70	102.9	117.6	8	6080
264	81	117.6	132.3	9	6840
297	91	132.2	147.0	10	7600

ATA, atmospheres absolute; fsw, feet of seawater; psia, pounds per square inch absolute; psig, pounds per square inch gauge.

tines, and middle ears, the body behaves as a liquid. The law that describes the behavior of pressure in liquids is named for the 17th century scientist Blaise Pascal. Pascal's law states that a pressure applied to any part of a fluid is transmitted equally throughout the fluid. Thus when a diver reaches 33 fsw, the pressure on the surface of the skin and throughout the body tissues is 29.4 psia or 1520 mm Hg (Fig. 70-2). The diver's body is generally unaware of this pressure, except in the air-containing spaces of the body. The gases in these spaces obey Boyle's law (Fig. 70-3), which states that the pressure of a given quantity of gas for which temperature remains unchanged varies inversely with its volume. Thus, air in the middle ear, paranasal sinuses, lungs, and gastrointestinal tract is reduced in volume during compression or descent underwater. Inability to maintain gas pressure in these body spaces equal to the surrounding water pressure leads to various untoward mechanical effects, which are discussed later.

Because of the weight of the water exerting pressure over the chest wall, humans can breathe surface air through a snorkel or tube connected to the surface for only a short distance underwater, typically only to a depth of 1 to 2 ft. Attempts to breathe at greater depths through the tube are not only impossible but are dangerous, because the respiratory effort greatly augments the already physiologic negative-pressure breathing. In other words, when the respiratory muscles are relaxed at sea level, alveolar pressure is equal to surrounding air pressure. At a depth of 1 ft, the total water pressure on the chest wall is nearly 200 lb (91 kg). Because of the loss of normal chest expansion and the pressurization of intra-alveolar air, the diver has to use forceful negative-pressure breathing to draw surface air into the lungs through the tube. Even at a depth of 1 ft, the great respiratory effort required is rapidly fatiguing, and respiration becomes impossible at further depths of only a few inches. Forced negative-pressure breathing can ultimately result in pulmonary capillary damage, with intra-alveolar edema or hemorrhage. Symptoms include dyspnea and hemoptysis. Should this occur, there is no specific treatment; therapy is purely supportive.

BAROTRAUMA

Gas pressure in the various air-filled spaces of the body is normally in equilibrium with the environment. However, if anything obstructs the passageways of gas exchange for these spaces and a change in ambient pressure occurs, pressure disequilibrium develops. The tissue damage resulting from such pressure imbalance is known as barotrauma.

Overall, barotrauma is the most common medical problem in scuba diving, potentially involving any structure or combination of structures that leads to entrapment of gas in a closed space. This includes the ears, paranasal sinuses, lungs, gastrointestinal tract, the portion of the face under a facemask, and skin trapped under a fold in a drysuit.

Barotrauma of Descent

Mask Barotrauma

For humans to see underwater, an air space must be present between the eye and water. In scuba diving, this is created by use of a facemask consisting of tempered safety glass in a soft malleable mask that seals across the forehead, on the sides of the face, and under the nose to allow nasal exhalations into the mask space to maintain air pressure inside the mask. As a diver descends in the water, the ambient pressure increases and air must be added to the gas inside the facemask to equalize water pressure. If inexperience or inattention cause the diver to forget to maintain this balance, negative pressure in the mask can rupture capillaries, causing skin ecchymosis, subconjunctival hemorrhage, lid edema, and rarely hyphema. This unusual condition is known as *mask squeeze* (Fig. 70-4; see also Figure 25-19).

Most divers with mask squeeze are asymptomatic, albeit sometimes horrified by their appearance. The condition usually resolves over a few days to a week without any intervention, but can be treated with cold compresses and analgesics if needed.

Facemask barotrauma is easily prevented just by exhaling through the nose during descent. It may also be prevented by wearing a full-face facemask.

In recent years, full-face facemasks, which are standard issue for commercial, military, and public safety divers, have become more popular with recreational divers as more models have become available, but they are still not commonly used. A full discussion of the advantages and disadvantages of full-face facemasks is beyond the scope of this section, but they should be remembered as a potentially good alternative for persons who have trouble with facemask fit, facemask flooding, or feelings of claustrophobia.

Orbital hemorrhage from facemask barotrauma is an unusual complication and can be associated with diplopia, proptosis, and visual loss.[29] Although such neurologic findings after scuba diving may suggest arterial gas embolism or neurologic decompression sickness, the presence of the unmistakable stigmata of mask squeeze and a consistent history should prompt consideration of orbital hemorrhage. Under these circumstances, instead of immediate referral to a recompression facility, the diver should be referred for immediate magnetic resonance imaging (MRI) and ophthalmology consultation because of the possibility of permanent vision loss due to elevated intraocular pressure. Recompression is contraindicated for orbital hemorrhage unless the diver also suffers from arterial gas embolism or decompression sickness.

Sinus Barotrauma

The four paired paranasal sinuses—frontal, maxillary, ethmoid, and sphenoid—have narrow connections to the nasal cavity via

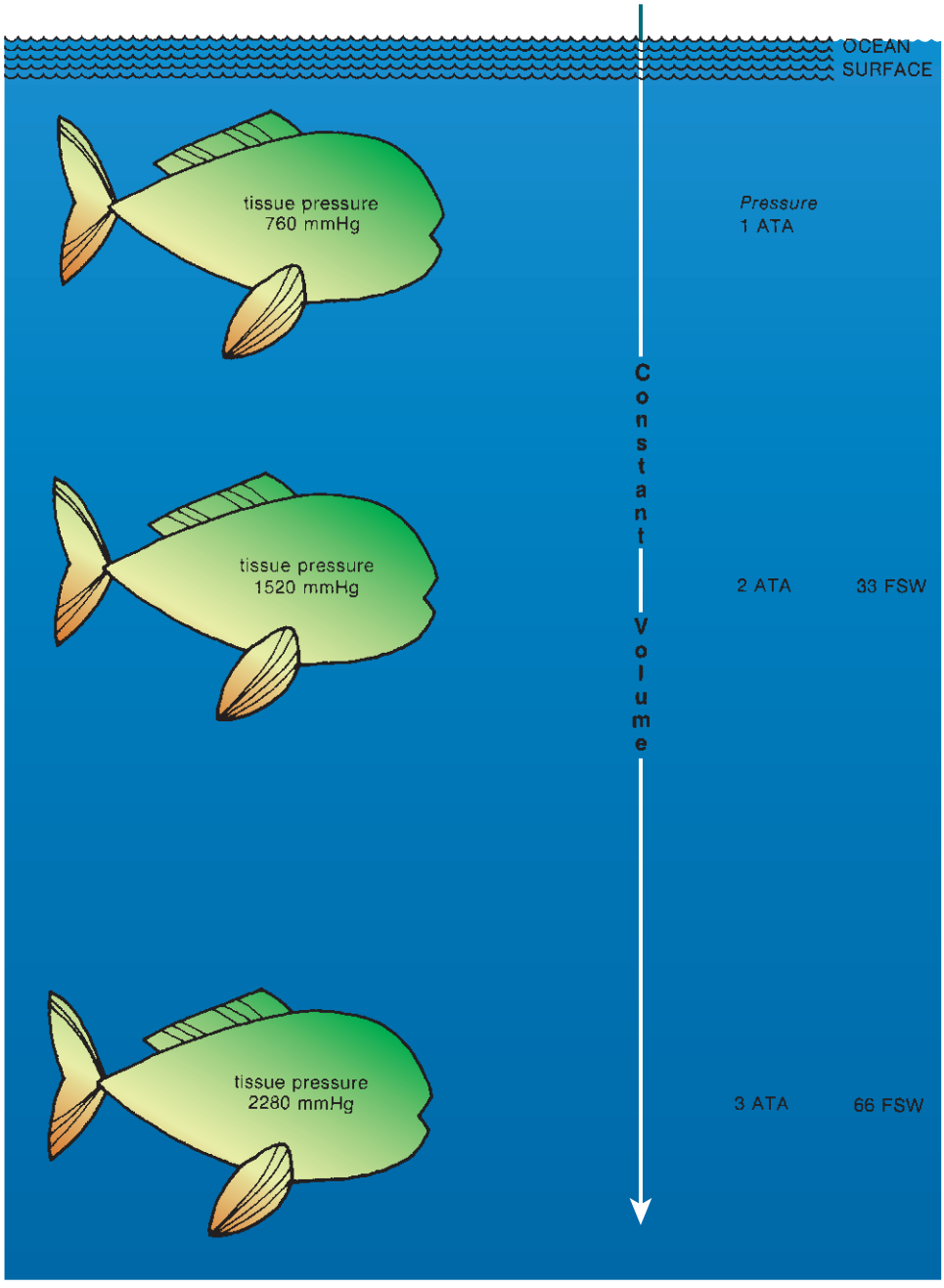


Figure 70-2. Pascal's law. Pressure applied to any part of a fluid is transmitted equally throughout the fluid.

the sinus ostia. If there is inability to maintain the air pressure in any paranasal sinus during descent, a relative vacuum develops in the sinus cavity. This negative pressure causes congestion of the mucosal lining with subsequent edema and intramucosal bleeding and possible formation of hematoma, hemorrhagic bullae, and bleeding into the sinus (Fig. 70-5). In cases of sinus squeeze, the diver usually experiences increasingly severe pain over the affected sinus during descent. On ascent, the remaining gas in the sinus expands and may force mucus and blood into the nose and mask.

The frontal sinus is most commonly affected by barotrauma, followed by the maxillary sinus. With maxillary sinus involvement, the diver often experiences pain in the maxillary teeth due to compression of the posterior superior branch of the 5th cranial nerve, which runs along the base of the maxillary sinus. Additionally, maxillary sinus barotrauma can cause compression or ischemic neuropraxia of the infraorbital branch of the 5th cranial nerve causing tingling and numbness of the cheek and upper lip.[28,136] Other complications include sinusitis from infection of the intrasinus fluid, or periorbital emphysema from

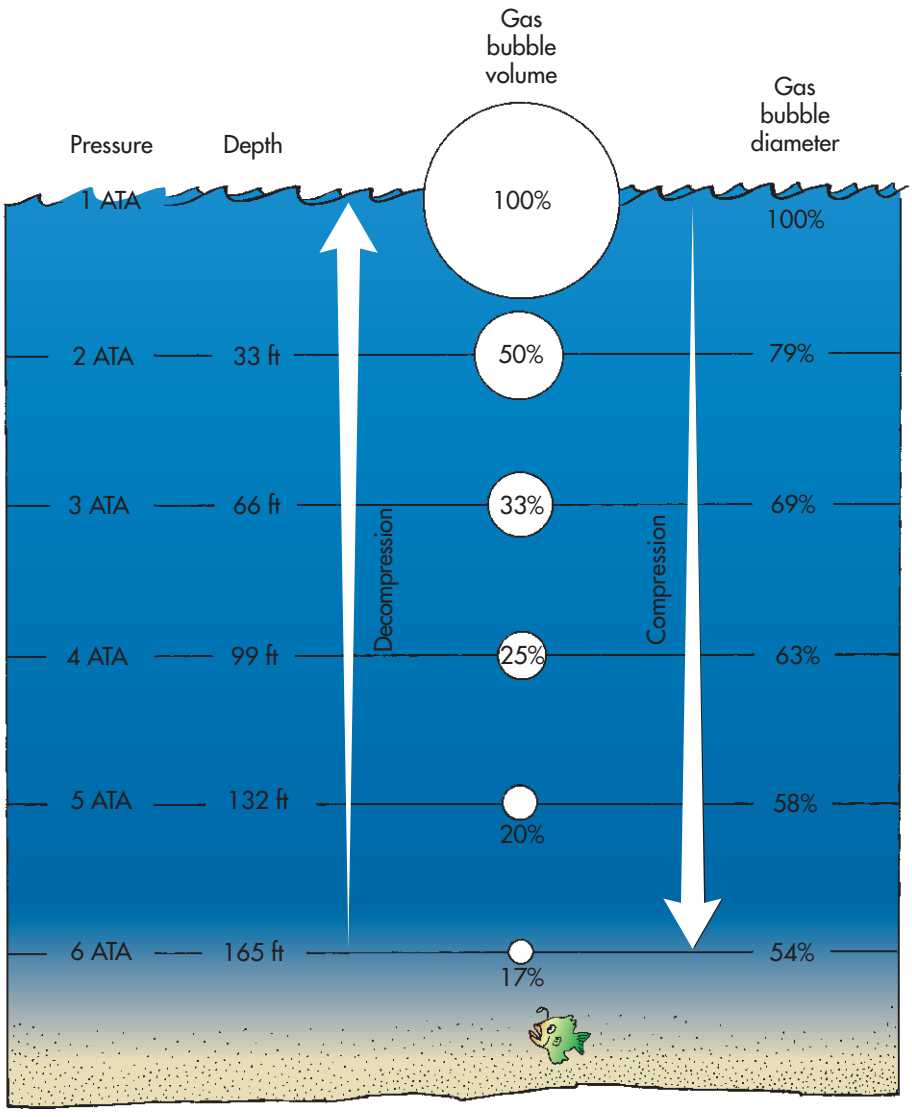


Figure 70-3. Boyle's law. The volume of a given quantity of gas at constant temperature varies inversely with pressure: $P_1V_1 = P_2V_2$.

Figure 70-4. Mask barotrauma. A novice diver with mask barotrauma showing subconjunctival hemorrhages.

air dissecting through the lamina papyracea from the ethmoid sinus into the orbits.

Treatment of sinus barotrauma involves use of systemic (e.g., pseudoephedrine) and topical (e.g., phenylephrine or oxymetazoline) vasoconstrictors, analgesics, abstinence from diving until resolved, and antihistamines if needed. Antibiotics are indicated only if signs of sinusitis are present, including fever or purulent nasal drainage. In the field, a 3 to 5 day course of corticosteroids has been sometimes used to hasten recovery and allow an otherwise healthy diver to return to diving. On rare occasions, drainage of the affected sinus by an otolaryngologist is required for persistent pain.

Sinus barotrauma usually occurs in the setting of a diver who has an upper respiratory infection or severe allergies or who has an anatomic deformity such as nasal polyps or deviated septum. Consequently, prevention of sinus barotrauma includes avoid-

Figure 70-5. Frontal sinus barotrauma 3 days earlier. Note the persistent air-fluid level. (Courtesy Kenneth W. Kizer, MD.)

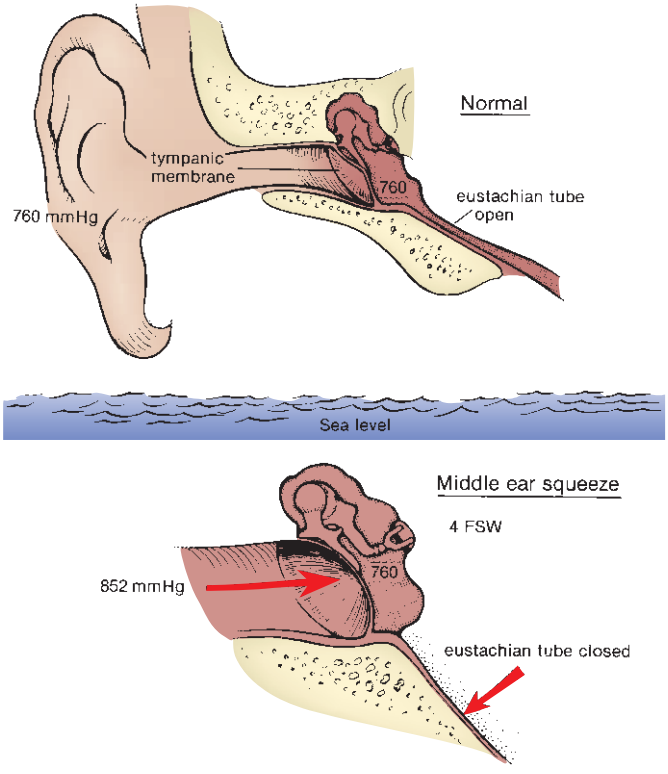


Figure 70-6. Middle ear barotrauma. Symptoms include fullness and pain caused by stretching of the tympanic membrane.

ance of diving when suffering from an upper respiratory infection or while symptomatic from allergic rhinitis, and when sinusitis, nasal polyps, or any other condition is present that impairs free flow of air from sinus cavity to nose. Significant nasal deformity may predispose to sinus barotrauma and may warrant surgical correction to allow one to continue diving.

External Auditory Canal Barotrauma. A tight-fitting wetsuit hood or drysuit hood can trap air in the external auditory canal and potentially lead to a painful external ear squeeze during descent as the volume of air is reduced according to Boyle's law. External ear squeeze also can occur if the canal is blocked by cerumen, exostoses, or foreign objects.

Symptoms and signs of external ear canal squeeze include pain, swelling, erythema, petechiae, or hemorrhagic blebs of the ear canal wall and possible bleeding when the hood is removed. Bullae may be present in the canal and on the tympanic membrane. In very severe (and very rare) cases, the tympanic membrane can rupture from the negative pressure in the ear canal. The diver, feeling pain in the canal, may believe there is inadequate equalization of the middle ear and attempt a forceful Valsalva maneuver, which increases pressure on the tympanic membrane, leading to rupture. If this occurs, further diving is contraindicated until the tympanic membrane has healed.

Treatment of ear canal barotrauma includes washing the canal with lukewarm water. Bullae should not be incised. Antibiotic drops, such as Cortisporin otic or a fluoroquinolone preparation combined with hydrocortisone, can be used to prevent infection due to contamination with seawater. For tympanic membrane perforation, fluoroquinolone otic drops should be given.

Ear canal barotrauma can be prevented by remembering to break the seal of the wetsuit hood to allow water to fill the external ear canal before descent. Ear plugs should never be worn when scuba diving.

Middle Ear Barotrauma (Barotitis Media). Referred to in diver parlance as *ear squeeze*, barotitis media is the most common medical problem in scuba diving, probably affecting more than 40% of divers at one time or another.[81] The problem is a direct application of Boyle's law (Fig. 70-6), potentially compounded by the structure of the eustachian tube.

As previously noted, Boyle's law describes the inverse relationship of pressure and volume in an enclosed airspace. It also explains why the greatest relative volume change for a given depth change occurs near the surface, which is why the greatest risk of middle ear squeeze occurs near the surface. As the diver descends, hydrostatic water pressure forces the tympanic membrane inward, and the volume within the middle ear cavity is reduced. The diver can add air into the middle ear through the eustachian tube, equalizing the pressure in the middle ear cavity with the external ambient pressure.

TABLE 70-2. Teed Grading System for Middle Ear Barotrauma

GRADE	DESCRIPTION
0	Symptoms without otologic findings
1	Erythema and mild retraction of the TM
2	Erythema of the TM with mild or spotty hemorrhage within the TM
3	Gross hemorrhage throughout the TM
4	Grade 3 changes plus gross hemorrhage within the middle ear (hemotympanum)
5	Free blood in middle ear plus perforation of the TM

TM, tympanic membrane.

Because each foot of seawater exerts a pressure of about 23 mm Hg, a diver who descends 2.5 ft (76 cm) and does not equalize pressure in the middle ear will develop a relative vacuum in the middle ear because of contraction of air volume. Typically, the diver notices slight pain at a 60-mm Hg pressure differential between air in the middle ear and ambient water pressure. This pressure differential causes the tympanic membrane to stretch and bulge inward, causing increasing discomfort and eventually severe pain. Additionally, when the tissues lining the middle ear cavity are exposed to this vacuum, vasodilatation, edema, transudation, and vascular rupture occur, causing bleeding into the mucosa and middle ear cavity.

At a depth of 4 fsw (1.2 m), a 90-mm Hg pressure differential is generated, and the unsupported, flutter-valve medial third of the eustachian tube collapses and becomes obstructed. At this point, attempts to autoinflate the middle ear by Valsalva or Frenzel maneuvers may be unsuccessful. The diver must ascend to equalize middle ear pressure with ambient environmental pressure.

If a diver does not heed the initial symptoms of barotitis media and allows the pressure differential to reach 100 to 400 mm Hg (i.e., at depths of 4.3 to 17.4 ft [1.3–5.3 m]), the pressure imbalance may lead to rupture of the tympanic membrane.[74] In such a case, the problem for the diver may be compounded by entry of cold water into the middle ear. This may cause severe vertigo.

Symptoms of middle ear squeeze include ear pain during descent, a sensation of fullness, and reduced hearing in the affected ear. There may be mild tinnitus or vertigo. With tympanic membrane rupture, the pain is relieved, but the cold caloric stimulation of seawater entering the middle ear cavity causes severe vertigo with nausea, vomiting, and disorientation underwater. The vertigo may resolve after a few minutes or continue for hours after surfacing.

The otoscopic appearance of the tympanic membrane in cases of barotitis media varies with the severity of the injury. A commonly used grading scheme is the Teed classification, which grades the severity according to the amount of hemorrhage in the tympanic membrane (Table 70-2).[66] Each higher grade tends to be more painful than the preceding one, except for grade 5, which may be relatively painless. With grade 5, the cessation of pain corresponds with the membrane tearing, which immediately equalizes the pressure in the middle ear with the external environment. Use of this grading scheme facilitates communication when describing these injuries.

In addition to having an abnormal-appearing tympanic membrane, persons with barotitis media occasionally have a small amount of bloody drainage around the nose or mouth. Audiometry usually reveals a conductive hearing loss.

Middle ear squeeze should be treated with decongestants and analgesics, although most cases will clear spontaneously in 3 to 7 days without complication. Antihistamines may be used if the eustachian-tube dysfunction has an allergic component. Divers should abstain from diving until the condition has resolved. Combining an oral decongestant with a long-acting topical nasal spray (such as 0.5% oxymetazoline or phenylephrine) for the first few days is usually most effective. Repeated gentle autoinflation of the middle ear by use of the Frenzel maneuver also can help to displace any collection of middle ear fluid through the eustachian tube. Antibiotics (e.g., amoxicillin-clavulanic acid) should be used when there is a tympanic membrane rupture or preexisting infection. If the tympanic membrane has ruptured, no diving should be done until it is fully healed. The majority of tympanic membrane perforations from diving heal spontaneously without complications in 1 to 3 months. Surgical repair can be considered if healing has not occurred within 1 month.

Prevention is key for barotitis media. Training must emphasize the importance of early and correct pressure equalization techniques. The diver should start equalizing immediately on leaving the surface. Without equalization, the eustachian tube may become "locked" by 1.2 msw (4 fsw), as described above.

There are several maneuvers for equalizing pressure in the middle ear. The Valsalva maneuver involves blowing with an open glottis against closed lips and nostrils to increase pressure in the nasopharynx to inflate the middle ear through the eustachian tube. This may force open a collapsed eustachian tube. Toynbee's maneuver is performed by swallowing with a closed glottis while the lips are closed and the nostrils are pinched. The Frenzel maneuver is performed by pinching the nose, closing the glottis, and keeping the mouth closed while moving the jaw forward and down. This moves the pharyngeal muscles, which open the eustachian tube. This move does not increase intracranial pressure. Descending feet first underwater also makes it easier to clear the middle ear.

Divers who understand the pathophysiology of barotitis media generally take steps to inflate the middle ear immediately on submerging and thereby prevent the problem as they descend in the water. If middle ear pressure is kept equal to or greater than water pressure, no problem should occur. However, if the diver forgets to inflate the middle ear or suffers from eustachian tube dysfunction (caused by mucosal congestion secondary to upper respiratory infection, allergies, smoking, mucosal polyps, excessively vigorous autoinflation maneuvers, or previous maxillofacial trauma), middle ear barotrauma may occur. This most often happens just after the diver leaves the surface, with the diver complaining of ear fullness or pain. Generally, the pain rapidly becomes so severe that the diver either corrects the problem or aborts the dive.

Topical and oral decongestants are often used prior to diving to facilitate clearing the ears.[143] Pseudephedrine has been reported to reduce the incidence and severity of middle ear barotrauma in novice divers.[22] Divers are sometimes taught not to use decongestants prior to diving because of the theoretical concern of the medication wearing off while diving and causing problems during ascent; however, no data support this concern,

and the judicious use of oral or nasal decongestants can facilitate pressure equalization.

Inner Ear Barotrauma

A serious but relatively unusual form of aural barotrauma is inner ear barotrauma causing labyrinthine window rupture. This is the most serious form of aural barotrauma because of possible injury to the cochleovestibular system that may lead to permanent deafness or vestibular dysfunction.[64,73]

Inner ear barotrauma results from rapid development of markedly different pressures between the middle and inner ear, such as may occur from an overly forceful Valsalva maneuver or an exceptionally rapid descent, during which middle ear pressure is not adequately equalized. During descent, the tympanic membrane is pressed inward, pushing the stapes against the oval window. The perilymph and endolymph are not compressible; the resulting increased pressure causes the round window to bulge outward. The diver may attempt a forceful Valsalva to equalize the middle ear; this will raise intracranial pressure, which is propagated through the perilymphatic duct to the inner ear, causing the round or oval window to rupture. Rupture of either window can also occur by a sudden increase in middle ear pressure by a forceful Valsalva. This pressure disequilibrium may cause several types of injury to the cochleovestibular apparatus, including hemorrhage within the inner ear; rupture of Reissner's membrane, leading to mixing of endolymph and perilymph; fistulation of the oval or round window, with development of a perilymph leak; or a mixed injury involving any or all of these conditions.[141] During ascent, the expanding middle ear gas may be forced through the perilymph fistula and enter either into scala tympani or scala vestibule, which may damage cochlear or vestibular structures, leading to a permanent hearing loss.

The classic triad of symptoms indicating inner ear barotrauma is roaring tinnitus, vertigo, and hearing loss. In addition, a feeling of fullness or "blockage" of the affected ear, nausea, vomiting, nystagmus, pallor, diaphoresis, disorientation, or ataxia may be present in varying degree. Symptoms of inner ear barotrauma may develop immediately after the injury or may be delayed for hours, depending on the specific damage and the diver's activities during and after the dive. Vigorous isometric exercise after a dive may complete an incipient or partial membrane rupture. Findings on physical examination may be normal or may reveal signs of middle ear barotrauma or vestibular dysfunction. Audiometry may demonstrate mild to severe high-frequency sensorineural hearing loss or a severe loss of all frequencies.

Symptoms usually improve with time. Tinnitus tends to decline over time, and vestibular injury is centrally compensated. The diver may be left with a residual high-pitch tone and a high-frequency hearing loss.

Persons with inner ear barotrauma should be treated with bed rest (with the head elevated to 30 degrees), avoidance of any strenuous activity or any straining that can lead to increased intracranial pressure, and symptomatic measures as needed. There is a good prognosis for full recovery of hearing in 3 to 12 weeks. Labyrinthine window fistulas usually heal spontaneously, and data support conservative treatment initially.[141] Deterioration of hearing, worsening of vestibular symptoms, or persistence of significant vestibular symptoms after a few days heralds the need for detailed otolaryngologic evaluation and possible surgical exploration and fistula closure. No consensus exists as to how long to wait before surgical intervention. If a perilymph fistula is suspected, it has been recommended to explore the ear surgically as soon as possible or if symptomatic after 24 hours.[17] Patients with a tear in Reissner's membrane have manifestations similar to those with inner-ear hemorrhage, although there will be persistent localized sensorineural hearing loss commensurate with the area of membrane tear. Management is the same.

Prevention of this condition is aimed at avoiding sudden, dramatic increases in middle ear pressure. Special emphasis during diver training and education should be placed on gentle pressure equalization. Upper respiratory tract infections and allergies reduce eustachian tube function and may be a precursor to inner ear barotrauma.

A diagnostic dilemma exists whenever a diver complains of vertigo, tinnitus, and hearing loss after diving. These symptoms are classic for labyrinthine rupture, in which case recompression is contraindicated because of the potential for further barotrauma to worsen the injury. Conversely, these symptoms may indicate a diagnosis of inner ear decompression sickness, which requires expeditious treatment in a hyperbaric chamber. In such cases, the most important differential feature for diagnostic use on a dive boat or other diving site is a careful history as to time of onset and dive activities preceding the onset of symptoms. If symptom onset was during descent, and ear clearing was difficult or impossible, requiring forcible Valsalva maneuvers, perilymph fistula is more likely. If the onset was during or after a decompression dive, decompression sickness must be assumed and chamber treatment sought. In some cases, however, it simply is not possible to rule out decompression sickness, or rarely, air embolism, and a "trial of pressure" in the recompression chamber may be necessary.

Suit Squeeze

If an area of the diver's skin becomes trapped under a fold or wrinkle of a drysuit, causing a closed airspace, the pressure-induced contraction of air under the fold, and resulting partial vacuum, can cause transudation of blood through the skin. This is unusual and generally benign, although the resultant skin ecchymosis may have a dramatic appearance. Suit squeeze requires no treatment and resolves in a few days to a couple of weeks.

Dental Barotrauma

Dental barotrauma or barodontalgia (tooth squeeze) is one of the most infrequent yet dramatic types of barotrauma. This painful condition, sometimes called aerodontalgia, is caused by entrapped gas in the interior of a tooth or in the structures surrounding a tooth. The confined gas develops either positive or negative pressure relative to ambient pressure, which exerts force on the surrounding sensitive dental structures and causes pain.

Barodontalgia may be caused by an array of dental conditions, including caries, defective restorations, oral tissue lacerations, recent extractions, periodontal abscesses, pulpal or apical lesions or cysts, and endodontal (root canal) therapy. If a pocket of trapped air remains at sea level pressure while ambient pressure increases during descent, the tooth can implode or the cavity fill with blood. Conversely, air that is forced into a tooth during descent can expand during ascent, causing the tooth to explode. To prevent barodontalgia, a diver probably should wait at least 24 hours after dental treatment (including fillings) before diving.

Other causes of tooth pain associated with pressure changes are less well understood. Pulpitis or other dental infections can produce pain during a dive. Upper tooth pain associated with pressure changes should raise a question about a pathologic condition in the maxillary sinus.

Lung Squeeze

Lung squeeze is a very unusual form of barotrauma that has been observed with breath-hold diving. Persons having this syndrome complain of shortness of breath and dyspnea after surfacing from a deep (greater than 100 fsw) breath-hold dive. The diver may cough up frothy blood, and a chest radiograph may show pulmonary edema. The condition is treated with supplemental oxygen and respiratory support as needed. Symptoms typically resolve within a few days.

The classic understanding of lung squeeze is that it occurs when a diver descends to a depth at which total lung volume (TLV) is reduced to less than residual volume (RV). At this point transpulmonic pressure exceeds intra-alveolar pressure, causing transudation of fluid or frank blood (from rupture of pulmonary capillaries) and the overt manifestations of pulmonary edema and hypoxemia.

According to this scenario, a breath-hold diver with a TLV of 6000 mL and an RV of 1200 mL could dive to only 6000/1200 or 5 ATA (equal to 132 fsw) before lung squeeze would occur. However, breath-hold divers have dived much deeper without apparent problem.

In 1968, Schaefer and associates reported that breath-hold divers pool their blood centrally, accumulating a central volume increase of as much as 1047 mL at 90 fsw.[156] If it is assumed that this adjustment in pulmonary blood volume reduces the RV, then theoretically it should be possible for the diver with a TLV of 6000 mL to breath-hold to 6000/(1200 – 1047 = 153) or almost 40 ATA. Although deep breath-hold dives seem to support the beneficial effect of central pooling of blood, cases of lung squeeze continue to occur at much shallower depths. The exact pathophysiology of this condition remains unclear. Fortunately, it occurs very infrequently.

Underwater Blast Injury

Barotrauma can be caused by underwater explosions. Shock waves from a blast are propagated farther in the dense medium of water than in air.[36] Underwater explosions may result from ordnance or ignition of explosive gases during cutting or welding operations.

Underwater blasts can cause serious injuries to divers. Air-containing body cavities such as the lungs, intestines, ears, and sinuses are most vulnerable. Pneumothorax, pneumomediastinum, and air embolism may result from laceration of the lung and pleura.[93] There may be intestinal perforation, subserosal hemorrhage, and subsequent peritonitis. The occurrence of blast-related air embolism at depth, which worsens with ascent to the surface, requires treatment by recompression, if possible. Otherwise, management of underwater blast injuries is the same as for terrestrial blast injury.

Barotrauma of Ascent

Reverse Sinus or Ear Barotrauma (Reverse Squeeze)

The sinuses and ears are subject to barotrauma during ascent as well as descent. As the ambient pressure drops, the volume of the gas within the sinus or ear cavities expands, causing pain and tissue damage if not released. Gas pressure can exceed intravascular pressure in adjacent tissue, causing local ischemia. In the sinuses, a cyst or polyp can act as a one-way valve; air enters the sinus as the diver descends but cannot escape as the diver ascends. Fainting underwater due to frontal sinus pain has been reported.[75]

Pain can develop in the ear during ascent due to expanding gas pressure in the middle ear that is not released through the eustachian tube. If a diver makes frequent descents and ascents during a dive or multiple short dives back-to-back ("bounce diving"), the mucosal lining of the eustachian tube often becomes inflamed, making equalization of pressure in the ears difficult. The eustachian tube may not allow air to escape fast enough and the pressure rises in the middle ear during ascent. This results in pain, often tinnitus and vertigo, and may lead to an outward rupture of the tympanic membrane.

Alternobaric Vertigo

An unusual type of aural barotrauma is alternobaric vertigo (ABV). This usually occurs with ascent and is caused by sudden development of unequal middle ear pressure, which causes asymmetric vestibular stimulation and resultant pronounced vertigo.[120] Vertigo, nausea, and vomiting may occur as the diver ascends. Although usually only transient and requiring no treatment, ABV may precipitate a panic response, leading to near drowning, pulmonary barotrauma, and resultant air embolism; however, the incidence of diving injuries due to ABV is unknown. Rarely, alternobaric vertigo lasts for several hours or days, in which case it should be treated symptomatically after excluding inner ear barotrauma. Diving should be avoided when middle ear equalization is compromised.

Alternobaric Facial Palsy

The 7th cranial nerve courses through the middle ear and mastoid process via a bony channel. Parts of the nerve may be directly exposed to middle ear pressures through a defect in the canal wall. During ascent, if the eustachian tube mucosa is swollen due to irritation, infection, or allergy, the middle ear pressure may exceed the capillary pressure of the facial nerve and cause an ischemic neuropraxia.[63,130] The diver may complain of ear fullness and pain after surfacing, along with dysesthesias of the face and tongue. The diver is unable to close the eye on the affected side, and the mouth may be affected. Otoscopy reveals a bulging tympanic membrane.

The diver can try the Toynbee maneuver, as described above, to release middle ear overpressure. Oral and topical decongestants should be used. The middle ear gas is eventually absorbed, which reduces the pressure. However, permanent damage of the nerve can occur if the pressure is elevated too long. Recompressing the diver to 1 msw (3 fsw) can restore capillary circulation in the nerve. A myringotomy may be necessary. The diver's diving profile and lack of other symptoms and signs help exclude the diagnosis of arterial gas embolism (AGE) or DCS.

Gastrointestinal Barotrauma

Because the intestines are pliable, contraction of intraluminal bowel gas during descent does not cause barotrauma. In unusual situations, however, expanding gas can become trapped in the gastrointestinal tract during ascent and cause gastrointestinal barotrauma, which is also known as aerogastralgia, or gas in the gut.[39,119] This infrequent condition has been noted

most often in novice divers, who are more prone to aerophagia; in divers who repeatedly perform the Valsalva maneuver in the head-down position, which may force air into the stomach, or who chew gum while diving; and in divers who consume large quantities of carbonated beverages or legumes shortly before diving.

Divers with gastrointestinal barotrauma typically complain of abdominal fullness, colicky abdominal pain, belching, and flatulence. Rarely, syncope has been reported and is presumed to result from a combination of decreased venous return and vagal reflexes. Most often, gas accumulates in the gastric antrum.

The physical examination of a diver having symptoms of gastrointestinal barotrauma is usually normal because the condition typically resolves by the time medical care is obtained. However, abdominal distention, tympany, and abdominal tenderness may be found. In an extreme case, there may even be signs of cardiovascular compromise as a result of obstruction of venous return.[65]

Gastrointestinal barotrauma is most often self-remedied by elimination of the excess gas. Recompression may be necessary in very rare, severe cases.

Pulmonary Barotrauma (Pulmonary Overpressurization Syndrome)

The most serious type of barotrauma is pulmonary barotrauma of ascent, which results from expansion of gas trapped in the lungs. If a diver does not allow the expanding gas to escape, a pressure differential develops between the intrapulmonary air space and the ambient pressure. The combination of overdistention of the alveoli and overpressurization causes the alveoli to rupture, producing a spectrum of injuries collectively referred to as pulmonary barotrauma or the pulmonary overpressurization syndrome (POPS)—the latter term being used in reference to the condition being a dramatic clinical demonstration of Boyle's law.

Divers suffering pulmonary overpressurization typically give a history of rapid and uncontrolled ascent to the surface before the onset of symptoms (typically as a result of running out of air, panic, or sudden development of uncontrolled positive buoyancy, such as may occur when a diver drops his weight belt or inadvertently inflates his buoyancy compensator). However, pulmonary barotrauma does occur in divers who ascend slowly with no discernible cause. There is presumed to be localized overinflation of the lung in these cases.

Localized overinflation of the lung from focally increased elastic recoil may occur in divers who ascend at a proper rate.[37,38] Theoretically, if there are focal areas of decreased compliance in the lungs, the adjacent areas of normal compliance would be subjected to greater forces, leading to barotrauma.[78] With immersion in diving, central pooling of blood causes an increase in intrapulmonary blood volume, and the lungs become stiffer; this decreased compliance may increase the risk of pulmonary barotrauma.

If a given intrapulmonary gas volume is trapped by forcible breath-holding or a closed glottis, or even in a small portion of the lung by bronchospasm during ascent, intrapulmonary volume increases (according to Boyle's law) until the elastic limit of the chest wall is reached. After that, intrapulmonary pressure rises until, at a positive differential pressure of about 80 mm Hg, air is forced across the pulmonary capillary membrane. This air usually enters either the pulmonary interstitial spaces or the pulmonary capillaries.

It is important to remember that there is significant change in barometric pressure in shallow water. Boyle's law dictates greater volume changes for a given change in depth near the surface than at greater depths. Thus, shallow depths are the most dangerous for breath-holding ascents. A pressure differential of only 80 mm Hg (alveolar air) above ambient water pressure on the chest wall, or about 3 to 4 ft (1 to 1.3 m) of depth under water, is adequate to force air bubbles across the alveolar-capillary membrane. Fatal pulmonary barotrauma has occurred from breath-holding during ascent from a depth as shallow as 4 ft (1.3 m) of water.

The diagnosis of pulmonary barotrauma is based on development of characteristic symptoms after diving. The actual clinical manifestations may take several forms, depending on the course that the extra-alveolar air travels. Once the alveoli rupture, air can remain in the interstitium causing localized pulmonary injury and alveolar hemorrhage. Air can travel along the perivascular sheaths and dissect into the mediastinum. This air can track superiorly to the neck, resulting in subcutaneous emphysema and can dissect inferiorly and posteriorly, causing pneumoperitoneum. The air may dissect to the visceral pleura causing a pneumothorax. If air enters the pulmonary vasculature, it can travel to the heart and embolize systemically, causing an arterial gas embolism (AGE) (Fig. 70-7).

Clinical Manifestations of Pulmonary Barotrauma. The specific clinical manifestations of pulmonary barotrauma depend on the location and amount of air that escapes into an extra-alveolar location.

Local Injury. Air can rupture the alveoli, causing localized pulmonary injury and capillary bleeding without other signs of pulmonary barotrauma such as pneumomediastinum or arterial gas embolism. Diffuse alveolar hemorrhage has been described as a rare manifestation of pulmonary barotrauma.[4] The diver may complain of chest pain, cough, and hemoptysis without any neurologic findings. Intraparenchymal lung injury and bleeding may be seen on a chest x-ray. A diver with local pulmonary injury without any evidence of AGE does not require recompression and should be treated with supportive care.

Pneumomediastinum or mediastinal emphysema is the most common form of pulmonary barotrauma, resulting from pulmonary interstitial air dissecting along bronchi to the mediastinum. The diver may be asymptomatic or complain of substernal chest pain. Respiratory distress is typically not present. If air dissects from the mediastinum up to the neck, the diver may experience hoarseness and neck fullness. Subcutaneous emphysema may be present and palpated as crepitance under the skin of the neck and anterior chest. In severe cases, the diver may complain of marked chest pain, dyspnea, and dysphagia. Hamman's sign (also known as Hamman's murmur or Hamman's crunch)—a crunching, rasping sound, synchronous with each heartbeat, heard over the precordium during auscultation of the chest—may rarely be present when air is present in the mediastinum around the heart. Radiographs may show extra-alveolar air in the neck, mediastinum, or both, although radiographs are rarely necessary to make the diagnosis. The presence of air on radiograph may be very subtle and is often best visualized along the pulmonary artery and aorta and along the edge of the heart.

Treatment of pneumomediastinum is conservative, consisting of rest, avoidance of further pressure exposure (including flying

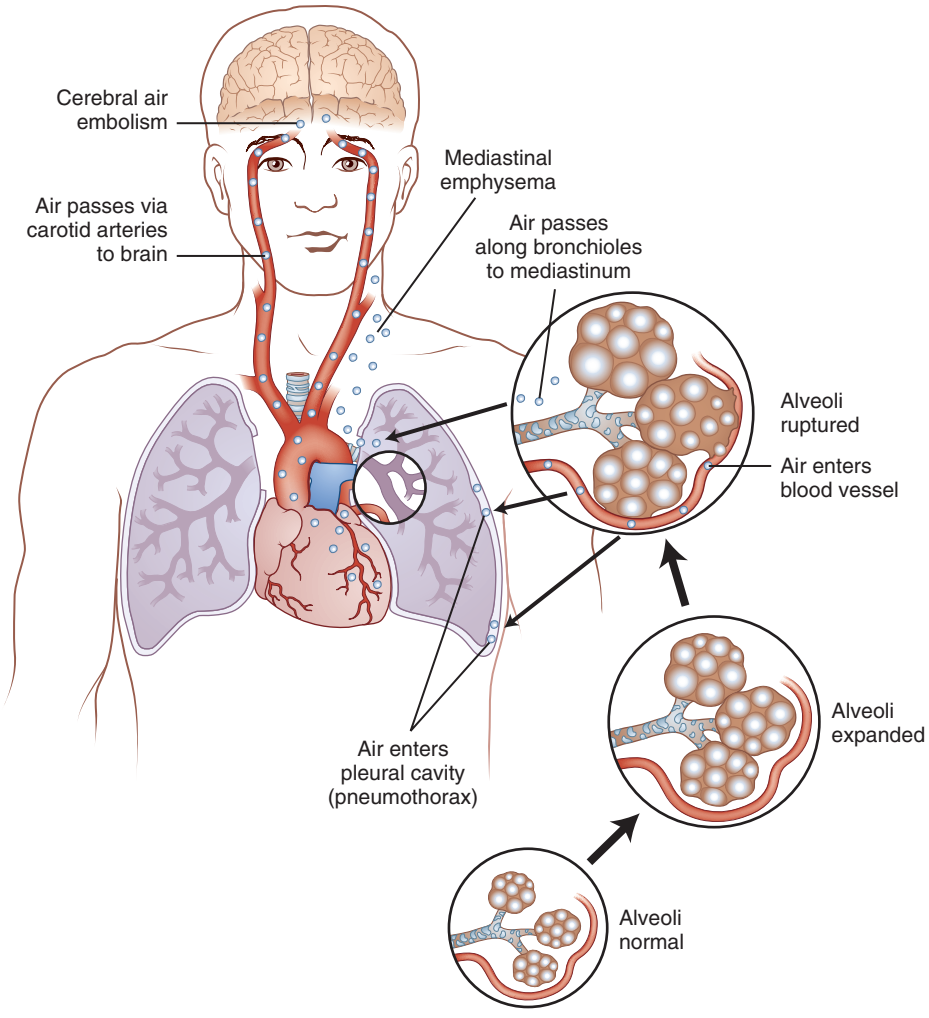


Figure 70-7. Pulmonary barotrauma. Diagram of different manifestations of pulmonary barotrauma including mediastinal emphysema, subcutaneous emphysema, pneumothorax, and arterial gas embolism (AGE).

in commercial aircraft), and observation. Supplemental oxygen administration may be useful in severe cases. Recompression is indicated only in cases associated with AGE.

Pneumothorax is an infrequent manifestation of diving-related pulmonary overpressurization, because it requires that air be vented through the visceral pleura, a path generally having greater resistance than air tracking through the interstitium. Although pneumothorax has been reported to occur in 5% to 10% of cases of AGE,[146] its occurrence is much less frequent in the authors' experience. Despite being infrequent, pneumothorax must be considered and excluded whenever pulmonary barotrauma or AGE is suspected, because recompression can turn a simple pneumothorax into a tension pneumothorax during ascent in the chamber.

In cases of diving-related pneumothorax, the diver usually complains of pleuritic chest pain, breathlessness, and dyspnea, just as in cases of pneumothorax from any other cause. Radiographs may confirm the diagnosis. Because the majority of diving-related pneumothoraces are small, treatment may consist simply of supplemental oxygen and close observation, repeating the chest x-ray daily to ensure resolution. Tube thoracostomy is usually reserved for a larger pneumothorax or if the diver is to undergo recompression treatment. A chest tube is necessary if the diver is recompressed because the expanding intrapleural gas of the pneumothorax cannot otherwise be vented to the environment during depressurization from hyperbaric treatment, and it may convert to a lethal tension pneumothorax.

Tension pneumothorax is one of very few diving-specific disorders that can quickly kill a diver at the dive site (or in a recompression chamber), but is curable if recognized. Although pneumothorax may result from pulmonary barotrauma, it is also associated with severe blunt chest trauma (e.g., from an underwater landslide or cave-in or resulting from a collision with a boat) or may develop spontaneously, as has been observed with other outdoor sports.[107] Blunt chest trauma is more likely to occur in commercial diving.

Divers developing a pneumothorax underwater will almost always develop shortness of breath, dyspnea, and pleuritic chest pain, although these symptoms may not be recognized or their significance may not be recognized at the time. Shortness of breath will always worsen during ascent, although the chest

pain often improves during ascent as the pneumothorax enlarges and the lung no longer touches the chest wall. Breath sounds will be diminished when the chest is auscultated.

Treatment of tension pneumothorax in divers is by closed chest thoracostomy, the same as in other settings.

ARTERIAL GAS EMBOLISM

Arterial gas embolism (AGE) is the most feared complication of pulmonary barotrauma. AGE is one of the most dramatic and serious injuries associated with compressed-air diving and is a major cause of death and disability among sport divers.[104] Many diver deaths officially listed as drowning probably have been cases of cerebral air embolism. Unfortunately, the accident investigation and postmortem evaluation of many diving-accident victims are insufficient to establish the precise cause of death. Autopsies of diving-accident victims should be performed according to special procedures.[50]

Pathophysiology of AGE

AGE results from air bubbles entering the pulmonary venous circulation from ruptured alveoli. When air is introduced into the pulmonary capillary blood, gas bubbles are showered into the left atrium, to the left ventricle, and subsequently into the aorta where they are distributed throughout the vasculature. Bubbles may enter the coronary arteries and produce electrocardiogram (ECG) changes and elevation of cardiac enzymes, but rarely myocardial injury.[163] Gas embolization to the coronary arteries also may induce arrhythmias.[100]

Most of the bubbles entering the aorta pass into the systemic circulation, lodging in small arteries and occluding the more distal circulation. Bubbles travel up the carotid or vertebral arteries to embolize the brain, causing a combination of mechanical and reactive sequelae. Vascular occlusion causes distal ischemia, which is compounded by damage to the vascular endothelium and disruption of the blood-brain barrier and resultant cerebral edema. Systemic hypertension occurs, cerebrospinal fluid (CSF) pressure rises, and there is reactive hyperemia causing loss of autoregulation of cerebral perfusion. Cerebral blood flow now reflects changes in systemic blood flow.

Depending on the site or sites of circulatory occlusion, AGE produces myriad and often disastrous consequences. The neurologic pattern may be confusing as showers of bubbles randomly embolize the brain's circulation, producing ischemia and infarction of diverse brain regions. Combined carotid and vertebral artery embolization may produce severe, diffuse brain injury (Fig. 70-8).[104]

Arterial gas embolism typically develops during ascent or immediately after the diver surfaces, at which time the high intrapulmonary pressure resulting from lung overpressurization is relieved, allowing bubble-laden pulmonary venous blood to return to the heart and pass into the systemic circulation. It is axiomatic that symptoms of AGE develop within 10 minutes of surfacing from a dive, although most often they are clearly evident within the first 2 minutes. Sudden loss of consciousness on surfacing from a dive should be considered to represent air embolism until proven otherwise.

In a pulmonary overpressure accident, as soon as normal breathing resumes at the surface of the water, the pressure differential that drives air bubbles into the pulmonary capillaries is equalized. From this point on, usually no further intra-arterial air is introduced.

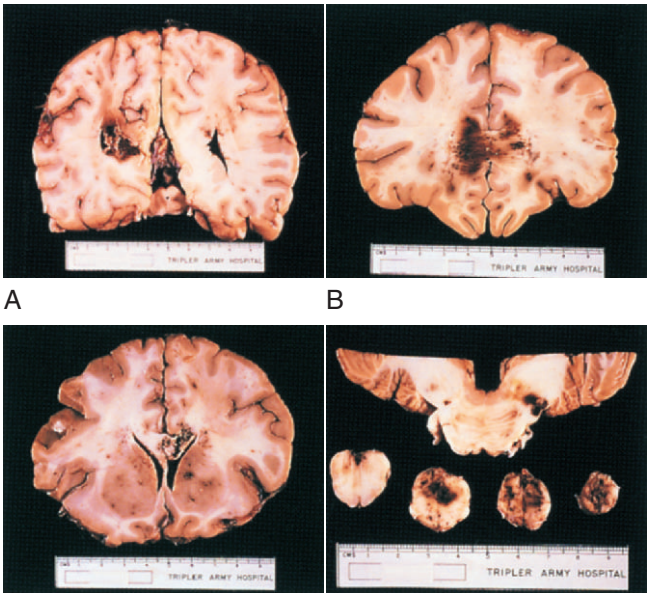


Figure 70-8. **A** to **D,** Cross sections of the brain of a 31-year-old male sport diver who suffered an arterial gas embolism, dying 4 days after the accident and extended hyperbaric oxygen treatment. Bubble-induced infarcts are found throughout the brain in the distribution of both the carotid and vertebral arteries. (Courtesy Kenneth W. Kizer, MD.)

Sudden Death due to AGE

Approximately 5% of divers who suffer an AGE will die immediately, presenting with sudden collapse, pulselessness, and apnea. These victims are not responsive to immediate cardiopulmonary resuscitation or recompression. It was previously thought that the cause of sudden death from AGE was due either to reflex arrhythmias from brain stem embolization or to myocardial ischemia and death due to coronary artery embolization and occlusion.[30,70] However, these mechanisms do not explain what is seen in animal models or clinically in humans. In sudden death from AGE, there is complete filling of the central vascular bed with air.[137] Radiographs of fatal cases of AGE demonstrate massive amounts of air in the central vasculature (Fig. 70-9).[139] Currently, it is believed that the primary mechanism of cardiac arrest in most cases of AGE is vascular obstruction due to air leading to pulseless electrical activity.[139]

Clinical Manifestations of AGE

Clinical manifestations of cerebral air embolism are sudden, dramatic, and often life threatening. Approximately 5% of victims of AGE suffer immediate cardiac respiratory arrest and die. Another 5% die in the hospital due to consequences of the AGE or severe near-drowning that can accompany AGE. Over half of the remaining victims of AGE have complete functional recovery.

Victims of AGE present with varied clinical symptoms depending on the amount and distribution of air. Neurologic manifestations of AGE are typical of an acute stroke, although hemiplegia and other purely unilateral brain syndromes are

infrequent. Loss of consciousness, monoplegia or asymmetric multiplegia, focal paralysis, paresthesias or other sensory disturbances, convulsions, aphasia, confusion, blindness or visual field defects, vertigo, dizziness, or headache are most often observed (Table 70-3).[56,70,104,127,135]

The physical findings of AGE are extremely variable and depend on the specific site or sites of vascular occlusion. Neurologic findings generally dominate the clinical picture because of the frequency of cerebral involvement. All patients with suspected AGE should be carefully examined for neurologic deficits. Often, the neurologic findings are subtle and require detailed examinations, including testing of cognitive function.[135] Such testing is rarely possible at the dive site, so divers who have a history suggestive of AGE (e.g., loss of consciousness after ascending from a dive) but who do not manifest any gross symptoms or signs of neurologic injury in the field should be given the benefit of the doubt and referred to a hyperbaric treatment facility. Specific manifestations of pulmonary barotrauma (such as subcutaneous or mediastinal emphysema) may or may not be present but should always be carefully sought.

Persons suffering an AGE may manifest various hematologic and biochemical abnormalities. Gas bubbles distribute systemically and are thought to cause direct organ injury or injury to vascular endothelium or both. AGE patients usually manifest hemoconcentration, most likely due to third-spacing from endothelial injury. The degree of hemoconcentration correlates with the neurologic outcome of the diver.[165] Creatinine kinase (CK) is elevated in almost all cases of AGE and also correlates with eventual neurologic outcome of the diver.[163] The more elevated the CK, the less likely is a full functional recovery. The majority of the elevated CK is from skeletal muscle (the MM component). CK-MB is elevated in some cases, and nonspecific ECG changes can occur; however, true myocardial infarction due to AGE is extremely rare.[21] Even in cases of AGE with elevated CK-MB, functional studies of the heart show no evidence of wall motion abnormalities following recovery.[163] Bubbles cause injury to other organs. Elevated serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), and lactate dehydrogenase (LDH) can be found in victims of AGE.[164] Despite elevations of these enzymes, organ function usually does not decline.

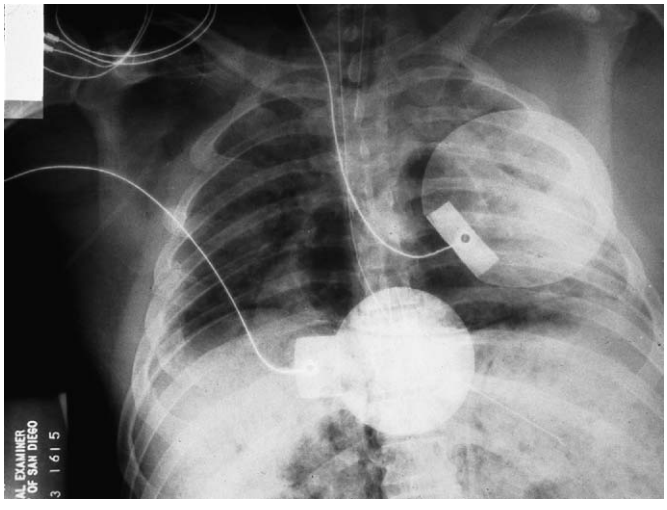


Figure 70-9. Chest radiograph of a diver who died suddenly from an AGE showing complete filling of the central vascular bed with air. Note the air in the heart and carotid arteries.

AGE can lead to loss of consciousness upon surfacing; hence, it is not unexpected to find evidence of aspiration and near drowning clinically and on chest x-ray. Radiographic findings consistent with aspiration can be found in over 50% of chest x-rays on victims with AGE.[86] However, many of the same radiographic findings may be seen with venous gas embolism.[106]

Rarely, air bubbles may be visualized in the retinal arteries, or sharply circumscribed areas of glossal pallor (Liebermeister's sign) may be noted, but these findings cannot be relied on to make the diagnosis. The diagnosis of AGE is clinical and based on the diving history and symptoms. Any diver who loses consciousness or manifests symptoms or signs of serious neurologic injury within 5 minutes of surfacing from a dive must be considered to have suffered an AGE.

Treatment of AGE

All cases of suspected AGE must be referred for recompression treatment (hyperbaric oxygen therapy) as rapidly as possible. This is the primary and essential treatment for the condition.[61]

Prehospital Care

Before recompression, whether in the field, emergency department, or clinic, or during transport to a recompression chamber, the affected diver should be given supplemental oxygen at a high flow rate (10 L/min by nonrebreather face mask). Supplemental oxygen will enhance the rate of resolution of inert gas bubbles and treat arterial hypoxemia.

Historically, much attention was directed toward keeping the AGE patient in the Trendelenburg position in the field. This

TABLE 70-3. Pulmonary Overpressure Accident: Typical Symptoms and Signs

CONDITION	SYMPTOMS	SIGNS
Arterial gas embolism	Seizure (focal or general), unconsciousness, confusion, headache, visual disturbances, bloody sputum	Hemiplegia, monoplegia, altered level of consciousness, blindness, visual motor deficit, focal motor or sensory loss
Mediastinal-subcutaneous emphysema	Substernal pain, "brassy voice," neck swelling, dyspnea, bloody sputum	Subcutaneous crepitus, gas patterns on radiograph of mediastinum and neck
Pneumothorax	Chest pain, dyspnea, bloody sputum	Loss of breath sounds, hyperresonant chest percussion, tracheal shift

was based on anecdotal reports and limited experimental data.[80,110,171] The rationale for keeping the patient with AGE head down was the belief that the weight of the column of blood would force bubbles through the cerebral capillary bed, that the buoyancy of the bubbles would keep them in the aorta or heart, and that the weight of the spinal fluid might compress bubbles in the cord. These benefits were never well demonstrated or experimentally confirmed, and more recent studies showed that the Trendelenburg position did not keep bubbles from being distributed to the systemic circulation.[23] Additionally, it is more difficult to oxygenate persons in a head-down position, and if one is maintained in this position for longer than 30 to 60 minutes, it may cause or worsen cerebral edema. Therefore, it is now recommended that AGE patients be maintained in the supine position, both in the field and during transport to an emergency medical treatment facility or recompression chamber.

Because the AGE event so often occurs while the victim is in the water, he or she frequently suffers concomitant near-drowning. Thus, the rescuer must be prepared to provide cardiopulmonary resuscitation (CPR) and to protect the airway from aspiration of gastric contents secondary to vomiting.

While life support measures are being instituted, a member of the diving or rescue team should telephone the nearest civilian or military recompression chamber and contact an air ambulance if air evacuation is required. Aircraft selection is crucial because the stricken diver should not be exposed to a significantly lower atmospheric pressure in the aircraft. Ideally, the diver should be transported by aircraft pressurized to sea level so any intra-arterial bubbles do not expand further. In the case of helicopter evacuation or in the event that an unpressurized aircraft is required, the flight altitude must be maintained as low as possible, not to exceed 1000 ft (305 m) above sea level, if possible.

An intravenous infusion of isotonic solution should be started, and urine output maintained at 1 to 2 mL/kg/hr.[46] As mentioned above, victims of AGE present with hemoconcentration due to endothelial injury from gas bubbles. It is important to maintain adequate intravascular volume because inert gas cannot be effectively eliminated from tissues or from intravascular bubbles at the arteriolar-capillary level unless adequate capillary perfusion is maintained. Additionally, autoregulation of blood flow in the brain is lost following AGE, and cerebral perfusion passively follows systemic blood pressure. Hypotension should be avoided in cases of AGE.

The AGE-affected diver should be transported to the recompression chamber as quickly as possible. Delay prolongs cerebral ischemia and cellular hypoxia, resulting in significant cerebral edema, which typically leads to a more difficult course of therapy. Nonetheless, transport should still be undertaken even if delay is unavoidable. Remarkable improvement has been seen in cases in which treatment was delayed for more than 24 hours after the onset of neurologic manifestations.[101]

If the AGE-stricken diver is first seen at a hospital emergency department or clinic, and if transport to a hyperbaric treatment facility will not be delayed, baseline laboratory, radiographic, and other diagnostic tests (such as an ECG) should be obtained before the patient is sent to the chamber. Computed tomography (CT) or MRI of the brain should be deferred until after initial hyperbaric treatment unless intracranial hemorrhage, carotid artery dissection,[133] or other nondiving injury is strongly suspected.

Recompression Treatment

Hyperbaric treatment consists of rapidly increasing ambient pressure (recompression) to reduce intravascular bubble volume and to restore tissue perfusion; it uses oxygen-enriched breathing mixtures to enhance bubble resolution and to supply oxygen to hypoxic nervous tissue, and then providing slow decompression to avoid the reformation of bubbles.[19,46,49] Additional details about the hyperbaric regimens used for treating AGE are provided in Chapter 71. Ideally, these patients should be treated in multiplace hyperbaric chambers (Fig. 70-10) capable of being pressurized to 6 ATA and in which both air and oxygen can be administered to the patient. There is growing evidence that recompression to 2.8 ATA on 100% oxygen may be more effective treatment than initial pressurization to 6 ATA for sports divers, because of the typical several-hour delay in getting to a chamber and, thus, less need for the higher pressure to resolubilize bubbles (and perhaps a greater immediate need for tissue oxygenation).[57,103,104,114,115]

Although the majority of victims with AGE have neurologic deficits when examined, the initial manifestations may spontaneously resolve by the time the victim is seen by medical personnel. Occasionally, a person has symptoms but no reproducible neurologic deficits on physical examination.[104,113] Nonetheless, all patients must be referred for diving medicine consultation and hyperbaric treatment if the history is suggestive of AGE, because neurologic impairment is impossible to exclude in the acute care setting, and waiting to complete definitive diagnostic studies may allow subtle neurologic injuries to become irreversible.[135]

Adjunctive Treatment

Over the years, numerous medications have been proposed as adjuncts to recompression and hyperbaric oxygen for the treatment of AGE (e.g., heparin, low–molecular-weight dextran, aspirin, corticosteroids); however, experimental and clinical data do not support the use of any of these except for lidocaine.

In recent years, lidocaine has been proposed as an adjuvant to recompression for the treatment of AGE, and limited experimental data support its use. Lidocaine is a class 1b antiarrhythmic agent and a local anesthetic that has cerebroprotective effects. In animal models of AGE and brain ischemia, lidocaine acts to preserve brain blood flow, reduce brain edema and intracranial pressure, and preserve neuroelectrical function.[128,129] When given prophylactically, lidocaine reduces brain dysfunction after AGE in cats[71] and improves recovery of brain function in cats and dogs with AGE when given therapeutically.[61,72] Human data are less impressive, since many of the case studies contain patients with both AGE and decompression sickness (DCS).[35,55] When lidocaine is used prophylactically during left heart valve surgery, patients had a greater recovery of brain function after surgery and fewer deficits in psychometric test performance compared to a control group.[129] Overall, the evidence supports the use of lidocaine as an adjunct to recompression for the treatment of AGE.

Prevention of Pulmonary Barotrauma and AGE

In view of the potentially catastrophic consequences of AGE, one of the key goals of scuba-diving training is to prevent pulmonary overpressure accidents. Divers must be warned of the potentially great intrathoracic volume changes that can occur at shallow depths and trained to keep an open airway during ascent, particularly through the last 10 ft (3 m) to the surface. If

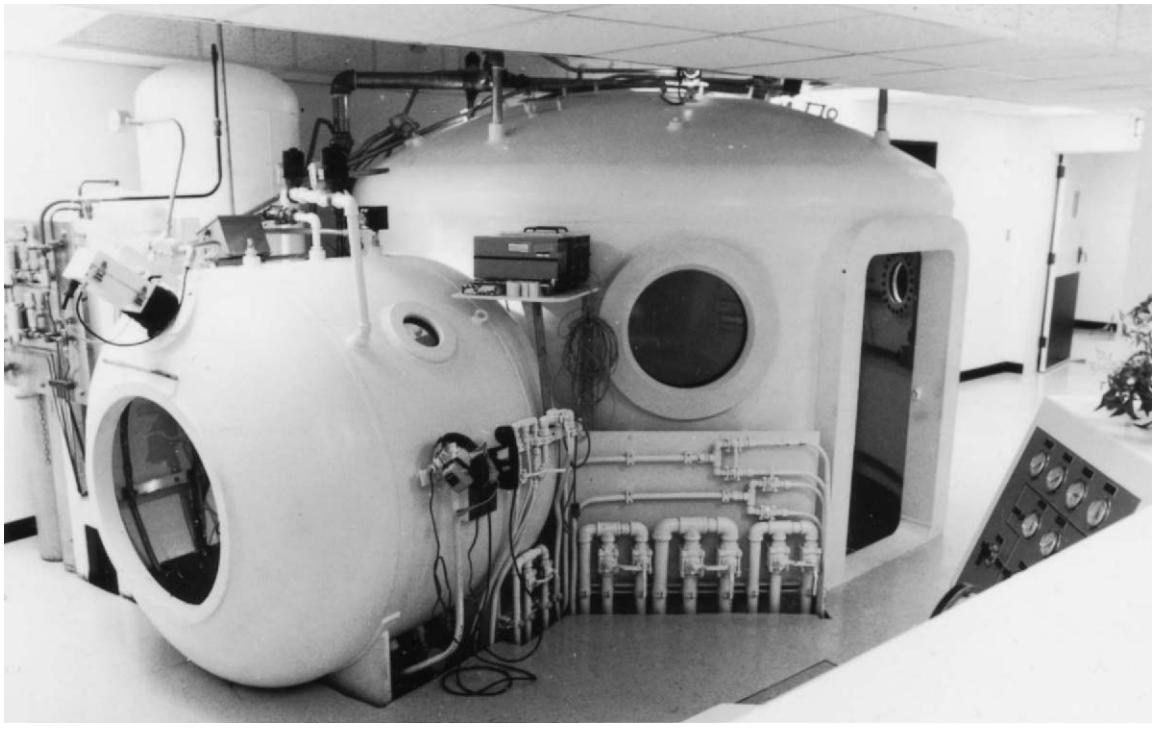


Figure 70-10. An example of a large modern hyperbaric chamber. Preferably, a hyperbaric chamber for treating decompression sickness or arterial gas embolism should have a pressure capability of at least 6 ATA (165 fsw) and should have space for an attendant to provide ongoing hands-on care and repeated neurologic examinations. It must have provisions for supplying 100% oxygen and other gases for treatment of the stricken diver. (Courtesy Kenneth W. Kizer, MD.)

equipment malfunction or depletion of air supply at depth makes this impossible, the diver must make every attempt to exhale continuously during an "emergency swimming ascent" in order to vent the increasing volume of air in the lungs. With a satisfactory air supply, the diver should simply breathe normally on ascent to the surface, taking care to ascend slowly near the surface.

INDIRECT EFFECTS OF PRESSURE

Several diving-related problems may develop as a result of breathing gases at higher than normal atmospheric pressure. Chief among these are nitrogen narcosis, oxygen toxicity, and decompression sickness.

Dalton's Law of Partial Pressures

Dalton's law of partial pressures states that the total pressure exerted by a mixture of gases is the sum of the pressures that would be exerted by each of the gases if it alone occupied the total volume. The partial pressure of a gas in a mixture is the pressure exerted by that gas alone. The symbols for partial pressure of oxygen, nitrogen, carbon dioxide, and water vapor are PO_2, PN_2, PCO_2, and PH_2O, respectively. Dalton's law states that in an air mixture the total pressure (PT) = $PN_2 + PO_2 + PH_2O + P_{other}$. The partial pressure of each gas in the mixture is found by multiplying the percentage of that gas present by the total pressure. In Figure 70-11, in a mixture of air, nitrogen is assumed to be present in a proportion of 78%, oxygen at 21%, carbon dioxide at 0.03%, and the balance composed of water vapor and other trace gases.

The partial pressures of inspired gases in a gas mixture, not their percentages, are of prime importance in diving. For example, it has been shown that in hyperbaric chamber treatment of decompression sickness, 100% oxygen can be safely used at depths to 60 fsw (2.8 ATA) for 20-minute periods with the subject at rest in the dry chamber. On the other hand, with 21% oxygen in a helium-oxygen mixture at 600 fsw (20 ATA), the diver would breathe 0.21 × 20 ATA, or 4.2 ATA of oxygen, which would rapidly produce CNS oxygen poisoning. This type of problem is avoided in deep diving by reducing the oxygen percentage in the gas mixture to between 0.35 and 0.50 ATA, so that the PO_2 will be between 266 and 380 mm Hg.

The scuba diver who uses open-circuit compressed air is subject to the effects of the component gases in the air according to their partial pressures. Thus, even though the gas mixture is simply air with normal percentages of oxygen and nitrogen, the increases in partial pressures of these gases and those of the trace contaminants at sea level create numerous potential breathing medium–related problems. Most notable among these for scuba divers is the problem of nitrogen narcosis.

Nitrogen Narcosis

Nitrogen narcosis, also known as rapture of the deep, inert gas narcosis, or the narcs, is development of intoxication due

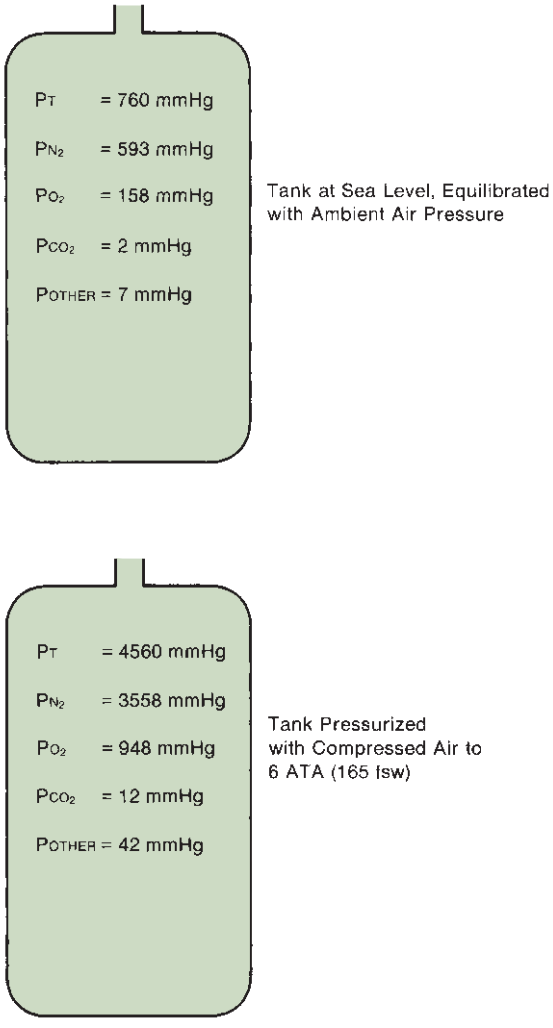


Figure 70-11. Dalton's law of partial pressures. The total pressure exerted by a mixture of gases is the sum of the pressures that would be expected of each of the gases if it alone were present and occupied the total volume.

to the increased partial pressure of nitrogen in compressed air at increased depth. Nitrogen narcosis is important to divers because it causes anesthetic-like euphoria, overconfidence, and deterioration in judgment and cognition, all of which can lead to serious errors in diving techniques, accidents, and drowning. Many divers have died as a consequence of nitrogen narcosis.

During the 1930s, Behnke and associates[9,10] first suggested that the mood changes described by divers breathing compressed air at 200 fsw were caused by high inspired P_{N_2}. Much has been learned since that time, and a complete compilation of data on inert gas narcosis has been provided by Bennett.[13–15] The subject was more recently reviewed by Hamilton and Kizer.[84]

The means by which nitrogen causes its intoxicating effect is believed to be similar to how gaseous anesthetics work in general. According to currently accepted theory, an alteration in electrical properties of cellular membranes is affected by absorption of gas molecules into their lipid component. Supporting this theory is the observation that the greater the lipid solubility of a given gas, the greater is its narcotic potency. Thus, higher partial pressures of pure nitrogen are required to produce an anesthetic effect than one that can be achieved by a much lower partial pressure of nitrous oxide, which is much more soluble in lipid than is nitrogen. Helium's lack of narcotic effect is in accord with its low lipid solubility. Indeed, substitution of helium for nitrogen as the inert gas in the diver's breathing gas prevents nitrogen narcosis and is the main reason helium-oxygen mixtures are used for deep diving.

Typically, a scuba diver breathing compressed air develops symptoms of nitrogen narcosis at depths between 70 and 100 fsw (21–31 msw). These symptoms include lightheadedness, loss of fine sensory discrimination, giddiness, and euphoria. Symptoms progressively worsen at deeper depths. At depths over 150 fsw (46 msw), a diver becomes severely intoxicated, manifesting increasingly poor judgment and impaired reasoning, overconfidence, and slowed reflexes. At depths of 250 to 300 fsw (76–91 msw), auditory and visual hallucinations may occur, along with feelings of impending blackout. Most divers lose consciousness when they reach a depth of 400 fsw (122 msw).

Of note, individual and diurnal variability occur in the depth of onset and severity of symptoms in nitrogen narcosis. Also, some degree of acclimatization allows experienced divers to work more safely at greater depths than can inexperienced divers. Nonetheless, nitrogen narcosis is a major problem for all compressed-air divers at depths greater than 100 fsw (31 msw). This is one of the reasons why it is recommended that sport divers not dive deeper than 100 fsw.

Treatment of nitrogen narcosis simply requires ascent to a shallower depth (usually less than 70 to 100 fsw [21–31 msw]), where symptoms promptly clear. Of course, the condition is prevented by avoiding deep dives. In commercial diving, where there may be good reasons to dive deeper than 100 fsw, the problem is prevented by using heliox.

Oxygen Toxicity

Although oxygen is essential for most life on Earth, it becomes a poison at elevated partial pressures, so oxygen toxicity is another indirect effect of pressure that divers need to understand. Oxygen toxicity in divers can affect either the central nervous or the pulmonary systems.

High inspired P_{O_2} can occur in diving in two ways. Breathing 100% oxygen in underwater or hyperbaric chambers is one. The second results from Dalton's law of partial pressures. If a normal 21%-oxygen gas mixture is breathed at 300 fsw (10 ATA), an inspired P_{O_2} of 2.1 ATA is generated, which is equivalent to breathing 100% oxygen at 36 fsw. Again, it is the partial pressure of a gas that determines its biologic effects.

Pulmonary Oxygen Toxicity

Retrolental fibroplasia in premature infants and pulmonary oxygen toxicity in adults are well-known problems associated with the use of therapeutic oxygen. Pulmonary oxygen toxicity is induced by breathing an above-normal but relatively low P_{O_2} for prolonged periods. The limit for indefinite exposure without demonstrable lung damage is generally considered to be a P_{O_2} of about 0.5 ATA. On a time-dose curve, it is generally considered safe to breathe 100% oxygen at 1 ATA for up to about 20 hours, or at 2 ATA for up to 6 hours. This time can be length-

ened significantly by using intermittent exposures, such as interspersing a 5-minute air break between every 20 minutes of oxygen breathing.[88] At 2.8 to 3 ATA (60 to 66 fsw), at which depth 100% oxygen is used to treat decompression sickness and gas gangrene, pulmonary oxygen toxicity is rarely a problem because CNS manifestations usually intervene before sufficient time elapses to induce pulmonary damage. This subject has been thoroughly reviewed by Clark and Lambertsen.[34] Use of hyperbaric oxygen according to USN Treatment Table 6 (as is used in treating decompression sickness; see Chapter 71) does not produce clinical manifestations of pulmonary oxygen toxicity. However, animal studies with more extreme oxygen exposures have shown a pathologic sequence of alveolar capillary endothelial damage, with increased permeability leading to pulmonary edema and hemorrhage. While species variation in susceptibility occurs, interruption of exposure usually results in reversal of these pathologic changes.[34]

The most common clinical manifestation of pulmonary oxygen toxicity is substernal discomfort on inhalation. If exposure continues, this can progress to severe burning substernal pain and persistent coughing. Reduction of inspired oxygen partial pressure to 0.21 to 0.5 ATA usually results in prompt relief. Severe cases of pulmonary oxygen toxicity may require endotracheal intubation and positive end-expiratory pressure (PEEP) ventilation to achieve adequate arterial oxygenation at the required lower partial pressures of inspired oxygen.

Central Nervous System Oxygen Toxicity

During the 1880s, the French physiologist Paul Bert described convulsions in animals breathing 100% oxygen at elevated chamber pressures. The same phenomena were observed in humans in the 1930s by Behnke and others. Donald's classic observations on divers were published in 1947 and provide much of the current knowledge on predisposing factors and clinical manifestations of CNS oxygen toxicity.[58,59]

In brief, Donald found that at a given duration and pressure of oxygen, a diver in the water was more susceptible to oxygen-induced seizures than was the same diver in a warm, dry hyperbaric chamber. Based on this observation, a limit of 25 fsw (7.6 m) was imposed for military special operations dives using 100% oxygen.

Most people can tolerate breathing 100% oxygen for 30 minutes at rest at 2.8 ATA in a dry chamber, although some manifest toxicity even at this exposure. Common symptoms and signs of CNS poisoning are shown in Box 70-4. Unfortunately, some persons may have no warning symptoms, and the first manifestation of CNS oxygen toxicity may be a generalized convulsion.

Box 70-4. Typical Manifestations of Central Nervous System Oxygen Poisoning

Apprehension
Feeling of air hunger
Sweating
Nausea
Focal muscle twitching
Isolated jerking of a limb
Auditory changes (such as "bells ringing")
Tunnel vision
Diaphragmatic flutter
Convulsion

Treatment for CNS oxygen toxicity in a hyperbaric chamber involves removal of the oxygen mask, maintenance of the airway, and keeping the patient from injuring himself or herself. Of note, chamber pressure should be kept constant until the seizure activity ceases to prevent a possible pulmonary overpressure accident.

After a postictal period, the patient who has had an oxygen-induced seizure recovers without sequelae. Although anticonvulsant drugs theoretically should suppress oxygen-induced seizures, none has been tested to determine efficacy or therapeutic dosage.

Anticonvulsant therapy is not usually considered necessary for persons who have had oxygen-induced seizures because termination of oxygen breathing routinely stops the seizure, and recurrent seizures are exceedingly rare. Removing oxygen at the first sign of CNS toxicity (e.g., when feelings of apprehension are noted or when sweating, nausea, or twitching is observed) has to date prevented any documented permanent CNS damage after an oxygen-induced seizure. Importantly, there is no reason for a person who has had an oxygen-induced seizure not to receive normobaric 100% oxygen if it is medically indicated.

Contaminated Breathing Gas (Carbon Monoxide Poisoning, Hypercarbia)

As breathing-gas cylinders are pressurized or filled, the sea level partial pressure of each gaseous component is multiplied. Thus, any contaminant in the air source can potentially become dangerous to the diver at the elevated pressure found underwater. Compressor motors must be free of oil that could be pumped into tanks; otherwise, the oil mist in the air may cause the diver to suffer lipoid pneumonitis.[105]

Compressed air inlets should always be situated so that they avoid engine exhaust from the compressor, parking lots, or other combustion sources that produce carbon monoxide. Because carbon monoxide is colorless, odorless, and tasteless, the diver cannot detect it unless it is accompanied by other contaminants. The first warning of carbon monoxide poisoning may be headache, nausea, or dizziness during the dive. Examination at the surface may show lethargy, mental dullness, and nonspecific neurologic deficits, which may be confused with those accompanying decompression sickness or air embolism. The cherry-red skin color often mentioned in standard medical texts is actually rarely observed in carbon monoxide poisoning. Fortunately, the treatment of choice for serious acute carbon monoxide poisoning, decompression sickness, and air embolism is hyperbaric oxygen.

Another potential breathing-gas problem involves carbon dioxide. Alveolar partial pressure of carbon dioxide (P_{ACO_2}) reflects arterial P_{CO_2}; thus, even as ambient pressure increases at depth, P_{ACO_2} remains constant at about 40 mm Hg unless environmental or physiologic changes occur. Hypercapnia can occur because of increased P_{CO_2} in the breathing gas or decreased pulmonary ventilation. Unless there is regulator malfunction or contaminated breathing gas, hypercapnia is exceptionally rare in open-circuit scuba diving. Hypercapnia can occur in helmet or chamber diving if these closed spaces are inadequately ventilated or the breathing gas becomes contaminated by carbon dioxide, and in closed-circuit scuba diving (i.e., using a rebreather) if there is failure of the CO_2-absorbent (scrubbing) material. It is believed that hypercarbia was the

Box 70-5. Causes of Unconsciousness in Divers

BREATH-HOLD DIVING
Underwater hypoxemia after hyperventilation before the dive ("shallow-water blackout")
Near-drowning

COMPRESSED GAS EQUIPMENT
Hypoxic breathing gas
Contaminated breathing gas (such as carbon monoxide)
Equipment failure or exhaustion of breathing gas
Near-drowning
Inert gas narcosis
Oxygen toxicity
Pulmonary overpressure accident with arterial gas embolism

REBREATHING EQUIPMENT
Carbon dioxide toxicity
Oxygen poisoning

primary cause of David Shaw's death at Boesmansgat in January 2005.

At sea level, a concentration of 5% to 6% inspired CO_2 leads to dyspnea, increased respiratory rate, and mental confusion. At 10% inspired CO_2, pulse rate and blood pressure may fall to the point that unconsciousness occurs. With prolonged exposure to 12% to 14% inspired CO_2, such that P_{ACO_2} exceeds 150 mm Hg, central respiratory and cardiac depression can be fatal.

Hyperventilation and Shallow Water Blackout

Hypocapnia can result from hyperventilation during or before diving. The well-known symptoms of hyperventilation—dizziness and paresthesias around the mouth and in the distal extremities—have been postulated to cause unconsciousness among divers, but whether this actually occurs is unclear. In contrast, unconsciousness associated with hyperventilation before a breath-hold dive is caused by hypoxia, rather than hypocapnia.

In what is commonly described as *shallow water blackout,* a diver hyperventilates before a dive, lowering alveolar P_{CO_2} to 20 to 30 mm Hg. However, because hemoglobin is nearly saturated with oxygen during normal respiration, there is little gain in arterial P_{O_2} by hyperventilating. In an underwater swim, even in a shallow swimming pool, exercise-induced hypoxia sufficient to cause unconsciousness may occur before arterial P_{CO_2} reaccumulates to provide sufficient stimulus to breathe.[8]

In a deep breath-hold dive, the problem is compounded by the effect of increased pressure. In addition to the initial depression of arterial P_{CO_2} secondary to hyperventilation, elevations in alveolar and arterial P_{O_2} occur. During the dive these serve to suppress the respiratory response to hypercarbia. During descent in the water, $PaCO_2$ increases from the increased pressure, but after oxygen consumption at depth, depressurization on return to the surface causes a dramatic drop in alveolar and arterial P_{CO_2}. Even if P_{ACO_2} rises to the stimulatory breakpoint during ascent, hypoxemia may cause unconsciousness and near drowning. An expansion of Edmonds' commonest causes of unconsciousness in divers is presented in Box 70-5.[65]

DECOMPRESSION SICKNESS

In the mid-19th century, tunnel and bridge workers who labored in caissons pressurized with compressed air were sometimes observed to suffer joint pains, paralysis, and other medical problems after leaving the high-pressure caissons. The condition was not understood and became dubbed "caisson disease" or "compressed air illness."[147] Of course, these early high-pressure workers were experiencing the same symptoms that were later observed in divers and aviators and which we now know to be decompression sickness (DCS).

For many decades caisson disease remained a medical curiosity, but because of its occurrence in increasingly important areas of activity in the 20th century, considerable research has been directed to better understanding its causes and finding effective treatment.

Today, we know that DCS is caused by the formation of bubbles of inert gas (e.g., nitrogen) within both the intravascular and extravascular spaces after a reduction in ambient pressure. This may occur during or after decompression from being underwater or in a caisson or hyperbaric chamber in which pressures are greater than at sea level, or in aviators, astronauts, or hypobaric (high-altitude) chamber workers who travel rapidly from sea level to pressures less than 0.5 ATA. Although DCS is a major concern in commercial divers who breathe heliox, this discussion concentrates on compressed-air diving situations likely to be encountered by recreational scuba divers.

Etiology of Decompression Sickness

To understand the etiology of DCS, one must understand the temporal uptake and elimination of inert gases supplied in the diver's breathing medium. Clearly, if it were possible for a diver to breathe 100% oxygen while underwater, there would be no problems with decompression sickness because oxygen is rapidly metabolized by the body and for all practical purposes does not contribute to bubble formation on ascent from depth. Unfortunately, breathing pure oxygen at increased atmospheric pressure causes CNS toxicity. The need to have an inert gas diluent (e.g., nitrogen) in the diver's breathing medium is the crux of the problem in DCS.

As alveolar air pressure increases at depth, the partial pressures of inspired gases increase. At 99 fsw (4 ATA), the absolute pressure is 3040 mm Hg (see Table 70-1). Seventy-nine percent of this pressure is nitrogen (2400 mm Hg, as compared with 600 mm Hg P_{N_2} at sea level). Accounting for water vapor and carbon dioxide, this results in an alveolar partial pressure of nitrogen (P_{AN_2}) of about 2360 mm Hg. This P_{AN_2} is rapidly reflected across the alveolar-capillary membrane to the arterial blood, where (according to Henry's law) nitrogen becomes physically dissolved in the blood. *Henry's law* states that the amount of gas dissolved in a liquid at any given temperature is a function of the partial pressure of the gas in contact with the liquid and the solubility coefficient of the gas in that particular liquid. As nitrogen-laden blood is presented to tissues at the capillary level, a complex set of variables dictated by perfusion, diffusion, and inert gas solubility results in a family of nitrogen uptake curves similar to the pharmacokinetic drug uptake curves commonly displayed for medications. Thus, tissue nitrogen saturations on any given dive are a function of depth (pressure) and time.

TABLE 70-4. Common Symptoms and Signs of Decompression Sickness

CONDITION	SYMPTOMS	SIGNS
Musculoskeletal decompression sickness, limb bends	Severe joint pain, single or multiple joints involved, paresthesia or dysesthesia about joint, lymphedema (uncommon)	Tenderness, which may be temporarily relieved by local pressure with blood pressure cuff; pain worsened by movement of joint
Neurologic decompression sickness		
Spinal cord	Back pain, girdling abdominal pain, extremity heaviness or weakness, paralysis, paresthesia of extremities, fecal incontinence, urine retention	Hyperesthesia or hypoesthesia, paresis, anal sphincter weakness, loss of bulbocavernosus reflex, urinary bladder distention
Brain	Visual loss, scotomata, headache, dysphasia, confusion	Visual field deficit, spotty motor or sensory deficits, disorientation or mental dullness
Fatigue	Profound generalized heaviness or fatigue	May precede signs of other forms
Cutaneous manifestations	Intense pruritus	No visible signs, mottling, local or generalized hyperemia or marbled skin (cutis marmorata)
Chokes	Dyspnea, substernal pain that is worsened on deep inhalation, nonproductive cough	Cyanosis, tachypnea, tachycardia
Vasomotor decompression sickness (decompression shock)	Weakness, sweating, unconsciousness	Hypotension, tachycardia, pallor, mottling, hemoconcentration, decreased urine output

Clinical Manifestations of Decompression Sickness

DCS is a multisystem disorder caused by a rapid decrease in ambient atmospheric pressure such that inert gas (nitrogen) comes out of solution, causing the formation of bubbles in tissue and venous blood. Conceptually, DCS is the same illness whether it occurs in high-altitude aviators or deep-sea divers, although there are some differences in the symptoms of the disease depending on whether it is caused by hyperbaric or hypobaric exposure.

The physiologic sequelae of bubble formation in tissue and venous blood are myriad. These effects include cellular distention and rupture; mechanical stretching of tendons or ligaments, producing pain; and intravascular or intralymphatic occlusion, resulting in congestive ischemia and infarction or lymphedema.

Intravascular bubbles also cause multiple biophysical effects at the blood–bubble surface interface because bubbles are viewed by the immune system as foreign matter and incite an inflammatory reaction. The key step in the process is activation of Hageman factor, which in turn activates the intrinsic clotting, kinin, and complement systems, producing platelet activation, cellular clumping, lipid embolization, increased vascular permeability, interstitial edema, and microvascular sludging. The overall effect is decreased tissue perfusion and ischemia.

The clinical manifestations of DCS are protean (Table 70-4), with the neurologic and musculoskeletal systems being most often affected. Symptoms of DCS are often categorized into type I and type II, with type I referring to the mild forms of DCS (cutaneous, lymphatic, and musculoskeletal) and type II including the neurologic and other serious forms. Some investigators have advocated use of the term *type III decompression sickness* to refer to combined AGE and DCS with neurologic symptoms.[134] Other numerical classification schemes have also been espoused.

Although the categorization of DCS as types I and II is firmly entrenched in the literature, its use is not advocated. It is clinically more meaningful to refer to the body systems affected when discussing patients with DCS, especially in light of the growing awareness that all DCS must be considered serious and treated vigorously.

Musculoskeletal Decompression Sickness

Musculoskeletal DCS, or *the bends,* is the development of pain in and around the major joints after diving or other hyperbaric exposure. It is the most common manifestation of DCS, occurring in about 70% of patients.[122,147,151] This form of DCS is often referred to as limb bends, joint bends, or pain-only bends.

The term *bends* originated at the beginning of the 20th century when caisson workers on the Brooklyn Bridge who were suffering from DCS of the hips were noted to walk stiffly, bending forward at the hips.[147] Coworkers would describe the stricken men as walking as if they were trying to do the "Grecian bend," a forward bending, stiff-at-the-hips way that stylish women of the day would walk because of their tight corsets. Over time the term became shortened to just "bends."

The shoulders and elbows are the joints most often affected by DCS in scuba divers, but any joint may be involved. Conversely, the hips and knees are most commonly affected in saturation divers, caisson workers, and aviators. The reason for the different anatomic predilection is not known.

The pain of joint bends is usually described as "boring" or a dull ache deep within the joint, although it may also be characterized as sharp or throbbing. It is sometimes described as "tearing" or feeling like tendinitis or bursitis. It may radiate to surrounding areas. Movement of the joint worsens the pain, so the joint is usually held immobile. An area of vague numbness or dysesthesia may surround the affected joint, but this typically does not conform to any anatomic distribution and should not be confused with neurologic involvement. There also may be erythema around the joint, and it may be mildly swollen. The joint may also be tender to touch.

Sometimes divers will complain of having a "niggle" after a dive. "Niggle" is a British colloquialism referring to mild pain in a single joint that improves 10 to 15 minutes after the onset of pain and then disappears without treatment. Whether niggles should be treated with recompression is controversial, but the safest approach is to treat them with at least a USN Treatment Table 5.

Differentiating between limb bends and trauma or other cause of joint pain may be aided by inflation of a sphygmomanometer cuff placed around the joint to 150 to 250 mm Hg.[154] If the pain is due to DCS, inflation of the cuff may immediately relieve the pain by reducing the gas volume in tendons and ligaments. This relief suggests that the mechanism of pain is gas expansion (bubbles) in tendons and ligaments that stretches nerve endings. The pain of limb bends recurs when the cuff is deflated. The test is helpful when it is positive, but a lack of response cannot be used to rule out the presence of DCS; this must be done with a "test of pressure" in a hyperbaric chamber. Importantly, most often there may be neither abnormal physical signs (other than splinting or stiffness from pain) nor abnormal radiographic findings.

Limb bends pain itself is not immediately threatening to life or function but indicates that bubbling may be occurring in venous blood. Often, patients who begin with musculoskeletal DCS and are not treated progress to more serious forms of DCS. In addition, it is impossible to completely exclude subtle neurologic signs in a field setting. Likewise, as discussed later, untreated DCS may lead to osteonecrosis of the major joints.

Fatigue

Profound fatigue that is out of proportion to the activity performed underwater, or otherwise while under increased atmospheric pressure, may be an early manifestation of DCS. Although its etiology is unknown, a feeling of severe fatigue after diving demands careful evaluation for other manifestations of DCS and may in and of itself justify a test of pressure.

Skin Bends or Cutaneous Decompression Sickness

DCS may present a variety of cutaneous manifestations, including scarlatiniform, erysipeloid, or mottled rashes, pruritus, and formication. Occasionally, localized swelling or peau d'orange may result from lymphatic obstruction, and rarely, an entire limb may become edematous.

Skin manifestations are relatively uncommon, and in and of themselves are usually not serious. However, mottling or marbling of the skin *(cutis marmorata)* is often a harbinger of more severe DCS (Fig. 70-12). The exact physiologic basis of the mottled skin lesion is unknown. Skin bends should be easily distinguished from cutaneous barotrauma, "wetsuit dermatitis,"[111,155] marine envenomation, or other skin rashes often seen in divers.

Itches, or "the creeps," is a type of skin bends seen during decompression in hyperbaric chamber workers when the skin is exposed to the high partial pressure of nitrogen in compressed air. This is a highly pruritic skin reaction most intensely felt on body parts exposed to the compressed air. The sensation is often described as feeling like ants crawling over one's body.

In hyperbaric chambers, inert gas from the external environment is absorbed directly into skin, and itches represent bubble formation in the skin during decompression. The concentrations of dissolved gases in ocean water are essentially constant at all depths, so the skin is not exposed to elevated partial pressures of inert gases underwater.

Figure 70-12. Cutis marmorata. Mottling of the skin seen in severe cases of decompression sickness.

Chokes or Pulmonary Decompression Sickness

The *chokes* is an unusual but very serious form of DCS characterized by burning substernal pain (especially on inhalation), cyanosis, dyspnea, and nonproductive cough. Animal studies have demonstrated gas bubbles or foam in the pulmonary arteries, right atrium, and right ventricle after unsafe decompression. Chokes probably represents massive pulmonary gas embolism with mechanical obstruction of the pulmonary vascular bed by bubbles. Typically, symptoms of pulmonary venous air embolization begin when 10% or more of the pulmonary vascular bed is obstructed. Patients with chokes can progress rapidly to profound shock or neurologic DCS. The specific clinical and radiographic manifestations of the chokes are similar to those seen with venous gas embolism (VGE) from other causes.[106]

Symptoms of VGE include air hunger, dyspnea, cough, and chest pain. Findings may include pallor, diaphoresis, tachypnea, tachycardia, hypotension, cyanosis, expiratory wheezing, neurologic signs, and a "mill wheel" heart murmur. Victims may also exhibit increased central venous or pulmonary artery pressure, electrocardiographic changes of ischemia or cor pulmonale, decreased end-tidal carbon dioxide fraction, and precordial Doppler sounds of circulating gas bubbles. Rarely, air may be visualized in the main pulmonary artery on chest radiographs; this is pathognomonic for pulmonary air embolism.[93,106,139]

Neurologic Decompression Sickness

Neurologic impairment may occur as the sole manifestation of DCS or as part of a larger dysbaric syndrome. Neurologic DCS is manifested by myriad symptoms and signs because of the random nature by which DCS affects the nervous system. Although any level of the central nervous system may be affected, the most commonly involved site in divers is the spinal cord, and specifically the lower thoracic and lumbar regions. While much less frequently affected than the CNS, the peripheral nervous system also may be involved.[95]

Based on military experience, neurologic DCS was believed to occur in only 10% to 20% of DCS cases,[151] but neurologic man-

ifestations of DCS have been found in 50% to 60% of scuba-diving casualties treated in Hawaii[69,97] and have been reported in similarly high frequencies in other populations of sport divers.[46,56]

Classically, dysbaric spinal cord injury occurs in the lower thoracic, lumbar, and sacral portions of the cord, producing low back pain, subjective "heaviness" in the legs, paraplegia or paraparesis, lower extremity paresthesia or dysesthesia, and possible bladder or anal sphincter dysfunction. General malaise or fatigue is often noted as well. Involvement of the cervical and thoracic cord may cause chest or abdominal pain and weakness or sensory disturbances in the upper extremities. Absence of the bulbocavernosus reflex, elicited by gently squeezing and pulling the glans penis to seek reflex contraction of the anal sphincter, often foretells a poor prognosis, as does absence of the superficial anal reflex, which can be elicited in the male or female by stroking the perianal region.

The mechanism of spinal cord DCS is multifactorial, involving autochthonous inert gas bubble formation in the cord[77,142] and in the epivertebral venous system (Batson's plexus), with resulting congestive infarction of the spinal cord,[83] and other mechanisms that are not well understood.[89,142]

DCS of the brain produces a variety of symptoms, most of which are indistinguishable from AGE. These include dizziness, vertigo, altered mentation or level of consciousness, generalized weakness, and visual deficits (e.g., diplopia, scotoma, visual field defects, and blindness). Involvement of the cerebellum or inner ear may produce ataxia and loss of balance.

Inner Ear or Vestibular Decompression Sickness

The *staggers* is another classic DCS syndrome. In this case, the inner ear is primarily affected, and the name derives from the unsteady gait that results from the vestibular damage. Manifestations include dizziness, vertigo, nystagmus, tinnitus, nausea, and vomiting. In contrast to inner ear barotrauma, which generally has a favorable prognosis, inner ear DCS carries a high risk for residual inner ear damage despite appropriate treatment.

Inner ear DCS is most often seen in saturation divers when there is a rapid ascent on heliox, or when switching gases on ascent during very deep dives.

Vasomotor Decompression Sickness

Vasomotor DCS, or decompression shock, is a rare, life-threatening form of DCS. The pathogenesis of this shock syndrome is not completely understood, but it is believed to be caused by a rapid shift of fluid from intravascular to extravascular spaces secondary to diffuse bubble embolization, ischemia, and hypoxia.[33] Hypotension may also result from massive venous air embolization of the lungs.

Despite vigorous intravenous fluid replacement, the hypotension of decompression shock may not respond until recompression is undertaken. Unfortunately, the condition is highly lethal, and most patients do not survive to undergo recompression unless a hyperbaric chamber is immediately available.

Long-Term Sequelae of Decompression Sickness

Although DCS is the most overt manifestation of inadequate decompression after diving, it has now been clearly established that there may be long-term sequelae of diving related to inadequate decompression, even if the diver never manifests overt DCS. The best characterized of these problems is dysbaric osteonecrosis.

Dysbaric Osteonecrosis

Dysbaric osteonecrosis (DON) is a form of avascular or aseptic necrosis of bone associated with pressure changes. The major joints (shoulders, elbows, hips, and knees) are most often affected, although any bone can be involved.[32,67,126]

DON was first recognized in compressed-air workers in the early 1900s.[140] Since then, its incidence in professional divers has been found to range from less than 1% to over 80%, depending on the age of the diver and the type of diving done. Its occurrence correlates well with deep diving, decompression diving, the occurrence of DCS, and missed decompression.[52,67,94,126,174] Most diving medicine experts consider DON a long-term sequela to inadequate decompression.

Of interest, fossil evidence of avascular necrosis has been found in marine mosasaurs and plesiosaurs of the Cretaceous period, suggesting that at least some of these extinct giant marine lizards dived deeply.[153] Likewise, although modern marine mammals were previously thought to be immune to decompression sickness, it has been recently found that sperm whales, which dive to depths of over 10,000 fsw (3048 m) and stay underwater for as long as an hour, also suffer from DON.

Dysbaric Retinopathy

Infrequently, DCS affects the eyes, producing a wide array of acute ophthalmic effects, including homonymous hemianopsia, cortical blindness, central retinal artery occlusion, retinal hemorrhage, nystagmus, convergence insufficiency, and optic neuropathy.[26,27] Some long-term ophthalmic findings in divers also have been observed.

A retinal fluorescein angiography survey of asymptomatic divers found a higher incidence of retinal pigment epithelium than in nondivers and various capillary changes at the fovea.[148] The significance of these abnormalities is unclear because none of the divers had visual loss. Similarly, the cause of such changes is unclear, although they are postulated to be the result of small bubble microembolization.

Diagnosis of Decompression Sickness

As with arterial gas embolism, the diagnosis of DCS is a clinical diagnosis based on the history of exposure to increased atmospheric pressure and subsequent development of characteristic symptoms and signs. The majority of patients with DCS become symptomatic in the first hour after surfacing from a dive, with most of the rest noticing symptoms within 6 hours after diving. A very few DCS patients (1% to 2%) may not note their symptoms until 24 to 48 hours after diving. In these latter patients, the symptoms tend to be less specific.

A variety of laboratory abnormalities may be demonstrated in DCS, but most have little or no usefulness in the immediate management of patients. Two tests that may be useful, however, are measurement of the urine specific gravity and hematocrit; intravascular volume depletion and hemoconcentration are common in serious DCS because of increased vascular permeability caused by endothelial damage and release of kinins. The results of these tests can help guide replacement fluid therapy. Hematocrit percentages are commonly in the high 50s or 60s in serious DCS.

As with laboratory tests, radiographic evaluation of patients with suspected DCS may yield various findings, but the radiographs are rarely useful in acute management of the patient. Bone radiographs of patients with acute joint bends do not show abnormalities. Months to years later, they may

demonstrate findings of DON. Noncardiogenic pulmonary edema may be seen on chest radiographs of persons with pulmonary or vasomotor DCS.

Both CT and MRI have been used to evaluate neurologic DCS injury, although conventional CT suffers from poor sensitivity of early lesions and inability to image spinal cord lesions.[91,99] Limited clinical data support the feasibility and efficacy of MRI of these conditions,[116,166,176] especially when intracranial injury is suspected, although the exigency of obtaining recompression treatment makes these modalities useful primarily in the post-recompression evaluation of residual deficits.

Treatment of Decompression Sickness

All persons with suspected DCS must be referred to a hyperbaric treatment facility as quickly as possible because recompression is the primary and essential treatment for this condition. The physician must have a high index of suspicion when diagnosing DCS, because the often diverse manifestations of DCS may present a very confusing clinical picture. The history of the dive profile is helpful if the diver knowingly violated decompression procedures, but DCS may occur on dives that should be safe according to current decompression schedules.[1] In addition, the reported depth and time of the dive are often not accurate.

Management of DCS must be commenced as soon as the condition is suspected.[46] Sport divers are usually far from a recompression chamber when their symptoms develop, so treatment is often initiated in the field or at a general acute care facility. One hundred percent oxygen should be administered by a tight oronasal mask to facilitate off-gassing of nitrogen. Of equal importance is maintenance of intravascular volume to ensure capillary perfusion for elimination of microvascular inert gas bubbles and for tissue oxygenation. An intravenous infusion of isotonic solution should be started and run at a flow rate sufficient to maintain urine output at 1 to 2 mL/kg/hr. With spinal cord involvement, an indwelling urinary catheter may be needed because of sacral nerve root dysfunction. Intractable vomiting or vertigo should be treated with appropriate parenteral agents. Diazepam has been quite effective in providing relief from the vertigo associated with labyrinthine DCS when other agents have failed. Oral aspirin (5 to 10 grains) also may be useful for its antiplatelet activity. Advanced life support measures should be undertaken appropriate to the patient's clinical condition.

While these measures are undertaken, someone should arrange for emergency transportation to the nearest recompression chamber. Because of the large number and frequently changing status of recompression chambers, no list is provided here. Instead, the reader is referred to the Divers Alert Network (DAN) (Fig. 70-13). Help with the treatment of dive-related incidents may be obtained 24 hours a day by calling 919-684-8111 and requesting DAN. This telephone number should be readily available to all divers and medical facilities. DAN's diving medicine experts provide help with diagnosis and immediate care of the patient and provide information about the location of the nearest hyperbaric chamber.

Before a patient is transferred to the hyperbaric treatment facility, it is imperative to contact the chamber to determine its availability. The chamber may be out of service or tied up with the treatment of another patient. The physician should never send a patient without first discussing the transfer with the hyperbaric treatment personnel.

Figure 70-13. Logo for Divers Alert Network. (Courtesy Divers Alert Network.)

If airborne evacuation is required, it is critical to obtain an aircraft that can maintain sea level cabin pressurization during flight. Examples of such aircraft are the military C9 and C-130 Hercules, Learjet, and Cessna Citation. In the case of helicopters (which cannot be pressurized), the crew must maintain the lowest possible flight altitude, and preferably never greater than 1000 ft (305 m) above the starting elevation. This is always problematic in evacuations from mountain lakes. All resuscitative measures must be maintained in flight.

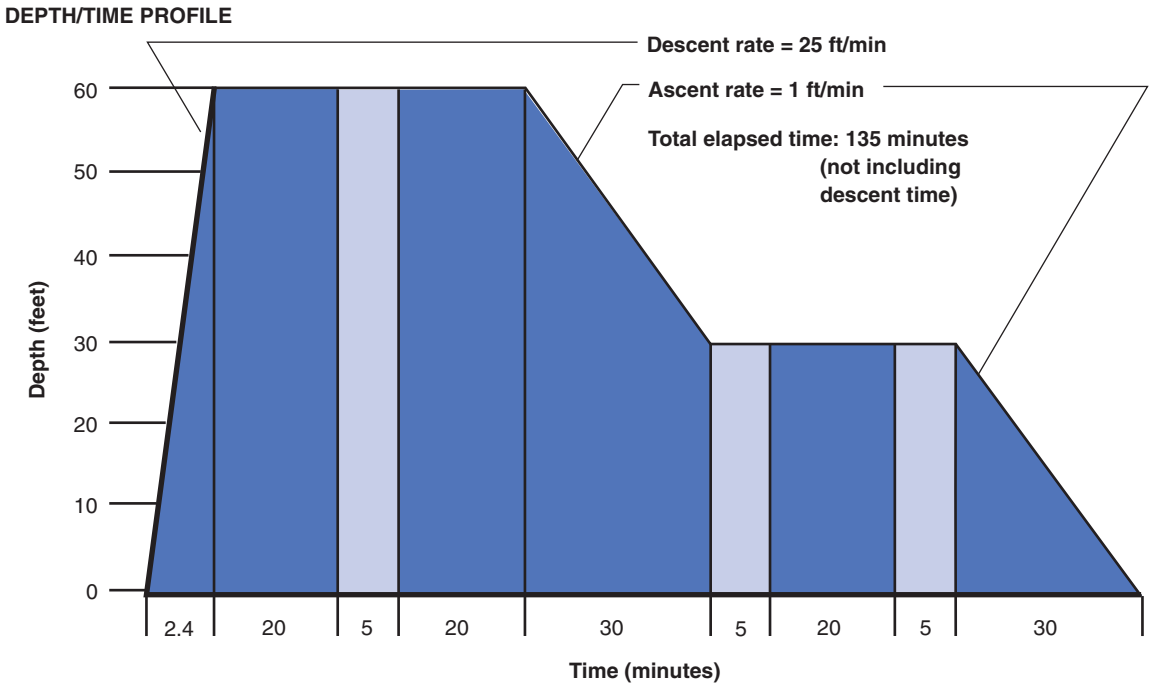
At the recompression chamber, one of several standard hyperbaric treatment protocols is followed. In a multilock compressed air chamber, the patient and an attendant can be pressurized with compressed air and the patient given 100% oxygen by mask.

As with AGE, hyperbaric treatment of decompression sickness has undergone significant evolution over the past two decades and is discussed in more detail in Chapter 71. It is beyond the scope of this chapter to discuss recompression treatment in detail.

Recompression treatment is most often successful, but the likelihood of success is difficult to predict for any given diver. In general, the sooner after the onset of symptoms that treatment begins, the better the outcome,[5] although treatment after delays of many hours, or even days, often results in full functional recovery. In one series of 92 sport scuba divers treated after a significant delay between the offending dive and the start of recompression treatment, 85% had good results when standard USN treatment tables were followed.[46] Similar results were achieved in another series of 50 patients.[102] Such treatment is usually given according to USN Tables 5 and 6 (Fig. 70-14). Any patient with neurologic or pulmonary DCS requires treatment with at least the protocol in Table 6 (see Figure 70-14B), with extension of the hyperbaric oxygen periods depending on how the patient responds.

Monoplace hyperbaric chambers are used to treat decompression sickness and air embolism,[87,103] although the conventional wisdom is to use a multiplace chamber whenever possible because of the free access to the patient that is possible in these larger chambers and the greater flexibility in possible treatment regimens.

USN Treatment Table 7 is an option for serious DCS cases; however, this treatment table should be reserved for patients with major deficits because of its length and commitment of



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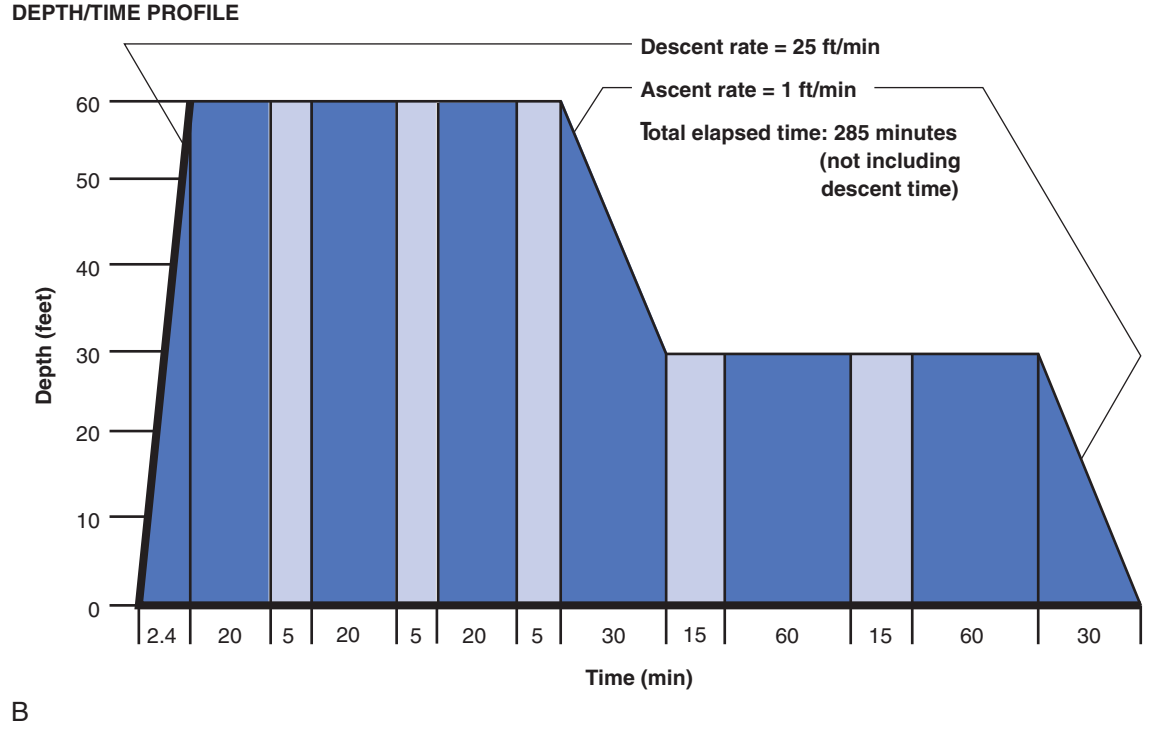

B

Figure 70-14. Examples of U.S. Navy (USN) decompression tables. Dark shading represents oxygen breathing; light shading represents air breathing. **A,** USN Treatment Table 5, oxygen treatment of type-1 decompression sickness. **B,** USN Treatment Table 6, oxygen treatment of type-2 decompression sickness. (Modified from Department of the Navy: U.S. Navy Diving Manual, vol 2, rev 2, Flagstaff, AZ, Best Publishing Company, 1988.)

resources. The diver and attendant are held at 2.8 ATA for at least 12 hours, and longer if needed, with the patient breathing oxygen in 30-minute periods as tolerated. A final, slow, 32-hour decompression follows, regardless of the time spent at 2.8 ATA. Details of Table 7 are found in the *U.S. Navy Diving Manual*.[54]

Another potentially useful recommendation for dealing with the recurrence of neurologic symptoms and signs after apparently successful chamber treatment of neurologic DCS has been advanced by Edmonds.[65] Frequent post-recompression recurrence of neurologic manifestations led him to institute multiple 30-minute sea level oxygen-breathing periods with 30-minute air breaks for 6 to 8 hours during post-treatment hospital observation, rechecking vital capacity frequently to detect and prevent pulmonary oxygen toxicity.

Although experimental proof of their efficacy is lacking, high-dose parenteral corticosteroids were widely recommended and used in the past as an adjunct to recompression treatment of both neurologic DCS and AGE. Rapid-acting hydrocortisone hemisuccinate (1000 mg) or methylprednisolone sodium succinate (125 mg) followed by dexamethasone, 4 to 6 mg every 6 hours, with continuation of the dexamethasone for 72 hours, was the standard regimen.

The use of steroids became prevalent based on the belief that they were beneficial in the treatment of cerebral edema, shock, and other conditions pertinent to DCS, although their benefit in many of these conditions has been questioned. Anecdotal data suggesting that steroids were beneficial in combination with hyperbaric oxygen treatment have been reported,[46,98,113] but there have been no published clinical series or controlled trials demonstrating their efficacy. In contrast, controlled studies of high-dose parenteral dexamethasone and methylprednisolone in DCS-affected dogs showed that the use of glucocorticoids as an adjunct to conventional hyperbaric oxygen therapy produced no benefit, and even suggested that the steroid-treated animals did less well.[76] Thus, the benefit and role of steroids in the treatment of DCS continue to be a matter of controversy, and they are much less often used today than in the past.

Because intravenous bubbles can induce platelet adherence, antiplatelet agents, such as aspirin and nonsteroidal anti-inflammatory drugs (NSAIDs), have been tried both prophylactically and therapeutically without success. Although the use of tenoxicam, an NSAID, has been shown to decrease the number of treatments required to treat DCS, the outcomes of the divers in the control and treatment groups were unchanged.[12] Therefore, NSAIDs and aspirin are not routinely recommended as adjuncts in the treatment of DCS, although it is recognized that these drugs, and especially aspirin, are commonly used. Of note, divers who are paralyzed from DCS should be treated to prevent thromboembolic disease (e.g., with subcutaneous low–molecular-weight heparin).

Because the outcome of recompression treatment is influenced by the time interval from onset of symptoms to the initiation of treatment, every emergency department and every diver should know the location of the nearest recompression chamber from their dive site. Spinal cord decompression sickness treated after a few hours delay may result in residual deficits ranging from mild weakness or sensory changes to permanent paraplegia or worse. On the other hand, dramatic improvements have been witnessed even in persons arriving at a chamber many days after the insult. It is not possible to predict what the outcome of treatment will be for any individual patient.

Prevention of Decompression Sickness

Ever since DCS was first recognized, efforts have been directed at preventing its occurrence. Such efforts have utilized all manner of intervention, but to date the only proven way to prevent DCS is to limit the time a diver spends at increased pressure (depth) and to ensure that decompression from increased pressure is slow enough, or staged, so that the body's burden of excess inert gas is eliminated without forming bubbles. Such depth/time ascent schedules have given rise to a variety of decompression tables.

Two very recently reported studies suggest that a single bout of moderately intense aerobic exercise within 24 hours of diving significantly decreased ultrasonic detection of bubbles in the pulmonary artery after diving.[16a,60a] The mechanism of this possible protective effect of predive exercise is unclear but may be due to a reduction of the number of circulating gaseous micronuclei from which bubbles form.

Decompression Tables

Navies and commercial diving companies around the world have developed various decompression schedules based on theoretical calculations and actual testing (animal and human) that allow divers to avoid exceeding safe rates of decompression after specified depth-time dive profiles. These listings of "safe" depth-time diving profiles are generally referred to as decompression tables. Many different sets of decompression tables exist, with the USN Standard Air Decompression Tables being the most widely used.[54] Decompression tables continue to be the subject of considerable scientific controversy, however, resulting in periodic revision and continual search for improved safety.

Over the years, the USN Standard Air Decompression Tables have been the ones most often used in recreational diving. Various rearrangements or modifications of these tables have been promulgated by sport diving groups in recent years in efforts to improve the safety of recreational diving, which is qualitatively different from military diving (for which the USN decompression tables were developed). In general, these sport-diving modifications have involved applying various "safety factors" to the standard USN protocols.

Recent "multilevel" dive tables also have been developed to address the depth fluctuations that typically occur in sport diving, because the diver ranges from shallow to deep repetitively during the course of a dive. These multilevel tables give the diver decompression "credit" for the time spent at shallower depths, as opposed to the Standard USN tables, which require the diver to use the maximum depth of the dive to select no-decompression limits or decompression schedules and repetitive-dive group designations. The reason for doing this has been the belief that the USN tables are too restrictive and excessively limit the diver's time underwater. Multilevel tables allow a diver to spend more time underwater, although there continues to be concern that they provide less safety margin.

Dive Computers

Automatic decompression meters that measure and record time and pressure underwater give the diver an indication of decompression status.[167] These dive computers track the exact dive profile and then calculate a decompression requirement according to the actual depth-time dive profile.

Over the years, these devices have used different physiologic and decompression models and technologies. Early automated

decompression meters presented problems because their use was associated with an unacceptable rate of DCS. More recent technologic innovations, however, have overcome many of the earlier difficulties. The most recent generation of dive computers is considerably more advanced than the initial devices.[92,117] Today, the sport scuba diver has access to a variety of sophisticated dive computers that offer the convenience of automatic and accurate depth-time recording, together with accurately computed multilevel decompression schedules.

Currently available dive computers typically use microprocessors with decompression tables stored in memory. The microprocessor in the dive computer quickly reads a pressure transducer (which converts pressure into electrical impulses) and applies nitrogen uptake and elimination algorithms to this information every few seconds. The computer tracks a diver's exact profile and calculates decompression requirements accordingly.

Use of dive computers has become the norm, and when used correctly, today's dive computers appear to be quite safe. Dive computers have made diving easier and have eliminated the clerical aspects of diving (i.e., the need to write down the depth and time of all dives, consult the decompression tables, calculate and record residual nitrogen time and surface intervals, and so on). Dive computers also offer advantages for some types of diving over use of the USN Standard Decompression Tables. They are especially useful in multilevel diving because they give bottom-time credit for time spent underwater at depths shallower than the deepest depth attained. However, it must be remembered that dive computers calculate decompression status based on models designed to simulate nitrogen uptake and release in a diver's body. These models may not accurately predict gas flow into and out of a given diver's tissues. Human physiology is not always as predictable as the computer model, and many factors can influence the actual rate of inert gas uptake and elimination. Also, not all computers have built-in safety margins to compensate for the difference between predicted and actual gas uptake and elimination, in contrast to decompression tables, which usually contain an inherent safety margin and require "rounding up" intermediate depths and times, providing a further safety factor.

Because of the multiple-tissue compartment modeling that dive computers use, another concern with their use is that they integrate the entire depth-time profile, so that in cases of repetitive diving (multiple dives per day, or multiple successive days of diving), dive computers would be more liberal than the USN tables, allowing more time under water and potentially allowing a gradual unsafe accumulation of nitrogen in slow tissues. In such settings the risk of developing DCS may be greater.

Other notable concerns associated with the use of dive computers are their allowance of multiple reverse-profile dives (shallow, then deep dives—an inherently risky way to dive), risk of equipment failure (e.g., battery failure or flooding of the battery compartment), loss of information by inadvertently turning off the computer, or failure to turn on the computer at the beginning of a dive. In the last three scenarios, the safety of the dives done after the one in which the problem occurred would be jeopardized.

The biggest problem with dive computers, however, is that these devices are too often not used as intended or not adhered to. Of course, they are of little benefit if not used properly.

Despite the widespread use of dive computers in recent years, it is not possible, at this time, to say whether they are more or less safe than the USN Standard Decompression Tables when the tables are actually followed. The fact that substantial numbers of divers treated for DCS in recent years (more than half of diving casualties treated at some recompression chambers) were using dive computers underscores that they do not inherently protect a diver from getting the bends.[57,124,125]

Decompression Sickness in Breath-Hold Divers

Conventional wisdom has always held that decompression sickness does not occur in breath-hold divers because they do not breathe air at increased atmospheric pressure. However, it appears that neurologic DCS does occur among professional breath-hold divers in some settings.

A condition known as *taravana* was first described among pearl divers in the Tuamotu Islands of the South Pacific by Cross in the early 1960s.[41,42] These breath-hold divers make 40 to 60 dives per day to depths of 80 to 100 fsw (24 to 30 m), spending about 2 minutes underwater each dive and remaining on the surface for 3 to 4 minutes between dives. These divers often complain of suffering vertigo, nausea, euphoria, numbness, and partial or complete paralysis of the extremities after several hours of diving. In some cases the diver loses consciousness for seconds to minutes after noting symptoms. Many of these divers have died either suddenly or after developing other symptoms. Nonfatal symptoms are usually transient, resolving in minutes to a few weeks. The symptoms immediately resolve with recompression.[145]

The symptoms of the taravana diving syndrome are similar to what has been more recently reported as occurring among some Japanese ama divers, who have been reported to suffer dizziness, nausea, euphoria, numbness and weakness of the extremities, dysarthria, and loss of consciousness after several hours of making 20 to 40 dives per hour to depths of 30 to 90 fsw (9 to 27 m).[108,109]

With both the Tuamotu pearl divers and the Japanese ama, as well as in one instance of a USN diver making repetitive breath-hold dives to depths of over 100 ffw in a submarine escape tank training tower, only neurologic symptoms have been observed. Joint pain and skin signs that are often seen in DCS have not been observed.

The mechanism of CNS damage after repetitive breath-hold diving is poorly understood. Venous gas bubbles were described in an ama diver after making 30 dives to 45 to50 fsw in less than an hour.[150] It is possible that inert gas accumulates in the peripheral tissues of these divers consequent to the repeated back-to-back exposure to elevated atmospheric pressure; then gas bubbles are released from the peripheral fatty tissues, bypass the usual lung filtering mechanism via pulmonary arteriovenous or cardiac intra-atrial shunts, enter the systemic circulation and cause cerebral embolization. Nothing has been reported about the prevalence of patent foramen ovale in these populations, but there is no reason to believe that it is less than in other populations where it has been studied (i.e., about 25% of people). It is also possible that intravascular microbubbles (i.e., bubbles <21 μm in diameter), that would pass through the lungs[24,25] could impair the blood–brain barrier transiently,[90] although in and of itself, this seems unlikely to explain the cerebral lesions that have been demonstrated by MRI.[108] Whatever the mechanism, the symptoms and neurologic lesions that these divers suffer after repetitive breath-hold dives are now well documented.

LONG-TERM HEALTH EFFECTS OF DIVING

Long-term untoward health effects related to diving may result from CNS injury due to AGE or DCS (e.g., motor paresis or paralysis; paresthesias and other sensory disturbances; bladder, bowel, or sexual dysfunction; seizures); hearing loss; vertigo, tinnitus, and balance disorders from inner ear injury; dysbaric osteonecrosis; dysbaric retinopathy; hearing loss; and possible loss of intellect and personality changes.

A number of informal observations have been made over the years suggesting that long-time divers suffer some loss of intellect and may experience significant changes in personality, although confounding variables (e.g., use of alcohol) have always complicated such observations. However, there is now increasing evidence that divers may well suffer subclinical CNS injury from paradoxical gas embolization and possibly other mechanisms.[174a] For example, long-time divers have been found to have more subcortical cerebral lesions than controls in MRI studies. Overall, there is growing evidence that long-time divers suffer untoward health effects related to repeated exposures to a hyperbaric environment and the breathing of compressed gas.

UNUSUAL POST-DIVING CONDITIONS OF UNCERTAIN ETIOLOGY

Immersion Pulmonary Edema

Acute pulmonary edema has been associated with both scuba diving and swimming. Several case reports describe scuba divers developing pulmonary edema during a dive.[85,149,162,185] In the majority of these cases, the divers are healthy with no predisposing factors for the development of pulmonary edema (e.g., no evidence of cardiac disease or hypertension). Other reports describe pulmonary edema associated with heavy exertion in surface swimmers.[118,121,160,178] Many of these reports are of healthy males in military fitness training programs who presented with dyspnea, cough, tachypnea, hypoxemia, and hemoptysis after a strenuous swim.

The exact mechanism for stress failure of the pulmonary capillaries is unknown. Race horses are known to develop extremely high pulmonary vascular pressures leading to stress failure of the capillaries.[179] The physiologic changes that occur with immersion most likely contribute to the overall pathophysiology. Immersion causes central pooling of blood, which increases preload. Decreased core body temperature from cold water causes redistribution of blood flow to thoracic vessels and vasoconstriction. Increased sympathetic outflow also leads to vasoconstriction. Hence, immersion causes increased cardiac output and increased pulmonary vascular resistance. Reduction occurs in functional residual capacity and vital capacity by blood displacement into the lungs, and there is also reduction in lung compliance.

Increased pulmonary blood flow and vascular pressure most likely cause stress failure of the pulmonary capillaries. In scuba divers, use of a regulator with a high inspiratory resistance could lead to negative-pressure pulmonary edema.

Radiographic findings include Kerley-B lines, cephalization of flow, airspace consolidation, and normal cardiac size (Fig. 70-15). Treatment is conservative because hypoxemia usually resolves within 12 hours. Treatment includes supplemental oxygen and inhaled beta-2 agonists for symptomatic relief. Diuretics are often given; however, most patients improve dramatically within the first 12 hours without their use. Older divers with cardiac risk factors need to be evaluated for a cardiac event that may have precipitated acute pulmonary edema.

Figure 70-15. Immersion pulmonary edema. Chest radiograph of a diver with immersion pulmonary edema showing bilateral patchy opacities consistent with pulmonary edema after an uneventful dive.

There is no way to predict whether or not a scuba diver will develop acute pulmonary edema. Many of the divers in the case reports have returned to diving without any recurrence of symptoms. No predisposing factors for recurrence have been identified, except for possibly low lung volumes.[160]

Internal Carotid Artery Dissection

Dissection of the internal carotid artery (ICA) and associated thromboembolic stroke has been reported rarely after scuba diving, in which cases it has been initially incorrectly diagnosed as arterial gas embolism.[133] Although conceivably related to intravascular bubble-induced endothelial injury, its etiology is a mystery.

ICA dissection has been reported after a variety of situations involving neck trauma, hyperextension of the neck in athletic activities, and invasive diagnostic procedures, as well as spontaneously in persons having connective tissue disorders (e.g., Marfan syndrome, fibromuscular dysplasia, and cystic medial necrosis), although its etiology is unknown in most cases.

Box 70-6. Absolute Contraindications for Diving

Spontaneous pneumothorax
Acute asthma with abnormal pulmonary function
Cystic or cavitary disease of the lungs
Obstructive or restrictive lung disease
Epilepsy or seizure disorder
Atrial septal defect (ASD)
Symptomatic coronary artery disease
Chronic perforated tympanic membrane
Chronic inability to equalize sinus and/or middle ear
Intraorbital gas
Pregnancy
Sickle cell disease
Ménière's disease

Symptoms of ICA dissection include unilateral headache, with or without ipsilateral neck pain, and other neurologic symptoms of intracranial origin. In all published cases involving divers, the condition was diagnosed after hyperbaric treatment for presumed AGE or DCS produced no improvement in symptoms or the condition worsened after hyperbaric oxygen treatment. The preferred diagnostic modality is magnetic resonance or conventional arteriography, although ultrasound may be helpful as well.

Primary treatment of this condition entails anticoagulation aimed at preventing further thromboembolic complications. Most patients regain full function if appropriately treated.

MEDICAL FITNESS FOR DIVING

General Considerations

Persons who want to take up scuba diving should first be cleared medically. The diving examination should focus on the pulmonary, otolaryngologic, cardiac, neurologic, and integumentary systems, as well as the person's psychological stability.

Many medical conditions are contraindications for diving because of the changes in pressure that occur with excursions underwater; the physical and sometimes psychological stress of diving; the potential for nitrogen narcosis, altered sensory stimulation, and other factors to interact with pharmaceuticals; and the inherent nature of being underwater (Box 70-6).[47,48] In general, these can be divided into five categories. Persons falling into any of these categories are at increased risk for a diving-related problem:

1. Persons who are unable to equalize pressure in one or more of the body's air spaces are at increased risk for barotrauma.
2. Persons who have a medical or psychiatric condition that may become manifest underwater or at a remote diving site and endanger the diver's life because of the condition itself, because it occurs in the water, or because inadequate medical help is available.
3. Persons who have impaired tissue perfusion or diffusion of inert gases and thus have an increased risk of DCS.
4. Persons who are in poor physical condition and thus at increased risk of DCS or exertion-related medical problems. The factors compromising physical condition may be physiologic or pharmacologic.
5. Women who are pregnant, because the fetus may be at increased risk of dysbaric injury.

Specific Conditions of Concern with Regard to Diving

Neurologic Disorders

Seizures. A history of epilepsy or other seizure disorder is usually an absolute contraindication for diving. Seizures occurring underwater almost always lead to drowning. Even if seizures are well controlled on medication, the individual should be disqualified from diving. However, once an individual is off antiseizure medication for 5 years and has a normal neurologic workup (including an electroencephalogram [EEG]), then the person may return to diving since their risk of having another seizure is the same as the general population. A history of febrile seizures in childhood, seizures due to sepsis or drug ingestion, or post-traumatic seizures of childhood are not disqualifying for diving as long as there is no ongoing seizure problem.

Head Injury. Any significant head injury that increases the risk of late seizures is a contraindication for diving. Such injuries include a significant brain contusion, subdural hematoma, skull fracture, or loss of consciousness or amnesia for greater than 24 hours. In a case of minor head injury that does not have any associated symptoms and that does not require anticonvulsant medication, scuba diving can be considered after 6 weeks.[2]

Unexplained Syncope. Unexplained loss of consciousness should be viewed as a contraindication for diving, much the same way as seizures.

Pulmonary Disorders

Pneumothorax. A history of a spontaneous pneumothorax is an absolute contraindication for diving. Individuals with a history of spontaneous pneumothorax are often found to have small blebs on the surface of the lungs that may be at increased risk for rupture while diving, causing pulmonary barotrauma and AGE. A history of previous spontaneous pneumothorax carries a significant incidence of recurrence, even after many years, and the candidate must be advised against compressed-gas diving. A pneumothorax that occurs while the diver is underwater or in a recompression chamber can rapidly progress to become a life-threatening tension pneumothorax as the pleural cavity air expands (per Boyle's law) during ascent. A history of a traumatic or iatrogenic pneumothorax is not a contraindication for diving. In these cases, inspiratory/expiratory high resolution chest CT scan can rule out significant air trapping.

Asthma. Asthma used to be considered an absolute contraindication for diving due to the asthmatic's increased risk for air trapping and pulmonary overinflation while diving. However, many persons with asthma dive with no apparent increased risk of pulmonary barotrauma.[138] Criteria for clearing a person with a history of asthma to dive include:

1. Asymptomatic adult with a past history of childhood asthma
2. Well-controlled asthma with known triggers
3. Normal pulmonary function tests (with or without medication) with a reduction of less than 20% in peak mid expiratory flow after exercise
4. No evidence of cold-induced wheezing or exercise-induced bronchospasm

A more complete discussion of the issues relating to asthma and diving can be found in Elliott.[68]

Bullous Lung Disease. Pulmonary blebs or cysts can trap air and lead to local pulmonary overpressure accidents during decompression. If a ball-valve or flutter-valve effect allows such a bleb or cyst to equalize with the elevated breathing pressure during compression or descent, but blocks the escape of air during decompression, rupture could cause pulmonary barotrauma and air embolism. Similarly, individuals with significant obstructive lung disease should be disqualified from diving.

Cardiovascular Disorders

Atrial Septal Defect. An atrial septal defect (ASD) allows right-to-left intracardiac shunting of blood and increases the risk for paradoxical embolization of bubbles[184]; hence, the presence of an ASD is an absolute contraindication for diving. A ventricular septal defect does not allow right-to-left shunting and is not a contraindication or diving.

Patent Foramen Ovale. A patent foramen ovale (PFO) has the potential for a right-to-left shunt, and several studies suggest that a PFO may increase risk for serious DCS.[79,131,157,170,173,183] However, the overall risk of DCS in divers with a PFO is very small and routine echocardiogram screening for a PFO is not indicated.[20] It is reasonable to look for a PFO in cases of neurologic DCS, because the risk of right-to-left shunting increases with the size of the PFO, and some recent data suggest a significantly higher risk of recurrent serious DCS in persons having a larger (grade 2 or 3) PFO.[170]

Coronary Artery Disease. As scuba diving has become more popular, the diving population has become older, and more deaths from coronary artery disease are occurring in the water while scuba diving. In addition to the need for cardiac reserve during an in-water emergency, carrying tanks, donning equipment, and swimming against current entail significant physical stresses. Symptomatic coronary artery disease is a contraindication for diving. However, a history of myocardial infarction is not a disqualification for sport diving per se, particularly after revascularization or comparable procedures, and the diving candidate is able to achieve 13 METs on an exercise stress test.

Dysrhythmias. Benign arrhythmias that do not interfere with exercise tolerance are not disqualifying. Individuals with well-controlled atrial fibrillation on anticoagulation should be able to scuba dive without undue risk.

Hypertension. Whether a hypertensive person may safely dive is based largely on the therapy required for blood pressure control and the presence of any other contraindications that are often associated with hypertension (e.g., coronary artery disease or diabetes). Diving has little effect on blood pressure, and when a regimen of weight control, diet, and mild diuretics is successful, diving usually can be allowed. If more potent antihypertensive medications are needed, diving may be contraindicated, depending on the medication.

Ear, Nose, and Throat Disorders. Any condition that inhibits or precludes the ability to clear the ears or sinuses may be disqualifying for diving. Likewise, any condition that impairs eustachian tube function may preclude an individual from diving; such conditions include frequent ear infections, history of middle ear surgery, mastoiditis, and cholesteatoma. A chronic perforated tympanic membrane is a contraindication for diving. A healed or repaired tympanic membrane perforation is usually not a problem for diving. Persons who have had a stapedectomy or stapedotomy should not dive due to the increased risk of oval window fistula and inner ear barotrauma. Chronic paranasal sinus disease or polyps, allergic rhinitis, or nasal septal deviation may make clearing the sinuses difficult. Individuals with inner ear disease with recurrent vertigo, such as Ménière's disease, should not dive.

Diabetes. Like asthma, insulin-dependent diabetes mellitus (IDDM; aka Type 1 diabetes mellitus) previously was considered a contraindication for diving because of the risk of a hypoglycemic reaction underwater leading to incapacitation, unconsciousness, and drowning. Hypoglycemic reactions may result from sudden bursts of energy expenditure, as may occur in dealing with emergencies. Underwater incapacitation due to hypoglycemia not only endangers the life of the individual but may also risk the lives of other persons during rescue attempts and can lead to drowning. However, many patients with IDDM have been diving safely for years, and guidelines for divers with IDDM exist through the American Diabetes Association and the YMCA. Diabetics who are well controlled on insulin, used to vigorous physical exercise, knowledgeable about their condition, and who dive with prepared buddies usually can be cleared for diving.

Pregnancy. Currently, there is near unanimity that a woman who is pregnant, or who may be pregnant, should suspend compressed-air diving until after delivery. Animal studies have produced conflicting results in different species and laboratories, but the possibility of bubble formation in fetal or placental tissues leading to fetal demise or malformation is a concern, even on a dive that is safe for the mother.

Sickle-Cell Disease or Trait. The chances that a sport diver will breathe a hypoxic gas mixture are remote, but possible. Other concerns are heavy exertion in cold water or local compromise of microvascular blood flow by bubble evolution during decompression, which could lead to sickling and a vicious cycle of hypoxia and further sickling.

Migraine Headaches. Scintillating scotomatas, paresthesias, weakness, and other neurologic symptoms associated with migraines and other types of vascular headaches may be confused for symptoms of AGE or DCS if they occur soon after diving. If such symptoms are misinterpreted, it may lead to unnecessary medical evacuation and recompression treatment. Depending on the specific situation, migraine headaches may be a contraindication to diving.

Panic Disorders. A person prone to panic attacks may have such an attack underwater, prompting the diver to make a rapid uncontrolled ascent that may precipitate pulmonary barotrauma and AGE. The majority of dive accidents usually result from human error, not medical problems. Any condition that can lead to poor judgment and panic underwater should be disqualifying for diving.

Abdominal Hernias. This would rarely be a problem, but there is a potential risk of trapping expanding gas in a herniated loop of bowel during ascent. In general, diving should be suspended until surgical repair is completed.

Poor Physical Condition. Sport scuba diving is deceptively easy until an emergency occurs that requires swimming against a current, rescue of a buddy diver, or other vigorous activity. The diver should be capable of performing sudden strenuous activity before entering the water. Regular swimming or other exercise programs to ensure cardiovascular fitness are encouraged.

MEDICATIONS AND DIVING

Many recreational divers today have underlying conditions that require the regular use of medication, although few drugs have been assessed for their effects or effectiveness in a hyperbaric environment or under actual diving conditions. This is of concern since increased atmospheric pressure causes a number of physiologic changes that may affect the action of medications. For example, increased atmospheric pressure alters the configuration of cellular membranes, slows conduction velocity in peripheral nerves and cardiac tissues, and depresses neuromuscular transmission.[60,62,112,175] Although these changes are most notable at very high pressures (>10 ATA), some effects have been detected at the depths routinely frequented by recreational scuba divers. It is unclear how even minor physiologic changes might affect the behavior of drugs in a diver who may be anxious, vigorously exercising, dehydrated, cold, suffering from nitrogen narcosis, or receiving altered sensory stimulation underwater.

Many drugs, including nonprescription medications, have undesirable side effects that may be intensified by increased partial pressures of breathing gases. For example, dimenhydrinate, which may be used to prevent motion sickness, has a sedating effect that potentiates the decreased mental performance caused by nitrogen narcosis.[169] Likewise, some of the antituberculous drugs (e.g., ethambutal, isoniazid, rifampin) are thought to lower the threshold for oxygen convulsions.[175] An unusual situation in this regard, but which is certainly plausible with today's scuba diving population, involves persons who have been treated with bleomycin (e.g., for lung cancer). The concern in this case is that bleomycin sensitizes the lungs to oxygen toxicity. Scuba divers diving to depths of 60 to 120 fsw on compressed air would be exposed to the equivalent of 0.63 to 1.05 ATA oxygen, which is sufficient to cause concern, because there is no known minimal safe oxygen exposure in bleomycin-treated patients.[96,186]

Another potential problematic scenario is where side effects of drugs may be confused with a dysbaric condition. For example, mefloquine used for malaria prophylaxis (which is recommended when traveling to many diving destinations) is known to cause vertigo, nausea, dizziness, anxiety, hallucinations, mania, and seizures. Depending on the specific circumstances, the side effects of the drug could be confused with decompression sickness or AGE. Another example of this is anisocoria and visual disturbances from direct ocular contamination with scopolamine from touching antimotion sickness patches.[123,152] In this case, though, the presence of the patch, the history of how it was handled, and the lack of other symptoms usually make clear the cause of the cycloplegia.

Another concern in this regard is the increased blood–brain barrier permeability caused by dysbaric conditions. Drugs that would not otherwise penetrate the blood–brain barrier could do so in a diver suffering AGE or cerebral DCS and possibly cause unpredictable effects.

The side effects of some drugs may be especially problematic for divers because of the environment of diving. For example, divers in tropical locations may be exposed to intense sunlight, increasing the risk of phototoxic reactions—a recognized side effect of many commonly prescribed medications. Similarly, beta blockers used to treat hypertension or various other conditions may be problematic because they impair the exercise capacity that a diver may need in emergency situations.

A few drugs have been studied under hyperbaric conditions and found to be at least relatively safe for divers; these include pseudoephedrine,[22,168] transdermal scopolamine,[16,180] and the antihistamine clemastine.[161] Other drugs that appear to be safe by virtue of their extensive use by divers and the absence of reported untoward effects include aspirin, acetaminophen, warfarin, chlorpheniramine, and oxymetazoline.

Given the unknowns about the behavior of most medications in the diving environment, the following precautions are appropriate:

1. Whenever possible, avoid all drugs when diving.
2. Any medication that impairs mental judgment or physical capacity should be very carefully considered before being used when diving. If used, the expected benefits of its use should outweigh its risks.
3. Never take a drug for the first time before diving.
4. Always consider whether the reason for taking a drug is reason itself not to dive.
5. Always consider the known side effects of a drug and whether those would be a problem when diving.

DIVING WITH DISABILITIES

In recent years, many persons with limb amputations or other serious orthopedic impairment, spinal cord injury, cerebral palsy, or similar physical condition have sought to participate in scuba diving. Some of these persons were accomplished athletes or divers before an accident changed their physical status. For others, scuba diving is a completely new experience, being offered as part of a rehabilitation program. Whatever the case, it is clear that persons having "disabilities" can enjoy diving as much as anyone else if they are properly trained, understand their condition and the limitations it causes them, and make appropriate adjustments, when necessary, for their condition.[53] Indeed, when done in this context, scuba diving can open exciting new vistas for some disabled persons. Williamson and coworkers[181] demonstrated significant improvement in self-concept and body image among a group of young people with disabilities ranging from post-head injury brain damage, congenital deafness, blindness, and spinal cord dysfunction to major limb amputations. They were examined according to standard diving medicine practice regarding pulmonary and ear, nose, and throat status and detailed psychological testing. Motivation proved to be an important predictor of success. The subjects were given extensive scuba-diving training with at least one-on-one instructor attention.

In any of these situations, the diver and his or her buddy need to fully understand the concerns attendant to his or her condition (e.g., autonomic dysreflexia, skin breakdown, personal hygiene, and a possible increased risk of DCS) and the wisdom of taking a conservative no-decompression approach to diving.

FLYING AFTER DIVING

Diving is often done at remote destinations, and many divers must fly thousands of miles to return home after multiple days of diving several times a day. The question of when it is safe to fly after diving comes up often. Flying too soon after diving can seriously jeopardize decompression safety, leading to development of DCS during or after the flight because of the reduced atmospheric pressure present in most commercial aircraft.

The normal commercial aircraft cabin pressure is equivalent to an altitude exposure of about 5000 to 8000 ft (about 1500 to 2500 m), which is sufficient pressure reduction to cause dissolved nitrogen to come out of solution and form intravascular bubbles. Depending on the age and maintenance of the aircraft, the internal cabin pressure may be equivalent to an even higher altitude. The Divers Alert Network reports that 5% to 7% of divers with DCS contacting them in recent years reported flying before the onset of their symptoms. The actual significance of these statistics is uncertain, because the total number of divers who flew after diving is unknown, but the total is estimated to be very large compared with the number who developed DCS.

With the continued growth of the dive-travel vacation industry, the issue of when it is safe to fly after diving has become even more important. Unfortunately, limited experimental and experiential data make it difficult to precisely quantitate the risks or to establish precise surface intervals for various types of diving profiles. However, this matter has been the subject of a number of workshops sponsored by the DAN, the Undersea and Hyperbaric Medical Society, and other concerned groups.[158,159,172]

Currently, the generally accepted guidelines for flying in commercial aircraft after recreational diving are:

1. For single dives within the no-decompression limits, a minimum preflight surface interval of 12 hours is suggested.
2. For multiple dives per day or multiple days of diving, a minimum preflight surface interval of 18 hours is suggested.
3. For dives requiring decompression stops, there is insufficient experimental or experiential evidence on which to base a recommendation. However, it would seem prudent that such divers should exercise caution, and a preflight surface interval substantially longer than 18 hours seems warranted. The longer the interval between diving and flying, the less likely the risk of DCS.

Having noted the above, it is important to emphasize that no rule about flying after diving can be guaranteed to prevent DCS. These are guidelines that represent the best estimate of a safe surface interval for the majority of sport scuba divers.

SAFE SCUBA DIVING

Sport scuba divers are fortunate that the most colorful marine life and abundant natural light exist at shallow depths. This obviates the need to dive deep. Indeed, 100 fsw should be considered a deep dive for sport diving and, for all intents and purposes, the maximum depth for recreational diving.

Recreational diving is usually done hours to days away from the nearest recompression chamber, so the occurrence of DCS or AGE usually necessitates a major effort to evacuate an afflicted diver to the chamber. This often requires the use of special aircraft. Unfortunately, the delay between symptom onset and treatment may cause the damage to be poorly responsive to hyperbaric treatment. Therefore, divers should do their utmost to avoid developing AGE or DCS.

The need to take a conservative approach to depth and time is even clearer when one considers that individual variability in DCS susceptibility, workload during the dive, water temperature, and postdive exercise or altitude exposure may confound any set of decompression tables or dive computers. Indeed, the potentially devastating consequences of DCS, even with the most vigorous hyperbaric treatment, mandate that divers always dive with the prevention of this disease foremost in their minds.

Adherence to the following safe diving guidelines should help recreational divers remain safe:

- Dive within the limits of "no-decompression" ("no-D") tables. When a decompression computer is used, monitor it closely and do not encroach upon the limits of no-decompression diving.
- When using the decompression tables, always use the next greater depth to determine the no-D limits, then "jump" tables. For example, a 37-minute dive with maximum depth of 68 ft is considered a 70-ft dive, and the no-decompression limit should be determined for the next jump, or in this case, that of an 80-ft dive for 40 minutes. Ascent toward the surface at a rate no faster than 1 ft per second should begin before the end of the prescribed limit (40 minutes in this example), regardless of how much time was actually spent at shallower depths. This penalty for dive time spent at shallower depth is an important safety factor.
- After any dive deeper than 60 fsw, and at the conclusion of all repetitive dives, make at least a 3- to 5-minute safety stop at 10 to 15 fsw.
- When using the decompression tables, use the surface-to-surface time or "total time of dive" in selecting the repetitive group designation.
- Carefully plan any repetitive dive so that you stay within no-decompression limits.
- Remember that any device or table that allows prolonged time at shallow depths after a deeper excursion also allows continued uptake of inert gas.
- Maintain hydration during diving days to ensure normal capillary perfusion for inert gas exchange. Remember that immersion diuresis and topside sweat loss in tropical regions can result in significant dehydration, which is often not overtly apparent. Keep the urine "clear and copious" on diving days.
- Do not engage in heavy exercise, such as jogging or wind surfing, for at least 6 hours after a dive.
- Do not fly, even in the pressurized cabin of commercial airlines, for at least 12 hours after no-decompression diving. If decompression stops were required, wait at least 24 hours before flying. If aircraft cabin altitude (e.g., an unpressurized airplane) will exceed 8000 ft, wait 24 hours before flying after any compressed gas dive.

Diving in mountain lakes requires significant adjustments in decompression tables to account for the decreased atmospheric pressure at the surface of the lake. Dive computers must be calibrated for altitude. Boni and associates[18] pointed out that for

the same depth and the same bottom time of a dive, surfacing at a lower ambient air pressure than sea level necessitates longer decompression times. Several decompression tables for altitude diving have been calculated and tested in the field.[11,40,132] The USN Standard Decompression Tables can be used up to an altitude of 2300 ft (701 m).

Even when these safe diving practices are followed, unexplained DCS cases sometimes occur.[1] Two mechanisms are postulated to account for these cases. After an otherwise safe dive, one well within the specified decompression procedure, elevated inert gas tensions exist in tissues and venous blood, which carry gas to the lungs for elimination. If all else is normal and conservative depths and times have been followed, the inert gas remains in solution, or, at worst, "silent" or asymptomatic volumes of venous gas emboli are returned to the lungs. If a physiologic PFO allows intermittent retrograde flow from the right to the left atrium, bubbles can become arterial gas emboli. The potential for this type of paradoxical gas embolism to occur has been demonstrated in recent years for both PFO and other types of intracardiac shunts, as further discussed below.[43,131,173] Whether the presence of a PFO should disqualify someone from diving is currently unclear because conflicting data have been found as to the association of a PFO and the occurrence of neurologic DCS.[43,131,173,182–184]

Another plausible explanation for DCS occurring after safe diving postulates that focal pulmonary barotrauma, or inadvertent breath-holding during decompression, produces local air trapping during ascent and releases microbubbles into the systemic circulation. These microscopic "seed" bubbles in arterialized blood then pass through the capillary bed to become bubble nuclei in venous blood, precipitating overt bubble formation and the classic manifestations of DCS.

DIVE ACCIDENT INVESTIGATION

When investigating a dive accident, whether for treatment purposes or as part of a forensic evaluation in the case of a fatal accident, the investigator begins by taking a detailed dive-accident history. A number of specific details must be determined about the patient's diving activities, the time of symptom onset, and the nature and progression of the symptoms, in addition to medical history and other information that should be obtained on any patient. The diving-related history should specifically solicit information in the following areas[102]:

1. The type of dive and equipment used. Inquire specifically as to whether a decompression computer, diving watch, and depth gauge were used. Was compressed air or mixed gas used, and what was the source of the gas? Was a rebreather used?
2. The number, depth, bottom or total dive time, and surface interval or intervals between dives for all dives in the 72 hours preceding symptom onset. This information will be needed by the diving medicine consultant because it allows calculation of any omitted decompression and thus helps decide the likelihood of the patient having DCS or other problems. Unfortunately, the diver's interpretation of whether required decompression was omitted cannot be the sole source of data; this is notoriously unreliable. If a decompression computer was used, it should be checked for information about the need for recompression and whether it was worn on all dives.
3. Whether in-water decompression was taken and if so, how much. This is relevant to the likelihood of the diver having DCS. If a decompression computer was used, the diver should be asked whether the specified decompression profile was followed.
4. Whether in-water recompression with compressed air was attempted after the onset of symptoms. For all practical purposes, this should never be done because it almost always leaves the diver in a worse condition and is fraught with other hazards. (Of note, special protocols for in-water recompression treatment using 100% oxygen have been developed for emergency situations in exceptionally remote places.)
5. The site of diving (e.g., ocean, lake, or quarry) and the environmental conditions (e.g., water temperature or presence of current or surge) associated with the dive. These factors enter into the differential diagnosis and may raise the possibility of the symptoms being caused by something other than a bubble-related problem. For example, DCS is more common after diving in cold water (other things being equal), and motion sickness may develop in a diver swimming back to shore on a choppy sea even if there was no problem with seasickness before or during the dive.
6. Primary diving activity (e.g., spearfishing, underwater photography, or shell collecting). Like knowledge of environmental conditions, this helps in the differential diagnosis. For example, DCS is more likely after an arduous dive, such as may occur with spearfishing, than a dive to take photographs.
7. Presence of predisposing factors for DCS. A number of factors have been associated with increased risk of DCS, including dehydration, vigorous exercise underwater, advanced age, obesity, poor physical conditioning, local physical injury, and multiple repetitive dives in unacclimatized individuals.
8. Whether the dive was complicated by running out of air, an untoward marine animal encounter, trauma, or other unexpected event. For example, low back pain suggestive of DCS may be caused by a muscle strain from lifting the scuba tank or climbing into the dive boat, and tingling or numbness in an extremity may be caused by jellyfish envenomation.
9. Whether the patient flew in an airplane, went jogging, or engaged in any other particular activity after diving but before the onset of symptoms. If so, the effect of the activity on the symptoms should be ascertained. Some activities (e.g., flying in an unpressurized aircraft or vigorously exercising immediately after diving) may precipitate DCS in someone who might otherwise not be affected, and trivial dysbaric symptoms may become severe after similar activities.
10. Time of symptom onset. Symptoms that began soon after getting in the water (e.g., nausea from motion sickness), even if they worsen afterward, are not likely to be from DCS.

In fatal diving accidents, information gathered from the diving accident history should be supplemented with an appropriate diving accident autopsy, thorough evaluation of the diving equipment used, and detailed environmental history.[50] Environmental factors, such as weather, currents, wave action, visibility, water temperature, potential for entanglement, and dangerous marine life, must be also be considered. The diving equipment should be carefully studied for proper function and

amount of compressed air in the tanks, and the air should be analyzed for contaminants. The diver's medical and psychological histories should be sought because they may contain clues to a coincidental medical event that led to the diver's death but was unrelated to the dive. Aside from the obvious psychosocial risk factors such as alcoholism, drug abuse, or propensity to panic, the use of a scuba dive for suicide or homicide must be considered.

Unique aspects of the diving-victim autopsy should include a careful search for subcutaneous emphysema or other physical signs of pulmonary overpressurization syndrome (POPS) and a search for signs of marine envenomation. For example, before the surface of the body is washed, it should be examined for evidence of nematocysts from coelenterate stings. In addition, in contrast to the thoracic incision being made first, the calvarium should be opened before other incisions to prevent accidental introduction of air into the intracranial circulation. A finding of gas bubbles in intracranial vessels may result from AGE or DCS. Postmortem introduction of gas into the cerebral veins can be avoided if the calvarium is opened under water.[50] Likewise, the initial thoracic incision must be made with care to determine whether pneumothorax is present. A careful search should be made for gas bubbles in the major blood vessels and the heart. The middle ear should be examined for the presence of blood, and the tympanic membrane and paranasal sinuses should be examined for evidence of barotrauma.

Meticulous investigation of dive accidents is important to find equipment, procedural, or medical causes that could be useful in improving the safety of diving and to gather information for legal procedures that often follow diving accidents.

The references for this chapter can be found on the accompanying DVD-ROM.

71 Hyperbaric Medicine

Karen B. Van Hoesen

Hyperbaric oxygen therapy (HBOT) is a method of treating both acute life-threatening and chronic conditions by delivering oxygen (O_2) at partial pressures greater than the normal barometric pressure at sea level of 760 mm Hg. The field of hyperbaric medicine has shown an increasing interest and usefulness in the past 40 years. More than 500 hyperbaric centers currently exist in the United States, and that number continues to grow. HBOT has been shown to be useful in treating diving injuries, acute carbon monoxide poisoning, necrotizing fasciitis, crush injuries, problem wounds, and radiation injuries.

Portable hyperbaric chambers are available for use in remote wilderness settings; however, their use is currently limited to diving injuries and altitude illness. The use of HBOT has been proposed for other applications related to wilderness medicine, such as frostbite, brown recluse spider bites, and, most recently, heatstroke (Box 71-1). However, few data exist to support the routine use of HBOT for these indications. Additionally, HBOT has been suggested as an adjunct to field management of combat trauma in remote settings.

One-year training fellowships in diving and hyperbaric medicine are available throughout the United States. The American Board of Preventive Medicine and the American Board of Emergency Medicine offer a subspecialty board certification in Undersea and Hyperbaric Medicine for individuals who have completed fellowship training.

HISTORY OF HYPERBARIC MEDICINE

Treatment with compressed air therapy dates back to 1662, when the British clergyman Henshaw treated acute medical disorders with increased pressure and chronic disorders with decreased pressures.[60] Compressed air was used to treat caisson disease or decompression sickness (DCS) in the late 1800s. Medical use of HBOT began in the 1930s with the first systematic studies on the physiologic effects of oxygen.[7] Extensive research on oxygen toxicity and safe oxygen limits was conducted during the 1940s, when oxygen was incorporated into the treatment tables of the United States Navy (USN) for diving injuries.

Clinical hyperbaric medicine really began in the 1950s and 1960s with the report of the use of HBOT for anaerobic infections, including gas gangrene and clostridial infections, and for carbon monoxide poisoning.[21,143] During the 1970s, HBOT was used widely to treat a variety of conditions with no scientific basis. In 1976, the Undersea Medical Society (UMS) established a multidisciplinary committee to review research and clinical data on the uses of HBOT. Every 3 years, the committee publishes a critical review of the approved indications for HBOT (Table 71-1).[39] In 1986, the UMS changed its name to the

amount of compressed air in the tanks, and the air should be analyzed for contaminants. The diver's medical and psychological histories should be sought because they may contain clues to a coincidental medical event that led to the diver's death but was unrelated to the dive. Aside from the obvious psychosocial risk factors such as alcoholism, drug abuse, or propensity to panic, the use of a scuba dive for suicide or homicide must be considered.

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Undersea and Hyperbaric Medical Society (UHMS) to incorporate the expanding field of hyperbaric medicine.

MECHANISMS OF HYPERBARIC OXYGEN

HBOT is inhalation of oxygen at a pressure greater than atmospheric pressure at sea level, or 1 atmosphere (atm). An individual is placed inside a hyperbaric chamber that is pressurized with air or oxygen. The atmospheres absolute (ATA) is the sum of the atmospheric pressure and the hydrostatic pressure as measured in the chamber. For every increase of 33 feet of seawater (fsw) (10 m), a pressure increase of 1 atm (760 mm Hg) is present. At 66 fsw, the absolute pressure in the chamber is 3 ATA. Increasing the atmospheric pressure in the hyperbaric environment causes both mechanical reduction in bubble size and increased partial pressure of oxygen in the tissues.

Box 71-1. Wilderness Applications for Hyperbaric Oxygen Therapy

Altitude illnesses
Diving injuries
Decompression sickness
Arterial gas embolism
Frostbite
Brown recluse spider bite
Heatstroke
Necrotizing fasciitis from marine-acquired wounds
Field management of combat trauma

Effects of HBO on Oxygen Content

The oxygen content of blood is the sum of oxygen carried by hemoglobin and oxygen dissolved in the blood plasma. At 1 atm at sea level, 98% of the oxygen content of normal blood is bound to hemoglobin. The oxygen content of blood plasma is determined by the solubility of oxygen in plasma at 37° C (98.6° F), which at a partial pressure of oxygen (Po_2) of 100 mm Hg results in oxygen in solution of only 0.31 mL/dL. Once hemoglobin is fully saturated, further increases in oxygen partial pressure can only increase the amount of oxygen physically dissolved in the plasma. As Po_2 increases in inspired air, the amount of oxygen dissolved in plasma increases linearly (Fig. 71-1). For every atmosphere of pressure increase, 1.8 mL/dL is dissolved in plasma. At 3 ATA, approximately 6.8 mL/dL of oxygen can be dissolved in plasma. Because normal oxygen extraction at the tissue level is 5 mL/dL (at normal cardiac output), plasma alone can carry enough oxygen to meet the metabolic needs of the tissues. In addition, this increase in oxygen carrying capacity dramatically increases the driving force for oxygen diffusion.

Effects of Elevated Po_2

The increase in tissue oxygen concentration has important physiologic and biochemical effects in both normal and diseased tissue. These include vasoconstriction, antimicrobial effect, wound healing, and ischemia-reperfusion injury (Box 71-2).

Vasoconstriction

Hyperoxia-induced vasoconstriction occurs in both arterial and venous vasculature and has been demonstrated in cerebral, retinal, renal, and myocardial blood flow. Despite reduction in blood flow from vasoconstriction, tissue oxygen partial pressures actually increase because of the increased amount of

TABLE 71-1. Approved Indications for Hyperbaric Oxygen Therapy (HBOT)

INDICATION	TREATMENT PROFILE	NUMBER OF TREATMENTS
Air or gas embolism	USN Table 6 or 6A Repeat treatment with USN Table 5, 6, or 2.4 ATA for 90 minutes	1 to 10
Carbon monoxide poisoning	2.4 to 3.0 ATA for 90 to 120 minutes	1 to 5
Clostridial myositis and myonecrosis (gas gangrene)	3.0 ATA for 90 min tid in first 24 hours, then bid for 2 to 5 days	3 to 10
Crush injury, compartment syndrome and acute ischemia	2.0 to 2.5 ATA for 90 to 120 minutes	2 to 12
Decompression sickness	USN Table 6 Repeat treatment with USN Table 5, 6, or 2.4 ATA for 90 minutes	1 to 10
Selected problem wounds	2.0 to 2.5 ATA for 90 to 120 minutes	10 to 30
Exceptional anemia	2.4 to 3.0 ATA for 90 to 120 minutes	No data
Intracranial abscess	2.0 to 2.5 ATA for 60 to 90 minutes	10 to 20
Necrotizing soft tissue infection	2.0 to 3.0 ATA for 90 to 120 minutes	5 to 30
Refractory osteomyelitis	2.0 to 2.5 ATA for 90 to 120 minutes	20 to 40
Delayed radiation injury and soft tissue and bony necrosis	2.0 to 2.5 ATA for 90 to 120 minutes	30 to 60
Skin grafts and flaps	2.0 to 2.5 ATA for 90 to 120 minutes bid initially until graft/flap appears stable	10 to 20
Thermal burns	2.0 to 2.5 ATA for 90 to 120 minutes tid in first 24 hours, then bid	20 to 40

ATA, atmospheres absolute; USN, United States Navy.

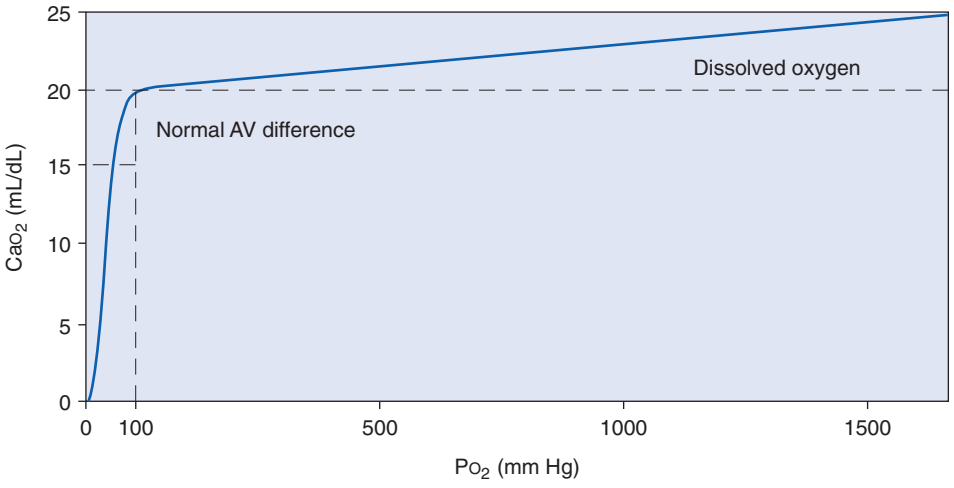


Figure 71-1. Effects of hyperbaric oxygen on the oxygen content of arterial blood. The blood oxygen content (CaO_2) under normal conditions is approximately 20 mL/dL and is carried almost exclusively by hemoglobin. The normal oxygen extraction (arterial-venous [AV] difference) is approximately 5 mL/dL or about one quarter of the total CaO_2. During hyperbaric oxygen therapy, dissolved O_2 in plasma can be increased to provide the entire AV difference without unloading O_2 from hemoglobin.

Box 71-2. Mechanisms of Hyperbaric Oxygen Therapy

Mechanical decrease in bubble volume
Vasoconstriction and reduction of edema
Antibacterial effects
Bactericidal effect on anaerobic bacteria
Bacteriostatic effect on selected facultative and aerobic organisms
Enhance transport and augment antibiotic efficacy
Wound healing
Enhance collagen synthesis
Stimulate angiogenesis
Improve leukocyte function of bacterial killing
Blunt systemic inflammatory responses
Stimulate vascular endothelial growth factor (VEGF) release
Induce platelet-derived growth factor (PDGF) receptor appearance
Diminish ischemia-reperfusion injury
Decrease leukocyte venular endothelial adherence
Decrease release of toxic oxygen species

oxygen dissolved in plasma. Edema reduction occurs because filtration of capillary fluid is decreased, whereas vascular outflow and improved oxygen delivery are maintained. This vasoconstriction can be beneficial in peripheral edema and intracerebral edema and decreases edema in burns and postischemic tissues.[12,114,117,118] Hence, blood flow in the microcirculation is improved through decreasing interstitial fluid pressures from edema reduction.

Antibacterial Effect

HBO has been shown to have a potent inhibitory effect on growth of various microorganisms. HBO causes a direct bactericidal effect on anaerobic bacteria through production of toxic radicals in the absence of free radical degrading enzymes, such as superoxide dismutase, catalases, and peroxidases.[67] HBO also has a bacteriostatic effect on selected facultative and aerobic organisms.[77] Tissue Po_2 of 30 to 40 mm Hg is necessary for oxidative killing of microorganisms by neutrophils. Increased Po_2 in infected and hypoxic tissue enhances neutrophil function and return of antimicrobial activity. HBO also augments the bactericidal action of aminoglycosides and potentiates the effect of certain sulfonamides.[54,72] Aminoglycosides and cephalosporin antibiotic transport across the bacterial cell wall requires tissue oxygen tensions above 30 mm Hg. Overall, HBO enhances transport and augments antibiotic efficacy.

Wound Healing

The relationship between tissue wound hypoxia and wound healing has been well documented.[64] Tissue hypoxia decreases fibroblast replication, collagen deposition, angiogenesis, resistance to infection and intracellular leukocyte bacterial killing, which are all oxygen sensitive responses essential to normal wound healing. Intermittent elevations of tissue Po_2 reverse localized tissue hypoxia and correct the pathophysiology related to oxygen deficiency and impaired wound healing. HBO enhances collagen synthesis, stimulates angiogenesis, improves leukocyte function of bacterial killing, and blunts systemic inflammatory responses.[65,90] In addition, hyperbaric oxygen stimulates vascular endothelial growth factor (VEGF) release and induces platelet-derived growth factor (PDGF) receptor appearance.[14,140,183] The net result of serial increased Po_2 from HBOT is improved local host immune responses, clearance of infection, enhanced tissue growth, angiogenesis with progressive improvement in local tissue oxygenation, and epithelialization of hypoxic wounds.

Ischemia-Reperfusion Injury

Ischemia-reperfusion injury is mediated by oxygen-derived free radicals, such as superoxide and hydroxyl free radicals, that are produced during prolonged periods of ischemia followed by reperfusion. These oxyradicals can lead to cell death by lipid peroxidation and generation of further free radicals. Oxyradicals are generated from xanthine oxidase and neutrophils. Neutrophil endothelial adhesion and subsequent release of free radicals play an important role in endothelial and microcirculatory damage.[133]

HBO protects tissues from reperfusion injury. HBO provides additional oxygen for reperfused tissues to generate scavengers such as superoxide dismutase, catalase, peroxidase, and flu-

Figure 71-2. Monoplace hyperbaric chamber. Monoplace chambers hold a single patient, usually in an environment of pure oxygen. Attendants remain outside the chamber during treatment.

tathione, which detoxify destructive oxygen radicals before they damage tissues.[42] HBO antagonizes the $beta_2$ integrin system, which initiates the adherence of neutrophils to post capillary venule endothelium.[155,156] Reperfusion injury may be inhibited by HBO through a decrease in leukocyte venular endothelial adherence, release of toxic oxygen species, and arteriolar vasoconstriction; hence, progressive arteriolar vasoconstriction is inhibited.[181] HBO inhibits intracellular adhesion molecule (ICAM-1) expression, which plays a role in neutrophil adhesion and ischemia-reperfusion injury.[22] Additionally, HBO upregulates transforming growth factor beta 1 (TGF-β1), which decreases reperfusion injury by upregulating bcl-2 and inhibiting tumor necrosis factor alpha (TNF-α).[53] HBO has been shown to be beneficial in almost all animal models of ischemia-reperfusion studied and results in increased microvascular perfusion in skeletal muscle and improved skin flap survival.[85,177,181,182] It may be an important adjunct for treatment of tissue at risk for reperfusion injury that can accompany crush injury, limb replantation, compartment syndrome, and decompression illness. New data suggest a role of HBOT in preventing ischemia-reperfusion injury associated with myocardial infarction and cerebral ischemia.

TYPES OF CHAMBERS

Monoplace Chambers

Monoplace or single-person chambers account for the majority of chambers in the United States. They accommodate a single patient, and the chamber is pressurized either with 100% oxygen or with air while the patient breathes oxygen through a mask or hood. Monoplace chambers are easier to maintain, require less space, and are less expensive than are multiplace chambers. They can be configured with noninvasive and invasive monitoring. Specially adapted ventilators and monitoring systems in some monoplace chambers allow for treatment of critically ill patients. During treatment, there is no direct access to the patient. Therefore, the chamber must be decompressed for an emergency, which can limit its use in some critically ill patients (Fig. 71-2). Most monoplace chambers cannot exceed 3 ATA treatment depth.

Figure 71-3. Multiplace hyperbaric chamber. Multiplace chambers allow more than one patient at a time to receive hyperbaric oxygen. Attendants may accompany the patients during treatments. (Courtesy Duke University Photography. Photo by Les Todd.)

Multiplace Chambers

Multiplace chambers hold two or more people and allow more than one patient to be treated at a given time, accompanied by attendants including technicians, nurses, and respiratory therapists (Fig. 71-3). The chamber is pressurized with air; 100% oxygen is delivered by face mask, with a head tent, or through an endotracheal tube. Multiplace chambers allow for intensive care–level monitoring, mechanical ventilators, vascular pressure monitoring, intravenous infusion pumps, blood gas analysis, and medication administration. Multiplace chambers can maintain a treatment pressure of 6 ATA and are ideal for treatment of diving injuries, particularly arterial gas embolism (AGE). General anesthesia, surgery, chest tube placement, and cardiopulmonary resuscitation have been performed inside multiplace chambers. They have the disadvantage of a risk of DCS to attendants during long treatments.

Portable Recompression Chambers

Portable monoplace recompression chambers have been developed for use in remote areas for treatment of both diving injuries and high-altitude illnesses.

Altitude Illness

Several portable hyperbaric chambers are available for emergency treatment of severe acute mountain sickness, high-altitude pulmonary edema, and high-altitude cerebral edema, particu-

A

B

Figure 71-4. Chinook Medical Gear's Gamow portable altitude chamber. **A,** In the field. **B,** Close-up. (Courtesy Chinook Medical Gear, Inc.)

A

B

Figure 71-5. **A** and **B,** Certec chamber. (Courtesy Certec Company.)

Figure 71-6. Treksafe Portable Altitude Chamber (PAC). (Courtesy Treksafe Company.)

larly when descent is not feasible or while waiting for evacuation (Figs. 71-4 to 71-6). They are constructed of relatively lightweight fabrics and require no use of portable oxygen. The portable chambers are pressurized with ambient air using manual foot pumps or hand pumps and can simulate a descent of 1500 to 2500 m (5000 to 8200 ft). Hyperbaric treatment should only be used as an emergency measure and does not substitute for descent or evacuation.

The patient should be told to breathe normally and instructed on equalizing his ears by swallowing or Valsalva maneuver. The patient is placed inside the portable chamber, the zipper is pulled

shut, and the bag is inflated with the foot pump to 2 psi (104 mm Hg) above ambient pressure. Two pop-off pressure values are set at 2 psi and prevent overpressurization of the bag. All the bags must be pumped continually 8 to 12 times per minute in order to flush air through the chamber to prevent carbon dioxide (CO_2) buildup. Patients should be treated for approximately 1 hour and then removed from the bag and reassessed. Additional cycles of descent and reassessment are continued until the patient improves and is able to descend or until evacuation is available. Patients with severe high altitude pulmonary edema (HAPE) may not be able to lie flat; hence, one end of the chamber can be propped up 30 to 40 cm (12 to 16 inches). In severe cases, a patient can breathe oxygen at 4 to 6 L/minute during the treatment by placing an oxygen bottle inside the chamber.

Three portable chambers are currently available for use at high altitude. The Gamow Bag (see Figure 71-4) was invented by Dr. Igor Gamow at the University of Colorado. It is cylindrical, lightweight (the bag, pump, and carrying day pack weigh approximately 7 kg (15 lb), and easy to use. It measures 2.5 by 0.6 m (8 × 2 ft) and can maintain an internal pressure of 2 psi (104 mm Hg or 0.13 atm). The Certec hyperbaric chamber (Certec, Le Bourg, France) comprises two bags: an outside envelope made to withstand tension and provide stability and an inside envelope of polyurethane that allows for more durability of the chamber (see Figure 71-5). The Certec chamber weighs 4.8 kg (10.5 lb) and measures 2.2 × 0.65 m (7 × 2 ft); it can be pressured to a slightly higher inflation pressure (internal pressure of 165 mm Hg or 0.22 atm or 3.2 psi) than can the Gamow bag. This is equivalent to about 800 m (2625 ft) greater simulated altitude descent; however, it is unclear if this is important clinically. The Portable Altitude Chamber (PAC) (Treksafe, Repton, Australia) is about the same size as the Gamow bag but allows more room at the head and shoulders and has a radial zipper at the head allowing easier assess for the patient (see Figure 71-6). All three chambers have been used successfully at altitude; early symptom relief and improvement in peripheral oxygen saturation and cerebral oxygenation have been reported.[4,66] These chambers have no utility in the treatment of diving injuries.

Diving Injuries

Sturdier portable chambers exist for treatment of diving related injuries. Although there are a number of hard, small recompression chambers used in commercial diving operations, these chambers are not collapsible or easily portable. Two collapsible chambers are available for treatment of diving injuries. These chambers are used primarily by the USN and commercial diving operations. They are collapsible and pressurized with air from a scuba cylinder while the patient breathes 100% oxygen from a face mask. The patient can be transported within the portable chamber to a large recompression facility and moved under pressure into a large multiplace chamber. Unlike the lightweight portable chambers used for treatment of altitude illnesses, these have a working pressure of 2.8 ATA or 27 psi (equivalent of 60 fsw).

The SOS Hyperlite Hyperbaric Stretcher (SOS Ltd., London, England) is the most widely used collapsible chamber for treatment and transportation under pressure of a patient suffering from DCS or AGE to a permanent recompression chamber (Fig. 71-7). The chamber is made of Kevlar and has two removable endplates with communication system and availability to deliver

Figure 71-7. The Hyperlite by SOS Limited is compressed with air from a scuba cylinder. The injured diver breathes 100% oxygen from a separate oxygen bottle.

100% oxygen to the patient. It is capable of a standard treatment depth of 2.82 ATA and weighs approximately 165 lb (75 kg). Also called the Emergency Evacuation Hyperbaric Stretcher (EEHS), it has received certification for use within the U.S. Department of Defense and is currently in use by the U.S. Coast Guard, all four services of the U.S. military, the Russian Navy, and the U.S. space agency, NASA.

Another collapsible and portable hyperbaric chamber is the Italian-made GSE chamber. A smaller version, the Hyperbaric Backpack, is available. It is a 30-inch diameter (76 cm) and 7-ft-long (2 m) chamber that weighs 92 lb (42 kg). It is a double bag of translucent composite polyester and specified for 6 ATA. Another chamber, the Chamberlite 15, is probably the simplest and one of the lightest units on the market. It is capable of providing 100% oxygen at 2 to 2.4 ATA pressure with special adapters. This unit is constructed of foldable polyurethane, has ten viewing ports, and weighs less than 40 lb (18 kg), thus making it highly portable by standard stretcher or two-person carry. However, depth of treatment is limited, because treatment of diving injuries requires a minimum treatment depth of 2.82 ATA. It has been used in studies on carbon monoxide (CO) poisoning and may be a useful tool for emergency treatment of CO poisoning in the field.[141]

CONTRAINDICATIONS TO HBOT

Absolute

HBOT is safe with very few contraindications. Untreated pneumothorax is an absolute contraindication to treatment, because it may progress to a tension pneumothorax during decompression. Once a chest tube has been placed, the patient can safely undergo HBOT. Treatment with cisplatin, bleomycin, disulfiram, doxorubicin, and mafenide acetate may have toxic effects when used concurrently with HBOT and is considered absolute contraindications to HBOT.[76]

Relative

Any condition that interferes with the ability to equalize pressure in the sinuses, ears, or lungs (e.g., acute or chronic sinusitis, otitis media, upper respiratory infection) is considered a

relative contraindication. Nasal and oral decongestants can be used prior to treatment; myringotomy or placement of tympanoplasty tubes can facilitate treatment in certain situations. The use of bronchodilators and slow ascent rates can allow individuals with chronic obstructive pulmonary disease (COPD) to undergo HBOT.

COMPLICATIONS OF HBOT

Oxygen Toxicity

In 1878, Bert described seizures and death in animals exposed to 3 to 4 ATA. This central nervous system (CNS) manifestation of oxygen toxicity became known as the Paul Bert effect.[9] In 1899, pulmonary oxygen toxicity was described after prolonged exposures to 0.74 to 1.3 atm of oxygen.[88] Tissue injury from oxygen is mediated by reactive oxygen species (ROS) that are produced by chemical reactions involving single electron transfers to molecular oxygen or its metabolites. These ROS include superoxide anion (O_2^-), hydroxyl radial (OH^-), and hydrogen peroxide (H_2O_2).

Under normal oxygen conditions, cells have adequate antioxidant defenses. These defenses include antioxidant enzymes such as superoxide dismutase and catalase, and low–molecular-weight scavengers such as glutathione and vitamin E. As P_{O_2} increases, these defenses are overwhelmed and ROS cause lipid peroxidation, protein oxidation, sulfhydryl depletion, and oxidation of pyridine nucleotides, leading to cell membrane disruption, cell injury, and death.[43]

CNS Oxygen Toxicity

CNS toxicity occurs during oxygen exposures of 1.4 atm or greater. Symptoms may include dizziness, irritability, tunnel vision, tinnitus, nausea, and seizures. The incidence of oxygen-induced seizures is about 1/10,000 patients treated at 2.4 ATA. Susceptibility to oxygen-induced seizures varies among individuals and in any single individual from day to day. Factors that increase susceptibility include exertion, increased P_{CO_2}, increased metabolic rate from fever, thyrotoxicosis, adrenal stress, and acute cerebral injury. Catecholamines can lower the seizure threshold and increase the risk of CNS O_2 toxicity. Oxygen-induced seizures are self-limited and resolve quickly after discontinuing oxygen. No permanent neurologic sequelae have been reported from HBO-induced seizures, and anticonvulsants are not indicated. Intermittent exposure to brief periods of air during HBOT significantly extends oxygen tolerance and prevents oxygen toxicity.

Pulmonary Oxygen Toxicity

Symptoms of early pulmonary oxygen toxicity include substernal burning, slight cough, mild dyspnea, and chest tightness and can be seen in patients treated for decompression sickness, air embolism, and gas gangrene when treatments are prolonged. These symptoms resolve after discontinuing therapy. Pulmonary oxygen toxicity is rare using current treatment tables. Patients do not experience significant alteration in pulmonary gas exchange when treated with standard HBO treatment protocols.[176]

Barotrauma

The most common side effect of HBOT is middle ear barotrauma caused by inadequate equalization of pressure in the middle ear. This may result from an upper respiratory infection, eustachian tube dysfunction, or inadequate techniques of equalization. Middle ear barotrauma typically occurs during compression, causing pain and hemorrhage within the middle ear, rarely leading to tympanic membrane rupture. It can be prevented by slow compression rates with frequent stops, by proper autoinflation techniques, and by use of decongestants. For individuals who have significant difficulty clearing their ears, bilateral tympanotomies can be placed. In an emergency, myringotomies can be performed. Sinus barotrauma results from blockage of the sinus ostia due to upper respiratory infection or allergic sinusitis or rhinitis. Symptoms include pain in the maxillary or frontal sinus. Oral or nasal decongestants before treatment may allow the patient to proceed with HBOT.

Claustrophobia

Some patients may experience claustrophobia or confinement anxiety in both monoplace and multiplace chambers. Benzodiazepines can be given prior to treatment to help relieve anxiety.

Visual Refractive Changes

Patients who undergo multiple (>20) HBOT treatments often develop progressive myopia that is temporary and reverses completely within a few weeks after discontinuing HBOT. The exact mechanism is not known but appears to be lenticular in origin.[26,91,121] Treatment with HBOT beyond 150 treatments is associated with increased risk of irreversible refractive changes or development of new cataracts. In the U.S., HBOT treatments rarely exceed 60 in number.

PRACTICAL ASPECTS OF HYPERBARIC TREATMENT

Evaluating the Patient for HBOT

For elective treatments, patients are assessed for appropriateness of HBOT and contraindications to treatment. Patients should be screened for history of seizures, pulmonary disease including asthma or COPD, implanted devices such as cardiac pacemakers or intrathecal pumps, and claustrophobia. Assessment of eustachian tube function and ability to clear the ears can be assessed by examining the tympanic membrane with an otoscope and asking the patient to hold his nose and swallow or perform a simple Valsalva maneuver. If the tympanic membrane moves with either maneuver, the eustachian tube is patent.

In emergency situations, patients may be unconscious and cannot be screened for contraindications to HBOT. If the patient is not alert, it may be necessary to intubate for airway protection; it is safer to sedate combative or semicomatose patients and mechanically ventilate them than to restrain and attempt to control an agitated patient in the chamber. Patients who are sedated and intubated generally do not need myringotomies, because passive inflation of the middle ear space during hyperbaric compression will occur. If necessary, an emergency myringotomy can be performed. Under direct visualization of the tympanic membrane, a perforation is made in the anterior-inferior quadrant of the tympanic membrane with a 21-gauge spinal needle. Patients with diving injuries and CO poisoning should be maintained on 100% oxygen by face mask at 10 to 15 L until treatment is initiated in the hyperbaric chamber.

Preparation for Hyperbaric Treatment

Preparation of a hyperbaric patient requires strict attention to certain safety issues and protocols. For fire safety reasons, patients must wear hospital-supplied clothing of either 100% cotton or a blend of 50/50 polyester-cotton. Undergarments

Box 71-3. Equipment Used in Hyperbaric Chambers

NOT ALLOWED FOR USE IN CHAMBER:
Telemetry device
Defibrillator
Brush driven motor (noninvasive blood pressure [NIBP] machines)
Laser printer (ECG strips)
Pneumatic stockings
Patient-controlled anesthesia (PCA) pump
Non–pressure tested IV pump
Transport ventilator
Unnecessary equipment, such as pulse oximetry device
Off-gassing batteries and equipment supplied by >12 volts
48-watt power source

ALLOWED INSIDE CHAMBER:
ECG monitor
Harvard Bard pump
HBO-approved IV pump
HBO-approved ventilator
Doppler
Permanent pacemakers (check with manufacturer for pressure tested)
Swan-Ganz and arterial line
Pressure bag
Drains, Foley catheter, nasogastric tube, endotracheal tube cuff
Glass bottles inspected by HBO staff and vented if necessary

ECG, electrocardiogram; HBO, hyperbaric oxygen; IV, intravenous.

must be 100% cotton. Silk, wool, waffle weave, and nylon and other synthetic materials are prohibited. Watches must be inspected for pressure compatibility. Excessive cosmetics, lotions, and perfumes should be removed. Patients must be inspected for matches, lighters, and smoking materials, all of which are prohibited. The hyperbaric staff should follow established protocols at their facility. In general, staff must (1) ensure eustachian tube patency, (2) remove all medical devices not required during hyperbaric treatment, (3) cap all intravenous (IV) catheters that are not in use, (4) empty Foley bag or have patient void before chamber entry, (5) have labeled medications available, (6) confirm a good signal on all monitors and check that monitors and IV pumps are fully charged, and (7) ensure that the ventilator is functioning correctly on the surface. A list of equipment that is prohibited and allowed inside a hyperbaric chamber is presented in Box 71-3.

Manifestations of CNS Oxygen Toxicity

Convulsions due to CNS oxygen toxicity can occur in patients breathing oxygen at pressures of 1.4 ATA or greater. Convulsions may occur abruptly or be preceded by signs of CNS irritability, including facial twitching, diaphoresis, apprehension, visual field constriction, tinnitus, nausea, or vertigo (Box 71-4). Manifestation of any of the premonitory symptoms of CNS oxygen toxicity is treated by switching the patient's breathing gas from oxygen to chamber air. Once all symptoms have resolved, oxygen therapy can be resumed; however, shortening the duration of oxygen exposure can prevent recurrence of symptoms. A small dose of benzodiazepines, such as IV diazepam 5 to 10 mg, will usually abate CNS oxygen toxicity symptoms if oxygen discontinuation fails to provide relief.

Management of Oxygen-Induced Seizures

If a patient has an oxygen-induced seizure, the breathing gas must be switched from oxygen to chamber air and the patient protected from mechanical trauma.

It is critical to avoid decompression during the tonic phase of a seizure. Expanding gas within the lungs could rupture alveoli, producing an arterial gas embolism. After tonic-clonic activity has subsided and respirations have normalized, the chamber may be decompressed. An oxygen-induced seizure is not a contraindication to further HBOT; anticonvulsant medication is not indicated for a patient with no previous seizure history.

Box 71-4. Signs and Symptoms of CNS Oxygen Toxicity

Diaphoresis
Bradycardia
Palpitations
Euphoria
Apprehension
Visual fields constriction
Tinnitus
Nausea
Vomiting
Vertigo
Lip or facial twitching
Seizures

CNS, central nervous system.

Additional seizures can be prevented by shortening the duration of oxygen exposure or decreasing the oxygen pressure. There are no residual effects of oxygen-induced convulsions if mechanical trauma is avoided.

Pediatric Considerations

Generally, indications for HBOT in the pediatric population are similar to those in adult patients. Accidental carbon monoxide poisoning and serious burns are common in pediatric age groups. Invasive procedures can result in iatrogenic cerebral gas embolism in children as in adults. Figure 71-8 shows a 7-day-old infant with clostridial omphalitis from an umbilical vein catheter that developed into gas gangrene of the abdominal wall. The child was treated successfully with HBOT at University of California, San Diego, Hyperbaric Center.[128] This center has also successfully treated a child with acute hepatic artery thrombosis after liver transplantation.[52]

Special attention must be paid to reduce evaporative, conductive, and convective heat losses in the chamber because children have an increased risk of hypothermia. An oxygen hood can be used for most children older than 3 to 4 years. In younger children, the neckdam can be pulled down to waist level, which allows the child's hands to be free inside the hood (Fig. 71-9). Although there are no studies on the safe limits of exposure to hyperbaric oxygen for children, in practical experience there is no increased risk for developing pulmonary or neurologic oxygen toxicity. Additionally, there are no significant differences in side effects or morbidly with HBOT for children compared with adults.[135] One theoretical problem with HBOT exists for

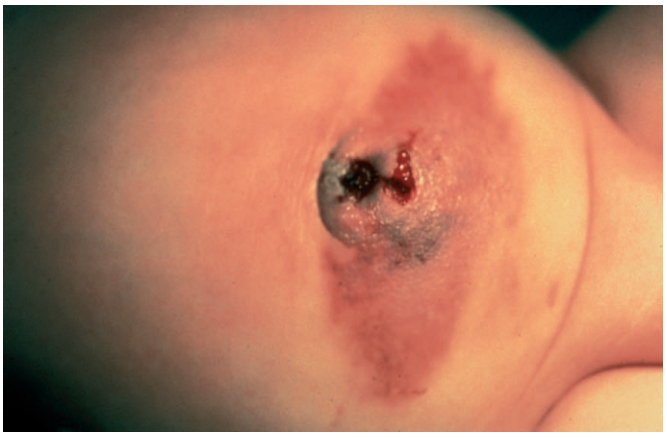


Figure 71-8. A 7-day-old infant with gas gangrene of the abdominal wall due to *Clostridium perfringens* treated with hyperbaric oxygen therapy with good outcome.

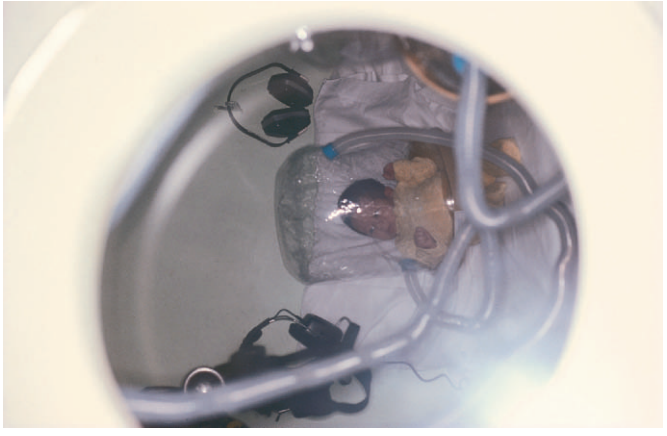


Figure 71-9. Child being treated in a hyperbaric chamber with the neckdam of the oxygen hood around his waist.

the child with a ductus arteriosus–dependent congenital heart disorder. The increased Po_2 could result in ductal constriction, limiting cardiac output, and must be considered when treating an infant with congenital heart disease.[166]

APPROVED CLINICAL APPLICATIONS OF HBOT

Arterial Gas Embolism

AGE can result from pulmonary barotrauma while diving (see Chapter 70). Pulmonary barotrauma typically occurs when a diver experiences rapid ascent while breath-holding due to panic or running out of air. Rapid expansion of gas causes pulmonary overdistention and disruption of the alveolar capillary membrane. Air enters the pulmonary veins, travels to the heart, and enters the systemic circulation. Air embolism may result from nondiving causes, many of which are iatrogenic. Documented AGE has occurred during central venous catheterization, mechanical ventilation, cardiopulmonary bypass, angioplasty, lung biopsy, percutaneous hepatic puncture, liver transplant, hemodialysis, penetrating chest trauma, oral sex, sexual intercourse after childbirth, transurethral prostatectomy, laparoscopy, arthroscopy, and cesarean delivery.[5,6,8,30,31,38,59,75,103,111,160,163,164,169] Abnormal neurologic findings after cardiovascular or neurologic surgery or dialysis should make one consider AGE.

AGE presents with sudden alteration in consciousness, confusion, focal neurologic deficits, cardiac arrhythmias, and death. AGE requires immediate recompression. HBOT reduces the volume of gas bubbles, thereby restoring blood flow, and improves oxygen delivery to ischemic, hypoperfused tissues. HBOT reduces cerebral edema and decreases ischemia-reperfusion injury. Treatment for AGE had traditionally been with USN Table 6A with an initial excursion to 165 ft (6 ATA) for 30 minutes to enhance bubble compression (Fig. 71-10). Animal studies showed no additional benefit from compression to 6 ATA compared with 2.82 ATA.[86] Current recommendations include initial recompression to 2.82 ATA breathing 100% oxygen. If there is no improvement or clinical deterioration, deeper recompression to 6 ATA can be instituted. The diver

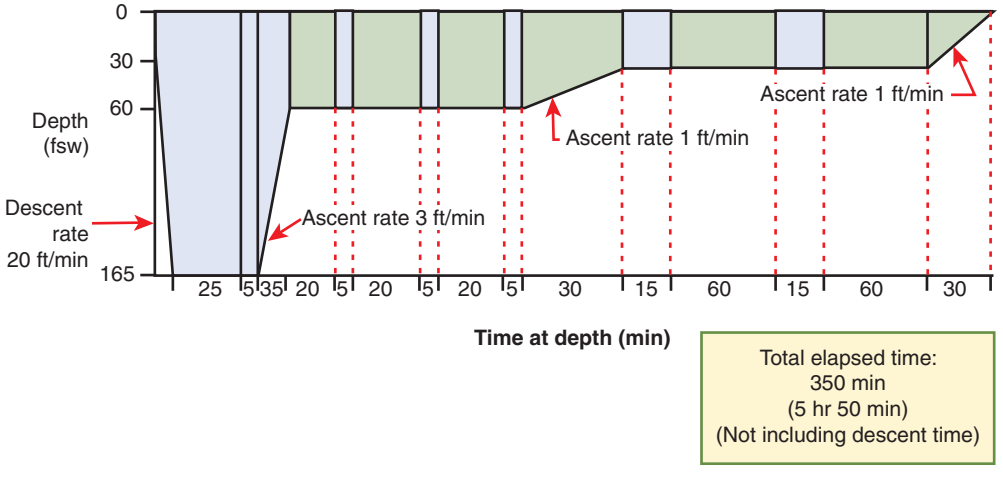


Figure 71-10. U.S. Navy Treatment Table 6A. fsw, feet of seawater. (Redrawn from U.S. Navy Diving Manual.)

breathes a mixture of 50% nitrogen and 50% oxygen below 60 ft (18 m). Repetitive treatments are recommended until there is no further stepwise improvement.[108]

The Divers Alert Network (DAN) has an on-call diving physician available 24 hours a day, 7 days a week, who can assist in triage and arrangement of transport and treatment for all diving injuries. In the U.S., DAN can be reached at 919-684-8111.

Decompression Sickness

DCS results from generation of bubbles of inert gas in the tissues and blood that interfere with organ function by occluding blood flow and other biochemical events (see Chapter 70). DCS may result from rapid ascent from diving, flying after diving, ascent in an unpressurized aircraft, or exposure in a hyperbaric or hypobaric chamber. The definitive treatment of DCS is immediate recompression and HBOT. HBOT causes immediate reduction in bubble volume, increase in the diffusion gradient for inert gas from the bubble into surrounding tissues, oxygenation of ischemic tissue, and reduction in cerebral edema. Other beneficial effects of HBOT that probably play a role in treating DCS are reduction in neutrophil adhesion to capillary endothelium and prevention of ischemia-reperfusion injury.[94]

Various hyperbaric treatment regimens for DCS have been described. However, there are no human prospective, randomized studies for treatment of DCS. In general, USN Table 6 has become the standard of treatment for pain-only and neurologic DCS.[153] Occasionally, USN Table 5 is still used for pain-only DCS, but many diving physicians have discontinued its use.[50] (USN Tables 5 and 6 are shown in Chapter 70 in Figure 70-14.) These tables consist of compression to 60 fsw, or 2.8 ATA, initially with intermittent oxygen breathing. Theoretical advantages of using helium and oxygen in the Comex-30 table have been reported.[78] Longer oxygen treatment tables exist, with extended decompression profiles for refractory cases of DCS involving the CNS.

The choice of treatment table and number of treatments required depend on the clinical severity of illness, response to treatment, and residual symptoms after initial recompression. If the delay to treatment is not excessive, the majority of cases of DCS have complete resolution of symptoms after a single hyperbaric treatment. Patients with severe DCS that involves the spinal cord or a prolonged delay to recompression may have incomplete resolution of symptoms and often require repetitive treatments. Although delay to treatment worsens outcome for severely injured divers, the data currently available have not established a maximum time after which recompression is ineffective.[3,35] Patients with DCS should continue treatments until symptoms have completely resolved or until the patient shows no further improvement in response to two consecutive treatments. Most individuals need no more than 5 to 10 repetitive treatments. Repetitive treatments can be given daily or twice daily as USN Table 5, USN Table 6, or 2-hour treatment at 2.4 ATA. A small number of patients have residual neurologic deficits that may either be permanent or improve gradually over 6 to 12 months.

Carbon Monoxide and Cyanide Poisoning

Carbon monoxide (CO) poisoning is a significant health problem in the United States. Poisonings occur primarily from fire smoke, exhaust from internal combustion engines, and inhalation of fumes from faulty gas furnaces. CO poisoning has also occurred in recreational boaters and campers who use internal combustion engines and fossil fuel heaters in enclosed spaces.

CO toxicity is mediated by a number of mechanisms that lead to hypoxic stress. CO binds to hemoglobin with an affinity 200 times that of oxygen, resulting in a shift of the hemoglobin-oxygen dissociation curve to the left, which results in tissue hypoxia. Additionally, CO binds to myoglobin and cytochrome oxidase, interfering with intracellular respiration and increasing oxidative stress, both of which contribute to cell death. Poor correlation exists between tissue hypoxia and blood carboxyhemoglobin (COHb) levels. The most common signs and symptoms of CO poisoning are headache, nausea, vomiting, confusion, and a flu-like illness. CO poisoning can result in a delayed neuropsychiatric syndrome (DNS) characterized by deterioration in cognitive and motor function that appears days to weeks after the initial insult. DNS is reported in 3% to 20% of acute CO poisoning victims.

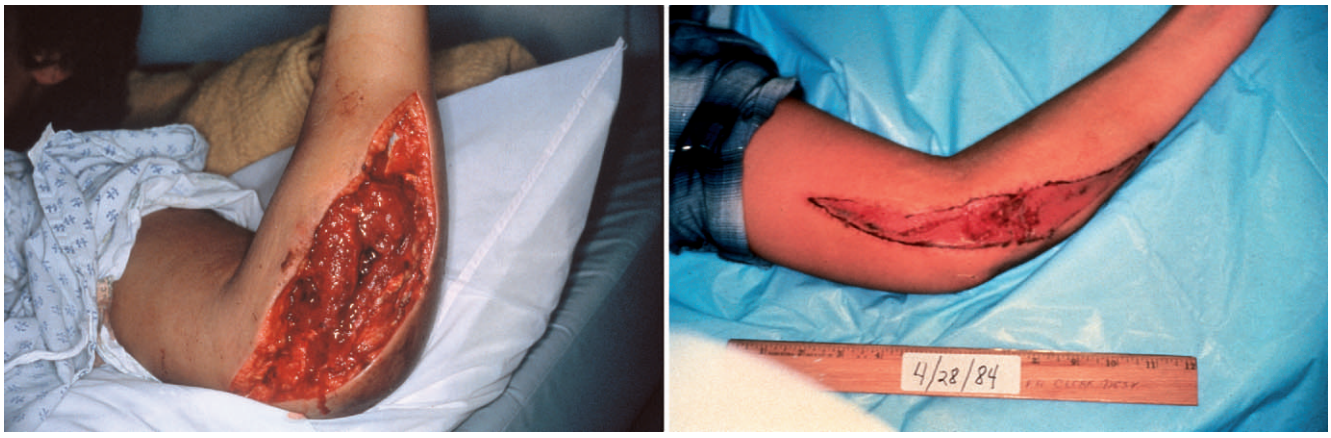
Supplemental oxygen is the standard treatment for CO poisoning. Oxygen hastens dissociation of CO from hemoglobin and provides enhanced tissue oxygenation. Use of HBOT for the treatment of CO poisoning is based on the following mechanisms:

1. HBOT accelerates the rate of CO dissociation from hemoglobin; COHb half-life is decreased from 5.5 hours breathing air to 23 minutes breathing 100% oxygen at 3 ATA.[18,120]
2. HBOT at 3 ATA accelerates dissociation of CO from cytochrome oxidase, thereby improving oxidative phosphorylation.[20]
3. HBOT can prevent CO-mediated brain lipid peroxidation and prevent leukocyte-mediated inflammatory changes in the brain by inhibition of leukocyte beta$_2$ interferons.[154,155] The CO neurotoxicity may be mediated by lipid peroxidation in the CNS.
4. HBOT decreases cerebral edema and maintains adequate cerebral oxygen delivery.

Animal models of CO poisoning and HBOT demonstrate more rapid improvement in cardiovascular status, lower mortality, and lower incidence of neurologic sequelae.[69,122]

HBOT is recommended for patients with COHb greater than 25% or with signs of serious CO poisoning, including prolonged unconsciousness, neurologic symptoms, cardiovascular dysfunction, or severe acidosis, irrespective of COHb level. HBOT should be used more liberally in the pregnant patient because of enhanced fetal vulnerability to CO and hypoxia.[166] Controversy exists surrounding HBOT for mild to moderate cases of CO poisoning and whether HBOT can prevent the DNS. Six randomized, controlled clinical trials in acute CO poisoning had conflicting results; however, the studies varied in quality, study design, and outcomes. The studies that showed no benefit from HBOT had long treatment delays and poor follow-up.[36,98,129,137,157,175] The largest randomized clinical trial with the best study design, and follow-up has demonstrated a significant benefit of HBOT in decreasing the incidence of DNS.[175] A recent review article discusses the state of the art in acute CO poisoning, including recommendations for HBOT and unanswered questions.[127] Recommended treatment protocol for CO poisoning is 2.4 to 3 ATA for 90 to 120 minutes for a single treatment, reserving repetitive treatments for residual neurologic symptoms.

Use of HBOT in cyanide poisoning is less clear. Hydrogen cyanide (HCN) often accompanies CO poisoning; the toxic effects of cyanide and CO are synergistic.[116] Treatment for HCN



A B

Figure 71-11. Gas gangrene due to *Clostridium perfringens*. **A,** The patient presented with myonecrosis from gas gangrene and was aggressively treated with surgical debridement, antibiotics, and hyperbaric oxygen therapy. **B,** Once her wound was stabilized, a graft was placed to cover the wound.

poisoning includes amyl nitrite, sodium nitrite (which oxidizes the iron in hemoglobin to generate methemoglobin), and sodium thiosulfate. HBO may have a direct effect in reducing the toxicity of cyanide and maintaining adequate oxygen delivery to the brain during peak insult from HCN and after induction of significant amounts of methemoglobin with sodium nitrate. The use of HBOT is supported only by anecdotal cases; the clinical literature is limited.[48,138]

Necrotizing Soft Tissue Infections

Clostridial Myonecrosis (Gas Gangrene)

Invasive clostridial infection usually results from injury or by contamination of wounds. Clostridial myositis with myonecrosis or gas gangrene is an acute, rapidly progressive invasive infection of muscles characterized by toxemia, edema, tissue death, and gas production. *Clostridium* is an anaerobic, spore-forming gram-positive encapsulated bacillus. The most common species isolated is *C. perfringens* (isolated in 80% to 90% of wounds); others include *C. novyi, C. septicum,* and *C. histolyticum*. *C. perfringens* is a facultative anaerobe that can multiply freely in tissues with an oxygen tension of 30 mm Hg. It demonstrates restricted growth at oxygen tensions of 70 mm Hg. Although more than 20 toxins are produced, the most prevalent and lethal is alpha-toxin. HBOT does not kill clostridial organisms; it has a bacteriostatic effect and at oxygen tensions of 250 mm Hg inhibits alpha-toxin production.[167]

Treatment for clostridial infections consists of surgical debridement, antibiotics, and HBOT as soon as possible to stop alpha-toxin production and to save potentially viable tissue. Multiple retrospective clinical studies report improved overall survival and decreased rate of amputation. The lowest morbidity and mortality are achieved with initial conservative surgery and rapid initiation of HBO therapy.[49,62,79,124,130] This approach is not only life saving, but it is limb and tissue saving, because no major amputations or excisions are done prematurely, and demarcation of dead tissue is allowed. HBOT for gas gangrene should be given at 3 ATA every 8 hours for the first 24 hours, then twice per day at 2.4 to 3.0 ATA for 2 to 5 days. When daily assessment reveals no further evidence of ongoing tissue necrosis, HBOT can be stopped. Figure 71-11 shows a young female who fell while riding her horse and sustained gas gangrene due to *C. perfringens*. After aggressive surgical debridement, antibiotics, and HBOT, a graft was placed to cover her wound, and she made an excellent recovery.

Necrotizing Fasciitis

HBOT is recommended as an adjunct to surgical and antibiotic treatment for necrotizing soft tissue infections (including crepitant anaerobic cellulitis, progressive bacterial gangrene, necrotizing fasciitis, Fournier's gangrene, and nonclostridial myonecrosis), particularly for compromised hosts when mortality is expected to be high. These infections are typically mixed aerobic–anaerobic infections of the subcutaneous tissues, fascia, and muscle. HBOT adversely affects anaerobic bacterial growth and improves neutrophil function. The hyperoxygenated tissue zone surrounding the infected area may be of significance in preventing extension of invading microorganisms. In humans, HBOT in combination with surgery and antibiotics can reduce morbidity and mortality.[49,61,79,130] Although there are no randomized, controlled clinical trials with HBOT, case studies and retrospective reviews have shown a reduction in mortality and reduced number of debridements. HBOT may be life and limb saving when used early in the course of these infections.

HBOT for necrotizing fasciitis is given at 2.0 to 2.5 ATA for 90 to 120 minutes twice daily until the patient has stabilized and the infection is controlled. If the infection is severe, three treatments at 3.0 ATA are recommended in the first 24 hours, followed by twice-daily treatments up to 30 treatments.

Crush Injuries, Compartment Syndrome, and Acute Ischemias

HBOT is a therapeutic adjunct to treatment of crush injury and acute traumatic ischemia. Post-traumatic cytogenic and vasogenic edema reduce oxygenation of tissues contributing to tissue hypoxia and ischemia. There may be secondary tissue destruction caused by reperfusion injury. HBOT increases tissue oxygen tension in hypoxic tissue. HBOT causes vasoconstriction and edema reduction while maintaining hyperoxygenation.

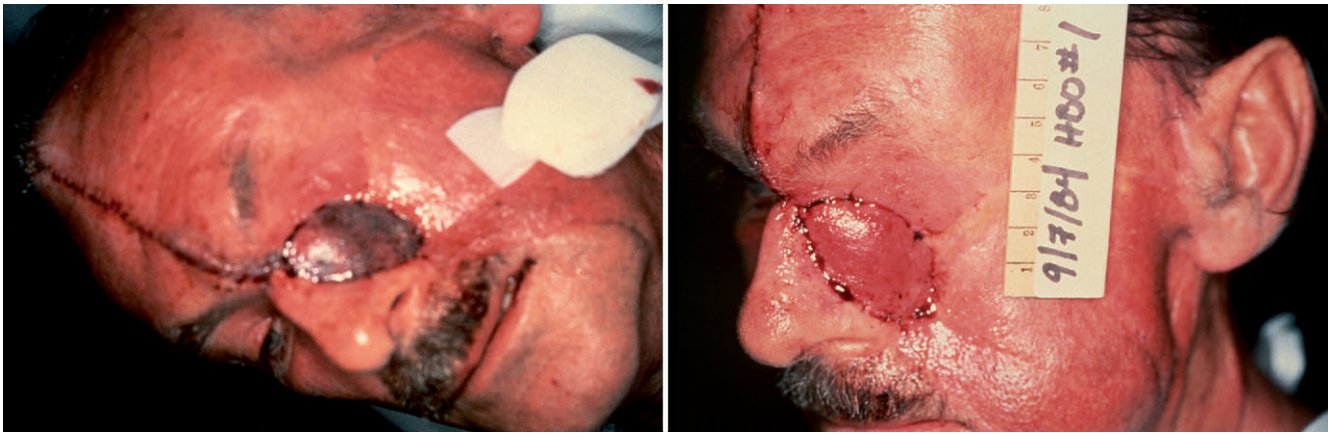


Figure 71-12. Hyperbaric oxygen therapy and compromised flap. **A,** Shortly after surgery, the flap appeared dusky and ischemic. **B,** The patient was treated with one hyperbaric treatment and had immediate improvement in color and blood flow to the wound.

Blood flow in the microcirculation is improved through decreasing interstitial fluid pressures from reduction in edema. Additionally, HBOT protects tissues from reperfusion injury that accompanies crush injuries, compartment syndrome, and acute traumatic ischemia.

Numerous animal studies and clinical series support the use of HBOT for acute peripheral ischemia, crush injuries, and compartment syndrome.[117,142,149,181] A randomized, double-blinded clinical trial in crush injuries showed 94% healing in the HBOT group compared to 59% ($p < 0.01$) in the controls.[15] Early application of HBOT within 4 to 6 hours of injury or ischemia is essential. HBOT is administered at 2.0 to 2.5 ATA for 90 to 120 minutes. The number of treatments depends on the pathophysiology. Patients with crush injuries require HBOT three times a day for the first 2 days, two times a day for the next 2 days, and daily for an additional 2 days. Suspected reperfusion injuries after reimplantations, free flaps, or transient ischemia only require 1 or 2 treatments. Signs of impending compartment syndrome include increasing pain, particularly with passive stretch, tense compartment, and hyperesthesia or weakness in the distal part of an extremity. If compartment pressures indicate that fasciotomy is not required, HBOT should be given three times within 24 hours. If clinical indications and compartment pressure measurements dictate immediate fasciotomy, HBOT can be used after surgery to reduce morbidity from residual injury from swelling and ischemia.

Exceptional Anemia

Exceptional anemia occurs when loss of sufficient red cell mass to compromise tissue oxygenation occurs in a patient who, for medical, personal, or religious reasons, cannot or will not receive a blood transfusion. HBOT can dissolve enough oxygen to meet the oxygen requirements of the tissues in the severely anemic patient until the bone marrow restores hemoglobin and red cell mass. Animal studies demonstrate the beneficial effect of HBOT in hemorrhagic shock models.[13,97,178] HBOT has been used successfully to correct accumulating oxygen debt in untransfusible patients and to reverse organ dysfunction in severe anemia associated with hemorrhagic shock.[51,105] The best published series for the use of HBOT for treatment of exceptional anemia is reported by Hart; 26 patients with severe exceptional blood loss anemia, class IV hemorrhage, demonstrated a 70% survival.[58] A systematic review of the use of HBOT for treatment of severe anemia was recently published.[165] HBOT may be used concurrently with erythropoietin, fluorocarbons, or stroma-free polymerized hemoglobin.

Compromised Skin Grafts and Flaps

HBOT is extremely useful in flap salvage when tissue is compromised by decreased perfusion, irradiation, or hypoxia. HBOT can help maximize viability of compromised grafts and flaps by improving tissue oxygenation and increasing flap capillary density. Experimental animal studies since 1966 have shown improved skin flap survival in animals treated with HBOT compared to controls.[104,113] In an ischemic flap model in rats, HBOT increased microvascular blood flow during reperfusion compared with untreated ischemic controls by reducing neutrophil endothelial adherence in venules and blocking progressive arteriolar vasoconstriction associated with reperfusion injury[181] A beneficial effect of HBOT has been described in numerous case studies and in one controlled clinical trial. Flap survival with HBOT averages 90%, compared with other studies with failure rates as high as 67% in compromised tissues.[16,123,174]

HBOT is most effective when started as soon as signs of flap compromise appear. Initial treatments should be twice daily at a pressure of 2.0 to 2.5 ATA for 90 to 120 minutes. Once the graft or flap appears more viable, HBOT can be given once a day until stable. The average number of treatments varies from 10 to 20. In patients who have had previous graft or flap failure, 20 treatments prior to flap placement can prepare the site with granulation tissue, followed by 10 to 20 treatments after flap or graft placement to ensure survival of the tissue. Figure 71-12 shows a dusky, ischemic flap after surgery that improved dramatically with one HBOT treatment.

Problem Wound Healing

Problem or chronic wounds are usually present in a compromised host and fail to respond to medical and surgical management. Such wounds include venous leg ulcers, pressure ulcers, and foot ulcers in patient with diabetes or significant

Figure 71-13. Diabetic foot wound. **A,** 67-year-old woman with exposed tendon at the base of her left foot wound. **B,** After 50 hyperbaric treatments, she had complete closure of her wound, eliminating the need for amputation.

vascular disease. Local tissue hypoxia, hypoperfusion, and infection are underlying problems in these wounds. The use of HBOT to restore tissue Po_2 into the normal range can stimulate fibroblast proliferation, collagen synthesis, neutrophil oxidative killing of microorganisms, and angiogenesis.

The most common problem wound treated with HBOT is the diabetic foot wound. Evidence-based reviews have addressed the effectiveness of HBOT.[80,132,172] Five randomized, controlled clinical trials of HBOT in diabetic lower extremity wounds reported improved wound healing and lower amputation rate in HBOT groups compared with controls.[1,34,37,71,74] Other treatments to hasten recovery include negative pressure wound therapy (wound VAC [vacuum-assisted closure]), bioengineered tissue grafts, topical debriding agents, platelet derived growth factor, and surgical closure in combination with HBOT. Not all wounds should be treated with HBOT. For example, HBOT is not a substitute for surgical revascularization in arterial insufficiency, for which a comprehensive, multidisciplinary approach is advisable.

HBOT is administered at 2.0 to 2.5 ATA for 90 to 120 minutes once or twice daily. Patients hospitalized with limb-threatening infections or significant peripheral arterial occlusive disease may require twice daily treatments until stabilized. Many of these wounds are slow to respond and can require 30 to 60 HBO treatments. Appropriate wound care is extremely important to facilitate healing, and many hyperbaric chambers are often associated with wound care centers.

Figure 71-13 shows a 67-year-old female with diabetes mellitus and peripheral vascular disease with a nonhealing wound to her left foot with exposed tendon at the base of the wound. She refused amputation and was treated with 50 hyperbaric treatments. She had granulation of her wound and eventual epithelialization and closure.

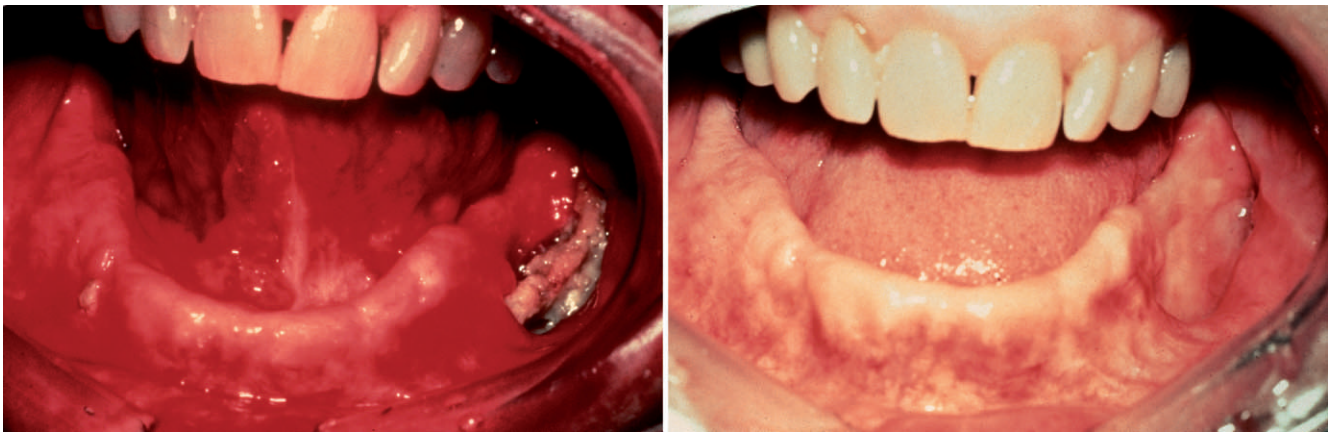
Delayed Radiation Injuries (Soft Tissue and Bony Necrosis)

Radiation kills normal tissue and causes loss of vascularity and cellularity. Delayed radiation complications are seen after a latent period of 6 months or more and may develop many years after exposure. Radiation leads to progressive obliterative endarteritis with resultant tissue ischemia and fibrosis. Dental extractions and other surgical procedures are associated with high complication rates when done in heavily irradiated tissues. HBOT has been used prophylactically before oral surgery in a radiated tissue field and to treat delayed radiation tissue injury. HBOT stimulates fibroplasia, angiogenesis and increased cellularity in radiated tissue by mechanisms similar to those described above for wound healing.[109] HBOT has been used successfully for many years in treatment of mandibular osteoradionecrosis (ORN) and, more recently, for radiation injuries at other sites.

From randomized, prospective trials by Robert Marx, DDS, of bone grafts to irradiated tissue in humans, a standard protocol has been developed for use of HBOT when surgery is performed in radiated tissue.[40,95,96] This protocol of 20 to 30 preoperative HBOT treatments followed by 10 postoperative treatments has been shown to be effective for head and neck after radiation surgery, including bone graft reconstruction, soft tissue vascular flaps, and tooth extraction. Figure 71-14 demonstrates improvement in ORN with use of HBOT.

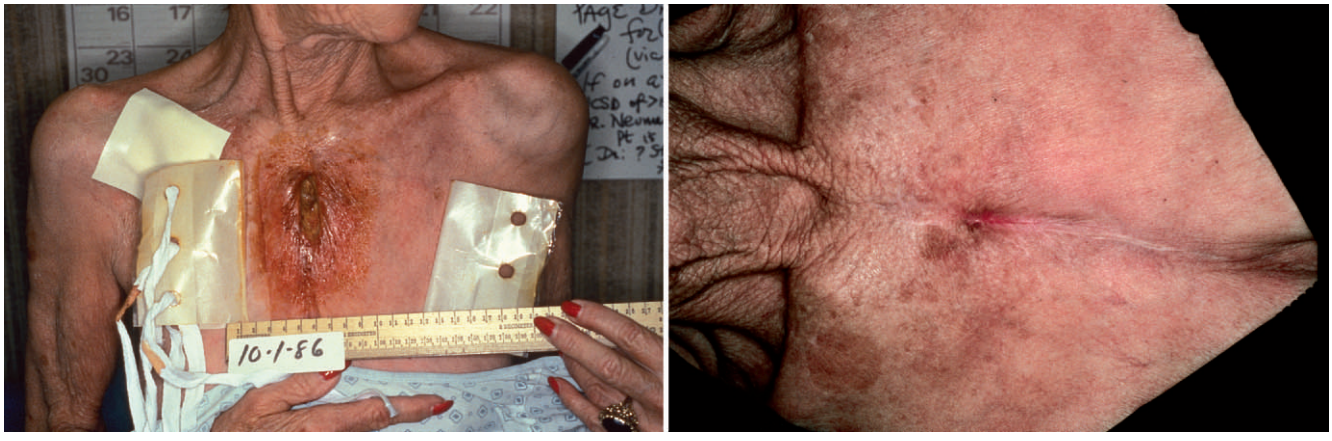
This clinical success has led scientists to apply HBOT to radiation injuries at other sites. HBOT has been shown to be beneficial in other forms of soft tissue radionecrosis, including radiation proctitis, enteritis, and cystitis.[10,17,25,41] Additionally, HBOT is useful in treatment of laryngeal and tracheal radionecrosis and in soft tissue necrosis of the head, neck, and chest wall, the latter particularly after mastectomy.[23,44] A more recent application has been use of HBOT to treat neurologic injuries secondary to radiation, including transverse myelitis, brain necrosis, optic nerve injury and brachial plexopathy.[40] Although evidence of beneficial HBOT for radiation-induced neurologic injuries is anecdotal, given the severe consequences and progression of injury in the CNS, serious consideration for HBOT should be given.

HBOT for radiation injury is administered at 2.0 to 2.5 ATA daily for 20 to 40 treatments. Some patients benefit from additional treatments, and utilization review should occur at 60 treatments. To prevent ORN in patients who require extraction



A B

Figure 71-14. Patient with osteoradionecrosis of the mandible before **(A)** and after **(B)** hyperbaric oxygen therapy.



A B

Figure 71-15. **A,** Woman with sternal osteomyelitis following coronary artery bypass graft that failed repeated debridements and two 6-week courses of IV antibiotics. **B,** After 40 sessions of hyperbaric oxygen therapy, she had complete closure of her wound and resolution of osteomyelitis.

of teeth in previously irradiated jaws, they should receive 20 treatments before extraction followed by 10 treatments after extraction. If ORN is already present, the treatment protocol changes to 30 pre-extraction treatments or surgical resection followed by 10 post-resection treatments.

Refractory Osteomyelitis

Refractory osteomyelitis recurs or persists after standard therapy with aggressive surgical debridement and antibiotics. HBOT can elevate tissue oxygen tensions in infected bone to normal or above normal levels.[93] Osteoclast function of removing necrotic bone is an oxygen-dependent process; osteoclasts do not function properly in hypoxic bone. HBOT has a stimulatory effect on osteoclast function.[106,109] Experimental animal models have demonstrated the benefit of HBO for difficult cases of osteomyelitis.[106,161] However, no prospective, randomized human trial has been conducted. Recent clinical reports have shown long-term success rates with HBOT. Of patients who failed to respond to repetitive surgery and antibiotic care, 63% to 86% have had infections successfully arrested with addition of HBOT.[2,24,102] HBOT is indicated in patients who fail to respond to surgical debridement and appropriate antibiotics or in whom osteomyelitis recurs after appropriate management.

HBOT is used as an adjunct to standard surgical and antibiotic treatment for refractory osteomyelitis. Although most studies consider extremity infections, HBOT is effective in management of chronic sternal wound osteomyelitis.[125] Figure 71-15 shows a woman who developed sternal osteomyelitis after coronary artery bypass grafting. She was treated with two debridements and two separate 6-week courses of IV antibiotics. There was no improvement in her wound, and she was eventually referred for HBOT. After 40 treatments, she had complete resolution of her sternal infection and closure of her wound. Additional indications include refractory osteomyelitis

of the jaw and malignant otitis externa, a progressive and potentially fatal *Pseudomonas aeruginosa* osteomyelitis involving the external auditory meatus with osteomyelitis of the temporal bone.[32] HBOT is administered at 2.0 to 2.5 ATA for 90 to 120 minutes once per day, for an average of 20 to 40 treatments.

Thermal Burns

HBOT is used as an adjunct for treatment of thermal burns and has been shown to improve morbidity and mortality, reduce length of hospital stay, and decrease the need for surgery. The rationale for use of HBOT is to minimize edema, enhance host defenses, preserve marginally viable tissue, and improve wound healing. Experimental data have shown HBOT reduces generalized edema and wound size in animals treated.[70,118] Additionally, HBOT has a beneficial effect on angiogenesis and epithelial regeneration.[11] Biopsies of burned animals showed progression to full-thickness injury in control animals and preservation of dermal elements and capillary patency in HBOT-treated animals.[147]

Clinical experience in humans suggests improved wound healing, reduction in mortality, decrease in surgical procedures and resuscitative fluid requirements, and reduced hospital stay in burn patients who received adjunctive HBOT.[27,28,29,55] In a prospective, randomized, controlled, double-blinded trial comparing HBOT with sham controls in a human burn model, Niezgoda and colleagues demonstrated significant reduction of wound size, hyperemia, and wound exudates in the HBOT group.[114] Current recommendations include serious burns greater than 20% total body surface area or involvement of the hands, face, feet, or perineum that are deep partial- or full-thickness injuries. Although use of HBOT for burn treatment is approved by the UHMS, some burn centers around the country do not use HBOT, except for CO poisoning. A recent Cochrane review found insufficient evidence to support or refute use of HBOT in management of thermal burns.[170]

HBOT should be initiated as soon as possible after injury. Initially, three treatments at 2.0 to 2.5 ATA for 90 minutes of oxygen delivery within the first 24 hours are recommended, then twice daily treatments for 10 to 14 days. Further treatments are often used to optimize graft uptake. Rarely do patients need more than 50 treatments. Careful attention to fluid management is critical because initial fluid requirements of burn patients can be several liters per hour. Ambient temperature in the chamber should be comfortable because thermal instability can be problematic, particularly within 1 to 2 hours of burn wound dressing changes. In cases of concurrent inhalation injury, patients can be maintained on ventilator support during treatment.

Intracranial Abscess

HBOT as an adjunct to treatment for intracranial abscess (ICA) is the most recent addition to approved indications for HBOT. The overall mortality rate for ICA is 20%. In certain patients with complications or in patients who pose therapeutic problems, HBOT has been beneficial when used as an adjunct. Approximately 48 cases have been reported in the literature, with an overall mortality rate of 2%.[82,83,84,99,151] Proposed mechanisms of HBOT in ICA include reduction in brain swelling, inhibition of anaerobic flora found in ICA, enhancement of host defense mechanisms, and treatment of concomitant skull osteomyelitis. HBOT has been recommended as a complement to currently accepted standard procedures in conditions with multiple abscesses, abscess in a deep or dominant location, compromised host, when surgery is contraindicated or when the patient is a poor surgical candidate, or with no response or further deterioration in spite of standard surgical and antibiotic treatment.[39] Because of significant mortality and long-term sequelae, it is unlikely that a rigid, human, double-blind, controlled study can be done. The UHMS HBOT Committee is collecting case reports.

For the treatment of ICA, HBOT is administered at 2.0 to 2.5 ATA, with 60 to 90 minutes of oxygen administration per treatment. Initially, HBOT should be administered twice daily depending on the condition of the patient. The optimal number of treatments is unknown; treatments should be based on the patient's clinical response and radiologic findings. In reported series, the average number of HBOT sessions was thirteen. Utilization review is recommended after 20 treatments.[39]

EMERGING RESEARCH

Acute Myocardial Ischemia

In animal studies, HBOT reduces the ischemic effect of coronary artery occlusion.[146,158] Thomas and coworkers demonstrated that a combination of thrombolytic therapy and HBOT is more effective in reducing size of myocardial infarction than either treatment alone.[158] In a rabbit model of aortic atherosclerosis, HBOT not only halted progression of disease but also accelerated regression of atherosclerotic lesions.[81] HBOT may lessen or inhibit reperfusion injury by protecting oxidative metabolism in reperfusion-stunned myocardium. The Hyperbaric Oxygen and Thrombolysis in Myocardial Infarction (HOTMI) study showed that treatment with HBO in combination with thrombolysis resulted in attenuated creatine phosphokinase rise, more rapid resolution of pain, and improved ejection fraction (EF).[145] Dekleva and colleagues demonstrated that HBOT in conjunction with streptokinase in acute MI reduced left ventricular volumes with associated increases in EF.[33] In a controlled trial, patients who underwent percutaneous coronary intervention (PCI) for acute MI or unstable angina and received HBOT in the early peri-PCI period had a lower clinical restenosis rate and less frequent development of late anginal symptoms.[139] The authors postulate that HBOT may induce expression of antioxidant enzymes that offer protection against atherosclerosis and reduce oxidation products in high-density lipoproteins. Additionally, endothelial cell–derived fibrinolysis and blood flow are enhanced by HBOT, which could result in reduction in recurrent thrombosis.[159] These results are provocative; larger randomized trials are indicated.

Acute Cerebral Ischemia

Effectiveness of HBOT in cerebral ischemia is controversial. It is difficult to compare studies due to different stroke models in different species with different HBOT protocols. Recent work demonstrates that HBOT appears to be protective in various models of focal cerebral ischemia.[144,150,179] In three recent reports utilizing magnetic resonance imaging (MRI) scans in an animal model, HBOT given 40 minutes to 6 hours after stroke onset was neuroprotective; HBOT improved infarct volume reduction and neurologic outcome.[89,136,168] Early HBOT may stop the process of ischemic infarct growth by rapidly restoring oxygen and energy to ischemic but still viable brain tissue. Other proposed mechanisms of HBOT include reduction of extracellular dopamine and inhibition of cyclooxygenase-2 (COX-2) overexpression in the cerebral cortex.[179,180] Clinical studies on humans

have failed to show a significant benefit from HBO, probably due to the long time window between symptom onset and HBO therapy.[115,134]

WILDERNESS MEDICINE APPLICATIONS OF HBOT

HBOT plays a significant role in wilderness medicine for treatment of diving-related illnesses and altitude illness. HBO has been investigated in other wilderness-related entities, such as frostbite, brown recluse spider bite, and more recently, heatstroke. HBOT has also been successful in treating necrotizing soft tissue infection from a stingray puncture and *Vibrio vulnificus* septicemia and cellulitis from eating raw fish.[131,173] HBOT has been tried in an experimental model of sea nettle envenomation in a rat model. Although it had no effect on mortality, HBOT protected against venom-induced decreases in brain blood flow and maintained oxygenation in envenomed animals.[110]

Frostbite

The first case of HBOT in cold injury was reported by Ledingham in 1963, followed by a number of reports for its use in the 1970s. There are numerous case reports available, but control data are lacking. Initial experiments in rabbits showed that HBOT had no effect on tissue loss and variation of injury.[46,47,56] However, other studies have shown benefit. If HBOT was administered immediately after rewarming, mean tissue loss was decreased in rabbits. This benefit was significantly reduced by delays in treatment, particularly beyond 24 hours.[47,119] Research into the pathophysiology of frostbite has revealed marked similarities to inflammatory processes seen in thermal burns and ischemia-reperfusion injury.[112] Hence, HBOT has theoretical advantages as an adjunct in the treatment of frostbite. An 11-year-old boy who suffered deep frostbite on six fingers was treated with HBOT and had complete recovery of his hands after 14 days.[171] In another report, HBOT was shown to improve skin microcirculation by increasing the number of nutrient capillaries in frostbitten areas on the toes.[45]

The ideal time to initiate HBOT for frostbite is during the rewarming period because of the reperfusion aspect of the injury. Although there is no standard protocol, HBOT can be given at 2.0 to 2.5 ATA for 90 to 120 minutes three times in the first 24 hours, followed by twice-daily treatments until there is no longer threatened tissue loss. If a frostbitten limb has already been rewarmed and is at risk for significant tissue loss, HBOT is still indicated to help hasten demarcation, decrease risk of infection, enhance survival of the damaged tissue in the gradient of injury, and improve flap survival.

Brown Recluse Spider Bite

The brown recluse spider *(Loxosceles reclusa)*, found commonly in the southern United States, is known to cause necrotic skin lesions in humans. Most bites resolve spontaneously, but a few progress to severe local necrosis and tissue loss (see Chapter 46). Treatment of brown recluse spider bites with HBOT remains controversial. HBOT has been proposed to directly inhibit neutrophil adherence to the endothelium and to decrease venom-induced ischemia-reperfusion injury, as well as perhaps inactivate sphingomyelinase or other components of the venom.[107] HBOT may minimize wound size by increasing O_2 tension within the wound and thereby increase angiogenesis and fibroblast proliferation.

In animal models, the data are conflicting. In experimental models using rabbits, there was neither reduction in lesion size nor histologic improvement from HBOT.[126] Similarly, there was no decrease in lesion size in a swine model.[63] Two reports have shown benefit in controlled animal studies. One showed histologic improvement in a rabbit model, but found no difference in lesion size.[148] A randomized, controlled trial using rabbits demonstrated that HBOT reduced the size of lesions, even when delayed by 2 days.[101] A single treatment given immediately was as effective as multiple treatments.

Several case studies in humans suggest that HBOT may be beneficial in managing dermatonecrosis associated with *L. reclusa*. Svendsen first reported use of HBOT in the treatment of brown recluse spider bites in 1986.[152] He treated 6 patients who had clinically deteriorating lesions with HBOT 2 to 6 days after the bite with twice-daily treatments for 3 days. All lesions healed well without surgery, skin sloughing, or significant scarring. Kendall and Canigalia reported 47 cases with good outcomes; only 1 patient out of 48 required grafting.[73] Mayor and colleagues treated 14 patients, who all healed without scarring, disability, or need for skin grafting.[100] Two other more recent cases of potentially devastating bites, one to the glans penis and another to the periorbital region on a child's face, were both treated with HBOT and had good outcomes.[19,68]

Treatment of brown recluse spider bites with HBOT may be effective at reducing scarring and complications. However, there are no randomized, controlled trials of HBOT in human cases of *Loxosceles* envenomation and no standardized HBOT protocols. HBOT can be given at 2.0 to 2.5 ATA for 90 to 120 minutes on a daily basis for 2 to 10 treatments. Systemic symptoms, size of the dermatonecrotic lesion, and surrounding erythema should be assessed on a daily basis.

Heatstroke

Heatstroke is characterized by significant hyperthermia, altered mental status, and varying degrees of multiple organ failure. In rodents, heatstroke causes vasoplegic shock, intracranial hypertension, and cerebral ischemia and injury.[87] In a recent experimental study on rats, HBOT was found to be beneficial in resuscitating rats with experimental heatstroke. HBOT reduced heatstroke-induced arterial hypotension, hypoxia, plasma tumor necrosis factor-alpha overproduction, and cerebral ischemia. HBOT improved survival during heatstroke by augmenting mean arterial pressure and local cerebral blood flow.[162] Currently, HBOT is not standard therapy for heatstroke. Further work is needed to support its use as an adjunct to conventional treatment options in severe cases.

Field Treatment of Combat Trauma

HBOT has been proposed for the treatment of combat-related trauma.[57,92] Hart reviewed preliminary wound statistics from operation Iraqi Freedom.[57] Approximately 70% of the injured soldiers sustained extremity trauma. Of 560 surgical procedures performed, 31% were complicated by persistent tissue necrosis, wound infection, graft failure, or delayed wound healing. HBOT is effective in correcting tissue ischemia and hypoxia, as an adjunctive treatment for compartment syndrome, and for controlling wound infections and augmenting healing. Field use of HBOT for combat injuries could limit the extent of surgical debridements, improve tissue flap and graft survival, decrease wound infection rates, and improve complex wound healing. HBOT could be used as a temporizing measure pending operating room availability.

Several portable hyperbaric chambers have been suggested for use in field treatment. The Hyperlite or EEHS is ideal for remote locations without hospital support. The USN's Transportable Recompression Chamber System (TRCS) is a multiplace chamber designed for the treatment and evacuation of injured divers, but can be used for any emergency hyperbaric treatment; it can be moved to almost any location. The Fly Away Recompression Chamber (FARC) is a much larger system consisting of a 200 cubic foot (61 m) double-lock recompression chamber capable of treating divers or any hyperbaric patient. A multiplace hyperbaric chamber could be put aboard a designated hospital ship for the treatment of combat wounds.

The references for this chapter can be found on the accompanying DVD-ROM.

72 Injuries from Nonvenomous Aquatic Animals

Paul S. Auerbach and George H. Burgess

The expanses of ocean and freshwater that cover the earth are the greatest wilderness. Seventy-one percent (or 139,768,981 million square miles [362 million km^2]) of the earth's surface is composed of ocean, the volume of which exceeds 325 million cubic miles (523 km^3). Underneath the surface lie huge mountain ranges, deep valleys, and many active volcanoes. Nearly half of the sea floor is composed of the *abyssal plain,* which lies at an average depth of 2.5 miles (4 km) and is largely devoid of life forms. The December 2004 tsunami that devastated parts of Thailand, Sri Lanka, and India was generated by an undersea earthquake of Richter magnitude 9.0. Within the undersea realm exist four fifths of all living organisms. We are aware of 275,000 ocean species.

The opportunity for direct encounters with aquatic organisms continues to increase because of enhanced recreational, industrial, scientific, and military oceanic and riverine activities related to ever-rising human populations. The most common cause of injuries is handling of animals that bite and sting in self-defense, followed by provoked, then unprovoked attacks.

Nearly 80% of the world's population resides in coastal regions. In the United States, 50% of the population lives within 50 miles (80 km) of a coastline. It is estimated that 127 million U.S. citizens will live along the coasts by the year 2010. A significant proportion of this population will be directly involved as entrants into the aquatic world. Therefore, it is imperative that clinicians be familiar with hazards unique to the aquatic environment.

Although noxious marine organisms are concentrated predominantly in warm temperate and tropical seas, particularly in the Indo-Pacific region, hazardous animals may be found as far north as 50° latitude. Saltwater aquariums in private homes and public settings, intercontinental seafood shipping, and accessibility of air travel to sport scuba and skin divers contribute to the risks.

Like the rainforest, the ocean depths have the potential to reveal virtually limitless active pharmaceutical agents. For example, genetically engineered reproduction of the adhesive protein of the marine mussel *Mytilus edulis* has created a tissue adhesive agent that may one day prove superior to cyanoacrylic compounds. Toxins isolated from ascidians (tunicates, or sea squirts) include cyclic peptides, some of which (esteinascidin 743, aplidine) have undergone evaluation for cancer chemotherapy; others (e.g., thiocoraline and kahalalide F) may follow. Investigative techniques continue to improve. In pursuit of anatomic information that can elucidate the biology and ecology of fish, evolution, cellular physiology, and aquatic models of human disease, nuclear radiologists have performed in vivo nuclear magnetic resonance imaging (MRI) and spectroscopy of anesthetized (tricaine methsulfonate [MS222]) aquatic organisms.[22] Most marine organisms rely on antimicrobial components of their innate immune defenses to combat pathogens. From this unique perspective, scientists seek to identify novel antimicrobials, among which the most promising are marine cationic antimicrobial peptides, defined as small (10 to 40 amino acids) peptides containing a prevalence of positively charged residues (lysine and arginine).[102] As examples, these have been found in teleost fishes (pardaxin, pleurocidin, hepcidin), tunicates (styelin, clavanin), chelicerates (big defensin,

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tachyplesin), crustaceans (callinectin), gastropods (dolabellanin), and mollusks (mytilin). Their activities include synergy to induce cell lysis, modulating the host immune response, chemotaxis, macrophage development, production or inhibition of cytokines, and so forth.

Despite the wondrous nature of the deep, danger exists. The ubiquity of hazardous creatures and their propensity to appear at inopportune times make it imperative to be aware of them, to respect their territorial rights, and to avoid unpleasant contact with them.

DIVISIONS AND DEFINITIONS

Dangerous aquatic animals are divided into four groups: (1) those that bite, rip, puncture, or deliver an electric shock without envenomation; (2) those that sting (envenom), discussed in Chapters 73 and 74; (3) those that are poisonous on ingestion (see Chapter 66); and (4) those that induce allergies (see Chapter 67). Aquatic skin disorders are discussed in Chapter 75.

IN DEFENSE OF THE FISH

As in all nature (except for humans), indiscriminate aggression is rarely involved when injuries are inflicted by aquatic animals. Most injuries result from gestures of warning or self-defense; with the exception of some sharks, few aquatic creatures attack humans without provocation. Attacks are made in defense of young, in territorial dispute, or to procure food. *Caution* is the key word when dealing with potentially injurious aquatic creatures.

GENERAL PRINCIPLES OF FIRST AID

The physician must adhere to fundamental principles of medical rescue. Although many injuries and envenomations have unique clinical presentations, the cornerstone of therapy is immediate attention to the airway, breathing, and circulation. Along with specific interventions directed against a particular venom or poison, the rescuer must simultaneously be certain that the victim maintains a patent airway, breathes spontaneously or with assistance, and is supported by an adequate blood pressure. Because marine attacks and envenomations often affect swimmers and divers, the rescuer should anticipate near-drowning (see Chapter 68), immersion hypothermia (see Chapter 6), and decompression sickness or arterial air embolism (see Chapter 70). Any victim rescued from the ocean for any reason should be thoroughly examined for external signs of a bite, puncture, or sting.

WOUND MANAGEMENT

Whether the injury is a bite, abrasion, or puncture, meticulous attention to basic wound management is necessary to minimize posttraumatic infection.

Wound Irrigation

All wounds acquired in the natural aquatic environment should be vigorously irrigated with sterile diluent, preferably normal saline (0.9% sodium chloride) solution. Seawater is not recommended as an irrigant because it carries a hypothetical infection risk. Sterile water or hypotonic saline is acceptable. Tap water (preferably disinfected) appears to be a suitable irrigant and should be used when the alternative is delay to irrigation.[5] Irrigation should be performed before and after debridement to maximize the benefits. A 19-gauge needle or 18-gauge plastic intravenous (IV) catheter attached to a syringe that delivers a pressure of 10 to 20 psi will dislodge most bacteria without forcing irrigation fluid into tissue along the wound edges or deeper along dissecting tissue planes. Convenient ring-handle syringes with blunt irrigation tips and IV tubing that connects to standard IV bags are useful. At least 100 to 250 mL of irrigant should be flushed through each wound. If a laceration is from a stingray, proteinaceous (and possibly) heat-labile venom may be present in the wound. Therefore, if the wound is still painful at the time of irrigation, the irrigant may be warmed to 45°C (113°F).[9]

Antiseptic may be added to the irrigant if the wound appears to be highly contaminated. Povidone-iodine solution in a concentration of 1% to 5% may be used with a contact time of 1 to 5 minutes.[97,145] When antiseptic irrigation is completed, the wound should be thoroughly irrigated with normal saline or tap water to minimize tissue toxicity from the antiseptic. Antiseptics that are particularly harmful to tissues include full-strength hydrogen peroxide, povidone-iodine scrub solution, hexachlorophene detergent, and silver nitrate.

Scrubbing should be used to remove debris that cannot be irrigated from the wound. Sharp surgical debridement is preferable to sponge scrubbing, which may increase infection rates, particularly when applied with harsh antiseptic solutions. Poloxamer 188 (Pluronic F-68), a nontoxic, nonionic surfactant skin wound cleanser (found in Shur-Clens 20%) that may be used to irrigate or scrub wounds, does not offer any significant advantage over traditional sterile saline irrigation.

Wound Debridement

Debridement is more effective than irrigation at removing bacteria and debris. Crushed or devitalized tissue should be removed with sharp dissection to provide clean wound edges and encourage brisk healing with minimal infection risk. The limitations are those imposed by anatomy, specifically skin tautness or the presence of vital structures. Anesthesia of wound edges may be attained by regional nerve block or local infiltration with lidocaine, which do not damage local tissue defenses. A topical anesthetic mixture of tetracaine, epinephrine, and cocaine may be less desirable because of the vasoconstrictive effect of epinephrine and theoretical infection-potentiating effects. Definitive wound exploration, debridement, and repair should be undertaken in an appropriate sterile environment. It is often impractical to explore complex wounds in the emergency department. When necessary, operating loupes should be used to inspect the wound for residual foreign material, such as sand, seaweed, teeth, spine fragments, or integumentary sheath shards. Standard radiographs, static soft tissue techniques, MRI, or fluoroscopy may be used preoperatively or perioperatively to localize spines or teeth.

Wound Closure

The decision to close a wound must weigh the cosmetic result against the risk of infection. The incidence of infection is high in wounds acquired in natural bodies of water because such wounds may be contaminated with venom, potentially virulent

microorganisms, or both; because early adequate irrigation and debridement are often unavailable; and because definitive care is often delayed. Tight wound closure restricts drainage and promotes bacterial proliferation, particularly from anaerobes, which are common contaminants. Wounds at high risk in this regard include those on the hands, wrists, or feet; punctures and crush injuries; wounds into areas of fat with poor vascularity; and wounds to victims who are immunosuppressed. Whenever possible, the use of sutures to close dead space in contaminated wounds should be minimized because the absorbable sutures act as foreign bodies.

Prophylaxis against Tetanus

Any wound that disrupts the skin can become contaminated with *Clostridium tetani.* Anaerobic bacteria, predominantly of the genus *Clostridium,* have been isolated in shark tissue and as part of the oral flora of alligators and crocodiles. Proper immunization with tetanus toxoid virtually eliminates the risk of disease. However, although it was previously accepted that the protective level of toxin-neutralizing antibody is 0.01 antitoxin unit/mL, it appears that clinical tetanus can develop despite an antibody level many times that amount. Therefore it is imperative to provide an early and adequate booster injection. If the victim is over 50 years of age, is from an underdeveloped country, or cannot provide a definite history of tetanus immunization, it is likely that circulating toxin-neutralizing antibody will be suboptimal. Prophylaxis should be provided according to the scheme shown in Chapter 20 in Table 20-3.

BACTERIOLOGY OF THE AQUATIC ENVIRONMENT

Wounds acquired in the aquatic environment are soaked in natural source water and sometimes contaminated with sediment. Penetration of the skin by the spines or teeth of animals, the razor edges of coral or shellfish, or mechanical objects such as the blades of a boat propeller may inoculate pathogenic organisms into a wound. Sports activities, such as surfing, snorkeling, and diving, lead to ubiquitous abrasions and minor lacerations that heal slowly and with marked soft tissue inflammation. Wounds acquired in the aquatic environment tend to become infected and may be refractory to standard antimicrobial therapy. Not infrequently, indolent or extensive soft tissue infections develop in the normal or immunocompromised host.[13,106] A clinician faced with a serious infection after an aquatic injury frequently needs to administer broad-spectrum antibiotics to a patient before definitive laboratory identification of pathogenic organisms has been obtained.

Marine Bacteriology

Marine Environment

Ocean water provides a saline milieu for microbes. The salt dissolved in ocean water (3.2% to 3.5%) is 78% sodium chloride (sodium 10.752 g/kg; chlorine 19.345 g/kg). Other constituents include sulfate (2.791 g/kg), magnesium (1.295 g/kg), potassium (0.39 g/kg), bicarbonate (0.145 g/kg), bromine (0.066 g/kg), boric acid (0.027 g/kg), strontium (0.013 g/kg), and fluorine (0.0013 g/kg). The temperature of the surface waters varies with latitude, currents, and season. Tropical waters are warmer and maintain a more constant temperature than do temperate and subtropical waters, which are subject to substantial meteorologic variation. Shallow and turbulent coastal waters are generally richer in nutrients than is the open ocean, which is reflected in the diversity of life that can be identified in the intertidal zone. Although the greatest number and diversity of bacteria are found near the ocean surface, diverse bacteria and fungi are found in marine silts, sediments, and sand and within the oral cavities of marine organisms. In ocean waters having marked differences in density, the greatest concentration of bacteria is noted at the thermocline, where changes in both temperature and salinity are usually found.[114] This effect is lost in active coastal waters, where the constant admixture creates an even distribution of sediments, microbes, salinity, and temperature. Microbes are most abundant in areas that have the greatest numbers of higher life-forms. Marine bacteria are generally halophilic, heterotrophic, motile, and gram-negative rod forms. Growth requirements vary from species to species with respect to use of organic carbon and nitrogen sources, requirements for various amino acids, vitamins and cofactors, sodium, potassium, magnesium, phosphate, sulphate, chloride, and calcium. Most marine bacteria are facultative anaerobes, which can thrive in oxygen-rich environments. Few are obligatory aerobes or anaerobes. Some marine bacteria are highly proteolytic, and the proportion of proteolytic bacteria seems to be greater in the oceans than on land or in freshwater habitats.[114] It has been observed that sharks may harbor multidrug-resistant bacteria, which indicates that these animals may have encountered synthetic drugs that have found their way into rivers and other waterways.

Diversity of Organisms

Unique conditions of nutrient and inorganic mineral supply, temperature, and pressure have allowed the evolution of unique, highly adapted marine microbes.[160,161] In addition, numerous other bacteria, microalgae, protozoa, fungi, yeasts, and viruses have been identified in or cultured from seawater, marine sediments, marine life, and marine-acquired or marine-contaminated infected wounds or body fluids of septic victims. In their natural environment the bacteria presumably serve to scavenge and transform organic matter in the intricate cycles of the food and growth chains. Some of these bacteria are listed in Box 72-1.[112] Enteric pathogenic bacteria have been isolated from sharks.[54] A shark attack victim in South Africa who sustained serious injuries to his lower extremities was reported to have developed a fulminant infection attributed to *Bacillus cereus* shown to be sensitive to fluoroquinolones, amikacin, clindamycin, vancomycin, and tetracyclines and resistant to penicillin and cephalosporins (including third generation). In another report, one shark (presumed bronze whaler) attack victim in Australia grew both *Vibrio parahaemolyticus* and *Aeromonas caviae* from his wounds, whereas another grew *Vibrio alginolyticus* and *Aeromonas hydrophila* from his wound.[115] It is now fairly well known that *Vibrio* species and *Aeromonas* species are potential pathogens residing in ocean water and fresh water.

For practical purposes, most marine isolates are heterotrophic (require exogenous carbon and nitrogen-containing organic supplements) and motile gram-negative rods. *Halomonas venusta,* a halophilic, nonfermentative, gram-negative rod, was reported as a human pathogen in a wound that originated from a fish bite.[146] Previous opinions that enteric pathogens (associated with the intestines of warm-blooded animals) deposited into marine environments ultimately succumb to sedimentation,

Box 72-1. Bacteria and Fungus Isolated from Marine Water, Sediments, Marine Animals, and Marine-Acquired Wounds

Achromobacter
Acinetobacter lwoffii
Actinomyces
Aerobacter aerogenes
Aeromonas hydrophila
Aeromonas salmonicida
Aeromonas sobia
Alcaligenes faecalis
Alteromonas espejiana
Alteromonas haloplanktis
Alteromonas macleodii
Alteromonas undina
Bacillus cereus
Bacillus subtilis
Bacteroides fragilis
Branhamella catarrhalis
Chromobacterium violaceum
Citrobacter
Clostridium botulinum
Clostridium perfringens
Clostridium tetani
Corynebacterium
Edwardsiella tarda
Enterobacter aerogenes
Erysipelothrix rhusiopathiae
Escherichia coli
Flavobacterium
Fusarium solani
Klebsiella pneumoniae
Legionella pneumophila
Micrococcus luteus
Micrococcus sedentarius
Moraxella lacunata
Mycobacterium marinum
Neisseria catarrhalis
Pasteurella multocida
Photobacterium damsela
Propionibacterium acnes
Proteus mirabilis
Proteus vulgaris
Providencia stuartii
Pseudomonas aeruginosa
Pseudomonas cepacia
Pseudomonas maltophila
Pseudomonas putrefaciens
Pseudomonas stutzeri
Salmonella enteritidis
Serratia
Staphylococcus aureus
Staphylococcus epidermidis
Streptococcus
Vibrio alginolyticus
Vibrio carchariae
Vibrio cholerae
Vibrio fluvialis
Vibrio furnissii
Vibrio harveyi
Vibrio hollisae
Vibrio parahaemolyticus
Vibrio mimicus
Vibrio splendidus I
Vibrio vulnificus

predation, parasitism, sunlight, temperature, osmotic stress, toxic chemicals, or high salt concentration may be untrue.[53] Pathogens may accumulate in surface water in association with lipoidal particulates, from which they are rapidly dispersed toward shore by wave and wind activity. In addition, dredging, storms, upwellings, and other benthic disturbances may churn enteric organisms into the path of wastewater nutrients.

Wound Infections Caused by *Vibrio* Species

Vibrio organisms can cause gastroenteric disease (gastroenteric *Vibrio* infections are discussed in Chapter 66) and soft tissue infections, particularly in immunocompromised hosts. Extraintestinal infections may be associated with bacteremia and death. *Vibrio* species are the most potentially virulent halophilic organisms that flourish in the marine environment. The teeth of a great white shark were swabbed and yielded *V. alginolyticus, V. fluvialis,* and *V. parahaemolyticus*.[25] Mako shark tooth culture has yielded *V. damsela, V. furnissii,* and *V. splendidus I*.[6] *V. parahaemolyticus* has also been identified in freshwater habitats.[10] Water that is brackish (salinity of 15 to 25 parts per thousand [ppt]) allows the growth of *Vibrio* species if appropriate nutrients are present; *V. vulnificus* infection has been documented after exposure to waters with salinities of 2 and 4 ppt. The optimal season for exposure appears to be summer, when water temperatures encourage bacterial proliferation. In most studies reported, infections seem to cluster during the summer months; this may be related to increased numbers of people at the seashore. This has been corroborated to some degree by the observation that *V. parahaemolyticus* cultured from marine mammals was recovered only in the warmer months of the year in the northeastern United States or in animals from subtropical regions. Sharks appear to develop some immunity to autochthonous *Vibrio* species, as suggested by the detection of a binding protein similar to the immunoglobulin M (IgM) subclass of immunoglobulin. Allochthonous (for the shark) *Vibrio* species, such as *V. carchariae,* may be the agents of elasmobranch disease when the animal is under stress.

Vibrio species are halophilic, gram-negative rods that are facultative anaerobes capable of using D-glucose as their sole or principal source of carbon and energy.[36] They are part of the normal flora of coastal waters not only in the United States, but also in many exotic locations frequented by recreational and industrial divers and seafarers. Vibrios are mesophilic organisms and grow best at temperatures of 24° to 40°C (75.2° to 104°F), with essentially no growth below 8° to 10°C (46.4° to 50°F). Certain other "marine bacteria" are facultative psychrophiles, barophiles, or both. *Vibrio* species seem to require less sodium for maximal growth than do other more fastidious marine organisms, a factor that allows explosive reproduction in the 0.9% saline environment of the human body. At least 11 of the 34 recognized *Vibrio* species have been associated with human disease.[36] Wound infections have been documented to yield *V. cholerae* 0 group 1 and non-01, *V. parahaemolyticus, V. vulnificus, V. alginolyticus,* and *V. damsela*. Septicemia, with or without an obvious source, has been attributed to infections with *V. cholerae* non-01, *V. parahaemolyticus, V. alginolyticus, V. vulnificus,* and *V. metschnikovii*. *Vibrio* may infect fish, causing significant mortality in fish culture facilities. The affliction manifests with lethargy, loss of appetite, skin sores, exophthalmia, and gastrointestinal hemorrhage.

Vibrio parahaemolyticus. *Vibrio parahaemolyticus* is a halophilic gram-negative rod. The organisms are found in waters along the entire coastline of the United States. Generally the incidence of clinical disease is greatest in the warm summer months when the organism is commonly found in zooplankton. *V. parahaemolyticus* absorbs onto chitin and to minute crus-

tacean copepods that feed on sediment. It has been postulated that unusual warm coastal currents (such as El Niño) may contribute to increased proliferation of *Vibrio* species. The optimal growth temperature of *V. parahaemolyticus* is 35° to 37° C (95° to 98.6° F); under ideal conditions the generation time has been estimated at less than 10 minutes, with explosive population growth from 10 to 10^6 organisms in 3 to 4 hours.

Extraintestinal wound infections are most common in persons who suffer chronic liver disease or immunosuppression. Although over 95% of *V. parahaemolyticus* strains associated with human illness are positive, the relationship to pathogenicity of the Kanagawa reaction (production of a cell-free hemolysin on high salt-mannitol [Wagatsuma] agar), caused by a heat-stable direct hemolysin, is not yet clear. Furthermore, most marine strains are not Kanagawa positive. Some primary soft tissue infections previously attributed to *V. parahaemolyticus* may theoretically be attributed to misidentified *V. vulnificus*. Panophthalmitis requiring enucleation occurred in a man who suffered a corneal laceration.[133]

Vibrio vulnificus. *Vibrio vulnificus* (formerly known as a "lactose [fermenting]-positive" vibrio) is a halophilic gram-negative bacillus. *V. vulnificus* (Latin for "wounding") is found in virtually all U.S. coastal waters and has been reported to cause infection worldwide.[11] It prefers salinity of 0.7% to 1.6%; although it prefers a habitat of warm (at least 20° C [68° F]) seawater, it can be found in much colder water. It does not appear to be associated with fecal contamination of seawater. It has been shown to exist in Chesapeake Bay with bacterial counts comparable to those reported from the Gulf of Mexico.[158]

The organism may or may not have an acidic polysaccharide capsule (opaque colony), which confers protection against bactericidal activity of human serum and phagocytosis and thus renders the organism more virulent in animals. At extremely low frequency, some strains can shift between unencapsulated (avirulent; translucent colony) and capsulated (virulent) serotypes. The encapsulated isolates show exquisite (positive) sensitivity to iron. Virulent isolates can use 100% but not 30% saturated (normal for humans) transferrin as an iron source, as well as iron in hemoglobin and hemoglobin-haptoglobin complexes. *V. vulnificus* exhibits enhanced growth and virulence in the presence of increased serum iron concentration or saturated transferrin-binding sites.[2] *V. vulnificus* is classified in three biotypes: biotype 1 is pathogenic for humans and biotype 2 is pathogenic for fish.[36,37] Biotype 3 causes soft-tissue infections and septicemia following contact with fish from freshwater ponds; it was first noted in Israel. Within biotype 1, various genetically distinct subgroups identified by randomly amplified polymorphic deoxyribonucleic acid (DNA) polymerase chain reaction (PCR) appear to be especially virulent.[8] Furthermore, complete genomic sequencing of *V. vulnificus* YJ016, a biotype 1 strain, reveals gene clusters related to pathogenicity (cell adhesion, colonization, cytotoxicity, and tissue destruction), including capsular polysaccharide, siderophore biosynthesis and transport, and heme receptor and transport.[37] In vivo antigen technology can identify virulence genes produced and expressed in humans.[69] The ability of biotype 1 to multiply and produce a toxic metalloprotease in human serum may be a prominent virulence factor.[151]

Infection worsens rapidly after the initiation of symptoms and has been noted most frequently in men over 40 years of age with preexisting hepatic dysfunction (particularly cirrhosis), end stage renal impairment, leukopenia, or impaired immunity (malignancy, human immunodeficiency virus [HIV] infection, diabetes, bone marrow suppression, long-term corticosteroids), although it has been reported in young, previously healthy individuals.[72] One case followed application of fish blood by a healer as a traditional remedy to a chronic leg ulcer in an obese patient suffering from recurrent erysipelas.[134] Preexisting liver disease is a predictor of death, with 50% of such individuals succumbing to the illness in one series.[59] Persons with high serum iron levels (from chronic cirrhosis, hepatitis, thalassemia major, hemochromatosis, multiple transfusions [such as are given for aplastic anemia]) or achlorhydria (low gastric acid; may be iatrogenically induced with H_2 blockers) may be at greater risk for fulminant bacteremia.[2,129,139] This has been attributed in part to the protective effect of gastric acid, the iron requirement of the organism, and the effects of liver disease (decreased polymorphonuclear leukocyte and macrophage activity, flawed opsonization, shunting of portal blood around the liver). It has been proposed that an effective host response against *V. vulnificus* and similar iron-sensitive pathogens (e.g., *Listeria monocytogenes, Klebsiella* spp., and *Yersinia* spp.) is in part augmented by hepcidin, a cysteine-rich cationic antimicrobial peptide central to iron metabolism.[8,150] *V. vulnificus* produces a siderophore *(vulnibactin)* and a protease that may enhance pathogenicity.[2]

The syndrome consists of flu-like malaise, fever, vomiting, diarrhea, chills, hypotension, and early skin vesiculation that evolve into necrotizing dermatitis and fasciitis, with vasculitis and myositis (Figs. 72-1 and 72-2).[159] Hematogenous seeding of vibrios to secondary cutaneous lesions is probable. Primary wound infections (approximately 30% of cases) rapidly show marked edema, with erythema, vesicles, and hemorrhagic or contused-appearing bullae, progressing to necrosis. This may require radical surgical debridement or amputation. Up to 25% of these victims may have sepsis. When *V. vulnificus* is recovered from the blood of a victim with sepsis attributable to a wound infection, the case fatality rate may exceed 30%.[59] Extracellular elastin-lysing proteases elaborated by the organism, as well as a potent collagenase, probably contribute to the rapid invasion of healthy tissue. *V. vulnificus* also produces a cytotoxin-hemolysin and phospholipases. Cytolysin produced by most pathogenic strains of *V. vulnificus* is extremely toxic to mice when injected intravenously and results in severe perivascular edema and neutrophil infiltration in lung tissues.[64] The precise roles of these and other factors (pili, mucinase, chondroitinase, hyaluronidase) in the in vivo pathogenicity of the

Figure 72-1. Echthyma gangrenosum associated with *Vibrio vulnificus* sepsis. (Courtesy Edward J. Bottone, MD, Dept. of Microbiology, Mt. Sinai Hospital, NY.)

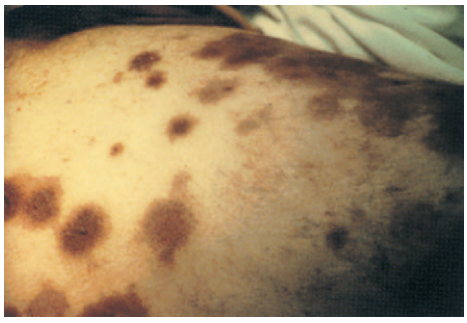


Figure 72-2. Torso of a victim with *Vibrio vulnificus* sepsis.

organism have yet to be determined. Bleeding complications (which may include gastrointestinal hemorrhage and disseminated intravascular coagulation) are common and may be attributed in part to thrombocytopenia. Gastroenteritis is more common (15% to 20%) with the septicemic presentation than with primary wound infection and may exist as an isolated entity (approximately 10% of cases), although it is debated that illness has been erroneously attributed to the asymptomatically carried organism. *Vibrio vulnificus* endometritis has been reported after an episode of intercourse in the waters of Galveston Bay, Texas.[168] Other presentations of *V. vulnificus* infections have included meningitis, necrotizing fasciitis following lightning strike in a windsurfer, spontaneous bacterial peritonitis, corneal ulcers, epiglottitis, and infections of the testes, spleen, and heart valves.[140]

The explosive nature of the syndrome can lead to gram-negative sepsis and death, reportedly in up to 50% of cases. The mortality rate may be as high as 90% in victims who become hypotensive within 12 hours of initial examination by a physician. For wound infections from all *Vibrio* species, the organism may only be recovered from blood specimens in less than 20% of victims.[59] Appropriate antibiotics should be administered as soon as the infection is suspected (see later). In one report, *V. vulnificus* sepsis was treated with antibiotic therapy, debridement of necrotic tissues, and direct hemoperfusion using polymyxin B immobilized fiber, which served as an artificial reticuloendothelial system and removed endotoxin from the circulating blood.[116] In an immunocompetent victim who acquired a *V. vulnificus* hand infection from peeling shrimp, treatment with oral ciprofloxacin was successful. In a series of seven patients treated for primary skin and soft tissue infections secondary to *V. vulnificus*, prompt operative exploration and debridement were correlated with a decrease in the intensive care unit and hospital length of stay, particularly if the surgery occurred within 72 hours from the time of infection.[57] The authors noted that all patients had necrosis of underlying subcutaneous tissue, whereas some did not demonstrate skin necrosis.

Vibrio mimicus. *Vibrio mimicus* is a motile, nonhalophilic, gram-negative, oxidase-positive rod with a single flagellum. It can be distinguished from *V. cholerae* by its inability to ferment sucrose, inability to metabolize acetylmethyl carbonyl, sensitivity to polymyxin, and negative lipase test. An ear infection may follow exposure to ocean water. Isolates are sensitive to tetracycline. Physicians who collect stool samples for culture to identify suspected *V. mimicus* must alert the laboratory to use appropriate culture media (thiosulfate–citrate–bile salts–sucrose [TCBS] agar).

Vibrio alginolyticus. *Vibrio alginolyticus*, found in seawater, has been implicated in soft tissue infections (e.g., those caused by coral cuts or surfing scrapes), sinusitis, and otitis, particularly after previous ear infections or a tympanic membrane perforation. Although bacteremia has been reported in immunosuppressed patients and patients with burns, *V. alginolyticus* does not generally carry the virulent potential of *V. vulnificus*.[87] Typical symptoms include cellulitis, with seropurulent exudate. Its distinguishing microbiologic features are stated to be sucrose and lactose fermentation, growth in 1% tryptone broth plus 10% NaCL, positive Voges-Proskauer reaction, negative urease reaction, and susceptibility to vibriostatic compound O/129. However, because these indices may vary, identification may be difficult.

Photobacterium damsela. *Photobacterium* (formerly *Vibrio* or *Listonella*) *damsela*, formerly enteric group EF-5 and so-named because it is pathogenic for the damselfish, causes wound infections similar to those attributed to vibrios. Rapidly progressive infection leading to muscle necrosis and fasciitis or to sepsis and death may transpire in an immunosuppressed victim or person with normal immunity.[50,52,169] This may be related to an extracellular cytolysin (damselysin) or other unidentified enzymes or virulence factors.

Vibrio cholerae. A case of necrotizing fasciitis and septic shock caused by *V. cholerae* non-O1 (not agglutinated in cholera polyvalent O1 antiserum) acquired in San Diego, California, has been described.[148] The victim suffered from preexisting diabetes mellitus complicated by chronic plantar ulceration of the affected limb.

Growth in Culture. Although plating on standard clinical laboratory media may detect only 0.1% to 1% of the total number of microorganisms found in seawater or marine sediment, most marine bacteria that are pathogenic to humans can be readily recovered on standard media. Although pathogenic *Vibrio* species can grow on conventional blood agar media, other marine bacteria may require saline-supplemented media and incubation at 25°C (77°F) instead of the standard 35° to 37°C (95° to 98.6°F). In culture, marine bacteria may grow at a slower rate than terrestrial bacteria, which delays identification. Pleomorphism in culture may be attributed to adaptation to small concentrations of nutrients in seawater. Most organisms require sodium, potassium, magnesium, phosphate, and sulfate for growth; a few require calcium or chloride.

All *Vibrio* species grow in routine blood culture mediums and on nonselective mediums, such as blood agar. TCBS agar is selective and recommended for the detection of marine *Vibrio* organisms, although cellobiose–polymyxin B–colistin (CPC) agar may be as good or better.[17,82] An alternative is Monsur taurocholate-tellurite-gelatin agar. A large clinical laboratory near the ocean might consider the use of TCBS or CPC agar routinely. Pathogenic vibrios generally grow on MacConkey agar. All species except *V. cholerae* and *V. mimicus* require sodium chloride for growth. Enrichment broth (alkaline peptone water with 1% NaCl) is recommended for isolation of vibrios from convalescent and treated patients. Another enrich-

ment broth that may be more effective is 5% peptone, 1% NaCl, and 0.08% cellobiose (PNC) at pH 8.0.[61] A comparison of strategies for the detection and recovery of *V. vulnificus* from marine samples of the western Mediterranean coast determined that the best strategy consisted of the combination of culture-based methods (3-hour enrichment in alkaline-saline peptone water at 40° C [104° F], followed by culture on CPC agar) and DNA-based procedures (specific PCR amplification of the presumptive colonies with primers Dvu 9V and Dvu 45R).[6]

Key characteristics that aid in the separation of *Vibrio* species from other medically significant bacteria (Enterobacteriaceae, *Pseudomonas, Aeromonas, Plesiomonas*) are the production of oxidase, fermentative metabolism, requirement of sodium chloride for growth, and susceptibility to the O/129 vibriostatic compound. *Vibrio vulnificus* can be cultured from the blood, wounds (bullae), and stool. The laboratory must be cautioned to use selective culture media with a high salt content (3% NaCl) for prompt identification. Suggestive features include positive fermentation of glucose, positive oxidase test, positive indole test, positive reaction for both lysine and ornithine decarboxylase, positive *o*-nitrophenyl-β-D-galactopyranoside, and inability to ferment sucrose. A useful identification scheme for pathogenic *Vibrio* species is found in the chapter on *Vibrio* in the most recent edition of the American Society for Microbiology's *Manual of Clinical Microbiology.*

Because growth of *V. vulnificus* in culture generally requires 48 hours, current research is directed at a more rapid diagnostic test. Direct identification of *V. vulnificus* in clinical specimens by nested PCR has been accomplished utilizing serum specimens and bulla aspirates from septicemic patients.[79] A nested PCR method for rapid and sensitive detection of *V. vulnificus* in fish, sediments, and water has been developed.[7] Simultaneous detection of five marine fish pathogens (*V. vulnificus, Listonella anguillarum, Photobacterium damselae, Aeromonas salmonicida,* and *V. parahaemolyticus*) has been accomplished by using multiplex PCR and a DNA microarray.[51] This technique has not yet been applied to diagnosis of pathogens afflicting humans.

Mycobacteria must be cultured in media such as Middlebrook 7H10 or 7H11 agar or Lowenstein-Jensen medium; fungi require a medium such as Sabouraud dextrose or brain-heart infusion/Sabhi agar. Antibiotic susceptibility testing can be performed using established procedures, except for the addition of NaCl 2.3% to the Mueller-Hinton broth or agar used for disk diffusion. Certain commercial test kits may not accurately identify marine organisms. In the setting of wound infection or sepsis, the clinician should alert the laboratory that a marine-acquired organism may be present. If a laboratory does not have the time or resources to perform a complete identification, the bacteria may be sent to a reference laboratory. Marine bacteria are kept in the American Type Culture Collection. Because of the diversity of species, complete agreement has not yet been reached on comprehensive taxonomic criteria for identification.

Antibiotic Therapy. The objectives for the management of infections from marine microorganisms are to recognize the clinical condition, culture the organism, and provide antimicrobial therapy. Management of marine-acquired infections should include therapy against *Vibrio* species. Third-generation cephalosporins (cefoperazone, cefotaxime, or ceftazidime) provide variable coverage in vitro; first- and second-generation products (cefazolin, cephalothin, cephapirin, cefamandole, cefonicid, ceforanide, or cefoxitin) appear to be less effective in vitro. The organism has been reported in some cases to be resistant in vitro to third-generation cephalosporins, mezlocillin, aztreonam, and piperacillin.[104] A combination of cefotaxime and minocycline seems to be synergistic and extremely effective against *V. vulnificus* in vitro.[39] Oral cultures taken from two captive moray eels at the John G. Shedd Aquarium in Chicago, Illinois, demonstrated *V. fluvialis, Photobacterium damsela, V. vulnificus,* and *Pseudomonas putrefaciens* sensitive to cefuroxime, ciprofloxacin, tetracycline, and trimethoprim-sulfamethoxazole.[48] Imipenem-cilastatin is efficacious against gram-negative marine bacteria, as are trimethoprim-sulfamethoxazole and tetracycline. Gentamicin, tobramycin, and chloramphenicol have tested favorably against *P. putrefaciens* and *Vibrio* strains. Nonfermentative bacteria (such as *Alteromonas, Pseudomonas,* and *Deleya* species) appear to be sensitive to most antibiotics. In a mouse model, combination therapy with minocycline and cefotaxime was more effective than either drug alone.[40]

Quantitative wound culture has no advantage before the appearance of a wound infection. Pending a prospective evaluation of prophylactic antibiotics in the management of marine wounds, the following recommendations are based on the indolent nature and malignant potential of soft tissue infections caused by *Vibrio* species:

1. Minor abrasions or lacerations (such as coral cuts or superficial sea urchin puncture wounds) do not require prophylactic antibiotics in the host with normal immunity. Persons who are chronically ill (as with diabetes, hemophilia, or thalassemia) or immunologically impaired (as with leukemia or acquired immunodeficiency syndrome [AIDS], or undergoing chemotherapy or prolonged corticosteroid therapy), or who suffer from serious liver disease (such as hepatitis, cirrhosis, or hemochromatosis), particularly those with elevated serum iron levels, should be placed immediately after the injury on a regimen of oral ciprofloxacin, trimethoprim-sulfamethoxazole, or tetracycline (or doxycycline) because these persons appear to have an increased risk of serious wound infection and bacteremia. Preliminary experience suggests that cefuroxime may be a useful alternative. Penicillin, ampicillin, and erythromycin are not acceptable alternatives.[92,94] Norfloxacin may be less efficacious against certain vibrios.[93,108] Other quinolones (ofloxacin, enoxacin, pefloxacin, fleroxacin, lomefloxacin) have not been extensively tested against *Vibrio;* they may be useful alternatives, but this awaits definitive evaluation. The appearance of an infection indicates the need for prompt debridement and antibiotic therapy. If an infection develops, antibiotic coverage should be chosen that will also be efficacious against *Staphylococcus* and *Streptococcus* because these are still quite common perpetrators of infection. In general, the fluoroquinolones, which are particularly effective for treating gram-negative bacillary infections, may become less and less useful against resistant staphylococci.[138] If *Staphylococcus* is a β-lactamase–producing strain, a semisynthetic penicillin (nafcillin or oxacillin) should be chosen, with a cephalosporin such as cefazolin or cephalothin used if there is a history of delayed-type penicillin allergy. Vancomycin is recommended in the event of methicillin resistance.[83]

2. Serious injuries from an infection perspective include large lacerations, serious burns, deep puncture wounds, or a retained foreign body. Examples are shark or barracuda bites, stingray spine wounds, deep sea urchin punctures, scorpaenid (scorpionfish) spine envenomations that enter a joint space, and full-

thickness coral cuts. If the victim requires hospitalization and surgery for standard wound management, recommended antibiotics include gentamicin, tobramycin, amikacin, ciprofloxacin, and trimethoprim-sulfamethoxazole. Cefoperazone and cefotaxime may or may not be effective. There is a recommendation in the literature advocating the use of ceftazidime (in combination with tetracycline).[73] Chloramphenicol is an alternative agent less commonly used because of hematologic side effects. Imipenem-cilastatin is an extremely powerful antibiotic that should be used in a circumstance of sepsis or treatment failure. Patients who simultaneously receive imipenem or ciprofloxacin and theophylline may have an increased tendency to seizures.[119] Meropenem has shown excellent in vitro activity against the Vibrionaceae and may be useful for eradicating infections produced by these organisms.[41a]

If the victim is managed as an outpatient, the drugs of choice to cover *Vibrio* are ciprofloxacin, trimethoprim-sulfamethoxazole, or tetracycline. Cefuroxime is an alternative. It is a clinical decision whether oral therapy should be preceded by a single intravenous or intramuscular loading dose of a similar or different antibiotic, commonly an aminoglycoside.

3. Infected wounds should be cultured for aerobes and anaerobes. Pending culture and sensitivity results, the patient should be managed with antibiotics as described previously. In a person who has been wounded in a marine environment and has rapidly progressive cellulitis or myositis, *Vibrio parahaemolyticus* or *V. vulnificus* infection should be suspected, particularly in the presence of chronic liver disease. If a wound infection is minor and has the appearance of a classic erysipeloid reaction *(Erysipelothrix rhusiopathiae),* penicillin, cephalexin, or ciprofloxacin should be administered (see Chapter 75). *E. rhusiopathiae* attributed to a pet goldfish has been reported to cause necrotizing fasciitis in a diabetic patient.[121] It is also cultured in episodes of infective endocarditis, although the origin of the infections are often not identified.[80,141]

If sepsis is severe, additional aggressive measures beyond surgery and antibiotics, such as administration of recombinant human activated protein C, may be required.[4] Hyperbaric oxygen therapy has been used as adjunctive therapy in *V. vulnificus* septicemia and cellulitis, but there is not a standard recommendation for this modality for this indication.[199]

Freshwater Bacteriology

Diversity of Organisms

Although it has not been as extensively studied as the marine environment, the natural freshwater environment of ponds, lakes, streams, rivers, lagoons, harbors, estuaries, and artificial bodies of water is probably as hazardous as the ocean from a microbiologic standpoint. Waterskiing accidents, propeller wounds, fishhook punctures, lacerations from broken glass and sharp rocks, fish fin or catfish stings, and crush injuries during white-water expeditions are commonplace. A large number of bacteria have been identified in water, sediments, animals, and wounds. In fringe areas of the ocean that carry brackish water (NaCl content <3%), marine bacteria, salt-tolerant freshwater bacteria, and brackish-specific bacteria, such as *Agrobacterium sanguineum,* are noted. The combined effects of human and animal traffic and waste disposal increase the risk for coliform contamination. In Great Britain, antibiotic-resistant *Escherichia coli* have been documented in rivers and coastal waters.[124] Coxsackievirus A16 has been isolated from children stricken ill after bathing in contaminated lake water.[43] Of particular note is the presence of virulent species, such as *Chromobacterium violaceum, Vibrio parahaemolyticus,* and *Aeromonas hydrophila,* associated with serious and indolent wound infections.[90,147] The last can be cultured from natural bodies of water, as well as from the mouths of domesticated aquarium fish, such as the piranha.[113] Biologic control agents, such as guppy fish bred in wells to control mosquito proliferation, can carry bacterial pathogens such as *Pseudomonas.*[35]

One investigation sampled water, inanimate objects, and animals from freshwater environments in California, Tennessee, and Florida.[10] Bacteria isolated were predominantly gram-negative and included *Aeromonas hydrophila, Flavobacterium breve, Pseudomonas* species, *Vibrio parahaemolyticus, Serratia* species, *Enterobacter* species, *Plesiomonas shigelloides, Bacillus* species, *Acinetobacter calcoaceticus,* and *Alcaligenes denitrificans.*

Wound Infections Caused by *Aeromonas* Species. *Aeromonas hydrophila* (Latin for "gas producing" and "water loving") is a gram-negative, facultatively anaerobic, polarly flagellated, non–spore-forming, and motile rod member of the family Vibrionaceae that commonly inhabits soil, freshwater streams, and lakes.[3,12,76,143] *Aeromonas* species are widely distributed and found at wide ranges of temperature and pH. Five species (*A. hydrophila, A. sobria, A. schubertii, A. veronii,* and *A. caviae*) of nine species that have been recovered from clinical material have been linked with human disease; there are thirteen or more distinct genotypes.[1] *A. hydrophila* is pathogenic to amphibians, reptiles, and fish. Soft tissue and gastroenteric human infections occur predominantly during the period from May to November. Virulence factors elaborated by *Aeromonas* species include hemolysin, cytotoxin, enterotoxin, cholera toxin–like factor, and hemagglutinins.[23,142] *Aeromonas* species are sometimes misidentified as members of the genus *Vibrio* by commonly used screening tests.[1]

A wound, particularly of the puncture variety, immersed in contaminated water may become cellulitic within 24 hours, with erythema, edema, and a purulent discharge.[155] The lower extremity is most frequently involved. This usually occurs from stepping on a foreign object or being punctured underwater. The appearance may be indistinguishable from a typical streptococcal cellulitis, with localized pain, lymphangitis, fever, and chills. Untreated or managed with antibiotics to which the organism is not susceptible, this may rarely progress to a severe gas-forming soft tissue reaction, bulla formation, necrotizing myositis, or osteomyelitis. Appearance similar to ecthyma gangrenosum caused by *Pseudomonas aeruginosa* has been reported in *Aeromonas* septicemia.

Fever, hypotension, jaundice, and chills are common manifestations of septicemia.[70] Additional clinical manifestations include abdominal pain or tenderness, altered consciousness, acute renal failure, bacteremic pneumonia, and coagulopathy.[65] In a manner analogous to the pathogenicity of virulent *Vibrio* species, the chronically ill or immunocompromised host (e.g., chronic liver disease, neoplasm, diabetes, uremia, corticosteroid therapy, extensive burns) is probably at greater risk of a severe infection or complication, such as meningitis, endocarditis, or septicemia. Freshwater aspiration may result in *Aeromonas hydrophila* pneumonitis and bacteremia. Infection has followed the bite of an alligator. A 15-year-old boy suffered *A. hydrophila* wound infection after a bite from his pet piranha. For unknown

reasons there is a marked preponderance of male victims. This may represent the phenotypic variation of critical bacterial adhesins. Corneal ulcer caused by *A. sobria* was reported after abrasion by a freshwater reed.[33]

Because of the microbiologic similarity of *Aeromonas* on biochemical testing to members of the Enterobacteriaceae family, such as *Escherichia coli* or *Serratia* species, it is important to alert the laboratory to the clinical setting. In the microbiology laboratory, *Aeromonas* species may be identified on the basis of positive oxidase reaction, no growth on TCBS agar, growth on MacConkey agar, and resistance to the vibriostatic compound O/129.[70]

Gram's stain of the purulent discharge may demonstrate gram-negative bacilli, singly, paired, or in short chains. Given the appropriate clinical setting (after a wound acquired in the freshwater environment), this should not be casually attributed to contamination.[64] *Aeromonas hydrophila* is generally sensitive to chloramphenicol, aztreonam, gentamicin, amikacin, tobramycin, trimethoprim-sulfamethoxazole, cefotaxime, cefuroxime, moxalactam, imipenem, ceftazidime, ciprofloxacin, and norfloxacin. In one case, culture of a severe wound infection demonstrated the presence of two species, *Aeromonas hydrophila* and *A. sobria*. Notably, the latter was resistant to tetracycline in vitro. For a severe infection, initial therapy that includes an aminoglycoside provides coverage against concomitant *Pseudomonas* or *Serratia* infection. As has been demonstrated with *Vibrio* species, the first-generation cephalosporins, penicillin, ampicillin, and ampicillin-sulbactam are not efficacious, perhaps because of the production of a β-lactamase by the organism. *Aeromonas* species are capable of producing chromosomally encoded β-lactamases induced by β-lactam antibiotics. This leads to resistance to penicillins, cephalosporins, and monobactams. The β-lactamase inhibitors, such as clavulanate, are not effective against these β-lactamases, so that amoxicillin-clavulanate may not kill *Aeromonas*.[101] The optimal therapy for invasive infections caused by cefotaxime-resistant *A. hydrophila* is not known, but a recent study of in vitro and in vivo (mice) activities of fluoroquinolones suggest that ciprofloxacin may be as effective as cefotaxime-minocycline.[71] In this study, ciprofloxacin and levofloxacin showed greater activity than did gatifloxacin, moxifloxacin, and lomefloxacin.

Initial therapy of a severe soft tissue infection related to *Aeromonas* should include aggressive wound debridement to mitigate the potentially invasive nature of the organism. In one case of severe cellulitis unresponsive to debridement, fasciotomy, and antibiotic therapy, treatment with hyperbaric oxygen was felt to contribute to successful infection control.[86]

Curiously, medicinal leeches can harbor *Aeromonas* in their gut flora; soft tissue infections related to this phenomenon have been reported.[126] The genus *Plesiomonas* also belongs to the family Vibrionaceae; it has been definitively linked with aquarium-associated infection complicated by watery diarrhea and fever.

Infections Caused by a Fish Pathogen, *Streptococcus iniae*. *Streptococcus iniae* is a pathogen of fish, noted to cause subcutaneous abscesses in Amazon freshwater dolphins (Mammalia *Inia geoffrensis*) kept in captivity.[152] Epizootic fatal meningoencephalitis in fish species caused by streptococci has been observed in outbreaks affecting tilapia, yellowtail, rainbow trout, and coho salmon. *S. iniae* has emerged as a serious pathogen of farmed barramundi in Australia.[167] Persons who handle these fish are at risk for bacteremic illness, which can manifest as cellulitis or sepsis. Endocarditis, meningitis, and arthritis have been noted to accompany *S. iniae* infection.

The tilapia (*Oreochromis* and *Tilapia* species) are also known as St. Peter's fish or Hawaiian sunfish. The surface of these commonly aquacultured fishes may be colonized with *S. iniae*. Persons of Asian descent have been identified as prone to infection, probably because they often prepare this fish with the intent to dine. Typically, the victim recalls puncturing the skin of the hand with a fin, bone, or implement of preparation. Cellulitis with lymphangitis and fever is common, without skin necrosis or bulla formation.[152]

In culture, *S. iniae* shows β-hemolysis. However, it may appear to be α-hemolytic because the narrow zone of β-hemolysis is ringed by more prominent zone of α-hemolysis. Therefore it may be misidentified as a *viridans* streptococcus and therefore considered a contaminant. A reasonable approach to antibiotic therapy includes penicillin, cefazolin, ceftriaxone, erythromycin, clindamycin, or trimethoprim-sulfamethoxazole. In one series, ciprofloxacin showed slightly less efficacy in vitro.

A General Approach to Antibiotic Therapy. Management of freshwater-acquired infections should include therapy against *Aeromonas* species. First-generation cephalosporins provide inadequate coverage against growth of freshwater bacteria. Third-generation cephalosporins provide excellent coverage, whereas second-generation products are less effective. Ceftriaxone may not be efficacious against *Aeromonas* species. Ciprofloxacin, imipenem, ceftazidime, gentamicin, and trimethoprim-sulfamethoxazole are reasonable antibiotics against gram-negative microorganisms. Trimethoprim or ampicillin alone may be inefficacious.

Whether to begin antimicrobial therapy before establishment of a wound infection is controversial. Pending a prospective evaluation of prophylactic antibiotics in freshwater-acquired wounds, the following recommendations are based on the potentially serious nature of soft tissue infections caused by *Aeromonas* species:

1. Minor abrasions or lacerations do not require the administration of prophylactic antibiotics in the normal host. Persons who have chronic illness, immunologic impairment, or serious liver disease, particularly those with elevated serum iron levels, should be placed immediately on a regimen of oral ciprofloxacin or norfloxacin (first choice), trimethoprim-sulfamethoxazole (second choice), or doxycycline-tetracycline (third choice—use only in the setting of allergy to the first two choices, since resistance has been observed) because these persons appear to have an increased risk of serious wound infection and bacteremia. Penicillin, ampicillin, erythromycin, and trimethoprim do not appear to be acceptable alternatives. The appearance of an infection indicates the need for prompt debridement and antibiotic therapy. If an infection develops, antibiotic coverage that will also be efficacious against *Staphylococcus* and *Streptococcus* should be chosen, because these are likely the most common perpetrators of infection.
2. If the victim requires surgery and hospitalization for wound management, recommended antibiotics include ciprofloxacin, gentamicin, or trimethoprim-sulfamethoxazole. Imipenem-cilastatin is an extremely powerful antibiotic that should be used in a circumstance of sepsis or treatment failure. If the victim is to be managed as an outpatient, the oral drug

of choice is norfloxacin (or ciprofloxacin), trimethoprim-sulfamethoxazole, or tetracycline or doxycycline. It is a clinical decision whether oral therapy should be preceded by a single IV or intramuscular loading dose of a similar or different antibiotic, commonly an aminoglycoside.

3. Infected wounds should be cultured. Pending culture and sensitivity results, the patient should be managed with antibiotics as outlined previously. If fever or rapidly progressive cellulitis characterized by bullae and large areas of necrosis develops, *Aeromonas hydrophila* infection should be suspected. Less rapidly progressive *Aeromonas* infections may have the appearance of streptococcal cellulitis.[67]

SHARKS

Myth and folklore surround sharks, the most feared of sea creatures. These occasionally savage animals have been the subjects of many behavioral investigations, but until more reproducible data are available, a degree of mystery will remain. Shark attacks on humans have always held enormous fascination for scientists, adventurers, and clinicians. The problem was highlighted for the U.S. military in 1945 during World War II when crewmembers from the *USS Indianapolis* perished in shark-infested waters. On July 30, the heavy cruiser was sunk by a Japanese torpedo, resulting in hundreds of deaths plus an estimated additional 60 to 80 shark attack fatalities of survivors left adrift for 5 days. This estimate is likely a high figure inflated by historic misinterpretation of scavenge bites on already dead servicemen, but nevertheless, this tragedy represents the largest reported incidence of mass shark attacks.

The International Shark Attack File (ISAF) has its origin from the Shark Research Panel created by the Office of Naval Research (ONR) in 1958. The File was initiated by Perry W. Gilbert and Leonard P. Schultz in 1958 for the Smithsonian Institution, American Institute of Biological Sciences, Cornell University, and the ONR. In 1967, the data were sent to Mote Marine Laboratory in Sarasota, Florida, where H. David Baldridge prepared a special technical report, *Shark Attack Against Man,* for the U.S. Navy Bureau of Medicine and Surgery in 1973, analyzing 1165 reported attacks and case histories. After a period of maintenance at the National Underwater Accident Data Center at the University of Rhode Island, the Shark Attack File moved to the University of Florida at Gainesville, where it is maintained by the International Elasmobranch Society and the Florida Museum of Natural History under the direction of George H. Burgess.[26] It remains an authoritative collection of analyzed data, containing a series of more than 3500 individual investigations from the mid-1500s to the present. Other regional scientific records of shark attacks are maintained by the California Department of Fish and Game, the Natal Sharks Board (South Africa), Taronga Zoo (Australia), the University of Sao Pãulo (Brazil), and the National Marine Fisheries Service Honolulu Laboratory (Hawaii). All of these organizations serve as cooperators with ISAF, feeding results of regional investigations into the ISAF database. Hundreds of cooperating scientific observers located throughout the world act in a similar capacity, ensuring broad coverage.

Although dreaded, sharks are among the most graceful and magnificent denizens of the deep. Sharks may be found in oceans, tropical rivers, and lakes. The bull shark *Carcharhinus leucas* (Fig. 72-3) is a frequent visitor and occasional resident

Figure 72-3. Bull shark. (Photo by Howard Hall.)

of tropical and warm-temperate rivers. It has been witnessed to swim 100 miles (161 km) up freshwater rivers, such as the Ganges, Nile, and Mississippi. These sharks also live in Lake Nicaragua and have had their aggressive behavior attributed in part to high levels of testosterone. During the summer of 2001, the "summer of the shark" in the U.S. media, a bull shark was believed to have attacked 8-year-old Jessie Arbogast off the Gulf Coast of Florida. Sharks range in size from 10 to 15 cm (3.9 to 5.9 in) (spined pygmy shark *Squaliolus laticaudus*) and dwarf dogshark *Etmopterus perryi* (15.4 cm or 6 in) to over 18 m (59 ft) and more than 22,700 kg (50,000 lb) (the whale shark *Rhincodon typus,* a plankton feeder).

The world's shark populations are in danger from overfishing. Each year, more than 100 million elasmobranchs (sharks, skates, and rays), including 1.6 million metric tons, or approximately 3.5 billion lb of shark, or 10 million elasmobranchs for each human shark-related fatality, are killed in fisheries. Half of this total represents incidental by-catch (sharks caught in fishing nets or on longlines intended for swordfish or tuna).[19] The National Marine Fisheries Service estimates that 20 million metric tons of marine wildlife are killed and thrown back into the sea as by-catch. This activity may double the estimated shark mortality figures. Commercial fishing mortality of sharks in U.S. waters averages 20,000 metric tons (44,092,000 lb) per year. Great declines in shark populations along the east coast of the U.S. have occurred over the past two decades, a trend that is worldwide. Some commercially targeted species have declined by as much as 80%. The most dramatic declines were seen in dusky sharks. Innumerable animals are ground into fertilizer. More than 90% of captured sharks are discarded.

The fishery interest in sharks centers on the fins. These are of great value in the Orient, where they are made into shark fin soup, a traditional dish that signifies high economic status and is reputed to be an aphrodisiac. Interest in fins has also spawned the heinous practice of finning, in which a shark is captured, its fins are sliced off (Fig. 72-4), and then it is returned to the water (Fig. 72-5).[85] Shark-fin soup is sold for upward of $150 per bowl. The prepared fins themselves may sell for more than $800 per pound. It has been estimated that 350 tons of shark fins may be consumed each year. The International Commission for the Conservation of Atlantic Tunas created a ban on shark finning in November 2004, to join the United States, which banned shark finning in the Atlantic Ocean in 1993 and in the

Figure 72-4. Finning a shark. (Photo by Marty Snyderman.)

Figure 72-5. This dead shark is a victim of finning. (Photo by Marty Snyderman.)

Figure 72-6. *Watson and the Shark,* by John Singleton Copley, 1778. (Photograph © 2005 Museum of Fine Arts, Boston.)

Pacific Ocean in 2002, in such a prohibition. Shark flesh is a major food source in both developed (commonly the fish in European "fish and chips") and undeveloped (artisinal fisheries) countries. Mako shark flesh is similar to that of swordfish and often serves as a more than adequate culinary substitute. To date, sharks do not routinely appear to carry ciguatera toxin, except in the liver. However, serious poisoning from shark ingestion has been reported[24] (see Chapter 66).

In Tahiti and some other Polynesian locations, sharks are occasionally mercilessly killed simply to acquire their teeth for jewelry manufacture. The great white shark has been declared a protected species in South Africa, Australia, the Maldives, California, and the Atlantic waters of the United States. In 1991 the South African government declared the great white shark a protected species within 200 miles (322 km) of its coast. The U.S. government has lowered allowable fishing quotas in Atlantic waters, and Australia closely monitors its shark fisheries. In October 2004, a global wildlife treaty by the Convention on International Trade in Endangered Species (CITES) offered new protection to great white sharks. In other areas, however, sharks and their relatives are largely unregulated and populations are in serious decline. Some species, especially those that enter rivers, are potentially at risk of extinction. This is in part due to the fact that sharks have slow growth rate, late sexual maturation, and low reproductive capacity.

Life and Habits

Sharks (from *xoc,* pronounced "shock," a Yucatee word from the Mayan language and a glyph for "fish") have inhabited the oceans for at least 400 million years.[20] They appeared on the planet during the Devonian period, approximately 200 million years before the dinosaurs. Indeed, many living species of sharks belong to the same genera as species from the Cretaceous period, one hundred million years ago.[128] Ancestral sharks may have been enormous; *Carcharodon megalodon,* which roamed the seas between 20 million and 1.5 million years ago, probably grew to a length of more than 15 m (49 ft), with teeth longer than 15.2 cm (6 inches). This was a predator of astronomic proportions that apparently largely fed on whales.

Some 35 of about 375 species of sharks have been implicated in the 75 to 100 shark attacks on humans that are estimated to occur annually worldwide, and another 35 to 40 species are considered potentially dangerous. Shark attack is perhaps first depicted on a vase dated circa 725 B.C. from the Island of Ischia west of modern day Naples, and it is recorded in early Greek literature.

U.S. coastal waters typically are the setting for nearly one quarter to one third of the annual number of shark attacks. The great American colonial painter John Singleton Copley's 1778 painting *Watson and the Shark* (Fig. 72-6), which depicts an encounter between the Englishman Brook Watson (1735–1807)

Figure 72-7. Great white shark *(Carcharodon carcharias)*. The most dangerous of sharks, this animal has been implicated in many attacks on humans. (Photo by Howard Hall.)

and a shark that bit off his right foot in Havana Harbor in 1749, is one of the earliest authenticated records of a shark attack.[20] ISAF has records of attacks going back into the mid-1500s. Between 6 and 10 deaths from shark attacks are reported worldwide each year. The most frequently documented offenders are the larger animals, such as the great white (Fig. 72-7), bull, and tiger *(Galeocerdo cuvieri)* sharks. However, blacktip *(Carcharhinus limbatus)* and possibly spinner *(Carcharhinus brevipinna)* sharks, which are thought to be involved in most of Florida's numerous (20 to 30 per year) minor bites, may be involved in even more incidents. Identification of attacking species is a difficult task because victims seldom see the attacker well enough to make an accurate identification and because identification of most shark species, especially requiem sharks of the family Carcharhinidae, is notoriously difficult, even for trained scientists. Definitive identification can be made by examining tooth fragments left behind in a wound, but this is an infrequent occurrence. Thus, easily identified species such as white, tiger, bull, and nurse sharks sit high on documented attacker lists whereas most requiem shark incidents are grossly underreported. Less commonly reported attackers include the sandtiger (ragged tooth or grey nurse), blue (Fig. 72-8), blacktip reef, bronze whaler, lemon (Fig. 72-9), shortfin mako (Fig. 72-10), grey reef, oceanic whitetip (Fig. 72-11), sandbar, sevengill, Caribbean reef, and dusky sharks (ISAF data). Hammerhead (Fig. 72-12), Ganges River, Galápagos, and nurse (Fig. 72-13) shark attacks are rarely reported. The famous series of attacks along the New Jersey shore in the summer of 1916 were attributed to a rogue great white shark. Tiger sharks are the most commonly identified attackers in the Hawaiian Islands and other tropical regions. In 2003, it was noted that reported unprovoked shark attacks worldwide had declined for 3 straight years, dropping from 79 in 2000 to 63 in 2002 to 55

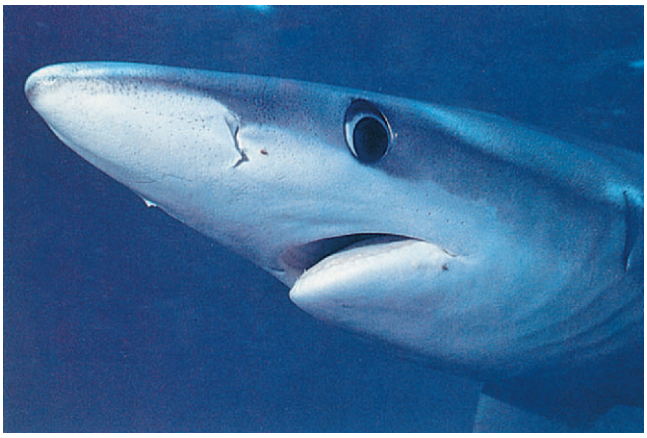


Figure 72-8. Blue shark, considered a dangerous species. (Photo by Marty Snyderman.)

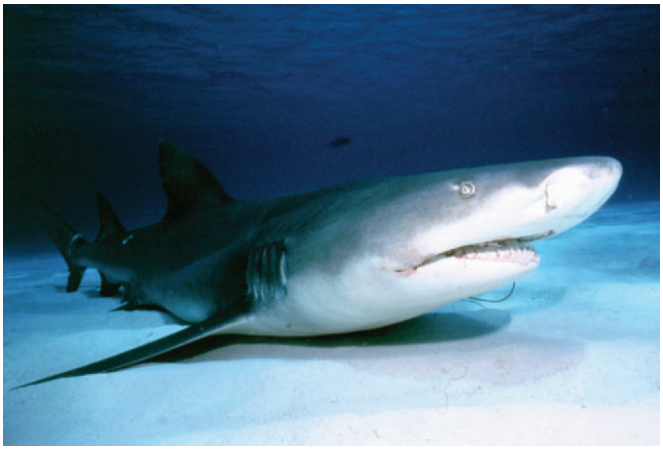


Figure 72-9. Lemon shark. (Photo by Howard Hall.)

Figure 72-10. Mako shark, *Isurus oxyrincus*. (Photo by Howard Hall.)

Figure 72-11. Oceanic whitetip shark, *Carcharhinus longimanus.* (© 2000 Norbert Wu, www.norbertwu.com)

Figure 72-14. Whale shark *(Rhincodon typus),* the largest fish in the sea, is fortunately a plankton eater. (© 2000 Norbert Wu, www.norbertwu.com)

Figure 72-12. Hammerhead sharks, schooling off Cocos Island. The positioning of the eyes is reputed to increase the peripheral vision of these apex predators. (Photo by Howard Hall.)

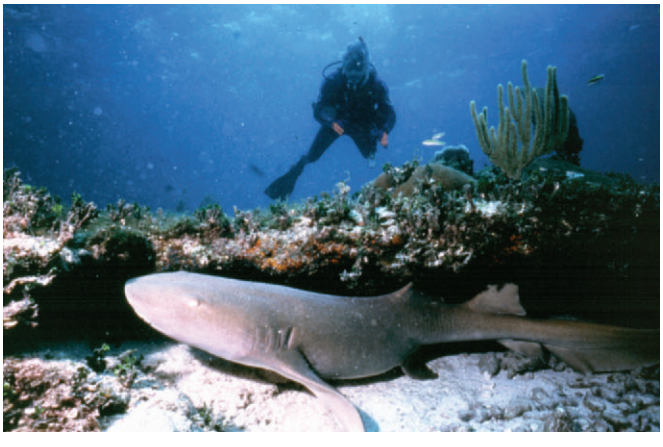


Figure 72-13. Diver with nurse shark. (Photo by Howard Hall.)

in 2003, with a similar decline in overall mortality from 13% in the 1990s to 7% in 2003.[157] Most attacks are reported from North America, with Florida (and its central east coast) leading the list. It is possible that people are becoming a bit more intelligent about when and where they enter the water.

Sharks are carnivorous; many are apex predators. Their danger to humans results from the combination of size, aggression, and dentition. Some species are also aggressive. The three largest sharks, the whale shark (the largest fish at 50 ft [15.2 m] in length and more than 40,000 lb [18,181 kg]), basking shark, and megamouth shark, eat plankton and use their teeth as filters (Fig. 72-14). Even small sharks have powerful jaws and sharp teeth. Any species reaching a length of 2 m (6.5 ft) must be considered potentially dangerous, because its dentition and jaw strength at that size can inflict serious injury on a human. White shark attacks are most common in the waters of southern Australia, the south coast of South Africa, the middle Atlantic coast of North America, and the American Pacific coast north of Point Conception, California. Attacks by great white sharks, which reach a length of nearly 20 ft (6 m) (making it the largest predatory shark), off the coast of northern California have led to the designation of a "red [or bloody] triangle" bordered on the north by Point Reyes and Tomales Bay, through the Farallon Islands to the west, and down south to Año Nuevo and Point Sur facing the Monterey Bay. This is a breeding area for elephant seals *(Mirounga angustirostris)* (Fig. 72-15), which yield 200-lb (91-kg) pups, perfect food for the immense predators. In an analysis of California attacks, Miller and Collier[91] attributed unprovoked attacks north of San Miguel Island to white sharks, while those south of this area involved members of the families Carcharhinidae (requiem sharks), Sphyrnidae (hammerheads, which can grow to more than 18 ft [5.5 m] in length), and possibly Squatinidae (angel sharks) (Fig. 72-16).

The white, tiger *(Galeocerdo cuvieri)* (up to 18 ft [5.5 m] and 2000 lb [909 kg]) (Fig. 72-17), and bull *(Carcharhinus leucas)* (11.5 ft [3.5 m]; 800 lb [364 kg]) sharks are considered the next most dangerous with regard to attacks on humans. The great hammerhead *(Sphyrna mokarran)* (Fig. 72-12) shark has a reputation as an attacker in equatorial waters, but its reputation is overstated, probably because of its appearance. The hammer-

Figure 72-15. The elephant seal, shown here swimming through a kelp bed, is a favorite food for the great white shark. (Photo by Howard Hall.)

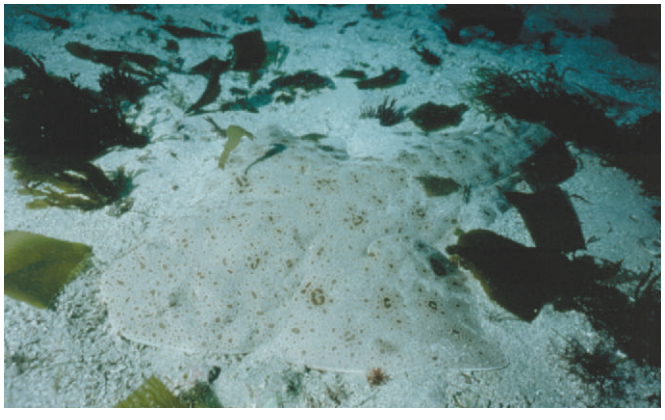


Figure 72-16. Pacific angel shark, *Squatina californica.* (Photo by Howard Hall.)

Figure 72-17. Tiger shark. (Photo by Marty Snyderman.)

Figure 72-18. Lateral eye placement of the hammerhead shark. (Photo by Marty Snyderman.)

head has ampullae scattered over its entire undersurface that are sensitive to electromagnetic fields, which in combination with its highly developed sense of smell and lateral eye placement (Fig. 72-18), may make this shark a superior predator. In the pelagic realm, the oceanic whitetip shark is to be respected because of its aggressive nature. This species was implicated in many of the attacks and scavenge bites on survivors of the *USS Indianapolis*.

Sharks are members of the class Chondrichthyes (subclass Elasmobranchii), or cartilaginous (skeleton) fish, which also includes skates, rays, and chimaeras. The class comprises approximately 780 species.[34] Unlike many bony fish, sharks are not buoyant because they do not possess swim bladders for flotation. In most species, the large liver, which contains the oil squalene, contributes to their limited buoyancy. Thus, most sharks must stay in nearly constant motion to keep from sinking and to drive oxygenated water past the gills. Only a few bottom-dwelling species rest for extended periods of time. Although sharks are not highly intelligent, they are endowed with remarkable sensory systems. These systems allow the shark to locate struggling fish, swimmers, or divers. Their color vision is poor but well compensated for by the acute perception of motion; the eye musculature is adapted for fixation with any body motion. Whether or not sharks are attracted to certain colors, such as international orange, is not entirely determined, but research biologists refer to this color as "yum yum yellow." The empirical observation is that sharks seem to be attracted to bright colors in preference to black. The tapetum lucidum ("bright tapestry," responsible for the eye shine seen at night) is a series of reflecting plates containing silver guanine crystals in the choroidal layer behind the retina, which reflects light from a photoreceptor back along the same optical path to restimulate the rods and cones and thereby increase the sensitivity of the eye. This is present in sharks that are active in low-light environments.[135] The tapetum also functions to shield the retina

Figure 72-19. Blue shark bites a mackerel, demonstrating the nictitating membrane that protects the eyes. (Photo by Marty Snyderman.)

from bright light near the surface by manipulating pigment granules. The eyes of many species are protected by upper and lower eyelids and the nictitating membrane (Fig. 72-19). The great white shark, which does not have the membrane, rotates its eyes in the sockets to avoid injury. Keen olfactory and gustatory chemoreceptors permit taste and the recognition of blood, urine, or peritoneal fluid in the water (in some cases, 1 part blood in 100 million parts of water, or concentrations of fish flesh in dilutions of 1 part in 10 billion parts of seawater). Sharks are most sensitive to chemicals that are similar to those produced by normal prey, such as amino acids, amines, and small fatty acids.[95] The nostrils are located on the underside of the snout just in front of the mouth. They open into sacs lined with folds of tissue containing cells used to detect smells. The odor-detecting system is constantly bathed by currents of water in both the resting and moving state.

Sharks have relatively large brains for their body size. Up to two thirds of the shark brain can be devoted to smell. The nostrils are the openings of the olfactory organs and do not take part in breathing, which is accomplished by oxygen extraction from the water through a series of five to seven gill slits. Additionally, sharks possess skin chemoreceptors that detect chemical irritants. The lateral line organs are small holes along the sides of the shark's body that register motion in the water. The lateral line system extending from the back along the side to the tail responds to sonic vibrations or pulsed low-frequency (20 to 60 cycles/sec; <800 Hz) sound waves.[34] Perhaps the most important series of telereceptors is located about the head, within the jelly-filled ampullae of Lorenzini, which are extremely sensitive to electrical voltage gradients (generated by the muscle contractions of fish; down to a $1/10^9$ volt per cm). Continuing research is directed at delineating the piscine ability to recognize electric fields. For instance, the common smooth dogfish *(Mustelus canis)* can detect an electrical voltage gradient of 5/1000 of a microvolt. Sharks also have extremely sensitive hearing, which may detect prey underwater from a distance of 914 m (3000 ft). Hammerhead sharks have a laterally expanded skull (cephalofoil), which spreads sensory receptors over a wider area and perhaps provides anterior lift during swimming.

Shark skin *(shagreen)* is composed in part by placoid scales (dermal denticles). These microscopic appendages have the same origin as teeth, with a pulp cavity, dentine, and vitreodentine (enameloid) covering. The denticles are embedded in the skin of sharks and rays, and protect them from predators and parasites, reduce mechanical abrasion to the animal, accommodate sensory organs, and minimize swimming-induced drag.[127] Some sharks can change color because melanin in the skin darkens near the ocean surface and lightens at depth distant from sunlight. Scale rasping behavior has been demonstrated in the juvenile lesser spotted dogfish *(Scyliorhinus canicula)*, which uses this body armor to anchor food items near its tail in order to allow bite-sized pieces to be torn away by rapid jaw and head movements.

Research has been conducted on isolation of potential antineoplastic agents from shark cartilage, organs, and body fluids.[38,78,103,105] For instance, sphyrnastatins have been isolated from the hammerhead shark *Sphyrna lewini*.[105] Squalamine, produced by the spiny dogfish, is a low-molecular-weight aminosterol as well as a broad-based antibiotic with antibacterial, antifungal, and antitumor properties. Squalene derived from shark liver boosted the immune system in experimental mice with sarcoma. Shark cartilage (which despite marketing claims does not appear to have significant antineoplastic activity) is used in the creation of artificial skin for humans and is an ingredient in cosmetics, and shark blood and liver oil are being investigated for hematologic and immunologic properties. The latter is used in preparations to shrink hemorrhoids. Angiogenesis inhibitors (U-995 from the blue shark *Prionace glauca* and water-soluble neovastat [AE-941]) have been derived from shark cartilage.[38,44] Shark cartilage may also affect lymphocyte migration into murine tumors.[49]

Shark Feeding and Attack

As previously noted, sharks are well equipped in the sensory aspects of feeding. They seem particularly able to avoid detection by potential prey, by virtue of coloration and a stealthy approach. Sharks feed in two basic patterns: (1) normal or subdued, with slow, purposeful group movements, and (2) frenzied or mob, as the result of an inciting event. The latter is precipitated by the sudden presentation of commotion or food or blood in the water. Frenzied behavior is enhanced by the proximity of other sharks in large numbers. In a frenzy, sharks become fearless and savage, snapping at anything and everything, including each other. Shark feeding frenzies are rare in nature, but may occur in association with major aviation or maritime disasters. Most reports of feeding frenzies have been the result of humans providing food to induce the event. After a shark decides to attack, it may "posture," swimming erratically with elevated snout, arched back, pectoral fin depression, stiff lateral bending of the body, and rapid tail motion, in contrast to its normal sinuous and graceful swimming style.[30,63] In bursts of speed a shark can use its powerful caudal fin muscles and attain speeds in the water of 20 to 40 miles (32 to 64 km) per hour. The mako shark swims with a tail sweep similar to that employed by tuna, facilitated by interposition of aerobic, fatigue-resistant red muscle and anaerobic, rapidly fatiguing white muscle.[131] Because sharks do not possess a swim bladder, they can ascend rapidly in pursuit of prey without incurring barotrauma.[128] As the carchariniform shark prepares to strike, it rapidly opens and closes its jaws up to three times each second, depresses the pectoral fins (from horizontal to as much as 60 degrees downward) in a braking action, and elevates the head. During a bite of a large prey item, the shark shakes its head and forebody in an effort to tear flesh from its prey. If the

object of such a bite is human, there may be gouge marks left on bones or teeth shattered and embedded in the human skeleton. The white shark often bites and releases its prey prior to returning to consume the weakened creature. Large sharks swallow food whole without chewing it. Some smaller sharks or juveniles, perhaps because they are gape-limited with respect to feeding, may repeatedly suck and spit prey into and out of the mouth in order to reduce food in size.[96] Analysis of human remains recovered from a tiger shark indicate that the human victim was dismembered and then swallowed and digested.[62]

It is difficult to postulate hunger as the sole attack motive, since more than 70% of victims are bitten only once or twice. "Hit-and-run" attacks, in which the shark bites, then releases and leaves the area, are most common. Usually the spike-like lower teeth are used first in feeding; these anchor the victim to allow the serrated upper teeth to carve through the flesh. Solitary upper tooth slashes might indicate attacks unrelated to feeding. Up to 60% of wounds involve only the upper teeth. To aid in cutting, the shark shakes or rolls its head. At the moment of the strike the shark rolls its eyes back in the sockets and uses the ampullae of Lorenzini to home in on the victim.

Sharks commonly attack young, old, injured, or sick prey. They are selective feeders with clear dietary preferences. Sea turtles, penguins, seals, and stingrays are consumed by certain large shark species; fish, squid, and shrimp are commonly taken by moderate- and small-sized species. Humans are not preferred food items because they are not normal denizens of the sea. Sharks often eat other sharks. The great white shark cruises along the bottom of the ocean preparing to launch an attack on an unsuspecting surface animal. It can strike with enough force to lift the animal out of the water and breach itself, tearing a 50-lb chunk of flesh from its victim or even decapitating the animal (Fig. 72-20). The cookie-cutter (or cigar) sharks *Isistius brasiliensis* (Fig. 72-21) and *Isistius plutodus* (Fig. 72-22) create circular craterlike wounds approximately 5 to 6 cm (2 to 2.4 in) in diameter when they attack pinnipeds, tunas, billfishes, and other large fish. Sharks have short intestines, seem to be able to selectively digest ingested foodstuffs, and may be able to keep portions of what they ingest intact for prolonged periods of time, perhaps as a method to regulate nourishment. Relatively intact human limb segments have been removed from shark stomachs many days after ingestion.[27]

Figure 72-20. Great white shark *(Carcharodon carcharias)* with open jaws on surface, Neptune Islands, South Australia. Protected species. (© Gary Bell/oceanwideimages.com)

Figure 72-21. Cookie-cutter shark *(Isistius brasiliensis)*. (© 2000 Norbert Wu, www.norbertwu.com)

Figure 72-22. The cookie-cutter shark *(Isistius plutodus)* cuts pieces of flesh off its prey with its unusual tooth configuration. (Photo courtesy of George Burgess.)

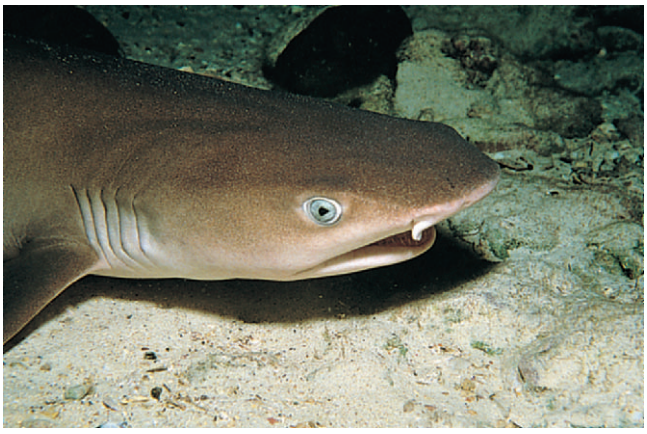


Figure 72-23. Whitetip reef shark. This species tends to be fairly docile. (Photo by Paul S. Auerbach, MD.)

Figure 72-24. Shark bite of the buttocks and thigh. (Courtesy T. Hattori, MD.)

Figure 72-25. The presence of a shark tooth in the arm is revealed by x-ray examination.

It is difficult to generalize about shark attacks on humans. Most attacks likely occur as cases of mistaken identity in which the shark misinterprets the splashing of humans at or near the water surface as the activity of normal prey items. Less commonly, attacks may be direct feeding events in which large sharks simply perceive the human as appropriate sized and demonstrating appropriate behavior patterns. Finally, overt agonist behavior related to territoriality or personal space may contribute to some attacks, especially in the reef environment. Current explanations suggest that frightened persons engaged in erratic escape activity are more likely to incite attack. This has been demonstrated in the case of the gray reef shark, *Carcharhinus amblyrhynchos*. Aggression may be aggravated by purely anomalous behavior, the violation of courtship patterns, or territorial invasion. More docile behavior tends to be the rule with other reef sharks, such as the silvertip *(Carcharhinus albimarginatus)*, blackfin *(C. melanopterus)*, or whitetip *(Triaenodon obesus)* (Fig. 72-23).

Arms and especially lower legs are the most common trauma regions in shark attacks on humans. The legs and buttocks are most commonly bitten (Fig. 72-24), perhaps an indication of dangling legs or buttocks displayed by an abalone diver facing a rock or ledge. Since sharks do not chew their food, their method of biting and rolling or thrashing allows flesh to be stripped from the victim. Teeth may be embedded in prey (Fig. 72-25), but fractured bones are surprisingly rare.[62]

The great white shark *(Carcharodon carcharias)* has been captured off Cuba at a length of 19.5 ft (5.9 m) and an estimated weight of 4500 lb (2045 kg); it is claimed, but not proven, that it can attain a length of 25 ft (7.6 m) and a weight of 5500 lb (2500 kg). It attains maturity at a length of approximately 2.5 m (8.2 ft). It is a man-attacker, but not always a man-eater. This statement reflects the observation that this highly feared animal usually releases its victim following a single "inquisitory" bite, a behavior it also employs on floating pieces of Styrofoam, surfboards, and marine mammals it does not consume, such as sea otters. Humans may survive and avoid consumption by having the ability to retreat to boats or surfboards prior to return of the shark, a luxury unavailable to the white shark's normal prey. This is small consolation to the unfortunate victim, who may have an entire hemithorax or limb removed (Fig. 72-26). The great white shark has only recently been closely observed in the wild and is thus the subject of much speculation about predation strategies.[89] The feared trait of the great white sharks is that they initiate contact with humans.[46] Their unpredictable nature ranges from a seemingly docile approach to a research boat to a powerful attack on a surface sea lion. Since adults feed largely on pinnipeds, the "bite-and-spit" behavior is considered a means of avoiding injury from struggling prey (Fig. 72-27) while allowing the prey to weaken from exsanguination. One theory is that a shark that largely consumes a human victim does so because the victim was solitary in the water.[47] Breath-hold diver behavior and the similarity of the silhouette of a contemporary surfboard to that of a surface seal may be responsible for attacks on humans. Most attacks on humans occur at the water's surface. One fatal attack in 1989 with two victims was on sea kayakers off the coast of southern California. At the time of this writing, there have been (since 1580) 212 documented unprovoked attacks by great

Figure 72-26. Victim of great white shark attack. **A,** Damaged surfboard ridden by the victim. **B,** Massive thoracic injury after a single bite. It was estimated that the shark was 20 ft (6 m) long. (Courtesy P. Crossman, Coroner's Division, Salinas, CA.)

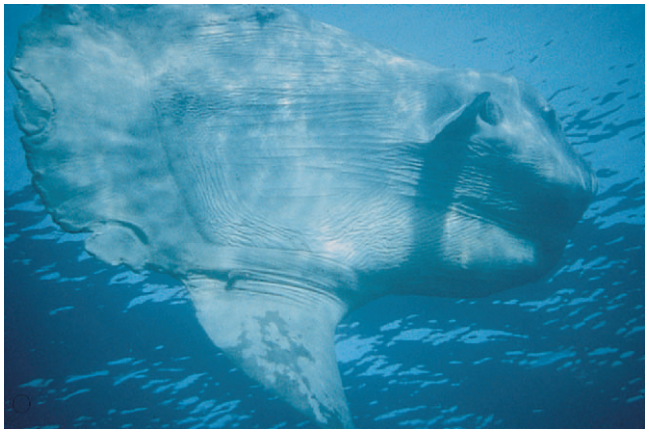


Figure 72-27. Giant sunfish *(Mola mola).* Note the shark bites on the posterior end of the fish. (Photo by Paul S. Auerbach, MD.)

white sharks upon humans, with approximately 61 fatalities (ISAF data). The most recent attack statistics for most species can be obtained from the International Shark Attack File at the Internet URL http://www.flmnh.ufl.edu/fish/sharks/statistics/species2.htm.

Shark attacks have occurred from the upper Adriatic Sea to southern New Zealand, with most between latitudes 46° N and 47° S, likely because of the increased presence of humans in warmer waters. The odds of being attacked by a shark along the North American coastline are approximately 1 in 11.5 million (ISAF data). The danger is greater during the summer months (more persons in the water), in recreational areas, during late afternoon and nighttime feeding, and in murky, warm (20° C [68° F]) water. White sharks prefer cooler water, and attacks have occurred in waters as cold as 10° C [50° F]).[175] Attacks in northern California occur more frequently in clearer water at temperatures of less than 16° C [60° F]).[91] Shark attacks in Hawaiian waters are relatively rare, with the exception of recent events.[153] Tiger shark increases in Hawaii may be due in part to attraction to seagoing green turtles that come close to land, but most likely are simply reflective of rising human recreational utilization (especially surfing) of coastal waters.

Although most attacks occur within 100 ft (30.5 m) of shore, this is an artifact of the high human density in this region rather than a reflection on shark distribution. Other areas, such as mouths of rivers and inlets, edges of channels and dropoffs, and reefs and other hard-bottom relief are areas of shark abundance. Because of their ability to detect contrasts, sharks have a predilection to attack bright, contrasting, or reflective objects. Movement is an added attraction to sharks, which have been known to bite surfboards, boats and propellors, and buoys. Some shark attacks in northern California coastal waters have involved swimmers on surfboards (black on white), who entered migratory elephant seal (white shark food) habitats (Fig. 72-28). Great white sharks congregate in the autumn in the waters around the ten rocky Farallon inlands 27 miles off the Golden Gate Bridge near San Francisco, where they actively pursue seals.

Most victims are attacked by single sharks, violently and without warning. In the majority of attacks the victim does not see the shark before the attack. The most common (80%) attack type is hit and run, in which the victim is seized and released, or slashed on an extremity.[171,172] This frequently occurs in shallow water and has been attributed to (1) mistaken identity of potential prey, (2) juvenile sharks with poor predatory ability, or (3) situational provocation.[16,26] Another type is a "sneak" attack upon a diver or swimmer in deeper water whereby a

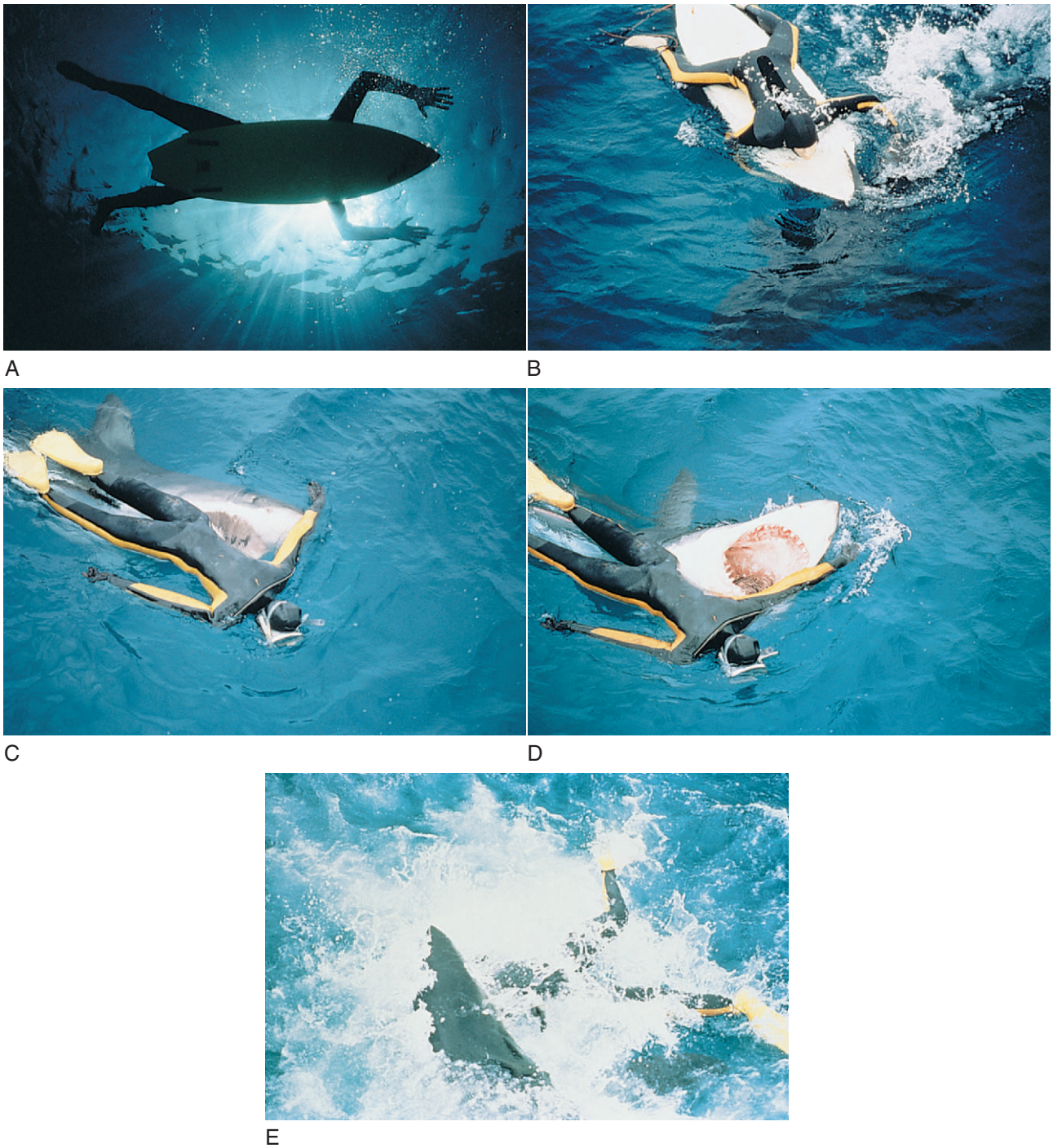


Figure 72-28. **A,** Silhouette of diver on surfboard to demonstrate similarity in shadow and contour to a sea lion at the surface. **B,** A great white shark passes underneath a dummy on a surfboard before making an attack. **C,** The shark begins to elevate its head from the water. **D,** With jaws wide, the shark attacks the dummy. **E,** With great commotion, the dummy and surfboard are dragged beneath the surface. (All photos courtesy of Images Unlimited, Inc. **A,** Al Giddings. **B,** Rosemary Chastney. **C** to **E,** Walt Clayton.)

Figure 72-29. Teeth of bull shark *(Carcharhinus leucas)*. (© 2000 Norbert Wu, www.norbertwu.com)

shark attacks without being seen. Finally, "bump-and-bite" attacks have the shark make contact with the victim prior to actually biting. This may be an attempt by the shark to determine the defensive abilities of its prey or to wound the victim before the definitive strike. A shark may circle a victim prior to bumping. Severe skin abrasions and bruising from the shark skin can be produced in this manner. The sneak and bump-and-bite attacks generally are perpetrated by larger animals and are thought to be part of intentional feeding behavior.

Clinical Aspects

The jaws of the major carnivorous sharks are crescent-shaped and contain up to five or six rows or series of sharp ripsaw triangular teeth, which are replaced every few weeks by advancing inner rows (Fig. 72-29). The teeth of sharks are not attached directly to the jaw cartilage, but are held in place by a collagenous membrane in a shallow depression known as the tooth bed.[95] Each species has distinctively shaped teeth.[135] However, teeth of the great white shark reveal no consistent pattern of size or arrangement of the marginal serrations that are sufficiently characteristic within an individual shark to serve as a reliable index of identification of a tooth as originating from that particular shark. Tooth shape and size are useful in determining its place in the jaw, and serrations are sufficiently distinctive to enable the potential identification of an individual tooth as having been the cause of a particular bitemark.[98] Furthermore, a nomogram has been developed that can be used to estimate the body length of a great white shark from measurements of the tooth or bite mark morphology.[99] Although normal tooth replacement takes 7 to 14 days, in some species a lost tooth can be replaced within 24 hours. Sharks are born with a full set of teeth, and in some cases have up to 15 rows of replacement teeth available. Amazingly, some sharks produce up to 25,000 teeth in a lifetime, as teeth are shed frequently; this explains the abundance of fossil shark teeth in paleontological excavations. The upper jaws generally have larger cutting teeth, whereas the sharp lower teeth are designed to fasten onto and hold prey during capture.[55] The bamboo shark *(Chiloscyllium plagiosum)*, which dines largely on crustaceans, is able to fold its teeth inwards on a flexible broad ligament in order to crush and consume its normal prey.[120]

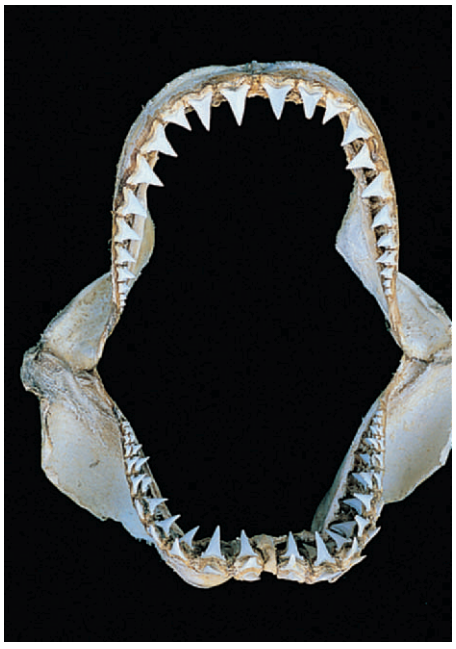


Figure 72-30. Jaw of a great white shark. (© 2000 Norbert Wu, www.norbertwu.com)

Shark teeth are cartilaginous, strengthened by the deposition of calcium phosphate crystals (apatite) in a protein matrix, all covered by an enameloid substance. They are considered to be as hard as granite and as strong as steel. In a large great white shark, the largest serrated triangular teeth can grow to 2.5 inches (6.4 cm). There are 26 upper and 24 lower teeth exposed in the front row. The height of the enamel of the largest tooth in the upper jaw is proportional to the animal's length, so a body length of up to 25 ft (7.6 m) may be possible based on recovered teeth. Teeth and fragments embedded in clothing or the victim may assist in identification of the biting species. The upper jaw is advanced forward and protruded to allow its participation in the biting action (Fig. 72-30). The biting force of dusky sharks is estimated at 18 tons per square inch across the tooth tips. By comparison, humans have been estimated to bite with a force of 15 tons per square inch. Severe shark bites result acutely in massive tissue loss, hemorrhage, shock, and death. Even a smaller animal, such as a lemon shark, can bite with bone-crushing force.[56] The potential for destruction is enormous, in parallel with other predators, such as tigers, lions and bears.

The human leg(s) is most frequently bitten, as the shark most often attacks from below and behind (Fig. 72-31). This is followed in frequency by the hand(s) and arm(s), as the victim attempts to fend off the shark.[27] Degloving injuries occur when victims attempt to pull an extremity from the mouth of a biting shark, raking soft tissue across sharp teeth (Fig. 72-32). Skin lacerations may display tooth patterns (Fig. 72-33). Proximal femoral artery disruption carries a poor prognosis because of the torrential hemorrhage (Fig. 72-34). Although fractures are not common, broken ribs often accompany intrathoracic, intraperitoneal, and retroperitoneal injuries. Spiral gouges on bones indicate the bite and roll activity of an attacking shark. Because the victim is generally far from medical assistance,

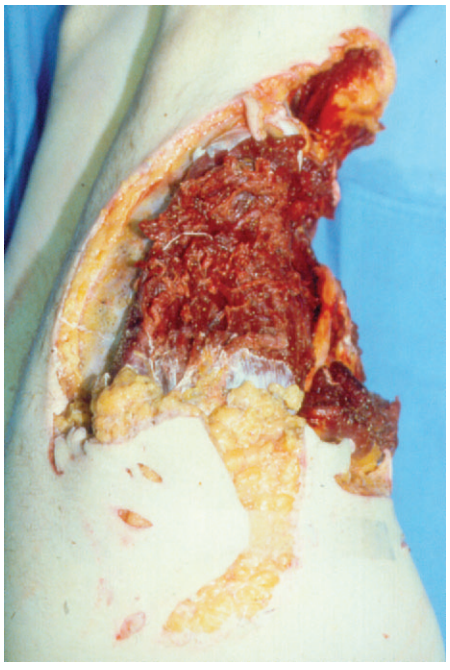


Figure 72-31. Typical thigh wound from shark bite. (Photo courtesy of George Burgess.)

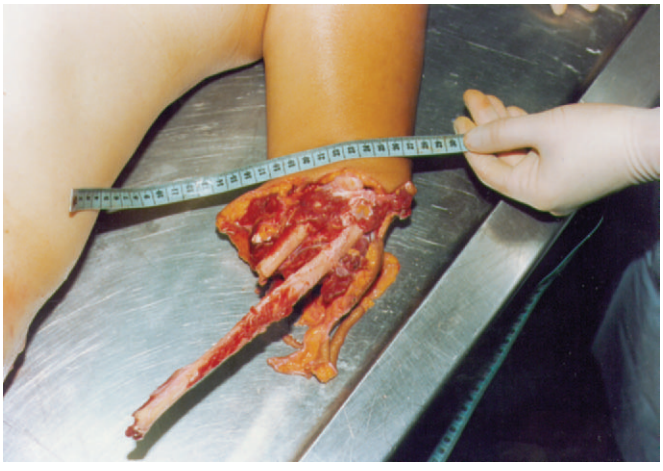


Figure 72-32. Tiger shark "bite and spin" behavior creates a degloving injury. (Photo courtesy of George Burgess.)

A

B

Figure 72-33. **A,** Shark bite creates wound edge that displays tooth pattern. **B,** Tooth pattern and wounds created by bite of a tiger shark. (Photos courtesy of George Burgess.)

blood loss may be profound. The wounds have historically been fatal in 15% to 25% of attacks (but have averaged about 10% over the past decade), with major causes of death listed as hemorrhage and drowning. Rapid response and prehospital care are undoubtedly improving this statistic, as have advances in major trauma treatment gained in part from lessons learned in otherwise regrettable 20th century wars.

Postmortem bites from sharks and other marine creatures, such as sea lice, can create the appearance of inflicted injury and obscure the true cause of death.[28] It therefore becomes important to differentiate postmortem artifacts from injuries that may have been inflicted while the victim was alive. For instance, the drowned victim may be bitten by sharks. If the wounds do not penetrate blood vessels or vital structures, they should be suspected of being noncontributory to the fatal event. However, when only part of a body is recovered, it is difficult to determine if a shark bite was the inciting event. Furthermore, sharks are capable of maintaining tissues within their stomachs for weeks at a time without digestion occurring, which may further obscure determination of time of death.[84] Bite configurations may be pathognomonic for some species, but species identification is difficult in most cases. The circular and C-shaped injuries

Figure 72-34. Shark bite of the proximal thigh. This unfortunate victim exsanguinated from a disrupted femoral artery. (Courtesy Kenneth W. Kizer, MD.)

A

B

Figure 72-35. **A,** Jaws of the cookie-cutter shark. **B,** Post-mortem wounds created by cookie-cutter shark bites. (Photos courtesy Y. Makino.)

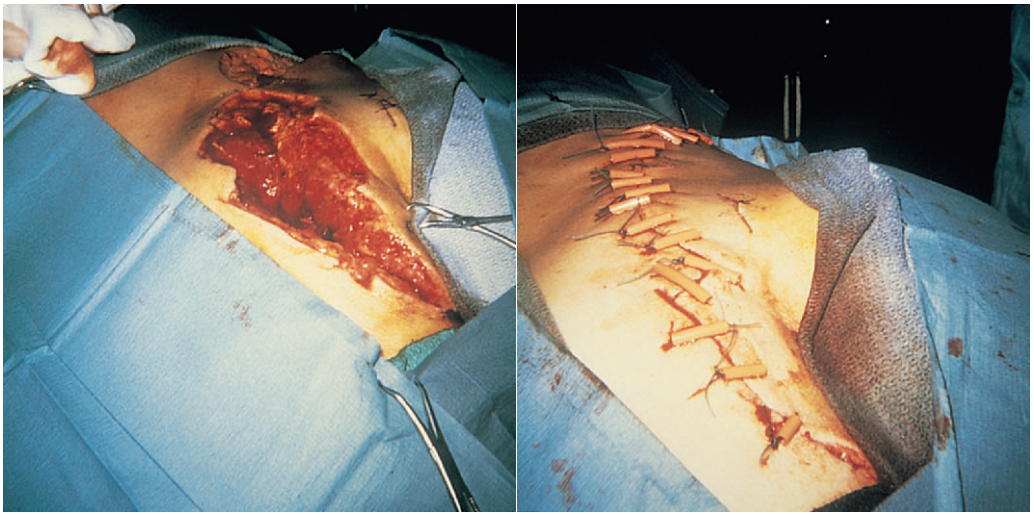
inflicted by the cookie-cutter shark *(Isistius brasiliensis, I. plutodus)* are made by small erect teeth in the upper jaw and triangular teeth in the lower jaw, resulting in the distinctive carving of a plug of flesh (Fig. 72-35).[156] However, there are no documented cases of attack by these small pelagic species on live humans.

Sharks can grow to be quite large and are strong creatures. Fishers are injured by thrashing animals on boat decks and in fishing nets. In one unusual incident reported to the author (PSA) by Dr. Edward Paget, the captain of a fishing ship jumped into the ocean in order to free a whale shark caught in a net. The enormous animal bumped him against the ship. The captain suffered mild abdominal pain, which worsened to the extent that he sought care in a hospital at Majuro in the Marshall Islands. He was transferred 300 miles by airplane to Dr. Paget, who observed signs of blood loss and localized peritonitis. At surgery, the victim was found to have 3400 mL of intraperitoneal blood with clots and a gangrenous 2-inch segment of sigmoid colon that necessitated a diverting colostomy.

Treatment

In most cases the immediate threat to life is hypovolemic shock. In one series compiled by the South African Natal Sharks Board, death occurred most frequently as a result of exsanguinating hemorrhage from a limb vascular injury. Thus it is occasionally necessary to compress wounds or manually to constrict arterial bleeding while the victim is in the water. As soon as the victim is out of the water, all means available must be used to ligate large, disrupted arteries or to apply compression dressings. In once recent incident, dental floss was used by a fast-thinking companion of the victim to ligate a severed vessel while in the field, likely saving the victim's life. If necessary, judicious use of pressure points or tourniquets should be entertained, taking care to avoid excessive ischemia time in treated tissue. If intravascular volume must be replaced in large quantities, at least two large-bore IV lines should be inserted into the uninvolved extremities to deliver crystalloid (lactated Ringer's solution, normal saline, or hypertonic saline), colloid, or blood products.[97,136] If prolonged transport is necessary, the rescuers should be prepared to attempt "central" IV access.[109]

The victim should be kept well oxygenated and warm while being transported to a facility equipped to handle major trauma. Central venous cannulation should be achieved if necessary. Blood losses should be replaced with whole blood or packed red blood cells and fresh-frozen plasma.[137] The precise ratio of crystalloid to blood products and proper mean arterial blood pressure endpoint of primary resuscitation in the presence of a major vascular injury are the subjects of ongoing investigations.[130] The victim should be thoroughly examined for evidence of cervical, intrathoracic, and intra-abdominal injuries. Because *Clostridium* can be cultured from ocean water, tetanus toxoid 0.5 mL intramuscularly (IM) and tetanus immune globulin (Hyper-Tet, Cutter) 250 to 500 units IM must be given. The administration of prophylactic antibiotics is more controversial. The victim of a shark bite should be treated with an IV third-generation cephalosporin, trimethoprim-sulfamethoxazole, an aminoglycoside, ciprofloxacin, or some reasonable combination of these agents. Imipenem-cilastatin should be reserved for established wound infections or early indications of septicemia, particularly in the setting of immunosuppression. The rationale



A B

Figure 72-36. Operative repair of shark bite wound shown in Figure 72-24. **A,** Debridement and exploration of the wound in the operating room. **B,** Proper closure technique with tension-releasing sutures. (Courtesy T. Hattori, MD.)

for prophylactic antibiotics is that shark wounds are prone to heavy contamination with seawater, sand, plant debris, shark teeth, and shark mouth flora. After a clinical infection is recognized, wounds should be cultured for aerobes and anaerobes by insertion of sterile swabs deeply into available lesions.

Proper operative intervention is mandatory.[173,174] It is inappropriate to attempt emergency department exploration of what often prove to be extensive and complicated wounds. In the operating room, devitalized tissue should be widely debrided and the wound irrigated copiously to remove all foreign material (Fig. 72-36). An x-ray may reveal one or more shark teeth in the wound (see Figure 72-25). Shark teeth and fragments should be identified and removed to avoid infection initiated by a contaminated foreign body. Vascular repair should be completed when necessary.[156] Unless it is absolutely necessary to achieve tight closure, the wound should be closed loosely around multiple drains (preferably closed systems) or packed open to await delayed primary closure. Although there is debate about whether to use internal or external fixation of grossly open and contaminated fractures, it seems logical to recommend surgical stabilization to facilitate vascular and soft tissue repair. In the pediatric population, damage to the physis and future limb length discrepancy should be anticipated.[56]

The abrasion associated with a shark bumping should be managed like a second-degree burn, with daily debridement and application of antiseptic ointment.

Postoperative management may be prolonged and complicated by acute renal failure attributed to hypovolemia and shock, massive blood transfusion, myoglobinuria, and administration of nephrotoxic antibiotics. Rehabilitation may include creation of prosthetic devices.

A reasonable "shark pack" should be available in emergency facilities and rescue vehicles near shark-infested waters. This must be portable and should include items necessary to control hemorrhage and initiate IV therapy.

Prevention

Every precaution should be taken to avoid shark attack, beginning with an intimate knowledge of the local waters. The following is precautionary advice and a list of alternatives for action in the event of a confrontation:

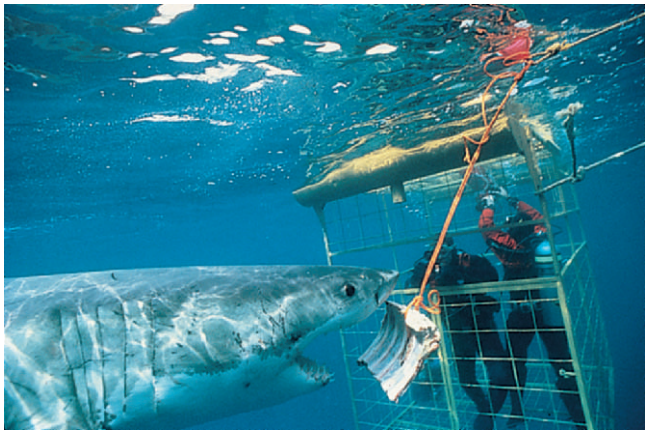


Figure 72-37. Caged divers view an inquisitive great white shark. (Photo by Carl Roessler.)

1. Avoid shark-inhabited water, particularly at dusk and at night. Do not swim through schools of bait fish in the presence of sharks. Do not enter waters posted with shark warnings. Do not wander too far from shore. Surfers are generally at greater risk than divers. Do not disguise yourself as a pinniped (seal). Do not swim with animals (such as dogs or horses) in shark waters. Shark behavior can be unpredictable, so it is best not to remain in the water with sharks, particularly if you are fearful. Although some persons believe that sharks can be domesticated, there is no such thing as a friendly shark. Photograph hazardous sharks from within the confines of a protective cage (Fig. 72-37), rather than from an unprotected position.
2. Swimmers should remain in groups. Isolation creates a primary target, eliminates companion surveillance, and removes the opportunity for post-attack assistance. When

Figure 72-38. Blue water hunter with white sea bass off Catalina Island, California. (© 2000 Norbert Wu, www.norbertwu.com)

Figure 72-39. Diver carrying a "bang stick" in the presence of a blue shark. (Photo by Howard Hall.)

diving, maintain constant vigilance. Do not wander far from shore, particularly if you are a solitary swimmer.

3. Turbid water, drop-offs, deep channels, inlets, mouths of rivers, and sanitation waste outlets are areas frequented by larger sharks and should be avoided. Water clarity is cited as turbid in 64% of all attack incidents, so it appears that sharks attack at least one third of the time in clear water. Humans are most often attacked in shallow water or beyond the breakers. Do not swim in waters frequented by recreational or commercial fishers. Do not swim in water that has been recently churned up by a storm. Be alert when crossing sandbars.
4. Blood and other body fluids (including peritoneal fluid) attract sharks. No person should be in shark waters with an open wound. Women have historically been advised to avoid diving during menstruation, although there are no data to support attraction of sharks to the discharge of menstruation. Given the shark's well-documented acute sense of smell, it certainly seems possible that menstrual blood is attractive.
5. Brightly colored swimwear or diving equipment and shiny snorkeling gear attract certain sharks. Bright (international) orange and other contrasting colors appear to be particularly attractive to sharks. Flat black is probably the least attractive color. There is scant evidence that sharks are more attracted to light-skinned bathers, but differential tanning and base skin color producing lighter palms of the hands and soles of the feet may contribute to bites to these appendages, especially when movement is involved.
6. Captured fish must be tethered at a distance from any divers. There is no greater chemical attractant for a shark than fish blood, and the thrashing of a speared fish is greatly attractive. Do not dive or swim in the presence of spearfishermen (Fig. 72-38). Divers who harvest abalone should be aware that the banging and prying noise of an "abalone iron" might attract sharks.
7. The presence of porpoises in the water does not preclude the presence of sharks (it is not uncommon for both to be pursuing the same prey items). Be alert for the presence of a shark whenever schools of fish behave in an erratic manner or when pods of porpoises cluster more tightly or head toward shore.
8. Do not tease or corner a shark. This is particularly true with captive animals. Do not pull on a shark's tail. Do not chase after a shark. If a shark begins to act in an erratic manner, do not photograph it at close range using a strobe flash apparatus. There is no evidence that shark feeding, as perpetrated by shark diving operators, causes sharks to attack humans.
9. If a shark appears in shallow water, swimmers should leave the water with slow, purposeful movements, facing the shark if possible and avoiding erratic behavior that could be interpreted as distress. If a shark approaches in deep water, the diver should remain submerged, rather than wildly swimming to the surface to escape. The diver should move to defensive terrain with posterior protection to fend off, as best as possible, a frontal attack. It is inadvisable to trap a shark in a position from which it must attack to obtain freedom. Fighting sharks is difficult; they are best repulsed with blunt blows to the snout, eyes, or gills. If possible, the bare hand should not be used, to avoid severe abrasions or lacerations. A stream of air bubbles from a scuba regulator directed into the face of a shark may serve as an initial deterrent, but this does not work most of the time. Although spears, knives, shotgun shell- or 30.06-loaded powerheads ("bang sticks" [Fig. 72-39]), strychnine-filled spears, and carbon dioxide darts can kill small sharks, they can worsen the situation if they are misapplied or their application promotes frenzy in a school of sharks.
10. Do not splash on the surface or create a commotion in a manner that might cause a shark to interpret your behavior as that of a struggling fish. Surface activity, including the sound of marine engines and perhaps the sounds created by helicopter rotor wash, attracts sharks. During a helicopter rescue, exit the water as soon as possible.

Although there is no question that shark avoidance is the most reliable maneuver, shark defense techniques and repellents are constantly evolving. In response to shark attacks on downed airmen and sailors during World War II, copper acetate (20%)

Figure 72-40. Diver wearing chain mesh metal suit for shark protection. (Photo by Howard Hall.)

Figure 72-41. Moses sole, *Pardachirus marmoratus,* Thailand. (© 2000 Norbert Wu, www.norbertwu.com)

blended with a water-soluble wax and nigrosine-type black dye (80%) was packaged as a slowly dissolving (3 to 4 hours) 6-oz waxy cake for deployment as "shark chaser" by the Office of Naval Research of the U.S. Navy.[15] It was theorized that the dye released as a dark (often black or purple) cloud similar to defensive mollusk secretions, and that the copper acetate both inhibited feeding and resembled the decaying carcasses of sharks, so that sharks would thus be repelled. Unfortunately, while a morale booster, this was not a reliable deterrent. Its use was discontinued by the Navy in 1976. Limited progress has been made since that time. Recreational beaches in Australia and South Africa are protected with extensive gill net systems ("meshing") to kill animals seeking to enter the surf zone. These work by reducing population sizes over time, thereby minimizing shark-human interactions. This type of prevention measure is increasingly under attack by biologists and conservationists because of its highly negative impact on threatened sharks, sea turtles, sea birds, and sea mammals. Electric shark barriers ("cable device") using 0.8-msec pulses 15 times per second to create a field of 4 volts per meter seem to generate a fright response in sharks longer than 1.2 m (4 ft) and are being investigated.[31,123] Their benefits include repulsion rather than shark capture or destruction. However, some sharks respond weakly to these stimuli. Abalone divers in South Australia work from one-person, self-propelled shark cages.

Experimental devices for individuals include chain-mesh diving suits (Fig. 72-40), inflatable dull-colored plastic protective bags (yellow is easy for aircraft to spot, but most attractive to sharks), acoustic and hand-held electrical field transmitters, surfactants and other chemical repellents (e.g., firefly and the Red Sea and western Indian Ocean Moses sole [flatfish; *Pardachirus marmoratus*] (Fig. 72-41) glandular extract [pardaxin]).[41] Pardaxin is an excitatory polypeptide neurotoxin that forms voltage-gated pores and triggers neurotransmitter release.[77] The ichthyotoxic secretion from the fish, which appears as a milky substance from a series of glands located along the dorsal and anal fins, also contains shark repellent lipophilic constituents that appear to be steroid monoglycosides.[107,132] Another sole, *Pardachirus pavoninus,* which lives in the tropical region of the western Pacific and eastern Indian Ocean, secretes pavoninins 1 through 6. These shark-repelling substances are ichthyotoxic steroids, *N*-acetyl-glucosaminides.[154] It appears that in sharks, the gills or pharyngeal cavity is the target organ(s) for the repellent action. For the exudate from the Moses sole, concentrations of 10 to 25 g/m^3 are needed to elicit an immediate indication of repellency. However, it has been estimated that about 24 kg (53 lb) of any effective drug would have to be contained within an enveloping "drug cloud" in the volume of water through which a slowly approaching shark might swim in its final 10 seconds of approach as it attacked a human in the ocean.[15]

Studies have indicated that shark repellent efficacy of alkyl sulfate surfactants is due to their hydrophobic nature.[122] In tests conducted on juvenile swell sharks *(Cephaloscyllium ventriosum),* the aversive response of sharks to these surfactants increased with carbon chain length from octyl (8) to dodecyl (12), decreased with the addition of ethylene oxide groups and was not affected by counterions (e.g., magnesium for sodium). Still, for the most effective synthetic detergent repellent (e.g., sodium dodecyl sulfate), the concentration needed in the water is 800 g/m^3. These findings show that a chemical carried in a life jacket cannot be reliably useful against sharks, because to meet the Navy's potency requirement for a nondirectional surrounding-cloud–type repellent, it would have to be instantaneously effective at a concentration of no less than 100 parts per billion (0.1 µg/mL). Future direction of chemical shark repellent research include delineation of the morphological target sites of chemical and natural shark repellents, investigation of natural bioactive toxins as repellents, and identification and purification of semiochemicals (e.g., exudates or body secretions of predators that act as alarm pheromones) to be used as repellents.[122]

The Shark Shield series of shark deterrent devices are manufactured by SeaChange Technology Ltd. (www.sharkshield.com) in South Australia, based on the original technology that led to development of the SharkPOD (which is now discontinued). The Shark Shield produces an electrical field that is detected by sharks through the ampullae of Lorenzini and that is believed to cause discomfort and muscle spasms in the animals. The Shark Shield unit incorporates two electrodes, which project the field from the unit to create an invisible protective shield that surrounds the user. Both electrodes must be

immersed in the water in order to give protection to the wearer. One electrode is encased in a short antenna, which trails out from the ankle, secured with a neoprene cuff. The second electrode is a pad designed to be worn on the scuba cylinder. This pad electrode attaches to the cylinder by means of a special quick-release fastening device. Under no circumstances should the two electrodes be placed any closer than 900 mm, as this will reduce the deterrent effect. A full-length wetsuit or drysuit must be worn when using the Shark Shield Scuba unit to reduce the skin stimulation effect caused by the configuration of the two electrodes. The unit is strapped to the thigh or can be placed in the buoyancy compensator pocket. In tests conducted to date, the unit has deterred sharks at distances of up to 8 m (26 ft), but with an average distance of 4 to 5 m (13 to 16 ft) from the unit.

Observations about this device indicate that sharks already in the process of feeding may not be repelled by the device, so the current thought is that the device should be turned on at the beginning of a dive to create a situation of "exclusion." As for any shark-deterrent strategy, it cannot be assumed to provide perfect protection, because the animals are unpredictable, and it is impossible to state with assurance that they will always be deterred. A fatal attack by a great white shark upon a SharkPOD-equipped diver in 2002 has been attributed to the device not being used in accordance with the manufacturer's specifications. Regardless of assignment of fault, it emphasizes that no device should be assumed 100% effective in all circumstances. The reader is advised to check on the availability and status of warranties and representations prior to committing to any particular purchase.

BARRACUDA

To many divers, the barracuda appears more sinister than the shark and is more highly feared. Barracuda are distributed from Brazil north to Florida, and in the Indo-Pacific from the Red Sea to the Hawaiian Islands. Of the 22 species of barracuda, only the great barracuda *(Sphyraena barracuda)* has been implicated in human attacks. Smaller species of barracuda may be found in large schools.

Life and Habits

The great barracuda is encountered in all tropical seas and is reputed to grow to 2.5 m (8.2 ft) and 50 kg (110 lb) but is rarely sighted at a length greater than 1.5 m (5 ft) (Fig. 72-42). The world record weight of a captured barracuda is approximately 38.6 kg (85 lb). A solitary swimmer, the fish is extremely swift and swims in a disconcerting darting fashion. The barracuda possesses an elongated narrow mouth filled with large knifelike teeth, similar in appearance to those of canines (Fig. 72-43).

Although great barracuda seldom attack divers, they do so rapidly and fiercely, often out of confusion in murky waters. More commonly the fish charges through or leaps from shallow water to bite the dangling legs of a boater, particularly if a shiny anklet (which resembles a fishing lure) is worn. Persons have been bitten on the scalp while wearing a barrette or on the face when trying to feed a barracuda by holding a dead fish bait in their mouths. Wounds to the hand are commonly incurred during removal of hooks. Barracudas normally feed with sudden and rapid bursts of speed. They often slice a natural prey item in two with almost surgical precision as they literally race through the prey. Amputations of fingers and hands at the wrist may occur if victims are wearing jewelry, carrying speared fishes, or are cleaning hands in the water from a boat or dock. In a recent case of an unprovoked attack, a snorkeler was severely bitten on the hand, creating a partial amputation. Considering the great frequency with which barracuda are encountered and the low number of reported attacks, they do not pose nearly the hazard of sharks.

Figure 72-42. Great barracuda *(Sphyraena barracuda)*. (Photo by Paul S. Auerbach, MD.)

Figure 72-43. Jaws of the great barracuda, with canine-configured teeth next to the author's (PSA) hand.

Clinical Aspects

Barracuda jaws contain two nearly parallel rows of teeth, which produce straight or V-shaped lacerations, in contradistinction to the crescent-shaped bite of the shark. Except for this difference and the magnitude of injury, the surgical problems generated by the barracuda do not differ from those of the shark. The clinician encounters tissue loss, moderate hemorrhage, and wound infections.

Treatment

Barracuda bites are treated identically to shark bites. If a barracuda is captured, it should not be eaten in ciguatera toxin-endemic regions.

Figure 72-44. California moray eel *(Gymnothorax mordax)*. (Photo by Howard Hall.)

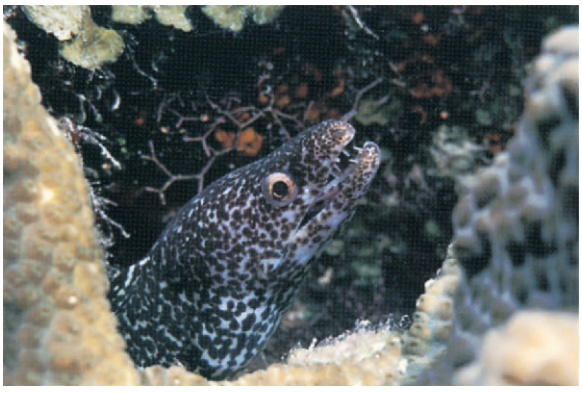


Figure 72-45. Moray eel, demonstrating the sharp teeth and biting potential. (Photo by Paul S. Auerbach, MD.)

Prevention

Barracuda are attracted to turbid waters, underwater commotion, irregular motion, surface splashing, shiny objects, and speared or tethered fish. These should all be avoided. It is unwise to dangle a body part adorned with reflective jewelry before the jaws of a barracuda. In general, divers should avoid wearing rings, exposed watches (the glass or plastic face plate is attractive when reflecting light), necklaces, earrings and bracelets, as these are contributors to attacks by both barracudas and sharks.

MORAY EELS

Life and Habits

Moray eels are found in tropical, subtropical, and some temperate waters. Members of the order Anguilliformes, family Muraenidae, some individuals of the larger species may attain lengths of 3.5 m, diameters of more than 35 cm and weights in excess of 34 kg (75 lb). Morays are muscular and powerful bottom dwellers, residing in holes or crevices or under rock and coral (Fig. 72-44). They have a snakelike appearance and usually lack scales or pectoral fins. The distinguishing feature of the morays is the small, round gill opening. The skin of moray eels is rubbery to leathery and mucus (to protect from infection) coated. They typically have poor eyesight, and rely on their sense of smell to locate prey. Moray eels exhibit three different types of reproductive ability: gonorchist (dedicated male or female, without the ability to change sex), simultaneous hermaphrodite (both sex organs, able to mate with either sex), or protogynous hermaphrodite (can be born as a female or male; female can change to male if need be).

Fortunately, eels usually evade confrontation unless cornered or provoked. Bites occur when a diver intentionally probes into a coral bed or cave, or a fisher reaches into a net and offers a hand to an aggravated eel. Aquarium-housed morays may strike when handled improperly. Most moray eels are easily intimidated. However, an aggressive eel may strike out in competition for prey. Elderly, vision-impaired eels or seemingly docile animals may attack without specific provocation, especially at night. In U.S. coastal waters, the most often observed species are the California, green (which has a third row of teeth, presumably to help hold prey), and spotted morays.

Clinical Aspects

Morays are forceful and vicious biters that can inflict severe puncture wounds with their narrow and viselike jaws, which are equipped with long, sharp, retrorse, and fanglike teeth (Fig. 72-45). Normal eel prey includes fish, crustaceans, octopi and other small marine animals. Molar-type teeth are present in some species; these species typically consume crustaceans. A moray eel has the tenacity of a bulldog and will hold on to a victim, rather than strike and release. Multiple small puncture wounds are common after the bite of smaller eels, with the hand most commonly involved. Large avulsion injuries may occur (Fig. 72-46).[166] If the eel is ripped forcefully from the victim, the resulting lacerations may be even more extensive.

Treatment

Moray bites are treated in a manner analogous to that of shark bites. If the eel remains attached to the victim, the jaws may need to be broken or the animal decapitated to effect release. The primary wound should be irrigated copiously and explored to locate any retained teeth. The risk of infection is high, particularly in bites to the hand, as the symbiotic flora associated with the moray's oral cavity is apparently unusually rich. Uncomplicated small puncture wounds generated by individual teeth should be left unsutured to allow drainage and the victim given appropriate prophylactic antibiotics. If the wound is extensive and more linear in configuration (resembling a dog bite), the wound edges may be debrided and loosely approximated with nonabsorbable sutures or staples, in which case antibiotics should be administered. If the wound is severe, vital structures should be assessed in routine operative fashion. In all cases, it is prudent to inspect the wound at 24 and 48 hours to detect the onset of infection. If not appropriately treated initially, infections may persist for months, even years.

Prevention

It is unwise for a snorkeler or diver to place a hand underneath unexplored coral or rock unless it has been probed or otherwise disturbed specifically in search of an eel. All fishing nets should be handled carefully. A dive guide who feeds moray eels (Fig. 72-47) by holding a loaf of bread or bait fish in his mouth

Figure 72-46. Moray eel bite. (Photo by Marty Snyderman.)

Figure 72-47. Diver handling large moray eel. (Photo by Marty Snyderman.)

is foolishly offering his nose and mouth as a target for an unpredictable eel.

GIANT GROUPERS

Some of the larger species of sea basses or groupers (family Serranidae) may grow to exceed 3.6 m (11.8 ft) and 227 kg (500 lb) (Fig. 72-48). Distributed in both tropical and temperate seas, they are curious, occasionally pugnacious, and voracious feeders. Although not aggressive like a shark, a giant grouper

Figure 72-48. Giant grouper or potato cod, Great Barrier Reef, Australia. (© 2000 Norbert Wu, www.norbertwu.com)

should be respected for its fearlessness, bulk, and cavernous mouth. Groupers can be found frequenting shipwrecks; swimming in caves, caverns, and holes; and lurking behind large rocks and coral outcroppings. They are territorial and may become aggressive while protecting their domain. Bite wounds may be ragged with extensive maceration and are treated the same as shark bites. Large groupers should not be eaten in ciguatera toxin–endemic regions. It is always wise to visually survey an underwater cave before entering or exiting. The diver should not block the exit if a grouper is attempting to escape and should not carry speared fish. Many scare tactics used against sharks are of no avail with groupers.

SEA LIONS

Sea lions (family Otaridae) and seals (family Phocidae) are mild-mannered mammals except during the mating season, when the males may become aggressive, and during the breeding season, when both genders attack in defense of their newborn pups (Fig. 72-49). Divers have been seriously bitten and therefore should avoid ill-tempered and abnormally aggressive animals. There is nothing unique about the clinical aspects of these injuries, except for the post-traumatic infections. The bites are treated the same as shark bites.

"Seal finger" (spekk finger, blubber finger) (see Chapter 75) follows a bite wound from a seal or from contact of even a minor skin wound with a seal's mouth or pelt. It has traditionally been an occupational hazard of seal hunters, but has now been noted in persons trying to save seals and aquarium workers. One case was attributed to a polar bear (which may or may not have eaten a seal) bite. The affliction is characterized by an incubation period of 1 to 15 (typically, 4) days, followed by painful swelling of the digit, with or without destructive articular involvement. Severe pain may precede the appearance of the initial furuncle, stiffness, or swelling. As the lesion worsens, the skin becomes taut and shiny, and the entire hand may swell and take on a brownish violet hue. It is quite possible to have involvement of adjacent fingers. Tenosynovitis and arthritis have been noted, which may progress to joint destruction and arthrosis. There may be painless, nonsuppura-

Figure 72-49. Sea lion and pup. Parents of both genders aggressively protect their young. (Photo by Paul S. Auerbach, MD.)

Figure 72-50. Saltwater crocodile, Queensland, Australia. (© 2000 Norbert Wu, www.norbertwu.com)

tive lymphadenopathy. It is common for the affliction to run a protracted course.[58] Current thinking focuses on *Mycoplasma* species (such as *phocidae* or *phocacerebrale*) as the inciting pathogens, which is consistent with their cytopathogenic potential.[14] Therefore, microbiologic evaluation of a lesion should include culture for mycoplasmas in addition to standard aerobes and anaerobes. Infection with *Erysipelothrix rhusiopathiae* is in the differential diagnosis, but usually is characterized by a more erythematous and bordered rash spread among multiple fingers.

Tetracycline 1.5 g initially, followed by 500 mg four times a day for 4 to 6 weeks is the recommended therapy.[29] β-Lactam antibiotics, cephalosporins, and erythromycin are not efficacious. Fluoroquinolone or macrolide antibiotics may be useful if tetracycline is not available. Early (in the first week) incision to relieve elevated tissue pressure may be efficacious, but delayed joint debridement has not proven useful. Early antibiotic therapy is key to successful treatment. Preventive measures include wearing gloves and washing all wounds vigorously with soap and water, perhaps followed by an isopropyl alcohol rinse.

CROCODILES AND ALLIGATORS

Crocodiles (genus *Crocodylus*) and alligators (genus *Alligator*) are long-bodied loricates with reputations as ferocious aquatic reptiles. Considered to be less sluggish than alligators, crocodiles (*C. niloticus* in the Nile and the larger estuarine *C. porosus* [Fig. 72-50] of eastern Asia and the Pacific islands) may attack and severely injure a human. *C. porosus*, which ranges over an extensive geographic area, including India, Sri Lanka, southern China, the Malay Archipelago, Palau, the Solomon Islands, and northern Australia, has been claimed to be a prolific man-eater. Estimates of human fatalities may be exaggerated based on isolated reports of atrocities committed by this beast. An adult crocodile devours prey much larger in size than a human. According to one report, the stomach of an Australian estuarine crocodile contained the remains of an aborigine and a 4-gallon drum containing two blankets. At a length greater than 20 ft (6 m) and a weight exceeding 2500 lb (1,136 kg), the crocodile can travel in water at a speed of 20 mph (32 km/h) and can charge a short distance over land at 15 to 30 mph (24 to 48 km/h). The enormous jaws and canine teeth (Fig. 72-51) can bite with sufficient force to sever an outboard boat propeller. However, the teeth are not well suited for tearing apart or chewing, so most prey is killed and allowed to rot, which makes them easier to swallow. Most crocodiles are content to eat fish, turtles, kangaroos, and wild pigs. However, feeding in freshwater rivers and adjacent land has introduced them to cows, horses, and humans, who are attacked when they cross rivers, catch fish, draw water, or work in the fields.[144] The majority of attacks occur at twilight or at night. A crocodile attacks by grasping its prey in its powerful jaws and dragging it underwater, where it drowns and dismembers it with a constant twirling motion ("death rolls"). In a series of 16 crocodile attacks reported from the Northern Territory of Australia, four fatalities involved transection of the torso or decapitation.[90] Most attacks (fatal and nonfatal) were on persons swimming or wading in shallow water. In riverine areas of Tanzania, crocodiles are a considerable health hazard. In the Korogwe District from 1990 to 1994, 51 human and 49 crocodile deaths were reported. The attacks are attributed to increased waste products in the water, which reduces the crocodiles' primary food

Figure 72-51. Saltwater (or estuarine) crocodile *(Crocodylus porosus)* covered in duck weed, North Queensland, Australia. (© Gary Bell/oceanwideimages.com)

Figure 72-52. Needlefish in mangrove swamp, Solomon Islands. (© 2000 Norbert Wu, www.norbertwu.com)

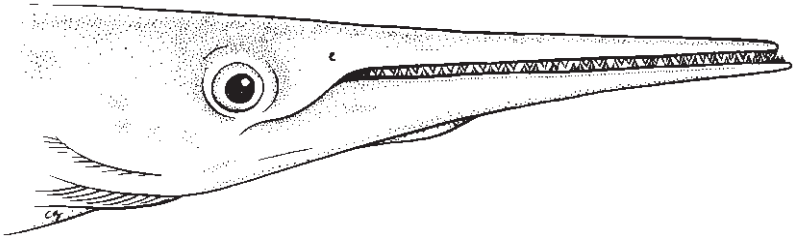


Figure 72-53. Needlefish beak, capable of causing a penetrating injury.

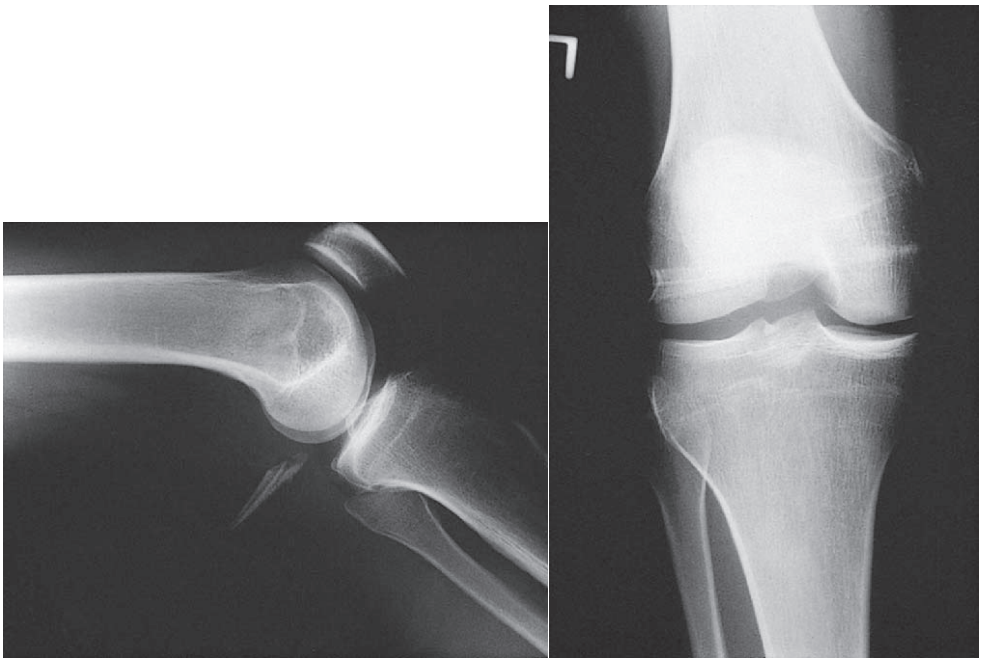
supply. In addition, "tamed" crocodiles are not hunted because of local superstitions and fear of reprisal by witchcraft.[117]

The American alligator *Alligator mississippiensis* most commonly attacks in the water but will also attack on land. These attacks seem to be motivated by feeding. Most alligator attacks in the U.S. seem to be on swimmers, waders, or fishermen. Alligators residing in lakes and ponds associated with golf courses, parks, and tourist attractions have attacked humans in the United States.[111] During the period 1948–2003, there were 326 alligator attacks resulting in 13 deaths in Florida, a fatality rate of 4%; by comparison, 459 Florida shark attacks with eight fatalities produced a 1.7% mortality rate during the same time frame (ISAF analyses). The American crocodile *Crocodylus acutus* inhabits the southern tip of Florida, Cuba, Hispaniola, Jamaica, Central America, and northern and northwestern South America. Attacks by this species are more infrequent than those by the American alligator.

Crocodile bites produce large crush injuries and lacerations. Surgical wound management is similar to that for dog bites. The bacteriologic considerations have not been extensively studied but should be assumed to be the same as for other animals that reside in the aquatic environment. Wound infections should be anticipated in the largely contaminated extensive crush injuries. *Aeromonas hydrophila* is a common inhabitant of eutropic (more organic matter is being made during photosynthesis than is being consumed) lakes and ponds. Prophylactic antibiotics to cover *Aeromonas hydrophila*, *Pseudomonas*, and *Vibrio* should be administered. Coverage against anaerobes, such as *Bacteroides*, is prudent. Antitetanus primary or booster immunization is mandatory. Residual deformities are common and it is hope amenable to surgical reconstruction.[66,144] When approached by an alligator, it is important to prevent the animal from opening its mouth, as the muscles elevating the jaw are relatively weak. It is also important to avoid the powerful, lashing tail.[111]

NEEDLEFISH

Marine needlefish (family Belonidae) are slender, tubular, elongate, silver, and lightning-quick surface swimmers found in temperate and tropical seas (Fig. 72-52). They resemble, but are not related to, the freshwater gar and may attain streamlined lengths of up to 2 m (6.6 ft). Possessed of an elongated pointed snout, which comprises one-quarter the length of the fish and contains small pointed teeth (Fig. 72-53), the fish moves rapidly, often leaping out of the water in fear or when attracted to lights or windsurfers. It has been hypothesized that a needlefish can exit the water at a speed of approximately 40 mph (64 km/hr), which approximates the speed of a flyingfish. The needlefish, or garfish, is an occupational hazard for persons who fish from small canoes at night in tropical Indo-Pacific ocean waters.[13] On occasion they have flown into people, spearing them in the chest, abdomen, extremities, head, and neck. In one reported case, a fish caused brain injury by creating an internal carotid-cavernous sinus fistula after orbitocranial perforation.[88] In



A B

Figure 72-54. Sailfish bill lodged in the posterior knee. **A,** Lateral view. **B,** Anterior view.

another case, the calcified elongated jaws of a needlefish embedded in a woman's neck were retained for over a month before removal.[21] In another, penetration of the knee by *Tylosurus crocodilus* ("little sea crocodile") occurred in an ocean surface swimmer in New Caldedonia.[74] Exsanguination from a neck wound has been anecdotally reported from Papua New Guinea. A penetrating leg injury from a needlefish, with major vascular injury (popliteal vein, anterior tibial and peroneal arteries), occurred in the leg of a windsurfer off the coast of the Outer Banks of North Carolina.[81] A chest wound can be accompanied by a pneumothorax. Death may occur from chest or abdominal penetration. The depth of penetration of the animal may be augmented by the speed at which the victim approaches the piscine projectile. Treatment is according to the nature of the injury. All wounds should be debrided and irrigated, followed by a search for foreign material. Radiographs appropriate to identify foreign bodies should be obtained. Two semiparallel lines of opacity representing the jaws of the fish are pathognomic of a needlefish beak.[68,74,81] A small superficial wound may cause the physician to underestimate an internal injury. The major risk is wound infection, attributed to bacteria in the carnivorous fish teeth. Injury prevention is difficult, although it has been suggested that canoes be positioned in a circle to allow spearing of fish in a central pool of light. Flyingfish pose less risk, because they have blunt heads.

Many other fish leap from the water, but injuries are extremely uncommon. A case of a wahoo (150 cm; 22.5 kg [5 ft; 50 lb]), family Scombridae, leaping from the water and biting a victim on the upper extremity has been reported.[60] The sharp teeth generated extensor tendon lacerations on the dorsal hand and forearm that required surgical repair. A careless fisherman can easily be cut by a wahoo (and other large mackerels of the genus *Scomberomorus*, including the king mackerel, *Scomberomorus cavalla*) when extracting a fishing lure. As previously described, barracuda frequently exit the water in pursuit of shiny metallic objects that resemble fishing lures as well as when chasing baitfishes. Bluefish *(Pomatomus saltatrix)* school and feed in a frenzy, but most bites occur as the fish are handled out of the water; in-water attacks on humans are largely theoretical.[75] During a World War II beach landing along the north African coast, bluefishes were reported to attack wading soldiers. The fish has sharp, conical canine teeth in both the upper and lower jaws, is quick to bite while on deck (earning the nickname "choppers") and can grow to 1.2 m (4 ft) and over 12 kg (26.4 lb).

The sailfish, which can approach speeds in the water of 70 mph (113 km/hr) in short bursts, sports an elongated bill. Although not considered a predator, a sailfish has on at least a few occasions driven its bill into a human victim (Fig. 72-54), in one case causing a colon perforation in a snorkeler. In another case, a young man had the left upper lobe of his lung pierced by the tip of a swordfish, requiring left upper lobe segmentectomy.[32]

KILLER WHALES

The killer whale, *Orcinus orca,* is probably not a ferocious killer of humans. The largest of the living mammalian dolphins, these magnificent animals (Fig. 72-55) grow to 33 ft (10 m) and 10 tons (9090 kg) and are found in all oceans. They usually travel in pods of up to 40 individuals. Swift and enormously powerful creatures, they feed on squid, fish, birds, seals, walruses, and other whales. Their powerful jaws are equipped with cone-shaped teeth directed back into the throat, designed to grasp

and hold food. The killer whale can generate enough crushing power to bite a seal or porpoise in two with a single snap.

In captivity, killer whales are playful creatures and seem intelligent, without the primal behavior of sharks. Nonetheless, although killer whales are believed not to prey on humans, they should be regarded with respect and kept at a distance in their natural habitat. Mistaken for a sea lion, a human would be a nice snack for a killer whale. In a highly publicized event, a captive killer whale appeared to pursue its handler, attempting to cause injury by repetitive battering.

Figure 72-55. Distinctive markings of a killer whale near the San Juan Islands, Washington State. (© 2000 Norbert Wu, www.norbertwu.com)

Other whale species, such as the finback, have rammed boats, theoretically in defense of their young. Territorial behavior should be anticipated and respected.

GIANT CLAMS

Although many adventure stories describe divers being caught in the clamp of a giant clam (family Tridacnidae), there are no verifiable reports of such a calamity resulting in a major injury. *Tridacna gigas* can attain a length of 1 m (3.3 ft) (Fig. 72-56) and weigh as much as 300 kg (660 lb). The hazard to divers is hypothetical.

COLOSSAL SQUID

The giant ("colossal") cephalopod (10 arms) squid, possibly *Mesonychoteuthis hamiltoni,* grows to a length in excess of 57 ft (17.4 m) and weight of 2000 lb (909 kg), with long (10 m [32.8 ft]) menacing tentacles, eyes with a diameter of nearly 35 cm (13.7 inches, the size of a dinner plate), and a razor-sharp beak that it uses to eat prey. *Architeuthis dux,* the "giant squid," may have a mantle that does not exceed 2.25 m (7.4 ft). The tentacles are armed with chitinous serrated rings equipped with teeth on each of the suckers. The suckers are approximately 1.5 inches (4 cm) in diameter. The giant Humboldt squid (Figs. 72-57 through 72-59) demonstrates typical giant squid features. Sperm whales have been examined with sucker wounds with a diameter of 18 inches (46 cm), which would extrapolate to a truly monstrous squid, estimated at 60 m (197 ft) in length. However, sucker scars expand in size as a whale grows, casting doubt on these projections. The battles between sperm whales and colossal squid are legendary, but humans are unlikely to

A B

Figure 72-56. Giant clams. **A,** Giant clam with diver. **B,** Giant clam mantle (*Tridacna* sp.) obtains its coloration from algae utilized for photosynthesis. (**A,** Photo by Howard Hall. **B,** © 2000 Norbert Wu, www.norbertwu.com)

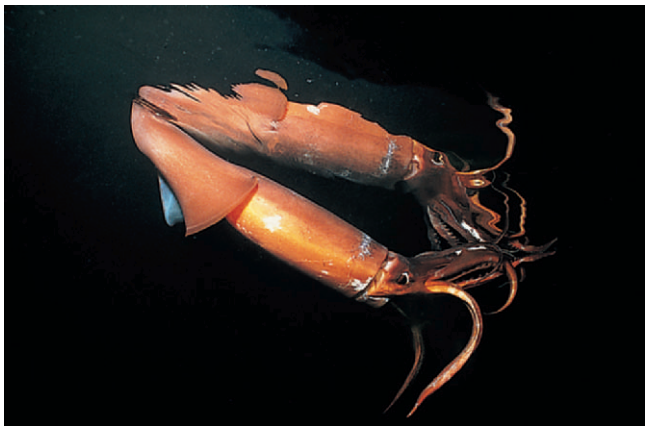


Figure 72-57. Giant Humboldt squid *(Dosidicus gigas)* attains a length of 15 feet and weight of 50 pounds. It is a voracious carnivore. (© 2000 Norbert Wu, www.norbertwu.com)

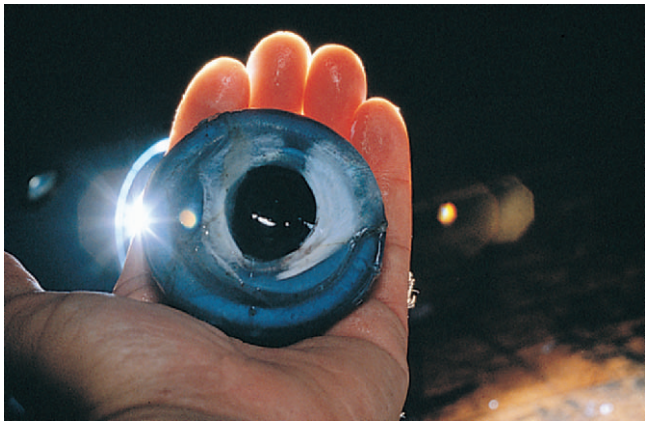


Figure 72-58. Eyeball of a giant Humboldt squid cut up by local fishermen. (© 2000 Norbert Wu, www.norbertwu.com)

Figure 72-59. Suckers of the giant Humboldt squid are lined with razor-sharp teeth. (© 2000 Norbert Wu, www.norbertwu.com)

Figure 72-60. Giant manta ray. The sting on the caudal appendage is vestigial, but the "wings" can slap and abrade the unwary individual. (Photo by Paul S. Auerbach, MD.)

encounter this awesome animal, which is found at depths far beyond the range of a sport scuba diver.[45] With increased deep sea exploration by small submersibles, we may learn more about this fascinating creature. It is possible that a hungry colossal squid might ingest a human, but this has not yet been observed and is not likely to occur.

GIANT OCTOPUS

The Pacific giant octopus *Octopus dofleini* is a predator that has been captured at 272 kg (598 lb) with an arm span of over 30 ft (9.1 m). It ranges off the western North American coast from northern California to Alaska and off eastern Asia southward to Japan. The cephalopod is armed with suckers on eight arms and a parrotlike chitinous mouth located centrally underneath the head. Although it exhibits curiosity, it does not exhibit aggression directed against humans. However, it possesses the strength and agility to easily overwhelm a human. The animals have been reported to remove a dive mask or pull a regulator from divers. In open water, it is capable of squirting a large cloud of ink, which it sheds as evasive strategy. Folklore from the South Pacific tell of native breath-hold divers being subdued and drowned by angered captive octopuses.

GIANT MANTA RAY

The giant manta ray *Manta birostris* can have a wingspan of more than 6 m (19.7 ft) and a weight of 1600 kg (3520 lb) (Fig. 72-60). The caudal appendage carries a vestigial stinger that poses no threat to humans. However, the coarse dermal denticles can create severe abrasions, which generally occur when divers attempt to ride these gentle and accommodating creatures.

MANTIS SHRIMP

The mantis shrimp *(Gonodactylus bredini, Hemisquilla ensigera californiensis)* is not a true shrimp but resembles a large flattened shrimp or miniature lobster (up to 36 cm [14.2 inches]) and is equipped with a pair of legs that serve as specialized jack-

Figure 72-61. Mantis shrimp, ready to strike with its claws. (© 2000 Norbert Wu, www.norbertwu.com)

Figure 72-62. Teeth of the piranha. (Photo by V. Haddad, Jr.)

Figure 72-63. Piranha. (© 2000 Norbert Wu, www.norbertwu.com)

knife claws (Fig. 72-61). The tail carries numerous sharp spines that may project beyond the edge of the sturdy tail fin. Lacerations may be induced by either the front raptorial (prey-acquiring) claws or the tail, particularly when the mantis shrimp attacks an unwary victim. The strike from the paired claws may be completed in a few milliseconds. It has been claimed that an attacking mantis shrimp struck with enough force to crack a diver's face mask. In the Caribbean, the stomatopod ("foot-mouth") mantis shrimp is known as "thumb splitter." *Odontodactylus scyllarus* from the Indo-Pacific can be afflicted with a disease that digests areas of its dorsal cuticle and eventually is lethal. This may explain one anecdotal report of a human finger wound (which led to amputation) characterized by cartilage destruction and from which no pathogenic organism could be cultured. The mantis shrimp is a superb predator, in part because it has the most highly developed eyes of any crustacean.

PIRANHA

South American freshwater characins include the piranha *Serrasalmus nattereri,* equipped with a formidable set of razor-sharp teeth (Fig. 72-62). They are attracted by blood or commotion. Piranhas are widespread in rivers and lakes, and number approximately 30 species of the genera *Pygocentrus* and *Serrasalmus*.[162] These small fish (Fig. 72-63) may attack in schools of several hundred, although this reputation is largely borne of folklore rather than of documentation. Its reputation as an attacker of humans, like that of the barracuda, is greatly overstated. Although an overwhelmed human could theoretically be reduced to a shiny skeleton in short order, most attacks on humans are caused by a single fish biting only once, resulting in a single, circular, crater-like wound (Fig. 72-64). Bathers are injured most often in dammed waters because of fish proliferation, spawning, and parental-care behavior.[163,164,165] In one series of attacks by speckled piranhas *(Serrasalmus spilopleura),* it was noted that most bites occurred on the lower extremities, particularly on the heel. One bite was sufficient to amputate a toe.[162] Prevention measures might include clearance of waterweeds at bathing sites or placement of net enclosures around bathing areas. Other characid freshwater fish with fearsome teeth include the South American dogfish (Fig. 72-65). Natives living near piranha-inhabited waters express much more concern over freshwater stingrays (genus *Potamotrygon*) than about piranhas.

SNAPPING TURTLE

Snapping turtles (family Chelydridae) may bite humans when they are provoked on land; bites are not initiated while the animals are in the water. The biting speed is quite rapid and powerful, as evidenced by the aggression of the common snapper *Chelydra serpentina*. The larger alligator snapper *Macroclemys temminckii* is less aggressive.[26] The Florida snapping turtle *Chelydra osceola* is found only in Florida.

TRIGGERFISH

Triggerfish may be gregarious or unimposing, but during mating season the females of at least two species (*Pseudobalistes fuscus* and the larger *Balistoides viridescens*) can become extremely territorial in guarding their nests and thus aggressive, inflicting painful bites. The former can grow to 55 cm (22 inches) and the

A

B

Figure 72-64. Crateriform bite wounds **(A** and **B)** caused by piranhas. (Photos by V. Haddad, Jr.)

latter to 75 cm (30 inches). The strong jaws each carry eight long, protruding, and chisel-like teeth (Fig. 72-66) in an outer row, backed by an inner row of six teeth.[71] Usually the fish "bites and runs," but the orange-striped triggerfish *Balistoides undulatus* has been reported to bite and not release. It is common to have to strike the fish in some manner to get it to release. In the Gilbert Islands, a release technique is to bite the fish on the top of the head.

STONY CORALS

Life and Habits

The anthozoan Madreporaria, or true (stony) corals, exist in colonies that possess calcareous outer skeletons (the origin of

Figure 72-65. South American freshwater characid dogfish. (Photo by V. Haddad, Jr.)

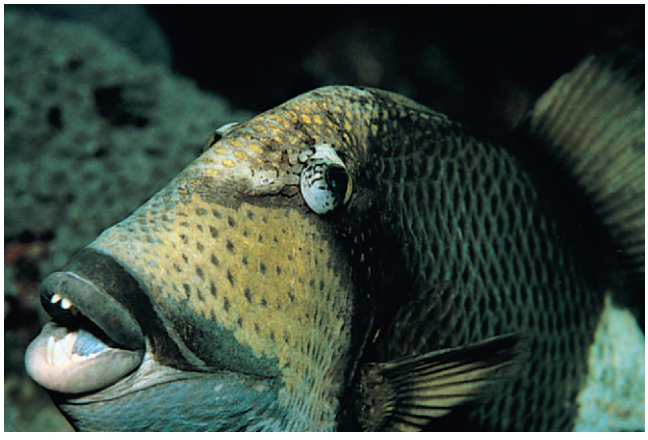


Figure 72-66. Triggerfish. (Photo by John Randall.)

calcium carbonate, or limestone) with pointed horns, razor-sharp edges, or both (Fig. 72-67). There are nearly 1000 species of corals. Corals live in waters at temperatures of 20° C (68° F) or higher, generally at depths of up to 20 fathoms (120 ft), although they are seen at depths of up to 83 fathoms (500 ft). A "coral head" is actually a colony of individual polyps. Certain coral species, such as *Plexaura homomalla,* have been investigated as sources of prostaglandins and other pharmaceutical precursors to treat conditions as diverse as asthma, leukemia, and infections. Pieces of coral have been evaluated for use as bone grafts.

Coral reefs are under pressure worldwide from climatic changes (e.g., El Niño), chemical poisons (e.g., cyanide used for fishing), natural predators (e.g., crown-of-thorns sea star), and mechanical destruction (e.g., ship anchors, diver contact, and explosives).

Clinical Aspects

Snorkelers and divers, particularly photographers and spear fishermen, frequently handle or brush against these living reefs, resulting in superficial cuts and abrasions on the extremities

Figure 72-67. Coral garden. (Photo by Paul S. Auerbach, MD.)

Figure 72-68. Abrasions of the leg from bumping against sharp coral. (Photo by Paul S. Auerbach, MD.)

(Fig. 72-68) while simultaneously injuring the corals. Coral cuts are probably the most common injuries sustained underwater. The initial reaction to a coral cut is stinging pain, erythema, and pruritus, most commonly on the forearms, elbows, and knees. Divers without gloves frequently receive cuts to the hands. A break in the skin may be surrounded within minutes by an erythematous wheal, which fades over 1 to 2 hours. The red, raised welts and local pruritus are called coral poisoning. Low-grade fever may be present and does not necessarily indicate an infection. Blistering may occur. With or without prompt treatment, the wound may progress to cellulitis with ulceration and tissue sloughing. These wounds heal slowly (3 to 6 weeks) and result in prolonged morbidity. There may be a stage of subacute fleshy granulomatous dermatitis, followed by chronic lichenoid dermatitis, in which the lesions harden, become smaller, and take on a shiny, lichenoid appearance.[42] In an extreme case the victim develops cellulitis with lymphangitis, reactive bursitis, local ulceration, and wound necrosis. Chronic dermal granulomata followed a coral scrape or cut should invoke suspicion for *Mycobacterium* infection, including species *marinum* or *haemophilum*.[125]

Treatment

Coral cuts should be promptly and vigorously scrubbed with soap and water, then irrigated copiously with a forceful stream of freshwater or normal saline to remove all foreign particles. Using medicinal hydrogen peroxide to bubble out "coral dust" is occasionally helpful. Any fragments that remain can become embedded and increase the risk for an indolent infection or foreign body granuloma. If stinging is a major symptom, there may be an element of envenomation by nematocysts (see Chapter 73). A brief rinse with diluted acetic acid (vinegar), papain solution, or isopropyl alcohol 20% may diminish the discomfort (after the initial pain from contact with the open wound). Topical decontamination should be followed by a normal saline or tap water rinse. If a coral-induced laceration is severe, it should be closed with adhesive strips rather than sutures if possible; preferably it should be debrided for 3 to 4 consecutive days and closed in a delayed fashion.

A number of approaches can be taken with regard to subsequent wound care. One method is to apply twice-daily sterile wet-to-dry dressings, using saline or a dilute antiseptic (povidone-iodine 1% to 5%) solution. Alternatively, a nontoxic topical antiseptic or antibiotic ointment (mupirocin, bacitracin, or polymyxin B–bacitracin–neomycin) may be used sparingly and covered with a nonadherent dressing (e.g., Telfa). Secondary infections are dealt with as they arise. A final approach is to apply a full-strength antiseptic solution, followed by a powdered topical antibiotic, such as tetracycline. An alternative medicine technique that has been reported to the author (PSA) to be effective is to apply Tibetan red flower seed oil 4 times a day, noting that the application will cause burning pain for a short period of time. No method has been supported by any prospective trial.

Despite the best efforts at primary irrigation and decontamination, the wound may heal slowly, with moderate-to-severe soft tissue inflammation and ulcer formation (Fig. 72-69). All devitalized tissue should be debrided regularly using sharp dissection. This should be continued until a bed of healthy granulation tissue is formed. Wounds that appear infected should be cultured and treated with antibiotics as previously discussed. Lichenoid papules, which may be flat or dome-shaped, may respond to treatment with betamethasone dipropionate 0.05% cream applied twice daily for 2 weeks under occlusive dressings.[42] Residual postinflammatory hyperpigmentation is possible.

The victim who demonstrates malaise, nausea, and low-grade fever may have a systemic form of coral poisoning or be manifesting early signs of a wound infection. It is prudent at this point to search for a localized infection, procure wound cultures or biopsy specimens as indicated, and initiate antibiotics pending confirmation of organisms. If the victim is started on an antibiotic and does not improve, a supplemental trial of a systemic glucocorticoid (prednisone 80 mg tapered over 2 weeks) is not unreasonable. In the absence of an overt infection, the natural course of the wound is to improve spontaneously over a 4- to 15-week period.

Prevention

Divers exploring near coral reefs must take every care to avoid coral cuts. Protective clothing and gloves should be impenetrable. Snorkelers and underwater photographers in shallow water should wear adequate hand, elbow, and knee protection.

Figure 72-69. Poorly healing wound following coral cut. (Photo by Paul S. Auerbach, MD.)

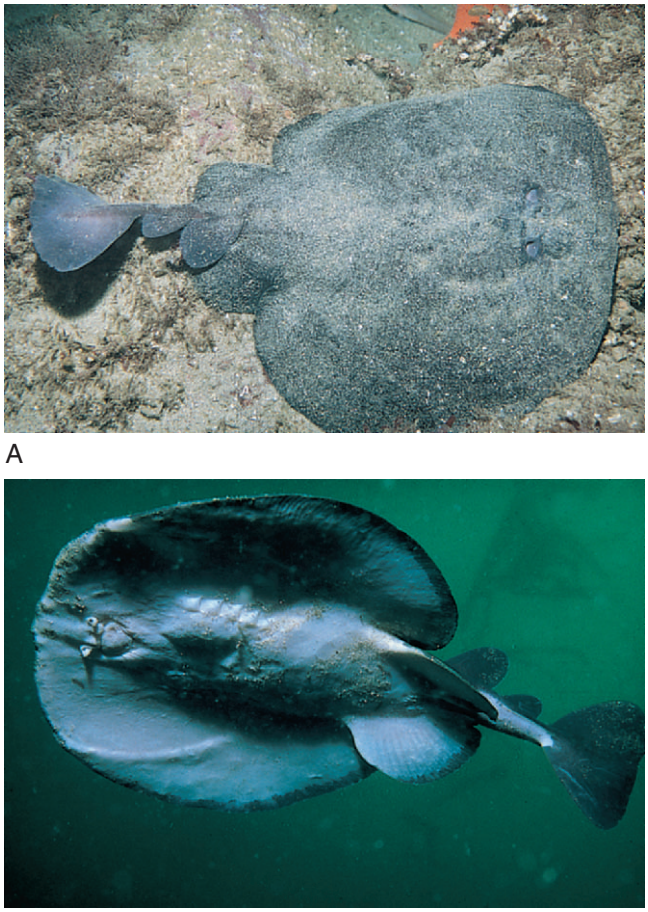
Figure 72-70. Stargazer *(Astroscopus zephyreus)* with electric plates above each eye. (© 2000 Norbert Wu, www.norbertwu.com)

SHOCKING MARINE AND FRESHWATER ANIMALS

Only two groups of electric fish are marine; the remainder are freshwater animals. They rarely pose a health hazard but rather are curious creatures surrounded by superstition and folklore. The marine electric fish include the stargazers *(Astroscopus)* (Fig. 72-70) and the electric rays (*Torpedo* and *Narcine*). The electric eel (Fig. 72-71) is a freshwater Amazonian animal.

Electric rays are found in temperate and tropical oceans. Of the class Chondrichthyes, they are round-bodied, with short tails and thick bodies (compared with stingrays). In California, *Torpedo californica* (Fig. 72-72) attains a length of 4 ft (1.2 m)

Figure 72-71. Electric eel, Steinhart Aquarium. (© 2000 Norbert Wu, www.norbertwu.com)



A

B

Figure 72-72. Electric ray *(Torpedo californica)*. A, Dorsal view. B, Ventral view. (© 2000 Norbert Wu, www.norbertwu.com)

and weight of 80 to 90 lb (36–41 kg). It swims slowly and sluggishly and is usually found partially buried in bottom mud and sand. Well camouflaged, its dorsal surface is multicolored and the ventral surface creamy white. The externally visible electric organs are located on each side of the anterior part of the disc between the anterior extension of the pectoral fin and the head, extending from above the level of the eye backward past the gill region onto the ventral surface. The electric organs are composed of a honeycomb network of modified muscles organized into columnar prismlike structures and connective tissue, which generate an electrical charge by neuromuscular activity. The muscle cells *(electroplaques)* are stacked 500 to 1000 deep, creating up to 500 cm^2 of surface area. The electroplaques depolarize in series and in parallel simultaneously, producing amperage sufficient to stun prey. Species in the tropical eastern Pacific include the smaller-bodied lesser ray, *Narcine entemedor,* and the bulls-eye ray *Diplobatis ommata.*

Generally the ventral surface of the ray is negative and the dorsal side is positive. An electrical discharge is reflexively produced on contact, often in a series exhaustive for the fish. This necessitates a period of recharging. Electricity is delivered in doses of 8 to 220 volts. The Atlantic *Torpedo nobiliana* produces 180 to 220 volts. Although the shock is of low amperage, it is sufficient to stun a grown man and might induce drowning. Recovery from the shock has been reported anecdotally to usually be uneventful. An electric ray should not be handled. The energy generated by skates is considerably less, measured in millivolts to 1 to 2 volts.

The references for this chapter can be found on the accompanying DVD-ROM.

Envenomation by Aquatic Invertebrates

73

Paul S. Auerbach

Stinging aquatic animals pose a hazard for swimmers and divers. They constitute a large collection of marine organisms that include invertebrates and vertebrates, and that range from primitive to extremely sophisticated organisms. This chapter discusses envenomation by aquatic invertebrate life-forms. Chapter 74 discusses envenomation by aquatic vertebrate life-forms. Chapter 72 discusses infections associated with aquatic wounds and the relevant antimicrobial therapies. Standard wound care measures, such as antitetanus immunization, should be undertaken whenever there is penetration of the skin.

The science of poisons, biotoxicology, is divided into plant poisons, or phytotoxicology, and animal poisons, or zootoxicology. *Toxinology* connotes the science of toxic substances produced by or accumulated in living organisms, their properties, and their biologic significance for the organisms involved.[114] Animals in which a definite venom apparatus is present are sometimes called phanerotoxic, whereas animals whose body tissues are toxic are termed cryptotoxic.[141] Naturally occurring aquatic zootoxins may be designated as oral toxins (which are poisonous to eat and include bacterial poisons and products of decomposition), parenteral toxins (venom produced in specialized glands and injected mechanically [by spine, needle, fang, fin, or dart]), and crinotoxins (venom produced in specialized glands and administered as slime, mucus, or gastric secretion). Within these three subdivisions, further classifications are by phylogeny, chemical structure, and clinical syndrome.

Although all venoms are poisons, not all poisons are venoms. Venoms can be released in varying amounts and have evolved for conquest and defense. It is theorized that offensive (prey capture and digestion) venoms are generally perioral (mouth, fang, or tentacle) and that defensive venoms are aboral (tail and sting) or dermal (barb and secretion). In the evolutionary scheme, it appears that many venomous fish seek specific self-defense, whereas poisonous fish are noxious in a nonspecific manner.[7] A brief comparison of the features of venoms and poisons shows that, generally, poisons produced in skin, muscle, blood, or organs are heat stable (46° to 49° C [115° to 120° F]), are gastric acid stable, and carry seasonal toxicity. They are not "released," and they may lack a well-defined biologic function. Venoms are more commonly heat labile, gastric acid labile, and nonseasonal in toxicity.

In snakes, the latency, toxicity, and duration of venom effects are related to the route of envenomation. Intravascular injection is significantly more lethal than intraperitoneal or transcutaneous injection, as determined by the dose that produces 50% lethality in a group (LD_{50}). This principle is not commonly applied to marine venoms because few encounters involve direct intravascular injection.

Most venoms are high-molecular-weight amalgams of vasoactive amines, proteolytic enzymes, and other biogenic compounds. These substances denature membranes, catabolize cyclic 3′,5′-adenosine monophosphate, degranulate mast cells,

and weight of 80 to 90 lb (36–41 kg). It swims slowly and sluggishly and is usually found partially buried in bottom mud and sand. Well camouflaged, its dorsal surface is multicolored and the ventral surface creamy white. The externally visible electric organs are located on each side of the anterior part of the disc between the anterior extension of the pectoral fin and the head, extending from above the level of the eye backward past the gill region onto the ventral surface. The electric organs are composed of a honeycomb network of modified muscles organized into columnar prismlike structures and connective tissue, which generate an electrical charge by neuromuscular activity. The muscle cells *(electroplaques)* are stacked 500 to 1000 deep, creating up to 500 cm^2 of surface area. The electroplaques depolarize in series and in parallel simultaneously, producing amperage sufficient to stun prey. Species in the tropical eastern Pacific include the smaller-bodied lesser ray, *Narcine entemedor,* and the bulls-eye ray *Diplobatis ommata.*

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Most venoms are high-molecular-weight amalgams of vasoactive amines, proteolytic enzymes, and other biogenic compounds. These substances denature membranes, catabolize cyclic 3′,5′-adenosine monophosphate, degranulate mast cells,

provoke histamine release, initiate arachidonate metabolism, accelerate coagulopathy, interfere with cellular transport mechanisms, disrupt metabolic pathways, impede neuronal transmission, and evoke anaphylaxis and shock. Although many marine venoms are composed of protein and polypeptide subunits, they lack sufficient immunogenicity to allow development of antitoxins or antivenoms. Poisons represent metabolic byproducts and are usually of smaller molecular weight.

Taxonomy of marine animals can sometimes be confusing. The hierarchy, in descending order, is kingdom, phylum, class, order, family, genus, and species.

ALLERGIC REACTIONS

Anaphylaxis

An envenomation or the administration of antivenom can elicit an allergic reaction. In the previously sensitized individual, the antigen (venom, aquatic protein, or animal serum) complexes with immunoglobulin E (IgE) and perhaps with IgG homocytotropic antibodies or activated complement cleavage products attached to the membranes of mast cells and basophils. This induces membrane permeability, which allows degranulation or membrane production of histamine, serotonin, kinins, prostaglandins, platelet-activating factor, eosinophil and neutrophil chemotactic factors, leukotrienes, and other bioactive chemical mediators.[9]

The signs and symptoms of anaphylaxis may occur within minutes of exposure. They include hypotension, bronchospasm, tongue and lip swelling, laryngeal edema, pulmonary edema, seizures, cardiac arrhythmia, pruritus, urticaria, angioedema, rhinitis, conjunctivitis, nausea, vomiting, diarrhea, abdominal pain, gastrointestinal bleeding, and syncope. Most severe allergic reactions occur within 15 to 30 minutes of envenomation, and nearly all occur within 6 hours. Fatalities are often related to airway obstruction or hypotension. Acute elevated pulmonary vascular resistance may contribute to hypotension that results from generalized arterial vasodilation.[10,12]

Treatment

Decisive treatment should be instituted at the first indication of hypersensitivity. Specific treatment recommendations for anaphylaxis are found in Box 56-3.

Antivenom Administration. A number of marine envenomations, such as those by the box-jellyfish and certain sea snakes, may require the administration of specific antivenom. Marine antivenoms are raised in horses or sheep and therefore may be antigenic in humans, inducing both immediate and delayed hypersensitivity. Most authorities recommend that a skin test be performed for sensitivity to horse serum, if the clinical situation permits, after a sea snake envenomation. (There is rarely time for a skin test when there has been a *Chironex* envenomation, which requires immediate intervention.) A skin test should be done only after deciding to administer antivenom; it is *not* done to determine whether antivenom is necessary. The purpose of sensitivity testing is to allow adequate prophylaxis against anaphylaxis. The skin test is performed with an intradermal injection into the upper extremity of 0.02 mL of a 1:10 dilution of horse serum test material in saline, with 0.02 mL saline in the opposite extremity as a control. Erythema and a wheal with pseudopodia are present in 15 to 30 minutes in a positive response. Because antivenom contains many times the protein content of horse serum used for skin testing, the use of antivenom for skin testing may increase the risk of anaphylactic reaction. If the skin test is positive, the antivenom intended for intravenous (IV) infusion should be diluted in sterile water to a 1:100 concentration for administration. Successive vials should be less dilute if the allergic reaction is minimal (controlled by antihistamines and epinephrine). A negative skin test does not preclude the possibility of an anaphylactic response to antivenom administration.

The rationale for administering antivenom is to provide early and adequate neutralization of the toxin at the tissue site of entry before it gains systemic dominance. Except for stonefish antivenom, the product is preferentially administered intravenously, taking care to provide adequate doses for children and older adults, who have a decreased volume of distribution and increased sensitivity to venom effects. The antivenom intended for IV administration should always be diluted with normal saline, Ringer's lactate, or dextrose 5% in water.

Marine antivenoms are produced and distributed in the Indo-Pacific regions. They include the following:

1. *Chironex fleckeri* (box-jellyfish) antivenom, from Commonwealth Serum Laboratories (CSL), Parkville, Victoria, Australia. This hyperimmune sheep globulin preparation may be used to neutralize the stings of *Chironex fleckeri* and *Chiropsalmus quadrigatus*.
2. *Enhydrina schistosa* (beaked sea snake) sea snake antivenom, from CSL. This hyperimmune horse globulin preparation may be used to neutralize the bites of most sea snakes. It is prepared by immunizing horses with venom from *E. schistosa* and the Australian tiger snake *Notechis scutatus*.
3. *Notechis scutatus* (tiger snake) antivenom, from CSL, has traditionally been recommended as the antivenom of second choice against the bites of most sea snakes. However, recent work suggests that it is not effective against sea snake bites, and so it should not be relied on for clinical efficacy in humans.[187]
4. *Enhydrina schistosa* (beaked sea snake) monovalent antivenom, from the Haffkine Institute in Mumbai, India, appears to be no longer available, as the product is not noted on their website. It was formerly used to neutralize the bites of most sea snakes and was most effective against the bite of *E. schistosa*.
5. *Synanceja trachynis* (stonefish) antivenom, from CSL. This hyperimmune horse globulin preparation may be used to neutralize the stings of stonefish and more virulent scorpionfish species.

A person who is known to be sensitive to horse or sheep serum, has a positive skin test, or develops signs of an allergic reaction or anaphylaxis during antivenom therapy requires aggressive medical management. A recipient of antivenom should be pretreated with 50 to 100 mg of IV diphenhydramine (1 mg/kg in children). After this, the initial dose of antivenom is administered at a rate no faster than one vial each 5 minutes. If no allergic manifestation ensues, the antivenom can be administered at a more rapid rate. If signs of anaphylaxis develop, usually heralded by an urticarial eruption or pruritus, 0.1 to 0.2 mL aliquots of antivenom should be alternated with 3 to 10 mL (0.03 to 0.1 mg) IV doses of aqueous epinephrine 1:100,000 (infused over 5 to 10 minutes). Alternatively, an epinephrine drip may be prepared as discussed in Chapter 56. The

victim should be managed in an intensive care unit, with electrocardiographic and blood pressure monitoring. The dose of epinephrine should not elevate the pulse rate to greater than 150 beats/min. The administration of IV epinephrine may cause transient hypokalemia as potassium is driven intracellularly; cessation of the epinephrine infusion may create a transient hyperkalemia as the potassium regains entry into the extracellular space. If a victim is highly allergic to antivenom, serious consideration should be given to supportive therapy (including hemodialysis) without antivenom administration.

In one series, stonefish antivenom was administered to 24 victims in a dose of one or two ampules by the intramuscular (IM) route, without any "immediate reactions" reported.[161] In this same report, six victims received box-jellyfish antivenom by the IV route without immediate or delayed reactions. Anecdotal reports indicate that box-jellyfish antivenom has been administered by the IM route in the field approximately 80 times to date without any episode of anaphylaxis.

Serum Sickness

The formation of IgG antibodies in response to antigens present in antivenom (prepared in heterologous serum) results in the deposition of immune complexes in many tissue sites, notably in the walls of blood vessels. These complexes induce vascular permeability, activate the complement cascade and chemotactic factors, degranulate mast cells, and trigger the release of proteolytic enzymes. Decreased levels of C_3 and C_4 are accompanied by increased C_{3a}/C_{3a} des-arginine, a split product C_3.[63,93] Although immune complexes can be measured by various tests (Raji-cell IgG assay and C_{1q}-binding assay), levels of immune complexes may not correlate with the clinical presentation.[63,121] Cutaneous venulitis may precede vasculitis. Dermal biopsy of lesional skin may reveal leukocytoclastic vasculitis.

Symptoms are generally present within 8 to 24 days and include fever, arthralgias, malaise, urticaria, lymphadenopathy, urticarial and morbilliform skin rashes, peripheral neuritis, and swollen joints. It is not uncommon for the primary urticarial lesion to be noted at the injection site. Serum sickness is managed with the administration of corticosteroids. An initial loading dose of prednisone (40 to 60 mg for adults, and 2 to 5 mg/kg, not to exceed 50 mg, for children) should be administered and maintained daily until symptoms markedly resolve. The corticosteroid should be tapered over a 2- to 3-week course to avoid induction of adrenal insufficiency. Aspirin or other nonsteroidal anti-inflammatory agents are rarely helpful and may be contraindicated because of circulating immune complex–induced platelet dysfunction.

SPONGES (PHYLUM PORIFERA)

Life and Habits

There are approximately 5000 species of sponges (phylum Porifera, predominantly of class Desmospongiae), which are supported by horny, but elastic, internal collagenous skeletons of spongin, some forms of which we use as bath sponges. Sponges are without digestive, excretory, respiratory, endocrine, circulatory, and nervous systems. Embedded in the connective tissue matrices and skeletons are spicules of silicon dioxide (silica) or calcium carbonate (calcite), by which some sponges can be definitively identified. In general, sponges are stationary acellular animals that attach to the sea floor or coral beds and may be colonized by other sponges, hydrozoans, mollusks, coelenterates, annelids, crustaceans, echinoderms, fish, and algae. These secondary coelenterate inhabitants are responsible for the dermatitis and local necrotic skin reaction termed sponge diver's disease (maladie des plongeurs).[156] In recognition of a medicinal property, the ancient Greeks burnt sea sponges and inhaled the vapors for prophylaxis against goiter.[44] Sponges harbor various biodynamic substances, with possible antineoplastic, antibacterial, growth-stimulating, antihypertensive, neuropharmacologic, psychopharmacologic, and antifungal properties. A number of sponges produce crinotoxins that may be direct dermal irritants, such as subcritine, halitoxin (*Haliclona* species), *p*-hydroxybenzaldehyde, and okadaic acid. These may be present in surface or internal secretions. Murine monoclonal antibodies against okadaic acid intended for use in an assay system for the detection of diarrhetic shellfish poisoning have been prepared from the sponge *Halichondria okadai*.[171] The causative agent of Dogger Bank itch, (2-hydroxyethyl) dimethylsulfoxonium chloride, has been isolated from the marine sponge *Theonella aff. mirabilis*.[176]

Figure 73-1. Pacific fire sponge. (From Norbert Wu, with permission; available at www.norbertwu.com.)

Clinical Aspects

Two general syndromes, with variations, are induced by contact with sponges. The first is a pruritic dermatitis similar to plant-induced allergic dermatitis, although the dermatopathic agent has not been identified. Rarely, erythema multiforme or an anaphylactoid reaction may be present. A typical offender is the friable Hawaiian (Fig. 73-1) or West Indian fire sponge *(Tedania ignis)*, a brilliant yellow-vermilion-orange (Fig. 73-2) or reddish-brown organism with a crumb-of-bread appearance found off the Hawaiian Islands and the Florida Keys.[146,154] This sponge grows in thick branches, which extend from a larger base and are easily broken off. Other culprits include *Fibula* (or *Neofibularia*) *nolitangere*, the poison bun sponge (Fig. 73-3) (and the related sponge *Neofibularia mordens*), and *Microciona prolifera*, the red moss sponge (found in the northeastern United States).[89] *F. nolitangere* is found in deeper water and grows in clusters, with holes (oscula) large enough to admit a diver's finger. It is brown (Fig. 73-4) and bready in texture, so it may crumble in the hands.

Figure 73-2. Atlantic fire sponge. (Photo by Dee Scarr.)

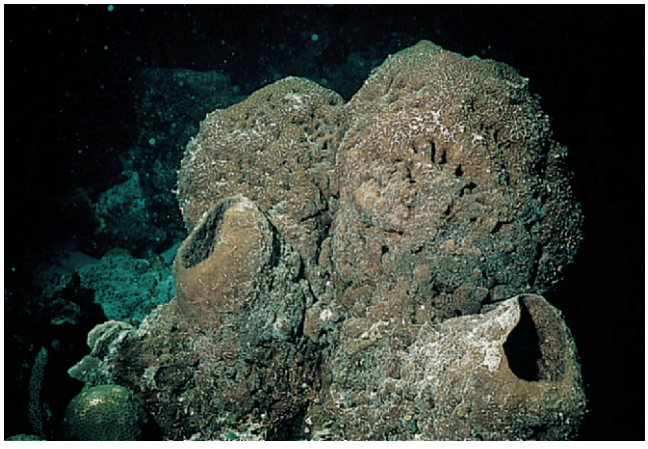


Figure 73-3. Poison bun sponge *Neofibularia nolitangere.* (Photo by Dee Scarr.)

Within a few hours after skin contact, but sometimes within 10 to 20 minutes, the reactions appear. They are characterized by itching and burning, which may progress to local joint swelling, soft tissue edema, vesiculation, and stiffness, particularly if small pieces of broken sponge are retained in the skin near the interphalangeal or metacarpophalangeal joint. Most victims of sponge-induced dermatitis have hand involvement, because they handled the sponges without proper gloves. In addition, abraded skin, such as that which has been scraped on stony coral, may allow more rapid or greater absorption of toxins.[141] When the sponge is penetrated, torn, or crumbled, the skin is exposed to the toxic substances. Untreated, mild reactions subside within 3 to 7 days. When large skin areas are involved, the victim may complain of fever, chills, malaise, dizziness, nausea, muscle cramps, and formication. Bullae induced by contact with *M. prolifera* may become purulent. Systemic erythema multiforme or an anaphylactoid reaction may develop 1 to 2 weeks after a severe exposure.[193] The skin may become mottled or purpuric, occasionally after a delay of up to 10 days.[154]

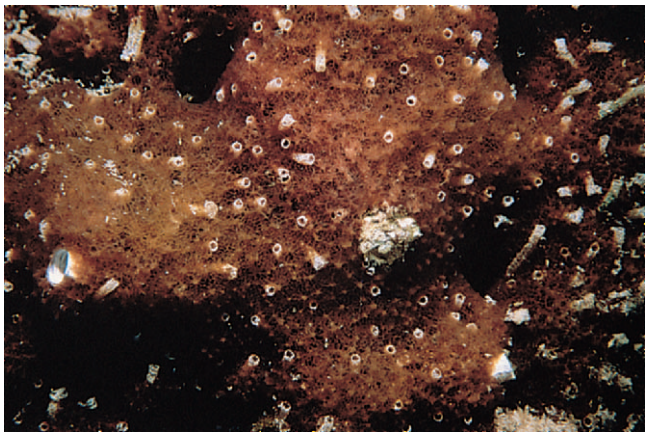


Figure 73-4. Crumb-of-bread appearance of poison bun sponge. (Photo by Dee Scarr.)

The second syndrome is an irritant dermatitis and follows the penetration of small spicules of silica or calcium carbonate into the skin. Most sponges have spicules; toxic sponges may possess crinotoxins that enter microtraumatic lesions caused by the spicules.

In severe cases, surface desquamation of the skin may follow in 10 days to 2 months. No medical intervention can retard this process. Recurrent eczema and persistent arthralgias are rare complications.

Treatment

Because distinguishing clinically between the allergic and spicule-induced reactions is usually impossible, it is reasonable to treat for both. The skin should be gently dried. Spicules should be removed, if possible, using adhesive tape, a thin layer of rubber cement, or a facial peel. As soon as possible, dilute (5%) acetic acid (vinegar) soaks for 10 to 30 minutes 3 or 4 times a day should be applied to all affected areas.[154,157,192] Isopropyl alcohol (40% to 70%) is a reasonable second choice. Although topical steroid preparations may help relieve the secondary inflammation, they are of no value as an initial decontaminant. If they precede the vinegar soak, they may worsen the primary reaction. Delayed primary therapy or inadequate decontamination can result in the persistence of bullae, which may become purulent and require months to heal.

Erythema multiforme may require the administration of a systemic glucocorticoid, beginning with a moderately high dose (prednisone, 60 to 100 mg) tapered over 2 to 3 weeks. Anecdotal remedies for the management of sponge envenomation that have been suggested without demonstration of efficacy include antiseptic dressings, broad-spectrum antibiotics, methdilazine, tripelennamine, phenobarbital, diphenhydramine, promethazine, and topical carbolic oil or zinc oxide cream.[154]

After the initial decontamination, a mild emollient cream or steroid preparation may be applied to the skin. If the allergic component is severe, particularly if there is weeping, crusting, and vesiculation, a systemic glucocorticoid (prednisone, 60 to 100 mg, tapered over 2 weeks) may be beneficial. Severe itching may be controlled with an antihistamine.

Because *Clostridium tetani* has been cultured from sea sponges, they should not be used to pack wounds. Proper antitetanus immunization should be part of sponge dermatitis

therapy. Frequent follow-up wound checks are important because significant infections sometimes develop.[90] Infected wounds should be cultured and managed with antibiotics (see Chapter 72). If sponge poisoning induces an anaphylactoid reaction, standard resuscitation using epinephrine, bronchodilators, corticosteroids, and antihistamines should be undertaken.[193]

As mentioned previously, sponge diver's disease is not caused by any toxin produced by the sponge but rather is a stinging syndrome related to contact with the tentacles of the small coelenterate anemone *Sagartia rosea* (family Sagartiidae) or anemones from the genus *Actinia* (family Actiniidae) that attach to the base of the sponge. Treatment should include that for coelenterate envenomation.

Prevention

All divers and net handlers should wear proper gloves. Sponges should not be broken, crumbled, or crushed with bare hands. If the victim brings a specimen, the physician should take care to document its appearance. Dried sponges may remain toxic.

COELENTERATES (PHYLUM CNIDARIA)

Coelenterates are an enormous group, comprising approximately 10,000 species, at least 100 of which are dangerous to humans. Coelenterates that possess the venom-charged stinging organoids commonly called nematocysts are known as cnidaria (nettle); those without nematocysts are acnidaria. For practical purposes the cnidaria can be divided into three main groups: (1) hydrozoans, such as the Portuguese man-of-war, (2) scyphozoans, such as true jellyfish, and (3) anthozoans, such as soft corals (alcyonarians), stony corals, and anemones. Gorgonians (order Gorgonacea, class Anthozoa, subclass Alcyonaria) secrete mucinous exudates having toxic effects in experimental animals that can be characterized as hemolytic, proteolytic, cholinergic, histaminergic, serotonergic, and adrenergic.[61] Fenner divides jellyfish into three main classes: schyphozoans (true jellyfish), with tentacles arising at regular intervals around the bell; cubozoans (e.g., box-jellyfish), with tentacles arising only from the corners (and these may be further divided into carybdeids [e.g., Irukandji], with only one tentacle [except in rare cases] arising from each lower corner of the bell, and chirodropids, which have more than one tentacle in each corner of the bell); and other jellyfish, such as the hydrozoans (e.g., *Physalia* species).

Morphology, Venom, and Venom Apparatus

Coelenterates ("hollow gut") are predators that feed on other fish, crustaceans, and mollusks. They are radially symmetric animals of simple structure (95% water) and exist in two predominant life-forms—either sedentary, asexual polyps (hydroids) or free-swimming and sexual medusae.[82] They are the lowest form of life organized into different layers.[141] Generally, the polyps are saclike creatures attached to the substrate at the caudal (aboral) end, with a single orifice or mouth at the upper end surrounded by stinging tentacles (dactylozooids). This form predominates in the hydrozoans and anthozoans. The medusa is a bell-shaped creature, with a floating gelatinous umbrella from which hang an elongated tubular mouth and marginal nematocyst-bearing tentacles. This form predominates in the scyphozoans and is also found in the hydrozoans.

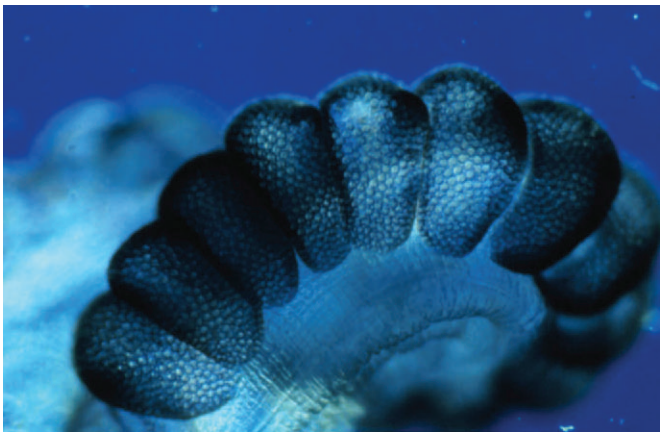


Figure 73-5. Unfired nematocysts on a *Physalia* tentacle. (Photo by Peter Parks.)

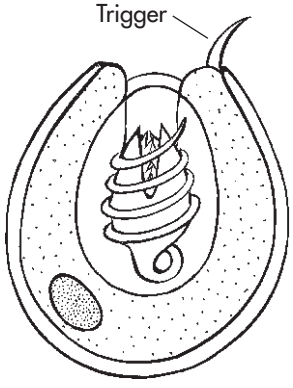


Figure 73-6. Nematocyst before discharge.

Cnidocytes are living cells that encapsulate the nonliving intracytoplasmic stinging organoids called cnidae (which include nematocysts, spirocysts, and ptychocysts). Nematocyst capsules are formed in clusters of differentiating nematocysts. After differentiation into the different types of capsules, the clusters break up to allow single cells to migrate to tentacles, where they become mounted in specialized tentacle epithelial cells, called battery cells.[163] The cnidocytes are located on the outer epithelial surfaces of the tentacles (Fig. 73-5) or near the mouth and are triggered by contact with the victim's body surface. The nematocyst is contained within an outer capsule called the cnidoblast, to which is attached a single pointed "trigger," or cnidocil. The nematocyst (3 to 10 µm in diameter) is filled with fluid and contains a hollow, sharply pointed, coiled or folded "thread" tubule (nema) (Fig. 73-6). This tubule may attain lengths of 200 to 850 µm and is sufficiently hardy to penetrate a surgical glove. The tubule is initially formed outside the capsule and then invaginates within the wall, so that in the undischarged state, the toxin is located in the folds and invaginations of the tubule's membrane. This membrane hardens via disulfide bond isomerization to form bridges between minicollagen peptides as the capsule attains its final size.[163]

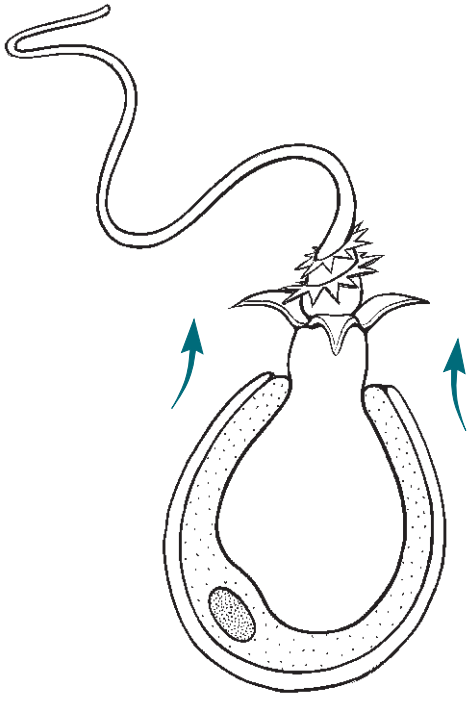


Figure 73-7. Nematocyst after discharge.

The tubule is lined with hollow barbs, which help it penetrate and anchor into the victim. In the undischarged state, the barbs occupy the lumen of the twisted and folded tubule. When the cnidocil is stimulated, either by physical contact or by a chemoreceptor mechanism, it causes the opening of a trapdoor (operculum) in the cnidoblast, and the venom-bearing tubule is everted (Fig. 73-7) within 3 µsec. This exocytosis has been hypothesized to occur because of osmotic swelling of the capsular matrix caused by high concentration of poly-γ-glutamate, influx of water (leading to a hydrostatic pressure of up to 150 atm), release of intrinsic tensile forces (up to 375 MPa on the inner capsule wall), or deformation of the wall-induced internal pressure.[80,163] The sharp tip of the thread tube enters the victim's skin (Figs. 73-8 and 73-9) and envenomation occurs as toxin is translocated by hydrostatic forces from the surface of the everted and extended tubule through the now helically arranged (Fig. 73-10) and extended hollow barbs.[99,100] It has been estimated that the velocity of ejection attains 2 m/sec, which corresponds to an acceleration of 40,000 g, with an estimated skin striking force of 2 to 5 psi.[46] This is one of the most rapid mechanical events found in nature. A human encounter with a large Portuguese man-of-war could conceivably trigger the release of several million stinging cells (Fig. 73-11). It has been estimated that more than 2000 sting penetrations can occur within a single square millimeter of skin. The threads penetrate the epidermis and upper dermis, where the venom diffuses into the general circulation. The agitated victim moves about and assists the venom's distribution by the muscle-pump mechanism. On the basis of mouse studies, it appears that the rapid death of a victim is related to the venom that is discharged directly into capillaries, as opposed to that which must diffuse from the dermis into the bloodstream.

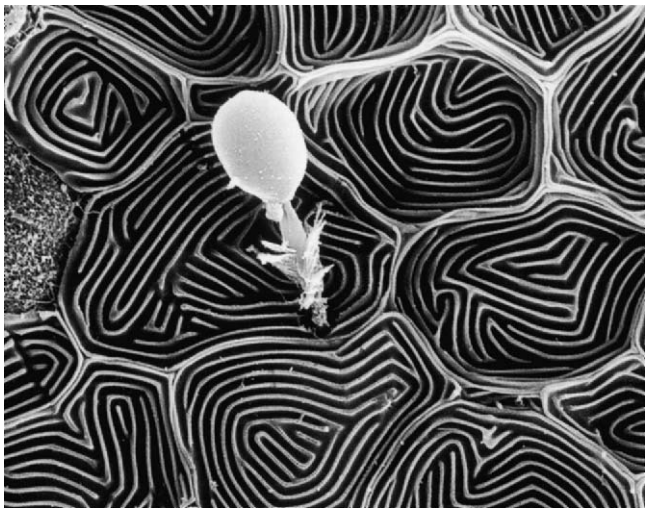


Figure 73-8. Discharged nematocyst that penetrated human skin. (Scanning electron micrograph by Dr. Thomas Heeger.)

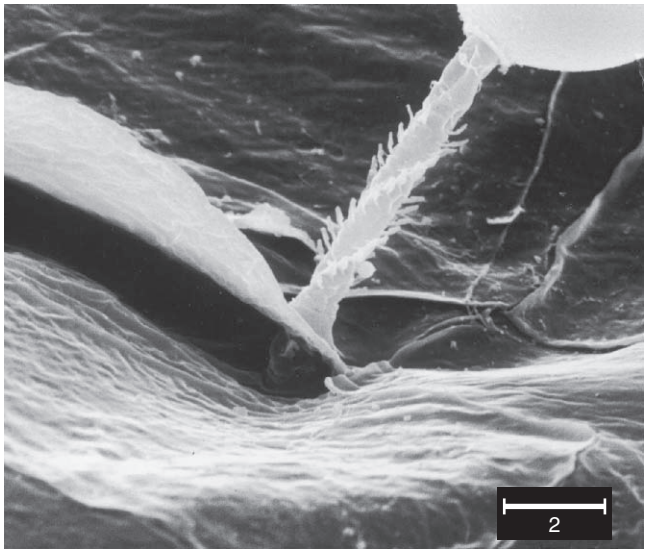


Figure 73-9. An everted tubule of a nematocyst from a lion's mane jellyfish *(Cyanea capillata)* has entered the skin and has lifted an epithelial cell. (Scanning electron micrograph by Dr. Thomas Heeger.)

In the case of the Indo-Pacific box-jellyfish *Chironex fleckeri*, which may carry up to 59 tentacles bearing millions of nematocysts, it is the cigar-shaped microbasic p-mastigophores that are most important in human envenomation (Fig. 73-12). The capsule of the structure holds a hollow coiled tube and granular matrix. The thread tube has a thick butt end that is attached to the operculum. The tube contains three rows of helically arranged spines. When the nematocyst fires into the human victim, the tube everts through the opercular end of the nematocyst, with the butt anchoring first to keep the nematocyst adherent to the victim. The thread then everts through the hollow butt and uncoils, presenting the spines and accompanying

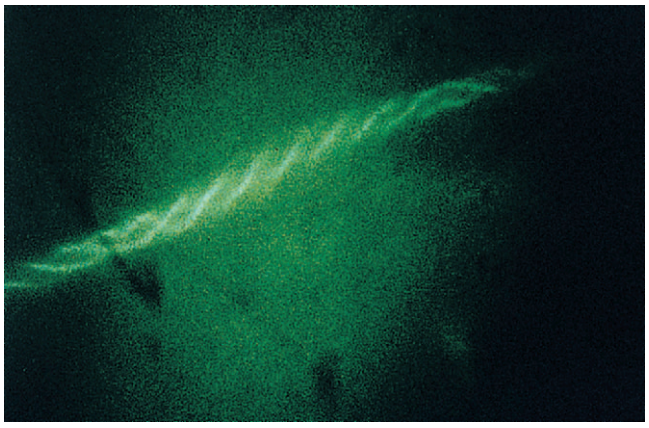


Figure 73-10. Helical arrangement of barbs on the tubule of a nematocyst. (Photo by Amit Lotan.)

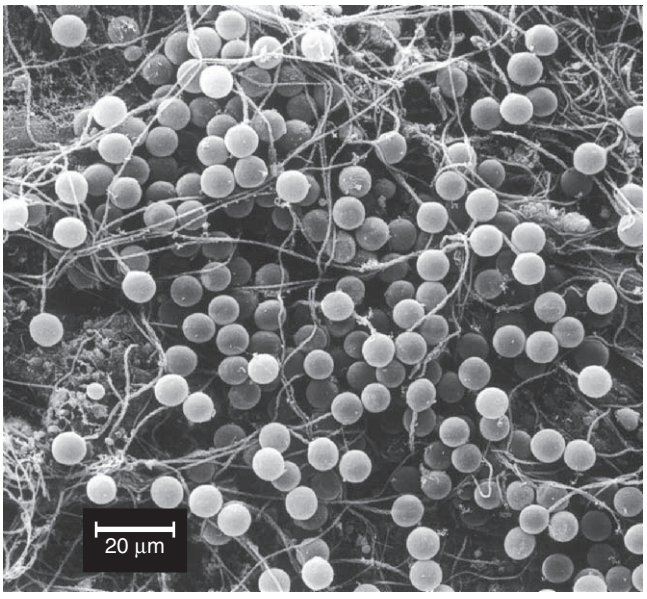


Figure 73-11. Nematocysts of a jellyfish (*Versuriga anadyomene,* Philippines), mostly discharged as seen by everted tubules. (Scanning electron micrograph by Dr. Thomas Heeger.)

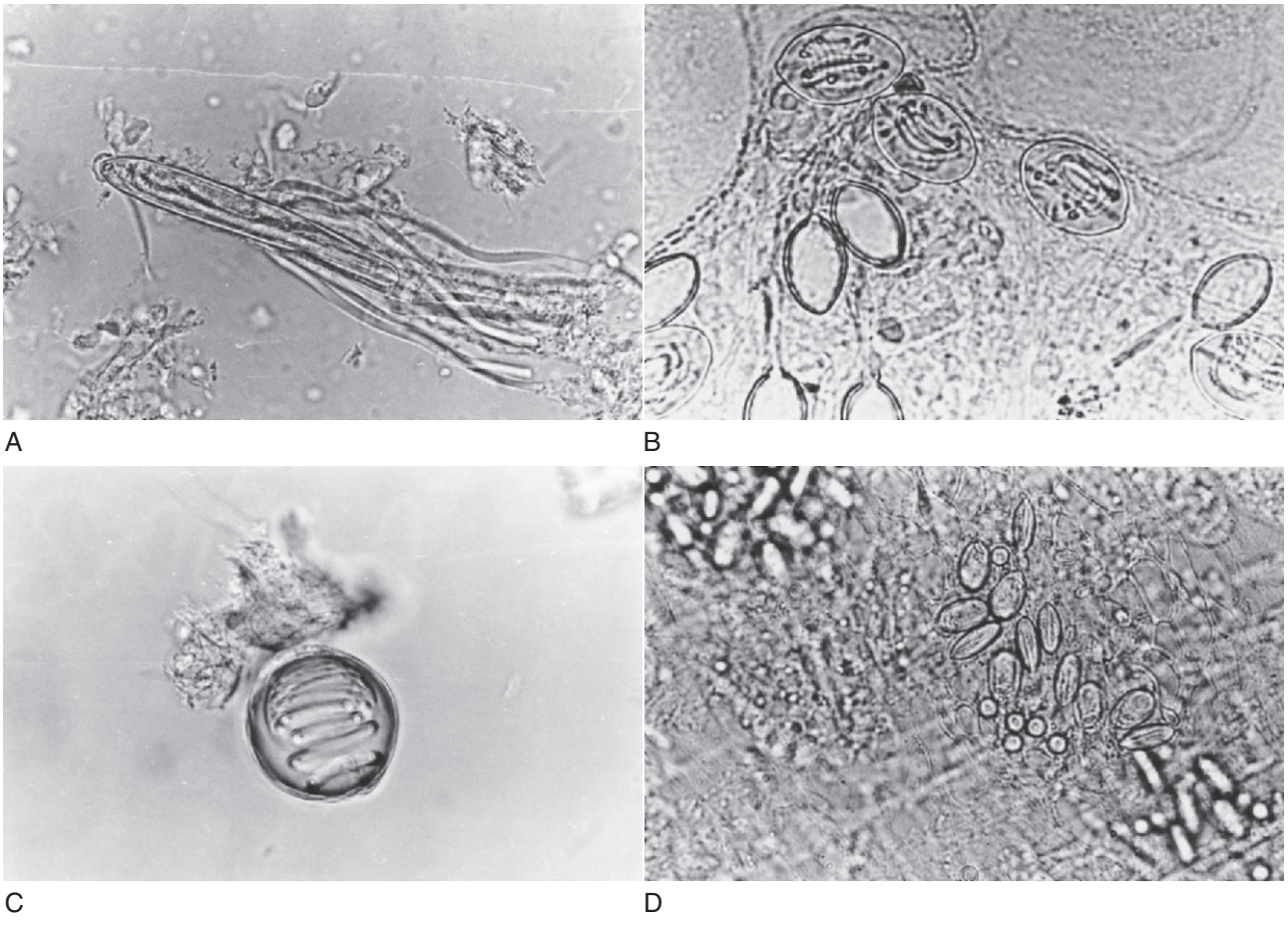


Figure 73-12. Nematocyst identification guide. **A,** Microbasic p-mastigophore (undischarged) of *Chironex fleckeri.* Capsule length, 75 µm. **B,** Same (discharged and undischarged) of Irukandji. **C,** Isorhiza (undischarged) of bluebottle—*Physalia physalis.* **D,** Clustered isorhizas and euryteles on tentacle of "hair jelly"—*Cyanea.* (**A** to **D** courtesy Bob Hartwick.)

toxins to the living tissue. Although the major toxic fractions appear to be present in the nematocysts, there appears to be toxic material present in tentacles denuded of such organelles.[21] The largest nematocysts of *C. fleckeri* can penetrate human skin to a depth of 0.9 mm.[114]

Coelenterate venoms are viscous mixtures of proteins, carbohydrates, and other nonproteinaceous components. Although they are heat labile in vitro, this does not seem to apply in the clinical setting. To date, they have been difficult to fractionate. The primary difficulties encountered in jellyfish venom purification have been the lack of stability and the tendency of active toxins to adhere to each other and to support matrices.[128] Lyophilized crude venom can be prepared in water by homogenization, sonication, and rapid freeze-thawing. A second technique consists of grinding samples with a glass mortar and pestle and using phosphate-buffered saline. This has been done to prepare crude venom from isolated nematocysts of the box-jellyfish, the bells of Irukandji jellyfish, and the oral lobes of blubber jellyfish.[186] Analysis of Western blot tests showed that box-jellyfish antivenom reacted specifically with the venom of each jellyfish, but there is not yet any clinical significance to this observation. Because toxicity was found in the Irukandji jellyfish venom derived by the mortar and pestle method, but not by the lyophilization method, the former was deemed the more efficacious method. Within box-jellyfish venom are protein components ranging from 18 to more than 106 kDa.

Cytolytic toxins have been characterized from *Physalia physalis, Rhizostoma pulmo* (Fig. 73-13), *Chironex fleckeri,* and *Carybdea marsupialis*.[140] Hemolytic activity, phospholipase A_2, and α-chymotrypsin–like serine protease activity have been noted in the venom of *Rhopilema nomadica*.[69] Many jellyfish and marine animal venoms generate autonomic neurotoxicity.[34] This may be a result of their ability to affect ion transport (sodium and calcium in particular), induce channels or pores in nerve and muscle cell membranes, alter membrane configurations, and release mediators of inflammation. Coelenterate venoms can target the myocardium, Purkinje fibers, atrioventricular (AV) node, and aortic ring, as well as injure the hepatic P-450 enzyme family.

Clinical Aspects

For clinical purposes, a considerable phylogenetic relationship exists among all stinging species, so that the clinical features of the coelenterate syndrome are fairly constant, with a spectrum of severity. The severity is related to the season and species (venom potency and configuration of the nematocyst), the number of nematocysts triggered and the size of the animal (venom inoculum), the size and age of the victim (the very young and old and the smaller person tend to be more severely affected), the location and surface area of the sting, and the health of the victim. The wise clinician suspects a coelenterate envenomation in all unexplained cases of collapse in the surf, diving accidents, and near drownings. Any victim in distress pulled from marine waters should be carefully examined for one or more cutaneous lesions that may provide the clue to a coelenterate envenomation.

Mild envenomation may result in only an annoying dermatitis, whereas severe envenomation can progress rapidly to involve virtually every organ system, resulting in significant morbidity and mortality. Clinical envenomation is described here by severity, with the understanding that there is a fair amount of overlap.

Mild Envenomation

The stings caused by the hydroids and hydroid corals, along with lesser envenomations by *Physalia, Velella velella* (Fig. 73-14), *Drymonema dalmatinum* (stinging cauliflower), *Olindias sambaquiensis* (known as *relojinho* in Portuguese; endemic

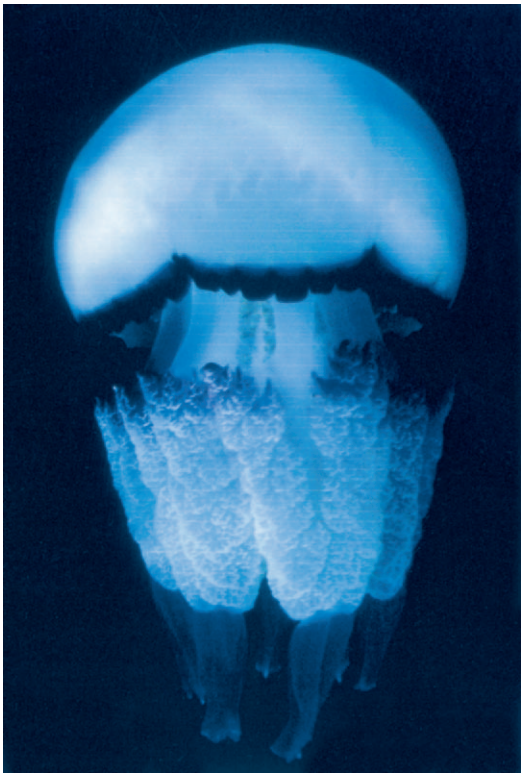


Figure 73-13. Rhizostome medusa, *Rhizostoma pulmo* (Mediterranean Sea). (Photo by Dr. Thomas Heeger.)

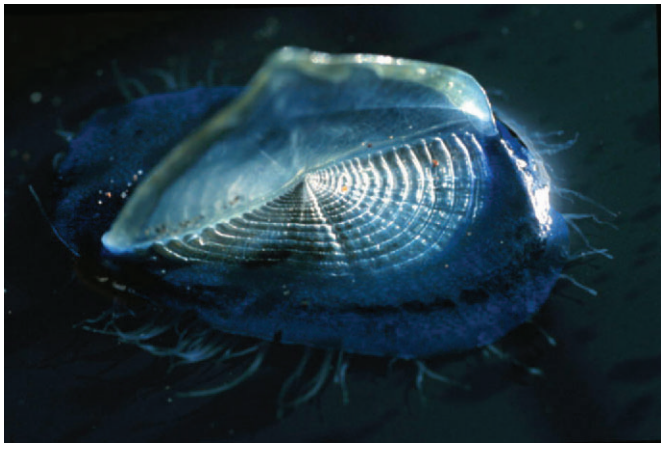


Figure 73-14. By-the-wind sailor, *Velella velella.* (From Norbert Wu, with permission; available at www.norbertwu.com.)

Figure 73-15. Skin irritation from sting of *Olindias* species. (Photo by V. Haddad, Jr.)

to the Blanca Bay area south of Buenos Aires province and found on the southeastern Brazilian coast) (Fig. 73-15), scyphozoans, and anemones, result predominantly in skin irritation.[71,91] There is usually an immediate pricking or stinging sensation, accompanied by pruritus, paresthesias, burning, throbbing, and radiation of the pain centrally from the extremities to the groin, abdomen, and axillae. The area involved by the nematocysts becomes red-brown-purple, often in a linear whiplike fashion, corresponding to tentacle prints. Other features are blistering, local edema, angioedema, and wheal formation, as well as violaceous petechial hemorrhages. Dyspnea due to upper airway obstruction associated with severe facial swelling is possible.[5] The papular inflammatory skin rash is strictly confined to the areas of contact and may persist for up to 10 days. Areas of body hair appear to be somewhat more protected from contact than hairless areas. If the envenomation is slightly more severe, the aforementioned symptoms, which are evident in the first few hours, can progress over a course of days to local necrosis, skin ulceration, and secondary infection. This is particularly true of certain anemone (*Sagartia, Actinia, Anemonia, Actinodendron,* and *Triactis*) stings. A painless "jellyfish sting," in which there is a pattern of hyperpigmented linear streaks, might represent the occurrence of phytophotodermatitis (e.g., from citrus juice spilled on skin and later exposed to light).[20]

Untreated, the minor to moderate skin disorder resolves over 1 to 2 weeks, with occasional residual hyperpigmentation for 1 to 2 months. Rubbing can cause lichenification. Local hyperhidrosis, fat atrophy, and contracture may occur.[25] Facial swelling with sterile abscess formation has been reported.[164] Permanent scarring or keloids may result. Persistent papules or plaques at the sites of contact may demonstrate a predominantly mononuclear cell inflammatory infiltrate, which may represent a delayed hypersensitivity response to an antigenic component of the coelenterate nematocyst or venom. This may be accompanied by localized arthritis and joint effusion. It has been suggested that sensitization may occur without a definite history of a previous sting, because coelenterates may release antigenic and allergenic venom components into the water. Granuloma annulare, which is usually both a sporadic and a familial inflammatory dermatosis, has been associated with a *Physalia utriculus* envenomation.[104] Gangrene has been observed.

Moderate and Severe Envenomation

The prime offenders in this group are the anemones, *Physalia* species, and scyphozoans. The skin manifestations are similar or intensified (as with *Chironex*) and are compounded by the onset of systemic symptoms, which may appear immediately or be delayed by several hours:

- Neurologic: Malaise, headache, aphonia, diminished touch and temperature sensation, vertigo, ataxia, spastic or flaccid paralysis, mononeuritis multiplex, Guillain-Barré syndrome, parasympathetic dysautonomia, plexopathy, radial-ulnar-median nerve palsies, brainstem infarction (not a confirmed relationship), delirium, loss of consciousness, convulsions, coma, and death[26,37,55,115,131]
- Cardiovascular: Anaphylaxis, hemolysis, hypotension, small artery spasm, bradyarrhythmias (including electromechanical dissociation and asystole), tachyarrhythmias, elevated serum troponin I level in the absence of myocardial injury, vascular spasm, deep venous thrombosis, thrombophlebitis, acute myocardial infarction, congestive heart failure, and ventricular fibrillation[81,109,144]
- Respiratory: Rhinitis, bronchospasm, laryngeal edema, dyspnea, cyanosis, pulmonary edema, and respiratory failure
- Musculoskeletal or rheumatologic: Abdominal rigidity, diffuse myalgia and muscle cramps, muscle spasm, fat atrophy, arthralgias, reactive arthritis (seronegative symmetric synovitis with pitting edema),[178] and thoracolumbar pain
- Gastrointestinal: Nausea, vomiting, diarrhea, paralytic ileus,[134] dysphagia, hypersalivation, and thirst
- Ocular: Conjunctivitis, chemosis, corneal ulcers, corneal epithelial edema, keratitis, iridocyclitis, elevated intraocular pressure, synechiae, iris depigmentation, chronic unilateral glaucoma, and lacrimation[64,65,188]
- Other: Acute renal failure, lymphadenopathy, chills, fever, and nightmares

The extreme example of envenomation occurs with *Chironex fleckeri,* the dreaded box jellyfish. *Physalia* and anemone stings, although extremely painful, are rarely fatal. Death after *Physalia* stings has been attributed to primary respiratory failure or cardiac arrhythmia, which may have reflected an element of anaphylaxis.[30,158] Confirmed deaths after coelenterate envenomation have been attributed to *Chironex fleckeri, Chiropsalmus quadrigatus,* and *Chiropsalmus quadrumanus* (Fig. 73-16).[114] *Stomolophus nomurai* (the sand jellyfish) has caused at least eight deaths in the South China Sea.[48] Although there have been other deaths, the animals have not been definitively identified.

Clinical reports and studies on the serologic response to jellyfish envenomation suggest that allergic reactions may play a significant pathophysiologic role in humans. When crude or partially purified nematocyst venom and an antigen are used in an enzyme-linked immunosorbent assay (ELISA), both IgG and IgE can be detected.[62,142] Elevated specific anti–jellyfish IgG and IgE may persist for several years, recurrence of the clinical cutaneous reaction to jellyfish stings may occur within a few weeks without additional contact with the tentacles, and serologic cross-reactivity between the sea nettle *(Chrysaora quinquecirrha)* and *Physalia physalis* occurs. In a case of significant envenomation by the moon jellyfish *Aurelia aurita* (Fig. 73-17),

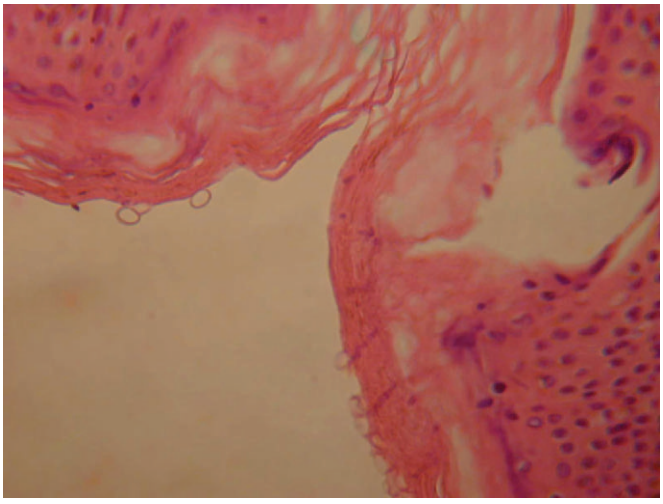


Figure 73-16. Skin biopsy from a child after fatal sting from *Chironex fleckeri.* Nematocysts are seen on the skin. (Photo by Dr. Jamie Seymour.)

Figure 73-17. Moon jellyfish, *Aurelia aurita.* (From Norbert Wu, with permission; available at www.norbertwu.com.)

the victim developed significant cross-reacting antibodies to *Chrysaora quinquecirrha* antigens.[29]

Persons with extracutaneous or anaphylactoid responses to a coelenterate sting have been noted to have higher specific IgG and IgE antibody levels.[142] However, elevated persistent specific anti-jellyfish serum IgG concentrations are not protective against the cutaneous pain resulting from a natural sting.[28] A false-positive ELISA serologic test to venom may occur, as demonstrated by negative skin testing.

A person stung by *Physalia physalis* may have recurrent cutaneous eruptions for 2 to 3 weeks after the initial episode, without repeated exposure to the animal. This may take the form of lichenification, hyperhidrosis, angioedema, vesicles, large bullae, nodules that resemble erythema nodosum, granuloma annulare, or a more classic linear urticarial eruption.[8,27,105] Recurrent eruptions have also followed a solitary envenomation by the cnidarian *Stomolophus meleagris*.[24] In a histologic study of delayed reaction to a Mediterranean Sea coelenterate, skin biopsy demonstrated grouping of human leukocyte antigen–DR-positive cells with Langerhans cells and helper/inducer T lymphocytes, which indicates the possibility of a type IV immunoreaction.[133]

Venom-specific IgG antibodies appear to persist for longer periods than IgM antibodies. The binding of brown recluse spider venom and purified cholera toxin to anti-*Chrysaora* and anti-*Physalia* monoclonal antibodies indicates that there may be a common or cross-reacting antigenic site or sites between these toxic substances and certain coelenterate venoms.[126]

Acute regional vascular insufficiency of the upper extremity has been reported after jellyfish envenomation. It can be manifested by acral ischemia, signs and symptoms of compartment syndrome, and massive edema.[181]

Treatment

Therapy is directed at stabilizing major systemic decompensation, opposing the venom's multiple effects, and alleviating pain.

Systemic Envenomation

Generally, only severe *Physalia* or Cubomedusae stings result in rapid decompensation. In both cases, supportive care is based on the signs and symptoms. Hypotension should be managed with the prompt IV administration of crystalloid, such as lactated Ringer's solution. This must be done in concert with detoxification of any nematocysts (particularly those of *Chironex* or *Chiropsalmus*) that are still attached to the victim, to limit the perpetuation of envenomation. Hypotension is usually limited to very young or older adult victims who suffer severe and multiple stings, the effects of which are worsened by the fluid depletion that accompanies protracted vomiting. Hypertension is an occasional side effect of a cubomedusan envenomation, such as that of the Irukandji *Carukia barnesii*. Excessive catecholamine stimulation is one putative cause, which has prompted clinical intervention with phentolamine, an alpha-adrenergic blocking agent (5 mg intravenously as an initial dose, followed by an infusion of up to 10 mg/hr). Bronchospasm may be managed as an allergic component. If the victim is in respiratory distress with wheezing, shortness of breath, or heart failure, arterial blood gas measurement may be used to guide supplemental oxygen administration by face mask. Seizures are generally self-limited but should be managed with IV diazepam for 24 to 48 hours, after which time they rarely recur.

Any victim with a systemic component should be observed for a period of at least 6 to 8 hours, because rebound phenomena after successful treatment are not uncommon. All older adult victims should undergo electrocardiography and be observed on a cardiac monitor, with frequent checks for arrhythmias. Urinalysis demonstrates the presence or absence of hemoglobinuria, indicating hemolysis after the putative attachment of *Physalia* venom to red blood cell membrane glycoprotein sites.[68] If this is the case, the urine should be alkalinized with bicarbonate to prevent the precipitation of pigment in the renal tubules, while a moderate diuresis (30 to 50 mL/hr) is maintained with a loop diuretic (such as furosemide or bumetanide) or mannitol (0.25 g/kg intravenously every 8 to 12 hours). In rare instances of acute progressive renal failure, peritoneal dialysis or hemodialysis may be necessary.

If there are signs of distal ischemia or an impending compartment syndrome, standard diagnostic and therapeutic measures apply. These include Doppler ultrasound, angiography, or both for diagnosis, regional thrombolysis for acutely occluded blood vessels, measurement of intracompartmental tissue pressures to guide fasciotomy, and so forth. Reversible regional sympathetic blockade may be efficacious if vasospasm is a dominant clinical feature. However, the vasospasm associated with a jellyfish envenomation may be severe, prolonged, and refractory to regional sympathectomy and intra-arterial reserpine or pentoxifylline.[1]

A small child may pick up tentacle fragments on the beach and place them into his or her mouth, resulting in rapid intraoral swelling and potential airway obstruction, particularly in the presence of exceptional hypersensitivity. In such cases, an endotracheal tube should be placed before edema precludes visualization of the vocal cords. In no case should any liquid be placed in the mouth if the airway is not protected. In 1999, a lifeguard in Cairns, Australia, drank from a container containing 4-day-old *Chironex fleckeri* tentacles. He fortunately suffered only a sore throat and transient shortness of breath.

Chironex fleckeri, the box-jellyfish, produces the only coelenterate venom for which a specific antidote exists. To date, the venoms of *Physalia* and *Chrysaora* species have not been sufficiently purified as antigens to permit the production of an antitoxin. Antivenom administration may be lifesaving and should accompany the first-aid protocol previously described.

Pain Control

Often, the pain can be controlled by treating the dermatitis. However, if pain is excruciating and there is no contraindication (such as head injury, altered mental status, respiratory depression, allergy, or profound hypotension), the administration of a narcotic (morphine sulfate, 2 to 10 mg intravenously; nalbuphine, 2 to 10 mg intravenously or intramuscularly; meperidine, 50 to 100 mg, with hydroxyzine, 25 to 50 mg intramuscularly; or meperidine, 15 to 30 mg, with promethazine, 12.5 to 25 mg, or prochlorperazine, 2.5 mg intravenously) is often indicated. Severe muscle spasm may respond to 10% calcium gluconate (5 to 10 mL intravenously by slow push), diazepam (5 to 10 mg intravenously), or methocarbamol (1 g, no faster than 100 mg/min through a widely patent IV line).

Treatment of Dermatitis

If a person is stung by a coelenterate, the following steps should be taken:

1. Immediately rinse the wound with seawater, not with freshwater. Do not rub the wound with a towel or with clothing to remove adherent tentacles. Nonforceful freshwater rinsing or a rubbing variety of abrasion (the latter in the absence of simultaneous application of a decontaminant such as papain or vinegar) is felt to stimulate any nematocysts that have not already fired. Surf life savers (lifeguards) in the United States and Hawaii have reported that a freshwater hot shower applied with a forceful stream may decrease the pain of an envenomation. If this is successful, one theoretical explanation is that the mechanical effect of the water stream (which dislodges tentacle fragments and stinging cells) supersedes the deleterious (sting-stimulating) effect of the hypotonic water. Remove any gross tentacles with forceps or a well-gloved hand. In an emergency, the keratinized palm of the hand can be used because it is relatively protected, but care must be taken to avoid becoming envenomed.

   Commercial (chemical) cold or ice packs applied over a thin dry cloth or plastic membrane have been shown to be effective when applied to mild or moderate *Physalia utriculus* (or bluebottle [see later]) stings.[46] Whether the melt-water from ice applied directly to the skin can stimulate the discharge of nematocysts has not been determined. It has recently been observed by physicians in Australia that hot packs and hot showers (45° C [113° F]) may be more efficacious than application of cold. Neither cold nor heat is as effective as applying vinegar or another decontaminant (see below).

2. Acetic acid 5% (vinegar) is the treatment of choice to inactivate *Chironex fleckeri* toxin. Vinegar does not alleviate the pain from a *Chironex* sting, but it interrupts the envenomation. It may not be extremely effective against *Chrysaora* or *Cyanea.* The detoxicant should be applied continuously for at least 30 minutes or until the pain is relieved. A sting from the Australian *Physalia physalis,* a relatively recently differentiated species, should not be doused with vinegar, as this may cause discharge of up to 30% of nematocysts.[54]

   For a sting from *Chironex fleckeri,* if vinegar is immediately available, a liberal dousing should occur and at least 30 seconds should pass before removing the tentacles. It is no longer recommended to use the pressure-immobilization technique for venom containment. A venolymphatic occlusive tourniquet should be considered only if a topical detoxicant is unavailable, the victim suffers from a severe systemic reaction, and transport to definitive care is delayed.

   For stings from other species, there are substances that may be more specific and therefore more effective. Depending on the species, these include isopropyl alcohol (40% to 70%), dilute ammonium hydroxide, sodium bicarbonate (particularly for stings of the sea nettle *Chrysaora quinquecirrha*), olive oil, sugar, urine, and papain (papaya latex [juice] or unseasoned meat tenderizer powdered or in solution). The last is supposed to work by cleaving active polypeptides into nontoxic amino acids. Lime or lemon juice has been observed on occasion to be effective. Ammonia has been noted to be relatively ineffective for stings of *Carybdea marsupialis* in the Adriatic Sea.[130]

   Perfume, aftershave lotion, and high-proof liquor are not particularly efficacious and may be detrimental. Other substances mentioned to be effective at one time or another, but that are to be condemned on the basis of inefficacy and toxicity, are organic solvents such as formalin, ether, and gasoline.

   There is some evidence that alcohol may stimulate the discharge of nematocysts in vitro; the clinical significance is as yet undetermined. The rescuer must remember that pain relief may not equate with nematocyst inhibition.[114]

   A commercial aqueous solution of aluminum sulfate (20%) and 1.1% anionic surfactant in aqueous solution (Stingose) has sometimes been mentioned as effective on the basis that the aluminum ion interacts with proteins and long-chain polysaccharide components to denature and inactivate venom. Prior treatment with topical alcohol or methylated spirits reduces the effectiveness of the aluminum sulfate solution. This product has for the most part fallen out of favor with clinician jellyfish experts in Australia.

3. No systemic drugs (other than antivenom for a *Chironex* envenomation) are of verifiable use. Ephedrine, atropine, calcium, methysergide, and hydrocortisone have all been touted at one time or another, but no proof exists that they help. Antihistamines may be useful if there is a significant allergic component. The administration of epinephrine is appropriate only in the setting of anaphylaxis.
4. Immersing the area in hot water has generally not been recommended, on the premise that the hypotonic solution causes nematocysts to discharge. However, one study compared hot (40° to 41° C [104° to 105.8° F]) water immersion to papain meat tenderizer or vinegar for treatment of a single-tentacle *Carybdea alata* (Hawaiian box) jellyfish sting to the forearm, and the hot water immersion was found to be the most efficacious.[123] This is attributed to the protein nature of this venom. In a crayfish model of envenomation, exposure to heat reduced the lethality of extracted *C. fleckeri* venom.[36] At temperatures of 43° C (109.4° F) and greater, venom lost its lethality more rapidly the longer the exposure time. Because of the speed of onset of symptoms after *C. fleckeri* envenomation, this approach may be of limited clinical utility, and until human clinical confirmation against other species is obtained, hot water application should not be extrapolated to other species.
5. Once the wound has been soaked with a decontaminant (e.g., vinegar), remaining (and often essentially invisible) nematocysts must be removed. The easiest way to do this is to apply shaving cream or a paste of baking soda, flour, or talc and to shave the area with a razor or similar tool. If sophisticated facilities are not available, the nematocysts should be removed by making a sand or mud paste with seawater and using this to help scrape the victim's skin with a sharp-edged shell or piece of wood. The rescuer must take care not to become envenomed; bare hands must be rinsed frequently. If a scrub brush or pad has been used to treat the envenomation, this step may not result in much, if any, clinical improvement.
6. A topical anesthetic ointment (lidocaine, 2.5%) or spray (benzocaine, 14%), antihistaminic cream (diphenhydramine or tripelennamine), or mild steroid lotion (hydrocortisone, 1%) may be soothing. These are used after the toxin is inactivated. Paradoxical reactions to benzocaine are rarely noted.
7. Victims should receive standard antitetanus prophylaxis.
8. Prophylactic antibiotics are not automatically indicated. Each wound should be checked at 3 and 7 days after injury for infection. Any ulcerating lesion should be cleaned three times a day and covered with a thin layer of nonsensitizing antiseptic ointment, such as mupirocin. A jellyfish sting to the cornea may cause a foreign body sensation, photophobia, and decreased or hazy vision. Ophthalmologic examination reveals hyperemic sclera, chemosis, and irregularity of the corneal epithelium with stromal edema. Depending on the extent of the wound, the anterior chamber may demonstrate the inflammatory response of iridocyclitis (flare with or without cells).[190] The victim should be referred to an ophthalmologist, who may prescribe steroid-containing eye medications, such as prednisolone acetate 1% with hyoscine 0.25%. Applying a traditional skin detoxicant directly to the cornea is not recommended, because it is likely to worsen the tissue injury. Cycloplegia achieved with topical cyclopentolate (0.5% to 1%) may prove useful to achieve pain relief.[188]

Delayed Reaction

A delayed reaction, similar in appearance to erythema nodosum, may be noted in areas of skin contact and may be accompanied by fever, weakness, arthralgias, painful joint swelling, and effusions. This may recur multiple times over the course of 1 to 2 months. The treatment is a 10- to 14-day taper of prednisone, starting with 50 to 100 mg. Prednisone administration may need to be prolonged or repeated with each flare of the reaction.

Persistent Hyperpigmentation

Postinflammatory hyperpigmentation is common after the stings of many jellyfish and other lesser coelenterates. A solution of 1.8% hydroquinone in a glycol and alcohol base (70% ethyl alcohol and propylene glycol mixed at a 3:2 ratio), twice a day as a topical agent for 3 to 5 weeks, has been used successfully to treat hyperpigmentation after a *Pelagia noctiluca* sting.

Persistent Cutaneous Hypersensitivity

Persistent local dermal hypersensitivity may occur after a jellyfish sting, such as that from the Hawaiian box-jellyfish *Carybdea alata*.[166] This is characterized by erythematous papulonodular lesions in the pattern of the original sting, which may persist for months. Treatment, which may be unsatisfactory, consists of topical and intralesional steroids.

Prevention

A topical jellyfish sting inhibitor has been commercialized. Safe Sea ("jellyfish-safe sunblock") by Nidaria Technology, Ltd., Jordan Valley, Israel (www.nidaria.com) was compared in a blinded fashion with conventional sunscreen for protection against *Chrysaora fuscescens* (sea nettle) and *Chiropsalmus quadrumanus* jellyfish. Subjects were stung with jellyfish tentacles on each forearm for up to 60 seconds, and erythema and pain were assessed at 15-minute intervals over a 2-hour period. The jellyfish sting inhibitor prevented sting symptoms of *C. fuscescens* in 10 of 12 subjects and diminished the pain of the jellyfish sting in the remaining two subjects.[88] It was equally impressive with *C. quadrumanus*. The inhibitor is formulated to inactivate jellyfish stinging in several ways: (1) it is hydrophobic and thus prevents tentacles from making sufficient skin contact to induce a sting; (2) glycosaminoglycans in the inhibitor mimic the same compounds found in the jellyfish bell, thus causing self-recognition; (3) the inhibitor contains a competitive antagonist to nonselective receptors on the jellyfish that bind to amino acids and sugar secretions from prey; and (4) calcium and magnesium within the inhibitor block transmembrane signaling channels of the jellyfish, thereby reducing the osmotic forces required to generate the firing pressure within the nematocyst capsule.[88] The product has not yet been tested prospectively against *Physalia, Carukia,* or *Chironex* species, to name a few.

Derma Shield is a topical formulation that contains lanolin, aloe vera, and vitamin E. According to the manufacturer, this chemically inert (1-vinyl-2-pyrrolidione) barrier protectant is hydrophobic (dimethicone and stearic acid) and does not wash off but is shed as the epithelium sloughs naturally. It has been reported anecdotally by ocean bathers to protect against the agents of seabather's eruption. To this author's knowledge, no prospective evaluation of the use of Derma Shield to protect against any coelenterate stings has been published.

Smerbeck and coworkers were assigned a U.S. patent in 1999 for a method and a composition of polymeric quaternary ammonium salts for protecting the skin from jellyfish stings.

A protocol has been developed to establish the effectiveness of topical agents to block firing of nematocysts.[33] Unreliable topical barriers include petrolatum, mineral oil, silicone ointment, cocoa butter, and mechanic's grease.

If jellyfish are sighted, they should be given a wide berth because the tentacles may trail great distances from the body. All swimmers and divers in hazardous areas should be on constant alert. Persons should not dive headfirst into jellyfish-infested waters; it is far safer to walk in. Bathers should wear protective clothing in infested areas. This includes "stinger suits" or a double thickness of panty hose. If "stinger enclosures" are present, bathers should stay within the netted barriers.

Divers concerned about jellyfish tentacles dangling from the surface or congregations of creatures at the surface should remain deeper than 20 feet and should always check snorkel and regulator mouthpieces for tentacle fragments before entering the water in endemic areas. In areas inhabited by anemones and hydroid corals, protective gloves should be worn when handling specimens. Beached dead jellyfish or tentacle fragments washed up after a storm can still inflict serious stings. Any person stung by a jellyfish should leave or be assisted from the water because of the risk of drowning.

The Consortium of Jellyfish Stings was formed to gather reports and encourage prospective investigations. The consortium publishes a newsletter edited by Dr. Joseph W. Burnett. A jellyfish sting report form can be obtained from the Internet (http://medschool.umaryland.edu/dermatology/jellyfish.asp) or from the Consortium of Jellyfish Stings, c/o Department of Dermatology, University of Maryland at Baltimore, 4401 Roland Avenue, Baltimore, MD 21210.

Class Hydrozoa

The hydrozoans range in configuration from the feather hydroids and sedentary *Millepora* hydroid coral to the free-floating siphonophore *Physalia* (Portuguese man-of-war).

Hydroids

Hydroids are the most numerous of the hydrozoans. The feather hydroids of the order Leptomedusae, typified by *Lytocarpus philippinus* (fire weed or fire fern), are featherlike or plumelike (Fig. 73-18) animals that sting the victim who brushes against or handles them.[137] After a storm, the branches may be fragmented and dispersed through the water, so that merely diving or swimming in the vicinity causes itching and may induce visible skin irritation.

Clinical Aspects. Contact with the nematocysts of a feather hydroid induces a mild reaction, which consists of instantaneous burning, itching, and urticaria. If the exposure is brief, the skin rash may not be noticeable or it may consist of a faint erythematous and miliary irritation (Fig. 73-19). A second variety of envenomation consists of a delayed papular, hemorrhagic, or zosteriform reaction (Fig. 73-20) with onset 4 to 12 hours after contact. Rarely, erythema multiforme or a desquamative eruption may develop. In turbulent waters or in a strong current, fragments may be washed into a diver's mask or regulator mouthpiece; this will be evident as a burning sensation in the conjunctivae or oral mucous membranes. Systemic

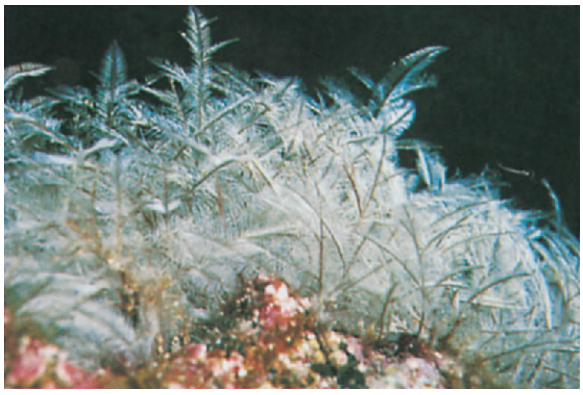


Figure 73-18. Coelenterate hydroid. (Photo by Paul Auerbach, MD.)

Figure 73-19. Hydroid sting on the arm of a diver. (Photo by Neville Coleman.)

Figure 73-20. Fernlike hydroid print on the knee of a diver. (Photo by Paul Auerbach, MD.)

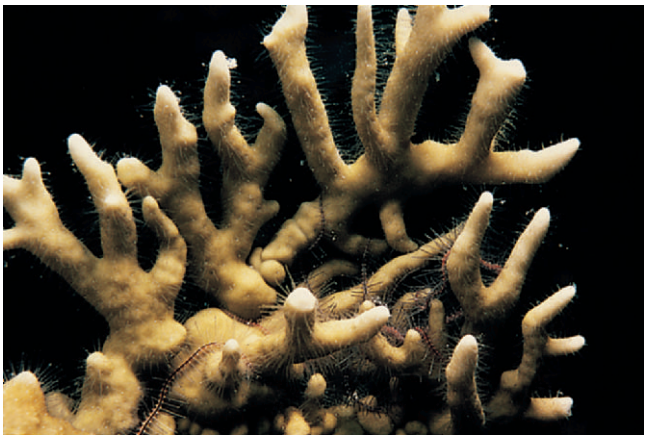


Figure 73-21. Fire coral. (Photo by Paul Auerbach, MD.)

Figure 73-22. Fire coral sting of the author. (Photo by Kenneth Kizer, MD.)

manifestations (such as abdominal pain, nausea, vomiting, diarrhea, muscle cramps, and fever) are rarely reported and are associated with large areas of surface involvement. Allergic sensitization and subsequent anaphylaxis have been proposed.

Treatment. The skin should be rinsed with seawater and gently dried without abrasive activity. Application of freshwater and brisk rubbing are strictly prohibited because they encourage any nematocysts remaining on the skin to discharge and thus worsen the envenomation. An application of 5% acetic acid (vinegar) or isopropyl alcohol (40% to 70%) to the skin for 15 to 30 minutes has traditionally been recommended to relieve the cutaneous reaction. In an in vitro evaluation, vinegar and urine caused discharge of a few nematocysts in 10% to 15% of defensive tentacle polyps; methylated spirits were found to cause gross discharge of microbasic mastigophores in all defensive polyps.[137] Freshwater did not cause discharge. On the basis of this study, the authors recommended that freshwater irrigation and the application of ice be used to treat acute stings. However, the clinical correlation remains to be described.

Alternative topical agents are addressed in the larger discussion on therapy for coelenterate stings. After pain relief is achieved, a mild steroid cream (hydrocortisone, 1%) or moisturizing lotion may be applied.

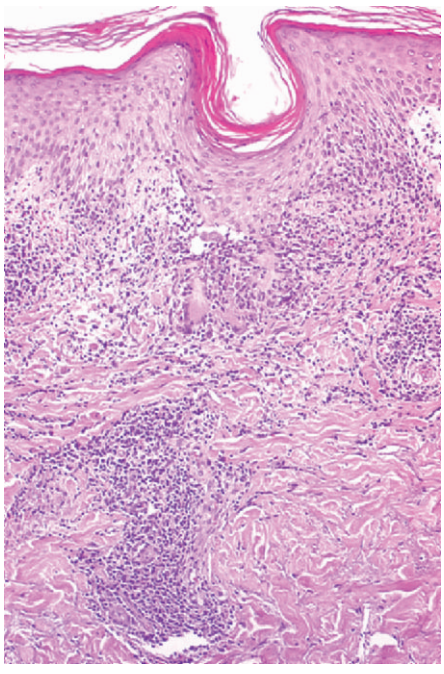
Fire Coral

The stony, hydroid, and coral-like *Millepora* species (e.g., *Millepora alcicornis*), or fire corals, are not true corals. They are widely distributed in shallow tropical waters. Sessile creatures, they are found attached to the bottom in depths of up to 1000 m (3281 ft). They are often mistaken for seaweed because they attach to pilings, rocks, shells, or coral. Although smaller segments resemble Christmas trees or bushes 3 to 4 inches (7.6 to 10.2 cm) in height, they may attain heights of 6.6 ft (2 m). The color ranges from white to yellow-green, with pale yellow (Fig. 73-21) most common. Rare purple fire corals exist. Fire coral is structured on a razor-sharp calcium carbonate (calcic limestone) exoskeleton, which is an important component in the development of coral reefs. The outcroppings assume upright, clavate, bladelike, honeycomb, or branching calcareous growth structures that form encrustations over coral and objects such as sunken vessels. From numerous minute surface gastropores protrude tiny nematocyst-bearing tentacles, wherein lies the stinging apparatus. *M. alcicornis* probably accounts for more coelenterate envenomations than any other species. Unprotected and unwary recreational scuba enthusiasts handle, kneel on, or lean on this marine stinger.

Figure 73-23. Hyperpigmentation of forearm depicted in Figure 73-22 after a fire coral sting. (Photo by Kenneth Kizer, MD.)

Clinical Aspects. Immediately after contact with fire coral, the victim suffers burning or stinging pain, rarely with central radiation. Intense and painful pruritus follows within seconds, which frequently induces the victim to rub the affected area vigorously, worsening the envenomation. Over the course of 5 to 30 minutes, urticarial wheals develop, marked by redness, warmth, and pruritus (Fig. 73-22). The wheals become moderately edematous and reach a maximal size in 30 to 60 minutes. Untreated, they flatten over 14 to 24 hours and resolve entirely over 3 to 7 days, occasionally leaving an area of hyperpigmentation (Fig. 73-23) that may require 4 to 8 weeks to disappear. The pain generally resolves without treatment in 30 to 90 minutes. In the case of multiple stings, regional lymph nodes may become inflamed and painful. This does not necessarily

A



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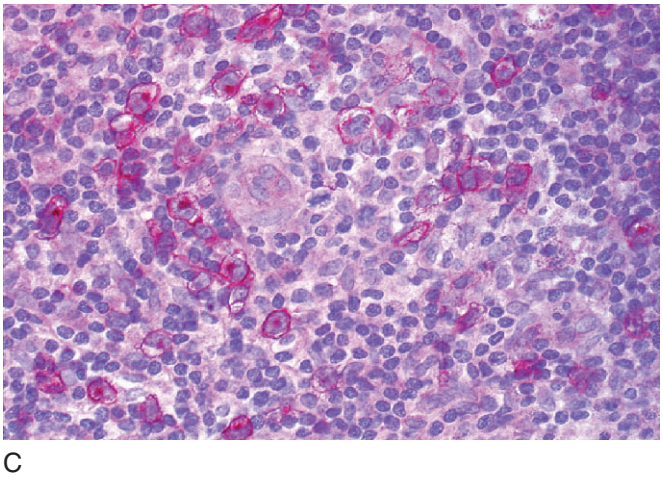


C

Figure 73-24. **A,** Streaks of red papules on the knee of a victim stung by a Red Sea fire coral. **B,** Wedge-shaped inflammatory infiltrate with edema of the papillary dermis and an epithelioid granuloma (hematoxylin and eosin, ×100). **C,** CD30+ atypical lymphoid cells (alkaline phosphatase–antialkaline phosphatase, ×400). (Photos courtesy Dr. Clelia Miracco.)

indicate a secondary infection. Long thoracic mononeuritis with serratus anterior muscle paralysis has been described after *Millepora* sting, confirmed by demonstrated presence of immune-specific IgG.[117] Delayed skin reaction after Red Sea fire coral injury was characterized by superficial granulomas and atypical CD30+ lymphocytes (Fig. 73-24).[116] In another series, contact with fire coral resulted in a typical pruritic urticarial lesion and blister formation, followed by a lichenoid stage that developed 3 weeks after the initial injury; resolution, with residual hyperpigmented macules, required 15 weeks.[2] Grouped or linear papulonodular lesions, round or oval in shape, may follow as a delayed reaction to jellyfish sting.[173] In a rare case, a full-thickness skin burn may occur.[143]

Renal minimal change disease (nephrotic syndrome, renal failure) responsive to corticosteroid therapy has been associated with fire coral exposure.[134a]

Treatment. The skin should be rinsed liberally with seawater and then immediately soaked with acetic acid 5% (vinegar) or isopropyl alcohol (40% to 70%) until pain is relieved. Alternative topical agents are discussed in the larger coelenterate treatment section, earlier. Residual dermatitis is generally not

Figure 73-25. Atlantic Portuguese man-of-war. (From Norbert Wu, with permission; available at www.norbertwu.com.)

very severe and can be managed in a fashion similar to that used after a feather hydroid sting. If the rash becomes eczematous and indolent, it may respond to a course of systemic corticosteroids (prednisone, 60 to 100 mg, tapered over 2 weeks). Divers should avoid touching with bare skin anything resembling coral. For example, the underwater statue of Jesus at John Pennycamp Park in Key Largo, Florida, is encrusted with fire coral, so posing divers have been envenomed.

Physalia (Man-of-War)

The Atlantic Portuguese man-of-war *(Physalia physalis)* of the phylum Coelenterata, order Siphonophora, is a pelagic (open sea) polymorphic colonial siphonophore that inhabits the surface of the ocean. It is constructed of a blue or pink-violet and iridescent floating sail (pneumatophore) that is filled with nitrogen and carbon monoxide and up to 30 cm (11.8 in) in length, from which are suspended multiple nematocyst-bearing tentacles, which may measure up to 30 m (98 ft) in length (Fig. 73-25). It has recently been reported that an Australian version of *Physalia physalis* is present in northern Australian waters.[53] This jellyfish is characterized by float lengths of up to 15 cm (5.9 in), up to five thick dark blue "main" tentacles, and up to 10 other long, thin, and pale-colored tentacles. The smaller Pacific bluebottle *(Physalia utriculus)* usually has a single fishing tentacle, which attains lengths of up to 15 m (49.2 ft). In some species, the sail can be deflated to allow the animal to submerge in rough weather.

The physaliae depend on the winds, currents, and tides for movement, traveling as individuals or in floating colonies that resemble flotillas. They are widely distributed but seem to abound in tropical waters and in the semitropical Atlantic Ocean, particularly off the coast of Florida and in the Gulf of Mexico. Envenoming has been reported as far south as the coast of Brazil.[42] Their arrival at surf's edge can transform a halcyon vacation into a stinging nightmare. Unfortunately, peak appearance time for both the man-of-war and sea nettle is July through September, which is prime beach season.

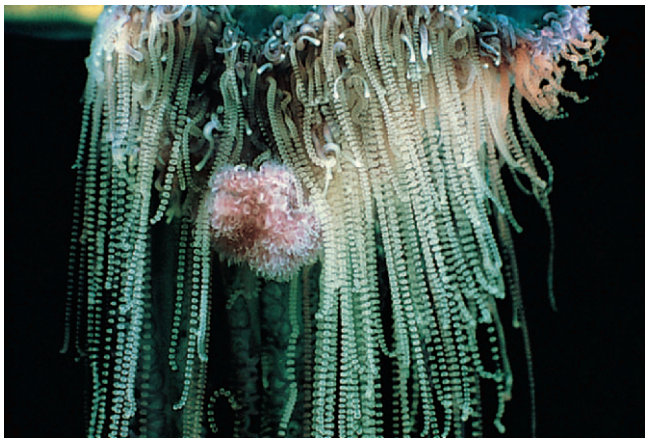


Figure 73-26. Tentacles of the Atlantic Portuguese man-of-war. Nematocysts may number in the hundreds of thousands on tentacles coiled into "stinging batteries." (Courtesy Larry Madin, Woods Hole Oceanographic Institution.)

As is the case for icebergs, much of the story is below the water surface. Because the tentacles are nearly transparent, they pose a hazard to the unwary. As the animal moves in the ocean, the tentacles rhythmically contract, sampling the water for potential prey. If the tentacle strikes a foreign object, the nematocysts are stimulated and discharge their contents into the victim. Each tentacle in a larger specimen may carry more than 750,000 nematocysts. To increase the intensity of the "attack," the remainder of the tentacle shortens in such a way as to create loops and folds, presenting a greater surface area and a greater number of nematocysts for offensive action in "stinging batteries" (Fig. 73-26).

Detached moistened tentacles, often found by the thousands fragmented on the beach, carry live nematocysts capable of discharging for months. Air-dried nematocysts may retain considerable potency, even after weeks (Fig. 73-27). The loggerhead turtle *(Caretta caretta)* (Fig. 73-28) feeds on *Physalia*. Like the clownfish with the sea anemone, the brightly colored fish *Nomeus gronovii* has a unique symbiotic relationship with the man-of-war, living freely among the tentacles. Two species of nudibranch (sea slug), *Glaucus atlanticus* and *Glaucus glaucilla,* eat the tentacles and nematocysts of *P. physalis*. The nematocysts are not digested and ultimately reside in the dorsal papillae of the nudibranchs, where they may sting on contact. Other nudibranchs are also able to ingest hydroids and store their stinging cells in the cerata, or flesh appendages. Dermatitis can also result from contact with water containing venom that has already been released from stimulated nematocysts. The Mediterranean octopus *Tremoctopus violaceous* stores intact dactylozooid segments in its suckers for later use.[27]

Gonionemus Species

These small hydrozoans are distributed worldwide but have been reported as causing severe envenomation only in the Sea

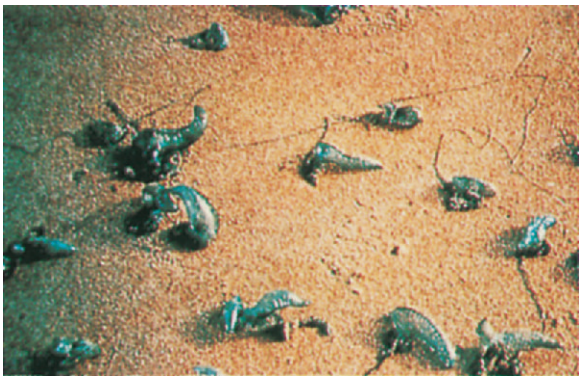


Figure 73-27. Pacific man-of-war washed ashore may retain stinging potency for weeks. (Courtesy John Williamson, MD.)

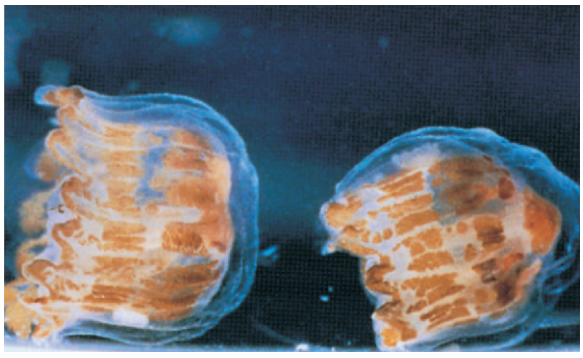


Figure 73-29. Mature *Linuche unguiculata,* the causative agents of seabather's eruption. The planula or larvae of these coelenterates were collected from plankton tows and grown to maturity at the University of Miami. Slightly smaller than their brethren found in the open ocean, these specimens are approximately 2 cm in diameter when open and 1 cm when contracted. (Courtesy David Taplin and Terri L. Meinking.)

Figure 73-28. The loggerhead turtle sometimes dines on jellyfish tentacles. (Photo by Howard Hall.)

of Japan near Vladivostok, Russia, and at the northwestern shores of Honshu Island, Japan.[48] It is a small creature of 5 to 15 mm in diameter across the bell, with a symmetric, right-angled cross visible in the transparent part.

When the reaction is painful, the victim suffers muscle, joint, chest, and pelvic pain for up to 3 days. There may be muscle fasciculations. In a respiratory presentation, the victims suffer rhinitis, tearing, hoarseness, cough, and shortness of breath. In addition, there may be a combination of symptoms, such as sore throat, tachycardia, vomiting, and mild hypertension. Psychiatric depression and hallucinations may occur.[124]

It has been noted that envenomation may occur beneath a bathing suit. In addition, a similar syndrome was reported after ingestion of raw seaweed, to which was presumably attached the jellyfish.[48]

Seabather's Eruption

Seabather's eruption, commonly termed sea lice (pika-pika around the Belize barrier reef; sea poisoning, sea critters, and ocean itch are other names), refers to a dermatitis that results from contact with ocean water.[82] It has become a seasonal problem afflicting oceangoers in southern Florida and across the Caribbean; it has been reported in Brazil and Papua New Guinea.[70,172] It predominantly involves covered areas of the body and is commonly caused by pinhead-sized (0.5 mm) greenish brown to black larvae of the thimble jellyfish *Linuche unguiculata* (Fig. 73-29), which breeds in Caribbean waters throughout the summer with a peak in May.[170] *L. unguiculata* exists in three swimming stages during its life cycle: planula (free-swimming larva), ephyra (immature medusa), and adult medusa. It is likely that all three swimming stages initiate the eruption.[135,147] Another culprit off Long Island, New York, has been the planula larval form (visible at 2 to 3 mm) of the sea anemone *Edwardsiella lineata,* which carries hundreds of nematocysts.[56,57] Given the number of coelenterates that inhabit the oceans of the world and the cross-reactivity of antigens, it is likely that etiologic organisms are numerous.

A swimmer who encounters the stinging forms usually complains of cutaneous discomfort (stinging, tingling, or a pins-and-needles sensation) after contact, often while in the water or soon after exiting. Application of freshwater may intensify the sting. The eruption occurs a few minutes to 12 hours after bathing and consists of erythematous and intensely pruritic wheals, vesicles, or papules that persist for 2 to 14 days and then involute spontaneously. When a bathing suit has been worn by a woman, the areas commonly involved include the buttocks, genital region, and breasts (Fig. 73-30). A person at the water's surface (commonly a person who surfaces after a dive) may suffer stings to the exposed neck (Fig. 73-31), particularly if there has been recent motorboat activity in the vicinity, which may disturb and fragment the causative jellyfish. Nematocysts adherent to scalp hair may sting the neck as the hair hangs down. Individual lesions resemble insect bites. Coalescence indicates a large inoculum. Surfers develop lesions on areas that contact the surfboard (chest and anterior abdomen). The rash may also be seen under bathing caps and swim fins or along the edge of the cuffs of wet suits, T-shirts, or stinger suits (Fig. 73-32).[170] In children with extensive eruptions, fever is common. Low-grade fever may be noted in adults.[172] Other symptoms may include headache, chills, fatigue and malaise, vomiting, conjunctivitis, and urethritis. Itching is often pronounced at night and awakens

Figure 73-30. Seabather's eruption. (From Wong DE, Meinking TL, Rosen LB, et al: J Am Acad Dermatol 30:399–406, 1994.)

Figure 73-31. Seabather's eruption on the neck of a diver in Cozumel, Mexico. (Photo by Paul Auerbach, MD.)

the victim from sleep. Burnett and Burnett[19] reported blurred vision and left arm weakness in a teenager stung by an adult *Linuche*. Persons who note a stinging sensation during the primary contact while still in the water may have a higher incidence of previous sensitization to the antigen or antigens. Persons who wear clothing that has been contaminated with the larvae may suffer recurrent reactions. Prior sensitization may precede prolonged (up to 6 weeks) reactions (rash and pruritus).

Elevated IgG levels specific for *Linuche unguiculata* can be measured by ELISA in the sera of victims who have suffered from seabather's eruption. The extent of the cutaneous eruption or sting severity appears to correlate with the antibody titer.[31]

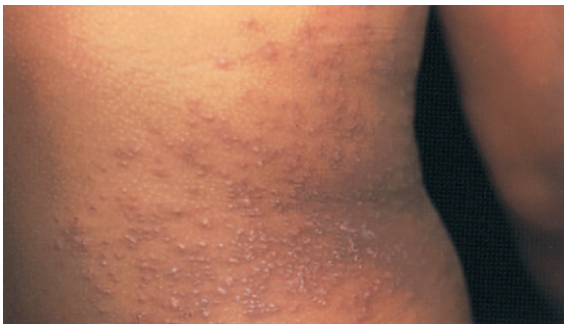


Figure 73-32. Seabather's eruption in an area under the weight belt. (Courtesy Doug Wong, MD.)

In an evaluation of southeastern Florida victims envenomed by *L. unguiculata,* histopathologic examination of inflammatory papules demonstrated superficial and deep perivascular and interstitial infiltrate consisting of lymphocytes, neutrophils, and eosinophils.[189]

Field management is identical to that for any coelenterate sting (see earlier), with the empirical observation that topical papain may be slightly more effective as an initial decontaminant than vinegar, isopropyl alcohol, or other substances. Papain application may be more effective if undertaken with a mildly abrasive scrub pad. Whether the pain relief is due to nematocyst inactivation or counterirritation is not yet known. Substances that are believed to be ineffective include hydrogen peroxide, garlic, antifungal spray, anti–head lice medication, petroleum distillates, fingernail polish, and citrus juice.

The skin eruption is self-limited and usually remits within 10 days. However, in a severe envenomation, the rash may persist for up to 4 weeks and leave atrophic scars.[102] Further treatment is palliative and consists of calamine lotion with 1% menthol. Because the lesions rarely extend into the dermis, a potent topical corticosteroid may be helpful in mild cases, but benefit is not invariably attained. In a more severe case, an oral or parenteral antihistamine or systemic corticosteroid may be used. A thorough soap and water scrub (not a casual rinse) on leaving the water provides partial prophylaxis. Avoidance logically includes advice to ocean bathe in abbreviated swimwear (which may, however, expose a person to other stings), to maintain tightly occlusive cuffs on dive skins and wet suits, to change swimwear as soon as possible after leaving the water, and to use caution during high season for *L. unguiculata* (April to July off southern Florida) or *E. lineata* (August to November off Long Island) and when there are strong onshore winds. Swimwear worn and suspected to be contaminated with nematocysts should be washed in detergent and freshwater and dried before wearing.[146]

True sea lice (see Chapter 75) are parasites on marine creatures and do not cause this disorder.

Class Scyphozoa

The class Scyphozoa comprises the larger medusae or jellyfish, including the deadly box-jellyfish and sea wasps (e.g., *Chironex*, *Cyanea,* and *Chiropsalmus*). These creatures are armed with some of the most potent venoms in existence. Jellyfish are mostly free-swimming pelagic creatures; however, some can be found at depths of more than 2000 fathoms. They may be trans-

Figure 73-33. The author snorkels in Jellyfish Lake in Palau, Micronesia. The jellyfish have evolved to subsist on algae and thus no longer pose a stinging hazard to humans. (Photo by Avi Klapfer.)

parent or multicolored and range in size from a few millimeters to more than 2 m ($6^1/_2$ ft) in width across the bell, with tentacles up to 40 m (131 ft) in length. Like physaliae, the scyphozoans depend on the wind, currents, and tides for transport and are widely distributed. Some vertical motion may be produced by rhythmic contractions of the gelatinous bell, from which originate the feeding tentacles.

Some jellyfish contain less than 5% solid organic matter. Regardless, they can withstand remarkable temperature and salinity variations, although they do not fare well with violent activity and thus may descend to great depths during stormy surface weather. Some scyphozoans avoid sunlight; others follow an opposite pattern. Certain jellyfish have adapted to local nutrient (largely algal) supply and have lost their ability to sting humans (Fig. 73-33).

In eastern coastal waters of the North American continent, the creatures appear to grow larger as they progress north (Fig. 73-34), so that true giant jellyfish, typified by *Cyanea capillata* (lion's mane), are found in Arctic waters (Fig. 73-35). Tentacles (which may number up to 1200) of larger specimens may exceed 30 m (100 ft) in length.[27] *Pelagia* species (purple-striped or mauve stingers) are commonly found in large numbers off the California coast and appear in the Mediterranean Sea in abundance every 10 to 12 years.[136] *P. noctiluca* (Fig. 73-36) phosphoresces at night, hence its name.[114] *Olindias sambaquiensis* is a jellyfish that stings bathers in South American coastal waters. *Rhopilema nomadica* is a tropical jellyfish that has invaded the eastern Mediterranean.[51,98,100] As a further example, stings from *Stomolophus nomurai* in the Bohai waters of China produce severe pulmonary edema, coma, convulsions, psychoses, and death. Australian jellyfish include the blubber jellyfish (*Catostylus* species), hair jellyfish (*Cyanea* species), little mauve stinger *(Pelagia noctiluca),* and the cuboid-shaped jellyfish (*Chironex fleckeri* and *Chiropsalmus quadrigatus*). A number of cubomedusan ("box-shaped jellyfish") scyphozoans of highly toxic nature inhabit Indo-Pacific and, less frequently, Caribbean waters. These include *Carybdea rastoni* (jimble) (Fig. 73-37) and *Carybdea marsupialis* (sea wasp), *Chiropsalmus quadrumanus* (box-jellyfish or sea wasp), *Chiropsalmus quadrigatus* (sea wasp), and *Chironex fleckeri* (box-jellyfish). The carybdeids

Figure 73-34. Lion's mane jellyfish *(Cyanea capillata).* (Photo by Carl Roessler.)

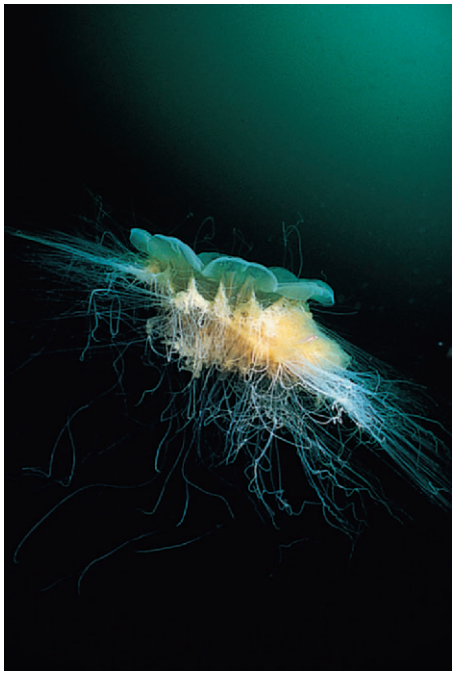


Figure 73-35. Lion's mane jellyfish *(Cyanea capillata)* can reach 3 m (10 ft) in diameter in Arctic waters. (From Norbert Wu, with permission; available at www.norbertwu.com.)

Figure 73-36. Mauve stinger *(Pelagia noctiluca).* (Courtesy Larry Madin, Woods Hole Oceanographic Institution.)

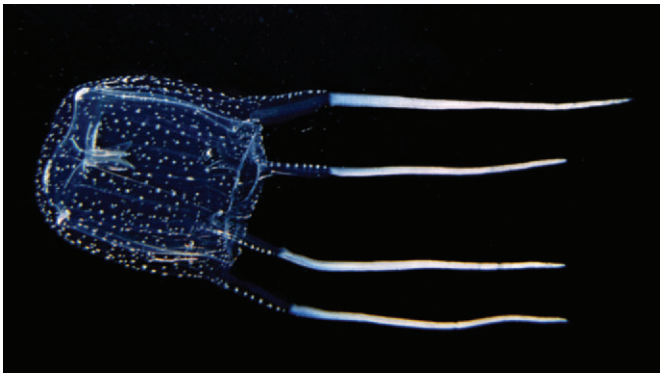


Figure 73-37. Jimble box-jellyfish or southern sea wasp *(Carybdea rastoni),* in Southern Australia. (From Gary Bell; available at www.oceanwideimages.com.)

of the order Carybdeidae have four tentacles only, whereas the chirodropids of the order Chirodropidae may have up to 60 tentacles. All are frequently called box-jellyfish.

Chironex (Box-Jellyfish)

The dreaded chirodropid box-jellyfish (*Chironex fleckeri* Southcott), often misnamed the sea wasp, is a most venomous sea creature that can induce death in less than 60 seconds with its potent sting. Like all other scyphozoans, it is a carnivore, adapted to deal rapidly with prey. A member of the group of Cubomedusae jellyfish, it ranges in size from 2 to 30 cm across

Figure 73-38. Box-jellyfish *(Chironex fleckeri),* swimming just beneath the surface of the water. (Courtesy John Williamson, MD.)

the bell. Although these creatures seem to prefer quiet, protected, and shallow areas, chiefly in the waters off northern Queensland, Australia, they can be found in the open ocean. A seasonal alternation of polypoid and medusoid generations from winter to summer, respectively, appears to account for the shift in preferred habitat from tidal estuaries to the open eulittoral zone.[76] Stinger season in the Northern Territory of Australia is from October 1 to May 31.[39] Swimming and bathing are precluded in the littoral and estuarine waters of Indonesia, Malaysia, and Northern Australia during this season, which coincides with the hottest tropical months in the Southern Hemisphere.[129] However, it is likely that *Chironex* may be present year-round in the Northern Territory.[50] *Chironex* are fragile and photosensitive and thus are found submerged during bright sunlight hours (Fig. 73-38), seeking the surface in the early morning and late afternoon and evening. They are swift and graceful travelers, capable of sailing along at a steady 2 knots.

An adult *Chironex* carries up to 15 broad tentacles (Fig. 73-39) in each corner (pedalium, or foot) of its bell (up to 60 tentacles total, each with a length of up to 3 m [10 ft]) and has enough venom (in excess of 10 mL) to kill three adults.[40,159] As *Chironex* grows in size, the ratio of mastigophores (nematocysts believed to hold the lethal venom component for prey) to less injurious organelles increases.[35] Two fractions have been isolated from the venom: a "lethal" fraction of molecular weight 150,000, and a lethal-hemolytic-dermatonecrotic fraction of molecular weight 79,000. At least 72 fatalities have been verified in Australian and Southeast Asian waters, with greater numbers probably lacking official documentation. Thus the box-jellyfish is a much greater true hazard than the more fearsome shark. Other jellyfish, such as *Carybdea rastoni* and

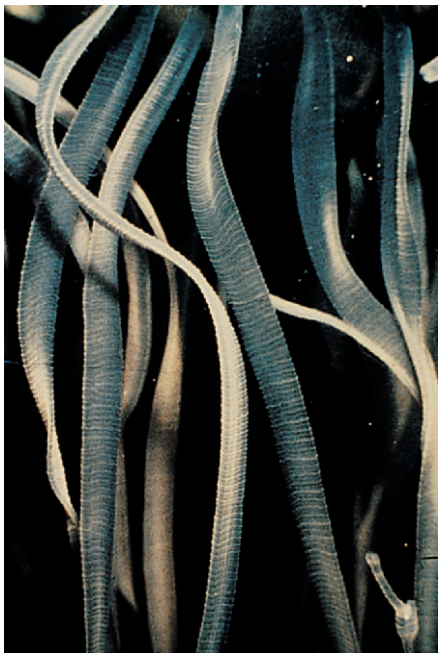


Figure 73-39. Close-up of the tentacle mass of an adult box-jellyfish *(Chironex fleckeri)*. (Courtesy Bob Hartwick.)

Pelagia noctiluca, infrequently cause severe prolonged reactions and have rarely been reported to lead to death, but they are capable of causing dramatic immediate reactions. Sudden death in a child has followed envenomation by *Chiropsalmus quadrumanus* in the Gulf of Mexico at Crystal Beach, Texas.[14] Death was attributed to acute arrhythmia after a catecholamine surge, followed by cardiogenic shock and pulmonary edema.

Clinical Aspects. The extreme example of envenomation occurs with the chirodropid *Chironex* ("the assassin's hand") *fleckeri* (after Dr. Hugo Flecker).[129] Death is attributed to hypotension, profound muscle spasm, muscular and respiratory paralysis, and subsequent cardiac arrest. The overall mortality after box-jellyfish stings may approach 15% to 20% in selected locales. Most commonly, bathers are stung—frequently, aboriginal children in shallow and remote coastal waters who do not recognize the small, semitransparent, and submerged creature, which may approach as a member of a small armada. Most stings are minor; severe reaction or death follows skin contact with tentacles longer than 6 or 7 m (20 to 23 ft), although 10 cm of tentacle is capable of delivering a lethal dose of venom.[129,160] The sting is immediately excruciatingly painful, and the victim usually struggles purposefully for only a minute or two before collapse. The toxic skin reaction may be intense, with rapid formation of wheals, vesicles, and a darkened reddish brown or purple whiplike flare pattern with stripes 8 to 10 mm in width (Fig. 73-40). With major stings, skin blistering occurs within 6 hours, with superficial necrosis in 12 to 18 hours (Fig. 73-41). The skin defects that result from a severe envenomation can be profound (Fig. 73-42). On occasion, a pathognomonic frosted appearance with a transverse crosshatched pattern has been observed (Fig. 73-43). This appear-

A

B

Figure 73-40. Intense necrosis (here, at 48 hours) is typical of a severe box-jellyfish *(Chironex fleckeri)* sting. **A,** Involvement of nearly an entire limb. **B,** Skin darkening can be rapid with cellular death. (Courtesy John Williamson, MD.)

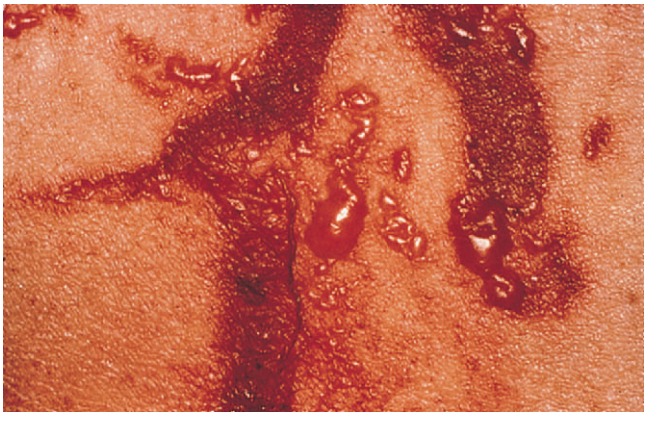
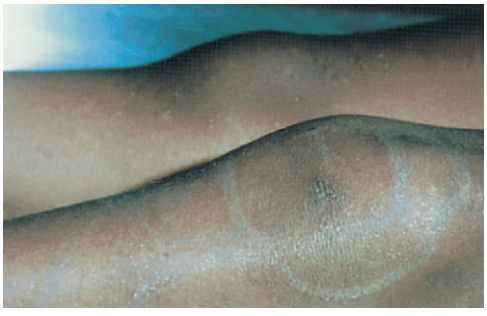


Figure 73-41. Incipient necrosis and blistering within 24 hours of box-jellyfish *(Chironex fleckeri)* envenomation. (Courtesy John Williamson, MD.)

Figure 73-42. Skin destruction 3 weeks after an untreated box-jellyfish *(Chironex fleckeri)* envenomation. (Courtesy John Williamson, MD.)



A

B

Figure 73-43. Frosted cross-hatched pattern pathognomonic for a box-jellyfish envenomation. **A,** The victim of this sting expired rapidly. **B,** The enhanced frosted appearance is a result of application of a spray of aluminum sulfate. (Courtesy John Williamson, MD.)

ance may be primarily the result of the application of aluminum salts used for decontamination. More severe reactions and increased mortality in women and small children have been attributed to their greater hairless body surface area and smaller body mass.

One case of *Chironex* envenomation in a pregnant woman has been reported.[92,182] A 20-year-old woman in the 34th week of pregnancy suffered apparent respiratory arrest but was successfully revived with rescue breathing at the scene. The victim received antivenom in the hospital and 9 weeks later delivered a healthy child by caesarean section. It is interesting to note that one rescuer was 37 weeks pregnant and received a sting from tentacles adherent to the victim, but she also delivered uneventfully.

Identification of *Chironex* envenomation is sometimes possible by nematocyst recovery from the skin. This can be done by scraping with a scalpel or by applying sticky tape. In the former technique, the skin is firmly scraped with a sterile scalpel blade, which is then placed in a container. Five to 10 mL of distilled water is added and the container is sonicated for 5 minutes to remove any nematocysts adherent to the blade. The solution is then syringed through a 13-mm Millipore filter, which leaves the nematocysts, debris, and skin cells on the paper. A 0.5% eosin stain is then syringed through the filter paper, which is then allowed to dry, after which it is placed on a glass slide, fixed and mounted with a cover slip. In the sticky tape technique, transparent household sticky tape is applied to the sting site, stroked several times to ensure adherence, and then removed and placed sticky side up on a glass slide, with the ends secured to the slide with additional tape.[41]

Treatment. In the case of a known or suspected box-jellyfish envenomation, the victim must be assessed rapidly for adequacy of breathing and supported with an airway and artificial ventilation if necessary. The victim should be moved as little as possible. It is essential to immediately and liberally flood, for a minimum of 30 seconds, the skin surrounding any adherent tentacles with 5% acetic acid (vinegar) before any attempt is made to remove them; this paralyzes the nematocysts and avoids worsening the envenomation (Fig. 73-44). Significant pain relief should not be expected from this maneuver, which may actually worsen the pain briefly.[13] Although most nematocysts cannot penetrate the thickened skin of the human palm, the rescuer should pay particular attention to his or her own skin protection. If acetic acid is not available, aluminum sulfate surfactant (Stingose) may be substituted, although its efficacy has not been well demonstrated for a *Chironex* envenomation. A number of experts recommend that isopropyl alcohol *not* be used as a topical decontaminant for a box-jellyfish envenomation, based on in vitro observations of inefficacy and nematocyst discharge after application of this detoxicant.[75,160] Clinical confirmation of this recommendation has not been published.

Pressure-immobilization is no longer uniformly recommended to prevent absorption of *Chironex* venom.[95] Some experts have questioned its efficacy and noted that large affected skin surfaces cannot be effectively bandaged. Others have noted that application of pressure might promote nematocyst discharge, which is believed to be more harmful than foregoing any attempt to devascularize the area immediately below the bandage in order to prevent distribution of venom into the general circulation.[8,132,148,159,184,185] In any event, it is reasonable to splint or otherwise immobilize the limb to prevent motion.

In the absence of antivenom and facing a prolonged transport prior to supportive intensive care, a rescuer might apply a constriction bandage proximal to the site of an extremity sting, to impede lymphatic and superficial venous return. Such a bandage should be loosened for 90 seconds every 10 minutes and should be completely removed after 1 hour. In no case

Figure 73-44. Surf lifesavers pour vinegar on the leg of a simulated box-jellyfish envenomation. Note how they restrain the victim's arms to prevent him from handling the harmful tentacles. (Courtesy John Williamson, MD.)

Figure 73-45. Box-jellyfish antivenom. (Courtesy John Williamson, MD.)

should an arterial tourniquet be applied. Use of a proximal constriction band has not been proven to be helpful.

Chironex antivenom should be administered intravenously as soon as possible. The IM route is less preferred, because peak blood levels may not be obtained for 48 hours after administration by this route. One author has recommended consideration of intraosseous administration if the IV route is not available.[40] The antivenom is supplied in vials (1.5 to 4 mL of liquid) containing 20,000 units by CSL (Fig. 73-45). The initial dose is one vial (diluted 1 : 5 to 1 : 10 in isotonic crystalloid; dilution with water is not recommended) administered intravenously over 5 minutes, or three vials into three different sites (generally on the thigh) intramuscularly. IM antivenom has been administered successfully over the years by members of the Queensland Surf Life-Saving Association and the Queensland Ambulance Transport Brigade.[52] Although the antivenom is prepared by hyperimmunizing sheep and adverse reactions reported have been rare and mild, the prudent physician is always prepared to treat anaphylaxis or serum sickness.[39] It cannot be overemphasized that timely administration of antivenom might be lifesaving, particularly in light of the fact that most deaths from *Chironex* stings occur in the first 5 to 20 minutes.[182] In addition to its lifesaving properties, early administration of antivenom may markedly reduce pain and decrease subsequent skin scarring.[183] Antivenom administration may be repeated once or twice every 2 to 4 hours until there is no further worsening of the skin discoloration, pain, or systemic effects. A large sting in an adult may require an initial IV administration of up to three vials. The antivenom may also be used to neutralize the effects of a *Chiropsalmus* envenomation.[159] Antivenom should be stored in a refrigerator at 2° to 10° C (35.6° to 50° F) and must not be frozen.[27] Concomitant administration of a glucocorticoid (such as hydrocortisone 200 mg intravenously) is often recommended for its anti-inflammatory activity but is no substitute for administration of antivenom.

If a sting is mild (not life-threatening), one may use nonmoist ice packs for initial pain relief, along with a parenteral analgesic.

Even with successful treatment, skin irritation may persist for months, marked by discolored striae, intermittent desquamation, and pruritus. Burnett and Calton[16,23,32] discovered that verapamil can prolong the life of mice challenged with box-jellyfish, sea nettle, or Portuguese man-of-war venom. Verapamil was considered to be inactive or deleterious in anaesthetized laboratory pigs envenomed with box-jellyfish venom.[169] Extrapolation of these data to humans is as yet untested. Although there is logic to using verapamil from a theoretical pharmacologic perspective (venom affects calcium influx through voltage-dependent channels), the suitability of using verapamil as an adjunct to therapy in humans has been questioned because of the perceived problem of administering a

hypotensive agent during an episode of cardiac decompensation.[78] Type IV hypersensitivity reactions may occur more commonly than previously thought with *Chironex* stings, perhaps attributable to retained foreign material.[127]

Irukandji

Carukia barnesii, the carybdeid jellyfish known as Irukandji, is a small (1 to 2.5 cm across the bell) translucent jellyfish with four thin nematocyst-covered tentacles (5 to 7 cm in length at rest, and up to 70 cm (27.5 inches) extended) found off the coast of northern Australia in both inshore and open waters.[11,118,155] Most stings occur near shore and during the afternoon.[6,129] Because the jellyfish tend to aggregate, victims often present in clusters. Furthermore, victims can be stung inside stinger-resistant enclosures, even when the mesh is as small as 2 cm diagonally.[47] After causing a severe immediate skin reaction characterized by pain and erythema without wheal formation, the venom may induce restlessness, muscle pain and spasm, severe lumbosacral back pain, lower leg pain, priapism, abdominal pain, parasympathetic dysautonomia, respiratory difficulty (including painful breathing), headache, shivering, tremor, nausea, and vomiting, which progress to profound weakness and collapse. Localized piloerection and sweating have been reported to occur commonly. Generally, the discomfort remits in 6 to 24 hours; however, it occasionally recurs. The Irukandji syndrome (named by Hugo Flecker for an Aboriginal tribe in the Cairns region of Australia) presupposes massive catecholamine release, with abdominal and chest pain, a sensation of chest tightness, pallor or peripheral cyanosis, vomiting, diaphoresis, hypertension (diastolic blood pressure to 140 mm Hg), oliguria, tachycardia, severe pulmonary edema, cerebral edema, and hypokinetic heart failure.[40,54,106] This resembles what might be seen with a pheochromocytoma. Papilledema and coma in a child have been described.[50a] Although the systemic syndrome can be quite distinctive, there may be minimal cutaneous signs of envenomation.[72] At least two deaths have been reported, both attributed to intracerebral hemorrhage associated with hypertension.[49]

It is interesting to note that many Irukandji-like stings occur inside stinger enclosures (bathing nets) designed to exclude *Chironex fleckeri.* Although residents of Irukandji-endemic areas are often aware that stinger-resistant enclosures do not prevent entry of the smaller jellyfish, many tourists, particularly those from countries other than Australia, are not aware.[74]

Other carybdeid medusae that envenom with lesser severity include the jimble *(Carybdea rastoni)* and fire jelly *(Tamoya haplonema).* The morbakka is a stinging creature that resembles the Irukandji but is larger. Its bell, which measures up to 12 by 16 cm, is covered with clumps of nematocysts and may be as dangerous to handle as the meter-long tentacles. This animal may have been previously misidentified as *Tamoya.* An Irukandji-like syndrome has been reported in South Florida divers, but the jellyfish species was not identified.[66]

Treatment. In addition to the standard coelenterate measures, IV phentolamine (5 mg initially, followed by 5- to 10-mg doses as needed) may be administered to control high blood pressure. However, because acute cardiac failure may be a feature of envenomation, administration of an alpha-adrenergic blocking drug should be undertaken with close cardiovascular monitoring and perhaps preliminary echocardiography, supplemented as needed by pulmonary artery catheterization.[97] Administration of box-jellyfish antivenom does not significantly relieve symptoms. Propranolol or other beta-adrenergic blockers should not be used to control tremor, as these might precipitate catastrophic hypotension, or might contribute to unopposed alpha-adrenergic stimulation predisposing to myocardial ischemia.[47,79] Fentanyl has been suggested for pain relief because it does not cause cardiac depression.[96] One report noted resolution of agitation and sympathetic features, and significant resolution of pain, in a victim of Irukandji syndrome treated with magnesium sulfate (loading dose 10 mmol [approximately 2.5 g or 20 mEq], followed by an infusion of 5 mmol/h).[38]

Chrysaora (Sea Nettle)

Sea nettles (such as *Chrysaora quinquecirrha* and *Cyanea capillata*) are considerably less lethal animals and can be found in both temperate and tropical waters, particularly in Chesapeake Bay, where they are found in seasonal plague proportions.[114] Not as dangerous as the Indo-Pacific box-jellyfish, they are still capable of inducing a moderately severe sting. *Chrysaora quinquecirrha* and similar species carry a proteinaceous venom that contains at least seven enzymes, with at least one antigenic and thermolabile component that is cardiotoxic, neurotoxic, and dermatonecrotic. The venom also contains histamine, histamine releasers, prostaglandins, serotonin, and kininlike factors (kininlike factors have also been found in venoms of *Chironex fleckeri* and *Physalia physalis*).[21] Large intradermal injections of crude sea nettle venom in normal saline produced immunosuppression (T cells) for several days, with a homologous reaction against the same coelenterate antigen and a heterologous reaction against antigens contained within vaccinia and herpes simplex viruses and tetanus bacillus.[174]

Clinical Aspects. The clinical presentation of a sea nettle envenomation is similar to that of *Physalia* species, with perhaps a greater incidence of systemic complications. Death is exceedingly rare. Elevated levels of serum anti–sea nettle venom IgM, IgG, and IgE may persist for years in victims who suffer exaggerated reactions to *Chrysaora quinquecirrha* stings. These antibodies cross-react with *Physalia* venom and have been postulated to be of value in identifying victims at risk for a severe reaction.[22] This technique is not widely available or frequently used, and its reliability and reproducibility require further verification.

The reaction after a sting by the blubber jellyfish (*Catostylus* species) is relatively mild, with the formation of wheals, erythema, and pruritus limited to the areas of contact. Systemic effects are exceedingly rare. *Cyanea* species carry long thin tentacles that induce a similar effect, with occasional muscle aching, nausea, and drowsiness, particularly in small children. *Pelagia* species also induce wheals, which are more circinate or irregularly shaped and may not follow a linear pattern. The venom is sufficiently toxic to cause severe generalized allergy, with bronchospasm and pruritus.

Treatment. Treatment for a sea nettle envenomation is similar to that for the sting of *Physalia* species. Baking soda may be the most effective commonly available initial detoxicant. Monoclonal antibodies to jellyfish venoms have been developed that demonstrate cross-reactivity among venoms of a variety of coelenterates, which may allow the development of a single protective antivenom or vaccine.

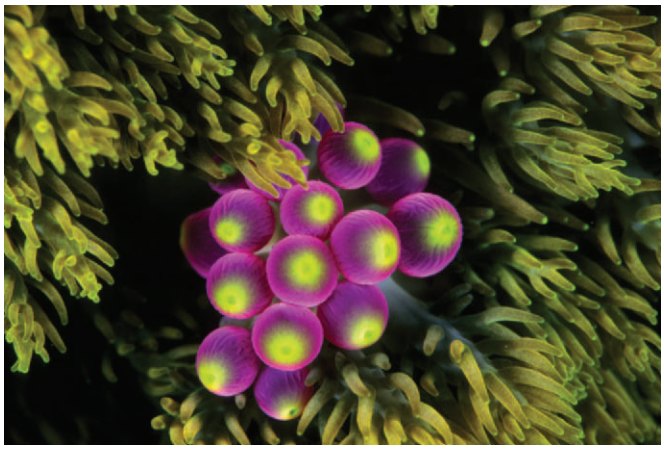


Figure 73-46. Detail of grape-like vesicles of sea anemone (*Actinaria* species). (From Gary Bell; available at www.oceanwideimages.com.)

Class Anthozoa

The class Anthozoa includes the sea anemones, stony (true) corals (subclass Zoantharia), and the soft corals (subclass Alcyonaria). The anemones are considered here because they envenom.

Actinaria (Anemones)

Actinarians (sea anemones) are abundant (1000 species) multicolored animals with sessile habits and a flowerlike appearance (Fig. 73-46). They are composed of stalked, fingerlike projections capable of stinging and paralyzing passing fish. Their sizes range from a few millimeters to more than 0.5 m (1.7 ft); they are found at depths of up to 2900 fathoms (5303 m). The insides of some anemones can be eaten after they are dried.

Anemones can be colorful creatures and may be found in tidal pools, where the unwary brush up against them or inquisitively touch them. Other anemones burrow into bottom mud or sand. Like other coelenterates, they possess tentacles loaded with one of two variations of the nematocyst, either the sporocyst or the basitrichous isorhiza (basitrich). These wreak havoc once stimulated by an unfortunate victim. Some sporocysts are adhesive and act to hold and to envenom prey. To present a greater number of nematocysts to the victim, an exposed anemone inflates the tentacles by filling them with water. Many anemones also secrete mucus, which covers the anemone's body and may contain cytolytic and hemolytic protein toxins. These may serve to repel potential predators.

Although a number of sea animals, such as clownfish (anemonefish) of the genera *Amphiprion* and *Premnas,* live in symbiosis with certain anemones (*Heteractis* species, *Stichodactyla* species, *Macrodactyla doreensis, Entacmaea quadricolor,* and *Cryptodendrum adhaesivum*), humans are not so fortunate and are frequently stung when attempting to handle these not so delicate "flowers." The clownfish have evolved resistance to the anemone's sting by repeated contact and development of a mucous coat (Fig. 73-47), and perhaps by immunity.[112]

Sea anemones contain biologically active substances, including neurotoxins (sodium channel interaction), cardiotoxins, hemolysins (for erythrocytes and platelets), and proteinase inhibitors.[113,151] A cytolytic toxin has been isolated from the Indo-Pacific sea anemone *Stoichactis kenti.*[15] The anemone *Actinia equina* elaborates cytolytic polypeptide toxins known as equinatoxins, which may induce hemolysis and cardiorespiratory arrest in animals, attributed by some to coronary vasospasm.[101] Tenebrosin-C from the anemone *Actinia tenebrosa* is a positive inotrope that can be inhibited by the cyclooxygenase blockers indomethacin and aspirin, a lipooxygenase blocker and leukotriene antagonist, and mepacrine (a phospholipase A_2 inhibitor).[59] Potassium channel toxins have been isolated from the sea anemones *Bunodosoma granulifera* and *Stichodactyla helianthus.*[77] Cytolytic toxins elaborated by anemones include cytolysins, which are thought to exert their effect by damaging membranes via pore or channel formation.

Figure 73-47. Clownfish in peaceful coexistence with a sea anemone. (Photo by Kenneth Kizer, MD.)

Clinical Aspects. Most victims are stung when they handle or accidentally brush against an anemone in shallow water. Nudists may acquire genital injuries; small children may accidentally or intentionally ingest tentacles. The dermatitis caused by contact with an anemone is similar in all regards to that from fire coral or a small man-of-war; it is often likened to a bee sting. The variation in skin reaction is related to the specific toxicity of the venom, so that although *Actinia* species produce painful urticarial lesions, *Anemonia* species induce paresthesias, edema, and erythema. Most commonly, the initial skin lesion is centrally pale with a halo of erythema and petechial hemorrhage. This is soon followed by edema and diffuse ecchymosis. If the envenomation is severe, intense local hemorrhage, vesiculation, necrosis, skin ulceration, and secondary infection may occur, particularly after the stings of certain species (*Sagartia, Actinia, Anemonia, Actinodendron,* and *Triactis*). In Floridian waters, the turtle grass anemone *Viatrix globulifera,* translucent-white and less than 1 inch (2.5 cm) in diameter, is very hazardous, particularly for fishermen wading on grass flats. The Hell's fire sea anemone *(Actinodendron plumosum)* is aptly named. Systemic reactions are less likely after the sting of an anemone than after that of a man-of-war; reactions include fever, chills, somnolence, malaise, weakness, nausea, vomiting, and syncope. Fulminant fatal hepatic failure 3 days after a sea anemone sting of approximately 3 cm (just greater than 1 inch) in diameter on the scapula and complicated by coma, severe coagulopathy, and renal failure has been attributed to *Condylactis* (Fig. 73-48) (commonly found in reefs and lagoons of south Florida, the Bahamas, and the Caribbean) on the basis of a positive serum test of IgG by ELISA at a dilution of 1:450.[60]

In most cases, mild envenomations resolve within 48 hours. More severe reactions, characterized by discoloration and

Figure 73-48. Giant anemone *(Condylactis gigantea).* (From Norbert Wu, with permission; available at www.norbertwu.com.)

vesicle formation, may become indolent, with eschar leading to residual hyperpigmentation, hypopigmentation, or keloid formation.

Sponge fisherman's (diver's) disease is caused by contact with an anemone (*Sagartia* or *Actinia*) that attaches itself symbiotically to the base of a sponge. A few minutes after contact with the sponge, the victim's skin begins to itch and burn, with development of erythema and small vesicles. As described previously, this transforms to a darkened purple appearance, with frequent systemic components (headache, nausea, vomiting, fever, chills, and muscle spasm).

Treatment. Treatment for an anemone envenomation is similar to that for the sting of *Physalia* species. The dermatitis is frequently more severe and may require prolonged wound care consisting of debridement and antibiotic therapy for secondary infection. The healing process is generally slower after an anemone sting than after a man-of-war envenomation.

PHYLUM ECHINODERMATA

The phylum Echinodermata ("spiny skin") has five classes: sea lilies, brittle stars, starfish, sea urchins, and sea cucumbers. Only the last three are of medical interest in humans, although some brittle stars carry toxins capable of causing paralysis and death in small animals.

Starfish

Life and Habits. Starfish are simple, free-living, and stellate echinoderms covered with thorny spines of calcium carbonate crystals held erect by muscle tissue. The creatures move on the ocean floor by means of tube feet located under the arms (rays). They eat other echinoderms, mollusks, coral, worms, and poisonous shellfish. Starfish proliferation and the destruction of coral beds within the Great Barrier Reef off the coast of Australia is a conservation issue of international concern. The starfish everts its membranous stomach through its mouth and secretes digestive enzymes that destroy coral polyps. Only the stark white coral skeleton remains. The crown-of-thorns starfish *(Acanthaster planci)* is found in the coral reef communities of the Great Barrier Reef, throughout the Pacific and Indian Oceans, in the Red Sea, and in the Gulf of California.

Figure 73-49. Crown-of-thorns starfish *(Acanthaster planci).* (Photo by Paul Auerbach, MD.)

Venom and Venom Apparatus. Glandular tissue interspersed in or lying underneath the epidermis (integument) produces a slimy venomous substance. The carnivorous *Acanthaster planci* is a particularly venomous species, normally 25 to 35 cm (10 to 14 in.) in diameter but up to 70 cm (27.5 in.) in diameter, with 7 to 23 arms (Fig. 73-49). The sharp, rigid, and venomous aboral spines of this animal may grow to 4 to 6 cm (1.6 to 2.4 in.) (Fig. 73-50). Potentially toxic saponins and histamine-like compounds have been isolated from the spine surfaces; crude venom extracts demonstrate hemolytic, capillary permeability–increasing, myotoxic (via phospholipases A_2-I and -II), myonecrotic, and anticoagulant effects. The *A. planci* lethal factor is a potent hepatotoxin in laboratory animals.[150,152] Plancinin is an anticoagulant purified from the crown-of-thorns starfish. This peptide shows activity in mice that suggests a longer duration of action than heparin.[85] Severe systemic hypotension, thrombocytopenia, and leukopenia were induced by *A. planci* venom in dogs.[153] Indomethacin, a cyclooxygenase inhibitor, suppressed the hypotension. *A. planci* venom caused smooth (uterine) muscle contraction in rats, which was blocked by inhibitors of prostaglandin synthesis but not by atropine.[86]

Other starfish that might envenom humans are those of the genus *Echinaster.* The slime (cushion) star *Pteraster tessalatus,* which inhabits Pacific coastal waters from Puget Sound to Alaska, generates the unique defense of copious gelatinous or rubbery, poisonous mucus to repel natural enemies. No human injuries have been reported to date.

Clinical Aspects. The ice pick–like spine of *A. planci* can penetrate the hardiest of diving gloves. Most spines are composed

Figure 73-50. Spines of the crown-of-thorns starfish *(Acanthaster planci)*. (Photo by Paul Auerbach, MD.)

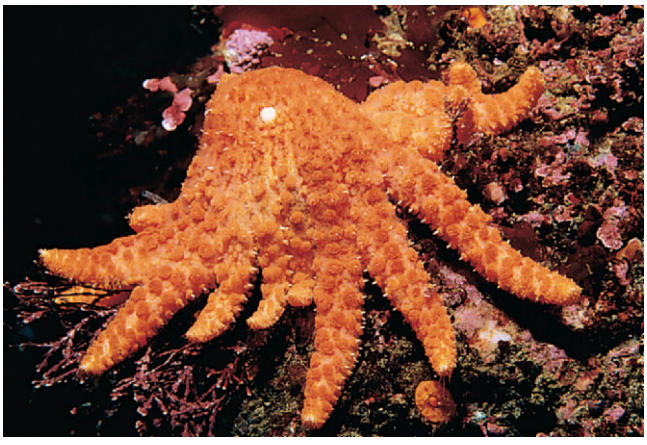


Figure 73-51. Sun starfish *(Solaster)*. (From Norbert Wu, with permission; available at www.norbertwu.com.)

of porous crystalline magnesium calcite, articulated at the base and extremely sharp, with three raised cutting edges at the tips. As the spine enters the skin, it carries venom into the wound, with immediate pain, copious bleeding, and mild edema. The pain is generally moderate and self-limited, with remission over a period of 30 minutes to 3 hours. The wound may become dusky or discolored. Multiple puncture wounds may result in acute systemic reactions, including paresthesias, nausea, vomiting, lymphadenopathy, and muscular paralysis. If a spine fragment is retained, a granulomatous lesion may develop akin to that seen after a sea urchin puncture wound. A previously sensitized victim may suffer a prolonged reaction lasting for weeks and consisting of local edema and pruritus. Tenosynovitis may affect multiple fingers simultaneously after a single puncture wound.[3] Contact with other, less injurious starfish may induce a pruritic papulourticarial eruption (irritant contact dermatitis).

Treatment. Immersion therapy may provide some relief from the pain. The wound should immediately be immersed into nonscalding hot water to tolerance (45°C [113°F]) for 30 to 90 minutes or until there is significant pain relief. The pain is rarely severe enough to require local anesthetic infiltration. The puncture wound should be irrigated and explored to remove all foreign material. Because of the stout nature of the spines, it is rare to retain a fragment. However, if any question of a foreign body exists, a soft tissue radiograph often identifies the fractured spine. Not infrequently, the victim suffers an indolent contact dermatitis from handling a starfish such as *Solaster papposus,* the sun (Fig. 73-51) or rose star. The dermatitis may be managed in standard fashion with topical solutions, such as calamine with 0.5% menthol, or a corticosteroid preparation. Systemic therapy is supportive. Granulomas from retained spine fragments may require excision. Starfish that have ingested poisonous shellfish are themselves toxic on ingestion.

Sea Urchins

Life and Habits. Sea urchins are free-living echinoderms that have an egg-shaped, globular, or flattened body. A hard skeleton (test) composed of fused calcareous plates surrounds the viscera and is covered by regularly arranged spines and triple-jawed (pincerlike) pedicellariae. These pedicellariae (globiferous, or glandular) are sometimes used for defense (Fig. 73-52). Urchins are nocturnal and omnivorous (mostly in pursuit of algae) eaters yet are shy, nonaggressive, and slow-moving animals found on rocky bottoms or burrowed in sand and crevices (Fig. 73-53). Their bathymetric range extends from the intertidal zone to great depths. The raw or cooked gonads of several species are eaten as a great delicacy by humans.

Figure 73-52. Globiferous pedicellariae are equipped with venom glands. (Photo by Dietrich Mebs.)

Venom and Venom Apparatus. Of the approximately 600 species of sea urchins, roughly 80 may be venomous to

Figure 73-53. Needle-like spines of sea urchins in their natural habitat. (Photo by Kenneth Kizer, MD.)

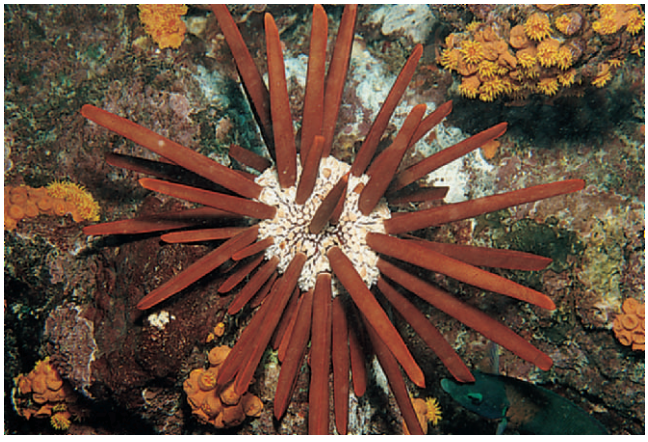


Figure 73-54. Nontoxic "pencil" urchin with blunt, rounded tips. (Photo by Paul Auerbach, MD.)

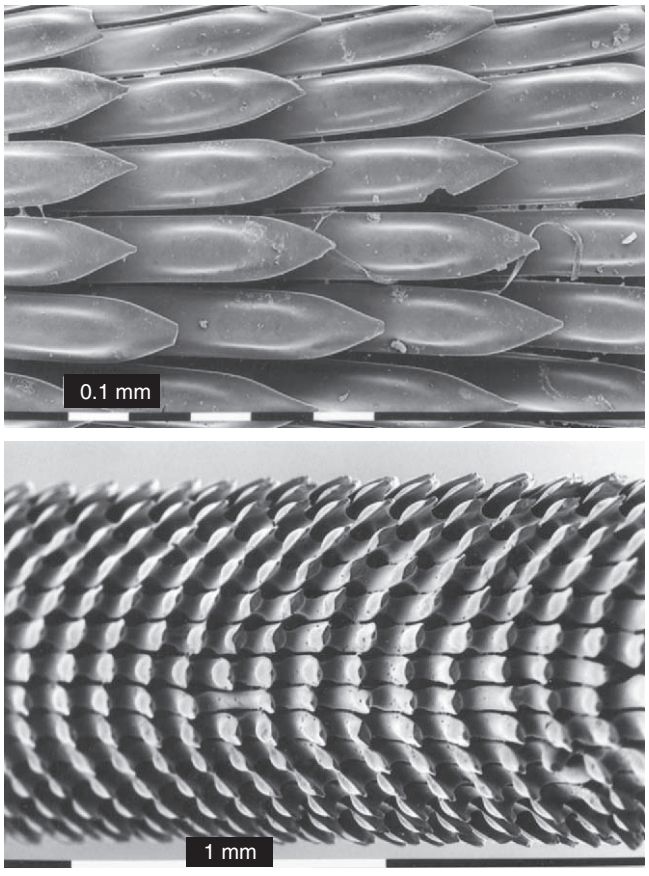


Figure 73-55. The spines of the diadematid sea urchin (*Diadema* species) are covered with small, tile-like structures. (Photos by Dr. J. Meier.)

humans.[94] The venom apparatuses of sea urchins consist of the hollow, venom-filled spines and the triple-jawed globiferous pedicellariae. Venom may also be released from within a thin integumentary sheath on the external surface of the spines of certain urchins.

The spines of sea urchins, formed by calcification of a cylindrical projection of subepidermal connective tissue, may either be non–venom bearing, with solid blunt and rounded tips (Fig. 73-54), or venom bearing (such as in the families Echinothuridae and Diadematidae [Fig. 73-55]), with hollow, long, slender, and sharp needles (Fig. 73-56). These are extremely dangerous to handle. The spines, which are attached to the shell with a modified ball-and-socket joint, are brittle and break off easily in the flesh, lodging deeply, and removal is difficult. They are keen enough to penetrate rubber gloves and fins. *Diadema setosum* (black sea urchin) spines may exceed 1 foot in length. *Echinothrix* species also carry lengthy spines. The purple sea urchin *Strongylocentrotus purpuratus* (Fig. 73-57) of California has much shorter spines. The genera *Asthenosoma* and *Aerosoma* have special venom organs (sacs) on the sharp tips of the aboral spines (Fig. 73-58), which introduce the potent venom.

Pedicellariae are small, delicate seizing organs attached to the stalks scattered among the spines. These are considered to be modified spines with flexible heads.[141] Globiferous pedicellariae, typified by those found in *Toxopneustes pileolus* (flower urchin) (Fig. 73-59) and *Tripneustes* species, have globe-shaped heads that contain the venom organs (Fig. 73-60). The terminal head, with its calcareous pincer jaws (two to four, but usually three), is attached by the stalk to the shell plates of the sea urchin. The outer surface of each opened "jaw" is covered by a large venom gland, which is triggered to contract with the jaw on contact. When the sea urchin is at rest in the water, the jaws are extended, slowly moving about (Fig. 73-61). Anything that touches them is seized. As long as the object is moving, the pedicellariae continue to bite and envenom. Once a pedicellaria attaches to a victim, it will be torn from the shell rather than let go. Detached pedicellariae may remain active for several hours. The *Toxopneustes* sea urchin also has solid spines, but these are nonvenomous.

The venom of sea urchins contains various toxic fractions, including steroid glycosides, hemolysins, proteases, serotonin, and cholinergic substances. The Pacific *Tripneustes* urchin carries a neurotoxin with a predilection for facial and cranial nerves. A toxic substance from the sea urchin *Toxopneustes pileolus* induces histamine release from rat peritoneal mast cells.[165] Contractin A (a mannose-containing glycoprotein) from the pedicellariae of the same species causes contraction of iso-

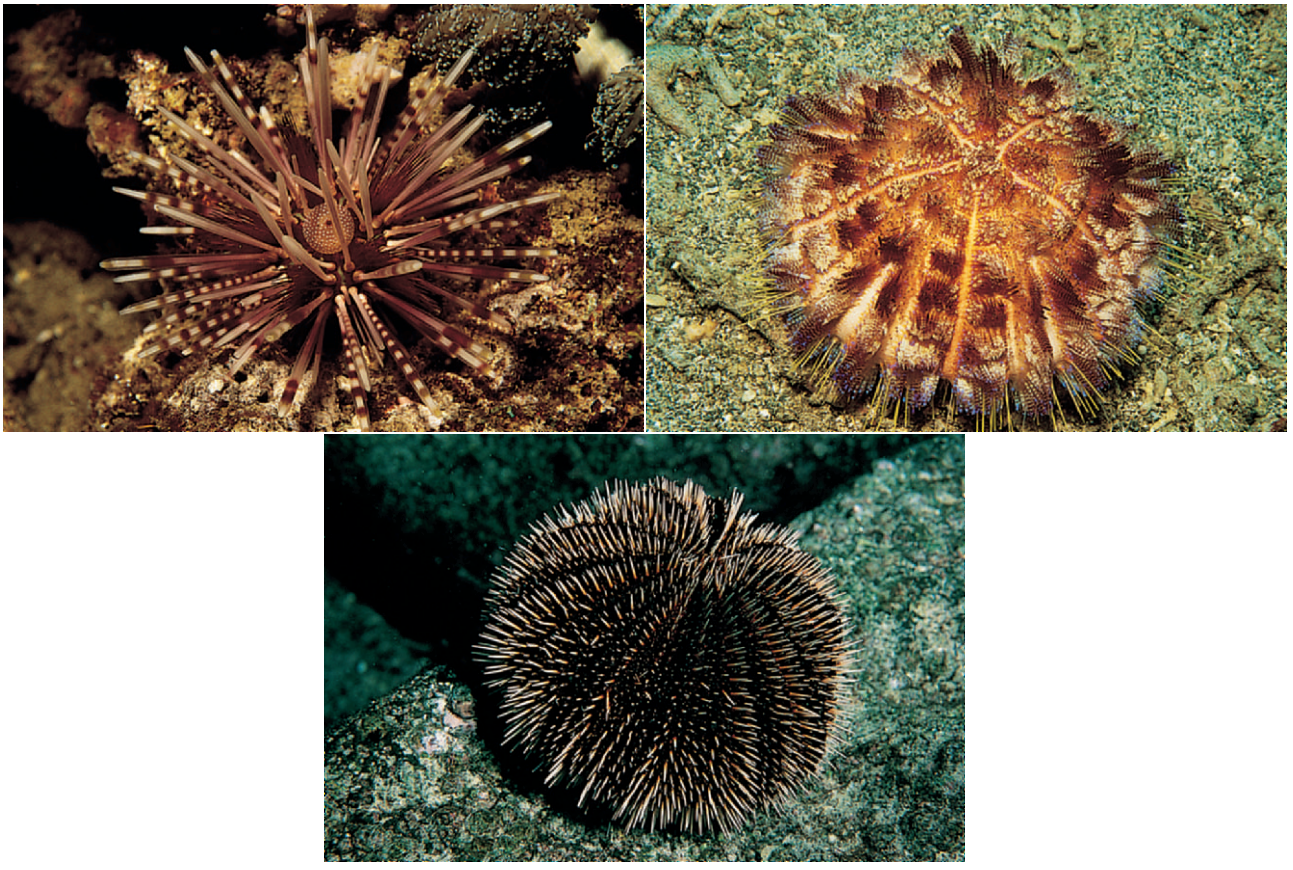


Figure 73-56. Three examples of sharp-spined (venomous) sea urchins. (Photos by Paul Auerbach, MD.)

lated guinea pig tracheal smooth muscle.[120] Other substances that have been identified from sea urchin spines or pedicellariae include D-galactose–binding lectins, and heparin-binding and hemolytic lectins.[119]

Clinical Aspects. Most victims are envenomed when they step on, handle, or brush up against a sea urchin. Because the creatures tend to be nocturnal, divers are most commonly injured in dark waters during night diving activities, particularly in small caves or in shallow turbulent waters. Young inquisitive children who explore tidepools frequently handle urchins incorrectly and may be injured. If a diver moves a hand slowly toward a spiny (venomous) sea urchin, the spines may align to offer the greatest defense.

Venomous spines inflict immediate and intensely painful stings. The pain is initially characterized by burning, which rapidly evolves into severe local muscle aching with visible erythema and swelling of the skin surrounding the puncture site or sites. Frequently, a spine breaks off and lodges in the victim. Some sea urchin spines (such as those of *Diadema setosum* or *Strongylocentrotus purpuratus*) contain black-purplish dye, which may give a false impression of spines left in the skin (Fig. 73-62). Soft tissue density x-ray techniques or magnetic resonance imaging (MRI) may reveal a radiopaque foreign body. If a spine enters a joint, it may rapidly induce severe synovitis. Over time, if the spine remains embedded in or near the joint, this may progress to arthritis.[94] If multiple spines have penetrated the skin, particularly if they are deeply embedded, systemic symptoms may rapidly develop, including nausea, vomiting, paresthesias, numbness and muscular paralysis, abdominal pain, syncope, hypotension, and respiratory distress. The presence of a frank neuropathy may indicate that the spine has lodged in contact with a peripheral nerve. The pain from multiple stings may be sufficient to cause delirium. Secondary infections and indolent ulceration are common. A delayed hypersensitivity–type reaction (flare-up) at the site of the punctures has been described, in which the victim demonstrates erythema and pruritus in a delayed fashion, 7 to 10 days after primary resolution from the initial envenomation.[3] The sensitizing antigen in such cases has yet to be identified. Hepatic transaminasemia after a relatively minor puncture has been

Figure 73-57. Purple sea urchins. (Photo by Howard Hall.)

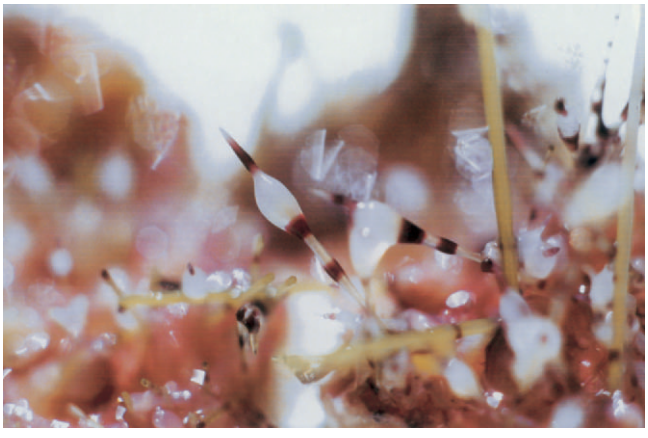


Figure 73-58. The tips of the short spines of the leather urchin (e.g., *Asthenosoma* from the Indo-Pacific) are encased by a venom gland. (Photo by Dietrich Mebs.)

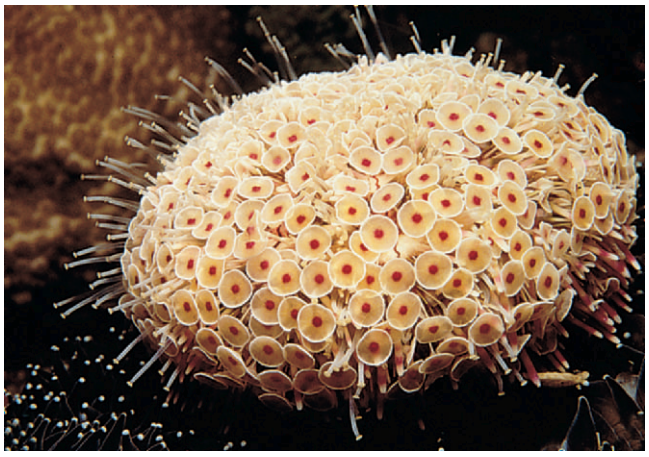


Figure 73-59. Flower urchin *(Toxopneustes pileolus)*. (Photo by Ken Kizer, MD.)

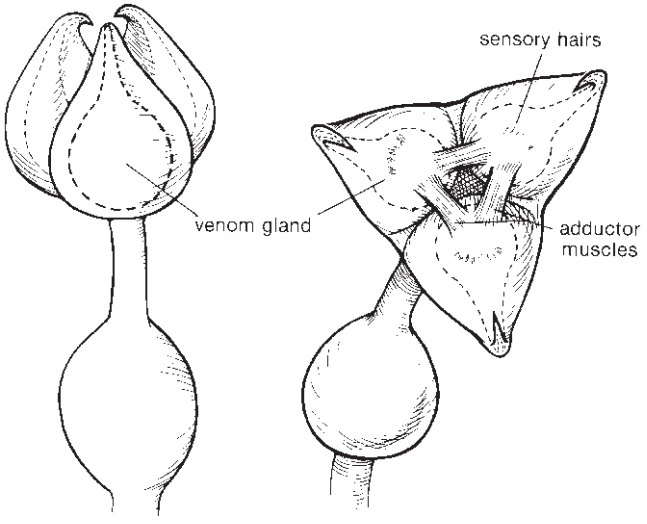


Figure 73-60. Globiferous pedicellaria of a sea urchin, used to hold and envenom prey.

reported, which may have been caused by the envenomation or by therapy with cephradine and mefenamic acid.[191]

Three separate unusual cases have been reported to this author since 1993 by neurologists. In each case, the victim sustained multiple punctures from one or several black sea urchins in Hawaiian waters. The immediate clinical reaction was typical, but it was followed in 6 to 10 days by severe bulbar polyneuritis with respiratory insufficiency. In two cases, the victims were hyporeflexic and appeared to suffer a Guillain-Barré variation with elevated protein levels in the cerebrospinal fluid. In the other case, the victim manifested meningoencephalitis documented by MRI. The temporal relationship to the urchin stings suggests an autoimmune phenomenon.

A spine that enters a finger in proximity to the nail apparatus may cause a subungual or periungual granulomatous nodular lesion. Excision may cause permanent nail plate dystrophy.

The stings of pedicellariae are often of greater magnitude, causing immediate intense radiating pain, local edema and hemorrhage, malaise, weakness, paresthesias, hypesthesia, arthralgias, aphonia, dizziness, syncope, generalized muscular paralysis, respiratory distress, hypotension, and, rarely, death. In some cases, the pain disappears within the first hour but the localized muscular weakness or paralysis persists for up to 6 hours.

Treatment. The envenomed body part should immediately be immersed in nonscalding hot water to tolerance (upper limit,

Figure 73-61. Close-up of opened flower urchin *(Toxopneustes pileolus)* pedicellariae seeking prey. (Photo by Ken Kizer, MD.)

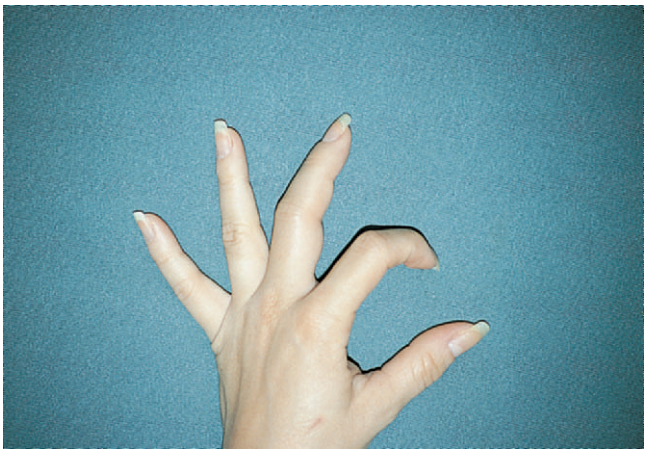


Figure 73-63. Finger swelling from sea urchin puncture. A single spine entered the palm over the mid third metacarpal bone. Swelling was severe in the second and third digits. (Photo by Paul Auerbach, MD.)

Figure 73-62. Thigh of the author demonstrating multiple sea urchin punctures from black sea urchins *(Diadema)*. Within 24 hours, the black markings were absent, indicative of spine dye without residual spines. (Photo by Ken Kizer, MD.)

45°C [113°F]) for 30 to 90 minutes in an attempt to relieve pain. Any pedicellariae still attached to the skin must be removed or envenomation will continue. This may be accomplished by applying shaving foam and gently scraping with a razor. Embedded spines should be removed with care because they easily fracture. Black or purplish discoloration surrounding the wound after spine removal is often merely spine dye and therefore may be of no consequence. Although some thin venomous spines may be absorbed within 24 hours to 3 weeks, it is best to remove those that are easily reached. All thick spines (calcium carbonate, magnesium carbonate, and silica) should be removed because of the risk of infection, foreign body encaseation granuloma, or dermoid inclusion cyst.

External percussion to achieve fragmentation may prove disastrous if a chronic inflammatory process is initiated in sensitive tissue of the hand or foot. If the spines have acutely entered joints or are closely aligned to neurovascular structures, the surgeon should take advantage of an operating microscope in an appropriate setting to remove all spine fragments. The extraction should be performed as soon as possible after the injury. If the spine has entered an interphalangeal joint, the finger should be splinted until the spine is removed to limit fragmentation and further penetration. This also may control the fusiform finger swelling (Fig. 73-63) commonly noted after a puncture in the vicinity of the middle or proximal interphalangeal joint. It is inappropriate to rummage about in a hand wound in the emergency department, virtually looking for a needle in a haystack. After a spine has been embedded in soft tissue for 24 to 48 hours, the spine dye may be absorbed, and the spine becomes flesh colored and very difficult to locate. If the spine is lodged in avascular tendon or ligament, the spine dye may persist for a longer period, allowing easier identification of the foreign body.

If the presence of a spine is in question, soft tissue density radiographic techniques for a radiopaque foreign body may be diagnostic. MRI (Fig. 73-64) may be quite useful to locate spine fragments. Although the calcium carbonate is relatively inert, it is accompanied by slime, bacteria, and organic epidermal debris. Therefore, secondary infections are common (Fig. 73-65), and deep puncture wounds are an indication for prophylactic antibiotics.

Some sea urchin spines are phagocytosed in the soft tissues and ultimately dissolve. The granulomas caused by retained sea urchin spine fragments have sarcoidal histologic features and generally appear as flesh- or dye-colored surface or subcuticular nodules 2 to 12 months after the initial injury (Fig. 73-66).[139] In thin-skinned areas, these nodules are erythematous and

Figure 73-64. Magnetic resonance imaging of the hand of a victim of multiple sea urchin spine punctures, demonstrating the presence of spine fragments in the soft tissues. (Photo by Paul Auerbach, MD.)

Figure 73-65. Infection after sea urchin puncture. The rapid-onset, gas-containing hemorrhagic blister and severe cellulitis with sepsis are common features of infection with *Vibrio* species. (Photo by Paul Auerbach, MD.)

rubbery, painless, and infrequently umbilicated. In thicker-skinned areas (palms, soles, and knees) that are frequently abraded, they have a keratinized appearance. Although necrosis and microabscess formation may be evident microscopically, suppuration is unusual. Rarely, the destructive nature of the inflammatory process may be severe enough to necessitate amputation of a digit. If a spine cannot be removed and becomes a nidus for cyst or granuloma formation, the lesion may be removed surgically. Intralesional injection with a corticosteroid (triamcinolone hexacetonide, 5 mg/mL) is less efficacious but may be successful. Systemic anti-inflammatory drugs may be minimally helpful but are not substitutes for removal of the spine. A diffuse delayed reaction, consisting of cyanotic induration, fusiform swelling in the digits, and focal phalangeal bony erosion, may be treated with a systemic corticosteroid and antibiotics.

Figure 73-66. Subcuticular nodule after sea urchin puncture. (Photo by Paul Auerbach, MD.)

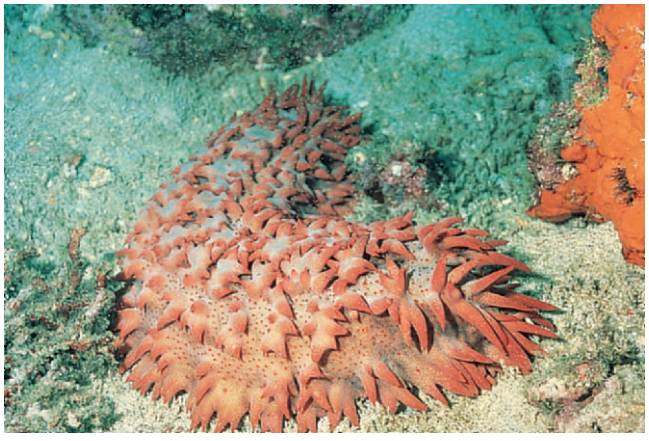


Figure 73-67. Sea cucumber. (Photo by Paul Auerbach, MD.)

Sea Cucumbers

Life and Habits. Sea cucumbers are free-living worm- or sausage-shaped bottom feeders of diverse external patterns and coloration (Fig. 73-67) that are essentially scavengers. They are cosmopolitan in distribution, found in both shallow and deep waters. Cucumbers are harvested as a food (trepang, bêche-de-mer) in the South Pacific.

Venom and Venom Apparatus. Cucumbers produce in their body walls a visceral cantharidin-like liquid toxin (holothurin). Holothurin is concentrated in the tentacular organs of Cuvier, which can be projected and extended anally when the animal mounts a defense (Fig. 73-68). Toxic genera include *Actinopyga*, *Stichopus*, and *Holothuria*. Some cucumbers dine on nematocysts and thus can secrete coelenterate venom as well.

Clinical Aspects. Holothurin may induce contact dermatitis when the tentacular organs directly contact the skin. Generally the substance is diluted in the surrounding ocean water and the reaction is minimal. However, persons who dissect sea cucum-

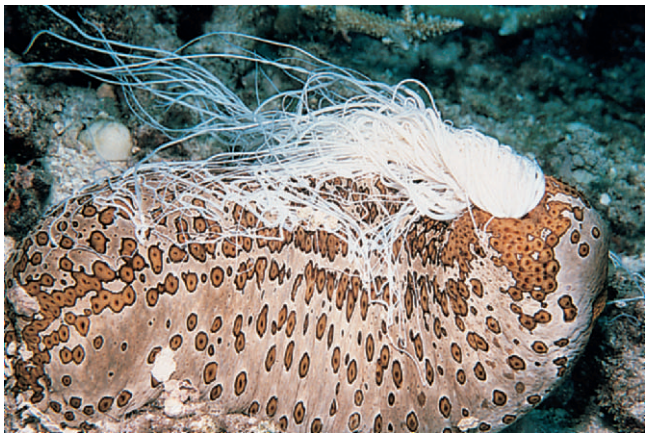


Figure 73-68. Extruded tentacular organs of Cuvier from within a sea cucumber. (Photo by Paul Auerbach, MD.)

bers topside in the preparation of food products may inadvertently handle the toxin and develop a papular skin irritation. The major risk underwater is to the corneas and conjunctivae, which may become intensely inflamed if directly contacted by tentacular fragments or high concentrations of the toxin. This may occur if the mask is cleared in the immediate vicinity of recent sea cucumber manipulation. A severe reaction may lead to blindness. Holothurin is a potent cardiac glycoside and may cause severe illness or death on ingestion.

Treatment. The management of holothurin-induced contact dermatitis is similar to that for starfish dermatitis. A topical or systemic corticosteroid may be necessary to manage a severe reaction. Because cucumbers that dine on nematocysts may secrete coelenterate venom, the initial skin detoxification should include topical application of 5% acetic acid (vinegar), papain, or 40% to 70% isopropyl alcohol. If an eye is involved, it should be anesthetized with 1 or 2 drops of 0.5% proparacaine and then irrigated with 100 to 250 mL of normal saline to remove any residual foreign matter. The cornea should then be stained with fluorescein to identify corneal defects. A proper slit-lamp examination is optimal to determine whether inflammation extends into the anterior chamber or involves the iris. If there is no sign of infection, a moderate approach to the inflammatory keratitis includes regular instillation of cycloplegic, mydriatic, and corticosteroid ophthalmic solutions. Prompt referral to an ophthalmologist is essential.

ANNELID WORMS (PHYLUM ANNELIDA)

Life and Habits. There are 6200 species of segmented marine worms (phylum Annelida, class Polychaeta), either free moving or sedentary. Some free-moving members are considered toxic and may attain 1 foot (30 cm) in length. The worms are predominantly carnivorous and exist in the tidal zone to depths of 5000 m (16,405 ft), mostly as bottom feeders. Each segment of the worm possesses paddlelike appendages (parapodia) for locomotion. From these project numerous silky or bristlelike setae, which are capable of puncturing the victim (Fig. 73-69).

Figure 73-69. The chitinous spines of a bristleworm are easily dislodged into the skin of an unwary diver. (Photo by Paul Auerbach, MD.)

The chitinous urticating bristles are arranged in soft rows about the body. When a worm is stimulated, its body contracts and the bristles are erected. There are no associated venom-producing cells. Easily detached, the bristles penetrate skin like cactus spines and are difficult to remove. The ubiquitous bottom-dwelling bristleworm *Hermodice carunculata* is frequently handled in Floridian and Caribbean waters by snorkelers and divers. This worm can attain a length of 1 foot and a width of 1 inch (2.54 cm). It is found on coral, under rocks, and moving among sponges. The body is green or reddish with tufts of white bristles. *Chloeia flava* is found along the Malayan coast, *Chloeia viridis* in the West Indies, Gulf of California, and Gulf of Mexico south to Panama, and *Eurythoe complanata* in Australia and other tropical seas. Other worms, such as *Chloeia euglochis,* are free swimming. Some marine worms possess strong chitinous jaws with pharyngeal teeth and can inflict painful bites.

Clinical Aspects. The bite or sting of an annelid worm may induce intense inflammation typified by a burning sensation with a raised, erythematous, and urticarial rash, most frequently on the hands and fingers (Fig. 73-70). Edema and papules ensue, with rare necrosis. The setae are easily fractured into the skin and are generally not visible on external inspection, although the victim may report a sensation of pricking or abrasion. Untreated, the pain is generally self-limited over the course of a few hours, but the inflammatory component of erythema and urticaria may last for 2 to 3 days, with total resolution of the skin discoloration over 7 to 10 days. With multiple stings, marked local soft tissue edema and pruritus may develop. Secondary infections and cellulitis may occur if the eczematous component is severe.

Treatment. All large visible bristles should be removed with forceps. The skin should be dried (without scraping, to avoid breaking or embedding the spines further into the skin) so that a layer of adhesive tape may be applied to remove the remaining smaller spines, which are too tiny for individual extraction. Application of tape may force spines into the tissue, causing pain. Alternatively, a facial "peel" or thin layer of rubber cement may be applied and removed. After this maneuver, 5% acetic acid (vinegar), 40% to 70% isopropyl alcohol, dilute ammonia, or a paste or solution of unseasoned meat tenderizer (papain) or application of a papain-impregnated scrub brush

Figure 73-70. Skin rash caused by a bristleworm. (Photo by Paul Auerbach, MD.)

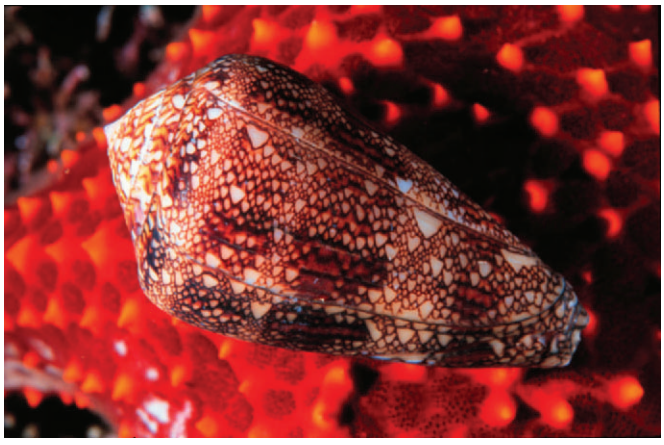


Figure 73-71. *Conus dalli,* in the Sea of Cortez. (From Norbert Wu, with permission; available at www.norbertwu.com.)

may provide some pain relief. If the inflammatory reaction becomes severe, the victim may benefit from the administration of a topical or systemic corticosteroid.

MOLLUSKS (PHYLUM MOLLUSCA)

The phylum Mollusca (45,000 species) encompasses a group of unsegmented, soft-bodied invertebrates, many of which secrete calcareous shells. Generally, a muscular foot is present with various modifications. Of the five main classes, three predominate in their hazard to humans: the pelecypods (such as scallops, oysters, clams, and mussels), the gastropods (such as snails and slugs), and the cephalopods (such as squids, octopuses, and cuttlefish). Mollusks are often implicated as the transvectors in poisonous ingestions.

Cone Snails (Cone Shells)

Life and Habits. There are approximately 500 species of these circumtropical, beautiful, yet potentially lethal, univalve and cone-shaped shelled mollusks of the class Gastropoda, family Conidae, genus *Conus* (Fig. 73-71).[111] Most of these carnivores carry a highly developed venom apparatus, and at least 18 species have been implicated in human envenomations, with occasional fatalities (approximately 16 to 30 have been recorded).[67] These include *Conus aulicus* (court), *Conus geographus* (geographer), *Conus gloria-maris* (glory of the sea), *Conus marmoreus* (marbled), *Conus omaria* (pearled), *Conus striatus* (striated) (Fig. 73-72), *Conus textile* (textile) (Fig. 73-73), and *Conus tulipa* (tulip).

Most harmful cone snails ("cones") are creatures of shallow Indo-Pacific waters; the variance in feeding habits and venom production accounts for the varying toxicity. Atlantic species, such as *Conus ermineus* (turtle) are less toxic. *Conus regius* (crown or queen) and *Conus spurius* (Chinese alphabet) are found in Florida waters. Apparently, cones that feed on fish or mollusks are the most dangerous. Less toxic stings are attributed to cones that feed on marine worms. Predominantly nocturnal creatures, cones burrow in the sand and coral during the daytime, emerging at night to feed. They have two eye stalks, but vision is poor, so chemosensory prowess is required to identify and approach prey.[167]

Figure 73-72. *Conus striatus.* (From Norbert Wu, with permission; available at www.norbertwu.com.)

Venom and Venom Apparatus. Cone snails are predators that feed by injecting rapid-acting venom by means of a detachable, dartlike radular tooth (or radula) (Fig. 73-74). To do this, the head of the animal must extend out of the shell. The venom apparatus is composed of a set of minute, harpoonlike, chitinous, and hollow radular teeth associated with a venom bulb, a long convoluted duct, and a radular sheath (Fig. 73-75).[114] The barbed teeth, which may attain a length of 1 cm (~$^3/_8$ inch), are housed within the radular sheath. The act of envenomation is performed by the release of a radular tooth from the sheath into the pharynx, where it is "charged" with venom from the venom duct and then transferred to the extensible proboscis. This appendage, which may extend in some species as far back as the spire of the shell, grasps the venom-impregnated and barbed tooth and thrusts it into the flesh of the victim. In normal small fish prey, the cone snails may deploy a hunting method of initial rigid paralysis with fin tetanus to tether the prey to the radular tooth, then flaccid paralysis to allow consumption. This has been observed in the fish-hunting snail *C. purpurascens*.[168]

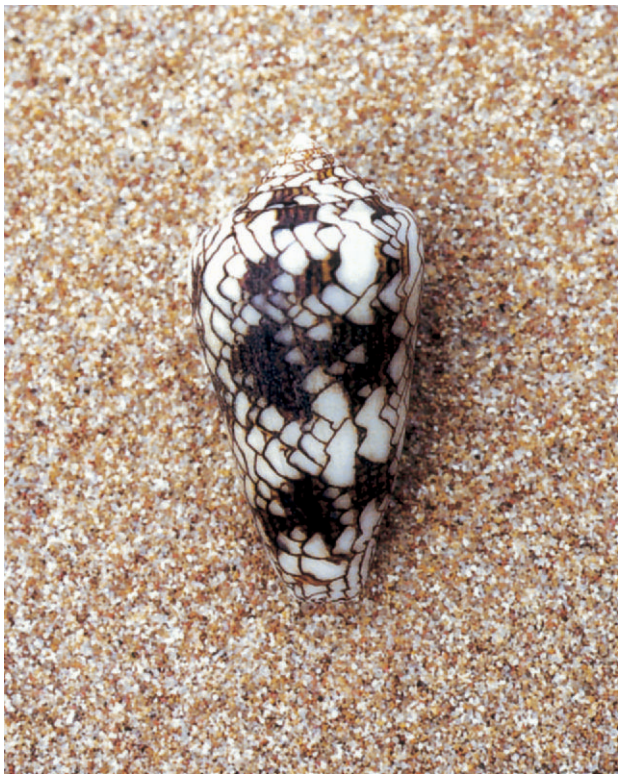


Figure 73-73. Cone snail, *Conus textile,* from the Red Sea. (Photo by Dietrich Mebs.)

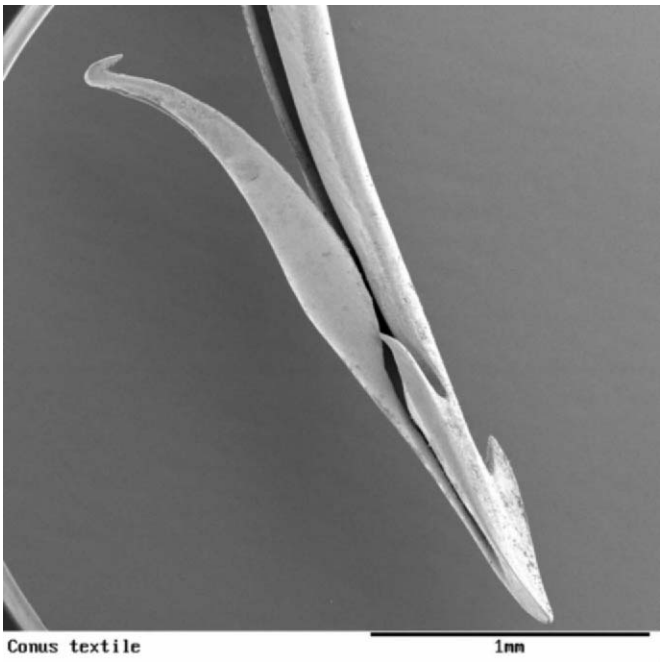


Figure 73-74. Radula tooth of *Conus textile.* (Photo by Dietrich Mebs.)

The venom is composed of biologically active peptides (more than 100 conotoxins have been identified) of 13 to 35 amino acids in length.[125] The majority of the unique *Conus* peptides appear to be derived from a few gene superfamilies (A, M, and O), which results in the biologically active venom components.[45] Peptide families in the A-superfamily include the α-conotoxins and the αA-conotoxins, which antagonize the nicotinic acetylcholine receptor, as well as the κA-conotoxins, which may act by blocking voltage-gated potassium channels. The μ-conotoxins, in the M-superfamily, block voltage-gated sodium channels. The ψ-conotoxins are noncompetitive antagonists of the nicotinic acetylcholine receptor. In the O-superfamily are the ω-conotoxins, which block voltage-sensitive calcium channels; δ-conotoxins, which delay inactivation of voltage-sensitive sodium channels; μO-conotoxins, which block voltage-gated sodium channels; and κ-conotoxins, which block voltage-gated potassium channels.[111]

Smaller peptides are probably strategic from an evolutionary perspective because of the speed of diffusion through a poisoned fish. The venom targets are neuromuscular transmission and ion channels.[67] Because there is a redundancy of sites of action at the neuromuscular junction, presynaptically and postsynaptically, minute amounts of conotoxins effect neuromuscular blockade.[177]

At the same site as tetrodotoxin and saxitoxin, μ-conotoxins bind and modify muscle sodium channels.[73,120] Voltage-dependent calcium uptake at the presynaptic cleft, and cholinergic transmission in avian and mammalian neuromuscular junctions are inhibited by ω-conotoxins such as that from *C. geographus*.[43] These ω-conotoxins bind to neuronal (N-type) rather than the cardiac (L-type) calcium channels, which prevents the calcium influx necessary for neurotransmitter release. N-type calcium channels are expressed almost exclusively on neurons, and they are implicated in synaptic release of neurotransmitters such as substance P and calcitonin gene-related peptide within nociceptive sensory neurons. Ziconatide, a synthetic form of the ω-conopeptide MVIIa, is a potent analgesic intended for human application directly to the spinal cord and to prevent cell death in the brain after head trauma and ischemic events.[177] The α-conotoxins block the nicotinic acetylcholine receptor.[67,120] A sleeper peptide in *C. geographus* venom causes test animals to enter a deep sleeplike state.[67] Conantokins G and T, which are selective inhibitors of certain subtypes of the *N*-methyl-D-aspartate (NMDA) receptor, exhibit potent antinociceptive effects in several models of injury-induced pain, which holds promise as a novel therapeutic approach for the control of pain.[103] Furthermore, if conotoxins targeting NMDA receptors can be translated into effective drugs, this may lead to another approach to the treatment of epilepsy.[83] A novel conotoxin isolated from *Conus virgo* inhibits vertebrate voltage-sensitive potassium channels.[87] Serotonin is present in venom from the cone snail *Conus imperialis,* which is a worm feeder.[110] In the act of envenomation, milky venom from the venom duct is transformed into a clear product, which may indicate conversion from an ineffective to an effective toxin.

Clinical Aspects. Most stings occur on the fingers and hand, as the unknowledgeable fossicker (i.e., a prospector, or collector) incorrectly handles a hazardous specimen. Mild stings are puncture wounds that resemble bee or wasp stings, with associated burning or sharp stinging sensation. The initial pain is followed

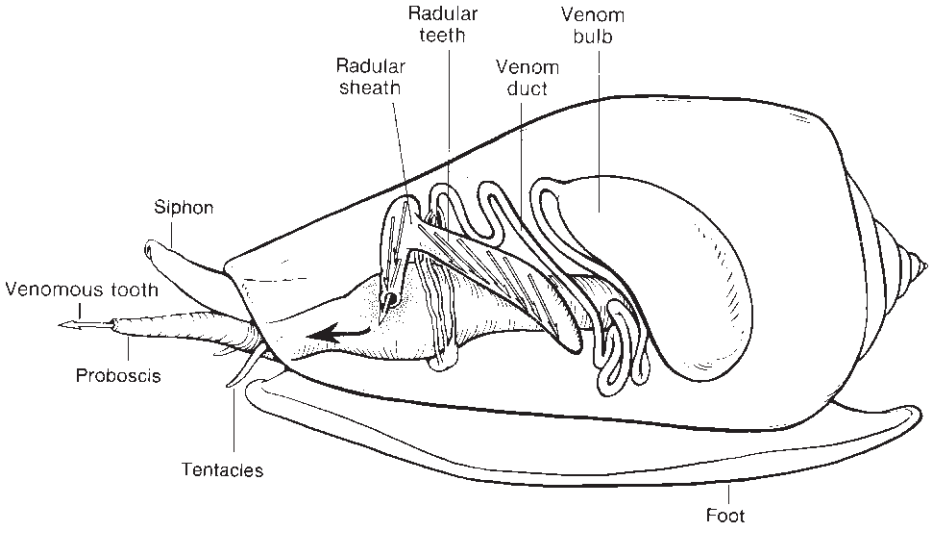


Figure 73-75. Venom apparatus of the cone snail.

by localized ischemia, cyanosis, and numbness in the area surrounding the wound. Numbness may occur without preceding pain, or in a rare case, the envenomation may be without any specific dermal sensation. More serious envenomations induce paresthesias at the wound site, which rapidly encompass the limb and then become perioral before generalized. Partial paralysis transitions to generalized muscular paralysis, causing diaphragmatic dysfunction and respiratory failure; bronchospastic respiratory distress is not commonly seen. Coma has been observed, and death is attributed to diaphragmatic paralysis or cardiac failure. Other symptoms include dysphagia, syncope, weakness, failing coordination, areflexia, aphonia, dysarthria, diplopia, ptosis, absent gag reflex, blurred vision, and pruritus. The sting of *C. geographus* may be rapidly toxic, with progression to cerebral edema, coma, respiratory arrest, and cardiac failure within a few hours, perhaps even 1 hour. Although mild stings may cause symptoms of nausea, blurred vision, malaise, and weakness for only a few hours, a severe envenomation may induce symptoms that require 2 to 3 weeks to achieve total resolution. *C. textile* and *C. marmoreus* have been reported to kill humans. A fatality has been attributed to *C. gloria-maris,* but this has not yet been confirmed.[114]

Treatment. No antivenom is available for a cone shell envenomation. Numerous therapies have been recommended, including the pressure-immobilization technique (see Figure 48-27), application of a proximal lymphatic–venous occlusive bandage, incision and suction, soaking in nonscalding hot water to tolerance (upper limit, 45° C [113° F]) until pain is relieved, injection of a local anesthetic (1% to 2% lidocaine without epinephrine), and local excision. The pressure-immobilization technique makes sense and should be applied.

Cardiovascular and respiratory support are the usual priorities after a severe envenomation. The wound should be inspected for the presence of a foreign body (the radula). Edrophonium (10 mg intravenously for an adult) has been suggested as empirical therapy for paralysis. A rational approach would be to administer an edrophonium (Tensilon) test to determine effectiveness. The clinician should choose a weak muscle group for which strength can be objectively measured, then inject edrophonium (2 mg intravenously). If there is improvement, this is followed by edrophonium (8 mg intravenously). Adverse reactions to edrophonium (an anticholinesterase inhibitor) include salivation, nausea, diarrhea, and muscle fasciculations. These can be ameliorated with atropine, 0.6 mg intravenously.

Cone shells should be handled only when wearing proper gloves; if the proboscis protrudes, the cone should be dropped. If the animal must be carried, it should always be lifted by the large posterior end of the shell, although this does not afford complete protection. A collector should never carry a live cone inside a wet suit, clothing pocket, or buoyancy compensator pocket.

Octopuses

Life and Habits. Octopuses and cuttlefish are cephalopods that are usually harmless and retiring. On occasion, they are noted to manifest "curiosity" or "play behavior," by navigating mazes or manipulating objects without intent to feed or create a habitat. True octopuses are inhabitants of warmer waters and have little tolerance for extremes in salinity. They prefer rocky bottoms and rock pools in the intertidal zones. The entertainment media have created the image of a giant creature that envelops its victim in a maze of tentacles and suction cups. However, most dangerous (envenoming) creatures are smaller than 10 to 20 cm (4 to 8 inches) and do not squeeze their victims at all. On the other hand, there are reports in the South Pacific of breath-hold spearfishermen drowned while hunting octopuses. The method used to kill the animals was to allow an octopus to cling to a diver, who would bite the animal between the eyes as the combatants surfaced. Apparently, the octopuses were large enough (4-m [13-foot] tentacle span) to resist the technique. *Octopus apollyon* can be pugnacious and may bite, in one case causing an immediate reaction of bradycardia and hypotension.[18]

Octopus bites are rare but can result in severe envenomations. Fatalities have been reported from the bites of the Australian blue-ringed (or "spotted") octopuses, *Octopus (Hapalochlaena) maculosa,* and *Octopus (Hapalochlaena) lunulata*. These small creatures, which rarely exceed 20 cm (8 inches) in length with tentacles extended, are found throughout the Indo-Pacific

Figure 73-76. Extremely venomous temperate blue-ringed octopus *(Hapalochlaena maculosa),* in Southern Australia. (From Gary Bell; available at www.oceanwideimages.com.)

(Australia, New Zealand, New Guinea, Japan) in rock pools, under discarded objects and shells, and in shallow waters, posing a threat to curious children, tidepoolers, fossickers, and unwary divers.[162] Divers rarely spot them in water deeper than 3 m (10 ft). The bodies are oblong and pyriform, with a pointed tail and conspicuous excrescences on the upper surface.[17] In Australian waters, *H. maculosa,* the southern species, is smaller and yellow. *H. lunulata* is found in the north; larger, darker, and predominantly brownish, it favors the warmer tropical water. A third species, the blue-lined octopus *(Hapalochlaena fasciata)* has been described along the east coast (New South Wales) of Australia.[122] It has blue lines on the body and blue rings on the arms. When any of these animals is at rest, it is covered with dark brown to yellow-ochre bands over the body and arms, with superimposed blue patches or rings.[159] When the animal is excited or angered, the entire body darkens and the blue circles or stripes glow iridescent peacock blue, a trait shared by other animals, such as the peacock flounder *(Bothus lunatus)*. The colorful appearance is attractive to small children, who can easily handle the 25- to 90-g animal (Fig. 73-76). The smallish *Octopus joubini* of the Caribbean, which lives in small shells and empty containers, such as submerged bottles, is dangerous to a lesser degree; envenomation causes pain followed by numbness, fever, and nausea. The large common octopus, *Octopus vulgaris,* is nontoxic. Many octopuses can release inky fluid into the water, which is used to confuse attackers, but this mechanism is not present in the blue-ringed octopus. The chameleon-like changing of colors to match the surroundings is accomplished with pigment cells (chromatophores).

Venom and Venom Apparatus. The venom apparatus of the octopus consists of the salivary glands (anterior and posterior), salivary ducts, buccal mass, and beak. The mouth is located ventrally and centrally at the base of the tentacles and is surrounded by a circular lip fringed with fingerlike papillae, leading into a muscular pharyngeal cavity. This anatomic complex (buccal mass), concealed by the tentacles, is fronted by two parrotlike, powerful, and chitinous jaws (the beak), which bite and tear with great force at food held by the suckers. The salivary glands, particularly the posterior ones, secrete maculotoxin (or cephalotoxin) via the salivary ducts into the pharynx. This venom, normally released into the water to subdue crabs, may be injected into the victim with great force through the dermis down to the muscle fascia.[162] Only the venom of *H. maculosa* has been extensively studied. The toxin, maculotoxin (molecular weight less than 5000), contains at least one fraction identical to tetrodotoxin ($C_{11}H_{17}O_8N_3$) of molecular weight 319.3, which blocks peripheral nerve conduction by interfering with sodium conductance in excitable membranes by blocking voltage-gated sodium channels.[84,149] The toxin, as well as the tetrodotoxin precursor anhydrotetrodotoxin, is produced by bacteria of the Vibrionaceae family, and it is passed along the food chain to the octopus.[177]

This paralytic agent rapidly produces neuromuscular blockade, notably of the phrenic nerve supply to the diaphragm, without any apparent direct cardiotoxicity. It has been estimated that enough venom (25 g) may be present in one adult octopus to paralyze 750 kg of rabbits or 10 adult victims.[159,162] An adult blue-ringed octopus can inject a second fatal dose of toxin after a 1-hour interval. The venom is active on ingestion or by parenteral administration, the latter being much more effective. Other components of the venom, which include hyaluronidase, histamine, 5-hydroxytryptamine, tyramine, serotonin, and hapalotoxin (thought to derive from tyrosine, but still not confirmed as being present), are not thought to be major contributors to the clinical effects of an octopus bite.[145] Because most venoms and toxins with molecular weights less than 30,000 are poor antigens, octopus venom elicits no good antivenom.[162]

Clinical Aspects. Most victims are bitten on the hand or arm as they handle the creature or "give it a ride." No blue-ringed octopus bites have yet been reported from an animal in the water.[180] An octopus bite usually consists of two small puncture wounds produced by the chitinous jaws. The bite goes unnoticed or causes only a small amount of discomfort, described as a minor ache, slight stinging, or a pulsating sensation. Occasionally, the site is initially numb, followed in 5 to 10 minutes by discomfort that may spread to involve the entire limb, persisting for up to 6 hours. Local urticarial reactions occur variably, and profuse bleeding at the site is attributed to a local anticoagulant effect or may rarely be a harbinger of coagulation abnormalities. Within 30 minutes, considerable erythema, swelling, tenderness, heat, and pruritus develop. By far the most common local tissue reaction is absence of symptoms, a small spot of blood, or a tiny blanched area.[179] More serious symptoms are related predominantly to the neurotoxic properties of the venom. Within 10 to 15 minutes of the bite, the victim notices oral and facial numbness, rapidly followed by systemic progression.[180] Voluntary and involuntary muscles are involved, and the illness may rapidly progress to total flaccid paralysis and respiratory failure. Other symptoms include perioral and intraoral anesthesia (classically, numbness of the lips and tongue), diplopia, blurred vision, aphonia, dysphagia, ataxia, dizziness, myoclonus, weakness, a sense of detachment, nausea, vomiting, peripheral neuropathy, absent deep tendon reflexes, flaccid muscular paralysis, a sensation of chest tightness, and respiratory failure, which may lead to death. Ataxia of cerebellar configuration may occur after an envenomation that does

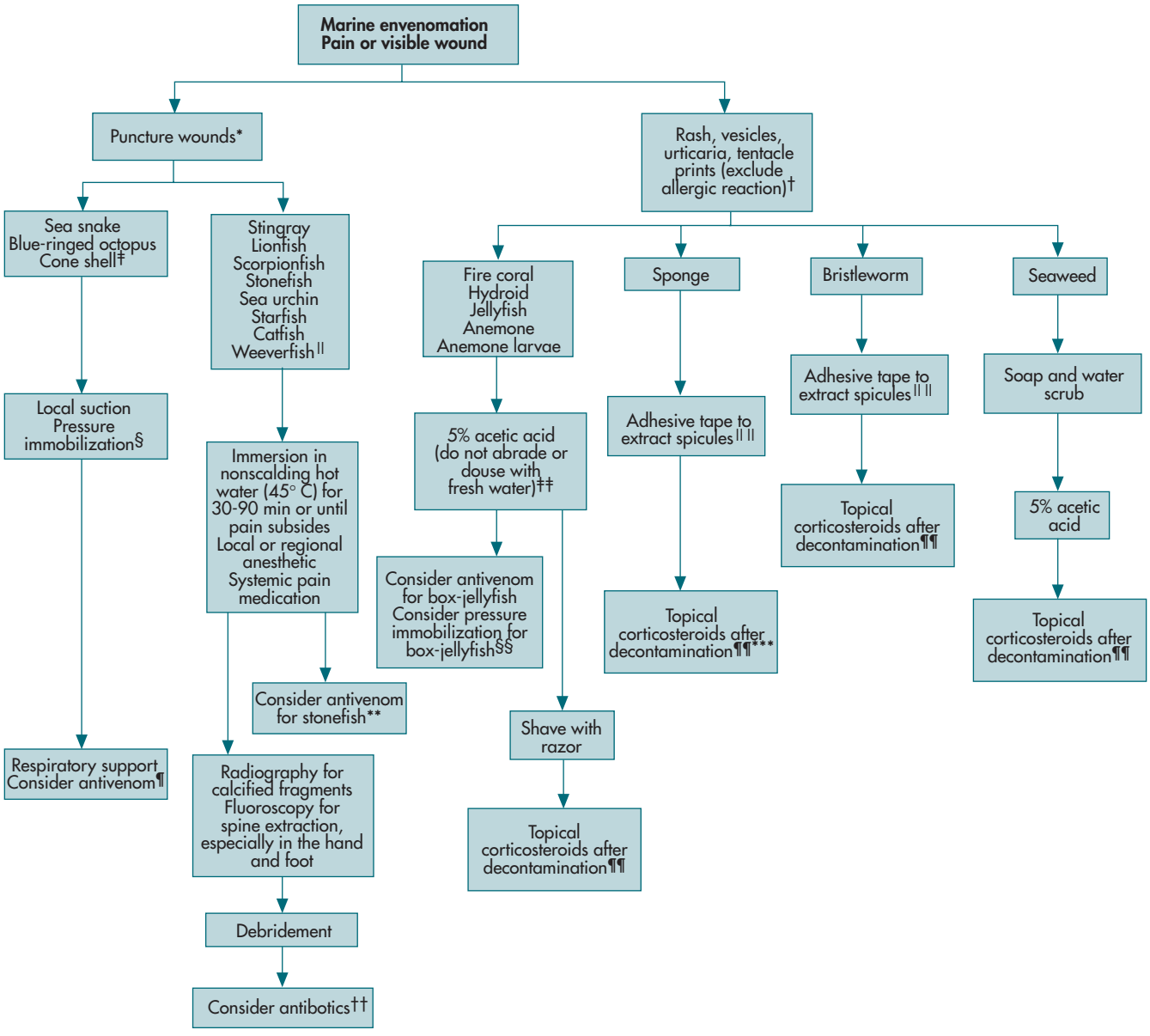


Figure 73-77. Algorithmic approach to marine envenomation.

*A gaping laceration, particularly of the lower extremity, with cyanotic edges suggests a stingray wound. Multiple punctures in an erratic pattern with or without purple discoloration or retained fragments are typical of a sea urchin sting. One to eight (usually two) fang marks are usually present after a sea snake bite. A single ischemic puncture wound with an erythematous halo and rapid swelling suggests scorpionfish envenomation. Blisters often accompany a lionfish sting. Painless punctures with paralysis suggest the bite of a blue-ringed octopus; the site of a cone shell sting is punctate, painful, and ischemic in appearance.

†Wheal and flare reactions are nonspecific. Rapid (within 24 hours) onset of skin necrosis suggests an anemone sting. "Tentacle prints" with cross-hatching or a frosted appearance after application of aluminum-based salts suggest a box-jellyfish *(Chironex fleckeri)* envenomation. Ocular or intraoral lesions may be caused by fragmented hydroids or coelenterate tentacles. An allergic reaction must be treated promptly.

‡Sea snake venom causes weakness, respiratory paralysis, myoglobinuria, myalgias, blurred vision, vomiting, and dysphagia. The blue-ringed octopus injects tetrodotoxin, which causes rapid neuromuscular paralysis.

§As soon as possible, venom should be sequestered locally with a proximal venous–lymphatic occlusive band of constriction, or (preferably) by the pressure-immobilization technique, in which a cloth pad is compressed directly over the wound by an elastic wrap that should encompass the entire extremity at a pressure of 9.33 kPa (70 mm Hg) or less.[4] Incision and suction are not recommended.

¶Early ventilatory support has the greatest influence on outcome. The minimal initial dose of sea snake antivenom is one to three vials; up to 10 vials may be required.

Figure 73-77, cont'd. Algorithmic approach to marine envenomation.

||The wounds range from large lacerations (stingrays) to minute punctures (stonefish). Persistent pain after immersion in hot water suggests a scorpionfish or stonefish sting or a retained fragment of spine. The puncture site can be identified by forcefully injecting 1% to 2% lidocaine or another local anesthetic agent without epinephrine near the wound and observing the egress of fluid. Do not attempt to crush the spines of sea urchins if they are present in the wound. Spine dye from already-extracted sea urchin spines will disappear (be absorbed) in 24 to 36 hours.

**The initial dose of stonefish antivenom is one vial per two puncture wounds.

††The antibiotics chosen should cover *Staphylococcus, Streptococcus,* and microbes of marine origin, such as *Vibrio.*

‡‡Acetic acid (5%; i.e., vinegar) is a good all-purpose decontaminant and is mandated for the sting from a box-jellyfish. Alternatives, depending on the geographic region and indigenous jellyfish species, include isopropyl alcohol, bicarbonate (baking soda), ammonia, papain, and preparations containing these agents.

§§The initial dose of box-jellyfish antivenom is one ampule intravenously or three ampules intramuscularly.

¶¶If inflammation is severe, steroids should be given systematically (beginning with at least 60 to 100 mg of prednisone or its equivalent), and the dose should be tapered over a period of 10 to 14 days.

|| ||An alternative is to apply and remove commercial facial peel materials.

***An alternative is to apply and remove commercial facial peel materials followed by topical soaks of 30 mL of 5% acetic acid (vinegar) diluted in 1 L of water for 15 to 30 minutes several times a day until the lesions begin to resolve. Anticipate surface desquamation in 3 to 6 weeks.

not progress to frank paralysis. Jerking limbs have been mentioned, as have poorly reactive or unreactive pupils. The victim may collapse from weakness and remain awake, so long as oxygenation can be maintained. When breathing is disturbed, respiratory assistance may allow the victim to remain mentally alert despite being paralyzed. Cardiac arrest is probably a complication of the anoxic episode.[175] Although tetrodotoxin is a potent vascular smooth muscle depressant, it does not appear to often produce significant hypotension in humans. However, hypotensive crisis has been mentioned in the literature as a complicating factor.

Treatment. First aid at the scene might include the pressure-immobilization technique (see Figure 48-27), although this is as yet unproven for management of octopus bites. A monoclonal rabbit serum IgG antibody has been effective against tetrodotoxin injected into mice.[107,138] This raises the possibility of the practical use of passive immunotherapy in the event of tetrodotoxin poisoning.

Treatment is based on symptoms and is supportive. Prompt mechanical respiratory assistance has by far the greatest influence on the outcome. Respiratory demise should be anticipated early, and the rescuer should be prepared to provide artificial ventilation, including endotracheal intubation and the application of a mechanical ventilator. The duration of the intense clinical venom effect is 4 to 10 hours, after which the victim who has not suffered an episode of significant hypoxia shows rapid signs of improvement. If no period of hypoxia occurs, mentation may remain normal. Complete recovery may require 2 to 4 days. Residua are uncommon and related to anoxia rather than venom effects.

Management of the bite wound is controversial. Some clinicians recommend wide circular excision of the bite wound down to the deep fascia, with primary closure or an immediate full-thickness free skin graft, whereas others advocate observation and a nonsurgical approach. Because the local tissue reaction is not a significant cause of morbidity, excision is presumably recommended to remove any sequestered venom. Kinetic studies of radiolabeled venom absorption are necessary to track the movement of octopus bite–introduced tetrodotoxin. As previously mentioned, there is no antivenom. Granuloma annulare of the hand developing over a 2-week period after an octopus (presumed to be *O. vulgaris* of the Florida Gulf Coast) bite of the hand has been reported.[58] On biopsy, histologic sections demonstrated superficial and deep dermal foci of altered dermis, presumably degenerated collagen, surrounded by histiocytes, lymphocytes, and fibroblasts. Intralesional triamcinolone acetonide injections were temporarily successful in treating the primary lesion.[108]

Prevention. All octopuses, particularly those less than 20 cm (8 inches) in length (including *O. joubini* of the Caribbean), should be handled with gloves. Divers need to be familiar with the lethal creatures in their domain. Giving an octopus a ride on the back, shoulder, or arm is not recommended.

SUMMARY

A summary algorithmic approach to marine envenomation (Fig. 73-77) can be followed when the causative agent cannot be definitively identified.

The references for this chapter can be found on the accompanying DVD-ROM.

74 Envenomation by Aquatic Vertebrates

Paul S. Auerbach

See Chapter 72 for a discussion of infections associated with aquatic wounds and the relevant antimicrobial therapy. A recent analysis that compared DNA sequences from 233 fish species was used to create a new family tree for spiny-rayed fishes. This indicates that previous estimates of approximately 200 venomous fishes should be revised to suspect at least 1,200 fishes as perhaps venomous.[125a]

STINGRAYS

Stingrays are the most commonly incriminated group of fish involved in human envenomations. They have been recognized as venomous since ancient times, known as "demons of the deep" and "devil fish." Aristotle (384–322 B.C.) made reference to their stinging ability.

Stingrays are members of the class Chondrichthyes (cartilaginous fish), subclass Elasmobranchii (plates and gills; with sharks and chimaeras), order Rajiformes (which contains stingrays [Dasyatidae], guitarfish [Rhinobatidae], skates [Rajidae], electric rays [Torpedinidae], eagle rays [Myliobatidae], mantas [Mobulidae], and freshwater rays [Potamotrygonidae]). Twenty-two species of stingrays are found in U.S. coastal waters, 14 in the Atlantic, and 8 in the Pacific. The family Dasyatidae includes most of the species that cause human envenomation. Skates are harmless. It is likely that at least 2000 stingray injuries take place each year in the United States. On the west coast of the United States, the round stingray (*Urolophus* [or *Urobatis*] *halleri*) is a frequent stinger; along the southeastern coast, it is the southern stingray *(Dasyatis americana)*. Most attacks occur during the summer and autumn months, as vacationers venture into the surf that may be laden with congregating (for spawning purposes) rays. Freshwater species do not inhabit U.S. waters. They are found in South America, Africa, and Southeast Asia.

Life and Habits

Stingrays are usually found in tropical, subtropical, and warm temperate oceans, generally in shallow (intertidal) water areas, such as sheltered bays, shoal lagoons, river mouths, and sandy areas between patch reefs.[59] Although rays are generally found above moderate depths, at least one deep-sea species has been discovered. Rays can enter brackish and fresh waters as well. For instance, freshwater stingrays are common in rivers and tributaries in South America.[42]

Rays are small (several inches) to large (up to 4 m × 2 m [12 ft × 6 ft]) creatures observed lying on top of the sand and mud or partially submerged, with only the eyes, spiracles, and part of the tail exposed (Fig. 74-1). Their flattened bodies are round-, diamond-, or kite-shaped, with wide pectoral fins that look like wings (Fig. 74-2). Rays are nonaggressive scavengers and bottom feeders that burrow into the sand or mud to feed on worms, mollusks, and crustaceans. The mouth and gill plates are located on the ventral surface of the animal (Fig. 74-3). The flattened shape is largely configured by the modified pectoral fins, or "wings," of the animal. These wings ripple or flap to propel the animal through the water.

Venom and Venom Apparatus

The venom organ of stingrays consists of one to four venomous stings on the dorsum of an elongate, whiplike caudal appendage. Anatomic types of stingray venom organs, and thus stinging ability, are differentiated into four groups based on their adaptability as a defense organ (Fig. 74-4): (1) the gymnurid type (butterfly rays, or Gymnuridae), with a poorly developed sting of up to 2.5 cm (1 inch) placed at the base of a short tail; (2) the myliobatid type (eagle and bat rays, or Myliobatidae), with a sting of up to 12 cm (4.7 inches) placed at the base of a cylindrical caudal appendage that terminates in a long whiplike tail; (3) the dasyatid type (stingrays and whip rays, or Dasyatidae), with a sting of up to 37 cm (14.5 inches) placed at the base or further out on the caudal appendage that terminates in a long whiplike tail; and (4) the urolophid type (round stingrays, or Urolophidae), with a sting of up to 4 cm (1.5 inches) located at the base of a short, muscular, well-developed caudal appendage. The efficiency of the apparatus is related to the length and musculature of the tail and to the location and length of the sting. Eagle rays and some mantas (Atlantic *Mobula mobular* and Pacific *Mobula japanica*) have a stinging apparatus, but it is less of a threat because the spine is located at the base of the tail and is not well adapted as a striking organ. Although the manta *(Manta birostris)* may grow to a width ("wingspan") of 6 m (20 feet) and weight of 1800 kg (4000 pounds), it dines on small fish, crustaceans, and microorganisms (Fig. 74-5). Many divers have "hitched" a ride on the wings of a manta; there are no reports of envenomation. However, manta skin is rough and can abrade unprotected human skin. A stingray "hickey" is a mouth-bite created by powerful grinding plates that produces superficial erosions and ecchymosis in an oral pattern. Persons who hand-feed stingrays may incur this

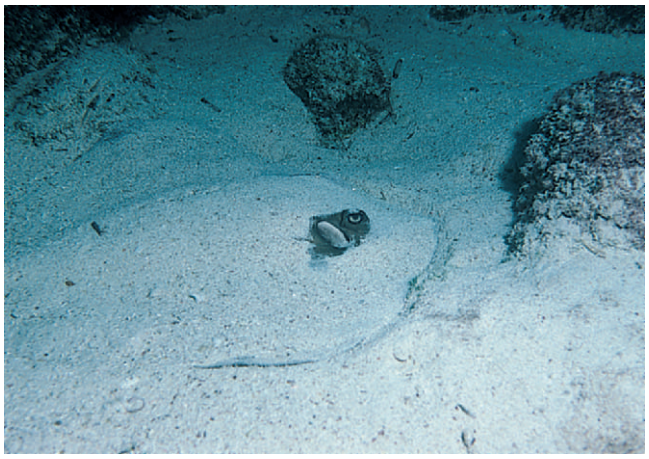


Figure 74-1. Stingray nestled in the sand. Only the eyes and spiracles are visible. (Photo by Paul Auerbach, MD.)

Figure 74-2. Diver cavorting with a large stingray. (Photo by Howard Hall.)

type of injury.[27] The suction force generated by a stingray is sufficient to pull in a large amount of soft tissue, for example, from an obese thigh. This may result in a large and painful contusion or hematoma.

In all cases, the venom apparatus of stingrays consists of a bilaterally retroserrate spine or spines and the enveloping integumentary sheath or sheaths. The elongate and tapered vasodentine spine is firmly attached to the dorsum of the tail (whip) by dense collagenous tissue and is edged on either side by a series of sharp retrorse teeth. Along either edge on the underside of the spine are the two ventrolateral grooves, which

Figure 74-3. Ventral surface of a stingray, demonstrating mouth and gill plates. (Photo by Paul Auerbach, MD.)

house the soft venom glands. The entire spine is encased by the integumentary sheath, which also contains some glandular cells. The sting is often covered with a film of venom and mucus. The spine is replaced if detached.

The venom contains various toxic fractions, including serotonin, 5′-nucleotidase, and phosphodiesterase. Russell[70] investigated the pharmacologic properties of stingray venoms. In animal studies he demonstrated significant venom-induced peripheral vasoconstriction, bradycardia, atrioventricular block, ischemic ST-T wave abnormalities, asystole, central respiratory depression, seizure activity, ataxia, coma, and death. The venom did not appear to be a paralytic neuromuscular agent. Research on stingray venom from the 1950s observed that heating the venom to a temperature above 50° C (122° F) diminished some biologic effects. Haddad analyzed proteins from freshwater stingray *(Potamotrygon falkneri)* venom utilizing SDS-polyacrylamide gel electrophoresis and identified components with gelatinolytic, caseinolytic, and hyaluronidase activities.[42]

Clinical Aspects

Stingray "attacks" are purely defensive gestures that occur when an unwary human wading in shallow waters handles, corners, or steps on a camouflaged creature (Fig. 74-6). The tail of the ray reflexively whips upward and accurately thrusts the caudal spine or spines into the victim, producing a puncture wound or jagged laceration (Fig. 74-7). The integumentary sheath covering the spine is ruptured and venom is released into the wound, along with mucus, pieces of the sheath, and fragments of the spine. On occasion the entire spine tip is broken off and remains in the wound (Fig. 74-8).[71] "Domesticated" stingrays, such as those that congregate at "Stingray City" in the waters of Grand Cayman Island, are habituated to the presence of humans and apparently pose less hazard for a spine puncture, but may still be induced to bite.

A stingray wound from a spine puncture is both a traumatic injury and an envenomation. The former involves the physical damage caused by the sting itself. Because of the retrorse serrated teeth and powerful strikes, significant lacerations can result. Secondary bacterial infection is common. Osteomyelitis may occur if the bone is penetrated. Most injuries occur when the victim steps on a ray; another common cause is handling a

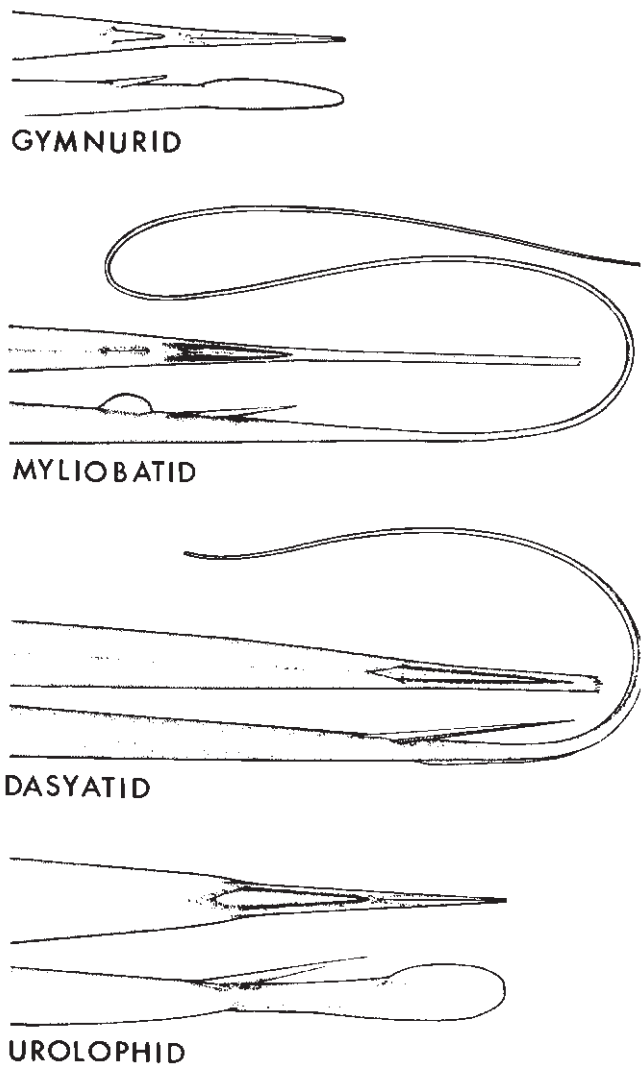


Figure 74-4. Four anatomic types of stingray venom organs.

Figure 74-5. Manta ray. (Photo by Paul Auerbach, MD.)

Figure 74-6. Stingray puncture wound through neoprene boot in typical location near the Achilles tendon. (Photo by Bob Luce.)

ray during its extraction from a fishing net or hook. The lower extremities, particularly the ankle and foot, are involved most often, followed by the upper extremities, abdomen, and thorax. In a rare case, the heart may be directly injured.[69] The tragic death in September 2006 of naturalist Stephen Irwin occurred at Batt Reef off the remote coast of northeastern Queensland, Australia, when he swam directly over a stingray that put a stingray spine into his chest. Death was attributed to a direct heart puncture. He was filming a documentary entitled *Ocean's Deadliest*. There have been two reported cases of survival following cardiac injury.[81] Pseudoaneurysm of the superficial femoral artery caused by stingray envenomation has been reported.[14] Fatalities have occurred after abdominal or thoracic (cardiac) penetration and from exsanguination from the femoral artery. One death has been attributed to tetanus complicating a leg wound. A spine partially or totally denuded of its sheath and venom glands may not cause an envenomation.[28] A detached stingray spine may be used as a weapon. A man stabbed between the shoulder blades with stingray spine suffered a direct spinal cord injury at the level of T7–T8. He was extremely fortunate to make nearly a complete recovery after delayed operative removal.[39]

The envenomation classically causes immediate local intense pain, edema, and variable bleeding. The pain may radiate centrally, peaks at 30 to 60 minutes, and may last for up to 48 hours. The wound is initially dusky or cyanotic and rapidly progresses to erythema and hemorrhagic discoloration, with rapid fat and muscle hemorrhage and necrosis.[9,48] If discoloration around the wound edge is not immediately apparent, within 2 hours it often extends several centimeters from the wound. Hemorrhagic blisters resembling a severe thermal burn or frostbite may occur and may be worsened by overzealous therapeutic hot water immersion.[73,80] Minor stings may simulate bacterial cellulitis. Delayed healing seen following stingray injuries is usually attributed to direct venom toxicity and infections. One analysis of the tissue surrounding a necrotic center

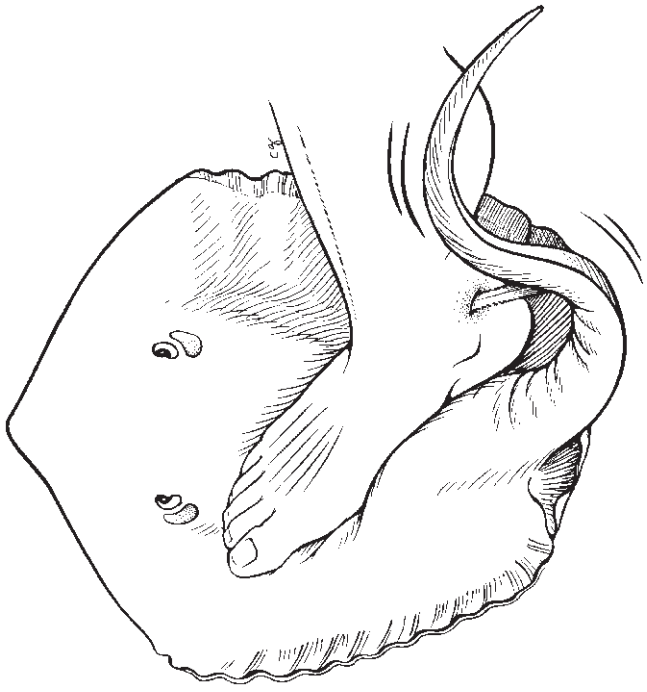


Figure 74-7. The stingray lashes its tail upward into the leg and generates a deep puncture wound.

Figure 74-8. Stingray spine tip broken off into the heel of a victim. (Photo by Robert D. Hayes.)

96 hours after envenomation revealed a perivascular and interstitial mononuclear cell infiltrate with numerous eosinophils and rare neutrophils. The phenotype of the lymphoid population was predominately $CD3^+$ T cells that coexpressed CD4 and contained T cell–restricted intracellular antigen $(TIA)^+$ granules corresponding to the NK1.1 subpopulation of $CD4^+$ T cells. Abundant eosinophils in the vicinity of a stingray soft tissue wound have been noted.[68] All of this is indicative of an immunologic reaction, which, if present, might contribute to the delayed healing of stingray injuries.[35]

Systemic manifestations include weakness, nausea, vomiting, diarrhea, diaphoresis, vertigo, tachycardia, headache, syncope, seizures, inguinal or axillary pain, muscle cramps, fasciculations, generalized edema (with truncal wounds), paralysis, hypotension, arrhythmias, and death.[38,47] The paralysis may represent spastic muscle contractures induced by pain, which are a tremendous hazard for a diver or swimmer.

When handled, a stingray may place its underside adjacent to a human limb or even wrap itself around a leg. The stingray may then bite the victim with a powerful crushing force sufficient to sever a digit or to create a substantial hematoma (Fig. 74-9).

Figure 74-9. Stingray suction bite incurred at "Stingray City," Grand Cayman Island.

Treatment

The success of therapy is largely related to the rapidity with which it is undertaken. Treatment is directed at combating the effects of the venom, alleviating pain, and preventing infection. If hot water for immersion and irrigation (see below) is not immediately available, the wound should be irrigated immediately with nonheated water or saline. If sterile saline or water is not available, tap water may be used. This removes some venom and mucus and may provide minimal pain relief.

As soon as possible, the wound should be soaked in nonscalding hot water to tolerance (upper limit 45°C [113°F]) for 30 to 90 minutes. This might attenuate some of the thermolabile components of the protein venom (although this has never been proven in vivo) or interrupt nerve impulse transmission, and, in some envenomations, relieves pain. Hot water immersion likely has minimal or no effect on the ultimate degree of soft tissue necrosis. There is no indication for the addition of ammonia, magnesium sulfate, potassium permanganate, or formalin to the soaking solution. Under these circumstances they are toxic to tissue and may obscure visualization of the wound. During the hot water soak (or at any time, if soaking is not an option), the wound should be explored and debrided of any

Figure 74-10. Blue-spotted stingray. (Photo by Paul Auerbach, MD.)

readily visible pieces of the sting or its integumentary sheath, which would continue to envenom the victim. Although the standard recommendation is to remove the spine and fragments as soon as possible (to limit the extent of envenomation and pain), if a spine is seen to be lodged in the victim and has acted as a dagger deeply into the chest, abdomen, or neck (this is extremely rare) and may have penetrated a critical blood vessel or the heart, it should be managed as would be a weapon of impalement (e.g., a knife). In this case, the spine should be left in place (if possible) and secured from motion until the victim is brought to a controlled operating room environment where emergency surgery can be performed to guide its extraction and control bleeding that may occur upon its removal.

Cryotherapy is disastrous, and no data yet support the use of antihistamines or steroids. One local remedy, application of the cut surface of half a bulb of onion directly to the wound, has been reported to decrease the pain and perhaps inhibit infection after a sting from the blue-spotted stingray *Dasyatis kuhlii* (Fig. 74-10).[82] The author noted that this approach is used in the Northern Territory of Australia for other fish spine stings, and that the medicinal use of the Liliaceae plant family has been recorded in many cultures. No other folk remedy, including the application of macerated cockroaches, cactus juice, "mile-a-minute" leaves, fresh human urine, or tobacco juice, has been proven effective.[62]

Local suction, if applied in the first 15 to 30 minutes, has been suggested by some clinicians to be of potential value (this is controversial), as may a proximal constriction band (also controversial) that occludes only superficial venous and lymphatic return. If a constriction band is deployed, it should be released for 90 seconds every 10 minutes to prevent ischemia.

Pain control should be initiated during the first debridement or soaking period. Narcotics may be necessary. Local infiltration of the wound with 1% to 2% lidocaine (Xylocaine) or bupivacaine 0.25% (not to exceed 3 to 4 mg/kg total dose in adults; not approved for children under the age of 12 years) without epinephrine may be useful. A regional nerve block may be necessary.

After the soaking procedure, the wound should be x-rayed (Fig. 74-11), then prepared in a sterile fashion, reexplored, and thoroughly debrided, particularly of hemorrhagic fat and obviously necrotic tissue. Wounds should be packed open for delayed primary closure or sutured loosely around adequate drainage in preference to tight closure, which might increase likelihood of wound infection. Another approach that has been mentioned is wound excision followed by packing with an alginate-based wick dressing.[29,62] Prophylactic antibiotics are recommended because of the high incidence of ulceration, necrosis, and secondary infection. Necrotizing fasciitis caused by *Vibrio alginolyticus* has followed stingray injury in a victim with preexisting hepatic cirrhosis.[46] It has also been attributed to *Photobacterium damsela* (formerly *Vibrio damsela*) in a person with normal immunity punctured by a stingray in the tibialis anterior muscle.[8] If the abdominal cavity is penetrated, the victim should receive cefoxitin, clindamycin–gentamicin, or another intravenous regimen intended to cover bowel flora in addition to any antibiotic(s) chosen to cover marine microbes.

If the treatment plan is to treat and release, the victim should be observed for at least 3 to 4 hours for systemic side effects. Properly treated wounds may require a few months to fully heal with complete resolution of local tissue swelling (Fig. 74-12). Wounds that are not properly debrided or explored and cleansed of foreign material may fester for weeks or months.[31] Such wounds may appear infected, but what really exists is a chronic draining ulcer initiated by persistent retained organic matter. Within the first few weeks after an envenomation, a foreign body can sometimes be observed by soft tissue radiograph, ultrasound, or magnetic resonance imaging. After a few weeks, exploration may reveal erosion of adjacent soft tissue structures and the formation of an epidermal inclusion cyst or other related foreign body reaction.[10] As with other marine-acquired wounds, indolent infection should prompt a search for unusual microorganisms. A case of invasive fusariosis *(Fusarium solani)* after stingray envenomation responsive to sequential debridement and ketoconazole (the latter of indeterminate effect) has been reported.[45] Necrotizing fasciitis due to *Photobacterium (Vibrio) damsela* followed a leg laceration caused by a stingray. Notably, the patient had the wound sutured primarily and was not prescribed an antibiotic at the time of the repair.[8] Hyperbaric oxygen therapy has been cited to contribute to wound healing in a refractory case of stingray-induced soft tissue necrosis and postulated infection.[68] Another treatment to accelerate wound healing in a refractory case is topical recombinant human platelet-derived growth factor-BB (becaplermin gel 0.01%) every 12 hours underneath a moist dressing.[6]

Prevention

A stingray spine can penetrate a wetsuit, leather or rubber boot, and even the side of a wooden boat; therefore, a wetsuit or pair of athletic sneakers is not adequate protection. Persons walking through shallow waters known to be frequented by stingrays should shuffle along and create enough disturbance to frighten off any nearby stingrays. The same precautions hold true when accompanied by animals such as horses.[67]

SCORPIONFISH AND SIMILAR VENOMOUS FISH

Scorpionfish are members of the family Scorpaenidae and follow stingrays as perpetrators of piscine vertebrate stings. Distributed in tropical and less commonly in temperate oceans, several hundred species are divided into three groups typified by different genera on the basis of venom organ structure: (1) *Pterois*

Figure 74-11. Radiographs demonstrating stingray spine tip at level of the first metacarpophalangeal joint. **A,** Oblique view. **B,** Lateral view. (Photos by Chris Fee.)

Figure 74-12. Initial severe inflammatory response from stingray puncture near the Achilles tendon. This injury required many weeks to heal. (Photo by Bob Luce.)

(zebrafish, lionfish, and butterfly cod), (2) *Scorpaena* (scorpionfish, bullrout, and sculpin), and (3) *Synanceja* (stonefish). All have a bony plate (stay), which extends across the cheek from the eye to the gill cover. Each group contains a number of different genera and species; at least 80 species of the family Scorpaenidae have been implicated in human injuries or studied anatomically, biochemically, or physiopharmacologically.

Other venomous fish that sting in a manner similar to scorpionfish include the Atlantic toadfish (family Batrachoididae, genus *Thalassophyrne*) (Fig. 74-13), with two venomous dorsal fin spines and venomous spines on the gill covers, and the Pacific ratfish *(Hydrolagus colliei)* (Fig. 74-14) and European ratfish *(Chimaera monstrosa),* both with a single dorsal venomous spine.[41] Toadfish hide in crevices and burrows, under rocks and debris, or in seaweed, sand, or mud. They may change coloration rapidly and remain superbly camouflaged. Rabbitfish (family Siganidae) and leather jacks (leather backs or leather jackets, family Carangidae) carry venomous spines or fins and pose additional risks. Stargazers (family Uranoscopidae) have spines but do not appear to be venomous (Fig. 74-15).

Life and Habits

Zebrafish (lionfish, firefish, or turkeyfish) are beautiful, graceful, and ornate coral reef fish generally found as single or paired free swimmers or hovering in shallow water (Fig. 74-16). They are increasingly popular as aquarium pets and are imported ille-

Figure 74-13. Toadfish. (Photo by Marty Snyderman.)

Figure 74-15. Stargazer *Kathetostoma averruncus.* (© 2006 Norbert Wu / www.norbertwu.com.)

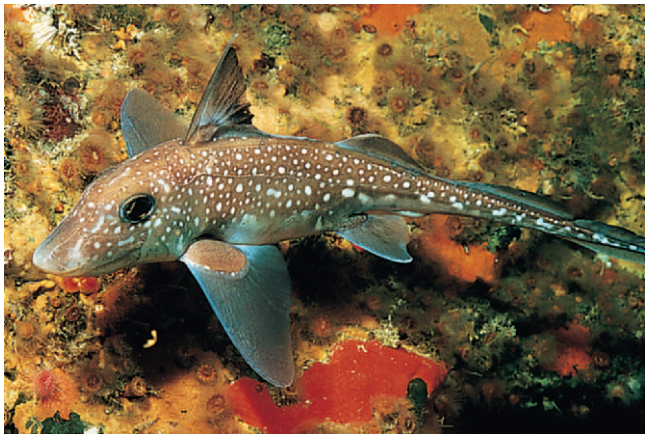


Figure 74-14. Ratfish *(Hydrolagus colliei).* (Photo by Howard Hall.)

gally as part of the "underground zoo." They have recently been introduced to the Atlantic Ocean and have been spotted from North Carolina to South Florida.

Scorpionfish proper *(Scorpaena)* dwell on the bottom in shallow water, bays, coral reefs, and along rocky coastlines, to a depth of 50 fathoms. Their shape and coloration provide excellent camouflage, allowing them to blend in with the ambient debris, rocks, and seaweed (Fig. 74-17). They can be captured by hook and line and serve as important food fish in many areas. The protective coloration and concealment in bottom structures make scorpionfish difficult to visualize. Some species bury themselves in the sand, and most dangerous types lie motionless on the bottom. In the United States, they are found in greatest concentration around the Florida Keys and in the Gulf of Mexico, off the coast of southern California, and in Hawaii.

Stonefish live in shallow waters, often in tide pools and among reefs (Fig. 74-18). They frequently pose motionless and absolutely fearless under rocks, in coral crevices or holes, or buried in the sand or mud. The fish use their pectoral fins to dredge sand or mud from beneath themselves, so that they can settle deeply with only the mouth and eyes exposed.[49] They are so sedentary that algae frequently take root on their skin (Fig. 74-19). They are usually 15 to 20 cm (6 to 8 inches) in length, but can grow to 30 cm (12 inches). Stonefish are not indigenous to North American coastal waters.

Venom and Venom Apparatus

The venom organs are the 12 or 13 (of 18) dorsal (Fig. 74-20), 2 pelvic, and 3 anal spines, with associated venom glands. Although they are frequently large, plumelike, and ornate, the pectoral spines are not associated with venom glands. Each spine is covered with an integumentary sheath, under which venom filters along grooves in the anterolateral region of the spine from the paired glands situated at the base or in the mid-portion of the spine. It is estimated that the two venom glands of each dorsal stonefish spine carry 5 to 10 mg of venom, closely associated with antigenic proteins of high molecular weight (between 50,000 and 800,000).[17] Scorpionfish venom contains multiple toxic fractions and, in the case of stonefish venom, has been likened in potency to cobra venom. It contains a mixture of proteins containing several enzymes, including hyaluronidase.[49] Hyaluronidase is a spreading factor in venoms because it degrades hyaluronate, which helps structure connective tissue. The major toxic component of *Synanceja* venom (stonustoxin) is a protein of molecular weight 148,000 (comprising alpha and beta subunits of molecular weight 71,000 and 79,000, respectively) that is both antigenic and heat labile. Similar purified toxins from other species are trachynilysin from *S. trachynis* (Australian estuarine stonefish) and verrucotoxin (a glycoprotein) from *S. verrucosa* (reef stonefish).[72] The principal action of stonefish venom appears to be direct muscle toxicity, resulting in paralysis of cardiac, involuntary, and skeletal muscles.[36] In an analysis of biologic activity, stonefish (*Synanceja horrida,* the Indian stonefish) venom exhibited edema-inducing, hemolytic, hyaluronidase, thrombinlike, alkaline phosphomonoesterase, 5′-nucleotidase, acetylcholinesterase, phosphodiesterase, arginine esterase, and arginine amidase activities.[50] In a recent evaluation, chromatographic analysis with electrochemical detection showed the presence of substances comigrating with norepinephrine, dopamine, and tryptophan. Serotonin (5-hydroxytryptamine)

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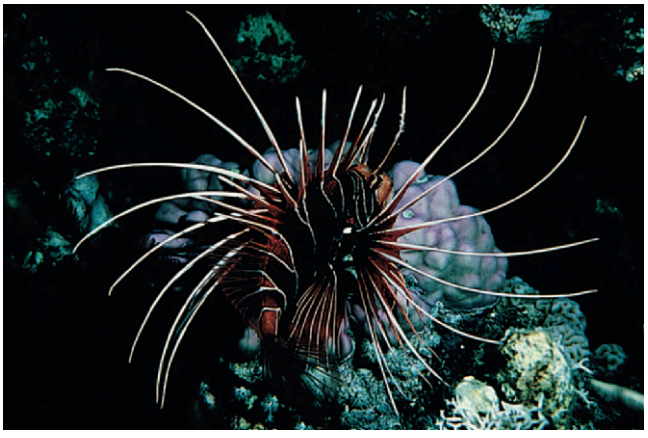

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Figure 74-16. Three examples of lionfish. **A,** Juvenile lionfish from Sulawesi, Indonesia. **B,** Adult lionfish. **C,** Lionfish from the Red Sea. (Photos by Paul Auerbach, MD.)

Figure 74-17. Scorpionfish assuming the coloration of its surroundings. (Photo by Paul Auerbach, MD.)

Figure 74-18. The deadly stonefish *(Synanceja horrida)*. (Photo by Paul Auerbach, MD.)

Figure 74-19. Some stonefish are so sedentary that algae grow on their skin. (Photo by Paul Auerbach, MD.)

was not detected.[34] Crude venom of the stonefish *Synanceja verrucosa* possesses numerous enzymatic properties, including hyaluronidase, 8 esterases, and 10 aminopeptidases.[33] Intracellular Ca^{2+} levels are increased by venoms of the soldierfish *(Gymnapistes marmoratus),* lionfish *(Pterois volitans),* and stonefish *(Synanceia trachynis),* possibly via the formation of pores in the cellular membrane, which, under certain conditions in experimental animals, may lead to necrosis.[22] In addition, trachynilysin activity upon the heart, often noted as negative inotropy, may also be a function of Ca^{2+} influx.[72] The hemolytic activity of stonustoxin may in some part depend on surface tryptophan residues.[84] The cardiovascular effects of stonefish venom have been attributed in part to its activity at muscarinic receptors and adrenoceptors, and pain effects perhaps to its activity at bradykinin receptors.[18] Similar receptor activity, neutralized by stonefish antivenom, has been noted with lionfish venom.[20] The pain-causing protein in bullrout *(Notesthes robusta)* venom is an algesic protein (169.8 to 174.5 kDa) called *nocitoxin*.[43]

Stonefish venom causes pulmonary edema in laboratory animals, which may reflect a general vascular permeability.[50,54] It also causes species-restricted (nonhuman) hemolysis and platelet aggregation.[51] Profound endothelial relaxation may contribute directly to hypotension.[57] The neuromuscular toxicity appears to be a consequence of the venom's dose-dependent presynaptic and postsynaptic actions at the myoneural junction, which include release and depletion of neurotransmitter from the nerve terminal, followed by irreversible depolarization of muscle cells and microscopically observable muscle and nerve damage.[55] Hypotension observed in envenomed laboratory animals may be due in part to binding to receptors on endothelial cells, causing production of nitrous oxide and activation

Figure 74-20. Scorpionfish spines. (Photo by Kenneth Kizer, MD.)

of potassium channels.[77] The nondialyzable opalescent venom retains full potency for at least 24 to 48 hours after the death of a scorpionfish.[53] Extrapolating from the LD_{50} of 0.36 µg/g in mice, it is estimated that 18 mg relayed by six intact spines might cause death in a 60-kg (132 lb) human.[50]

Pterois species carry long, slender spines with small venom glands covered by a thin integumentary sheath. An extract of lionfish spine tissue contains acetylcholine and a toxin that affects neuromuscular transmission.[23] *Scorpaena* species carry longer heavy spines with moderate-sized venom glands covered by a thicker integumentary sheath. *Synanceja* species carry short, thick spines with large, well-developed venom glands covered by an extremely thick integumentary sheath (Figs. 74-21 and 74-22). However, the skin over the venom gland is loosely attached, so when a human treads on the fish, the skin is pushed down the spine and the venom gland is compressed by the crumpled sheath. The pressure forces the venom gland to empty up the paired narrow ducts so that venom and glandular tissue spurt into the wound.[37]

When any of these fish is removed from the water, handled, stepped on, or otherwise threatened, it reflexively erects the spiny dorsal fin and flares out the armed gill covers and pectoral and anal fins. If provoked while still in the water, it actually attacks. The venom is injected by a direct puncture wound through the skin, which tears the sheath and may fracture the spine, in a manner analogous to that of a stingray envenomation. Fishermen are commonly injured, particularly when emptying nets or extracting hooks from captured fishes.[40]

Clinical Aspects

Native residents of the Indo-Pacific islands have great fear of a sting from the dreaded venomous stonefish, such as the "ikan hantu" (devil fish), Tahitian "nohu" ("nofu" or "no'u," the waiting one) or the Australian "warty ghoul." The presentation of the injury is similar to that of stingray envenomation in that the unwary diver or fisherman steps on or handles the fish. In the United States, marine aquarists and beneficiaries of illegal importation of tropical animals are increasingly envenomed as they unknowledgeably handle *Pterois volitans, P. radiata,* or *Scorpaena guttata*. In Indo-Pacific waters, envenomations of the foot and lower extremity are more commonly caused by the

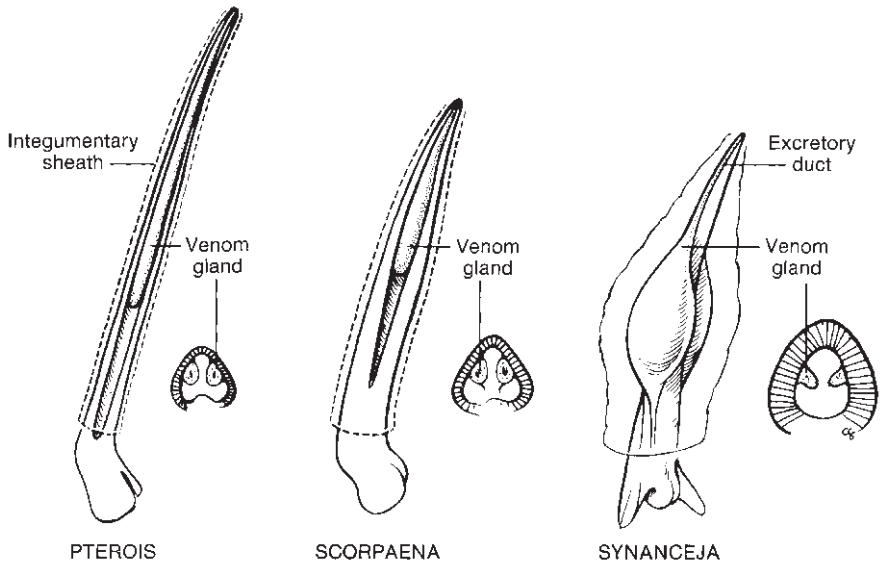


Figure 74-21. Lionfish (*Pterois*), scorpionfish (*Scorpaena*), and stonefish (*Synanceja*) spines with associated venom glands.

Figure 74-22. Spines of the venomous stonefish, demonstrating venom glands. (Courtesy John Williamson, MD.)

Figure 74-23. Stonefish puncture wound. (Photo by Dr Richard Lyon.)

stonefish, such as *Synanceja horrida*, *S. trachynis*, or *S. verrucosa*. Scorpionfish stings vary according to the species, with a progression in severity from the lionfish (mild) through the scorpionfish (moderate to severe) to the stonefish (severe to life-threatening). The severity of the envenomation depends on the number and type of stings, species, amount of venom released, and age and underlying health of the victim. Pain is immediate and intense, with radiation centrally. Untreated, the pain peaks at 60 to 90 minutes and persists for 6 to 12 hours. With a scorpionfish or stonefish envenomation, the pain may be severe enough to cause hallucinations or delirium and may persist at high levels for hours (scorpionfish) or days (stonefish).[40] The wound and surrounding area are initially ischemic and then cyanotic (Fig. 74-23), with more broadly surrounding areas of erythema, edema, and warmth. Vesicles may form (Fig. 74-24). Human (hand) vesicle fluid after the sting of the lionfish *Pterois volitans* was analyzed for mediators of inflammation and demonstrated an appreciable quantity of prostaglandin $F_{2\alpha}$; thromboxane B_2, prostaglandin E_2, and 6-keto-prostaglandin $F_{1\alpha}$ were present in negligible quantities. Whether or not residual venom is present in blister fluid is a matter of conjecture. Rapid tissue sloughing and close surrounding areas of cellulitis, with anesthesia adjacent to peripheral hypesthesia, may be present within 48 hours. Necrotic ulceration is rare, but may occur after a lionfish envenomation.[66] Severe local tissue reaction is more common after the sting of a scorpionfish or stonefish.

Systemic effects include anxiety, headache, tremors, maculopapular skin rash, nausea, vomiting, diarrhea, abdominal pain, diaphoresis, pallor, restlessness, delirium, seizures, limb paralysis, peripheral neuritis or neuropathy, lymphangitis, arthritis, fever, hypertension, respiratory distress, bradycardia, tachycardia, atrioventricular block, ventricular fibrillation, congestive heart failure, pericarditis, hypotension, syncope, and death. Pulmonary edema is a bona fide sequela.[56] Death in humans, which is extremely rare, usually occurs within the first 6 to 8 hours. The wound is indolent and may require months to heal, only to leave a cutaneous granuloma or marked tissue

Figure 74-24. Vesiculation of the hand 48 hours after the sting of a lionfish. (Photo by Howard McKinney.)

defect, particularly after a secondary infection or deep abscess. Mild pain may persist for days to weeks. Lionfish envenomation has been used as a fabricated chief complaint to seek a prescription for narcotic drugs.[76] After successful therapy, paresthesias or numbness in the affected extremity may persist for a few weeks.

Treatment

As soon as possible, the wound or wounds should be immersed in nonscalding hot (upper limit 45°C [113°F]) water to tolerance. This may inactivate at least one of the thermolabile components of the protein venom that might otherwise induce a severe systemic reaction. Platelet aggregation in blister fluid is inhibited by heat treatment, which suggests that the venom or some other active component may be neutralized. The soak should be maintained for a minimum of 30 minutes and may continue for up to 90 minutes. Recurrent pain that develops after an interval of 1 to 2 hours may respond to a repeat hot water treatment. As soon as is practical, all obvious pieces of spine and sheath fragments should be gently removed from the wound. Vigorous irrigation should be performed with warmed sterile saline to remove any integument or slime. If pain is severe or inadequately controlled (in terms of degree or rapidity or relief) by hot water immersion, local tissue infiltration with 1% to 2% lidocaine without epinephrine or regional nerve block with an anesthetic such as 0.25% bupivacaine may be necessary. After injection with a local or regional anesthetic, the hot water immersion should be discontinued or closely observed to avoid inadvertent creation of a burn wound in the now insensate body part. Infiltration with emetine hydrochloride, potassium permanganate, or Congo red has been abandoned, despite reports of favorable experiences with acidic emetine. The biochemical bases for the success of folk remedies, such as the application of meat tenderizer, mangrove sap, or green papaya (papain), have yet to be confirmed. The effectiveness of alternative remedies may be related to the protein behavior of the venom, which is inactivated by heat, extremes of pH (it is partially inactivated at pH of greater than 8.6 and completely at a pH of less than 4), hydrogen peroxide, iodine, and potassium permanganate (which is, unfortunately, tissue toxic). Currently, no data are available to support the topical administration of empirical remedies, such as mineral spirits, organic dye, ground liver, or formalin. Cryotherapy is absolutely contraindicated, to avoid an iatrogenous cold-induced injury.

Although the spine rarely breaks off into the skin, the wound should be explored to remove any spine fragments, which will

Figure 74-25. Stonefish antivenom. (Courtesy John Williamson, MD.)

otherwise continue to envenom and act as foreign bodies, perpetuating an infection risk and poorly healing wound. If the spine has penetrated deeply into the sole of the foot, surgical exploration should be performed in the operating room with magnification. Vigorous warmed saline irrigation should be performed. Wide excision and debridement are unnecessary. Because of the nature of the puncture wound, tight suture or surgical tape closure should not be undertaken; rather, the wound should be allowed to heal open with provision for adequate drainage. If the puncture wound is high risk (deep, into the hand or foot or both), prophylactic antibiotic(s) should be administered. It is probably wise to remove blister fluid using aseptic technique.

A stonefish antivenom is manufactured by the Commonwealth Serum Laboratories (CSL Limited, Parkville, Victoria, Australia) (Fig. 74-25). In cases of severe systemic reactions from stings of *Synanceja* species, perhaps from soldierfish *(Gymnapistes marmoratus)* or bullrout (*Notesthes* robusta), and rarely from other scorpionfish, it is administered intramuscularly or diluted for intravenous administration.[19,25] The antivenom is supplied in vials containing 1.5 to 3 mL of liquid containing 2000 units of hyperimmune $F(ab')_2$ horse serum active against *Synanceja trachynis,* with 1000 units (one half a vial) capable of neutralizing 10 mg of dried venom.[24] $F(ab')_2$ preparations are obtained by pepsin treatment of IgG at pH 2, whereas Fab fragments are produced by papain treatment at pH 7 to 8.[78] The former product is believed to be easy to standardize

than the latter, and better in its plasma distribution and venom neutralization. After skin testing to estimate the risk for an anaphylactic reaction to equine sera, the antivenom should be given. As a rough estimate, one vial should neutralize one or two significant stings (punctures). For one or two puncture wounds, administer 1 vial; for three or four puncture wounds, 2 vials; for more than four puncture wounds, administer 3 vials. One or more additional vials may be necessary if there is recurrent severe pain. When not in use, the antivenom should be protected from light and stored at 2° to 8°C (35.6° to 46.4°F), never frozen. Unused portions should be discarded.

The fact that stonefish antivenom cross-reacts with most piscine venoms suggests that piscine venoms may possess structural similarities in addition to their functional similarities, which include the induction of profound cardiovascular changes, release of nitric oxide from endothelial cells, smooth muscle contraction, depolarizing action on nerve and muscle cells, and potent cytolytic activity.[21]

Prevention

The most effective way to prevent envenomation is to avoid handling a scorpionfish. A diver should make a careful inspection before setting down on the bottom. Amateur aquarists should be exceedingly cautious when handling exotic tropical fish. Seemingly dead fish may yield an unpleasant surprise for the unwary.

CATFISH

Life and Habits

Approximately 1000 species of catfish inhabit both fresh and salt waters; many of these are capable of inflicting serious stings. Marine animals include the Oriental catfish (*Plotosus lineatus*), which lurks in tall seaweed and can inflict extremely painful stings, the larger sailcat *(Bagre marinus),* and the common sea catfish *(Galeichthys felis),* which hovers along the sandy bottom. The coral catfish *(Plotatus lineatus)* has also been reported to sting humans.[74] Ocean catfish, particularly juveniles, "swarm" and feed along the bottom (Fig. 74-26). There are 39 species of catfishes native to the North American continent. Freshwater catfish of North America include the brown bullhead *(Noturus nebulosus),* Carolina madtom *(N. furiosus),* channel *(Ictalurus punctatus),* blue *(I. furcatus),* and white *(N. catus)* catfish.

The catfish derives its name from the well-developed sensory barbels ("whiskers") surrounding the mouth. The barbels of catfish carry well-developed sensory organs that are used to transmit both touch and taste. All catfishes are adapted to foraging in muddy and dark waters where feeding by senses is essential.

Catfish possess a slimy skin without any true scales. Marine catfish, unlike freshwater catfish, frequently travel in large schools. Most freshwater catfish are bottom feeders noted for their junkyard diet. They are poor swimmers and not very evasive.

The South American astroblepids have flattened suctorial lips that allow them to scale cliffs. Tiny South American (Amazonian) catfish of the genus *Vandellia* (species *cirrhosa, balzanii, plazaii, sanguinea,* and *becarrii*) are known as "urethra fish" in English, *candirú* by Brazilians, and *canero* by Spanish speakers.[12] Approximately 2.5 to 7.5 cm (1 to 3 inches) long, they carry short spines on their gill covers (Fig. 74-27). This "vampire fish" is predominately a bottom-feeding "junkfish" found in murky or muddy waters in the Amazon and Orinoco Rivers and perhaps selected tributaries and is putatively attracted to urine (water motion, warmth). It can swim up the human urethra or other urogenital apertures, where it extends the spiny gill covers and thus becomes embedded, preventing removal by pulling on the fish's tail. Within the gills of a fish, it anchors itself with its spines and rasps with teeth to obtain a blood meal. Within the human urethra, it causes extreme pain and inflammation. Because the animal normally seeks the outflow stream from a larger fish's gills (where it may enter and parasitize the host fish), perhaps it is not urinophilic, but merely swimming into a stream. Others theorize that it is attracted to ammonia. Natives wear pudendal shields when urinating in natural bodies of water. A tight-fitting bathing suit is certainly prudent.

Figure 74-26. Juvenile ocean catfish. (© 2006 Norbert Wu / www.norbertwu.com.)

At best, extraction is painful. Amputation of the penis by natives has been described in the older literature. Ingestion of the green fruit of the jagua (xagua or xaqua) tree or buitach apple (*Genipa americana* L.) as a concoction (tea) apparently works to dispel the urethra-lodged candirú by the action of a large quantity of citric acid (megadose vitamin C), which softens calcium spines. Other references cite placement of either or both plants (or their extracts) within the urethra (or other invaded body orifice) in order to dispatch and dissolve the fish. One recent case requiring surgical removal from the urethral tract of a male was graphically documented on the Internet by Dr. Anoar Samad.

Venom and Venom Apparatus

The venom apparatus of the catfish consists of the single dorsal and two pectoral fin spines ("stings") and the axillary venom

glands. Both the dorsal and pectoral spines are exquisitely sharp and can be locked into an extended position by the fish when it is handled or becomes excited. The spines are enveloped by glandular tissue within an integumentary sheath; some spines are barbed or have sharp retrorse teeth. Scattered reports note envenomation in persons who handled only the tail of the fish, such as the Arabian Gulf catfish *(Arius thalassinus)*, which suggests the presence of a toxic skin secretion (crinotoxin). Other observers note that toxin released from epidermal skin cells can cause throbbing pain, tissue necrosis, and perhaps muscle fasciculations.[32] Oriental catfish toxin, which is poorly antigenic, contains vasoconstrictive, hemolytic, edema-forming, dermatonecrotic, and other biogenic fractions.[75] It behaves in vivo much like a milder version of stingray venom. In contrast, the crinotoxin of the Arabian Gulf catfish contracts smooth muscle and stimulates the release of prostaglandins; pretreatment with atropine and indomethacin attenuates the response.[2,74] Furthermore, wound healing responses are accelerated by repeated local application of preparations from the epidermal secretions of another Arabian gulf catfish (*Arius bilineatus*, Valenciennes).[3]

Figure 74-27. Amazonian catfish (candirú), which can enter the human urethra. (Photo courtesy V. Haddad, Jr.)

Clinical Aspects

Most stings are incurred when a fish is handled, which creates an injury out of proportion to the mechanical laceration. When the spine penetrates the skin, the integumentary sheath is damaged, and the venom gland exposed. Catfish stings are described as instantaneously stinging, throbbing, or scalding, with central radiation up the affected limb. Normally the pain subsides within 30 to 60 minutes, but in severe cases it can last for 48 hours. The area around the wound quickly appears ischemic, with central pallor that gradually becomes cyanotic before the onset of erythema and edema. Swelling can be severe, and secondary infections are frequent; gangrenous complications have been reported. Common side effects include local muscle spasm, diaphoresis, and fasciculations. Bleeding from the puncture wounds may be more severe than expected. Less common sequelae are peripheral neuropathy, lymphedema, adenopathy, lymphangitis, weakness, syncope, hypotension, and respiratory distress. Death is extremely rare. "Finning" occurs when a person is punctured by a fin while handling a fin. This often occurs when removing a hook from a fish or a fish from a net. In one instance of catfish finning, in addition to the immediate typical immediate toxic reaction, the victim suffered recurrent episodes of pain and swelling on the dorsum of the hand over the course of 6 months, which eventually led to spontaneous skin rupture and blood-tinged fluid drainage.[1] Plain radiography revealed two catfish spines embedded in the soft tissues between the third and fourth metacarpal bones. Thirteen months after the initial injury, one spine was removed easily with local exploration; more extensive surgery did not lead to successful localization of the second spine, but revealed extensive edematous tenosynovitis. Because of the clinical course, the patient was treated presumptively for *Mycobacterium marinum* infection. In another case, the radial artery was lacerated by a spine that became embedded in the volar-radial aspect of the nondominant wrist (Fig. 74-28).[30] This was repaired by lateral arteriorrhaphy rather than segmental resection and reanastomosis (Fig. 74-29).

The sting of the marine catfish is usually more severe than that of its freshwater counterparts and may have a propensity to more local hemorrhage.[61] Infection risk is similar to that for any aquatic-acquired wound, in that *Vibrio* and *Aeromonas*

Figure 74-28. Plain films with a catfish fin in the volar wrist. (Photo courtesy of Ekkehard Bonatz.)

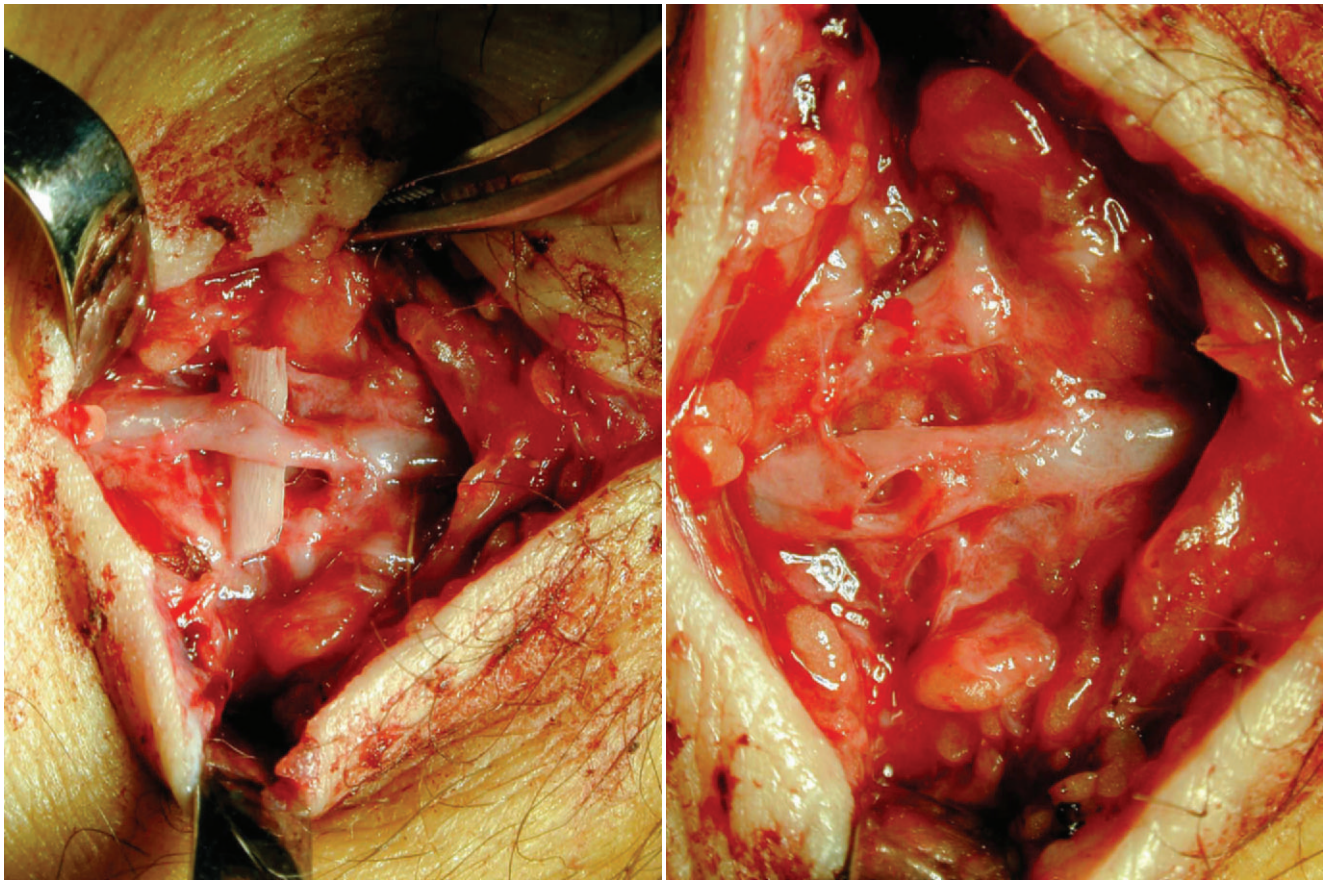
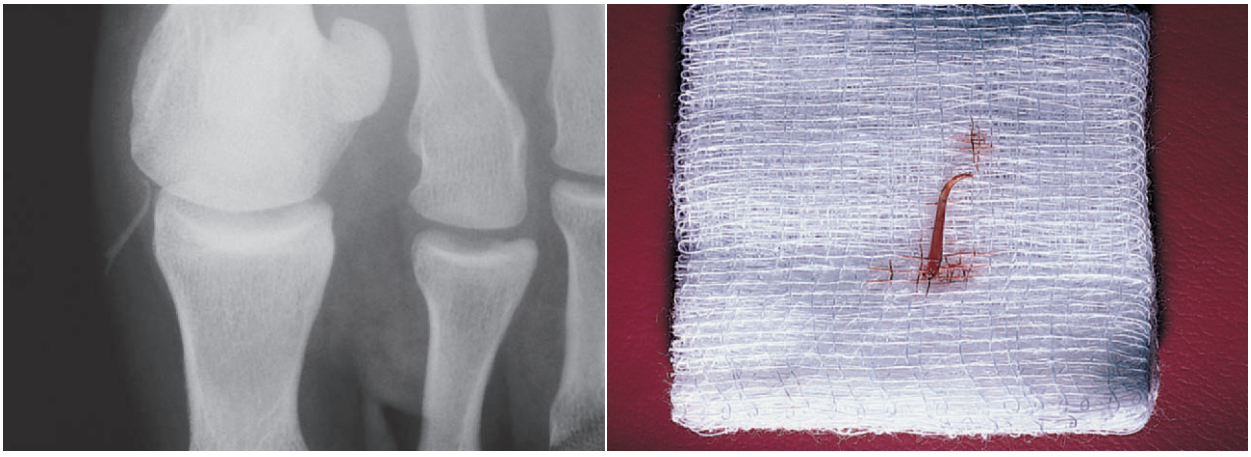


Figure 74-29. **A,** Catfish spine piercing the radial artery. **B,** Radial artery with residual holes from catfish spine, which has been removed. **C,** The retro-barbed structure of the catfish spine is apparent. (Photos courtesy of Ekkehard Bonatz.)



A B

Figure 74-30. Catfish spine lodged in the foot. **A,** Radiograph demonstrating the presence of a foreign body. **B,** The spine removed. (Photos by Paul Auerbach, MD.)

species may be pathogens and the infection may be polymicrobial.[63,64] Other organisms that have been reported to be associated with marine or freshwater catfish-related injuries include *Edwardsiella tarda, Citrobacter freundii, Fusobacterium mortiferum, Morganella morganii, Providencia rettgeri, Enterococcus faecalis, Pseudomonas aeruginosa, Mycobacterium terrae,* and *Enterobacter cloacae.*[64] *E. tarda* is a gram-negative bacillus of the family Enterobacteriaceae that is mainly associated with aquatic environments and the animals that inhabit them, particularly catfish and other cold-blooded animals.[4,7] It may be a pathogen for eels and catfish. If *E. tarda* infection is determined, it is sensitive in vitro to ampicillin, aminoglycosides, β-lactamase stable cephalosporins, quinolones, tetracycline, and trimethoprim–sulfamethoxazole.[7]

Treatment

There are no specific antidotes. As with stingray and scorpionfish envenomations, the success of therapy is related to the rapidity with which it is undertaken. With catfish envenomations, in contrast to those of stingrays, constriction bandages have never been recommended for first aid. The wound should be immediately immersed in nonscalding hot water to tolerance (upper limit 45° C [113° F]) for 30 to 90 minutes or until there is significant pain relief. This may inactivate heat-labile components of the venom and perhaps helps to reverse local toxin-induced vasospasm. There is no evidence that adding mineral salts, solvents, antiseptics, or other chemicals to the water is of additional benefit. Cryotherapy is not efficacious. A popular and unstudied local (U.S. rural) remedy is to rub the sting with skin mucus (slime) from the catfish. If the hot water soak is not sufficient to control pain, local infiltration of the wound with buffered (alkalinized) bupivacaine or lidocaine without epinephrine or a regional nerve block may be necessary. It has been theorized that the pH alteration offered by the alkalinized local anesthetic may neutralize venom.[58] The wound should be explored surgically to remove all spine and sheath fragments. Standard radiographs or soft tissue exposures may locate a radiopaque foreign body (Fig. 74-30). The wound should be left unsutured to heal, to allow adequate drainage and minimize the risk of infection. All wounds must be carefully observed for infection until healed. If the puncture wound is high risk (i.e., deep or into the hand or foot), prophylactic antibiotic(s) should be administered.

Figure 74-31. Greater weeverfish *(Echiichthys draco),* Mediterranean Sea. (Photo by H. Göthel.)

Prevention

Catfish should be handled without grabbing the dorsal or pectoral fins, preferably by using a mechanical instrument or gaff. If possible, *Plotosus lineatus* should not be handled at all.

WEEVERFISH

Life and Habits

The weeverfish (*Echiichthys* species, formerly named *Trachinus*) (Fig. 74-31) is the most venomous fish of the temperate zone. It is found in the Black Sea, Mediterranean Sea, eastern Atlantic Ocean, North Sea, and European coastal areas. Common names for the weeverfish include the adder-pike, sea dragon, sea cat, and stang. Weeverfish are small (10 to 53 cm [4 to 21 inches]) marine creatures that inhabit flat sandy or muddy bays, usually

burying themselves in the soft bottom with only the head partially exposed. They lead sedentary lives but when provoked can strike out with unerring accuracy. Weevers are terrors to fishermen working in shallow sandy areas.

Venom and Venom Apparatus

The venom apparatus consists of four to eight elongate and needle-sharp dorsal (up to 4.5 cm [1.75 inches] in length) and two opercular and daggerlike dentinal spines, associated holocrine glandular tissue, and thin, enveloping stratified squamous epithelium integumentary sheath. When excited, the fish extends the dorsal fin and expands the operculum, projecting the opercular spine out at a 35- to 40-degree angle from the longitudinal axis of the body. Weeverfish survive for hours out of the water, and the toxin remains potent for hours in dead animals, particularly when they are well refrigerated. Although incompletely characterized, the unstable (heat-labile) protein venom (ichthyoacanthotoxin) contains several peptides, at least one protein of high molecular weight (324,000), and possibly 5-hydroxytryptamine, epinephrine, norepinephrine, histamine, and mucopolysaccharide components. To date, serotonin has not been identified in weeverfish venom. The greater weeverfish *(Echiichthys draco)* releases a protein venom, dracotoxin, which has membrane depolarizing and hemolytic activities. It appears to be a single polypeptide of molecular weight 105,000.[16] Other weeverfish of significance include *E. vipera, E. radiatus,* and *E. lineolatus*.

Clinical Aspects

Weeverfish stings usually afflict professional fishermen or vacationers who wade or swim along sandy coastal areas. The thrust of the spine is sufficient to penetrate a leather boot and creates a substantial puncture wound. The integumentary sheath is torn, and venom is injected into the wound. The onset of pain is instantaneous, described as intensely burning or crushing, and spreads rapidly to involve the entire limb. The pain usually peaks at 30 minutes and subsides within 24 hours but can last for days. Its intensity can induce irrational behavior and syncope; even narcotics are poorly effective. An account dating from 1782 informs that a fisherman amputated his own finger to alleviate the pain caused by a weeverfish sting.[13] If an upper extremity is envenomed, the pain may radiate into the thorax and mimic the symptoms of myocardial ischemia.[44] The puncture wound bleeds little and often appears pale and edematous initially. The sting of *E. vipera* may bleed freely. Over the course of 6 to 12 hours the wound becomes erythematous, ecchymotic, and warm. The edema may increase for 7 to 10 days, causing the entire limb to become markedly swollen. Secondary bacterial infections are common, and gangrene has been reported. The indolent wound may require months to heal, depending on the nature of the sting and underlying health of the victim. Raynaud's phenomenon in an envenomed digit occurring a few weeks after a weeverfish sting has been reported.[15] This may develop in a delayed fashion and persist for months after envenomation.[60] Persistent edema has been noted to last for more than 1 year.

Systemic symptoms associated with weeverfish envenomation include headache, delirium, aphonia, fever, chills, dyspnea, diaphoresis, cyanosis, nausea, vomiting, seizures, syncope, hypotension, and cardiac arrhythmias. Death has been reported, perhaps attributable to direct intravascular injection of venom.[11]

Treatment

The wound should be immersed immediately in nonscalding hot water to tolerance (upper limit 45° C [113° F]) for 30 to 90 minutes or until there is significant pain relief. This may inactivate heat-labile components of the venom and perhaps helps reverse local vasospasm that might contribute to local sequestration of venom and the inhibition of free bleeding. The addition of mineral salts, ammonia, vinegar, urine, or other substances to the water is of no proven value. Immersion in hot water is often a less successful therapy for a weeverfish sting than for that of a scorpionfish. When the heat inactivation method is inadequate to control pain, it is necessary to infiltrate the wound with a local anesthetic (1% to 2% lidocaine without epinephrine) or perform a regional nerve block. The liberal use of narcotics is often required. Prolonged immersion cryotherapy is contraindicated. However, a practice known as "thermic shock" has been touted by practitioners along the French Mediterranean coast. This consists of application of intense local temperature variation (heat for 2 to 10 minutes, followed by application for 10 to 30 minutes of an ice cube insulated within a tissue or thin cloth).

Rarely, a spine breaks off into the skin. The wound should be explored gently, all fragments of sheath should be removed, and the wound should be irrigated vigorously with warmed saline. Wide excision and debridement are unnecessary. Because of the nature of the puncture wound, tight suture or surgical tape closure should not be undertaken; rather, the wound should be allowed to heal open with provision for adequate drainage. If the puncture wound is high risk (i.e., deep or into the hand or foot), prophylactic antibiotic(s) should be administered. No commercial antivenom is currently available. An effective experimental antivenom was created at the Institute of Immunology in Zagreb, Yugoslavia, but is no longer available.

Prevention

Weeverfish hide in bottom sand and mud; thus persons must shuffle along with adequate footwear. These fish are easily provoked and should be avoided by scuba divers. They should never be handled alive and must be treated with extreme caution even when dead. Weeverfish survive for hours out of the water, and careless handling of a seemingly dead fish may result in an envenomation.

VENOMOUS (HORNED) SHARKS

Life and Habits

Horned sharks are species that possess dorsal fin spines. In the United States the group is essentially limited to the spiny dogfish *(Squalus acanthias)* (Fig. 74-32). These and similar animals are distributed throughout sub-Arctic, temperate, tropical, and sub-Antarctic seas. The Port Jackson shark *Heterodontus portusjacksoni* (Fig. 74-33) is particularly dangerous.

The fish are sluggish and prefer cooler water and shallow protected bays. They are erratic in their migration and may be found singly or in schools. Voracious feeders, they eat other fish, coelenterates, mollusks, crustaceans, and worms.

The venom apparatus consists of a spine anterior to each of two dorsal fins and the associated venom glands.

Clinical Aspects

As with other vertebrate stings, there is immediate intense stabbing pain that may last for hours and is accompanied by

Figure 74-32. Spiny dogfish *(Squalus acanthias).* (© 2006 Norbert Wu / www.norbertwu.com.)

Figure 74-33. Port Jackson shark. (Photo by Marty Snyderman.)

erythema and edema. Although systemic side effects are rare, fatalities are possible.

Treatment

Treatment is the same as for stingray envenomation.

SURGEONFISH

Life and Habits

The surgeonfish (doctorfish, or tang) is a tropical reef fish of the family Acanthuridae that carries one or more retractable jackknife-like epidermal appendages ("blades") on either side of the tail (Fig. 74-34). When the fish is threatened, the blade may be extended out at a forward angle, where it serves to inflict a laceration. There does not appear to be any associated envenomation.

Clinical Aspects

A victim cut by a surgeonfish notes a laceration or deep puncture wound that is immediately painful; it usually bleeds freely. The pain is moderate to severe and of a burning nature. Systemic reactions are infrequent and consist of nausea, local muscle aching, and apprehension.

Figure 74-34. Surgeonfish "blades." (Photo by Paul Auerbach, MD.)

Treatment

The wound should be irrigated and then soaked in nonscalding hot water to tolerance (upper limit 45°C [113°F]) for 30 to 90 minutes or until pain is relieved, although this may be of variable efficacy. It should be scrubbed vigorously to remove all foreign material and watched closely for the development of a secondary infection. Unless absolutely necessary for hemostasis, sutures should not be used to close the wound.

PLATYPUS

Venom and Venom Apparatus

The platypus *Ornithorhynchus anatinus* is a furry venomous mammal that inhabits riverine systems of eastern Australia between northern Queensland and southern Tasmania.[26] These strange, fat animals have bills like a duck, webbed feet, a paddlelike tail, and claws on the feet. The male animal has an erectile keratinous spur on each hind limb linked via a distensible duct to a venom gland. The spur attains a length of 15 mm ($^{9}/_{16}$ inch) in mature animals. There is a duct on each side that connects the spur to a venom gland situated under the thigh muscles. The venom appears to have components that mediate a type I hypersensitivity reaction with mast cell degranulation, which is consistent with the clinical presentation of soft tissue edema. Other venom fractions include a natriuretic peptide, proteases, and hyaluronidase. Venom-induced local edema in laboratory rats is attenuated by ketanserin and, to a lesser degree, by cimetidine, which may indicate a role of 5-hydroxytryptamine and histamine in the pathogenesis of the envenomation.

Clinical Aspects

Normally, the platypus is a shy creature. However, when provoked, it grasps its opponent with the hind legs and thrusts a spur or spurs into the victim. From 2 to 4 mL of venom may be released. When a human is envenomed, symptoms include immediate severe pain, tissue edema, and prolonged local sensitivity to painful stimuli. Movement, even remote (such as coughing), worsens the pain. The pain and hyperesthesia may generalize for several days before the pain recedes back to the envenomed limb. The pain may last for weeks, and in a severe case, muscle mass may be lost.

Figure 74-35. Olive sea snake. (Photo by Michele Hall.)

Treatment

Therapy is supportive and includes pain medication, wound care, and physical therapy after the acute episode. Hot water immersion does not appear to be of benefit acutely. Short-term corticosteroid therapy has been suggested to diminish pain and mitigate swelling, but there is no proof that anti-inflammatory agents are definitively useful.

SEA SNAKES

Life and Habits

Sea snakes (Fig. 74-35) of the family Hydrophiidae (subfamilies Hydrophiinae [genera *Hydrophis, Hydrelaps, Kerilia, Thalassophina, Enhydrina, Acalyptophis, Thalassophis, Kolpophis, Lapemis, Astrotia, Pelamis,* and *Microcephalophis*] and Laticaudinae [genera *Laticauda, Aipysurus,* and *Emydocephalus*]) are probably the most abundant reptiles on earth. There are at least 52 species, all venomous. Species implicated in serious envenomations or human fatalities include *Astrotia stokesii, Enhydrina schistosa, Hydrophis ornatus, Hydrophis cyanocinctus, Lapemis hardwickii, Pelamis platurus,* and *Thalassophis viperina.*

The snakes are distributed in the tropical and warm temperate Pacific and Indian Oceans, with the highest number of envenomations occurring along the coast of Southeast Asia, in the Persian Gulf, and in the Malay Archipelago. No sea snakes live in the Atlantic Ocean or in the Caribbean Sea. Hawaii is the only U.S. state that has sea snakes (predominantly *Pelamis platurus*). The Pacific snakes usually inhabit sheltered coastal or coral reef waters and congregate about river mouths, and only on rare occasion do they venture into the open ocean. *P. platurus,* the most widely distributed sea snake, is pelagic and may be found in the Pacific coastal waters of Central and South America. It does not migrate to the Caribbean because of the freshwater barrier of Gatun Lake in the center of the Panama Canal.

Although sea snakes have the general appearance of land snakes, true sea snakes and sea kraits have valvelike nostril flaps and rudimentary ventral plates, without gills, limbs, ear openings, sternum, or urinary bladder. Most species of sea snakes are 0.9 to 1.2 m (3 to 4 feet) long, but some attain lengths of up to 2.7 m (9 feet). They are sinuous scaled creatures whose bodies are compressed posteriorly into a flat, paddle-shaped tail designed for marine locomotion (Fig. 74-36). They swim in an undulating fashion and can move backward or forward in the water with equal speed. On land, however, they are awkward and do not survive readily. They may be brightly colored, such as the yellow-bellied sea snake, *P. platurus*. With a single lung, the sea snake is capable of diving to 100 m (328 feet) and remaining submerged for 2 hours. The sea snake is an air breather and must surface periodically. The sea snake can be distinguished from a sea eel (Fig. 74-37) by the presence of scales and the absence of gills and fins.

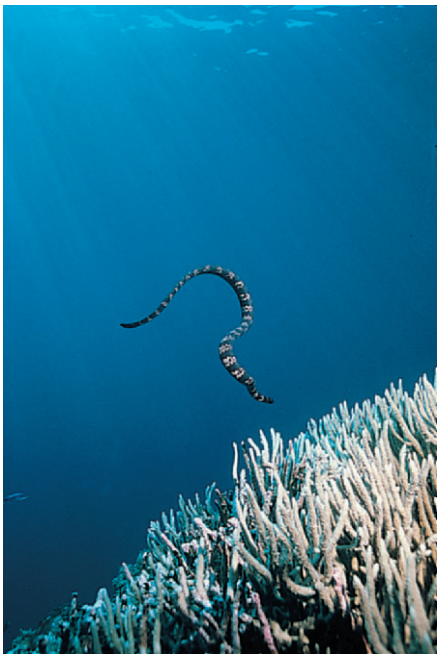


Figure 74-36. Sea snake in the Coral Sea. (Photo by Carl Roessler.)

Sea snakes use an air retention mechanism in the lung to control buoyancy. Their food, small fish swallowed whole, is captured underwater, usually around bottom rocks and coral. In general, sea snakes are docile creatures and flee when approached. However, when cornered or handled, they may become aggressive and strike out. During the reproductive season, some males adopt more irritable attitudes. The banded sea snake (sea krait) *Laticauda semifasciata* is served as a food (raw, smoked, or cooked) in certain Asian countries, notably Japan and the Philippines.

Venom and Venom Apparatus

The well-developed venom apparatus consists of two to four hollow maxillary fangs and a pair of associated venom glands. Fortunately, because the fangs are short and easily dislodged from their sockets, most bites (approximately 80%) do not result in significant systemic envenomation. Most fangs, except for those of *Astrotia stokesii* and *Aipysurus laevis,* are not long enough to penetrate a wetsuit. The venom yield of sea snakes varies with species and is largely related to the size of the venom glands. An average-sized snake can produce 10 to 15 mg of

Figure 74-37. Harmless snake eel mimics venous sea snake, Sulawesi Island, Indonesia. (© 2006 Norbert Wu / www.norbertwu.com.)

venom, which is approximately 10 times the lethal dose in humans.

The protein venom is highly toxic and includes stable peripheral neurotoxins more potent than those of terrestrial snakes. Neuromuscular transmission is blocked predominantly at the postsynaptic membrane and caused by attachment of toxin to the alpha subunit of the acetylcholine receptor. Presynaptic toxin in sea snake venom has been less well studied but appears to be related to inhibition of transmitter release by blocking resynthesis of acetylcholine from choline. It seems probable that the action of *Laticauda semifasciata* venom on excitable membranes is to alter ionic permeability, particularly that of sodium and chloride, without effect on Na^+,K^+-dependent adenosine triphosphatase activity. Calcium transport abnormalities are currently under investigation. Among other fractions of the venom are phospholipases, nerve growth factors, capillary permeability factor, anticomplement-active factor, enzymes (including acetylcholinesterase, hyaluronidase, leucine aminopeptidase, 5′-nucleotidase, phosphomonoesterase, phosphodiesterase), and hemolytic and myotoxic compounds, which result in skeletal muscle necrosis, intravascular hemolysis, and renal tubular damage. Myonecrosis is related to phospholipase A, which may inhibit calcium uptake into the sarcoplasmic reticulum. Neurotoxins are thought to exert their toxicity by binding in a nondepolarizing fashion to the nicotinic acetylcholine receptor and blocking neuromuscular transmission.[65]

The venoms of sea snakes are similar, as is reflected in positive reactions during immunodiffusion, immunoelectrophoresis, and cross-neutralization by antivenom against heterologous venoms, and amino acid composition and sequences of neurotoxins. This is a reflection of phylogenetic relationships and is a logistic aid in the preparation of effective antivenom.

Although large venom yields have been obtained from *Astrotia stokesii, Enhydrina schistosa* is considered the most dangerous sea snake (Fig. 74-38). *E. schistosa* is the most widely distributed sea snake in the Arabian sea. *Aipysurus duboisii* and *Acalyptophis peronii* from the Coral Sea have recently be shown to carry venoms of high human lethality potential. In an evaluation of poisonous land and sea snakes representative of those encountered in Saudi Arabia, sea snakes were noted to have an average lethal dose in dogs of 0.05 mg/kg, in comparison with the average lethal dose of vipers (1.13 mg/kg) and elapids

Figure 74-38. Beaked sea snake *(Enhydrina schistosa)*. A common sea snake of Southeast Asia, the average length is about 1 m (3 feet). This creature inflicts a high proportion of the sea snake bites recorded in Asian coastal waters. (Photo by Sherman Minton, MD.)

(0.69 mg/kg).[79] Deaths were attributed to respiratory paralysis and failure.

Clinical Aspects

Bites are usually the result of accidental handling of snakes snared in the nets of fishermen or of accidentally stepping on a snake while wading. Most sea snake poisonings occur in remote fishing villages and in boats engaged in fishing. Nearly all bites involve the extremities.

The diagnosis of sea snake bite is based on the following:

1. Location. A person usually must have been in the water or handling a fishing net containing a sea snake to have been bitten. Some snakes may foray briefly onto land, particularly in areas of heavy mangrove growth, but it is quite unusual for a bite to occur out of the water. Because snakes may inhabit sheltered coastal waters and frequently congregate near river mouths, a bite can occur in an estuarine setting, up to 5 km (3 miles) inland.
2. Absence of pain. Initially, a sea snake bite does not cause great pain and may resemble no more than a pinprick.
3. Fang marks. These are multiple pinhead-sized, hypodermic-like puncture wounds, usually 1 to 4, but potentially up to 20. If the skin is not broken, envenomation cannot occur. In some cases, particularly with a superficial injury through the arm or leg of a neoprene wetsuit, the fang marks may be difficult to visualize because of the lack of a localized reaction.
4. Identification of the snake. Snakes should be captured or killed carefully with a nonmacerating blow behind the head and retained for identification by an expert.
5. Development of characteristic symptoms. These include painful muscle movement, lower extremity paralysis, arthralgias, trismus, blurred vision, dysphagia, drowsiness, vomiting, and ptosis. Neurotoxic symptoms are rapid in onset and usually appear within 2 to 3 hours. If symptoms do not develop within 6 to 8 hours, there has almost certainly not been a clinically significant envenomation.

Envenomation by sea snakes characteristically shows an evolution of symptoms over a period of hours, with the latent period being a function of venom volume and victim sensitivity. The onset of symptoms can be as rapid as 5 minutes or as long as 8 hours. There is no appreciable local reaction to a sea snake bite other than the initial pricking sensation. The first complaint may be euphoria, malaise, or anxiety. Over 30 to 60 minutes, classic muscle aching and stiffness (particularly of the bitten extremity and neck muscles) develop, along with a "thick tongue" and sialorrhea, indicative of speech and swallowing

dysfunction. Within 3 to 6 hours, moderate to severe pain is noted with passive movements of the neck, trunk, and limbs. Ascending flaccid or spastic paralysis follows shortly, beginning in the lower extremities, and deep tendon reflexes diminish and may disappear after an initial period of spastic hyperreactivity. Nausea, vomiting, myoclonus, muscle spasm, ophthalmoplegia, ptosis, dilated and poorly reactive pupils, facial paralysis, trismus, and pulmonary aspiration of gastric contents are frequent complications. Occasionally, bilateral painless swelling of the parotid glands develops.

Severe envenomations are marked by progressively intense symptoms within the first 2 hours of symptoms. Victims become cool and cyanotic, begin to lose vision, and may lapse into coma. Failing vision is reported to be a preterminal symptom. If peripheral paralysis predominates, the victim may remain conscious if hypoxia is avoided. Leukocytosis may exceed 20,000 white blood cells per milliliter; elevated plasma creatine kinase is variable. Elevated glutamic oxaloacetic transaminase reflects hepatic injury. Pathognomonic myoglobinuria becomes evident about 3 to 6 hours after the bite and may be accompanied by albuminuria and hemoglobinuria. Cerebrospinal fluid is normal. Respiratory distress and bulbar paralysis, pulmonary aspiration–related hypoxia, electrolyte disturbances (predominantly hyperkalemia), and acute renal failure (attributed in part to myonecrosis and pigment load) all contribute to the ultimate demise, which can occur hours to days after the untreated bite. Preterminal hypertension may occur. The mortality is 25% in victims who do not receive antivenom and 3% overall.

It is interesting to note the effects of sea snake *(Aipysurus laevis)* venom on prey fish.[85] The prey are subdued in six stages, which correlate roughly to certain aspects of a human envenomation: stage 1, increased ventilatory rate; stage 2, loss of mouth control, fin control, coordination, and buoyancy; stage 3, depressed ventilation, weakness, ineffective swimming; stage 4, apnea; stage 5, near paralysis, body color darkening; and stage 6, death.

Treatment

If possible, the offending snake should be captured for identification, taking care not to increase the number of victims. The therapy for bites by snakes of the family Hydrophiidae is similar to that for terrestrial snakes of the family Elapidae. The affected limb should be immobilized and maintained in a dependent position while the victim is kept as quiet as possible. The pressure-immobilization technique (see Figure 48-27) for venom sequestration should be applied. If the bite is on a digit where a compression bandage cannot be applied, a loose constriction bandage that constricts only the superficial venous and lymphatic flow may be applied proximal to the wound. This should be released for 90 seconds every 10 minutes and should be completely removed after 4 to 6 hours. If the bite is older than 30 minutes, neither technique may be very effective.

There is little clinical enthusiasm for the perpetuation of incision and suction therapy, which has nearly been universally relegated to therapeutic history.

The victim must be kept warm and as still as possible. As with terrestrial snakebite, cryotherapy (immersion into ice water) is inefficacious and potentially harmful.

With any evidence of envenomation, sea snake antivenom (an equine pepsin-digested immunoglobulin from CSL Limited) prepared against the venom of *Enhydrina schistosa* and the Australian tiger snake *Notechis scutatus* should be administered intravenously after appropriate skin testing for equine serum hypersensitivity. Tiger snake *(Notechis scutatus)* antivenom was formerly recommended for use if sea snake antivenom is unavailable, but this is no longer recommended, because tiger snake antivenom does not appear to be efficacious against sea snake bites in humans.[83] However, it is still commented in product literature that one vial of CSL sea snake antivenom is equivalent to 2 to 4 vials of CSL tiger snake antivenom. Sea snake antivenom is specific and absolutely indicated in cases of envenomation. Supportive measures, although critical in management, are no substitute. The administration of antivenom should begin as soon as possible and is most effective if initiated within 8 hours of the bite. Each vial of sea snake antivenom contains 1000 units of antivenom in 15 to 35 mL of liquid. The minimum effective adult dosage is one vial (1000 units), which neutralizes 10 mg of *E. schistosa* venom. The victim may require 3000 to 10,000 units (3 to 10 vials), depending on the severity of the envenomation. The proper administration of antivenom is clearly described on the antivenom package insert. Antivenom should be protected from light and stored refrigerated at 2° to 8° C (35.6° to 46.4° F). It must not be frozen.

Commercial Thai cobra *(Naja kaouthia)* antivenom was found to be effective in neutralizing sea snake *(Lapemis hardwickii)* venom in mice. The application of this finding to humans is as yet undetermined.[52]

Sea snake envenomation may induce severe physiologic derangements that require intensive medical management. Urine output and measured renal function should be closely monitored because hemolysis and rhabdomyolysis release hemoglobin and myoglobin pigments into the circulation, which precipitates acute renal failure. If hemoglobinuria or myoglobinuria is detected, the urine should be alkalinized with sodium bicarbonate and diuresis promoted with a loop diuretic (furosemide or bumetanide) or mannitol, to avoid progressive nephropathy. Acute renal failure may necessitate a period of peritoneal dialysis or hemodialysis. Hemodialysis offers an alternative therapy that may be successful if antivenom is not available.

Respiratory failure should be anticipated as paralysis overwhelms the victim. Endotracheal intubation and mechanical ventilation may be required until antivenom adequately neutralizes the venom effects. Serum electrolytes should be measured regularly to guide the administration of fluids and electrolyte supplements. Hyperkalemia related to rhabdomyolysis and renal dysfunction must be promptly recognized and treated.

As previously mentioned, symptoms usually occur within 2 to 3 hours after envenomation. If there is no early evidence of envenomation, the victim should be observed for 8 hours before discharge from the hospital.

SUMMARY

A summary algorithmic approach to marine envenomation can be followed when the causative agent cannot be positively identified (see Figure 73-77).[5] Once the physician has made a commitment to a course of treatment based on a presumption of what creature has caused the injury, the subtleties of therapy can be utilized.

The references for this chapter can be found on the accompanying DVD-ROM.

75 Aquatic Skin Disorders

Vicki Mazzorana and Edgar Maeyens, Jr.

Dermatitis acquired from the aquatic environment can be related to exposure to diving equipment, chemicals, or animal and plant irritants. Hypersensitivity reactions to water have been documented. Bacteria, algae, and parasites can be the etiologic agents of a variety of aquatic infections. Wounds resulting from stings, bites, punctures, and suction may result from inadvertent contact with marine life (see Chapters 72, 73, and 74).

CONTACT DERMATOSES

Allergic Dermatitis

Diving Wetsuits

Neoprene (polychloroprene), the synthetic rubber used in wetsuits, contains many chemicals that are allergenic. An allergic reaction to wetsuits may reflect sensitivity to either the rubber or any of the other chemicals, such as dodecyl mercaptan and the accelerator ethyl butyl thiourea within the neoprene suit.[1,85,88] N/N-diethylurea is a chemical compound used within the wetsuit to bond the nylon lining to the rubber. It can cause contact dermatitis of the neck, trunk, and extremities in sensitive individuals.[1,29,85,148] Contact allergy may be enhanced by heat, sweating, and maceration. The cutaneous reaction varies from generalized redness to severe, pruritic papulovesicles (Figs. 75-1 and 75-2). Wearing a tight diving suit may produce irritant skin lesions at the pressure points. Systemic antihistamines (Table 75-1) are recommended to provide symptomatic relief from the pruritus. Systemic corticosteroids (Table 75-2) may be necessary in the treatment of severe reactions.

Diving Masks, Goggles, and Mouthpieces

The facial condition termed *mask burn* has been described as a reddish imprint of the mask on the face and was initially thought to be a requisite annoyance of snorkeling and scuba diving.[1,6,85] More severe reactions, consisting of painful, disabling eruptions characterized by vesiculation, weeping, and crusting have been described. Mouthpieces attached to regulators and snorkels may cause minor oral irritation described by sufferers as a mild burning sensation. Persons with sensitivity to the components of the mouthpiece may suffer severe intraoral ulceration and inflammation accompanied by vesiculation of the oral mucosa, gingiva, and tongue.[6] Neoprene swim goggles can cause raccoon-like periorbital contact dermatitis in sensitive individuals.[8,102]

Rubber mouthpieces and masks contain antioxidants similar to those that cause contact dermatitis in surgeons who are allergic to rubber gloves. Mercaptobenzothiazol, tetraethylthiuram, and isopropyl paraphenylene diamine have been implicated in such reactions.[88] N-isopropyl-N-phenyl-paraphenylenediamine, an antioxidant used in rubber scuba masks, can cause facial dermatitis.[181,275] Use of diving equipment can result in both irritant and allergic reactions.[84,87]

Treatment and Prevention. Acute facial dermatitis may be treated with the application of cool compresses containing Burow's solution and the administration of oral systemic corticosteroids, such as prednisone, beginning with a supraphysiologic dose (see Table 75-2) For serious intraoral reactions, rinsing twice daily with a mouthwash consisting of equal parts antihistamine, such as diphenhydramine elixir, and milk of magnesia may be efficacious.[88] Individual mucosal ulcerations may be coated twice daily and again at bedtime with triamcinolone acetonide (0.1%) dental paste (Kenalog in Orabase) for 5 to 7 days. The problem may be avoided at the outset by using clear silicone rubber hypoallergenic masks and mouthpieces.

Rubber Swim Gear

Nose clips, ear plugs, and swim fins produce allergic contact dermatitis in some individuals.[88] Equipment made of nonallergenic silicone is available. Mercaptobenzothiazole, which is a constituent of some bathing caps, also can cause contact dermatitis.[88] Silicone swim caps are available for persons who are allergic to rubber bathing caps.

Irritant Dermatitis

Chlorine

Chlorinated pool or spa water rarely causes irritant dermatitis or an allergic reaction, but it may produce contact urticaria. Nitrogen trichloride, which is produced when chlorine is added to water containing ammonia, has a pungent odor and may cause eye and nose irritation.[86,207]

Bromine

Bromine-based disinfectants are used in swimming pools and spas. They are more effective than chlorine-based disinfectants against pseudomonads at high temperatures.[91] Bromine dermatitis is described as a pruritic, generalized, eczematous irritant contact dermatitis arising within 24 hours of exposure to a spa or pool recently disinfected with 1-bromo-3-chloro-5, 5-dimethylhydantoin (Halobrome)[232] (Fig. 75-3). The dermatitis resolves spontaneously after spa use is discontinued. Treatment is symptomatic.

Figure 75-1. Severe allergic contact dermatitis secondary to a neoprene hood. (Courtesy of Edgar Maeyens, Jr., MD.)

Figure 75-3. Bromine: An extensive generalized irritant dermatitis secondary to excessive bromine used in a home spa. (Courtesy of Edgar Maeyens, Jr., MD.)

Figure 75-2. Contact dermatitis secondary to neoprene knee brace. (Courtesy of Edgar Maeyens, Jr., MD.)

TABLE 75-1. Topical Antipruritics and Oral Antihistamines

PRODUCT (MANUFACTURER)	DESCRIPTION/DOSAGE
Topical	
Sarna Ultra (Stiefel)	1.0% pramoxine HCl, 0.5% menthol, 30% petroleum in a lotion base
Soothing Relief Cream (Neutrogena)	3% lidocaine (Xylocaine) in emollient cream
Pramosone Cream, Ointment, Lotion (Ferndale)	Hydrocortisone acetate 1.0% and 2.5% with pramoxine HCl 1.0%
Oral	
Allegra (Aventis)	60-mg tablets, capsules 1 PO tid *or* 180 mg capsule daily
Bendadryl (Parke-Davis)	25 mg capsule, 1 PO q 6 h prn
Zyrtec (Pfizer)	5-mg or 10-mg tablet, 1 once daily 5 mg per teaspoon syrup once or twice daily

Sulfur

A dermatitis consisting of erythematous pea-sized papules has been attributed to bathing in green sulfur springs[266] (Fig. 75-4). The lesions develop within 24 hours after bathing and are distributed over the trunk and limbs. Allergic contact dermatitis has not been documented in persons who bathe in hot springs. The water's high soluble chloride and sulfur content and its extreme acidity predispose bathers to irritant reactions. It has not been shown that microorganisms play a role in any of these dermatoses.

Algaecides

Copper solutions or quaternary ammonium compounds may be used to combat algae growth in pools and spas. Copper algaecide solutions used to treat pool and spa water can cause greenish discoloration of the scalp hair, most obvious in blonds[164] (Fig. 75-5). Wetting the hair with fresh water before immersion

TABLE 75-2. Systemic Steroids

STEROID	DOSING
Oral	
Prednisone	Dosing Schedule A: 40 mg PO every morning for 7 days Dosing Schedule B: 2-wk tapering course starting at 35 mg PO q am × 2, then decrease by 5 mg q2d
Intramuscular	
Kenalog	40 to 60 mg deep IM into a large muscle mass (gluteus maximus)

Figure 75-4. Sulfur: Widely scattered erythematous papules after "hot tubbing" in a sulfur hot spring. (Courtesy of Edgar Maeyens, Jr., MD.)

Figure 75-5. Green hair discoloration secondary to copper algaecide. (Courtesy of Edgar Maeyens, Jr., MD.)

in pool or spa water decreases the incidence and severity of hair discoloration. Shampooing decreases the green tint, and immersing the hair in 3% peroxide solution removes the green discoloration in a couple of hours.

Animal and Plant Irritants

Sea Cucumbers

Sea cucumbers (see Chapter 73) are invertebrates that extrude sticky, white tentacular organs used to entangle prey. Humans who come into contact with these moplike strands suffer dermatitis. Cutaneous erythema, pain, and pruritus may occur. The skin manifestations are self-limited. Prompt washing with soap and water appears to remove any toxins.[182]

Figure 75-6. Soapfish *(Rypticus saponaceus)*. Skin contact with soapy mucus causes dermatitis. (Courtesy of Carl Roessler.)

Ichthyocrinotoxic Fish

A number of fish species possess glands that secrete skin toxins (crinotoxins) but do not have venomous defense organs, such as spines. These include soapfish, trunkfish, gobies, sole, toadfish, clingfish, and pufferfish.[114] The toxins can be extremely powerful, but resulting dermatoses are poorly documented. The soapfish (family Grammistidae) *Rypticus saponaceus* (Fig. 75-6) receives its name from the soapy mucus it releases when handled or disturbed. Fishermen in the Caribbean know that the presence of a soapfish in a restricted volume of seawater with other fish results in death of the other fish.[120] Human contact results in eczematous dermatitis. The skin irritant is called grammistin; similar substances have been isolated from boxfish and sea bass species.[37,120,219]

Treatment and Prevention. Cool compresses of aluminum sulfate and calcium acetate (Domeboro), Aveeno (one package per bathtub), or Burow's solution applied to affected areas may alleviate the burning and itching caused by soapfish dermatitis. In severe cases, topical corticosteroids (Table 75-3) are beneficial. Systemic corticosteroids are rarely required. Wearing protective clothing and gloves when handling fish prevents dermatitis from developing. Washing protective wear with soap and water after use is required to remove toxins.

Sea Moss Dermatitis

Sea moss dermatitis, also known as "dogger bank itch," is an eczematous dermatitis caused by a plant *(Fragilaria striatula)* or by bryozoarian animals referred to as sea chervils (*Alcyonidium hirsutum* and *Alcyonidium gelatinosum*).[182] The irritant contact dermatitis is most common among fishermen of the North Sea. These fishermen make contact with sea mosses or sea mats that are drawn up within fishnets. The dermatitis first appears on the palms and forearms (Fig. 75-7) and often disappears spontaneously with the discontinuance of fishing.[201] Recurrent exposure causes subsequent attacks to be more severe.

The cutaneous reaction initially consists of erythema but may progress to vesiculated, edematous eruptions of the hands, arms, legs, and face. It is controversial as to whether or not this is an allergic reaction or an irritant dermatitis.[201] Positive reactions to patch tests performed with an extract from the causative source and lack of reaction in controls support an allergic etiology.

Treatment. Treatment is symptomatic. Application of dilute (26%) isopropyl alcohol in calamine lotion as needed may provide relief from the itch. Severe reactions may require the use of oral antihistamines (see Table 75-1) and oral corticos-

TABLE 75-3. Potency Ranking of Topical Steroids

POTENCY	BRAND	GENERIC	SIZES
Super high potency	Temovate Cream, Ointment 0.05%	Clobetasol propionate	15, 30, 45, 60 gm
	Psorcon Ointment	Diflorasone diacetate	15, 30, 60 gm
Medium potency	Westcort Cream 0.2%	Hydrocortisone valerate	15, 45, 60 gm
	Locoid Cream 0.1%	Hydrocortisone butyrate	15, 45 gm
Low potency	Aclovate Cream, Ointment 0.05%	Aclometasone diproprionate	15, 45, 60 gm
	Desowen Cream, Lotion 0.05%	Desonide	15, 60, 118 mL

These topical steroids must be applied twice daily. However, the actual application rate can vary upward a maximum of 3 to 4 times per day according to the prescriber's discretion.

Figure 75-7. Sea moss dermatitis. Dermatitis of palms and forearms from a moving sea moss entangled in nets. (Courtesy of Edgar Maeyens, Jr., MD.)

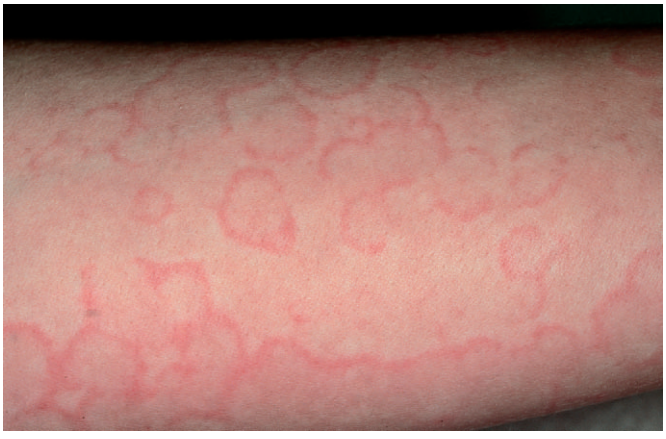


Figure 75-8. Red tide dermatitis. Petaloid pattern of urticaria following swimming in red tide contaminated water. (Courtesy of Edgar Maeyens, Jr., MD.)

teroids (see Table 75-2) or potent topical corticosteroids (see Table 75-3) Wearing proper protective clothing may prevent this self-limited disorder.

Red Tide Dermatitis

A "red tide" occurs when large numbers of unicellular microscopic free-swimming dinoflagellate organisms are present in the water. A variety of plant life proliferations, such as *Ptychodiscus brevis*, have been implicated as causative agents of red tides. Unicellular algae reproduce rapidly when conditions are favorable. They secrete toxins that can cause dermatitis, conjunctivitis, and respiratory symptoms in humans (see Chapter 57). Swimming during a red tide may result in an acute urticarial eruption (Fig. 75-8). Massive blooms of these types of phytoplankton are toxic to fish. Avoidance of affected areas, especially during windy conditions, is the best prevention, because wind aerosolizes the algae blooms.

Treatment. There is no specific treatment. Symptomatic treatment is all that is needed for this dermatitis.

Ciguatera Dermatitis

Blooms of toxic dinoflagellates, such as *Gambierdiscus toxicus*, synthesize toxins that become incorporated into the food chain.[296] Ciguatera toxins can be extremely harmful to humans (see Chapter 66). Dermatologic manifestations include intense generalized pruritus associated with a diffuse maculopapular eruption, leading to blisters and desquamation (Fig. 75-9). Other manifestations have been reported, such as hair and nail loss, intense diaphoresis leading to dehydration, cyanosis, and urticarial reactions. Symptom severity depends on factors such as geographic location.[296]

Treatment. Treatment is symptomatic, with the use of topical and systemic therapy depending on the severity of the skin manifestations[296] (see Tables 75-1, 75-2, and 75-3).

Algal Dermatitis

Seaweed Dermatitis. Seaweed dermatitis is almost always secondary to irritation resulting from contact with algae, of which more than 25,000 species are known. Algae are loosely defined as chlorophyll-bearing or colorless organisms that are thallous (without true roots, stems, or leaves). They vary in size, shape, and color. Some are equipped with flagella and propel themselves through the water.[271] Algae range from microscopic diatoms (1 µm in length) to kelp plants (100 m [328 ft]in length). Algae are found in all marine environments and are extremely adaptive to temperature and depth variation. For instance, one form of blue-green algae thrives at hot spring temperatures of 71° C (160° F); others dwell in frigid Arctic water. Although sunlight can penetrate only to a depth of 900 ft (274 m) in the ocean, some blue-green algae exist at depths up to 12,000 ft (3658 m).

Figure 75-9. Ciguatera dermatitis. Thirty-year-old male's posterior hemithorax showing papules and rare blister as a cutaneous manifestation of his ciguatera intoxication. (Courtesy of Edgar Maeyens, Jr., MD.)

Figure 75-10. *Microcoleus lyngbyaceus.* Rare and extreme example of superficial necrosis and inflammation secondary to dermonecrotic toxins. (Courtesy of Edgar Maeyens, Jr., MD.)

Microcoleus lyngbyaceus. *Microcoleus lyngbyaceus,* formerly known as *Lyngbya majuscula Gomont,* is a blue-green saltwater alga that has strains of varying toxicity, often within geographic proximity. The "stinging seaweed" is dark green or olive, drab, and finely filamentous, occurring in abundance at depths of up to 100 ft (30 m).[245,246,251] It grows in hairlike masses or mats throughout the Pacific and Indian Oceans and Caribbean Sea. There have been large epidemics reported in Hawaii and Okinawa, Japan.[135] Outbreaks of *Microcoleus* organism–induced dermatitis occur between March and September in Florida and between June and September in Hawaii. In Hawaii, the largest reported series involved windward beaches.[106] The assumption is that strong currents and winds dislodge the alga from its normal habitat, fragment it, and blow the fragments into the surf line.[135] The alga produces a dermatonecrotic and potent inflammatory agent called debromoaplysiatoxin, as well as an indole alkaloid dermatitis–producing toxin called lyngbyatoxin A.[48,198,251] A radioimmunossay to identify the presence of lyngbyatoxin A is in development.[135]

Vibrio alginolyticus and *Vibrio parahaemolyticus* have been cultured from marine specimens of *M. lyngbyaceus* collected from the beaches of Hawaii. Typically, the affected individual swam in turbid water infested with plant life and did not remove his or her bathing suit after leaving the water. Within minutes to hours, a pruritic, burning, moist, and erythematous dermatitis develops in a bathing suit distribution, followed by bullous escharotic desquamation in the genital, perineal, and perianal regions[246] (Fig. 75-10). Lymphadenopathy, pustular folliculitis (Fig. 75-11), and local infections occur in some individuals. It may be difficult to differentiate this reaction from sea bather's eruption (which is attributed to thimble jellyfish or larval anemone organisms) (see Chapter 73). There also are occasional outbreaks of similar skin reactions after exposure to freshwater blue-green algae.[60,123] Oral and ocular mucous membrane lesions, facial dermatitis, conjunctivitis, and perhaps interstitial pulmonary edema have been attributed to *M. lyngbyaceus*.[135,245]

Treatment. Treatment of dermatitis caused by *M. lyngbyaceus* consists of copious irrigation with soapy water, followed by isopropyl alcohol rinses and application of topical corticosteroids

Figure 75-11. *Microcoleus lyngbyaceus.* Folliculitis in the bathing trunk area. (Courtesy of Edgar Maeyens, Jr., MD.)

(see Table 75-3). Severe cases may require oral administration of a systemic corticosteroid, such as prednisone, tapered over 2 weeks (see Table 75-2).

Prevention. Prevention is accomplished by showering with soapy water and removing all swimwear immediately upon leaving the water. Avoid swimming in areas laden with seaweed.

Sargassum natans. *Sargassum* species algae collect as seaweed masses in great numbers off the southeastern coast of Florida. Breezes blow the algae toward the coast in the winter. The algae are generally considered to be harmless to man. After *Sargassum* species seaweed was placed in the swimwear of two children at play, they both developed an erythematous skin eruption with urticarial lesions. The eruption disappeared within 4 hours.[43]

Blue-Green Algae of Freshwater

Definition. In freshwater environments, warm weather can cause rapid growth of blue-green algae (group Cyanophyta) in

Figure 75-12. Blue-green algae. Facial and lip swelling after accidental ingestion of contaminated water. (Courtesy of Edgar Maeyens, Jr., MD.)

a "bloom."[255] During conditions of a bloom, toxins are concentrated and become a significant hazard.[73,78] Species such as *Microcystis aeruginosa, Aphanizomenon flos-aquae, Nodularia spumigena, Nostoc* species, *Oscillatoria agardhii, Anabaena spiroides,* and *Anabaena flos-aquae* have been implicated in the acute poisoning and death of wild and domestic animals.[199,231] In most species of toxic cyanobacteria, the toxins are cyclic heptapeptides called microcystins or cyanoginosins.[248,261] More than 40 microcystins have been isolated from blue-green algae.[248] The toxins are of multiple configurations and include alkaloids, polypeptides, and lipopolysaccharides (endotoxins).[255] The rapid-acting toxins, called "death factors," resemble saxitoxin and tetrodotoxin.

Animals exposed to these toxins may collapse quickly and appear to suffer neuromuscular paralysis, with features of staggering muscle fasciculations, gasping, and convulsions.[117] A different dominant symptom complex includes gastrointestinal hemorrhage and hepatic failure. Although ingestion of contaminated water may cause considerable animal mortality, human exposures to date have resulted primarily in local effects, including allergic reactions, conjunctivitis, ear infection, facial swelling (Fig. 75-12), lip swelling, papulovesicular dermatitis, and gastroenteritis.[33,168]

Treatment. Treatment is supportive in both animals and humans. Cyclosporin A has been shown to inhibit the fatal effects of microcystins administered to mice, but has not demonstrated the same effect in humans.

Prototheca

Definition. The genus *Prototheca* consists of nonpigmented algae from the family Chlorellaceae. Human and animal infections have been caused by achlorophyllic mutants of the green alga *Chlorella pyrenoidosa*. Two species of *Prototheca* are recognized: *Prototheca wickerhamii* and *Prototheca zopfii*. Of these algae, *P. wickerhamii* and *P. zopfii* are the most commonly isolated pathogens in human protothecosis.[36,64,156,286]

Microbiology. These organisms are ubiquitous and have been isolated in fresh and marine water, streams, lakes, sewage treatment systems, tree slime, and animal feces.* *Protothecae* are unicellular, aerobic, and achlorophyllic spherical organisms with hyaline sporangia that reproduce asexually. They are unable to produce energy from photosynthesis and therefore

*References 36, 154, 189, 190, 195, 197, 215, 268, 269, 273, 280, 281, 286.

Figure 75-13. Protothecosis of anterior leg. (Courtesy of Edgar Maeyens, Jr., MD.)

exist as saprophytes.[36] These organisms are distinct from fungi and bacteria in size, morphology, and method of reproduction. They resemble *Chlorella* organisms and probably represent a mutation of this genus.[36,268] Infection with these algae can present after recreational exposure in swampy areas or after aquarium management.[36] Preexisting skin wounds exposed to contaminated water or soil are potential sites of entry of these organisms. Percutaneous inoculation with contaminated water after trauma is the most common mechanism of infection.[268,286]

Signs and Symptoms. Clinical features of human infection include:

1. **Superficial cutaneous lesions:** They present as papulonodules or verrucous plaques with or without ulcerations[57] (Fig. 75-13). Bullous lesions may occur with subsequent rupture, drainage, and crusting.[111] Rarely, eczematous and cellulitis-like lesions occur.
2. **Olecranon bursitis:** The elbow is swollen and erythematous, and on occasion drains spontaneously. This is not accompanied by fever or chills. The presentation is similar to other causes of bursal inflammation. A history of preceding trauma should suggest protothecosis.[57,111]
3. **Systemic infection:** Immunosuppressed patients, such as those on chemotherapy or afflicted with human immunodeficiency virus (HIV), are more predisposed to disseminated infection than are persons who are immunocompetent. At least 50% of the reported victims of cutaneous protothecosis are immunosuppressed, owing to either local or systemic factors.[49,217]
4. **Mucosal:** Lacoviello and colleagues reported a case of protothecosis of the esophagus, complicating prolonged endotracheal intubation.[162]

In cases associated with a traumatic episode, the initial lesion is a nodule or tender red papule, which enlarges, becomes pustular, and ulcerates. A purulent, malodorous, and blood-tinged discharge may be present. Satellite lesions surrounding the primary lesion usually develop and frequently become confluent. The lesions may become verrucous and resemble chromomycosis. Regional lymph nodes may develop metastatic granulomas. The lesions extend centrifugally and occasionally disseminate. In the olecranon bursitis form, the infection develops several weeks after an elbow injury and is localized to the

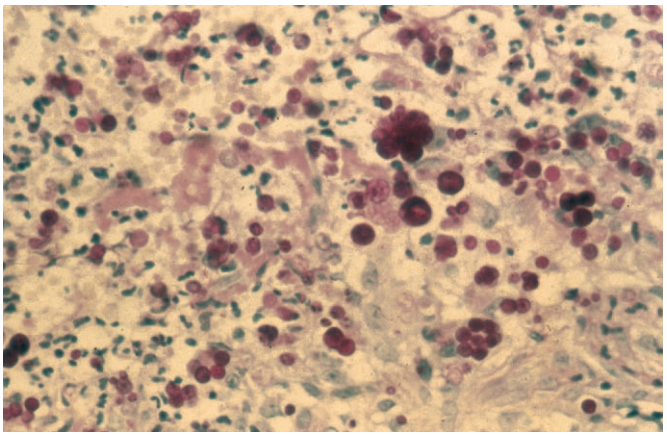


Figure 75-14. Protothecosis histology. (Courtesy of Edgar Maeyens, Jr., MD.)

bursa.[200] Overlying sinus tracts may develop. The diagnosis is confirmed with histology and tissue culture.

Histology. Histologically, the dermis and subcutaneous tissue show granulomatous inflammation containing organisms within the cytoplasm of multinucleated giant cells or lying freely in the dermis. The characteristic endospores of *Prototheca* are rounded, with a central spore surrounded by a corona of molded endospores forming a morula (from the Latin word for mulberry) (Fig. 75-14). The organisms are spherical, basophilic, and gram positive; they vary in diameter from 3 to 15 µm *(P. wickerhamii)* or 7 to 30 µm *(P. zopfii)* and stain well with Grocott-Gomori methenamine silver nitrate, colloidal iron for acid mucopolysaccharides, and periodic acid-Schiff.[111] Larger organisms have thick walls and characteristic internal septation.

Microbiology. *Prototheca* species grow on Sabouraud agar, blood agar, or brain-heart infusion agar at an optimum temperature of 30°C to 32°C (86°F to 90°F). The colonies are cream colored, yeastlike, and microaerophilic and reproduce in 1 to 2 days.[24,149] Antisera for identification of species of *Prototheca* can be used on fresh or formalin-fixed tissues.[265]

Treatment. Protothecosis is a chronic and progressive disease.[143,200] Effective therapy has not been established. Localized lesions can be excised, which may be curative.[190,212] Topical medications are unsatisfactory. Algaecidal agents, including ketoconazole, itraconazole, fluconazole, miconazole, fluorocytosine, and tetracycline, may inhibit or kill the organisms in optimal conditions. There is early indication that parenteral therapy with amphotericin B combined with oral tetracycline may be effective.[154,197,200,269,272,273,286]

HYPERSENSITIVITY REACTIONS

Aquagenic Urticaria

Definition

Aquagenic urticaria is a rare condition that occurs when water of any temperature contacts skin, resulting in an urticarial eruption at the site of exposure (Fig. 75-15).[209,211,239] The appearance

Figure 75-15. Aquagenic urticaria. Pruritic, punctate, and perifollicular wheals characteristic of the rash of aquagenic urticaria. (Courtesy of Edgar Maeyens, Jr., MD.)

of urticaria is usually preceded by itching. Morphologically, it is characterized by pruritic, punctate, and perifollicular wheals 1 to 2 mm in diameter that develop as early as 10 minutes after contact with water and persist for up to 60 minutes. The eruption is usually confined to the neck, upper trunk, and arms, while the face, hands, legs, and feet are spared.[209] An episode is typically followed by a refractory period lasting several hours. No systemic complications have been reported. This entity most often occurs in postpubescent individuals, and it is reported more frequently in women than in men.[176] There may be a family history of atopy.[31] The coexistence of other forms of physical urticaria has been demonstrated in some patients.[65,187]

Pathogenesis

The pathogenesis of this phenomenon is not completely understood. Histamine involvement is supported by the finding of elevated local tissue histamine levels and by suppression of symptoms via various antihistamines.[63,65,242,288] Dermographism typically is not present, and serum IgE levels usually are normal.[242] In some cases, prophylaxis with antihistamines fails to prevent the urticarial response to contact with water, suggesting a mechanism unrelated to the release of histamine. Some have suggested that water may react with a component of sebum on the stratum corneum, forming a toxic substance that diffuses into the skin and may either directly cause urticaria or stimulate mast cells to release histamine, resulting in urticaria.[63,239] Another theory is that there is a change in the osmotic pressure in the skin around hair follicles, allowing for passive permeation of water through the skin.[288] In some victims there is a significant increase in acetylcholinesterase activities, suggesting that the pathogenesis of aquagenic urticaria may be related to the release of acetylcholine in the nerve fibers of sebaceous glands.[242] The ability of anticholinergics to suppress the

hypersensitivity reaction in some individuals suggests that aquagenic urticaria is acetylcholine mediated.[192]

Differential Diagnosis

Aquagenic urticaria must be differentiated from other forms of physical urticaria that are induced by pressure, exercise, cold, or stress.[187] Clinically, the morphology and distribution of the eruption are similar to those of cholinergic urticaria. Aquagenic urticaria can be differentiated from cholinergic urticaria by pretreating the skin with topical atropine, which does not prevent wheal formation in aquagenic urticaria but does so in cholinergic urticaria.[242] Aquagenic urticaria also must be differentiated from aquagenic pruritus, in which exposure of skin to water elicits intense pruritus without cutaneous lesions.[109] Before the diagnosis of aquagenic urticaria can be made, other physical urticarias must be ruled out and the patient must respond to a water provocation test.[187]

Treatment

Aquagenic urticarial reactions can be inhibited by applying petroleum ointment to the skin before water exposure.[242] There may be a role for prophylaxis with antihistamines (see Table 75-1), given 1 hour prior to water exposure. Exposure to ultraviolet (UV) light (psoralen plus UVA [PUVA] and UVB) may be considered as an alternative treatment if others fail.[147,185,211] PUVA and UVB radiation may cause immunosuppression or inhibit mast cell response to the application of water. UV light treatment may increase epidermal thickness, thereby inhibiting penetration of water. It is dosed according to guidelines of the American Academy of Dermatology and delivered by a dermatologist. A case report describes a patient with aquagenic urticaria who responded well to treatment with the anabolic steroid stanozolol. In this study, at a dose of 10 mg/day, symptoms of aquagenic urticaria were controlled.[82]

Aquagenic Pruritus

Definition

Aquagenic pruritus is a condition in which contact with water at any temperature consistently induces intense, disabling itching without any visible cutaneous changes. This condition is rare.[71,89,109] Transient itching of the skin after exposure to water is not unusual. Common etiologies of nonspecific water-mediated itching include dermatitis, xerosis, cold urticaria, heat urticaria, vibratory urticaria, cholinergic urticaria, symptomatic dermographism, polycythemia rubra vera, aquagenic urticaria, aquagenic pruritus of the elderly, and aquagenic pruritus.[259]

Aquagenic pruritus is a distinct clinical entity with clearly described diagnostic criteria. Criteria for diagnosis include: (1) severe pruritus, prickling, stinging, or burning that consistently develops after skin contact with water, regardless of water temperature or salinity; (2) lack of visible cutaneous lesions; (3) reaction within minutes of exposure and lasting between 10 minutes and 2 hours; (4) lack of a concurrent skin disease, internal disorder, or medication to account for the reaction; and (5) exclusion of all other physical urticarias, symptomatic dermographism, and polycythemia rubra vera.[71,259,260] Symptoms may occur only in areas exposed to water. Typically the head, palms, soles, and mucosa are spared.[260] The disabling nature of this intense, unrelenting skin discomfort may account for the mood changes demonstrated by more than half of patients.

Aquagenic pruritus is slightly more prevalent in males, can occur at any age, and may have a familial component.[71,222] Aquagenic pruritus has been associated with hematoproliferative conditions, including polycythemia rubra vera, myelodysplasia, and hypereosinophilic disorders. Recently, an association between histiocytic disorders, such as juvenile xanthogranuloma, and aquagenic pruritus has been established.[115]

Pathogenesis

The pathogenesis of aquagenic pruritus is poorly understood, but it has been demonstrated that water, not other liquids, elicits the symptom complex. Increased mast cell degranulation (with an increase in circulating histamine), release of acetylcholine, and increased cutaneous fibrinolytic activity have all been implicated as potential etiologies for the hypersensitivity component of aquagenic pruritus.[89,109,173] Several neuropeptides play a significant role in the pathophysiology of pruritus as described in the section on cutaneous decompression syndrome.

Treatment

Treatment for aquagenic pruritus has been disappointing. Alkalinization of water and application of petrolatum have had limited success. Administration of antihistamines, anticholinergics, aspirin, iron, serotonin antagonists, propanolol, recombinant interferon-alpha, and triamcinolone is under investigation and has produced inconclusive results.[108,121,194,230] Topical application of capsaicin, the spicy component in the hot pepper plant, has been reported beneficial. It induces release of neuropeptides, including substance P.[121,172] Continued application of capsaicin depletes substance P, thereby blocking further stimulation of nociceptor C fibers, the fibers responsible for the axon reflex in the skin. The use of UV radiation and psoralen photochemotherapy has had promising results. UVB exposure at varying periods of time causes decreased sensitivity to chemical mediators of pruritus. It is postulated that UV irradiation is effective because it may reduce end-organ responsiveness by producing ultrastructural changes within nerves or by raising sensory nerve thresholds.[121,194,250] It may stabilize mast cells at nerve endings, inhibiting the release of histamine. Although impossible, complete avoidance of water exposure is the only guaranteed effective prevention.

Sea Urchin Granuloma

Sea urchin spine injuries frequently develop a sarcoidal granulomatous response (Greek *sarcos,* "flesh"; *eidos,* "form"; so called because its histologic features were originally thought to resemble a sarcoma). This is usually a delayed response to remnants of spine epithelial covering, spine fragments, or associated slime, sand, or surface microbes. Histologically, sarcoidal granulomas are devoid of inflammatory cells other than epithelioid histiocytes. Foreign body giant cells can be seen engulfing spine fragments or other extraneous material. This reaction pattern is the result of an intense inflammatory response to foreign material, antigenic stimuli, or both (Fig. 75-16).

See also Chapter 73.

BACTERIAL INFECTIONS

Pseudomonas aeruginosa

Definition

P. aeruginosa (predominantly serotype 0–11) is a ubiquitous, motile, nonfermentative, obligate aerobic, gram-negative bacillus.[130,195] It is a fastidious organism, surviving in extremes of temperature, under hostile conditions, and with minimal nutritional support. It infects humans, other vertebrates, animals,

Figure 75-16. Sea urchin granuloma. Intradermal nodule near the metacarpophalangeal joint. (Courtesy of Edgar Maeyens, Jr., MD.)

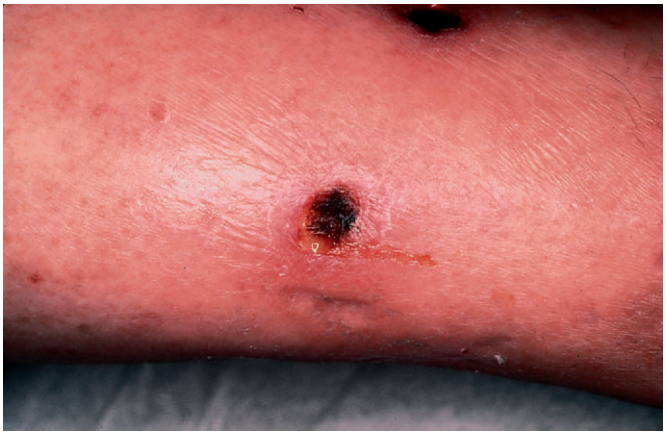


Figure 75-17. Ecthyma gangrenosum. Hemorrhagic necrotic ulcerations on the leg of a man with *Pseudomonas aeruginosa* sepsis. (Courtesy of Edgar Maeyens, Jr., MD.)

and plants. *Pseudomonas aeruginosa* is the most common microbe causing skin disorders in occupational saturation divers and can occur after recreational use of diving suits.[3,161,234]

Pathogenesis

Extracellular toxins and enzymes produced by *P. aeruginosa* contribute to its virulence and are responsible for cutaneous manifestations, including necrotizing subcutaneous nodules, cellulitis, ecthyma gangrenosum, and hemorrhage (Figs. 75-17, 75-18, and 75-19). For *P. aeruginosa* to cause a cutaneous infection, there must be a break in the epidermal barrier in addition to hypersaturation of the stratum corneum. *P. aeruginosa* has a predilection for moist environments. No unusual host factors have been identified. High water temperature, hydration of the skin, high concentration of organisms, and chemical irritants in the water all contribute to this dermatitis. In several outbreaks, deficiencies in disinfection have been noted.

Hot Tub Folliculitis

Definition. One of the more common types of cutaneous infections is hot tub folliculitis. Hot tub folliculitis is an infection of the superficial portion of the hair follicle by *P. aeruginosa*.

Figure 75-18. *Pseudomonas aeruginosa*. Primary infection of the penis in a young man with atopic dermatitis following hot tubbing. (Courtesy of Edgar Maeyens, Jr., MD.)

Figure 75-19. *Pseudomonas aeruginosa*. Primary infection of the forearm in a young man with atopic dermatitis following hot tubbing. (Courtesy of Edgar Maeyens, Jr., MD.)

Signs and Symptoms. This infection is seen most often following immersion in whirlpools or hot tubs, but can occur following swimming or scuba diving (secondary to maceration under the wetsuit). Numerous cases of "hot tub" or "whirlpool" dermatitis have been described.[53,235,287] Eruptions occur after the use of heated recreational water sources, such as whirlpools, hot tubs, swimming pools, and water slides.* Family bathtubs, contaminated bath toys, loofah sponges, moisturizing creams, and diving suits have been implicated in cases of pseudomonal folliculitis.[32,90,96,130] In this situation, hyperhydration of the follicular ostea facilitates colonization by *Pseudomonas*.

An inflammatory response, primarily composed of polymorphonuclear leukocytes, surrounds and infiltrates the follicular epithelium. At this point, one sees a pustule surmounting an erythematous papule or nodule. Depending on the stage of evolution of this infection, purulence may or may not be present and there are only inflammatory papulonodules (Figs. 75-20, 75-21,

*References 76, 112, 131, 136, 191, 214, 235, 236, 241, 254, 270, 301.

Figure 75-20. Hot tub folliculitis. (Courtesy of Edgar Maeyens, Jr., MD.)

Figure 75-21. Hot tub folliculitis. (Courtesy of Edgar Maeyens, Jr., MD.)

and 75-22). Histopathologically and microbiologically, this folliculitis rarely demonstrates the bacterium.

The eruption is perifollicular in distribution and appears within 48 hours of exposure. It usually resolves spontaneously without therapy in 7 to 14 days. The eruption is most pronounced in areas covered by bathing garments, and the head and neck are spared unless they are submerged. The extent and severity of the eruption depends on the concentration of bacteria in the water source, duration of exposure time, presence or absence of preexisting skin disease, water temperature, and individual susceptibility. Other symptoms include external otitis; conjunctivitis; tender breasts; enlarged, tender lymph nodes, fever; and malaise.[130,236] Serious infections arise in immunosuppressed and debilitated individuals. The erythematous,

Figure 75-22. Hot tub folliculitis. (Courtesy of Edgar Maeyens, Jr., MD.)

maculopapular, and vesiculopustular eruption usually is accompanied by pruritus.

Although rare, papules and pustules may reappear for up to several months without re-exposure. Skin lesions heal without scarring but can cause postinflammatory hyperpigmentation that fades with time. Mild pruritus and pain are usual accompaniments. These can be managed with topical antipruritics or oral antihistamines. Rapid progression to severe systemic disease, manifested by hemorrhagic bullae, pneumonia, or septicemia, suggests immunosuppression.[76,101,228,253] *P. aeruginosa* is resistant to many antibiotics. Systemic infection may be treated with an aminoglycoside or ciprofloxacin. Prevention of *P. aeruginosa* infection requires either use of adequate disinfectant or avoidance of recreational closed-water systems and disinfection of reservoirs and vehicles of transmission. Showering does not appear to prevent the disorder.[236]

Green Nail Syndrome

Definition. Green nail syndrome is defined as greenish-black discoloration of a nail plate secondary to the pigment pyocyanin secreted by the bacterium *Pseudomonas aeruginosa*.

Pathophysiology. For this condition to occur, there must be an infection of the paronychial tissues with *Pseudomonas*. *Pseudomonas* colonizes the epidermis, producing inflammation and infection of the paronychial tissues. Pigments diffuse from the skin into the adjoining nail plate, resulting in a greenish-black discoloration. *Pseudomonas* species produce the following fluorescent pigments: pyocyanin (green), pyomelanin (black), and pyorubin (red). In the course of the skin infection, pigments spread into the adjoining nail plate. As a consequence of the infection, the nail plate becomes deformed, thereby allowing further colonization by bacteria, so the cycle progresses (Fig. 75-23). Pain and swelling of the nail fold is inevitable. Often, the infection extends into the hyponychium, resulting in onycholysis.[56] Prolonged or frequent exposure to water, such as tending bar, house cleaning, or water sports, predisposes a person to pseudomonal skin infection of the lateral nail folds and the distal subungual space. Commonly, the cuticle is lost, allowing one or more of the pigments to diffuse into the nail plate with subsequent nail pigmentation. Green nail syndrome is asymptomatic except when there is an acute, painful

Figure 75-23. Green nail syndrome secondary to *Pseudomonas aeruginosa.* (Courtesy of Edgar Maeyens, Jr., MD.)

infection of the nail folds. This condition rarely clears spontaneously.[2,113]

Treatment. Removal of the affected nail plate and elimination of any subungual debris is key to successful therapy. One must avoid excessive exposure to water. Gentamicin sulfate cream or ointment 0.1% applied twice daily to the nail bed and nail folds effects a cure. Occasionally, a topical steroid cream (see Table 75-3) is used to eliminate inflammation and swelling of the paronychial tissues. Within approximately 3 months, the fingernail and cuticle regrow.

Otitis Externa

Definition. Otitis externa is an infection of the pinna and external auditory canal, most commonly caused by *P. aeruginosa* and *Staphylococcus aureus*.[127,300]

Etiology. Infection of the external ear canal is the most common of all aquatic dermatoses. It causes extreme discomfort and interferes with participation in aquatic activities. The incidence of otitis externa is directly related to the duration of exposure to water. Otitis externa is referred to as "swimmer's ear" (Fig. 75-24) because swimmers and scuba and deep-sea divers frequently are afflicted with this problem. The adult ear canal is a cul-de-sac approximately 5 mm in diameter and 25 mm in length lined with stratified squamous epithelium. The outer one third of the ear canal has a physiologic barrier to infection consisting of an acidic waxy mantle with sloughed epithelial cells. However, this barrier is not present to the same degree on the thinner and more delicate epithelium of the inner two thirds of the ear canal.[262]

Pathophysiology. The pathophysiology of otitis externa involves the interaction of moisture retention, moderate to high temperatures, and infection. Other factors that predispose a diver to the development of otitis externa include canal occlusion (e.g., exostoses, cerumen plugs, entrapped particles of sand, earplugs), trauma related to mechanical attempts to clean the ear canal, intrinsic dermatoses (e.g., allergic conditions, eczema, psoriasis, contact and seborrheic dermatitis, neurodermatitis), cerumen degradation, and pH variation above normal pH of 4 to 5.

Figure 75-24. Otitis externa. Scaling and erythema of the external auditory canal and surrounding pinna colonized by *Staphylococcus aureus* and *Pseudomonas aeruginosa.* (Courtesy of Edgar Maeyens, Jr., MD.)

Signs and Symptoms. The initial symptoms of otitis externa include itching of the ear canal, pain, pressure or fullness within the ear, and diminished hearing. As the inflammatory process within the ear canal progresses, the pain becomes more intense. Pain is noted upon applying pressure to the external auditory meatus or the tragus, or pulling the ear lobe. A severe infection may be associated with or progress to otitis media, canal occlusion, cervical lymphadenopathy, headache, nausea, fever, toxemia, and cellulitis with associated purulent discharge. Infection can spread to the periauricular soft tissues, parotid gland, and temporomandibular joint. A secondary periauricular infectious eczematous dermatitis may occur when exudate from the ear canal discharges onto the surrounding skin.

Otitis externa is polymicrobial, so other bacterial isolates may include *Acinetobacter calcoaceticus, Proteus mirabilis, Enterococcus faecalis, Bacteroides fragilis,* and *Peptostreptococcus magnus*.[59,127,300] Otitis externa does not appear to be associated with bacterial indicators of recreational water quality, such as fecal coliform bacteria, enterococci, or *Pseudomonas* organisms.[46] A recent study showed that even when current water standards for recreational aquatic activities are met, swimming can increase an individual's risk of otitis externa because of exposure to *P. aeruginosa*.[277]

Treatment. Treatment is directed at changing the environment within the ear canal. The goals of topical therapy are reacidification and desiccation of the ear canal, accomplished by the topical administration of a mixture of alcohol and acetic acid (vinegar) or Burow's solution (aluminum sulfate and calcium acetate [Domeboro]). Mild otitis externa characterized by slight pain and itching can be treated with eardrops alone or with a mild topical corticosteroid (see Table 75-3). An excellent topical preparation is nonaqueous acetic acid with or without hydro-

Figure 75-25. Malignant otitis externa. (Courtesy of Edgar Maeyens, Jr., MD.)

Figure 75-26. *Aeromonas hydrophila.* Trauma-induced necrotic ulcer of the anterior leg of a fisherman. (Courtesy of Edgar Maeyens, Jr., MD.)

cortisone 1% (VoSol Otic). If suppuration occurs, antibiotic eardrops, such as hydrocortisone 1%, polymyxin B, and neomycin (Cortisporin Otic) or 0.3% ofloxacin otic, are indicated.[144] Products not currently recommended include benzocaine, antipyrine, camphor, ichthammol, and thymol.[262] If the ear canal is so swollen that drops cannot be instilled, a gauze wick should be placed and continually soaked with the topical agent for 24 to 72 hours. Systemic antibiotics, such as oral trimethoprim-sulfamethoxazole, amoxicillin-clavulanate, or ciprofloxacin for 7 to 10 days, are indicated if cellulitis, adenopathy, profuse purulent discharge, or fever is present. In patients who do not respond within 2 days to the standard antimicrobial management, a course of antibiotics that cover anaerobic bacteria is warranted.[59] Intravenous antibiotics are indicated when the infection worsens or does not resolve rapidly within 48 hours.

Analgesics are required for pain control. Systemic steroids should be avoided unless an underlying dermatosis exists because they may interfere with the host's response to infection. Oily suspensions or moisturizers must be avoided. Otitis externa rarely progresses to the life-threatening condition of malignant otitis externa, which is a severe, progressive, and destructive infection of the pinna characterized by excessive granulation tissue at the junction of the osseous and cartilaginous portions of the auditory canal (Fig. 75-25). Malignant otitis externa characteristically occurs in diabetics and immunosuppressed individuals. When this condition does not respond to other treatment measures, some suggest that hyperbaric oxygen can be effective.[64a]

Prevention. To prevent otitis externa, the individual must take prophylactic measures intended to inhibit moisture accumulation within the external ear canal. Head shaking, tilting, fanning, use of a blow dryer, and use of desiccating and acidifying agents such as diluted vinegar (1 teaspoon of vinegar to 1 pint of water) applied to the auditory canal three times daily for 1 to 2 days and followed with topical corticosteroids (see Table 75-3) all prevent moisture accumulation and decrease the incidence of otitis externa. Cotton-tipped applicators should not be used to absorb moisture or remove cerumen because they may damage the ear lining or press cerumen deeper into the canal. Cerumen softening products are not useful because they only soften hardened or impacted cerumen without removing it. If warranted, a trained professional may remove cerumen with irrigation or curettage. Earplugs should not be used because they produce ischemia of the ear canal lining. Oily substances, such as petroleum jelly, should not be used to form a water-tight seal because they will act as a moisture trap for debris, increasing the incidence of otitis externa.

Aeromonas hydrophila

Definition

Aeromonas hydrophila is a gram-negative, facultatively anaerobic, polarly flagellated, non–spore-forming, motile rod member of the family Vibrionaceae that commonly inhabits soil, freshwater streams, and lakes.[97,116,146,150,230,284] *Aeromonas* species are widely distributed and found at wide ranges of temperature and pH.[122,283] Three species (*Aeromonas hydrophila, A. sobia,* and *A. caviae*) have been associated with human disease. Medicinal leeches can harbor *Aeromonas* species in their gut flora; soft tissue infections related to leech attachment have been reported.[285]

See also Chapter 72.

Signs and Symptoms

Although *A. hydrophila* may produce soft tissue infection or cellulitis in normal hosts, serious infection is more commonly noted in immunocompromised persons.[188,249,291,297] *A. hydrophila* may cause gastroenteritis, endocarditis, peritonitis, meningitis, and septicemia. Soft tissue and gastroenteric human infections occur predominantly between May and November.[137,283,292] A wound (frequently of the lower extremity) immersed in contaminated water may develop cellulitis within 24 hours, with erythema, edema, and a purulent discharge[291,294] (Fig. 75-26). The wound is usually sustained when the individual steps on a foreign object or suffers a laceration or puncture wound while in water.[151,238] The symptoms may be indistinguishable from those of streptococcal cellulitis, with localized pain, lymphangitis, fever, and chills. If managed with inappropriate antibiotics or if untreated, this infection may progress to a severe gas-forming soft tissue reaction, bullae formation, necrotizing myositis, or osteomyelitis.[66,97,150,167,249,291] The appearance of aeromonad septicemia is reportedly similar to septicemia subsequent to ecthyma gangrenosum caused by

P. aeruginosa.[297] In a manner analogous to the pathogenicity of virulent *Vibrio* species, the chronically ill or immunocompromised host probably is at greater risk for severe complications, such as meningitis, endocarditis, or septicemia.[67,97,150,167,170,249,291,297] In the individual who has aspirated freshwater contaminated with *A. hydrophila,* pneumonitis and bacteremia may develop.[221] Infections also have occurred after individuals have been bitten by an alligator or a pet piranha.[223,230] For unknown reasons, there is a marked preponderance of male patients.[15]

Treatment

A. hydrophila generally is sensitive to tetracycline, chloramphenicol, gentamicin, tobramycin, trimethoprim-sulfamethoxazole, cefotaxime, moxalactam, imipenem, ceftazidime, and ciprofloxacin.[12,80,150,208] However, it is typically resistant to penicillin. In one case, culture of a severe wound infection demonstrated the presence of both *A. hydrophila* and *A. sobia*.[146] Notably, the latter species was resistant to tetracycline in vitro. Initial therapy for a severe infection should include an aminoglycoside to provide coverage against concomitant *Pseudomonas* or *Serratia* species infection.[116] As has been demonstrated with *Vibrio* species, the first-generation cephalosporins, penicillin, and ampicillin are not effective against *A. hydrophila,* perhaps because of the production of β-lactamase by the organism.[12,41,80,81,208] In addition to antibiotic therapy, initial treatment of a severe soft tissue infection related to *Aeromonas* organisms should include aggressive wound debridement and adequate drainage to mitigate the potentially invasive nature of the organism. A case of *A. hydrophilia* cellulitis that did not respond to antimicrobials and surgical debridement responded successfully to adjunct hyperbaric oxygen therapy.[61,188] High oxygen concentrations may suppress virulence factors and inhibit or kill anaerobic organisms.[61]

Because of the microbiologic similarity of *Aeromonas* species on biochemical testing to members of the Enterobacteriaceae family, such as *Escherichia coli* and *Serratia* species, it is important to alert the laboratory to the clinical setting. A Gram stain of the purulent discharge may demonstrate gram-negative bacilli. Given the appropriate clinical setting (after a wound has been acquired in the freshwater environment), this finding should not be casually attributed to contamination.[151]

Figure 75-27. *Chromobacterium violaceum.* A minor abrasion while snorkeling led to this forearm infection. (Courtesy of Edgar Maeyens, Jr., MD.)

Figure 75-28. *Chromobacterium violaceium.* Lymphangitis of the forearm. (Courtesy of Edgar Maeyens, Jr., MD.)

Chromobacterium violaceum

Definition

Chromobacterium violaceum is a mesophilic (prefers a temperature of 20° C to 37° C [68° F to 98° F]), saprophytic, facultatively anaerobic, and gram-negative rod. It is motile, with both polar and lateral flagella. This bacterium produces a violet pigment (violacein) during oxidative and fermentative glucose use. It is an abundant inhabitant of tropical and subtropical freshwater rivers and soil, especially in the southeastern United States, and is rarely considered pathogenic.[133,196]

Signs and Symptoms

The infection usually presents after the organism enters an individual's body through a minor skin injury or after the individual ingests contaminated water.[139,196,202,218,257] Sepsis is sometimes a feature of this infection. Bacteremia leads to diffuse pustular dermatitis, with *C. violaceum* cultured from both blood and skin pustules.[257] Other skin manifestations include vesicles, ecchymotic maculae, maculopapular eruption, ulcers (Fig. 75-27), subcutaneous nodules, cellulitis, lymphangitis (Fig. 75-28), and digital gangrene.[177] Additional manifestations are secondary to systemic disease and include suppurative lymphadenitis; osteomyelitis; urinary tract infection; sinusitis; orbital cellulitis; lung, kidney, and liver abscesses; and meningitis.[177,196,257,274,282] Persons with chronic granulomatous disease, which is characterized by the inability of granulocytes and monocytes to convert oxygen into hydrogen peroxide and other oxygen metabolites necessary for microbicidal activity, are at particular risk for severe infection.[104,141,177] Fortunately, *C. violaceum* infection, which is potentially fatal and associated with a high mortality rate, is rare. Of 34 reported cases, 65% were fatal.[218] The interval between the onset of the illness and death ranged from 7 to 15 months. Although most patients with *C. violaceum* infection are not immunocompromised, the presence of immunodeficiency, such as that caused by chronic granulomatous disease, can predispose to infection.

Treatment

Treatment consists of antibiotics and prompt surgical drainage of purulent collections. The organism is generally sensitive to imipenem, carbenicillin, ciprofloxacin, chloramphenicol, tetracycline, gentamicin, kanamycin, tobramycin, and trimethoprim-sulfamethoxazole. Resistance to cephalosporins, penicillins, and aztreonam has been reported.[196,257,264] Physicians in the southeastern United States should be highly suspicious of *C. violaceum* when caring for a septic patient with a history of exposure to stagnant water and typical skin lesions, organ abscesses, or both.

Mycobacterium marinum

Definition

Mycobacterium marinum is an atypical mycobacterium found in freshwater and saltwater environs.[107,225] It is an acid-fast, rod-shaped bacillus of the Runyon group 1. It is a photochromagen in that it produces yellow-orange pigment after light exposure. In contradistinction to typical mycobacteria, which cannot multiply outside an animal host, atypical mycobacteria are free-living soil and water saprophytes, rarely pathogens. Optimal growth occurs at 31° to 33°C (88° to 91°F). The organism is resistant to chlorine as a disinfectant. Human infections are limited to cooler acral locations such as forearms, hands, feet, and legs and rarely involve deeper, warmer soft tissues.[107]

Pathophysiology

Cutaneous lesions are typically granulomatous, although deeper infections, including bursitis, tenosynovitis, septic arthritis, and osteomyelitis, may occur.[7,21,58,118,163,240,252] *M. marinum* may be rapidly invasive, leading to sepsis in both healthy and immunocompromised individuals.[83,132] Infections may occur after exposure to fresh, salt, or brackish water and have been reported in commercial fish handlers, aquarium tenders, pool cleaners, and dolphin trainers.[35,39,62,70,92,142,160,163,252] *M. marinum* invades tissues through a break in the skin.

Signs and Symptoms

Three types of lesions occur: ascending lymphatic sporotrichoid lesions (nodular lymphangitis), a solitary granulomatous verrucous papule or nodule that may ulcerate and express purulent drainage, and rare disseminated cutaneous lesions.[103,216] Seven to 10 days after the individual sustains a puncture wound or laceration, particularly of a cooler distal extremity, a localized area of cellulitis develops. Serious sequelae can ensue. In the absence of appropriate treatment, the cellulitis may progress to localized arthritis, bony erosion, formation of subcutaneous nodules, and superficial desquamation.[17,129,158,295] Aspiration of a joint effusion may reveal the acid-fast bacilli. Alternatively, within 3 to 4 weeks of the primary inoculation, a red papule develops and slowly grows, becoming a hard purple nodule. This nodule often is scaly, ulcerated, and verrucous with a violaceous base, resembling tuberculosis verrucosa cutis.[84,157,171,237,244] The lesion may enlarge to 6 cm ($2^1/_2$ inches) in diameter, although a diameter of 1 to 2 cm is more common. New lesions may develop in a pattern that resembles sporotrichosis, with dermal granulomas in a linear distribution along the superficial lymphatics (Figs. 75-29 and 75-30). These granulomata may become secondarily infected, resulting in polymicrobial cellulitis or lymphangitis. The differential diagnosis of the nodular lymphangitis includes sporotrichosis, cutaneous leishmaniasis, nocardiosis, verrucous lichen planus, verruca vulgaris, iodine and bromine granulomas, sarcoidosis, syphilis, gout, other cutaneous mycobacteriosis, and chronic pyogenic infections.[140] Risk factors for the development of cutaneous atypical mycobacterial infection include immunosuppression

Figure 75-29. *Mycobacterium marinum.* Cutaneous granulomas on the forearm. (Courtesy of Edgar Maeyens, Jr., MD.)

Figure 75-30. Nodular lymphangitis in *Mycobacterium marinum.* (Courtesy of Edgar Maeyens, Jr., MD.)

secondary to lymphoma, leukemia, HIV infection, and medical therapies that alter host immune response.

Diagnosis

The diagnosis of *Mycobacterium marinum* is confirmed by wound culture or by isolation of the organism from a homogenized skin biopsy (minimum 4 mm punch) specimen on standard mycobacterial culture medium (Lowenstein-Jensen) at 31°C to 33°C (88°F to 91°F), rather than the standard 37°C (98°F).[7,100,140,160,293] Acid-fast bacilli are visualized directly in only 10% to 13% of biopsies, and approximately 50% of cultures are positive.[79] Cutaneous intradermal skin tests show cross reactivity between *M. marinum* and *M. tuberculosis*.

Treatment

The literature does not indicate consensus on treatment. Conservative therapy, aggressive surgical excision, and multiple antibiotic therapies have been described.[7,47,72,103,163,165,276] Because most lesions are self-limited and heal spontaneously within a few months to 3 years, leaving only scarring and residual pigmentation, some advocate only conservative therapy. Small lesions often are easily excised, but may recur. *M. marinum* is susceptible to many antibiotics. Those used previously with inconsistent results include amoxicillin-clavulanate, azithromycin, amikacin, kanamycin, clarithromycin, minocycline, tetracycline, levofloxacin, trimethoprim-sulfamethoxazole, and tuberculostatic drugs, such as streptomycin, isoniazid, ethambutol, and rifampin.* Because clinical evidence regarding the most efficacious treatment of *M. marinum* is lacking, the current recommendation is to treat the patient empirically with one of the aforementioned antibiotics until sensitivities are obtained.[39,171] Susceptibility testing of *M. marinum* usually takes several weeks to perform due to the slow growth of this organism, and therefore, empiric therapy is implemented.[163] Recent reports favor the use of trimethoprim-sulfamethoxazole or ethambutol plus rifampin as first-line therapy.[72,160,163] Trimethoprim-sulfamethoxazole is safe, efficacious, and inexpensive.[160] Subsequent treatment should be based on the sensitivity and clinical response. All studies agree that antibiotic treatment should be long term, with the duration of treatment varying from months to years.[72,103] Although the growth rate of the organism is sensitive to heat degradation both in vitro and in vivo, heat therapy is no longer recommended. Intralesional steroids are contraindicated.[75]

Vibrio vulnificus

Definition

V. vulnificus is a curved, flagellated, gram-negative rod. The genus *Vibrio* is classified in the family Vibrionaceae, along with the genera *Photobacterium, Aeromonas,* and *Plesiomonas*.[11] *Vibrio vulnificus* is a part of normal marine flora and is recognized as a virulent pathogen in the United States and other parts of the world. The organism has been isolated in warm (20°C [68°F] or warmer) coastal water and in water with salinity of 0.7% to 1.6%.[203,223] It has been found in brackish inland waters (see Chapter 72). *Vibrio vulnificus* may be found in high concentrations in filter-feeding sea life, such as aquatic animals, oysters, mussels, clams, scallops and crabs, and also fish inhabiting coral reefs.[263]

*References 21, 30, 39, 58, 70, 72, 84, 94, 100, 134, 153, 157, 159, 163, 165, 171, 210, 220, 237, 244, 252, 276, 289.

Figure 75-31. *Vibrio vulnificus* infection. (Courtesy of Edgar Maeyens, Jr., MD.)

Signs and Symptoms

Vibrio vulnificus gains entry into the skin by direct inoculation or indirectly via the bloodstream during septicemia.[206] Inoculation results from contamination of minor skin wounds, lacerations, and punctures by contaminated objects or from skin trauma suffered while exposed to contaminated water. Victims frequently report having handled shellfish or fish in a marine environment when they obtained a small wound. The wound rapidly progresses to a painful cellulitis that can be characterized by various clinical signs that include edema, erythema, or ecchymosis.[27] Initially, there is erythema, edema, and pain, which if not promptly treated, progresses to bulla formation and necrosis of cutaneous vessels, collagen, fascia, and muscle[152] (Figs. 75-31 and 75-32). The stage where the infection has progressed to the point of fasciitis, septicemia is virtually inevitable.[213]

Pathophysiology and Histology

The lesions caused by *Vibrio vulnificus* may be attributed to destructive activities of proteolytic, collagenolytic, and elastase enzymes. Later stages of cutaneous *V. vulnificus* infections are characterized by necrosis of the epidermis, dermis, and subcutaneous fat. There is a mixed inflammatory cell infiltrate composed of neutrophils, lymphocytes, and histiocytes. Blood vessels are thrombosed and infiltrated by inflammatory cells.

Treatment

Because of the severity and rapid progression of *V. vulnificus* wound infection, prompt diagnosis and antimicrobial treatment, along with early surgical debridement, are mandated.[138] Antibiotics are the mainstay of therapy. *Vibrio vulnificus* shows sensitivity to doxycycline, ciprofloxacin, chloramphenicol, gentamicin, imipenem-cilastatin, third-generation cephalosporins, amikacin, tobramycin, aztreonam, and trimethoprim-

Figure 75-32. *Vibrio vulnificus* infection. (Courtesy of Edgar Maeyens, Jr., MD.)

sulfamethoxazole. Multiple antimicrobials may be required. Surgical debridement and fasciotomies are necessary to control infection in the presence of necrotizing fasciitis.[138] Despite prompt diagnosis and treatment; the mortality rate remains high, especially in persons who are chronically ill or immunologically compromised.

Erysipelothrix rhusiopathiae

Definition

Erysipelothrix rhusiopathiae, formerly known as *Erysipelothrix insidiosa,* is a facultatively aerobic, nonmotile, non–spore-forming bacillus. *Erysipelothrix* is hardy enough to survive saltwater or freshwater exposure, drying, salting, pickling, and smoking. It can survive 12 days in direct sunlight, 4 months in putrefied flesh, and 9 months in a buried carcass. It is the cause of the acute skin infection called erysipeloid. Erysipeloid occurs in fishermen, butchers, and others who handle raw fish, crab, poultry, or meat products.[99,229]

Signs and Symptoms

Erysipelothrix enters the skin through a puncture wound or abrasion, usually on the finger or hand. Between 1 and 7 days later, a lesion develops. The puncture wound takes on the appearance of a minor purple-red skin irritation or infected paronychia, with edema and a small amount of purulent discharge. This lesion is surrounded by an area of central fading or noninvolvement ("clearing"), which is surrounded by a centripetally advancing, raised, well demarcated, and marginated erythematous or violaceous ring. The lesion located on the finger or hand is usually warm and tender, with characteristic proximal progression along the dorsal edge of the finger into the web space and then distally along the adjoining finger[290] (Fig. 75-33). The lesion is painful and often pruritic, with regional painful adenopathy.

Figure 75-33. Typical appearance of *Erysipelothrix* skin infection. (Courtesy of Paul S. Auerbach, MD.)

Figure 75-34. *Erysipelothrix rhusiopathiae* infection. (Courtesy of Edgar Maeyens, Jr., MD.)

Pitting and suppuration are absent. The infection seldom affects the palm. Although the infection is generally limited to the hand, it may spread to the wrist or forearm.[155] Low-grade fever and malaise are not uncommon. In the diffuse cutaneous form, the local lesion enlarges and dissemination results in multiple satellite lesions distant from the original site, occasionally with vesiculation or formation of large bullae[74] (Fig. 75-34). Bacteremia, severe generalized cutaneous infections, systemic infections, and endocarditis are rare.[84,110]

Diagnosis

E. rhusiopathiae may be cultured from biopsy tissue specimen or by injecting sterile (not bacteriostatic) saline into the edge of the lesion and then aspirating without withdrawing the needle.[300] For transport, the specimen should be immediately placed into an infusion broth containing 1% glucose. The microbiology laboratory should be alerted to the possible presence of *E. rhusiopathiae* because it is closely related bacteriologically to *Listeria monocytogenes* and *Corynebacterium* species and may be easily confused on Gram stain with streptococci or diphtheroids.[110]

Treatment

Erysipeloid usually is self-limited and runs its course within 3 weeks. For isolated skin involvement, penicillin VK or cephalexin (250 to 500 mg by mouth four times daily) or ciprofloxacin (500 to 750 mg by mouth twice daily) is rapidly effective.[20] Erythromycin is no longer recommended. *E. rhusiopathiae* is resistant to aminoglycoside antibiotics.[243] If arthritis, septicemia, or endocarditis is present, aqueous penicillin G

should be administered in a dose of 2 to 4 million units intravenously every 4 hours for 4 to 6 weeks.

Shewanella putrefaciens

Definition
Shewanella putrefaciens is a marine gram-negative bacterium identifiable by its biochemical reactions at 24 hours.[98] It is a ubiquitous bacterium isolated from seawater, soil, fish, and oily foods. In the food industry, it is a common cause of spoilage of marine fish.[16] This organism has rarely been isolated clinically.[16]

Signs and Symptoms
This is a rare human pathogen that has been associated with lower limb ulceration, otitis media in divers, peritonitis, and bacteremia. Direct contact with seawater and inoculation of a preexisting wound are potential mechanisms of infection. Infection manifests as cellulitic ulcerations. Complications include purulent inflammation and sepsis. *Shewanella putrefaciens* has been documented to produce the extracellular enzymes lecithinase, lipase, and DNAase. These enzymes may be involved in the destruction of skin and subcutaneous tissue.

Treatment
Ciprofloxacin 500 mg bid has been successfully used to treat this infection.[210] Aminoglycosides and chloramphenicol are also effective.

▶ *MYCOPLASMA*

Definition
Seal finger is the sobriquet given to label a unique infection, usually of a digit, acquired by exposure to Pinnipedia (seals, walruses, and sea lions).[119] Characteristically, after exposure to the skin or mucous membranes of one of these animals, the infected digit becomes swollen and very painful. If treatment is not instituted promptly, damage to the joints of the infected finger(s) can ensue.[69,179,186]

Pathogenesis
This disorder is thought to be secondary to infection with *Mycoplasma* strains *(M. phocarhinis, M. phocacerebrale)* initially isolated from seals.[19] These strains are new species of *Mycoplasma* unique to seals and possibly to other pinnipeds.[119] Some believe that these *Mycoplasma* species may be part of the normal flora of seals.[178] Several species of *Mycoplasma* are commensals in the animal's genitourinary tract and in the human oral cavity and genital mucosa.[22] They represent the smallest known free-living forms of microbes.[22] In fact, when initially discovered, they were thought to be viruses.

Signs and Symptoms
The clinical manifestations begin after an incubation period of hours to 3 to 4 days, although some report an incubation period of 1 to 15 days.[19,119] Initially, an inflammatory papule occurs that promptly develops into a nodule with swelling, slight purulence, and severe pain (Fig. 75-35). Edema and stiffness of the digit are common sequelae.[69,186] If joint involvement occurs, it is usually that which is nearest the site of entry of the organism.[119] The affected joint becomes swollen, inflamed, and stiff. Untreated, atrophy of cartilage, bone resorption, and ultimately, arthrosis develop.[186] Secondary bacterial infection may occur in the skin lesions, which manifests as purulence and lymphangitis. Fever and leukocytosis may accompany infection. Seal finger

Figure 75-35. Seal finger secondary to *Mycoplasma.* (Courtesy of Edgar Maeyens, Jr., MD.)

can resolve spontaneously with little or no sequelae or can be protracted over several months. No immunity is conferred.[119]

Prevention
Prevention is simply being aware of the potential for infection when handling pinnipeds or their by-products, good hygiene, wearing gloves, and washing with soap and water after every exposure. If the integrity of the skin is breached, as with a laceration or cut, seek immediate attention and treatment with tetracycline.

Treatment
Tetracycline is given orally at 1.5 g initially and 500 mg four times a day for 4 to 6 weeks.[119] The sooner therapy is begun, the greater the chance for resolution of the infection and avoidance of joint destruction.

▶ YEAST

Pityrosporum folliculitis

Definition
Pityrosporum folliculitis is a condition occurring in young or middle-aged adults and characterized by follicular papulopustules primarily involving the upper torso. Less frequently, this condition occurs on the face.[14] The pathogenesis follows overgrowth of normal yeast flora of the genus *Pityrosporum*. Two species commonly associated with *Pityrosporum* folliculitis are *P. ovale* and *P. orbiculare* which, collectively, are classified as *Malassezia furfur.*[166]

Pathophysiology
Malassezia furfur are saprophytic, lipophilic, dimorphic, gram-positive, double-walled, and budding yeasts.[14] Growth and proliferation occur in an environment rich in free fatty acids. Sebaceous glands attached to follicles are the source of triglycerides, which, when metabolized by *Malassezia furfur* into free fatty acids, stimulate an inflammatory response within the follicle.[40,183]

Signs and Symptoms
This is manifested clinically as acneiform folliculitis. The lesions are typically dome-shaped papules of 2 to 4 mm in diameter.

Figure 75-36. *Pityrosporum folliculitis.* Follicular papulopustules on the back of a scuba diver after removing his wetsuit. (Courtesy of Edgar Maeyens, Jr., MD.)

Some papules are topped by tiny pustules. Pruritus is common. The surface of normal skin is colonized by *Pityrosporum* species in 90% to 100% of humans.[126] Individuals living in humid and hot climates experience an increased incidence of *Pityrosporum* folliculitis.[14] In addition, cosmetics, sunscreens, body lotions, and occlusive clothing in this environmental setting are predisposing factors.[14] Scuba divers are prime candidates for *Pityrosporum* folliculitis as their skin is hydrated, macerated, and occluded by wearing a wetsuit (Fig. 75-36).

Medical disorders predisposing to *Pityrosporum* folliculitis include diabetes, leukemia, lymphoma, and immunodeficiency states, either infectious or pharmacologic in origin.[40,77] Nonmedical predisposing agents include antibiotics, anticonvulsants, immunosuppressants, and systemic corticosteroids.[40] All of these medications tend to alter normal skin flora, favoring proliferation of yeasts.

Treatment

Treatment is with either topical or systemic antifungals, or both.[93] Of the several antifungals available, these authors recommend oral ketoconazole in combination with ketoconazole cream (Nizoral 2%). The adult dosage is 200 mg once daily for 2 to 3 weeks plus twice daily application of the cream for 4 weeks. For children older than 2 years, the oral dosage is 3.3 to 6.6 mg/kg once daily and the topical regimen is the same as for adults.

WORMS AND LARVAE

Schistosome Cercarial Dermatitis

Definition

Schistosome cercarial dermatitis, also known as "swimmer's itch," is the result of an inflammatory response to cutaneous infestation with one of any of several water-borne trematode parasites. Some examples are the genera *Trichobilharzia, Ornithobilharzia, Gigantobilharzia, Orientobilharzia, Austrobilharzia,* and *Bilharziella.*[10,128,180,233] The geographic distribution of this dermatosis is worldwide, occurring in Arctic, temperate, and tropical zones. It is most commonly seen after exposure to cercariae-laden fresh or brackish water or, less often, saltwater.[169] This process is nearly exclusively cutaneous, but rarely, systemic effects such as fever and malaise occur.[55]

Life Cycle

The definitive hosts of these nonhuman schistosomes are aquatic birds, such as ducks, gulls, and geese, and mammals such as beavers, mice, muskrats, and ungulates. Eggs of the adult schistosomes are eliminated via the feces of infested hosts. If eggs are deposited in water, they hatch, releasing small free-swimming larvae (miracidium). Upon finding specific snail intermediate hosts, they penetrate soft snail flesh and metamorphose into different larval form, the cercariae. Cercariae exit snails in search of definitive avian or mammalian hosts to complete the life cycle[23] (Fig. 75-37).

Most cercariae are released during the warm mid-day hours from 12:00 P.M. to 2:00 P.M. They lack free-swimming skills, so they congregate at the water's surface. Currents and wind carry the cercariae miles from their origin.[52] Once released from snails, cercariae are infective for 24 hours. One infected snail can release up to 4000 cercariae per day.[18] The larvae are more likely to be found at the shoreline where snails are most numerous.[42]

Pathophysiology

Penetration of human skin by cercariae induces a localized inflammatory response. The degree of inflammation varies depending on previous exposure to and the numbers of penetrating cercariae. Once in human skin, these nonhuman cercariae are unable to develop further, and are destroyed by the inflammatory infiltrate. The clinical presentation is predicated on the magnitude of inflammatory response. As an example, mild inflammation produces macules and papules, whereas marked inflammation results in vesicles.[204]

Histopathology

The epidermis shows varying degrees of spongiosis. There is edema of the upper dermis and a superficial perivascular inflammatory infiltrate composed of lymphocytes, histiocytes, and eosinophils.[38,299] This histologic pattern is similar to that seen in arthropod assaults and sea bather's eruption.[298]

Signs and Symptoms

Within minutes of epidermal or subsequent dermal penetration, tingling, burning, and itching appear. Initially, an erythematous macule develops, which promptly evolves into a papule. If the allergic response is severe, papules vesiculate (Figs. 75-38 and 75-39). Without therapy, the eruption disappears in 7 to 14 days.[128] Exposed body surfaces are prime locations; however, covered surfaces may also be involved.[42] Itching with scratching can lead to secondary infection, most commonly with bacteria such as *Staphylococcus aureus* or *Streptococcus* species.

Risk Reduction

Methods to reduce the risk of cercarial dermatitis include the following[52,54]:

1. Avoid wading or swimming in marshy areas, areas with dense vegetation, or other areas where snails are plentiful.
2. Vigorously towel dry immediately upon exiting the water. This mechanically removes the larvae.
3. Swim as far away from the shore as safety allows.
4. Obey posted signs noting unsafe water.
5. Use of waterproof sunscreens has been reported to prevent infestation.

Treatment

Because the inflammatory response is self-limited, therapy need only be symptomatic. Use of over-the-counter antihistamines plus antipruritic lotions or creams (see Table 75-1) are usually all

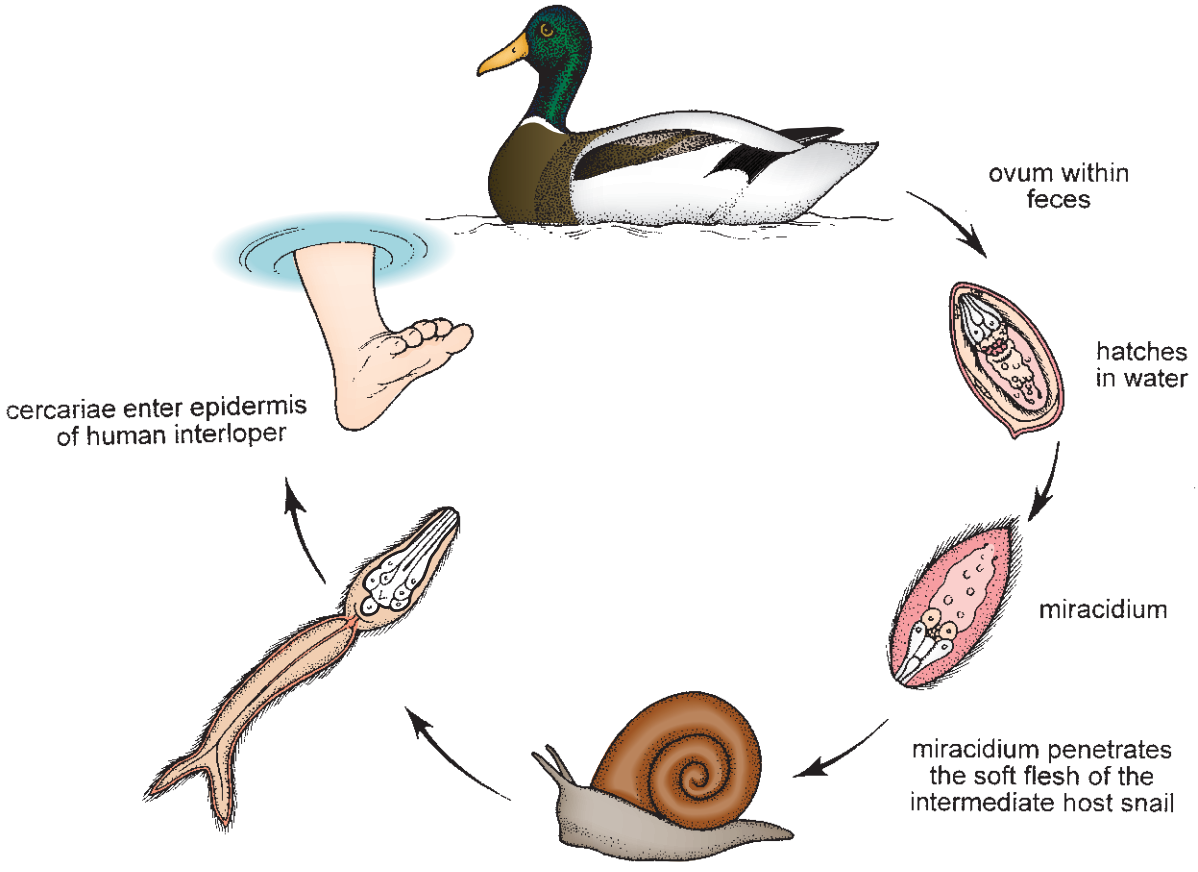


Figure 75-37. Schistosome cercarial life cycle. (Courtesy of Edgar Maeyens, Jr., MD.)

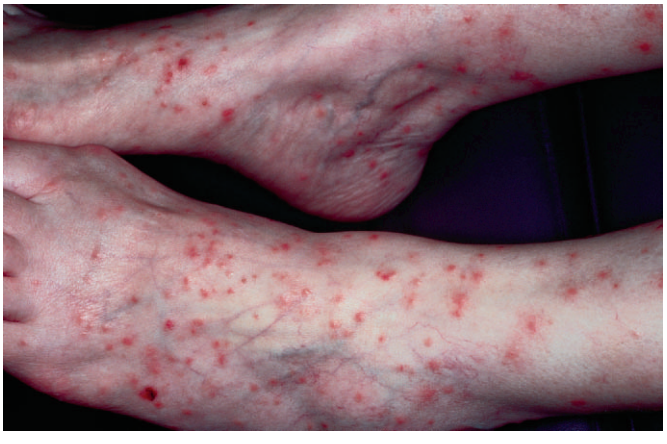


Figure 75-38. Schistosome cercarial dermatitis of the feet and ankles. (Courtesy of Edgar Maeyens, Jr., MD.)

Figure 75-39. Schistosome cercarial dermatitis: Multiple papulovesicles in a sensitized individual following clam digging. (Courtesy of Edgar Maeyens, Jr., MD.)

that is needed. If a moderate to severe dermatitis occurs, topical steroid creams (see Table 75-3) in conjunction with parenteral corticosteroids (see Table 75-2) may be indicated.[51] Secondary infection, depending on the degree and extent, is treated with topical antiseptic ointments or creams (mupirocin or bacitracin) or systemic antibiotics (cephalexin or erythromycin). Cephalexin dosage is 500 mg four times per day for 7 to 10 days and erythromycin 250 mg four times per day for 7 to 10 days. Culture and sensitivity are indicated when necessary to precisely identify the infecting organism and establish appropriate antibiosis.

Figure 75-40. Cutaneous larva migrans. (Courtesy of Edgar Maeyens, Jr., MD.)

Cutaneous Larva Migrans

Definition

Cutaneous larva migrans ("creeping eruption") is a superficial infestation of the skin usually caused by *Ancylostoma braziliensis*, the canine and feline hookworm. Other less commonly reported species that can cause this condition are *Ancylostoma caninum*, *Gnathostoma spinigerum*, *Uncinaria stenocephala*, and *Bunostomum phlebotomum*.[125,224] Although this disorder is not directly related to marine environs, it is occasionally acquired by humans while participating in water-related recreational activities. Hookworm-infested dogs and cats shed eggs of the hookworm in their feces, which, when in a moist and temperate environment, metamorphose into infectious larvae. A transient creeping eruption also results from infestation with the larvae of the human hookworms *Ancylostoma duodenale* and *Necator americanus*.

Pathophysiology

Humans can become accidental hosts when they contact soil that has been contaminated with the feces of parasitically infested animals. Eggs are eliminated in the feces, and the ova that are deposited in the soil hatch into larvae, which can penetrate human skin. Soil in sandy, warm, and shady areas (e.g., above the ebb and flow of the ocean) is most favorable for larvae. Other persons commonly affected include ocean bathers, children, gardeners, farmers, and those who work under buildings. Athletes also have been affected.[26]

Signs and Symptoms

In humans, the larvae penetrate the epidermis and lodge either at the epidermal–dermal junction or in the papillary dermis. The larvae migrate along the upper dermis, leaving in their wake inflammatory serpiginous tracts called lava currens. Tracks can be single or multiple, depending on the degree of infestation. In at least one quarter of the afflicted, only a nonspecific dermatitis exists for a few weeks prior to the onset of the migration (Fig. 75-40). Erythematous papules and petechiae develop at the site of the infestation. Although the vast majority of the lesions occur on the foot or lower extremity, they may occur anywhere on the body. Some larvae remain quiescent for a few weeks or months, and then migration is manifested by a wandering, thin, linear, raised, and tunnel-like lesion 2 to 3 mm in width. Older lesions become dry and crusted. Nonspecific symptoms include pruritus, urticaria, and maculopapular exanthem. A case of cutaneous larva migrans complicated by a systemic inflammatory disorder (erythema multiforme) has been documented.[279] The larvae travel a few millimeters to a few centimeters each day. Larva migrans may be associated with eosinophilia (in 10% to 35% of cases) and Löeffler's syndrome.

Larva currens is a form of cutaneous larva migrans caused by *Strongyloides stercoralis*. Usually, this eruption is associated with intestinal strongyloidiasis and initially presents in the perianal skin. It may involve the buttocks, thighs, back, and shoulders, but the genitalia are spared. Pruritus is intense, and the lesions spread rapidly, advancing 5 to 10 cm each hour. The larvae leave the skin, enter the bloodstream, and later settle in the intestinal mucosa, whereupon the cutaneous manifestations fade.

Treatment

Therapy for cutaneous larva migrans includes cryotherapy (application of liquid nitrogen spray and carbon dioxide with ethyl chloride spray), topical 10% thiabendazole (two 0.5-gm tablets crushed and mixed with 10 gm of petrolatum applied twice daily until clear), and systemic administration of oral thiabendazole (25 to 50 mg/kg) for 2 to 4 days, albendazole (400 mg daily) for 7 days, or ivermectin (12 mg) as a single dose.[28,50,174,224,226,278] Oral albendazole is an effective treatment, with fewer side effects, recurrences, and failures.[145,205] Secondary infection sometimes occurs and may require incision and drainage of pustules or furuncles and the use of topical and systemic antibiotics, depending on the bacterium cultured. Without treatment, the larvae spontaneously die within a few weeks and dermatitis clears.

Prevention

In endemic areas, precautions include not sitting or lying on damp soil or sand, especially during rainy seasons; not walking barefoot or using water shoes with fenestrated fabric[267]; draping the ground with impenetrable material before crawling in the dirt; and covering sandboxes with a tarp to prevent contamination by prowling cats.

Leeches

Definition

Leeches are members of the phylum Annelida, class Hirudinea. These segmented worms are found in freshwater and marine environments. Their biting jaws allow them to attach to the unsuspecting victim's skin. The freshwater leech may attach without causing significant pain, whereas the marine leech may produce a significant sting upon attachment.[182]

Pathophysiology

Upon attachment of the leech, anticoagulants (hirudin and fibrinase) are released into local tissues, which cause moderate, painless bleeding at the site after leech removal. A substance that has local analgesic qualities also may be released.[105,247] The release of such a substance could account for the absence of significant pain during freshwater leech attachment. Leeches feed on their host until engorged, at which time they detach.

Signs and Symptoms

Signs and symptoms of leech attachment include local pain, redness, swelling, pruritus, and evidence of puncture wounds

(caused by the leech's fangs).[182] The wounds may bleed for a prolonged time, heal slowly in unsensitized individuals, and are inclined to become secondarily infected by bacteria. Previous sensitization to leeches may produce a more severe reaction. Leech bites can cause serious systemic allergic reactions, including anaphylaxis.[124] The wound may become necrotic, urticarial, or bullous.

Treatment

Leeches must be removed from the skin carefully. If a leech is ripped from the skin, its fangs may break off and remain embedded, which induces ulcer formation. Leech removal is facilitated by application of a few drops of alcohol, vinegar, or brine. An alternative removal method involves the application of a lit match to the leech at the attachment site.[84] After leech removal, the site should be inspected carefully to ensure that the fangs are absent. Hemostasis can be achieved by applying direct pressure and using a styptic pencil (a short medicated stick, often of alum, applied to a cut to stop bleeding), oxidized regenerated cellulose absorbable hemostat, or topical thrombin solution. The wound should be cleansed thoroughly with an antiseptic agent several times daily to prevent infection.

Prevention

To prevent leech attachment, avoid wading through waters infested with leeches. Individuals who must enter these waters should wear protective clothing and cover all exposed skin with a body suit or wetsuit.

Parasitic Sea Lice (Cymothroidism)

Sea lice (cymothroids) are small marine crustaceans of the order Isopoda, suborder Cymothoidra. Water skiers, skin divers, and swimmers who frequent coastal and estuarine waters of temperate and tropical seas may encounter these free-swimming crustaceans.[25] Sea lice may live as parasites on invertebrates and fish. Sea lice bites are commonly reported along the coast of southern California. Cymothroids are usually buried in sandy bottoms below water level, but they attack any creature that encroaches on their space.[68] They typically attack marine animals but also attack humans. They are equipped with a biting apparatus that readily allows them to attach to fish or to human skin.[25] The bite is rapid and sharp, causing punctate hemorrhagic wounds (Fig. 75-41) referred to as sea lice dermatitis (cymothroidism).

Treatment

The wounds should be cleansed initially with hydrogen peroxide or a brisk soap and water scrub. A topical antiseptic ointment (neomycin, mupirocin, or bacitracin) should be applied, and the wound should be inspected daily for secondary infection. The injury resolves over 5 to 7 days with appropriate care.

Crab Larvae

An unusual sensation and irritation described as "pine needles sticking in the skin" is reported when crab larvae contact exposed skin.[44] This sensation resolves upon removal of the larvae. The larvae, found floating on the water's surface, are described as floating sand particles or small, gray seeds. These larvae have small, translucent gray bodies with no glandular structures. Calcified spines induce traumatic injury on contact with skin. Contact does not produce urticaria or any other form of dermatitis.

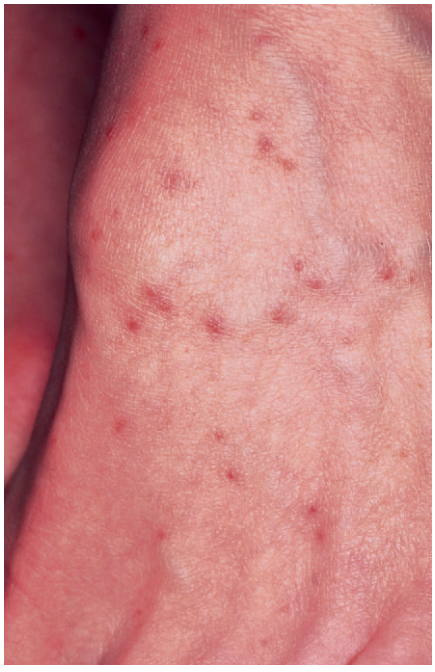


Figure 75-41. Sea lice. Pinpoint purpuric lesions typical of Cymothroidae (sea lice) bites or punctures. (Courtesy of Edgar Maeyens, Jr., MD.)

DIVING-RELATED DERMATOSES

Diver's Hand

The condition is described as extensive desquamation of the palms and occasionally the soles.[4] Diver's hand affects most long-term occupational saturation divers at one time or another. Victims complain that their skin splits and peels off in large flakes. Peeling starts at the fingers and progresses proximally. There are no vesicles, papules, or hyperkeratosis. Secondary infection rarely occurs. The dermatitis continues for 1 to 2 weeks when out of water. The condition remains painful for 2 to 4 weeks until the epidermis regenerates and fissures resolve.

Diver's hand may occur in divers without any previous skin problems. Preexisting dermatitis is not a prerequisite for the development of diver's hand; this condition occurs as readily in normal skin. Biopsies of affected skin reveal formation of horizontal clefts in the upper two thirds of the epidermis. Absence of an inflammatory infiltrate and infectious organisms indicates that physical or chemical factors are the etiologic agents. Diver's hand is theorized to develop in all phases of saturation (compression, bottom time, and decompression during a dive), as well as after the end of saturation. There is no effective protection or treatment available.[3,4]

Mask Squeeze

Divers may experience bilateral subconjunctival hemorrhages and may sustain pressure marks in the shape of the diving mask if they are unable to equalize pressure within the face mask during descent (see Figure 25-19).

Nitrogen Dermatitis

If scuba divers linger longer than the recommended time at depth, oversaturation of nitrogen under pressure into the sub-

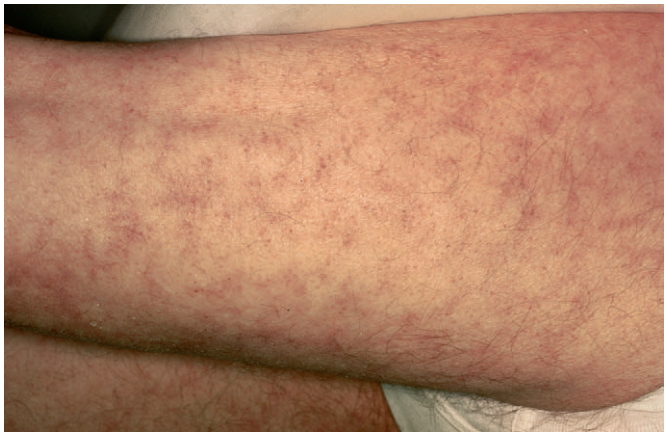


Figure 75-42. Marbling of the thigh in cutaneous decompression sickness. (Courtesy of Edgar Maeyens, Jr., MD.)

Figure 75-43. Blotchy erythema of the breast in cutaneous decompression sickness. (Courtesy of Edgar Maeyens, Jr., MD.)

cutaneous tissue can rarely cause transient dermatitis. Nitrogen dermatitis is manifested by pruritic, tender, and erythematous lesions involving primarily the flanks and elbows.[182] Diving should be discontinued until all symptoms and signs resolve.

Cutaneous Decompression Sickness

Definition

Cutaneous decompression sickness is a spectrum of clinical signs and symptoms, which are the result of neural stimulation via release of chemical mediators, neurotransmitters, and stimulation of mechanoreceptors through expanding bubbles plus the alteration of blood flow. Thermal cooling is a contributory factor. It is defined as a disorder of the skin subsequent to reduction of ambient pressure resulting in the formation of bubbles from dissolved gas in tissues or blood.[95] Characteristically, this process is accompanied by itching, pain, and various "rashes." Excessive skin gas pressure over ambient pressure (nitrogen supersaturation) is critical in development of bubbles occurring during or after ascent from depth. Cutaneous decompression sickness is the consequence of this bubble formation.[95] Once bubbles form, they may initiate the release of mediators of inflammation, obstruct vascular or lymphatic vessels, or stimulate directly or indirectly the release of neurotransmitters. Cutaneous decompression sickness can occur as an isolated occurrence or in conjunction with other organ system decompression sickness or decompression illness with associated arterial gas embolism.

Skin, like any other organ system, is vulnerable to the effects of expanding gases during decompression. Depending on the location of the bubbles, the cutaneous manifestations will vary. For example, when bubbles are present in the larger vessels of the deep dermis or subcutaneous tissue, the clinical presentation is that of marbling (Fig. 75-42). If the capillaries or venules of the superficial plexus are affected, the skin develops a blotchy erythema (Fig. 75-43). When lymphatics are occluded by bubbles, edema results.[34]

Pathophysiology

The precise pathophysiology of cutaneous decompression sickness remains unclear. Pruritus is a common symptom. Itching and pain share common neural pathways, but are distinct sensory modalities. The sensation of itch is produced primarily near the dermal-epidermal junction by stimulation of free nerve endings of the unmyelinated C-fiber-type.[68,175]

C-fibers comprise 70% of the primary afferent neurons and are categorized into three classes:

1. C-mechanoreceptors
2. C-polymodal nociceptors
3. Cold thermoreceptors

Eighty percent of the C-fiber afferents are C-polymodal nociceptors. These nerves in the dermis respond to thermal, electrical, mechanical, and chemical stimuli. The sensation of itch is conveyed by C-polymodal nociceptors.[175,184]

In the skin, there are chemical mediators (histamine) and neurotransmitters (neuropeptides) responsible for the itch sensation.[175] Neuropeptides are produced primarily by cutaneous sensory nerves. Examples of these include substance P, endopeptidases (e.g., kallikrein, bradykinin), prostaglandins, and serotonergic compounds.[5,184,256] On exposure to one or more of these transmitters, the C-polymodal nociceptor is activated and the itch sensation occurs. Histamine is stored in dermal mast cells that surround substance P–secreting cells and are adjacent to microvascular endothelial cells.[13] It is probable that expanding gases in cutaneous decompression sickness stimulate neurotransmitter release or alter mast cell membranes, thereby releasing histamine.

Histamine has numerous functions, all of which can affect cutaneous decompression illness. Through its effects on vascular smooth muscle and endothelial cells, histamine allows plasma protein and fluid to escape into the extracellular spaces.[13] Combined with histamine-induced increase in lymphatic flow and lymph protein content, this results in formation of edema. Wherever there are large numbers of mast cells, such as in skin, intestinal mucosa, and bronchial mucosa, histamine content is high.

Substance P is a neuropeptide synthesized in the dorsal root ganglia within the cell bodies of the polymodal nociceptor C-fibers. It induces vasodilation, immune cell activation, leukocyte infiltration, and cytokine secretion.[9] An activated C-nociceptor in the skin signals afferent transmission to the spinal cord. In response, antidromic (neuronal cross-talk) signals are sent via efferent neurons to the skin, triggering

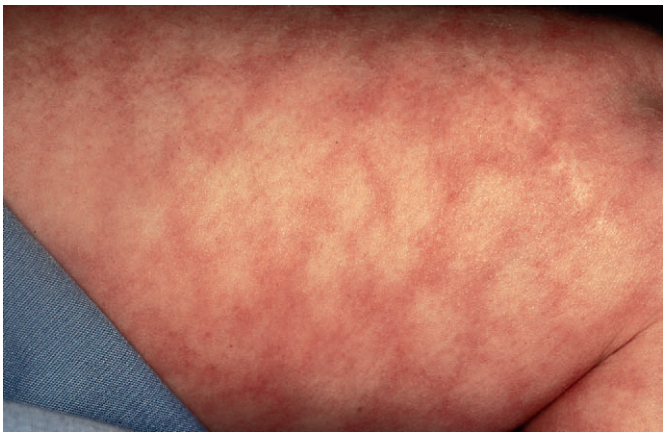


Figure 75-44. Cutis marmorata. (Courtesy of Edgar Maeyens, Jr., MD.)

Figure 75-45. Cutis marmorata. (Courtesy of Edgar Maeyens, Jr., MD.)

further histamine release and prolongation or accentuation of itch.[258] For the sensation of itch to occur, C-nociceptors must be stimulated by chemical mediators or neurotransmitters.

It is likely that expanding bubbles in the dermis stimulate mechanoreceptors by distortion and, thereby, produce pain. With recompression, symptoms usually promptly abate. In addition, histamine, when present in the dermis, can produce pain and itch.

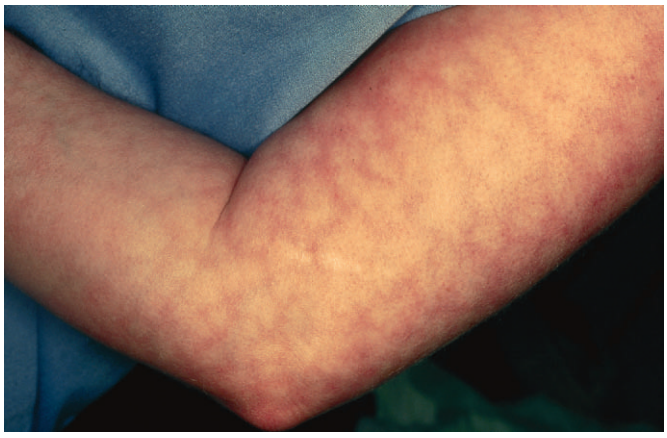


Figure 75-46. Cutis marmorata. (Courtesy of Edgar Maeyens, Jr., MD.)

The term "cutis marmorata" is often used erroneously to describe cutaneous decompression sickness. True cutis marmorata presents as a blue reticulated (marbled) vascular pattern occurring when skin is cooled (Figs. 75-44, 75-45, and 75-46). It is most pronounced in areas of greatest fat deposition because fat insulates the skin surface from the central warmer core of the body. This condition is the result of slowing blood flow in the capillary/venous plexus. Other contributing factors are arterial narrowing, vasoconstriction, and increased blood viscosity.[227] This is in contradistinction to cutaneous decompression sickness, which is almost universally an erythematous, blotchy condition. The erythema is the result of vasodilatation and vessel engorgement.[45]

Buttolph and colleagues, using a swine model to study cutaneous decompression sickness, were able to demonstrate vascular congestion as the most common finding.[45] They also described neutrophil adhesion to vessel walls and vascular occlusion. They demonstrated by electron microscopy a perturbation of either endothelium or neutrophils or both. These authors concluded that "the marbling or cutis marmorata forms of cutaneous lesions in cutaneous decompression sickness are principally vascular congestion, possibly as a result of inflammation."[45]

The references for this chapter can be found on the accompanying DVD-ROM.

Safety and Survival at Sea

76

Michael E. Jacobs and Charles G. Hawley

It is difficult for us to grasp the idea that parts of our planet remain in an almost primordial state of wildness and isolation. There are only a few places left on earth where merely getting across them is an achievement: Antarctica . . . the Sahara . . . the Southern Ocean . . . the wilderness of ice or sand or water or terrible places where nature retains power over humans to terrify and diminish.

—Derek Lundy, Godforsaken Sea

The greatest wilderness on earth is the sea. It covers two thirds of the planet and, with the exception of the sun, has the greatest influence on global weather patterns. Although it may take hours or days to succumb in other environments, death at sea can happen in less than a minute. Compared with desert heat, high-altitude hypoxia, and polar subzero temperatures, water is the most hostile and life-threatening natural environment for inadequately equipped survivors.

The 1979 Fastnet Race distinguished itself as the worst disaster in the 100-year history of ocean yacht racing. When a killer storm approached the Irish Sea between southwest England and southern Ireland, it exploded without warning in the midst of the Fastnet racing fleet. Suddenly, 2700 men and women in 303 ocean sailing yachts unwittingly became participants in a study of survival at sea. Winds of 55 knots and seas of 50 ft (15 m) knocked down one third of the fleet and left 26 yachts capsized. Despite a massive response of personnel and equipment, 24 ships were abandoned and five of those sank. Fifteen sailors drowned and 136 were rescued from disabled yachts. The official Fastnet Race Inquiry noted, "The common link among all fifteen deaths was the violence of the sea, an unremitting danger faced by all who sail." It concluded: "The sea showed that it can be a deadly enemy and that those who go to sea for pleasure must do so in the full knowledge that they may encounter dangers of the highest order."[2]

Twenty years later, on the edge of the Southern Ocean, the Sydney to Hobart race between the southeast coast of Australia and the island of Tasmania became a terrifying ordeal for 115 yachts. Within a day of leaving Sidney harbor, an explosive low-pressure cell, a "southerly buster," formed over the fleet as it entered Bass Strait. In its aftermath, six men died, 55 were rescued, and 12 boats either sank or were abandoned. One hundred participants were seriously injured and five drowned. Fractured ribs, lacerations, and head trauma were the most common injuries. The 330-page investigation report recommended strict guidelines for improved safety gear, life rafts, and telecommunication equipment. For the first time, attendance at Safety at Sea conferences became mandatory.

Mastering seamanship and survival skills requires training and experience. The ultimate challenge for every mariner is to confront and handle the fears that frequently render a person helpless in a survival situation. Survival depends on philosophical, psychological, and physical preparation in addition to good seamanship. This chapter discusses some of the basics for survival at sea.

EMERGENCIES AT SEA

The Decision to Abandon Ship

Flooding, fire, or collision generally causes sinking. Only when the vessel is about to sink or is burning out of control should the crew abandon ship. The adage "always step up to the life raft" means it is recommended to abandon the vessel no sooner than when the decks are awash. Consider all options; do not abandon the ship before it abandons you! Abandoning ship prematurely puts the crew at greater risk than remaining with a disabled craft.

With rare exception, a floating disabled vessel is always your best lifeboat. The mother ship provides a stronger and more visible rescue platform than a life raft. It is better stocked with provisions and equipment for communication and survival; conditions will always be harsher in a life raft.

Flooding

Flooding is the most common disaster at sea. It may result from failure of systems or materials or structural damage from collision and extreme weather (Box 76-1). Before abandoning ship, quickly assess the damage. The limiting factor is time. Stock the proper tools and repair supplies in a damage control kit (Box 76-2), and know how to respond quickly and skillfully.

Early detection of flooding is crucial for an effective response. Make frequent visual inspections of the bilge, engine room, galley, and head while underway, and maintain watertight integrity at all times. The most reliable way to keep a boat afloat is to keep the water on the outside. Hatches for the main companion way, engine room, lazarettes, cockpit lockers, fish holds, and elsewhere require gaskets and proper dogging (locking) devices to ensure a watertight seal; secure them while underway. After a knockdown or wave breaking over the cockpit, water may go straight below decks if the companionway drop boards are not in place. Severe flooding, with damage to the electronics, navigation station, and engine, usually results. These boards require robust dead bolts capable of locking from both sides.

Box 76-1. Sources of Flooding

1. Failure of through-hull fittings involving the following systems: head, galley, wash basins, shower sump, bilge pumps, centerboard pins and cables, engine exhaust, engine cooling, deck and cockpit drains, bait box, drain plugs (for small boats), knot meter sensor, depth sounder sensor, propeller shaft stuffing box, shaft struts, shaft log, rudder post
2. Failure of hose connections, clamps, pipes, and fittings
3. Open hatches, portholes, and ventilators
4. Siphoning of seawater back into the boat because of poor system design or failure of a check valve
5. Collision with large floating or submerged objects
6. Structural failure in hull, deck, or rigging
7. Punctured hull from overboard broken spars (e.g., dismasting)
8. Ruptured on-board water tanks (a boat will not sink from leaking internal water tanks)
9. Loosened or broken keel bolts
10. Clogged scuppers or cockpit drains

Box 76-2. Damage Control Kit

Assorted hose clamps
Extra through-hull conical wooden plugs and hammer or mallet to bang them home
3M 5200 or equivalent sealant/glue (e.g., G-5 sealant tape)
Caulking gun, extra cartridges
Collision mat with 15-ft (4.5-m) lines in corners or old storm sail with lines rigged at the head, tack, and clew
Water-activated fiberglass repair fabric (Syntho-Glass)
Pruning saw with spare blades
Bolt and rigging cutters
Drifts matching size of clevis pins
Duct tape, electrical tape, and self-amalgamating tape
Polyvinyl chloride (PVC) cloth from old foul weather suits, neoprene from old wet suits
Self-tapping stainless steel screws, threaded rod, bolts, nuts, and large washers
Large screwdriver, pry bar, and hatchet
Shoring materials and wood wedges
Vise-grip pliers
Premade square plywood patches (e.g., 2 to 3 ft), bendable, 3/16″ five-ply birch, with predrilled holes at 4-inch (10-cm) intervals set about an inch (2.5 cm) in from the edge)
Hand drill or cordless drill, and bits
Extra canvas buckets
Extra dry chemical fire extinguishers
Portable VHF emergency ship antenna
Band-it tool, SS bands, box of buckles
Emergency single-sideband (SSB) coaxial antenna

Discharge pipes require seacocks. Regularly inspect through-hull fittings, engine drive shaft stuffing box, clamps and hoses. Avoid using polyvinyl chloride (PVC) or any other domestic plastic plumbing fittings for through-hull fittings below the waterline; they can easily break off or fracture if struck by shifting stores. The preferred materials are Marelon (reinforced plastic) or bronze, together with stainless steel hose clamps. Post a diagram in the cabin showing the locations of all through-hull fittings and the routes of connecting hoses. Keep seacocks accessible and unobstructed and be able to find them blindfolded. Install U-shaped antisiphon valves above the highest waterline (it changes as the boat heels). Without these valves, water can siphon back through the hose and eventually into the bilge.

Figure 76-1. Large vessels may elect to have substantial emergency pumps onboard, which can keep up with flooding from modest hull breaches. Note also the crew overboard device on the pushpit and the throw rope.

Reliable bilge pumps are the best defense against flooding (Fig. 76-1). An excellent pump can buy time for locating and plugging the leak. If a boat is equipped with an automatic bilge pump (or pumps), it is a good idea to install a cycle counter on the pump and an "on" light to alert the crew when the pump is activated. A second emergency pump mounted above the first, using a separate float switch, can provide added pumping capability if the first pump cannot keep ahead of the leak. In this case, a loud horn installed in the circuit can alert the crew to flooding.

Keep the bilge clean and free of debris to avoid clogging the pump strainer. Perform regular inspection and maintenance of the entire pumping system. Aluminum-body bilge pumps corrode from the inside out, especially while retaining saltwater. They may appear to be in perfect condition, yet be completely useless. Hoses crack with age, rubber components become dry and brittle, valves jam, and moving parts deteriorate through wear and corrosion. To guarantee reliability, disassemble, inspect, and clean manual bilge pumps annually. Bilge pump handles should be easily accessible and secured with a lanyard to avoid loss after a knockdown or rollover. Offshore boats require at least two manual bilge pumps, one operable from the helm station and one below decks. Know the capacities of the boat's compartments and have a means to pump out any that flood. Test the pump's capabilities by intentionally flooding the bilges (preferably with fresh water), and monitor how long it takes to pump them out.

If flooding occurs, the following actions are recommended:

1. Alert the crew, don life jackets, and (in cold water) prepare immersion suits.

2. Switch on electric bilge pumps and run engine-driven pumps.
3. Radio a mayday (distress message).

Sending a mayday early is important, because the electrical system may short-circuit from water rising in the cabin. The call for assistance can be cancelled later if the vessel is saved.

The next four tasks should be to:

1. Locate the source of flooding.
2. Ascertain the type and extent of damage.
3. Stop or reduce the inflow temporarily.
4. Repair the damage.

One expert suggests walking around your boat when it is on the jack stands in the boatyard and tapping on the hull. Ask yourself where a puncture at that spot would flow to a corresponding area on the inside. Having that picture in mind might save your vessel and your life.

The volume of water rushing in depends both on the size of the breach and its depth below the waterline. A 1-inch (2.5-cm) diameter hose disconnected from a sink drain, usually 1 ft (30 cm) below the waterline, allows 20 gallons per minute (gpm) of seawater into the cabin. A typical 1-inch diameter open seacock just 2 ft below the waterline admits 62 gpm, and if located 4 ft below, 87 gpm. If the propeller shaft is lost, the 1.5-inch hole left 3 ft below the waterline will admit 77 gpm or 4620 gph.

A boat equipped with the largest manual double-action twin-diaphragm bilge pump can pump a maximum of 42 gpm, a limit easily surpassed by the examples given. It is therefore critically important to locate and stop the leak, rather than fight what may be a losing battle by pumping to prevent sinking. For a variety of reasons, electrical bilge pumps rarely operate at their rated capacity, and none can keep ahead of the common sources listed above. A filled bucket holds 3 gallons (11.4 L) and weighs about 20 lb (9 kg). A bucket brigade may be effective for a limited time until the source is located or the crew fatigues.

As the water rises in the cabin, a leak becomes more difficult to locate. The inflow rate decreases as the depth of water in the boat increases, because the pressure gradient is reduced. At a critical level of flooding, the inflow rate may slow sufficiently to allow pumps to handle the volume, so do not stop pumping. A point of zero net flooding may be reached from the inherent buoyancy of the boat as it settles in the water (another reason the ship should not be abandoned unless it continues to flood). Many small wood and fiberglass boats float when fully flooded or swamped. Boats less than 20 ft (6 m) in length constructed in the United States after July 1972 are required to have sufficient built-in flotation to remain afloat when swamped. Small boats (e.g., daysailers, small open motorboats) can obtain additional buoyancy by lashing down unused life jackets, cushions, and fenders to increase flotation.

A tapered, soft, dry wood plug sized to fit a leaking through-hull fitting can serve indefinitely as an adequate seal; it will swell to seal the fitting or any small puncture in the hull. Take the plugs out of the damage control kit and attach them to their respective through-hull fittings with lanyards; this will make them instantly available and help the crew find them in the dark.

Large holes can be overlaid with a collision mat (Fig. 76-2) placed outside the hull to supplement a temporary interior patch (see below). The mat is a piece of heavy canvas or vinyl-coated fabric with grommets and lines that enable it to be positioned and secured on the exterior surface. It is held in place by pressure and lines. Collision mats can be purchased or improvised

Figure 76-2. Triangular patches made from coated fabrics can be held in place with lines attached to each corner.

by using a small sail or awning material, although it is a mistake to make the mat too large; generally 4 ft (1.2 m) on an edge of the triangular shape is adequate. An umbrella-like device called a Subrella can be pushed through the damaged area from the inside and then opened on the outside of the hull by pulling it sharply back. The water pressure automatically spreads the patch over the hole to form an effective seal and holds it in place. A commercial hull repair kit is available and features flexible oval concave sheet metal plates with rubber gaskets. A bolt is welded to the intended exterior piece. The idea is to slip one oval through the hull breach, then back it from the inside with the second metal plate and tighten both together with a thumbscrew.

Lacking special equipment, any soft, pliable material, such as a life jacket, mattress, blanket, towel, cushion, clothing, or foam pad, can be used to slow water rushing through a jagged break in the hull. When placed against the exterior hull, the suction effect created by flow and hydrostatic pressure will generally provide some clamping pressure to the plug or patch. If a plug is positioned from inside the cabin, shore it with a board and brace it with a pole (e.g., oar, mop handle, boat hook, whisker pole, strut, bunk rail).

Underwater patching compounds can be used to bond a solid plate over the hole or to impregnate an expandable material or packing to serve as plugging material. There is great variability in their cure speed, ease of mixing, mixed viscosity, and adhesiveness. Some products work only on specific hull materials (e.g., wood, fiberglass, or aluminum). A complete review and test of these compounds can be found in the December 2001 issue of *Practical Sailor* magazine (vol. 27).[6]

Supplement repairs with other measures to help slow the inflow. Heel the boat away from the area of damage to decrease hydrostatic water pressure. This is easy to accomplish under sail, but for powerboats, shift heavy items to the side opposite the leak and slow forward speed if water is entering a hole in the bow.

When ingenuity and improvisation fail to stop flooding, the U.S. Coast Guard can supply a portable gasoline-powered dewatering pump to assist a sinking vessel. The unit is simple to operate and comes in a waterproof barrel with hoses, gasoline, and illustrated operating instructions. When dropped from

an aircraft into the sea, a retrieving line will be also dropped to the deck crew. It takes two people to lift the barrel from the sea onto the deck. The standard CG P-1B dewatering pump can pump 120 gpm at 10 ft lift, and is capable of running 4 to 5 hours on one tank of gas. The P-1B pump, known as the "drop pump," is likely to be carried by helicopter and by 22-ft rigid hull inflatable Coast Guard rescue craft. A larger pump, the CG P-6 (classified as a dewatering/fire fighting pump) is carried by Coast Guard search and rescue ships and is placed either onboard or passed via lines, depending on weather conditions. Pump capacity is 250 gpm at 12 ft lift; it runs 4 to 5 hours on a tank of gas. Both pumps are dramatically more effective than even the most robust manual or electric bilge pump. However, 250 gpm is equivalent to the inflow of water from a 3-inch hole (a relatively small breach) only 2 ft below the waterline; any larger hole at this depth will exceed the capacity of even the P-6. Remember to run either pump outside, not in an enclosed space where carbon monoxide may accumulate. Refuel them only after the engine is stopped.

Some oceangoing cruising vessels are built with watertight compartments to confine flooding to a limited area. Maintain watertight bulkheads watertight! Know the location of watertight doors and how to operate them.

If flooding and sinking are inevitable, the captain should consider running the boat onto shore.

Fire

Uncontrolled fire is a disaster aboard ship. Fires aboard wood and fiberglass boats have the potential to double in size every 10 seconds. Approximately 7500 pleasure boat fires and explosions occur annually; of these 10% are declared total losses. More than half of the 2700 fire-related injuries incurred each year occur on small, open motor boats.

Ninety percent of fires start in the engine compartment, commonly from an overheated engine, turbocharger, or exhaust manifold; leakage of fuel, hydraulic fluid, or lube oil provides the fuel. Faulty electrical wiring and short circuits in the power panel or battery compartment are also frequent causes, as are the ship's batteries and galley stove. Lightning is a major cause of boat fires at marinas in Florida. Box 76-3 lists ways to prevent fires aboard ship.

The explosive potential of fuel depends on its chemical properties and where the vapors accumulate in enclosed, unventilated spaces. Hazardous liquids are classified according to flash point, the lowest temperature at which a liquid releases enough vapor to sustain burning. Flammable liquids, such as gasoline, turpentine, lacquer thinner, and acetone, have flash points below 38° C (100.4° F), meaning they release enough vapor at common ambient temperatures to form burnable and explosive mixtures. Combustible liquids, such as diesel oil, kerosene, and hydraulic fluid, have flash points above 38° C (100.4° F).

Gasoline is the most hazardous fuel. Vapors can ignite from the heat in the engine compartment and from liquid spills over hot engine parts. One teaspoon (5 mL) of gasoline can vaporize and cause an explosion, and 1 cup (237 mL) of gasoline has the explosive potential equal to several sticks of dynamite. Vaporized gasoline is heavier than air, so it accumulates in the lowest part of any enclosed space. Diesel fuel is much less explosive. However, pressurized diesel fuel spurting from a burst fuel line will ignite and burn when it strikes something hot, such as the exhaust manifold. Charging batteries generates hydrogen, which accumulates in the battery compartment or in the compartment overhead; the gas is lighter than air, highly flammable, and potentially explosive. Sparks from a nearby electric motor may set off an explosion from excess hydrogen produced by overcharging.

The best protection against fire is to install a properly sized, automatic discharging extinguisher system, which interrupts the chemical process of combustion with chemical materials or gases. Fire-suppressing alternatives to halon gas (which is now banned because it breaks down atmospheric ozone) are fluoroethane and fluoropropane (called FM 200 and FE-241, respectively). These fire suppressants can be automatically dis-

Box 76-3. Fire Prevention at Sea

1. Sniff the engine compartment before starting a gasoline engine and before starting the electric bilge blower.
2. Ventilate the bilge; run the bilge blower for at least 4 minutes before starting a gas engine.
3. Use appropriately sized marine-grade wire. Periodically inspect wiring for cracks, charring, and deteriorating insulation. Wiring connectors and terminals should be tightly set and periodically inspected for looseness. Electrical systems should have fuses or circuit breakers. Check electric motors for sparks.
4. Ventilate the battery compartment; never create sparks around a battery until the surrounding space has been ventilated. Inspect and tighten battery cable connections. Never smoke around a battery.
5. Install a lightning ground.
6. Observe all precautions when taking on fuel. Close all hatches and ports before filling the tanks, and extinguish all flames.
7. Never leave the stove unattended while in use.
8. Store extra fuel on deck in approved plastic containers, or in a locker that is sealed from the main cabin and drains overboard.
9. Store outboard motor and its fuel tank on deck.
10. Transfer gasoline and other flammable liquids from one container to another on deck or off the boat.
11. Inboard engines require meticulous inspection and maintenance. Perform a routine hourly visual inspection of any operating engine and the exhaust system. Keep seawater strainers for the engine cooling system clean. Become an expert in examining and repairing all components of the fuel system.
12. Install a heat-activated automatic fire-extinguishing system in the engine compartment, together with smoke and flame detectors.
13. Properly store fuels, solvents, paints, brushes, and combustibles in a deck storage locker. Keep all rags in metal containers with the lid tightly closed. Do not keep oily rags below decks or in the engine compartment.
14. Place fire extinguishers away from the intended area of use so that they are accessible, and have them inspected and maintained annually.
15. Read the extinguishers' instructions periodically, and practice on a controlled fire away from the boat.

charged from extinguishers by devices that sense ultraviolet radiation or temperatures above 79°C (175°F). Automatic systems should also have a manual trigger for activation. Halotron is a new "no residue" agent for portable extinguishers and a good alternative to the older dry chemical powders, which leave a messy, highly corrosive, and destructive residue. When using portable extinguishers, discharge them into the engine compartment through small fire ports to avoid introducing large quantities of fresh air to the fire. Remember to have the engine shut down either automatically or manually when fighting fire in the compartment. Let the area cool before opening hatches or inspection ports, lest the entry of air start the fire anew by diluting the concentration of extinguishing agent or by introducing oxygen.

A popular galley stove fuel is liquefied petroleum gas (LPG), either propane or butane. Both are highly explosive, heavier than air, and may accumulate in the bilge. Free propane in the bilge, like gasoline, is a bomb waiting to go off. An ignition-protected independent bilge blower can safely remove LPG. Proper LPG tank installation requires a completely self-contained vapor-tight locker opening only above decks. The drain should be located at least 20 inches (51 cm) from any opening to the boat's interior and should not be submerged while the boat is underway. A pressure gauge connected to the LPG cylinder valve will help to indicate a leak somewhere in the system. It is not, as many believe, intended to show the level of gas in the tank. A regulator to reduce the pressure in the gas line to the stove and an electric solenoid valve complete the delivery system. Seek expert professional assistance when installing the complete fuel supply system. As of April 2002, propane cylinders must be equipped with an overfill protection valve, because overfilled cylinders may explode after overheating.

Leaks in the line can be detected with an electronic gas detector installed in the bilge and beneath the stove. If the alarm sounds, turn on the blower and run it until gas is no longer detected by smell or by an electronic sensor. The system should be pressure-tested for leaks periodically, especially after rough weather.

To check for a leak, open the cylinder valve with the solenoid switched on and all appliance valves closed, and record the pressure gauge reading. Next, close only the cylinder hand valve to see if the pressure drops over intervals of 3 to 5 minutes; if it does, there is a leak.

To avoid accidents after cooking, switch the solenoid off first, let the burner continue to flame until the line is cleared of gas, and then turn off the burner. When appliances are not in use or the boat is unattended, close the cylinder valve. Never use the stove as a cabin heater; the flame can deplete a cabin of oxygen and asphyxiate the sleeping crew. Carbon monoxide, a colorless, odorless gas, is a byproduct of incomplete combustion; severe exposure can be lethal. Mild early symptoms consist of fatigue, sleepiness, headache, malaise, nausea, vomiting, and ataxia (loss of balance); these are also the symptoms of seasickness, and can lead to a fatal misdiagnosis!

Stove alcohol is also hazardous, especially if one burner is accidentally extinguished and liquid alcohol used in priming the burner pours onto an adjacent flaming burner, causing a flare-up. A nonpressurized burner can also reignite if refilled with alcohol while still hot. Alcohol fires can be extinguished with fire blankets or wet towels. A grease fire on the stove can be extinguished with these items or by liberally sprinkling the fire with baking soda. A good precaution is to place a kettle of water on the burner before lighting it; this helps to contain any high flames arising from the excess alcohol used in priming the burner.

Figure 76-3. Dry chemical fire extinguishers should be inspected to ensure the pressure gauge indicates a full charge and should be inverted regularly to unpack the extinguishing agent.

The Coast Guard requires all recreational vessels to have portable fire extinguishers (Fig. 76-3). The standard multipurpose dry chemical extinguisher (filled with ammonium phosphate) and the newer dry chemicals discussed previously can be used on all types of fires, class A, B, or C. They extinguish and suffocate the fire by preventing access to oxygen. A major drawback is the extremely brief window of opportunity provided to put out the fire. The common B-1 extinguisher discharges completely in just 10 to 13 seconds. Units with greater capacity simply deliver more chemical over the same period. Because of the short discharge time, more than one fire extinguisher should be available to cope with a large blaze.

The thick cloud of dust coming from the extinguisher impairs visibility. Aiming the extinguisher becomes more a function of memory than of visibility. Direct the nozzle to the base of the fire with a sweeping, side-to-side motion. Fires extinguished with dry chemicals should be considered a hazard until they cool down to room temperature.

Dedicate one portable extinguisher to the engine compartment, a second to the galley, a third to the area under the fore hatch, and place the fourth (and largest size practical to stow) in a cockpit locker. One should never have to walk more than half the boat's length to reach an extinguisher. The ideal location for a portable extinguisher in a closed compartment is next to the exit door. Fires involving common combustible solid material can be brought under control by cooling them with large amounts of water. Fires involving flammable or combustible liquids can be smothered to remove the oxygen by using a fire blanket. Electrical fires, where a short circuit is generating sufficient heat to cause other materials to combust, can often be controlled by cutting the electric power. Water does not completely extinguish electrical fires (Box 76-4).

Collisions

Collisions were responsible for approximately 40% of all recreational boating accidents reported to the U.S. Coast Guard in

Box 76-4. Guidelines for Fighting Fire at Sea

1. Attack the fire immediately at the source. Detection and reaction time must be within a few minutes, before the fire burns out of control. Prepare a plan that has been shared with the crew so that everyone knows the location of equipment and their responsibilities.
2. Initiate a mayday call immediately. The purpose is to alert ships in the area that the crew may have to abandon ship.
3. When fire is discovered, all crew should be on deck as quickly as possible with life jackets and fire extinguishers in hands. Ensure that the life raft is away from the fire.
4. Slow the boat to reduce relative wind, and steer to keep smoke and flames clear of the crew and vessel. Keep the fire on the downwind side of the ship, exposing the smallest amount of the boat's structure to the flames.
5. Position personnel between the fire and a clear escape route.
6. Cut off the source(s) of the fire (e.g., fuel supply, electric current, ventilation system). Turn off blowers, and stop the engine immediately.
7. Shut off the air supply to the fire. Close the hatches, doors, and vents to all compartments free of people.
8. If you must open a hatch to discharge a portable extinguisher, beware of burning your hands or face. As fresh air enters the compartment, the fire will rise to the air source and flare up. The safest way to open a hatch is to wear gloves and stand on the hinged side of the hatch while pulling it open.
9. If the fire is too large or out of control, abandon ship before the fuel tanks explode.
10. Be alert for signs of overheating when sailing with the engine running, especially when the boat is heeled.
11. Check the engine compartment frequently and take action if you smell smoke or burning wire.

2005.[8] Only capsizing and crew overboard cause more fatalities.[8] Collision with another boat or with a fixed or floating object is responsible for the greatest number of nonfatal injuries and accounts for more than three times as many injuries as any other type of marine accident. The most common injuries are head, brain, and neck injuries; fractures; and lacerations. Two thirds of watercraft involved in collisions are open motorboats under 26 ft (8 m) in length and jet ski personal watercraft (PWC). Most fatalities occur with open motorboats. Seventy percent of fatalities result from drowning. Nearly 85% of drowning victims fail to wear a personal flotation device (PFD or lifejacket). Jet ski riders tend to wear life jackets, which may explain the lower number of fatalities in this group. Approximately 80% of all reported boating accidents involve operator-controllable factors, not equipment or environmental factors (see later). The boats generally have no major mechanical problems; fair weather with good visibility and calm seas are the rule. The majority of recreational boating accidents occur not offshore, but on inland or near coastal waters, which corresponds to the most commonly used areas for recreational boating.

The most common types of boats involved in reported accidents are open powerboats (45%), personal watercraft (26%), and cabin cruisers (14%). In 2005, a record-breaking 13 million motorized crafts were registered in the United States.[8]

Operator factors responsible for collisions include inattention and inexperience, carelessness, recklessness, excessive speed, and improper lookout. Seventy percent of all reported fatalities occur on boats whose operators have not received boating safety education.[8] Alcohol consumption in reported accidents is believed to be a contributing factor in 25% of the fatal accidents.[8]

Collisions with Submerged Objects

For adventurous boaters cruising around the world's oceans, collisions are a major hazard. Encounters with whales may be the most memorable, but are still rare. The major threats to small boat cruisers are collisions with partially submerged objects (rogue containers, 55-gallon drums, uncharted coral heads) or being run down by a commercial ship along standard shipping routes.

An estimated 1000 to 10,000 shipping containers fall from the decks of merchant ships every year during storms; these ships are not equipped to retrieve them. Exact numbers are difficult to obtain due to underreporting because of the potential liability involved. Unfortunately, shipping companies are reluctant to report either the location or numbers of containers lost. In a single instance, more than 300 containers were lost from a container ship caught in Typhoon Babs in 1998. The containers and cargo from the 900-ft ship may still be adrift in the Pacific! It is estimated that half of the containers continue to float, like steel icebergs, with only one corner above the water, or submerged and invisible just below the surface. The most common shipping container is 40 ft long and 8 ft wide (12 × 2.4 m), constructed of aluminum or steel; it is capable of holding up to 50,000 lb (22,680 kg) of cargo. A refrigeration container weighs about 4 tons empty and may float partially submerged, even with the doors ajar, because of its thick insulation.

The sea is rapidly becoming littered with athletic shoes, toys, dolls, plastic bags, and other types of floating cargo. A transport carrying 12,000 tons of 40-ft white spruce logs to British Columbia intentionally dumped 90% of its cargo into the Gulf of Alaska during a storm. During the same weather pattern, a Pacific storm overwhelmed four container ships en route from Japan to Seattle, causing them to lose 400 containers overboard.

Massive stumps and log debris float in the coastal waters off northern California, Oregon, and Washington because of logging practices in northern river watersheds. The waterlogged stumps flow down the rivers, enter the ocean, and often float just below the surface. Unfortunately, even with technology, the sailor's best and only defense is still to keep a sharp lookout, because partially submerged objects, especially floating logs and cargo containers, are difficult to detect by radar.

A number of factors determine a boat's vulnerability to puncture. Steel and aluminum hulls offer good protection. Solid fiberglass laminates are less tough, but stronger than wood. Hull form is another consideration; traditional deep draft, heavy displacement designs with their sharp entries are more likely to ride over or push away debris than modern light displacement boats with fin keels and spade rudders. Offshore multihulls sailing at full speed are particularly prone to damage; the bows of their light, composite, unprotected amas (pontoons)

can easily shear off or hole in a collision with a heavy floating object.

Collisions with Other Vessels

International rules for preventing collisions at sea, referred to as COLREGS, define the responsibility of ships when collision is possible between two boats, as when crossing each other, overtaking, or meeting head on. Although the *stand on* vessel, which has the right of way over the *give way* vessel, is permitted to hold course and speed, the rules require that both ships take any actions necessary to avoid an imminent collision. In the presence of large ships, small boats must be especially vigilant. Consider any tow vessel, large freighter, or tanker operating upon inshore waters to be restricted in maneuverability. Rules 3 and 18 of the Inland Navigational Rules make it clear that a sailing vessel does *not* have right of way over these vessels. As a professional tug captain declared, "If it looks like it is going to be close, it already is too close." In commercial traffic areas, maintain a deck watch at all times, and hail any approaching ship on very high frequency (VHF) channel 13 (or 16 if no response) if uncertain of the ship's course or intentions. Be prepared to take defensive action. If two vessels are in a crossing situation and the bearing (angle) between them remains constant, collision is inevitable. Visibility from the pilothouse of a large vessel may be partially obstructed by containers, fishing gear, or other items on deck; small craft may not be seen by the ship's lookout or pilot, show up on radar, or be granted right of way. This is a common cause of collisions along the shipping lanes around the world for all commercial and recreational small craft.

The 47-ft (14-m) sailing vessel (S/V) *Melinda Lee* was run down by the 540-ft (165-m) bulk carrier *Pan Grace* off the coast of New Zealand in 1995; the cargo ship had no running lights, had not turned on its radar and ARPA (see later), and had disregarded the sighting of *Melinda Lee*'s "dim, red light," which appeared faintly and then disappeared on the horizon. Two children and their father drowned within hours following the collision. A review of this tragedy in *Blue Water Sailing* magazine (August 2002) concluded: "Big boats don't see you; they don't have to. They may be required to look for you, but as a sailboat of virtually any size represents no threat at all to a freighter, you must rely on yourself, not them, to avoid being run down."[1]

Unfortunately, commercial ships and solo ocean racers often run on autopilot without a lookout on deck. This is a clear violation of rule 5 of the COLREGS: "Every vessel shall at all times maintain a proper lookout by sight and hearing."

For collision avoidance, many cruising sailboats install a masthead strobe light. Although they are highly visible lights, they are not accepted internationally as legal running lights, and the Coast Guard does not recommend them. A flashing strobe light may be interpreted as a distress signal, paradoxically inviting an unwelcome convergence of ships. Shining a powerful spotlight on the mainsail, turning on spreader lights, or igniting a white "anti-collision" flare can be an effective alternative means of alerting a ship to the danger of collision. It is recommended that cruising sailboats have masthead running lights for coastal and offshore cruising (to extend the range of visibility), in addition to deck level lights for meeting, crossing, and passing maneuvers where vessels are nearby.

Mariners should recognize the different patterns of lights displayed by commercial ships, but be aware that outside U.S. coastal waters, many ships do not always show the appropriate lights. The risk of collision is increased; be vigilant, and be prepared to make quick decisions and emergency maneuvers when cruising in foreign waters.

Figure 76-4. Although radar reflectors can increase the visibility of your vessel to other radar-equipped vessels, an active radar watch is still the best technique for collision avoidance.

Radar is invaluable in poor visibility (Fig. 76-4). At night or in fog, radar can be used to identify other ships, hazards to navigation, squalls, and other local weather that may endanger a vessel.

Small, affordable radar units are available for recreational boaters. Most units are simple to operate; skill with their use comes with practice, study, and patience. Under normal atmospheric conditions, the radar horizon is longer than the optical one by 6%, making radar equally useful while navigating by day. The horizon can be increased by raising the antenna height (radar horizon=2.2 × [square root of antenna height in meters + square root of target height in meters]). Modern units integrate a variety of data, including electronic bearing lines, rings that give a fast reference point to a target, and the variable range mark (VRM), which marks the range to a target at a particular time. These data are invaluable for avoiding collision with another vessel. Many units can be integrated with the global positioning system (GPS), electronic charts, and interfaced with computers for precision course plotting and tracking during navigation in narrow and complicated passages. A big advance in collision avoidance is ARPA (automatic radar plotting aid); ARPA works in conjunction with the main radar and is required by COLREGS rule 7 to be used whenever risk of collision exists.

It provides automated long-range scanning, sounds an alarm when a vessel comes within a predetermined distance, calculates the speed and bearing of other vessels, and automatically calculates the course alternatives needed to avoid collision.

One problem common to units on small boats is that, during storm conditions, large commercial ships and yachts may be lost on the radar screen because of the mass of echoes from nearby tall waves. The intense reflection of radar signals from the sea is called sea clutter. Fiberglass and wooden boats and wood spars are nearly invisible to radar. Regardless of construction, all boats should have a radar reflector mounted at all times while in shipping lanes, on the open sea, or under conditions of reduced visibility. Ideally, a reflector should provide consistent reflective performance in all directions (most do not). The larger the reflector, the better the reflection. Doubling a reflector's diameter increases its effective area 16 times. The effective area increases with the fourth power of the diameter of the device.

To make a vessel the brightest reflection on the other vessel's radar screen, mount an active transponder on the masthead. This device receives the radar signal and transmits it back to the radar unit with a signal that is electronically enhanced and stronger than one from a passive reflector.

Another important function of radar is the ability to maintain an electronic guard zone. The radar scans this zone, which is preset as a circle of a given radius, and sounds an alarm when a new target enters the guard zone. Box 76-5 reviews measures to prevent collisions at sea.

The sinking of the 19-ton, 43-ft schooner *Lucette* in the Pacific after attack by three killer whales reminds all sailors that yachts can be destroyed by unprovoked whale attacks. Cruising at 10 knots and accelerating underwater to speeds up to 30 knots, a whale has little time to reevaluate its target while attacking torpedo style. Other attacks may be reprisal by a whale accidentally rammed while sleeping at the surface. This is probably what occurred to the cruise ship *Royal Majesty,* which arrived in Bermuda with a dead 60-ft finback whale balanced atop its bow.

Sailors crossing the North Atlantic in summer along a great circle route to Europe need to beware of colliding with icebergs. Every year from February to July, more than 10,000 icebergs are separated from Greenland glaciers. At least a thousand of them drift in the Labrador Current south and east of Newfoundland and become a hazard to mariners above 41° north latitude. Navigation around icebergs is complicated by dense fog often present over the Grand Banks region and some difficulty of detecting icebergs with radar. Iceberg reports are broadcast twice daily by U.S. and Canadian Coast Guards. The International Ice Patrol broadcast times and frequencies can be obtained by telephoning 203-441-2626.

Box 76-5. Preventing Collisions at Sea

1. Post a lookout with a 360-degree view of the horizon.
2. Know the rules for steering and right of way.
3. Use radar (if available) and running lights. A tricolor masthead light is most visible.
4. Use collision avoidance radar detector (CARD) if available.
5. Mount radar reflectors, and place them as high as possible.
6. Do not assume other boats have operational or unobstructed running lights. Be aware outside of U.S. waters that navigation lights on foreign ships may not match standard configurations.
7. Observe rule 7 of the COLREGS: "When a vessel has any doubt as to whether a risk of collision exists, she shall assume it does and avoid it." Be prepared to change course and speed.
8. Ask yourself, "Can I be seen?" Do not assume you are seen, even in daylight. The height of the steering station of a large commercial vessel makes it difficult if not impossible to see a small boat near the bow or alongside the ship.
9. If you hear a foghorn, stop and let the other ship steer around you.
10. If a collision threatens, sound the danger signal (five or more short blasts on the horn), and take whatever actions are necessary to save your boat.
11. Maintain radio contact with other ships in the area. VHF channel 13 is the bridge-to-bridge channel. Use VHF channel 16 if there is no response.
12. Avoid shipping routes if possible, and avoid navigating in the harbor's incoming and outgoing traffic separation lanes designated for commercial traffic.
13. When your boat is in the way of another vessel, the right of way does not automatically grant you safe passage. Regardless of the COLREGS, be prepared to take evasive action. Caution is the primary rule of the sea to avoid collision.
14. To avoid collision with another vessel, make changes in course and/or speed early and recognizable. Port-to-port is a good rule for most head-on passing situations (sound one whistle). In overtaking situations, one whistle means you are overtaking on the starboard side of the overtaken vessel, and two whistles mean the port side.
15. Give large ships a wide berth. Whenever possible, avoid meeting or overtaking tugs near river bends, bridges, and narrow channels.
16. Observe the bearing of an approaching vessel (the direction relative to you). If the bearing does not change, and if the distance between the two boats is decreasing, you are on a collision course.

COLREGS, international regulations to prevent collisions at sea.

Seasickness

Seasickness is probably the most frequent medical problem responsible for maritime search and rescue operations around the world. During stormy weather, mariners frequently consider seasickness a medical emergency and justification for medical evacuation. Each year, dozens of seaworthy yachts are abandoned because their exhausted and despondent crews have lost their collective will to persevere. "They are wet, seasick, scared, and want to go home," observed a merchant marine captain (personal communication with author).

Such was the case for the crew of *Miss Manhattan,* a 43-ft ocean-cruising sailboat caught in a powerful northeast gale 45 miles east of Cape Hatteras on Memorial Day 2000. With nightfall approaching and rescue available, the wet, cold, panicked, and seasick crew had enough. They radioed an already overburdened HH-J60 Coast Guard helicopter on duty from the Coast Guard Air Station in Elizabeth City, North Carolina.

With the chopper hovering overhead, they risked their lives (and those of the Coast Guard crew) by jumping into 30-ft seas, where they were individually hoisted aboard the helicopter with the assistance of a rescue swimmer and basket. A few days later, they drove to meet and secure their perfectly intact and seaworthy craft as it drifted ashore on the Outer Banks beach about 15 miles north of Cape Hatteras. Had they persevered, they could have simply walked ashore 2 days later and not imperiled their lives and those of their rescuers. Halsey Herreshoff, a respected sailor and renowned yacht designer, once said, "The crew gives up before the boat."

At the very least, seasickness *(mal de mer)* is moderately disabling. It can also lead to rapid mental and physical deterioration marked by progressive dehydration, loss of manual dexterity, ataxia, loss of judgment, and loss of the will to survive. Fatalities from seasickness have occurred because of poor seamanship and complications arising during hazardous emergency evacuations.

Seasickness impairs cognitive function, especially short-term memory. Sailors often lose the ability to multitask, making it difficult to analyze and integrate complex data, which leads to faulty decisions.

The underlying mechanism of seasickness involves a conflict of sensory input processed by the brain to orient the body's position. Place someone in the cabin of a heeling or rolling boat and you immediately invite mal de mer. Below decks, the eyes oriented to the cabin sole and ceiling detect no tilt from vertical, while fluid in the inner ear's vestibular system constantly shifts. Position sensors (proprioceptors) in the neck, muscles, and joints send additional signals depending on how a person shifts and secures himself from falling. This mix of sensory data from the eyes, inner ear, and position sensors arrives in complex and conflicting combinations, creating a neural mismatch that activates the emetic center (vomiting center) in the brainstem.

The relative importance of visual and vestibular information in the etiology of seasickness is not well understood. Each stimulus can independently initiate symptoms. Sickness occurs most commonly with acceleration in a direction perpendicular to the longitudinal axis of the body, which is why head movement toward and away from the direction of motion is so provocative. In the laboratory (and in flight simulators and movie theaters), visual stimulation is more important than vestibular input in causing motion sickness when these stimuli are presented in isolation, but there is no increase in symptoms when combined stimulation is applied. Of interest is that blind people can become seasick; the conflict arises when input from the vestibular system does not match the individual's expectations derived from previous motion experience. The intensity of conflicting input can also be amplified when compared to these expectations. In response to visual or vestibular input, the neurotransmitter dopamine stimulates the medulla oblongata's chemoreceptor trigger zone, which stimulates the vomiting center within the lateral reticular formation of the brainstem.

The vomiting center is also directly stimulated by motion and by high levels of released acetylcholine. Neurotransmitters such as serotonin and neurokinin also appear to activate the chemoreceptor trigger zone and vomiting center. These neurotransmitters and their receptors are the targets for most of the drugs used to ease symptoms of seasickness. The benefit of antihistamines is derived from their anticholinergic properties rather than antihistamine properties. Sympathomimetic drugs counteract seasickness directly and in synergy with anticholinergic medication. In summary, if one eliminates the sensory conflict so that "the eyes are seeing what the ears are feeling," then one is likely to avoid seasickness. Several preventive measures one can take aboard ship incorporate these principles.

The balance center has the ability to adapt to new environments, commonly called "getting your sea legs." Medication is more effective in preventing symptoms than in reversing them; therefore, drugs should be used early, before leaving port. Begin the trip well hydrated and free of the after-effects of alcohol, which impairs vestibular function by sensitizing the vestibular apparatus to motion (recall a staggering drunk). Eat lightly; anecdotal reports favor eating carbohydrates rather than protein, but no conclusive study favors any particular food or diet. Many sailors favor eating saltines or bread. Snack on bland foods throughout the day, even if anorectic, to maintain energy levels until meals are regularly tolerated. Cheese and crackers, energy bars, fruit, trail mix, dry granola, and popcorn work best. Drink small amounts of fluid frequently to avoid dehydration. Ginger is often recommended as an antiemetic and may be clinically useful in individual cases. Ginger was tested in a double blind, randomized, placebo-controlled study of 80 naval cadets in rough seas. Seasick cadets were given a gram of ginger or placebo hourly for 4 hours. Ginger significantly reduced vomiting and cold sweats, and minimally decreased nausea and dizziness. Ginger is available in 500-mg capsules; the suggested dose is 1000 mg every 6 hours, starting one-half hour prior to the trip; it is less effective when given to someone who is already nauseated. The capsules can be supplemented with foods containing lower concentrations of ginger, such as gingersnap cookies, ginger ale or tea, and candied ginger. Eating too much ginger may be counterproductive; it may cause heartburn and should not be taken by people with gallstones, because it can provoke an attack of biliary colic by stimulating the flow of bile.

Both field and laboratory experiments have documented the efficacy of acupressure in preventing seasickness; however, some experts on space and motion sickness still consider acupressure no better than placebo. One sea trial showed that acustimulation suppressed the symptoms of motion sickness.[3] Pressure should be applied on the Neiguan P6 point of the forearm. This is found two to three fingerbreadths proximal to the wrist joint between the two prominent finger flexor tendons. There are commercially available elastic wrist straps with plastic studs that create pressure over the P6 point. Sea-band, BioBand, and Queaz-Away are some of the popular inexpensive bands. The ReliefBand is a wristwatch-like device worn over the P6 area on the underside of the wrist to deliver transcutaneous electrical stimulation to the median nerve. It can be used before or after the start of symptoms and offers an alternative to people sensitive to the side effects of medication. This device is very effective for postoperative nausea and vomiting, and may be as effective for many seasick sailors as are commonly used drugs. The electrical stimulation can be felt as a tingling sensation or an annoying distinct shocking sensation in the fingers. Adjustment to the lowest effective power setting is recommended for comfort.

Recommendations for preventing mal de mer include limiting the time below decks, keeping mealtime preparation in the galley to a minimum, and providing easily prepared meals, such as freeze-dried meals. Keep personal items ready and easily accessible.

After departure, stay on deck and amidships (center) or aft (toward the stern), where pitching and rolling are less severe.

TABLE 76-1. Medications for Seasickness

MEDICATION	DOSE	INTERVAL
Dimenhydrinate (Dramamine)	50 mg liquid/capsule/chewable tablet	4 to 6 hr
Meclizine (Antivert)	12.5/25 mg tablet	6 to 8 hr
Meclizine (Bonine)	25 mg chewable tablet	6 to 8 hr
Cinnarizine (Stugeron)	15 mg tablet	6 to 12 hr
Scopolamine (Transderm-Scop)	1.5 mg skin patch	Every 72 hr
Scopolamine (Scopace)	0.4 mg (1 to 2 tabs)	8 hr
Promethazine (Phenergan)	12.5/25/50 mg tablet, suppository, injection	6 to 12 hr
Phenergan plus ephedrine	25 mg tablet of each	6 to 12 hr

Look out and obtain a broad view of the horizon using direct and peripheral vision. This provides a stable and level point of reference. Avoid close visual tasks such as prolonged reading, writing, and navigation. Avoid areas with fumes (especially diesel) and odors that can stimulate nausea. Continue medication started prior to departure for preventing seasickness at the suggested intervals, and begin to taper the dose after the first or second day, depending on conditions.

The early signs and symptoms of seasickness are yawning, sighing, dry mouth or salivating, drowsiness, headache, dizziness, and lethargy. With sustained exposure to the stimulus, gastric emptying is inhibited, and pallor, cold sweats, belching, nausea, dry heaves, and vomiting ensue. The window of opportunity for early intervention is often missed because early signs are not recognized or the person is in denial.

At the first sign of seasickness, take the helm and steer. Feel the waves and focus on clouds, horizon and distant marks for reference points. When not steering, sit or stand in the cockpit and actively posture yourself to anticipate the boat's motion and "ride" the waves. Keep head and shoulders balanced over hips. Stay in balance and gain postural control gracefully.

If symptoms progress, lie down in a secure, well-ventilated bunk, face up with eyes closed and head still; try to sleep. Use parenteral antinausea medication such as the phenothiazine derivative promethazine hydrochloride (Phenergan). This drug has powerful antidopaminergic, anticholinergic, and antihistamine properties. It is useful for both prophylactic and active treatment of seasickness and can be administered as a suppository, intramuscularly, and orally as a tablet or syrup. Continue to take sips of water, crackers, and hard candy.

Debilitated seasick persons can easily fall or be washed overboard. They should always wear a safety harness on deck and be closely monitored. In storm conditions, the safest place is to be secured below in a bunk.

Table 76-1 lists medications available for motion sickness. Time-release forms and longer-acting drugs may be preferable when storms are expected to last a few days.

Transdermal scopolamine hydrobromide (Transderm-Scop) is the most popular anticholinergic agent used for treatment of motion sickness. Scopolamine prevents motion-induced nausea by inhibiting vestibular input to the central nervous system, resulting in inhibition of the vomiting reflex. It may also act directly on the vomiting center. The drug is delivered via an adhesive patch placed behind the ear 4 or more hours before departure. Scopolamine enters the bloodstream at a constant rate to provide a therapeutic blood concentration for up to 3 days, often with minimal side effects. The most common adverse effects are dry mouth (66%) and drowsiness (17%). Additional undesirable side effects include blurred vision (which may persist for weeks), dry mucous membranes, short-term memory loss, and other problems denoted by the well-known mnemonic "hot as hell, dry as a bone, blind as a bat, mad as a hatter." To reduce the dose of scopolamine, allow only half of the intact patch to contact the skin by placing the other half onto a Band-Aid or tape attached to the area. Do not disrupt the integrity of the disc by cutting it. Follow the directions carefully, wash hands thoroughly after application, and apply only one disc at a time. Scopolamine is contraindicated for children, persons with narrow-angle glaucoma (remove the patch immediately if eye pain suddenly occurs), and men with prostatic hypertrophy (enlargement). Long-term use may produce withdrawal symptoms such as nausea, dizziness, headache, and equilibrium disturbances. Scopolamine in pill form (Scopace) is an alternative to the patch. The chief advantage is the dosing flexibility; the fixed dose from the patch may be excessive for small individuals, and inadequate for larger people. The side effects can therefore be minimized by taking the lowest effective dose. The current recommendation is one to two tablets (0.4 mg) 1 hour prior to departure, and thereafter one to two tablets every 8 hours as needed.

Over-the-counter antihistamines have potential side effects including drowsiness, dry mouth, blurred vision, irritability, urinary retention, dizziness, and headache. NASA astronauts use a combination of intramuscular promethazine and oral ephedrine to counter the drowsiness. An effective nonprescription substitute for ephedrine is the decongestant pseudoephedrine, which is available in 30- to 100-mg tablets.

Whichever drug is selected, be sure to try it on shore before the trip, to evaluate potential side effects. It is not uncommon for one drug in a category (e.g., antihistamine) to be effective and a related drug to provide no benefit; the same is true for side effects. Finally, if all else fails, follow Samuel Johnson's 18th century advice: "To cure seasickness, find a good big oak tree and wrap your arms around it."

THUNDERSTORMS AND ASSOCIATED WEATHER EVENTS

A single thunderstorm generally encompasses an area less than 2 miles (3 km) in diameter. It lasts approximately 30 minutes, with marked fluctuations in wind, temperature, and barometric pressure. The National Weather Service (NWS) considers a thunderstorm severe if it produces hail at least $^{3}/_{4}$-inch (2-cm)

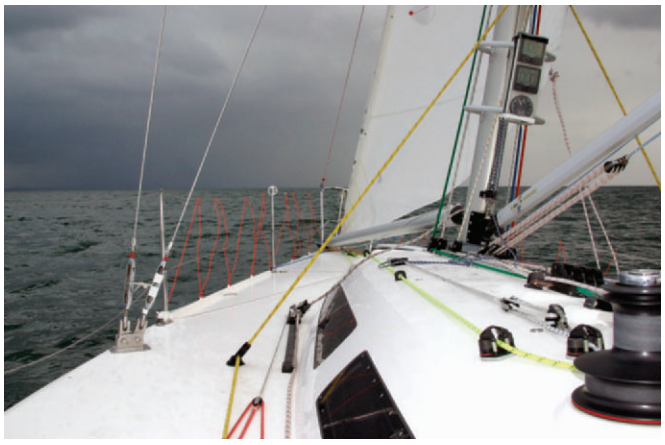


Figure 76-5. Mariners have to be continually vigilant for changes in their environments. Rapidly approaching storm fronts call for reduced sails, harnesses and tethers, and secured hatches.

in diameter, winds of 58 mph or stronger, or a tornado. Of the estimated 100,000 thunderstorms that occur on inland and coastal areas in the U.S., 10% are classified as severe. Eighteen hundred thunderstorms occur at any moment around the world, totaling 16 million a year. A squall line is a fast moving row of violent thunderstorms, often more than 100 miles (160 km) long. An intense cold front 40 to 300 miles (64 to 483 km) behind the squall line contains most of the gusting winds and rain normally found in the front. It is visible on radar, and to the naked eye it appears as a wall of boiling black clouds arising from the sea.

Rapid growth of cumulus clouds is the primary indicator of a forming thunderstorm (see below). The faster the clouds build, the more violent the resulting storm, because steep pressure gradients within the cloud generate high winds. Within the thunderhead are columns of rapidly sinking air called *downdrafts*. Downdrafts along the leading edge of the thunderstorm form the gust front. This zone of advancing cold air is characterized by a sudden increase in wind speed. Strong and highly localized downdrafts are called downbursts, the smallest of which are microbursts. Airplane pilots refer to these as wind shear.

Downbursts are extremely intense concentrations of sinking air. On reaching the surface, they fan out radially in all directions, often generating winds at the gust front in excess of 150 knots. They are short-lived, typically lasting less than 15 minutes. A single thunderstorm can produce a series of downdrafts affecting an area several miles long and persist an hour or more. Blowing spray under or slightly ahead of the storm may be the only indicator of its presence. A gust front often precedes a microburst. The combination of these two extremely strong and shifting wind systems can blow equipment and personnel off the deck and can easily capsize small craft.

In the United States, squalls occur predominantly during the spring and summer in association with thunderstorms generated by towering cumulus clouds (Fig. 76-5). The larger the cloud's size (and radar echo), the more wind potential in the cloud. The taller the cloud (especially above 20,000 ft [6096 m]), the more energy potential for stronger winds. Most squall formations occur at night when cloud tops radiate heat back into space, enhancing their ability to grow. Squall-generated winds rarely strike without warning. A rapid fall in temperature is the precursor of a local storm. The bigger the drop, the stronger the winds, and rain that precedes the wind suggests stronger winds are coming.

Occasionally, thunderstorms may form with the building of the cumulus cloud and not be associated with the typical rain, thunder, and lightning. These violent winds are called white squalls. The only warning may be the sudden appearance of a cold, shifting wind with an increase in velocity. Large sailboats, such as the clipper topsail schooner *The Pride of Baltimore,* have been knocked down and sunk by these unexpected powerful winds. In May 1986, a sudden storm rumbled across the Atlantic and unleashed its strength over a small patch of the Bermuda Triangle. With furious precision, its 70-knot winds overwhelmed the 97-ton, 110-foot ship, an historically authentic clipper ship heading home from Europe; survivors reported that the ship immediately barrel-rolled in the heavy wind and sank.

To avoid or prepare for the potential fury of a thunderstorm, the mariner must monitor local weather conditions. The best up-to-date weather information for coastal waters is the continuous 24-hour NWS broadcasts (Box 76-6). The National Oceanographic and Atmospheric Administration (NOAA) VHF-FM radio transmissions repeat recorded messages every 4 to 6 minutes (Box 76-7), updating them every 2 to 3 hours with the latest local information. The broadcasts usually can be received 20 to 60 miles from the transmitting antennas. Nationwide, more than 860 transmitters provide coverage to all 50 states and the adjacent marine areas. In many areas, the Coast Guard broadcasts weather information on VHF channel 22. Listings of schedules and frequencies for coastal and offshore weather broadcasts are available in a number of publications, including the *Admiralty List of Radio Signals,* Volume III, and *Reed's Nautical Almanac*.

Box 76-6. National Weather Service Broadcasts

NOAA WEATHER RADIO CHANNELS
WX-1 162.55 MHz
WX-2 162.40 MHz
WX-3 162.475 MHz

CANADIAN WEATHER CHANNELS
Canadian Channel 21 161.65 MHz

WEATHER WARNINGS BY RADIO
National Bureau of Standards: WWV in Fort Collins, Colorado, and WWVH in Hawaii broadcast time signals and weather warnings on 5, 10, and 15 MHz. WWV broadcasts at 8, 10, and 12 minutes after the hour. WWVH broadcasts at 47, 49, and 50 minutes after the hour.

U.S. COAST GUARD (USCG) BROADCASTS
- USCG VHF Voice: Broadcasts coastal and offshore forecasts and storm warnings on VHF channel 22A after an initial call on VHF channel 16.
- USCG MF Voice: Broadcasts offshore forecasts and storm warnings on 2670 kHz following an initial call on 2182 kHz.

NOAA, National Oceanic and Atmospheric Administration.

Box 76-7. Storm Warnings

1. Small craft advisory: generally associated with sustained winds 18 to 33 knots (l9 to 38 mph) and/or sea conditions dangerous to small boats.
2. Gale warning: sustained winds 34 to 47 knots (39 to 54 mph)
3. Storm warning: sustained winds over 48 knots (55 mph). If winds are associated with a tropical cyclone, the storm warning display indicates forecast winds of 48 to 63 knots (55 to 73 mph).
4. Hurricane warning: sustained winds of 64 knots (74 mph) and greater as the result of a tropical cyclone
5. Special marine warning: winds of 35 knots or more generally lasting less than 2 hours. These are usually associated with an individual thunderstorm or an organized series of thunderstorms, as in a squall line.

Many of the newer VHF radios have a weather-alert function known as Weather Watch. When the radio receives a special warning signal from NOAA, it sounds a special alerting tone, signifying an urgent NOAA weather forecast. Some radios automatically tune to the active weather channel, whereas others require manual tuning. In 1997, NOAA improved their severe weather warnings by encoding both the area affected by the severe weather and the nature of the severe weather (e.g., hurricane, winter storm, high wind, severe thunderstorm). Called SAME (Specific Area Message Encoding), this feature is intended to make weather warnings more customized so that users will not tune out possibly life-saving warnings.

Offshore sailors can receive a variety of high-seas marine weather broadcasts with single-sideband (SSB) high-frequency (HF) radios. The time schedules and frequencies for these transmissions can be found in the *U.S. Defense Mapping Agency (DMA) Publication 177, Radio Navigation Signals*. A less comprehensive list for specific locations can also be found in *Reed's Nautical Almanac*.

The U.S. Coast Guard sends a facsimile of a weather chart (weatherfax) from their East, Gulf, and West Coast communication centers. These marine weather charts are updated every 6 hours and available within $3^1/_2$ hours of the valid update time. Weatherfaxes contain graphic charts and forecasts compiled by the NWS; they are broadcast on high-frequency radio bands between 3.5 MHz and 30.0 MHz. Lists of transmitters, frequencies, and times can be found in *Reed's Almanac* or online at www.nws.noaa.gov/om/marine/fax.pdf.

The NWS Internet page at www.nws.noaa.gov lists all weather charts broadcast over SSB. This site contains text forecasts, ocean current and surface analysis charts, wave charts, buoy reports, and prognosis charts for weather outlooks of 12, 24, 36, 48, and 96 hours (500-millibar [mb] charts).

An onboard cellular connection, modem, and laptop computer are required to obtain this Internet information. Beyond cellular range, a dedicated weather fax unit or SSB, laptop, or weatherfax module is needed. New software can integrate weather forecasting with optimal routing for safety and sailing performance. Weather files (called GRIB files) can be downloaded and overlaid onto an electronic chart, and routing formulas can then be applied incorporating the specific boat's known performance characteristics. MaxSea, obtained from SetSail.com, is one of the most popular offshore electronic charting and navigation programs. Mariners can obtain sea state and weather data directly from NOAA buoys moored offshore by using the Web. The National Data Buoy Center website is www.ndbc.noaa.gov; clicking on a specific station shows data gathered by the buoy in the preceding minutes.

NAVTEX is a worldwide land-based radio navigation warning service, transmitting text-only messages (in English) to a dedicated onboard receiver. The unit can be programmed to receive both specific stations and message categories; it can print out area weather forecasts, gale warnings, navigation warnings, ice warnings, and relayed distress messages on the assigned frequency of 518 kHz. Vital messages (e.g., gale warnings, search and rescue information) activate an alarm in the receiver as the message is being printed.

There are also compact radio facsimile receivers that receive only graphic data. Dedicated weatherfax units have the advantage of automatic reception without keeping a laptop and SSB turned on for extended periods.

Visualization of characteristic cloud formations at night can be accomplished with radar or night-vision binoculars. Even without expensive electronic equipment, mariners can predict changes in local weather by using their own observations. Know the cloud types or use a photo atlas of cloud formations to identify the variety of clouds. During the day, a sailor can learn to recognize a squall line, the growth of cumulus to a cumulonimbus cloud with its characteristic anvil head on the downwind side (a thunderhead), and the cloud sequence and wind changes of an approaching cold and warm front. "Mare's tails and mackerel scales; soon it's time to shorten sails," refers to cirrus and cirrocumulus formations (long puffy clouds at high level, commonly known as a mackerel sky). This cloud formation signifies unsettled weather with the approach of a warm front. Two weather concepts can be helpful for predicting changes in the weather: the "crossed-winds rule," popularized by the meteorologist Alan Watts, and Buys Ballot's law. The crossed-winds rule states that whenever the upper level wind flow (determined by observing the direction of the cirrus clouds at 20,000 to 30,000 ft [6096 to 9144 m]) and lower level surface winds are crossed, the weather is going to change. Buys Ballot's law states that in the Northern Hemisphere, when you stand with the wind blowing at your back, a high pressure will be at 90° to your right, and a low pressure 90° to your left (remember "low-left"). If the application of crossed-winds rule and Buy Ballot's law indicates a low is approaching, the weather will certainly deteriorate; expect a warm front with increasing winds and rain. Once past, a cold front follows. If the upper-level flow either matches or directly opposes the surface wind, the weather is likely to remain stable for a while.

During daylight, a band of low, dark, and smooth tubular roll clouds can often be seen at the leading edge of a squall, preceding a cold front. The faster a roll cloud approaches, the stronger the wind is likely to be, and the more agitated the sea appears under it. When the sea is observed beneath the roll cloud, the cloud is about 2 miles away; because clouds typically move at about 25 mph, the face will arrive in about 5 minutes.

The barometer is one of the most important weather instruments onboard a small boat. One to 4 hours before an approaching thunderstorm, there is a sharp drop in barometric pressure of about 1.5 mb. When the atmospheric pressure is fluctuating very little, it means the weather is likely to remain stable. A pronounced rise in pressure heralds fair weather.

Sea state is also a valuable clue to changing weather patterns. Large ocean swells precede a heavy weather system, whereas

Box 76-8. Defensive Measures in Thunderstorms*

1. Alert everyone on board of the imminent storm threat.
2. When boating on a small pond or lake, return to shore and get off the boat.
3. If practical and time permits, take evasive action and set a course perpendicular to the expected thunderstorm track. In rough water, a small boat is most stable going downwind with the seas on the stern quarter (about 45 degrees to the back of the boat).
4. Ascertain position and identify any shoals, rocks, and other boats in the immediate area that may pose a hazard to navigation if visibility becomes severely reduced.
5. If practical, seek shelter from wind and waves in the lee (downwind) side of a land mass.
6. Secure all loose gear on deck and below, and verify that lockers, cabinets, and drawers are properly secured.
7. Close all hatches, ventilators, ports, and lazarettes to prevent flooding in the event of a knockdown or capsize. Close all seacocks not immediately required. Secure doors or boards to companionways leading below.
8. Reef the sails (reduce sail area) or lower them completely, and start the engine.
9. Pump the bilge dry, and be sure the pumps are ready for operation. Water shifting in the bilge can adversely affect the boat's stability.
10. All crew and passengers should wear foul weather gear and life jackets.
11. All crew on watch should be secured with a safety harness.
12. All remaining personnel should go below with clear knowledge of how to egress in the event of a capsize.
13. Safety gear, communication (hand-held VHF radio), and visual distress devices should be readily available.

*Measures are listed in sequence, as storm approaches.

chop without swells often reflects a local, more isolated, and temporary disturbance.

An excellent early-warning device is the SkyScan Lightning and Severe Storm Warning unit. This unit has a sophisticated receiver for listening to the characteristics of lightning and can detect and report air-to-ground lightning strikes in four different mileage ranges reaching up to 40 miles. The unit can calculate the distance of a ground strike to within 1 mile 97% of the time.

An alert observer can calculate the distance between a vessel and a thunderstorm within earshot. Lightning's distance from an observer can be determined by noting the time between the flash and the bang of associated thunder. For each 5-second count from flash to bang, lightning is 1 mile (1.6 km) away.

Because it is impossible to predict the power of thunderstorms, mariners should always prepare for the worst and initiate defensive tactics (Box 76-8).

Lightning

Lightning is one of nature's most destructive phenomena (see Chapter 3). Boating, fishing, and swimming rank second only to playing sports on an open field as the most dangerous activities associated with a lightning strike. Although the odds that an individual will be struck and killed by lightning are about 1 in 3 million, the odds for a boat being struck are greater. A cruising sailor in Florida, the lightning capital of America, can expect at least one strike to his boat in its lifetime. Although there is no lightning-proof boat, it is possible to reduce the likelihood of a lightning strike and provide a high degree of protection for the boat, crew, and equipment.

Tall and narrow objects, with highly charged and focused electric fields, are likely to attract lightning. Metal itself does not attract lightning. A sailboat mast, radio antenna, fishing outrigger, fishing rod, and even persons standing in a motorboat all make good targets. In a marina or anchorage, the boat with the tallest mast is most vulnerable to a strike. The lesson: Do not be the tallest object.

The most critical factor initiating the streamer emanating from the boat is not only the height of the boat's mast, but also its electric potential. The crackling bluish-green light sometime seen in a ship's rigging at night during thunderstorms is not lighting, but a type of electrical discharge (corona discharge) called St. Elmo's fire. It may even appear like a stream of fire as it trails from the mast. Magellan's storm-battered crew regarded the "fire" as a sign of divine protection by Saint Elmo, the patron saint of mariners. Captain Ahab saw St. Elmo's fire and reassured his ill-fated crew aboard the whaler *Pequod,* "The white flame but marks the way to the white whale."

St. Elmo's fire occurs when there is a large difference in electrical charge between the mast and the surrounding air. This causes air molecules to be split apart by the voltage streaming off the mast, and the resulting gas begins to glow. Do not climb the rigging to check it out; there are 30,000 volts per centimeter of space surrounding the masthead! Fires and explosions destroy many boats struck by lightning. A strike exiting through the bilge can instantly vaporize trapped bilge water and raise it to explosive pressures. Boats sink because of ruptured through-hull fittings or large exit holes burned in the hull. Protect the structural integrity of the boat by providing a path of least resistance for the major current to travel through and off the boat into the water. Protection from a side flash (see below) is accomplished by taking precautions to equalize the electric potential of all metal objects aboard. By understanding the components of a lightning protection system (LPS), the mariner can appreciate how best to shield the boat and crew. However, lightning is far too erratic and unpredictable for absolute protection to be possible.

The traditional way is to mount a pointed copper or aluminum rod 1 to 2 ft (30 to 60 cm) long on the highest part of the vessel to attract and control the strike; the rod can then safely discharge the strike down a path through the bonding system (see later) to a ground plate. This is usually a metal plate attached to the hull below the waterline. Products such as static (ionic) dissipaters decrease the probability of a strike (in theory) by preventing the charge from building on the vessel. They bleed off and remove the lightning-attractive static charge, so that leaders approaching from a storm cloud are not attracted to the masthead (ironically, they also increase the height of the masthead). On a powerboat without a dominant high structure, the dissipater should be mounted on a special mast (air terminal). The grounded air terminal or mast theoretically creates an electrically safe zone called the cone of protection. The apex of this cone is the top of the mast, and the

base is a circle around the boat with a radius equal to the mast height. Without additional precautions, the cone of protection does not guarantee safety from lightning strikes. Theories concerning lightning protection for boats are widely debated, and marine experts do not universally accept concepts such as the cone of protection. Lightning, it appears, can easily curve around the lightning rod, as it did when it curved around the giant launch tower and struck the base of the launch pad of space shuttle *Challenger STS-8* on August 30, 1983, at the Marshall Space Flight Center. (View the NASA photo at http://snrs.unl.edu/amet498/nemunaitis/damage.html. Accessed October 25, 2006.)

To protect the boat from lightning, a lightning bonding system is required to guide the massive energy from the air terminal to an underwater ground in the shortest, most direct route possible. This is especially important for boats constructed from wood, composites, and fiberglass; these nonconductive hulls impede the passage of the electrical charge to the water. Steel and aluminum hulls with traditional aluminum spars are excellent conductors of electricity and can carry electrical charges to the ground with ease. The objective is to prevent injury to the crew, catastrophic damage to the boat, and failure of electrical systems and electronic equipment. The paradox (and controversy) about bonding systems is that by providing a conductive path though the boat, one may invite more lightning strikes, even though the system might minimize the damage. Without grounding, a bolt will find its way to the sea from the base of the mast, usually through the hull. The path to the ground must be of a highly conductive material of low resistance so that the current passing through will not create heat sufficient to melt the conductor itself. The bonding system must be complete. Half measures may invite massive electrical charges into the boat and then fail to provide a safe path to the water; it would be preferable to remove the half measures and sail without a bonding system (other than one exclusively for the electronics). Copper wire with a minimum of 4 American Wire Gauge (AWG)—not 8 AWG, as previously suggested—is required in salt water, and a ground plate ideally made of solid copper or bronze with a dimension of at least 1 square foot (30.5 cm) is recommended. It should be located as close to the mast as possible on the bottom exterior of the underwater hull. An alternative is to install a copper ground strip $^{3}/_{16}$-inch thick, $^{3}/_{4}$-inch wide, and 4 ft long (5 mm thick × 2 cm wide × ~1 m long), running along the bottom exterior of the hull directly under the base of the mast to the bottom of the transom. All ground plates and ground strips should be centerline to remain submerged at all heeling angles.

Lead keels on sailboats make excellent ground plates only when properly connected to the base of the mast and if not encapsulated with fiberglass. Some motor and auxiliary sailboats use the exposed surface of the engine prop and shaft as a ground plate. With engine grounding, damage may occur from the heat generated by a powerful strike. All masts constructed of wood or other nonconductive materials (carbon fiber with epoxy resins) require wire or a solid copper strap from the masthead to the ground plate.

Lightning can also generate a side flash, which is the secondary flow of current from the surrounding charged area to some object near the path the strike follows to the water. This current is especially dangerous to crew who are accidentally near the path to the ground. Simple grounding of the mast to the ground plate prevents major hull damage but does not prevent these side flashes. As the current follows a designated path to the ground, another electric potential is created between the ground system and the objects surrounding it. The entire boat becomes high voltage, and the secondary electric current, the side flash, is created.

The key to preventing secondary current flows is to equalize the voltage of all the metallic objects on board by establishing a common electrical ground for the entire boat; that is, a complete bonding system. Any area capable of collecting a large static charge, each piece of metal equipment on board, and all electronic instruments and radio equipment must be bonded to the same discharge system used to protect the boat from the effects of the initial lightning strike. This is accomplished by connecting copper wire from all metal objects to the common ground. Metal through-hull fittings are also capable of building a static charge and require connection to the ground plate. Box 76-9 outlines the general principles to protect the boat from lightning damage.

Crew members may become part of the current path if they are in contact with, or come between, two different metal objects that are not interconnected (e.g., by grasping the stanchion or rigging while holding an aluminum steering wheel). The best protection in the event of a direct lightning strike, even if the boat is grounded and bonded, is to remain low in the boat (preferably in a cabin) and away from metal objects, wiring, and electric conductors (Box 76-10).

Box 76-9. Protecting Boats from Lightning Damage

1. Install a lightning protection system (LPS), and have it tested for conductivity from masthead to grounding plate by a marine electronic specialist.
2. Install temporary lightning protection for motorized dinghies and open small boats. Battery jumper cables connected to a railing and dropped into the water will help.
3. Keep water out of the bilge, and install a large-capacity manual bilge pump. Pump the bilge dry when a thunderstorm approaches.
4. Place tapered wooden plugs next to all through-hull fittings in case of blowout. Have a diagram of the hull indicating location of all through-hull fittings.
5. Conduct a regular maintenance and inspection schedule. All ground conductors and bonding connections must be clean, secure, electrically sound, and free of corrosion.
6. During a severe thunderstorm, avoid altering the boat's electrical potential profile, such as dropping anchor.
7. Lower, remove, or tie down the radio antenna and other similar upright devices if they are not part of the LPS.
8. In severe lightning storms, unplug all electronics and place computers, cameras, and GPS (global positioning system) units in the oven (it may protect them from lighting) and small items in a covered pot. When practical, design the original installation of electronic instruments inside metal cabinets that are bonded to the lightning bonding system.

Box 76-10. Protecting Crew from Lightning Injury

1. Get out of the water. If possible, get off the water and away from the water.
2. Get off the beach. If stranded, squat down and place legs and ankles together. Keep surface area of the body in contact with the ground to a minimum. Spread people out to maximize the possibility that some will survive a lightning strike.
3. If possible, get off the boat, including boats at anchor or in a marina.
4. Remove wetsuits and other wet clothing. Put on dry clothing and foul weather gear, hat and boots, and a personal flotation device. Wet bodies make good electric conductors, but wet foul weather gear may provide protection by lowering surface resistance and guiding the current around you.
5. Remove all metal articles, especially jewelry, scuba tanks, and weight belts.
6. Avoid direct contact with all metal objects, including handrails, engine, stove, rigging, and spars.
7. Do not touch any of the boat's installed electrical equipment, including navigation instruments and radios. Use a hand-held VHF radio for emergency communications during the storm.
8. Avoid contact with objects connected to the lightning protection system. Do not simultaneously touch two components connected to the system, such as the engine throttle and spotlight handle.
9. Stay away from the mast, stays, shrouds, and wet sails.
10. Stay out of areas where bridging between two highly charged areas, or side flashes, may occur. These areas include the foredeck, between the mast and the head stay, or a seat between an outboard engine and the portable metal fuel tank or steering wheel.
11. If caught in a storm while on a sailboard, lower the sail and mast and sit down on the board.
12. If fishing or trawling, stop and lay the rods and trawls horizontal in the boat.
13. Put nonessential crew and passengers below decks, in the center of the cabin. Stay out of the companionway, engine room, and head. If there is no cabin, stay low in the boat.
14. Place the boat on autopilot to minimize contact with a metal wheel.
15. Avoid activity that may suddenly alter your electric potential profile, such as stepping into a dinghy or stepping onto a beach from a small boat.

Other Weather Phenomena

Waterspouts are maritime tornadoes. Although less common than lightning and downbursts, they are generated by the same dynamic forces found in the squall line at the leading edge of an advancing cold front or in a rapidly building summer afternoon thunderstorm. The danger inherent in a waterspout lies in the powerful revolving winds, which may exceed 250 mph, and the very low pressure at its center, which may cause tightly enclosed spaces to explode. Waterspouts are visible during the day, when the majority of them occur, and can also be located and tracked by radar. The average forward speed is 30 mph, but may vary from nearly stationary to 70 mph. An area of turbulent water in the distance is the earliest visible sign of a waterspout; as it approaches, spray rises upward and joins the funnel cloud with its characteristic snakelike, gyrating appendage. Waterspouts usually last only 30 to 60 minutes. Preparation of the boat and crew is similar to that for a thunderstorm. Because a waterspout is relatively narrow, steering a course perpendicular to its projected path (the direction the clouds are going) is a logical avoidance maneuver.

A hurricane is a type of tropical cyclone: an organized rotating weather system that develops in the tropics. Tropical cyclones are classified as follows:

- **Tropical depression:** an organized system of thunderstorms with a closed low-level circulation and maximum sustained winds of 38 mph (33 knots) or less.
- **Tropical storm:** an organized system of strong thunderstorms with a well-defined circulation and maximum sustained winds of 39 to 73 mph (34 to 63 knots).
- **Hurricane:** an intense tropical weather system with a well-defined circulation and sustained winds of 74 mph (64 knots) or higher.

The Saffir-Simpson Hurricane Scale is a 1 to 5 rating based on the hurricane's intensity. Category 1 has sustained winds up to 95 mph; category 5, sustained winds in excess of 155 mph. In the western North Pacific Ocean, hurricanes are called typhoons ("super typhoons" have sustained winds exceeding 150 mph), and in the Indian Ocean, cyclones. On average each year, 10 tropical storms, six of which become hurricanes, develop in the Atlantic Ocean, Caribbean Sea, or Gulf of Mexico.

The center (eye) of a hurricane is relatively calm. The most violent winds and rainfall are found in the eye wall, a ring of thunderstorms 50,000 ft (15,240 m) high. Coastal sailors seeking harbors of refuge from the destructive wind and sea still have to prepare for the greater threat of the storm surge; this is a large dome of water, often 50 to 100 miles (80 to 160 km) wide, that sweeps across the coastline where the hurricane makes landfall. The surge of high water, topped by huge waves, is devastating. In the summer of 2005, Hurricane Katrina flooded and destroyed cities of the Gulf Coast with its 20-ft storm surge. When given the choice, some sailors head out to sea, risking their lives to save their boats. This may be a reasonable strategy for a battleship, but rarely a prudent decision for an offshore sailing craft. The best tactic for dealing with the fury of hurricanes at sea is to avoid them. Although recommendations include placing the boat in the so-called safe or navigable semicircle and avoiding the strongest winds surrounding the eye of the hurricane, the safest course is to stay out of their path.

In the Northern Hemisphere, the wind blows counterclockwise around the eye. Facing into the wind and stretching the right arm back 120 degrees will point at the eye of the hurricane. In the Southern Hemisphere, the wind blows clockwise around the eye. Facing into the wind and stretching the left arm back 120 degrees will point to the eye. In the Northern Hemisphere, the strongest winds are to the right side of the hurricane's path, where the forward speed over the water adds to the local wind speed; this is the more dangerous semicircle. In the Southern Hemisphere, the strongest winds are on the left side of the path.

Hurricane track forecasting has acknowledged limits and errors, and the U.S. National Hurricane Center is not infallible. Hurricanes are inherently unpredictable, even when the best computer models are used to predict their path. In October 1998, when category 5 Hurricane Mitch (the fourth-strongest Atlantic basin hurricane in recorded history), slammed into Honduras, the 286-ft, four-masted, steel-hulled *Fantome* was doomed. Despite every evasive action taken by the experienced captain (based on the updated forecast track), the massive hurricane swirled menacingly into his path and eventually sank the cruise ship with loss of the entire crew. The plots of the ship's daily locations off the coast of Central America and those of the hurricane exactly overlap each other, as though Mitch was stalking the ship before finally devouring it.

The expected track error for a hurricane is 100 miles (160 km) on either side of the predicted track for each 24-hour forecast period. For a 72-hour forecast, an error of 300 miles to the left or right of the predicted forecast track can be expected, and for 96 hours, 400 miles to either side is applied. That would make the storm's potential swath for destruction 800 miles wide, a considerable area to avoid if a vessel can only travel at 6 to 7 knots in storm conditions. In order to take meaningful evasive action, a hurricane needs to be monitored at least every 6 hours, which is the official forecast interval for the NWS Tropical Prediction Center. The National Hurricane Center in Coral Gables, Florida, provides advisory updates on developing tropical storms and hurricanes 24 hours a day. Voice recordings can be heard by calling 305-229-4483 or online at www.nhc.noaa.gov.

Sea Conditions

In storm-tossed seas, expect larger, more dangerous waves; their height varies, and some can be double the height of the average waves in a given area. Strong tidal currents, common near interisland passages, inlets, canal exits, and river mouths, can interact with wind-driven waves to produce high waves and perilous conditions. The same concept applies to adverse currents. When a current's speed approaches or exceeds 25% of the speed of oncoming wind-driven waves, the current stops the wave energy from moving forward; wave energy builds vertically until the steep waves begin to break, endangering small craft. Check tide and current tables in the regional *Nautical Almanac* to calculate the optimal time for safe passage by coordinating the local weather with the predicted currents. Try to avoid sailing parallel (beam-to) to high breaking seas; the curl of a plunging breaker can easily capsize a boat. Capsizing may occur in seas that are not exceptionally high. On Dec. 21, 2004, the 75-ft *Northern Edge*, with six fishermen aboard, capsized off the New England coast in a snow squall with 15-ft seas and 35-mph winds (an average North Atlantic winter storm). The lone survivor said, "We were dredging (for scallops) . . . then came a wave and the boat was hit broadside, and it just flipped." The bodies of the other five fishermen were never recovered from the 40° F (4° C) water after the U.S. Coast Guard searched a 1,850-square-mile area for more than 40 hours. None of the fishermen were wearing survival suits, and according to a preliminary investigation, the watertight door to the engine room and forward compartment was open, but the scuppers to the main deck (the openings along the deck that allow water to escape) were closed!

EMERGENCY COMMUNICATION AND DISTRESS SIGNALS

Cellular Telephones

Sailors frequently use cellular phones rather than marine radios to call for assistance. A variety of emergency services, including regional Coast Guard stations, can be called directly, and in some coastal areas, the Coast Guard can be accessed immediately by dialing *CG.

The cellular phone has several disadvantages when used in search and rescue (SAR). Currently, 10 to 20 miles (16 to 32 km) offshore is the average effective range of cellular phones; range is determined by line of sight to the cell antenna. Therefore, use is restricted to high-traffic areas where there are both cellular antennas and relay stations. Gaps in transmission make them unreliable even for coastal use. Cellular communication is private, in contrast to the more public (party line) broadcast over VHF radio; it excludes potential rescuers who might be in the immediate vicinity. In SAR operations, no practical way exists to maintain continuous communication with a number of rescue craft via cellular phone. The Coast Guard is unable to use a radio direction finder to locate a cellular signal, as it can with the VHF-FM signal.

Reception and transmission of cellular ultra–high-frequency (UHF) signals can be strengthened by using an external antenna placed as high as possible on the boat or by standing in the highest location, such as the cabin top. Signal strength can also be improved by reprogramming the phone for the best reception in a specific location using wireline (B-side) or nonwireline carriers.

If a cellular phone is the only communications link on board, extra charged phone batteries and a waterproof pouch are needed. A comprehensive list of emergency phone numbers should include local hospitals and physicians, regional Coast Guard stations, harbormasters, and maritime towing services.

Similar to the cellular phone, but utilizing low earth-orbiting (LEO) satellite constellations, are the Globalstar and Iridium communication systems. Using a hand-held transceiver reminiscent of the original bulky mobile phones, these telephones can communicate from virtually anywhere in the world (Iridium) or from major landmasses and coastal areas (Globalstar) to any phone in the world. These are portable handsets, requiring a clear view of the sky to operate (the antenna must be visible to the satellite), or they may be used with a docking station and remote external antenna. Cost runs approximately $1.50 per minute. When the mast comes down and the antennas for the SSB and VHF are lost, a satellite phone may be lifesaving, especially when help is out of range of the hand-held VHF radio.

Marine Radios

The VHF-FM radio transceiver is the most popular, user-friendly, inexpensive, and reliable form of marine communication. It is the single most important radio system you can buy. It is easy to use, even for crew with minimal experience (Box 76-11). The VHF signal range is limited to line of sight and therefore depends on the height of the transmitting and receiving antennas. A transmission range of 15 to 30 miles can be expected between boats having masthead-mounted antennas.

A simple formula to calculate the maximum line of sight between two antennas is to add together the square roots of the

Box 76-11. Operating Instructions for VHF Marine Radio

Turn power on and adjust the volume. Turn the squelch control knob counterclockwise until static noise is heard, and then clockwise until the noise is quieted.

1. Transmit distress calls on VHF channel 16. All sets have a designated button that immediately selects this channel. Switch to another frequency if instructed to do so.
2. Set the transmit power to high (25 W). Use low power (1 W) to contact nearby boats, which will conserve the ship's battery and reduce interference with distant vessels.
3. Depress the call button on the microphone to transmit and keep it depressed throughout the message. Release the button to hear the response. You cannot transmit (talk) and receive (listen) at the same time.
4. Speak in a normal voice and normal volume. It is unnecessary to shout. Speak slowly and distinctly. Keep text simple and terse. Keep mouth close (1 inch [2.5 cm]) to microphone.
5. Know or estimate the boat's location before commencing transmission. Refer to any recognizable landmarks on shore or preferably, the position from a GPS (global positioning system).
6. Establish radio contact before the crisis is fully developed and while electronic equipment is functional.
7. Use the appropriate alerting signal. "Mayday" is the top-priority distress signal if the vessel or crew is in imminent danger and requires immediate assistance. Urgent messages relating to non–life-threatening emergencies are prefaced with the signal "pan-pan" (pronounced pahn-pahn). Seeking medical advice for a sick or injured crew would fall into this category.

If there is no response, wait 5 minutes with the radio turned on, and repeat according to the probability of other ships being in the area.

two antenna heights (in feet), then multiply by 1.17 to find the distance in miles. Two sailboats with antennas at 40 ft can theoretically communicate, under optimal conditions, up to 14.8 miles apart. The Coast Guard places antennas along the coast, connects them with land lines to their operations centers, and is able to monitor and communicate with all vessels that have a masthead-mounted aerial within 20 to 25 miles (32 to 40 km) of the coast. The range may extend up to 300 miles (483 km) when communicating with aircraft.

The VHF radio is limited to near-shore, ship-to-ship, and ship-to-shore communication. Communication is not private, which is a distinct advantage in maritime emergencies and SAR operations. VHF is the open party line connecting all vessels within the signal range. Any boats in the area monitoring distress channel 16 (which they automatically do when the radio is initially turned on) will receive the distress call. Rescue boats and aircraft are equipped with radio direction-finding equipment that can precisely locate a boat's VHF-FM signal.

Cruising boats should have both a fixed mounted radio wired to the ship's electrical system and a portable hand-held VHF radio (Fig. 76-6). After a knockdown or capsize, a hand-held radio can operate independently of the ship's radio antenna (the mast may be lost or the antenna damaged). It is indispensable in SAR operations and helicopter evacuation, where one is required to be mobile and on the deck. Hand-held radios can be used to communicate with the mother ship from a dinghy, and are essential for communicating with rescue personnel from the life raft.

Figure 76-6. Waterproof VHF radios can make an effective *crew overboard* alarm, by allowing the victim to contact the mother ship or other vessels in the vicinity.

During an emergency, use the more powerful, permanently installed radio for the initial distress call. In the spare parts inventory, include an emergency antenna that can be inserted into the VHF radio jack to allow short-range communication should the boat be dismasted (the same problem can befall a powerboat if its antenna is broken).

The range of the portable unit is up to 5 miles (8 km). To conserve batteries, the user should transmit only when a ship is in sight and whenever a message is received (even if the calling ship is not in sight). Transmitting uses 15 times more battery power than does receiving; transmitting at full power uses dramatically more power than at low (1 W) output. Although range is less than a high-power fixed-installation VHF radio, reliable communication can be established with any visible aircraft or vessel.

The new multichannel VHF survival radios are ideal for life raft or lifeboat use: They are waterproof, submersible to 10 ft (3 m), and float. The antenna, circuitry, and power are tuned for maximum range. The operating life with the lithium battery is a minimum of 8 hours. The basic waterproof and submersible VHF radios sink, so place them in a foam flotation case for protection.

The most important channel on the VHF band is channel 16, the distress and safety frequency (156.8 MHz). This calling frequency is used to initiate contact between any two vessels and is the only frequency constantly monitored by the Coast Guard. When a radio is not active on another channel, it should be left eavesdropping for distress calls on channel 16. Box 76-12 lists other VHF-FM channels often used by pleasure craft.

Be advised, there are still coverage gaps ("dead zones") in the Coast Guard's National Distress System. That means that in approximately 88 locations along the coastal continental United States, mayday calls may not be received by the Coast Guard.

Box 76-12. Useful Marine Channels (VHF Radio)

06	Intership safety communications
09	Boater calling. Use this to initiate contact with other boats, and ship to shore (hailing channel). Commercial and noncommercial.
12	Port operations, traffic advisories
13	Intership navigation safety (bridge-to-bridge). **Use this channel to contact a ship when there is danger of collision.**
14	Port operations and some Coast Guard (CG) shore stations
16	International distress, safety, and calling
22A	Working CG ships and shore stations, CG marine information broadcasts
24 to 28	VHF marine operator
68 to 69	Noncommercial intership and ship to coast
70	Digital selective calling (voice communications not allowed)
71	Noncommercial ship-to-ship and ship-to-coast
72	Noncommercial intership only
78A	Noncommercial intership and ship to coast
WX 1 to 7	NOAA Weather radio broadcasts on 162 MHz

NOAA, National Oceanic and Atmospheric Administration.

A new system with greater capability is currently being built. It will include channel 16 and add channel 70 Digital Selective Calling (see later). It is expected to be completed by 2006. At that time, a sailor within 20 miles (32 km) of the coastline transmitting a distress call on low power (1 W) will have a 98% chance of being heard.

When receiving a mayday call, listen and do not interrupt. If the Coast Guard or another vessel does not respond, then you must answer. Acknowledge the mayday call as follows:

- Repeat the name of the distressed vessel three times.
- Transmit your name three times and then say, "Received mayday."
- Listen to see if any other vessel is closer and preparing to offer assistance. If not, recontact the vessel in distress and give your position and estimated time of arrival.
- If you are able to contact the Coast Guard or another ship in the vicinity, transmit "Mayday-relay, mayday-relay, mayday-relay," then give your vessel name and location and the information you have received.

For communication beyond the VHF range, a more powerful and elaborate SSB radio transmitter is required. Medium-frequency/high-frequency marine radio-telephone equipment operates between 2 MHZ and 26 MHz using single sideband emissions. This equipment can also be used to receive high seas weather broadcasts (see discussion on thunderstorms and weather) and, in combination with a laptop computer and a special HF modem, can provide an easy and relatively inexpensive way to send and receive e-mail. The four principal SSB e-mail system providers for the recreational market are CruiseEmail, MarineNet, SailMail, and SeaWave. Each provider uses a different software package with the Pactor II modem. E-mail is not just for social exchanges; it offers cruising boats a safety advantage for communicating safety-related data to boats around the world. Depending on the radiofrequency band and atmospheric conditions, communication range may be several thousand miles.

The international distress and calling frequency is 2182 kHz, located in the medium-frequency range. The frequency 2182 kHz is used for distances from 20 to 100 miles day or night. The frequency 2670 kHz is designated for distress and safety communications with the U.S. Coast Guard. On the high-frequency bands, the frequencies 4125 kHz (Channel [Ch.] 450), 6215.5 kHz (Ch. 650), 8291.0 kHz (Ch. 850), 12,290.0 kHz (Ch. 1250), and 16,420 kHz (Ch. 1650) have all been designated for distress and safety calls. The high-frequency transceivers can call and receive voice and digital communications to and from anywhere in the world.

The best way to select an optimum emergency frequency is to listen to the quality of a radio broadcast. A station that your radio receives loud and clear will also provide good reception for your broadcast at that time. SSB is an excellent receiver for voice weather and weatherfax broadcasts, as discussed previously.

WLO Marine Radio in Mobile, Alabama, is currently the sole provider of worldwide ship-to-shore SSB high-frequency radiotelephone service in the United States. HF radiotelephone service is now available from their radiostations on the West Coast. Complete information regarding their radiotelephone channels can be obtained by calling 251-666-5110 or through their Web site at www.wloradio.com.

Global Maritime Distress and Safety System

Since February 1, 1999, ships and coastal stations have not been required to monitor the traditional distress frequencies, such as VHF channel 16 or 2182 kHz. Until the new Global Maritime Distress and Safety System (GMDSS) is implemented, the Coast Guard and many recreational and commercial ships will continue a radio watch on these frequencies. A complete transition to the new GMDSS is not expected until at least 2007. The GMDSS is a worldwide infrastructure, controlled from a shore-based communications center, to coordinate the assistance of vessels in distress. This fully automated system uses satellite and digital communication techniques. New radio equipment and communication protocols are required. GMDSS simplifies routine communications at sea and facilitates regular weather forecasts, navigation warnings, and distress relays in the form of maritime safety information (MSI). Digital selective calling (DSC) technology permits a VHF radio to call another radio selectively using digital messages, similar to the modem on a computer. As with a direct-dial telephone call on land, only the vessel called receives the message. The usual VHF party line connecting boats in the area capable of monitoring the message is eliminated. Every vessel has its own unique maritime mobile service identity (MMSI). The vessel's MMSI is manually coded into the DSC radio. Part of that number may characterize a particular group, enabling calls with MSI to alert everyone in the group. The digital information is carried on a designated channel. For VHF radios, channel 70 (156,525 MHz) is authorized exclusively for distress alerts, safety announcements, and calling using DSC techniques. No other uses are permitted. The digital signal is able to carry other essential information, such as the caller's identity, location, and nature of the call (see Box 76-12).

Distress messages can be sent automatically. The vessel's identity is permanently coded into the unit, and its position can be determined from the data output of a GPS receiver linked to the radio. On a DSC-equipped radio the receiver sounds an alarm

Box 76-13. How to Make a Distress Call at Sea

Tell them who you are, where you are, and what is wrong:

1. "Mayday, mayday, mayday" or "Pan-pan, pan-pan, pan-pan"
2. Name of vessel in distress (spoken three times) and call sign or boat registration number, spoken once
3. "We are located. . . ." Report position data: latitude and longitude (from GPS), bearings to landmarks, vessel's course, and speed and destination.
4. "We need assistance. . . ." Specify nature of distress (e.g., injury, fire, flooding).
5. "The boat is. . . ." Provide full description of craft (type, color, size) with any distinguishing characteristics.
6. "There are ___ people on board."
7. "This is (repeat boat's name) standing by on channel (16 or 2182)."
8. End message with "Over."
9. Repeat this call and listen for a response between transmissions. If no one responds to your mayday call, activate your EPIRB!

EPIRB, Emergency position–indicating radio beacon; GPS, global positioning system.

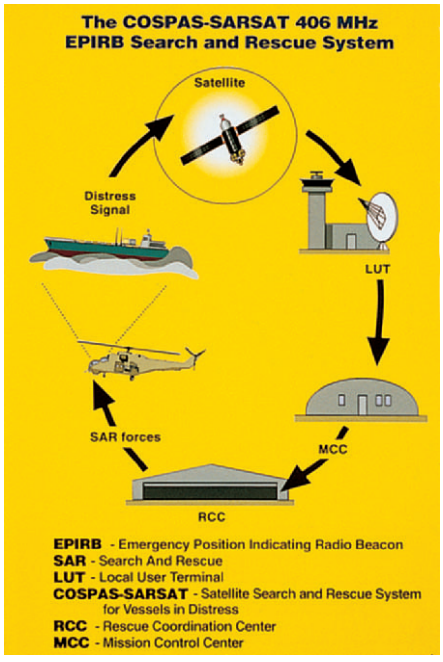


Figure 76-7. Anatomy of a rescue. The 406-MHz emergency position–indicating radio beacon (EPIRB) sends a digitally coded signal to a low-Earth-orbit satellite, which relays the signal to a nearby ground receiving station (LUT). The location of the EPIRB is calculated and forwarded to the nearest mission control center (MCC), which matches the EPIRB user's registration with the specific signal. These data are relayed to the local rescue coordination center (RCC), which initiates the search and rescue. (Courtesy ACR Electronics.)

if it receives an "all ships call" (distress or otherwise), group call, or call specifically to that vessel. All other DSC calls are ignored.

A distress alert (equivalent to mayday) can be sent automatically as a digital signal to a shore-based rescue coordination center (RCC) covering the area. Once the alert has been sent, the radio will automatically repeat the call at intervals between $3^1/_2$ and $4^1/_2$ minutes until the RCC acknowledges receipt of the message on channel 70. Subsequent communication should continue on channel 16 or another selected frequency for voice transmission. A verbal mayday can also be sent immediately on channel 16 after the first alert. Some DSC-equipped transceivers can listen simultaneously on channel 70 for DSC and channel 16 or other channels for voice transmission. A verbal mayday follows the same sequence as outlined in Box 76-13 and includes the MMSI as part of the vessel's identification.

To make a ship-to-ship or ship-to-shore call on DSC, the nine-digit MMSI number of the station to be called must be keyed in on the radio together with a proposed working radio telephone channel for voice communication. This announcement is transmitted on channel 70 and should be acknowledged on channel 70. Four VHF channels are available for special use within the GMDSS: channel 06 is for SAR coordination, channel 13 for intership safety of navigation (bridge to bridge), channel 16 for distress and safety, and channel 70 for DSC alerting.

The GMDSS also utilizes satellite communication links such as the International Maritime Satellite Organization (INMARSAT M) to provide long-range global communication. Geostationary (geosynchronous) INMARSAT satellites cover the whole world between 70° north and 70° south latitude (there is no coverage at the poles, and spotty coverage in the Southern Ocean). The satellites pass over the equator at an altitude of 22,500 miles with an orbital period of 24 hours, allowing them to remain stationary over fixed points on Earth. They provide high-quality worldwide speech (voice is full duplex, just like a telephone), data, and facsimile communication as well as reliable distress alerting and follow-up communication within the GMDSS. Presently, the size of the equipment and power consumption make this system unsuitable for smaller recreational craft. INMARSAT Mini-M uses small units with tracking antennas. The radome designed for smaller commercial boats and yachts over 45 ft (14 m) is 12 inches (31.5 cm) tall, weighs a mere 15 lb (7 kg), and requires a clear view of the sky.

INMARSAT C is the gold standard for global marine data communication. It offers digital data messaging (no voice) and is the only two-way marine satellite data message system approved for GMDSS safety at sea. The hardware is small enough to fit in any boat. It can send a distress signal at the touch of a button and can easily be integrated with a GPS receiver. Pressing the distress key sends a message with the ship's identity and location to the nearest RCC.

Emergency Beacons

Emergency position–indicating radio beacons (EPIRBs) are hand-held portable radio transmitters that can transmit signals interpreted as mayday calls (Fig. 76-7). These signals are the satellite-linked equivalent of a 911 call for mariners in distress. In the absence of a marine radio, EPIRBs are the most important piece of signaling equipment. An EPIRB should be used only as a last resort when all other means of communication have failed, and when there is a life-threatening emergency. It should be located in a readily accessible location.

EPIRB signals are transmitted on established distress frequencies 121.5/243.0 MHz and 406/121.5 MHz. The signals are monitored by the global COPAS-SARSAT (search and rescue

Figure 76-8. Personal locator beacons (PLBs) are now small enough to be carried by each crew member.

satellite-aided tracking) satellite system coordinated by the United States, Canada, France, and Russia. This system is a constellation of polar orbiting and geostationary satellites fitted with transponders to receive the distress signal and locate the beacon. The polar satellites orbit 600 miles above the earth and have an orbital time of 105 minutes.

The first-generation class A and class B beacons, which transmit on 121.l/243.0 MHz, can also be monitored by SAR ground stations, aircraft, and ships with appropriate receivers. Satellite monitoring of these older beacons is scheduled to be discontinued in 2009, and most dealers have stopped selling them.

The second-generation 406-MHz EPIRB provides the most reliable worldwide coverage. Satellites with 406-MHz transponders can store the signal in memory until a ground station is in view and then can retransmit the signal. The distress signal is quickly relayed to a ground station called a local user terminal (LUT), passed on to the mission control center (MCC), and relayed to the appropriate maritime rescue coordination center (MRCC). The satellites are able to compute the position of the beacon within 2 km. The 406-MHz EPIRB transmits a digital signal with a unique identification code, which can be instantly identified through a NOAA encoded-transmission program. If the unit is properly registered with NOAA, vital information regarding the vessel can be passed on to SAR units. All registered EPIRBs are issued a dated decal that provides proof of registration and includes a unique 15-character hexadecimal code, registration expiration date, and the vessel's eight-digit registration code.

As of July 2003, boaters can also select a beacon primarily intended for wilderness use, called a PLB or personal locator beacon (Fig. 76-8). PLBs have several drawbacks compared with EPIRBs, including half the battery life and inability to float upright. Their benefits include being compact and less expensive; some have user-replaceable batteries to lower the cost of ownership.

Failure to register the EPIRB can lead to unnecessary and tragic delays. NOAA receives many EPIRB signals daily, some in error and some justified. Signals are processed by computer and matched by updated information provided biannually by the owner. Signals from unregistered EPIRBs are considered questionable and are not given top priority. The 406-MHz EPIRB is now required aboard all commercial vessels operating at or beyond 3 nautical miles from shore.

The newest generation of EPIRB is the 406-MHz unit with a GPS interface or a built in GPS receiver. The GPS-enabled beacon (also called a self-locating beacon) transmits its exact location within 100 meters, using GPS-derived latitude and longitude coordinates, along with the EPIRB signal. It utilizes polar orbiting satellites and new geostationary search and rescue (GEOSAR) satellites in high earth orbit. The GEOSAR satellites receive the signal as soon as the beacon is activated. Position information is continually updated and stored every 20 minutes in the unit as long as it maintains a direct connection to the GPS receiver. The battery has a minimum operating life of 48 hours (like all EPIRBs) and operates longer in temperate climates.

All EPIRBs have a homing signal at 121.5 MHz. This is the homing frequency used by SAR vessels and aircraft equipped with radio direction-finding equipment to locate the craft in distress. Once activated, the EPIRB should be left on until the emergency is over; to update the position, it must broadcast continuously. EPIRB radio signals cannot penetrate water, wood, metal, or fiberglass (the unit must be outside the cabin), but the signals will be received when transmitted from inside a life raft, on deck, or on the water's surface. All EPIRBs are required to transmit for 48 hours at –40° C (–40° F).

Many cruising sailors believe a 406 EPIRB alert will bring rescuers in a matter of hours, or at most a couple of days. These expectations are unrealistic in many parts of the world. Although the SARSAT system and RCCs that support 406 EPIRBs save more than 1000 lives each year, the system is far from perfect. Many nations responsible for RCCs covering specific areas have failed to ratify the Search and Rescue Convention of 1979, and even after signing, lack the resources to meet their obligations under the guidelines. They are not equipped to carry out effective SAR operations in their designated area, and understandably, SAR may not be a top priority for the planet's poorest countries.

On June 8, 2002, when the 32-foot Down East cutter *Leviathan* went down in a storm off the island nation of Madagascar, a 6-hour EPIRB signal was not picked up by the RCC in Madagascar; the rescue center is not available on a 24-hour basis. Rescue ships and planes were not immediately diverted to the beacon's location, and 10 days passed before an air search was launched in the Western Indian Ocean. By then, the task was futile; the cruising couple and lifeboat were never found.

Potential delays justify carrying a second 406-MHz EPIRB when cruising in remote areas and far offshore. While the EPIRB is activated, continue signaling by all the other methods available. EPIRBs with lithium batteries have a shelf life of 10 years, making it feasible to store one in the life raft. If a single EPIRB is carried on a vessel, it should be packed into an abandon-ship bag or other storage area for safety gear, unless it is mounted in a special bracket for automatic activation and deployment.

Visual and Sound Distress Signals

In an age in which we are dependent on electronic navigation and sophisticated electronic and satellite communication, the simplest signaling strategies and devices are often overlooked (Box 76-14).

Visual pyrotechnic distress signals have a high failure rate and age rapidly, so they require replacement according to the labeled expiration date.

Box 76-14. Visual and Sound Distress Signals at Sea

1. If other vessels are in sight, stand in an unobstructed area and slowly and repeatedly raise and lower outstretched arms as though you are flapping wings.
2. Sound a foghorn (SOS: 3 short blasts, 3 long, 3 short), ring a ship's bell, or blow a whistle continuously.
3. At night, point a flashlight at another boat and flash SOS (dot dot dot, dash dash dash, dot dot dot) repeatedly.
4. Fly the ship's ensign upside down, or fly the recognized distress flag: an orange flag with a black square under a black ball. Any square flag with a ball shape above or below is also a recognized distress signal.
5. Wave any brightly colored clothing or foul weather gear attached to a paddle or pole.
6. Use controlled flames or smoke on the vessel, or pour a dye marker in the sea near the vessel.

Hand-held flares have varying effectiveness for attracting attention during daylight (Fig. 76-9). Luminosity ratings range from 500 to 15,000 candlepower. The lowest rated flares are virtually invisible in daylight at $^1/_4$ mile (0.4 km), and the highest rated flares are only slightly more visible. For daytime use, orange smoke devices are the most effective way to attract attention. Smoke canisters are superior to hand-held smoke flares. They float and emit orange smoke for 2 to 5 minutes. Immediately after ignition, throw the canister in the water downwind of the craft. Smoke signals have a visible range of 1 to 3 miles (1.6 to 5 km) in daylight, depending on the wind. Helicopter pilots find smoke signals especially useful to indicate the strength and direction of the wind at sea level (Fig. 76-10).

After sunset, consider a flare's luminosity, burn time, and attained altitude. Use an aerial flare (meteor or parachute) to attract attention at night, and then ignite a hand-held flare to guide the rescue craft to your location. Due to the curvature of the earth, sighting distances are limited. A high-altitude flare enables a ship beyond your line of sight to be alerted. The greater the height, the longer and farther the signal can be seen by a distant viewer. A parachute flare at 1000 feet (305 m) is seen as a brief flash of light on the horizon from 40 miles (64 km) away, but from 20 miles (32 km), it appears to be 500 ft (152 m) above the horizon and is visible longer. Luminosity and burn time are more important than altitude to help assisting craft home in on the ship's location. The rated visible range assumes a ship in the area has an alert lookout standing on the bridge watching for and anticipating your signal, on a clear night with calm seas.

Whenever possible, choose flares meeting SOLAS standards (International Convention for Safety of Life at Sea). They exceed U.S. Coast Guard standards and are much more visible. The flares are easy to ignite, do not require pistol launchers, are waterproof, are safer to use, and have the lowest failure rate.

All pyrotechnics are hazardous. They can melt life-raft rubber and burn skin. Hand-held red flares may drip considerable ash and slag; point them downwind while holding them high, at arm's length, away from the raft or boat deck, and over the water. Be in a stable position in the raft or boat when igniting a flare. Remove all adhesive fastenings and covers on the flare and be sure to identify the end from which the rocket or flare exits. After the signal is activated, turn away from the intense glare to protect the eyes. If a rocket or flare fails to ignite immediately, it may not ignite at all or there may be a lag time before firing. Hold the flare and count to 60. If there is still no ignition, throw the flare into the sea. Never look into a flare tube to see why it failed, and never put it back in the raft. Treat it as an activated and live explosive.

Figure 76-9. Hand-held flares are reasonably effective during the day, and dramatic at night. They also produce white smoke that can be distinctive for aerial searchers.

Figure 76-10. Coast Guard rescue swimmers carry multipurpose pyrotechnics, including a very effective smoke flare, to show wind direction and to make their own recovery easier for air crews.

Figure 76-11. Pyrotechnic signals (flares) need to be appropriate for the time of day and the distance offshore.

Parachute flares are designed to attract the attention of potential rescuers out of sight over the horizon. For practical purposes, a parachute flare has a useful range of about 10 miles (16 km) regardless of its rated range or altitude (they go up 1000 ft [305 m]). These flares drop slowly beneath a nonflammable parachute and burn for 45 seconds. The SOLAS models burn the brightest and longest. Red is the distress signal, but a white parachute flare provides far more illumination than a red one. The same is true for meteor (rocket) flares. They should be fired downwind at an angle just slightly less than vertical. The main body of the rocket is contained within the launch tube. After activating the firing mechanism, hold the tube with both hands, with gloves or a dry cloth, to prevent the tube from slipping due to recoil.

Red meteor (aerial) hand-launched or pistol-launched flares (Fig. 76-11) should be also launched downwind approximately 80 degrees above the horizon. These flares are less effective than parachute flares because burn time is only 5 to 9 seconds. It is best to fire them in sequence, the first to attract attention, the second about 10 seconds later to confirm distress and general position. Due to their generally lower altitude and brightness, visibility is limited to 3 to 5 miles (5 to 8 km) at night. Because of these limitations, these flares are best suited for guiding nearby rescuers to the boat, after it has been spotted.

Do everything possible to attract attention with signal mirrors, flashlights, kites, or any other means before using flares. Do not fire flares in the direction of high-flying commercial aircraft. They cannot see your signal when they are flying at 30,000 ft (9144 m). Do not waste all the flares when the first ship or low-flying aircraft appears, because it might pass close by without seeing your signals. Flares are labeled with expiration dates; keep them on board for a while, then schedule a time to practice using them (notify the Coast Guard and local authorities).

Signal mirrors are inexhaustible devices and more effective than flares in daylight. Mirrors are especially useful when trying to attract the attention of aircraft, even those flying at high altitudes. The new $10 Hot Shot signal mirror features a holographic red dot viewing spot in its center, allowing the user to align the sun with the target; this ensures that the reflection, created by a telescope-quality mirror, is in fact reaching those who may see it. Aim the signal at the front end of the fuselage while the airplane is still in the distance and coming toward your vessel. The flash from a signal mirror can be seen up to 40 miles (64 km) from the air on a clear day. If a plane is heard, begin signaling with the mirror, because the aircraft can see the flashing light before the plane comes into view. Mirrors are also useful at night. Like reflector tape, they reflect back a ship's searchlight, making the raft or ship easier to locate in the dark. Utilize the entire crew to improvise signals with other reflective materials, such as compact discs, foil space blankets, jewelry, eating utensils, fishing lures, and credit cards. The sea rewards resourcefulness!

Coast Guard rules state that every boat must carry a device capable of producing a 4-second blast audible from half a mile (800 m). This requirement can be met with chemical-propellant horns (that can damage ozone), lung-powered horns, and whistles. Plastic horns threaded into a metal canister often malfunction. Have at least two aboard, with a big inventory of spare canisters. Virtually all hand-held horns (tested by *Practical Sailor* magazine in 2002[5]) are barely audible at half a mile and are inaudible at three-quarters of a mile (1.2 km). The same is true for lung-powered horns and whistles, which are barely audible at a quarter of a mile (400 m). In the absence of a working sound device aboard ship, try striking the inside of a large pot with a metal spoon or winch handle to create a continuous, albeit crude, bell-like sound.

Rescue ships and aircraft use radar to locate a life raft or vessel in distress. Visibility on the radar screen can be improved by using an electronic radar reflector known as a search and rescue transponder (SART). Once activated by an incoming radar signal (the interrogating radar), it is capable of sending back an electronic signal to any commercial radar located on a ship within a 10-mile radius and up to 50 miles (80 km) on the radar screen of an aircraft flying at 3000 feet (914 m). It is much more effective than a simple radar reflector. When activated by interrogating radar, a line of 12 intense blips will appear on the radar screen radiating outward from the position of the SART. As the search vessel approaches, these blips widen to form a series of concentric circles around the transponder's position on the screen. In standby mode, the batteries have an operating life of 96 hours; in the operation mode, battery life is more than 8 hours. As with a radar reflector, a SART should be mounted at the highest possible point. On a raft, the telescopic pole normally provided or the man-overboard pole can be used as a mast to support the SART. Alternatively, it can be lashed to the top of the raft. If a radar reflector is supplied with the life raft, it should not be deployed at the same time, because it will block some of the SART signals.

Fluorescent dye marker can be seen by airplanes and has a daytime visibility of 2 to 5 miles. In moderate weather, it will last for half an hour; in rough weather, it will rapidly dissipate. Use it whenever an aircraft is expected or the raft is beneath a known flight path. The sea anchor will keep the drift rate the same as that of the dye.

Hand-held waterproof strobe flashers offer portable, compact signaling and can flash for hours on a single replaceable battery. Although not an internationally recognized distress signal, they are effective when supplemented with other recognized distress signals.

CREW OVERBOARD

Personal Flotation Devices

Annual recreational boating fatality statistics underscore the need for boaters to wear personal flotation devices. In 2003, for example, 481 (70%) of 703 recreational boating fatalities were due to drowning.[7] The vast majority of these cases involved open motorboats, PWCs, canoes, and kayaks, whereas only seven involved sailboats. It is estimated that at least 86% of these deaths could have been prevented if a PFD had been worn.[7]

Drowning often occurs in less than three minutes. Sudden immersion in water below 15° C (59° F) is an equalizer for all immersion victims, regardless of swimming ability. In the United Kingdom, it was observed that half of the open cold water immersion deaths occurred within 10 ft (3 m) of safety; two-thirds of persons who died were considered good swimmers. Statistics suggested that a rapid-onset incapacitating response, termed the *cold shock response,* prevented the individuals from successfully swimming 10 ft to save their lives. This incapacitating response is characterized by a series of cardiovascular and respiratory reflexes that peak in 30 seconds, last 3 minutes, and increase the risk of drowning (see Chapter 68). If the victim survives the first few minutes, the short-term response evolves over the next 3 to 30 minutes. Muscles, nerves, and joints malfunction. There is loss of muscle strength, coordination, manual dexterity, and overall muscle performance leading to asynchrony of the swim stroke and breathing (swimming failure). Ability to maintain airway freeboard (distance from mouth to water) is compromised, increasing the risk of aspiration and drowning. Immersed victims cannot perform self-rescue skills, such as clinging to lines, climbing into life rafts, activating rescue flares, and climbing up the cargo net or the Jacob's ladder of the rescue boat. Victims are unable to assist in their own rescue. These lethal responses to cold water immersion can be prevented by donning a well-insulated, fully zipped survival suit (see later), a drysuit, immersion suit, or even a wetsuit.

If one has to enter cold water voluntarily (e.g., to free a fouled propeller) without the protection of a specialized suit, take the following steps in anticipation of the cold shock response: Improvise an immersion or wetsuit by dressing in a foul weather suit and snugly wrapping the ends of the sleeves, legs, and waist with duct tape. Wear thermal underwear or insulating fleece under the suit. Stockings, gloves, and a hat will complete the outfit. Pour warm water slowly through the open collar and saturate the clothing covering the torso, then seal the neck. Put on a PFD and safety harness, with the tether held by an alert crew member. Enter the water slowly, and feel the water seep through all the openings. When breathing is under full control, proceed with the task.

Few boaters routinely wear PFDs, and most people put them on only in storm conditions. The main reasons for veteran sailors not wearing PFDs are discomfort and inconvenience. All the conventional type I offshore life jackets and type II nearshore life vests are bulky, uncomfortable, and awkward to wear; they are too warm in the summer, and limit mobility. The common type III vest is comfortable and wearable, but has poor reserve buoyancy and freeboard (distance from the water to the mouth); it cannot turn the unconscious victim face up (righting ability) and cannot support the head (body angle). It is suitable only for calm water and should be worn only by active boaters who are able to swim. PFDs should be selected according to comfort and practicality in order to maximize compliance. Nonswimmers, children, and inexperienced crew should wear PFDs at all times whenever on deck. Everyone on deck should wear a PFD in heavy weather, at night, or when sailing in water colder than 60° F (15° C). In warm waters, experienced boaters who are strong swimmers must acknowledge a degree of risk by not wearing a PFD. Inflatable PFD vests (on the market for two decades) have a flat and lightweight design that allows them to lie flush against the body so they do not restrict movement. They have excellent wearability and superb flotation capabilities. The vests provide as much as 35 lb (16 kg) of buoyancy, compared with $22^1/_2$ lb (10 kg) in type I and $15^1/_2$ lb (7 kg) in type II and III. In most cases, the added buoyancy enables the person to float higher, making it easier to breathe, and reduces the risk of aspirating seawater in rough sea conditions. By keeping the head and chest higher out of the water, it is easier to adopt the heat escape lessening position (HELP; see Chapter 6). With buoyancy high on the chest, there is also better righting ability and head support. The offshore model can also be purchased with an integral safety harness (Fig. 76-12).

Figure 76-12. Comfortable, lightweight, and convenient integrated safety harness and water-activated inflatable life vest. It is Coast Guard approved and features both a backup rip cord and an oral inflation system.

The U.S. Coast Guard has approved a wide variety of inflatable life jackets, including models listed as type I, II, III, or V. Versions are available with different buoyancies (16, 24, 35, and 60 lb), with and without a safety harness. Autoinflation vests should be worn at all times by nonswimmers, because they may panic after falling overboard; children must be older than 16 years and weigh more than 80 lb (36 kg) to wear these vests. Even strong swimmers should consider wearing an automatic/manual model. Head injury from a surprise fall or an inadvertent jibe might render a person unconscious and leave him face down in the sea with a deflated manual vest. The newest models have inflators that have a single point indicator to show if the vest is armed with an unused CO_2 cylinder. It provides an almost foolproof method for the user to determine

whether the PFD is properly charged and ready for use. The new inflators will not deploy unless the wearer is immersed in water and will not be activated by spray, humidity, rain, or a rogue wave boarding the boat, as some older models have done. All automatic vests have a manual back-up ripcord for inflation and a second back-up oral inflation tube.

Like all mechanical systems on board ship, inflatable vests require regular inspection and maintenance according to the manufacturer's instructions. Every season, orally inflate the vest (after removing the CO_2 cylinder and autoinflation device) and leave the vest inflated for 24 hours to check for leaks. Inspect the water soluble bobbins and replace them as scheduled. Conduct an in-water test to experience the righting ability of the vest. Each time a vest is used, unscrew the CO_2 cartridge to make sure the seal is not punctured. Know how to rearm an automatic vest, and how to repack it according to instructions. Care for your PFD as though your life depended on it.

Boaters who resist wearing any type of vest may tolerate an inflatable vest packed in a compact belt pack. Worn around the waist, it is the least cumbersome of any PFD. After inflation in the water, it must be pulled over the body while the swimmer works to stay afloat; it offers 22 to 35 lb of buoyancy.

When boating in cold weather, a float coat can be worn under a PFD. In addition to providing $15^1/_2$ lb flotation, the coat offers excellent protection against hypothermia and cushions the rib cage from traumatic injury during a fall.

Test and wear any new vest in a pond or pool and practice the HELP position. The wearer should float in a slightly reclining position with the water rising no higher than armpit level. PFDs should be close fitting, with small arm holes. Children require a properly fitted model with crotch straps to prevent the vest sliding up over the arms and head. Some children require a safety harness (see below) to keep them aboard. Never buy an oversized PFD, expecting the child to grow into it in the future. Incorrect sizing of a PFD compromises usefulness and may have tragic consequences.

An immersion (survival) suit is the ultimate protection from hypothermia and drowning. It is a combination life raft and drysuit. Features include a watertight full-length zipper, watertight hood, face seal for wind and water protection, detachable mitts, neoprene wrist seals, integral boots, inflatable head pillow for optimum flotation angle, water-activated safety light, whistle, and buddy line. Their general bulkiness (one size fits a 110- to 300-lb [50- to 136-kg] person) and built-in gloves, however, make them impractical for continued wear while actively working aboard ship. The Coast Guard now requires personnel on board their vessels to wear a drysuit when ocean water temperature is less than 10° C (50° F) and a less bulky antiexposure suit with insulating underwear and clothing when temperatures are between 10° to 15° C (50° to 60° F). A type III PFD is still necessary both to provide adequate flotation if a head pillow and flotation are not integral to the suit.

One potential disadvantage of survival suits (as with autoinflatable PFDs) is that their buoyancy may impede escape from an overturned craft. The person may become trapped in the cabin, under the cockpit, or under the trampoline of a multihull. "When the trampoline is on top of you, buoyancy is your enemy," said one multihull sailor, who barely survived after capsizing his vessel while wearing his survival suit. Always don the suit topside (you're a cork when wearing a survival suit below decks), and get away quickly from a rolling, unstable craft. Have a suit for each crew member when cruising in nontropical cold water below 15° C (60° F). Crew should read the instructions; there is a specific technique and sequence for getting into the suit, pulling the hood over the head, zipping, and closing the face flap. Practice, with a stopwatch running, until the suit can be closed and made water tight in less than 60 seconds. Inspect the suit regularly for any tears or deterioration, and lubricate the zippers with Zipper-Ease or other zipper lubricant. A partially closed immersion suit serves only to keep the victim afloat and alive until he dies of the cold shock response or succumbs to immersion hypothermia.

The crew of the fishing vessel *Cape Fear* learned the tragic consequences of failure to follow these simple rules. All five crew members had to quickly abandon ship 5 miles off the coast of Rhode Island on the evening of January 8, 1999. While returning to port and without warning, the ship took several waves over the stern, flooding the fish holds. The vessel then slowly down-flooded, rolled to port, and capsized in rough seas. Water temperature was 2° C (36° F), seas 6 to 8 ft, winds 20 to 30 knots, and air temperature a brisk 0° C (32° F). Visibility was reduced in the nighttime rain and snowstorm. Three crew members in zipped closed, hooded survival suits clung to a plank for 25 minutes before being rescued by the nearby fishing vessel, *Misty Dawn*. One deckhand was seen to enter the water with his survival suit partially on up to his hips. His screams for help (he said his suit slipped off) were heard about 10 ft from the crew for a couple of minutes, and then they stopped. His body was never recovered. The crew last saw the second deckhand donning his survival suit in the cabin. *Misty Dawn* remained on scene and searched for the two young men without success over the next several hours. The next day, a body was recovered from a nearby beach floating face down, with the hood off; the survival suit was saturated with diesel fuel and filled with water, and the rusty zipper was open down to the waist. The survival suit was labeled *Cape Fear*.

Safety Harness

Always wear a safety harness in rough weather, at night, and whenever on deck alone, out of sight of crew, or when both hands are occupied. In heavy weather, wear a harness even while steering from the relative protection of the cockpit. Adjust the harness to fit snugly around the chest, two inches below the armpits. It should be constructed with webbing at least two inches wide, and have a breaking strength exceeding 3300 pounds. Tethers connect the harness to through-bolted deck fittings or to a dedicated jackline, made from either uncoated stainless steel wire or webbing running fore and aft to a through-bolted padeye or cleat. Designate a jackline specifically for the cockpit that can be reached from the companionway, so that crew can clip on before entering the cockpit. The jacklines should be continuous, allowing the crew to roam without having to unclip the harness. Webbing is preferable to wire because feet will not roll on it. Wire jacklines should be inspected for broken strands, and webbing for UV damage and weakening. A length of marine rope is not an acceptable substitute; neither is the stanchion-mounted lifeline. Specifications of each of these components require a breaking strength of over 4950 lb (2245 kg), because the force generated by a 150-lb (68 kg) body falling 12 ft (4 m) is more than 3000 lb (1360 kg). Being dragged in the water by a safety harness can be a bruising experience; therefore, tethers should be no more than 2 m (6.5 ft) long and fitted with highest quality snap shackles at both ends. An elastic cover helps to keep them from dragging under-

foot; a second tether 1 m (3 ft) long helps to triangulate support and insures continuous contact with the ship while changing to different positions; it is also best for use in confined places such as the cockpit and at the helm's station. The shackle at the chest should be easy to disconnect for quick release from a capsized boat.

Don the harness before going on deck, and secure the tether before climbing up the companionway. While on deck and underway, hook onto the windward (uphill, upwind) jackline whenever possible. One is more likely to fall down to leeward, and the shorter length of the tether will keep a person on deck instead of dragging him through the water. Women should not adjust the chest strap below their breasts. Injury may occur from the upward force that is placed on the harness when it suddenly comes under tension; some harnesses are designed for females to avoid possible injury. PFDs are available with integral safety harness. This convenient combination may be the most important piece of personal safety gear at sea. A harness not only will keep crew aboard and prevent separation from the vessel if overboard, but can guide escape from an overturned craft, especially when someone is disoriented.

Remaining Aboard

Falling overboard (most often from collision) and then drowning is the most common cause of marine fatality in recreational boating and commercial shipping. In stormy weather, the safest location is in the cabin.

Virtually all man-overboard incidents can be prevented by following these rules:

1. Remain sober, especially if you expect to go on deck for any reason.
2. Wear nonskid footwear when working on deck and have nonskid paint or pads in critical work areas.
3. Walk in a crouched position with a low center of gravity and wide-based stance when the boat is rolling, heeling, or pitching.
4. If the boat's motion is too violent to stand, then crawl or slide along the deck.
5. Use a safety harness (with two shackles) as a "third hand," secured to a strong attachment point.
6. Use a safety harness whenever going aloft in the rigging or climbing any superstructure.
7. Avoid leaning overboard with all your weight on a lifeline or stanchion.
8. Know the location of secure handholds and grab rails so you can find them at night.
9. Know the safe routes to avoid tripping on deck hardware, vents, and hatches, especially at night.
10. Do not urinate from the afterdeck in rough weather unless you are kneeling and attached with a safety harness.
11. Wear a safety harness whenever seasick; vomit into a bucket rather than leaning overboard.
12. In heavy weather, sleep in the harness and be ready to attach the tether quickly if called on deck for an emergency.
13. In heavy weather, stay below in the cabin unless absolutely needed on deck.
14. Remember: One hand for you and one for the boat.
15. Wear an LED headlamp while working on deck at night.

Recovery of Crew Overboard

If one falls overboard without a safety harness, don't panic! Maintain a positive attitude about survival and rescue. The will to live makes all the difference. In cold water, be prepared for violent shivering and intense pain. To survive, the single most important step is to immediately bring one's breathing under control, to overcome the reflex hyperventilation and risk of aspiration. Do not undress; clothing traps air, providing insulation and added buoyancy. Trapped water next to the skin retards heat loss, similar to a wetsuit's effect. If wearing a hat and gloves, leave them on. Move slowly to decrease heat loss from blood circulating through the skin and active muscles. Slowly tread water, facing away from the oncoming waves to minimize wave splash, and maintain airway freeboard.

Assume the HELP position if wearing a life jacket. If a short swim presents the best chance for survival, remove extra clothing and footwear and maintain airway freeboard by employing a breast-stroke or dog-paddle. Keeping the head above the waves reduces heat loss, because up to 50% of the body's heat loss can be from the head. Allow a rescue vessel to approach downwind. Do not swim to the boat you fell from unless it is completely disabled and unable to maneuver. Conserve strength for the rescue. Whenever possible, get out of the cold water (e.g., onto a capsized or partially submerged boat, or rocky ledge), and stay out of it, no matter how low the air temperature and wind chill effect, Water saps the body's heat 25 times faster than air at the same temperature. Keep on all clothing to reduce wind chill.

Time is the critical factor in recovering a person overboard. A well-rehearsed rescue under expert leadership with clear communication during the rescue maneuvers is most likely to succeed. When someone is observed falling overboard, shout "man overboard" and sound the all stations alarm: a continuous blast of the ship's whistle, supplemented by continuous ringing of the general alarm bell for not less than 10 seconds, or three short whistle blasts. Designate one or more crew members to spot and point to the victim continuously without losing sight of him (not even for an instant). Floating objects should be thrown overboard, including buoyant cushions, horseshoe buoys, ring buoys, and extra life jackets, in order to litter the water surrounding the victim. The gear may provide extra flotation and will help mark the area for the spotter. Unfortunately, most of these objects will drift faster in winds over 10 knots than a person can swim, so the man overboard (MOB) cannot expect to retrieve a PFD after falling overboard.

Special equipment designed for locating and retrieving a person overboard should be deployed immediately (Fig. 76-13). This gear should be ready for easy deployment and must release instantly. Too often, the gear is protected against accidental loss by extra wraps of line to the stern pulpit or rigging. Any delay in releasing MOB gear will leave it too far from the victim. A crew overboard pole is a 12-ft (4 m) flagpole that is ballasted to remain upright in rough seas. Without the drogue accessory (a parachute-shaped device to slow a vessel's drift downwind), it will quickly drift away from the designated area. A man overboard module (MOM) automatically deploys a CO_2-activated horseshoe buoy and a 6-ft inflatable locator pole equipped with a drogue and water-activated, lithium-powered light.

A variety of lights have been developed to serve as rescue beacons. The overboard marker strobe marks the site and illuminates the scene for the rescuers. It automatically activates when thrown into the water. Waterproof personal rescue strobe lights attached to a PFD can flash for 8 hours at 1-second intervals and are visible a mile away. Other personal strobe lights can last up to 60 hours, with variable rates to conserve battery

Figure 76-13. Crew overboard alarms are capable of alerting the vessel's crew to an emergency, while storing the precise coordinates where the crewmember fell overboard.

power. A U.S. Navy whistle has a special flat design to prevent the whistle body from holding water and dampening the sound. Attach one to every PFD.

Electronic overboard alarms are available for crew; these are small transmitters alerting the mother ship receiver. The personal alarms are activated when they contact the water or when manually turned on.

All onboard GPS units have a MOB function, which, if activated, will create a waypoint at the vessel's position when pressed. It will also make the waypoint "active," so the vessel can return to the latitude and longitude of the person at the time of the fall. The GPS is a powerful search and rescue tool, but is not a substitute for maintaining strict visual contact and using signal lights and markers. The inherent small degree of error with GPS in marking the waypoint of the MOB is magnified in heavy winds, large seas, and strong currents, because the person may drift downwind while the boat returns to the scene. GPS receivers receive signals from U.S. Air Force satellites and then compute the location accurately to within 10 m. It may still be close to impossible to locate the person in rough seas or during reduced visibility.

Rather than rely on the ship's emergency locator beacon, sailors can wear them on their person. A small unit can be worn (on a PFD) by a crew member as a personal locator beacon (PLB). Weighing as little as 17 ounces, and not much larger than a cigarette pack, it also transmits on the 406- and 121.5-MHz frequencies. The battery life for this compact unit exceeds 24 hours. Category I PLBs are inherently buoyant but do not float upright in the transmitting position. The transmitter requires manual activation and must be held out of the water with the antenna pointed skyward. It would be difficult to do this without wearing a PFD. Some units have an integrated GPS to yield location accuracy to within less than a 300-ft (91 m) radius, versus a 3-mile (5 km) radius from the 406 MHz operating alone. These units also require registration with NOAA. A new product combines a 125.5 MHz PLB together with handheld radio direction finder. The direction finder is designed to pinpoint the signal sent from the PLB from the deck of the mother ship. Together, they work as a micro-SAR unit. The devices will function on batteries for at least 20 hours and are effective over a range of 8 nautical miles. PLBs have been the subject of controversy in 2004. There are some serious performance issues with these compact units, relating to the accuracy of transmitted data (GPS position), transmit power, signal strength, and battery life. One should anticipate future development and improvement.

A less technical signaling device designed for night use is the Rescue Laser Flare. This is really not a flare, but a light that produces a visible line of laser beam to attract the attention of search boats and aircraft within a 10-mile radius.

The goal in overboard recovery is to return as quickly as possible to the MOB using the simplest maneuver. Begin the process immediately. A boat traveling at a speed of 8 knots moves away from the MOB at about 13 ft (3.9 m)/second or 800 ft (243.8 m)/minute. At that rate, half a mile is traversed in 3 minutes. Motorboats should reduce speed and return in a simple circle. Sailboats under power alone can return by simply circling back and approaching downwind of the victim. Establish contact by using a heaving line, such as a floating polypropylene line in a throw bag. If the boat is drifting downwind, slowly advance forward to complete the recovery over the windward side. Placing the boat upwind of the victim entails the risk of drifting onto the person. Sailboats under sail can choose from a variety of options.

The "quick stop" recovery maneuver is designed for rapid MOB recovery. This method enables the boat to reduce speed immediately by turning into the wind while trimming in the mainsail and keeping the headsail (jib) aback. Thereafter, the helmsman keeps the boat turning downwind while steering to remain close to the victim. After passing abeam of the victim, the jib is dropped (or furled), and the boat heads up to the wind (on a close reach) to stop alongside the victim at an angle of about 60 degrees to the wind with the sails luffing (flapping into the wind). By sailing the final approach to the MOB on a close reach, the sails can be fully luffing or trimmed in to maintain forward movement if short of the mark. The technique is similar to picking up a mooring under sail. The boat can also be left beam-to-wind with the sails luffing while contact is made with the victim. The engine can be started and left in neutral, ready to be used if needed in the final approach. Rescuers must ensure all lines are aboard before engaging the engine to avoid fouling the propeller. It is advisable to return to neutral when close to the victim. The main danger to the victim is being sucked under the stern while the propeller is turning and the boat is moving forward under power.

The direction of approach to the victim used by the rescue boat is controversial and involves judgment based on many variables, including the sea state, wind strength, drift of the boat relative to the victim, maneuverability of the boat, and condition of the MOB. If the seas are large, approach to leeward (downwind) of the MOB so the boat cannot fall off a wave and injure the person in the water. Gentle seas permit an approach to windward (upwind) with a slow drift down to the victim (the boat will always drift faster than the person in the water, so have retrieval gear ready).

An injured, hypothermic, or unconscious person (not waving or looking at the rescue boat) requires assistance by a rescue swimmer who should take steps to avoid the cold shock response. The rescue swimmer should be tethered to the boat during recovery of the MOB. Ideally, the rescue swimmer

Figure 76-14. Successful rescues of a crew overboard requires consistent methods for contacting the victim, and for getting him back on board.

should be trained in water rescue and lifesaving techniques and be able to recognize the warning signs of panic as he or she approaches the victim. If the MOB is unconscious, assume possible head and neck injury, and stabilize the cervical spine before hoisting the victim out of the water.

The goal is to have the MOB back on the boat as quickly as possible (Fig. 76-14). Practice the different techniques and decide which method and modifications work most effectively. The Lifesling, developed to enable one person to retrieve a person overboard, is a flexible floating collar that doubles as a hoisting sling. Deploy the collar from the stern pulpit and deliver it by repeatedly circling the victim, much as a ski boat maneuvers to deliver the tow rope to a fallen water skier. After securing the horseshoe over the head and under the arms, pull the victim back to the boat and hoist him in the apparatus with the assistance of a halyard and winch.

Lifelines are an obstacle to bringing the MOB back on deck, but are an important source of protection for the remaining crew. You may elect to secure lifelines at the stern (or transom) with lashing rather than shackles or pins so they can be easily cut and released in a recovery should the need arise.

If the crew has lost sight of the victim, immediately call for assistance. A mayday call on VHF-FM channel 16 will notify the Coast Guard and simultaneously alert all ships in the area monitoring this channel. The last known position should be obtained using GPS or reference to any navigational buoys and landmarks on shore. Rescuers should perform repeat searches of the area, because it is easy to miss someone in poor visibility or choppy seas. The chances for recovery are close to 100% if the victim is well-equipped. Ideally, this includes a high-visibility (orange or yellow) PFD with reflective tape combined with a safety harness, personal strobe light, loud whistle, packet of waterproof self-launching meteor flares (Skyblazer), fluorescent dye marker, and personal EPIRB.

HOW TO ABANDON SHIP

Before abandoning ship, the first priority is to dress warmly and put on a life jacket. Layer on as much warm and quick-drying clothing as possible. If a survival suit is available, wear that instead of a life jacket, but bring the jacket for later use in the raft. One crew member should broadcast a mayday message on all available radios: VHF-FM channel 16 (good up to a 20-mile range) and SSB 2182 kHz (good beyond 50-mile range). Make a satellite telephone call to contact someone who can continue coordinating the rescue after abandoning ship. Repeat the coordinates of the ship's location slowly and distinctly. Activate a 406-MHz EPIRB. Simultaneously, other crew should launch the life raft and locate the abandon ship bag (a survival kit). It is recommended to augment the modest amount of survival gear found in most life rafts by having select items packed inside the raft container at the time of its annual repack (include VHF radio, EPIRB, SART, water maker, medical kit, signal pack, sharp knives). However, it is not recommended to put those items in the raft if they might be needed independently. Those items can be packed into a waterproof bag securely attached by a short line to the raft.

Each crew member should have an easily accessible prepacked waterproof bag containing extra dry clothing, personal medications, passport, prescription glasses and sunglasses, personal strobe, safety harness and tether, wallet, and any other personal valuables and necessities. If the ship's bag has been properly stocked, little else is needed from the sinking ship except synthetic blankets (not down sleeping bags, which never dry), jerry jugs of fresh water (two-thirds full so they float), extra food (especially high-carb sports bars), and navigation tools. If time permits, additional communication and signaling equipment should be collected from the stricken vessel, even if equipment was already placed in the bag or raft. This includes a 406-MHz EPIRB, a SART, and a waterproof handheld VHF radio. In a life raft, one can never have too many EPIRBs, radios, flares, and fishhooks (Box 76-15).

There are three loosely defined classes of life rafts: ocean, offshore, and coastal. These classes refer to the areas in which the vessel normally operates and reflect differences in size, design, construction, quantity of survival stores, and kinds of equipment. Another practical way to categorize life rafts is in terms of performance. A poor life raft will only keep its occupants from drowning until they die of hypothermia or heatstroke. A mediocre life raft will protect the survivors from dying of exposure until they succumb to thirst and starvation. The best life raft will provide food and water, have a means of replenishing both, and be sufficiently equipped to sustain life and facilitate rescue. To be a survivor, a person must manage the emergency, remain alive, and be successfully rescued.

For coastal cruisers on a small boat, an inshore rescue platform (e.g., Rescue Pod or Rescue Raft) can afford a level of safety and protection. It bridges the gap between PFDs and life rafts. Although not recognized as a lifesaving device (e.g., a life raft), it is a good solution as an emergency craft for near-shore (inshore) cruising on a small boat lacking storage space for a conventional life raft. For a variety of reasons, it should not be seen as a substitute for a coastal life raft. It is only suitable for

Box 76-15. Abandon Ship Bag for Ocean Passage

SIGNALS AND COMMUNICATION

1. Registered 406-MHz category II COSPAS/SARSAT emergency position indicating radio beacon (EPIRB)
2. Waterproof and submersible portable VHF hand-held radio transmitter with spare batteries
3. Search and Rescue Transponder (SART)
4. Flares, smoke signals, canister and lung-powered horn, ship's bell, whistle, kite, dye marker, mirror
5. Radar reflector
6. Waterproof binoculars
7. "Cyalume" chemical light sticks
8. Waterproof LED flashlight with extra batteries; consider the Night Star, an LED light with a reliable self-contained generator and storage capacitor
9. Strobe light with spare batteries

PROTECTION

1. Survival suits, gloves, spare sunglasses, hats
2. Expanded medical pack with extra seasick medications, SAM splints, narcotics, sunscreen, lip balm, emollient skin cream, tampons.
3. Backup sea anchor and line

SUSTENANCE

1. Portable reverse-osmosis desalinator (Katadyn Survivor 06 or equivalent)
2. Fishing harpoon, spear gun with spare heads, extra fishing gear, including fishing line, wire leaders, assorted barbed hooks, sinkers, lures
3. Knife, can opener, sharpening stone, small cutting board, scissors, storage bags, eating utensils, nylon mesh bag, waterproof bags, zip-lock freezer bags
4. High carbohydrate, low protein energy bars
5. Collapsible bucket, urine bottle
6. Packs of chewing gum
7. Comprehensive survival manual, fish identification book, cards, reading material
8. Collapsible water containers

TOOLS

1. Small ax, pliers, and screwdriver or multi-tool
2. Dive mask
3. Duct tape for raft repair and crew repair
4. Raft patching clamps
5. Waterproof pad and pencils

use on protected bays, sounds, and inland waters, especially in cold climates. A stable inflatable dinghy can also serve to support the crew on inland waters and very near shore in an emergency requiring abandoning ship.

A tender that can also serve as a lifeboat may be a good alternative for inland and near coastal sailors who do not want to climb into a raft, flip on the EPIRB and wait. The 8-foot polyethylene Portland Pudgy lifeboat/tender (Portland Pudgy Inc., Portland, Maine) can be rowed, sailed, or powered to shore. It is stable and unsinkable and has an exposure canopy and numerous watertight hatches for storage. There are no anecdotal reports to evaluate its performance; however, its use in near coastal waters sounds appealing and reasonable.

Box 76-16. Ocean 16 Life Raft Equipment

1. Inflation pump with hose
2. Two 4-ft paddles
3. Two sea anchors (one stowed inside ready for use, one outside automatically deployed)
4. Two bailers
5. Two sponges
6. Drogue and cord
7. Repair kit containing sealing clamps, patches, cement
8. 100-ft buoyant heaving line with quoit
9. Floating sheath knife
10. Scissors
11. Can openers
12. Thermal protection bags
13. Provisions
14. Water
15. Graduated drinking vessel
16. First-aid kit (primarily for wound management)
17. Seasickness pills (6 per person)
18. Fishing kit
19. Survival instructions
20. Operating instructions
21. Two red parachute flares (rocket propelled, red)
22. Six hand-held flares (stowed in watertight container)
23. Signaling mirror and whistle
24. Signal flashlight with spare batteries and spare bulb
25. Two smoke canisters

Have the life raft serviced annually by an authorized service facility and be present when the raft is both unpacked and repacked. One has a vested personal interest in seeing how well the job is done, and this is an invaluable opportunity to become familiar with all components of the raft.

Box 76-16 lists the gear found in a six-man inflatable life raft approved by SOLAS for ocean service (SOLAS A). The SOLAS B raft is designated for limited service. It carries no food, water, or fishing kit. These rafts also carry half the number of flares and smoke signals found in SOLAS A life rafts. Ocean-service rafts (and some lower specification rafts) are designed for use in rough offshore conditions. They are constructed with two complete, stacked inflation chambers. One flotation chamber is sufficient to support the number of people for which the raft is rated.

Give careful thought to how and where the life raft is stowed. The raft should be securely stowed either on deck, with hardware capable of withstanding the shearing forces due to capsize, or in the cabin. The lid on a deck locker or lazarette is likely to open in a knockdown or capsize, unless it closes with deadbolts. Carry SOLAS-grade rafts on deck in hydrostatic release brackets.

If the boat has a dinghy, prepare to tow it behind the raft. It is useful for storing additional supplies and can be used with an improvised sailing rig. Although a solid dinghy is less seaworthy in a storm than a life raft, it is far more durable against shark attack and is immune to chafe. A dinghy has more maneuverability and strength than a raft.

To launch the raft, the canister should be thrown overboard on the downwind (leeward) side of the boat and then inflated.

It is dangerous to inflate a life raft aboard the ship with the CO_2 cartridge because the raft may become wedged in the rigging of the sinking ship or be punctured accidentally. Attach the line coming out of the canister (painter) to the boat before the raft is inflated. In the confusion surrounding an emergency, this critical step is sometimes forgotten, and after the raft inflates, it drifts away. A sharp jerk on the outstretched painter triggers the CO_2 cartridge and inflates the raft. If the first pull is unsuccessful, give a second, stronger tug. If the raft fails to inflate in the water, however, bring it aboard and inflate with the hand pump.

Station one person with a knife by the painter, prepared to cut the line. Another safety knife is located in a pocket by the entrance of the raft to cut the painter should the ship begin to sink after the crew has entered the raft.

Once everyone is aboard, get away from the sinking vessel quickly by paying out all the painter line. Risks for the raft and its occupants at this moment include being struck from below by surfacing wreckage or becoming entangled with the boat's rigging if it rolls and sinks. If the boat remains afloat, attach a quick release line to it. The wreckage is always easier to spot than a small life raft; it remains a source of additional food and supplies, and may be salvaged later.

Large-capacity rafts, usually found on commercial ships, are often mounted in a cradle with an automatic disconnect device called a hydrostatic release mechanism. This mechanism can be manually activated by pressing a button, which releases the straps holding the canister to the cradle, or it may have a snap shackle or other quick-release in series with the hydrostatic release. The canister can then be dropped overboard. In the event the boat begins to sink before the life raft is launched, a hydrostatic release mechanism will open when the vessel sinks to a depth of 10 to 15 ft, allowing the canister to float up to the surface. As the vessel sinks to the end of the painter's length, it will jerk the painter and initiate inflation. The specially designed weak link on the cradle where the painter is attached is designed to break and free the raft from the vessel completely before it is pulled under by the sinking boat. An unoccupied inflated raft will blow downwind quickly, certainly faster than someone in a life jacket can swim; if in the water while the raft inflates, hold onto the raft or painter.

Full inflation normally takes less than 30 seconds. If possible, allow time for the canopy arches to inflate completely before boarding. The additional pressure caused by body weight may cause gas to escape through the pressure-relief valves before full inflation has occurred. If entering the raft before it is fully inflated is unavoidable, crawl under the collapsed canopy.

When possible, try to remain dry and enter the raft directly from the sinking boat rather than from the water. If you enter the water, ease yourself in to avoid the cold shock response. Even when wearing a survival suit, it is much more difficult to enter the raft from the water. The effort can be exhausting or impossible without assistance. All rafts are strong and stable enough so that you can jump directly onto the canopy and supporting arch tube when entering the raft. If jumping is necessary, it is preferable to jump into the entrance from as low a height as possible; climb down a ladder, or lower yourself down with a line. Be careful not to jump on top of the occupants or tear the canopy. The canopy serves to collect rainwater, helps to keep the raft dry, and shields survivors from the elements.

When entering from the water, use the automatically deployed boarding ladder and handholds at the entrance to the raft. If the crew is forced to enter from the water, the first person into the raft should throw the heaving line to the others. The line has a rubber doughnut-shaped quoit attached to the end and will allow a person to hold on firmly. It is important to get everyone out of the water quickly. With the recovery of each crew member from the water, a significant volume of water enters the raft, adding to what may enter through wave action. If you recover someone unconscious, be sure not to leave him or her lying facedown in this water. Some experts suggest that the fittest be recovered first, so they can distribute themselves in the raft to assist with stability and attend to the injured. If you must swim away from the raft to help a crewmate, be certain you are attached to the craft with a safety line.

The hissing sound heard after inflation does not signify a leaking or defective raft. The relief valve is simply releasing excess gas pressure. Immediately after the release of the CO_2–nitrogen mixture into the raft's interior space, ventilate the life raft thoroughly and periodically thereafter. Inflate the floor separately with the manual pump to provide insulation from the cold sea. Because the craft is so vulnerable to capsize (see later), it is extremely important to inventory and then secure all equipment as it comes aboard. Then check the life raft for any damage or leaks and the crew for any life-threatening injuries. Activate the EPIRB immediately and leave it on.

The first medical action should be distribution of seasickness medication. In a life raft (sometimes referred to as "an inflatable vomitorium"), everyone is susceptible to seasickness, especially in the first 24 hours. Make the raft as dry as possible, and remove wet clothing. Close the door, and huddle together to conserve heat. Do everything possible to conserve body heat, strength, and spirit, and prepare for rescue.

Life rafts have ballast pockets under the floor to trap seawater and thereby increase stability. Unfortunately, if the wind gets under the craft as it rises on a wave, it can still capsize. A capsize is most likely immediately after launching when the raft is empty. Have one or two of the fittest, heaviest crew members board the raft early to provide stability and ensure that the ballast water pockets are filling. They can also assist in transferring additional equipment and help others into the raft. The raft may even initially inflate upside down; most rafts can be properly righted from the deck of the boat without entering the water.

No immediate danger exists if the raft capsizes with crew inside, because sufficient air is available in the space under the canopy. It is necessary to enter the water in order to right the raft, however, since it is unlikely to right itself unless rolled again by successive waves. With the raft empty, it can be righted by pulling on the righting straps at the bottom. Kneel on the downwind side with feet braced on the CO_2 cylinder, and lean back. The raft will right itself easily when the wind catches it.

To prevent recurrent capsizes in heavy wind and seas, additional measures need to be taken. Every raft is equipped with a cone-shaped sea anchor. This device is essential in rough weather. It provides stability in high winds, helps to prevent capsizing in rough seas, serves to aid in directional control of the raft, and helps reduce drift. The benefit of reducing drift in storm conditions is controversial, because the raft may become sluggish with the sea anchor deployed. If sea room is sufficient, the occupants may be more comfortable and the raft less likely to capsize if it is allowed to drift at the same rate as the waves. Deployment of the sea anchor is automatic on some rafts.

Stream the sea anchor after the raft is clear of the sinking vessel. Check that the rope does not become entangled, and protect the raft from chafe at the point where the rope is attached. Weight distribution is also critical to avoid capsize. Position most of the crew on the windward side, the same side from which the sea anchor is deployed, to act as ballast. Weight on the windward side reduces the chance of the raft being lifted and flipped over by the wind. In seas with high wave crests, be prepared to maintain the raft's balance and quickly shift as needed to prevent capsize in the opposite direction.

Since a survivor can quickly become separated from a capsized raft, have everyone attach a line to the handholds if capsize is possible. Whenever a craft capsizes, gather the crew in the water and check that no one is under the overturned raft. Everyone should return as quickly as possible to avoid the progression of the cold shock response and hypothermia, and to conserve precious energy.

PREPARATION FOR RESCUE: LIFE IN THE RAFT

After successfully abandoning ship and securing both crew and equipment in the life raft, prepare for rescue. Panic, fear, helplessness, and hopelessness can easily defeat the best-equipped and most experienced crew. A crew that is optimistic is more likely to survive. If the EPIRB is activated in busy shipping lanes, the probability of rescue in a few days is high. The leader can set the example and improve morale by staying calm and formulating an active strategy to be rescued. Assigning meaningful duties to everyone by structuring work and rest time instills in the crew some sense of control over their destiny. In Pornichet, France, the Center for the Study and Practice of Survival proposes the following "seven tools of the mind to help you survive":

1. Size up the situation rationally. Analyze and evaluate all options and plan the best course of action. Consider the resources available, the time frame, and risks.
2. Understand and control. Get in touch with your feelings. Identify and acknowledge your fears; own and control them.
3. Risk the least, achieve the most. Evaluate and estimate the risks of any course of action. Ask what can be done to lessen the risk, and consider if the crew can handle the increased stress.
4. Value your reason for living. Find your own personal reason for wanting to survive. It will motivate you and powerfully counter any urge to give up.
5. Imagine new ways to do things. Use unconventional, lateral thinking. Improvise and try imaginative, creative solutions to problems. Trust your intuition.
6. Victory comes with time. Time is your asset, and problems often resolve themselves if given the opportunity. Avoid looking for quick solutions. Rushing a task often leads to failure.
7. Enrich yourself with a permanent positive mental attitude. Appreciate that you are alive, and keep your sense of humor.

AMSEA (Alaska Marine Safety Education Association) teaches the Seven Steps to Survival most succinctly:

1. Recognize you are in a survival situation
2. Inventory your equipment
3. Create a shelter
4. Signals
5. Water
6. Food
7. Attitude

Signals and Watch Schedules

Review how to use each type of distress signal. Take inventory of the signaling devices available, and agree on priorities for their use. Turn on the EPIRB and leave it on. The battery is good for 48 hours, during which time the signal should have activated an SAR operation and transmitted an updated location.

The most important duty aboard the raft is to maintain a continuous and effective lookout for rescue boats and airplanes. Create a watch schedule to rotate duties every 2 hours. The lookout's responsibility is to monitor changes in the weather, wind, sea conditions and to look for signs of nearby land and passing ships. In calm seas, make intermittent observations while standing up in the raft. The visible horizon is 2.6 nautical miles if the observer's eye is at 5 feet (1.5 m); raising the eye to 10 feet (3 m) (standing up in the raft as it rides on a swell) adds another mile. Other crew should monitor the well-being of the sleeping survivors. Their duties include providing first aid and comfort to injured victims, fishing, making potable water, organizing the stores, keeping the log, bailing, and maintaining the raft.

Raft Maintenance

The raft is subject to tears, punctures, chafe, and structural deterioration, which cause air chambers to leak and lose watertight integrity. As the raft deflates, it begins to flex and chafe. The buoyancy chambers should therefore be kept taut with no apparent folds. To prevent overinflation, the pressure relief valves must be operational during the daytime to let off heated expanding air. This is especially important in the tropics. As the temperature drops at night, the raft should be reinflated with the manual pump.

Protect the raft against sharp, pointed, and abrasive objects such as knives, tools, fishhooks, spears, fish bones and teeth, shells, belt buckles, jewelry, and pens. All pointed tools must be kept sheathed or wrapped except when in use.

Attached to the canopy are internal and external dome lights. These help survivors find the raft at night when abandoning ship and provide interior lighting. These battery-operated lights last 12 to 24 hours, so turn them off when not in use. They are not intended to be rescue lights.

Health Issues and Hypothermia

After seasickness, no other condition is more debilitating than chronic sleep deprivation. Sleep deprivation causes decreased alertness with apparent blackouts of attention, which become increasingly prolonged and frequent. A groggy, inattentive lookout can easily miss the opportunity to observe and signal a passing ship. Conversely, the person may begin to hallucinate and see ships and planes. Impaired responsiveness to new situations and inability to make quick decisions further jeopardize the rescue.

Whenever possible, lie down and rest. Stretch out to relax muscles that are constantly working to keep the body stable in the raft. Insulate the recumbent body against heat loss, especially from the cold floor of the raft. Always keep the double bottom of the floor of the raft fully inflated and make every

effort to keep the raft dry (hypothermia rather than exposure to severe weather is the greatest threat to survival in most abandon-ship scenarios). Line the floor with sails, tarps, and extra clothing. The double layer floor also protects against the bumps of sharks, dorado, and other fish. Spraying salt water directly on the skin or clothing to cool the body by evaporation is not recommended. Salt-encrusted skin is more likely to break down and become infected. Dry clothing protects the skin from painful saltwater boils and other bacterial infections. Apply an emollient to knees, hands, elbows, and buttocks to decrease skin abrasion. Common lubricants that moisturize the skin are petrolatum, mineral oil, and baby oil. Any area of skin breakdown should be smeared with an antiseptic or antibiotic cream and covered with a dry dressing.

Hypothermia becomes a threat with an air temperature of 0° to 10° C (32° to 50° F) or with repeated immersion in cold water (see Chapters 6 and 7). Hypothermia is likely if the raft repeatedly capsizes. Sustained, uncontrollable shivering is the earliest clinical sign of a drop in core temperature. Other early clues of hypothermia are changes in motor skills and mental status. Manual dexterity, balance, and strength are impaired. Ability to climb back into the raft may be lost. Changes in mental status may involve judgment and cognitive skills.

Recognition of the early signs of hypothermia is critical. Prompt treatment usually prevents progression to severe hypothermia, which is fatal since it is untreatable in the raft. Treatment of mild to moderate hypothermia consists of preventing further heat loss by sheltering the victim from the wind, sea spray, or rain. Whenever possible, remove all wet clothing and dry the skin completely, then dress the victim in multiple layers of dry clothing. If a change of clothing is impossible, provide a vapor barrier with foul weather gear, a sail bag, or sleeping bag. If a sail or blanket is available, wrap the victim like a burrito, covering the head, neck, and face. Let the victim shiver. If shivering is vigorous, the heat generated internally provides the most effective method of rewarming. Calories from simple sugars and carbohydrates and ample water are necessary for the muscles to continue the shivering response.

A new SOLAS requirement for survival craft is a thermal protection suit for each crew member. These suits are ideal for keeping a person warm and dry in the survival craft. Foil space blankets made of aluminized Mylar are useful as sun reflectors on the canopy, and they effectively minimize heat loss through radiation. A Mylar blanket reduces body heat loss by 80% to 90%. However, the major heat loss in raft survivors is by conduction through the raft floor, against which a foil blanket affords little protection.

Water

To prevent heat-related illness (see Chapters 10 and 11) and dehydration, it is imperative to drink water at regular intervals. If possible, keep drinking until the urine is clear. Mechanisms for obtaining water are discussed later.

In tropical climates, keep clothing on to help reduce fluid losses and prolong the cooling effect of evaporative heat loss. Wear long-sleeved shirts, trousers, and a hat. Clothing protects skin from sunburn and reduces passive heating from solar radiation. Apply a waterproof sun protection cream to exposed areas of the face, hands, arms, neck, and feet. Stay in the shade of the raft canopy and make sure the raft is well ventilated. The best rafts have a double canopy with an air layer in between that acts as insulation from the heat of the sun and from severe cold. Space blankets with a reflective surface may also be tied over the canopy to reflect the sun's rays. Deflating the raft's floor chambers helps to cool the raft. It may be tempting to take a dip in cool seawater if you feel very hot. Be sure there is no hazardous marine life in the area and that you have the strength and ability to climb back aboard; post an alert lookout.

The body's requirement for water depends on body weight, individual physiology, degree of exertion, diet, ambient humidity, and temperature. The average person can survive approximately 12 days without a supply of fresh water (the longest reported survival is 15 days); however, they will remain fit for only 5 to 6 days, and then become delirious. Body water losses in excess of 8% to 10% of body weight cause significant deterioration in mental and physical performance. Hallucinations and delirium from hypernatremia (high serum sodium) are common with progressive dehydration; death occurs with acute dehydration when water loss approaches 15% to 20% of body weight. By contrast, complete starvation leads to death in 40 to 60 days (see Chapter 63), provided there is enough fresh water to drink. Decrements in physical and cognitive performance do not begin in well-hydrated individuals until they lose 10% or more of body weight acutely. Fit and healthy individuals are able to maintain normal work capacity during short periods (less than 10 days) on severely restricted diets. For the average adult at rest, the daily energy expenditure is 1400 kilocalories. Reducing activity to a minimum will decrease the requirement for food and water.

The human body resting at sea level, in a thermoneutral environment (air temperature 28° to 30° C [82° to 86° F]) and 50% relative humidity, will lose approximately 500 mL (17 ounces) daily through insensible loss from the skin, 500 mL from the lungs as it humidifies inspired air, and 500 mL from the kidneys to excrete metabolic waste. Therefore, an estimated 1500 mL (1.6 quarts) is the minimal daily body water loss; at 35° C (95° F), 2500 mL is required. Besides drinking water, another source of water available is that produced from the metabolism of food. This amounts to about 350 to 500 mL per day on an average mixed diet. The net result is, ideally, a minimum water requirement of about 1 L/day (1 quart/day). Carbohydrate and fat metabolism contribute to body water stores, whereas protein metabolism is depleting. The kidneys require 2 to 3 mL of water for every gram of protein ingested in order to excrete the urea from protein metabolism. This obligatory loss of body water may hasten death from dehydration long before death from starvation could occur.

Water requirements change dramatically with exercise, sweating, diet, and ambient conditions. The evaporative loss from the action of sunlight in an open boat in the tropics is estimated at 5 pints (2.4 L) per day if the body is at rest.

Life rafts, if they have any water rations, generally carry 1 L per person in 125-mL sachets; some rafts only carry as little as a pint per person. It is recommended that castaways should restrict water intake to about 600 mL (20 ounces, or 1.25 pints) daily, unless supplies are plentiful. However, this is not the absolute minimal requirement. Analysis of protracted voyages in life craft during World War II by McCance and colleagues found that the critical amount of potable water for survival was 110 to 220 mL (4 to 8 ounces) per day. This figure was based on the relationship between survival rates and availability of water in 121 life-craft voyages involving 3616 men. Reviewing 1314 men at risk who drank only 4 to 8 ounces, 96, or 7%, died. Consumption under 4 ounces resulted in much higher

death rates. In a subgroup of 62 men who were adrift in two boats for periods of 37 and 49 days with water rations between 4 and 8 ounces, only one died.[4]

Hand-operated, portable reverse-osmosis desalination units contain a semipermeable membrane that allows only fresh water to pass through when pressurized to approximately 800 psi. Using a motion similar to that employed with garden shears, these water makers produce a cup of water in 15 minutes, and can hydrate as many as 25 individuals indefinitely if operated continuously. An added benefit is that the units remove bacteria, viruses, protozoa, and other contaminants.

Without a water maker, solar stills can evaporate fresh water from salt water. The solar still was included in survival kits used by naval aviators during World War II. It provides about a quart of fresh water daily in temperate climates with sunshine. When inflated, the 24-inch (61 cm) ball containing a saltwater-soaked cloth collects fresh water after it condenses on the inner surface of the ball. The still is reusable and requires no chemicals. Unfortunately, older solar stills are fragile and will not work in rough weather.

Rainwater can be collected with the canopy of the life raft. An exterior gutter collects and routes the water to a large container for storage. Daily washing of the canopy with seawater, whenever practical, can help to remove the buildup of salt deposits. In a heavy sustained downpour, allow the rain to rinse the canopy before the water is collected.

Fish and other sea creatures contain water with extremely low salt content in their eyes, flesh, and cerebrospinal fluid. Fish blood has a high salt concentration and is not recommended. Juices can be pressed out of the flesh by twisting pieces of fish in a cloth. The blood from sea birds and turtles is also a reliable source of hydration for castaways.

Seawater is not potable. After continuous exposure to the harsh elements, intolerable thirst may drive castaways to drink seawater. Succumbing to this overpowering temptation is a major cause of death and actually hastens the process of dying. Seawater is approximately four times saltier than blood. Drinking seawater usually causes immediate vomiting. If ingested and absorbed, seawater triggers a process of osmosis in which free water is shifted from the intracellular space into the blood and other extracellular fluids to restore equilibrium. The process of osmosis dehydrates every cell in the body. It is postulated that reduction in the intracellular fluid of brain cells causes the reported madness in those who have drunk large quantities of seawater.

Survivors can reduce body water loss by optimizing the use of shade and the convective cooling effect of the sea breeze. In tropical climates, maintenance work on the raft should be limited to the cooler evening or early morning; one should rest during the heat of the day.

In the high latitudes, old sea ice is a good source of water. Sea ice loses its salt content after 1 year. The ice is brittle, bluish in color, and has round edges. New sea ice is gray, salty, opaque, and hard. Melting the ice allows tasting and judging the salinity. If the temperature drops to freezing, seawater can be collected in a can and allowed to freeze. Fresh water freezes first. Therefore, the salt concentrates in the center, forming slush surrounded by ice containing very little salt. Sea ice should not be confused with ice from an iceberg. Water from melted iceberg chunks is glacial fresh water. As a last resort, chewing a piece of gum or cloth will help to moisten the mouth and reduce thirst.

Unless survivors are assured of an early rescue or have a reverse-osmosis watermaker, they should consume no water in the first 24 hours, and utilize the body's reserves. Thereafter, survivors should restrict intake to about 500 mL (just over a pint) a day. If water is plentiful, drink up to 1 L (just over a quart) daily. Nutrition is the last priority for survival. If rescue is expected to take many days or weeks, plan to eat very little for the first 3 days. Thereafter, begin the lifeboat rations of carbohydrates. Save some rations until rescue is imminent, when extra energy will be needed.

Food

Life raft rations are similar to energy bars; they are high in carbohydrates and low in salt. They are bland, slightly moist, and sweet. Their primary purpose is to provide sugar in order to reduce catabolism and dehydration and thereby extend survival time. They should be regarded as more of a medicine than a meal and should be consumed as such. A daily ration should provide between 600 and 1400 kilocalories. Do not eat dry food unless water is available, and limit protein intake to conserve the body's water. With 2 quarts of fresh water available daily, eat as desired.

Fish are usually the mainstay of a diet at sea once survival routine and water consumption has been established. The raft casts a dark shadow beneath the surface, which appears as a safe haven for a variety of fish, especially dorado ("school dolphin"). With practice and patience, fish can be taken near the sea surface with a harpoon, gaff, or spear gun. Care must be taken not to puncture the raft with these devices.

Successful fishing with a spear gun requires proficiency. Refraction makes it difficult to hit the fish, so it is advised to minimize these effects by aiming straight down at the target, rather than obliquely. The ideal area to strike is just behind the gill cover. Attach all fishing gear to the raft with a lanyard to avoid losing it. Trailing a line with a baited hook can catch fish far from the raft. Bait can be obtained by using the guts, stomach contents, or thin strips of flesh from the first fish caught. Lures can be made from any shiny object.

At night, fish are attracted to bright lights. Instead of a flashlight, try a signal mirror or any other shiny surface to reflect moonlight onto the water.

When bringing a fish aboard the raft, use a cloth or piece of canvas to wrap the fish. Both dolphin and wahoo, which have serrated teeth, thrash wildly when they come out of the water. Wounds from fish heal poorly and easily become infected. Have a cutting board ready to kill the fish quickly by cutting through the spine right behind the head. A large fish can be stunned with a blow to the top of the head at eye level, and simply covering its eyes will calm it down sufficiently to position it for a quick kill.

Special care is required when bringing a shark aboard. A paddle or stick should be thrust in its mouth, and the fish held on its back over the flotation chamber tail first, until it is dead. The jaws should be cut out and thrown over the side before the fish is dressed.

Certain fish are inherently poisonous (see Chapter 66); these are usually located around shoals and reefs in shallow waters. These include puffer fish, porcupine fish, and the ocean sunfish *(Mola)*. Any fish with spines or bristles instead of scales should not be eaten.

As a general rule, do not eat any fish that smells bad after storage. If uncertain about the safety of a fish, test it first for

edibility. If it burns, stings, or tastes bad on the tip of your tongue, do not eat it. If it has an acceptable taste, try a small piece every hour for 3 to 4 hours initially, and if there are no ill effects after 12 hours, you can assume the food is edible. To save fish for future meals, cut the flesh into thin strips about an inch wide and half inch thick, and spread it out to dry in the sun on a flat surface. If you cut the strips with the fibers running the long way, the strips can be hung on a piece of string and allowed to dry in the open air under the raft canopy. Fish spoils within hours in the heat, so start drying some as soon as it is caught. Fresh-caught fish (except tuna), as well as dried turtle and bird meat, are good for days when the heat and humidity are not too high. Most ocean fish can safely be eaten raw. Freshwater fish should not be eaten raw, because they harbor parasites.

Seaweed is valuable in two ways. As a floating nest, it can harbor a variety of small edible creatures, including small fish, barnacles, crabs, and other crustaceans. All kelp and almost all brown and green seaweed are edible. Red seaweed is highly toxic, and any seaweed that tastes bitter may be one of the rare poisonous varieties. Leafy green seaweed can be dried for storage. Before eating seaweed, wash it in fresh water to eliminate any toxic plankton and salt that may be adhering to the weed. Seaweed must be chewed well and swallowed only after it becomes a soft paste. It has limited nutritive value because the carbohydrates are not digestible and the cellulose has laxative properties.

Plankton also has a high cellulose and chitin content and cannot be ingested in large quantities. Some toxic plankton is difficult to detect. The red tides that cause paralytic shellfish poisoning result from the blooms of dinoflagellates (see Chapter 58).

Many sea creatures follow a raft. There are reports of killer whales attacking small boats, but no documented accounts of attacks on life rafts. Other dolphins and whales may accompany the boat, but are not likely to harm the survivors in a raft. Sharks can be a menace as they swim about the raft for days or even weeks and drive away other potentially edible fish. The shark's habit of frequently bumping a raft with its abrasive skin causes wear on the flotation chambers. Every effort should be made to avoid attracting sharks. Take special care to dispose of blood and offal at night, preferably when the raft is moving. Try not to create a waste trail for the sharks to follow, and bring hooked or speared fish aboard as quickly as possible.

Sea turtles' sharp claws and beaks can damage a raft or cause lacerations, so always bring them aboard cautiously by holding the hind flippers, then flipping them over onto their backs; they can easily be killed by severing the vital arteries on the underside of the neck and collecting the spurting blood in a cup. The blood must be consumed immediately, because it coagulates in about 30 seconds. Draining the blood also preserves the quality of the meat for storage and future use. The eggs, heart, and bone marrow are edible, but the liver should not be eaten. Dry whatever meat is not immediately consumed. The bones inside the flippers have tasty marrow. Turtle eggs are rich in protein and fat. If the liver is crushed, the skimmed oil can be used as skin oil. Use the shell to bail the raft.

All seabirds are edible, but catching them may be more luck than skill. Their beaks and wings can be dangerous, so grab them by the feet. Float a baited hook on the water (fish guts are best), and let the bird hook itself. After the bird is hooked, throw a piece of canvas or article of clothing over the bird. With the head and beak well covered, compress the chest to suffocate the animal, or twist and quickly pull its neck. Rather than plucking the bird, slit the skin over the breastbone and peel off the skin with the feathers intact. The entire bird, except for the intestines and the small green gallbladder, is edible. The flesh may contain bioluminescent substances from ingested plankton, giving the dead bird a ghostlike glow at night, but it is nontoxic. The forewings and legs make excellent bait, and the feathers can be made into fishing lures.

RESCUE AND EVACUATION OF SICK AND INJURED

Transferring personnel from a boat or a life raft to a rescue ship or helicopter entails risk for everyone. It may well be the most dangerous aspect of the survival ordeal.

The Automated Mutual Assistance Vessel Rescue System (AMVERS) provides resources to help any vessel in distress on the high seas. AMVERS is the only worldwide safety network available to all authorized rescue centers. Participation is free, voluntary, and available to ships of all nations. More than 2700 merchant ships participate by being available to respond to emergencies at sea. The positions of all these ships around the world's oceans are tracked by computer and continuously updated. With these data, AMVERS creates a "surface picture" with the location of the ships in any given area. This vital information is used to deploy ships nearest the scene of the emergency. These ships are not designed for SAR and have crews generally untrained for recovering survivors from small boats or life rafts under storm conditions. Offshore, they may be the only rescue option.

In ship-to-ship rescue, collision between the vessels is the greatest risk. Be prepared to sacrifice your vessel in this situation. The typical scenario is of a large merchant ship with very limited maneuverability approaching a smaller vessel in distress with almost no maneuverability. Unless the rescue craft is designed for rescue work, the captain and crew experienced, and the seas relatively calm, it is much safer for the boats and crew to use a smaller craft to transfer personnel between the boats. The options include a rigid-hull inflatable boat (RHIB), a lifeboat, or even a life raft. In rough seas, never secure a boat or raft to the rescue vessel. The constant battering of the two hulls is likely to damage and may sink the smaller craft. Whether to approach the rescue vessel upwind or downwind depends on the wind, sea, and size and relative drift of the two vessels. The advantages and disadvantages are similar to those for retrieving a person overboard. Becoming pinned and capsizing are risks when sitting in the lee (downwind) side of a large, rolling ship.

The transfer of personnel between ships is also hazardous if sick, injured, or exhausted crew are required to climb a cargo net or pilot ladder. Under the best of circumstances, it can be extremely difficult even for a healthy crewman.

A large rescue ship most often approaches upwind of the survival craft unless the ship's rate of drift is much greater than that of the craft. This provides a calmer sea in the lee of the rescue vessel as it slowly drifts down to the survivors' craft. If the sea anchor is deployed, be sure to pull it in to prevent entanglement in the rescue vessel's propeller. Remain calm, and do not rush to board the ship. Wait to see if a lifeboat or rescue swimmer is lowered to facilitate the transfer. Attempt to com-

Figure 76-15. Coast Guard HH-60 Jayhawk helicopters carry a wide variety of gear to hoist victims and to assist crews in saving their vessels.

municate on channel 16 with the rescue craft to coordinate the rescue. Clarify and follow instructions carefully to ensure safety. If the raft is to be lifted aboard with injured survivors, be sure the floor is fully inflated. Attach the lifting lines to the towing bridles on both sides of the raft, and attach two steadying lines to each side as well.

Never attempt to scramble up a net, pilot ladder, or Jacob's ladder without a safety line. When boarding a rescue ladder, wait until the craft being departed is on the crest of a wave. At that moment, transfer to the ladder. This drops the survival craft down while you are ascending and eliminates the danger of the craft rising up on a wave and striking you. The safest procedure is to be hoisted up to the deck in a harness by a deck cargo crane.

Helicopter emergency evacuation and rescue has now become commonplace within 300 miles (483 km) of the coastline. A detailed briefing is radioed to the crew when the helicopter is en route to the vessel in distress. All loose gear onboard must be well secured, including cockpit cushions, coils of line, winch handles, dive gear, hats, and clothing. Any gear not secured on deck will become a flying missile in the 100-mph downdraft generated by the helicopter. This debris may be sucked into the intake of the helicopter's engine or become tangled in the rotor blades.

All crew on deck should wear life jackets. Add extra clothing layers because the helicopter's downdraft creates a windchill effect. Avoid shining flashlights on the helicopter; the light may blind the pilot and rescue team. For the same reason, never fire aerial flares in the vicinity of a helicopter.

The transfer device is either a rescue basket or a Stokes litter (Fig. 76-15). Selection depends on the victim's medical condition and the necessity to remain horizontal during the hoist. The horizontal position is particularly important for persons with spine injuries or severe hypothermia. A basket is the preferred device for lifting. It is easy to enter, especially in rough weather, and has positive flotation so that it will not sink. Just climb in it and fasten the straps. The basket will settle on the sea surface, enabling someone in the water to float into it.

A "horse collar" sling is a padded loop that is placed over the body around the back and underneath the armpits. The hoist is made with the line in front of the face. Always wear a PFD when entering the basket or hoist, and follow the directions for securing the safety straps.

The helicopter builds up static electricity traveling to the rescue scene, and the charge is transferred down the cable to the basket. Allow the basket to touch the deck or the water first to discharge any static electricity. Failure to follow this procedure will give one a strong, but nonlethal, electric shock. The orange steadying line, which is lowered first, is safe to handle and will not produce any shock.

Unhook the hoist cable only if it becomes necessary to move the litter below decks; let the cable free to be hauled back. When it is lowered again, allow the hook to ground on the vessel, and then reattach it to the rescue device. Never attach the hoist cable or the steadying line to any part of the vessel or life raft, even temporarily. The winch operator, who is intently watching the hoist cable, will react and instantly sever the cable from the hauling winch to prevent disaster.

If a helicopter picks up survivors from a life raft, the downdraft from the chopper's blades may capsize it, especially if the raft has small ballast pockets. Similarly, rafts loaded to capacity with crew become increasingly unstable as the occupants are removed and winched aboard the helicopter. The survivors should sit on the roof and on the inflated support arch to decrease the amount of surface exposed to the downdraft. Evacuate the strongest crew from the raft last.

The raft may also be used as an intermediate rescue platform between the distressed vessel and the helicopter. This is especially useful if the boat's mast and rigging interfere with the positioning of the helicopter or threaten to entangle the basket hoist. In this situation, the raft is allowed to drift downwind, attached to the distressed vessel by a line. The helicopter pilot cannot see the raft directly below. The winch operator therefore guides the rescue operation. When being winched up by cable and harness, follow directions to secure yourself, and keep on your life jacket. With an integrated safety harness and inflatable life vest, it is much easier to put on the helicopter's rescue harness when not encumbered by a bulky life vest. It may be preferable to leave the vest uninflated when being hoisted from the ship.

The helicopter has enormous potential for safe and effective SAR. The new HH-60 Jayhawk can fly 300 nautical miles offshore, remain on scene for 45 minutes, hoist up to six people on board, and return to base at 140 knots. Request this form of evacuation judiciously, especially if it is for transfer of sick or injured victims. Communicate medical data accurately so that the appropriate method of evacuation is selected. The C-130 Hercules is the largest of the U.S. Coast Guard SAR fleet, with a range in excess of 1000 miles (1609 km). It can air drop an enormous amount of lifesaving equipment, including dewatering pumps, life rafts, survival and signaling equipment. The most recognizable airplane in the CG fleet is the Falcon jet. This medium-range, fast-response plane flies at 350 knots and has a 2000-nautical-mile range. Sophisticated onboard electronics, including infrared scanners and surface search radar, allow the jet to fly a variety of search patterns on autopilot.

The references for this chapter can be found on the accompanying DVD-ROM.

77 Travel Medicine

Charles D. Ericsson

International travelers, particularly those participating in wilderness and outdoors activities, have unique health needs based largely on their underlying health and specific geographic destination.[70] Exposure to unfamiliar cultures, poor sanitation, and harsh environments may have a deleterious effect on health and interfere with the purpose and enjoyment of the trip.

The multidisciplinary specialty of travel medicine borrows principles from the fields of public health, infectious diseases, tropical medicine, and environmental and wilderness medicine. Travel medicine integrates features of these other disciplines with geographic and chronologic data, to formulate an approach to health risk assessment for a given journey.[74]

SOURCES OF INFORMATION

As a basic introduction to travel medicine, this chapter focuses on advice for the healthy adult U.S. traveler. Closely related topics are covered extensively in other chapters. Pediatric and unhealthy travelers are beyond the scope of this chapter. Excellent information about travel medicine–related vaccines for children has been published[24,79,93] and reference tables for both drugs and vaccines can be found at www.istm.org (see Appendix at the end of the chapter). A review on the approach to travelers with underlying medical conditions was recently published.[53]

The Centers for Disease Control and Prevention (CDC) publishes several authoritative sources of information on travel medicine. The "yellow book," *Health Information for International Travel,* is updated annually. Two other periodicals, the weekly *Morbidity and Mortality Weekly Report (MMWR)* and *Summary of Health Information for International Travel* (the "blue sheet," published biweekly), provide updated information on the status of immunization recommendations, worldwide disease outbreaks, and changes in health conditions. A reliable way to obtain current travel health information, including vaccine requirements, malaria chemoprophylaxis, and disease outbreaks for various regions of the world, is to consult the CDC Database of Health Information for International Travel website at www.cdc.gov/travel/yb/index.htm. For nonmedical information of interest to the traveler, the U.S. State Department can be accessed at http://travel.state.gov/. Additional resources for travel medicine information are given in the Appendix at the end of the chapter.

Professional societies, foundations, and private publishers are additional sources of telephone advice, topical brochures and information sheets, newsletters containing information on travel medicine topics, schedules of travel medicine meetings and continuing education courses, and other information, including lists of travel medicine clinics in the United States and abroad and English-speaking physicians worldwide. A convenient way to access both official and proprietary information sources can be found at the home pages of the International Society of Travel Medicine (www.istm.org) and the American Society of Tropical Medicine and Hygiene (www.astmh.org), where links can be found to the CDC, the World Health Organization (WHO) and other websites where the health needs for individual itineraries can be explored.

Standard textbooks on infectious diseases and tropical medicine and a number of monographs on travel medicine provide in-depth information on travel medicine–related subjects; many of these resources are listed in the suggested readings at the end of the chapter. A number of computer-based interactive programs for travel medicine clinics are commercially available. The best sources of current information on travel medicine software are published reviews and "hands-on" demonstrations at medical and scientific meetings.

American travelers abroad who experience an emergency of any sort should contact the nearest U.S. consulate or embassy, or call the U.S. Department of State (see Appendix). If an extended stay in a given country is planned, the traveler should register with the consulate or embassy shortly after arrival in the country.

Several months in advance of departure, travelers should ascertain whether their health insurance policy covers the costs of treatment and hospitalization for illness or injuries occurring abroad. They should consider a policy that covers emergency medical evacuation back to the United States, particularly if their travel involves remote locations where medical care is marginal or nonexistent. Medicare usually covers only health care expenses arising in the United States and its territories. Some credit card services provide worldwide medical referrals and arrangements for emergency transportation for their card-holders but do not actually cover the costs incurred. What the traveler needs is a short-term health insurance policy that specifically covers medical expenses and medical evacuation during foreign travel. Depending on the insurer, chronic medical conditions may be excluded, or they may be covered only if they are certified to be under control for a period of time before departure. Elderly travelers may find it more difficult to get medical insurance of this kind.

Box 77-1. History for Travel Risk Assessment

TRAVEL DETAILS

- Geographic itinerary
  Sequence of countries visited
  Urban vs. rural travel
- Duration and season of travel
- Style of travel and accommodations
  Airline and resort hotel vs. bus and camping
- Reason for travel and planned activities
  Packaged tour vs. business vs. adventure
- Access to competent medical care during travel

GENERAL AND SPECIAL PERSONAL HEALTH ISSUES

- General health status
  Age and weight
  Pregnant or lactating
  History of routine immunizations
  Allergies to drugs and vaccines
  Medications taken on a regular basis
- Special medical issues
  History of travel immunizations
  Impaired immunity from disease or treatment
  - HIV
  - Transplantation
  - Malignancy and its treatment
  - IgA deficiency
  - Asplenia
  - Use of immunosuppressive drugs, such as corticosteroids

  Underlying medical or physical conditions
  - Diabetes mellitus
  - End stage renal disease requiring dialysis
  - COPD requiring oxygen therapy
  - Heart disease, including recent MI
  - GI disease, including cirrhosis
  - Disability requiring special transport or accommodations

TRAVEL HEALTH RISK ASSESSMENT

Pretravel medical preparation is individualized by review of the geographic destination, duration and purpose of the trip, style of travel, underlying health of the traveler, and access to medical care during the trip (Box 77-1).[134] Information about the level of sanitation and environmental hazards at the destinations can be used to identify special health concerns; likewise, risk assessment includes whether the traveler might be exposed to extreme weather conditions, high altitude, or aquatic activities. In this context, immunizations and prevention and self-treatment of malaria and travelers' diarrhea are always addressed. Prevention and treatment of common ailments, such as jet lag, motion sickness, sun exposure, altitude illness, and insect and animal bites, should be reviewed when the issues are appropriate to the specific itinerary. Some attention, at least in patient education brochures, should be given to personal safety, sexually transmitted diseases, prevention of motor vehicle injury, and emergency medical evacuation.

Multiple destinations and travel lasting longer than a few weeks increase the complexity of the medical preparation; the contents of the travel medical kit become more inclusive with longer travel, in that a greater number of health needs over time can be anticipated. Travelers staying in urban air-conditioned hotels or in well-developed resorts have less exposure to mosquitoes than do those camping or living among residents in small villages. If accommodations in malarious areas are without screens, travelers should plan to take with them portable bed nets and permethrin-containing knock-down sprays against mosquitoes and other biting insects. Teachers, students, missionaries, relief workers, agricultural consultants, field biologists, and adventure travelers are more likely than persons on standard tourist packages and business travelers to be exposed to endemic infectious diseases. Such diseases include those transmitted by the local residents (e.g., hepatitis B, tuberculosis, and meningitis), insect-borne diseases (e.g., malaria, yellow fever, leishmaniasis, filariasis, plague, and typhus), and diseases associated with animal exposure (e.g., rabies, leptospirosis, and anthrax). Travelers need also to be aware of the risks of blood-borne infections from the use of contaminated needles, syringes, and other medical or dental devices, such as emergency dental care, injections, tattoos, and transfusions.

All travelers should be cautioned about the hazards of unprotected sexual contact with new partners, especially with commercial sex workers. Gonorrhea and chlamydial infection are common in the industrialized world but also have worldwide distribution. Human immunodeficiency virus (HIV) infection, syphilis, chancroid, and lymphogranuloma venereum are more prevalent in the developing world.

Most international travelers should begin pretravel medical preparation 4 to 6 weeks before their date of departure so that multidose immunization schedules can be completed, protective immunity developed, and necessary medications and special supplies obtained. Medical preparation for an international trip to many tourist destinations is relatively straightforward for people in good health. However, advance planning and consultation with a travel medicine expert are recommended for people with allergies, special health needs (e.g., pregnancy, infancy, advanced age, and handicaps), or chronic or underlying health conditions (e.g., cardiovascular disease, respiratory conditions, compromised immune status, diabetes, renal failure, organ transplant, and seizure disorders).

Vaccine-preventable diseases (e.g., diphtheria, measles, polio, hepatitis, and typhoid fever) and exotic infectious diseases (e.g., malaria, schistosomiasis, leishmaniasis, and trichinosis) are important; however, cardiovascular diseases and trauma account for more morbidity and mortality among American travelers and expatriates than do infectious diseases. This underscores the importance of addressing not only the travelers' health needs but also educating them about accident prevention.

Injuries are the leading cause of preventable death among travelers. Road traffic accidents account for the majority of injury-related deaths.[58,113,159] If travelers consider driving an automobile in a developing country, they must be aware of the possible lack of safety features on the rented vehicle, poorly maintained roads, and sometimes very different and aggressive behavior of local drivers. Even driving in a developed country can be hazardous when signs cannot be read or the traffic

pattern is reversed from what is customary for the traveler. If travelers ride bicycles, mopeds, or motorcycles, they should insist on wearing helmets. Travelers should avoid driving any vehicle at night and should never drink alcohol and drive. Injuries due to falls, drowning, animal bites, fire, and poisoning are also important sources of travel-related morbidity.

Travelers who participate in relief or other activities in regions affected by political unrest, war, terrorist activity, famine, or natural disaster represent special problems in preparation. Ideally, such travelers who know they might volunteer for such activities should try to prepare themselves well in advance and receive immunizations so that they are prepared for travel to virtually any part of the world. Likewise, such travelers might face unique risks when an outbreak of disease is occurring at the destination. For instance, if they must work where avian influenza is actively being transmitted, it is logical to consider oseltamivir prophylaxis or self-therapy in the event of a flulike illness. If an outbreak of plague occurs, they might consider taking doxycycline for both malaria prevention and protection against plague, because plague vaccine is not readily available and requires an extended series of injections. If SARS (severe acute respiratory syndrome) or one of the hemorrhagic viruses is a concern, they must plan to arrive with enough gloves and gowns, and the correct type of masks to practice appropriate infection-control precautions. Too frequently, volunteers who are anxious to help in the face of a disaster literally fly off poorly prepared to protect their own health.

All travelers should remain vigilant to help guarantee personal safety. They should consciously avoid risky situations. Up-to-date information concerning personal risk at destinations can be obtained from the U.S. Department of State website (www.state.gov). Sometimes overlooked among travelers to politically dangerous locations is the possibility of kidnapping. Such travelers might benefit from education about how to behave as a hostage (see Chapter 37). Likewise, women traveling alone or under difficult circumstances must consider the possibility of rape and might plan on the possibility of needing emergency contraception.[106]

HAZARDS OF AIR TRAVEL

Modern jetliners fly very high and represent a measurable risk for cosmic ray exposure to long-haul pilots and crew. The risk to the occasional traveler, however, is negligible.[14] While the cabin is pressurized to an altitude of approximately 5000 ft (1524 m), pilots are authorized to climb to avoid weather, so the cabin altitude can effectively rise to 8000 ft (2438 m). At this altitude everyone is slightly hypoxic; however, a patient with chronic obstructive pulmonary disease (COPD) can become dangerously hypoxic. A pulmonologist can expose the patient before traveling to 15% oxygen and determine whether supplemental oxygen might be necessary during the flight. Planning is necessary because airlines need several days' notice and not all flights offer supplemental oxygen. Travelers who have had myocardial infarctions are advised not to travel for at least 3 weeks after the event.[53,112]

Medical problems during flight relate mostly to barotrauma. Transmission of respiratory pathogens like *M. tuberculosis* is possible; however, air in a modern jet is HEPA-filtered, so risk is mostly to travelers seated near the infected person. Mosquitoes that might transmit diseases like malaria or dengue fever can be a problem on airliners, but such concerns are generally adequately dealt with by disinsection of the aircraft as it departs high-risk locations.

Jet Lag

Symptoms of fatigue, loss of concentration, and impaired performance and sleep can occur when the normal circadian rhythm is disrupted by travel across multiple (usually five or more) time zones.[14] In most persons, more symptoms occur and a longer period of adaptation is required with travel eastward than with travel westward. Without special interventions, the typical adjustment time is 1 day per hour of time zone change. Short-acting hypnotics, timed exposure to bright light, and ingestion of melatonin have been used to shorten the period of adaptation.

To avoid possible periods of amnesia associated with hypnotics,[90] the drugs should be avoided during flight and should only be used when the traveler can schedule uninterrupted sleep during the time that the drug is active. Any hypnotic (e.g., benzodiazepam, triazolam, or the nonbenzodiazepam drugs zolpidem, zaleplon, and eszopiclone) should be used in the lowest effective dose. It should be tried before travel to determine whether it is well tolerated. Hypnotics are recommended to be taken for the first several nights after arrival in the new time zone and after return to the old time zone. Alcohol should be avoided, because it interferes with REM sleep.

Exposure to bright light, which suppresses melatonin production by the pineal gland, has been advocated to suppress the "leftover, wrong-time" melatonin production after arrival at the new destination. For instance, after traversing 12 time zones, one's internal clock may still be beginning to produce melatonin at 6 AM in the new time zone because it "thinks" it is still 6 PM. Therefore, 6 AM in the morning at the new destination would be a good time to get exposure to bright sunlight. While this approach is logical, scientific data supporting its application are scant.

To facilitate adaptation, melatonin may be taken at the new bedtime in a dose of 2 to 5 mg for several nights after arrival.[61] In the United States, melatonin is a dietary supplement. Over-the-counter melatonin preparations may be contaminated with impurities and potency is not guaranteed. It is not available in many parts of the world and its use is not routinely advocated.[85]

Deep Venous Thrombosis

Deep venous thrombosis (DVT) and its associated risk for pulmonary embolism are recognized as potential complications of flights that last for 6 or more hours. DVT occurs more commonly in persons using oral contraceptives and with cardiovascular risk factors, active malignancy, or recent surgery.[10,56,81,87] Dehydration is also an important risk factor. Pulmonary embolism is rare and occurs in only 1 to 2 travelers per million long-haul flights.[76] On the other hand, up to 10% of travelers who were not using support stockings sustained asymptomatic DVT in the calf after a flight of 8 hours or longer.[126]

As safety permits during long-haul flights, travelers should be encouraged to move about the cabin periodically or at least isotonically exercise their calf muscles if turbulent weather forces them to remain seated. Keeping well hydrated ensures the necessity to move about the cabin in order to urinate. Avoiding alcohol helps prevent dehydration. Use of below-the-knee support stockings is encouraged. Low-molecular-weight heparin

could be used in high-risk travelers with the concurrence of the travelers' primary care physician; however, aspirin is not recommended for reduction of DVT.[28]

IMMUNIZATIONS FOR TRAVEL

Immunizations may be divided into three categories: required, recommended, and routine. Vaccine schedules and booster intervals for adult travelers who are assumed to have received primary series of routine vaccines as children are given in Table 77-1.[17,23,110] Vaccinations for traveling children can be found in tables referenced in the Appendix. The international traveler should have all current immunizations recorded in the WHO International Certificate of Vaccination. This yellow document is recognized worldwide and has a special page for official validation of the yellow fever vaccine. Recent copies of the document do not contain a separate page for cholera vaccine validation because the WHO officially removed cholera vaccination from the International Health Regulations in 1973. If given, cholera vaccination can be recorded in the space provided for "Other Vaccinations" in the newer booklets.

Contraindications to vaccinations are often overstated. An excellent review of situations that are not contraindications to vaccination is included in a recent book on travel vaccinations.[114] In general, live virus vaccines and attenuated bacterial vaccines are contraindicated during pregnancy and in persons with altered immunocompetence. Immune-altering conditions include underlying disease or conditions (e.g., malignancy, HIV infection, asplenia, congenital immune deficiency like IgA deficiency) and medical therapy (e.g., corticosteroids, cancer chemotherapy, radiation therapy, immunosuppressants for organ transplant). Although a comprehensive review of contraindications to immunizations by underlying host deficiency is beyond the scope of this chapter, a recent review of the subject is available.[53] Vaccination in the HIV-infected patient is outlined in Box 77-2, which in general terms is a reasonable approach to vaccination in most immunocompromised hosts in whom live virus and live bacterial vaccines should generally be avoided.

Required Travel Vaccines

The "required" immunizations refer not only to those regulated by the WHO but also to those required in practice. Yellow fever vaccine may be legally required for entry into some WHO member countries. Smallpox and cholera vaccines are no longer required for international travel according to WHO regulations; however, some countries continue to "require" cholera vaccine in practice. Meningococcal vaccine is required in practice by Saudi Arabia, particularly for persons arriving for the Hajj or Umrah pilgrimages.

Yellow Fever Vaccine

Yellow fever is a viral infection transmitted by mosquitoes in equatorial South America and Africa (see Figures 42-11 and 42-12). The yellow fever (YF) vaccine is a live attenuated viral vaccine that is highly protective.[89,160] YF vaccine is given as a single dose for primary immunization; the booster interval is 10 years. Because of age-related risk of encephalitis after immunization, most authorities agree that YF vaccine is contraindicated in infants less than 6 months of age, and immunization should usually be delayed until the infant is 9 months of age or older.[29] The vaccine is generally not recommended for persons who are immunocompromised or during pregnancy. If the pregnant traveler cannot avoid or postpone travel to a highly endemic area, then risk of the disease should be greater than the theoretical risk of adverse effects from the vaccine.

The vaccine virus is cultured in eggs and is contraindicated in persons with a history of an anaphylactic reaction to eggs. If a person can eat eggs without a reaction, he or she can receive YF vaccine.

Recently, vaccine-associated viscerotropic disease has been reported in a small number of first-time recipients of YF vaccine.[16] Many of the subjects died in fulminating multiorgan failure. Altered thymic function or thymectomy was documented in 4 of 23 cases and the syndrome may occur more frequently in persons older than 60 years of age; however, the small number of cases makes accurate risk factor assessment impossible.[7] The disease is thought to be infection by the attenuated vaccine strain facilitated by an altered host response rather than a change in virulence of the vaccine strain. Persons who have successfully received a first dose of YF vaccine are unlikely to be at risk with a booster dose if they have remained immunocompetent hosts.

Although YF-vaccine–associated viscerotropic disease is rare (approximately one event per 250,000 to 350,000 doses sold), the potentially deadly outcome prompts careful risk assessment especially when considering first-time vaccination in older travelers. Some countries may be listed as endemic for YF, but certain locations within the country may pose no risk. If a person for whom the vaccine is contraindicated (or ill-advised because the person is not truly at risk) must travel to a country where yellow fever vaccine is required for entry, then according to WHO regulations, a signed statement indicating that the YF vaccine could not be given because of medical contraindications should be acceptable in lieu of documented vaccination. The statement should be written on letterhead stationery and accompanied by authoritative stamps or seals, if available. Contacting the embassy or consulate of the country may be necessary to help guarantee that the letter of waiver will be accepted.

Cholera Vaccine

The injectable cholera vaccine is not highly efficacious, even when the primary series of two doses given a week or more apart is received.[115] The WHO no longer endorses a requirement for this vaccine for entry into any country. For countries that might still require cholera vaccination for travelers arriving from cholera-endemic areas, recording a single cholera dose in the travelers' International Certificate of Vaccination should suffice to meet this requirement.

Travelers going to areas endemic or epidemic for cholera are encouraged to follow food and water precautions gauged to prevent all forms of travel-associated diarrhea. Oral killed and live attenuated vaccines are available in some countries, but at present, there are no standardized recommendations for use of cholera vaccines, although health care workers and relief workers going to outbreak areas might be suitable candidates. Likewise, travelers to cholera endemic areas who have achlorhydria or have had partial gastric resection are logical candidates for cholera vaccine.

Smallpox Vaccine

The requirement for smallpox vaccine for international travel was removed from the WHO regulations in 1982. The CDC has embarked on an initiative to immunize health care providers, first responders, and others involved in bioterrorism

TABLE 77-1. Vaccines and Immunoglobulin for Adult Travelers Who Completed Childhood Immunizations

VACCINE	ROUTE (DOSE)	SCHEDULE	SIDE EFFECTS, PRECAUTIONS, AND CONTRAINDICATIONS*	COMMENTS
Hepatitis A (Havrix and Vaqta)	IM (1.0 mL)	Primary: single dose Booster dose at 6–12 mo for long-lasting protection Additional booster doses: not recommended	Local reactions: <56% Fever: <5% Headache: 16%	Prevaccine hepatitis A serology may be cost effective for some travelers (see text).
Hepatitis B (Recombivax and Engerix-B)	IM (adult and pediatric formulations)	Primary: 1 dose at 0, 1, and 6 mo Booster: not routinely recommended Accelerated schedules: see text	Local reaction: 3–29% Fever: 1–6%	
Hepatitis A and B antigens combined (Twinrix)	IM (1.0 mL)	Primary: 1 dose at 0, 1, and 6 mo Booster: not routinely recommended Accelerated schedules: see text	Local reactions: approximately 56% Systemic reactions: similar to single antigen products	Give at least 2 doses of vaccine prior to departure to provide protection against hepatitis A.
Immunoglobulin (Ig)	IM (buttocks)	Travel of <3 mo duration: 0.02 mL/kg Travel >3 mo: 0.06 mL/kg every 4–6 mo	Local discomfort: common Systemic allergy: rare	Should not be given <2 wk after or 3 mo before MMR, or varicella vaccines. Hepatitis A vaccine is preferred.
Influenza	IM (0.5 mL)	One dose of current vaccine annually	Local reactions: <33% Systemic reactions: occasional Allergic reaction: rare Avoid in persons with history of anaphylaxis to eggs	
Influenza (Flumist)	Intranasal (0.5 mL)	Primary: 1 dose per season	Mild upper respiratory symptoms: occasional Avoid in persons with history of anaphylaxis to eggs, Guillain-Barré syndrome, or immunosuppression	Approved for persons 5–49 yr
Japanese B encephalitis	SC (1.0 mL)	Primary: 1 dose at 0, 7, and 30 days Booster: 1 dose at 24-mo intervals	Local reactions: 20% Mild systemic reactions: 10% Allergic reactions (urticaria, rash, angioedema, or respiratory distress): approximately 0.6% Sudden death or encephalomyelitis: rare Avoid in pregnancy or in persons with multiple allergies.	Observe recipients for delayed allergic reactions for 30 min after each dose. Complete series ≥10 days before departure.
Measles (monovalent or combined with rubella and mumps [MMR])	SC (0.5 mL)	Primary: 2 doses separated by at least 1 yr Booster: none	Fever, 5–21 days after vaccination: 5–15% Transient rash: 5% Local reaction among persons who received killed vaccine (1963–1967): 4–55% Severe allergic reactions, CNS complications, thrombocytopenia (MMR): rare Avoid in pregnancy, immunocompromised hosts, and persons with history of anaphylaxis to eggs or neomycin	Do not give Ig within 3 mo of vaccine dose. If MMR and yellow fever vaccine are not given simultaneously, separate by ≥28 days.
Meningococcal polysaccharide-protein conjugate tetravalent vaccine (Menactra)	IM (0.5 mL)	Primary: Single dose Booster: Not determined	Local reactions: 10–60% Systemic reactions: occasional fever, headache, and malaise	Replaces tetravalent polysaccharide vaccine (Menamune).
Mumps	SC (0.5 mL)	Primary: 1 dose (usually as MMR) Booster: none	Mild allergic reactions: uncommon Parotitis: rare Avoid in pregnancy, immunocompromised hosts, and persons with history of anaphylaxis to eggs or neomycin.	Do not give Ig within 3 months of vaccine dose. If MMR and yellow fever vaccine are not given simultaneously, separate by ≥28 days.
Pneumococcal polysaccharide	SC or IM (0.5 mL)	Primary: single dose at age 65 or age 60 if high risk.	Mild local reactions: approximately 50%	Opportunity to update routine vaccination in older travelers.

TABLE 77-1. Vaccines and Immunoglobulin for Adult Travelers Who Completed Childhood Immunizations—cont'd

VACCINE	ROUTE (DOSE)	SCHEDULE	SIDE EFFECTS, PRECAUTIONS, AND CONTRAINDICATIONS*	COMMENTS
		Booster: high-risk patients after 5 yr	Systemic symptoms: <1% Arthus-like reaction with booster doses may occur Avoid in persons with moderate to severe acute illness	
Poliomyelitis	SC or IM (0.5 mL)	Booster: one adult dose	Local reactions: occasional	Additional boosters not recommended. Access CDC or WHO databases for current regions with polio transmission.
Rabies: human diploid cell vaccine (HDCV); purified chick embryo cell (PCEC); rabies vaccine adsorbed (RVA)	IM (1.0 mL)	Preexposure: 1 dose at 0, 7, and 21 or 28 days Booster doses depend on ongoing risk and results of serology (see text).	Mild local or systemic reactions: occasional Immune complex-like reactions after booster dose of HDCV (2–21 days after vaccination): 6%	Target children in endemic areas who might not tell parents about bites.
Rubella	SC (0.5 mL)	Primary: 1 dose (usually as MMR) Booster: none	Transient arthralgias in adult women beginning 3–25 days after vaccination: up to 25% Arthritis: <2% Avoid in pregnancy, immunocompromised hosts, and persons with history of anaphylaxis to neomycin.	Do not give Ig within 3 mo of vaccine dose. If MMR and yellow fever vaccine are not given simultaneously, separate by ≥28 days.
Tetanus–diphtheria (Td)	IM (0.5 mL)	Booster dose every 10 yr	Local reactions: common Systemic symptoms: occasional Anaphylaxis: rare Arthus-like reactions possible after multiple previous boosters Avoid if Guillain-Barré syndrome occurs ≤6 wks after previous dose.	Consider booster at 5 years for travelers to remote areas or regions without adequate health care facilities when sustaining punctures or other significant wounds is possible.
Tetanus-diphtheria acellular pertussis (Tdap)	IM (0.5 mL)	One dose to replace a Td dose	See Td	After one dose Tdap give Td every 10 yr
Typhoid Ty21a	Oral capsules	Primary: 1 capsule every other day for 4 doses Booster: every 5 years	Gastrointestinal upset or rash: infrequent Avoid in pregnancy and in persons with febrile illness, taking antibiotics, or immunocompromised state.	Refrigerate capsules. If already taking mefloquine, separate doses by 24 hr.
Typhoid Vi polysaccharide	IM (0.5 mL)	Primary: single dose Booster: every 2 years	Local reaction: 7% Headache: 16% Fever: <1%	
Varicella	SC (0.5 mL)	Primary: 2 doses at ≥4 wk interval No booster	Local reactions: 20% Fever: 15% Localized or mild systemic varicella rash: 6% Avoid in pregnancy and immunocompromised hosts, if severe allergic reactions to gelatin or neomycin or if serum immune globulin within 5 mo.	Rare transmission of vaccine strain to susceptible hosts; therefore, avoid if close contacts are immunosuppressed.
VZV vaccine (Oka/Merck)	SC (0.65 mL)	One dose	Local reactions: 33% Fever: 1.8% See Varicella for contraindications.	Consider for all adults ≥60 yr of age to lower the burden of herpes zoster and decrease the incidence of postherpetic neuralgia.
Yellow fever	SC (0.5 mL)	Primary: single dose Booster: every 10 yr	Mild headache, myalgia, fever (5–10 days after vaccination): 25% Immediate hypersensitivity: rare Viscerotropic syndrome or neurotropic disease: rare (see text) Avoid if allergic to eggs	If subject can eat eggs without a reaction he or she can take vaccine.

*Moderate or severe acute illness with or without fever or a serious reaction to a previous dose is a contraindication to all vaccines.
IM, intramuscularly; SC, subcutaneously.
Adapted from information in Health Information for International Travel 2004–5 (http://www.cdc.gov/travel/yb/index.htm) and Hill DR, Ericsson CD, Pearson RD, et al: The practice of travel medicine: Guidelines by the Infectious Diseases Society of America. Clin Infect Dis 43:1499–1539, 2006.

Box 77-2. Vaccination in HIV-Positive Adults

GENERALLY AVOID
VZV
BCG
Oral polio
Oral typhoid

AVOID IF CD4+ CELLS<200
- Yellow fever
- Measles

GIVE ROUTINELY
Tetanus/diphtheria (or dTaP, when approved)
Hepatitis B
S. pneumoniae
HiB
Influenza, yearly
Hepatitis A

GIVE IF INDICATED FOR TRAVEL
- Typhoid Vi
- Meningococcal
- Polio, IPV
- Rabies
- Japanese encephalitis
- Tick-borne encephalitis

preparedness, but the vaccine is not otherwise available and travel is not considered a sufficient reason for vaccination.[86]

Recommended Travel Vaccines

Recommended vaccines are those that are not routinely given during childhood in the United States but are given to travelers based on their travel health risk assessment. Vaccines in this category include those for hepatitis A and B, typhoid fever, meningococcal meningitis, Japanese encephalitis virus, rabies, tick-borne encephalitis, varicella zoster virus (VZV), influenza, and Bacillus Calmette-Guérin (BCG). Some vaccines have become routine in children (e.g., hepatitis B since 1991), so in the future, travelers will have been vaccinated. Likewise, in the future, travelers will already have received meningococcal vaccine as adolescents or young adults for entry into institutions for higher education or as 11- to 12-year-olds for whom the vaccine has just recently been recommended as a routine childhood vaccine. Arguably, varicella zoster virus (VZV) vaccine should be used routinely in the United States, but for many adult U.S. travelers it remains a vaccine that can sometimes be recommended. Influenza vaccine is often, but not routinely, used in children; each year it can be recommended for many travelers. While BCG vaccination is used in children in the developing world, it is not used in U.S. children.

Hepatitis A Vaccine

Hepatitis A virus (HAV) infection is the most common vaccine-preventable disease among international travelers to the developing world.[137,138,157] Without protection, HAV infection occurs in 1 to 10 persons among 1000 travelers at risk during 2 to 3 weeks of travel, and risk is high even among those residing in first-class accommodations. Adventure travelers who venture off usual tourist routes may be at increased risk compared with other groups of travelers. While HAV infection is asymptomatic in young children and self-limiting in most adults, it still accounts for greater than 2% mortality among adults older than 40 years of age, considerable morbidity during travel, and lost productivity after travel. Vaccination against HAV should be considered for all travelers to regions where sanitation and hygiene is poor.

Individuals who have a history of jaundice, who were born before the 1950s, or were born or resided for lengthy periods in endemic regions are likely to have natural immunity to HAV.[145] They should be screened for IgG antibodies against HAV, because if they are positive, they will avoid the cost of vaccination, which is usually more than serologic testing. However, because vaccination of HAV-immune persons is not associated with adverse consequences, a traveler who does not have time before departure to receive the testing results should simply be vaccinated.

A single dose of monovalent hepatitis A vaccine affords adequate protection by 7 to 10 days following vaccination.[21] Amelioration of HAV clinical infection occurs if serum immune globulin is administered up to 14 days after exposure to HAV. HAV challenge experiments in a simian model and indirect evidence in humans suggests that immunization with monovalent vaccine immediately before travel obviates the need for serum immune globulin.[118] Some authorities continue to advocate a dose of immune serum globulin (ISG) if HAV vaccination cannot be given sooner than 2 weeks before travel. Others feel ISG is seldom indicated except in immunocompromised persons who might not respond to the hepatitis A vaccine.[33]

HAV vaccine products are thought to be interchangeable. After two full doses separated by 6 to 12 months, protection is likely life-long, so booster doses are not recommended in immunocompetent travelers.[149] Travelers who fail to receive their second dose of HAV vaccine within 6 to 12 months should simply receive one full dose of monovalent vaccine with the anticipation of life-long immunity.

Hepatitis B Vaccine

Hepatitis B vaccine was added to the list of vaccines recommended for routine immunization of children in the United States in 1991, and consideration should be given to vaccinating all U.S. adults regardless of travel. Risk to short-term travelers is low; however, travelers should be vaccinated when they might have contact with body fluids or blood (e.g., through sex or medical work), when they anticipate receiving medical care in a developing country, or when they are frequent short-term travelers. Long-term travelers and expatriates should be vaccinated.[67]

Two recombinant vaccines are available and are thought to be interchangeable. The standard regimen for both vaccines is doses at 0, 1, and 6 months. One vaccine is approved by the U.S. Food and Drug Administration for an accelerated dosage schedule of 0, 1, and 2 months with a booster dose at 12 months for long-lasting protection. While a highly accelerated 3-week schedule is not approved, literature supports dosing at 0, 7, and 21 days with a 12-month booster.[13,80] This regimen affords 65% protection at the end of 1 month and is an attractive option for at-risk travelers who plan to depart in the next 3 to 4 weeks.

A combined hepatitis A and hepatitis B vaccine is now available and is dosed at 0, 1, and 6 months. Because a smaller dose of HAV antigen is used in this preparation, travelers must receive their second dose before traveling for reliable protection.

Literature also supports a highly accelerated 3-week dosing regimen with a 12-month booster.[98]

After any of the HBV vaccination regimens, additional boosters are not recommended for normal hosts. A protective amnestic response after exposure to wild type virus can be expected even after serum concentrations of antibody have fallen to undetectable levels.

Typhoid Fever Vaccine

The incidence of typhoid fever among American travelers is relatively low (1 to 10 cases per 100,000 travelers), but among reported cases in the United States, the majority were acquired during international travel.[88] Risk is highest among visitors to the Indian subcontinent and particularly high among those visiting friends and relatives. Visitors to Central and South America, Africa, and Asia should be considered for typhoid vaccination when they might be exposed to conditions of poor sanitation and hygiene, even for short periods of time.[34,141]

Increasing antibiotic resistance among *Salmonella enterica* serotype typhi is another reason to be vaccinated.[1] However, the current vaccines afford only 50% to 70% protection, and the emergence of *S. enterica* serotype paratyphi, against which protection is not afforded by the current vaccines, underscores the importance of hygienic food and beverage choices among travelers.[46]

Two vaccines are currently available and offer a similar degree of protection. The parenteral purified Vi polysaccharide typhoid vaccine is administered as a single injection with a booster recommended every 2 years. The oral Ty21a typhoid vaccine utilizes a live attenuated strain of *S. enterica* serotype typhi. One capsule is taken every other day for four doses. A booster regimen is recommended every 5 years.

Meningococcal Vaccine

Vaccine protection against meningococcal meningitis is recommended for long-term travelers to the sub-Saharan "meningitis belt" (Fig. 77-1).[111] Short-term travelers to this region should receive vaccine if they will travel during dry season (December to June) or have extensive contact with local people. Meningococcal vaccine is required for travel to Saudi Arabia during the time of the annual Hajj and Umrah religious pilgrimages. Regardless of travel, the classic recommendation has been to immunize young adults who will live in school dormitories, those with complement deficiencies, and persons with surgical or functional asplenia. Travelers to regions where outbreaks are occurring should be vaccinated. Practitioners who do not subscribe to commercial information services that are routinely updated should check the CDC website (www.cdc.gov/travel) periodically to learn the current locations of epidemic disease.

The quadrivalent meningococcal polysaccharide vaccine induces immunity against serogroups A, C, Y, and W-135. A single dose appears to provide immunity for at least 3 years. A booster every 3 years is recommended if the traveler continues to reside in high-risk locations.

A new quadrivalent meningococcal polysaccharide-protein conjugate vaccine has just been released for approved use in persons 11 to 55 years of age and approval is being sought for 2- to 10-year-olds. Compared to the polysaccharide vaccine, the conjugate vaccine promises longer-lasting immunity. It is now recommended by the Advisory Committee on Immunization Practices for routine use in 11- to 12-year-olds in the United States.[20]

Travelers who had previously been vaccinated with polysaccharide vaccine and need revaccination should receive conjugate vaccine. Whether routinely vaccinated children or travelers who remain at risk in meningococcal endemic regions might need a booster dose of conjugate vaccine at some time is a matter of ongoing research.

Japanese Encephalitis Virus Vaccine

Japanese encephalitis (JE) is a mosquito-transmitted viral infection prevalent in Asia and Southeast Asia. Transmission is year round in the tropical and subtropical areas and during the late spring, summer, and early fall in temperate climates. JE virus is not considered a risk for short-term travelers visiting the usual tourist destinations in urban and developed resort areas.[19]

Risk of infection can be greatly decreased by personal protective measures that prevent mosquito bites. For visitors to rural areas during the transmission season, the estimated risk for JE during a 1-month period is 1:5000, so vaccination should probably be offered to both long- and short-term visitors to rural areas during transmission season, particularly when mosquito exposure might be intense and when rice and pig farming occurs nearby.

The standard schedule for the inactivated viral vaccine is doses injected at 0, 7, and 30 days. An accelerated schedule of doses at 0, 7, and 14 days results in a lower rate of seroconversion. When risk of exposure continues, a booster dose of vaccine may be given every 2 to 3 years.

Adverse reactions to JE vaccine include local pain and swelling at the injection site in about 20% of recipients, systemic symptoms (fever, headache, malaise, rash) in about 10%, and hypersensitivity reactions at a rate of 0.1 to 5 per 1000 administrations.[11,144] The hypersensitivity reactions can rarely be fatal and can occur immediately or with delays of up to 2 weeks. Limited data suggest that persons who have had urticarial reactions to hymenopteran envenomation and to other stimuli might be at greater risk of JE vaccine–induced hypersensitivity reactions. JE vaccine recipients should be directly observed for 30 minutes after injection and should not travel until 10 days after their last dose because of the risk of delayed adverse reactions.

Rabies Vaccine

Although rabies is endemic in much of the world, the risk of rabies for most travelers is very low. Avoidance of dog bites would eliminate much of the risk.[156] Other animal species important to the transmission of rabies to travelers include monkeys, mongooses, bats, and foxes. Preexposure rabies immunization should be considered for rural travelers, particularly adventure travelers who go to remote areas, those with occupational (veterinarians) or recreational (spelunkers) exposure, and for expatriate workers, missionaries, and their families living in countries in which rabies is a recognized risk. Children should be specially targeted for preexposure vaccination in high-risk regions because they may not tell their parents that they have been bitten.

Any of the three tissue culture–derived inactivated virus rabies vaccines can be administered intramuscularly in a preexposure schedule of 0, 7, and 21 or 28 days.[18] For those who continue to be at risk of exposure, a booster can be given every 2 years or less, frequently based on annual serology testing. In the event of an exposure, the vaccinated traveler must understand how to clean the wound thoroughly and immediately seek

Figure 77-1. Meningitis belt. Consult details in the CDC Yellow Book for locations within each country where, and seasons when, pretravel vaccination is recommended. (From CDC Health Information for International Travel, 2005–2006, available at www2.ncid.cdc.gov/travel/yb/utils/ybGet.asp?section=dis&obj=menin.htm&cssNav=browseoyb.)

two additional doses of vaccine. Preexposure vaccination obviates the need for postexposure administration of rabies immune globulin (RIG), which is an important consideration because it may be very difficult to find in some regions.[73,105] Those who are exposed without preexposure rabies vaccination require both RIG and a five-dose course of rabies vaccine given on days 0, 3, 7, 14, and 28. Intradermal (ID) administration results in slightly lower and shorter-lasting protection, and is not routinely recommended. Also, chloroquine impairs the immune response, so an ID-administered preexposure series should be completed well before chloroquine prophylaxis begins.

Mild local reactions to rabies vaccine occur in about 20% of recipients and are self-limiting in 1 to 3 days. Up to 10% of recipients may develop mild fever, myalgia, headache, dizziness, or gastrointestinal upset. Much less commonly, allergic reactions characterized by urticaria, angioedema, and respiratory symptoms can develop, with serum sickness–like syndrome possible after doses of human diploid cell–derived vaccine. Sudden death and encephalomyelitis are rare.

Tick-Borne Encephalitis (TBE) Vaccine

TBE is a viral disease transmitted predominantly by the bite of *Ixodes* ticks during the spring and summer months in rural forested areas of central and eastern Europe, Scandinavia, and Siberia. Infection after ingestion of unpasteurized dairy products from infected cows, goats, or sheep can also occur.[82]

The two inactivated vaccines (Encepur [Novartis], and FSME–Immun [Baxter AG]) are not available in the United States.[37] The standard dosing regimen is three doses given over a year. Accelerated schedules exist, but doses would likely need to be administered in the destination country. Whereas expatriates can consider obtaining vaccine at their new location, it is much more practical for most travelers to at-risk areas to practice comprehensive tick precautions (using repellents and residual insecticides, wearing protective clothing, and performing careful tick checks) and to avoid consuming unpasteurized dairy products.

BCG Vaccine (Tuberculosis)

BCG is not recommended for most travelers, including expatriates. Persons going on short trips for tourism or business to developing countries in which tuberculosis is common among the indigenous population are not at great risk of contracting tuberculosis. However, expatriates or travelers who will live among foreign residents or work in foreign orphanages, schools, hospitals, or similar facilities are at significant risk of exposure to infection with tuberculosis.[31] Such travelers should be PPD skin tested before their trip, and if the test is negative, they should be retested yearly and 3 months after their return to a developed country. People whose skin test results convert to skin test positivity should be treated with isoniazid for 9 months, regardless of age.

VZV (Chickenpox)

VZV infection is common throughout the world. A traveler with a history of chickenpox can be considered immune. Many adults have had inapparent disease, so if time permits, lack of serum immunity should be documented before considering vaccination. Exactly who should be vaccinated is uncertain, but nonimmune long-term travelers to developing countries and short-term travelers likely to have contact with ill children probably should be vaccinated.

Influenza

For U.S. travelers, influenza is a potential risk when traveling during winter in the Northern Hemisphere; between April and September to the Southern Hemisphere; and year-round to the tropics. Travelers from diverse locations may be brought together during cruises, resulting in an outbreak of influenza during periods when influenza transmission might otherwise not occur.[149] Risk for acquiring influenza during long-haul flights exists if an ill person is seated near the traveler. For these reasons, influenza should be considered a travel-related infection and should be prevented.[59]

Because influenza vaccine antigen composition is based each year on projections of winter influenza activity in North America, the vaccine may not protect against strains circulating elsewhere in the world and may not be available for at-risk travel from the United States during late spring through early fall.[148] The current influenza vaccine is not protective against avian influenza A (H5N1) that has caused outbreaks, including fatalities, in Southeast Asia and China.[147,158] Travelers to locations where avian influenza might be transmitted should avoid visits to markets or farms or other activities that might bring them into close contact with fowl. Oseltamivir prophylaxis or self-treatment is not routinely recommended for dealing with the possibility of avian influenza, because most travelers should be able to avoid risky contacts. The approach does have merit, however, for at-risk persons going on cruises at times of the year when they cannot be vaccinated against influenza.

Other Vaccines

Anthrax vaccine is not recommended for travelers. A killed bacterial vaccine for plague exists, but the dosing schedule is long, and protection is uncertain. An alternative for select persons at risk for plague (e.g., field biologists) is a daily 100-mg dose of doxycycline, which can double as protection against malaria. The protective efficacy of this regimen against plague is inferred from treatment recommendations.

Routine Vaccines

Routine immunizations are those customarily given in childhood, then updated in adult life regardless of travel.[13,17] Visits to travel medicine clinics afford opportunities to update booster doses of routine immunizations, particularly among middle-aged and older travelers.

The routine vaccines currently recommended in childhood include those against tetanus, diphtheria, pertussis, measles, mumps, rubella, poliovirus, *Haemophilus influenzae* type b (Hib), and hepatitis B. Routine immunization schedules for children, including nuanced changes in routine schedules for traveling children, can be found in an excellent set of informational tables at www.istm.org. Pneumococcal vaccine should be routinely offered to adults at 65 years of age.

Diphtheria, Tetanus, and Pertussis Vaccine

Primary immunization in young children is accomplished with a combination vaccine containing acellular pertussis antigen. The diphtheria/tetanus vaccine (dT) classically used for booster doses in older children and adults has no pertussis component and a lower dose of diphtheria antigen. Absence of a pertussis booster for adults has led to waning immunity and susceptibility to pertussis.[25] Booster doses of dT vaccine given at 10-year intervals are recommended to maintain immunity. For adventure and other travelers to remote locations who might sustain puncture wounds and be unable to safely obtain a tetanus booster, a dT booster could be given after 5 years. Particularly for travelers to areas of the world where diphtheria remains a risk (e.g., most countries of Africa, Asia, and the Middle East, countries of the former Soviet Union, and focal areas of Latin America), care must be taken to recognize that the last "tetanus" booster might really have been tetanus toxoid alone without the diphtheria component.

Combination vaccines with acellular pertussis and adult dose of diphtheria (dTaP) are now approved for use in adolescents and adults in the United States. This vaccine should replace dT for use in all adults regardless of travel.

Poliomyelitis Vaccine

All traveling adults should have received a primary course of polio vaccine. Following vaccination campaigns around the world, the WHO has been able to declare some regions of the world polio-free (the entire Western Hemisphere, Europe, and the western Pacific)[22]; however, some regions continue to have

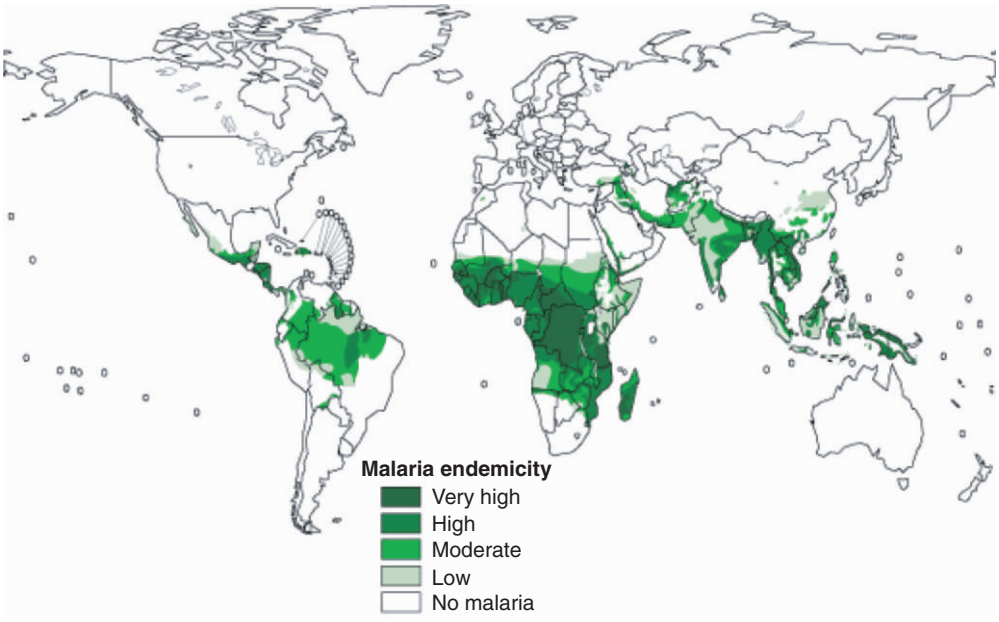


Figure 77-2. Worldwide distribution of malaria. (From WHO World Malaria Report 2005, available at http://rbm.who.int/wmr2005/.)

polio transmission because civil strife or political obstinacy has interfered with vaccination programs, because regional reimportation of polio virus has occurred, or because the vaccine strain has reverted to wild type and caused small outbreaks.[26,27] Travel medicine practitioners should check the CDC or WHO websites on a regular basis for latest information about polio status of travel destinations. Adult travelers to at-risk regions should receive a one-time booster dose of inactivated polio vaccine.

Measles, Mumps, and Rubella Vaccine

Not only is transmission of measles higher among populations in developing countries than in the United States, cases in the United States are now most related to importation of measles.[103] Travelers to developing countries are at risk of acquiring measles. If travelers have received two doses of measles vaccine, usually given in childhood as the measles, mumps, and rubella (MMR) vaccine, they are protected. Persons born after 1956 should receive a second dose of measles vaccine, which can be given as single antigen measles vaccine or as MMR vaccine.[13,17] Measles or MMR vaccines are live attenuated virus preparations. They are contraindicated in pregnancy and in immunocompromised persons.

Haemophilus influenzae B Vaccine

The risk for *Haemophilus influenzae* type B disease is the same in traveling children as it is in children resident in developed countries. Schedules for children can be found in pediatric travel information tables at www.istm.org.

Pneumococcal Vaccine

The pneumococcal polysaccharide vaccine should be offered routinely, regardless of travel, at 65 years of age, if not sooner depending on underlying disease and history of pneumonia. Many older travelers have not been vaccinated or high-risk vaccine recipients might not have been boosted, so the visit to the travel clinic is an ideal opportunity to bring this immunization up to date.

MALARIA

Malaria is a mosquito-transmitted, blood-borne, parasitic infection present throughout tropical and developing areas of the world (Fig. 77-2; see Chapter 43 for details). In the United States, approximately 1350 cases and several deaths are reported annually to the CDC. Although the risk to travelers is relatively low compared with other medical problems (e.g., diarrhea and respiratory problems), malaria is the most common preventable infectious cause of death among travelers and the most common cause of fever in the returned traveler. Most travelers who develop malaria have used no chemoprophylaxis, were prescribed inappropriate chemoprophylaxis, or were not compliant with their medication regimen.[124] Furthermore, travelers often fail to use appropriate personal protective measures. Visitors to friends and relatives (VFR) are now recognized as a group that disproportionally account for malaria occurring among travelers. They return to a developing region from a sojourn in a developed country, having lost much of their protective immunity. They must be counseled that they need to practice personal protective measures (PPM) and take medications in the same fashion as typical tourists or business travelers do after arrival from developed countries into a malarious region. Whereas the introduction of new antimalarial medication has somewhat facilitated ease and acceptance of chemoprophylaxis, rising drug resistance, breakdown in malaria control in some regions, and continuing reports of real or perceived adverse effects from antimalarial medication contribute to the difficulty of protecting travelers against malaria.

Malaria Risk Assessment

Assessment of risk for malaria requires knowing the details of a travelers' itinerary, including specifics of travel within a country. A geographic area may be listed as endemic for malaria, but urban travel may represent no appreciable risk, so chemoprophylaxis might be delayed until rural travel begins. Some destinations represent so little risk, and have no appre-

ciable risk for *Plasmodium falciparum* malaria, that when health facilities in the area are reliable, the traveler might only need to practice PPM and perhaps only consider taking along stand-by therapy if the duration of travel exceeds the minimal incubation time for malaria (approximately 1 week). The season of travel (dry vs. rainy) and the elevation of a destination influence risk. Type of accommodation (camping out vs. sleeping in an air-conditioned, well-screened room) influences whether to recommend permethrin products or bed nets. Duration of stay bears on how much medication to prescribe, but also which product (e.g., weekly mefloquine vs. daily doxycycline or atovaquone-proguanil for long-term travel). The medical history must address the possibility of known intolerance of an antimalarial, drug-drug interactions, and contraindications (e.g., mefloquine and depression).

Any travelers determined to be at-risk for malaria should be educated about how serious malaria is; what their risks are by their individualized itineraries; how to avoid being bitten by mosquitoes; how to take their antimalarial drugs compliantly; and how to seek medical care expeditiously if they develop fever during or after travel. Approximately 20% of cases of *Plasmodium vivax* malaria in travelers occur greater than 6 months after travel. A helpful mnemonic for travelers to learn is the "ABCs and D" of malaria prevention: *A*—awareness of risk; *B*—bite avoidance; *C*—compliance with drugs; *D*—diagnose promptly.

Insect Precautions

Exercising PPM against insect bites is arguably more important than is taking drugs for preventing malaria, in that only avoidance of mosquito bites will successfully prevent other mosquito-borne diseases such as dengue fever.[54,83,95] Likewise, resistance to antimalarial drugs is a concern in many regions of the malarious world; many chemoprophylactic regimens cannot completely prevent the liver phase of *P. vivax* infection from emerging and causing disease, despite an appropriate course of preventive medication. A pregnant traveler, for instance, might be obliged to take a suboptimal antimalarial drug regimen, further emphasizing the importance of PPM.

The malaria parasites are transmitted by the female *Anopheles* mosquito, which tends to bite between dusk and dawn, when travelers should limit time outdoors. They should wear protective clothing and apply insect repellents to exposed areas of skin while outdoors. They should sleep in well-screened or air-conditioned rooms or within bed nets, whose protective efficacy can be increased by the application of insecticide (e.g., permethrin). Treated bed nets and clothing retain residual insecticide activity for weeks (sprayed) to months (soaked).[127] In addition to repelling mosquitoes, permethrin insecticides are effective against gnats, ticks, chiggers, bedbugs, scorpions, centipedes, beetles, and flies. Protection in enclosed spaces may further be increased by burning permethrin insect coils or employing electric vaporizing mats. Permethrin is a chemical derivative of alkaloids (pyrethrums) that naturally occur in the chrysanthemum plant family. When permethrin is allowed to air dry before treated items are used, it is relatively nontoxic and suitable for treatment of external clothing (Fig. 77-3) and mosquito nets. It is not recommended for direct skin application, because skin hypersensitivity reactions may occur.

The most effective insect repellents for skin application contain *N,N*-diethyl-meta-toluamide (DEET) (see also Chapter 41).[54] Higher concentrations of DEET do not protect better; rather, protection lasts longer. Concentrations of DEET that afford a reasonable duration of protection are in the range of 30% to 40%. Particularly in concentrations less than 30%, DEET appears to be safe for use on infants and children older than 2 months of age. Care should be taken when one is applying DEET to avoid the eyes and mouth. When sunscreen and insect repellent need to be used simultaneously, the sunscreen should be applied first.

Malaria Chemoprophylaxis

Selection of malaria chemoprophylaxis is determined by the geographic destination and the pattern of drug resistance among malaria strains transmitted at the destination. As can be seen in Table 77-2, chloroquine is still appropriate for chemoprophylaxis in some regions of the world. For much of the malaria-endemic world, chloroquine-resistant *P. falciparum* (CRPF) malaria dictates the drug of choice. In a limited region of the world, chloroquine-, mefloquine-, and sulfonamide-resistant types of malaria are a concern. Chloroquine-resistant *P. vivax* (CRPV) malaria is widespread on the island of New Guinea (which comprises the Indonesian province of Papua [formerly Irian Jaya] and the nation of Papua New Guinea) and is also documented on Sumatra and other islands of Indonesia; on the islands of Vanuatu; in Myanmar; in Colombia; and in Guyana. Where CRPV malaria is a concern, recommendations for CRPF, which is also present in these regions, suffices for CRPV as well. Doses and schedules for drugs commonly used in malaria chemoprophylaxis are given in Table 77-3.

Travelers should start taking drugs with a weekly dosing regimen (chloroquine or mefloquine[77,122,136]) 1 or 2 weeks before departure. This allows time for familiarity with the side effects of the drug while the drug builds up to steady-state levels in the body, enables the traveler to habituate to the timing of doses, and gives the traveler time while at home to switch to an alternative drug in the event of intolerable side effects. Antimalarial drugs with a daily dosing schedule (doxycycline,[101,104] atovaquone-proguanil,[78,128,129,143] or primaquine phosphate[4,5,125,131,154]) are started 1 to 2 days before entering malarious areas. Chloroquine, mefloquine, and doxycycline chemoprophylactic regimens should be continued for 4 weeks after the traveler leaves a malaria area to prevent attacks of malaria in the immediate post-travel period. Atovaquone-proguanil should be continued for 7 days and primaquine should be continued for 2 days after leaving a malarious area. Travelers should be warned not to stop or switch antimalarial regimens during the trip without the advice of a knowledgeable health care provider.

In addition to geographic considerations, convenience of dosing regimen, and cost, adverse effects of the malaria chemoprophylaxis agents play a role in choice of one over another. Although it is inexpensive, doxycycline is associated with sun sensitivity rash.[55] Mefloquine has gotten considerable bad press for its neuropsychiatric side effects[12,32,153]; however; comparative studies show that the prevalence of mild or severe side effects is not appreciably dissimilar among the agents studied even if the specific side effects are different.[121] Nevertheless, because of past perceptions concerning side effects of other drugs and its relatively advantageous side effect profile, atovaquone-proguanil has become very popular, despite its expense.

Some travelers may not be able to take an optimal chemoprophylactic regimen. During pregnancy, doxycycline (FDA risk category D), atovaquone-proguanil (FDA risk category C), and primaquine (potential risks of hemolysis in the fetus) are contraindicated. Based on limited data, mefloquine is probably safe for use in the second and third trimesters of pregnancy, and if a pregnant traveler absolutely must travel to a CRPF region, expert consensus concurs it is probably safe in the first trimester

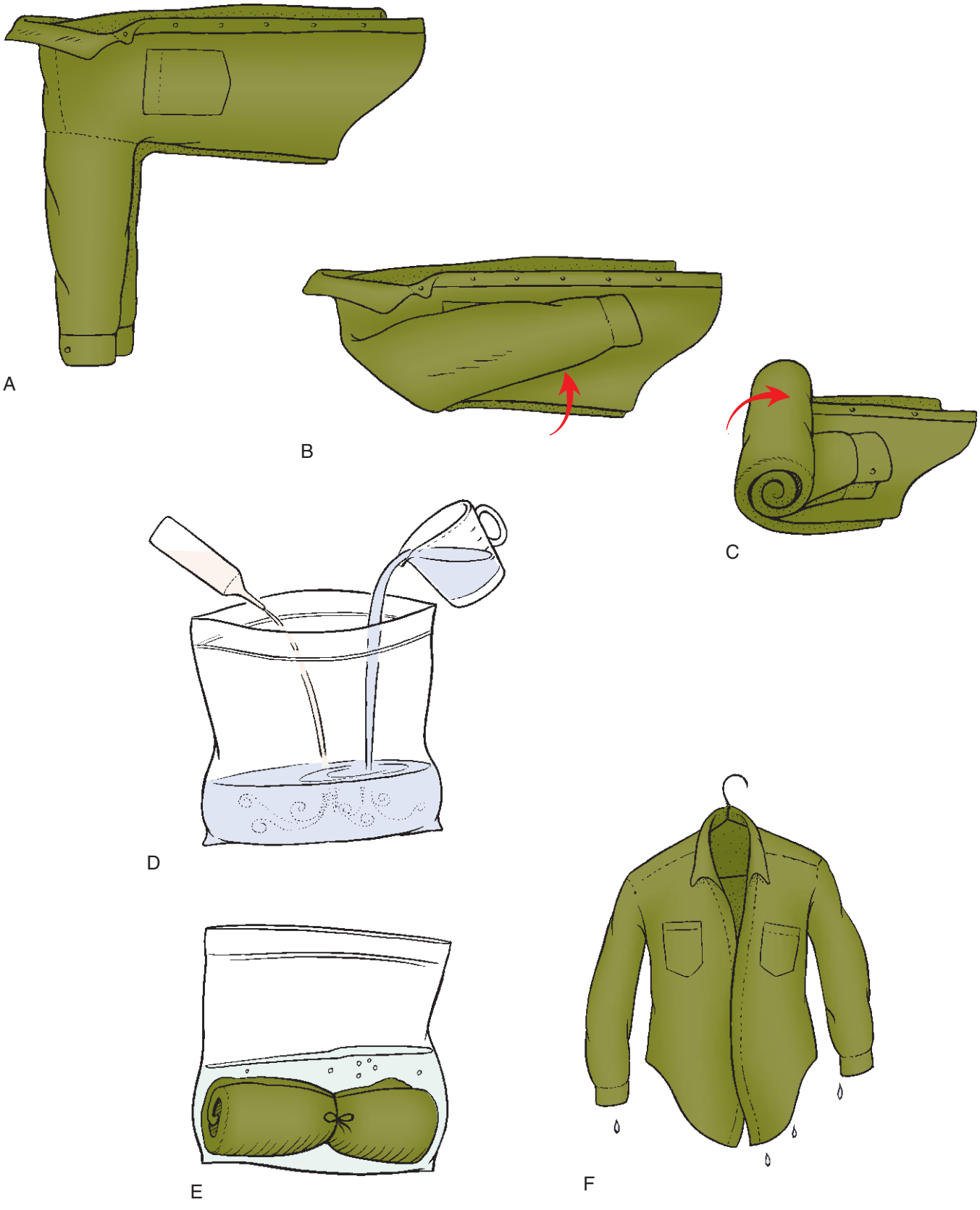


Figure 77-3. Technique for impregnating clothing or mosquito netting with permethrin solution. **A** to **C,** Lay jacket flat and fold it shoulder to shoulder. Fold sleeves to inside, roll tightly, and tie middle with string. For mosquito net, roll tightly and tie. **D,** Pour 2 oz (60 mL) of permethrin into plastic bag. Add 1 quart (1 L) water. Mix. Solution will turn milky white. **E,** Place garment or mosquito netting in bag. Shut or tie tightly. Let rest 10 minutes. **F,** Hang garment or netting for 2 to 3 hours to dry. Fabric can also be laid on a clean surface to dry. (Redrawn from Rose S: International Travel Health Guide. Northampton, MA, Travel Medicine, 1993, with permission.)

TABLE 77-2. Malaria Chemoprophylaxis Drugs by Regions of Drug Resistance

MALARIA DRUG RESISTANCE	COUNTRY OR REGION	DRUGS	
		PREFERRED	ALTERNATIVES
Chloroquine-sensitive	Central America (west of Panama); parts of Mexico; Haiti/Dominican Republic; most of the Middle East; states of the former Soviet Union; northern Africa; Argentina and Paraguay; parts of China	Chloroquine	Atovaquone-proguanil Doxycycline Mefloquine Primaquine Hydroxychloroquine
Chloroquine-resistant	Most of malarious South America, including Panama west of the former Panama Canal Zone; most of Asia and SE Asia; sub-Saharan Africa; Oceania; parts of Iran, Oman, Saudi Arabia, and Yemen	Atovaquone-proguanil Doxycycline Mefloquine	Primaquine
Chloroquine-, mefloquine-, and sulfonamide-resistant	Thailand borders with Myanmar and Cambodia	Doxycycline Atovaquone-proguanil	

TABLE 77-3. Malaria Chemoprophylaxis Drug Regimens for Adults

DRUG	DOSE	REGIMEN	COMMENTS AND ADVERSE EFFECTS
Chloroquine	500 mg/wk	Begin 1–2 wk prior to arrival in malarious area, continue drug weekly while in, and for 4 wk after leaving, the malarious area.	May exacerbate psoriasis. Pruritus (persons of African descent), headache, bitter taste: common Transient visual blurring, partial alopecia, skin eruptions, reversible corneal opacity: occasional
Hydroxychloroquine	200 mg/wk	As with chloroquine	As with chloroquine
Mefloquine	250 mg/wk	As with chloroquine	Take with food. Avoid concurrent alcohol. Avoid if history of depression, psychosis, seizures, or cardiac conduction abnormality. Dizziness, nausea, diarrhea, nightmares, insomnia, mood alteration, headache: common
Doxycycline	100 mg daily	Begin 1–2 days prior to arrival in malarious area. Continue drug daily while in and for 4 wk after leaving the malarious area.	Use sunscreen. Stains teeth (fetuses and children <8 yr). GI upset, photosensitivity, *Candida* vaginitis: common
Atovaquone-proguanil	One adult tablet (250 mg atovaquone/ 100 mg proguanil) daily	Begin 1–2 days prior to arrival in malarious area. Continue drug daily while in and for 7 days after leaving the malarious area.	Take with food. Avoid if CL_{cr} < 30 mL/min. Nausea, abdominal pain, headache: common Transient increase in transaminases: occasional
Primaquine	30 mg base daily	Begin 1–2 days prior to arrival in malarious area. Continue drug daily while in and for 2 days after leaving the malarious area.	Take with food. Avoid if G6PD deficiency. GI upset: common

GI, gastrointestinal; CL_{cr}, creatinine clearance; G6PD, glucose-6-phosphate dehydrogenase.

as well.[97,109,130,142,150] Doxycycline is contraindicated in children younger than 8 years of age. Although the combination of weekly chloroquine plus daily proguanil is considered safe in pregnant women and young children, protection during travel to a CRPF region is suboptimal. Also, proguanil is not licensed or marketed in the United States and must be purchased abroad.

Stand-by Self-Diagnosis and Drug Treatment

For travelers who spend prolonged time in very-low-risk areas where risks of chemoprophylaxis might arguably exceed the risk of malaria, the concept of self-diagnosis[68,99,155] and self-treatment,[120,123] instead of taking malaria chemoprophylaxis, has been popularized, particularly in European countries including Germany and Switzerland. The approach may also be appropriate on occasion for those taking suboptimal chemoprophylaxis or for those taking appropriate chemoprophylaxis who travel to remote areas where risk of breakthrough clinical illness due to resistant malaria is exceptionally high. This approach mandates that the traveler who develops illness and treats it with an antimalarial agent also seek knowledgeable

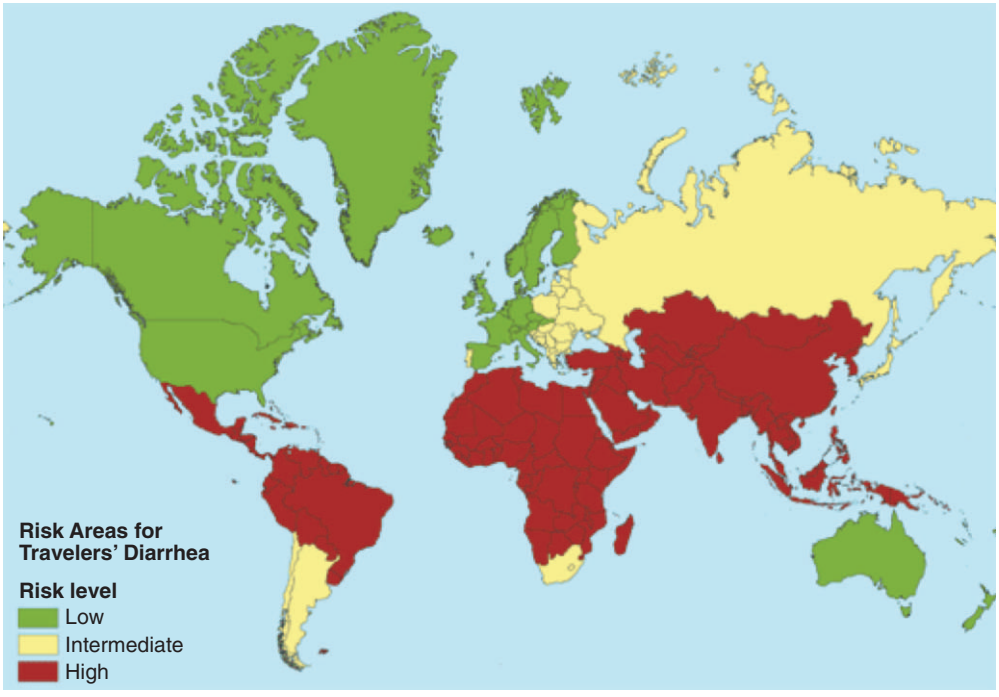


Figure 77-4. Risk areas for travelers' diarrhea. (From CDC Health Information for International Travel, 2005–2006, available at www2.ncid.cdc.gov/travel/yb/utils/ybGet.asp?section=dis&obj=travelers_diarrhea.htm&cssNav=browseoyb.)

medical care as soon as possible. The problem with self-diagnosis is that use of new rapid diagnostic tests by travelers in the field is fraught with a high false-negative rate of results. While the sensitivity and specificity of self-administered diagnostic tests can be improved in controlled settings (e.g., when instructions are clarified and the traveler is not ill), even if such tests become licensed in the United States, they cannot be recommended for routine use in U.S. travelers. Furthermore, when febrile travelers who elect to self-treat are evaluated medically, they often prove not to have malaria. Recent expert consensus concludes that self-diagnosis and self-treatment cannot routinely be recommended and should be considered only after expert consultation. In the United States, the currently preferred recommendation for self-treatment of suspected malaria is atovaquone-proguanil (four 250-mg tablets taken as a single daily dose for 3 days).

TRAVELERS' DIARRHEA

The term *travelers' diarrhea* (see also Chapter 62) usually refers to the abrupt onset of passage of soft or watery stools, accompanied by nausea, vomiting, abdominal cramps, or low-grade fever. Tenesmus and passage of bloody stools is uncommon. Rates of diarrhea for persons traveling from industrialized countries to developing regions are on the order of 40% to 60% over a 2- to 3-week vacation.[43,47,63,138,152] Whereas most travelers' diarrhea is self-limiting within 3 to 5 days, a substantial percentage of travelers will change their activities and some will be confined to bed, which has been the major reason for considering arming the traveler with drugs for self-treatment. Mounting evidence indicates up to a 10% prevalence of irritable bowel syndrome that persists 6 months after a bout of travelers' diarrhea, despite treatment of the initial bout of diarrhea with antimicrobial agents.[94] This finding, coupled with the largely disappointing status of vaccine prevention of enteric pathogens,[30,107] has prompted renewed interest in prevention of the syndrome through education and chemoprophylaxis.

Diarrhea associated with travel can result from multiple causes, including a change in the normal diet, food poisoning (toxins), and infection with viruses (rotavirus and norovirus) and parasites (*Giardia lamblia, Entamoeba histolytica,* and *Cryptosporidium*). However, bacteria (enterotoxigenic and enteroaggregative *Escherichia coli, Shigella, Campylobacter, Aeromonas, Salmonella, Vibrio*, and *Plesiomonas* species, among others) account for the majority of causes. The major causal role of bacteria explains the benefits of antimicrobial agents in treatment and prevention of the syndrome.

The world can be divided into high-, intermediate-, and low-risk regions for travelers' diarrhea (Fig. 77-4).[40,50,139] The risk of travelers' diarrhea is highest wherever food storage, fuel for cooking, and sanitation are inadequate. Contaminated water appears to be a risk factor for some viruses and parasites, but contaminated food is the most common risk factor. Inadequate public health practices appear to be a more important risk than contamination of specific food items.[62] As such, travelers find it difficult to prevent diarrhea only by attempting to exercise safe beverage and food choices. Nevertheless, travelers should seek locations of food consumption that have an excellent reputation for safety. Piping hot, thoroughly cooked food, dry food, and fruits and vegetables peeled by the traveler are generally safe. Tap water, ice cubes, unpasteurized fruit juices, fresh salads, unpasteurized dairy products, cold sauces and toppings, open buffets, and undercooked or incompletely reheated foods should be avoided.

TABLE 77-4. Recommended Agents for Travelers' Diarrhea Chemoprophylaxis

AGENT	DOSAGE	COMMENTS AND ADVERSE EFFECTS
Bismuth subsalicylate (Pepto Bismol)	Two tablets chewed four times a day	Avoid in persons who should not take aspirin or who are on anticoagulants. Black stool and tongue may occur.
Fluoroquinolones*		Occasional GI upset, rash, and allergic reaction.
Norfloxacin	400 mg PO daily	
Ciprofloxacin	500 mg PO daily	
Rifaximin	200 mg PO daily	Well tolerated because rifaximin is not systemically absorbed.

*Other fluoroquinolones can be predicted to be effective but they have not been studied in prophylaxis.

TABLE 77-5. Recommended Agents for Symptomatic Treatment of Travelers' Diarrhea

AGENT*	DOSAGE	COMMENTS AND ADVERSE EFFECTS
Bismuth subsalicylate (Pepto Bismol)	1 oz. (30 mL) PO every half hour for 8 doses	Delayed onset of action. Avoid in persons who should not take aspirin or who are on anticoagulants. May interfere with absorption of other antimicrobials, notably fluoroquinolones and doxycycline. Black stools and tongue may occur.
Loperamide (Imodium)	4 mg PO then 2 mg after each loose stool, not to exceed 8 mg daily	Rapid onset of action. Best results occur with loperamide plus an antimicrobial agent. Very well tolerated.

*See text for discussion of other agents.

Chemoprophylaxis of Travelers' Diarrhea

Bismuth subsalicylate–containing compounds and antimicrobial agents are successful in preventing travelers' diarrhea (Table 77-4).[40,50] Chemoprophylaxis might be a reasonable strategy for a traveler who is taking a brief trip to a high-risk area and cannot afford to be ill for even a single day. Traditionally, the benefits and safety of chemoprophylaxis with antimicrobials was weighed against obtaining prompt relief of diarrhea by the use of loperamide plus an antimicrobial agent. Travelers who might have been offered chemoprophylaxis included competitive athletes, politicians, business travelers, and people going to special events. The confirmation that up to 10% of those developing travelers' diarrhea have symptoms of irritable bowel syndrome up to 6 months later[102] and the availability of a safe, new antimicrobial agent, rifaximin,[42] have rekindled interest in the chemoprophylaxis of travelers' diarrhea.

Lactobacillus GG is a safe agent for prevention of travelers' diarrhea. However, it achieved only modest efficacy in one study and was less effective in others.[45,64] Likewise, other probiotics have demonstrated geographically spotty and inconsistent efficacy.[71,72] Probiotics cannot be recommended for routine prophylaxis of travelers' diarrhea.

Bismuth subsalicylate (BSS), which is active in large part because it is an antimicrobial agent, can successfully prevent approximately 65% of travelers' diarrhea.[38] Disadvantages to this regimen include cost, bulk of the BSS in luggage, the potential risks of absorbed salicylate, and development of a black tongue and stools. Also, BSS is not readily available in many parts of the world.

Trimethoprim-sulfamethoxazole can no longer be advocated for prevention of travelers' diarrhea owing to developing resistance worldwide. Fluoroquinolones successfully prevent more than 80% of travelers' diarrhea,[60] but a cogent argument can be made for reserving this class of agents for treatment of numerous conditions and not risk development of fluoroquinolone resistance through their use as preventive agents. Rifaximin is a newly approved, nonabsorbed antimicrobial agent with an excellent safety profile and usefulness only in the management of enteric diseases. A daily dose of rifaximin prevents over 70% of cases of travelers' diarrhea.[42] Emergence of resistance has not been a clinical problem. Although some experts still question the wisdom of routine chemoprophylaxis of travelers' diarrhea and favor self-treatment instead, prevention of irritable bowel syndrome following travelers' diarrhea has caused others to reconsider use of chemoprophylaxis of travelers' diarrhea in all short-term travelers.

Symptomatic Treatment of Travelers' Diarrhea

Fluid replacement has long been the cornerstone of therapy for travelers' diarrhea. Arguably, dehydration from loss of fluids and decreased oral intake is the biggest risk to health in young children and debilitated or older people. However, when loperamide was used to treat the syndrome, additional use of oral rehydration solution (ORS) did not add to the clinical benefit of loperamide alone.[15] Generally, pasteurized fruit juices or flavored mineral water suffice for oral rehydration during most episodes of travelers' diarrhea. Packets of oral rehydration salts, which can be reconstituted with clean water to make oral rehydration solution, are increasingly available in pharmacies throughout the world. Treatment with ORS is a wise consideration for diarrhea in children, elderly adults, and those who might develop diarrhea far removed from medical attention. In adults, a diet restricted to liquids and bland foods may not offer additional treatment benefit when diarrhea is also being treated with antibiotics.

Medications and their doses for symptomatic relief of travelers' diarrhea are listed in Table 77-5. Agents that offer no or insufficient relief include anticholinergics, adsorbents like kaolin-pectin preparations, and probiotics like *Lactobacillus*. Owing to insufficient information or lack of avail-

TABLE 77-6. Recommended Antimicrobial Agents for the Treatment of Travelers' Diarrhea

AGENT	DOSAGE	COMMENTS
Fluoroquinolones		
Norfloxacin	400 mg PO bid for 3 days	Occasional GI upset, rash, and allergic reactions. After 24 hours, patients can reevaluate themselves before taking next dose. If diarrhea persists or if fever or passage of bloody stools was present, finish 3 days of therapy.
Ciprofloxacin	500 mg PO bid for 3 days	
Ofloxacin	200 mg PO bid for 3 days*	
Levofloxacin	500 mg PO qd for 3 days*	
Azithromycin	1000 mg PO once *or* 500 mg daily×3 days	Unpublished data indicate a single 500-mg dose suffices when *Escherichia coli* is the predominant enteropathogen. Agent of choice for diarrhea occurring in southeast Asia.
Rifaximin	200 mg PO tid for 3 days†	Should not be used to treat persons with fever, with grossly bloody stools, or in whom an invasive enteropathogen is otherwise suspected.

*Single doses of ofloxacin and levofloxacin have been studied and appear equivalent to 3-day regimens. It is likely that single doses of any fluoroquinolone will suffice.
†400 mg PO bid for 3 days was also efficacious in one study.

ability, calmodulin or enkephalinase inhibitors cannot be recommended.

BSS reduces the number of stools passed in travelers' diarrhea by approximately 16% to 18%.[44,139] Although BSS can be recommended in mild diarrhea, for moderate to severe disease loperamide works better and with a faster onset of action.[69] The opiates and diphenoxylate are effective, but central nervous system and other side effects, plus poor tolerance among the elderly, limits their usefulness. Because it is safe and efficacious, loperamide has become the symptomatic treatment agent of choice.[51]

The combination of loperamide and an antimicrobial agent (e.g., a fluoroquinolone or azithromycin) is especially efficacious in the treatment of travelers' diarrhea.[2,48,49,52,108,146] Loperamide appears to be safe, even in children, as long as doses are kept in the recommended range and the drug is stopped if diarrhea persists despite several days of treatment. Most experts prefer, however, to avoid using loperamide in children. Even though the combination of loperamide and an antibiotic was more efficacious than antibiotic alone in the treatment of *Shigella* dysentery,[92] most experts prefer not to use loperamide when the patient has high temperature and passage of grossly bloody stools.

Antibiotic Treatment of Travelers' Diarrhea

Travelers' diarrhea can be relieved in a little over 1 day after empiric antibiotic therapy is instituted. With the concurrent use of loperamide, relief is realized in a matter of hours. With reports of increasing resistance among enteric enteropathogens worldwide,[65,117,151] trimethoprim-sulfamethoxazole can no longer be recommended. Until recently, one of the fluoroquinolones had been the drug of choice for empiric treatment of travelers' diarrhea. With recognition that *Campylobacter jejuni* is a common cause of travelers' diarrhea in Southeast Asia and of emerging resistance (up to 90%) to fluoroquinolones among *Campylobacter* in this region, the preferred agent for the empiric treatment of travelers' diarrhea has become azithromycin.[3,75] Rifaximin, a nonabsorbed agent with broad activity against enteric pathogens, is effective in the treatment of travelers' diarrhea in regions of the world where enterotoxigenic *E. coli* is the predominant pathogen.[39,41,140] However, it is not recommended for treatment of bloody diarrhea or when invasive pathogens are suspected, limiting its usefulness as a therapeutic agent in regions like Southeast Asia. The most appropriate use of rifaximin will likely be as a chemoprophylactic agent, because it can prevent diarrhea caused by invasive pathogens. Either a fluoroquinolone or azithromycin can be used for treatment of any illness that occurs despite prophylaxis. Recommended dosages of antimicrobial agents are shown in Table 77-6. Often a single dose suffices.[49,96,119] Travelers who do not respond to empiric antibiotic treatment or who have persistent diarrhea of more than 1 week's duration should seek medical attention.

HIGH-ALTITUDE ILLNESS

Acute mountain sickness (AMS) (see also Chapter 1) or the more dreaded complications, high-altitude cerebral edema and pulmonary edema, can occur when travelers ascend suddenly to altitudes greater than 3000 m (9800 ft), particularly if the traveler fails to acclimatize as higher altitudes are reached.[66,91] Up to 25% of persons who ascend rapidly to moderate altitude (1900 m to 3000 m [6200 to 9800 ft]) can develop at least mild AMS. Symptoms of AMS are usually experienced within 12 hours of arrival at altitude and are characterized by headache, fatigue, sleep disturbance, dizziness, and anorexia.[6,9] These symptoms often abate spontaneously over a few days as long as travelers do not further ascend and particularly if they are able to sleep at altitudes lower than those reached during the day. In the absence of previous experience at high altitude, the risk of AMS in the individual traveler is uncertain, although travelers with underlying hematologic, cardiac, or pulmonary disease should carefully consider whether or not to travel to high altitudes.

Acclimatization is the way to prevent AMS. Gradual ascent is critical. Ideally, travelers should spend a few days at altitudes below 3000 m (9800 ft), then gradually ascend above that level such that the elevation at which they sleep does not rise more than 300 to 500 m (1000 to 1600 ft) per night.

The carbonic anhydrase inhibitor acetazolamide (Diamox) hastens acclimatization by increasing ventilation, bicarbonate diuresis, and arterial oxygen levels.[36] A dose of 125 mg to 250 mg twice daily (or a single 500-mg dose of the extended-release formulation) can be taken 1 day before ascent and continued for at least 2 days once the highest altitude is reached.[8] Acetazolamide is contraindicated in persons allergic to sulfonamides. Extremity and circumoral paresthesias can occur.

Even though dexamethasone has been used for prevention, it does not hasten acclimatization; rather, it is usually reserved for treatment of altitude illness.[57] For experienced climbers who previously developed high-altitude pulmonary edema, nifedipine can be prescribed to help prevent a recurrence, but it is no substitute for staged ascent. While sildenafil (Viagra) increases maximum workload and cardiac output at high altitude, it cannot be recommended at this time for routine prophylaxis of AMS.

If mild AMS does not resolve spontaneously, particularly if acetazolamide is also being taken or symptoms actually worsen, then descent is imperative. Descending as little as 500 to 1000 m (1600 to 3300 ft) can be life-saving. Various interventions to control symptoms at high altitude have been tried (acetazolamide, dexamethasone, nifedipine, oxygen, and hyperbaric chambers), but these should only be employed by very experienced mountaineers in consultation with altitude experts. For all others, the solution is simple: descend!

ULTRAVIOLET (SUN) EXPOSURE

Sunscreens should be applied in sufficient quantities to achieve protection (see also Chapter 14).[84] At least 1 ounce (30 mL) is required to adequately protect an adult in a skimpy bathing suit. Sunscreen should be applied at least 30 minutes before exposure to the sun. Care must be taken to apply sunscreen under the chin and on any other body part that might be burned because of reflected UV rays (especially when one is on water or snow, which reflect a lot of rays). A sun protection factor of at least 15 is preferred, but SPF relates only to protection against UVB. UVA is also deleterious to skin, and only sunscreens with specific added ingredients will guarantee protection also against UVA rays. As a general rule, a product with SPF 30 or higher will often also provide UVA protection. While products vary somewhat in their ability to withstand water exposure, sunscreen should be reapplied at regular intervals after swimming, profuse sweating, or ongoing exposure to the sun. Sunblocks containing emulsified titanium dioxide are useful to protect particularly sensitive skin or to block UV exposure in persons taking doxycycline for malaria prophylaxis.

MOTION SICKNESS

Medications to prevent motion sickness should be taken before boarding moving vehicles in which a traveler might experience symptoms (see also Chapter 76). Oral scopolamine preparations are available and are convenient for short-duration travel.[132] For longer-duration travel (e.g., cruises and prolonged or frequent automobile or bus trips), transdermal, sustained-release scopolamine (Transderm Scop) may be employed. Scopolamine is contraindicated in persons with urinary obstruction or glaucoma and can cause drying of mucous membranes and drowsiness. Other medications for motion sickness prevention are meclizine and over-the-counter dimenhydrinate, but sedation can be a limiting factor. Wrist bands or other devices that provide electric stimulation or pressure to acupuncture points associated with controlling motion sickness may also be useful.

TRAVEL MEDICAL KIT

Travelers are advised to prepare a travel medicine kit appropriate to their itineraries. Such a kit should contain adequate supplies of all prescription medications normally taken, medications for malaria and diarrhea, and remedies for common problems such as headache, musculoskeletal pain, allergies, nasal and sinus congestion, cough, jet lag, and constipation. Travelers should be instructed to carry prescription medications in their handheld luggage and copies of their prescriptions; however, finding exact replacements for medications can be difficult in some parts of the world. A second supply of critically necessary medication might also be placed in checked luggage to guard against loss in the event that hand luggage is stolen.

Depending on the individual and the itinerary, insect repellent, sunscreen, topical antibiotic ointment, antifungal cream or powder, and medications for motion sickness, allergic reactions, and high-altitude illness might be included. Female travelers should be reminded to carry personal sanitary supplies, because disposable tampons and pads may be difficult to obtain in developing countries. Sexually active travelers of both sexes should carry a supply of high-quality latex condoms.

POSTTRAVEL MEDICAL CARE AND SCREENING

Even though travel medicine practitioners focus on pretravel advice and prevention and on self-treatment of disease and are not expected to be tropical medicine experts, they may be called upon for advice by returned travelers who have become ill. Physicians should be generally familiar with common and life-threatening posttravel conditions and how to triage posttravel complaints.[135] Particularly when the traveler presents with only constitutional symptoms, the differential diagnosis may be broad but can often be narrowed by considering incubation time, geographic area of disease acquisition, immunizations received, malaria chemoprophylaxis taken, dietary habits, insect and rodent exposure, animal bites, and intimate or sexual contact with foreign residents or fellow travelers.

The approach to posttravel illness has recently been reviewed in detail.[100,116,133] In a traveler who develops high temperature weeks, months, or even years after travel in a malarious area, malaria should be actively sought and treated.[35] Many physicians practicing in developed countries are unfamiliar with the clinical presentation of malaria and may fail to include malaria in the differential diagnosis. Likewise, typhoid fever is another relatively common cause of fever in the traveler recently returned from a developing country.

Diarrhea that develops in the first week or so posttravel can be assumed to be travelers' diarrhea and treated empirically. Persistent or remittent diarrhea should prompt a diagnostic workup. Antibiotic-resistant bacterial enteropathogens, intestinal parasites, or even hepatitis may account for prolonged symptoms after return home. A bout of travelers' diarrhea may unmask inflammatory bowel disease, and persistent symptoms raise concern for coincidental biliary tract disease or even intestinal malignancy.

Returned travelers may complain of unusual skin lesions or rashes. Tropical infections that might present with cutaneous manifestations include cutaneous myiasis, cutaneous larva migrans, and cutaneous and mucocutaneous leishmaniasis. Because secondary bacterial infection can occur with these conditions, the skin lesion may initially improve with therapy for bacterial skin infection. However, if the lesion fails to resolve completely, consultation with a tropical medicine specialist is recommended.

Many parasitic infections may have indiscriminate signs and symptoms. Eosinophilia may be a clue for some parasitic diseases. Nonspecific symptoms in a recently returned traveler, in whom common non–travel-related illness has been excluded, probably should be referred for expert evaluation.

SUGGESTED READINGS

DuPont HL, Steffen R (eds): Textbook of Travel Medicine and Health, 2nd ed. Hamilton, Ontario, BC Decker, 2001.
Ericsson CD, DuPont HL, Steffen R (eds): Travelers' Diarrhea. Hamilton, Ontario, BC Decker, 2003.
Freedman D (ed): Travel medicine. Infect Dis Clin North Am 12:543–547, 1998.
Hill DR, Ericsson CD, Pearson RD, et al: The practice of travel medicine: Guidelines by the Infectious Diseases Society of America. Clin Infect Dis 43:1499–1539, 2006.
Jong EC (ed): Travel Medicine. Med Clin North Am 83:903–922, 1999.
Jong EC, Zuckerman JN (eds): Travelers' Vaccines. Hamilton, Ontario, BC Decker, 2004.
Keystone JS, Kozarsky PE, Freedman DO, et al (eds): Travel Medicine. London, UK, Mosby, 2004.
Schlagenhauf P (ed): Travelers' Malaria. Hamilton, Ontario, BC Decker, 2004.
Spira A: Preparing the traveller. Lancet 361:1368–1381, 2003.

APPENDIX: RESOURCES FOR TRAVEL MEDICINE INFORMATION

Telephone Information

- Centers for Disease Control and Prevention (CDC) Travelers' Health Hotline: 877-FYI-TRIP
- United States Department of State Overseas Citizens' Services: 888-407-4747

Official References

- Centers for Disease Control: Health Information for International Travel, 2004–5, Washington, DC, US Government Printing Office (revised annually). Telephone: 202-738-3238
- World Health Organization: International Travel and Health, Vaccination Requirements and Health Advice, 1999, World Health Organization Publications Center USA, 49 Sheridan Avenue, Albany, NY 12210 (revised annually)
- Morbidity and Mortality Weekly Report, Centers for Disease Control and Prevention, Atlanta, GA 30333; subscriptions available at the website www.cdc.gov

Pretravel Clinic Directories

- International Society of Travel Medicine, website www.istm.org
- American Society of Tropical Medicine and Hygiene, website www.astmh.org

Travelers' Clinic Directory

English-Speaking Physicians: International Association for Medical Assistance to Travelers (IAMAT), 1623 Military Rd. #279, Niagara Falls, NY 14304-1745. Telephone: 716-754-4883

Reference Tables for Pediatric Travel

- Access www.istm.org, then at the left-hand column click on "Education and Training," then "Education and Scientific Information," then "Drugs and Vaccines for Pediatric Travelers: an Integrated Table."

The references for this chapter can be found on the accompanying DVD-ROM.

78 Non–North American Travel and Exotic Diseases

James W. Kazura

Travelers to tropical and subtropical areas of the world where hygienic conditions are poor and ecologic conditions are permissive may encounter infectious agents that are no longer endemic or have never existed in temperate regions of the world. Although economic development and industrialization of developing countries of the tropics have resulted in a decreased health burden of many tropical infectious diseases, it is important to realize that there is still a risk of exposure for the traveler who is unaware of appropriate measures to prevent or treat such conditions. The most important consideration in the management of this problem, which is increasing as international travel expands, is appropriate preventive measures through counsel with a travel medicine specialist and prophylaxis using safe drugs and vaccines. This topic has recently been reviewed in several excellent publications.[6,16,35,38,45,69]

This chapter is concerned with infectious diseases that are uncommon or do not exist in North America and with which most health professionals in North America have scant familiarity. Other chapters give specific details relevant to malaria (Chapter 43), tick-borne diseases (Chapter 45), infectious diarrheas (Chapter 62), and travel medicine (Chapter 77). The infectious diseases considered in this chapter should not be con-

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- World Health Organization: International Travel and Health, Vaccination Requirements and Health Advice, 1999, World Health Organization Publications Center USA, 49 Sheridan Avenue, Albany, NY 12210 (revised annually)
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This chapter is concerned with infectious diseases that are uncommon or do not exist in North America and with which most health professionals in North America have scant familiarity. Other chapters give specific details relevant to malaria (Chapter 43), tick-borne diseases (Chapter 45), infectious diarrheas (Chapter 62), and travel medicine (Chapter 77). The infectious diseases considered in this chapter should not be con-

sidered a complete listing. This is especially important to keep in mind in an era when diseases once thought to be eliminated or nonexistent in North America are emerging or reemerging coincidental with large-scale movements of human and vector populations.

MAJOR VIRAL INFECTIONS

A vast number of viruses cause human disease. This section describes selected viral infections that may be acquired outside North America. Emphasis is placed on viral infections that have recently been recognized as being highly pathogenic and endemic in selected tropical areas, such as those due to filoviruses, and those that for which there are effective preventive or therapeutic measures, such as viral hemorrhagic fever due to the yellow fever virus, selected types of viral hepatitis, and Japanese B encephalitis.

Major Viral Hemorrhagic Fevers

A diverse group of ribonucleic acid (RNA) viruses can produce the hemorrhagic fever syndrome. Fever, headache, myalgia, and malaise generally characterize the early phase of the clinical presentation of all these infections, which develops over several hours to 3 to 4 days. In the full-blown hemorrhagic fever syndrome, the somewhat nonspecific clinical manifestations are followed by hemorrhagic signs that include petechiae and bleeding from the gums and gastrointestinal tract. Loss of plasma volume and a capillary leak syndrome may ensue, manifested as increased hematocrit with hypotension and shock in some individuals. Elevated blood urea nitrogen and creatinine levels indicative of renal dysfunction may also develop. Death is caused by a combination of intractable hypotension, bleeding, electrolyte imbalances, and renal failure. There are many viral causes of this syndrome but these are not discussed here (e.g., Rift Valley fever). In general, the management principles are the same and consist primarily of supportive therapy.

Yellow Fever

European physicians did not recognize until the late 1490s the clinical syndrome now known as yellow fever. Initially described by Columbus in the West Indies, large-scale epidemics were later observed throughout the Americas and tropical Africa in the 1700s and 1800s. After epidemic yellow fever in Texas, Louisiana, and Tennessee caused 20,000 deaths in the 1880s, the Yellow Fever Commission was organized to study the problem. Identification of the mosquito vector, *Aedes aegypti,* and definitive studies conducted by the United States military under the leadership of Walter Reed were followed by massive campaigns to eradicate mosquito breeding sites. This led to virtual elimination of urban yellow fever from the Americas. The last case of yellow fever acquired in the continental United States was reported in 1911. Because it is difficult if not impossible to eliminate jungle reservoirs, there continue to be cases reported annually from South America and tropical Africa, respectively. Larger outbreaks secondary to resurgent vector populations have occurred in recent years in tropical West Africa.[4,22,52]

Virology and Pathophysiology. Yellow fever is a single-stranded RNA flavivirus. Strain differences are of little clinical relevance, although they may be of use in epidemiologic studies. The pathophysiologic mechanisms operating in viral hemorrhagic fevers are not well defined. In general, viral replication occurs at the site of inoculation. After the virus spreads to lymph nodes and monocyte-rich organs, further reproduction results in massive viremia.

The liver is the principal target organ. Pathologic studies show coagulative necrosis of hepatocytes and the appearance of various markers of cell involvement (Councilman and Torres bodies). However, the degree of physiologic derangement is usually much more severe than expected for the extent of hepatic damage seen on pathologic examination. Perivascular edema and occasional focal bleeding occur in the kidneys, heart, and brain, but these changes are less severe than expected for the degree of clinical disease.

Ecology and Epidemiology. In the Americas, primates in the forest canopy serve as hosts for the yellow fever virus. Mosquitoes of the genus *Haemogogus* transmit infection. Since this vector does not travel far from the forest, jungle yellow fever occurs when humans enter jungle areas or the forest border zones. Urban yellow fever involves a different vector, *A. aegypti.* This mosquito is highly anthropophilic, lives in and around human habitations, and prefers domestic water storage containers for breeding. The presence of a large population of *A. aegypti* breeding sites in an urban area is a significant risk for epidemic spread of yellow fever once the virus is introduced from a nearby forest area. In Africa the presence of larger numbers of mosquito species that can serve as vectors has hindered complete understanding of the ecology of the disease.

Currently, both the Americas and Africa have a constant low level of jungle yellow fever because of inability to control either the monkey reservoir or the mosquito vector. Overall there are about 2000 cases per year in Africa and 200 per year in the Americas.[76] Some suggest that these rates are underestimated by at least tenfold. Persons at risk include workers or travelers in or near the tropical rainforest canopy. Urban yellow fever had been reduced in the Western Hemisphere through massive campaigns to control the breeding and spread of the *Aedes* vector. However, the benefits of these campaigns have declined, and there is currently an increased threat of further outbreaks of disease. Introduction of *Aedes albopictus,* an aggressive anthropophilic dengue vector from Southeast Asia, and reemergence of *A. aegypti* into the Americas raise the specter of increased yellow fever transmission in the Western Hemisphere.[50] Less intense vector control measures and a more complex ecology have made eradication of urban yellow fever in Africa even more difficult.

Clinical Presentation. Although yellow fever may appear as an undifferentiated viral syndrome, classic disease is characterized by a triphasic pattern. The infection phase begins with sudden onset of headache, fever, and malaise, often accompanied by bradycardia and conjunctival suffusion. After approximately 3 to 4 days, victims often experience brief remission. Within 24 hours, however, the intoxication phase develops, characterized by jaundice, recrudescent fever, prostration, and, in severe cases, hypotension, shock, oliguria, and obtundation. Hemorrhage is usually manifest as hematemesis; however, bleeding from multiple sites may occur. Signs of a poor prognosis include early onset of the intoxication phase, hypotension, severe hemorrhage with disseminated intravascular coagulation (DIC),

renal failure, shock, and coma. Death occurs in one quarter to one half of all cases.[76] Diagnosis in the infection phase is difficult. With development of the classic syndrome, the differential diagnosis narrows somewhat, but still includes malaria, leptospirosis, typhoid fever, typhus, Q fever, viral hepatitis, and other viral hemorrhagic fevers. The standard means of diagnosis is evaluation for neutralizing antibodies in acute and convalescent sera (available through the Centers for Disease Control and Prevention [CDC] and state health departments in the United States). Several new systems for early detection of immunoglobulin M (IgM) or viral antigen are now being evaluated for more rapid diagnosis. A specimen of whole blood (at –70° C [–57° F] on dry ice) should be sent to the state health laboratory for isolation. Growth of the virus is possible in a number of systems, including Vero cells and infant mice. The virus is most easily isolated during the first 4 days of fever.

Management. Appropriate management of viral hemorrhagic fevers requires awareness of the geographic distribution of the disease and travel history of the victim. In the first several days of infection, differentiation of a viral hemorrhagic fever from other infectious diseases is nearly impossible. However, the occurrence of an undifferentiated febrile syndrome in a traveler from a yellow fever–endemic area warrants a careful physical examination, thick and thin blood smears to rule out malaria, and blood cultures for bacterial pathogens *(Salmonella typhi)*. In recently returned travelers, dengue serology should be considered. Progression to the intoxication phase or any sign of volume disturbance, renal failure, or hemorrhage mandates immediate admission to an intensive care unit.

There are no effective antiviral therapies for yellow fever. Supportive care should address several important problem areas. Fever should be controlled with acetaminophen, not with salicylates. Evaluation of volume status should include Swan-Ganz or central venous pressure monitoring to direct volume expansion and use of vasopressor agents. Calculation of fractional excretion of urinary sodium may help differentiate prerenal azotemia (for which volume expansion is appropriate) from acute tubular necrosis (which may require temporary dialysis). Plasma volume loss may contribute to serious electrolyte disturbances, necessitating close observation of serum electrolytes and arterial blood gases. Rapid correction of these abnormalities, which commonly include hyperkalemia and acidosis, is essential. Hypoglycemia is presumably a result of impaired hepatic gluconeogenesis.

Serial coagulation studies (including platelet count, prothrombin time, partial thromboplastin time, fibrinogen, and fibrin degradation products) should be obtained. Whole blood or fresh-frozen plasma is indicated to maintain the prothrombin time and hematocrit value at near normal levels. If DIC is documented, the use of heparin may be considered, although no controlled trials have documented the effectiveness of heparin in yellow fever. To reduce the risk of upper gastrointestinal hemorrhage, H_2 blockers have been suggested. Although the efficacy of intensive care treatment has not been studied for victims of yellow fever, clinical experience with dengue shock syndrome suggests that similar benefits might be obtained.

Prevention. Avoidance of this potentially fatal infection is possible through use of the yellow fever vaccine. The vaccine strain 17D is an attenuated live virus grown in chicken embryos. Greater than 95% of persons vaccinated achieve significant antibody 1evels. Repeat vaccinations are recommended every 10 years, although persistent antibody titers have been detected as long as 30 to 40 years after vaccination. Yellow fever vaccine is generally well tolerated, with headache or malaise occurring in less than 10% of those vaccinated. Rare allergic side effects occur primarily in persons with hypersensitivity to eggs. Other serious adverse events, including death, have been reported, with the greater risk being associated with age older than 60 years.[36,43,44,51] Vaccination is not recommended in the first 6 months of life or in other situations where live virus vaccines are contraindicated. Although pregnant women have received the vaccine without adverse effect to themselves or their infants, it is not recommended for use in this group because of possible teratogenic effects. Other means of reducing the risk of yellow fever (and any mosquito-borne infectious disease) include liberal use of mosquito repellent and netting in endemic areas.

Treatment of severe yellow fever is difficult and often unsuccessful. Avoidance through mosquito protection measures and administration of the highly effective vaccine before entry into endemic areas is of utmost importance.

Dengue

Dengue fever has been reported since the late 1700s. Since World War II, increased attention has focused on the dengue virus, largely as a result of recognition of dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS). First noted in Southeast Asia, DHF and DSS have attained worldwide distribution in the last 30 years.[22,23,40,74] Dengue is the most common insect-borne viral infection in the world. The infection has been reported in more than 100 countries, with DSS and DHF estimated to affect 100 million and 250,000 persons, respectively, annually.

Virology and Pathophysiology. The etiologic agent is a single-stranded RNA flavivirus, which may be one of four serotypes, denoted DEN1 through DEN4. As with yellow fever, local viral replication is followed by dissemination to lymphocyte- and macrophage-rich areas, where most of the reproductive activity occurs. Infection with one virus serotype provides long-lasting protection against that type only. For the DEN2 serotype, previous infection with a heterologous serotype may result in a more severe clinical course than noted in those experiencing dengue 2 infection without such a history. Non-neutralizing antibodies produced in response to infection with other dengue serotypes aid entrance of the virus into host macrophages. Although cases of DHF and DSS may result from this "immune enhancement,"[26] severe DHF and DSS also occur with other serotypes in the absence of previous infection with a heterologous dengue virus serotype.[13] Pathologic studies of DHF and DSS show hemorrhage, congestion, and perivascular edema of multiple organs. The liver may show areas of focal necrosis. As with yellow fever, the extent of pathologic findings does not correspond to severity of the clinical course.

Ecology and Epidemiology. *A. aegypti* is the principal vector for dengue viruses worldwide. In the Americas and Asia, viral transmission is maintained through a mosquito–human cycle without a major animal reservoir. Monkey carriers have been identified in Africa and Asia, but their importance in transmission is unclear. *A. albopictus,* an anthropophilic dengue vector

from Southeast Asia, has also been recognized recently in the Western Hemisphere. Both of these mosquitoes are capable of large-scale transmission to humans in endemic areas. Currently, dengue is endemic in tropical and subtropical Asia, Africa, South America, and the Caribbean basin. In the early 1920s, large epidemics occurred in Texas, where dengue infections were reported in 500,000 inhabitants. In the last 30 years, endemic transmission on the mainland United States has been documented only in Texas. Travelers to Southeast Asia have highest risk of dengue, especially when travel takes place during periods of high transmission and epidemic dengue.[84]

Clinical Presentation. Most dengue infections appear after an incubation period of 2 to 14 days, either as an undifferentiated viral syndrome with fever and mild respiratory or gastrointestinal symptoms or as dengue ("break-bone") fever with bone pain, generalized myalgia, severe headache, and retrorbital pain. Febrile illnesses that appear more than 2 weeks after putative exposure to dengue virus are unlikely to be due to this virus. After 1 to 3 days, a quiescent period may ensue. There may be a subsequent second episode of fever accompanied by a patchy maculopapular or morbilliform rash that spreads outward from the chest and that ultimately desquamates. Lymphadenopathy and leukopenia occur during this phase of the illness. The distinct severe forms of dengue disease referred to as either DHF or DSS may occur around the usual time of recovery. These are due to the development of capillary leak syndrome with associated hemorrhagic manifestations (Fig. 78-1), and the advanced forms have the unique feature that the platelet count decreases to less than 100,000 per mm^3 and hematocrit increases by more than 20%. The severity is classified as grade I to IV, according to World Health Organization guidelines. In cases of grade I DHF, the only hemorrhagic manifestation is a positive tourniquet test, in which inflation of a tourniquet to midway between systolic and diastolic blood pressure leads to development of 20 or more petechiae distal to the tourniquet. A complete blood cell count classically shows a decreased platelet and leukocyte count and an increase in hematocrit value. Grade II DHF is defined as the above with hemorrhage from any site. DSS grade III includes clammy skin, hypotension, or a narrow pulse pressure (<20 mm Hg) in a patient with DHF. An undetectable blood pressure defines grade IV DHF and DSS. Most studies have noted DHF and DSS primarily in infants and young children, usually with a history or serologic evidence of previous heterologous dengue infection.

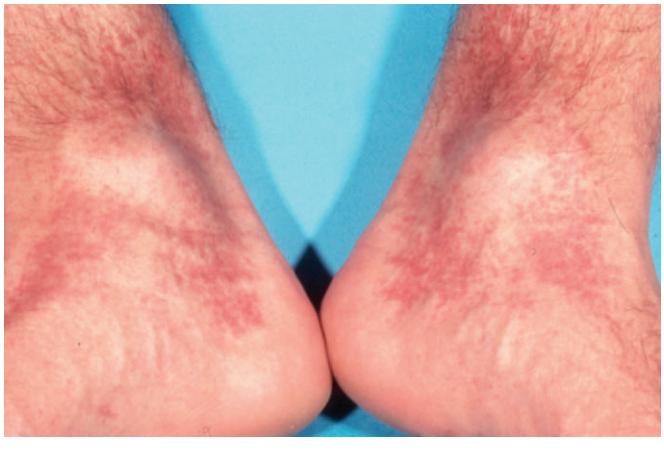

Figure 78-1. Hemorrhagic manifestations of dengue hemorrhagic fever or dengue shock syndrome. (From Ryan ET, Wilson ME, Kain KC: Illness after international travel. N Engl J Med 2002;347(7):505–516.)

Prevention and Management. There is currently no vaccine to protect against dengue infection or disease, though several experimental products are under development and progressing in clinical trials.[61,71]

Awareness of the local epidemiology of DHF and DSS, especially the occurrence of other cases, is important in establishing the diagnosis. The diagnosis may be confirmed by a fourfold change in antibody titer between acute and convalescent sera or by presence of anti-dengue IgM antibodies. In the United States, isolation of the virus from serum can be arranged through state health departments. Management is symptomatic and directed in severe cases to fluid management. In the DF syndrome, acetaminophen may be given for fever and myalgia. Salicylates should not be used. Hydration should be vigorously maintained.

Selected victims of grade I or II DHF may be managed as outpatients. However, outpatient care requires careful monitoring of hematocrit value, platelets, and electrolytes. If significant bleeding develops, hospitalization is appropriate for rapid and continuous assessment. Progression to DSS (grade III or IV) is a medical emergency and requires immediate hospitalization. There is no specific antiviral chemotherapy. Supportive measures described in the section on treatment of severe yellow fever are appropriate. A recent double-blind study of Vietnamese children with DSS has shown that Ringer's lactate is indicated for management of moderately severe illness.[85]

Lassa Fever

Four viral hemorrhagic fevers—Lassa, Marburg, Ebola, and Crimean-Congo—have been associated with outbreaks of fatal person-to-person spread. Although the overall number of clinical cases in travelers caused by these viruses is small, they represent potentially significant threats as emerging diseases. They have also achieved notoriety as a group as a result of media interest and their potential use as agents of bioterrorism. Lassa fever was first recognized in 1969, when several nurses caring for febrile patients at a mission hospital in Nigeria became ill. Since that time, seroepidemiologic studies have established a large area of endemicity and a broad spectrum of clinical manifestations of infection.

Epidemiology. The principal animal host for this virus is a rat, *Mastomys natalensis,* which prefers living in and around human dwellings. The rodents become chronically infected, secreting viral particles for long periods. Natural infection in humans occurs after rodent contamination of food and drink, inhalation of aerosolized rodent secretions, or contact with rodent material through skin abrasions. Lassa fever has been reported in several areas of sub-Saharan West Africa, and large outbreaks have been noted in Nigeria, Sierra Leone, Guinea, and Liberia.[11,46] Complete seroprevalence data are lacking, making definition of an endemic area impossible at this time. Secondary human infection has been reported and may occur after contact with infected secretions.

Virology and Pathophysiology. Lassa virus is a single-stranded RNA arenavirus. Proliferation and dissemination presumably occur after initial replication at the inoculation site. As with the flaviviral diseases, the extent of end organ involvement noted at autopsy does not account for the rapid death of infected patients. Recent work in an animal model provides evidence for platelet dysfunction and an endothelial cell defect in shock caused by Lassa fever virus. DIC, believed to be a major cause of bleeding and death in patients with other viral hemorrhagic fevers, appears to play a relatively minor role in arenavirus infections. The liver is most consistently the organ in which pathologic changes are observed at autopsy.

Clinical Presentation. Most seroconversions to Lassa virus are not accompanied by obvious symptoms.[47–49] Only 5% to 14% of seroconverters experienced a febrile illness, and the fatality-to-infection ratio was 1:100 to 2:100. Patients hospitalized with Lassa fever show a distinct clinical syndrome. Fever, malaise, and purulent pharyngitis often develop after the insidious onset of headache. Retrosternal chest pain, possibly a result of pharyngitis and esophagitis, suggests the diagnosis. The combined presence of retrosternal chest pain, fever, pharyngitis, and proteinuria is the best predictor of Lassa fever.[46] Hemorrhagic complications (hematemesis, vaginal bleeding, hematuria, lower gastrointestinal bleeding, and epistaxis) were seen in fewer than 25% of patients with Lassa fever. Nonfatal disease usually begins to resolve in 8 to 10 days. The combined presence of fever, sore throat, and vomiting was associated with a poor prognosis (relative risk of death = 5.5). Terminal stages of fatal disease were accompanied by hypotension, encephalopathy, and respiratory distress caused by stridor (presumably secondary to laryngeal edema). Laboratory indicators of a poor prognosis include an admission serum aspartate aminotransferase (AST) of greater than 150 IU/L (55% death rate) and viremia greater than 10^4 $TCID_{50}$ ($TCID_{50}$ is the titer at which sera containing the virus infects 50% of tissue culture test plates).

Diagnosis. Establishing an accurate diagnosis is extremely difficult during the early phase of the infection. As the classic clinical syndrome develops, differentiation from other viral hemorrhagic fevers depends on serologic confirmation. Serologic diagnosis is made by indirect fluorescent antibody analysis of acute and convalescent sera or detection of Lassa-specific IgM antibody. Clotted whole blood may be sent to the CDC for viral culture if handled appropriately. If the diagnosis is suspected, the CDC should be contacted immediately for assistance in diagnosis, isolation, and management.

Management. As mentioned above, Lassa fever has been associated with outbreaks of fatal person-to-person spread. Secondary infection occurs through direct contact with infected persons or their secretions. The role of aerosols in person-to-person spread is unclear. Blood and body fluids should be considered infectious. In light of the potentially fatal outcome of Lassa fever and the relative ease of transmission, the CDC has published specific recommendations for management of possible or confirmed cases. If a person has (1) a compatible clinical syndrome (especially pharyngitis, vomiting, conjunctivitis, diarrhea, and hemorrhage or shock); (2) a relevant travel history, including time spent in an endemic area; and (3) prior contact within 3 weeks of presentation with a person or animal from an endemic area suspected of having a viral hemorrhagic fever, he or she should be isolated and local, state, and federal health officials contacted. Ideally, an isolation unit with negative air pressure vented outside the hospital should be used. However, lack of a negative-pressure room alone is not a reason for transfer to another medical care facility.

The probability of transmission of Lassa fever virus to medical and nursing staff can be reduced by the use of routine blood and body fluid precautions as well as strict barrier nursing. Barrier nursing includes wearing gloves, gown, mask, shoe covers, and, if there is risk of splashing fluids, goggles whenever entering the patient's room. Decontamination of solid articles and rooms may be accomplished with 0.5% sodium hypochlorite solution. Recommendations for the management of patients with viral hemorrhagic fever have been published.[3,58]

Ebola and Marburg Viruses

Ebola and Marburg viruses are closely related large-RNA viruses known as filoviruses. They cause severe viral hemorrhagic fever syndromes with high mortality rates. Both are endemic in focal areas of central and southern Africa.[56] Ebola virus seropositivity has been noted in Sudan, Democratic Republic of the Congo, the Central African Republic, Côte d'Ivoire, and Kenya. A strain of Ebola known as Ebola Reston has been found in monkeys imported into the United States from the Philippines. More recently, there have been outbreaks with fatalities in Gabon, Democratic Republic of Congo, and Angola. Marburg disease is found in South Africa, Zimbabwe, and Kenya. In 2005, there was an outbreak that caused over 300 deaths in Angola.[53] Although there is not definitive evidence indicating the animal reservoir that maintains these filoviruses in nature, current evidence strongly suggests that bats are involved.[54,57] Person-to-person transmission has been well documented, primarily through contaminated needles and contact with the secretions of infected individuals.[54,57]

Pathophysiology and Clinical Presentation. Marburg and Ebola viruses are presumed to act through similar pathophysiologic mechanisms that involve activation of procoagulants through tissue factor and marked suppression of innate immunity and interferon-alpha production.[33,41,67]

Patients present after an incubation period of 4 to 10 days with fever, headache, and myalgias. Diarrhea and abdominal pain occur commonly. In many victims, rash, conjunctivitis, sore throat, and chest pain appear early in the disease. As in other hemorrhagic fevers, hemorrhage, hypotension, shock, and electrolyte abnormalities mark fatal courses. The high mortality reported in various outbreaks (25% for Marburg virus and 55% to 88% for Ebola virus) and transmission to health care workers taking care of patients emphasizes the importance of intensive supportive care and precautions that limit contact with body secretions of infected individuals.[54]

Diagnosis and Treatment. If these diseases are suspected, strict isolation procedures should be instituted and the local health authorities and the CDC notified immediately. Diagnosis may be made on a serologic basis or by polymerase chain reaction (PCR). There appears to be no serologic cross reactivity between the two viruses. Although anecdotal reports suggest the efficacy of immune sera in therapy, this has not been consistently observed in experimental studies. There are currently no specific antiviral therapies for Marburg or Ebola virus infection.

Care is supportive, and the therapeutic considerations given for treatment of severe yellow fever should be followed. There is promise of a vaccine based on observations that primates immunized with this product are protected against lethal challenge with Ebola virus.[27,73,81]

Crimean-Congo Hemorrhagic Fever

Virology and Epidemiology. The etiologic agent of Crimean-Congo hemorrhagic fever (CCHF) is a bunyavirus. Ixodid ticks serve as both reservoirs and vectors of the virus. Infection in humans results from tick bites or direct contact with infected secretions from crushed ticks, animals, or humans. Most cases occur in individuals with occupations or living conditions that bring them in contact with domestic goats, sheep, or cattle on which ticks feed. The disease has been observed in southeastern Europe, south central Asia, the Middle East, and much of Africa.[83] Nosocomial transmission through contact with infected body fluids has been well documented.[78–80]

Pathophysiology and Clinical Presentation. Pathophysiologic mechanisms are presumably similar to those of other hemorrhagic fevers.[17] One in five infections results in clinical disease. Among those clinically ill, mortality ranges from 15% in sporadic cases to 70% in rare hospital outbreaks.[21,75]

Diagnosis. The diagnosis can be confirmed with acute and convalescent serologic evaluation for a four-fold rise in IgG antibody titers. The virus can be cultured from whole blood if it is drawn during the first week of symptoms and kept on dry ice (or at –70° C [–57° F]) during shipment to the CDC.

Management. Initial management is similar to that for Lassa, Marburg, and Ebola virus infections, with strict patient isolation and notification of health authorities. Supportive therapy should be instituted as discussed for yellow fever. Although not confirmed in clinical trials, ribavirin has good activity in vitro against CCHF virus. The CDC recommends that patients believed to have CCHF receive intravenous (IV) ribavirin in the doses suggested for treatment of Lassa fever.[17] Persons in contact with CCHF patients should receive prophylactic ribavirin as suggested for Lassa fever contacts. Treatment with oral ribavirin has been successfully used in a nosocomial setting when IV therapy was not available.[18]

Hemorrhagic Fever with Renal Syndrome and Hantavirus Pulmonary Syndrome

Hemorrhagic fever with renal syndrome (HFRS) first came to the attention of Western medical science during the Korean Conflict, when febrile illness accompanied by bleeding and renal failure developed in 3000 United Nations troops.[24] Mortality ranged from 5% to 10%. A similar, less severe syndrome *(nephropathia epidemica)* had been recognized in Scandinavia since the 1930s. More recent investigations have resulted in the identification of a group of RNA viruses, known as hantaviruses, as a cause of this syndrome. A novel infection associated with a new hantavirus, the Muerto Canyon or Sin Nombre virus, was described after a cluster of deaths in the Southwestern United States in 1993. This is hantavirus pulmonary syndrome (HPS), in which a nonspecific febrile illness is followed by shock and alveolar pulmonary edema.[14]

Epidemiology. The agent of HFRS causes chronic, nondebilitating infections of rodents. Human infection is initiated by contact with rodent secretions or inhalation of aerosolized rodent material. The disease occurs most commonly in rural areas, although occasional urban outbreaks, presumably with the common house rat as vector, have been described. Cases have been described most often from the Far East, including China, Korea, Japan, and the Soviet Union, but the disease also occurs in Eastern Europe. A recent epidemiologic study from China found the highest rates of infection in men who engaged in heavy farm work and slept on the ground (rather than on raised wooden beds).[86] The Sin Nombre agent appears to cause a chronic infection of the deer mouse, *Peromyscus maniculatus*, which is the main reservoir of the virus in the United States. Since the initial outbreak, additional cases have been described across the United States and South America. The risk of infection is likely to be related to rodent exposure, but transmission is infrequent.

Virology and Pathophysiology. The etiologic agent of HFRS is the prototype hantavirus, Hantaan virus, a member of the bunyavirus family. A related hantavirus, the Puumala agent, is the apparent cause of the more benign nephropathia epidemica. Other closely related viruses can cause similar, although less frequently recognized, syndromes. Seoul virus causes a milder form of HFRS in the Far East, and a severe HFRS-like syndrome in Eastern Europe has been attributed to a related virus. As described above, Sin Nombre virus is the usual cause of hantavirus pulmonary syndrome (HPS) in the United States, but other hantaviruses probably cause the same syndrome.

Clinical Presentation. As with most viral hemorrhagic fevers, infection may be asymptomatic or accompanied by a mild nonspecific illness. In the classic severe form, an initial febrile phase is associated with petechiae, proteinuria, and abdominal pain. After 3 to 5 days, a hypotensive phase occurs, with decreased platelet count and more severe hemorrhagic phenomena. An oliguric phase follows with concomitant electrolyte abnormalities. Subsequently, a diuretic phase usually commences 10 days after the onset of illness. Death occurs from hemorrhage, hypotension, and pulmonary edema, presumably secondary to fluid overload and renal failure. With modern management, the case-fatality rate of classic HFRS is about 5%. The more benign nephropathia epidemica syndrome has a case-fatality rate of less than 1%. In this disease, hypotension, shock, and hemorrhagic manifestations are rare. With HPS, there is usually a prodromal illness with fever and mild respiratory or gastrointestinal symptoms, followed by shock and pulmonary edema. The tempo of the disease at this stage may be rapid and require respiratory and circulatory support in an intensive care unit.

Diagnosis. The diagnosis of HFRS, nephropathia epidemica, and HPS is confirmed by indirect fluorescent or enzyme-linked immunosorbent assay (ELISA) for antibodies in acute and convalescent sera. IgM antibody determination may also be helpful. Virus isolation is difficult, but PCR and immunohistochemical staining may be useful in affected tissues.

Management. Care of patients with HFRS is supportive and should follow the guidelines given for yellow fever. With HFRS, renal dysfunction occurs early and may require institution of

dialysis soon after diagnosis to prevent fluid overload and to correct electrolyte disturbances. Patients' secretions should be handled with care, and enteric precautions (but not strict isolation) are prudent. It is not clear whether person-to-person transmission of the virus through direct inoculation occurs. For the hantaviruses, viremia recedes and antibody levels rise as the clinical phase appears. Accordingly, nosocomial transmission or hematogenous transmission with hantavirus infections has not been frequently documented, although presumed nosocomial transmission has been reported.[86]

Japanese B Encephalitis

Japanese B encephalitis has been recognized in Japan since the 19th century. It is the only arboviral encephalitis for which an effective inactivated vaccine has been developed. Vaccine use in Japan and elsewhere since the 1960s has resulted in a significant decrease in the disease rate. However, there is concern regarding the routine use of the vaccine for travelers because of the description of several adverse reactions described in Western countries. These tend to occur on the day after vaccination.[32] Therefore, use of the vaccine depends on a risk-benefit analysis. Relevant considerations include the duration of travel (minimum of at least 30 days), travel in an endemic area during the transmission season, and the likelihood of exposure to mosquitoes. A history of adverse reactions to the vaccine is a contraindication to its use.

Epidemiology. Rice field-breeding and other culicine mosquitoes serve as the vectors for Japanese B encephalitis. In addition to humans, birds and pigs can be infected. Pigs play an important role as amplifying hosts because they develop high-grade viremia from which large numbers of mosquitoes may be infected. Most infections in endemic areas occur in children, whereas all age groups of previously unexposed populations are at risk. Transmission of Japanese B encephalitis currently occurs in India, Southeast Asia, China, Korea, Indonesia, the Western Pacific region, and eastern Russia.[77] Routine use of Japanese B encephalitis vaccine in Japan is being eliminated because of low risk in this country. Recent outbreaks and case reports of Japanese B encephalitis in islands of the Torres Strait that runs between Northern Australia and Papua New Guinea indicate that the virus has spread southward from Asia, presumably by migratory ardeid birds.[28]

Virology and Pathophysiology. Japanese B encephalitis is caused by a neurotropic flavivirus that is phylogenetically related to West Nile and dengue viruses. After initial replication near the mosquito bite, viremia occurs, which if prolonged may seed infection to the brain. The cytopathologic effect of the flavivirus is believed to cause nerve cell destruction and necrosis.

Clinical Presentation. Most infections do not cause clinical illness. Many patients recall a mild undifferentiated febrile illness, which probably coincides with the viremic phase of infection. Patients with encephalitis often report a similar prodrome. The encephalitis syndrome is not easily distinguishable from other arboviral encephalitides. The patient usually complains of headache, lethargy, fever, and confusion and may display tremors or seizures. Reported mortality of persons with clinical encephalitis ranges from 10% to 50%.[77] One clinical series suggested that the presence on admission of (1) unresponsiveness to pain, (2) low levels of anti-Japanese B encephalitis virus IgG or IgM antibodies (in serum or cerebrospinal fluid [CSF]), or (3) virus in CSF culture was associated with death.[5] Of the 16 patients with fatal disease, all died within 7 days of hospitalization.

Diagnosis. Acute and convalescent sera for antibody determination (virus neutralization or hemagglutination inhibition assays) provide the only reliable method of diagnosis. Paired sera should be sent for these assays through state health departments. Sensitive assays for determinations of IgG and IgM antibodies in serum and CSF have been developed but are not yet widely available. Because most patients seek treatment long after the viremic phase, blood cultures are rarely positive for the virus and CSF cultures are often positive only in patients with a poor prognosis.

Management. There is no specific therapy. The main interventions are prophylactic: vaccination and reduced arthropod exposure. Supportive care may require an intensive care unit. Because the virus is present in body fluids, especially CSF, blood and body fluid precautions should be considered. Japanese B encephalitis is only one of several of arthropod-borne viruses that may cause encephalitis in different areas of the world; others include Murray Valley encephalitis in Australia, tick-borne encephalitis in Europe (for which a vaccine exists), and Lacrosse, West Nile, and St. Louis encephalitis in the United States.

Named Hepatitis Viruses

Although infectious hepatitis has been a well-known clinical entity for hundreds of years, it is only in the last few decades that identification of specific viral pathogens has been possible. The causes of hepatitis may be divided into two groups. First, the so-called named, or more accurately, lettered viruses, now include hepatitis A to G. These are associated with defined clinical syndromes and elevated liver function tests. Second, other organisms that cause hepatitis as part of a more systemic infection include Epstein-Barr virus, cytomegalovirus, toxoplasmosis, and leptospirosis. Only selected examples in the former group are discussed here.

Hepatitis A

Epidemiology. Hepatitis A virus is transmitted primarily by the fecal-oral route by either person-to-person contact or ingestion of contaminated food or water. Food items commonly associated with outbreaks are raw or undercooked clams and shellfish. Homosexual men have higher rates of seropositivity than the population at large. Occasional cases are associated with exposure to nonhuman primates. Transmission by blood transfusion has been reported, but this is an uncommon source of infection. Hepatitis A is endemic world-wide, but underdeveloped nations have a higher prevalence than those in North America. Most persons in these areas show serologic evidence of past infection with hepatitis A virus. Several recent studies suggest that hepatitis is the most common serious viral infection occurring in travelers and that hepatitis A virus is the most common identifiable cause.[20,25]

Virology and Pathophysiology. Hepatitis A virus is a picornavirus with a single-stranded RNA genome. Although the

pathophysiologic mechanism has not been delineated, most infections begin with introduction of viral particles into the proximal gastrointestinal tract. Brief viremia precedes seeding of hepatocytes, where viral replication has been documented. With replication, hepatocellular necrosis is accompanied by lymphocytic infiltration. In the vast majority of cases, hepatic regeneration occurs after acute disease and no significant sequelae are observed.

Clinical Manifestations. The incubation period ranges from 2 to 7 weeks. The infection may be asymptomatic or mild, especially in children, but also in a minority of adults. The classic syndrome includes initially anorexia, followed by nausea, vomiting, fever, and abdominal pain. These symptoms may be accompanied by hepatosplenomegaly. AST and alanine aminotransferase (ALT) levels rise within a few days of the onset of symptoms. In children, AST and ALT return to normal levels in 2 to 3 weeks, whereas in adults resolution of elevated serum aminotransferase levels may take several months. The bilirubin level rises shortly after AST and ALT elevations. Jaundice usually follows gastrointestinal symptoms by several days to a few weeks. Resolution of jaundice may take another 3 to 4 weeks. The syndrome is occasionally preceded by arthralgias and rash, but these prodromal symptoms are uncommon. Resolution of acute disease is permanent in most instances, but rare cases of relapse have been noted. It has become clear in recent years that severe illness and death after acute hepatitis A are not as rare as initially believed.[20] The fatality rate in cases reported to the CDC is 0.6%.[60] Pregnant women have a higher risk of severe illness than does the general adult population. Anti–hepatitis A antibody (primarily IgG) is detectable in the blood for many years after infection. The presence of the antibody confers immunity. Accordingly, reinfection with hepatitis A virus is not believed to occur.

Diagnosis. The clinical presentation of hepatitis A is usually milder than other types of viral hepatitis. Consequently, the symptoms are not distinctive enough to allow a firm diagnosis, which requires detection of hepatitis A antigen in the stool or serologic evaluation for hepatitis A–specific IgM or total anti–hepatitis A virus antibody. Stool hepatitis A antigen is maximal before the onset of symptoms but may be detected as long as 2 weeks after the onset of disease. A more practical test is measurement of hepatitis A–specific IgM antibody, which is usually present by the time symptoms are recognized and generally absent 6 months later. Measurement of anti–hepatitis A antibodies may be helpful in evaluating possible causes of past icteric episodes or for seroepidemiologic studies, but their presence does not differentiate recent from past infection.

Management. No specific therapy exists for hepatitis A. Affected persons are usually managed as outpatients and should be instructed on enteric precautions to avoid transmission to others. Although infectivity drops sharply soon after the onset of jaundice, it is prudent to maintain enteric and blood-drawing precautions for about 2 weeks after jaundice appears. Nosocomial transmission has also been documented, but most spread probably occurs before jaundice and diagnosis.

Prevention. Active immunization with hepatitis A vaccine is recommended for most travelers to at-risk areas.[59] This is discussed in Chapter 77.

Hepatitis B

The spread of hepatitis by parenteral means was noted in 1885. Recognition in the 1960s of specific viral particles (the Australia antigen) in the serum of hepatitis patients led to identification of the responsible agent.

Epidemiology. With the widespread use of serologic markers for hepatitis B disease, it became apparent that spread occurs through exchange of blood, semen, or, rarely, saliva of infected people. Although spread is possible from persons with acute disease, the primary sources of viral particles are chronic carriers. Persons are defined as carriers if blood samples obtained 6 months apart contain hepatitis B surface antigen particles (HBsAg). The carrier state follows acute infection in up to 90% of infected infants and 10% of adults. Risk factors for acquisition of hepatitis B infection in the United States include IV drug use, homosexual activity, and working in health care. In the United States, most victims are adults, and the carrier rate in the general population is less than 0.5%. In many areas of the developing world, most infections occur in infancy or childhood, and chronic carriers may comprise as much as 10% to 20% of the total population. Thus, travelers are more likely to be exposed to carriers than is the nontraveling population. The risk is higher in persons regularly exposed to body fluids, including medical personnel and persons with many sexual partners.

Virology and Pathophysiology. Hepatitis B virus is a deoxyribonucleic acid (DNA) virus unrelated to the agent responsible for hepatitis A. Infection occurs naturally in humans and can be induced easily in some nonhuman primates. Most hepatitis B infections are subclinical. In those resulting in clinical disease, entry of the virus into the liver is followed by viral replication and hepatocyte necrosis. HBsAg, a viral particle, appears in the bloodstream within 3 months of infection. In most cases, IgM antibody to the hepatitis B core antigen (HBcAg) appears first, followed by anti-HBsAg (surface) antibody. Antibody to a third hepatitis antigen, the e antigen (HBeAg), is present for variable periods. The course of the disease varies widely, depending on a number of factors that are not well defined. In brief, most cases are self-limited and resolve in 4 to 6 months. In these patients, anti-HBsAg or anti-HBcAg IgG antibodies can be detected for years after the episode of hepatitis. Chronic carriers do not develop anti-HBsAg antibody, but rather maintain measurable levels of HBsAg. Similarly, carriers with persistent HBeAg detectable in blood samples appear to be more infectious than carriers without circulating HBeAg. The intricate network of antibody-antigen relationships in hepatitis B is believed to play a role not only in development of acute and chronic hepatitis but also in the many extrahepatic syndromes associated with hepatitis B. Immune complex formation has been suggested as etiologic in hepatitis B–associated arthritis, rash, arteritis, and renal disease.[55]

Clinical Presentation. The incubation period for hepatitis B ranges from 7 to 22 weeks; however, the patient may be antigenemic for a large portion of that time. The manifestations of hepatitis B infection are similar to hepatitis A, including fever, anorexia, nausea, vomiting, and abdominal pain. In addition, a prodrome of rash, arthralgia or arthritis, and fever is seen in up to 20% of hepatitis B patients compared with their rarity in hepatitis A. Glomerulonephritis is occasionally seen. Jaundice

usually appears a short time after the onset of gastrointestinal symptoms. In the self-limited disease, recovery is complete by 6 months. Some infections follow a fulminant course; case-fatality rates are 2% or less in most series. In addition to complete resolution or death, three other sequelae are possible with acute hepatitis B. A person may become an asymptomatic chronic carrier and remain HBsAg positive but have no detectable active hepatitis. Another sequela is *chronic persistent hepatitis*, a term used to describe persistent but not progressive, hepatic inflammation (usually monitored by serum transaminase levels), often with HBsAg in the serum. Persons with chronic active hepatitis may be HBsAg positive and have progressive hepatitis, which may result in cirrhosis and death directly related to liver disease. Any of these three conditions results in the presence of hepatitis B viral particles in the blood.

Diagnosis. A variety of antigen and antibody tests have been developed for the diagnosis and monitoring of hepatitis B disease. The most practical and widely available test for diagnosis of acute disease is the assay for HBsAg. Antigen is usually present before the onset of symptoms and persists during symptomatic disease. Occasionally, HBsAg may be undetectable in patients with clinical disease caused by hepatitis B. In these cases, antibody to HBcAg is often present. Later, antibody to HBsAg will appear, but this is often long after the episode of clinical hepatitis.

Management. Management is similar to that of hepatitis A. Prolonged viremia makes blood and body fluid precautions necessary until the absence of HBsAg antigen and the presence of antibody to HBsAg are established. For patients with chronic infection, therapy with interferon-alfa is recommended.[42] Even in individuals with good prognostic indicators, the response rate in terms of long-term clearance of virus and seroconversion approaches only 30%.

Prevention. Travelers to highly endemic areas who stay for 6 or more months or have close contact with inhabitants should be vaccinated. The various available vaccines are discussed in Chapter 77.

Delta Hepatitis (Hepatitis D)

Hepatitis with the delta agent was first suspected in 1977, when cases of severe hepatitis B disease and exacerbations of hepatitis were being evaluated.

Epidemiology. Delta virus infection is found only in patients concomitantly or previously infected with hepatitis B.[64] Transmission follows a pattern similar to hepatitis B. In the United States, affected populations are IV drug abusers and multiply transfused hemophiliacs. Serologic evidence of delta virus disease has been documented in the Mediterranean basin, West Africa, and parts of South America.[63]

Virology and Pathophysiology. The delta agent has been termed a *defective virus* because it requires hepatitis B virus activity for its own replication.[65] The agent is a single strand of RNA enclosed in a protein coat of HBsAg. The delta agent infects cells at approximately the same time as does hepatitis B virus (coinfection), or it may be introduced later in the course of persistent hepatitis B infection (superinfection). In coinfected patients, the clinical picture may not differ from hepatitis B, but a higher percentage of such patients develop severe disease than do those with hepatitis B alone. Patients superinfected with the delta agent develop flare-ups of hepatitis, which may become fulminant. After the acute infection, the delta agent can cause progressive disease in previously stable hepatitis B patients. In general, infection with the delta agent worsens the prognosis of hepatitis B disease. The diagnosis can be made by detection of antibody to the delta antigen in the serum[63] or, less commonly, by detection of free delta antigen in serum.[62]

Management and Prevention. Management of acute hepatitis consists of supportive care. Precautions against transmission are the same as for hepatitis B. There is no specific vaccine or IG for the delta agent. The best preventive measure is to be vaccinated for hepatitis B because delta agent infection cannot occur in the absence of the former virus.

Hepatitis C

As serologic methods for the diagnosis of hepatitis A, hepatitis B, and delta agent were developed, it became apparent that there was a group of persons with hepatitis for which no etiologic agent had been identified. This syndrome was previously termed non-A, non-B (NANB) hepatitis and thought to be caused by a heterogeneous group of etiologies. It is now clear that a majority of such cases were due to hepatitis C.[37]

Epidemiology. Risk factors for hepatitis C include IV drug use and, before routine testing, transfusion of blood products. Nonparenteral routes of infection are less important than for hepatitis B. Hepatitis C is a global problem. Approximately 80% of exposed individuals develop chronic infection, which may lead to cirrhosis in 20% of subjects and hepatocellular carcinoma in up to 5% of this subset of infected persons. Rates of infection vary from 1% to 5% in most Western countries to 20% in parts of the Middle East, such as Egypt.

Virology and Clinical Manifestations. Hepatitis C is a single-stranded RNA virus of the Flaviviridae family. Transition to chronic hepatitis after an insidious asymptomatic infection is the usual pattern. Chronic hepatitis may be asymptomatic or associated with nonspecific symptoms, such as lethargy, nausea, and abdominal discomfort. The patterns of cirrhosis and hepatocellular carcinoma, when they occur, do not differ significantly from those of other conditions. Extrahepatic syndromes associated with hepatitis C infection include porphyria cutanea tarda, membranous glomerulonephritis, and mixed cryoglobulinemia.

Diagnosis. Diagnosis of hepatitis C has been a vexing problem. First-generation ELISA tests available soon after the discovery of the virus led to a significant number of false positive results. These tests have subsequently become more sensitive and specific, and radioimmunoassays (RIA or RIBA) have become available and are useful in confirmatory testing. To clarify the significance of a positive screening test, it should be repeated, and if possible, a second confirmatory test should be done (usually a RIBA). Interpretation of the significance of such tests should be tempered by the presence or absence of risk factors for infection. PCR tests for hepatitis C may also provide further evidence of infection and a direct measure of viral replication.

If the PCR results are positive, it should be interpreted with two caveats. First, a minority of infected individuals clear the virus, so a negative test result does not exclude past infection. Second, the serum PCR may be negative in the face of viral replication in the liver.

Management and Prevention. Prevention of hepatitis C is largely dependent on risk reduction, especially with respect to drug use. Pooled immunoglobulin has been used after exposure, but this should be procured from donors screened for hepatitis C. It is, however, not generally recommended. Unlike hepatitis B, protective antibody responses have not been demonstrated. Treatment of acute hepatitis C is supportive. Treatment of chronic infection is with interferon-alfa, though the overall results are disappointing and side effects significant. Response rates increase to approximately 40%, however, when interferon-alfa is used in combination with ribavirin.[10]

Hepatitides E, F, and G

Hepatitis E is an RNA virus provisionally placed in the Caliciviridae family. It is the second most common cause of viral hepatitis transmitted via the enteric route. The epidemiologic characteristics are similar to hepatitis A. This group of infections is especially important in the Indian subcontinent, the Middle East, and Africa. The incubation period is 2 to 6 weeks. The disease is usually self-limited but may be associated with severe illness in pregnant women. Diagnosis in travelers from endemic areas can be made on the basis of IgM antibody to hepatitis E in serum or testing of stool for viral antigen. PCR for hepatitis E may be available in some centers. In the United States, testing for hepatitis E is best undertaken in returned travelers with clinical hepatitis, although a more severe illness may occur in persons with underlying liver disease. Vaccines are not available. Prophylaxis is appropriate advice for travelers and involves counseling with respect to precautions regarding ingestion of food and water in endemic areas.

Hepatitis F is a putative hepatitis virus of uncertain significance, first described in France. Hepatitis G is a member of the flavivirus family with limited homology to hepatitis C. Its significance as a cause of hepatitis is also unclear.

MAJOR BACTERIAL INFECTIONS

This section reviews several bacterial diseases of relevance to the overseas traveler, including typhoid fever, meningococcal disease, pertussis, diphtheria, and tetanus. Other chapters deal with bacterial causes of gastroenteritis and diarrhea, tick-borne diseases, and zoonoses.

Typhoid Fever

Typhoid fever was recognized as a clinical entity in the 1800s and was first associated with transmission by the fecal-oral route in the 1870s. Although effective treatment with chloramphenicol became possible in 1948, the disease continues to be a major cause of morbidity and mortality in the developing world.[15]

Epidemiology. Typhoid fever occurs worldwide, but its prevalence and attack rates are much higher in under-developed countries.[16] Humans are the only host for *Salmonella typhi,* the most common cause of the typhoid fever syndrome. Nearly all cases are contracted through ingestion of contaminated food or water. Transmission occurs through a variety of mechanisms, the most common of which are contact with a chronic carrier of the organism, especially food handlers, and ingestion of untreated waste material or sewage. Improved sewage and tracking of chronic carriers have markedly reduced the incidence in developed countries, although several hundred cases a year are reported to the CDC. Evaluation of cases reported between 1975 and 1984 revealed that 62% of typhoid fever cases in the United States were acquired during foreign travel. The highest attack rates occurred in persons traveling to India, Pakistan, Peru, and Chile (58 to 174 cases per 1 million travelers).[68]

Bacteriology and Pathophysiology. *Salmonella* species are gram-negative enteric bacilli. *S. typhi* is the prime cause of typhoid fever, but other species, including many of the *S. enteritidis* serotypes and some non-*Salmonella* enteric organisms such as *Yersinia* or *Campylobacter,* may cause a typhoid fever–like syndrome. *Salmonella* species are easily grown on routine bacterial culture plates, but if multiple organisms are present, media with selective growth inhibitors may be needed for optimal sensitivity. After ingestion of food or water containing the pathogen, organisms are subjected to the acid stomach environment, which results in significant bacterial killing. If the organisms pass through the small intestine, several processes may occur. The bacteria may simply pass through, causing few clinical symptoms. If the bacteria multiply and invade the mucosa, a gastroenteritis-like syndrome will result. Typhoid fever requires penetration of the intestinal mucosa and intestinal lymphatics, where intracellular replication of *S. typhi* occurs. Soon thereafter, bacteria seed the bloodstream and are transported to reticuloendothelial cells throughout the body, where further intracellular replication can take place. After the acute episode of infection is over, *Salmonella* species may remain and asymptomatically reproduce in scarred or chronically inflamed tissues. Persons may shed organisms from such foci for years and serve as a source of outbreaks while they themselves are asymptomatic. The most common site for such colonization is the chronically diseased gallbladder.

Clinical Presentation. After exposure to the pathogen, 10 to 14 days usually passes before the onset of clinical illness. Some patients may experience gastroenteritis early in the course of disease, and abdominal pain or diarrhea may be present at the time the classic typhoid fever picture develops. Fever is usually the first sign of disease. Fever increases slowly over several days and may remain constant for 2 to 3 weeks, after which time defervescence begins.[30] With antibiotic therapy, fever resolves more rapidly, often within 3 to 4 days.[68] Relative bradycardia may accompany fever. Most victims also report headache, malaise, and anorexia. Rose spots (2- to 4-mm maculopapular blanching lesions) are classically described on the trunk, although they are not seen in the majority of patients. Hepatomegaly and splenomegaly have been reported in a large number of patients. Laboratory investigations early in the course may show a high white blood cell (WBC) count, anemia, and mild elevations of serum hepatic enzyme levels, including AST, lactate dehydrogenase, and alkaline phosphatase. Later in the course of the disease, leukopenia ($WBC < 3500/mm^3$) develops. Uncomplicated and untreated typhoid fever resolves in 3

to 4 weeks. Several complications may herald or contribute to death. Intestinal perforation, presumably secondary to necrosis of lymphoid areas of the bowel wall, may lead to peritonitis and death. Significant gastrointestinal hemorrhage may occur but rarely is fatal. Secondary pneumonia is common. A subgroup of patients has more severe disease, which may include myocardial involvement, mental status changes, hyperpyrexia, and multisystem failure. The overall case fatality rate has ranged from 12% to 32% in the developing world but is less than 2% in industrialized nations.[15]

Diagnosis. Culture of a bacterial species associated with the syndrome (most likely *S. typhi*) from a normally sterile fluid makes the diagnosis. Multiple studies have evaluated the usefulness of various diagnostic tests. In general, bone marrow culture is the most sensitive method, detecting up to 90% of cases, whereas blood cultures are less sensitive. Both methods are most useful in the first week of disease. Stool cultures (and string test cultures) may be positive later in the course of disease but provide only circumstantial evidence of the causative agent. Work is currently being done on serodiagnostic methods, but no clear consensus has been reached on the relative usefulness of any test.[7]

Management. Chloramphenicol had been the mainstay of treatment for typhoid fever since the late 1940s. Ampicillin and trimethoprim-sulfamethoxazole were the traditional alternatives. In the last decade, multiple drug resistance has increased so ciprofloxacin is now the first-line antibiotic. Unfortunately, increasing resistance to quinolones has been observed, especially from the Indian subcontinent. In cases of quinolone resistance, either laboratory or clinical, ceftriaxone or other third-generation cephalosporins are indicated.[66] Other treatment modalities include fluid support and adequate nutrition. Corticosteroids have been used empirically for many years. A single randomized double-blind study showed that administration of high-dose dexamethasone (3 mg/kg for the first dose, followed by 1 mg/kg every 6 hours for eight more doses) with chloramphenicol resulted in significantly lower mortality in patients with severe typhoid fever than in those treated with chloramphenicol alone.[29] Severe typhoid fever was defined in this study by the presence of obtundation, delirium, stupor, coma, or shock (systolic blood pressure <90 mm Hg for those 12 years or older and <80 mm Hg in younger children). High-dose steroids were not recommended for those with less severe disease and should be used cautiously in those with severe disease.[9]

Prevention. Three vaccines exist for prophylactic use. The oldest in use is a heat-killed, acetone-precipitated vaccine. Efficacy in Americans, however, is only about 50%, and significant incidences of local inflammatory reactions and febrile responses occur in persons receiving the vaccine. Two doses of 0.5 mL are given subcutaneously separated by a 4-week interval. An oral vaccine was licensed for use in the United States in 1989. The vaccine contains the Ty21a strain of *S. typhi* and is associated with minimal side effects. The Ty21a vaccine is given as a four-dose series, with 2 days between each dose. Immunosuppression, antibiotic use, and gastroenteritis are contraindications to the use of this vaccine. A newer parenteral vaccine contains the Vi (virulence) polysaccharide of *S. typhi* purified from formalin-killed bacteria. It is administered as a single intramuscular injection and is therefore most useful when vaccination is required at short notice. A booster is required every 2 years.[8,38] Even when the vaccine is given before travel, it is important to observe routine precautions for ingestion of food and water to prevent typhoid fever and acquisition of other pathogens by the fecal-oral route.

Meningococcal Disease

Classically, meningococcal meningitis attacks children and young adults and is often seen in epidemic form. Although the advent of effective antibiotic therapy and a useful vaccine have greatly improved the ability to deal with this disease, it remains a major problem in many parts of the world.

Epidemiology. Cases of meningococcal disease occur sporadically worldwide, with epidemic disease generally limited to developing nations. Epidemic situations clearly pose the greater health problem to both travelers and resident populations. Since 1970, large outbreaks have occurred in Brazil; China; the Sahel region of sub-Saharan Africa (from Mali and Burkina Faso to Ethiopia and northwestern Somalia); New Delhi, India; and Nepal. Particularly in sub-Saharan Africa and China, the disease demonstrates yearly incidence peaks and periodic massive outbreaks, the exact determinants of which are unknown. Transmission of the organism occurs by exchange of respiratory secretions; contact is believed to be important in the spread of the disease. Asymptomatic transient nasopharyngeal carriage of the meningococcus, occurring with a baseline prevalence of 5% to 10%, may increase during epidemic periods and in close contacts of cases. The secondary attack rate among household contacts of patients with sporadic disease is 2:1000 to 4:1000, whereas that in epidemics ranges from 11:1000 to 45:1000 household contacts.

Bacteriology and Pathogenesis. *Neisseria meningitidis* is a gram-negative diplococcus that grows easily on several common media, including chocolate and blood agar. The organism is characterized further on the basis of serologic analysis of capsular antigens. The most common serogroups are A, B, C, Y, and W135. Serogroups A and C are often associated with epidemic disease, whereas serogroup B is the major cause of sporadic disease in the United States. Asymptomatic persons may carry various serotypes of *N. meningitidis* in the nasopharynx for short periods of time. An antibody response is often generated to these strains during asymptomatic carriage. The conditions that cause one person to become clinically ill with invasive disease while another carrier remains healthy are not well understood. The route of entrance of the organism to the bloodstream and central nervous system (CNS) is presumably through the nasopharynx or respiratory tract.

Clinical Presentation. Meningococcal disease may appear in a variety of forms, including, but not limited to, bacteremia with septic shock; meningitis, often accompanied by bacteremia; and pneumonia. Sustained meningococcemia may lead to severe toxemia with hypotension, fever, and DIC. In the fulminant presentation, adrenal hemorrhage may lead to Waterhouse-Friderichsen syndrome, and death may follow intractable shock. In the United States, the case-fatality rate for sustained meningococcemia is generally higher than for meningococcal meningitis. There is also a clinical syndrome of chronic meningococcemia with a much more insidious onset.

Meningitis caused by *N. meningitidis* classically begins with fever, headache, and a stiff neck. It may also be accompanied by bacteremia and any of several skin manifestations, including petechiae, pustules, or maculopapular rash. In either meningitis or bacteremia, progression of petechiae to broad ecchymoses is a poor prognostic sign. As with septic meningococcemia, severe meningitis may progress with mental status deterioration, hypotension, congestive heart failure, DIC, and death. The case-fatality rate of meningococcal meningitis with or without bacteremia is now estimated to be about 10%.[82] In classic cases of meningitis or bacteremia with sepsis, the peripheral WBC count is elevated, with polymorphonuclear cell predominance. CSF typically is purulent, usually with more than 500 polymorphonuclear cells/mm^3. There may be a more heterogeneous cell population and fewer cells if CSF is obtained early in the course or if the patient has been treated with antibiotics. The CSF glucose level is usually low and protein high, as in other bacterial meningitides. Gram stain of CSF may show the gram-negative diplococci. Meningococcal pneumonia is a well-known but less common clinical entity described in military recruit populations involving serogroup Y organisms.

Diagnosis. The presumptive diagnosis in an epidemic can be made on the basis of clinical presentation and purulent spinal fluid. The presence of characteristic bacterial forms on Gram stain is also suggestive. A definitive diagnosis requires culture of the organism from CSF or a normally sterile fluid (usually peripheral blood). This may be impossible in the case of a patient who was previously treated. Several commercial kits for measuring meningococcal antigen are now available for use on CSF or blood samples.

Management. Treatment of meningococcal meningitis or sepsis is a medical emergency. Fortunately, the organism remains sensitive to a large number of antibiotics. The treatment of choice is penicillin G, 300,000 units/kg/day (up to 24 million units a day), given intravenously in divided doses every 2 hours. Between 7 and 10 days of therapy for serious disease is appropriate. Ceftriaxone is an alternative antimicrobial agent. If the patient is allergic to penicillin, chloramphenicol (100 mg/kg/day) may be given, although the emergence of chloramphenicol-resistant strains of *N. meningitidis* is of great concern.[19] Notably, antibiotic treatment before culture or hospital referral is recommended in clinically suspected cases. Supportive care should include close monitoring for hypotension and cardiac failure. Fluids and vasoactive and cardioselective agents may be important. This type of support necessitates invasive monitors and intensive care unit technology. Development of DIC is an ominous sign. The role of heparin in this disease is still debated. Although focal bleeding and adrenal necrosis may lead to acute adrenal insufficiency, the role of replacement steroids in the treatment of Waterhouse-Friderichsen syndrome is unclear. Since the infectious agent has been found in household contacts and in those with exposure to oral secretions, contacts should receive prophylaxis to eradicate the organism. Rifampin, 600 mg by mouth every 12 hours for four doses, is standard adult prophylaxis. Children should receive 10 mg/kg of rifampin every 12 hours for four doses if they are older than 1 month and 5 mg/kg every 12 hours for four doses if they are younger than 1 month. More recently, alternate regimens using ceftriaxone and ciprofloxacin have also been proven to be efficacious, although rifampin remains the standard.

Prevention. An effective meningococcal vaccine that has been available in the United States for several years contains purified polysaccharide from the bacterial capsule of *N. meningitidis* serogroups A, C, Y, and W135. The recommended dose is one subcutaneous injection of 0.5 mL. Clinical efficacy of more than 65% is maintained for at least 3 years in persons immunized at 4 years of age or older, although protection wanes more rapidly in children vaccinated at younger than 4 years of age. The incidence of meningococcal disease in the United States is so low that mass administration of the vaccine is inappropriate; however, vaccination is recommended for persons without spleens, those with specific complement deficiencies, or those planning to travel to high-risk areas. Epidemics have occurred since 1980 in sub-Saharan Africa; New Delhi, India; Nepal; and Saudi Arabia. Because the areas in which epidemic meningococcal disease occur change from time to time, it is prudent to check with appropriate authorities to determine current recommendations for meningococcal vaccination before travel.

Pertussis

Pertussis, or whooping cough, was first recognized as a major threat in the 1500s. After the introduction of a vaccine in the 1940s, the incidence of pertussis dropped sharply among immunized populations. However, because recognition of rare side effects to pertussis vaccine, immunization rates have fallen. The age-specific incidence of pertussis in all age groups increased between 1981 and 1985 in the United States, with rates in persons 20 years and older increasing by thirteenfold during that period.

Epidemiology. Pertussis is found throughout the world. The incidence is currently highest in undeveloped countries, where immunization rates are low and socioeconomic conditions predispose to many communicable diseases. Pertussis is highly infectious, with attack rates of greater than 90% in unvaccinated household contacts. In the United States, most infections and the most severe disease occur in children under 5 years old. Transmission is by airborne particles from respiratory secretions of infected persons.

Bacteriology and Pathophysiology. *Bordetella pertussis* is a gram-negative coccobacillus. The organism produces several toxins when present in the respiratory tract. Pertussigen stimulates lymphocytosis and hemagglutination. Dermonecrotic toxin and tracheal cytotoxins damage respiratory epithelium. In addition, endotoxin is produced. During the course of the somewhat protracted disease, complications can occur that may cause death. The most serious of these are secondary pneumonia or encephalopathy. In addition, fits of coughing often result in pneumothorax, hemorrhage (facial, conjunctival, and CNS), and aspiration.

Clinical Presentation. Classic pertussis develops after an incubation period of 7 to 10 days. The disease appears in three stages: catarrhal, paroxysmal, and convalescent. The catarrhal stage lasts 1 to 2 weeks and resembles an undifferentiated upper respiratory tract infection with cough and mild fever. Progression of the cough to yield the classic whoop (which results when the patient gasps for breath after a prolonged coughing episode) marks the paroxysmal stage, which again can last as long as 2 weeks. During this stage, the WBC count may show marked lymphocytosis. Finally, cough resolves during the convalescent

stage. Death may occur from pertussis alone or from complications such as aspiration pneumonia. Recent case-fatality rates for Americans were 0.4% for all persons and 1% for patients younger than 1 year. The disease in adults is often milder, although it may show a severe classic pattern. Some investigators believe mild or atypical disease in adults may serve as a reservoir for infection of susceptible children.

Diagnosis. Culture of the organism from a nasopharyngeal swab is the most efficient way to diagnose pertussis; however, the culture is positive most frequently early in the illness, and late cultures (after the second week of illness) are rarely helpful. For optimal recovery, Bordet-Gengou media should be used, with methicillin added to reduce overgrowth by other bacteria present in the nasopharyngeal flora. Direct fluorescent antibody techniques may also be used to evaluate nasopharyngeal swabs or sputum samples for the presence of organisms.

Management. Treatment with antibiotics, unless begun in the incubation or catarrhal period, has little effect on the course of the disease. Antibiotics can reduce subsequent transmission to contacts, however, and should be instituted as soon as the diagnosis is made. Erythromycin, the drug of choice, should be given for at least 2 weeks. Other useful agents include doxycycline, trimethoprim-sulfamethoxazole, and chloramphenicol. In patients with severe disease, corticosteroids may provide some improvement. Perhaps more important than specific antibiotics is supportive care, including hydration, nutrition, care to maintain adequate ventilation, and supplemental oxygen. In addition, external stimuli, which seem to exacerbate symptoms, should be kept to a minimum.

Prevention. Immunization of children with diphtheria-pertussis-tetanus (DPT) vaccine is recommended by the CDC, because the benefits of immunization are greater than the risk of neurologic damage from the vaccine.[12] Introduction of new acellular pertussis vaccines may reduce this risk even further. Vaccination is not recommended for persons older than 7 years unless there is specific risk. The goal of treatment is to reduce secondary transmission. Close contacts under 7 years old who are unimmunized should be immunized, and persons who have not had a dose of DPT in 3 years should receive one. Furthermore, all close contacts under 1 year old should receive a 14-day course of erythromycin or trimethoprim-sulfamethoxazole, as should unimmunized contacts under 7 years old.

Diphtheria

Diphtheria, once a highly feared cause of morbidity and mortality in young people, can be controlled with appropriate use of a vaccine. However, according to some surveys, waning immunity has left many adults (18 years or older) with inadequate circulating levels of antitoxin against diphtheria.

Epidemiology. Humans are the natural host for *Corynebacterium diphtheriae*. Person-to-person spread occurs through contact with respiratory secretions or diphtheritic skin lesions. A carrier state exists in which people who have either been immunized or previously infected harbor the organism and asymptomatically transmit it to others. Evidence suggests that diphtheria can be transmitted through food or water, but this is not a major route of transmission.

Bacteriology and Pathogenesis. *C. diphtheriae* is a gram-positive, club-shaped bacillus. On Gram's stain, the clustered bacteria have the characteristic "Chinese letter" configuration. The organisms grow on standard media, but to avoid overgrowth of other oral flora, selective media (Loffler's culture medium or cysteine-tellurite agar) are suggested. The presence of a lysogenic bacteriophage in some *C. diphtheriae* organisms induces production of diphtheria toxin. The toxin is produced as a single molecule with two subunits, fragments A and B. Fragment B facilitates attachment to the cell membrane of host cells, and after attachment, fragment A enters the cell. Cell death results from large-scale disruption of protein synthetic capabilities.

Clinical Presentation. The most important manifestation of diphtheria is respiratory tract infection. Illness begins after an incubation period of about a week with nonspecific symptoms of malaise, fatigue, mild sore throat, and slight fever. The classic lesion is exudative pharyngitis progressing to a greenish-gray membrane that is difficult to dislodge. This membrane may spread over the posterior pharynx, tonsils, and uvula and down the respiratory tree to involve the larynx and trachea. Any one of these areas may be involved selectively, and the severity of illness is to some extent related to the area grossly involved. In severe disease, swollen tissues may result in a bull-neck appearance. Major complications include obstruction of the respiratory tract, which may result from direct parapharyngeal swelling or laryngeal involvement in young children, and sloughing of the tracheobronchial membrane in older patients. In addition to respiratory tract damage, toxin directly injures myocardial and neural tissue. Endocarditis occurs in some patients. Early signs in the first week of disease include ST-T wave depression and atrioventricular conduction abnormalities on the electrocardiogram. Congestive heart failure and cardiac enlargement may develop. Neurologic deficits usually begin with pharyngeal and cranial nerve paralysis. Cranial nerve paralysis may progress to bilateral motor paralysis, which generally resolves over a period of 3 to 6 months. In the tropics, cutaneous diphtheria is seen frequently. The skin lesions are not consistent in appearance and range from very superficial impetigo-like lesions to deep ulcers. In most cases of cutaneous disease, absorption of toxin is not great enough to cause the multisystem involvement seen in respiratory tract disease. The prevalence of skin lesions increases the overall likelihood of coming in contact with toxigenic *C. diphtheriae*.

Diagnosis. Reliable isolation of the organism requires a selective medium and several days of culture. Treatment should be started as soon as the patient is evaluated and is guided by clinical manifestations.

Management. Because the toxin and not the organism per se mediates life-threatening clinical manifestations of diphtheria, neutralization of absorbed toxin is crucial. A horse-derived antitoxin is available and should be administered as soon as the diagnosis is seriously considered. A 0.1 mL test dose of intradermal antitoxin diluted to a 1:1000 concentration (with a saline control) is observed for 20 minutes. If no reaction occurs, full doses can be given intravenously. Antitoxin should be diluted to 1:20 in saline and given no faster than 1 mL per minute. For mild cases, 20,000 units may be adequate, 40,000

units for moderate cases, and as much as 80,000 to 120,000 units for severely ill patients. Erythromycin or penicillin may be given to eradicate the carrier state, although their use has no effect on the clinical course of disease. Close observation is crucial to evaluate the need for respiratory support, especially in young children. Serial electrocardiograms and neurologic evaluation establish the onset of complications. If significant conduction abnormalities are present, continuous heart monitoring should be undertaken. Strict bed rest is recommended for all patients for 2 to 3 weeks.

Prevention. Close contacts of patients with respiratory diphtheria should receive diphtheria vaccine if they have not received at least three doses previously or if 5 or more years have elapsed since the last dose. In addition, unimmunized or partially immunized contacts should receive either intramuscular benzathine penicillin (600,000 units if younger than 6 years, 1.2 million units if older than 6 years) or 7 to 10 days of erythromycin (40 mg/kg/day for children or 1 g/day for adults, in four divided doses). Antitoxin is not recommended for contacts. The most important way to prevent diphtheria in adults, however, is to ensure that all adults receive a booster dose of diphtheria-tetanus toxoid every 10 years.

Tetanus

Tetanus was recognized by the early Greeks and is still a cause of infant and adult mortality. Today, the mortality approaches 90% and 40% for untreated infants and adults, respectively. Tetanus toxoid immunization has drastically reduced the incidence of disease in populations with high coverage rates.

Epidemiology. The bacterium and its spores are ubiquitous. Approximately 10% of people in the general population carry *Clostridium tetani* in fecal flora. Person-to-person spread is not an important cause of this disease. Disease occurs when the organism is introduced into an environment suitable for its growth, specifically wound sites with an anaerobic environment. In the developing world, the vast majority of cases are in neonates as a result of umbilical stump infections.

Bacteriology and Pathophysiology. *C. tetani* is a gram-positive, anaerobic, spore-forming rod. The spores are hardy and can occasionally survive boiling for short periods. After proliferating in an appropriate anaerobic environment, *C. tetani* releases the toxin tetanospasmin, which, in generalized disease, reaches the spinal column and CNS by hematogenous spread. The toxin is taken up by inhibitory neurons, where it interferes with release of inhibitory neurotransmitters, resulting in disinhibition of motor groups. Disinhibition of the sympathetic nervous system neurons occurs through a similar mechanism. The result is muscular spasm of varying severity and signs of sympathetic nervous system hyperactivity, including tachycardia, sweating, arrhythmias, and high blood pressure.

Clinical Presentation. A tetanus-prone wound precedes most adult disease, which may not be evident at the time of presentation. Localized tetanus, with spasm of a focal set of muscle groups, may occur and remain localized for weeks, then slowly resolve. This form of tetanus is much less common than is the generalized form, which often begins with trismus, or spasm of the masticator muscle group. Gradual onset of spasm of other muscle groups usually involves the trunk and extremities. Because the posterior muscles are stronger during spasms, the victim exhibits lumbar lordosis, with the neck and legs extended and arms flexed at the elbows (opisthotonos). Spasms seem to be exacerbated by external stimuli, such as sudden sound or light. The primary danger is loss of ability to breathe, especially during prolonged spasms. Respiratory failure is the main cause of death. The clinical picture in neonatal tetanus is similar, but begins with restlessness and failure to nurse, with progression to tetany and sympathetic overactivity. There is no definitive laboratory test to confirm the diagnosis of tetanus, but the clinical picture is adequate in the majority of cases.

Management. Emergency medical treatment of tetanus patients should include (1) excision of the wound, (2) administration of human tetanus immunoglobulin (at least 500 units, but a range of 500 to 3000 units is commonly used), and (3) administration of an antibiotic effective against *C. tetani*, such as penicillin or metronidazole.[70] Depending on the severity of the disease, different levels of supportive care and sedation may be appropriate. Diazepam may be given to mildly affected patients for sedation. Patients should be evaluated carefully for dysphagia. If dysphagia is present or other respiratory difficulties arise, endotracheal intubation or a tracheostomy should be performed. With prolonged spasms, hypoxia and cyanosis may occur; mechanical ventilation with pharmacologic paralysis is appropriate. At the same time, attention must be given to fluid balance and nutrition. Enteral feeding by a nasogastric tube is the least invasive way to supply both. Beta-blockers have been suggested to relieve symptoms of autonomic overactivity, such as tachycardia and hypertension, but there is no proven benefit to their prophylactic use. Sources of sensory stimulation should be reduced when the spasms are uncontrolled.

Prevention. Although rare cases of tetanus have occurred in previously immunized persons, immunization is considered at least 99.9% effective. Several vaccine formulations are now available in the United States. Children younger than 7 years may receive either DPT or DT (diphtheria and tetanus toxoid only) vaccine. A third vaccine, Td, is manufactured for use in persons at least 7 years old and consists of tetanus toxoid and a smaller amount of diphtheria toxoid than is present in the pediatric vaccines. A reduced amount of diphtheria toxoid is used in the adult preparation, because both the amount of toxoid and increasing age are associated with more severe reactions to vaccination. Adults who are unimmunized should be given a series of three doses (0.5 mL intramuscularly) of Td, with the second dose 4 to 8 weeks after the first and the third dose 6 to 12 months after the second. A booster should be given every 10 years thereafter. All travelers should know when they were last immunized and stay up to date with booster doses. From the standpoint of tetanus prevention, care of wounds is crucial. The tetanus-prone wound, contaminated with dirt or feces or caused by puncture, crush, avulsion, or frostbite, should be cleaned and debrided appropriately. Persons with tetanus-prone wounds should receive 250 to 500 units of human tetanus Ig if their immunization history is unknown or their immunization series is incomplete. These persons should also receive a dose of Td and complete an immunization series. Persons fully immunized and given an appropriate booster before a tetanus-prone wound should not receive tetanus Ig. If they have not

received a booster within 5 years, however, they should get a dose of Td.

MAJOR PROTOZOAN INFECTIONS OTHER THAN MALARIA

African Trypanosomiasis

Trypanosoma brucei rhodesiense and *T. brucei gambiense* are important infectious diseases in Africa and have provided remarkable insights into the importance of antigenic variation as a strategy used by parasites to avoid the immune response.[34,39,72] *T. brucei gambiense* causes African sleeping sickness, and *T. brucei rhodesiense* causes an acute disease that may end in heart failure. The parasites are transmitted to humans by tsetse flies (*Glossina* species) in sub-Saharan Africa. Metacyclic promastigotes are injected into the bloodstream through the saliva of the biting tsetse fly and divide into long slender forms in the bloodstream. These eventually differentiate into short stumpy forms, which are taken up in the blood meal of the tsetse. Once in the fly, the parasite differentiates into procyclic forms. It takes approximately 3 weeks for the protozoa to develop into infective metacyclics within the tsetse fly.

Clinical Manifestations. In nonimmune individuals, the initial sign of infection is a nodule at the site of the tsetse fly bite. This lesion becomes erythematous and painful over a period of 1 week and recedes after several days. Dissemination of the trypanosome throughout the body causes clinical symptoms, notably fever, headache, and severe malaise. On physical examination, enlarged supraclavicular and posterior cervical lymph nodes are noted. This phase of illness lasts several days and is followed by an asymptomatic period of several weeks. The acute phase may then recur. In the case of *T. brucei gambiense* infection, symptoms are less severe and evolve into a syndrome characterized by behavioral changes and chronic somnolence. *T. brucei rhodesiense* infections cause severe anemia, frequent episodes of fever, and eventual heart failure and severe CNS involvement.

Diagnosis. Definitive diagnosis depends on identification of parasites in the blood, lymphatics, or CSF. Thick blood smears and buffy coat preparations should be stained with Giemsa stain and examined for the presence of trypanosomes. The CSF should be subjected to centrifugation and the sediment examined for parasites. Associated laboratory abnormalities include anemia, monocytosis, and elevated serum and CSF IgM levels.

Management. Suramin (available from the CDC) should be used for treatment of early *T. brucei rhodesiense* infection, although the drug may cause proteinuria. A test dose of 100 mg intravenously is first given to detect possible idiosyncratic reactions. If tolerated, 1 g should be given on the initial day of treatment and 3, 7, 14, and 21 days later. If CNS involvement is diagnosed or strongly suspected (CSF lymphocytosis and elevated IgM), melarsoprol (available from the CDC) should be administered. This drug should be given at an initial dose of 1.5 mg/kg of body weight, with gradually increasing doses (e.g., increase every 48 hours to 3.6 mg/kg over 2 weeks). After a week with no drug given, additional injections of 3.6 mg/kg are given every other day 3 times. This arsenical compound is toxic, causing encephalopathy and exfoliative dermatitis, and should be used only in a controlled hospital setting. For early *T. brucei gambiense* infection, pentamidine (4 mg/kg body weight intramuscularly, up to 300 mg/kg given over 7 days) is the treatment of choice. Eflornithine, 100 mg/kg every 6 hours for 14 days, should be used for more advanced cases of this infection.

South American Trypanosomiasis (Chagas' Disease)

T. cruzi is transmitted to humans by triatomids that live in the cracks of mud-built homes in Central and Latin America. These insects are common in areas of Brazil, Venezuela, and Argentina with poor socioeconomic development. The infection has been reported as far north as the southern United States. Affected individuals generally do not recall initial contact with the insects, during which time triatomid feces containing the protozoan organisms are deposited on broken skin or mucous membranes multiply within local macrophages. The macrophages rupture and elicit an inflammatory reaction that appears as a nodule with slightly painful satellite nodules or draining lymph nodes. A symptomatic phase, characterized by fever and diffuse lymph node enlargement, develops subsequently. Hepatosplenomegaly may also occur. In severe cases, acute myocarditis, pericarditis, or endocarditis is seen. After several months, the acute phase resolves, and chronic disease appears, characterized by cardiomyopathy, megaesophagus, or megacolon.[1] It is rare for the traveler to develop these signs or symptoms. Diagnosis during the acute phase may be made by demonstration of parasites in leukocytes in Giemsa-stained blood smears. Amastigotes of *T. cruzi* may also be present in biopsy specimens of lymph nodes or muscle. Elevated IgM antibody titers to *T. cruzi* (performed by the CDC) also support the diagnosis. In the chronic phases of Chagas' disease, the clinical findings of cardiomyopathy, megaesophagus, or megacolon, in concert with isolation of *T. cruzi* from blood, support the diagnosis. To detect trypanosomes in blood, uninfected triatomids are permitted to feed on the patient's forearm for 30 minutes. The insects are then kept for 30 days, and the intestinal contents of the insect inspected for *T. cruzi*. If negative, the examination may be repeated 60 days later. This test is positive in about 50% of cases. Serologic tests, including complement fixation anti-*T. cruzi* antibodies, are also useful but may be positive in long-term residents of endemic areas. Acute Chagas' disease is treated with nifurtimox, 8 to 10 mg/kg body weight orally per day in 4 divided doses for 120 days. The drug is available from the CDC.

Leishmaniasis

Humans may be infected by *Leishmania* species that cause skin, mucocutaneous, or visceral disease. These intracellular parasites are transmitted by phlebotomine sandflies. Various forms of the infection occur throughout Latin and Central America, Africa, the Middle East, and Asia (Fig. 78-2). Cutaneous lesions caused by *L. tropica* and *L. tropica major* are referred to as "Oriental sores" in Asia and the Middle East. In Central and South America, *L. mexicana* and *L. braziliensis* cause skin lesions characterized in the chronic phase as nonhealing ulcers that frequently become secondarily infected by bacteria. Espundia, or mucocutaneous leishmaniasis caused by *L. braziliensis,* begins as a single nodule and eventually involves the oropharyngeal or nasal mucosa, where it causes severe destruction. This disease occurs primarily in residents of the Amazon basin. Kala-azar, or visceral leishmaniasis, is caused by *L. donovani* in Africa and Asia. Affected individuals generally do not recall an initial skin

Figure 78-2. Old-World leishmaniasis. (Courtesy of Richard Kaplan, MD.)

Figure 78-3. Mild local dermatitis associated with contact with cercariae, the infective stage of *Schistosoma* spp. released by snails. (From Ryan ET, Wilson ME, Kain KC: Illness after international travel. N Engl J Med 2002;347(7):505–516.)

lesion. Several months after inoculation, fever, abdominal discomfort, and weakness develop and become progressively more severe. Nausea and vomiting are protracted, the skin becomes dry and dark, and abdominal distention with hepatosplenomegaly eventually appears. This disease is rare in travelers and nonresidents of endemic areas.

MAJOR HELMINTHIC INFECTIONS

Worm infections are common among travelers to developing countries, especially among persons who spend time in rural areas. However, unlike many viral and protozoan infections, helminths rarely cause life-threatening disease, and infested persons are often asymptomatic.

Schistosomiasis

Three major species of schistosomes infect humans: *Schistosoma mansoni, S. haematobium,* and *S. japonicum*. *S. mansoni* infection occurs in South America and Africa. *S. haematobium* infection occurs primarily in Africa, especially Egypt and East Africa. *S. japonicum* infection is present exclusively in the Far East. Schistosomiasis is transmitted by freshwater snails. These snails release cercariae that penetrate the skin of humans. The cercariae rapidly transform into schistosomulae, which migrate to the lungs and eventually the portal (in the case of *S. mansoni* and *S. japonicum*) or vesical (in the case of *S. haematobium*) venous system to differentiate into adult worms. Fecund female worms release eggs, which may be passed in feces or urine. Miracidia released from this stage may then infect snails in water used for bathing, washing clothes, or other communal activities.

Signs and symptoms of infection vary among the three schistosome species.[2] The initial presentation of acute *S. mansoni* infection may include fever, anorexia, weight loss, and abdominal pain. This unusual symptom complex, which occurs in individuals with heavy infection, has been referred to as Katayama fever and appears 18 to 60 days after exposure. Travelers with light or moderate exposure, however, usually have no specific signs or only mild local dermatitis associated with contact with cercariae, the infective stage of the parasite released by snails (Fig. 78-3). In persons with established infections, the prevalence of clinical manifestations is low. Most individuals have no signs specifically attributable to *S. mansoni* infection. Hepatomegaly or splenomegaly, attributable to portal hypertension after granulomatous reactions to eggs deposited in the liver, occurs in 15% of subjects. Eggs may also embolize to the lungs and induce granulomatous lesions and cor pulmonale. Those at greatest risk are persons who have the heaviest intensity of infection as judged by fecal egg counts. These complications may ultimately result in esophageal and gastrointestinal varices, which cause acute blood loss. The manifestations of schistosomiasis japonicum are similar to schistosomiasis mansoni, except that Katayama fever appears to be more frequent in the former case. In addition, there is a unique manifestation of *S. japonicum* infection attributable to embolization of eggs to the brain. Generalized or jacksonian seizures are the major signs of cerebral schistosomiasis. Since *S. haematobium* adult worms inhabit the venous system of the genitourinary tract, the signs and symptoms of this helminth infection are primarily secondary to granulomatous reactions to eggs present in the ureters and bladder wall. Dysuria and hematuria have been reported in many individuals who reside in endemic areas.

The major risk to travelers is encountered when exposure to large numbers of cercariae occurs by bathing in fresh water that contains infective snails. Cases of transverse myelitis have been reported in these circumstances. Appropriate preventive measures include counseling to avoid bathing or swimming in fresh bodies in endemic areas.

Filariases

Three major types of human filariasis exist. Infections caused by *Onchocerca volvulus* are manifest primarily as skin and eye disease. *Brugia malayi* and *Wuchereria bancrofti* cause lymphatic filariasis. *Loa loa* infection may cause skin disease. Each of these is described separately, because their ecologies and manifestations are distinctive.

Onchocerciasis

O. volvulus is transmitted to humans by *Simulium* species of blackflies in Central America and West and Central Africa. Infective, or third-stage, larvae eventually develop into adult

Figure 78-4. Calabar swellings of loiasis (loaiasis). (From Ryan ET, Wilson ME, Kain KC: Illness after international travel. N Engl J Med 2002;347(7):505–516.)

worms contained in deep subcutaneous nodules that are asymptomatic and may be palpable. Microfilariae are released from adult female worms and cause dermatitis as they migrate through the skin. The organisms have a propensity to invade the eye (especially the anterior chamber and cornea), where they cause blindness. Diagnosis is based on prolonged residence in an endemic area (e.g., as in Peace Corps volunteers) and parasitologic identification in skin snips or slit-lamp examination of the eye.

Lymphatic Filariasis

B. malayi and *W. bancrofti* are transmitted by mosquitoes. Infective larvae eventually develop into lymphatic-dwelling adult worms, which release microfilariae into the bloodstream. Although chronic infection and recurrent exposure are associated with a wide variety of clinical manifestations, including tropical pulmonary eosinophilia, acute lymphangitis, and elephantiasis, these manifestations are rare in nonresidents of endemic areas. The only definitive diagnostic test is identification of parasites in the bloodstream. Because nonresidents and many residents who are infected may not have detectable parasitemia, other laboratory studies (eosinophilia, elevated serum IgE level) must be used as aids in diagnosis. Diethylcarbamazine (cumulative dose of 72 mg/kg body weight given over 2 weeks) is the treatment.

Loiasis (Loaiasis)

Loa loa is transmitted to humans by the bites of tabanid flies that live along river edges in Central and West Africa. Microfilariae migrate in the bloodstream, whereas adult worms migrate in cutaneous tissues. The major disease manifestation is calabar swellings, which are characterized as egg-sized or smaller raised lesions, predominantly over the extremities, that are tender and surrounded by edematous skin (Fig. 78-4). They may migrate and last several days. Their pathogenesis may be related to migration of adult worms or release of antigens that elicit immunologic hypersensitivity reactions. Treatment is with diethylcarbamazine at a dose of 9 mg/kg body weight/day for 3 weeks. Retreatment is occasionally required.

Intestinal Helminth Infections

Ascariasis

Approximately 25% of the world's population is infected with *Ascaris lumbricoides*. Although this nematode contributes significantly to morbidity in children with poor nutrition, it generally does not cause significant health problems for the traveler. The helminth is transmitted by eggs contained in ingested pieces of soil, such as may be found on vegetables grown in countries with poor hygienic conditions. It is not limited to tropical climates and occurs in North America and Europe. Ingested eggs enter the small intestine. Larvae leave the eggshell to penetrate the mucosa and eventually enter the bloodstream and lymphatics. Between 1 and 5 days after infection, they enter the liver and, at about 14 days, the lungs. The larvae then rupture through the alveoli, ascend the trachea, and return to the intestine on being swallowed. In the small intestine, adult males and females develop into macroscopic worms (12 to 25 cm long). Eggs passed via feces continue the life cycle. *Ascaris* infection is often asymptomatic, but several syndromes are associated with tissue and intestinal phases of infection. Persons who are recurrently exposed may develop pulmonary ascariasis, characterized by cough, wheezing, eosinophilia, and fleeting pulmonary infiltrates on chest x-ray examination. Children may suffer from intestinal or biliary tract obstruction from infestations with large numbers of worms as a consequence of repeated ingestion of *Ascaris* eggs. Intestinal symptoms are seen mainly in persons with heavy infection, an uncommon situation in the traveler.

Diagnosis of ascariasis may be made by identification of one of several parasite stages. Adult ascarids occasionally migrate from the mouth or anus. Ascaris larvae may rarely be observed in sputum or gastric washings. The most common means of diagnosis is identification of eggs in feces. Eggs are ovoid, 35 to 70 mm in diameter, and consist of an outer white shell and brownish ovum internally. The eggs are not produced until approximately 9 weeks after infection. Intestinal ascariasis is treated with albendazole or mebendazole.[13] An alternative regimen that avoids the use of benzimidazoles (e.g., for treatment of pregnant women) is pyrantel pamoate (11 mg/kg body weight to a maximum of 1 g).

Hookworm

Ancylostoma duodenale and *Necator americanus* infections occur most commonly in the tropics but also in temperate climates where sanitation is poor. Hookworm is second only to *Ascaris lumbricoides* in terms of the number of people infected. Humans are infected percutaneously by third-stage larvae in the soil. The larvae enter the bloodstream, pass to the lungs, and rupture the alveolar lining to eventually ascend the trachea and descend the esophagus to differentiate into adult worms. These adult worms contain cutting plates on the anterior end and feed on host blood obtained through their attachment sites in the upper small intestine. It has been estimated that each *N. americanus* infection causes 0.03 mL of blood loss per day, whereas the *A. duodenale* hookworm consumes 0.26 mL per day. Iron deficiency anemia, especially in persons with low iron intake, is the major clinical manifestation of hookworm infection.[31] The diagnosis may be made by identification of hookworm eggs in feces. The eggs are round, 40 to 60 mm in diameter, and have a "smoother" shell than do *Ascaris* eggs. Although multiple drugs are effective in treatment, albendazole is most readily

Figure 78-5. Migrating animal hookworms, such as *A. braziliense* and *A. caninum*, may create serpiginous lesions in superficial tissues of humans. (From Ryan ET, Wilson ME, Kain KC: Illness after international travel. N Engl J Med 2002;347(7):505–516.)

available.[13] Supplemental iron should be given to persons when necessary. Infection with hookworm is rare in the traveler from a developed country. Migrating animal hookworms, such as *A. braziliense* and *A. caninum*, may create serpiginous lesions in superficial tissues of humans (Fig. 78-5).

Strongyloidiasis

Strongyloides stercoralis infection occurs in tropical and temperate regions. The infection is initiated by contact with soil containing infective third-stage larvae. The helminth follows a route within the host similar to that described for hookworms. In addition, there is an autoinfection cycle in which larvae released in the intestine may penetrate the mucosa directly and then migrate through the liver and lungs. This occurs only in immunocompromised individuals. Many persons with *S. stercoralis* infection are asymptomatic. Some persons, however, have cutaneous or intestinal manifestations. The former are urticarial lesions around the buttocks and waist that last 1 to 2 days. These are secondary to penetration of larvae present in the feces. Other symptoms include indigestion, abdominal cramps, and diarrhea. Diagnosis is made by identification of larvae in fresh stools or gastrointestinal washings. Rhabditiform larvae with a length of 250 mm and a width of 10 to 20 mm are most commonly observed, although filariform larvae may also be present. Treatment is with ivermectin.[13]

Enterobiasis

Enterobiasis, or pinworm infection, exists in all parts of the world. Eggs are passed from female worms in the colon. Infection is transmitted by ingestion of *Enterobius vermicularis* eggs, which develop into gravid adult female worms in the large bowel. The infection is especially common in crowded settings where sanitation is poor. The diagnosis may be made by identification of adult worms migrating along the perianal area or by eggs deposited in the same area. Eggs are detected by applying a piece of sticky cellophane tape to the area and inspecting it microscopically. Treatment is with pyrantel pamoate or albendazole.[13] Repeated treatments as a result of reinfection in crowded settings is commonly required.

The references for this chapter can be found on the accompanying DVD-ROM.

Natural Disaster Management

79

Eric K. Noji and Catherine Y. Lee

Throughout history, natural disasters have exacted a heavy toll of death and suffering.[1] Most recently, the great tsunami in South Asia and the earthquake in Bam Iran resulted in hundreds of thousands of deaths, injuries, and homelessness[126,133] (Table 79-1 and Figs. 79-1 to 79-6). The compounding toll of disasters and human suffering has not improved despite much attention by the international scientific community.[63] Global climate change brings the potential for severe weather events, flooding, and introduction of tropical vector-borne diseases into more temperate regions.[52,137] Increasing population density near coasts, in flood plains, and in regions of high risk points to the probability of future catastrophic natural disasters, with millions of casualties.

Disasters affect communities in numerous ways. Roads, telephone lines, and other transportation and communication links are often destroyed.[20] Public utilities and energy supplies may be disrupted.[73] Substantial numbers of victims may be rendered homeless.[92] Portions of the community's industrial or economic

Figure 78-5. Migrating animal hookworms, such as *A. braziliense* and *A. caninum*, may create serpiginous lesions in superficial tissues of humans. (From Ryan ET, Wilson ME, Kain KC: Illness after international travel. N Engl J Med 2002;347(7):505–516.)

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TABLE 79-1. Selected Natural Disasters, 1970–2005

YEAR	EVENT	LOCATION	APPROXIMATE DEATH TOLL
1970	Earthquake/landslide	Peru	70,000
1970	Tropical cyclone	Bangladesh	300,000
1971	Tropical cyclone	India	25,000
1972	Earthquake	Nicaragua	6,000
1976	Earthquake	China	250,000
1976	Earthquake	Guatemala	24,000
1976	Earthquake	Italy	900
1977	Tropical cyclone	India	20,000
1978	Earthquake	Iran	25,000
1980	Earthquake	Italy	1,300
1982	Volcanic eruption	Mexico	1,700
1985	Tropical cyclone	Bangladesh	10,000
1985	Earthquake	Mexico	10,000
1985	Volcanic eruption	Columbia	22,000
1988	Hurricane Gilbert	Caribbean	343
1988	Earthquake	Armenia SSR	25,000
1989	Hurricane Hugo	Caribbean	56
1990	Earthquake	Iran	40,000
1990	Earthquake	Philippines	2,000
1991	Tropical cyclone	Bangladesh	140,000
1991	Volcanic eruption	Philippines	800
1991	Typhoon/flood	Philippines	6,000
1991	Flood	China	1,500
1992	Hurricane Andrew	USA	52
1993	Earthquake	India	10,000
1995	Earthquake	Japan	6,000
1998	Hurricane Mitch	Central America	10,000
1999	Earthquake	Turkey	18,000
1999	Earthquake	Taiwan	1,000
2001	Earthquake	India	20,000
2003	Earthquake	Algeria	3,000
2003	Earthquake	Iran	25,000
2004	Tsunami	South Asia	300,000

Data from Office of U.S. Foreign Disaster Assistance: Disaster history: Significant data on major disasters worldwide, 1900-present. Washington, DC, Agency for International Development, 2005.

base may be destroyed or damaged. Casualties may require medical care, and damage to food sources and utilities may create public health threats.[84,97] The more remote the area, the longer it takes for external assistance to arrive, and the more the community must rely on its own resources, at least for the first several hours, if not days.[1] Good disaster management requires accurate information and must link data collection and analysis to an immediate decision-making process.[107] The overall objective of disaster management from a public health perspective is to assess the needs of disaster-affected populations,[77,125] match available resources to those needs, prevent further adverse health effects, implement disease control strategies for well-defined problems, evaluate the effectiveness of disaster relief programs,[160] and improve contingency plans for various types of future disasters.[109] Common patterns of morbidity and mortality after certain disasters can be identified[111] (Table 79-2). Effective emergency medical response depends on anticipating these different medical and health problems before they arise[105] and delivering appropriate interventions (relief supplies, equipment, and personnel) at the precise times and places where they are needed most.[62,102]

NATURE OF DISASTER

The World Health Organization (WHO) defines disaster as a sudden ecologic phenomenon of sufficient magnitude to require external assistance.[118,164] At the community level, this can be defined operationally as any community emergency that seriously affects people's lives and property and that exceeds the community's capacity to respond effectively.[65] The essence of a disaster is substantial environmental damage, which may be accompanied by large numbers of casualties. This chapter refers to limited incidents creating relatively small numbers of casualties and slight environmental disturbance as "multiple casualty incidents." The term *disaster* is reserved for incidents that cause great disruption of the physical and social environments and that require extraordinary resources and special medical care, even in the absence of mass casualties.

PAST PROBLEMS IN NATURAL DISASTER MANAGEMENT

In ancient times, little mitigation was possible against the effects of disaster. Today, communications inform us rapidly of disasters and allow us to provide effective medical aid to victims. This requires adequate planning and brisk execution. Medical aid in many previous disasters has been well intentioned but poorly organized, with limited benefits.[43,118]

Health decisions made during emergencies are often based on insufficient, nonexistent, or even false information, which results in inappropriate, insufficient, or unnecessary health aid, waste of health resources, or countereffective measures.[135] For example, large amounts of useless drugs and other consumable supplies are frequently sent to a disaster site. After the 1976 earthquake in Guatemala, 100 tons of unsorted medicines were airlifted to the country from foreign donors.[35,36] Of these supplies, 90% were of no value because they consisted of medications that had expired, were already opened, or carried labels written in foreign languages. A similar situation occurred after the 1988 Armenian earthquake, when international relief operations sent at least 5000 tons of drugs and consumable medical supplies. Because of the difficulties with identification and sorting, only 30% of the drugs were immediately usable by the health workers in Armenia, 11% were useless, and 8% had expired. Ultimately, 20% of all the drugs provided by international aid had to be destroyed.[3] Other examples of inappropriate aid include sending mobile hospitals and teams of specialized trauma or emergency medicine specialists that arrive much too late and sending unprepared medical volunteers when nonmedical relief workers (e.g., sanitation engineers) would be more appropriate. The arrival of unprepared and inexperienced foreign personnel may damage the relief effort by tying up communication, transportation, and housing. These problems are all compounded in the vacuum created by the disaster, including the lack of communication, transportation, local supplies and support, and a decision-making structure. Because these relief operations are often conducted under the watchful eye of the media, medical relief efforts are often pejoratively called the second disaster.[75,102]

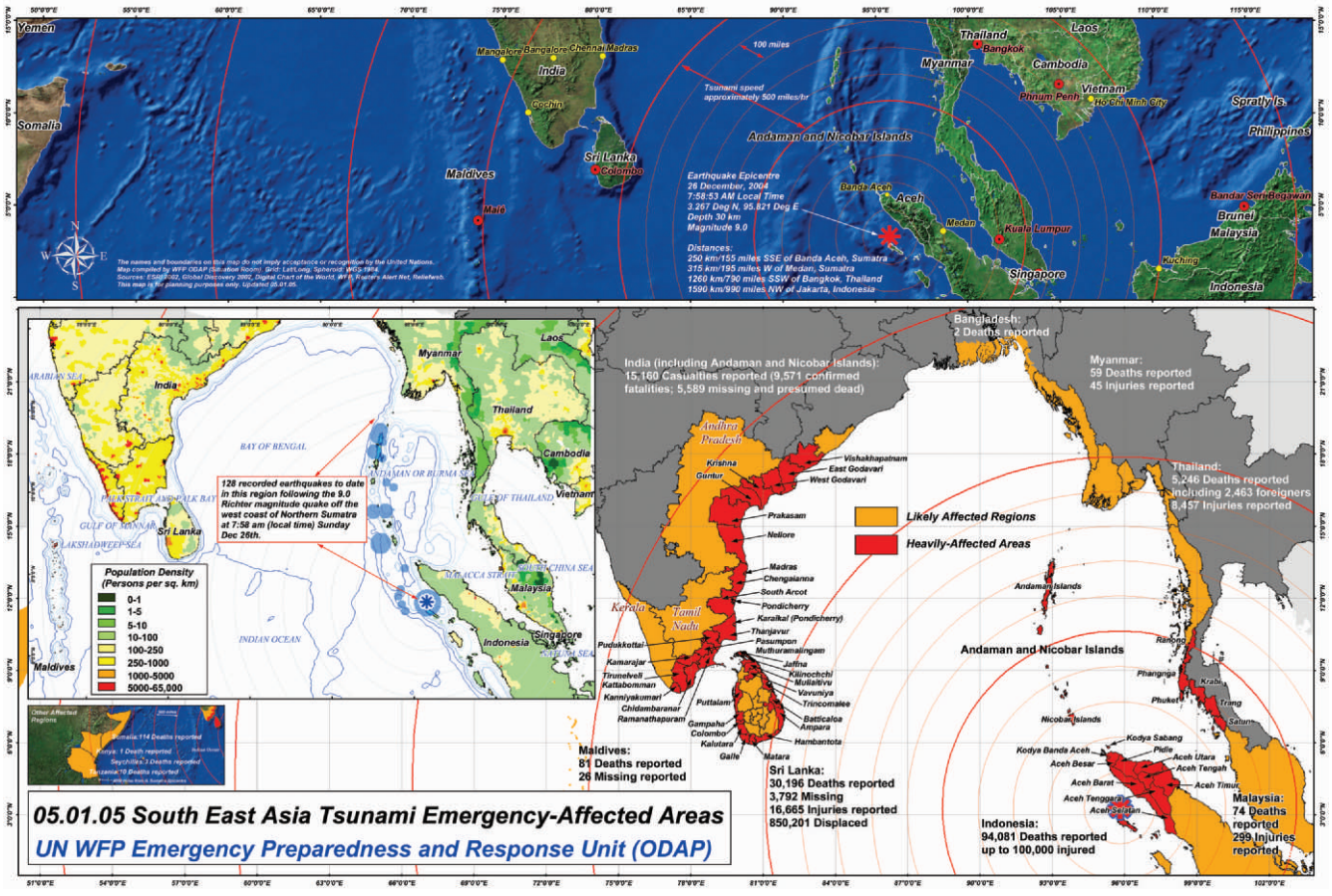


Figure 79-1. Map showing the areas most affected by the 26 December 2004 tsunami. (Courtesy Humanitarian Early Warning Service [HEWSweb]. Available at www.hewsweb.org/downloads/home_page/maps/pdf/20041227Tsunami.pdf.)

Figure 79-2. The 26 December 2004 tsunami in South Asia. Incoming waves rushed in quickly with great force and volume, leading to the deaths of nearly 400,000 people across 15 nations. (Courtesy U.S. Navy.)

Figure 79-3. Thailand suffered great damage in the 26 December 2004 tsunami.

Figure 79-4. Photo of the 26 December 2004 tsunami in south Asia. (Courtesy U.S. Navy.)

INFORMATION MANAGEMENT SYSTEMS FOR DISASTER RESPONSE

Over the past several years, efforts have been made to develop rapid and valid disaster damage assessment techniques.[153,164] These techniques must be able to define quickly the overall effects of the disaster impact, nature and extent of the health problems, groups in the population at particular risk for adverse health events, specific health care needs of the survivors, local resources to cope with the event, and extent and effectiveness of the response to the disaster by local authorities.[65,143] Guha-Sapir and Lechat[53–55] have developed indicators for needs assessment in earthquakes ("quick and dirty" surveys), highlighting simplicity, speed of use, and operational feasibility. The techniques employed (e.g., systematic surveys, simple reporting systems) are methodologically straightforward. With suitable personnel and transport, estimates of relief needs can be quickly obtained.[26] Problems may arise, however, with interpretation of

Figure 79-5. The 26 December 2003 earthquake in Bam, Iran, magnitude 6.6, resulted in tens of thousands of injuries and at least 25,000 deaths (early reports estimated as high as 45,000 deaths). (Photo by Medhi Ghasemi, Iranian Students' News Agency.)

Figure 79-6. The 26 December 2003 earthquake in Bam, Iran, demolished many of the mud brick and clay homes of the town. (Courtesy U.S. Geological Survey.)

TABLE 79-2. Short-Term Effects of Major Natural Disasters

EFFECT	EARTHQUAKES	HIGH WINDS	TSUNAMIS	FLASH FLOODS
Deaths	Many	Few	Many	Few
Severe injuries requiring extensive care	Overwhelming	Moderate	Few	Few
Increased risk for communicable diseases	Potential (but small) risk after all major disasters (probability rises as overcrowding increases and sanitation deteriorates)			
Food scarcity	Rare (may occur for reasons other than food shortage)	Rare	Common	Common
Major population movements	Rare (may occur in heavily damaged urban areas)	Rare	Common	Common

Modified from Office of Emergency Preparedness and Disaster Relief Coordination: Emergency Health Management After Natural Disaster. Washington, DC, Pan American Health Organization, 2002.

data, particularly incomplete data, and in developing countries in which predisaster health and nutritional levels are unknown.

The ultimate goal of surveillance is to prevent or reduce adverse health consequences of the disaster itself, as well as to optimize the decision-making process associated with management of the relief effort. These epidemiologic objectives can be simply defined as the surveillance cycle: the collection, analysis, and response to data.[43] The surveillance cycle must be repeated many times: immediately, with rapid assessments of problems using the most rudimentary data collection techniques; then short-term assessments involving establishment of simple but reliable sources of data; and subsequently, ongoing surveillance to identify continuing problems and monitor response.

Field surveillance methods vary greatly by disaster setting and by personnel and time available. Early field surveys must be simple and provide immediate answers that will directly prevent loss of life or injury. Subsequently, surveys can address issues such as availability of medical care, assessment of the need for specific interventions, and epidemic control (a rumor clearinghouse), each of which demands more careful investigation. Surveillance must determine whether the effort is having a tangible impact on the population, or whether new strategies are needed.[147] Surveillance becomes an iterative cyclical process in which simple health outcomes are constantly monitored and interventions assessed for efficacy.

Finally, linking postdisaster information to a decision-making process is important.[55] In the rapid evolution of a disaster relief program, major decisions regarding relief are made early, hastily, and often irreversibly, so reliable early data to assist in making these decisions are vital.[165] Organized data collection in disaster situations can greatly improve decision making. Adaptable questionnaires can assist in an efficient data collection operation.

Operational decisions depend on the phase of the disaster. In the early phase of relief, basic needs of water, food, clothing, shelter, and medical care must be met, after which the long-term process of rebuilding proceeds. Relief aid can often be squandered by overreacting to minor problems when excitement is great, needs are extensive, and scrutiny by the media is omnipresent.[135] Because everyone in the disaster area has needs and experiences loss, the challenge of early assessment is to decide where initial interventions will prevent the greatest loss of life or severe morbidity. The postimpact phase requires information on long-term rehabilitation and restoration of health services. Epidemiologic assessment, prioritization of needs, and ordering an appropriate response can have a major impact on the community's ability to return to normalcy in both the short and longer term.[101]

HEALTH CARE NEEDS IN SPECIFIC NATURAL DISASTERS

Natural hazards that can cause substantial property damage, economic dislocation, and medical problems include earthquakes and associated phenomena, volcanic eruptions, and extreme weather incidents, such as heat waves and blizzards. Accounts of morbidity and mortality recorded after previous disasters can predict the medical care needs of future disaster victims and provide a foundation for disaster response planning.[87]

Figure 79-7. During summer 1988, monsoon rains resulted in the most severe flooding ever recorded in Bangladesh. Water covered three fourths of the land area of Bangladesh, displacing up to 40 million persons from their homes. (Courtesy U.S. Centers for Disease Control and Prevention [CDC] and U.S. Public Health Service.)

Figure 79-8. Florence, South Carolina, September 6, 2004. A car floating in a flooded road. The flooding is a result of Hurricane Frances. (Courtesy U.S. Federal Emergency Management Agency [FEMA].)

Floods

Floods are the most common natural disasters. They affect more people worldwide and cause greater mortality than any other type of natural disaster.[35,114,162] They occur in almost every country, but 70% of all flood deaths occur in India and Bangladesh[96] (Fig. 79-7). In the United States, floods cause more deaths than any other natural disaster, with most fatalities resulting from flash floods.[45]

Fast-flowing water carrying debris such as boulders and fallen trees accounts for the primary flood-related injuries and deaths. The main cause of death from floods is drowning, followed by various combinations of trauma, drowning, and hypothermia with or without submersion (Fig. 79-8).[9] Persons submerged in

cold water for up to about 40 minutes have been successfully resuscitated with 100% recovery of neurologic function.[114] Unfortunately, such resuscitations from clinical death require technologically advanced measures, which may not be available for days after a flood, even in a highly developed country such as the United States. Among flood survivors, the proportion requiring emergency medical care is reported to vary between 0.2% and 2%.[136,144] Most injuries requiring urgent medical attention are minor and include lacerations, skin rashes, and ulcers.[118] However, flood-associated lacerations are frequently contaminated, so primary wound closure should be done with caution. Primary closure without careful evaluation of the wound almost always requires reopening the wound and additional treatment within 24 to 48 hours.[9]

Increased incidence of snakebites was reported after floods in India and the Philippines.[158] In India, most snakebites were by cobras that had been driven by rising water to seek higher ground near towns and villages. For some floods, substantial numbers of casualties caused by fire have been documented.[114] Fast-flowing water can break oil or gasoline storage tanks. If the film of oil is ignited, the fire may spread to buildings on land.

From a public health viewpoint, floods may disrupt water purification and sewage disposal systems, cause toxic waste sites to overflow, or dislodge chemical containers stored aboveground.[39] In addition, makeshift evacuation centers with insufficient sanitary facilities may become substantially overcrowded.[118] Combination of these events may contribute to increased exposure to highly toxic biologic and chemical agents. Examples include the potential for waterborne disease transmission of such agents as enterotoxigenic *Escherichia coli, Shigella, Salmonella,* and hepatitis A virus.[136] The risk of transmission of malaria and yellow fever may be increased because of enhanced vector-breeding conditions.[118] In 1973, Ussher[158] reported that the most serious problems encountered after a flood in the Philippines were viral upper respiratory tract infections, probably caused by crowded conditions in temporary shelters.

Despite the potential for communicable diseases to follow floods, mass vaccination programs are counterproductive for a variety of reasons. They not only divert limited personnel and resources from other critical relief tasks, but also may create a false sense of security and cause persons who have been vaccinated to neglect basic hygiene.[37] Unfortunately, after floods the public often demands typhoid vaccine and tetanus toxoid, although no epidemic of typhoid after a flood has ever been documented in the United States.[82] In addition, antibodies to typhoid after immunization take several weeks to develop, and even then, vaccination protects only moderately. Likewise, mass tetanus vaccination programs are not indicated. Management of flood-associated wounds should include appropriate evaluation of the injured person's tetanus immunization history, and the person should be vaccinated only if indicated.

The proper approach to the problem of communicable diseases is to set up an epidemiologic surveillance system so that an increase in cases of communicable diseases in the flood-stricken area can be identified quickly. Particular attention should be given to diseases endemic to the area. For example, when floods occur in areas with endemic arthropod-borne encephalitides, arthropods known to transmit the disease should be monitored and areas should be sprayed if the vector population increases significantly after the flood.

Figure 79-9. New Orleans, Louisiana, following flooding caused by Hurricane Katrina in September 2005. (Courtesy Luanne Freer.)

Tropical Cyclones (Hurricanes or Typhoons)

Cyclones (rotating storm systems that may be called *cyclones, hurricanes,* or *typhoons* depending on which area of the globe they occur in) have killed hundreds of thousands and injured millions of people during the last 30 years (see Table 79-1).[57,113] From 1900 to 1999, more than 14,000 people lost their lives in hurricanes in the United States (see Table 79-1).[42] The greatest natural disaster in U.S. history, as measured by the number of deaths, occurred on September 8, 1900, when a hurricane struck Galveston, Texas, and killed more than 6000 persons.[46,96] In 1970, deaths resulting from a single tropical cyclone striking Bangladesh were estimated to exceed 250,000. As population growth continues along vulnerable coastal areas, deaths and injuries resulting from tropical cyclones will increase.[55,139]

Although hurricane winds do great damage, wind is not the primary killer in a hurricane. Hurricanes are classic examples of disasters that trigger secondary effects such as tornadoes and flooding that, together with storm surges, can cause extraordinarily high rates of morbidity and mortality. This was seen after the 1991 cyclone and sea surge in Bangladesh, in which 140,000 persons drowned; during Hurricane Mitch in Central America in 1998, with thousands of drowning deaths[50]; and after Hurricane Katrina in the United States in 2005 (Figs. 79-9 and 79-10). Nine of 10 hurricane fatalities are drownings associated with storm surges.[44,46,120] The major rescue problem is locating persons stranded by rising waters and evacuating them to higher land. Other causes of deaths and injuries include burial beneath houses collapsed by wind or water, penetrating trauma from broken glass or wood, blunt trauma from floating objects or debris, or entrapment by mud slides that may accompany hurricane-associated floods.[22,56] Many of the most severe injuries occur to persons who are in mobile homes during the storm, or those who are injured or electrocuted during postdisaster cleanup.[23,25,122]

Most persons who seek medical care after hurricanes do not require sophisticated surgical or intensive care services and can be treated as outpatients.[104,129] The majority have lacerations caused by flying glass or other debris[56]; a few have closed frac-

Figure 79-10. Photo of New Orleans, Louisiana, following flooding caused by Hurricane Katrina in September 2005. (Courtesy Luanne Freer)

tures and other, mostly penetrating, injuries.[21,29] Longmire and colleagues[80,81] studied injuries associated with hurricanes Frederic (1979) and Elena (1985). They found a statistically significant increase in lacerations, puncture wounds, chain saw injuries, burns, gasoline aspiration, gastrointestinal complaints, insect stings, and spouse abuse in the two weeks after the hurricane. The authors concluded that minor trauma treated in the outpatient setting created an urgent demand for primary care physicians and nurses skilled in managing minor surgical emergencies. In addition, although the number of chain saw injuries was small, the time-consuming nature of treating such wounds significantly increased the demands placed on remaining emergency department personnel to treat those with other injuries. As with flood-related wounds, emergency medical care providers should be aware that wounds may contain highly contaminated material such as soil or fecal matter.[119,121] Because of this danger, primary wound closure should be done with caution.

Storm shelters are often severely crowded.[24] As with flood disasters, this crowding increases the probability of disease communication through aerosol or fecal-oral routes, particularly when sanitary facilities are insufficient.[14]

Trauma after a cyclone is not usually a major public health problem when compared with the need for water, food, clothing, sanitation, and other hygienic measures.[144] Studies demonstrate that sending fully equipped mobile hospitals and specialized surgical teams that arrive much too late at the disaster site is an ineffective response to a cyclone disaster. Nonmedical relief (e.g., epidemiologists, sanitation engineers, shelter, food, agricultural supplies) is probably more effective in reducing mortality and morbidity. On the other hand, field hospitals and emergency medical teams from outside the disaster-affected area may be useful in providing ongoing primary health care services to the community when all other health care facilities have been destroyed or severely damaged.[58] This was the case in St. Croix after Hurricane Hugo[129] and in south Florida after hurricane Andrew.[157] These situations reemphasize the importance of conducting rapid assessments of public health needs before sending relief personnel and materials to a disaster.[117,118]

Figure 79-11. Tornado striking McConnell Air Force Base, Kansas, April 26, 1991. (Courtesy U.S. Air Force.)

Tornadoes

Tornadoes are among the most violent natural atmospheric phenomena, as has been witnessed after recent devastating tornadoes in Oklahoma, Texas, and Alabama (Fig. 79-11; see Table 79-1).[96] Although approximately 700 tornadoes occur in the United States each year, only about 3% result in severe injuries requiring hospitalization.[78] Of 14,600 tornadoes studied between 1952 and 1973, only 497 caused fatalities, and 26 of these events accounted for almost half of the fatalities.[47] The Centers for Disease Control and Prevention (CDC) has reviewed the public health impact of tornadoes in great detail.[78]

Destruction caused by tornadoes results from the combined action of strong rotary winds and the partial vacuum in the center of the vortex.[95] For example, when a tornado passes over a building, the winds twist and rip at the outside. Simultaneously, abrupt pressure reduction in the tornado's eye causes explosive pressures inside the building. Walls collapse or topple outward, windows explode, and debris from this destruction can be driven as high-velocity missiles through the air. Buildings made of nonreinforced masonry, wood frame buildings, and those with large window areas will likely suffer the most extensive damage.[71] Building practices may be largely responsible for the severity of injury resulting from tornadoes.[18] In the

last 50 years, tornadoes have been responsible for more than 9000 deaths in the United States.[49] About 4% of all injuries sustained were fatal. For every person seriously injured or killed, approximately 44 others required emergency medical attention.[51]

Victims of tornado disasters show characteristic patterns of fatal and nonfatal injuries. The leading cause of death is craniocerebral trauma,[17,85] followed by crushing wounds of the chest and trunk.[5,136] Fractures are the most frequent nonfatal injury. Lacerations, penetrating trauma with retained foreign bodies, and other soft tissue injuries also frequently occur. A high percentage of wounds among tornado casualties are heavily contaminated.[19,59] In many instances, foreign materials such as glass, wood splinters, tar, dirt, grass, and manure are deeply embedded in areas of soft tissue injury.[85] Wound contamination appears to be a major factor contributing to the high rate of postoperative sepsis for tornado victims who require surgery, even under conditions in which patients receive highly skilled and prompt surgical debridement. Sepsis is common in both minor and major injuries; sepsis affects one half to two thirds of patients with minor wounds.[5] In 1956, Hight and coworkers[59] examined the postoperative course of patients after the Worcester tornado and found sepsis in 12.5% to 23.0% of orthopedic and neurosurgical patients with lacerations and three cases of gas gangrene, but no cases of tetanus.

Three studies have looked specifically at the species of bacteria that contaminate wounds sustained during tornadoes.[15,48,64] These revealed frequent infection with aerobic gram-negative bacilli, presumably derived from soil.

Volcanic Eruptions

Volcanic eruptions have claimed more than 266,000 lives in the past 400 years, with fatalities occurring in about 5% of eruptions (see also Chapter 15).[15,141] Some of the more catastrophic eruptions in history include that of Krakatoa (Indonesia, 1883), which caused 36,000 deaths; of Mt. Pelee in 1902, which caused the destruction of St. Pierre in Martinique and the deaths of 28,000 persons; of Nevado del Ruiz in Colombia, which claimed 25,000 lives; and of Mt. Pinatubo in the Philippines, with effects still ongoing because of persistent mudflows. The U.S. Geological Survey has identified about 35 volcanoes in the western United States and Alaska that are likely to erupt in the future. Most of these are in remote rural areas and are not likely to result in disaster. A few, such as Mt. Hood, Mt. Shasta, Mt. Rainier, and the volcano underlying Mammoth Lakes in California, are near population centers.[4,11] Because of increasing population density in areas of volcanic activity, volcanic hazards are of growing concern.[31,57,69,96]

Eruptions have immediate life-threatening health effects through suffocation from inhalation of massive quantities of airborne ash, scalding from blasts of superheated steam, and surges of lethal gas.[6,10] Pyroclastic flows and surges are particularly lethal.[8] These are currents of extremely hot gases and particles that flow down the slopes of a volcano at tens to hundreds of meters per second and cover hundreds of square kilometers. Because of their suddenness and speed, pyroclastic flows and surges are difficult to escape.

Mudflows, or lahars, account for at least 10% of volcano-related deaths.[161] These are flowing masses of volcanic debris mixed with water. The mud is sometimes scalding hot, and entrapped persons may sustain severe burns. A relatively minor eruption of snow-capped Nevado del Ruiz in 1985 triggered lahars from the volcano's ice cap that buried more than 22,000 persons in Colombia, South America.[141]

An indirect effect of volcanic activity is accumulation of toxic volcanic gases in deep crater lakes.[115,142] Sudden release of these gases can be catastrophic; carbon dioxide released from Lake Monoun and Lake Nyos in Cameroon in 1984 and 1986, respectively, claimed 1800 lives. Other toxic effects of these gas releases include pulmonary edema, irritant conjunctivitis, joint pain, muscle weakness, and cutaneous bullae. In the rare event of a ground-level release of toxic gases or aerosols (e.g., from a vent opening to the atmosphere from the side of the volcano), equipment for monitoring atmospheric concentrations of sulfur dioxide, hydrogen sulfide, hydrofluoric acid, carbon dioxide, and other gases should be available.[6,10]

A volcanic eruption may also generate tremendous quantities of ashfall.[93,130,142] Buildings have been reported to collapse from the weight of ash accumulating on roofs, resulting in severe trauma to the occupants.[86] Ash can also be irritating to eyes (causing corneal abrasions), mucous membranes, and the respiratory system.[84] Upper airway irritation, cough, and bronchospasm, as well as exacerbation of chronic lung disease, are common findings in symptomatic patients.[8] In extremely high concentrations, as in the path of a pyroclastic flow or near the volcanic vent during an ashfall, volcanic ash may cause severe tracheal injury, pulmonary edema, and bronchial obstruction, leading to death from acute pulmonary injury or suffocation.[86] After the eruption of Mt. St. Helens in 1980, 23 immediate deaths were reported. Postmortem examinations revealed that 18 of these resulted from asphyxia.[40] In most asphyxiated victims, the ash mixed with mucus and formed plugs, obstructing the trachea and main bronchi. Finally, delayed onset of ash-induced mucus hypersecretion or obstructive airway disease may occur.[7]

Victims who recover from volcano-generated mudflows may have severe dehydration, burns, and eye infections.[41] Reports of surgical care after the volcanic eruption in Colombia in 1985 showed that primary closure of wounds contaminated by mud and other volcanic material resulted in major complications.[116] These complications included gangrene necessitating amputation, osteomyelitis, compartment syndrome, and sepsis.

Most volcanic deaths are caused by immediate suffocation and, to a lesser extent, by burns or blunt trauma. Advanced cardiac and trauma life support capabilities, even if immediately available, would probably arrive too late to save asphyxiated victims. Persons who may develop severe respiratory distress syndrome should be admitted to an intensive care unit (ICU) for appropriate respiratory supportive measures, ranging from continuous positive airway pressure to mechanical ventilation with positive end-expiratory pressure.[86] Hospitals in the vicinity of both active and dormant volcanoes should be prepared to deal with a sudden influx of victims with severe burns and lung damage from inhalation of hot ash, as well as multiple varieties of trauma.[8,10]

Earthquakes

An earthquake of great magnitude is one of the most destructive events in nature. During the past 20 years, earthquakes have caused more than a million deaths and injuries worldwide.[52] In the United States, approximately 1600 deaths attributed to earthquakes have been recorded since colonial times, of which more than 1000 have occurred in California.[112,113] Hospitals and other health care facilities are particularly vulnerable to the

damaging effects of an earthquake. Because of loss of power and water supply, equipment (e.g., x-ray and kidney dialysis machines, ventilators, blood analyzers) and hospital facilities (e.g., ICUs and surgical theaters) cannot function normally when they are most needed.[2,100,127]

Disaster medical planners should note injury type and/or diagnostic classification among survivors to determine the medical care needed. The primary cause of death and injury from earthquakes is collapse of buildings that are not adequately designed for earthquake resistance, are built with inadequate materials, or are poorly constructed.[49,83] Factors determining the number of people killed when a building collapses include how badly trapped they are, how severely they are injured, how long they must wait for rescue, and how long they can survive without medical attention.[27,28,98]

Deaths resulting from major earthquakes can be instantaneous, rapid, or delayed.[73,149] Instantaneous death can be caused by severe crushing injuries to the head or chest, external or internal hemorrhage, or drowning from earthquake-induced tidal waves (tsunamis). Rapid death occurs within minutes or hours and can be caused by asphyxia from dust inhalation or chest compression, hypovolemic shock, or exposure (e.g., hypothermia). Delayed death occurs within days and can be caused by dehydration, hypothermia, hyperthermia, crush syndrome, or postoperative sepsis.[131,132]

As with most natural disasters, the majority of persons requiring medical assistance have minor injuries such as superficial lacerations, sprains, and bruises.[2,124,140] The next most frequent reason for seeking medical attention is simple fractures not requiring operative intervention.[136] For example, after the 1968 earthquake south of Khorasan, Iran, only 368 (3.3%) of 11,254 persons injured required inpatient care. Hospitalized patients included those with serious multiple fractures or internal injuries, hypothermia, sepsis from wound infections, or multiple organ failure requiring surgery or other intensive care services.[89]

More detailed inpatient information is available from data collected on 4832 patients admitted to hospitals after the 1988 earthquake in Armenia.[98] Consistent with findings from other major earthquakes, combination injuries constituted 39.7% of the cases. Superficial trauma, such as lacerations and contusions, was the most frequently observed injury (24.9%), followed by head injuries (22%), lower extremity injuries (19%), crush syndrome (11%), and upper extremity trauma (10%).

Infected wounds and gangrene were major problems after the Armenian earthquake.[99] Persons who have been trapped by rubble for several hours or days may also develop compartment syndromes requiring fasciotomy or amputation. These persons may have significant rhabdomyolysis and must be watched closely for signs and symptoms of crush syndrome such as hypovolemic shock, hyperkalemia, renal failure, or fatal cardiac arrhythmias.[12,103,110] After the 1988 earthquake in Armenia, more than 1000 victims trapped in collapsed buildings developed crush syndrome as a result of limb compression; 323 developed secondary acute renal failure requiring renal dialysis.[103]

Heavy dust is produced by crumbling buildings immediately after earthquakes. For trapped victims, this dust is a life-threatening hazard that may cause asphyxiation or upper airway obstruction.[60] Fulminant pulmonary edema from dust inhalation may also be a delayed cause of death.[131] Asbestos and other particulate matter in the dust are both subacute and chronic respiratory hazards for trapped victims, as well as for rescue and cleanup personnel. The degree of hazard depends on the characteristics and toxicity of the dust.[106]

Burns and smoke inhalation from fires used to be major hazards after an earthquake. For example, after the 1923 earthquake in Tokyo, more than 140,000 persons perished, principally because of fires that broke out in a city in which most buildings were constructed from highly flammable paper (shoji) and wood material. Since 1950, however, the incidence of burns worldwide has decreased considerably.[27]

To maximize saving trapped victims and increase their chances of survival, search and rescue teams must respond rapidly after a building collapses.[108] Studies of the 1980 Campania-Irpinia, Italy, earthquake[32–34] and the 1976 Tangshan, China, earthquake[166] show that the proportion of trapped people found alive declined as delay in extrication increased. In the Italian study, a survey of 3619 survivors showed that 93% of persons who were trapped and survived were extricated within the first 24 hours and that 95% of the deaths recorded occurred while the victims were still trapped in rubble.[32] Estimates of the survivability of victims buried under collapsed earthen buildings in Turkey and China indicate that within 2 to 6 hours, fewer than 50% of those buried are still alive.

Although it cannot be determined whether a trapped person dies immediately or survives for some time under the debris, more people would be saved if they were extricated sooner. Safar,[132] studying the 1980 earthquake in Italy, concluded that 25% to 50% of victims who were injured and died slowly could have been saved if lifesaving first aid had been rendered immediately. As suggested by these data, if any significant reduction in earthquake mortality is to be achieved, appropriate search and rescue action must be provided within the first 2 days after the impact.[2]

Paralleling the speed required for effective search and extrication is the speed with which emergency medical services must be provided.[134] The greatest demand occurs within the first 24 hours.[150] In fact, injured people usually seek medical attention at emergency departments only during the first 3 to 5 days, after which time hospital case-mix patterns return almost to normal.[148] The critical importance of early emergency care was seen in the number of admissions to a field hospital after the 1976 earthquake in Guatemala.[35,36] From day 6 on, admissions fell dramatically despite intensive efforts to find injured people in remote rural areas of the impact zone, indicating that specialized field hospitals that arrive a week or more after an earthquake are generally too late to help during the emergency phase. After the Armenian earthquake, only 22 (2.4%) of the 902 patients requiring hospitalization at a large hospital were admitted 7 or more days after the impact.[98]

With most earthquakes, trauma caused by the collapse of buildings is the cause of most deaths and injuries (Fig. 79-12).[2] However, a surprisingly large number of victims require acute care for nonsurgical problems, such as acute myocardial infarction, exacerbation of chronic diseases (e.g., diabetes, hypertension), anxiety and other mental health problems, respiratory disease from exposure to dust and asbestos fibers from rubble, and near-drowning because of flooding from broken dams.[2] An example of the adverse effects of an earthquake on medical conditions was observed after a magnitude 6.7 earthquake in Athens, Greece. A 50% increase in deaths from myocardial infarction was documented during the first 3 days after the earthquake, peaking on the third day.[67,155] Finally, an earth-

Figure 79-12. Northridge earthquake, California, January 17, 1994. Many roads, including bridges and elevated highways, were damaged by the 6.7 magnitude earthquake. Approximately 114,000 residential and commercial structures were damaged and 72 deaths were attributed to the earthquake. Damage costs were estimated at $25 billion. (Courtesy U.S. Federal Emergency Management Agency [FEMA].)

quake may precipitate a major technologic disaster by damaging or destroying nuclear power stations, hospitals with dangerous biologic products, hydrocarbon storage areas, and hazardous chemical plants. As with most natural disasters, the risk of secondary epidemics is minimal, and only mass vaccination campaigns based on results of epidemiologic surveillance are appropriate after earthquakes.[154]

In the aftermath of a tsunami, there are acute traumatic injuries, some of which may be fatal, followed by infectious diseases. This may include sequelae of seawater aspiration, including pneumonia, and virulent soft tissue infections, such as fasciitis.[72] In the tsunami that struck the Asian subcontinent and Africa on December 26, 2004, patients were stricken with pneumonia and lung abscess attributed to *Burkholderia pseudomallei*, which led to melioidosis after the disaster. Other organisms identified in the blood, sputum, or pus of patients included *Pseudomonas aeruginosa*, *Stenotrophomonas maltophilia*, *Acinetobacter baumanii*, *Escherichia coli*, *Klebsiella*, *Enterobacter*, *Neisseria*, *Citrobacter*, *Corynebacteria*, and *Streptococcus viridans*. In a rare case, brain abscess from hematogenous spread of infectious organisms may follow polymicrobial pulmonary infection from seawater aspiration.[66]

CRITICAL PUBLIC HEALTH INTERVENTIONS AFTER DISASTERS

Critical public health interventions after disasters focus on the following areas:

Environmental Health: Water, Sanitation, Hygiene, and Vector Management

General Issues

Overcrowding, inadequate and contaminated water supplies, and poor hygiene and sanitation are well-known factors that increase the incidence of diarrhea, respiratory infections, and other communicable diseases. A good system of water supply and excreta disposal must be put into place quickly.[79] The objectives of an adequate system should meet criteria in (1) providing the minimum quantity of clean drinking water, (2) reducing the transmission of fecal-oral diseases and exposure to vectors, and (3) establishing conditions that allow people to perform daily tasks such as using the toilet, bathing, comfort, and security.

No amount of curative health measures can offset the detrimental effects of poor environmental health planning.[79] Such planning includes consequence management and forecasting of how public infrastructure and public works will influence and put at risk access and provision to basic human needs like shelter, food, potable water, and sanitary facilities. A well established plan should be laid out before the disaster occurs, in collaboration with various sectors of the community (health, public works, policy officials, public safety), addressing those parts of the public infrastructure that are most vulnerable or that, if corrupted, will most put at risk the society's health.

Important postdisaster environmental interventions include access to adequate sources of potable water and the collection, disposal, and treatment of excreta and other liquid and solid wastes.[163] This is achieved through installation of an appropriate number of suitably located excreta disposal facilities, such as toilets, latrines, or defecation fields; solid waste pickup points; water distribution points; and availability of bathing and washing facilities and of soap, together with effective health education. Control of disease vectors, such as mosquitoes, flies, rats, and fleas, is an important part of an environmental health approach to protecting community members from disease.[90] This is achieved through measures that include attacking the source of the disease and disrupting its transmission.[94] For example, in the case of malaria, the mosquito population can be reduced by distributing mosquito nets impregnated with insecticide. This provides a shield effect even for those who may be at risk and do not use nets.[97] Traditionally, chemical insecticides have been used to suppress vector populations by eliminating breeding sites.[30]

Water Disposal

Adequate quantities of potable, relatively clean water are preferable to small amounts of high-quality water. Minimum standards for potable water of intermediate quality are indicated through specific, measurable criteria, such as (a) no more than 10 fecal coliforms per 100 mL, (b) a sanitary survey that indicates low risk of fecal contamination, (c) water that is treated with a residual disinfectant (e.g., 0.2 to 0.5 mg per liter of chlorine), and (d) turbidity less than 5 nephelometric turbidity units (NTU).[163] Each person would ideally receive a minimum of 15 to 20 L of clean water per day for personal domestic needs such as washing clothes, washing cooking and eating utensils, and bathing.[145] Unfortunately, it is frequently difficult to provide even this minimum quantity of water to disaster-affected populations.[30]

Excreta Disposal

During the early acute phase, latrine construction begins, but initial sanitation measures may be nothing more than simply designating an area for defecation, hopefully segregated from the community's source of potable water. Construction of one latrine for every 20 persons is recommended but is rarely achieved in camp settings.[145]

Shelter

Surveys of settlements and towns around Bam, Iran, after the December 2003 earthquake indicated that 80% to 90% of the 200,000 displaced persons were living with relatives and friends; 5% to 10% were living in parks, city squares, and vacant lots; and the remainder were living in schools and other public buildings.[94] Regarding temporary living space allocations, 3.5 square meters is the absolute minimum floor space per person in emergency shelters.[145,146] The first priority in areas where large numbers of people are living in damaged housing is to diminish as much as possible the penetration of wind and rain into the structure.[90] In these situations, plastic sheeting for roof and window repairs, along with the required materials for attaching them to the damaged structures, is often provided by relief organizations.[90] Most people who lose their homes will initially be able to take shelter with friends and relatives.[94,146] When housing losses reach more than approximately 25%, there is a need to find other forms of shelter.[94] The decision to provide shelter can have significant long-term consequences, especially in poor communities.[126,134] For example, simple shelters provided on an emergency basis may unintentionally evolve into permanent shantytowns or squatter settlements and end up attracting many more homeless people to the site.

Mass Casualty Care

A disaster may create casualties in excess of the capacity of the local health care system. The approach to patient evaluation and treatment is quite different under disaster situations that result in large numbers of casualties. While some principles of nondisaster medical care are unchanged in a mass casualty incident, others must be altered to achieve the best overall result. The health care system must adapt to this situation with four measures: simplifying care (austerity); rationing care (adopting a triage ethic); calling for outside help; and in circumstances of catastrophe, instituting mass care measures typical of battlefield medicine. Many compromises in work methods eliminate attention to details that would be required in less urgent situations. These compromises may include collection of only a brief patient history, such as data on allergies, medicines, last meal eaten, and events or the environment. Secondly, only basic first aid measures and "damage control surgery" (nondefinitive surgical intervention) would be performed on the casualty. Another constraint unique to the disaster situation is that local hospitals might undergo rapid conversion to excess capacity. Physicians and nurses often perform procedures beyond the scope of their usual practices. Professional functions and roles are widely shared among physicians, nurses, and paramedics. These adaptations allow available resources to serve more victims.[123]

Austerity

To be effective, disaster medical care must be confined to basic measures, adequate to preserve life and function. Examinations, techniques, appliances, and drugs that are not essential to patient survival or preservation of function are luxuries. It may be necessary to perform fracture reductions and other minor surgical procedures with oral narcotic analgesia only. Orthopedic devices are often improvised. Use of outdated drugs is sometimes acceptable, rather than using no drugs. The level of austerity is determined by the health care personnel, supplies, and equipment available at the disaster treatment site.

COMMUNICABLE DISEASE CONTROL AND EPIDEMIC MANAGEMENT

Epidemics

Natural disasters, such as the recent tsunami disaster in South Asia, are often followed by rampant rumors of epidemics (e.g., cholera and plague) or unusual conditions, such as increased snakebites and dog bites. Such unsubstantiated reports gain great public credibility when printed as facts in newspapers or reported on television or radio.[76] For example, after disasters in developing countries, any disruption of the water supply or sewage treatment facilities has usually been accompanied by rumors of outbreaks of cholera or typhoid.[156] Such rumors may well have reflected psychological fears and anxieties about a disastrous event, rather than the true perception of an imminent problem. However, information on disease incidence in most developing countries is poor, and some outbreaks may have been missed entirely by public health authorities. Although natural disasters do not usually result in outbreaks of infectious disease, under certain circumstances disasters may increase disease transmission. The risk of epidemic outbreaks of communicable diseases is proportional to population density and displacement. These conditions increase pressure on water and food supplies and the risk of contamination (e.g., as occurs in refugee camps), disruption of preexisting sanitation services such as piped water and sewage, and failure to maintain or restore normal public health programs in the immediate post-disaster period. The most frequently observed increases in communicable disease are caused by fecal contamination of water and by respiratory spread (e.g., influenza in evacuation camps).[74] In the longer term, an increase in vector-borne diseases occurs in some areas because of disruption of vector control efforts, particularly after heavy rains and floods. Residual insecticides may be washed away from buildings, and the number of mosquito breeding sites may increase. Moreover, displacement of wild or domesticated animals near human settlements brings the additional risk of zoonotic infection.

Disposition of Dead Bodies

The public and government authorities are usually greatly concerned about the danger of disease transmission from decaying corpses (Fig. 79-13). Responsible health authorities should recognize, however, that health hazards, such as epidemics associated with unburied bodies, are minimal, particularly if death resulted from trauma. It is far more likely that survivors may become the source of disease outbreaks. Although the risks of disease transmission for rescue workers who handle dead bodies are higher than for the survivors of a disaster, those risks can be limited through a set of simple measures. Appropriate precautions include training military personnel and others who might have to provide assistance after a disaster, vaccinating those persons against hepatitis B and tuberculosis, using body bags and disposable gloves, washing hands after handling cadavers, and disinfecting stretchers and vehicles that have been used to transport bodies.[128]

Unjustified worries about the infectiousness of bodies can lead to rapid, unplanned disposal of the dead, sometimes before proper identification of the victim has been made, as well as to taking needless "precautions" such as mass cremation, burying the deceased in common graves, and adding chlorinated lime as

A B

Figure 79-13. **A,** Coffins lining the street in the city of Leninakan (now Gyumri) after the 1988 earthquake in Armenia. **B,** Three horses were killed as a result of falling debris during the 1906 San Francisco earthquake. (**A,** courtesy Eric K. Noji.; **B,** courtesy Eric Swenson, U.S. Geological Survey.)

a "disinfectant." Despite the negligible health risk, dead bodies represent a delicate social problem. Disposal of bodies should respect local custom and practice when possible. When there are large numbers of victims, burial is likely to be the most appropriate method of disposal. There is little evidence that proper burial of bodies poses a threat to groundwater that serves as a source of drinking water.[138]

Immunization

Mass immunization during situations of natural disasters is usually counterproductive and diverts limited human resources and materials from other more effective and urgent measures. Immunization campaigns can give a false sense of security, leading to neglect of basic measures of hygiene and sanitation, which are more important during the emergency. Mass vaccination would be justified only when the recommended sanitary measures do not have an effect and if there is evidence of the progressive increase in the number of cases with the risk of an epidemic. A vaccine with the following characteristics could be considered useful in this situation:

- A vaccine of proven efficacy, high safety, and low reactogenicity
- A vaccine that is easy to apply (single dose)
- A vaccine that confers rapid and long-lasting protection for people of all ages
- Avoilability of sufficient quantities of vaccine to guarantee the supply for the entire population at risk
- Low-cost vaccines

For example, immunization of children against measles is one of the most important (and cost-effective) preventive measures in emergency-affected populations, particularly those housed in camps. Immunization of refugee children against measles in Thailand in 1979 clearly saved many lives. Although measles was an early problem in Somalia, immunization of the refugee population was effective in preventing outbreaks after 1981.[159] Because infants as young as 6 months of age may contract measles in refugee camp outbreaks and are at greater risk of dying as a result of impaired nutrition, it is recommended that measles immunization programs, along with vitamin A supplements, in emergency settings target all children from the ages of 6 months through 5 years (some would recommend as old as 12 to 14 years of age). Ideally, one should strive for measles immunization coverage in refugee camp settings of better than 80% of persons present.[145,146]

Nutrition

Food shortages in the immediate aftermath of a disaster may arise in two ways. Food stock destruction within the disaster area may reduce the absolute amount of food available, or disruption of distribution systems may curtail access to food, even if there is no absolute shortage. Generalized food shortages severe enough to cause nutritional problems usually do not occur after natural disasters. Flooding, tsunamis, and sea surges can damage household food stocks and crops, disrupt distribution, and cause major local shortages. Food distribution, at least in the short term, is often a major and urgent need, but large-scale importation/donation of food is not usually necessary.[91] In extended droughts, such as those occurring in Africa, or in complex disasters, the homeless and refugees may completely depend upon outside sources for food supplies for varying periods of time.[152] Depending on the nutritional condition of these populations, especially of more vulnerable groups such as pregnant or lactating women, children, and the elderly, it may be necessary to institute emergency feeding programs.[61] The highest nutritional priority in the postdisaster setting is timely and adequate provision of food rations containing at least 2100 calories per day and that include sufficient protein, fat, and micronutrients.[145]

DISASTER RESPONSE ORGANIZATIONS

Department of Homeland Security

The Department of Homeland Security (DHS), a cabinet-level department formed following the terrorist attacks of September 11, 2001, is the federal government's lead organization for emergency management activities in the United States. The

Federal Emergency Management Agency (FEMA) has been incorporated intact into DHS and retains its name. DHS has a coordinating responsibility for the entire spectrum of natural, technological, and terrorist disasters. DHS assists state and local organizations to mitigate, prepare for, respond to, and recover from emergencies and is a major source of funding for these endeavors. Starting in 1992, the Federal Response Plan (FRP) was used to coordinate federal assistance into 12 categories known as Emergency Support Functions (ESFs). Examples of ESFs include food, health and medical services, transportation, and communications. In 2003, the President of the United States directed that the DHS develop a National Incident Management System and a National Response Plan (NRP). Existing federal plans, including the FRP, are being incorporated into the NRP.

National Disaster Medical System

The National Disaster Medical System (NDMS) is a federally coordinated initiative designed to augment the emergency medical response capability of the United States in the event of a catastrophic disaster. This system is a cooperative program of four agencies in the federal government: (1) Department of Defense (DOD), (2) Department of Health and Human Services (HHS), (3) DHS, and (4) Department of Veterans Affairs (VA). DHS has the lead for the NDMS via FEMA. The NDMS provides an interstate medical mutual aid system linking the federal government, state and local agencies, and private sector institutions to address the medical needs of victims from catastrophic disasters. Its medical response element includes dozens of volunteer civilian disaster medical assistance teams (DMATs) that supplement the local medical infrastructure.

In a disaster, the NDMS is activated when state resources are overwhelmed and the governor makes a request for federal assistance. The DMATs must meet specific NDMS standards. Throughout the NDMS, emergency physicians (EPs) are taking key roles in defining training standards, deployment of clinical services, and administration of field operations, and in developing the concept of a civilian-federal disaster response capacity during national emergencies.

Department of Veterans Affairs (VA)

The VA has not traditionally been regarded as a disaster response entity. However, one of the four mandated missions of the VA is emergency management. A unique feature of the VA is that its facilities and personnel are situated nationwide. These are used to support federal health and medical assistance to state and local governments during disasters. The VA has highly trained specialty personnel who can support disaster medical activities. In addition to the vast pool of human resources, the VA provides vast amounts of the pharmaceuticals and expendable supplies for on-site disaster support. Support is coordinated through DHS and HHS, as the lead federal agency for health and medical response.

Urban Search and Rescue

When a building collapses because of an earthquake, terrorist bombing, structural failure, or other reason, various challenges confront rescue and medical personnel. Some victims require field amputations to facilitate extrication, and use of urban search and rescue (USAR) teams and effective emergency medical care are essential for successful lifesaving efforts. This national system of multidisciplinary task forces is designed for rapid deployment to the sites of collapsed structures. The medical team's responsibilities include caring for task force members, victims recovered by search and rescue activities, and the search team's canines. There are now WMD-USAR teams (weapons of mass destruction–urban search and rescue) trained by FEMA to respond to NBC (nuclear/biological/chemical) terrorist attacks.

Centers for Disease Control and Prevention

The CDC, based in Atlanta, is a U.S. federal agency under HHS. Its major responsibilities include preparing for and responding to public health emergencies (e.g., disasters) and conducting investigations into the health effects and medical consequences of these events. Major aims of CDC researchers are to assess the risks of death and injury and to develop strategies for preventing or mitigating the impact of future disasters. Other responsibilities of CDC staff in the area of emergency preparedness and response include (1) rapid assessment of the health and medical needs of disaster victims in the immediate post disaster period; (2) development and maintenance of national systems for acute environmental hazard surveillance; and (3) provision of epidemiologic, sanitary, laboratory, and other relevant scientific support services to agencies involved in disaster planning and response.

Strategic National Stockpile (SNS)

To ensure availability, procurement, and delivery of medical supplies, devices, and equipment that might be needed to respond to terrorist and other disaster-caused illness or injury, the CDC maintains the Strategic National Stockpile (SNS). The mission of the SNS program is to ensure availability of lifesaving pharmaceuticals, vaccines, antidotes, and other medical supplies and equipment necessary to counter the effects of nerve agents, biologic pathogens, and chemical agents and to augment depleted state and local resources for responding to terrorist attacks and other emergencies (Box 79-1). The SNS program stands ready for immediate deployment to any U.S. location in the event of a terrorist attack using a biologic agent, toxin, or chemical agent directed against a civilian population. Both 12-Hour Push Packages and Vendor Managed Inventory (VMI) are stored in strategic locations around the United States to ensure rapid delivery anywhere in the country.

Box 79-1. Contents of the Strategic National Stockpile (SNS) (also known as the National Pharmaceutical Stockpile [NPS])

- Pharmaceuticals
- Antibiotics
- Mark I kits, diazepam, atropine, pralidoxime
- Intravenous supplies
- Catheters, syringes, fluids, heparin-locks, administration sets
- Airway supplies
- Ventilators, Ambu-bags, endotracheal tubes, laryngoscopes, suction devices, oxygen masks, nasogastric tubes
- Other emergency medications for hypotension, anaphylaxis, sedation, pain management
- Bandages and dressings
- Vaccines

Metropolitan Medical Response Systems

Metropolitan Medical Strike Teams (MMSTs) are highly trained, readily deployable, and fully equipped groups of medical, fire, and rescue professionals. As a component of the larger Metropolitan Medical Response System, they support other local personnel in treating victims of a chemical, biologic, or nuclear attack. At least 25 such teams are currently planned throughout the United States. Although composed of local personnel, MMSTs are under the direction of HHS. Their goal is to enhance local planning and response capability. However, hospital and community planners must still create an independent response, because MMSTs require 90 minutes or longer to deploy. MMSTs are equipped with chemical agent monitoring devices, protective equipment, and pharmaceutical supplies.

Department of Defense

On April 17, 2002, Secretary of Defense Donald Rumsfeld announced formation of the Northern Command, or NorthCom, to assume responsibility over all military forces that operate within the United States in response to external threats and in support of civil authorities. Furthermore, Secretary Rumsfeld stated that NorthCom will "help the department better deal with natural disasters, attacks on U.S. soil, or other civil difficulties. It will provide for a more coordinated military support to civil authorities such as FBI, FEMA, and state and local governments."

MYTHS AND REALITIES OF NATURAL DISASTERS

Many mistaken assumptions are associated with the impact of disasters on public health. Disaster planners and managers should be familiar with the following myths and realities[151]:

Myth: Volunteers with any kind of medical background are needed.
Reality: The local population almost always covers immediate lifesaving needs. Only medical personnel with skills that are not available in the affected community may be needed.

Myth: Any kind of assistance is needed, and it is needed immediately!
Reality: A hasty response that is not based on an impartial evaluation only contributes to the chaos. It is better to wait until genuine needs have been assessed. In fact, most needs are met by the victims themselves and their local government and agencies, not by external relief agencies.[124]

Myth: Epidemics and plagues are inevitable after every disaster.
Reality: Epidemics do not spontaneously occur after a disaster, and dead bodies will not lead to catastrophic outbreaks of exotic diseases. The key to preventing disease is to improve sanitary conditions and educate the public.[38]

Myth: Disasters bring out the worst in human behavior (e.g., looting, rioting).
Reality: Although isolated cases of antisocial behavior exist, most people respond spontaneously and generously.[16]

Myth: The affected victims are too shocked and helpless to take responsibility for their own survival.
Reality: On the contrary, many find new strength during an emergency, as evidenced by the thousands of volunteers who spontaneously united in search of victims after the December 2004 tsunami catastrophe.

Myth: Disasters are random killers.
Reality: Disasters strike hardest at the most vulnerable groups, such as the poor, especially women, children, and the elderly.

Myth: Locating disaster victims in temporary settlements is the best alternative.
Reality: It should be the last alternative. Many agencies use funds normally spent for tents to purchase building materials, tools, and other construction-related support in the affected community.

SUMMARY

This chapter discusses health effects of disasters and outlines the requirements for effective emergency medical and public health response to these events.[70] Sound epidemiologic knowledge of the causes of death and of the types of injuries is clearly essential when determining what relief supplies, equipment, and personnel are needed to respond effectively in emergency situations.[88] The overall objective of disaster management is to assess the needs of disaster-affected populations, to match resources to needs efficiently, to prevent further adverse health effects, to evaluate relief program effectiveness, and to plan for future disasters.[13,68]

The references for this chapter can be found on the accompanying DVD-ROM.

Natural and Human-Made Hazards

80

Disaster Risk Management Issues

Sheila B. Reed

The term *hazard* is usually applied to rare or extreme events in the natural or human-made environment. Hazards can adversely affect human life or property to the extent of causing a disaster or major disruptive situation. Natural hazards are caused by biologic, geologic, seismic, hydrologic, or meteorologic processes in the natural environment and include drought, flood, earthquake, volcanic eruption, and severe storms. When natural hazards affect vulnerable human settlements, structures, and economic assets, they can be disastrous, disrupting the normal functioning of a society and necessitating extraordinary emergency interventions to save lives and the environment.

Human-made hazards are derived from human interactions with the environment, human relationships and attitudes, and the use of technology. For example, transportation accidents, petrochemical explosions, mine fires, building collapses, oil spills, hazardous waste leaks, and nuclear power plant failures are disasters in which the principal and direct causes are human actions. Many hazards have both natural and human components. Desertification results from arid conditions, erosion, and overgrazing; landslides may occur from poorly planned construction on unstable hillsides, and flooding may be caused by dam failures.

The distinction between many natural causes of hazards and contributions of humans to disastrous situations is becoming increasingly blurred. As populations grow and expand, pressure on land resources may force settlement in vulnerable areas, where hazards such as volcanic eruptions, earthquakes, or floods can become major disasters. Pest infestations may lead to famine in food-deficient areas, incidences of disease might become an epidemic because of overcrowding, and drought may become famine where food shortages result from combinations of lack of rainfall, displacement of people and lack of access to food supplies. The recent focus on global warming emanates from studies of the effects of climatic conditions and environmental pollution. Variables in these studies form such complex interactions that even computerized models have difficulty predicting the outcomes. Hazards with a combination of causes result in complex disasters and often in complex emergencies. Whatever their causes, disasters have serious political, economic, social, and environmental implications. In less developed areas, disasters can severely set back or reverse development efforts.

DISASTER RISK MANAGEMENT

This chapter focuses on hazards with a significant geophysical component, from a disaster risk management perspective. Disaster risk management is a sustainable hazard mitigation approach that is meshed with development agendas globally and nationally. It includes reducing the probability of disasters by using methods that are financially, environmentally, and culturally sensitive and using mitigation methods that are agreed upon through public consultation. A critical feature is training for communities to take more direct responsibility for disaster reduction. Another key component is improving on unsustainable predisaster conditions through well-planned disaster recovery programs.

The practice of disaster risk management encompasses all aspects of preventing, planning for, responding to and recovering from disasters, including predisaster and postdisaster activities. It refers to the management of both the risks and the consequences of disasters. Components of disaster management include vulnerability and risk assessment, disaster mitigation and preparedness, and disaster impact assessment. Selection of management options depends on the type of hazard and its characteristics. Box 80-1 lists the elements usually found in a disaster preparedness plan for sudden-onset hazards, such as earthquakes, tsunamis, volcanic eruptions, tropical cyclones, and floods. Preparedness measures for slow-onset disasters, such as drought, include early warning systems that alert authorities to precursory conditions and allow preparations to avert famine and displacement.

Slow-Onset versus Rapid-Onset Hazards

The distinction between slow- and rapid-onset hazards is useful in that the methods to deal with them often differ. However, disasters are a product of hazards combined with human vulnerability. The socioeconomic forces that make people vulnerable to disaster are likely to result from long-term trends. Rapid-onset hazards often occur with violent intensity and have profound effects on the surrounding environment, resulting in measurable numbers of casualties and damage. Slow-onset climatic changes brought on by deforestation, drought, desertification, or environmental pollution change the suitability of

Box 80-1. Essentials to Prevent or Prepare for An Emergency

1. Identification and mapping of the hazard zones; registration of valuable and movable property
2. Identification of safe refuge zones to which the population will be evacuated in case of danger
3. Identification and maintenance of evacuation routes
4. Identification of assembly points for persons awaiting transport for evacuation
5. Means of transport and traffic control
6. Shelter and accommodation in the refuge zone
7. Inventory of personnel and equipment for search and rescue
8. Hospital and medical services for treatment of injured persons
9. Security in evacuated areas
10. Formulation and communication of public warnings and procedures for communication in emergencies
11. Longer-term recovery plans (e.g., social services for trauma victims)
12. Provisions for revising and updating the plan

different parts of the world for agriculture and also affect flora and fauna. The effects of slow-onset disasters are often insidious. Their impact can be measured only through environmental studies and in terms of reduction in quality of life and productivity for the affected population. The study of disaster management, which formerly focused on natural hazards, now encompasses a range of slow-onset and rapid-onset disasters and their natural and human causes.

Conservatively, an estimated 1.5 to 2 million people have been killed in rapid-onset disasters since 1946, for an average annual death toll of 35,000 to 50,000. The primary killers are earthquakes, tropical cyclones, and floods. Most deaths are concentrated in a relatively small number of communities, predominantly in poorer nations of Africa, Asia, Latin America, and Oceania. In comparison, North America, Europe, Japan, and Australia have average annual death tolls that rarely exceed a few hundred persons.

Although no comprehensive data are available for economic losses from rapid-onset hazards, a few examples illustrate the scale of the problem. Annual worldwide losses from tropical cyclones are estimated at between $6 and $7 billion. For landslides, the comparable figure exceeds $1 billion. These figures only hint at the human impact of destruction. The eruption of Colombia's Nevado del Ruiz volcano in 1985 killed approximately 22,000 people and left 10,000 more homeless. An earthquake in Bam, Iran, in December of 2003 claimed at least 30,000 lives and destroyed 80% of the city. The 2005 Atlantic hurricane season caused record damages of $100 billion. Hurricane Katrina alone, in August of 2005, killed 1417 people in three U.S. states, displaced 1.5 million, and caused $75 billion in damages.

The relative human, economic, and social impacts of rapid-onset disasters are usually greatest in small, poor nations. The 1985 earthquake in Mexico City caused economic losses equivalent to about 3.5% of Mexico's gross national product (GNP). Hurricane Allen in 1980 caused losses in St. Lucia equivalent to 89% of this island nation's GNP and destroyed 90% of its banana crop, which normally accounts for 80% of the country's agricultural output. One of the strongest storms in recent history, Hurricane Mitch in 1998 devastated the economies and infrastructure of Honduras and Nicaragua.

Economic losses from rapid-onset hazards are increasing at a fast pace. In the United States, damage to buildings from earthquakes, tropical cyclones, and floods was estimated to increase from approximately $6 billion in 1978 to more than $11 billion in 2000 without additional loss reduction measures. A major earthquake in Tokyo would probably kill more than 30,000 people, cause the collapse of 60,000 houses, and set fire to more than 400,000 homes.

Slow-onset disasters take an even greater toll, but precise figures are difficult to find. Drought currently affects more people than any other disaster; worldwide, droughts have been estimated to have affected more than 18 million people each year during the 1960s, more than 24 million people during the 1970s, and 101 million from 1980 to 1989. During the early 1980s, drought affected up to 30 million people in Africa alone. Droughts have led to famines, resulting in large numbers of deaths and displacements. Increasing desertification in arid areas may be contributing to droughts. Desertification, or decline in biologic productivity, extends to 70% of total productive arid lands or 3.6 billion acres worldwide and may adversely affect the quality of life for 10% of the world population, including urban dwellers.

Possible global warming is predicted in the next century from increased atmospheric carbon dioxide caused by burning of fossil fuels, deforestation, and generation of methane. If this occurs, sea levels will rise and coastal cities worldwide will be inundated. A rise of 1 m ($3^1/_3$ ft) in sea level could flood 15% of arable land in Egypt's Nile Delta and completely submerge the tiny islands of the Maldives, the latter inhabited by 200,000. Hundreds of millions of people will also be affected if increased ultraviolet radiation is delivered to the earth's surface as a result of stratospheric ozone depletion caused by continued release of chlorofluorocarbons.

Although global warming and ozone depletion are threats to the future, other forms of environmental pollution, such as water and air pollution, affect life today. Massive oil spills make headlines, and adverse health effects are seen from contamination and smog. Deforestation, particularly in the tropical rainforests, is highly significant. In addition to its contribution to possible global warming, it increases vulnerability to droughts, landslides, and floods.

Assessing Vulnerability and Risk

Not all hazards become disasters. Whether or not a disaster occurs depends on the magnitude, intensity, and duration of the event and the vulnerability of the community. For example, a severe earthquake is not a disaster unless it significantly disrupts a community by creating large numbers of casualties and substantial destruction. Effective disaster risk management requires information about the magnitude of the risk faced and how much importance society places on the reduction of that risk. Risks are often quantified in aggregated ways (e.g., a probability of 1 in 23,000 per year of dying in an earthquake in Iran). The importance placed on risk of a hazard is likely to be influenced by the nature of the risks faced on a daily basis. In Pakistan, where communities are regularly affected by floods, earthquakes, and landslides, people use their meager resources to protect against what they perceive to be greater risks, such

as disease and irrigation failure. In California, where risk of disease is low, communities choose to initiate programs against natural disasters.

Vulnerability is often measured as the susceptibility of buildings, infrastructure, economy, and natural resources to damage from hazards. Many aspects of vulnerability, however, cannot be described in monetary terms and should not be overlooked, such as personal loss of family, home, and income and related human suffering and psychosocial problems. Although communities in developed nations may be as prone to hazards as communities in poor nations, the wealthier communities are often less vulnerable to damage. Although southern California and Managua, Nicaragua, are both prone to earthquakes, California is less vulnerable to damage because of strictly enforced building codes, zoning regulations, earthquake preparedness training, and sophisticated communications systems. In 1971, the San Fernando earthquake in California measured 6.4 on the Richter scale but caused minor damage and 58 deaths, whereas an earthquake of similar magnitude struck Managua 2 years later and reduced the center of the city to rubble, killing approximately 6000 people. Similarly, in wealthy countries, drought and resulting loss of food production and groundwater are managed by use of food surpluses and treated water, but drought in poor nations often leads to deaths from famine, and to sickness and death from contaminated water supplies.

Disaster Mitigation Strategies

Mitigation involves not only saving lives and reducing injury and property losses, but also reducing the adverse consequences of hazards to economic activities and social institutions. Where resources are limited, they should be directed toward protecting the most vulnerable elements. Vulnerability also implies a lack of resources for rapid recovery.

For most risks associated with natural geophysical hazards, such as volcanic eruptions, tsunamis, and tropical cyclones, little or no opportunity is available to reduce the hazard itself. In these cases, the emphasis must be placed on reducing the vulnerability of the elements at risk. However, for technologic and human-made hazards or slow-onset hazards, such as environmental pollution and desertification, reducing the hazard is likely to be the most effective mitigation strategy.

Actions by planning authorities to reduce vulnerability can be active, where desired actions are promoted through incentives, or passive, where undesired actions are prevented by use of controls and penalties. Discussion of mitigation options follows.

Engineering and Construction. Engineering measures range from large-scale engineering works to strengthening individual buildings and implementing small-scale community-based projects to incorporate better protection into traditional structures, such as buildings, roads, and embankments.

Physical Planning Measures. Careful location of new facilities, particularly community facilities such as schools, hospitals, and infrastructure, plays an important role in reducing settlement vulnerability. In urban areas, deconcentration of elements especially at risk is an important principle. Specific procedures include hazard mapping and development of a master plan containing land use control guidelines. Hazard occurrence probabilities can be extrapolated from historical data and used to create hazard maps to show regional variation. Hazard mapping can be detailed by an inventory of people or things that are exposed or vulnerable to the hazard. In France, the Zones Exposed to Risks of Movements of the Soil and Subsoil (ZERMOS) plan produces landslide hazard maps at scales of 1:25,000 or larger that are used as tools for mitigation planning. The maps portray degrees of risk of various types of landslides, including activity, rate, and potential consequences.

Economic Measures. The linkages among different sectors of the economy may be more severely disrupted than the physical infrastructure. Diversifying and strengthening the economy are important ways to reduce risk. Within a strong economy, governments can use economic incentives to encourage individuals or institutions to take disaster mitigation actions.

Management and Institutional Measures. Building disaster protection takes time and requires support from programs of education, training, and institution building to provide the professional knowledge and competence required. Development of forecasting and warning systems are important protective measures.

Societal Measures. Mitigation planning should aim to develop a "safety culture" in which all members of society are aware of the hazards they face, know how to protect themselves, and support the protection efforts of others and communities as a whole. Specifically, these include conducting community education programs and planning and practicing evacuation procedures.

Disaster Assessment and Postdisaster Needs

Assessments regularly conducted during the recovery process help to identify needs that lead to appropriate types of assistance. In the cases of slow-onset hazard types such as desertification, deforestation, and environmental pollution, a distinct postdisaster period usually does not exist; thus ongoing impact assessment is vital. The initial response to a rapid-onset hazardous event includes the following steps by local authorities:

1. Evacuation
2. Emergency shelter
3. Search and rescue
4. Medical assistance
5. Provision of short-term food and water
6. Water disinfection and purification
7. Epidemiologic surveillance
8. Provision of temporary lodging
9. Reopening of roads
10. Reestablishment of communications networks and contact with remote areas
11. Brush and debris clearance
12. Disaster assessment
13. Provision of inputs for recovery and rehabilitation

Nature of Hazards

Many types of natural hazards and many hazards partially rooted in natural systems exist. Many of these occur infrequently or affect only small populations. One example is the eruption of toxic gases from several volcanic lakes in Cameroon that killed 2000 people in 1984 and 1986. Other rare events, such as meteor impacts, may occur only once every few centuries. Other widespread but minor phenomena that damage

property but do not generally cause loss of life include land subsidence and sinkholes. Some hazards, such as snowstorms, often occur in areas that are prepared to deal with them so that they rarely become disasters.

This chapter discusses twelve hazards that affect large populations and that can be categorized as follows:

- Geologic hazards—earthquakes, tsunamis, volcanic eruptions, landslides
- Climatic hazards—tropical cyclones, tornadoes, floods, drought, winter storms
- Environmental hazards—environmental pollution, deforestation, desertification

To plan appropriate responses to implement emergency medical care and other measures to save or restore physical and mental health of affected populations, governments and communities first need to understand the causal phenomena, characteristics, and predictability of the hazards and the factors that contribute to vulnerability. Examination of the hazard's effects on humans, property, and the environment can promote measures to prevent or lessen casualties and destruction.

EARTHQUAKES

Earthquakes are among the most destructive and feared of natural hazards. They may occur at any time of year, day or night, with sudden impact and little warning. They can destroy buildings in seconds, killing or injuring the inhabitants. Earthquakes not only destroy entire cities but may destabilize the government, economy, and social structure of a country.

Causal Phenomena

The earth's crust is a rock layer of varying thickness from a depth of about 10 km (6.2 miles) under the oceans to 65 km (40.4 miles) under the continents. The theory of plate tectonics holds that seven major and about six minor crustal plates, varying in size from a few hundred to many thousands of kilometers, ride on the mobile mantle. When the plates contact each other, stresses arise in the crust. Stresses occur along the plate boundaries by pulling away from, sliding alongside, and pushing against one another. All these movements are associated with earthquakes.

The areas of stress at plate boundaries that release accumulated energy by slipping or rupturing are known as faults. Elastic rebound occurs when the maximum point of supportable strain is reached and a rupture occurs, allowing the rock to rebound until the strain is relieved (Fig. 80-1). Usually, the rock rebounds on both sides of the fault in opposite directions. The point of rupture is called the *focus* and may be located near the surface or deep below it. The point on the surface directly above the focus is termed the *epicenter* (Fig. 80-2).

The energy generated by an earthquake is not always released violently and can be small or gradual. Minor earth tremors are recorded daily in the United States, but whether they are caused by the same processes that can level a city is not known. Most damaging earthquakes are associated with sudden ruptures of the crust.

Characteristics

The actual rupture process may last from a fraction of a second to a few minutes for a major earthquake. Seismic (from the Greek *seismos*, meaning "shock" or "earthquake") waves are generated that last from less than a tenth of a second to a few minutes, and cause ground shaking. The seismic waves propagate in all directions, causing vibrations that damage vulnerable structures and infrastructure.

There are three types of seismic waves. The body waves (P, or primary, and S, or secondary) penetrate the body of the earth, vibrating quickly (Fig. 80-3). P waves travel at about 6 kilometers per second (kps) (3.7 miles per second [mps] and provide the initial jolt that causes buildings to vibrate up and down. S waves travel about 4 kps (2.5 mps) in a movement similar to the snap of a whip, causing a sharper jolt that vibrates structures from side to side and usually resulting in the most destruction. Surface waves (L waves) vibrate the ground horizontally and vertically and cause swaying of tall buildings, even at great distances from the epicenter.

Earthquake focus depth is an important factor in determining the characteristics of the waves. The focal depth can be deep (from 300 to 700 km) 186 to 435 miles or shallow (less than 60 km [37 miles]). Shallow-focus earthquakes are extremely damaging because of their proximity to the surface. The earthquake may be preceded by preliminary tremors and followed by aftershocks of decreasing intensity.

Earthquake Scales

Earthquakes can be described by use of two distinctly different scales of measurement demonstrating magnitude and intensity. Earthquake magnitude, or amount of energy released, is determined by use of a seismograph, which records ground vibrations. The Richter scale mathematically adjusts the readings for the distance of the instrument from the epicenter. The Richter scale is logarithmic; an increase of one magnitude signifies a tenfold increase in ground motion, or about 30 times the energy. Thus, an earthquake with a Richter magnitude of 7.5 releases 30 times more energy than one with 6.5 Richter magnitude. The smallest quake to be felt by humans is of magnitude 3. The largest earthquakes that have been recorded under this system are 9.25 (Alaska, 1969) and 9.5 (Chile, 1960).

The *moment magnitude scale* is a successor to the Richter scale and is most often used to estimate large earthquake magnitudes. Theoretically, all magnitude scales should yield approximately the same value for any given earthquake. However, controversy exists over the measurement of the great Indian Ocean earthquake that occurred on December 26, 2004, which generated a tsunami, killing over 280,000 people. The Pacific Tsunami Warning Centre (PTWC) estimated the magnitude as 8.5 on the Richter scale shortly after the earthquake. The U.S. Geological Survey (USGS), using the moment magnitude scale, increased its estimate from 8.1 to 9.0 Other scientists, using moment magnitude, have revised the estimate to 9.3, and the PTWC has accepted this, but the USGS has so far not changed its estimate of 9.0. The most definitive estimate so far has put the magnitude at 9.15. (Source: Wikipedia, The Free Encyclopedia).

The earthquake intensity scale measures the effects of the earthquake where it occurs. The most widely used scale of this type is the modified Mercalli scale, which expresses the intensity of earthquake effects on people, structures, and the earth's surface in values from I to XII (Table 80-1). Another, more explicit scale used in Europe is the Medvedev-Sponheuer-Karnik (MSK) scale.

Location and Predictability

Most earthquakes (95%) occur in well-defined zones near the boundaries of the tectonic plates. These areas bordering the

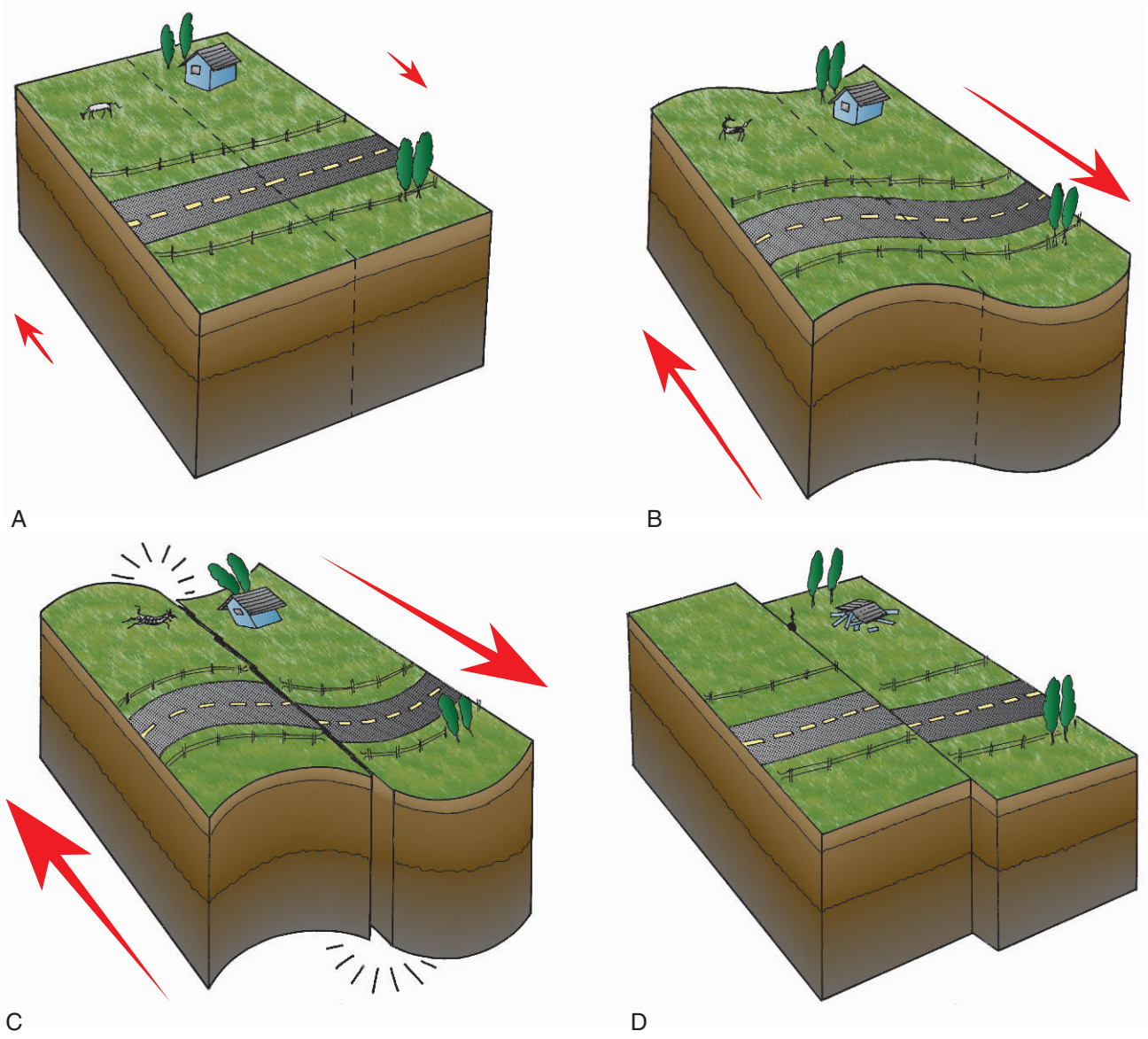


Figure 80-1. Elastic rebound in earthquake. **A,** Forces build up over time. **B,** Crust deforms. **C,** Crust snaps. **D,** Plates slide.

Pacific Ocean are called the circum-Pacific belt. Areas traversing the East Indies, the Himalayas, Iran, Turkey, and the Balkans are called the Alpide belt. Earthquakes also occur along the ocean trenches, such as those around the Aleutians, Tonga, Japan, and Chile and within the eastern Caribbean. Some earthquakes occur in the middle of the plates, possibly indicating where earlier plate boundaries might have been. These have included the New Madrid earthquake in 1811 and the Charleston earthquake in 1816 in the United States, the Agadir earthquake in 1960 in Morocco, and the Koyna earthquake in 1967 in India.

Earthquake prediction was a constant preoccupation for early astrologers and prophets. Some signs of earthquake noted by observers were buildings gently trembling, animals and birds becoming excited, and well water turning cloudy and smelling bad. Although some modern scientists claim ability to predict earthquakes, the methods are still controversial. For example, the 1995 earthquake in Kobe, Japan, which killed more than 5000 people, was not predicted. However, mechanical observation systems make it possible to issue warnings to nearby populations immediately after detection of an earthquake. Reasonable risk assessments of potential earthquake activity can be made with confidence based on the following:

1. Knowledge of seismic zones or areas most at risk, gained through study of historical incidence and plate tectonics
2. Monitoring of seismic activity by use of seismographs and other instruments (The USGS monitors global seismic activity through 2150 seismic stations.)

3. Use of community-based, scientifically sound observations, such as elevation and turbidity of water in wells and radon gas escape into well water

Earthquake Hazards

Earthquakes produce many direct, and sometimes indirect, effects. Landslides, flooding, and tsunamis are considered secondary hazards and are discussed separately in this chapter.

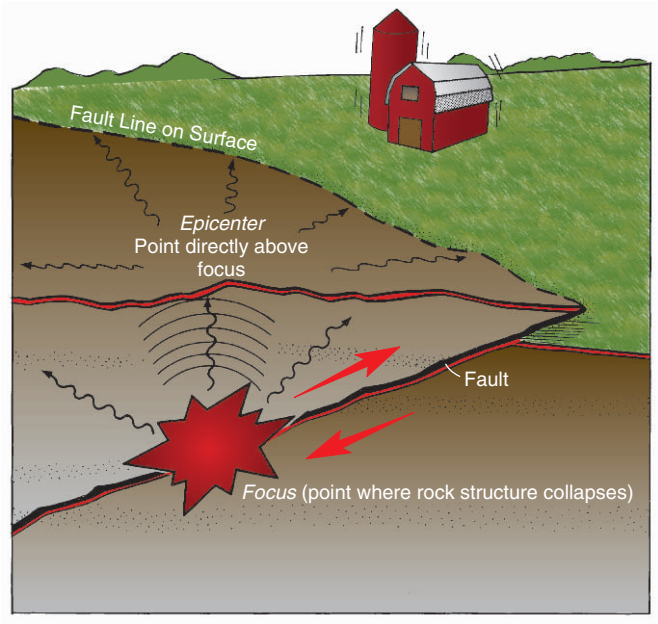


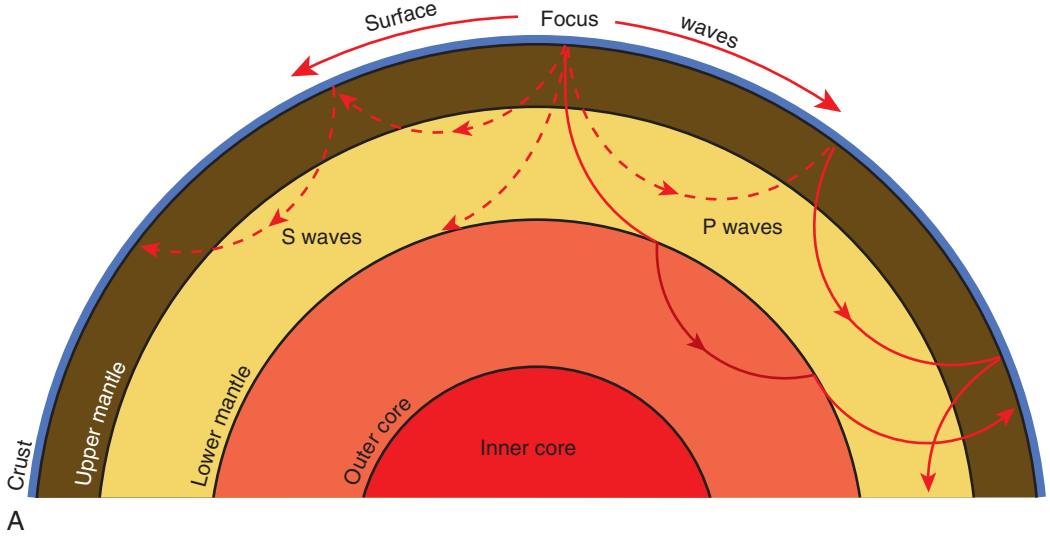
Figure 80-2. Motion of the earth's plates causes increased pressure at faults where the plates meet. Eventually the rock structure collapses, and movement occurs along the fault. Energy is propagated to the surface above and radiates outward. Waves of motion in the earth's crust shake landforms and buildings, causing damage. (Courtesy Disaster Management Center, University of Wisconsin.)

Fault Displacement and Ground Shaking. Fault displacement, either rapid or gradual, may damage foundations of buildings on or near the fault area or may displace the land, creating troughs and ridges. Ground shaking causes more widespread damage, particularly to the built environment. The extent of the damage is related to the size of the earthquake, the closeness of the focus to the surface, the buffering power of the area's rocks and soil, and the type of buildings being shaken. The Northridge, California, quake in 1994 was one of the most costly, producing U.S. $44 billion in damage, due to the position of the event directly below a population center.

Aftershocks may cause further damage and may recur for weeks or even years after the initial event. The Kashmir earthquake (also known as the Northern Pakistan earthquake) occurred on October 8, 2005 and registered 7.6 on the moment magnitude scale. Affecting three countries, this earthquake killed over 90,000 people. Between October and February 2006, there were more than 978 aftershocks with a magnitude of 4.0 and above.

Ground Failure and Soil Liquefaction. Seismic vibrations may cause settlement beneath buildings when soils consolidate or compact. Certain types of soil, such as alluvial and sandy soils, are more vulnerable to failure. Liquefaction is a type of ground failure that occurs when saturated soils lose strength and collapse or become liquefied. During the 1964 earthquake in Nigata, Japan, the ground beneath earthquake-resistant buildings became liquefied, causing the buildings to lean up to 45 degrees from the vertical. Most of these buildings were later jacked back into an upright position and reoccupied. In the 2001 earthquake in the Buhj area of Gudjarat, western India, many reservoir dams were damaged because of water-saturated alluvial foundations.

Lateral Spreads and Flow Failure. Lateral spreads involve the lateral movement of large blocks of soil as a result of liquefaction in a subsurface layer. During the 1964 Alaska earthquake, more than 200 bridges were damaged or destroyed by lateral



A

Figure 80-3. **A,** Propagation of seismic waves in an earthquake.

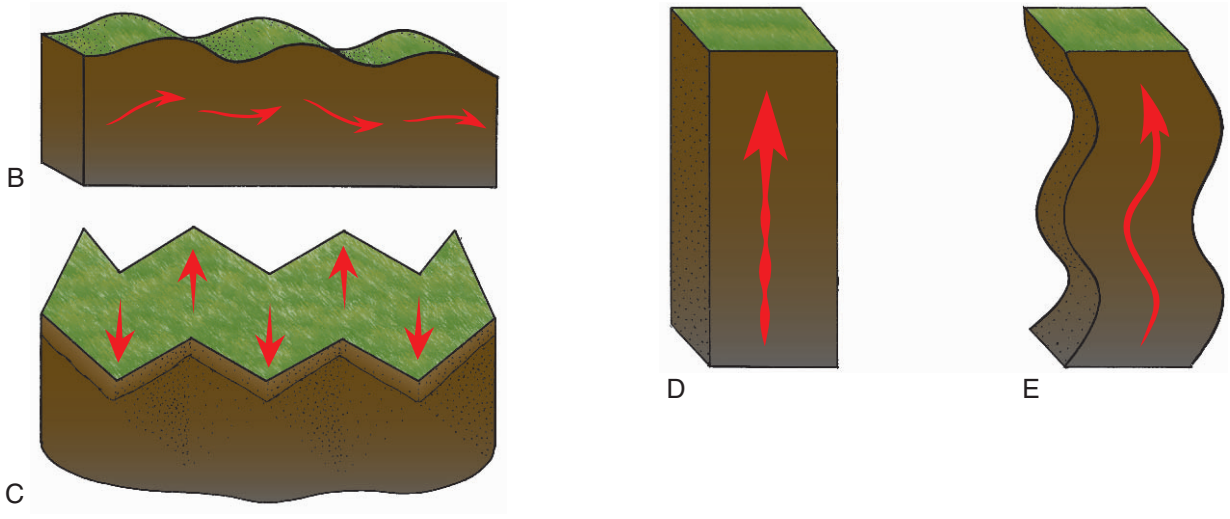


Figure 80-3, cont'd. Surface waves vibrate the ground horizontally (**B** and **C**) and vertically (**D** and **E**).

TABLE 80-1. Modified Mercalli Intensity Scale of 1931

SCALE	DESCRIPTION
I	Not felt except by very few persons under especially favorable circumstances.
II	Felt only by a few persons at rest, especially on upper floors of buildings. Delicately suspended objects may swing.
III	Felt quite noticeably indoors, especially on upper floors of buildings, but many people do not recognize it as an earthquake. Standing motor vehicles may rock slightly. Vibration similar to passing of truck. Duration estimated.
IV	During the day felt indoors by many but outdoors by few. At night some awakened. Dishes, windows, doors disturbed; walls make creaking sound. Sensation resembles heavy truck striking building. Standing motor vehicles rocked noticeably.
V	Felt by nearly everyone; many awakened. Some dishes, windows, etc., broken. A few instances of cracked plaster. Unstable objects overturned. Disturbances of trees, poles, and other tall objects sometimes noticed. Pendulum clocks may stop.
VI	Felt by all; many frightened and run outdoors. Some heavy furniture moved; a few instances of fallen plaster or damaged chimneys. Damage slight.
VII	Everybody runs outdoors. Damage negligible in buildings of good design and construction, slight to moderate in well-built ordinary structures, considerable in poorly built or badly designed structures. Some chimneys broken. Noticed by persons driving motor vehicles.
VIII	Damage slight in specially designed structures, considerable in ordinary substantial buildings with partial collapse, great in poorly built structures. Panel walls thrown out of frame structures. Fall of chimneys, factory stacks, columns, monuments, and walls. Heavy furniture overturned. Sand and mud ejected in small amounts. Changes in well water. Persons driving motor vehicles disturbed.
IX	Damage considerable in specially designed structures. Well-designed structures thrown out of plumb, greatly in substantial buildings with partial collapse. Buildings shifted off foundations. Ground cracked conspicuously. Underground pipes broken.
X	Some well-built wooden structures destroyed. Most masonry and frame structures with foundations destroyed; ground severely cracked. Rails bent. Landslides considerable from river banks and steep slopes. Shifted sand and mud. Water splashed (slopped) over banks.
XI	Few, if any, (masonry) structures remain standing. Bridges destroyed. Broad fissures in ground. Underground pipelines completely out of service. Earth slumps and land slips in soft ground. Rails bent greatly.
XII	Damage total. Practically all works of construction are damaged greatly or destroyed. Waves seen on ground surface. Lines of sight and level are distorted. Objects are thrown upward into the air.

spreading of flood plain deposits toward river channels. In the 1906 San Francisco earthquake, major pipelines were broken by lateral spreading, hampering efforts to fight fires. In 1989, the Marina District in San Francisco, built on soft landfill, was damaged by lateral spreading from the Loma Prieta earthquake.

Flow failure, in which either a layer of liquefied soil rides on top of another layer or blocks of intact material ride on top of liquefied soil, can be catastrophic. Some of the most damaging flow failures have occurred underwater in coastal areas, carrying away large sections of port facilities and generating large sea waves. Some flow failures on land have been as much as a mile in length and breadth, such as those induced by the 1920 earthquake in Kansu, China, which killed 200,000 people.

Landslides and Avalanches. Slope instability may cause landslides and snow avalanches during an earthquake. Steepness, weak soils, and presence of water may contribute to vulnerability from landslides. Liquefaction of soils on slopes may lead to disastrous slides. The most abundant types of earthquake-induced landslides are rock falls and rock slides, usually originating on steep slopes. The Kashmir earthquake of 2005 was characterized by numerous landslides that blocked access by assistance organizations to people in high mountain areas.

Tsunamis. Tsunamis may be generated by undersea or nearshore earthquakes and may break over the coastline with great destructive force. The great tsunami of December 2004 that devastated Banda Aceh, Indonesia, was generated by an earthquake occurring 240 km (149 miles) off the coast of Sumatra at the boundary between the Indian and Burmese tectonic plates in the Andaman-Sumatran subduction zone. A second earthquake of 8.7 magnitude occurred along the same fault in March 2005, but this event produced a much smaller tsunami (4 m [13 ft] versus 9 m [30 ft] in height). The December earthquake ruptured a longer segment of the fault and occurred in much deeper water, creating a larger movement of the sea floor.

Fires. One of the most destructive consequences of an earthquake is fire, particularly in urban centers. Great post-earthquake fires played a major role in the destruction of Lisbon, Portugal, in 1755 and in San Francisco in 1906, and caused considerable damage in Kobe, Japan, in 1995. The million wooden buildings in Tokyo pose a major risk of fire if an earthquake strikes, as predicted in the next few decades.

Typical Adverse Effects

Ground shaking can damage human settlements, buildings and infrastructure (particularly bridges), elevated roads, railways, water towers, water treatment facilities, utility lines, pipelines, electricity generating facilities, and transformer stations. Aftershocks can do great damage to already weakened structures. Significant secondary effects include fires, dam failures, and landslides, which may block waterways and cause flooding. Flooding may be also be caused by seiches (back-and-forth wave action in bays) or by failures in dams and levees. Damage may occur to facilities using or manufacturing dangerous materials, resulting in chemical spills. Communications facilities may break down. Destruction of property may have a serious impact on shelter needs, economic production, and living standards of the affected community. Depending on their level of vulnerability, many people may be homeless in the aftermath of an earthquake.

The casualty rate is often high, especially when earthquakes occur in areas of high population density, particularly when streets between buildings are narrow, buildings are not earthquake resistant, the ground is sloping and unstable, or adobe or dry stone construction is used, with heavy upper floors and roofs.

Casualty rates may be high when quakes occur at night because the preliminary tremors are not felt during sleep and people are not tuned in to receive media warnings. In the daytime, people are particularly vulnerable in large unsafe structures such as schools and offices. Casualties generally decrease with distance from the epicenter. As a rule of thumb, quakes result in three times as many injured survivors as persons killed. The proportion of dead may be higher with major landslides and other secondary hazards. In areas where houses are of lightweight construction, especially with wood frames, casualties are generally much fewer, and earthquakes may occur regularly with no serious, direct effects on human populations.

The most widespread acute, serious medical problems are broken bones. Other health threats may occur with secondary flooding, when water supplies are disrupted (earthquakes can change levels in the water table) and contaminated water is used or water shortages exist, and when people are living in high-density relief camps, where epidemics may develop or food shortages exist.

In the aftermath of the Colombia earthquake of January 1999, which most heavily affected Armenia, the death toll was 1185, and 160,000 people were homeless, most in urban areas. In Armenia, 60% to 70% of homes had been destroyed, movement was restricted by fallen debris, and unemployment rose from 12% to 35%. People were living in unsatisfactory shelters made with plastic sheeting. Many migrated from the area to other places that could not absorb them. Although international response to aid Colombia was strong, the overwhelming need continued to pose problems. Five weeks after the earthquake, supplies of food, clean drinking water, and shelter materials were still urgently required. Hygiene and sanitation services and essential medicines were badly needed. Social services were required to work toward normalizing the lives of victims, especially children. In Gujarat, India, where 30,000 people died in 2001, assistance agencies struggled for years to help rebuild the more than 300,000 houses that were lost. In the Kashmir earthquake of 2005, 3.3 million were made homeless in Pakistan, and many were at risk of dying from the winter cold and spread of disease.

TSUNAMIS

Tsunami is a Japanese word meaning "harbor wave." Although tsunamis are sometimes called "tidal waves," they are unrelated to the tides. The waves originate from undersea or coastal seismic activity, landslides, and volcanic eruptions. They ultimately break over land with great destructive power, often affecting distant shores.

Causal Phenomena and Characteristics

The geologic movements that cause a tsunami are produced in three major ways (Fig. 80-4). The foremost cause is fault movement on the sea floor, accompanied by an earthquake. The second most common cause is a landslide occurring underwater or originating above the sea and then plunging into the water. The highest tsunamis ever reported were produced by a landslide at Lituya Bay, Alaska, in 1958. A massive rock slide produced a wave that reached a high water mark of 530 m (1740 ft) above the shoreline. A third cause of a tsunami is volcanic activity, which may uplift the flank of the volcano or cause an explosion.

Tsunamis differ from ordinary deep ocean waves, which are produced by wind blowing over water. Normal waves are rarely longer than 300 m (984 ft) from crest to crest. Tsunamis, however, may measure 150 km (90 miles) between successive wave crests. Tsunamis travel much faster than ordinary waves. Compared with normal wave speed of around 100 kph (62 mph), tsunamis in the deep water of the ocean may travel at the speed of a jet airplane—800 kph (497 mph). Despite their

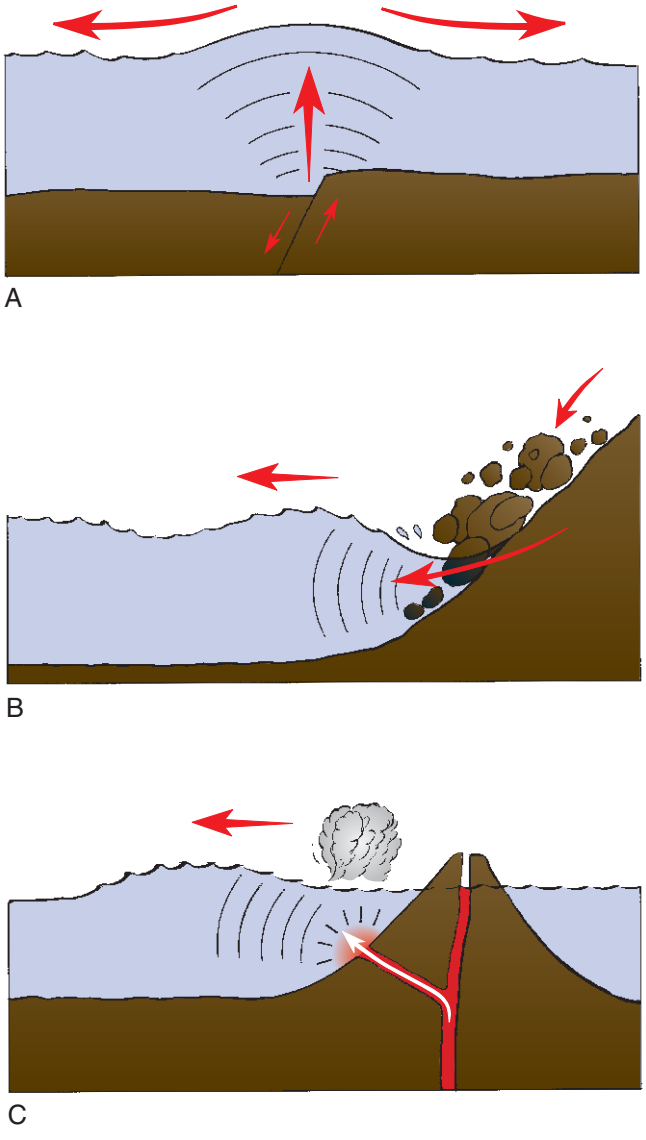


Figure 80-4. Tsunamis are produced in three ways. **A,** Fault movement on the sea floor. **B,** Landslide. **C,** Submarine explosion from volcanic eruption.

speed, tsunamis increase the water height only 30 to 45 cm (12 to 18 inches) and often pass unnoticed beneath ships at sea. In 1946, a ship's captain on a vessel lying offshore near Hilo, Hawaii, claimed he could feel no unusual waves beneath him, although he saw them crashing on the shore.

Contrary to popular belief, the tsunami is not a single giant wave. A tsunami can consist of 10 or more waves, termed a *tsunami wave train*. The waves follow each other in 5- to 90-minute intervals. As tsunamis approach the shore, they travel progressively slower. The final wave speed depends on the water depth. Waves in 18 m (59 ft) of water travel about 50 kph (31 mph). The shape of the near-shore sea floor influences how tsunami waves will behave. Where the shore drops off quickly into deep water, the waves are smaller. Areas with long shallow shelves, such as the major Hawaiian islands, allow formation of very high waves. In the bays and estuaries, seiches where the water sloshes back and forth can amplify waves to some of the greatest heights ever observed.

On shore, the initial sign of a tsunami depends on what part of the wave first reaches land; a wave crest causes a rise in the water level, and a wave trough causes a recession. The rise may not be significant enough to be noticed by the general public. Observers are more likely to notice the withdrawal of water, which may leave fish floundering on the sea floor. A tsunami does not always appear as a vertical wall of water, known as a bore, as typically portrayed in drawings. More often, the effect is that of an incoming tide that floods the land. Normal waves and swells may ride on top of the tsunami wave, or the tsunami may roll across relatively calm inland waters.

The flooding produced by a tsunami may vary greatly from place to place over a short distance, depending on submarine topography, shape of the shoreline, reflected waves, and modification of waves by seiches and tides. The Hilo tsunami of 1946, originating in the Aleutian Trench, produced 18-m (59-ft) waves in one location and only half that height a few miles away. The sequence of the largest wave in the tsunami wave train also varies, and the destructiveness is not always predictable. In 1960 in Hilo, many people returned to their homes after two waves had passed, only to be swallowed up in a giant bore that in this case was the third wave.

Predictability

Tsunamis have occurred in all oceans and in the Mediterranean Sea, but the majority of them occur in the Pacific Ocean. The zones stretching from New Zealand through East Asia, the Aleutians, and the western coasts of the Americas all the way to the South Shetland Islands are characterized by deep ocean trenches, explosive volcanic islands, and dynamic mountain ranges.

Since 1900, the recorded effects of tsunamis included only local casualties and significant damage. Nine tsunamis struck areas throughout the Pacific. The Tsunami Warning System (TWS) was developed in Hawaii shortly after the 1946 Hilo tsunami and is headquartered in the Pacific Warning Center in Honolulu. There are 26 member countries in the Pacific basin. The TWS works by monitoring seismic activity from a network of seismic stations. A tsunami is almost always generated by an undersea earthquake of magnitude 7 or greater. Therefore, special warning alarms sound when a quake measuring 6.5 or more occurs anywhere near the Pacific. A tsunami watch is declared if the epicenter is close enough to the ocean to be of concern. Government and voluntary agencies are then alerted, and local media are activated to broadcast information. The five nearest tide stations monitor their gauges, and trained observers watch the waves. With positive indicators, a tsunami warning is issued.

The TWS met with general success in saving lives during the tsunamis of 1952 and 1957 in Hawaii. In 1960, however, two major earthquakes occurring a day apart rocked the coast of Chile in South America. The first registered 7.5 on the Richter scale and produced a small but noticeable wave in Hilo Bay. The second registered a stunning 8.5, more than 30 times the energy of the first, and authorities predicted generation of a large, destructive tsunami. When the waves hit Hilo, 15 hours after the earthquake, not all the public had taken the warnings

seriously, and 61 people were killed. About 7 hours later the tsunami struck Japan, killing 180. By the time that information of conditions in Chile reached TWS, three giant waves had already destroyed villages along a 500-mile (805-km) stretch of coastal South America, arriving only 15 minutes after the earthquake.

Lack of an effective warning system has been blamed for the extensive loss of life from the tsunami generated in the Indian Ocean in December 2004. Over 290,000 are estimated to have died in 11 countries and thousands remain missing. Tsunamis have been relatively rare in the Indian Ocean, and the area has no international warning system. The first tsunami-generated wave crashed into Sumatra only 30 minutes after shaking from the earthquake had subsided. The tsunami ultimately traveled nearly 5000 km (3107 miles) to Africa. In contrast to stronger preparedness levels in the Pacific countries, citizens and tourists were not fully aware of the dangers and many watched from the beach with catastrophic results. In Kobe, Japan, the World Conference on Disaster Reduction in January of 2005 laid the groundwork for the first tsunami warning system in the Indian Ocean, expected to be in place in 2006.

Vulnerability

The following major factors contribute to vulnerability to tsunamis:

1. Growing world population, increasing urban concentration, and larger investments in infrastructure, particularly on the coastal regions, with some settlements and economic assets on low-lying coastal areas
2. Lack of tsunami-resistant buildings and site planning
3. Lack of a warning system or lack of sufficient education for the public to create awareness of the effects of a tsunami
4. Unpredictable intensity

Typical Adverse Effects

The force of water in a bore, with pressures up to 10,000 kg/m^2, can raze everything in its path. The flooding from a tsunami, however, affects human settlements most, by water damage to homes, businesses, roads, and infrastructure. Withdrawal of the tsunami also causes significant damage. As the water is dragged back toward the sea, bottom sediments are scoured out, collapsing piers and port facilities and sweeping out foundations of buildings. Entire beaches have disappeared, and houses have been carried out to sea. Water levels and currents may change unpredictably, and boats of all sizes may be swamped, sunk, or battered (Figs. 80-5, 80-6, and 80-7).

Casualties and Public Health. Deaths occur principally from drowning as water inundates homes or neighborhoods. Many people may be washed out to sea or crushed by the giant waves. Some injuries occur from battering by debris. Little evidence exists of tsunami flooding directly causing large-scale health problems. Rapid effective assistance to the Banda Aceh area in early 2005 prevented widespread outbreaks of disease in displacement camps (Fig. 80-8). Malaria mosquitoes may increase because of water trapped in pools. Open wells and other groundwater may be contaminated by salt water and debris or sewage. Normal water supplies may be inaccessible for days because of broken water mains.

Crops and Food Supplies. Flooding and damage by tsunami waves may result in the following:

Figure 80-5. Ship washed ashore in tsunami, December 2004, in Koh Lanta, Thailand. (Photo by Sheila Reed.)

Figure 80-6. Boat perched on a house near Banda Aceh, Indonesia, following the December 2004 tsunami. (Photo by Sheila Reed.)

1. The harvest may be lost, depending on time of year.
2. Land may be rendered infertile from saltwater incursion from the sea.
3. Food stocks not moved to high ground are damaged.
4. Animals not moved to high ground may perish.
5. Farm implements may be lost, hindering tillage.
6. Boats and fishing nets may be lost.

In July 1998, an earthquake of magnitude 7 occurred close to the northwest coast of Papua New Guinea. Although the tremor was felt over a large area, no earthquake damage was reported. Only 10 minutes after the quake, however, the first of three 7- to 10-m (23- to 33-ft) waves came ashore in Sandaun Province. The tsunamis struck at high speed after dark and penetrated up to 1 km (over half a mile) inland, totally destroying

Figure 80-7. Generator ship washed ashore 2.3 miles inland, near Banda Aceh, Indonesia. (Photo by Sheila Reed.)

Figure 80-8. Displaced people in temporary settlements in Aceh province, Indonesia. (Photo by Sheila Reed.)

villages and vegetation along 50 km (31 miles) of the coast. Of the 9000 people affected, more than 2000 died, mainly from being battered by debris as they were swept away by the water.

VOLCANIC ERUPTIONS

A volcano is a vent or chimney to the earth's surface from a reservoir of molten rock, called magma, deep in the earth's crust (see Chapter 15). Approximately 500 volcanoes are active (have erupted in recorded history), and many thousands are dormant (could become active again) or extinct (are not expected to erupt again). On average, about 50 volcanoes erupt every year; only about 150 are routinely monitored. Since 1000 AD, more than 300,000 people have been killed directly or indirectly by volcanic eruptions, and currently about 10% of the world's population lives on or near potentially dangerous volcanoes.

Volcanology, the study of volcanoes, has experienced a period of intensified interest after five major eruptions in the 1980s and early 1990s: Mt. St. Helens in the United States (1980), El Chichón in Mexico (1982), Galunggung in Indonesia (1982), Nevado del Ruiz in Colombia (1985), and Mt. Pinatubo in the Philippines (1991). Although the Mt. St. Helens eruptions were predicted with remarkable accuracy, predictive capability on a worldwide basis for more explosive eruptions has not been achieved. No recognized immediate precursors to the eruption of El Chichón were known. It caused the worst volcanic disaster in Mexico's history. Despite sufficient warnings, ineffective implementation and evacuation measures resulted in more than 22,000 deaths from the eruption of Nevado del Ruiz. Galunggung erupted for 9 months, disrupting the lives of 600,000 people. Despite a major evacuation effort from Mt. Pinatubo, 320 people died, mainly from collapse of ash-covered roofs. A study of these eruptions underscores the importance of predisaster geoscience studies, volcanic hazard assessments, volcano monitoring, contingency planning, and enhanced communications between scientists and authorities. The world's most dangerous volcanoes are in densely populated countries where only limited resources exist to monitor them.

Causal Phenomena

The basic ingredients for a volcanic eruption are magma and an accumulation of gases beneath an active volcanic vent, which may be either on land or below the sea. Magma is composed of silicates containing dissolved gases and sometimes crystallized minerals in a liquidlike suspension. Driven by buoyancy and gas pressure, magma, which is lighter than surrounding rock, forces its way upward. As it reaches the surface, the pressures decrease, enabling the dissolved gases to effervesce, pushing the magma through the volcanic vent as the gases are released.

The chemical and physical composition of magma determines the amount of force with which a volcano erupts. Magmas that are less viscous allow gas to be released more easily. More viscous magma, perhaps containing a greater concentration of solid particles, may confine these gases longer, allowing greater pressures to build up. This greater pressure may lead to more violent eruptions.

Types

Volcanic eruptions may be described as follows in descending order of intensity (Fig. 80-9).

Pelean. This is the most disastrous type of eruption. The hardened plug at the volcano's throat forces the magma to blast out through a weak spot in the volcanic flank. The great force of the blast devastates most objects in its path, as occurred in the Mt. St. Helens eruption of 1980.

Plinean. As the pressure on the magma is released, a violent upward expulsion of gas can extend far into the atmosphere. In 1991, Mt. Pinatubo sent a plume of tephra 30 km (19 miles) above the surface.

Vesuvian. As in the eruption of Mt. Vesuvius in Italy in 79 AD, this type is very explosive and occurs infrequently. The

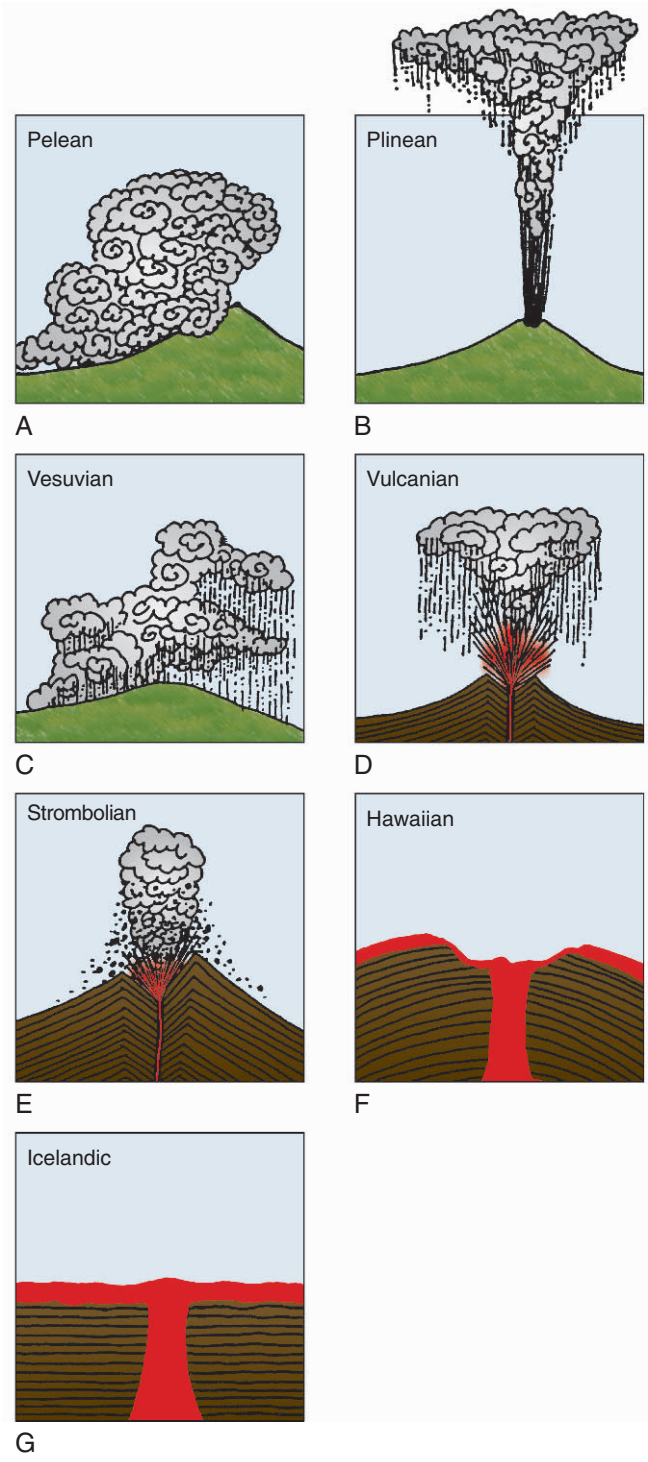


Figure 80-9. Eruption types. **A,** Pelean. **B,** Plinean. **C,** Vesuvian. **D,** Vulcanian. **E,** Strombolian. **F,** Hawaiian. **G,** Icelandic. (Modified from Introduction to Hazards, 3rd ed. New York, Disaster Management Training Programme, United Nations Development Program, UN Organization for Coordination of Humanitarian Assistance, 1997.)

explosion of built-up magma discharges a cloud of ash over a wide area.

Vulcanian. Lava forms a crust over the volcanic vents between eruptions, building up the volcano. Subsequent eruptions are more violent and eject dense clouds of material. The Paracutin, Mexico, volcano originated in a cornfield in 1943 and eventually covered 260 square km (162 miles). A major eruption occurred in 1947.

Strombolian. Gases escape through slow-moving lava in moderate explosions that may be continuous. Volcanic "bombs" of clotted lava may be ejected into the sky, as occurred in the 1965 eruption of Irazu in Costa Rica.

Hawaiian. The lava is mobile and flows freely. Gases are released quietly, as in the Kīlauea, Hawaii, volcano, which has continued to erupt since 1983.

Icelandic. Similar to the Hawaiian type, lava flows from deep fissures and forms sheets spreading out in all directions, as in the Laki, Iceland, eruption of 1783.

Characteristics

No international scale exists to measure the size of volcanic eruptions. The volcanic explosivity index (VEI) estimates the energy released in a volcanic eruption, based on measurements of the ejected matter, height of the eruption cloud, and other observations. The VEI scale ranges from 0 to 8. The largest eruption recorded was in Tambora, Indonesia, in 1815, which had a VEI of 7.

The primary volcanic hazards are associated with products of the eruption: pyroclastic flows, air-fall tephra, lava flows, and volcanic gases. The most destructive secondary hazards include lahars, landslides, and tsunamis.

Pyroclastic Flows. Pyroclastic (meaning "fire-broken" in Greek) flows are the most dangerous of all volcanic phenomena because there is virtually no defense against them. They are horizontally directed explosions or blasts of gas containing ash and larger fragments in suspension. They travel at great speed and burn everything in their path. The flows move like a snow or rock avalanche because they contain a heavy load of dust and lava fragments that are denser than the surrounding air. Gas continues to be released as they travel, creating a continuously expanding cloud.

Pyroclastic flows are responsible for the majority of deaths associated with volcanic eruptions. The pyroclastic flows from the Mt. St. Helens eruption in 1980 moved at rates up to 870 kph (541 mph), and pyroclastic deposits found 2 days after the blast at the foot of the mountain registered temperatures of more than 700° C (1292° F). The greatest distance recorded of such flows in historical times is 35 km (22 miles).

Air-Fall Tephra. Tephra smaller than 2 mm is classified as ash. Almost all volcanoes emit ash, but emissions vary widely in volume and intensity. Heavy ashfalls can cause complete darkness or drastically reduce visibility. Fine material from great eruptions may travel around the world and affect world climate. Clouds of dust and ash can remain in the air for days or weeks and spread over large distances, causing difficulty in driving and

breathing as well as contributing to building collapse and air traffic disruption. The largest tephra are rocks or blocks, sometimes called "bombs," which have been known to travel more than 4 km (2.5 miles). Tephra may be hot enough to start fires when it lands on structures or vegetation.

Lava Flows. Lava flows are formed by hot, molten lava flowing from a volcano and spreading over the surrounding countryside. Depending on the viscosity, a flow may move a few meters per hour. It is usually slow enough that living creatures can move to safety. Sometimes the edges break off, causing small hot avalanches.

Volcanic Gases. Gas is a product of every eruption and may also be emitted by the volcano during periods of inactivity, either intermittently or continually. Volcanic gas is composed mostly of steam, although often present are large amounts of toxic sulfur dioxide, hydrogen sulfide, and smaller but measurable amounts of toxic hydrochloric and hydrofluoric acid gases. Carbon dioxide is often a major component of volcanic gas and is an asphyxiant because it is much denser than air and tends to travel to and through low-lying areas and valleys. Several mountain climbers and skiers in Japan were overcome by hydrogen sulfide fumes in a valley near the Kusatsu-Shirane volcano, and eventually an alarm system was installed. In 1986, approximately 1800 people were asphyxiated by gas bursts from crater lakes in Cameroon.

Lahars and Landslides. Enormous quantities of ash and larger fragments (tephra) accumulate after an eruption on the steep slopes of a volcano, sometimes to a depth of several meters. When mixed with water, the volcanic debris is transformed into a material resembling wet concrete that flows easily downhill. *Lahar* is an Indonesian word for debris flows or mudflows. A primary debris flow is caused by eruptive activity, such as melting of snow and ice by hot volcanic materials, and a secondary debris flow results when heavy rainfall saturates the deposits.

The rate of flow is affected by the volume of mud and debris, viscosity, and slope and character of the terrain. Velocity may reach 100 kph (62 mph), and distance traveled may exceed 100 km (62 miles). Mudflows and debris flows can be very destructive. They have buried entire towns, such as Armero, Colombia. They can silt up waterways, causing floods and changing river courses.

Landslides and debris avalanches are common where stress from intruding magma causes fractures along cracks in the volcano. Ground deformation from swelling and hardening of volcanic material can produce landslides.

Tsunamis. Tsunamis, described previously, are generated by movement of the ocean floor, possibly caused by a volcano. In a study of volcanic eruptions in the past 1000 years, human fatalities resulting from indirect tsunami wave hazards were as significant as those from pyroclastic flows and primary mudflows.

Location

The distribution of volcanoes, as with earthquakes, is determined by the location of geologic forces involving the tectonic or crustal plates. About 80% of the active volcanoes are located near subduction boundaries. Subduction volcanoes occur where denser crustal plates are shoved beneath less dense continental plates, which occurs in most of the Pacific Ocean, known as the Pacific Ring of Fire. Subduction volcanoes are found in the Aleutian Islands stretching from Alaska to Asia, Japan, the Philippines, Indonesia, and the Cascade Range in the United States. Many volcanoes are located beneath the ocean, and submarine eruptions may cause tsunamis and other effects.

Rift volcanoes occur at divergent zones where two distinct plates are slowly being separated. Rift volcanoes, such as those in Iceland and East Africa, account for about 15% of active volcanoes. Hot spot volcanoes are located where crustal weaknesses allow molten material to penetrate, but not necessarily on the plate boundaries. These isolated regions of volcanic activity exist in about 100 places in the world. The Hawaiian islands, in the middle of the Pacific plate, and Yellowstone Park, within the North American plate, are good examples.

Predictability

Systematic surveillance of volcanoes, begun early in the 20th century at the Hawaiian Volcano Observatory, indicates that most eruptions are preceded by measurable geophysical and geochemical changes. Short-term forecasts of future volcanic activity in hours or months may be made through volcano monitoring techniques that include seismic monitoring, ground deformation studies, and observations and recordings of hydrothermal, geochemical, and geoelectric changes. By carefully monitoring these factors, scientists were able to issue a high confidence forecast of the 1991 Mt. Pinatubo eruption, allowing a largely successful evacuation. The best basis for long-term forecasting (within a year or longer) of a possible eruption is through geologic studies of the past history of each volcano. Each past eruption has left records in the form of lava beds. These are deposits and layers of ash and tephra that can be studied to determine the extent of the flows and length of time between eruptions.

Problems in Eruption Forecasting and Prediction. Although significant progress has been made in long-term forecasting of volcanic eruptions, monitoring techniques have not progressed to the point of yielding precise predictions. For the purposes of warning the public and avoiding false alarms that create distrust and chaos, ideal predictions should provide precise information concerning the place, time, type, and magnitude of the eruption. The importance of enhanced communications between scientists and authorities is also emphasized. Despite sufficient warning, evacuation orders were not issued by local authorities, which resulted in more than 22,000 deaths from lahars produced by Nevado del Ruiz. The eruption of Mt. St. Helens was adequately monitored and forecasted, but the main explosion surprised authorities because the volcano did not exhibit expected signs before eruption and because the blast was lateral rather than vertical; 57 people who remained in the danger area were killed.

The greatest constraint to predictability is lack of baseline monitoring studies, which depict the full range of characteristics of the volcano. Interpretation of baseline data enables differentiation of the precursory pattern of an actual eruption from other volcanic activity, such as intrusion of magma under the surface, which is sometimes termed *aborted eruption*. Before the 1982 eruption of El Chichón, virtually nothing was known of

its history of frequent and violent eruptions. No monitoring was conducted before or during the brief eruption.

Developing countries suffer the greatest economic losses from volcanic eruptions. More than 99% of eruption-caused deaths since 1900 have been in developing countries. Because of shortages of funds and trained personnel, monitoring is also poorest in these countries.

Vulnerability

Rich volcanic soils and scenic terrains attract people to settle on the flanks of volcanoes. These people are more vulnerable if they live downwind from the volcano, in the path of historical channels for mudflows or lava flows, or close to waterways likely to flood because of silting. Structures with roof designs that do not resist ash accumulation are vulnerable even miles from a volcano. All combustible materials are at risk.

Typical Adverse Effects

Casualties and Health. Deaths can be expected from pyroclastic flows and mudflows and to a much lesser extent from lava flows and toxic gases. Injuries may occur from impact of falling rock fragments and from being buried in mud. Burns to the skin, breathing passages, and lungs may result from exposure to steam and hot dust clouds. Ashfall and toxic gases may cause respiratory difficulties for people and animals. Nontoxic gases of densities greater than air, such as carbon dioxide, can be dangerous when they collect in low-lying areas. Water supplies contaminated with ash may contain toxic chemicals and cause illness. Deaths have also occurred indirectly from starvation and from tsunami waves.

Settlements, Infrastructure, and Agriculture. Complete destruction of everything in the path of pyroclastic or lava flows should be expected, including vegetation, agricultural land, human settlements, structures, bridges, roads, and other infrastructure. Structures may collapse under the weight of ash, particularly if the ash is wet. Falling ash may be hot enough to cause fires. Flooding may result from waterways filling up with volcanic deposits or from melting of large amounts of snow or glacial ice. Rivers may change course because of oversilting. Ashfall can destroy mechanical systems by clogging openings, such as those in irrigation systems and airplane and other engines. Communication systems could be disrupted by electrical storms developing in the ash clouds. Transportation by air, land, and sea may be affected. Disruption in air traffic from large ash eruptions can have serious effects on emergency response.

Crops in the path of flows are destroyed, and ashfall may render agricultural land temporarily unusable. Heavy ash loads may break the branches of fruit or nut trees. Livestock may inhale toxic gases or ash. Ash containing toxic chemicals, such as fluorine, may contaminate the grazing lands.

The Caribbean island of Montserrat has undergone volcanic activity for years. In June 1997, the famous Soufrière Hills volcano erupted, causing at least nine deaths. The resulting pyroclastic flows buried and destroyed seven villages. Only one third of the island is now considered relatively safe. In 2002, in the Democratic Republic of the Congo, lava poured from the Nyiragongo volcano, devastating the city of Goma and forcing 300,000 to flee some crossing the border into Rwanda. A multidonor funded observatory was ultimately established to monitor the volcano, which emits 12,000 to 50,000 metric tons of sulfur dioxide each day.

See also Chapter 15 for more information on volcanic eruptions.

LANDSLIDES

Landslides are a major threat each year to human settlements and infrastructure. *Landslide* is a general term covering a wide variety of landforms and processes involving the downslope movement of soil and rock. Although landslides may occur with earthquakes, floods, and volcanoes, they are much more widespread and over time cause more property loss than any other geologic event.

Causal Phenomena

Landslides result from sudden or gradual changes in the composition, structure, hydrology, or vegetation of a slope. These changes may be natural or caused by humans, and they disturb the equilibrium of the slope's materials. A landslide occurs when the strength of the material in the slope is exceeded by the downslope stress. The resistance in a slope may be reduced by the following:

- Increase in water content, caused by heavy rainfall or rising ground water
- Increase in slope angle, for new construction or by stream erosion
- Breakdown or alteration of slope materials, from weathering and other natural processes, placement of underground piping for utilities, or use of landfill

Downslope stress may be caused by the following:

- Vibrations from earthquakes (triggering some of the most disastrous landslides), blasting, machinery, traffic, or thunder
- Removal of lateral support by previous slope failure, construction, or excavation
- Removal of vegetation by fires, logging, overgrazing, or deforestation that causes loosening of soil particles and erosion
- Loading with weight from rain, hail, snow, accumulation of loose rock or volcanic material, weight of buildings, or seepage from irrigation and sewage systems

Characteristics

Landslides usually occur as secondary effects of heavy storms, earthquakes, and volcanic eruptions. The materials that compose landslides are divided into two classes: bedrock and soil (earth and organic matter debris). A landslide may be classified by its type of movement (Fig. 80-10).

Falls. A fall is a mass of rock or other material that moves downward by falling or bouncing through the air. These are most common along steep road or railroad embankments, steep escarpments, or steeply undercut cliffs, especially in coastal areas. Large individual boulders can cause significant damage.

Slides. Resulting from shear failure (slippage) along one or several surfaces, the slide material may remain intact or break up.

Topple. A topple is caused by overturning forces that rotate a rock out of its original position. The rock section may have

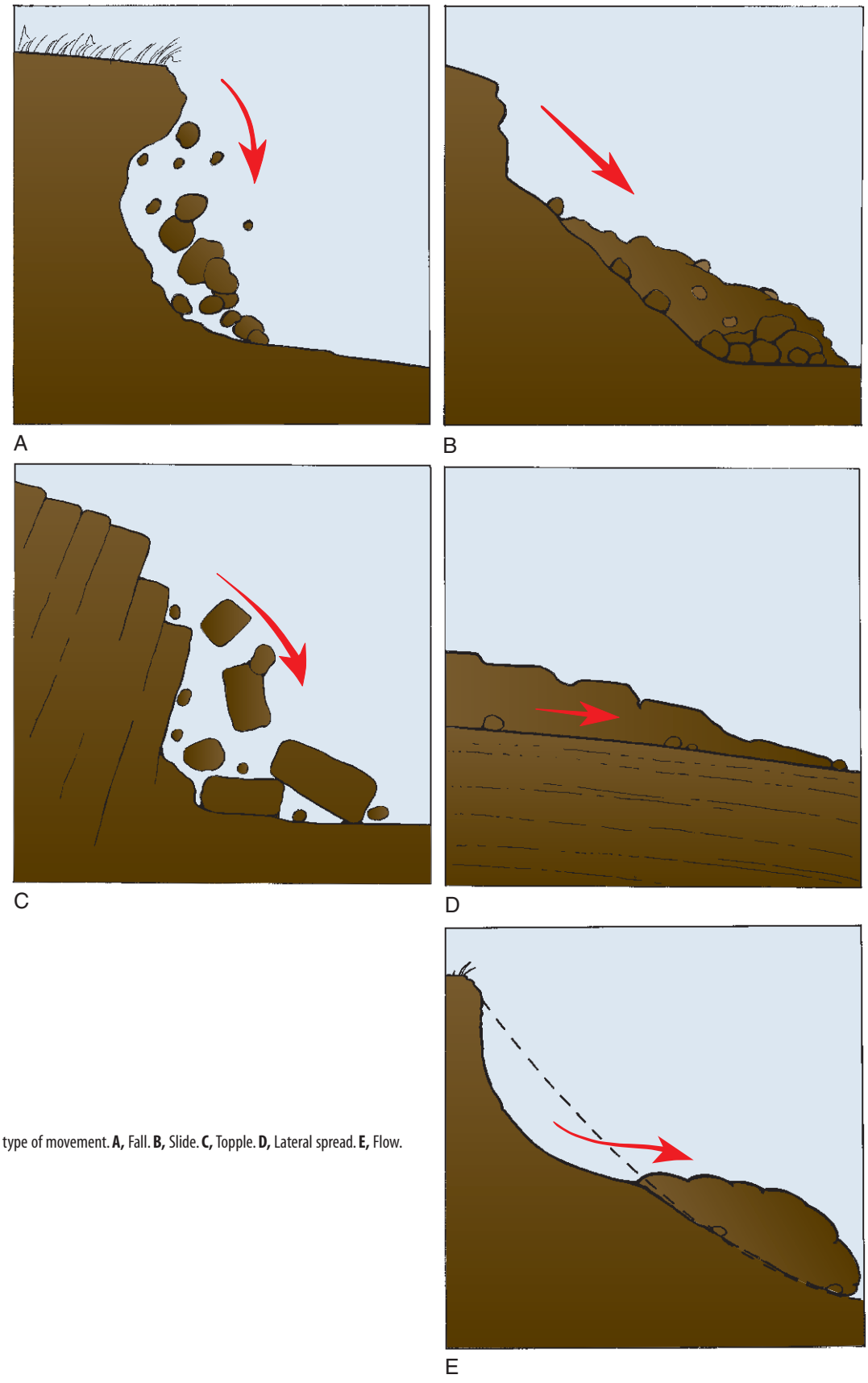


Figure 80-10. Landslides classified by type of movement. **A,** Fall. **B,** Slide. **C,** Topple. **D,** Lateral spread. **E,** Flow.

settled at a precarious angle, balancing itself on a pivotal point from which it tilts or rotates forward. A topple may not involve much movement, and it does not necessarily trigger a rockfall or rock slide.

Lateral Spread. Large blocks of soil spread out horizontally by fracturing off the original base. Lateral spreads occur generally on gentle slopes, usually less than 6%, and typically spread 3 to 5 m (10 to 16 ft), but may move from 30 to 50 m (100 to 160 ft) where conditions are favorable. Lateral spreads usually break up internally and form numerous fissures and scarps. The process can be caused by liquefaction, in which saturated loose sands or silts assume a liquefied state. A lateral spread is usually triggered by ground shaking (as with an earthquake). During the 1964 Alaskan earthquake, more than 200 bridges were damaged or destroyed by lateral spreading of floodplain deposits near river channels.

Flows. Flows move as a viscous fluid, sometimes very rapidly, and can cover several miles. Water is not essential for flows to occur; however, most flows form after periods of heavy rainfall. A mudflow contains at least 50% sand, silt, and clay particles. A lahar is a mudflow that originates on the slope of a volcano and may be triggered by rainfall, sudden melting of snow or glaciers, or water flowing from crater lakes. A debris flow is a slurry of soils, rocks, and organic matter combined with air and water. Debris flows usually occur on steep gullies. Very slow, almost imperceptible flows of soil and bedrock are called creeps. Over long periods, creeps may cause telephone poles or other objects to tilt downhill.

Predictability

The velocity of landslides varies from extremely slow (less than 0.06 m/year) to extremely fast (greater than 3 m/sec), which might imply a similar variation in predictability. In absolute terms, however, predicting the actual occurrence of a landslide is extremely difficult, although situations of high risk, such as forecasted heavy rainfall or seismic activity combined with landslide susceptibility, may lead to estimation of a time frame and possible consequences.

Estimation of landslide hazard potential includes historical information on the geology, geomorphology (study of landforms), hydrology, and vegetation of a specific area. Structural features that may affect stability include sequence and type of layering, lithologic changes, planes, joints, faults, and folds. The most important geomorphologic consideration in the prediction of landslides is the history of landslides in a given area. Such was the case with the tragic slide in Santiago Atitlán, Guatemala, which partially buried its hospital and killed many hundreds of unfortunate victims on October 5, 2005 (Fig. 80-11).

The source, movement, amount of water, and water pressure must be studied. Climatic patterns combined with soil type may cause different types of landslides. For example, when monsoons occur in tropical regions, large debris slides of soils, rocks, and organic matter may occur. Plant cover on slopes may have either a positive or negative stabilizing effect. Roots may decrease water runoff and increase soil cohesion; conversely, they may widen fractures in rock surfaces and promote infiltration. In Nepal in 2002, a heavy monsoon season caused flooding and landslides, killing 500 people. The vulnerability to landslides was increased due to the proximity of most communities to slopes and the poor quality of housing.

Figure 80-11. Former hospital in Santiago Atitlán, Guatemala, buried in mudslide. (Photo by Paul S. Auerbach, MD.)

Vulnerability

Settlements built on steep slopes, in weak soils, on cliff tops, at the base of steep slopes, on alluvial outwash fans, or at the mouth of streams emerging from mountain valleys are all vulnerable. Roads and communication lines through mountainous areas are in danger. In most types of landslides, damage may occur to buildings even if foundations have been strengthened. Infrastructural elements, such as buried utility lines or brittle pipes, are vulnerable.

Typical Adverse Effects

Anything on top of or in the path of a landslide will be severely damaged or destroyed. In addition, rubble may damage lines of communication or block roadways. Waterways may be blocked, creating a flood risk. Casualties may not be widespread, except in the case of massive movements caused by major hazards such as earthquakes and volcanoes.

In addition to direct damage from a landslide, indirect effects include loss of productivity of agricultural or forest lands (if buried), reduced real estate values in high-risk areas and lost tax revenues from these devaluations, adverse effects on water quality in streams and irrigation facilities, and secondary physical effects, such as flooding.

Casualties. Fatalities have resulted from slope failure where population pressure has prompted settlement in areas vulnerable to landslides. Casualties may be caused by collapse of buildings or burial by landslide debris. Worldwide, approximately 600 deaths occur per year, mainly in the circum-Pacific region. The estimate for loss of life in the United States is 25 lives per year, greater than the average loss from earthquakes. Catastrophic landslides have killed many thousands of persons, such as the debris slide on the slopes of Huascaran in Peru triggered by an earthquake in 1970, which killed more than 18,000 people. In January 1989, only 6 weeks after an earthquake killed 25,000 people in Armenia, another quake struck the

Republic of Tajikistan, 50 km (31 miles) southwest of the capital city of Dushanbe. This quake registered 5.8 on the Richter scale. The earthquake triggered a landslide of hillside soils that had become wet with melted snow. The liquefied soil spilled downhill and eventually covered an area about 8 km (5 miles) long and 1 km ($^3/_5$ of a mile) wide. The total volume of mud was more than 10 million m^3. The epicenter of the earthquake was located in the village of Sharora. This village and several others were engulfed with mud that killed 200 persons and left 30,000 homeless. Mud deposits reached a height of 25 m (82 ft) in Sharora, causing rescue efforts to be abandoned. The area was later declared a national monument.

TROPICAL CYCLONES

The World Meteorological Organization (WMO) uses the generic term *tropical cyclone* to cover weather systems in which winds exceed gale force (minimum of 34 knots or 63 kph). Tropical cyclones are rotating, intense low-pressure systems of tropical oceanic origin. Winds of hurricane force (63 knots or 117 kph) mark the most severe type of tropical storm. They are called hurricanes in the Caribbean, the United States, Central America, and parts of the Pacific; typhoons in the northwest Pacific and east Asia; severe cyclonic storms in the Bay of Bengal; and severe tropical cyclones in south Indian, South Pacific, and Australian waters. For easy identification and tracking, the storms are generally given alternating masculine and feminine names or numbers that identify the year and annual sequence.

Tropical cyclones are the most devastating of seasonally recurring rapid-onset natural hazards. Between 80 and 100 tropical cyclones occur around the world each year. Devastation by violent winds, torrential rainfall, and accompanying phenomena, including storm surges and floods, can lead to massive community disruption. The official death toll in individual tropical cyclones reached 140,000 (Bangladesh, 1991), and damages approached $10 billion from hurricane Gilbert (1988) and hurricane Hugo (1989). The damages from hurricane Andrew in Florida and Louisiana in 1992 totaled $16 billion. Hurricane Katrina, in August 2005, killed 1417 people in three U.S. states and caused $75 billion in damages. The deadliest tropical cyclone on record, the Bhola cyclone in Bangladesh in 1970, killed 500,000.

Causal Phenomena

The development cycle of tropical cyclones may be divided into three stages: formation and initial development, full maturity, and modification or decay. Depending on their tracks over the warm tropical seas and proximity to land, tropical cyclones may last from less than 24 hours to more than 3 weeks (the average duration is about 6 days). Their tracks are naturally erratic but initially move generally westward, then progressively poleward into higher latitudes, where they may make landfall, or into an easterly direction as they lose their cyclonic structure.

Formation and Initial Development Stage. Four atmospheric and oceanic conditions are necessary for development of a cyclonic storm (Fig. 80-12):

1. A warm sea temperature (greater than 26°C [78.8°F] to a depth of 60 m [197 ft]) provides abundant water vapor in the air by evaporation.
2. High relative humidity (degree to which the air is saturated by water vapor) of the atmosphere to a height of about 7000 m (23,000 ft) facilitates condensation of water vapor into water droplets and clouds, releases heat energy, and induces a drop in barometric pressure.
3. Atmospheric instability (an above-average decrease of temperature with altitude) encourages considerable vertical cumulus cloud convection when condensation of rising air occurs.
4. A location of at least 4 to 5 latitude degrees from the equator allows the influence of the forces as the earth's rotation (Coriolis force) takes effect and induces cyclonic wind circulation around a low-pressure center.

The atmosphere can usually organize itself into a tropical cyclone in 2 to 4 days. This process is characterized by increasing thunderstorms and rain squalls at sea. Meteorologists can monitor these processes with weather satellites and radar from as far as 400 miles (645 m) away from the storm. The existence of favorable conditions for cyclone development determines the cyclone season for each monitoring center. In the Indian and south Asian region the season is divided into two periods, from April to early June and from October to early December. In the Caribbean and United States, tropical storms and hurricanes reach their peak strengths in middle to late summer. In the Southern Hemisphere, the cyclone season extends from November to April or May, but occasionally cyclones occur in other months in lower latitudes.

Maturity Stage. As viewed by weather satellites and radar imagery, the main physical feature of a mature tropical cyclone is a spiral pattern of highly turbulent, giant cumulus thundercloud bands. These bands spiral inward and form a dense, highly active central cloud core that wraps around a relatively calm and cloud-free "eye." The eye, where light winds occur, typically has a diameter of 20 to 60 km (12 to 37 miles) and appears as a black hole or dot surrounded by white clouds.

In contrast to the light wind conditions in the eye, the turbulent cloud formations extending outward from the eye accompany winds of up to 250 kph (155 mph), sufficient to destroy or severely damage most nonengineered structures in the affected communities. These strong winds are caused by a horizontal temperature gradient that exists between the warm core of the cyclone (up to 10°C (18°F) higher than the external environment) and the surrounding areas, resulting in a correspondingly high-pressure gradient.

Decay Stage. A tropical cyclone begins to weaken, in terms of its central low pressure, internal warm core, and extremely high winds, as soon as its sources of warm moist air begin to ebb or are abruptly cut off. This would occur during landfall, by movement into higher latitudes, or through influence of another low-pressure system. The weakening of a cyclone does not mean that danger to life and property is over. When the cyclone hits land, especially over mountainous or hilly terrain, widespread riverine and flash flooding may last for weeks. The energy from a weakening tropical cyclone may be reorganized into a less concentrated but more extensive storm system, causing widespread violent weather.

Characteristics

Tropical cyclones are characterized by their destructive winds, storm surges, and exceptional level of rainfall, which may cause flooding.

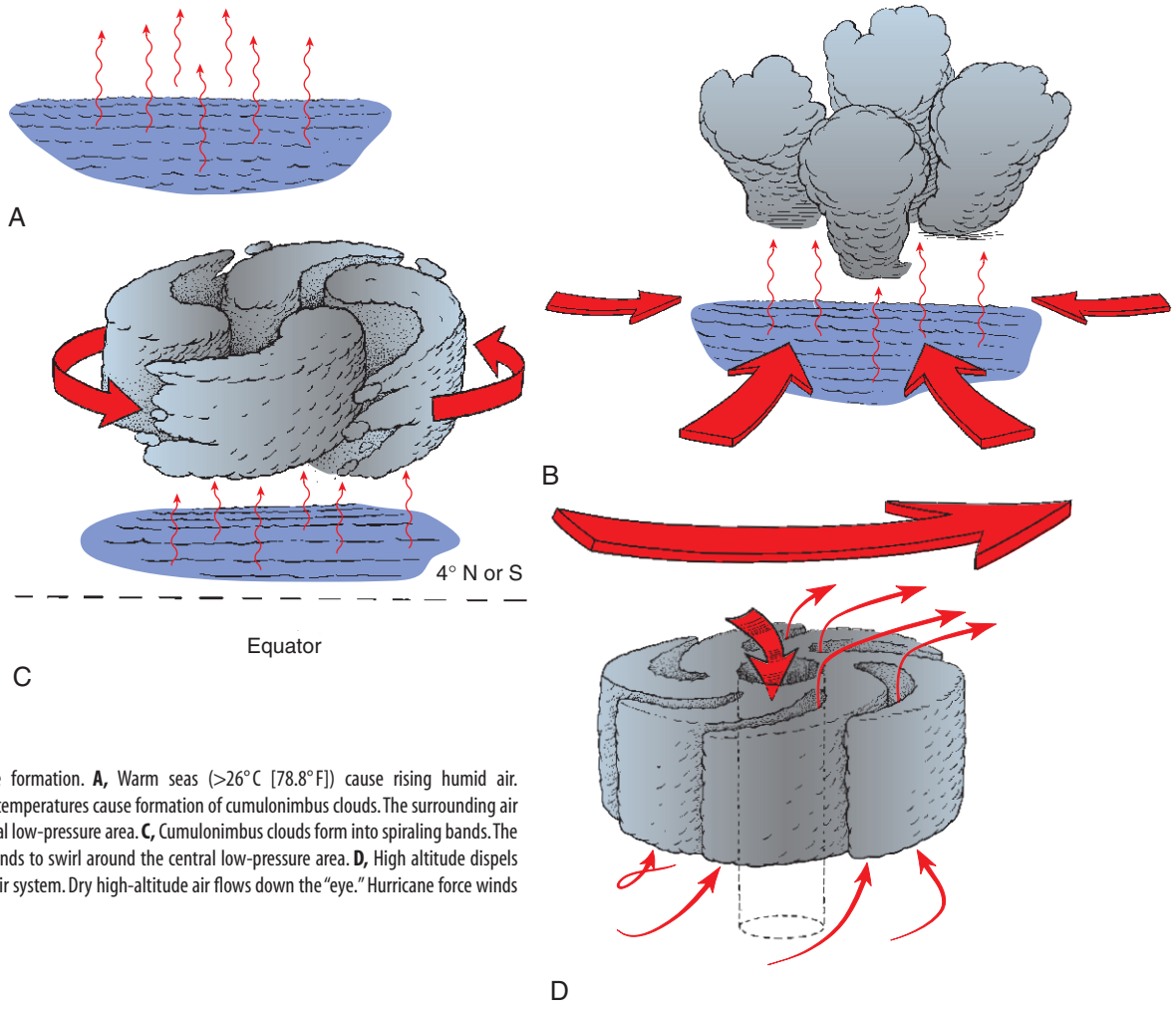


Figure 80-12. Cyclone formation. **A,** Warm seas (>26°C [78.8°F]) cause rising humid air. **B,** Cooler high-altitude temperatures cause formation of cumulonimbus clouds. The surrounding air moves toward the central low-pressure area. **C,** Cumulonimbus clouds form into spiraling bands. The Coriolis effect causes winds to swirl around the central low-pressure area. **D,** High altitude dispels the top of the cyclonic air system. Dry high-altitude air flows down the "eye." Hurricane force winds circle around the eye.

Destructive Winds. The strong winds generated by a tropical cyclone circulate clockwise in the Southern Hemisphere and counterclockwise in the Northern Hemisphere, while spiraling inward and increasing toward the cyclone center. Wind speeds progressively increase toward the core as follows:

1. 150 to 300 km (93 to 186 miles) from the center of a typical mature cyclone, winds of 63 to 88 kph (39 to 54.7 mph)
2. 100 to 150 km (62 to 93 miles) from the center, storm force winds of 89 to 117 kph (55 to 73 mph)
3. 50 to 100 km (31 to 62 miles) from the center, winds in excess of hurricane force, 117 kph (73 mph) or greater
4. 20 to 50 km (12 to 31 miles) from the center, the edge of the inner core containing winds 250 kph (155 mph) or greater

As the eye arrives, winds fall off to become almost calm, but they rise again just as quickly as the eye passes and are replaced by hurricane force winds from a direction nearly the reverse of those previously blowing.

The Beaufort scale is used to classify the intensity of the storms. It estimates the wind velocity by observations of the effects of winds on the ocean surface and familiar objects. Both the United States (Saffir-Simpson Hurricane Scale) (Box 80-2) and Australia (Cyclone Severity Categories) use country-specific scales that estimate potential property damage in five categories. The Philippines recently increased its typhoon warning signal numbers from three ranges of wind speeds to four in order to take into account the lower standards of building structures and regional variations.

Storm Surges. The storm surge, defined as the rise in sea level above the normally predicted astronomic tide, is frequently a key or overriding factor in a tropical storm disaster. As the cyclone approaches the coast, the friction of strong onshore winds on the sea surface, in combination with the "suction effect" of reduced atmospheric pressure, can pile up sea water along a coastline near a cyclone's landfall well above the predicted tide level for that time. In cyclones of moderate intensity, the effect is generally limited to several meters, but exceptionally intense cyclones can cause storm surges up to 8 m (26 ft).

Of the countries experiencing cyclonic storms, those most vulnerable to storm surges are characterized by low-lying land

Box 80-2. Saffir-Simpson Potential Hurricane Damage Scale

The Saffir-Simpson Hurricane Scale is a 1 to 5 rating based on the hurricane's present intensity. This is used to give an estimate of the potential property damage and flooding expected along the coast from a hurricane landfall. Wind speed is the determining factor in the scale, as storm surge values are highly dependent on the slope of the continental shelf and the shape of the coastline, in the landfall region. Note that all winds are using the U.S. 1-minute average.

CATEGORY 1 HURRICANE
Winds 74 to 95 mph (64 to 82 kt or 119 to 153 kph). Storm surge generally 4 to 5 ft above normal. No real damage to building structures. Damage primarily to unanchored mobile homes, shrubbery, and trees. Some damage to poorly constructed signs. Also, some coastal road flooding and minor pier damage.

CATEGORY 2 HURRICANE
Winds 96 to 110 mph (83 to 95 kt or 154 to 177 kph). Storm surge generally 6 to 8 ft above normal. Some roofing material, door, and window damage of buildings. Considerable damage to shrubbery and trees with some trees blown down. Considerable damage to mobile homes, poorly constructed signs, and piers. Coastal and low-lying escape routes flood 2 to 4 hours before arrival of the hurricane center. Small craft in unprotected anchorages break moorings.

CATEGORY 3 HURRICANE
Winds 111 to 130 mph (96 to 113 kt or 178 to 209 kph). Storm surge generally 9 to 12 ft above normal. Some structural damage to small residences and utility buildings with a minor amount of curtainwall failures. Damage to shrubbery and trees with foliage blown off trees and large trees blown down. Mobile homes and poorly constructed signs are destroyed. Low-lying escape routes are cut by rising water 3 to 5 hours before arrival of the center of the hurricane. Flooding near the coast destroys smaller structures with larger structures damaged by battering from floating debris. Terrain continuously lower than 5 ft above mean sea level may be flooded inland 8 miles (13 km) or more. Evacuation of low-lying residences with several blocks of the shoreline may be required.

CATEGORY 4 HURRICANE
Winds 131 to 155 mph (114 to 135 kt or 210 to 249 kph). Storm surge generally 13 to 18 ft above normal. More extensive curtainwall failures with some complete roof structure failures on small residences. Shrubs, trees, and all signs are blown down. Complete destruction of mobile homes. Extensive damage to doors and windows. Low-lying escape routes may be cut by rising water 3 to 5 hours before arrival of the center of the hurricane. Major damage to lower floors of structures near the shore. Terrain lower than 10 ft above sea level may be flooded requiring massive evacuation of residential areas as far inland as 6 miles (10 km).

CATEGORY 5 HURRICANE
Winds greater than 155 mph (135 kt or 249 kph). Storm surge generally greater than 18 ft above normal. Complete roof failure on many residences and industrial buildings. Some complete building failures with small utility buildings blown over or away. All shrubs, trees, and signs blown down. Complete destruction of mobile homes. Severe and extensive window and door damage. Low-lying escape routes are cut by rising water 3 to 5 hours before arrival of the center of the hurricane. Major damage to lower floors of all structures located less than 15 ft above sea level and within 500 yards of the shoreline. Massive evacuation of residential areas on low ground within 5 to 10 miles (8 to 16 km) of the shoreline may be required. Only three category 5 hurricanes have made landfall in the United States since records began: the Labor Day Hurricane of 1935, Hurricane Camille (1969), and Hurricane Andrew in August, 1992.

From The National Oceanic and Atmospheric Administration—National Weather Service, National Hurricane Center, Tropical Prediction Center, Miami, FL. Accessed at http://www.nhc.noaa.gov/aboutsshs.shtml (2006).

along the closed and semi-enclosed bays facing the ocean. These countries include Bangladesh, China, India, Japan, Mexico, the United States, and Australia. Most of the casualties from the 1970 Bhola cyclone occurred from drowning in the storm surge. Prevailing onshore winds and low pressures from winter depressions in nontropical latitudes, as in countries bordering the North Sea, are also subject to storm surges that require substantial mitigation measures, such as dikes.

Rainfall Events. The world's highest rainfall totals over 1 to 2 days have occurred during tropical cyclones. The highest 12- and 24-hour totals, 135 cm (53 inches) and 188 cm (74 inches), respectively, have both occurred during cyclones at La Réunion, an island in the southwestern Indian Ocean. The very high specific humidity condenses into exceptionally large raindrops and giant cumulus clouds, resulting in high precipitation rates. When a cyclone makes landfall, the rain rapidly saturates even dry catchment areas, and rapid runoff may explosively flood the usual water courses as it creates new ones.

The relationship between rainfall and wind speed is not always proportional. For instance, if the atmosphere over land is already saturated with moisture, rainfall will be strongly enhanced, and the cyclone will weaken slowly. If the atmosphere is dry, the rainfall will be greatly reduced, and the cyclone will decay faster. Thus, landfall of even a relatively weak tropical cyclone may result in extensive flooding, as occurred in 2004 with hurricane Jeanne, which had weakened to a tropical storm and dumped torrential rain on Haiti, killing 3,000 people.

Predictability

Tropical cyclones form in all oceans of the world except the South Atlantic and South Pacific east of 140° W longitude.

Figure 80-13. Damage in Grenada from hurricane Ivan, September 2004. (Photo by Sheila Reed.)

Figure 80-14. A man standing on the site of his house in Jamaica, destroyed by hurricane Ivan, September 2004. (Photo by Sheila Reed.)

Nearly one quarter form between 5° and 10° latitude of the equator and two thirds between 10° and 20° latitude. It is rare for a tropical cyclone to form south of 20° to 22° latitude in the Southern Hemisphere; however, they occasionally form as far north as 30° to 32° in the more extensive warmer water of the Northern Hemisphere. They are mainly confined to the warmer 6 months of the year but have occurred in every month in the western North Pacific.

The locations, frequencies, and intensities of tropical cyclones are well known from historical observations and, more recently, from routine satellite monitoring. Tropical cyclones do not follow the same track, except coincidentally over short distances. Some follow linear paths, others recurve in a symmetrical manner, and still others accelerate or slow down and seem stationary for a time. For this reason, predicting when, where, and if a storm will hit land is often difficult, especially with islands. In general, the difficulty in forecasting increases from lower to higher latitudes, whereas the margin of error in determining the cyclone center decreases as landfall approaches.

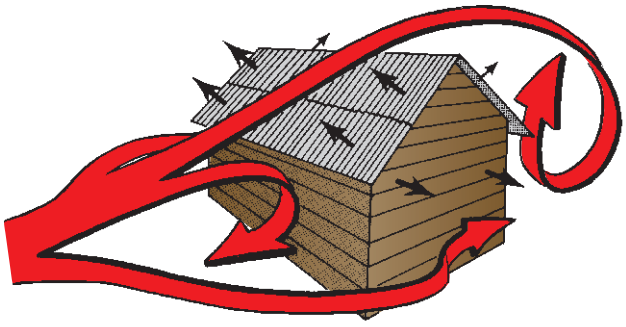
Special warning and preparedness strategies for evacuation from offshore facilities or closure of industrial plants must relate the costs and benefits of those strategies against the uncertainties of precision in the forecasts. For general community purposes that require a minimum 12 hours of preparedness time, the imprecision in forecasting the location of landfall within 24 hours should be generally tolerable, bearing in mind that highly adverse cyclonic weather usually commences about 6 hours before landfall of the cyclone. In September 2004, a category 4 hurricane, Ivan, veered unexpectedly northward and directly hit the Caribbean island of Grenada, home to 90,000 people. The island had not experienced a major hurricane in 40 years. Citizens received warnings in advance but generally did not have adequate preparedness measures in place. Remarkably few (39) died, but 90% of housing was damaged or destroyed (Figs. 80-13 and 80-14).

Regrettably, progress in reducing forecasting errors has remained slow in the last two decades despite huge investments in monitoring systems. However, substantial progress has been made in the organization of warning and dissemination systems, particularly through regional cooperation. The activities of national meteorologic services are coordinated at the international level by the WMO. Forecasts and warnings are prepared within the framework of the WMO's World Weather Watch (WWW) program. Under this program, meteorologic observational data are provided nationally, and data from satellites and information provided by the regional centers are exchanged around the world. The WWW system includes 8500 land stations, 5500 merchant ships, aircraft, special ocean weather ships, automatic weather stations, and meteorologic satellites. A tropical cyclone is first identified and then followed from satellite pictures. A global telecommunications system relays the observations.

Ultimately, however, national services are responsible for providing forecasts and warnings to the local population regarding tropical cyclones and the associated winds, rains, and storm surges. Unfortunately, many of the less developed countries, where most deaths from tropical cyclones occur, do not possess state-of-the-art warning systems.

Vulnerability

Human settlements located in exposed, low-lying coastal areas are vulnerable to the direct effects of a cyclone, such as wind,



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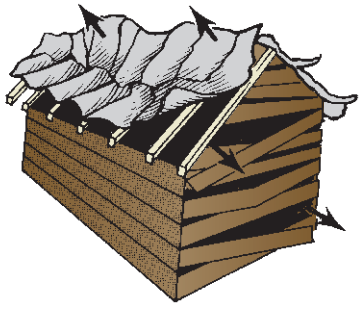

B

Figure 80-15. How high winds damage buildings. **A,** Wind blowing into a building is slowed at the windward face, creating high pressure. The airflow separates as it spills around the building, creating low pressure or suction at the end walls, roof, and leeward walls. **B,** The roof may lift off and the walls blow out if the structure is not specially reinforced. (Courtesy Disaster Management Center, University of Wisconsin.)

rain, and storm surges. Settlements in adjacent areas are vulnerable to floods, mud slides, or landslides from the resultant heavy rains. The death rate is higher where communications systems are poor and warning systems are inadequate.

The quality of structures determines resistance to the effects of the cyclone. Those most vulnerable are lightweight structures with wood frames, older buildings with weakened walls, and houses made of unreinforced concrete block (Fig. 80-15). Infrastructural elements particularly at risk are telephone and telegraph poles, fishing boats, and other maritime industries. Hospitals may be damaged, reducing access to health care and essential drugs.

Typical Adverse Effects

Structures are damaged and destroyed by wind force, through collapse from pressure differentials, and by flooding, storm surge, and landslides. Severe damage can occur to overhead power lines, bridges, embankments, nonweatherproofed buildings, and roofs of most structures. Falling trees, wind-driven rain, and flying debris cause considerable damage.

Casualties and Public Health. Relatively few fatalities occur because of the high winds in cyclonic storms, but many people may be injured and require hospitalization. Storm surges may cause many deaths, but usually few injuries among survivors. Because of flooding and possible contamination of water supplies, malaria organisms and viruses may be prevalent several weeks after the flooding.

Water Supplies. Open wells and other groundwater supplies may be temporarily contaminated by flood waters and storm surges. They are considered contaminated by pathogenic organisms only if dead people or animals are lying in the sources or if sewage is present. Normal water sources may be unavailable for several days.

Crops and Food Supplies. The combination of high winds and heavy rains, even without flooding, can ruin standing crops and tree plantations. Food stocks may be lost or contaminated if the structures in which they were held have been destroyed or inundated. Salt from storm surges may also be deposited on agricultural lands and increase groundwater salinity. Fruit, nut, and lumber trees may be damaged or destroyed by winds, flood, and storm surges. Plantation-type crops, such as bananas, are extremely vulnerable. Erosion can occur from flooding and storm surges. Food shortages may occur until the next harvest. Tree and food crops may be blown down or damaged and must be harvested prematurely.

Communications and Logistics. Communications may be severely disrupted as telephone lines, radio antennas, and satellite dishes are brought down, usually by wind. Roads and railroad lines may be blocked by fallen trees or debris, and aircraft movements may be curtailed for at least 12 to 24 hours after the storm. Modes of transportation, such as trucks, carts, and small boats, may be damaged by wind or flooding. The cumulative effect of all damage is to impede information gathering and transport networks.

Preparedness Measures in India

Tropical cyclones twice struck the same coastal areas of the Indian state of Andhra Pradesh. In 1978, 10,000 people perished. In 1990, despite significant increases in population, fewer than 1000 died. A program of improved monitoring and warning dissemination had been developed by the Indian government. This included use of a domestic satellite, an upgraded cyclone contingency preparedness plan, and enhanced community awareness. Widespread evacuation procedures were initiated by the state counterdisaster committee 48 hours ahead of forecasted landfall. A total of 651,865 people were evacuated from 546 villages to 1098 emergency relief camps by 2019 evacuation teams using 745 transport vehicles. People had also been instructed by the media to go to the camps before the commencement of the cyclonic weather.

Deadly Hurricane Seasons

The 1998 Atlantic hurricane season, from June 1 to November 30, was one of the deadliest in 200 years, killing more than 10,000 people in eight countries and causing billions of dollars in damage. Fourteen named storms, four more than average, formed in the Atlantic Ocean, Caribbean Sea, and the Gulf of Mexico. Of these, 10 became hurricanes. Hurricane Georges followed a path across the U.S. Virgin Islands, Puerto Rico, the Dominican Republic, Haiti, and Cuba, killing more than 500 and causing $5 billion in damages. Hurricane Mitch moved across Central America, killing an estimated 10,000 in

Figure 80-16. Aftermath of hurricane Mitch in Honduras in 1998. (Courtesy Paul Thompson, InterWorks.)

Honduras and wiping out the country's infrastructure (Fig. 80-16). Mitch regenerated as a tropical storm and then passed over southern Florida.

The 2005 Atlantic hurricane season, however, was the most active season on record, lasting into January 2006. Twenty-seven tropical storms formed, of which 15 became hurricanes. Of these, seven were major hurricanes, five becoming category 4 and three reaching category 5. Hurricane Wilma was the most intense ever recorded in the Atlantic. The record damages were over $100 billion and at least 1918 deaths. Hurricanes Dennis, Emily, Katrina, Rita, and Wilma struck Mexico, Cuba, and the U.S. (Florida, Alabama, Louisiana, Texas, and Mississippi). The most catastrophic effects of the season were felt in New Orleans, Louisiana, where hurricane Katrina, a category 5, caused a storm surge that breached levees and flooded most of the city.

TORNADOES

Tornadoes are the most dramatic example of a class of storms, often called severe local storms, which includes thunderstorms and hailstorms. Severe local storms a few miles to a few tens of miles in diameter are often accompanied by unusually strong, gusty winds that can cause severe damage, by heavy local rain that can cause flash floods, and by lightning, hail, and sometimes tornadoes. These intense vortices may be only a few hundred feet in diameter but can contain winds in excess of 300 mph (483 kph), capable of tearing roofs off houses and lifting houses, trees, and vehicles hundreds of feet through the air. Tornadoes have been known to occur in swarms, with as many as several dozen affecting an area of hundreds of thousands of square miles in a single day. A new one-day record was set in the United States on January 21, 1999, when 38 tornadoes were hatched in Arkansas, surpassing the previous record of 20 statewide.

Causal Phenomena

Tornadoes and other severe local storms result from intense, local atmospheric instability, usually caused by solar heating of the earth's surface, which causes intense convective columns. A tornado is a vortex in which air spirals inward and upward. It is frequently, but not always, visible as a funnel cloud hanging part or all of the way from the generating storm to the ground. The upper portion of the funnel consists of water droplets, and the lower portion usually consists of dust and soil being sucked up from the ground. The funnel size may range from a few meters to a few hundred meters in diameter and from 10 meters to several kilometers high. The funnel may undergo changes in appearance during the tornado's lifetime. There may be a single well-defined funnel, multiple funnels, or funnels that appear to consist of several ropelike strands. Tornadoes may be as loud as the roar of a freight train.

Tornadoes are the most violent event associated with thunderstorms. They occur in many parts of the world but are most frequent and fierce in the United States. As many as 1000 may strike the United States each year, mostly in the central plains and southeastern states, although they have occurred in every state, mostly in the spring and summer. Of all the natural hazards in the United States, thunderstorms with associated winds, rain, hail, and lightning rank first in number of deaths, second in number of injuries, and third in property damage.

The most common type of tornado is small and lasts only a minute or two, causing minor damage over a track often less than 300 ft wide and 1 to 2 miles long. Most tornado-related deaths, injuries, and property damage are caused by relatively infrequent, large, and long-lasting tornadoes with paths more than 1 mile wide and more than 100 miles long over several hours.

Predictability

Although conditions favorable to tornado formation can often be predicted a number of hours in advance, the areas in which these conditions are found may cover hundreds of thousands of square miles. It is impossible to predict where individual tornadoes will occur. When a warning is issued, a tornado has already formed, and the threatened population may have only a few minutes to take cover. In the United States, when tornadoes are considered likely within a well-defined region, a tornado watch is issued. When a tornado is actually detected, either visually or on radar, a tornado warning is issued.

Vulnerability

Most injuries from tornadoes are caused by flying or falling debris, usually from destroyed structures. The quality of structures will determine resistance to the effects of the tornado. Those most vulnerable are lightweight structures with wood frames, older buildings with weakened walls, mobile homes, and houses made of unreinforced concrete blocks. Thorough education regarding taking shelter from flying debris is essential to reduce deaths and injuries.

Examples of Tornado Outbreaks

In March 1925, a tornado struck Missouri, Illinois, and Indiana, killing 689 people. On April 11, 1965, an outbreak of at least 37 tornadoes struck Iowa, Wisconsin, Illinois, Indiana, Michigan, and Ohio, killing 271 people, injuring more than 3000, and causing $300 million in damage. On April 3 and 4, 1974, an outbreak of 147 tornadoes struck Illinois, Indiana, Michigan, Ohio, West Virginia, Virginia, Kentucky, Tennessee, North Carolina, South Carolina, Georgia, and Alabama, killing 335 people, injuring more than 5500, affecting more than 27,000 households, and causing more than $600 million in damage. More than half the deaths were caused by fewer than

5% of the tornadoes. The worst of these struck Xenia, Ohio. It cut a swath of destruction half a mile wide and 16 miles long, killed 34 people, injured 1150, and damaged or destroyed 2400 homes. On May 31, 1985, 43 tornadoes struck Ohio, Pennsylvania, New York, and southern Ontario, killing 87 people. The 1999 tornado damage season began early with multiple tornadoes in Arkansas in January. In early May, tornadoes hit Oklahoma and Kansas, killing 43, the largest number in more than a decade.

FLOODS

Throughout history, people have been attracted to the fertile lands of the floodplains, where their lives have been made easier by proximity to sources of food and water. Ironically, the same river or stream that provides sustenance to the surrounding population also renders humans vulnerable to disaster by periodic flooding. Flooding occurs when surface water covers land that is normally dry or when water overflows normal confinements. The most widespread of any hazard, floods can arise from abnormally heavy precipitation, dam failures, rapid snow melts, river blockages, or even burst water mains. However, floods can provide benefits without creating disaster and are necessary to maintain most river ecosystems. They replenish soil fertility, provide water for crop irrigation and fisheries, and contribute seasonal water supplies to support life in arid lands.

Every year in Bangladesh, large tracts of land are submerged during the monsoon season, a normally beneficial process that deposits a rich layer of alluvial soil. The floods originate from three great river systems in the Himalayan mountains: the Ganges, Brahmaputra, and Meghna. In summer 1988, the rivers reached their highest levels in history, and 60% of the land was flooded. At least 1500 people died in the floods, and 49 million were affected by crop loss and damaged homes. In 1998, high sea levels and silting from increased deforestation upriver contributed to massive floods, killing 1500, and causing $2 billion in damages. Farms were inundated and 26,000 cattle perished, destroying livelihoods for millions.

In the aftermath of the flooding, cases of diarrheal diseases reached epidemic proportions, with 50,000 cases reported daily. The risk of other diseases, such as hepatitis, typhoid fever, and measles, was elevated because of contaminated water supplies. Destruction of almost 4 million hectares of crops and partial damage to 3 million hectares left a shortfall in annual grain requirements of 1 million tons and placed the population at risk of famine. In the flood of 1974 in Bangladesh, affecting 50% of the land, 27,500 persons perished from subsequent disease and starvation. Fortunately, timely arrival of food aid averted a famine crisis.

Causal Phenomena

The most important cause of floods is excessive rainfall. Rain may be seasonal and occur over wide areas, or may form localized storms that produce the highest intensity rainfall. Some storms are attributed to atmospheric and oceanic processes, such as the El Niño Southern Oscillation (ENSO) or strong jet streams. Melting snow is another major contributor.

Types

Flash. These are usually defined as floods that occur within 6 hours of the beginning of heavy rainfall. This type of flooding requires rapid localized warnings and immediate response by affected communities if damage is to be mitigated. Flash floods are normally a result of runoff from a torrential downpour, particularly if the catchment slope is unable to absorb and hold a significant part of the water. Other causes of flash floods include dam failure or sudden breakup of ice jams or other river obstructions. Flash floods are potential threats, particularly where the terrain is steep, surface runoff is high, water flows through narrow canyons, and severe rainstorms are likely.

River. River floods are usually caused by precipitation over large catchment areas, by melting of the winter accumulation of snow, or sometimes by both. The floods take place in river systems with tributaries that may drain large geographic areas and encompass many independent river basins. In contrast to flash floods, river floods normally build up slowly, are often seasonal, and may continue for days or weeks. Factors governing the amount of flooding include ground conditions (the amount of moisture in the soil, vegetation cover, depth of snow, cover by impervious urban surfaces such as concrete) and size of the catchment basin (Fig. 80-17).

Coastal. Some flooding is associated with tropical cyclones (also called hurricanes and typhoons). Catastrophic flooding from rainwater is often aggravated by wind-induced storm surges along the coast. Salt water may flood the land by one or a combination of effects from high tides, storm surges, or tsunamis. As in river floods, intense rain falling over a large geographic area will produce extreme flooding in coastal river basins.

Contribution by Humans

Floods are naturally occurring hazards, but can become disasters when they affect human settlements. The magnitude and frequency of flooding often increase because of human actions. Settlement on floodplains contributes to flooding disasters by endangering humans and their assets. However, the economic benefits of living on the floodplain outweigh the dangers for some societies. Population pressure is now so great that people have accepted the risk associated with floods because of the greater need for a place to live. In the United States, billions of dollars have been spent on flood protection programs since 1936. Despite this, the annual flood hazard has become greater because people have built on floodplains faster than engineers can design better flood protection. The Mississippi River, which had been protected by 5600 miles of levees, flooded in 1993, affecting nine states. Some 70% of levees had failed to protect against the record rainfall.

Urbanization contributes to urban flooding. Roads and buildings prevent infiltration of water, so runoff forms artificial streams. The network of drains in urban areas may deliver water and fill natural channels more rapidly than natural drainage routes, or drains may be insufficient and overflow. Natural or artificial channels may become constricted by debris or obstructed by river facilities, impeding drainage and overflowing the catchment areas. Failure to maintain or manage drainage systems, dams, and levees in vulnerable areas also contributes to flooding. Central Europe experienced severe flooding in 2002 and incurred $18 billion in damages, with the cities of Dresden and Prague particularly affected.

Deforestation and removal of root systems increase runoff. Subsequent erosion causes sedimentation in river channels, which decreases their capacity.

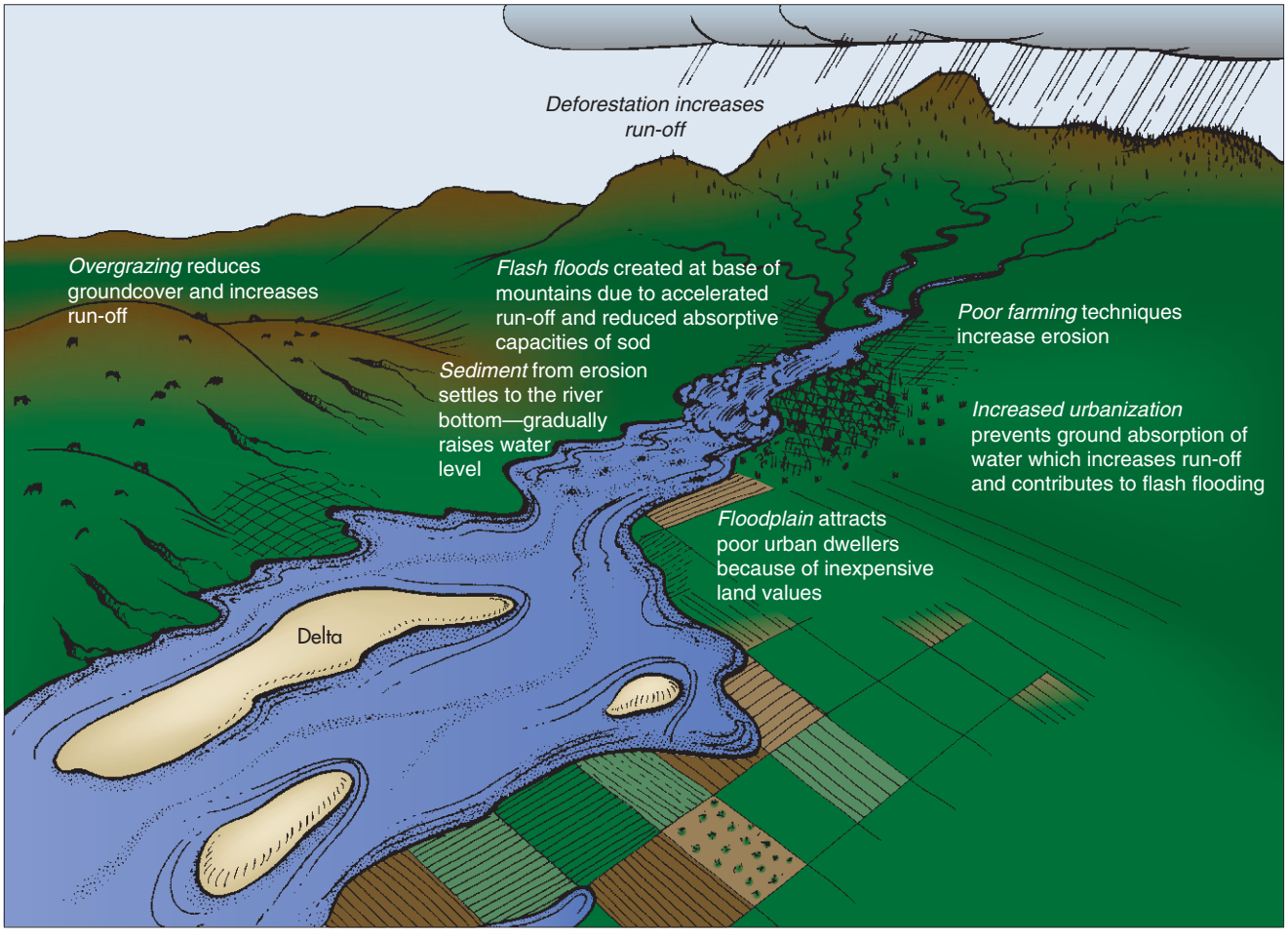

Figure 80-17. Flooding and its causes. (Courtesy Disaster Management Center, University of Wisconsin.)

Predictability

Riverine flood forecasting estimates river level stage, discharge, time of occurrence, and duration of flooding, especially of peak discharge at specific points along river systems. Flooding resulting from precipitation, snowmelt in the catchment system, or upstream flooding is predictable from 12 hours to as much as several weeks ahead of events. Forecasts issued to the public result from regular monitoring of the river heights and rainfall observations. Flash flood warnings, however, depend solely on meteorologic forecasts and knowledge of local geographic conditions. The very short lead time for the development of flash floods does not permit useful monitoring of actual river levels for warning purposes.

For comparison with previous flood events and conversion to warning information, assessment of the following elements should be included: flood frequency analysis, topographic mapping and height contouring around river systems with estimates of water-holding capacity of the catchment area, precipitation and snowmelt records, soil filtration capacity, and, if in a coastal area, tidal records, storm frequency, topography, coastal geography, and breakwater characteristics.

An effective means of monitoring floodplains is through remote sensing techniques. The images produced by satellites can be interpreted to map flooded and flood-prone areas. Other efforts to improve forecasting are being implemented by United Nations organizations, such as the WMO, using WWW and the Global Data Processing System (GDPS). These systems are strategic when flood conditions exist across international boundaries. The great majority of river and flash flood forecasts, however, depend on observations made by national weather services for activation of flood alert warnings.

Vulnerability

At notable risk in floodplain settlements are buildings made of earth or with soluble mortar, buildings with shallow foundations, or buildings that are nonresistant to water force and inundation. Infrastructural elements at particular risk include utilities, such as sewer systems, power and water supplies, and machinery and electronics belonging to industry and communications. Of great concern are food stocks and standing crops, confined livestock, irreplaceable cultural artifacts, and fishing boats and other maritime industries.

Other factors affecting vulnerability are lack of adequate refuge sites above flood levels and accessible routes for reaching those sites. Also, lack of public information about escape routes and other appropriate response activities renders communities more vulnerable. Vietnam, which has 3444 km (2149 miles) of coastline and a complex and ancient system of sea dikes, is chronically vulnerable to floods. The government is promoting a strong institutional network to support citizens to "live with floods," reducing their vulnerability and providing social safety nets to assist with recovery.

Typical Adverse Effects

Structures are damaged by receiving the force of impact of flood waters, floating away on rising waters, becoming inundated, collapsing because of undercutting by scouring or erosion, and being struck by waterborne debris.

Damage is likely to be much greater in valleys than in open, low-lying areas. Flash floods often sweep away everything in their path. In coastal areas, storm surges are destructive both on inward travel and on outward return to the sea. Mud, oil, and other pollutants carried by water are deposited and ruin crops and building contents. Saturation of soils may cause landslides or ground failure.

Casualties and Public Health. Currents of moving or turbulent water can knock down and drown people and animals in relatively shallow depths. Major floods may result in large numbers of deaths from drowning, particularly among young and weak persons, but generally inflict few serious, nonfatal injuries requiring hospital treatment. Slow flooding causes relatively few direct deaths or injuries, but often increases the occurrence of snakebites.

Endemic disease will continue in flooded areas, but little evidence exists of floods directly causing any large-scale additional health problems besides diarrhea, malaria, and other viral outbreaks 8 to 10 weeks after the flood.

Water, Crops, and Food Supplies. Open wells and other groundwater supplies may be contaminated temporarily by debris carried by flood waters or by salt water brought in by storm surges. They are contaminated by pathogenic organisms, however, only if bodies of people or animals are caught in the sources or if sewage is present. Normal sources of water may not be available for several days.

An entire harvest may be lost along with animal fodder, resulting in long-term food shortages. Food stocks may be lost by submersion of crop storage facilities, resulting in immediate food shortages. Grains quickly spoil if saturated with water, even for a short time. Most agricultural losses result from the inundation of crops or stagnation of standing water, as in the 1988 Bangladesh flood.

Large numbers of animals, including draught animals, may be lost if they are not moved to safety. This may reduce the availability of milk and other animal products and services, such as preparation of the land for planting. These losses, in addition to possible loss of farm implements and seed stocks, may hinder future planting efforts.

Floods bring mixed results in terms of their effects on the soil. In some cases, land may be rendered infertile for several years after a flood because of erosion of the topsoil or salt permeation, as in the case of a coastal flood. Heavy silting may have adverse effects or may significantly increase fertility of the soil.

In coastal areas, where fish provide a source of protein, boats and fishing equipment may be lost or damaged.

On the positive side, floods may flush out pollutants in the waterways. Other positive effects include preserving wetlands, recharging groundwater, and maintaining the river ecosystems by providing breeding, nesting, and feeding areas for fish, birds, and wildlife.

DROUGHT

Of all natural disasters, droughts potentially have the greatest economic impact and affect the greatest number of people. They invariably have a direct and significant impact on food production and the overall economy. Due to the slow onset of droughts, their effects may accumulate over time and linger for many years. Their impact may be less obvious than that of other natural hazards but may be spread over a wider geographic area. Because of the pervasive effects of droughts, assessing their impact and planning assistance become more difficult than with other natural hazards.

No universal definition exists for drought. In general, drought is a temporary reduction in water or moisture availability that is significantly below the normal or expected amount for a specified period. Because droughts occur in nearly all regions of the world and have varying characteristics, however, working definitions must be regionally specific and focus on the impacts that result from discrepancies between the supply and demand for water.

Droughts are most often associated with low rainfall and semiarid climate. However, they also occur in areas with normally abundant rainfall. Humans tend to stabilize their activities around the expected moisture environment. Thus, after many years with above-average rainfall, people may perceive the first year of average rainfall as a drought. A rainfall level that meets the needs of a pastoralist may constitute a serious drought for a farmer growing corn. To define drought in a region, it is necessary to understand both the meteorologic characteristics and the human perception of drought.

Types

Meteorologic. Meteorologic drought results from a shortfall in precipitation and is based on the degree of dryness relative to the normal or average amount, and on the duration of the dry period. This comparison must be region specific and may be measured against daily, monthly, seasonal, or annual rainfall amounts.

Hydrologic. Hydrologic drought involves a reduction of water resources, such as streams, groundwater, lakes, and reservoirs. It involves data on availability and offtake rates in relation to the normal operations of the system (domestic, industrial, irrigated agricultural) being supplied. One impact is competition between users for water in these storage systems.

Agricultural. Agricultural drought is the impact of meteorologic and hydrologic droughts on crops and livestock production. It occurs when soil moisture is insufficient to maintain average plant growth and yields. The impact of agricultural drought is difficult to measure because of the complexity of

plant growth and the possible presence of other factors that may reduce yields, such as pests, weeds, low soil fertility, and low crop prices.

Famine drought can be regarded as an extreme form of agricultural drought in which food shortages are so severe that large numbers of people become unhealthy or die. However, famine disasters have complex causes, such as civil war or external conflict, chronic food insecurity, or inflicted food insecurity due to political manipulation. Although scarcity of food is the main factor in a famine, death can result from other complicating influences, such as disease or lack of access to water and other services. Most famine-related deaths occur in the semi-arid areas of sub-Saharan Africa. It might be hoped that the lessons learned in mitigating the famine droughts in Ethiopia and Sudan in 1984–1985, where an estimated 900,000 people died, will prevent such tragedy.

Socioeconomic. Socioeconomic drought correlates the supply and demand of goods and services with the three other types of drought and emphasizes the relationship between drought and human activities. When the supply of some goods or services, such as water, hay, or electric power, is weather-dependent, drought may cause shortages.

Causal Phenomena

The reasons for lack of rain are not well understood. Displacement of the normal path of the jet stream may steer rain-bearing storms elsewhere. Recent research has focused on teleconnection, or linkages to global interactions, between the atmosphere and the oceans. Sea surface temperature anomalies (SSTAs) influence heat and moisture, such that warm surface water may create air conditions favorable for cyclone formation. A large-scale SSTA is linked to ENSO events in the Pacific. These involve the periodic (every 2 to 7 years) invasion of warm surface waters into the normally colder waters off the coast of South America. Droughts of 1982–1983 in Africa, Australia, India, Brazil, and the United States coincided with a major El Niño.

Human causes of drought that include land use practices that give rise to desertification, such as deforestation, overcultivation, overgrazing, and mismanagement of irrigation, are thought to result in greater persistence of drought. Traditional drought coping systems in Africa, such as pastoralists' use of seasonal grazing lands and farmers' use of fallow periods, have been reduced because of population pressures and economic policies (see Desertification).

Droughts vary in terms of intensity, duration, and coverage. Droughts tend to be more severe in drier areas of the world because of low mean annual rainfall and longer duration of dry periods. In dry areas, drought builds up slowly over several years of poor rainfall. Dry conditions in the African Sahel over a 16-year period led to widespread famine in 1984–1985. The quarter century of drought conditions in the Sahel was interrupted by heavy rains in 1994. The area affected by drought in a country has important implications for food security. Larger countries, such as India and Brazil, are rarely completely affected by drought, but smaller countries may be totally affected (Fig. 80-18). Worldwide food availability may be adversely affected by drought in grain-exporting nations.

Predictability

Modern meteorologic monitoring and telecommunications systems can prevent casualties from drought-induced food shortages. The slow onset of drought allows a warning time between the first indications, usually several months, and when the population will be affected. In 1987, satellite imagery and rainfall reports indicated areas within Ethiopia with below-normal moisture and allowed timely intervention to avert a major food shortage. Longer term prediction requires analysis of a century of rainfall data, which do not exist for some parts of the world. The World Meteorological Organization has established a base in Niamey, Niger, to promote regional training on agricultural production and drought response.

Most countries in sub-Saharan Africa have installed famine early warning systems after the 1980s drought. The FAO Global Information and Early Warning System (GIEWS), as well as the USAID-sponsored Famine Early Warning System (FEWSNET), issues regular bulletins on rainfall, food production, and famine vulnerability. These systems rely on satellite remote sensing to detect reduction in vegetation. In addition to the unique vantage point and condensed view, remote sensing provides a permanent historical record. The National Oceanic and Atmospheric Administration (NOAA) satellites provide twice-daily coverage of the planet's surface. These data are available at many receiving stations around the world. NOAA has developed crop-monitoring technology for large areas of the Sahel.

Figure 80-18. Victims of drought in Ethiopia. (Courtesy United Nations.)

Vulnerability

Although drought is more likely in dry areas with limited rainfall, physical factors, such as the moisture retention of soil and timing of rains, influence the degree of crop loss. Dependency on rain-fed agriculture increases vulnerability. Farmers unable to adapt with repeated plantings may experience crop failure. Livestock-dependent populations without adequate grazing territory are also at risk. Farmers dependent on stored water resources or irrigation are more vulnerable to water shortages and may face competition for water.

Drought-related effects are more severe in countries with yearly food deficiencies and in largely subsistence-level farming and pastoralist systems. Food shortages have the greatest impact where malnutrition already exists. Most food shortage–related deaths occur in the semiarid countries of sub-Saharan Africa, whereas in more developed countries the consequences are largely economic. Adverse effects may be more serious where

drought response has not been adequately planned and where assistance measures may be poorly targeted or ineffective.

Typical Adverse Effects

The effects of drought can be grouped as economic, environmental, and social. Economic effects include losses in crops, dairy and livestock, timber, fisheries, national economic growth, and income for farmers and others. Decreased tourism, loss of hydroelectric power and increased energy costs, increased food prices, unemployment, and losses of revenue to governments are other economic effects. Environmental losses include damage to animals, fish, and plant species and habitat; wind and water erosion of soils; reduced water quality or altered salination; and reduced air quality from dust and pollutants. Social effects include food shortage (malnutrition, famine), loss of human life, conflict between water users, health problems from decreased water flow, decline in living conditions, increased poverty, social unrest, and population migration for employment.

WINTER STORMS

Winter storms feature strong winds, extreme cold, ice storms, and heavy snowstorms. These are often deceptive killers because most deaths are indirectly related to the storm, such as from traffic accidents and hypothermia. In the United States, of deaths related to ice and snow, about 70% occur in automobiles, and 25% are people caught out in a storm. The majority are men older than 40 years. For deaths related to cold, 50% are people over 60 years old, more than 75% are males, and about 20% occur in the home.

When temperatures are below freezing, everyone is at potential risk from winter storms. In areas of the world where roads are rarely maintained to mountainous areas, such as in Lebanon, Iraq, and Russia, local populations cope by storing provisions for the winter months. In more heavily populated areas, individual and societal precautions must be taken to avoid the effects of winter storms. The cost of cleaning up after winter storms and loss of business during the storm can have significant economic impact.

Causal Phenomena

Cold air and below-freezing temperatures in the clouds and near the ground are necessary to make snow and ice. Moisture is needed to form clouds and precipitation. The source of moisture may be air blowing across a body of water, such as a large lake or the ocean. Lift, or the required force needed to raise the moist air to form the clouds and cause precipitation, can occur when warm air collides with cold air and is forced to rise over the cold dome. The boundary between the warm and cold air is called a front. Lift might also occur from air flowing up a mountainside.

Strong Winds. Strong winds that sometimes accompany winter storms can create blizzard conditions with blinding wind-driven snow, severe drifting, and dangerous windchill factor. Strong winds with these intense storms and cold fronts can knock down trees, utility poles, and power lines. Storms near the coast can cause coastal flooding and beach erosion, as well as sink ships at sea. Winds descending from mountains can gust to 100 mph (160 kph) or more, damaging roofs and other structures.

The windchill factor is based on the rate of heat loss from exposed skin caused by combined effects of wind and cold. As the wind increases, heat is carried away from the body at an accelerated rate, driving down the body temperature. Animals are also affected by windchill.

Extreme Cold. Extreme cold often accompanies a winter storm or is left in the aftermath. What constitutes extreme cold varies in different areas. For example, in areas unaccustomed to winter weather, cold may be extreme only at the freezing mark. Freezing temperatures can cause severe damage to citrus fruit crops and other vegetation.

Prolonged exposure to cold can cause frostbite (damage to body tissue caused by tissue being frozen) or hypothermia (low body temperature) (see Chapters 5 and 8). Infants and elders are most susceptible.

Ice Storms. Even a small amount of ice poses a significant hazard to motorists and pedestrians. Accumulations of ice can bring down trees, electrical wires, telephone poles and lines, and communication towers. Communication and power can be disrupted for days while utility companies work to repair extensive damage.

Snowstorms. Snow may fall as flurries, showers, squalls, blowing snow, or blizzards, where winds over 35 mph (56 kph) and blowing snow reduce visibility. Sleet (raindrops that freeze into ice pellets before reaching the ground) can accumulate and cause problems. Heavy snow can immobilize a region and paralyze a city. Travelers can be stranded and emergency services disrupted. In rural areas, homes and farms may be cut off for days, and livestock may die if unprotected. The probability of avalanches increases in the mountains.

Predictability

Although winter storm patterns are known in most areas of the world, predicting the intensity and characteristics of winter storms is not an exact science. The effects of El Niño on the winter storm patterns of 1998 are still being debated. Typical U.S. storm patterns include the "Nor'easter," which affects the mid-Atlantic coast to New England from low-pressure areas off the Carolina coast. Research is continuously underway to improve forecasting tools and techniques. For example, the Center for Analysis and Prediction of Storms (CAPS) at the University of Oklahoma provides feedback for the National Weather Service, NASA, and the Department of Defense.

The capacities of most national weather services allow individuals and public services to prepare for winter storms. Winter storm watches and warnings and winter weather advisories are normally issued in most vulnerable areas.

Vulnerability

Lack of a preparedness plan by individuals and communities and lack of understanding of the effects of winter storms increase vulnerability. Failure to heed warnings, lack of communication facilities to receive warnings, and insufficient preparation to cope with the cold or possible isolation from heavy snow can lead to casualties. For example, a person stranded in a vehicle or home during a winter storm without a storm survival kit or without adequate heat, food, or water may become hypothermic or dehydrated. Lack of protection for infrastructure, utilities, and houses can result in damage, loss of service, and roof collapse from heavy snow. Downed trees may later

cause forest fires. Motorists unaccustomed to driving in winter storms cause more accidents. People living in uninsulated or unheated buildings are at greater risk for hypothermia.

The 1998 Ice Storm

A storm of unprecedented impact began on January 5, 1998, and ultimately damaged about 18 million acres of rural and urban forests throughout Maine, New Hampshire, Vermont, upstate New York, and southeastern Canada. The storm severely impacted the dairy industry, maple sugar industry, small businesses, public facilities, and infrastructure. Power outages lasted for up to 23 days. Thousands of people required shelter for an extended period, and nine people died in the United States.

The causal factors of the storm were both natural and human-made. Population and urbanization had recently increased in the area. Cold surface temperatures were overrun by a warm moist tropical air mass, resulting in record rainfall of 2 to 6 inches (5 to 15 cm). Below-freezing temperatures caused the rain to freeze on contact, producing ice accumulations of more than 3 to 4 inches (7 to 10 cm). These factors were intensified by the long duration and significant scope of the storm, resulting in severe flooding and ice damage. Much of the damage could not be assessed until the spring thaw. As a result of the storm, the Federal Emergency Management Agency (FEMA) reviewed the mitigation measures in place and made new recommendations.

ENVIRONMENTAL POLLUTION

The world population, now around 6 billion, is expected to grow another 2 billion by 2030 and reach 8.9 billion by 2050. Despite the pressures placed on natural resources by the expanding population, many poor countries still desperately need the benefits accompanying industrialization and economic growth. In general, people in developing countries are much more vulnerable to the effects of environmental degradation because they are poorer and depend more directly on the land.

Causal Phenomena

Various parts of the environment are subjected to the effects of toxic (poisonous) chemicals produced in manufacturing, such as paint and metal production, and the burning of fossil fuels, such as gasoline, coal, and oil. Some of these chemicals are heavy metals, such as lead, which are essentially nondegradable. Other toxic compounds, such as pesticides, are purposely introduced into the environment. Toxic chemicals may accumulate and affect the quality of air and water. Other pollutants of importance are from biologic sources, such as human wastes, soil sediments, and decaying organic matter.

Air Pollution. Much of the world's urban population breathes polluted air at least part of the time. Sulfur dioxide, a major pollutant, is a corrosive gas harmful to humans and the environment. Electricity generation using fossil fuels is the key source of sulfur dioxide in industrialized countries. In developed countries the burning of fossil fuels also contributes to its creation. Other air pollutants include nitrogen oxides, carbon dioxide (CO_2), and lead, mainly from vehicle exhaust.

Marine Pollution. Sewage is the major cause of ocean pollution. Raw sewage containing human excreta and domestic wastes is disposed of in large quantities directly into the ocean. In the summer of 1993, thousands of ocean beaches were closed in the United States because of high levels of pathogens from human and animal wastes. Industrial effluents are also piped into the ocean. Other pollutants include marine litter, oil spills, and dumped chemical compounds, such as those containing mercury and radioactive substances.

Freshwater Pollution. Human waste and other domestic wastewaters are often discharged directly into nearby bodies of water, particularly in urban areas. In developing countries, this waste may be completely untreated. Industrial effluents from papermaking, chemical, metalworking, textile, and food-processing industries reach bodies of water by direct discharge or by leaching from dumps.

Clearing the land for agriculture and using irrigation, fertilizers, and pesticides have seriously affected water quality in many countries. Unprecedented deforestation has led to soil erosion, causing accelerated runoff and sediment deposits in riverbeds. The sediment level in rivers may increase 100-fold in deforested areas during rainy seasons.

Runoff of nitrogen from fertilizers, particularly in industrialized nations, renders some water unfit to drink without treatment. Use of irrigation systems may lead to increased salinity of water sources and saltwater intrusion on coastal areas where water is withdrawn. Approximately 25% of the world's pesticide production is used in developing countries, mainly on cash crops. Accumulations of pesticide toxins are found in food, soil, and water. Although data on Africa are lacking, studies in Asia indicate that rivers and lakes in Indonesia and Malaysia have very high levels of polychlorinated biphenyls (PCBs) and some pesticides.

Ozone Depletion

Ozone is a form of oxygen composed of three atoms of oxygen. Most atmospheric ozone is concentrated in the upper atmosphere, or stratosphere. The ozonosphere, or the ozone layer, is 11 to 24 km (7 to 15 miles) above the earth. Ozone screens out harmful wavelengths of ultraviolet radiation (UV-B) that originate from the sun, protecting life on Earth (see Chapter 14). Ultraviolet light is associated with increased nonmelanoma skin cancer, ocular cataracts, and deterioration of the retina and cornea. In addition, oceanic phytoplankton are reduced, with damage to fish larvae and young fish. Because fish provide 14% of the animal protein consumed worldwide (60% of that in Japan), the impact could be significant. A hole in the ozone layer has been detected over Antarctica. This hole appears seasonally and is roughly the size of the United States. Thinning of the ozone layer is caused by chlorofluorocarbons (CFCs), chemicals used in refrigeration, foam products, and aerosol propellants. The CFCs that damage the ozone layer may also contribute to global warming. Although they compose a fraction of greenhouse gases, they account for 20% of the warming trend caused by radioactive trapping potential (10,000 times greater than that of CO_2).

Global Warming

Global temperatures appear to be higher today than they have been since 1862, when temperatures were first recorded by instrumentation. Over the past 50 years, the average global temperature has increased at the fastest rate in recorded history; the three hottest years on record have occurred since 1998. One

explanation for this increase is perhaps global warming caused by the greenhouse effect.

The term *greenhouse effect* is used to describe the role of atmospheric gases, such as CO_2, methane, and water vapor, in trapping radiation that would otherwise leave the atmosphere. Without this canopy of gases and clouds, the temperature of the earth would be extremely cold. The atmospheric gases therefore behave similarly to a greenhouse.

Since the beginning of the Industrial Revolution in the late 18th century, CO_2 in the atmosphere has increased by almost 25%, mainly from combustion of coal, oil, natural gas, and gasoline. A strong scientific consensus states that buildup of greenhouse gases is warming the global atmosphere. Computer models used to examine the climatic effects of increasing CO_2 suggest that if it doubles, global temperatures would increase on average by 3° to 5°C (37.4° to 41.0°F).

Trees play a vital role in recycling CO_2 by taking it in, transforming it chemically, storing the carbon, and releasing oxygen into the air. When trees are cut down, left to decay, or burned, they release stored carbon to the air as CO_2. Recently in Central Africa, virgin rainforests were found to have air pollution levels comparable to those in industrial areas. A major cause of this pollution is the smoke from fires that rage for months across huge stretches of land. These fires are set to clear shrubs and trees for the production of crops and grasses. Deforestation has been estimated to account for 20% of total atmospheric content of CO_2. The effects of acid rain (pollutants that are held in the clouds and fall back to Earth in rainwater) and air pollution in Europe, Canada, and the United States also contribute to the increase of CO_2.

Another greenhouse gas is methane. Methane is generated by bacteria as they break down organic matter. It is emitted largely by landfills, cattle, and fermenting rice paddies. The concentration of methane gas in the atmosphere has doubled in the past 200 years, mainly because of expanded animal husbandry and rice cultivation, more landfills, and leaking natural gas pipelines.

Global warming is still a subject of controversy in the scientific community. Both the magnitude and timing of the warming and future climatic changes are uncertain. The status can be summarized as follows:

- Fact: Greenhouse gases are responsible for keeping the planet warmer than it would be otherwise.
- Fact: Concentrations of greenhouse gases are increasing at unprecedented rates.
- Theory: Continued greenhouse gas emissions will lead to global warming.

Characteristics and Typical Adverse Effects

Air Pollution. Pollution of the troposphere (lower atmosphere) is damaging to agricultural crops, forests, aquatic systems, buildings, and human health. Primary pollutants often react to form secondary pollutants (acidic compounds), a frequent cause of environmental damage. The following effects are possible:

1. Crop and vegetation damage by injury to plant tissue, increasing susceptibility to disease and drought
2. Decline in forests caused by leaf damage by acidic compounds, acidic soils, and stresses of multiple pollutants
3. Damage to aquatic ecosystems so that they no longer support life
4. Degradation of building materials, such as metal, stone, and brick
5. Adverse impact on human health by damage to respiratory tracts

Marine Pollution. Marine pollution has the following major effects:

1. Spread of pathogens from human wastes, including viruses and protozoa that cause hepatitis, cholera, typhoid, and other infectious diseases
2. Release of undegradable materials, such as plastics and netting, that may injure marine mammals
3. Oil pollution from oil spills
4. Spread of hazardous chemicals and radioactive substances into the marine ecosystem, where they may accumulate in seafood

Freshwater Pollution. Freshwater pollution results in the following adverse effects:

1. Untreated wastewater carries viruses and bacteria from human feces into human drinking water, which can result in illness or even in infant mortality
2. Eutrophication, or decay of organic matter, which decreases oxygen levels in water, upsetting the balance of the aquatic ecosystem
3. Adverse health effects in persons drinking untreated water from tainted sources
4. Water acidification, which reduces water's capacity to support aquatic life
5. Runoff sediment from eroded soil deposits in drainage basins, reducing the basins' capacity and exacerbating flooding
6. Salinization from irrigation, with harmful effects on downstream agriculture
7. Pesticides and fertilizer chemicals, which accumulate in water and affect tissues in living organisms

Global Warming. The impacts of global warming are still uncertain. Computer models are unable to make reliable predictions of regional changes. Theoretically, the changes could lead to increase in disasters, including those caused by drought, floods, tropical cyclones, and tornados. The following changes may occur.

Rise in Sea Level. Melting of the Arctic ice sheets and alpine glaciers could cause the seas to expand and sea levels to rise. Depending on the degree of global warming, the seas may rise 30 cm to 2 m (12 inches to 7 ft) by 2075, jeopardizing coastal settlements and marine ecosystems. A rise of 1 m ($3^1/_3$ ft) in sea level could flood 15% of arable land in Egypt's Nile Delta and would flood 12% of Bangladesh, displacing 11 million people. The tiny island of the Maldives, inhabited by 200,000, would be submerged.

Climate Change. Natural disasters such as superhurricanes could become common. A temperature increase of a few degrees in tropical seas can intensify hurricane production. The warmer oceans may increase the El Niño phenomenon near the coast of Peru. ENSO inhibits phytoplankton growth, causes fish and shellfish to migrate or die, and forces higher forms of life (e.g., birds, humans) dependent on this sea life to migrate or die.

Other climatic changes could lead to warmer and drier conditions in middle latitudes, higher temperatures in semitropical

and tropical areas, and higher rates of evaporation. Rainfall patterns may also change. The combined effects of increased CO_2 and climate changes may alter plant and animal productivity. Plants may grow faster and larger but may have reduced nutritive value.

Changes in Ecosystems. In warmer climates, grasslands, savannas, and deserts may expand, rendering them vulnerable to increased degradation through erosion and fire. Animal species that do not adapt to the temperature increases may have to relocate to survive, which would be difficult, given population pressures on land. Plant species unable to adapt would perish.

Public Health Impact. Global warming may affect mortality because of heat stress and may increase the incidence of respiratory diseases, allergies, and reproductive illnesses. Geographic ranges of vector-induced diseases (e.g., mosquito-borne malaria, yellow fever) and parasitic diseases might increase.

Measurement

Air and Water Pollution. Pollutants are measured worldwide, but to a much lesser degree in developing countries. The most comprehensive data collection system is the Global Environment Monitoring System (GEMS) of the United Nations Environmental Programme (UNEP), which provides data on sulfur dioxide and particulate matter in urban air and contaminants in water resources. Pollution production is related to per capita consumption, so that as countries develop, pollution also tends to increase.

Ozone Depletion. Ozone levels are regularly monitored each year, especially in the Southern Hemisphere, where a seasonal ozone hole opens over Antarctica every year. Twenty million tons of CFCs were manufactured and have either escaped or will escape to the atmosphere. An international agreement to reduce ozone depletion, the 1987 Montreal Protocol, was ratified by 170 countries and has successfully decreased the ozone depleting chemicals in the lower atmosphere.

Greenhouse Effect. Greenhouse gas emissions are regularly measured throughout the world. Even if the exact levels of future greenhouse emissions were available, however, predicting the effects on global climate would be difficult. Climatic models are used to study climate change, but the models differ in their interpretation of the various interactions in the earth's systems, partly because information put into the system is incomplete and linkages between variables are poorly understood.

Risk Reduction Measures

Air and Water Pollution. Most nations are acting individually to control air pollution. However, since the Chernobyl nuclear power plant accident, transboundary pollution has been recognized as an environmental hazard necessitating a multinational approach. Basic requirements are to set ambient air quality standards that measure pollutants away from the source and set controls on acceptable levels, and to require that every source of an air pollutant meet certain emission limits. In some cases, humans may have to develop the technologies to make the latter possible.

Pollution control of coastal areas in the past has proved that recovery is possible to some extent. The banned pesticide DDT, which was present in many forms of marine life, is now decreasing in concentration. Most strategies for protecting the oceans must address broader ranges of pollutants from sewage to industrial effluents. More national and international efforts should focus on establishing policy for protection of coastal areas.

Improvement of soils can decrease the possibility of water contamination by toxic chemicals and decrease runoff, thereby lessening silting and sedimentation of waterways. Establishing terraces and contour bounds, stabilizing sand dunes, building check dams, and planting trees and shrubs can help to stabilize soil. Watershed mapping, management, and protection are also of vital importance in ensuring a safe and plentiful drinking water supply. Proper systems to dispose of human waste should be promoted.

Regulations must be established and enforced by government agencies to protect citizens against the toxic effects of pesticides and other chemicals. Improvement of soils will also help to absorb and degrade toxins. Further studies must be made on the effects of pesticide residues. Farmers may use crop types resistant to pests or an integrated approach to pest management requiring less pesticide.

Ozone Depletion. International cooperation to limit CFC emissions should reduce production and use of CFCs in industrialized nations from 1986 levels by 50%, with developing countries allowed to increase their use slightly. Research is addressing the need for CFC substitutes, for minimizing loss to the atmosphere, and for recycling. Countries can regulate import and use of aerosols and disposal of refrigeration units.

Global Warming. Because burning of fossil fuels (at least in theory) is the primary cause of global warming, developed countries are mainly at fault, and poorer countries are more likely to be the victims. However, scientists estimate that 20% of greenhouse gases (mainly CO_2) are generated by deforestation, a trend occurring at a devastating rate in developing countries, particularly in tropical rainforests. Because global warming could affect the entire planet, steps can be taken to prepare for its effects and to prevent its acceleration, as follows:

1. Reduce the rate of deforestation. Plant trees to solve community needs for wood, such as fuel wood, or to provide profits for individual farmers with agroforestry.
2. Increase energy production and use. Promote energy efficiency in urban areas, and support renewable energy sources, such as wind, water, geo-thermal, and solar power. These may be of great use in areas where no electricity sources exist.
3. Develop regulations to curb pollution from traffic emissions and industry in urban areas.

Education is a vital tool for environmental awareness. By understanding the relationships of ecosystems and the long-term effects of degradation, people are motivated to act. Women's groups in India have established a tree protection lobby. Their motto is "trees are not wood," a concept that promotes trees as a vital part of the ecosystem, involving CO_2 exchange in the air and a root system to hold down the soil. Education regarding the environment should begin in the early grades. Education for adults may take place in farmers' cooperatives, women's cooperatives, and village settings or may accompany programs to distribute seeds and tools.

Trying to Save the Black Sea

The Black Sea, named for the dark clouds and fierce storms that affect its shores each autumn, faces an even darker future. The residues of modern agriculture and industry now threaten its marine life and the air quality of its bordering countries of Bulgaria, Georgia, Romania, the Russian Federation, Turkey, and Ukraine. The Black Sea is particularly vulnerable to pollution, because it collects 10 times more water per square meter of surface area than any other sea or ocean. It is fed by several major rivers, which deposit many of the pollutants. The most important is the Danube, which flows through eight highly industrialized countries, all using chemically intensive agricultural practices. Other threats to the sea include insufficiently treated sewage and inputs of harmful substances, such as oil and exotic species from sea vessels.

In addition, the Black Sea has natural pollutants—organic matter collected over thousands of years, now decaying and diminishing the supply of oxygen in the water. In the unique two-stratum structure of the sea, where salt water from the Mediterranean forms a bottom layer and fresh water a top layer, toxic hydrogen sulfide from the decomposing matter remains on the bottom layer, where oxygen is not present. Construction of irrigation works and dams has reduced the flow of fresh water into the Black Sea, so the toxic layer that was previously 200 m (656 ft) below the surface has now risen to a depth of only 80 to 100 m (262 to 328 ft).

Further deterioration of the Black Sea and air pollution from industries around it could be economically disastrous to the surrounding countries that depend on it to draw tourists. Acting on the mandate of the six Black Sea countries, The Black Sea Commission, in 1992, ratified a Convention on the Protection of the Black Sea Against Pollution. A strategic action plan was developed in 2002. The plan sets out measures to conduct environmental impact assessments and audits, promote environmentally sound technologies and public involvement in environmental decision making, and promote green tourism and sustainable livelihoods.

DEFORESTATION

Deforestation is removal or damage of vegetation in a region that is predominantly tree covered. Deforestation is a slow-onset hazard that may contribute to disasters caused by flooding, landslides, and drought. Deforestation reaches critical proportions when large areas of vegetation are removed or damaged, harming the land's protective and regenerative properties. The rapid rate of deforestation in some parts of the world is a driving force in the yearly increase of flood disasters in these areas.

During the 1990s, the FAO reported that the loss of natural forests averaged 14.6 million hectares per year, minus 5.2 million hectares of afforested and expanded forest areas, for a total of 9.4 million deforested hectares. This corresponds to annual losses of 0.4 percent globally and 0.8 percent in the tropics. Deforestation was highest in Africa and South America. The countries with the highest net loss of forest area between 1990 and 2000 were Argentina, Brazil, the Democratic Republic of Congo, Indonesia, Myanmar, Mexico, Nigeria, the Sudan, Zambia, and Zimbabwe. Those with the highest net gain of forest area during this period were China, Belarus, Kazakhstan, the Russian Federation, and the United States. Plantations contributed to the gain in forest area, with 1.9 million hectares of new plantations per year in tropical countries and 1.2 million hectares in nontropical areas.

Although the amount of forest land coming under protection or conservation is growing, the future still poses problems because of rapidly increasing pressures of development and exploitation. Forests provide subsistence and income to close to 1.6 billion people; thus, the alarming rate of deforestation warrants international attention.

Causal Phenomena

The principle causes for loss and degradation of forests are conversion to other land uses (mainly agriculture and grazing) and overexploitation of forest products (industrial wood, fuelwood).

Beneath the obvious causes are fundamental problems in development, such as the use of inefficient agricultural practices such as overgrazing, insecure land tenure, rising unemployment, rapid population growth, and failure to regulate and preserve forest lands. Contributing factors are air pollution and storms, and pests and diseases. A significant contributing cause in the 90s were wildfires that occurred in the western United States, Ethiopia, the western Mediterranean, and Indonesia.

Conversion to Agriculture. The major cause of forest loss is the spread of farming. Land may be cleared for commercial ventures such as sugarcane, coffee, or rubber plantations, which is a principal cause of deforestation in Central America. In tropical rainforests, both legal and illegal colonists are trying to farm the former jungle lands, where soil conditions are fragile. Up to 90% of the nutrients are in vegetation rather than in the soil. When the forest is cut and burned, a nutrient surge occurs in the soil, lending initial fertility. After cropping and exposure to sun and rain, however, soil fertility rapidly declines, and the area becomes unproductive, perhaps prompting the farmer to slash and burn new forest areas.

Many traditional people in the Amazon Basin, Central Africa, and Southeast Asia still practice shifting cultivation techniques, allowing fallow periods between cropping for soils to regenerate. This practice becomes unsustainable if populations increase to the extent of forcing people into smaller areas. Insecure land tenure or fixed land titles may also force overuse of the land.

Because of crowded conditions in cities and farm areas, many people migrate to areas of marginal fertility, where they must keep moving their fields to produce sufficient food. Where this occurs, the migrant farmer may damage timber, wildlife, and human resources. In Venezuela, which has a high rate of unemployment and rising numbers of landless peasants, 30,000 families live and farm in national parks, forest reserves, and other legally protected areas. An influx of cultivators who settled on the watershed above the Panama Canal has caused increased silting of a major reservoir that supplies Panama City.

In Central and South America, large areas of tropical forest have been cleared to create grazing lands. A major portion of this can be attributed to economic enterprises designed for meat production. The Brazilian government has granted large land concessions to both domestic and foreign corporations wanting to raise cattle in the Amazon area. In Central America, virgin forest is being destroyed by ranchers who intend to export beef to the United States.

Overexploitation of Forest Products. Extensive logging in humid tropical forests, particularly in Asia and in temperate and mountainous forests, is conducted by large multinational corporations for export or to fill building needs in cities. The procedure usually involves either "clear cutting" or "creaming" (selective logging) of the forest's small proportion of valued species. Creaming, even though a less radical alternative to clear cutting, causes significant damage to vegetation and wildlife that is not apparent from statistics. A study in Indonesia revealed that logging operations damaged or destroyed about 40% of trees left behind. The roads created by logging operations may encourage settlers to enter the forest and begin slash and burn agriculture, so that eventually even more of the forest is lost.

Firewood collection can contribute to the depletion of tree cover, particularly in lightly wooded areas. Because of a lack of alternative fuels and fuel-efficient stoves, this is especially a problem in Africa and in Asian highland countries such as Nepal. In areas of dense woods, dead material may fill local requirements for fuel. The outright destruction of trees for fuel occurs most commonly around cities and towns, where commercial markets for firewood and charcoal exist. Well-organized groups and individuals bring fuel wood by vehicle, pack animal, and cart into many cities, hastening local deforestation.

Fuel Wood Crisis. About 100 million people in developing countries cannot meet their minimum needs for energy, and almost 1.3 billion consume fuel wood resources faster than they are being replenished. In parts of West Africa today, some urban families spend one fourth of their income on wood or charcoal for cooking. In India, firewood is subsidized for the poor to prevent starvation.

Characteristics

Trees play a vital role in regulating the earth's atmosphere, ecosystems, and weather systems. They recycle CO_2, a gas now increasing in the atmosphere and thought to contribute to global warming. They release moisture to the air, thus contributing to rainfall and moderating local and global climate. Their roots trap nutrients, improve soil fertility, and trap pollutants, keeping these from the water supply. They provide habitats for species, engendering diversity. They nurture traditional cultures by giving shelter, wood, food, and medicinal products. These benefits are lost as trees are destroyed.

The root systems of vegetation help retain water in the soil, anchor the soil particles, and provide aeration to keep soil from compacting. When vegetation dies, the nutrients go back to the soil. When root systems are removed, soil becomes destabilized. Water tends to flow off the top of the soil instead of percolating in, and it carries valuable topsoil along with it. This soil eventually forms sediment in the drainage basins.

Deforestation poses the most immediate danger by its contribution to the following hazards:

1. Destabilized soils are more susceptible to landslides and may increase the landslide risk in areas vulnerable to earthquakes and volcanoes.
2. Loss of moisture from deforestation may contribute to drought conditions, which in turn may trigger famines. Soil nutrients may also be lost through erosion of topsoil, resulting in decreased food production and possible chronic food shortages.
3. Erosion and dry conditions, combined with loss of vegetation and soil compaction, result in desertification and unproductive lands.
4. Dryness may accelerate the spread of fires.
5. Loss of CO_2 from dying trees and fires may add to global warming.
6. Deforestation of watersheds, especially around smaller rivers and streams, can increase the severity of flooding, reduce stream flows, evaporate springs in dry seasons, and increase the amount of sediment entering waterways.

Of all the hazards associated with deforestation, flooding may be the most serious. Usually, curative measures, such as dredging and dam building, are taken rather than preventive measures to solve flooding problems. As flooding worsens in developing countries, more attention is given to protection of watersheds. In India, flood damages between 1953 and 1978 averaged $250 million per year. Today, even more people live in flood-prone areas. Flood problems may not be lessened without reforestation of the increasingly denuded hills of northern India and Nepal.

Predictability

Measurement and monitoring of forested areas may be conducted through ground-level sampling and aerial or satellite surveys. Each method has drawbacks. Ground sampling is tedious and difficult to extrapolate, aerial surveys are expensive, and satellite imagery poses difficulty in distinguishing forest from other vegetation. Combinations of methods usually produce the best results. Vague definitions in the study of deforestation still make exact determinations and forecasting difficult. Three different prediction methods follow:

1. One type of study predicts future deforestation rates by extrapolating present rates of deforestation into the future. If the present rate of deforestation at 6.1 million hectares per year were to continue, the tropical moist forests would be completely cleared in 100 years. Where deforestation is more acute, the losses will be more serious. Cote d'Ivoire and Nigeria annually lose about 5.2% of their forests, and Costa Rica, Sri Lanka, and El Salvador lose between 3.2% and 3.6%. Each of these countries could lose all forests between the years 2007 and 2017.
2. Another forecast for 43 tropical countries was made using a mathematical model, which assumed that when forests in a country fell to a critical level, governments would take action to prevent further deforestation. Considerably more optimistic, the results predicted deforestation rates to decrease to between 0.9 and 3.7 million hectares per year in 2030.
3. Perhaps the bleakest theoretical forecast incorporates the effects of population growth and increasing consumption, which might be assumed to increase the rate of deforestation worldwide. However, growth of economies and technologies at the same time may assist to curtail the deforestation process if governments take appropriate action.

Typical Adverse Effects

The specific impacts of deforestation include the following:

1. Loss of soil fertility in the tropics and loss of productive capacity
2. Soil erosion and deposition of sediment
3. Increased runoff
4. Reduction in rainfall and increase in temperature
5. Destruction of biodiversity and traditional cultures

6. Loss of "free" goods, such as fuels, food, and medicines
7. Exacerbation of other disasters

Economic Impact. Most developing countries are already importers of forest products, especially paper. Because the amount of wood and wood products available per person in the world is falling and thus increasing in price, combined with shortages of foreign currencies, import of forest products may be increasingly prohibitive for these countries. Commercially marketed firewood is becoming more scarce, and prices are climbing. Wood for construction is also scarce in many countries, which adversely affects the availability of housing.

Risk Reduction Measures

Various types of forest management, reforestation, and community participation can reduce deforestation. Most governments now recognize the vital importance of national forestry programs. Foresters help people meet their basic needs for forest products, and not always from the traditional forest or concentrated woodlot. For instance, forestry practiced by many farmers on their lands has been shown to be more environmentally effective. Reforestation has become intrinsically interwoven with other government policies that affect the population. Forestry therefore should be considered an integral part of land use and natural resource planning sectors of government.

Forests should be viewed by governments as capital resources to be managed. Management of the system should discourage concessionaires or other land users from practices that are not sustainable. Good management encourages highly selective harvesting without undue waste of remaining trees, especially in tropical forests. The involvement of communities in forest management is now a significant feature of national forest policies throughout the world. Forest management policies must consider the need to protect forests in conflict areas and to avoid exacerbating conflict over forest resources. For any country to address its loss of forests and ensure that forests will yield economic benefits well into the future, the following steps must be taken:

1. Forest law or basic forest policy must be written that clearly states the objective of long-term sustainable management of the forest.
2. Forest regulations or management guidelines must be written and followed.
3. Sufficient financial and human resources must be allocated.

Forest management must be considered in the broadest sense of land use planning to include solutions for people as well as for trees. Compromises between complete destruction of the forests and complete conservation might entail regulated clearing of forests for shifting cultivation, habitation, or hunting; voluntary and intentional protection of forests or individual species by designating areas for reserves or national parks; and enrichment of the forest with species from other places. The last option may be considered risky, since pests and other species-specific problems may accompany introduction of new species.

Many unresolved scientific issues in forest management remain. How can the ever-expanding areas of secondary vegetation and degraded soils be managed to be more productive for the local people? Because most primary forests have disappeared, what type of forest can be established that would be stable and productive and that would ensure the conservation of biologic diversity? What further types of basic ecologic research are needed to manage natural forests?

DESERTIFICATION

Desertification is defined as land degradation in arid, semiarid, and dry subhumid areas resulting mainly from human actions. Poor land use is a significant contributing factor, but desertification can also be caused by natural cycles of climate change. It affects both developed and developing regions, including Africa, the Middle East, India, Pakistan, China, Australia, Eastern Europe, Central Asia, the central and southwestern United States, and many Mediterranean countries. A slow-onset disaster, desertification worsens conditions of poverty, brings malnutrition and disease, and destabilizes the social and economic bases of affected countries.

Causal Phenomena

Role of Climate. Vulnerability to desertification and the severity of its impact are partially governed by climatic conditions of an area. The lower and more uncertain the rainfall, the greater is the potential for desertification. Other influencing factors are seasonal patterns of rainfall and high temperatures that increase evaporation, land use, and the type of vegetation cover.

The world's drylands are found in two belts centered approximately on the Tropic of Cancer and the Tropic of Capricorn (23.5 degrees north and south of the equator, respectively) and cover one third of the earth's surface. More than 80% of the total area of drylands is found on three continents: Africa (37%), Asia (33%), and Australia (14%). The drylands can be further classified into hyperarid, arid, and semiarid zones, depending on the average amount of rainfall received per year. Other factors, such as temperature and soil conditions, must be considered when determining the dryness ratio.

Both natural and human-derived climatic changes may contribute to desertification. Natural effects, such as long-term climatic cycles and the basic Earth-sun geometry, have resulted in drier conditions in the Sahara. Human influence is associated with the predicted global warming trend and local climatic changes, in which deforestation has reduced the moisture-holding capacity of soil and has decreased cloud formation. The result is less rainfall and higher temperatures.

Despite the common misperception that desertification is caused by the desert advancing itself, land degradation can occur at great distances from deserts. Desertification usually begins as a spot on the land where land abuse has been excessive; from that spot, land degradation can spread outward with continued abuse (Fig. 80-19). Desertification does not cause drought but may result in greater persistence of or susceptibility to drought. Drought, on the other hand, contributes to desertification and increases the rate of degradation. When the rains return, however, well-managed lands recover from droughts with minimal adverse effects. Land abuse during periods of good rains and its continuation during periods of deficient rainfall contribute to desertification.

Role of Land Use Management. Desertification can be caused by five main types of poor land use: overcultivation, cash cropping, overgrazing, deforestation, and poor irrigation practices.

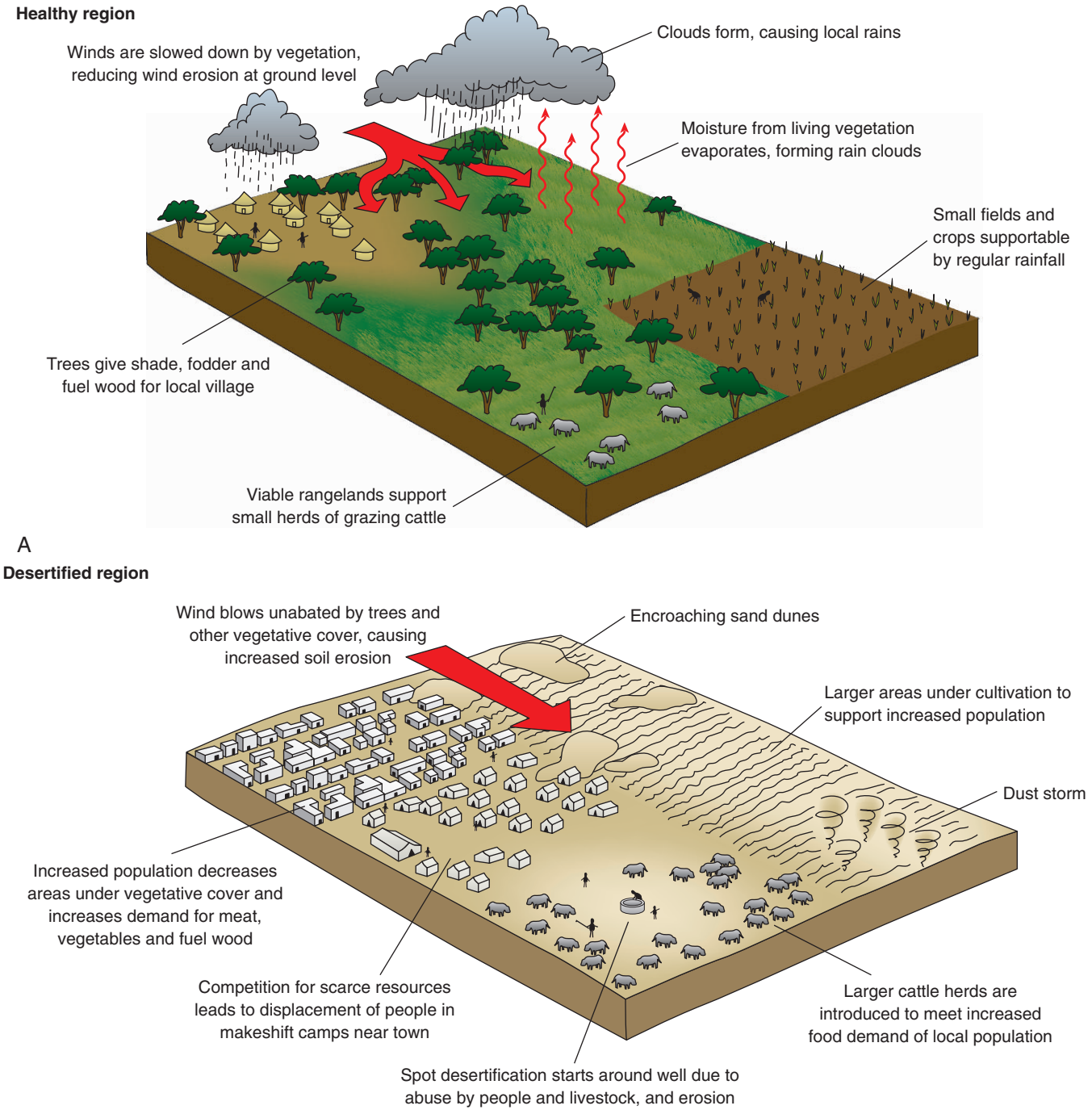

Figure 80-19. Comparison of healthy and desertified regions. (Modified from Introduction to Hazards, 3rd ed. New York, Disaster Management Training Programme, United Nations Development Program, UN Organization for Coordination of Humanitarian Assistance, 1997.)

Overcultivation. Overcultivation damages the structure of the soil or removes vegetation cover, leaving the soil vulnerable to erosion. Reasons for overcultivation include drought, increasing demand for food because of population growth, cropping on marginal range lands unsuitable for long-term production, land tenure restrictions confining sectors of the population to marginal lands, mechanized farming, and expansion of cash cropping.

Cash Cropping. Although a large part of agricultural production in developing countries fills subsistence needs, some cash crops are grown for foreign exchange. A feature of most cash crops, however, is their extreme demand for nutrients and optimum conditions. Degradation of land occurs directly through improper management of such crops and indirectly by displacing subsistence crops and pastoralism to marginal lands.

Overgrazing. Overgrazing is a major cause of desertification (range lands account for 90% of desertified lands) and occurs when too many animals are pastured. The number of cattle in Niger, for example, increased an estimated 450% between 1938 and 1961 and an additional 29% by 1970, when the majority were killed by starvation. Livestock density increases when herd sizes grow too large in wet years and cannot be sustained in dry years. Lucrative markets for meat, such as Nigeria and the Middle East, have resulted in cattle ranches, where concentrated activity threatens land, with poor returns for the investment.

Better veterinary care has decreased mortality rates. Deep wells increase availability of water, allowing larger, less mobile herds that congregate in the well area and degrade the vegetation and soil.

Deforestation. Land is cleared for agriculture, livestock, and fuel wood production. Deforestation is the first step toward desertification, removing vegetative barriers and exposing land to sun, wind, and rain. In Africa, demands for fuel wood and charcoal exert considerable stress on wood resources.

Poor Irrigation Management. The concept of using irrigation to ward off the threat of crop failure during drought has been promoted by many development agencies. Ironically, poor management of irrigation projects has been a cause of desertification. In some cases, productivity falls and soils become salinized, alkalized, or waterlogged. The major problem is usually inadequate drainage, and damage may be irreversible. A key example is the Greater Mussayeb irrigation project in Iraq, begun in 1953. By 1969, waterlogging was widespread, and two thirds of the soil was saline. In 1970, a project to reclaim the salinized land was begun, but by 1976, because of technical and organizational limitations, the project still had not been successful. Egypt, Iraq, and Pakistan have lost more than 25% of their irrigated areas to salinization and waterlogging.

Role of Government Policy. Population growth and economic expansion also contribute to desertification. As populations grow, government and multilateral policies must promote increased food production through appropriate technologies that prevent soil degradation and erosion. Government policy should also address the causes of poverty, or disadvantaged peoples will place more stress on land to obtain needed resources. Some governments choose to expand cash crop cultivation to improve foreign currency holdings rather than promote food security for the poor, or they fail to resolve conflicts over scarce resources. Policies may mandate land uses that are difficult to enforce and that result in breakdown of customary land tenure or natural resource management institutions.

Characteristics

The two main characteristics of desertification, the degradation of soil and degradation of vegetation, have the same result: reduction of productivity.

Degradation of Vegetation. Vegetation in arid lands adapts to the cycle of water availability by adjusting its growth. The drier the area, the farther apart plants grow. Some plants grow only during the rainy seasons. Degradation of vegetation occurs initially in the early stages of desertification with deforestation but continues after soil fertility declines.

The two main forms of vegetation degradation are overall reduction of density of the vegetation cover, or biomass, and a more subtle change in types of vegetation to less productive forms. For example, range land perennial grasses may be replaced by less palatable annual varieties, or more saline-tolerant crops, such as barley, may be substituted for traditional crops because of low yields from waterlogging and salinization.

Degradation of Soil. Soil degradation occurs in four major ways: water erosion, wind erosion, soil compaction, and waterlogging, which result in salinization and alkalinization.

Water Erosion. Vegetation normally protects soil from being washed away by rain and also from splash erosion by raindrops. The raindrops move the soil particles and pack them together on the surface, sealing the pores and thereby decreasing infiltration and increasing runoff. Sheet erosion is a more serious form of erosion in which fine layers of topsoil carrying soil nutrients are washed away. Unless the nutrients are replenished artificially, crop yields decline. Gullies are created by the runoff and, unless reclaimed by conservation measures, render the land unusable.

Wind Erosion. Wind erosion occurs when finer components of the soil, such as silt, clay, and organic matter that contain most of the nutrients, are blown away, leaving behind the less fertile sand and coarse particles. Sand itself may start to drift, forming dunes, but this accounts for a minor proportion of the effects of wind erosion. Strong winds may form dust storms that damage crops by shredding leaves.

Soil Compaction. Nearly complete compaction can occur when soil of poor structure is compressed by heavy machinery or by hooves of large herds of animals. A less serious form of compaction, called *surface crusting,* results when high-speed mechanical cultivation or dry season cultivation turns particles into thin powder, which then forms a crust when pelted with raindrops. Crusting and compaction make the soil less permeable for germination of new plants.

Waterlogging (Salinization and Alkalinization). These effects result from poor management of irrigation and water supplies in general. When the soil is waterlogged, the upward movement

of saline in groundwater leaves salt on the surface when the water evaporates.

Predictability

Desertification is a direct socioeconomic threat to more than 200 million people and a less direct threat to 700 million, but data are still insufficient to quantify the extent of the problem and its progression. It leads to estimated losses in agricultural production of $42 billion. Databases are incomplete or do not exist for many countries. More information is needed on the characteristics and status of dryland ecosystems. An understanding of climatic changes, including the effects of possible global warming, is crucial to predictability. Socioeconomic indicators showing trends in human health, income, and welfare must be collected to understand related issues.

The International Soil Reference Centre and UNEP support the Global Assessment of Soil Degradation (GLASOD), which uses a geographical information system (GIS) to access data for different areas of the world and estimate land degradation.

Rate and Scope

Although the numbers remain controversial, yearly losses to desertification are estimated at 1.0 to 1.3 million hectares of irrigated land, 3.5 to 4.0 million hectares of rainfed cropland and 4.5 to 5.8 million hectares of range land. These estimates represent an increase from estimates in 1994 of 3.4% and indicate that the situation is worsening. The area of desertification is thought to be increasing at about 60,000 square kilometers per year.

Desertification affects drylands in more than 110 countries but is concentrated in Asia and Africa, which together account for 70% of all desertified land. Scientists have tried to quantify the areas desertified on a worldwide basis. The physical damage can be measured in reduced productivity of soils and loss of vegetation. The number of casualties cannot be scientifically extrapolated, but deaths occur, directly from famine or indirectly from reduced standards of living.

ACKNOWLEDGMENTS AND RESOURCES

This chapter builds on the original teaching concept developed in *Introduction to Hazards,* Third Edition (1997), a module prepared for the United Nations Development Programme's Disaster Management Training Programme (UNDP-DMTP) and UN Organization for Coordination of Humanitarian Assistance (OCHA) by InterWorks of Madison, Wisconsin. Another background document is *Natural Hazards: Causes and Effects,* published by the Disaster Management Center of the University of Wisconsin.

Websites and e-mail addresses for more information on hazards include the following:

Federal Emergency Management Agency: www.fema.gov
Global Information and Early Warning System on Food and Agriculture: GIEWS@fao.org
Hazlit Data Base, University of Colorado: www.colorado.edu/hazard/litbase
International Tsunami Information Center: itic@itic.noaa.gov
National Earthquake Information Center: quake@gldfs.urgs.gov
National Oceanic and Atmospheric Association: www.noaa.gov.
United Nations Office for Coordination of Humanitarian Affairs, Relief Web: www.reliefweb.int.
United Nations Environmental Program: www.unep.ch.
Wikipedia, The Free Encyclopedia: www.en.wikipedia.org.

SUGGESTED READINGS

1. Bryant E: Natural Hazards. Cambridge, Cambridge University Press, 2005.
2. Dregne HM, Rozanov B: A new assessment of the world status of desertification. Desertification Control Bull 20, 1991.
3. Dudley WC, Lee M: Tsunami! 2nd ed. Honolulu, University of Hawaii Press, 1998.
4. Erikson J: Quakes, Eruptions and Other Geologic Cataclysms, rev. ed. New York, Facts on File, 2001.
5. Food and Agriculture Organization of the United Nations: The State of the World's Forests, 2001. Rome, Food and Agriculture Organization, 2001.
6. Grainger A: The Threatening Desert: Controlling Desertification. London, Earthscan, 1990.
7. Hanley ML: Can the Black Sea be saved? World Dev 3:6, 1990.
8. International Federation of Red Cross and Red Crescent Societies: World Disasters Report 2002, 2003, and 2004. Geneva, IFRC 2002, 2003, 2004.
9. International Tsunami Information Center: Tsunami: The Great Waves. Honolulu, International Tsunami Information Center, 1995.
10. Kovach R, McQuire B: Guide to Global Hazards. Richmond Hill, Ontario, Firefly Books, 2004.
11. Nuhfer E, Proctor R, Moser P: The Citizen's Guide to Geologic Hazards. Westminster, CO, American Institute of Professional Geologists, 1993.
12. Smith K: Environmental Hazards: Assessing Risk and Reducing Disaster. 4th ed. London, Routledge, 2004.
13. Tilling RI: Volcanic hazards and their mitigation: progress and problems. Rev Geophys 27:237, 1989.
14. Twigg J: Disaster risk reduction: mitigation and preparedness in development and emergency programming. Good Pract Rev 9, 2004.
15. United Nations Inter-Agency Secretariat of the International Strategy for Disaster Reduction: Living with Risk: A Global Review of Disaster Reduction Initiatives. New York, UN/ISDR, 2004.
16. United States Agency for International Development: Disaster Reduction: A Practitioner's Guide. Washington, DC, United States Agency for International Development, Bureau for Democracy, Conflict and Humanitarian Assistance, 2002.
17. Vickers DO: Tropical cyclones. Nature Resources 27:31, 1991.
18. Wernly D: The Roles of Meteorologists and Hydrologists in Disaster Preparedness. Geneva, World Meteorological Organization, 1995.
19. Wilhite DA: Preparing for Drought: A Guidebook for Developing Countries. New York, Earthwatch Climate Unit, United Nations Environmental Programme, 1992.
20. Wilhite DA (ed): Drought: A Global Assessment, vols 1 and 2. London, Routledge, 2000.
21. World Meteorological Organization: Global Perspectives on Tropical Cyclones. Geneva, World Meteorological Organization, 1995.
22. World Resources Institute: World Resources 1996–97. Oxford, UK, Oxford University Press, 1996.

81 Wilderness Preparation, Equipment, and Medical Supplies

Matthew R. Lewin, Suzanne C. Jensen, and Timothy F. Platts-Mills

Wilderness travel and recreation expose individuals to illness and injury far from medical care. Commonplace problems, such as blisters, diarrhea, and dehydration, may become serious obstacles to a journey, and major illness may go undiagnosed. Some wilderness activities may result in traumatic injuries that threaten life or limb. In experienced hands, the correct medications and equipment can greatly reduce the morbidity experienced by individuals who encounter medical problems in a remote setting. This chapter identifies medicines and medical equipment useful in a wilderness setting and provides recommendations for trip planning, vaccinations, and prescribing and transporting medications. Also detailed are the basics of preparation and procurement, taking into consideration the limited space and budgets commonly allocated to wilderness expeditions.

EPIDEMIOLOGY

Before any travel in the wilderness, it is important to understand the epidemiology of wilderness injuries. However, it is difficult to make statements on the precise statistical risk for injury or illness associated with outdoor travel. In the absence of required reporting of medical problems, information has come from special interest groups and prehospital care providers (emergency medical services) in national parks. Future wilderness epidemiologic data may better characterize injury and illness patterns.

Despite such limitations, the proportion of accidental injuries to illness occurring during travel is remarkably constant. Data on outdoor travel suggest that traumatic injury, generally of a minor nature, exceeds medical illness by roughly threefold. When reviewing injury patterns, most are attributable to soft tissue damage (e.g., abrasions, lacerations, and sprains); serious dislocations and fractures account for less than 5% of all trauma. The lower extremities are by far the most likely to be involved in minor orthopedic injuries,[12] emphasizing the importance of appropriate footwear selection and foot care.

Most medical illnesses reported by wilderness travelers are attributable to nonspecific syndromes, such as gastroenteritis or upper respiratory illness. These illnesses probably result in part from exposure to new pathogens and from travel conditions that preclude adequate preemptive hygiene measures. Other commonly reported medical problems include headache (exacerbated by high altitude), dyspepsia from local food intolerance, dehydration, heat-related illness, dermatitis, sunburn, allergic reactions, blisters, and other integument-related problems.[3,9,12]

After excluding deaths from cardiac causes, drowning remains the number one cause of accidental death among outdoor participants. Ice and rock climbing generate a unique array of injuries, including rare traumatic death from severe head trauma. Environmental causes of illness predominantly relate to high altitude and extremes of temperature.[3,9,12,13] Awareness of weather patterns, participant condition, and journey duration is critical to ensure that appropriate first-aid equipment, not routinely found in most kits, is available to deal with relevant emergencies. Recommendations for such unique supplies are covered in the section on specialized equipment found in Part 2 of this chapter.

GENERAL PREPARATION

Prevention and management of injury and illness are among the foremost challenges of wilderness recreation. Medical care may be greatly delayed for the injured person many hours from civilization. Educating travelers, planning carefully, and carrying appropriate medical and nonmedical emergency supplies can minimize unnecessary suffering. Large expeditions frequently enlist experienced medical professionals in logistic planning. However, most small groups trekking into the wilderness do not have access to this expertise. Even when a physician is a party member, he or she is usually not specifically trained in wilderness medicine.

This chapter is organized in five parts. The first part is an overview of general preparation, including planning strategies and preventive measures, with additional attention to travelers with chronic medical conditions. The second part describes different types of medical kits and details specialized medical equipment. The third part describes various medications useful in the wilderness setting, followed by a discussion regarding preparation for common medical problems encountered in the wilderness. The final part suggests strategies for both organizing materials in a kit and estimating optimal quantities of supplies.

Box 81-1. Guidelines for Travel in Developing Countries

BEFORE TRAVEL

Consult local sources of medical help; the International Society of Travel Medicine (ISTM) Clinic Directory is available by phone request (770-736-7060) or online (www.istm.org).

Obtain up-to-date travel warnings from the Centers for Disease Control and Prevention (CDC): International Traveler's Hotline (www.cdc.gov).

Consider purchasing the CDC's *Yellow Book*, a reference published every 2 years for those who advise international travelers of health risks. It is written primarily for health care providers, although others may find it useful.

Obtain necessary vaccinations. Recommendations are available from the CDC (www.cdc.gov) and the World Health Organization (www.who.int/en/). Update vaccinations for tetanus, measles, mumps, and rubella as needed.

DURING TRAVEL

Contact U.S. embassy on arrival. Have extra copies of essential documents such as passports and itineraries. Carry phrase books and dictionaries with phonetic and native script translations.

Use a repellent containing high concentrations of DEET. Citronella and other homeopathic remedies are falsely reassuring.

AVOID

Ice, unboiled or unbottled water, and uncooked food

Wading or swimming in lakes and canals of populous regions

Blood transfusions and use of reusable needles

Straight-razor shaves and acupuncture unless the blade or needles are changed in your presence

Nocturnal travel

Part 1: Planning Strategies, Preventive Measures and Risk Factors for Wilderness Travel

Mode of travel, destination, duration, environment, and the number of people on any trip vary such that no single recipe exists for the perfect medical kit. Physicians face the challenge of advising travelers on medicines and medical equipment, knowing that their patients may face decisions they are not prepared to make. Travelers and their physicians must recognize the limits of their knowledge and seek appropriate consultation from physicians, books, wilderness medicine courses, experienced colleagues, and myriad high-quality Internet resources relevant to travel anywhere around the globe. Pretravel planning, resourcefulness, and the ability to communicate and improvise are key elements to minimize risk and successfully diagnose and treat medical problems in the wilderness. Regardless of a physician's prior wilderness medicine experience, it is essential to have detailed discussions with expedition leaders and past members regarding the occurrence of medical problems, trauma, and the usefulness of various medical supplies on previous, similar expeditions. Local rangers and emergency medical service personnel may provide valuable information about weather patterns and risks specific to local terrain. Climbers and mountaineers will benefit from reading *Accidents in North American Mountaineering*, an annual publication that describes and analyzes climbing injuries and fatalities occurring each year in North America.

Familiarity with evacuation resources (e.g., satellite phones) and facility with Global Positioning System (GPS) devices are helpful. In the past, these items were prohibitively expensive, but they are now quite affordable. One can now purchase a basic GPS unit for about U.S. $100 or rent one on weekly or monthly bases. Like GPS units, satellite phones can be rented, charged by battery, or charged and recharged on car batteries or solar panels. In developed countries, a cellular telephone may have good reception even in remote areas. Using satellite or cell phones to activate a rescue can expedite patient care, but neither device should be regarded as a substitute for proper preparation and sound judgment during wilderness travel. Rescue services in most mountainous regions outside the United States require accident insurance or a large amount of cash before helicopter transport. If traveling in these areas, medical designees should work with group leaders to ensure that appropriate rescue and accident insurance coverage is obtained for all expedition members.

Before the Trip

Travelers and their physicians should take an educated approach to the places they are going and the people who will accompany them. Attention should be given to regional hazards and locally available health resources. For example, drinking water quality and treatment options, endemic infectious diseases, and environmental exposures should all be considered. Malaria prophylaxis should be used based on Centers for Disease Control and Prevention (CDC) or World Health Organization (WHO) recommendations (Box 81-1), and, in areas with mosquitoes, members should carry mosquito nets and insect-repellent containing a high concentration of N,N-diethyl-m-toluamide (DEET).

If a physician or designated medical leader accompanies the group, that person should obtain the medical history of each member, including vaccinations relevant to the area of travel, chronic diseases, prior surgical history, allergies, and regular medications (see Appendix A). Travelers should receive tetanus boosters and destination-appropriate vaccinations. Dental problems should be treated before the trip, and diabetic travelers should be instructed to bring an ample supply of their routine medications as well as a functioning and a spare glucose meter. Musculoskeletal complaints from overuse are common during travel. Proper conditioning can reduce the incidence of these injuries, and group members should be encouraged to prepare for their trip by training in a way that simulates anticipated activities during the expedition. For groups without trained medical leadership, physicians should address these issues with their patients before departure directly or by questionnaires (see Appendix A). When not accompanied by a physician, the trip leader or coordinator assumes responsibility for assessing health limitations of the group. The coordinator should confidentially but frankly discuss medical problems with

Box 81-2. Checklist for General Preparedness

- Advance physical conditioning, medical and dental checkups
- Immunizations (especially tetanus, hepatitis A and B)
- Planning of potential rescue and evacuation routes and leaving an itinerary with someone to initiate rescue if individual or group does not return on time. Working knowledge of GPS, satellite, and local phone systems.
- Planning for water disinfection (chemicals or devices)
- Medical alert medallions or cards (illnesses, allergies, medications such as steroids, insulin, and oral hypoglycemics) and emergency contact information
- Historical appraisal of weather conditions: National Climatic Data Center (www.ncdc.noaa.gov); Precipitation and temperature, immediate and short-term forecasts: National Weather Service, The Weather Channel, large general search engines such as Yahoo! and MSN (www.nws.noaa.gov, www.weather.com, www.yahoo.com, www.msn.com)

each candidate and require a medical evaluation if uncertainty exists. Safety of the individual and the group are the coordinator's first priorities. Box 81-2 addresses general preparedness and travel in developing countries.

Before the trip, consideration should be given to caffeine, alcohol, and drug dependencies. Management of such dependencies is a sensitive and complex issue unlikely to be satisfactorily resolved in a wilderness setting. Eliminating access to substances to which individuals have chronic dependencies will often cause unnecessary hardship. Strategies for addressing dependencies should be discussed with both the individual member and group leader. Excess alcohol should be avoided because it causes peripheral vasodilation, which can result in excess heat gain in hot environments and heat loss in cold or wet environments. Alcohol also tends to exacerbate symptoms of acute mountain sickness and should be avoided at altitudes above 8000 feet (2438 m). In addition, alcohol's effects on judgment and sensory perception may result in failure to acknowledge early symptoms of environmental illness.

An additional challenge facing the medical designee is the allocation of opioid-containing analgesics, sleep agents, and other medications. Expedition members naturally hope to be as comfortable as possible and to sleep well, even though this may be an unreasonable expectation at times. Likewise, medical designees want to provide adequate treatment for pain and anxiety. Because of the potential for inappropriate use of these medications, it is advisable to limit access and consistently apply predetermined policies on the use of opioids and anxiolytics.

Healthy Participants

Even the most active and healthy individuals should begin a graduated exercise program at least 2 months before departure to minimize the deleterious effects of muscular, metabolic, and mental fatigue. This is especially important for people traveling to a high altitude; aerobic capacity in a sedentary person drops about 4% for each 300 meters (1000 feet) above the 1200-meter (4000-foot) level, but the loss is only half as great in an aerobically fit individual.[4,10] Careful stretching of muscle groups may increase efficiency and lessen the likelihood of soft tissue injury during exertion and minor accidents.

If excessive environmental heat is anticipated, preparatory exercise in a hot, humid environment (simulated with sweat clothing) for 1 hour daily, for at least 7 days before departure, will help preserve plasma volume (aldosterone effect) and sweat rate while lowering myocardial oxygen demand and sweat sodium content.[8] This acclimatization will be lost within a week if not maintained. Such conditioning should be practiced with caution because of the risk for dehydration. During the trip, it is essential to wear a comfortable and sufficiently sun-protective hat. Hats are essential for both sun protection and as a means to protect against excessive insensible fluid loss from the scalp.

People with Preexisting Medical Problems

(see Chapter 29)

Patients with preexisting medical conditions should discuss in detail their travel plans and request recommendations from their primary doctor. It is advisable for the physician to speak to the trip coordinator about the itinerary if there are any lingering doubts about medical clearance for the patient. At-risk patients should wear medical identification bracelets and be encouraged to acquire and manage their own medications. The trip medical provider should know about these illnesses and carry replacement medications provided by these individuals for safe keeping. Patients with a history of chronic obstructive pulmonary disease (COPD), asthma, heart disease, diabetes, allergies, or seizures require special consideration. Pulmonary hypertension, recent pulmonary embolism, history of recurrent spontaneous pneumothorax, sickle cell disease, and sleep apnea are considered absolute contraindications to high-altitude travel.

Patients with a history of significant allergic reactions should carry an epinephrine autoinjector or equivalent (see Bites and Stings in Part 4). Patients with a history of seizures should continue routine medications and also carry an injectable form of benzodiazepine, such as lorazepam (Ativan). Suppositories may be appropriate if traveling in a cool or cold environment or with juveniles.

Participants with preexisting cardiopulmonary disease and those with some of the selected medical problems described later deserve special attention. Caution should be taken when people with a history of COPD or asthma are attempting high-altitude travel. A plan for rapid descent is essential because people with asthma and COPD may experience difficulty as a result of hypoxia from high altitude. Similarly, dry air, exercise, or noxious stimuli (e.g., smoke or red tide[11]) may exacerbate reactive airway disease. Thus, a plan for rapid treatment should be in place before departure. Exercise in cold, dry air may trigger wheezing during travel. Poor air quality, a by-product of fossil fuel burning or even remote volcanic activity, along with winds that can "stir up" larger particulate matter such as dust or sand, can also cause irritation. Besides carrying a β-agonist metered dose inhaler, travelers with COPD or asthma should carry a 2-week course of an oral steroid (e.g., prednisone) plus a broad-spectrum oral antibiotic. Studies of aircraft pressurized to 8000-foot (2438 m) altitude reveal that people with moderately severe COPD may have significant dyspnea; this may serve as a surrogate marker for the altitude to which such individuals can safely travel.[1,5] People with mild to moderate COPD should not sleep above 3048 m (10,000 feet) because of nocturnal desaturation.

Patients with known history of significant cardiac disease should not participate in wilderness activities that require excessive exertion. During travel, these patients should continue routine medications and carry copies of their most recent electrocardiogram. Outdoor adventure travel can provoke angina in people with underlying heart disease. There is continued debate concerning the evaluation and advice a physician should provide to patients with cardiovascular disease. In general, people with predictable angina of mild to moderate level may engage in outdoor travel if able to exercise by Bruce protocol for at least 9 minutes; such individuals must moderate their activity in proportion to the decrease in available oxygen that limits work capacity at higher altitude. As a guideline, activity levels should be titrated to not exceed a heart rate that is roughly 75% of one's ischemic threshold. For those with unstable angina, congestive heart failure, or valvular disease such as aortic stenosis, vigorous adventure travel is contraindicated.

A person with a history of cerebral transient ischemic attacks can participate in outdoor travel if attention is given to proper hydration and use of aspirin or other anticoagulants such as warfarin (Coumadin) or clopidogrel (Plavix), as indicated.

Outdoor travel often disrupts the normal meal schedules of diabetic travelers. Insulin-dependent diabetic travelers should carry their own insulin and glucose meter. Although some individuals may need less insulin when participating in high levels of exercise such as backpacking, this phenomenon is not true for all. Patients should monitor their glucose at least twice a day, regardless of how well they feel, and modify their insulin regimen accordingly. Other group members in close contact with insulin-dependent diabetic travelers should know that the first two interventions for an ill-appearing diabetic person are a small amount of sugar under the tongue and measurement of blood glucose. For those taking oral hypoglycemic agents with hypoglycemia refractory to sublingual sugar, injectable glucagon may be a worthwhile investment, provided there is the medical expertise to administer it and manage any adverse effects. During air travel, an insulin-dependent diabetic person should take his or her daily dose of insulin and eat according to the local time (departure) schedule. For a diabetic person traveling eastbound across multiple time zones, the day is effectively shortened; on arrival, the person should eat and administer insulin according to local time but reduce the dose by one third. For travel westbound, the day will lengthen, and a second dose of insulin after 18 hours of travel may be administered after glucose monitoring, if indicated.

Human immunodeficiency virus (HIV) infection should not preclude outdoor travel as long as an HIV-positive person pays meticulous attention to water disinfection and receives immunizations against pneumonia, influenza, hepatitis A, and hepatitis B.

Many prescription drugs predispose to heat, cold, and altitude-related illnesses. Diuretic use may lead to intravascular volume contraction, impaired heat transfer to the skin, dehydration, and potentially life-threatening electrolyte abnormalities such as hypokalemia. People taking diuretics should carry a packaged electrolyte replacement (oral rehydration solution) and a source of potassium (e.g., dried bananas or potato chips). The anticholinergic action of antihistamines, phenothiazines, and tricyclic antidepressants may result in hypothalamic dysfunction and diminished sweating. Whenever possible, alternative preparations should be considered for use during wilderness travel.

Patients with serious medical allergies or active illnesses should have an appropriate medical identification bracelet, anklet, medallion, or wallet card and store their personal medications in a protected but accessible location in their pack. Everyone should carry a complete medication list during foreign travel.

Education in First Aid and Wilderness Safety

Participants should be encouraged to take general courses in first aid and wilderness safety. Some agencies that offer general and specialized training in skiing, mountaineering, river rafting, and other types of wilderness medicine are listed in Appendix C. Locally organized programs may be found through the American Red Cross, sporting goods stores, and local colleges' continuing education departments.

Before departure, the trip coordinator should review emergency supplies with the group. He or she should demonstrate proper use of mechanical devices and discuss medication indications. Groups planning an extended or high-risk outing may wish to conduct a mock injury evaluation and management exercise.

Factors in Trip Planning

The primary considerations in planning a trip relate to (1) duration of the outing and maximum anticipated delay in obtaining medical assistance, (2) risks imposed by environmental extremes, and (3) hazardous recreational activities that require use of specialized equipment and supplies.

Availability of Medical Care. The longer the delay in obtaining advanced medical assistance, the more likely the irreversible loss of physiologic function, limb, or life. One must anticipate delays in care when in rural areas because the nearest physician or hospital may not be equipped to handle a major injury or illness. An extreme case example is the planning of emergency access to a recompression chamber for members of a deep-sea diving expedition. A more typical example is a deeply penetrating arm laceration. As hours pass, the likelihood of infection grows. If the victim can reach advanced medical help within an hour, it will suffice to control bleeding and apply a sterile dressing held in place by improvised cravats or tape. If definitive care is several hours away, irrigation with water containing a topical disinfectant may be desirable. If the delay in care will be 6 hours, a decision must be made whether to close the wound before evacuating the victim (see Chapter 20). Estimated time delay depends on the type of rescue services, method of contact, terrain, weather, and number of able (carrying) people. Party members should agree in advance on simple emergency distress signals, such as whistle or flashlight bursts in groups of three.

Manually evacuating a victim is an option but requires a relatively mobile victim or a minimum of six carriers if the victim is immobilized. In this regard, it is important to know whether other groups might be trekking in the same vicinity. If access is controlled by permit, the administering agency should be asked about neighboring parties' itineraries. The decision to carry a victim out must be based on a realistic appraisal of the time it will take messengers to reach aid versus the time, effort, and risk to the patient and party members performing an evacuation unassisted by an outside party. The likelihood of mishap rises as trip duration increases. This is partly attributable to unpredictable weather and the cumulative effects of fatigue and repetitive strain injuries. In addition, long trips usually involve

Box 81-3. Contents of a Personal Medical Kit

ON-PERSON ITEMS
Identification
Pencil and notepad
Hat and sunglasses
High-SPF sunscreen and lip balm
Topographic map and compass
Multiple-tool knife (e.g., Swiss Army, Leatherman) or razor blade
Nylon cord
Whistle and small reflective mirror
Lighter or waterproof matches
Poncho and/or space blanket
Adhesive compress and tape
Bandanna
Nonperishable, high-energy bar or gummy bears
Personal first-aid and hygiene material
Survival guide/first-aid booklet
Prescription medications, labeled (in plastic or waterproof aluminum box)
Over-the-counter medications noted in the comprehensive medical kit (see Box 81-4)

extensive planning, significant financial investment, and time away from work. Party members are therefore reluctant to cut the trip short and likely would rather continue in the face of mild medical disability and equipment failure. Groups planning to be away from civilization for more than a week should have a maximally diversified list of medical and contingency items.

Environment and Risk of Activities. Weather, terrain, and activity interact and increase the risk for illness or injury. Particularly hazardous combinations include winter climbing, mountaineering, skiing, and white-water kayaking. The anticipated ranges of weather and terrain must be figured into estimates of maximum delay to medical assistance. U.S. and global historical summary data indicating temperature ranges, winds, and duration, type, and amount of precipitation can be obtained from the National Climatic Data Center (see Box 81-1 and Appendix C). State and national park services and state climatology offices are also sources of such information about their territories. The National Weather Service office nearest the travel site can provide short-term forecasts and in many regions broadcasts weather information between 162.40 and 162.55 MHz VHF (see Appendix C).

Part 2: Medical Kits and Equipment

Medical supplies may be broken down into five components (Boxes 81-3 to 81-8): (1) personal medical kits, (2) more comprehensive community medical kits, (3) devices and medications for expeditions and the medically trained, (4) specialized equipment for particular environmental and recreational hazards, and (5) supplies stored in the vehicle.

Personal Medical Kit

The purpose of carrying a minimum amount of items on one's person relates to the ever-present danger of separation from travel partners. A sudden fall, avalanche, or swamping can quickly separate victim and gear. In anticipation of this, experienced wilderness travelers need to carry a bare minimum of items on their person at all times (see Box 81-3). Such supplies provide protection from the elements, permit self-treatment of minor traumatic injuries, and allow signaling for purposes of search and rescue. These items typically include assorted adhesive compress strips, knife or razor blade, butane lighter or matches (preferably the waterproof, strike-anywhere type), plastic whistle, small reflective mirror, length of thin nylon cord, and bandanna (which can double as a cravat or sling). A nonperishable source of quick high-energy food may be of value during isolation to maintain strength. These items can be compactly stored inside a plastic bag or small stuff sack and may be carried in either a pocket or a small pack (zippered, passport-size waist belts are comfortable and inexpensive). As noted previously, all travelers should carry some form of identification, and those with serious medical allergies or active illnesses should have an appropriate medical alert bracelet, anklet, medallion, or wallet card. Those with a history of bee, wasp, or other anaphylactic allergy should carry at least two preloaded syringes of 1:1000 aqueous epinephrine solutions (e.g., EpiPen, Ana-Kit) in a cool, dark compartment and should inform others in the party of the medicine's location and proper use. Certain contingency "essentials" should be carried on virtually every venture. In addition to one's personal first-aid, hygiene, and clothing items, a flashlight, coins for telephone calls, and extra pair of sunglasses are recommended. Extra clothing, food, and water should be carried in proportion to the risk associated with the trip.

A host of over-the-counter medications may be useful for wilderness travel, especially for low-risk outings of short duration. Some commonly required nonprescription items that may be of value are listed in Box 81-4. Of prime importance is control of pain associated with trauma. For mild to moderate pain, aspirin, acetaminophen, or a nonsteroidal anti-inflammatory drug (NSAID) is effective. Decongestants are helpful in treating symptoms associated with upper respiratory infections, and their antihistaminic effects are useful for the treatment of allergies and insomnia. Gastrointestinal complaints may necessitate antacids and an antidiarrheal agent.

Saline eyewash is helpful for irritated eyes and in removing foreign objects. Antiseptic cream or ointment is useful in treating superficial infections of the skin, and a steroid ointment is of value for treatment of certain rashes or contact dermatitis. Aloe vera gel is useful for treatment of frostbite and injuries from burns or excessive sun exposure. However, sunburn is best avoided by fastidious application of high SPF (>30) sunscreen and lip balm and wearing protective clothing.

Comprehensive Community Medical Kit

Traumatic injuries represent the greatest concern during wilderness travel. A comprehensive medical kit is the most important community item (see Box 81-4). Emphasis is placed on the management of wounds and stabilization of soft tissue and orthopedic injuries through appropriate bandaging and splinting (Fig. 81-1). Bulky and heavy items, including most stock first-aid and contingency supplies, should be labeled and distributed among group members for storage and transport. A cross made from strips of tape or cloth may be placed on the pack overlying the compartment containing the first-aid items. This allows ready identification by any member of the party in an emergency situation. Repair materials are best kept in clearly labeled stuff sacks or watertight aluminum (e.g., Zarges) or plastic (e.g.,

Box 81-4. Contents of a Comprehensive Community Medical Kit

WOUND MANAGEMENT
Liquid soap
Alcohol-based gel for hands (e.g., Purell)
Clean and sterile gloves
Splash shield and face protection
Irrigation syringes (1 cc to 60 cc)
18-gauge needle
Irrigation saline
Morgan eye lenses
Sterile surgical scrub brush
Alcohol pads and gels
Antiseptic towelettes
Povidone-iodine solution 10% (Betadine)
Wound closure strips, butterfly closures
Tincture of benzoin
Skin adhesive glues
Suture materials, needle driver, tissue forceps
Disposable skin stapler and remover
Silver nitrate sticks
Scalpel with no. 11 or 15 blade
Tourniquet or blood pressure cuff

COMMONLY AVAILABLE OVER-THE-COUNTER MEDICATIONS AND REMEDIES
Acetaminophen (Tylenol)
Aloe vera gel
Aerosolized epinephrine (Primatene Mist)
Antibiotic or antiseptic ointment (Bacitracin, Neosporin)
Calamine lotion
Diphenhydramine (Benadryl)
Famotidine (Pepcid)
Glucose paste or tablets
Glycerin rectal suppositories
Hemorrhoidal ointment or witch hazel pads
High-SPF sunscreen and lip balm
Hydrocortisone cream 1%
Ibuprofen 200 mg (Advil, Motrin, Nuprin)
Loperamide (Imodium AD)
Meat tenderizer (for bee stings)
Meclizine (Antivert, Bonine)
Omeprazole (Prilosec)
Oxymetazoline (Afrin)
Phenylephrine (Neo-Synephrine, Sinex)
Pseudoephedrine (Sudafed)
Ranitidine (Zantac 75)
Saline eye wash
Simethicone (Mylanta II antacid)
Tinactin antifungal cream

PRESCRIPTION MEDICINES
Function of trip duration and interval to care; see Tables 81-2 and 81-3.

BANDAGES, SPLINTS, AND SLINGS
4-×4-inch (10 × 10 cm) sterile dressing pads
5-×9-inch (15 × 27 cm) sterile dressing pads
3-inch (9 cm) sterile gauze bandage
Cotton-tipped applicators
Nonadherent sterile dressing
Xeroform and petroleum jelly (Vaseline) gauze burn dressings
Elastic bandage wraps with Velcro closures
Adhesive cloth tape
Adhesive bandages (Band-Aid)
Tegaderm
Eye pad and eye shield
Moleskin, Blistoban silver duct tape
SAM Splints (4.4×36 inch [15 × 110 cm])
Aluminum finger splints
Kendrick (or improvised) femur traction device
Triangular (cravat) bandage and safety pins

MISCELLANEOUS ITEMS
Waterproof flashlight and matches
Signal mirror/dental mirror and whistle
CPR mouth barrier
Plastic resealable bags
Permanent markers (Sharpie)
Notebook and record-keeping supplies
Adhesive labels
Pill bottles and cotton balls
Nail clippers (toe and finger)
Steel sewing needles, paperclips, safety pins
Forceps for removal of splinters and ticks
Pocket knife or multiple tool knife
Trauma shears and medical scissors
Duct tape (silver)
Tongue depressors
Chemical ice packs and heating packs
Sun hats, high-SPF sunscreen and lip balm
Emergency Blanket (e.g., SPACE, Protech)
DEET-containing insect repellent
Contact lens solution and case

EQUIPMENT
Diagnostic instruments: see Table 81-1
Specialized Equipment: see Box 81-8

DENTAL SUPPLIES
Oil of cloves (eugenol), 3.5 mL; combine with zinc oxide powder to make temporary fillings
Zinc oxide powder
Cavit (7 g), IRM (Intermediate Restorative Material), Express Putty
Wooden spatulas (for mixing and applying)
Paraffin (dental wax) stick
Dental floss
Dental mirror
Cotton rolls and pellets
Injectable lidocaine 2% or bupivacaine 0.25%

TABLE 81-1. Portable Diagnostic Instruments for Wilderness Travel

DEVICE	INDICATION
Urine pregnancy test (Baby Check Midstream, SureStep, many other generic and name brands)	Essential for the evaluation of abdominal pain in women of child-bearing age. A positive pregnancy test raises the possibility of ectopic pregnancy; immediate evacuation should be considered.
Glucometer (e.g., Therasense)	Useful for routine diabetes management and for the evaluation of ill-appearing diabetic individuals who may have a too-low or too-high serum glucose level.
Fluorescein dye strips and fluorescent light sticks	Evaluate for corneal abrasions. If present, the eye should be flushed, the lid flipped to search for a foreign body, and the patient treated with topical antibiotic drops or ointment.
Hemoccult cards and developer	Patients with traveler's diarrhea and bloody stool should not be given loperamide or other antiperistaltic agent.
Low-reading (hypothermia) thermometer (e.g., ADTEMP 419 digital)	Essential for the evaluation of hot or cold patients, particularly those for whom alternative diagnoses are being considered.
Sphygmomanometer (blood pressure cuff)	Useful for accurate measurement of blood pressure, particularly in trauma patients and in patients with tachycardia or altered mental status. May be used as an adjustable tourniquet.
Stethoscope	Useful for auscultation of the abdomen and chest, particularly to evaluate for the presence of wheezing, pulmonary edema, or pneumothorax.
Urine test strips (e.g., Clinitek)	Useful for the evaluation of abdominal pain, urinary symptoms, and hyperglycemia. Hyperglycemia and the presence of urine ketones suggest diabetic ketoacidosis.
Chronometer with seconds hand	Useful for accurate measurement of heart rate and respiratory rate. Also important in planning evacuations.
Magnifying glass	Foreign-body identification and removal.
Pulse oximeter (e.g., Respiron, Nonin)	Finger-sized, digital light-emitting diode (LED) readouts for estimating tissue oxygenation.
End-tidal CO_2 detector (e.g., Nellcor)	Colorimetric devices are available to aid in the confirmation of endotracheal tube placement. Quantitative devices are coming onto the market (Nanomix).

Box 81-5. Contents of a Medical Kit for Expeditions and the Medically Trained

- Medical guidebooks, PDA, and Internet sources
- Comprehensive first-aid kit (see Box 81-4)
- Appropriate prescription medications for general illness (see Tables 81-2 and 81-3)
- Devices and medications for the medically trained (see Box 81-6)
- Indicated equipment based on recreational and environmental hazards (see Box 81-7)

Otter, Pelican) boxes independent of the first-aid and medical supplies.

Medical Kits for Expeditions and the Medically Trained

Medically trained individuals traveling with a group at high risk for trauma or illness may wish to carry supplies requiring special expertise for proper use (see Boxes 81-5 and 81-6). Some of the necessary skills may be acquired in advanced first-aid, paramedic, or nursing classes. Intramuscular and intravenous (IV) medications should be administered only by those with formal training in the indications, dosing, and risks of those drugs. Inclusion of a limited supply of emergency oxygen or IV saline may be reasonable to carry for a high-risk expedition. Adequate rehydration can generally be accomplished in a conscious person with a motile bowel using commercially available oral rehydration packets. Conversely, a variety of IV fluids and medications (e.g., antibiotics, pain medicines, dextrose in saline) are a basic component of either a medical base support camp or situations of search and rescue. Items such as surgical tools, chest tubes, and mechanical suction devices are appropriate only for extremely high-risk expeditions and military excursions.

Specialized Equipment for Environmental and Recreational Hazards

A host of recreational activities have inherent risks that may dictate specialized equipment beyond a basic medical kit. Mountain climbing not only poses a risk from traumatic injury but also subjects the climber to altitude-related illnesses, possibly requiring portable oxygen or a pressure bag (e.g., Gamow Bag). Extreme cold exposure might dictate the need for technical devices to provide warmed IV fluids and humidified oxygen. Adventures in white-water sports place the participants at risk for freshwater drowning, and such expeditions might consider carrying adjuncts for airway management. Cycling poses risk for soft tissue injuries from abrasions that require occlusive water-based gel dressings for optimal wound care. Travel to underdeveloped nations in which mosquitoes transmit deadly infectious diseases requires specialized equipment, such as protective netting for sleeping and chemical insecticides or repellents. A newer option for control of nausea associated with motion sickness is the ReliefBand, a U.S. Food and Drug Administration (FDA)-approved device intended to stimulate nerves and reduce nausea.

Box 81-6. Devices and Medications for the Medically Trained

DEVICES

Airway, nasopharyngeal (impaired mental status; resuscitation)
Cricothyrotomy cannula or catheter (e.g., Abelson cannula)
Adult endotracheal tube(s), laryngeal mask airway (LMA)
Chest tube set (chest trauma; empyema—practical only on major expeditions)
Glucose testing strips and buccally absorbed glucose preparation (strips must be protected from freezing)
Intravenous tubing with high-flow drip chamber and spike (see Fig. 19-2 for use as emergency cricothyrotomy device)
Needles and syringes (for IV hydration and emergency injectables)
Ophthalmoscope with blue filter and fluorescein strips to stain corneal lesions (retinal hemorrhages; anterior eye examination)
Oxygen (hypoxemia, shock, cerebral/pulmonary edema, impaired mental status)
Sphygmomanometer (aneroid, plastic housing)
Stethoscope
Suction device (mechanical, for clearing oral cavity; chest tube drainage)
Surgical tools
Surgical or fine dust masks

MEDICATIONS

General Use

Intravenous (IV) solutions (for medications, hydration and wound irrigation)
Antibiotic, potent oral with wide-spectrum coverage (e.g., ciprofloxacin)* or injectable (e.g., ceftriaxone)*
β-Agonist metered-dose inhaler (for asthma, anaphylactic reaction)*
Ophthalmic anesthetic*

High Risk for Insect Bites/Allergies

EpiPen* or Twinject
Diphenhydramine oral (for allergic reaction, mild sedation/insomnia)
Glucagon
Oral corticosteroid*

High Risk for Trauma

Fentanyl patch (Duragesic) applied to skin on chest when mental status precludes oral opioids*
Alprazolam (Xanax), Zolpidem (Ambien) for sedation*

High Risk for Altitude Illness or Snowblindness

Acetazolamide for mountain sickness*
Corticosteroid oral or injection for cerebral edema*
Nifedipine for pulmonary edema*
Ophthalmic cycloplegic for pain from snow blindness (e.g., cyclopentolate 1%)*
Ophthalmic corticosteroid-antibiotic combination (e.g., Maxitrol)*

*See Table 81-2 for considerations in dispensing medication.

Box 81-7. Specialized Equipment for Environmental and Recreational Hazards

HIGH ALTITUDE

Gamow Bag and accessories
Pulse oximeter
Oxygen canisters, nasal cannulae, face masks, oxygen tubing and connections

COLD AND AVALANCHE EXPOSURE

External thermal stabilizer bag
Res-Q-Air
Hot-Sack IV Warmer
Chemical warmers (e.g., Grabber)
Electric foot warmers (e.g., Hotronic)
SPACE Thermal Reflective Survival Bag
Low-reading thermometer
Adhesive climbing skins
Ice axe
Adjustable ski/probe pole
AvaLung avalanche vest
Tracker Digital Transceiving System
Avalanche beacon

WATER SPORTS (LOW IMPACT)

CPR Micro shield
Water disinfection equipment (filter, iodine)

WATER SPORTS (HIGH IMPACT)

Cervical spine immobilizer
Pelvic immobilizer (e.g., SAM sling)

BICYCLING

All-terrain cyclist kit
Occlusive dressings

TROPICAL AND THIRD WORLD TRAVEL

Pressure immobilization equipment (snake bite)
Permethrin-containing repellent
Mosquito nets
Oral rehydration salt packets
Water disinfection equipment (filter, iodine)

MOUNTAIN CLIMBING AND HIKING

SAM Splints and pelvic immobilizer (sling)
Slishman Splint (not available in the United States)
Ankle brace (Aircast)

Box 81-8. In-Vehicle Emergency Supplies

FIRST-AID, RESCUE, AND SURVIVAL
Comprehensive medical kit (see Box 81-4)
Large burn dressings
Boards for splint construction
Backboard, short or folding long (e.g., Junkin—see Appendix B)
Bags, large plastic
Blankets—wool and "space" blankets
Climbing rope and hardware†
Candles, long burning
Flares
Flashlights and batteries in water-tight storage
Food and water
Matches (waterproof) or lighter
Radio, citizens band (CB)
Satellite phone
GPS (e.g., Garmin *e*Trex)
Rope
Saw with metal-cutting blade
Small stove, pot or coffee can, and utensils
Tarp, plastic
Toilet paper
Whistle

AUTOMOTIVE
Aluminum foil to cover windows
Cables to jump battery
Chains with tighteners (with repair links and special pliers)*
Fire extinguisher
Flares, 10-minute (at least six; can also serve as fire starters)
Industrial or welding gloves
Oil, extra can
Shovel, metal or Lexan with short or collapsing handle
Tool kit (consider inclusion of a small ax)
Tow chain or cable
Wheel chock or wedge blocks
Siphon hose or hand-cranked gas pump
5- and 10-gallon water cans
Propane tank with regulator and hoses
Solar panels or rolls
Car cigarette lighter adapters and cables
Country appropriate plug adapters
Batteries
Gasoline-powered generator
Propane cigarette lighters

*Winter weather supplies.
†Mountain terrain supplies (special training needed).

The following paragraphs review specialized items not basic to the comprehensive medical kit that may be needed for certain high-risk recreational activities. Box 81-7 lists common recreational activities and identifies specialized equipment that may be considered for high-risk expeditions. Suppliers and their unique products are listed in Appendix B. Many of these items are expensive, but also potentially lifesaving.

High-Altitude Exposure (see also Chapter 1). The Gamow Bag (Fig. 81-2) is a portable hyperbaric chamber resembling a large sleeping bag with a window. It has been shown to be effective for the treatment of high-altitude pulmonary and cerebral edema. Constructed of nylon, the bag is foldable and has a packing weight of 14.5 pounds (6.6 kg). Inflated, it is 7 feet (2.1 m) long and has a diameter of 21 inches (64 cm). The bag is pressurized with a foot pump at a rate of 10 to 20 strokes per minute and has relief valves pressurized to 2 pounds (approx. 1 kg) per square inch, allowing venting of expired air. In a situation such as a remote clinic with electricity, a mechanical compressor can be used to avoid the fatiguing task of foot pumping. In addition, a breathing bladder has been introduced. One end of this large nylon bag connects to a face mask, and the other to one of the pressure relief valves of the Gamow Bag. The patient inhales uncontaminated air from the Gamow Bag, and the exhaled air flows down a plastic tube into the bladder. This obviates the need for manual foot pumping of the bag for a 15- to 30-minute period until the bladder becomes full. It is then necessary to operate the foot pump again to repressurize the bag. The bladder is an excellent adjunct for expeditions at very high altitude that are far from medical evacuation and when a prolonged period of resuscitation is anticipated. The recently developed Gamow Tent operates on the same principles as the Gamow Bag. It is nearly 50% larger, and the added height allows a victim to sit upright.

The aforementioned devices are expensive and intended for expeditions going to extreme altitude (greater than 4267 m [14,000 feet]) or in dangerous situations that prevent an easy, rapid descent (e.g., ice climbing on a steep glacial crevasse).

Cold Exposure. Equipment purchases for cold exposure relate to the stabilization and resuscitation of victims with accidental exposure hypothermia. For most travel, removal of an individual from exposure and prevention of further heat loss are sufficient measures when medical assistance is nearby. People engaging in low-risk travel can use a sleeping bag wrapped with a space emergency blanket to provide a capsule that preserves heat and allows thermal recovery. The latter product is made of a lightweight material capable of reflecting and retaining more than 80% of radiated body heat. Another helpful product is the Grabber Warmer. Useful in the prevention of frostbite, this small pad undergoes a chemical reaction on exposure to the air, producing heat. Grabber brand warmers can maintain a temperature of 66°C (150°F) for 7 hours or longer. They can be placed in gloves, shoes, and pockets for the prevention of frostbite, but should be used only as directed because of the high temperatures that can be generated.

For more serious expeditions involving high-risk travel or distant from medical rescue or assistance, several highly sophisticated products may be of value. Of value in circumstances of high avalanche danger is a lightweight vest with a breathing

A

B

Figure 81-1. **A,** Schematic diagram suggesting arrangement of a comprehensive expedition medical kit. **B,** Medical kit from the 2004 Mongolian American Expedition to the Gobi Desert set up in watertight, Zarges aluminum freight box.

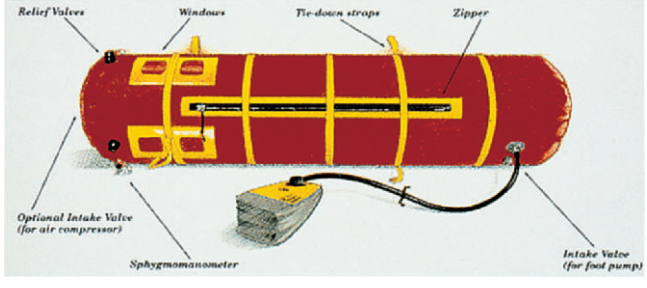

Figure 81-2. Gamow Bag. Attached is the foot pump required to pressurize the compartment to 2 pounds per square inch. Four windows are strategically located to permit observation. Entry is through a lengthwise zipper.

tube that permits extraction of oxygen and release of carbon dioxide when trapped under snow (Fig. 81-3). Such a device can significantly prolong survival chances until the victim is found by another person appropriately manned with a snow shovel and an avalanche beacon or probe. For victims of severe exposure hypothermia, elevation of core temperature and prevention of further body heat loss may be critical. Inhalation of warm, humidified air provides a method of heat exchange and is now possible with portable field units. The Res-Q-Air provides warm, saturated air by using a battery-operated device that requires a small amount of water and delivers heat for inhalation through an attached hose and face mask. The unit is simple to operate, has a temperature-control valve, and runs for 1 hour on battery power. It is small (9 × 3 × 2 inches [27 × 9 × 3 cm]), weighs approximately 4.5 lb (2 kg), and is portable. In the event that emergency IV fluids may be required, an IV bag warmer, the Hot-Sack, can be used. This product comes in a soft, rugged portable case and has its own battery power source. Temperature within the bag remains at 37° C (98° F), so that IV fluids are kept warm. The bag has a protective sleeve to place over IV tubing to maintain warmth.

Advanced expeditions and people involved in cold-water search and rescue should consider the purchase of a Hypothermic Stabilizer Bag by MARSARS. This product consists of an internal, high-pile fabric that wicks water to allow quick drying of hypothermic victims. The thermal properties far exceed those of an equivalent-thickness conventional down sleeping bag, and the product requires no additional insulation underneath for support and comfort. A key feature involves the ability to perform complete cardiopulmonary resuscitation through an access window over the chest. The stabilizer's outer cover is made of water-resistant material and has carrying handles to allow for safe transport of the victim.

Figure 81-3. The AvaLung (Black Diamond, Ltd.). A lightweight vest designed to improve avalanche survival. Victims are able to draw air from even a dense snow pack through the front using an air-exchange mouthpiece. A one-way valve permits exhalation of carbon dioxide expelled through the vest back.

Water Disinfection. The key design features of a filtration system that produces acceptable water for consumption are (1) adequate pore size of the filter to remove bacteria and protozoan cysts (0.2 mm or less), (2) filter element that either has activated charcoal or is impregnated with silver or iodine for local antibacterial action, (3) pump-feed mechanism that forces water through the filter housing, (4) device that can be easily disassembled and cleaned for proper maintenance, and (5) product having durability and simplicity of use. Noteworthy filters that have most of the desirable features include those from Katadyn, PUR, and MSR WaterWorks.

Bicycling. Several prepackaged medical kits for the cyclist can be purchased. People who want to upgrade a basic first-aid kit to one for treating cycling injuries should consider the abrasions and minor burns likely to be encountered. Proper wound management requires a protective pad that is nonadherent, cools the skin, and absorbs exuded fluids. A number of breathable water-based gels, such as Spenco Second Skin, exist for this purpose.

Mountain Climbing and Hiking. In wilderness travel above the tree line or on shipboard, the inclusion of more advanced splinting products should be considered. A long-bone SAM Splint weighs only 4 ounces. The foam-coated aluminum strip unfolds to provide rigid longitudinal support. It can also be configured as a cervical collar, although the injured neck may require additional immobilization. At least two SAM Splints are needed to stabilize an entire extremity. The SAM Sling is a force-controlled circumferential pelvic sling belt for reduction and stabilization of pelvic fractures. The Slishman Splint is a lightweight splint made of a pulley system and collapsible ski pole used as a lower extremity traction device for femur and hip fractures. The Kendrick Traction Device is similar but uses a dedicated pole and nylon straps. It can also be used for shoulder reductions, skiing, or hiking. Ankle injuries are common, and air-inflatable or gel-filled splints provide stability and fit comfortably in a wide range of footwear (e.g., Aircast).

Items Stored in the Vehicle

When feasible, a complete emergency kit in the vehicle is highly recommended (see Box 81-8). It will facilitate further stabilization of an injured person evacuated to a trailhead. The vehicle kit should also provide material necessary to manage accidents encountered along the highway and to cope with the environment if the occupants are stranded by automotive trouble or natural disaster. Several large burn dressings and a neck-board with strapping will fit in a standard trunk or other recess. Although only large vehicles can accommodate the usual full-length backboard, a folding backboard is now available from Junkin Safety Appliance (Appendix B). The remaining contents of an emergency kit will fit in a medium (6 inches wide × 12 inches long × 9 inches high [18 × 37 × 27 cm]) or large (8 inches wide × 18 inches long × 14 inches high [24 × 55 × 43 cm]) war surplus ammunition box or a toolbox of similar size (Fig. 81-4).

Part 3: Medications

Physicians taking care of travelers will often be asked to prescribe medicines that will be used or withheld based on the judgment of someone who is not a physician (Fig. 81-5). Patients should be educated about the appropriate and inappropriate use of medicines and encouraged to consult a local physician or communicate with a physician from their country of origin before taking any prescription medications. When assembling medicine kits for a group, always include copies of the manufacturer's package insert with each medicine. For more than legal reasons, it is advised to prescribe medications only for patients with whom the doctor has met. Other group members should visit their own health care providers. Although sharing of medications within a group of travelers is common and may be appropriate, it is not an activity for which the prescribing physician should be responsible unless the doctor is acting as an adviser to the entire group. All physicians should be aware of their potential liability when traveling with, prescribing medications for, or advising a travel group from a remote location.

Table 81-2 lists commonly carried over-the-counter and prescription medications. This list is not comprehensive, but provides options from which a medical designee can choose. A number of the medications have significant systemic effects (e.g., atropine, epinephrine, dexamethasone, nifedipine, nitroglycerin). Administration of these medications should generally be limited to physicians, nurses acting under the direction of a physician, or designated medical providers with appropriate training.

Injectable drugs are rarely needed for the management of wilderness-associated trauma and illness because most emergencies can be managed by either oral or transdermal application of medicines. IV medications are temperature sensitive and fragile, expire quickly, and require monitoring of vital signs because of potency and immediate onset of action.

Figure 81-4. First-aid kit for vehicles. Medications are stored in zipper-lock bags with dosing instructions. Each kit has an inventory card. A small kit such as this is appropriate for short excursions and while driving in a caravan.

Figure 81-5. Medications. See Table 81-2 for a list of commonly carried over-the-counter and prescription medications. (Photograph by Matthew R. Lewin.)

Opioid analgesics should be used to treat pain only if mild analgesics such as ibuprofen and acetaminophen are inadequate, mental status is clear, and respiratory distress is not present (unless it is due solely to discomfort). Oral opioids are appropriate for moderately severe pain. More potent, transdermal delivery systems of opioids (e.g., fentanyl patch) are useful, especially when oral administration is not possible. Opioids and dehydration can predispose to constipation, an uncomfortable and potentially dangerous problem. Anyone initiating opioid therapy should be advised to take a stool softener such as docusate sodium. Any expedition carrying opioids should also have a reversal agent such as naloxone. Opioid and opioid antagonist preparations such as pentazocine (Talwin) are also available and may reduce the possibility of abuse.

Whenever possible, medicines should be purchased as tablets, not capsules, because of the tendency for capsules to break apart or dissolve (Fig. 81-6). Under extremes of temperature, creams may become unusable; in such environments, oral medications are preferred. For example, creams for genital yeast infections have a tendency to separate, whereas oral antifungals such as fluconazole, although more expensive, are compact and stable and may be discretely and hygienically administered.

Purchasing medications on arrival may be necessary and may save money. Many medications that require prescriptions for purchase in industrialized countries are available over-the-counter in developing countries, including opioids. However, a recent study of the quality of antibiotics in a developing country found that in about 30% of the products studied, the drug was either not present or present in lower dosages than stated.[14] If treatment failure poses a significant health threat, the traveler should make every effort to obtain the medication from a reliable source. The advice of a local physician may be helpful.

In an attempt to reduce costs, expedition members may wish to use expired medications. The use of expired medications is discouraged unless there is an experienced pharmacist or physician available to guide their use. Whether expired medications should be used depends on two factors: stability and safety. Stability refers to the tendency of a drug to become less potent over time. Manufacturers provide an expiration date up to which point the drug will retain greater than 90% of its potency if kept in its original closed container. Once opened, the expiration dates on bulk containers no longer apply, but retail pharmacies typically label repackaged drugs with a "beyond-use" date that is generally 1 year from the date the prescription was filled. Many drugs maintain potency greater than 90% for long after the expiration date. For example, ciprofloxacin can remain stable for several years after the expiration date, even under conditions such as high temperature and humidity. Passing an expiration date does not indicate that the drug becomes harmful to use. One known exception is tetracycline antibiotics, which are known to degrade into toxic metabolites that can damage the kidneys.[6] The tetracycline class of antibiotics, which

Text continued on p. 1912

TABLE 81-2. Sample Medications for Wilderness Travel

DRUG	DOSAGE	INDICATIONS	CONTRAINDICATIONS AND ADVERSE EFFECTS	COMMENTS
Analgesics				
Acetaminophen 500-mg tabs (Tylenol, paracetamol)*	500 mg PO q 4–6 hr, max dose 4 g/24 hr	Pain relief, decreases fever	Chronic or acute overdose can cause liver injury.	Many cold and flu remedies contain acetaminophen and should be accounted for when consuming acetaminophen. Paracetamol is metabolized to acetaminophen, and its combination with acetaminophen can be toxic.
Bupivacaine 0.25% solution (Marcaine, Sensorcaine)	Max dose 1.5 mg/kg	Local anesthesia, injectable	Use with caution on feet because it may allow further skin damage to go unnoticed; hypotension and dysrhythmias can occur at toxic doses.	Injections around nerves can be effective for tooth pain, extremity fractures; may last up to 10 hr. Cardiotoxicity is greater than that of lidocaine at equivalent doses. May be used to soak gauze for topical and dental applications.
Codeine 60-mg tabs	15–60 mg PO q 4–6 hr	Pain relief	Not to be used while driving; may cause respiratory depression and decreased alertness.	Codeine and morphine have large therapeutic windows and should be titrated to effect.
Fentanyl 400-µg lozenges (Actiq)	400 µg PO/SL q 2–6 hr	Pain relief	Same as codeine	
Fentanyl patches (Duragesic)	25–100 µg/hr for 72 hr	Pain relief	Same as codeine	May combine more than one patch to achieve correct dose.
Hydrocodone/APAP 5-mg/500-mg tabs (Vicodin, Lortab)	5 mg/500 mg q 4–6 hr, max dose 8 tablets/24 hr	Pain relief	Contains acetaminophen, may cause severe constipation.	Do not give large amounts to patients who do not comprehend risk for acetaminophen toxicity.
Hydrocodone/APAP 10 mg/325 mg tabs (Norco)	10 mg/325 mg q 3–6 hr, max dose 12 tablets/12 hr	Pain relief	Same as above, except contains less acetaminophen	Higher concentration of opioid than in opioid/acetaminophen above
Ibuprofen 200 mg tabs (Advil, Motrin)*	200–800 mg PO q 6 hr	Pain relief	Not to be used if gastrointestinal bleeding, pregnancy, or dehydration is present.	This medication can be combined with acetaminophen, codeine, or morphine.
Lidocaine 0.5%–2% injection solution with or without epinephrine (Xylocaine)	Max dose 4 mg/kg	Local anesthetic	Conventional teaching is that lidocaine with epinephrine should not be used when injecting distal extremities (finger tip, penis, nose).	Shorter acting than bupivacaine; may be used as advanced cardiac life support (ACLS) drug in setting of cardiac arrest. Toxic doses may result in seizure or cardiac arrest.
Lidocaine jelly 2%, 5-mL packages	Apply to skin PRN or 30 min before procedure	Pain relief, prophylaxis for catheter insertion		May be used as part of a "GI cocktail" for upset stomach.
Morphine sulfate 20 mg/mL	2–10 mg IV/IM q $^1/_2$ to 6 hr	Pain relief	Same as codeine	Concentrated formulations such as hydromorphone (Dilaudid) are easier to give IM.
Naloxone (Narcan)	0.4–2.0 mg SC, IM, IV or by endotracheal tube q 2–3 min	Opioid overdose	May precipitate opioid withdrawal in chronic opioid users.	Rare adverse effects include pulmonary edema and seizure; may be helpful for some types of neuropathic pain.
Pentazocine (Talwin)	50 mg PO q 3–4 hr	Opioid agonist-antagonist	Still has abuse potential.	Also comes in IV/IM formulation with recommended starting dose 30 mg q 3–4 hr for an adult >50 kg.
Antimicrobials				
Amoxicillin/clavulanic acid 825-mg/125-mg tabs (Augmentin)	875 mg/125 mg PO bid	Animal bites, oral infection	Contraindicated in patients with penicillin allergy.	All animal bites should be irrigated with a large volume of clean water.

TABLE 81-2. Sample Medications for Wilderness Travel—cont'd

DRUG	DOSAGE	INDICATIONS	CONTRAINDICATIONS AND ADVERSE EFFECTS	COMMENTS
Azithromycin 250 mg tabs (Zithromax)	500 mg PO ×1 day, then 250 mg PO×4 days	Pneumonia, otitis media	May cause nausea and vomiting.	May use in combination with cephalosporin. If treating traveler's diarrhea, dosage is 500 mg PO daily for 3 days. Erythromycin is a less expensive alternative but has more GI side effects.
Cefazolin solution (Ancef, Kefzol)	1 g IM/IV q 6 hr	Soft tissue infection, dirty wounds, crush injuries, open fractures	Penicillin allergy is a risk factor for cephalosporin allergy.	
Ceftriaxone solution (Rocephin)	1–2 g IM/IV q 12–24 hr	Pneumonia, meningitis, gonorrhea		Consider dexamethasone IM or IV injection in patients with suspected bacterial (particularly meningococcal) meningitis.
Cephalexin 500-mg tabs (Keflex, Keftab)	500 mg PO qid	Soft tissue infection, dirty wounds, crush injuries, open fractures		This is the oral equivalent of cefazolin.
Chloroquine 500-mg tabs (Aralen)	500 mg PO q wk for prevention; 1500 mg PO on day 1, then 500 mg PO daily × 2 days for treatment	Malaria in chloroquine-sensitive areas (parts of Central America, Caribbean, and Middle East)	May cause nausea, diarrhea.	Chloroquine resistance is common; information on local resistance patterns is available at www.cdc.gov.
Ciprofloxacin 500-mg tabs (Cipro)	500 mg PO bid	Traveler's diarrhea, urinary tract infections	Not recommended for patients under age 16 years because of risk for cartilage injury	Associated with Achilles' tendon rupture in adults.
Clindamycin 150-mg tabs (Cleocin)	450 mg PO q 6 hr	Anaerobic infections: oral or aspiration pneumonia	Can cause *Clostridium difficile*–mediated toxic diarrhea.	Also useful for suspected methicillin-resistant *Staphylococcus aureus* (MRSA) soft tissue infections.
Doxycycline 100-mg tabs (Vibramycin)	100 mg PO daily—bid	Malaria prophylaxis and treatment, fever of unknown origin	Not recommended in patients <8 years because of teeth staining.	Do not give with dairy products or antacids because drug binds to calcium, causing decreased absorption.
Fluconazole 150-mg tabs (Diflucan)	150 mg PO daily	Vaginal candidiasis, other fungal infections	Liver toxicity may occur.	A single dose treats vaginal candidiasis; higher, repeated doses treat systemic fungal infections; more convenient than nystatin preparations but requires a prescription.
Mebendazole 100-mg tabs (Vermox)	100 mg PO daily—bid	Intestinal nematodes	Not to be used by pregnant patients or children <2 years old	Frequency and length of treatment vary depending on worm type.
Mefloquine (Lariam)	250 mg PO weekly from 1 wk prior until 4 wk after exposure, or 1250 mg PO once for treatment	Chloroquine-resistant malaria prophylaxis and treatment	Prior adverse reactions to mefloquine, including hallucinations and night terrors	There are many alternative regimens for chloroquine-resistant malaria including atovaquone + proguanil (Malarone) and sulfadoxine + pyrimethamine (Fansidar); seek expert consultation for region-appropriate regimens.
Metronidazole 500-mg tabs (Flagyl)	250 mg PO tid × 5 days	Suspected giardiasis; severe diarrhea or diarrhea with fever, blood, or leukocytes	Alcohol consumption may cause disulfiram reaction. May increase toxicity of lithium, phenytoin, and anticoagulants.	In case of severe diarrhea, use in combination with a fluoroquinolone; check other medications for alcohol content.
Penicillin V 500-mg (Pen-Vee)	500 mg PO bid	Dental infections, streptococcal pharyngitis		

Continued

TABLE 81-2. Sample Medications for Wilderness Travel—cont'd

DRUG	DOSAGE	INDICATIONS	CONTRAINDICATIONS AND ADVERSE EFFECTS	COMMENTS
Quinine 325-mg tabs	650 mg PO tid × 7 days	Malaria in chloroquine-resistant areas	May prolong QT interval; IV form available if patient unable to tolerate PO.	Doxycycline 100 mg PO bid × 7 days should be given concurrently.
Rabies vaccine (RVA, RabAvert)	1 mL IM on days 0, 3, 7, 14, and 28	Postexposure rabies treatment		Wash wound. Give vaccine and immune globulin immediately if known rabies exposure.
Rabies immune globulin (Imogam, BayRab)	20 units/kg, as much as possible around the bite, the rest IM	Postexposure rabies treatment		Immune globulin is given <7 days after vaccine.
Trimethoprim-sulfamethoxazole 160 mg/800 mg tabs (Bactrim, Septra)	160 mg/800 mg tab PO bid	Urinary tract infections, traveler's diarrhea, soft tissue infections		Resistance to this medication is common in some areas.
Cardiovascular				
Aspirin 325-mg tabs (Bayer, ASA, Ecotrin)*	325 mg PO daily	Acute coronary syndrome	Not recommended for patients at high risk for bleeding.	
Atropine 1 mg/mL	0.5–1.0 mg IV q 3–5 min, max dose 0.04 mg/kg	Symptomatic bradycardia	Causes tachycardia and stool/urine retention.	Reduces respiratory secretions.
Clonidine 0.1 mg tabs (Catapres)	0.1 mg PO q 24 hr	Hypertension	May cause drowsiness and dizziness.	If a routine medication, stopping it may cause severe rebound hypertension.
Epinephrine 1 mg/mL	0.3 mg IM/IV q 30 min for anaphylaxis; 1 mg q 5 min for cardiac arrest	Anaphylactic reaction, hypotension, cardiac arrest, severe asthma	Because of cardiac stimulation, use with caution in elderly patients or patients with cardiac history.	Considered essential because of its use in anaphylaxis. Multiple doses may be required.
EpiPen or Twinject	Inject into extremity large muscle group, usually thigh	Anaphylactic reaction, hypotension, cardiac arrest, severe asthma		Single use only (see alternative, above).
Metoprolol 50-mg tabs (Lopressor, Toprol)	50 mg PO bid	Tachycardia in presence of ischemic symptoms	Do not give to patients with hypotension, bradycardia, or pulmonary edema.	
Nitroglycerin 0.4-mg sublingual tabs or spray (Nitrostat, Nitrolingual)	0.4-mg tab under tongue q 5 min	Angina, congestive heart failure exacerbation	Hypotension, potentially life-threatening interactions with drugs for erectile dysfunction (Viagra, Cialis, Levitra, Silagra)	Relief of chest pain with nitroglycerin does not identify pain as cardiac in origin.
Oxygen	Titrate to effect	Respiratory distress, acute coronary syndrome, other ischemic event		Tanks are explosive.
Central Nervous System				
Alprazolam 0.5-mg tabs (Xanax)	0.5 mg PO qhs	Insomnia, anxiety	Causes sedation and respiratory depression.	May increase dose to treat agitation or violent behavior; half-life is 6–23 hr; high abuse potential.
Dextroamphetamine 5-mg tabs (Dexedrine, Dextrostat)	5 mg PO q 8 hr	Fatigue, difficulty concentrating	May cause nervousness, diarrhea, or loss of appetite.	Only recommended for emergency use.
Diazepam 5-mg tabs (Valium)	5–10 mg PO bid–qid	Anxiety, agitation, seizures	Causes sedation and respiratory depression.	Similar to lorazepam but half-life of 1–3 days.
Dimenhydrinate 50-mg tabs (Dramamine)*	50–100 mg PO q 4–6 hr	Motion sickness	Sedation	

TABLE 81-2. Sample Medications for Wilderness Travel—cont'd

DRUG	DOSAGE	INDICATIONS	CONTRAINDICATIONS AND ADVERSE EFFECTS	COMMENTS
Haloperidol 5 mg/mL (Haldol)	2–5 mg IM or PO q 8–24 hr	Agitation, mania, psychosis, migraine	Dystonic reactions may occur; treat with diphenhydramine or benztropine (Cogentin).	This medication is very effective in providing temporary sedation; use need not be limited to patients with history of psychosis. Newer, but more expensive, antipsychotic formulations such as ziprasidone (Geodon) and olanzapine (Zyprexa, see below) may have lower incidence of dystonic reactions.
Lorazepam 2 mg/mL (Ativan)	2 mg IM/IV q 10 min (seizures) to q 12 hr (anxiety, insomnia)	Anxiety, agitation, seizures, alcohol withdrawal	Causes sedation and respiratory depression.	The appropriate dose varies considerably; titrate to effect. Half-life is 10–20 hr. Can be taken orally.
Meclizine 12.5-mg or 25-mg tabs (Antivert, Bonine)*	12.5–25 mg PO q 6 hr PRN (vertigo); 25–50 mg PO 1 hr before travel (motion sickness)	Vertigo, motion sickness prophylaxis	Causes sedation.	For motion sickness prophylaxis, may repeat as needed q 24 hr; pregnancy class B.
Olanzapine (Zyprexa) 10-mg tabs	5–20 mg PO, SL, or IM q 24 hr	Agitation, mania, psychotic disorders	Can cause tardive dyskinesia, neuroleptic malignant syndrome, orthostatic hypotension, and hyperglycemia.	Orally disintegrating tabs are useful for acute episodes; not for use in dementia-related psychosis.
Dermatologic, Oral, and Topical				
Clotrimazole 1% cream (Lotrimin, Mycelex)*	Apply to skin bid	Topical fungal infection		May be used in combination with oral fluconazole.
Hydrocortisone 1% cream (Cortaid, Hytone)	Apply bid–tid	Contact dermatitis	Not for infectious rashes; repeated use can cause skin atrophy especially on face, genitals, and dorsum of hands.	
Triamcinolone 0.5% cream (Aristocort, Kenalog)	Apply bid–tid	Contact dermatitis, severe itching	Not to be used for infectious rashes; repeated use can cause skin atrophy, especially on face, genitals, and dorsum of hands.	Many substitutes are available, including betamethasone, fluocinolone, and halcinonide; high-potency formulations are recommended.
Mupirocin (Bactroban)	Apply bid	Infected or contaminated wounds; suspected nasal MRSA		Apply bid to inside of nares for 5 days for suspected MRSA.
Diphenhydramine 50-mg tabs (Benadryl)*	50 mg PO q 4–6 hr	Allergies, allergic reactions, dystonic reactions	Causes sedation, not recommended while driving.	Nonsedating antihistamines are also available.
Metronidazole (Metrogel—vaginal)	0.75% 1 applicator qhs for 5–7 days	Bacterial vaginosis	Safe in pregnancy	Formulation tends to separate at high temperatures; consider oral formulation for more compact and stable transport.
Nystatin vaginal suppositories and creams (Mycostatin)*	100,000 units qhs for 14 days for vaginitis; bid—tid application for skin infection	Vulvovaginal candidiasis, balanitis, localized skin infections	Safe in pregnancy	Creams and suppositories tend to melt in warm or hot environments; fluconazole is more stable and can be taken as a single oral dose, but it is pregnancy class C and requires a prescription.

Continued

TABLE 81-2. Sample Medications for Wilderness Travel—cont'd

DRUG	DOSAGE	INDICATIONS	CONTRAINDICATIONS AND ADVERSE EFFECTS	COMMENTS
Permethrin cream 5% (Elimite, Nix)	Cover body from neck down, wash off after 8 hr	Scabies; head lice may be treated with 1% permethrin rinse.	Temporary stinging; may repeat treatment in 7 days if unsuccessful	The group should be treated simultaneously; clothes and bedding should be washed.
Polymyxin, bacitracin, and neomycin ointment, 15-g packs (Neosporin)*	Apply tid	Lacerations, superficial skin infections		Not a substitute for systemic antibiotics for soft tissue infections; may be appropriate for some ophthalmic infections and injuries.
Silver sulfadiazine 1% cream (Silvadene)	Apply bid	Burns, large soft tissue injuries, open fractures		Remove old cream before reapplying; contains sulfa.
Tolnaftate 1% cream, gel, spray, or powder (Tinactin)*	Apply bid to affected area	Localized skin infections suspected to be fungal in origin (e.g., jock itch, athlete's foot)	Safety profile is unknown in pregnancy.	
Eyes, Ears, Nose, and Throat Topical Medications				
Cyclopentolate 1% eye drops (AK-Pentolate, Cyclogyl)	2 drops q 24 hr	Snow blindness	Not to be used in patients who need to walk or drive; may cause acute angle-closure glaucoma.	Cyclopentolate decreases pain by decreasing ciliary muscle spasm.
Dexamethasone + neomycin + polymyxin ointment (Maxitrol)	1–2 drops tid–qid or $^1/_2$-inch ribbon bid	Snow blindness, disabling allergic conjunctivitis		Cortisporin (neomycin + polymyxin + hydrocortisone) is interchangeable with Maxitrol for otic and ophthalmic irritation.
Erythromycin ophthalmic ointment 0.5% (Ilotycin) 3.5-g tube	Ribbon in affected eye before bed	Corneal abrasion, ulcerations	Ointment stays on eye but blurs vision; use at night or while resting.	May substitute bacitracin, mupirocin, polymyxin, or neomycin ointments.
Gentamicin (Garamycin) or tobramycin (Tobrex) drops	2 drops hourly × 24 hr, then reduce to qid	Corneal abrasion, ulceration	May cause chemical keratitis.	For ulcerations due to contact lenses, discard contacts and case; do not use new lenses until well healed.
Ofloxacin 0.5% otic solution (Floxin Otic)	10 drops bid	Otitis externa		A wisp of cotton wool placed in the ear as a wick will draw medication into ear canal.
Oxymetazoline 0.05% nasal spray (Afrin)*	2 drops each nostril bid × 3 days	Congestion, epistaxis		May be sprayed on lacerations to temporarily decrease bleeding. Causes rebound congestion with prolonged use.
Polymyxin B and trimethoprim eye drops (Polytrim)	2 drops q 3–6 hr	Corneal abrasions or ulcerations, snow blindness	Sulfa allergy	Ointments can be used instead of drops but impair vision and should only be used when patients are resting or sleeping. Worsening eye irritation suggests chemical keratitis caused by medication.
Prednisolone 1% eye drops (AK-Pred, Pred Forte)	1 drop q 1–12 hr	Allergic keratitis	Corneal infections	Because steroids may worsen eye infections, they are typically only prescribed with advice from an ophthalmologist.
Proparacaine 0.5% eye drops (Alcaine, Ophthetic)	1–2 drops	Facilitate eye examination, temporary relief of eye pain	Repeated use may cause worsening of infection or abrasion.	Complete relief of eye pain with application of proparacaine suggests that pathology is limited to the cornea.
Sulfacetamide 10% ointment or drops (Sulamyd)	2 drops or $^1/_2$ inch ribbon tid–qid for 7–10 days	Blepharitis, conjunctivitis	Sulfa allergy	May also be used in setting of snowblindness and corneal abrasions.

TABLE 81-2. Sample Medications for Wilderness Travel—cont'd

DRUG	DOSAGE	INDICATIONS	CONTRAINDICATIONS AND ADVERSE EFFECTS	COMMENTS
Gastrointestinal				
Bismuth subsalicylate 262-mg tabs (Pepto-Bismol, Kaopectate)	262-mg tabs PO q 1 hr, max 8 doses/day	Abdominal pain, vomiting, diarrhea	Stool and tongue may turn black; excessive intake can cause salicylate poisoning.	May provide good GI prophylaxis from coliform infections if taken before meals.
Docusate sodium 100-mg tabs (Colace)*	100 mg PO q 6–24 hr	Constipation	May cause diarrhea, abdominal cramping	Works by softening stool. Polyethylene glycol (MiraLax), Senna (Senokot), and myriad other agents are available. Liquid Colace can be used to remove earwax.
Famotidine 20-mg tabs (Pepcid)*	20 mg PO qhs—bid	Peptic ulcer disease	Decreased stomach pH may predispose patient to gastrointestinal infections.	Consider treatment with tetracycline and metronidazole for *Helicobacter pylori* infection.
Lactase (Lactaid)*	1–3 pills with first bite of dairy	Lactose intolerance		α-Galactosidase (Beano) is useful for more general coverage of gassy foods.
Loperamide 2-mg tabs (Imodium)*	4 mg PO initial dose, then 2 mg PO to max 16 mg/day	Diarrhea	Not recommended for patients with bloody diarrhea	Decreases symptoms in most cases of traveler's diarrhea, may prolong infection in patients with enteroinvasive diarrhea.
Omeprazole 20-mg tabs (Prilosec)*	20–40 mg PO q 24 hr	Peptic ulcer disease	Decreased stomach pH may predispose patient to gastrointestinal infections.	For duodenal ulcer, start at 20 mg/day; for suspected stomach ulcer, 40 mg/day for 14 days.
Ondansetron 8-mg tabs or 4 mg/5 mL (Zofran)	8 mg PO or 4 mg IV q 8 hr	Nausea, vomiting		This newer, more expensive agent likely causes fewer dystonic reactions than phenothiazine antiemetics.
Prochlorperazine 25-mg suppositories (Phenergan)	25 mg PR bid	Nausea, vomiting	Causes sedation and dystonia. Not for patients <2 years old.	Acute dystonic reactions can be treated with diphenhydramine or benztropine.
High Altitude				
Acetazolamide 250-mg tabs (Diamox)	250 mg PO bid	Acute mountain sickness (AMS) treatment (125 mg for prevention)	Contraindicated in patients with sulfa allergy; may cause vertigo, diuresis-induced hypovolemia, and paresthesias.	Test dose before trip (for sulfa allergy). Begin preventive treatment on first day of ascent and stop at maximum elevation; makes beer taste flat.
Dexamethasone 4 mg/mL in 1-mL vials or 4-mg tabs (Decadron)	2–4 mg IM/IV q 6 hr, initial dose of 8 mg for high-altitude cerebral edema (HACE)	HACE	May cause agitation and mood disturbances.	Onset in 2–8 hr, can be lifesaving for HACE; may be substituted for prednisone and vice versa in an emergency, but doses are not equivalent.
Nifedipine extended-release 30-mg tabs (Procardia, Adalat)	30-mg tab PO bid	High-altitude pulmonary edema (HAPE)	Do not give if hypotensive.	Can be used to treat hypertension.
Sildenafil 50-mg tabs (Viagra)	50 mg PO q 24 hr	May enhance cardiovascular performance at high altitude, but still experimental	May cause hypotension, headache, lightheadedness, and blue scotomata. Not for patients taking nitrites or with a history of retinal disease.	Benefit demonstrated during single use; unknown benefit with repeated use; possible but unknown benefit for HAPE[†]
Respiratory				
Albuterol inhaler (Ventolin, Proventil, Salbutamol)	2 puffs q 4 hr	Asthma exacerbation, chronic obstructive pulmonary disease	May cause tachycardia or provoke anxiety.	In managing acute exacerbations of asthma, albuterol may be given continuously.

Continued

TABLE 81-2. Sample Medications for Wilderness Travel—cont'd

DRUG	DOSAGE	INDICATIONS	CONTRAINDICATIONS AND ADVERSE EFFECTS	COMMENTS
Beclomethasone 40 µg/dose aerosol (QVAR, Vanceril)	1–4 puffs bid	Chronic asthma		One of many inhaled steroids for chronic asthma; may have fewer systemic side effects than oral steroids.
Epinephrine—aerosolized 0.22 mg/puff (Primatene Mist)*	2 puffs every 4 hr PRN for asthma exacerbation	Asthma exacerbation, allergic reactions, anecdotal use for symptomatic bradycardia	Use with caution in older adults and those with known coronary disease.	
Fexofenadine 60-mg tabs (Allegra)	1 tab PO bid	Allergy, urticaria		Nonsedating antihistamine useful for treatment of nasal congestion and allergy-induced itching; it costs more than diphenhydramine but CNS effects are less pronounced.
Ipratropium 18 µg/puff (Atrovent)	2–3 puffs qid	Asthma exacerbation	Not for patients with life-threatening soy or peanut allergies.	Available in combination with albuterol (e.g., Combivent).
Prednisone 20-mg tabs (Deltasone, Pred-Pak)	60 mg or 1 mg/kg/day × 3–5 days	Asthma exacerbation, allergic reactions	Short-term side effects include insomnia and nervousness.	Also used to treat autoimmune diseases; use taper to stop after long-term treatment.
Throat lozenges*	1 lozenge PO q 10 min PRN	Sore throat, cough		Many types available; Ricola cough drops are preferred by many.

*Does not require a prescription in the United States.
†Kleinsasser A, Loeckinger A: Are sildenafil and theophylline effective in the prevention of high-altitude pulmony edema? Med Hypotheses 59:223–225, 2002.

Figure 81-6. Gelatin capsules exposed to heat and rough roads over the course of one expedition. When possible, obtain pills rather than capsules. The manufacturer's drug information is attached to the top of the bottle.

includes minocycline and doxycycline, is also associated with acute nephropathies in nonexpired forms. For this reason, they should be used with caution in older patients and those with known renal insufficiency. For medications that are dosed to effect, such as those to treat pain, gastric, respiratory, and psychiatric symptoms, decreases in potency usually present little danger to the patient. For medications with specific recommended doses, such as antibiotics and treatments for high-altitude illnesses, decreased potency could result in ineffective treatment of a serious condition. In all cases, recently expired medications are preferable to long expired ones.

Gathering large quantities of medications, particularly controlled substances, can be difficult and expensive. As with any other aspect of an expedition, advanced planning lessens frustration as departure draws near. Soliciting the help of an experienced pharmacist can be helpful, if not essential, to maximizing a medical kit's pharmacologic armamentarium.

Passing through Customs with Medications

Each medication should be labeled with the patient's name, prescribing physician's name, dosing schedule, indications, and warnings. In most parts of the world, individuals carrying properly labeled medications receive little attention at customs. For those leading a group and carrying its medications, appropriate documentation is important. To facilitate passage through customs, all medication bottles should be labeled appropriately, including "*For use by the __ Expedition only.*" Medical designees should carry multiple copies of their credentials and letters of purpose from their sponsoring institutions. A letter from the sponsoring institution or company designating the carrier of the medications as the group's medical officer or physician will usually suffice.

Inevitably, something will be overlooked or needed unexpectedly. A visit to a local hospital or clinic may require creative pantomime and drawing. These visits will provide local

TABLE 81-3. Indications for Antibiotic Treatment in Wilderness Travelers

Traveler's diarrhea—sensitivity to various antibiotics varies based on region and should be researched before departure (e.g., there is high resistance to ciprofloxacin by *Campylobacter* species in the Himalayan region).	Ciprofloxacin, 750 mg PO × 1, *or* azithromycin, 1 g PO × 1, if diarrhea treated within first 24 hours. If diarrhea greater than 24 hours' duration, then: azithromycin, 500 mg PO daily × 3 days, *or* ciprofloxacin, 500 mg PO bid × 3 days. Antimotility agents should not be given in the setting of dysentery, fever, or other abnormal vital signs.
Diarrhea (suspected giardiasis)	Metronidazole, 250 mg PO tid × 5 days
Diarrhea in pregnant women or children	Trimethoprim-sulfamethoxazole DS, 1 tablet PO bid × 3 days
Fungal infections (e.g., yeast vaginitis)	Fluconazole, 150 mg PO × 1
Lacerations from animal bites	Amoxicillin–clavulanic acid, 875/125 mg PO bid × 3–5 days (recommendation is controversial)
Lacerations with gross contamination, or bone, tendon, or cartilage exposure	Cephalexin, 500 mg PO qid × 3–5 days
Pneumonia	Doxycycline, 100 mg PO bid × 7 days, *or* azithromycin, 500 mg PO day 1 and then 250 mg daily days 2 to 5, *or* levofloxacin, 500 mg PO daily, *or* moxifloxacin, 400 mg PO daily
Sexually transmitted urethritis	Ciprofloxacin, 500 mg PO × 1, *and* azithromycin, 1 g PO × 1, *or* doxycycline, 100 mg PO bid × 7 days
Urinary tract infections	Ciprofloxacin 250 mg PO bid × 3 days
Appendicitis or other intra-abdominal infection	Ciprofloxacin, 500 mg PO bid, *and* metronidazole 500 mg PO qid

medical contacts and help identify appropriate sites for donation of medications and equipment upon return. Urban areas where expeditions and tours are often staged can provide a good opportunity to supply forgotten, lost, or confiscated materials. Sterile items such as disposable needles, IV tubing, and catheters may be much less expensive. However, it should not be taken for granted that they will be available or fit correctly on items purchased before international travel.

A dictionary of the local language is essential. Many developing countries have hospitals and training modeled after countries that once occupied them. Thus, when traveling in Central Asia (e.g., Kazakhstan or Mongolia), a Russian dictionary is helpful because a high percentage of pharmacists and physicians speak Russian and read Cyrillic. Similarly, local medical providers in many parts of Africa and Southeast Asia will be familiar with, or perhaps fluent in, French.

Part 4: Preparing for Common Medical Problems

Thresholds for administering medications, including analgesics, sleep agents, and antibiotics during travel depend on practice style, stocks, and evacuation options. However, treatment thresholds tend to be lower (more aggressive) in the wilderness than at home. A review of common indications for antibiotic therapy is provided in Table 81-3. To ensure timely care, all trip members should feel comfortable seeking consultation or clarification from the expedition's medical designee. A secure log of all patient encounters is important for both the patient and the physician. The following paragraphs discuss the evaluation and treatment of common medical problems in the wilderness setting; the last few paragraphs relate specifically to medical problems and considerations in underdeveloped countries.

Gastrointestinal and Genitourinary Care

Groups camping in a delicate ecology or near freshwater should use a lightweight, portable commode with sturdy, disposable plastic bags. Urine containers may be appropriate if prolonged adverse weather is a possibility. Funnel-like devices that connect to urine containers (e.g., Lady-J, available from Campmor; see Appendix B) are helpful for women. People embarking on long journeys should be aware that their normal bowel habits are likely to change. Lack of public sanitation may compel the more fastidious to bring their own supplies of toilet paper, baby wipes, and items for the treatment of perianal itching and hemorrhoids, such as pads saturated in witch hazel (e.g., Tucks). Other familiar brand-name formulations include Anusol and Preparation H. Women should predict their needs for absorbent pads and tampons based on their menstrual cycles. Some women who use oral contraceptive pills may change their cycles to avoid menstruating on long journeys. Women should discuss this possibility with their personal physicians several weeks before departure.

Diarrhea and Abdominal Pain

Diarrhea is a common problem for travelers in developing countries. Careful selection of foods and drinks can decrease the risk for infection. Bottled or boiled drinks and cooked or peeled foods are less likely to contain pathogens. Conversely, cold drinks, ice cubes, and raw or undercooked foods are more likely to contain pathogens. Fomites can be highly contaminated with feces even if people are fastidious; flies land on feces and transport pathogens from plate to plate. Treatment of traveler's diarrhea should include fluid replacement, antibiotics, and symptomatic treatment. Each participant should be encouraged to report the first loose stool. Treatment at the onset of symptoms can cut the duration of symptoms from 3 days to 1 day and minimize the opportunity to infect other members of the group. Patients with diarrhea should increase fluid intake, but sharing or preparing drinks or food for others is absolutely contraindicated.

Patients with diarrhea who are very young or very old, have significant underlying disease, or are unable to tolerate oral fluids should seek local medical attention and evaluation; parenteral fluids may be necessary in these individuals. Bacterial pathogens cause about 80% of acute traveler's diarrhea, which explains why antibiotic treatment is so effective. Traveler's diarrhea may be treated with 3 days of a fluoroquinolone, azithromycin, or trimethoprim-sulfamethoxazole. Sensitivity to various antibiotics varies greatly from country to country. It is

critically important to research this before departure. In the Himalayas, because a high percentage of *Campylobacter* species infections are resistant to ciprofloxacin, azithromycin is a more appropriate choice. Trimethoprim-sulfamethoxazole is adequate in certain areas of some countries, such as Mexico, with low levels of resistance; this drug is also a reasonable alternative to fluoroquinolones in pregnant patients and children. Chemoprophylaxis is generally not recommended but should be considered for patients who are immunosuppressed, have inflammatory bowel disease, have significant cardiac disease, are taking diuretics, or are on a "critical" trip. Loperamide given with ciprofloxacin has been shown to decrease diarrhea and is a recommended addition, except in patients with bloody diarrhea (dysentery), because of the risk for prolonging infection. Bismuth subsalicylate also decreases the number of loose stools. However, even bismuth preparations can cause illness if used inappropriately; its benign flavor and familiarity to most consumers may lead to salicylate poisoning. Overzealous consumption of bismuth subsalicylate can also cause stomach upset, pain, and tinnitus. Providing clearly labeled medications and emphasizing the importance of consultation and following instructions will help avoid complications.

Travelers with diarrhea and abdominal pain occurring within the first few weeks of arriving in a developing country usually have traveler's diarrhea. However, travelers and physicians should be aware that there are other pathologies of the abdomen, some of which require immediate medical attention. Diarrhea and fever may indicate typhoid fever, but it is important to be aware that this disease can also present with constipation preceding diarrhea or in the absence of diarrhea.

All women with abdominal pain without an obvious explanation should receive a pregnancy test; if positive, the patient should seek immediate medical attention given the risk for ectopic pregnancy. Women who know they are pregnant should consider ultrasound studies before travel to identify the location and number of pregnancies. Nonpregnant patients with abdominal pain and a stiff or rigid abdomen or abnormal vital signs should also seek immediate medical attention.

Proton-pump inhibitors and histamine-2 receptor antagonists, such as famotidine and ranitidine, are commonly prescribed for acid reflux and symptomatic treatment of dyspepsia. Both medications cause hypochlorhydria (decreased levels of stomach acid and elevated stomach pH), which compromises defense mechanisms of the upper gastrointestinal tract. Both appropriate and inappropriate use of these medications may predispose travelers to bacterial and parasitic intestinal infections.[15] Patients taking histamine-2 receptor antagonists and proton-pump inhibitors should be aware of their potential to increase the incidence and perhaps the severity of gastrointestinal infections. Temporarily stopping these medications during travel should be considered on a case-by-case basis.

Sexually Transmitted Infections

Travelers should know that, because of variations in reporting and surveillance, HIV is a risk even in regions where HIV is not considered prevalent. Anyone considering sexual activity with a new partner should carry a supply of lubricated, synthetic condoms. It is also prudent to carry condoms in the medical kit. Of note, unlubricated condoms can also serve as waterproof pill carriers, small tourniquets, and makeshift canteens (as used by the British military). All travelers of reproductive age should receive vaccinations for hepatitis B. Hepatitis A can be transmitted by fecal–oral route, and vaccines for this potentially serious disease are available alone or in combination with the hepatitis B vaccine.

Oral Hygiene and Health

Mild sore throat and a foul taste are common when traveling in the mountains and in cool weather, probably because of mouth breathing and enhanced loss of moisture from the upper respiratory tract. Both problems benefit from either hard candies or medicated lozenges (e.g., Cepacol, Chloraseptic). In addition, saline spray can keep nasal passages hydrated. Because treatment of early caries and loose fillings (which may trap expanding air) can prevent dental demise, each party member should have a dental examination before the trip. On the trail, frequent brushing may seem impractical; flossing after meals, rinsing well with water, and chewing sugar-free gum helps maintain oral hygiene en route. Toothache is common at high altitudes. For painful cavities or decay, temporary filling materials, such as Cavit, can be obtained in small squeeze tubes and applied to a tooth with a wet cotton applicator to prevent sticking.

Fractures and Dislocations

Splints can be improvised from cardboard, plaster, cloth, duct tape, water cans, and a phenomenal array of other materials. A number of lightweight splinting products, including moldable splints, ankle braces, and pelvic immobilizers, are now commercially available (see the mountain climbing and hiking section of Part 2 and Box 81-7). There are several types of cold packs: chemical, flexible, and rigid. Chemical cold packs are available at most wilderness and outdoor outfitters and should be used with caution when there is a break in the skin. Chemical cold packs are usually pouches of chemicals that when combined create a very cold chemical reaction. They can be useful if the group is away from a solar-powered cooler or refrigerator. Some cold packs have the potential to cause frostbite, and inattention to their application can cause or worsen injury. Flexible ice packs are pouches that contain small bits of frozen material such as ice chips or frozen peas or corn. Gel packs are stored in the freezer and might only be seen in large expeditions with solar or fuel-powered generators. Similarly, rigid ice packs are refillable and reusable.

Sleeping and Staying Awake

Difficulty sleeping during travel is common. Stress associated with a new environment and jet lag, a disruption in sleep pattern resulting from changing time zones, both contribute to this problem. Decreased sleep affects daytime performance and trip enjoyment. Medications that aid induction and maintenance of sleep can be useful. Oral diphenhydramine is generally a safe and effective soporific. A 50-mg dose results in sedation for about 6 hours. Diphenhydramine also causes mild mental impairment lasting about 2 hours and, because of its anticholinergic effect, can cause urinary retention in predisposed individuals. Benzodiazepines belong in a travel medical kit to treat seizures and painful injuries. They are also effective sedatives and may be used with discretion as a sleeping medication if diphenhydramine is ineffective. Patients should be warned about potential adverse effects and the possibility for abuse with benzodiazepines and other sleep remedies. Benzodiazepines should not be used with alcohol and are contraindicated in pregnancy (Pregnancy Class D) except in emergencies such as

seizures. Physicians prescribing benzodiazepines to women should document last menstrual period or require a pregnancy test in addition to detailed discussion about the risks and benefits of these medications when sexual activity and pregnancy are a possibility.

Additional problems in sleeping are experienced at high altitudes, where hypoxia, frequent awakenings, and periodic breathing are almost universal. Zolpidem (Ambien), temazepam (Restoril), acetazolamide, and theophylline have each been examined in the treatment of sleep problems at altitude. Drugs such as zolpidem and temazepam have been shown to subjectively improve sleep quality and decrease nocturnal variation in oxygen saturation in subjects at 5300 meters (17,388 ft).[7,16] In addition, acetazolamide and theophylline have been shown to decrease periodic breathing. Acetazolamide can significantly reduce nocturnal hypoxia from periodic breathing.[7,16] Climbers and trekkers who have difficulty sleeping at altitude should consider a trial course of acetazolamide. If acetazolamide is ineffective, then zolpidem or temazepam may be useful. Patients should be aware that the effect of benzodiazepines on daytime performance at high altitudes is unknown. Also, patients should be cautioned against the use of temazepam, alprazolam (Xanax), or other benzodiazepines during technical ascents because of the risk for impaired judgment and performance. Nevertheless, a supply of these medications should be available for emergency use.

Some travelers will encounter situations that require staying awake for an extended period; long drives and rescues are two possible scenarios. For these situations, caffeine-containing drinks or over-the-counter stimulant medications may be invaluable. Caffeine is also useful in the temporary relief of caffeine withdrawal headaches and is available in 200-mg tablets, equivalent to 2 cups of strong coffee. Caffeine use should be discouraged in patients with a history of seizures, high blood pressure, or heart disease. Because of the diuretic effect, patients using caffeine will need to consume additional fluids to stay well hydrated. Newer (but expensive) drugs such as modafinil (Provigil) are becoming popular, but have not been formally evaluated in the wilderness setting.

Blisters and Foot Care

Participants should bring footwear they have already worn and found comfortable. Socks should be kept clean and dry; a second pair is usually necessary. Identifying and addressing painful areas on the feet early can prevent blisters. There are many ways to prevent blisters by minimizing friction and pressure at hotspots using basic elements of the medical kit. Petroleum jelly on a gauze pad, covered by duct tape, is a reliable method of preventing or treating blisters. Also, duct tape placed on the inner lining of shoes decreases friction between the sock and shoe. A long-acting local anesthetic such as bupivacaine can provide exceptional relief if there is no alternative to walking and a blister injury must be overcome. However, local anesthesia may allow further tissue trauma to go unrecognized, so this is not a solution for blisters encountered at the beginning of a multiday trip. Finally, a small square of silk can be glued to the heel or other pressure point. This durable method of blister care is another example of simple, inexpensive alternatives to commercial preparations that may not be available or sufficiently durable in suboptimal conditions. Similarly, methyl acrylate–based glue can be used to repair skin fissures (Fig. 81-7). People who anticipate problems with blisters should consider reading John Vonhof's book, *Fixing Your Feet*, which provides detailed information on ways to protect the feet during extended travel (see Appendix C).

Figure 81-7. Methyl-acrylate glues may be useful for closing skin fissures and small lacerations. Skin glues should not be used to close lacerations in cosmetically significant areas such as eyebrows or eyelids.

Nail clippers are an important addition to the medical kit for people on extended journeys. For shorter trips, trimming toenails before departure reduces the likelihood of injury to the toenail and surrounding soft tissue. However, caution must be taken to evert the distal nail edges in people who are prone to ingrown toenails. Hikers whose feet sweat excessively may benefit from talc or medicated powder (e.g., tolnaftate [Tinactin]; see Table 81-2). Travelers in cold and aquatic environments are especially prone to dry skin. Petroleum jelly and skin creams such as Eucerin, Lubriderm, and Keri lotion may help forestall microtrauma and epidermal cracking.

Wounds

The medical kit should include tourniquets and a blood pressure cuff to help stop bleeding. Also, nasal sprays containing vasoconstrictors can help provide hemostasis. In some cases, particularly the scalp, closure of the wound with staples will provide quick hemostasis; thus, a small "ten shot" skin stapler (e.g., ConMed Reflex or 3M Precise) may be a useful addition to the medical kit.

Water for irrigation should be clean but does not need to be sterile; therefore, IV fluids should be conserved if rescue is not close at hand. Forcefully ejecting irrigant solution from a 20- or 35-mL syringe through an 18- or 19-gauge needle generates pressures adequate to dislodge bacteria and microscopic particles from contaminated wound surfaces. A squeezable bottle with holes punched in the top or a plastic bag with small holes suffices for lower-pressure irrigation. When appropriate, surgical excision of crushed, devitalized, or severely contaminated tissue can be achieved with sharp eyelet or suture-cutting scissors from the medical kit's surgical or bandage-cutting tools.

Needles and other surgical tools may be crudely disinfected by rubbing vigorously with a prepackaged towelette containing alcohol, chlorhexidine (Hibistat), povidone-iodine (Betadine), or benzalkonium chloride. After cleaning the instruments with alcohol or chlorhexidine towelettes, igniting the residual alcohol will provide additional sterilization. Flame sterilization to red-hot is also acceptable. Although less efficient, immersion

in boiling water (for about the same duration as one boils drinking water) will also adequately sterilize materials.

Significant advances in wound care include Tegaderm adhesive dressings and Spenco Second Skin. Tegaderm is a transparent 3- × 3-inch (9- × 9-cm) adhesive covering for clean wounds that provides a barrier to water, dust, and dirt while allowing oxygen to penetrate. It can remain in place for several days provided no wound complications arise. A polyethylene oxide gel laminate known as Spenco Second Skin reduces friction damage to the skin underlying blisters and burns; it may offer additional protection by redistributing pressure and absorbing exudates. It is a useful product to carry, especially if abrasion injuries are likely to occur.

Necessary quantities of dressings, gauze, and suture material can be calculated similarly to the way medications are calculated (see sample calculations in Part 5). However, the prevalence of traumatic injury during travel should be considered. Thus, it is not necessary to bring enough packing for 10 lacerations if there are 10 people on the trip.

Bites and Stings

In the United States, arthropods cause more venom-related fatalities than do snakes and other reptiles or amphibians. Insect stings usually kill because of acute, severe allergic reactions resulting in hypotension and airway compromise. Arachnid bites and stings can cause necrotic ulcers (e.g., *Loxosceles* species), abdominal wall tetany mimicking an acute abdomen (e.g., *Latrodectus mactans*), or respiratory compromise (e.g., *Centruroides* species).

Travelers should know the signs of and treatments for allergic reactions. Epinephrine preparations, diphenhydramine (Benadryl), and oral corticosteroids are essential components of the medicine supply. Individuals with a history of allergy to insect stings should carry their own epinephrine (EpiPen, EpiPen Jr). A new device, the Twinject autoinjector (Verus Pharmaceuticals, San Diego, CA.), carries two doses of injectable epinephrine, and is available with either two 0.15 mg doses or two 0.3 mg doses. For trained medical professionals, an inexpensive alternative is to carry an insulin syringe and a 1-mg ampule of epinephrine (Fig. 81-8). The benefit of this approach is that a 1-mL vial provides at least three doses of epinephrine in the case of a severe reaction or reactions in multiple patients. Because epinephrine ampules are glass, they require careful handling. Primatene Mist is an inexpensive and commercially available over-the-counter formulation of epinephrine. One puff delivers about 0.2 mg of epinephrine. Although Primatene Mist has been used for the treatment of asthma, this medication has not been studied for the treatment of acute allergic reactions. Although it might be a helpful "improvisation" treatment, it cannot be considered equivalent to subcutaneous or intramuscular epinephrine.

Topical corticosteroids are especially useful for insect bites and contact dermatitis. They may alleviate suffering and obviate the need for systemic drugs. Maximally potent fluorinated corticosteroid preparations (e.g., betamethasone, fluocinolone, fluocinonide, halcinonide) are recommended (see Table 81-2) for use under the harsh circumstances of wilderness travel; short-term use rarely causes any complications.

The topic of snakebite treatment and the proper first aid and medical management of snake envenomation remain highly controversial. Suction applied to the wound has theoretical appeal, but a recent study performing suction 3 minutes after a simulated bite removed less than 0.1% of the injected fluid from the wound.[2] Thus, purchase of suction devices for snakebite should be discouraged because their presence in the kit might be falsely reassuring. Current recommendations for the treatment of elapid snakebites from Australia and Southeast Asia emphasize the use of immobilization and a tourniquet or Ace bandage and splint to restrict lymphatic and capillary flow. Patients traveling to areas with elapids should be familiar with the immobilization and compression technique and should have the appropriate splinting equipment and technical training.

Figure 81-8. Three different preparations of epinephrine. On the left, Primatene Mist aerosol spray delivers about 0.2 mg epinephrine per puff. In the middle, a 1-mg vial of epinephrine and insulin syringe, and on the right, a single-use (0.3 mg) autoinjector device, EpiPen.

Travelers expecting frequent exposure to snakes might consider obtaining antivenom specific for the area of travel. All antivenoms, including the new formulation for rattlesnakes (CroFab), carry a significant risk for severe allergic reactions and should not be administered except by people who are properly trained and equipped to handle a severe allergic reaction. In countries with highly developed medical care, capturing or killing snakes for identification is probably unnecessary. However, in some countries, broad-spectrum antivenom is not available, and capturing or killing the snake may be the only way to guide specific therapy in the absence of species-identifying photographs or the presence of an expert herpetologist.

Sunburn, Snowblindness, and Sunglasses

Sunburn and snowblindness are entirely preventable injuries that occur because people underestimate the importance of sun protection. One can prevent sunburn with a variety of measures. Anyone even mildly susceptible to sunburn should use a sunscreen with sun protection factor (SPF) of at least 30. The efficacy of sunscreen, however, is diminished by sweat, water, wind, dirt, and friction inherent in wilderness travel. A hat and long-sleeved clothing plus sunscreen will prevent sunburn in most environments. Zinc oxide sunblock or facemasks provide additional protection in snow, high altitudes, and deserts.

In 1997, the incidence of snowblindness among trekkers and porters in central Nepal was about 3%, indicating the need for further education on this subject. Snowblindness, a burn injury to the cornea, results from cumulative exposure to sunlight. Patients experience symptoms about 6 to 10 hours after expo-

sure. These include pain, tearing, photophobia, and spasm of the muscles surrounding the eye with decreased visual acuity, injected conjunctivae, and diffuse punctate corneal lesions sometimes defined by the eyelids. Topical ophthalmic anesthesia provides pain relief and facilitates examination, but should not be given repeatedly because of the increased risk for ulcer formation. Snowblindness is very painful, but full recovery usually occurs in 24 to 72 hours. Short-term treatment includes prevention of further sun exposure, oral pain medications, short-acting cycloplegics to decrease ciliary spasm and pain, and topical antibiotics.

Optimal eye protection can be achieved using sunglasses with 100% ultraviolet (UV) light protection. The major variables to consider when buying sunglasses are durability, light transmission, photochromicity (darkening with increasing sun intensity), and polarization.

Although plastic lenses are lightweight, they scratch easily and are generally not recommended for extensive wilderness trekking. Glass lenses with the best photochromic properties can be coated for greater than 99% UV absorption, but they are heavy, susceptible to breakage, and moderately susceptible to scratching. The most durable lenses are made of polycarbonate and are lightweight and shatterproof. For this reason, polycarbonate is usually the preferred material for serious wilderness sports. Polycarbonate models that absorb at least 99% UV are manufactured by such brand names as Orvis, All Weather, Bolle, Coyote (polarized), Gargoyles, Gentex, Learjet, Ski-Optics, Suncloud, Transitions (photochromic), and Wings. Although polarization does not protect from UV light, it reduces glare and increases contrast, making it highly desirable for winter mountaineering, skiing, and aquatic wilderness sports. External clip-on plastic polarizers are available. Also, some soft contact lenses absorb UV radiation.

Because light reflects off snow and sand, sunglasses with side protection are recommended in these environments. Side shields, nose guards, and eye patches can be fashioned from almost any material. Because sunglasses are often forgotten, misplaced, or broken, for long trips, every member of the expedition should carry two pairs of sunglasses. Expedition physicians should carry extra pairs. Retention (neck) straps are recommended for virtually all outdoor activities. For water sports, a lanyard may be attached from the glasses' frame and over the back to a belt loop.

Foreign bodies in the eye may occur. Eye protection should be worn at all times when working with or in the vicinity of others using hand tools, machinery, or flammable substances. A loupe or other handheld lens can be very helpful for identifying ulcerations and removing corneal foreign bodies.

Pain

Most individuals and groups will want to carry at least an NSAID, such as ibuprofen, and an opioid, such as hydrocodone or acetaminophen with hydrocodone. Unlike more potent opioids, hydrocodone does not have the same degree of prescribing restrictions and should be easy to acquire and transport across borders. Ten milligrams of hydrocodone has the approximate effect of 5 mg of morphine. The combination of acetaminophen with an NSAID may be more effective than either medication alone. Many drug formulations contain acetaminophen, and no individual should consume more than 4 g of this drug in any 24-hour period (see Analgesics, Table 81-2). Overuse of pain medications and sedatives is dangerous, can impede travel, and will reduce their availability when they are actually needed. Opioid–antagonist combinations such as pentazocine (Talwin) may reduce abuse potential. For these reasons, access to opioid pain medications should be restricted to one or two group leaders if the supply is designated for group use). Any group carrying opioids should also carry an adequate supply of docusate sodium (Colace), senna (Senokot), or other agent to prevent constipation. (See also Chapter 17.)

Travel in Underdeveloped Countries

Travelers to underdeveloped countries face additional considerations. Traveler's diarrhea is common in Third World countries, and oral rehydration packets should be carried. Commercially prepackaged units contain essential elements that allow oral rehydration when combined with 1 L of disinfected water. Insect bites pose an additional risk for infection from a host of diseases. For travel outside of urban areas, the risks are especially great when sleeping unprotected outdoors. A lightweight bed net that can enclose two persons should be carried. Spraying these nets with permethrin further protects against mosquitoes. Several topical medications that combine an anesthetic (benzocaine) along with a cooling agent (menthol), such as Sawyer's Sting Aid or Itch Balm Plus, may help treat bites or minor allergic rashes.

In underdeveloped countries, donations of leftover, nonexpired medications to a local hospital or clinic are most often greatly appreciated, even in modest quantities. Meeting with a local pharmacist or clinic before departure can be helpful for arranging a donation and engendering good will should someone in the group need it. Donation of expired medications to even the most impoverished regions is generally considered taboo. However, physicians traveling to areas with limited health care resources should be encouraged to discuss the donation of expired medications with an accepting pharmacist or physician because the benefits of old medications likely outweigh the risks of using less potent forms, especially when the alternative is nontreatment.

Related to the subject of donating medications is the issue of providing medical care to local people encountered during an expedition. Often the medical designee, recognizing a significant illness in a local person, will want to provide care. Such interventions are generally beneficial and without significant cost to an expedition. However, the medical designee should keep in mind that this care is in almost all cases temporary and may affect the perceived value of local healers or the existing medical system. In addition, owing to lack of medical translation experience, the group's translator may not be able to effectively communicate instructions to patients, which may lead to dangerous misunderstandings. For example, nitroglycerin sublingual tabs might be given to a nomad with stable angina for "chest pain." The recipient might perceive this as a general pain medication and give it to a child with headache or another adult with aortic stenosis. Nevertheless, the medical designee will be expected to provide care for both native and foreign members of the expedition and must plan accordingly, unless the hosting team is accompanied by a local health care provider. Because these issues will directly affect the assembly of the kit, they should be discussed with the group leader in detail before departure.

Part 5: Strategies for Assembling Medical Kits

A medical kit can be assembled by customizing kits already available or starting from scratch. Budget and temporal con-

TABLE 81-4. Repair Supplies to Consider for Wilderness Travel

ITEM	DESCRIPTION	USES, COMMENTS
Needle or sewing awl	With heavy thread (1 oz [30 g])	Clothing and pack repair
Screwdrivers	Flat and Phillips No. 2 (3–6 oz [88–177 g])	For skis, No. 3 "posidrive" (or filed-down No. 2 Phillips)
Duct tape or reinforced strapping	1–2 inches (2.5–10 cm) wide, 5 yards (1.5 m) (2 oz [59 g]) (per person per trip)	
Wire and nylon cord	Braided steel and braided nylon 3–6 ft (0.9–1.8 m) (2 oz [59 g])	Repair of binding, boot, snowshoe, and pack
Needle-nose pliers with wire cutter		
Vise-grip pliers	5-inch (13 cm) (5–8 oz [148–237 g])	
Awl	One multifunction knife (2 oz [59 g]) (per person per trip)	Repair of clothing, pack, and shelter
Glue	Two-component epoxy, or meltable nylon glue stick (1 oz [30 g])	
Spare bale and screws	Two	Repair of ski binding
"P-tex" ski base stick	Meltable No. 1 (1 oz [30 g])	Repair of plastic ski base
Spare ski tip	Plastic or aluminum (3–5 oz [88–148 g])	
Spare crampon wrench	No. 1 (one)	
Knife sharpener	Diamond-bar, ceramic, or stone (2–3 oz [59–89 g])	

straints, as well as skill and differing levels of training, influence this process. As with any area of wilderness medicine, resourcefulness and opportunism may aid in the endeavor. Each New Year, catalog and Internet-based outfitters have clearance sales including many of the items discussed in this and other chapters. In addition, high-quality and normally expensive items such as laryngoscopes, water-tight boxes, and sunglasses are available in new and used condition on popular public websites such as www.eBay.com and www.craigslist.com. However, the reader is cautioned to purchase only from reputable individuals and businesses and to inspect all purchases for integrity and functionality.

Large kits must be well-organized to allow timely access to equipment and medicine. A water-tight aluminum or plastic box is a preferable container. The underside of the lid is an ideal place to store equipment for treating immediately life-threatening emergencies (e.g., treatment of respiratory distress or failure, altered mental status, anaphylaxis, cardiovascular disease, or tension pneumothorax). A duffel bag makes a convenient holder for medications, which can be organized into separate zipper-locked, heavy-duty plastic bags by category (e.g., antibiotics, analgesics/local anesthetics) and systems (e.g., respiratory/allergy, cardiovascular, gastrointestinal, ear-nose-throat, genitourinary/gynecology, dermatologic).

Additional equipment can occupy the space beneath the medicine bag. Miscellaneous items such as skin adhesive glues (or their hardware store counterparts), small reagent diagnostics, large bulb suction, sphygmomanometer, stethoscope, thermometers, urinary catheters, gloves, glasses, hefty supplies of large bandages, and other basics of minor-injury care, can be stored under the medicine duffel. Other useful items, such as repair supplies (Table 81-4), women's hygiene products, sunscreen sticks, and sunglasses may be considered.

Many items have multiple uses and can be adapted for improvisational use (see Chapter 21). For example, a large-bore IV catheter and the finger of a latex glove can be used as a Heimlich valve, and the spike from high-flow IV tubing can be used as a makeshift cricothyrotomy device with a good fit to a bag-valve mask. The uses of duct tape and acrylate glues are too numerous to count and include blister control and femoral traction splints. In addition to standard pill bottles, small zipper-locked plastic bags can provide convenient storage for medications. Tablets can be placed in the small bags; instructions placed in or affixed to the bags should clarify dosing schedules.

How Much to Bring?

The duration of the outing or expedition and the maximum interval to medical care are the prime factors when considering what and how much to bring into the wilderness. A simplistic, but effective approach to answering the question of how much to bring is to calculate the dosing schedule of each medication under consideration, then multiply it by the maximum interval to medical care and the number of people who are likely to require the specified therapy at any one time. For example, on a 1-week rafting trip 3 days away from the nearest pharmacy with 10 people, determine how much ciprofloxacin or azithromycin should be available for the treatment of traveler's diarrhea if it takes more than 1 day to identify each case (see Table 81-3 for indications for antibiotic treatment of some commonly encountered infections).

Sample calculations for commonly prescribed antibiotics for traveler's diarrhea are as follows:

Ciprofloxacin 500 mg: 1 pill twice a day × 3 days for 10 people
$= 1 \times 2 \times 3 \times 10 = 60$ pills

Azithromycin 500 mg: 1 pill each day × 3 days for 10 people
$= 1 \times 3 \times 10 = 30$ pills

The rationale for medicine selection among a host of prescribed and over-the-counter items is based on one's expected time frame for support and rescue in the event of unexpected injury and the cumulative likelihood of experiencing a medical illness

as trip duration increases. A low-risk outing (e.g., a day hike) is one in which the interval to medical care is short (4 hours) and the trip duration (1 day) places one at low likelihood of acquiring a medical illness. Therefore, expectations for a prompt rescue and medical support preclude the need to carry most prescription medicines. Because traumatic injury or a life-threatening allergic reaction are always of prime concern, even in a low-risk outing, pain control and management of anaphylaxis are most important. In such circumstances, pain control and management of anaphylaxis are most important. A moderate-risk trek (e.g., a 1-day "ski-in" with 2 nights winter camping) will have an interval to medical care that is longer (4 to 12 hours) and a trip duration that increases chances for medical illness. With a "ski-out" time of 4 to 12 hours plus added time to return with organized help, expectations are that an ill group member will not receive sophisticated rescue efforts until the following day. In such instances, an opioid to control pain, antibiotic to prevent infection from soft tissue injuries, and antiemetic or antidiarrheal medications to control gastrointestinal upset and preserve fluid and electrolyte balances become paramount. A high-risk outing (e.g., a weeklong expedition with mountain climbing) precludes seeking formal help within a 12-hour period and has a long trip duration, with the added challenge of encountering bothersome medical illnesses. With such an expedition, nonthreatening problems (e.g., athlete's foot, infected blisters, dermatitis, asthma, bronchitis, or sinusitis) are apt to occur, limiting performance and enjoyment. Besides carrying the medicines noted previously for low- and moderate-risk treks, one should have treatments to manage more persistent medical illness, including a full course of oral antibiotics for upper respiratory, abdominal, and soft tissue infections, oral and potent topical steroids for dermatologic problems, and assorted ophthalmic and otic medications to deal with bothersome infections or allergic reactions of vital sensory organs.

A Sample Journey

The month is March, and the goal is a 4-day climb to the peak of Mount Shasta (elevation 4317 m [14,162 feet]) in the Cascade mountain range. There are 17 established routes. However, in winter, these are limited, and the team has decided to climb Avalanche Gulch, a popular route. The team consists of 10 cross-country skiers who have completed an American Red Cross advanced first-aid course and an avalanche seminar offered to the public by the National Ski Patrol System. The base camp will be at 2134 m (7000 feet). Were a skier to become disabled en route, one of the others would remain with the victim while the remaining two would ski out to the nearest telephone or source of help (no longer than an 8-hour downhill ski from the farthest point along the route, in good weather). Helicopter evacuation to the nearest hospital would usually be accomplished within the next 2 to 4 hours, making the maximum interval to medical care 12 hours, weather permitting. The trip poses risks related to trauma, cold injury, avalanche danger, snowblindness, and high-altitude illness. With the possibility of an overnight stay until rescue arrives, medicine selection focuses on pain control for traumatic injury and acute mountain sickness. Appropriate topical medicines include sunscreen and those for managing corneal injury from snowblindness. Systemic medicines of value include a limited supply of antibiotics to prevent infection of soft tissue injuries plus specialized medicines for prevention and treatment of acute mountain sickness.

How much acetazolamide (Diamox) should be carried? If the acetazolamide supply is intended for both prophylaxis and treatment, there are 10 members, and the maximum anticipated time to rescue is 24 hours, then a generous calculation (assuming all members require treatment and that evacuation can be accomplished in 24 hours) can be estimated as follows:

Prophylaxis: 1 pill (125 mg) twice a day during 4 days of ascent for 10 people = 80 pills
Treatment: 2 pills (250 mg) twice a day for 1 day awaiting rescue for 10 people = 40 pills
Total: Prophylaxis + Treatment = 80 + 40 = 120 pills

Finally, emphasis must be placed on the recreational and environmental hazards likely to be encountered during travel. In this example, the dangers relate mainly to high altitude and cold exposure. Specialized equipment that may be of value under the conditions described is listed in Box 81-7. Because the peak altitude is only 4317 m (14,162 feet) and the nearby medical center has prompt helicopter evacuation abilities, a Gamow Bag would not be mandated for treating high-altitude illness. However, because of unpredictable weather and visibility, its addition to the medical kit could be considered, weight and bulk permitting. In addition, the need to carry technical equipment to provide warmed IV solutions or humidified oxygen is offset by a prompt helicopter rescue. However, frostbite can occur rapidly, and each individual might wish to carry a personal supply of chemical hand warmers (e.g., Grabber) because of their low weight and ease of packing. Additionally, certain repair equipment should be carried, as denoted in Table 81-4. Overall, thoughtful advanced consideration of proximity to care, duration, and probability of various illnesses and injuries will guide both selections and quantities in preparing for a wilderness outing.

SUMMARY

Physicians advising travelers must be prepared to identify possible problems and provide advice, equipment, and medications to allow their patients to both avoid and treat these problems. This challenge requires the medical designee to obtain information about the health of the travelers, the region of travel, and environmental and recreational exposures. A basic first-aid kit, essential medical knowledge, and a small number of medications will allow travelers to provide a first line of medical care for themselves and their group. Travelers who are not medical professionals should exercise caution when making decisions about how to care for themselves and other group members. In addition, they should seek advice from either local physicians or physicians from the country of origin whenever possible. Particular attention to hygiene, water quality, hydration, proper diet, sun protection, and foot care can prevent many of the hardships associated with travel.

An array of supplies to deal with potential problems can be appropriately tailored to augment the contents of a basic medical kit. Based on trip duration and remoteness from medical care, specific medications may be chosen and dispensed by knowledgeable and responsible individuals. Such efforts may permit a sick group member to continue travel in the setting of non–life-threatening medical illness or forestall serious discomfort or disability while one awaits search and rescue efforts.

SUGGESTED READINGS

Adventurous Traveler Bookstore, 245 South Champlain St., PO Box 64769, Burlington, VT 05406-4769; 800-282-3963. Website: www.adventuroustraveler.com. A complete source of outdoor travel books, maps, videos and CD-ROMs.

American Red Cross, American Red Cross, PO Box 37295, Washington, DC 20013 Website: www.redcross.org. Many tutorials on accepted guidelines for advanced first aid, best reviewed in a classroom format before travel.

Auerbach PS: Medicine for the Outdoors, 4th ed. Guilford, CT, Lyons Press, 2003. Website: www.lyonspress.com (800-836-0510). This is an excellent, compact wilderness medicine guide for laypersons. It covers all situations and environments and is meant to be carried by anyone who will treat an illness or injury outdoors.

Backer H: Medical limitations to wilderness travel. Emerg Med Clin North Am 15:17, 1997.

Baird JK: Drug therapy: Effectiveness of antimalarial drugs. N Engl J Med 352(15):1565–1577, 2005. Excellent review of malaria therapies and resources for finding appropriate recommendations for prophylaxis and treatment of this disease in the field from several perspectives, including adverse effects and cost.

David JH, et al (eds): Outdoor Emergency Care: Comprehensive First Aid for Non-urban Settings, 4th ed. Lakewood, CO, NSPS, 2002 (133 S. Van Gordon, Lakewood, CO 80228; 303-988-1111). Website: www.nsp.org. Developed for ski patrollers, provides an up-to-date, comprehensive guide to the prevention, recognition, and management of injuries and illness encountered in nonurban settings. Highly recommended reading for participants in all varieties of skiing.

Darvill F: Mountaineering Medicine, 14th ed. Berkeley, CA, Wilderness Press, 1998 (2440 Bancroft Way, Berkeley, CA 94704; 510-843-8080). Website: www.wildernesspress.com. Light-weight, easily carried reminder of the principles of first aid and basic medical care for the mountain environment.

Forgey W: Wilderness Medicine, Beyond First Aid, 5th ed. Guilford, CT, Globe Pequot Press, 2000.

Gentile DA, Morris JA, Schimelpfenig T, et al: Wilderness injuries and illnesses. Ann Emerg Med 21:853, 1992.

Hackett P, Roach R: High-altitude illness. N Engl J Med 345:107–113, 2001. An excellent review of cerebral and pulmonary syndromes and their appropriate medical therapies.

Lentz M, et al: Mountaineering First Aid, 4th ed. Seattle, The Mountaineers, 1996 (306 Second Ave West, Seattle, WA 98119; 800-553-4453). Website: www.mountaineers.org. Good review of mountaineering first aid, most appropriate for classroom or individual reading in advance of departure.

McFadyen JE, et al: International Drug Price Indicator Guide, 2005. Published on the Internet for free by Management Sciences for Health. This guide lists medications deemed essential by the World Health Organization and gives an idea of wholesale purchase costs for myriad drugs. Website: www.erc.msh.org. This website is run by a consortium of health management experts from around the globe and is also a superb guide for understanding local medical economies. Published on the Internet in three languages: French, English, and Spanish.

Montalvo R, Wingard DL, Bracker M, Davidson TM: Morbidity and mortality in the wilderness. West J Med 168:248–254, 1998.

Olsen LD: Outdoor Survival Skills, 6th ed. La Vergne, TN, Ingram Trade, 1998. There are good chapters on shelter, fire, water, plants, and special skills, as well as on building a lean-to and how to harvest and prepare plants in the wild.

Vonhof J: Fixing Your Feet, 3rd ed. Berkeley, CA, Wilderness Press, 2003 (1200 5th Street, Berkeley, CA 94710; 510-558-1666, 800-443-727, fax 510-558-1696. E-mail: mail@wildernesspress.com. A superb foot care book applicable to foot care in all environments.

Wilderness Medicine Letter (3595 E. Fountain Blvd. Ste A1, Colorado Springs, CO 80910; 719-572-9255). Website: www.wms.org. This is the newsletter of the Wilderness Medical Society. They review recent publications, notification of events and meetings, official position statements, and commentary by experts in the field. Highly recommended reading for physicians serving as advisors to travelers.

Wilkerson J, et al (eds): Medicine for Mountaineering and Other Wilderness Activities, 5th ed. Seattle, The Mountaineers, 2001 (306 Second Ave. West, Seattle, WA 98119; 800-553-4453). Website: www.mountaineers.org. This is a comprehensive coverage of the prevention, recognition, and advanced field management of mountaineering injuries and illnesses. Many sections are easily understood by general readers, but chapters on medical illness require familiarity with medical terminology and concepts, and a working knowledge of basic first aid is assumed. It is recommended that a single copy be carried by the person responsible for medical care of a large group on an extended or high-risk mountaineering outing.

Wilkerson J, Bangs C, Hayward J: Hypothermia, Frostbite and Other Cold Injuries, 5th ed. Seattle, The Mountaineers, 1998 (306 Second Ave. West, Seattle, WA 98119; 800-553-4453). Website: www.mountaineers.org. Excellent overview that explains, in lay terms, the metabolic disturbances underlying cold injury, as well as practical methods of preventing and treating these disorders. Especially welcome is the chapter on immersion injuries containing valuable information for persons involved in aquatic sports. Highly recommended reading for all travelers to cold weather, mountain, and aquatic wilderness environments.

Williamson JE (ed): Accidents in North American Mountaineering. Golden, CO, The American Alpine Club, 2004.

Zell SC: Common questions: environmental and recreational hazards associated with adventure travel. J Travel Med 4:94, 1997.

APPENDIX A: HEALTH QUESTIONNAIRE FOR WILDERNESS TRAVEL

TRIP COORDINATOR: _____ Dates of trip: _____
Trip location: _____ Major type of activity: _____
_____

Please provide the following information (use another sheet of paper if necessary): (All information will be used confidentially by the coordinator to promote the safety of the individual and the group)

Name _____ Age _____ Height _____ Weight _____ Last Menstrual Period _____
_____

Address _____

Emergency Contact Information (names, phone numbers, addresses): _____
_____

Primary Care Doctor contact information (names, phone numbers, addresses): _____
_____

Are you trained in basic first aid (Yes) (No) or CPR (Yes) (No)?
Date of most recent certification, if available _____.
What is your overall HEALTH? (circle one)
Poor Fair Good Excellent
What is your overall PHYSICAL condition? (circle one)
Poor Fair Good Excellent

Medical Data

1. Allergies (drug, pollen, food, etc.—be specific) . . . . . (YES) (NO)
2. Allergies (insect bites—be specific) . . . . . (YES) (NO)

3. Diabetes . . . . . (YES) (NO)
4. Epilepsy, seizures, or convulsions . . . . . (YES) (NO)
5. Heart or lung problems . . . . . (YES) (NO)
6. Kidney or genitourinary system problems . . . . . (YES) (NO)
7. Injuries or problems with joints (specify which side, EXPLAIN problem in space provided below or on a separate sheet of paper):
   A. Ankle . . . . . (YES) (NO)
   B. Knee . . . . . (YES) (NO)
   C. Hip . . . . . (YES) (NO)
   D. Fingers/toes . . . . . (YES) (NO)
   E. Wrist . . . . . (YES) (NO)
   F. Elbow . . . . . (YES) (NO)
   G. Shoulder . . . . . (YES) (NO)
   H. Back/spine, including neck . . . . . (YES) (NO)
   I. Other . . . . . (YES) (NO)
8. Have you ever had (EXPLAIN problem):
   A. Mountain sickness . . . . . (YES) (NO)
   B. Cerebral edema . . . . . (YES) (NO)
   C. Pulmonary edema . . . . . (YES) (NO)
   D. Heatstroke . . . . . (YES) (NO)
   E. Sunblindness/snowblindness . . . . . (YES) (NO)
   F. Frostbite . . . . . (YES) (NO)
   G. Hypothermia/exposure . . . . . (YES) (NO)
   H. Immersion . . . . . (YES) (NO)
   I. Excessive nosebleeds . . . . . (YES) (NO)
   J. Asthma . . . . . (YES) (NO)
   K. Ulcers . . . . . (YES) (NO)
   L. Bowel problems . . . . . (YES) (NO)
   M. Broken bones . . . . . (YES) (NO)
   N. Do you have any metal clips, plates, screws or implants? . . . . . (YES) (NO)
9. Have you been or are you being treated for any psychiatric conditions such as depression, bipolar disease, post-traumatic stress disorder (PTSD)? Do you have any phobias that may affect your ability to participate in the group's activities? Be as specific as possible.

   _____
   _____

10. List any medical problems, illnesses, injuries, surgeries (including cesarean birth) or chronic conditions that you have presently or have had in the past 3 years (be as specific as possible):

    _____
    _____
    _____

11. List any MEDICATIONS that you are currently taking (include the dosing, if possible):

    _____
    _____
    _____

Modified from Green P: The Outdoor Leadership Handbook. Tacoma, Wash, Emergency Response Institute, 1982.

APPENDIX B: SUPPLIERS LISTED IN TEXT AND TABLES

Many of the products cited in this chapter can be found in outdoor equipment retail stores or pharmacies. The sources listed below are for specialized products referred to in the text.

1. Black Diamond Equipment, Ltd., 2084 East 3900 South, Salt Lake City, UT 84124; 801-278-5552. Website: www.bdel.com. Manufacturer of the AvaLung, which is the first and only active avalanche safety device that may enable the avalanche victim to breathe while waiting to be excavated. Website: www.avalung.com.
2. Campmor, PO Box 700, Saddle River, NJ 07458; 888-CAMPMOR (888-226-7667). Website: www.campmor.com. Lady-J urinal guide and other camping equipment.
3. Chinook Medical Gear, Inc., 3455 Main Avenue, Durango, CO 81301; 800-766-1365. Website: www.chinookmed.com. One of the most complete catalog retailers for wilderness medical supplies; carrier of the Gamow Bag; good beginning source for the person in need of a wide variety of medical supplies.
4. Conmed Corporation, 525 French Road, Utica, NY 13502; 315-797-8375. Website: www.conmed.com. Makers of skin staplers and other surgical instruments.
5. Garmin International Inc., 1200 East 151st Street, Olathe, KS 66062; 913-397-8200. Website: www.garmin.com. Maker of a wide variety of easy-to-use GPS devices and instructional videos for GPS education.
6. Gilbert Surgical Instruments, Inc., PO Box 458, Bellmawr, NJ 08031; 856-933-2770. Website: www.gilbertsurgical.com. Manufacturer of the Abelson emergency cricothyrotomy cannula and other respiratory care instruments.
7. Grabber Performance Group, 4600 Danvers Drive SE, Grand Rapids, MI 49512; 800-518-0938. Website: www.grabberwarmers.com. Manufacturer of a variety of packaged miniature heaters with accessories useful for preventing cold-related injuries of the hands, feet, and face.
8. Junkin Safety Appliance Co., 3121 Millers Lane, Louisville, KY 40216; 888-458-6546. Website: www.junkinsafety.com. Manufacturers of several folding aluminum full-length backboards, splints, and stretchers as light as 15 lb. Other safety equipment includes fire blankets and cervical spine immobilizers.
9. MARSARS/Great Eastern Marine, Inc., 205 Myrtle Street, Shelton, CT 06484-4015; 203-924-7315 or 866-426-2423. Website: www.marsars.com. Manufacturer of water and ice rescue equipment; a hypothermic stabilizer bag and additional accessory heat packs are available.
10. Mountain Safety Research, 4000 1st Avenue South, Seattle, WA 98134; 800-531-9531. Website: www.msrcorp.com. Manufacturer of high-performance and light-weight stoves, as well as the WaterWorks filtration system.
11. National Ski Patrol System, Inc. (NSP), 133 South Van Gordon, Suite 100, Lakewood, CO 80228; 303-988-1111. Website: www.nsp.org. Various first-aid supplies and equipment for winter skiing and mountaineering sold by catalog to NSP members.
12. Otter Products, LLC Bldg 1, Old-Town Square, Suite 303, Fort Collins, CO 80524; 888-695-8820. Website: www.otterbox.com, also available at

www.watertightcase.com. Otter manufactures a variety of water-tight cases useful for everything from PDAs to large medical kits.
13. Outdoor Research, 1000 1st Avenue South, Seattle, WA 98134; 800-421-2421. Website: www.orgear.com. Diverse array of tote bags, medical travel kits, and stuff sacks useful for international travel.
14. Patagonia, Inc., PO Box 150, Ventura, CA 93002; 805-643-8616. Website: www.patagonia.com. Capeline underclothing; variety of outdoor clothing.
15. Pelican Products, Inc. 23215 Early Avenue, Torrance, CA 90505; 310-326-4700 within California, 800-473-5422 outside California, fax, 310-326-3311. Website: www.pelican.com; e-mail: sales@pelican.com. Manufacturer of water-tight freight boxes, bright and durable headlamps.
16. Recreational Equipment, Inc. (REI), 800-426-4840. Website: www.rei.com. Thermax underclothing; variety of sunglasses, outdoor gear and clothing.
17. Res-Q Products, Inc., PO Box 661, Quathiaski Cove, BC, Canada VOP 1N0. Contact: Robert Douwens. Websites: www.hypothermia.org (educational site) or www.hypothermia-ca.com. Res-Q-Air and IV HOT-SACK plus accessories; sophisticated equipment for the serious expedition traveler to areas of extreme cold.
18. SAM Splints, 4909 South Coast Highway #245, Newport, OR 97365; 541-867-4726. Website: www.samsplint.com. SAM folding splint, 4-oz and many other splinting and first-aid products made for outdoor travel and first aid.
19. Slishman Splint. Website: www.geocities.com/slishmansplint/. Available from Bound Tree Medical (UK). Not currently available for sale in the United States.
20. Survival Suppliers, PO Box 1860, Los Gatos, CA 95031 Website: www.survivalsuppliers.com. A multitude of well-designed medical kits that is prepackaged for a range of specific recreational activities, such as bicycling, white-water rafting, and backcountry travel.
21. Travel Medicine Inc., 351 Pleasant Street, Suite 312, Northampton, MA 01060; 800-872-8633. Website: www.travmed.com. Specialize in educational books and handy supplies for international travel; source of insect repellents, clothing, and nets for mosquito protection.
22. Zarges Aluminum Boxes, Soanar Inc., Mayer Krieg Components, 9 Civic Square, Croydon, Victoria 3136 Australia. Phone: 61 2 9741 0192. Website: www.soanar.com. Extremely durable, light-weight, water-tight aluminum freight boxes ideal for storing medical and communications equipment.

APPENDIX C: SOURCES OF INFORMATION ABOUT WILDERNESS EMERGENCIES AND SUGGESTED READINGS

1. American River Touring Association, 24000 Casa Loma Road, Groveland, CA 95321; 800-323-2782. Website: www.arta.org. This is a nonprofit organization that sponsors basic and leadership courses for river rafting and kayaking.
2. Emergency Response Institute, 4537 Foxhall Drive NE, Olympia, WA 98516; 360-491-7785. Website: www.eri-intl.com. Publishes books and sponsors symposia on search and rescue, emergency preparedness, survival, and outdoor leadership.
3. National Association for Search and Rescue (NASAR), PO Box 232020, Centreville, VA 20120-2020; 703-222-6277. Website: www.nasar.org. Educational association that provides conferences, symposia, and training in search and rescue and emergency response, including communications; sponsors *Rescue* magazine (JEMS Communications, 1947 Camino-Vida Roble, Suite 200, Carlsbad, CA 92008; 619-431-9797).
5. National Climatic Data Center, 151 Patton Avenue #120, Asheville, NC 28801-5001; 828-271-4800. Website: www.ncdc.noaa.gov. Provides historical weather summaries for U.S. and many foreign cities; small fee for materials. Telephone first to determine availability of relevant data.
6. National Ski Patrol System, Inc. (NSP), 133 South Van Gordon, Suite 100, Lakewood, CO 80228; 303-988-1111. Website: www.nsp.org. Sponsors training programs in winter emergency care, avalanche, and ski mountaineering, some that are open to the public; sells equipment and first-aid supplies through catalog to NSP members.
7. National Weather Service, 1325 East West Highway, Silver Spring, MD 20910. Website: www.nws.noaa.gov. The NWS site nearest the travel site can provide forecasts and in many regions provides broadcasts over NOAA Weather Radio on frequencies between 162.40 and 162.55 MHz, including 3- to 5-day forecasts and avalanche warnings.
8. Outward Bound Training Institute, 800-779-7935. Website: www.outwardbound.com. Nonprofit educational organization that uses mountain, river, and ocean wilderness settings to provide stimuli for personal development; separate leadership training courses available.
10. The Travel Doctor, 7515 Greenville Avenue, #600 Dallas, TX 75231. Website: www.thetraveldoctor.com. An excellent website for travel alerts and learning about the pitfalls of travel to different international locales, also lists prescription medications useful for various travel-related illnesses
11. Undersea and Hyperbaric Medical Society, 10531 Metropolitan Avenue, Kensington, MD 20895; 301-942-2980. Website: www.uhms.org. Nonprofit organization that sponsors workshops and meetings on the prevention and treatment of diving injuries and illnesses treatable with hyperbaric oxygen. Publishes bimonthly newsletter, *Pressure,* and two research publications, *Undersea Biomedical Research* and *Journal of Hyperbaric Medicine*.
12. Wilderness Education Association, 900 East 7th Street, Bloomington IN, 47405; 812.855.4095. Website: www.weainfo.org. Nonprofit educational organization that specializes in training and certifying outdoor leaders.

13. Wilderness Medical Society, 810 East 10th, PO Box 1897, Lawrence, KS 66044; 800-627-0629. Website: www.wms.org. Nonprofit organization of medical and related professionals interested in the prevention and treatment of wilderness injuries and illnesses. Publishes quarterly newsletter, *Wilderness Medicine Letter,* covering wilderness medicine meetings, literature review, field management, and position statements. Publishes *Wilderness and Environmental Medicine* (formerly *Journal of Wilderness Medicine*), the official academic publication of the society.

The references for this chapter can be found on the accompanying DVD-ROM.

Outdoor Clothing for Wilderness Professionals*

82

Christopher Van Tilburg

Humans are tropical animals, not designed to be in the elements—sun, wind, rain, and snow—without protection. Outdoor clothing is extremely important for survival and comfort. Like any piece of equipment, outdoor clothing for work, recreation, rescue, and research should be chosen for specific activities, climates, and weather conditions. Design of materials, styles, and accessories are key to function and comfort, especially in extreme weather, rough terrain, or vigorous activity in which survival and safety may hinge on clothing. This chapter examines general principles of fabrics, clothing design, and dressing, with emphasis on choosing proper clothing for specific applications, activities, climates, and weather.

When one is choosing clothing, basic guidelines should be followed. Clothing type and amount depend on several factors: climate, season, weather, length of travel, degree of remoteness from civilization, specific activity, and level of exertion. For example, one who typically travels in harsh climates or foul weather needs warmer clothing than someone working in mild climates. Long trips and remote locations may require more clothing and a different selection than half-day outings close to civilization. Also, one who is participating in a highly aerobic activity such as hiking or skiing may want different clothing than someone working at a relatively sedentary job such as research or photography, especially in extremely hot or cold environments. Personal preference makes a difference; one person may prefer several layers of light clothing, while another may be more comfortable with bulky insulating layers.

One should carry enough layers to adapt to different work levels and climatologic extremes. Information in selecting gear that allows comfort during average highs and lows must also take into account the extremes. Always carry extra clothing for foul weather, unexpected delay in returning to shelter, and other contingencies. One should carry at least enough clothing to survive an unexpected night in the field.

*The present chapter is based on John Gookin's fine treatment of this topic in the previous edition.

FABRICS

The wide variety of synthetic and natural fibers used in outdoor clothing are quite variable in cost, comfort, durability, and function. A brief overview highlights common natural and synthetic fabrics used for a variety of applications. New fabrics improve the function of some garments, but time-tested designs and fabrics often work equally as well. Many fabrics are blends of several natural and synthetic fibers and are proprietary in construction and materials.

Outdoor clothing is made from fabrics woven of fibers. A fiber is a single strand of threadlike material. The following four material properties pertain to fibers and fabrics:

1. The thicker the material, the greater the insulative value, as long as the material stays dry. Thickness of an inch (2.5 cm) or more in any layer is rarely needed with modern materials.
2. The following qualities of fiber reaction to moisture are important:
   a. Ease of wicking action: Hydrophilic fabric transfers moisture from the body surface to the material; hydrophobic fabric transfers moisture from the body surface across the fabric to other clothing or to the air.
   b. Evaporative ability: rate of drying
   c. "Moisture regain": amount of moisture a material can absorb before it feels cold. With higher moisture regain, the fabric will be more functional for activity in cold or wet environments.
   d. Amount of insulative value a material retains when wet.
3. The less the thermal conductance, the better the insulation.
4. Tighter weaves and coated fabrics help block wind; this attribute decreases convective cooling but allows more perspiration to accumulate in insulation layers.

13. Wilderness Medical Society, 810 East 10th, PO Box 1897, Lawrence, KS 66044; 800-627-0629. Website: www.wms.org. Nonprofit organization of medical and related professionals interested in the prevention and treatment of wilderness injuries and illnesses. Publishes quarterly newsletter, *Wilderness Medicine Letter,* covering wilderness medicine meetings, literature review, field management, and position statements. Publishes *Wilderness and Environmental Medicine* (formerly *Journal of Wilderness Medicine*), the official academic publication of the society.

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   d. Amount of insulative value a material retains when wet.
3. The less the thermal conductance, the better the insulation.
4. Tighter weaves and coated fabrics help block wind; this attribute decreases convective cooling but allows more perspiration to accumulate in insulation layers.

Natural fibers tend to be soft on the skin and durable, but they are usually more expensive. Some people are allergic to wool, down, or silk; allergy to synthetic fibers is rare. Synthetics are usually light, durable, quick to dry, and warm. They are often not as durable, especially with repeated washings, but are less expensive. They are often flammable, tend to retain body odor after repeated use, and often do not feel as luxurious on the skin.

Natural Fibers

Cotton is durable, light, and comfortable. It breathes extremely well, absorbs sweat, and dries slowly. However, it has little insulating value, especially when wet. Thus, cotton excels only in summer desert climates when one wishes to protect skin from the sun's damaging rays or stay cool in extremely hot temperatures. Cotton is a poor choice for staying warm or protecting oneself from wind or rain.

Wool is a time-tested fabric that is a good insulator, is durable, and maintains some insulating properties when wet. It has a high coefficient of friction on snow and ice and thus does not slide like other fabrics. It is not flammable. However, wool does not dry quickly. Traditional wool fabrics tend to itch; thus, wool can be uncomfortable when worn next to skin. In the past, wool has been used for shirts, jackets, hats, and socks.

Merino wool is relatively new to the garment industry. Wool from Merino sheep from New Zealand is spun into longer and finer diameter strands than can be spun from other types of wool; the barbs are microscopic, too small to "prickle" human skin. Thus, when woven into a smooth finish, the fibers don't itch, have more elasticity, and shrink minimally. Merino wool is excellent for persons who prefer a natural fiber next to the skin, such as for long underwear or socks, and need the durability that wool provides. It is also machine washable.

Silk is warm, light, and soft. It is an excellent next-to-skin layer because it is very smooth and soft and exceptionally light. It isn't used often for outdoor clothing because it tends to be more expensive and less durable than other fabrics.

Down is a superior insulator, perhaps the lightest available. It is expensive, but long-lasting, and lighter than synthetic materials for the same warmth. It comes in different fill power or insulating value. Fill power is the amount of space, in cubic inches, that down occupies for a specific weight. In other words, with equal weight, the higher the fill power, the more space the down takes up. Hence, the more dead air space, the warmer the down. Every manufacturer measures down slightly differently, so comparisons can be difficult. In general, the highest quality down has a fill power of 800 in^3 (13 L; 0.013 m^3) or more. Lower quality is often 600 in^3 (9.8 L; 0.0098 m^3); it is less expensive, but to achieve the same warmth as 800 fill power, more down is used. Thus, the garments are heavier. Higher quality down feathers are usually procured from geese. Lower quality down often comes from ducks.

When down soaks up water, the feathers lose much of their insulating power and take a long time to dry. Down is best used in cold, high-altitude climates where there is little chance of rain, or as an insulating layer under a waterproof parka.

Synthetic Fabrics

A huge variety of synthetic materials are available. These synthetics are chiefly derived from petroleum-based polyester and nylon fibers, proprietary fibers, and blended fabrics. They are manufactured in many different thicknesses, weaves, and knits.

Polyester

Polyester is a general term for petroleum-based fiber. It is perhaps the most common next-to-skin layer and insulator. It is light, stretchy, warm, inexpensive, and durable. It wicks moisture away from the skin and dries quickly because it has minimal absorbency. It is not as soft on the skin as natural fibers, can retain body odor, and, like most synthetics, is flammable. For most applications, polyester is the standard for long underwear and has virtually replaced polypropylene, another synthetic with similar properties that does not feel as soft on the skin.

In addition to its use in thin next-to-skin layers, polyester is used for insulation in sweaters, vests, shirts, jackets, pants, booties, hats, and gloves. Polyester can be spun into thick fleece, also called *pile,* which is soft, washable, durable, and warm. Some examples of fleece include:

- Polartec Classic by Malden Mills is one of the first polyester fleece fabrics. Its bulk enables much air to be trapped; for its weight, it is exceptionally warm. The fleece is available in a variety of denier, or fabric thicknesses.
- Polartec Thermal Pro, also by Malden Mills, is more advanced. It is a softer and higher loft velour polyester that is lighter and warmer than Polartec Classic.
- Other types of fleece have specific properties, as listed in Box 82-1. Wind resistance, high loft for warmth, and stretch for aerobic activities are common desirable functions of fleece.

Polyester can be spun into sheets of insulation. Sheets of insulation are not as durable as fleece, so the sheets are usually encased in nylon for use in jackets, sweaters, and vests. Newer versions tend to be lighter and more compressible than older versions for the same warmth rating. This is because the fibers are spun into smaller sheets and are hollow. Unlike down, synthetic-filled coats, jackets, shirts, and vests dry quickly and stay moderately warm when wet. The main drawback is that they are neither as light nor as compressible as down of the equivalent warmth rating. Also, because synthetic fibers don't settle against the body as well as down, these garments tend to be less comfortable and a bit drafty. Some brands of sheets of high-loft polyester insulation include those listed. Each fabric comes in several thicknesses and weaves.

- Thinsulate, by 3M, is a polyester-based insulation fill material that was introduced in 1978 as a down substitute. It is warm and durable, traps air, and reflects body heat back to the wearer.
- Polarguard, by Invista, is a polyester fabric that has long continuous filaments and thus resists matting and clumping.
- Hollofil, Quallofil, and Thermolite are types of polyester-based fleece from Du Pont. Hollofil is softer and conforming. Quallofil is a high loft insulation used in parkas as a down alternative. Thermolite is thin for aerobic activities.
- Primaloft, by Albany International, is a proprietary polyester constructed as a down alternative. It carries a high loft-to-weight ratio and thus is warmer for its equivalent weight than other fabrics. It is softer and more compressible than other types of polyester or blended insulating fill fabrics, and also has improved water repellency.

In addition to polyester insulation materials, many companies manufacture blends such as polyester-polyolefin or proprietary fabrics. These fabrics take the benefits of polyester and

Box 82-1. Design Properties for Clothing

Moisture wicking
Lightweight
Low volume
Compressible
Easy to clean and repair
Elasticity
Durability
Abrasion resistance
Low moisture absorption
Comfort
Weather protection
Insulation
Flame resistant
Fashion
Cost
Insect protection
Sun protection
Stain resistance
Pockets for tools, hand warming
Accessories for venting excess sweat, heat
Accessories to improve weather protection

add additional fibers to improve one or more of the design characteristics, as listed in Box 82-1. An example is Thinsulate Lite Loft, by 3M, a polyester-polypropylene (see description earlier) blend that offers much more warmth for equivalent weight than Thinsulate; it has a higher loft-to-weight ratio. It is also softer.

Nylon

For outwear, nylon has long been the standard for wind- and water-resistant jackets and pants. Nylon is a generic term for petroleum-based polyamide fabrics made in numerous weaves and thicknesses. Nylon is light, strong, resistant to abrasion, easy to wash, and has low absorbency. Nylon is used for everything from windbreakers to heavy patches for abrasion resistance that reinforce elbows and knees of an alpine climbing suit or wear points in gaiters or overboots.

Some examples of the wide variety of nylon applications are listed here:

- Ripstop, an extremely lightweight nylon woven in square grids that is resistant to tears. It is used in windbreakers, wind shirts, and hiking pants.
- Taffeta, a light, soft fiber that is used for lining of parkas and pants, which are made from heavier, bulkier fill material.
- Supplex, trade name for Du Pont's fine-strand nylon that is soft and pliable; used for sun and watersport clothing.
- Cordura, trade name for Du Pont's thick, abrasion-resistant nylon used in backpacks or clothing in areas of heavy wear, such as knee patches on pants.

Laminates

For severe weather in highly aerobic activities, laminates provide an extra measure of wind- and waterproofness. Laminates are usually bonded to nylon because they are not as durable as other fabrics. "Three ply" has the laminate with nylon on both sides. "Two ply" has nylon on the outside to save weight and bulk for ultralight clothing. These fabrics work moderately well to protect from rain, wind, and snow. However, perspiration can condense on the inside of the jacket without evaporation, rendering the wearer wet even without ambient precipitation.

Laminates usually need to be seam-sealed or taped by the manufacturer because the stitching causes a weak point in the fabric that can disrupt the waterproof membrane.

Some examples are described here:

- Polyurethane is a petroleum-based coating that is applied to nylon or similar materials to increase impermeability. It is inexpensive and durable and often used in jackets, pants, and tent rain flies.
- Gore-Tex by Gore Industries is a fabric in which polytetrafluorethylene (known commonly by Du Pont's trade name Teflon) is laminated to nylon to improve wind- and water-resistance. Gore-Tex is made by stretching sheets of polytetrafluorethylene, which causes microscopic tears or holes. The holes are large enough to let perspiration in the form of water vapor escape, but they are small enough to prevent water droplets from rain or snow to leak through from the outside. This concept creates a breathable, waterproof fabric; sweat escapes and rain or snow stays out.

Other Materials

Many other synthetic materials are used in wilderness clothing. Those commonly used in blended fabrics are discussed under "Blends." Manufacturers use the design properties in Box 82-1 as a guideline for choosing fabrics. Like most synthetics, these are soft, washable, light, and durable. They have low absorbency and thus dry quickly. In addition, they are usually stain resistant.

- Acrylic is the generic term for polyacrylonitrile fiber woven into soft fabric that is often used in hats and sweaters as a wool substitute. It is inexpensive and easy to dye.
- Spandex is the generic name for thin polyurethane fiber that is extremely elastic. It is most commonly manufactured as Lycra, a Du Pont trade name. It is commonly used in high-stretch fabrics, such as tights and tops for aerobic activity in warm weather.
- Polypropylene is an early petroleum-based fiber, also called olefin or polyolefin. It was frequently used in next-to-skin layers, but has largely been replaced by polyester. Polypropylene is the least absorbent of all synthetics and it is the only one that floats.

Blends

Outerwear blends are now frequently used in place of single-material garments; blends may include both synthetic and natural fibers. These allow clothing designers to combine the benefits of more than one fabric to improve durability, comfort, and breathability.

There are a multitude of blends from many manufacturers. They prioritize the properties in Box 82-1, then enhance fabrics to maximize function. Some examples of blended fabrics follow:

- Wool is blended with polyester to provide durability and stretch.
- "Soft shell" jackets and pants, which will be discussed below, blend many types of fibers to create a breathable, wind- and water-resistant fabric.
- Polyester fleece can be blended with spandex to provide stretch, or with a laminate to improve wind- and waterproof functions.

CLOTHING DESIGN AND PROPERTIES

Clothing is designed according to the intended use. Important properties that manufacturers consider when designing outdoor clothing are listed in Box 82-1. Manufacturers weight the properties differently; some give highest priority to style, color, and accessories; some focus on durability and performance in extreme conditions; and others give weight and mobility highest priority.

Fibers are measured in *denier*. One denier is 1 gm per 9,000 m of a particular fiber. Light, thin fibers may be 30 denier; heavy and thick fibers can be 1000 denier or more. Whereas denier refers to a fiber, *loft* describes the thickness of a particular fabric.

Clothing warmth can be compared with clo units. One clo is the amount of clothing needed for a person to be warm at a temperature of 70° F (21° C). The clo value can also be described as square meter of clothing-degree Celsius per watt, that is, square-meter-kelvins per watt (m^2K/W), usually estimated by the manufacturer using guidelines from the American Society of Heating, Refrigeration, and Air-Conditioning Engineers. Corrections are made for surface area, fit, weave, and other design properties listed in Box 82-1. These guidelines are used because the clo unit is analogous to the R-value, the insulating property of home construction. One clo equals 0.155 m^2K/W. This is roughly equivalent to the insulation provided by $^1/_4$-inch thickness of wool. One R-value is 1.137 clo units.

The lowest clo value for clothing is 0; the highest is around 4 to 6, as listed below. In general, 10 lb of clothing are worth around 1.5 clo. Unfortunately, the clo value of a particular piece of clothing is estimated by the manufacturer. In addition, an article of clothing that insulates one person adequately in certain weather conditions may not insulate another as well because of different metabolism, perspiration, body shape and size, nutritional state, and hydration status. Clo values need to be interpreted with caution when comparing manufacturers or users. Clo units are generally useful to the outdoor clothing and fabric industries, but are not usually used in marketing and labeling information available to consumers. Clo values are most useful to someone scrutinizing clothing in detail. Examples of approximate clo values are:

- Cotton T-shirt: 0.06 to 0.12
- Summer clothing: 0.6
- Insulated work coveralls: 1.03
- Down jacket: 0.55
- Winter clothing: 1
- Fur Eskimo clothing: 4
- Military cold weather survival suit: 6

DRESSING IN LAYERS

It has long been accepted that dressing in layers is the key to outdoor clothing use. The exception is soft shell or multilayer clothing, as discussed below, or special clothing for extreme cold temperatures. Using the layer system allows one to easily add or subtract clothing depending on the weather and activity level, to easily replace soiled or wet layers, and to more easily pack for wilderness ventures. Using layers, one can usually carry less clothing while accommodating a broad range of temperature and weather conditions and activities.

One usually dresses in three types of layers: base, insulating, and outer. Layers need to fit comfortably on top of one another without binding. The outer layer protects from the environment. In cooler weather, inner layers of appropriate thicknesses are chosen so that they can be continually adjusted. For instance, at –29° C (–20° F) alongside a fjord (a wet, cold environment), one should wear expedition-weight undergarments, two or three insulation layers, and a snow-proof parka and pants.

Anticipate changes in body temperature and climate and adjust layers accordingly. As one begins climbing a steep hill, it is wise to remove layers and ventilate zippers before overheating. To avoid sweating, one should expose more skin around the head, neck, and wrists; remove gloves; walk in the shade instead of the sun; and turn the head slightly to face into the breeze.

Prevention of sweating in cold climes is more important than just avoiding discomfort. It keeps insulation drier, fluffier, and warmer. Garments remain cleaner for extended use. Experienced expedition members know that this strategy is not just for peak performance during work cycles; it helps achieve a lower metabolic rate and therefore conserves energy and water.

One should add clothing layers when heading over a windy pass or downhill, always adding insulation to trap heat before resting in cold weather. If one begins to feel cold and begins shivering, one or more layers are added.

Base Layer

The base or next-to-skin layer should create warmth while allowing for quick transfer of sweat to outer layers and eventually to the air. It should be tight yet stretchy to allow for adequate motion without restrictions. Sometimes it is useful to have a very thin layer, especially in mild climates or with extended aerobic activity. Other times, the base layer should be thick to retain as much heat as possible. The base layer is designed to be hydrophobic and repel water. The primary function is to retain heat while transferring moisture from the body surface to the outer layers of clothing and eventually to the air. This function of clothing base layers is called wicking.

Several types of materials are adequate for a base layer. In most cases, a hydrophilic material such as polyester, silk, or Merino wool works well. Some people like the durability of wool or the natural soft feel of silk. Others prefer inexpensive and durable polyester. Zippered turtle necks are versatile and allow ventilation.

In a few circumstances, hydrophilic base layers such as cotton, which retain sweat and water, are useful. In desert travel, for example, cotton can keep one cool and protect one from the sun's rays. For cold weather, cotton is a poor insulator, especially when wet.

Middle Layer

The middle layer serves as insulation and is widely variable. This may be a shirt, vest, sweater, or two layers of bulky fleece. Basically, the thicker the fabric and the more dead air space trapped, the warmer the layer. Insulation layers should be tight enough to minimize excess bulk or air pockets, but should be loose enough and stretch to allow free movement. With two layers of insulation, one should easily fit over the other.

Most insulating layers consist of polyester fleece, synthetic fiber filler, down, or wool. For tops, the most versatile combination is a vest and zippered sweater or a long-sleeve shirt in

which sleeves can be rolled up. A vest keeps the core warm while giving full motion of arms without restriction. A zip-up sweater is easy to put on and take off. Pullover sweaters are sometimes lighter. For cold climates, two layers may be necessary.

For pants, full zippers are useful, and fleece or a synthetic blend is the most durable. This allows pants to be easily donned or removed without removing boots. However, this convenience adds weight and may not be necessary for some wilderness travelers. Pockets that zip are extremely useful for storing small items. These often include handwarmer, rear, and side thigh pockets.

Outer Layer

The outer layer is for wind and water resistance. This may be a heavy parka, light windbreaker, rain suit, or down suit. The tighter the weave, the more wind and waterproof the fabric. However, tighter weave lessens the chance of ventilating excess sweat and heat generated from activity. This conundrum has been solved in four ways:

1. Outerwear has been designed to ventilate excess sweat and heat through armpit zippers, vents, two-way front zippers, and other accessories.
2. Laminates such as Gore-Tex allow smaller droplets of sweat to escape, but repel larger droplets of external moisture. They are breathable and resistant to rain, snow, and wind. In heavy rain and snow or in situations of profuse perspiration, laminates don't work optimally.
3. Looser weaves block wind and rain to some extent, and allow rapid heat and moisture transfer from the body to the air. These weaves promote quick drying and ample ventilation. This is the principle behind the "soft-shell clothing" discussed later. This outer layer is most useful in highly aerobic activities to ventilate excess sweat and heat, particularly in mild climates when large storms are not anticipated.
4. For activities in which there is little aerobic activity and thus little internal heat and sweat accumulation, rubberized or polyurethane-coated nylon may work better. This is more waterproof, but tends to be hot and not breathable in moderate or mild temperatures. It works best for sedentary activity in rainy weather.

Parkas should have large handwarmer pockets as well as chest pockets that can easily be accessed while wearing a backpack. It is helpful to have a hood for foul weather; it should easily fit over a helmet if one is used. Arm pit zippers help ventilate. Storm flaps prevent drafts from coming through the zippers. Shock cord drawstrings at the waist and at the cuffs help seal out the weather.

Pants should have full zip sides so they can be put on or taken off without removing boots in foul weather. It is helpful to have at least one zippered pocket for storing valuables or small items that need to be accessed frequently. Bibs are sometimes preferred because they cover the chest and are warmer, keep out weather at the midriff, and stay up during aerobic activities or heavy labor.

ACCESSORIES

Accessories protect appendages and the head, and are critical to prevent frostbite, hypothermia, and heat illness.

Handwear

Gloves vary depending on the application. One may use leather gloves for search and rescue, rubberized gloves for fishing, or warm polyester and nylon wind- and waterproof gloves for winter activity. Hybrid gloves use leather on palms and fingers for grip and nylon on the dorsum for warmth and wind- and waterproofing. Some gloves are insulated to provide extra warmth. An inner layer of polyester liner gloves adds extra warmth but can decrease grip strength.

Mittens are generally warmer than gloves because they keep all fingers together and have less surface area exposed to the cold. Gloves allow better dexterity and grip. For cold environments, mittens or gloves with long gauntlets seal out the weather. Retention cords are essential to prevent loss in high winds or on slopes.

Headgear

Cold weather hats are most often polyester fleece, acrylic, or wool. For persons who perspire frequently or profusely, a washable hat works best. In general, hats should provide adequate coverage over ears as well as the head. For persons needing sun protection, a wide-brim canvas hat provides excellent shade. A wide-brim rubber- or urethane-coated nylon or rubber hat keeps rain off the head and from running down the neck. If a helmet is to be worn, the hat must be thin enough to fit under the helmet and be free from tassels.

A balaclava or neck gaiter is important to cover exposed skin of the face. A balaclava, sometimes called a ski mask, is a hat that is pulled over the head and down the neck, with a cutout for the eyes and nose. A neck gaiter is a tube-shaped scarf that can be pulled over the head to cover the face from just below the eyes down to the neck, including the lower part of the ears, which are often exposed even when wearing a hat.

Eyewear

See Chapters 21 and 25.

Socks and Overboots

Synthetic or wool blends are good for warmth, durability, and fit. Socks should be stretchable to eliminate any bunching that causes blisters. Polyester fleece socks are warm, but tend to wear out sooner. Neoprene socks are the warmest for prolonged walking in water.

With proper boot choice and fit, wearing one pair of socks may suffice. However, wearing two pairs of socks, a thin liner plus a bulky insulating layer, adds warmth, comfort, and protection from friction blisters.

Gaiters and overboots provide extra warmth and weather protection in mountain environments. They also seal out sand and dirt in desert and forest environments. Gaiters are tube-shaped cuffs made from nylon or nylon with a laminate that attach to boots and legs with straps, and fit from the mid foreleg to the ankles. They seal out the weather from the boot top to the lower pant leg. Some specialized skiing and mountaineering pants have built-in gaiters with tight elastic cuffs. For extreme winter and mountain environments, gaiters are insulated with polyester sheets and encased in nylon; these are commonly called *overboots*. For mountain biking, road cycling, and water sports, overboots are made from neoprene.

MULTILAYERED AND SOFT SHELL GARMENTS

Newer clothing designs attempt to combine two or even three layers into one piece. This may be more efficient for certain

applications, but if a person tends to add and remove layers frequently during an outing, these designs may not be the best choice. A few examples of multilayer designs are described here.

Windproof fleece bonds bulky insulating fleece to a laminate as described earlier in this chapter. This is designed to function as both an insulating layer and a windproof layer in mild climates. These garments are great for sedentary activity in cool or occasionally windy and drafty conditions. This type of fleece tends to be more bulky than regular polyester pile. Brands include Gore Industries' Windstopper, which laminates polyester fleece to Gore-Tex, and Malden Mills' Polartec Windbloc, which laminates polyurethane film to polyester fleece.

Insulated parkas combine a wind- and waterproof outer shell to a bulky layer, usually polyester or down. These are best for cold climates in which a person doesn't add or remove insulating layers frequently or doesn't require a wind or rain shell separate from the insulating layer. Typical users are Arctic researchers or high-altitude climbers. The warmest type of suit is a down or polyester fill jacket.

Soft-shell clothing is stretchy outerwear that has some insulating value and moderate wind and water protection. It is highly breathable and dries quickly. Although it provides significantly less wind and water resistance and insulation when compared to traditional parkas, a soft shell allows ventilation and is usually light, stretchy, comfortable, and durable. It also allows the user to wear one layer that functions both as a middle insulating layer and an outer shell layer. In mild weather with aerobic activity, where one is less concerned with wind and rain but more apt to become wet and cold from excess sweat, these garments work well.

FABRIC TREATMENT

Some fabrics are treated chemically to enhance their function. For example, polyester can be treated with antimicrobial chemicals that resist bacterial and fungal growth. This is useful for expeditions in which regular washings may not be practical. Some chemicals can enhance sun and insect protection.

Fabric can be treated with durable water repellent (DWR). DWR is an industry term that refers to chemical enhancement of water repellency. Most DWR treatments are proprietary chemicals, often including fluorocarbons, that enhance hydrophobic properties. When DWR is applied to fabric, water beads up and is repelled. DWR treatment is often applied to the fabric or fibers before garments are constructed. After repeated use and several washings, they need to be reapplied.

DWR is available as an aftermarket additive such as Nikwax Polar Proof, Scotchgard, and Blue Magic Tectron Wet Guard. DWR applied as an aftermarket product, usually as a spray, is not as durable as that applied to the fibers before the garment fabric is woven. Thus, aftermarket DWR needs to be reapplied every few months with heavy use or after every few washings.

SPECIAL USE CLOTHING

Sun Protection Clothing

Most clothing blocks solar radiation simply by mechanically covering the skin. Cotton, nylon, or polyester long-sleeve shirts and pants work best for full coverage. In hot climates, loose-fitting garments are more comfortable because they allow ventilation of internal heat and sweat. Sun protection garments use a tight weave to block radiation. The tighter the weave, the more the sun's rays are blocked. However, if the weave is too tight, it doesn't ventilate well, which in hot climates can be uncomfortable.

Light colors reflect light better than do dark colors. Garments sold as sun-protective clothing are usually light pastel or white. Heavier fabric, although hotter, blocks more sun because the fibers are thicker. Less stretch improves sun protection. Clothing blocks sun better when dry than when wet.

Special laundry additives can help improve sun protection. For example, Rit Sun Guard is washed onto clothing to help absorb ultraviolet radiation.

Sun protection clothing is rated by its ultraviolet protection factor (UPF), similar to the manner sunscreen is rated using sun protection factor, or SPF. Several rating systems used for outdoor clothing are:

- The American Association of Textile Chemists and Colorists (AATCC) uses Test Method 183.
- The American Society for Testing and Materials (ASTM) uses D6544.
- Australia and New Zealand use Standard 4399, which is recognized globally.

Examples of UPF ratings are below. Generally, ratings must hold up after 40 washings.

- UPF of 15 to 20 provides "good" protection, from 93.3% to 95.8% of ultraviolet light blocked.
- UPF of 25 to 35 provides "very good" protection, from 95.9% to 97.4%.
- UPF of 40 or greater provides "excellent" protection, above 97.5%.

Some examples of sun protection factors for clothing are:

- White cotton T-shirt: 5 to 9 UPF
- Tight weave long-sleeve shirt: 10 UPF or more
- Commercial sun protection clothing 30 UPF or more

Insect-Repellent Clothing

Insect-repellent clothing uses several properties to ward off biting insects and ticks. First, loose-fitting, long-sleeve, and long-legged clothing block skin from insects. A tight weave, as in sun-protection clothing above, blocks insects better than do loose weaves.

Insect repellents can be impregnated into the fibers of garments. These can reduce the need for lotions applied to the skin. For example, Buzz Off clothes are treated with permethrin. This odorless chemical, a synthetic form of a chemical found in chrysanthemums, repels mosquitoes, ticks, flies, and other insects. Permethrin is an insecticide. As mosquitoes and other insects land on clothing, the permethrin kills them by extending depolarization of sodium channels in presynaptic neurons.

Permethrin is bound to the fabric fibers; it is much longer lasting when added to fibers before the garment is constructed. Although bound tightly, it does wash off after repeated washings. Buzz Off is effective through 25 washings. It is registered with the U.S. Environmental Protection Agency as insect-repellent clothing.

Fire-Resistant Clothing

Most fire-resistant clothing, usually jackets, pants, shirts, and hats, is made from Nomex. Nomex is the trade name for Du Pont's fire- and flame-resistant chemical. The proprietary material is durable and is used by urban and wildfire firefighters.

Vapor Barrier Clothing

A vapor barrier system is a specialized clothing application used in cold weather, especially favored by someone who perspires profusely or is on a long expedition in which drying clothing and sleeping bags becomes difficult. It is often found in boots and sleeping bags.

The concept is that when a person perspires, sweat is wicked away from the skin into clothing and sleeping bags, which become damp. The idea is to allow the base layer to soak up sweat and keep the insulating layer dry for extended use. For example, one can wear a thin layer of socks, then put an impermeable waterproof vapor barrier sock over the top. Sweat is soaked up by the inner sock, and keeps the boot liner and footbed dry. However, many people do not like the clammy feeling of having socks wet with sweat next to the skin.

With sleeping bags, the system works similarly. One has a vapor barrier lining the same size and shape of the sleeping bag. One crawls in at night wearing a thin base layer of long underwear. Then, during sleep, moisture generated from the body stays in the fibers of the long underwear or condenses in the liner. Although one may wake slightly damp, the insulating fibers of the sleeping bag stay dry.

CLIMATE-SPECIFIC CLOTHING

Climates vary widely around the world. Here are some general tips for the five major types of climates with respect to clothing. Local temperature, weather, and altitude and activity play a roll in choosing a clothing system. One needs to be protected from the elements, but should avoid generating so much internal heat and perspiration from activity that one's inner layers become damp, or worse, soaked.

Cool, Wet, Windy Weather

Garments must repel the elements of rain, wind, snow, and cold, but still allow ventilation of excess sweat and heat. It doesn't do much good to keep the rain out if one becomes drenched with sweat. Choosing appropriate base and middle layers is essential.

Outerwear is the key factor in staying dry, whereas insulation is the key for staying warm. For mild temperatures and wet climates, select an uninsulated, breathable, water- and windproof parka. This usually has tight weave nylon and a laminate to improve water and wind resistance, but allows ventilation of excess perspiration. This may be a lightweight wool shirt or a heavy fleece sweater. Insulation varies with the factors discussed earlier.

For highly wet conditions, a nonbreathable jacket of rubberized or polyurethane-coated nylon is the most waterproof. These excel at blocking wind and rain, but do not ventilate heat well. Thus, they are suitable for sedentary activities in foul weather. The better that a fabric blocks wind and rain, the more it retains internally generated heat and moisture.

For windy conditions, wind chill can quickly cause hypothermia and frostbite. A proper windproof or wind resistant jacket is important. In some cases, a heavy nylon shell parka is necessary. In mild wind, a simple wind shirt or windproof fleece will suffice.

For situations of prolonged and extensive aerobic activity, one should select a highly breathable outer jacket that has less protection from wind and rain. These looser weaves of softer materials, the so-called soft shells, provide a good balance to spill excess heat and sweat, but offer some protection from wind, rain, and cold.

Insulation varies by conditions and persons. For cool temperatures, or people who always feel cold, bulky insulation layers such as fleece or fiberfill sweaters and pants are useful. People doing highly aerobic activities or who usually generate a lot of internal heat may have thin base and middle layers. One should have a spare set of base layer clothing to subsitute should the first set become wet.

Cold, Dry Weather or High Altitude

When ascending big mountains or visiting the polar caps, one typically needs clothing for extremely cold weather. Because cold air does not carry much moisture, these climates are usually cold, but not typically wet. Precipitation most often falls as snow. Wind tends to be more of a factor.

For high-altitude mountaineering or polar exploration, the down suit is standard. This one-piece, thick, light suit is encapsulated in a tightly woven nylon shell. It does not function well when wet. However, it is perhaps the warmest outfit for cold, dry climates. The down suit should be reinforced at the elbows and knees so it withstands rugged use. Parkas should have a hood with an extended face-shielding area, sometimes called a tunnel or snorkel. Fur or fleece lining in the face shield can help block wind, snow, and rime. The hood should accommodate a helmet if necessary. Pockets should be large enough to store heavy mitts and small items. Sometimes, water bottle pockets are necessary so water can be kept inside the parka to keep it from freezing. A waist drawstring helps seal out the weather. Some climbers prefer short parkas, so they don't interfere with a climbing harness. Others, such as workers or researchers, prefer long parkas cut to cover the buttocks and pelvis.

Pants should have abrasion patches on the seat and knees and should be thick enough to provide adequate insulation without restricting movement. Bib overalls are warmer than pants because they have extra material over the torso and cover the midriff, where wind and snow can enter under the trim of a parka.

Insulated canvas duck provides excellent durability and mobility, especially with activity that may be very hard on clothing. It is warm and wind-resistant when dry, but offers very little protection from rain.

One should choose clothing that can easily be layered for extreme cold; thus, it may need to be one or more sizes larger to allow for ample movement. Some climbers prefer to add a layer of insulation, such as a down or fiber-fill parka, on the outside of the outer layer. This is useful when an aerobic activity, such as mountain climbing, is temporarily halted, to allow adding a layer quickly without removing the outer shell.

One should wear thick long underwear and socks. Heavy-duty mittens with long gauntlets and retention cords, thick hats, neck gaiters, and goggles are the norm. Insulated gaiters and overboots help keep feet warm and dry. All exposed skin should be covered.

Mild Climates

In mild climates, where rain and cold are minimal, one should choose a light next-to-skin layer with a layered garment for insulation and wind and water protection. This is usually windproof fleece, a soft shell jacket, or a vest. The key in mild

climates is to layer and be prepared for the worst possible weather conditions that could likely occur.

In some climates, shorts and T-shirts may be adequate. One is able to dissipate excess heat and perspiration easily. Backpackers in sunny climes prefer nylon shorts and T-shirt because they are comfortable and rugged. Nylon shirts dry quickly. However, sun damage is a risk if the skin is not covered, so sun protection guidelines should be followed.

Some hikers prefer a lightweight long-sleeve shirt for sun protection and lightweight nylon pants. The pants dry quickly and protect legs from brush. Wool or fleece pants tend to be too heavy, and cotton often is not warm enough. Plus, unlike nylon, other materials tend to snag on brush. Alternatively, a polyester-cotton blend, such as the pants worn by many park rangers, is durable, comfortable, and quick-drying.

In slightly wetter or cooler conditions, especially at dawn or dusk, one may need a long-sleeve shirt and pants to protect from the mild wind, light rain, or cool temperatures.

Hot and Dry

Sun protection is best done with both sunscreen and sun protective clothing. Lightweight, loose-fitting cotton, cotton blend, or synthetic fabrics improve cooling as they allow sweat to evaporate and allow air to circulate next to the skin. They also directly block the sun's harmful rays.

A wide-brim hat that shields the face and sunglasses are essential. In jungle or tropical environments, long-sleeve, long-leg garments also provide protection from sticks, rocks, and other debris and from dangerous plants, insects, and other animals.

Water

Water clothing is a special issue because water can promote cooling that leads to hypothermia.

For occupations or recreations that include significant time in water, neoprene is the best insulator. For water sports and occupations, noncoated or "smooth skin" neoprene provides excellent wind protection and mobility. For activities in which someone is regularly submersed, such as diving or snorkeling, nylon-coated neoprene is much more durable.

Neoprene wetsuits come in many styles and models. They should be snug-fitting to limit excess water in the suit, but not so tight as to restrict movement. For occasional dips into water, a neoprene shirt, shorts, or both may suffice. For longer escapades, one may need a "farmer john," which is a sleeveless, long-leg suit. This is sometimes coupled with a neoprene jacket for versatility. Alternatively, one can wear a wetsuit with short arms and legs, or long arms and legs. The more neoprene that covers the skin, the warmer the wearer.

Thickness is important. Cold-water surfers and divers have long-leg, long-arm suits that are 5 mm (0.2 inches) thick. In more temperate climates, rafters or kayakers may choose a 3-mm (0.13 inch) suit.

Neoprene accessories that dramatically improve warmth include hats, balaclavas, gloves, booties, and socks. In addition to providing warmth, neoprene booties protect the feet from trauma in aquatic environments.

FOOTGEAR

Boots should be sized and fitted professionally and may require "break in" time to soften them. Some boots are ready to wear right from the box because they are made from supple materials such as canvas and soft leather. Others, especially mountaineering and heavy backpacking boots made of leather, require several days of wear to soften the leather and allow the materials to form to the feet before they are taken into the wilderness.

The sole should be comfortable and the laces snug. The boot should be neither so tight as to cut off circulation nor so loose as to allow the foot to slide in the boot. One should be able to flex the foot and walk or hike comfortably. The heel should not slide in the heel cup, and the foot should not be pinched or squeezed. The toes should have ample space and touch the tip of the shoe. Feet should not slide forward when walking down hills. Heels should not lift when hiking up steep slopes.

Consider a custom fit using a moldable liner and custom foot bed such as an orthotic. These should be fitted professionally and dramatically improve the comfort and life of the boots.

Some soles have a half or full shank for added support and stiffness. Soles are often constructed with a lasting board or platform for stability and stiffness. Some come with stiff lug soles for added traction. Boots come in low, mid, or high top, depending on the need for support versus the desire for light weight and mobility.

Construction can be canvas, synthetic material, or leather. Canvas and synthetics are light, ventilate well, and are less expensive. Leather provides the best support and more durability, but can be hot, heavy, and expensive. Split-grain leather is lighter and softer than full-grain leather. However, full-grain leather, because it is thicker, is usually more resistant to water and abrasions. A laminate layer such as Gore-Tex improves water resistance.

Many boots are constructed to be water resistant and durable and include baffles in the tongue, seam sealers, extra stitching, and high cuffs. These features also add weight and expense. Uppers are connected to the sole by two methods. Sewing is more durable and sometimes gives a stiffer feel and longer break-in time. Cementing the upper and sole is less expensive and generally lighter and softer, but not nearly as durable. Laces are generally nylon, which are lighter and more durable than leather or cloth laces.

For leather, canvas, or nylon shoes, a waterproof spray or wax-based coating is sometimes added by the manufacturer or the consumer. These help improve water repellency and keep feet drier. However, most treatments only last for a few outings, and thus need to be frequently reapplied, especially in heavy rain or mud.

Examples of common outdoor footwear include:

- Double plastic mountaineering boots are the standard for mountaineering. These are plastic lace-up or buckle shells. The inner insulating boot is usually lace-up.
- Pack boots work for general all-purpose snow travel. These are soles and lower boots of rubber sewn to high top leather lace-up uppers. They usually have a thick felt lining and are warm and durable.
- High-top leather work boots are standard for many occupations. They usually have a stiff rugged sole, full shank, and steel toes.
- High-top hikers are for backpacking or hiking. These come in a huge variety of materials, designs, and constructions. They range from heavy high-top leather backpacking/mountain boots to lightweight desert travel boots.
- The low-top hiker, also called an approach shoe, is a lighter version of the backpacking boot. It is meant for day hiking, scrambling, or approach trails.

- Water boots for fishing, kayaking, and rafting are made of soft rubber soles for traction on wet rocks, with neoprene uppers to provide warmth and durability.
- Rock climbing shoes have sticky rubber soles and a very tight fit to provide friction and grip on vertical rock.
- Sport sandals are used by water enthusiasts to allow adequate ventilation, quick drying, and some support to protect feet from water hazards.
- Trail running shoes are similar to approach shoes in that they provide good under-foot support and are very light. They do not provide much ankle support or torsional stability for rough terrain.

Most boots do not need any type of aftermarket coating if chosen properly for the conditions and activities. The exception is that when hiking and mountaineering in wet conditions, one may choose to spray or apply a DWR. These can be wax, petroleum, or synthetic based and are applied as a rub or spray to make leather, canvas, and synthetic fabrics repel water. However, they only last for one trip, so they need to be reapplied frequently.

CLEANING, STORAGE, AND REPAIR OF CLOTHING

In general, most outdoor clothing performs better if it is kept clean. In general, you should follow cleaning instructions on the manufacturer's label. These usually provide specific instructions. There are many types of cleansers, powdered or liquid detergents, and liquid fine washable solutions. Some clothing may need to be hand washed or dry cleaned.

In general, front-loading washers that tumble clothing are much less damaging to clothes than a top-loading washer, which has a central agitator. Follow temperature guidelines recommended by the manufacturer and use a dryer only if recommended, because some fabrics are better maintained if air dried. Always store clothing properly when not in use, either hung or folded in a clean, dry place.

Always get clothing repaired professionally, especially if it is expensive laminated outerwear with taped seams, special zippers, or specialized fabrics.

Box 82-2. Basic Backcountry Clothing Repair Kit

Awl
Buttons
Sewing needles, #18 and #7
Safety pins
Seam ripper
Straight pins
Thimble
Thread, #69 nylon

For emergency repair, carry thread and a needle. For heavy-duty clothing or backpacks, carry nylon thread, a heavy needle, and a thimble for driving the needle through heavy clothing. See Box 82-2 for a list of a basic backcountry clothing repair equipment.

Store all clothing in a clean, dry indoor closet. Avoid storing clothing in a damp garage. For wool, a variety of clothes-moth larvae can eat the fibers. Kill the eggs by running the clothes through a warm clothing dryer or washing them, then store them in an airtight bin or garment bag. Use repellents. Cedar is the most common natural repellent. It comes in forms such as balls, sticks, or liners for drawers and closets. Mothballs made from paradichlorobenzene work, but are more toxic to humans and impart an odor to clothing.

Footwear should be cleaned with a warm, damp cloth. Shoes and boots should be allowed to air dry, not put in a clothes dryer or next to a hot fire. If they are soaked, a few wads of newspaper placed inside will help them dry quicker and retain their shape. Aftermarket treatments to soften or waterproof footwear should only be used at the manufacturer's recommendation.

The references for this chapter can be found on the accompanying DVD-ROM.

83 Nonmedical Backcountry Equipment for Wilderness Professionals*

Christopher Van Tilburg

Whether for work, research, or recreation, specialized outdoor equipment enhances safety, ensures survival, and provides comfort, sustenance, and shelter. When chosen carefully and cared for properly, equipment may last a long time. This chapter examines basic concepts for choosing outdoor gear.

GENERAL CONCEPTS FOR CHOOSING EQUIPMENT

There are two basic concepts for selecting equipment and packing for wilderness travel. One concept is to carry as much of the best equipment as one can reasonably haul and afford. This theoretically enables one to prepare for as many outdoor conditions and mishaps as possible, and allows for maximum variability and flexibility. This works well for long trips with vehicles, boats, or porters for support, especially for research or scientific expeditions. However, this style is not practical if terrain, weight, space, or funds limit equipment choices.

The more common concept, especially endorsed by recreational travelers, is using light, compact equipment. This is sometimes referred to as "light is right." This author promotes the concept as "weight less, take less" because even with light, compact equipment, one may elect to leave a piece of gear at home because the luxury or increased margin of safety is not worth the extra weight and bulk.

An alpine mountaineering style perfected by long-distance hikers has been developed around this concept. Some basic principles of choosing and using light, compact equipment and omitting gear are:

1. Carrying less and using lighter equipment saves energy, and often leads to a safer, more enjoyable experience. One can travel faster and maneuver better and select from a wider variety of gear that is easier to repair. Pay attention to weight when shopping; attempt to avoid accessories that add weight but are not really necessary.
2. Using light gear has some risk. It can be less durable, less reliable, and more expensive. For example, aluminum ice axes and crampons are tempting, but they may not withstand years of hard use or even one big fall on an icy glacier. A plastic avalanche shovel is not nearly as durable as a heavier aluminum one.
3. It makes sense to skimp on weight when maximal performance is not necessary. For most recreational outdoor enthusiasts, maximal performance is rarely necessary. For example, instep crampons may be adequate in place of full-frame crampons for low-angle, nontechnical snowfield hiking. Even when full-frame crampons are used, they are rarely used at the limit of the integrity of the structure or the strength of the materials.
4. Choose simple tools and gear, because they are often more reliable and durable. Try to get gear that is easy to adjust and repair in the field. Avoid complex gear that requires special tools for set-up, repair, or maintenance. For example, choose "tool-free" crampons that adjust without a wrench or screwdriver; use a compass with an easily adjustable declination correction.
5. Plan well but learn to improvise. One should never cut corners on an emergency kit or survival gear.
6. Multifunction gear is a great way to shed weight and space. However, performance of each function may be compromised. For example, multitool pliers do not have the strength or power of simple tongue-and-groove pliers. Also, if the tool breaks or is lost, one has lost more than just a single item. For example, most experts agree that in a rescue, an avalanche probe is superior to ski poles that convert to a probe.
7. Practice is key to using equipment properly, especially with equipment that is highly technical, complicated, or electronic.
8. Everyone does not need the same gear. Members of a party should divide equipment and carry different tools if necessary. This way, the team has a greater selection to match tasks and can split up the weight and bulk of items like stoves, tents, medical gear, and research equipment.
9. Some newer, lighter, and "high-tech" tools use cutting-edge materials. Carbon fiber, titanium, magnesium, aluminum, plastics, and other materials create strong, light equipment. If weight, durability, and maximal performance are an issue, consider using tools made of these materials. If you do not need extremely lightweight equipment, then traditional materials may work just fine. For example, a standard aluminum avalanche probe is more durable and only slightly heavier than a new carbon fiber probe.

*The present chapter is based on John Gookin's fine treatment of this topic in the previous edition.

ESSENTIAL EMERGENCY EQUIPMENT

When planning any sort of wilderness activity for work, research, or recreation, travelers should prepare a basic emergency kit. An example of a basic kit is shown in Box 83-1. Previously, mountaineers and climbers referred to this list as the "10 essentials" because earlier writings described 10 key ingredients to an emergency survival kit. However, most authors and experts now recommend a basic list of essential equipment that may include more or less than 10 items and varies according to context, as follows:

Who?

The number of people and level of expertise are crucial to how much equipment to bring. For example, if on a private raft trip with no other medical providers and elderly or young participants, one might take a large emergency kit. For a research trip with a group of doctors and two guides, one might carry only a small personal kit. Number, medical background, and age of companions contribute to deciding what to carry.

What and Where?

Most kits are designed for basic outdoor adventures like climbing, hiking, backpacking, and mountain biking. However, a dive photography expedition to Hawaii requires much different gear than an international medical relief program to Africa. Kayakers and rafters need different equipment than do cross country mountain bikers. Persons traveling overseas, to high altitude, or upon the ocean require specialty equipment related to climate and terrain.

When?

Time of year may be a factor in determining how much gear is carried. During winter, one may carry more survival gear, such as equipment for avalanche safety, snow camping, and hypothermia and frostbite prevention, than in summer while on the same itinerary.

How Far? How Long?

Longer trips farther from help dictate more advanced equipment for safety and survival.

Commercially available emergency kits usually include first aid, survival, and repair supplies. Alternatively, a wilderness enthusiast can prepare his or her own custom kit. Three basic sizes for emergency kits are generally in use.

- A basic emergency kit may be used for day outings (see Box 83-1 for an example of a basic compact kit). This includes enough equipment to cover one unexpected night out in mild weather. A small kit only provides the bare minimum of survival gear for food, shelter, water procurement, navigation, fire building, first aid, and equipment repair. Everyone should carry a personal emergency kit, even if there is a large group emergency kit.
- A large multiday kit is intended for climbing, backpacking, kayaking, or rafting overnight trips that may last from 1 day to 2 weeks. It anticipates advanced needs for water procurement, shelter building, and navigation. These are often carried by guides, trip leaders, or outdoor professionals.
- Lengthy expeditions of a week or more may require specialized equipment and a broad range of supplies for situations that may involve large numbers of people, encountering problems in extreme environments.

Information about retail emergency kits is found at www.orgear.com or www.omnikit.com. Complete medical supplies are listed in Chapter 81. Equipment for vehicles is listed in Box 83-2.

LIGHTS

Headlamps

When light is needed in the backcountry, headlamps are the most reasonable tool because they allow hands to be free for tasks. Brighter headlamps often consume batteries faster. Features to consider include light distribution, durability, water resistance, and maintenance. The headlamp should be compatible with a helmet if that is a requirement. Small lamps usually have limited battery power. Large battery packs, such as those stored on the back of the helmet or on a waistbelt, provide much longer life for caving or winter night travel.

A good headlamp has a spare bulb stored within it, is weather resistant, and has smooth light distribution with an adjustable beam. A good field of illumination provides even lighting in front of the user and well into the field of peripheral vision. Waterproof lamps have O-rings or other gaskets to resist rain and sweat. Dive headlamps are completely sealed and submersible. Cavers often use long-lasting carbide lamps.

For headlamps, light bulbs are usually incandescent, tungsten, halogen, xenon, or light-emitting diodes (LEDs). The bulb has three key properties: brightness (distance), duration (time of usable light), and shape of beam. These depend on type of bulb, shape of housing, and battery type and size.

Incandescent bulbs are less expensive and consume moderate amounts of battery power. Halogen and xenon bulbs emit whiter and brighter light, but usually consume power at much higher rates. LEDs are small, have a smooth light, and use batteries efficiently. Because they are not nearly as bright as other bulbs, headlamps using LEDs usually have three or more diodes lit simultaneously.

With two equal AA batteries, most headlamp bulbs perform as follows:

- Xenon halogen: 3 to 4 hours on maximum power; 10 hours on less power
- Standard tungsten: 7 to 10 hours on maximum power
- LED: 30 to 40 hours on maximum power, up to 120 hours on limited power

Some headlamps have two types of bulbs: a bright xenon or halogen for important tasks that require bright light and three or more LEDs for functioning around camp or reading. These lamps are an excellent choice for professionals. The bright light can be used for night search and rescue or identifying animals. The LED can be used for documenting research or repairing gear.

Shape of the beam depends on housing. Some have a narrow beam with a longer range, sometimes up to 20 or 30 meters; these headlamps are good for night searches or route finding. Headlamps with a wide but short beam concentrate light for near work such as within a tent or vehicle. The best lights have adjustable beams to allow either close or distance illumination.

Batteries influence brightness and duration. For example, headlamps with AA or AAA batteries have neither the bright-

Box 83-1. Basic Emergency Kit

A basic emergency kit should include first aid, survival, and repair materials.

FIRST AID KIT
Waterproof cloth first aid tape is an essential first aid item, because it has a wide range of uses and is difficult to improvise in the field. A basic kit should include wound care supplies and personal protective equipment (CPR mask, gloves, face mask) at a minimum. Wilderness medicine professionals may carry more advanced supplies, tools, and medicines (see Chapter 81). This kit should also include sunscreen.

FIRE-STARTING MATERIALS
Carry windproof and waterproof matches in a watertight jar with a striking swatch, a lighter, or a metal match (flint with steel striking blade). One may carry fire starter as well, such as petroleum jelly–impregnated cotton balls or commercial fire-starting tablets.

NAVIGATION
The bare minimum is a compass with declination correction and a topographic map. An altimeter is useful, especially in the mountains or canyons. One may choose to carry a GPS (global positioning system) unit, but should always have a compass for backup (see Chapter 85). Surveyor's tape can be used to mark dense woods. Wands (3-foot or 1-meter bamboo poles with surveyor's tape on the top) are useful for marking crevasses and snow routes.

POWER
Carry extra batteries for headlamp, camera, avalanche beacon, medical equipment, GPS units, and other electronics.

SUN PROTECTION
Carry sunscreen and sun-protective eyewear, either goggles or glasses. A sun hat and sun protective clothing are important.

HEAT
Chemical hand or foot warmers are useful.

LIGHT
Headlamp—include a spare bulb and spare batteries.

REPAIR MATERIALS
The basic repair supplies can include a multitool. This should include pliers, wire cutters, screwdrivers, small knife blade, and scissors (see Box 83-4).

Duct tape, 2 inches × 5 feet (rolled then squeezed flat), is extremely useful and difficult to improvise in the field. Basic repair materials should include thread and awl, wire or paper clips, plastic cable ties, polyurethane plastic straps, and nylon cord (3 m × 4 mm).

CLOTHING
In addition to what one is wearing on a wilderness outing, one should carry at least one layer more than what one expects to use on the trip. Consider carrying enough clothing to survive the unexpected night out. See Chapter 82 for detailed information on clothing.

EMERGENCY SHELTER
Have the ability to improvise an emergency shelter using materials in the wilderness or carry an emergency bivouac sac. This can be a simple, compact plastic tube shelter or even a large plastic garbage bag.

COMMUNICATION
For basic communication in varied terrain or storms, a plastic whistle can be much louder than the human voice. A signal mirror is useful for communicating with rescue aircraft. Cell phones, satellite phones, VHF radios, and FRS (family radio station) radios can aid in emergencies.

ness nor the duration of those powered by a C battery belt pack or a flashlight with two, three, or four D batteries.

Flashlights

In the wilderness, handheld flashlights are not as useful as headlamps because the hand occupied by the flashlight is unavailable for other tasks. Smaller lights house smaller batteries, such as AA or AAA, and thus have relatively limited brightness and burn-time. Larger flashlights that house C or D batteries are brighter and burn longer. Plastic housings are lightweight but not as durable, depending on the thickness of the plastic. Aluminum housings are a bit heavier, but much more durable. Flashlight bulbs, housings, and other features are similar to those of headlamps discussed earlier.

Lanterns

Lanterns are valuable for base camp operations or on trips in which much light is needed after dark, such as when cooking for large groups, reading, or doing research. Liquid fuel lanterns burn white gas, a purified form of gasoline, yielding more light per pound of fuel than do battery-powered lamps. With a gas lantern, one should carry spare mantles and transport the lamp in a plastic case. The mantles and glass globe are fragile, and gas must be stored safely. These lanterns get very hot. Hazards associated with liquid fuel are listed in Box 83-3.

Compressed gas lanterns that run on butane, propane, or a mixture of both are fuel-efficient and lightweight. The gas is contained in a compressed cylinder and thus is not as messy as liquid gas. For the same weight, these lanterns are brighter than liquid gas lamps. Like white gas fuel lanterns, those with compressed gas have fragile globes and mantles.

Battery-operated fluorescent-bulb lanterns provide efficient, smooth, usable light without the risks associated with fuel or the fragility of mantles or glass globes. These lanterns are replacing liquid and compressed gas lanterns; they are great for commercial trips, where one might have inexperienced campers who are unfamiliar with the hazards of lanterns, and for international travel, where breakage can occur.

Box 83-2. Suggested Emergency Equipment for Vehicles

Two spare tires
Tire jack and iron
High lift jack
Sand and snow plate for jack
Jumper cables
Tow strap
Tow rope
Extra gas, at least 5 gallons (20 liters)
Extra oil, 2 quarts (2 liters)
Extra food and water for emergency rations
Tire pump
Snow shovel
Sand or dirt shovel
Fire extinguisher
Headlamp for night repairs
Road flares
Tie-down straps
Flat tire repair kit: awl, rubber cement, patch, or plug material
Flat tire repair canister: pressurized glue that inflates tire and plugs flat
Spare valve stems for tires
Tool kit: screwdrivers, wrench or socket set, pliers, and tongue-and-groove pliers
Repair kit: electrical tape, wire, duct tape
Tarp and spare blanket or sleeping bag
Comprehensive first aid kit

Box 83-3. Stove and Liquid and Gas Lantern Safety

Backpacking stoves often tip over. Burns commonly come from spilled pots of scalding water, from using bare hands near a flame or frying pan, or directly from burning stove fuel. Lantern globes become very hot. To minimize problems:

- Supervise the stove area so that no one tips over a pot.
- Do not allow people to sit in the impact area around a burning stove.
- Use a ladle instead of pouring from the pot.
- Use a handle when picking up a pot or lid.
- Do not carry a pot of boiling water.
- Do not pour boiling water into a cup that is in someone's hand.
- Use caution when placing a lantern on a rock or log.
- Avoid cooking in an enclosed tent or snow cave. Avoid using a fuel lantern in a tent. Carbon monoxide poisoning can result in death.

Box 83-4. Suggested Multitool Options

Awl
Bottle opener
Can opener
Corkscrew
File
Knife
Pliers
Saw
Scissors
Screwdriver, flat
Screwdriver, Phillips
Tweezers
Wire cutters

Oil and kerosene lanterns have been replaced by the aforementioned liquid fuel, compressed gas, and battery-operated types. Lamp oil and kerosene are difficult to find, burn with noxious fumes, and are not practical for wilderness travel.

Candle lanterns are used occasionally by campers. These are simple and reliable, but they emit minimal light that is bright enough to read or eat a meal by but usually not enough for cooking or procuring water. They have an open flame, so they can burn the user or ignite tents, sleeping bags, or clothing.

Lantern Safety

Liquid fuel and compressed gas lanterns should never be used in a tent, vehicle, cave, or other enclosed space, because lanterns emit carbon monoxide. Even in extreme emergency and survival situations, campers who would otherwise survive the night in a storm die of carbon monoxide poisoning. In addition, all lights with an open flame or even those enclosed in a glass globe can ignite clothing, sleeping bags, or tents. They should be used with extreme caution. For these reasons, many professional guides and outfitters prefer battery-operated lanterns.

TOOLS

Multifunction Tools

Recreationalists most often use a compact multifunction tool (multitool) or a pocket knife with accessories like bottle and can openers. Professional guides or outfitters may need a compact folding knife blade and a separate multipurpose tool with many tool components. See Box 83-4 for a basic idea of multitool components.

Most experts agree that a pocket knife or a pocket multitool with pliers is sufficient for emergency use or basic camp chores. However, multifunction tools do not work as well as do specific tools. For example, a real screwdriver may be needed to apply sufficient torque and control to repair a ski binding. To repair a broken bike chain, tongue-and-groove pliers are much easier to use than are small multitool pliers. If rafting, one may need a boater's knife, rather than the small blade of a multitool, to cut rope.

At a minimum, a multitool should have pliers, wire cutters, a knife blade, a can opener, and flat and Phillips head screwdrivers. It is useful in many situations to have a saw, awl, tweezers, and scissors.

Multitools are available in two configurations. Some, especially the newest designs, are based on the platform of a pair of pliers. These tend to be heavier, but many wilderness enthusiasts and professionals agree that the pliers are indispensable for equipment repair. The second style is based on a folding pocket knife, such as Swiss Army brand. These contain many features and components, but usually do not have pliers. Thus, multitools based on pliers are usually preferred. Sometimes one chooses to carry a separate, larger knife, as described in the next section. If one carries a folding-knife style multitool, then he or she must carry a separate pair of pliers.

Choose the multitool that fits specific needs by performing these comparisons between different models:

1. Open all accessories. Blades that lock open are generally more controllable than are nonlocking blades. Blades that are difficult to open when new may be impossible to open after a few trips.
2. Determine which tools will really be used.
3. Tightly grip the pliers and test them on a piece of wood or on a fat tent pole. Some tools have sharper handle corners than others; the smoother edges may provide more control, better fit, and less pain on gripping. In general, there is more torque with a larger tool or with compound leverage.

For specific applications, one may need specific tools. Mountain bikers need bike chain repair tools, a tire pump, a patch kit, an extra tube, and hex wrenches. Skiers and snowboarders need a #3 Phillips screwdriver for binding screws. Rafters need tools as well as a pump and a patch kit to repair a boat or an oar.

Knives

For knife blades, many survival experts feel that a nonfolding knife is best. However, often this is not practical because folding knives can be stowed in the handle and are more compact. Persons who prefer a folding knife usually choose one with a locking blade so that more force can be applied without risk of the knife folding accidentally. These knife blades are generally 2 to 6 inches in length. Sometimes they are serrated to assist in cutting rope.

Blade Shapes

Knife blades have many shapes, depending on their intended use, for example:

- Hunting knives range from all-purpose utility knives to special skinning or gut hook knives for field dressing game. Large animal knives generally have longer and wider blades compared to those for upland birds and waterfowl.
- Fishing knives, such as fillet knives for field gutting and dressing fish, usually have long, narrow blades.
- Dive knives are usually straight and have symmetric blades and waterproof handles. They usually come with a specialized holster to attach to a diver's leg on the outside of a wetsuit.
- Rescue and river knives, such as those used by kayakers, rafters, and canoers, are often serrated to easily cut rope and cord in emergencies. The holsters are designed to attach to a life vest.

Construction

Most blades are made from high-carbon steel. Steel is durable, and when it contains high carbon content (typically 0.5% to 1.5%), it is strong, easy to sharpen, and holds an edge during regular use. However, it oxidizes easily and thus discolors after use or with weather exposure. There are many types of steel alloys, using more or less carbon and other alloying elements.

Stainless steel, similar to high-carbon steel, contains enough chromium to resist stains and rust, so it is excellent for use on long trips. Surgical stainless steel is less frequently used. It has more chromium, so it is stain- and rust-resistant and cleans easily. However, the blade does not hold an edge as well as high-carbon steel.

Titanium is light, durable, and resists stains well. The main drawback is that titanium is more expensive than steel.

Ceramic, or zirconium oxide, is extremely hard and thus needs to be sharpened infrequently. But ceramic knives are brittle, and they need to be sharpened with a special diamond sharpener.

Handles

Be sure that the handle is strong, easy to clean, and fits comfortably in the hand. Wood handles look nice, but plastic handles are more durable and easier to clean and sterilize. Some knives have rubberized grips; if present, these should be well bonded to the blade.

Care

Knife maintenance starts with cleaning. Even the finest stainless steel corrodes if moisture, food, or dirt remain in contact. When a knife becomes dirty, it should be cleaned immediately. Keep knives clean in the field by wiping with a clean cloth, with the sharp side of the blade pointed away from the wiping hand. If used for food or edible game, they should be disinfected using hot soapy water or alcohol-based cleanser. Locking knives should be lubricated with light oil as recommended by the manufacturer.

If a tool is immersed in salt water, it should be rinsed in fresh water, dried, and then lubricated. Lubrication not only greases the joints but also displaces water. Joints are more prone to corrosion because of electrolysis between different metals in the tool.

Keep knives sharp by using a whetstone or ceramic stick in the field just before or after use. If a knife is used excessively, it may need to be professionally sharpened. Use caution when sharpening with a whetstone to avoid thinning and reshaping of the blade.

Shovels and Trowels

A small military folding steel shovel is often adequate for emergency use for vehicles or for expeditions using pack animals. However, for vehicle expeditions or trips, a full garden spade to shovel dirt or mud and a large grain scoop to move snow are necessary for extricating a vehicle or clearing a road.

For backpacking, small and light trowels work well for digging cat holes for improvised latrines. These usually have blades of 4 to 6 inches. Stainless steel trowels usually have a folding handle. Plastic trowels are not as durable, especially if one encounters rocks or dense, hard dirt.

For backcountry snow travel, a compact snow shovel with a removable handle for storage in a backpack is necessary for avalanches, digging snow shelters, and similar uses.

Saws and Axes

Saws are likewise necessary for vehicle trips for removing downed logs from the road or for large expeditions that need to cut downed timber for fuel. For backpacking, a saw attached to a multitool is sufficient only for dire emergencies, because they are not much good for cutting firewood and work only for cutting tiny limbs for emergency shelters.

Cord saws, also called cable or chain saws, are also best carried for emergencies only. These are long strands of cord, cable, or chain that are gripped at both ends, then used to cut wood.

Box 83-5. Basic Backcountry Gear Repair Kit

Adhesive nylon patches for clothing, backpacks, and tents
Cable ties
Duct tape
Flat 1-inch (2-cm) nylon webbing
Nylon cord, 2 mm × 30 m
Safety pins
Seam adhesive for repairing clothing, backpacks, and tents
Sewing kit
Paper clips or similar wire
Tent pole splint, aluminum tube with hose clamps

Folding saws are usually around 8 to 12 inches long when collapsed and are double that size when expanded. They are small enough to carry in a backpack, on a pack animal, or in a vehicle. Importantly, the blade is stowed in a collapsed position, protecting against accidental injury to a person or to other gear.

For vehicle- or pack-animal–supported expeditions, larger folding saws are available that have blades of 12 to 24 inches in length. A compact ax can be useful for chopping firewood.

Gear Repair

One should carry some basic materials with which to repair gear. Anything that can serve as a fastener, patch, or adhesive can potentially put a broken or torn piece of equipment back into service. Box 83-5 suggests contents for a basic backcountry gear repair kit.

WATER STORAGE

Water disinfection is discussed in Chapter 61. Options for water containers are discussed below.

Rigid plastic bottles are common. Choose 1-liter (1 qt) bottles that have a secure, watertight lid. Narrow-mouth bottles are easier for drinking; wide-mouth bottles are easier to fill. Bicycle water bottles with snap-open or pop-up spouts are flimsy and tend to leak when inverted or squished in a backpack. In cold weather, put bottles in insulated sleeves to help prevent freezing, or pack water in a parka to use body heat to prevent freezing. Thawing frozen water bottles is extremely difficult in the field and consumes much fuel. Water containers crack when water inside them freezes, rendering them useless after the water is thawed.

Larger bottles, up to 5-gallon (20-liter) size, are usually carried in vehicles. Collapsible vinyl bottles and bags can be carried empty in a backpack, to be filled at camp for ample water for cooking, bathing, cleaning dishes, and refilling personal water bottles. Accordion-style collapsible bottles tend to crack after repeated use.

Nylon-lined water bags are light and collapse when not in use. They are more common for large expeditions when extra water storage is needed in base camp and when carrying bottles is cumbersome, such as with pack animals. Water bags pack easier because they are relatively flat when filled, unlike rigid bottles.

Vinyl hydration pack bags have an attached drinking tube that allows sipping while walking, biking, or boating without having to open a backpack or unscrew a lid. The tube has a mouthpiece that consists of a slit valve, which remains closed when not in use; the user bites the valve, opens the slit, and sucks out water. The vinyl bags come in 1-, 2-, or 3-liter sizes. Many backpacks now have an internal sleeve to specifically hold a hydration bag. Drinking tubes freeze quickly in very cold conditions, but they are very helpful in hot or moderate conditions. Neoprene sleeves help minimize, but do not eliminate, frozen drinking tubes.

Between trips, clean plastic water containers by running them through the dishwasher. Soft plastic containers and hydration bags need to be scrubbed by hand with hot soapy water, then rinsed thoroughly. To disinfect and minimize mold and bacterial growth, rinse with chlorinated water (1 teaspoon [5 cc] of bleach in 1 liter [1 quart] of water) and dry thoroughly.

Before using a water container that has been stored for more than a few weeks, eliminate odors by washing with baking soda solution (1 teaspoon [5 cc] of baking soda in 1 liter [1 quart] of water), then rinsing with water. Clean tubes and valves on the drinking hoses after each use. Replace them when worn or excessively soiled.

HEAT PACKS AND HEATERS

For wilderness travel, heat packs can be useful to prevent hypothermia and frostbite. These are available for feet, hands, and backs. Most disposable heat packs use a chemical reaction to generate heat. When the pouch is opened and agitated, iron is exposed to oxygen, oxidizes, and produces iron oxide, commonly known as rust. This reaction generates heat and lasts for 4 to 8 hours. Sodium chloride is used as a catalyst and carbon disperses the heat. The elements are contained in a polypropylene pouch, and use cellulose as filler and vermiculite as a heat retainer.

Heat packs are effective for preventing frostbite when used in gloves and boots, and for core warming if placed under the axillae, in the groin, or on the torso. Some heat packs are configured in a belt so they can be worn on the low back. Many have adhesive so they can be attached to gloves or socks. Most manufacturers recommend against using them directly on skin to eliminate risk for burns.

Boot heaters for skiing, biking, hiking, or climbing boots have small rechargeable battery packs that fit on the back of the upper boot, near the cuff. Cords run into the boot. The heating element is placed just under the ball of the foot on a footbed. These heaters can provide up to 18 hours of heat production at low power. They are most useful for 1- or 2-day cold weather outings, because they need to be recharged, and for persons prone to frostbite.

OPTICS

Compact binoculars and telescopes are useful for backpacking when space and weight are priorities. Larger binoculars and telescopes add bulk and weight, but usually have wider objective lens, better magnification, and wider field of view. These are best for base camps, research stations, and vehicles.

Optic devices use two numbers to identify the two chief properties: magnification and objective lens diameter. For example, 6 × 25 has a magnification of 6, which means the object appears six times closer than with the unaided eye. The objective lens, the one farthest from the eye, is measured in millimeters. Larger

objective lenses allow more light into the binocular or telescope, which is better for low-light situations such as dawn or dusk. In the example above, 6 × 25 has an objective lens of 25 mm (1 inch).

Other factors are useful when comparing optics. Exit pupil is the diameter of light beam that reaches the eye measured in millimeters, calculated by dividing object lens diameter by magnification. In the example above, 25 divided by 6 is approximately 4. The larger the number, the more light that reaches the wearer's eye; thus, the optic device's view is brighter. Field of view, measured in feet, describes the width at 1000 yards (305 feet). This usually correlates with magnification; lower magnification allows a broader field of view. High magnification, such as with a telescope or gun scope, yields a smaller field of view. Eye relief is the distance in millimeters that the field of view is visible. This is important for persons who wear glasses, who may require eye relief of 11 mm (0.43 inch) or more to accommodate wearing glasses while looking through the device.

Other factors when choosing optics include the following:

- Lens coatings cut glare.
- Waterproof housings protect internal lenses from rain or snow.
- Plastic housings, sometimes called armor, protect the device when stowed during travel, when dropped, and in rugged environments.
- Focus types should include one or two adjustments. One is a central focus found on binoculars and monoculars. Binoculars usually have a separate focus on one eyepiece. This is used when the user needs a different focus for each eye. First the user focuses the eyepiece to equalize eyes, then he or she uses the central focus to bring the object into view.
- Zoom changes magnification and thus field of view. This adds weight because of additional lenses, but can be useful for multipurpose binoculars and telescopes.

PACKS

One ideally should have a professional at an outdoor store assist in fitting any pack for correct size, type, body habitus, and specific application.

Lumbar Packs

Lumbar ("fanny") packs are small and work well for brief hikes in clear weather or for carrying emergency supplies when skiing in a developed winter resort. They have capacity to hold food, water, an emergency kit, an extra windbreaker, a hat, and gloves.

Day Packs

Day packs, or rucksacks, are small packs that carry gear for a 1-day outing, usually a spare jacket, food, water, and emergency supplies. Day packs are usually around 1000 in^3 (16,400 cm^3). Usually, the shoulder straps and belt are unpadded or have minimum padding. They are often light and compact, and usually do not have any frame.

Backpacks

The two types of backpacks are internal- and external-frame packs. Internal-frame packs are the current standard and an excellent choice for many situations. Aluminum or plastic stays are sewn into pockets inside the pack to add support. Internal-frame packs are more flexible and narrower than rigid external-frame packs, offer much more space than does a day pack, and have thicker padding on the waistbelt and shoulder straps.

Compared to external-frame models, internal-frame packs, which are more flexible and narrower, are easier to maneuver when hiking in tight, narrow spaces such as caves, canyons, and thick forests.

Because they stay tight against the body, they are warmer and allow better balance and maneuverability when hiking over rough terrain, skiing, or climbing. Compression straps allow one to cinch the load; this keeps the pack tighter and more stable, especially when it is partially full.

Top-loading packs have one large compartment that opens at the top. Usually these have a drawstring closure and a lid that clips over the top. These packs can be loaded with much gear, sit well on the torso, and are easy to unload with the pack upright. Panel-loading packs have a large zipper on the front of the pack. These allow easy access to the entire contents of the pack.

External-frame packs have a stiff aluminum tube frame on the outside of the pack. The frame holds the straps and waistbelt. The nylon pack is attached to the frame. External-frame packs are more comfortable when carrying heavy loads, in that they distribute the weight efficiently between the shoulder straps and waistbelt. Some people can carry more than 50% of body weight with an external-frame pack. External-frame packs can be cooler than internal-frame packs, because the pack sits away from the wearer's back, and they can be easier to pack, because they usually have multiple compartments. However, they are bulkier, so they are not a good choice for tight spaces, aerobic activity, climbing, airline travel, or off-trail hiking.

Size is a consideration. For overnight trips, 3000 to 6000 in^3 (49,000 to 98,000 cm^3, or 49 to 98 L) may be needed. To carry medical, rescue, research, or work equipment in addition to personal gear, one obviously needs a large pack.

If a pack is routinely used half full, the pack itself will weigh more than necessary and the fit will not be optimal, because packs are designed to be used full or nearly so. One can always add pockets to packs if they are not quite large enough, or if one wishes to make access to certain equipment easier. However, pockets tend to flop around on packs and are a less efficient way to carry heavy items. Alternatively, some gear like sleeping pads and spare clothing can be strapped on the outside of the pack.

Suspension systems vary from a simple webbing belt and shoulder straps to well-padded systems that wrap around the torso and are specific for gender, body shape, and height. Women should use packs designed for their anatomy. For full loads, choose a system that comfortably rests the weight on the hips but snugly pulls the load toward the back. For larger packs, choose thick padding on the hips and shoulders, and a fit that easily adjusts while walking to take the pressure off hot spots. A padded hip belt and lumbar pad should fit snugly with ample room to tighten the belt when in the field. For small day packs, choose a lightweight and comfortable suspension system that does not have more padding than needed for the relatively light load (Fig. 83-1).

Load-adjusting straps are usually necessary for packs of 3000 in^3 (49,000 cm^3 [49 L]) or more. A sternum strap brings the shoulder straps into position over the collarbones. Load-lifting straps on top of the shoulder strap bring the top of the pack closer to the shoulders. Belt-stabilizer straps at the waist

Figure 83-1. Backpack suspension system. (Redrawn from Harvey M: The National Outdoor Leadership School's Wilderness Guide: The Classic Handbook. New York: Fireside, 1999.)

bring the bottom of the pack closer to the hips. It is essential that these be adjustable in the field, to shift the ride of the pack depending on how it is loaded.

Trying on a pack before purchase is essential. Load the pack with as much weight as intended for the average trip, hoist the pack up and over obstacles, and climb up and down stairs. A professional fitter can assist with size and selection and adjust the straps and harness system. Certain adjustments, such as shoulder strap height, can be fitted once and then not changed much, but other features, such as side load adjustment straps, will be adjusted at the trailhead and while walking.

Accessories for backpacks should be considered with care. Sometimes accessories add essential space, but they also add weight, extra stitching that can fail, and bulk. Some packs come with integral map pockets, hydration bag holders, water bottle pockets, a shovel pocket, a crampon patch, ice ax straps, a removable lid that converts to a lumbar pack, and an extension cuff to extend the volume on a top-loading pack. Many packs have lash patches that allow one to strap on additional items like sleeping pads, tents, or climbing gear.

Packs for special use have added features:

- Climbers and mountaineers require ice ax loops, crampon patches, and wand pockets.
- Backpackers need water bottle pockets, hydration bladder pouch, and lash patches for lashing on tents, sleeping bags, and tents.
- Mountain bike backpacks are usually compact, around 250 in^3 (4100 cm^3), so they fit between the shoulders when biking and do not impede the rider. They are designed to carry a hydration bladder, food, emergency kit, and bike repair tools.
- Mountain rescuers and ski patrollers may need internal pockets to organize gear like snow shovels, avalanche probes, and first aid supplies.
- Backcountry skiers and snowboarders need straps to hold their skis or snowboard on the outside of the pack when hiking.
- Photographers need compartments lined with foam to protect equipment.
- International travelers can use compartments that stow the suspension system for airlines, and can use wheels for easy transport.

Packing a backpack efficiently is an art form. Nylon stuff sacks can be used to compartmentalize gear. Mesh bags or plastic zip freezer bags work well for small items. Pack heavy objects low and forward to improve balance. Balance the pack so that left and right are equal. Stuff sleeping bags and bulky clothing into compression stuff sacks to reduce volume. Minimize the amount of gear lashed on the outside and, if tied outside the pack, make sure everything is secured tightly. In wet climates, consider waterproofing the pack by lining it with a tough garbage bag, using a pack rain cover, or pack gear in dry bags, such as those for kayakers and rafters.

DUFFELS, STUFF SACKS, AND DRYBAGS

Duffels should be large enough to hold equipment, clothing, and other gear, but they should not be too large that they are cumbersome to transport. All duffels for wilderness travel should be constructed with heavy-duty zippers and stitching. It helps if they have heavy-duty shoulder straps or straps that convert to a backpack. Wheels are a great benefit for transporting bags in airports. They are not much use in the wilderness and add weight. Choose a size that can be reasonably carried and used. This may be as small as 1500 in^3 (27,600 cm^3) or as large as 10,000 in^3 (164,000 cm^3). Duffels that are larger than 7000 in^3 (114,800 cm^3) are usually too heavy to be carried for more than a short distance. It is often better to use two smaller packs when packing items like ski, camping, and climbing gear.

For baggage carried in commercial airline cabins ("carry-on"), the height, weight, and depth should be no more than 45 inches (114 cm). Most airlines restrict checked bags to 70 lb (32 kg), but newer restrictions have been imposed on some flights down to 40 lb (18.1 kg). For airline travel, bags can be locked with luggage padlocks or plastic zip ties. However, in many countries, they will need to be opened for inspection.

Box 83-6. Important Tips on Battery Selection and Use

Alkaline batteries are the standard. Usually, name brands tend to provide more useful hours than do budget brands. Alkaline batteries are shorter lasting and perform worse in cold environments compared to lithium batteries. Lithium batteries provide a constant voltage output until they fail, unlike alkaline batteries, which slowly decrease voltage output as they are drained.

Rechargeable batteries are an option for short outings. Disposable alkaline cells yield roughly double the light duration per weight of rechargeable batteries. Consider lithium ion batteries for all rechargeables, because they do not have "memory" and thus do not need to be deep-charged; they can be "topped off" without a full discharge. Older rechargeable batteries were nickel metal hydride types that needed to be frequently fully discharged, then recharged.

It is helpful to keep all electronics on one type of battery so you only need to carry one type of spare. For example, carry a headlamp, laryngoscope, otoscope, avalanche beacon, radio, and GPS unit that all take size AA batteries. Newer, lighter electronics may use AAA, rechargeable, or camera-size batteries.

Make sure to have a backup plan if batteries fail. For example, be prepared to navigate with a compass if a GPS unit stops working.

ELECTRONICS

Electronics are ubiquitous in the backcountry. Before purchasing, consider the following:

- Do they enhance the outdoor experience, improve the margin of safety, or increase emergency response capabilities?
- Are they easy to use? Some manuals are difficult to read and are complicated. Does one need the manual to remember how to operate or calibrate the device?
- Does the device still work after a week in the field? The device can be rendered useless due to exposure to cold weather or water, or from being dropped. One small slip and a global positioning system (GPS) unit can explode on a rock; a small water leak can cause it to malfunction.
- Are the batteries fresh and fully charged? See the important information about batteries in Box 83-6.

When considering carrying and using an electronic of any type, always have a contingency plan for emergencies. For example, carry a standard compass and altimeter to use when the GPS or wrist-top computer compass fails.

Common electronics are highlighted below.

- Global positioning system (GPS) units have become small, light, and easy to use. Still, they weigh much more than a map and compass, which should be carried anyway for backup. GPS units are not always reliable, especially when in a deep canyon where it is not possible to receive the signals of at least three satellites.
- Wrist-top computers are as valuable as they are versatile. They most often contain a compass, an altimeter, a stopwatch, a timer, multiple alarms, a barometer, and, the most recent addition, a heart rate monitor. They are fairly accurate, but more complicated to use than a standard non-electronic compass.
- Family radio station (FRS) radios are inexpensive and valuable, especially for outdoor families. These small UHF-frequency walkie-talkies are easy to use and great for communication within the effective range. Some ski patrols and park rangers monitor FRS channels. However, most FRS radios transmit at only 0.5 to 1 watt of power; thus, they only have a range of 1 to 2 miles in clear, line-of-sight terrain. Terrain and trees can block the signal.
- VHF radios are commonly used by marine travelers. They are also available for backcountry land travelers. They need to be registered with the Federal Communications Commission in the United States. These usually transmit at 5 watts, and thus have much longer range than FRS radios. Certain channels may be monitored by local law enforcement, park service, or forest service personnel.
- Personal locator beacons (PLBs) are used by mariners and aviators. Now widely available for all backcountry travelers, the unique number is registered with an international satellite locator system. If activated, the PLB broadcasts a signal with its identification number. The signal is relayed through a satellite to a ground station rescue center.
- Cell and satellite phones are common in the wilderness. Cell phones have made rescues much easier, by allowing quicker and more accurate responses. However, these phones cannot always connect with a cell tower or satellite, and they exhaust batteries quickly.
- Weather radio is a broadcast in the United States by the National Weather Service. The continuous broadcast operates on 162.40, 162.475, or 162.55 MHz VHF frequency, depending on the area, and gives detailed weather information, depending on the region, on a continuous, round-the-clock basis that is updated daily or, in some cases, hourly.

OVERNIGHT GEAR

For overnight wilderness travel, cooking and sleeping equipment are necessary. Some backpackers and climbers prefer minimal equipment. Long-distance Pacific Crest Trail hikers have been known to take this to the extreme, for instance, by cutting down and drilling holes in the handles of their toothbrushes or hiking for 6 months in tennis shoes.

Stoves

Camp stoves are valuable to disinfect water, improve nutrition and hydration, and help keep up spirits in foul weather by allowing for hot food and drink. Camp stoves may be needed to melt drinking water from snow.

The simplest stove is a solid-fuel stove. These burn wood, natural charcoal, or other burnable materials. They are light and compact because fuel is not carried but usually gathered from the wilds. They are great for emergency or limited use, especially when abundant fuel is available. However, it is difficult to control the heat and flame; thus, heating is unpredictable. They are best for day trips during which one is not reliant upon the stove for survival.

Alcohol-fuel stoves are usually aluminum with brass burners. Liquid alcohol is poured into the burner and lit. For the most part, denatured alcohol is used. Also called methylated spirits or solvent alcohol, denatured alcohol is a mixture of ethyl and methyl alcohols. It is often available in hardware stores. Otherwise, isopropyl alcohol, grain alcohol (pure ethanol), or high-proof food-grade drinking alcohol can be used.

Alcohol stoves are lightweight, compact, and simple with no moving parts or complicated valves. They burn quietly. As with solid-fuel stoves, it can be difficult to control the flame and it can take a long time to boil water. For example, the Trangia takes 7 to 10 minutes to boil 0.65 liter (1.37 pints) of water. These stoves sometimes are preferred by "ultralight" campers who prefer to avoid the more complex fitting of pressurized stoves.

Canister stoves burn fuel from a sealed pressurized canister containing either butane or a butane-propane blend. These stoves are easy to ignite and burn hot. They usually boil 1 liter (1 qt) of water in 3 to 4 minutes, and thus are much more efficient than solid-fuel or alcohol stoves. The main benefits are that they are clean-burning, do not leave soot like other stove types, and rarely clog. However, the canisters are not refillable and, in most areas, not recyclable. These stoves are starting to replace white gas stoves as the standard for backcountry travel.

White gas stoves require a purified form of gasoline. They require canisters that have to be filled and pressurized with a tiny pump before use. Sometimes they require priming with isopropyl or ethyl alcohol before lighting, especially in cold temperatures and high altitudes. They burn hot but can be messy, because the fuel sometimes gets splashed when the user is setting up the system. Also, they can clog and require frequent cleaning from carbon deposits. The gas is found in most camping stores, often sold as Coleman Fuel. White gas stoves usually come with a windscreen and heat reflector, to improve heat transfer, and a cleaning kit to remove carbon clogs. The fuel is stored in refillable aluminum bottles of 0.3-, 0.6-, or 1-liter sizes. These are slightly less efficient than canister stoves, and boil 1 liter (1 qt) of water in 4 to 5 minutes.

Multifuel stoves are variations of the white gas stove with a more versatile jet that accepts a variety of petroleum-based fuels, many of which are less pure than white gas. In addition to white gas, they may burn kerosene, unleaded or leaded auto fuel, or jet fuel. These are useful for international travelers who might not have access to white gas or pressurized canisters. However, the jets clog frequently with carbon soot, especially with poor-quality fuel, and they do not burn as hot when using fuel with impurities. They should be used primarily with white gas; alternate fuels should be used in emergencies or on a trip in which standard white gas is unavailable.

Dual-fitting stoves, such as a new stove by Primus, the Omni Fuel, has fittings for both liquid fuel, like the multifuel stoves, as well as pressurized canisters with butane-propane mix. Although expensive and heavier with fittings for both canister and liquid fuels, this stove burns any available fuel. This may be the best choice for expeditions abroad.

Two-burner folding propane camp stoves usually burn propane or white gas. They work well when camping with vehicle support, pack animals, or boats. A two-burner stove is superb for cooking large meals for groups and when car camping. Although propane canisters are heavier than white gas bottles, propane burns hot and efficiently.

Accessories

Accessories are available for certain stoves, depending on the type and model:

- Flame adjuster: Many canister and liquid fuel stoves allow flames to be adjusted from full power to simmer.
- Auto lighter: A small hand-operated piezoelectric sparker ignites the flame. It can be unreliable in wind or rain, so one should always carry matches or a lighter for backup.
- Cleaning tool: Some stoves are maintenance-free. However, many liquid fuel stoves become clogged with soot. The cleaning tool unclogs the valve. A safety pin can suffice if the tool is lost.
- Heat reflector: Some stoves, especially multifuel types, include a heat reflector to channel heat from the flame directly to the pot. For other stoves, like most pressurized canister models, this is not recommended, because it makes the stove too hot and can damage valves and seals.
- Windscreen: For some stoves, this improves transfer of heat to pot and minimizes wind blowing away flame and heat. Again, pressurized canister stoves and other models burn hot and thus windscreens are often not recommended by the manufacturers. Many stoves have built-in windscreens to protect the flame.
- A case to protect the stove in a pack is recommended.

It is important to be able to disconnect fuel from the stove for careful storage. Folding or collapsing the stove makes storage easier, but the stove should be simple to reassemble. Airline regulations prohibit travel with fuel. Box 83-3 contains information on stove safety.

Cook Sets

For most 1- or 2-night trips with one or two campers, a single 1-liter (1 qt) pot is usually adequate for boiling water for morning drinks and melting snow for water. For more people, consider carrying two pots or a larger 2- or 3-liter (2 or 3 qt) pot. Nesting pots that fit inside each other pack well and offer more versatile sizes, such as 0.5, 1, and 2 liters (0.5, 1, and 2 qt). Lids shorten cooking time and protect cooking food from debris. A pot handle is essential to lift hot pots from the burner.

Cookware is constructed from aluminum, stainless steel, or titanium. Aluminum is light, inexpensive, and conducts heat well. However, it is not as durable as other materials and can dent easily. Titanium is extremely light and strong, but is expensive. It does not conduct heat as well as other materials. Stainless steel is the heaviest and most durable. It does not dent as easily as aluminum and is scratch resistant.

Nonstick coatings are cleaned easily when adequate water and soap are available. However, sand, dirt, and utensils wear down these coatings quickly. Buy an inexpensive pan and replace it often.

Cooking utensils may include pot grips, serving spoon, ladle for dishing out hot water, and spatula. For personal utensils, ultralight campers carry only a spoon for basic trips. Plastic utensils are the norm because they are durable, light, and easy to clean. A small bowl and covered mug usually are adequate for personal dishes.

Dish soap and a small sponge are usually adequate for cleaning cook sets and utensils. This should be biodegradable camp soap sold in most outdoor and camping supply stores.

Personal Toiletries

Personal hygiene is extremely important in wilderness travel. A typical list of toiletries includes the following:

- Toothbrush, collapsible
- Toothpaste
- Dental floss
- Liquid or bar soap
- Shampoo
- All-purpose camp soap (can be used for dishes, hair, and skin)
- Alcohol-based gel hand cleaner
- Lotion
- Toilet paper
- Feminine hygiene products
- Razor and shaving cream
- Compact hair brush or comb

Sleeping Bags

Products made of better down, softer synthetics, and lighter construction make buying a new sleeping bag tempting. Box 83-7 suggests points of comparison.

Material

Down settles closely around the skin and is thus warmer and cozier than synthetic fillers. Down is a more efficient insulator for the same weight of synthetic fill and thus lighter for the same degree of insulation. It compresses into a smaller stuff sack and is more durable in the long run. Down is best for alpine trips where cold temperatures are encountered; down is also a good choice for trips that mandate the lightest and most compact gear, such as mountain climbing or international travel.

For down, 800-fill-power is warmer than 600-fill-power. In other words, for the same warmth rating, the 800-fill-power bag is lighter.

Synthetic materials are easier to clean and store (down should not be stored compressed in a stuff sack). Synthetics do not absorb as much water or sweat as down and are still quite warm when they become damp. Therefore, synthetic bags are preferred for outings in which the bag may get wet, such as boating or rafting, or in climates in which rain may be anticipated. They are also great for persons who frequently encounter dirty conditions, or for children, because synthetics are easy to launder.

Polyester insulators include polyester, polypropylene, blends, and proprietary fabrics such as Polarguard, Hollofil, and Primaloft. These are extruded petroleum-based polymers spun into sheets. Some are more compressible than others because they are spun into shorter strands and more compact sheets. Some are warmer because the fibers are hollow. See Chapter 82 for more information on materials.

Temperature

Temperature rating varies between manufacturers, quality of down or synthetic filler, construction, design, and shell material. Choose a lightweight 30° to 45°F (–1.1° to 7.2°C) rated bag for summer fair weather, travel to the tropics, or an emergency bag for search and rescue. For three-season below-timberline use, select a sleeping bag rated at around 20° to 15°F (–6.7° to –9.4°C). Choose a warmer bag, rated 0°F (–17.8°C), if taking fewer clothes or if you tend to get colder than other people when sleeping (some manufacturers refer to the latter concept as "sleeping cold"). For four-season above-timberline use, most bags are rated at 5° to –20°F (–15° to –29°C). Mountain expedition bags are usually rated at or below –20°F (–29°C).

Comfort rating in degrees is only a rough guide. Metabolic rate, type of sleeping pad (described later), type of tent or shelter (described later), amount and type of clothing worn to bed, design and construction of sleeping bag, and nutrition and hydration status affect the comfort of a sleeper.

Winter and mountain expedition bags usually have 6 to 9 inches of loft, midweight bags may have 4 to 6 inches of loft, and those for fair weather and summer use may have 2 inches of loft.

Some sleeping bags may be constructed from water-resistant laminates such as Gore-Tex. These improve weather resistance to keep the insulation dry, but often they do not breathe well; thus, a person may perspire profusely in the bag. Uncoated nylon, the typical shell material for sleeping bags, breathes much better but is not waterproof. In this case, one typically would use a tent or bivouac sack.

Mummy-shaped bags are the only reasonable choice for wilderness travel because they insulate efficiently. Rectangular bags are bulky and waste materials around the legs and feet. However, because they allow more space, they can be more comfortable for someone who changes positions frequently during sleep. Most bags are around 6 feet (1.8 m) long. However, a few models are designed for taller people.

For a more versatile system, consider a convertible sleeping bag with an accessory blanket that zips on in winter and off in summer. One can add warmth to a sleeping bag in other ways (Box 83-8). A draft tube is an insulated tube covering the zipper,

Box 83-7. Down Versus Synthetic Sleeping Bags, Compared for Equal Weight Bags

DOWN
Warmer
More compressible
Softer
Longer lasting

SYNTHETIC
Easy to clean and store
Retains some warmth when wet
Dries quicker
Less expensive initial cost
Less likely to be allergenic

Box 83-8. Tips to Stay Warmer at Night

- Use a liner of silk or fleece.
- Wear more clothes to bed, especially wool or synthetic long underwear.
- Wear a hat to bed.
- Use a thicker pad.
- Use a bivouac sack or outer bag.
- Consider buying a warmer tent.
- Eat a snack or high-calorie beverage before going to bed to provide additional calories for generating heat at night.
- Consider a water-resistant shell material for the sleeping bag.

preventing wind from entering the bag and heat from escaping. A shoulder collar to seal in warmth, and a hood to cover the head and most of the face can improve warmth. Both should have drawstrings and should be able to cinch loosely to fully cover the user, all but the nose, mouth, and eyes.

Sleeping Pads

In addition to a quality bag, a sleeping pad is very important for warmth. The thicker the pad, the more one is insulated from the ground, and the warmer one sleeps. A snow camper should use a 2-inch-thick pad. A summer camper may be able to stay warm and comfortable with a pad ranging from $^3/_4$ inch (2 cm), for lightweight campers, to 1.5 inches (3.8 cm). In addition to warmth, thicker pads provide more comfort, but are bulkier and heavier.

Closed-cell foam pads are the least expensive, lightest, and most durable. The dense foam is filled with sealed air cells. If strapped outside of a backpack, it can take a beating and still function well. In addition, closed-cell foam does not soak up water. It provides superior insulation against the cold ground but not as much cushioning or softness as other pads. These are favored by alpinists, climbers, and adventure sports participants.

Open-cell foam is similar to a sponge with tiny open air cells. These are comfortable, light, and compressible, but are not as durable and do not insulate as well as closed-cell pads.

Two-inch-thick, dual-density pads provide a blend of warmth, durability, and comfort. These usually have thick, abrasion-resistant closed-cell foam on one side and lighter, more compressible open-cell foam for comfort on the other.

Inflatable pads are preferred by many backpackers because they can be more comfortable and pack into smaller bundles. These are nylon-encased open-cell foam with an air valve. The nylon shell protects the foam from absorbing water and contains air to improve insulation and comfort. Although manufacturers sometimes call these self-inflating, they usually require a camper to blow up the pad. Inflatable pads are heavier than and not as durable as closed-cell foam pads, but they can be just as comfortable as open-cell pads. In addition, one can adjust the amount of air in the pad for comfort. With a small puncture, an inflatable can be pancake-flat by morning, so one should always carry a patch kit, which usually includes a strip of adhesive-backed shell material and/or some nonadhesive material and rubber cement. A bike repair patch kit usually works, as does duct tape, if nothing else is available.

Some pads have accessories that may improve comfort but add weight. Pads may have built-in pillows, separate air chambers that can be adjusted for comfort, and non-slip surfaces so the sleeping bag does not slide off the pad during the night. To save weight, consider a mummy-shaped pad that matches the footprint of the sleeping bag or use a $^3/_4$-length pad in which the feet and forelegs hang off the pad.

Shelters

In addition to blocking out rain, snow, and cold, shelters block out heat and sun in the desert, insects, small animals, and dirt. They provide privacy in crowded areas and a place to store or operate equipment.

Construction and Design

Most tents used by wilderness professionals are freestanding, in that they will stand up without guy lines or stakes. The exceptions are tents or tarps used by ultralight backpackers or cyclists, where space and weight are at a premium. Tents that are not freestanding are lighter because they do not need as many poles in the design. However, because they require guy lines, they take longer to set up and are not quite as sturdy in wind and rain.

Almost all shelters are constructed of nylon. *Nylon* is a generic term for petroleum-based polyamide fabrics made in numerous weaves and thicknesses. It is light, strong, and resistant to abrasion; it is easy to wash, has low absorbency, and is easy to stow. Most tents use thin-strand ripstop, an extremely lightweight nylon woven in square grids that is resistant to tears, or taffeta, a light, soft nylon. Some tents and almost all rain flies are made more waterproof and windproof by using a urethane coating on one side of the nylon.

Poles are usually aluminum, or in some cases, fiberglass or carbon. Aluminum is stronger and less expensive.

Most tents are double-walled and have a separate rain fly. This double-wall system allows the tent to be used without the rain fly in mild weather. The rain fly, usually waterproof urethane-coated nylon, sheds rain, enhances protection from the wind, and provides warmth because it traps a layer of air. The double-wall system allows the tent to breathe through the tent and still accomplish weatherproofing with the second layer.

Certain three- or four-season tents are made with specialized fabrics in a single-wall construction without a separate rain fly. These do not breathe as well and are not as warm, but they are compact and light. They allow weatherproofing without the bulk and weight of two-layer tents. These are favored by ultralight mountaineers, backpackers, mountain bikers, and cyclists when foul weather may be encountered, but weight and space are at a premium and simplicity of setup is essential.

Shape and Size

Most wilderness tents are dome-shaped because they are lower-profile, stronger in the wind and rain, easier to set up, lighter, and less bulky when carried than are square or triangular tents. Lightweight tents, such as those used by ultralight backpackers and cyclists, are sometimes tunnel-shaped or hoop tents. They are set up with one or two hoops with guy lines and are usually not freestanding. A-frame tents are an older style with a rectangular floor and a center pole at each end. These tents are not freestanding but are very light because they have the fewest poles. Large 12- × 12-foot (4- × 4-meter) or greater camping tents with external poles are good for car-supported camping and expeditions, but they are too bulky and heavy to carry otherwise. Specialty tents, like plastic- and canvas-walled military or research tents, are used in extreme climates.

Tents are measured in square feet or the number of persons that will reasonably fit without backpacks or gear. A typical two- to three-person tent is around 25 to 45 ft^2 (2.3 to 4.2 m^2). A typical four- to five-person shelter can be around 50 to 70 ft^2 (4.7 to 6.5 m^2). Six- to eight-person shelters can be as large as 120 ft^2 (11.2 m^2).

Bivouac Sack

The basic shelter is a bivouac sack. This one-person overbag provides protection from the elements in a very small package, just large enough to encase a sleeping bag. Some have an aluminum pole that bends to form a hoop to keep the bivouac sac off the face when sleeping. Others are simple nylon bags that slide over the sleeping bag. These provide warmth, and with

laminated waterproof, windproof coatings, can keep a person fairly dry. They are especially useful for 1- or 2-day outings, for trips that require the least amount of weight and bulk, and for mild-weather adventures.

Tarps

Tarps and wings are floorless shelters that provide sun and rain protection, but little protection from the wind, insects, or cold damp ground. These minimum shelters are great for ultralight backpacking, cycling in clear, warm weather, and for an emergency shelter. Some wings are freestanding, while others need to be tied to trees or vehicles with guy lines.

Tents

Summer tents are usually all-mesh bug shelters or partial-mesh with solid nylon panels on the apex to block the sun and provide shade.

Three-season tents are designed to withstand most weather conditions that occur outside of winter. They work well for all applications except mountaineering or severe wind and rain. Full rain flies that extend to the ground are the most wind- and waterproof. Some less expensive tents have a rain fly that only covers the top third of the tent. Three-season tent sizes range from one-person shelters to eight-person family tents.

Four-season tents are designed for mountain and winter use. Most have a full rain fly with a vestibule, and they minimize the use of mesh panels to conserve warmth. They are usually lower-profile than three-season tents to minimize snow and wind effects and are also usually constructed with four to six poles to maximize stability and handle loads of snow. The main disadvantage of a four-season tent is that it is bulky and heavy.

Accessories

Many tents use accessories to improve function and versatility:

- Gear lofts and mesh pouches store clothing and equipment off the tent floor.
- Internal lash points are used to rig clotheslines for drying wet clothing or to hang a flashlight.
- A footprint tarp protects the floor from rocks and dirt on the ground. This thin cloth keeps the tent dry as well.
- Mesh panels, mesh doors, and skylight panels work well for star gazing and ventilation, and keep insects outside the tent.
- Large loops for stakes and guy lines keep the tent secure in wind and rain. The guy lines should be adjustable.
- Tent stakes help hold the tent on the ground, sometimes necessary for tents that are not freestanding. These can be thin aluminum nails or hooks for dry, hard ground. In soft dirt, wide or large-diameter plastic stakes hold much better. Long, wide stakes reach down to more compressed sand or snow.
- Many tents have a vestibule on the rain fly; this provides additional dry storage outside of the tent.
- Many tents have more durable, thicker material on the floor to protect from dirt and moisture.
- A few tents are "convertible," like the sleeping bags mentioned earlier. Weight and bulk can be adjusted depending on the weather conditions, length of outing, and terrain. For example, a four-season tent can be set up without one or two poles, or a vestibule can be detached. Some tents offer moderate wind and rain protection without the rain fly. Others allow the rain fly to be set up alone, to provide a simple floorless shelter.

CARE OF OUTDOOR EQUIPMENT

Gear needs to be cleaned and inspected regularly. Replace worn or broken parts before beginning a trip. Carry spare parts and repair material. Make sure equipment, especially tents and sleeping bags, are dried thoroughly after use. Knives rust and tents mildew when stored damp.

Follow the manufacturer's recommendations on washing and cleaning. Some hardware can be cleaned in warm soapy water. However, other equipment should only be rinsed with warm water. All equipment works better when it is clean and well lubricated, when appropriate.

Cooking utensils and pots should be washed with hot soapy water. If hot soapy water is unavailable, an alcohol-based cleanser may be used. Backcountry cooking is not the place to skimp on cleaning or hygiene.

SELECTED RESOURCES

Adventure Medical Kits. Available at www.adventuremedicalkits.com.
Backcountry.com. Available at www.backcountry.com.
Backpacker Magazine. Available at www.backpacker.com.
Cox SM, Fulsaas K: Clothing and equipment. In Mountaineering: The Freedom of the Hills, 7th ed. Seattle, The Mountaineers Books, 2003, pp 18–42.
Davenport GJ: Personal Protection. In Wilderness Survival. Mechanicsburg, PA, Stackpole, 1998, pp 10–49.
Ganci D: Desert dress and paraphernalia plan. In Desert Hiking. Berkeley, CA, Wilderness Press, 1993, p. 39–72.
Handwarmers.com. Available at www.handwarmers.com.
Hotronics. Available at www.hotronics.com.
Lanza M: Clothing and gear. In Winter Hiking and Camping. Seattle, The Mountaineers Books, 2003, pp 27–62.
Leatherman. Available at www.leatherman.com.
Petzl Equipment. Available at http://en.petzl.com/petzl/accueil.
Johnston G: The 10 essentials: A revised list for emergency wilderness survival. Seattle Post-Intellingencer. Thursday, March 17, 2005.
Townsend C: Clothing for the snow. In Wilderness Skiing and Winter Camping. Camden, ME, Ragged Mountain Press, 1994, pp 160–184.
Trangia Stoves. Available at www.trangia.se.
Van Tilburg CS: Equipment. In Canyoneering: Beginning to Advanced Techniques. Seattle, The Mountaineers Books, 2000, pp 33–53.
Van Tilburg CS: Backcountry Snowboarding. Seattle, The Mountaineers Books, 1998.
Useful Knives. Available at www.usefulknives.com.

84 Ropes and Knot Tying

Loui H. McCurley and Steve E. Hudson

Ropes, and the knots that hold them in place, are considered by many to be a staple of the avid outdoors person. Ropes are commonly used for lashing down equipment, setting up tents, and stowing food in trees to protect it from bears, as well as in wilderness adventure activities like climbing and caving. In an emergency, ropes can be a lifesaving resource for the person who knows how to use them properly.

A rope, simply stated, is a flexible cord of intertwined fiber. Most modern-day ropes are made of human-made materials, such as nylon, polyester, polypropylene, or a derivative of these. Natural fiber ropes have lost favor with most informed users because of their tendency to mold, mildew, degrade under ultraviolet (UV) light, and lose significant strength over time.

ROPE FOR LIFE SAFETY

Being able to assess the characteristics of a rope in light of its intended use is a critical skill for the outdoors person and rescuer. Not every rope is appropriate for every purpose—a point that becomes increasingly important as one considers life-safety ropes versus the types of ropes used to rig camp.

Ropes used in non–life-safety applications are commonly known as *commodity ropes*. They can be found at hardware stores, discount stores, and probably in the garage. These ropes are fine for use in noncritical applications, where failure of the rope is unlikely and would have relatively minor consequence.

No rope should ever be used to support a human life unless it has been specifically built for that purpose. Stories abound of towropes being used (fatally) as climbing ropes, utility ropes coming apart when used as hand lines, and natural fiber ropes rotting away to nothing. One good way to be certain that a rope is designed for life-safety purposes is to check whether it is certified to any life-safety standards, such as the Union of International Alpine Associations (UIAA), Cordage Institute (CI), American Society for Testing and Materials (ASTM), or National Fire Protection Association (NFPA).

Various types of life-safety rope are made for specific purposes and applications. Rock climbers have different priorities than do mountaineers, who in turn have different priorities from cavers, who prioritize differently yet from rescue personnel. Interestingly, the requirements of each of these categories of life-safety rope users tend to revolve around similar performance considerations, but different users want different combinations of these considerations. Important performance considerations for life-safety rope, for whatever purpose, can be categorized as follows:

- Strength
- Impact force transmitted during a fall
- Number of falls held
- Elongation
- Diameter
- Abrasion resistance
- Compatibility with other equipment
- Hand

Life-safety rope users generally select which rope to use based on whether they want a rope that stretches a little, a lot, or somewhere in between. Life-safety rope is generally classified into three types: dynamic, static, and low stretch. Each of these three types of rope is tested to different standards and criteria.

Although there are no cookie-cutter solutions to rope selections, some rough generalizations can be made. A climber who could potentially take a significant fall on a rope will opt for a higher stretch rope for its ability to absorb the forces of a fall—in other words, a dynamic rope. A rescuer who wants to lower a load without a lot of excess elongation may choose a rope with as little stretch as possible in order to more effectively manage the load—a static rope. The user who wants a limited amount of stretch, but would like at least some force absorption capability, may opt for the third type of rope, low stretch.

It is important to understand the job at hand, as well as performance characteristics of ropes, in order to select the right rope. Climbing and rope rescue techniques are not the subject of this text, but other texts are available that give guidelines on appropriate selection, care, and maintenance of life-safety ropes for various applications. Such activities should be undertaken only by experienced personnel. With appropriate knowledge of the intended use of a rope, performance characteristics can be more accurately evaluated.

Strength

Strength requirements for life-safety rope are most important to people who are using the rope in raising, lowering, ascending, or rappelling. Although most rope users never come anywhere close to pushing the strength limits of their equipment, a necessary margin of safety should be factored into any system. In some extreme environments and uses, heavy loads and complex systems combined with safety margin requirements can create a challenge. Acceptable safety factors range from 4:1 to 15:1. These ratios are most appropriately applied to the completed system, not just the rope or other individual components. This requires not only a fair amount of sophistication on the part of the user, but also quite a high starting strength in order to

compensate for strength reductions as the equipment is integrated into a system. According to CI specifications, static and low-stretch rope must meet the following minimum strength requirements:

Minimum Breaking Strength (MBS) by Size (Diameter) noted in pounds-force (lbf) and kilonewtons (kN)

9/32″	(7 mm)	2,200 lbf	(9.8 kN)
5/16″	(8 mm)	2,875 lbf	(12.8 kN)
3/8″	(10 mm)	4,500 lbf	(20.0 kN)
7/16″	(11 mm)	6,000 lbf	(26.7 kN)
1/2″	(12.5 mm)	9,000 lbf	(40.0 kN)
5/8″	(16 mm)	12,500 lbf	(55.6 kN)

Impact Force

Impact force is an important consideration, especially for sport climbers who are climbing above their protection, thereby exposing themselves to a fall with significant impact potential. Dynamic ropes are most commonly used for such applications. They are designed to absorb energy during a fall so that the force is not transmitted to the climber or to anchorages. Dynamic ropes are tested to verify their performance using an 80-kg mass and are certified to either UIAA or European EN standards. In these tests, the 80-kg (176-lb) mass is attached to a 2.5-meter (8.2 ft) rope, anchored over an edge, then raised 2.3 meters (7.5 ft) above the anchor. It is then dropped a total distance of 4.8 meters (15.7 ft), with the requirement that the resulting impact force be less than 12 kN (2698 lbf).

When it comes to static and low-stretch ropes, impact force is an important consideration, but impact force testing is not performed in the same way as on dynamic rope because static and low-stretch ropes are not intended to be used where significant impact may occur.

Number of Falls Held

Number of falls held applies only to dynamic climbing rope, on which falls are expected to be taken. In the test, the impact force test described previously is repeated until the rope breaks. In order to qualify for UIAA or EEN certification, a rope is required to sustain a minimum of five falls. The maximum number of falls achieved becomes the *fall rating*.

Elongation

An important attribute of nylon and polyester ropes is an inherent ability to absorb force. Virtually any loading of a rope results in at least some impact force, which could potentially be damaging to equipment and systems if the rope did not have at least some stretch. Using high-quality nylon and polyester life-safety rope helps to protect against the effects of such loading. Although absorption of impact force generally translates into a high-stretch rope, elongation poses a practical concern where heavy loads are being raised, lowered, and positioned on a vertical plane. Ropes with too much elongation can require more effort to raise, "bounce" the load, and cause a stopped load to creep dangerously. For this reason, ropes with lower elongation are preferred for raising, lowering, and positioning heavy loads.

Diameter

Most life-safety ropes range in diameter from 7.5 to 13 mm (0.23 to 0.57 in). Accurate assessment and reporting of diameter are critical for life-safety ropes. Most of the auxiliary equipment designed for use with life-safety ropes works with very specific rope sizes. Friction, ability to be gripped, and weight are important considerations. Balance of the two factors is largely a matter of personal preference.

Abrasion Resistance

Life-safety ropes are built for abrasion resistance on rock and ice and in industrial settings. This translates to better protection against cutting and damage and greater security. Because ropes with the best abrasion resistance generally do not also boast the softest hand, experienced rope users are often identified by the fact that they prefer ropes with a tighter sheath weave and stiffer characteristics.

Compatibility with Other Equipment

Auxiliary equipment selected for use with life-safety rope should be selected specifically according to purpose and with consideration to the specific rope to be used. Rope construction is important with some devices, as is sheath material, flexibility (too much or too little), and even sheath slippage.

Hand

The hand of a rope refers to its flexibility and handling characteristics. A rope must be manageable and easy to work with, but these terms are really very subjective. An experienced user will have different priorities than an inexperienced user. Although a soft hand and flexibility are often preferred by inexperienced rope users, the ropes with the best abrasion resistance, the least sheath slippage, and the greatest efficiency in systems are usually those with a stiffer hand.

In addition to the technical considerations involved with the manufacture of life-safety rope, quality must be considered a key factor. Some user groups of life-safety rope now mandate that qualifying manufacturers meet specific quality assurance criteria, such as certification to an ISO quality standard.

LIFE-SAFETY ROPE CONSTRUCTION

Most life-safety ropes in the 21st century are of a kernmantle construction. The German word *kernmantle* means "core" (kern) and "sheath" (mantle). Kernmantle rope sheaths are braided around the core, and their design is crucial to the hand, knotability, and abrasion resistance of the rope. A tightly woven sheath will prove more durable than a loose one, but this feature must be finely balanced to maintain knotability. Other variables include fiber denier, number of strands in the braid, and angle of weave.

Materials

Before the development of synthetic fiber ropes, the standard for many years was rope made of natural fibers, such as manila. Natural fiber rope degrades in strength even when carefully stored, lacks the ability to absorb shock loads, lacks continuous fiber along the length of the rope, and has low strength compared with certain artificial fibers. For these reasons, natural fiber ropes are no longer considered appropriate for life-safety applications. Synthetic fibers, including polyolefin, aramids, ultra-high-modulus polyethylene (UHMPE), polyester, and nylon, are more commonly used in modern-day rope making.

Polyolefin
Polyolefin (polypropylene or polyethylene) fiber ropes are used when their flotation property is desired, such as in water rescue. They also have good resistance to most acids. However, polyolefin fibers rapidly degrade, especially under UV exposure. Because the fiber has low abrasion resistance, strength, life expectancy, and melting point, it is a poor choice for most life-safety applications.

Aramids
Aramids, commonly known by the trade name Kevlar, are extremely strong fibers and resist high temperatures. However, aramid is very susceptible to internal and external abrasion. Because it cannot absorb dynamic energy and breaks if bent too tightly (such as in a knot or rappel device), it is dangerous to use in life-safety rope applications.

UHMPE
UHMPE, more commonly known by trade names Spectra and Vectran, is a newer type of high-modulus lightweight fiber. It has better abrasion resistance than aramid but still has too little stretch to absorb dynamic energy. UHMPE fibers also have too low a melting point to be used safely with most rappelling equipment. In addition, they tend to be very slippery and do not hold knots well under high tension.

Polyester
Polyester fibers are used in many ropes. Dacron has a melting point of about 249°C (480°F), which is in the range of nylon 6.6 and above the melting point for nylon 6. Polyester fiber has a high tensile strength even when wet, has low elongation at break, and can be as effectively UV stabilized as nylon. These factors make polyester rope well suited for marine applications, such as for boat-rigging lines, and make it an interesting choice for life safety. However, polyester fiber has low dynamic energy absorption, which means that the fiber cannot handle shock loads or repeated loading as well as does nylon fiber.

Nylon
Nylon is the most common and most suitable fiber for general life-safety use. Nylon is about 10% stronger than polyester, but nylon fiber may lose as much as 10% to 15% of its strength when wet. However, it regains its strength when it dries. Nylon can handle about twice as much shock loading per pound as polyester when both are wet.

Nylon strongly resists most chemicals, but certain alkalis, acids, or bleaches can cause degradation, especially in high concentrations. For the high-angle technician, the source of most damaging acids is batteries, including lead acid and so-called sealed or dry-cell batteries. Users must scrupulously protect ropes from direct contact with batteries or exposure to acid fumes or residues that might be found in vehicle storage compartments, trunks, and garage floors. Industrial users should take special storage and handling precautions because of chemicals used in their rescue environments.

Rope Type
The core of a kernmantle rope primarily determines the elongation, force absorption, and strength properties of the rope. The terms dynamic, low stretch, and static, introduced earlier in this chapter, are technically misnomers, in that all the ropes are dynamic, at least to some degree. However, these are the industry-standard terms and are quite useful in relating the degree of elongation inherent in each type of rope.

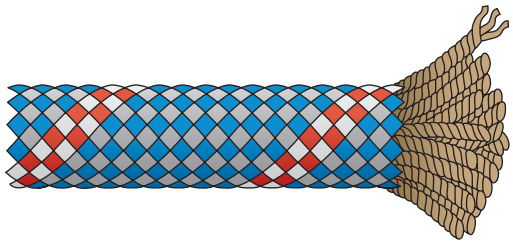

Figure 84-1. Dynamic kernmantle rope.

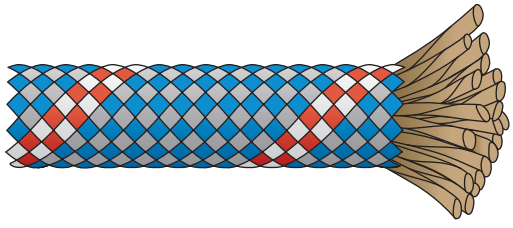

Figure 84-2. Static kernmantle rope.

Dynamic Kernmantle Rope
A well-designed dynamic rope that is intended to absorb the shock load of a fall will also be very stretchy: as much as 30% elongation at 10% of minimum breaking strength. Thus, a dynamic kernmantle rope would be very difficult to use effectively for positioning heavy loads (such as a rescue load), contending with changing loads (i.e., loading a patient midface on a rock wall), or rigging into a haul system (in which energy would be wasted with each pull because of the inherent elongation). It would also be very difficult to use effectively under high tension (as in a highline).

Dynamic ropes also tend to have a lower tensile strength than do static or low-stretch kernmantle ropes because of the same design characteristics that allow it to stretch. Furthermore, dynamic kernmantle designs are often softer and have a lower percentage of sheath than do static kernmantle ropes, making them more susceptible to abrasion and wear (Fig. 84-1).

Static Kernmantle Rope
A static kernmantle rope is designed to be very strong and to have minimal stretch: as little as 3% to 6% at 10% minimum breaking strength. For consistent strength, inner bundles run continuously and unbroken throughout the length of the rope, usually in a near-parallel manner to reduce stretch and spin (Fig. 84-2). This load-carrying core is protected from dirt, abrasion, and cutting by a tightly braided outer sheath. Static kernmantle ropes are ideal for lowering and raising heavy loads (as in rescue), work positioning, and fall protection. Static ropes should not be subjected to a fall factor greater than 0.25 unless additional force absorption provisions are made in the system.

Low-Stretch Kernmantle Rope

Low-stretch kernmantle ropes elongate 6% to 10% at 10% breaking strength, fulfilling the needs not met by highly dynamic climbing ropes or very stable static kernmantle designs. This is achieved using a different core construction and a better sheath–core relationship and typically gives such ropes a softer profile than static lines. However, these same characteristics decrease abrasion resistance and make it a less desirable rope for positioning. Low-stretch ropes are often used for belaying heavy loads, especially where low fall factor potential exists.

ROPE SELECTION CONSIDERATIONS

Selecting the right tool for the job means first and foremost understanding how a rope is affected by, and performs with, the way you use it. The following discussion provides some background for balancing your need against the available alternatives.

Fall Factors

Fall factors can be calculated by dividing the fall distance by the length of the rope between the load and the anchor or belay. Thus, a 3-foot (0.91 m) fall on a 9-foot (2.74 m) rope would be a fall factor of 0.3, a 3-foot fall on a 3-foot rope would be a fall factor of 1.0, and a 3-foot fall on a 100-foot (30.48 m) rope would be a fall factor of 0.03. This formula assumes that the fall takes place in free air without rope drag across the rock face or through intermediate equipment (Fig. 84-3).

The danger of a high fall factor on a low-stretch or static rope is not that the rope will break but that the high-impact forces may subject the person to great discomfort and possible injury or death. A static rope transfers more of a shock load to the anchors than does a dynamic rope, increasing damage potential there as well.

Another concern in any impact load situation is the effect such forces have on equipment such as belay devices, rope grabs, or ascenders. Some of these devices can damage a rope when subjected to relatively low-impact forces. The study of impact forces and ways that different pieces of equipment relate to different constructions of rope when subjected to such forces is fairly complicated. Two general rules of thumb are to (1) design and operate your rope systems to minimize the potential for high-impact forces and (2) exercise great caution in selecting belay methods.

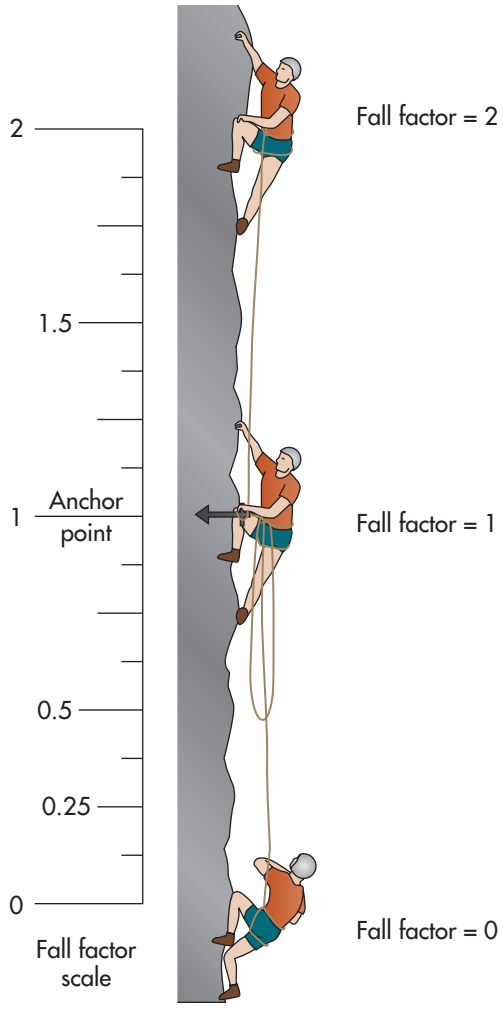

Figure 84-3. Fall factors.

Rope Diameter

Just as no single type of rope is appropriate for all activities, no single size fits all conditions. Purchasing the largest rope in the hope that it will fit all situations because it is so strong could lead to problems in working with the rope and the auxiliary equipment. Rope strength and elongation are actually the more critical determinations, and size considerations should follow.

Rope Strength

Rope strength is a fairly misunderstood topic. Strength of life-safety rope is usually referred to as *minimum breaking strength*. Unfortunately, reported numbers do not necessarily reflect numbers adequate for comparison. Variations in test methods, as well as in analysis of results, provide little more than marketing fodder that can create great confusion.

One way to report strength is as the rope's "ultimate, or maximum, breaking strength," which is the highest score of a given rope in a series of tests. An alternative is to list the "average breaking strength" of several tests. A more conservative method is to define breaking strength at a value 2 or 3 standard deviations below the average test result. Another method is to define minimum as not greater than 10% below the average. Often, figures for tensile or breaking strength are reported without any explanation as to whether they are average or minimum or whether some other measure was used. Simply stated, "breaking strength" might refer to any one of these reporting methods, or even to another.

To add to the confusion, a number of factors affect test results. The rate at which the pull is applied to the rope, temperature, humidity, diameter of the object to which the rope is attached, and other factors all have an effect on test results. Unless ropes are tested in exactly the same way, results cannot be meaningfully compared.

One common test method that has been adopted by several standards organizations is CI-1801 from the Cordage Institute. CI-1801 for life-safety rope is specific and gives a common baseline from which to attain results. This standard also calls for a very conservative reporting method, wherein minimum break-

ing strength is defined as 3 standard deviations below the mean of several break tests. This helps normalize the results. In addition to type of construction, the strength of a rope comes from the amount of nylon used to make it; similar rope constructions of the same diameter should have similar strengths if they have the same amount and quality of nylon.

Results from laboratory tests may differ greatly from rope performance in real-world applications, which cannot be consistently and accurately quantified. A knotted rope loaded over a building edge, for example, may not come close in strength to a rope loaded in a laboratory tensile test machine.

Safety Factors

It is clear that a rope should be stronger than the force of the intended load, but how much stronger? The ratio of a rope's strength to load is often called a *design factor*. If the strength of a rope is 2500 pounds (1134 kg) and the load is 500 pounds (227 kg), then the design factor is 5:1. The design factor takes into consideration only the new condition of an item as it is designed to perform. When translating a design factor into a system safety factor, factors such as age, wear and tear, dry or wet condition, and system rigging should be considered.

Because a rope system is only as strong as its weakest link, selecting a sufficiently strong rope is just the beginning. Generally, component design factors tend to be very high, often as high as 15:1. Once all those components are joined into a larger system, however, and the real world is factored in, the remaining system safety factor may be less than half that value.

The *system safety factor* is the ratio between the weak link in a system and the load to be applied. System safety factors also take into account the way that loads are applied to a system. For instance, redirectional pulleys, rigging angles, and mechanical advantage systems can all increase the forces within a system.

As a rule, the higher the probability of and consequences of failure, the higher the system safety factor should be.

Service Life

The service life of a rope cannot be determined in advance. How long a rope lasts depends on a large number of variables, including individual care, frequency of use, type of hardware used, speed of descent on rappels, exposure to abrasion, local climate, and type of loading. DuPont claims at least a 10-year shelf life for nylon, and rope tests have confirmed that an unused rope can last up to 10 years without significant strength loss. Used ropes degrade more quickly.

Any rope can fail after poor care or under extreme conditions, such as shock loading and sharp edges. A shock load of 0.25 fall factor for a static rope or 1.5 fall factor for a dynamic rope will likely cause internal, invisible damage to the rope, although damage may also be caused under lower fall factors. Regardless of how long it has been in service, you should immediately discard a rope when it becomes cut, when abrasion has caused significant wear to the sheath, after a hard shock load, when you suspect chemical contamination, or any time you are in doubt about the rope.

KNOTS IN LIFE-SAFETY ROPES

The medical professional encounters many knots in the course of his or her career. From the square knot in a stitched laceration to neck ties at administrative functions, practical and symbolic representations of knots abound.

In modern society, knots are used as a form of expression, in art, as mathematical structures, and for security purposes. Determination of "good" versus "bad" in the analysis of a knot lies solely in the knot's ability to achieve the purpose for which it was created. Therefore, you may find that several of the good and clever knots you have learned are useless when it comes to functioning in the wilderness.

Uses

Knots are applied in several ways in the wilderness environment. They are used to stake out tents or shelters, tie flies to fishing lines, create suspended "bear-proof" gear caches, and occasionally tie things together.

In wilderness medical evacuation, knots take on great significance. Many medical professionals find themselves working alongside rescue technicians in steep terrain where knots are used for the safety of rescue personnel and for patient evacuation. Improvisational medical techniques often involve knots. Knotted rope or fabric can be used to secure a splint, create a hasty patient-transport device, or provide secure shelter to people in precarious environmental situations.

How Knots Work

It has been said "if you cannot tie it right, at least tie it big"—and thus the "lotta knot" was born. In a lotta knot, the greater the mass of rope and the more twists and turns it takes in relation to itself, the higher the probability that the knot will hold. Although the theory is somewhat humorous, it is seldom effective.

Clearly, certain types of twists and bends hold better than others; the reason the lotta knot sometimes holds together is because the larger mass increases the odds of getting a bend to hold in the mix. There are many disadvantages to the lotta knot, however. It takes up a lot of rope and is very difficult to tension. The varying twists and turns are not conducive to tightening the knot or positioning it properly, and it is usually quite difficult to untie. There is also the unpredictability factor; often the lotta knot fails to perform altogether.

The internal friction of a knot is essentially what holds it together. This friction can be attained by the rope taking twists and turns around itself or from friction against another object, such as a capstan or another rope.

The serious professional should take knot tying seriously because in a wilderness rescue situation, lives may depend on this skill. One should be able to select the correct knot without hesitation, tie knots correctly the first time, and be able to tie knots with gloved hands, on muddy or icy rope, in the dark, and under stress. Finally, one should be able to determine by looking at a knot whether it is tied correctly.

Rope and Knot Terminology

The *working end* of the rope is the section used to tie or rig the knot.

The *standing part* (or end) of the rope is the section not actively used to form the knot or rigging.

The *running end* (or simply the *end*) is the free end of the rope.

A *line* is a rope in use. For example, a rope used to rappel is a rappel line.

A *bight* of rope is formed when the rope takes a U-turn on itself so that the running end and standing end run parallel to each another. The U portion, where the rope bends, is referred to as the bight.

A *loop* of rope is made by crossing a portion of the standing end over (or under) the running end. Note that a loop closes, as compared with a bight. Thus, many knots that form a loop from a bight in the standing part of the rope are named something on a bight, such as figure-8 on a bight.

The *tail* of a rope is the (usually short) unused length of rope that is left over once the knot is tied.

Categories of Knots

The most practical way to select a particular knot is to first evaluate what role that knot is expected to perform. For the purposes of this book, knots are addressed based on five basic functions: stopper knots, end-of-line knots, midline knots, knots to join two ropes, and safety knots.

There are subsets of knots for the terminology purist. A knot that is tied around something (e.g., a tree, a standing rope, or the rail of a litter), that conforms to the shape of the object around which it is tied, and that does not keep its shape when the object around which it is tied is removed, is called a *hitch*.

In its simplest form, a loop is a section of rope that crosses itself. A tied loop is a knot that forms a fixed eye or loop in the end of a rope. Regardless of the name or the way in which the terminology applies, there are basic rules that apply to any of these ties.

When working with rope, it is critical to be aware of the type of material into which the knot is tied. Some fibers have a low coefficient of friction and require special considerations in knot tying. Knots that are effective on rope do not always perform well in webbing or sling material.

Stopper Knot

A stopper knot is often used in rappelling. Before an end of the rope is thrown down for the rappel, a stopper knot is tied into the end to prevent the rappeller from rappelling off the end should the rope not reach the ground. Stopper knots are also used in other applications to perform a similar function.

The most common stopper knot is a *figure-8* (Fig. 84-4). When two ropes are used in tandem, the stopper knot should be tied into both lines together. A simple overhand knot (Fig. 84-5) may also be used for this purpose, but its relatively low bulk makes it less desirable than a figure-8.

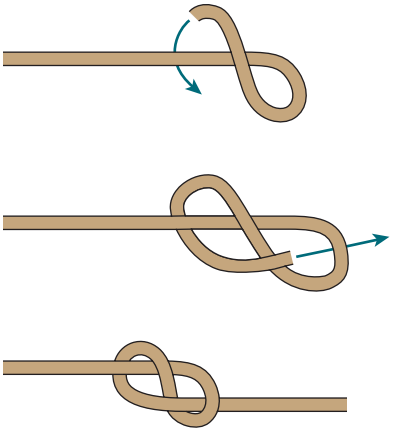

Figure 84-4. Figure-8 knot.

Stopper knots such as the figure-8 and the overhand, basic though they may be, are the foundation for many of the other knots one learns for wilderness use. It is important to learn these before progressing further.

End-of-Line Knot

Perhaps the most common use of a knot is to make a loop in the end of a rope to anchor, tie in, or attach the rope to something. Borrowed from mariners, bowlines have been used by mountaineers for years. However, this knot can "capsize" into a slipknot quite easily when the tail is pulled; therefore the *double bowline* (or high-strength bowline) is preferred (Fig. 84-6).

Another interesting bowline variation is the *bowline on a coil* (Fig. 84-7). Having several large loops, the bowline on a coil can be creatively used in a number of ways. For example, this knot is sometimes used to attach a person to a belay line if a harness is not available, or to create a load-distributing anchor.

Many people prefer to use forms of the figure-8 for multiple applications on the theory that they have to learn only one knot. One advantage is that the figure-8 may be tied directly onto a bight (Fig. 84-8), or it may be tied as a retrace (Fig. 84-9). Although the versatile nature of the figure-8 knot is indeed

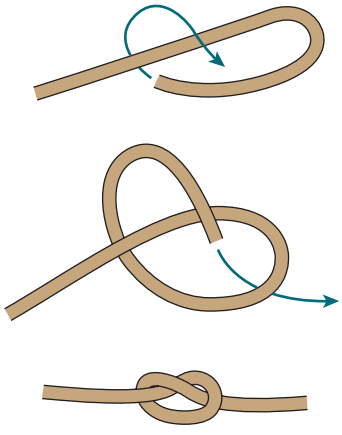

Figure 84-5. Overhand knot.

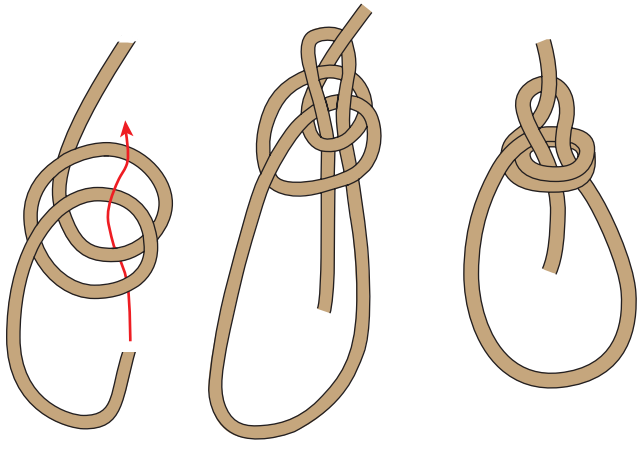

Figure 84-6. Double bowline.

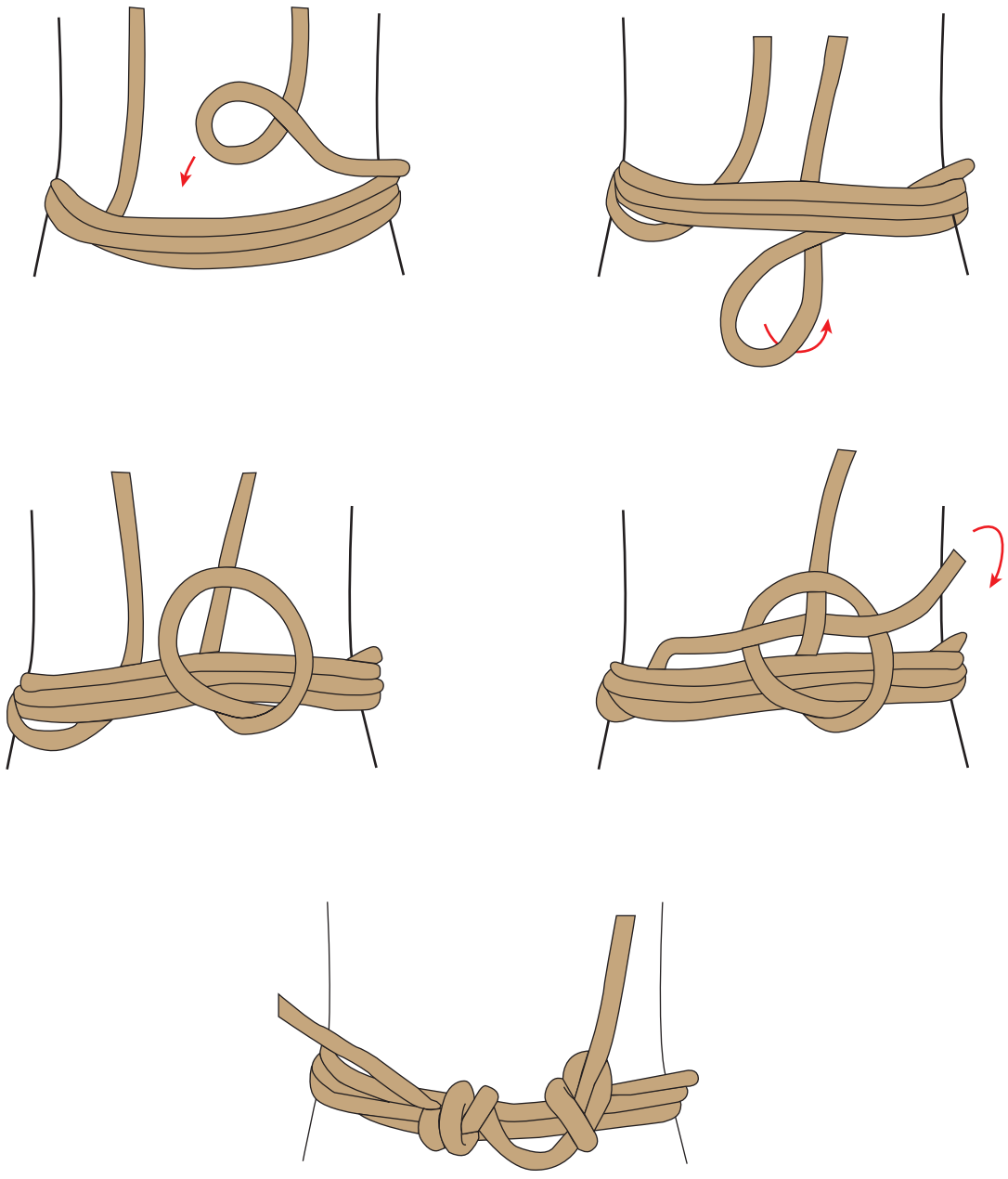

Figure 84-7. Bowline on a coil.

attractive, it should be noted that learning only one knot may prove limiting. Some people feel that the figure-8 is easier to tie and easier to check than other knots. In truth, the redundant nature of the figure-8 retrace can make it deceiving on visual inspection, a factor that has resulted in accidents.

Midline Knot

Knots are often used to form loops in the middle of the rope, for clipping into, for grasping, or for bypassing a piece of damaged rope. Perhaps the easiest and most common method of throwing such a loop is with a simple *overhand on a bight* (Fig. 84-10). Alternatively, a figure-8 on a bight (see Figure 84-8) may be used for this purpose. Either of these options works well as long as the load is attached to the bight. However, both these loops are susceptible to deformation when not loaded. Perhaps more importantly, if the rope below the knot is loaded, the knot deforms and weakens.

More preferable by far is the *butterfly knot* (Fig. 84-11), particularly if the loop and the line beneath it will be placed under significant load. The butterfly can be pulled effectively either from the loop or from below the knot without negative effect. Caution must be taken with this knot, however, because if the

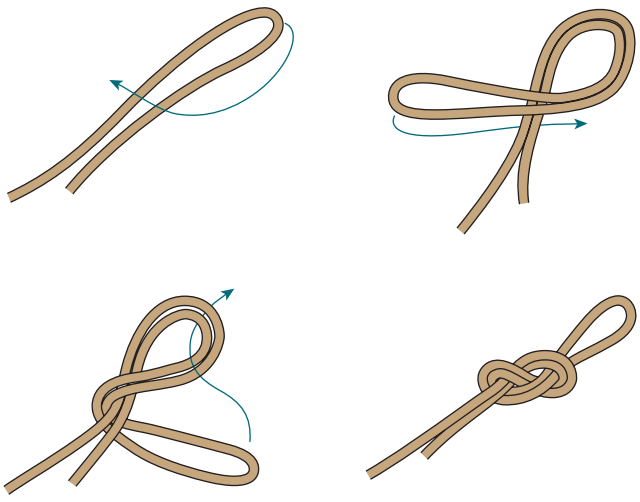

Figure 84-8. Figure-8 on a bight.

loop is not big enough and not loaded, it can pull out under tension.

For a quick and versatile solution to creating twin loops in the middle of a rope, a bowline can be tied on a bight of rope. The *bowline on a bight* (Fig. 84-12) results in two relatively symmetric loops that can be used to make an emergency boatswains chair, hand loops, or a towing bridle.

Knots to Join Two Ropes

Tying a knot that will not untie itself is very important when joining two ropes, particularly because the knotted ends are unlikely to be in a place where they can be constantly monitored.

Most people, by about the age of 3 years, have learned to tie a *square knot* (Fig. 84-13) for the purpose of joining rope ends. Although well known and easy to tie, this knot is far from being a secure or reliable tie. It can easily untie itself, either by pulling through when tightened or by shaking apart when loose. In short, the square knot should be avoided for all but decorative purposes.

The *overhand bend* (Fig. 84-14) is proclaimed by many in the climbing community to be the preferred choice for joining two ropes. However, this knot may untie while in use and is not deemed secure enough for rescue purposes. Instead, the more secure *double fisherman's bend* (Fig. 84-15) should be used.

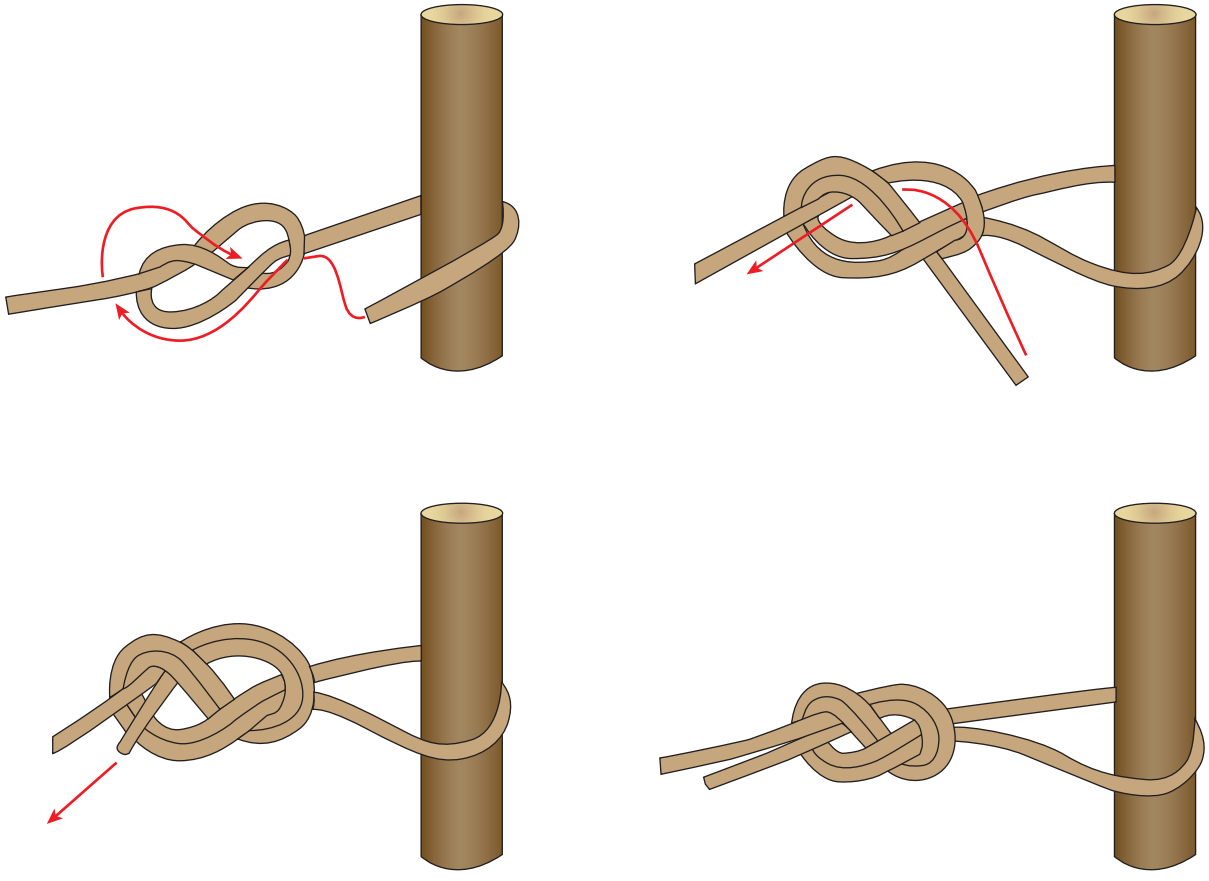

Figure 84-9. Retrace figure-8 on a bight.

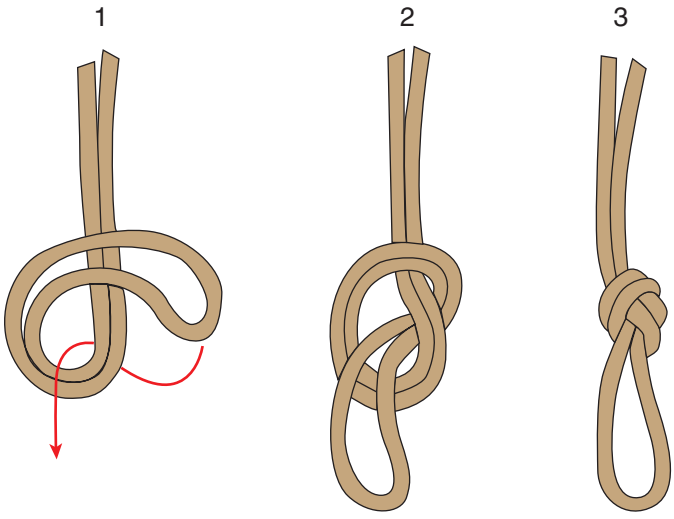

Figure 84-10. Overhand on a bight.

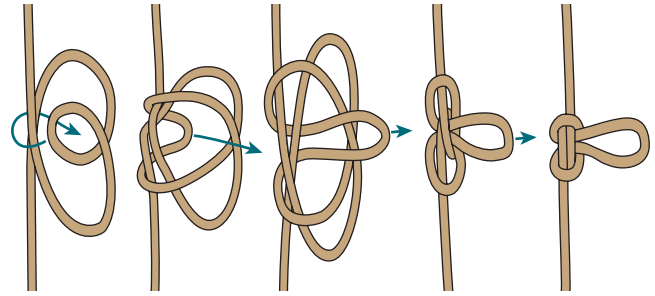

Figure 84-11. Butterfly knot.

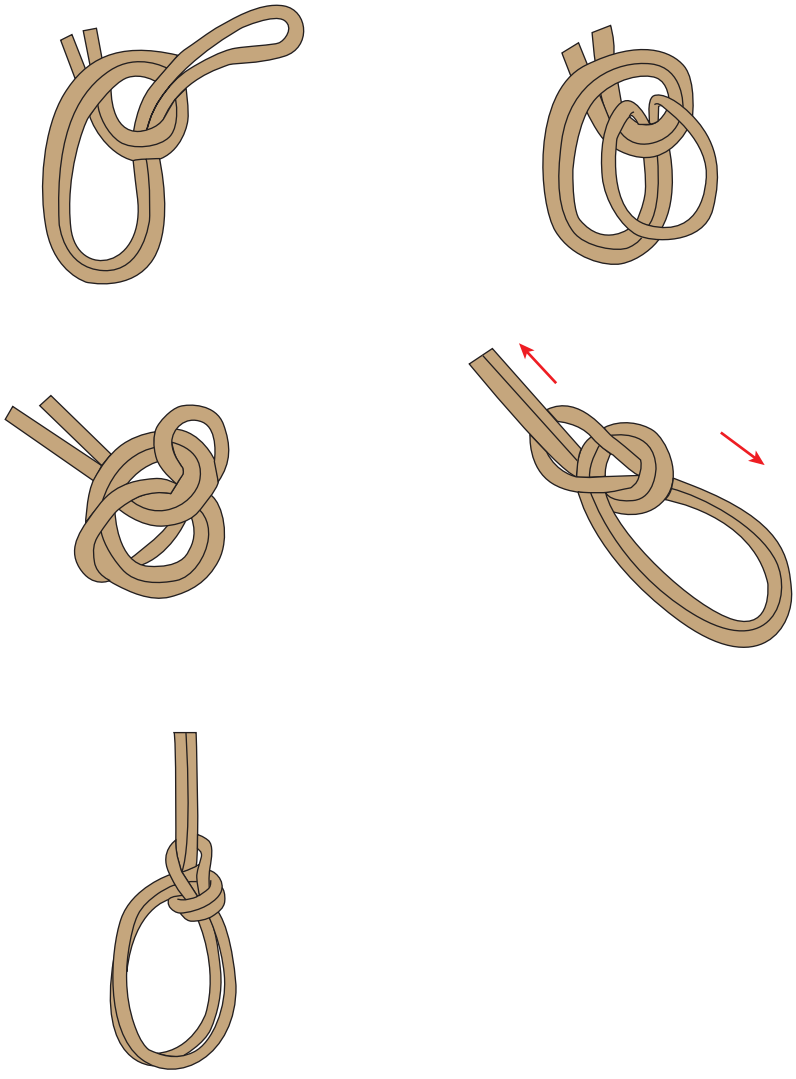

Figure 84-12. Bowline on a bight.

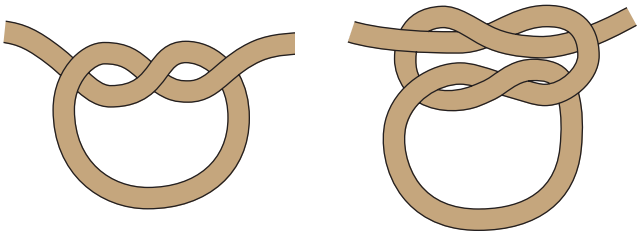

Figure 84-13. Square knot.

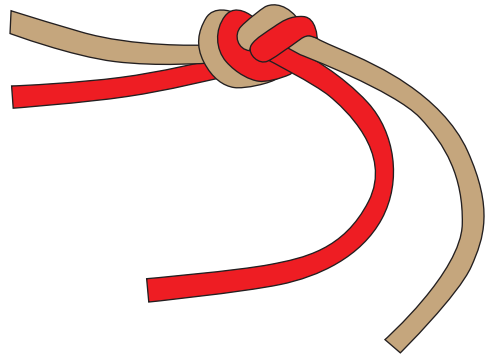

Figure 84-14. Overhand bend.

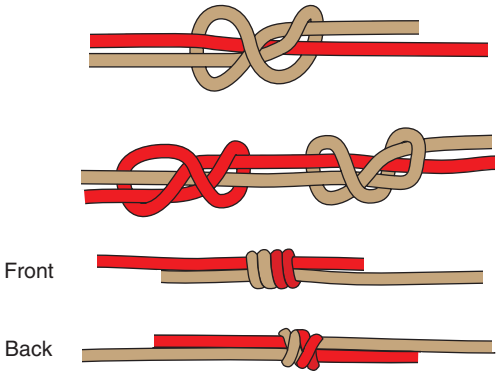

Figure 84-15. Double fisherman's bend.

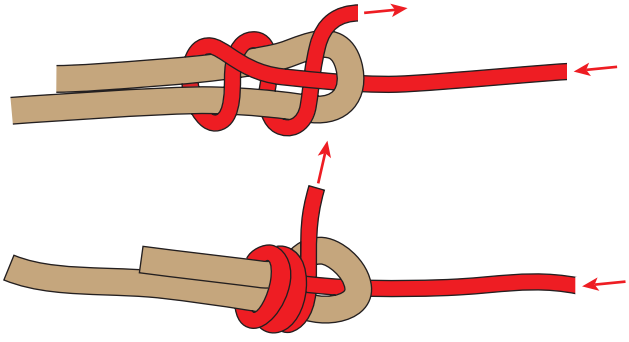

Figure 84-16. Double-sheet bend.

This bend is very effective for joining ropes of relatively equal diameter. Care should be taken to ensure that the two halves of the bend nestle against each other and that there is enough tail protruding from the knot to keep the knot from unraveling. This knot is also used to join two ends of a short length of cordage for use as a prusik (see Hitches).

When ropes of unequal diameter are joined, the *double-sheet bend* (Fig. 84-16) is a more effective tie. This is a bulkier alternative, and although perhaps not quite as strong, can be easier to untie and, in the case of joining ropes of different diameters, is preferred.

Of course, the versatile figure-8 deserves honorable mention here as well. Retracing a figure-8 in the opposing direction by a second rope results in what is commonly referred to as a figure-8 bend, an effective means of joining rope ends (Fig. 84-17). Care must be taken with this method to ensure that the rope ends exit from opposite ends of the bend. If tied simply as a figure-8 knot, this bend has a tendency to deform and pull itself apart (Fig. 84-18).

Most knots work best in cordage or rope that has a rounded surface. Flat webbing and similar materials perform differently under tension. The preferred bend for joining webbing ends is known as the ring bend, sometimes also called the tape knot (Fig. 84-19). This is most useful for forming webbing slings into a loop but can also be used for lashing.

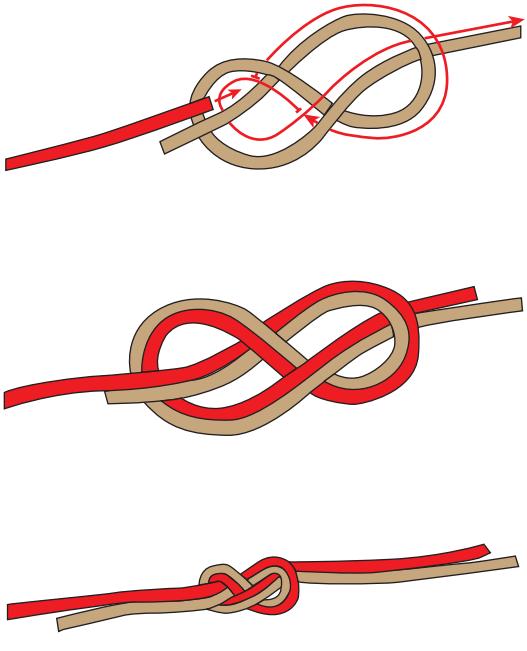

Figure 84-17. Figure-8 bend.

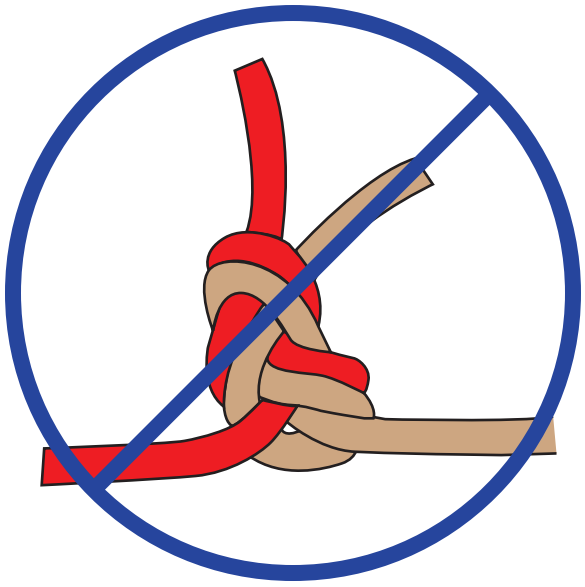

Figure 84-18. Incorrectly tied and loaded figure-8 bend.

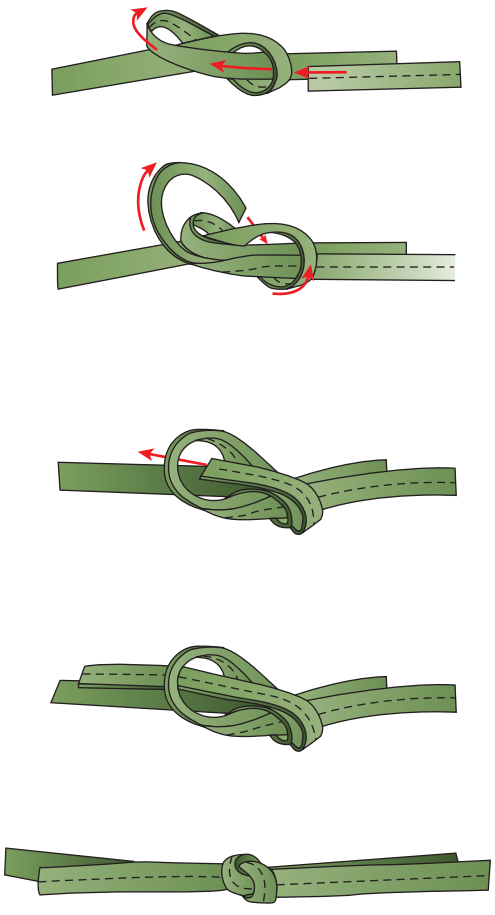

Figure 84-19. Ring bend (tape knot).

Hitches

Hitching refers to a method of tying a rope around itself or an object in such a way that the object is integral to the support of the hitch. Hitches are seldom used in rescue and should be considered for use only by a skilled technician because there are severe consequences when a hitch comes untied or does not perform as intended. Specifically, disintegration of a hitch results in immediate release of whatever load it is holding.

One of the most commonly used hitches is the *prusik hitch* (Fig. 84-20). A prusik is a sliding hitch by which a cord can be attached to a rope and slid up and down the rope for positioning. However, under tension, the hitch will not slide. A prusik is created by tying a length of cordage into a loop by means of a double fisherman's bend. Wrapping the loop around the main rope and through its own loop two or three times and then pulling it tight forms the hitch.

Another fairly common hitch used in climbing and rescue applications is the *clove hitch* (Fig. 84-21). This hitch can be useful when trying to shorten the distance between two objects, such as the climber's belay and the climber or the litter rail and the rescuer. It is also useful in some lashing techniques, but can have a tendency to roll loose.

Another type of hitch, called the *Munter hitch* or Italian hitch (Fig. 84-22), can be used around a carabiner or pole to add friction to a system, as in a belay. This hitch is particularly useful in that it effectively adds friction regardless of which direction the rope is moving. However, care should be taken when using the hitch around a carabiner because there can be a tendency for the moving rope to slip through the gate of the carabiner, rendering the hitch useless.

No discussion of knots would be complete without mention of the *trucker's hitch*, useful for pulling a cord tight across something, such as a load in the bed of a pickup truck (hence the name), or securing a patient snugly into a litter (Fig. 84-23).

Lashing

This process incorporates several concepts already discussed in the preceding sections on knots and hitches, and refers generally to the process of binding items together, such as poles for a shelter or drag. One basic technique used in starting or finishing a lashing involves the *round turn with two half hitches* (Fig. 84-24). This is a more secure, but bulkier, solution for lashing than the aforementioned clove hitch. There are several variations on the concept of lashing, including *square lashing* (Fig. 84-25) for arranging the poles to create a corner, *diagonal lashing* (Fig. 84-26) for joining poles to create a triangular shape, and *sheer lashing* (Fig. 84-27) for joining poles side by

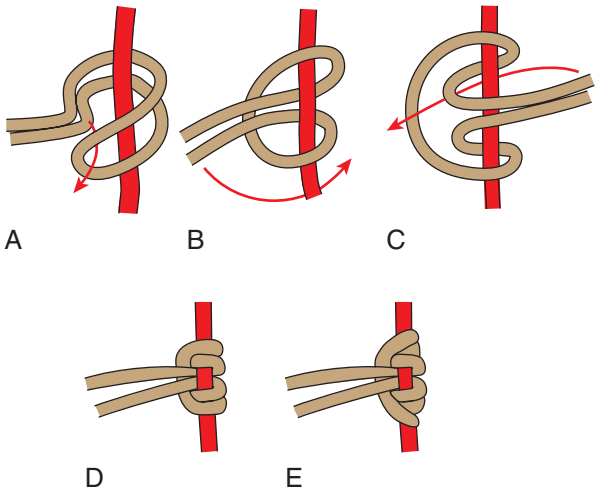

Figure 84-20. Prusik hitch. **A–C,** Tying sequence for the prusik hitch. **D,** Two-wrap prusik hitch. **E,** Three-wrap prusik hitch.

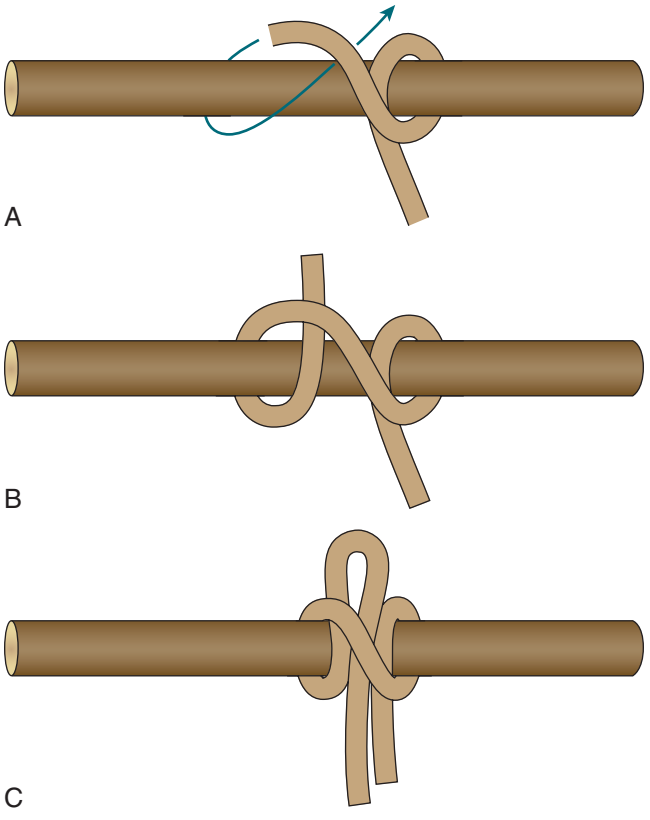

Figure 84-21. **A** and **B,** Clove hitch. **C,** Clove hitch with draw loop for temporary attachment.

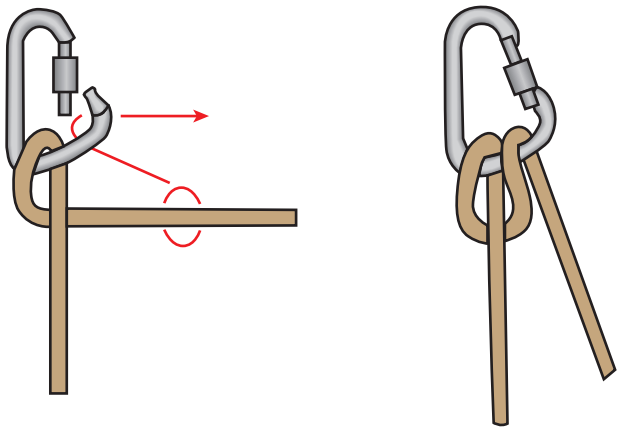

Figure 84-22. Munter hitch.

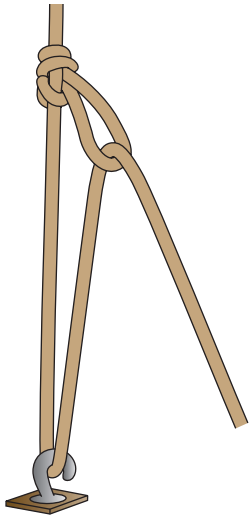

Figure 84-23. Trucker's hitch.

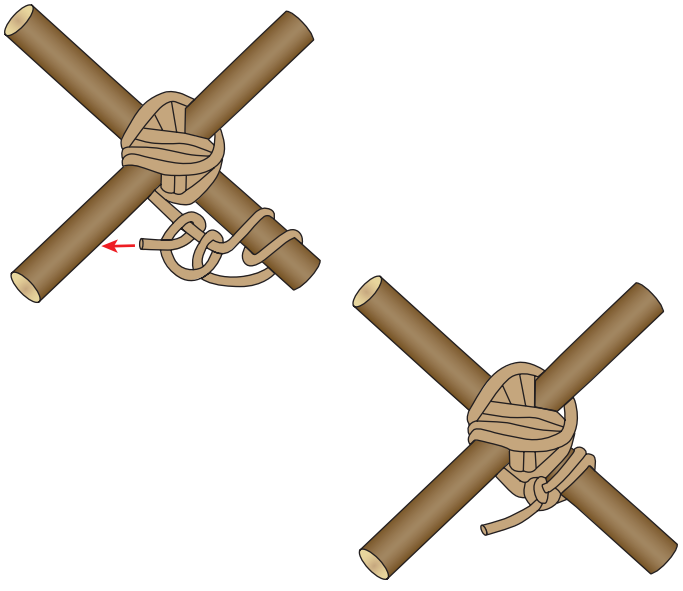

Figure 84-24. Round turn and two half-hitches.

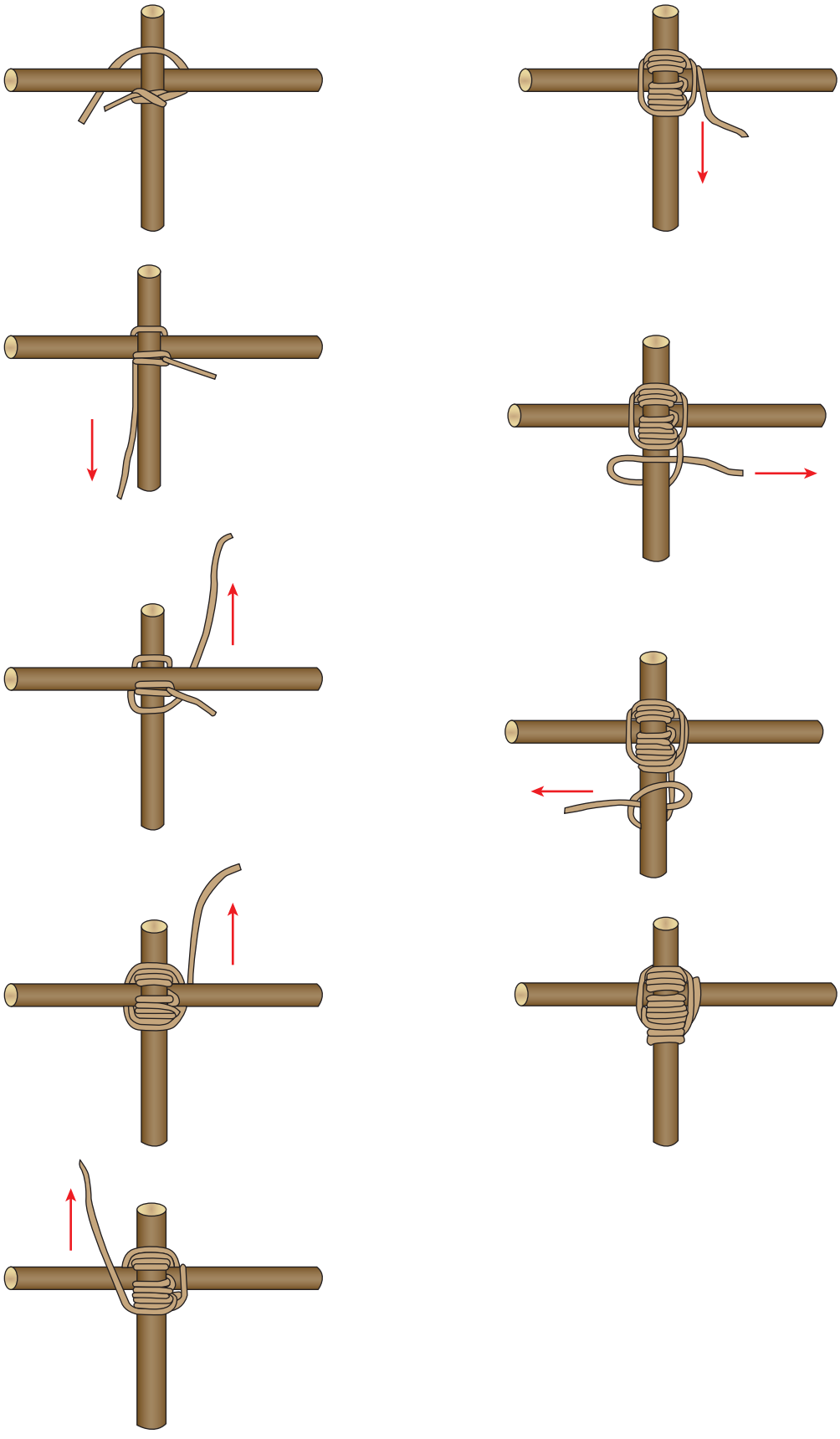

Figure 84-25. Square lash.

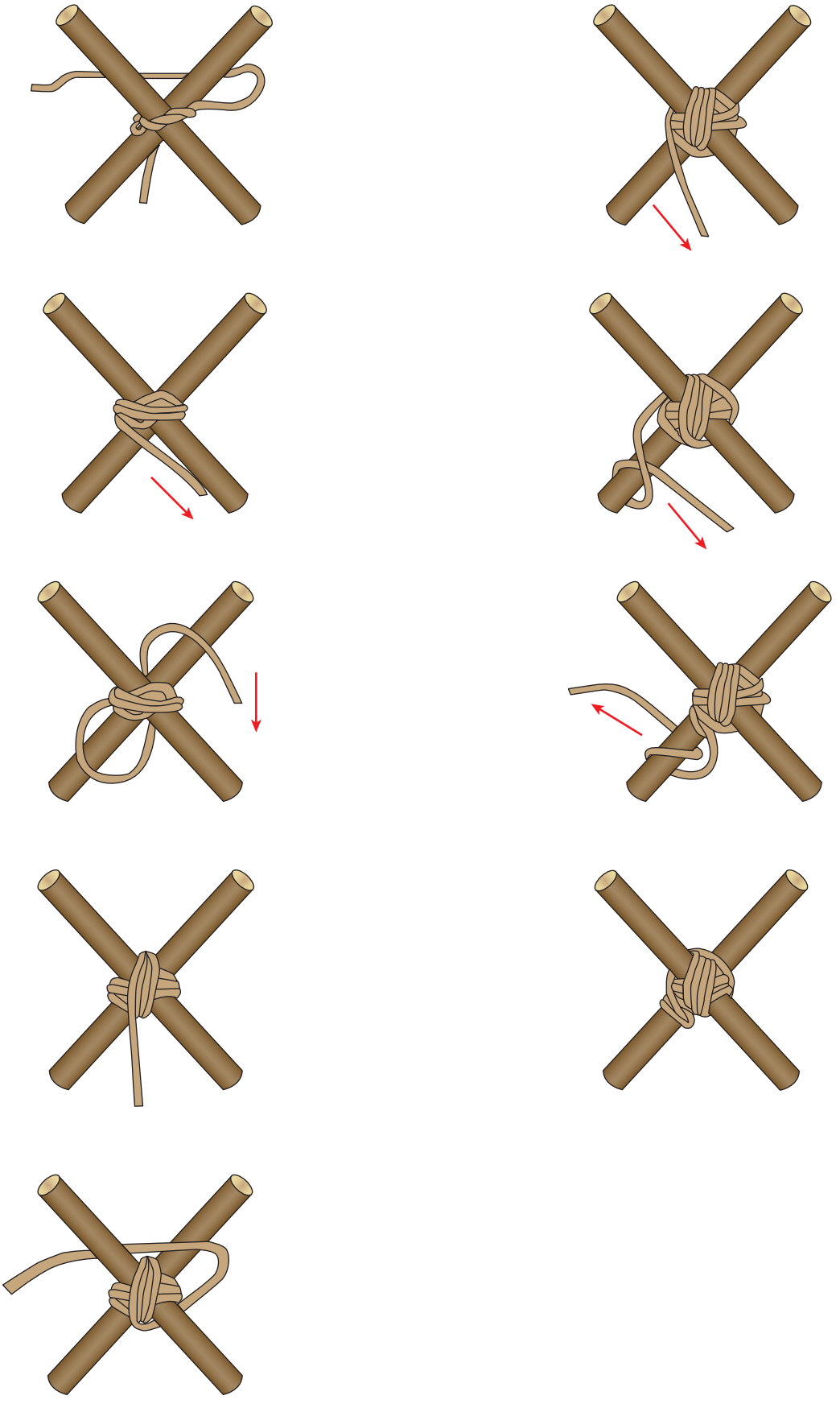

Figure 84-26. Diagonal lash.

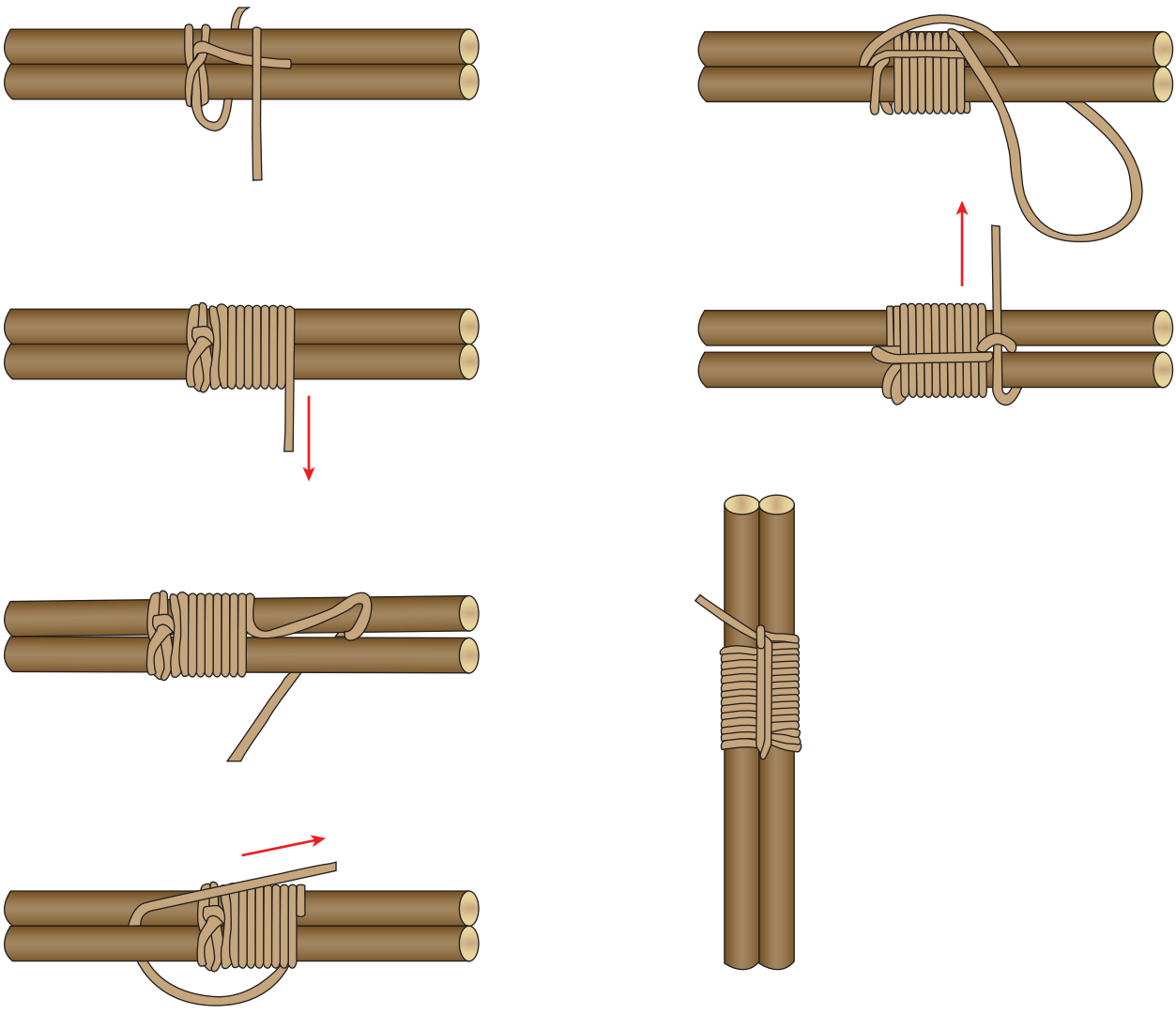

Figure 84-27. Sheer lash.

side. Lashings can be extremely useful tools for creating conveniences at camp as well as for emergency use in creating tripods and other mechanical aids.

Knot Safety

Every knot should be checked (preferably by someone other than the person who tied it) to ensure that it is tied properly and should be monitored at intervals thereafter. Many knots have a tendency to loosen, and some can even change forms, for instance, into a slipknot.

A safety knot can help prevent mishaps. A safety knot is an overhand knot (see Fig. 84-5) or *barrel knot* (Fig. 84-28) tied into the tail of the rope after the knot is tied. This safety knot is placed to keep the knot from deforming or unraveling.

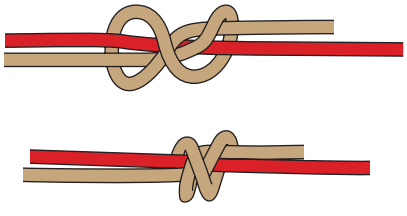

Figure 84-28. Barrel knot.

KNOTS AND ROPE STRENGTH

Knots inevitably reduce the strength of a rope to some degree. Knot strength is affected by the tightness of bends as well as by the "pinching" effect that a knot has on itself.

The strength of any given knot is directly proportional to the strength of the material into which it is tied. Knot strengths are usually expressed in terms of efficiency ratio. That is, a knot rated at 85% efficiency is said to maintain about 85% of the reported breaking strength of the rope.

Some individuals and agencies have reported that any knot reduces the strength of a rope by at least 50%. This information is erroneous because the efficiency of any knot depends on which knot is used, which rope it is tied into, whether it is tied correctly, and how it is cared for. Most knots recommended for use in wilderness rescue reduce the strength of a typical rescue rope to no less than 65% of that rope's minimum breaking strength. Most commonly used knots are even more efficient, in the range of 80%.

Unfortunately, accurate data on knot efficiencies are hard to find. Comprehensive testing that takes into account statistically significant sampling, differences between rope fibers, construction and diameter, static versus dynamic loading, and other variables is virtually nonexistent.

The following data, taken from many different sources and reflecting limited testing, should be referenced for trend information only.

The relative breaking strength of kernmantle design ropes with knots follows:

Double fisherman's bend: 65%–70%
Bowline: 70%–75%
Figure-8 on a bight: 75%–80%
Overhand knot: 60%–65%

The best way to know the strength of a knot on a given rope is to test the type of knot you use on the types of rope you use and test enough samples using statistical analysis to provide a reasonable margin of error.

LEARNING MORE ABOUT ROPES AND KNOTS

For more information on using ropes to move patients, we suggest the third edition of *High-Angle Rescue Techniques* by Hudson and Vines (see Suggested Readings), supplemented by training through a respectable course. A good basic vertical class on rappelling and ascending a rope requires a minimum of 2 to 3 days. After a basic rope skills class is completed, regular practice is required to maintain proficiency. Many excellent advanced classes in patient movement by rope or vertical rescue are offered. Figure on at least a week of training for a beginning class on rope rescue.

SUGGESTED READINGS

PMI product catalogs. LaFayette, GA, Pigeon Mountain Industries, 2005.

Vines T, Hudson S: High Angle Rescue Techniques, 3rd ed. St. Louis: Mosby, 2005.

85 Wilderness Navigation Techniques

Steven C. Carleton

Many environmental illnesses occur as a consequence of becoming lost. Even well-prepared individuals may suffer hypothermia, heat stroke, frostbite, immersion foot, sunburn, dehydration, starvation, and a variety of other conditions if they become separated from the resources of their expedition, or have an unanticipated extension of their time outdoors. As with other areas of medicine, so too in wilderness medicine: an ounce of prevention is worth a pound of cure. This chapter discusses use of the magnetic compass for route finding, map reading, application of celestial navigation techniques for the determination of direction and position, navigation with the Global Positioning System (GPS), orienteering, and use of alternative methods for recovering when lost.

All of navigation boils down to two processes: determination of direction and establishment of position. In the wilderness setting, the determination of direction and fixing of position are challenging. Generally, routes and landmarks are provided by a map, but local knowledge from memory may have to suffice. Generally, direction is derived from a magnetic compass, but other methods of direction finding can be exploited when the compass is forgotten, lost, damaged, or unreliable. Generally, *lines of position* (LOPs) are established by trails or by bearings to identifiable landmarks. However, shorelines, watercourses, firebreaks, power lines, altimeter readings, and relative bearings to celestial bodies or radio sources can substitute. Finally, position can be fixed through the use of technologies such as the

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GPS, or the time-tested methods of celestial navigation and triangulation of bearings. None of these navigational techniques is prohibitively expensive or so equipment-intensive as to be incompatible with a hiker's kit. Each method requires an understanding of its practice and limitations. Nothing substitutes for preparation, but effective wilderness navigation can be practiced using nothing more than the clues offered by the environment and the wits of the navigator. For travelers in the continental United States, it is significant that there is no site that is more than 29 miles from a road, and thus from a source of help.

COMPASS NAVIGATION AND DEAD RECKONING

The directional properties of lodestone (magnetite) were recognized by a variety of civilizations in ancient times. References to the use of a directional magnetized needle at sea appear in Chinese literature dating from the 12th century CE. Descriptions of the magnetic compass in European writings followed in the 13th century, by which time it was noted that a needle stroked on lodestone pointed to the vicinity of the North Star.[2,38] Discovery of the magnetic compass was a seminal event in the exploration of the planet; the compass allowed reasonably accurate steering in all weather and permitted development of a fundamental process in navigation, the process of *dead (deduced) reckoning*. Dead reckoning is the estimation of current location based on knowledge of the direction, rate, and time of travel from a known starting point. Whenever traveling in the wilderness, dead reckoning should be practiced so that a general awareness of position is never lost. Estimates of time of travel, rate of travel, and direction of travel should be recorded whenever possible so that the last known position and subsequent movement from that position are preserved.

Magnetic Dip, Deviation, and Declination

The directional properties of the compass result from the interaction between magnetized iron in the compass needle and the magnetic lines of force generated by metals in the earth's core. These lines of force have both a vertical and horizontal component. The vertical component is termed *magnetic inclination*, or *dip*. Dip causes a compass needle to incline downward from horizontal, potentially to a degree that interferes with the ability of the needle or card to pivot freely. Dip is 90° at the magnetic poles, and 0° at the magnetic equator. Most modern compasses are manufactured to compensate for the average dip likely to be encountered in the region of intended use. Others allow a small weight to be moved along the indicator needle to compensate for dip in any region of use.[32]

The horizontal component of the magnetic lines of force causes the compass needle to point to the earth's north magnetic pole. It is an unfortunate fact that the earth's magnetic and geographic poles do not correspond in location, and that the earth's magnetic lines of force are not straight lines, but meander in an irregular fashion dictated by irregularities in the density of the core. The irregular directionality of the earth's magnetic field is called *magnetic declination*. The compass needle is also influenced by local magnetic forces. These forces may result from natural sources such as ore deposits, or from artificial sources such as ferromagnetic metals in vehicles, equipment, and clothing fasteners. Displacement of the compass needle resulting from local magnetic influences is termed *deviation*. As a result of magnetic declination and deviation, compasses point to geographic north only when used with care in selected locations. In general, compasses point northward, but not exactly due north.

Direction in compass navigation is expressed in three ways: (1) *true direction*, or direction measured in reference to the earth's meridians and geographic poles; (2) *magnetic direction*, or direction measured in reference to the earth's magnetic poles; and (3) *compass direction*, or direction as measured by the magnetic compass. Magnetic direction varies from true direction by the sum of declination and deviation. Compass direction varies from magnetic direction by the quantity of deviation.[5,26] The definitions of magnetic and compass direction point out the necessity for minimizing preventable sources of compass deviation when taking bearings. For practical purposes, when preventable sources of deviation are minimized and the compass is used with caution, magnetic direction and compass direction can be considered to be equivalent.

Wandering lines of points with equal magnetic declination can be graphed on maps and charts. These are called *isogonic lines*. Lines representing points on the surface of the earth where the magnetic declination is zero, and magnetic north and true north are aligned, are termed *agonic lines*. In the Americas, an agonic line follows a relatively straight, slanting course extending from the east coast of Victoria Island in north-central Canada through western Lake Superior, along the west coast of Florida, and traversing South America from the Gulf of Venezuela to the southeastern coast of Brazil. At locations east of the agonic line, the compass needle declines to the west (counterclockwise) of true north; at points west of the agonic line, the compass needle declines to the east (clockwise) of true north. By convention, magnetic declination is given a positive sign when east, and a negative sign when west (Fig. 85-1). Declination is quantified as the angle between true and magnetic north.

By way of example, in southwestern Ohio, the current magnetic declination is negative 5.175°, or about 5° west. This means that a compass needle actually points 5° to the west of true north, and that the true bearing given by the needle is 355° when the needle points to 360° on the compass rim. Any magnetic bearing taken with a compass will thus be 5° greater than the true bearing. To correct from magnetic to true, 5° must be subtracted from any indicated magnetic bearing.

The mnemonic "declination east, compass bearing least; declination west, compass bearing greatest" may be helpful in converting magnetic direction to true direction when taking a bearing. Restated, to convert from magnetic to true while taking a bearing from the compass, add east declination to the compass bearing, or subtract west declination from the compass bearing. When taking a true bearing from a map and converting it to a compass bearing to follow in the environment, subtract east declination from the true bearing, or add west declination to the true bearing. The interconversion between magnetic and true bearings is an essential skill in compass navigation. Failure to recognize the relationship will result in significant errors when following a map route by compass, because directional references on the map are based on true direction. At a location where the magnetic declination is 10°, travel over a straight course derived from a map and guided by compass bearings will result in a 0.18-mile error for each mile traveled if the declination is not considered.[16]

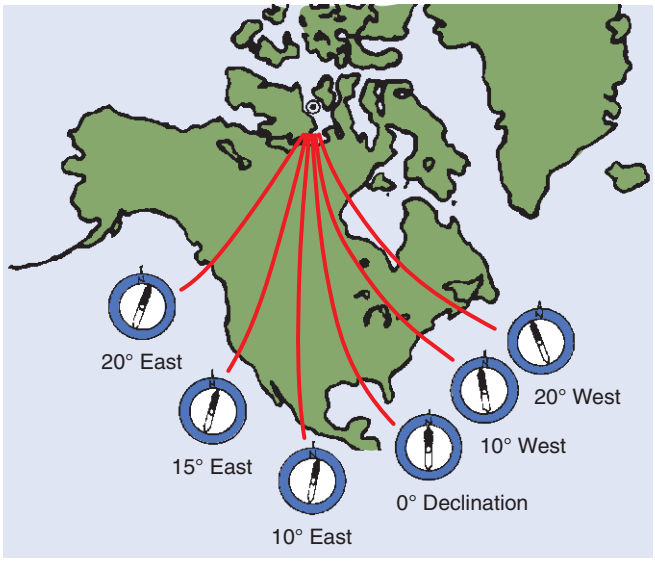

Figure 85-1. Schematic representation of North America showing declination at various locations as the difference between true north ("N" on the compass rim) and magnetic north (tip of the compass needle). (From Seidman D: The Essential Wilderness Navigator. Camden, ME, Ragged Mountain Press, 1995, with permission.)

Figure 85-2. The three basic compass types. *From left to right:* Fixed-dial compass, magnetic card compass, baseplate compass. Note the deviation in indicated north resulting from local magnetic influences.

Magnetic declination for any location can be determined by reference to the *Isogonic Chart for Magnetic Declination* produced every 5 years by the U.S. Geological Survey (USGS). In the United States, magnetic declination varies from +23° in Washington State to –22° in Maine[7] (see Figure 85-1). On standard USGS 7.5-minute and 15-minute squares, magnetic declination is indicated by a pointer next to the pointer indicating true north at the bottom of the map. Without recourse to an *Isogonic Chart,* declination for any location in the northern hemisphere can be empirically determined with reasonable accuracy by comparing the magnetic bearing of north with the true bearing, as indicated by a line pointing to the star Polaris. Polaris lies up to 45 minutes of arc (0.75°) away from true north at some times of day, but this offset is negligible for wilderness navigation situations. Declination at any location also can be determined by comparing the magnetic bearing of a prominent landmark to the true bearing between the observer's known location and the location of the landmark as read from a map.[3]

Compass Types

The three compass types used in land navigation are the fixed-dial compass, magnetic card compass, and baseplate compass (Fig. 85-2). The simplest compass is the fixed-dial, which utilizes a magnetized needle balanced on a pivot and enclosed in a case that is graduated around its periphery into 360°. The magnetic card compass uses a magnetized needle or wire fixed to a circular card that is graduated around its periphery from 0° to 360°. The housing of the compass is marked with a line, the lubber line, that allows magnetic bearings to be determined when the line is pointed at an object of interest. The lensatic compass used by the military, which has a lens for magnification of the compass card and sights for alignment to distant objects, is a magnetic card compass. The most useful compass for land navigation is the baseplate compass,[16,19,29,33] which consists of a fixed-dial compass (or capsule) mounted to a baseplate in a manner that allows the capsule to rotate in relation to the baseplate. The baseplate is marked with a line used to indicate the direction of travel. This line functions in a manner identical to the lubber line of the magnetic card compass. The capsule of the compass has an orienting arrow inscribed on its lower surface that points to the graduation denoting north on the capsule rim. On different models, this graduation may be labeled "0°," "360°," or "N." Rotation of the capsule such that the compass needle is superimposed on the orienting arrow and points to "N" on the capsule rim allows the user to easily read the magnetic bearing indicated by the direction-of-travel line. As long as the direction-of-travel line is followed and the needle remains superimposed on the orienting arrow, the user is assured of maintaining the desired magnetic bearing during travel.

Many baseplate compasses allow the orienting arrow to be adjusted relative to the rim of the capsule to compensate for magnetic declination. When the orienting arrow of such a compass is adjusted to point to the bearing of magnetic north, the "N" graduation on the capsule rim will indicate true north when the compass needle aligns with the orienting arrow. All bearings as read on the capsule rim will now represent true, rather than magnetic, direction. Baseplate compasses have other features particularly suited for use with a map, including plotting scales, a straightedge, and often a protractor and magnifier.[33]

Correction for declination when using a fixed-dial or magnetic card compass requires addition or subtraction of the declination, as appropriate, from the magnetic bearing indicated by the compass rim or lubber line.

Compass Use

The magnetic compass is used to establish the cardinal directions, bearings for use in route finding, and back-bearings for use in returning to a known starting location. A *back-bearing* is the reciprocal of the bearing followed on the outbound leg of a journey (outbound bearing minus 180°), or the reciprocal

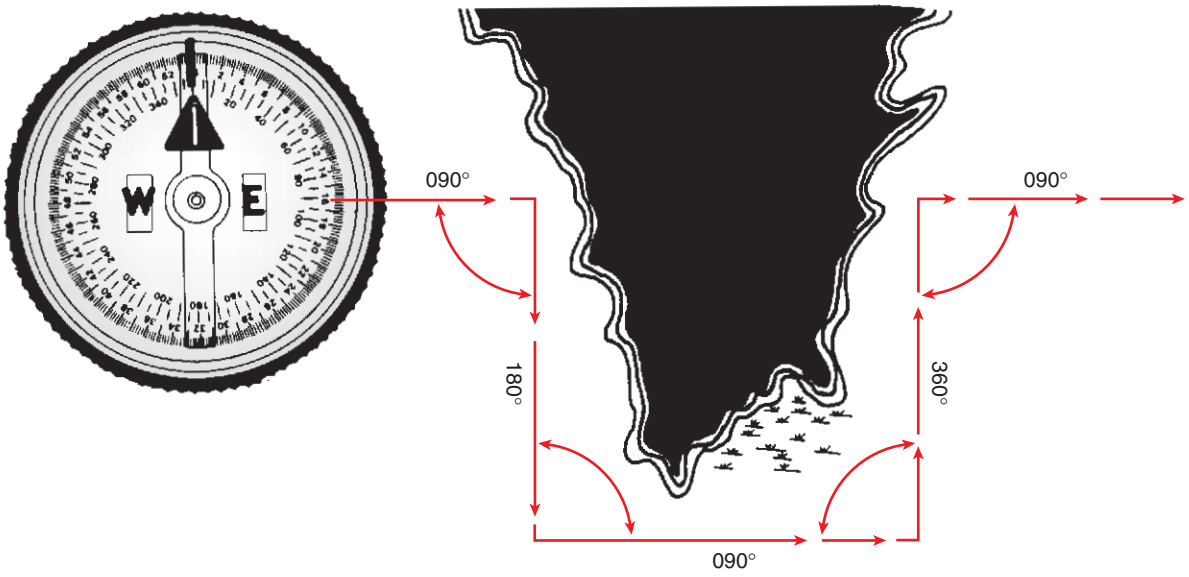

Figure 85-3. Use of the compass to return to an intended route when faced with an obstacle. A course 90° to the intended course is walked for a known number of steps, the obstacle is bypassed, and the original course is regained by walking the reciprocal course of the initial detour for the same distance. (From Department of the Army: Map Reading and Land Navigation. Field Manual 21-26. Washington, DC, Headquarters, Department of the Army, 1987, with permission.)

of a measured bearing to a prominent terrain feature. Any route can be subdivided into legs that can be defined by magnetic bearing lines. Ideally, each leg should pass between prominent, identifiable landmarks that will remain recognizable even in the dark or in poor weather. However, even when weather or lighting conditions prevent visual acquisition of the landmark from a distance, careful compass work should permit the user to reach an objective. Early in the course of travel over each leg, the observer should visually check the back-bearing of the direction of travel to become familiar with the view of the starting point as it will appear on the return journey. If possible, the bearing and back-bearing of each leg of a route, and the landmarks defining each leg, should be recorded on paper rather than trusted to memory.

Use of the compass in this manner permits the user to return easily to the desired direction-of-travel if an obstacle to the intended route is encountered. The course around the obstacle is recorded as a series of legs of known direction and estimated (by stride) length. When permitted by the terrain, right-angle detours are the simplest to follow. The user returns to the intended route by traveling the reciprocal of the course of the detour for the same distance as that required for bypass of the obstacle[5] (Fig. 85-3). Return to the intended course is greatly augmented by using *natural ranges*. A natural range is formed by two landmarks lying along the same bearing line, one of intermediate distance from the viewer and one of greater distance—for example, a large tree or rock formation several kilometers distant, and the silhouette of a hill or mountain on the horizon. As the traveler deviates from the intended course, the near and far landmarks will fall out of line. When the traveler returns to the intended route, the objects forming the natural range will return to alignment.

Use of a compass with a map allows the user to orient the map to the environment and relate bearings taken from the map to bearings measured with the compass. Correction for declination is essential when the compass is used for this task if true bearings are to be used in plotting a route. However, there is no absolute need for the use of true bearings in navigation; it is important only that the map and compass agree. Agreement can be accomplished either by correcting the compass to the map or by correcting the map to the compass. If a baseplate compass with declination adjustment is used, it is relatively simple to correct the compass to the map and use true bearings for all subsequent travel. With all types of compasses, however, it is easiest to use magnetic bearings exclusively. If the declination is known or can be determined observationally, magnetic meridians can be drawn on the map to be used in place of the true meridians represented by the map margins (Fig. 85-4). These magnetic meridians will form an angle with the true meridians equal to the declination angle. A map modified in this manner permits magnetic bearings, rather than true bearings, to be taken from the map for use in following a course. The internal consistency of this method is always much less confusing than the method requiring conversion between true bearings and compass bearings. Because of the ease with which bearings may be taken from the map when it is marked with magnetic meridians, maps for use in navigational sports such as orienteering are prepared exclusively with magnetic meridians. The choice between use of true versus magnetic bearings should be made in advance of travel to permit modification of the map. Plotting magnetic meridians on a map requires a pencil, straightedge, flat surface, and protractor, items unlikely to be available in a field emergency.[32,33]

To orient a map with a baseplate compass corrected for declination, the compass capsule is rotated such that "N" on the capsule rim is aligned to the direction-of-travel arrow. An edge of the baseplate parallel to the direction-of-travel arrow is then placed on one of the vertical borders of the map. The map,

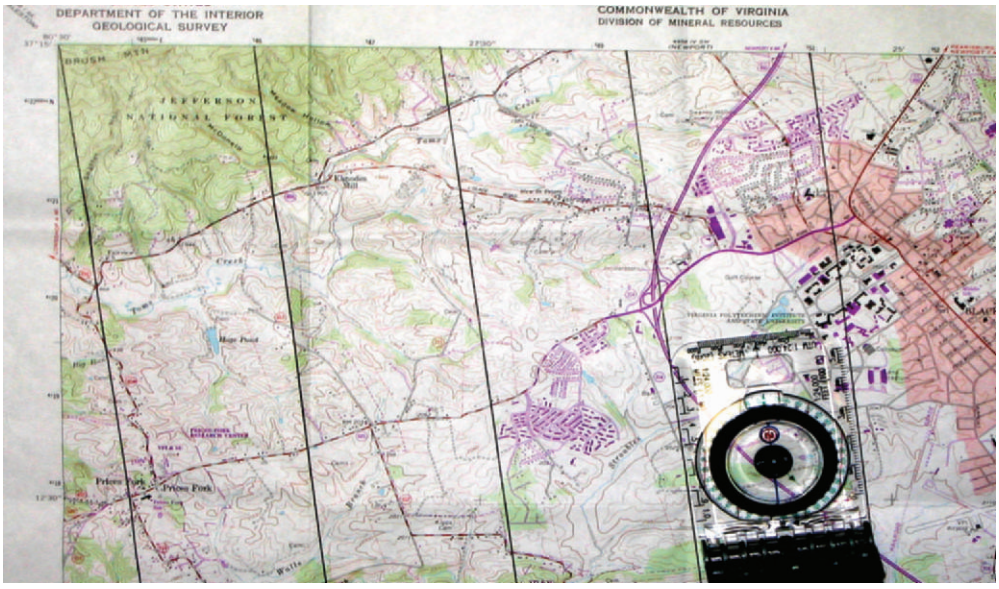

Figure 85-4. A U.S. Geological Survey (USGS) 1 : 24,000 scale map modified with magnetic meridians *(dark lines)*. The magnetic meridians reflect the 8° west declination of the area represented on the map. The compass has been placed with its edge parallel to a magnetic meridian, and the compass capsule rotated such that the orienting arrow on the bottom of the capsule points to north on the map. The map and compass were then rotated in concert to align the compass needle and the orienting arrow. The map is now oriented to the terrain, and directions as indicated on the capsule rim represent magnetic bearings to objects in the environment.

with the compass in place, is then rotated until the compass needle is superimposed on the orienting arrow on the base of the capsule. True north on the map is now aligned with true north on the planet.

To orient a map that has been modified with magnetic meridians using an uncompensated baseplate compass, the compass capsule is rotated until "N" on the capsule rim aligns with the direction-of-travel arrow. An edge of the compass parallel to the direction-of-travel arrow is placed on one of the magnetic meridians plotted on the map. The map, with the compass in place, is rotated until the compass needle is superimposed on the orienting arrow. True north on the map now corresponds to true north in the surrounding landscape.[16,29,32,33] If an uncompensated compass of another type is used, the north–south line of the compass face or lubber line is superimposed on a magnetic meridian, and the map and compass are rotated in concert until the indicator needle points to "N."

To plot and follow a bearing using a baseplate compass and map, the map is held horizontally and a straightedge of the baseplate parallel to the direction-of-travel arrow is placed on a line connecting the starting and ending points of the leg. The compass capsule is then rotated until the orienting arrow points to north as indicated on the map. The map and compass are held together and rotated until the compass needle is superimposed on the orienting arrow. The intersection of the direction-of-travel arrow and the capsule rim now indicates the bearing of the leg, and the direction-of-travel arrow points to the objective (Fig. 85-5). To follow the bearing, the user walks in the direction indicated by the direction-of-travel arrow while keeping the compass needle and orienting arrow in alignment.

Once a map is oriented to the environment, back-bearings from landmarks that are visible both on the map and in the landscape can be used to obtain a positional fix by *resection,* or triangulation.[29] Each back-bearing from a landmark represents a LOP that can be plotted on the map. The point of crossing of two or more LOPs fixes the position of the observer (Fig. 85-6). Alternatively, the intersection between the LOP represented by a bearing line and a shoreline, riverbank, road, firebreak, or celestial position line can be used to fix position on a map.

Makeshift Compasses

A field-expedient compass can be fabricated with relative ease. Items containing iron, nickel, or cobalt are suitable for use as an indicator needle. Iron in the form of a steel needle, pin, wire, staple, or paper clip is most commonly available. Most of these items are magnetized as purchased. If not, they can be magnetized by stroking them on a magnet salvaged from an electric motor or radio speaker, on a magnetized screwdriver or similar item, or on a piece of silk. A dry cell also can be used to magnetize a needle by wrapping an insulated wire tightly around the needle and connecting the ends of the wire to the battery terminals for several minutes. There is a fire risk associated with shorting the battery terminals in this manner; expect sparks and heat. Trial and error will often yield a suitable magnetizer. The indicator needle is floated in water by placing it on a wood chip, leaf, slip of paper, or small piece of cork or closed-cell foam. The container, which may be the cupped palm of the hand or a puddle, should be protected from the wind (Fig. 85-7). A compass so constructed will reliably indicate a magnetic north-to-south line. Absolute determination of directions may require external cues, such as the general direction of sunrise, sunset, or the sun at mid-day.

CELESTIAL NAVIGATION

Celestial navigation exploits the predictable relationship between the apparent positions of selected celestial bodies and the surface of the earth. The influence of celestial navigation on world history is immense. Millennia before European cultures conceived of celestial navigation as a means for facilitating exploration, commerce, and military domination of the seas, the people that populated the islands of the Pacific Basin were

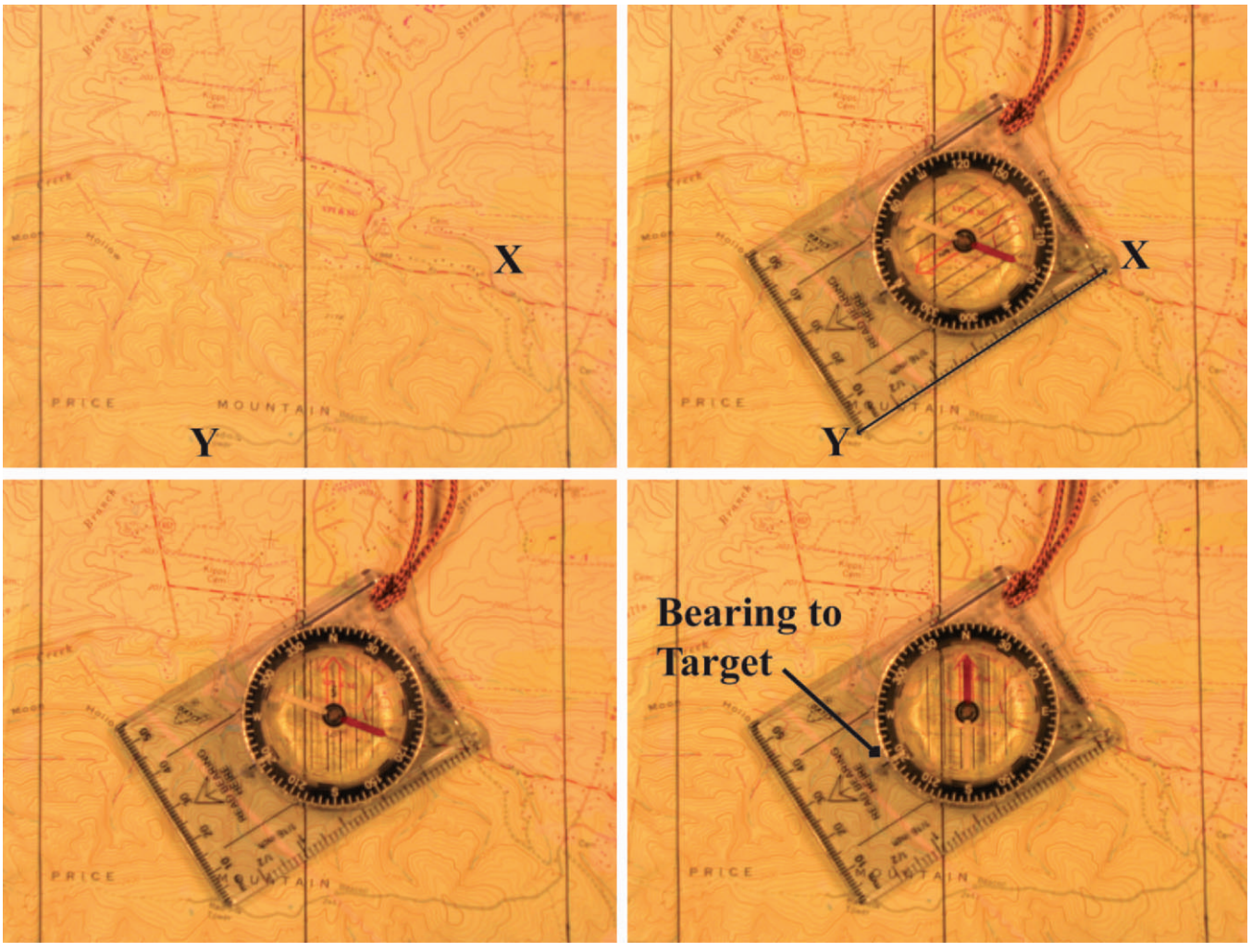

Figure 85-5. The steps for plotting and following a bearing using map and compass. The current position (road junction at X) and destination (watch tower at Y) are identified **(A).** The compass is then placed on the map with an edge along the line connecting X and Y **(B).** The compass capsule is rotated so that the orienting arrow points to north as indicated by the meridians on the map **(C).** The compass is now oriented to the map. The compass and map are now held horizontally and rotated until the magnetic needle is superimposed on the orienting arrow **(D).** The map is now aligned to the environment, and the bearing to Y is indicated at the intersection of the compass capsule and the direction-of-travel arrow (240° in this example). As long as the needle is kept superimposed on the orienting arrow, the direction-of-travel arrow will point toward the destination.

making open ocean voyages over thousands of miles guided by memorized "star paths."[12] The use of simple altitude measuring devices (the Greek gnomon, Arab kamal, and Chinese stretch board) for the qualitative determination of latitude was practiced by mariners in antiquity. As the Age of Exploration dawned, competing governments of Western Europe devoted enormous energy to systematization of celestial navigation, funding legions of astronomers in the development of coordinate systems and accurate tables of stellar, solar, lunar, and planetary positions. Instruments such as the mariner's astrolabe, quadrant, octant, and sextant permitted accurate and reproducible quantitative measurement of the altitude of celestial bodies, and accurate clocks became available to permit the ready determination of longitude.[2,38]

Celestial navigation has its greatest utility and easiest application at sea. Standard celestial practice depends on the availability of a sea horizon as a reference point for the measurement of altitude. The horizon reference for land navigation is necessarily artificial (a plumb bob, bubble level, or level reflective surface). Positions resulting from celestial fixes, even with scrupulous technique, are only approximations of the navigator's actual position. Errors of several miles are common. This is of little consequence in the open ocean, even as landfall approaches; the target destination is generally large enough to be seen from many miles distant. This degree of imprecision may prove troublesome on land, but the accuracy of celestial navigation should still suffice for many wilderness situations.

Although developed for use on trackless seas, celestial navigation has a long and rich tradition of use on land. Lewis and Clark established the positions of landmarks during their exploration of the American West by celestial observation. Various expeditions that led to the exploration of the North and South

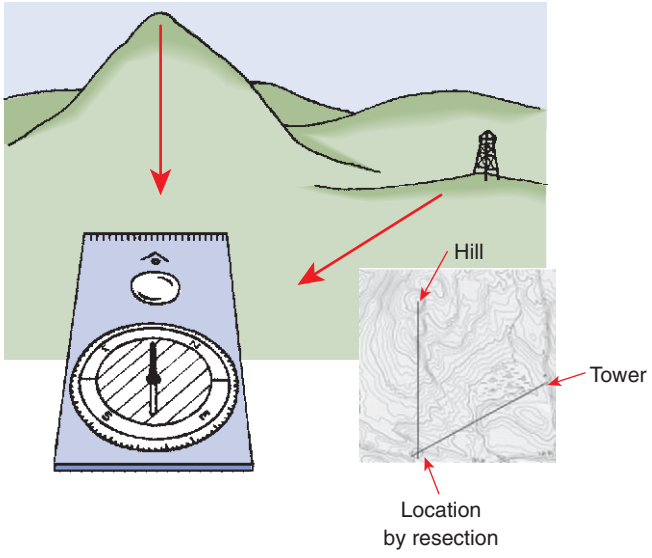

Figure 85-6. Establishing a magnetic fix by crossing the back-bearings from two prominent landmarks. This process is known as *resection.*

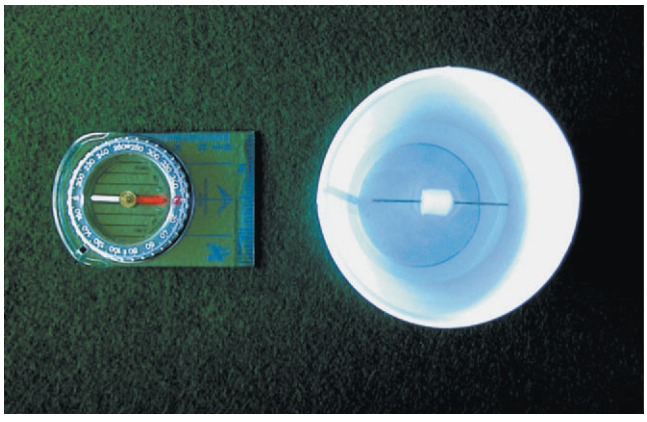

Figure 85-7. Makeshift compass constructed from a plastic cup, straight pin, and foam packing peanut. Note the correspondence between north as indicated by the makeshift device and the commercial compass.

Poles depended entirely on celestial observations to determine direction and position. Celestial navigation techniques can be applied in a wilderness setting at a variety of levels. Traditional celestial navigation, in which sextant observations lead to the fixing of terrestrial position, can be accomplished with relatively little equipment but requires considerable preplanning. This methodology is unlikely to be adopted for use by the vast majority of wilderness travelers. Simplified celestial techniques requiring little or no equipment are well suited to field-expedient navigation, however, and can provide accurate directional information in the absence of a compass. In particular, an understanding of the movement of the sun and several prominent stars can allow accurate route finding without recourse to technology. To understand the relationship between the position of a celestial body and the position of an observer on Earth, it is necessary to discuss the various coordinate systems that are used to describe terrestrial position, celestial position, and the appearance of the sky from the earth's surface (horizon coordinate system).

Terrestrial Coordinates

In the terrestrial coordinate system, the earth has a North Pole and a South Pole that define its axis of rotation. This axis passes through Earth's center. Any plane that passes through the center of the earth describes a circle on the surface called a *great circle.* The equator is the great circle described by the plane that passes perpendicular to the earth's axis. The great circle of a plane that contains the earth's axis is termed a *meridian.* Meridians always run due north and south and converge at the poles. The *Prime Meridian* is the great circle that passes through Greenwich, England. Greenwich was assigned the Prime Meridian by treaty in 1884, in recognition of the work on astronomy and navigation performed at the Greenwich Royal Observatory. Every point on the earth's surface lies on a meridian. The angular measurement, or arc, between the Prime Meridian and the meridian of any other point on the planet's surface is the *longitude* (λ) of that point. Longitude is measured in degrees, minutes, and seconds of arc, east or west of the Prime Meridian, from 0° through 180°. The angular measurement between the plane of the equator, as measured north or south from the center of the earth to a point on the surface, is the *latitude* (L) of that point (Fig. 85-8). All points at the same latitude form a *parallel* of latitude. Latitude is measured in degrees, minutes, and seconds of arc from 0° through 90° north or south. As such, the latitude of the equator is 0°, whereas that of each pole is 90° north or south, respectively. Every point on the surface of the earth is defined by a specific longitude and latitude.

A *nautical mile* (1852 meters, 6076 feet, or 1.15 statute miles) is the distance on a great circle that subtends an angle of 1 minute of arc as measured from the center of the earth. A degree of arc (60 minutes of arc) is thus 60 nautical miles, and one second of arc is equal to about 100 feet (30.5 m). It must be recognized that 1 minute of latitude will always equal 1 nautical mile, whereas one minute of longitude will equal 1 nautical mile only at the equator. At all points north or south of the equator, 1 minute of longitude will be less than 1 nautical mile, owing to the convergence of the meridians toward the poles.

Longitude bears a special relationship to time. Within reasonable standards of accuracy, the earth rotates once about its axis in 24 hours. As such, our planet moves through 360° of longitude in 24 hours, or 15° each hour, or 1° for every 4 minutes of time. It is this fact that establishes the conventions by which sundials work, clocks run, and time is defined. It also forever links the modern practice of celestial position finding to the accurate keeping of time. An alternative terrestrial coordinate system, the Universal Transverse Mercator (UTM) grid system, was specifically designed to facilitate land navigation. This is discussed in a subsequent section.

Celestial Coordinates

To a terrestrial observer, the sky appears to be an immense hollow sphere with the earth at its center, and the stars painted on its inner surface. The sphere rotates about the earth once daily. The sun, moon, and planets wander across the background of stars on concentric spheres of their own. This is exactly the conception of the universe forwarded by Ptolemy in

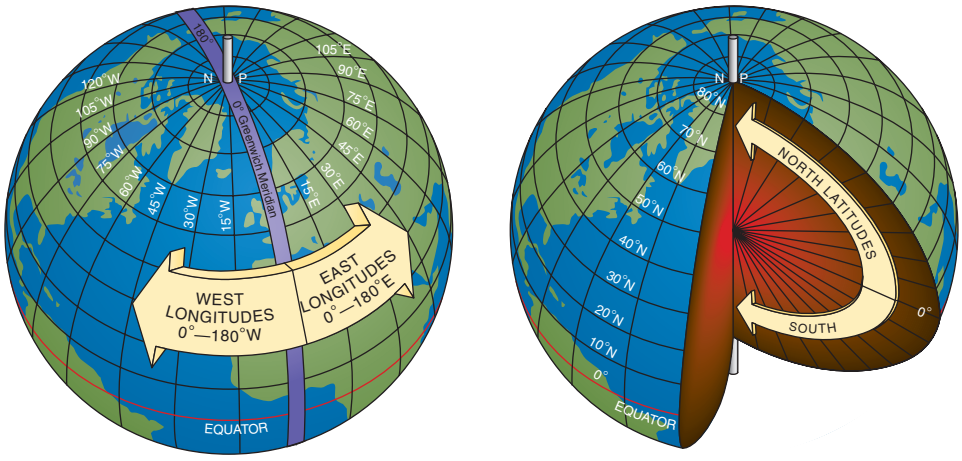

Figure 85-8. Meridians of longitude, including the Prime Meridian, and parallels of latitude as measured north and south of the equator in degrees of arc from the center of the earth. (From Department of the Air Force: Survival—Training Edition. Manual 64-3. Randolph AFB, TX, Air Training Command, 1969, with permission.)

the 2nd century CE, and is termed the *geocentric model*. This theory was highly popular with the Catholic Church; challenging it led to the persecution of many intellectuals during the era of the Spanish Inquisition. It held sway until the 16th century, when it was replaced, gingerly, by an equally incorrect heliocentric (sun-centered) model. The geocentric model, although false, is a useful construct for ordering the heavens, and is the model used in the definition of celestial coordinates.

The celestial coordinate system plays off of the terrestrial coordinate system. The terrestrial poles are projected outward to the surface of the imaginary celestial sphere to form the north and south celestial poles. The terrestrial equator is similarly projected outward to form the celestial equator, or *equinoctial*. The celestial correlate to latitude is *declination* (unrelated to magnetic declination in compass use). The declination of a celestial body is the angle measured from the center of the earth (and geocentric universe) north or south of the celestial equator from 0° through 90° of arc. Declinations north of the celestial equator are positive (+), whereas those south of the celestial equator are negative (–). The declinations of the celestial poles are thus +90° and –90°, respectively, whereas that of the celestial equator is 0°. The declinations of the sun, moon, four navigational planets (Venus, Mars, Jupiter, and Saturn), and 57 navigational stars are listed in the daily pages of the *Nautical Almanac*.[7,15]

The celestial correlate to longitude is less intuitive and requires some explanation. Projections of terrestrial meridians onto the celestial sphere form *celestial meridians*. The projection of the Greenwich meridian onto the celestial sphere forms the *Greenwich celestial meridian*. The projection of an observer's meridian onto the celestial sphere forms the *local celestial meridian* (Fig. 85-9). A meridian on the surface of the celestial sphere that contains a celestial body and the celestial poles, and is perpendicular to the celestial equator, is termed the *hour circle* of that body. Hour circles converge toward the celestial poles in a manner identical to meridians on Earth. As the celestial sphere rotates about its polar axis, the hour circles of all celestial bodies appear to rotate above a terrestrial observer, rising in the east, sweeping overhead, and setting in the west. A reference hour circle on the celestial sphere called the *first point of Aries*, or the hour circle of Aries, substitutes for the terrestrial Prime Meridian. The first point of Aries is represented by

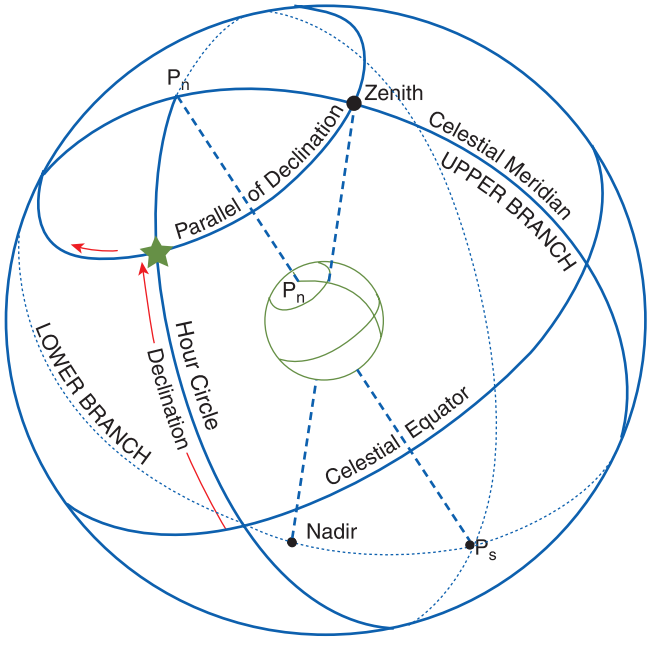

Figure 85-9. Diagram showing the celestial meridian of an observer on the earth's surface, and the celestial meridian corresponding to the hour circle of an observed star. (From Bowditch N: The American Practical Navigator: An Epitome of Navigation. Washington, DC, Defense Mapping Agency Hydrographic/Topographic Center, 1984, with permission.)

an hour circle that intersects the celestial equator at the point where the sun crosses it at the moment of the spring equinox. The angular measurement between the hour circle of a celestial body and the Greenwich celestial meridian is defined as the *Greenwich hour angle* (GHA) of the body. GHA is the celestial equivalent of longitude, and is measured westward from the Greenwich celestial meridian from 0° through 360°. The Greenwich hour angle of Aries (GHA^{Aries}) is the angle between the Greenwich celestial meridian and the hour circle of Aries at any given second in time. The GHAs for Aries and for the sun,

moon, Venus, Mars, Jupiter, and Saturn are listed for each hour of each day of a year in daily pages of the *Nautical Almanac*. Interpolation tables permit determination of hour angles for each second of the year.[15,26]

The GHAs for the 57 navigational stars are defined by their predictable and nearly constant relationship to the hour angle of Aries. The angular measurement between the hour circle of Aries and the hour circle of any of the navigational stars is called the *sidereal hour angle* (SHA) of that star. The GHA of each of the navigational stars is thus equal to the sum of the hour angle of Aries and the SHA of the star in question ($GHA^{star} = GHA^{Aries} + SHA^{star}$).

In the practice of celestial navigation, the hour angle of a celestial body is ultimately defined in reference to the observer's local celestial meridian. The angular measurement between the local celestial meridian and the hour circle of a body is the *local hour angle* (LHA) of that body. The LHA, like the GHA, is measured westward from the local celestial meridian from 0° through 360°. The LHA of a celestial body equals the GHA of the body plus the observer's longitude if east, or minus the observer's longitude if west (LHA = GHA + east λ; LHA = GHA – west λ). When a celestial body is on an observer's meridian, its GHA equals the longitude of the observer's position.[2]

Horizon Coordinate System

The horizon coordinate system defines celestial position from the point of view of the earthbound observer. The point on the celestial sphere directly over the observer's head is the *zenith*. The point on the celestial sphere directly beneath the feet of the observer is the *nadir*. The zenith and nadir lie on a line that includes the observer's terrestrial position and the earth's center. The plane that passes though the center of the earth perpendicular to this line is the observer's *celestial horizon*. The celestial horizon lies parallel to the observer's visible horizon. Any great circle on the celestial sphere formed by a plane passing perpendicular to the celestial horizon is termed a *vertical circle* (Fig. 85-10). Vertical circles converge at the observer's zenith and nadir in a manner analogous to the convergence of the terrestrial and celestial meridians at their poles. Three of the infinite number of potential vertical circles for any surface position are of particular importance: (1) the *principal vertical*, that vertical circle lying on the observer's celestial meridian; (2) the *prime vertical*, that vertical circle passing through points due east and west of the observer; and (3) the vertical circle containing a celestial body of interest[15] (see Figure 85-10).

The angular measurement between an observer's celestial horizon and a celestial body is the *altitude* of that body. Altitudes are expressed in degrees, minutes, and seconds of arc from 0° (the horizon) through 90° (the zenith). In the traditional practice of celestial navigation, altitudes are measured using a sextant, octant, or quadrant. Altitude, as measured by an instrument in reference to the horizon, is termed *sextant altitude* (Hs), regardless of the type of instrument used. Hs, once corrected for various atmospheric and geometric factors, becomes *observed altitude* (Ho). The corrections to be applied are tabulated in the *Nautical Almanac*. The true altitude of a body can be calculated for any given terrestrial position at any instant of time through application of spherical trigonometry. This altitude is termed *calculated altitude* (Hc).[2] The minimum required equipment for altitude measurement and position calculation based on celestial observation is surprisingly portable and is shown in Figure 85-11.

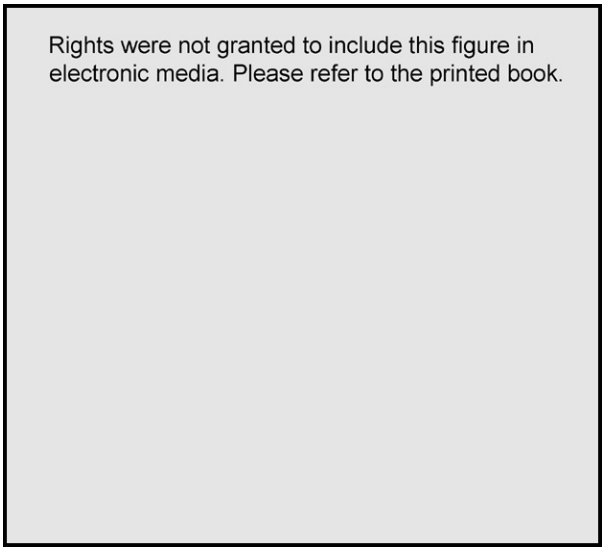

Figure 85-10. Schematic representation of the horizon coordinate system showing the observer's zenith and nadir, the celestial horizon, the prime and principal vertical circles, the elevated pole (Pn), and the altitude and azimuth of the body observed. (From Hobbs RR: Marine Navigation: Piloting and Celestial and Electronic Navigation, 4th ed. Annapolis, MD, Naval Institute Press, 1998, with permission.)

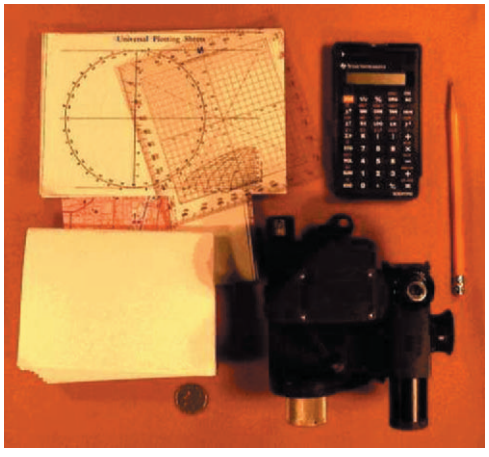

Figure 85-11. A complete portable kit for celestial navigation. *Clockwise from upper right:* Scientific calculator, pencil, A-10 aeronautical sextant, folded copies of almanac daily pages and sight reduction pages from *H.O. 249,* protractor/straightedge, plotting sheets, and topographic map. The sight reduction pages and calculator can substitute for one another. The topographic map and plotting sheets can substitute for one another, although the map is more useful for land navigation. Not included in the image is an accurate watch set to Greenwich Mean Time (GMT). A U.S. quarter is included to provide scale.

The other determinant of the observed position of a celestial body is the angular measurement along the celestial horizon between true north and the vertical circle including the body. This angle is termed the *azimuth* (Zn) of the body, and is measured clockwise from 0° through 360° (see Figure 85-10).

Celestial Lines of Position—Methods for Latitude

The simplest celestial LOP is derived from the *noon sight,* or *meridian altitude of the sun.* This method dominated celestial navigation from the 17th century until the late 1800s. The noon sight is simple but inflexible. It is very adaptable to wilderness navigation, requiring minimal mathematics and little equipment. The noon sight allows the direct determination of latitude by measuring the altitude of the sun at the moment that it passes the observer's meridian. The instant of solar meridian passage is termed *local apparent noon* (LAN), and is that moment when the sun achieves its greatest altitude. The sun is observed with the sextant for a brief period of time preceding LAN until the altitude ceases to increase and begins to fall. The maximum sextant altitude so determined is corrected using *Almanac* data to yield Ho, which is then converted to *zenith distance* (ZD). Zenith distance is the angular distance between observer's zenith and the body; thus ZD = 90° – Ho. The algebraic sum of ZD and the declination of the sun (d; from the *Nautical Almanac*) at the approximate time of observation gives the observer's latitude by the following formulas[7,20]:

1. L = ZD + d, when L and d have the same sign and L > d
2. L = d – ZD, when L and d have the same sign and d > L
3. L = ZD – d, when L and d have different signs

Because the declination of the sun changes very slowly on an hourly basis, accuracy is acceptable if the sun's declination is known for merely the approximate time of observation. The noon sight requires only the sun's declination and precise altitude. Determination of latitude by meridian passage can be accomplished through observation of any celestial body of known declination, although observation is easiest with the sun.

Another extremely simple observation to determine latitude involves measuring the altitude of Polaris (also known as α Ursae Minoris, the Pole Star, or the North Star). Polaris rotates about the true north celestial pole on a very short radius (about 45 minutes of arc). At two times each day, the altitude of Polaris will be equal to the altitude of the true pole, and that altitude will equal the latitude of the observer. The altitude of Polaris most closely approximates the altitude of the pole, and thus latitude, when the constellations Ursa Major (the Big Dipper) and Cassiopeia are positioned as indicated by Figure 85-12. For the remainder of the day, there will be a predictable discrepancy ranging from zero to 45 minutes of arc between the measured altitude of the star and the latitude of the observer. The *Nautical Almanac* has a brief table that corrects for this discrepancy, allowing an observer to determine accurate latitude by the altitude of Polaris at any time when the star is visible and a suitable horizon is available. The Polaris sight requires Greenwich Mean Time (GMT) to within several minutes, a precise measurement of altitude, and data from the *Nautical Almanac.*[2,7]

The latitude obtained by the noon sight or Polaris sight represents the simplest available celestial LOP. This line can be used to determine a fix if coupled with a sight that yields longitude, or with another LOP obtained by any other method. In land navigation, this second LOP might represent a river, shoreline, road, trail, compass bearing, or radio bearing that crosses the determined latitude line at a single point.

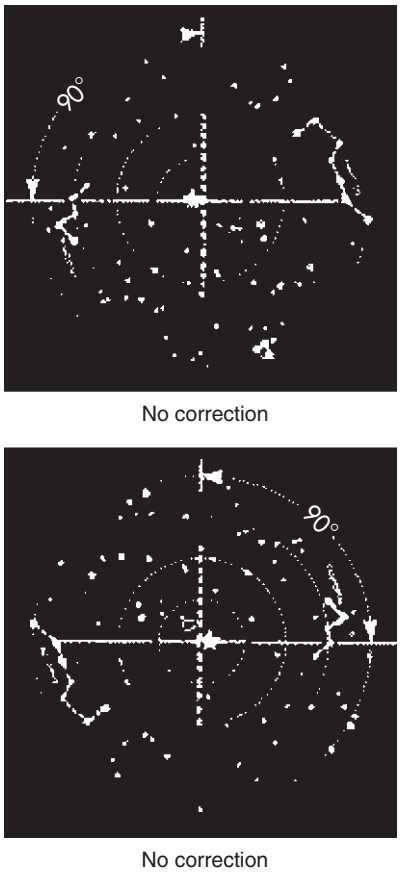

Figure 85-12. Diagram indicating the appearance of the north circumpolar sky at the two times of day when the observed altitude of Polaris is equivalent to the latitude of the observer. (From Department of the Air Force: Survival—Training Edition. Manual 64-3. Randolph AFB, TX, Air Training Command, 1969, with permission.)

Celestial Lines of Position—Methods for Longitude

Determination of longitude is considerably more involved than finding latitude, requiring precise timekeeping, meticulous observation, extensive preplanning, and detailed tables of celestial data. The seminal advance in the search for a method of determining longitude was the invention of the chronometer by John Harrison in 1735. With an accurate timepiece, longitude could be directly determined by measuring the altitude of any celestial body of known coordinates at a precisely known instant of time. This method, termed the *time sight,* revolutionized exploration but involves formidable mathematics. A simpler alternative exists that is adaptable to the wilderness setting. This traditional technique for establishing longitude is called the *equal altitude method.* The equal altitude method requires accurate timekeeping, the ability to measure altitude with precision, and data from the *Nautical Almanac.* At a convenient interval before the meridian passage of the body (1 to 2 hours), the altitude of the body is taken and the GMT is noted. The sextant is left at the altitude setting of the first observation. After the body passes the meridian, it is observed until it falls to the exact altitude of the first reading. The time is again noted. Meridian passage will have occurred at the midpoint between these two times. The GMT of meridian passage is then known. At the moment of meridian passage, the LHA of the body equals zero, and the GHA of the body (from the *Nautical Almanac*) equals the observer's longitude.

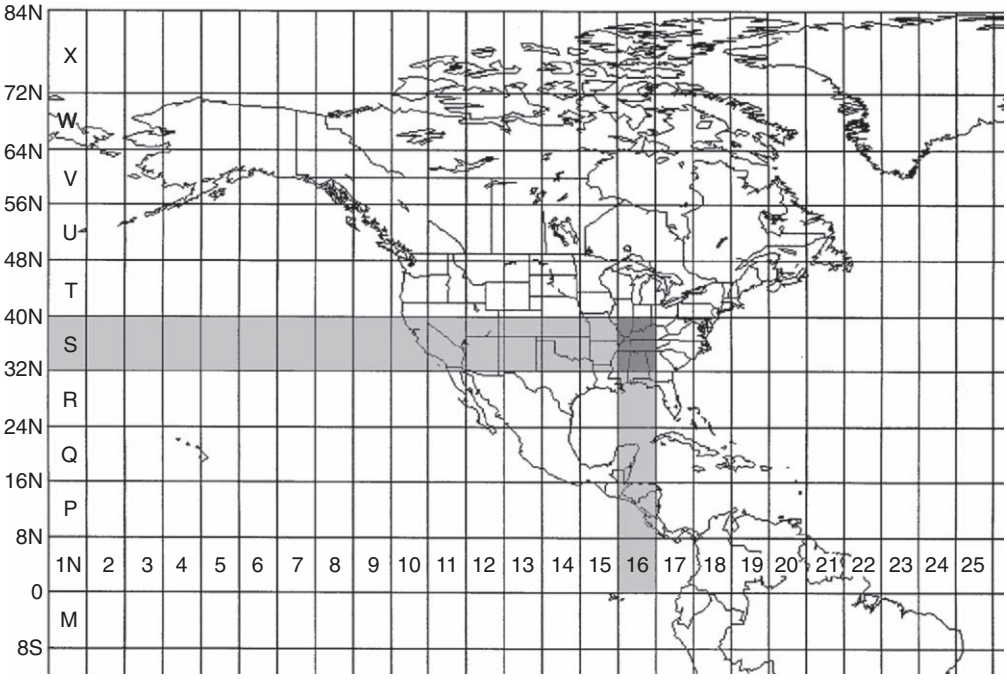

Figure 85-13. Map with the Universal Transverse Mercator (UTM) grid overlaid on a portion of the western hemisphere. Zone 16 and row S are highlighted in light gray. The area of their intersection, designated as *16S,* is highlighted in dark gray. The central meridian for this zone lies at 87° west longitude. The uppermost right-hand corner of the map represents the area used in the examples of UTM coordinates discussed in the text.

In theory, the instant of meridian passage could be found by noting the moment the body reached maximum altitude, achieving the same result. This is highly inaccurate in practice because of the extremely slow rate of change of altitude in the minutes immediately surrounding LAN.[20] Other elegant and highly accurate methods of determining longitude and position exist. These use Sumner lines, a particularly flexible form of celestial LOP discovered by Thomas H. Sumner in 1837, and the altitude-intercept method discovered by Marcq St. Hilaire in 1875. These methods have a rich and colorful history, and serve today as the principal methods for celestial position finding at sea. However, these techniques are unlikely to be practiced by the land navigator and are not discussed further here. Interested readers are referred to several references for further study.[2,7,15,20,37]

Alternative Coordinate System

The latitude and longitude coordinate system evolved in response to the requirements of navigation at sea, where identifiable landmarks may be absent for thousands of miles, and unobstructed visibility in good weather may permit identification of a landfall from a considerable distance. Although conceptually useful, latitude and longitude are less well suited to land navigation, where precision on the order of tens of meters is often required, and where visibility may be limited by terrain features and vegetation. The determination of latitude and longitude is inextricably linked to external references such as celestial bodies or satellites. Without recourse to these external references, calculation of a new position based on the direction and distance traveled and from a known starting point requires intimidating mathematics. Furthermore, the interconversion of minutes or seconds of arc to meters or feet is cumbersome and confusing.

An alternative terrestrial coordinate system, the Universal Transverse Mercator (UTM) grid or Military Grid Reference System (MRGS), was adopted by the U.S. Mapping Agency in 1947 to cope with the specific exigencies of maneuvering and delivering ordinance on land. The UTM and MGRS grids are metric, use the transverse Mercator map projection in common with maps distributed by the USGS, and require no conversion between distances expressed as angles and linear measure distances such as meters, kilometers, feet, or statute miles. The UTM system divides the earth's surface, from 80° south latitude to 84° north latitude, into 60 zones arrayed around the surface of the planet like narrow slices of an orange. Each zone is 6° wide in longitude. The zones are consecutively numbered from 1 through 60 beginning at an index line, the International Date Line (IDL), and progressing eastward. Each zone is subdivided from south to north in 8° increments of latitude (except the northernmost zone, which encompasses 12° of latitude [72° north to 84° north]). The south-to-north divisions of each zone are labeled consecutively and alphabetically from C through X, excepting the letters I and O to avoid confusion with the numbers 1 and 0 (Fig. 85-13). In UTM, terrestrial coordinates are expressed in meters east of a *false origin,* and north of a latitude index line. The false origin for any zone is an arbitrarily assigned north–south line 500,000 m to the west of the central meridian of the UTM zone of interest. Progress to the east of the false origin is termed *false easting,* or simply *easting.* Progress to the north of the latitude index is termed *northing.* In the northern hemisphere, the latitude index is the equator. In the southern hemisphere, the latitude index is the southern limit of strip C (80° south latitude).[17,21,34]

UTM coordinates are printed on all USGS maps produced in the last several decades. These appear as numbered, blue tick marks occurring at 1-km intervals along the horizontal and vertical map margins. The numbers express UTM eastings and

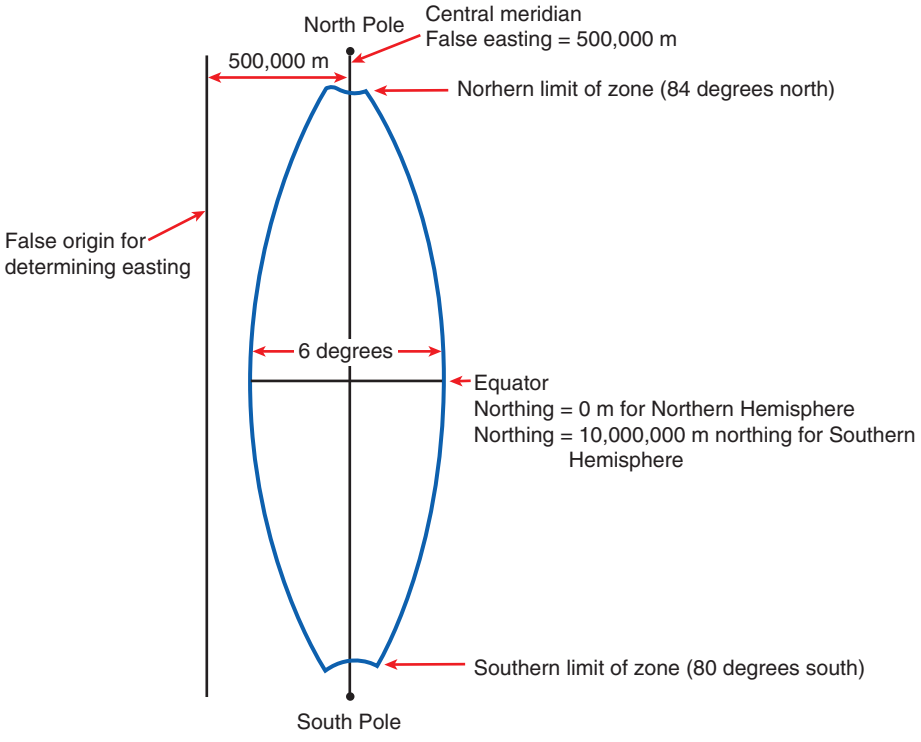

Figure 85-14. Diagram of a representative Universal Transverse Mercator (UTM) zone showing the false origin 500,000 m to the west of the central meridian of the zone. The zone extends from 80° south latitude to 84° north latitude and spans 6° of longitude. Note that the equator forms the index line for reckoning northing for the northern hemisphere, whereas the southern limit of the zone forms the northing index for the southern hemisphere. (Redrawn from Cole WP: Using the UTM Grid System to record historic sites. National Register of Historic Places Bulletin no. 28. Washington, DC, U.S. National Park Service, 1977. Available at www.cr.nps.gov/nr/publications/bulletins/nrb28/.)

northings, and thus increase from left to right, and from bottom to top (i.e., to the right and up). By convention, the central meridian of each of the 60 UTM zones is assigned an easting value of 500000 m E (500,000 m east of a false origin of the zone). Easting values less than 500,000 lie west of the central meridian for the applicable UTM zone. Easting values greater than 500,000 fall east of the central meridian for the UTM zone in question. Slight overlap (80 km) between adjacent zones prevents negative easting values from occurring. Similarly, easting values of greater than 1,000,000 are never encountered; they would lie beyond the overlap in the next zone to the east. For the northern hemisphere, northing values are expressed as meters north of the equator, and range from zero to 10000000 m N (10,000,000 m north of the equator; Fig. 85-14).

Most UTM tick marks on a map margin are labeled with three-digit numbers, representing kilometers. On the horizontal margins of the map in the right upper or left lower corners, one of the last blue tick marks is labeled with a six-digit number in mixed large and small numerals (716000 m E in Fig. 85-15). This means, "716,000 m east of the false origin, or 216,000 m east of the central meridian of the applicable UTM zone, because the false easting value is greater than 500,000" (the central meridian in this example is in UTM zone 16 at 87° west longitude). Similarly, one of the blue tick marks at the right lower or left upper vertical margins of the map image is labeled with a multidigit number (4374000 m N), indicating a line 4,374,000 m north of the equator in UTM row S. Note that within the six-digit false easting value and seven-digit northing value, the numerals in larger type represent thousands of meters, or kilometers.

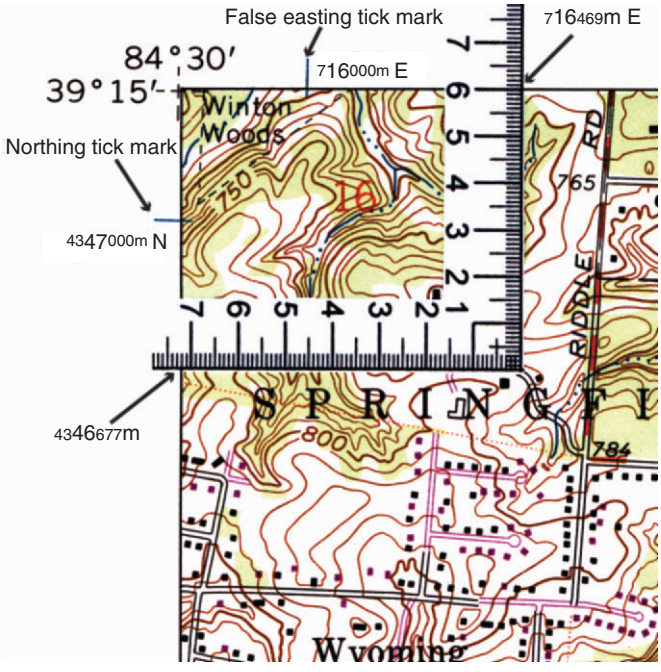

Figure 85-15. Portion of a U.S. Geological Survey (USGS) 1:24,000 map showing the blue Universal Transverse Mercator (UTM) tick marks on the map margin that give values for false easting (716000 m E) and northing (4347000 m N). A 1:24000 UTM roamer scale is superimposed on the map. The points of intersection between the arms of the roamer scale and the map margin allow the grid reference for the point at the outside corner of the roamer scale to be read.

Every point on the earth's surface can be described by a unique false easting and northing value, described as a *grid reference*. For practical purposes, when navigating within an area of several dozens to several hundreds of square miles, the UTM margin ticks on any large-scale USGS map can be thought of as a simple kilometer scale. The long and intimidating numeric labels on the tick marks can be ignored, except to recognize that they indicate 1-km increments.

Use of the UTM grid system for plotting position based on a map is actually much simpler than its conceptual framework would suggest. Position plotting is greatly assisted by using a specialized ruler called a *roamer scale* that is compatible with common USGS map scales (see Figure 85-15). Roamer scales are included on many baseplate compasses, can be easily made from a scrap of paper, or can be printed from Internet sources. The large graduations on a roamer scale denote 100-m increments, whereas the smaller divisions represent a 10-m span. The steps for determining UTM coordinates for any point on a compatible map follow:

1. Mark the position of interest on the map with an "X."
2. Examine the margins of the map to become familiar with the three-digit labels for the UTM ticks. For most hiking trips covered by a single map or by a small number of maps, the UTM zone and row can be ignored.
3. Draw lines connecting tick marks of equal value on the top and bottom margins of the map. This need be done only for the area of the map surrounding the position of interest. Connect tick marks on the vertical margins of the map in the same manner. This will form a grid of 1-km squares on the desired portion of the map.
4. Position the corner of the roamer scale or ruler at the "X." Measure from the "X" to the nearest false easting line to the left, and record the easting value of the "X." Measure to the nearest northing line below the "X" and record the northing value of the "X."
5. The unique position of point "X" is now known, and can serve as a reference point for subsequent navigation. This might include use as a GPS waypoint, to identify the position of a prominent landmark to be used in triangulation of bearings, or to label a known starting point for the next leg of the hike.

Once a degree of comfort is attained with UTM coordinates, navigation from point to point becomes much more intuitive than with latitude and longitude. Using the method just described, it is a simple matter to determine the bearing and the distance in meters between any two points on the map, to plot GPS waypoints on the map, or to identify a probable position based on dead reckoning from a known starting point. Note that it is standard for GPS receivers to be able to express the user's position in any of several formats, including as UTM coordinates. A location near the author's home has the geodetic coordinates of 39° 14.68′ north latitude, and 84° 29.5′ west longitude as determined by a commonly available, inexpensive GPS receiver. With several simple key presses, the grid reference is expressed in UTM format as:

16S 0716469
4346677

This indicates that the location is in UTM grid zone 16S (zone 16, row S). The meridians bounding zone 16 are 90° west longitude on the west, and 84° west longitude on the east. Row S is bounded by 32° north latitude to the south, and 40° north latitude to the north. The latitude and longitude boundaries of the identified zone and row include southwestern Ohio. The numbers specifying the unique location within the zone and row must be read, per UTM convention, to the right (easting) and then up (northing). The first seven-digit number indicates that the site of interest has a false easting value of 716469 m E (716,469 m east of the central meridian for the zone), or 469 m east of the 16-km tick mark on the horizontal margin of the relevant USGS map (see Figure 85-15). The second number indicates that the location has a northing of 4346677 m N (4,346,677 m north of the equator), or 323 m south of the 47-km tick mark on the vertical margin of the map.

CELESTIAL METHODS FOR DIRECTION FINDING

Shadow Methods

The axis of rotation of the earth is inclined at about 23.5° from the orbital plane of the solar system. It is this phenomenon that results in the seasons, in the variable path of the sun through the sky with each season, and in the terrestrial definitions of the tropics, the temperate zones, and Arctic and Antarctic zones. The apparent path of the sun is lower in the sky, and shadows cast by the sun are longer, at any given time of day in winter than in summer. A plot of the tips of the shadows cast in daylight by a vertical object (a gnomon) on any given date results in a curved line called a *declination curve*. The declination curve is so-named because shadow lengths are proportional to the declination of the sun on the date of the plot. The shadows used in plotting a declination curve are shortest at noon when the sun is at its greatest altitude, and longest near sunrise and sunset. A family of declination curves can be plotted empirically for various dates, and can then be used in subsequent years to predict shadow lengths for future dates and times. The shadows themselves can be plotted to indicate the direction of the sun, which gives the approximate time of day. This is the principle by which sundials were constructed before the discovery of trigonometry. A family of declination curves will be useful for any point on the earth's surface near the same latitude as the location where they were plotted. The curves will not be accurate at more distant latitudes because the altitude of the sun changes with the latitude of the observer. This lack of universality can be overcome by moving the gnomon to compensate for changes in latitude. Since the advent of plane and spherical trigonometry, declination curves and gnomon positions can be calculated for any date and location.[30]

The sundial is most commonly used to tell time, but also can be used as a sun compass to indicate direction. Shadows cast in the morning hours point westward, whereas those in the afternoon point eastward. The variation between the rising and setting points, and due east or west, are predictable for any date and latitude (see section on Direction by Amplitudes). The shortest shadow cast by the sun occurs at LAN and always lies on a due north–south line. Alternatively, a north–south line can be found by bisecting the angle between two points on a declination curve that are at equal distances from the gnomon. Knowing this, a sun compass can be constructed that indicates true direction with a high degree of accuracy whenever the shadow tip of the appropriately positioned gnomon lies on the declination curve for the approximate date of observation. Such a sun compass, called a *Universal Pocket Navigator* (UPN), with a scale to compensate for the latitude of the user, is shown

Latitude	Dec 22	Jan 22 Nov 22	Feb 22 Oct 22	Mar 22 Sep 22	Apr 22 Aug 22	May 22 Jul 22	Jun 22	Latitude
	SELECTOR CHART							
	NORTHERN HEMISPHERE							
0°	0-N	0-M	1-K	0-J	0-I	1-G	2-F	0°
10°	2-N	3-M	4-K	3-J	2-I	3-G	4-F	10°
20°	6-N	6-M	7-K	5-J	4-I	6-G	7-F	20°
30°	10-P	10-N	11-K	9-J	9-H	11-F	12-E	30°
40°	20-P	18-N	14-L	12-J	12-H	13-F	14-E	40°
50°	30-Q	29-Q	23-L	18-J	18-G	21-D	21-C	50°
60°	Wait for rescue party			25-J	25-F	28-B	29-A	60°
	Jun 22	Jul 22 May 22	Aug 22 Apr 22	Sep 22 Mar 22	Oct 22 Feb 22	Nov 22 Jan 22	Dec 22	
	SOUTHERN HEMISPHERE							
	In Southern Hemisphere, the arrow points to the south, and west (a.m.) is interchanged with east (p.m.)							

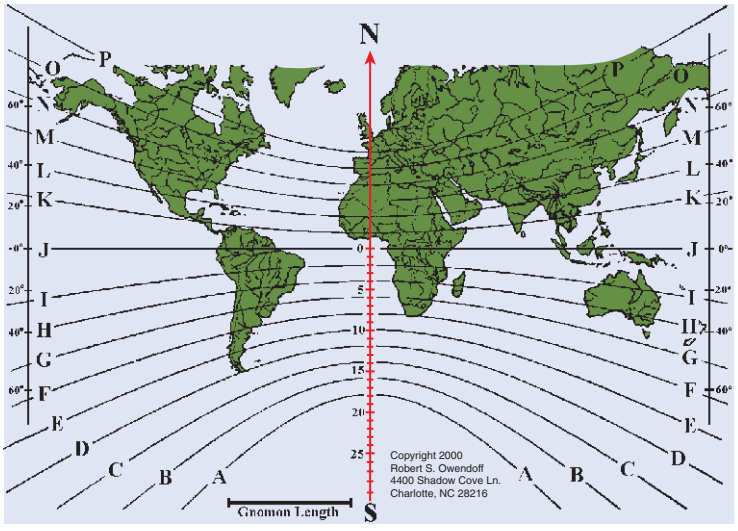

Figure 85-16. The Universal Pocket Navigator (UPN). Enter the Selector Table with arguments for the approximate date and the approximate latitude as determined from the background map. Stick a pin or toothpick vertically in the center line at the cross mark indicated by the Selector Table to serve as a gnomon. The tip of the gnomon should stand above the figure by the length indicated by the gnomon scale. Hold the figure horizontally, and rotate your body until the shadow of the tip of the gnomon lies on the appropriate lettered curve (from the Selector Table). The arrow now points north. (From Owendoff RS: Better Ways of Pathfinding. Harrisburg, PA, Stackpole, 1964, with permission.)

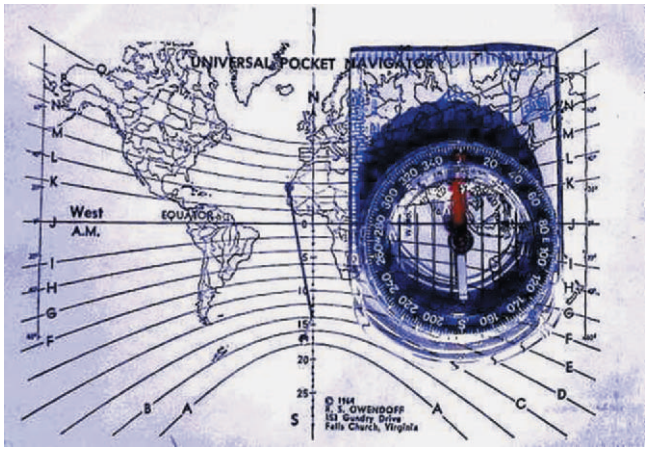

Figure 85-17. Use of the Universal Pocket Navigator (UPN) to find north. The selector chart was entered with the date of October 25 and the assumed latitude of 40°, yielding declination curve "L" and gnomon position "14." Note the correspondence between magnetic north and north as indicated by the center line of the figure. The magnetic declination at the sight of observation was 41/2° west. The UPN reading corresponds to true north.

in Figure 85-16.[30] A copy of this sun compass, a pin or toothpick greater than 1 inch (2.5 cm) in length, and sunshine are all that is necessary to determine the cardinal directions with an accuracy greater than that commonly achievable with a magnetic compass (Fig. 85-17).

In the absence of a copy of Figure 85-16, the same principle can be applied to construct a sun compass on the fly. Any flat surface (e.g., a chip of wood, piece of bark, scrap of paper) is placed horizontally in the sun and a gnomon of convenient length is stuck in the surface in a vertical position. The tip of the shadow cast by the gnomon is marked at various times beginning shortly after sunrise, and a curve is traced between the marks. At some point, the sun will pass the meridian, and the shadow will begin to lengthen again. A line between the base of the gnomon and the point of closest approach of the curve represents solar noon, and runs due north to due south. Whether the shadow at noon points north or south depends on whether the observer is in the northern hemisphere, the southern hemisphere, or the tropics. The actual direction indicated by the shadow is easily determined by noting the general direction of sunrise, which is on the eastward side of the sun compass. The remaining afternoon portion of the curve can either be completed freehand as a mirror image of the morning curve, or by continued observation and marking of shadow tips (Fig. 85-18). On subsequent days, the sun compass is reoriented to true north by rotating the horizontal surface until the tip of the gnomon's shadow touches the curve. A sun compass prepared in this manner is highly portable and remains accurate for many days until there is significant change in the latitude of the traveler, or in the declination of the sun. New curves can be prepared on the same compass as needed.

Other methods of direction finding by shadows exist but are significantly less accurate. The direction of travel of a shadow tip over the period of an hour or so will point generally eastward throughout the day.[4] The accuracy of this method is degraded in direct proportion to the length of time before or after noon that the observations take place, and in inverse proportion to the declination of the sun. Errors of greater than 30° are possible when conditions are unfavorable, such as at higher latitudes and during winter when the path of the sun is low in the sky. However, errors incurred during morning hours will tend to be canceled by reciprocal errors incurred during afternoon hours as long as the rate of change in latitude while

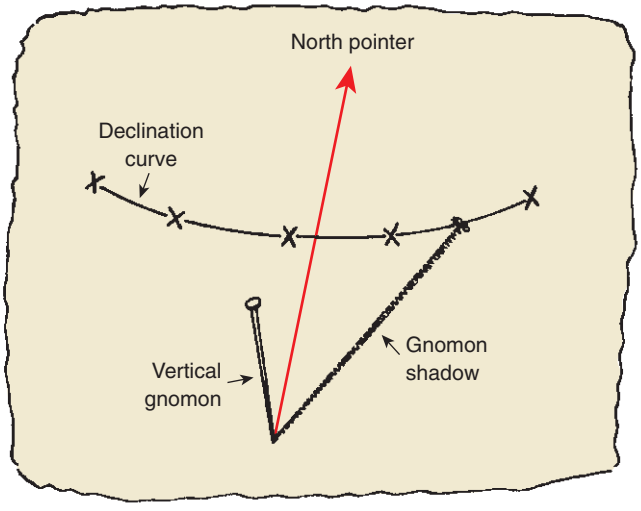

Figure 85-18. A makeshift sun compass inscribed on a scrap of paper. The declination curve has been empirically determined by tracing a line through points representing the tip of the gnomon shadow at various times throughout the day. On subsequent days, rotation of the figure such that the shadow tip touches the curve will orient the line connecting the base of the gnomon to the point of closest approach of the curve to point north. (From Owendoff RS: Better Ways of Pathfinding. Harrisburg, PA, Stackpole, 1964, with permission.)

traveling is relatively slow during the period of observation.[30] The shortest shadow cast by a vertical gnomon through the course of the day lies on a north–south line at all latitudes. The point of closest approach of a declination curve to the gnomon may be difficult to determine accurately when the rate of change of the sun's altitude around LAN is slow. As an alternative method, a line bisecting the angle between any two shadows of equal length cast by a vertical gnomon will lie on a north–south line at all latitudes.

An ordinary analog wristwatch or pocket watch can be used to give rough north–south direction using the shadow technique. Because a conventional watch has a 12-hour cycle of rotation, the hour hands rotates at 30° per hour, or twice the rate of the apparent angular movement of the sun. Thus, if the hour hand of the watch is aligned with the shadow of a vertical object (i.e., the hand is pointing directly at the sun), the bisected angle between the hour hand and 12 o'clock on the dial yields a line that runs generally north to south. At times before 0600 or after 1800, the larger of the two possible angles between the hour hand and 12 o'clock should be used. This method is fraught with errors that increase in magnitude when the sun's altitude is high (tropical or subtropical latitudes; late spring, summer, and early fall months). It is also subject to errors that result from differences between zone-time and local time, and from the *equation of time* (the difference between mean solar time and true solar time). However, the watch method can be useful as a direction finder if the potential for inaccuracy is kept in mind, and if techniques are used to reduce avoidable errors. Correspondence between direction determined by the watch method and true direction will be greatest between latitudes of 40° to 60° in the winter months. Accuracy is improved if the watch is set to local solar time at the approximate longitude of observation, rather than zone-time (local solar time = GMT + east longitude expressed in time; local solar time = GMT − west longitude expressed in time [where 15° of longitude = 1 hour]). Directional errors are further reduced by tilting the watch face to lie in the plane of the sun's apparent path, rather than in the horizontal plane.[10,30,32] A digital watch also may be used if the indicated time is drawn as an analog clock face on paper or in the dust.

Direction by Amplitudes

The *amplitude* of a heavenly body is the angular measurement between the body when on the horizon and the observer's prime vertical circle. When the body is rising, the amplitude is reckoned from due east; when the body is setting, the amplitude is reckoned from due west. The amplitude is designated as north or south, depending on the relative position of the body on the horizon and the prime vertical circle. The amplitude will always be north when the declination of the body is positive, and vice versa. For objects of constant declination (the "fixed stars"), amplitude is constant. For the sun, moon, and planets, amplitude varies with the seasonal variations in the declination of these bodies. Knowledge of the amplitude of the sun for various dates is useful as a means of determining direction at sunrise and sunset. Amplitude of the sun is zero on the dates of the spring and fall equinoxes, and the sun rises due east and sets due west of an observer at any latitude between the Arctic and Antarctic Circles on those dates.[2,7]

The formula for calculating the amplitude of any body at rising or setting (altitude = 0) is known as Napier's rule.[2,24,25]

Given latitude (L) and declination (d), solve for amplitude (A):

$$A = \sin^{-1} (\sin d / \cos L)$$

The maximum amplitude of the sun for a given latitude is calculated by entering the maximum declinations of the sun occurring at the solstices (±23.5°) into the formula.[1] The amplitude for any date is calculated by entering the declination of the sun on the date of interest. Note that directions as established by amplitudes are true, not magnetic, directions.

For most of the year in most of the world, the sun's amplitude lies within 30° of due east and west. In fall and winter in the northern hemisphere, the sun rises and sets south of east and west, whereas in the spring and summer the opposite is true. Maximum amplitudes occur at the solstices, whereas minimum amplitudes occur at the equinoxes.

A useful table of amplitudes for the sun or other selected celestial body can be easily calculated by Napier's rule and carried on an index card to provide a directional reference in the area of intended travel. The approximate latitude of the area of travel can be determined from a map. The declinations of the body to be used for the dates of travel can be located in the *Nautical Almanac* or from any of several websites. A ready-made table of amplitudes covering latitudes from 0° to 77°, and declinations from 0° to 24°, is available in Bowditch's *American Practical Navigator,*[2] Volume II, as Table 27.

Direction by Observation of Circumpolar Stars

Polaris provides the most reliable directional indicator in the night sky. Polaris can be used to indicate direction within a degree of true north at any location above 10° north latitude. Use of the star becomes progressively more difficult as its altitude increases above 60° north because of the difficulty in relat-

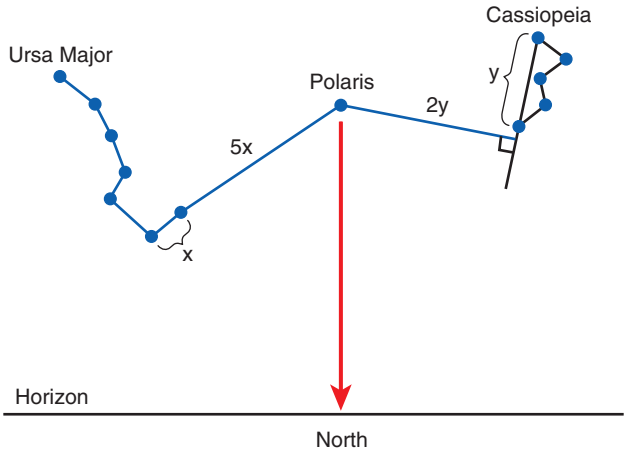

Figure 85-19. The azimuth of Polaris is always within 45 arc minutes of true north. Lines as indicated from the "Pointers" of the constellation Ursa Major, or from the trailing star of the constellation Cassiopeia, also can be used to find north when Polaris is obscured by clouds or below the horizon. (From Burch D: Emergency Navigation. Camden, ME, International Marine Publishing, 1986, with permission.)

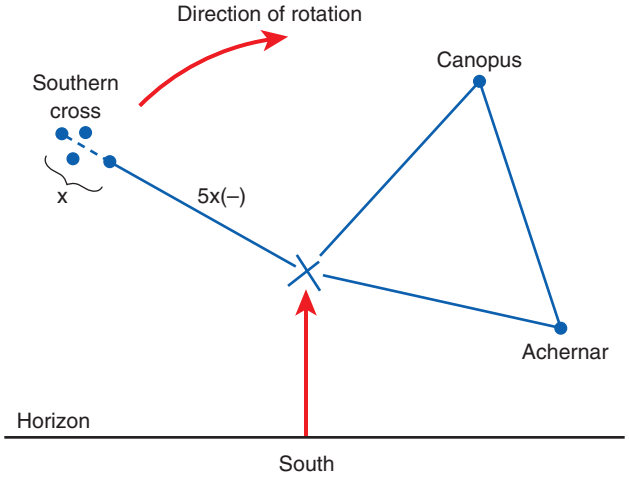

Figure 85-20. Determination of south by circumpolar stars. The long axis of the Southern Cross lies along an approximate radius extending from the south celestial pole at the distance indicated in the figure. The apex of an equilateral triangle with the stars Achernar and Canopus forming the vertices of the base also approximates the position of the south celestial pole. (From Burch D: Emergency Navigation. Camden, ME, International Marine Publishing, 1986, with permission.)

ing the star's azimuth to the horizon. A stick with a string tied to the end and weighted with a bolt or washer can be used to find the point on the horizon representing north in this setting. The stick is held such that the string hangs in a line from Polaris to the horizon. The point of intersection of the string with the horizon indicates north.[3]

The Big Dipper (Ursa Major) can be used to identify Polaris or true north by extending a line from the "Pointers" (α Ursae Majoris, or Dubhe, and β Ursae Majoris, or Merak) toward the north celestial pole. These stars form the leading edge of the Dipper. The distance between them multiplied by five indicates the approximate position of Polaris. When the Dipper is low in the sky or below the horizon, a similar process can be followed in identifying north from the constellation Cassiopeia. Cassiopeia has the appearance of a flattened letter "M" when above the pole, and "W" when below. If a line drawn between the stars forming the feet of the "M" (β Cassiopeiae, or Caph, and ε Cassiopeiae) is assigned length *y*, a perpendicular line of length 2*y* extending from the trailing star indicates approximate north[4] (Fig. 85-19).

In the southern hemisphere, there is no conspicuous star that marks the south celestial pole. However, the distinctive asterism of the Southern Cross can be used to point to the approximate location of the pole and thus to indicate south. The Southern Cross consists of four stars. The declination of the crossbar is approximately –60°. The long axis of the cross lies on a line that passes within 3° of the south celestial pole. The distance to the approximate pole from the star forming the base of the cross (α Crucis, or Acrux) is approximately five times the length of the long axis of the cross (Fig. 85-20). At latitudes where the cross is visible but the south celestial pole is below the horizon, the long axis of the Southern Cross indicates south when the constellation is vertically oriented.[3,4,33]

When the Southern Cross is below the horizon or too low in the sky for reliable observation, the bright stars Canopus (α Carinae) and Achernar (α Eridani) can be used to find south. If a line between these stars is considered to represent the base of an equilateral triangle, the apex of the triangle points to the approximate location of the south celestial pole[3,4] (see Figure 85-20).

Direction by Observation of Other Stars

It should be clear from the amplitude formula that any object with a known declination of less than ±5° could be used to give a reasonably accurate indication of east at the time of rising, or west at the time of setting. By virtue of its brightness and of the familiarity of the constellation in which it is located, the star δ Orionis (Mintaka; declination –0°, 18′) is particularly useful in this regard. Mintaka is the leading star in the belt of the constellation Orion. It rises due east and sets due west at any latitude from which it is observed[11] (Fig. 85-21). Unfortunately, the visibility of Mintaka at rising or setting is limited to the months of October through April. When the star is obscured by haze or by clouds at rising, or the time of rising is missed, the location of the rising can be extrapolated. Within 1 to 2 hours of the rising time, hold a straightedge connecting the star to the horizon at the rising angle of the star, where rising angle = 90° minus the latitude. The point where the straightedge touches the horizon indicates the position the star occupied when on the horizon. Because the rising and setting angles of any body are the same, the same technique can be used to determine the point on the horizon where the star will set.[3]

The constellation Scorpius is prominent in the southern sky at mid-northern latitudes during the summer. This constellation contains a distinctive reddish star, Antares (α Scorpii), at the position of the neck of the scorpion. To the east of Antares, the tail of the scorpion hangs toward the horizon. Three stars just before the sharp bend in the tail of the figure (ε, μ and ζ Scorpii) form a nearly straight line. The stars comprising the head and claws of the figure (β, δ [Dschubba], and π Scorpii) lie in a fairly straight line located immediately to the west of Antares. The configuration of the constellation is such that the stars forming

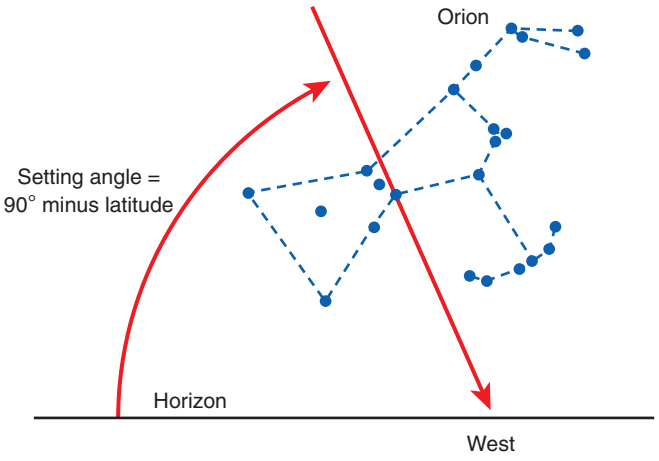

Figure 85-21. Determination of true west by observation of the setting point of the star Mintaka (declination zero) in the belt of the constellation Orion. The rising point of the same star indicates true east. (From Burch D: Emergency Navigation. Camden, ME, International Marine Publishing, 1986, with permission.)

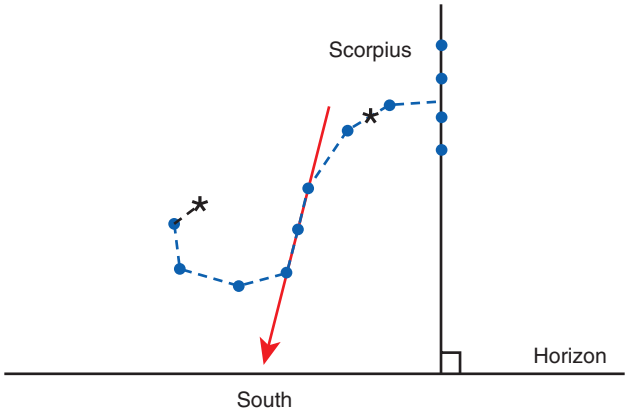

Figure 85-22. Determination of south by observation of the constellation Scorpius. When the best-fit line connecting the stars forming the head and claws of the figure is perpendicular to the horizon, the linear array of stars in the tail of the asterism points south. (From Burch D: Emergency Navigation. Camden, ME, International Marine Publishing, 1986, with permission.)

the linear array of the tail point due south when the line of the head and claws has passed the meridian and is perpendicular to the horizon[3] (Fig. 85-22).

In the Carolinas Islands of the South Pacific, the bearings at rising and setting of 32 prominent stars are memorized by navigators to permit determination of direction at sea. The fidelity of this "star compass" for direction finding is demonstrated by the ability of these navigators to make successful landfall on minute atolls after open-ocean voyages of hundreds of miles.[12] Memorization or recording of the rising or setting azimuths of a few prominent stars at the latitude of a planned trip will afford the same directional reference to the land navigator in case the compass or other navigational aid is lost or damaged. These azimuths could be precalculated in minutes using Napier's rule.

Finally, observation of the movement of any overhead celestial body relative to a fixed reference (e.g., a branch, guy line) over a span of 15 to 20 minutes (4° to 5° of movement) will give a reasonably accurate indication of east and west.

Practical Field-Expedient Celestial Navigation

The following discussion demonstrates the practical use of the methods just described on a hypothetical trip to one of the most remote areas in the 48 continental United States. Before leaving on a trip to the Florida Everglades in mid-May, a traveler consults the map for the intended area of travel, noting the approximate latitude (25° north) of the area in question. The traveler also consults the *Nautical Almanac*, Bowditch's *American Practical Navigator,*[2] or a website to obtain the approximate declination of the sun (18.5° north) during the week of intended travel. By applying Napier's rule and using a calculator, or by consulting Bowditch,[2] the traveler notes that the sun will rise approximately 20° north of due east, and set 20° north of due west during the trip. This information is recorded on a 3″ × 5″ index card. The traveler then checks the same sources to see which easily recognized stars will be visible after sunset, near midnight, or before sunrise during the planned dates of the excursion. In this case, the traveler notes that Polaris will be visible all night, and that the constellation Scorpius (see Figure 85-22) will be prominent, and almost due south, just before 1 AM. A prominent red star in Scorpius, Antares, rises shortly after twilight. Antares has a declination of 26° south. By the formula or table, the amplitude of Antares at rising will thus be approximately 29°, and the star will rise at a bearing of about 119° and set at a bearing of about 251°. This information is also recorded on the index card. On the morning of arrival at the destination latitude, the traveler places the index card on a flat surface in sunlight and sticks a pin into the card. A sun compass is constructed by the method described earlier (see Figure 85-18). The card and pin are then placed in a pocket.

The traveler paddles to a remote location in the park on a cloudy day and travels further than anticipated because of an unusually high tide. He suffers a spill from the canoe en route, and while in the middle of the Everglades becomes stranded when the tide recedes. He discovers that his compass and map are missing, and that his watch is broken, but the index card is still in his pocket. The weather worsens and the traveler loses all directional reference and is forced to camp. That evening, the eastern horizon clears and Antares is observed rising at low altitude. The traveler retrieves the index card, recalls the rising amplitude of the star, and scratches a line pointing toward the rising point in the dirt and labels it "119°." He then draws a complete compass rose in the dirt, and labels all of the cardinal directions. In the morning, the sky is obscured, but the traveler recalls that a highway crosses the Everglades to the south-southeast. The drawn compass rose is consulted, and a small rise is noted on the horizon south of his position. Through a combination of paddling and dragging the canoe, the traveler moves toward the rise but is forced well to the west of its position by the terrain, and it is lost from view after several hours. The traveler continues moving southward as judged by the position of the brightest area of cloud, presumably representing the position of the sun. Several hours later, the sun breaks though the clouds, and the traveler retrieves the index card, pins it flat to the seat of the canoe with the pin, and rotates the card until the shadow tip touches the curve. The cardinal directions are now re-established, and the traveler proceeds southeast as indi-

cated by the sun compass. As the weather worsens, the traveler again identifies a landmark on the southern horizon and moves toward it after the shadow is lost. As night falls, the clouds thin on the western horizon, the setting sun is observed, and the cardinal directions are again established by the amplitude method. Travel progresses to the southeast. That night, the northern sky transiently clears enough for Polaris to be identified. The traveler continues southward away from the direction of the star, but becomes disoriented. During a rest period, the traveler lies on the ground and observes the movement of overhead stars for 15 minutes through a break in the overcast, reacquiring the cardinal directions. He continues southward and notices lights in the direction of travel, reaches the highway, and is rescued by motorists.

The successful outcome in this example resulted from preparation in advance of travel, and familiarity with the navigational clues offered by the environment. Being lost in the middle of a desert or in mountainous terrain in winter may represent a more desperate situation than that just described, but the methods used for recovery would be identical if sophisticated navigational aids were unavailable.

NAVIGATION WITH A POCKET RADIO

Radio transmission and reception are inherently directional. This is particularly evident with reception in the broadcast band (500 to 1600 kHz) and is a principle that is more apparent in inexpensive portable radios than in larger, more expensive models. This method of navigation was used extensively in aviation from the 1930s through the 1950s. There are anecdotal reports of successful sailings from the west coast of the United States to Hawaii using only an AM radio and jet contrails as navigational aids.[23]

The internal antenna of the typical pocket AM radio is formed by a ferrite bar wrapped in multiple loops of copper wire. This ferrite-loop antenna responds most strongly to radio waves when oriented perpendicular to them. Reception is minimized when the antenna is oriented parallel to the transmitted radio waves (i.e., pointed at the transmission tower of the station). The point of minimum radio reception, or *null point*, can be used to find the direction to a source of radio transmission with surprising precision.[3,32] Use of this technique assumes that a broadcast band station is audible, and that the geographic location of the radio transmission can be identified by listening to the station. Once these conditions are met, the procedure for establishing a direction line that runs through the broadcast source and the observer is relatively easy. Radio homing does not determine true direction, but can identify the direction to safety. When used in conjunction with a map, radio bearings act as LOPs, and multiple bearings to different stations can result in a fix. The procedure for navigation by radio bearings follows:

1. If not known, determine the orientation of the radio's internal antenna. This can be done by opening the case and looking for the antenna (a dark gray or black bar wrapped in fine copper wire), or by rotating the radio while listening to a station and determining the plane of rotation that results in a null point (rotation about the long axis of the antenna will not change the reception, while rotation perpendicular to the long axis will result in nulling).
2. Select an audible AM station and listen until the location of the station is identified. Hold the radio in a manner that allows the long axis of the internal antenna to act as a pointer. Rotate the radio parallel to the ground until the radio signal becomes faintest or disappears. Nulling of the signal can be augmented by tuning to the fringes of the station frequency if the reception is strong.
3. The actual direction to the source of radio transmission will be one of the reciprocal bearings of the LOP connecting the observer and the station location. The bearing that actually points to the radio source can be determined if the observer has even a crude understanding of his or her position relative to the station (e.g., generally north of, versus generally south of). The bearing to the broadcast source also can be determined by serially checking the null point of the station while traveling. The angle over which nulling occurs becomes greater as the signal weakens with increasing distance from the source. Thus, if the angle over which the signal nulls is widening as travel progresses, the observer is moving away from the radio source. If the angle over which the station nulls narrows as travel progresses, the observer is moving toward the source.
4. If a compass is available, determine the azimuth of the null point. Keep the compass far enough from the radio to avoid influencing the needle with the radio's metal parts. A compass allows the magnetic bearing to the station to be determined. This bearing can then be followed to safety without further use of the radio.
5. If a map and compass are available, orient the map using the compass and draw a line though the broadcast source on the map at the azimuth of the null point. Extend this line to the edges of the map. Your location is somewhere on or near this line. If you have a map but no compass, and can properly orient the map by natural cues, place the radio on top of the broadcast location on the map and rotate it until the signal nulls. Draw a line parallel to the internal antenna through the broadcast location and extend it to the map margins. Again, your position lies somewhere on this line. When two (or more) stations can be tuned and identified, the LOPs connecting the observer to each radio source will cross, and a fix is obtained at the point of crossing. The uncertainty of this fix will be least when the angle between two position lines is close to 90°, or when the angles between three position lines are close to 120°.

NAVIGATION WITH THE GLOBAL POSITIONING SYSTEM

The NAVSTAR Global Positioning System (GPS) exploits the logical framework of celestial navigation in using a predictable extraterrestrial reference for the determination of terrestrial position. In GPS navigation, artificial satellites substitute for celestial bodies as reference points. Calculation of position is based on *circles of equal distance* from the satellites.

The current system consists of a constellation of satellites arrayed in six orbital planes, with four functioning satellites per orbital plane. The orbital planes are inclined to the earth's equator by 55°. The orbital paths of the satellites are nearly circular and have an altitude of approximately 20,000 km with an orbital period of 11 hours, 58 minutes. At any given time, five

to eight satellites are available in line of sight to a receiver anywhere on the surface of the earth.[8,17]

The method of position determination using GPS depends on calculation of the range between the satellite and receiver. GPS signals are transmitted on two L-band frequencies by each satellite: L1 (1575.4 MHz) and L2 (1227.6 MHz).[6] Transmitted information includes the precise time as kept on board the satellite by multiple atomic clocks, a satellite ephemeris (catalog of predicted positions), and data concerning corrections for atmospheric propagation of radio signals and satellite clock errors. The GPS receiver decodes the positional data for each satellite and compares the timing information transmitted by the satellite with time as kept by the receiver's onboard clock. The signal received is in the form of a pseudorandom code. The GPS receiver shifts the code contained in memory to match the received code in phase. The duration of the shift represents the time required for the satellite signal to reach the receiver, usually on the order of 50 to 60 msec. Because distance = speed × time, the transit time of the signal allows a calculation of the distance between receiver and satellite.[14] At any given instant, a GPS receiver in contact with a NAVSTAR satellite will lie on the surface of a sphere of equal distance from the satellite. The intersection of this sphere of equal distance with the surface of the earth forms a circle of equal distance, analogous to the circle of equal altitude in celestial navigation. The intersection of two such circles occurs at two points. The intersection of three such circles occurs at a single point on the surface[8,17,18,28] (Fig. 85-23). This is the position of the receiver. If the intersection of the sphere of equal distance of a fourth satellite is added, the approximate altitude of the receiver can be determined. Software allows the GPS receiver to choose the optimal group of four satellites for position determination among the subset of satellites within line of sight of the receiver.

As originally configured, the GPS system provided two levels of service: the Standard Positioning Service (SPS) and the Precise Positioning Service (PPS). SPS provided civilian users with positional accuracy to 100 m 95% to 98% of the time, to 50 m 65% of the time, and to 40 m 50% of the time. Altitude accuracy was relatively poor at 150 m. The intentional inaccuracy of the SPS system resulted from the introduction of timing errors into the broadcast signal from the satellites on frequency L1. This degradation of precise data from the satellites is termed selective availability (SA), and is controllable from the ground. SA was formally implemented 1 year after public availability of GPS signals was granted in 1994, and was discontinued in 1999. There are no immediate plans for reimplementation, and PPS is the standard level of service available to military and civilian users at the time of this writing. PPS provides positional accuracy of 15 m, velocity accuracy of 0.1 m/sec, and time accuracy of 100 nanoseconds.

To comply with the requirements for safety-of-life aviation applications, the Wide Area Augmentation System (WAAS) was developed in cooperation with the Federal Aviation Administration (FAA) and Department of Transportation (DOT). WAAS consists of 25 North American ground stations that monitor and correct errors in the GPS satellite signals caused by ionospheric interference, orbital drift, and clock errors. Corrections are transmitted on the L1 frequency by geostationary satellites and can be received by enabled receivers. WAAS was implemented for non–safety-of-life applications in 2000, and for aviation safety-of-life applications in 2003. WAAS-enabled, civilian GPS receivers allow less than 3-m horizontal and vertical positional accuracy 95% of the time, with 7-m accuracy the remainder of the time.[9] The system is scheduled for augmentation over the course of the next decade that may permit even greater recreational accuracy. Currently, WAAS is available only in North America.

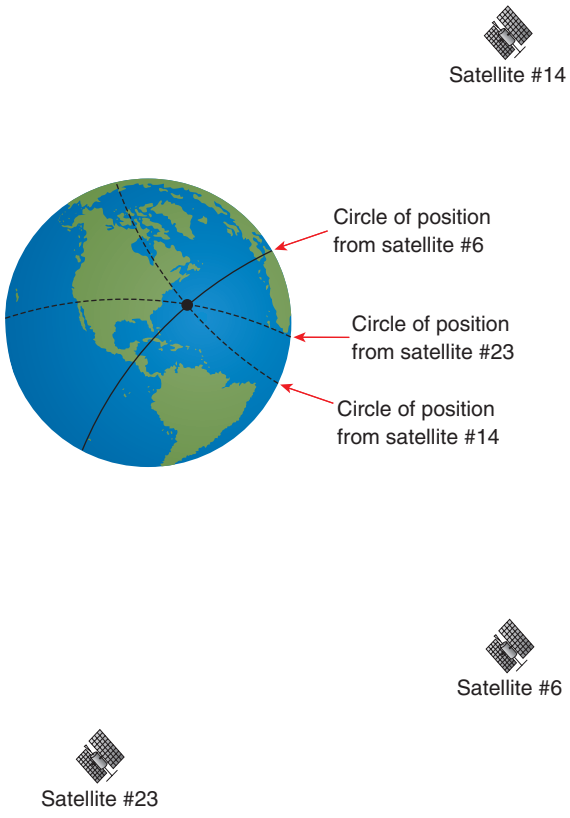

Figure 85-23. Illustration of a Global Positioning System (GPS) fix from the intersection of three circles of equal distance from three separate satellites. (From Monahan K, Douglass D: GPS Instant Navigation. Bishop, CA, Fine Edge Productions, 1998, with permission.)

A Soviet-era Russian system, GLONASS (Global Navigation Satellite System), also exists. GLONASS has marginally better satellite coverage in higher northern and southern latitudes (>50°). Positional accuracy is 30 m. GLONASS receivers are not widely available, however, and the system offers no compelling advantages over GPS for the surface navigator. The European Union (EU) is currently developing another satellite navigation system, Galileo.

The GPS system reaches its greatest utility when used in conjunction with a topographic map and compass. The raw output of the receiver is in latitude and longitude to the nearest second of arc, or in UTM grid coordinates to the nearest meter. This information is somewhat abstract in isolation, but software included with the receiver permits the user to understand absolute and relative position and perform sophisticated navigational feats, even in the absence of other aids to navigation. When used with a map, GPS allows the user to plot position and route at will, determine bearings to landmarks, and enter the locations of landmarks into memory for use as waypoints. Even without a map, GPS allows the user to determine precise

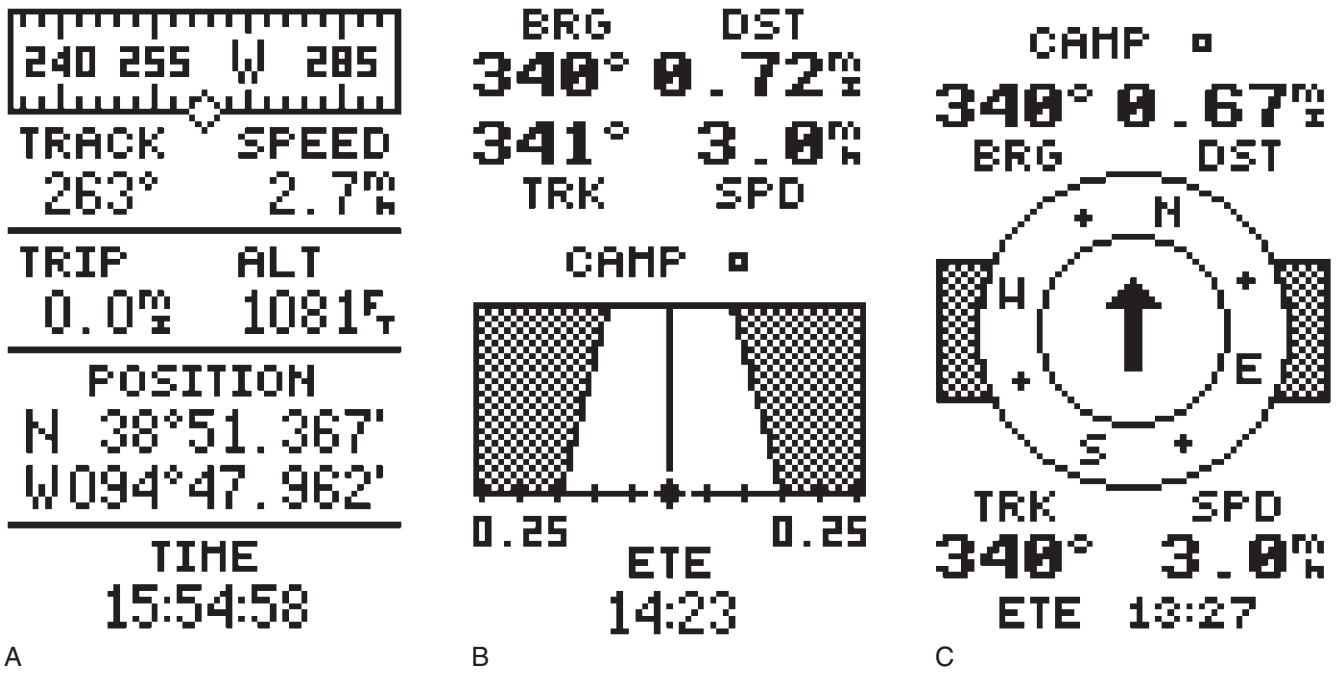

Figure 85-24. Screen images from a common portable Global Positioning System (GPS) receiver showing various navigation screens. Screen A displays the time, the terrestrial coordinates and altitude of the observer's position, and the direction and velocity of travel. Screen B shows the bearing and distance to a selected waypoint (CAMP), the track made good by the observer, the velocity of travel, and a graphic representation of deviation from the intended course. Screen C shows the distance and bearing to the selected waypoint in compass format. (Courtesy of Garmin International, Olathe, KS.)

position, approximate altitude, bearing and linear distance to established waypoints, deviation from the intended course to a waypoint, and velocity of travel.

Waypoint navigation is a feature of particular utility in wilderness navigation. Waypoints can be entered into the GPS receiver manually by a keypad, or current position can be entered and stored as a waypoint with a single keystroke. Groups of waypoints can be stored as a route. As a course is walked, waypoints can be stored and labeled with reference to a prominent landmark or terrain feature. On the return route, the receiver sequentially gives the bearing and distance to any waypoint in the stored route, and displays a graphic representation of the deviation of the current course from the course needed to reach the waypoint (Fig. 85-24). The receiver informs the user that a waypoint has been reached by sounding an alarm. Theoretically, this allows a user to follow a route in whiteout conditions or in total darkness, using no visual references other than the display of the GPS receiver. Most current receivers have memory capacity sufficient for the storage of several hundred waypoints. Identification of a large number of waypoints on a complex route essentially allows the user to follow a "breadcrumb route" on return to their objective.[27]

All receivers can calculate the heading of travel, and thus provide the user with a method of determining a bearing and the cardinal directions by walking a brief, straight course. Bearings can be expressed as either true or magnetic at the preference of the user by toggling a software switch. Speed of travel is displayed, and estimated time en route at the current speed is given. Simple moving maps displaying the route traveled are available on even the least expensive GPS receivers, and permit the user to "walk the line" of a route on the receiver's graphic display (Fig. 85-25). Many units incorporate low-resolution base maps with a resolution of kilometers. Some models permit the download and storage of high-resolution topographic maps in random access memory, and display the user's position on the map image. When applied with common sense and a routine awareness of approximate location, even the simplest GPS receiver renders it virtually impossible to become lost, and effectively eliminates much of the pathfinding challenge inherent in wilderness travel.

In practice, the GPS should be applied to a wilderness trek in the following manner: before embarking on a trip, the user should chose a terrestrial coordinate system (UTM versus latitude and longitude), and then enter into the receiver the precise coordinates of various important landmarks on the intended route of travel. For reasons articulated earlier, UTM coordinates offer a substantive benefit in land navigation compared with the traditional geodetic coordinate system. The locations to be entered can be obtained from a trail guide or from a USGS topographic map. The waypoints obtained in this manner are labeled and stored as a route in the receiver's memory. At the beginning of the trip, the location of the nearest town or source of assistance also is entered as a waypoint, as would the location of the trailhead where the trip is begun. The trip then progresses using map, compass, established trails, or the GPS receiver to follow the intended route. When pausing to camp, the position of the camp would be named and entered as yet another waypoint. At any time, the bearing and distance to any waypoint of interest is available to the user. If, in a spasm of self-reliance, the user decides to lay aside the GPS receiver and pursue tradi-

Figure 85-25. Photograph of a moving map image from a typical Global Positioning System (GPS) receiver showing a "breadcrumb" route of plotted points acquired during a hike. Note the directional reference arrow indicating north in the left upper corner, and the map scale showing a 300-m bar at the bottom left. The route contains four waypoints of known coordinates. The position of the user is indicated by the "?" symbol. In poor-visibility conditions, the user could walk back along the indicated route to reach each waypoint in sequence. The receiver can also display the bearing and direction to any waypoint, and indicate the deviation from that bearing line as the user walks toward the waypoint.

tional methods of navigation and he or she becomes lost, reactivation of the receiver will allow the direction and distance to safety to be immediately determined.

Highly capable GPS receivers, including WAAS-enabled receivers, are commonly available at prices of less than $100, fit into a shirt pocket, and have sufficient battery power for weeks of navigation if used judiciously. Models that incorporate full-color, topographic maps can be purchased for about $500. Still, GPS suffers from several limitations. Most receivers have a battery life of between 12 and 30 hours. Dead batteries yield a useless receiver. The receivers are relatively fragile, and many are not waterproof or even particularly water resistant. Obstruction of the sky by terrain features or heavy foliage interferes with the reception of satellite signals, and may render the receiver unable to acquire a sufficient number of satellites to provide a fix. In an era of national security risk, SA could be reimplemented or WAAS could be restricted, with resultant degradation of the accuracy of recreational GPS navigation. Still, GPS is unsurpassed in ease of use, accuracy, and utility for wilderness navigation. Reliance on GPS as the sole navigational resource for any wilderness expedition is a grave error, however. As with all high-tech methodology, a GPS receiver can be easily disabled. The more self-contained methods of navigation discussed in earlier sections should be used whenever possible to maintain positional awareness and navigational skill in anticipation of the possibility that GPS might fail.

ORIENTEERING AND GEOCACHING

Orienteering is a competitive sport in which an unfamiliar course is navigated using map and compass. Most commonly, participants are required to find a number of *controls*, or identified sites, in a set sequence (point-to-point orienteering). In a variant, participants have a fixed time to visit as many controls as possible in any sequence (score orienteering).[1,21] Orienteering is usually practiced as a cross-country footrace over a distance of kilometers or miles, but shorter courses can be set up in paved or inhabited areas for participants of differing abilities. Appropriately constructed courses can be negotiated on foot, on skis, or by bicycle or wheelchair.

Orienteering had its genesis in military land navigation exercises. The term *orienteering* was coined in Sweden in 1900 to describe a ski relay race over a distance of greater than 100 miles. By the close of World War I, orienteering clubs conducting competitive meets were well established in Scandinavia. The sport rapidly gained popularity in Europe, and was introduced in North America in the late 1940s. By the 1970s, an International Orienteering Federation (IOF) was established to codify standards and rules.[1] Orienteering is now practiced by many thousands of people on all continents.

Orienteering has spawned several offshoot sports. *Rogaining* is a team form of score orienteering practiced over a 24- to 48-hour period, requiring overnight camping as a component. Participants set out from a central base (the "hash house") to find as many controls as possible over the allotted time span. Return to the hash house for meals, rest, refreshment, or socializing is permitted. *Adventure racing* is a form of team orienteering practiced over long distances and time spans of days to weeks, and may involve running, hiking, biking, and canoeing as components.[21]

The Orienteering Meet

Orienteering meets are usually sponsored by local clubs operating under guidelines established by the IOF, United States Orienteering Federation (USOF), or other national federations. Events may be advertised in local newspapers or on websites maintained by the sponsoring clubs. Most events offer multiple courses to accommodate participants of varying skill and fitness levels. An internationally standardized color coding identifies the technical difficulty of each course[31] (Table 85-1).

The only required equipment for competitive orienteering is an orienteering compass, a pencil or pen, and appropriate athletic clothing and shoes. Participants register for a course, pay a fee, and receive a start time, a course map, a printed description of the controls along the course (i.e., "at bend in creek"), and a control card that will be marked with a coded hole-punch attached to each control point. The map displays magnetic meridians, eliminating the need to correct for declination. The map may also have the controls and lines connecting them printed on it (Fig. 85-26), or participants may be required to copy these features onto their map from a master map.

Several minutes before the appointed start time, the participant reports to the starting line. The starter tears a stub from the control card to allow a tally of participants on the course. The participant orients the map to the environment using the

TABLE 85-1. Color-Coded Events—Standards for Competitors

COLOR	LENGTH (km)	CONTROL SITES	TYPE OF LEG	TECHNICAL LEVEL	TIME (min)	AGE (yr)
String	0.5–1.0	On the line	Along the string	Easy	10–15	3+
White	1.0–1.5	On major line features and at junctions	Along line features, no route choice	Easy	15–40	6–12
Yellow	1.0–2.5	On line features and at easy adjacent features	Along line features, minimal route choice, no compass legs	Easy	20–45	8+
Orange	2.0–3.5	On minor line and easy point features	Route choice with compass legs and collecting features near controls	Medium	35–55	10+
Red	4.5–6.0	Same	Same	Medium	50–80	10+
Green	3.5–4.5	At small point and contour features	Fine compass and contour legs, more physical	Hard	25–55	10+
Blue	4.5–6.5	Same	Same	Hard	50–75	10+
Brown	6.5+	Same	Same	Hard	60–85	10+

Modified from Renfrew T: Orienteering. Champaign, IL, Human Kinetics, 1997, p 27, with permission.

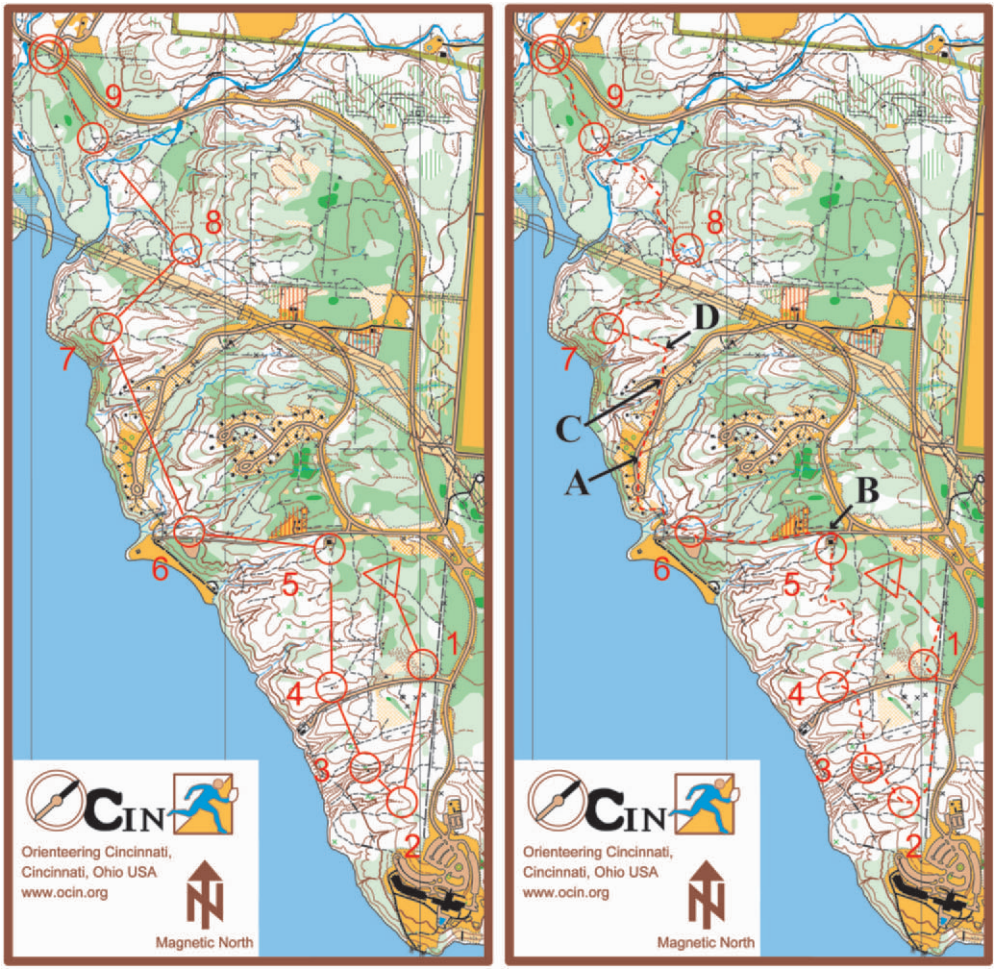

Figure 85-26. Paired orienteering maps for a typical Yellow-level course showing the starting point as a triangle, the control points as numbered circles, and the finish point as a double circle. Note the magnetic meridians printed on the map as a directional reference. In the left-hand map, bearing lines connect the controls but may not represent the fastest, least strenuous, or safest routes of travel. The actual route followed is shown in the right-hand map. Note the use as a handrail of the road segment **(A)** between Controls 6 and 7. The road immediately to the north of Control 5 **(B)** represents a collecting feature should the participant overrun the control. The branch in the road between Controls 6 and 7 **(C)** forms an attack point for reaching the path that leads to Control 7. On this leg, the participant deliberately aimed off to meet the path at a point known to be east of the target **(D)** so that the path could be followed in the proper direction toward the objective. (Courtesy of Michael Minium, Orienteering Cincinnati, Inc., and United States Orienteering Federation, 2005. Used with permission.)

compass and visual landmarks and starts the course when told by the starter. The starter records the starting time with the participant's name or number for comparison with the finish time and determination of elapsed time to run the course. When running the course, the participant continually reorients the map to the environment using the compass, and the environment to the map by comparing visual cues with map features. A relatively direct, or circuitous, route between control points is chosen by considering terrain features and the presence or absence of readily identifiable landmarks that may assist in navigation. As the participant approaches the area of a control, the area is scanned for descriptive features and the orange and white flag that identifies the site, and the control point is located and gained. The participant then punches his or her control card with the punch attached to the control as proof of having been there, then reorients the map and progresses to the next control in sequence. This process is repeated until the course is competed. As the participant crosses the finish line, the time is recorded and the number on the control card is checked against the stub that was collected by the starter. The card is then inspected to establish that all of the control points were visited, and the course time is calculated and recorded. At the completion of the meet the winners for each course are determined and announced, and prizes may be awarded.[1,31]

Several concepts that receive particular emphasis in orienteering are extremely useful for general map and compass navigation.[22,31] A *handrail* is a linear feature on the map that can be easily recognized in the terrain, and used as a guide to an objective. Typical handrails include paths or roads, streams, fences, shorelines, firebreaks, ridgelines, or abrupt contour changes. Once a handrail is encountered, the navigator can move along it with speed until the chosen route deviates from it, or the objective is encountered. An *attack point* is a prominent and obvious feature in the terrain from which an objective can be easily located. This may be a knoll, a bend in a path, a narrowing in a stream, or other point feature. An attack point can be chosen in advance of travel, allowing the bearing and distance from the point to the objective to be calculated in advance. Once the attack point is reached, discovery of the ultimate objective is greatly simplified. A *collecting feature* is an easily recognized terrain feature that lies beyond a desired objective. If the collecting feature is encountered, the navigator knows that the target has been passed, and can retrace his steps to the last known position for a second attempt on the objective. *Aiming off* is a technique for reducing confusion when navigating toward a particular point that lies on handrail. A common problem in land navigation involves encountering such a linear feature, then not knowing which way to turn to follow the handrail to the objective. Aiming off involves introducing a deliberate error into the bearing to the handrail, such that it will be encountered at a point that is absolutely known to be left or right of the intended target. The navigator then follows the handrail in the direction that corrects the error, and is certain to lead to the objective (see Figure 85-26).

Handrails, collecting features, attack points, and opportunities for aiming off should be determined in advance of travel by studying the map and the visible terrain. This permits selection of a route that facilitates speed, safety, or ease of travel. These features should be recorded whenever possible, rather than trusted to memory. Critical evaluation of the map for such features greatly increases the navigator's awareness of terrain and competence in map reading.

Orienteering for Children

Successful orienteering depends on an ability to apply basic land navigation skills with speed and accuracy. The skills required for completion of even simple orienteering courses are completely translatable to following a wilderness route. Note in Table 85-1 that *String* courses, in which participants literally follow a string, ribbon, or length of yarn strung from one control to the next, are available as an introduction for the very young. Courses classified as *White, Yellow,* and *Orange* are also of a length and technical level that are appropriate for children. As such, orienteering offers perhaps the most reliable and accessible avenue for teaching map and compass skills to children. Map reading, orientation of the map with the compass, orientation of the map to the environment, route selection, terrain recognition, and outdoor safety are all concepts that are compatible with training in a classroom or backyard setting. These skills can then be applied in any large indoor space, or at a local park.

Most orienteering clubs offer programs for teaching children. The USOF sponsors a four-step program to foster progressive mastery of necessary skills in a logical sequence.[35,36] The program begins at the *Little Troll* level using String courses. Participants complete five USOF-sponsored courses with adult assistance. At the conclusion of each course, the child receives a sticker that he or she affixes to a special card. The completed card is redeemed with the USOF for a Little Troll patch. The child then progresses to the *Chipmunk* level. As Chipmunks, participants learn to be more comfortable in an outdoor setting, and are introduced to map symbols, the concept of control sites, and the procedures of running a course. After completion of five USOF-sponsored White courses with adult assistance, the child receives a Chipmunk patch and advances to become a *Rabbit.* At this level, knowledge of map symbols and colors is expanded, safety awareness is increased, and the child learns to orient the map to the terrain, mark his or her current location, select routes to follow, and follow a path between controls. Some adult assistance continues at this level. After completion of seven sponsored White courses, the child receives a Rabbit patch and advances to training as a *Roadrunner.* The training at this level occurs without direct adult assistance, although an adult follows the child on each course. Roadrunners are required to know safety rules, and learn to adjust the orientation of the map as they cover the course, make independent route selections, and orienteer in sequence from control to control. After completion of another seven White courses, the child receives the Roadrunner patch and progresses to solo orienteering.[36] The IOF supports a similar progressive training program for children called the *Swedish Step System.*[31]

In orienteering, correction for declination is not required because the maps are prepared with reference to magnetic meridians, creating internal consistency between the map and compass. Separate training in the conversion of magnetic and true bearings is required to allow the practice of safe and comprehensive map and compass navigation.

Geocaching

The sports of orienteering and rogaining do not provide any training in GPS use and, in fact, prohibit the use of GPS technology. However, another recently developed sport, *geocaching,* offers an excellent opportunity for training children and inexperienced adults in the use of terrestrial coordinates and GPS receivers. Geocaching is essentially a treasure hunt in a chal-

lenging environment. The sponsors of a cache leave a container of objects in a concealed location identified only by latitude and longitude. Locations of caches, relevant maps, and location descriptions are published on websites maintained by cache sponsors. Persons seeking the cache use the provided information and a GPS receiver to locate the site. Once located, the participant removes an item from the cache, replacing it with another token item. The visit to the cache is recorded on an enclosed logbook, and the cache is replaced. Note that the cache may be located in an easily discovered spot, or in an extremely challenging position such as on a cliff face, in a tree, buried, or underwater. Many variations on the basic theme exist, including caches that contain coordinates for locating the next cache in a sequence, and caches that contain hints for locating a final cache after the accumulation of clues from several other caches.[13] Geocaching is greatly facilitated through the use of a WAAS-enabled GPS receiver, owing to the increased accuracy available with this technology.

CONCLUSION

The body of literature covering the topic of navigation is immense. Many techniques for determining direction and position exist in addition to those described in this chapter. Several of the references[2,3,20,24,37,38] catalog these methods. It is sufficient to the task of navigation if the aforementioned methods are understood in terms of how they work, even if why they work remains obscure. Practice is essential, and the reader is encouraged to increase his or her familiarity with the motion of celestial bodies, the inconstancy of the earth's magnetic field, and the use of a variety of common navigational aids such as the compass and topographic map. Inexperienced persons anticipating a backcountry trip are strongly encouraged to increase their navigational skill by participating in the readily accessible sports of orienteering and geocaching. A review of land navigation techniques, advance study of the area of intended travel, and familiarity with the appearance of the day and night sky during the planned time of travel should be routine components of the preparation for any wilderness expedition.

The references for this chapter can be found on the accompanying DVD-ROM.

86 Exercise, Conditioning, and Performance

Jolie Bookspan

Being in good physical condition helps one hike and climb more comfortably, withstand harsher conditions, and be better able to rescue oneself and others. Unfortunately, a surprising number of things commonly done for exercise and conditioning are not healthy or useful for daily life, the trail, or survival itself in the wilderness. This chapter covers healthy, productive training for daily health and wilderness travel.

EXERCISE AS A LIFESTYLE

To many people, fitness means stopping normal routine to do arbitrary motions in a gym with little similarity to movement patterns in daily life or sport, and then returning to sedentary behavior, bending incorrectly, moving with poor ergonomics, riding elevators, and slouching when driving and sitting. *Fitness as a lifestyle* does not mean isolated bouts of working out at a gym on selected days or hiking on weekends. Rather, it is moving in healthy patterns throughout daily life. In much of the developing world, people commute by bicycle, lift and carry loads all day, sit and rise from the floor throughout the day, and meet in the park mornings and afternoons for social physical activity. They are mostly lean, fit, and mobile to their last years. It is important to be active, but not as a different, often inconvenient, part of the day. A gym is not needed to develop fitness. Bending, movement, lifting, stretching, and balance should become part of the ordinary day.

Specificity

Strength, power, muscular endurance, balance, and cardiovascular fitness are different body systems. Each develops through different exercise modalities. Stressing one part, such as arms or legs, or one system, such as strength or flexibility, does little to develop other parts or systems.[8] This is the concept of specificity. Running, for example, makes cardiovascular, metabolic, and structural changes specific to running but not necessarily to hanging from rock faces. Trained runners may exhaust themselves during long swims, and paddlers during climbs, even though all may have high aerobic capacity. Another example is a weightlifting practice of lifting at lower than normal speed. This does not train the more rapid joint angle movement needed in common wilderness, daily life, and rescue situations.[13] Slow lifting may build strength, just as with any other weightlifting, but not the power that depends on speed, or the injury-prevention capability that comes from training rapid stabilization. To get off the ridge before weather hits, and be fast and safe when it is most needed, one needs to train for it.

Specificity encompasses a range. For wilderness fitness, it is not necessary to train each degree of motion, range, or intensity to be encountered. There is a degree of carryover. However, it is best to train closely to the way needed for function, and to train abilities together. Wilderness skills and activities of daily living are multisystem, multijoint, multispeed, and multifunction. The wilderness often demands endurance, strength, power, and balance simultaneously—for example, holding healthy joint positioning under the weight of a pack while crossing a rope bridge, descending carrying a stretcher on skis or in foul weather, rock hopping, getting out of a flash flood, or just hiking with small children.

Strength, Muscular Endurance, Power

The definition of strength generally relates to how much you can move or carry in a single or a few efforts. Muscular endurance is how long you can continue being strong. Power in a fitness setting is how quickly you can be strong; power is work per unit of time.

Ability for strength, endurance, and power tasks depends on individual level of fitness, work load, type of work, exercise efficiency, and leverage, and on whether the work is external, such as carrying weights,[56] or internal, such as carrying oneself,[200] which varies with bodyweight. On ascents, a heavy person may work harder and closer to his maximum than a lighter person of similar fitness or, depending on the work load, even of lesser fitness. Internal work like hiking and climbing may favor the smaller, lighter person. For external work like portage, rescue, and hauling gear, a larger person of high muscular fitness may have an advantage.

Different activities require different ratios of strength to muscle contraction speed. A higher strength component compared with speed of contraction (e.g., portage and rock-face scrambling) is strength-dominated power.[8] Kicking with fins, swimming, running, jumping, and deploying safety equipment primarily employ speed-dominated power.[141] Many situations, like navigating rapids, require both strength and speed in constantly changing proportions.

Abdominal Muscles and the Core

It is a common assumption that a strong core supports the back and body for activity. However, it is not strength of abdominal or other muscles that supports the back. Rather, it is voluntary use of abdominal and core muscles to change vertebral and pelvic angle to reduce load and injury potential.[21] Even slightly built people who use their abdominal muscles (abs) to flex the

Figure 86-1. It is common to arch the back when reaching or lifting *(left)*. Arching shifts the load to the low back. Instead, use abdominal muscles to tuck the hips and reduce low back lordotic angle *(right)*. This movement (not strengthening or tightening) is how abdominal muscles prevent back strain.

spine to reduce a lordotic low back arch to a straighter position will reduce loading of the low back, and thus tolerate a heavy pack without strain, whereas someone with a strongly built torso who stands arched imposes much load on vertebral facets from the weight of the upper body on the unfavorable vertebral angle.[118] To understand how use of abdominal muscles reduces low back strain, try the following:

- Stand up and arch the low back, allowing ribs to lift and upper body weight to shift backward. You may feel a familiar discomfort. (Do not do this if you have back pain.)
- Remove exaggerated lordosis by tucking hips and ribs forward, as if starting a crunch sit-up without curling the upper body forward. Lower back pressure should be gone. Do not tighten abdominal muscles. Just move spine and pelvis away from the backward lean or arch, and into a healthy position.

Use abdominal muscles functionally in this way to reposition the spine during all activity and to reduce unhealthy lordotic arch, particularly when lifting overhead and carrying loads (Fig. 86-1). See Conditioning, later, for specific functional strengthening exercises for core muscles.

Flexibility

Flexibility training may reduce the incidence of activity-based injury.[148] One proposed mechanism is the increase of muscle length before reaching a tearing point,[155] probably from change in the viscoelastic properties of muscle–tendon units.[202] Another is reduced tendon organ activation.[132] However, the number of injuries that occur seems to dispel the hope of stretching as preventive.[170] The disparity seems to lie in how stretching is commonly accomplished, and in movement patterns used during exercise and daily life.

Many common stretches promote the original problem of thoracic rounding and stretch-weakness of the back and hip. After rounding forward all day over the computer, desk, steering wheel, handlebars, or backpack, more forward rounding is not healthful or needed. The widespread phenomenon of forward bending for the majority of stretching and exercise creates a widely occurring lack of extension range of motion needed to simply stand up straight, often resulting in chronic low-grade aches, injury, and wear and tear from habitual unhealthy ergonomics and positioning.

To understand functional flexibility for resting muscle length, try the following: Stand against a wall with the back of your head, shoulders, hips, and heels touching the wall. This is aligned standing position. A small space remains between low back and wall, but not a large space. Are your back and shoulders too rounded to stand straight without strain? Do you have to arch your back to touch your head? Does your chin jut forward or lift up? Are your hips so tight that your back is too uncomfortable to stand straight? When lying supine, can you rest comfortably without a pillow under head or knees? Anterior muscle tightness is often the culprit.

Tight, rounded anterior chest and shoulders contribute to neck pain, upper back pain, and shoulder pain.[76] Tight anterior hip muscles, common in people who sit for extended periods, change the normal angle of hips and low back, inhibiting normal standing, walking, and running, and adding a large share of low back pain.[47] Tight hip muscles, calf muscles, and Achilles tendon contribute to walking duck-footed, or toe-out.[171] The resulting change in gait and stance may wear on ankles, knees,[87] hips, and big toe,[171] and contribute to bunion formation.[161] Tight feet add to plantar fasciitis.[183] Tight hamstrings are prone to pulls and may contribute to low back pain.[40] See Conditioning, later, for functional stretches to remediate these problems.

Aerobic and Anaerobic Systems

In humans, one aerobic and two anaerobic systems generate the high-energy compound ATP, which fuels all movement and life

processes. ATP is not stored in appreciable amounts but generated at the moment needed, in quantities suiting the need, from sleep to maximal exercise. The aerobic ATP-generating system uses oxygen to combust food substrate into ATP and combustion by-products. Two anaerobic systems make ATP from stored fuels without oxygen when quick, hard exertions outstrip the aerobic system's slow generating capability. All three systems contribute in different proportions for different-length and different-intensity activities. Activities that use more aerobic than anaerobic processes are called aerobic exercises. Other activities use more anaerobic processes, and are not considered aerobic exercises. Aerobic metabolism operates during all daily life, as well as exercise, as it is the system that produces the most continuous supply of ATP.

Aerobic reactions to make ATP begin in the Krebs cycle. Carbohydrate, fat, and protein can all be used. With each step in the cycle, removed carbon becomes carbon dioxide to be exhaled, and hydrogen is removed from the food substrate to enter the electron transport system in the inner membrane of mitochondria. There they combust to water and ATP by oxidative phosphorylation. Below a certain level of activity, steady state is preserved, with acetyl-S-CoA and oxaloacetate entering the Krebs cycle, and carbon dioxide and hydrogen attached to nicotinamide adenine dinucleotide (NAD) leaving. This level is determined by the amount of oxygen that the body can deliver to end-processing organelles, and that can be processed by them. This amount is determined in part by aerobic fitness. Regular aerobic training increases blood volume and number of oxygen-carrying blood cells, expands the network of blood vessels, reduces incidence of vessels clogged with fatty deposits, and increases the number of cellular organelles associated with oxidative enzymes.[176]

The electron transport chain uses oxygen to transport removed hydrogen ions. Without oxygen, ions bond to pyruvate, producing lactate in anaerobic glycolysis. Lactate cannot enter the Krebs cycle, losing the majority of ATP production. Lactic acid also interferes with fatty acid mobilization for fuel.[91] Ability to process more oxygen through higher fitness makes the difference between stopping energy-making processes with lactate (because of gasping and rubbing of burning limbs) and restoring lactate to pyruvate in aerobic glycolysis. Pyruvate reenters aerobic energy-making processes and lactate levels fall, allowing continued activity.

Although the aerobic system is a dependable and large supply of ATP, it cannot make enough quickly. With onset of exercise, or high-intensity movement, the first few seconds are fueled by the ATP-phosphocreatine (ATP-PC or ATP-CP) anaerobic system of stored ATP and phosphocreatine. Not much can be stored, so this depletes in seconds. With continued activity, the second anaerobic system, anaerobic glycolysis, makes ATP from glucose and muscle and liver stores of glycogen. Anaerobic glycolysis is also limited with respect to ATP production. After several minutes of continued activity, the aerobic energy system contributes increasingly to energy needs, whereas the anaerobic glycolysis contribution is reduced. The higher the aerobic fitness level, the more the aerobic system can contribute, so that stored carbohydrate is not depleted through glycolysis. One theory of exercise-fatigue end-point is that when carbohydrate is depleted, capacity for athletic activity is greatly impeded,[134] and athletes are said to "hit the wall."

Energy Substrate. During heavy exertion of less than 2 hours' duration, the principal fuel is muscle glycogen. As duration of exercise increases, the preferred fuel for aerobic metabolism is increasingly fat.[153] With a low-carbohydrate diet, amino acid use may increase,[103] although not all studies support this.[139] Fat enters the Krebs cycle by beta oxidation. Increasing fat substrate through increased dietary fat intake does not enhance aerobic capacity, however. It is training that increases ability to use fat from body stores. A trained endurance athlete's body begins the change to fat-burning from glycolysis sooner than an out-of-shape body. The in-shape body extends muscle carbohydrate use, extending exercise duration.[83] Preferentially using fat and less glycogen is called glycogen sparing, a training adaptation that delays fatigue by providing Krebs cycle intermediates.[22] Ingesting carbohydrate during exercise allows continued use of carbohydrate and fat, delaying fatigue.

CONDITIONING

This section covers specific functional exercises for health during wilderness travel.

Functional Conditioning

Training in the manner used for actual activity is called functional exercise. Many common exercises may strengthen or provide exercise but not train fitness or movement in the manner needed for health, daily life activity, or wilderness skills. For example, strength increase through lifting weights by isolating specific muscles with discrete exercises does not train the multifunction movement needed to prevent injury when opening a window or carrying a heavy pack. Many people accustomed to running on a treadmill or elliptical trainer later turn an ankle on uneven terrain because they have not trained balance, proprioception, or stabilization muscles.

An effective approach to training for wilderness skills is to change the practice of exercise for each body part to natural, integrated, functional motion for real life health and physical ability. Instead of using a treadmill, walk and move over uneven ground for movement and balance training to reduce risk of sprain on uneven footing and unstable slopes. Use muscles to hold neck, back, and legs in healthy position all the time for built-in, functional exercise and injury prevention, rather than slumping under heavy packs. Climb stairs and hills, not a stair-climber machine, to train for the hills. Use good bending technique for the innumerable activities of daily living, to strengthen legs and accustom the body to functional movement. Lift, move, lunge, walk, and squat to prepare to portage, haul, climb, and hoist, and to squat where there is no plumbing.

Strength, Endurance, Power for Wilderness Preparedness

To increase strength, lift a weight heavy enough to produce muscle failure after 8 to 10 lifts. Continuously lifting weight light enough to be moved or lifted more than 10 times increases muscular endurance. Lifting the same weight faster trains power.

For external work of dragging sleds, building shelters, carrying gear and packs, and rescuing, train by carrying and moving weights in functional movement patterns, at various speeds, while maintaining healthy positioning of back, neck, and knees. Have fun devising training to simulate conditions. Drag a tire on a rope. Run while dragging children on the tire. Run with a rickshaw. Give piggyback rides. Haul a trailer on the bike.

To develop muscular ability to move your own body for hiking, climbing, skiing, and hauling up inclines, practice lifting your body at various speeds for various repetitions with pull-ups, push-ups, lunges, and squats. Lifting external weight strengthens, but the joint and muscle mechanics of lifting weights are different from moving yourself against the ground or overhead support. To progress, lift yourself while wearing a backpack or other weight. Loading bodyweight also trains the important skill of safely moving your body under the weight of external loads.

Climbing with packs and other skills requiring both internal and external strength to lift and haul is best prepared for with intelligent cross training to optimize several fitness components. Vary lifting and training workouts to avoid injury and stagnation.

Practice different components and variations of strength, power, and endurance training on different days and weeks, in the manner to be used on the trail. All strength training programs have the common goal of trying to maximize muscle work done to maximize gain.

Figure 86-2. Note the low back arch in the first, third, and fourth exercisers. When abdominal muscles are not used to prevent low back arching, bodyweight presses on the low back. Use abdominal muscles to tuck the hips and remove the low back arch to hold a straight position (second from left).

Upper Body

Push-Ups. Push-ups are an effective overall upper body exercise that is convenient and easy to begin. It is functional for strengthening, for endurance if done over many repetitions, and for skill in maneuvering your own bodyweight. A supplementary benefit is to strengthen forearms and wrist bones. (One of three main sites of osteoporosis is the wrist.) Although principal movers for the push-up are three arm and chest muscle groups, back, hip, and abdominal muscle use is needed to prevent the low back from swaying into an arch—the same stabilization repositioning knowledge critical for good posture and back pain control when this positioning is transferred to standing. Push-ups done without preventing low back sway are not effective exercise, do not train stabilization, and strain the back. Use a mirror when possible to determine positioning. To do push-ups with safe, effective positioning, follow these guidelines:

- Do not allow the lower back to sway like a hammock. Tuck hips to straighten low back arch, as if starting a crunch. When abdominal muscles are used enough to straighten the back, you will feel the abdominal muscles working. The more tuck, the more abdominal involvement. Do not hitch the hips up. Hold straight position throughout the push-up (Fig. 86-2).
- Hold head up, straight in line with your back. Keep the upper back from rounding.
- Keep elbows slightly bent, not locked straight. If arms are too weak to support bodyweight, strengthen them, but do not injure elbow joints by locking.
- Keep weight distributed over the entire hand, to strengthen the wrist without compressing the joint.
- When proficient in holding the posture without arching during slow push-ups, increase power with increasingly rapid executions of single push-ups, then multiples. Try to push off quickly and powerfully so that you can clap your hands. Try pushing off enough to lift feet as well, in safely executed jumping push-ups that maintain back posture. Land with shock absorption, without allowing low back to sway into an arch on landing. Keep hips tucked without lifting them.
- Push-ups can be done in inventive ways—for example, push-ups with one foot lifted, both feet propped on a bench, both hands on a medicine ball or exercise ball that rolls, or even hands and feet on two separate medicine balls or other rolling object. Use abdominal muscles to prevent low back arch, not by tightening, but moving the spine into a healthy position.
- Prop feet on increasingly high supports until you can do a handstand. Use a wall at first. Do small elbow bends, like reverse pull-ups. Work up to holding a handstand without a wall, doing full dips.

If holding a tucked push-up position even for a few seconds without arching is difficult, it becomes clear why posture sags under bodyweight throughout the day, pressuring the low back. Properly done push-ups train muscular positioning in the manner needed for real life posture, and for wilderness situations of carrying loads with healthy ergonomics. If you cannot do many push-ups, do not be discouraged or do them on knees. Hold a properly tucked push-up position to functionally train torso muscles. Progress by bending arms, not lowering hips. Try one push-up, then two, then more.

Pull-Ups. Pull-ups are functional training for hauling yourself up, an often-needed outdoor skill. Hang from a bar, door jamb, or tree limb for pull-ups.

- Tuck hips under, as described for push-ups. This lordosis-reducing maneuver specifically trains core muscles in a functional manner with the bonus of training proper standing posture.
- Use torso muscles to maintain straight posture as a functional posture exercise. Do not flex hips or swing legs when doing chin-ups or pull-ups.
- If full pull-ups cannot yet be done, just hang from a bar to train grip, arms, and body posture. Make increasing efforts to pull. Use a step to boost the initial lift, then lower bodyweight unassisted.
- Avoid exclusive use of machines that allow pull-ups using less than bodyweight. These machines are appealing, and they beguile you to think you are doing pull-ups, but they prevent needed training of hanging against full bodyweight.
- To progress to full pull-ups and practice for special climbing situations, hang feet on another overhead support while doing pull-ups. Reducing weight on arms assists pull-ups while adding tilt and stabilization components.

Lower Body

Lunges and squats are functional and specific lower body exercises that transfer strength and ergonomics to daily life and wilderness situations.

Lunges. The lunge is a functional lower body strengthener that enhances the ability to lift oneself to a stand carrying overloaded packs or a rescued friend, to duck falling rocks, and to climb difficult terrain. Use the lunge for a healthy way to bend and pick things up, and to get free leg training all day. Done properly, lunges are beneficial to knee strength and health. The lunge is often done improperly with strain on the knee and back, and with ineffective use of muscles. To do healthy and effective lunges, do the following:

- Stand with feet at normal width apart. Slide one foot back as far as comfortable.
- Center your weight between both legs, not leaning on front leg.
- Keep the back foot straight, not turned out. Lift the rear heel.
- The key to healthy lunges is torso and knee position. Reduce low back arch by tucking hips under, as if starting a crunch, to straighten torso. You will feel a stretch in your back anterior hip. Do not lean forward or arch backward.
- Keep the front knee over the front heel as you lower gently. Do not let front knee come forward.
- Keep weight distributed on both legs, not leaning on the front leg. Keep body upright as you lower and rise back up. Lower anywhere from an inch to almost to the floor, as you progress. Keep the back knee off the floor.
- Use lateral thigh muscles to keep the front knee over the heel, not sagging inward (knock-kneed, or valgus knee), which stresses the knee.
- Keep feet in position and do lunges one after the next without moving the feet.
- Start with 10 repetitions and increase.
- Do these lunges with front foot on a bench. Keep weight centered on both legs, and weight on entire front foot, not toes. Try the same lunge facing backward, turning so that the foot on the bench becomes the back foot, heel raised on the bench. Center weight, and keep back foot straight, not turned outward.
- Practice landing from jumps in proper lunge or squat form. Do not let bodyweight fall inward on your knees or arches. Land with soft shock absorption using torso, thigh, lower leg, and gluteal muscles. Learn to hold proper posture during jumps to train power, strength, and speed, while learning to reduce injury potential. Begin to add sequential jumps and landings for plyometric training (see Power and Plyometrics, later).
- To progress, hold hand weights during lunges. To progress further, lift hand weights with each lunge: curls, presses overhead, shrugs, and lifts to the side and to the front. Keep good knee, back, and neck posture. Do not arch your back to lift overhead. This multimuscle activity simulates actual daily needs such as lifting laundry baskets and babies, and it trains lifting skills for the trail.
- Practice lunges first slowly, then more quickly (and carefully) to train functional speed and power.

Squats. The squat is a functional strengthener, enhancing ability to lift, carry, jump, and rise from the ground quickly and easily and while carrying loads. Done properly, squats strengthen legs while training good bending positioning to avoid knee and back injury. Do partial squats for household lifting to train whole body movement that combines balance, posture, strength, and flexibility, and to save the back from bad bending. Done incorrectly, squats may strain back and knees. Practice with a mirror to observe positioning, and start without holding additional weight. The following is healthy positioning for squats:

- Stand facing the mirror and observe your knees. Keep feet apart and parallel or slightly turned out, but not turned out like a dancer.
- As you squat, keep your bodyweight back toward your heels, not toes, to protect knees. Hips move back without arching the back. Keep hips tucked under.
- Just as with the lunge, keep knees over ankles, not drooping forward or knocking inward. Keep kneecaps straight ahead in line with the direction of the feet. Use lateral thigh muscles to keep knees over feet, and legs parallel.
- Keep weight on the sole of the foot, not the arch. You will feel the effort in leg and hip muscles, and off the knee joints.
- Stand sideways and try the squat again, watching knees in the mirror, adjusting posture so that knees do not move forward when bending. Keep hips tucked under, not stuck out in back. Back is upright and straight.
- Keep feet in position and do squats one after the next without moving the feet.
- To progress, do squats holding hand weights, then lifting the weights with each squat to do curls, presses overhead, shrugs, and lifts to the side and to the front, among others. Keep good knee, back, and neck posture. Notice if you arch the low back to lift overhead. Prevent that by tucking hips and standing straight.
- Standing (with healthy posture) puts less load on the back than sitting with a weight. Instead of sitting to do biceps curls or other weightlifting, combine repetitions of curling a weight with repetitions of squatting.
- If you use a barbell, hold the weight across the chest. Avoid weight behind the neck.
- Try upside-down squats by lying on your back and lifting your pack or a friend with your legs. Do not curl your back, which stresses vertebral discs. Keep hips on the floor.

Watch positioning in a mirror to learn where knees are in relation to where they feel they are. Use this kinesthetic practice to learn healthy positioning. Transfer this learned healthy knee and foot posture for all daily bending and walking. Do not be afraid or disappointed if this is work. Using muscles is the point.

Avoid full squats, or squatting on the balls of the toes. Such acute knee angle while under bodyweight is tough on the knees. The meniscal injuries of professional baseball catchers come from chronic squatting on their toes.[46] Instead, to sit in a full squat to rest, or for toileting, keep heels on the floor, a customary sitting posture in much of the world. This reduces pressure on the knees and is an Achilles tendon stretch. Maintain feet parallel without turning them out or letting bodyweight press inward on the knees or arches. Keep knees over feet, not drooping inward.

Abdominal and Core Muscle Conditioning

Strengthening core muscles is commonly thought to solve back pain by correcting the muscle weakness that underlies pain. But on scrutiny, core strengthening has not been found to reduce

pain[75] or to effect any greater relief than aerobic exercise or non-strength programs.[1,59,111] Exercising for stronger abdominal and torso muscles does not alter the poor mechanics that are the source of much pain.[128] Using abdominal and core muscles to unload the facets by reducing lordosis is how the abdominal muscles support the back.[21] One uses abdominal muscles to tuck the hips and spine enough to reduce low back arch when standing, and to prevent increase in arch when lifting overhead—for example, to put away groceries and pull off a shirt overhead. Use abdominal muscles in this way, not by tightening, but to hold healthy posture for all daily activity.

Crunches, and other common flexion-based exercises of hunching forward, may increase strength for flexion beyond a straight position, but they do not provide functional strength for standing and lifting in the upright position in real activity.[21] For effective abdominal muscle strengthening specific to daily life and trail ergonomics, try the following:

- Use a pair of light dumbbells (5 to 10 lb [2.3 to 4.5 kg]). Lie face up on the floor with knees bent comfortably.
- Extend arms straight back, biceps next to ears and dumbbells about an inch off the floor.
- Keep elbows slightly bent, and the back of your head on the floor.
- The key to using abdominal muscles is to prevent the ribs from lifting up, or the low back from arching from the floor. Press ribs downward.
- Slowly raise and lower the dumbbells to about an inch from the floor, as many times as you can (at least 8 to 10 times), continuously using abdominal muscles to keep the low back from rising from the floor at any point during the exercise. Keep breathing.
- When you have mastered controlling torso positioning during slow arm movement, increase the speed of raising and lowering the weights. Raise the weights no more than a few inches from the floor, not overhead where you can see them. As the weight lowers, the momentum and weight will encourage lifting the ribs and arching to shift the work of the exercise off the abdominal muscles to the vertebral joints. Use abdominal muscles to prevent this. This abdominal exercise also works arms and back. The benefit is to learn to use abs to hold safe, effective, and functional posture when using the rest of your body.
- To progress, reduce knee bend, eventually straightening legs to the floor, without increasing the low back arch. Keep hips tucked and ribs down, or core training benefits are lost. It is commonly said that knees must be bent to protect the back. However, normal standing and function during real activity is not done with knees bent. It is not bent knees that protect the back, but using core muscles to maintain vertebral angle.[21] Use this straight-leg exercise to retrain use of abdominal muscles to hold spine and pelvis in position when standing.

Holding posture against a stationary or moving load is the key to learning how to use abs and core muscles for back pain and posture control. Next, using the technique just described to prevent lordotic arching under load, do push-ups, with hips tucked. Without the tuck to remove low back arch, core muscles are not in use, and bodyweight is loaded on the vertebral joints.

- Holding the tucked push-up position, turn to the side, raising one arm from the floor, not allowing hips to sag to use oblique abs in functional training for posture under side loads.
- Try this side push-up, raising one leg.
- Hold a straight, face-down, tucked push-up position, with one arm raised just above the level of the ear. Keep the body level, not turned or dipped. Raise the opposite back leg, keeping posture.
- Do push-ups on a medicine or exercise ball. Practice holding straight torso posture, using abdominal and core muscles to train healthy postural habits. Do not take a good exercise adjunct like a ball and lose benefits by doing crunches on it.
- Do push-ups with tucked hips and one leg out to the side 90 degrees and parallel to the floor.

Using abdominal muscles does not mean "sucking them in," "tightening," or "pressing navel to spine." Breathing and healthy movement are restricted or impossible with tightened abs, and tightening does not change the poor posture that loads the low back. To understand this, try the following: Tighten abs as commonly taught. Press navel to spine. Tighten the entire area. Now breathe. Note that such tightening would not be possible or useful for daily activity. Next, stand with arched posture. Tighten the abs and surrounding musculature. Note that posture does not change. Stop tightening the area so that movement is unrestricted. Tuck the spine and hips to remove the lordotic arch, straightening posture. Train the abdominal musculature for wilderness activity by using muscles to hold healthy spine and pelvic positioning under load, during standing, lifting, and movement. This will provide efficient exercise at the same time as retraining posture and back pain prevention habits.

Hands and Wrists

Hands and wrists are often a weak link. They require strengthening and training like the rest of the body for daily use, and for wilderness needs of grabbing, holding, lifting, hanging, pulling, belaying, carrying, and rescues.

- Squeeze things. Open jars. Wring wet clothes. Work with clay. Give massages. Use pliers and screwdrivers. Saw wood.
- Hang from a bar or tree limb to strengthen grip. Increase time held. Hang from fingers, then fewer fingers, and groupings of fingers. Perhaps, eventually, hang from one finger. Who knows when that will be handy?
- Carry groceries with hands, not a cart. Do not lean back when carrying. Stand straight, torso tucked using core muscles (see previous abdominal muscle section).
- Carry suitcases and other items with handles, arms by your sides, holding with finger grip (farmer's carry). Carry heavy dumbbells across the room (lifting and setting down properly, using legs, not bending forward). Use farmer's carry on unwieldy items like plate weights, and heavy books like encyclopedias, as if they were suitcases.
- Train pinch-grip and holding-grip strength of fingers and palm by carrying partially inflated medicine balls of different weights or rubber balls filled with water.
- Do push-ups to strengthen wrists and arms, and become accustomed to holding and pushing weight with your hands. Keep weight on whole hand and fingers, not just concentrated on the wrists.
- Do push-ups, or hold the push-up position on fingertips, then fewer fingers, and groupings of fingers.
- Use fingers to push and pull things.
- Instead of hanging directly from a pull-up bar, loop a towel over the bar. Grip the towel at each end and hang to train grip for climbing and rescue.

- Hang from door jams and oddly shaped overhead structures for finger grip training.
- Jump up to hang from fingers, instead of first gripping and then lowering bodyweight, to practice unexpected situations requiring quick, sure grasp.
- Get weeks of rest between intense periods of training finger and hand grip to prevent tendon overuse.
- Regularly stretch fingers back softly, and open fingers as wide as possible.

When lifting hand weights, keep wrists straight instead of compressed backward. When using your hands for everything from typing to driving, do not angulate wrists back, compressing the joint. Train hand strength and dexterity to prevent hand and wrist injury, to reduce risk of osteoporosis, and for strong climbing, carrying, rescue, and self-rescue.

Feet and Ankles

Feet and ankles need exercise, an often overlooked activity in the gym. Tight, weak feet and ankles are more likely to cramp, hurt, strain, and develop plantar fasciitis[114,208] and deformed toes. When the big toe joint does not bend when walking (hallux rigidus, or stiffness in the first metatarsophalangeal joint), it alters gait and posture, reduces needed plantar stretch, and promotes bunion. Low back muscles and discs bear the brunt. Weak, unused toes easily deform and curl. Toes must be straight and strong for balance and healthy gait. Weak, overly stretched ankles are prone to recurrent sprains. Feet are easy to condition, because they routinely bear bodyweight and movement forces greater than bodyweight.

Exercise feet by retraining muscles and increasing general exercise with attention to using foot muscles to hold foot posture, instead of letting muscles atrophy in tight shoes. Here's how:

- Use lateral ankle and thigh muscles to keep weight off arches. Do not let feet roll inward on arches to stand and walk. Many cases of "flat feet" are acquired pronation (bad posture and weak muscles), not a structural problem.
- Keep feet facing straight ahead for gait. Standing toe-in strains the foot, knee, and hip, and affects normal gait. Standing with feet turned out contributes to hip and knee pain, bunion formation, and is often mistaken for flat-foot because it weights the medial edge of the foot, pronating, everting, and flattening arches.
- Stretch feet daily using the lunge, described earlier. Keep the back foot straight, not turned out even slightly, or stretch on the foot and calf will be lost. Lift the heel to stretch the bottom of the foot. Then turn the top of the back foot (volar surface) toward the floor and stretch the other way. When stretching the sole of the foot with your hands, do not pull back only the toes; stretch the entire sole.
- Lift heels and rise up on toes. Keep weight over the big toe and second toe, not teetering ankles outward. Practice relaxed, straight foot posture.
- Pick up increasingly heavy things with toes and pass them to your hand while standing.
- Put toes under something like a door, your other foot, or a friend, and lift toes.
- Play hopscotch. Hopscotch was possibly developed for Roman soldiers to exercise their feet and ankles. Do other fun combination balancing and hopping games.
- Have fun with the balance exercises (see Balance Skills for the Trail, later). They strengthen and train ankles and feet in ways needed for normal life and to keep balance on narrow, rocky, uneven, and slippery trails.
- Stand on one foot while on the phone or other activity. Move other leg in inventive directions at varying speeds.
- If toes do not move freely, or shoes make toes touch each other, the shoes are too tight for daily wear. Practice moving toes in all directions often. Stretch toes with your hands and by using your foot muscles. When standing, do not clench toes, or lift toes from the ground. Avoid heeled shoes; they are prone to deform feet and are detrimental to posture and gait, affecting the kinetic chain from feet to back and neck.
- Hold foot and ankle posture against pronation. Do not wear a tight boot to do what your own muscles and kinesthetic sense can do. You would not wear a neck collar for upper body posture. Do not let foot muscles atrophy from the disuse that a tight, supportive shoe allows.
- Walk softly; heel first, rolling to your toes. Keep foot straight, not turning in or out. Bend at the toe knuckles and push off the ball of the foot, not the side of the toes. When descending stairs, come down toe first, then bring the heel down. Do not crash down on heels. Bend knees for shock absorption with each step down.

Power and Plyometrics

Plyometrics are exercises designed to train muscles for quick powerful moves.[18] The muscle is first quickly stretched under load (contracted eccentrically), then immediately, forcefully contracted concentrically—for example, push-ups with a clap between each pair, and rapidly jumping over a line of boxes with quick deceleration crouches between each pair. Plyometric exercises stress muscles and associated attachments more than other exercises.[57] Learning and maintaining healthy joint positioning and good shock absorption are the key to safety in plyometric training. Here are some fun training examples:

- Throw and catch a heavy medicine ball against a (sturdy) wall or in the air in a quick succession of forceful throws and quick deceleration catches, fully bending arms. Many small children love being thrown in the air and safely caught. Remember safety for all in this exercise. Do not arch the low back when throwing or catching. Keep hips tucked, using torso muscles to handle the load, not the low back. Do not lean back when standing and throwing overhead. (See Abdominal and Core Muscle Conditioning, earlier.)
- Lie back on an incline or a sofa, with arms extended, feet on the floor. Have a friend stand between your legs, his back to you, holding his body rigidly so he can lie back balancing his back on your hands. Fling him to an upright stand. When he falls back, catch him on your hands, then fling him forward again. Experiment with various safe variations on this exercise. Progress until this can be done lying down. Try this using your feet instead of hands to push and catch. Employ good shock absorption with each catch, bending knees and hips. Keep healthy knee positioning. Do not let knees sway in (valgus knee, or knock-knee) under load.

Stretching for Wilderness Preparedness

Heavy packs and difficult terrain make it tempting to let posture sag in directions already favored by tightness. Strain and injury often result. Flexibility training for the wilderness is best served by addressing healthy joint positioning (posture) and movement dynamics. The main areas to stretch for functional wilderness

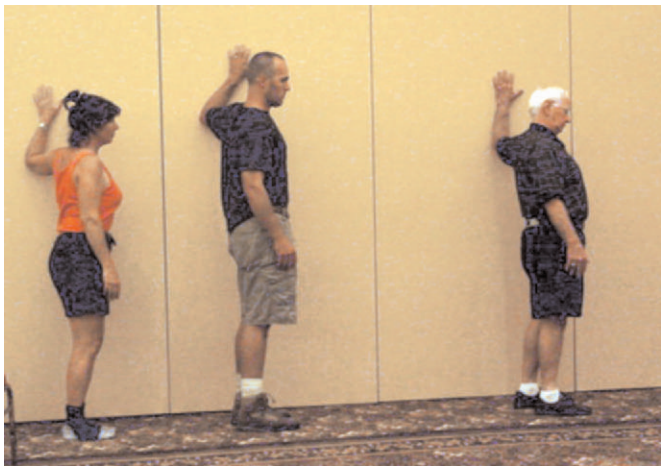

Figure 86-3. This is an excellent anterior chest stretch to prevent round-shouldered tightness that promotes neck and upper back pain. Pull your bent elbow behind you. The wall holds your elbow back as you turn away, stretching the chest muscles. Do not bend the hip *(left)*, tilt the head forward *(center)*, or arch the back *(right)*.

health and ergonomics are anterior shoulder and chest, hamstring, hips, and feet. Following are a few main stretches.

Anterior Shoulder and Chest

- Stand upright near a wall. Notice the positioning of your hands and note if thumbs point toward each other. This internal rotation usually indicates pectoral shortening. Note that trying to hold arms with thumbs facing forward instead of inward may feel tight or unnatural, as tightness prevents it. To restore healthy resting length to the anterior chest, face the wall and pull one bent elbow out to the side behind you. Turn your body away from the wall and use the wall to gently pull the elbow back. Feel the stretch in the anterior chest muscles. Do not hunch or tighten the shoulder. Hold a few seconds on each side. Drop arms and observe thumb positioning, which this stretch corrects by lengthening the previously tight anterior chest. Muscle length should now be more comfortable, making it possible to stand straight, with heels, hips, back, and the back of the head touching a wall (see Exercise as a Lifestyle, earlier). Do this stretch in the morning, before exercise, and throughout the day to restore healthy shoulder and head positioning (Fig. 86-3).
- To stretch the top of the shoulder (trapezius stretch), stand straight, placing one hand behind the opposite hip, as if in an opposite pants pocket. Tilt head toward the hand, stretching the side of the neck, body, and, as you slide the other hand down toward the knee, the side of the hip. Do both sides for a few seconds each. Keep breathing. Do not lean forward. Try the preceding wall-stand posture check again. It should become even more comfortable and possible.
- Lie on the floor flat on your back without a pillow. Do not lift your chin or arch your back. Practice relaxed straight posture. Can you put your arms straight out to the side on the floor without arching your back? Can you bend your elbows with hands against the floor, as if in a stick-up? Can you hold upper arms against your ears, still touching the floor without arching your back? Many people are so round shouldered that this is uncomfortable or impossible. Use the two preceding stretches.
- Lie supine with a small roll, pillow, or soft log-like support vertically between your shoulders. Retract shoulders to the floor over this roll without arching the low back.
- Lie face down, hands at sides and off the floor. Slowly lift your upper body a few inches, then lower. This upper back extension is an effective postural strengthener that combines range of motion. As you progress, move hands to the side, then overhead.

Hip and Thigh

- The lunge described in the strengthening section is an important functional stretch for the anterior hip and thigh, Achilles tendon, and foot. Tuck hips under without leaning back to gain the crucial hip flexor stretch.
- Lie supine with bent knees. Lift hips from the floor.
- To stretch the deep lateral rotators, lie supine with knees bent, both feet on the floor. Cross one ankle over the opposite knee. Gently press the crossed knee away. Lift the other foot from the floor. Hold briefly, then drop both legs to each side gently. Repeat with other ankle crossed. For this stretch in a chair, sit up, one ankle crossed over the other knee. Press the crossed knee down. Arch the entire back and lift head up, chin in, as you lean slightly forward.
- An effective low back and hip strengthener that includes a functional stretch is prone back extension. Lie face down, hands under chin or at your sides. Slowly lift legs a few inches, then lower. Keep knees straight. Or start by doing one leg at a time, then progressing to both.
- To stretch the quadriceps while standing, hold one foot behind you. Tuck your hip. Arching the back will lose the stretch. To protect the knee, push your foot away into your hand, but do not pull your foot in to your behind.
- To stretch the quadriceps lying down, curl on one side, both knees bent in front. Extend the top leg behind you, foot in hand. Keep the bottom knee bent in front.

Hamstrings

Lack of sufficient hamstring flexibility reduces capacity to sit without rounding the back, which in turn puts pressure on the lumbar intervertebral discs. The result is that many people sit (and stretch) rounded because tightness makes slouching more comfortable than healthy position. Stretching the hamstrings can restore resting length sufficient for healthy sitting ergonomics.

- Bending forward from a stand overloads low back structures.[166,191] It does not become healthy by calling it a stretch. To stretch hamstrings without this bending, lie on the floor and lift one leg, pointing the foot to the sky. Keep the other leg straight and flat on the floor. Notice if your anterior hip is so tight that the leg on the floor lifts also. Include anterior hip stretches by lunges, described earlier, until remedied. Keep shoulders and neck relaxed on the floor. Do not round the back and call it a leg stretch.
- To stretch both legs at the same time, lie supine with both legs overhead. Keep the low back and hips on the floor. Arch the low back to lengthen the ischial tuberosities where the hamstrings originate. Flex ankles to further stretch.
- To stretch standing, face a bench or chair and prop one foot on it directly in front of you. The key to this stretch is to keep the standing foot straight forward, not turned out. Keep the

foot on the support, toe up. Drop the hip of the lifted leg down and square with the standing leg. Arch the low back slightly to lengthen the hamstring, without rounding the back.
- Another hamstring stretch is downward dog, described next.

Achilles Tendon and Foot

- A common Achilles tendon stretch is to stand in a lunge position against a wall. It is ineffective when done with the hip flexed and protruding posteriorly instead of tucked inward. Bring the hip forward, as if trying to touch it to the wall. Keep the back foot straight, not turned out, or the stretch is lost and the medial knee bears the pressure.
- A more effective Achilles tendon stretch than the standing stretch is the lunge (see Lower Body, earlier). Keep the hip tucked and back foot straight, not turned outward.
- Downward dog is an effective multijoint stretch with bodyweight supported on hands, protecting the back. Put hands and feet on the floor, hands far forward of the feet as if starting a push-up, weight mostly on hands. Keep feet where they are, lift hips up in the air, pushing hips backward until heels relax to the floor. Arch the back, rather than letting it round or hunch. Relax head downward. Keep feet straight, not turned, weight on rims, not arches. Push fingers forward with straight, not locked, elbows. You have probably seen dogs and cats stretch this way.
- During daily walking and movement, do not let bodyweight fall inward to the arches; keep weight on the sole of the foot. Point toes straight ahead. This prevents uneven and unhealthy stretch forces that gradually deform feet, ankles, and knees. Make sure straight leg posture continues through the knee and hip, to prevent straightening the foot from overstraining another part up the kinetic chain.

Flexibility-Enhancing Techniques

There are several methods to augment stretching gains. Stretch regularly. Be warmed before stretching.[60,198] *Warming up* means raising body temperature, because elasticity increases with temperature. Active warming is easily and quickly accomplished with exercise, like a few push-ups and lunges until breaking a light sweat. Do not be afraid of exercise without air conditioning. Within limits, a warmer environment helps. Passive warming in a hot-tub or shower, or locally applied heat, also works well.

Another quick and effective technique to improve immediate flexibility is called push-pull, contract-relax, or proprioceptive neuromuscular facilitation (PNF).[51] While holding any given stretch at maximal comfortable stretch, push (contract) against resistance in the direction opposite the stretch for 4 or 5 seconds without moving or reducing the stretch, to fatigue the muscle. Then pull (relax) into the stretch. Use this technique slowly and safely for any desired stretch. For example, do the following for hamstring flexibility:

- Lie face up with one leg lifted, and shoulders, head, and other leg flat on the floor.
- Hold the back of the lifted leg at maximal comfortable stretch.
- Push the leg downward against the resistance of your hands. Hold stationary while pushing downward for about five counts to fatigue the hamstrings.
- Stop pushing down. Pull the leg up gently toward your chest. It should immediately gain a greater stretch.
- Repeat, pushing against hands at the new stretch length, then gently pull in to the next greater stretch. Keep breathing. Repeat for three stretches on each leg.

To stretch the triceps using PNF, do the following:

- Lift one arm overhead, elbow bent by your ear, allowing the hand to hang straight back. Do not arch your back to raise the arm. Keep torso and head posture straight, not bent forward or back.
- Without moving the raised elbow forward, press the raised elbow in front against the other hand (or a wall) for five counts to fatigue the triceps.
- Release the press and pull back. An immediate and easily gained farther stretch should result. Repeat for three stretches on each arm.

Another technique for gaining greater stretch is the active assist.[34] The antagonist muscle is used to assist in pulling the stretch. For example, to use this for hamstring stretching:

- Lie supine with one leg lifted. Shoulders, head, and other leg remain flat on the floor.
- Instead of holding the back of the lifted leg with the hands, assist the stretch of the lifted leg toward the body only using quadriceps muscles (antagonist to the hamstrings). Maintain the held position in the air for 4 to 5 seconds, then relax and pull the leg farther in toward the body using hands. Repeat for three stretches on each leg.

To stretch triceps using the active assist:

- Lift one arm overhead, elbow bent by the ear, allowing the hand to hang straight back. Do not arch the low back to raise the arm. Keep torso and head posture straight without bending forward or back.
- Use the muscles of the raised arm to pull the raised arm further back, without using the other hand. Maintain the held position in the air for 4 to 5 seconds, then relax and push the arm further back using the other hand. Repeat for three stretches on each arm.

Remember that functional movement is training in the manner useful to real life. Many stretches are static and nonmoving. However, muscle lengths needed on the trail and during exercise are achieved while moving, such as when navigating large boulders, or performing a high brace in kayaking. Incorporate stretches as needed for movement, rather than solely practicing static stretches. A good example is the lunge (see Lower Body, earlier). With properly held posture throughout the movement, this exercise dynamically stretches the anterior hip, quadriceps, calf and Achilles tendon, and plantar surface of the foot. When used with various upper body lifts, it trains functional use of abs and shoulders to maintain healthy ergonomics during stretching and movement.

Aerobic and Anaerobic Conditioning

Aerobic capacity allows steady climbs carrying packs, long runs and swims, and any continuous exertion that would be difficult or undoable at lower aerobic capacity. Anaerobic capacity allows fast, hard exertions, like dashing for a bus or away from danger, swimming underwater, running inclines and stairs, throwing a ball or rescue device, sliding home in baseball, lifting and moving gear, and carrying someone to safety. Both aerobic and anaerobic reactions are used in varying amounts whenever you move over varying speeds and distances. Train both for intended needs.

There appears to be reliable correlation at usual intensities between perceived and actual exertion,[100] making exercise level

fairly easy to estimate. A pace perceived as moderate effort is usually a moderate heart rate and oxygen consumption (VO_2). Depending on level of fitness, moderate exercise is 40% to 60% of the maximal volume of oxygen a person can use to do exercise (VO_{2max}). Aim for aerobic exercise with long, increasingly paced activity at moderate intensity. If intensity is low, as in walking or in exercise class floor-work of leg lifts and crunches, effort is below aerobic benefit. As exercise intensity increases, lactate production eventually exceeds removal. This point is the anaerobic threshold, or lactate threshold. Training reduces lactate accumulation during exercise.[207] A person with low aerobic fitness may reach threshold at close to 55% of VO_{2max}. High-level athletes can work at 80% to 90% of VO_{2max} before reaching the point where their systems cannot remove lactate as fast as it is produced. To improve aerobic capacity, keep a target pace just past comfortable breathing level to retrain brain and body to handle exertion at levels that were previously too hard.

Anaerobic training enhances mechanisms to store and restore anaerobic fuels, and it trains muscles and body systems for short, intense movement. For bursts of less than about 7 to 10 seconds, like a tennis serve or 100-m dash, it is the ATP-PC that is primarily trained. Improve ATP-PC capacity through intense sprint runs, swims, lifts, throws, stairs, and other target activities hard enough to create fatigue in seconds. Hard efforts lasting a few minutes train the glycolytic system. Finish the last 3 minutes of long training efforts with the fastest possible pace. Alternate sprints with slower recovery activity—for example sprint runs with walks, sprint bicycling with slower cycling, and sprint swims with easy laps. Jump speed rope for 15 seconds, and rest for 15 seconds. Increase to jumping 30 seconds, with 15 seconds rest, and so on, increasing time and speed of exertion. Challenge speed by training with all-out efforts. Vary resting time between bouts to train both ability to recover and ability to work fatigued. Keep moving following intense exercise, as light exercise removes lactate faster than does complete rest.

Train aerobic and anaerobic systems together and separately. Vary exertion levels for variety of ability and use. Vary the type of workout. Cross train with varied activities. Intersperse long efforts with speed bursts. Vary workouts and skill combinations from week to week and month to month. Vary direction in different planes with zigzags, rapid directional change, ducks, swoops, and jumps for balance, stability, proficiency, and safety in emergency situations. Of many possible training strategies, chose efforts similar to projected wilderness needs.

Training is all too often curtailed after injuries and pain from poor positioning and ergonomics of ankle, leg, back, and neck during activity and training. Often, it is not the activity that precipitates injury but poor body mechanics—for example, running with a lordotic arch, head jutting forward, ankles rolling inward, and poor shock absorption. Good body mechanics are the key in training for aerobic and anaerobic benefits.

Balance Skills for the Trail

Balance is easily and highly trainable, but it is usually forgotten. Good balance is crucial for ease of movement, independence, variety of activity, and preventing falls, ankle sprains, and slips on the trail. Injury and disuse diminish balance, which is a use-or-lose skill. Vicious cycles grow of poor balance, injury, and decreasing activity because of inability and reduced activity. Recurring ankle sprains are often a matter of lack of retraining foot and ankle proprioceptors that give information about positioning. Weak untrained ankles turn without warning muscles to regain balance and footing.

A gym is not needed to practice balance. Most gyms neither have balance programs nor incorporate balance exercises into classes or workouts. Try the following in a safe environment:

- Stand to exercise. It is functional and healthier for the back than sitting to lift weights or stretch.
- Stand on one foot for increasing periods of time. Try it when you are on the phone and washing dishes. Do both legs. Do not let your weight fall inward on your arches. Maintain good foot posture by using muscles and balance.
- Throw and catch things standing on one foot.
- Practice balancing on one foot, one knee, or one knee and hand while on the floor.
- Practice getting up from the floor and back down in a smooth manner. Repeat without hands. Repeat holding a package.
- Put on and take off pants, socks, and shoes standing up for balance and flexibility.
- Stand on tiptoe. Keep ankles straight and weight over the big toe and second toe, not teetering on the outside of the foot or inverting the ankle. Raise and lower 10 times.
- Raise and lower on the toes of only one foot. Maintain healthy foot position with weight centered over the big and second toe. When proficient, try balancing with eyes closed.
- Walk over uneven ground.
- Walk a line, and then walk the line backward.
- Walk a line sideways. Cross feet, first one in front then the other behind—this is called grapevine walking. Sports players practice running like this quickly.
- Hop on a line, and then hop the line backward.
- Hop on one foot for increasing periods of time.
- Slalom hop the line, and then slalom hop the line backward.
- Try going without trekking poles before the first climb.
- Hop from one line, space, marking, or crack in the sidewalk or ground to the next. When landing from any hop or jump, use shock absorption by bending knees and using muscles to slow and pad landing.
- Kneel on a large exercise ball. Stand on rolling surfaces. Walk on fence rails. Use a skateboard and balance board. Go skating.

Developing Speed for Wilderness Situations

Wilderness conditions often require ability to react, move, grasp, or run quickly. Speed training aids outdoor activities, from catching falling equipment or companions, to paddling around unexpected logs, to running from bees. Each person has different muscle fiber distributions that favor speed, strength, or endurance. Some have greater distribution of slow, oxidative (type I) fibers, which are highly fatigue resistant, benefiting long steady efforts. Others may have a greater percentage of fast twitch fibers (type IIb), favoring bursts of strength and speed. Both types respond to training. Individuals with a predominance of slow-oxidative fibers may need to train speed as a corrective countermeasure.

The way to be fast is to train fast. The practice of slow weightlifting, sometimes called super slow, is a limited way to train with weights. Slow training increases the total time spent lifting, so it increases strength, but it will not develop speed for moving heavy objects. Similar strength and endurance gains can be made from lifting weights more quickly, by using more lifts

so that total muscle work remains the same. As the amount of weight lifted quickly increases, the power component increases. When speed training, maintain posture and safe lifting technique. Injury potential rises with speed of movement, another reason to properly train speed. Following are a few examples of speed games to increase speed of movement. With each, imagine application to wilderness performance situations.

- Have a friend hold a piece of paper near your hand and drop it without warning. Practice catching.
- Punch at a candle flame to extinguish it.
- Have a friend point a flashlight beam at the ground, moving it quickly from point to point. Jump quickly to each new point. Try jumping with both feet and hopping with one foot.
- Kneel on the floor and try that game using your hand, grabbing or tapping at the flashlight beam on the floor. Do a few dozen points, and then change hands.
- Run a short distance—for example, 15, 25, or 50 yards—at maximal speed. Rest 10 seconds. Repeat 10 times. Work to increase speed.
- Practice moving a heavy weight quickly, both bodyweight and external weight. Do one push-up at high speed, rest, and repeat, until reaching 10 push-ups. Practice lifting your pack, or throwing and catching a weighted ball (medicine ball).
- Do 8 to 10 push-ups quickly without stopping and without letting the low back arch or hitch upward.
- Try lunges quickly, first singly, then in groups.
- Punch (or grab to simulate catching something) as fast as possible. Then repeat twice in rapid succession. Then three times, then four, and so on, until you can punch or grab like a machine gun. Bring the elbow all the way back to your side each time. Launch your fist straight forward and back like a piston, or like sawing wood.
- Quickly sit or kneel on the floor, and rise to a stand. Try this (carefully) wearing your pack.
- Have a friend poke at your feet quickly with a (nonsharp) stick or squirt you with a water pistol. Jump and duck to avoid hits.
- Practice throwing a heavy ball or rescue device. Practice throwing and catching heavy thrown objects (carefully).
- Devise other speed games that simulate needs and varying proportions of speed and strength—assemble gear, break camp, don rescue clothing, dodge hurtling objects, and so on.

Brain Conditioning for the Wilderness

The brain is an important use-or-lose part. Mental, like physical, functioning requires stimulation and conditioning throughout life to deter effects of disuse, often mistaken for aging, and situations compromising brain cell health such as hypoxia or overindulgence in alcohol and other substances. It seems to be the case that keeping physically fit helps mental functioning. It also seems effective to practice mental skills separately. Add brain games to develop ability for simultaneously coping, evaluating, and decision making. Choose fun and stimulating cognitive activities for memory, reasoning, and speed of processing. Practice puzzles, map reading, tracking, orienteering, and charades around the campfire. Learn celestial navigation. Learn a foreign language, paying attention to accent. Learn to identify bugs, flowers, spoor, scat, constellations, and edible food in the wild. Examples of thinking for better exercise ergonomics and injury prevention are given throughout the chapter. *Mens sana in corpore sano:* A healthy mind in a healthy body.

Conditioning for Special Environments

To handle special environments, train in the expected situation. For heat, train in the heat (safely) without returning to an air-conditioned microclimate. For cold, get out in the weather. For a 30-mile hike, practice the whole 30 miles. For a journey at night, train at least a portion at night. For extended caving, accustom yourself to lengthened wake cycles and lack of external circadian cues. Show your system what to expect. Cross train, taking into consideration the several interacting environmental conditions of wilderness travel expected. Keep general health high.

Heat

Because exercise in the heat (see Chapters 10 and 11) is one of the greatest cardiovascular loads on the body, aerobic fitness is a major factor in heat tolerance.[36] Exercise in high heat may be expected on desert treks, on ultramarathons, on jungle expeditions, when working in tropical medical clinics, and during other operations in deserts and tropics. Exercising in heat produces heat acclimatization more effectively than heat exposure without exercise.[130]

Fitness training in heat produces several adaptations. A major adaptation is expansion of blood plasma volume.[167] More blood is pumped with each beat, supplying blood simultaneously to muscles for exercise and skin for cooling. Increased plasma volume increases the sweating reservoir and functions as a heat sink, absorbing heat without an increase in body temperature. Sweating begins earlier, at a higher rate and with greater electrolyte conservation.[174] Sweating distributes more to limbs, furthering cooling from areas of high heat loss. With increased fluid available for cooling and muscular activity, a fit, heat-acclimated person's core temperature will not rise as high at rest[27] or at the same exercise intensity as that of an out-of-shape person.[122] The in-shape body produces more of several protective heat-shock proteins, further increasing tolerance to heat exposure.[126] Physical training can negate the decrease of heat tolerance with age.[5] Heat adaptation occurs quickly, with dramatic changes in the first 4 to 5 days. Acclimatization is almost complete in 1 to 2 weeks of continuous exposure to exercise in the heat.[138] Acclimatization does not occur if time is spent in air-conditioned environments. A protective environment prevents discomfort and heat illness, but such a microclimate reduces acclimatization and heat conditioning. With reduced physical conditioning, adaptations are lost over several weeks.[5]

Physical training in a cool environment improves tolerance to exercise in the heat and the rate of heat acclimatization, but not to the extent that training in the heat does.[137] To condition for high exertion in extreme heat, spend time in environments matching expected temperatures. Ease into activity, gradually increasing exercise intensity and duration. Stay well hydrated. For a single exertion in the heat, pre-cooling with air-conditioning or cold baths attenuates rise in core temperature[20] and provides a margin for increased heat accumulation before overheating occurs.[80] Pre-cooling provides no acclimatization to exercise in the heat and will not prevent heat illness. It is best used when one is already acclimatized. Some people in heat training for ultramarathons use a sauna while wearing heat-trapping clothing. When they travel by automobile, they close windows and turn on the heater, to increase acclimatizing time spent in the heat. They avoid air-conditioning for sleep or rest, and they avoid wearing cooling garments when training. Such training should be approached slowly, with full understanding

and education about health-preserving methods to avoid dangerous dehydration or raising body temperature. To condition for milder heat, spend increasing periods in the target temperature, avoiding air-conditioning. Gradually increase physical activity, closely matching expected conditions.

Cold

To increase cold tolerance and prepare for conditions (see Chapter 4), spend increasingly long times exposed to the temperatures and humidity expected, while training to levels of physical activity at or greater than expected levels. Stay well fed, hydrated, and rested to be able to train hard, as cold tolerance is reduced by calorie deficit[58,78] and exertional fatigue.[206] However, with that knowledge, also train for cold, hungry, and tired conditions.

Cold acclimatization involves at least three adaptations, with the extent of each varying between individuals and with exposure. Shivering begins at lower body temperatures as more heat is generated without shivering.[116] There may be both increased[156] and decreased skin temperature,[48] depending on circumstances and on body location.[97] In some cases, skin blood flow increases to keep extremities warm and to resist cold injury.[157] In other cases, it decreases to reduce heat loss.[158] For example, skin temperatures of Australian Aborigines[28] and Arctic dwellers[104] were found to be lower while they slept than those of the unacclimatized European investigators. It is also the case that different people regulate body temperature to different set points.[106] A third hallmark of cold-acclimatized people is improved ability to sleep in the cold.[26] Cold acclimatization is noted in indigenous people of cold climates—for example, in the African Kalahari, the Australian desert, and Tierra del Fuego in southern Chile. Many sleep outdoors nearly naked in freezing temperatures. When chronic exposure ends, cold adaptation lessens. Cold tolerance decreases with dehydration, lack of sleep, and food deprivation.

The relationship between fitness and cold is not as marked as that between fitness and heat tolerance. However, structural and metabolic changes that occur with exercise training benefit cold tolerance. A fit person tolerates a lower body temperature than an unfit person before onset of shivering, and can generate more heat through shivering.[16] Increased muscle mass in trained athletes increases metabolic heat production ability[17] and insulation, better maintaining body temperature without shivering.[205] Physical fitness allows exercise at a higher intensity to generate heat. Cold tolerance is improved to a greater extent by exercise in cold conditions than from exercise alone. For example, seasonal acclimatization[79] occurs in people working outdoors in the cold and fishermen who dunk their hands in cold water to tend their winter nets.

Altitude

Exercise endurance is initially diminished at altitude (see Chapter 1). An interesting dichotomy of training results. With decrease in work rate and VO_{2max} at altitude, maximal accustomed training levels cannot be reached at altitude, and detraining may result. Athletes preparing for specific events at altitude generally recommend alternative training (see Reduced Oxygen Environments, and Performance Enhancement, later). However, to prepare for wilderness travel at altitude, training at altitude is beneficial.

High-altitude acclimatization adjusts the body to decreased oxygen, a process taking days to weeks. Several physical changes from extended exposure to the hypoxia of altitude are similar to changes induced by exercise training, including elevated hemoglobin[159] resulting in increased VO_{2max}, increased muscle vascularization,[135] increased brain capillary vascularization,[19] more efficient pulmonary gas exchange,[12] and changes in fluid balance.[193] Unlike physical training effects, the process of altitude acclimatization decreases total body water, particularly at altitude in the cold. The osmotic center, which detects blood concentration, resets so that blood is more concentrated[112] as the kidneys excrete fluid during altitude diuresis. Hematocrit increases,[23] improving oxygen-carrying ability to a small extent. Oxygen delivery can be maintained with lower cardiac output. Cardiac output decreases below sea-level resting levels.[73] Acute altitude exposure stimulates the sympathetic nervous system, increasing heart rate[73] and elevating basal metabolic rate above sea-level values.[29] Resting cardiac output and heart rate return to sea-level values when fully altitude acclimatized, although working heart rates remain higher.

Physical fitness does not appear to decrease susceptibility to altitude sickness, and training does not appear to prevent it. However, aerobic fitness to carry out work at altitude is benefited by aerobic training. Water and muscle weight loss is common among climbers at altitude. Stay hydrated and well fed. Exercise to retain muscle mass and athletic ability.

Scuba Diving

Being in good physical condition is beneficial to divers, even though average sport diving is not generally rigorous (see Chapter 70). Fitness reduces risk of several diseases affecting general health, increases tolerance to heat and cold, increases ability to lift and carry gear, delays several common effects of aging, and reduces chance of sudden death from unaccustomed exertion. Physical fitness can improve diving safety and the ability to dive more comfortably, possibly making the difference between a diving trip and a diving accident. Divers with preexisting small lung cysts or end-expiratory flow limitation may be at risk of pulmonary barotrauma, but physical fitness or training does not appear to be a factor in susceptibility.[181] Whether or how much physical condition may relate to risk of decompression sickness is not firmly established. Anecdotally, risk is higher in poorly conditioned divers, although that may relate to lack of strength or swimming ability for handling situations that incur decompression problems. Again anecdotally, higher body fat may predispose to decompression sickness risk, but there is not a firm correlation. It may be that fatter subjects in studies of risk were in poorer physical condition, had higher incidence of fatty blood vessels that compromise circulation, or were older. It also is not known if the risk involves total body fat or percentage body fat. For example, a 120-lb woman with 20% body fat has 24 lb of fat, whereas a 180-lb man with 15% body fat carries 27 lb of fat.

To condition for diving, swim with fins regularly. Wear tanks or simulate tanks by increasing resistance to forward movement by tilting a kickboard to increase presented surface area, and by using devices such as drag suits, webbed gloves, tethers, and other commercial and home-made resistance tools. Maintain speed and finning mechanics. On land, lift, carry, and walk around wearing tanks, weight belt, and other gear. Avoid bending over to pick up gear while wearing tanks, which is injurious to the back. Bend and lift properly using legs. Practice climbing ladders in gear, to simulate boat diving.

PERFORMANCE AND INJURY

The most common wilderness injury is not avalanche, sting, bite, or thermal injury, but the same aches, pulls, strains, and pains that often occur from suboptimal body mechanics and injurious exercise practice.

Avoiding Exercise Injuries

Many common exercises are less effective than desired and may do harm. Other useful exercises can be rendered ineffective or injurious if carried out with commonly accepted but poor ergonomics. One common injurious practice is to make an exercise easier by transferring bodyweight to the joints, rather than doing the muscular work of holding the body in a healthy position. Examples include lifting weight overhead by arching the back and rotating the hips forward, instead of preventing lordotic low back arch by countering the load with abdominal flexion (see Abdominal and Core Muscle Conditioning, earlier); allowing knee and ankle joints to sway inward under load, rather than countering with leg muscle repositioning; craning the neck forward while exercising or lifting; and lack of shock absorption when stepping down from terrain, stairs, or a step bench. Allowing bodyweight to sag into joints instead of using supporting muscles loses the benefit of the exercise, and slowly wears joints. Poor positioning (suboptimal posture) during lifting may be associated with higher rating of exertion.[43] The following paragraphs describe how to exercise while maintaining healthy positioning to avoid injury.

Safer Back Positioning

- Forward bending to pick up a package is injurious to the low back.[166,191] It is just as unhealthy to bend over to lift weights in the gym or to stretch by bending over to touch toes. Spending much of the day rounded over desks, computers, handlebars, and steering wheels, and bent under packs, and then exercising with much forward bending, followed by stretching (knees to chest and touching toes), may stretch the back but at the price of damaged discs and soft tissue, whether done with straight or rounded back.[166] Instead, lift weights standing upright. Use legs to lift. Stretch hamstrings (see Flexibility-Enhancing Techniques, earlier). Instead of forward bending to exercise abdominal and core muscles, as commonly done in conventional abdominal training classes or Pilates classes that emphasize hip flexion, use functional training exercises (see Abdominal and Core Muscle Conditioning, earlier).
- Instead of sitting to lift weights, stand and practice stabilizing and balancing a weight with healthy standing posture.
- When lifting weights, do not allow the low back to arch under the load. The lordotic arch transfers bodyweight plus the weight of the load to the low back.[118] Remove the arch and straighten posture by tucking the hips and ribcage, as if starting a crunch, but without rounding forward or leaning back.

Carrying Loads and Packs. Backpacks and heavy shoulder bags produce bad posture from forward leaning[136] and lordotic arching.[96] However, the injury potential is not the pack or asymmetric distribution of the load[152] but not using torso muscles to counter the pull of the load.[21] Maintain torso posture against the weight of carried objects by consciously using abdominal and back muscles to pull to upright posture.

- Practice using abdominal musculature to counter lumbar lordosis when carrying anterior loads. Pick up a chair, laundry basket, or other commonly lifted heavy object. Notice if you lean back. Instead, tuck hips, using abdominal muscles to bring upper body to upright position.
- For posterior loads like packs, notice if you lean back, hunch forward, or hitch to the side to offset the load. Use the above technique to straighten posture and prevent lordosis (Fig. 86-4).

Figure 86-4. It is not the pack that creates low back arching but the failure to use anterior (abdominal) muscles to counter the posterior pull. When the back is allowed to slouch back into an arch (*left*), upper body weight plus the weight of the pack rests on low back joints (facets). Using abdominal muscles to tuck hips and reduce low back arch shifts weight to the musculature and off the joints (*right*).

- When pushing or pulling heavy loads, or reaching or lifting overhead, use the back-positioning technique to prevent poor low back dynamics. Convert gear to a built-in core-muscle trainer.

Safer Knee Positioning

Letting knees sway inward (excessive valgus knee [genu valgum], or knock-knee) when bending or walking strains the medial knee,[117] wearing on the cartilage[39] and interfering with normal muscle use and kneecap tracking.[180]

- When stepping up stairs and in rocky terrain, step onto the whole foot where possible, pushing through the heel, not the front of the foot. Leaning forward and stepping up onto the ball of the foot increases transfer of bodyweight through the knee joint rather than within the thigh musculature.
- Stand and step with feet parallel, not turning toe-in or toe-out, where terrain allows.
- When stepping down, step lightly. Use conscious muscle control to decelerate. Plant toe first, then heel. Bend knees with descent for shock absorption. Use the thigh muscles of the upslope leg to slow descent.
- Use these techniques for step aerobic and other exercise classes and training.

Safer Neck Positioning

- Looking overhead is a common position during exercise and stretching, and for experiencing the wilderness through stargazing, belaying, rock climbing, scouting, and drinking from a canteen. Two poor positioning habits are common. One is to arch the low back to look upward instead of extending at the upper back and shoulder. This presses upper body weight on the low back, a common contributor to low back ache.[21] To prevent lordotic arching, use trunk and abdominal muscles to tuck ribs and hip. A second common habit is to crane the neck and chin forward when looking upward, and to hyperextend one cervical segment, rather than distributing the extension throughout the upper vertebral column. Chronic hyperextension is a factor in disc degeneration[31]; it shears intervertebral discs between the vertebrae,[133] and it overly lengthens the posterior muscles and longitudinal ligament, which promotes a cycle of overly stretched, weak posterior structures, forward head position, and neck and shoulder pain. Use upper back and neck muscles to lift the head, keeping the chin in. Lift from shoulders and chest without arching the low back.
- Keep shoulders back and ears positioned over shoulders, chin in gently, without strain. Prevent the neck from jutting forward at an angle. Forward head posture commonly results in neck, shoulder, and upper back pain,[74] often mistaken for stress. The forward head also rotates the shoulder forward, interfering with raising the arm and contributing to impingement.[71]
- Test head and neck posture by standing against a wall. Heels, hips, upper back, and the back of the head should all touch without arching your back, or raising or dipping your chin. See the earlier stretching section on stretches for anterior shoulder and chest to remedy tightness preventing straight standing posture.
- Maintain chin in, gently, not rigidly.
- Keep the chin-in, shoulders-back position, particularly when driving or sitting at the desk or computer. Lean back against the chair back. (See Long Sitting in an Automobile, later.)

Avoiding Stretching Injuries

Just as not all foods are beneficial, neither are all common stretches. Many promote poor positioning or impose undue force on joints, deforming or injuring them over time:

- Shoulder stands and the yoga plow position (lying on the upper back with neck bent forward, legs overhead) force the cervical and thoracic spine into hyperflexion. Such pressure on the posterior longitudinal ligament may cause stretch deformation,[204] promoting the common poor posture of forward head and round shoulders. Overstretched ligaments are plastic, not elastic. They do not return to normal length and cannot hold vertebrae in position or function as a firewall to intervertebral discs. The posterior force of the forced flexion under the body's weight may eventually degenerate and herniate cervical discs and promote bone spurs.[166,191]
- A common shoulder stretch involves bending forward with arms lifted behind with clasped hands. Resultant unsupported weighted lumbar flexion promotes disc degeneration and herniation. Forced shoulder hyperextension overloads the constitutionally susceptible anterior shoulder capsule.
- The hurdler's stretch (sitting with one knee rotated and bent laterally) and yoga knee stretches involving lying back on folded, rotated knees forcibly overlengthens the medial collateral ligament. Highly flexing the knee under load also exerts excessive pressure on the kneecap.
- Avoid assisted stretches where anyone presses your back into rounded position or flexion.
- Avoid vigorously oscillating knees (butterfly knees) when sitting with bent knees and soles of feet touching. Do not let anyone stand on or push your knees to facilitate stretching.
- Add extension stretches to reverse thoracic kyphosis. When leaning back to stretch, whether standing, sitting, or lying, keep chin in and do not crane the neck.
- When sitting cross-legged, stretch from the hip rather than inverting the lateral ankle (turning the sole medially or upward). Overstretching lateral ligaments leaves the ankle prone to inversion sprain.
- Stretch muscles, not joints. Do not force joints into such ligament laxity that they no longer seat properly.
- Strengthen, rather than just stretching. Unstable joints slowly wear and tear. In a sudden situation, weak, loose joints are predisposed to pulls or dislocations.

Hard stretching just before an athletic event may reduce the tendinous stiffness that assists elastic recoil in high-exertion situations,[98] reducing maximal force development.[154] Rather than stretching to maximize length before competitive events, use stretches described under Conditioning, earlier, to regain functional muscle length for healthy ergonomics. Similarly, chronically holding a muscle in a stretched position weakens it. Chronic slouching and lengthy sitting weaken the lengthened muscles of the back and posterior hip. Slouching is a stretch, but not a beneficial one. Avoid compounding the problem: avoid forward rounding and bending.

Back Pain Prevention during Long Travel to the Wilderness

Travel to the wilderness can involve sitting long hours while driving or flying. Vehicle seats often have concave backs. Long sitting in lumbar, thoracic, and cervical flexion pressures intervertebral discs and soft tissue. A pillow behind the head exacerbates such flexion. Sitting stresses discs to a higher extent than

does standing, and poor sitting posture amplifies the strain. Neck and back pain from airline seats far exceeds the incidence of deep vein thrombosis. Try the following to train proper sitting mechanics to easily prevent strain:

- It is common to conform low back posture to a concave chair back, pressuring the low back with forward rounding. A lumbar roll pads the space between the chair and the lower back, leading to the proper low back position for healthy sitting without loading the low back. To understand lumbar roll function and appropriate size, sit in a chair with your entire back against the back of the chair. Put your forearm behind you and nestle it in the natural lumbar space between your low back and the chair. Lift your upper back against the chair so that the low back does not press your arm, but rests lightly. It should feel comfortable. Your forearm is usually about the right size for a lumbar roll.
- Make a lumbar roll from a small folded towel, small pillow, or other roll placed between the low back and the chair. Lift the upper back against the chair instead of pressing against the roll. Do not force into unnaturally straight or arched posture. Keep head up, not tilted forward.
- For travel, a small towel, article of clothing, or inflatable pillow are inexpensive lumbar rolls that pack flat and are lightweight.

Long Sitting in an Automobile

- Use a lumbar roll, as just described.
- Car seats are often positioned too far back, encouraging hunching and leaning for the wheel. Slide the seat position forward. Tilt the seat back to a slight backward incline. Sit up and lean back against the seat, instead of craning and rounding.

Buses and Flights

Commercial transport seats are often concave, encouraging prolonged, forced flexion.

- Use two pillows, one in the natural curve of the low back, and the second in the thoracic space still left by the extreme concavity of the seat. Lean back in the chair, instead of forward. Sit upright to rest the back of your head against the headrest. For in-flight sleeping, lean back against a pillow or companion while preventing as much weighted rounding of the neck and low back as feasible.
- Flights sometimes have a video message encouraging in-seat stretching. Often the advice is forward bending. Instead, extension is indicated to counter long flexion. Stretch shoulders back, not forward. Pull the chin in while leaning back. Breathe.
- Press hands to thigh, arch back with chin in, looking upward. Breathe.
- Extend legs as much as space allows, lift hips, and arch the back. Breathe. Try lifting hips without extending legs by pushing on feet and armrests.
- Get out of the seat. Push hips forward to stretch the front of the hip. Tip head to sides to stretch the side of the neck. Try anterior chest stretches and small lunges with tucked hips (see Hips and Thighs, earlier).
- Pump feet. Squeeze knees together and apart, upward and downward against hands. Open and shut hands. Squeeze shoulder blades together. Press the sides of your legs to the seat leg or against your companion for isometric exercise to encourage blood flow in dependent limbs.

Soreness after Exertion

Anyone can be sore after hard activity, not just those who are unaccustomed to exercise. Soreness develops in the days following activity, rather than during activity, so is called delayed-onset muscle soreness (DOMS). Many components are involved in the initiation and consequences of DOMS. Mechanical stress initiates a chain of events. At the time of injury, cell membrane damage in the injured muscle fibers disrupts calcium homeostasis.[6] Abnormal influx of the ion inhibits cellular respiration in the mitochondria. Lack of ATP starves and damages structures, activating lysosomal proteases[41] that degrade and release proinflammatory cytokines[172] and other serum proteins[30] that induce inflammation. Free radicals promote release of arachidonic acid from cell membranes.[165] Arachidonic acid (an omega-6 fatty acid) is converted by cyclooxygenase-2 (COX-2) into proinflammatory prostaglandins,[123] which, along with histamines and kinins accumulated in the interstitium, stimulate nociceptors, causing pain.[6]

The inflammatory response begins the healing process by attracting white blood cells[110] and polymorphonuclear neutrophils.[41] Activation of the complement system may also contribute to mobilization of neutrophils.[32] The inflammatory process is self-regulated by a balance of proinflammatory and anti-inflammatory mediators.[44] The extent and magnitude of the response is determined by the presence or absence of both types of mediators.[123] The result of this balance is that one is stiff, sore, and weak for 1 to 5 days.

Although soreness is more common and pronounced with eccentric contraction (lengthening under tension), soreness results with sufficient stimulation after all types of muscular contraction.[77] Concentric muscular contraction is contraction that shortens muscles—for example, contracting the biceps to lift a load. Concentric contractions against a high load, such as ascending while carrying a heavy pack, creates conditions for soreness among the prime movers of the hamstring, gluteal, and calf muscles. After hard swims, soreness may occur in prime movers of latissimus dorsi and leg extensors, depending on the stroke. Isometric contraction maintains the muscle at one length while under tension—for example, gripping a rock ledge. Soreness after technical climbing is common in wrist and forearm flexor muscles. Eccentric muscular contraction is contraction while the muscle lengthens. When lowering a pack to the ground, biceps lengthen while in a high contractile state, to guide the load at the chosen speed and direction. The high load of eccentric muscle contraction of descending slopes is often felt primarily in the decelerating muscles of the thigh, shins, and ankles, particularly when stepping down hard and not using muscles to decelerate and absorb shock during gait. Use shock absorption to walk and step down lightly.

To prevent delayed soreness, eat healthful food before physical training, as tolerated, to fuel exertion. One study found a single bout of physical activity prevents soreness after exercise bouts for up to 6 weeks,[30] although there are athletes who might state otherwise from their own experience. Sometimes pain is not soreness, but injury from strained muscle fibers from poor posture, efforts above tolerance, or joints twisted into harmful positions. During exertion, if pain, tearing, or pinching is felt in a joint, or a feeling like electricity radiates down a limb, that is probably an injury process. Similarly, if joints feel hot, swollen, or sore after effort, that may be an injury. Identify and change injurious movements to healthy ergonomics. Soreness from carrying packs with poor body ergonomics, such as

forward head, round shoulders, and torso hunched against the load of packs or the cold, is often felt later in the neck, upper back, and shoulders. This pain is from both muscle overuse and misuse, which overstretches muscles while loading them. Keep body position as upright as possible, instead of rounding against the load. Pain from lordotic posture (low back overly arched) under the load of packs is often felt focally in the low back and posterior hip. Prevent lordosis when standing and carrying, by reducing anterior hip tilt (tucking hips under). Do not tighten abdominal muscles—just use them to change vertebral and pelvic angle, similar to changing any other posture. Tight muscles reduce shock absorption and ability to breathe and move properly. Eat as soon after hard exercise as feasible to restock muscle glycogen, preferably within the first 30 minutes, to reduce fatigue on subsequent exertions. Take a hot bath or warm yourself. Reward yourself for the grand effort. After resting, return to activity to become better accustomed to it and induce less soreness on subsequent efforts.

Specific warm-up or stretching before activity does not prevent delayed soreness,[77] but to help alleviate soreness after activity, stretch affected areas gently (see Conditioning, earlier), and keep sore areas moving. Eat food with anti-inflammatory components to help soothe the inflammatory component of delayed soreness. Such foods include leafy green vegetables, cherries, and blueberries, which contain quercetin (a plant pigment belonging to the flavonoids); curcumin (tumeric); ginger; and plant foods containing resveratrol (especially grape skins). Several foods high in essential fatty acids including omega-3 are known for their anti-inflammatory properties—flax seeds, pumpkin seeds, olive oil, and soy products. These are light and easily carried as fresh food or supplements for wilderness travel.

The yellow spice curcumin inhibits two potent proinflammatory mediators, tumor necrosis factor alpha and interleukin-1 (IL-1).[33] Anti-inflammatory properties of resveratrol in grape skins[178] and ginger[105] act via suppression of the proinflammatory catalyst COX-2. Green tea contains the polyphenolic compound GTP, found to inhibit the proinflammatory cytokine IL-1.[127] Salicylic acid is a chemical produced by plants to protect themselves from pathogens, and it is responsible for the anti-inflammatory action of aspirin. Organic vegetables were found to have higher concentrations of salicylic acid (117 ng/g) than were nonorganic vegetables (20 ng/g), possibly because they were less likely to be protected from infection.[11]

Arachidonic acid, a component of human nutrition, is also a precursor of prostaglandins involved in the inflammatory response. Avoid proinflammatory foods that promote arachidonic acid in excess, such as foods containing omega-6 fatty acids, which are converted by COX-2 into prostaglandins[123] and leukotriene B_4.[165] Sources of proinflammatory mediators include peanuts, beef, chicken, and eggs.[165] Another food popularly implicated as proinflammatory is sugar. Diets heavy in inflammatory components may produce what is described as the "diet-induced proinflammatory state."[165]

FITNESS MYTHS

Myth: Walk and run toe-out because that is the natural direction of the foot muscles.

Fact: Limbs are pulled to the toe-out (duckfoot) position from tightness in the plantar fascia, Achilles tendon, and calf, and from weak external rotators of the lateral thigh in a cycle of tightness and loss of the stretch that is normally obtained from straight position during gait. Slight anatomic differences in femoral position usually do not factor in the many degrees of turnout commonly seen. Toe-out walking imposes a high degree of strain on feet, ankles, knees, and hips. It contributes to pronation, bunion formation, and the cycle of tightness. It decreases the push-off phase by pressing medially off the big toe, rather than off the ball of the foot and all toes, reducing speed and jumping ability. Without addressing the cause of deviation in gait, altering a natural stride may lead to other complications. Stretch and retrain gait to keep feet parallel and bodyweight on the rims, not the arches, for standing, walking, jumping, and movement. Give attention to training straight position of the entire kinetic chain, rather than forcing change in a single segment.

Myth: High-top shoes and elastic bracing prevent ankle sprain.

Fact: Main predisposing factors in repeat sprains are overly stretched lateral ligaments and muscles, weak leg muscles, disuse atrophy from "supportive" shoes rather than holding up bodyweight with ankle and foot postural muscles, prior ankle sprains incompletely rehabilitated, and poor proprioception and balance. Prevent ankle sprain with ankle and foot strengthening, but, more importantly, with balance and kinesthetic exercises to retrain foot and ankle positioning (see Feet and Ankles, earlier).

Myth: Women should not lift weights.

Fact: Weightlifting (including body weight) is a health factor for everyone. Women need strength to do daily activities like carrying several 20-lb grocery bags and a 40-lb (or more) child. Extra muscle helps thermoregulation in winter and is the body's chief calorie burner. Weightlifting strengthens bones and is important to osteoporosis prevention. Weightlifting benefits everyone.

Myth: A gym is necessary to get in shape.

Fact: Gyms are artificial substitutes for real life movement. Instead of a treadmill, get out and walk, run, skate, row, and enjoy. Instead of a stationary bicycle, go outside and ride a real bike. Go dancing in a club. Give children piggyback rides. Volunteer at a community garden. Help people with chores. A set of weights is not needed, because you already have one—your body. Move as part of your ordinary day. Use legs each time you bend and obtain free lunges and squats every day. Go out in the fresh air every day, no matter what the weather. Put on appropriate clothing, mentally rehearse your wilderness and climbing moves, and go.

Myth: Women are more prone to lower body injuries than men.

Fact: Several studies have identified training and fitness, not gender, as primary risk factors. Lt. Col. Bruce H. Jones, MD, Chief of the Occupational Medicine Division of the U.S. Army Research Institute of Environmental Medicine, reported on data from army recruits. He found that those of lower physical fitness had a consistently higher injury rate. When comparing men and women of equal aerobic fitness, their rates of injury were similar.[85] In a study of 861 Army recruits, Nicole S. Bell, ScD, MPH, of Boston University stated that the higher rate of injuries among women "appears to be explained by physical fitness" rather than sex differences.[14] More confirming data

came from a 13-year study by Stephen G. Rice, MD, of 60,000 high school athletes in 18 sports. Dr. Rice concluded: "What we have here is not a gender issue, but instead a classic combination of training error and [lack of] physical fitness."[150]

Myth: Carbohydrate is unnecessary and unhealthy, raising insulin and blood fat.

Fact: Insulin concentrations do not rise during exercise in response to carbohydrate as they do at rest, because of glucose transporter recruitment. Triglycerides are also checked by exercise. Simple sugar is physiologically handled differently from the way complex carbohydrates are handled. Simple sugars have been found to be unhealthy for the heart, blood vessels, skin, and eyes. Refined sugar snacks raise blood sugar too quickly and unevenly, and may contribute to mood swings in people susceptible to low-blood-sugar-induced crankiness. Carbohydrate provides energy necessary to exercise and gain skills. The body changes complex carbohydrates into the types and amounts of sugars needed. Diets excluding complex carbohydrates do not provide the chemical energy to do exercise, nor do they provide the calming compounds, important vitamins, or cancer-fighting phytochemicals found only in fruits and vegetables. Eat legumes, vegetables, and whole fruit for healthful carbohydrate. If you eat grain, emphasize whole grain and oats (without added sugar), instead of "junk" sugar foods like white bread, white rice, donuts, pretzels, and breakfast cereals. Wash foods with edible skins (apples, pears, mangos, and sweet potatoes), and eat the skin too. The skin has fiber, which helps maintain even blood sugar and insulin, and has important nutrients. Drink juice with the skin and pulp, or you'll be drinking little more than sugar water. Avoid sweetened juices. Eat the seeds when you prepare squash and pumpkin. Avoid soda (pop) beverages; the phosphorus in them demineralizes bones. Do not be fooled by ingredients called "natural sugar" or "no sugar added." Fructose, galactose, corn syrup, rice syrup, and high-fructose corn syrup are simple sugars, not a healthy alternative. Do not be fooled by junk food labeled "sugar free." A block of butter is sugar free (and a bag of sugar is fat free). Do not eat donuts and Danish. Refined sugar is like distilling alcohol to unnaturally high proof. It is overly concentrated, and in an unhealthy form. If you safeguard health with avalanche preparedness and bug repellent, do not undo gains by eating junk that injures health. For quick energy, eat fruit, not candy. Increase carbohydrate at altitude to offset muscle glycogen loss. Reduce the proportion of carbohydrates in meals when not exercising. No need to substitute fat or protein; less food is needed when not exercising. When exercising, eat healthful meals with complex carbohydrates for energy.

Myth: Devices can improve aerobic fitness by raising heart rate or blood flow without exercise.

Fact: Several things increase heart rate without benefiting cardiovascular status. Claims have been made that standing on your head, massage, or lying head-down improves circulation to the head, or that heating the body or holding a difficult yoga stretch increases heart rate, conferring aerobic benefit. Aerobic conditioning involves more than increasing blood flow and heart rate. Stage fright and moving the legs of a person lying in bed raise heart rate, but these are neurogenic increases, produced as a nervous system effect, not cardiogenic. Raising body temperature through a hot shower or tub or sweating in a sauna suit increases heart rate through skin vasodilation to dump extra heat. Blood pressure decreases, with consequent increase in heart rate to maintain pressure. There is no aerobic benefit to overheating or sweating, and caloric expenditure is not raised. Improve aerobic ability through long-duration, moderate-to-high-energy exercise like biking, swimming, rowing, skating, dancing, and skiing, repeated regularly over time.

Myth: Older people need less exercise.

Fact: Exercise becomes more necessary and protective with aging. Deconditioning over years is often confused with aging. Regular exercise, stretching, and balance practice slows the progression of decline, or can even confer gains equivalent to stopping or reversing years of aging. Older people who exercise, move, balance, and stretch have many of the functional characteristics of a chronologically younger person. They can reach, bend, and move easily, negotiate uneven sidewalks and other terrain, and carry their own gear. The anti-aging effect of exercise is effective in all age groups. Geriatric populations on exercise programs considerably improve strength and mobility. Physical gains often eliminate the need for walkers, wheelchairs, and canes, and self-sufficiency returns. Many people do not kneel or sit on the floor because they are too weak to get back up. That is not aging, but the need to regain strength to do it. Exercise and skill practice are the key to independence and retention of physical ability.

PERFORMANCE ENHANCEMENT

Performance-Enhancing Drugs and Nutrients for Exercise and Expeditions

Foods to increase physical ability have been sought throughout history. Indigenous and mountain people have long used stimulant plants like coffee, kola, khat, betel nut, and coca to withstand cold, hypoxia, hunger, and fatigue. Aztec warriors hoped to increase bravery by eating the hearts of brave enemies. Berserkers, Norse warriors legendary for savagery and reckless frenzy in battle, ate *Amanita muscaria* mushrooms. Dervishes whirled longer using coffee. Before the first Olympic games in 776 BC, athletes used wine to enhance performance by dulling pain, while concoctions of wine and substances from strychnine to cocaine were used in later Olympic games. Amphetamines were long given to soldiers to delay fatigue, and were widely used in medical practice until recently when their dangers became widely known. Today, ergogenic aids include stimulants, muscle growth promoters, energy substrates, and oxygen utilization enhancers. They range from healthy to illegal.

Methylxanthines. Methylxanthines are a class of stimulant alkaloids found in plants, including coffee, tea, cocoa, guarana, yerba mate, and kola (cola). They may possibly be humankind's most commonly consumed drugs. Animals also seek them out. In South America, animals chew coca leaves, and goats in the Middle East are credited with showing the effects of eating coffee seeds to humans. (Although popularly called coffee beans, coffee seeds look like, but are not, true beans.) Methylxanthines are purinergic, acting on purine neurotransmitters such as adenosine.[162] They elevate cyclic adenosine monophosphate (cAMP) in several tissues, including the brain, with stimulant, diuretic, and vasodilator properties.[82] All xanthines have a similar stereochemistry, but each has unique properties. In

various products promoted for health and energy, they are often combined ("stacked"), yielding compound effects.

Caffeine. Caffeine is the primary methylxanthine in coffee and kola nut, from which many cola sodas are made, and is present in smaller amounts in chocolate and tea. It was probably discovered in the Stone Age, and has been widely used ever since for its several effects. Caffeine increases endurance and is particularly used for long endurance events.[68] How caffeine helps athletic performance is still studied and debated. It is thought to help endurance by directly stimulating the central nervous system, increasing plasma epinephrine,[151] which enhances excitation–contraction coupling mechanisms.[179] Some work shows epinephrine induced enhanced free fatty acid oxidation for fuel,[146] which in turn spares muscle glycogen,[175] whereas others dispute lipid mobilization by caffeine[67,187] and show ergogenic effect of caffeine without an increase in plasma epinephrine.[66,187] Caffeine decreases perception of effort, perhaps through adrenocortical axis stimulation,[102] which promotes cortisol and beta-endorphin release.[42,101] The caffeine molecule is similar to the depressant molecule adenosine. As a competitive antagonist of adenosine receptors, caffeine replaces adenosine.[177] Reported effects on short-duration intense anaerobic activity are inconsistent about whether power or endurance is aided. Positive effects are attributed to increased motor unit recruitment[108] and resistance to fatigue through mobilization of intracellular calcium from the sarcoplasmic reticulum.[179] Although caffeine in isolation increases endurance, some work that suggests that the effects do not extrapolate fully to coffee, perhaps due to components present in coffee that moderate the effect.[68] Caffeine with phenylpropanolamine (PPA) interacts adversely, increasing the risk of hemorrhagic stroke.[25] PPA is used as a decongestant and weight-control drug. The U.S. Food and Drug Administration (FDA) banned this drug combination in 1983. Habitual caffeine users do not derive ergogenic effects from usual amounts of caffeine. Dependence and withdrawal symptoms, often severe, are common.

Theophylline. Theophylline is the methylxanthine in tea leaves, along with a small amount of caffeine and theobromine.[65] Theophylline is a smooth muscle relaxant, diuretic, cardiac stimulant, and vasodilator; it is used to treat asthma and other obstructive pulmonary disease. Theophylline delays skeletal muscle fatigue[113] and is thought to increase aerobic endurance through increased blood glycerol levels without increase in plasma epinephrine.[70] A study of Mt. Everest basecamp expeditioners found no evidence that tea acts as a diuretic in regular tea drinkers, even at high altitude where fluid balance is stressed, and that it exerts a positive effect on mood.[164]

Theobromine. Theobromine is the primary methylxanthine in chocolate. Other compounds in cocoa include caffeine, serotonin, histamine, tryptophan, tryptamine, tyramine, phenylethylamine, octopamine, and anandamide.[144] Theobromine is found in smaller quantities in the kola nut. It is a weak diuretic, bronchodilator, and stimulant, and it may increase mood and motivation to work.[129] Dark chocolate contains higher levels of theobromine than lighter chocolate, along with substantial amounts of flavonoids and phenolics, possibly good for the heart. The healthiest way to use theobromine on the trail is in unsweetened cocoa, rather than in sweetened candy.

Guarana. Guarana is made from the crushed seeds of *Paullinia cupana,* a South American vine. It is considered to be potent caffeine (often called herbal caffeine), with similar effects.[3] However, guarana may not contain caffeine but rather the isomer guaranine.[15] Coffee contains between 1% and 4% caffeine. Guaranine is reported to either contain or be the equivalent of 5% caffeine. It also has lower amounts of the methylxanthines theophylline and theobromine.[149] Guarana is frequently found in herbal or nutritional supplements and has been used for energy and headache relief. Use is linked to unpleasant overstimulation, and withdrawal weakness and depression in an unhealthy cycle.

Yerba Mate. Yerba mate is a South American tea-like beverage made from mate *(Flex paraguariensis),* an evergreen holly. Among other plant substances and vitamins, mate contains a small amount of xanthines, primarily mateine, and smaller amounts of theophylline and theobromine. Mateine is a simple stereoisomer of caffeine, but it has distinct effects. Like other xanthines, it relaxes smooth muscle airways and peripheral blood vessels and is a mild diuretic. Central nervous system effects are disputed.[54] Although occurring in only minute amounts, the xanthenes in mate attract much speculation regarding possible benefit. Anecdotal claims are that persons with caffeine sensitivities seem able to drink mate for stimulation without insomnia and irritability. The cytotoxic and antioxidant activity of components in mate is speculated to confer possible anticancer benefit,[145] but at least three studies link mate consumption with oropharyngeal or esophageal cancer.[62,63,168] Confounding variables make this still unspecific.

Cholinergic Stimulants (Tobacco, Areca, and Lobelia)

Cholinergic agents stimulate nerve cells or fibers that employ acetylcholine as neurotransmitter. They cause cardiac inhibition, vasodilation, gastrointestinal peristalsis, and other parasympathetic effects.

Nicotine. Nicotine is an alkaloid found in various plants, especially tobacco. It is poisonous, but in small doses is used for its stimulant effects. These effects have long been used to combat hunger and fatigue in various difficult environments, although nicotine has not been specifically found to enhance physical ability. It has mixed effects because it stimulates both sympathetic and parasympathetic systems.[209] It is used in various forms, usually as chewing or smoking tobacco, although nicotine patches and gum have been used to try to boost alertness, each with intrinsic health effects.

Betel Nut. Betel nut *(Areca catechu)* is a mild stimulant habitually chewed by many millions of people, primarily in Asia. It contains the alkaloids arecoline and guvacoline. Chewers, known by their reddish black teeth, primarily chew the areca nut wrapped in betel leaf, with mineral lime often added as a catalyst. The lime hydrolyzes the arecoline and guvacoline into arecaidine and guvacine, which are strong inhibitors of gamma-aminobutyric acid (GABA) uptake. Aromatic phenolic compounds stimulate release of catecholamines. As a result of complex interactions, betel chewing affects parasympathetic, GABAergic, and sympathetic functions.[38] The dilute plant alkaloids are chewed over hours and slowly absorbed through the

mucous membranes of the mouth to furnish a subtle stimulation and not-so-subtle parasympathetic salivation.

Lobelia. This plant, also called asthma weed, wild tobacco, pukeweed, and vomit wort, is another parasympathetic agent used in folk medicine to aid breathing, and more recently as a source of chromium.[109] It is found in several "health" tonics, with side effects reported to be breathing difficulty, rapid heartbeat, low blood pressure, diarrhea, vomiting, dizziness, and tremors.

Sympathomimetic Stimulants-Monoaminergic (Amphetamines, Coca, Khat, and Ephedra)

Sympathomimetics mimic the sympathetic nervous system and stimulate release of noradrenaline and adrenaline. They enhance alertness and reduce hunger and mental and physical fatigue. Examples are amphetamines, coca, khat leaves, and ma huang (ephedra). Overstimulation is common, with addiction to some resulting in various unhealthy effects.

Amphetamine. Methamphetamine (Methedrine), levoamphetamine (Benzedrine), and dextroamphetamine (Dexedrine) are collectively known as amphetamines. Amphetamines are intensely stimulating, and this effect is followed by intense withdrawal depression, fatigue, and rebound appetite as neuronal dopamine stores, peaked by use,[37] are depleted in the mesolimbic pleasure center of the brain.[9] Development of amphetamines in the United States was first supported as a substitute for Chinese ephedra. Amphetamines were once prescribed to soldiers and widely used in clinical medicine to combat weight loss, asthma, depression, Parkinson's disease, hyperactivity in children, and travel sickness. They have been used for their alerting, enjoyable effects, and to reduce hunger and fatigue. Users may experience abnormally high or irregular heart rates, raised blood pressure, and, sometimes, mental states resembling paranoid schizophrenia. Abdominal cramps, incoordination, dizziness, dry mouth, nausea, and vomiting may also accompany initial use. Chronic users are found to have structural deficits in several brain areas.[182] Acutely, amphetamines increase risk of overheating by two mechanisms: increased endogenous heat production and decreased heat dissipation due to peripheral vasoconstriction.[93] Deaths in the heat have resulted in endurance athletes and in soldiers,[69,192] and in a 12-year-old taking appetite suppressants.[93] Amphetamines usually physically and psychologically addict the user after about 12 weeks, with abusers increasing doses from 10 to 1000 times to retain effects. Although amphetamines extend endurance, they are counterproductive to healthful wilderness travel.

Coca. The leaves of the South American shrub *Erythroxylon coca* contain from 0.1% to 0.9% of the alkaloid cocaine. Cocaine is a powerful cardiovascular stimulant, blocking catecholamine reuptake and prolonging effects of released neurotransmitters.[115] Unresequestered norepinephrine increases blood pressure[184] and arrhythmias.[72] Resulting catecholamine depletion leads to intense depression (cocaine crash).[94] Sudden heart attack may occur in an otherwise healthy person.[99,142,188] Until the early 1900s, cocaine was widely sold over the counter in tonics, toothache cures, and patent medicines. Combined with alcohol, cocaine alkaloid yields cocaethylene, a potently reinforcing compound, becoming a popular ingredient in wines like Vin Mariani. As an export drug, cocaine is extracted as a cocapaste from soaked, mashed coca leaves, often in the form of cocaine hydrochloride salt with high percentages of cocaine. Addictive and socially and medically unhealthy, cocaine is unsuitable and profoundly unwise for athletic efforts.

Khat. Khat grows in eastern Africa and southern Arabia. It is frequently chewed for amphetamine-like stimulatory effects and to reduce fatigue and appetite. Khat releases catecholamines from presynaptic storage.[90] Leaves of the khat bush contain the alkaloid cathinone, which functions like amphetamine, and a milder form of cathinone, cathine,[196] one of the alkaloids found in ephedra. Cathinone increases blood pressure, heart rate, and psychostimulation.[24] Both cathinone and cathine are controlled substances (cathinone is Schedule I and cathine is Schedule IV).

Ephedra (Ma Huang). Ephedra is a plant native to Pakistan, China, and northwestern India. The three ephedra species, *Ephedra sinica, Ephedra equisetina,* and *Ephedra intermedia,* are collectively known by their Chinese name, ma huang. The ephedra plant has two main active compounds: ephedrine (2-methylamino-1-phenyl-1-propanol) and pseudoephedrine.[194] Ephedrine and pseudoephedrine are both sympathomimetic, with effects similar to amphetamine but to a smaller degree. Unlike pseudoephedrine, ephedrine mediates effects through circulating epinephrine[49] and is a bronchial dilator used in treating asthma. Many now-banned "energy" and weight loss products contained one or more of the several forms of ephedra. Ephedrine can have several nervous system effects, such as insomnia, tremors, anxiety, and seizures.[140] Effects do not always depend on dosage. Ephedra is associated with a small number of cases of cardiovascular toxicities attributed to sympathomimetic adrenergic effects, including myocarditis, arrhythmias, myocardial infarction, cardiac arrest, and sudden death.[131] The effect to increase blood pressure is postulated as a factor in increased risk of stroke.[124] Ergogenic effects are debated and risks do not seem to show that ephedra is advantageous for wilderness travel. The FDA banned ephedra in December 2003.

Other Stimulants

Ginseng. Ginseng collectively refers to several different plant species from different countries, each with possibly differing effects. Main active agents are ginsenosides, which are triterpene saponins. It is usually the root that is used, with active agents varying with species of the plant, age, location, and method and timing of harvest and processing. Because of ginseng's adaptogen and stimulant qualities, it is used in numerous candies, pills, and drinks, with numerous claims. Investigations have found that many ginseng preparations contain other stimulating compounds, usually caffeine, and that ginsenoside content varies greatly, with some preparations containing none at all, making interpretation of claims difficult. Although some animal studies show effect, of many human studies, there does not seem to be ergogenic effect on oxygen consumption, respiratory exchange ratio, minute ventilation, blood lactic acid concentration, heart rate, perceived exertion,[50,125] exercise time, workload, plasma lactate, or hematocrit.[4] A study reviewing clinical trials showed no beneficial effect on physical performance, psychomotor performance, cognitive function,

immunomodulation, diabetes mellitus, or herpes simplex type II infections.[189] Excess may result in diarrhea, nervousness, blood glucose changes, and insomnia. Ginseng may potentiate or interact adversely with caffeine, phenelzine (Nardil),[84] warfarin (Coumadin),[185] and monoamine oxidase inhibitors.[121]

Anabolic Steroids

Anabolic steroids are controlled drugs with growth promoting and androgenic effects. They improve athletic performance when strength is a primary component, and when used with intense training programs and adequate nutrition. For ergogenic effect, anabolic steroids are taken at 10 to 100 times the therapeutic dose for medical conditions. A large black market exists for controlled medical and veterinary anabolics like Trenbolone, used to increase weight of cattle. A related steroid is tetrahydrogestrinone (THG), a designer anabolic steroid derived from gestrinone, a European drug for endometriosis. Several popular steroids have progestogenic activity. Others are aromatizing (synthesize estrogens), including methandrostenolone (Dianabol), boldenone (Equipoise), and to a lesser extent fluoxymesterone (Halotestin). Users seeking to avoid estrogenic side effects or recover depressed testosterone production after a "cycle" of use, may "stack" (combine) antiestrogens (aromatase inhibitors or receptor blockers) such as aminoglutethimide (Cytadren) and clomiphene (Clomid). The pattern of increasing a dose through a cycle is referred to as pyramiding. The risk for injury increases, as tendons and ligaments do not strengthen at the same rate as muscle. Side effects can be common and injurious. One study indicates that reports of hepatotoxicity may be overstated[45]; however, other adverse effects of anabolic steroids seem to be documented, including apoptosis of skeletal muscle,[2] cardiovascular damage,[120] decreased endogenous androgen production and testicular atrophy,[95] thrombotic phenomena such as strokes, myocardial infarctions, and limb loss,[52,53] uncontrolled behaviors,[7] and dependency behavior and withdrawal depression.[92]

Anabolic (Growth-Promoting) Nonsteroids

DHEA. DHEA (dehydro-3-epiandrosterone) is a weak intermediate steroid, the most abundant naturally made in the body. DHEA is manufactured mostly in the adrenals from cholesterol, and to a smaller extent in the ovaries and testes. DHEA converts to dozens of hormones, including estrogen and testosterone in both men and women. DHEA levels wane with age and some illnesses, leading to the question of whether restoring those levels can mitigate effects of age and disease, even though it is unknown if lowered DHEA causes such problems. Although DHEA is commonly purchased in hopes of enhancing strength, gains are not confirmed in controlled investigation.[88] The estrogenic potential seems to produce results in the laboratory for women to restore lost bone and stopped menstrual periods. Before taking DHEA, one should have blood levels checked for deficiency.

Androstenedione. Androstenedione ("andro") is made in the body from DHEA. Androstenedione is a precursor molecule (prohormone) of both testosterone and estrogen. Which hormone pathway it takes depends on several factors, including percentage of body fat: a high percentage of body fat increases estrogenic potential. Androstenedione is accepted as an aromatizer, being readily converted to estrone. Popularity seems to stem from its being a precursor to testosterone. For perspective, cholesterol is also a precursor molecule to testosterone, but eating it does not increase testosterone. Reports from gyms and andro manufacturers state that athletic ability is always greatly helped. Studies do not find ergogenic potential.[88] Some studies show increased strength, but no more than in control groups with matched exercise without andro. Another study found no increase in plasma testosterone and no anabolic effect on muscle protein in young men.[147] Supplementation may suppress endogenous production. Over-the-counter androstenedione supplements have sometimes been found to have other elements not on the label that can cause a positive drug test for other banned substances.

Creatine. As quickly as ATP is used for fuel in cells, it is rebuilt for more activity using phosphocreatine, stored mostly in skeletal muscle, and a smaller amount of free creatine. Use of supplemental creatine to fuel intense, short efforts is behind sales of creatine monohydrate, the synthetic form of endogenous creatine phosphate. Creatine seems to assist repetitive, high-intensity, short-term efforts with brief recovery.[186] Whether it is an important adjunct for extended exercise is debated. Individual variation is wide,[89] which may be why some studies and some people find results and others do not. Advice to eat steak, fish, and pork, which contain creatine, is misguided because creatine denatures once cooked. Creatine (methylguanidine-acetic acid) is an amino acid made in the liver, pancreas, and kidneys from arginine and glycine, which are obtained from balanced meals, including vegetarian meals. The ergogenic effect of creatine supplementation seems to be counteracted by caffeine.[186] Long-term effect of supplementation is not known.

Foods

Carbohydrate. Ingested and stored carbohydrate is ergogenic, providing fuel and delaying fatigue during intense, long-duration exercise, and aiding recovery after exercise.[197] The larger the body stores, the longer exercise can be extended before fatigue, depending on fitness level. Depletion of blood glucose and stored carbohydrate is related to fatigue and reduced athletic ability.[190] Storing muscle glycogen is accomplished by hard physical training and restocking muscles by eating healthy carbohydrate, preferably within the first 30 minutes after a bout of exertion.

Vitamins and Minerals. No evidence supports vitamin or mineral supplementation as ergogenic. Supplementation is useful when nutritional deficiency exists,[201] such as for athletes in weight-restricted events, or for extended and extreme wilderness travel with inadequate nutrition. However, increasing intake will not improve physical ability with adequate nutrition. Oversupplementation may alter utilization of other micronutrients.

Bee Pollen. Bee pollen claims include various effects, from vague vitality to specific athletic improvements. Bee pollen has nutritional components, but it has not been experimentally shown to enhance any aspect of athletic performance[203] or to have any effect on level of physical stimulation.[81] It may provoke allergic reactions in susceptible people.

Water

An often overlooked athletic ability aid is water. Although possessing no ergogenic chemical or property, water is a necessary,

inexpensive, and easy fluid replacer for healthy functioning during exercise and daily activities. It aids heat balance and recovery from exercise. Drinking too much will not enhance health, and in extremes, can result in dilutional hyponatremia (see Chapter 64).

Fitness Water, Fitness Carbo and/or Protein Drinks with "Buzz." Many packaged "health" and sports drinks contain stimulants of all kinds—ginseng, guarana, and caffeine among them. These are advertised as energy drinks and vitality enhancers. Check the label. Remember that they are stimulants with associated health risks. Also note if they contain refined sugar, hydrogenated fat, and fillers that are equally problematic.

Blood Doping

Doping involves physiologic methods of altering body chemistry to increase athletic ability.

Transfusion Doping. A major performance-limiting factor in endurance sports is maximal oxygen uptake. Transfusion doping is the practice of increasing red blood cell count by transfusion. Transfusion doping is (illegally) used to increase hemoglobin and VO_{2max},[61] increasing ability to perform submaximal and maximal endurance exercise,[169] and to reduce physiologic load during exercise in the heat. In autologous transfusion doping, one to four units of the athlete's blood is removed approximately 6 weeks before an event. The blood is centrifuged, and the plasma components are immediately reinfused. Remaining red cells are stored to be reinfused 1 to 7 days before the event. Homologous blood doping uses blood obtained from relatives or friends. Problems have included rejection and disease transfer. Effects of doping are heightened by training at high altitude.

Cell-Generating Hormone Doping. Hormonal blood doping involves injecting synthetic human erythropoietin (EPO), the hormone naturally secreted during the renal hypoxia of hard aerobic exercise or exposure to altitude. Darbepoetin (darbepoetin-alpha), an erythropoiesis-stimulating protein with a longer half-life than EPO, has also been used.[195] Administration increases reticulocytes, red blood cells, hemoglobin, and hematocrit.[173] The drugs can increase cell counts to a dangerous level, leading to cardiac problems. Increased concentration of cells can increase blood viscosity and decrease cardiac output, blood flow velocity, and peripheral oxygen content, paradoxically reducing aerobic capacity. Doping can be a cumbersome and unhealthy process and has occasionally proved fatal.[55]

Artificial Oxygen Carriers. Artificial carriers are solutions based on recombinant, bovine, or human hemoglobin or perfluorocarbon emulsions. The carriers improve oxygen delivery to muscles and aerobic exercise capacity. These substances have potentially lethal side effects, including renal toxicity, increased blood pressure, and immune depression.[163]

Soda Doping. Soda doping (or soda loading) involves drinking concentrated sodium bicarbonate (baking soda) or sodium citrate before short intense activity, in an attempt to delay muscle fatigue. During hard exercise, lactic acid in working muscles builds, fatiguing the muscles. It is hypothesized that the alkaline sodium bicarbonate neutralizes lactate. Studies find extension of time to exhaustion[86] and increased speed over short distances.[64,199] Although in endurance events muscles accumulate less lactic acid, soda doping may possibly enhance endurance.[143] Common side effects are diarrhea, cramps, bloating, and thirst, all of which can inhibit performance.[119] Drinking extra water may alleviate the problem, but soda doping does not seem to have benefit for wilderness activities.

Reduced Oxygen Environments. Red cells increase in response to the low-oxygen environment of high altitude. The expectation is that by living at altitude, athletes improve oxygen-carrying capacity and sea-level athletic ability. However, because their ability to exercise at altitude is lower, improved sea-level performance has not been noted. Various strategies have been proposed to combine the advantages of high altitude with sea-level training levels.

Counter to the climb-high, sleep-low dictum for avoiding altitude sickness is the live-high, train-low motto, whereby athletes live and sleep at high altitude in an attempt to improve athletic hemodynamics, and then drive to lower elevations only to train. Because of operational and cost concerns of constant travel, various in situ training analogs have been developed. "Altitude houses" were developed by Finnish scientists in the early 1990s. Nitrogen was pumped into a structure to reduce oxygen percent from 21% to 15%, simulating altitudes above 9000 ft (2700 m). Smaller, individual versions became "altitude tents" or "nitrogen tents." Nitrogen–oxygen masks worn during workouts have been used in hopes of achieving the same effect.

Sleep-high, train-low is another method hoped to raise red cell count, thereby increasing aerobic capacity. Athletes sleep at altitude high enough to stimulate endogenous erythropoietin, and then return to sea level to train, limiting altitude exposure time to the hours of nightly sleep. Athletes either drive to altitude to sleep and return to sea level to train, or they stay at sea level and sleep in commercial hypoxic (low-oxygen) chambers, and then emerge to train each day. The low pressure chamber reproduces the reduced air pressure at high altitude, without requiring a physical move to altitude. It is not yet known how little time is needed at altitude for intermittent exposure to produce sufficient effect on exercise potential. Critics state that hypoxic enclosures are ethically not different from the banned practice of doping.

Supplemental Oxygen. Oxygen administered during endurance exercise increases performance by assisting hydrogen ion removal during the hypoxia of hard exercise (see Aerobic and Anaerobic Conditioning, earlier) or high altitude. Such oxygen would have to be carried in a tank on a vehicle or on the athlete's back. There is no evidence that oxygen administration (e.g., as breathed by football players) hastens recovery after intense or prolonged exercise.

Alerting or Wakefulness Adjuncts. Products may increase alertness or stimulation without augmenting physical ability to do work and thus may be represented as ergogenic without being work-enhancing (e.g., nicotine). Some, like amphetamines, are globally stimulating and then require recovery sleep, becoming counterproductive in settings requiring health, not solely a time or score in a competition. Newer products such as modafinil (Provigil [U.S.] or Alertec [Canada]) and adrafinil aim to focus on wakefulness and vigilance by targeting specific sleep-promoting areas of the suprachiasmatic nuclei,[160] without

binding to dopaminergic or adrenergic receptors.[35,107] These drugs are labeled for narcolepsy, with the intended use being wakefulness during an ordinary day, followed by a normal night's sleep. Off-label use as a lifestyle drug, to extend wakefulness for stretches of 40 hours and more without the need for recovery sleep, seems to be increasing among those seeking advantage in an ultramarathon, the military, or a competitive workplace. Prolonged sleep deprivation is known to depress immune function. Animals in experiments and humans deprived of sleep by torture have psychotic effects and die from infections. The effects and safety of these drugs over extended periods is not yet known.[10]

FURTHER READING

Bookspan J: Fix Your Own Pain Without Drugs or Surgery. Monterey, CA, Healthy Learning, 2006.
Bookspan J: Healthy Martial Arts. Philadelphia, Neck and Back Pain Sports Medicine Press, 2006.
Bookspan J: Stretching Smarter Stretching Healthier. Philadelphia, Neck and Back Pain Sports Medicine Press, 2006.
Bookspan J: The Ab Revolution, 3rd ed. Philadelphia, Neck and Back Pain Sports Medicine Press, 2006.
Bookspan J: Health & Fitness in Plain English, 2nd ed. Monterey, CA, Healthy Learning, 2002.
Bookspan J: Diving and Hyperbaric Medicine Review for Physicians. Kensington, MD, Undersea and Hyperbaric Medical Society, 2000.
Bookspan J: Diving Physiology in Plain English, rev. ed. Kensington, MD, Undersea and Hyperbaric Medical Society, 2006.

The references for this chapter can be found on the accompanying DVD-ROM.

Children in the Wilderness

87

Judith R. Klein

Once the realm of a few adventurous individuals, the wilderness today attracts an ever-broader range of explorers. This includes many in the pediatric age group, as parents seek to share the joys and lessons of wilderness travel with their children. In 2002, of the estimated 15.5 million people participating in backpacking and wilderness camping, nearly 25% were under 17 years of age.[79] Millions of other children annually visit national parks and recreation areas.

Wilderness travel with children requires special preparation and places extra demands on parents. However, it also affords unique opportunities. Parents and children interact in a setting distant from the stresses of work and school. Isolated from the distractions of television and modern life, children experience new environments, interact with individuals of different cultural heritage, and participate in activities that enrich their lives. These activities bring families together as they learn to rely on one another for support and entertainment.

Physicians and other health care professionals can encourage and facilitate such undertakings by providing preventive health and treatment guidelines for families planning wilderness travel with children. This chapter focuses on how children differ from adults and how to prevent, recognize, and treat the medical problems children are likely to encounter in a wilderness setting.

Wilderness travel may also involve travel to foreign countries. The number of families traveling outside the United States, particularly to developing countries, is on the rise, as is the number of families relocating abroad for prolonged periods. This chapter also reviews risk avoidance techniques, pediatric travel immunizations and prophylaxis, and common pediatric medical problems seen during travel to and within developing countries.

WHAT MAKES CHILDREN DIFFERENT?

Size and Shape

Children are distinct from adults in a variety of physical, physiologic, and psychological ways. The most obvious difference is size. During development, children may grow from the average 7-lb baby to a 140-lb adolescent, a 20-fold difference. Accordingly, medications and fluids must be calculated on an individual basis, based on the weight of the child. Table 87-1 lists average weights for age.

This variation in size also influences a child's risk of developing serious complications from envenomations. Many snakes, spiders, scorpions, and poisonous marine animals deliver the same dose of venom regardless of the size of the victim. Chil-

binding to dopaminergic or adrenergic receptors.[35,107] These drugs are labeled for narcolepsy, with the intended use being wakefulness during an ordinary day, followed by a normal night's sleep. Off-label use as a lifestyle drug, to extend wakefulness for stretches of 40 hours and more without the need for recovery sleep, seems to be increasing among those seeking advantage in an ultramarathon, the military, or a competitive workplace. Prolonged sleep deprivation is known to depress immune function. Animals in experiments and humans deprived of sleep by torture have psychotic effects and die from infections. The effects and safety of these drugs over extended periods is not yet known.[10]

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dren often experience greater toxicity because of the increased dose of venom per kilogram of weight.

Children are not only smaller than adults; they also have a larger surface-area-to-mass ratio. For example, a 7-lb infant has 2.5 times more body surface area per unit weight than a 140-lb adult. Not only is the surface area of the body larger but also the head, which is the part of the body most often left exposed, takes up a larger proportion of the body in a young child (Fig. 87-1). As a result, children experience greater exposure to environmental factors, such as cold, heat, and solar radiation. They are also more likely to suffer toxic effects from topical agents, such as medications.

Musculoskeletal System

The musculoskeletal system in children differs from that in adults in several important ways. A child nearly doubles in height between birth and 2 years, and again between 2 and 18 years. In some respects, this rapid growth makes a child's bones much more forgiving. Because of the active osteogenic potential of the periosteum, nonunion or permanent angulation deformities at the metaphysis are unusual in children. For the same reason, fractures in the pediatric population heal quite rapidly. For example, a fractured femur typically heals in 3 weeks in a newborn, compared with 20 weeks in a 20-year-old. The strong, pliable periosteum also permits the development of greenstick and buckle fractures, which are not seen in the adult population. These fractures with their intact periosteum are quite stable, with little swelling or crepitus. If nondisplaced, they are often incorrectly dismissed as sprains.

Another key difference between the musculoskeletal systems of children and adults is that children have an open growth plate, or physis, at the ends of long bones. The physis connects the metaphysis to the epiphysis and consists of soft cartilaginous cells that have the consistency of rubber and act as shock absorbers (Fig. 87-2). They protect the joint surfaces from suffering the grossly comminuted fractures seen in adults. However, because the growth plate is more vulnerable to injury than the strong ligaments or capsular tissues that attach to the epiphysis, a true sprain in a child is rare. Any significant juxtaarticular tenderness in a child should be assumed to be a growth plate injury. Such an injury is most common at the ankle (lateral malleolus), the knee (distal femur), and the wrist (distal radius). Physeal fractures have been classified into five Salter-Harris groups (see Figure 87-2). Salter-Harris I and II fractures generally heal without complications. However, Salter-Harris III and IV fractures often require open reduction of displaced fractures to realign the joint and growth plates and to permit normal growth. A Salter-Harris V fracture has a poor prognosis; the impaction and crushing of some or all of the growth plate may result in a bony bridge that inhibits further growth or causes unequal, angulated growth. Consequently, any significant injury, especially if it involves the growth plate, requires full evaluation in a medical facility.

TABLE 87-1. Average Weight for Age

AGE (yr)	WEIGHT	
	kg	lb
1	10	22
3	15	33
6	20	44
8	25	55
9.5	30	66
11	35	77
13	45	100

From U.S. Centers for Disease Control and Prevention National Center for Health Statistics (www.cdc.gov/nchs/).

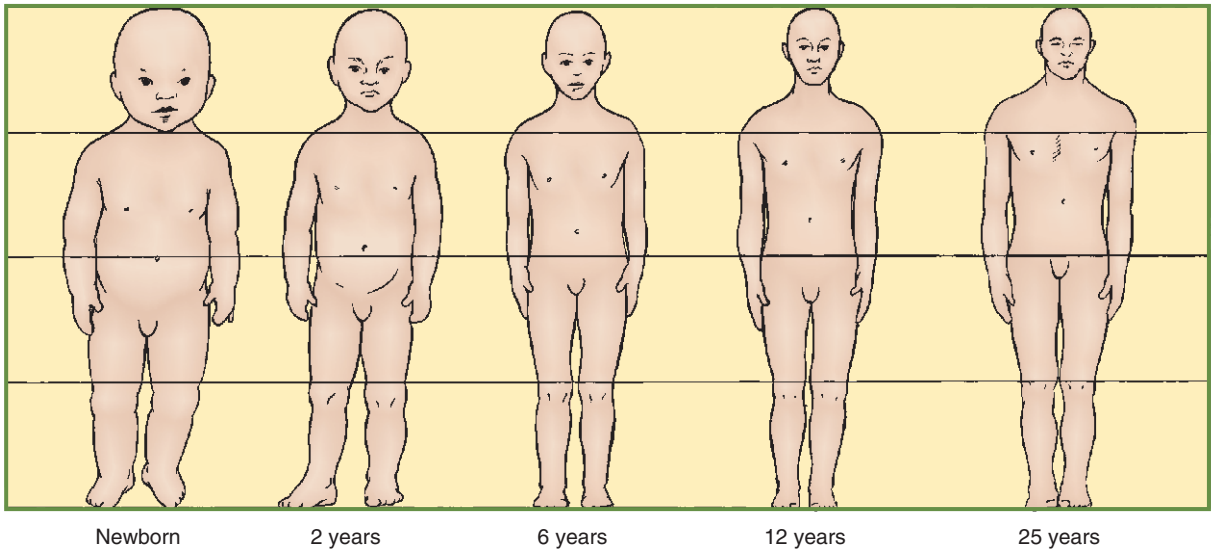

Figure 87-1. Body proportions.

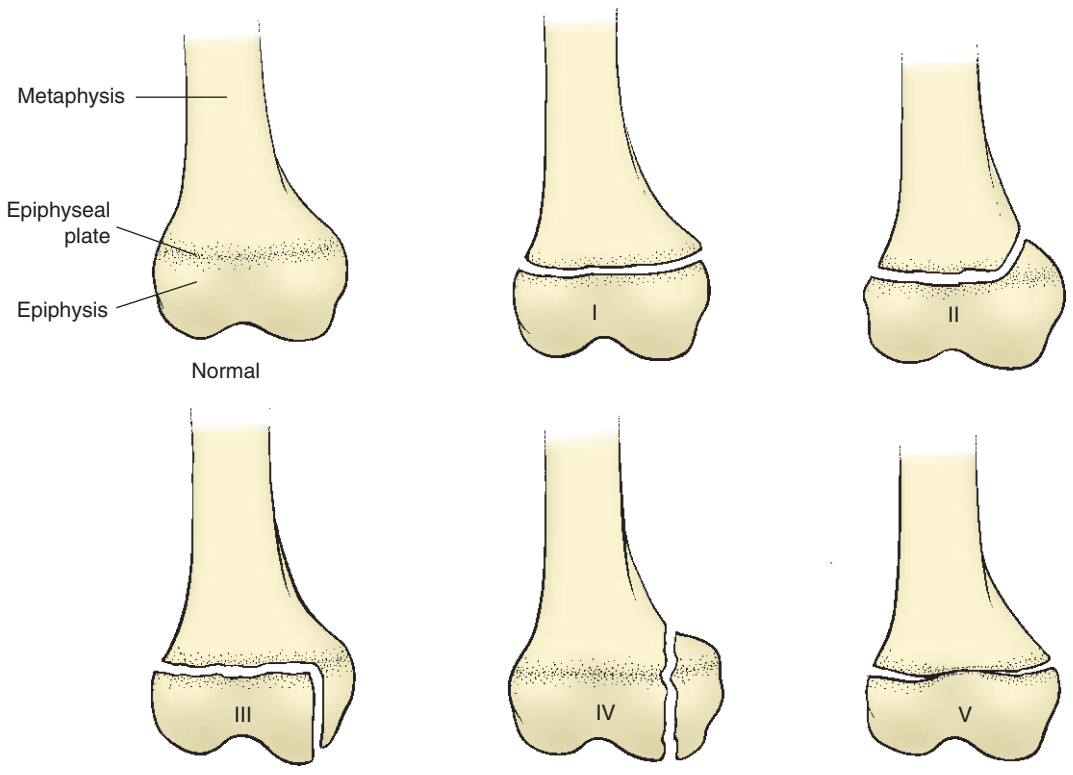

Figure 87-2. Salter-Harris classification of physeal fractures (I to V).

Cardiovascular and Respiratory Systems

The basic physiologic parameters change greatly during the transition from infancy to childhood to adulthood. Recognizing these differences is important to avoid unnecessary and potentially harmful interventions in healthy children, and to intervene aggressively when abnormal vital signs are truly present. For example, blood pressure of 65/35 mm Hg, pulse rate of 160 beats/min, and respiratory rate of 40 breaths/min are considered ominous vital signs for an adult. However, these vital signs are normal in a 2-month-old infant.[31] Although blood pressure readings may not be available in a wilderness setting, it is possible to assess the work of breathing, the respiratory rate, and the pulse strength and rate of an ill child. In general, infants and children have higher respiratory and heart rates and a lower blood pressure than do adults. The normal values for various age groups are presented in Table 87-2.

TABLE 87-2. Age-Specific Resting Heart Rate and Respiratory Rate*

AGE	HEART RATE (Beats/Min)	RESPIRATORY RATE (Breaths/Min)
0–5 mo	140±40	40±12
6–11 mo	135±30	30±10
1–2 yr	120±30	25±8
3–4 yr	110±30	20±6
5–7 yr	100±20	16±5
8–11 yr	90±30	16±4
12–15 yr	80±20	16±3

*Mean rate, ±2 SD.

Thermoregulation

Because environmental extremes are often encountered when traveling in wilderness areas, it is important to recognize that thermoregulation is less efficient in children than in adults. A number of physiologic and morphologic differences make children more susceptible than adults to heat illness.[80] During exercise, children generate more metabolic heat per unit mass than do adults. Children also have lower cardiac output at a given metabolic rate, resulting in a lower capacity to convey heat from the body core to the periphery. Because they have a larger surface area–to-mass ratio, children also gain heat more rapidly from the environment than do adults when ambient temperature exceeds skin temperature. Under these same conditions, cooling from conduction, convection, and radiation ceases to be effective, leaving evaporation (sweating) as the only effective means of heat dissipation. Unfortunately, children have a lower capacity for evaporative cooling, possibly because of decreased sweat volume, regional differences in sweat patterns, and a higher sweat point (the rectal temperature when sweating starts). Finally, children acclimatize to hot environments at a slower rate than do adults.

Children are also at greater risk for hypothermia. Their larger surface area–to-mass ratio causes them to cool more rapidly than adults in cold environments. Children have less subcutaneous fat and thus less body insulation. Infants, in particular,

have an inefficient shivering mechanism.[10] Humans are poorly adapted to a cold environment and must rely on adaptive behavioral responses, such as seeking shelter and dressing appropriately, to maintain body heat. Infants and young children are not capable of these responses and must rely on their caregivers to provide shelter and appropriate clothing.

Immunology and Infections

Children experience a greater number of infections than do adults. The average 1-year-old has six to eight infections per year, whereas the average adult has only three to four infections per year. Infections in children tend to be more severe. The younger the child, the more likely it is that a given infection represents a first exposure. A first-exposure infection is more likely to cause fever and produce severe symptoms than a reexposure, in which the infection is modified by the antibodies produced from the first exposure. Young children are also less likely to have cross-reacting antibodies from a previous infection with an antigenically related organism.

Many common respiratory or viral infections produce more severe symptoms in children because of anatomic differences. The pediatric bronchioles, eustachian tubes, and larynx are narrower and, therefore, more easily obstructed by edema and mucus. This obstruction worsens the symptoms, prolongs clearance of infection, and increases the risk of secondary infection. Pertussis (whooping cough) is a classic example of the difference in severity of infection between children and adults. Nearly 20% of infants with pertussis have severe complications such as pneumonia, seizures, or encephalopathy; adult pertussis, although a common cause of chronic cough, is generally indistinguishable from a common cold.[61]

Trauma

Blunt trauma is the leading cause of morbidity and mortality in children ages 1 to 18 years. Closed head injuries are responsible for 80% of pediatric trauma deaths. Although pedestrian and motor vehicle accidents are the source of many of these injuries, falls and drowning are close behind. Children differ from adults in their susceptibility to injuries from blunt trauma, and the injuries themselves differ. By nature of their smaller size, children's airways are more prone to obstruction, particularly by their relatively large tongues. Their rib cages are more pliable and hence provide less protection to the lungs and mediastinum. Similarly, the abdominal musculature in children is underdeveloped relative to adults, leaving the intra-abdominal organs more vulnerable to injury. Pelvic fractures are uncommon in children; when they do occur, they rarely result in life-threatening bleeding or genitourinary injury.

The cranium of younger children is also softer and hence less protective of the underlying brain. Fortunately, open fontanelles in infants provide a valve of sorts for children with intracranial hemorrhage or swelling. The relatively large head size, loose ligaments, poor cervical muscular support, and underdeveloped facets result in a different pattern of cervical spine injuries in children than is seen in adults. Although cervical spine injuries in children are uncommon, when they do occur, they tend to be higher in the cervical spine and may involve injuries to the spinal cord not visible with conventional radiography or computed tomography (CT) scanning—thus the acronym SCIWORA, for spinal cord injury without radiographic abnormality.

GENERAL CONSIDERATIONS AND EXPECTATIONS

Children of different ages have different needs and abilities. Expectations regarding distances of travel, pace, and safety issues vary depending on age (Table 87-3). This section explores the key issues regarding wilderness travel with children of various ages and provides general expectations for each age group. A number of helpful books that discuss different aspects of wilderness activities with children are listed under Suggested Readings.

First 2 Years

Travel Expectations

Because they are typically carried, children in their first 2 years can travel long distances, depending on the adult's hiking abilities. However, they place extra demands on their caretakers and require attention and care nearly all of their waking hours. Most children in this age group are content in front carriers (infants of <6 months) or back carriers (older infants and toddlers weighing <15 kg) and can easily travel for hours at a time. However, because of their increased risk of illness and limited communication skills, infants must be watched closely for signs

TABLE 87-3. Age-Specific Expectations for Wilderness Travel

AGE	EXPECTATION	SAFETY ISSUES
0 to 2 yr	Distance traveled depends on adults. Use child carriers.	Provide "safe play area" (e.g., tent floor, extra tarp laid out), bells on shoes, ipecac syrup
2 to 4 yr	Difficult age; stop every 15 min, hike 1 to 2 mi on own	Dress in bright colors, teach how to use whistle; ipecac syrup
5 to 7 yr	Hike 1 to 3 hr/day, cover 3 to 4 mi over easy terrain, rest every 30 to 45 min	Carry whistle (three blows for "I'm lost"), carry own pack with mini first aid kid and water
8 to 9 yr	Hike a full day with easy pace, cover 6 to 7 mi over variable terrain; if over 4 ft tall (1.2 m), can use framed pack	As for 5 to 7 yr, plus teach map use and route finding, precondition by increasing maximal distances by <10%/wk, watch for overuse injuries, keep weight of pack <20% of bodyweight
10 to 12 yr	Hike a full day at moderate pace, cover 8 to 10 mi over variable terrain	As for 8 to 9 yr
Teens	Hike 8 to 12 mi at adult pace; may see a decrease in pace or distance with growth spurt	As for 8 to 9 yr

of infection, hypothermia, hyperthermia, and altitude illness. Parents must be prepared to give prompt treatment or evacuate to seek medical attention should signs of serious illness develop. Evacuation plans should be formulated before departure.

Entertainment in this age group is simple. A few small toys (attached to the carrier on a short string), the natural surroundings, and a little parental attention provide ample amusement. A toddler can spend hours examining rocks, leaves, and sticks and rarely tires of a parent's undivided attention. If a child is comforted by a pacifier, it can be attached to the child's shirt or carrier, with extras packed for emergencies.

Safety

As babies become more active with rolling, crawling, and then walking, they require constant attention. Bells attached to their shoes may function as an alerting device, ringing when they are on the move. These children often "graze," putting everything they come across into their mouths. When they are not being carried, it is best to have a child-proofed area for them to play in, such as a tent floor or an extra tarp. Toxic ingestions are common in this age group, and parents should be vigilant to avoid unwanted objects or plants landing in the child's mouth.

Food and Drink

Nourishment in the first 2 years is fairly simple. Infants in their first 4 to 6 months require only breast milk or formula. As long as the mother remains healthy, breast feeding is the safest and most convenient way to feed an infant. However, if the mother is not nursing or not available, formula may be used. Formula is most conveniently carried in a powdered form and mixed as needed. The water for formula may be boiled once a day and stored in individual bottles with airtight lids. The powder for the formula is added just before feeding. Any unused, reconstituted formula should be discarded after 2 to 3 hours at room temperature.

Baby cereals can be carried conveniently in a dry form to be mixed with formula or breast milk. Dry cereals mixed with breast milk or formula have a higher nutritional value than ready-to-feed cereals in jars. Jars of commercial pureed foods may be carried, but the empty jars must be packed out. Once a jar of baby food has been opened, it should be used for only that meal. Without refrigeration, opened jars of baby food spoil quickly. Some families prefer to bring a hand grinder and make their own pureed foods. Resources on preparing homemade baby food are included in the suggested readings at the end of the chapter.

By age 9 to 12 months, many babies are eating finger foods. Parents should be cautioned to avoid any hard round foods such as peanuts or candies on which a baby may choke. Up to 1 year of age, honey should be avoided because of an increased risk of botulism. Parents may also want to also avoid citrus fruits, which may cause rashes around the mouth and in the diaper area. Any new food should be tested at home prior to travel to be certain the baby will accept it when away from home.

All water for drinking must be disinfected by boiling, iodination, and/or the use of small-pore filters, depending on the water source. Chronic iodide poisoning and neonatal goiter have been associated with prolonged ingestion of large amounts of iodine, although small amounts ingested for water disinfection appear safe. Nonetheless, if both boiled water and iodinated water are available, it is prudent to use boiled water for infants and small children. In addition, infants and small children often reject the taste of iodinated water. Iodine must be kept out of reach of small children; severe acute toxicity can occur with an ingestion of just 2 to 4 g.

Diapers

Most children under the age of 2 years are in diapers, either disposable or cloth. Soiled diapers in a wilderness environment require special care. Thin paper diaper liners may be purchased to help collect the stool. The stool and liner should be buried in a trench at least 6 inches deep and 200 feet (60 m) from any water source. If disposable diapers are used, they should be packed out after the stool has been removed and buried. The used disposable diaper should be wrapped and placed in a double bag for packing out. To reduce weight, urine-soaked diapers may be set out in the sun to dry before repacking. Superabsorbent diapers may be left on babies much longer and still function to keep them dry. However, these diapers cannot be dried out as easily and, consequently, add significant weight for the rest of the trip.

On longer trips, some families prefer to use cloth diapers, which may be washed out and reused. Cloth diapers must be changed more frequently, as they are not as absorbent. Washing cloth diapers is labor intensive, time consuming, and requires an abundant supply of water. A washbasin is needed, and the diapers must be washed in hot soapy water. The diapers should be rinsed at least twice to remove irritating soap residue, and the waste water dumped where it will not pollute, at least 200 feet (60 m) from a water source.

Equipment

Because infants and young children are not capable of extended hikes, they are typically transported in carriers. Excellent front and back carriers are now available. Most front carriers work well from infancy until an age when babies can sit fairly well, typically around 6 to 9 months (Fig. 87-3). It is important that a front carrier extend up high enough in the back to completely support the baby's head. Once a child is sitting well, back carriers are better (Fig. 87-4). Back carriers function on the same principle as framed backpacks, redistributing the weight off the shoulders and onto the hips. Many back carriers are able to stand alone and can double as a highchair. Children must be strapped into back carriers, because it is easy for a child to be catapulted out of a carrier if the adult bends over or falls.

Sleeping bags are available for infants and toddlers. However, in a warm climate, a few blankets generally suffice, and, in a colder climate, an insulated snowsuit is often adequate. Children, including young infants, need their own sleeping pads. Such pads protect them from hard, rough ground under the tent and insulate them from the cold ground at night.

Shoes for young children should protect their feet and allow for full range of movement. The best shoes for toddlers are lightweight and flexible. They need shoes that stay on well, because children often flip their shoes off while in a carrier. Velcro-strapped shoes stay on well and are easy to put on and take off. Because children often lose shoes, an extra pair should be included.

2 to 4 Years

Travel Expectations

Children 2 to 4 years old are the most challenging to take into the wilderness. Two-year-olds become easily frustrated and

Figure 87-3. Young infant in front carrier. (Courtesy Judith R. Klein, MD.)

Figure 87-4. Toddler in back carrier. (Courtesy Judith R. Klein, MD.)

throw temper tantrums, often as a result of the collision between adult restrictions and their desires for independence and control. By 2 years of age, children are becoming too heavy to carry for prolonged periods, but they are still incapable of hiking long distances on their own. They are just gaining bladder and bowel control, and accidents are frequent. Despite these difficulties, wilderness trips with this age group can be successful with appropriate planning, preparation, and adjustment of expectations.

A key ingredient to successful wilderness trips with small children is to keep things slow, simple, and flexible. This is the age of independence and assertion. The children need to be given some control and allowed to set a pace. Adults should encourage young children to express their natural curiosity and enthusiasm for the outdoors by letting them stop to explore their surroundings. Parents can enjoy rediscovering nature through the eyes of their children by exploring rocks and tide pools and observing a caterpillar's crawl. Parents should expect to stop at least every 15 minutes while hiking. If a diversion or a stimulus is needed to get the children hiking again, parents can begin a story or favorite song and continue it while hiking. With patience and plenty of time, parents can expect children in this age group to travel 1 to 2 miles under their own power over easy terrain.

Safety

Unfortunately, 2- to 4-year-olds are notorious for exploring their environment by either wandering off or putting whatever they come across into their mouths. Young children must be watched closely and cautioned to keep wild mushrooms, plants, stones, and any nonedible item out of their mouths. Children should be kept within sight at all times, as their desire to explore often defies good judgment and exceeds their physical abilities. Although attacks are rare, mountain lions may view small children as easy prey and can strike quickly. Parents should, therefore, discourage their children from wandering ahead unaccompanied. Toddlers should also be encouraged to step only where they can see to avoid the unsuspecting reptile or large insect.

When selecting campsites, dangerous features, such as steep drop-offs and fast, deep water, should be avoided. Children should be dressed in brightly colored clothing, so they are more easily located if they are lost (Fig. 87-5). As children get older, they may carry a whistle to call for help when they are lost. The standard distress signal is three blows to indicate "I'm lost" or "I need help"; the response is two blows to indicate "help is coming." Parents should teach children to stay put once they discover they are lost and wait to let help come to them. If children panic and start running when they realize they are lost,

Figure 87-5. Appropriate wilderness wear: brightly colored clothing and whistle. (Courtesy Judith R. Klein, MD.)

they increase the chance not only of getting injured but also of getting farther from the family. The concept of "hugging a tree" will be described later.

Food

The diet of 2- to 4-year-olds is usually quite simple but very individual. They tend to have strong preferences and dislikes. Unfortunately, most children at this age do not care for the convenient "all-in-one-pot" cooking common around campfires. Foods should be tested at home first to be sure they are acceptable to the child. Nutritious snacks such as raisins, granola bars, bagels, crackers, string cheese, and fruit bars can be packed. These snacks may become a child's meal. Small children should not be given items such as peanuts, grapes, hard candies, or hot dogs on which they may choke. At least one adult member should be trained in basic cardiopulmonary resuscitation (CPR) and know how to assist a choking child.

Toileting

Most children become toilet trained by the end of their third year. However, accidents are common and parents need to be prepared with extra dry clothing that is readily accessible. Children should be taught correct toileting procedure for a wilderness environment. Stools should be deposited at least 200 feet (60 m) from a water source, buried in a hole approximately 6 inches deep, and completely covered. Many families carry a special trowel for this purpose. Some groups staying in one location for more than a day dig a specific toileting trench, 12 to 18 inches deep, to be used multiple times. They then add enough dirt after each use to cover all waste. Children need help learning to squat over the trench and to bury their stools.

It may be years before children gain reliable nighttime bladder control. Cotton and down sleeping bags should be avoided because they lose their insulating abilities and take a long time to dry. Fortunately, many synthetic bags are available, with fills such as Polarguard, Quallofil, and Hollofil, which maintain warmth and loft when wet.

School Age (5 Years and Up)

Travel Expectations

Once children enter kindergarten, their abilities and attention span increase dramatically. This enables them to participate more actively in many outdoor activities. Children are hungry for knowledge and readily absorb information about nature and outdoor activities. They enjoy being included in the initial planning, as well as in the field activities, such as setting up camp, cooking, and cleaning up. School-age children can understand maps and often enjoy following their progress from one point to another. This is an ideal age to explain to them the rules of living in and traveling through wilderness areas. The examples and rules parents set for appropriate behavior in a wilderness at this age may become lessons engraved for a lifetime.

When parents are planning hiking trips, it is important that they have appropriate expectations for children's differing abilities (see Table 87-3). Children enrolled in organized sports activities are likely to have greater endurance in the wilderness. A child's hiking ability can be estimated by walks around the neighborhood or in a local park. If this practice becomes a routine, children become preconditioned, increase their endurance, and learn to pace themselves. More importantly, parents can learn what to expect and can test methods for motivating their children. It is better to underestimate than to overestimate a child's ability. Parents should also remember that children, like adults, have good and bad days, so allowances should be made.

Safety

School-age children can learn to become more self-sufficient and in tune with their surroundings. They can be taught to recognize landmarks in their environment, so they are less likely to get lost. Such landmarks can be pointed out, and children should be encouraged to view their surroundings from different angles so that they can see their way back if they stray off. Children should periodically turn around so they can see where they came from, as well as where they are going. As children advance in school years, they can learn survival skills, such as how to maintain warmth, build a shelter, secure food and water, and use a signal mirror, map, and compass. As with the previous age group, they should carry a whistle and know how to use it appropriately.

Equipment

Children like to feel important, capable, and independent. These feelings are enhanced if they are allowed to carry some of their own gear. Even 5-year-olds like to carry their own soft back-

packs. Items they can carry in the packs include snacks, a favorite small toy, extra clothing, sunscreen, a small trash bag, and a whistle. As the children grow, the contents of their backpacks should reflect their increasing independence, with more self-care and survival items. In addition to the preceding items, they may wish to carry their own water bottle, a mini first-aid kit (adhesive bandages, wipes, personal medication), insect repellent, and other survival items as they learn to use them. The maximal weight of these packs should be 20% of the child's bodyweight until he or she has had significant backcountry experience and can comfortably carry more. Once children reach 4 feet (1.2 m) in height, they can be fitted for a framed backpack. Internal-frame backpacks tend to be more comfortable than external-frame packs. When a backpack is properly fitted, the waistband should rest at the hips and the shoulder strap should be adjusted so that the weight is carried on the hips, not on the shoulders. With a framed pack, children can carry even more of their own gear. However, the total weight should be gradually increased to allow the child to become comfortable with heavier loads and should not exceed 30% of the child's bodyweight.

TABLE 87-4. Signs and Symptoms of Hypothermia*

RECTAL TEMPERATURE		SIGNS AND SYMPTOMS
Mild	(33° to 35° C) (91° to 95° F)	Sensation of cold, shivering, increased heart rate, progressive incoordination in hand movements, developing poor judgment
Moderate	(28° to 32° C) (82° to 90° F)	Loss of shivering, difficulty walking or following commands, paradoxical undressing, increasing confusion, decreased arrhythmia threshold
Severe	(<28° C) (<82° F)	Rigid muscles, progressive loss of reflexes and voluntary motion, hypotension, bradycardia, hypoventilation, dilated pupils, increasing risk of fatal arrhythmias, appearance of death

*Data from adult subjects.

ENVIRONMENTAL ILLNESSES

Dehydration

Children are at greater risk of dehydration than are adults. Because the surface area–to-bodyweight ratio of a child is greater than that of an adolescent or adult, insensible fluid losses through the skin account for a larger percentage of total fluid losses as the size of the child decreases. In addition, the sodium concentration of children's sweat is generally less than that of adults, leading to a greater relative free water loss. Infants are unable to report thirst, an important marker of fluid deficit, increasing their risk of dehydration. Even once they become verbal, children are often preoccupied and fail to report or meet their need for fluids, even when water is freely available.

Symptoms

As little as a 2% decrease in bodyweight through fluid loss results in mildly increased heart rate, elevated body temperature, and decreased plasma volume. Water losses of 4% to 5% of bodyweight reduce muscular work capacity by 20% to 30%.[80] Symptoms of dehydration include weakness, fatigue, nausea, vomiting, and, ultimately, lethargy. Dehydration also predisposes a child to other environmental hazards, such as hypothermia, hyperthermia, and acute mountain sickness.

Treatment

It is the caregiver's responsibility to provide fluids and coax the child to drink frequently. For short (<2-hour) periods of activity, water is as efficacious a rehydration solution as carbohydrate-electrolyte drinks.[54] A child eating a normal diet does not require electrolyte replacement unless sweating is prolonged or excessive. By closely monitoring a child's urine output, fluid deficits can be recognized and promptly managed. A child with decreased urine output or dark, concentrated urine needs extra fluids.

Hypothermia

Children cool more rapidly than do adults because they have a proportionally large surface area and often lack the knowledge and judgment to initiate responses that will maintain warmth in a cold environment (see Chapter 5). In addition, they have a more difficult time, physiologically, maintaining their body temperature in cold climates. As a result, parents participating in cold weather recreation with children should be able to recognize, treat, and, preferably, prevent hypothermia and frostbite.

Hypothermia is defined as a core body temperature below 35° C (95° F). At this temperature, the body no longer generates enough heat to maintain body functions. The condition is considered mild when the core temperature is 33° to 35° C (91° to 95° F); moderate at temperatures between 28° and 32° C (82° and 90° F), and severe when it is less than 28° C (82° F). The signs and symptoms of hypothermia are listed in Table 87-4, although these may be quite variable. The most important clue to significant hypothermia is altered mental status. The child who is shivering, stumbling, or appears confused should be evaluated promptly for hypothermia. The presence or absence of shivering is *not* a reliable marker of the severity of hypothermia. Physicians should caution parents that hypothermia can develop at moderate ambient temperatures if adverse climatic conditions are compounded by illness, fatigue, dehydration, inadequate nutrition, or wet clothing.

Prevention

When preparing for cold weather activities, children should dress in layers to allow clothing to be added or subtracted as necessary (Fig. 87-6). This avoids excessive perspiration while maintaining warmth. An inner, wicking layer should be followed by a middle, insulating layer and, finally, by an outer, protective layer.

Because children generally avail themselves of any opportunity to get wet, clothing that maintains low thermal conductance when moist is particularly important. Conductive heat loss may increase fivefold in wet clothing and up to 25-fold if the child is completely immersed in water. Wool retains warmth when wet because of its unique ability to suspend water vapor within the fibers; however, it is heavier than synthetics and takes much longer to dry. Cotton has a high thermal conductance that increases greatly when wet and is therefore a poor choice for wilderness activities in cold weather. Newer synthetic materials

Figure 87-6. Layering of clothing for cold environments. (Courtesy Judith R. Klein, MD.)

(polypropylene, Capilene, Thermax, Coolmax) wick moisture away from the skin and dry quickly, making them ideal for an inner layer. The middle, insulating layer may incorporate wool, polyester pile or fleece, down, or similar materials. Windproof and water-resistant outer garments (e.g., Gore-Tex) decrease heat loss from convection and keep children dry. Hats and mittens are also essential; the uncovered head of a child dissipates up to 70% of total body heat production at an ambient temperature of 5° C (41° F). These are available in materials that not only provide warmth but also resist the penetration of wind, further reducing convective heat loss.

Treatment

For the hypothermic child, field rewarming begins with limiting further exposure to the cold environment. Find immediate shelter for the child. Wet clothing should be removed, and the child's head and neck should be protected from further heat loss. Place the child together with a normothermic person in a sleeping bag insulated from the ground to provide external warmth. Hot water bottles, insulated to prevent burns, may also be placed at the axillae, neck, and groin. If the child is alert, oral hydration with warm fluids containing glucose repletes glycogen and corrects dehydration, which frequently accompanies hypothermia. Signs of severe hypothermia dictate immediate evacuation as conditions permit; rescuers should handle the victim gently to prevent precipitating arrhythmias.

Frostbite

Localized cold injury results in frostbite (see Chapter 8). Predisposing factors include wet skin, constricting garments that hinder blood circulation, fatigue, dehydration, contact with cold surfaces, and wind. If skin temperature drops below 10° C (50° F), cutaneous sensation is generally abolished and injury may go unnoticed. Skin cooled to –4° C (25° F) freezes.

Frostbite has traditionally been divided into degrees of injury, much like burns. Determination of depth of injury should occur 24 to 48 hours after rewarming; prior to this, frostbitten skin generally appears hard and feels numb. Skin with superficial frostbite is typically swollen, pink or erythematous, painful, somewhat warm, and often blistered. Sites with deep frostbite are cooler, not edematous, pale, anesthetic, and do not have blisters or bullae. In children, frostbite that extends into bone may affect the growth plate and result in skeletal deformities.[39]

Treatment

All wet and constricting clothing should be removed and hypothermia treated aggressively. Rapid rewarming, the primary treatment for frostbite, should be initiated as soon as possible. This is best accomplished by immersion of the frostbitten area in water warmed to 40° to 42° C (104° to 108° F). This narrow temperature range maximizes rewarming speed while preventing thermal burn injuries. Thawing usually takes 30 to 45 minutes and is complete when the skin is soft and pliable. Field rewarming is indicated unless evacuation is imminent and rapid; however, great care should be taken to avoid refreezing. Refreezing causes far more damage than delayed thawing, due to the formation of ice crystals in connective tissue. Vigorous rubbing should also be avoided because it is ineffective and potentially harmful. After thawing, proper wound care is essential. Frostbitten sites should be kept clean, ruptured bullae should be debrided, and the area should be dressed in a bulky dressing. Oral ibuprofen and topical aloe vera facilitate healing.

Hyperthermia

Families participating in wilderness activities in hot climates must take special precautions to avoid heat illnesses (see Chapters 10 and 11). Children do not tolerate the demands of exercise in the heat as well as adults. They generate more heat per kilogram and are less able to disperse heat from the core to the periphery. Parents planning wilderness ventures with children in hot climates can follow some simple guidelines for avoiding heat illness. The most obvious entails reducing the duration and intensity of activities under conditions of high climatic heat stress. The likelihood of heat illness depends on relative humidity, wind velocity, and radiant heat, as well as standard dry bulb thermometer temperature. Figure 87-7 gives a rough guide for activity levels based on temperature and relative humidity.

Prevention

Children should be fully hydrated before prolonged exercise and actively encouraged to drink fluids at regular intervals. Infants and neonates are most vulnerable to heat illness. Under high climatic heat stress, infants fed undiluted cow's milk or formula may develop marked salt retention and dehydration. They should be given extra water or dilute feedings. The lower

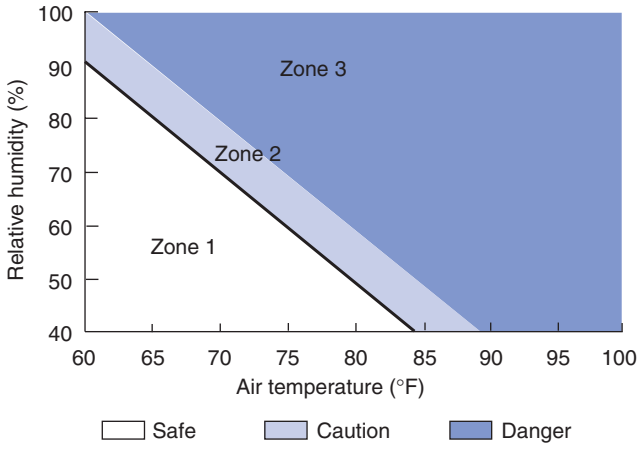

Figure 87-7. Activity levels based on temperature and relative humidity.

osmolar load of breast milk appears to protect against heat illness and hypernatremia.[62]

Because their mechanism of evaporative heat loss (sweating) is immature relative to that of an adult, children should be encouraged to engage in activities in the shade to maximize other means of dissipating heat, such as radiation (skin-to-air gradient). Also, because sweat evaporated from clothing contributes less to cooling than sweat evaporated from skin, children should be changed out of sweat-soaked clothing and wear dry, lightweight, loose-fitting clothing. Finally, children acclimatize to the heat more slowly than adults, often taking 10 to 14 days to fully adapt. The intensity and duration of exercise should be gradually increased over this period.

Symptoms and Treatment

Early signs and symptoms of heat illness include flushing, tachycardia, weakness, mild confusion, headache, and nausea. Vomiting often occurs in children. Sweating may be present or absent and should not be relied on as a clinical indicator of the severity of hyperthermia. If heat illness develops, children should be removed from obvious sources of heat, including direct sunlight, and have their clothing removed. Convective cooling can be increased in the field by vigorous fanning after spraying or sprinkling the victim with water. If available, ice packs or cold compresses placed on the groin, axillae, and scalp will aid cooling. Cool-water immersion is also a very effective means of rapid cooling if the child's head position can be controlled. If the child is alert, dehydration should be corrected with oral fluids. Progression of symptoms or failure to respond to treatment mandates immediate evacuation.

Sun Damage

The hazards of overexposure to sunlight include sunburn, photoaging, skin cancer, and phototoxic and photoallergic reactions (see Chapter 14). Climatic changes such as global warming and ozone degradation have only increased these hazards. Preventing ultraviolet damage to the skin should begin in childhood, as 80% of a person's lifetime sun exposure occurs before 21 years of age.[59] Adolescence is the period when children are most at risk. In one study, 83% of children 12 to 18 years old reported at least one sunburn per summer; 36% reported three or more sunburns per summer.[25] Recent evidence suggests that the risk of developing malignant melanoma increases significantly with the number of sunburns in childhood.[38] This risk is even higher if a child is light-skinned with a propensity to burn rather than tan. Tolerance to sun exposure is determined by the amount of melanin in the skin and the ability of the skin to produce melanin in response to sunlight. In general, children have lower melanin levels and thinner skin than do adults and are, therefore, at greater risk of sun damage.

The ultraviolet wavelengths UVA and UVB are principally responsible for the harmful effects of solar radiation. UVB is primarily responsible for suntan and sunburn and also promotes the development of skin cancer and aging of the skin. UVB increases 4% for every 1000-foot gain in elevation over sea level. Therefore, a backpacker at 10,000 feet (3050 m) will have a 40% increase in UVB exposure. UVA, which is 10 to 100 times more abundant than UVB, is only 0.001 as potent at inducing sunburn. It is also less affected by changes in season or solar zenith angle than UVB. UVA is primarily responsible for photosensitivity reactions and solar urticaria. It also contributes to skin cancer and skin aging. A number of drugs often used in adolescence, such as tetracycline, vitamin A derivatives (Retin-A, Accutane), and nonsteroidal anti-inflammatory drugs (NSAIDs), increase the risk of photosensitivity reactions and the need for UVA protection. Consequently, it is important to use sunscreens that protect against both UVA and UVB.

The harmful effects of ultraviolet radiation from the sun can be reduced if parents are educated regarding the dangers of sun exposure and encouraged to use sun-protective clothing and sunscreens early in their children's lives. One study demonstrated a 60% reduction in childhood sunburns with good parental role-modeling and sunscreen vigilance.[59] Regular use of a sunscreen with a sun protection factor (SPF) of at least 15 for the first 18 years of life reduces a person's lifetime risk of developing nonmelanoma skin cancer by 78%.[38]

Prevention

The most effective means of preventing sun damage is avoidance of excessive sun exposure and the use of protective clothing. Midday hours, particularly around bodies of water, at high altitude, and at the equator, are the most dangerous in terms of quantity of ultraviolet exposure. Shady areas should be utilized for activities during these times. Hats with wide brims and neck drapes help to protect the face and neck from sun exposure. Clothing made from tightly woven fabrics is more protective than clothing made from loosely woven fabric. For example, loosely woven fabrics, such as that used in most T-shirts, have an SPF of only 5. Most clothing loses even more of its protective effect when wet. However, several manufacturers (www.coolibar.com; www.sunprotectiveclothing.com) are marketing high-SPF (25 to 30) protective clothing. This specialized sun-protective clothing is cool and lightweight, dries quickly, and can maintain its full SPF capabilities even when wet.

Proper eye protection is often overlooked in infants and young children. Excessive ultraviolet light, particularly during snow and water activities, can result in ultraviolet keratitis (see Chapter 25) and corneal burns with even brief exposures. Properly fitting sunglasses that transmit fewer than 10% of ultraviolet rays should be part of a child's outdoor activity armamentarium (Fig. 87-8). Side shields and polarizing lenses are particularly important on the snow.

Figure 87-8. Child with appropriate sun wear: wide-brimmed hat and sunglasses. (Courtesy Judith R. Klein, MD.)

Sunscreens. Most sunscreens have ingredients, such as PABA, PABA-esters, cinnamates, salicylates, and anthranilates, that protect the skin from sun damage by absorbing primarily UVB. Dibenzoylmethane absorbs only UVA. Benzophenones block UVB and to a lesser degree UVA.[37] Sunscreens that combine ingredients that protect the skin from both UVB and UVA are the most effective. Micronized titanium dioxide, which reflects ultraviolet light in a fashion similar to the sun block zinc oxide, is nearly colorless yet retains the ability to scatter sunlight.

The SPF is a measure of a sunscreen's effectiveness. It is measured in terms of the minimal dose (in length of time) of UV radiation required to cause skin erythema. Sunscreens with an SPF of 30 or higher provide a superior degree of photoprotection and almost completely prevent the cellular changes seen with sunburn.[36]

Water-resistant sunscreens maintain effectiveness for up to 40 minutes of water immersion, whereas waterproof sunscreens maintain effectiveness for up to 80 minutes of water immersion.[37] Both should be reapplied if longer times are spent in the water or if the water is turbulent. Excessive sweating and rubbing of the skin are also indications for reapplication.

Treatment

When sunburn occurs, the mainstays of treatment are cool compresses, topical antipruritics, and NSAIDs. Topical aloe vera cream or gel often provides a soothing effect for an uncomfortable child. Further sun exposure should be avoided while the skin is healing. As always, prevention is superior to any treatment.

Drowning and Near-Drowning

According to the Centers for Disease Control, drowning (see Chapter 68) is the number two cause of injury-related death in children, resulting in 859 fatalities in 2001 (www.cdc.gov/ncipc/factsheets/drown.htm). Those most at risk are unsupervised toddlers and male teenagers, in particular those with poor swimming skills and even poorer judgment. Morbidity and mortality typically result from asphyxiation, hypothermia, or trauma.

If a child is pulled from the water after a submersion event, cervical spine precautions should be maintained unless the event is witnessed and no trauma has occurred. All children who have experienced a period of near-drowning should be transported to a hospital for observation, particularly if they demonstrate any sign of distress. If the child is apneic or pulseless, CPR should be initiated immediately and the child transported to an emergency care facility as quickly as possible. All wet clothing should be removed and rewarming initiated to the extent possible. Prognostic assessments based on the initial appearance of the child should not be made in the field, particularly in the setting of cold water immersion. The exception would be in a remote wilderness area where transport and evacuation times may be extremely long. Survival with good neurologic outcome has been documented in children with prolonged (>40 minutes) submersion in cold waters. The emphasis in the field should be on rapid rescue and immediate CPR.

Prevention

Preventive measures are critical to reducing the number of drowning and near-drowning incidents. Most importantly, children should be taught to swim at an early age and should learn to "read" the water and make appropriate judgments regarding water safety. They should be taught to swim with a buddy, particularly in moving and deep water. Young children should always be supervised by an adult skilled in CPR and water rescue. Use of a personal flotation device is highly encouraged until the child is a strong swimmer capable of treading water for prolonged periods. Finally, cold water with rapidly moving current, water hazards, or large waves should be approached with extreme caution and entered only by strong swimmers.

High-Altitude Illness

High-altitude illness (see Chapter 1) can be viewed as a continuum from acute mountain sickness (AMS) to life-threatening conditions such as high-altitude pulmonary edema (HAPE) and high-altitude cerebral edema (HACE). The true incidence of high-altitude illness is unknown but probably falls between 10% and 20% at 2500 m (8200 ft) in adults and children. AMS usually develops within 24 hours of ascent. The incidence and severity of AMS depend on individual susceptibility, as well as the rate of ascent and the altitude attained. The sleeping altitude is particularly important to the development of symptoms.

Box 87-1. Preventing High-Altitude Illness

- Avoid abrupt ascent to a sleeping altitude higher than 3000 m (10,000 ft).
- Spend 2 or 3 nights at 2500 to 3000 m (8000 to 10,000 ft) before going higher.
- Avoid abrupt increases of greater than 500 m (1600 ft) in sleeping altitude per night above 3000 meters (10,000 ft).
- Climb high, sleep low.
- Drink lots of fluids.
- Eat high-carbohydrate foods.
- Avoid artificial sleep aids (e.g., diphenhydramine).
- Consider using prophylactic acetazolamide.

Symptoms

The cardinal symptoms of AMS are a throbbing headache, anorexia, and malaise. Children are particularly prone to nausea and vomiting. Other symptoms include dizziness and fragmented sleep. Infants may display nonspecific findings such as irritability, poor feeding, and sleep disturbance. As AMS worsens, headaches become more severe and nausea and anorexia progress to vomiting. Dyspnea at rest and confusion/ataxia mark the development of the life-threatening conditions of HAPE and HACE, respectively.

Prevention

The safest and most effective method of preventing high-altitude illness is to allow for acclimatization via a graded ascent. No precise, scientifically proven guidelines exist, given the markedly variable individual susceptibility to altitude illness. However, general recommendations for children (and adults) without altitude experience are listed in Box 87-1. After day trips to higher altitude, children should return to lower altitude to sleep in order to aid acclimatization. A high-carbohydrate diet and plenty of fluids can also help to reduce the risk of high-altitude illness.

Acetazolamide has been convincingly shown to reduce the incidence of AMS in adults.[4] Pretreatment with this agent mimics the acclimatized state by inducing hyperchloremic metabolic acidosis, allowing for a compensatory increase in respiration. There are no published studies of its efficacy in children, but clinical experience suggests that it is beneficial. The primary indication for acetazolamide prophylaxis in children is a history of recurrent AMS despite graded ascent.[87] Acetazolamide is given at 5 mg/kg/day, in two divided doses, up to a maximum daily dosage of 250 mg. It should be started 24 hours before ascent and continued for 3 to 5 days while at maximal altitude. Side effects include nausea, mild somnolence, and paresthesias that can be particularly bothersome in children. Dexamethasone also prevents or reduces symptoms of AMS in adults, but its use is highly discouraged in the prevention of AMS in children, because it masks the early symptoms of mountain sickness and thereby encourages continued ascent. *Ginkgo biloba* has also been studied recently as an herbal alternative to acetazolamide for the prevention of AMS; unfortunately, its efficacy was found to be no greater than placebo.[4] Salmeterol, an inhaled long-acting beta agonist, has also been studied as a prophylactic agent against HAPE in adults, but it has not been evaluated in children.

Treatment

Treatment of mild AMS requires prompt recognition of symptoms, cessation of ascent, and allowance of time for acclimatization to occur. *Proceeding to higher altitude in the presence of symptoms is strongly contraindicated and may lead to the life-threatening conditions HAPE and HACE.* Symptomatic therapy includes rest, acetaminophen for headache, and adequate hydration. Promethazine (Phenergan) may be used to relieve nausea and vomiting in older children. Dystonia in response to phenothiazines, such as promethazine, occurs disproportionately in young children, so their use should be avoided. The dose is 0.2 to 0.5 mg/kg/dose up to every 6 hours, preferably per rectum. If symptoms resolve, the child may continue to ascend slowly. However, if symptoms progress or fail to improve, descent is mandatory. Although descent should proceed as far as necessary for improvement, 500 to 1000 m (1600 to 3200 ft) is often sufficient. If immediate descent is not possible, oxygen should be administered if available. Studies examining dexamethasone and acetazolamide for the treatment of AMS suggest that both are effective.[4] Dexamethasone should be reserved for patients with severe AMS or HACE. The symptoms of HACE or any symptoms of HAPE demand immediate descent and possible evacuation.

Bites and Stings

Bites and stings occur commonly in the pediatric age group. In 2002, the American Association of Poison Control Centers recorded 90,896 calls regarding bites and envenomations; nearly half involved patients 17 years of age or younger.[91] Remarkably, major morbidity occurred in less than 0.1% of cases, and there were only two deaths in patients less than 17 years of age.[91] This emphasizes the need for appropriate triage to determine which children require aggressive therapy so that potentially harmful field interventions can be avoided.

Dangerous interactions between children and surrounding fauna can be limited by the judicious use of protective clothing, sturdy footwear, appropriate chemical barriers, and education. These principles apply to snakes, Hymenoptera, mosquitoes, and ticks.

Snakes

Of the 7000 venomous snakebites (see Chapters 48 and 49) that occur in the United States each year, about half occur in those under the age of 20. Although mortality is uncommon, about 20% of snakebite-related deaths occur in children under the age of 5 years. Over two thirds of bites in children are on the lower extremities; these are predominantly in younger children walking or running in the wilderness. Most snakebites, however, can be prevented. Children should be instructed not to handle snakes, not to reach blindly into crevices, and not to turn over rocks and fallen limbs. A useful adage is that hands and feet should never go where the eyes cannot see. When walking through endemic areas, hikers should stay on trails and wear long, loose pants and boots that extend above the ankle. Campsites should be on open ground, away from woodpiles or rock piles.

If a bite occurs, the child should back well away from the snake and be calmed. Agitation and movement of the bitten extremity will encourage venom circulation. The wound should be cleansed rapidly and any constricting items of clothing or jewelry should be removed from the extremity. The bitten extremity should be immobilized promptly and positioned at

the level of the heart. No incision over the bite should be made. The use of mechanical suction (e.g., Sawyer extractor) is ineffective at removing snakebite venom and no longer recommended. Advanced techniques for potentially limiting venom spread, particularly with exotic snake envenomations, are discussed in detail in Chapters 48 and 49. All pediatric victims of potentially poisonous snakebites should be transported to a medical facility for prompt evaluation, local wound care, and possible antivenom administration.

Hymenoptera

Hymenoptera (bees, wasps, hornets, and ants) stings are the most common cause of envenomations in children (see Chapter 44). Although Hymenoptera venom possesses intrinsic toxicity, the amount delivered is small and multiple stings are necessary for significant human morbidity. However, the venom components are potent antigens capable of producing IgE-mediated anaphylaxis in sensitized individuals. Although children appear less susceptible to systemic reactions than adults, physicians should educate parents in the management of Hymenoptera stings, particularly if a child has previously had a severe reaction.

Hymenoptera stings usually produce local pain, swelling, and erythema. If a stinger is imbedded, it should be removed as quickly as possibly by whatever means available, for the speed of removal of the stinger is far more important than the method of extraction.[90] Within 20 seconds, 90% of the contents of the venom sac are discharged into the victim, and 100% within 1 minute.[75] Applying ice or cool compresses reduces pain and swelling. Elevation and immobilization are indicated for large local reactions on extremities. In older children, oral antihistamines may provide additional symptomatic relief.

Early signs of a systemic reaction include generalized pruritus, urticaria, angioedema, bronchospasm, and laryngeal edema. The presence of these signs or symptoms mandates immediate medical evaluation. Epinephrine (1:1000) is the drug of choice for systemic sting reactions and should be administered in the field if available (0.01 mg/kg subcutaneously up to 0.3 mg). Parents without a medical background may prefer the EpiPen or EpiPen Jr. Both contain a spring-loaded automatic injector that delivers 0.3 mg (EpiPen) or 0.15 mg (EpiPen Jr) of epinephrine subcutaneously when triggered by pressing the device against the thigh. The EpiPen Jr is appropriate for children up to 15 kg, and the regular EpiPen is appropriate for larger children and adults. Because up to half of all patients with anaphylactic reactions have no forewarning, epinephrine belongs in all wilderness medical kits.

Mosquitoes

Mosquitoes not only present a high nuisance potential but also serve as vectors of disease (see Chapters 41 and 42). A number of steps can be taken to avoid mosquito bites and the diseases they carry (Box 87-2). A proper wardrobe provides an excellent physical barrier and is the first defense against mosquitoes. This should include ankle-high footwear, pants cinched at the ankles or tucked into socks, a long-sleeved shirt, and a full-brimmed hat. Mosquito head netting draped over a child's hat will protect the face and neck (Fig. 87-9). Mosquito netting, especially in the sleeping area, has been found to reduce the mosquito attack rate by 97%.[81]

Repellents containing DEET (*N,N*-diethyl-meta-toluamide) are effective against mosquitoes, ticks, black flies, and many other arthropods. Although generally of low toxicity, high concentrations of DEET can cause dermatitis with erythema, bullae, skin necrosis, and residual scarring. DEET is also absorbed cutaneously, and rare toxic encephalopathy associated with its use has been reported. Recently, a large study based on

Figure 87-9. Mosquito netting draped over a child's hat. (Courtesy Judith R. Klein, MD.)

Box 87-2. Mosquito Avoidance

- Wear long-sleeved shirt and pants.
- Minimize outdoor activities at dusk.
- Use mosquito netting to cover the heads of infants and children and sleeping area.
- Spray clothing, netting, and screens with permethrin (Permanone or Duranon).
- Use insect repellents with no more than 35% DEET or picaridin on exposed skin only, avoiding children's hands.
- Apply repellents over any other creams such as sunscreen to minimize absorption and maximize the repellent effect.
- Keep repellents out of reach of small children.

DEET, *N,N*-diethyl-meta-toluamide.

data from the American Association of Poison Control Centers refuted the commonly held belief that children are more susceptible to DEET toxicity. The study demonstrated that children actually have less severe outcomes after DEET exposures than do adults.[5] Although products containing 100% DEET are commercially available, long-acting formulations of 35% DEET appear equally effective in protecting against mosquitoes, with much less potential for toxicity.[81] Although available data do not permit precise safety guidelines, infants younger than 6 months should avoid DEET, but those older than 6 months can use products with up to 30% to 35% DEET. The repellent effect can last up to 8 hours, but heavy perspiration or swimming should prompt reapplication every 6 to 8 hours. Dawn and dusk are particularly risky times; outdoor activities should be limited at these times if possible. Parents should not allow children under 8 years old to apply DEET for themselves, because of the risk of DEET exposure to eyes or mouth in this age group. DEET products can be applied over other creams such as sunscreen to minimize absorption and maximize repellent effect. Parents should also be cautioned to keep DEET out of the reach of small children, since ingestion may be fatal.

Picaridin (Bayrepel) is a recently developed alternative to DEET. Commonly used in Europe and Australia, it is odorless, minimally toxic, and considered by the World Health Organization (WHO) and the U.S. Environmental Protection Agency (EPA) to be an effective insect repellent. Oil of lemon eucalyptus is another EPA-approved insect repellent but its efficacy is similar to that of low concentration DEET products. Its use is therefore not recommended in areas heavily infested with mosquitoes. Other alternative insect repellents are widely available and often more appealing to parents, since their active ingredients are "natural." Unfortunately, agents such as citronella, lavender oil, and soybean oils have demonstrated very limited efficacy in repelling arthropods.[43] While natural products may be acceptable in areas where mosquitoes do not carry disease, with West Nile Virus now present across the United States and malaria and dengue fever endemic in many parts of the developing world, these products are simply inadequate. In addition, the above oils have been associated with aspiration pneumonitis when accidentally ingested by young children.

The pesticide permethrin, available as a 0.5% spray (Permanone, Duranon Tick Repellent), is safe and effective against arthropods, especially ticks. Unlike DEET repellents, these permethrin products are applied to fabric or netting, not to skin. Permethrins as a class have low toxicity in mammals. The combination of DEET applied to exposed skin and permethrin treatment of clothing is particularly effective in protecting against mosquito bites and ticks. In one study, this combination provided 99% protection for 8 hours.[47] The protective effect of mosquito netting is also greatly enhanced when the netting is impregnated with permethrin.[81] These effects are longer lasting with permethrin soaks (up to 20 washings) than with permethrin spraying of clothing or netting.

West Nile Virus (WNV) has become a great concern in recent years given its rapid spread via migrating birds across the United States (see Chapter 42). WNV is a zoonotic disease transmitted from animal hosts to humans via infected *Culex* mosquitoes. 20% of infected individuals will develop a mild nonspecific febrile illness lasting less than a week. Fewer than 1% of infections will result in encephalitis. Severe manifestations of WNV are more common in adults than children but more than 110 cases of WNV-related neurological disease were reported in children in 2003.[33] Protection against mosquito bites is the only means of prevention of WNV-related disease in humans.

Ticks

Like mosquitoes, ticks serve as vectors for disease, most notably Lyme disease (see Chapter 45). Lyme disease is rare on the Pacific coast but endemic in the Northeast, where up to 30% to 50% of *Ixodid* ticks are carriers.[77] Transmission of the Lyme spirochete, *Borrelia burgdorferi,* typically requires 48 to 72 hours or more of tick contact.[78] Therefore, tick checks should be conducted regularly while traveling through wilderness areas. If a tick is found imbedded in skin, it should be grasped with forceps close to the skin surface and gentle traction should be applied. The use of alcohol or open flames is strongly discouraged, because these techniques do not tend to work and can induce tick salivation or regurgitation into the wound.

After tick removal, parents should remain vigilant for the appearance of a large erythematous or targetoid annular lesion at the bite site within 7 to 10 days. This rash, erythema migrans, is either painful or pruritic and is characteristic of early localized Lyme disease. If neglected, the rash will spread and fever, neurologic symptoms, and arthritis may develop. Treatment for early-stage Lyme disease is amoxicillin 40 mg/kg/day (500 mg max; divided into three doses) for 14 to 21 days (up to age 8 years), or doxycycline 2.2 mg/kg (100 mg max) twice a day for 14 to 21 days (older than 8 years). A vaccine against Lyme disease (LYMErix) is no longer commercially available. As with mosquitoes, clothing and chemical barriers such as DEET and permethrin are the first lines of defense against ticks and the diseases they carry.

FOREIGN TRAVEL WITH CHILDREN

Visits to foreign countries provide superb educational, social, and cultural experiences for children. Unfortunately, traveling to wilderness or rural areas within developing nations entails the risks not only of wilderness travel but also of poor sanitation conditions with consequent exposure to bacteria, viruses, protozoa, and helminths not usually seen in the developed world. Although there is little consensus among experts, parents should be made aware of the greater risk of traveling to a developing country with a child under 2 years or weighing less than 15 kg. This increased risk is the result of incomplete immunizations, an underdeveloped or naive immune system, poor hygiene, and age- and weight-based contraindications for vaccinations and various medications.

It is the role of the physician to provide guidance and information to parents planning foreign travel. The emergence and widespread availability of the Internet has provided both the physician and the traveler with easily accessible and up-to-the-minute guides to disease outbreaks, immunizations, and symptoms and treatments for various tropical diseases. Reliable sources for this type of information are listed in Table 87-5.

More specifically, preparation for foreign travel with children includes identification and avoidance of risky endeavors; administration of appropriate immunizations and prophylactic medications; knowledge of common childhood diseases and the means of treating them; and, finally, good post-travel follow-up by an informed physician.

TABLE 87-5. Resources for Current Travel Immunizations, and Malaria Prophylaxis Recommendations

RESOURCE NAME	URL	COMMENTS
U.S. Centers for Disease Control and Prevention (CDC)	www.cdc.gov	General website
	www.cdc.gov/travel/	Index of travel information, vaccines, disease outbreaks
		Access to "Bluesheet" summary of indicated vaccines and "Yellow Book" online text of health information by destination country
	www.cdc.gov/mmwr/international/world.html	*Morbidity and Mortality Weekly Report* online: International bulletin on disease outbreaks
	www.cdc.gov/ncidod	National Center for Infectious Diseases
Medical College of Wisconsin International Traveler's Clinic	http://healthlink.mcw.edu/travel-links.html	Excellent links
U.N. World Health Organization (WHO)	www.who.int/en/	—
Travel Medicine Providers	www.tripprep.com	—
Travel Internationally with Your Kids	www.travelwithyourkids.com/	Parent-friendly website with much practical information

Box 87-3. Risk Avoidance during Travel

- Select appropriate settings: supervised swimming, safe campsites, protective devices.
- Protect skin: sunscreen, repellents, protective clothing, closed shoes.
- Eat and drink cleanly: water disinfection, careful food selection, "Boil it, peel it, cook it, or forget it."
- Avoid bugs and wild animals: clothing, netting, repellents, vigilance, safe practices.

General Recommendations

Physicians should emphasize to families that risk avoidance is the most important aspect of safe travel. The basic tenets of safe foreign travel with children are listed in Box 87-3. Parents should select transportation, activities, and overnight settings carefully to avoid unnecessary hazards. Fresh-water swimming should be avoided in developing countries to prevent parasitic infections such as schistosomiasis. Where swimming is appropriate, parents should provide close supervision. In one study in Washington State, drowning was the leading cause of pediatric mortality in the wilderness, followed by closed head injury and exposure.[57] Because traumatic injury is the leading cause of morbidity and mortality among children, prevention is the key. Protective devices such as car seats, helmets, personal flotation devices, and protective clothing and pads should be used as often as possible (Fig. 87-10). Skin protection is vital in the outdoors, particularly in tropical environments. The use of closed shoes can prevent infections with various hookworms, *Strongyloides,* and other parasites that enter through the skin. Clothing selections should be based on ambient temperature and expected conditions. Even in hot, sunny climates, light clothing should cover as much body surface as possible to provide protection from parasites, insects, and ultraviolet light. Sunscreen should be used liberally and reapplied every few hours, particularly after swimming.

Figure 87-10. Appropriate helmet use by a child. (Courtesy Judith R. Klein, MD.)

Box 87-4. Prevention of Traveler's Diarrhea

- Eat only well-cooked vegetables, meat, and seafood.
- Eat only fruit that can be peeled.
- Drink only disinfected or boiled water, carbonated drinks, hot teas, or coffee.
- Drink or eat only pasteurized dairy products.
- Avoid ice cubes or use only those made from disinfected water.
- Breastfeed infants.
- Prepare formula with disinfected or boiled water only.
- Wash hands thoroughly before eating or preparing food.
- Brush teeth with disinfected water.
- Beware of food from street vendors.

Parents should be advised to take great care in selecting foods and safe drinking water for themselves and their children, who are particularly vulnerable to disease (Box 87-4). All foods should be either well-cooked, canned, or peeled. All milk products consumed should be either pasteurized or boiled. Water should never be consumed from the tap; only bottled, 1-minute-boiled, or chemically treated (iodine) *and* microfiltered (0.2-µm pore size) water should be ingested. In certain areas, even bottled water is suspect. Breastfeeding is safest for young infants. If formula is used, only properly disinfected water should be used for its preparation. Finally, good and frequent hand-washing, particularly with infants and toddlers and especially around mealtimes, does successfully interrupt the fecal–oral passage of disease.

Parents should also focus their efforts on avoiding contact between their children and insects or wild animals. Mosquitoes and ticks can be avoided by limiting outdoor activities between dusk and dawn and by wearing protective clothing and repellent (see Box 87-2). As mentioned previously, DEET is an effective repellent but should not be used at concentrations greater than 35% in children because of the risk of toxicity.[81] Children's hands should be free of DEET to prevent accidental eye and mouth contact. Outer clothing and bed netting should be treated with permethrin. Check for ticks daily when in tick-infested areas. Children should be warned to watch where they place their hands and feet (in crevices, unattended shoes, sleeping bags) to avoid the unexpected arthropod or snake.

Immunizations

Foreign travel with children requires advance planning because vaccines recommended for travel to certain countries may take up to 6 weeks to complete. The Centers for Disease Control (CDC) website (www.cdc.gov/travel) provides up-to-date information on immunizations and prophylaxis based on the following:

- Countries of travel
- Length of time in each country
- Location of destinations (rural versus urban)
- Time of year
- Type of lodging and eating facilities
- Previous immunizations
- Age and weight of the child

Vaccines may be categorized as routine (polio, diphtheria-tetanus-pertussis, *Haemophilus influenzae* B, pneumococcus, hepatitis B, measles-mumps-rubella, varicella, influenza); routine for travel (hepatitis A, immunoglobulin); geographically indicated or required (yellow fever, typhoid, meningococcus, Japanese encephalitis); and indicated for extended stay (rabies)[86] (Table 87-6). The indications, dosages, and schedules for administration of these immunizations are listed in Table 87-7. The risk of acquiring diseases covered by many routine childhood immunizations (diphtheria, pertussis, tetanus, measles, polio, *Haemophilus influenzae* B, pneumococcus, influenza, and hepatitis B) is greater when traveling to developing countries because of the inconsistent immunization and subsequent loss of herd immunity. Influenza A vaccine, although not routinely given to all children in the United States, should be given to all children older than 6 months traveling during influenza season: December to April in the Northern Hemisphere and April to October in the Southern Hemisphere. In addition, children who have not completed their primary series of immunizations may require an acceleration of the vaccination schedule or extra doses to maximize protection before travel.

TABLE 87-6. Categories of Vaccines for Children

TYPE OF VACCINE	EXAMPLES
Routine	Polio, diphtheria-tetanus-pertussis (DTaP), *Haemophilus influenzae* B (Hib), measles-mumps-rubella (MMR), pneumococcal (PCV-7), varicella (VZV), hepatitis B, influenza
Routine for travel (developing countries)	Hepatitis A or immune globulin (IgG)
Required or geographically indicated	Yellow fever, typhoid, meningococcal, Japanese encephalitis
Extended stay	Rabies

From U.S. Centers for Disease Control and Prevention (www2.ncid.cdc.gov/travel/yb).

Ideally, a medical visit to discuss travel plans and start immunizations should be made 6 weeks before travel. Not all immunologic agents recommended for travel are compatible, and some require multiple doses. Therefore, the selection of immunizations to be given at any one time and the interval between immunizations are important. Table 87-8 presents recommendations for the timing and sequence of specific travel immunizations. In general, all toxoid, recombinant, inactivated, and live-attenuated vaccines may be given simultaneously. Live-attenuated vaccines should be given either simultaneously or at least 30 days apart to avoid reduced immunoreactivity to each vaccine.

In the United States, hepatitis A vaccine is recommended for children greater than 2 years old. Of note, the World Health Organization (WHO) recommendations include children 1 year of age and older. For children unable or unwilling to receive this vaccine, immunoglobulin (Ig) can provide passive hepatitis A prophylaxis. Administration of immune globulin interferes with the humoral response to the live-attenuated virus vaccines, such as measles, mumps, and rubella (MMR) and varicella. If immune globulin is given first, then these vaccinations should be delayed by at least 6 weeks and preferably 3 months to obtain an adequate immunogenic response. When both are needed for travel, it is best to give MMR or varicella vaccine first; the immune globulin can be given closer to the time of

TABLE 87-7. Vaccines for Children Who Travel

VACCINE	AGE AT VACCINATION	DOSING	COMMENTS/CONTRAINDICATIONS
Routine			
Polio (IPV)—inactivated injectable	2 mo	2, 4, 6 to 12 mo, 4 to 6 yr	Oral PV is no longer recommended due to risk of vaccine and associated paralytic polio in undiagnosed immunocompromised infants
Diphtheria-tetanus-acellular pertussis (DTaP) Booster (Td)	2 mo	DTaP at 2, 4, 6, 15 to 18 mo and 4 to 6 yr; Td booster at age 12 then every 10 yr thereafter	Large retrospective study indicated no increased risk of autism with DTaP
Haemophilus B (Hib) Polysaccharide conjugate	2 mo	2, 4, 6, 12 to 15 mo	Typically given as combination with DTaP
Pneumococcal (PCV-7): 7-valent conjugate vaccine	—	2, 4, 6, 12 to 15 mo	Use protein polysaccharide vaccine for certain high-risk groups >5 yr old
Hepatitis B Inactivated viral antigen	Birth	3 doses: 0, 1, 6 mo	Some protection after just 1 or 2 doses
Measles-mumps-rubella (MMR) live-attenuated virus	12 to 15 mo	12 to 15 mo, then booster at 4 to 6 yr or 11 to 12 yr	Give at least 2 to 3 wk before any IgG
Varicella live-attenuated virus	12 mo	12 mo to 12 yr: single dose >12 yr: 2 doses, 4 to 8 wk apart	Give at least 2 to 3 wk before any IgG, may be given with MMR at different sites, avoid if immunocompromised
Influenza A			
IM: inactivated virus	6 mo	Single dose If 1st vaccination, 2 doses, 1 mo apart	Influenza season Dec to April in Northern Hemisphere, April to Oct in Southern Hemisphere. All children traveling during influenza season should receive vaccine.
Intranasal: live-attenuated virus	5 yr	Single dose	Live-attenuated vaccine only for healthy, nonimmunocompromised children >5 yr
Routine for Travel			
Hepatitis A			
Inactivated virus	2 yr	2 doses, 6 to 12 mo apart	Preferred for hepatitis A protection if ≥2 yr, WHO recommendation ≥1 yr. Protects in 4 wk after first dose.
Immune globulin (Ig) antibodies	Birth	<2 yr: 0.02 mL/kg Good for 3 mo, administer 2 wk prior to travel	Hepatitis A protection for those <2 yr; beware of timing with live virus vaccines.
Required or Geographically Indicated			
Yellow Fever			
Live virus	9 mo	At least 10 days pre-departure; booster every 10 yr	Required for parts of sub-Saharan Africa and tropical S. America, may give at 6 to 9 mo if traveling to epidemic area; under 9 mo, risk of vaccine-related encephalitis
Typhoid			
(ViCPS) injectable polysaccharide	2 to 6 yr	Single dose: booster every 2 yr	Important for Latin America, Asia, and Africa Vaccine not a substitute for eating and drinking cleanly
(Ty21a) oral live attenuated	>6 yr	1 capsule every 2 days, 4 times; booster every 5 yr	
Meningococcal (ACYW-135) polysaccharide vaccine	2 yr	Single dose, booster in 1 yr if 1st dose <age 4; otherwise, every 5 yr	Use for central Africa, Nepal, and epidemic areas; minimal efficacy under 2 yr
Japanese encephalitis inactivated virus	1 yr	At 0, 7, 14 to 30 days Last dose >10 days before travel	Indicated for parts of India and rural Asia if stay >1 mo; no safety data for under 1 yr of age; high rate of hypersensitivity
Extended Stay			
Rabies			
Human diploid cell vaccine (HDCV)	Birth	Days 0, 7, 21 to 28 if >1 mo stay	If exposed to rabies and previously immunized: • Repeat vaccine 1 mL IM days 0, 3 If exposed and not previously immunized: • Give rabies Ig (RIG) 20 IU/kg half at site of exposure and half IM • Give vaccine 1 mL IM days 0, 3, 7, 14, 28

Data from Centers for Disease Control and Prevention: Vaccine recommendations for infants and children. In Travelers' Health: Yellow Book: Health Information for International Travel, 2005–2006. Available at www2.ncid.cdc.gov/travel/yb/utils/ybGet.asp?section=children&obj=child-vax.htm&cssNav=browseoyb

TABLE 87-8. Recommended Timing and Sequence of Nonroutine Immunizations for Foreign Travel

4 TO 6 WEEKS BEFORE DEPARTURE	1 WEEK AFTER INITIAL VISIT	WEEK OF DEPARTURE
Hepatitis A (need 2nd dose 6 to 12 mo later)	—	**Or** immunoglobulin (Ig) for hepatitis A prevention
Yellow fever	—	
Typhoid ViCPS **or** Ty21a	—	
Meningococcal	—	Meningococcal (if not given at initial visit)
Japanese encephalitis	Japanese encephalitis	Japanese encephalitis
Rabies	Rabies	Rabies

Give only immunizations indicated for area of travel, length of stay, and age of child.
Simultaneous administration of routine and travel-related vaccines is acceptable with the exception of measles-mumps-rubella or varicella with Ig. Administer Ig at least 3 wk after these live virus vaccines.

travel, at least 2 weeks and preferably 4 weeks later. Immune globulin does not interfere with antibody production of the live virus vaccines (yellow fever) and may be given at the same visit.[65] Travel vaccines that require multiple doses or early administration include hepatitis A, Japanese encephalitis, rabies, and yellow fever. Of note, cholera vaccine is no longer recommended by the CDC because of its limited efficacy. The only exception is when travel is planned to a region threatened or affected by cholera.

Prophylaxis: Malaria

The risk of acquiring malaria (see Chapter 43) during visits to developing countries in the tropics is significant. Even areas where the overall risk is relatively low may have foci of intense transmission. Children younger than 5 years are particularly vulnerable and represent the largest proportion of fatalities from this disease. Of the 30,000 to 50,000 cases of malaria acquired by travelers from Europe and North America per year, most occur among visitors to sub-Saharan Africa (82%) and Asia (8%).[48] In 1999, 21% of the 1421 cases of malaria in the United States reported to the CDC were in children.[81]

Protective measures to prevent mosquito bites help interrupt the transmission of malaria but are not foolproof (see Box 87-2). Therefore, chemoprophylaxis is highly recommended for travelers to countries where malaria is endemic. *Plasmodium vivax* and *Plasmodium falciparum* are the two most abundant species responsible for malaria. The most lethal plasmodium, *P. falciparum,* has developed widespread resistance to chloroquine and, in some areas (extreme northern and southern Thailand, Cambodia, Burma), resistance to mefloquine. Therefore, the choice of prophylactic agent depends on the presence of resistant malaria in the area of travel. Table 87-9 lists the available options for malaria chemoprophylaxis.

Chloroquine (Aralen) is still the drug of choice for travelers to the few areas where chloroquine-resistant strains of *P. falciparum* are not a problem; this includes parts of northern and western Central America, the Caribbean, the Middle East, and North Africa. Chloroquine prophylaxis should be given weekly, starting 1 week before travel and continued for 4 weeks thereafter. Chloroquine is passed through the breast milk but not in sufficient quantities to protect an infant. Therefore, a breastfed infant should receive chloroquine prophylaxis in standard recommended doses (see Table 87-9). Chloroquine is not readily available in a liquid form in the United States. The powder, which is extremely bitter, may be suspended in a syrup or mixed with food. Instant pudding effectively masks the bitter taste and makes the medicine more palatable. An acceptable-tasting chloroquine liquid (Nivaquine) is available in most developing countries. Chloroquine should be kept out of the reach of children. As little as 300 mg may be fatal in small children. If a toxic chloroquine ingestion occurs, syrup of ipecac should be administered immediately to provoke emesis, and the child should be transported promptly to a medical facility.

For children traveling to chloroquine-resistant areas, mefloquine (Lariam) is the preferred agent. It is started 1 to 2 weeks before travel and dosed weekly, during, and for 4 weeks after return from travel. It should be avoided in children with psychiatric illnesses, epilepsy, or underlying cardiac arrhythmias. It is available only in tablet form, but tablets may be administered crushed. Alternatively, a compounding pharmacist may prepare the child's dose in a gelatin capsule and the contents of the capsule can be placed in food just prior to administration. Doxycycline is an option for children older than 8 years with contraindications to mefloquine use. Unlike mefloquine, however, it must be dosed daily. It should be started 1 to 2 days before and continued for 4 weeks after travel to a malarial area. Side effects include diarrhea and photosensitivity. An alternative to mefloquine or doxycycline for children traveling to chloroquine- or mefloquine-resistant areas is atovaquone plus proguanil (Malarone). Malarone is approved in the United States for children over 11 kg and requires daily administration. A few, as yet unpublished, studies from Africa have demonstrated the safety of Malarone in children as small as 5 kg, but dosing data have not yet been made available.[81] It must be taken 2 days before entry into a malarial area and continued for 7 days after leaving. It is contraindicated in the setting of severe renal impairment (creatinine clearance, <30 mL/min).

In the event that a child develops an acute febrile illness in a malaria-endemic area where medical care is not immediately available, pyrimethamine-sulfadoxine (Fansidar) may be used as standby treatment. Fansidar is given as a one-time dose. Prophylaxis with this drug is not recommended because of the risk of Stevens-Johnson syndrome. Fansidar should not be given to an infant less than 2 months old or to anyone allergic to sulfa medications. Malarone is an alternative standby treatment that has been used successfully in Fansidar-resistant areas (parts of Southeast Asia and the Amazon basin).[50,63] All patients who take Fansidar or Malarone for presumptive treatment of malaria should be transported to a medical facility as soon as possible for definitive care.

TABLE 87-9. Malaria Chemoprophylaxis

MEDICATION	INDICATIONS/CONTRAINDICATIONS	DOSING
Chloroquine (Aralen) (liquid form is Nivaquine)	Travel to chloroquine-sensitive areas (Caribbean, Central America north of Panama, Middle East) Safe in children under 15 kg	5 mg base/kg/wk up to 300 mg for 1 wk pre- and 4 wk post-exposure; 10 mg/mL liquid available outside United States
Mefloquine (Lariam)	Travel to chloroquine-resistant areas Do not use in setting of epilepsy, psychiatric illness, cardiac arrhythmias	Given weekly: 1 wk pre- and for 4 wk post-travel Dosage: 5 mg/kg/wk 5–10 kg: 1/8 tab every wk 10–20 kg: 1/4 tablet every wk 20–30 kg: 1/2 tablet every wk 30–40 kg: 3/4 tablet every wk >40 kg: 1 tablet (250 mg)
Doxycycline	>8 yr of age only Alternative to mefloquine in chloroquine-resistant areas if contraindication to mefloquine exists; beware photosensitivity	2 mg/kg/day up to 100 mg/day 1 to 2 days pre-exposure and for 4 wk after exposure
Atovaquone (A)+Proguanil (P) (Malarone)	>11 kg Travel to chloroquine- or mefloquine-resistant areas Alternative to mefloquine or doxycycline Contraindicated if creatinine clearance <30 mL/min	Ped tab: 62.5 mg A/25 mg P 11–20 kg: 1 tab qd 21–30 kg: 2 tab qd 31–40 kg: 3 tab qd >40 kg: 1 adult tab qd (250 mg A/100 mg P)
Pyrimethamine-sulfadoxine (Fansidar)	Standby for treatment if malarial symptoms develop and medical attention >24 hr away Contraindicated if age <2 mo or sulfa allergy	Single dose: 2 mo–1 yr: 1/4 tablet 1–3 yr: 1/2 tablet 4–8 yr: 1 tablet 9–14 yr: 2 tablets >14 yr: 3 tablets
Primaquine	Prevention of relapse with *P. vivax* or *P. ovale* Use after prolonged stay in malaria-endemic area Avoid if G-6-PD deficient	0.6 mg/kg/day for 14 days after leaving area

G-6-PD, glucose-6-phosphate dehydrogenase.
Data from Centers for Disease Control and Prevention: Information for health care providers: Prescription drugs for malaria. Available at www.cdc.gov/travel/malariadrugs2.htm.

Primaquine is an antimalarial drug used to prevent emergence of *P. vivax* and *Plasmodium ovale* after heavy or prolonged (many months) exposure to mosquitoes. Routine chemoprophylaxis does not kill the exoerythrocytic stages of these *Plasmodium* species. Primaquine is taken daily for 2 weeks after leaving a malarial area. Primaquine should not be given to anyone with glucose-6-phosphate dehydrogenase (G-6-PD) deficiency.

TRAVEL-RELATED PROBLEMS

Next to boredom and restlessness, motion sickness and eustachian tube dysfunction are the most common problems encountered by children during travel. Parents can minimize the first two problems by preparing small activity packs or bags with paper, pencils, crayons, cards, travel puzzles, or small toys. Once at their destination, children most frequently suffer from respiratory tract infections, diarrhea, and fevers. Parents should be aware of the basic elements of diagnosis, treatment, and reasons to seek immediate medical attention.

Motion Sickness

Motion sickness can occur with air, land, or sea travel (see Chapter 76), particularly in children ages 2 through 12 years. Emotional upset, noxious odors, and ear infections can make symptoms worse. Children experiencing motion sickness are often pale and diaphoretic, and they feel nauseated and weak. They may vomit, but unfortunately this does not provide prolonged relief. Children known to be susceptible to motion sickness should be seated in the middle or near the front of the boat, plane, or car, where motion is minimized. They should be encouraged to look at objects far away and avoid focusing on close objects such as books. Some children get significant relief from using headphones to listen to music or stories.

Dimenhydrinate (Dramamine, 1.0 to 1.5 mg/kg) administered 1 hour before departure and repeated every 6 hours can help those known to be prone to motion sickness. If dimenhydrinate is not available, diphenhydramine (Benadryl, 1.25 mg/kg every 6 hours up to 50 mg) may also be effective. Both medications may cause drowsiness, and diphenhydramine occasionally causes paradoxical excitability in children. Scopolamine patches, commonly used in adults, should not be used in children because children are particularly susceptible to the side effects of belladonna alkaloids. Whether this particular administration system might release too much scopolamine and consequently produce serious side effects in children is not known.

Eustachian Tube Dysfunction

Eustachian tube dysfunction is the result of disequilibrium between pressure in the eustachian tube and the surrounding atmospheric pressure. As atmospheric pressure rises (e.g., with descent in an airplane), the eustachian tube becomes compressed if the pressure within the tube does not rise as quickly

as the ambient pressure. If compressed enough, the eustachian tube cannot equalize pressure in the middle ear with that of the environment, resulting in a sense of compression on the outer aspect of the tympanic membrane. Far more children than adults (nearly 15% of the pediatric population) suffer from this problem, because of the relatively smaller, and hence more compressible, eustachian tube in children. Swallowing often helps relieve the pressure disequilibrium and may be facilitated by drinking or, for the breastfed infant, by nursing. Older children may wish to chew gum or yawn to equalize middle ear and atmospheric pressure. Contrary to popular belief, decongestants are not useful with eustachian tube dysfunction in children and may cause paradoxical drowsiness.[8] This class of medications should be avoided.

Traveler's Diarrhea

Traveling to wilderness areas or developing countries requires leaving behind modern sanitation and reliably disinfected tap water. Unfortunately, this places travelers at increased risk for diarrheal illness (see Chapter 62). In one study, 40% of children less than 3 years of age developed prolonged diarrhea during travel in tropical or subtropical areas.[62] This risk was highest in young visitors to North Africa (71%) and India (61%), with much lower incidences in travelers to Asia and Latin America. Young children are at greater risk for traveler's diarrhea (TD) and its complications because of their relatively poor hygiene, oral exploration, immature immune systems, lower gastric pH, more rapid gastric emptying, and difficulties with adequate hydration.

TD is defined by the National Institutes of Health (NIH) as a twofold or greater increase in the frequency of unformed stools, or any number of such stools when accompanied by symptoms such as fever, abdominal cramping, vomiting, or blood or mucus in the stools. In small children, the course tends to be more severe and prolonged, lasting from 3 days to 3 weeks.[62] TD can be caused by preformed toxins, viruses, invasive bacteria, or parasites (Table 87-10). Enterotoxigenic *Escherichia coli* is responsible for 50% of TD.[6] Rotavirus and Norwalk-like viruses account for another 30%. Invasive bacterial pathogens account for most of the remaining cases of TD, with *Shigella* topping the list. In certain seasons and geographic areas, *Campylobacter* is also a frequent agent of invasive TD.

TABLE 87-10. Causes of Traveler's Diarrhea in Children

AGENT	EXAMPLES
Preformed toxin	Enterotoxigenic *Escherichia coli**
	Staphylococcus aureus
	Bacillus cereus
Viral	Rotavirus*
	Norwalk agent*
	Adenovirus
	Enterovirus
	Influenza
	Hepatitis
Bacterial	*Shigella**
	Campylobacter
	Salmonella
	Enteroinvasive *E. coli*
	Yersinia enterocolitica
	Vibrio cholerae
Parasitic	*Giardia lamblia*
	Entamoeba histolytica
	Cryptosporidium

*Most common.

Prevention

Standard recommendations for prevention of TD are based primarily on known potential vehicles for transmission of the illness (see Box 87-4). Transmission is through fecal–oral contamination, with water, food, and fingers as the most common vehicles. Careful selection and preparation of food and beverages can decrease the risk of acquiring TD. As mentioned previously, washing hands thoroughly before eating decreases bacterial carriage and also serves as a reminder to children of the need for precautions. The "boil it, cook it, peel it, or forget it" rule implies that all raw vegetables and salads should be avoided, meats and seafood should be well cooked, and fruits need to be properly peeled. See Chapter 61 for a complete discussion of water disinfection.

The use of antibiotics to prevent TD in children is not currently recommended by the CDC or the American Academy of Pediatrics (AAP) because of the risks of medication side effects and the development of antibiotic resistance. Bismuth subsalicylate (Pepto-Bismol) has been shown in adults to be an effective form of chemoprophylaxis against TD. No studies to evaluate dosing and safety in children have been performed. Therefore, and because of the risk of administering salicylates to children, the use of bismuth subsalicylate for TD prevention in children is not recommended. Probiotic agents, such as *Lactobacillus acidophilus,* have been studied and shown to be effective for both prevention and treatment of TD in children.[23,29,70] Their mechanism of action is not entirely clear but most likely involves competition with pathogens for intestinal receptors, improvement of immune function in the gut, or production of antibiotics by the probiotic agent with efficacy against pathogens.[23] *Lactobacillus* is available over the counter and dosing is one tablet or capsule a day for children less than 2 years old, and two a day for children older than 2 years. Capsules can be opened and placed into food or drink for children unable or unwilling to take pills.

Treatment

The major cause of morbidity and mortality in infants and small children with diarrhea is dehydration.[1] Signs of dehydration in children are listed in Box 87-5. According to the WHO, dehydration is best categorized as mild to moderate or severe.[89] This distinction is based on changes in behavior and mental state, quality of mucous membranes, oliguria or anuria, changes in vital signs, and decreasing peripheral perfusion. Parental assessment of dehydration using urine output, tearing, and fontanelle contour has been shown to be quite accurate.[82] Children young enough to be wearing diapers should have some urine output at least every 8 hours. If they do not, they are very likely dehydrated.

The cornerstone of treatment for TD is oral rehydration therapy (ORT), which, if instituted early, can be used alone in 90% to 95% of cases.[24] ORT is often tolerated by children who are vomiting if administered in small quantities frequently. ORT is as effective as intravenous hydration in mild to moderate dehydration caused by gastroenteritis.[24] Parents traveling to developing countries or wilderness areas with children should

Box 87-5. Signs of Dehydration

MILD TO MODERATE (5% TO 10%)
Irritability
Sunken eyes
Dry mucous membranes
Very thirsty

SEVERE (>10%)
Lethargy
Very sunken eyes
Very dry mucous membranes
Unable to take liquids orally
Cool, mottled extremities
Rapid, thready pulse
Tachypnea

Box 87-6. Homemade Oral Rehydrating Solutions (ORSs)

GLUCOSE-BASED*
1 teaspoon (5 mL) salt
8 teaspoons (40 mL) sugar
1 quart (1 L) disinfected water

RICE CEREAL–BASED*,†
1 teaspoon (5 mL) salt
1 cup (50 g) rice cereal
1 quart (1 L) disinfected water

*Can be used directly or diluted in half to reduce osmolarity.
†The rice cereal–based solution is stable for only 12 hours, after which it should be discarded and a new solution made.
Data from www.rehydrate.org.

TABLE 87-11. Field Treatment of Dehydration

	REHYDRATION SOLUTION	MAINTENANCE SOLUTION
Volume	50 to 100 mL/kg/4 hr (1 to 1.5 oz/lb/4 hr)	75 to 150 mL/kg/day* (1 to 2.5 oz/lb/day)
Electrolytes		
Na^+ (mEq/L)	75 to 90	50 to 60
Glucose (%)	1.5 to 2.5	1.5 to 2.5
K^+ (mEq/L)	20	20
HCO_3^- (mEq/L)	30	30

*Add 10 mL/kg or about 4 oz for each diarrheal stool and 5 mL/kg or about 2 oz for each bout of emesis.
Data from King CK, Glass R, Bresee JS, Duggan C: Managing acute gastroenteritis among children. MMWR 52(RR16):1–16, 2003.

carry powdered oral rehydration solution (ORS) or a recipe for a homemade solution (Box 87-6) Powdered ORS is readily available in most developing countries in pharmacies and drug stores. Most available ORS is glucose based. According to several studies, rice-based ORS is more effective in reducing stool output than standard glucose-based ORS.[17,55] Rice-based ORS has a lower osmolarity and higher concentration of organic solutes. As a consequence, the osmotic gradient across the intestinal lumen is lower and intestinal sodium transport is decreased, resulting in less stool water. Unfortunately, any advantage gained by rice-based ORS is lost after 6 hours of rehydration.[17,55] In addition, due to hydrolysis of starches, the rice-based ORS is stable for only 12 hours, after which time it must be discarded and a new solution made.[71] Alternatively, glucose-based ORS can be diluted 2:1 with clean fresh water to reduce the osmolarity of the solution. A Turkish study demonstrated reduced vomiting and diarrhea in children rehydrated with half-strength ORS.[60]

For rapid treatment of mild-moderate dehydration, 50 to 100 mL/kg of ORS should be administered over the first 4-hour period to rehydrate the child. Reduced-osmolarity ORS contains 75 to 90 mEq/L of sodium, 75 mEq/L (1.5%) glucose, and 20 mEq/L of potassium (Table 87-11). If a solution with more glucose is used, the osmotic pressure exerted by the carbohydrate in the intestinal lumen produces fluid losses greater than fluid absorption, thereby exacerbating diarrhea. Most colas, sports drinks, and juices contain 6% to 15% carbohydrate and, therefore, are not appropriate rehydration solutions. Once the child is rehydrated, the high sodium concentration in the rehydration solution poses a risk for hypernatremia if the solution is used for maintenance fluids or to prevent dehydration. For maintaining hydration, alternating ORS with a lower-sodium fluid, such as water or breast milk, will avert hypernatremia. Alternatively, a separate solution containing 50 to 60 mEq/L sodium can be used. Maintenance fluid volumes are 75 to 150 mL/kg/day. An additional 10 mL/kg ($^1/_3$ oz/kg) can be given for each diarrheal stool and 5 mL/kg ($^1/_6$ oz/kg) for each episode of emesis. If vomiting develops, most children will still tolerate ORS if given in small volumes (5 to 10 mL) every 5 minutes. Feeding of solid food, particularly complex carbohydrates and yogurt, should be resumed as soon as vomiting resolves; solid food promotes enterocyte regeneration and reduces duration of diarrhea.[1,82]

Although oral hydration is the cornerstone of therapy for TD, medications may occasionally be helpful. Antimotility agents, such as loperamide and diphenoxylate, are of no proven efficacy according to the AAP guidelines for acute gastroenteritis.[1] In general, they should be avoided. However, loperamide (see Table 87-12 for dosing information) may be used with children over 2 years of age if fever, bloody stool, and abdominal distention are absent. Other antidiarrheal agents, such as bismuth subsalicylate and attapulgite, have not been shown to be effective in children and should be avoided, particularly in those less than 2 years.[1] Treatment with antibiotics for TD is safe and effective in reducing TD severity and duration in adults but is not currently recommended by the AAP for use in children.[1] The use of antibiotics should be limited to children with fever, bloody stool, or abdominal distention in whom invasive bacterial diarrhea is suspected. Under age 3 to 6 months, the risk of bacteremia with *Salmonella* infections is significant and antibiotic treatment is also indicated. With increasing bacterial resistance to trimethoprim-sulfamethoxazole, azithromycin is now the drug of choice for treatment of bacterial diarrhea.[34,82] The dosage is 10 mg/kg on day 1, then 5 mg/kg once daily for 5 days. Fluoroquinolones, such as ciprofloxacin, are an alternative but are approved in the United States only for children over 18 years. Multiple studies have, however, demonstrated safety and efficacy in children as young as 6 months of age,

TABLE 87-12. Pediatric Medications in Wilderness Medical Kit

MEDICATION	INDICATION	DOSE
Topical Medications		
Antibiotic ointment (e.g., bacitracin or polymyxin)	Superficial skin infections	Apply as directed qd to tid
Topical corticosteroid (e.g., 1% hydrocortisone)	Contact or atopic dermatitis, insect bites, sunburn	Apply to affected areas bid to tid (use sunscreen aggressively; avoid >1% corticosteroid on face)
Antifungal cream (e.g., clotrimazole or miconazole)	Yeast at diaper area, groin, scalp, feet; ringworm	Apply bid for 7 to 10 days and for several days after rash has resolved
Desitin cream	Sun block, diaper area erythema	Apply thick coat as sunscreen or thin coat for diaper area
Permethrin (Elimite)	Scabies, lice, treatment for clothing and mosquito netting	Apply 5% cream from chin to soles of feet and wash after 8 to 14 hr, do not use in children <2 mo of age or on eyes, nose, mouth
Anesthetic eye drops (e.g., proparacaine)*†	Removal of superficial ocular foreign body	1 drop in affected eye for removal of foreign body, must patch eye for protection for at least 1 hr
Antibiotic eye ointment (e.g., erythromycin)†	Purulent conjunctivitis, suspected corneal abrasion	Use as directed tid to qid
Oral Medications		
Diphenhydramine 12.5 mg/5 mL elixir 25- or 50-mg capsules	Allergic symptoms, pruritus, insomnia, nausea, motion sickness	1.25 mg/kg/dose every 6 hr (up to 25 to 50 mg/dose), may cause paradoxical restlessness in children
Pseudoephedrine 15-mg chewable tabs 15 or 30 mg/5 mL elixir	Decongestant	2 to 6 yr: 15 mg every 6 hr 6 to 12 yr: 30 mg >12 yr: 60 mg
Acetaminophen 80 mg/0.8 mL drops 160 mg/5 mL elixir 80- and 160-mg chewable tabs	Fever control, pain	15 to 20 mg/kg every 4 to 6 hr up to 650 mg/dose
or Ibuprofen 40 mg/1 mL drops 100 mg/5 mL elixir 50- or 100-mg chewtabs 100- or 200-mg caplets	Fever control, pain, anti-inflammatory	10 mg/kg/dose every 8 hr up to 600 mg/dose
Acetaminophen with codeine† 120 mg acetaminophen and 12 mg of codeine/5 mL elixir	Severe pain, severe cough	As for acetaminophen, or 0.5 to 1.0 mg/kg of codeine every 4 to 6 hr
Dimenhydrinate (Dramamine) 12.5 mg/5 mL elixir 50 mg chewable tab	Motion sickness	1 to 1.5 mg/kg/dose 1 hr prior to departure and every 6 hr thereafter; may cause drowsiness
Oral Antibiotics (as Appropriate for Age of Child)		
Amoxicillin† 125 mg or 250 mg chewable tab 250 mg capsule	AOM, sinusitis, pharyngitis, pneumonia, UTI	80 mg/kg/day for UTI or pneumonia, divided tid
Amoxicillin-clavulanate (Augmentin)† 200 and 400 mg chewable tabs 200 and 400 mg/5 mL elixir	Penicillin-resistant organisms, AOM, sinusitis, animal bites, soft tissue infections	80 mg/kg/day divided bid
Azithromycin (Zithromax)† 125 and 250 mg/5 mL elixir 250 mg tabs (best)	AOM, sinusitis, pharyngitis, pneumonia, traveler's diarrhea, skin infections, animal bites	10 mg/kg on day 1, then 5 mg/kg/day qd×5 days
Ciprofloxacin (Cipro)† 100, 200,500 mg tabs	In U.S., approved for >18 yr of age only: UTI, traveler's diarrhea, wounds acquired in an aquatic environment	20 to 30 mg/kg/day divided bid up to 500 mg bid
Other Preparations		
Epinephrine (premeasured)† 0.15 mg EpiPen Jr 0.3 mg EpiPen	Anaphylaxis, severe asthma	0.15 mg SQ up to 15 kg, 0.3 mg SQ if over 15 kg
Oral rehydration packet (ORS)	Dehydration	See Table 87-11 for administration guidelines

TABLE 87-12. Pediatric Medications in Wilderness Medical Kit—cont'd

MEDICATION	INDICATION	DOSE
Foreign Travel		
Loperamide 1 mg/5 mL 1 mg caps	Nonbloody, minimally febrile, significant diarrhea over 2 yr of age	13 to 20 kg: 1 mg tid 20 to 30 kg: 2 mg bid >30 kg: 2 mg tid
Appropriate malaria prophylaxis if indicated†	—	See Table 87-9
Pyrimethamine-sulfadoxine (Fansidar)†	Standby treatment for malaria if immediate medical attention unavailable	See Table 87-9
Travel to High Altitude		
Acetazolamide (Diamox)† 30 or 50 mg/mL suspension 125 mg tab	Recurrent AMS despite graded ascent	5 mg/kg/day divided bid up to 250 mg/day
Phenergan suppositories† 12.5 or 25 mg/suppository	Nausea and vomiting associated with AMS; avoid with young children	0.25 to 0.5 mg/kg/dose every 6 hr up to 25 mg/dose

*Administration of this medication by other than trained medical personnel is strongly discouraged, given the risk of overuse and subsequent worsening of eye injury; if significant eye irritation persists, medical attention must be sought to evaluate for corneal injury.
†Available by prescription only.
AMS, acute mountain sickness; AOM, acute otitis media; bid, twice a day; qd, every day; tid, three times a day; UTI, urinary tract infection.

Box 87-7. Indications for Seeking Immediate Medical Attention for Children with Diarrhea

- Severe dehydration
- High fever
- Failure of oral rehydration therapy
- Failure of antibiotic treatment for bloody diarrhea
- Altered mental status

but increasing bacterial resistance to quinolones makes azithromycin an even better choice.[34,44,45] Severe dehydration, high fever, failure of ORT, failure of antibiotic therapy for bloody diarrhea, or altered mental status require prompt medical attention and administration of intravenous fluids for rehydration (Box 87-7). *Salmonella typhi*, or typhoid fever, should be managed initially in a hospital setting with a quinolone, such as ciprofloxacin, or a third-generation cephalosporin, such as ceftriaxone.

Constipation

Although not as serious a problem as diarrhea, constipation can be a significant and uncomfortable issue for children during travel. Changes in diet, a decline in fruit, vegetable, and water consumption because of sanitary concerns in developing countries, and viral illnesses can all contribute to the development of constipation. Constipation is defined as not only a decrease in stool frequency but also an increased firmness of the stool. Firm stool is more difficult to pass and can result in painful anal fissures and hemorrhoids. Children who experience anal pain during defecation may also choose to retain stool, exacerbating the constipation problem.

Parents should be encouraged to prevent the development of constipation or to manage it early in its course by providing safe fruits and vegetables for their children during travel and actively encouraging clean water consumption. Anal fissures and hemorrhoids should be managed with sitz baths, vitamin A&D ointment, and anti-inflammatory hemorrhoidal creams such as Anusol. If these approaches fail, bulk stool softeners (e.g., Metamucil) and, in extreme cases, hypertonic phosphate enemas (e.g., Fleets) can be used in children older than 1 year. Laxatives are not recommended because chronic use can actually result in paradoxical constipation. If constipation is accompanied by severe abdominal pain, distention, vomiting, or fever, children should be brought to medical attention for further evaluation.

Abdominal Pain

Abdominal pain is another common complaint in children. The etiology of abdominal pain ranges widely, from extremely benign viral infections or constipation to serious or life-threatening appendicitis or ectopic pregnancy. Caregivers accompanying children into the backcountry or to developing countries should be taught the basic indications for seeking medical treatment for this complaint. Abdominal discomfort accompanied by vomiting or diarrhea may be observed for 12 hours with appropriate hydration. If the pain worsens or fails to resolve, or the child develops a high fever, vomiting for greater than 24 hours, focal abdominal tenderness, or abdominal distention, the child should be brought to medical attention. If a child complains of suprapubic pain and dysuria, a 7-day course of antibiotics (e.g., amoxicillin or ciprofloxacin in older children) may be administered for presumed bladder infection. If the symptoms do not improve in 2 or 3 days, or if high fever or back pain develops, the child should be brought to medical attention because these symptoms suggest pyelonephritis. In adolescent girls, the presence of lower abdominal pain, amenorrhea, or vaginal bleeding suggests the possibility of ectopic pregnancy. If this is suspected, immediate medical evaluation with ultrasound is indicated.

Respiratory Infections and Otitis Media

Respiratory tract and otolaryngologic infections are very common in the pediatric population. The majority of infections, including acute otitis media (AOM), sinusitis, pharyngitis,

croup, bronchiolitis, and bronchitis, involve the upper respiratory tract. Most of these are viral in etiology; the remainder are the result of infections with *Streptococcus pneumoniae, Haemophilus influenzae,* or *Moraxella catarrhalis*. Because definitive diagnoses based on otoscopy and auscultation of the lungs often cannot be made in the course of wilderness travel, parents must rely on symptoms and the presence of fever to determine whether treatment is necessary. The presence of high fever, otalgia, facial pain, sore throat without cough or rhinorrhea, or mucopurulent nasal discharge lasting longer than 10 days probably merits both antimicrobial and symptomatic treatment. It should be noted, however, that 20% to 80% of AOM and 50% of streptococcal pharyngitis cases resolve without intervention.[12] Furthermore, antibiotic treatment of upper respiratory infections, such as bronchitis, bronchiolitis, croup, and most pharyngitis, does not shorten the course, minimize symptoms, or decrease complications.[53] If streptococcal pharyngitis is suspected based on the absence of cough or coryza and the presence of fever, cervical lymphadenopathy, and tonsillar exudate, then antibiotic therapy is warranted. Although antibiotics for streptococcal pharyngitis have minimal effect on the duration of the disease, they do reduce the incidence of suppurative complications, such as peritonsillar abscess, and nonsuppurative complications, such as rheumatic fever and poststreptococcal glomerulonephritis.

With increasing use of antibiotics, bacterial resistance to these agents has risen dramatically. Resistance is highly correlated with the rate of antibiotic use in a given population. Ten percent to 50% of *S. pneumoniae* isolates are resistant to penicillin, and 5% to 35% are resistant to macrolides such as erythromycin.[11] Up to 20% of *H. influenzae* and 70% of *M. catarrhalis* isolates are resistant to amoxicillin. However, minimal inhibitory concentration (MIC) testing has demonstrated sensitivity to high-dose amoxicillin, even among resistant strains. First-line therapy for suspected bacterial respiratory infection is, therefore, high-dose amoxicillin at 80 mg/kg/day divided into three doses per day. Second line therapy is high-dose amoxicillin-clavulanate (80 mg/kg/day divided into two doses per day), cefuroxime (15 mg/kg twice a day), or ceftriaxone (50 mg/kg once daily). Uncomplicated AOM in children older than 2 years may be treated for 5 to 7 days; otherwise, the standard treatment for AOM is 10 days, and for sinusitis 14 days.[53] Suspected streptococcal pharyngitis can be treated for 10 days with penicillin or amoxicillin, or for 5 days with azithromycin (10 mg/kg on day 1, then 5 mg/kg/day on days 2 to 5).

Fever of Unclear Etiology

High fever that develops in a child during the course of foreign travel may be the result of a common infection (AOM, pharyngitis) or the manifestation of a tropical disease. If malaria is suspected, the child should immediately be taken to the closest medical facility. If this cannot occur within 24 hours, any child older than 2 months should be given Fansidar as standby treatment as recommended in Table 87-9.

Typhoid fever, caused by *Salmonella typhi,* can also present with high fever, paradoxical bradycardia, and, at least initially, without diarrhea. Children who appear ill with high fevers may have invasive typhoidal disease and should be treated in a medical facility. *Salmonella* resistance is rising in developing countries, but strains are typically susceptible to quinolones and third-generation cephalosporins (e.g., ceftriaxone).

Dengue fever, caused by a mosquito-borne arbovirus, can also cause high fever, headaches, and severe myalgias. With the exception of recurrent dengue, which can result in hemorrhagic complications, the treatment of dengue fever is purely symptomatic. Of note, dengue is a disease found not only in the developing world. The southeastern United States and Hawaii are infested with the dengue-carrying *Aedes aegypti* mosquito.

Headache

Headache is a common travel-related problem in the adolescent population. The most common causes of headaches in children are febrile illnesses, migraines, dental infections, sinusitis, and tension or fatigue. Life-threatening causes of headache, such as meningitis, spontaneous or post-traumatic intracranial bleeding, or mass lesions, should be considered in any child with this complaint. Parasitic diseases, such as dengue fever and malaria, can also cause severe headache.

A child with a tender tooth or tenderness to palpation of his sinuses should be managed with analgesics (e.g., acetaminophen with or without codeine or ibuprofen) and antibiotics. Dental infections respond well to penicillin and a visit to the dentist for definitive management. See Respiratory Infections and Otitis Media, earlier, for information on the antibiotic treatment of sinus infections. A decongestant, such as pseudoephedrine, should also be given to children with sinusitis to improve sinus drainage. If a child has had previous migraine headaches or has a history that is classic for migraine (e.g., unilateral headache, aura, visual scotoma, photophobia, nausea, family history of migraine), analgesics and rest in a quiet, dark room are the appropriate first-line therapies. Caffeine, in the form of a strong cup of coffee, is also a helpful improvisational treatment for migraine headaches. If a child's headache is accompanied by high fever, neck stiffness, vomiting, confusion, neurologic abnormalities, or a recent history of trauma, the child should be brought to medical attention immediately.

Conjunctivitis

Conjunctivitis, or "pinkeye," is another common pediatric problem. Most conjunctivitis in children is either allergic or infectious (adenovirus, *Staphylococcus, Streptococcus,* or *Haemophilus*). Itching and clear discharge suggest an allergic etiology that is best treated with a topical or systemic antihistamine (e.g., diphenhydramine). The absence of itching, along with a slightly thicker or colored discharge from one or both eyes, is more consistent with an infectious cause of conjunctivitis. Distinguishing between viral and bacterial etiologies can be very difficult, so all suspected infectious conjunctivitis should be treated with a topical antibiotic, such as erythromycin ointment, four times a day for 1 week. Eye discharge should be wiped from the eye with a warm moist cloth, and contact precautions should be exercised to prevent spread of what may be a very infectious condition until the discharge resolves.

If the presence of a foreign body in the eye is suspected or there is a chemical exposure to the eye, a drop of a topical ocular anesthetic, such as proparacaine, should be applied and the eye flushed vigorously with water. Caregivers should then administer oral analgesics to the child and apply a topical antibiotic ointment (e.g., erythromycin) to the eye four times a day for 24 to 48 hours. If the foreign body sensation persists after 1 to 2 days, there is likely to be a retained foreign body and the child should be brought to medical attention. In addition, the presence of eye pain, periorbital swelling, fever, photophobia, or visual blurring

with or without a history of antecedent eye trauma or contact lens use suggests a more serious diagnosis and should prompt immediate medical attention.

Rashes

Identification of rashes in the pediatric population is an art. Although parents cannot be expected to become familiar with all types of rashes, they should learn to distinguish common rashes from potentially dangerous exanthems. Petechial, purpuric, or mucosal lesions are markers for potentially serious disease and should prompt parents to seek medical attention immediately. Standard dermatology textbooks with photographs can be used to instruct parents to recognize common viral exanthems and rashes caused by varicella, scabies, and contact dermatitides (e.g., poison oak or poison ivy). Scabies can most safely be abolished with permethrin. Contact dermatitis often can be simply treated with 1% hydrocortisone cream and diphenhydramine (see Chapter 57).

Soft Tissue Injuries

Soft tissue injuries are extremely common among children, particularly as they become more ambulatory. All wounds sustained in the wilderness should be thoroughly irrigated with clean water and explored to ensure that no foreign bodies remain. In general, lacerations should not be sutured in the backcountry because of the risk of infection. They should be covered with a water-resistant dressing, splinted if located over a joint, and observed. If no evidence of infection develops over 4 to 5 days, the laceration may be repaired, if necessary, by delayed primary closure.

Poisoning

In their first few years of life, children explore their environments with their hands and their mouths. Plants, flowers, mushrooms, and medications, including those in the pediatric first-aid kit, are all potentially tasty treats in the eyes of a toddler. Travel into the wilderness expands the repertoire of available objects for a child to explore orally. In an urban environment or during travel in the United States, a call to the local poison control center (800-222-1222) can provide immediate information regarding the dangers of a particular ingestion and the need for emergency treatment. In the wilderness and when traveling abroad, particularly in developing countries, this is rarely an option. Because it is unrealistic to expect a parent to know whether a certain medication, cosmetic, plant, or mushroom is toxic, the best advice to parents is prevention.

All medications and cosmetics should be kept out of reach and stored in child-resistant containers. Children should never be enticed to take their own medications by referring to medicines as "candy." During forays into the wilderness, parents should not let toddlers out of eyesight and should explain to their children why plants or mushrooms should not be put in their mouths. If a child's skin or eyes are exposed to a toxic substance, the area should be flushed vigorously with clean, preferably disinfected, or tap water for 15 to 20 minutes. Ipecac syrup, a powerful emetic and once a mainstay of home treatment for ingested poisons, is no longer recommended by the AAP. Ipecac's efficacy in removing toxic substances from the stomach is limited and inconsistent and, therefore, not worth the severe vomiting and lethargy that result from its administration. If a child ingests a plant, mushroom, or medication whose toxicity the caregiver is unfamiliar with, and calling a poison center is not an option, the child should be taken to a medical facility for evaluation.

Foreign Bodies

In addition to exploring the world around them with their mouths, toddlers and even young school-age children enjoy placing objects into their noses and ears. Such objects, particularly if they are organic in nature, can result in infection of the sinuses and external ear, respectively, and are best removed promptly.

Objects that are easily visible in the nose near the nares can be removed by two caregivers, one holding the child's head motionless while the other carefully removes the object with forceps or tweezers. If the object is not near the nares or cannot be easily visualized, a "mouth-to-mouth" technique can be used to try to remove the object. While occluding the uninvolved nare with one finger, the parent should place her mouth over the child's mouth and blow hard several times. Ideally, this creates enough pressure to expel the object from the nasal passage. If an object is lodged in the child's ear, it is best removed with forceful irrigation with warm (37° C [98° F]) (or clean, preferably disinfected) water using a bulb syringe. If the object is an insect, the ear should first be filled with isopropyl alcohol for several minutes to kill the insect prior to irrigation, unless there is a possibility of tympanic membrane perforation. If these techniques fail to remove the foreign body from the child's ear or nose, the child should be brought to medical attention so that the object can be removed by direct visualization under proper procedural sedation.

SPECIAL CONSIDERATIONS FOR CHILDREN WITH MEDICAL PROBLEMS

Asthma

Parents of children with asthma face unique challenges when traveling into the wilderness or to the developing world. In both situations, access to medical care may be limited and parents may need to treat exacerbations that would normally require advanced medical attention. In addition, exposure to known asthma triggers, such as tobacco and specific allergens, may not be as controllable while traveling. A study of adult adventure travelers demonstrated that the major risk factors for asthma exacerbations while traveling include poorly controlled asthma prior to departure and intensive physical exertion.[27]

To prepare for travel, parents should work with their pediatrician to stabilize their child's asthma to the extent possible. Any physical exertion planned should be graded so that the child's response can be gauged and activities scaled back as necessary. In locales with heavy particulate pollution or in settings with significant tobacco exposure, the child's activities should be limited. In addition to packing extra canisters of the child's inhalers and an extra spacer/aerochamber, parents should carry a course of high-dose steroids for administration during an acute exacerbation not responding immediately to an inhaled bronchodilator (e.g., albuterol). Options for steroids to carry along include prednisone or prednisolone 2 mg/kg on day 1, then 1 mg/kg on days 2 to 5 (max, 60 mg per day); or dexamethasone 0.6 mg/kg/day for 2 days (max, 10 mg/day). For children 5 years and older, a peak flow meter is also a useful item to carry to assist in gauging the severity of an asthma exacerbation.

Sickle Cell Disease

For children with sickle cell disease, the focus during travel should be on prevention. Parents should be aware of the triggers for the pain crisis characteristic of this disease, and should emphasize good hydration and hygiene to prevent diarrheal illness. Travels to high altitude should in general be avoided, because of the possibility of altitude-related hypoxia resulting in a pain crisis. If a crisis develops, the child should be treated with rest, oral hydration, and appropriate pain medications (ibuprofen or narcotics). If this therapy is ineffective or if the crisis involves chest or abdominal pain, parents should seek immediate medical attention for their child.

Fever is a common and significant problem for children with sickle cell disease. By the age of 5 years, children with this disease are effectively asplenic as a result of sickling-related infarction of the spleen. As a consequence, infections with encapsulated organisms normally cleared by the spleen can rapidly lead to sepsis in children with sickle cell disease. Vaccinations against *Haemophilus influenzae* and *Streptococcus pneumoniae* are the first line of defense. Children with sickle cell disease who develop fever should receive prompt medical attention. If this is not immediately available, they should be treated with an antibiotic, such as azithromycin. Children under the age of 5 years are at particularly high risk for sepsis. Penicillin prophylaxis (125 mg twice a day), particularly during travel, is recommended for children in this age group by the AAP.

Congenital Heart Disease and Pulmonary Hypertension

Traveling with children with cardiac or pulmonary problems can be a particular challenge. During flight, most airline cabins are pressurized to allow a fraction of inspired oxygen (FIO_2) of 0.17 or 0.18 as opposed to the typical FIO_2 of 0.21 at sea level. Depending on the nature and severity of the child's disease, this may require arranging for an oxygen tank and delivery apparatus during flight. Travel to high altitude should be cleared by the child's primary or specialty physician for the same reasons. Respiratory infections are also more common during travel. Because of the potentially significant impact of seemingly minor respiratory infections on children with cardiopulmonary disease, hand hygiene is particularly important to prevent inoculation with infected secretions via hand-to-nose and hand-to-eye contact. An emergency plan should be available for every country on the child's itinerary, including a specialist contact and a full description of the child's condition and health care needs.

PEDIATRIC WILDERNESS MEDICAL KITS

When a family travels in remote areas, any medical kit must be adapted to meet the special needs of the children. Actual items carried vary depending on the ages of the children, preexisting medical conditions, length of travel, specific environmental conditions likely to be encountered, and medical sophistication of the adults. Although there is considerable room for individual preference in assembling a medical kit, certain items are essential for management of problems commonly encountered during wilderness travel with children (Box 87-8).

Box 87-8. Pediatric Wilderness Medical Kit: Basic Supplies

- Identification and basic health information for child: past medical history, medications, allergies, blood type, weight
- First-aid supplies: adhesive bandages, gauze pads, gauze roll, tape, nonadherent dressings, moleskin/Spenco 2nd Skin/New-Skin, benzoin, alcohol wipes, povidone-iodine solution for dilution and disinfection, safety pins, tweezers, lightweight malleable splint (SAM splint), syringe (20 to 35 mL), and 18-gauge plastic catheter for wound irrigation
- Powdered oral rehydration solution (homemade or commercially available formula that meets the World Health Organization recommendations)
- Sunscreen: SPF 15 or greater
- Insect repellent: DEET <35%
- Whistle for child
- For infant <3 mo: rectal thermometer, bulb syringe

DEET, *N,N*-diethyl-meta-toluamide; SPF, sun protection factor.

To reduce weight and bulk, medications selected for a wilderness medical kit should have multiple uses (see Table 87-12). For example, diphenhydramine is effective for allergic symptoms, pruritus, motion sickness, nausea, and insomnia. Desitin, best known for its use in preventing diaper rash, is also an excellent sun block, as it contains 40% zinc oxide. A broad-spectrum antifungal cream, such as miconazole or clotrimazole, covers not only tineal infections (ringworm, jock itch, and athlete's foot) but also *Candida* infections (diaper rash and vaginitis). Medications in liquid form should generally be avoided because they add excess weight to the medical kit and can leak. If an infant under the age of 6 months is traveling, liquid medications can be carried in light- and leak-proof powder form. Most children are able to chew tablets once their first molars are present (by about 15 months). Before that time, chewable medications or tablets can be crushed between two spoons and mixed with food. If a child dislikes the taste of medication, it may be camouflaged in a food such as instant pudding, which is easily carried in a powdered form.

Painful musculoskeletal injuries are a potential complication of wilderness activities, so pain medication for children should be included in every medical kit. Acetaminophen not only relieves minor aches and pains but also is effective for fever control. It is well tolerated by most children and available in many pleasant-tasting forms for children unable to swallow pills: chewable 80- or 160-mg tabs, elixir (160 mg/5 mL), and concentrated drops (80 mg/0.8 mL). Ibuprofen is also effective for pain and fever control. Its duration of action is 6 to 8 hours, compared with 4 to 6 hours for acetaminophen. Ibuprofen is available in many forms: infant drops (40 mg/mL), children's elixir (100 mg/5 mL), 50- or 100-mg chewtabs, and 100- or 200-mg caplets. Tylenol with codeine combines a centrally acting agent, codeine, with a peripherally acting analgesic, acetaminophen. It is indicated for treatment of moderate to severe pain. Acetaminophen has the added effect of fever control,

whereas codeine has antitussive properties. Codeine can cause respiratory depression, particularly in children less than 12 months of age, and may elevate intracranial pressure, especially in the presence of head injury. Its use should be avoided in these situations. Dosing is based on the codeine component: both the liquid formulation (12 mg/5 mL) and tablets (10, 20, or 30 mg of codeine per tablet) are dosed at 0.5 to 1 mg/kg up to every 6 hours.

Two or three antibiotics will cover most bacterial infections encountered in children. Age of children traveling, allergies, intolerance, and past experience must be taken into account when antibiotics are selected. Given increasing bacterial resistance to common antibiotics, antimicrobial choices should be reevaluated periodically to ensure that infections likely to be encountered can be successfully treated with these agents. All oral antibiotics require a prescription. Amoxicillin is available in pleasant-tasting chewable tablets (125 and 250 mg) and in powdered form for suspension (125 and 250 mg/5 mL). Because of current resistant patterns, high-dose therapy with 80 mg/kg divided, three times daily is indicated. Amoxicillin is familiar to and well tolerated by most children. Amoxicillin-clavulanate (Augmentin) provides excellent coverage for recurrent pharyngitis, soft tissue infections, animal bites, and infections when a penicillin-resistant organism is suspected. The most common side effect is diarrhea, but this has been reduced with the newer formulations of Augmentin. The new formulation of this antibiotic is available in 200- and 400-mg (amoxicillin component) chewable tabs and in 200 and 400 mg/5 mL liquid. Dosing is 80 mg/kg divided, twice a day. Azithromycin (Zithromax) is a long-acting macrolide without the gastrointestinal side effects of erythromycin. It is effective in the treatment of otitis media, sinusitis, pharyngitis, lower respiratory infections, traveler's diarrhea, and common animal bites. It is particularly useful in children with penicillin allergies. Dosing is extremely convenient due to azithromycin's long half-life; a 10 mg/kg dose on day 1 is followed by 5 mg/kg/day for a total of 5 days. Ciprofloxacin (Cipro) has a broad spectrum of coverage and is useful for treatment of bacterial diarrhea, complex urinary tract infections, and wounds acquired in an aquatic environment. Its use has not been approved for children less than 18 years in the United States because of a potential problem with arthropathic effects on weight-bearing joints. However, a study involving more than 600 children found only a 1.3% rate of reversible arthralgia and no evidence of arthropathy.[73] Nonetheless, ciprofloxacin should not routinely be recommended for children unless a special situation arises in which the benefits of its use outweigh the risks.

Parents of children with special medical conditions should carry a pertinent medical summary with them and should have resources to access specialty physicians in the destination countries. A generous supply of necessary medications with instructions for caregivers and consulting physicians on what is to be done should symptoms worsen must be included in the medical kit. An extra supply of all essential medications should be kept with either the parents or the patient in case the medical kit gets lost or separated from the patient when the medication is needed. Some travelers in endemic areas for hepatitis B and acquired immunodeficiency syndrome (AIDS) carry their own needles and syringes for emergency use. The International Association for Medical Assistance to Travelers (www.iamat.org) has a directory of qualified physicians worldwide who speak English.

INFANTS AND NEONATES

A family traveling with an infant should be particularly vigilant about monitoring their child's state of health. Infants become hypothermic, hyperthermic, septic, and dehydrated more rapidly than adults or older children. A thermometer and appropriate lubricant should be included in the medical kit for monitoring rectal temperature. A rectal temperature above 38°C (100.4°F) in a child less than 3 months of age requires immediate medical evaluation. Digital thermometers are recommended, as they are less likely to break, are easy to read, and acquire a temperature reading three to four times faster than a glass thermometer. Some emit an audio alarm when the reading is ready.

Infants are less tolerant of problems with excess mucus. A bulb syringe is handy for suctioning mucus from the oropharynx and nasal passages. A few drops of saline solution ($^1/_4$ tsp of salt in 1 cup of water) instilled into the nares a few minutes before aspiration helps to loosen the mucus. Nasal aspiration should be reserved for times of most need, such as before feeding and sleep, as the procedure is irritating to a child. Other uses for a clean bulb syringe include flushing foreign bodies from ears and administering enemas.

Away from the conveniences of home, diapers tend to be changed less frequently. Consequently, diaper rash may become a problem. A good barrier cream containing zinc oxide may be helpful, and its use should be started at the first signs of irritation. If the rash progresses despite appropriate cleaning and barrier cream use, an antifungal cream, such as miconazole or clotrimazole, should be applied two to three times a day.

In very young infants, 3 to 12 weeks of age, colic can be a very challenging and vexing problem. A few hours of inconsolable crying a day is not uncommon in this age group but should prompt some investigation. If the infant is feeding well, is urinating and stooling regularly, and has neither fever nor a rash, the most likely etiology for this recurrent, seemingly inconsolable crying is colic. Colic is defined as a pattern of daily paroxysms of irritability and crying without a significant identifiable cause. The pathophysiology of colic is not clear but has been postulated as immaturity in the gastrointestinal or nervous system, a response to maternally ingested foods in breastfed babies, or a product of over- or understimulation of an infant. Colicky infants can often be soothed with rocking motions and close parental contact. Gentle abdominal massage can also be helpful. Breastfeeding mothers should consider eliminating caffeine, spicy foods, citrus, and dairy from their diets. Colic typically resolves by 3 to 4 months of age.

LOST CHILDREN AND SURVIVAL

Lost children in the wilderness is a common, preventable problem. Children should be taught to recognize landmarks and to turn around and look backward periodically to familiarize themselves with the terrain. Those who are capable of reading a compass and topographic map should carry these items at all times. Young children should wear brightly colored clothing to facilitate a search should they become lost (see Figure 87-5). They should carry a whistle around their necks and should be taught the universal signal for help: three blasts in a row. It should be emphasized to the child that the whistle is intended

for emergency use only. Portable two-way radios can also be used for emergency communication if a child is lost. Depending on the model, the range can extend 2 to 5 miles, but mountains and ridges can cause interference. As soon as they are able to carry a backpack, children should also be equipped with some survival items. A flashlight, a bottle of water, extra food, and a few brightly colored garbage bags can make all the difference for a child forced to spend several hours or a night out in the wilderness. Also, all children should either know or carry with them a piece of paper with their parent's name, address, and phone number. Older children who venture out without their parents should always tell an adult where they are going, with whom, and when they expect to return.

A few programs, such as Hug-a-Tree (www.gpsar.org/hugatree.html), instruct children in the basics of survival and orienteering when lost. The title of the program is intended to remind children of three important tenets of survival when lost: stay in one place to facilitate any search, take advantage of the natural shelter provided by a tree, and feel the security and calming effects of a large natural protector. By hugging a tree and not wandering around, children can work on making signals out of rocks or branches, thereby indicating their location. Children should be taught to avoid getting wet, to wear a hat, and to stuff pine needles or dry grasses into their clothes to insulate themselves if they are cold. Children can practice making temporary shelters out of logs, branches, and leaves and thereby experience the warmth and protection provided by these natural features. They should avoid lying directly on the ground and should use leaves and branches to insulate themselves from the cold earth or snow. Finally, children should be advised not to eat anything with which they are not familiar.

Older children and adolescents who plan on spending time in the wilderness should be encouraged to participate in a program that teaches basic survival and first aid skills. Several organizations such as Lifeschool in Bodega, California, (www.lifeschooladventures.org) and the National Outdoor Leadership School or NOLS (www.nols.edu) provide instruction for children. Lifeschool is intended for children ages 12 to 16 years, whereas NOLS provides more technical instruction in various forms of wilderness travel in addition to first aid and survival training.

HOMESICKNESS

Most children experience some degree of distress when faced with separation from home, particularly when they are not accompanied by a parent. Predisposing factors to the depression and anxiety referred to as homesickness include young age, little prior separation experience, high parental separation anxiety, great perceived distance from home, few initial positive experiences after separation, preexisting anxiety or depression, and little perceived control over the situation.[88] Parents should be encouraged to introduce short periods of separation from home and family, leading up to longer periods. They should discuss the exciting aspects of any future adventure and encourage active decision-making regarding activities and destination. Parents should also try to alleviate their own separation anxiety and ensure positive early post-separation experiences for their child.[73] The presence of familiar faces, such as friends or favorite playmates, can significantly reduce a child's feeling of homesickness. Activities that include a child's favorite games and meals will be remembered by the child as fun experiences in which he would like to reengage. Through careful planning and coordination, parents can do much to allay the fear and anxiety of children as they travel away from home.

ENVIRONMENTAL CONCERNS AND CHILD HEALTH

Human activities have affected our global environment in dramatic ways in recent decades. These changes have been driven primarily by increasing reliance on fossil fuels. The use of leaded gasoline has resulted in significant lead contamination of soil. Childhood exposure to lead can cause anemia, gastrointestinal problems, and neurologic damage. In recent years, a more insidious and far-reaching problem associated with the use of fossil fuels that has emerged is accumulation of carbon dioxide and methane. The accumulation of these greenhouse gases has resulted in some of the warmest average air and sea temperatures ever recorded.[32] These changes in temperature will very likely result in extremes of precipitation—floods and drought.

The implications of these climate changes on child health are several-fold. As described previously, children are more at risk than adults for development of dehydration and hyperthermia, particularly during exercise. Higher temperatures further increase this risk. The rates of sunburn in children and of subsequent melanoma are likely to rise. Furthermore, warmer temperatures typically result in higher levels of ozone and other air pollutants. These can significantly affect children with asthma and other respiratory diseases. Warmer winters and rising carbon dioxide levels may also result in earlier and more abundant release of grass pollen and other pollens,[9,32] which could increase the incidence and severity of atopic syndromes in children. Finally, higher temperatures, along with changes in humidity and flood frequency, are likely to result in significant increases in vector- and rodent-borne disease transmission. Recent modeling experiments suggest that the distribution of malaria, a disproportionate killer of children, is likely to increase by 5% to 7% in Africa by 2100.[32] The rate of dengue virus replication in the *A. aegypti* mosquito vector increases directly with temperature. Higher global temperatures might result in a higher infection rate, particularly in children who are less able to protect themselves from mosquitoes. Other tick-borne diseases may also become more prevalent as greater flooding expands the rodent population that is an integral part of the tick life cycle.

If for no other reason than preserving our children's health, it is incumbent on us to mitigate these effects of fossil fuel consumption. One approach is to improve our energy efficiency through the use of hybrid and fuel-cell technologies and to develop renewable energy sources, utilizing solar, wind, and geothermal power. Finally, by preserving our world's forests, we may limit greenhouse gas accumulation and thereby slow the progress of global warming. Physicians and other health care professionals can educate communities about the impacts of climate change on child health and emphasize the need for policies supportive of a clean, healthy environment.

SUGGESTED READINGS

Cary A: Parents' Guide to Hiking and Camping: A Trailside Guide. Hong Kong, New Media, 1997.

Euser B: Take 'Em Along: Sharing the Wilderness with Your Children. Evergreen, CO, Cordillera Press, 1989.
Foster L: Take a Hike! San Francisco, Sierra Club, 1991.
Hodgson M: Wilderness with Children. Harrisburg, PA, Stackpole Books, 1992.
Jeffrey N: Adventuring with Children: An Inspirational Guide to World Travel and the Outdoors. Ashland, MA, Avalon House, 2000.
Rockwell R: Hug a Tree and Other Things to Do in the Outdoors with Young Children. Beltsville, MD, Gryphon House, 1993.
Ross C, Gladfelter T: Kids in the Wild. Seattle, Mountaineers, 1995.
Yaron R: Super Baby Food. Archbald, PA, FJ Roberts, 1998.

The references for this chapter can be found on the accompanying DVD-ROM.

Women in the Wilderness

88

Susan Anderson

Travel not only stirs the blood . . . it also gives birth to the spirit.

Alexandra David-Neel, French explorer and writer (1868–1969), who walked 2000 miles across the Himalayas to enter Lhasa at the age of 55 in 1924

More women are traveling to remote areas of the world at all stages of life, from adolescence and pregnancy to their wild adventuresome 80s. Wilderness travel health issues vary according to the life stage and style of the woman. Situations range from the first trimester of pregnancy and determination to climb a 16,000 peak to an adolescent kayaking in the jungles of the Amazon to a 72-year-old researcher spending a year in Antarctica.

Over the centuries, women have strived to prove their equality with men in areas professional, intellectual, and physical. Wilderness experiences have the potential to increase a woman's self-esteem and empowerment. Physical activity has numerous health benefits for women, including stress reduction and prevention of osteoporosis, heart disease, breast cancer, and depression.[34,57] Women from around the world are reaching new "personal summits," from a Nepalese woman climbing Mount Everest to a series of climbing expeditions by breast cancer survivors. Clothing and recreational equipment designed specifically for women represent a multibillion-dollar industry.

Problems common and unique to women might diminish the wilderness experience or place them at risk. The framework for wilderness travel health recommendations for a particular woman revolve around considerations including age and life stage, background health history, level of fitness, itinerary, and activity plans. Physicians and other health care providers can assist women embarking on wilderness experiences by providing individualized prevention and treatment guidelines. Because wilderness travel may include travel to remote locations, special considerations for wilderness travel to developing countries are discussed.

GENDER-BASED RESEARCH

In 2001, the Institute of Medicine (IOM) of the National Academy of Sciences released the report, "Exploring the Biological Contributions to Human Health: Does Sex Matter?"[170] The IOM report was the first significant review of the biomedical research to date related to sex and gender differences. The report confirmed the importance of gender and recommended more funding for multidisciplinary research to address the issue of gender and health in all areas of medicine.

Women's health and gender-based research initiatives have been developed at many academic institutions and organizations around the world. The number of training programs and fellowships, along with journals, textbooks, and other related publications focused on women's health and gender medicine, has grown exponentially.[34,73,86,105,114,124]

Until recently, researchers paid little attention to gender issues in the field of wilderness medicine. The focus was predominately on the effects of extremes of environment on fertility and pregnancy outcome. Pregnant women who live at altitude experience a high rate of fetal intrauterine growth retardation. What does this mean for the pregnant short-term traveler to altitude? Certain infectious diseases carry special risks for women. They may run an accelerated or more severe course, especially during pregnancy. Infections that have a more severe course during pregnancy include malaria, typhoid, and amebiasis. Women of any age exposed to schistosomiasis through adventurous kayak-

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ing, canoeing, or rafting may become infected. Female genital schistosomiasis can lead to subtle inflammatory changes of the genital tract and a risk for future infertility.[43,46,50,85] Thus, for a woman, exposure to certain environmental extremes or pathogens at a particular stage of life may lead to severe long-term consequences.

There are many practical questions to be answered. What is the safety of recommended immunizations and medications for a specific itinerary in pregnancy? What is the risk for deep venous thrombosis in a breast cancer survivor taking tamoxifen who wants to trek over an 18,000-foot pass? Do antimicrobials prescribed for malaria chemoprophylaxis or for self-treatment of traveler's diarrhea interfere with the efficacy of oral contraceptive pills? Gender and life-stage issues are important when considering a woman's risks for wilderness and environmental exposure, preventive measures, treatment options, and possible long-term complications.

PRE–WILDERNESS TRAVEL WOMEN'S HEALTH ASSESSMENT

The key areas that should be addressed for any woman wilderness traveler are outlined in the Pre–Wilderness Travel Checklist for Women (Box 88-1).

The priority is to identify life-stage and medical issues that could be exacerbated by physical demands and the wilderness setting. Environmental and infectious disease risks should be assessed. These must be evaluated in relation to the length of the excursion, need for additional conditioning and acclimatization, and any difficulty in accessing sophisticated medical care.

For anticipated physically challenging conditions or prolonged stays, women should undergo a thorough medical assessment (Box 88-2).

After historical information is collected, a comprehensive physical examination is performed, with special attention to the cardiovascular and respiratory systems, musculoskeletal system, genitourinary tract, and breasts.

Laboratory screening, as indicated by age, medical status, and risk factors, include hemoglobin/hematocrit (Hgb/Hct), thyroid panel, urinalysis, urine culture, pregnancy test, Pap smear, and screening for sexually transmitted infections and hepatitis.

Other tests might include an electrocardiogram (ECG) with stress testing, pulmonary function tests, and a mammogram if over age 40 or with a strong family history of breast cancer. Bone densitometry should be considered in perimenopausal and postmenopausal women and in young women with a history of fractures or abnormal menses consistent with a hypoestrogenic state.

Recommendations for a medical kit should be made and adapted to life stage and health needs. Maintenance medication should provide optimal control of medical conditions and cover planned time, emergency needs, and expected dose adjustments. Contraceptive needs should be reviewed and options explored. Basic supplies for hygienic and anticipated therapeutic needs related to menses, the urinary tract, and vaginitis should be included. Suggestions are given in Table 88-1 and Box 88-3).

After medical evaluation, a final risk assessment with guidelines for prevention and treatment versus adaptation of the itinerary to decrease a particular risk should be discussed.

Box 88-1. Pre–Wilderness Travel Checklist for Women

- Current age or life stage
- Personal level of risk taking
- Itinerary
- Gender-specific recommendations related to:
  - Environmental risks
    - Altitude, heat, cold, water, etc.
    - Preventative measures
  - Infectious disease risk
    - Vaccine-preventable diseases
    - Chemoprophylaxis
    - Personal protective measures
  - Sports and exercise
    - Level of fitness
    - Equipment
- Practical issues
  - Genitourinary issues
    - Menstruation or dysfunctional uterine bleeding
    - Contraception and emergency contraception
    - Breast health
    - Vaginal health
    - Urinary tract issues
    - Sexually transmitted infection risk
  - Pregnancy
    - Planned or unplanned
    - Lactation
  - Perimenopausal and menopausal issues
- Medical issues
  - Past medical history
  - Long-term travel
    - Pap smear and mammogram
    - Copy of electrocardiogram if older than 50 years
- Psychological issues
- Cultural issues
- Personal safety issues
  - Self-defense training
  - Personal protection devices
- Wilderness medicine kit
- Emergency plans
  - Medical and evacuation insurance
  - Copy of medical data

WHAT MAKES WOMEN DIFFERENT?

Gender-Related Performance

Gender-specific differences in anatomy and physiology are mainly due to hormonal differences that vary across the lifespan. In general, women are smaller than men and have less lean muscle mass and 8% to 10% more fat mass for a given body size, differences attributed to increased estrogens in the female and increased androgens in the male. Females reach physiologic and skeletal maturity and achieve peak height before males. Women have a larger surface area–to–body mass ratio. Blood volume and hemoglobin, stroke volume, and cardiac output are all lower in women than in men. These and other factors contribute to lower maximal aerobic power (even for similar training status) in women. $\dot{V}O_2max$ is the gold standard as an index of car-

Box 88-2. Assessment of a Woman's Health: Screening Highlights

Menstrual history: age of menarche, regularity, characteristics, timing, extent of blood flow (length, amount), intermenstrual bleeding, perimenstrual symptoms (e.g., dysmenorrhea, headache, premenstrual syndrome), plans for managing periods (hygienic, therapeutic)
Sexual history: age of coitarche, number of partners, sexual orientation, sexually transmitted infections, contraceptive history, plans for sexual activity in the wilderness, dyspareunia
Gynecologic history: ovarian cysts, uterine fibroids, endometriosis, cervical dysplasia, surgical history, pelvic pain, vaginal discharge, vaginal infections and treatment, pregnancy and complications
Breast: galactorrhea, discharge, masses, surgery
Gastrointestinal system and urinary tract: ulcers, irritable bowel syndrome, gallbladder disease, constipation, urinary tract infections, kidney stones, stress incontinence, urgency incontinence
Musculoskeletal system and skin: injuries (exercise related and accidents), limitations, muscle cramps, joint pain and swelling, arthritis, rashes, acne, sun sensitivity, hirsutism, hair loss
Exposure to abuse: battering, sexual harassment, sexual assault
Habits: smoking, alcohol, illicit drug use
Current problems: condition, medications, status of control, complications
Immunizations: measles, mumps, rubella, polio, diphtheria, tetanus, hepatitis, others
Family history: thyroid disease, hypertension, autoimmune disorders, diabetes, breast and gynecologic malignancies, osteoporosis
Allergies: general, drug related, bite and sting sensitivity
Nutritional: eating disorders, weight changes, food sensitivities, dietary preference (e.g., vegetarianism), caloric intake, assessment of mineral and vitamin intake in diet, supplements (e.g., iron, vitamins, calcium), including homeopathic compounds

TABLE 88-1. Medications for Medical Kit for Women

INDICATION	MEDICATION*	DOSE
Dysmenorrhea	Ibuprofen	200 mg 1 to 4 tabs q 4 to 6 hr
Headache, pain, fever	Acetaminophen	325 to 500 mg
Nausea and vomiting	Phenergan (tablet or suppository)	25 mg q 4 to 6 h
Urinary tract infection	Nitrofurantoin	100 mg bid × 7 days
	Ciprofloxacin	250 to 500 mg bid × 3 days
	Trimethoprim-sulfamethoxazole	160 mg/800 mg bid × 3 days
Urinary analgesic	Pyridium	200 mg tid PRN
Pyelonephritis	Ciprofloxacin	500 mg bid × 14 days
Yeast vaginitis	Miconazole cream or suppository	One applicator HS × 3 to 7 days
	Fluconazole	150 mg single dose
Bacterial vaginosis	Metronidazole tablets	250 to 500 mg bid to tid
	Metronidazole vaginal gel; clindamycin vaginal cream	One applicator HS × 3 to 7 days
Menstrual regulation or breakthrough bleeding	Oral contraceptive pills	As directed
	Conjugated estrogen	2.5 mg qd
	Medroxyprogesterone acetate	5 to 10 mg qd
Nutritional supplements	Ferrous sulfate	300 mg qd to tid
	Calcium carbonate multivitamin	1250 mg qd

*Suggested medications or equivalent depending on tolerance, allergy history, and patient preferences.
qd, daily; bid, twice daily; tid, three times daily; qid, four times daily; HS, at bedtime; PRN, as needed.

diorespiratory fitness. Women of comparable training status usually have $\dot{V}O_2$max values that are 5% to 15% lower than those of men. Correction of $\dot{V}O_2$max values for lean body mass does not correct the discrepancy between genders completely, which may relate to lower hemoglobin concentrations in women, leading to less oxygen-carrying capacity.[37]

Estrogen and progesterone can influence ventilation and thermoregulation during exercise. Women have a smaller lung capacity than men. The smaller capacity of the lungs to oxygenate blood during intense exercise may limit exercise performance in women. Control of ventilation is influenced by progesterone, which increases central ventilatory drive, affecting breathing responsiveness during exercise and at altitude.[37]

Thermoregulatory control varies over the course of the menstrual cycle The thresholds at which cutaneous vasodilation and sweating are initiated are increased about +0.5° C (+1.8° F) in the luteal phase of the menstrual cycle, when progesterone and estrogen are elevated, as compared with the early follicular phase when these hormones are low. However, these subtle thermoregulatory differences across the menstrual cycle have not been found to affect exercise performance.[97] The physiologic mechanisms that affect adaptation and performance in women are subtle, and there is a substantial biologic variability among individuals.[6,37,59]

Box 88-3. Supplies for Medical Kit for Women

- Menstrual supplies
  - Calendar to keep track of menses
  - Supplies and devices
    - Pads, tampons, menstrual cups
    - Towelettes, plastic disposal bags, matches
    - Premenstrual symptom medications
    - Dysfunctional uterine bleeding medications
- Urinary voiding
  - Medication for urinary tract infections and burning
  - Toilet tissue, towelettes
  - Funnels—paper or plastic
  - Pads for incontinence
  - Urinary dipstick to screen for infection
- Vaginitis
  - Self-treatment medications
  - pH paper
  - Vaginal speculum and gloves if trained medical professional on expedition
- Contraception
  - Contraception option
  - Emergency contraception
  - Chart to keep track of pills, patches, or ring use
  - Timer—special wristwatch "alarm"
- Pregnancy tests
- Perimenopausal and menopausal issues
  - Stress incontinence—pads
  - Atrophic vaginitis—vaginal moisturizers and lubricants
- Pregnancy supplies
  - Blood pressure cuff
  - Urine protein and glucose strips to screen for diabetes and preeclampsia
  - Urine leukocyte esterase strips to check for urinary tract infection
- Supplies for lactation
  - Breast pump
  - Nipple cream
  - Self-treatment—mastitis
- Personal safety
  - Alarms
  - Pepper spray
  - Other self-defense measures

Gender-Related Issues Concerning Environmental Exposure

Women and Altitude

Studies have found no difference in the incidence of acute mountain sickness (AMS) between women and men.[74,109] In early studies, women seemed to have a lower incidence of pulmonary edema than men.[90,92,156] However, in retrospect, many of the epidemiologic studies on altitude show a predominance of male subjects; thus, these early studies may not reflect true gender predilection. Women have been found to have a higher incidence of peripheral edema than men.[75,139] An excellent review of the research to date on women and altitude was recently published.[94]

A difference in how men and women respond to altitude is thought to be hormonally mediated. Adaptation to high altitude involves a series of physiologic responses triggered by hypoxemia. The sigmoidal shape of the oxyhemoglobin dissociation curve prevents a drop in the oxygen saturation below 90% in healthy persons until an altitude of about 2400 m (8000 ft) is reached. At higher altitudes, hypoxia stimulates respiratory, cardiovascular, and hematologic changes that depend on both degree of hypoxia and time.

The earliest and probably most important response to altitude is increase in minute ventilation caused by increases in tidal volume and respiratory rate. This hypoxic ventilatory response (HVR) is mediated by chemoreceptors in the carotid bodies, which respond to a decrease in arterial oxygen pressure (Pa_{O_2}) and signal the respiratory center in the medulla to increase ventilation. HVR has been closely related to adequacy of acclimatization and the risk for developing AMS.[145] The degree of HVR to a given Pa_{O_2} varies among individuals and is genetically influenced. The HVR is inhibited by respiratory depressants, such as alcohol and sedatives, and stimulated by respiratory stimulants, such as caffeine and cocoa. Progesterone is a potent respiratory stimulant and acts primarily through activation of peripheral arterial chemoreceptors.

Progesterone is produced by all steroid-forming glands, including the ovaries, testes, adrenal cortex, corpus luteum, and placenta. Although progesterone has been found to increase hypoxic ventilatory response and ventilation at sea level, there is no difference between men and women in ventilatory response to high altitude.[125,126,160] Despite higher levels of progesterone in women, particularly in the luteal phase of the menstrual cycle when progesterone levels increase as much as 10-fold, no difference has been observed between males and nonpregnant females in the incidence of AMS.[74,109] The incidence of AMS is not affected significantly by menstrual phase.[23,142,175]

Most studies on pregnancy and altitude have been done with permanent residents of high altitude.[56,94] More studies are needed to evaluate the combined effects of altitude and exercise on pregnancy in the short-term sojourner. One study of pregnant sojourners at moderate altitude of less than 2500 m (<8200 ft) found the incidence of AMS during pregnancy did not differ from the nonpregnant state.[130] Studies of high-altitude residents indicate that pregnancy and altitude act together to increase ventilation. Arterial oxygen saturation in the pregnant woman is increased by the increase in ventilation.[120] Uterine artery blood flow may be lower at high altitude than at low altitude, which would suggest that oxygen delivery to the fetus might fall.[118,178] The effect of a reduction in uterine blood flow, decreased arterial oxygen content, and anemia in the mother suggest that fetal oxygen delivery would be compromised during exercise.[56] Some studies suggest a higher incidence of spontaneous abortion in the first trimester.[40,167] For women traveling to altitudes up to 2500 m (8200 ft) in the second half of pregnancy, there is little risk for fetal complications in those who otherwise have a normal pregnancy and do not smoke.[19,91] There are no data on safety at higher elevations. Increased incidence of preeclampsia, gestational hypertension, and placental abruption has been found in women who stay at higher altitudes for periods of weeks to months. Infants of women found to have preeclampsia were at increased risk for intrauterine growth retardation (IUGR).[101,118,119,174,177] Exercise has the potential to cause fetal hypoxia or preterm labor at high altitude if competition occurs for blood supply between maternal skeletal muscles and the uteroplacental circulation.[94] Recommendations and guidelines for pregnant women traveling to altitude are discussed in the pregnancy section.

Hot and Cold Environments

Because of lower sweat rate, women rely more heavily than men on dimensional characteristics and circulatory mechanisms for heat dissipation in hot environments.[148] Females adapt more easily than males to hot, wet environments because of lower sweat rate and decreased risk for dehydration.[14] In hot, dry environments, however, a higher sweat rate is advantageous because perspiration decreases the risk for hyperthermia, provided adequate fluid replacement is available. With acclimation to dry heat, women are able to increase the sudorific response to an extent equal to that of men. With similar acclimatization and physical training, women tolerate physical activity in hot environments as well as men. Women planning wilderness expeditions to hot, dry destinations are advised to plan a 1- to 2-week acclimation period in a hot environment.[16] In general, heat tolerance depends more on cardiovascular fitness than on gender.[82]

In moderately cold environments, women on average adapt better than men, partly because of thicker subcutaneous fat. At extreme cold temperatures, however, women may be at a disadvantage because of the decrease in insulation from lower muscle mass. Although adaptation to cold environments depends more on body size, physical fitness, and degree of acclimation than on gender, exercise performed during cold exposure may be more effective in maintaining body heat in women than in men.[111] Raynaud's syndrome is more common in women. Vasospasm precipitated by a cold environment results in numb or painful extremities, usually the digits. Rewarming the affected area is therapeutic. Preventive measures, such as wearing gloves and using hand warmers and boot warmers before exposure, is helpful. Treatment with a calcium channel blocker has been used in extreme circumstances.[28]

Gender-Related Issues Concerning Infectious Disease

Wilderness travel may increase exposure to infectious diseases. There are some differences in pathology when anatomic differences or physiologic differences (e.g., pregnancy) are taken into account.[102,155,163,173]

For example, immunity is compromised during pregnancy, making a woman more likely to become infected with malaria. Malaria during pregnancy is an important cause of maternal mortality, spontaneous abortion, and stillbirth. Most of the studies on malaria during pregnancy have been done on pregnant women living in endemic areas. These studies have demonstrated that women living in endemic areas have an increased susceptibility to *Plasmodium falciparum* infections during pregnancy compared with women who are not pregnant. Increased susceptibility to malaria during pregnancy may be due to sequestration of parasites in the placenta or suppression of selected components of the immune system associated with increased production of several hormones and other proteins.[49,53,60,127]

Female genital schistosomiasis has been associated with a wide range of pathobiologic manifestations, such as infertility, miscarriage, and preterm delivery. Women may have lesions of the vulva, vagina, cervix, uterus, and fallopian tubes.[57,98,136,149] The risk for schistosomiasis in women adventure travelers and expatriates living in endemic areas is considerable.[46,72,89,98] The Centers for Disease Control and Prevention (CDC) investigated the incidence of schistosomiasis among expatriates in Malawi. Of 917 persons serologically tested, 302 (33%) had schistosomal antibody; of these, 292 (93%) had antibody to *Schistosoma hematobium*.[36] Physicians may be confronted with parasitic infections causing gynecologic pathology not only in women originating from endemic countries but also in women who travel to exotic and subtropical countries for work or wilderness expeditions.[46,68] Women should be educated about risk and preventive measures.[43]

Hepatitis E infection acquired during pregnancy has a mortality rate of 15% to 25%.[35,104,146] Hepatitis E is a major cause of hepatitis outbreaks in India, Nepal, China, Pakistan, Africa, and the former Soviet Union and has been reported from Central America and Southeast Asia. Transmission of the virus occurs through fecal–oral exposure. Most outbreaks result from fecal contamination of drinking water. In nonpregnant women, severe disease occurs in less than 1% of individuals. However, 20% to 30% of pregnant women may experience fulminant disease. The causes of increased severity during pregnancy are not known. Hepatitis E infection acquired during the third trimester is also associated with fetal morbidity and mortality. A vaccine was tested in clinical trials.[150] Ideally, pregnant women should avoid travel to areas with a high rate of hepatitis E. Passive immunization with immune globulin is not effective in preventing hepatitis E infection.

Wilderness and adventure travel can increase a woman's risk of acquiring a particular infectious disease. Understanding the most common infectious diseases acquired during travel, geographic location, mode of transmission, clinical manifestations, diagnosis, how the infection might affect a pregnancy or lead to changes in the female genital tract, and other gender-related issues is important to advise women on travel risk issues, especially for the long-term or adventure traveler to endemic and remote areas.

PRACTICAL ISSUES FOR WOMEN DURING WILDERNESS TRAVEL

Women between the ages of 10 and 50 years menstruate on average once a month, with a high degree of individual variability. Women should be prepared to experience changes in their cycle and premenstrual symptoms during wilderness travel. Changes in exercise, diet, sleep patterns, and time zones can affect the hypothalamic-pituitary axis and cause menses to become irregular or cease. It is helpful for women on long-term expeditions to chart their cycles. Guidelines can be followed in cases of amenorrhea or dysfunctional uterine bleeding.[7] Reproductive-age women who are sexually active with men should carry pregnancy tests to rule out pregnancy if they miss their menstrual period. The options for feminine hygiene products that might be the best for a particular trip should be considered. Environmental considerations, such as the weight or bulk of the products and their disposal, should be considered. A number of alternative menstrual products are available (Fig. 88-1). One example is a soft reusable rubber cup (the Keeper) that is reported to last 10 years. Another option is a menstrual cup (Instead), which is sold for single use and can easily be washed and reused. Feminine hygiene products can also be used for wilderness first aid. For example, tampons may be used to stop nose bleeds or as ear plugs, and menstrual pads may be used as compression dressings for wound management (Fig. 88-2).

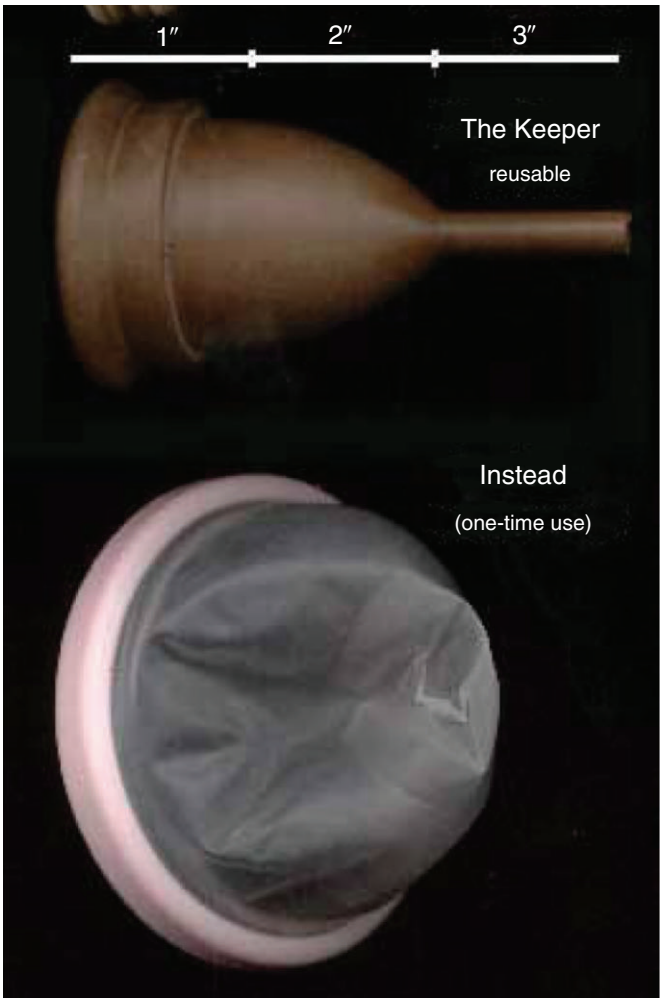

Figure 88-1. Options for feminine hygiene. (Photograph courtesy of Harry Finley, http://www.mum.org/MenCups.htm.)

Figure 88-2. Multiple uses of feminine hygiene supplies in the wilderness. (Photograph by Susan Anderson.)

Dysmenorrhea

Women may experience the worst premenstrual symptoms of their lives while traveling, even if they have never experienced symptoms before. Medications for self-treatment of these symptoms, such as ibuprofen, should be included in the medical kit.

Controlling the Menstrual Cycle

Some women prefer not to have a menstrual period during long-distance trekking, bicycling, or other endeavors. Women have the option of controlling their menstrual cycles by taking oral contraceptives or using a contraceptive patch or ring without a hormone-free interval. This option decreases the need for menstrual supplies and reduces premenstrual symptoms. "Tri-cycling" involves taking three to four cycles continuously without the pill-free days. Women should be warned that a small amount of breakthrough bleeding may occur because the endometrium may not remain stable without the physiologic curettage of the pill-free interval.[161]

Another option to decrease menstrual bleeding is to try a method of contraception that leads to thinning of the endometrium over time and amenorrhea in many women, such as progestin-only pills, Depo-Provera, or the progesterone intrauterine device (IUD) (Table 88-2). However, in some women, progestin-only contraception may be less predictable for cycle control and lead instead to irregular bleeding or spotting between menses. If a progestin-only method is chosen, it is recommended that it be started at least 3 to 6 months in advance of the trip in order to evaluate the effect on the menstrual cycle.

Menstrual Cycle Disturbance

Menstrual cycle disturbance can occur at any age and is one of the most frequent reasons a woman seeks medical care.[7,88] Among highly trained athletes and in environments that impose extreme physical and psychological demands, such disturbances can be found in greater than 50% of women not taking oral contraceptives (OCs). Menstrual problem during long-distance hiking was found to the most common complaint of women backpackers. A study of backpackers on the Appalachian trail found that 87% of normally menstruating women had menstrual abnormities while backpacking.[27] Menstrual cycle complaints are also common at high altitude. Mountain ascent, in

TABLE 88-2. Contraceptive Choices

METHOD	MECHANISM	ADVANTAGES/DISADVANTAGES	WILDERNESS TRAVEL ISSUES
Vaginal Barrier Methods			
Spermicides			
Creams, jellies, foams, melting suppositories, sponges, foaming tablets, films	Surface active agents that damage the cell membranes of sperm, bacteria, and viruses	Chronic exposure may cause mucosal injury that increases risk for human immunodeficiency virus (HIV) transmission	Available over the counter Store in location that is cool and dark Bring own supplies Female controlled
Diaphragm	Domed-shaped rubber cup Must use with spermicide Protection for 6 hr	Requires clinician fitting Insert extra spermicidal with repeated intercourse After use leave in for 6 hr May increase risk of urinary tract infection (UTI)	Carry in climate-resistant case Spermicide may not be available in developing countries
Cap	Mechanical barrier Requires spermicide	Needs clinician fitting Provides protection for up to 48 hr Small risk for toxic shock syndrome (TSS) Small risk for cervical dysplasia	Easy to carry Rubber may deteriorate in heat and humidity
Sponge (e.g., Today Sponge)	Polyurethane sponge containing nonoxynol-9 continuously released into vagina over 24 hr Sponge traps and absorbs semen before sperm reach cervix Leave in place for 6 hr after intercourse	One size Over the counter Moisten with water before use and insert Loop for removal Do not wear longer than 30 hr due to rare risk for TSS	Easy to carry, use Use bottled water for moistening in countries with questionable water supply Protects for 24 hr no matter how many times intercourse occurs
Condoms			
Male	Latex	Possible allergy Do not use oil-based lubricants May be stored in wallet up to 1 mo if kept away from heat and sunlight	Quality varies from country to country Bring supply from U.S. of latex and polyurethane types
	Polyurethane	Thinner, stronger, more resistant to deterioration Can use oil-based lubricants	Latex may break down in heat and humidity. Carry in special case. Estimated to last 1 mo in wallet
	Lambskin, natural	Small pores permit passages of viruses—hepatitis B, herpes simplex virus, HIV, so use only for contraception Brands and materials differ in quality	Use emergency contraception if condom breaks or slips and no backup method is in place (e.g., oral contraceptives, diaphragm, sponge) Store in cool, dry place Check expiration date
Female (Reality)	Polyurethane pouch Spermicide not required One use only	Can be inserted 8 hr before intercourse	Female controlled Does not deteriorate in heat and humidity Bring supply from U.S.
Hormonal Methods			
Oral contraceptives (OCs) Combined pill Estrogen Progesterone Many different types	Inhibition of ovulation by blocking luteinizing hormone (LH) surge Thickens cervical mucus to prevent sperm penetration to upper genital tract	Increased menstrual cycle regularity Blood loss reduced by 50% Cramping reduced by >60% Fewer ovarian cysts Fewer fibroids Less endometriosis Reduce ectopic pregnancy by 90%	Convenient, effective, easy to carry Need to take every 24 hr May use to delay menses by starting next package of active pills after 3 wk of previous package

Continued

TABLE 88-2. Contraceptive Choices—cont'd

METHOD	MECHANISM	ADVANTAGES/DISADVANTAGES	WILDERNESS TRAVEL ISSUES
Monophasic Triphasic New lower-dose forms 3-mo packaging option	Inhibits capacitation of sperm, which limits ability of sperm to fertilize egg Slows tubal motility, delaying sperm transport	If nausea and vomiting are present, need to use backup method or consider placing pill in vagina for absorption or switch to patch or ring May use as emergency contraception—check instructions May use to treat dysfunctional uterine bleeding Slight increased risk for DVT and stroke, especially in smokers	Consider drug interactions Rare risk for deep vein thrombosis (DVT) Bring supply for trip Research availability of OC or other method to use if OC lost or stolen See IPPF Guide to Hormonal Contraception for availability worldwide
Transdermal patches (Ortho Evra)	Same as above Matchbook-sized device placed on skin Designed to deliver 1 wk of hormone, used 3 wk on, 1 wk off	Same as above Better compliance Place in nonexposed area of skin	Better compliance during travel owing to weekly dosing Not dependent on gastrointestinal absorption, so is a good method in women with symptoms of nausea and vomiting
Intravaginal Rings NuvaRing: soft, flexible, transparent vaginal ring Daily hormone EE 15 µg Etonogestrel, 120 µg	Absorption of hormones across the vaginal wall Similar mechanism to OC Ring inserted for 3 wks Removed for 1 wk Insert new ring at 4 wk	Better compliance Less breakthrough bleeding than other hormonal options as steady release of hormone SE vaginal discharge, irritation,	Better compliance during travel, as lasts 3 wk Not dependent on GI absorption, so is a good method in women with nausea and vomiting
Progestin Only Pills	Inhibition of ovulation (may occasionally ovulate) Thickened and suppressed cervical mucus Suppression of midcycle LH and follicle-stimulating hormone (FSH)	Use if cannot take estrogen Take same type of pill every day—no pill-free week Decreased menstrual cramps, less bleeding Can use when breastfeeding Older women, smokers	Need to be prepared for irregular bleeding MUST take pill at same time every day—set alarm watch to help with time zone changes Must use additional method to protection against sexually transmitted infections (STIs)
Injectable Depo-Provera Depo medroxyprogesterone acetate (DMPA)	Depo-Provera 150 mg dose administered intramuscularly every 3 mo Blocks midcycle LH surge and inhibits ovulation	Increased compliance No interaction with antibiotics Side effects: weight gain, irregular bleeding, acne mood changes, decreased libido, osteoporosis Option if cannot take estrogen	Good compliance if travel <3 mo May be difficult to get if traveling in some areas Prepare for irregular bleeding
Intrauterine Device (IUD) Two types approved for use in U.S. Other types worldwide	Inhibition of sperm migration, fertilization, and ovum transport Creates spermicidal environment by provoking a sterile inflammatory reaction that is toxic to sperm and implantation	Option for nulligravid and multiparous women at low risk for STIs who desire long-term contraception Small risk for IUD-induced infection at insertion and if more than one partner Option for women with history of diabetes, history of thromboembolism, or who are breastfeeding	Long-term contraception (5–10 yr) Need fewer supplies for contraception Need to know how to check for string Need backup method if IUD falls out
Copper T 380A Replace every 10 yr	T-shaped polyurethane frame holding 380 mg of exposed surface of copper	Increased menstrual bleeding and cramping many occur in the first few months after insertion, which is usually relieved with nonsteroidal anti-inflammatory drugs	Need to protect against STIs
Levonorgestrel intrauterine system (LNG-IUS) Replace every 5 yr	T-shaped with a polydimethylsiloxane sleeve containing 52 mg of levonorgestrel Releases 20 µg of levonorgestrel per day	Less bleeding and cramping due to the effect of levonorgestrel	Need to get medical evaluation for missing string, excessive vaginal bleeding, abdominal pain, vaginal discharge, pregnancy

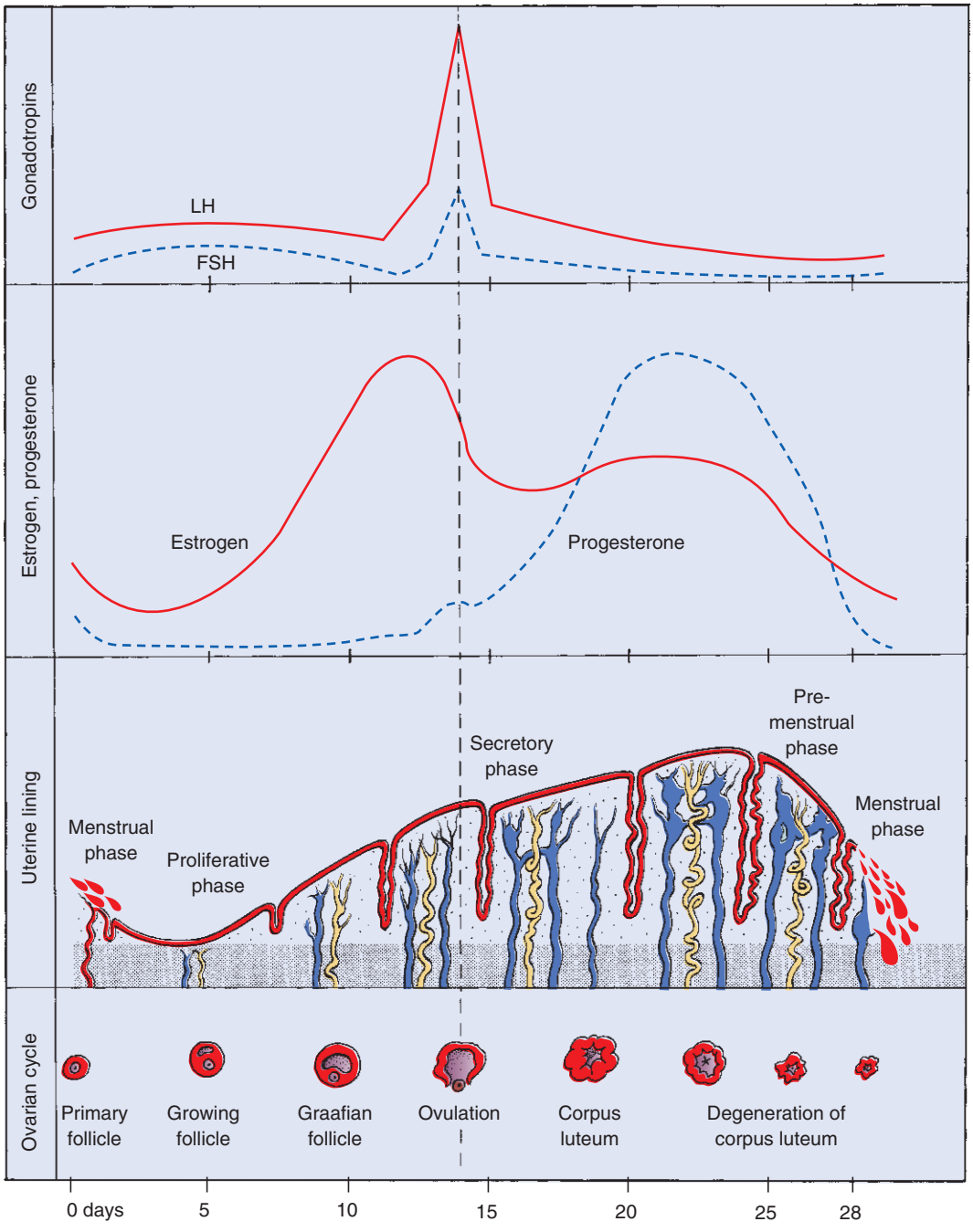

Figure 88-3. Normal human ovarian and endometrial (menstrual) cycle. FSH, follicle-stimulating hormone; LH, luteinizing hormone. (Modified from Shaw ST Jr, Roche PC: Menstruation. In Finn CA (ed): Oxford Reviews of Reproduction and Endocrinology, vol 2. Oxford, UK, Oxford University Press, 1980.)

combination with other stressors, such as time change, cold, and weight loss, is likely to modify the menstrual cycle.[44]

Understanding menstrual cycle abnormalities requires a basic understanding of the normal menstrual cycle (Fig. 88-3).

In premenopausal women, the hormones progesterone and estrogen fluctuate. Normal cycles occur at regular intervals, require ovulation, and typically average 28 days (range, 21–35 days). By convention, the first day of the menstrual cycle corresponds to the first day of the menstrual period, which usually lasts about 4 days (range, 3–7 days). The onset of menstrual bleeding represents desquamation of the functional endometrium, resulting from progesterone withdrawal in the absence

of conception during the previous cycle, and coincides with recruitment of a new group of primordial follicles for maturation. The first stage of the menstrual cycle, up to the time of ovulation, is referred to as the *follicular phase,* or *endometrial proliferative phase*. In the early follicular phase, estrogen and progesterone levels are low. In the late follicular (also called the *preovulatory*) phase (days 8–11), there is a brief surge in estrogen levels, which promotes ovulation. The second stage is the *luteal phase,* or *endometrial secretory phase*. After ovulation, there is an increase in the levels of estrogen and progesterone, which peak at about days 19 to 21 and then decrease until the following menstrual phase. During the menstrual cycle, pulsatile release of gonadotropin-releasing hormone (GnRH) by the hypothalamus occurs hourly during the follicular phase and every 1.5 hours during the luteal phase. Factors that disrupt the normal pulsatile release of GnRH or reduce the ability of the pituitary to respond to it are the major contributors to menstrual cycle abnormalities in premenopausal women.[29] In women who use OCs, contraceptive patch, or contraceptive ring, the hormone levels are elevated when the hormone (active) pills (or patch or ring) are taken (normally 3 weeks) and decrease during the week of placebo (no pill or no patch or no ring) interval. After menopause, the levels of circulating estrogen and progesterone decrease substantially; estrogen replacement (or combined estrogen and progestin) increases the levels of these hormones.

Causes of Abnormal Uterine Bleeding

Understanding the physiology of the normal menstrual cycle and differential diagnosis of dysfunctional uterine bleeding at each phase of a woman's reproductive life will help to diagnosis and manage the problem.

Abnormal uterine bleeding (AUB) can be loosely defined as any aberration in the normal menstrual bleeding pattern in premenopausal women and as any bleeding episode in postmenopausal women. The most common causes of AUB are problems related to pregnancy and hormonal contraceptive therapy. Initial evaluation of AUB in any premenopausal woman includes a urine or serum screen for human chorionic gonadotropin (hCG), regardless of contraceptive history. Non–pregnancy-related causes of abnormal bleeding can be divided into episodes in ovulatory women, in anovulatory women, and from extrauterine causes, either ovulatory or anovulatory.

Historical information is useful in developing a presumptive etiology and empiric therapeutic approach for AUB, particularly in the absence of laboratory resources. Past history includes age at onset of menses, regularity of menses, usual length of menses and blood flow, perimenstrual symptoms, medical problems, pregnancy history, sexually transmitted infection (STI) history, surgical history, and basic endocrinologic review of systems.[88] The current problem is then characterized by changes in the frequency of bleeding, amount of flow, new symptoms, relationship to activities, recent sexual history, systemic symptoms, weight change, change in medications or use of other health-related products, and change in exercise patterns.

Ovulatory Women

When the premenopausal woman who has consistently had regular cyclic menses presents with a history of progressively increasing amount and duration of menstrual flow, the most common causes are uterine fibroids, particularly submucosal (just beneath the endometrium) fibroids, endometriosis, and adenomyosis (endometriosis of the uterine muscle wall). These conditions are frequently associated with progressive dysmenorrhea and are more likely to occur with advancing age. Use of certain medications (e.g., estrogen, tamoxifen, warfarin, nonsteroidal anti-inflammatory drug [NSAID]) or homeopathic compounds should always be part of the evaluation of AUB. Abrupt change in duration or amount of blood flow should suggest a corpus luteal cyst, often associated with unilateral lower abdominal discomfort, or acute pelvic inflammatory disease (PID), often accompanied by more diffuse abdominal pain and systemic symptoms. Hyperplasia of the endometrium is unusual in ovulating women, and malignancies of the uterus or ovaries (estrogen secreting) are rare.

Perimenopausal women who are still ovulatory may have progressively more frequent menses that may be accompanied by changes in flow and duration, with or without the underlying causes noted above.

Intermenstrual or postcoital bleeding should also be evaluated for cervicovaginal infections, such as bacterial vaginosis (BV), yeast, trichomoniasis, and condylomas. Endocervical polyps, cervical dysplasia, and even invasive cervical carcinoma can occur in this group. When precipitated by coitus, profuse bleeding and dyspareunia can result. Acute pain with coitus and not usually accompanied by bleeding is more likely to be the result of PID, rupture of an ovarian cyst, or adnexal torsion.

Anovulatory Women

Anovulatory AUB is more common than ovulatory abnormalities among women who partake in wilderness experiences. Anovulatory bleeding problems can be divided into those occurring with adequate estrogen production and those accompanied by low estrogen production. Women with adequate estrogen stores are often characterized by hypersecretion of gonadotropins and frequent heavy bleeding. An example is women with polycystic ovary syndrome (PCOS). Oral contraceptives are helpful in management to stabilize the endometrium and synchronize and decrease menstrual flow.

In contrast, anovulation can be caused by inadequate release of gonadotropins from the pituitary, which leads to decreased estrogen production and presents as amenorrhea. Amenorrhea can be a symptom of the female athletic triad,[93,100,107] which includes disordered eating, amenorrhea, and osteoporosis. More commonly, anovulatory cycles and amenorrhea result from changes in a woman's usual routine of sleep, diet, and exercise patterns, all of which are extremely common to a wilderness experience.

The main goal when evaluating vaginal bleeding in the wilderness is to differentiate a true emergency, such as ectopic pregnancy or miscarriage, from a more benign cause that could be stabilized and then further evaluated by a woman's personal physician at home. If the pregnancy test is positive, immediate medical evacuation should be arranged.

If the pregnancy test is negative and there is no other evidence of an anatomic abnormality or systemic illness, vaginal bleeding may be a form of dysfunctional uterine bleeding (DUB). The following measures could be tried to stop the bleeding until further evaluation can be obtained.

For women without contraindications to estrogen or progestin, hormonal therapy with either estrogen or progestin or a combined hormonal preparation may be used to stabilize the endometrial lining. One option is to take an OC pill containing

30 to 35 µg of ethinyl estradiol every 4 to 6 hours until bleeding is under control, then to continue taking 1 tablet a day until the 21 active pills are finished. A few days later, synchronized withdrawal bleeding will occur. A woman may opt to continue taking one OC pill a day to prevent the withdrawal bleeding until evaluation can be obtained. Although the mechanism of action of a bolus of estrogen and progesterone is not well understood, its effect is thought to be mediated by accelerated proliferation of the endometrial basal layer, which seals the bleeding vessels. OC may be used as a temporizing measure for treatment of DUB until a more thorough evaluation is can be obtained.

In perimenopausal women with irregular bleeding, a low-dose OC containing 20 µg of ethinyl estradiol may be tried. Advantages include predictable cycle control for the trip, reliable contraception, decrease in perimenopausal symptoms (such as mood swings, irritability, decreased libido, and hot flashes), and possibly the prevention of accelerated bone and mineral loss that is believed to start during the perimenopausal years.

Menopause is associated with amenorrhea as a result of cessation of ovarian estrogen production and inactive atrophic endometrium. Occasionally, a menopausal woman may produce sufficient estrogen from peripheral conversion of ovarian and adrenal androgens to stimulate the lining and cause resumption of bleeding. Postmenopausal bleeding can also be due to atrophy. Although postmenopausal bleeding is usually mild and does not need emergent treatment, a medical evaluation is important to rule out more serious causes, such as underlying malignancy.[7]

Self-Diagnostic Pregnancy Tests

Pregnancy tests are helpful to evaluate amenorrhea, vaginal bleeding, pelvic pain, and other symptoms that may be related to a pregnancy. For women who are sexually active with men, pregnancy tests should be carried in the medical kit. Urine pregnancy kits are not always available in remote areas, and the reliability of kits in some countries is not known. Pregnancy test kits have limited shelf life, so the expiration date should be checked. Environmental extremes can affect accuracy of the test, so the manufacturer's recommendations regarding storage should be observed. As with most self-diagnostic tests, it is important to review the test procedure with the woman before travel.[41,137]

Contraceptive Options during Wilderness Travel

Contraceptive advice is an important area to address for all women wilderness travelers of reproductive age (see Table 88-2). The number of women that experience unplanned pregnancy remains high. The range of contraceptive options has increased during the past 10 years with introduction of lower-dose oral contraceptive pills, several new oral progestins, progestin-only implants, and a progestin-bearing IUD. New delivery methods, such as the contraceptive patch and vaginal ring, offer great new options for wilderness travelers. These options do not require daily compliance with taking a pill at the same time every day, which might be difficult to remember during an expedition. The other main advantage is that their absorption is not affected by a gastrointestinal (GI) illness such as nausea, vomiting, or diarrhea. Contraceptive efficacy is improved with use of the contraceptive patch or ring and there is less risk for irregular bleeding due to fluctuating hormone levels.[87,96,135]

TABLE 88-3. Contraceptive Method Efficacy in the United States

	PERCENTAGE OF WOMEN EXPERIENCING AN UNINTENDED PREGNANCY WITHIN THE FIRST YEAR OF USE	
METHOD	TYPICAL USE	PERFECT USE
No method	85	85
Barrier		
Spermicide	29	18
Diaphragm	16	6
Cap	32 (Parous) 16 (Nulliparous)	26 (Parous) 9 (Nulliparous)
Sponge	32 (Parous) 16 (Nulliparous)	20 (Parous) 9 (Nulliparous)
Condom		
Male	15	2
Female (Reality)	21	5
Hormonal		
Combined pill	8	0.3
Evra patch	8	0.3
NuvaRing	8	0.3
Progestin pill	8	0.3
Depo-Provera	3	0.3
Intrauterine device (IUD)		
ParaGard (copper-T)	0.8	0.6
Mirena (LNG-IU)	0.1	0.1
Female sterilization	0.5	0.5
Male sterilization	0.15	0.15

The best contraceptive method for a particular woman depends on her planned activities and itinerary. Considerations when choosing a method include accessibility and ease of use, weight and bulk of method, effects of environment such as extreme heat or cold, immersion in water on durability of the method, and known reliability of the method under perfect use (and under imperfect use)[80] (Table 88-3).

Wilderness medicine providers should have access to reference books and Internet sites on contraception because this is a rapidly changing field.[70,80] Relevant sites include the following:

American College of Obstetrics and Gynecology: www.acog.org
International Planned Parenthood: www.ippf.org
Princeton Emergency Contraception site: http://ec.princeton.edu

If a woman is already using a contraceptive method, the method should be evaluated for its ease of use and reliability, along with any special recommendations concerning its use during travel. If a woman wishes to try a new method, she should begin several months before travel, especially if she is planning to be overseas on a long-term assignment or in a remote area. Backup methods should be discussed. The International Planned Parenthood Federation maintains a worldwide guide to contraceptives and a list of family planning clinics. The Princeton Emergency Contraception site lists emergency contraception (EC) options available worldwide.

Barrier Contraceptives

The most common barrier contraceptives used during travel include the diaphragm, cervical cap, sponge, vaginal spermicide, and male condom.[80] Barrier contraceptive methods have the widest safety profile, excluding latex allergy and method failure resulting in pregnancy, but all have the same disadvantages for regular use in a wilderness setting. Failure rates even under ideal use with a spermicidal foam or gel are relatively high, estimated at 20% to 30% with "typical use" and 6% to 20% with "perfect use." The compounds in barrier devices are variably susceptible to extremes of heat or cold, which may compromise their tensile strength and effectiveness. Some couples find these methods inconvenient and messy, which might discourage compliance in an environment not conducive to cleanup. Bulk and weight complicate transport of sufficient spermicidal compound. Barrier methods decrease risk for gonorrhea, chlamydial infection, and human immunodeficiency virus (HIV) transmission, but offer minimal protection against human papillomavirus (HPV) or genital herpes simplex virus (HSV).

Hormonal Contraceptives

Combination (estrogen and progestin) OCs, either monophasic or multiphasic, offer the most reliable, convenient, cost-effective, and sensible risk-to-benefit ratio for the healthy reproductive-age woman. Brand selection should be done with a qualified health care professional. Previous experience of use, complications of therapy, menstrual history, and skin type should be considered. Failure rates with optimal use are extremely low (1%) but may be worsened by difficulty with compliance or GI illness during travel. Common side effects include nausea, vomiting, weight gain, and breakthrough bleeding. In most patients, breakthrough bleeding decreases with consistent use, and there is no need to change the brand of OC as a result of this effect.

Potential benefits of OCs include normalization of cycles, fewer midcycle ovulatory (i.e., mittelschmerz) and perimenstrual (e.g., dysmenorrhea, headaches) symptoms, lighter menstrual flow, suppression of ovarian cyst formation, reduced risk for endometrial cancer, prevention of osteoporosis, and increased bone mass. Condoms must be used to prevent STIs.

Major contraindications include history of thromboembolic disease, certain autoimmune disorders with thromboembolic risk factors, uncontrolled hypertension, hepatic dysfunction, diabetes with complications, and cigarette smoking.[61,122]

Transdermal Hormonal Contraception

The U.S. Food and Drug Administration (FDA) approved the first transdermal contraceptive patch in 2001. The contraceptive patch is a matchbook-sized device that is placed on the skin (abdomen, upper outer arm, buttocks, or upper torso; not the breasts). It consists of a three-layer matrix with an outer polyester protective layer and a medicated adhesive layer that contains the contraceptive steroids. A clear, polyester release liner, which protects the adhesive layer, is removed before application. The patch results in daily hormone release of 22 µg of ethinyl estradiol (EE) and 150 µg of norelgestromin. The hormones are absorbed into the blood and reach a steady state in 2 days. Each patch lasts 7 days. The patch is started on day 1 of menses and replaced weekly for 3 weeks. Week 4 is patch free. Each patch should be placed at a different site.[20,32,63,87,95,96,129] The efficacy is similar with various application sites. The contraceptive efficacy is not thought to be affected by humid climates, vigorous exercise, exposure to saunas, or water baths. The patch has similar side effects to OCs with the addition of occasional mild to moderate application-site reactions and an increased incidence of breast tenderness during the first few months of use. Most women have found the patch to be more convenient then a daily pill when traveling. There are no hormonal peaks and troughs. It is easy to check to make sure it is in place. Taking oral antibiotics is not believed to interfere with efficacy.[2,3] The FDA has issued a warning about Internet sales of counterfeit contraceptive patches.[164] To discard a used patch, it should be folded in half and discarded in solid waste garbage, not flushed down a toilet.

Vaginal Ring

NuvaRing is a soft, flexible, and transparent ring made of an ethylene vinyl acetate copolymer. It has an outer diameter of 54 mm and a cross section of 44 mm. There are two steroid reservoir cores in the ring that provide daily hormone release of 15 µg of EE and 120 µg of etonogestrel (an active metabolite of desogestrel).[96] NuvaRing provides hormone bioavailability comparable with an OC, such as desogestrel ethinyl estradiol (Ortho-Cept). It is inserted by a woman and removed after 3 weeks. Following 1 "ring-free" week, a new ring is inserted. If the ring is expelled accidentally, it can be washed and reinserted. If the ring is out for more than 3 hours, a backup contraceptive method is recommended. Advantages include good contraceptive efficacy, with continuous release leading to better compliance. The ring is not dependent on GI absorption, so it will continue its effectiveness even if the woman has a GI illness.[63] Side effects rarely include prolonged menstrual bleeding, vaginal discomfort, and foreign-body sensation. Use of antibiotics, such as amoxicillin and doxycycline, does not appear to affect efficacy.[52]

Special Issues Related to Hormonal Contraceptives and Wilderness Travel

Changes in Time Zone. Many of the low-dose OC formulations are time sensitive, so that forgetting to take a pill because of time zone changes or a change in schedule could result in ovulation. It is advised that a woman use a special wristwatch alarm dedicated to OC dosing control for precise determination of 24-hour intervals.

Absorption. Nausea, vomiting, or diarrhea during travel may decrease absorption of the pill. If vomiting occurs within 3 hours of taking a pill, a woman should take another. If a replacement pill cannot stay down (equivalent to missing a pill), she should use a backup method for the rest of the month. Elimination of the pill-free interval and starting the next package right away can also be considered if the missed pill was less than 7 days from the end of the package. For continued nausea and vomiting, some clinicians recommend inserting the OC pill into the vagina for absorption, although this has not been evaluated in clinical trials. Estrogens are actually absorbed better through the vaginal mucosa than through the GI tract.[81] For women at risk for GI illness during travel, a contraceptive patch or a ring is ideal.

Drug Interactions That May Affect OC Efficacy. A common question is whether antibiotics affect OC efficacy. The metabolism of OCs is increased by any drug that increases liver microsomal enzyme activity. Despite numerous case reports of

penicillins, tetracyclines, metronidazole, and nitrofurantoin causing contraceptive failure in humans, no large-scale studies have demonstrated that antibiotics other than rifampin lower steroid blood concentrations.[17,67,168] However, individual women have been shown to have decreased ethinyl estradiol while taking antibiotics. Because these women cannot be identified in advance, most clinicians and the OC package insert take a conservative approach and advise women to use a backup method of contraception when taking antibiotics.[51] Recent data suggest that efficacy of the contraceptive patch and the ring are not affected by concurrent antibiotics.[2,3,52]

Gender and the Risk for Venous Thromboembolism. The association between pregnancy and venous thromboembolism (VTE) is well established, and pulmonary embolism (PE) is one of the leading causes of peripartum mortality.[162] Increased risk for VTE is also associated with use of OCs, hormone replacement therapy, and estrogen-antagonist therapies. These exposures are unique (pregnancy and OCs) or nearly unique (estrogen agonist and antagonist therapies) to the female gender.[122] The risk for VTE may be important to consider in some wilderness settings, such as high-altitude expeditions, that may include a combination of risk factors such as immobility due to inclement weather, dehydration, cold, and exhaustion.

OCs are believed to exert their effect on the risk for thrombosis by causing alterations in endogenous coagulation and fibrinolytic systems. OC use is associated with increases in prothrombin, fibrinogen levels, and factors VII, VIII, and X, and decreases in factor V levels. Use of OC is associated with a twofold to threefold increase in the risk for VTE. Women who use third-generation OC drugs containing gestodene or desogestrel as the progestin component likely have a slightly greater risk than women who use second-generation preparations. Obese patients and smokers are at increased risk.[165]

The term *hormone replacement therapy* (HRT) refers to a variety of estrogen or combined estrogen and progestin formulations prescribed to relieve menopausal symptoms and prevent osteoporosis. Effects of HRT on the coagulation system appear to be similar to those of OCs, with evidence for increased markers of coagulation, factor VII levels, and activated protein C (APC) resistance and a decrease in antithrombin levels.[140]

Two selective estrogen receptor modulators (SERMs) are currently used with some frequency. Tamoxifen is used primarily for prevention and adjuvant treatment of breast cancer, and raloxifene has been approved for prevention of osteoporosis and is being investigated for use in breast cancer prevention. Although these agents have antiestrogenic properties, they also have partial agonist effects at selected receptors and might be expected to have some of the same side effects and associated risks as other estrogens.

The Breast Cancer Prevention Trial[58] reported a threefold increased risk for PE and a nonsignificant increased risk for deep vein thrombosis (DVT) among women with breast cancer receiving tamoxifen.

Pregnancy is a well-recognized risk factor for thromboembolic disease. It is estimated that pregnancy confers a threefold to fivefold increase in VTE risk compared with the nonpregnant state. Physiologic changes that occur during pregnancy pose several risks for VTE. Hypercoagulability results from increased levels of fibrinogen and several clotting factors (II, VII, VIII, IX, X, XII), as well as decreased levels of inhibitors of coagulation, such as protein S. This altered state of coagulation persists throughout pregnancy and for up to 6 weeks postpartum. Venous stasis occurs from hormonally induced increases in venous distensibility and capacity, as well as vena cava compression by the enlarging gravid uterus.[162] Vascular injury resulting from childbirth, especially cesarean birth, further escalates risk for VTE during the peripartum period; cesarean birth is associated with increased incidence of VTE compared with vaginal delivery. Other risk factors for VTE during pregnancy include obesity, maternal age, parity, prolonged bed rest, infection, prior history of VTE, and thrombophilia.[69]

Exposure to high altitude may be a risk factor for thromboembolic events due to polycythemia, dehydration, and cold, but increased risk from use of OCs or other hormonal therapy has not been systematically studied. As a precaution, women planning high-altitude excursions who desire hormonal contraception should consider a combination pill containing the lowest dose of ethinyl estradiol (20 µg) or its equivalent or a contraceptive patch or ring. Venous thromboembolism occurs more frequently in users of third-generation OCs (20 µg estrogen with desogestrel or gestodene as progestins). The contraceptive patch and the ring are absorbed though the skin and thus avoid first-pass metabolism in the liver. It is theorized that they may have less effect on the coagulation cascade than oral hormones.[122]

If a thrombotic or thromboembolic event is suspected on an expedition, aspirin therapy may be started empirically while awaiting medical evacuation. However, there are no studies evaluating the role of aspirin in reducing DVT related to altitude or air travel. Aspirin prophylaxis has been shown to reduce the incidence of venous thrombosis and pulmonary embolism in high-risk medical and surgical patients.[138]

Intrauterine Devices

An IUD is a highly effective and convenient form of contraception, especially for the parous woman in a stable relationship.[13,96] Women who cannot take estrogens due to a history of breast cancer, thromboembolism, or diabetes, or who are breast-feeding, are candidates for an IUD. Ideally, the IUD should be inserted at least 3 months before departure because complications occur most often within the first month after initial insertion. IUDs are occasionally associated with increased menstrual flow, dysmenorrhea, intermenstrual spotting, and expulsion. However, the Copper T 380A IUD and the levonorgestrel system (LNG-IUS or Mirena) carry minimal risk for these side effects. The LNG-IUS releases about 20 µg of levonorgestrel per day, which is about 10% of that of a levonorgestrel-containing OC. The levonorgestrel system requires replacement every 5 years. The Copper T 380 A IUD is effective for 10 years or longer. For certain women, an IUD is a great option for wilderness travel.[13]

The most serious risk, affecting less than 1% of IUD users, is acute or indolent pelvic infection that might become clinically significant in the wilderness. The risk for this is greatest within the first month after insertion or replacement of the IUD and among women at increased risk for STIs. Women with multiple partners, previous pelvic infections, unrecognized chlamydial infection or gonorrhea, recurrent episodes of bacterial vaginosis, or tobacco use are at highest risk. When acute PID occurs with lower abdominopelvic pain, peritonitis, purulent vaginal discharge, and fever, the IUD should be removed immediately by simple traction on the string protruding from the external cervical os. Broad-spectrum antibiotics should be started. Evac-

TABLE 88-4. Emergency Contraceptive Methods

	NO. OF PILLS PER DOSE	ETHINYL ESTRADIOL (μg)	LEVONORGESTREL (μg)	INSTRUCTIONS
Progestin only				
Levonorgestrel (Plan B)	1 tablet	0	0.75	Take first dose within 72 hr of risk Take second dose 12 hr later
Ovrette	20 tablets	0	0.75	May take both doses at once
Combined OC				
Ovral	2 white	100	0.5	Take first dose within 72 hours of risk
Lo-Ovral	4 white	120	0.6	Take second dose 12 hr later
Nordette	4 orange	120	0.6	May need antinausea medication
Levelen	4 orange	120	0.6	
Levora	4 white			
TriLevelen	4 yellow	120	0.5	
Triphasil	4 yellow	120	0.5	
Trivora	4 pink	120	0.5	
Alesse	5 pink	100	0.5	

See also emergency contraceptive options worldwide at http://ec.princeton.edu/worldwide/.

uation is mandatory when the device cannot be removed. Pelvic infection should be suspected, even without fever and peritoneal signs, if irregular bleeding occurs, particularly when accompanied by pelvic discomfort and discharge. When the IUD is removed at this stage, the infection may respond to therapy with oral or simple parenteral antibiotics.[13]

Another potentially serious risk for IUD users is pregnancy, occurring in approximately 1 in 1000 users per year with the levonorgestrel system. Both intrauterine and extrauterine (ectopic) pregnancies can occur with an IUD in place. Unfortunately, the latter is more common, but the risk is still only half that in women who use no contraception. Confirmed or suspected pregnancy in a woman with an IUD is an indication for immediate evacuation.

Emergency Contraception

EC is defined as a method of contraception that women can use after unprotected intercourse or contraceptive failure to prevent pregnancy (Table 88-4). EC should be used when desired in cases of unprotected intercourse, contraceptive failure, or sexual assault. EC should be included in every woman's wilderness medical kit. It decreases a woman's risk for pregnancy in an 8:100 to 1 to 2:100 ratio. The Princeton Emergency Contraceptive website (http://ec.princeton.edu) has an excellent review of the EC options available and commonly asked questions. It also has a chart of how a number of brands may be used as EC and what is available in different countries.[65,66]

EC options available in most countries include either two doses of estrogen-progestin combination OC pills or two doses of levonorgestrel pills (Plan B) taken 12 hours apart within 72 hours of unprotected intercourse. More recent studies have shown that Plan B is equally effective when both doses of pills are taken at the same time rather then 12 hours apart.[55,135] Plan B is better tolerated then the other ECs that contain estrogen. Presently, Plan B is the only option packaged specifically for EC available in the United States and Canada. In Canada, the United States, and many other countries, EC is available over the counter.

EC regimens using standard OC formulations can be used. Options include Ovral (0.05 mg ethinyl estradiol and 0.50 mg norgestrel), two tablets; Lo-Ovral (0.03 mg ethinyl estradiol and 0.30 mg norgestrel), four tablets; and Nordette or Levlen (0.03 mg ethinyl estradiol and 0.15 mg levonorgestrel), four tablets. The dose is repeated at 12 hours. Common side effects include nausea, vomiting, irregular bleeding, cramping, and headache, but these are usually transient and can be managed symptomatically. Administering an antiemetic may be helpful when using OCs containing estrogen and progesterone.[70]

Breast Health

Clothing and sports equipment should be tested ahead of time for breast comfort. Sports bras are helpful for many women. All women should examine their breasts for masses before travel. Women older than 40 years and women with a strong family history of breast cancer should have a breast exam and mammography screening before travel. Although there are reports of self-diagnosis and treatment in the field,[84] ideally a breast mass would be evaluated before travel to a remote location.

A woman who has had a mastectomy and lymph node dissection with resulting lymphedema may want to use an arm sleeve to decrease swelling during air travel and extended backpacking or trekking expeditions. Women should be taught how to recognize cellulitis and how to treat it with appropriate antibiotics.[79] There have been no large studies on the safety of breast implants at altitude or during scuba diving. In one small study evaluating breast implants in simulated dives, there were no ruptures, but there were morphologic changes.[71] Issues pertinent to breast-feeding are discussed in the pregnancy section.

Urinary Tract Issues

Women are prone to urinary tract infections (UTIs) during wilderness travel. Contributing factors include dehydration, less frequent urination owing to lack of convenient toilets, fewer available facilities for hygiene, and increased sexual activity.

Figure 88-4. Urinary director. (Photograph by Susan Auderson.)

Sexual intercourse increases the risk for UTI at all ages because of urethral massage, which introduces bacteria into the bladder. Voiding after intercourse lessens this risk. The risk for UTI is increased during pregnancy owing to urinary stasis and pressure of the uterus on the ureters. UTI in older women may be due to changes in the urogenital epithelium. Thus, women of all ages should be taught to recognize the symptoms of UTI and how to treat it with oral antibiotics and a urinary analgesic agent. Urinary dipsticks used as a screening test to check for leukocytes and nitrites may be carried in the medical kit.

Measures to prevent UTIs include adequate hydration and urination when needed, even if there is no formal facility or a tree behind which to hide. Some women find the squatting position awkward. Practicing deep knee bends before the trip is helpful. Women should consider attire, such as a free-flowing skirt, that would both facilitate this stance and add some privacy. There are a number of plastic and paper funnels that have been designed to assist women to urinate in the standing position (Fig. 88-4). These methods require practice to avoid getting wet. It is best to be with one's back to the wind and to point the funnel downwards. When wearing pants, it is important to make certain that the zipper or other opening is long enough to fit the device. The funnels are especially useful in extremes of cold weather and altitude when a woman might not want to wear a skirt or pull down her pants. It is also possible to connect the device to a longer tube that is connected to a container for urine storage if it is too cold to urinate outside the tent or a woman is on a long road trip.

One product, called the WHIZ, mixes an antibacterial agent into the plastic (similar to what is done with cutting boards) and then coats the device so that it repels fluid and is easily cleaned. For the extremely cautious user, the device may be cycled in a washing machine.

Options for female urine directors include the following:
Freshette: www.freshette.com
My Sweetpee: www.mysweetpee.com
Travelmate: www.travelmate.com
WHIZ all-terrain director: www.whizaway.com

If an older woman has a problem with stress incontinence or bladder control, she should consult a physician specializing in female urinary tract problems before her trip. For minor problems, women can be taught to do pelvic floor exercises and to bring a supply of panty liners. Older women may experience vaginal dryness and urinary frequency or urgency without dysuria. Recent data suggest that estrogen vaginal cream, vaginal rings, or even an OC pill inserted intravaginally may decrease urogenital dryness and frequency symptoms.[21,64]

Vaginal Discharge or Itching

Environmental conditions and constraints on hygiene during wilderness travel promote a change in a woman's vaginal ecosystem that may result in increased vaginal discharge and vulvar itching and irritation. Causes range from infection to contact irritation due to a new soap, feminine hygiene product, or contact with a poison oak twig when urinating. Because diagnostic capabilities in the wilderness are limited, clinical features (Table 88-5) can help guide diagnosis and treatment. Women should be taught self-diagnostic skills and given the appropriate treatment to include in the medical kit. Even if a woman has never before had a vaginal infection, she should be prepared for this possibility, especially for extended itineraries. Women should be advised to seek medical evaluation if the symptoms do not improve with self-treatment. The most common causes of vaginitis during wilderness travel are yeast infection, bacterial vaginosis, and chemical irritation.

Vulvovaginal Candidiasis

Symptomatic yeast infections are often referred to as vulvovaginal candidiasis (VVC). About 80% to 90% of these result from overgrowth by *Candida albicans*. Risk factors for yeast infections include pregnancy, hormonal therapy, recent antibiotic use, corticosteroid therapy, postovulatory phase of menstrual cycle, frequent coitus, condom use, and intravaginal use of spermicidal compounds.[54]

The most common complaint of women with VVC is vulvar pruritus or burning, not vaginal discharge. In more severe cases, redness, irritation, burning, soreness, swelling, and external dysuria are variably present. The characteristic white, flocculent ("cottage cheese"), and adherent discharge is often diagnostic, but is not consistently present or visible externally. The yeast discharge is thicker than that seen with bacterial vaginosis or trichomoniasis, is usually not frothy or malodorous, and often has a pH of 4.5 or less, unless a mixed infection is present. About 50% of yeast infections are confirmed by direct microscopic examination of the discharge diluted in saline; however, diagnosis is most reliably accomplished by detection of budding yeast hyphae or spores using a slide preparation with 10% potassium hydroxide (KOH) added to lyse background epithelial cells and bacteria.

The foundation of treatment for VVC is a variety of azole derivatives, many of which (e.g., butoconazole, clotrimazole, miconazole, tioconazole) are available over the counter as topical creams, vaginal tablets, and suppositories. Therapy periods range from 1 to 14 days, depending on the formulation and severity of the infection. Generally, a treatment regimen of at least 3 days results in a greater initial response rate and a lesser chance of immediate recurrence. Symptoms related to inflammatory vulvar involvement respond most rapidly to the topical creams, although their application at first may be accompanied by burning pain. Oral fluconazole (150 mg, single dose) is a convenient therapeutic agent to include in the basic pharmacopoeia of a wilderness expedition. Women with frequent

TABLE 88-5. Differential Diagnosis of Vulvovaginitis

FACTORS	NORMAL	BACTERIAL VAGINOSIS	VULVOVAGINAL CANDIDIASIS	TRICHOMONIASIS	ATROPHIC VAGINITIS	OTHER
Discharge	White, clear, finely granular	Gray-white, thin, homogeneous, adherent, frothy	White, thick, curdlike, adherent	Gray to yellow-green, occasionally frothy, adherent	Thin, clear to serosanguineous	Normal
pH	3.8–4.2	>4.5	≤4.5	>4.5	>4.5	3.8–4.2
Amine odor	Absent	Present	Absent	Variably present	Usually absent	Absent
Primary complaints	None	Malodorous discharge	Pruritus, irritation	Severe pruritus, discharge, dyspareunia, dysuria	Burning, soreness, dyspareunia	Burning, irritation, swelling, soreness
Microscopic appearance	Normal epithelial cells, lactobacilli	"Clue cells," no WBCs	Budding yeast, hyphae, spores	Trichomonads, many WBCs (PMNs)	Small, round (parabasal) epithelial cells, many PMNs	Normal
Other findings and diagnostic features	None	Minimal vulvar involvement	Vulvar and vaginal erythema, predisposing medical conditions	Intense vulvovaginal erythema, "strawberry cervix," other STIs	Atrophy of vulva and vaginal epithelium	Highly variable

PMNs, polymorphonuclear neutrophil leukocytes; STIs, sexually transmitted infections; WBCs, white blood cells.

recurrences or predictable outbreaks at specific times in their cycle, most often premenstrually, should consider prophylactic suppressive therapy with fluconazole (150 mg orally) or clotrimazole (500 mg vaginal tablet) weekly.[154] To treat local symptoms, a low-potency steroid cream can be used in conjunction with a topical antifungal.

Bacterial Vaginosis

BV is the most common cause of vaginitis in women of childbearing age. The most common complaints of women with BV are discharge and odor, itching, and irritation of the vulva and vagina. More than 50% of women with BV do not complain of symptoms or are unaware that symptoms result from a treatable condition.

BV is believed to result from a disturbance in the normal vaginal flora whereby the normal levels of hydrogen peroxide–producing lactobacillus are replaced by less dominant organisms.[131,152] The discharge accompanying BV is typically thin, watery, grayish white, frothy, and homogeneous (not flocculent), uniformly coating the vaginal walls and introitus. More than 50% of women with BV complain of a fishy odor, particularly during menstruation and immediately after unprotected sexual intercourse. Blood and semen can alkalinize the vagina and volatilize a variety of amines (e.g., cadaverine) produced by anaerobic organisms.

The diagnosis of BV is confirmed by presence of three of the following: (1) discharge, (2) pH greater than 4.5, (3) release of amines (fishy odor) when discharge is exposed to 10% KOH ("whiff test"), and (4) microscopic detection of "clue cells" (epithelial cells coated with bacteria) in saline solution. Microscopic appearance of pure BV is characterized by few if any leukocytes and few motile, curved rods (lactobacilli).

The antimicrobial agent most successful in treating BV is metronidazole, which can be administered orally (500 mg 2 or 3 times daily for 5 to 7 days) or as a 0.75% vaginal gel (once or twice daily for 5 days).[152] These regimens have initial response rates in excess of 90%. Single-dose oral treatment with metronidazole (2.0 g) yields initial response rates of 80% to 90% but is accompanied by higher recurrence rates within 1 month of treatment. This regimen should be considered when drug supplies are limited or when the GI side effects of oral metronidazole are not well tolerated. Clindamycin (300 to 600 mg orally twice daily for 5 to 7 days or 2% vaginal gel daily for 7 days) has comparable efficacy, but disadvantages are expense, deleterious effects on normal vaginal lactobacilli, and increased risk for pseudomembranous enterocolitis, a rare effect that could be life-threatening in the wilderness.

Trichomonas Vaginitis

Trichomonas vaginalis is a single-celled parasite that causes vaginitis in 2 to 3 million women annually in the United States.[172] It is predominantly sexually transmitted and is found most often in individuals with multiple sexual partners and those with a history of or current STIs. Diagnosis and treatment of a woman and her sexual partner are best accomplished at a screening visit before departure on a wilderness excursion. Unlike BV and yeast vaginitis, detection of *T. vaginalis,* even in asymptomatic women, is an indication for treatment and more complete STI screening.

The most common complaints include severe vulvovaginal pruritus, dyspareunia, and dysuria. Physical examination often reveals intense erythema of the vagina and introitus and petechial lesions of the cervix ("strawberry cervix"). The vaginal discharge is typically gray or yellow-green, somewhat cloudy, and variably frothy and malodorous. Presence of the latter two findings frequently indicates a mixed infection with the amine-producing organisms seen in BV. The pH of vaginal fluid is usually above 5 and frequently exceeds 6.

Because of diagnostic difficulties in the wilderness, empiric therapy is justified. Metronidazole is the only drug approved for trichomoniasis in the United States. It is administered in a single oral dose (2 g), and in severe cases or single-dose failures, for a week (500 mg bid) or longer. Tinidazole, 2 g orally, may also be used. For optimal results, sexual partners should be treated simultaneously and sexual activity curtailed during therapy.[153] To minimize GI side effects, metronidazole should be taken with plenty of water. This may not reduce the unpleasant metallic taste, but may reduce the risk for nausea, vomiting, and gastric irritation. Because a disulfiram-like effect is possible, alcohol should be avoided while taking metronidazole. At present, metronidazole 0.75% vaginal gel is not appropriate for treatment of trichomoniasis.

Atrophic Vaginitis

Decrease in estrogen levels during perimenopause and menopause can cause vaginal atrophy. This effect can also occur in hypoestrogenized premenopausal women, such as some amenorrheic athletes and women on ovarian suppressive therapy with GnRH agonists. The presumed etiology is lost estrogen effect on the vaginal epithelium, causing thinning of the vaginal epithelium and reduction in epithelial cell glycogen, an important substrate for lactobacilli, which leads to changes in vaginal pH and flora with subsequent overgrowth of non-acidophilic organisms. Symptoms include burning or soreness, dyspareunia, and vaginal discharge that is often watery or even serosanguineous. Typically, the vagina is uniformly erythematous and may have areas of petechiae. The pH of the vagina usually exceeds 5 (often 6 to 7), and microscopic evaluation of the discharge reveals small, round, immature epithelial cells (parabasal cells), increased neutrophils, and a paucity of lactobacilli. Treatment includes the use of topical or oral estrogen replacement therapy.[131] Treatment with low-dose topical estrogen (cream, ring, or tablet) usually provides complete relief of symptoms within weeks. In the interim, women may obtain relief by using vaginal moisturizers and lubricants (Astroglide, Replens).

Noninfectious Vulvovaginitis

Certain noninfectious causes of vulvovaginitis result from environmental exposures that cause local irritation or allergic reactions. These may be of greater significance when attention to personal hygiene is limited.[76] Common causative agents include latex condoms, spermicidal compounds, soaps, detergents, fabric softeners, deodorant products, menstrual pads and tampons, and topical medications such as antimycotics and povidone-iodine. Exclusion of an infectious etiology and identification of the source of the reaction usually make the diagnosis. Once a potentially offending cause, such as a recent change in laundry detergent, is identified, the first step in management is removal from exposure. Treatment is usually symptomatic (pain relief, antihistamines, sitz baths). Topical corticosteroids creams may be tried; however, in rare instances, the symptoms may be exacerbated.

Sexually Transmitted Infections

If a woman has a new discharge and pelvic pain following a sexual encounter, she may have an STI and should be evaluated as soon as possible. The importance of safe sex practices and a discussion of the risks associated with the range of STIs pertinent to the destination should be included as part of the pre–wilderness travel visit.[83,172]

A woman is at a higher risk for acquiring an STI from an infected man than a man would be from an infected woman. The long-term complications related to STIs may be more severe in women than in men, and include PID, chronic pelvic pain, and infertility. To prevent acquisition of sexually transmitted diseases, women should avoid casual sex and always practice safe sex by using condoms no matter what other means of contraception are used simultaneously. High-quality latex condoms are an essential part of a traveler's medical kit regardless of gender. A male or female condom made of polyurethane is an effective alternative for persons allergic to latex.

If a woman has a history of genital HSV infection, she should bring a supply of antiviral medication (acyclovir, valacyclovir) in case she has an outbreak during travel.

Nonoccupational Postexposure HIV Prophylaxis

Women should be educated about the availability of postexposure HIV prophylaxis for high-risk sexual exposures or sexual assault.[151] If a woman is traveling to a remote area, the initial week of treatment could be included in the traveler's medical kit. Travelers should check with the Centers for Communicable Disease or similar source for the latest information on nonoccupational postexposure HIV prophylaxis recommendations:

www.cdc.gov
www.mass.gov
www.hivguidelines.org

Home testing kits for HIV are available in many countries. The sensitivity and specificity of these tests may vary. The HIV test should be repeated with a reliable laboratory analysis.[133,157]

Personal Safety

Women of all ages should take a self-defense course. There are many self-defense devices available, such as pepper spray (made from cayenne pepper), Mace (tear gas), capsules that let out a pungent odor, and sound alarms. Some examples may be found at www.homesecuritystore.com.

Sexual Assault

Sexual assault of women in the wilderness is a rare occurrence. Wilderness morbidity and mortality statistics are limited, but one study of eight National Park Service areas in California over 3 years reported only one incident of sexual assault.[117] Many incidents are probably not reported, however, because only 7% of all rapes are reported.[24] The World Health Organization estimates that between 20% and 50% of women in the world have been physically or sexually assaulted by a man at some point in their lives.

The best defense against sexual assault is not going into the wilderness with unfamiliar people. The chance of meeting an assailant is quite low. A woman traveling into the wilderness alone or with someone she does not know well is advised to tell friends or family exactly where and with whom she is traveling and when she anticipates returning.

If sexually assaulted, the woman is advised to seek medical attention as soon as possible. Most emergency departments are prepared to handle the evaluation and treatment of sexual assault victims. It may be impossible to reach a medical facility for many hours or even days, but an attempt should be made to preserve potential evidence. Women are advised to avoid douching, gargling, brushing teeth, or changing clothes. If

clothes are removed, clothes should be placed in a paper bag and brought to the medical facility. The CDC recommends STI prophylaxis, postexposure prophylaxis for HIV, and EC for women at risk.[24] Post-traumatic stress counseling should also be offered.

WILDERNESS TRAVEL DURING PREGNANCY

Many women involved in wilderness activities expect to continue their adventures at all stages of pregnancy. These women understand the benefits of exercise and the positive effect that participating in a wilderness experience has on their general health and well-being.[9] However, pregnancy is considered a relative contraindication to wilderness activities unless access to medical care is available in the field or provisions are made for rapid evacuation. The pregnant traveler's itinerary should be reviewed and assessed for risks relating to a destination or a specific activity that may be a risk to the mother or fetus. If there are risks involved, the pregnant traveler should be counseled so that she can take preventive measures or consider an alternative plan. Although prior pregnancy is a relatively good predictor of outcome after the first trimester, pregnancy is still characterized by its unpredictability. A pregnant woman should consider the potential risks involved if an unexpected complication were to occur and how she would feel if the outcome resulted in preventable maternal or fetal morbidity and mortality.

Much of the information on the pregnant traveler is based on small studies, anecdotal information, and extrapolation from nonpregnant travelers. Although evidence-based recommendations are ideal, they are lacking for pregnant women. This section summarizes the available data and experiences in this growing field of wilderness medicine.

Pre–Wilderness Travel Evaluation during Pregnancy

Box 88-4 is a checklist for pregnant travelers.

Basic questions to ask when counseling pregnant women anticipating travel to remote places include the following[15]:

- What are the medical and obstetric risks associated with wilderness travel?
- What medical services are available during transit and in the areas of destination?
- What does health insurance cover if the traveler is out of area for delivery or pregnancy-related complications?
- What are signs of serious pregnancy-related illness for which emergency medical help should be sought?
- What are some general guidelines to follow for the medical management of illness that will safeguard the pregnant woman and her fetus?
- Are the required and recommended immunizations for the itinerary safe in pregnancy?
- What medications are safe in pregnancy?
- Are there special concerns related to the environment that need to be considered?
- Are there special concerns related to a sports activity that need to be considered?
- Is the traveler prepared for a "wilderness delivery"?

Pre–wilderness travel preparation starts with a review of the women's obstetric and medical history. Health practitioners who advise pregnant women should work closely with the woman's obstetrician to assess potential benefits and risks involved in a particular wilderness trip. Any history of pregnancy problems, including bleeding, preterm labor, or chronic illness, increases risk. Categories that define potential high-risk pregnancies for which travel should be delayed or the itinerary adjusted are summarized in Box 88-5.

The obstetric history should be reviewed for complications for which there may be a high risk of recurrence, including preterm labor, premature rupture of membranes, preeclampsia, gestational diabetes, fetal growth restriction, group B hemolytic streptococcal infection, UTI, chorioamnionitis, blood group isoimmunization, thromboembolic event, surgical delivery, and postdelivery complications. A complete physical examination should be done.

Laboratory evaluation before departure includes standard blood work recommended by the American College of Obstetricians and Gynecologists: complete blood count, blood type,

Box 88-4. Checklist for Pregnant Travelers

PRETRAVEL RISK ASSESSMENT
Stage of gestation
Obstetric risk factors
Medical risk factors
Destination risk considerations
- Access to care
  - Medical services available during transit and at destination
  - Emergency evacuation insurance
  - Review emergency signs and symptoms
    - Vaginal bleeding, abdominal pain, contractions, proteinuria, headache with visual change, severe edema and/or accelerated weight gain, rupture of membranes
- Due to infectious disease
  - Chloroquine-resistant *Plasmodium falciparum* malaria
  - Outbreak of disease requiring a live virus vaccine
  - Outbreak of disease for which no vaccine is available but has a high risk for maternal and fetal morbidity and mortality
- Due to food water exposure
- Due to insect exposure
- Due to environment
- Exercise risk
  - Altitude
  - Heat
  - Cold
  - Open water

Recommendations
Medical insurance and evacuation coverage
Immunizations to reflect actual risk for disease and probable benefit
Medications—review safety during pregnancy
Preventive measures
Medical kit adaptations for pregnancy
Plans for emergency delivery
Postpone wilderness travel if risks outweigh benefit or adapt itinerary to decrease risk

Box 88-5. Potential Contraindications for Wilderness Travel during Pregnancy

OBSTETRIC RISK FACTORS
Extremes of maternal age
Vaginal bleeding this pregnancy
Multiple gestation this pregnancy
History of gestational diabetes or hypertension
History of miscarriage, preterm labor, or premature rupture of membranes

MEDICAL RISK FACTORS
History of thromboembolism
Cardiac disease
Severe anemia
Medical disease requiring ongoing assessment and medication: diabetes, pulmonary, or kidney disease

RISK FACTORS AT WILDERNESS DESTINATION
Lack of access to medical care
High altitude
Disease requiring a live virus vaccine (yellow fever, measles)
Mefloquine-resistant *Plasmodium falciparum* malaria
Epidemic of infectious disease leading to high-risk maternal and fetal morbidity
Natural disasters

RISK FACTORS DUE TO SPORTS ACTIVITY
Scuba diving
High risk for trauma

antibody screen (and screen of partner if the woman is Rh negative or isoimmunized), and basic serology (rapid plasma reagin, rubella, hepatitis B, HIV). Serologic screening for HSV-2 should be considered in the woman with no history of genital herpes because of the potential for first-time outbreaks during pregnancy in women with unrecognized infection. Individuals at risk and those not previously evaluated should also be offered hemoglobin electrophoresis to assess for hemoglobinopathies. Urinalysis and urine culture are performed because of the high frequency of asymptomatic infections during pregnancy that complicate outcome. Vaginal fluid should be assessed for BV because treatment early in pregnancy may prevent premature rupture of membranes and preterm labor. A Pap smear should be done. A woman beyond 15 weeks' gestation before departing should be offered biochemical screening (e.g., maternal serum alpha-fetoprotein, estriol, hCG) to assess for certain congenital and chromosomal abnormalities. Abnormal biochemical markers may indicate complications, such as fetal growth restriction and preeclampsia, resulting primarily from early abnormalities in placentation that can become clinically significant later in gestation. These conditions also increase the risk for premature delivery as well as for maternal and fetal morbidity and mortality.

Screening for diabetes should be done. If the woman had gestational diabetes with a previous pregnancy, it is best to assume she will have it again. Proper dietary counseling and regular blood sugar monitoring with a portable plasma glucose monitor should be conducted throughout the pregnancy. Insulin, sufficient syringes, and alcohol wipes should be included with the basic medical supplies in the event glycemic control deteriorates. The goal is to maintain fasting plasma glucose levels at 90 mg/dL or less and lower than 120 mg/dL 2 hours after meals. Physical conditioning reduces but does not eliminate the risk for developing diabetes during pregnancy. Women with pregestational diabetes of any duration should have a baseline electrocardiogram and possibly an echocardiogram before participation in any wilderness-related activities.

All pregnant wilderness travelers should have an obstetric sonogram before departure for additional risk assessment. Early in the pregnancy, sonography can accurately confirm gestational age, viability, intrauterine location, and number of babies. It is also useful in ruling out the presence of an ectopic pregnancy, adnexal mass, or molar pregnancy that could lead to life-threatening complications. In the second trimester, an ultrasound is useful not only in estimating gestational age but also in determining the presence of major fetal abnormalities, the location of placenta in relationship to the cervix, and cervical length and integrity of the internal cervical os. Beyond 20 weeks' gestation, normal fetal growth and blood flow patterns in the umbilical and middle cerebral arteries can be assessed by Doppler velocimetry. Normal results indicate lower risk for complications, such as intrauterine growth restriction, preeclampsia, and preterm labor. Any findings that significantly increase maternal or fetal risk are contraindications to travel.

Counseling on timing of wilderness activity should be done, although no interval during pregnancy is considered absolutely safe. The American Academy of Obstetrics and Gynecology states that the safest time for women to travel is the second trimester. The pregnancy is established and the extra weight is not usually a functional limitation for the mother. During the first trimester, the risk for miscarriage is the highest. The first trimester is also the period when theoretical effects on a fetus from medications or vaccines needed by the mother would be the greatest. The primary risks in the third trimester are complications such as bleeding, preeclampsia, and preterm labor and delivery.

One of the most important considerations for a pregnant woman planning wilderness travel is the available options in case of an emergency. The complete itinerary should be evaluated with attention to both availability and quality of medical care during transit and at the final destination. Access to high-quality care during travel is essential in case of preterm labor or an unexpected complication of pregnancy.

All pregnant women traveling to a more remote area or to a less developed country should check their health insurance policy for coverage guidelines. An additional travel health insurance policy that provides a worldwide 24-hour medical assistance hotline number may need to be purchased. This service would provide telephone contact with medical personnel who can help arrange emergency medical consultation and treatment, monitor care, and provide emergency evacuation to a more advanced medical facility if necessary. It must be remembered that medical evacuation can take hours to days from remote locations, so one should not hold a false sense of security. Each policy must be reviewed carefully to make sure that it covers the expenses associated with a normal pregnancy (such as delivery) as well as with the possible complications of pregnancy, such as a miscarriage early in pregnancy or preeclampsia in the third trimester. The policy should also cover expenses associated with care of the neonate.

All pregnant women should be taught the warning signs of a potentially serious problem: bleeding, passing tissues or clots, abdominal pain or cramps, rupture of membranes, headache, or visual changes. The pregnant woman should have a plan for what to do if any of the above occurs. She should carry a copy of her medical record (including blood type and Rh) and her physician's phone number, fax number, and email address. This information may be helpful for routine questions or if there is an emergency.

Physiologic Changes Accompanying Pregnancy

The normal physiologic changes of pregnancy may impact wilderness travel. These include reduced exercise and heat tolerance, and elevated heart rate related to physiologic anemia and increased plasma volume.

Hormonal changes accompanying pregnancy result in physiologic adaptations affecting every organ system within weeks of conception. For example, progesterone has smooth muscle relaxation effects that help to maintain uterine quiescence, but these also contribute to vasomotor instability, hypotension, gastric reflux, and constipation. Estrogens stimulate hepatic production of many hormone-binding globulins and of coagulation factors II, V, VII, VIII, IX, X, and XII, and fibrinogen, which contributes to the hypercoagulable state of pregnancy and risk for thromboembolism.[42]

Cardiovascular Adaptation

Pregnancy affects maternal hemodynamics by inducing an increase in blood volume, heart rate, and stroke volume and a decrease in systemic vascular resistance (SVR). Cardiac output increases 30% to 50%, much by the end of the first trimester, from an increase in stroke volume secondary to an increase in preload. The balance comes from a gradual increase in maternal heart rate that usually peaks between 24 and 28 weeks' gestation. Uterine blood flow increases from 50 to more than 500 mL/min, corresponding to an increase from about 1% to 20% of total cardiac output. SVR decreases, primarily from the low-resistance placental vascular bed, the equivalent of a large arteriovenous shunt, and also from the peripheral vasodilatory effects of progesterone, estrogen, and other factors. These hemodynamic changes help to provide the circulatory reserve necessary to provide nutrients and oxygen to the mother and fetus at rest and during moderate exercise.[9]

Changes in Blood Volume

Total blood volume increases 40% to 50% during normal pregnancy from a rapid expansion of plasma volume. A disproportionate increase in plasma volume over red cell mass results in the so-called physiologic anemia of pregnancy. The physiologic effects of hypervolemia and anemia during pregnancy have several benefits. Decreased blood viscosity (from greater increases in plasma volume than red cell volume) results in reduced resistance to flow, facilitating placental perfusion and lowering cardiac work. The increase in blood volume to about 50% above nonpregnant values provides some reserve against the normal blood loss during parturition. Most of the increase in cardiac output is distributed to the placenta, kidneys, and skin to provide nutrients to the fetus, excrete maternal and fetal waste products, and assist maternal temperature control, respectively.[9]

Respiratory Status

Respiratory system changes that occur during pregnancy help to compensate for the physiologic anemia to maintain fetal and maternal homeostasis. Progesterone directly stimulates the respiratory center and increases its sensitivity to CO_2, resulting in a 30% to 40% increase in tidal volume (TV). Respiratory rate (RR) does not change significantly, but as a result of TV changes, minute ventilation (TV × RR) increases 25% to 30%, despite a slight decrease in total lung capacity. A 20% reduction of functional residual capacity, resulting from decreases in both expiratory reserve volume and residual volume, completes the picture of relative hyperventilation with a compensated respiratory alkalosis. Many of these changes are completed by the end of the first trimester; their sum is a dramatic 50% increase in alveolar ventilation, increased Pa_{O_2}, 30% increase in one-minute O_2 uptake, and significant decrease in P_{CO_2}. The overall effect is to increase the O_2-carrying capacity of maternal blood to accommodate fetal and maternal metabolic needs, while facilitating diffusion of CO_2 from the fetus. For the wilderness traveler, these changes lead to higher O_2 saturation under hypoxic conditions, such as at altitude.

Urinary System

Significant changes in the urinary system account for many of the complaints and complications of pregnancy, including urinary frequency, incontinence, and increased risk for urinary tract infections. Beginning in the first trimester, the ureters become dilated, elongated, and more tortuous, presumably under the influence of progesterone. Further dilation of the proximal ureters occurs when the uterus reaches the level of the pelvic brim at about 20 weeks' gestation and compresses the ureters. Frequently at this stage, women present for the first time with pyelonephritis, more often on the right than on the left owing to dextrorotation of the uterus by the descending colon. Contributing to the risk for upper tract infection, vesicoureteral reflux occurs secondary to decreased competency of the ureterovesical junction. This is exacerbated by progressive decrease in bladder capacity and doubling of intravesicular pressures (from 10 to 20 cm H_2O) during gestation. These factors also contribute to frequent complaints of urinary incontinence that plague pregnant women.

Integumentary and Musculoskeletal Status

Integumentary and musculoskeletal changes can have significant effects on the well-being of the pregnant wilderness traveler.[25] Under the influence of estrogen, proliferation and dilation of small arterioles in the skin help to compensate for increased demands to remove heat generated by maternal and fetal metabolism. Because of these inherent changes, pregnant women have limited capacity to respond further to heat stress and are at increased risk for hyperthermia in hot and humid environments. Estrogen and other pregnancy-associated hormones also increase sensitivity of the skin to damage by sun exposure, particularly in fair-skinned individuals. Some pregnant women have a predisposition to develop skin hyperpigmentation in a nonuniform distribution because of excessive melanin deposition in the dermis and epidermis. This is enhanced by sun exposure, and it often affects the face (melasma), midline abdomen (linea nigra), nipples, axillae, and perineum. It may require a prolonged period for resolution after delivery. It may never resolve completely.

Weight gain, weight redistribution, and ligamentous relaxation may pose the greatest risks, even to the well-conditioned pregnant woman.[12] Overall weight gain during pregnancy is usually about 20 to 35 pounds. Some weight gain is important during pregnancy to avoid a catabolic state and may be more

important to the highly conditioned athlete who enters pregnancy with limited fat stores. This weight increases stress on the weight-bearing skeleton and ligaments throughout the body and may accumulate more rapidly than conditioning can handle. Much of the weight gain is contributed by uterine and fetal growth, resulting in forward displacement of the center of gravity. This is usually accompanied by progressive lordosis of the lower spine and increased strain on spinal ligaments, disks, and paravertebral muscles. When lumbar lordosis is exaggerated, traction and compression on the sciatic nerves can cause significant pain and weakness in the buttocks and lower extremities. Changes in the lower spine are frequently followed by compensatory flexion of the cervical spine. This in turn can place traction on the median and ulnar nerves, resulting in pain, paresthesias, and weakness of the upper extremities.[25]

The challenges of weight gain are accompanied by dramatic changes in ligamentous support throughout the body. Under the influence of relaxin and other hormones, ligaments become more compliant and hydrophilic. Benefits of this include relaxation in the sacroiliac joints and symphysis pubis to facilitate delivery, but hormonal effects on other ligaments can lead to complications. For example, fluid retention by the flexor retinaculum in the wrist can cause compression of the median nerve, resulting in carpal tunnel syndrome, a common complaint of pregnancy. This may be more than just a nuisance to the wilderness traveler because pain and hand weakness can compromise activities, such as climbing and canoeing, that require hand strength and endurance. Even more troublesome, pelvic girdle instability, accompanied by weight gain, shift in center of gravity, and spinal lordosis, usually leads to gait and balance disturbances, interfering with enjoyment of many wilderness activities such as hiking or skiing. These changes also lead to an increase in the severity of trauma accompanying falls. Maternal trauma remains one of the leading causes of fetal death due to placental abruption or preterm labor.[144] The anterior cruciate ligament is especially prone to severe trauma, accounts for 3 to 4 times more injuries in women than in men, and is especially susceptible to trauma in the active pregnant woman.[169] Difficult terrain also poses a risk to the pregnant wilderness traveler.

Changes in Immune Status: Response to Infection and Vaccine

Pregnancy results in a number of changes in the maternal immune system. Evidence from a number of studies suggests that there is a reduction in cell-mediated immunity during pregnancy. Pregnancy has been found to result in increased susceptibility or predisposition to more severe disease during a number of infections in which the cell-mediated immune response is the most important. Examples include malaria, amebiasis, coccidiomycosis, leishmaniasis, leprosy, listeriosis, and tuberculosis.[132] Thus, when the itinerary is being reviewed, risk for these infections should be determined. By contrast, infections in which the humoral system is the most important response show no increase in susceptibility to infection. B-cell numbers and functions do not appear to be reduced during pregnancy.

Immunizations during Pregnancy and Lactation

Ideally, all women should be vaccinated before pregnancy.[144] The risks and benefits of immunizations during pregnancy must be weighed against the woman's risk for illness, likelihood of adverse fetal outcomes if the mother becomes ill, and risks for the vaccine. The risks for the immunizations during pregnancy are largely theoretical. Benefits of vaccinating pregnant women usually outweigh potential risks when risk for exposure to the disease is high.[1,11,62]

Breast-feeding is not a contraindication to vaccination. For a woman trying to conceive, it is recommended that she wait for 1 month after receiving a live virus vaccine (e.g., MMR, varicella) to get pregnant. If a woman receives a vaccine and later finds out that she is pregnant, there is no indication to terminate pregnancy.[1,11]

Ideally, vaccinations should be avoided in women during the first trimester owing to the uncertain effect on the developing fetus. Serologic studies for hepatitis A or B, varicella, measles, and rubella may be checked to assess potential risk for infection.

In general, inactivated vaccines are safe, and live vaccines are contraindicated during pregnancy. Tetanus and diphtheria toxoids and influenza vaccine are routinely given during pregnancy; hepatitis A and B, Japanese encephalitis, meningococcal, rabies, and typhoid fever (inactivated) vaccines may be administered during pregnancy if there is a risk for exposure to these infections.

The safety of yellow fever vaccine during pregnancy is unknown. If the risk for yellow fever infection is low but proof of immunization is required, a medical waiver can be given. If the risk for yellow fever is high and travel is unavoidable, vaccine should be given. A small study that evaluated pregnant women who had inadvertently been vaccinated with the yellow fever vaccine did not find adverse fetal or maternal outcomes.

All vaccines are listed as category C under the FDA pregnancy categories[1,30] (Table 88-6).

FDA Guidelines

The FDA has developed a set of guidelines to categorize drugs, vaccines, and toxoids with regard to developmental toxicity and adverse fetal outcome. The assessments are based on the degree to which available information has ruled out a risk to the fetus balanced against potential benefits to the pregnant woman[30] (Box 88-6). Most medications fall under FDA category C. Few double-blind studies have been done in pregnant women to categorize drugs.

Medication Use during Pregnancy

All physicians advising pregnant women should have ready access to references related to medication use during pregnancy. *Drugs in Pregnancy and Lactation*[30] includes reviews of the reproductive literature relevant to drugs and immunizations. Each medication is categorized by the risk classifications outlined by the FDA. There are Internet resources with information on teratogenic risk of particular medications or vaccines.[103] Table 88-7 lists some of the medications commonly used by pregnant women during travel.

Infectious Disease Risk

Food- and Water-borne Disease

The main concern with traveler's diarrhea during pregnancy is dehydration, which can compromise placental blood flow and adversely affect the fetus. Reduced gastric acidity during pregnancy may predispose to GI illness.

Boiling water is the most effective and safest method of disinfection. Iodination of water is not recommended owing to the risk for fetal goiter. Other options are chlorination or a filter

TABLE 88-6. Immunizations during Pregnancy

VACCINE/ IMMUNOBIOLOGIC AGENT	TYPE OF VACCINE	ISSUES IN PREGNANCY
Measles-mumps-rubella (MMR)	Live attenuated	Contraindicated Delay pregnancy for 1 mo after MMR is given If exposed, check titer if immunity unknown May give immune globulin for exposure
Polio	Inactivated virus	Use if indicated
Varicella	Live attenuated	Contraindicated in pregnancy Check titer if exposed to varicella during pregnancy; give varicella zoster immune globulin if not immune by history or titer
Tetanus-diphtheria	Combined toxoid	Safe in pregnancy
Influenza	Inactivated vaccine	Recommended for all pregnant women in 2nd and 3rd trimester Indicated for pregnant women in any trimester if history of high-risk medical condition
Pneumococcal	Polysaccharide	Vaccine used only in high-risk pregnancies After splenectomy
Meningococcal	Polysaccharide	Administer for high-risk exposure
Typhoid (Ty21a)	Live-attenuated bacterial, oral	No data in pregnancy Avoid in pregnancy on theoretical grounds (live vaccine)
Typhoid Vi	Vi capsular polysaccharide, parental	No data in pregnancy Use only if clearly indicated
Hepatitis A	Formalin-inactivated vaccine	Use if clearly indicated **Risk for complications of hepatitis A increased in pregnancy** Consider checking titer if prior exposure
Hepatitis B	Recombinant or plasma derived	Pre-exposure and postexposure prophylaxis indicated in pregnant women at risk for infection
Yellow fever	Live attenuated	Indicated if exposure unavoidable Give letter of waiver for travel to low-risk area
Japanese encephalitis (JE)	Inactivated virus	Data on safety in pregnancy not available. The theoretical risk of vaccination should be weighed against risk for disease. JE virus infection acquired during 1st or 2nd trimester of pregnancy may result in uterine infection and fetal mortality
Tick-borne encephalitis	Inactivated	Not recommended in pregnancy Practice strict tick bite precautions
Rabies	Killed virus Human diploid cell rabies vaccine (HDCRV) or rabies vaccine adsorbed (RVA)	Postexposure prophylaxis is indicated during pregnancy Pre-exposure prophylaxis only when substantial risk for exposure exists
Immune globulins (IGs) Pooled or hyperimmune	IGs or specific antitoxic serum including antivenom for snakebite, spider bite, diphtheria antitoxin, hepatitis B IG, rabies IG, tetanus IG, varicella zoster IG	Give appropriate IG or antitoxin as indicated for exposure
Cholera	Killed oral cholera toxin B subunit whole cell (BS-WC) Live-attenuated oral cholera vaccine (CVD 103 HgR strain)	No live organisms. May be efficacious in high-risk situations but not recommended during pregnancy at this time. Not available in U.S. Not recommended in pregnancy Not available in U.S. Available in Canada and Europe

Adapted from Centers for Disease Control and Prevention: Health Information for International Travel 2005–2006. Atlanta, US Department of Health and Human Services, Public Health Service, 2005, pp. 494–495.

containing three elements: microfiltration, iodine resin, and activated charcoal. Meat should be well cooked and all dairy products (including cheeses) pasteurized to decrease the risk for toxoplasmosis, listeriosis, and other food-borne pathogens.

Treatment of GI illness during pregnancy should emphasize oral rehydration. Pharmacologic measures are limited. Products containing bismuth and salicylate (Pepto-Bismol has both) carry the risk for congenital malformations (bismuth) and fetal bleeding (salicylate). To control frequency of bowel movements, loperamide and diphenoxylate are two antimotility drugs (category B) that may be used with severe diarrhea. Antimicrobial choices are limited. Fluoroquinolones (category C) are not recommended, although data from inadvertent exposure during pregnancy do not show association with adverse outcomes. Adverse effects have been shown in animals but not in humans. Azithromycin (FDA category B) is safe in pregnancy. Cefixime and other cephalosporins are safe during pregnancy, but effectiveness is unclear.

Box 88-6. U.S. Food and Drug Administration Use-in-Pregnancy Classifications

Category A: Adequate and well-controlled studies in women show no risk to the fetus.
Category B: No evidence of risk in humans. Either studies in animals show risk, but human findings do not, or, in the absence of human studies, animal findings are negative.
Category C: Risk cannot be ruled out. No adequate and well-controlled studies in humans, or animal studies are either positive for fetal risk or lacking as well. Drugs should be given only if the potential benefit justifies the potential risk to the fetus.
Category D: There is positive evidence of human fetal risk. Nevertheless, potential benefits may outweigh the potential risks.
Category X: Contraindicated in pregnancy. Studies in animals or humans or investigations or postmarketing reports have shown fetal risk that far outweighs any potential benefit to the patient.

Vector-borne Disease: Malaria

Women are more susceptible to malaria during pregnancy and the postpartum period.[49,110] Pregnancy increases the clinical severity of *P. falciparum* malaria in women, both with and without existing immunity.[128] There is preferential sequestration of parasitized red blood cells in the placenta along with suppression of selected components of the immune system during pregnancy. This can result in IUGR, premature delivery, anemia, fetal loss, maternal death, or congenital malaria. Maternal and perinatal mortality are markedly increased with infection.

Pregnant wilderness travelers need to scrutinize their itinerary for increased risk to themselves and their fetus. If a woman is pregnant or plans to become pregnant and cannot defer travel to a high-risk area, appropriate chemoprophylaxis and maximal personal protective measures are essential.[38,159]

Mefloquine-resistant strains of *P. falciparum* are increasing worldwide. In these areas, there is no ideal safe option for prophylaxis at this time, and travel should be strongly discouraged.

Personal Protective Measures

Pregnant women should use a combination of physical and chemical barriers (Fig. 88-5). Permethrin (or deltamethrin) to treat clothing and diethyltoluamide (DEET) in a concentration of 30% to 35% can be used on the exposed skin. DEET crosses the placenta in small amounts but has not been associated with adverse effects in the fetus when used as directed.[112] Clothing that has already been treated with permethrin is also available.

Pregnancy may lead to "increased attractiveness" to mosquitoes. This may relate to physiologic changes during pregnancy, such as greater body heat and surface area, that promote a greater host signal for the mosquito, and to human behavioral factors, such as having to urinate more outside the tent, which may give mosquitoes a greater chance to attack.[106]

Chemoprophylaxis

Options for chemoprophylaxis and their uses and contraindications during pregnancy and lactation are listed in Table 88-7. For travel to chloroquine-sensitive malaria areas, chloroquine can be prescribed. Chloroquine has been used for decades for the prophylaxis and treatment of chloroquine-sensitive malaria without adverse fetal or maternal effects. For travel to chloroquine-resistant *P. falciparum* areas, travel should be deferred during pregnancy if possible. If travel is unavoidable, mefloquine is the only available antimalarial currently recommended. Limited studies have not found an association of use with increased rates of stillbirth or congential malformations when used in the second and third trimester. Postmarketing surveillance suggests that administration in the first trimester is also safe.[166] Doxycycline and primaquine are contraindicated in pregnancy. Atovaquone proguanil is not currently recommended for prophylaxis during pregnancy owing to insufficient data. If a pregnant traveler is unable to take mefloquine chemoprophylaxis, the CDC Malaria Hotline (770-488-7788) should be consulted for guidance. Malaria during pregnancy can lead to serious morbidity and mortality for the infant and the mother. Each pregnant woman should carefully consider how she might feel if she acquired a severe case of malaria that resulted in a poor fetal outcome.

Sports and Wilderness Adventure Risks

Exercise during Pregnancy

Exercise during pregnancy is recommended for most women.[9,59] Potential benefits and possible risks of particular exercises have been reviewed in the literature.[10,19,39] Unless there are other medical or pregnancy-related contraindications (e.g., risk for premature labor, incompetent cervix, multiple gestation, bleeding), exercise and recreational training should be encouraged throughout pregnancy, taking precautions to avoid prolonged exposure to extremes of temperature, dehydration, hypoglycemia, prolonged anaerobic conditions, and excessive skeletal stress.[9,99]

Even well-conditioned athletes experience limits on strenuous activity due to hemodynamic changes of pregnancy. Exceeding this limit could have deleterious effects on fetal status because of decreased uterine perfusion, increased uterine contractions, maternal acidosis, and hypoglycemia. Mild to moderate exercise is not associated with increased pregnancy loss. Running and other endurance sports may divert blood from the uterus and should be done with care as pregnancy progresses.

The American College of Obstetricians and Gynecologists[9] provides the following recommendations for exercise during pregnancy:

- Maternal heart rate less than 140 beats/min during exercise
- Strenuous activity for less than 15 minutes
- Avoid core temperature higher than 38° C (100.4° F)
- No exercise in the supine position after the fourth month

Altitude

Although exercise during pregnancy has been shown to be safe and is now recommended for all healthy women,[9] little is known about the combined effects of high altitude and exercise during pregnancy. Owing to a paucity of experimental data, recommendations regarding exercise during pregnancy at elevations higher than 1600 m (5200 ft) must be based on synthesis of available information regarding independent effects of high altitude and exercise on uteroplacental oxygen delivery.[56]

TABLE 88-7. Medication Use during Pregnancy and Lactation

MEDICATION	CATEGORY	ISSUES DURING PREGNANCY	ISSUES DURING LACTATION
Analgesics/antipyretics		Try nonpharmaceutical methods first to treat pain such as rest, heat, massage	
Acetaminophen	B	Safe in low doses short term	Compatible
Aspirin	C/D	Avoid first and last trimester. Has been associated with premature closure of ductus and excessive bleeding. Low-dose aspirin (60–80 mg) may be used for preeclampsia	Use caution
Nonsteroidal anti-inflammatory (ibuprofen, naproxen)	B/D	Should not be used in first and last trimester owing to effects on premature closure of ductus and effects on clotting. Not teratogenic	Safe
Codeine	C/D	Use cautiously. May cause respiratory depression and withdrawal symptoms in fetus if used near term	Compatible
Hydrocodone	C	Use cautiously. May cause respiratory depression in infant if used near term	Use caution. Probably compatible
Antibiotics for URI, UTI, GI, Skin, Other		Use antibiotics only if strong evidence of bacterial infection	
Amoxicillin, amoxicillin + clavulanic acid (Augmentin) Amoxicillin + sulbactam (Unasyn)	B	Safe. Use for treatment of otitis media, sinusitis, strep throat	Safe
Azithromycin	B	Safe. Use for bronchitis, pneumonia, *Campylobacter, Shigella, Salmonella, Escherichia coli*	Safe
Cephalosporins	B	Safe. Use for otitis, streptococcal infections, sinusitis, pharyngitis	Safe. Can be used to treat mastitis
Clindamycin PO or clindamycin vaginal cream	B	Safe. Treat bacterial vaginosis (BV) orally or locally in 2nd or 3rd trimester. Avoid 1st trimester	Safe
Ciprofloxacin, other quinolones	C	Controversial. Sometimes used short term in severe infections and/or long term in life-threatening infections (e.g., anthrax). May be used if potential benefit justifies risk to fetus	Safe
Dicloxacillin	B	Safe. Used for skin infections	Safe. Used to treat mastitis
Doxycycline, tetracycline	D	May cause permanent discoloration of the teeth during tooth development, including the last half of pregnancy, infancy, and childhood to the age of 8 years	Avoid
Erythromycin (base or state)	B	Safe. Treatment of bacterial causes of URI	Safe
Nitrofurantoin	B	Drug of choice for UTI in pregnancy	Safe
Penicillin	B	Safe	Safe
Sulfonamides	C/D	Safe. Not recommended 3rd trimester owing to risk for hyperbilirubinemia	Avoid owing to risk for kernicterus
Trimethoprim	C	Safe	Safe
Gastrointestinal			
Antidiarrheal		Replace fluids	
Atropine sulfate diphenoxylate hydrochloride (Lomotil)	C	Avoid during pregnancy	Avoid
Loperamide (Imodium)	B	Use if severe symptoms	Compatible
Antiemetics for nausea, heartburn, esophageal reflux		Encourage supportive measures first rather than medications: crackers upon arising, frequent small meals, protein meal at bedtime	
Antacids	B	May use sparingly for symptoms as needed	Safe
Bismuth subsalicylate (Pepto Bismol)	D	Avoid. Contains salicylate	Avoid
Cimetidine, ranitidine, omeprazole	B	Safe. Study during the 1st trimester found not associated with an increase in congenital malformations.	Safe
Metoclopramide (Reglan)	B	Safe in small doses	Safe
Dimenhydrinate (Dramamine)	B	Safe for severe nausea	Safe
Phenothiazines (Compazine)	C	Rare cases of congenital malformations have occurred after use during pregnancy	Avoid

TABLE 88-7. Medication Use during Pregnancy and Lactation—cont'd

MEDICATION	CATEGORY	ISSUES DURING PREGNANCY	ISSUES DURING LACTATION
Promethazine (Phenergan)	C	Used for nausea	Probably compatible
Acupressure (Sea Bands)		Safe	Safe
Emetrol (fluid replacement)	B	Safe. Oral solution	Safe
Ginger	B	Safe	Safe
Meclizine	B	Safe for treatment of severe nausea and vomiting	Safe
Pyridoxine (B_6)	A	Safe. Used for nausea	safe
Constipation		Increase fiber + fluid in diet first	
Bisacodyl	B	Safe to use occasionally	Safe
Milk of magnesia	B	Safe in small amounts	Safe
Psyllium hydrophilic mucilloid	B	Safe	Safe
Hemorrhoids		Increase fiber + fluid in diet	
Anusol HC suppositories	B	Safe	Safe
URI, Congestion, Cough		Symptomatic Rx: steam, rest, fluids	
Antihistamines			
Chlorpheniramine	B	Use cautiously for severe symptoms	Unknown
Cetirizine (Zyrtec)	B	Safe Nonsedating. Use cautiously	Unknown
Diphenhydramine (Benadryl)	B	Safe. Use cautiously	Avoid
Loratadine (Claritin)	B	Safe. Nonsedating. Use cautiously	Unknown
Dextromethorphan	C	Probably safe. Use in small amounts	Unknown
Guaifenesin	C	Probably safe. Use only if needed	Unknown
Pseudoephedrine (Sudafed)	C	Avoid 1st trimester. Use cautiously	Unknown
Saline nasal spray	A	Safe	Safe
Topical nasal decongestants Oxymetazoline (Afrin)	C	Safe. Do not use for more then 3 days	Safe
Asthma, Allergy			
Inhaled bronchodilators	C	Safe for use of wheezing during pregnancy	Unknown
Inhaled steroids	C	Use if indicated	Safe
Nasal steroids	C	Use if indicated	Safe
Antimalarials			
Mefloquine (Larium)	C	Avoid during 1st trimester unless unavoidable travel to high-risk area. Safe in 2nd and 3rd trimester for high-risk travel	Excreted in breast milk Infant still needs own chemoprophylaxis
Chloroquine	C	Avoid in 1st trimester unless travel to high risk area	Safe. Excreted in milk in small amounts. Infant still needs chemoprophylaxis
Atovaquone, proguanil (Malarone)	C	Avoid in 1st trimester. Not recommended for prophylaxis at this time due to insufficient data. Used for treatment of malaria	Safe if infant is >11 kg (24 lbs) or if benefit for mother outweighs possible risk
Doxycycline	D	Contraindicated for malaria prophylaxis. May be considered for treatment of severe infections	Avoid
Primaquine	C	Do not administer during pregnancy because of the possibility the fetus may be G6PD deficient. If a causal cure with primaquine is indicated, continue to suppress with chloroquine (or other chemoprophylaxis) until delivery	Avoid
Proguanil	C	Not associated with teratogenicity. Not effective as single agent	Probably compatible
Insect Repellent			
DEET		Safe. Use sparingly as directed	Compatible
Antiparasitics			
Albendazole	C	Teratogenic in animal studies. Avoid during 1st trimester. Treat after delivery if possible. May be indicated for serious infections	Probably compatible
Metronidazole	B	Contraindicated during 1st trimester. Use in 2nd and 3rd trimesters only if clearly indicated	Single dose: hold breastfeeding 12–24 hrs. Use caution

Continued

TABLE 88-7. Medication Use during Pregnancy and Lactation—cont'd

MEDICATION	CATEGORY	ISSUES DURING PREGNANCY	ISSUES DURING LACTATION
Antivirals			
Acyclovir	B	Use when indicated	Compatible
Altitude Sickness			
Acetazolamide (Diamox)	C	Do not use during 1st trimester. Use only if benefit outweighs risk	Compatible
Dexamethasone (Decadron)	C	May use if needed for treatment for altitude illness	Avoid breastfeeding during use
Calcium channel blockers (Nifedipine)	C	Use only to treat severe symptoms pulmonary edema	Probably compatible
Water Disinfection		Boil water, use filters	
Iodine	D	Avoid. May lead to goiter and fetal hypothyroidism	Avoid

GI, gastrointestinal; URI, upper respiratory infections; UTI, urinary tract infection.
Data from Briggs G, Freeman R, Yaffe S: Drugs in Pregnancy and Lactation. Baltimore: Lippincott Williams & Wilkins, 2005; Micromedex Online; American Academy of Pediatrics, 2005; and Lexi-Comp Online UptoDate.

Figure 88-5. Personal protective measures during pregnancy.

Short-term travel to moderate altitude does not appear to have negative effects on pregnancy. For women living at altitude, pregnancy-induced hypertension and preeclampsia are more common. Infant birth weights are lower at altitude compared with infants born at sea level. Chronic placental changes occur at altitude but have not been seen or conclusively studied in the short-term traveler. Low oxygen tension and pressure changes result in IUGR and increased risk for premature labor in women who spend most of their pregnancy above 2500 m (8200 ft).[8]

Maternal hyperventilation and elevated blood hemoglobin concentration maintain resting arterial oxygen content at or above sea-level values. During exercise, however, arterial hemoglobin saturation falls, lowering oxygen content. Resting uterine artery blood flow is lower in residents at 3100 m (10,000 ft) than at 1600 m (5200 ft), and flow is likely to decrease further during exercise in proportion to intensity and duration. The combined reduction in arterial oxygen content and uterine artery flow suggest that fetal oxygen delivery is compromised during exercise at altitude.[56]

Studies done on pregnancy at high altitude have demonstrated that chronic hypoxia plays a key role in the etiology of IUGR and preeclampsia. Recently, genetic factors also have been elicited that may relate to underlying susceptibility to complications of pregnancy and fetal life.[121]

Altitude alone does not determine whether the fetus becomes stressed. The maternal (and fetal) arterial oxygen pressure and arterial oxygen saturation are critical. Altitude illness, especially pulmonary edema, must be avoided. Lung disease, smoking, and other disorders of oxygen transport can render the pregnant patient at altitude more hypoxemic and at a higher physiologic altitude.

A Medical Commission from the International Mountaineering and Climbing Federation recently reviewed the research to date on women and altitude and published a consensus paper.[94] The following recommendations were adapted from that paper:

Recommendations for Pregnant Women Going to Altitude

First trimester: Short stays (hours to days) at altitudes up to 2500 m (8200 ft) without heavy exercise

- Healthy pregnant women in first trimester with good access to care may go to altitudes of 1600 m (5200 ft) to 2500 m (8200 ft). Plan 2 to 3 days to acclimatize.
- Women who are at an increased risk for spontaneous abortion should avoid altitude exposure during the first trimester.
- Women with risk factors for preeclampsia or placental abruption or whose babies are at risk for IUGR should not go to high altitude, even for short stays.

Later trimesters: Short stays (hours to days) at altitudes up to 2500 m (8200 ft) without heavy exercise pose little risk to pregnancy or of fetal complications during the second half of pregnancy.

Contraindications for going to altitude after 20 weeks of pregnancy include the following:

- Chronic hypertension or other factors that increase the risk for preeclampsia
- Preeclampsia
- Impaired placental function (e.g., ultrasound diagnosis of partial abruption, clots)
- IUGR
- Maternal heart or lung disease
- Anemia
- Smoking

Any trimester: Exercise

- Mild to moderate exercise: Plan at least 2 to 3 days for acclimatization before exercising at altitude over 2500 m (8200 ft).
- Strenuous exercise: Plan at least 2 weeks for full acclimatization before strenuous exercise and avoid heavy exertion at higher altitudes.
- Most would advise against travel to altitude after 36 weeks because of lack of access to optimal care.

Any trimester: Longer stays at high altitude >2500 m (>8200 ft)

- Increased risk for preeclampsia, placental abruption, and IUGR.[101,121,176,178]
- All women should be monitored for signs and symptoms of preeclampsia, placental pathology, and fetal IUGR.
- Prenatal visits should include blood pressure monitoring, urine protein check, and Doppler ultrasound monitoring of the uterine artery for waveform and volumetric flow.
- For "prenatal self-checks," women should be taught how to take their own blood pressure, screen for protein and glucose in the urine, and assess for other early symptoms of preeclampsia.
- Women at risk should be identified and referred for advanced health care services immediately.
- Ideally, maternal artery and fetal umbilical arterial waveforms and growth after 20 weeks should be followed in high-risk women.

Acute mountain sickness

- The incidence of AMS does not differ between pregnant and nonpregnant women.[130]
- There are few data on treatment of AMS during pregnancy.
- Strict guidelines for acclimatization should be followed as outlined above.
- Acetazolamide and other sulfonamides are contraindicated during the first trimester owing to studies demonstrating teratogenicity in animals,[141,147] and are not recommended after 36 weeks owing to increased risk for neonatal jaundice.[31]
- If a pregnant woman has symptoms of AMS, the theoretical risk of medication must be weighed against symptoms.
- Descent and oxygen therapy are the preferred treatment.
- Use of acetazolamide or dexamethasone may be considered on an individual basis.

There are no studies on skiing while pregnant, although for a normal healthy pregnancy, there is probably little risk during the first trimester. Access to care is the most important issue in case of an emergency. Women skiing later in pregnancy are at higher risk owing to the increase in weight, change in the center of gravity, and increase in joint laxity that may predispose her to trauma leading to placental disruption or other devastating consequences. Short-term travel to a favorite mountain escape with adequate resources is probably safe for a pregnant adventurer with a normal pregnancy. More research is needed to evaluate the risks of intense exercise at altitude during pregnancy.[19,56,94,130]

Water Sports

Swimming and snorkeling are safe during pregnancy and considered to be an excellent form of exercise for pregnant women. Scuba diving is potentially hazardous. The fetus is at risk from nitrogen bubbles in the fetal-placental circulation during decompression on ascent. Most authorities consider pregnancy a contraindication to diving.[26,33,49]

Diving is compromised by increased abdominal girth, difficulty breathing due to engorgement of the mucous membranes of the nose and oropharynx, and increased buoyancy secondary to fat deposition. Higher levels of body fat also increase the risk for decompression sickness because nitrogen tends to be retained in these tissues. Dyspnea may be exaggerated and lead to panic, even in the experienced diver.

As with exertion at high altitude, the pregnant woman may have limited ability to maintain anaerobic metabolism for prolonged periods because of fetal needs. The pregnant woman should also limit prolonged nondiving immersion in cold water that might lead to hypoventilation and hypothermia. The effects of scuba diving on pregnancy were reviewed in detail.[33] There is no contraindication to diving for the nonpregnant woman. Advice for a woman who finds out she was pregnant during the time she was diving is to not terminate the pregnancy. There are case reports of normal pregnancies despite continued diving.

Water skiing, jet skiing, and other water sports that might force water up into the vagina and through the cervix, thereby causing a risk for miscarriage or peritonitis, are not recommended during pregnancy.[108]

Heat

Safe limits for exposure to heat during pregnancy have not been established. Acclimation to environments characterized by high temperatures, particularly with high humidity, may be especially difficult for the pregnant woman and may pose a risk to the fetus. Hyperthermia has been shown to be teratogenic in various animal models. A higher incidence of birth defects, particularly neural tube defects, has been found among offspring of women who experienced hyperthermia by environmental exposure or febrile illness in the first trimester.[115] Later in pregnancy, the fetus depends on the woman to eliminate excess heat. Elevation of ambient temperature decreases the heat gradient and ability of the pregnant woman to dissipate heat. Elevated humidity exacerbates this situation by decreasing the contribution of perspiration to heat loss. This increases risk for elevated maternal core temperature, which further raises fetal metabolic activity and heat generation. Hyperthermia increases the risk for premature labor, particularly with dehydration and loss of electrolytes. Fetal stress, resulting from decreased uterine perfusion secondary to compensatory peripheral vasodilation and depletion of intravascular volume, further increases the likelihood of preterm labor. Pregnant women exposed to hot climates should pay particular attention to hydration.

Data regarding the effects of exercise on core temperature are limited. To date, hyperthermia associated with exercise has not been found to be teratogenic in humans. However, it is recommended that pregnant women avoid hot tubs and saunas.[158]

Exposure to Venomous Animal Bites and Stings

Venomous animal bites and stings during pregnancy may have serious effects on the fetus and mother. A recent review of the literature[104a] demonstrated that the occurrence of fetal death is 30% to 40% in snakebite cases in pregnant women reported since 1966. There have been no formal epidemiologic studies on the effect of antivenom on fetal development. Although a large percentage of fetal deaths occurred in the mothers who received antivenom, the deaths may have been related to severity of bite as opposed to therapy. It is thought in general that the risks of significant venom poisoning to the mother outweigh any theoretical concerns about safety of the antivenom to the unborn child.

Envenomation during pregnancy should be reported to the poison control center and to the pharmaceutical company that produces the venom, so that information on medical management and fetal outcomes can be evaluated and lead to evidence-based recommendations. More research is also needed to evaluate the risk-to-benefit profiles of snake, spider, and scorpion antivenoms on pregnant women, embryos, and fetuses.

REMOTE WILDERNESS TRAVEL DURING PREGNANCY

Prolonged wilderness excursions during pregnancy, particularly those extending into late second and third trimesters, should include preparations for ongoing assessment and emergency delivery.

Routine antepartum care usually includes visits to a health care provider at least monthly until 26 to 28 weeks, every 2 weeks thereafter until 36 weeks, then weekly until delivery. Beyond the due date, more frequent visits are often recommended. Routine visits focus on uterine activity, vaginal discharge, abdominopelvic pain, bleeding, headaches, symptoms of UTI, current medications, and fetal activity.

The basic antepartum examination includes measurements of blood pressure, weight, and uterine size (height of uterine fundus above symphysis) and subjective assessments of peripheral edema and reflexes. Urine is tested at each visit with a multitest strip that provides estimates of glycosuria, proteinuria, pH, and presence of nitrites. For the wilderness traveler, elevated pH and presence of nitrites may be associated with UTI. In Rh-negative women with Rh-positive partners, the antibody screen is often repeated at 26 to 28 weeks, before administration of Rh immune globulin for prophylaxis against third-trimester sensitization. If an antibody screen cannot be done, Rh immune globulin should be administered empirically. Maternal hemoglobin and hematocrit are frequently done in each trimester to assess need for iron supplementation. Because many women require supplemental iron during pregnancy, this should be taken prophylactically if determination of hemoglobin cannot be done, unless the woman has a contraindication, such as hemochromatosis.

Box 88-7. Supplies for Management of Wilderness Delivery

STANDARD
Clean towels
Surgical sponges
Surgical gloves
Umbilical cord clamps
Suction bulb
Suture kit
Scalpel
Scissors
Syringes and needles
Local anesthetic
Injectable oxytocin
Injectable and oral methylergonovine
Oral analgesics (e.g., ibuprofen, acetaminophen with codeine)
Oral broad-spectrum antibiotic
Sanitary napkins

OPTIONAL
Injectable magnesium sulfate
Intravenous fluids and administration supplies
Injectable narcotic
Naloxone
Misoprostol
Prostaglandin F_{2a}
Injectable antibiotic
Neonatal mask and Ambu bag

Prenatal Care in the Wilderness

Basic supplies for the pregnant wilderness traveler might include a diary to record progress, reminders of scheduled testing, and complications; tape measure; stethoscope; sphygmomanometer; urine test strips; and prenatal vitamins and iron. Other supplies include a glucometer with test strips, calcium supplement, and basic medications for the most common pregnancy complaints: antiemetic suppository such as promethazine, prochlorperazine, or chlorpromazine for nausea and vomiting; acetaminophen for headaches and pain; stool softener for constipation; and antibiotics to cover UTIs and vaginitis. In addition to basic medical supplies, the pregnant woman should have changes in clothing size and possibly shoe size.

Ideally, delivery in the wilderness should not be planned. If delivery in the wilderness is a possibility, special preparations need to be made (Box 88-7), including plans for an emergency evacuation if needed.

Complications during Pregnancy

Miscarriage

Women who are pregnant or become pregnant for the first time while in the wilderness are at high risk for complications. About 15% to 25% of all pregnancies abort spontaneously during the first trimester, and this number may exceed 60% to 70% in the true primigravida.[143] Reasons may be related to immunologic naiveté to paternal antigens expressed by the fetal tissues. In contrast, isolated miscarriages in women who have successfully carried pregnancies are often the result of chromosomal abnormalities. Pending miscarriage in the first trimester is usually pre-

ceded by embryonic demise and accompanied by reduction or loss of early pregnancy-related symptoms, such as breast tenderness and nausea. Bleeding and uterine contractions eventually occur and accompany expulsion of the products of conception. Under most circumstances, hemorrhage during miscarriage is self-limited and not life-threatening but at times can be heavy, especially if fetal death does not precede the event and occurs late in the first trimester or during the second trimester, or if the expulsion process is prolonged or incomplete. Spontaneous abortion after the first trimester is much riskier but less common. It also can result from chromosomal abnormalities or fetal anomalies but is more likely to be caused by chorioamnionitis, UTI, severe abnormalities of placentation, poorly controlled maternal medical conditions, or cervical incompetence.

Any acute and significant blood loss in a physiologically hostile environment can compromise endurance of the most highly trained individual. Under wilderness conditions, control of significant maternal hemorrhage accompanying miscarriage may be difficult unless provisions, facilities, and medical supplies are available. Uterine curettage is the method most often used to complete evacuation of the uterus when medical facilities are available. Once empty, uterine involution, spontaneous or aided by uterine massage, is usually sufficient to impede bleeding from the implantation site. In the absence of the ability to perform curettage, treatment with methylergonovine, 0.2 mg orally or intramuscularly, can enhance uterine contractions, accelerate expulsion of products of conception, and promote uterine involution to maintain hemostasis while plans for evacuation are being made. Methylergonovine should not be used in patients with hypertensive disorders, underlying vascular disease, and certain cardiac abnormalities unless the benefits clearly outweigh the risks of acute generalized vasoconstriction. As an alternative, the prostaglandin F_{2a} drug carboprost tromethamine, 250 µg intramuscularly, or misoprostol, 100 µg orally or vaginally, can be administered to stop uterine bleeding with less risk for cardiovascular compromise.

Ectopic Pregnancy

Far more dangerous than miscarriage, ectopic pregnancy must always be considered a life-threatening emergency that requires immediate medical attention. Ectopic pregnancy refers to implantation at any location outside the uterine cavity, most often (more than 95%) within the fallopian tube. Hemorrhage resulting from ectopic pregnancy is still the leading cause of maternal death in the first trimester. The incidence of ectopic pregnancy has tripled over the past 30 years and now exceeds 1 in every 100 pregnancies.[134] This increase is directly proportional to the rise in incidence of acute and chronic PID. The most common predisposing risk factors include a history of infections, multiple sexual partners, early age of onset of sexual activity, delayed childbearing, and previous IUD use. Independent risk factors are a history of abdominal and tubal surgery, including previous tubal sterilization procedures, endometriosis, diethylstilbestrol (DES) exposure, and pregnancy by assisted reproductive interventions. Regardless of the etiology, a prior ectopic pregnancy increases the risk for another ectopic pregnancy about 10-fold. A woman with a history of ectopic pregnancy should not intentionally plan to conceive again in the wilderness and should have her intrauterine pregnancy confirmed sonographically before departure.

Most women with a tubal ectopic pregnancy become symptomatic before 12 weeks' gestation and present with complaints of abdominal pain and altered menses. Early pregnancy symptoms may be minimal or absent. The pain often begins unilaterally with sudden onset and is usually severe, distinguishable from the cramping, intermittent pain accompanying miscarriage by its persistence and intensity. Clinical findings include a tender adnexal mass, nontender cervix, small to slightly enlarged nontender uterus, and absence of high-grade fever. Low-grade temperature elevation to 38° C (100.4° F) may occur in as many as 20% of women with ectopic pregnancy. When intraperitoneal hemorrhage accompanies rupture of the tube, the pain becomes diffuse with peritoneal signs of tenderness, guarding, and rebound. Shoulder pain from diaphragmatic irritation may be present as well. Pelvic examination at this point usually elicits discomfort with movement of the cervix and uterus. Fullness of the cul de sac posterior to the uterus and abdominal distention suggest significant intraperitoneal blood loss.

Vaginal bleeding accompanying an ectopic pregnancy usually follows a variable period of amenorrhea. It often begins with minimal flow of blood darker than that seen during miscarriage and results from inadequate progestational support of the decidualized endometrium. Uterine cramping may ensue, with passage of organized clot and tissue in the form of a decidual cast resembling products of conception, leading to the mistaken diagnosis of spontaneous abortion. However, cessation of pain is typical with completion of miscarriage and quite atypical with passage of a decidual cast concurrent to an ectopic pregnancy. With rupture of an ectopic pregnancy, heavier, bright-red bleeding may occur vaginally and if accompanied by significant intraperitoneal hemorrhage, may rapidly result in hemodynamic decompensation that only can be controlled surgically. The differential diagnosis of ectopic pregnancy includes normal intrauterine pregnancy with a corpus luteal cyst or hemorrhagic corpus luteum, threatened or incomplete abortion, PID, adnexal torsion (usually associated with adnexal enlargement from a benign or neoplastic process), endometriosis, UTI or ureteral stone, degenerating fibroid, and appendicitis. Although simultaneous intrauterine and ectopic pregnancies were once considered extremely rare, the incidence is now estimated at 1 in 6000 in the general population and greater than 1 in 100 among recipients of assisted reproductive techniques. The diagnosis of ectopic pregnancy is considered presumptively in any woman with a positive pregnancy test, abnormal bleeding, and abdominal pain. In a full-service medical care facility, the first step in management is to assess serum hCG levels and ascertain location of the pregnancy. An intrauterine pregnancy can usually be confirmed by transvaginal sonography once the serum hCG exceeds 1000 mIU/mL, corresponding to 2 to 3 weeks after conception or 4 to 5 weeks from the last normal menstrual period. In contrast, absence of an intrauterine pregnancy at hCG levels of 1000 mIU/mL or greater suggests ectopic pregnancy. In a wilderness setting that precludes the ability to perform a sonogram and quantitative determinations of hCG, plans for evacuation must be made at first suspicion of the diagnosis.

Later Pregnancy Complications

Complications that cause bleeding at 20 weeks' gestation or later cannot be optimally managed in most wilderness settings (Box 88-8). Plans should be made for immediate evacuation. Second- and third-trimester bleeding could simply be the result of cervical effacement, labor, cervical polyps, coital trauma, or

Box 88-8. Later Complications of Pregnancy Requiring Evacuation

Placenta previa
Placental abruption
Preterm labor
Premature rupture of membranes
Chorioamnionitis
Preeclampsia, eclampsia, HELLP syndrome

HELLP, hemolysis, elevated liver function enzymes, and low platelets.

vaginitis, but it could also be much more serious if the source is placenta previa or placental abruption. Historical information and physical findings may suggest an etiology, but a definitive diagnosis is not possible without verification.

Placenta Previa. Placenta previa results from implantation of the placenta in the lower uterine segment over or near the internal cervical os. It occurs in about 1 of 200 births. Risk increases with age and parity, multiple gestations, women who have submucosal fibroids or have had multiple dilation and curettage procedures or cesarean births, and cigarette smokers.[4] The classic presentation of placenta previa is painless, sudden, heavy, and bright-red vaginal bleeding.[22] It may occur with exertional activity, straining on the toilet, or intercourse, but also occurs at rest with no obvious precipitating factor.

Placental Abruption. Placental abruption is defined as separation of the placenta from the maternal interface before delivery. One of the most common etiologies is trauma. Risk factors include hypertensive disorders (e.g., preeclampsia, chronic hypertension), other chronic diseases with vascular compromise (e.g., diabetes, renal disease, certain autoimmune disorders), coagulation disorders (presence of lupus anticoagulants or anticardiolipin antibodies, protein S or C deficiencies, factor V Leiden, antithrombin III deficiency), trauma, chorioamnionitis, advanced maternal age, and multiparity. Placental abruption is highly variable in presentation, depending on the location and the extent of the separation and hemorrhage.[116] About 80% of placental abruptions result in visible bleeding accompanying the onset of other symptoms. Unlike placenta previa, abruption is usually accompanied by sudden onset of sharp pain. The pain may be focal and continuous, intermittently intensifying with the frequent uterine contractions and irritability that usually accompany and can extend the placental separation.[22]

Premature Labor. Despite advances that have been made in obstetric and neonatal care in the past 50 years, rates of preterm delivery, defined as delivery before 37 completed weeks' gestation, have not changed, persisting in the range of 8% to 10% of all pregnancies in the United States.[47] It is still the leading cause of perinatal morbidity and mortality. Preterm labor, defined as regular uterine contractions resulting in progressive cervical change, as assessed by effacement, dilation, and softening, complicates twice as many pregnancies as actually progress to preterm delivery. Symptoms of preterm labor include mild and menstrual-like to painful uterine contractions, intermittent low back pain or pressure, pelvic pressure, increase in vaginal discharge resulting from effacement with compression of endocervical glands or leaking of amniotic fluid, and bloody "show." The more common risk factors for preterm labor and delivery include premature rupture of membranes, subclinical or overt chorioamnionitis, UTI, preeclampsia, multiple gestation, hydramnios, dehydration, constipation, chronic stress, and incompetent cervix.[47,113] The pregnant wilderness traveler is at risk for several of these factors and should strive to reduce their occurrence.

Thorough evaluation and management of preterm labor cannot be done in most wilderness settings. Some empiric measures can be taken, however, based on the woman's symptoms and palpation of uterine contractions alone, while awaiting evacuation or to prepare for delivery if evacuation is delayed or impossible. Pelvic examination should be avoided unless placenta previa has been previously excluded and sterile supplies are available. If done by an experienced person, cervical examination should determine dilation, effacement, and consistency, as well as the station and presentation of the baby. Once it has been concluded that the woman is in preterm labor and evacuation is required, examinations should not be repeated unless delivery appears imminent. If she clearly has ruptured membranes, characteristics of the fluid should be noted (clear, bloody, meconium stained), but no internal vaginal examination should be performed initially to minimize risk for introducing infection. Plans should be made for evacuation at any initial signs of premature labor. If premature labor cannot be stopped before evacuation, guidelines exist for management of delivery, as detailed later.

Preeclampsia. The diagnosis of preeclampsia is based on the triad of hypertension, proteinuria, and edema.[48] Hypertension is defined as systolic blood pressure of 140 mm Hg or higher or diastolic blood pressure of 90 mm Hg or higher measured on two separate occasions 6 or more hours apart. Proteinuria is defined as 0.3 grams or more of protein in a 24-hour urine collection, which usually corresponds with it or greater on a urine dipstick test. Edema is considered significant to the diagnosis of preeclampsia only if it generalized or if the woman has had a sudden weight gain of 5 pounds (2.27 kg) or more in a week. The strict diagnosis of preeclampsia requires the presence of hypertension with proteinuria, edema, or both. Women meeting these criteria have at least mild preeclampsia. If preeclampsia is suspected, plans should be made for immediate evacuation from the wilderness setting.

Diagnostic criteria for severe preeclampsia require only one of the following: blood pressure of 160 mm Hg systolic or higher or 110 mm Hg diastolic or higher on two occasions at least 6 hours apart; proteinuria (5 g/24 hours or higher); oliguria (400 mL/24 hours or higher); persistent epigastric pain; pulmonary edema or cyanosis; impaired liver function of unclear etiology; thrombocytopenia (<100,000/mm^3); and eclampsia (grand mal seizures). Most cases of severe preeclampsia are associated with IUGR or abnormalities of fetal umbilical (increased resistance) and middle cerebral (decreased resistance) arterial flow consistent with relative placental insufficiency. Eclampsia as a subset of severe preeclampsia is a major cause of maternal and fetal morbidity and mortality worldwide, occurring in 1 of 2000 pregnancies in the United States. Although difficult to anticipate, the presence of visual disturbances, severe headache, irritability, epigastric or right upper quadrant pain, nausea and vomiting, and cerebral dysfunction must be considered predictors of eclampsia. Thus, if a woman has early signs of preeclampsia, she should be evacuated because she may progress to severe eclampsia with high morbidity and possible mortality.

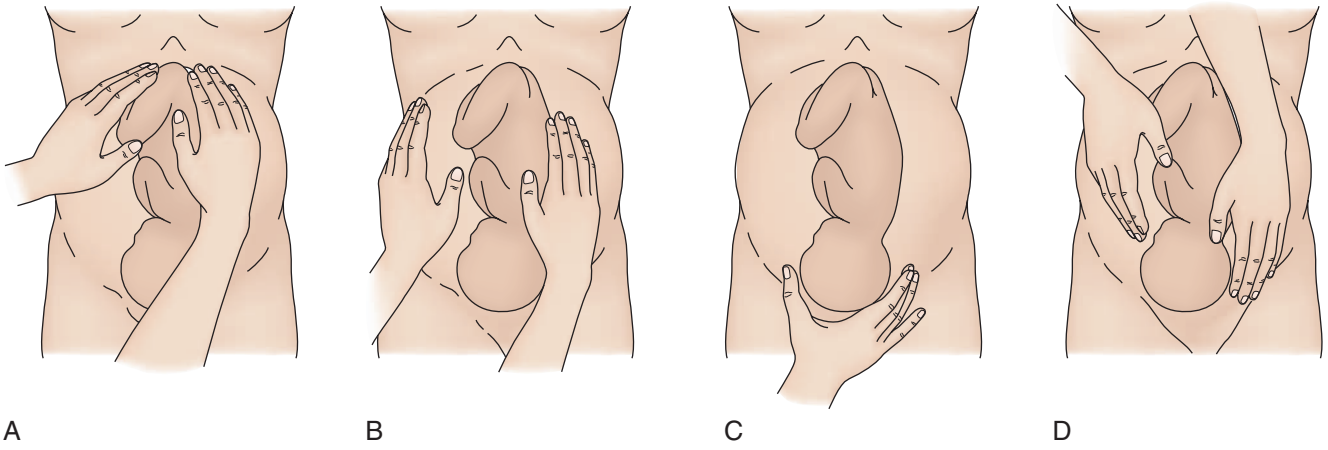

Figure 88-6. Ascertaining fetal position by Leopold's maneuvers. **A,** First maneuver. Assess part of fetus in upper uterus. **B,** Ascertain location of fetal back. **C,** Identify presenting part. **D,** Determine descent of presenting part.

Emergency Delivery

Although a delivery should never be planned for a wilderness expedition, unexpected delivery may occur. A pregnant woman planning a wilderness experience of any duration that will carry beyond 20 weeks' gestation should include emergency provisions and plans. The location, duration of stay, and distance dictate the extent of these plans related to medical care facilities, convenience of evacuation routes, and ease of communication with and availability of evacuation support. Any woman planning a wilderness excursion should review detailed emergency plans with her physician and her expedition partners. Some of the considerations are briefly reviewed below.

If a woman goes into labor unexpectedly on a trip, either a health care provider or the person with the most childbirth experience who is willing to assist in the delivery should be identified as team leader and "midwife." Participatory roles for other members of the party should be defined in cooperation with the pregnant woman. Requests for privacy and intimacy should be respected to the extent possible.

By necessity and practicality, delivery in the wilderness dictates a laissez-faire approach. Excessive intervention (e.g., repeated cervical examinations, artificial rupture of membranes, augmentation of uterine contractions by oxytocin or nipple stimulation, manual cervical dilation) is neither warranted nor appropriate because delivery cannot be expedited for concerns of fetal distress, and such intervention may increase maternal and fetal risks. To the extent possible, a clean, comfortable, and quiet site is prepared for the delivery. If clean and sterile supplies and medications are available, these should be brought to this location and inventoried by the team leader (see Box 88-7). Otherwise, clean towels, clothing, bedding, soap, and water should be made readily accessible.

Fetal position should be determined. In late third-trimester pregnancies, this can be accomplished by external abdominal palpation using Leopold's maneuvers (Fig. 88-6). In preterm pregnancies, this may first require internal digital examination, but fetal position is extremely important because the risk for malpresentation (breech, transverse, or compound lie) is inversely proportional to gestational age, as is the disparity between fetal head and abdominal circumferences. At term, the incidence of breech presentation is about 3%, whereas it may exceed 25% before 30 weeks' gestation. If the woman reports fetal activity, or if this is visible or palpable on abdominal examination, evaluation of fetal heart activity is usually unnecessary. If the woman or the examiner cannot detect fetal activity, and because fetal activity frequently diminishes with onset of labor, viability is assessed by auscultation. A stethoscope or the ear can be positioned over the location of the baby's back and shoulder. A stethoscope bell placed on the abdomen with minimal pressure is usually more sensitive than the diaphragm. Normal fetal heart rate at term usually falls to between 120 and 160 beats per minute. Under most wilderness situations, once viability is confirmed, further auscultation is probably unnecessary and may even provoke anxiety because of the inherent difficulties of fetal heart rate detection with unamplified methods, subtle changes in fetal position, descent of the presenting part, and the increasing discomfort of labor as it progresses.

In the early stages of labor or with spontaneous rupture of membranes in the absence of regular uterine contractions, rest interspersed with brief periods of ambulation, fluid intake, and frequent light meals should be encouraged. Prophylactic antibiotics should be started, especially when there has been premature rupture of membranes. Antibiotic coverage is the same as that recommended for preterm labor. Digital cervical examination is usually not necessary at this point and is contraindicated with rupture. As labor becomes more active, as gauged by increased frequency, regularity, and strength of uterine contractions, pelvic pressure, and discomfort level, the safest approach is to limit oral intake to clear liquids only. The GI tract becomes quiescent with active labor, and any stomach contents present at the outset will likely be present at the end. Because vomiting is not unusual, especially during the "transition" phase of labor, clear liquids minimize discomfort and decrease risk for aspiration. Intermittent ambulation may also decrease the discomfort associated with contractions and can be continued, if the woman desires, until she feels the need to push. During this time, she should also be reminded to empty her bladder because she may not be able to differentiate the sensation to void from that of pressure from the presenting fetal part. A full bladder not only adds to the discomfort of labor but also can impede descent of the baby into the pelvis, prolonging the process.

Although some women become irritable as labor intensifies before complete dilation and do not want to be touched, others appreciate low-back or extremity massage between contrac-

tions. Assistance with breathing and relaxation techniques to distract, maintain composure, and preserve the energy that will be required during the second stage of labor is also beneficial. During labor, no oral pain medication should be given, and parenteral narcotics, although acceptable, should be administered sparingly (ideally, intravenously with the onset of a contraction) unless naloxone is available to manage the fetal depression that may result.

When the woman begins to feel involuntary efforts to push with contractions, cervical examination should be performed with a clean or sterile glove or freshly washed hands. At the same time that cervical dilation and effacement are assessed, the presenting fetal body part should be identified, determining its station in relation to the ischial spines in midpelvis. If the cervix is completely dilated and effaced so that no cervical tissue is palpable between the presenting part and the vaginal wall, the first stage of labor is complete, and the woman can begin pushing with contractions. If the cervix is not completely dilated, the woman should be encouraged not to push with contractions so that she does not become exhausted. She also risks entrapping the cervix between the presenting part and the pelvis, which can lead to cervical edema and thickening, although this usually results from cephalopelvic disproportion. If membranes have ruptured, presence or absence of meconium should be noted. If membranes have not ruptured, they should not be ruptured intentionally, particularly if the baby is premature or in a breech presentation.

Usually, once the cervix is completely dilated, the desire to push is involuntary, and pain is less of an issue until the moment of delivery. Pushing is done only with uterine contractions. At the onset of a contraction, the woman takes in a deep breath, then exhales. Then she takes in and holds another deep breath, bearing down without releasing air as if straining to have a bowel movement. Most contractions are long enough to permit two or three attempts at this maneuver. Proper pushing is evident by expansion of the introitus and rectum during the effort and should not be accompanied by tensing of the extremities. Once the contraction is over, she should expel any held air and begin restful breathing, trying to relax completely to conserve energy and recover for the next contraction. The woman may push in any position in which she feels comfortable. However, she should avoid lying flat on her back because uterine compression of the inferior vena cava can lead to hypotension and decreased uterine perfusion. Common positions include semirecumbent, with back and head elevated and legs drawn up or supported at the knees during contractions; lateral recumbent, with superior leg flexed and supported during contractions; squatting; sitting; kneeling on all fours; and standing while being supported from behind around the torso. These positions can also be used for the actual delivery, as long as the attendant has adequate access. Once the presenting part reaches, distends, and remains at the vaginal introitus between contractions, final preparations are made for the delivery. A delivery position should be selected that allows control of the presenting part, protection of the perineum, and room to accomplish completion of the birth with as little trauma to the baby as possible. Delivery should be performed during a contraction.

Vertex Delivery

The most common fetal presentation is the cephalic (or vertex) presentation, with the fetal head facing the perineum (occiput anterior) (Fig. 88-7). When the perineum begins to distend with a contraction, the woman should be instructed to bear down. Intentional cutting of an episiotomy in a wilderness setting is not recommended. Spontaneous lacerations are more likely to occur along tissue planes that are less vascular and less likely to extend into the rectum. The perineum should be supported between the rectum and the introitus by the index finger and thumb of the nondominant hand while the fetal head is maintained in flexion until the crown has just begun to clear the symphysis. The woman stops pushing while the attendant exerts steady inward and upward pressure at the perineum against the fetal chin, thereby extending the head and completing its delivery while protecting the perineum. Once delivered, the fetal head will usually rotate laterally to align itself with the shoulders. The infant's mouth and nose should be cleaned by bulb aspiration or simple swabbing with a clean gauze or cloth. This step is especially important when meconium is present to prevent aspiration of this fluid when the baby is free to take its first breaths. Once the oropharynx is cleaned, the fetal neck should be palpated to ascertain the presence of a nuchal cord. If present, one or more loops of umbilical cord are often loose enough to be slipped over the baby's head before completion of the delivery. If they cannot be slipped over the head but are not tight, the baby can frequently be delivered through the loops. If the cord is tightly applied around the neck, the attendant should doubly clamp or tie a section of one loop, cut between the clamps, and then deliver the baby.

In the final stages of delivery, the woman resumes pushing while steady downward (toward the maternal sacrum) traction is applied with hands cupping both sides of the fetal head. When the anterior shoulder has cleared the symphysis, the perineum should again be supported while the head is elevated and the posterior shoulder delivered. The rest of the baby's body usually follows without effort. The baby is held below the perineum (to prevent loss of blood to the placenta from the baby) while the oropharynx is again cleaned and the baby dried. Usually, rubbing the baby dry is sufficient to stimulate breathing and crying. The umbilical cord should then be doubly clamped or tied and then severed. The baby should be thoroughly dried and wrapped in clean, dry, and warm fabric with its head covered and given to the mother if she desires. If the baby does not cry within 10 to 15 seconds after delivery, has obvious airway obstruction, or is premature, the umbilical cord should be cut immediately and resuscitative efforts begun.

Shoulder Dystocia

If there is difficulty delivering the anterior shoulder (shoulder dystocia) by the method outlined, immediate steps should be taken to accomplish this. True shoulder dystocia occurs in less than 1% of deliveries and is rare in uncomplicated labors but is a substantial cause of fetal and maternal morbidity. Shoulder dystocia is often anticipated when the fetal head snaps back tightly and fails to rotate after its delivery. If available, other individuals can assist. Throughout the steps necessary to accomplish delivery, excessive traction on the fetal head is avoided because it may stretch the brachial plexus, resulting in Erb's or Klumpke's palsy. The first step is to position the woman so that the buttocks are elevated to allow at least 12 inches of free space beneath the perineum to maneuver. Both legs should then be flexed upward to the chest (McRobert's maneuver) while the woman is supported in a semirecumbent position behind her back. Delivery should then be attempted again by downward traction on the fetal head.

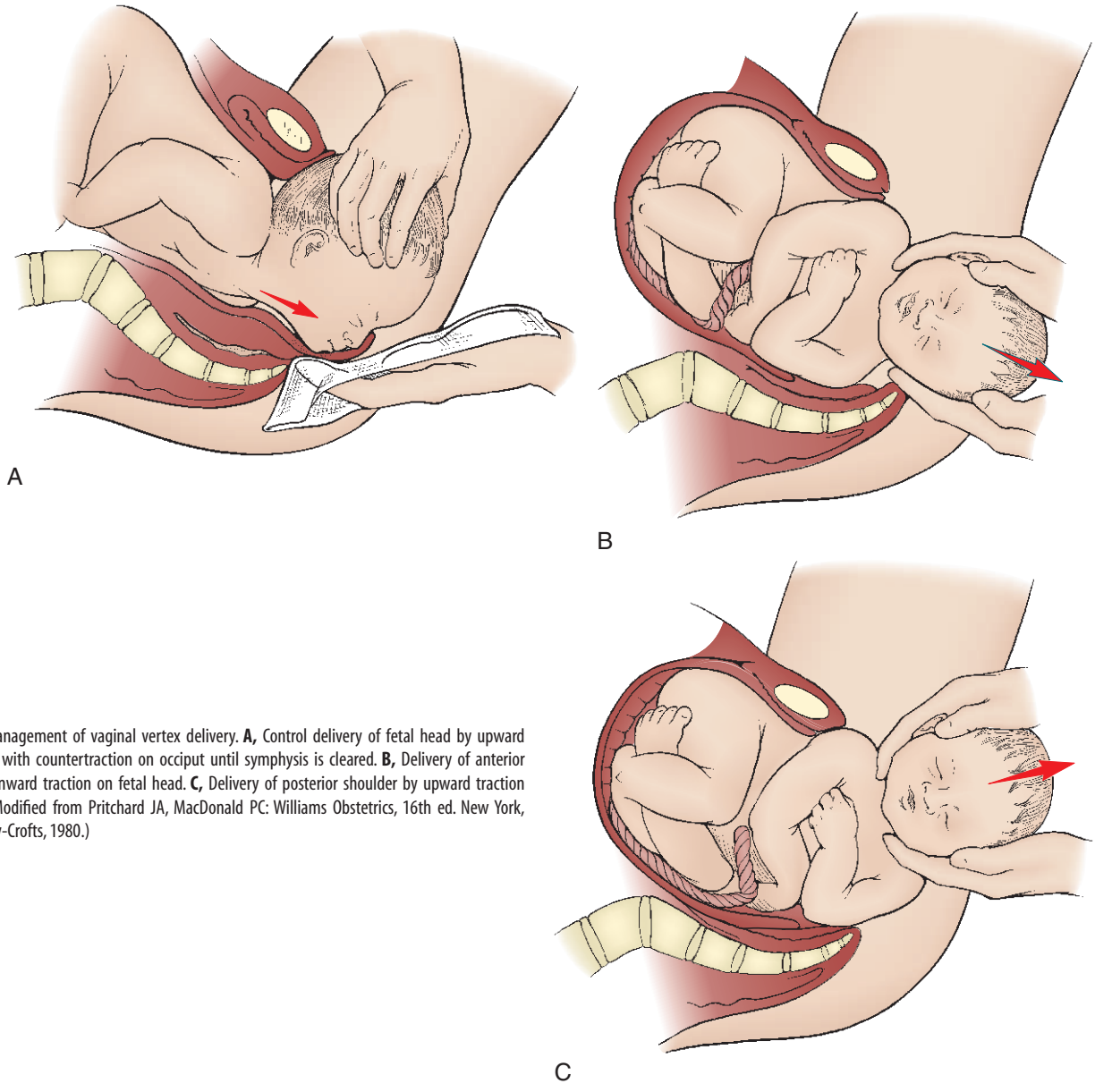

Figure 88-7. Management of vaginal vertex delivery. **A,** Control delivery of fetal head by upward pressure on chin with countertraction on occiput until symphysis is cleared. **B,** Delivery of anterior shoulder by downward traction on fetal head. **C,** Delivery of posterior shoulder by upward traction on fetal head. (Modified from Pritchard JA, MacDonald PC: Williams Obstetrics, 16th ed. New York, Appleton-Century-Crofts, 1980.)

If the shoulder is still impacted against the symphysis, pressure applied with the fist or heel of the hand just above the symphysis in the midline may reduce it sufficiently to accomplish the delivery. The assistants should not push on the uterine fundus because this can further impact the shoulder. If these maneuvers fail, an episiotomy should be cut to admit several fingers or the hand beneath the posterior shoulder. Once the hand has been inserted, the baby should be rotated by applying pressure to the shoulder and scapula (Wood's maneuver). The corkscrew rotation will deliver the posterior shoulder as it turns anteriorly, the anterior shoulder will dislodge, and the baby can be delivered without further difficulty. If this rotational maneuver fails, the posterior arm is delivered by grasping it along the forearm and sweeping it across the chest and out the vagina. This technique may fracture the humerus or the clavicle but is preferable to losing the baby because of inability to complete a delivery. Once the posterior arm is out, the anterior shoulder can usually be displaced downward, or the baby can then be rotated, allowing completion of the delivery. This approach is preferable to intentionally fracturing the clavicle, which can be technically difficult and does not provide as much room for the delivery.

If the baby is in a vertex presentation but facing the symphysis (occiput posterior), the labor is often more prolonged and uncomfortable, particularly in the lower back. The delivery is basically accomplished as described, however, except the final maneuvers to deliver the fetal head are extension first, then flexion. Perineal and introital trauma is a greater risk with an occiput posterior delivery. Management of this fetal presentation by the wilderness birth attendant should be a minor challenge compared with delivery of a breech baby.

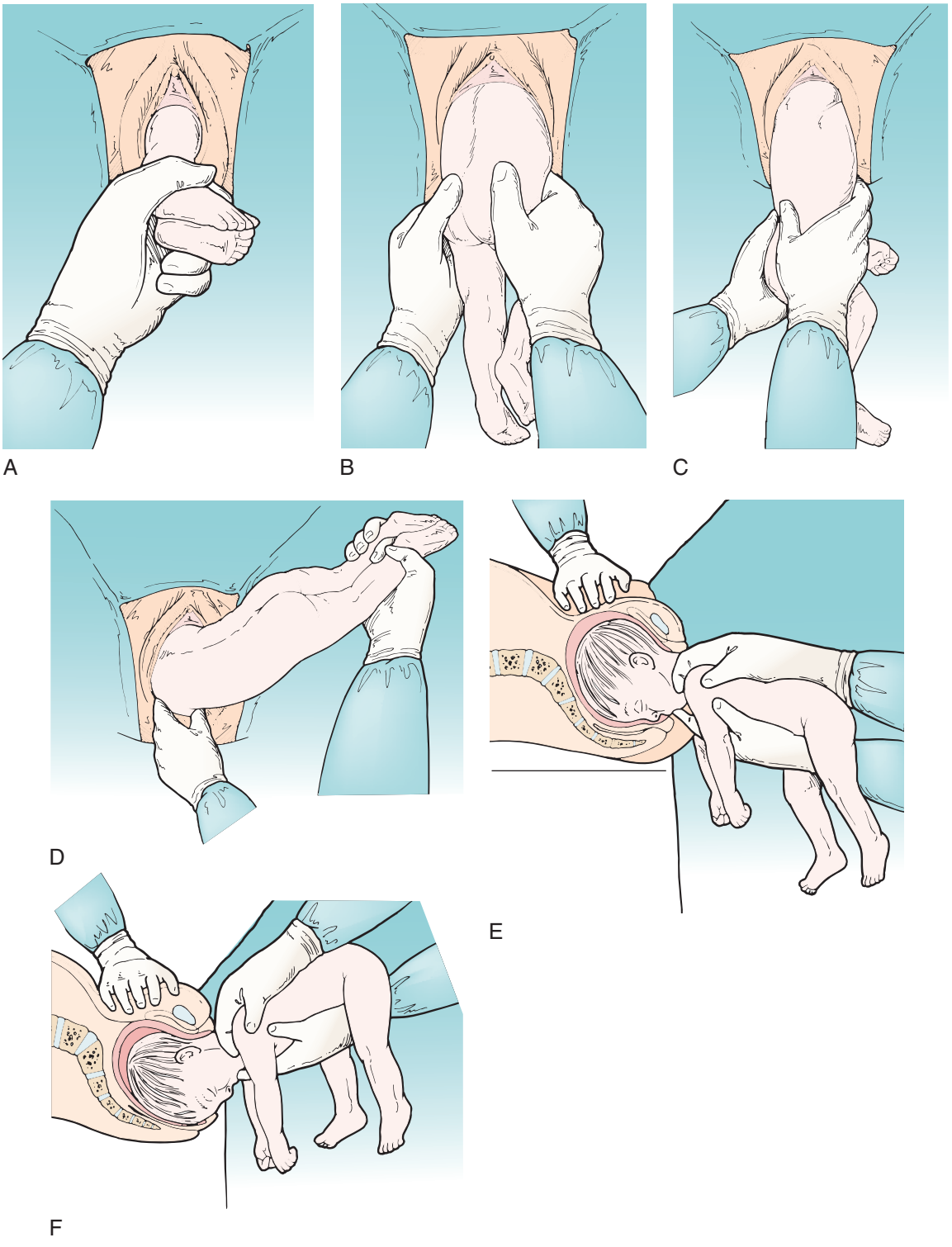

Figure 88-8. Management of vaginal breech delivery. **A,** Downward traction at ankles until buttocks clear the introitus. **B,** Traction on pelvic girdle until an axilla becomes visible. **C,** Delivery of posterior shoulder and arm. **D,** Delivery of anterior shoulder and arm with downward traction. **E,** Cradling the baby on forearm, finger is inserted into mouth or against chin. **F,** Delivery completed by outward traction while maintaining fetal head in flexed position. (Modified from Pritchard JA, MacDonald PC: Williams Obstetrics, 16th ed. New York, Appleton-Century-Crofts, 1980.)

Breech Delivery

Because most wilderness deliveries are "unexpected" and more likely to be premature, the baby will also more likely be in a breech lie (Fig. 88-8). Other than chance and prematurity, the greatest risk factors for a baby to be in a breech presentation are unsuspected congenital fetal anomalies, chromosomal abnormalities, and maternal uterine abnormalities. Each of these adds a new level of challenge to the birth attendant. Under the best of circumstances, delivery of a breech carries a threefold to fourfold greater risk than a vertex presentation for morbidity resulting from prematurity, congenital abnormalities, and trauma at delivery. The trauma often results from disproportion between the larger fetal head and the smaller body circumference of a premature infant. This disproportion can lead to entrapment of the head and is especially problematic when the fetal body has negotiated an incompletely dilated cervix.

Breech babies come in many forms: frank breech (hips flexed, knees extended, buttocks presenting), complete breech (both hips and both knees flexed, buttocks and feet presenting), incomplete breech (one hip flexed, one hip partially extended, knees flexed, buttocks and feet presenting), and footling breech (hips and knees extended, feet presenting). Regardless of the form, the approach in a wilderness setting demands patience. No effort should be made to deliver a breech baby until the presenting part is visible at the introitus and the cervix is completely dilated. Membranes should not be artificially ruptured in breech presentations. As the amniotic sac balloons into the birth canal, it helps to dilate the cervix completely. This facilitates descent of the baby, providing a lubricated smooth surface against which the body can freely move, and cushions the umbilical cord against compression in the birth canal.

When the cervix is completely dilated, the woman is instructed to push. Regardless of the type of breech presentation, the safest course is to allow the body to be extruded to at least the level of the umbilicus by maternal efforts alone. This increases the chance that the fetal head has begun to pass through the pelvic inlet. A baby in a frank or complete breech lie should have the posterior leg delivered by gently grasping the thigh and flexing the leg at the knee as it is rotated medially and toward the introitus. The baby should then be rotated to the sacrum anterior position, then another 45 degrees in the same direction to facilitate delivery of the other leg using the technique described for the first. The legs and buttocks can be wrapped in a clean towel to provide a firmer grip and decrease trauma to the baby. The delivery from this point is the same as for footling breech presentations. The upper legs should be grasped on each side, with the index fingers crossing the infant's pelvic girdle and both thumbs positioned just above the crease of the buttocks. Using gentle side-to-side rotational motion over an arc of 90 degrees outward and downward, traction should be applied while the mother pushes, until the upper portion of a scapula is visible at the introitus. With the baby's body rotated 45 degrees toward the opposite side, the arm is delivered by flexion and medial rotation across the chest. The baby is rotated to the opposite side in the same position, and the other arm is then delivered. If assistants are present, the woman should be helped into the McRobert's position, with hyperflexion at the hips to maximize the space between the symphysis and the sacrum.

Maintaining the baby in the same plane as the vagina, the birth attendant reaches palm up between the baby's legs and into the vagina, supporting the baby's entire body on the forearm while placing the second and fourth fingers over the infant's maxillae and placing the middle finger into the mouth or on the chin. The other hand is positioned over the infant's upper back so that those fingers are overlying each shoulder. If there is sufficient room, the middle fingers can be applied to the fetal occiput. Then with the woman pushing, the baby's head is flexed downward, completing the delivery. Firm suprapubic pressure can help to maintain the head in flexion. During this final stage, the baby's body should not be elevated more than 45 degrees above the plane of the vagina to avoid hyperextension of the head. If the fetal head cannot be delivered because the cervix is incompletely dilated, the cervix can be cut at the 2- and 10-o'clock positions (Dührssen's incisions) to provide sufficient room to complete the delivery. Once delivered, if the baby breathes and cries spontaneously or with minimal stimulation, cutting the umbilical cord can be delayed while the baby is dried. This allows some of the blood retained in the placenta from umbilical vein compression (common with breech deliveries) to return to the baby. On the contrary, if the baby is clearly depressed, the umbilical cord should be immediately clamped and cut and neonatal resuscitation begun.

Neonatal Resuscitation

The first steps in neonatal resuscitation are to dry the baby thoroughly, keep the baby warm, and clear the nose and mouth of excess fluid. Respiratory effort and heart rate (by auscultation or palpation at the base of the umbilical cord) are then assessed. If breathing spontaneously with pulse greater than 100 beats/min, the baby should be kept warm and observed. If the pulse falls below 100 beats/min and respiratory effort is poor, the next step is to improve ventilation. If further stimulation of the baby by rubbing with a towel or flicking the heels fails to elicit improvement in respiratory effort and pulse, the next step is to provide ventilatory support, ideally by applying positive-pressure ventilation with a neonatal mask and Ambu bag (preferably with oxygen). If this is available, gentle (15–30 cm H_2O) and rapid (40–60 breaths/min) ventilation should be performed for 30 seconds and the heart rate reassessed. If no equipment is available, the resuscitator's mouth is placed over the infant's nose and mouth, and rapid shallow breaths are delivered at a rate of 30 to 40 breaths/min. If this restores heart rate and respiratory effort, the baby should be observed for deterioration in status and the maneuvers repeated as necessary until support is available.

If the baby's heart rate falls below 60 beats/min, full infant cardiopulmonary resuscitation (CPR) should be started. Cardiac compression is performed by (1) placing both thumbs on the sternum just above the xiphoid and facing the fetal head, (2) gently stabilizing this position with the other fingers around the chest, and (3) supplying compressions to a depth of $^1/_2$ to $^3/_4$ inch (1.2 to 1.9 cm) at a rate of about 90 per minute. Care should be taken not to deliver compression to the baby's ribs. Ventilation should be continued simultaneously as described. If the heart rate after 30 seconds of chest compression is 80 to 100 beats/min, the resuscitator continues with ventilatory support only. If the heart rate is more than 100 beats/min, the person discontinues CPR and observes. If the heart rate is still less than 80 beats/min, CPR is continued.

Delivery of Placenta

After the baby is delivered and stabilized, attention is redirected to the mother. The first step is to assess the status of placental separation. The heel of the nondominant hand is placed just

above the symphysis to hold the uterus in position, and then the fingers are cupped to apply pressure to the uterine fundus while providing gentle, steady downward traction on the umbilical cord. If this maneuver does not promote placental separation, as indicated by a gush of bleeding, lengthening of the cord, and descent of the placenta into the vagina, efforts should be interrupted until these signs ensue. When the placenta does descend, the mother should be instructed to push once again to complete the third stage of labor. Rotating the placenta several times once it has passed through the introitus will usually result in complete extrusion of the attached chorioamnionic membranes. With signs of placental separation but resistance to extraction, the hand should be placed through the vagina and into the cervix. If the placenta is filling the cervix, it should be grasped and gently extracted. If the placenta does not separate spontaneously or is adherent to the uterine wall (placenta accreta), no effort should be made to separate it manually in a wilderness setting because this could precipitate uncontrollable hemorrhage. Excessive traction on the umbilical cord also could result in uterine inversion, causing vasomotor collapse and hemodynamic decompensation.

Once placental expulsion has occurred, the uterus can be gently massaged abdominally to promote contraction and involution. Usually, this is sufficient to control hemorrhage from the placental bed. If this is ineffective, it may be necessary to explore the uterus manually for retained placenta while compressing the fundus externally until it contracts. Additional measures to aid uterine involution and control bleeding include administration of oxytocin (10 U/mL intramuscularly), nipple massage to promote endogenous release of oxytocin, methylergonovine (0.2 mg intramuscularly or orally every 2 to 4 hours), prostaglandin $F_{2\alpha}$ carboprost tromethamine (250 µg intramuscularly), misoprostol (100 to 200 µg orally every 4 to 6 hours), and prostaglandin E_2 (20 mg suppository every 2 hours).[123] If none of these measures is successful, and if bleeding from a laceration has been eliminated as a source, the uterus can be packed with clean sponges or towels until additional medical assistance arrives.

Once the placenta is removed and uterine bleeding controlled, maternal damage is assessed and repaired. The most common sites of lacerations are the perineum, periurethral tissues surrounding the external meatus, lower vagina, and cervix. Significant cervical lacerations in unhurried deliveries are rare unless uncontrollable pushing has occurred before complete dilation. Other lacerations from a spontaneous delivery usually occur along tissue planes that do not disrupt vital areas; they will heal naturally or can be repaired later. Significant bleeding at any of these sites can usually be easily controlled by direct pressure. If available, application of ice packs to the perineum for the first 12 to 24 hours after delivery provides relief.

During the postpartum period, the woman should be encouraged to drink fluids and void frequently. Bladder overdistention resulting from impaired sensation and pain after delivery is a common cause of permanent bladder dysfunction. It may also contribute to uterine subinvolution and continued blood loss. Once the baby has stabilized after delivery, the mother should begin breastfeeding, which also helps control excessive postpartum blood loss. After a wilderness delivery, a broad-spectrum antibiotic, if available, should be continued for 24 to 48 hours. If the mother is Rh negative and the baby's blood type is Rh positive or unknown, Rh immune globulin, 300 µg intramuscularly, should be given within 48 to 72 hours after delivery.

Breastfeeding

Unless the baby is too premature or too unstable to nurse, breastfeeding should be encouraged as soon as possible after birth. Benefits include promotion of uterine contractions that control hemorrhage at the placental insertion site, encouragement of maternal–newborn bonding, provision of easily digestible and balanced nutritional support for the baby, and transmission of antibodies (immunoglobulin A) that protect the enteric mucosa against invasion by colonizing bacteria.[5]

During the first 24 hours after delivery, frequent brief feedings are recommended (every 2 to 3 hours; 5 minutes on each breast, alternating first breast). Although a small amount of breast fluid (colostrum) is present initially, it contains electrolytes, minerals, and a high concentration of protein and protective immunoglobulin A antibodies. The length of time spent breastfeeding and the interval between feedings can be increased as milk production is established over the next 2 to 3 days. The feeding schedule is typically 10- to 15-minute periods on each breast 8 to 12 times per day. It should be made clear to the mother that babies are not restricted to this regimen.

Breastfeeding women should be instructed to drink plenty of fluids (2 L/day); increase their caloric intake by 500 to 600 kcal/day, including a total protein intake of 60 to 70 g/day; and consume foods rich in calcium (1200 mg/day).

Breastfeeding Practicalities. Several conditions can interfere with breastfeeding or cause maternal frustration. Breast engorgement 48 to 72 hours after delivery, signifying the onset of milk production accompanied by lymphatic obstruction, can cause pain and low-grade fever, interfere with the baby latching on, and inhibit milk letdown. Frequent feedings and warm compresses just before nursing help to stimulate milk letdown. Cool compresses after nursing, a supportive nursing bra, and acetaminophen usually provide symptomatic relief until milk production and newborn consumption are in equilibrium and lymphatic obstruction is resolving. Engorgement usually resolves within 24 to 48 hours.

Sore and cracked nipples are a frequent complaint of women nursing for the first time.[110] Short, frequent feedings with several rotations between breasts at each sitting can be beneficial. After each feeding, gently cleansing with water and applying a small amount of milk, expressed from the breast and spread around the areola to dry, help protect the nipples. Lanolin formulations designed for breastfeeding women can be used as well. Dry absorbent nursing pads should be placed over the nipples between feedings. If contact with clothing or even the nursing pads creates discomfort, breast shells can be used to prevent surface contact. Occasionally, nipple shields can be beneficial for the mother, as well as for the infant who has difficulty latching on to the breast.[170]

Mastitis occurs in 2% to 3% of lactating women and should not be confused with breast engorgement because it rarely occurs until at least 3 to 4 weeks postpartum.[110] Unlike breast engorgement, mastitis is usually unilateral and accompanied by localized pain, erythema, brawny edema, fever, and malaise. It is more common among women who report painful and cracked nipples and who participate in vigorous exercise- and work-related upper-body activities. These women should be encouraged to empty their breasts by nursing or pumping before these activities and to wear a properly fitting and supportive bra as preventive measures for mastitis. Women who develop mastitis should continue to nurse from the affected breast and may benefit from warm compresses and pumping between nursing.

They should be placed on a course of antibiotics (e.g., dicloxacillin, 500 mg, or cephalexin, 500 mg, four times daily) for 10 to 14 days, with coverage for *Staphylococcus* and *Streptococcus* species and *Escherichia coli,* the most common bacterial isolates from affected breasts. Failure to improve or worsening while on this regimen, as determined by consolidation, widening of erythema and induration, and abscess formation, occurs in 10% to 15% of women. Incision and drainage may be required.[110] An antibiotic-resistant organism, such as a methicillin-resistant *Staphylococcus aureus* (MRSA), should also be considered.

For the breastfeeding mother who is traveling without her infant, a breast pump can be used to maintain milk production and prevent engorgement and mastitis. Manual, battery-powered, and electric breast pumps available.

Medications during Breastfeeding. All physicians treating breastfeeding mothers should have access to relevant and accurate sources providing data on medication safety during pregnancy and breastfeeding.[5,30,78]

Useful References

The Committee on Drugs of the American Academy of Pediatrics published a list of drugs and chemicals that transfer into human milk. The list is updated on a regular basis. The statement may be found online at www.aap.org.

Medication and Mother's Milk, written by Thomas Hale,[77] is another excellent reference that provides an alphabetical list of medications with references at the end of each review. This source calculates a theoretic infant dose of medication that a breastfed infant might receive.

Because no randomized controlled trials exist on the safety of medications during lactation, any medication given to a lactating woman should be carefully considered. Most routinely prescribed drugs are safe to use during lactation. Short-acting drugs administered after a feeding have the least opportunity to be excreted into milk. Few drugs are absolutely contraindicated while breastfeeding. With important exceptions, most categories of medications are safe for mothers to take without discontinuing breastfeeding. Most medications have no effect on milk supply or on infant health (see Table 88-7). Lactating mothers may safely receive all vaccinations. Strains of live viral vaccines are not known to be transmitted in breast milk, with the exception of attenuated rubella virus. Infants are usually not infected by the vaccine strain of rubella.

Box 88-9. Checklist for the Older Adventuress

- Review itinerary
- Review past medical history and medications carefully
- Immunizations
  - Risk vs. benefit
  - Issues related to immunogenicity in older adult
- Chemoprophylaxis
- Environmental
  - Heat, cold, performance data in older individuals
- Menopausal issues
  - Estrogen replacement therapy
  - Osteoporosis prevention
  - Incontinence, urinary tract infection, constipation, other
- Sexually transmitted infection prevention
- Medical kit
- Safety issues
- Pre–wilderness travel evaluation: electrocardiogram, mammogram, Pap smear
- Evacuation insurance

WILDERNESS HEALTH ISSUES FOR THE OLDER ADVENTURESS

Box 88-9 is a useful checklist for the older female traveler.

ACKNOWLEDGMENTS

Many thanks are due to Dr. Kenneth F. Trofatter, Jr., and Dr. Barbara D. Dahl for their excellent work on this chapter in the previous edition.

The references for this chapter can be found on the accompanying DVD-ROM.

89 Elders in the Wilderness

Blair Dillard Erb

There is still no cure for the common birthday.
John Glenn, Senior Astronaut[18]

"The elderly still climb mountains: it's just that their definition of mountains has changed considerably."[13] The Gray Eagles prove the point. This organization consists of elders with a passion who venture regularly into the wilderness.[9] They recognize that they are not champions, but enjoy the fellowship, camaraderie, and adventure of wilderness activities. However, altered physiologic function, unrecognized impairments, or the effects of illness and their various treatments can cause serious health issues among elders. Consequently, in the elder population, health problems and untoward medical events associated with outdoor activities can be found more frequently than in younger, more resilient participants. Prevention of such problems is of utmost importance. Intervention may be critical for relief or can even be lifesaving (see the section on Counseling and Teaching Elders Before Wilderness Ventures: The Gray Eagles).

Most extreme performance ventures do not include elders, but there are instances of incredible physical performance from elderly individuals. Ulrich Inderbinen (Fig. 89-1), born in the shadow of the Matterhorn in Zermatt, was a mountain guide who climbed the Matterhorn 370 times, the last time at age 90. He died in 2004 at age 103.[8] Keizo Miura (1904–2006), a Japanese ski legend in his own time, became the oldest man to climb Mount Kilimanjaro in 1981, skied down Mont Blanc at age 99, and was a dynamo even beyond his 100th birthday.[16]

Data regarding elders come from organizations and government services, such as the National Park Service and some local, county, and state emergency medical services, which maintain and report records of wilderness emergencies. The highly respected Explorers Club and the American Alpine Club report on and can advise on health hazards and their preventive measures. There is a vigorous elder women's group, which, in its own words, "transformed the image of helpless old ladies to one of power and strength as they unite to protect America's roadless wild lands."[42] Other active organizations, some of which have medical links, can be found on the Internet. The Wilderness Medical Society provides an excellent source of medical and related information, through its journal *Wilderness and Environmental Medicine*.

THE AGING PROCESS

Aging is a natural consequence of life. It leads to anatomic, biochemical, and physiologic alterations in every organ system (Table 89-1). Common degenerative disorders and diseases include atherosclerotic and cerebrovascular disease; chronic obstructive pulmonary disease; emphysema; diabetes mellitus; arthritis; emotional, mood, and memory disorders (e.g., depression, Alzheimer's disease); and impaired thermoregulation. Polypharmacy (including excesses in medication intake) is a problem. Accidents due to unsteadiness, falls, forgetfulness, and operating machinery and vehicles take their toll. The cumulative effect of these changes on elders subjected to stressful environments produces a major increase in health risk and health care demand.[4,12]

Anatomic changes are organ specific, as organs appear to age independently and not necessarily in parallel fashion. Furthermore, laboratory tests must be carefully interpreted. For example, glomerular filtration rate (GFR) and renal blood flow (RBF) decrease with age, but many elders have normal serum creatinine levels because there is a concomitant loss of muscle mass due to aging and, as a result, lower creatinine production. Physical and physiologic alterations may occur slowly. They may not be apparent for many years, yet result in "silent" functional and anatomical changes.

The degree of loss of function in various physiologic systems can be approximated by using the "1% rule," which states, "most organ systems lose function at roughly 1% per year after the age of 30 years."[25] Some age-related biologic changes and their resulting functional changes are listed in Table 89-1.

Inasmuch as aging reflects the effects of the passage of time, injuries and illnesses that occur along the path of life may produce cumulative anatomic scars, which, when combined with the degenerative changes of aging, may result in a functionally impaired elderly person. The risk may be greatly exaggerated by wilderness ventures. The challenge to health professionals is to determine the extent of that risk and to take appropriate action.

ETIOLOGY OF THE AGING PROCESS

Some individuals age faster than others. Why? Lifestyle and genetic predisposition are most commonly incriminated "causes," but this does not explain the biologic basis for aging.[21] Genetic events may determine longevity somehow. For example, there is an increased risk of development of Alzheimer's disease in the presence of an allele of apolipoprotein E *(ApoE)* gene, which encodes a carrier of cholesterol.[2] Support for genetic determination of tissue longevity is scant. Most researchers believe that the processes of aging are multifactorial. Speculative theories suggest that damage to deoxyri-

Figure 89-1. Ulrich Inderbinen, the world's oldest mountain guide, died in 2004 at his home in Zermatt, Switzerland, at age 103. He lived his entire life in the shadow of the Matterhorn, which he climbed 370 times, the last time when he was 90 years old.[8] (Courtesy Corbis.)

bonucleic acid (DNA) and proteins occurs from a variety of sources, such as reactive oxygen species, including superoxide, the hydroxyl radical, and hydrogen peroxide. Mitochondria are important sources of reactive oxygen species and a major site of damage.

Hayflick in 1961 observed that each body cell undergoes a finite number of divisions, roughly 50, after which the cell dies.[21] This number of divisions is known as the "Hayflick limit." Each cell replication has a span of about 18 months and may be a predictor of individual human longevity. For example, with a cellular life span of 18 months, 50 doublings would result in a human life span of 75 years. Variation in the duration of each doubling has been speculated to be a predictor of length of life.

The Hayflick limit is a function of caps at each end of the chromosomes called telomeres. These caps are a DNA-protein complex, which shorten after each cell division, protected by a cellular enzyme, telomerase. Eventually, after about 50 divisions, the telomeres become too short for further division and the cell dies.

Molecular concomitants of aging include alteration in chromosomal structure, mitochondrial deterioration, DNA cross-linking and single strand breaks, a decline in DNA methylation, and loss of telomeric sequencing.

Other theories on the aging process include cellular changes, autoimmune mechanisms, neuroendocrine factors, and even a "biological clock," but Strehler[40] feels that the cause should explain the progressive deleterious and intrinsic changes universal within the species. For example, some animals, usually cold-blooded fish or amphibians, which may grow to an indeterminate size, may have an indeterminate life span, whereas warm-blooded animals with a limited or fixed size after maturation may die at a more predictable time and at an actuarially determined rate.[41]

Questions concerning the nature and cause of aging include the following:

- *Does aging affect everybody?* Yes, but with considerable variation.
- *Is aging a disease? Are there decrements in or natural losses of function and anatomic content representing "normal aging"?* If so, there must be such a thing as "abnormal aging."
- *Is aging genetically programmed as a sequence of events?* Evidence suggests that genetic events may influence longevity, especially by modifying various influences, such as cholesterol and blood sugar. Three genetic theories include: (1) mutations that impair long term survival, (2) pleiotropic antagonism affecting the genes, and (3) variations in environmental survival hazards among clans.
- *Is aging a result of natural selection?* Darwin's laws of natural selection and evolution have been considered, but in the 2 to 3 million years of human existence, there have been too few old humans in any generation until recently to provide proof of a selective advantage favoring genetic expressions related to aging.
- *Is there a finite number of population doublings of human fibroblasts?* As noted earlier, Hayflick suggested that there are 50 doublings of fibroblasts during their life span (the "Hayflick limit").
- *Is an individual responsible for personal longevity?* Lifestyle factors, such as lack of exercise, dietary habits, tobacco use, and drug and alcohol use, not only influence anatomic and functional characteristics, but also may have deleterious effects on health and length of life.

In perhaps the clearest summation of these theories, Hayflick suggests that the ultimate effect from the many factors influencing and affecting human life is that we simply exceed our reserve capacity. This lends support to the mountaineering dictum, "Always keep your reserve," which is particularly appropriate for elders.

DEFINITION OF ELDERS

Classifying Elders by Age and Health

Definition of "elders" can be complicated. Barry and Eathorne[3] suggested defining them as the *hale* or the *frail*. This is a delightful play on words, but lacks the precision needed for medical decision-making. Smith[36] reported by Howley recommends a classification according to chronologic age: (1) *athletic old* (younger than 55 years), (2) *young old* (55 to 75 years of age), and (3) *old old* (older than 75 years of age). This classification focuses only on the chronologic age and fails to recognize non-uniform functional change during the passage of years and residual effects of remote illnesses and injuries.

A more precise and comprehensive classification that consists of three components is preferred: (1) *chronologic:* simple time-based classification of years (e.g., age 55, age 63); (2) *pathologic:* describing morphologic and anatomic changes associated with disease or degenerative processes; and 3) *functional:* describing changes in function resulting from impairment.

Functional classification of individuals is based on an idealized bell-shaped distribution curve that places participants in one of five categories labeled alphabetically as explained later in this chapter: (A) high performance persons, (B) healthy vigorous persons, (C) healthy deconditioned persons, (D) persons with risk factors, and (E) persons who are manifestly ill. Specific aerobic capacity, defined as maximal physical work capacity (PWCmax) can be derived from graded exercise testing. Other specific functional data can be determined from testing physical modalities in a human performance laboratory, cardiac rehabilitation center, or physical/occupational therapy unit.

TABLE 89-1. Age-Related Biological Changes and Their Functional Consequences

ORGAN SYSTEM	AGE-RELATED ANATOMIC CHANGES	AGE-RELATED PHYSIOLOGIC CHANGES	AGE-RELATED FUNCTIONAL CONSEQUENCES
General	Decreases in organ mass and muscle	Decreased organ function; decreased oxygen consumption	Decreased flexibility, endurance, and maximal performance
Cardiovascular	Fibrosis of arterial media; thickening of arterial intima; sclerosis of arterial valves (especially aortic and mitral); elongation and tortuosity of aorta	Decreased maximal heart rate of 6 to 10 beats/min per decade; decreased beta adrenergic responses; increased left ventricular ejection fraction; decreased arterial compliance	Decreased cardiac output of 20–30% by age 70 years; decreased PWCmax; orthostatic hypotension; decreased endurance; syncope; shortness of breath
Lungs	Decreased lung elasticity; decreased activity of cilia; reduced cough reflex	Decrease in vital capacity of approximately 30 mL/year after age 30 years; microaspiration	Shortness of breath; cough; aspiration pneumonia
Kidneys	Increase in number of abnormal glomeruli	Decreased glomerular filtration rate; decreased renal blood flow; decreased urine concentration; compensatory reduction in muscle mass neutralizing creatinine elevation; proteinuria	Delayed response to salt or fluid restriction; nocturia
Genitourinary	Prostatic enlargement; vaginal/urethral mucosal atrophy	Urinary retention (increased residual volume); bacteriuria; atrophic vaginitis	Nocturia; tenesmus; incontinence; urinary tract infection
Gastrointestinal	Atrophic mucosa; atrophic taste buds; anorectal incompetence	Decreased salivary flow; decreased gastric hydrochloric acid; decreased hepatic function; decreased motility	Regurgitation with aspiration; food intolerances; constipation; incontinence; modified appetite, food intake, and motility
Hematologic/immune	Bone marrow fibrosis; metaplasia	Decreased bone marrow reserve; decreased T-cell function; antibody dysfunction	False negative immunological skin tests; false positive laboratory immune tests (i.e., rheumatoid and antinuclear antibody)
Musculoskeletal	Decreased height, weight, lean body mass, muscle, and bone density; sarcopenia	Loss of skeletal calcium; reduced elasticity in connective tissue; decreased viscosity of synovial fluid	Loss of cartilaginous surfaces; hypertrophic changes in joints; increased ratio of fat to muscle mass; osteoporosis; FTT; loss of muscle mass and strength of 20% by age 65 years
Endocrine	Osteoporosis; vertebral collapse; changes in fluid volumes	Altered glucose homeostasis; decreased thyroid and testosterone hormone; renin, aldosterone production and vitamin D absorption; increased ADH	Hyperglycemic response to stress; diabetes mellitus; ± hyponatremia; ± hyperkalemia; osteopenia; osteoporosis; impotence
Nervous	Reduced brain mass; decreased cortical cell count	Decreased brain catechol and dopamine synthesis; impaired thermal regulation	Decreased nerve conduction; impaired cerebral and cognitive functions; dementia; depression; forgetfulness; sleep changes; loss of agility; balance; falls; hypothermia, hyperthermia; sensory impairment, including taste, smell, vision, hearing, and touch
Eyes	Decreased translucency of lens; decreased size of pupil; increase in intraocular pressure; macular degeneration; arcus senilis	Decreased accommodation; requirement for increased illumination; susceptibility to glare	Decreased vision including color and night vision; impaired accommodation; presbyopia
Ears	Loss of auditory neurons; atrophy of cochlear hair cells	Decreased hearing, especially high tones greater than 2000 Hz; decrease in directional discrimination; vestibular dysfunction	Loss of hearing; loss of click pitch hearing and constant discrimination; balance impairment with falls
Skin	Flattening, atrophy, and attenuation in dermal collagen and rete pegs and cytoplasm of basal keratinocytes	Decreased skin thickness; risk of dermo-epidermal separation; loss of elasticity	Decreased resistance to tear

PWCmax, maximal physical work capacity; FTT, failure to thrive.

TABLE 89-2. Classification of Wilderness Ventures

CLASS	GENERAL DESCRIPTION	EXAMPLES
1	Extreme-performance ventures	High-altitude mountaineering, such as a Mount Everest climb or other Himalayan trekking
2	High-performance ventures	Remote hunting activities, particularly at high altitude or under stresses of heat, dust, or cold Jungle trekking
3	Recreational activities	Trail walking is generally considered recreational, but because of endurance demands and environmental risks, it may present physiologic hazards. Other activities that may fit this classification: Alpine hiking National Park Trail walking Forest-based orienteering
4	Therapeutic activities	For more than a century, physical activity has been recommended for certain individuals with cardiovascular disease and other physical limitations to improve their functional capacity.

This classification is useful for matching an individual with various wilderness activities according to physical and environmental demands, described in sections on Physical Activity and Medical Examination.

As an example of this classification scheme, a patient age 58 years, with coronary artery disease and angina with a stent in place who is symptomatic during a graded exercise test at 6 METs, would be functionally classified as:

1. Chronologic: 58 years of age
2. Pathologic: stented coronary artery disease
3. Functional: symptomatic (angina) 6-MET maximal physical work capacity

This person is manifestly ill and is considered class E.

Demography of Elders and the Wilderness

A striking increase in longevity during the 20th century has changed the age composition of Western civilization. Life spans have continued to increase in the 21st century. The median age in the United States in the year 1900 was 23 years. It rose to 30 years in 1950, 33 years in 1990, and 36 years in 2000. More importantly, the population in the United States older than 65 years has been projected to increase from 35 million (12.4%) in the year 2000 to 71 million (19.6%) in 2030.[30] Octogenarians comprised 9.3 million (3.3%) in 2000 and will number 19.5 million (5.4%) in 2030.

It is not simply the size and growth of this group of seniors that is responsible for its changing medical needs, but rather the nature of the lifestyle and activities adopted by them. Of the 18 million persons between the ages of 65 and 74 years, most are retirees who have stimulated a surge in vigorous outdoor recreational activities. Many are at medical risk. The largest increases in elders leading up to 1990 occurred in regions in the United States most commonly associated with active outdoor lifestyles.[7,17] Florida led the way in 1995, when 19% of the population exceeded 65 years of age. A consequent increase in medical needs warrants consideration of the nature of illnesses, injuries, and services unique to the elderly.

Lifestyle is considered important as a major factor in longevity. Leaf reported three locations in the world where individuals not only live to ages beyond 100 years, but they even expect to do so.[29] These locations are in relatively remote mountainous areas: the Caucasus Mountains in Georgia, the Andes in Ecuador, and the Karakoram range in Pakistan-controlled Kashmir. Speculation suggests that this longevity is due to a combination of factors, including genetic selection and a physically active lifestyle.

In keeping with these data and exposure to various life events, it is helpful to classify wilderness ventures according to demands required by the activity. A useful classification includes: (1) extreme performance ventures, (2) high-performance ventures, (3) recreational activities, and (4) therapeutic activities (Table 89-2). Mechanics for matching individuals according to their functional classification with the demands of the venture are described in the section on Physical Activity and Medical Examination.

WHY SOME ELDERS VENTURE INTO THE WILDERNESS

Hard work, family and fiscal responsibilities, and delayed gratification are characteristics of Western industrialized cultures. As retirement years approach and leisure time increases, seniors begin to increase their participation in recreational and outdoor activities.

The U.S. National Park System studied the frequency of recreational activities in the National Parks from 1982 to 1983 and found, in order of frequency, the following activities: driving for pleasure, sightseeing, walking for pleasure, picnicking, stream/lake/pool swimming, and motor boating.[5]

A repeat survey, completed by the National Park Service in 1994, found that walking has become the most popular single outdoor activity and now involves over 134 million participants. Walking is especially attractive for an older person because it is healthful, self-paced, and socially rewarding when enjoyed with others. With population increases of persons age 70 and older, predictions indicate that many new trails and walking venues will be needed. The most striking change in activities is the dramatic increase in bird watching, having risen from 21 million in 1982 to 54 million in 1994, an increase of 155%. Growth in participation within other activities includes hiking (94%), backpacking (73%), downhill skiing (59%), and primitive area camping (58%).[6]

Why go? Nash concluded that the personal reasons for elders to venture into the wilderness are for enjoyment of nature, physical fitness, tension reduction, tranquility and solitude away

Figure 89-2. President George H. W. Bush lives up to his remark, "Even old guys can still do stuff!," on his 75th and 80th birthday parachute jumps.[33] (Courtesy of the George Bush Presidential Library.)

from noise and crowds, experiences with friends, enhancement of skill and competency, and excitement or even the thrill of risk-taking[32] (Fig. 89-2). Some ventures, however, such as off-road motorcycling, kayaking, and cross-country skiing, take place in difficult and inaccessible areas under extraordinary environmental circumstances (cold, altitude, rough terrain) that in the event of an emergency may require search and rescue services or medical intervention.

PHYSICAL ACTIVITIES OF ELDERS IN THE WILDERNESS

For elders, the physical and environmental demands of certain activities may be excessive. The physical workload of a wilderness venture depends on its nature and the characteristics of its component parts. For example, is the venture a walk or a climb? What is the nature of the terrain? What is the altitude? What is the ambient temperature? It is prudent for elders to examine plans for prospective wilderness ventures and to select activities consistent with their personal capacity, skill, and tolerance.

Wilderness activities can be classified according to their physical, technical, and environmental characteristics. The skill, judgment, and capacity of all participants, elders included, can be matched with the characteristics of the venture to determine if individual capacity is adequate for the demands.

CLASSIFYING WILDERNESS VENTURES

One classification scheme for wilderness ventures ranges from those that are extremely demanding and risky undertakings to those with minimal physical and environmental requirements

TABLE 89-3. Classification of Participants in Wilderness Ventures

GROUP	GENERAL DESCRIPTION	EXAMPLES
A	Demonstrated high-performance individuals	Athletes in training Mountaineers continually active and in training Workers involved with heavy physical tasks
B	Healthy, vigorous individuals	Athletes Active hunting guides
C	Healthy deconditioned individuals	Young to middle-aged, healthy business and professional people who are moderately active
D	Individuals with risk factors	Individuals at risk because of age, lifestyle, smoking, excessive alcohol consumption, or factors not under their control. Most elders are in this group.
E	Individuals who are manifestly ill	People at any age with chronic illness or physical limitations, such as heart disease, diabetes, or neuromuscular or orthopedic problems

(see Table 89-2). Although this classification is very general, it can be helpful in examining prospective participants and matching demands of the wilderness task or activity facing that individual.[10,11]

PHYSIOLOGIC WORKLOAD OF WILDERNESS ACTIVITIES

Workload of wilderness ventures and functional capacity of individuals can be defined with a degree of precision by using measurements of energy demands from indirect calorimetry derived from oxygen consumption techniques. Oxygen consumption may be expressed as $\dot{V}O_2$, calories (1000 cal = 1 Kcal or 1 Calorie), or metabolic equivalents (METs).

The term *MET,* considered to be the energy cost of sitting, is defined by convention as 3.5 mL oxygen per kilogram of body weight per minute. Multiples of the MET are used to define the energy cost of various activities. For example, walking 2 miles an hour on a level, smooth surface requires 7 to 9 mL O_2/kg/minute or approximately 2 METs; 3 miles an hour, 10.5 mL O_2/kg/minute or 3 METs, and so on. Lists of the energy cost of activities given in Calories or METs are available. Most activities of daily living (ADLs) require an energy expenditure of less than 3 METs. When a task is complex, there may be several levels of energy cost of various components.

Armed with the energy cost of a planned activity, a venture planner can proceed with a comparison of the task requirements and the functional capacity of the participant. The average 40-year-old man in this country can reach a 10-MET activity at the point of exhaustion, the maximal physical work capacity (PWCmax). Derived from this is a useful scale of 1 to 10; that is 1 MET is the energy cost of sitting increasing to 10 METs as the PWCmax of the average 40-year-old man. Tolerance to physical activity among women, increasing for several decades, is now close to that of men, within 1 to 2 METs. Well-conditioned athletes can reach a PWCmax of around 13 to 23 METs.

A natural decline in PWCmax occurs with age and results in a loss of roughly 1 MET of PWCmax per decade after 40 years of age. The average maximal functional capacity of a healthy 70-year-old man is approximately 7 METs.

It has been empirically observed that a healthy individual can function at an energy expenditure of about 25% to 40% of personal PWCmax over an 8-hour period of time without being too exhausted to get up the next morning and repeat that level of energy expenditure. A healthy person with a 10-MET PWCmax should be able to handle roughly a 3-MET activity for 8 hours.

These figures are very general approximations only, and the circumstances of each prospective venturer need to be treated individually. Of course, skill and experience with any activity is the best predictor of a safe and successful venture.

CLASSIFICATION OF INDIVIDUALS CONSIDERING WILDERNESS VENTURES

Two basic physiologic factors affect the physical relationship between the venture and the participant: (1) the physical workload of the activity and (2) the capacity of the individual to tolerate the physical workload as influenced by the environmental stressors. Physical demands of the venture were classified previously. A similar classification assists in matching the participant with the venture (Table 89-3). The populations presenting to the physician may fit the distribution curve shown in Figure 89-3, where the groups defined in Table 89-3 are graphed. These classifications serve as starting places for more precise recommendations for the individual presenting to the physician for advice concerning physiological capacity to tolerate the physical demands of a specific adventure.[10,11,12]

MATCHING INDIVIDUALS WITH WILDERNESS PHYSICAL ACTIVITIES

Individuals who plan to participate in wilderness ventures probably have already been through a form of natural selection. For example, a person who aspires to participate in an expedition to Mount Everest will in all likelihood have already participated in a similar activity and will have proven his or her capacity to function at an extreme level of performance. This person would most likely be in participant group A or B. An individual who is healthy but has not recently been involved in vigorous activities and has become "deconditioned" may be in group C. The motivation may be a desire to reaffirm youth or vigor in some form of exciting or hazardous activity. These individuals deserve the scrutiny of alert organizers with, perhaps, an assessment prior to the venture in order to consider risk factors or occult health problems.

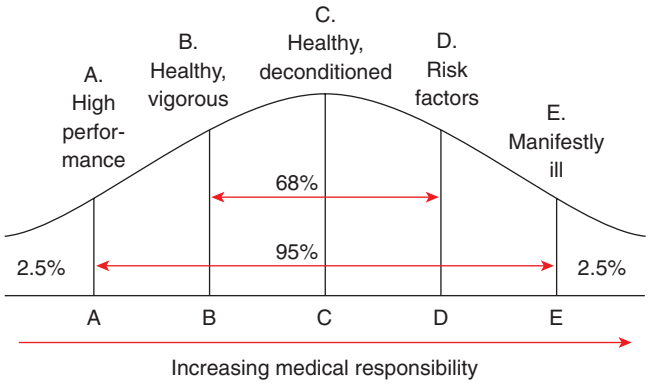

Figure 89-3. Classifying prospective participants according to physical characteristics, functional capacity, and experience helps match the candidate with demands of the venture. Here, prospective participants are arranged with an idealized representation of participants divided by standard deviations according to the classification in Table 89-3.

Particular attention should be directed toward any individual at risk of illness or injury (group D), even though manifest evidence of disease may not be superficially apparent. For example, cardiovascular risk factors, such as smoking, a fat-laden diet, and high blood pressure, may warrant detailed medical examination to determine level of functional capacity considered safe for that individual. An examination may even disclose the presence of diseases, asymptomatic or symptomatic.

Group E includes persons with definite manifestations of illness. Supervised outdoor activities can still be of value in such cases and have even been used as a form of physical therapy and rehabilitation for persons with various illnesses, including cardiovascular disease. Persons in this category should be individualized in their assessment and require a high degree of medical evaluation and supervision.

PHYSICAL CONDITIONING TO PREPARE ELDERS FOR WILDERNESS VENTURES

Elders considering wilderness activities should consider the appropriateness of their participation in a contemplated venture. After a medical and functional examination, the classification may suggest the need for a physical conditioning program to increase individual reserve capacity. An individual can tolerate an energy cost of approximately one fourth to one third of his or her maximal physical work capacity over a period of 8 hours. If the demands of a venture exceed predicted tolerance, PWCmax can be enhanced by 15% or more by a 3- to 6-month graduated conditioning program. A conditioning program should begin with a warm-up session that includes flexion and extension exercises, followed by a cardiovascular conditioning component and a cool-down component. The aerobic component should last at least 20 minutes at a level of about 50% of individual PWCmax. Some persons can learn to monitor the pulse rate corresponding to this level of activity as a monitoring technique. Mouth breathing, which usually occurs at about 60% of maximal work capacity, also is a useful body signal for marking the intensity of the activity. As intensity increases, catecholamines increase. Above 70% of maximal work capacity, the catecholamine level may be hazardous for an individual with subtle cardiovascular disease. Consequently, if the subject keeps the intensity of the activity below that requiring mouth breathing, the aerobic activity should be in the range below 60% of the individual maximal capacity, a relatively safe range in healthy individuals. This level of conditioning is helpful and may be reasonably safe for seniors, although not at the level necessary for conditioning competitive athletes. If cardiovascular disease is present, a conditioning program should be individualized and supervised. A structured program is an ideal time for teaching an individual to read their personal body signals, a collection of markers for physiologic responses to activity (Box 89-1).

The American College of Sports Medicine has developed a position paper on exercise and physical activity for older adults. This should be reviewed by the serious student of the subject.[1] Recommendations include endurance training to help maintain and improve cardiovascular function and strength training to help offset loss of muscle mass. Standards for exercise programs for subjects with cardiovascular disease have been available from the American Heart Association since 1979.[14] Additional benefits from regular exercise include improved bone health, reduction of risk from osteoporosis, improved postural stability, and increased flexibility and range of motion. Psychological benefits include a feeling of well being, relief of symptoms of depression often associated with the elderly, and a general *joie-de-vivre*.

ENVIRONMENTAL STRESSES AND ELDERS

Environmental variables encountered in the great diversity of outdoor wilderness activities may produce significant physiologic stresses. These variables include extremes of heat and cold, high altitude, water immersion, tropical humidity, desert aridity, and ultraviolet exposure. The common denominator in nearly all wilderness ventures is physical activity, often at extreme levels. To compound the complexity of physical activity influenced by environmental stress, the physician may have to deal with a senior afflicted with subclinical or manifest disease. When the physiologic demands from environmental stresses are added to the increased and prevalent degenerative conditions and diseases associated with aging, risk for illness and injury is multiplied. The complete package of age, conditioning, environment, nature of the activity, and experience must be considered when an elder is advised or treated in the wilderness.

Heat (See also Chapters 4, 10, and 11)

Tolerance to heat depends on characteristics of the host, including health status, medications, frequency and duration of exposure, history of recent acclimatization, and prevalent environmental factors. Industry has considered levels for permissible exposure limits (PEL), threshold limit values (TLV), and standards for maximal exposure, but there has been no consensus on an exact environmental stress index for heat.

Elders in a hot wilderness setting may have personal host characteristics, in addition to the environment, that further limit tolerance and safety. Weight, fractionated body mass, cardiovascular, renal or pulmonary problems, and the presence of

Box 89-1. Body Signals

PRIMARY

Cardiovascular

Heart rate: used to estimate physiologic effect of physical workload
Pounding in head: possibly related to physiologic or pathologic systolic hypertension
Chest pain: physiologic or pathologic
Fluttering in chest: arrhythmia

Respiratory

Mouth breathing: 60% of maximal physical work capacity
Breathing too hard to talk: >85% maximal physical work capacity
Cough: dry air; early high-altitude pulmonary edema (HAPE)
Cheyne-Stokes respiration: high altitude
Cyanosis: nails, nose, earlobes (consider hypoxia)

SECONDARY

Musculoskeletal

Tremulousness: quadriceps weakness and cramps (consider volume depletion and hyponatremia)
Shivering: cooling, body temperature still >96° F (35.5° C)
Stopped shivering with confusion: possibly further dangerous cooling, probably <93° F (33.9° C)
Sweating: air temperature/humidity or heavy physical work above 7 METs

Gastrointestinal

Abdominal distention (with flatus), high-altitude flatus expulsion (HAFE)
Dry mouth: dehydration
Loss of appetite: altitude or dehydration

Genitourinary

Urinary frequency: altitude (the *Hochdiurese* ["high-altitude diuresis"] of alpine mountaineers)

Sensory

Tubular vision: altitude/hypoxia
Loss of color discrimination: hypoxia
Loss of night vision discrimination: hypoxia
Visual central white spot: hypoxia
Sense of touch: wind against cheek >5 mph
Taste: diminished with dehydration and hypoxia
Smell: diminished with dehydration and hypoxia

Neurologic

Headache: altitude, high altitude cerebral edema, acute mountain sickness
Lack of analytical decisiveness: altitude, hypoxia
Confusion and irrational behavior: altitude, hypoxia, possible cerebrovascular abnormalities

Box 89-2. Contributors to Heat Exposure Vulnerability in Elders

1. Obesity
2. Decreased physical functional capacity
3. Infrequent heat exposure
4. Altered thermoregulatory center in the hypothalamus or insensitive skin sensors
5. Metabolic and serum electrolyte abnormalities
6. Heart disease, coronary artery disease, pulmonary disease, diabetes, and renal disease
7. Peripheral vascular disease
8. Multiple medications, often in combination: anticholinergics, antipsychotics, tranquilizers, and beta blockers
9. Alcohol

various medications may influence individual response to heat.

Regulation of body heat may be affected by altered function of the thermoregulatory center located in the anterior preoptic hypothalamic nuclei, by deranged skin sensors, or by medications used to treat various diseases. These include anticholinergics, beta-adrenergic blockers, antipsychotic medications, and major tranquilizers. Side effects influence adaptation of sweat mechanisms to thermal stress. Diuretics may produce hypovolemia with loss of adequate subcutaneous circulation for heat dissipation. Because elders as a rule consume more medications than younger persons, it is very important to approach heat injury from a position of prevention.

The cardiovascular system plays a major role in heat regulation through heat dissipation. Circulatory abnormalities, peripheral vascular disease, hypertension, and reduced cardiac output may modify heat dissipation, resulting in vulnerability to heat injury. Physical work capacity as measured by maximal oxygen consumption ($\dot{V}O_2$ max) decreases 5% to 15% per decade after age 25 years. Beta-adrenergic blockers and calcium channel blockers may also influence cardiac output by modifying heart rate and myocardial contractility.

To prevent heat-related illness associated with wilderness activities in the elderly, it is sometimes helpful to suggest a regular exercise program in the heat for adaptation. A regular program consisting of 60 to 100 minutes of low-intensity exercise per day for 7 to 14 days at tolerable heat levels before the planned exposure should result in significant adaptation in normal individuals. The exercise level should require an oxygen consumption of less than 50% of the individual's $\dot{V}O_2$ max. Experience teaches us that a degree of adaptation results from frequent and extended periods of exposure.

Acclimatization to heat yields improved response to exercise generally. Physiologic responses to adaptation include lower heart rate, enhanced tolerance to physical activity, predictable core temperature in response to heat stress, increased sweat rate, and decreased sodium loss through sweating.

Additional environmental factors, such as high humidity, high winds, and infrared and ultraviolet radiation exposure, may modify levels of an individual's tolerance to heat, partly through skin changes. It is valuable to teach individuals to be aware of the environment and associated responses such as warmth, coldness, or dampness in the skin. It is also helpful to advise the prospective wilderness venturer that, as a general rule, it takes a breeze of greater than 5 knots (5.75 mph) to be felt on the skin of the face. This is one of a large group of recognizable somatic and sensory observations known as "body signals" (see Box 89-1). Characteristics of elders who are vulnerable to heat exposure are listed in Box 89-2. Actions that may help prevent heat injury are suggested in Box 89-3.

Box 89-3. Prevention of Heat Injury in Elders

1. Assess the health status, with particular emphasis on history, cardiovascular status, obesity, and previous history of problems associated with heat exposure.
2. Maintain adequate hydration.
3. Maintain adequate nutritional status: food, fluid, and electrolyte intake.
4. Use estrogen replacement therapy where indicated (see Clinical Medicine, Menopause).
5. Participate in a proper acclimatization program.
6. Avoid the use of superfluous medication.

Cold (See also Chapters 4 to 8)

Cold exposure is poorly tolerated among the elderly. The complex mechanisms that control body temperatures in the elderly are not as responsive as in younger people. Peripheral vasoconstrictive response to cold is diminished. Systolic hypertension through stimulation of the sympathetic nervous system is exaggerated in a cold environment. Cardiac workload is increased; consequently, in the presence of coronary artery disease, angina is frequently precipitated by exertion and cold. Four avoidance factors for persons with coronary artery disease include exertion, emotion, eating excessively, and exposure to cold, the "four E's of angina."

With aging, diminished metabolic rate occurs. When associated with age-related reduction in muscle mass, the shivering response is blunted and there is reduced capacity for heat generation.

Exhaustion added to hypoglycemia and dehydration compounds the problem of impaired metabolic function, making the elder individual more vulnerable to the effects of cold. Adequate food intake is essential for maintaining body heat and may become critical. Other physical environmental influences, such as wind, humidity, ultraviolet and infrared radiation, and altitude, should be factored into the exposure equation. The wind chill index provides a useful teaching device for reminding explorers about the hazards of combined cold and wind. The classic combination of cold, dampness, wind, and exhaustion may prove fatal, especially in an elder with decreased physical reserve.

Medical conditions such as cardiovascular disease, metabolic diseases such as hypothyroidism and diabetes, compromised nutritional status, and modified thermoregulatory responses resulting from central nervous system disease or medication may influence heat conservation and contribute to hypothermia. Heat loss may also be increased by damp, wet clothing. All persons should be cautioned to carry ample clothing for changes after saturation with moisture.

Peripheral vasoconstriction, the fundamental mechanism for heat conservation, may be enhanced to some small degree by long-term exposure to cold. When an elder recognizes personal intolerance to cold, he or she may begin a program of gradual increase in exposure to cold. Prevention of cold injury, however, is best achieved through a learning process derived from experience. Elders should never venture unaccompanied into the cold wilderness. Judgment, orientation, and independent responsibility may be impaired in elders who find themselves lost in the cold or in a rescue situation. Characteristics of elders who are vulnerable to hypothermia and cold injury are listed in Box 89-4. Some good preventive measures are suggested in Box 89-5.

Box 89-4. Characteristics of Elders Vulnerable to Cold Exposure

1. Peripheral vascular disease (impaired vasoconstriction)
2. Hypertension (perhaps cold-induced)
3. Heart disease, including coronary artery disease, decreased cardiac output, congestive heart failure
4. Metabolic diseases (diabetes, obesity, hypothyroidism)
5. Hematologic disorders (anemia, dysproteinemias)
6. Pulmonary disease (cold-induced asthma, chronic obstructive pulmonary disease)
7. Drugs and alcohol
8. Medications, particularly beta blockers and tranquilizers

Box 89-5. Prevention of Cold Injury in Elders

1. Avoid exhaustion during wilderness ventures.
2. Limit exposure.
3. Carry and wear adequate clothing, including rain gear.
4. Stay dry and avoid damp undergarments from excessive sweating.
5. Maintain adequate nutrition with high carbohydrate intake and fat. Carry adequate food for the trip.
6. Maintain adequate fluid intake. Do not consume snow or ice.
7. Participate in a pre-expedition physical training program.
8. Pay attention to medication effects.
9. Avoid alcohol and illicit drugs.
10. Always maintain access to an adequate shelter.

Altitude

The effects of altitude and incidence of altitude illness among the elderly are not clearly understood in spite of extensive study on altitude among younger, more vigorous subjects. Hackett[19] reported that aging reduces physiologic components of the gas exchange process that maintain oxygenation, such as vital capacity and hypoxic ventilatory drive. Older persons are known to have a lower arterial Po_2 because of the thickening of the pulmonary alveolar-capillary membrane.[27]

Houston,[23] Honigman,[22] and others studied the general population at moderate altitude (2000 to 3000 m [6500 to 9750 ft]) elevation at ski resorts in Colorado. Predictors of mountain sickness included chronic residence at altitude greater than 1000 m (3250 ft) before a high altitude venture, underlying lung problems, previous history of acute mountain sickness ($p < 0.05$), and, surprisingly, age younger than 60 years. This apparent paradox probably is due to natural self-selection among the small group of elderly individuals who are exposed to altitude and activity.

Although 25% of visitors to moderate altitude develop some degree of mountain sickness, data reflect a protective effect from living at moderate elevations of 1500 to 2000 m (4900 to 6500 ft), which is consistent with empiric observations that altitude staging reduces the incidence of acute mountain sickness.

Honigman and colleagues[22] found that the most frequent predictors of acute mountain sickness are (1) altitude of greater than 1000 m for residence, (2) history of previous episode of

Box 89-6. Characteristics of Elders Vulnerable to Altitude Illness

1. Abrupt ascent in altitude from near sea level to 3000 m (9750 ft) or greater, without an extra night for acclimatization for every additional 600 to 900 m (1950 to 2900 ft) of continuing ascent
2. History of previous episode of altitude sickness
3. Preexisting lung disease characterized by decreased capacity and decreased hypoxic ventilatory response
4. Preexisting cardiovascular disease
5. Metabolic abnormalities associated with diabetes and renal disease
6. Medication that influences respiratory drive
7. Low physical functional capacity
8. Obesity

Box 89-7. Prevention of Altitude Sickness in Elders

1. Avoid strenuous physical activity at altitude if there is a history of acute mountain sickness.
2. Limit the intensity of physical activity in the presence of cardiovascular disease.
3. Limit physical activity in the presence of pulmonary disease, especially if there is decreased vital capacity.
4. Avoid significant physical activity for at least 12 hours after arrival at an altitude 2500 m (8100 ft) or more above sea level, and delay physical activity for an additional 24 hours for every 600 to 900 m (1950 to 2900 ft) altitude gain.
5. Be aware of all medications and their effect on hypoxic ventilatory drive.
6. Use carbonic anhydrase inhibitors (acetazolamide) according to recommendations of the venture leaders, considering the altitude, medical history, and other medications.

acute mountain sickness, (3) age younger than 60 years, (4) poor or average physical condition, and (5) lung disease. The risk of altitude sickness among elders is increased by poor physical condition, alcohol intake, preexisting pulmonary disease, medication, and excessive activity within the first 12 hours after arriving at altitude. Alcohol tolerance is variable, so total abstinence during a wilderness venture is recommended. It is prudent to avoid sedatives and hypnotics at high altitude.

Acetazolamide, 62.5 mg at bedtime, is the drug of choice for prophylaxis against periodic breathing.[20] Acute mountain sickness may warrant 125 to 250 mg up to twice a day for treatment. Elders may experience side effects such as weakness, nausea, and paresthesias with large doses, so caution is encouraged. Characteristics of elders who are vulnerable to altitude illness are listed in Box 89-6. Some good preventive measures are suggested in Box 89-7.

CLINICAL MEDICINE IN ELDERS

Wilderness medicine is in part defined as traditional medicine in nontraditional settings. The principles of risk, pathophysiology, and treatment of health problems are those of the established medical community. However, characteristics of the acute care, setting, and environmental conditions affect care, search and rescue, transportation, and medical support. When elder age is a factor, an additional set of concerns is added, including reduction in physical reserve, increase in rate of physical deterioration during exercise, and reduction in margin of safety. Enhanced skills of analysis, planning, intervention, and action are demanded of the rescuers and medical team.

There are two important occasions when wilderness medical leaders should consider health/medical evaluation for elderly venturers: (1) during the planning phase before the venture and (2) following symptoms that may occur during or after the venture. Components of the pretrip evaluation are based on the characteristics of the individual and on the nature of the proposed venture. These are discussed in the section on Medical Examination. Evaluation after the onset of symptoms or for intervention requires clinical judgment dictated by the specific problem or symptom and is considered in discussions of specific illnesses.

CARDIOVASCULAR DISEASE

Age-related changes in the cardiovascular system affect heart rhythm, myocardial pump function, and afterload. With aging, there is progressive reduction in the number of pacemaker cells in the sinoatrial (SA) node, increase in myocyte cell volume per nucleus in both ventricles, increase in peripheral vascular resistance, and increase in aortic impedance from loss of elasticity. Increased levels of circulating catecholamines are associated with aging, especially with stress, but beta-adrenergic–induced vasodilation decreases with age. This is important for exercise tolerance in elders.

Cardiac rhythm disturbances range from the nuisance effect of premature systoles to significant rhythm disturbances, such as atrial fibrillation, to potentially life-threatening ventricular arrhythmias. An elder who is aware of symptoms related to an arrhythmia should seek evaluation by a physician before a wilderness venture. The evaluation should include history of onset of symptoms and initiating factors, medications taken, physical examination with emphasis on auscultation of the mitral and aortic valves, electrocardiogram, ambulatory monitoring if there is a history of heart disease or light-headedness, and possibly an echocardiogram.

Valvular changes, especially of the aortic and mitral valves, may be recognized by auscultation and confirmed by echocardiography, and perhaps by cardiac catheterization. The subject with a prosthetic cardiac valve may be at risk during prolonged ventures with exposure to inclement weather, infectious diseases among fellow venturers, or diseases among the local populace.

The presence of pump failure, either systolic or diastolic, may be indicated by recent weight gain, shortness of breath, orthopnea, or ankle edema. It is confirmed by physical examination, electrocardiography, echocardiography, and chest x-ray. Although digitalis, diuretics, and angiotensin I converting enzyme (ACE) inhibitors may relieve symptoms of systolic dysfunction, the subject with heart failure should limit remote wilderness ventures to those activities that do not cause excessive shortness of breath, that is, to the point of requiring breathing through the open mouth, or excessive fatigue.

Peripheral vascular disease, claudication, symptomatic carotid disease, and aortic aneurysms warrant caution in regard

to the difficulty of return or evacuation in the event of incapacity.

Coronary Artery Disease

A heart attack is the ultimate medical emergency. In the United States, approximately 1.5 million persons sustain myocardial infarctions annually, 30% of whom do not survive. Of these 50% die within the first hour of the onset of chest pain. Because of the remoteness and delay in instituting treatment, heart attack in the wilderness is high risk for poor outcome. If a victim survives the first hour, which may be the time required to initiate a search and rescue, there may be approximately a maximum of 85% chance of survival expected subsequently, using urban statistics.

Only half of people with myocardial infarction have premonitory symptoms, but interrogation about this during a medical history could alert one to the potential for myocardial infarction. The history should include a detailed assessment of risk factors for coronary artery disease. Subsequent physical examination and testing may provide recommendations aimed at reducing life-threatening cardiac events.

Many physiologic stresses related to environmental extremes have as a common denominator an increase in catecholamine output with a concomitant rise in heart rate, blood pressure, and cardiac output. This results in increased cardiac workload and myocardial oxygen demand.

Because the myocardium depends almost entirely on aerobic metabolism, workload is reflected in myocardial oxygen consumption. Delivery of oxygen to the myocardium depends on coronary blood flow. Hence, integrity of the coronary arteries is essential for a viable response to wilderness stresses.

Knowledge of the existence and extent of coronary artery disease will influence physician recommendations for the nature and level of activity, expected tolerance to environmental stresses, and recommendations as to the degree of remoteness, level of exertion, and environmental factors that the individual should endure in the proposed wilderness ventures.

Cardiovascular Assessment Prior to Wilderness Venture

Since there is no absolutely risk-free activity for a person with significant cardiovascular disease, prevention is the fundamental principle for reducing risk. Before vigorous outdoor activity by an elder, the physician should:

1. Question the individual regarding planned activities
2. Have a knowledge of the planned activity and be aware of anticipated exertion and environmental circumstances
3. Have a thorough understanding of any previous history of coronary artery disease, other illnesses, and medications
4. Be prepared to make specific recommendations regarding physical activity and exposure (see Medical Examination)

There are currently no published guidelines to aid in advising elderly individuals intending to venture into the wilderness, but the American College of Cardiology 26th Bethesda Conference produced "Recommendations for Determining Eligibility in Competitive Athletes with Cardiovascular Abnormalities." These guidelines divide patients into two categories: (1) those considered to have mildly increased risk have a left ventricular ejection fraction (LVEF) of greater than 50% (or revascularization of such lesions), and (2) those with a substantially increased risk have an LVEF of less than 50%, exercise-induced myocardial ischemia, exercise-induced complex ventricular arrhythmias, and occlusive lesions of greater than 50% in one or more coronary arteries. Transposing the recommendations for athletes to the elderly, people with mildly increased risk are advised to restrict activities to levels of low dynamic activity, that is, activities requiring energy expenditure equivalent to walking, and a low to moderate static level equivalent to lifting less than 15 to 20 lb (7 to 9 kg). Those persons with substantially increased risk are advised to participate only in low-level dynamic and static activities.

Persons with atrial fibrillation, but without structural heart disease, who can maintain an appropriate heart rate response to exercise have no limitations. For persons with atrial fibrillation, structural heart disease, and an appropriate rate response to exercise, activities consistent with the limitations of the structural heart disease are allowed. Persons with atrial fibrillation requiring anticoagulation should not participate in activities risking physical contact and injury.

In considering these recommendations, one must remember that these guidelines were developed for competitive athletes and are not necessarily applicable to wilderness and recreational activities. Nevertheless, the guidelines provide a framework for an initial evaluation.

A fascinating philosophical discussion dealing with the limitations of wilderness activities in people who have undergone cardiac surgery appeared in a series of letter exchanges and editorials in the *Journal of the American Medical Association*. The conclusion places the decision-making process squarely on the clinical judgment of the cardiologist on a case-by-case basis.[35]

Hypertension

Hypertension, the "silent killer," is an important marker for potential cardiovascular problems. Cardiovascular mortality has been shown to be three times as high in hypertensive elders compared to those with normal blood pressure. Complications include angina, myocardial infarction, left ventricular hypertrophy, heart failure, stroke (both ischemic and hemorrhagic), and renal failure. The most widely accepted values of blood pressure considered to represent hypertension are systolic above 140 mm Hg or diastolic over 90 mm Hg. In a health survey in England,[34] systolic blood pressure over 160 mm Hg was observed in 35% of men and 37% of women aged 65 to 74, increasing to 41% and 49%, respectively, after age 75 years. Isolated systolic hypertension is almost exclusively a disorder of elders, with a prevalence of 0.8% at age 50 years, 12.6% at 70 years, and 23.6% at 80 years.[39]

There is an increased effect of oral sodium intake on blood pressure in the elderly. An increase of 100 mmol/day is associated with a 10 to 15 mm Hg rise in systolic pressure, compared with a 4 to 5 mm Hg rise in younger adults who ingest the same sodium load.[28] In elders, isolated systolic hypertension is recognized as a predictor of stroke.

Treatment of hypertension is mandatory before elders venture into the wilderness. Medications, however, may cause significant side effects. Diuretics, an integral part of treatment, may deplete volume and electrolytes such as potassium and sodium. Beta-adrenergic blockers, of particular value for the systolic component, may limit heart rate response to activity or modify body temperature control during exposure to heat or cold. Calcium channel blockers are of value in treatment of hypertension, but headache, flushing, and edema are reported in about one third of users. The least complicated treatment for hypertension is control of obesity and restriction of dietary sodium intake.

GASTROINTESTINAL DISORDERS

There is an increased prevalence of certain gastrointestinal disorders among elders. The most common are constipation and diverticulitis. Constipation is a frequent complaint among 26% of elderly men and 34% of elderly women. There is a difference between the medical definition and elder perception of constipation and their need for laxatives. Normal frequency of bowel movements ranges from three defecations per day to three per week. However, laxatives are used by 15% to 30% of elders on a regular basis. When such individuals find themselves in the wilderness, away from the convenience of their bathrooms, alteration in bowel habits, sometimes to the point of fecal impaction, may occur. Diverticulitis may flare at such times, requiring dietary change, stool softeners, and antibiotics. It is prudent to obtain a detailed history of bowel habits and medications before embarking on a wilderness venture. Dietary fruit, fiber, and grain with stool softeners for elders during a wilderness venture may help avoid impaction, rectal fissures, hemorrhoidal bleeding, fecal incontinence, and chafing.

Gastrointestinal disorders noted among elders may also include malignancies of the colon and pancreas, gastric ulceration that may bleed, especially after nonsteroidal anti-inflammatory drug (NSAID) ingestion, and *Helicobacter pylori* infection. Prospects for bleeding or obstruction from colon malignancies, bleeding from gastric ulcers, and possible perforation of ulcers should alert the individual or group leader to the need for medical attention. A surgical emergency may arise from an incarcerated or strangulated inguinal or ventral hernia.

Diarrhea among elders contracted in Third World countries may be severe, leading to dehydration, gastrointestinal bleeding, or perforation of a hollow organ. Exacerbation of diverticulitis or acute cholecystitis, with or without pancreatitis, persistent or unusual abdominal pain, vomiting, bleeding, and worsening dehydration are reasons for ending the venture and initiating evacuation to medical care.

When traveling to remote countries, it is prudent for expeditions to carry intravenous (IV) fluids and to consider appropriate antimicrobial prophylaxis or medications for the presumptive treatment of infectious diarrhea.

GENITOURINARY DISORDERS

Elders are particularly vulnerable to urinary tract disorders, including infections, obstructions, and ureteral, renal, or bladder stones. Bladder infections in women are common in remote settings because of dehydration and difficulty with hygiene. Incontinence after hysterectomy is frequent, and discomfort from chafing and bladder infection may be present. It is prudent for women to carry medicated skin pads and cotton diapers. Men may incur bladder infections from benign prostatic hypertrophy with obstruction. Anticholinergic medications may induce bladder relaxation with subsequent retention. Terazosin hydrochloride, an α_1 selective adrenoreceptor blocking agent, may improve urine flow in instances of obstruction resulting from benign prostatic hypertrophy by reducing bladder outlet obstruction without affecting bladder contractility. On extended trips in remote areas, it may be prudent to carry a catheter for emergency relief of acute urinary retention.

Dehydration from fluid loss or inadequate oral replacement may precipitate infection. Adequate fluid intake is the key to genitourinary health in the wilderness, but the participant must be cautioned about water quality.

MENOPAUSE

Menopause in women is a normal phenomenon associated with aging. Rather than occurring in a discrete event, the menopause transition may take place over a period of several years, beginning around 40 years of age or as late as 50 to 55 years of age. Also known as the climacteric, this is a time in life when the opportunity for leisure activities may be greatest. Women who participate in wilderness activities must deal with the symptoms and somatic changes of menopause in addition to the physical and environmental stresses of the wilderness.

Symptoms that foretell the onset of menopause include vasomotor flushing, night sweats, insomnia, vaginal dryness, and variation in menstrual cycle and flow. Convention accepts that 12 months of cessation of menses is a confirmation of menopause.

As ovarian production of estrogen declines, the androgen/estrogen ratio changes dramatically. Gonadotropin feedback results in increased follicle-stimulating hormone (FSH) in a range of up to 30 MIU/mL. Progesterone secretion is variable and may either increase or decrease.[38] Resulting anatomic changes from these hormonal alterations may affect lipid ratios, the coronary arteries, cortical and trabecular bone, and changes in body fat distribution with a shift in fat toward the center of the body. Changes in cognitive functions have also been reported.

Vulnerability to osteoporotic fractures is a serious hazard for elderly women in the wilderness. In trabecular bone found in the spinal column, resorption and formation occur 4 to 8 times as fast as that of cortical bone. As a result, there is an increased risk of compression fracture of the spine or fracture of hip in the event of a fall.

Hormonal replacement therapy should be considered for menopausal or postmenopausal women or in those who have had surgical hysterectomy. The three classic indications for hormone replacement therapy are: (1) for symptoms related to estrogen deficiency, such as vasomotor symptoms or genitourinary tissue atrophy; (2) for prevention or treatment of osteoporosis; and (3) for prevention of cardiovascular morbidity and mortality, all of which may occur in the wilderness setting.

Combinations of estrogens and progestins may be used in treatment.[25] Examples include:

- Daily estrogen as 0.625 mg conjugated estrogen or 1 mg micronized estradiol
- Daily progestin as 2.5 mg medroxyprogesterone acetate or 0.35 mg norethindrone

Transdermal route of administration may be convenient in the wilderness setting.

MUSCULOSKELETAL DISORDERS

Musculoskeletal disorders are common among elders. In the wilderness, joint stress associated with extended hiking, injuries from repetitive motion, falls, and other trauma, when superimposed on the aging body, can induce functional impairments. Limitations in mobility and from pain lead to a loss of success or even to significant hazards during wilderness ventures.

Arthritis, found in about 15% of the U.S. population in 1990, is among the most common causes of disabilities in elders. More significant is a reported prevalence of nearly 50% among persons 65 years of age or older.

Musculoskeletal conditions with increased incidence in adults over age 50 years include osteoarthritis, osteoporosis, polymyalgia rheumatica, giant cell arteritis, gout (especially associated with diuretic use), and other crystal-associated arthritides such as calcium pyrophosphate deposition disease, basic calcium phosphate deposition disease, rheumatoid and other inflammatory arthritides, and spinal stenosis.

The aging joint undergoes degenerative changes in its components. Cartilage has very little ability to heal. Therefore, injuries to cartilage tend to accumulate with age, leading to irreversible damage and osteophyte formation, developing into hypertrophic osteoarthritic change in joints of the extremities and spine.

Skeletal muscle undergoes specific age-related changes with loss of muscle mass. Changes in hormones, growth hormone/insulin-like growth factor-1 (GH/IGF-1), the androgen/estrogen ratio, cytokines/growth factor, interleukin-6, and free radical production may be related to these atrophic changes.

The syndromes causing loss of muscle mass in elders are either (1) intrinsic, from sarcopenia, resulting in diminished reserves of muscle mass, or (2) extrinsic, from failure to thrive (FTT) syndrome, diseases, or increased metabolic demands.

Prevention and treatment of sarcopenia and FTT include a regimen of exercise, including both aerobic training and strength training. Progressive resistance training (PRT) is effective in the treatment of sarcopenia. Lean mass is lost along with bone mass at menopause. Estrogen replacement appears to be helpful in some postmenopausal women. Evidence supporting testosterone replacement for FTT in elderly men is lacking.

A detailed history and physical examination, with emphasis on prior and present symptoms, is of value for an elder contemplating physical activity and is essential for an elder planning remote wilderness activities. Although radiologic studies and laboratory tests may help establish a diagnosis, a common tendency is to rely too heavily on the radiograph to explain symptoms. Joint aspiration and synovial fluid analysis helps in the diagnosis of metabolic or inflammatory arthritides.

Treatment of arthritis in a layered stepwise manner begins with rest, acetaminophen, NSAIDs, and topical capsaicin. Elders especially must be cautioned about the gastric irritation from NSAIDs and their danger in the presence of comorbid conditions and anticoagulant therapy. Intra-articular corticosteroids may help reduce inflammation. Injection serves the extra function of providing access for aspiration of synovial fluid from a joint effusion.

Joint replacement, especially of the hip and knee, has made it possible for otherwise limited individuals to return to wilderness activities. The procedure, known as arthroplasty, is performed for relief of pain and enhancement of function in a joint whose cartilage has been damaged by trauma, infection, or arthritis.

In the traditional total hip replacement, the integrity of the prosthetic femoral head–acetabulum ball and socket joint is maintained.

Sometimes joint resurfacing is possible. In this procedure, resurfaced joint space is capped and a metal prosthesis may be possible. Smoothing, reshaping, and cementing the artificial joint leads to a 3- to 4-day hospital stay, especially with knee replacement, followed by 4 to 8 weeks of limitation, subject to variation based on age, general health, and other factors. Total rehabilitation after surgical hip replacement takes at least 6 months, and wilderness activity during that time should be discouraged.

There are risks from joint replacement, such as infection, clotting, embolization, dislocation, and breakage. Infection may occur immediately after surgery or years later and may result from infection elsewhere in the body. In the rare case when infection does not respond to antibiotic therapy, the joint must be replaced surgically. A temporary artificial hip, such as Prostalac, may be inserted for 3 months, allowing the patient some movement until the joint is permanently replaced.

Physical therapy before and after the onset of symptoms, including body weight reduction, PRT, aerobic conditioning, and spa therapy, may be effective in treatment or when preparing for a wilderness venture. Corsets, braces, and canes prescribed by the treating specialist should be used, but the layperson is cautioned against self-prescription.

A conditioning program before the venture may help prevent musculoskeletal problems or may precipitate symptoms that alert the individual to a problem not previously recognized. It is better for symptoms to occur at home and serve as a warning for potential future untoward events than to occur in a remote location far from medical care. Because degenerative changes in the lumbar and cervical spine may become symptomatic, it is appropriate to carry a backpack during a conditioning program before a hiking venture. At least one and preferably two hiking sticks are recommended for hiking activities.

GROWTH HORMONE IN ELDERS

With advancing age, there are decreases in lean body mass, body fat, and protein synthesis. Growth hormone (GH) deficiency is associated with changes in body composition and protein metabolism similar to those of aging. With advancing years, men and women with no clinical evidence of pituitary abnormalities show decreases in GH secretion and serum levels of insulin-like growth factor 1 (IGF-1). Administration of recombinant human GH (rhGH) to these GH/IGF-1–deficient elders has been reported to result in improvements in nitrogen balance, increase in lean body mass, and decrease in percent body fat. Studies also suggest that there may be an increase in lumbar vertebral bone density but no significant change in the mineral density of the radius or proximal femur. Long-term administration of GH has been reported to cause some side effects, including edema, hypertension, carpal tunnel syndrome, and arthralgia.

Further studies are needed to determine if normalization of GH and IGF-1 levels in healthy elders will lead to improvements in the physical and functional quality of their lives. The prospect of improved muscle mass and strength for elders considering wilderness activities should be followed closely by those interested in human performance.

NEUROLOGIC DISORDERS

Organic neurologic disorders, clinically apparent or subclinical, are frequent among elders. Symptoms of neurologic problems may be precipitated by minor activities or environmental stresses in the wilderness. Physical activity, to which a partici-

pant may be unaccustomed, especially when extreme environmental conditions are superimposed, can make subtle changes more apparent. However, if a victim responds with denial, the result can be devastating.

A history of ischemic or hemorrhagic stroke that in the past resulted in a degree of paresis, especially when accompanied by symptoms such as dizziness, changes in rational thought, or seizure, should alert leaders to potential problems in a wilderness venture.

Altitude, especially above 2500 m (8200 ft), may induce changes in mentation not apparent at lower levels. Leaders should be especially diligent in the assessment of individuals with a previous history of altitude sickness or who are taking medications, including anticoagulants, for cerebrovascular disease.

THE SENSES

Our senses are our warning system for hazards in the wilderness. The five classic sensory "instruments" of vision, smell, taste, touch, and hearing (including its vestibular function) send signals to the central nervous system during interpretation of the physical environment. With age, these sensors undergo functional degeneration. Up to 75% of elders have visual and auditory impairments not reported to their physicians. Changes may be subtle. Again, the aging rule is 1% loss per year after age 30 years of physiologic function in most organ systems.

Vision

There is decreased visual acuity caused by morphologic changes in the lens, choroid, retina, macula, rods and cones, and other neural elements and by an increase in intraocular pressure. Night vision and color vision are notably decreased after age 50 years. Hypoxia, to which the elder's eye may be sensitive, may cause tunnel vision. This may be a valuable body signal indicating an altitude that may be hazardous to the individual.

The most troublesome ophthalmologic effects of age are glaucoma and changes in refractive power. Increased intraocular pressure may be associated with halos and declining vision. This is a serious medical condition warranting continuing care by an ophthalmologist. Manifestations and treatment of farsightedness range from purchasing a pair of nonprescription reading glasses to elaborate multipower prescription lenses and surgical correction of lens abnormalities. Negotiating rough terrain may be difficult while wearing bifocals. An older person walking on a wooded trail may be unable to see roots or rocks in his or her path. Trifocals and various lens designs have been tried, but experience suggests that trail glasses should be configured for distance, with separate reading glasses for close-up work such as map reading.

Complete healing after eye surgery should be confirmed by the ophthalmologist before the adventurer heads into the wilderness, especially with altitude changes.

Decreased tear secretion and "dry eyes" may affect elders in a dusty wilderness environment.

Smell

Much of the pleasure of wilderness exposure is the aroma of trees, flowers, and an array of botanical treasures. With age, the olfactory bulbs undergo significant atrophy, resulting in a form of senile anosmia. Olfactory memory, fortunately, is powerful.

Taste

Some lingual papillae are lost because of age, diminishing the ability to taste. Salivary secretion is reduced. Dentures may cover secondary taste sites. Because olfactory bulbs also undergo atrophy with age, combined taste and olfactory sensory deprivation may account for decreased "pleasure" of trail food taste.

In the event of serious facial injury, the first responder should examine the mouth carefully for fractures of dentures, which could cause respiratory obstruction.

Touch

In addition to assisting in fine movement needed for technical work, sensitivity of touch against a hostile environment may be lifesaving. For example, conventional wisdom holds that the threshold for feeling a gentle breeze against the cheek is about 5 miles per hour. Impairment of cold awareness, which originates from stimuli at the end bulbs of Krause in the skin, along with age-attenuated metabolic adaptation to cold, can interfere with signals that alert an individual to assume cover from the weather.

Hearing

Hearing problems are common in elders, affecting 60% of Americans over 65 years. Presbycusis may be the result of previous middle ear infection, vascular disease, exposure to noise, or the natural loss of sensitivity and distortion of signals associated with advancing age. Acoustical trauma may cause a permanent reduction in sensitivity to high-frequency sound, and the failure to hear the "click pitch" of consonants may obscure communications, since consonants often are found at the beginning and ending of words. Speech in that circumstance in the elderly may be interrupted as a continual drone of vowels.

Problems with sound localization may result in loss of directional hearing. An aging brain cannot process confused sound signals as accurately. Add tinnitus and a hostile environment, and the hearing sense loses its value as an important survival tool.

Hearing aids are useful for enhancing volume, sound direction, and pitch discrimination. Traditional analog hearing aids have limitations, especially in high-frequency ranges, obscuring click pitch discrimination. Newer digital devices, some with a range of 500 to 6000 Hz, provide increased clarity and sound quality and can enhance volume, as well as directionality, a valuable warning signal in the wilderness setting. Programmable units can be changed according to needs, such as listening to music and background noise reduction. Unilateral hearing loss can be improved by "cross-over" hearing aids.

In the wilderness setting, a hearing aid user must be cautious to prevent sweat, rain, and other moisture from entering the components. A drying kit containing desiccating crystals is available and should be carried by the wilderness hearing aid wearer.

The "inner ear," or vestibular apparatus, is especially valuable for balance and stability and combined with proprioception may provide a "biological gyroscope" of crucial importance. Increasing age seems to be associated with more frequent episodes of vestibular dysfunction, including vertigo.

There is a "sixth sense" built into the aging wilderness adventurer derived from years of experience and exposure, study, and review of wilderness activities. This is *wisdom!* As a result of assimilating and processing these data, elders frequently acquire a reputation for survival. One who develops this aura is affectionately known as a "salty dog."

NEUROPSYCHIATRIC DISORDERS

Situational stresses are often superimposed on environmental stresses during wilderness ventures. Difficulties with group interaction, changes in rational thought in individuals with organic brain disease, or behavioral upheaval from bipolar states can jeopardize the health and safety not only of the individual, but also of an entire group.

Problems associated with drug abuse or alcoholism are a danger to the individual and are potentially devastating to the group. Subtle alcoholism may evolve into full-blown withdrawal psychosis during a hypoxic event or during extreme physical exertion. Drug use in elders may not be "recreational," but withdrawal from ethical drugs may be quite severe. Leaders of wilderness ventures must recognize the danger of disruptive psychiatric problems and attempt to prevent them by careful screening before the venture.

As people age, a variety of cognitive disorders become apparent, including progressive dementia, confusional states, and cognitive disorders resulting from psychiatric syndromes. Symptoms may be precipitated in vulnerable individuals by physical or environmental stresses of the wilderness. The two usual sources of information concerning the status of patients are patients' families and the patients themselves. It is rare for a family member to approach the physician with concerns about the cognitive function of a relative; thus, an interview and brief mental status testing should be included in the medical examination.

A cognitive interview should provide the examiner with insight into prior cognitive skills and personality traits to serve as a baseline. Included should be the nature of the subject's memory at that time, the interval since onset of symptoms, the nature of onset (slow or sudden), and current state of cognitive function.

Frontotemporal dementia, such as Pick's disease, may manifest itself early with a change in personality with inappropriate and often volatile behavior. Alzheimer's disease is frequently manifested by apathy and decline in ability to learn new information. Physician assessment of cognitive function and neuropsychiatric disorders may identify potential disaster during wilderness undertakings.

DIABETES MELLITUS AND OTHER METABOLIC DISORDERS

Type 2 ("adult-onset") diabetes mellitus, whether requiring insulin or controlled by oral hypoglycemic medication, is not necessarily a contraindication to wilderness activities; however, other problems related to diabetes may increase risk of medical emergencies. Cardiovascular disorders, coronary artery disease, cerebrovascular disorders, peripheral and autonomic neuropathy, nephropathy, poor wound healing or infection, and vulnerability to frostbite and cold injury all may be associated with this metabolic disorder.

Insulin-dependent (type 1) diabetes is a very different situation because of the additional problems with caloric intake and energy expenditure. Hypoglycemic episodes may lead to accidents from mental confusion. The vestibular apparatus in elders is particularly sensitive to such episodes. The "buddy system" should be deployed to assist the insulin-dependent diabetic in the wilderness. Replacement therapy of other metabolic disorders should be maintained as recommended by the endocrinologist.

OBESITY

Almost two thirds of adults in the United States are overweight and over 30% are obese, according to National Institutes of Health data collected from 1999 to 2000.[43] There is widespread recognition that weight has become a very significant problem affecting morbidity, mortality, health care services, production, and economic costs.

The term *overweight* refers to an excess of body weight compared to a set of standards. Excess weight may come from muscle, bone, fat, or body water. *Obesity* refers specifically to an abnormally high proportion of body fat. Because of their high proportion of muscle to fat, some athletes and body builders can be overweight without being obese. Obese people, however, are all overweight.

The most commonly accepted measure for determining overweight status is the body mass index (BMI), reflecting the relation between height and weight. The BMI is derived from the following formula:

$$\text{BMI} = \text{weight (kg)/height squared (m}^2\text{)}$$

Overweight is identified by NIH as a BMI of 25 to 29.9 kg/m^2 and obesity as a BMI of 30 kg/m^2 or greater. The World Health Organization also recommends defining overweight as a BMI of greater than 25.[43]

Illnesses associated with overweight and obesity include diabetes, hyperlipidemia, hypertension, heart disease, stroke, osteoarthritis, respiratory problems, sleep apnea, gallbladder disease, menstrual irregularities, complications of pregnancy, increased surgical risks, depression, and even certain forms of malignancy. Injuries, especially falls with fractures, warrant awareness and caution along trails in the wilderness. Respiratory difficulties and excessive fatigue are also frequent complaints among the obese in the wilderness.

Among people with type 2 non–insulin-dependent diabetes, 67% have a BMI greater than 25. Hypertension is found in 22.1% of men with a BMI greater than 25, but is found in only 14.9% of those with a BMI less than 25.[43] These figures indicate that weight is a risk factor and a predictor of illness. Life expectancy is shortened 2 to 5 years with moderate obesity. Obese people have a 50% to 100% increase in all-cause risk of premature death.[43]

People with obesity-related problems are at risk if they happen to venture into the wilderness. In the event of an emergency requiring evacuation, more effort is required by rescuers of an obese victim as well as by the victim him- or herself.

Many obese individuals who recognize their problems may begin a weight reduction program that includes not only dietary controls but also exercise activities, especially walking. Energy expenditure while trail walking[15] may be calculated from speed of walking and grade of the trail using the following formula:

$$\dot{V}O_2 \text{ (mL } O_2\text{/kg/min)} = \text{speed (m/min) } [0.1 + (1.8 \text{ grade})] + 3.5$$

Caloric expenditure may be calculated from this energy cost. The energy calculated from the above formula may be modified by the physical characteristics of the trail terrain such as soil, asphalt, scree, and snow. Terrain coefficients are available for estimating the increased energy expenditure while traversing difficult terrain.[37]

Until the overweight/obesity epidemic is controlled, obesity-related illnesses and injuries will continue to concern wilderness health care providers.

PHARMACOLOGY, PHARMACOKINETICS, AND POLYPHARMACY

Older people are the major consumers of all categories of medications. Health status and pharmacokinetics of elders influence drug choices, dosages, prospects for adverse reactions, and even therapeutic goals. Physician assessment of elders before wilderness activities should include careful review of all medications.

Age-related physiologic changes that may influence pharmacokinetics include reduced gastric acid production and altered gastric emptying that affect absorption; reduced splanchnic blood flow that affects first pass (or presystemic) clearance; reduced body water, serum albumin, body fat, and body mass that affect protein binding; changes in hepatic size and blood flow that affect hepatic clearance; and reduced glomerular filtration rate and renal tubular function that affect renal clearance. As a result, physicians recognize that it is almost always prudent to begin with a lower starting dose and lower maintenance dose of certain prescribed medications in elderly patients.

Renal clearance of some drugs is reduced as a result of aging. The clinical significance of altered renal clearance resulting in elevated blood levels is, of course, related to the drug. Drugs with a very narrow therapeutic ratio should be carefully monitored. These include antibiotics such as gentamicin, streptomycin, and kanamycin; beta blockers such as atenolol and sotalol; cardiac glycosides such as digoxin; and psychotropic drugs such as lithium.

Hepatic clearance of drugs is of even more concern in the elderly because of a silent reduction in clearance capacity of up to 50% or more in this age group. Causes of reduced hepatic clearance include reduction of absolute liver size, decrease in hepatic blood flow, and perhaps altered enzyme activities. Benzodiazepines, nitrazepam, chlordiazepoxide, midazolam, and colabazepam, eliminated primarily by oxidation, should be reduced in dosage. Oxazepam, eliminated by glucuronidation, also should be given in reduced dosage.

Comorbidity, the presence of multiple pathologic problems, makes drug choice particularly important because of the prospects of worsening a coexisting disease when a prescription is given for another disease condition. For example, thiazide diuretics may worsen control of diabetes; calcium channel blockers may result in ankle edema; beta blockers may increase symptoms of peripheral vascular disease; dopamine blockers given as antiemetics may exacerbate parkinsonism; and NSAIDs may render hypertension, heart disease, or renal failure more difficult to control. Counseling, packaging labels, diaries, and supervision are important instruments for proper control of toxic and side effects of medications in elders.

Excessive use of medicines is often seen.[24] Polypharmacy represents a less-than-desirable state of duplication of medications, failure to recognize potential drug interactions, and inadequate attention to pharmacokinetics and pharmacologic principles.[31] Alcohol abuse frequently compounds the problem of polypharmacy. It is a great help to have each participant carry all presently consumed medications to his or her physician for review before the wilderness venture.

FIRES, STORMS, AND NATURAL DISASTERS

Anything can happen at any time in the wilderness. The suddenness with which fires, storms, and natural disasters can strike threatens not only safety but also survival. Elders, who may be less mobile and more vulnerable than younger people, must be especially alert. They have no business struggling against the elements. For all of these events, the first rule is *evacuate* with the first warning. The second rule is *maintain communication,* if possible, if only one-way by radio. Small emergency radio receivers powered by batteries, photoelectric cells, or crank generator are inexpensive.

For elders, fire hazard is not just flames; it includes the fatigue, smoke inhalation, and possible cardiovascular emergencies associated with the exertion and excitement. Common sense measures that may help if trapped in the area of fire are to seek shelter inside, keep smoke inhalation minimal, avoid strenuous outdoor exercise, drink copious amounts of fluids, and keep containers of water and wet towels available.

Elders are more likely to have experienced the violence of meteorological storms and realize that shelter is the critical defense. Injuries associated with such storms are not just from the dynamics of the weather, but also from the injuries sustained from debris hurled as projectiles by the wind.

FALLS AND SAFETY

Falls are especially common in the wilderness setting and can lead to devastating environmental exposure. Risk-taking invites problems. Planning, prevention, and alertness are essential for safety.

Falls rarely occur because of a single cause; most often they are a result of the interaction among intrinsic deficits, activities, and environmental hazards. Intrinsic deficits among elders impair judgment, sensory input, reaction time, proprioception, balance, and gait. Medication effect often adds to the risk of falls. Individuals with these deficits are vulnerable to environmental hazards such as scree, rock, roots, water, and unstable footing. About one in four elders who fall sustains significant injury and about 5% result in fractures. Known as "the big fall," this life changing event may be followed by progressive deterioration of health and vigor.

Effective prevention entails a review and assessment of individual health status and the proposed wilderness activity by an experienced wilderness leader. On site, many find a walking stick or a lightweight alloy pole may help with stability. Cell phones and radios may speed rescue for the injured. Some principle precautions include the following suggestions:

- Stay together on the venture.
- Don't overestimate your own ability.
- Budget time realistically with rest stops included.
- Teach the team the practical aspects of first aid.
- Cross-check lists of gear, clothing, footwear, and raingear.
- Plan, plan, plan!

Focused instructions are available through specialty organizations such as the National Ski Patrol, Outward Bound, National Outdoor Leadership School, and Princeton University's Outdoor Activities Program, among others.

Box 89-8. Components of the Medical Examination

1. **Personal characteristics,** which provide a profile of the subject regarding age; gender; education; occupation; status as volunteer or recruit; history of a recent or remote similar venture; use of tobacco, alcohol, drugs, or steroids; history of psychological or interpersonal problems especially during wilderness ventures; and history of athletics.
2. **Historical features,** such as illness, with particular emphasis on cardiovascular, pulmonary, musculoskeletal, and neurologic problems; problems associated with a previous venture; intolerance to altitude, heat, or cold; psychological problems; accidents; and pertinent family history.
3. **Medical examination** to include examination of heart rate, blood pressure, precise cardiac examination (including auscultation), examination of peripheral pulses and carotid arteries, auscultation of the chest, abdominal palpation with rectal examination, musculoskeletal system with range of motion of joints and back, and height and weight.
4. **Physiologic examination,** which is only rarely required, depends largely on the nature of the venture and may range from simple simulation of the planned activity to functional aerobic testing for $\dot{V}O_2$max using a treadmill, cycle, or step ergometry. Functional testing with electrocardiographic monitoring is frequently used for diagnostic testing and for predicting cardiovascular response to exercise. Testing by running for speed or endurance and evaluation of dynamic strength and agility are rarely used but are interesting during skill assessment. Testing for hypoxic ventilatory response (HVR) may be primarily of research interest but nonetheless should be considered if precision is needed for elders going to high altitude.
5. **Psychological interviews** largely depend on the skill and technique of the examiner but should attempt to uncover a history of previous difficulties with group interaction and team activities, or fears related to physical and environmental stress.

MEDICAL EXAMINATION

By the time an individual qualifies for the designation "elder," that person has usually made some arrangements for medical care, examination, and advice. However, as an elder considers a venture into the wilderness, there is often confusion or insecurity about the prospect of developing some form of medical problem. A medical examination may be necessary.

Components of the Medical Examination

A survey of wilderness leaders served as the basis for developing specific components of a medical examination for prospective participants in wilderness activities.[10–12] The five categories of characteristics included in the health examination are shown in Box 89-8.

In the survey of wilderness leaders, the best predictors of a successful venture were found to be a history of similar successful venture and the psychological profile of the individual. The best single predictor of failure on a wilderness venture was difficulty with interpersonal relations. Consequently, the most important part of the medical examination before a wilderness venture was determined to be an in-depth interview by an examiner experienced in medical care *and* the nature of the venture. After the interview, appropriate medical, laboratory, radiographic, and physiologic studies may be performed.

The content of a medical examination, consisting of any or all of the 5 categories listed in Box 89-8, matches the characteristics of the individual with the characteristics of the venture and identifies the components of the medical examination that may be needed (Table 89-4).

COUNSELING AND TEACHING ELDERS BEFORE WILDERNESS VENTURES: THE GRAY EAGLES

The love of the wilderness fuels the spirit that hears the call of the wild. Aging seems only to sharpen this desire and to deepen its significance. One group of aging individuals who answered the song was the Gray Eagles (Fig. 89-4), the brainchild of Memphis pathologist Dr. John K. Duckworth and retired food executive Mr. John C. Johnson.[9] After years of recreational hikes, these seasoned outdoorsmen formalized their group in 1989. They invited individuals to participate in wilderness backpacking trips to such places as the Wind River Range in Wyoming, Banff National Park in Canada, the Beartooth Range in Montana, and the Olympic Range in Washington State. Their experiences have been invaluable in providing a unique opportunity to learn about the medical problems of groups in the wilderness.

With a record of over 3500 person-days in the wilderness, this group has sustained very few serious medical problems. Most problems consist of consequences of environmental exposure, trauma, infectious processes, and neuropsychiatric disorders (Box 89-9). Fatigue, minor musculoskeletal problems, and manageable trauma were encountered relatively often. One successful year of participation by an elderly person does not necessarily guarantee that the next year will be uneventful. With increasing age causing reduction of reserve, tolerance to the effort may change dramatically, and subtle symptoms may evolve into a full-blown manifest emergency. Problems confronted by the Gray Eagles with the most serious potential have been behavioral aberrations from organic brain syndrome not previously recognized but precipitated by the environmental and physical stress of the venture.

From these experiences, the need for a structured program of preparation prior to undertaking the trip became apparent. Analysis of the events of each trip led to a program that included:

1. Pre-selection based on experience, interviews, and medical history
2. A stringent 6-month conditioning program with good boots, well "broken in" with 100 miles of use
3. Walking with a backpack, unless contraindicated, weighing one-third of total body weight
4. Orientation meetings that include features of safety, review of risk, and recognition of clinical problems, such as the symptoms of hypothermia, hyperthermia, hypoxia, and fatigue

TABLE 89-4. Construction of the Medical Examination of Prospective Participants in Wilderness Ventures[10,11]

CLASSIFICATION OF CHARACTERISTICS OF PARTICIPANTS	CLASSIFICATION OF VENTURE			
	1. EXTREME PERFORMANCE	2. HIGH PERFORMANCE	3. RECREATIONAL	4. THERAPEUTIC
A. Demonstrated high performance	2, 3, **5**	2, 3, **5**	2	*
B. Healthy, vigorous	2, 3, 4, **5**	2, 3, **5**	**2**	**2**
C. Healthy, "deconditioned"	1, 2, 3, **4, 5**	2, 3, **4, 5**	2, **4**	2, **3, 4**
D. Risk factors	1, 2, **3, 4, 5**	1, 2, **3, 4, 5**	2, **3, 4,** 5	2, **3, 4, 5**
E. Illness	*	*	1, 2, **3, 4, 5**	1, 2, **3, 4,** 5

Category of Components (from Box 89-8):
1. Personal data
2. Historical data
3. Medical findings
4. Physiologic assessment
5. Psychological evaluation

*Individualized assessment indicated

A reason for classifying wilderness ventures and participants is to help design an examination that includes the features ideally examined depending on the physical status of the individual and the nature of the venture. All categories listed in Box 89-8 should be considered in the examination, but those shown here in bold numbers are of prime importance and should be emphasized.

Figure 89-4. The Gray Eagles. At top, with the flag, this group of senior outdoorsmen gathered 1 year after the 9/11 tragedy, memorializing those who suffered or died. Counterclockwise: John C. Johnson, one of the founders, rides in a wheelbarrow while Dr. Justin Towner pushes; left lower corner, founder Dr. John K. Duckworth; right lower corner, llamas help with the load. (Courtesy Dr. John K. Duckworth and Mr. John C. Johnson.)

Box 89-9. Medical Problems Encountered by the Gray Eagles during 3500 Person-days in the Wilderness

ENVIRONMENTAL PROBLEMS
- Acute mountain sickness (climbed as high as 13,500 ft [4100 m]) with nausea, vomiting, vertigo, and insomnia
- Hypothermia, mild
- Immersion foot (100% of one group of 8), numbness and tingling persisted for 6 months
- Wind burn, sunburn
- Numerous lightning storms

TRAUMA
- Lacerated knee with hemarthrosis (on warfarin)
- Splinters
- Ocular foreign bodies, conjunctivitis
- Many foot blisters, some serious
- Osteoarthritis of knees with exacerbation (subsequent arthroscopic meniscus surgery)
- Fractured thumb (climbing in rough terrain)
- Osgood-Schlatter disease of the knee with crippling pain, required walking with assistance
- Lymphedema of upper extremities secondary to pack straps (with paresthesias of hands)
- Fatigue from hiking on obliterated trail from storm and a "blow-down" in Bitteroot Mountains (required 9 hours over 2 days to go 3 miles)

INFECTIOUS DISEASES
- Giardiasis symptomatic after return
- Minor gastrointestinal upsets

NEUROPSYCHIATRIC PROBLEMS
- Dissension among the leaders of the group
- Blatant disregard for authority by one person who proceeded alone into remote dangerous area (set a small stove fire that endangered nearby hikers)
- Refusal to continue on hike (one person only)
- Emotional breakdown secondary to fatigue in middle-aged male
- Temporary dementia (later diagnosed as Alzheimer's disease)
- Fear of heights (acrophobia)
- Excessive use of alcohol

None of the problems was life-threatening, even though the group was in the vicinity of a fatal moose attack on one occasion. "I would do it all again," said Dr. John K. Duckworth.

(Courtesy of John K. Duckworth, MD.)

5. The "three season" principle for packing clothes because of the prospects of weather change
6. Orientation sessions with officials in the area to determine safety precautions unique to the region and to let the officials know of the presence of the Gray Eagles

With further aging, they have added llamas, mules, horses, and river trips. Their experiences provide a unique opportunity to examine the effects on elders of wilderness activities and the effects of exposure, lightning, and other environmental hazards. The remarkably safe record of the trips could be attributed to precise planning, education of participants regarding any eventuality, and the requirement that each member of the group have a health review prior to the venture. The security of physician oversight was reassuring. These activities displayed new horizons for participants and their doctors.

SUMMARY AND CONCLUSION

Seniors in our society are encouraged to remain mentally, physically, and socially active. They perceive this recommendation as a welcome invitation to live life to its fullest. In response, elders in increasing numbers have come to enjoy the fellowship, camaraderie, and adventure of wilderness activities. Unfortunately, health risk associated with high-performance activities increases with age because of altered physiologic function, unrecognized impairments, or effects of illnesses and their treatment.

To reduce risk, seniors should be advised to temper their enthusiasm with the caution derived from the wisdom of experience. John Glenn made his second space flight safely at age 77 because of thoughtful planning and training. A maxim from his aeronautical colleagues reminds us, "There are old pilots, and there are bold pilots, but there are no old, bold pilots." Nevertheless, a healthy respect for risk should not go to an extreme where it becomes a fear that precludes wilderness experience entirely. The golden years hold many opportunities for adventure. Remember that the optimist sees sunset as sunrise in reverse.

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The references for this chapter can be found on the accompanying DVD-ROM.

Persons with Special Needs and Disabilities

90

Sheryl K. Olson and Donna L. Nimec

The wilderness has been a place for rest, relaxation, work, exploration, life, and death since the beginning of human existence. People of all ages, races, health, and abilities participate in outdoor activities. Increasing populations have encroached on open spaces, threatening an enduring wilderness for present and future generations. To preserve and protect wildland areas, the U.S. Congress passed the Wilderness Act in 1964 and established the National Wilderness Preservation System (NWPS). The Wilderness Act prohibited roads and the use of motor vehicles, motorized equipment, or other forms of mechanical transport. Because of this, any person with a disability or functional limitation requiring assistive devices for mobility was prohibited from accessing the wilderness.

With increased awareness of the special needs and rights of people with disabilities, the unfair restrictions of the Wilderness Act needed to be addressed. In 1990, Congress passed the Americans with Disabilities Act (ADA). This Act supports persons with disabilities in using necessary assistive devices in the wilderness, thereby removing physical barriers of wilderness access. These devices include, but are not limited to, wheelchairs, respirators, and service dogs.

Focusing on functional issues, the ADA defines persons with disabilities as those with[8]

1. A physical or mental impairment that substantially limits one or more of the major life activities of such individual;
2. A record of such impairment; or
3. Being regarded as having such impairment.

For many individuals with chronic illnesses, the limitations are related more to intrinsic factors of the underlying disease process, not the environment. For example, if an individual has a seizure disorder, scuba diving may be contraindicated. For individuals with underlying pulmonary disease, high-altitude hiking activities may present increased risk for pulmonary complications. In this instance, it is not accessibility of the environment that is at issue. Individuals with chronic illnesses need to be willing to understand the physiologic limitations of disease processes and make appropriate decisions and preparations.

With regard to access to medical care, *wilderness* is defined as a remote geographic location more than 1 hour from definitive medical care.[78] People with disabilities and chronic illnesses are at risk for all the hazards associated with an activity plus the unique set of potential complications associated with the disability or the illness. To create a safe and positive wilderness experience for persons with disabilities and chronic illnesses, preparation for the adventure must include identification of potential medical complications, plans to prevent or minimize the risk of the complications, and a treatment protocol to follow if a complication arises.

The goal of this chapter is to help health care professionals become more aware of some of the adaptations that need to be considered when people with disabilities enter the wilderness environment. The authors also review some medical considerations that need to be made before an individual with a chronic illness participates in a wilderness activity.

PREVALENCE OF DISABILITY IN THE UNITED STATES

The U.S. Bureau of the Census provides data on disability using three primary sources: the decennial census of population, the Survey of Income and Program Participation (SIPP), and the Current Population Survey (CPS).[245] Inclusion of questions about disability status began with the 1970 decennial census. A comparable indicator for 1970, 1980, 1990, and 2000 could not be constructed because survey questions on disability became more descriptive of the physical capabilities of a person with a disability. There was also a variation in age of the population sampled. In 1990, the population 15 years of age and older were queried about disabilities; in 2000, the age category was 5 years and older. SIPP is a longitudinal national household survey that began in 1984. A panel of households enters the survey and is interviewed every 4 months for 3 years. The questions on disability are extensive and consistent.[244]

The third source for disability data is the CPS. This survey identifies persons who are out of the labor force because of a disability and identifies those who have a health problem that "prevents them from working or limits the kind or amount of work they can do."[245] This monthly survey has a primary focus on labor force data for the civilian noninstitutionalized population. Work disability is the only disability measured by the CPS.

The U.S. Census Disability Status: 2000 counted 49.7 million people with some type of long-lasting condition or disability.[245] This represented 19.3% of the 257.2 million people who were aged 5 years and older in the civilian noninstitutionalized population, as noted in Table 90-1.

A survey sample of one in six people was queried on disability issues using two multipart questions. The first question asked about any long-lasting condition such as blindness or deafness or a condition that substantially limited basic physical activities. The second question correlated a physical, mental, or emo-

tional condition lasting six months or more with difficulty in doing daily activities such as learning, dressing or bathing, going outside the home to shop or working at a job. Results of the disability questions from the 2000 census are shown in Table 90-2. Estimates may vary from actual values due to sampling variation and other sampling errors. The estimates were based on responses from a sample of the population. The responses were weighted to represent the total population and are significant at the 90% confidence level.[245]

The population was subdivided into three age categories identifying the type and frequency of disability in each group. The numbers and percentages of people with a sensory, physical, mental or self-care disability in each age category are identified in Table 90-3.

The SIPP has a specific set of definitions for disabilities and uses an extensive set of disability questions, making it the preferred source for examining most disability issues. All household members 15 years of age and older are interviewed by self-response, if possible.

The SIPP defines disability as relating to functional limitations for seeing, hearing, speaking, walking, and carrying or lifting weight. Box 90-1 provides a complete list of SIPP functional limitations.[136] Disability for a person age 15 years or older is defined as the need to use a wheelchair or other mobility aid, difficulty with a functional activity, difficulty with activities of daily living (ADLs) or instrumental activities of daily living (IADLs), or as having a developmental, emotional, or mental disability. Disability is further identified if a person age 16 years or older had a condition that made it difficult to do housework or limited the amount or kind of work he or she could do on a job. A person younger than 21 years of age was considered disabled if the parents reported receipt of developmental services for that child or limitation in usual activities, such as the ability to do regular schoolwork, walk, run, or use stairs. Last, one definition of total disability includes a person younger than age 65 years who is covered by Medicare and has received Social Security benefits. The defining characteristics of disability are listed in Box 90-2.[136]

Activities of daily living include getting into and out of bed or a chair, bathing, dressing, eating, toileting, and moving around the home, as listed in Box 90-3. IADLs, listed in Box 90-4, are slightly more advanced and require a higher level of functioning, such as is required for meal preparation, simple housework, money management, and going outside the home for shopping. Severity is measured by the level of inability to perform physical functions.

The National Center for Health Statistics (NCHS) also queries a sample of the civilian, noninstitutionalized households in the United States. For disability purposes, the National Health Interview Survey on Disability (NHIS-D) focuses on two specific components: limitation of activity and chronic conditions.[210] NHIS identifies major activities associated with a particular age group according to the predominant activity, as noted in Table 90-4.[183]

A long-term reduction in a person's ability to perform a normal amount of activity associated with his or her particular age group is deemed "limitation of activity." A chronic condition is defined as one that persists for 3 months or more. Some conditions, such as arthritis, cancer, diabetes, and heart conditions, are considered chronic regardless of duration. Persons must have one or more chronic health conditions reported as the cause of the activity limitation in order to meet the "limitation of activity" classification. Disability is thus defined by the NHIS as any long- or short-term reduction or limitation of a person's activity as a result of an acute or chronic condition.

Chronic health conditions are the most common cause of disability. Conditions that cause activity limitations occur an estimated 61,047,000 times in the U.S. population. The three conditions causing the most limitations are heart disease (7,932,000), back problems (7,672,000), and arthritis

TABLE 90-1. Civilian Noninstitutionalized Persons Aged 5 Years and Older: Disability

	TOTAL NUMBER	PERCENTAGE
Population ≥5 yr	257,167,527	100.0
With any disability	49,746,248	19.3

From U.S. Census Bureau, Census 2000, Summary File 3, Table 1.

TABLE 90-2. Questions on Disability from Census 2000

QUESTION ON CENSUS	NUMBER IN MILLIONS	PERCENTAGE OF POPULATION
1. Does this person have any of the following long-lasting conditions:		
a. Blindness, deafness, or a severe vision or hearing impairment?	9.3	3.6
b. A condition that substantially limits one or more basic physical activities such as walking, climbing stairs, reaching, lifting, or carrying?	21.1	8.2
2. Because of a physical, mental, or emotional condition lasting 6 months or more, does this person have any difficulty in doing any of the following activities		
a. Learning, remembering, or concentrating?	12.4	4.8
b. Dressing, bathing, or getting around inside the home?	6.8	2.6
c. (Answer if this person is 16 YEARS OLD OR OVER.) Going outside the home alone to shop or visit a doctor's office?	18.2	8.6 (Of the 212.0 million people this age.)
d. (Answer if this person is 16 YEARS OLD OR OVER.) Working at a job or business?	21.3	11.9 (Of the 178.7 million people this age.)

From U.S. Census Bureau, Census 2000 questionnaire.

TABLE 90-3. Characteristics of the Civilian Noninstitutionalized Population by Age, Disability Status, and Type of Disability

	TOTAL NUMBER	PERCENTAGE
Population 5–15 yr	45,133,667	100.0
With any disability	2,614,919	5.8
Sensory	442,894	1.0
Physical	455,461	1.0
Mental	2,078,502	4.6
Self-care	419,018	0.9
Population 16–64 yr	178,687,234	100.0
With any disability	33,153,211	18.6
Sensory	4,123,902	2.3
Physical	11,150,365	6.2
Mental	6,764,439	3.8
Self-care	3,149,875	1.8
Going outside the home	11,414,508	64
Employment disability	21,287,570	11.9
Population ≥65 yr	33,346,626	100.0
With any disability	13,978,118	41.9
Sensory	4,738,479	14.2
Physical	9,545,680	28.6
Mental	3,592,912	10.8
Self-care	3,183,840	9.5
Going outside the home	6,795,517	20.4

From U.S. Census Bureau, Census 2000, Summary File 3, Table 1.

Box 90-1. SIPP Limitations in Functional Activities

- Seeing ordinary newspaper print (with glasses or contacts if normally used)
- Hearing normal conversation (using aid if normally used)
- Having speech understood
- Lifting or carrying 10 lb
- Walking a quarter of a mile without resting
- Climbing a flight of stairs without resting
- Getting around outside
- Getting around inside
- Getting into and out of bed

From The Survey of Income and Program Participation, U.S. Census Bureau.

(5,721,000). The top 10 chronic conditions causing activity limitations are listed in Table 90-5.[183]

The three conditions causing the highest percentage of limitations in major activity are mental retardation, multiple sclerosis, and malignant neoplasm of the stomach, intestine, colon, and rectum. The top 10 most frequently limiting chronic conditions are listed in Table 90-6. Of all people with mental retardation, 87.5% have such a limitation. For those with multiple sclerosis, 69.4% have a limitation; 62.1% of persons with the aforementioned types of malignant neoplasm have a limitation in major activity.[183]

As noted in Table 90-7, the NHIS classifies each person into one of four degrees of disability: unable to perform the major

Box 90-2. SIPP Definition of Disability

1. Use of a wheelchair
2. Use of a cane or similar aid 6 months or longer
3. Has difficulty with a functional activity
4. Has difficulty with an ADL
5. Has difficulty with an IADL
6. Is identified as having a developmental disability or a mental or emotional disability
7. Is 16 years or older and has a condition that makes it difficult to do housework
8. Is between 16 and 67 years of age and has a condition that limits the amount or kind of work the person could do at a job
9. Is younger than 21 years of age and parents responded on the survey about receipt of developmental services, and limitations in usual activities such as the ability to do regular schoolwork, or the ability to walk, run, or use stairs
10. Is younger than age 65 years and covered by Medicare or received SSI

ADL, activity of daily living: IADL, instumental activity of daily living; SSI, supplemental security income.
From The Survey of Income and Program Participation, U.S. Census Bureau.

Box 90-3. Activities of Daily Living

- Getting around inside the home
- Getting into and out of bed or a chair
- Bathing
- Dressing
- Eating
- Toileting

From The Survey of Income and Program Participation, U.S. Census Bureau.

Box 90-4. Instrumental Activities of Daily Living

- Going outside the home
- Keeping track of money or bills
- Preparing meals
- Doing light housework
- Using the telephone

From The Survey of Income and Program Participation, U.S. Census Bureau.

TABLE 90-4. Major Activities for Each Age Group

AGE GROUP	MAJOR ACTIVITIES
<5 yr	Ordinary play
5–17 yr	Attending school
18–69 yr	Keeping house, working, or ability to work at a job or business
≥70 yr	Capacity for independent living

From Chartbook on Disability in the U.S. Appendices: Surveys.

TABLE 90-5. Top 10 Chronic Conditions Causing Activity Limitations, NHIS 1990–1992 Survey

CHRONIC CONDITION	NO. OF CONDITIONS CAUSING LIMITATIONS
Heart disease	7,672,000
Back problems	5,721,000
Arthritis	2,592,000
Asthma	2,569,000
Diabetes	2,035,000
Mental disorders	1,577,000
Disorders of the eye	1,575,000
Learning disabilities and mental retardation	1,342,000
Cancer	1,294,000

From National Center for Health Statistics (NCHS) National Health Interview Survey (NHIS), 1996.

TABLE 90-6. Top 10 Most Frequently Limiting Conditions

CHRONIC CONDITION	PERCENTAGE OF CONDITIONS CAUSING MAJOR LIMITATIONS
Mental retardation	87.5%
Multiple sclerosis	69.4%
Malignant neoplasm of stomach, intestine, colon, and rectum	62.1%
Paralysis of extremities, complete or partial	60.7%
Malignant neoplasm of lung, bronchus, and other respiratory sites	60.6%
Blindness, both eyes	60.3%
Other deformity or orthopedic impairment	54.4%
Paralysis of other sites, complete or partial	48.0%
Other diseases of the heart, excluding hypertension	47.8%
Epilepsy	44.4%

From National Center for Health Statistics (NCHS) National Health Interview Survey (NHIS), 1996.

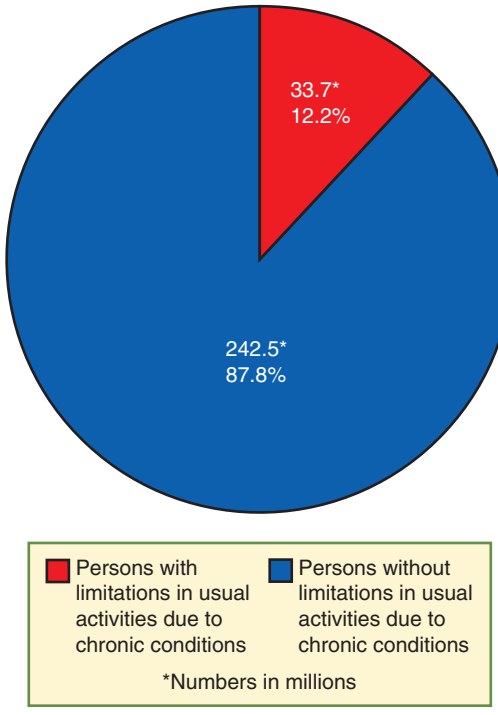

Figure 90-1. Limitations due to chronic conditions: civilian noninstitutionalized persons. (From Schiller JS, Bernadel L: Vital Health Stat 10[220], 2004.)

TABLE 90-7. NHIS Classification of Disability

CLASS	DESCRIPTION
1	Unable to perform the major activity.
2	Able to perform the major activity but limited in the kind or amount of this activity.
3	Not limited in the major activity but limited in the kind or amount of other activities.
4	Not limited in any way.

From 2002 National Center for Health Statistics (NCHS) National Health Interview Survey (NHIS) Summary of Health Statistics.

activity, able to perform the major activity but limited in the kind or amount of this activity, not limited in the major activity but limited in the kind or amount of other activities, or not limited in any way.

According to the 2002 NHIS Summary of Health Statistics for the U.S. Population survey, approximately 33.7 million persons were limited in their usual activities because of one or more chronic health conditions (Fig. 90-1). The Summary of Health Statistics for the U.S. Adult Population categorizes limitations into four categories: hearing trouble, vision trouble, unable or very difficult to walk one-quarter mile, and any physical functioning difficulty (Fig. 90-2).

The NCHS examines the use of assistive technology devices for vision, hearing, mobility, and orthopedic impairments. This is the first analysis of this kind from the NHIS's Disability Supplement. Highlights from the report, titled *Trends and Differential Use of Assistive Technology Devices: United States, 1994*, include[181]:

- 7.4 million persons in the U.S. household population use assistive technology devices for mobility impairments, the most frequent reason for using an assistive device. Almost 5 million people use canes, the single most utilized assistive device.
- 4.6 million persons use assistive devices such as back braces and artificial limbs to compensate for orthopedic impairments.
- 4.5 million persons use hearing aids, amplified telephones, closed-caption television, and other assistive devices for hearing impairments.
- 500,000 persons use assistive technology devices for vision impairments.
- The majority of persons using these devices are older than 65 years of age. Sixty-two percent of persons using mobility

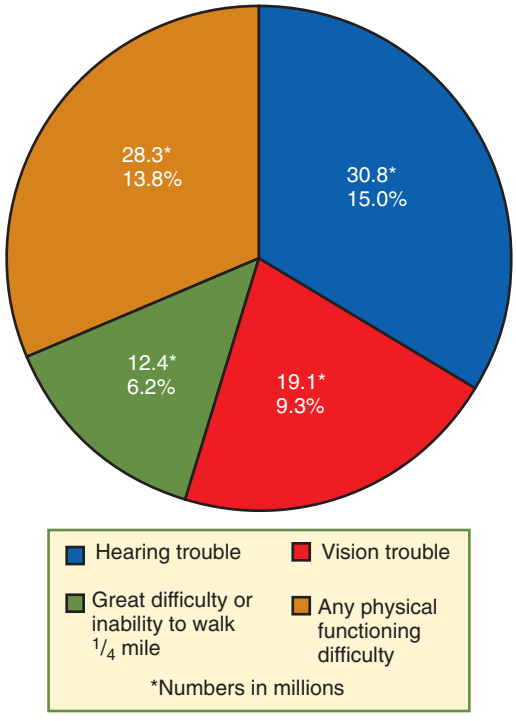

Figure 90-2. Types of limitations: civilian noninstitutionalized adults. (From Schiller JS, Bernadel L: Vital Health Stat 10[220], 2004.)

devices, 69% of persons using hearing devices, and 51% of persons using vision devices are older than 65 years of age.

- Use of assistive devices has increased dramatically over the past decade, in part because of the aging of the population, but also because of technologic advances, public policy initiatives, and changes in the delivery and financing of health care.

With the passage of recent legislation, the wilderness is more accessible now than in past years. As a result, more people are willing and able to take advantage of its beauty and opportunities. People with chronic illnesses who may or may not have had interests in wilderness activities in the past may want to commence participation in these activities. They must have a thorough understanding of the physical limitations that require adaptations and perhaps different types of preparation.

THE NECESSITY OF WILDERNESS

The beauty of the trees,
the softness of the air,
the fragrance of the grass
speaks to me.
The summit of the mountain,
the thunder of the sky,
the rhythm of the sea,
speaks to me.
And my heart soars.

—Chief Dan George

The search for self-fulfillment and actualizing one's potential has been suggested as one of the highest human motives.[159] Outdoor activities and wilderness journeys can have a powerful effect on the lives of individuals. They create opportunities for experiential learning, adventure, and self-fulfillment, and they can promote self-esteem and build confidence. A 1990 study by McCleary and Chesteen in which people with disabilities spent 2 days in the wilderness on a river trip showed an improved self-image of the participants. Forty-seven percent of these people believed that they could do more things than they had previously thought possible after participating in the trip. Being able to do things better after the trip as compared to before the trip was reported by 41%. Over 90% reported a positive experience of making new friends and experiencing adventure. Improvement in areas of problem solving and decision making (41%), human relations (76%), and self-assurance (65%) were also reported. Over a third of the participants claimed increased effectiveness in their work environment after the wilderness experience.[163] The wilderness classroom offers its own unique pathway for personal growth.

Opportunities to access the wilderness are openly available to both the able and the disabled. Education and adventure groups have expanded their programs worldwide to be inclusive of people with functional limitations. Programs for people with disabilities incorporate outdoor activities such as mountaineering, canoeing, caving, scuba diving, hunting, fishing, skiing, bicycle journeys, and many other sport activities. Although no data exist for the number of disabled persons participating in wilderness activities, the number of organizations advertising to and including people with disabilities is large.

The national organization for a particular disability can be used as a reference and launch point leading to the discovery of adventure opportunities for people with a particular type of disability. Many adventure groups offer outdoor activities and wilderness experiences to people with a broad scope of disabilities and welcome all people regardless of ability or level of dependency.

Canada began a campaign in the 1980s with the goal of promoting public awareness that disabled people can function independently within society.[74] Great Britain has an organization, the British Mountaineering Council, that lists all the adventure clubs for climbers, hill walkers, and mountaineers. It specifically notes which have accessibility for the disabled.

Ski for Light, Inc., one of the first organizations for people with disabilities, was founded in 1975 to promote the physical fitness of vision- and mobility-impaired adults. The philosophy is to replace "we can't" with "we can." Disabled and able-bodied people join together in a one-on-one partnership to learn from each other through physically demanding activities (Fig. 90-3). Annual events include regional and international programs that teach cross-country skiing to participants with these disabilities.

The presence of physical disability does not imply or indicate lack of physical ability. There are many notable wilderness adventure success stories of people with a variety of disabilities. John Hawkridge, a climber with cerebral palsy (CP), tackled part of Mt. Everest in 1988. Jeff Pagels, a paraplegic, climbs mountains using adaptive equipment, including a hand-powered mountain bike and a snow sit-ski that is arm-powered with fixed ropes and ascenders. He has climbed Mt. Whitney in California, Mt. Bross in Colorado, and Mt. Kilimanjaro in East Africa (Fig. 90-4). Dean Juntunen, paralyzed below his diaphragm, accesses the wilderness on his kayak. Spring and fall, he can be found running his favorite rivers (Fig. 90-5).

Figure 90-3. Participants of Ski for Light, 2005. (Photograph courtesy of Craig Gray.)

Figure 90-5. Dean Juntunen at a rugged kayak launch. (Photograph courtesy of Sheryl Olson.)

Figure 90-4. Jeff Pagels and friends at 3050 m, Mt. Bross, Colorado. (Photograph courtesy of Sheryl Olson.)

Mike Schmitz has a C6-7 incomplete spinal cord injury with limited strength in his hands and no trunk balance. He has found ways to adapt equipment and technique to be able to hunt. Erik Weihenmayer, a totally blind mountaineer, summited Mt. Everest in 2001. He stated in his book, *Farther Than the Eye Can See,* "The dark is a great equalizer."

PREPARTICIPATION EVALUATION FOR THE INDIVIDUAL WITH A DISABILITY

Over the last several decades, the attitudes of health care providers have changed to encourage more activity for individuals with disabilities and chronic illnesses. The focus has turned toward helping individuals achieve their goals despite their disabilities. For example, exercise has been promoted to keep individuals with chronic diseases and disabilities healthier. Maximizing functional independence and minimizing health risks are keys to safe adventures. Physical activity programs have evolved to help develop and maintain cardiovascular endurance, flexibility, and muscle strength.

Fitness programs for individuals with disabilities generally address cardiovascular endurance, muscle strength, and range of motion. Each individual's limitations act as a road map for planning an effective exercise program. For example, exercise management for the child with CP would include a combination of interventions that are designed to improve efficient patterns of movement. Exercise planning for individuals with pulmonary disease should focus on interventions to reduce symptoms of dyspnea and maximize endurance.

The American College of Sports Medicine has put forth a proposal that problem-oriented exercise management should be used to develop exercise programs for people with a variety of chronic illnesses and disabilities.[174] Problem-oriented exercise management uses exercise testing to reveal physiologic dysfunction. It also directs an exercise therapy program toward problems that might be improved by training. In addition, it integrates exercise into medical management problems. It takes components of an individual's movement or medical difficulties and puts a program together with manageable goals.

Before participating in a wilderness experience, a complete functional assessment should be completed to help a person with a chronic illness or disability to understand potential limitations. The level of independence for transfers, ambulation, or mobility using adaptive equipment, and the proficiency demonstrated by an individual in using the equipment should be noted.

A general cognitive assessment may also be helpful in some cases. This is important for skill acquisition and safety awareness. An individual with a disability should be educated in all areas of potential risks for given activity.

The goal of the preparticipation physical examination is to assess, educate, and prevent. It is important to assess the physical capability to understand the individual's physical requirements for a given activity. It is also important to educate the individual about the potential medical risks of the selected activ-

ity. Finally, providing information to the individual and team members about potential medical complications is a way to prevent a catastrophic event. This can be done by using both the screening assessment and a complete physical examination.

Different aspects of an individual's medical treatment may influence how well a person performs a specific activity. For example, there are a variety of drugs that interfere with the body's response to stress. In addition, risks, costs, and benefits of an activity or exercise program need to be addressed. Risks are disease dependent. For instance, if an individual has arthritic joints, increased activity such as a wilderness hiking experience may increase the pain and discomfort. There are some components of activity that depend on the deficits associated with an underlying condition, such as weakness or muscle imbalance. The risks associated with these activities often include musculoskeletal injury or exhaustion.

Costs involved in the training required to maximize participation in a wilderness experience need to be determined. This may include an exercise training program or evaluation of medications. Benefits include all activities and experiences gained from being in the wilderness with other individuals.

Appropriate physical screening should also be performed to rule out contraindications and or develop a list of precautions. The skin should be evaluated for insensate areas and checked for vasomotor changes in extremities that are weak or painful. Vital capacity should be measured, if indicated, in individuals with muscular or neurologic impairment that could affect respiration, especially if they have a history of respiratory illnesses such as asthma. This may involve formal pulmonary function tests or submaximal cardiopulmonary testing.

Pulmonary function tests in neuromuscular conditions typically reveal a decrease in vital capacity. This is initially due to the large residual volume and later due to low total lung capacity measurements. Vital capacity, forced expiratory volume in the first second (FEV_1), forced vital capacity (FVC), and total lung capacity are generally considered normal in the able-bodied population, if they are greater than 80% of the predicted normal value pertaining to the appropriate table. If the FEV_1 is less than 30% of the predicted normal, cardiopulmonary exercise testing is contraindicated.

The presence of increased motor tone and how it affects function and joint alignment should be described. The neurologic evaluation might also include manual muscle testing. This provides information to the individual about which muscle groups may be weak. Certain compensations may be needed if an activity requires a degree of strength not available to the individual. Injuries sustained in recreational activities are often soft tissue injuries related to the weakness or muscle imbalance. The evaluation should involve inspection, palpation, and assessment of passive and active ranges of motion of major joints.

Structural deformities should be noted as well. Conditions such as muscle contractures and associated joint deformities are frequently seen in ambulatory individuals with CP and should be noted because these may contribute to biomechanical stresses during rigorous activity.

GENERAL PREPARATION GUIDELINES

A team of people may be required to support a person with a disability during a wilderness adventure. There are no standards or specific guidelines in the literature indicating how many people are needed safely to support a disabled person participating in an outdoor sport or wilderness adventure.

Trip preparation for an individual with a chronic illness or disability is the same as for anyone desiring a wilderness experience. The first steps are to decide what the activity is, where it will occur, and how long it will last. Time of year and extremes of weather conditions should be considered. Information must be obtained about accessibility and any special equipment necessary to facilitate access to the area identified. Inquiry about the condition of pathways and trails for mobility assist devices should be obtained before the trip.

The degree of assistance needed is unique for every person and every situation, but could range from one to five or more people. The functional ability and support needs of each team member should be evaluated in preparation for a wilderness adventure. There needs to be enough strength within the entire group to balance a limitation of a single member. The most limited member may not be the person with the disability. The first time a summit of Mt. Whitney was attempted by a paraplegic, the photographer documenting the trip was more limited by lack of outdoor skills than the paraplegic was limited by lack of functional legs.

Adventure in the wilderness environment creates a change from the normal ADLs. People with disabilities rely on ADL routines for maintenance of daily habits. Disruption of the comfortable and familiar can sometimes result in frustration and unpleasant consequences. For example, a change in diet can cause a change in bowel function. For a paraplegic, this is undesirable and potentially stressful. Paraplegics rely on regular bowel routines in hopes of preventing a surprise public stooling. Trip planning should include appropriate plans for diet and maintenance of the daily bowel routine.

The level of understanding the individual has about his or her injury or illness is important information because not everyone has the same foundation of knowledge. It is possible that the wilderness participant is newly injured or was injured in a country where education was not part of the rehabilitation process, as noted in the following report:

> *A young man with a high spinal cord injury was going on a day hike. He was from South America and had been living in the United States for 10 years. Before the hike, he was asked if he had trouble with heat and he assured the guide he did not. He did develop "major problems," and the only way the guides got him through the day was "by keeping his clothing and hair saturated with cold water." This mobility-impaired person became disabled in South America. He had never received any education or training about his injury and, having never before ventured into the wilderness, was not aware he was susceptible to the effects of heat.*

It is helpful if the individual with the disability is able to communicate all pertinent medical information to the team leader, team medical person, or a designated team member before the trip. The designated person(s) should be aware of and understand the potential complications of the illness or injury, even those not previously expressed or experienced by the individual. They should have knowledge of the signs and symptoms and be prepared to make a decision and implement a care plan if indicated. This person must also be able to differentiate between a life-threatening medical emergency and a situation that can be managed in the field. Wilderness trip plans should include knowledge of rescue and evacuation options. If desired,

communication devices such as satellite phones or emergency personal radio beacons (EPRBs) can be carried.

Chronically ill or disabled people may have an enhanced predisposition to complications when pursuing wilderness activities. Several questions should be considered during preparation for the trip:

1. What are the potential complications of this condition?
2. What complications most commonly affect this person?
3. How is the complication managed in the home environment?
4. What complication(s) will the wilderness environment and the chosen activity have on a person with this condition?
5. How can the complication(s) be prevented?
6. What specialty equipment is needed to maximize safety?

Knowledge of the individual's normal routines will aid in preventive measures, thus promoting safety and minimizing risk during the wilderness adventure. Answers to these questions will also provide insight into equipment that might need to be purchased or special adjustments that need to be made on assistive devices before the trip. Adapting to new equipment should be accomplished before commencing the trip. It is important to allow time for the individual to try the equipment for ease of use, comfort, and fit and to identify and resolve potential equipment problems before they occur in the wilderness.

The National Center on Physical Activity and Disability (NCPAD) recommends the following general guidelines be followed during an outdoor experience[182]:

1. Frequently check for hypothermia.
2. Maintain adequate hydration by drinking plenty of water.
3. Maintain the same eating schedule and the same bowel and bladder routine.
4. Bring enough medication to last at least 3 days beyond the length of the planned trip.
5. Bring an extended family member or personal care attendant (PCA) if needed.
6. Let other people know the planned itinerary and overnight locations.

With adequate planning, team support, and proper equipment, the wilderness experience can be as safe for the disabled person as for the nondisabled.

MOBILITY IMPAIRMENTS

Spinal Cord Injury

The current estimate of the total number of individuals in the United States living with spinal cord injury (SCI) or spinal dysfunction is 250,000 to 400,000.[186] The National Spinal Cord Injury Association Resource Center (NSCIARC) also reports an estimated 32 injuries per 1 million population, or 7800 new injuries, in the United States each year using data from regional SCI Centers and compiled by researchers at the University of Alabama at Birmingham. Eighty-five percent of SCI trauma patients who survive the first 24 hours are still alive 10 years later, compared with 98% of the non-SCI population, given similar age and sex.[187]

Complications

Long-term secondary medical complications in individuals with traumatic SCI were analyzed for incidence, risk factors, and trends in a 1999 study.[165] The three most common long-term medical complications were autonomic dysreflexia, pressure ulcers, and pneumonia/atelectasis.

Autonomic Dysreflexia

Autonomic dysreflexia (AD), or hyperreflexia, is a potentially life-threatening complication for a person with a high-level SCI and requires immediate treatment. It most commonly occurs when the SCI is at or above the major sympathetic outflow at T6.[66] It rarely develops in persons with a level of injury below T6, but has been reported by persons with a lesion as low as T8.[172] Although most common and unpredictable during the first year after SCI, episodes of AD can occur at any time throughout the individual's life.[7]

The acute clinical syndrome manifests as paroxysmal hypertension affecting both systolic and diastolic blood pressure.[66] The syndrome is caused by noxious stimuli below the level of injury that produce an exaggerated and unopposed sympathetic response.[145] The reported incidence of AD is variable throughout the literature. Stover and colleagues reported the incidence as 15.4% in cervical injuries and 2.6% in high thoracic injuries.[230] Colachis reported that AD occurs in up to 85% of individuals with SCI above the major splanchnic sympathetic outflow.[45] Other investigators state that AD can occur in both incomplete and complete SCI and has an incidence of 19% to 70%.[33,224]

Autonomic dysreflexia can readily be recognized and treated in the wilderness environment. The severity of AD can be decreased with prompt and early intervention. It can nearly always be managed successfully but can cause severe complications, such as cerebral or subarachnoid hemorrhage and death, if left untreated. Early recognition and rapid intervention decrease the incidence of complications.

Cause. Bladder or bowel distention accounts for most episodes of AD, although a variety of stimuli can provoke an autonomic response.[45] Urologic stimuli, such as bladder distention and spasm, account for 90% of all episodes of AD.[60] Bladder distention, the most common precipitant, is present in 75% to 85% of cases.[148] The other most common causes include bowel impaction or constipation and pressure ulcers.[60] Using many literature resources, Eltorai compiled an excellent comprehensive review classifying the triggers of AD by a review of systems.[66] Table 90-8 identifies the mechanisms that could present as noxious stimuli potentially predisposing to AD during a wilderness adventure.

The list of stimuli presented by Eltorai[66] includes a few specific environmental factors that could stimulate the onset AD for the individual participating in wilderness sports and activities. These include exposure to extremes of temperature, wounds, sunburns, and insect bites. Although not reported in the literature, other environmental hazards (e.g., snakebites or contact dermatitis from poison ivy, oak, or sumac) could precipitate AD.

The risk of trauma to the skin or musculoskeletal system increases with outdoor activity. Blisters and abrasions are not uncommon in the wilderness. Even though statistics indicate bladder distention as the most common cause of AD, bowel impaction and pressure sores may be more common. If they are found not to be the cause and AD remains unresolved, factors common to the wilderness environment should be included in the differential of identifying causes.

Signs and Symptoms. Early subjective symptoms experienced by the patient can forewarn of an impending crisis. The cluster of findings may include anxiety, apprehension, restlessness,

TABLE 90-8. Stimuli Leading to Autonomic Dysreflexia

SYSTEMS	CAUSATIVE FACTORS
Genitourinary	**Both sexes:** bladder distention; plugged catheter; prolonged intervals between intermittent catheterizations; hypertonic bladder spasm; urinary tract infections; stones in the bladder, urethra, or ureters **Male:** epididymitis; torsion of the testicles; compression of the testicles; sexual intercourse; or electroejaculation **Female:** menstruation, menstrual cramps, pregnancy, labor, vaginal inflammation, sexual intercourse, ectopic pregnancy
Gastrointestinal	Constipation, fecal impaction; distention during digital stimulation; enemas; multiple suppositories; sphincteric dilatation; prolapsed, thrombosed, or inflamed hemorrhoids; anal infections; peptic ulcers; acute gastritis; acute gastric dilatation; gastrectasis; severe flatulence; acute abdomen (e.g., bowel obstruction, appendicitis, pancreatitis, acute cholecystitis)
Skin stimuli	Any direct irritant below the level of injury: pressure ulcers, abscesses, ingrown toenails, paronychia, tight clothing/shoes, applied splints, cast, exposure to extremes of temperature, wounds, applied tourniquets, bullets or shrapnel, burns, insect bites, pressure of hard or sharp object in bed or in wheelchair

Data from references 33, 48, 57, 66, 152, 158, 209, 259.

nasal congestion, mild shortness of breath, beads of perspiration, and occasionally penile erection.[66] At this point, the crisis can be prevented.

As the episode progresses, the individual might complain of a severe, pounding headache, nasal congestion, and a flushed face.[24] The headache can be frontal, bitemporal, or occipital.[66] The individual may experience visual changes and be diaphoretic and flushed above the level of the SCI with pallor and piloerection below the level of the SCI.[3] Other reported signs and symptoms include malaise, nausea, bronchospasm or respiratory distress, mydriasis, cardiac irregularities, patchy erythema above the level of the SCI, and a metallic taste in the mouth.[145] Early symptoms increase in intensity until the cause is eliminated. Delayed treatment can result in complications of a hypertensive crisis such as seizures, stroke, retinal hemorrhage, subarachnoid hemorrhage, cardiac dysrhythmia, cardiac arrest, or death.[3]

The hallmark sign of AD is severe hypertension with systolic blood pressure as high as 250 to 300 mm Hg and diastolic as high as 200 mm Hg.[128] Resting blood pressure declines after SCI, with normal often in the range of 90/60 mm Hg.[160] A significant rise in blood pressure is an increase greater than 40 mm Hg.[145] Thus, a resting blood pressure of 130/90 might be considered elevated. Reflex bradycardia (<60 beats per minute) frequently accompanies the hypertension.

When participating in a wilderness adventure with a paraplegic or tetraplegic, team members must be aware of the level of SCI for that person. If the injury is at T6 or above, they should be aware of the possible causes of AD and the presenting signs and symptoms. They must be trained to recognize the signs of AD and intervene appropriately. The normal resting blood pressure of the person with the SCI should be known and documented before the trip.

Differential diagnosis for AD includes essential hypertension, epilepsy, cerebral hemorrhage, migraine and cluster headaches, posterior fossa tumors, pheochromocytoma, and eclampsia.[66]

Treatment. Systolic blood pressure of 150 mm Hg is the accepted defining parameter of hypertension caused by AD. The treatment goals for this syndrome are first to lower the blood pressure and second to identify and eliminate the noxious stimuli. The first step is to sit the person upright if he or she is in a recumbent position. This position optimizes pooling of blood in the lower extremities, decreasing cardiac preload. Any tight clothing or restrictive devices should be loosened. Any straps securing legs to a mobility assist device should be removed. A personal flotation device (PFD), climbing harness, ski boots, or other equipment, if in use, should be loosened or removed.

The second step is to identify and remove the precipitant, beginning with assessment of the bladder and urinary drainage system. When intermittent self-catheterization is the chosen bladder management program, the bladder should be catheterized and emptied of urine. If an indwelling catheter is used, patency should be checked by observing for urine flowing through the tube and into the collection device. The drainage tube should be checked for kinks or other obstructions impeding free outflow of urine from the bladder. If blockage is suspected, the catheter can be gently irrigated. The goal is to prevent further bladder distention or irritation while flushing and reestablishing catheter patency. Bladder irrigation with 60 mL of a 2% lidocaine solution can reduce bladder irritation from the flush.[242] Temperature of the irrigant should be close to body temperature to avoid additional stimulation from instillation of hot or cold solution.

If the catheter is blocked and cannot be successfully irrigated, it should be promptly removed. After removal, 10 mL of 2% lidocaine jelly inserted into the urethra may minimize additional irritation. Prefilled syringes of lidocaine jelly are available from several medical companies. International Medical Systems produces a two-part sterile syringe filled with 10 mL (200 mg) of 2% lidocaine HCl jelly 20 mg/mL (Fig. 90-6). The tip of the syringe is smooth and rounded and is inserted directly into the urethra (Fig. 90-7).

After inserting lidocaine jelly and waiting 10 to 15 minutes, an attempt can be made at gently inserting another catheter. If a regular catheter cannot be passed, an attempt can be made using a coudé catheter. If the urethra cannot be catheterized and urine cannot be drained from the bladder, a medical emergency exists and the individual needs to be evacuated from the wilderness as quickly as possible.

After the bladder is emptied and when the systolic blood pressure remains 150 mm Hg or greater, pharmacologic therapy should be initiated before further manipulation of the patient.[162] Preferred medications for initial management are fast-acting agents with a short half-life, such as nitroglycerin. Nitroglycerin ointment or paste in a 2.5-cm strip should be applied to the chest wall above the level of the injury.[66] A second application can be applied as needed. The individual's blood pressure should be monitored every few minutes throughout the treat-

Figure 90-6. Syringe prefilled with 2% lidocaine HCl jelly. (Photograph courtesy of Sheryl Olson.)

Figure 90-7. Catheter tip syringe prefilled with 2% lidocaine HCl jelly. (Photograph courtesy of Sheryl Olson.)

ment process until the crisis has resolved. When taking frequent blood pressures in a cold environment, it is important to protect the person in an AD crisis from unnecessary exposure to the elements because this could further aggravate the symptoms.

Rapid onset of action, short half-life, and ease of application make nitroglycerin paste or ointment an excellent choice for acute management of an AD-triggered hypertensive crisis in the wilderness. Unlike oral, parenteral, or longer-acting antihypertensive agents, a significant advantage of this medication is that it can be readily removed when the AD trigger has been eliminated and the hypertension resolves.

Nitroglycerin paste is easy to transport in medical kits, easy to apply, and requires no extra supplies for administration. The manufacturers recommend storing the medication at controlled room temperature, between 15°C and 30°C (59°F to 86°F). Because the effects of temperatures outside this range are unknown, the medication should be properly stored during a wilderness expedition to ensure product stability.

Once the systolic blood pressure is less than 150 mm Hg, further assessment of the patient can be accomplished. Digital rectal examination should be done to check for fecal impaction. If impaction is present, and if the systolic pressure remains less than 150 mm Hg, digital disimpaction can be done. With a systolic pressure greater than 150 mm Hg, medical management for hypertension should be continued and digital evacuation of the rectum delayed until the blood pressure is controlled.[67] Movement and manipulations of the person with paraplegia or tetraplegia in autonomic dysreflexia should be minimized until the blood pressure is less than 150 mm Hg.[162]

Digital stimulation requires use of a generous amount of 2% lidocaine gel, which is introduced into the rectum to minimize further noxious stimuli.[162] The rectum can then be gently assessed for impacted fecal matter, with subsequent removal if present. Frequent monitoring of blood pressure should continue during all maneuvers. If it begins to increase, the procedure should be stopped, an antihypertensive agent administered, and more lidocaine gel inserted into the rectum; when the systolic pressure is less than 150 mm Hg, the procedure can then be continued.

If no impaction is present, a physical examination should be done to assess for tissue pressure points and necessary adjustments made. If bladder distention, rectal impaction, and pressure point irritation are not the cause of the event, assessment for other potential noxious stimuli must ensue. Once pharmacologic management of hypertension has been initiated, it should be continued until the cause is identified and eliminated and the blood pressure has returned to normal limits.

The possibility always exists that injury or illness may occur in the wilderness that cannot be promptly remedied. Examples of such problems include bladder infection, severe blister, fracture, pressure sore, sunburn, contact dermatitis, or laceration. If the trigger of AD has been identified and will take time to resolve, or if the trigger cannot be resolved in the wilderness, the person should be placed on a maintenance dose of antihypertensive medication until the negative stimulus of the condition is remedied.[242]

Hypertension can be managed in the wilderness with a variety of medications. Nifedipine was the most commonly used medication for over a decade.[33,47,61,92,128,145,238] Grossman and colleagues conducted a study on the use of nifedipine and concluded that absorption of nifedipine from buccal mucosa showed inconsistent delivery and recommended abandoning its use.[92] Hydralazine (Apresoline), mecamylamine (Inversine), diazoxide (Proglycem), prazosin (Minipress), and clonidine (Catapres) have all been reported to be effective in managing the hypertensive crisis of AD.[33,128,224] However, there have been no randomized, controlled studies reported comparing the use of various medications for the control of hypertension in AD. Information reported comes from case reports and a small series of patients.

Eltorai[66] reports the following medications and dosages as helpful in mild cases of acute hypertension due to AD:

1. Nitroglycerin: 2.5-cm (1-inch) strip of 2% paste or ointment applied to the upper chest or back and a second 1-inch strip applied as needed; alternatively, 0.15- to 0.6-mg tablet sublingually, repeated as needed

2. Clonidine (Catapres): 0.1 to 0.2 mg orally every hour up to 0.8 mg (central α_2-adrenergic receptor stimulator)
3. Captopril (Capoten): 25 mg orally and repeated as needed (angiotensin-converting enzyme inhibitor)
4. Prazosin (Minipress): 1 to 2 mg orally and repeated every hour as needed (peripheral α_1-adrenergic blocker)
5. Labetalol (Normodyne, Trandate): 200 to 400 mg orally every 2 to 3 hours

If hypertension is unresolved by either nitropaste or oral medication, parenteral treatment may become necessary. If possible, the individual should be transported to a medical facility for further treatment. However, evacuation from the wilderness can take hours or days and treatment must not be delayed. Intravenous nitroglycerin is easily titrated and can be initiated for a rapid drop in blood pressure. The hypotensive effects are eliminated in 2 to 3 minutes when the medication is discontinued. Hydralazine intravenously or intramuscularly is another option. It has an onset of action of 10 minutes, but the drawback is the long duration of action, which is 6 to 8 hours. This medication is best used when it is known that the trigger of AD will not be resolved within a shorter time.

Because bradycardia can be one of the symptoms of AD, antihypertensive agents with a β-adrenergic blocking action are not recommended for persons experiencing bradycardia.[162]

Prevention. The first step in prevention is for the team to know the level of the SCI and the pertinent medical history of the disabled individual. Information critical for the medical leader includes

1. Level of the injury
2. Preferred method of bladder management
3. Previous complications from prior wilderness excursions
4. If the SCI is T6 or above:
   a. Frequency of AD events
   b. Normal blood pressure
   c. Most frequent cause
   d. Most effective management

An injury at T6 or above should alert the team physician or leader to further inquire about the medical history. If the individual is prone to AD, the usual stimuli for that individual should be communicated with the team physician, team leader, or other designated team member. Medications previously found useful in treating AD for that person should be carried in the medical kit along with a selection of other antihypertensive agents. The individual should carry pertinent information about his or her history of AD, the signs and symptoms previously experienced, and a treatment protocol for this condition.

Urinary Tract Infection

Urologic complications were once a leading cause of morbidity and mortality for people with neurogenic bladder.[230] Antibiotics, improved equipment and bladder management techniques, and increased awareness and education of patients and caregivers have contributed to a reduction of urinary tract infections (UTIs).

Bladder disorders continue to be nearly universal in people with SCI,[36] and bacteriuria in patients with neurogenic bladder continues to be a problem. Chronic bacterial colonization is present in the bladder in the spinal cord–injured person who depends on catheterization.[11] Individuals with a complete SCI are at high risk of having bacteriuria with positive culture results. UTIs in this population are frequently initially asymptomatic, polymicrobial, and very likely to recur or relapse.

Bladder management for the individual with a neurogenic bladder is accomplished most frequently by one of three methods: indwelling catheter (suprapubic or urethral), condom catheter, or clean intermittent self-catheterization (CISC). Indwelling catheters are used most often for the paralyzed individual with a high-level injury and no or very limited hand dexterity. CISC is widely accepted as the method of choice to manage the neurogenic bladder in the SCI population and is the most commonly used method of bladder management at time of discharge from rehabilitation centers.[165,240,264]

The bladder and sphincter both contract in the neurogenic bladder, but are not in synergy. Condom catheters are used for reflex voiding. To enable reflex voiding, the bladder neck and urethral sphincter are relaxed with α-adrenergic blocking agents such tamsulosin HCl (Flomax), doxazosin (Cardura), or terazosin HCl (Hytrin). The bladder continues to contract involuntarily and the outflow of urine into the urethra is unobstructed. A sphincterotomy can also decrease impedance of the urine from the bladder into the urethra. The condom is placed and remains on the penis at all times. The urine flows into the condom collection device in varying intervals and amounts. The device should be changed at least once a day.

Clean intermittent self-catheterization is the most frequent method chosen by paraplegics to manage a neurogenic bladder. For CISC to be successful, the urethral sphincter must remain constricted to retain urine in the bladder while the bladder is relaxed to allow for filling. Medications of choice to relax the bladder are oxybutynin chloride (Ditropan) or tolterodine tartrate (Detrol). The bladder is catheterized and drained intermittently. Condom catheters and CISC bladder management are not interchangeable systems because of alternative methods of managing the neurogenic bladder.

A device worthy of mention for bladder management for men with tetraplegia is a recently invented device called the HouseHold. This device, invented by J. Glen House, MD, a C7 complete tetraplegic, allows men with little or no finger dexterity to perform independent self-catheterization (Figs. 90-8 and 90-9). Individuals who previously were dependent on others for catheterization have been able to perform independent CISC on their first attempt.

This assistive device was evaluated in a study by Adler and Kirshblum.[2] The results demonstrated a simple learning curve with participants able to perform all aspects of CISC independently within 5 minutes on their first attempt. Dr. House used the HouseHold with clean catheterization technique without infection for 2 years. It is easily cleaned with soap and water. Information about the HouseHold self-catheterization device is available by e-mail at flexlife@msn.com or telephone at (281) 360-0067.

The wilderness presents an environment that could potentially increase the risk of UTI in the CISC population. Risk factors of intermittent catheterization include less-than-optimal hygiene (hand-washing and perineal cleanliness), dehydration, or too infrequent catheterization owing to inconvenience or the need to conserve supplies. The use of condom catheters for bladder management also increases risk for UTI; risk factors include poor hygiene and less-than-daily condom catheter changes.

Signs and Symptoms. People without sensation do not experience dysuria and the other common symptoms of a bladder

Figure 90-8. The HouseHold in open position. (Photograph courtesy of Glen House.)

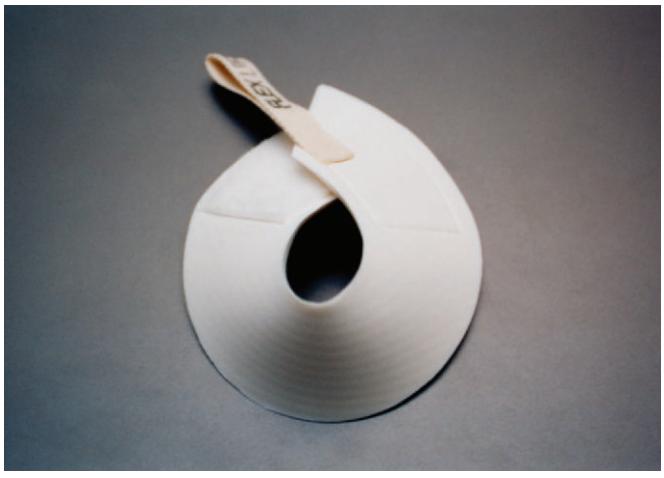

Figure 90-9. The HouseHold in functional position. (Photograph courtesy of Glen House.)

infection. Besides developing signs and symptoms of AD from bladder distention or infection, other symptoms may include tachycardia, malodorous and cloudy urine, increased muscle spasticity in patients with upper motor neuron lesions, fever, lethargy, nausea, and vomiting.[141]

Treatment. The question arises as to when to treat a bladder infection in the wilderness. It is very helpful to know the history of the person's experience and frequency of UTIs, the signs and symptoms he or she usually experiences, and the effective treatment protocol.

For the person with SCI, antibiotic therapy should be initiated when the bacterial colony count exceeds 100,000/mL.[162] However, without access to laboratory equipment for a urinalysis, a bacterial count in the wilderness is impossible. Therefore, treatment should be initiated based on the presenting signs and symptoms of the patient.

Color and clarity of the urine can be observed for indication of blood or sediment. Urine can be collected into a clear container and held against snow, a white shirt, or other white item for improved accuracy of color assessment. Ambient light conditions must also be considered when looking at urine color, because many things, including urine in a clear container, can take on the orange hue of a brilliant sunrise or a blazing sunset. Even such things as the vivid pink, orange, and red colors of clothing can impart an ambient glow affecting apparent color of a clear tube of urine. For example, being inside an orange tent will most certainly affect an accurate color assessment. Urine clarity and inspection for sediment is less affected by ambient light hues. Based on the individual's signs and symptoms and the gross appearance of the urine, a clinical diagnosis can be made, leading to the decision to begin antibiotic therapy.

A urine dipstick test kit can test up to 10 components of urine. The ability to detect leukocytes could be an invaluable resource. Because people with SCI always have colonized bacteria in their urine, the marker for an infection is elevated leukocyte count. The test strip indicates whether white blood cells are present in the urine. The pathologic concentration is more than 20 leukocytes/μL. The strip gives a good qualitative and only a fair quantitative representation. The intensity of the color that forms is proportional to the number of leukocytes present in the urine sample. Low specific gravity, oxalic acid, or traces of oxidizing agents may cause false-positive results. Sensitivity may decrease when urine is concentrated or contains glucose, albumin, blood, tetracycline, cephalexin, or gentamicin. The test kit instructions should be taken into the wilderness to enable accurate readings. It is important for the prospective wilderness adventurer to test his or her urine at home before the trip, when the person is asymptomatic and when it is known that no bladder infection is present. This provides a baseline of information against which to compare wilderness test results. The outcome of the leukocyte test before the trip should be documented in the wilderness medical record and available for reference.

The urine test kit is a 10-in-1 chemical strip. In addition to leukocytes, it tests for bilirubin, blood, glucose, ketones, nitrite, pH, protein, specific gravity, and urobilinogen. This product is available for purchase at www.anytestkits.com.

If UTI occurs in the wilderness and treatment is ineffective, it can cause serious illness for an individual, resulting in early termination of the trip for the disabled person and supporting team members. The following case study shows an example of

Figure 90-10. Jeff Pagels pedaling the One-Off wheelchair on a rough road where vehicles require four-wheel drive. (Photograph courtesy of Sheryl Olson.)

an individual becoming septic as a result of UTI during a climb on Mt. Kilimanjaro.

Case Study

A mountain climber, RB, became paralyzed at age 34 after a T10 complete SCI.

Before 1984, RB was an able-bodied athlete. In college, he was an All-American swimmer. As a teenager and into his thirties, he was a top-ranked canoe and kayak racer. He also completed the prestigious 55-km Birkebiner cross-country ski race six times and completed many triathlons.

RB became disabled in 1984 when a tree fell on him. He was left with a complete T10 SCI. A wheelchair user since then, he has become the United States' most decorated cross-country skier, with five Paralympic medals. He is always trying to go deeper into the woods and climb higher. RB spends much of his time helping others to challenge themselves while enjoying outdoor sports.

To celebrate his 55th birthday, RB decided to attempt a climb of Mt. Kilimanjaro. The expedition began on September 23, 2003 with a 20-hour airline flight. RB noticed that he was developing upper respiratory symptoms on arriving in Africa. The first 3 days were spent in the town of Marangu. On day 4, RB awoke with a full-fledged cold, runny nose, and sore throat. The team climbed for 6.5 hours, alternately using the one-off specialized chair and the wheelchair as needed, arriving in camp by dusk (Fig. 90-10). Day 5 was a 15-mile (24-km), 3000-vertical-foot (900-vertical-meter), 10-hour trek. (This same trek would take an abled person an average of 4 to 6 hours.) The trail was packed with obstacles—rocks, roots, and drainage ditches. It was very slow going for the team as they lifted, pulled, and carried RB and the mountain wheelchair up and over the barriers.

Day 6, a rest day, was followed by an elevation gain of 3500 ft (1070 m) on day 7. On summit day, day 8, RB and his mountain chair successfully reached 17,500 ft (5330 m). The scree on the trail was like sinking sand. As very short distances were gained forward, the wheels sank deeper and were quickly buried to their hubs. RB had climbed as high as his equipment would allow, so he turned around and returned to the camp he had left earlier that day. What took 4 hours to ascend took only 1 hour on the return.

October 1, day 9, the team completed its descent down the mountain. RB had "a full-blown head cold with plugged ears and a head that felt like it was going to burst." In his diary, he wrote ". . . and the farther I went down the mountain, the less I could hear and the larger my head felt." He had difficulty equalizing the pressure in his ears. The road down contained many obstacles. RB described the ride down as "bone-jarring and dusty." One team member reported the incredible amount of pounding RB's body must be taking and noted that RB could not even know what was happening to half of his body as it bounced and bumped traveling over the mountain trail. The team checked into the local hotel, elevation 6000 ft (1830 m), at completion of the climb.

The next morning, day 10, RB awakened quite ill. He was coughing violently, unable to retain liquids or food, nauseated and vomiting, and had a rising temperature. RB noted his urine had been dark and cloudy for several days. The team physician started an intravenous (IV) line. RB remained in bed throughout the day and received 2 L of saline the team had in their medical kit. At 2200 hours that evening, without signs of improvement, RB was taken to the hospital. He was admitted, given an antiemetic, tested for malaria, rehydrated, and started on ciprofloxacin and levofloxacin for UTI.

He was visited the next morning by three physicians, an African, a German, and a Norwegian. They recommended that he remain in the hospital for a week before making plans to fly home. He chose not to remain confined and was discharged under the care of the team physician. He returned to the hotel with continuation of IV fluids and antiemetic medication. He commented on the culminating challenge of the events. "Several days of medications, hit-and-miss food, and a return from dehydration all played a part in a bad onset of diarrhea." Diarrhea is not easy to deal with when one cannot feel or anticipate its occurrence.

Oct. 3 to 5 were spent relaxing, resting, and sightseeing. Oct. 6, day 14, the team began the long flight home.

During the flight, RB developed severely swollen legs about 13 hours into the journey. The second day home, he had a Doppler venous ultrasound that was negative for deep vein thrombosis (DVT). The leg edema gradually resolved over 48 hours.

Three weeks after return to the United States, RB, still not feeling well, was admitted to the hospital. He had a UTI and blood culture results were positive for *Escherichia coli*, resistant to levofloxacin. He was started on cephalexin for sepsis. The infection cleared over the next 10 days.

DISCUSSION

Controversy about the relationship of long-distance travel and DVT and pulmonary venous thromboembolism (VTE) continues to exist. Evidence that prolonged travel predis-

poses to DVT comes from empirical observations of travelers presenting with DVT immediately after landing. A recent study evaluating the effects of sitting quietly for 2 hours reported an increase in blood viscosity and hematocrit level locally in the legs, but not systemically.[112] Long-term and quiet sitting causes local venous blood stasis and decrease of venous blood return from the legs. Several hours of airplane travel may create a risk for development of DVT, especially in persons with recognized risk factors, including a previous episode of thrombosis, hormonal therapy, recent surgery, malignancy, pregnancy, and certain hematologic abnormalities.[84] Scurr found a much higher incidence of asymptomatic venous thrombosis than expected in a prospective study linking flying and development of thromboses.[213] The LONFLIT1/2 study results established the incidence of DVT between 4% and 6% in high-risk individuals after flights greater than 10 hours.[41] DVT or VTE can also occur in persons with no other health-related risk factors.[73,260] A Norwegian study concluded asymptomatic DVT may occur in 10% of healthy airline passengers older than 50 years of age and up to 4.5% of those younger than 50 years.[21] Deaths from pulmonary embolism caused by DVT have been attributed to long-distance flights.[14]

RB did not develop leg edema during the flight to Africa. The plane was only partially full for the longest leg of the journey, so he was able to lie down by stretching across four seats. On the return, the planes were full and he was confined to the space of a single seat. He donned graduated elastic stockings in Amsterdam when he noticed that his legs were edematous. He also was able to move to a bulkhead seat, where he could prop his legs against the wall in front of the seat in hopes of minimizing or preventing continued swelling. At present, there is no literature discussing the potential increased risk of DVT due to airplane flights in the mobility-impaired individual.

Risk factors for increased blood stasis and viscosity include sitting in a cramped position for several hours without exercise or change in position. Low humidity and reduced fluid intake contribute to the risk for dehydration and increased blood viscosity.[211] Information is widely available to the public describing preventive behaviors to be used during travel, including leg exercise, adequate fluid intake, and use of compression stockings.[23,104,105,106,119,146,222,260] Compression stockings create a pressure around 20 mm Hg at the ankle, gradually decreasing further up the leg. The cutaneous veins are compressed, shifting some blood to central sites and increasing deep vein blood flow.[217] Studies have demonstrated that graduated elastic stockings reduced the incidence of edema and asymptomatic venous thrombosis.[119,213] Thromboprophylaxis with low–molecular-weight heparin may be considered for individuals at high risk for DVT and VTE.[21,211,212]

RB noted that during the trip he chose to minimize his liquid intake in order to minimize the need to catheterize. The mobility-impaired person should be aware of the risk factors involved in travel longer than several hours' duration and should take precautions to minimize risk of DVT or VTE. Adequate fluids should be consumed in-flight to maintain hydration and decrease the risk of concentrating the urine. Sitting in an upgraded class provides more room for position change and movement and for accomplishing bladder maintenance tasks. RB developed edema of his legs during the flight. Although the use of graduated compression elastic stockings has been studied only in people with normal mobility, their use may also prevent edema in the mobility-impaired individual.

Pressure sores are another risk on long flights. The mobility-impaired person should shift position frequently and be certain there is not an unusual pressure point on an extremity.

During the expedition, the usual bladder routine for RB consisted of self-catheterization whenever he thought it was probably needed. He was able to know this based on time intervals, amount of fluids consumed, visually observing leg spasms, and sometimes sensation. Leg spasms are his most common symptom indicating the need for catheterization. Occasionally, if his bladder is quite full, he can feel a dull painful sensation in his lower left side. Incontinence, although not a common problem for RB, can be a problem for some paralyzed individuals when changes in daily routines occur.

Even if one wears gloves for warmth or protection while hand cycling, hands still get very dirty. The team was limited on the amount of water available during each day's climb, which made soap-and-water hand cleaning impossible. Other waterless methods of hand cleaning had not been planned and were not available. RB would wipe the dirt from his hands without benefit of antisepsis before catheterizing. His inability adequately to clean or disinfect his hands undoubtedly predisposed to an increased risk of bladder infection.

RB's cold symptoms were quite severe and (as can be seen retrospectively) were masking the early UTI symptoms. RB knows that sometimes with a cold he gets warm, achy, and stiff, which are the same symptoms he gets with onset of a UTI. In addition, he was taking hydrocodone as needed for syringomyelia pain and meloxicam for arthritis symptoms related to increased strenuous physical exertion. Both medications could have been masking UTI symptoms.

Prevention. An individual bladder routine for a disabled person embarking on an extended travel or wilderness adventure requires careful planning and preparation to minimize the risk of developing an infection.

Hands should be cleansed with soap and water when it is available. If water is in limited supply, options are to use waterless antiseptic gel or to wipe hands with a moist towelette before catheterizing. Clean or sterile gloves are not a prerequisite for CISC, but could certainly be used if it is not possible to clean the hands adequately.

Prevention also includes provision of adequate bladder management supplies, whether the individual uses CISC or condom catheters. The person using CISC technique should self-catheterize frequently to prevent bladder distention. A condom catheter should be changed daily and the bag emptied every few hours. Equipment should be kept clean and protected from environmental contaminants.

The closed catheter system is ideal for CISC in the wilderness (Figs. 90-11 and 90-12). Although not yet studied in a wilderness environment, it can theoretically decrease the risk of bladder infection because the catheter is not easily contaminated by hands during insertion. It is a prelubricated sterile catheter contained in a urine collection bag. The tip covering the catheter

Figure 90-11. Sterile, closed-system field catheter as packaged. (Photograph courtesy of Sheryl Olson.)

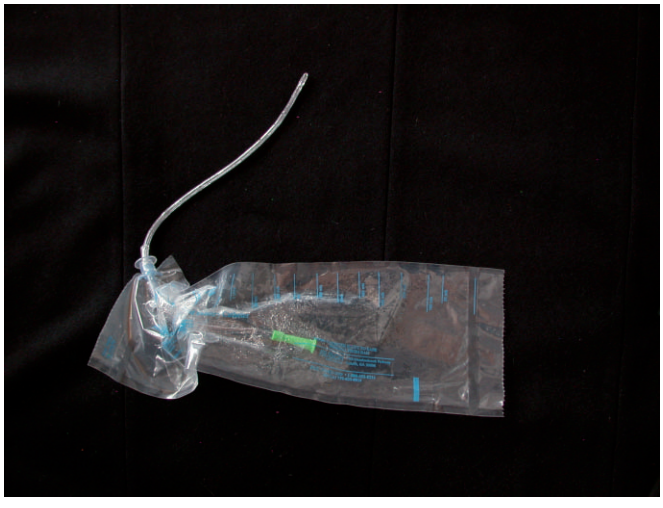

Figure 90-12. Sterile, closed-system catheter ready for insertion. (Photograph courtesy of Sheryl Olson.)

is removed and the individual inserts the catheter directly into the urethra while handling only the outside of the collection bag. The urine drains into the bag, and after the catheter is removed, the entire device can be disposed of properly. In the wilderness, a small hole could be cut in the end of the collection bag to allow the urine to drain onto the ground or into a river, according to appropriate wilderness protocol. The closed catheter system can be used with the HouseHold device for persons with limited dexterity (it can be purchased at www.sportaid.com). Several companies make similar products. Abundant catheter or condom supplies should be included in the expedition equipment.

An adequate potable water supply should be available throughout the wilderness excursion to encourage maintenance of adequate hydration. It is wise to have at least two optional methods of water disinfection available for the entire time of the trip. The average adult person during normal daily activity requires approximately 2 L of water daily. Fluid intake during prolonged or strenuous exercise should be increased appropriately. Changing the amount of exercise and amount of fluid consumed could potentially change the catheterization schedule. It is wise to catheterize more frequently than might be deemed necessary in the early stages of the wilderness experience. As the individual with SCI becomes accustomed to his or her personal needs and learns to balance fluid intake with varying degrees of exercise, the bladder routine can be adjusted and scheduled to minimize risk of bladder distention, spasm, and AD.

Conscientious monitoring of fluid intake is mandatory in the prevention of urinary problems for the person with SCI. If the water supply is ample and the outdoor adventurer is limiting his or her intake, inquiry should be made as to the reason. Inadequate fluid intake may be the result of the individual wanting to catheterize less frequently in the wilderness environment.

Cranberry juice supplementation has not been found to be an effective preventive treatment regimen in individuals with indwelling catheters. It may be beneficial as an adjunct to other types of bladder management. There are no data to support prophylactic antibiotics as a preventive measure against UTI.

It is important to remember when planning the wilderness expedition that the normal bladder routine for the disabled traveler is disrupted on commencement of the trip. Airplane travel in itself necessitates adaptation to a new routine. Normal eating, drinking, and bladder and bowel schedules are interrupted. Frequently, fewer fluids are consumed during flight, which predisposes to dehydration. Airplane bathrooms are generally not handicap accessible; therefore, catheterization must be accomplished in the airplane seat under a blanket. Crowded planes, small seating space, minimal lighting at night, lack of accessible hand-washing facilities, and disposal of urine are all obstacles that can decrease the desire to consume adequate liquids. The mobility-impaired traveler should be mentally prepared to manage his or her bladder needs for a long airplane flight. Practicing CISC under a blanket and in dim lighting can build the confidence of the traveler preparing for flight. The bowel routine schedule should also be adjusted to accommodate the flight schedule so that an adequate restroom facility is available for the necessary regimen.

Inability to clean adequately after a bowel routine can contribute to an increased risk of bladder infection. At home, it is convenient for a disabled person to shower after a bowel routine. In the wilderness, where cleaning is just as important and much less convenient, an improvised bidet can be made by using a solar shower. After the bowel routine, the person sits on the wheelchair with the false seat (as described later in this chapter) removed. The nozzle of the solar shower can be sprayed on the perineum without touching the nozzle to the skin. The solar shower used for this purpose should be labeled as such and not shared by the group for hand-washing or other bathing purposes. Another method is to use a 60-mL syringe and gently spray water over the perineum. Hands should be cleansed thoroughly after each bladder and bowel routine.

Special Considerations for a Wilderness Adventurer with Spinal Cord Injury

Logistical preparation for any kind of wilderness adventure requires focused attention and energy. If specialized equipment

Figure 90-13. Leg and ankle securely strapped to off-road wheelchair to prevent injury to leg. (Photograph courtesy of Sheryl Olson.)

for mobility aid is needed, it must be disassembled and packaged in a sized container that will fit through the cargo door of an aircraft. Airplanes flying to remote areas are frequently quite small with cargo doors critically limiting the size of luggage or gear that will fit. A call in advance to the airline asking what requirements and special procedures need to be taken may save frustration on the trip. Asking the size of the cargo door provides important information when packaging a large piece of equipment. Obtaining a letter from a doctor stating that a piece of baggage is required medical equipment may result in allowance of more than two bags without a surcharge.

Some parks and wilderness areas do not usually allow any travel device with wheels to be used on trails. Permission to use such devices should be obtained before the trip. The local regulatory agency of the area to be traveled can be contacted to secure written permission for use of a wheelchair or other mobility assist device.

Maintaining safety in use of the mobility equipment must be foremost. The paralyzed parts of the body should be well secured to the equipment that is being used (Fig. 90-13). It is the responsibility of the support team to monitor ongoing safety throughout the trip. If a paralyzed extremity becomes unsecured, it could loosely flop around or become entangled in the equipment, potentially causing severe injury. Such was the case for a paraplegic enjoying an outing on an all-terrain vehicle (ATV). He reported "...I broke my foot and leg when my foot fell off the ATV and I rolled over it with the rear tire." (Personal interview.)

Prolonged use of new or unfamiliar equipment can cause localized pressure or abrasions. Equipment needs to fit properly to minimize pressure sores and overuse injuries. When possible, new equipment should be broken in gradually with training intervals increasing gradually for time and intensity. Equipment such as a climbing harness, scuba buoyancy control device (BCD), or simply unpadded straps around the legs to keep them in proper position on a mobility device can all cause potential points of irritation.

Preparation for an expedition or for any activity that is above and beyond the usual daily level of activity requires training before the event. The individual must acquire technical and physical skill in use of the piece of equipment that is being used. Cross-training routines and programs should follow similar standards for both physically disabled and able-bodied persons. Training should focus on a combination of cardiovascular and strength-building programs. For longer expeditions, such as mountain climbing, training should develop the ability to do tolerable amounts of work over an extended period of time. For example, in preparation for the climb of Mt. Kilimanjaro, RB had a very specific daily routine. He typically got up in the morning, hand-peddled his one-off wheelchair for 10 miles, ate breakfast, peddled another 10 miles, arrived home, sat in the hot tub, and then went out to peddle another 10 miles.

It is necessary that every member of the team spend some time training with the disabled individual before an extended wilderness adventure. This provides the support crew an opportunity to learn about the needs of the individual and what assistance will be required. Roles and responsibilities can be identified during this time, allowing the team to work through actual problems that arise, identify potential complications, and implement problem-solving techniques. Adequate preparation will result in fewer delays during the actual event, enhance the ability to follow the preplanned time scheduled, and provide for an overall safer, more satisfying event.

The athlete should have intermittent assessment of skin integrity with careful observation for skin damage of any kind including pressure sores, blisters, or exposure. Any point of contact of a body part with a piece of equipment should be looked at initially every 5 to 10 minutes. This includes situations such as a foot or ankle rubbing against the inside of a canoe or kayak; checking for a stone or other type of debris getting between the buttocks and the seat, or legs being flexed too severely, thus impeding circulation to the periphery. Adjustments to body positioning and equipment may need to occur frequently, and depend somewhat on the strenuousness of the physical activity. If the participant sits on the ground, a rock, or a log, the team should check the underlying surface for irregularities to help avoid a raised pressure area. If necessary, the seat surface can be padded by using a closed-cell foam pad, inflatable mattress, or extra clothing. Any sleeping surface should be well protected from small stones or other lumps that could cause an unwanted pressure point.

Because paraplegics use their hands so frequently, it is important to minimize the risk of disruption of skin integrity by preventing extreme dryness, hangnails, cuts, and scrapes. A good-quality moisturizer should be applied each night to the hands. When using the hands for vigorous physical activity, use of a durable, well-fitted glove is essential for protection.

Gloves need to perform different functions in various wilderness environments. At present, there are no studies evaluating the best types of gloves for chair pushers participating in specific sports and activities. The most significant consideration for gloves, regardless of the activity, is high durability. The glove must withstand the constant friction between the hands and the mobility assist device. Gloves need to be soft and smooth on the inside to prevent blisters and skin irritations. The fit must be snug, but not constricting. Gloves that fit too loosely allow chafing and formation of blisters. In addition, gloves need to perform the required functions of warmth, wicking, water repellence, and wind blocking. Mittens provide greater warmth than

gloves, whether wet or dry. Gloves made from goat skin are more durable than those made from other types of animal hides.[138] Saranac gloves or mittens, used by law enforcement, football players, mechanics, motorcycle riders, and others, provide excellent protection for the hands. However, they are not waterproof. At the time of printing, no winter glove is manufactured that can stand up to the force of wet snow applied between the hand and wheelchair rim. The pressure drives the moisture through all layers of the glove, regardless of the type of glove used. The best advice is to carry several pairs of gloves or mittens and change them when they get too wet. If hands become cold, small hand warmers or heat packs can be used inside the glove or mitten to provide an element of active warming.

During an activity, the mobility-impaired person should change positions at regular and frequent intervals. This includes getting out of the wheelchair or other mobility-assist device to stretch the arms, legs, and back. Time and mileage goals for each day's journey should be adjusted appropriately to allow for these critical rest stops.

Exposed body parts should be covered with sunscreen of sufficient SPF to prevent sunburn to the skin. The exposed skin can also be covered with clothing that has built-in sunscreen protection. Particular care must be taken to protect or cover the parts of the body with decreased or absent sensation. If the person is taking sun-sensitizing medication, extra precautions for protection from the sun are imperative because of increased susceptibility to sunburn.

Frostbite can be a potentially severe problem in cold environments because of lack of normal skin sensation. Before embarking for an extended period in a cold environment, a trial run should be made to test the clothing and equipment for warmth and functional worthiness. It might be necessary for the individual with SCI to wear larger pants, shoes, or boots to accommodate extra layers of insulation. The toes and feet should be checked frequently for warmth and circulation. Wet clothing and foot gear should be removed immediately at the time of cessation of strenuous physical activity. Caution must be taken when around a campfire to prevent burning areas of the body that lack sensation. A visual inspection of the condition of the skin can be done to identify early signs of loss of skin integrity. The entire team must be vigilant about the need to protect, inspect, and maintain specific skin areas for the individual with SCI.

RB reported that before his injury, his feet would sweat, enhancing his cold misery. Since his SCI, his feet do not sweat, and when he touches them to assess warmth, they are warm. A more significant problem for him is difficulty in becoming warm once he is cold. When camping, he must avoid staying outside too long in the evening because he has a tendency to become chilled and then have difficulty sleeping. Changing into dry clothing and wearing multiple layers of clothing on both upper and lower body helps him to prevent chilling after a day of exercise. He finds that once he is inside his sleeping bag, hand warmers placed at the neck, groin, armpits, and hands help to keep him warm.

Compliance with medication routines should be maintained. If the person is taking a medication, such as an antibiotic for UTI, the primary care physician should be consulted for dosage management during the expedition. At least several extra doses of each medication should be taken on the trip. The specific amount carried depends on the number of days of the expedition, remoteness of the environment, location of a source for resupply, and potential for delay in departing the wilderness. In general, it is advisable to carry 5 to 7 days' additional supply of routine medications for extended trips in remote environments.

Dietary intake and the bowel program should be kept as close to the normal routine as possible. Changes in activity, diet, and water can cause constipation or diarrhea. If the adventure is going to require a significant dietary change, the new foods should be tried at home during the training period before the adventure. Adaptations to the bowel routine can then be made based on the physiologic needs as a result of the new diet. Once in the wilderness and eating the tested diet, the adventurer can usually prevent undesirable events through faithful adherence to the new bowel program.

Sometimes the wilderness environment or the activity itself is not conducive to maintaining a regular bowel routine. A cold environment where many clothes need to be worn for warmth, the need to pack out all human waste, lack of private personal space, or being otherwise preoccupied are all factors that can drive a decision not to perform a daily bowel routine. Such was the situation with SC in the following case report:

> *SC has a complete T11 SCI. Being quite athletic, one of his goals was to climb Mt. Whitney. Before an event of any kind, his routine was to empty his colon as thoroughly as possible. His last bowel routine was early morning on the first day of the climb. With increasing altitude, his appetite diminished, which was typical for him. He skipped his normal bowel routine for the following 3 days. He stated, "Of all the things I had to worry about (on the mountain), this was one that slipped between the cracks." On day 4, the team was off the mountain by 4:00 PM. The news media were present and there was much celebration with food and beverages that continued into the evening hours. SC forgot about the pending detail of a bowel routine. At 4:00 AM, while in his sleeping bag in the bunk house, he was awakened by a horrific smell. He had suffered an uncontrolled bowel movement. The greater difficulty was getting out of the bunk, down the hall to the bathroom, and into the shower, where he cut the sleeping bag off in chunks.*

Fecal elimination in the wilderness can be managed quite well with preparation, planning, and practice. Wheelchairs can be adapted to become toilet seats. The chair seat fabric is modified by cutting a toilet-seat–sized hole in the fabric. The edges of the fabric should be stitched or reinforced to prevent unraveling. One side of Velcro, either the loop or hook half, is sewn around the edge of the seat (Fig. 90-14). The original wheelchair seat, now with a hole in it, is covered with another layer of fabric the same size as the original wheelchair seat (Fig. 90-15). This becomes a false seat. It has the other side of Velcro around the edge and, when in place, allows the chair to be used as normal. The top padded cushion is placed on top of the false seat. Removing the cushion and the top false seat allows the chair to be used as a commode chair. This modified chair requires transferring off the chair in order to remove the pad and false seat.

Several options exist for easily and properly disposing of feces. Wilderness etiquette should always be followed for burying or carrying out stool, toilet paper, and other sanitary products. If not in an area requiring everything to be carried out, a cat hole, or pit, can be dug in the ground in a place that

Figure 90-14. Wheelchair seat modified to a commode seat; open position. (Photograph courtesy of Jeff Pagels.)

Figure 90-15. Wheelchair seat modified to a commode seat; closed position. (Photograph courtesy of Jeff Pagels.)

is wheelchair accessible. Using the wheelchair with the modified seat, the chair is then rolled over the pit and the bowel routine accomplished. If a cat hole cannot be dug, an option is to place a large plastic bag under the chair. After removal of the false seat, the bag can be arranged with the open end circling the hole in the seat. This procedure offers more privacy during the elimination process than wheelchair placement over a cat hole. It also offers easier management of the feces if the regulations of the particular wilderness area require that human waste be carried out.

Cat litter or other absorbent material can be placed in the plastic bag before the trip. The litter will absorb the odor and excess liquid from the stool, making the package easier to pack out without fear of liquid seepage should the stool be loose.

Box 90-5. Wilderness Medical Kit Options for Individuals with Spinal Cord Injury

- Nitropaste 2% (injury at T6 and above)
- Oral antihypertensive medication (to include one that is not a β-adrenergic blocking agent)
- Hydralazine (Apresoline), or other parenteral antihypertensive agent
- Nitroglycerin, sublingual and intravenous (IV)
- IV supplies for medication administration
- Lidocaine jelly 2%
- Antibiotics for urinary tract infection
- Suppositories for constipation
- Urine test kit
- Closed suction catheters
- Coudé catheter
- Antiseptic hand gel
- Moist towelettes
- Gloves, latex or nonlatex
- Sphygmomanometer
- Stethoscope (optional)
- Chemical hand warmer packets
- Personal medications with several days' extra supply of any medications taken on a routine, daily basis

Another option is to purchase travel toilet supplies from a company such as Travel Jon. This company sells individual bags for a one-time use or packages for a full day's supply of toilet items. A day's supply includes a large plastic bag for a bowel movement, several small plastic bags for urine (all with absorbent material), toilet tissue, and hand wipes. Anything not biodegradable should always be packed out of the wilderness.

To be adequately prepared to treat the most common and frequent complications of the mobility-impaired individual, adaptations should be made to the wilderness or expedition medical kit based on the individual's potential medical needs. Of paramount importance is the inclusion of several days' extra supply of routine medications. Box 90-5 summarizes the equipment and medications that have been discussed in this section. The items listed should be considered for inclusion in a wilderness medicine kit for individuals with SCI.

Cerebral Palsy

Cerebral palsy refers to nonprogressive neurologic conditions involving motor coordination and posture that result from an injury to the developing motor areas of the brain.[18] The physical manifestations depend on the extent and location of the injury in the brain. It is one of the most common physical disabilities in the United States. The reported incidence is 2 to 3 per 1000 live births.[91] As a result of difficulties with movement and coordination, activities in the wilderness may need to be modified for individuals with CP, depending on their level of function.

There is a great deal of diversity in the presentation of CP. An individual may be limited in his or her ability to move and maintain balance and posture. Some individuals have concomitant cognitive, auditory, visual, speech, and swallowing difficulties. In preparation for being in the wilderness, individuals with CP may benefit from an exercise program consisting of

strengthening, increasing range of motion, and maximizing endurance.

A study completed by a rehabilitation center in the Netherlands concluded 75% of people with CP are independent in ADLs, mobility, and communication.[247] The same study concluded that young adults with CP are poorly integrated in similar participation in employment opportunities and sports activities. People with CP require varying levels of mobility assistance based on the severity of their illness.

The Cerebral Palsy International Sports and Recreation Association (CP-ISRA) has developed a classification system based on an individual's functional abilities.[40] This system includes not only those diagnosed with CP, but individuals with other conditions that are characterized by nonprogressive lesions in the brain, including stroke, brain injury, and tumors. The classification system allows for the grouping of individuals with similar abilities. Based on an individual's level of function, recommendations for exercise testing and daily program can be developed in preparation for an individual to venture out into the wilderness. There are eight classes of ability level according to the CP-ISRA classification system. They are defined in terms of functional profiles, each with subsections for lower extremities, trunk control, and upper extremities (including hand control). Because the only rational way to make such classifications is through functional profiles that require several pages to define, the eight classes are not listed here in tabular format. Instead the reader is referred to the CP-ISRA's Classification and Sports Rule Manual, which is available free on the Internet[40] and provides a proper understanding of how the classification process works.

Whenever someone with muscle imbalance begins a new physically demanding activity, it is important to recognize the effect of exercise and the physiologic response. What constitutes a low-intensity exercise to an individual without CP (e.g., walking) may be a moderate- or high-intensity exercise for someone with a moderate form of CP. Suzuki used heart rate to compare subjects with and without CP to estimate exercise intensity while walking. The subjects were rated level one, two, or three according to the Gross Motor Function Classification System, with level one having the best motor function. Results of the study concluded that during the same activity, the heart rate of the level-one group increased significantly compared with healthy subjects ($P < .05$). The walking speed was decreased for level two and the heart rate continued to increase significantly ($P < .05$).[237]

Complications

The literature suggests that individuals with CP have a greater number of injuries to the knees, ankles, shoulders, and hands.[228] Wheelchair users are more prone to injuries to the shoulders and hands. Individuals who are ambulatory are more prone to injuries to the knees and ankles.

Individuals with significant neurologic impairment are more prone to development of dehydration and exhaustion because of the additional energy expenditure required for simple physical activities. There is reduced mechanical efficiency secondary to the extra energy required to overcome increased tone. Dehydration can be of significant concern in the wilderness. Not only does it predispose an individual to decreased muscle efficiency, it can lead to other problems, such as constipation. Fluids should be available and encouraged throughout the wilderness adventure.

Fatigue and stress also have a negative influence on a person's level of functioning. Exhaustion may result from uncontrolled or excessive movements. After stressful exercise sessions, individuals with CP were noted to have increased spasticity and decreased coordination. Pacing activity in the wilderness is important to minimize fatigue.

Musculoskeletal pain may also be a significant problem in adults with CP. From a response of 406 persons, a postal survey concluded almost one third of adults with CP had chronic pain, compared with 15% in the general population.[121] Back pain was the most common complaint. Other factors, such as sex, chronic fatigue, low life satisfaction, and deteriorating physical function, were associated with back pain.

Treatment

The level of severity of CP should be assessed and an activity in the wilderness carefully chosen and planned. The activity should be one in which the intensity and duration can be modified. Good examples of sports that can be adjusted to a slower pace include hiking, backpacking, cross-country skiing, or diving.

The individual should monitor and manage his or her level of pain during the activity and be supported by the adventure team to take rest periods and stop as needed. The management of CP-related pain is not well documented in the literature. A longitudinal study of 50 adults over 2 years found that only three treatments—whirlpool, ultrasound, and transcutaneous electrical nerve stimulation (TENS)—were associated with a decrease in pain.[124] None of these is readily available in the wilderness.

The participant should be monitored for overexertion, and frequent rest stops should be offered. The best activity will accommodate the individual according to the level of functional motor capability.

Multiple Sclerosis

Multiple sclerosis (MS) is an inflammatory disease of the central nervous system characterized by areas of demyelination. Destruction of myelin causes the formation of plaques on nerves that slow nerve impulses and produce symptoms of MS.[177] It is the third most common cause of disabling illness in persons between the ages of 15 and 50 years.[151] The etiology of this condition continues to be debated. Functional and cognitive impairments develop in the majority of patients. Frequently, gait, coordination, bladder control, and sexual function are affected. The course of MS is extremely variable, ranging from death within 5 years in 5% of patients to many cases that can be manifested by one episode in a lifetime.[150]

Very little research has focused on understanding how MS affects basic physiologic responses during increased physical activity. In one study,[198] four general areas were assessed, including autonomic control of heart rate (HR) and arterial blood pressure (BP), cardiorespiratory fitness, skeletal muscle function, and symptom instability under thermal stress. In the past, abnormalities in cardiovascular reflexes were observed in some individuals with MS during quiescent testing. In this study, HR and BP responses during exercise did not confirm such findings. Deficits in cardiorespiratory fitness were present in moderately impaired individuals, but not always present in people with minimal impairment. Similarly, abnormalities in skeletal muscle function were reported in some individuals with MS, while

absent in others. Training appeared to improve both cardiorespiratory fitness and skeletal muscle function.[198]

Weakness is often a significant problem in MS. It is important that general conditioning be maintained as long as possible. This has to be done in a way that does not increase fatigue or body temperature. Elevated body temperature can worsen symptoms of MS; many people with MS experience a worsening of symptoms when weather is hot or humid or if they become overheated from exercise. For example, some individuals notice that their vision becomes blurred when they get overheated, a symptom known as *Uhthoff's phenomenon*.[177] Heat generally produces only temporary worsening of symptoms and does not cause persistent tissue damage. Some people with MS notice that symptoms, particularly spasticity, become worse in cold weather. It is generally recommended that individuals with MS who are sensitive to temperature avoid extremes of either hot or cold.[184]

Fatigue is often a symptom seen with MS. The actual source of fatigue is not well understood. It may result from a combination of weakness, spasticity, ataxia, and depression.[120] The Social Security Administration has recognized the disabling aspects of fatigue and has allowed it to be considered as a major factor in granting disability benefits to individuals with MS.

Mobility problems are among the most common complaints in individuals with MS. Sixty percent of patients in surveys reported the need for some assistance with mobility.[17] Appropriate assistive devices increase safety and decrease energy expenditure.

A variety of other medical concerns have been described in individuals with MS. In the area of motor function, these include weakness, spasticity, ataxia, and dysphagia. Individuals sometimes experience eye muscle imbalance. Cognition can be affected, including decreased memory, attention, concentration, and problem-solving skills. Emotions may become labile. Bladder problems are usually characterized by urgency, frequency, incontinence, and retention. Constipation is one of the more frequent bowel concerns.

Individuals with MS can engage in wilderness experiences to the degree that they feel comfortable. Because MS is a progressive neuromuscular disorder, fatigue and stress may elicit attacks during exercise, causing acute increased disability.

Other Causes of Muscle Imbalance

Traumatic brain injury (TBI) is a significant source of disability.[134,237] It is defined as an insult to the brain caused by an external physical force that produces a diminished or altered state of consciousness. Each year, more than 2 million Americans sustain head injuries.[135] Just over 2% of the population lives with disabilities resulting from TBI.[39] Every year, 80,000 Americans develop long-term disabilities after TBI. Motor vehicle accidents are the leading cause. The risk is highest among adolescents, young adults, and individuals older than 75 years of age. Survival rates are improving secondary to improved care.

Individuals with TBI may fatigue more easily. Depression is also a potential long-term problem. Aerobic and neuromuscular training may improve oxidative capacity and improve locomotor efficiency.[122] From a psychological perspective, individuals with TBI who exercise report fewer negative symptoms and are less depressed.[88] There are a number of programs for people with TBI throughout the country that offer wilderness experiences, which help promote exercise and fight depression.

Stroke ranks second to TBI as a leading cause of neurologic disability. More than 500,000 new strokes occur annually in the United States. In the 1970s, nearly 2 million people in the United States survived a stroke.[17] More recent data indicate that there may now be almost twice this number. According to the NCHS (2002), 2.4% of noninstitutionalized adults have had a stroke.[261]

Hemiparesis is the most common major manifestation of stroke. This involves weakness or lack of coordination on one side of the body. In individuals with hemiparesis who are ambulatory, ankle dorsiflexion and eversion are usually weak, resulting in an altered gait pattern. The goal during rehabilitation is to normalize gait patterns. Normal gait promotes efficiency of ambulation and diminishes the risk of spine and joint complications. Using an ankle-foot orthosis can help provide stability around the ankle, promoting normal joint alignment and muscle function while walking. Normalizing ambulation with an orthosis decreases the amount of energy expended, decreasing oxygen consumption while walking. A more typical gait pattern enables people to walk or hike in the wilderness with maximum efficiency. If a person needs assistance, such as might be required on a mountain trail, it is usually most helpful to walk on the individual's affected side.

A metal walker with a wide base or a quad cane may be used to provide additional stability during ambulation. Each device is individually adjusted for proper height. Persons requiring a walker or quad cane most likely are too unstable to walk on a trail or other uneven wilderness terrain. Boardwalks have been constructed in some wilderness areas that allow an excellent wilderness experience, while at the same time providing a terrain free of mountain trail hazards. One such boardwalk is on Mt. Bierstadt (elevation 14,060 ft [4285 m]) in Colorado. The boardwalk passes through an amazing area of scrub willow below the summit and provides a mountain climbing experience that might otherwise not be possible.

Another option is to provide a wheelchair to enable expanded wilderness adventures. A hemiplegic individual who lacks independent ambulation usually has access to a hemiplegic wheelchair. This chair can be propelled independently using the unaffected arm on the wheel chair rim and the foot on the floor for propulsion and steering. Because of upper extremity involvement, individuals who have had a stroke may find it difficult to manipulate devices that require two hands. Equipment can be adapted to help promote functional independence, as described in the equipment section at the end of the chapter.

After both stroke and TBI, muscles can be spastic or dystonic. A variety of different treatments can be used to treat spasticity and dystonia. The goal is to minimize the risk of contractures and provide stabilization across joints for movement that is not well coordinated. Furthermore, slow motor responses are also associated with TBI and stroke. Motor slowness can be attributed to central processing delays or decreased coordination.

Disorders of balance and coordination may result from damage to the cerebellum or its connections. Some individuals who have good muscle strength are not able to ambulate or sit independently because of significant ataxia. Ataxia can interfere with a person's ability independently to perform ADLs, including eating, hygiene, and toileting. Ataxia is difficult to treat. Adaptations to equipment, such as weights or cuffs, may help with stabilization.

For individuals who have tremors, medications may be used to help control the frequency, amplitude, and occurrence during

different activities. Propranolol has been used to treat movement disorders. However, it may limit a person's cardiovascular response to exercise.

Amputation

According to the NCHS, it is estimated that there are over 1.2 million amputees in the United States. Approximately 75% of these are lower extremity amputations.[9] With advancing technology, amputees are now able to participate in a wider range of activities in the wilderness setting. Changes in prosthetic designs and materials allow more people with amputated limbs to lead a more active lifestyle.

As a result of widespread interest in recreational prosthetics, more information is available to amputees. In one study,[130] 100 individuals with lower extremity amputations were surveyed to determine their involvement and sports activities. Age rather than level of amputation appeared to be the major factor influencing participation in recreational activities. Most individuals returned to recreational activities in which they were involved before their amputation. Seven percent of individuals had special waterproof prostheses. Approximately 30% of the individuals believe that the prosthetist was knowledgeable about available components for sports and recreational activities.[130]

A socket is the connection between the residual limb and the prosthesis. The function of the socket is to protect the residual limb and transmit the forces associated with movement. The prosthesis attaches to the body using a suspension mechanism. There are many types of suspension mechanisms, depending on the limb that is affected and the desired function. Prostheses also have a pylon, joint, and terminal device. The pylon is a shell that attaches the socket to the terminal device. The joint connects the terminal device to the pylon. Different terminal devices have different functions. For example, in the case of a lower extremity amputation, there are five basic functions provided by a prosthetic foot. These include shock absorption, a stable weight-bearing surface, cosmetic appearance, anatomic joint function, and adequate strength.

Advances in new prosthetic foot and knee designs have enabled amputees to pursue more rigorous activity levels. Materials have been incorporated from the aerospace industry that allow designers to create lightweight feet that can be deformed under a load, then return to their original shape when the load is removed.[203] These dynamic elastic response feet have revolutionized the amputee's ability to function in a variety of environments. There are specially designed knee and foot mechanisms and lightweight sockets with excellent suspensions that allow people with amputations to move about more freely. The prosthetist is the best source of information about prosthetic technologies related to different sports.[71]

There are also a variety of prostheses for specific activities. For example, prostheses for rock climbing come in various shapes and sizes to accommodate different features in the rock. The sole of the terminal device can be fitted with a material to provide greater friction.

Prosthetic training techniques also take into account an amputee's recreational interests. Advanced athletic training concepts to achieve superior performance in a wide variety of activities are now being used. Before undergoing surgery, the patient's interests are taken into account by the surgeon. One goal of surgery is to allow an individual to have the longest possible residual limb segment. Energy consumption while walking with a prosthesis has been shown to be inversely related to the length of the residual limb.[254]

Cycling offers a chance for amputees to enjoy travel over longer distances. This provides an opportunity for aerobic conditioning without the high impact to the residual limb that occurs during running or hiking. A toe clip is often used to hold the prosthetic foot in place. Some transfemoral amputees choose not to use a prosthesis. Instead, the pedal on the prosthetic side is removed and a toe clip is used on the sound side to apply power on both downward and upward strokes of the pedal.[168]

Amputees often do not wear prostheses during aquatic activities. They can learn to swim without drifting toward the prosthetic side when kicking with the sound limb. However, it is sometimes challenging to maintain the trunk and shoulders parallel to the water surface. There are advantages to wearing a prosthesis while swimming. Propulsion is enhanced. It is also easier to get into and out of the water. Several types of swimming prostheses are available for below-the-knee amputees. The Otto Bock swimming leg is hollow. There is a hole drilled through the ankle. When one swims, water enters the hole, filling the airspace and decreasing the buoyancy of the leg. Once the swimmer leaves the water, the water in the airspace drains through the hole at the ankle. There is also an above-the-knee swimming leg, which has a quick-release lock mechanism. The single axis in the knee mechanism permits function in the water. The friction lock mechanism allows the amputee to walk on land. Swim fins can also be used with different prostheses.

Below-the-knee amputees can learn to snow ski on adaptive equipment with little difficulty. Bilateral below-the-knee amputees have two choices: they can use a three-track ski or one ski and two outriggers. Individuals with bilateral above-the-knee, hip-disarticulation, and hemipelvic amputations have the option of using a mono-ski or a sit-ski. A mono-ski usually has a seat that sits on top of a single or dual ski base. Shortened outriggers can be used for additional balance and to steer. The mono-ski may have up and down positions controlled hydraulically. The sit-ski is similar to a sled and is usually made of fiberglass. Picks are used to start, turn, and stop the sit-ski. Some bilateral above-the-knee amputee skiers have learned to use a four-track ski, which involves using two skis and two outriggers.[168]

Activities such as camping and fishing are fairly easy to perform with little adaptation to a prosthesis. Protective clothing can be used to help keep a socket dry. It is important to protect a prosthesis from getting wet if it is not meant to be wet. Elastomeric skins are also available to cover prostheses and provide protection from exposure to moisture. Hiking and hunting require walking over uneven ground. This puts more stress on the residual limb. Some people will use a prosthesis with a larger socket and pad the residual limb more to achieve added protection. Multiaxial feet make walking on uneven ground easier.

Upper extremity amputations are less common than lower limb amputations. The same type of treatment principles described for individuals with lower extremity amputations also apply to individuals with upper extremity amputations. Recent advancements have placed an emphasis on better grip strength and precision.

Complications

As part of the regular routine, it is important to check the components of the prosthesis for function. In preparing for more

rigorous activity, it may be necessary for a person to change his or her training regimen to adapt to a new prosthesis. Discomfort is the primary factor that limits most amputees from pursuing more rigorous activities. Causes of discomfort from prostheses may include poor fit, inadequate suspension, and limited shock absorbing properties.

When out in the wilderness, it is important to monitor the status of the residual limb(s), fit of the prosthesis, and body temperature. Back pain, an indicator of problems with prosthesis length, should also be addressed. Pressure sores can develop on the residual limb if a prosthesis does not fit properly. Blisters and abrasions should be treated immediately to minimize the risk of infection.

Occasionally, an individual will develop swelling over the distal part of the residual limb from a socket that is too tight proximally. Venous outflow causes obstruction. If there is too much space distally in the socket, swelling continues until the empty space is filled. The skin becomes red and indurated, developing an orange-peel appearance with prominent skin pores. Venous stasis ulcers can develop if the constriction is not relieved.

Amputees may also have difficulty with control of body temperature. For example, people with lower extremity amputations have decreased surface area over which perspiration and cooling can occur. As a result, there is a higher incidence of overheating. This can cause increased perspiration to the degree that the fit of the prosthesis is affected. A prosthesis is more likely to slip off when it is wet. Socks on the residual limb may need to be changed more frequently. Adequate hydration is essential.

MEDICAL CONDITIONS

See also Chapters 27, 28, and 29.

Hypertension and Cardiac Disease

Hypertension is one of the most common conditions in the general population. Blood pressure readings above 140 mm Hg systolic and 90 mm Hg diastolic for extended periods are considered high. In the short term, hypertension can cause a cerebrovascular accident. Long-term complications are associated with coronary artery disease, congestive heart failure, kidney disease, and cerebrovascular disease.

Studies have indicated that regular physical activity helps individuals with cardiac disease to maximize physical, psychosocial, and emotional well-being.[53,62,147] However, certain environmental conditions and strenuous activity place increased stress on the heart. For persons with coronary artery disease, high blood pressure, or diabetes, the response to stress due to increased activity and variable environmental conditions may be impaired.

There are a number of factors that affect cardiac function. For the purposes of this chapter, the two factors that will be addressed include cold environments and high altitude. The interactive effects of cold and the hypoxic stress of high altitude during rest or exercise are not completely understood.[193] However, given that both can have a potentially negative effect on an individual with coronary artery disease, it is reasonable to think that the potential risk in a cold, high-altitude environment is increased.

Cold exposure in individuals with coronary artery disease may predispose to an attack of angina pectoris secondary to increased myocardial oxygen demands. Exposure to cold generally results in increased arterial pressure, myocardial inotropic state, and cardiac work during rest.[194] One mechanism that might explain cold-induced coronary vasoconstriction is α-adrenergic input to receptors in coronary arteries.[109] This mechanism decreases the myocardial oxygen supply for a given level of demand.

Another mechanism is related to increased oxygen demand that arises from the local myocardial or peripheral vascular effects of sympathetic nervous and adrenomedullary hormonal stimulation. Individuals with coronary artery disease who inhale cold air during rest may also be at risk.[225] When one breathes in cold air, the temperature in the segmental bronchi is more than 3°C (5.4°F) warmer than in the upper trachea.[164] This has the potential to lower the core temperature. Lowering the core temperature by as little as 1°C (1.8°F) increases cardiac work and is associated with large increases in plasma levels of norepinephrine[79] and smaller increases in levels of epinephrine.[80]

Coronary vasoconstriction can also be triggered by cooling down one limb (the cold pressor test). Immersing a limb in cold water triggers coronary vasoconstriction, thereby decreasing coronary perfusion.[199] Coronary vasoconstriction during cold pressor testing appears to occur more often in people with atherosclerotic coronary artery disease, whereas healthy individuals respond with increased coronary perfusion.[178] The difference in the response is thought to be related to endothelial dysfunction secondary to the atherosclerosis.

The incidence of death from myocardial infarction increases during the winter months, even in moderate climates.[202] Winter recreational activities that require the use of the upper body are of particular concern because they increase metabolic demands. Walking over packed snow significantly increases energy requirements. Canoeing and cross-country skiing can involve high-intensity exercise and may be contraindicated for some individuals with coronary artery disease. Proper clothing is essential. Multilayered garments should be used. The face, ears, neck, hands, and feet should be adequately covered and kept warm.

Individuals with coronary artery disease who swim in cold water may also be at risk for the same reasons. Cardiovascular events cause 20% to 30% of all deaths that occur while scuba diving.[56]

If an individual has had a revascularization procedure to improve coronary blood flow, strict criteria must be met before return to diving. This would include a standardized multistage treadmill test ("stress test") for assessing cardiovascular health. The test was developed and described in 1963 by Robert Bruce, an American cardiologist. According to the original Bruce protocol,[263] an individual walks on an uphill treadmill in a graded exercise test with electrodes on the chest to monitor the heart rhythm. Every 3 minutes, the speed and incline of the treadmill are increased. There are seven stages, and only very fit individuals complete all seven stages. After 6 to 12 months, the individual should undergo a thorough cardiovascular evaluation, which includes an exercise stress test. The individual should be able to perform the level of 13 metabolic equivalents (METS)—stage 4 on the Bruce protocol. A MET represents the energy cost of activity in multiples of resting oxygen consumption (1 MET = 3.5 mL/kg/min). Because oxygen consumption is determined primarily by cardiac output in the absence of pulmonary or skeletal limitations, this information allows for

rough estimates of cardiac function. This is a fairly brisk level of exercise, equivalent to running progressively faster until the individual reaches a pace that is slightly faster than running an 8-minute mile. Performance at that level without symptoms or electrocardiographic (ECG) changes indicates normal exercise tolerance.[38]

High-altitude activities present another challenge for individuals with coronary artery disease. At high altitude, the partial pressure of oxygen remains 21%. However, because of decreased barometric pressure, the total amount of inspired oxygen decreases. This tends to stimulate ventilation during rest and exercise above rates experienced at low altitudes during the same activity. Alveolar and arterial oxygen pressures and arterial oxygen content fall below sea level values. Heart rate increases in an attempt to circulate more oxygen to the tissues. Cardiac output increases, thereby increasing the work of the heart. In turn, oxygen requirements for the myocardium increase, necessitating an increase in coronary blood flow. Activities with less energy expenditure can usually be tolerated without adverse effects.[161] However, an individual with coronary artery disease may have more difficulties with backpacking uphill or cross-country skiing.

Nine men with documented coronary artery disease who had exercise-induced angina or ST segment depression were studied by treadmill testing at an altitude of 1600 m (5250 ft) and again during acute exposure to 3100 m (10,170 ft).[173] Mean maximum oxygen uptake was reduced at altitude by an average of 11%. Ventilation, heart rate, and systolic pressure at submaximal workloads were increased at 3100 m, but maximum values were unchanged. Oxygen saturation was reduced at rest and during submaximal and maximal exercise. Angina or ST segment depression occurred at the same heart rate systolic pressure product (an indication of myocardial oxygen consumption), but at lower workloads. Systolic time intervals were unchanged at altitude. A target heart rate range of 70% to 85% of the ischemic end point rate at lower altitude predicted an appropriate level of tolerable exercise at high altitude. Based on results of the study, recommendation was made that the activity prescription for coronary patients with angina on arrival at high altitude should be based on heart rate rather than workload.

Huonker and colleagues evaluated the effects of highland mountain hiking on individuals with a history of myocardial infarction.[117] Cardiac rhythm was continuously recorded by a Holter monitor and analyzed for heart rate, ST segment deviations, and arrhythmias. Cardiac dysrhythmias were categorized as I to V according to the Lown classification. Doppler echocardiography was also performed. Individuals who had coronary artery disease showed a significantly lower systolic and not significantly higher diastolic resting blood pressure. The average maximum heart rate during mountain hiking did not exceed 110 beats per minute in both the control and target groups. Holter ECG monitoring during the mountain hike tour did not show ischemia-dependent ST segment alterations. Accelerated arrhythmias were not found in any subject.

Before commencing the mountain hiking tour for Huonker and colleagues' study, the patients with a history of myocardial infarction showed left ventricular dimensions in the upper normal range, which were significantly higher compared with corresponding values of the control subjects. This difference in the left ventricular dimensions was thought to be caused by left ventricular mural remodeling after myocardial infarction and may be responsible for changes in the left ventricular filling pattern observed after mountain hiking in these individuals.

The risk of an acute heart attack induced by severe arrhythmia or a myocardial infarction during a discrete episode of physical exertion is shown to be significantly increased in some studies.[257] However, other studies have shown that regular physical exercise is associated with a reduction in the long-term risk of coronary events, and also a decrease of the relative risk of a myocardial infarction triggered by an episode of heavy physical exertion.[258]

For people who have coronary artery disease or who have had a myocardial infarction, protocols for cardiac rehabilitation have been developed to enable gradual return to increased physical activities.[215] Before participation in a wilderness-based activity, adequate exercise tolerance and functional capacity should be documented.

Cardiac rehabilitation programs are usually divided into four phases.[197] The first phase takes place during the acute hospitalization. The second phase encompasses the first 3 months after hospitalization. It is during this time that scar tissue forms in the damaged myocardium. Usually, this rehabilitation is conducted in supervised outpatient medical facilities. During phase three, participation in an outpatient supervised community- or home-based program is encouraged. This is considered the training phase, during which aerobic conditioning is used to increase endurance. Phase four consists of a lifetime maintenance phase that is community based and usually unsupervised. A variety of activities are prescribed during each phase for individuals who have had a myocardial infarction. Goals include improving functional capacity and exercise tolerance. In addition, activities to increase cardiovascular efficiency and reduce atherogenic and thrombotic risk factors are used. These activities improve coronary blood flow and reduce myocardial ischemia.

Contraindications to exercise testing or training include unstable angina, severe uncontrolled hypertension, uncontrolled cardiac dysrhythmias, third-degree heart block, and severe aortic stenosis.[102] For these individuals, participation in a strenuous wilderness activity should be delayed until the condition is resolved or more stable.

In individuals with known coronary artery disease, a statement from a physician indicating that participation in a wilderness experience is not contraindicated may be helpful. To obtain this information, the individual may have to undergo exercise testing or training. Recommendations for activity level and tolerance can be generated. This could enable the individual and tour leaders to understand better the person's abilities, precautions, and functional limitations.

Asthma

Asthma is the most common respiratory disease causing impairment in the wilderness setting because it is so prevalent in younger populations with active lifestyles. Less common are individuals with emphysema or pulmonary fibrosis.[180] Asthma, whether mild, moderate, or severe, can affect the quality of life by preventing or limiting activity.[99] According to the Centers for Disease Control and Prevention (CDC), it affects approximately 9% of the U.S. population.[153]

The majority of people with asthma do not meet the current recommendations for physical activity.[77]

Although many studies exist about exercise and asthma, relatively little is known about exacerbation of asthma during

travel and wilderness sports and activities. In a study of risk factors and occurrence of acute exacerbation of asthma in adventure travelers,[85] the participants traveled to remote regions for a median duration of 13 weeks. The conclusion was that "asthma frequently worsens during travel and should not be ignored as a potentially life-threatening condition requiring pre-travel consideration." Two specific risk factors identified by the study were frequent use (at least three times per week) of inhaled bronchodilators before travel and intensive trekking during travel.

Eisner and Blanc[63] examined the prevalence of exposure to environmental tobacco smoke (ETS) and the short-term health impact during travel among adults with asthma. Thirty percent of the study group reported exposure to ETS during travel. The health impact of this exposure varied depending on presence or absence of regular ETS exposure before travel. Thirty-four percent of the exposed subjects indicated travel was the primary source for ETS exposure. Of this group, 66% reported cough, wheezing, or chest tightness, and 55% required extra inhaled asthma medication. There were no reported symptoms, increased asthma medication use, or exacerbation of asthma in persons with regular exposure to ETS.[63]

A literature search revealed one study comparing physiologic changes of nonasthmatic and asthmatic subjects. The study concluded that asthmatic subjects develop bronchoconstriction when they go to very high altitudes.[149] Information concerning the effects of altitude and wilderness environments on asthma is limited.

The National Heart, Lung, and Blood Institute, one of the U.S. National Institutes of Health, classifies asthma into one of four levels of severity: mild intermittent, mild persistent, moderate persistent, and severe persistent.[180] Mild intermittent asthma is defined as asthma causing symptoms (dyspnea that limits activity) less than two times per week, less than two nocturnal awakenings per month, and peak flow rate (PFR) variability of less than 20%.[29,58,187,229] The FEV_1 should be normal between exacerbations. Exacerbations are usually brief, from hours to a few days, although intensity may vary. Treatment is normally limited to a medium-term–acting β_2-adrenergic agonist bronchodilator, such as albuterol, as needed. If exercise is a trigger, then inhaled albuterol or nedocromil 30 minutes before exercise, and after exercise as needed, is usually sufficient.

Mild persistent asthma describes asthma symptoms more than 2 and less than 7 days per week, nocturnal awakenings (from asthma) two to six times monthly, and PFR variability of 20% to 30%. FEV_1 may be mildly abnormal but at least 80% of its predicted value. Exacerbations may affect activity. Treatment consists of at least one controller medication such as an inhaled corticosteroid, which is usually the first-line choice. Alternative treatment includes an oral antileukotriene, inhaled nedocromil/cromolyn, or possibly theophylline. Inhaled bronchodilators (inhaled β_2 agonist) should be administered as needed.

Moderate persistent asthma is characterized by symptoms nearly every day, nocturnal awakenings more than once per week, PFR variability greater than 30%, and FEV_1 greater than 60% and less than 80% of predicted value. Exacerbations may last days and affect activity. Treatment consists of multiple controller medications given in sufficient dosages that asthma symptoms require six or fewer puffs of medium-acting β_2 agonist per day.

Severe persistent asthma is diagnosed when daily symptoms are continual, nocturnal symptoms and exacerbations are frequent, and physical activity is significantly limited. FEV_1 is no greater than 60% predicted, and PFR variability is greater than 30%. These individuals are most likely unable to participate in wilderness activity because of the physical limitations imposed by the pathologic process.

Exercise can be the sole trigger or one of many triggers of asthma exacerbations. Exercise-induced asthma (EIA) affects both children and adults.[229] It is defined as "the clinical occurrence of shortness of breath, cough, or wheeze that occurs typically five to fifteen minutes after the cessation of the exercise."[143] Intensity and duration of exercise can precipitate EIA. The severity is related to the level of ventilation, to humidity of inspired air,[126] to heat and water loss from the respiratory tree, and to the rate of airway rewarming and rehydration after exercise.[169] It is estimated that 6% to 15% of the general population is affected.[143,169]

Moderate exercise does not predispose an individual to asthma. One study[131] found that the prevalence of physician-diagnosed asthma or exercise-induced bronchoconstriction (EIB) in moderately endurance-trained athletes (mean training time of 10 hours per week) was 4.2%. The study concluded that these athletes are not at higher risk of asthma or EIB than the general population.[131]

Symptoms of dyspnea during exercise are common, with 40% to 60% of asthmatic patients exhibiting EIA.[234] EIA symptoms include cough, shortness of breath, chest tightness, wheezing, and increased mucus production. Bronchospasm may appear during sustained exertion but more typically arises within 10 to 15 minutes of beginning exercise, peaks 8 to 15 minutes after conclusion of the exertion, and resolves about 60 minutes later.[169]

Diagnosis of EIA can be confirmed by history, physical examination, spirometry, serial peak flow rate measurements, and, occasionally, exercise testing.[229,234] The physical exam is frequently normal between attacks. Occasionally, when the diagnosis is suspected but no wheeze has been detected and airflow measurements (by spirometry or peak flow) have been normal, bronchial provocation testing is useful.[114] Bronchial provocation testing is a diagnostic technique designed to elicit bronchial reactivity (bronchospasm) as measured by a decline in serial FEV_1 measurements.

The presence of asthma does not imply the need for limited physical exertion due to exercise intolerance. Participation in exercise programs has been shown to enhance physical fitness and improve coping behavior of children with asthma.[37,249] A study of 164 children concluded the response to exercise cannot be consistently related to the clinical severity of asthma.[35]

Early diagnosis and proper preventive and maintenance therapy can reduce episodes of EIA and enable patients to continue to engage in wilderness activities and lead an active life.[234]

Treatment

The key to asthma management in the wilderness setting is prevention. Known intrinsic triggers and conditions associated with exacerbation of asthma, such as sinusitis and acid reflux, should be treated before embarking on a journey that takes an individual far from definitive care. Extrinsic asthmatic triggers should be avoided. The asthmatic participant should carry both prophylactic and therapeutic medications in sufficient quantities.

Box 90-6. Wilderness Medical Kit Options for Individuals with Asthma

- Controller medications
- Inhaled corticosteroids: fluticasone
- Long-acting inhaled β-adrenergic agonist: salmeterol (Serevent)
- Antileukotriene: montelukast or zafirlukast
- Oral corticosteroids: prednisone. Dosage is variable. Adults: 40 mg/d × 5–7 days *or* 40–60 mg/d tapered over 10–14 days.
- Anti-inflammatory cell agents (inhibit mast cell degeneration): Nedocromil: 2 to 3 puffs four times daily or 3 to 4 puffs three times daily; cromolyn: 2 to 3 puffs four times daily or 3 to 4 puffs three times daily
- Rescue inhaler: albuterol (Ventolin or Proventil)
- Antibiotics: levofloxacin, macrolide antibiotic
- Epinephrine: 0.3 mL of 1:1000 aqueous solution SC or IM every 20 min × 3 doses

An expedition participant with mild intermittent asthma should bring a corticosteroid for inhalation or even consider using this medication over the course of the trip. A wilderness adventurer with mild persistent asthma should consider bringing, or even initiating, a second controller medication, such as an oral antileukotriene or long-term β_2 agonist in addition to an inhaled corticosteroid.

Because respiratory infections can increase asthma symptoms, it is prudent to add a fluoroquinolone, such as levofloxacin, to the wilderness medical kit. This antibiotic can be used to treat pneumonia, chronic bronchitis, and sinus infection.

Increasing attention has been placed on pneumonias caused by *Chlamydia pneumoniae* or *Mycoplasma pneumoniae*.[29] *C. pneumoniae* can trigger acute episodes of wheezing.[29,68,93] *M. pneumoniae* was detected in 50% of lung biopsies from asthmatic patients and in less than 10% of control subjects.[133] Because both *C. pneumoniae* and *M. pneumoniae* can trigger local inflammatory reactions in the lungs, it can be rationalized that asthma may worsen in an individual with either infection.[29] Because both organisms are sensitive to macrolide antibiotics, it is wise to carry a macrolide antibiotic in the wilderness and during travel to remote environments.

Suggested medications to be included in the wilderness medicine kit for treatment of asthma are listed in Box 90-6.

Pretravel training should include physical activity comparable to the level of exertion anticipated for the wilderness trip. The individual should be evaluated to make certain the current treatment regimen is effective. If frequent use of an inhaler is necessary, further diagnostic evaluation should be done.

It is imperative that the person with asthma inform the medical advisor of any increased respiratory difficulties during the wilderness excursion. If an individual is using the primary "rescue" bronchodilator during wilderness activity more often than is his or her normal, it is time to reassess the individual's fitness to continue or consider an intervention.

Prevention

Short-acting medications, such as albuterol and pirbuterol, given in a timely manner before exertion can prevent an episode of EIA. Salmeterol and formoterol (Foradil), two long-acting bronchodilators, also provide excellent protection.[169] A 9-hour window of protection can be achieved from a single 50-μg dose of salmeterol. Cromolyn sodium and nedocromil sodium are effective in 70% to 87% of persons diagnosed with EIA.[169] Both have minimal side effects.

Nonpharmacologic measures can prevent an exacerbation or aid in reducing the severity of symptoms of EIA. If the activity is in a cold, dry environment, breathing through a mask can help warm inspired air and retain moisture from exhaled air. A gradual warm-up before aerobic activity can also aid in reduction of severity of EIA. Aerobic fitness and ventilatory capacity are improved through exercise rehabilitation.[16,98] Athletes and nonathletes with asthma have fewer symptoms after exercise conditioning. Physical conditioning also increases the level of intensity of exercise necessary to induce EIA.[143]

Chronic Obstructive Pulmonary Disease

Chronic obstructive pulmonary disease (COPD) refers to slowly progressive obstructive lung disease, usually associated with smoking, in which airflow obstruction is at least partly "fixed," that is, not completely reversible. When the term is used formally, other specific causes are excluded, such as cystic fibrosis (CF) or bronchiectasis. COPD affects approximately 15 million Americans and is the fourth leading cause of death in the United States.[180]

Current treatment modalities for COPD are seldom able to improve survival or halt decline in lung function.[241] Health-related quality of life (HRQOL) is often cited as an outcome measure in studies of COPD. Medical interventions that have shown a positive effect on HRQOL for persons with COPD include inhaled corticosteroids, inhaled bronchodilators, opioids, oxygen therapy, pulmonary rehabilitation, implementation of a disease-specific self-management program, and lung volume reduction surgery.[241] Even a modest change in lung function may be associated with clinically significant gain in health and well-being.[125]

Exercise tolerance can be improved through training programs, which also may improve the quality of life. A group of 50 normal-weight individuals with COPD participated in an inpatient standardized exercise training program that included cycle ergometry, treadmill walking, weight training, and gymnastics for 8 weeks. The results demonstrated improvement in exercise performance and muscle function.[81] This indicates that individuals with COPD can build muscle strength from a training program, which can augment physical fitness level.

Mental well-being and HRQOL for a person with COPD may be enhanced through travel to a remote environment or participating in a wilderness adventure. There are no specific studies in the literature precluding an individual with lung disease from traveling to hot, cold, humid, dry, or high-altitude environments. However, CF and other obstructive diseases may limit one's ability to participate in exercise or exertional activities because of reduced lung function. Although most individuals in good health have exercise limitation based on reaching "anaerobic threshold" (so-called cardiac limitation), patients with COPD usually have ventilatory limitation based on high minute volumes. This is a significant consideration in recreational planning, especially at altitude. Unlike asthma, COPD is less amenable to pharmacologic prophylaxis and less rapidly responsive to escalations in treatment intensity.

A scientific review of management of COPD supported the use of long-acting bronchodilators and inhaled corticosteroids

in reducing exacerbations.[219] It is crucial that COPD in affected participants is optimally managed before leaving on a wilderness expedition.

Cystic Fibrosis

Studies indicate that exercise and altitude are significant risk factors for adults with CF.[235] Moderate to high altitudes may be encountered during an expedition or airplane flight. People with CF suffer from hypoxemia under normobaric environmental conditions. The accepted critical value of the partial pressure of arterial oxygen (Pa_{O_2}) is 50 mm Hg.[204,235]

A decrease of aircraft cabin pressure during flight may be significant enough to compromise oxygenation. Respiratory function and blood gas variables during air travel with reduced environmental pressure have been measured. Arterial Pa_{O_2} in patients with CF with moderate airway obstruction was 58 mm Hg at 2500 m (8200 ft). Pa_{O_2} dropped to 46 mm Hg with atmospheric pressure equivalent to 3000 m (9840 ft).[235]

Another study of 10 patients with CF had similar results and included evaluation of subjective symptoms during simulated flight altitudes of 2000 and 3000 m. The Pa_{O_2} at 2000 meters (6560 ft) was 60 mm Hg, and at 3000 m (9840 ft) was 45.5 mm Hg.[204] No subject dropped below a Pa_{O_2} of 40 mm Hg. No patient developed dyspnea at any of the tested altitudes. The study concluded that "chronically adapted adult lung disease patients without accompanying heart disease and a Pa_{O_2} greater than 40 mm Hg can anticipate a safe flight trip."[204]

Individuals with CF desiring to travel above sea level could have pulmonary function testing (PFT) to calculate their anticipated Pa_{O_2} at their destination altitude. Anticipated Pa_{O_2} for a specific altitude can be estimated if FEV_1 and FVC are known. The formula is as follows[13]:

$$Pa_{O_2} \text{ at } 8000 \text{ ft} = [0.238 \times (Pa_{O_2} \text{ sea level})] + [20.098 \times (FEV_1/FVC)] + 22.26$$

If the result is Pa_{O_2} greater than 50 mm Hg, the accepted critical value, the individual should be able to tolerate the higher altitude at rest.

A Pa_{O_2} of at least 68 mm Hg at sea level predicts Pa_{O_2} of at least 55 mm Hg at 5000 ft (1520 m). A Pa_{O_2} of at least 72 mm Hg at sea level predicts Pa_{O_2} of at least 55 mm Hg at 8000 ft (2440 m).[54]

Exercise at altitude further compromises arterial oxyhemoglobin saturation. Evaluating 50 adult patients with CF with obstructive airway disease during exercise at an altitude of 1500 m (4920 ft), one study concluded that there is a significant risk for decreased arterial oxygen saturation.[207]

Barometric pressure decreases with increase in altitude. Even though oxygen concentration remains constant at 21%, the partial pressure of oxygen decreases. As a rule of thumb, barometric pressure (760 mm Hg at sea level) is reduced by half for each 6000 m (19,700 ft) gain in altitude. Partial pressure of inspired oxygen ($P_{I_{O_2}}$) can be estimated from the alveolar air equation[16]:

$$P_{I_{O_2}} = (\text{barometric pressure} - \text{partial pressure of water}) \times O_2 \text{ concentration}$$

$P_{I_{O_2}}$ at sea level is thus calculated as follows:

$$P_{I_{O_2}} = (720 - 47) \times 0.21 = 150 \text{ mm Hg}$$

$P_{I_{O_2}}$ at 6000 m (19,700 ft) is thus calculated as follows:

$$P_{I_{O_2}} = (380 - 47) \times 0.21 = 70 \text{ mm Hg}$$

Knowing $P_{I_{O_2}}$, the partial pressure of alveolar oxygen ($P_{A_{O_2}}$) can be calculated in anticipation of travel to high altitudes. The formula is[16]:

$$P_{A_{O_2}} = P_{I_{O_2}} - Pa_{CO_2}/0.8 \text{ [normal respiratory exchange ratio]}$$

At sea level and with a normal respiratory rate and Pa_{CO_2}, this is calculated as:

$$P_{A_{O_2}} = 150 - 40/0.8 = 100 \text{ mm Hg}$$

Calculating $P_{A_{O_2}}$ at 6000 m (19,700 ft) for an individual with a normal ventilatory rate and Pa_{CO_2} level can be accomplished as follows:

$$P_{A_{O_2}} = 70 - 40/0.8 = 20 \text{ mm Hg}$$

If minute ventilation is doubled, something normal climbers can easily do, then Pa_{CO_2} would decrease to 20 mm Hg, and $P_{A_{O_2}}$ becomes 45 mm Hg:

$$P_{A_{O_2}} = 70 - 20/0.8 = 45 \text{ mm Hg}$$

In the normal lung, alveolar oxygen saturation is slightly higher than arterial oxygen saturation. Assuming a normal alveolar–arterial (A-a) gradient, most subjects with healthy lungs, who are capable of doubling their minute ventilation for prolonged periods, have a Pa_{O_2} of about 38 mm Hg at 6000 m (19,700 ft) when breathing ambient air.[29] With acclimatization, this is potentially sustainable.

It is, however, difficult for individuals with hypoxic lung disease, such as patients with CF, to double minute ventilation, which is often already high at baseline. Furthermore, their A-a gradient is usually quite elevated above normal, which may reduce their Pa_{O_2} at altitude to unsustainable levels.

From these calculations, it is clear that high-altitude adventures in patients with respiratory diseases should probably be reserved for stable asthmatic patients without significant fixed airway obstruction. For the patient with COPD or CF, wilderness travel to moderate altitude should be feasible, provided that baseline PFT data are known, as per the preceding discussion. Wilderness travel to extreme altitude should generally be avoided in the patient with COPD or CF with significant disability.

Prevention

Commercial aircraft most commonly pressurize their cabins to the pressure equivalent of about 2000 to 2500 m altitude (about 6500 to 8000 ft). Although it is theoretically possible, this altitude does not normally result in a sufficient decrease in altitude pressure to cause rupture of a subpleural bleb, resulting in a pneumothorax. The overall incidence of pneumothorax is approximately 1% per year, increasing with age and severity of disease.[29] Approximately 20% of adults with CF experience at least one pneumothorax.[226] If needed, the airline can be queried to obtain the cabin pressure planned for a particular flight before the journey. If the flight altitude planned is on the higher

end of the spectrum and if the flight is of long duration, an oxygen supply and medical assistance might be required.

In circumstances where an individual will be flying at altitudes over 2500 to 3000 m (approximately 8000 to 10,000 ft) in an unpressurized aircraft such as a helicopter, an individual with CF may choose to appraise his or her risk for air travel before flight. An evaluation of response to altitude can be accomplished in an altitude or hypobaric chamber.

The individual with CF should be evaluated for exercise tolerance as part of trip planning. One study[25] evaluated the effects of an intensive summer camp on CF by measuring pulmonary function, exercise tolerance, and nutrition. The weekly program consisted of daily mountain climbing coupled with a high-calorie diet and chest physiotherapy. Arterial oxygen saturations during exercise were maintained at 80%. Results demonstrated improved exercise tolerance and nutrition in patients with CF. Although resting lung function was unchanged, the study concluded that exercise therapy for patients with CF is not detrimental for those with mild to moderate lung disease.[25]

In addition to sensitivity to altitude exposure, individuals with CF are quite prone to severe, life-threatening respiratory infections. Precautions should be taken to prevent unnecessary exposure to airborne infectious agents. In an enclosed space, such as an aircraft, it might be wise for the individual with CF to wear a medical face mask for protection from airborne pathogens. Children are taught in school to cough and sneeze into their elbows to decrease the risk of hand contamination and passing of germs. This method of protection is especially wise to implement during periods when hand-washing frequency is less than optimal. Empirical antibiotic choice might be a broad-spectrum drug with activity against *Pseudomonas*, such as levofloxacin.

Considerations for Wilderness Adventure for the Respiratory-Compromised Individual

Preparation for the trip should include optimizing medications.[75] If the journey includes exposure to high altitude, time needs to be allowed for adequate acclimatization. A person with respiratory complications should be physically trained to the degree that the activity or sport requires. Although the effects of physical training in the management of asthma are unclear, physical training does improve cardiopulmonary fitness without changing lung function.[200]

Travelers are at a greater risk of ETS exposure. Individuals exposed to ETS should carefully monitor their respiratory status and not venture further into rural or remote environments until their asthma management resides at baseline.

Approximately 9% of individuals with EIA are undiagnosed.[169] Although exercise itself does not precipitate bronchospasm, EIA should be considered if an individual participating in a strenuous activity develops shortness of breath, coughing, wheezing, or other respiratory symptoms.

When traveling to remote environments, the respiratory compromised individual, medical leader, and other identified team members must know how to recognize and treat emergent respiratory compromise. The individual with asthma should inform the medical advisor on the expedition about frequency of exacerbations and the treatment regime implemented for regaining control of the asthma. Medications for maintenance and management of an acute exacerbation should be included in the wilderness medical kit. Suggestions for such medications are listed in Box 90-6.

Seizure Disorders

A seizure is an abnormal electrical discharge of cortical neurons, resulting in involuntary change in level of consciousness, movement, perception, or sensation. A variety of factors can influence the onset of a seizure. Approximately 5% to 10% of the population will have at least one seizure during their lifetime, with the highest incidence occurring in early childhood and late adulthood.[34]

Epilepsy is defined as seizures that recur, usually because of a primary underlying abnormality in the brain. Epilepsy is a common neurologic disorder, with an incidence and prevalence of 50 and 650 per 100,000 population, respectively. Three fourths of individuals with epilepsy experience their first seizure before the age of 30 years.[137]

In the past, individuals with seizures were commonly counseled not to participate in athletic activities. There was a fear that exercise and increased activity would induce seizures. Evidence demonstrates that most persons with seizure conditions can participate in wilderness activities when given appropriate care from physicians. Medical guidance is needed to determine the appropriate doses of medication(s). Individuals with seizures should comply with medication routines that work to control factors that precipitate seizures. Causes predisposing to seizure activity include excessive fatigue, emotional stress, physical stress, strobe lights, stimulants, alcohol, and recreational drugs.[185] Other precipitating factors can include hypoxia caused by exposure to high altitude, hyponatremia associated with electrolyte loss, hypernatremia associated with dehydration, hyperthermia related to physical exhaustion and heat, and hypoglycemia associated with poor nutrition before an activity.[220]

Activities that involve the danger of falling are contraindicated for individuals with epilepsy. These include rope climbing, rock climbing, skydiving, and high diving. Individuals who experience seizures during physical activity likely do not have optimal medical control of seizure activity in general.[89] In fact, it has been shown that exercise may perhaps raise the seizure threshold and be protective against seizures.[179]

Complications

Individuals with epilepsy may have a lower physical work capacity. This is thought to be related to the fact that there may be a lower activity level at baseline. Exercise capacity appears to be similar overall to that in the general population.

Hyperventilation can provoke seizures in the laboratory. As a result, many researchers believe that the increased ventilation during strenuous activities will do the same. During intense physical activity, however, homeostatic mechanisms generally avoid hypocapnia. However, this does not reduce the sensitivity to respiratory alkalosis needed to precipitate seizures. During exposure to high altitude, hypoxia stimulates peripheral chemoreceptors and causes hyperventilation. This may produce respiratory alkalosis and could lead to seizures. It has been recommended, therefore, that people with epilepsy avoid exposure to rapid decreases in barometric pressure. The most common provoking factors for seizures are excessive mental labor, mental strain, and physical fatigue.[22]

The National Society for Epilepsy recommends that individuals with epilepsy make informed decisions about the types of

activities in which they choose to participate. Epilepsy is a very individual condition, so choices about whether to participate depend on the type and frequency of seizures, and the level of control with medications. Many activities can be made safer by adopting simple safety measures to help minimize potential risks.

Treatment

If a person has a seizure in the field, the most important task is to protect him or her from injury. This usually involves helping the individual to the ground, and clearing away any objects that may cause injury. It is not appropriate to place any object into an individual's mouth. Seizures are usually self-limited. If a seizure is prolonged, Diastat (diazepam rectal gel) should be given. Most epileptic patients are aware of the appropriate Diastat dose that is needed in such a situation and should communicate the information to the leader or medical advisor before the trip.

IMMUNE-MEDIATED DISEASES

Rheumatic Diseases

The five most common rheumatic diseases are osteoarthritis, rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), ankylosing spondylitis, and gout.

Joint pain, tenderness, and swelling are the most common symptoms of these inflammatory diseases. Additional joint manifestations include osteophyte formation, cartilage destruction, loss of joint integrity, and joint fusion. Fatigue is also a frequent symptom of rheumatic diseases.

Maintaining a healthy lifestyle with appropriate diet and exercise can contribute to improved well-being. Exercise has a similar effect on the various systemic forms of arthritis.[171] Although vigorous exercise is contraindicated during an acute exacerbation, the literature consistently indicates that intensive exercise is beneficial and does not negatively affect the rheumatic disease process.[171] It can reduce loss of flexibility, muscle atrophy, weakness, depression, and fatigue, all of which are problems common to both inflammatory and degenerative conditions. One study also concluded that long-term high-intensity muscle strengthening and aerobic fitness programs are effective in slowing the loss of bone mineral density at the hip.[51] Spending time in the wilderness and acquiring the endurance necessary for strenuous activities may enhance the well-being of an individual diagnosed with a rheumatic disease.

Rheumatoid Arthritis

Rheumatoid arthritis affects about 1% of the U.S. population and is three times more common in women than in men.[223] It is associated with increased morbidity and mortality, with cardiovascular disease being the most common cause of premature death.[87] The hallmark of RA is inflammation and pain in multiple joints, usually in a symmetric pattern. Stiffness of the joints, especially in the morning or after long periods of inactivity, is another feature of RA. Regular physical activity can preserve and improve joint function. Exercise is useful for patients with RA to reduce inflammation and pain.[127]

Aerobic capacity and neuromuscular performance are necessary for endurance activities, often required for wilderness adventure. Several studies have evaluated aerobic fitness, strength training, and physical function on the long-term impact of the disease process of RA. One study[95] concluded that physically active women with early or long-term RA can maintain their aerobic fitness to levels comparable with those of age-matched healthy subjects. The study also concluded, however, that explosive strength of the leg extensors cannot be maintained at a normal level with routine physical activities. This would potentially affect activities requiring a sudden burst of energy, such as sprinting.

Muscle weakness is a common progressive development in RA. Conservative and intensive exercise programs have been compared to determine which type of training provides the best outcome. Muscle strength is more effectively maintained or increased by a moderate- or high-intensity strength training program than a low-intensity training regimen and is well tolerated and without pain or other harmful effects on the disease process.[52,94,96,166,246] Regular dynamic strength training in combination with endurance physical activities improve muscle strength and physical function without detrimental effects on RA.[97] Endurance and strength training can safely be pursued in preparation for wilderness adventure without deleterious effect on disease activity.

There is a psychological benefit to remaining active, even during painful exacerbations of the disease process. Decreasing activity as a behavioral method to cope with pain has a negative impact on both disease course and subsequent psychological distress.[248]

Wilderness activities are safe for individuals with RA and offer an opportunity to maintain aerobic conditioning, endurance, physical fitness, and mood.

Complications. An acute flare-up of the disease can affect the ability to exercise. Energy expenditure during normal physical activity can increase as much as 50% because of pain, stiffness, biomechanical inefficiency, and abnormal gait.[171] Repetitive movements may be difficult to perform and range of motion may be limited during an exacerbation. This could affect performance in certain types of sports such as paddling, bicycling, hiking, or rock climbing.

Pulmonary involvement is the most frequent extra-articular manifestation of RA.[156] Other pulmonary complications include clinically significant interstitial lung disease, occurring in 5% to 10% of patients with RA, and bronchiectasis in 10%. Respiratory infections account for 15% to 20% of deaths.[156] Before venturing into the wilderness for an extended period of time, the individual should be evaluated for presence of respiratory complications and for tolerance to exposure to a high-altitude environment, should that be part of the trip itinerary. Respiratory infections should be assessed and treated before travel.

People with RA are at greater risk for developing osteoporosis and osteoporotic fractures than are their healthy peers. Pathologic changes in the cervical spine are common,[6] and instability of the atlantoaxial joint is a frequent complication.[12] The individual should be cognizant of neurologic deficits, including numbness, tingling, or weakness of the extremities. Cervical spine subluxation can be a life-threatening injury.

Joint inflammation, stiffness, and pain can cause alterations in gait. This can lead to instability on uneven terrain or when participating in wilderness sports. Hip fractures are common in women with RA.[129] Examination of visual acuity, ability to perform stand-ups, and heel-toe walking is helpful in predicting risk of falls.

Major ocular manifestations in RA include keratoconjunctivitis sicca, scleritis, and keratitis. Corneal ulcers are a rare

Box 90-7. Wilderness Medical Kit Options for Individuals with Rheumatic Diseases

- Topical pain relievers
- Narcotic pain relievers, oral and parenteral
- Nonsteroidal anti-inflammatory drugs
- Oral corticosteroid (prednisone) up to 10 times the normal dosage
- Corticosteroid, parenteral
- Diphenhydramine (Benadryl) or hydroxyzine (Atarax, Vistaril)
- Disease-modifying antirheumatic drugs
- Oral and injectable gold therapy
- Immunosuppressive agents
- Antibiotic for respiratory infection
- Epinephrine 1:1000 aqueous for intramuscular injection
- Assortment of 3-cc, 10-cc, and tuberculin syringes with needles
- Extra supply of all medications currently being used
- RA medications, which may include:
  - Remicade (given IV every 2 mo)
  - Humira (given SC every 2 wk)
  - Etanercept (given SC twice weekly)

complication but can lead to perforation because of an unstable epithelial barrier.[110,232] A literature search revealed no information on the effects of ultraviolet (UV) light on these complications. It would be prudent to suggest that effective UV eye protection in the outdoors is imperative.

A rheumatoid flare can occur, sometimes without much warning, and can often be debilitating. Severe inflammation causes marked increases in pain, swelling, and tenderness in one or more joints, leading to serious incapacity. A mobility-impairing flare could be devastating in the wilderness. Treatment for such an exacerbation is a bolus of high-dose prednisone, up to 10 times the usual amount.[139]

Treatment. Before wilderness travel, an individual should consult with his or her personal physician for guidelines for prednisone treatment should a flare-up occur. Any person on long-term steroid therapy needs to have an adequate supply of prednisone available during travel.

In consideration of RA, certain medications should be added to the standard wilderness medical kit. Suggestions for consideration are listed in Box 90-7. Medications must be stored at proper temperatures in the wilderness to ensure effectiveness of the drugs. Some medications require cold storage. One such drug used by patients with RA is etanercept (Enbrel). Etanercept is a protein and requires special handling. The dose tray for etanercept must be kept at a temperature of 2°C to 8°C (36°F to 46°F), which is the standard temperature in most refrigerators. The medication should never be frozen.

When traveling, the dose tray for etanercept can be carried in a small, insulated cooler bag with a frozen ice pack for a short period of time. It can be difficult or even impossible to keep the medication at appropriate temperatures when traveling for extended periods of time in the wilderness in warm climes. It is much easier to keep the medication at the necessary temperature in cold environs. A temperature gauge is invaluable.

Prevention. A regular, high-intensity muscle strengthening and aerobic exercise program has been found to aid in maximizing the health and well-being of an individual with RA. The individual must adequately condition the muscles before vigorous activity through flexibility, joint range of motion, and strength training. Overstretching or highly repetitive activity requiring use of unstable joints should not be performed. Activities requiring medial or lateral forces on joints should be avoided.

Appropriate footwear is essential during prolonged weight-bearing activities, such as hiking or mountain climbing. Insoles that attenuate shock should be inserted into the sport-specific shoe or boot. Overpronation should be corrected with orthotics when possible.

Systemic Lupus Erythematosus

Systemic lupus erythematosus, or "lupus," is a rheumatic disease whose pathogenesis includes development of autoantibodies that bind antigens such as DNA to form immune complexes. These immune complexes are deposited in various organ systems, including the joints, skin, kidneys, and brain, where they cause inflammation. Like patients with RA, persons with SLE should maintain optimal fitness.

A complication in SLE, not seen in RA, is photosensitivity. Patients with lupus may develop a rash on sun-exposed areas of their bodies. The rash is sometimes severe and can be associated with a generalized lupus flare as well. A person with lupus should protect his or her skin from sun exposure. Preparation for a wilderness experience must include not only sunscreen but protective clothing. Long-sleeved shirts and long pants are necessary for sun-sensitive patients with lupus. Regular clothing allows approximately 1/30 of UV rays to reach the skin. However, specially manufactured clothing now allows only 1/50 of UV rays to penetrate, significantly reducing UV exposure.

Anaphylaxis

Idiopathic anaphylaxis is a systemic syndrome of immediate hypersensitivity that presents clinically with bronchospasm, angioedema, hives, and, in severe cases, cardiovascular collapse. This disease process has no definable etiology or identifiable exogenous allergens.[55] As last reported in 1995, there was an estimated prevalence of 20,000 to 50,000 cases in the United States.[144]

Patients with idiopathic anaphylaxis exhibit the same signs and symptoms as patients with other forms of anaphylaxis, including hypotension, tachycardia, wheezing, stridor, hives, angioedema, pruritus, flushing, nausea, vomiting, diarrhea, dysphagia, lightheadedness, and loss of consciousness.[144] Loss of peripheral vascular tone leads to severe hypotension. Cardiovascular collapse can occur without the classic early signs of wheezing, hives, or angioedema.[250] Progression from the dermatologic manifestations of idiopathic anaphylaxis to life-threatening symptoms affecting the cardiopulmonary system may occur from 10 minutes to hours after onset.[144]

The etiology is unknown. Diagnosis is made by a process of exclusion. The most important information for the wilderness adventurer is awareness of the events surrounding onset of each episode. Pertinent information for the medical team includes the frequency of attacks and response to specific treatment.

It is unlikely that an unforeseen external allergen will be responsible for idiopathic anaphylactic symptoms.[144] Idiopathic anaphylaxis can be managed successfully with published algo-

rithms.[144] Most patients achieve remission with pharmacotherapy.[55] Treatment depends on the frequency and severity of exacerbations and may include prednisone, hydroxyzine, and albuterol.[55] Ketotifen has steroid-sparing effects and has been found to induce remission in persons previously corticosteroid-dependent for control of idiopathic anaphylaxis.[195]

Anaphylaxis can occur in response to specific allergens. Food allergy is the most common cause of anaphylaxis, occurring in approximately 1% to 2% of the population.[208] The onset of symptoms is usually 5 to 30 minutes after ingestion and can occur as long as 1 to 2 hours later, rarely longer. Food allergy can be of significance for the wilderness traveler predisposed to an anaphylactic reaction. It is often necessary to procure food from unfamiliar sources, and exact contents of these food items may not be known. Likewise, a food might be verbally reported to be free of a particular food substance and actually have trace amounts present, enough to cause a severe reaction. Not all countries require identification of entire contents and strict food labeling. Language may be a barrier in food product identification, especially with processed or prepackaged foods. Shellfish, fish, peanuts, tree nuts, milk, egg, soy, and wheat comprise 90% of food allergens.

Exercise can induce anaphylaxis, either in isolation or in conjunction with food consumption before exercise.[192] This alone does not preclude one from participating in a wilderness activity. It is imperative to know specific details surrounding an episode of exercise-induced anaphylaxis. The wilderness adventure can be planned with the activities scheduled within tolerable limits of the individual.

Treatment

In the wilderness environment, an anaphylactic reaction becomes a medical emergency regardless of its etiology. Treatment should be initiated immediately with onset of symptoms. Adult patients should receive 0.3 mL of 1:1000 aqueous epinephrine intramuscularly or subcutaneously, followed by oral doses of both an antihistamine (diphenhydramine 50 mg or hydroxyzine 25 mg) and 60 mg prednisone.[144] An individual with predisposition to severe anaphylactic reactions may want to treat prophylactically with an antihistamine to block the histamine response in case of inadvertent exposure.

If a team member skilled in advanced airway management is present on the expedition, carrying equipment for advanced airway management should be considered.

Hereditary Angioedema

Hereditary angioedema (HAE), although similar to idiopathic anaphylaxis, presents gradually, painfully, and without the typical anaphylactic dermatologic manifestations.[144] It is caused by a deficiency of the plasma protein C1 inhibitor (C1-INH).[27,28,86,265] The term *angioedema* describes localized, transient, and episodic edema of the deeper layers of the skin and intestinal mucosa.[27] It is characterized clinically by abdominal pain, skin swelling, and life-threatening upper airway obstruction. Abdominal pain is caused by extravasation of plasma and subsequent edema in the affected areas.[82] Episodes may be provoked by emotional stress or infection,[70] trauma (particularly associated with local pressure),[144] dental procedures,[116] or exercise.[65,157]

Hereditary angioedema has been estimated to affect between 1 in 10,000[86] and 1 in 150,000 persons.[43,65,116] Urticaria and angioedema affect nearly 20% of the population, whereas true HAE occurs in approximately 2% of the population in the United States.[65] Laryngeal edema can cause an immediate life-threatening emergency and is the major source of HAE-related mortality.

Figure 90-16. Hereditary angioedema, lateral view. (Photograph courtesy of Sheryl Olson.)

Clinical Presentation

The skin and gastrointestinal tract are more commonly involved than the airway. There is a ratio of laryngeal edema to skin swellings to abdominal pain attacks of approximately 1:70:54 in persons with laryngeal edema. Eighty percent of cases of laryngeal edema occur between the ages of 11 and 45 years. The mean time from onset to maximum development of laryngeal edema is 8.3 hours, and the mean age of first event is 26 years. The clinical signs most frequently described are dysphagia, a sensation of a lump or feeling of tightness in the throat, voice changes, including hoarseness and roughness, and dyspnea.[27,28]

Cutaneous edema is the most common symptom and can occur anywhere on the body, most frequently on the upper extremities and around the oropharynx.[65,70] It usually develops over several hours, progresses for up to 36 hours, and resolves over 1 to 3 days.

Intestinal wall and mesenteric edema cause abdominal pain, nausea, and vomiting.[218] It can cause intense pain mimicking peptic ulcer disease, biliary colic, appendicitis, or a perforated viscus.[65,116]

Edema of the upper airway can create a medical emergency (Figs. 90-16 and 90-17). The individual with airway involvement may first experience a full or tight sensation in the throat, dysphagia, or voice alteration.[70] Soft tissue edema can progress to stridor with complete airway obstruction in as little as 4 hours.[65]

Treatment

Long-term management of HAE consists of therapeutic prophylaxis to minimize frequency and severity of attacks. Individuals who experience frequent symptoms may require daily suppressive therapy with the androgen steroids danazol (Danocrine) or stanozolol (Winstrol).[144] Two other androgen steroids, oxymetholone (Anadrol-50) and oxandrolone (Oxandrin), are being studied. Limited data are available to suggest appropriate dosing, as noted in Table 90-9.

Figure 90-17. Hereditary angioedema, frontal view. (From Wüthrich B, Devay J, Späth P: Hereditary or acquired angioedema caused by functional deficiency of C1 inhibitor—a still unfamiliar disease picture. Schweiz Med Wochenschr 129:285–291, 1999.)

Acute management goals include decreasing the length and severity of signs and symptoms after onset. Narcotics may be required to relieve symptomatic pain. If edema is severe, fluid has been lost from the vascular compartment and fluid replacement should be considered. In the wilderness, intravenous fluid replacement may not be available. If there is no airway compromise or dysphagia, the individual can be encouraged to increase fluid intake until symptoms resolve.

Anticoagulant therapy can prevent recurrent or ongoing thromboembolic occlusion of the circulation.[65] Aerosolized heparin 30,000 units can be administered by nebulizer during an acute attack.[70]

Laryngeal edema is the most important component of this disease. The airway must be assessed for signs of laryngeal edema during an acute attack. If present, it can become a life-threatening medical emergency requiring immediate intervention. At first recognition of airway involvement, C1-INH concentrate, if available, should be given promptly IV at a dose of 1000 to 1500 IU.[70] Although not yet approved for use in the United States, it is available throughout Europe and has been used in Australia[142] and Canada.[70,252] It is anticipated that C1-INH protein will be available for treatment of acute attacks within the next several years.

Although angioedema attacks respond poorly to epinephrine, it is often used for treatment.[86] Racemic epinephrine has been used to decrease upper airway edema during an acute attack. In surveying individuals who have used it, it has been found to be effective in less than 27%. It is best if used early in an attack and can be administered as an oropharyngeal spray of 0.2 to 0.3 mL of a 1:1000 dilution.[116]

If airway edema becomes severe and compromises ventilation, advanced airway management is imperative. The trachea should be intubated before laryngeal edema is so severe it precludes successful endotracheal intubation. If such is the case, an emergent cricothyrotomy becomes the procedure of choice for airway management.

An algorithm for treatment of HAE was developed in October 2003 through consensus of European and North American investigators, patient care providers, patient group representatives, and individual patients.[32] Fresh frozen plasma is a first-line intervention in a hospital but is clearly not available in the wilderness setting. Wilderness team members must be aware that the first episode of laryngeal edema may be fatal.[28]

Even though an individual with known HAE may have had no prior episodes of airway obstruction, it is advisable to have supplies available for advanced airway management, providing someone on the wilderness expedition is knowledgeable and skilled in their use. A sedative should be available should intubation be required. Medications for rapid sequential intubation (RSI) should be considered if advanced airway management equipment is going to be available in the wilderness. RSI provides opportunity for earlier intubation if the person is conscious and alert and in severe respiratory distress.

Medications used for treating HAE and other considerations for the wilderness medical kit are listed in Table 90-9.

Prevention

Known causative stimuli should be eliminated when possible. In the wilderness, it is especially important to prevent pressure points, which may trigger an acute attack. Every sport has its unique piece of equipment that can cause pressure points on some part of the body. For example, when hiking or skiing, attention must be paid to the feet. Trail shoes, hiking boots, and ski boots should fit well and be well broken in before use during an outing. Ski boots often have liners in which tongue flaps and other segments of the boot are stitched together. These stitched areas tend to create potential pressure points. One-piece liners that are heat-molded to the foot and ankle are available and eliminate pressure points on the foot. Socks should be free of wrinkles.

Backpacks should fit well. Waist belts should be wide and well padded to distribute weight evenly, preventing pressure points on the iliac crest. Shoulder straps should be padded. The shoulder strap fit should not exert pressure on the axilla. A chest strap connecting the shoulder straps at the level of the sternum can aid in decreasing pressure of the lateral aspects of the shoulder strap on the axilla. During training for the expedition, the backpack should be worn and weighted gradually, allowing improved tolerance to increased weight and length of time it can be worn.

River sports require the use of helmets and paddles. Attention must be given to pressure points on the head and the chin or mandible. The hands can be protected from blisters by wearing gloves when paddling.

Because vigorous activities can precipitate HAE attacks, Elnicki[65] suggests that a short course of anabolic steroids before the planned event may lessen the frequency and severity of attacks.

Raynaud's Phenomenon

Raynaud's phenomenon (RP) is a syndrome described in 1862 by Maurice Raynaud that can be induced by exposure to

TABLE 90-9. Medications and Supplies Used in Treatment of Hereditary Angioedema

	DOSAGE
Medication	
Danazol (Danocrine)	Adult initial dose 150–200 mg PO qd, gradually reduce to a minimum effective maintenance dose, 50–100 mg qod.[70,116] Daily dose required, may be as high as 200–400 mg/day.[79,80]
Stanozolol (Winstrol) (This drug is no longer available in the United States)	Adult initial dose 6 mg PO qd, gradually reduce to maintenance dose 2 mg PO qd to qod.[70,116]
Oxymetholone (Anadrol-50)	Adult dose not yet established. Limited data suggest dose of 0.5–1 mg/kg/day PO.[116]
Oxandrolone (Oxandrin)	Adult dose not yet established. Limited data suggest dose of 2.5 mg PO tid, adjusting for response.[116]
Heparin	30,000 IU, aerosolized in a nebulizer for acute attacks.[70]
Aminocaproic acid (Amicar; antifibrinolytic agent)	Adult dose 4–5 g IV over 1 hr initially, followed by 1 g/hr IV for 8 hr. Solution should be diluted to a concentration of 1 g/50 mL.[116]
Tranexamic acid (Cyklokapron; immediate short-term treatment; helps relieve intestinal symptoms and prevent edema formation)	2 g/day IV until symptoms resolve.[116]
C1-INH concentrate (available in Europe, not available in the United States)	Used in acute treatment; adult dose 500–1500 IU IV, dose can be increased significantly depending on severity of attack.[70,116]
Epinephrine (adrenalin; racemic epinephrine 1:1000 oropharyngeal spray to reduce edema of the upper airway[116])	0.2–0.3 mL q 20 min for a total of 3 doses.
Supplies	
Advance airway supplies, including:	
Laryngoscope	
Endotracheal tube, appropriate size for the individual with hereditary angioedema	
Stylette	
10-cc syringes with 18-gauge needles	
Umbilical tape or other commercial tube security device	
Midazolam (Versed) or other benzodiazepine for sedation and amnesia	
Bag, valve, mask (optional)	
Succinylcholine (Anectine)—optional; storage without refrigeration is limited to 4 wk	
Vecuronium (Norcuron) or other paralytic agent—optional; does not require refrigeration	
Sterile saline or water for reconstitution of paralytic agent	

cold temperatures or by emotional stress. Small blood vessels in the fingers and toes become overly constricted and severely blanched, often without capillary refill. The phenomenon is caused by constriction of digital arteries, precapillary arterioles, and cutaneous arteriovenous shunts, perhaps influenced by abnormal sympathetic nerve activity or an immunologic process. Women constitute the majority of RP sufferers. RP may be primary (idiopathic) or associated with some underlying disease process, including connective tissue disorders, arterial disease secondary to atherosclerosis, angitis obliterans, and secondary effects of medications (e.g., β-adrenergic blockers, oral contraceptives).

Clinical criteria for RP include skin color change in response to cold or stress. The classic triad presents as digital or distal hand pallor with numbness, which persists for the duration of the cold stimulus (Fig. 90-18). Cyanosis develops after 10 to 30 minutes of rewarming as the small amount of flowing blood saturates the tissue. Last, reactive hyperemia and rubor of the skin develop. Severe pain is rarely associated with the process. Prolonged cold exposure or severe vasoconstriction can cause ischemia of the tissues, leading to infarction of a finger or toe, which may result in permanent tissue damage.

Figure 90-18. Raynaud's phenomenon. Note blanched fingertips with hyperemia of left little finger. (Photograph courtesy of Sheryl Olson.)

Treatment

Treatment depends on severity of the vascular problem. It can consist of nonpharmacologic measures such as biofeedback, medicinal therapy, or surgery.

The most widely used agents for treatment of RP are calcium channel blockers.[256] A study[1] compared the effectiveness of sustained-release nifedipine and temperature biofeedback for the treatment of RP. It concluded that treatment of RP with sustained-release nifedipine is safe and effective and is superior to temperature biofeedback.

The use of low-level laser irradiation (LLLI) has been studied for treatment of RP. Infrared thermography was used to assess the response to a cold challenge test before and after LLLI or the placebo treatment. The results demonstrated a statistically significant reduction of the frequency and severity of RP in the LLLI group at 6 weeks and 3 months.[5] LLLI may be a viable alternative to existing therapeutic regimens for treatment of RP.

The clinical efficacy of standardized *Ginkgo biloba* extract (Seredrin) in the treatment of RP in persons with no associated condition, such as systemic sclerosis, has been studied.[176] *G. biloba* extract may be effective in reducing the number of RP attacks per week.

In severe cases, individuals unresponsive to medical treatment may undergo microsurgery for a localized digital sympathectomy[256] or cervical sympathectomy.[243]

Prevention

Individuals with RP are often more comfortable recreating in warmer, as opposed to colder, wilderness environments. However, a person with RP does not need to be restricted to warm climates. Adequate planning and preparedness can enable someone with this phenomenon to enjoy cold environment adventures. Each individual should be aware of the degree to which cold affects this phenomenon. If it is unknown, a trial outing should be planned before the expedition. Assessment should be made for the severity of the vasoconstriction, how long the constriction lasts, what stimulates vasodilation, and if the digits can be rewarmed in the wilderness without prolonged ischemia.

Because of the peripheral vasoconstrictive process, the ability to remain warm in the wilderness is a priority. Gradual exposure to the cold may decrease the severity of sudden vasoconstriction. At the first sign of onset, the hands or feet should be placed in a warm area. The hands can be placed next to the skin on the torso, abdomen, or thighs if possible. Mittens are preferred to gloves for warmth and may be needed even when it is not extremely cold. When the fingers begin to grow cold, they should be curled into the palm to minimize exposure. Hand warmers inside the mittens may be necessary.

Socks should be made of wool or other warm insulating fiber. A wicking liner should be worn to help keep the feet dry. Prewarming mittens and boots before donning can help minimize cold exposure to the digits when first exposed. Exercising to generate body heat increases peripheral circulation.

Any prophylactic medication should be started well before the planned wilderness expedition to evaluate its effectiveness and side effects. Tachycardia, headache, dizziness, orthostatic hypotension, edema, and flushing are all side effects of calcium channel blockers[256] and may interfere with an individual's ability to meet the physical demands of the planned adventure.

SENSORY IMPAIRMENT

Vision

World perspective on the physical abilities of a blind person changed dramatically when Erik Weihenmayer climbed to the summit of Mt. Everest. His lack of vision did not rob him of passion or his desire to seek a physical challenge.

Blindness and low vision are among the most common disabilities.[46] At the time of the last accurate assessment of blindness and low vision in the world, an estimated 40 million people were blind and 110 million people had low vision.[239] Blindness is defined in the United States as best vision of no greater than 20/200 in the better eye, and less than 20/400 by the World Health Organization (WHO). Low vision is defined in the United States as less than 20/40, and less than 20/60 by the WHO. Causes of visual impairment include refractive error, increasing age, infectious diseases, nutritional deficiencies, metabolic conditions, and trauma.[46]

There is a paucity of literature on visual impairment relating to wilderness activity and travel[251] (see Chapter 25). Many recent studies discuss the effect of visual impairment on quality of life and indicate the need for additional research to gather information to understand the full spectrum of this disability.[42,49,101,206] Although visual function declines with age,[119] loss of visual acuity is only one dimension of the disability. Contrast and glare sensitivity, impaired stereoacuity, and diminishing visual fields are significant independent factors that can affect the quality of everyday life.[206] Eye diseases that can contribute to visual disability include cataracts, maculopathy, glaucoma, diabetic retinopathy, and retinal vein occlusion. Whether from ocular pathology or loss of acuity, impaired vision is generally associated with significantly lower levels of physical and social functioning and lower quality of life.[244] Interventions aimed at improving quality of life for visually impaired individuals may include strategies for vision-related rehabilitation.[140]

Cataracts

Disability for the person with cataracts goes beyond primary loss of visual acuity. Some types of artificial intraocular lens can increase glare, which can further impair vision. A hydrophilic acrylic intraocular lens had superior performance in reducing glare disability.[4] The wilderness adventurer may choose to travel during daylight hours to minimize the effect of glare, especially if climbing or using technical skills.

Glaucoma

Exercise has been found to cause acute visual loss, with complete or near-complete recovery after cessation of activity, in a small number of patients with advanced glaucoma.[214] It is hypothesized that the mechanism underlying the phenomenon is possibly due to a "vascular steal."[214] This information provides guidance for evaluation of the impact that strenuous wilderness activity may have on the eye. Diminished blood flow to the eye can cause a change in vision. The individual preparing to train for the activity should be questioned as to any previous experience with deterioration of vision during physical activity. The person with glaucoma should be aware of potential change in vision and limit activity should this occur.

Altitude

Changes in altitude may affect vision. Boes and colleagues[26] concluded that climbers who have had LASIK (laser-assisted in

situ keratomileusis), especially during the early postoperative period, should anticipate and be prepared for visual acuity fluctuations at high altitudes. Another study concluded that an increase in altitude from sea level to 10,000 ft (3050 m) has not been found to reduce intraocular pressure significantly in healthy subjects.[19]

Cane Use

Body posture and movements of individuals using canes during ambulation vary from the normal movement of walking. Repetitive movements of the wrist or forearm accomplish cane manipulation, while the remainder of the upper body maintains sustained postures.[175] More than half of long cane users lack the normal intersegmental movement that typically occurs in the neck, trunk, and shoulder girdle during walking.[175] Cane use on uneven terrain may be difficult.

The performances of expert gymnasts with normal vision, with impaired vision, and without vision have been compared. There were similar scores in normal and reduced visual conditions, with lowest scores in the no-vision performance.[113] The gymnasts with normal visual acuity were still able to perform a back aerial double somersault with their vision blocked and without prior practice. Individuals living with visual impairment also have the opportunity for high-level performance. With training opportunities, practice, equipment adaptations, and a support group, visually impaired people can experience even extreme wilderness adventures.

Hearing Impairment

The term *hearing loss* applies to people who are hard of hearing or deaf. In the wilderness, safety is of utmost concern. Communication between members of the group is essential. For people who have a hearing loss, there are challenges related to setting up a system of communication that will be effective and safe. Someone with hearing loss may use speech, lip reading, hearing aids, or amplification systems to enhance oral communication.

Hard of hearing (hearing loss of 35 to 69 decibels hearing level [dBHL]) is a condition that makes understanding speech difficult with use of the ears alone, with or without a hearing aid.

Deafness (a hearing loss at or above 70 dBHL) describes a condition in which a person is unable to understand speech through use of the ears alone, with or without a hearing aid. There are two types of hearing loss: sensorineural hearing loss and conductive hearing loss. Sensorineural hearing loss results from damage to the inner ear or auditory nerve. Conductive hearing loss results from abnormalities of the outer ear and middle ear.

With sensorineural hearing loss, the cochlea of the inner ear is affected. In this case, a person's balance may likewise be decreased because the vestibular apparatus is also located in the inner ear. Otherwise, hearing loss does not generally cause any changes in tolerance for activities in the wilderness area. If a person has decreased balance, however, he or she may have difficulty with spatial orientation and depth perception.

Assistive devices that amplify sound are often used by individuals with hearing loss. There are several different types of hearing aids, including chest- or body-worn aids, behind-the-ear aids, and in-the-ear aids. Many hearing aids work on similar basic principles. They have a built-in microphone that picks up sound that is processed electronically, either by analog circuits or digitally. The resulting signals are then passed to a receiver in the hearing aid, where they are converted back into sounds. A hearing aid also has an earmold, tubing, and batteries. A hearing aid does not create perfect hearing. It makes sounds louder, but not necessarily clearer. If an individual wears a hearing aid, it is important to pack extra batteries and tubing for wilderness or rural travel. The following are descriptions of various types of hearing aids[201]:

Behind-the-ear (BTE) hearing aids have an earmold that sits inside the ear. The hearing aid rests behind the ear and a plastic tube connects it to the earmold. These are the most common types of hearing aids.

In-the-ear (ITE) and *in-the-canal* (ITC) aids have their working parts in the earmold so the whole aid fits into the ear. The smallest ITC aids fit entirely inside the ear canal, where they cannot be seen.

Body-worn hearing aids have a small box that is clipped to clothes or put in a pocket. This is connected by a lead to an earphone and earmold. Body-worn hearing aids are the most powerful hearing aids, and are more suitable for people with sight problems or problems using their hands.

Bone conduction hearing aids are for people with conductive hearing loss or people who cannot wear a conventional hearing aid. They deliver sound through the skull by vibrations.

CROS/BiCROS hearing aids are for people with hearing in one ear only. CROS (contralateral routing of signal) hearing aids pick up sound from the side with little or no hearing and feed it to the better ear. BiCROS (bilateral-CROS) aids amplify sound from both sides and feed it into the ear that has some hearing.

Waterproof and *water-resistant hearing aids* have a thin membrane to keep water from getting into them. Waterproof aids are suitable for swimming; water-resistant aids can be used for water sports.

In preparation for wilderness adventure, written instructions or a video with closed captions may be beneficial. The group leader must understand the best way to communicate with the person(s) and method(s) used to communicate with other members of the group. Use of interpreters may help facilitate communication.

There are different types of sign language. American Sign Language (ASL) is most commonly used in the United States. Some individuals use ASL as their first language, with English as their second language. ASL is not equivalent to English. It is a visual-spatial language that has its own syntax and grammatical structure. Even if a person is able to read lips, only 30% to 40% of spoken English is distinguishable on the mouth and lips under the best conditions. If the individual cannot understand what is being said, the speaker can rephrase the wording.

It is important to be aware that a person who is hard of hearing may also have difficulties with speech, reading, and writing skills, given the close relationship between language development and hearing.

When communicating with an individual who is deaf, it is important to face the person and maintain eye contact and speak directly to the person. One must have the person's attention before speaking. A light touch on the shoulder or visual signal will help. Providing visual cues and as many concrete examples as possible may be useful. Attentive listening is required when the deaf person is speaking. If the person's speech

is unclear, do not pretend to understand what was said. Ask for clarification.

Individuals with hearing loss communicate in different ways depending on the amount of residual hearing, type of deafness, language skills, age of onset, speech abilities, lip reading skills, and educational background. Some are more easily understood than others. Some use speech only or a combination of sign language, finger spelling, writing, body language, and facial expression.

Slow down the pace of communication as needed to facilitate understanding. Allow extra time for the person to ask or answer questions. Demonstrate how to do something as the situation allows.

The wilderness guide and team members, including the individual with compromised hearing, need to communicate carefully to be certain the activities planned are clearly understood. Important information can be written and shared if there is concern about a hard-of-hearing individual understanding guidelines, rules, or other aspects of a trip. Paper and pencils should be in ample supply on a wilderness expedition. Pencils are preferable to pens because the ink can freeze, leak, or otherwise become unusable. Pencils can readily be sharpened.

Water sports enthusiasts may need to use individualized molded earplugs if a conductive disorder exists. When tympanic membrane tubes are used, care should be taken to keep water out of the ears. Paddlers and other water lovers should wear individualized earplugs whenever the potential of immersion exists.

Dogs are sometimes used to help facilitate interaction with the environment for the person with hearing loss. Service dogs are allowed in wilderness areas and can alert individuals with impaired hearing to changes of sound in the environment.

Traveling with Service Animals

With the passage of the Americans with Disabilities Act in 1990, service animals became defined as "any guide dog, signal dog, or other animal individually trained to do work or perform tasks for the benefit of an individual with a disability, including, but not limited to, guiding individuals with impaired vision, alerting individuals with impaired hearing to intruders or sounds, providing minimal protection or rescue work, pulling a wheelchair, or fetching dropped items."[8] Service animals are not considered pets and, in general, when accompanying a person with a disability, are allowed wherever visitors or employees are allowed and are allowed to go into areas where pets are traditionally prohibited. In some circumstances, the national park superintendent may close an area based on individualized assessment and a written determination that allowance of any domestic animal would pose a direct threat to the health and safety of people or wildlife.

Blind persons may decide to bring their seeing-eye dog when traveling to a foreign country. Three U.S. federal agencies will be encountered when traveling with an animal: the U.S. Department of Agriculture (USDA), Animal and Plant Health Inspection Service (APHIS), and Veterinary Services (VS).

International health certificates for export of animals from the United States are completed by an APHIS-accredited veterinarian who certifies animal health status, conducts tests, and records test results. Completed and signed international health certificates for the export of animals from the United States must be endorsed by a VS area office to be valid. To obtain the USDA endorsement of an international health certificate or any other documents relating to traveling with a service animal or pet, the documents must be completed by an APHIS-accredited veterinarian. The VS area office for each state can be located at www.aphis.usda.gov/vs/area_offices.htm.

Although the United States has minimal requirements for exportation of animals to other countries, each country may have specific health requirements for entry of animals. These requirements are established by the importing country. Regardless of the length of stay in another country, if a pet is in accompaniment, the country's consulate or embassy should be contacted for information about any requirements that must be met. A listing of consulates can be found at www.state.gov/s/cpr/rls/fco/ (U.S. Department of State website). The travel restrictions for most countries can be reviewed on the APHIS website at www.aphis.usda.gov/vs/ncie/iregs/animals/. If a country or a species within a country is not listed, the requirements are not known. The appropriate ministry in the destination country should be contacted for specific requirements.

Further information on taking animals to foreign countries can be obtained at www.aphis.usda.gov/vs/ncie/exp-pets.html.

COGNITIVE AND EMOTIONAL DISABILITIES

Traumatic Brain Injury

Traumatic brain injury is one of the most common causes of disability in the United States. There are a variety of ways to classify the degree of TBI. The most commonly used is the Glasgow Coma Scale (GCS). An initial GCS score of 13 or 14 is considered a mild TBI. An initial GCS score of 9 to 12 is considered a moderate TBI. An initial GCS score of 8 or below is considered severe. There is no consistent correlation with recovery based on the initial GCS score. However, the greater the initial degree of injury to the brain, the more deficits an individual tends to develop in the long term. Anoxic brain injury is associated with potentially worse outcomes.

Individuals may have movement disorders, as well as emotional and behavioral difficulties. One effect often associated with TBI is cognitive impairment, especially memory deficits. Providing written directions, specific instructions regarding planned activities, and details of what to do in an emergency may be helpful.

Some individuals may be impulsive, and others may have some degree of emotional lability. Sixty percent of people with TBI have sleep disorders. This can affect endurance. It is important to note level of endurance because decreased tolerance for rigorous activity may develop as the day goes on.

As much as 30% to 65% of individuals with TBI develop symptoms of traumatic vestibular pathology at some point during their recovery.[253] These symptoms include vertigo, disequilibrium, and gait ataxia. Understanding the effects on gait, posture, and perceptual function is important to maximize safety.

Psychological/Psychiatric Conditions

As defined by the American Psychiatric Association, mental illness is a clinically significant behavioral or psychological syndrome that occurs in an individual, associated with present distress or disability or with a significantly increased risk of suffering death, pain, disability, or an important loss of freedom. Approximately 5.5 million Americans suffer from severe mental

TABLE 90-10. Psychiatric Medications and Potential Side Effects

MEDICATION	POTENTIAL SIDE EFFECTS
β-Adrenergic blockers	Affect heart rate response
Prolixin	Increases blood pressure
Haldol	Causes tachycardia, possibly hypotension, and QT interval prolongation on electrocardiogram
Antipsychotic	Gait disturbance secondary to tardive dyskinesia; dehydration medications
Antidepressants	Insomnia, weight gain, dizziness
Antianxiety agents	Drowsiness, withdrawal, potentiate effects of alcohol

Data from references 10 and 221.

illness, the most common diagnoses being bipolar disorders, personality disorders, and schizophrenia.

Individuals are often treated with medications that may have an influence on cardiovascular function. Table 90-10 lists groups of medications and potential side effects.[10,221]

Before embarking on a wilderness adventure, it may be beneficial for an individual to participate in an orientation session to gain increased understanding of the expectations for a given activity. The psychological and emotional status of individuals with psychiatric disabilities vary from day to day. The psychological status may affect motivation and ability to perform in a wilderness environment. There may be gait disturbance or tardive dyskinesia as a side effect of antipsychotic medications. Activities that increase a person's sense of vulnerability and lack of control may increase anxiety levels.

Mental Retardation

The American Association on Mental Retardation defines mental retardation as "significantly sub-average general intellectual functioning existing concurrently with related limitations in two or more of the following applicable adaptive skill areas: communications, self-care, home living, social skills, community use, self-direction, health and safety, functional academics, leisure, and work."[216] Sometimes mental retardation is associated with a particular medical condition. For example, individuals with Down syndrome may have associated congenital heart problems or ligamentous laxity. They may also have ambulatory limitations or decreased coordination skills.

An outdoor adventure or wilderness experience is of great value in providing learning opportunities and interaction with the environment.[205] The focus of a program for an individual with mental retardation could include maximizing social skills and experiencing new physical challenges. Motivation may be one of the primary obstacles for ongoing participation and cooperation in a given activity. Levels of support may be considered across four dimensions: intellectual functioning and adaptive skills, psychological emotional concerns, physical/health/etiology concerns, and environmental concerns. These levels of support may be required intermittently, on a limited basis, extensively, or pervasively.[72]

SPORTS AND RECREATION EQUIPMENT

Through research and development, technology is enabling access to recreation in the outdoors for people with a wide range of abilities and interests. Technology has improved, strong and lightweight materials such as titanium and carbon fiber have been developed, and computer-aided design and manufacturing have created better products.

Hunting

Many adaptations are available to enable hunters to return to the wilderness. Some states allow handicapped people to hunt legally from a vehicle. However, many disabled people prefer to hunt without special privileges. Both the mobility assist device and the hunting equipment need to be adapted to meet the particular degree of disability.

Wheelchair backs must be high enough to support the torso or pelvis appropriate to the level of injury. Wheelchairs must have wide, knobby tires for greater traction on rough terrain, and they must be maneuverable by the rider to adjust for small position changes. Guns, rifles, or bows and arrows must be securely attached and in a position where they can be fired by a person with tetraplegia. If a person has gross arm movement and loss of hand, finger, and fine motor movement, a device can be rigged between the trigger and the arm so the trigger can be pulled.

Bow-and-arrow hunting is also possible with equipment changes. Bow-Pro Company adapts bows to the needs of the hunter. Compound bows can be attached directly to a wheelchair with a counterbalanced socket, allowing the bow to swivel to aim. A draw-lock device holds the arrow at full draw until released. Owen Orthmann of Minneapolis is a C3-C4 tetraplegic. He has a crossbow that draws power from his electric wheelchair. He aims it with chin movements while looking through a red-dot sight. He fires by inhaling on a tube, which triggers an electric solenoid to release the bolt.

Many states have established specific seasons and regulations for hunters with disabilities. Some states allow crossbows but prohibit lighted sight pins on crossbows. Other states prohibit the use of crossbows and still others prohibit draw-locks and mouth releases.

Horseback Riding

Horseback riding is an excellent venue for disabled people to experience the wilderness. Hippotherapy (physical therapy using the movement of a horse) combines physical therapy with the psychological therapy of an outdoor environment. One study found that only 8 minutes of hippotherapy in children with CP resulted in significant improvement in symmetry of muscle activity.[20]

Scuba Diving

Spinal Cord Injury

Diving can be safely enjoyed by people with SCI. It can give a paralyzed person a sense of freedom as the constraints of gravity are removed from the environment. For individuals who are able to tolerate exercise and have physical stamina, it opens the door to a whole new world.

Individuals with tetraplegia or paraplegia can meet most, if not all, of the standards used for scuba dive certification. Some of the basic skills, such as flooding, clearing, and removal of the face mask, may be difficult to achieve if there is significant paralysis of the hands and wrists.

A diver with SCI must deal with balance difficulty, hypothermia risk, and anxiety, as discussed in the following sections.

Balance Difficulty. Balance is an integral component of scuba diving. It enables the diver to remain in position of choice during ascents and descents and during the dive. Divers most frequently are in a vertical position on ascent and descent and more horizontal during the dive.

Balance on the water surface and while submerged is normally maintained by motion of the arms and legs and fine adjustments of the trunk. The individual with SCI is unable to use his or her torso, arms, or legs to compensate for slight alterations in the center of gravity. Counterweights can aid in creating symmetric balance. They must be carefully positioned to maintain gravity along the center axis of the body.[196] Placement of weights on a weight belt or integrated buoyancy compression device can change the center of gravity, pulling one forward, backward, sideways, or even upside-down. The position of the weights should initially be distributed symmetrically around the body. Adjusting placement of the weights can be made in the water to maintain the desired body position. Changes in current may affect body position. Weight placement should be reevaluated if there is difficulty maintaining balance and body position.

Neoprene wet suits and boots can increase the tendency for the legs to rise to the surface. The legs can be strapped together and weighted at the ankles to prevent them from floating in an uncontrolled manner. The weights necessary for surface floating, ascent, descent, and during the dive may differ. They should be adjusted for each part of the dive. If heavier weighting is needed for ascent and descent, the extra weights can be removed and left on the anchor line during the dive.[196] Amount and distribution of weights in all phases of a dive are useful dive log entries.

Hypothermia Risk. A diver with a disability may be at greater risk for hypothermia while diving. Normothermia is maintained by an intricate balance of heat loss and heat production. Heat loss is up to 25 times greater in water than air, exposing divers to potential hypothermia.[56] The body generates heat by energy expenditure during voluntary muscle activity and involuntary muscle shivering. Energy used by a nondisabled person during a dive is about eight times greater than energy required to sit at rest.[196] The paralyzed diver potentially uses less energy to dive and has less muscle mass to generate heat through shivering. In addition, heat may escape more easily from the body if the autonomic nervous system is damaged. Consequently, with increased heat loss and decreased heat production, preventing chills and hypothermia should be part of the dive preparation and plan for the person with SCI.

Anxiety. The underwater world is unfamiliar to the new diver and can precipitate a multitude of fears. It is not unusual for a new diver to have anxiety about equipment, entry and exit from the water, depth of dives, and simply being in the open water of the ocean. Anxiety can also arise when an experienced diver is faced with a new circumstance in the underwater environment.

A diver with limited physical capabilities may be very enthusiastic and interested in learning to dive but may have a high level of anxiety about the sport. The diver is not able to sense or control the body below the level of injury and can suffer from vertigo in the water.[196] The diver may be fearful of being unable to maneuver as quickly or easily as desired. It is helpful if the disabled person has been in a swimming pool or other body of water to experience flotation and swimming. If not, spending time in the water without dive equipment can be advantageous for enhancing comfort in the aqueous environment before learning to dive.

Causes of perceived fears should be identified and discussed. Dive equipment should be easy to use and training should be thorough (see following discussions). Meeting those two recommendations will go far toward allaying the diver's anxiety.

Equipment Considerations. A full-length wet suit and dive booties should be worn on all dives for protection from unexpected direct contact with ocean life and for increased thermal insulation. A neoprene hat or full head-and-neck gaiter aid in preventing heat loss. Foot fins are not necessary, but hand fins might be advantageous in increasing maneuverability in the water.

Neoprene dive suits are at best difficult to don. A thin nylon or polyester body suit worn under a wet suit makes donning the wet suit much easier. For maximum effectiveness, the wet suit should fit snugly and comfortably over the thin liner without constriction. The choice of wearing gloves for protection and warmth depends on individual preference and the governing rules of the dive site. Limited hand function in tetraplegia may preclude the choice to wear gloves because gloves will further decrease the ability to hold and operate dive equipment. If hands have a tendency to get very cold and stiff, the choice might be in favor of wearing gloves if allowed at the dive location.

Regulators should be easy to purge and have the capacity to adjust resistance for breathing. The disabled diver may or may not be able to roll to recover a dislodged regulator. The regulator should be attached to the front of the buoyancy control device (BCD) for rapid and easy recovery should this occur. The secondary stage should also be readily accessible to the diver without requiring physical maneuvers to find its location.

Training. Dive training for the disabled diver follows the same protocol as for anyone learning to dive. A disabled person with full use of hands is most likely able to meet the standard requirements for Professional Association of Diving Instructors (PADI) or National Association of Underwater Instructors (NAUI) dive certification. If the SCI is at a high level and paralysis affects the hands and arms, some skills, such as mask clearing, may be difficult to accomplish. Most basic diving skills do not require fine motor movement or dexterity of the hands and fingers. The international signals can be adjusted to meet the functional capability of the individual. This information needs to be clearly communicated with the dive buddy and dive master on each dive.

During the pool and open-water training modules, a higher staff-to-student ratio is recommended. Initially, one staff person for each student enables uninterrupted attention that can lessen the disabled diver's apprehension as the diver becomes accustomed to scuba gear and his or her body in the water. The first pool session should focus on balance and weight distribution to promote proper body position and comfort in the water. This deviates from the usual initial pool skills of clearing one's mask and exchanging snorkel and regulator.

Scuba diving off a boat with a dive platform provides easiest water entry and exit. There must be someone on the boat capable of lifting a disabled diver out of the water and onto the platform. Lifting a diver over the edge of a boat without using a dive platform is much more difficult. If extra weights on legs are used during ascent, they should be removed in conjunction with the weight belt or the integrated weight system before

lifting the diver from the water. The air tank can be disconnected from the BCD and passed up to the boat crew to decrease the lifting weight even further. The BCD should not be removed because it provides handholds with which to lift.

Beach diving is also a possibility, although water entry and exit require planning. A small diver can be carried to and from the water, but this is difficult if the person is very large. Devices that slide easily on the sand, such as a boogie board or an old surf board, can be used to pull the person into the water. Dive equipment can be put on and taken off while sitting in shallow water. If able, the diver can then rotate to his or her stomach and pull herself further into the water, timing the motion with incoming waves. If the dive equipment is already on the diver, the diver can be pulled or carried into deeper water and begin the dive. Big waves or a strong undertow create additional hazards for beach diving.

Although previously discussed, a full bladder and the potential complication of AD is worthy of mention again. Descending one, two, or more atmospheres creates increased pressure on the body, resulting in increased diuresis. It is common for a diver to feel the need to urinate soon after descent. Because a person with paraplegia may not sense a full bladder, it is important to recognize the need to empty the bladder more frequently, preferably just before and after each dive.

Dive sites should be evaluated for the strength of current and the potential need to swim against the current during the dive. Without the use of legs and foot fins for kicking, the disabled diver may not be capable of swimming against strong currents. It is also wise to guard against the need for a long surface swim for similar reasons.

Asthma

Diving with asthma is controversial because of the risk for air trapping and pulmonary overinflation during ascent.[44,107,190] Because asthma is unpredictable and persons with asthma may develop airflow obstruction in the lungs at any time, the question of if and when a person with a history of asthma can safely dive is problematic. Historically, recommendations on diving ranged from "never" to "no diving within 2 days of wheezing."*

There is no solid evidence that divers with asthma have an increased rate of accidents or decompression illness.[44] The tendency to restrict asthmatic people from diving has been more theoretical than evidence-based because pulmonary barotrauma has not been supported by clinical observation.[30] Elliott established pulmonary function criteria to be used as a guideline for safe diving by an individual with asthma. The suggested parameter is that expiratory airflow at 50% of peak (mid-expiratory flow) should not be reduced after exercise by greater than 50%.[64]

The choice to dive or not to dive is best made in collaboration between the patient and physician. A physical examination, detailed medical history, chest x-ray, and simple tests of lung function allow the physician to discuss the issues, enabling the potential diver to make an informed decision.[50,154]

Diabetes

Varied opinions prevail regarding the safety and prudence of scuba diving by diabetic patients. Most dive organizations and operators allow an individual with diabetes to dive if the diver has a medical release signed by the diver's physician.

*References 69, 90, 103, 111, 123, 155, 167, 170, 188, 189, 231.

Bove,[31] having made over 200 dives with diabetic individuals, wrote an excellent paper suggesting stringent requirements to be used as guidelines in determining the appropriateness of scuba diving for an individual with diabetes. He highlighted six conditions to be considered:

1. The prospective diabetic diver must be a responsible individual, understand the potential consequences of diving, and have physical capability for the activity.
2. The prospective diabetic diver should have a thorough understanding of the interrelationship of diet, exercise, and insulin, know how to make mild adjustments according to changes in activity, and be well controlled on a day-to-day basis.
3. The prospective diabetic diver must be able to perform hard physical activity on land before choosing to scuba dive and must be able to respond to an emergency situation underwater.
4. The prospective diabetic diver should have no other impairments or disabilities potentially causing predisposition to or confusion with a diving-related problem.
5. The diabetic diver should avoid difficult, physically demanding dives and dive a conservative dive profile, more conservative than the guidelines of the standard decompression tables.
6. The diabetic diver must tell dive buddies and the dive master that he or she is diabetic and should only dive with a buddy and dive master that are well informed about diabetes and know how to recognize and treat a hypoglycemic reaction.

The diabetic person should know her or his personal early symptoms of hypoglycemia. Increased awareness for onset of symptoms underwater is imperative because symptoms may differ from those on land. For example, if early terrestrial symptoms include cool, clammy skin and diaphoresis, these may not be discernible underwater. Symptoms more discernible underwater may be shortness of breath unrelated to increased exertion, weakness, and lightheadedness.

There is no universal underwater signal indicating the onset of a hypoglycemic reaction. Before the dive, the diver should devise an underwater signal with dive buddies for "I am feeling low on sugar." This signal should be practiced during training and reviewed before each dive with the dive master and members of the dive group.

Recognized early, hypoglycemia can be treated underwater. Unless carried in a waterproof container, hard candy could dissolve and be gone by the time it is needed. An oral glucose gel carried in a BCD pocket can be readily available. Glutose (40% dextrose) is available in a 15-g tube. The diabetic diver should be assisted as needed with removal of the regulator, ingestion of glucose, and reinsertion of the regulator. The diabetic diver and dive buddy should begin a controlled ascent with other divers assisting as necessary. Should the diabetic diver lose consciousness, the regulator can be held in the mouth for completion of controlled ascent. The head and neck need to be held in proper alignment to enable ventilation for the unconscious victim. If the regulator becomes dislodged, the next best action is to send the unconscious individual to the surface. The dive buddy should then complete his or her controlled ascent and at the surface begin resuscitation as needed.

Epilepsy

Swimming and scuba diving present the risk of neurologic complications or drowning during a seizure. One case was found in

the literature of a diver dying of cardiac complications from pulmonary barotrauma after rapid ascent during a seizure.[100] The dive certification agencies NAUI and PADI and most physicians will not authorize people with epilepsy to scuba dive.[59,91]

Visual Impairment

The underwater world is a world of sensory deprivation compared with noises on land, yet the ocean is full of the sounds of animal life. A blind person, often with greater acoustic adaptability than a sighted individual, may well enjoy listening to the sounds of the underwater world. The feel of water pressure on the body, flow of currents, and sounds of feeding fish are all opportunities worth experiencing.

The BCD, tank, and regulator require no special adaptation for the vision-impaired diver. However, there are currently no talking or Braille dive computers available to enable depth, time, and remaining air to be tracked. Because diving is a buddy sport, it can safely be done with proper dive planning. The direction of the dive in relation to underwater currents should be discussed before water entry. The swimming speed should be planned. It is important to agree on the desire to swim at a certain speed or to remain in a small area listening to sounds of the underwater environment.

The sighted diver can lead, with the blind diver following the sound of air bubbles, or the blind diver can lead, with the sighted diver tracking location for the course of direction to return to the boat. The sighted diver needs to monitor carefully the necessary gauges of his or her buddy. The divers should have a tank banger for communication and preestablish a simple code to meet underwater needs of communication. Two bangs could mean "go to your right" and four bangs could mean "go to your left." There should be a unique underwater sound code communicating the need for immediate help. The blind diver should be kept in sight by the dive buddy at all times. If an inadvertent separation occurs, the tank banger can be used as a means of finding and reconnecting the two parties within the audible distance of the sound.

Without proprioceptive inputs in an environment without gravity, the blind diver may become disoriented about up and down. The diver can easily remain oriented in a head-above-the-feet position by placing a hand over the bubbles as they leave the regulator, to feel in which direction they are rising. If the bubbles are going towards the feet, the diver can readjust position.

Hearing Impairment

Diving is not contraindicated for individuals with hearing loss. For divers that know sign language, it is an excellent way to communicate if other divers are nearby.

White-Water Sports

Canoeing and kayaking can provide river access to outdoors and wilderness environments that are not practicably accessible by land, or that are ordinarily inaccessible to people with mobility limitations.

Before a river experience, the mobility-impaired individual should spend time in a swimming pool learning how his or her body reacts in water. Some legs have a greater tendency to float, whereas others have a greater tendency to sink. It is important to assess the degree of arm mobility necessary to enable one to turn and control direction in the water.

Whether in a canoe, kayak, or raft, the risk of falling into the river, in part, depends on the difficulty of the rapids. Participants in commercially supported white-water activity are required to wear a personal flotation device (PFD). Helmets are also worn when running more difficult rapids. Before a river trip, the group is instructed in correct body position and maneuvers to be used should one accidentally fall out of the boat. The normal technique for managing body position in a river is to get on one's back with legs close together and pointing downriver. The legs are in front and naturally float close to the surface of the water. Without control over legs, they can randomly move apart and in any direction. They are more prone to sink than float, increasing the risk of a leg becoming caught underwater on a rock or other obstacle. This creates a significant hazard because the individual can become pushed or sucked under by the force of the moving water.

The legs should be strapped together before getting in a raft, canoe, or kayak. This keeps the legs together and going in the same direction while in the water. Any type of material for a strap will work, provided it does not constrict the extremity. The important aspect of strap use is that it be securely fastened so it cannot come apart in the water. It must not have loose ends that can be caught underwater and cause entrapment. If the legs tend to sink, a pull buoy can be attached above the knees or at a level to make them neutrally buoyant.

A paralyzed person sitting in a kayak may not be able to feel the cold water around his or her legs or buttocks. Frequent checks for adequate circulation and warmth should be made.

Canoe and kayak rental facilities and instructional programs currently rely on commercially available seats. Standard canoe seats provide a bench for the paddlers to sit on or lean against. Without placement of another type of chair with a back rest on top of the bench, the paddlers must use their own balance, torso, and leg strength to maintain an effective sitting position.

Similarly, an additional chair needs to be mounted onto the standard canoe bench to stabilize the pelvis and provide lower back support. The functional benefits of pelvic stability are well documented for wheelchair users. The disabled paddler must maintain truncal and pelvic stability to effectively paddle. Although commercially available backrests are available, they are not designed to support individuals who are unable to sit independently. The disabled paddler must rely on an external back support system. It should include lateral support for pelvic stability and a stable attachment to the existing seat. It should also be adequately padded to provide sufficient cushioning to prevent pressure ulcers or abrasions.

Ski or hiking poles are helpful for kayak entry and exit from the water (Figs. 90-19 and 90-20).

Off-Road Riding

Wheelchair users may want to enjoy the rugged ride offered by back-country trails. When one is mountain biking, it is important to be able to adjust one's center of gravity to optimal positions for ascents and descents. For a wheelchair, this adjustment capability needs to be possible without fundamentally modifying the wheelchair frame.

The Cobra Off-Road Wheelchair makes the mountain biking experience available to the wheelchair rider. The chair has four-wheel disc brakes, knobby mountain bike tires, and directional steering. The Cobra is capable of seat adjustment, positioning the rider's center of mass back and low when going downhill and high and forward for the uphill climbs.

Figure 90-19. Dean Juntunen at a take-out on Sturgeon River. (Photograph courtesy of Jim Waters.)

Figure 90-20. Dean Juntunen at a put-in on East Branch of Ontonagon River. (Photograph courtesy of Jim Waters.)

The One-Off (One-Off Titanium, Inc., www.titaniumarts.com) is also geared for pedaling on mountain terrain, but requires a double track or wide trail because of the dual wheels on the front of the bike (Fig. 90-21; see also Fig. 90-10).

Skiing

A variety of skis are available for the winter outdoor enthusiast. Sit-skis, toboggan skis, and cross-country skis offer a variety of modes to enjoy backcountry exploring or tracked ski trails (Fig. 90-22).

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Figure 90-21. Jeff Pagels maneuvering over snow on a winter mountain climb. (Photograph courtesy of Sheryl Olson.)

Figure 90-22. Single-track cross-country skier at Ski for Light, 2005. (Photograph courtesy of Craig Gray.)

APPENDIX A: LIST OF OUTDOOR-ADVENTURE RESOURCES FOR PERSONS WITH SPECIAL NEEDS

"Tools for Play" Program

Tools for Play is a multimedia program that was presented at the Opening Session of the Paralympic Congress. It emphasizes the importance of recreation and leisure activity for everyone and the ability of people with disabilities to participate. Produced by Gayle Curtis, Veterans Affairs Palo Alto Rehabilitation Research and Development Center.

http://guide.stanford.edu/toolsforplay/tfp.html

Tools for Play also provides an alphabetical list of recreation and leisure activities that can be selected to learn more about resources that support it. Several organizations have programs that support multiple activities.

http://guide.stanford.edu/toolsforplay/activities.html

Magazines

Sports 'N Spokes (www.pvamagazines.com/sns/)
New Mobility (www.newmobility.com/)

Equipment

Gloves, custom fit (www.saranacglove.com)
HouseHold: The self-catheterization device
Flexlife
4319 Broadleaf St.
Kingwood, TX 77345
(281) 360-6773
flexlife@msn.com

Associations, Clubs, and Travel Companies

The following organizations can provide further information on adventure trips, sports, and sports-related activities for individuals with disabilities.

Access Sport America
119 High St.
Acton, MA 01720
www.windsurf.org

Access to Sailing
19744 Beach Blvd., Ste. 340
Huntington Beach, CA 92648
(714) 722-5371 w
(310) 987-4047 f

Accessible Cruise Planners
Specializes in accessible cruising.
(800) 801-9002
www.love2cruise.com

Adapt Ability
Czeslaw Cimachowski
Custom Rehabilitation, Recreation, and Sport Equipment Designer
Power Wheelchair and Seating Systems Designer and Consultant
912-65 High Park Ave.
Toronto, Ontario M6P 2R7
Canada
(416) 604-2214
adapt@netcom.ca

Adaptive Aquatics
1473 Paradise Cove Ln.
Wilsonville, AL 35186
www.adaptiveaquatics.org
205-807-7519 w

American Canoe Association
7432 Alban Station Blvd., Ste. B-226
Springfield, VA 22150
(703) 451-0141 w
(703) 451-2245 f

American Handcycle Association
Mr. John Gardner
1744 Pepper Villa Dr.
El Cajon, CA 92021-1214
(619) 596-1986 w
cmgmai@aol.com

American Sled Hockey Association
10933 Johnson Ave., So.
Bloomington, MN 55437
(612) 644-2666 w

American Water Ski Association
1251 Holy Cow Road
Polk City, FL 33868
863-324-4341 w
863-325-8259 f
www.usawaterski.org

Americans with Disabilities Act
http://ada.gov

America's Athletes with Disabilities
143 California Ave.
Uniondale, NY 11553

Association of Disabled American Golfers
7700 E. Arapahoe Rd., Ste. 350
Englewood, CO 80112

Aqua Sports Association for the Physically Challenged
830 Broadway, Ste. 10
El Cajon, CA 92021
(619) 593-8777 w
(619) 593-1148 f

Bay Area Outreach Recreation Program (BORP)
830 Bancroft Way
Berkeley, CA 94710
(510) 849-4663 w
(510) 849-4616 f

BC Rehab
Ms. Anne Opelka-Salumay
4255 Laurel St.
Vancouver, British Columbia V5Z 2G9
Canada
(604) 737-6372 w
(604) 737-6349 f
asalumay@bcrehab.vancouver.bc.ca
www.bcrehab.bc.ca

Beasley Travel
Specializes in travel for the deaf and hard of hearing
866-584-5266
www.beasleytravel.com

Beneficial Designs, Inc.
1617 Water Street, Ste. B
Minden, NV 89423-4310
(775) 783-8822 w
(775)-783-8823 f
mail@beneficialdesigns.com
www.beneficialdesigns.com

Bloorview MacMillan Centre
Applied Technology Program
Mendal Slack
350 Rumsey Rd.
Toronto, Ontario M4G 1R8
Canada
(416) 424-3826 w
(416) 421-3821 f
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Wilderness and Endurance Events

91

David A. Townes

Perhaps the earliest record of an endurance event may be traced to ancient Greece in the 5th century B.C. The Persians invaded Greece in 490 BC, landing in Marathon, a small town about 26 miles from Athens. Seriously outnumbered by the Persians, the Athenians sent messengers to cities throughout Greece requesting assistance. Legend has it that after the battle, a man named Pheidippides was sent from Marathon to Athens to bring word of the Greek victory. He covered the 26-plus miles on foot only to drop dead after proclaiming *"Niki!"* (Victory). There is some debate among historians about what really occurred. There is evidence that Pheidippides was actually sent from Marathon to request help and that news of the victory was delivered by a man named Eukles.[16]

Although the exact details remain unclear, when the modern-day Olympic games were inaugurated in Greece in 1896, the legend of Pheidippides served as inspiration for the marathon. That first marathon covered a distance of 24.85 miles (40,000 meters), the distance from Marathon Bridge to Olympic Stadium.[47]

During the next 28 years, the marathon continued to evolve. In the United States, the Boston Athletic Association held its first marathon on April 19, 1897 to commemorate the famous ride of Paul Revere on that date in 1775. For the Olympic Games in London in 1908, the marathon distance was changed to 26 miles, the distance from Windsor Castle to White City Stadium, with an additional 385 yards added so the race would finish in front of the royal family's viewing box. Finally, in the 1924 Olympic Games in Paris, the distance was set at 26.2 miles, establishing the modern-day marathon.[47] Today, there are hundreds of marathons held throughout the world each year.

In addition to marathons, the past quarter century has seen tremendous growth in the number, type, variety, and popularity of endurance events, including cycling events, triathlons, ultra-triathlons, and ultra-marathons.

Advances and improvements in equipment, along with relatively efficient and affordable travel, have allowed increased participation in such activities as adventure travel, backcountry skiing, mountain biking, mountaineering, orienteering, rock climbing, sea kayaking, scuba diving, snowboarding, trekking, and white-water rafting and kayaking.

The growth of these activities, along with the continued popularity of endurance events, has led to the development of activities that combine aspects of both. During the past decade, wilderness multisport endurance events, also referred to as *adventure races* or *multisporting*, have soared in popularity throughout the world, with increasing numbers of events and participants each year.

With the growth of adventure races, cycling events, marathons, and triathlons has come increasing demand for medical support for these activities. The provision of medical care for wilderness and endurance events represents a unique area of wilderness and event medicine. This chapter introduces the subject and suggests strategies for the development of a *medical support plan* for these activities using adventure races as the primary example. Some of the injuries and illnesses associated with participation in these activities are discussed. In addition, some of the challenges and controversies encountered are highlighted. This information should prove useful for those charged with the provision of medical care for these activities as well as those participating themselves.

MEDICAL SUPPORT FOR MASS GATHERINGS

In terms of the provision of on-site medical care, events have traditionally been divided into three categories, classes, or types. Category I events are those in which spectators remain seated for a set period of time or for the duration of the event. Common examples include stadium sporting events and concerts. In category II events such as golf tournaments, carnival and Mardi Gras celebrations, and state fairs, spectators are mobile and may become participants in the events. A large geographic area and participants often outnumbering spectators characterize category III events, which include charity walks, bicycle rides, marathons, and triathlons.[33]

Because of their extreme nature and the unique challenges in providing medical support for them, several authors have labeled adventure races and similar events category, class, or type IV events.[5,48,49,51] These events occur in rough and often remote terrain where communication may be very difficult and transport times to definitive care prolonged. With no set course, the exact location of each team may be unknown, and technical search and rescue may be required (Fig. 91-1). In addition, many of these events are not staged, resulting in hundreds of miles separating lead teams from the back of the pack. A *wilderness event* has been previously defined as "an event with more than 200 participants and where the time from injury to care at a medical facility is greater than one hour."[5] The authors go on to suggest the evolution of a new specialty: *wilderness event*

Figure 91-1. Adventure races represent a unique area of wilderness and event medicine because they have no set course, and technical search and rescue may be required. (Photograph courtesy of Dr. David Townes.)

medicine (WEM).[5] This definition is perhaps a bit too restrictive, and the establishment of a new specialty remains to be seen. What is certain is that adventure races represent a new and important area of event and wilderness medicine.

Mass gatherings have been defined in the literature as events with more than 1000 participants and as events with more than 25,000 participants.[7,30] Category III and IV events do not generally meet these definitions because they often have fewer than 1000 participants, except in the case of some marathons and triathlons. Most investigations of medical support for mass gatherings have described the frequency and type of injury and illness treated, the rate of utilization of on-site medical services, and the rate of transfer to local care facilities for category I and II events. The focus of many of these investigations has been to determine what factors influence the type and frequency of injury and illness to better anticipate need and thus establish appropriate guidelines and standards of care. Much of the information is anecdotal and descriptive, and several studies have concluded that there is no standard of care for emergency medical services at mass gatherings.[2,7,38,41]

Most investigations have been of category I and II events, with a smaller number of investigations of category III events and few investigations of category IV events. Some of the general findings of the investigations of category I and II events are described below.

The incidence of true medical emergencies at mass gatherings appears to be relatively small. In one large study, respiratory illness, heat-related injuries, and minor problems including sunburn, blisters and headache accounted for 75% of medical encounters. Asthma was the most common reason to require acute medical intervention.[3]

The relationship between attendance or crowd size and utilization of on-site medical services is unclear. Several studies found that overall utilization grew with attendance but that utilization *rate* did not increase and, in some cases, actually decreased with larger attendance.[2,8,54] Rate of utilization of on-site medical services varies widely between events from 0.14 to 90 patients per 1000 participants, with most events in the range of 0.5 to 2 patients per 1000 participants.[2,7,41]

A number of factors influence the utilization rate and type of medical care required, including the type and duration of the event, the weather, the availability of alcohol and drugs, and the demographics of the crowd, including average age, density, and mood.[2,3,30]

Temperature and relative humidity have a major effect on utilization of on-site medical resources at mass gatherings. They are both associated with increases in demand for on-site medical services; however, humidity has a larger effect than temperature. The availability of water also influenced the incidence of dehydration and heat illness.[2,7,26] For any event, it is important to know the likely temperature and humidity and plan accordingly.

Crowd (participant) demographics, event type, and availability of alcohol and drugs may also be used to help estimate utilization of medical resources. As one might predict, studies demonstrate that when alcohol is readily available, there is an increase in medical problems related to intoxication.[2,30] One might expect to treat medical problems related to drug and alcohol use during a rock concert. In contrast, during a Papal visit, one might expect less intoxication but more cardiac-related problems.[26]

The provision of medical support for any event begins with the development of a medical support plan. Several authors have described this process for mass gatherings.[8,23,26] The basic goals are to provide rapid access and triage, stabilize and transport seriously injured or ill patients, and provide on-site care for minor complaints.[7] Nine elements of planning have been described, including attendance or crowd size, personnel, medical triage and facilities, communication, transportation, medical records, public information and education, mutual aid, and data collection.[23] The importance of early planning and good communication cannot be overemphasized.[52] For any event, the medical support plan should be based on *anticipation of need*. This begins with an estimation of both the number of patients and the type of injury and illness that will require treatment in both the best-case and worst-case scenarios. It is often helpful to review utilization of medical resources for similar events that have been held.[26] In addition, as previously described, a number of factors influence utilization of medical resources that should be considered.

Some general recommendations have been made about the location and staffing of on-site medical facilities at mass gatherings. One group of investigators recommend that advanced life support (ALS) units be in place so that the response time from collapse to ALS care is 5 minutes or less for all participants under all conditions.[41] Others have suggested the goal of basic first aid in 4 minutes, ALS care in 8 minutes, and evacuation to a medical facility within 30 minutes.[38] For staffing, it has been suggested that the minimum staffing for every 10,000 participants be a two-person team consisting of registered nurses, emergency medical technicians, or paramedics or a combination.[8,23]

Appropriate on-site medical support for mass gatherings and wilderness and endurance events is equally important to help ensure the health and safety of the participants. As the popularity of wilderness and endurance events grows, courses are longer and more demanding, events are held in more remote

and exotic locations throughout the world, and the potential for illness and injury increases. The information available from the study of mass gatherings is helpful in the provision of medical support for wilderness and endurance events. The basic influence of attendance, temperature, and relative humidity on utilization of medical resources is likely to be similar across all events. Caution should be used however, when applying *utilization rates* from category I and II events to category III and IV events. As one might predict, utilization rates of on-site medical resources tend to be higher for category III and significantly higher for category IV events.

Similar to mass gatherings, the provision of on-site medical care for wilderness and endurance events begins with development of a medical support plan. This should be done under the direction of the event's medial director who might be a physician, paramedic, emergency medical technician, nurse, or other medical professional. The primary responsibility of the medical director is the health and safety of the participants. Ideally, this individual should have prior experience as a medical director for similar events and will serve as physician, planner, advisor, educator, and liaison with the community.[19] It is essential that he or she be familiar with the location of the event, including the capability of local emergency medical services (EMS), local health care facilities, and in the case of category IV events such as adventure races, the local search and rescue (SAR) system. Medical support plan development should begin several months to a year before the event depending on its complexity.

In general, the medical support plan should be comprehensive and outline all aspects of medical support, including a complete list of medical supplies, equipment, and personnel (see Table 91-2). Treatment and transfer protocols should be clearly outlined, assigning any penalties for receiving medical care and establishing indications for medical withdrawal from the event. The medical support plan should be based on estimates of the type and frequency of injury and illness expected for the individual event in both the best and worst case scenarios.

Adventure races are used as the primary example because they present the greatest challenge in terms of complexity in the provision of medical support. Much of the information provided can be applied to other endurance events. There are, however, some unique aspects in the provision of medical support for marathons, triathlons, and cycling events that are discussed. In general, for these events, adjustments in the type and amount of injury and illness anticipated should be made and communication, EMS, and SAR protocols can be simplified or eliminated.

ADVENTURE RACES

In adventure races or wilderness multisport endurance events, athletes compete over a course that requires performance of multiple disciplines that may include caving, fixed-line mountaineering, flat- and white-water boating, hiking, in-line skating, mountain biking, navigation and orienteering, technical climbing and ropes skills, trail running, and trekking. Races are categorized by duration into sprint (<6 hours), intermediate (6–12 hours), long (12–36 hours), and expedition length (>36 hours).

Adventure racing as we know it today began in the early 1980s with the first large, well-organized events including the Coast-to-Coast, started in New Zealand in 1980, and the Alaska Wilderness Classic, started in 1983. These were followed by other well-known events, including New Zealand's Raid Gauloises and the Southern Traverse, begun in 1989 and 1991, respectively. The Eco-Challenge introduced adventure racing to the United States in 1995. The Primal Quest, started in 2002 in Telluride, Colorado, has brought adventure racing more into the mainstream of American sports through network television coverage. In addition to these expedition-length races, there are countless shorter races throughout the world.

Expedition-length adventure races are competitive, team events that require at least one member of the four- or five-person team to be of the opposite sex. Teams race together, with each team member completing each discipline along the course. The course may cover hundreds of miles and take up to 10 days or more to complete.

In many expedition-length adventure races, there is no set course. Teams are provided maps and the Universal Transverse Mercator (UTM) coordinates for each checkpoint and transition area (where teams change disciplines) that they must pass through. Unique to category IV events, it is left to the team to decide the best route between checkpoints depending on their strengths and weakness. Whereas one team may opt to go around a ridge, another may go over it. In addition, there are no built-in rest periods, and once the race begins, teams may race around the clock. An individual team must strategize if and when to rest.

When teams are racing on the course, they are governed by a set of instructions called the "rules of travel." These rules dictate multiple aspects of the race, such as where and when a team may travel on paved roads, existing trails, or water. For example, white-water travel is often prohibited at night in the interest of safety. In addition, the rules of travel mandate safety equipment that must be used for each discipline.

The rules of travel also dictate several aspects of the event pertaining to medical care that should be included in the medical support plan. They govern the use of medications including performance-enhancing substances, specify penalties for utilization of medical resources during the race, and outline criteria for medical withdrawal from the event.

A breach of the rules of travel results in a penalty for the offending team. Minor infractions such as travel on an unapproved section of paved road might result in additional hours being added to the team's total time at the end of the race. Major infractions such as not wearing a helmet during a mountain bike section, use of a banned substance, or severe injury or illness may result in disqualification.

The team to complete the course with the fastest time after all penalties have been allocated is declared the winner. In many events, prize money is awarded to the top teams.

DEVELOPMENT OF A MEDICAL SUPPORT PLAN

For the event medical director, adventure races and other category IV events present the greatest challenge in medical support plan development, often occurring in sparsely populated rural or very remote, rugged wilderness terrain. In these locations, there are inherent difficulties in communication, travel, and general logistics. It may be unrealistic to assume local EMS, health care facilities, and SAR will be able to handle the potential increased burden imposed by the event.

Figure 91-2. In wilderness and endurance events, the basic goal of the medical support plan should be to provide definitive treatment for minor illness and injury and to provide initial stabilization and facilitate transfer for more severe illness and injury. (Photograph courtesy of Dr. David Townes.)

TABLE 91-1. Injuries and Illness during the Primal Quest (2002)

TYPE OF INJURY/ILLNESS	NO. OF CASES (n = 302)	PERCENTAGE OF CASES
Skin/soft tissue	145	48.0
Respiratory	55	18.2
Altitude (AMS/HAPE)	36	11.9
Orthopedic	29	9.6
Dehydration	21	7.0
Gastrointestinal	6	2.0
HEENT	5	1.7
Genitourinary	3	1.0
Other	2	<1.0

AMS, acute mountain sickness; HAPE, high-altitude pulmonary edema; HEENT, head, ears, eyes, nose, throat.

From Townes DA, Talbot TS, Wedmore IS, Billingsly R: Event medicine: Injury and illness during an expedition-length adventure race. J Emerg Med 27:161–165, 2004.

The basic goal of the medical support plan should be to provide definitive treatment for minor illness and injury and to provide initial stabilization and facilitate transfer for more severe illness and injury[49] (Fig. 91-2). It is fundamental that the medial support plan be based on *anticipation of need*. Unfortunately, for category IV events, there is relatively little information available to assist the medical director in development of a medical support plan, and the challenge of anticipating need remains. The studies that are available demonstrate that medical providers should be prepared to treat a wide variety of injury and illness.[14,51]

Development of a medical support plan begins with a careful review of the course, including its location, the disciplines required, the time of year, and the environmental conditions, such as recent precipitation, temperature, and humidity. In this way, the occurrence and type of injury, illness, endemic disease, and environmental emergencies such as dehydration, heat and cold illness, and altitude illness can be roughly predicted.

Injury and Illness

Injury and illness are common during adventure races. Many are minor, and relatively few result in withdrawal from an event.[4,46,51] Most injuries involve skin and soft tissues.[4,14,51] In a survey of 223 adventure race athletes, 73% reported at least one injury (acute or chronic) during the 18 months before the survey.[14] This is comparable with overall (acute and chronic) injuries reported during orienteering and standard triathlons, although lower than in ultra-endurance triathlons, in which injury rates have been reported as high as 90%. In the survey, 44% of advanced, 35% of intermediate, and 19% of beginner racers reported acute injuries. Chronic injuries were more common, reported in 59%, 54%, and 56% of athletes, respectively.[14] Acute injuries are considerably more common during adventure races when compared with triathlons. Some of the difference has been attributed to lower extremity injuries sustained during orienteering over unstable terrain and the inherent risks of mountain biking. In the survey, the most common site of acute injury was the ankle, followed by the arm and shoulder, the knee, and the lower back.[14]

In a prospective cohort study of injuries and illness treated during the Primal Quest expedition-length adventure race held in Telluride, Colorado in 2002, 243 medical encounters and 302 distinct injuries and illnesses were reported among the 248 athletes who participated. Of the 179 (59%) injuries and 123 (41%) illnesses reported, skin and soft tissue injuries were the most common (48%), with blisters on the feet representing the single most frequent reason to utilize on-site medical resources (32.8%).[51] A complete list of injuries and illness by type, number, and frequency is given in Table 91-1.

Although injury was more common overall, illness resulted in more medical withdrawals from the event. Of the 28 athletes who withdrew for medical reasons, 60% withdrew because of illness. Respiratory illness, including upper respiratory infection (URI), bronchitis, and reactive airway disease–asthma, were the most common medical reasons to withdraw (32.1%), followed by dehydration (25.0%), altitude illness (14.3%), skin and soft tissue problems (14.3%), orthopedic problems (10.7%), and genitourinary problems (3.6%).[4] In a similar study, the only medical withdrawal from a 2-day wilderness multisport event was due to reactive airway disease–asthma in the setting of a viral respiratory infection.[4]

Reactive airway disease–asthma appears to be a common cause of illness during adventure races. The relatively high incidence observed may be partially explained by changes in respiratory function in adventure race participants. This has been demonstrated by several investigations. In one study of two consecutive years of a wilderness multisport endurance event, measurements of oxygen saturation (Sao_2), forced expiratory volume in 1 second (FEV_1), and forced vital capacity (FVC) were taken before and after the event and at 45-minute intervals during the event. During the event, there was a progressive decline in both FEV_1 and FVC from the normal baseline observed at the start. At the finish, the FEV_1 had declined 25% and 22% and FVC had declined 14% and 22%, respectively, for the 2 years. Changes in Sao_2 were less dramatic.[37] In a similar study of 25 adventure race athletes, a mean decrease in FEV_1 of 15.1% and FVC of 13.0% was observed between the start and finish of the

event. Fourteen (56%) of these athletes had a decrease in FEV_1 and FVC of more than 10%, and 7 (28%) had a decrease of more than 20%.[36] Decline in respiratory function has been described in other endurance athletes but to a lesser degree.[21,27,28] Possible explanations for these findings include bronchospasm, airway inflammation, muscle fatigue, and pulmonary edema.

Complicating the understanding of reactive airway disease–asthma observed during adventure races is the fact that many events have sections that occur at altitude. In an investigation of the 2002 Primal Quest, the incidence of altitude illness requiring medical treatment was 14.1%. Most cases were acute mountain sickness (AMS) (13.3%), and only 0.81% of cases were high-altitude pulmonary edema, but the role of altitude illness in exacerbating or alleviating other respiratory problems remains.[46]

Previous studies have demonstrated improvement in asthma at high altitude. Explanations offered for this observation include a reduction in pollen and pollution and the increase in cortisol levels at high altitude.[1,6] Other studies have found that exercise at altitude may exacerbate AMS.[34] Further studies are necessary to fully understand the interaction between reactive airway disease–asthma, respiratory tract infections, and altitude illness during adventure races.

In addition, injury and illness due to environmental exposure are common during adventure races. A study of Australia's Winter Classic, a 2-day event, found that 21% of participants developed symptoms consistent with exposure.[4] During the Primal Quest, 25% of medical withdrawals were attributed to dehydration or heat illness.[51]

Endemic Disease

Another potential source of illness during adventure races is endemic disease. The literature includes descriptions of everything from isolated cases to widespread infections affecting large numbers of participants.

In the 1997 Raid Gauloises held in Lesotho and Natal, South Africa, 13 French athletes were diagnosed with African tick-bite fever. This rickettsial disease is caused by *Rickettsia africae* and transmitted by *Amblyomma* ticks. Signs and symptoms included fever, headache, multiple inoculation scars, regional lymphadenopathy, and rash.[15]

The most widely publicized outbreak was of leptospirosis during the Eco-Challenge-Sabah 2000 adventure race held in Malaysian Borneo.[9,10,39,44] Of the 304 athletes competing, 189 were contacted, of which 80 met the case definition for leptospirosis. No deaths were reported. Risk factors identified were kayaking or swimming in the Segama River, swallowing water from the Segama River, and spelunking (caving). Swimming in the Segama River was the only risk factor independently associated with illness. Of the 20 athletes who reported taking doxycycline for prophylaxis of malaria or leptospirosis, 4 (20%) developed symptoms of leptospirosis. When this rate was compared with those not taking doxycycline and adjusted for exposure, doxycycline use was determined to be protective; however, the difference was not significant.[39] Leptospirosis infections have also been attributed to participation in an adventure race in Guam, triathlons in Illinois and Wisconsin, and white-water rafting in Costa Rica.[11,12,20]

Leptospirosis is a bacterial zoonotic infection associated with exposure to water or soil that has been contaminated by wild and domestic animals, which serve as a reservoir and transmit the infection by shedding the organism in their urine. The illness is characterized by symptoms including fever, chills, myalgias, headache, conjunctival suffusion, abdominal pain, vomiting, diarrhea, and rash. This may progress to aseptic meningitis, jaundice, renal failure, hepatic failure, and hemorrhage. The syndrome of fever, meningismus, and renal and hepatic failure is referred to as *Weil's disease*. The incubation period is most commonly 5 to 14 days but ranges from 2 to 30 days. Mild infections can be treated with tetracycline and severe cases with intravenous penicillin.

Myiasis, an infection of humans by fly larvae, has also been reported during an adventure race. In 2001, during a race in the Para jungle region of Brazil, an athlete fell and sustained a wound that was infected with a third-stage larva of the New World screwworm fly, *Cochliomyia hominivorax*. Although most cases of myiasis are generally benign, infection by invasive species like the screwworm may result in extensive tissue damage, pain, and even death because the larvae have powerful oral hooks that can invade cartilage and bone.[40]

Adventure races are held in a wide range of locations and a variety of conditions. Given the potential for participants to become ill during or after the event, it is essential that the medical director be familiar with endemic diseases for a particular event given its location, the time of year, and the current environmental conditions.

Major Trauma

Major trauma has occurred during adventure races, resulting in significant injuries and death.[24] Fortunately, the incidence is low, but owing to the potential morbidity and mortality, the medical support plan should include equipment, supplies, and personnel to provide initial stabilization and treatment of major trauma. It should also include a protocol for evacuation and transport developed in close conjunction with local EMS and SAR. Obtaining the appropriate personnel and equipment may require a significant financial commitment by the race organization. The medical director must emphasize the importance of these resources and insist on them even though they may go unused. Examples include full-time paramedic-staffed ALS ambulances, helicopters, and other rescue vehicles. Even litters and Gamow chambers can represent a significant expense for some events. The medical support plan should outline the location of these limited resources and their movement during the event. For example, it may be necessary to "leapfrog" ambulances from one location to another during the event to use them most effectively. This outline should be done before the start of the event.

Through careful and thorough review of the course, it is possible to make some estimate of the type and amount of environmental injury and illness that will require treatment during the event. For example, if the course requires travel at high altitude, the medical support plan should include a protocol for the treatment of altitude illness. This might include indications for oxygen, medications such as dexamethasone, and the use and location of the Gamow chamber.

Even with the best planning, the wide variety and variability of adventure races make it impossible to precisely predict the injury and illness pattern for any one event. In the author's experience, there may even be significant differences in one event from year to year. During the 2002 Primal Quest, a large

Figure 91-3. Foot-related problems, particularly blisters, are one of the most common reasons to seek medical care during wilderness and endurance events. (Photograph courtesy of Tim Holstrom.)

number of participants developed shortness of breath and wheezing during the event, requiring treatment with β agonists even though only a few athletes reported a history of exercise-inducted asthma. In contrast, during the 2003 Primal Quest, very few athletes required the use of β agonists; however, poison ivy on a section of the course left prednisone in short supply.

Foot Care

Foot-related problems, particularly blisters, are one of the most common, if not the single most common, reason to seek medical care during an adventure race (Fig. 91-3). In one investigation, blisters on the feet were the single most common reason, accounting for 53% of all medical encounters.[51]

Staffing and supplies should reflect the high incidence of foot-related problems. Ideally, the medical team should include providers with expertise in foot care. At a minimum, all providers should have some basic understanding of foot and blister care.

There seem to be as many different techniques for the prevention and treatment of blisters as there are feet. Athletes use everything including duct tape, antiperspirants, petroleum jelly, and multiple newer products designed specifically for blister prevention. In one text dedicated to the subject, the author lists 159 ways to prevent blisters.[53] Despite the variety of preventive methods, susceptibility to blisters will vary. Although some athletes will remain blister free with little effort, others will suffer from blisters no matter what measures are taken. The best method will depend greatly on the individual.

The treatment of blisters begins before they form. All blisters begin as "hot spots" that should be treated with tape or any one of the many blister products on the market. If the athlete is no longer continuing in the event, blisters need not be drained unless they are infected. If the athlete plans to continue, however, it may be necessary to drain the blister to allow application of a dressing that will stay in place. The blister can be drained with any sterile sharp object such as a needle, blade, scissors, or nail clippers. Ideally, a small hole is made in the blister to drain the fluid and the overlying dead skin is left in place. After the area has been cleaned, it should be dressed with tape or a blister care product. Blisters should be monitored closely for infection.

Equipment, Supplies, and Personnel

The medical support plan should include a list of all medical supplies. The list should be available at each medical station. In this way, personnel are not looking for items that are not available, and an inventory can be maintained. A sample supply list is shown in Table 91-2. Obviously, substitutions, adjustments, additions, and subtractions should be made based on the individual event. It is useful to have all supplies packaged in durable carts, with the location of supplies in each cart being identical (Fig. 91-4). Personnel can then move from one medical station to another and easily locate supplies. Supplies should be packaged in clear plastic bags and labeled (with an index card in the bag) to aid in organization, especially when supply carts are moved over rough terrain from one point to another on the course. Some adjustment should be made in the *amount* of supplies at each medical station. For example, foot care supplies will be in high demand after a long trek but less so after a kayaking section of the course.

On-site medical personnel should be able to recognize and initiate treatment for routine injury and illness, major and minor trauma, environmental conditions, and endemic diseases. Ideally, they should have experience in wilderness and event medicine. Essential skills include patient assessment, establishing intravenous access, administration of fluids and medications, and packaging of patients for transfer or evacuation. Personnel might include physicians, paramedics, emergency medical technicians, physician's assistants, nurse practitioners, and wilderness first responders. At a minimum, staffing of every medical station should include an individual or individuals with excellent patient assessment skills and the ability to establish intravenous access. Because of the high incidence of foot problems among race participants, the author has found it extremely useful to include personnel with expertise in foot care whenever possible.

Medical care is administered at medical stations located along the course at "transition areas" where racers change from one discipline to another and at designated checkpoints where it is anticipated that medical care may be required. As an example, in a 400-mile long race, there may be about 10 medical stations.

Personnel and equipment should also include full-time, on-site ALS-staffed ambulance and helicopter support. As discussed, this may represent a major expense for event management, yet every effort should be made to have an ALS ambulance on site, especially in remote locations where local EMS response may be prolonged. If a medically equipped helicopter is not available, one option is to have the helicopter that is used for general transportation and media also serve to evacuate and transport ill and injured participants.

TABLE 91-2. Suggested Medical Equipment and Supplies for Wilderness and Endurance Events

FOOT CARE
Coban self-adhesive wrap
Elastikon tape
Hypafix tape
Leukotape
Moleskin
New-Skin
GENERAL
Alcohol pad
Blanket
Cotton swab
Examination glove (S/M/L)
Flashlight/battery
Hand cleaner/sterilizer/sanitizer
Instant cold pack
Iodine swab
Portable bed/cot
Sharp/needle box
Sphygmomanometer
Stethoscope
Syringes/needle (3 mL/22gauge/1 inch)
Tape (various sizes)
Thermometer (oral/rectal)
Tongue depressor
Trauma shears
Utility towel
INTRAVENOUS FLUIDS
Angiocatheter (18, 22 gauge)
Fluid: 9% NaCl (1 L)
Intravenous starter kit
Intravenous tubing kit

MEDICATIONS
Injection
- Diphenhydramine (Benadryl)
- D50 (dextrose in water)
- Epinephrine
- Ketorolac
- Lidocaine (1%–2%)
- Promethazine

Inhaled
- Albuterol

Oral
- Acetaminophen
- Antihistamine
- Hurricaine gel
- Nonsteroidal anti-inflammatory drugs
- Antidiarrhea drugs
- Prednisone
- Pseudoephedrine (Sudafed); use with caution

Topical
- Antibiotic or antiseptic ointment
- 2% lidocaine jelly (Xylocaine)
- Hydrocortisone cream
- Ophthalmic antibiotic

MISCELLANEOUS
- Duct tape
- Fans/water sprayer
- Foley catheter
- Nasal pack (packing)
- Pregnancy test kit

ORTHOPEDIC
Elastic bandage
Cardboard splint
Finger splint
SAM splint
Respiratory
Ambu bag
Laryngoscope/blades
Endotracheal tubes (various sizes)
Nebulization pipe
Oxygen mask
Oxygen tank
Oxygen tank regulator
Oxygen tubing
Trauma/transport
14-gauge angiocatheter
Backboard
Cervical collar
Head immobilizer
Strap kit
WOUND
Bandage (various sizes)
Dressing (4 × 4, roll gauze)
Masks with eye shield
Needle for irrigation
Normal saline for irrigation (500 mL)
Sterile gloves
Steri-Strips
Suture kit
Suture (3.0, 6.0)
Forceps
Wound (tissue) glue

Adapted from Townes DA: Wilderness medicine: Strategies for provision of medical support for adventure racing. Sports Med 35:557–564, 2005.

Injury and Illness Classification

The medical support plan should include a protocol for notification of medical personnel of any medical emergency on the course. In the interest of simplicity, the author has found it useful to develop and employ a three-part classification system for injury and illness. Athletes use this system when notifying race and medical personnel of an emergency. A class I emergency requires no evacuation, and the athlete will proceed to the closest medical station for evaluation and treatment. A class II emergency requires evacuation but is not life-threatening, such as an injured ankle. A class III emergency requires immediate evacuation, as in the case of a head injury.

In the event of a medical emergency, athletes are instructed to first notify race personnel of their team number and the emergency classification number. The purpose of this system is to allow athletes to quickly communicate the severity of the situation to medical personnel. The need to relay information quickly and accurately is important because communication modalities may be unreliable or inconsistent. Athletes can then describe the situation in more detail if necessary.

EMS and SAR

As part of the medical support plan, it is essential to identify local ground and air ambulance services, search and rescue services, care facilities, and hospitals and trauma centers (and their capabilities) for each location along the course (Fig. 91-5). Expedition-length adventure races may cover multiple hospital, trauma center, and EMS and SAR jurisdictions. At each medical station, there should be a folder containing the location and contact information for these resources specific to that medical station. It should also include the location and description of the medical station that will be readily understood by local providers. For example, when requesting a local ambulance, it will be more efficient to describe "Spring Valley Camp Ground off of county road 14" rather than "medical tent 5." In this way, any medical personnel, even those without local knowledge, are able to quickly and accurately request the services they need and communicate accurately with them. It is also important to determine whether there is a national network such as "911" and whether it can by accessed with an out-of-area cellular phone. The importance of working with local providers cannot be overemphasized. It is strongly recommended that a member of the local EMS community act as a liaison between the race and the local medical community. This individual is often helpful in determining what EMS, SAR service, or care facility should be utilized.

Logistics

One of the biggest challenges in providing medical support for adventure races is the logistics of the location of equipment and supplies and the scheduling of personnel. Because medical sta-

Figure 91-4. Medical supplies should be packed in durable carts that can be easily moved from one location to another. (Photograph courtesy of Dr. David Townes.)

tions will open as the fastest teams reach them and remain open until the slowest teams have passed, this is a very dynamic process. It is impossible to predict the exact timing before the event and is thus important to build enough flexibility into the schedule of personnel and equipment to allow for this uncertainty.

Communication

Another important challenge in providing medical support for adventure races is communication among athletes, race and medical staff, and EMS and SAR personnel. Communication is especially important between race participants and medical personnel in the event of an emergency. Given the rugged terrain and remote locations in which these events take place, communication can be difficult and unreliable.

There are a variety of communication options, including cellular phones, satellite phones, and radios. Each has advantages and disadvantages, and the best modality will depend on the individual situation. The advantages of cellular phones include widespread availability and direct, private, person-to-person communication. Cellular phones are limited by network availability, making them useless in many situations. Satellite phones offer better coverage but are expensive, relatively large, heavy, and unreliable under certain conditions. They require the caller

Figure 91-5. As part of the medical support plan, it is essential to identify local ground and air ambulance services, search and rescue services, care facilities, and hospitals and trauma centers and their capabilities, for each location along the course. (Photograph courtesy of Dr. David Townes.)

to remain in one place with an unobstructed view of the sky to minimize the chance of dropping the call. Radios are usually readily available and offer reliable coverage in a variety of conditions. One disadvantage of radios is the need for battery-dependent repeaters to cover large areas. A second disadvantage particularly important to medical personnel is the lack of privacy of radio communication.

Global Positioning System (GPS) devices have been used to locate teams along the course during several expedition-length adventure races. Teams competing in the Primal Quest carried units that combined a GPS tracking unit with either a radio or satellite phone (Competitio Inc., Montreal, Quebec, Canada). In an emergency, teams notify race officials by radio or phone. Through computerized GPS tracking, their exact location can be determined almost immediately. In addition to emergency situations, this tracking system is used to follow the progress of the event.

The best communication system will depend on the individual event but may utilize multiple modalities such as cellular phones or radios as a primary modality with satellite phone backup. GPS tracking of teams has become an important part of the communication system during the race.

PENALTIES FOR ACCEPTANCE OF MEDICAL CARE

In the provision of medical support for adventure races, one area of controversy involves penalties for acceptance of medical support during the event. The administration of intravenous fluid (IVF) is a commonly cited example. If the acceptance of IVF results in disqualification, athletes may push themselves too hard in an attempt to remain in the event. If IVF is administered without penalty, racers may request it at every opportunity to gain a competitive advantage. The author and several race directors of both category III and IV events utilize an "IVF

Box 91-1. Intravenous Fluid Rule Used during the Primal Quest (2002–2004)

- Athletes who receive intravenous fluid (IVF) are automatically penalized 4 hours. The penalty period begins with the completion of the last liter of fluid.
- Athletes requiring more than 2 L of IVF at one time (one medical station) or any amount of IVF at more than one time (multiple medical stations) will be automatically disqualified from the event.
- All athletes who require IVF must be evaluated and medically cleared by the race medical director or his/her designee before returning to the race. Return to the race will occur only after the 4-hour penalty has been served.

From Townes DA: Medical Support Plan: Primal Quest 2004. Unpublished.

rule" intended to allow fair and safe competition. This should be tailored for each individual event. For example, the IVF rule used during the Primal Quest expedition-length adventure race is shown in Box 91-1. As more events are held and investigations undertaken, this may be refined.

MEDICAL DISQUALIFICATION

A potential conflict arises when the medical team and an athlete disagree about the athlete's ability to continue to race safely. In adventure races, one strategy the author has found helpful in this situation is to explain the concerns to the other seven members of the team (the four crew members and three remaining racers). They will often help convince their ill or injured teammate to withdraw from the race. In certain cases, these individuals agree that their teammate cannot continue but are unable or unwilling to convince him or her to stop. In these cases, the team may look to the medical director for help. In the end, the medical director for the event should have the final say on whether an athlete is able to continue the race. This should be clearly explained to all race participants.

Adventure races and other category IV events represent a new and exciting area of event and wilderness medicine. As the provision of medical support for these activities continues to evolve, guidelines can be developed and refined. Many of the basic principals discussed are applicable to other endurance events. Some of the unique aspects of the provision of medical support for category III endurance events are discussed in the following sections.

CYCLING EVENTS

Endurance cycling events are often noncompetitive group rides or events designed to raise money for charity. These are normally staged, multiday events with participants riding during the day and resting at night. Several investigations have demonstrated that those in charge of medical provision for these rides must be prepared to treat a wide variety of injury and illness. Because many of these events are designed to raise money for charity, the general health and experience of the participants may be more varied than in other endurance events.

The most common reasons to utilize medical care during cycling events include dehydration, heat illness, and soft tissue injuries. During the 1996 California AIDS Ride, heat-related illness accounted for 31% of all encounters, followed by pulmonary complaints (12%) and orthopedic problems (9%). During the 2001 Midwest AIDS Ride, dehydration accounted for 35% of encounters, followed by orthopedic problems (27%), skin and soft tissue problems (10%), ophthalmologic problems (6%), gastrointestinal problems (6%), respiratory problems (4%), head and neck problems (3%), allergic reactions (3%), cardiac problems (2%), psychiatric problems (<1%), and other problems (4%). The most common orthopedic problem was overuse injury of the knee primarily due to iliotibial band (ITB) syndrome.[17,50] Fortunately, major trauma is rare. This is likely in part because of mandatory helmet use and the protection from traffic that these events provide.

Because a large percentage of injuries sustained during endurance cycling events are overuse injuries resulting from inadequate training or conditioning, it may prove useful to send educational material directed at proper training techniques to the participants well in advance of the event.

MARATHONS AND TRIATHLONS

Marathons and triathlons are category III endurance events. They are the most popular endurance events in the world today. Standard marathons are 26.2 miles (42 km) long, whereas ultramarathons may be 100 miles (160 km) or more. Triathlons involve swimming, cycling, and running. Races range from sprint length (a quarter-mile to half-mile [400- to 800-meter] swim, 10- to 15-mile [16- to 24-km] bike, 3.1-mile [5-km] run) to international or Olympic length (0.9-mile [1500-meter] swim, 24- to 27-mile [38- to 43-km] bike, 6-mile [10-km] run) to Ironman or ultra-triathlon (2.4-mile [4-km] swim, 112-mile [180-km] bike, 26.2-mile [34-km] run) that may last tens of hours, or days in the case of staged races.

The overall injury rate for triathlons has been calculated at 13% to 25% of participants.[25] Each of the three disciplines is associated with a fairly unique set of injuries.

The swimming section has the greatest potential for mortality. It is essential to have sufficient rescue boats with qualified personnel in the water. Fortunately, the swimming section is associated with the fewest problems, accounting for only 3% of medical visits in one study.[25] Next to drowning, hypothermia is perhaps the greatest risk during the swimming section. The mandatory use of wetsuits may reduce its incidence. Blunt trauma from kicking and corneal irritation or abrasion from trauma, goggles, and defogging solutions are not uncommon. In addition, man-of-war stings, sea urchin punctures, coral lacerations, and aspiration have all been reported.[44] Leptospirosis has been described in triathlon participants as well.[11]

The biking section is associated with trauma, including fractures, sprains, and abrasions or "road rash." Biking accounts for 7% to 10% of injuries.[25] The occurrence of significant injury is reduced by not allowing drafting and by mandatory helmet use.

The running section, at the end of the triathlon, it is associated with the largest demand for medical assistance. In fact, 75% of medical care is delivered in the last 8 hours of the event, with a full 75% directly at the finish. Many of these injuries and illnesses are due to dehydration and electrolyte abnormalities.[25]

Figure 91-6. Dehydration and hyponatremia are common in wilderness and endurance events. (Photograph courtesy of Tim Holstrom.)

In terms of staffing for triathlons, some general recommendations include having 2.5 physicians and 7.5 nurses or other medical personnel for every 100 competitors. Although utilization rates have been reported as low as 2% to 5%, the medical team should be prepared to treat about 15% of the field.[25]

In a 12-year study of the Twin Cities Marathon in Minneapolis, 90% of medical encounters occurred at the finish line. The most common reason was exercise-associated collapse (59%), followed by skin problems (21%), musculoskeletal problems (17%), and other medical problems (3%).[35] The mechanism of collapse at the end of a marathon may be due to postural hypotension from inadequate venous return secondary to decreased skeletal muscle massage on stopping.[32] Sudden cardiac death has been reported during a marathon but is exceedingly rare.[29] About half of these cases are in individuals with cardiac disease.[13] Heat illnesses, including heat exhaustion and heat stroke, occur especially in warmer climates with participants who are not properly acclimatized. Heat illness is discussed elsewhere in the text.

For marathons, it has been recommended that an aid station be placed every 2.5 miles (approx. 4000 meters), with four roving vehicles on the course.[19]

Dehydration and Hyponatremia

Dehydration and hyponatremia are common during marathons and triathlons, and members of the medical team should be familiar with their recognition, diagnosis, and treatment (Fig. 91-6). During the Hawaii Ironman Triathlon, dehydration was the most common reason to receive on-site medical care and hyponatremia the most common electrolyte disturbance. Dehydration with hyponatremia is rare in races lasting less than 4 hours but becomes more common in races lasting longer than 8 hours.[22]

Hyponatremia is defined as serum sodium of less than 135 mEq/L associated with malaise, disorientation, hyperreflexia, nausea, and fatigue. More severe cases may result in seizures, stupor, coma, and death. Seizures have been reported in a triathlete with serum sodium of 116 mEq/L and rapid neurologic deterioration and encephalopathy in ultra-marathon runners.[18,43,45]

Hyponatremia occurs when free water intake exceeds free water loss. In the endurance athlete, this is thought to occur in two distinct ways, although there is some debate about the exact etiology of exercise-associated hyponatremia. Several authors have suggested that hyponatremia is a result of net water gain during exercise secondarily associated with sodium loss through sweat.[22] Others have attributed it so salt depletion due to massive sweat losses associated with net dehydration.[22] Additional studies have demonstrated that in fact, hyponatremia occurs both in the setting of *dehydration* and *overhydration*.[42]

In a study of 605 participants in the New Zealand triathlon, 58 (18%) were found to be hyponatremic. Of these, 18 were symptomatic, and 11 were severely hyponatremic, defined as having serum sodium of less than 130 mmol/L. Postrace serum sodium concentration was inversely related to weight change during the event. That is, athletes with hypernatremia were dehydrated, but athletes with *severe* hyponatremia were generally overhydrated. The relationship between *mild* hyponatremia and hydration status was less clear. Some athletes with mild hyponatremia were dehydrated, whereas others were overhydrated. It would appear that the mechanisms for mild hyponatremia in the endurance athlete include fluid overload, large salt losses through sweat, or a combination of the two.[42]

Whatever the mechanism, it is important to be aware of both dehydration and hyponatremia in participants of triathlons. Recommendations to help reduce the incidence of dehydration and hyponatremia include the following: (1) athletes should have 0.5 L of fluid intake for each pound lost during an event, (2) during races lasting more than 4 hours, athletes should use some form of sodium replacement (1 g/hr), (3) athletes from cooler climates should give themselves a week to acclimatize and should increase salt intake by 10 to 25 g/day, and (4) IVF therapy for races longer than 4 hours should be 5% dextrose in normal saline, and for races less than four hours either 5% dextrose in normal saline (D-5 NSS) or 5% dextrose in $^1/_2$ normal saline.[22] Studies of marathon runners have suggested that replacement of sodium at a rate of 20 mEq/hr and potassium at 8 mEq/hr will maintain normal blood levels.[31]

LEGAL CONSIDERATIONS

An important area of consideration in the development of a medical support plan for any event concerns licensing, liability, and insurance coverage for medical personnel. There will be variability in the necessary requirements depending on the location. Because events are held throughout the world, no single set of standards is applicable to each situation. It is essential to understand and adhere to the requirements of the specific location, and it may be beneficial for the medial director and race organization to solicit expert legal advice in this matter.

In addition to basic issues of licensing and liability, several interesting questions should be considered. Is the liability different for a volunteer compared with someone who is paid? If medical personnel are covered under the general liability policy of the event, do they require additional insurance? What is the validity of liability waivers signed by race participants? What constitutes the practice of medicine? For instance, does a volunteer who applies a dressing to a blister need to be licensed to do so? Do all medical personnel need to be licensed, or just the medical director? If narcotics are included in the medical kits,

what additional measures are required? The answers to these questions will depend on the laws that govern the location of the event and are beyond the scope of this text.

CONCLUSION

Wilderness and endurance events continue to push the limits of human performance. As a relatively new area of event and wilderness medicine, the provision of medical support for these events is both exciting and challenging. Although there is limited information to assist in this task, there is a growing body of literature dedicated to this endeavor. It is the hope that this growth will continue and more formal guidelines can be developed to ensure the health and safety of these athletes and thus the success of future events.

The references for this chapter can be found on the accompanying DVD-ROM.

Wilderness Medicine Education

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James R. Liffrig

NATURE OF THE PROBLEM

The features of wilderness medicine that make it so attractive to an ever-increasing number of health care professionals also present challenges to its educational programs. The need for special attention to the learning process in the discipline has been called for in the peer-reviewed literature since the early 1990s.[1,2,8,37] Some work has been done at the individual program level to incorporate evidence supporting modern concepts of adult education in health care. However, there are no universally accepted standards for the delivery of education or assessment of outcomes unique to this diverse discipline.

The spectrum of learners in wilderness medicine closely mirrors the types of experiences to which the discipline applies.[51] On one end of the spectrum are lay public who seek to acquire knowledge on basic first aid or exotic travel for reasons of safety or security. Next are those seeking formal search and rescue medical training, including conventional emergency medical technicians (EMTs) and paramedic training tailored to the wilderness setting. The next level is represented by expeditionary advisory and emergency care provided in isolated circumstances by multiple types of providers, including EMTs, nurses, and physicians. Finally, there are technically oriented researchers and other professionals who seek topic- or environment-specific experience and fellowship with others who share their interest.

In 1991, the Wilderness Medical Society (WMS) proposed a model for developing curricula to teach Wilderness Prehospital Emergency Care (WPHEC).[51] It was hoped that this would stimulate movement toward a standard that would elevate the quality of this type of training. In this work, the WMS noted the differences between wilderness and urban prehospital emergency care (PHEC). These lie at the heart of what makes wilderness medicine education unique. They are summarized as follows:

- Harsh environments greatly influence decisions on treatment and resourcing. This links basic survival needs to clinical care.
- Prolonged evacuation transport times of victims to definitive care are common.
- Uncommonly encountered injuries and clinical syndromes are due to exposure to extreme environments.
- Common illness and injuries require a different approach when complicated by austere circumstances.
- Advanced medical techniques may be required owing to the nature of the injury or illness or prolonged transport time.
- No immediate or reliable remote contact with a controlling physician in a definitive care setting is available.
- Reliance on improvisation of equipment is typical.
- Expectations for successfully managing serious injury or illness are tempered by the circumstances.
- Standard urban protocols may be unrealistic or hazardous to caregivers.

The evolution of traditional models of adult education in health care has had a positive effect. The models incorporate widely varied teaching techniques that stimulate adults to learn difficult material at the deepest levels. Wilderness medicine

what additional measures are required? The answers to these questions will depend on the laws that govern the location of the event and are beyond the scope of this text.

CONCLUSION

Wilderness and endurance events continue to push the limits of human performance. As a relatively new area of event and wilderness medicine, the provision of medical support for these events is both exciting and challenging. Although there is limited information to assist in this task, there is a growing body of literature dedicated to this endeavor. It is the hope that this growth will continue and more formal guidelines can be developed to ensure the health and safety of these athletes and thus the success of future events.

The references for this chapter can be found on the accompanying DVD-ROM.

Wilderness Medicine Education

92

James R. Liffrig

NATURE OF THE PROBLEM

The features of wilderness medicine that make it so attractive to an ever-increasing number of health care professionals also present challenges to its educational programs. The need for special attention to the learning process in the discipline has been called for in the peer-reviewed literature since the early 1990s.[1,2,8,37] Some work has been done at the individual program level to incorporate evidence supporting modern concepts of adult education in health care. However, there are no universally accepted standards for the delivery of education or assessment of outcomes unique to this diverse discipline.

The spectrum of learners in wilderness medicine closely mirrors the types of experiences to which the discipline applies.[51] On one end of the spectrum are lay public who seek to acquire knowledge on basic first aid or exotic travel for reasons of safety or security. Next are those seeking formal search and rescue medical training, including conventional emergency medical technicians (EMTs) and paramedic training tailored to the wilderness setting. The next level is represented by expeditionary advisory and emergency care provided in isolated circumstances by multiple types of providers, including EMTs, nurses, and physicians. Finally, there are technically oriented researchers and other professionals who seek topic- or environment-specific experience and fellowship with others who share their interest.

In 1991, the Wilderness Medical Society (WMS) proposed a model for developing curricula to teach Wilderness Prehospital Emergency Care (WPHEC).[51] It was hoped that this would stimulate movement toward a standard that would elevate the quality of this type of training. In this work, the WMS noted the differences between wilderness and urban prehospital emergency care (PHEC). These lie at the heart of what makes wilderness medicine education unique. They are summarized as follows:

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The evolution of traditional models of adult education in health care has had a positive effect. The models incorporate widely varied teaching techniques that stimulate adults to learn difficult material at the deepest levels. Wilderness medicine

challenges these models to deliver even more. The diverse nature of both teachers and learners adds a new dimension. Traditional medical education fosters the development of individualistic attitudes rather than members who routinely serve the needs of the group. Many wilderness medicine skills are not easy to learn and retain. They blend technical difficulty with the need for immediate recall and accuracy of application under adverse conditions with limited resources. Training often does not closely approximate the true impact of extreme surroundings on emergent or urgent care rendered in the wilderness.

A unique aspect of wilderness medicine is the close relationship of the often-harsh environment to maintenance of the knowledge base. Wilderness medicine practitioners are typically action-oriented professionals who prefer to learn and practice their craft in the outdoors. This speaks to the very nature of wilderness medicine. However, despite this link, there is a need to separate preparation and conditioning for the environment from education in order to facilitate the learning process.[1] Logistics and varying skill levels of learners make it unreasonable to carry out full practical exercises for every wilderness medicine topic in every venue at which they are taught.

Health care education serves independent groups that are homogeneous in their levels of education and learning experience. Student groups such as nurses, doctors, EMTs, and physician assistants have independent educational programs. Their professional preparation is generally addressed using subsets of educational techniques with scant cross-pollination. Once fully certified in their fields, they carry these differences forth as learners and teachers in the continuing medical education (CME) setting. It is from this educational melting pot that wilderness medicine draws its teachers. In wilderness medicine, the mixing of groups of learners is inevitable. They represent different learning styles and all levels of health care experience and certification. The growing international attention given to wilderness medicine as an academic discipline adds another dimension. Compounding the problem of diverse learners are the varied educational backgrounds and teaching styles of wilderness medicine educators. The process that prepares someone for a career as a pulmonologist who does high-altitude research produces different cognitive and practical skills than do mountaineering and scuba diving. The skill set required to effectively teach any of those highly technical fields is different still, albeit just as important. The logistics of career management versus discretionary time make the blending of these factors into the consummate wilderness medicine educator a rare event. This leads to a pool of teachers whose credentials to teach are derived largely from their practical experience or notoriety, rather than their having acquired specific training in adult education.

One might presume that the advanced educational levels of students might make the wilderness medicine educator's job easier. Except for the truly passive attendees to whom effective learning is less important than the setting and experience, this proves not to be the case. Learners expect to receive exciting, well-taught education lessons and hold educators accountable. The well-prepared educator should study and understand the students, just as an actor must know the audience.

Although there is little hard evidence to support the preference of any education or theory with this unique discipline, it is likely that established techniques have great utility in wilderness medicine. However, there remain two fundamental challenges: to determine which techniques work best for specific circumstances and how to apply them to specific types of learners; and to prepare a cadre of wilderness medicine educators formally trained to make these applications effective.

PRINCIPLES OF ADULT LEARNING

There are important differences between the education of children and adults. In modern educational parlance, these are referred to as pedagogy and androgogy, respectively. The differences go far beyond the need for adults to be directive of their own learning and for children to be directed. Because of their general clientele, the details of the various learning theories may not be as important to wilderness medicine educators as an appreciation of some of their fundamental concepts. Although most wilderness medicine education is delivered to adult learners, the keen observer may note similarities to the education of school-aged children. This speaks to the importance of attending to fundamental concepts. Much of the discipline is focused on acquiring emergent or urgent skills that are most effectively learned using basic educational models.

Basic Principles

Some basic modern academic thinking exists on adult education. The concept of proximity means that learning is enhanced and mastery achieved when new information or skill is used immediately. Classroom lectures in wilderness medicine are often necessary, but are less effective than hands-on and small-group seminars.

Learners generally prefer educational approaches that focus on concepts and principles, instead of fact-based information.[50] A concept derived from the teachings of Sir William Osler and known by nearly every classically trained physician holds that one should never spend time memorizing facts from a book at the expense of hands-on patient contact. This is why problem- and scenario-based learning has been incorporated into most modern health care education programs.

Learners respond favorably when they are able to participate in developing their own learning objectives.[50] This represents a pronounced difference between adult and child educational processes, but should be properly accounted for and leveraged. The negotiation process between student and teacher that leads to properly established objectives builds relationships and trust that are at the foundation of the adult learning process. Allowing this to occur may seem counterintuitive, especially to educators who adopt a more directive style of teaching. However, participation by learners in goal setting facilitates ownership of the process and leads to higher levels of performance.

Feedback to students may be the most important ingredient to solidify learning and complete the education cycle.[50] To be effective, this should be direct, specific, and individualized to each learner. The reasons this may not occur in health care education range from the simple logistics of managing large classes to litigation, and are often cited for failure to use this powerful educational tool.

Concepts, Theories, and Models

The Education Cycle

The notion that broad concepts of the process of learning can be described as a cycle is not new. Pattison and Mathews recently applied it to wilderness medicine while planning a

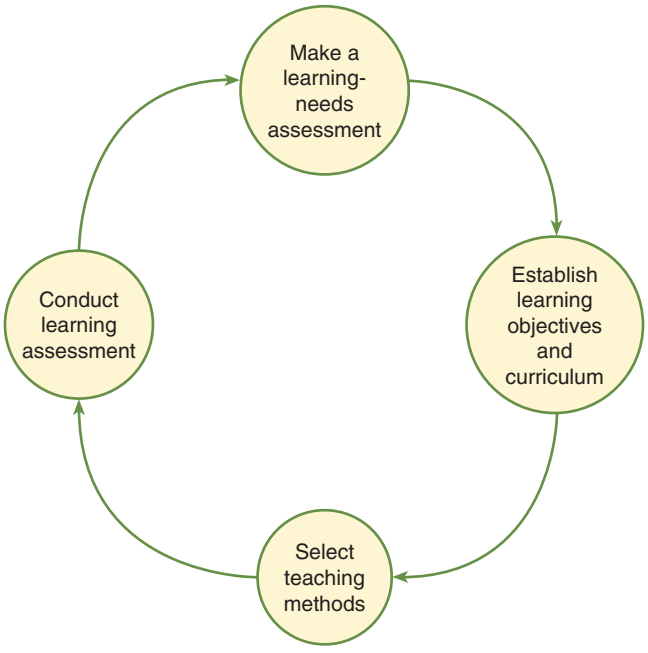

Figure 92-1. The educational cycle.

mountain medicine curriculum.[38] As depicted in Figure 92-1, the program director first makes an assessment of the needs of the learners; he or she gets to know the audience. Next is the often-underestimated task of establishing tailored and focused learning objectives. The educator then selects teaching methods and settings that best accomplish the objectives, while meeting the needs and expectations of the learners. Finally, after the experience, the educator makes an assessment to ascertain whether learning has occurred. This final step may be the most difficult and least enacted of all.

Experience-Based Learning

Kolb's model of learning is based on how individuals internalize and process learning experiences.[29] Learners perform an action, referred to as a "concrete experience." They then process the new information by "reflective observation." Next, they consider how the new information can be applied to their unique circumstances by "abstract conceptualization." Having internalized the experience, they "actively experiment" with what they have learned and apply it in new and unique ways. This model is reflected in several of the teaching and assessment techniques discussed later, especially those that address concrete skills.

Education and the Human Organism

Setting the conditions for learning can be conceptualized by using Maslow's famous explanation of how humans address fundamental needs.[34] Vella's work in popular education extends these ideas into the realm of education in the social context, but still deal with the rudiments of human nature.[47,48]

Unmet physiologic needs, such as warmth and hunger, tend to impair learning as the human organism prioritizes toward survival. Unfulfilled security needs, what Vella refers to as "safety," distract from any process that doesn't pose an immediate threat.[47] In adult education, safety issues may be subtle. Students who do not feel free to voice opinions or reveal a deficiency may be said to lack a safe learning environment. Identification with a group of learners addresses the need for belonging that is used by organized athletic teams and the military. The need for self-esteem may be met by recognition for academic achievement in front of one's peers. Finally, the highest level of Maslow's concept, self-actualization, is represented by satisfied expectations on the part of the learner. By expanding these concepts and directly addressing each in the classical hierarchical fashion, a program director can remove many obstacles to learning during the planning phase of the educational experience. A learning event that accounts for them will have a high likelihood of success and affect the student's life well beyond the experience.

Learner Sophistication

Another powerful tool available to teachers is the identification of the level of understanding, or "sophistication," of the learner. Neal Whitman proposed four levels that learners traverse as they acquire new knowledge and skills. Correctly matching teaching methods to these will improve the overall experience by saving time and increase the likelihood of fully meeting expectations.[50]

Most people begin the education journey at the level of being "unconsciously incompetent" and do not know enough about the material to even know what they do not know. After a time, they become "consciously incompetent" as they gain an appreciation for the amount and nature of the information they will have to master. Next, they become "consciously competent" as they learn how to perform the skill or use the information, but not yet with efficiency or at the level of mastery. The final stage, that of the teacher, is becoming "unconsciously competent." At this level, the learner becomes so practiced at the skill and the understanding is so well internalized that it can be performed to standard without consciously focusing on process. The learner simply knows what to do.

Principles of Androgogy

There have been numerous efforts to roll learning theories together into a concise package of tools for health care education. To this end, the work of Malcolm Knowles is widely read and often cited by education academics. He put structure to the concept of helping adults learn and called it "androgogy." He suggested that through the principles of androgogy, adult learners are most successful when they are assisted in the process rather than directed through it.[28] Knowles made five basic assumptions about adult learners from which he derived his principles. They can be summarized as follows: (1) adults tend to have internally and not externally focused motivations for learning; (2) the learning process should be related to solving real-life problems; (3) existing knowledge and experience greatly influence learning; (4) self-direction improves the learning experience; and (5) adults learn best with problem-based rather than subject-based methods. Knowles' seven principles of androgogy are summarized in Box 92-1.

Learner-Centered Education

Jane Vella applied learning theory in unique ways to the social context across different cultures. She recognized that education resides at the heart of many social issues. The teacher's message

Box 92-1. Summary of Knowles' Principles of Androgogy

1. The proper learning climate sets the stage for success.
2. Adults prefer to actively participate in and contribute to planning for methods and curricula.
3. Adults respond well to self-diagnosis of learning needs.
4. Adults prefer to self-determine learning goals and objectives.
5. Allow adults to identify their own resources for learning and formulate plans for their use.
6. It is better to support adults while they implement their own learning strategies rather than direct them through it.
7. Encourage reflective self-critique in the learning process.

Box 92-2. Summary of Learner-Centered Principles

1. Perform a learning needs assessment.
2. Provide for learners' safety.
3. Develop sound relationships to foster trust and encourage dialogue.
4. Properly sequence learning tasks from simple to complex and solo to groups.
5. Provide opportunity for praxis—performing learning tasks and reflecting on the experience.
6. Respect the learners' lives, circumstances, autonomy, ideas, opinions, and time.
7. Attend to ideas and feelings; link actions to them.
8. Provide for immediate application of new skills and knowledge.
9. Make dialogue more accessible by clarifying roles and reinforcing equity between teacher and learner.
10. Use teamwork to enhance education.
11. Engage learners directly by using small groups and open exchange of ideas.
12. Teachers and learners are accountable for what is learned. Learning must be tangible and observable.

is often lost in the delivery owing to avoidable cross-cultural and interpersonal obstacles. She maintains that the key to adult learning is clear dialogue between the teacher and learner.[47–49] Educators too often fail to establish productive dialogue and hence select ineffective teaching approaches. Vella suggested that traditional hierarchical teacher and student roles be discouraged. Teachers become facilitators. Barriers to dialogue are "addressed and eradicated." She offered to popular education a paradigm that places the learner at the center of the educational universe. Her principles link theory to practical use in a way that enables learning in challenging circumstances. Vella's principles are summarized in Box 92-2.

Learning-Oriented Teaching Model

Cate and colleagues recently published their notion of a model of teaching based on concepts from educational psychology.[5] Their proposals are meant to influence all aspects of adult education, especially those of curriculum design, teaching techniques, and teacher assessment. An attractive feature is a method to "inventory" and match teaching and learning styles to improve outcomes. The authors build a model around what they identify as the "components of learning" and the "amount of guidance" that learners need to navigate the experience. Their premise is that, because education seeks to enable people to "function independently," the process should foster self-regulation of learning. In the model, learners mature from "externally guided" learning, through "shared guidance," to self-guided or "internally guided" learning. This applies to both cognitive (what to learn) and affective (why to learn) components of learning.

Box 92-3. Kaufman's Principles to Guide Teaching Practice

1. The learner should be an active contributor to the process.
2. Learning should relate to understanding and solving real-life problems.
3. Learners' knowledge and experience are critical and must be accounted for.
4. Learners should have the opportunity to self-direct their learning.
5. Learners should have ample opportunity to practice, self-assess, and receive constructive feedback.
6. Learners should have frequent opportunity to reflect on the learning process.
7. Role models have great impact on learning.

Putting It All Together

Theories and models are meaningless without practical application. Directly applying ideas from theorists with diverse backgrounds and agendas into the realm of wilderness medicine education is difficult. Kaufman recently reviewed several adult education theories in the context of health care education.[27] He examined them for key areas of commonality and offered simple principles to "guide" medical educators as they think about and plan educational experiences (Box 92-3). Wilderness medicine educators can easily adopt these.

Educational Techniques

Lecture

Lecture is the most often used educational technique. It offers several important advantages. Large amounts of information can be delivered in relatively short periods of time. Planning is generally easier for the lecture format. It requires little logistical support and only one teacher. It works well for highly technical information that the learner will most likely have to study again in order to internalize. Lectures can be easily enhanced with audiovisual aids. All this adds up to a degree of efficiency that is highly attractive to the resource-limited educator.

The lecture technique has several equally important disadvantages. Learning is highly dependent on the delivery skill of the teacher. Because it is passive, many adult learners do not respond as well with this approach. It is generally accepted that levels of retention of material presented by pure lecture are lower than more active teaching techniques.[44] There is generally

Box 92-4. Methods to Enhance the Lecture

1. Be clear about the reasons to lecture. Communicate this to learners.
2. Research the audience's culture, attitudes, expectations, professions, and experiences.
3. Pace the presentation; attend to the 12- to 20-minute rule for limits of attention span. Break up the pace with discussion, question and answer period, or group work.
4. Personalize the presentation with examples.
5. Speak from notes, not complete scripts. Know the material!
6. Use visual aids.
7. Use critical incidents with which the learners are familiar to make teaching points and solidify learning.
8. Leave with a question or challenge to encourage learners to explore further.
9. Perform self-assessment through video, learner evaluation, etc.

limited opportunity for hands-on applications and practice. Thus, the usefulness is limited in some areas of wilderness medicine that are largely skills oriented. The more restricted the opportunity for student questioning and dialogue, the less effective this technique becomes. Students can often more effectively learn by themselves information that is presented by lecture. Brookfield discussed methods to enhance the lecture as a means to communicate information[4] (Box 92-4).

Problem-Based Learning

A highly effective trend in health care education is the use of problem-based learning (PBL). PBL can take various forms, but generally learners are presented with a problem and are guided through a structured discussion that leads to a pre-established solution. This learner-centered approach has proved popular among students and demonstrates comparable outcomes when compared with other, more traditional formats. An interesting aspect to PBL is that student-led experiences tend to be more highly favored among participants than those facilitated by faculty, yet the outcomes remain at least comparable in terms of satisfaction and examination scores.[20,43]

PBL offers great versatility to the curriculum planner. It can be used whether the focus is on acquiring pure fact-based knowledge or practical skills. It is most often applied in the small group setting and therefore may pose logistical challenges in some space- and resource-limited settings. It complements the strengths of small-group learning in that it reinforces communication and problem-solving skills, teamwork, individual responsibility for learning, and the need to share knowledge.[52] It tends to give the best results when it is structured and forces learners to use critical thinking skills. It fosters the process of analysis, organized problem solving, and decision-making using group discussion to direct and reinforce learning. The general format for problem-based learning is presented in Box 92-5.[3]

Case-Based Learning

Case-based learning (CBL) is a subtype of PBL that is extremely useful in wilderness medicine education. It makes use of several powerful aspects of theory to solidify learning. This method improves outcomes over pure lecture for certain types of material. Many of the skills-oriented topics in wilderness medicine can be easily presented through scenarios and cases. The teacher can easily modify case scenarios to suit various learner levels, styles, and objectives. It can be modified for learning experiences in the field setting because, if properly planned, it requires minimal logistical support.

Box 92-5. Steps of Problem-Based Learning

1. Presentation and evaluation of the problem
2. Generation of solutions to the problem (hypotheses)
3. Inquiry to review the hypothesis, including gathering
4. Information such as lab tests and physical exam
5. Interview information
6. Application of the information gathered to the original problem
7. Review and synthesis of what has been learned and evaluation of the process

This technique motivates the learner because it is directly relevant to the reasons for which he or she sought the experience. It offers an automatic sequence and information source for presentation because cases flow in accordance with actual or simulated events. The teacher generally facilitates discussion and assists the students with understanding and methods to organize information. Clinical problem-solving styles can easily be demonstrated.

Scenarios

Scenario-based training is familiar to PHEC educators, but less widely used in traditional medical education. It maximizes many of the strengths of newer approaches to adult education as it guides self-motivated students through a process of "discovery" of the information.[32] The main role for teachers in this format is to facilitate, not to direct, the learning process.[21] It is less important to be an expert in the clinical details of each case than in the application of problem-based learning. This method is often scripted and makes use of actors, props, and moulage to simulate real-life situations. The logistics of carrying out this aggressive training technique may be prohibitive to some programs, but the investment is worth the effort in terms of improved outcomes and retention of knowledge by learners. Nearly all organizations that train prehospital wilderness medicine practitioners use some form of complex case- or scenario-based learning program.

Subject-Based Learning

Subject-based learning (SBL) is a euphemism for traditional teacher-centered techniques of presenting information. The student in an SBL setting is the passive recipient of instruction rather than an active participant in the learning process. SBL methods typically present information by broad topic in a mainly lecture-based format. Health care curricula designed around SBL tend to be redundant because broad topics may deal with the same or similar types of information. For example, a traditional medical school first-year basic science curriculum will have a series of lectures on pharmacology, biochemistry, pathology, and microanatomy. Each will incorporate specific material on diabetes presented by different departments. The student may have to wait a year to receive a session that focuses on diabetes that incorporates elements of pharmacology, biochemistry, pathology, and microanatomy.

Although SBL has a role, most health care educators have incorporated more learner-centered techniques, such as PBL, into their curricula. The main drawback of SBL is that by favoring efficient delivery and lengthy, complex curricula, it often fails to take advantage of features in adult learning theory that are known to lead to higher rates of retention and deeper learning.

Discussion

The technique of discussion is a versatile and highly effective teaching tool rooted in behavioral science. People learn better when the information is presented in ways that challenge them to process it in more than one way. Although not as efficient as lecture in terms of the quantity of information that can be delivered, it offers advantages to the wilderness medicine educator when teaching problem-solving skills and broad concepts that can be applied to many types of scenarios. Students readily internalize material because they have to intellectually manipulate it in various ways. The information is introduced, processed, and discussed. Students modify their own notions and then formulate solutions based on their new internal constructs of the problem. Learning is guided by the facilitator-teacher and reinforced by the group members, who are all going through the same process and adopting a similar problem-solving skill set.

There are two basic modes of discussion-based learning.[30] The Socratic questioning method challenges students to identify the most important features of a specific problem and then reconstruct it using general principles that are the true focus of the discussion. Developmental discussion approaches the problem in parts. It keeps all students focused on one part at a time and takes advantage of the group setting to ensure that teaching points are addressed.

Teacher-facilitators may highlight discussion with the powerful tools of analogy, discovery, and induction to stimulate learning and ensure retention.[16] Analogy illustrates concepts by asking students to visualize using examples with which they are already familiar. The process of discovery leads students through a sequence of steps from the most basic to the more complex, in order to guide them to the final goal of deeper understanding of the principal learning objective. Induction asks students to take general lessons from specific examples or experiences, make comparisons, and draw new conclusions relevant to the learning objective. These three techniques can be applied in any setting that involves learner interaction with the teacher.

Small Groups

The selection of a teaching method is highly influenced by class size and the quantity of information that must be learned. Large classes that must digest substantial amounts of information tend to push faculty into selecting passive modes of teaching. The traditional CME conference at which hundreds of attendees review highly technical material is an example of this. However, passive, lecture-based methods are not the most effective and often not the most efficient, for all types of learning, especially in wilderness medicine. It is now widely accepted that skills-based learning presented in a small-group setting is a better way to teach practical skills.[31]

The problems inherent in teaching large groups can often be overcome by adopting a combined approach. In this strategy, the information is delivered in parts to the entire group. Learners are then broken into small groups to conduct activities that allow them to discuss it, use it, and solidify learning. There is usually a more favorable teacher-to-student ratio and greater self-direction of learning by the group.

Wilderness medicine offers ample opportunity to use the combined approach and the pure small-group venue for teaching. Both rely on the process and mechanics of group experiences. The simple act of breaking a large group into smaller groups does not constitute small-group teaching. This method requires skill and experience with group process on the part of the educator to avoid pitfalls that detract from learning. When done well, small-group learners take away solid lessons and the strong relationships that they had to build to get them. If done poorly, learners walk away dissatisfied with negative attitudes toward learning, the setting, and the discipline.

To understand group learning is to understand how individuals within the group interact.

Bruce Tuckman's original concept of the developmental sequence of small groups[45,46] should greatly influence how teachers plan and conduct these activities.[19,26] The general strategy for teachers is to become familiar with the stages, recognize their manifestations, and use a planned approach that gradually releases control of the teaching process to the group as members become more able to direct their own learning.

Group members get acquainted and become oriented to the setting during the *forming* stage. Student anxiety may hinder learning as interpersonal dynamics take shape. Students tend to adopt a passive mode of learning. They respond best during this stage to a more directive teaching style with clearly defined expectations and structured events.

During the *storming* stage, the group's identity begins to take shape among some members, whereas others continue to seek individual goals over that of the group. Dissent may be voiced about leader- or teacher-directed tasks. Teachers should demonstrate strong but patient leadership to move through this stage without alienating group members. They should openly encourage support for group-generated goals.

The group identity solidifies during the *norming* stage. Individual ownership of group goals and a greater affinity for teamwork are hallmarks. As they gain a sense of safety, members genuinely interact with other participants. Leadership tasks should be directed to group members. Learning activities, such as role-play and case discussions, become most useful.

During the *performing* stage, the teacher acts only as a resource for the group, which has developed to a point of relative autonomy. The group is able to plan and conduct its own activities as well as make self-assessments in an organized and productive fashion. Energy is spent on learning rather than on the interpersonal mechanics of the process.

The role of the teacher in a small group setting should be oriented toward facilitation of learning rather than direct delivery of material. However, the teacher remains accountable and cannot take a completely hands-off approach if the expectation is that objectives will be met. By attending to the details of process, the teacher ensures that the learners are free of this task. The teacher creates the proper learning environment and keeps the process on track. Some common mistakes made by small-group "facilitators" include[25] the teacher presents a lecture; the teacher talks too much; students do not participate unless prompted or directly questioned; the students are not adequately prepared (i.e., prereading) for the session; there are overbearing, domineering students; and participants want to be provided a quick and simple solution to the problem rather than engage in the process of group discovery.

Despite not being part of the learner group, the conduct of the teacher has great influence over the process and outcomes. A poorly prepared teacher ignores the internal dynamics of the group and sets up the experience for failure. Some techniques available to avoid these problems include[25] agreed-on rules for the conduct of sessions; clearly stated tasks and objectives; use of the rhetorical method of questioning to stimulate thinking; taking a lengthy pause after posing a question, allowing students to answer; not offering immediate solutions or guidance unless participants appear to be taking the wrong path; attending to body language and mannerisms of all participants, both when they are speaking and when they are listening; and addressing the entire group, rather than a single student, with effective mannerisms and eye contact.

The general steps in preparing for and conducting small-group teaching are summarized as follows:

- Establish the objectives or outcomes of the activity. What should the students be able to do?
- Select the specific tasks and methods to achieve the desired outcomes (i.e., case presentation with discussion or group project and presentation)
- Choose appropriate facilitators and faculty
- Select and modify the environment and group size; make group assignments
- Prepare and coordinate for training support materials
- Familiarize with individuals and dynamics of the group; identify potential problems early
- Conduct the activity; modify techniques as required to address unique needs of each group
- Assess learning; retrain as needed
- Assess methods and modify as needed for the next session or course.

Small-group learning is a rewarding and highly effective method of teaching. It requires preparation and experience. The characteristics of small-group learning make it the teaching method of choice for many wilderness medicine educational activities, especially when combined with other techniques, such as simulation, scenarios, and case presentations.

Web-Based Distance Learning

Distance learning, traditionally provided through correspondence courses, has taken an entirely new direction. With the proliferation of the Internet, more people than ever find it possible to further their education without physically entering the classroom. A little-used tool in wilderness medicine education is web-based distance learning. It has found widespread application in other academic areas, and health care educators have embraced many of its benefits.

Even courses that are not offered as complete web-based packages can be supported by limited Internet applications such as email, distribution of materials, enrollment, needs assessments, and testing. To this can be added videoconferencing, discussion boards, live topic-specific chat rooms, and presentation of live events by video streaming. Nearly any software application that can be used on a home computer can be offered in some fashion over the Internet. With the advent of high-speed connectivity and constantly changing security programs, obstacles to efficiency and learner security have been minimized. Virtual Learning Environment (VLE) software enables all of these applications to be managed efficiently while keeping the focus on the learner and not the technology.[22]

The Internet carries two main disadvantages. First is the need for learners to possess sufficient computing power to allow for rapid and efficient downloading and presentation of course materials. This may be difficult in remote locations away from an adequate Internet connection. The second drawback to web-based learning in wilderness medicine is its dependence on technology and the requisite support. Large organizations find it necessary to hire full-time support staff in order to provide comprehensive and reliable products. Many wilderness medicine educators find it difficult to dedicate the resources necessary to bring it to full implementation.

There are numerous ways that education can be delivered on-line. The two broad categories are pure distance learning and the hybrid web-based course. In a completely web-based program, all course work, materials, sessions, and assessments are transmitted over the Internet; even assistance from teachers occurs by email or live message applications. This method is a true virtual learning experience. Alternatively, many organizations offer hybrid on-line courses in which students use Internet applications as an additional tool to complete portions of the course, but are required to participate in periodic face-to-face sessions.

Many health care practitioners involve themselves in wilderness medicine because of the fellowship with other uniquely qualified and experienced professionals. As web-based applications become more widely used in wilderness medicine, educators will be increasingly challenged to ensure that learners do not feel isolated from their wilderness medicine peers and role models. The learner, not the technology, must remain at the center of the effort

Field Experiences

Medical schools and residencies are following the lead of commercial WPHEC programs and moving learners out of the classroom and into the field.[32] Rotations and electives that include field training experiences of various lengths are highly desirable. They offer direct relevance and immediate application of newly learned skills. When coupled with effective group process and feedback, the benefits of field training make this a powerful venue for skills-oriented education. These experiences are most often part of an overall curriculum that incorporates other techniques, such as lecture and PBL in a didactic setting.

Concerns include safety and security of participants as well as liability issues for the sponsoring organization. The logistical support package can be expensive and complex. The quality of the experience can be greatly influenced by uncontrollable factors, such as weather. Proper screening and selection of participants are critical to success, especially in programs conducted in remote locations or in physically demanding environments. Multiple techniques, such as scenarios and hands-on practical exercise, can be applied simultaneously to maximize the learning experience. Field training affords an opportunity to introduce nonmedical skills such as leadership, wilderness survival, and land navigation.[32] This not only generates interest but also creates well-prepared learners and an overall lower level of risk.

ASSESSING LEARNERS' NEEDS

Performing a proper assessment of the needs of students before designing a curriculum leads to a fulfilling experience for all.

Jane Vella claims that this critical step is necessary to "truly honor the time investment of the learner and create the conditions for meaningful dialogue between learner and teacher."[47] People whose motivations for learning are ignored "quickly become bored and indifferent." They often walk away from the experience dissatisfied or without completing the program. Some examples of information the wilderness medicine course planner would want to know are:

- Why do the students want to spend their valuable time learning this material?
- What are the levels of training and vocations of the students?
- Are there any specific outcomes that are more important to the group than others?
- What is the relevance of the wilderness medicine learning experience?
  - Job requirement
  - Prerequisite for another course
  - Enhance recreational activities
  - Prepare for emergencies
  - Acquire knowledge for safer exotic travel
  - Share fellowship with other wilderness medicine practitioners
  - Creative diversion
  - Academic advancement

Learner-centered education principles tell us that the needs assessment should lead to some degree of modification of course content or structure to suit the individualized needs of the learners. To become fully invested, students should be able to shape to some degree what will be taught to them. Thorough modification of curriculum content based on a formal needs assessment may be impractical for standardized certification experiences, such as Wilderness First Responder courses. Even though they may be of great value, detailed needs assessments are seldom done for wilderness medicine CME conferences, owing to great variation in the levels of professional education. Advance study of students' expectations and reasons for learning allows for more focused and tailored instruction. The needs assessment can be easily accomplished with a questionnaire delivered by postal or electronic mail, telephone contact, or face-to-face interview. The actual format is not as important as the mere act of soliciting input from students. Allowing them to actively dialogue about their learning will achieve buy-in. The curriculum design becomes overtly accountable to the students and results in much higher levels of internal motivation.

LEARNING OBJECTIVES

An area of academic wilderness medicine that is often given cursory attention is that of establishing objectives for learning activities. Many educators make an attempt, but well-written objectives are far too rare for the amount of education being delivered in the discipline.

A learning objective is a collection of words, pictures, or diagrams that tells others what the educator intends for learners to achieve.[33] When properly written, objectives establish outcomes, rather than describe process. The basic purpose of teaching is to facilitate learning that results in measurable outcomes. What students actually learn should be described in advance by objectives.[18] The basic functions of learning objectives are to:

- Enable prospective participants to determine whether the activity is appropriate for them
- Provide focus for instructors in preparing their offerings and guide selection of educational methods
- Serve as an evaluation tool to measure success of the program

Learning objectives should be tailored to a specific learning activity or session and not used as goals for an entire course. During precourse preparation, objectives should be provided to faculty to assist them in preparing their sessions and to the learners so they will know what to expect and how to prepare.

An objective is constructed by describing an activity that elaborates specific knowledge or skills that a learner will be able to demonstrate after a successful learning activity. Well-written learning objectives are measurable by written testing, observation, hands-on problem solving, or other methods of assessment. Words or phrases, such as "know, think, appreciate, learn, comprehend, remember, perceive, understand, be aware of, be familiar with, have knowledge of, grasp the significance of," are difficult to measure and are of little use when writing learning objectives in wilderness medicine.[6] Examples of learning objectives that illustrate the concepts of being specific and measurable are: The student will be able to . . .

. . . perform well during wilderness-setting role-play (not specific or measurable)
. . . carry an injured patient (specific; can be made more measurable)
. . . discriminate between edible and inedible wild plants (specific and measurable)
. . . feel more comfortable performing mass-casualty triage (neither specific nor measurable)
. . . express a point of view supported by valid evidence (specific and measurable)

Strong objectives, in addition to being performance-based, specific, and measurable, are preceded by a condition statement to set the stage and aid with measurement specificity.[33] In writing objectives, answer the following questions: "What should the participants be able to do?" "How well must they do it?" Objectives must be clear and attainable. They are often constructed in an "if . . . then" sequence to facilitate clarity. Focus on acquisition or reinforcement of a specific element of knowledge or skill.[18] Recommended styles for constructing objectives include the following:

"Given X materials and within Y minutes, the student will build a Z device strong enough to support 25 pounds."
"At the completion of this teaching activity, participants will be able to . . ."

This last phrase is followed by a specific performance verb and the desired learning outcome. Examples of performance verbs are found in Table 92-1. The following is an example of a properly prepared set of objectives[6]:

Aerospace Medicine Lecture: Fatigue, Desynchronosis, and Countermeasures
Time: 1200–1300
Speaker: Dr. Pasta Linguini
Lesson Objectives: At the conclusion of this activity, the student will:

1. Summarize the results of studies on the effects of fatigue on aircrews.
2. Give examples of the deleterious effects of fatigue on human performance and accident rate.
3. Define desynchronosis and give examples of applications of its understanding to aviation medical personnel.

TABLE 92-1. Examples of Performance Verbs for Learning Objectives

KNOWLEDGE	COMPREHENSION	APPLICATION	ANALYSIS	SYNTHESIS	EVALUATION
cite	associate	apply	analyze	arrange	appraise
count	classify	calculate	appraise	assemble	assess
define	compare	complete	contrast	collect	choose
draw	compute	demonstrate	criticize	compose	critique
identify	contrast	dramatize	debate	construct	determine
indicate	describe	employ	detect	create	estimate
list	differentiate	examine	diagram	design	evaluate
name	discuss	illustrate	differentiate	detect	judge
point	distinguish	interpret	distinguish	formulate	measure
read	explain	interpolate	experiment	generalize	rank
recite	estimate	locate	infer	integrate	rate
recognize	examine	operate	inspect	manage	recommend
relate	express	order	inventory	organize	revise
repeat	interpret	predict	question	plan	score
select	interpolate	practice	separate	prepare	select
state	locate	relate	summarize	produce	test
tabulate	predict	report		propose	
tell	report	restate			
trace	restate	review			
write	review	schedule			
	translate	sketch			
		solve			
		translate			
		use			

4. Outline various countermeasures that are available, as well as some that are currently undergoing investigation, such as melatonin.

More theoretical treatments of preparing learning objectives are based on complex notions of learning theory and behavioral psychology. They describe objectives as appealing to domains of information processing or whether objectives are based on achievement or outcomes. The details are beyond the scope of this text. To summarize, well-written learning objectives are tailored to a single learning activity or a closely related group of activities, address preidentified needs and interests of the learner, are specific to levels and types of performance, are achievable, realistic, and time specific, and use verbs that specify behaviors that can easily be measured.

ASSESSING LEARNING

The last step in the education cycle (see Figure 92-1) is making a formal assessment of whether or not learning has occurred.[38] There are several reasons for testing in wilderness medicine education beyond the obvious need to validate and certify students. Learning assessment can be a valuable extension of the learning process. Students seldom come away from any properly conducted and reviewed examination without having learned something. It can be said that this type of learning by assessment is the capstone of knowledge synthesis. Each exam completes the education cycle at a particular level and allows progression to the next level with a higher degree of competence.

Learning assessments can be grouped for convenience into two general categories: formative and summative. Formative assessments are made before and throughout a course of instruction and have both learning and testing dimensions. Feedback after formative assessment is critical to shape additional learning. This type of assessment may take the form of a diagnostic exam to assess learners' needs and establish lesson objectives, or an intermediate exam to assess progress at a particular phase of training.

Summative assessments are generally used at the end of either a critical phase of training or completion of a course. This type of assessment is more often applied for certification and validation than as an extension of learning. Final course examinations and tests that lead to state or national certification in a vocation are examples of summative assessments.

Reliability and Validity

Two key concepts to understand when designing learning assessment tools for wilderness medicine courses are reliability and validity. These are applied separately when discussing written[25] and skills-oriented[42] testing. They are particularly relevant to WPHEC courses, such as Wilderness First Responder (WFR) or Wilderness EMT (W-EMT), which rely heavily on skills-based testing.

A reliable testing instrument consistently measures the desired outcome no matter how many times it is administered. There are a host of factors that influence reliability. The length of time it takes to complete a test, characteristics of the examinees, logistical and administrative problems, and variation in examiner methods may decrease the reliability of an exam and mask learners' true level of competence. One cannot address the validity of an assessment tool until reliability is achieved.

A valid testing instrument is one that measures what it was designed to measure. Simply put, it reveals the actual level of learner competence. Apart from reliability problems, issues that negatively influence validity include cases and scenarios that are

not directly relevant to the learning objectives, poorly structured or improperly selected test questions, testing stations that do not adequately examine the skills that were taught, and failure to seek expert review of course content during the design phase. The measure of validity is done in terms of exam scores. Having removed obstacles to reliability and validity (see Evaluating the Assessment Tool, later), the educator may use scores to make a direct link from teaching methods to achieving learning objectives.

The two main approaches to learning assessment in health care that are pertinent to wilderness medicine are written and skills-based testing. One-on-one teaching with feedback is useful in other areas of health care education, such as primary nursing programs and the training of interns and residents, but it is often impractical for wilderness medicine teaching activities.

Written assessments remain the cornerstone of health care education, despite the proliferation of progressive teaching techniques such as hands-on skills development and problem-based learning. The questioning format of written assessment may take many forms, but all generally test reasoning ability and accumulation of knowledge, as opposed to practical application.

Schuwirth and van der Vleuten proposed a list of criteria to compare the advantages and disadvantages of various types of written test formats and questions[25]: reliability and validity (as discussed earlier), educational impact (how students will prepare for exams, hence learn the material), cost-effectiveness (expense in terms of money and time), and acceptability (how both students and teachers view the exam's effectiveness and relevance). To account for these factors, the most preferred method of developing written examinations is to use several types of test questions in each instrument. The types of exam questions that may be considered include: true or false, single best answer multiple choice, multiple true or false, short answer "fill in the blanks," essay, case-based key feature, and extended matching. A thorough treatment of each can be found in the education literature.

Skills-based assessment is particularly relevant to much of health care education, especially in wilderness medicine. It is the mainstay of competency testing for most prehospital courses. The two general subtypes of skills-based assessment used in wilderness medicine are skills subset testing (sessions focused on a single or a few closely related skills) and case- or scenario-based skills stations. Basic learning theory tells us that this popular testing method should be highly effective in achieving objectives and in delivering highly retained learning. This bears out in practical application. The disadvantages of skills-based assessments are centered around two areas. First, the logistical requirements necessary to conduct high-quality testing can be burdensome. Second, examiners require a high degree of skill with regard to testing process and mechanics to ensure reliability and validity of results. The general steps in conducting a skills-based learning assessment session are as follows:

- Decide whether the activity is for practice, formative (intermediate) assessment, or summative (end-of-course) assessment. Inform the learners.
- Match learning objectives to scenarios or cases in the exam.
- Select satisfactory examiners and role-players; conduct thorough briefings.
- Obtain logistical support and prepare the environment.
- Thoroughly brief the learners.
- Conduct the assessment; check progress; modify as required.
- Conduct an initial assessment of the instrument.
- Review results with learners.
- Conduct a thorough assessment of the instrument with broader comparisons.

Evaluating the Assessment Tool

Educators must evaluate their learning assessment tools to ensure reliability and validity. A couple of simple methods may be used. For written assessments, first review exam scores and compare them to attendance. It should come as no surprise that students who do not attend specific sessions would do poorly on quizzes or test questions designed to assess learning on that topic. If this level of analysis points to attendance problems, there are other issues in the course, such as objectives, content, and quality of instruction, that should be addressed before the quality of the exam.

The process of test item analysis correlates the number of examinees who missed a given question with overall test scores.[14] This can be done on two levels. The first is within the group that was being tested at a given course. The second is over a period of time among several groups that took the same exam. As with any internal assessment tool, the latter style leads to stronger conclusions, but takes longer to complete. Internal pattern analysis of test scores can reveal that either the instruction was bad or the test question was not well written. The basic principle is that if a large percentage of students received a high score on an exam but a similarly large percentage missed a particular test item, then either construction of the item or the teaching method is likely at fault. This technique loses power and specificity at lower overall exam scores among those who missed the items in question.

Skills-based learning assessments can be evaluated for quality using similarly simple analytical tools.[14] The attendance comparison mentioned previously is an obvious starting point when reviewing individual performance.

Mechanics or logistics of the testing setting that seem irrelevant to the actual demonstration of knowledge and skills may affect test scores. An examiner who did not attend precourse briefings and is not familiar with what was taught, or worse, with what is expected, poses a problem. This can be revealed by noting a large percentage of examinees doing poorly at a given skill station despite doing well at other stations designed to test related skills. Notably strong students who fail a particular station may be another warning sign. Testing stations that use role-playing with scenarios must account for the influence of the role players. Poorly briefed actors who do not follow the script can make or break an otherwise competent learner. Having made attendance comparisons and ruled out problems with the setting, examiners, and role-players, the problem is most likely the quality of teaching if many students are doing poorly at a given skill station. Retraining and retesting may be in order.

TEACHING IN WILDERNESS MEDICINE

Good Teachers

What is special about teaching wilderness medicine? What differentiates the discipline from other areas of health care educa-

tion and calls for unique attention to delivery? As mentioned before, wilderness medicine instruction is often disconnected from the environment about which it informs. Teaching the fundamentals of high altitude, great depths, extreme cold, or torrid heat and their impact on the human condition requires that the initial knowledge base be largely acquired in a "safe" environment. The experiential phase that we presume solidifies learning for wilderness medicine practitioners often occurs in another setting at a different time, if it occurs at all. Therefore, to be effective, the material must be presented by faculty who possess the ability to captivate and motivate learners. Paul Auerbach proclaimed, "The enthusiasm of the instructor is plainly apparent and can carry or lose the day. Regardless of the educational technique chosen, one must be 'into it' or the students will be soon flocking out of it."[2]

Just as wilderness medicine practitioners must draw from a solid base of knowledge to be creative when providing care in austere surroundings, wilderness medicine faculty must be familiar with and use all of the theory-based tools available to them to be successful even in less than optimal teaching settings. Steve Donelan highlighted the issue by noting, "Instructors tend to assume that if students stay awake and interested, pass the tests, and write nice comments on the evaluation forms, then the course is successful."[11] Good teaching doesn't just happen. Highly effective wilderness medicine teachers are as proficient with the teaching skill set as they are with the clinical "tools of the trade" in their unique area of expertise.

As in highly technical health care fields, the credibility of wilderness medicine faculty is paramount. Beyond the obvious reasons for needing credibility from their teachers, most wilderness medicine learners are already well placed in their respective fields with many years of educational experience. Moreover, it is not uncommon for wilderness medicine faculty to be addressing participants who not only are proficient at the topic in question but also may be leading experts or researchers in that area. According to Auerbach, "If a teacher wishes to do more than read from a script, he or she must have some firsthand experience in the environment. Students are better than we imagine at rating our technical skills."[2]

For years, medical educators have studied the notion that the best clinical teachers all display a common set of characteristics. Numerous authors list qualities of good teachers based on learner surveys and outcomes assessments.[23,24,50] Some of these with particular applicability to wilderness medicine education are as follows:

- Possess an excellent depth and breadth of knowledge about the topics
- Are highly competent at the skills being taught
- Are highly skilled at interpersonal interactions with learners
- Are enthusiastic about their role as teachers
- Possess good supervisory skills by providing appropriate feedback, timely and clear direction, and easy access to students for questions and guidance

Educational Environment

Hutchinson recently pointed out that "in adult learning theories, teaching is as much about setting the context and climate for learning as it is about the imparting knowledge or sharing expertise."[22] Nearly everything that the teacher does influences either the students' ability or willingness to learn. Wilderness medicine course planners who account for these factors will enjoy a much higher likelihood of success than those who simply present material with little account for process and environment. Two factors that can be influenced by providing the right environment are the motivation of the learners and their perception of how relevant the material is to their lives. Adult learners derive their energy directly from the faculty and will respond in kind to the amount of effort that has gone into providing the proper learning environment.

Motivating factors to consider when planning an educational experience include physical needs and comfort issues that may hinder learning, safety and security, group inclusion and identification, self-esteem (making learners feel important and relevant), and self-actualization through self-directed learning. This all leads to academic fulfillment resulting in deeply ingrained understanding of the material. These mirror Maslow's model for hierarchical needs satisfaction.

Tangible characteristics of the environment that affect learning seem insignificant to the overall goals of the course and are easily overlooked. If they are not properly accounted for in the course plan, learners may sense a lack of respect. The best presented material by the most captivating, world-renowned teacher will not hold students' attention if their primary motivation needs are not addressed. These include factors such as food and beverage availability; frequency of rest breaks; ambient lighting, noise, and temperature of the setting; lodging accommodations; access to telephone and Internet connections; access to public transportation and airports; and the availability of recreation during nontraining hours.

Although it is impossible to solve every problem in any setting that is remote or exciting enough to host a wilderness medicine educational experience, all problems should be addressed to the extent possible. Decisions should be made early about the adequacy of the setting balanced against the need to conduct realistic training in or near the wilderness environment. Compromises may be made that trade some degree of realism for comfort and vice versa. Potential problem areas should be made known to participants well in advance so that they may make choices about the importance of these factors and balance them against their own desire to receive the training.

Training Aids

Audiovisual

Audio and visual aids may take many forms, limited only by the creativity of the teacher. The most basic may be a sand-covered stone upon which simple diagrams are drawn that communicate simple ideas and relationships. The most complex may be a multimedia presentation that incorporates diagrams, video, audio, and a computer-based interactive application to enhance learning by appealing to multiple senses. Despite the obvious advantages afforded by technology in preparing appealing and effective learning activities, pitfalls exist. These are not often represented by the lack of visual aids, but rather by the overbearing, confusing, or complex nature of those used.

The basic roles of visual aids are to illustrate the organization of a topic as it develops, reinforce or highlight key material, and provide an organizational "anchor" for the group that allows members to take notes, think, pose questions, and keep pace with the session.[15]

Examples of simple low-tech aids include terrain models, sand tables, chalkboards, paper charts, maps, and butcher-block flipcharts. These offer significant advantages to wilder-

ness medicine teachers in that they are inexpensive and retain their usefulness in austere settings. The disadvantages of these dependable visual aids include the time required to prepare them, single use or single topic, and limited flexibility if changes in teaching direction are made. Faculty cannot easily hide lack of preparation behind these simple devices.

High-tech audio and visual aids include overhead projectors, video machines, 35-mm slide projectors, and computer software–driven and projected presentations. The obvious disadvantages to the wilderness medicine educator of high-tech methods include expense and reliance on electricity, light bulbs, software, power cords, connectors, remote controls, and computers. Many "high-powered" presentations have been aborted for want of a simple piece of equipment, an incompatible software program, or a nontechnically prepared speaker.

Books

Textbooks are designed for specific purposes. It is important to address this issue before selecting a book to support an educational activity. Some are intended to be encyclopedic and used mostly for reference. These tend to be larger and cover topics from a theoretical and evidence-based perspective. They may be unwieldy for use in wilderness medicine courses that focus on skills or are conducted in remote locations.

Smaller texts are intended as summaries of larger works. These may be condensed into "quick" reference pocket versions. They are seldom used as primary texts for courses, rather as adjuncts for rapid access to information.

A third type of book is designed for use as a practical study guide. These typically relate to the more practical areas of wilderness medicine that focus on prehospital care and the acquisition of hands-on skills. Course planners may organize an entire curriculum around these highly flexible texts or may use sections to support an existing program of instruction.

Formats for modern textbooks include hard copy, electronic (CD-ROM), and fully Internet based. Publishers now integrate some hard-copy texts with web-based tools that offer advanced features inaccessible with a book such as updates, searches, and downloading graphics.

Syllabus Material and Handouts

The main purpose of providing handouts is to supplement material provided in texts or presented in class. They are often used when faculty want to either summarize or reorganize information into a format that more closely matches learning objectives. Handouts may be used to provide background information when more superficial treatments are offered in texts designed for skills or case-based learning. Other roles for handouts may be to provide a resource for the conduct of projects or self-examination, and to update information (i.e., clinical guidelines and procedural protocols) presented in texts.

Handouts that are meant for preparation before class should be provided before the session. Those meant to recap or summarize may be best provided after the session to avoid distraction.[15] Some syllabus materials are meant to save students time in note taking by providing key points with spaces given to fill in additional information. This serves the dual purpose of keeping students engaged and making them think about the information before summarizing it in their own words. This technique carries a risk that important information may be misquoted or missed entirely.

Handouts are sometimes intended for use as comprehensive study and preparation resources. Settings where this may be most beneficial are resource-constricted courses held in remote locations that are not able to provide standard texts for all students.

A modern approach to handouts is that of providing course materials either on CD-ROM or through a web-based, downloadable version. This presents challenges to the wilderness medicine educator because activities are often conducted at locations where the Internet is inaccessible and students may not carry computers.

Simulations

The use of simulation with computer-based mannequins and live patient role-play adds a dimension to skills-based training. Although this tool limits the quantity of information that can be covered and is resource intensive, it is a highly effective means to solidify learning and conduct reliable and valid assessments of hands-on skills. Except for the important teaching paradigms of austerity, limited treatment resources, and improvisation, its use in wilderness medicine is fundamentally no different than how it is deployed in other areas of health care education.

Important factors to consider when planning training using patient simulation are the setting, scene, acting, evaluation, and makeup (moulage).[12] The setting can be either in or out of doors as long as the proper clues are available to the learners. Highly realistic indoors settings call for elaborate surroundings and are often unnecessary. The basic function is to allow learners to familiarize quickly with the details of the scenario and make treatment and resource allocation decisions based on that information. Outdoors settings add more realism and use fewer resources, but are subject to limitations imposed by weather and terrain. Even though prehospital courses in wilderness medicine orient their curricula toward anticipating, recognizing, and managing clinical syndromes using limited and often improvised equipment, the use of prefabricated and pre-positioned props may make the training more efficient and assessments more reliable.

Managing the acting can be challenging. Much of what role-players will be asked to do will seem unnatural. Some will simply not have the personality or behavioral repertoire to make good role-players. This results in either overacting or underacting, both of which hinder learning and assessment. Thorough briefings about training goals and setting ground rules for role-players are crucial. Having role-players who are thoroughly familiar with the script ensures that training objectives are met and that skills assessments remain reliable. Students within the group can be used for training and derive additional learning benefit from this experience. Outside role-players should generally be used as victims for skills testing. The variation in quality of role-players can be partly overcome by having experienced and thoroughly briefed faculty and examiners at each station.

When available, makeup devices enhance learning by adding a greater degree of realism. The overriding concern when improvising or selecting moulage is whether it enhances or detracts from achieving the learning objectives of the scenario. If it does not directly support the clinical syndrome being portrayed, it may confuse learners by causing them to make faulty assumptions. This has the overall effect of incorrect learning and leads to unreliable and invalid assessments.

Computerized patient simulation is particularly well suited for skills-oriented basic and advanced life support training in wilderness prehospital courses.[32] This tool merges computer technology with fundamental behavior-based adult learning principles to deliver effective teaching. Students are both challenged and stimulated to high levels of performance. Information is delivered in a tangible, practical fashion with immediate reinforcement. Learners may repeat scenarios, make rapid corrections of wrong decisions, and see immediate results. Simulation at this level is more efficient than both problem- and scenario-based teaching, largely because of the high quality of feedback learners receive as they observe the immediate consequences of their interventions.[32]

Life-sized mannequins are used that have complex analog-to-digital computer interfaces and run algorithm-based software designed to deliver a response to anything the learner does, including the absence of activity. Software can be programmed to demonstrate minor to life-threatening clinical presentations that are interpreted by the student as physiologic changes expected in any real patient with a similar history and status. Interventions, such as cardiopulmonary resuscitation, intravenous fluid administration, injecting medication, and airway management, can be made that, if done properly and in the correct sequence, will result in the expected clinical improvement.

An additional feature offered by this tool is assessment of learning. Computer patient simulation using scenarios is one of the most effective learning assessment methods. Computer-generated reports and video of the interaction can be viewed individually or in groups to reinforce teaching points and make corrections.

Evaluation of Teaching

Assessment of the teaching process is an integral part of completing the education cycle. It is hard to imagine how any course could flourish without a mechanism to self-evaluate and make periodic adjustments. This holds educators accountable. Organizations that track, report, and maintain standards for the awarding of CME and continuing education unit (CEU) credit require this step to ensure that the quality of health care instruction is maintained at a high level. All curricula should evolve in ways that are responsive to students' needs. Formal self-evaluation requires a method to organize this process. The main purposes of evaluation are as follows[35]:

- Ensure that teaching is meeting students' needs.
- Identify areas where teaching can be improved.
- Facilitate the allocation of faculty resources.
- Provide feedback and encouragement for faculty.
- Identify what is valued by educational organizations.
- Facilitate curriculum development.

Course directors should look for correlations between results of course evaluations and academic testing. This requires well-constructed evaluation tools. Although critique of the course should have no impact on whether students graduate, it must be treated like any other assessment in order to yield meaningful results. Several issues must be addressed when designing or selecting an effective instrument.

Method of Evaluation

There are three basic methods available: individual interview, open discussion, and questionnaire-survey. Interview and discussion have limited roles in the overall, long-term process improvement of most wilderness medicine courses, but have great utility in making short-term, on-the-spot adjustments. Questionnaires and surveys have the greatest applicability and will be discussed further.

Fairness and Confidentiality

All participants must be afforded the opportunity to evaluate the educational experience. Although it may not be feasible to make this mandatory, it should be highly encouraged. Questions should be constructed so that they do not unintentionally bias the results toward one group of learners. Most instruments are provided in a confidential fashion. The issue of anonymity has two schools of thought. One advocates anonymity to reduce the impact of bias and to make sure respondents feel free to offer honest critique without fear of reprisal. The other requires students to provide their name but takes steps to guard against unintended disclosure and reprisal. This provides for responsible commentary and allows for further clarification by individual students if needed. A reasonable compromise is to make it optional to provide names.

Usefulness of the Results

Like any other assessment, the ideal evaluation is reliable, valid, acceptable, and inexpensive.[35] A violation of any of these will decrease usefulness of the instrument. A poorly designed or administered critique wastes valuable resources and time for respondents and faculty. This lack of respect transforms the process into a meaningless exercise. The most important aspect to ensure that results will be useful is construction of the instrument. It is important to make students feel vested in the course, so that thoughtful assessment is encouraged. This must be nurtured in the course design and should have already occurred before the critique forms are handed out on the final day. Attend to the principle of proximity. For lengthy courses, ensure that there is time scheduled and reminders given each day to fill out critique forms for each session. A short, overall evaluation of the course can be done at the end. Students may be in a hurry to depart after academic activities are over and evaluations are being completed. A common technique to ensure that each student completes an evaluation is to require that they turn in a form before receiving a graduation or training certificate.

What to Evaluate

Participants may be queried about anything that is relevant to the conduct of the course. However, consideration must be given to the actual usefulness of the information and the time it takes to complete the form. At a minimum, students should be given the opportunity to rate and add additional commentary on the following components:[14]

- Course design
- Textbooks
- Supplementary readings
- Audiovisual aids
- Lecture and discussion sessions
- Skills practice sessions
- Scenarios and practice exams
- Written exams and quizzes
- Instructors
- The setting (administration, accommodations, and physical surroundings)

Format of the Evaluation Instrument

Selection of the proper format of the evaluation is critical. An improperly designed instrument with poorly worded questions

may lead to meaningless results or faulty assumptions that lead to inappropriate modification of course design. It should minimize potential for error and confusion by providing as much unique identifying course-related information as possible. Instructions must be brief and clear.

Written commentary format gives immediate comprehensible feedback to the faculty; however, it may be superficial and limited in scope. Comments may be difficult to translate into actionable information that leads to course improvements. Evaluations that use this method exclusively have limited reliability and validity.

The multiple-choice format facilitates tabulation and objective comparison. The choice of responses must be broad enough to avoid bias toward either end of the scale. In general, simple good–bad descriptors are less helpful than those that address the function and effectiveness of the item in question. Descriptive adjectives should be selected that match the component being evaluated. For example, adjectives describing helpfulness and effectiveness may be most appropriate when asking questions about class materials or procedures. A space for commentary should be provided after each question. Scaled forms upon which the student must fill in a circle or cross off a number to rate the specific features of the course are helpful in managing the results and take less time to complete. A quick reminder as to the nature of the item they are being asked to rate may accompany these questions. Rating scales can be from 1 to 5 or 1 to 10 and may correspond to various forms of descriptors.

No matter what format is chosen, it is a good idea to leave room for several open-ended questions that address the most and least valuable portions of the course, as well as what improvements the respondent recommends. Responses to these tend to be highly contextual based on the background and personality of the student. However, multiple responses from a single class that focus on the same issue or several that are similar across multiple courses may indicate need for change.

Provide the Results

Course directors should provide tabulated and summarized results of the evaluations to all faculty members. Both group and individual sessions with instructors may be beneficial to discuss the results and select process improvement strategies. Faculty members should only see specific comments about themselves and not those made about other instructors. There should also be some way to inform the students about the results of the evaluations and the resulting actions that were taken.

Evaluation of teaching is a critical component to the overall process of educating adults in any discipline. Learners and teachers at all levels expect this and often look forward to it. With some effort in planning and design work, the practice can become one of the most valuable tools available to wilderness medicine educators.

PROGRAM AND CURRICULUM DEVELOPMENT

The notion that teaching in health care should be organized and programmed continues to evolve. The uniqueness of wilderness medicine presents challenges and affords educators ample opportunity to be innovative in curriculum design. This applies to short certification-oriented courses, embedded curricula within longer programs of instruction, or limited CME/CEU programs.

In its most basic form, a curriculum is an expression of community values. In other words, if it is to remain viable, it should reflect the current and changing values of the community it serves. Because students will ultimately return to the community to practice, the community, made up of stakeholders, should have a say in what they are taught. What they are taught should be directly relevant to the needs of the community from which they come.

Concepts and Models

Prideaux recently described a curriculum as "existing at three basic levels: what is planned for the students, what is delivered to the students, and what the students experience." In practical application, a curriculum should be easily communicated to learners and educators, should be open to critique and modification, and should be easily implemented. More specifically, it should contain four elements: content, teaching and learning strategies, assessment processes, and teaching evaluation methods. The process of curriculum design attempts to form these elements into a tangible, usable device.[41]

Several models of health care curriculum design have been proposed over the years. These have evolved indirectly based on changing needs, values, and expectations of society. The one that has emerged as being most effective and has the greatest applicability to wilderness medicine is based on identifying, addressing, and assessing desired outcomes. This is appropriately referred to as *outcomes-based education*. Although usually discussed in the context of clinical teaching settings such as medical schools and residencies, the focus on outcomes is germane to wilderness medicine. No matter how the details of a curriculum unfold in practice, keeping the focus on outcomes places the wilderness medicine learner at the center of the education cycle. The basic concept of this model requires that educators first decide on what the students should know (outcomes), design a program of learning to allow them to achieve it, and then assess whether they achieved the desired results. The details of how educators make these determinations describe an elaborate and interesting process.

Numerous versions of curriculum design are in use throughout the United States and Europe. Many have been reported in the wilderness medicine literature.[7,32,38–40,51] Steve Donelan has written extensively on the subject and offers the most detailed guidance available.[9,10,12,13,17] Nearly all courses use a combination of techniques to deliver material to learners. A focus on outcomes is a common theme.

Steps in Designing a Curriculum

All aspects of curriculum design should be well thought out before resources are allocated. The general sequence starts with identification of the desired outcomes, or what the students should be able to do (including a learning needs assessment), addresses specific content (topics to be taught), selects teaching methods, develops learning assessment instruments, and finally, constructs tools to evaluate the process. All these have been addressed previously and are mentioned below.

Desired Outcomes

Outcomes are addressed on two levels: the overall course goals or objectives and specific learning objectives for each session or

activity. As the name implies, course goals describe the overall purpose for students attending the course. This may include a job- or skill-related certification or simply advancement of their general fund of knowledge. It addresses what they should be able to do or know after completing the entire program. All activities related to the formal curriculum should contribute in some way to these goals. This is similar to a large corporation that does institution-level analysis of mission, vision, goals, and objectives. Any action resulting from this strategic planning that does not contribute in some way to the mission of the organization is, at best, distracting and possibly a hindrance. Individual learning objectives can be viewed in this context. Accomplishment of the course goals depends on them.

Two sources, or stakeholders, should be queried to determine outcomes at both levels. Potential learners may be asked using various methods (polling, focus groups, and questionnaires) what they think is important and what they would most like to learn. Organizations that hire or interface with potential graduates of the course may be asked for their input on skills, knowledge, and qualities the graduates should possess. The goals and objectives should be formalized, written, and distributed to anyone involved with curriculum design. They serve as a compass to guide all planning and design efforts.

The set of general educational objectives for the WPHEC curriculum proposed in 1991 by the Wilderness Medical Society (summarized below) offer an example of how to organize course objectives for this type of program.[51] A learning needs assessment of the intended audience allows these to be rewritten in a more active tense using appropriate verbs to make them suitable for use as outcomes in any setting. Courses designed to provide certification or registration would address this requirement, as well in the course objectives.

- Review basic principles of anatomy, physiology, and emergency care, emphasizing their application to, and if necessary, modification for, unique problems in a wilderness environment.
- Review common illnesses and injuries, emphasizing modifications of assessment and care in a wilderness environment.
- Review the causes, assessment, and treatment of illnesses and injuries unique to the wilderness environment.
- Use extended-care techniques in a wilderness environment before evacuation of a casualty to definitive care.
- Apply the principles of wilderness survival, search and rescue, and extrication, packaging, and transport of victims from a wilderness setting.
- Review suggested contents of wilderness emergency care kits and improvisation of emergency equipment and supplies.
- Practice prevention strategies for wilderness injuries and illnesses.

Some wilderness medicine courses focus on specific environments such as dive, tropical, aerospace, or desert medicine. Course objectives for these should reflect the unique requirements of each and be tailored to the needs of the anticipated audience.

1. *Determine the content*

After the course objectives are established, they are used as a basis to begin selecting major content divisions or sections. There is no "best" method of dividing the information, but sections should be easy to support, with effective teaching and assessment methods based on sound learning theory. The organization of the Wilderness Medical Society WPHEC objectives suggests sections devoted to basic science, clinical science, emergency medical services, evacuation procedures, equipment, field craft, and prevention. The major sections are further broken down into subsections or topics. The basic science section might be divided by topic-specific reviews of anatomic or physiologic systems. The field craft section might be broken down into land navigation and wilderness survival. Land navigation may be further divided into sessions on map reading, compass use, celestial navigation, and use of Global Positioning Systems (GPS). It is this final level for which specific learning objectives are written. There are no absolutely right or wrong answers to selecting the specific content of a course. As long as the basic steps are followed, a suitable solution will reveal itself.

2. *Select teaching methods*

Methods to deliver the information should be selected after the course content has been established. Course planners should make use of several techniques and, if possible, vary them daily to maintain student interest. An entire day of hands-on practical exercise in frigid temperatures may distract from learning as much as 10 hours of back-to-back lectures. Intermixing the techniques by spending 2 hours outside, 2 hours indoors in small group work, followed by 2 more hours outside, capped off with 1 or 2 hours of lecture at the end of the day is an example of this type of variation. Planners should make maximum use of the various forms of problem-based instruction and small-group work. Lectures should be limited to introductions and topics that absolutely do not lend themselves to the aforementioned techniques.

Of the steps in curriculum design, the selection of teaching methods may be most influenced by the physical setting of the course. This includes climate, terrain, and accommodations for teaching and lodging, as well as available support. There is no best time in the curriculum design process to finalize the setting. It will sometimes be pre-established, forcing accommodations in design work. At other times, the curriculum may be set, pending the outcome of a suitable location to conduct the course.

3. *Select learning assessment instruments*

The assessment of learning derives directly from the course objectives that are supported by the learning objectives. Written testing will allow assessment of both. Some questions may be intended to evaluate knowledge of facts addressed by learning objectives from individual sessions. Others will address the synthesis of information drawn from multiple sessions and in different sections. Written tests or quizzes can be used for formative assessment, whereas hands-on skills testing using scenarios and role-players may be used in practice, formative, and summative (end-of-course) roles. For some courses, skills-based assessments may be out of place, forcing planners to be skilled at constructing written exams. The main concept in constructing assessment tools is to develop them based on well-written session and course objectives.

4. *Develop the course evaluation*

Evaluation of the course is the final step in the education cycle and in curriculum design. Reserving this until last should not undermine its importance. Many highly effective examples of evaluation tools exist. Adhering to the basic principles discussed previously will lead to an effective instrument that informs continuous improvement and curriculum modification.

Text continued on p. 2163.

TABLE 92-2. Wilderness Medicine Educational Resource Organizations

NAME	TYPE	LOCATION	TIME	CLASS SIZE	CONTACT INFORMATION	COMMENTS
Wilderness Medical Associates	Commercial Instruction	Bryant Pond, ME; specific to sponsoring organization	Varies by sponsoring organization	Variable	www.wildmed.com	WFR, WEMT, SAR, WFA, WALS
Wilderness Medicine Outfitters	Commercial Instruction	Elizabeth, CO; or client specific	Varies	Variable	www.wildernessmedicine.com	WFR, WEMT, WFA, others
CDS Outdoor School	Commercial Instruction	Valencia, PA	Varies	Variable	www.cdsoutdoor.org	WFR, WEMT, WFA, SAR, others
First Lead	Commercial Instruction	Norwood, CO; or client specific	Varies	Variable	www.firstlead.com	WFR, WFA
Wilderness EMS Institute	Commercial Instruction; Academic	Pittsburgh, PA	Varies	Variable	www.wemsi.org	WEMT, W-EMS Rotation to Emergency Med. Residents, Wilderness Command Physician (WCP)
Stonehearth Open Learning Opportunity (SOLO)	Commercial Instruction	Conway, NH; or client specific	Varies	Variable	www.soloschools.com	WFA, WFR, WEMT, Adv. WFA
Outward Bound (Regional)	Commercial Instruction	Multiple	Varies	Variable	www.obwilderness.org www.outwardboundwest.com	WFR
National Outdoors Leadership School–Wilderness Medicine Institute (NOLS-WMI)	Commercial Instruction	Multiple	Varies	Variable	www.nols.edu	WFA, WFR, WEMT
University of Utah	Commercial Instruction		Varies	Variable; physicians	www.awls.org	AWLS
WMS Resident-Student Elective in Wilderness and Environmental Medicine	Academic Elective	Townsend, TN	February, 1 month	24 Residents and Students, all health care disciplines	www.wms.org	Hosted by the WMS Comprehensive curriculum; didactic, WFR training; 5-day exercise
University of Massachusetts Wilderness Medicine Elective	Academic Elective	Utah	May, 1 month	12 Medical students	drh@pop.tiac.net	Combined didactic, hands-on, practical exercises

University of South Carolina Wilderness Medicine Course	Academic Elective-Course	Columbia, SC	September, 3 weeks	Residents and medical students	www.scwildernessmed.com	Combined didactic, hands-on, practical exercises, final exam
University of New Mexico Wilderness International and Improvisational Medicine Elective	Academic Elective	Various in NM, UT, CO	March, 1 month	10 Physicians, Residents, medical students	dmacias@salud.unm.edu	Comprehensive curriculum including didactic and WFR training; final exam
University of Washington Dept. of Pediatrics Elective in Wilderness Medicine	Academic Elective	Washington State	June, 1 month	14 (variable) Residents and medical students	diek@u.washington.edu	Comprehensive curriculum including didactic and hands-on training
University of California-SF at Fresno Wilderness Medicine Elective	Academic Elective	Fresno, CA	September, 2 weeks	10 MS-4 medical students	lori.weichenthal@ucsfresno.edu	Broad curriculum, swift-water rescue cert; no exam
University of Utah Advanced Wilderness Life Support	Academic Elective	Moab, UT	April, 2 weeks	100 medical students	www.awls.org	AWLS medical for students
Universities of Washington and Idaho Family Practice Programs: Rocky Mt. Wilderness Medicine Conference	Academic Elective/ Conference	Boise, ID	June, 2 weeks (biannual)	60 medical students; all professionals for first portion	hilvers@cableone.net	Two parts; CME conference then outdoors phase; swift-water training
Wilderness Medical Society	Conferences and Academic Resources	Colorado Springs, CO (home office)	Annual Scientific Conference	Variable	www.wms.org	Annual Scientific Conference, World Congress, Topic Specific Conferences

TABLE 92-2. Wilderness Medicine Educational Resource Organizations—cont'd

NAME	TYPE	LOCATION	TIME	CLASS SIZE	CONTACT INFORMATION	COMMENTS
University of California, San Diego: Southeastern Wilderness Medicine Conference	Conference	Chattanooga, TN	October, 5 days	Variable	http://cme.ucsd.edu	CME Conference
Wilderness and Travel Medicine Group	Conference and Field Courses	Variable	Variable	Variable	www.wilderness-medicine.com/about.asp	CME conferences and focused field experiences
National Ski Patrol	Educational Resources and courses	Variable	Variable	Variable	www.nsp.org	Courses and educational resources for winter sports injuries and safety
Undersea and Hyperbaric Medical Society	Educational Research conferences and courses	Variable	Variable	Variable	www.uhms.org	Professional and academic resources for undersea and diving activities; hyperbaric oxygen therapy and research
Aerospace Medical Association	Educational Resources Research and conferences	Variable	Variable	Variable	www.asma.org	Professional, educational, and research resources for aviation, space, and environmental medicine
International Society for Mountain Medicine	Educational Resources Research and conferences	Variable	Variable	Variable	www.ismmed.org	Professional, educational, and research resources for high-altitude and mountain medicine
International Society of Travel Medicine	Professional educational resources, and conferences	Variable	Variable	Variable	www.istm.org	Professional, educational, and research resources for travel medicine

AWFA, advanced wilderness first aid; AWLS, advanced wilderness life support; CME, continuing medical education; WALS, wilderness advanced life support; WEMT, wilderness emergency medical technician; WFA, wilderness first aid; WFR, wilderness first responder; SAR, search and rescue.

WILDERNESS MEDICINE EDUCATIONAL ORGANIZATIONS, RESOURCES, AND PROGRAMS

There are countless sources of training opportunities and information available today to the wilderness medicine educator and student. An explosion of information on the Internet provides an endless stream of ideas and resources. Professional societies around the world have blossomed, most with easily accessible websites. Organizations that provide wilderness-related prehospital emergency care at various levels continue to develop. Medical schools, graduate medical education programs, and fellowships that focus on wilderness medicine are becoming more popular.

In Europe, wilderness medicine practitioners have taken the professional and academic stature of the discipline to new heights. Many work full time in mountaineering and high-altitude environments. Professional societies have guided the establishment of academic standards that are used in numerous mountain medicine courses. For instance, practitioners who complete the appropriate level of training may be awarded the International Diploma of Mountain Medicine. Various specialty courses are available in areas such as mountain rescue and expedition medicine. Patrick Peters reviewed the European mountain medicine education process and offered examples of curricula based on these courses.[39] Professional societies like the International Society for Mountain Medicine and the Wilderness Medical Society have worked to improve international cooperation and sharing of knowledge.

Table 92-2 provides a list of some educational resource organizations in wilderness medicine.[36]

The references for this chapter can be found on the accompanying DVD-ROM.

Medical Liability and Wilderness Emergencies

93

Carolyn S. Langer

The following is an excerpt from the standard "fine print" found on the back of cruise passengers' tickets:

CARRIER NOT LIABLE FOR PASSENGER'S MEDICAL CARE

(a) Medical Services on Vessel. The Carrier does not undertake that a physician or medical personnel will be aboard the Vessel. If the Vessel does carry a physician or medical personnel, then they are independent contractors and work directly for the Passengers.

(b) Consent of Treatment. If, in the opinion of the Carrier, a Passenger in need of medical or surgical services is unable to request it, the Passenger hereby expressly consents to such treatment, if any, and to pay the cost thereof charged by the Vessel's physician or other physician or medical personnel designated by the Carrier who in doing so is acting on behalf of the Passenger.

(c) Treatment at Passenger's Risk. All medicines and all medical or surgical services furnished by the Vessel's physician, if any, or any other physician or medical personnel (all of whom are engaged by the Passenger as independent contractors) or ship's officers, employees or agents of the Carrier, shall be and are accepted by and at the Passenger's sole risk and expense, and the Carrier shall not be responsible for the quality, nature or consequence thereof.

(d) Passenger's Obligation to Report Medical Conditions. Before this Ticket is issued, the Passenger must report to the Carrier or its general agent, all pre-existing illness, disabilities or pregnancy and all other conditions for which the Passenger may require medical attention during the course of the Cruise. If any such condition arises after the Ticket is issued, the Passenger must report all such conditions to the Carrier, Purser or Vessel's Master or physician, if any, before boarding, or if the Passenger has boarded the Vessel, then before the Vessel leaves port. The Carrier, Vessel and the Vessel's physician, if any, shall have no liability in connection with any such condition.

(e) Lack of Obligation to Examine Passenger. Neither the Carrier, nor the Vessel's physician, if any, has any obligation to examine any Passenger for any purpose prior to boarding or sailing.

(f) Refusal of Passage. The Carrier reserves the right to refuse passage to a Passenger who has failed to give proper notice of any disability, illness, pregnancy or other condition requiring special care, attention or treatment or who in the

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Carrier's sole opinion is physically or mentally unfit for the Cruise. In such event, the Passenger is not entitled to any refund of fare.*

In recent years, personal wealth, general interest, increased leisure time, globalization of the economy, and sophisticated marketing have led to expanded participation by the general public (with an aging population) in expeditions and cruises to remote environments, including wilderness areas. Such travel poses a higher risk of injury and illness than does travel in urban areas and often involves exposure to extreme weather conditions, limited access to medical personnel and supplies, and extended patient management due to limited medical evacuation capabilities. What are the medical-legal ramifications for tour operators and trip physicians? Historically, the probability of a lawsuit was low because participants were younger, more physically fit, and more experienced adventurers who tended to be greater risk takers with an awareness of and a willingness to assume these risks. However, the increased involvement of a less prepared and marginally aware general population in these wilderness pursuits has undoubtedly generated a concomitant increase in exposure to liability. To minimize this liability, tour operators and trip physicians must implement sound risk management strategies for medical clearance, education, and provision of medical services to trip participants.

TORT LAW AND THE DOCTRINE OF NEGLIGENCE

Case Study: Snakebite and Medical Malpractice

On August 11, a victim sustained a rattlesnake bite on the index and middle fingers of his left hand. Suction cups and a tourniquet were immediately applied, and the victim was transported to the hospital within 15 minutes. Another 15 minutes passed before Dr. A arrived. Dr. A proceeded to treat the patient by injecting antivenom into the base of the fingers bitten by the snake and into the left deltoid, despite an instruction sheet accompanying the antivenom that cautioned, "Do not inject serum into a finger or toe." Dr. A packed the patient's hand in ice and admitted him to the hospital. Two days later the patient was sent home with instructions to keep ice on the hand.

Eight days later the victim's two fingers, hand, and arm were edematous, discolored, and odorous from gangrene. Dr. B assumed care of the patient, applying a heating pad and injecting antibiotics. Nevertheless, on September 9, the two fingers required amputation. Is Dr. A liable for medical malpractice?

In tort law, members of society owe a duty to others to act reasonably and in a way that will not hurt another person or his or her property. In the context of wilderness medicine, redress for a private civil wrong is typically sought under a medical negligence doctrine. To bring a cause of action under a negligence theory, the plaintiff (the party filing the claim) must establish the following four elements:

1. Duty: The defendant has a duty by virtue of the physician–patient relationship to use due care as a reasonably prudent physician would under similar circumstances. For example, a plaintiff could argue that a trip physician owes the participants a duty to "possess and bring to bear on the patient's behalf that degree of knowledge, skill, and care that would be exercised by a reasonable and prudent physician under similar circumstances"[7] given the prevailing state of medical knowledge and available resources. To meet this duty, physicians must typically adhere to the standard of care established by the medical profession.
2. Breach of duty: Defendants breach their duty when they fail to conform to the duty of care or to act in accordance with norms or standards of practice. This breach may occur either through commission or omission of certain acts. For example, a physician may breach the duty of care by failing to diagnose or treat a condition (omission) or by improperly treating a patient (commission).
3. Causation: The plaintiff must further prove that the defendant's conduct was the direct, foreseeable, and proximate cause of the resulting injury. In other words, absent the defendant's conduct, the harm would not have occurred. Establishing causation is often the most problematic step for plaintiffs. For example, an injured traveler might allege that a trip leader or medical practitioner breached the duty of care by delaying medical evacuation, but the defendant could argue that the injury had already occurred and that any delay in evacuation neither caused nor aggravated the injury.
4. Damages: Finally, the plaintiff must demonstrate damages, that is, harm to the individual's person, property, or interests for which the redress is customarily a monetary award. Awards may encompass special damages (e.g., out-of-pocket losses, medical expenses, lost earnings), general damages (pain and suffering, mental anguish, and other emotional injury), and punitive damages. Punitive damages are rare in the medical context because the plaintiff must prove that the defendant's conduct exceeded simple negligence and was intentional, grossly negligent, reckless, malicious or fraudulent. Nonetheless, physicians should be aware that their insurance policies generally do not provide malpractice coverage for punitive damages.

Medical malpractice is a specific form of negligence occurring during the execution of a physician's professional or fiduciary duties. Medical malpractice can be defined as medical care that falls below the standard of care expected of a reasonably prudent physician under similar circumstances, resulting in foreseeable harm to the patient. In all negligence suits, the plaintiff bears the burden of proof in establishing the requisite four elements under a "more probable than not" standard. In other words, the plaintiff must establish that there was a greater than 50% probability that the defendant's breach of duty caused the harm.

In the snakebite case presented earlier, the first element is clearly demonstrated. Dr. A's actions established a physician–patient relationship and an acceptance of the duty to render care. Establishing the second and third elements is more problematic. The court held that Dr. A did not breach his duty. The court elaborated, "There are wide variations in accepted methods of treatment of rattlesnake bites. The method of treatment chosen and used by the defendant was an acceptable method of treatment." In other words, even though physicians must act with the level of skill and learning possessed by minimally qualified members of the profession, they are judged by reference to the beliefs of the school that they follow. That is, their practice need not be followed by a majority consensus,

*From Levinson J (ed), Ger E (medical ed): Safe Passage Questioned: Medical Care and Safety for the Polar Tourist. Centreville, MD, Cornell Maritime Press, 1998.

provided that it is supported by a recognized school of practice in the medical community.

The court further held that the plaintiff failed to establish causation. Expert testimony showed that "rattlesnake bites in extremities always present some chance of tissue destruction" and that the most probable cause of tissue death in the two distal phalanges was the rattlesnake venom. Thus, Dr. A was found not liable. Whether Dr. A's intervention met the standard of care and caused the ultimate amputation is debatable. Typically, though, both parties to the litigation will generally need to use expert witness testimony to establish their respective positions vis-à-vis standard of care and causation issues.

Each state has its own body of statutory, regulatory, and case law (in addition to federal law). Thus, although this chapter presents general legal and risk management principles, physicians should always familiarize themselves with the laws and precedents in their own states before implementing policies and procedures related to wilderness or travel medicine. Unless the trip application has a jurisdiction clause explicitly limiting the venue for litigation (often the state in which the tour outfit has its headquarters or principal place of business), a plaintiff could theoretically bring a claim in his or her own state of residence, in the state where the physician is licensed, in any state where the tour outfit conducts business or has some nexus, or in the jurisdiction where the injury occurred.

LIABILITY CONCERNS IN WILDERNESS MEDICINE

The remainder of this chapter examines how the legal doctrines apply in the unique circumstances of wilderness medicine. Not surprisingly, increased participation of the general population in adventure travel has led to higher—and in many cases unrealistic—expectations on the part of the public. Some unseasoned travelers to remote destinations anticipate the same level of medical resources (including personnel, equipment, medications, and evacuation capabilities) to which they have access in their own large, urban tertiary care medical centers. It is therefore vital for tour operators and trip physicians to understand the duty and standard of care to which they must adhere and to educate trip participants regarding the risks of travel.

DUTY TO WARN AND EDUCATE TRIP PARTICIPANTS

Case Study: Duty to Warn

A middle-aged male traveler booked a tour to Bolivia through a travel agent. In the trip brochure, the tour operator represented itself as an experienced and reputable company and stated that it researched all locations to which it arranged tours and "would care for participants from 'portal to portal.'"[5] In addition, the travel agent asked both the tour operator and local health agencies about health requirements and necessary precautions. During the trip, the traveler developed high-altitude cerebral edema after flying from Chile (at sea level) to La Paz, Bolivia (elevation 3960 m [13,000 feet]) in less than 1 hour. He subsequently sued the travel agent and tour organizer for failure to warn him of the health risks associated with his travel.

In general, there is no duty to warn of dangers that are as obvious to the participant as to the organizer. Obvious dangers might include possible seasickness or airsickness, substandard sanitation, and poor environmental conditions. Moreover, travel agents and tour operators have no duty to investigate potential vacation sites (not even those where conditions and terrain are dangerous) or lower standards of medical care in foreign countries. However, they do have a duty to warn travelers of known unreasonable risks or dangers, such as political turmoil and criminal attacks, that are foreseeable or likely to occur. Typically, to incur this duty to warn, the travel agent or tour operator must have actual or constructive notice of the hazardous condition arising from their knowledge of special circumstances (e.g., prior occurrences). In addition, travel agents and tour operators may contractually expand their duty to warn through representations made in brochures or other advertisements. In the case mentioned earlier, for example, the court recognized that tour organizers are not insurers of the safety of tourists with whom they contract and need not warn of obvious hazards. Nonetheless, in this case the travel organizer contractually expanded its obligation through its representations and brochures and created a reasonable expectation on the part of the tourist that it would research the risks of high-altitude travel and warn him of accompanying dangers.

Even in the absence of liability, trip organizers and medical practitioners have incentive to prevent travel-associated injury or illness because they suffer financially and ethically when a tourist experiences physical or financial loss, fellow travelers are inconvenienced, or the reputation of the company or medical practice is potentially damaged. Sound risk management principles dictate that travel companies provide as much useful information as feasible to prevent adverse consequences. Companies lacking the wherewithal to research health hazards may benefit from the assistance of a medical adviser. Depending on the nature of the trip, some or all of the following information may be useful to participants:

1. Environmental conditions (e.g., climate, altitude, terrain)
2. Activity level (type, intensity, duration, and frequency of activity or exertion required)
3. Specific health hazards (e.g., tropical diseases, marine envenomation, frostbite, heat injuries)
4. General health information (e.g., safety of local food and water, local medical resources)
5. Known risks (e.g., political unrest, high crime areas)
6. Recommended immunizations

Whether an outfitter has an obligation to provide helmets and safety equipment depends on many factors, including anticipated hazards, experience of participants, industry practice, and so forth. However, even when the outfitter requires participants to furnish their own safety equipment, it is advisable for the outfitter to identify the potential hazards and required gear. Perhaps a more pressing concern is the noncompliant participant who refuses to wear or use furnished safety gear or who otherwise acts in a reckless manner. In these instances, signed waivers may be of some value, but juries are not always sympathetic to waivers because the trip operator is sometimes perceived as being in a better position to ensure participant safety. The more prudent risk mitigation step might be to include a statement in the trip brochure or application indicating that the trip operator may, in its discretion, dismiss any participant at any time during the trip for cause—including failure to wear appropriate safety gear or to comply with safety rules—at no liability or cost to the trip operator.

MEDICAL CLEARANCE OF TRIP PARTICIPANTS

Tour operators have no duty to medically screen trip participants. Travelers have a responsibility to exercise due caution for their own safety. Nonetheless, do tour operators have the right or an incentive to screen participants? Medical clearance of travelers may serve several beneficial purposes. It can function to educate participants about potential health hazards, thereby reducing their risk of harm. Medical screening affords the participant's private physician the opportunity to more effectively manage the patient's care. It also may enable a tour operator to arrange accommodations in advance for participants with special needs. Moreover, medical clearance may reduce the tour operator's liability, costs, and inconvenience.

On the other hand, medical screening has certain disadvantages. Some authorities consider medical screening useless because a high-risk patient with a strong desire to go on a trip may ignore the inherent dangers and conceal any significant medical history (or shop around for a physician willing to grant the medical clearance). In addition, since the enactment of the Americans with Disabilities Act (ADA) of 1990, many tour operators fear discrimination suits for medically screening out disabled persons. However, with a proper understanding of the legal issues underlying medical clearance and the ADA, tour operators and physicians can safeguard the interests of all parties involved.

The Americans with Disabilities Act

Title III of the ADA prohibits all places of public accommodation and services operated by private entities from discriminating against the disabled. Furthermore, a public accommodation may not apply eligibility criteria that screen out an individual or any class of individuals with disabilities "unless such criteria can be shown to be necessary for the provision of the goods, services, facilities, advantages, or accommodations."[8] For example, "a cruise line could not apply eligibility criteria to potential passengers in a manner that would screen out individuals with disabilities unless the criteria are 'necessary.'"[9]

A public accommodation is not required to permit an individual to participate in services when the individual poses a direct threat to the health or safety of themselves or others. This determination that the participant poses a significant risk must be an individualized assessment using reasonable judgment based on current medical knowledge or the best available objective evidence. In addition, the "safety exception" may justify excluding individuals when they pose a threat to themselves. The "safety exception" holds that a public accommodation may impose legitimate safety requirements that are necessary for safe operations. As an example, the regulations cite as a valid screening criterion the requirement for all participants in a recreational rafting expedition to meet a necessary level of swimming proficiency.

For screening criteria to be valid, however, they must be uniformly applied to all prospective participants, not merely to those with disabilities. Moreover, they must be based on actual risks, not on mere speculation, stereotypes, or generalizations about a person or class of persons with a particular disability. A tour organizer could not, for example, categorically exclude all persons with a history of angina from a high-altitude trip but rather must afford individuals with documented, well-controlled angina an opportunity to establish their fitness and eligibility for the trip.

Even when screening criteria are valid and the prospective participant cannot meet those requirements, a public accommodation must make reasonable modifications in policies and practices or must accommodate the disabled individual unless such modifications would fundamentally alter the nature of the goods, services, or facilities offered. For example, a cruise line could be required (as a reasonable modification) to provide an individual who relies on a wheelchair for mobility with a stateroom on the same level as the restaurant. However, a mountain trek would not be required to transform to a bus tour to accommodate this same individual.

Medical Screening

To minimize the potential for discrimination claims, tour operators should provide physicians granting medical clearance as much information as possible regarding the physical demands of the trip. Tour organizers must provide detailed information concerning environmental conditions, specific health hazards, health and living conditions in the destination site, and availability of local medical resources. Companies should be as specific as possible in describing environmental conditions (e.g., altitude in feet, temperature in degrees). Trip organizers also must delineate the type, intensity, duration, and frequency of activity (e.g., bus tour, mild walking [1.6 to 3 km or 1 to 2 miles per day], vigorous hiking [specify distance and terrain], backpacking [specify weight of gear], trekking to remote areas [e.g., 24 hours from definitive medical care], climbing, swimming, canoeing).

The health care professional in turn must tailor the medical history and examination to the physical demands of the trip. The physician should become familiar with the patient's medical history, including any medications that could interact with the environment to which the patient is traveling. If applicable, the clinician should advise the patient to bring extra medications or medical equipment (e.g., a spare pair of eyeglasses, extra hearing aid batteries, an anaphylaxis kit) in case complications or exacerbations occur. The physician also should inform the patient of applicable first aid procedures, and may also need to inform the trip organizer of special needs or accommodations.

Because patients and their physicians are sometimes hesitant to disclose confidential medical information to a travel company, medical clearance forms should emphasize functional abilities and limitations rather than diagnoses. If medical clearance forms request sufficiently detailed information about the prospective participant's medical fitness to meet the physical demands of the trip, travel companies frequently do not need to know about specific medical diagnoses. Companies that do request confidential medical information should disclose this information only to employees who have a need to know and should ensure that the company medical adviser or trip physician safeguards this information in a locked file, typically at the company's offices, unless arrangements have been made for the physician adviser to store medical records at his or her private office. The company also may want to encourage high-risk patients to share medical information with trip leaders or bring along key medical records. It may be desirable for the trip physician or individual trip participants to bring along key medical documents or summaries that can easily be stored in their backpacks, particularly when traveling under Spartan conditions or to remote sites. Even though a company-employed medical

adviser or private family physician makes determinations about medical fitness and medical clearance, the travel company bears the legal responsibility for excluding disabled participants or for failing to accommodate their needs.

PROFESSIONAL LIABILITY, MEDICAL MALPRACTICE, AND GOOD SAMARITAN LAWS

In addition to those complaints arising from a lack of proper health warnings, injured trip participants commonly base claims on the company's failure to provide adequate medical services and facilities and the negligent delivery of medical care. Historically, tour operators and cruise ships have had no obligation to ensure the health or safety of participants or to provide health care—particularly physicians' services—on trips. Trip organizers should nonetheless ascertain the appropriate standard of care for their particular circumstances by determining the medical resources that other companies have provided on similar expeditions to similar destinations. Companies generally have the duty to staff trips to remote locations with personnel who have "significant training to provide adequate first aid and medical care until evacuation can be arranged."[3] Typically, these staff members are emergency medical technicians (EMTs) or laypersons with basic first-aid training. Organizers of trips to locales with readily available medical care may have a lesser obligation to furnish staff trained in first aid. In the event of an adverse medical outcome, travel company employees are judged by a "reasonably prudent person" standard; that is, they have the duty to use reasonable care to furnish such aid and assistance as an ordinarily prudent person or trip leader would under similar circumstances.

Several professional medical organizations, such as the American College of Emergency Physicians and the Wilderness Medical Society, have sought to define and establish standards of care for the provision of medical services on cruise ships and expeditions. Many companies voluntarily staff physicians on their trips (and publicize that they have done so) for the comfort and convenience of the participants or to provide a competitive marketing advantage. Travel companies that advertise the presence of trip physicians and medical facilities in brochures, contracts, and other correspondence to participants or prospective participants contractually expand their obligation by creating an implied or express contract for the availability of certain medical services. At a minimum, it is expected that trip physicians will have the resources and capability to evaluate and manage emergencies and, when necessary, will arrange for more definitive care for the types of injuries and illnesses that might be reasonably anticipated on these types of trips.[1]

Who, if anyone, is liable when the physician commits medical malpractice? Is the physician protected by Good Samaritan laws? Although Good Samaritan laws vary from state to state, most of these laws generally hold physicians and other personnel free from liability when assisting in an emergency, provided their conduct was not grossly negligent (a higher threshold than simple negligence), wanton, or willful. Other provisions common to many Good Samaritan laws include the requirement to render care in an emergency or at the scene of an emergency, to act in good faith, to provide services gratuitously, and to have no preexisting duty to the victim to respond or provide aid. For example, if a physician happens upon a stranger in need of emergent care while hiking up a mountain and provides medical care, his or her services would most likely fall within the purview of Good Samaritan laws.

On the other hand, does the physician acting as the "trip physician" lose his or her Good Samaritan protection? A typical custom in the adventure travel industry is to offer physicians generous discounts on trips in exchange for the provision of medical support for fellow participants. Many of these physicians are under the mistaken impression that they are covered by Good Samaritan laws in the event of a malpractice claim. The discounted fee, however, is the equivalent of compensation, and these physicians, along with those on straight salaries, would in all likelihood be considered independent contractors with a preexisting duty to render medical care to fellow participants.

This potential exposure to malpractice claims has two important risk management implications for expedition physicians. First, physicians must familiarize themselves with the medical issues that they are likely to encounter on their trips and must possess the knowledge and skills to diagnose and treat relevant medical disorders. There is currently no prescriptive training, certification, or specialty requisite to serving as a trip physician. However, in determining the standard of care, a jury would expect the physician to anticipate foreseeable medical problems, assess the patient, and administer first-aid measures as a reasonably prudent physician would under similar circumstances, taking into account the remote location, the extremes of environment, and the limited capability to transport medical equipment on the trip. A physician employed on a cruise to known endemic areas must be familiar with the diagnosis and treatment of tropical diseases. A physician with a mountain trekking group should be able to recognize the signs and symptoms of high-altitude sickness and be proficient in the management of this condition.

The trip physician's specialty is generally not a shield from liability. For example, a psychiatrist who holds himself or herself out as a trek physician would not be exculpated in a malpractice claim merely because he or she lacked training and experience in infectious diseases, primary care, emergency medicine, and other relevant areas. Therefore, physicians who lack the necessary skills to diagnose, treat, or stabilize basic first-aid, primary care, or emergency conditions may want to consider declining participation as a compensated trip physician because they would likely be held to the same standard of care as, for example, a primary care or emergency medicine physician. On the other hand, specialty alone does not disqualify a physician from participating as a trip physician if he or she has acquired the appropriate skills through prior participation in similar activities, conferences, previous duty in the public health service or military, and so forth. The determinative issue is how the "reasonably prudent physician" would have prepared for the trip and diagnosed and treated the patient under similar circumstances. Certifications, such as Advanced Cardiac Life Support (ACLS), Advanced Trauma Life Support (ATLS), and various wilderness medicine programs, are useful in demonstrating that the trip physician had training and acquired a certain level of proficiency in understanding and applying the standard of care. However, these certifications will not, in and of themselves, absolve the physician of liability, because the physician must still demonstrate that he or she adhered to the appropriate standard of care in any particular malpractice claim.

Second, physicians should not assume that their medical malpractice insurance policies cover claims arising out of medical services rendered on trips. Many insurance policies carry limitations on coverage for care delivered outside the scope of a physician's normal practice or beyond a certain geographic area. In these cases, trip physicians should seek extended coverage through their own insurance policies or through the tour organizer.

Both the plaintiff and the defendant typically rely on expert witness testimony to identify the standard of care and to offer an opinion regarding the defendant physician's adherence to that standard of care. Various state licensing boards are beginning to grapple with what their roles should be, if any, in sanctioning expert witnesses who give false or misleading testimony or who otherwise carry out their functions in a negligent manner. State licensing board policies on disciplining expert witnesses are not uniform across the country, but the American Medical Association (AMA) and many component state medical societies are advocating greater state government oversight of expert witness conduct and formal sanctioning when indicated, including possible loss of licensure. Regardless, any physicians wishing to serve as expert witnesses should closely scrutinize their qualifications and suitability to testify about wilderness medicine issues. The AMA House of Delegates has proposed the following minimum requirements for qualification as an expert witness (Policy H-265.994[3a]):

> *The AMA believes that the minimum statutory requirements for qualification as an expert witness in medical liability issues should reflect the following: (i) that the witness be required to have comparable education, training, and occupational experience in the same field as the defendant or specialty expertise in the disease process or procedure performed in the case; (ii) that the occupational experience include active medical practice or teaching experience in the same field as the defendant; (iii) that the active medical practice or teaching experience must have been within five years of the date of the occurrence giving rise to the claim; and (iv) that the witness be certified by a board recognized by the American Board of Medical Specialties or the American Osteopathic Association or by a board with equivalent standards.*

The negligence of a physician generally will not be imputed under a theory of respondeat superior (master–servant relationship) to the tour organizer or carrier. Many courts have noted that the relationship between a trip member and a physician is not a traditional activity over which a cruise ship or tour organizer has control. Moreover, a shipping or travel company is not in the business of providing medical services to participants and does not possess the requisite expertise to supervise a physician brought along for the convenience of the participants. Nonetheless, once the carrier or tour organizer undertakes to hire a physician, it owes a duty to participants to exercise reasonable care in the selection of a competent and qualified physician. To the extent that the company fails to discharge this duty, it may be subject to a cause of action for the negligent hiring of an incompetent physician. Therefore, cruise lines and tour operators should ensure that trip physicians are proficient in general medical care, emergency treatment, and the medical management of diseases endemic to the destination site.

Furthermore, travel companies and cruise ships should brief trip physicians on available resources, host country medical facilities, and evacuation procedures. Finally, companies should conduct formal credentialing of prospective trip physicians, verifying medical school graduation, board certification, and state licensure and researching any history of disciplinary actions.

DUTY TO RESCUE AND ABANDONMENT DOCTRINE

A physician who is on an expedition to Alaska and approaching the summit of Denali happens upon a trekker with multiple fractures and hypothermia. Does the physician have an obligation to treat the victim or attempt a rescue?

Abandonment is the unilateral termination of the physician–patient relationship by the physician without adequate notice to the patient, despite the need for ongoing medical care. In general, a physician has no legal duty to provide care for or to rescue endangered strangers. "The law imposes no liability upon those who stand idly by and fail to rescue a stranger who is in danger."[4] Moreover, abandonment occurs only in the presence of an established physician–patient relationship. Thus, the physician who refuses to enter into a physician–patient relationship and initiate treatment will not be held liable for abandonment.

Traditionally, once an individual initiated a rescue attempt, he or she could abandon rescue efforts at any time. "[The] motives in discontinuing the services are immaterial. . . . [The rescuer] may without liability discontinue the services through mere caprice or because of personal dislike or enmity toward the [victim]."[6] However, if by giving aid the rescuer has put the victim in a worse position than before the attempt to aid, the rescuer may be liable. For example, the victim may have relied on the rescuer's efforts to his or her detriment, foregoing other opportunities to obtain assistance during the rescuer's intervention.

The best risk management tool is to assume that physicians who either implicitly or explicitly agree to treat patients create a duty to provide continuity of care. Given these circumstances, physicians can generally avoid liability for abandonment under the following conditions:

1. The physician and patient mutually consent to terminate the relationship.
2. The patient dismisses the physician.
3. The victim no longer requires care, recovers, or dies.
4. The physician dies or is disabled.
5. Further rescue efforts would place the rescuer's life in danger.
6. The physician gives the patient reasonable notice of his or her intent to withdraw from the care of the patient and, particularly in an emergency, continues to treat the patient until another qualified health care provider takes over the case.

Improper termination of the physician–patient relationship may lead to a cause of action for breach of contract or professional negligence, as well as for abandonment.

STANDING ORDERS AND MEDICAL KITS

There is tremendous variability in the medical expertise of trip leaders and participants and in the resources, including medical equipment and supplies, that accompany treks and trips into

the wilderness. The delivery of medical care by nonphysicians represents one of the most problematic and controversial issues for trip organizers. Absent any representations to the contrary in travel brochures or other documents sent to participants, there is no implied or express contract to provide medical care to trip participants. Nonetheless, because of the nature of the business proprietor–customer relationship, trip leaders have some preexisting duties, namely, to exercise due care in the performance of their duties and in facilitating evacuations when feasible, as would be considered reasonable under the circumstances. In other words, if they have made no claims to the contrary, trip leaders and staff have no duty to render care beyond basic first aid services that an ordinary layperson could provide.

May a nonmedical trip leader or an EMT, paramedic, or other allied health care provider render medical services beyond the scope of his or her training or certification? One state board of registration in medicine defines the practice of medicine as[10]:

> *Conduct, the purpose or reasonably foreseeable effect of which is to encourage the reliance of another person upon an individual's knowledge or skill in the maintenance of human health by the prevention, alleviation, or cure of disease and involving or reasonably thought to involve an assumption of responsibility for the other person's physical or mental well being: diagnosis, treatment, use of instruments or other devices, or the prescription or administration of drugs for the relief of diseases or adverse physical or mental conditions. A person who holds himself out to the public as a physician or surgeon, or with the initials M.D. or D.O. in connection with his name, and who also assumes responsibility for another person's physical or mental well being, is engaged in the practice of medicine. The practice of medicine does not mean conduct . . . engaged in by persons licensed by other boards of registration with authority to regulate such conduct; nor does it mean assistance rendered in emergency situations by persons other than licensees.*

Most states require that an EMT or paramedic act under the supervision of a physician. Supervision usually entails direct observation, radio or telephone communication, or written guidelines, protocols, or standing orders (although usually with reasonable access to a physician who can respond to questions and provide requested guidance). These statutes were enacted under the assumption that physician consultation would be fairly accessible. Even with the advent of cellular telephones and telemedicine, however, many trekkers and travelers to remote regions cannot easily obtain physician consultation. In general, trip leaders and staff who practice beyond the scope of their education, training, and certification could be engaging in the unlawful practice of medicine.

Even in an emergency situation, it is unclear whether the Good Samaritan statutes protect trip leaders and staff. As previously discussed, travel companies and their trip leaders are to a certain extent compensated for safeguarding the safety and welfare of participants. Therefore it seems most likely that courts would use a reasonable care standard in determining the negligence of trip leaders. In other words, the court would determine how a reasonable trip leader with similar education, training, and certification would respond in similar circumstances, taking into account the emergent nature of the victim's illness or injury, the remoteness of their location, the scarcity of medical equipment, the limited means of evacuation, and the inaccessibility of definitive medical care.

On many expeditions and treks into the wilderness, medical kits stocked with prescription drugs, including controlled substances, are brought along. Again, failure of nonmedical trip leaders to provide these medications would not be considered negligence when doing so is in violation of the law, particularly when the trip organizer did not create the peril and provided adequate advance warnings to trip participants. If a nonphysician dispenses these drugs, it could very likely constitute the unlawful practice of medicine. Moreover, physicians who do not accompany the travelers but who nonetheless write the initial prescriptions to stock the medical kits expose themselves to lawsuits arising out of any malpractice on the part of the trip leader with regard to use of those medications. For example, a nonmedical trip leader might improperly diagnose and dispense medications or might overlook certain drug interactions. In addition, physicians who write prescriptions to stock medical kits face the risk of being disciplined by their respective state boards of medicine for inappropriate prescribing practices, particularly if medications are dispensed to an unintended recipient or if there is an adverse event.

This legal dogma of course is of little comfort to trip leaders who find themselves in emergency situations. In some instances, trip leaders on overseas travel have been known to purchase or to recommend that participants purchase medications that would require a prescription in the United States but that are available over the counter in foreign countries. Although the purchase and use of these medications is legal on foreign soil, travelers should be cautioned about the potential lack of guarantees regarding the safety, quality, and efficacy of locally procured drugs. If the nonphysician trip leader chooses to dispense medications, whether originally stocked in the medical kit before departure from the United States or obtained abroad, a court would in all likelihood judge any adverse consequences in accordance with a "due care" standard. That is, a trip leader who dispensed medications with resultant harm to the patient would be expected to demonstrate the due care of a reasonably prudent person under similar circumstances, taking into account the emergent nature of the medical condition, the remoteness of the trip, the availability of definitive medical care, and so forth. To avoid these awkward situations, trip organizers should adhere to the following basic risk management principles:

1. Do not advertise medical capabilities and resources beyond the scope of expertise of trip staff members or those resources readily accessible in the country.
2. Emphasize the need for trip participants to bring along an adequate supply of their own prescription drugs, and even verify that they have done so.
3. Caution trip participants about the lack of safeguards concerning the safety, quality, and efficacy of medications obtained overseas.

If trip organizers choose to equip staff members with medical kits, one expert in wilderness medicine recommends that the contents of the kit be based on the following factors[2]:

- The environmental extremes encountered during the trip
- Endemic diseases
- The medical expertise of the medical officer
- The medical expertise of the expedition members
- The number of people on the trip
- The responsibility for local health care

- The length of the trip
- The distance from definitive medical care
- The availability of rescue (i.e., helicopter)

MEDICAL RECORD KEEPING IN WILDERNESS MEDICINE

Good record keeping is of paramount importance in the practice of wilderness medicine. The medical record is both a health care document and a legal document. Although storage space and the portability of written or electronic medical records on trips to remote locations are often limited, trip physicians should nonetheless strive to be as thorough as possible in documenting medical care. The medical record functions as a complete, written, and chronological record of a patient's medical history, condition, and treatment. In medical malpractice claims, which are frequently litigated years after the alleged occurrence, these records may be the only written source from which the sequence of events and subsequent treatment can be reconstructed. Moreover, memories fade and witnesses become less available over time. Therefore, courts tend to give tremendous weight to the written record and will often assume that documented events occurred and undocumented events did not occur, particularly when the oral testimonies of the litigants conflict.

Although the length and content of entries vary with the specific circumstances, notes should at a minimum include the patient's chief complaint, the results of the physical examination (including normal findings), an assessment, and a treatment plan. The key point to remember in the creation of medical records is that they may be used to prove or disprove that the medical practitioner adhered to the appropriate standard of care. Therefore, physicians should record sufficient details to reflect their thought processes and to justify the care provided under the circumstances. Moreover, physicians should document events chronologically and as soon as feasible after the delivery of medical care. Once notes have been entered into the written or electronic medical record, the medical practitioner should never alter (or write or type over) the original notes. Entries should not be removed or inserted. If corrections are necessary, for written records, the physician may place a single line through the error, enter the correction, and initial and date the correction. For written or electronic medical records, the clinician may draft an addendum. Addenda should be placed after the last entry in the chart. Of course, notes should always be legible, signed, and dated by the health care provider making the entry. Upon completion of the trip, the company should treat these medical records as confidential information and should place them in locked medical files, granting access to designated personnel (company medical advisers or others with a need to know) only. Some states have explicit laws or regulations that dictate the minimum period for retention of medical records. In the absence of such laws, travel companies and cruise lines should store medical records for the period of time corresponding to the statute of limitations for tort claims in their states, which, although varying from state to state, is typically about 2 to 3 years.

CONCLUSION

The services of physicians on cruises and expeditions can be invaluable to the safety and welfare of participants. The expanded participation of higher-risk individuals on trips to more remote regions of the world increases the liability exposure of health care providers. Legal issues, particularly causation, become more complex when medicine is practiced in these unconventional settings. By following the risk management principles outlined in this chapter, physicians can continue to provide effective care while minimizing their liability.

The references for this chapter can be found on the accompanying DVD-ROM.

94 The Ethics of Wilderness Medicine

Kenneth V. Iserson and Barry Morenz

Ethics is the application of rules and principles to guide human action. Bioethics is the area of ethics that applies specifically to health care providers and biomedical researchers. It involves the encounter between the patient and the health care provider, biomedical research, and health care policy. Wilderness medicine and its applicable bioethics, however, are not directly comparable to either the medicine delivered in health care facilities or even the care delivered by urban emergency medical services. Wilderness medicine is unique, and its special attributes provide unique ethical problems (Table 94-1). For example, a hospital's working environment is rarely a factor considered by the hospital-based practitioner in the determination of what medical care to deliver, but the working environment is of major concern in the wilderness. Similarly, whereas patients are

- The length of the trip
- The distance from definitive medical care
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TABLE 94-1. Differences among Hospital Practice, Emergency Medical Services, and Wilderness Medicine

	HOSPITAL PRACTICE	EMERGENCY MEDICAL SERVICES	WILDERNESS MEDICINE*
Environment	Controlled, known, static	Partly controlled, partly known, changeable	Uncontrolled, partly known, changeable
Patient	Known, requests care	Unknown, sometimes requests care	Unknown, sometimes requests care
Equipment	Sophisticated	Adequate	Rudimentary
Security	Safe	Usually safe	Questionable
Personnel	Highly educated, definitive care	Highly educated, basic care	Variable education, basic care
Evacuation	Rare	Built into system	Major concern
Rescue	No	Rare	Common

*Includes search and rescue.

usually known to the hospital practitioner and arrive requesting care, neither condition is necessarily true in the wilderness setting. Even more striking are the differences between the hospital and the wilderness setting with regard to equipment availability, personnel training, the need for evacuation or rescue, and the provision for the security of those involved. All these differences can lead to unique ethical dilemmas.

This chapter provides an overview of ethical values in general and these values as they are applied to bioethics. It then describes a model for bioethical decision-making and adapts that model to wilderness medicine, taking into account the bioethics of wilderness health care providers, researchers, and, in some cases, makers of health care policy. Finally, unique dilemmas in wilderness medicine are discussed.

APPLICATION OF RULES, PRINCIPLES, AND ACTION GUIDES TO HUMAN ACTIVITIES

Values are acquired throughout life from many sources and develop into ethical action guides. In everyday situations, individuals may be unaware that these values are guiding their actions. However, when faced with situations that are rarely encountered, often termed "ethical dilemmas," people may question how they should apply their values to solve practical problems. Such situations often develop in wilderness medicine, since the settings can challenge practitioners to demonstrate expertise outside the usual scope of their medical specialties.

Both patients' and clinicians' values control patient–clinician encounters. When patients express their values, clinicians can get an impression of the patients' views about necessary treatment, desired quality of life, and other complex attitudes that control the willingness to seek and accept medical care. Whether these expressions are deemed valid is based on the clinicians' "bedside" determination of their patients' decision-making capacity. The clinicians' own values, both personal and professional, are also part of the relationship and sometimes conflict with the patients' values.

Ethical discussions often revolve around applying ethical rules in a consistent manner or in a way that could be applied by all practitioners in the same situation (universalizability). Ethical decisions derived from accepted principles, rules, or values should be applied consistently across all scenarios. If an accepted rule is that patients with decision-making capacity may make their own decisions about health care, this rule should be applied to all situations, not just when it is convenient for the health care provider. Likewise, if the rule is truly ethical, all health care providers, not just a privileged or unique group, must be able to apply it to practice.

Sources of Values

The basic elements of any ethical discussion are personal values. Values are the guideposts used to structure an individual's actions in life. They signify what a person's duties and responsibilities are, what is important to them, and how they interact with others. Thomas Aquinas said that the three vital things for each person are "to know what he ought to believe; to know what he ought to desire; and to know what he ought to do."[1]

Personal values derive from many sources: family, society, school, religion, the media, and professional training and related interactions. Family and religion generally guide the development of values in the formative years. For nearly everyone, these values form the bedrock on which his or her life is structured. Emphasizing the importance of early childhood learning is the maxim: If you control a child's life until the age of 6 years, he is yours forever. Additional significant influences are the media, schooling, and society. In this electronic age, the media begin to influence an individual's values early in life. Secular education broadens a child's experiences and values beyond the small world of the home. Finally, societal pressures continue to influence most individuals' value systems throughout life. Taken as a whole, different individuals' values derived from these multiple sources may conflict, leading to disagreements over which action to take when ethical dilemmas arise.

Professional schooling and interactions further refine how a person's values are applied. For example, one reason that medical students take anatomy courses is to destroy an ingrained cultural value against mutilating the dead. This allows them to accept and acquire the values of beneficial mutilation (surgery), handling the dead (resuscitations, pathology, transplants), and invading another's body (invasive medical procedures).[11] In addition, when exposed to clinical practice, medical students, nurses, medics, and other health care providers learn to adopt the values of their preceptors. In any residency program, trainees learn intrinsic professional values, and the majority of them behave remarkably like the faculty.

Values in Modern Biomedical Ethics

Another category of values, sometimes referred to as the *Georgetown bioethics catechism,* has emerged as an ideal for modern medicine, especially in the United States. These values include autonomy, beneficence, nonmaleficence, and distributive justice.

For the past two decades in the United States, the overriding professional and societal bioethical value has been a patient's autonomy. **Autonomy** recognizes an adult's right to accept or reject recommendations for his or her personal medical care (even to the extent of refusing all care) in the presence of appropriate decision-making capacity (see Decision-Making Capacity and Consent). Current bioethical opinion demands that clinicians respect patient autonomy. This is the counterweight to the long-practiced paternalism (or parentalism) of the medical profession, wherein the physician alone determined what was good for the patient. Coupled with paternalism is coercion, the threat or use of violence to influence behavior or choice. The august figure in white (or in a medic's or search-and-rescue uniform) who implies that there will be dire consequences if medical recommendations are not followed remains a potent challenge to patient autonomy.

At the patient's bedside, **beneficence,** the act of doing good, and confidentiality, the holding of information in confidence, have been long-held and nearly universal tenets of the medical profession. Likewise, personal integrity, the adherence to one's own moral and professional standards, is basic to ethical thought and action. The basic tenet taught to all medical students is **nonmaleficence,** or "First, do no harm." This credo, often stated in the Latin form, *Primum non nocere,* derives from the historical knowledge that patients' encounters with physicians can be harmful as well as helpful. It recognizes every physician's fallibility.

The concept of comparative or **distributive justice** suggests that comparable individuals and groups in society should share equitably in the benefits and burdens of that society. Many society-wide decisions about the allocation of limited health care resources are based on this principle. Yet, it is a fallacy to extrapolate from this valid principle the idea that individual clinicians can arbitrarily limit or terminate care on a case-by-case basis simply because there exists a need to limit resource expenditures.[18]

Decision-Making Capacity and Consent

A significant concern in the wilderness setting is whether patients have decision-making capacity, thus retaining their autonomy to make reasonable health care decisions. Many ethical dilemmas in emergency medical care revolve around ascertaining a patient's decision-making capacity, often linked with consent to (or more often refusal of) a medical procedure. Since a basic canon of both ethics and law, as stated by Justice Cardozo, is, "Every human being of adult years and sound mind has a right to determine what shall be done with his own body,"[26] these decisions about what action to take can often be made clearer by understanding what is meant by decision-making capacity and how it relates to consent. (Note that the word *competent* is often used when *capacity* is really what is meant. *Competent,* meaning, "possessing the requisite natural or legal qualifications," is a legal term and can be determined only by the court.[22])

Box 94-1. Components of Decision-Making Capacity

Knowledge of the options
Awareness of the consequences of each option
Appreciation of personal costs and benefits of options in relation to relatively stable values and preferences

Buchanan AE: The question of competence. In Iserson KV, Sanders AB, Mathieu D (eds): Ethics in Emergency Medicine, 2nd ed. Tucson, AZ, Galen Press, 1995.

Capacity refers to a patient's decision-making ability as determined by clinicians. Capacity is always decision-specific rather than global. Although an inebriated person, for example, may have the capacity to refuse to have stitches put into a small laceration, especially with evidence that he has previously refused similar treatments without remorse, the same individual may not have the capacity either to accept an elective operation of any sort or to refuse an emergency lifesaving procedure or operation. To have adequate decision-making capacity in any particular circumstance, a person must understand the available options and the consequences of acting on the various options and be able to compare any option he or she chooses against the costs and benefits related to a relatively stable framework of personal values and priorities (Box 94-1).[3,4] This last requirement is the most difficult to understand and requires a subjective interpretation. The easiest way to assess it is to ask why an individual made such a decision. Disagreement with the physician's recommendation is not in and of itself grounds for determining whether a person is incapable of making his or her own decisions. In fact, even the refusal of lifesaving medical care may not prove the person is incapable of making valid decisions, if it is made on the basis of firmly held religious beliefs, as in the case of a Jehovah's Witness refusing a blood transfusion.

A person must be permitted to consent to or to refuse any medical intervention (implicitly or explicitly) if he or she has decision-making capacity for that decision and if the clinician respects the patient's autonomy. Three general types of consent exist: presumed, implied, and informed. **Presumed consent,** sometimes called emergency consent, covers the necessary lifesaving procedures that any reasonable person would wish to have if lacking decision-making capacity; controlling hemorrhage and securing an airway in an unconscious victim of a fall are common examples. **Implied consent** is when a person with decision-making capacity cooperates with a procedure, such as holding out an arm to donate blood or to allow initiation of an intravenous line. **Informed consent** is when a person who retains decision-making capacity is given all the pertinent facts regarding the risks and benefits of a particular procedure, understands them, and voluntarily agrees to undergo the procedure.[9]

Questions applying to consent in the wilderness setting can be difficult. Does the victim have the capacity to understand the situation? Will decision-making capacity be questioned only if a person refuses "good" medical care? Also, unresolved even in

standard medical practice, what procedures require informed, as distinct from implied, consent? The requirement to obtain informed consent varies in practice and law from area to area. This variation stems from differing local practice standards and state law and disparities in physician training. Determining decision-making capacity and providing an opportunity for a victim to consent to a procedure when appropriate are crucial to respecting a patient's autonomy.

Security: Another Value Applicable to Wilderness Medicine

Security, or safety, signifies a measure of responsibility toward oneself, one's companions, and the patient. In the unique setting of wilderness medicine, this responsibility extends to the wilderness team's safety from the environment, victims, and their own poor judgment, a concept more familiar to emergency medical services (EMS) personnel than to health care providers in normal medical practice. However, this value is of paramount importance in wilderness medicine. Security is the responsibility of any wilderness medical provider, even if he or she is not officially designated a provider but must take over in a medical crisis on the basis of special knowledge or skills. Decisions about rescue, evacuation, terminating group travel, or even attempts to perform certain medical interventions must include security considerations.

Security is wilderness medicine's controlling value in most circumstances. Concerns about safety are applied in the following order: oneself, other team members, and the patient. Ethical theory supports this hierarchy. Beneficence by medical personnel does not imply a need to endanger oneself, and, indeed, if medical skills are to be useful, medical personnel must be able to render care. In addition, inherent in any leadership position is a responsibility to protect one's team. Therefore, the team members' safety is the second responsibility. Finally, the patient's security should be ensured, but never at the expense of the medical team's safety. This is to say that in unknown or unknowable circumstances, the medical leader may have to weigh potential risks against benefits. All risks must be considered in these "calculations," such as in the case of a badly injured trekker who might survive if evacuated by aeromedical transport. If the helicopter team is willing to attempt a pickup, the wilderness medical care provider must determine whether local conditions are sufficiently safe, balanced against the chance of benefit to the patient, to justify the request.

One such example illustrating security issues occurred in the Pacific Northwest near Mount Baker. A group of adults and adolescents were on a hike above some snowfields when two parents and their daughter decided to glissade down one of the fields, something they had done before. As the mother and daughter sped over a crest, they dropped into a crevasse and were injured. The father pieced together what had happened and sought help. Eventually, a group of climbers were enlisted. No one was eager to descend into the trench but one man from the climbing group agreed to be lowered on a rope, telling the group, "Just make sure you get me out."

The ethical question here is how much risk and responsibility untrained volunteers have in this type of wilderness crisis. A second issue that has to be considered is the capability of the group to attempt a rescue without endangering themselves and possibly creating a need for a second rescue. As a member of the hiking group, the father above had a responsibility to help; however, since he was technically incapable of the rescue, his only responsible avenue of action was to seek help. Bystanders, on the other hand, have no fundamental responsibility to help or to assume any risk beyond what they are willing to assume. The man who agreed to be lowered into the crevasse would have been acting ethically if at any point in the rescue attempt he had signaled to the group to pull him up without helping the victims or if he had walked away without allowing himself to be lowered into the trench in the first place. Despite entreaties from others, bystanders need not justify their participation or nonparticipation to anyone but themselves.[23]

In contrast, Ernest Shackelton, the appointed leader of a 19th-century attempt to be the first to reach the South Pole, did have a responsibility to do his utmost to see his men safely home. During the voyage, their ship broke up in the ice, and the men had to pull lifeboats over ice to reach open sea, struggling against all odds to reach safety. Shackelton's steady and undaunted leadership is credited with helping get all his men to safety.[19]

A unique ethical problem that arises in wilderness settings—and that has often led to disasters—is when the team, especially the nonmedical team leader, ignores or overrides the medical person's decision. Individual team members have been harmed and multiple team members lost because factors other than the team members' safety and well-being were given priority.[16,25] Heeding the demands of security is especially important because the majority of people who are in the wilderness have risk-taking personalities, leading them to downplay security in favor of adventure.

Utility

In the language of ethics, utilitarian thinking plays a dominant role in wilderness ethics. Utilitarianism is the philosophy that promotes the greatest good or happiness for the greatest number of individuals. When applied to wilderness medicine, it promotes the well-being of the many over the well-being of the individual. This can be defended by simply recognizing the unique aspects of wilderness medical practice, such as the uncontrolled environment, unfamiliarity with the patient, rudimentary equipment, and changeable situations—all contributing to safety concerns.

The ultimate application of utility in remote settings was described in the great survivor story of the men of the *Essex*, the doomed whaling ship that was the basis for Herman Melville's *Moby Dick*.[24] As was common after shipwrecks, the men drew lots to decide who would be sacrificed and die so that the others in the small boat could live a little longer without starvation.[27] One can argue that if all the men consented to this process, then it was ethical, but the very nature of the situation put each man under such extreme duress that it would be questionable if any man's consent could be considered voluntary. In these types of extreme circumstances, the ethics of draconian decisions such as survivor cannibalism are always fraught with paradoxical ethical dilemmas.[11]

Ethical Dilemmas

Health care professionals commonly apply their values without much deliberation. They act instinctively based on their prior behavior and training. Values are constantly (although not nec-

essarily consistently) applied to everyday decisions. Of course, most decisions are not ethical decisions. Ethical dilemmas arise with a conflict between two seemingly equivalent values that are represented by different and mutually exclusive possible actions.

An example of a bioethical dilemma in wilderness medicine may help illustrate ethical decision-making. A distress call has been received from anxious relatives or by radio from a plane flying over a wilderness area. The victim is in a hazardous area or, more commonly, is caught in terrible weather. The clinician directing a search-and-rescue team must decide how to respond to the call in a setting that may put the team in danger. The standard bioethical value of beneficence directly competes with the bioethical value of security in wilderness medicine. Each has a strong pull on the decision maker, with each value providing good arguments for sending or not sending the rescue team. Although the value of security may often be considered paramount in the wilderness setting, the emotional and altruistic pulls of beneficence make this a difficult choice. Considering this case, a word should first be said about rights and duties in relation to health care.

Although the word *rights* is glibly used in many situations, a personal right is present only if another person or society as a whole has an identifiable duty to the individual. For example, if all U.S. citizens had a right to health care, the government would have the duty to provide this care. Health care providers would have a corresponding duty to deliver this care. However, except in limited circumstances, such as emergency departments and prisons, no such legal right currently exists. Morally and practically, citizens do not have the right to health, and how much health care people are owed is unresolved.[17] Correspondingly, no health care practitioner has a duty to provide all the health care people desire or need. Practitioners do, however, have a duty to provide security, when possible, for those they direct in wilderness settings.

Solving the Bioethical Dilemma

Because an ethical dilemma arises when two or more seemingly correct actions appear to have equal benefits, the choice of actions should be examined first. How are these proposed "correct" actions determined in the first place? After that, which of these actions is the more ethically acceptable?

Deciding on an Action in the Standard Setting. Jonsen and colleagues[15] have suggested four groups of factors to consider when determining a course of action in the face of a bioethical dilemma in the standard clinical paradigm. These include the medical indications for the action, the patient's (or surrogate's) preferences, consideration of the quality of life, and other contextual factors. These have been seen as an "ethical square," with the top two boxes weighing more heavily (Fig. 94-1).

In a broad sense, patient wishes equate to patient autonomy. In a growing number of instances, however, especially at the end of life, surrogate decision makers, rather than the patient, may exercise this autonomy. This may also be true in the wilderness setting, albeit with less legal backing. Many critically ill patients in health care facilities have an advance directive (durable power of attorney for health care) naming a surrogate to make health care decisions if they lack decision-making capacity. Finding such advance directives in the wilderness setting would be rare and, in emergency situations, may not be valid, especially if the message is in an unusual form, such as a tattoo or MedicAlert jewelry.[8,14] Experience has shown that these cannot be relied on to reflect a person's current wishes (e.g., tattoos) or can easily be misinterpreted (e.g., do-not-resuscitate jewelry).[12,14] Furthermore, it is not the wilderness practitioner's job to interpret either nonstandard legal or quasilegal documents that are not part of his or her training. Standard forms, such as area- or state-wide prehospital do-not-resuscitate forms or advance directives, if present, should be followed. Many of these, however, are specifically not applicable after trauma.[9,10]

If a person lacks decision-making capacity, a spouse, close friend, or medical care provider makes critical decisions for the patient, acting in his or her best interest. Parents normally act as decision makers for minor children. In rare cases, a court-appointed guardian is named to replace parents. A child may also be an *emancipated minor,* meaning that he or she lives alone and is self-supporting. Two key issues arise in children's medical care. First, children should not be denied emergency care simply because no permission is available. In these cases, emergency is defined broadly. Anything that causes pain or that may increase the child's morbidity or mortality should be treated. In wilderness situations, reasonable efforts to get parental consent usually do not apply if they are not already on the scene. They may have given blanket consent to treat their child, but that is not required if an emergency exists. Second, if the child is old enough to understand the situation and to cooperate, it is both ethically important and good medical practice to have him or her assent to the proposed treatment. The assent is the agreement but does not constitute legal permission, since the child is underage.[9]

Medical indications are often more straightforward in the wilderness setting than they are in standard health care. In the wilderness, treatment is basic, injuries and illnesses are generally acute, and intervention is normally life preserving rather than death prolonging. The clinicians use standard clinical algorithms for their appropriate level of training and expertise. In remote areas, of course, questions may arise about whether an ophthalmologist should attempt to reduce a hip dislocation or whether a nurse should attempt to establish a surgical airway. These dilemmas should, when feasible, be decided with input from the patient or surrogate. As a matter of proper planning, behavior in critical scenarios must be decided in advance. In general, however, medical indications are clear.

Bioethicists normally feel most comfortable helping to resolve cases using only the medical indications and patient (or surrogate) wishes, which are all above the double line in Figure 94-1. When these factors are ambiguous, however, two other sets of factors must be considered: contextual factors and quality of life. In the wilderness setting, the primary contextual factor is safety. This may overshadow all other considerations involved in a victim's treatment. Other contextual factors include financial implications of various treatments and the effect of various options on other trip members. In the standard medical situation, this is, admittedly, a fuzzy area. Related to these, and even more nebulous, are quality-of-life factors. These relate to the nature of a person's current and presumed future existence as viewed by others. For those who retain decision-making capacity, their autonomous decisions reflect their view

MEDICAL INDICATION	PATIENT WISHES
1. What is the patient's medical problem? Prognosis? 2. Is the problem acute? Chronic? Critical? Emergent? Reversible? 3. What are the goals of treatment? 4. What are the probabilities of treatment success? 5. What are the plans in case of therapeutic failure? 6. In sum, how can this patient be benefited by medical interventions, and how can harm be avoided?	1. What has the patient expressed about treatment preferences? 2. Has the patient been informed of benefits and risks, understood, and given consent? 3. Does the patient have decision-making capacity? What is the evidence of incapacity? 4. Has the patient expressed prior preferences, e.g., advance directives? 5. If the patient is incapacitated, who is the appropriate surrogate? Is the surrogate using appropriate standards? 6. Is the patient unwilling or unable to cooperate with medical treatment? If so, why? 7. In sum, is the patient's right to choose being respected to the best extent possible?
QUALITY OF LIFE	**CONTEXTUAL FEATURES**
1. What are the prospects, with or without treatment, for a return to patient's normal life? 2. Are there biases that might prejudice the provider's evaluation of the patient's quality of life? 3. What physical, mental, and social deficits is patient likely to experience if treatment succeeds? 4. Is the patient's present or future condition such that he or she might judge continued life undesirable?	1. SAFETY ISSUES. (In wilderness medicine, these are often the most important considerations.) 2. Are there family issues that might influence treatment decisions? 3. Are there provider (SAR or trip member) issues that might influence treatment decisions? 4. Are there financial and economic factors (evacuation/rescue costs)? 5. Are there problems of allocations of resources? 6. What are the legal implications of treatment decisions? 7. Any provider, organization-related, or institutional conflicts of interest?

Figure 94-1. The ethical square. SAR, search and rescue.

of life. In the wilderness setting, time and circumstances usually do not allow clinicians to make quality-of-life judgments.

Deciding on an Action in the Wilderness. The importance of security factors in the wilderness setting leads to the altered diagram of decision making for ethical problems in wilderness medicine (Fig. 94-2). This includes three groups of factors to consider when deciding on a course of action: patient autonomy, security, and medical indications. Within this decision-making model, security factors must be given the most weight.

Security factors include the safety of the medical and rescue personnel and victim, the proposed procedures, and the evacuation method. As mentioned previously, the security of the medical team is a valid consideration because of the inherent risk-taking nature of people in the wilderness. In recent legal actions pertaining to wilderness injuries, the law has recognized a "doctrine of reasonable implied assumption of risk." This implied risk is also part of an acceptable concept of wilderness triage. Wilderness triage takes place when the same injuries or illnesses that would cause minimal morbidity in a medically sophisticated environment inevitably cause death if they occur in the wilderness. A fractured femur in the lone wilderness traveler or an abdominal gunshot wound in a remote area is often a virtual death sentence. This is a risk that wilderness adventurers take, although not always with a clear understanding of the enormity of the risk.

Bioethical Decision-Making Process[13]

Both standard bioethics and wilderness medical ethics often involve difficult situations with no "correct" answer. Usually, more than two possible actions exist. When faced with such a dilemma, what should the practitioner do?

Using an Algorithm as a Guide for a Decision

In bioethics, although disagreements may arise regarding the optimal course of action using a specific set of values, general agreement often exists as to what constitutes ethically wrong actions. The method of ethical case analysis described in Figure 94-3 is designed to provide the emergency practitioner with prompt assistance in selecting an ethically correct, although not necessarily a theoretically "best," course of action.[12] This method applies equally well in both the wilderness setting and the normal hospital setting.

The first step in using the algorithm in Figure 94-3 is to use a known precedent. This is the simplest solution to an ethical dilemma but requires planning in advance, including reading and thinking about ethical problems. Many physicians and

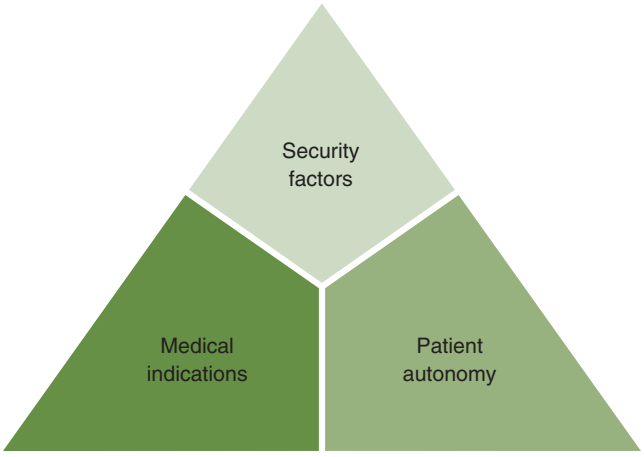

Figure 94-2. Wilderness medicine's ethical triangle.

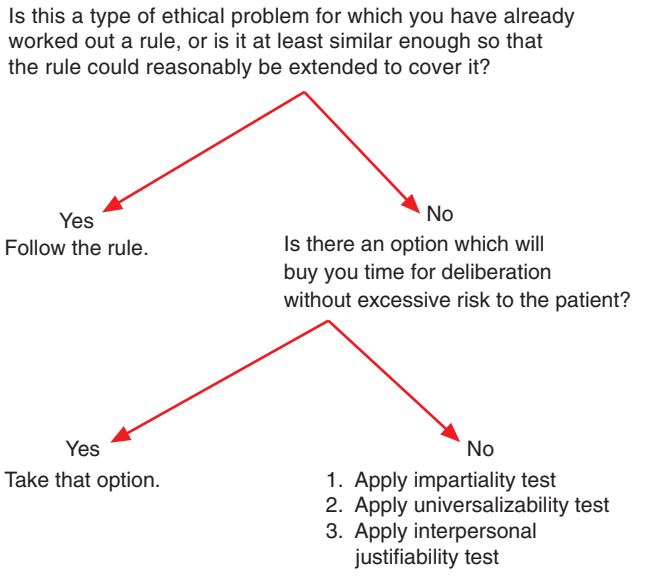

Figure 94-3. A rapid approach to emergency ethical problems. (Modified from Iserson KV: An approach to ethical problems in emergency medicine. In Iserson KV, Sanders AB, Mathieu D (eds): Ethics in Emergency Medicine, 2nd ed. Tucson, AZ, Galen Press, 1995.)

other health care professionals are not prepared to do this. Just as with any emergency procedure, wilderness medicine physicians and health care professionals should be prepared with a course of action for the most common ethical dilemmas likely to occur in the wilderness setting.

With no precedent, the second step is to "buy time." What action will not be harmful to the patient and will provide time for the consultation or information gathering needed to refine the action plan? In a wilderness medical setting, this might mean placing a person's arm in a sling for comfort while deciding whether an inexperienced provider should attempt to reduce a dislocation or fracture.

With no precedent on which to rely and no way to buy time, the health care professional must select a possible course of action and test it for ethical viability. The impartiality test, the universalizability test, and the interpersonal justifiability test are drawn from three different philosophical theories. First, the **impartiality test** is applied. The practitioner asks whether he or she would ask to have this action performed if they were in the patient's place. In essence, this is a form of the Golden Rule, "Do unto others as you would have done unto you." According to John Stuart Mill, this espouses "the complete spirit of the ethics of utility."[21] Second, the **universalizability test** asks if the health care professional would feel comfortable having all practitioners perform this action in all relevantly similar circumstances. This generalizes the action and asks whether developing a universal rule for the contemplated behavior is reasonable. This is merely a restatement of Kant's categorical imperative: "Act as if the maxim of thy act were to become by thy will a universal law of nature."[15a] Finally, the **interpersonal justifiability test** asks if the practitioner can supply good reasons to others for his or her action. Will peers, superiors, or the public be satisfied with the action taken and reasons for it? This test uses David Gauthier's basic theory of consensus values as a final screen for a proposed action.[6] If all three tests can be answered in the affirmative, the health care professional can be reasonably assured that the proposed action falls within the scope of morally acceptable actions. If, however, the proposed action fails any of these tests, the algorithm must be applied to another proposed action.

ETHICAL DILEMMAS IN WILDERNESS MEDICINE

With its unique setting and mode of practice, wilderness medicine provides practitioners with ethical dilemmas that are rarely seen by most other providers. These dilemmas can be grouped into three categories: standards of care, priority in care, and the decision-making process (Box 94-2). As might be expected, some of the issues in each group deal with provider–patient dilemmas, while others have more to do with group or governmental policies. These dilemmas have few parallels in other areas of medical practice, except perhaps battlefield medical practice or medical care during major disasters, resulting in ethical decisions that differ from those in standard medical settings. Such dilemmas include providing euthanasia for potentially nonfatal medical conditions, abandoning patients, and prioritizing medical care between original patients and rescue team members. The ethical decision-making process used to sort through these dilemmas, however, is similar to that used in other settings, a basic truism sometimes obscured by the unique setting and issues of wilderness medical care. A limited discussion of these ethical dilemmas and the values involved follows.

Box 94-2. Ethical Dilemmas in Wilderness Care

STANDARD-OF-CARE DILEMMAS
- Limited resources: The standard of care differs. What should be brought into the field? How are resources distributed?
- Cultural: Are Western standards of care and attitudes appropriate when treating locals in a foreign country?
- Nontrained personnel: How much authority is delegated to nontrained personnel?

PRIORITY-IN-CARE DILEMMAS
- Triage choices: Who should be rescued first? (Those most injured or ill? Injured or ill rescuers? Those with the best chance of survival? Women and children? Those with important information, such as scientists who have collected data? Those who do not volunteer to stay behind?)
- Issues of survival
- Issues of direct life-threatening situations for the provider or providers
- Motorized vehicle restrictions and environmental protection in wilderness areas

DECISION-MAKING DILEMMAS
- Unavailability of a surrogate or a family member
- Euthanasia
- Lack of ethics consultation
- Advance directives
- No-rescue areas

Standard-of-Care Dilemmas

Limited Resources

In the wilderness setting, resources are limited. Medical equipment is usually confined to supplies that can be carried into the field on foot or, in some cases, on horseback or by helicopter. Moreover, wilderness rescue personnel may have limited medical skills. The combination of limited skills and limited availability of supplies and equipment gives rise to ethical dilemmas. What should be included in wilderness medical kits? Their composition is resource allocation at its most basic. And who makes the decision?

Rarely do people consider advanced decision making a part of medical care. However, it is very much a part of wilderness medicine. For example, decisions regarding the contents of medical kits made well in advance rather than during triage affect the patient's care. Although the individual wilderness traveler usually determines what is carried into the field, he or she generally fails to realize that this decision may set a limit on treatment. Any traveler into a wilderness area must assume that the contents of the medical kit will be the only resources available for medical treatment. Although the group, a medical committee, or the medical director or advisor selects equipment for organized wilderness excursions or search-and-rescue teams, the selection still limits the medical care that can be given.

Although commercially available standardized medical kits are usually designed on the basis of "medical" criteria, it is vital to recognize that some types of treatment will be implicitly unavailable because of what is excluded from these kits. No one is expected to carry a fully stocked emergency department into the field, but clearly identifying the ethical dilemmas entailed in compiling these kits helps team members in their decisions. For example, if a decision not to carry antiarrhythmic medications or a defibrillator is made and if a team member suffers a cardiac rhythm abnormality, there will be little that can be done for him or her. Some people may omit medical kit items that could be useful, such as intravenous solutions. As the medical person on one doomed expedition to the Himalayan peak Nanda Devi recalled, "[My] irritation grew as [I] remembered [being] pressured into leaving intravenous fluids behind."[25] Such pre-expedition resource decisions may jeopardize a team member. It helps if team members know in advance that these decisions were made—and even participate in making them.

Explicit triage decisions, although harder to make, are often easier to recognize as ethical dilemmas. These are discussed later in the chapter.

Cultural Differences

Many wilderness emergencies occur in places outside of the United States or other Western countries. Are Western standards of care and attitudes appropriate when treating locals in a foreign country? Whose values control medical treatment and other actions?

Three circumstances may present ethical dilemmas in the delivery of medical care during expeditions to remote areas. The first is a lack of cultural sensitivity. Aggressive offers to care for disease or injury may frustrate or anger local patients or providers, whose methods of treatment fit within the region's cultural milieu and may be as good as or better than "modern" medicine. Temporarily replacing or upstaging traditional healers and their methods may degrade them in the eyes of the local population.

The second situation is when medical problems occur that are beyond the capabilities of an expedition's practitioners. After offering the care for which they are competent, practitioners may feel obligated to attempt treatments beyond their knowledge or abilities. An internist may face treating a gunshot wound to the chest, a psychiatrist may encounter a complicated obstetric emergency, or a paramedic may confront an epidemic. Often without any direction except a moral compass, these caregivers may be tempted to stretch their abilities beyond the limits of patient safety. Cultural ethical concerns should be considered when deciding which course of action to pursue.

The third situation relates to the larger question of the fairness of chance encounters: a woman's life is saved through the luck that a passing trekker could treat her pyelonephritis; after a surgeon relocates a hip, a man will continue to provide for his family; and a paramedic happens to be on hand to intubate a child with epiglottitis. These situations in themselves rarely encompass ethical issues. The larger question, which may be more philosophical than practical, is how these interventions interfere with the balance of life in the area. Are chance encounters an aberration or simply a part of life? One of the most common situations in this category is a wilderness team from a developed country leaving medications behind with individuals who would not normally have access to them. Beyond the questions of the medications' efficacy, continued availability, and safety in inexperienced hands, there are the ethical concerns about interfering and altering the life balance in other cultures.

Trekkers who traverse areas that others commonly visit do not face this dilemma, because medications are routinely distributed by the succession of groups. This question arises, however, in expeditions entering rarely visited areas, such as remote areas in Papua New Guinea or the Amazon basin.

How Much Authority Is Given to Nontrained Personnel?

Wilderness travelers face ethical dilemmas when they encounter medical situations for which they are untrained. This is certainly not restricted to laypersons. Medics, physician assistants, nurses, and physicians may quickly find themselves out of their depth in a wilderness setting. This occurs when they treat patients with conditions comfortably treated only in an urban environment or when an illness or injury is beyond the scope of personal experience and knowledge. In deciding whether to intervene in such a situation, the person planning to help must weigh the chance of benefiting the patient (value of beneficence) against the chance of doing the recipient of care harm (nonmaleficence).

The following hypothetical case illustrates both the questions raised in this type of dilemma and the application of the Rapid Approach to Emergency Ethical Problems (see Figure 94-3). A backcountry excursion sets out with a medical provider who is unprepared for orthopedic emergencies. When a group member dislocates her shoulder, the provider is unwilling to go beyond his level of training by attempting shoulder relocation, although the victim (as well as the rest of the party) encourages the attempt. Another member of the party with even less training volunteers to attempt the maneuver; the clinician is then in a double bind, seemingly forced to either overextend his or her skills or to acquiesce to even less knowledgeable medical care for the victim.

How could this dilemma be resolved using the Rapid Approach to Emergency Ethical Problems? The first step would be to anticipate such a situation in advance and to plan a course of action. Because orthopedic trauma is common in the wilderness, any medical provider should expect to face such a situation. (Note that planning may obviate this ethical dilemma, as it does in many other situations, since the provider may then learn the requisite orthopedic knowledge and skills in advance or may abandon plans to assume this wilderness medical role.) Whether or not the skill level is unchanged, the provider may also decide on an ethical course of action after discussing the potential problem in advance with knowledgeable peers or acquiring information from other sources. Perhaps the provider has previously decided to act in such situations (his or her paradigm in the Rapid Approach to Emergency Ethical Problems). It is reasonable to base intervention on (1) determining whether the patient has decisional capacity, (2) informing the patient fully and honestly of the apparent situation and options, and (3) acquiescing to the patient's desires, whether attempting relocation or simply securing the arm in place. Honest acceptance of the patient's autonomy to control his or her medical care often resolves a seemingly difficult ethical dilemma.

If the provider believes that the "experienced" layman offering to help has insufficient knowledge, the provider must then decide whether the paradigm case for which he or she prepared a response is similar enough to the current circumstances to use. If it is, the dilemma is resolved, and that rule should be followed. If, however, the provider believes that the current situation differs significantly from the paradigm case or if he or she has simply failed to decide in advance on an ethical course of action, the provider should move on to step 2: buying time. In the scenario presented, buying time may consist of making the patient comfortable before contacting help or thinking through the problem. Help may be available to organized wilderness excursions through radio or cellular telephone communication. The assistance may be experienced advice about other actions to resolve the dilemma or orthopedic advice on ways to reduce the shoulder. Sometimes, however, no help is available or not enough time can be bought to secure help. In that case, the health care provider must make a decision to act.

At this stage, step 3, the provider attempts to choose an action that is ethically acceptable (by applying Figure 94-2), even if it is not the optimal action he or she might select if more time were available to consider the problem. Possible actions in this case might include attempting a reduction, allowing the layperson to attempt a reduction, simply immobilizing the victim's shoulder, leaving the victim and going for help, or ignoring the situation and leaving the decision to someone else. The provider must first choose a course of action (remembering that not deciding is also a course of action) and then decide whether the choice falls within the scope of ethically acceptable behavior. If, for example, the proposed action is shoulder immobilization, the three tests of impartiality (golden rule), universalizability (should every practitioner do as I plan to do?), and interpersonal justifiability (would I be ashamed to have my actions publicized?) should be applied to this action. If the action passes all three tests, it is probably ethically acceptable and may be used. Remember that ethically acceptable actions may differ with the circumstances or the wilderness group involved.

Health care policy is another aspect of this type of ethical dilemma. Restricting medical practitioners from fully using their skills and knowledge may limit wilderness medical care. Paramedics, for example, are told that in some jurisdictions, on penalty of losing their licenses, they may not reduce fractures, perform cricothyrotomies, or, in a few locations, perform endotracheal intubations. Emergency medical technicians, first aiders, first responders, and the like are more severely restricted. Nurses may not know what procedures their licenses allow, and physicians are constantly concerned about liability. In general, many practitioners in wilderness settings feel that the laws and administrative policies under which they work restrict their actions. This attitude and their subsequent behavior may lead to substandard care for victims of wilderness injury or illness. The Wilderness Medical Society and other groups have begun working to overcome these limitations. Currently, however, an ethical dilemma may exist when practitioners face medical situations in the field that they know how to treat but that exceed their licenses or official certifications. A clear conflict may exist between the law and ethical responsibility. Practitioners have to decide the best course of action, preferably in advance of the problem.

Priority-in-Care Dilemmas

Triage takes on new dimensions in wilderness settings. Ethical dilemmas easily arise when health care providers face not only triage among victims but also critical decisions about whether to help victims at all. These settings also produce situations in which the rescuers or other members of the party may be placed in danger by helping an injured person.

Triage Choices: Whom to Rescue First and How to Distribute Resources

Medical practitioners, especially those in the fields of surgery and emergency care, are familiar with medical triage in which multiple patients need care and in which patients must be sorted by severity of injury, availability of resources, and possibility of successful treatment. These triage decisions have their own unique set of ethical dilemmas. Wilderness triage is unusual on several counts and may present ethical dilemmas markedly different from those encountered in non-wilderness environments.

Three ethical dilemmas result from wilderness triage questions that are unlikely to occur elsewhere, with the exception of battlefield settings.[5] The first dilemma arises when the wilderness practitioner knows all the victims and may have personal ties to at least one. This is unlike normal triage scenarios and complicates any decision about who receives treatment, especially if resources are limited. For example, in an outbreak of giardiasis in a party of twelve, the provider may have only enough metronidazole (Flagyl) to treat five people. Another, more serious example would be a lightning strike in the midst of six people, with only one other individual capable of providing assistance. In each case, the medical practitioner applying triage criteria may be torn between medical and personal concerns.

A second ethical triage dilemma arises in what may be termed the "us-versus-them" situation. Members of both the wilderness party and the local population may be in the victim pool to be triaged. To whom does the provider owe primary responsibility? Some may argue that the implicit or explicit contract between the provider and the group members warrants treating group members first. Yet, in the battlefield setting, which may often be analogous to the wilderness setting with regards to medical ethics, the Geneva Convention specifies that patients are always to be triaged for medical care *on the basis of medical need and the ability to treat*. Whether military caregivers follow this dictum in practice is moot. The wilderness caregiver must carefully consider this issue before venturing into the field.

Finally, ethical dilemmas arise because not all team members are equal. If triage among team members is necessary, treatment on the basis of pure medical necessity is not always realistic. In the giardiasis example, will the sickest patients be treated or will treatment be given to the less sick guide and translator, who are needed to lead the party safely out of the wilderness? The greatest good for the greatest number, or the concept of group safety, must prevail. However, this may be neither a comfortable nor an intuitively obvious decision.

An ethical dilemma also arises when a rescue team member is injured while out in the field. The question is asked whether rescue teams should treat their injured team member before, or instead of, victims. Wilderness rescue is an inherently dangerous operation. Although the safety record of some organized and experienced rescue groups has been excellent, this is not universal, particularly with ad hoc rescue attempts.[7] Where should the team's priorities lie? Again, an analogy can be drawn with triage parameters in emergency care. The principle of triage is that as long as resources are available, the most seriously injured are treated first. Those that cannot be saved with available resources or be evacuated in time to be saved are given only comfort measures. This situation logically and morally prevails in wilderness medical care. However, emotion rather than reason often influences actions, so the wilderness health care provider must ensure that ethical decision-making prevails.

Issues of Survival

In some situations, the lives of expedition members may be put at immediate risk if an injured person receives optimal assistance. One well-known example is high-altitude climber Simon Yates, who, while trying to lower his injured climbing partner, Joe Simpson, down to base camp in the Peruvian Andes, found himself in a situation in which he had to either cut the lowering rope tethering his partner, almost assuredly killing him, or risk also dying himself.[28] (He chose to cut the rope and, unbelievably, Simpson survived.)

> *I couldn't help him, and it occurred to me that in all likelihood he would fall to his death. I wasn't disturbed by the thought. In a way I hoped he would fall. I knew I couldn't leave him while he was still fighting for it, but I had no idea how I might help him. I could get down. If I tried to get him down I might die with him. It didn't frighten me. It just seemed a waste. It would be pointless. . . . The knife! The thought came out of nowhere. Of course, the knife. Be quick, come on, get it. . . . I reached down again, and this time I touched the blade to the rope. It needed no pressure. The taut rope exploded at the touch of the blade, and I flew backwards into the seat as the pulling strain vanished. . . . I was alive, and for the moment that was all I could think about. . . . There was no guilt, not even sorrow. . . . I was actually pleased that I had been strong enough to cut the rope. There had been nothing else left to me, and so I had gone ahead with it. I had done it. . . . I was alive because I had held everything together right up to the last moment. It had been executed calmly. . . . I should feel guilty. I don't. I did right.*

In another example, a diver may surface too quickly and suffer an air embolism. Reviewing the ethical considerations in wilderness medicine's ethical triangle (see Figure 94-2), both medical indications and possibly the patient (autonomy) influence the decision to rapidly transport the victim to a recompression chamber. However, even with the medical urgency of the situation, the other divers' safety mandates that the boat remain in the area until the other divers are on board. This example demonstrates again that in the wilderness setting, security factors are primary in making ethical decisions.

Issues of Direct Life-Threatening Situations for the Provider

Health care providers in a wilderness setting often have the opportunity to rescue others, which directly supports their underlying motivation to be of help. However, situations arise in which providing help puts the caregiver (or the entire team) at significant risk. This has already been discussed in Safety: Another Value Applicable to Wilderness Medicine. Wilderness medical leaders commonly decline to enter a dangerous situation to attempt to rescue a patient. However, a more direct and powerful ethical issue arises when the caregiver must directly and explicitly sacrifice the patient for personal or team safety (somewhat analogous to the difference between passive and active euthanasia). This occurs, for example, when a helicopter hoisting a patient encounters difficulties that endanger the craft. Standard procedure is to cut the hoist line, sacrificing the patient. In the abstract, the safety of the helicopter crew (and possibly rescuers on the ground) outweighs that of the patient.

Yet in reality, the conflict between safety and beneficence may not be intrinsically clear to the health care provider; an answer in favor of safety contradicts all professional education and experiences. This conflict must be resolved in advance or within a few seconds during the event if anyone is to survive. In the analogous scenario of the battlefield, the question is raised, "How many medics do you sacrifice to save one infantryman?" The same dilemma applies to rescuers.

Decision-Making Dilemmas

Health care decisions are generally the responsibility of the adult with decision-making capacity. If a patient lacks the ability to make these decisions, health care providers normally seek a surrogate decision maker, an advance directive, or the counsel of a bioethics committee or colleague. These resources are rarely available in the wilderness setting. Health care decisions can therefore become more problematic.

Unavailability of a Surrogate or a Family Member

When a person needing health care in the wilderness lacks decision-making capacity because of illness or injury, the health care provider must make decisions based on knowledge of the patient's values (often from a relatively brief period of interaction), the values of the group, or the provider's own perceptions of what is in the patient's best interest. When family or close friends are present, they may act as surrogates to make decisions for the patient, but this is much less frequent in the wilderness setting than in the urban environment. The wilderness medical provider must therefore be prepared to make difficult decisions without this guidance.

Advance Directives. To allay the problems of the absence of surrogate decision makers or knowledge about a patient's wishes, health care providers for organized expeditions, especially those in which significant risk of danger exists, may want to request that each team member complete an advance directive. However, as explained earlier, the normal forms of advance directive (durable power of attorney for health care and living will) may not suffice in the wilderness setting. Rather, a more specific directive should be used. It should detail how aggressive each individual would want the team to be in trying to extract him or her from a dangerous situation if the victim (1) had a reasonable chance of survival given available resources, (2) had a reasonable chance of survival but with serious physical disability, (3) had a reasonable chance of survival but with serious brain injury, or (4) had a poor chance of surviving. Any directive given by a team member would be tempered by the need to ensure the safety of other team members, but such a directive might give the medical provider a better idea of each team member's desires. Indeed, just discussing these scenarios with the team may be beneficial in elucidating attitudes and health care desires in the wilderness. Some team members might want to use a durable power of attorney, naming a surrogate within the team, for use with or in place of the more specific directive.

Euthanasia

Controversy continues to rage in society and medicine over the concept of active euthanasia (so-called mercy killing). In wilderness medicine, however, euthanasia may be less ethically problematic, although it is a very sensitive issue to discuss and a devastating event for those involved. Active euthanasia may be an ethically acceptable alternative in the rare situation when a patient will die either because he or she cannot be rescued from the wilderness environment or because the survival of group members would be jeopardized by attempting to evacuate or remain with him or her until help arrives. The seriously injured person on a high-altitude climb with inclement weather quickly approaching and the injured caver in a flooding cave are two examples. In these cases, euthanasia is based on the beneficence of relieving suffering in a doomed individual (although many in the medical profession believe that mercy killing violates professional principles), security for other members of the party (not creating more victims), and perhaps patient autonomy (although some psychiatrists might argue that by asking for death a person demonstrates a lack of decision-making capacity).[2] Indeed, a particularly difficult situation is the psychiatric patient who cannot be made to continue out of the wilderness and who will endanger the group and himself or herself if either remains. This has happened at least once in the dual peril of war and wilderness, when British soldiers of the 111th Indian Infantry Brigade in Burma euthanized their own during World War II.[29] In non-war situations, however, any episodes of active wilderness euthanasia have gone unreported.

Further complicating the preceding scenarios is the question of whether such patients should be simply left to die (passive euthanasia) or more humanely killed (active euthanasia). This question should be given serious consideration, because many incidents of passive euthanasia in wilderness settings occur, especially in high-risk or remote areas. Passive euthanasia has occurred, for example, at least several times on Everest expeditions when unconscious, hypothermic climbers were left to die when conditions made it difficult or impossible to get them down.[25] The ethical question of what is best for the injured individual almost always comes in direct conflict with other team members' lack of confidence in their (or their medical person's) prognosis and their unwillingness to implement active euthanasia. The lack of certainty about prognosis may sometimes be justified. For example, during a recent disastrous Everest expedition, a physician-climber who was left for dead (active euthanasia was not discussed among team members) survived by eventually making it to camp on his own.

Dilemmas in Wilderness Policies

Ethical decision-making plays a part in policies governing wilderness medicine. The values of beneficence and nonmaleficence make proposed and actual rules for wilderness medical practice untenable. These policies include when to stop searches, prohibition of motorized vehicles in wilderness areas, no-rescue areas, prohibition of environmental destruction, and restriction of medical providers' roles (see How Much Authority Is Given to Nontrained Personnel?).

When to Stop Searches

Without a body or corpse, it is difficult for managers of wilderness searches to know when to stop searching for someone who is presumed lost. Resource allocation decisions (distributive justice) create the contours of the solution to this kind of ethical dilemma. The parameters include available resources, probability of finding the lost person, danger to searchers, and the likelihood of survivability under existing conditions. An example of such a dilemma occurred near Mount Rainier when a hunter briefly lost consciousness and became separated from his group.

Fortunately, he was a strong, heavy man who could draw on fat stores for energy and warmth for several days. His hunting companions immediately began a search, followed by a formal search and rescue by a trained team the next morning. The dilemma was when to stop or pause the search because of bad weather, risk to the searchers, and the probability that the hunter was dead due to a preexisting heart condition. Severe weather did cause the search to be halted after 4 days because of danger to the searchers, but it was to resume the next day after the weather had cleared. Before the search could be resumed, however, the victim found his way to a road, where he encountered a ranger. As this case illustrates, searches will often last beyond the point when the victim is believed to be dead in hopes of finding them alive or at least finding the corpse. It is the search leader's responsibility to continue the search process as long as it is reasonable to do so.[23]

Motorized Vehicle Restrictions and Environmental Protection in Wilderness Areas

A policy occasionally imposed on wilderness medical practice is that of no motorized vehicles in designated wilderness areas. This rule has logical roots but is enforced only intermittently. When it is used to hinder rescue efforts or delay needed medical care, however, it defeats a basic purpose of society: the assurance of citizens' welfare.

A related issue is the basic tenet of wilderness travel that the environment should be left at least as pristine as it was found. Situations arise, however, when preservation of a wilderness area must be weighed against pain and suffering or life and death. Helicopter pads chopped into the forest or a new entryway blasted into a cave are only two examples. The preservation of wilderness areas is an important goal, but so is the preservation of human life and values, and these should not be overridden to reach a symbolic goal. Human life is a priority.

No-Rescue Areas

Perhaps the most pernicious concept proposed to govern wilderness medical care is that of the "no-rescue area," into which adventurers would go with the foreknowledge that no rescue would be available.[20] Akin to playing Russian roulette, people entering these wilderness areas would put life and limb at risk while society condoned and presumably enforced a requirement not to assist those in need. This macho concept disregards patient and societal beneficence and individual autonomy. Those familiar with advance directives, such as health care powers of attorney or living wills, know that patients in extremis often countermand their preconceived instructions. To prevent wilderness trekkers from doing the same to preserve their life contradicts basic ethical values.

The references for this chapter can be found on the accompanying DVD-ROM.

95 The Changing Environment

James K. Mitchell and Monalisa Chatterjee

Three miles southwest of the Kremlin, not far from Moscow's Olympic Stadium, lies the Novodevichiy Cemetery, one of the most celebrated burying places in Russia. Amid ornate memorials to former leaders such as Andrei Gromyko and Nikita Khrushchev, who were out of favor at the time of their deaths, and to giants of the arts, such as Chekhov and Shostakovich, stands a white marble pedestal carrying the sculpted head of Vladimir Illich Vernadsky (1863–1945). Little known in the West, Vernadsky was a prescient observer of the emerging role of humans as makers of the global environment. It was he who first announced that we are living at a time when the power of mankind to change the earth now rivals that of geologic processes.[8] In the past, students of natural history could regard the human life span as a mere blink of a cosmic eye that witnessed little environmental change. Today, we are faced with the prospect that the planet may be fundamentally transformed by humans, perhaps within a few decades, but more probably over the space of one or two generations.

This situation has not come about all at once, or equally everywhere. On a global scale, it has gradually built up over centuries, although the local manifestations of increased human agency have sometimes been masked by other processes. For example, conversion of "natural" ecosystems to "managed" ecosystems is a dominant feature on the global scale, but in some parts of the world (especially the United States and Western Europe), managed ecosystems are also being abandoned. Such is the case in New Jersey, where hilltop 19th century farm lands have largely reverted to regrowth forests.[88]

The complications of environmental change might best be appreciated with the aid of a time machine such as the one envisioned by H. G. Wells in 1895. Imagine, for a moment, being in a mature pine forest in southern New England. What might be observed as the machine slips into the past at this location?

The surrounding landscape comes clearly into view. The pine trees shrink slowly down into youth as the years wind back, for most of today's pines trace their origin to the abandonment of farm lands at or near the turn of the century. The pines disappear entirely in the late 19th century and are replaced by shrubs and eventually by grasses. By the mid-1800s, the local vicinity is completely open and appears as a shifting mosaic of agricultural crops and pasture, rotated in time and space. This is the high tide of farming in New England. Thereafter, the sequence goes into reverse. By the 18th century, trees begin to return, connecting the remnant patches of presettlement vegetation. Gradually, the forest closes in and traces of human presence fade. Little breaks the monotony, apart from occasional fires started by lightning or Native Americans clearing seasonal cultivation patches, or major windstorms that topple weaker trees. As the 16th century approaches, the landscape is essentially similar from decade to decade.

The time traveler's dominant impression is one of change. The preceding sequence of changes has been documented by many analysts of the New England landscape.[19,55,68] The sequence might be different elsewhere, but is no less dynamic.[92] Sometimes the changes are sudden and dramatic, and sometimes they are slow and imperceptible.* Sometimes they are "natural" (e.g., storms) and sometimes they are caused by humans (e.g., forest clearance). For the bulk of human history, natural changes have seemed to dominate, although in fact people have been major shapers of the environment for millennia.[17,53,86,92] A casual observer of the New England landscape might conclude that the well-wooded contemporary scene is more "natural" than the cleared fields of the 1850s. However, today's scene is just as much a product of human choices as that of the 19th century, albeit different in composition and appearance.

In any event, deciding whether human or natural factors are responsible for a given environmental change is often difficult; both factors tend to operate interdependently (Fig. 95-1). It is widely believed that people have reached a critical threshold as environmental modifiers; they are able to equal or surpass the

*Arguments between uniformitarians (who give primacy to continuous, small-scale changes) and catastrophists (who favor change as a function of intermittent large-scale events) have continued for more than a century (Oldroyd DR: Thinking about the Earth: A History of Ideas in Geology. Cambridge, Cambridge University Press, 1996, pp 131–144). Although the uniformitarian position has dominated among earth scientists, recent evidence in support of catastrophic explanations is compelling. For example, the fossil record indicates that mass extinctions have occurred throughout the globe at many times in the past. (See Martin PS, Klein RG [eds]: Quaternary Extinctions: A Prehistoric Revolution. Tucson, AZ, University of Arizona Press, 1984.) Some analysts argue that the age of dinosaurs ended 65 million years ago when a massive meteor collided with the earth, perhaps striking the Gulf of Mexico near the Yucatan Peninsula. (See Alvarez W, Asaro F: What caused mass extinction? Sci Am 263[4]:78, 1990.) The impact may have thrown sufficient dust into the atmosphere that solar radiation was reflected back into space, causing global temperatures to plummet. A similar, although smaller effect was noted in 1992 when volcanic Mt. Pinatubo in the Philippines spewed ash, dust, and gases into the atmosphere and lowered mean global temperature by an estimated 2° C. (See Dutton EC, Christy JR: Solar radiative forcing at selected locations and evidence of global lower troposphere cooling following eruptions of El Chichon and Pinatubo. Geophys Res Lett 19:2313, 1992.)

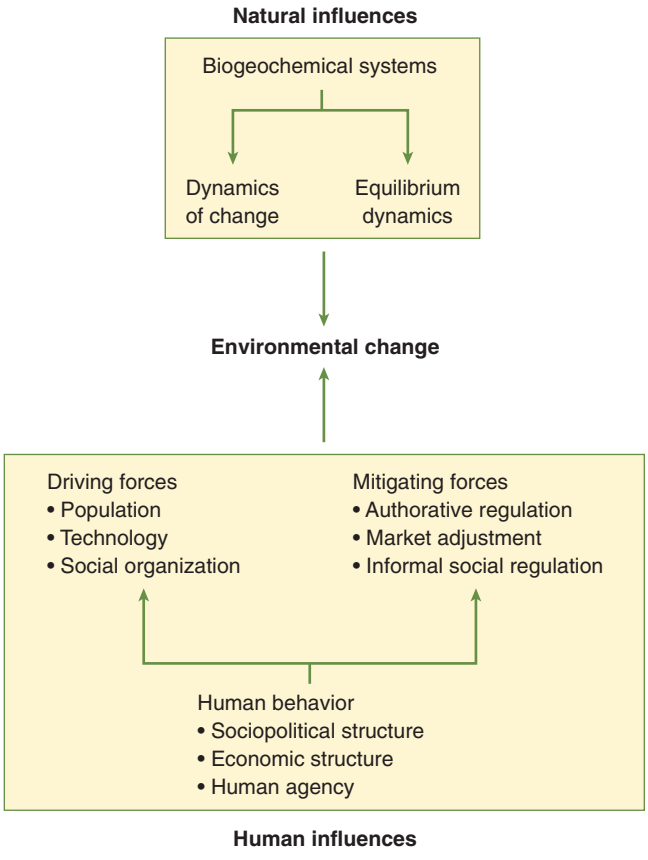

Figure 95-1. Human and natural forces for environmental change. (Modified from Kates RW, Turner BL II, Clark WC: The great transformation. In Turner BL II [ed]: The Earth as Transformed by Human Action. Cambridge, Cambridge University Press, 1990, with permission.)

effects of nature. Portentous changes are becoming manifest in the entire biosphere. We can at last begin to speak of a human "transformation" of the global environment.[87]

This chapter addresses environmental change and its human dimensions, with special attention to implications for environmental and wilderness medicine. What types of changes are likely to occur? How will they affect the natural environment, especially wilderness areas? What will be the consequences for society in general and for medical practitioners in particular? Can anything be done to improve our chances of successfully negotiating this impending time of dislocation and discontinuity?[58]

ISSUES OF ENVIRONMENTAL CHANGE

In recent years, a number of environmental change issues have come to prominence. They include climate change, stratospheric ozone depletion, erosion of biodiversity, population growth, and burgeoning pollution. These issues affect all environments, from urban centers to remote wilderness areas. In the discussion that follows, they are examined on a variety of scales. Although each scale is characterized by different expressions of change, all are interconnected. Local changes can aggregate to produce global effects, and global changes have many different disaggregated local effects.[87]

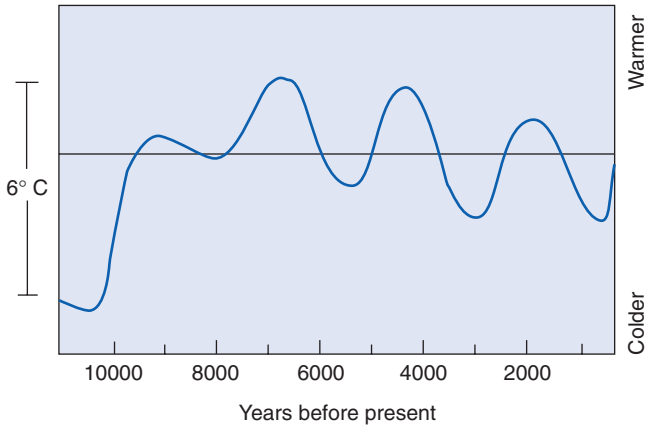

Figure 95-2. Variations in mean global temperature during the past 10,000 years. Horizontal line represents present global average temperature. (Modified from Henderson-Sellers A, Robinson PJ: Contemporary Climatology. New York, Wiley, 1991, with permission.)

Climate Change

Weather is the state of the atmosphere at any specific time. Climate is the average weather pattern at a particular location. Weather and climate are usually described by such measures as temperature, precipitation, pressure, humidity, and wind speed and direction. In most parts of the world these measures have been recorded for less than a century, so the actual historical record of direct observations is relatively brief compared with the human tenure of the earth. However, scientists are often able to extend the historical record by constructing synthetic climate data from other evidence such as tree rings, fossils, concentrations of plankton in ocean sediments, pollen in sedimentary rocks, and isotopes of carbon and oxygen in rocks and glacial ice. For example, narrow intervals between annual growth rings in trees and thin layers of organic material in lake sediments usually indicate cold, dry conditions. Clues like these permit investigators to open a window on past climates.

Figure 95-2 illustrates trends in average global temperature during the past 10,000 years. Note that the global temperature has been in flux throughout this period. Not only has weather varied concerning long-term average conditions, the averages themselves have changed over time. For example, during the most recent ice age (about 10,000 years ago), average global temperatures were approximately 6°C (about 8.5°F) cooler than at present.[32] In other words, a massive environmental change (the Wisconsin ice age) was connected with a relatively small climatic change. It is worthwhile remembering that regional changes in climate may or may not parallel global changes. For example, between 2600 and 2700 years ago—roughly at the time of Socrates and Confucius—North America was colder and wetter than the continental average since the end of the Wisconsin glaciation, whereas conditions in Europe were warmer and drier. Fortunately, the climate has remained within a range that sustains life for most of the earth's history, and the changes have occurred at very slow rates, over thousands to millions of years.

Currently, a broad consensus exists among atmospheric scientists that global temperatures may rise significantly in coming

decades.[54] One indicator of this trend is the fact that the 10 warmest years since 1861 all occurred after 1990.[94] Although the global climate system is enormously complex, two factors point toward warming. First, it is known that certain greenhouse gases warm the atmosphere by trapping short-wave radiation reflected from the earth's surface when it is heated by solar radiation. Second, atmospheric concentrations of these gases, which include carbon dioxide, methane, and nitrous oxide, are steadily increasing. Normally, the materials in greenhouse gases pass through long biogeochemical cycles between natural sources and natural sinks. For example, sulfur enters the atmosphere as sulfur dioxide from volcanic eruptions and washes back to the oceans in the form of mildly acid rainfall whose constituents are later incorporated into bottom sediments. Human activities can increase source loads (e.g., emissions) and reduce the absorptive capacities of natural sinks. In the case of carbon dioxide, the greenhouse gas about which there is most concern, both processes are at work simultaneously. Emissions of carbon dioxide have been increasing as energy-hungry societies burn petroleum hydrocarbons, coal, and wood. At the same time, forests that usually absorb huge amounts of atmospheric carbon dioxide continue to be cleared—although at rates that are less than was predicted a decade ago.

Atmospheric scientists are currently struggling to estimate how climate might change as greenhouse gases accumulate. For this purpose, they rely heavily on general circulation models (GCMs) that mathematically simulate the global climate system. The chemistry and physics of climate are complex and the models, although increasingly sophisticated, are still imperfect. Their accuracy is constrained both by the limits of current knowledge about the dynamics of the atmosphere and by the computational power of the most advanced supercomputers. They are also hedged with other limitations. For example, the GCMs presently generated are too coarse to provide more than a broad-gauge portrayal of atmospheric conditions in a lattice of regions over the earth's surface. They do not reveal storm systems that bring most of the weather to middle and high latitudes. They also do not incorporate the role of clouds as reflectors and absorbers of energy. They do not satisfactorily account for all of the carbon dioxide that is believed to have been liberated into the atmosphere through human activities. Nonetheless, many investigators have considerable confidence in the accuracy of GCMs because of their relative success in replicating present and past climates.

The Intergovernmental Panel on Climate Change (IPCC), a joint United Nations–World Meteorological Organization committee of leading earth scientists, has synthesized existing research on climate change. It has now completed three comprehensive assessments of the state of scientific knowledge and reached some sobering conclusions. (A fourth is due to be published in 2007.) In the most recent assessment (2001),[38] it concluded that "globally averaged surface temperature is projected to increase by 1.4° to 5.8°C over the period 1990 to 2100." The projected rate of warming is likely to be without precedent during the past 10,000 years on Earth.

Although the IPCC estimates embody a consensus about global warming, the level of agreement declines as researchers attempt to forecast the resulting impacts, especially at regional and local levels. An enhanced greenhouse effect would have a greater effect on the global climate than would temperature alone. Solar radiation provides the energy that drives the climate system. The effects of a warmer atmosphere could produce a cascade of changes in many climate variables. For example, precipitation and evaporation would also be likely to increase, especially over high latitudes, but with strong regional variations elsewhere.

TABLE 95-1. Effects of Global Climate Change in Different Latitudes

	LATITUDINAL ZONES IMPACTED		
INDICATOR	LOW	MIDDLE	HIGH
Biologic productivity	–	?	+
Precipitation	+	–	+
Food production	–	+	+
Malaria transmission	0	+	0

+, increase; –, decrease; 0, no change; ?, uncertain.
Adapted from data reported in Hulme et al[35] (1999), White et al[91] (1999), Arnell[6] (1999), Parry et al[63] (1999), and Martens et al[54] (1999).

However, we will soon know a great deal more about regional patterns of climate change, because a large number of studies that combine GCM data and other indicators of climate are now being conducted.[5,52]

In general, GCMs indicate that lower latitudes and lower elevations will be less affected by anticipated climate changes than will upper latitudes and higher elevations. However, the most recent generation of climate change impact simulations paints no simple pictures.[1,63] Different combinations of effects would be likely at different latitudes (Table 95-1). Probably, a mosaic of regional and local changes will occur along a spectrum from strongly positive to strongly negative depending on how, when, and where they occur. For example, tropical islands might experience heavier precipitation combined with more frequent severe storms and rising sea levels. The net impacts of such changes are difficult to assess, but the experience of Indian Ocean islands and coastlines during the massive tsunami of December 26, 2004 shows just how vulnerable to natural disasters these regions already are. For the Andamans, the Maldives, the Seychelles, and other heavily populated, low-lying islands of the Indo-Pacific Ocean, the results of sea level rise could be disastrous, whereas other places, such as high-standing islands of the Caribbean, could see offsetting agricultural benefits.[64]

More than any other factor, the rate of climate change is of concern to humans. GCMs indicate that absolute changes in temperature will be smaller than those that have occurred at other times during the earth's history. However, the anticipated climate changes would still occur at a rate and magnitude that are unprecedented in human experience. Whereas past changes usually occurred slowly enough for plants and animals to adapt or migrate, examples of mass extinctions after rapid change exist. Many scientists fear that today and in the future, insufficient time and undeveloped areas will be available for plants and animals to make similar adjustments.*

*Recent rates of temperature change seem to be speeding up. As noted in December 2003 by the World Meteorological Organization, "Over the 20th century, the global surface temperature increased by more than 0.6°C. The rate of change for the period since 1976 is roughly three times that for the past 100 years as a whole. In the northern hemisphere, the 1990s were the warmest decade with an average of 0.38°C. The surface temperatures averaged over the recent five years (2000–2004) were, however, much higher (0.58°C)." (United Nations Environment Programme: The Environment in the News [press release], December 19, 2003.)

Although changes of average climate would have important long-term consequences, variations in extreme weather might produce the most immediate and significant impacts.[57] Droughts, floods, and tropical cyclones are unusual events in today's climate. If mean climates change, changes in the frequency and severity of these extremes would probably also occur and become manifest well before the permanent shifts could be confirmed.[31] The geographic distribution of such events would also be affected. According to the IPCC, more hot days and heat waves would likely occur over most land areas, with frost days and cold waves becoming fewer. The frequency of extreme precipitation events would also likely increase everywhere.[38] As a result, natural hazards would be likely to pose increased risks to society. Moreover, exposure and vulnerability to extreme events would probably be exacerbated because populations at risk might respond to the new conditions on the basis of outdated information and assumptions.[22] We might find that our previous experience prepared us to "fight the last war" rather than the new one.

Stratospheric Ozone Depletion

The stratosphere is a distinct layer of the upper atmosphere that occurs between 15 and 58 km (9 and 35 miles) above the ground. It contains significant concentrations of ozone (O_3), a gas that is formed when solar radiation splits ordinary oxygen atoms.* The stratospheric ozone layer absorbs most of the ultraviolet (UV) radiation from space that would otherwise damage plant and animal species.

During the 1970s and 1980s, researchers discovered that stratospheric ozone was being lost and that the ozone layer was thinning to the point of disappearance, particularly in polar regions.[29,60] Chlorofluorocarbons (CFCs) were held to be at fault. For decades, these synthetic compounds had been manufactured in large quantities, mainly for use as aerosol propellants and refrigerants. Once CFCs escape into the atmosphere, they remain stable until reaching the stratosphere, where they decompose under the action of UV radiation. Chlorine atoms are released and bond with ozone atoms, breaking them down into oxygen and other products. As a result, the ozone shield is weakened or removed.

If the ozone layer is sufficiently depleted, intensity of UV radiation that reaches the earth's surface could be significantly increased. This could have deleterious consequences for human populations and for plant and animal species. For humans, increased incidence of skin cancer, cataracts, and immune system suppression are three recognized effects of high UV exposure. Although humans might take precautions to protect themselves against UV radiation, such as reducing time spent outdoors, or adding sun blocks, glasses, and clothes, nonhuman species may not be able to make the necessary adaptations. Serious disruption of human and agricultural systems is possible.

During their winter seasons, the Antarctic[65] and, to a lesser extent, the Arctic[51,72,85] have experienced elevated levels of UV radiation. "Ozone holes" have been clearly traced to CFCs, and an international agreement, the Montreal Protocol, was reached to phase out CFC usage by 1996. Much progress has been made toward that goal, but these compounds are still being produced in some developing countries and the substitute chemicals that were introduced elsewhere may also contribute to global warming.[47] In any case, chlorine atoms are extremely long-lived in the stratosphere, perhaps persisting for 100 to 200 years. There will continue to be potential for additional ozone depletion in the decades to come.

*Ozone also accumulates near ground level as a byproduct of the photochemical modification of exhaust gases from automobiles and other sources of pollution. Concentrations of this type of ozone are sometimes reported in local news media, but the ground-level "ozone problem" should not be confused with the stratospheric one.

Erosion of Biodiversity

Loss of species or the habitats that support them is a controversial and potentially serious global problem that comes under the heading "erosion of biodiversity." Biodiversity is not an agent of change like greenhouse gas buildup or ozone depletion. It is an index against which environmental changes can be assessed.[81] Like climate change and ozone depletion, biodiversity has a range of dimensions from global to local.[37] Two aspects of biodiversity that are of great importance are numbers and interconnections of species.

Estimates of the number of existing species are wide-ranging because the state of knowledge about the planet's biologic resources is both uneven and incomplete.[83] It is estimated that the earth hosts between 5 and 15 million species. Approximately 1.75 million of these have been named.[49] Higher-order mammals and birds in temperate ecosystems are well documented, but insects, worms, and micro–life forms in tropical regions are far less known. In the United States approximately 100,000 species are recognized,[24] but only about one fifth of these have been surveyed to date.[83]

Paleobiologic research indicates that the number and type of species have varied greatly over time. New species evolve through adaptive genetic mutations, whereas others perish because of competitive pressures of natural selection. Emergence and disappearance rates depend on the speed and direction of environmental change and the ability of species to adjust. What is most troubling about the recent record is the disappearance of so many species. "Between 1600 and 1994, at least 484 species of animals and 654 species of plants (mostly vertebrates and flowering plants) became extinct. The rate of extinction in groups such as birds and mammals also increased dramatically during this period: Nearly three times as many species of birds and mammals became extinct between 1810 and 1994 (112 species) as were lost between 1600 and 1810 (38 species)."[70] Plant losses are presumed to have been much greater. On some oceanic islands, such as Hawaii, the disappearance of native animal species is almost total. Of 269 extinct Hawaiian species, most were either invertebrates (135) or plants (105). A majority of the rest are birds (15) and land snails (11).[83] Commercial forestry and fishing have proved particularly injurious to biodiversity because they harvest desirable species and destroy undesirable species at the same time.[36] Agriculture and animal husbandry also contribute to species extinction, especially by modifying the habitats that support biota. Particular concerns have been expressed about threats to tropical forests and the near extinction of some marine species like the northern cod, blue whale, and leatherback turtle. However, the problem is general in scope and may be most important for the "noncelebrity" species that do not elicit much human compassion.

In the United States it is estimated that approximately 32% of all species are now under serious pressure, some to the point of threatened extinction[83] (Box 95-1). However, the picture varies widely among particular groups of species (Table 95-2). Denizens of freshwater ecosystems, such as shellfish, crus-

taceans, amphibians, and fish, are far more likely to be in danger than are flowering plants, conifers, mammals, and birds. Likewise, on the U.S. mainland, loss of biodiversity is more acute in sunbelt states and east of the Mississippi River than in states of the northern Great Plains and the northern Rocky Mountains.

For many people, the protection of threatened species is a moral imperative. For others it is a luxury. Quite apart from moral issues, the rising rate of species extinction has practical implications.[71] For example, loss of the planet's genetic stock hampers the search for wild strains of domestic crops that are resistant to pests and diseases that plague high-yield domestic varieties. The so-called Green Revolution, which has helped to alleviate world hunger in recent decades, owes much of its success to introduction of resistant wild genetic strains into commercial agriculture.

Biodiversity is also important for stability of global ecosystems. For example, the extent to which entire species can be eliminated from an ecosystem before it collapses is unknown. Likewise, the extent to which some nominally "wild" species may thrive under human management while others succumb is hotly debated.[26] Most ecologists believe that ecosystems containing a wide diversity of organisms are more resilient to change than those with few species. Regardless of the degree of resilience, biodiversity and environmental change may be connected by negative feedback relationships. Thus, environmental change may lead to a loss of biodiversity that in turn produces lowered resistance to pressures for further change.

Despite intuitive, theoretical and case study arguments in favor of preserving biodiversity, it has been difficult to agree on standardized measures of biodiversity or its loss. Deforestation of South American rainforests is a case in point. The Amazon Basin is one of the world's premier wilderness regions and is also regarded as its most important source of biodiversity. Perhaps impelled by dramatic and widely publicized reports of forest clearances by ranchers, homesteading farmers, and mineral firms in Brazil during the late 1980s, levels of international concern about the loss of biodiversity in Amazonia have been high. But, as in most developing countries, comprehensive and reliable data on Brazilian deforestation are difficult to secure and interpret[80,97]:

> *Up to 1988, the best estimates suggest that 6–7% of the forest has been subject to at least one cutting in the past 30 years. Inclusion of secondary forest, known to have been deforested prior to 1960, but with advanced secondary forest that satellite imagery cannot differentiate from primary forest, raises the estimate by roughly 100,000 km^2. The latest estimates from INPE, the Brazilian Space Agency, show that deforestation had grown to about 472,000 km^2 in 1994, including the deforestation that occurred prior to 1960. It should be emphasized that all of this remaining 372,000 km^2 of deforestation (9% of the total forest) should not be interpreted as land that has been deforested and transferred to agricultural use forever. Land that has been cleared and later abandoned will often return to forest cover, even if used for pasture, although there is some uncertainty about the length of the process of forest recovery from pasture abandonment to mature forest.*[25]

Given the foregoing uncertainties, it is difficult to predict what might be the future rates of biodiversity erosion. The best available estimates suggest that about 3.5% of today's bird species will likely become extinct by the year 2050, together with most large marine predators and much of the species richness of freshwater ecosystems.[40]

Box 95-1. Percentages of U.S. Species at Risk

Extinct	1.0%
Critically imperiled	6.5%
Imperiled	8.8%
Vulnerable	15.4%
Secure	69.3%

From Stein BA, Flack SR: Environment 39(4):6–11, 34–39, 1997.

Population Growth

Human population is one of the primary driving forces behind contemporary environmental change. Beginning with the Reverend Thomas Malthus (1766–1834), many people have argued that rising populations must eventually deplete resources and degrade environments because the Earth is, for all intents and

TABLE 95-2. Percentages of U.S. Species Groups at Risk

	EXTINCT	IMPERILED	VULNERABLE	SECURE
Freshwater mussels	16.4	39.7	11.8	32.1
Crayfish	17.3	32.8	0.9	49.0
Amphibians	14.0	23.9	2.5	59.6
Freshwater fish	14.1	22.0	2.6	61.3
Flowering plants	16.6	15.8	0.9	66.7
Conifers	12.2	14.0	0.0	73.8
Ferns	11.9	8.9	0.7	78.5
Tiger beetles	13.6	6.3	0.0	80.1
Dragonflies	10.4	7.6	0.4	81.6
Reptiles	11.9	6.1	0.0	82.0
Butterflies	12.3	4.0	0.5	83.2
Mammals	9.1	7.2	0.2	83.5
Birds	5.4	5.8	3.3	85.5

Adapted from Stein BA, Flack SR: Environment 39(4):6–11, 34–39, 1997.

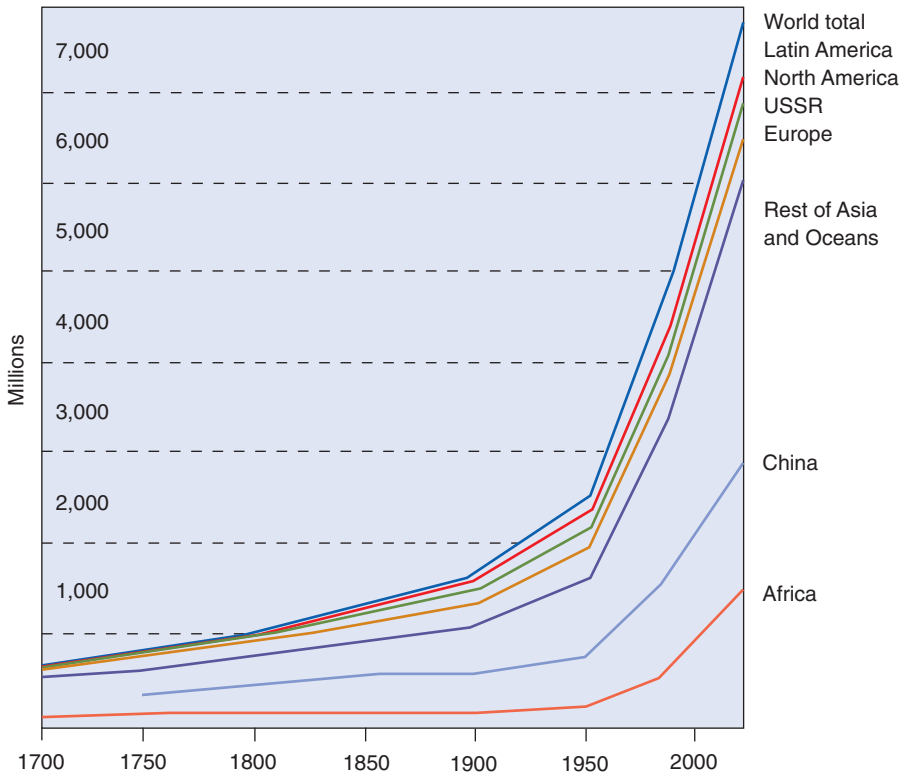

Figure 95-3. Global population (1700–2020). (Modified from Demeny P: Population. In Turner BL II [ed]: The Earth as Transformed by Human Action. Cambridge, Cambridge University Press, 1990, with permission.)

TABLE 95-3. Average Annual Percentage Rates of Population Increase

	1700–1750	1750–1800	1800–1850	1850–1900	1900–1950	1950–1985	1985–2020
Africa	0.0	0.0	0.1	0.4	1.0	2.6	2.7
Asia	0.3	0.5	0.5	0.3	0.8	2.1	1.4
Europe	0.3	0.6	0.7	0.7	0.6	0.6	0.1
Russia	0.3	0.7	1.0	1.0	0.7	1.2	0.6
North America	0.8	1.0	3.2	2.6	1.2	1.3	0.6
Latin America	0.8	0.5	1.2	1.6	1.6	2.6	1.6
Oceania	—	—	—	—	1.6	1.9	1.2
World total	0.25	0.44	0.55	0.54	0.84	1.88	1.45

From Demeny P: Population. In Turner BL II, Clark WC, Kates RW, et al (eds): The Earth as Transformed by Human Action: Global and Regional Changes in the Biosphere over the Past 300 Years. Cambridge, Cambridge University Press, 1990, pp 41–54.

purposes, a closed system.[45] In the absence of interplanetary space travel on a scale impossible at present, to destinations that are now unknown and perhaps nonexistent, the Earth is our only home. This does not mean that it will be impossible for the Earth to hold additional human populations. If the record of the last four centuries provides a demographic lesson, it is that the global carrying capacity is highly elastic—up to some, as yet unreached, limit.[16] Leaving aside the argument that human ingenuity can make possible the support for larger populations indefinitely, clearly from the perspective of burdens on the physical environment, how people live is more important than the number of people. All other things being equal, richer societies place heavier burdens on the physical environment than do poorer ones. For example, the United States, with just 5% of the global population, consumes approximately 25% of the world's energy resources.[18]

The global population has undergone unprecedented growth in the last several centuries (Fig. 95-3). By 1800, the Earth's population was approximately 1 billion. By 1920 it had doubled. Three billion was reached in 1960, and the present estimate is over 5 billion. The United Nations estimates that there will be somewhere between 6.3 and 8.9 billion people on Earth by 2050. Most of the new growth is likely to occur in developing countries of Asia, Africa, and Latin America (Table 95-3). The recent experience of China suggests that the process of development can itself perpetuate or increase historical rates

of population growth, at least until economic conditions improve significantly.

The composition of future populations is an increasing concern for governments and individuals. In places like Japan and Eastern Europe, natural increase is now well below the rate necessary to replace existing populations, whereas in the United States, increasing numbers are maintained largely by immigration. As a result, the fraction of national populations that is more than 65 years of age is growing rapidly in the more developed world. Meanwhile, increasing expertise in genetic manipulation holds out the prospect of significantly longer life spans for populations that can afford the kind of scientific research and medical care that will make this possible. In other words, some parts of the world's population will become increasingly healthier and older, whereas others may remain caught in a cycle of sickness-prone lives followed by early deaths. Quite apart from the staggering societal impacts that such a change would produce, the implications for wilderness are considerable. For those who can be assured of longer lives, the quality of life experience—including the quality of their environments—may become of foremost importance. Wilderness areas would be among the most cherished places, and decisions about their future all the more portentous. Among developing countries, a different future might obtain, perhaps dominated by intense pressure to convert all available resources into supports for survival. Although such scenarios are not difficult to envision, they carry with them a danger of indulging in stereotypical dichotomizations that ignore possibilities for a range of more nuanced outcomes.

Today, the greatest uncertainties about future populations pertain to rates of migration and composition of families.[15] If current patterns of migration continue, the lion's share of migrants will end up in large countries like the United States, India, Pakistan, France, and Germany, where, depending on their internal destinations, they may add to the burden of users on existing wilderness areas.

Pollution

Unwanted byproducts of production and consumption that exceed the absorptive capacity of the environment are known as *pollution*. Pollution comes in many forms, including solid physical materials, liquid chemical compounds, and energy (e.g., thermal pollution). Some pollutants (such as certain isotopes of plutonium) are highly toxic even in small amounts. Many materials that are beneficial in small amounts can be deleterious in large quantities. For example, phosphorus is a nutrient that limits biologic productivity in coastal and marine ecosystems. Small amounts of phosphorus can increase algal growth at the bottom of marine food chains. But when larger amounts of phosphorus-rich runoff from fertilizers or septic systems enter these environments, the entire population of algae can begin a period of explosive growth ("bloom"). Extensive blooms can produce so-called red tides or brown tides.[14] These occur when algae prevent light from penetrating coastal waters and the decomposition of dead algae consumes dissolved oxygen. Large fish kills are a frequent result.

The preferences of people for quick and convenient disposal of pollutants into available environmental sinks (e.g., soil, streams, groundwater, oceans, and atmosphere) have sometimes been validated by incomplete science. For decades in the United States and elsewhere, scientists advised policymakers that "the solution to pollution is dilution." As a result, physical and chemical wastes have been released into environments that had finite capacities for absorbing them. Once the absorptive capacities were reached, a variety of serious problems occurred. These included biologically "dead" rivers (such as Cleveland's Cuyahoga River), "dead" lakes (such as Lake Erie), and "dead" seas (such as the sewage sludge dumping ground in the New York Bight off the coasts of New Jersey and Long Island). Although some of these conditions can be reversed, the processes are slow, costly, contentious, and often incomplete. Also, a growing body of evidence suggests that the aggregate effect of pollution may be jeopardizing the functioning of fundamental Earth systems. The buildup of atmospheric carbon dioxide is an excellent example of this process.

Despite a large volume of evidence, the effects of pollutants on receiving environments are not fully known.[18] This is partly because of lack of scientific knowledge about the normal (unpolluted) functioning of some environments, such as deep oceans and tropical forests. The sheer volume and variety of materials released into the environment and their interactions complicate the study of effects of any single pollutant. Sometimes the effects of pollutants are subtle, long-delayed, and far removed from the point of origin, making it difficult to connect causes and consequences. Sometimes experts disagree about evidence of pollution impacts collected in the field and evidence acquired from laboratory experiments. Even the impacts of well-studied events like the *Exxon Valdez* tanker grounding are in dispute.[9,84] Nonetheless, there is a broad consensus that the absorptive capacity of receiving media is not inexhaustible and that pollution is a growing world problem that is pushing society against the limits of environmental resilience.

IMPACTS OF ENVIRONMENTAL CHANGE ON WILDERNESS AREAS

The task of assessing environmental change impacts in wilderness areas poses particularly difficult challenges to researchers. To begin with, the term *wilderness* is rarely used by scientists. For example, readers of the voluminous reports of the Intergovernmental Panel on Climate Change[34] might search in vain for references to wilderness impacts. Instead, there are comments about the effects of climate fluctuations on specific types of ecosystems, land covers, or land uses—such as forests or nature reserves—some of which may be defined as wilderness by different interest groups.[56] The lack of references to wilderness in the scientific literature can be explained by the emergence of a widely shared conviction among scientists that no part of the world is now truly "natural." As one prominent ecologist puts it, "Overall, any clear dichotomy between pristine ecosystems and human-altered areas that may have existed in the past has vanished."[89] In other words, our environments are arranged on a continuum from intensely human-constructed places, such as cities, to places where the human presence is small, intermittent, or nonexistent, such as wilderness areas. Most places show evidence of both human and natural influences.

If scientists shy away from the term "wilderness," the same cannot be said of political leaders and the general public. Unfortunately, in this larger arena there is little agreement about meanings of wilderness. "Naturalness" and "remoteness" are two commonly mentioned wilderness attributes, but measures

TABLE 95-4. Global Crop Land Changes (1700–1990)*

REGION	1700	1750	1800	1850	1900	1950	1990
United States and Canada	3,077	6,606	15,009	170,444	199,413	235,327	232,771
Central and South America	15,348	15,333	16,602	18,980	31,950	79,024	149,456
Europe	67,292	73,315	79,878	87,028	106,734	143,983	139,129
Asia and Soviet Union	135,514	179,054	239,826	324,859	424,820	603,186	698,279
Oceania	2,147	3,500	5,788	9,659	16,291	27,750	53,063
Japan	1,425	1,458	1,492	1,527	2,119	4,720	4,596
World	265,631	321,302	401,611	537,060	813,425	1,229,985	1,477,600

*In 1000 hectares (ha).
From History Base of the Global Environment—HYDE. Available at http://www.mnp.nl/hyde/

of both can be highly subjective. Depending on the definition that is adopted, a wilderness area might include some of the world's most biologically productive ecosystems (e.g., tropical forests of the Brazilian Amazon Basin) together with some of its least productive (e.g., Sahara Desert), and some of those that have been thoroughly transformed by human activities (e.g., so-called urban wilderness areas like Portland, Oregon's Forest Park). Some analysts have attempted to cut through this Gordian knot by equating wilderness with uninhabited areas or roadless places, but neither of these indicators is comprehensive in scope when it comes to accounting for all the places that humans perceive as "wild." In view of the potential for confusion that exists in discussions of wilderness, it is worthwhile sketching some of the philosophical and evolutionary background of the term.

The notion of wilderness can be found in several of the world's earliest cultures,[69] but formulation of a powerful philosophical and political movement that espouses the value of unaltered natural areas did not occur until the 19th and early 20th centuries in the United States.[74] Although the wilderness movement subsequently diffused elsewhere, widespread public concern for the preservation of wild lands remains a characteristically American preoccupation,[77] perhaps because the contrast between juxtaposed human-dominated landscapes and ostensibly "natural" ones is so apparent in the United States (e.g., California's Central Valley and Sierra Nevada mountains; Florida's Everglades and Gold Coast). Yet despite the volume of public debate about wilderness, the concept itself remains poorly defined, even in the United States.*

Most analysts recognize that the term refers to places that have some or all of the following three characteristics: few or no permanent resident human populations, unmanaged biogeochemical systems, and no significant modification by modern technology. Places that meet these criteria might include deep oceans, high mountains, deserts, circumpolar lands, certain oceanic islands, coastal fringes, most areas of active vulcanicity, and some of the world's great forests (such as taiga and tropical rainforests).†

The three criteria are best regarded as necessary but not sufficient to identify an area as wilderness. Spatial dimensions must also be taken into account. An acre of wetland that is surrounded by shopping malls would not be considered wilderness even if it is in biologically pristine condition. As a rule of thumb, a wilderness should encompass at least several square miles.

If the foregoing criteria were to be applied to the United States, they would include a great diversity of environments. Most would be marginal lands and waters, that is, beyond the boundaries of areas that are permanently settled at present and perhaps without prospects for human occupancy or use in the long term. Some protected areas within the ecumene (inhabited lands) might also qualify as wilderness because they are administered as such. But most protected areas, such as national parks, national forests, and national recreational areas, would probably not meet all three major wilderness criteria because they are often subject to intensive management of residual plant and animal populations as well as to human visitors.[31]

Conversion of Wilderness

In many parts of the world the frontiers of wilderness areas are being pushed back as land is converted to managed uses. Economic growth and population increases are the ultimate driving forces of this conversion at the global scale. At local and regional levels a variety of conversion processes are apparent. These include resource extraction industries, such as mining and forestry; agriculture; animal husbandry; tourism; and commercial and residential uses.[62] These processes are not confined to land. They also affect freshwater and shallow-water marine environments. Tourism is exerting pressure on coral reefs in Belize, Kenya, and many other countries.

Most land conversion is driven by demands for additional cropland. The highest levels of land conversion are found in developing countries with rapidly growing populations; by comparison, land formerly devoted to crops is reverting to an uncultivated state in much of Europe, North America, and Japan (Table 95-4). Most of the world's prime agricultural lands have been brought under cultivation, so attention has turned to

*The definition included in the Wilderness Act (1964) is typically vague: ". . . an area where the Earth and its community of life are untrammeled by man, where man himself is a visitor who does not remain." For practical purposes, "roadless areas" are often used as an indicator of wilderness in North America. Even in a well-researched region like North America, exhaustive inventories of the species present in wilderness areas are generally lacking, whereas at the global level, only the approximate distribution of wilderness areas has been mapped. (See the assessment of McClosky M, Spalding H: A reconnaissance-level inventory of the amount of wilderness remaining in the world. Ambio 18:223, 1989.)

†How should formerly developed areas that have reverted to unmanaged states be classified? Many such areas can be found in Western Europe and North America (e.g., the Adirondack Mountains of New York). Often, radical differences exist between predevelopment conditions and reverted conditions. For the purposes of this discussion, such areas are considered wilderness.

other terrains that are spatially and agriculturally marginal.[3] Often these are wilderness areas. For example, during the last 4 decades many formerly unpopulated parts of Sumatra and Borneo have been settled by government-sponsored "transmigrants" from the heavily populated Indonesian island of Java.

Land conversion may fragment existing wilderness areas by dividing them into smaller blocks. This process is well advanced in the Amazon rainforest of Brazil, where new, long-distance, government-built roads bring settlers.[61] As a result, ecologic "islands" that may not be sustainable are created. Forest edge environments replace deep forest ones. The islands may be too small to retain the previous diversity of species. Governments often attempt to protect such islands by designating them as parks or wilderness areas, but this may be insufficient to prevent further changes. In any case, to be effective, such places often require intensive management of ecosystems and visitors, which defeats the objective of designating them as wilderness in the first instance. Moreover, the management actions may ripple through the ecosystems in unforeseen ways, perhaps contributing to the long-term conversion process.

Human Penetration of Wilderness Areas

The recent deadly South Asian tsunami (December 26, 2004) was notable, in part, for the number of foreign tourists that were included among the estimated 300,000 dead. Citizens of 44 different countries from outside the region became victims because they were vacationing in resorts located on exposed islands and remote coasts. Many such places were in or near wilderness areas. Among other things, this "global" disaster marks yet another stage in the increasing spread and penetration of humans into formerly remote places.

Wilderness areas may be degraded without being converted to other uses. This usually occurs in one of three ways: direct impacts from increasing human presence; indirect effects of conventional industrial technologies in adjacent areas; and global effects of innovative, powerful, and often high-risk technologies.

Direct Impacts

Few parts of the planet have not been explored by humans at ground level. Formerly remote areas are being penetrated for a variety of reasons. Winter sports entrepreneurs are shifting attention to Europe's ecologically fragile High Alps as snowfields at lower altitudes shrink under the forcing action of rising temperatures.[76] Canada's James Bay is slowly being altered by a huge hydropower scheme; gold prospecting has intruded into the innermost recesses of Amazonia and Angola; and Philippine coral reefs are subject to cyanide poisoning in pursuit of aquarium fish.[21]

The penetration of wilderness is facilitated by modern industrial technologies, especially transportation technologies. For example, road building encourages invasion of wilderness areas for recreation, resource extraction, or other purposes. The roads themselves have environmental impacts ranging from vegetation clearance to drainage impedance, but their roles as conduits of change are even more significant. They bring new people, exotic materials, and different lifestyles to remote places. Similar inroads are made by boats and aircraft and their support facilities.

Just as economic gain is an important incentive for wilderness penetration, recreational and esthetic needs are also increasing visitation. Hunting and fishing have long attracted visitors to wilderness areas like the Boundary Waters Canoe Area of northern Minnesota. Such pursuits are being reinforced by "ecotourism." An increasing number of people want to visit remote areas to appreciate pristine beauty. For many people who formerly might have sought out Yellowstone National Park and the Grand Canyon, the destinations of choice include places like Antarctica, the high Himalayas, Amazonia, and even Siberia. The more remote the destination, the more attractive. Because most ecotourists want to visit the wilderness for only brief periods, they are whisked in and out by the most modern transportation technologies.

Visits from ecotourists can change wilderness environments. Seemingly insignificant impacts that are repeated can eventually become major problems. In the Masai Mara Reserve of Kenya's Serengeti Plains, the savanna ecosystem has been altered by successive photographic safaris. Safari camps require open campfires; fuel wood is scavenged from fallen trees that would otherwise provide important ecologic niches for local plants and animals. Climbing expeditions on Mt. Everest have reported large volumes of garbage left by earlier expeditions. Decomposition is slow in the dry mountain air. The scarring of scientific sites in Antarctica by discarded refuse and vehicle tracks is well known. Even the Galapagos Islands, the one-time archetypical wilderness of Charles Darwin, are succumbing to the effects of their popularity with ecotourists. Geographers from the United States are assisting the government of Ecuador in carrying out capacity studies that will be the basis for land use regulations and other development controls to limit further degradation of these internationally valued sites.

Indirect Impacts

One of the most potent indirect impacts on wilderness areas follows the introduction (inadvertent or intentional) of nonnative species. Negative impacts have been demonstrated in the United States countless times, such as after the introduction of English sparrows, Asian gypsy moths, and Africanized "killer" bees. Everglades National Park in Florida is now one of the best places to find imported Burmese pythons living in the wild.[27] Another recent example occurred in Glacier National Park. Pack trips in the park were curtailed because horses were introducing exotic species of grasses picked up from stable feed and passed through the digestive tract in the feces.

The problems of small islands and introduced species are legendary. Guam's experience with the brown tree snake is a good example.[96] These snakes are native to New Guinea, but several managed to travel to Guam on airplanes in 1962. They thrived in the absence of native snakes or predators. Now Guam has as many as 30,000 brown tree snakes per square mile, and they have devastated native bird species. These snakes are beginning to show up in the Hawaiian Islands, where conditions are also favorable for colonization. The potential outcome is discouraging, although efforts to intercept the snakes are being increased.

High-Risk Technologies

Technological risks are an increasingly familiar threat to modern industrial society. Such risks are usually perceived as limited to accidents in urban industrial zones like Bhopal, India, where over 3000 people died after the accidental release of methylisocyanate gas in 1984. However, some technologies have the potential to affect very large areas at great distances from the point of origin, up to and including the entire global environment.

Biotechnology exemplifies these powerful, high-risk technologies. Through genetic engineering, new organisms are being created, primarily for agricultural purposes. Nuclear technologies are another example. Like biotechnologies, they are characterized by considerable uncertainty about potential impacts. For decades after World War II, a massive nuclear war between the United States and the Soviet Union was a serious possibility that would have brought catastrophic changes to the earth as a whole.[66] Many military nuclear facilities were located in remote areas throughout the United States and Soviet Union. With the end of the Cold War, this threat has diminished, although regional nuclear conflicts among lesser powers are still possible. The risks for nuclear weapons accidents also remain. Nuclear bombs have been lost at sea, improperly managed nuclear wastes have exploded in the Ural Mountains and elsewhere, and military nuclear wastes are buried on small Pacific islands, often within reach of rising sea levels. Environmental contamination around nuclear weapons manufacturing plants in the United States has been reported, and nuclear submarine propulsion systems have been discarded into the Arctic Ocean north of Russia.

Civilian uses of nuclear technologies pose risks to wilderness areas. Accidents like the explosion and fire at the Chernobyl nuclear power station have had global repercussions. Deposition of highly radioactive fallout in Arctic areas of Scandinavia demonstrates that no wilderness is immune from the effects of major nuclear accidents.[4] The proposed placement of a repository for high-level nuclear waste in Yucca Mountain in the middle of semiarid Nevada provides another example of the connection between high-risk technologies and wilderness areas.[13]

CONSEQUENCES OF ENVIRONMENTAL CHANGE

Research on global environmental change continues to reveal an ever greater number of connections between human and natural systems. Linkages among species in a given ecosystem, among different ecosystems, and among global biogeochemical systems have been described. Providing details about all vulnerable systems is not possible, but the range of interconnections can be illustrated by two examples.

Scientists have recently explored the likely impacts of environmental changes on users of wilderness areas in northern Canada. In one case, rising temperatures and increased precipitation were judged likely to pose few problems for rafters and canoeists on the Mackenzie River, but accompanying forest fires were seen as much greater threats. Further north, on Bathurst Island, the likelihood of increased winter snowfall combined with larger summer insect populations seemed likely to stress the existing large caribou herds to a point where hunting might have to be curtailed. Throughout the region, a shift from consumptive uses of wilderness lands (e.g., hunting) to nonconsumptive ones (e.g., scenic tourism) is a potential outcome.[10] Elsewhere in the Arctic, there might be serious effects on resident and visitor populations. For example, "Indigenous people, dependent on climate conditions that support specific vegetation like forage for cattle and tundra climate commercial crops will need to change their lifestyle and adapt to suit the new environment. These changes in lifestyle would have long-term implications in all aspects, including health. It is projected that although health conditions from frostbite and hypothermia would decrease with the reduction of cold stress in the region, heat-related diseases would become more common. Indirectly, changes due to adjustments of dietary practices and weather conditions would change bacterial and viral proliferations that would result in specific health effects."[5]

Coral reefs provide a second illustration of environmental change effects. Such reefs are among the most prized of wilderness ecosystems. Major reefs, like the Great Barrier Reef of Australia and the reefs off Belize, are national and international treasures. Coral reefs cover only 0.17% of the ocean floor, an area about the size of Texas.[28,75,90] However, the importance of such reefs far exceeds their physical extent. Their biologic diversity is second only to that of tropical forests; their productivity is among the highest in the world; they protect adjacent lands from wave action, nourish valuable fish populations, and generate millions of dollars in tourist revenues.

When subject to physical or chemical stress, coral "bleaches," losing color as a consequence of biochemical changes. Such stresses may be caused by fluctuations in sea level, temperature, or salinity and by pollution. Although reefs sometimes recover, bleaching often leads to death of the coral organisms and decomposition or disintegration of the reefs. In 1987, marine scientists began to notice high levels of coral bleaching and mortality off Puerto Rico. A worldwide pattern of severe coral bleaching began to emerge.[11] Some scientists interpreted the problem as a harbinger of global warming, but it is unclear that this is the case. Nonetheless, coral reefs are vulnerable to temperature changes and sea level increases, so that the threat of future damage is considerable. The best estimate now available is that sea level may rise an average of 1 m by 2100. Healthy reefs can grow upward by as much as 10 cm per decade, which may allow some reefs to adjust to rising sea level. But if reefs are unhealthy—the evidence of bleaching suggests that many are—the rate of inundation may well exceed the coral's ability to keep pace.[79]

Among the stresses that afflict coral reefs are coral mining for cement, dredging for navigation, coral collection for aquariums, and disruption by divers and commercial fishing. Many places also experience significant biochemical effects from coastal pollution and sediment or pollution runoff from land.

The loss of coral reefs is already significant. Estimates suggest that 5% to 10% of the world's living reefs have been destroyed by human activities. An additional 60% are thought to be at risk over the next 20 to 40 years.[79] The consequences for society are potentially enormous. The physical protection of coastlines could be drastically reduced. The locally rich fisheries of coral islands could be diminished to the impoverished levels that typify deep oceans. Prized tourist attractions would disappear, together with the revenues they generate. Opportunities for the recovery of medicinal products from reef organisms could be lost.* Finally, the genetic resources of the planet could be further eroded. These are just some of the consequences of environmental change for one type of wilderness area. Similar, perhaps larger, effects may occur elsewhere.

Environmental Change and Medical Emergencies

The causes and characteristics of many medical emergencies, and perhaps also the appropriate responses, are directly and

*Kainic acid, used in the diagnosis of Huntington's chorea, has already been developed from coral reef organisms. (See *The Economist,* June 13, 1992.)

indirectly connected with the environment in which they occur. This text contains many examples of emergency medicine challenges that are posed by environments in general and wilderness environments in particular. In some cases an environmental agent causes a medical emergency (e.g., reptile bites, altitude sickness, wild animal attacks). In others, the environment affects the treatment of problems that are not environmentally created (e.g., wilderness trauma and surgical emergencies, hunting injuries, wilderness medical liability). In many cases the environment serves as both agent and context. Inasmuch as the process of environmental change is global in scope, it probably will also affect emergency medicine. A number of examples follow.

Increasing human penetration of wilderness areas by hikers, hunters, skiers, climbers, whitewater boaters, and others is steadily driving up the number and cost of wilderness emergencies. For example, throughout the country the National Park Service spent $3.5 million on 3108 rescues in 2003. Personnel from the Park Service, the U.S. military, and volunteers may be exposed to high risk when called on to retrieve inexperienced and underequipped parties. Given the rising cost of such operations, it has been proposed that individuals who participate in risky adventures should post rescue bonds before departing into the wilderness. California recently enacted a law that permits local authorities to charge the persons who were aided up to $12,000 for each rescue performed by public agencies.[72] Similar laws exist in Hawaii, Idaho, Oregon, and New Hampshire. The combination of increasing populations and projected changes in environmental conditions can only add to the costs and difficulties of search and rescue in wilderness areas in the future.

As settlement advances into wilderness areas, new patterns of disease are likely to form. For example, African land conversion from unmanaged wetlands to irrigated agriculture may spread the range of schistosomiasis and other waterborne diseases that are associated with drainage canals.[73] Likewise, more people may be exposed to virulent diseases of wilderness ecosystems. Conversion of tropical forest in Africa may increase exposure to malaria carried by mosquitoes, onchocerciasis (river blindness) carried by *Simulium* flies, and trypanosomiasis (sleeping sickness) carried by tsetse flies.[7]

Pollutants often migrate into wilderness areas ahead of people. Air pollution is particularly mobile. Higher smokestacks have been a common means of diluting airborne pollutants, but they also allow these materials to disperse more widely. Trees and lakes in New York State's Adirondack Mountains have been affected by acid rains transported from the Ohio Valley, and once-clear vistas in the Grand Canyon have been obscured by smoke from a distant coal-fired power plant. The growing severity of winter haze in the Arctic is a further problem.[82,85] Although the Arctic is a remote area, increasing haze has been observed there for almost a century. This smog consists of many different industrial pollutants that originate far to the south in industrial areas, especially the heavy manufacturing industries of Russia. Intense cold is perhaps the most obvious environmental health hazard in the Arctic, but the buildup of industrial air pollutants may also have significant health effects, both directly on the body and indirectly through uptake by food sources in the Arctic environment.

The effect of weather on human mortality has long been a focus of biometeorologic research.[41] A range of medical conditions that are weather-related are listed in Box 95-2. To take but one, well-established linkages exist between high summer temperatures and human mortality, especially among the elderly.[43] Although "global warming" need not mean that all parts of the earth will experience significantly elevated temperatures, some researchers are convinced that summer heat waves are likely to become more extreme, leading to increased mortality from this cause.[44]

Box 95-2. Causes of Death Considered to Be Weather Related

Active rheumatic fever
Adverse effect of medicinal agents
Cerebrovascular disease
Complications of medical care
Complications of pregnancy and childbirth
Contusion and crushing of intact skin surface
Diseases of the arteries, arterioles, and capillaries
Diseases of the blood and blood-forming organs
Diseases of the digestive system
Disease of the musculoskeletal system and connective tissue
Diseases of the nervous system and sense organs
Diseases of the skin and subcutaneous tissue
Diseases of the veins and lymphatics
Effects of foreign body entering through orifice
Endocrine, nutritional, and metabolic diseases
Fractures of the skull, spine, trunk, and limbs
Hypertensive disease
Influenza
Injury to nerves and spinal cord
Intracranial injury
Ischemic heart disease
Neoplasms: benign and malignant
Superficial injury
Toxic effects of substances of chiefly nonmedical sources

Modified from Kalk LS, Davis RE: Ann Assoc Am Geographers 79(1):44, 1989.

The medical effects of UV radiation have already been noted. Further erosion of the stratospheric ozone layer will undoubtedly increase the likelihood of cataracts, skin cancers, and immune system diseases. Reduction of biodiversity threatens to reduce the availability of natural materials that have medicinal value. Ethnobotanists are currently working with traditional shamans in Amazonia to catalog the medicinal properties of plants in tropical forests. Marine species are also an important source of new medicines. Scientists continue to extract from marine life powerful agents against bacteria, fungi, and parasites that may spur research into naturally occurring antibiotics.

The "ozone hole" is a dramatic example of the expanding capacity of humans to modify the biosphere. Usually the process is inadvertent, and wilderness areas are not singled out for attention. Sometimes, however, the very remoteness and isolation of wilderness areas encourages dramatic environmental changes. Such was the case in northwest Alaska in 1962, when the U.S. government buried 15,000 pounds of radioactive soil at Point Hope.[23] The project was conducted by the U.S. Geological Survey acting in conjunction with the Atomic Energy Commission. The intent was to study effects of Arctic environ-

ments on radioactive isotopes. However, the burial was illegal; no public hearings were held, no markers were erected, and high-level wastes instead of low-level wastes were included. When the land was returned to the Inupiat (Eskimos) in 1971, they were not informed about the buried soils. They now attribute current elevated cancer rates to living and hunting for many years in a contaminated area. Government officials reject this view. The Point Hope case is not an isolated example. There is significant evidence that metropolitan governments have often tended to regard wilderness peripheries and their populations as dispensable when issues of national security and the welfare of metropolitan residents are at stake.[20]

Complexity and Uncertainty

Although we have ample reason to be concerned about the environmental changes that lie ahead for wilderness areas, the subject is hedged with complexity and uncertainty. The potential for change exists, but it is difficult to be certain how fast and how far such changes will proceed. The following two cases illustrate some of the dimensions of complexity and uncertainty.

The north (Na Pali) coast of the Hawaiian island of Kauai is representative of wilderness areas that are particularly vulnerable to climate change. It is one of the most remote and beautiful places in Hawaii, accessible only on foot, from the ocean, or by air, weather permitting. The potential for increased rainfall, storminess, and sea level rise could radically alter this wilderness. For example, increased rainfall on Kauai's massive central peak, Mount Waialeale ("the wettest place on Earth"), would make hiking difficult on steep Na Pali access trails that are already subject to erosion and landslides. The few available campsites near beaches may be eliminated by rising sea level. Sea caves that can be entered only by inflatable small powerboats during calm conditions may become inaccessible. Flash floods in Na Pali streams may erode archeological sites, and increased moisture in the air would add to the mistiness that is now only an occasional feature of the area. Offshore waters host migrating whales that can be seen from the coast, but increased soil erosion might add to sediment loads and discourage these highly valued mammals.

As the Na Pali coast becomes increasingly hazardous to visitors on foot, larger numbers may try to enter by helicopter, with more high-technology–dependent visitors and fewer low-technology–dependent ones. Health and safety emergencies may increase, or the mix of emergencies may change. Already the skies over Na Pali are crowded with noisy aircraft. Several crashes and deaths occur every year. Leptospirosis from Na Pali streams is now a health hazard and may become worse. The bacteria were introduced from Southeast Asia in imported rats and pigs. In 1989, the Hawaiian Islands reported 66 cases of leptospirosis with 2 resulting deaths.[95] Despite the potential for problems, no one can yet say with certainty which, if any, of these changes will come about. Nonetheless, we see strong indications that the Na Pali coast will not remain in its present state.

A second case that illustrates the complex interplay of environmental linkages and the potential for problems is provided by the highlands of Papua New Guinea.[2] Since the sweet potato was introduced to this area in the 1500s, it has become a staple crop for residents of remote mountain valleys. Sweet potatoes are susceptible to frost damage and tend to deplete mountain soils. In response to these constraints, villagers have developed specialized social and agricultural adjustments, including the practice of "mounding" and a complex system of resource exchanges between residents of higher elevations and lower elevations. Global warming might reduce the frost hazard, but increased precipitation or increased UV radiation could also threaten crop survival. At present we have no way of confirming the extent and severity of possible changes. But clearly a delicately worked out system of human ecology like this one would not remain unaffected by climate changes of the type that are anticipated in the next several decades.

WHAT MIGHT BE DONE ABOUT LIMITING ENVIRONMENTAL CHANGE?

At the beginning of this chapter it was suggested that change is a dominant, perhaps "normal," feature of the world's landscapes and environments. What is different about the present era of environmental change is the extent to which it is directly attributable to human decisions and actions. It seems unlikely that people will stand by and do nothing if the anticipated changes are perceived as threatening, especially if they are also perceived as caused by humans. However, also likely are responses to environmental change that will not be motivated solely by concern about environmental hazards, such as medical emergencies in wilderness areas. Recognition is growing throughout the world that improved environmental quality is an appropriate goal for all countries, not just developed ones. Therefore, public policies toward the environment will seek both to mitigate risks such as those connected with environmental emergencies and to secure rewards by safeguarding and enhancing valued resources such as wilderness areas. Let us briefly examine some recent developments that provide clues to future public policies.

Changes in Environmental Science and Policymaking

Traditional concepts of wilderness have emphasized the value of nature in a pristine condition, a condition that must be protected and preserved against human modifications. As argued herein, such a view does not square with the vast bulk of scientific knowledge that recognizes the pervasiveness of past human impacts on natural systems and looks toward future environments that are even more completely human-dominated. Such recognition implies that science and society might both pay attention to restoration of degraded environments as well as to preservation of little-disturbed ones. Environmental management policies might focus on redevelopment of so-called "brownfields," as well as development of more familiar greenfield sites. Both of these trends are clearly detectable in practice. Restoration ecology has become a new growth area in the environmental sciences and a new entry in the toolkit of environmental managers.[42] The implications for wilderness areas of such a shift are considerable. On the one hand, a restored watershed is not the same as one that has never been allowed to deteriorate; neither is a hand-reared endangered species the same as one that survives without direct human help. But on the other hand, it is possible that environments might be restored to states that are functionally equivalent to wilderness. This raises fascinating, but as yet unanswerable, questions. Would the availability of human-constructed alternatives to natural areas mean a slackening in political pressures to preserve "authentic" wilderness? Would "synthetic wildernesses" contain suites of

medical risks similar to those in other kinds of wilderness? It is simply not possible to answer these questions at present. At this point, we must interpret and manage wilderness as it now exists using a more or less conventional battery of environmental policies, programs, and initiatives.

International Actions

The first Earth Day (April 22, 1970) ushered in an important era of environmental politics in the United States.[12] The immediate result was a striking increase in public awareness of environmental issues. The United Nations Conference on the Human Environment (Stockholm, 1972) played a similar role on the international stage. In June 1992, the United Nations Conference on Environment and Development (UNCED) marked the 20th anniversary of the Stockholm meeting and focused attention squarely on the global environment and emerging issues of environmental change. Several significant international agreements that addressed the task of slowing environmental change were signed[30]:

1. The Rio Declaration, a broad agreement on principles of environmental management and development
2. The Convention on Climate Change, a nonbinding treaty for industrialized and developing countries that is intended to stabilize and eventually reduce greenhouse gas emissions
3. The Convention on Biodiversity, a nonbinding treaty that establishes a framework to preserve the planet's biologic diversity and to share products derived from genetic stocks

These agreements are unusual because they broadly and directly link issues of environmental management to issues of economic development, and because they took place under the glare of international publicity in a global forum. Many other international agreements are more narrowly targeted and have been signed without such fanfare. They include, for example, the Montreal Protocol on CFCs, the Convention on International Trade in Endangered Species of Wild Fauna and Flora (CITES), the London Dumping Convention, the World Heritage List, and various agreements about the protection of Antarctica. CITES aims to limit trafficking in endangered species, and the Dumping Convention bans ocean disposal of wastes.

Important as are agreements among governments, they do not constitute the only kind of international action to limit environmental change. Increasingly, nongovernmental organizations (NGOs) have become significant international policy actors. Such organizations can serve as catalysts for responsible environmental management by marshaling public support for governmental agreements. They can also take action themselves, as in the case of so-called debt-for-nature swaps.

Debt-for-nature swaps were first begun in 1987 as an innovative way to accomplish three goals: to ease the debt burdens of developing countries, to relieve the international banking community of risky development loans, and to protect important environmental resources in debt-ridden developing countries. The mechanism operates as follows. An environmental organization purchases some of the debt of a developing country from a lien-holding institution. Because no one expects the loans to be paid back in the near future, they have lost their luster as financial instruments and can be purchased well below face value. The organization then forgives the debt in return for the debtor country's agreement to undertake environmental protection measures. These may include establishing preservation areas or deferring environmentally destructive development proposals. Debt-for-nature swaps offer one means for private organizations in the developed world to make a direct commitment to limiting unwanted environmental changes in developing countries. However, at present they fall short of being an adequate solution because the amount of debt greatly exceeds the funds that are available for buy-backs.

At the end of the 1990s, NGOs began to pioneer even more important roles in the shaping of international environmental policy. Perhaps potentially the most far-reaching of these has been the rise of broad-based coalitions of environmental, human rights, and labor groups to oppose what they perceive as the hegemony of the World Trade Organization (WTO). The collapse of WTO meetings in Seattle (December 1999) as a result of such opposition may be the opening engagement of a much broader global campaign toward striking a better balance between a desire for open markets and a potentially stronger impetus toward environmental protection and social equity. If it develops as forecast, the movement will be complex and changeable. For example, one dimension may involve challenges to scientific protocols, standards, and labels upon which many U.S. and international food safety regulations are based, as in the cases of genetically modified foods and "mad cow disease" (bovine spongiform encephalopathy). Another form may include wage rates, import quotas, and workplace environmental protections that affect the manufacture of goods in emerging markets in developing countries. Yet other dimensions may be concerned with regulating the differential burden of greenhouse gas contributions to the global atmosphere, or the constitutional rights of women and minorities.

National Actions

Because national states retain sovereignty over most of the resources within their borders, they are still the most powerful institutions for managing the global environment. For example, the U.S. government has authority to regulate pollution, manage federal lands, and control waste disposal.[67] It also has important supervisory and review powers over the actions of state governments. Finally, it has strong indirect influence on economic factors that are deeply implicated in environmental issues.

The federal government's record of regulating environmental change in wilderness areas is complex and illustrates both the strengths and limitations of existing national level public initiatives. Approximately 4.5% of U.S. territory (104 million acres) is now included in the National Wilderness Preservation System, together with about 10,700 miles of free-flowing waters that are part of the Wild and Scenic Rivers System.[18] The purpose of these designations is to protect the relevant areas against significant modification by humans. Most (60%) of the designated wildernesses are in Alaska, and the bulk of the rest in the 11 westernmost coterminous states. Typically, a wilderness area is embedded within, and surrounded by, other types of public land, such as National Forests, National Parks, or fish and wildlife reserves. Many types of human uses and human activities are permitted in the surrounding lands, which have spillover effects on the wilderness areas. Wilderness management is usually in the hands of the same agencies that administer the surrounding public lands and has often been a neglected stepchild of those agencies. Moreover, recent research has disclosed that the boundaries of wilderness areas are often ill-suited to permit the survival of many species they contain. Especially in the case of migratory animals or those with large territorial ranges, what happens to them outside the wilderness

TABLE 95-5. National Environmental Lobbying Organizations

ORGANIZATION (YEAR FOUNDED)	MEMBERSHIP (in 1000s)					
	1960	1969	1972	1979	1983	1990
Sierra Club (1892)	15	83	136	181	346	560
National Audubon Society (1905)	32	120	232	300	498	600
National Parks and Conservation Association (1919)	15	43	50	31	38	100
Izaak Walton League (1922)	51	52	56	52	47	50
The Wilderness Society (1935)	10	44	51	48	100	370
National Wildlife Federation (1936)	—	465	525	784	758	975
Defenders of Wildlife (1947)	—	12	15	48	63	80
Environmental Defense Fund (1967)	—	—	30	45	50	150
Friends of the Earth (1969)	—	—	8	23	29	30
Natural Resources Defense Council (1970)	—	—	6	42	45	168
Environmental Action (1970)	—	—	8	22	20	20
Total	123	819	1117	1576	1994	3103

Modified from Mitchell RC, Mertig AG, Dunlap RE: *Soc Nat Resources* 4:219, 1991.

is just as important as what happens within. It therefore makes little sense to restrict efforts for limiting environmental change to wilderness areas alone. Such efforts usually need to be applied to the private lands and waters that interdigitate with the federally managed wilderness and nonwilderness areas. In other words, holistic management principles that can be applied across governmental boundaries both within and between countries are necessary.

Together with business and industry, environmental interest groups are important nongovernmental participants in shaping U.S. environmental policy. Although direct-action groups like Greenpeace often receive publicity in the mass media, most U.S. environmental groups are political lobbying organizations. As shown in Table 95-5, membership in the major environmental organizations has increased greatly in the last 2 decades, especially during the Reagan presidency, when environmental issues were widely perceived to have been ignored by the U.S. government.[59]

New environmental interest groups are also joining the fray. For example, the American Medical Association has recently formed an Environmental Health Task Force that is charged with studying harmful environmental issues such as waste disposal and ozone depletion. In addition, the National Association of Physicians for the Environment was established in April 1992 to educate physicians about environmental hazards to human health and to develop recommendations for policymakers.[39]

Local Actions

One of the most popular environmental slogans of recent years is "Think Globally, Act Locally." This advice recognizes that most environmental issues need to be addressed at the grassroots level if they are to be resolved successfully. In local communities, the effects of environmental problems are most forcefully felt by individuals, and the "not in my back yard" (NIMBY) syndrome is a powerful deterrent to projects with perceived negative impacts on environment and health.[78] For example, opposition to creating sites for sanitary landfills has raised garbage disposal costs to the point that recycling has become an accepted part of daily activities for many Americans. In other words, the political pressure of the NIMBY syndrome has yielded significant environmental benefits.

Many different kinds of local initiatives can affect the rate at which wilderness areas change. Formal measures, such as planning and zoning regulations or restrictions on the availability of public utilities, are obvious examples. But less conventional approaches may also be significant. Some local communities in the United States have sought to discourage further development of fragile remote areas by informing prospective newcomers about the discomforts and drawbacks of rural living.*

Nontraditional leaders are an emerging facet of local environmental activism. In the United States, women play key leadership roles in many local environmental groups. The same is true in developing countries such as India (the Chipko Movement) and Kenya (the Greenbelt Movement).[48] Chico Mendes, a Brazilian rubber-tapper who urged a slowdown in clearance of Amazonian forests, is another widely heralded example.

Looking across the range of environmental change issues and responses, new institutions and new philosophies of human–nature relations are emerging and being linked to a broad range of public concerns. Issues of environmental change are now seen as intertwined with issues of economics and security. In other words, the principle of diversity in natural systems, which imparts resilience in the face of stress, is now being replicated in social systems. This is a hopeful sign at a time when environmental changes are unprecedented in rate and magnitude.

SUMMARY

Environmental change has been a characteristic feature of the earth's history, but today the changes are unprecedented in

*This is a strategy favored by officials in Spokane County, Washington, who point out that in many areas public services are irregular and unreliable, emergency response times for medical emergencies are not guaranteed, perceived amenities such as forested glades can turn into liabilities during wildfire season, whereas floods, snowstorms, and landslides frequently disrupt transportation and communications (*New York Times,* May 30, 1999).

human experience. Nonetheless, people are capable of taking positive steps to confront and manage anticipated changes, despite complexity and uncertainty. During the next several decades, wilderness areas will be particularly affected by environmental transformations that occur within and around them—perhaps more so than at any comparable period in the past. Like other sectors of society, emergency medicine and medical systems will face new challenges as a result. Wilderness medical systems may need to be redesigned with particular attention to improving their flexibility.

The references for this chapter can be found on the accompanying DVD-ROM.

96 Wilderness Management and Preservation

Chad P. Dawson and John C. Hendee

Wilderness is a word that carries various connotations and denotations among different people and cultures. Often, wilderness suggests a special place with human appeal or aversion that invokes an associated emotional, psychological, and mental state because of the natural and undeveloped characteristics of the area, whether real or perceived. Humans have a deep historical and cultural connection with "wild nature" because we have been shaped through human evolution by it and, in turn, we have modified it. Whether a person has a direct experience with wilderness, views wilderness in art and photography, or reads about the adventures of others in wilderness—the human reaction is complex and forms the basis for both conscious and subconscious memories.

In the United States and several countries around the world, the word *wilderness* is associated with a legal definition for places that are legislatively protected. The places selected and designated for protection follow certain general guidelines, and this political process is influenced by very strong and diverse groups of stakeholders and the general public. The management objectives for these areas include a variety of values, from preservation of the ecologic conditions and processes to human use and enjoyment (Fig. 96-1). Although there is widespread public support for wilderness, there are divergent and polarized viewpoints on how to define wilderness, ranging from extreme protectionists who believe that humans have no place in wilderness to the utilitarian interests that hold that wilderness is a setting for economic development for recreation and tourism activities.

Although there are a variety of definitions of wilderness, the United States has a legal definition of wilderness, albeit somewhat vague, that is the basis for the creation of the National Wilderness Preservation System (NWPS). By 2003, the NWPS included over 700 management units and more than 106 million acres of publicly owned lands managed by four federal agencies. The purpose of this chapter is to outline the legal designation, management, and preservation of wilderness areas in the United States.[7]

HISTORICAL DEVELOPMENT OF THE WILDERNESS CONCEPT

The term *wilderness* historically was used to describe places that were untamed and not under control of humans, whereas *civilization* was the place of human control.[7,10] Areas of civilization that were cultivated and heavily influenced by human activities often bordered or were surrounded by areas that had little human influence. As the population of the world has grown and more land area has come under human influence, wilderness has been lost to the point that it is now scarce in many areas of the world.

There are few places that are not now, or have not been at one time, under human control, habitation, cultivation, or influence. A gradient of human influence and impact exists from urban centers and rural areas to some wild country (e.g., wilderness) that has little or no human influence (Fig. 96-2). The so-called "human footprint" on the world is large and extending rapidly with population growth, road building, food production, power generation, industrialization, and human habitation.[11] Some identifiable "last of the wild places" exists on each continent and might continue to do so with careful conservation of resources and protection worldwide of some remaining representative or remnant areas of each ecologic community type.[11]

In the United States, there are few older adults who have not commented on the change in the landscape and extent of development in the country during their lifetimes. The early history of the United States during European immigration was one of cultivating and taming the wild places and taking dominion over the land for human habitation. Wilderness was seen as a

human experience. Nonetheless, people are capable of taking positive steps to confront and manage anticipated changes, despite complexity and uncertainty. During the next several decades, wilderness areas will be particularly affected by environmental transformations that occur within and around them—perhaps more so than at any comparable period in the past. Like other sectors of society, emergency medicine and medical systems will face new challenges as a result. Wilderness medical systems may need to be redesigned with particular attention to improving their flexibility.

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Figure 96-1. Day hikers en route to a summit attempt on snowcapped South Sister Mountain (3158 m [10,358 feet]) in the Three Sister Wilderness, a 286,708-acre area managed by the U.S. Forest Service in west-central Oregon. (Photograph by Chad Dawson.)

Figure 96-2. Wildlife is both an attraction and a danger to visitors in the Okefenokee Wilderness, a 353,981-acre wildlife refuge swamp and wilderness area managed by the U.S. Fish and Wildlife Service in southern Georgia. (Photograph by Chad Dawson.)

place for exploration and primitive travel, and was often feared and avoided by most of the population. As the amount of land with wild conditions began to diminish, it was more appreciated as a change from cities and civilization. The public's interest in wild places evolved as they became scarce. Special places were first set aside as National Parks, such as Yellowstone, Yosemite, and the Grand Tetons. These areas were at first seen as park destinations for the development of recreation and tourism, rather than as preserves.

The early interest in wilderness began with employees in the federal land managing agencies, such as the Forest Service and National Park Service. After World War II, a greater public interest began to emerge to save areas for wilderness character. Some of the emerging concern to save certain places by designating them for protection as wilderness was due to interest in recreation experiences coupled with a growing concern about rapid industrialization and population growth transforming the landscape through human activities. The human population in the United States stood at over 290 million by 2004. Some would argue that there are few places in the world that are wilderness in the strictest sense of the word. Thus, the more common usage of the term *wilderness* is in relation to our perception of areas that are little known or predominantly under the influence of natural processes and forces. Although the term had been commonly applied to any large, remote area with natural characteristics, conditions, and processes, by 1964 it gained a new legal definition that was applied to federally owned land areas designated as wilderness by congressional action in the United States.

WILDERNESS LEGISLATION AND POLICY IN THE UNITED STATES

The Forest Service and National Park Service did not begin to set agency policies to protect primitive and roadless areas from development until the 1920s. During the following decades, roadless area inventories and administrative designations of wilderness occurred with increasing public interest. As recreational use and interest in these lands increased, concerns were raised by professionals in the agencies and the public that the administrative regulation (1) allowed too many development activities, such as mining, grazing, motorized access, and water resource development; (2) shifted boundaries or removed designation to permit resource development; (3) promulgated different regulations and management in different areas; and (4) had neither a distinct policy for wilderness preservation nor a national system with coordinated management.

The concept gradually evolved that legislative protection was needed to create a more permanent and coordinated national system for wilderness preservation and management. From 1956 to 1964, more than 50 versions of a wilderness bill were introduced in Congress, heavily debated, supported, and challenged by different interest groups. Political compromises were deemed necessary finally to get a wilderness bill passed into legislation, and so human activities were permitted in some areas, even though they would be nonconforming with the intent of the wilderness legislation.[7,13] These included activities such as mining, grazing, aircraft landings, and water resources development.

In 1964, the U.S. Congress passed The Wilderness Act (U.S. Public Law 88-577).[15] This legislation created the NWPS and was heralded by the environmental community and the general public as one of the most important pieces of conservation legislation in U.S. history.[13] Scott comments[12] that, "Before there was a Wilderness Act, wilderness was, at best, an afterthought. Only the U.S. Forest Service had actually delineated wilderness areas, propelled by visionaries within its own ranks. . . ."

The Wilderness Act defines a broad statement of policy for designating wilderness under the Act and recognizes the need to set aside significant natural areas for present and future generations because of the rapid loss of such resources:

> *In order to assure that an increasing population, accompanied by expanding settlement and growing mechanization, does not occupy and modify all areas within the United States and its possessions, leaving no*

lands designated for preservation and protection in their natural condition, it is hereby declared to be the policy of the Congress to secure for the American people of present and future generations the benefits of an enduring resource of wilderness. For this purpose there is hereby established a National Wilderness Preservation System to be composed of federally owned areas designated by Congress as "wilderness areas," and these shall be administered for the use and enjoyment of the American people in such manner as will leave them unimpaired for future use and enjoyment as wilderness, and so as to provide for the protection of these areas, the preservation of their wilderness character, and for the gathering and dissemination of information regarding their use and enjoyment as wilderness. (U.S. Public Law 88-577, section 2a).

This paragraph is referred to by land management agencies as the "guiding management intent" because it specifically refers to human "use and enjoyment," provided the areas were "unimpaired" and that management would ensure "preservation of their wilderness character."

Section 2c of The Wilderness Act includes an important and often-quoted definition of wilderness that has led to much controversy and debate because although it is poetic in form, it has left room for interpretation, especially during legal hearings and court cases in the 40 years since its passage:

A wilderness, in contrast with those areas where man and his own works dominate the landscape, is hereby recognized as an area where the earth and its community of life are untrammeled by man, where man himself is a visitor who does not remain. An area of wilderness is further defined to mean . . . an area of underdeveloped Federal land retaining its primeval character and influence, without permanent improvements or human habitation, which is protected and managed so as to preserve its natural conditions and which (1) generally appears to have been affected primarily by the forces of nature, with the imprint of man's work substantially unnoticeable; (2) has outstanding opportunities for solitude or a primitive and unconfined type of recreation; (3) has at least five thousand acres of land or is of sufficient size as to make practicable its preservation and use in an unimpaired condition; and (4) may also contain ecological, geological, or other features of scientific, educational, scenic, or historical value. (U.S. Public Law 88-577, section 2c)

The definition emphasizes that wilderness is in contrast to civilization, human influences, and habitation. The definition is an ideal that is tempered by four conditions to make it practical and applicable in a world that has historic human activities and impacts that may no longer be noticeable to visitors.

One of the conditions refers to "outstanding opportunities for solitude or a primitive and unconfined type of recreation"; that phrase is often referred to as the guiding principle for recreation and visitor management. A careful reading of the definition of wilderness makes it clear that preservation is the overall principle and reason for designating an area "wilderness." Certain types and amounts of recreation are permitted, provided the area is "protected and managed so as to preserve its natural conditions." This is an important principle to keep in mind when discussing recreation use and management (Fig. 96-3), especially when it relates to primitive facilities and trails, backcountry travel, recreational equipment (e.g., removable climbing gear, backpacking stoves), and management interventions.

Figure 96-3. Tourists often test their snow-climbing skills on the easily accessible snowfields above Paradise in the Mount Rainier Wilderness (4393 m [14,410 feet]), a 228,480-acre wilderness area managed by the National Park Service in western Washington. (Photograph by Chad Dawson.)

The use of helicopters and other motorized equipment during search and rescue operations is often thought of as a necessary exception to the natural preservation principle of wilderness, with the humanistic and legal rationale being that human health and safety should take precedence. However, the extent and frequency of search and rescue operations have led to such an intrusion in high-use mountain areas that "outstanding opportunities for solitude" are being diminished. Balancing human interest in risk-taking activities, such as helicopter drop-offs for high-altitude skiing or multiday high-wall rock climbing, with human self-preservation instincts in the case of those injured or needing rescue, will remain a controversial subject in wilderness management.

Creation of the NWPS with The Wilderness Act in 1964 was just the beginning of legislative designations. By 2003, there were more than 130 different laws passed by the U.S. Congress designating new areas or adding acreage to existing areas[7]. As Hendee and Dawson[8] note,

in addition to The Wilderness Act, 40 years of subsequent legislation have clarified congressional intent to protect and manage our nation's wildest remaining lands as wilderness and has expanded the NWPS. . . . It is hard to identify any natural resource issue—or any issue—for which Congress has so consistently and so often confirmed their intent as they have with wilderness.

The initial designation of 9.1 million acres of wilderness was followed by congressional designations in 28 of the 40 years between 1964 and 2004 to add additional acres and units to the NWPS (Table 96-1). The greatest growth (64%) in the NWPS occurred during 1976 through 1980, and the largest

TABLE 96-1. The Acreage of Wilderness Designated in 1964 at the Creation of the National Wilderness Preservation System and in 5-Year Intervals through 2004

YEAR	ACREAGE	PERCENTAGE OF TOTAL
1964	9,139,721	8.6
1966–70	1,153,382	1.1
1971–75	2,612,902	2.5
1976–80	68,027,642	64.0
1981–85	8,563,271	8.1
1986–90	5,560,032	5.2
1991–95	8,772,384	8.2
1996–2000	1,119,621	1.1
2001–04	1,332,445	1.2
Total	106,281,400	100.0

Source: www.wilderness.net, accessed January 15, 2005.

single increase was the addition of approximately 56 million acres in Alaska under The Alaska National Interest Lands Conservation Act of 1980 (Public Law 96-487).

Many proposals for additional units and acreage are still being brought before Congress and its committees. Several authors and organization have predictions that more acreage will be added to the NWPS in coming decades, but the estimated amount of those additions varies widely.[7,13] Scott,[12] a historian of wilderness policy and legislation, offers the observation that, "However much wilderness Americans may choose to designate through their elected representatives, future generations are likely to judge that we preserved too little, rather than too much."

WILDERNESS STEWARDSHIP PHILOSOPHY

Some management of wilderness resources and experiences is necessary as visitor use increases and surrounding land management and use affect the wilderness area. The idea that we need management in an area that was intended to be free of the influences of modern human activities may appear paradoxical. However, wilderness stewardship is the management of human uses of wilderness and internal and external influences on wilderness to protect and preserve an area's solitude and naturalness, including natural processes and conditions. Hendee and Dawson[8] highlight the wilderness stewardship philosophy for managers:

> *Wilderness management should not mold nature to suit people. Rather, it should manage human use and influences so as not to alter natural processes. Managers should do only what is necessary to meet wilderness objectives and use only the minimum tools, regulations, and enforcement required to meet those objectives. In wilderness, people adapt to nature, to naturalness and solitude, and that is the source of human benefits from wilderness experience as well as the ecological and non-use benefits.*

This stewardship philosophy is based on the wilderness legislation and is balanced between two often-debated ends of a continuum. At one end of the continuum is wilderness for its own sake with protection of the naturalness foremost, and at the other end of the continuum is wilderness primarily for human use and enjoyment. The stewardship philosophy favors the natural integrity of the wilderness ecosystems, with some accommodation for primitive styles of recreation and the opportunity for solitude. The long-term results are that the natural forces and processes that shaped and formed the American wilderness will be evident in the wilderness that we leave for future generations.

POTENTIAL THREATS TO WILDERNESS

Designating an area as wilderness is just the first step and must be followed by stewardship to maintain those areas that represent all that is left of many ecosystems, as well as natural landscapes that have not been cultivated, mined, developed, urbanized, or otherwise heavily altered by human activities. Numerous types of internal and external conditions, influences, and changes threaten wilderness resources and values, now and in the future.[8] Three examples of categories of threats are summarized here to highlight the concern about the future sustainability of wilderness conditions and processes.

Wilderness areas, in many states, are increasingly isolated fragments or remnants of historic ecosystems. As the surrounding landscape becomes more developed and inhabited, wilderness areas become ecologic islands that can continue with various ecologic processes, provided they are large enough or are not disconnected from other natural areas. This concern is most pronounced in the eastern United States, with its smaller wilderness areas, but the threat is felt throughout the country as the natural landscape is replaced by human influences, habitation, and manipulation.

Exotic and non-native species of plants and animals are invading wilderness and are a direct threat to naturalness and wildness. Efforts to control and manipulate these invasive species can have additional impacts on wilderness conditions that are also undesirable. Invasive plant species, like knapweed, cheat grass, and purple loosestrife, can rapidly change an ecosystem and fundamentally alter its historic patterns and conditions for native plant and animal species.

Increasing commercial and public recreation use of wilderness and efforts to control its impacts are a serious threat to wilderness resources and values. High visitor use has obvious impacts that can be identified, but what may not be as easily observed are changes to opportunities for solitude and the impacts of regulations and enforcement on social conditions. Regulation takes away some of the freedom of choice and spontaneity to explore that many visitors associate with wilderness experiences.

Hendee and Dawson[8] identified 17 categories of internal and external threats as the change agents that affect wilderness conditions and values:

1. Fragmentation and isolation of wilderness areas as ecologic islands
2. Impacts on threatened and endangered species
3. Increasing commercial and public recreation use
4. Permitted livestock grazing
5. Invasion of exotic and non-native species
6. Administrative access, facilities, and intrusive management
7. Adjacent land management and use

8. Private and public land inholdings within wilderness
9. Established mining claims
10. Wildland fire suppression activities
11. Reduced air quality
12. Reconstruction and maintenance of water projects and reduced water quality
13. Advanced communication and navigation technology that reduces solitude
14. Motorized and mechanical equipment trespass and legal use
15. Aircraft noise and air space reservations
16. Urbanization and encroaching development
17. Lack of political and financial support for wilderness protection and management

The concern is that few of these threats will diminish, and most are projected to increase in the coming decades. Land managers will need to monitor these potential threats to prepare management plans and activities to steward designated wilderness areas and minimize, mitigate, or remove the threats. The responsibilities at the national level for these wilderness planning and management activities are primarily under the jurisdiction of four federal agencies.

WILDERNESS MANAGEMENT AGENCIES IN THE UNITED STATES

The NWPS included more than 700 units managed by 4 federal agencies and totaled more than 106 million acres of publicly owned lands by 2004 (Table 96-2). The four federal agencies administering the NWPS are the National Park Service (NPS), Bureau of Land Management (BLM), and Fish and Wildlife Service (FWS) in the Department of Interior, and the Forest Service (FS) in the Department of Agriculture. The NPS has the greatest total area of wilderness at 43.6 million acres and the fewest units for a federal agency; the largest area in the NWPS, Wrangell-St. Elias Wilderness (over 9 million acres) in Alaska, is under NPS management. The FS has the largest number of wilderness units to manage and approximately one third of the total NWPS acreage. The FWS manages 19% of the NWPS area, including the smallest unit in the system, 5-acre Pelican Island Wilderness in Florida. The BLM has significant acreage in some of the desert ecosystems of the west and manages many smaller units in its 6.8% of the NWPS.

The four federal land managing agencies have promulgated regulations based on the wilderness legislation and have developed policy and management documents to steward the lands under their jurisdiction. In addition, the process of evaluating additional lands and managing them for potential inclusion in the NWPS continues for all four agencies. Although the NWPS is a national system operating under the same legislation, each agency has developed its own procedures and organizational approach to accomplishing the task of protecting the "enduring resource of wilderness," based on each agency's administrative mission and structure. Some of the different approaches to visitor and resource management can be confusing to visitors who do not have an appreciation or understanding that the overall mission of each agency is different. For example, the FWS has a wildlife management mission that incorporates a national wildlife refuge system.

DISTRIBUTION OF WILDERNESS IN THE UNITED STATES

The 106 million–acre NWPS represents just over 4.5% of the U.S. land area, in contrast to the more than 6% of total acreage in urban and suburban land area or more than 20% of the total in agricultural cropland.[8] The NWPS is an attempt to designate wilderness areas that would represent the different geographic regions and ecosystems of the United States. The representation of ecosystems is not complete (less than 50% of types are represented), and it is more effective for some arid lands and mountain ecosystems of the west than for coastal lowlands, grasslands, and eastern hardwood forests.[8]

Forty-four of the states have federally designated wilderness, ranging from a 77-acre island wilderness area in Ohio to over 57 million acres in the state of Alaska.[7] The six states without federally designated wilderness are in the midwestern or northeastern United States. When the number of acres of designated wilderness in each state or region is compared with the total land area and total population, the pattern is an uneven distribution favoring the western United States. Less than 5% of the NWPS is located in the eastern United States, where more than one half of the population resides on over 40% of the U.S. land area.[7] The Pacific and mountain regions of the western United States have approximately 22% of the population and more than 95% of the NWPS. The greatest disparity is that Alaska has more than one half of the land area of the NWPS and less than 1% of the U.S. population.

Whereas the NWPS is based on The Wilderness Act of 1964 and federal land ownership, there are also eight states that have designated state wilderness areas on state-owned lands since the

TABLE 96-2. The Number of Designated Wilderness Management Units and Their Acreage by 2004

AGENCY	WILDERNESS UNITS	ACRES	PERCENTAGE OF TOTAL
Bureau of Land Management	175	7,280,521	6.8
Fish and Wildlife Service	71	20,699,108	19.4
Forest Service	406	34,867,591	32.8
National Park Service	55	43,650,796	41.0
Total	707	106,498,016	100.0

Source: www.wilderness.net, accessed January 15, 2005.

1970s. Six states in the midwestern and eastern United States have added more wilderness areas (i.e., state-designated wilderness). Most notable is the New York State system, which has added approximately 1.1 million acres in the Adirondack and Catskill Forest Preserves.[7] California and Alaska state wilderness systems also added more to each of those states' total wilderness areas through both federal and state wilderness designations. The eight state wilderness programs protect over 2.6 million acres.[8,12] These are managed by the state land managing agencies and are not part of the NWPS. Many states have legislation and management programs modeled after the federal Wilderness Act.

The NWPS is extensive and complex geographically and is often difficult for visitors to locate because it is generally shown on agency maps only as part of overall public land holdings. For example, the Boundary Waters Canoe Area Wilderness in Minnesota is part of the Superior National Forest. A very helpful source to see the geographic distribution and location of the units in the NWPS is available at www.wilderness.net and is provided by the Wilderness Institute at The University of Montana's College of Forestry and Conservation, the Arthur Carhart National Wilderness Training Center, and the Aldo Leopold Wilderness Research Institute. This site also links to the managing federal agency, the wilderness area's legislative history, and visitor information sources.

WILDERNESS VALUES AND PUBLIC PERCEPTIONS

The American public is strongly supportive of wilderness designation and the NWPS.[1,3] Scott[12] reported a summary of seven different surveys in the United States from 1999 through 2002 that showed 48% to 81% of respondents supported designating more wilderness land in the United States into the NWPS.

Observations on the relationship between public visitors to wilderness areas and the values they hold for its protection indicate that the striking trend over time is for a broad base of support across American society and an intense commitment from a subset to fight for wilderness protection:

> *Although wilderness means something different to everyone, four central themes have consistently emerged: experiential, the direct value of the wilderness experience; the value of wilderness as a scientific resource and environmental baseline; the symbolic and spiritual values of wilderness to the nation and the world; and the value of wilderness as a commodity or place that generates direct and indirect economic benefits.*[7]

National surveys in 1994 and 2000 reported[3] that over 50% of the public indicated that 12 of 13 wilderness values were very or extremely important to them (Table 96-3). The 1994 to 2000 surveys showed a trend to increase the percentage for all 13 values, indicating a higher level of value. The nonuse values tended to dominate the higher average value scores (scale ranged from "not important" to "extremely important"), with the highest value for protecting water quality and air quality. Strong support for the value of income for tourism industry use on wilderness lands tended to be reported by less than 30% of the respondents.

Western U.S. residents were somewhat more often aware of the NWPS than were easterners (60% versus 56%); conversely, eastern residents more often reported there was not enough land in the NWPS than did westerners (53% versus 48%).[3] Metropolitan and urban residents more often reported there was not enough land in the NWPS than did rural residents (54% versus 44%).[3]

TABLE 96-3. Percentage of Americans (≥16 Years of Age) Indicating a Response of Very or Extremely Important for 13 Wilderness Values

WILDERNESS VALUE	PERCENTAGE
Protecting water quality	93.1
Protecting air quality	92.3
Protection of wildlife habitat	87.8
For future generations	87.0
Protection for endangered species	82.7
Preserving ecosystems	80.0
Future option to visit	75.1
Just knowing it exists	74.6
Scenic beauty	74.0
Recreation opportunities	64.9
For scientific study	57.5
Providing spiritual inspiration	56.5
Income for tourism industry	29.7

From Cordell HK, Tarrant MA, Green GT: Int J Wilderness 9(2):27–32, 2003, with permission.

WILDERNESS VISITORS

The diversity of wilderness visitors ranges from those who take short walks and view scenery and wildlife in an hour to multiple-day backpackers, week-long backcountry hunting trips with pack animals, and mountain climbing expeditions. Growth in the amount of recreation demand and the increasing popularity of many forms of recreation in wilderness is due to U.S. population growth and a general upward trend in participation in many activities, on all types of sites, in the United States[2] on public or private lands. Although backcountry and wilderness use is distributed across the full geographic and sociodemographic spectrum, there is an identifiable 8.6% of the U.S. population that has been labeled as "backcountry actives" by one researcher because of their 2.5 times or greater above-average participation in such activities as backpacking, wilderness visits, cross-country skiing, and day hiking.[2]

Studies at high-use federal agency sites of visitor use of wilderness in the mid-1990s estimated that over 14 million visitors went to the wilderness per year.[1] Public surveys of the general population in recent years estimated that visitation was closer to 40 million per year.[1] Most estimates of future growth suggest that wilderness use will continue to increase (2% to 4% per year) and that participation will continue across a wide range of activities.[1,2]

Recreation enthusiasts spending 7 or more days in wilderness or primitive areas per year are a growing market segment that includes participation in strenuous physical activities on a regular basis.[2] Improvements in and availability of high-technology gear permit travel in all types of weather conditions and terrain, so that regular use across the entire landscape makes it more difficult to manage use in general, as well as more

challenging to conduct search and rescue operations. Risk-taking, exploring, and adventure activities are increasingly prevalent because of media exposure, easier access to sites, more available high-technology gear, more opportunities for initiation into activities, and more training and skill-building opportunities. There is some concern that communication equipment, like satellite cell phones, and navigational equipment, like hand-held Global Positioning System (GPS) units, may contribute to the impression that a person could call for help more readily and therefore take greater risks than their skills otherwise would warrant.

One of the ways that people are initiated into wilderness use and gain training in primitive travel and living is through participation in Wilderness Experience Programs (WEPs). The number of WEP organizations and their clientele have grown rapidly in the United States in the last 2 decades.[5] Wilderness land managers recognize WEPs as a significant user group with a focus on programs in wilderness and primitive areas as one of their defining characteristics.[4]

The most prevalent three types of WEPs are (1) education programs where the wilderness ecosystem is the focus of the instruction, research, and field trips; (2) personal growth and development, where wilderness is the setting and metaphor for everyday life, with insights achieved from challenging activities and reflection; and (3) therapy and healing, where wilderness is the setting to seek restored normal functioning and a healthy balance through primitive living and traveling.[4,5] Although the healing aspects of a natural environment[6] and wilderness areas are well documented, the inherent risks of traveling in remote areas must be recognized through risk management assessment and contingency planning,[14] especially for human health and safety.

DISTRIBUTION OF WILDERNESS VISITOR USE

Visitor use is very unevenly distributed in geographic space and across time. Each wilderness has popular access points because of easy highway access from urban centers, information in guidebooks, word-of-mouth information dissemination, and many other factors. There is often congestion in visitor use at these access sites and crowding along popular trails and at campsites and destinations (e.g., lakes and ponds, scenic overlooks, historic sites). Many wilderness areas have these types of heavy-use access sites; conversely, each area also has places with very low use or no use for a variety of reasons. This variation in visitation means that not every acre of wilderness or the associated experience has the potential to be experientially unique. Hence, there is a continuum of solitude and naturalness across wilderness.

Some of the greatest variations in visitation are due to seasonality and opportunity for use. Each area has a favored season for a given type of activity. Even though most hiking and camping activities occur during late spring through early fall, some areas have other peak use times because of weather and opportunities present. For example, spring fishing and white-water boating may occur in the same area, whereas fall hunting and backpacking during fall foliage may be followed by cross-country skiing and winter camping. Cooler weather in desert areas may bring visitors in nonsummer months (Fig. 96-4). Warmer months in alpine areas may bring large numbers of visitors. Weekend and weekday variations are normal fluctuations due to workweek schedules.

Figure 96-4. The lower Sonoran Desert in the North Maricopa Mountain Wilderness is a scenic and difficult place to access. Visitors may not understand the dangers of heat and dehydration in this 63,020-acre wilderness area managed by the Bureau of Land Management in southern Arizona, where temperatures commonly exceed 100° F. (Photograph by Chad Dawson.)

Some of the variation patterns in visitation are due to geographic location in the United States and proximity to urban population centers. The majority of use in each wilderness area comes from the surrounding region and states, regardless of whether one considers the urban proximate White Mountain National Forest wilderness in New Hampshire or the Bob Marshall Wilderness in Montana. However, there is considerable long-distance travel and higher visitation to some of the larger and more popular wilderness areas that have unique opportunities and features. Every wilderness area has a great variation in the number of people at one time and in any given place. Although such variation in visitation is natural, the impacts may be difficult to manage, leading managers to use more direct and heavier-handed management techniques to reduce the negative consequences to resources and other users.

WILDERNESS MANAGEMENT PRINCIPLES

The guiding principles for managing wilderness areas[7] revolve around the idea that wilderness needs to be managed as a pristine extreme in the landscape (over 4% of U.S. land area) to maintain the distinctive qualities that define and separate wilderness from other land uses (over 95% of U.S. land area). Wilderness is managed from the biologically centered perspective. Environmental integrity and primeval conditions of wilderness are the basis for any human enjoyment, values, and benefits.

Managing wilderness as an ecosystem and not as a separate set of resource types (e.g., water, forests, wildlife) focuses managers on a more comprehensive perspective on the protected area. Most wilderness areas represent the remnants of ecosystems or entire ecosystems and, as such, need to be protected for present and future generations if they are to be available to experience and enjoy. In addition, it is imperative that human uses and influences be managed to preserve wilderness condi-

tions and characteristics because without such stewardship these remaining areas would lose their unique value in the U.S. landscape.

If wilderness is to be managed to maintain or improve wilderness conditions and not allow degradation on sites or across the area, then an understanding of the carrying capacity of the area to sustain recreational use is essential. One of the major components in managing wilderness recreational use is to manage use in favor of recreation and human activities that depend on wilderness conditions to achieve their goals, while not degrading the wilderness conditions. In other words, there are other places to have various recreational experiences that do not require wilderness conditions. Thus, only those activities that require such conditions should be allowed in wilderness, and only as much as the area can sustain while maintaining its wilderness conditions and processes.

One of the implications of such a management philosophy is that wilderness is not primarily a place for recreational use, although it is permitted as long as it does not impinge on the capacity of the area to maintain its wilderness conditions and processes. All management activities, including search and rescue operations, should have as light an impact on the wilderness and user experience as possible. This is sometimes referred to as the "minimum tool or regulation" that achieves the management objectives for the area and maintains the highest levels of naturalness and solitude. Examples are using hand tools instead of gas-powered tools in wilderness maintenance activities, using educational materials in place of direct trip management, or using minimal directional trail signs and not mileage markers.

Section 4c of The Wilderness Act provides for the administrative use of vehicles, such as aircraft and motorized equipment, when it is considered the minimum necessary for certain operations like search and rescue. In the case of emergency situations, the use of aircraft, helicopters, and other motorized transport during search and rescue operations is often thought of as necessary for human health and safety. However, the extent and frequency of search and rescue operations has, in some places, become an intrusion affecting both the environment (e.g., wildlife behavior) and the human experience of solitude. Balancing the need for effective and rapid search and rescue operations with impacts on the wilderness resources and experiences will be an increasingly controversial subject in wilderness management. Determining the level of search and rescue operations and activities that is effective with a minimal impact on wilderness resources and experiences is an ideal goal that needs further attention and study. For example, in nonemergency search and rescue operations, the minimum may be determined to be nonmotorized access using humans on foot to search or to carry a minor accident victim out in a Stokes basket.

WILDERNESS PRESERVATION AS A NATIONAL AND INTERNATIONAL MOVEMENT

The number of and membership in organizations involved in promoting wilderness designations, stewardship, information, and education have grown dramatically over the last 40 years. Organizations can be found at international, national, state, and local levels.

The U.S. legislative model has influenced some forms of international wilderness protection,[9] although the variation in level and type of protection is complex and based on the cultural and legislative history in each country. The concept of wilderness is universal, but the national legislative approach used in the United States has been widely adopted by other countries, such as Canada, Australia, Finland, Russia, and South Africa.[9] There are dozens of international organizations that one can join to help protect wilderness worldwide. Some examples include the WILD Foundation (www.wild.org), Conservation International (www.conservation.org/xp/CIWEB/home), Sierra Club (www.sierraclub.org), The Wilderness Society (www.wilderness.org), and IUCN—The World Conservation Union (www.iucn.org). Many non–U.S. countries have strong public support for wilderness and related organizations that support wilderness designation and stewardship.

Wilderness protection and management of federal lands in the United States is under the jurisdiction of four federal agencies: the NPS, BLM, FS, and WFS. Each of these agencies has policy and operational information that is important for visitors to understand. This information is available at agency websites, but is easy to obtain through websites such as www.wilderness.net that allow access from a geographic map that, in turn, brings the viewer to useful information from the designating legislation to the managing agency and local-level contact offices for that wilderness unit. Obtaining local contact information and some general visitor management information makes wilderness visitation more enjoyable and improves the pretrip planning process, including compliance with local regulations.

Wilderness stewardship organizations in the United States include many that have been involved in wilderness issues for decades. Only a few examples are listed here: The Wilderness Society (www.wilderness.org), Sierra Club (www.sierraclub.org), American Wilderness Coalition (www.americanwilderness.org), Campaign for America's Wilderness (www.leaveitwild.org), Wilderness Watch (www.wildernesswatch.org), Wild Wilderness (www.wildwilderness.org), National Wildlife Federation (www.nwf.org), National Audubon Society (www.audubon.org), and the Izaak Walton League (www.iwla.org).

Wilderness preservation is a national and international movement comprising grass roots and membership organizations interested in protection and stewardship of our dwindling wild areas. Although the concept and values of wilderness are supported by the general population of the United States and many other countries, it is the continued support and work of many people and organizations that stimulate the legislative and administrative branches of our government to continue their efforts to maintain parts of our country wild for present and future generations. The reader is encouraged to become part of that wilderness preservation movement. For a comprehensive resource, the reader is referred to Hendee and Dawson.[7]

The references for this chapter can be found on the accompanying DVD-ROM.

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The New Frontier

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Wilderness medicine is often about making do with less—providing care in an austere or hostile environment, where medical personnel and resources are limited or absent. The aerospace environment embodies this frontier medicine attitude; not only are there constraints associated with available medical capabilities, but also the very physiology of the casualties themselves is altered. If space is, as one Doctor McCoy might suggest, the "final frontier," then space medicine largely remains the "undiscovered country." Relatively little is known about how the human body adapts to and recovers from long-term exposure to microgravity (notated as "μG" or, for many practical purposes, "0G"), let alone the best ways to treat illness or injury that occur in space.

In January 2004, U.S. President George W. Bush called for a renewal of exploration missions beyond low earth orbit (LEO). These missions will require not only application of lessons learned from past flights, but also additional investigation into the best ways to safeguard the health of travelers as they transition from the 1G environment of Earth to the free fall of interplanetary space to the partial gravity of the moon or Mars, and then back again. Each of these environments may require unique equipment and procedures, as well as improved understanding of how and when the physiologic changes associated with microgravity occur.

Space is arguably the most hostile wilderness humans have ever faced. It is an "extreme environment," imposing both direct (e.g., radiation) and indirect (e.g., isolation) challenges. For example, a Mars mission may well have medical evacuation capabilities no better than those of the Lewis and Clark expedition (though they will presumably not meet any helpful natives along the way), resupply lines similar to those of early explorers into the Australian interior, and psychological challenges as profound as those experienced by members of Antarctic winter-over teams. In order to provide the best possible care for their patients and to ensure success of the mission, space medicine physicians must make use of the knowledge acquired by all of those previous groups, as well as by other practitioners of wilderness medicine.

MEDICAL CHALLENGES ASSOCIATED WITH THE PHYSIOLOGY OF SPACEFLIGHT

Time Course of Changes and Adaptation to Microgravity

The microgravity environment provokes numerous changes to the space traveler's physiology. Some of these alterations occur immediately, while others have a more delayed onset; effects can be short-lived or last for the duration of the mission. As a result, "space normal" physiology can be difficult to define, and the ability of an astronaut to respond to a physiologic insult, such as injury or illness, can also be challenging to predict.

The physiology of the space traveler is most labile during the transition to or from microgravity. Within a few days, the body adapts to its new environment (as described later), but in the first 72 hours following a change to or from microgravity, most of the physiologic processes are in a state of flux.

Short-Term Effects

Immediately upon exposure to microgravity, certain physiologic systems exhibit altered function. Many of these are associated with maintaining moment-to-moment homeostasis or are directly affected by the physical effects of microgravity. Examples of these are the baroreceptor reflex, neurovestibular system, and gastrointestinal tract.

The majority of these systems adapt to microgravity by resetting to a new equilibrium state within a short time (hours to days). Upon return to a gravitational field, the same rapid effects may be seen in reverse.

Long-Term Effects

Several physiologic systems exhibit microgravity-related effects over a longer time frame (weeks to months) (Fig. 97-1). For short-duration missions, these changes may be minor or even undetectable, but on longer flights, the effects can become more pronounced. In some systems, it is unclear whether a "space normal" equilibrium is ever achieved, or whether changes continue so long as the crewmember remains in microgravity. Examples include alterations in red cell mass and bone demineralization.

Life Science Research Topics Have Changed Over Time

In the beginning days of the manned space program, missions were quite short (the Mercury program had six manned flights from 1961 to 1963; most were under 5 hours in duration, although the last was 34 hours). As a result, life sciences questions tended to focus on whether humans could survive in the space environment and ignored many of the normal physiologic functions as not (yet) relevant. For example, Alan Shepard's spacesuit for the first Mercury flight, Freedom 7 (Fig. 97-2), didn't even provide him with a way to relieve himself (a perhaps forgivable oversight, since the flight was only scheduled to last

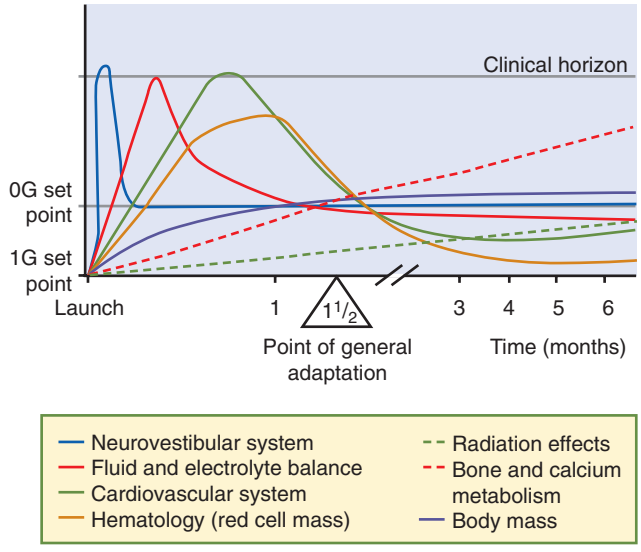

Figure 97-1. Time course of physiologic changes. (From DeHart RL, Davis JR [eds]: Fundamentals of Aerospace Medicine, 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2002.)

Figure 97-2. The Mercury-Redstone 3 rocket Freedom 7, carrying Alan Shepard on the first American manned suborbital flight, launches on May 5, 1961. (From NASA Image Exchange [NIX] at http://nix.larc.nasa.gov/info;jsessionid=1snd99vh206gk?id=GPN-2000-000859&orgid=12.)

15 minutes; unfortunately there was a lengthy delay on the launch pad).[73]

By the Gemini program (1965 to 1966, with flight durations from under 5 hours to nearly 14 days), researchers needed to know how best to help humans survive in the microgravity environment for longer periods (up to 2 weeks), and investigations began into what activities humans could reasonably do in that environment. For example, mission objectives for the Gemini astronauts included subjecting humans (and their equipment) to space flights of up to 2 weeks in duration, accomplishing a rendezvous and docking with orbiting vehicles, and conducting the first spacewalk (or extravehicular activity [EVA]).

During the Apollo program (1968 to 1972), the body's reaction to partial gravitational states (i.e., less than a full 1G) became important for the first time, along with questions as to whether the physiologic adjustments caused by the moon's ⅙ G would impair the astronauts' ability to do meaningful work while on the surface. In point of fact, the crew were not only able to drive the lunar rover, but also to collect geological samples and—when time permitted—to play golf!

Skylab (1973 to 1974) was America's first space station, as well as its first attempt to investigate the effects of long-duration (up to 84 days) exposure to microgravity. Associated issues, such as exercise programs, menu variety, and psychological issues, received heightened attention. Although the Apollo-Soyuz program was as much for political purposes as for scientific ones, it too added to the knowledge base by allowing comparisons between the Soviet and American methods for handling physiologic adaptations.

More recently, the success of the Shuttle program has led to a feeling of familiarity with short-duration (approximately 2 week) missions to LEO, but the International Space Station (ISS) program remains relatively new, with small numbers of crew. Although the ISS missions to date, along with historical data from the Soviet/Russian space station programs, have provided information about long-term effects of microgravity on the human body, much of what is known has been extrapolated from short-duration Shuttle missions. In addition to lingering questions about the mechanisms of adaptation to and from microgravity, as well as the time courses and the best way to prevent or recover from the undesirable changes, research also continues into how ground-based personnel can best support long-duration crews.

Effects on the Cardiovascular and Pulmonary Systems

The cardiovascular system undergoes several predictable adaptations in response to microgravity. Recalibration of baroreceptor homeostasis occurs after several days in space, likely due to cephalad fluid shifts induced by the microgravity environment[45] (Fig. 97-3). As a result of these shifts, the carotid baroreceptors interpret a (relative) hypervolemia and cause a diuresis. Relative hypovolemia then exists (approximately 10% decrease in total body fluid and a fall in central venous pressure (CVP) from a terrestrial average of 7 to 10 mm Hg to 0 to 2 mm Hg), compared with preflight terrestrial fluid status.[25] The fluid shift may also be associated with development of space motion sickness, with resultant nausea and vomiting that lead to further loss of fluids.

Although there is still debate on the effects of microgravity on hemodynamic parameters, the most recent data suggest that heart rate and diastolic blood pressure decrease and cardiac output increases. This implies reduction in peripheral vascular resistance and a similar decrease in sympathetic tone. Pulmonary physiology testing has revealed a decrease in tidal

Figure 97-3. The cephalad fluid shift is measured by determining venous pressure in the jugular vein. (From NP-119 Science in Orbit: The Shuttle & Spacelab Experience, 1981–1986, at http://history.nasa.gov/NP-119/ch2.htm.)

Figure 97-4. A Russian "Chibis" suit makes use of lower body negative pressure to offset orthostatic changes. (From http://vsm.host.ru/r_photos.htm.)

volume and an increase in respiratory rate.[143] There is also a decrease in dead space and improved CO_2 diffusion capacity, possibly due to increased intrathoracic perfusion.[115]

Although these changes have not been demonstrated to affect human health or cardiovascular stamina during spaceflight, orthostatic intolerance following return to Earth's gravity remains a concern. Compounding this effect may be a loss of adrenergic responsiveness; in a number of returning astronauts, lower norepinephrine levels have been found to correlate with deficient responses to orthostatic stress.[123]

The severity of orthostasis is generally proportional to the duration of time spent in space, and poses a problem if an emergency situation requires crew mobility immediately upon landing.[21] It is also a significant obstacle to planning a prolonged mission to Mars, where the crew will be required to function independently immediately upon entering a $\frac{1}{3}$-G gravity environment after several months' travel in microgravity.

Several strategies are currently used as countermeasures against postflight cardiovascular dysfunction. Five-bladder anti-G suits and liquid cooling garments (LCG) became standard equipment during landing, after experience from short-duration Shuttle flights demonstrated that they reduced orthostatic hypotension on landing. The former presumably is effective by minimizing venous pooling in the extremities, whereas the latter reduces heat stress, sweating, and dehydration during the high cabin temperatures of re-entry.[41] Another standard protocol is oral fluid loading with isotonic solutions (such as bouillon or a sports drink–like concoction called Astro-Ade) performed 2 hours prior to landing. This is a short-acting measure to mitigate the relative hypovolemia of spaceflight.

Previous experience with longer duration (>4 months) missions, such as those to the Mir space station, suggested that regular in-flight cardiovascular exercise benefited orthostatic stamina on re-entry, and an exercise regimen is now standard on all ISS missions.

The Russians also employ another in-flight countermeasure to improve postflight orthostatic intolerance: a lower body negative pressure device (LBNP, or Russian "Chibis" suit) (Fig. 97-4). This device draws blood away from the central circulation and into the lower extremities through a negative pressure gradient, simulating a gravitational stress on the cardiovascular system.[44] The Russians have also employed "brazlets," or thigh cuffs, to encourage greater blood distribution in the lower extremities. Fortunately, even severe postflight orthostatic intolerance is a temporary event and within a few days, virtually all long-duration flight crews are able to mount an appropriate response to orthostatic stress.

Effects on the Neurovestibular and Sensory Systems

In the terrestrial environment, gravity plays an important role in neurovestibular homeostasis, providing an internal compass for proprioception. Acute exposure to microgravity disrupts interpretation of linear and angular forces in the vestibular organs and may alter integration of visual and proprioceptive input.

One of the earliest effects of these changes is space motion sickness (SMS). This form of motion sickness, associated with exposure to microgravity, often manifests as headache, nausea, vomiting, anorexia, poor concentration, or general malaise. It is usually a minor and self-limiting condition (generally lasting for the first 1 to 2 days in orbit), yet it is one of the most

Figure 97-5. The Reduced-Gravity Program provides the unique weightless or zero-g environment of space flight for testing and training of human and hardware reactions. The reduced-gravity environment is obtained with a specially modified KC-135A turbojet transport that flies parabolic arcs. (From NASA Image Exchange [NIX] at http://nix.ksc.nasa.gov/info;jsessionid=31kakwlvw0uun?id=MSFC-0001123&orgid=11.)

Figure 97-6. Astronaut Franklin Chang-Diaz participates in an EVA on the ISS; some astronauts report feeling that they might "fall off" the space station. (From NASA Human Space Flight Gallery at http://spaceflight.nasa.gov/gallery/images/shuttle/sts-111/html/s111e5132.html.)

common reasons for pharmacologic intervention in space (most commonly treated with parenteral or rectal promethazine, 25 to 50 mg, administered at bedtime on the first in-flight day).[116] On occasion, however, SMS has persisted for the duration of a Shuttle flight, significantly impairing the affected crewmember's performance.

SMS is believed to be related to fluid redistribution patterns or alterations in bowel motility. It is only weakly correlated with the motion sickness associated with ship or air travel. Even the motion sickness experienced during parabolic flights on NASA's KC-135A Reduced Gravity Research Program (Fig. 97-5), when passengers experience intervals of microgravity, has been anecdotally reported to correlate poorly with in-flight SMS.[52,126] SMS affects over 70% of space travelers. Reliable prediction of its occurrence (particularly in first-time flyers) is difficult.[41] The incidence is somewhat lower among repeat flyers, suggesting some type of training effect.[11]

The operational impact of SMS is considerable. The first in-flight timeline change related to a medical cause occurred because a crewmember suffered SMS. To this day, extravehicular activities (EVAs) are not scheduled within the first few days of a mission because of concerns regarding the effects of SMS during a spacewalk (in particular, the potential for aspiration and fouling of a spacesuit).

Even though SMS can be expected to have resolved several days into a mission, other neurovestibular and neurosensory changes persist, including alterations in eye–head coordination, target tracking, and optokinetic reflex function.[11,41] These adaptations can significantly impact delicate technical operations, such as manipulation of the robotic arm or manually controlled docking maneuvers. In fact, neurovestibular dysfunction was implicated as one cause in the collision of the Russian space station Mir with a Progress resupply vessel in 1997, as well as the "bumping" of a satellite during a capture attempt by the Shuttle's robotic arm.

Neurovestibular dysfunction has also been suggested as a causative factor in the correlation between Space Shuttle mission duration and accuracy of landing speed, position, and touchdown vertical velocity. As mission duration increases, landing accuracy decreases, leading to concerns about the safety of manual landings following long-duration missions, such as exploration missions to Mars. This may necessitate, for example, design of a spaceship with automated landing capabilities and demonstrates why physicians and other life scientists need to take part in mission design and planning as well as participating in ongoing operations.

About 80% of space travelers experience perceptual illusions during or after flight. Several different types have been reported: illusory self-motion (both linear and rotational), a sensation of the floor dropping when doing a squat to stand, the sensation of things floating in space, visual streaming (blurring), visual scene oscillation (oscillopsia), object position distortion, visual axis distortion (tilting or inversion), and platform stability illusion. Some crewmembers also experience a sense of being upside-down early in spaceflight.[11] The term "EVA acrophobia" has been coined by spacewalking astronauts to describe a feeling that they might "fall off" their vehicle or "fall to earth" (Fig. 97-6).

Restoring sensory references is one goal of neurovestibular countermeasures. Research is currently underway using preflight virtual reality training to simulate conflicts between the proprioceptive and visual systems and to help flyers become accustomed to the ISS geometry prior to arrival. ISS modules also have glow-in-the-dark directional guides and standardized coloration of "floor" and "ceiling" surfaces.[128,129]

Crewmembers tend to choose an external frame of reference to orient themselves. "Down" direction may be toward Earth,

the vehicle "floor," or wherever their feet may be. Crewmembers using the latter strategy ("my feet = down") seem to experience less disorientation when encountering an unexpected visual stimulus. Additional countermeasures include use of in-flight sensory input stimulators and exercises to reaffirm "up/down" visual cues. For example, watching a video while exercising on a treadmill has anecdotally improved postflight symptomatology upon return to a 1G environment.

On return to Earth, the nervous system readapts to a 1G field remarkably well, often returning to preflight states within 48 hours. Postflight neurovestibular symptoms including nausea, illusory movements, clumsiness, and vertigo are experienced, at least briefly, by most crewmembers.[19] These symptoms are usually mild, but in the event of an emergency egress or bailout over the water, they could prove dangerous.[19] Crewmembers in such an emergency not only will have to overcome the musculoskeletal and aerobic deconditioning that may be present, but are also likely to have to cope with unsteadiness, poor coordination, vertigo, and seasickness. Pharmacologic prophylaxis (anti–motion sickness medications) were used for landings during the Skylab program, but were ultimately felt to be counterproductive because of side effect profiles that included sedation.

Effects on the Musculoskeletal System

With exposure to microgravity, mechanical load on the musculoskeletal system lessens dramatically, leading to muscle atrophy (particularly in the postural/antigravity muscles) and bone demineralization (disuse osteoporosis). The latter is one of the high priorities of space physiology research, because osteoporosis remains a limiting factor for astronaut health during and after long-duration flight.[99] Changes are seen within the first few days of space flight and continue throughout exposure to microgravity.

Although the mechanism of this muscle atrophy is not well understood, initial studies suggest that the loss of muscle mass is associated with both decreased protein synthesis (approximately 15%) and increased rate of protein breakdown. As muscle atrophy begins, there is concomitant increase in urinary excretion of nitrogen compounds, 3-methylhistidine, creatinine, and sarcosine. Over the course of a long-duration mission, muscle mass losses of up to 50% can be seen (up to 20% on short-duration missions).

These changes are readily seen in the "chicken legs" of a space traveler; although the cephalad fluid shift seen early in space flight can contribute to this effect, the fluid shift is complete after the first week or so of flight, but leg volume (particularly in the thigh area) continues to decrease throughout the course of a mission. Leg volume measurements do not revert to preflight levels immediately upon rehydration and return to Earth, providing further evidence that the changes are primarily associated with muscle atrophy and not fluid shifts.

Other changes in muscle morphology include decreases in muscle volume, cross-sectional fiber area (though not fiber number), contractile proteins, oxidative-to–glycolytic enzyme ratio, and capillary density within muscle tissue. This contributes to a more anaerobic metabolic profile and decreased muscle strength and endurance.

Changes in bone structure appear to be caused by increased osteoclastic activity and are particularly pronounced in the spine and legs. Losses of up to 20% of preflight bone mass have been documented and can continue for several weeks (or months) following return to a 1G field. Interestingly, the gender-based differences in bone demineralization rates that are seen terrestrially do not exist in space; men and women lose calcium at approximately the same rate (1% to 2% per month in the lower limbs).

These changes in musculoskeletal structure and function have implications for the amount and type of physical activity that can be performed during space missions and also suggest that crewmembers may be at high risk of injury. Animal studies have demonstrated a microgravity-associated reduction in muscle fiber regeneration and repair, which would imply that should an injury occur, recovery may be prolonged. Similarly, the relative reduction in osteoblastic activity could significantly impair healing of an in-flight fracture. Animal studies also reveal microgravity-related alterations in the architecture of bone, leading to further doubts about the body's ability to properly repair a fracture in-flight.

The persistent diminution in bone and muscle mass is a source of great concern for exploration-class missions. Depending on the mission profile, travel to the destination could require several months, and crewmembers will likely be expected to engage in physical activity on the surface immediately (e.g., specimen collection, habitation module construction). If there have been significant losses in strength and fitness en route, injuries may occur or mission objectives may be compromised. Further exacerbating any physical weaknesses will be neurologic abnormalities in balance, coordination, and gait, all of which increase the risk of falls, fractures, dislocations, and other injuries. It is therefore critical to ensure that space travelers arrive in acceptable physical condition.

To that end, a robust countermeasures program has been initiated on the ISS, making use of both strength and aerobic training. Because enthusiasm for exercise (and compliance with the scheduled program) varies from person to person, it is important for the aerospace physician to educate space travelers about the need for regular exercise, as well as to monitor fitness and strength levels during the course of a mission.

Mechanical countermeasures, such as low-intensity vibrations (to stimulate bone maintenance) or the Russian "penguin suit" (which makes use of elastic bands to force use of extensor muscles) (Fig. 97-7), have been studied to determine their efficacy. Unfortunately, none has as yet proved sufficiently useful to warrant routine use.[21] Pharmacologic and nutritional interventions, such as bisphosphonates and other antiosteoclastic agents or high-calcium diets, are currently under investigation as countermeasures, but none is (yet) routinely used. One concern is that use of drugs to alter calcium balance may have unintentional effects on renal physiology and enhance creation of kidney stones. Artificial gravity infrastructures could someday offer a solution to this problem, but they remain impractical for space travel in the near future.

In the immediate postflight period, crewmembers experience muscle weakness, fatigue, and soreness. Whether this is due to readaptation of cellular processes (i.e., readjustment of protein synthesis and degradation pathways), unaccustomed strain associated with reexposure to gravity, or microinjuries within the muscle tissue is unclear.

Muscle mass and strength tend to recover rapidly on return to Earth and initiation of a postflight exercise regime. Within 2 weeks, muscle strength is generally recovered, though it is unclear if there are any associated changes to connective tissue structures (such as tendons and ligaments).

Figure 97-7. The Russian "penguin suit," with built-in resistance bands. (From Russian/Soviet Space Suits at http://www.globaleffects.com/C_b06_frameset.html?Page=rentalC01_b06_S_russian.html.)

Despite rigorous rehabilitation programs, full recovery of preflight bone mass can take years. In part, this is due to the slower time course for terrestrial mineralization (100 mg of calcium per day) compared to space-associated demineralization (250 mg per day), which suggests that it will take two to three times as long to recover bone mass as it did to lose it. In other words, it may take up to 18 months to recover the amount of calcium lost from bones during a six-month mission. In addition, different parts of the skeleton lose and recover mass at different rates. For example, demineralization of the lumbar spine has been noted to linger for up to 6 months postflight, even as other parts of the spine were recovering and adding mass. As a result, there is a desire to maximize bone density prior to flight, and many crewmembers engage in preflight exercise programs to increase bone density. This is particularly important for groups at highest risk for the development of osteoporosis (e.g., white women, persons with a positive family history).

Effects on the Gastrointestinal and Genitourinary Systems

Weight loss, dehydration, and anorexia are common gastrointestinal symptoms during spaceflight.[41] Reduced gastric emptying and altered intestinal transport time have also been documented.[41,51a] Weight loss, likely augmented by fluid losses and muscle atrophy, may also be related to negative nitrogen balance, possibly due to persistent protein loss. Anorexia may be related to altered taste sensation, perhaps due to cephalad fluid shifts. Increased stressors and time pressures associated with mission objectives may influence food choices, with snack or "handheld" foods being preferred to full meals.

Significant efforts are made to offer space travelers a variety of nutritionally balanced foods, but weight loss remains a common finding. Lack of refrigeration and fresh produce necessitates reliance on processed foods. Research continues into developing agricultural methods for use on future long-duration and/or exploration missions.

With the exception of the nausea related to postflight SMS, there is no evidence of postflight gastrointestinal changes. Crewmembers appear to recover their preflight weight and nutritional balance rapidly, though it is unclear how much of a role is played by the physical rehabilitation program undertaken for the musculoskeletal system.

Regarding genitourinary changes, there is a well-documented increase in the incidence of renal stone formation. Micro-

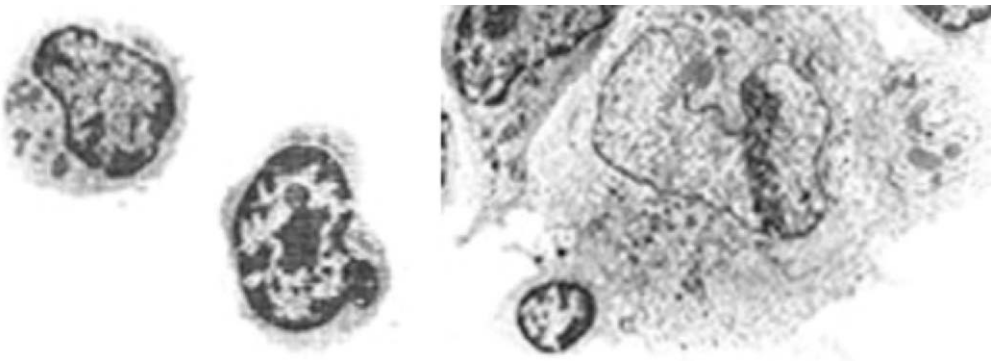

Figure 97-8. White blood cells cultured in microgravity (*left*) did not reproduce at normal rates, but white blood cells cultured in space on the 1-G centrifuge (*right*) proliferated normally. (From NP-119 Science in Orbit: The Shuttle & Spacelab Experience, 1981–1986, at http://history.nasa.gov/NP-119/ch2.htm.)

gravity-induced increases in bone metabolism contribute to increased calcium excretion in the urine, with a stone incidence of up to 5%.[21]

To prevent renal colic, the current hydration recommendation for crewmembers is to exceed 2.5 L per day.[72] Persistent bone loss during extended missions (and the increased risk of renal stones) remains a significant obstacle to planning a Mars mission.

The relatively short duration of missions to date has yielded sparse data on the male and female reproductive systems. Radiation exposure remains a concern for reproductive health. Reversible reductions in male testosterone levels have been reported, and although poorly studied, retrograde menstrual flow remains a concern needing further elucidation. Efforts to preserve gamete cells may be a future preflight consideration due to prolonged exposure to radiation.

Effects on the Endocrine System

The changes in plasma volume due to the cephalad fluid shift occur early in space flight and give rise to natriuresis through both pressure effects on the glomerulus and other factors, such as antidiuretic hormone, the renin-angiotensin-aldosterone system, and calcium-regulating hormones. There also appears to be relative downregulation of neurohormonal receptors during space flight.

Among the effects of microgravity on the endocrinologic system is establishment of a new balance between osteoblastic and osteoclastic activity. Reductions in circulating vitamin D and glutathione, presumably related to decreased load exerted on bones, lead to diminished calcium absorption.

Catecholamines also exhibit changes associated with space flight; in-flight levels of urinary norepinephrine are periodically increased, whereas postflight levels of both norepinephrine and epinephrine are elevated relative to preflight values. Epinephrine is felt to reflect stress levels, while norepinephrine is an indication of physical activity. It is interesting that given the stresses of space travel (e.g., isolation, busy schedules), in-flight levels of epinephrine are generally unchanged from preflight. This could suggest that the human body adapts relatively well to space flight and does not find microgravity per se to be an overly stressful environment. There have been general increases in cortisol levels noted during space flight, but studies into hormonal levels have demonstrated wide variability. Differences in diet, exercise, sleep, and emotional stress are likely contributors to the variation seen and suggest that endocrine changes are too subtle and complex to permit easy categorization. This is also why no countermeasures to these (generally adaptive) changes (yet) have been developed.

Neurohormonal changes affect the cardiovascular system postflight by blunting the baroreceptor reflex and impairing the body's ability to respond to an orthostatic challenge. This can contribute to residual orthostasis. Just as the body adapts to the microgravity environment through changes in the endocrine and nervous system, so too do the same systems facilitate readaptation back to the terrestrial environment.

Effects on the Immune System

Space travelers demonstrate a significant increase in the number of many types of circulating white blood cells (neutrophils, monocytes, T-helper cells, B-cells), but a decrease in natural killer cells. It is not clear whether these changes are due to increased production or decreased destruction of the cells. Studies have shown that the increase in plasma norepinephrine levels on landing are correlated with increased white blood cells, suggesting that landing stresses produce sympathetic redistribution of circulating leukocytes.

Although studies to date have produced conflicting results, anecdotal data persistently indicate that immune function is compromised in microgravity. In vitro studies show that lymphocytes in-flight are largely unaffected by stimulating agents, suggesting that the white blood cells circulating in the blood, although more numerous than terrestrial levels, may be unable to mount an effective immune response.[34a,146,147] Changes in leukocyte morphology have also been reported, further suggesting that immune function may be impaired (Fig. 97-8).

Some researchers who have documented an increase in plasma cortisol levels posit that it is associated with in vivo corticosteroid-induced immunosuppression. Because microorganisms may be more highly concentrated in the confined environment of a spacecraft, the issue of immunocompetence could be important. To date, there have been reports of (usually minor) illnesses during space flight, but no evidence that illnesses are more severe or longer lasting in space. Whether the findings to date demonstrate changes related to the microgravity environment or merely stress-related effects on the immune system remains unclear.

Subjects in analog environments, such as crews spending the winter in Antarctica, often display latent viral reactivation,[82] so it is not unreasonable to imagine that such an effect could be present in long-duration space flight.

Other than ensuring that space travelers are in good health and have had all standard vaccinations, no countermeasure program for the immune system is currently in place. The crew health care systems of the Shuttle and ISS contain medications, including antibiotics and antivirals, for treatment of disease, and the "health stabilization program" (or quarantine period) immediately before launch is intended to further diminish the likelihood of communicable disease during a mission. Unfortunately, the effects of space flight on the immune system are insufficiently understood to permit development of additional countermeasures at this time.

While in-flight changes to the immune system remain unclear, current work focuses on identifying any alterations in structure and function, along with the associated time course(s). Until these trends have been clearly established, the aerospace physician must assume that immunocompromise, if present in-flight, may persist in the postflight period. As a result, space travelers should be cautioned about the risks of exposure to infectious diseases and the importance of seeking medical help promptly should symptoms develop.

Effects on the Blood, Fluid, and Electrolyte Balance

There is in-flight loss of total body fluids, including extracellular fluid volume, plasma volume, and circulating blood volume.[58] The majority of this volume loss is due to changes in plasma volume and circulating red cell mass.

Blood sodium levels decrease in spaceflight, although the relative ratio between sodium and potassium remains unchanged.[58] Most of the other electrolytes are unaffected by exposure to space, with the one major exception being calcium. Both urine and plasma levels of calcium are increased in conjunction with bone demineralization, while negative nitrogen balance and muscle atrophy lead to elevated urinary levels of nitrogen and muscle breakdown products. Phosphorus levels mimic those of calcium and are also felt to be due to changes in bone metabolism.

Red cell mass has been found to decrease on orbit, regardless of whether an enriched oxygen (Skylab) or sea level equivalent atmosphere (ISS) is present. This "space anemia" appears to be due to decreased red cell production and thus is not one of the effects seen immediately upon exposure to microgravity. Researchers propose that the decrease in plasma volume leads to a relative increase in hematocrit and causes the body to decrease production of erythropoietin and other red blood cell (RBC)–producing factors. As red cells are consumed in the normal life cycle, but not replaced, the red cell mass decreases in what is thought to be an adaptive response to the microgravity environment.

Since the changes in blood volume, fluid status, and electrolytes are thought to be appropriate adaptations to the space environment, countermeasures are not currently employed to prevent or minimize them during the mission. Immediately prior to reentry, however, fluid loading occurs to re-expand intravascular volume and protect the crew against gravity-induced orthostatic intolerance. On return to Earth, the space-euvolemia is now perceived as a terrestrial-hypovolemic state, leading to expansion of plasma volume and relative dilution in hematocrit. This "anemia" triggers increased erythropoietin production and returns the red cell mass to preflight levels. Similarly, the thirst reflex and renal system expand the fluid volume, thus ensuring maintenance of proper fluid and electrolyte levels. In this way, normal physiologic processes reverse the changes following landing and return the space traveler to a terrestrial-euvolemic state. Some positional orthostasis may persist for a few days, so the aerospace physician should inform his or her crew about this possibility and provide suggestions on how to avoid it (e.g., rise from bed slowly), similar to the cautions provided to blood donors.

Effects on Pharmacokinetics and Pharmacodynamics: Implications for Therapeutic Intervention

Pharmacokinetics involves the absorption, distribution, utilization, and metabolism of drugs. Pharmacodynamics involves drug interactions in living systems. Although there is evidence that both vary in the space flight environment (for example, one study on in-flight use of intramuscular promethazine documented less than 5% experience of sedation, while ground-based controls had a sedation rate of over 70%[20a]; bioavailability of certain drugs, including scopolamine and acetaminophen, has been shown to be different in-flight), relatively little is known about microgravity-associated changes to these parameters.[138] In part, this is due to small sample size and limited opportunities for in-flight observations. Unfortunately, analog environments are largely unsuitable for pharmacologic studies, although some work has been done using bedrest and head-down tilt models. As a result, few data can be gathered from anything other than on-orbit research. Unfortunately, this research is often beset by confounding factors, such as relative hypovolemia, changes in diet and sleeping patterns, SMS, muscle atrophy (which could have effects on drug binding by circulating proteins), and stress levels. Other parameters, some as yet poorly understood, can also play a role; for example, hepatic blood flow may be altered in microgravity, and this would be expected to have a significant impact on first pass effects and drug excretion.

Routes of Drug Administration

To date, numerous routes of drug administration have been utilized during spaceflight, including intravenous, intramuscular, subcutaneous, intranasal, inhaled, oral, topical, and rectal. Crew medical officers are trained on drug administration through all of these routes, although oral and intramuscular are the routes most commonly used. However, there is little evidence to show whether drug availability via any or all of these routes is changed in space. As a result, additional research into the efficacy and bioavailability of each of these routes is needed. Particularly, more information is needed for exploration-class missions in order to select the most appropriate pharmacopoeia.

Operational Issues

One of the major operational issues related to drug administration deals with crew medical officer (CMO) training. Unless a physician-astronaut is a member of the crew, an infrequent occurrence on Shuttle flights and one that has not (yet) happened on ISS, the CMOs are minimally trained caregivers. Even for ISS crews, the medical training program is both short (usually around 40 hours) and intermittent (often spread over several months), with little time for hands-on training and development of clinical judgment. As a result, it is unlikely that the caregiver will be experienced in either drug administration or evaluation of drug efficacy. Even physician-astronauts have reported unusual difficulty in obtaining intravenous access in

healthy subjects in space, and alternate methodologies (intramuscular injection, sternal intraosseous infusion systems) may be needed, especially for use in medical contingencies when rapid access is required.

Preliminary data suggest that the shelf life for certain pharmaceuticals is reduced on orbit, presumably due to the higher radiation environment.[40a,42a,112a] Supplementary testing of all drugs intended for flight will likely be necessary, particularly for those intended for exploration-class missions. Without this research, it would be impossible for the aerospace physician to know if a patient's failure to respond to a prescribed drug was due to an erroneous diagnosis, worsening condition, or inactive pharmaceutical. This work may also result in the need for new spacecraft designs, with additional radiation shielding for medication kit(s). Another alternative might be to store drugs in powder form and reconstitute them as needed, because powdered forms tend to have longer shelf lives in the terrestrial environment. However, mixing drugs during space flight poses numerous challenges, from the current inability to create injectable-grade water in-flight, to the increased training requirements for CMOs, to the challenges of creating a homogeneous mixture in microgravity. Research is currently underway in this area, but the technical difficulties are considerable.

Prior to flight, many drugs are taken in test doses by crewmembers. This is done in an attempt to prevent atypical reactions during the mission. Although it is virtually impossible to rule out the possibility of anaphylaxis in-flight, testing the medications ahead of time to ensure there are no allergic reactions is thought to be protective. In addition, it is helpful to identify any side effects that could potentially impact the mission, such as extreme sedation.

MEDICAL CHALLENGES ASSOCIATED WITH SPACE AS THE MOST EXTREME ENVIRONMENT

Gravity

Microgravity

All tasks take longer in space. Experienced astronauts who have flown in space repeatedly and have spent extended periods on space stations acknowledge that, even after they have become familiar with the microgravity environment, "everything just takes longer to do." In addition, some things that we take for granted in earth's 1G field (movement of carbon dioxide away from your face, air-fluid separation, the ability to put something down and find it still there when you look again) do not exist in microgravity.

Partial-Gravity Environment

In the decades since humans last set foot on an extraterrestrial body, relatively little research has been focused on the specific requirements for living and working in a partial-gravity environment. Generally speaking, the assumption has been that so long as there is a gravitational vector, physiologic processes are likely to function more like they do on Earth than on orbit. We may soon be in a position to test that assumption. While it is logical to assume that some tasks that are extremely difficult in the microgravity environment will be less so in the partial gravity on the Moon (⅙ G) or Mars (⅓ G), it is likely that others will require significant adaptation. Furthermore, although the presence of gravity may ameliorate or resolve some of the things that caused difficulty for medical planning (e.g., lack of gravity-based air-fluid separation led to problems in creating an effective medical suction device for use on orbit), it causes other hazards—such as falls—to reappear.

Micrometeoroids and Space Debris Issues

Spacecraft routinely collide with "space dust" of size 1 μ and larger.[90] Meteors (also known as meteoroids or micrometeoroids, depending on their size) are small bodies of solid matter that move through space, having originated in the interplanetary region, presumably from crumbling asteroids or comets.[140]

Most meteors (61%) are made of stone, with a sizable number (35%) composed of iron.[90] The vast majority of the micrometeoroid material is <0.1 mm in diameter.[91] Although their size is tiny, micrometeorites travel at such high velocities that they have very great kinetic energy. When they enter Earth's atmosphere, they are heated by the friction of the thickening air around them and may become visible as "shooting stars." About 10 million kg of this interplanetary dust reaches Earth's surface every day as meteorites or micrometeorites.[60]

Orbital debris is any man-made object in orbit that no longer serves a useful purpose; a great deal of orbital debris is material that comes from other spacecraft. Some estimates suggest that millions of kilograms of debris are suspended in orbit, of which the most numerous component is "fragmentation debris" consisting of old satellite fragments (e.g., dead batteries, unused fuel cells) and deterioration products such as paint chips and aluminum oxide particles left over from rocket fuel exhaust.[110] The remainder is composed of rocket bodies, nonfunctional spacecraft, and mission-related debris.[110] Orbital debris can remain in orbit indefinitely, depending on its orbital altitude. Objects at altitudes less than 200 km (124 miles) are likely to fall to Earth within a few days, whereas those above 36,000 km (22,370 miles) will stay in orbit forever.[109] Debris at altitudes between 200 and 600 km will likely stay in orbit several years; debris at altitudes between 600 and 800 km will stay in orbit several decades; and between 800 and 36,000 km, will stay in orbit for centuries. Humanity's short history of spaceflight to date has already produced a remarkable amount of debris (Fig. 97-9).

Collisions with either form of matter (micrometeoroids or orbital debris [MMOD]) take place at very rapid speeds (~10 km/sec on average or 16 km/sec for head-on collisions), which releases a great deal of kinetic energy even for tiny objects such as paint flakes.[90] As a result, this material poses a danger to space travelers. For Shuttle missions, the risk of a lost mission increases from 1/107 to 1/76 when MMOD is considered.[64] MMOD was termed the "number one loss of crew vehicle risk driver" in a 2003 probabilistic risk assessment (PRA), although the risk and consequence(s) of the MMOD strike vary greatly depending on where on the orbiter the impact occurs.[40] In a September 2002 briefing, a senior ISS engineer reported that MMOD was predicted to cause one ISS evacuation every 214 years.[64] This was considered the second most likely cause for an ISS evacuation, with a medical emergency (including the sequelae of a radiation event) being the most likely cause (estimated at 1 evacuation every 5 years). In the same presentation, data were presented assuming a 40% chance of an MMOD-caused hole in the ISS (over a 15-year lifespan of the station), with a 12.8% chance (1 in 8) of the resultant hole necessitating a crew evacuation.[64]

Figure 97-9. An artist's image of earth showing the currently tracked orbital debris in low earth orbit. (From NASA Orbital Debris Program Office at http://www.orbitaldebris.jsc.nasa.gov/photogallery/photogallery.html.)

The longer a spacecraft is in orbit, the greater the risk of being struck by some form of space matter. The number of micrometeoroids is fairly constant in interplanetary space (e.g., during flights between Earth and Mars), but the gravitational forces near a planet alter density patterns and trajectories. In addition, orbital debris is naturally more plentiful in frequently used orbits, because there have been more opportunities for debris to detach from spacecraft in those areas. As a result, orbital debris varies with altitude and inclination, and NASA is currently able to estimate the probability of an impact with an object for a given orbit.

Approaches to Minimize Micrometeoroids and Space Debris

Very large objects (>10 cm [4 inches] in diameter) will usually be detected in time for the spacecraft to maneuver out of their way, whereas very small ones (<0.1 cm diameter) can be handled by current spacecraft shielding.[91] Accordingly, there remains only a small number that are too big to be controlled by shielding but too tiny to detect before impact. According to the NASA-Johnson Space Center (JSC) Office of Orbital Debris, "debris objects smaller than 0.1 cm generally do not penetrate spacecraft [but] 0.1 to 10 cm debris penetrate and damage spacecraft [whereas] 1 cm debris objects and larger will cause catastrophic failure (for example, loss of functionality of satellite due to impact)."[90] The North American Aerospace Defense (NORAD) Command and United States Space Surveillance Network tracks orbital debris >10 cm in diameter so that spacecraft can maneuver around them; currently ~11,000 of these objects are being monitored. Another approximately 100,000 objects are estimated to fall in the 0.1 to 10 cm range and can neither be accurately tracked nor shielded against. The vast majority of orbital debris (numbering in the millions) are <1 cm and are less likely to cause damage to a spacecraft.

In addition, during the in-flight phase of its mission, the Shuttle is oriented so that as it flies around Earth, the bulk of the vehicle will protect more fragile equipment (and astronauts during spacewalks) from impacts.

Objects 1 cm in diameter can penetrate typical spacecraft shielding, potentially causing depressurization of a habitation module.[90] The speed of such a depressurization depends on the module volume and the size of the hole caused by the debris.

One way to mitigate the risk of depressurization due to an impact is to add more shielding, but this increases spacecraft mass, requiring more lift to get into and maneuver within orbit. Other mitigation strategies include avoiding popular orbits, where more orbital debris exists, and orienting sensitive surfaces (such as solar arrays) away from the direction of travel in order to minimize the effects of an impact.

Crews make every effort to limit creation of orbital debris. Newer satellites are designed to avoid breakup (a more common occurrence in early models) and are placed in lower altitudes so that at the end of their lifespan their orbits will decay and permit destruction during reentry. All NASA flight projects are now required to provide debris assessments as part of the routine project development, and debris tracking continues.

Protection against Micrometeoroids and Space Debris during EVA

Although spacecraft walls are thick enough to protect astronauts from impacts with most of the tiny material, the risk to crewmembers increases when they leave the shielded spacecraft, as during spacewalk. Spacesuits are designed with a layer of shielding to protect the astronauts to the greatest extent possible, but even with this shielding, particles of 1 mm in diameter may still be able to penetrate the outer protective layer of the spacesuit, increasing risk by an order of magnitude.[79,95] Intravehicular astronauts generally only worry about objects larger than 1 cm, but EVA astronauts are at risk from objects larger than 1 mm.

In addition to using layers of material in the space suits to protect against MMOD, EVA astronauts are also safeguarded by limiting exposure through short duration of EVAs and by planning EVAs at times and locations outside of high-risk orbits. LEO is more dangerous for MMOD than will be lunar or Martian EVAs, because the latter do not have orbital debris. Of course, they will have dust from the surface, which is likely to have an abrasive effect. Apollo crewmembers found that the moon dust (actually a coarse grit) scratched their suits and visors and contaminated spacesuit seals and bearings. The electrostatic and mechanical properties made the dust cling to the suit, causing problems in removing it before reentering the habitat. Although Martian dust is likely to be more fine (like talcum powder) than moon dust and therefore less abrasive, the contamination issue will likely remain.

Impact Emergency Procedures

An example of an MMOD impact occurred on STS-94, when an impact crater in one of the orbiter's windows was found after landing.[90] The crater was ~1 mm diameter, and the MMOD object was estimated to be ~100 μ (0.01 cm) in size. Tests indicated that it was aluminum oxide residue from a solid rocket motor. Figure 97-10 shows an MMOD impact from STS-7.

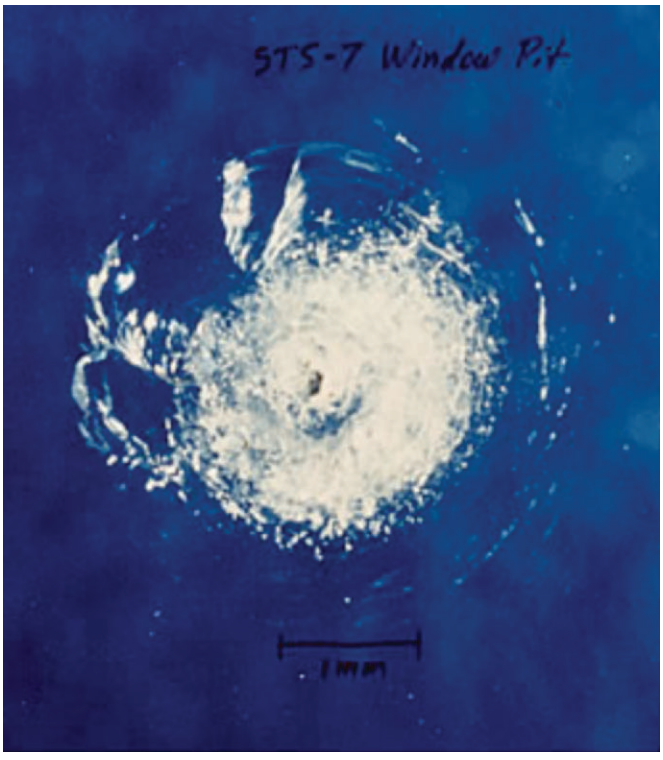

Figure 97-10. An MMOD impact on the orbiter window during mission STS-7. (From NASA Orbital Debris Program Office at http://www.orbitaldebris.jsc.nasa.gov/photogallery/photogallery.html.)

Another example, this time of an unmanned satellite, occurred in 1996, when a briefcase-sized piece of an Ariane 1 rocket body collided with a Cerise communication satellite at 14 km/sec (31,500 mph), severing the satellite's 6-m stabilization boom.[90,109]

Following the Mir/Progress collision in 1997, when a Spektr module was punctured and lost its pressure, the crew was able to evacuate safely in a controlled fashion because the leak was relatively slow. The Spektr module was sealed off, and although it eventually dropped its internal pressure to vacuum, the rest of the station never dropped below 675 mm Hg (normal is 750 mm Hg).[98,124]

Radiation

Radiation exposure remains one of the most challenging obstacles in executing a prolonged manned mission in space. On earth, humans are protected from ionizing radiation by the magnetosphere, the magnetic field that filters out high-energy solar and cosmic particles from deep space.

There are three main sources of radiation that pose risk to manned spaceflight: galactic cosmic rays (GCRs), solar radiation (solar particle events [SPEs]), and the Van Allen radiation belts. The Space Shuttle and ISS lie in LEO, where most exposure comes from protons and electrons trapped in the Van Allen belt and from SPEs.

In 1958, Dr. James Van Allen first described a "belt" of particles, predominantly protons and electrons, trapped in Earth's magnetic field (Fig. 97-11). The proton belts extend to an altitude of approximately 20,000 km (12,428 miles) with peak intensities at 5000 km (3107 miles), and the electron belts extend to 30,000 km (18,642 miles) with peaks at 3000 km (1864 miles) and 15,000 km (9321 miles). Although the magnetic field traps potential sources of radiation exposure, it also acts as a filter for the much more energetic heavy ion radiation of deep space. Galactic cosmic rays are extremely energetic ions ranging from protons to iron nuclei, and have the most potential for radiation exposure on deep space missions, such as landing on Mars.

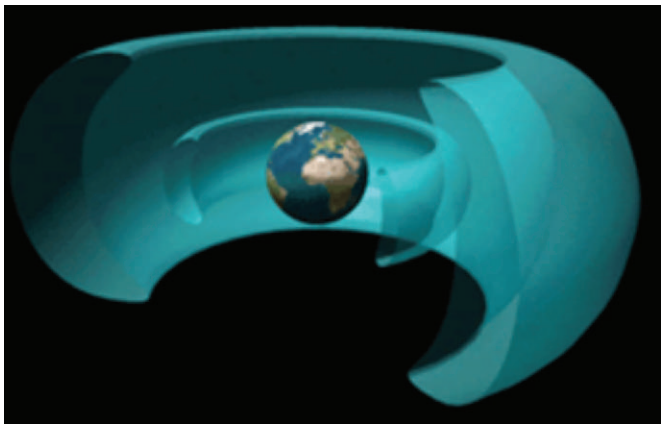

Figure 97-11. Artist's rendition of the Van Allen Belts. (From What Is Space Radiation? at http://srag-nt.jsc.nasa.gov/AboutSRAG/What/What.htm.)

Solar particle events emit very high doses of radiation over a short period of time. They mostly consist of protons and are strongly associated with solar flares. Although solar flares are known to vary in 11-year cycles, there are currently no reliable models that can accurately predict the onset and intensity of SPEs. Crewmembers on EVAs or inadequately shielded spacecraft remain vulnerable to SPEs, and could exceed career limits for radiation exposure in as little as 12 hours.

Radiation exposure in LEO comes primarily from the Van Allen belts and from GCRs. Crewmember exposure is measured in Sieverts (Sv), which is a product of radiation energy absorbed (Gray [Gy]) and a dose equivalent quality factor taking into account different types of radiation (alpha, beta, gamma rays). The amount of exposure varies with time, altitude, orbital inclination, and radiation shielding. A 1-week Shuttle mission has had exposures ranging from 0.005 to 0.05 Sv, and a 1-year Mir mission resulted in 0.584 Sv dose-equivalent exposure. NASA guidelines have established that the radiation dose limit for blood-forming organs (the most sensitive system to radiation effects) is 0.5 Sv/year. This is estimated to confer a 3% excess cancer mortality over a 10-year career.[97]

A round-trip exploration-class mission to Mars has been estimated to last 2.5 years, with approximately 1 year interplanetary travel and concomitant exposure to deep space radiation. This remains one of the key obstacles for the success of a Mars mission and is a current area of active research. Previous concern regarding astronaut radiation exposure has focused on cancer risk, cataract development, and reproductive health. One area that may affect performance during the mission is radiation damage to the central nervous system, specifically early

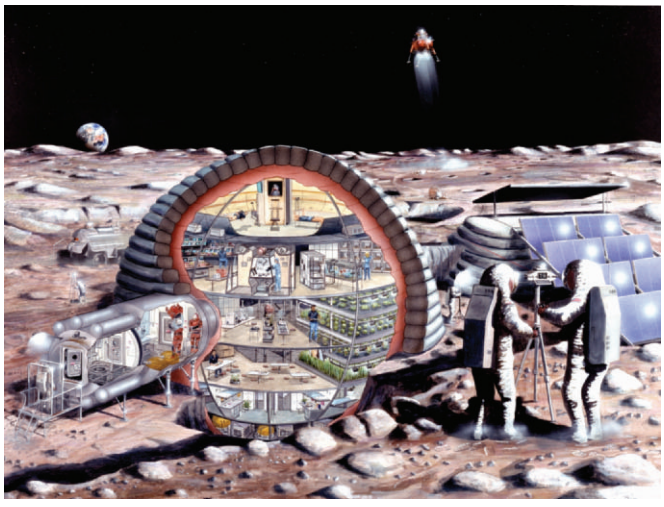

Figure 97-12. As in this artist's rendition, some plans for a lunar base envision subterranean habitats so as to use lunar soil for shielding. (From NASA Human Space Flight Gallery at http://spaceflight.nasa.gov/gallery.)

Figure 97-13. Astronaut Pam Melroy helps Dave Wolf to don his EMU. (From NASA Human Space Flight Gallery at http://spaceflight.nasa.gov/gallery/images/shuttle/sts-112/lores/sts112-332-002.jpg.)

onset of neurodegenerative disorders or acute effects that affect crew performance.

Countermeasures for prolonged radiation exposures are mostly unproven. Genetic screening has been proposed as a strategy for identifying crewmembers with vulnerability for oncogenic mutations.[100] Other proposals include prophylactically dosing the crew with radioprotective chemotherapy. Side effect profiles have limited this as a viable option but may hold promise for the future. Less toxic pharmaceutical interventions may incorporate antioxidants (vitamins A, C, E; trace amounts of iron, zinc), or tumor growth disruptors, such as dimethysulfoxide (DMSO) or protease inhibitors, as a way to minimize cell damage.[33] Certainly for exploration-class missions, preflight cryogenic gamete preservation would be recommended for all crewmembers of child-bearing age, and along the same lines, bone marrow preservation is recommended in case an autologous transplant should be necessary upon return.

Shielding is an obvious solution to ambient radiation, but it eventually becomes impractical from an engineering standpoint. Although the idea of a "storm shelter" has been proposed as a location on the vehicle where crew could weather SPEs, which are relatively short lasting, GCR is constant exposure, and the entire vessel would need to be shielded. Radiation exposure has an exponentially inverse relationship with protective shielding, meaning multiple layers of shielding confer small decreases in radiation exposure. Several meters of shielding would be required to eliminate GCR exposure—an impractical engineering demand given current technology. One creative solution for a Mars-bound vessel suggests using superconductors to create a synthetic magnetic field.[47] Until a combination of solutions emerge to mitigate the effects of GCR, manned spaceflight will likely be confined to LEO and the relatively short trip to the moon.

Even though the moon has no atmosphere and is also exposed to GCR, lunar soil has long been studied as an abundant source of radiation shielding for prolonged stays (Fig. 97-12). Although Mars has an atmosphere that consists mainly of CO_2, it is less than 1% as thick as Earth's, and Mars has no global magnetic field. The Mars Radiation Environment Experiment (MARIE), part of the Mars Odyssey Orbiter, entered Martian orbit for the purpose of determining GCR effects on Mars.[83] Previous estimates of dose-equivalent GCR exposure on the Martian surface are 0.12 Sv/year (to blood-forming organs).[127]

Decompression Hazards

A minimum barometric pressure of 0.9 psi (6.2 kPa) (the saturated vapor pressure of water at body temperature) is required to prevent ebullism, the spontaneous boiling of body tissues. Armstrong's line, or an altitude of 63,000 ft (19,202 m), is where this barometric pressure is reached in Earth's atmosphere.

In addition to the 0.9 psi (6.2 kPa) of water pressure present in the alveoli, there is also 0.7 psi (4.8 kPa) of carbon dioxide, leading to a total alveolar gas pressure of 1.68 psi (11.6 kPa). For gas exchange to occur, ambient pressure (i.e., pressure within the alveoli) must be greater than 1.68 psi (11.6 kPa).

Space suits, such as the American EMU (Extravehicular Mobility Unit) (Fig. 97-13) or the Russian Orlan (Fig. 97-14), contain only a single gas, oxygen, and can thus be pressurized to hypobaric levels (4.3 psi [29.6 kPa] for the EMU, 5.7 psi [39.3 kPa] for the Orlan) while still maintaining alveolar pressures of oxygen that do not induce hypoxia.[77] When the atmosphere contains more than one gas, however, ambient pressure must rise further in order to ensure that the partial pressure of oxygen in the alveoli is high enough to support normal physiologic processes.

Both the U.S. Space Shuttle Program and International Space Station make use of a 14.7 psi (101.4 kPa) normal cabin barometric pressure, as this requires the least adaptation by crewmembers and avoids confounding of scientific experiments that might be affected by atmospheric pressures different from terrestrial controls.

The Apollo program, by contrast, used 100% oxygen at 5 psi (34.5 kPa). (This caused difficulties during the Apollo-Soyuz program, because the Soyuz, then as now, used a nitrogen/oxygen environment at 14.7 psi [101.4 kPa].) While the single

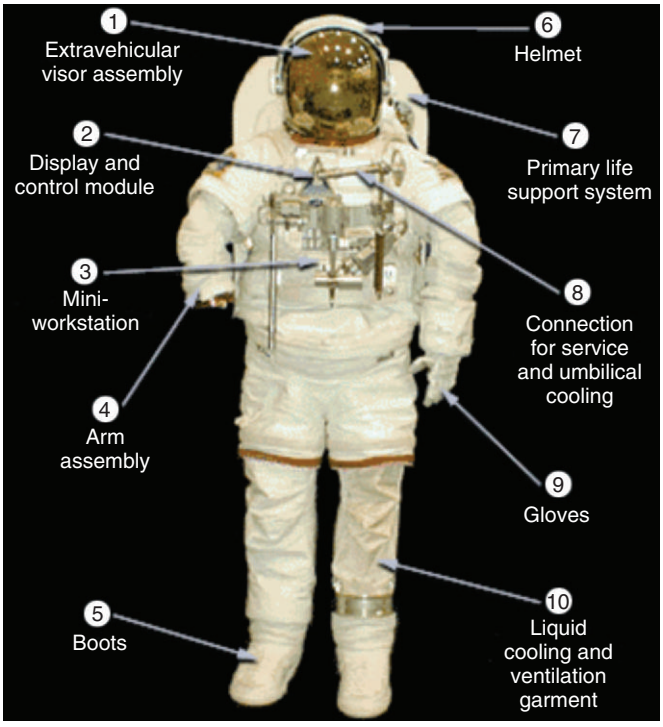

Figure 97-14. A Russian Orlan spacesuit. (From www.voentour.com/space/space_suit.shtml.)

Figure 97-15. Astronauts (*left to right*) Gus Grissom, Ed White, and Roger Chaffee, the crew of *Apollo 1*, posed for this picture a mere 10 days before their deaths in a fire on the launch pad. (From NASA Image Exchange [NIX] at http://grin.hq.nasa.gov/IMAGES/SMALL/GPN-2000-000618.jpg.)

Figure 97-16. Astronauts Piers Sellers *(left)* and Dave Wolf are in the midst of a prebreathe exercise in preparation for an EVA. They are wearing the liquid cooling garment (LCG). (From NASA Human Space Flight Gallery at http://spaceflight.nasa.gov/gallery/images/shuttle/sts-112/lores/sts112-309-033.jpg.)

gas environment simplified the atmospheric control system for these early space vehicles, there were concerns about the effects of prolonged hypobaric pressures on the crewmembers, as well as increased danger of fire in oxygen-enriched environments, particularly if the barometric pressure were to be raised above the hypobaric level. This was the case in the Apollo 1 disaster, where a 100% oxygen cabin environment was temporarily raised to 16 psi (110.3 kPa) for a test on the launch pad. In the resulting fire, Ed White, Virgil "Gus" Grissom, and Roger Chaffee died (Fig. 97-15).

The two main types of decompression in spaceflight are unexpected decompression, as with the June 1997 collision that ruptured one of the Mir space station's living modules, and scheduled decompression, as when spacewalks occur.

Scheduled decompressions occur routinely, often several times in the course of a mission, when space travelers move from the sea level environment of Shuttle or Station to the lower pressure of a spacesuit. The lower the atmospheric pressure within the suit, the more easily the astronaut or cosmonaut can move around and manipulate the suit's gloves. However, because all of the currently used spacecraft (Shuttle, Soyuz, Station) use a nominal dual gas atmosphere at sea level pressure, crewmembers face a risk of decompression sickness (DCS) whenever they move from the cabin environment to the spacesuit.

To prevent DCS during EVAs, preventive measures are routinely undertaken to maximize nitrogen washout in the EVA crewmembers. Among the procedures used are prebreathing 100% oxygen (Fig. 97-16), exercising during the oxygen prebreathe session, and (for the Shuttle only, whose smaller habitable volume makes this a feasible option) temporarily decreasing the cabin atmospheric pressure to 10.2 psi (70.3 kPa), in order to decrease the gradient between the cabin and spacesuit environments. During this decompression, if the oxygen concentration were held constant at 21%, the alveolar oxygen tension would be significantly reduced, so it is necessary to raise the concentration of oxygen in the cabin during this period to 23.8%.

In the event of a suspected or confirmed DCS event, limited hyperbaric treatment can be provided through overpressurization of the EMU spacesuit. When performed in a cabin pressurized to sea level, the patient will experience a 100% oxygen

Figure 97-17. Using power tools and maneuvering massive objects are routine activities during an EVA. (From NASA Human Space Flight Gallery at http://spaceflight.nasa.gov/gallery.)

environment at approximately 22 psi (151.7 kPa). For LEO missions, rapid medical evacuation to a terrestrial medical facility would then occur. Exploration-class missions, by contrast, are unlikely to be able to return stricken space travelers to Earth, and therefore a hyperbaric chamber functionally equivalent to terrestrial models may be included in medical systems for those vehicles.

Spacewalk Concerns

Extravehicular activities are an integral part of manned spaceflight. During an EVA, humans enter arguably the most hostile environment imaginable. The absence of air, heat, pressure, and gravity forces space travelers to bring along not only their own food and water but also a protective atmosphere, similar to that of Earth. Environmental systems control atmospheric composition, pressure, humidity, and temperature. Design of these systems is remarkably complex and consumes a significant amount of budget and resources, yet the life support systems are indispensable aspects of the manned space program. Spacesuits are incredibly sophisticated pieces of equipment. They consist of liquid cooling garments (LCGs) for thermal homeostasis, biomonitoring devices for medical support, and a pressurized environment, yet they allow for user dexterity in performing complex and demanding tasks (Fig. 97-17).[87] The dangers of EVAs are numerous. Manipulating extremely heavy objects—without weight, but with enormous mass—risks musculoskeletal and crush injuries. Decompression sickness (DCS) remains an ever-present concern. EVAs require extensive training, and crew repeatedly practice techniques in the terrestrial setting of large underwater tanks.

The danger of DCS illustrates the criticality of the life support systems in both the orbiter/ISS and EMUs.

Case Study

The effect of a loss of the cabin environment was starkly illustrated by the 1971 deaths of the entire *Soyuz 11* crew (cosmonauts Georgi Dobrovolksy, Vladislav Volkov, and Viktor Patsayev), when a pressure equalization valve opened too early in the reentry phase and vented the cabin to space at an altitude of over 100 miles. In order to prevent future tragedies of this nature, it is important not only to understand the crew's physical needs, but also the intricacies of the life support systems that supply them. See Steven Casey's *Set Phasers on Stun: And Other True Tales of Design, Technology, and Human Error*[28] for a more detailed description of this event.

Prolonged missions to Mars or the Moon will require new and innovative approaches to EVAs commensurate with longer missions, repeated field work, and remoteness from any support. Because such a mission would emphasize planetary science and exploration, EVAs would undoubtedly be frequent and likely to produce greater wear and tear on equipment.

Potential EVA tasks consist of constructing and maintaining the habitat, as well as conducting a scientific exploration program encompassing geologic field work, sample collection, and deployment, operation, and maintenance of instruments. Any EVA system must perform these tasks with the critical functional elements of a pressure shell, atmospheric and thermal control, communications, monitoring and display, and nourishment and hygiene support systems.[83,142] Balancing the desire for high mobility and dexterity against accumulated risk to the explorer is a major design driver on a Mars EVA system. Light weight, ease of dexterity, and durability will be essential to the design, as will a reliable airlock system.

Because of the unique challenges for a Mars EVA program (frequency and duration of sorties, quick turn-around times, necessary dexterity and mobility, dust and abrasion), a fresh strategy for interfacing the crew with the surface is required.[87]

Launch/Landing Stresses and Surface Concerns

Historically, launch and landing are the times of a mission that expose a crew to the most risk. Both the *Challenger* and *Columbia* disasters, as well as the *Soyuz-1* (parachute failure) and *Soyuz-11* (cabin decompression) tragedies were associated with leaving or re-entering the atmosphere.[101,124] These are also the times when crew physiology is most labile, especially with the return to a gravitational field and other landing stresses, including vibration and G-forces. A potentially osteopenic crew dealing with the forces of a Martian landing may have an increased risk for bony injuries. This possibility will have a strong effect on emergency protocols such as procedural sedation and fracture detection and management.

Once safely landed, surface exploration occurs in nearly as hostile an environment as deep space. The Moon has no atmosphere, magnetic field, or geologic activity, rendering the satellite a "dead" planet. High-energy deep space radiation reaches the lunar surface unimpeded, posing a risk to long-term inhabitants. The surface composition itself poses no toxicity to humans, although the fine particulate characteristics of the silicate soil may cause problems with allergic reactions or inflammatory lung conditions during prolonged exposures.

Similar concerns exist for exploration on the surface of Mars. With the lack of a magnetosphere and an atmosphere 1% as

dense as Earth's, long-term radiation exposure will be a major health risk. Seasonal changes exist, and weather phenomena such as dust storms may be strong enough to cover the planet for weeks. Although slightly more than half the diameter of earth, Mars has incredible geographic diversity.[87] Olympus Mons is the largest volcano in the solar system and is roughly three times the height of Mt. Everest. Likewise, the Valles Marineris is the largest canyon in the solar system. It is over 4 miles (about 6 km) deep, and its length would stretch from California to Virginia. Exploration of such features will provide challenges far beyond a stroll on a lunar plain and will require the incorporation of lessons from terrestrial wilderness medicine.

Stressful Environment: Psychological and Behavioral Issues

NASA has specifically identified 14 individual behavioral issues that are thought to have a direct bearing on productivity and habitability during routine operations onboard a permanently manned space station[62]:

- Sleep
- Clothing
- Exercise
- Medical support
- Personal hygiene
- Food preparation
- Group interaction
- Habitat aesthetics
- Outside communications
- Recreational opportunities
- Privacy and personal space
- Water disposal and management
- On-board training, simulation, and task preparation
- Behavioral and psychological requirements associated with microgravity

External sources of stress during space missions include limited ability to contact family and friends, real or imagined crises at home, supply shortages, a sense of "interference" from ground personnel, and feelings of rejection resulting from delays in relief. Internal sources of stress may include lack of privacy, forced social interaction, boredom, sexual and emotional deprivation, and perceived absence of status and role definition.

Studies show that isolated and confined populations frequently demonstrate mild psychiatric symptoms, including depression, anxiety, increased defensiveness, and belligerence.[30] It has also been suggested that prolonged isolation and confinement harms group dynamics, with social irritability reported among polar expeditioners, submariners, and space-simulator subjects.[30]

Interpersonal conflict is a real and concerning threat to crew health and mission performance.[61] Cramped, confined spaces, cultural differences (often including language differences), and busy schedules replete with complex physical and mental tasks can often exacerbate stressed relationships. Ground support currently plays a large role in monitoring and providing intervention for crewmember strife, but this strategy will need modification for a mission to Mars, where transmission time delays can be up to 40 minutes round-trip.

This delay will not only diminish reliance on ground support for problem solving, but may increase tensions between the crew and mission control. History is replete with strife between those on the "front" and the decision makers in the rear. During World War II, submariners on 2- to 3-month patrols complained about the "unrealistic" orders sent from rear echelon officers thousands of miles away. There have already been reports of animosity toward mission control from astronauts on long-duration missions.

The main psychological effects on space travelers can be summarized as[68]:

1. Impaired intellectual functioning: Isolation and confinement lead to decreases in alertness, concentration, and memory. It is worth noting that there are numerous anecdotal reports from astronauts that it is harder to think clearly in space.
2. Motivational decline: Prolonged isolation and confinement can result in loss of motivation. Polar expeditions have shown general decline in work satisfaction and group accomplishment, with fatigue, inertia, and general apathy occurring as the mission wears on.[119]
3. Somatic complaints: Psychosomatic symptoms, including insomnia, headaches, sleep disturbance, digestive problems, toothache, and fears of acute surgical emergencies, have occurred both in space and analog environments. Anecdotal reports from Russian long-duration space crews have shown concern (bordering on obsession) with physical well-being at various phases of a mission.
4. Morale: Substantial evidence suggests that regardless of mission length, morale reaches its nadir between the one-half to two-thirds mark.

The consequences of psychological dysfunction have resulted in early abandonment of at least one Salyut mission, when cosmonaut Vladimir Vasyutin began showing signs of severe stress and depression after 2 months in space. He was hospitalized on his return. In another example, chronic high stress among the crew may have contributed to the 1997 collision between Mir and a resupply vehicle during a docking maneuver.

In-flight communications remain key for assessment and improvement of crew behavioral health. Crew access to medical and emotional support from family members and mental health professionals is essential for mission morale. Favorite meals and family "care packages" are often dispatched on supply vehicle flights.

Limited Resources

The risk of scarce resources can trigger anxiety and panic. If fears become realities, group dynamics can break down. Both spaceflight and terrestrial analogs have witnessed numerous conflicts over favorite foods or when, for example, scarcity of water has limited desired activities (such as laundry or hygiene).

Lack of Privacy

Privacy (or the lack of it) is a feature not only of space exploration, but also of other stressful environments. Shuttle crews are at more of a disadvantage than are ISS crews. The larger crew size and smaller habitable volume force Shuttle crews to live in extremely close quarters (Fig. 97-18). By contrast, the larger ISS, with its 2- or 3-person crew, affords significantly more privacy and personal space. That said, the tiny crew complement ensures that there are few secrets on board.

Privacy may well become an even greater issue in interplanetary travel, where there will be larger crews, but the vehicle may not have the size to allow each crewmember the privacy he or she would like.

Worsening the issue is the fishbowl-like nature of life in space. Not only do members of the ground support team follow the

Figure 97-18. Sleeping astronauts on the crowded Shuttle middeck demonstrate the lack of privacy on board. (From NASA Human Space Flight Gallery at http://spaceflight.nasa.gov/gallery/images/shuttle/sts-112/lores/sts112-345-028.jpg.)

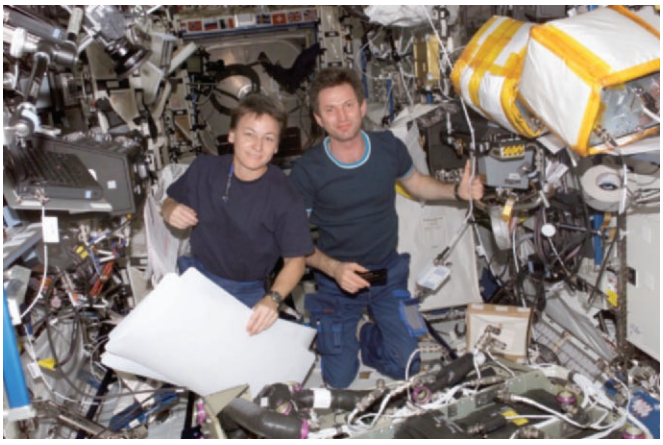

Figure 97-19. The highly mechanized environment of the ISS. (From NASA Human Space Flight Gallery at http://spaceflight.nasa.gov/gallery.)

crew's every action with close attention, but members of the world media do as well.

Isolation from Family and Social Networks

The isolation of space can lead to sleep disturbances, headaches, irritability, anxiety, depression, boredom, restlessness, and anger, all of which can negatively impact motivation and performance. Perceptual sensitivities may become altered with time. This has resulted in several instances of a "them versus us" situation developing, with conflicts between the flight crew and ground control team.

Monotony and Boredom

Monotonous tasks that require little cognitive processing or attention are common in flight, and may cause crewmembers' attention to drift and their performance to become lethargic. This represents a serious threat to the integrity and safety of the crew, due to the nature of system monitoring and interpretation of warning signals.

Measures to counter this situation have proved difficult to develop in both ground and flight operations, although suggestions include the use of intelligent computer-controlled monitoring systems.

Circadian Patterns and Sleep Disturbances

Sleep and circadian rhythm disturbances may also contribute to in-flight disorientation. Orbiting flights experience multiple sunrises throughout the day, creating contradictory light/dark cycle cues that can affect the body's circadian rhythm and interfere with sleep patterns. Sleep hygiene is encouraged through limited exposure to light, pharmacologic aids (45% of all medicines used by Space Shuttle crews are sleep aids[116]), and guidelines that limit sleep-shifting schedule changes.

Through the Apollo 9 mission, it was common practice to employ staggered sleep schedules for crewmembers. Even during later flights when crewmembers slept simultaneously, hectic schedules frequently required displacement of the sleep phase by several hours, leading to complaints of fatigue and sleep problems. The hazard of having all crewmembers sleeping simultaneously lies in the inability of most subjects to function optimally immediately upon awakening. During some of the past space missions, events have occurred that required the crew to arise from sleep and respond immediately.

Workload Issues

Managing workload is a challenging task; activity levels have to be delicately balanced between too much (stress of overwork) and too little (frustration from boredom, time to brood). In this respect, the differences between Russian and U.S./European procedures are marked. Shuttle activity is planned by ground personnel in great detail, to maximize efficient use of time on orbit. Russian practice involves more flexible schedules, with greater crew discretion. These differences in part reflect cultural differences and the longer-duration flight times of most Russian missions.

The Russian Space Agency (RSA) operated Mir on a 5-day-per-week/8-hour-per-day schedule, with planned time off. Failure to do this in Skylab resulted in near-revolt when after 3 weeks the crew refused to work any further without a day off. With Shuttle missions, NASA generally tries to pack in the maximum amount of activity through a tightly planned schedule. However, anecdotal reports show that time overruns on the Shuttle are common, with crews working an extra 4 hours per day just to keep up. Although crewmembers on ISS missions have been given greater flexibility in determining their own work schedules, both American and Russian space crews continue to ask for additional options in their work and leisure schedules.

Machine-Dominated Environment

The study of human-machine systems, where humans control their environment through technologic devices, is directly relevant to space missions. During emergencies on the ISS, for example, crewmembers base their actions on information provided by the caution and warning system. They also control aspects of the vehicle operations remotely via computer interfaces. When using the ISS manipulator arm, for example, the crewmember relies entirely on a computer display. The design of such systems is a key component for future vehicles, where the goal must be to maximize automation while maintaining the crewmember as the central figure in the decision loop (Fig. 97-19).

Figure 97-20. Astronaut John Glenn on his 1998 mission at age 77. (From NASA Human Space Flight Gallery at http://spaceflight.nasa.gov/gallery/images/shuttle/sts-95/html/s95e5055.html.)

SOLUTIONS TO THE CHALLENGES OF PROVIDING MEDICAL CARE IN THE AEROSPACE ENVIRONMENT

Starting Out with "The Right Stuff": Planning the Expedition

Selecting the Best Possible Crew

The initial medical qualification standards for astronauts are significantly higher than the recurrent qualification criteria, as the latter reflect the substantial training investment made in the astronauts over the intervening time frame.[88] A 42-year-old active astronaut with medical problems may thus be retained, whereas the same person as an astronaut applicant would be rejected.

It is important to understand the value placed on training and experience when reviewing the medical standards for space flight, as it reflects the stress placed on mission success. If the value added to the mission by a particular crewmember outweighs the medical risk (to both crewmember and mission) associated with his or her health, then he or she is likely to fly (Fig. 97-20).

Selection criteria are not always easily developed. For example, current society rightly considers it abhorrent to bar people from the astronaut corps because they belong to a certain racial, ethnic, or religious group, and there are laws to ensure that this does not occur. Gender, however, remains a somewhat thorny issue, as attested by the relatively low number of women astronauts. In addition, although the professional astronaut corps continues to bar groups based on certain medical criteria, standards appropriate for space tourists remain much more debatable.

Review and revision of medical standards are supposed to occur regularly, usually on a 5-year cycle for major changes (lesser changes every 2 to 3 years), but operational demands have been known to delay this review.[88,125] Since the days of the Apollo program, standards have been significantly relaxed, and more modern technology, such as endoscopy, ultrasound, magnetic resonance imaging (MRI), and other imaging modalities, are used extensively in evaluating crew health.

The criteria by which space travelers (and potential space travelers) are currently scrutinized differ depending on their mission role. Pilots and commanders are screened by more stringent criteria than are mission or payload specialists.

The first space travelers were selected from terrestrial pilots, whose physical condition (health, reflexes, strength) was considered most important. Subsequently, as the purpose of the space program moved from military to scientific objectives, the emphasis in crew selection has shifted to favor academic qualifications more strongly.

Each year, approximately 25% of finalists are disqualified on medical grounds. By far the most common reason for medical disqualification of astronaut candidates is inadequate distance vision. Applicants who pass the initial medical screening and are subsequently selected by the NASA administrator to be astronauts undergo annual medical examinations during their career as astronauts.

Several authors have suggested that future long-duration missions are more likely to be compromised by psychological reasons than physical ones.[68,119] It is therefore very important to ensure crewmembers are not only physically healthy, but also mentally and emotionally so. In addition, the blend of crewmembers should facilitate interpersonal relationships.

Current NASA selection procedures include standardized psychological tests and a psychiatric interview designed to identify any significant psychiatric disorder. NASA has thus chosen a "select-out" policy that eliminates individuals thought to present potential risk. After initial astronaut selection, little emphasis is placed on selecting specific crews based on the individuals' psychological aspects, interpersonal dynamics, or performance predictions. By contrast, since the late 1950s, the U.S. Navy has "screened in" participants for Antarctic missions. Using this paradigm, the Navy deliberately selects crewmembers based on their ability to work well with the other team members. It is not enough simply to be psychologically sound; good interpersonal and group dynamic skills are required as well.

A survey of NASA, CSA (Canadian Space Agency), ESA, and JAXA personnel identified 14 key multicultural factors[62,120]:

- Language
- Nonverbal communication styles
- Task- and relationship-oriented behavior
- Patience and tolerance
- Assertiveness
- Decision-making process
- Interpersonal interests
- Respect for other cultures
- Hygiene and cleanliness
- Gender roles and stereotypes
- Conflict management and resolution

- Trust
- Scheduling
- Sense of humor

Current crew rotations on the ISS involve Russian and U.S. crewmembers for long-stay missions, with shorter visits from ESA, CSA, and JAXA astronauts. The official language of the ISS is English, which is a second language for many of its crewmembers. This could itself cause operational difficulties, because during emergencies, crew-related errors often result from communication breakdown, not technical incompetence. Perhaps in recognition of this, the U.S. crew assigned to the ISS have all learned Russian, though fluency has varied.

Cultural components also influence how people display initiative and wield authority. In the aviation world, numerous accidents have resulted from failure of subordinates to question their leader or to assume command when circumstances dictated that course of action.

On the Apollo-Soyuz mission in 1975, a near-fatal accident occurred during reentry because all three crewmembers failed to set several critical switches. Two of the astronauts were long-time veterans, while the third, on his first flight, was an original Mercury astronaut and later Director of Flight Crew Operations. It is possible that confusion over roles, status, leadership, and Deke Slayton's position in the Astronaut Office contributed to a communication breakdown.

Longer missions will mean increasing leisure time and more unstructured activity, which may affect the cohesiveness of a crew, resulting in the formation of subgroups with "scapegoating." Lieutenant General Beragovoi, who headed cosmonaut training in the 1980s, reported that at about 30 days into a mission, hostility became evident within a space station crew, although the feelings were displaced to the earthbound ground crew.[29,54] However, prolonged Soviet Salyut/Mir missions of the 1980s and more recent ISS experience have shown that maintenance of good communications and psychosocial support are effective in maintaining high work capacity and a happy environment among crews.

Undersea research confirms that homogeneous crews that share values and interests behave more cooperatively than do heterogeneous crews; this has clear implications for the intended multinational crews of the ISS and exploration missions. Studies of the Navy Tektite undersea crews clearly showed that performance and motivation leading to greater group cohesiveness came from the sense of adventure, the selective nature of the crewmembers, and mission objective success.

As yet we cannot define the "right stuff" for space crews, although ironically, those who are fearless, technical, egotistical and individualistic may more appropriately be considered the "wrong stuff," at least from a team-based perspective. In past U.S. programs, there has been an emphasis on military pilots (particularly fighter pilots, many of whom are most experienced in single-seat aircraft), familiar with a recognized command structure and frequent contingency drills or simulations ("sims"). However, the ISS has a multicultural, multiskilled, mixed gender, and mixed civilian/military crew, which makes training and assessment of their actions in the event of a significant emergency more problematic.

NASA training is targeted at an increasingly broad audience: crewmembers, managers, mission control, and families. A significant component of maintaining performance in the space environment comes from planning and preparation stages of the mission, addressing such issues as:

Figure 97-21. Susan Helms, Yuri Usachev, and Jim Voss, the *ISS Expedition Two* crew, participate in preflight Soyuz winter survival training. (From http://spaceflight.nasa.gov/gallery/images/station/crew-2/lores/s98_04119.jpg.)

- Psychological/psychiatric prescreening
- Group training for crews prior to flight
- Preflight counseling for crewmembers and families
- Psychosocial support in-flight to crews and families
- In-flight stress management techniques
- 24-hour on-call psychiatric support

Preflight/Mission Planning

After the rigorous selection process, it may be several years before an astronaut actually performs a mission in space. Crews are encouraged to maintain physical fitness throughout preflight training and are subjected to routine wellness screening. More formal annual exams are conducted not only to ensure flight readiness, but also for biomedical longitudinal data collection to determine any long-term health effects of spaceflight. The data thus far have shown one concerning trend: astronauts have a higher number of (non-occupational) trauma-related deaths than do matched controls. Of course, this may be due to an adrenaline-loving lifestyle that led them to space in the first place.

Communication and team building exercises are also staples of preflight training, with crewmembers trained in conflict resolution and stress management. Classroom activities are interspersed with mission simulation in the mock-ups, in the Neutral Buoyancy Lab, and during KC-135A parabolic flights. Land and water survival training courses are also part of the training regimen (Fig. 97-21). Medical training for all crewmembers consists of the general principles of space medicine and physiology, as well as first aid and cardiopulmonary resuscitation (CPR) courses.

CMO and Buddy Care Training

During a mission, there is often no physician available, and the astronauts must rely on a fellow crewmember to be their primary caregiver. This system of "buddy care" training is formalized in the role of the Crew Medical Officer (CMO). ISS CMOs receive approximately 40 hours of training on topics such as diagnostics and therapeutics, phlebotomy, suturing, splinting, and endotracheal intubation (Fig. 97-22). Part of their

Figure 97-22. Astronaut Sergei Krikalev receives training in suturing from Tyler Carruth, PA, as part of his preflight CMO training. (From http://spaceflight.nasa.gov/gallery/images/station/crew-11/lores/jsc2004e26778.jpg.)

role is to provide information to the ground-based Flight Surgeon in Mission Control, with whom lies the ultimate responsibility for the health of the crew.

Creating a Habitable Habitat

The nominal atmospheres of the Space Shuttle, the Soyuz, and the ISS are dual-gas oxygen-nitrogen (21% oxygen with the balance nitrogen) maintained at sea level pressures. However, pure oxygen is breathed during extravehicular activities or during emergencies when the crewmembers are directed to don portable breathing apparatuses (PBAs). PBAs are used to protect the wearer from atmospheric contamination (as in cases of fire) or to maximize the alveolar partial pressure of oxygen and delay development of hypoxia (during depressurization).

The ISS ventilator (AutoVent 2000) also uses 100% oxygen exclusively.[66] As a result, medically compromised patients aboard the ISS can receive 100% oxygen or room air, but there is currently no means of providing supplemental oxygen between 21% and 100%. This may create difficulties in seriously ill or injured crewmembers who require mechanical ventilation or supplemental oxygen for prolonged periods, as studies have shown that deleterious effects from breathing 100% oxygen at sea level pressures occur in as little as a day.

A single-gas environment has several benefits. It permits a lower habitable atmospheric pressure, thus decreasing the risk of decompression sickness during EVA; it minimizes the required strength of the spacecraft hull since it will not need to withstand a higher internal pressure; and it requires smaller stores of gas. However, it is a highly artificial environment in which certain experiments cannot be performed at all, while it would be impossible to generalize from other experiments because they were not performed in the standard sea-level, dual-gas atmosphere. In addition, the higher oxygen concentration (albeit at a lower pressure) could pose a higher fire risk.

Air supplies for spacecraft repressurization and crew respiration are stored in high-pressure tanks as liquids in cryogenic storage or in other forms.[24,79] Nitrogen, for example, can be stored as hydrazine (N_2H_2), which is also a spacecraft propellant. Four high-pressure gas tanks (two each for nitrogen and oxygen) are located on the exterior of the joint airlock on the ISS, and can be either refilled or replaced by the Shuttle (Fig. 97-23).

Figure 97-23. An overhead crane at Kennedy Space Center lowers a gas tank onto the Spacelab Logistics Double Pallet while workers help guide it. Four of these storage tanks, two for gaseous oxygen and two for gaseous nitrogen, comprise the high pressure gas assembly that will be attached to the Joint Airlock Module during two spacewalks. (From NASA Image Exchange [NIX] at http://nix.larc.nasa.gov/info;jsessionid=1n21fjejgtner?id=KSC-01PP-0952&orgid=5.)

Ambient cabin pressure is maintained, and atmospheric leakage countered, by periodic injection of gas. Both ground control and on-board crew can control these valves and thus regulate ambient pressure within the spacecraft.

Hypoxia and hypercarbia are very real dangers during spaceflight, not only from malfunctions in the life support system, but also because in microgravity, gases do not diffuse away from the face as they do terrestrially. Carbon dioxide is heavier than breathing air, so on Earth it falls away from the face once it is exhaled. In addition, breezes, local airflows, and weather- or gravity-related convective forces tend to remove carbon dioxide from around the nose and mouth. These forces are absent in microgravity, and, in the absence of adequate ventilation systems to move the air from in front of the face, pockets of carbon dioxide can accumulate, leading to hypercarbia. Similarly, when the crew must operate in small, contained spaces where the ventilation system is less effective, such as behind a rack, inside an unventilated sleep station, or when numerous items are out in the cabin, extra caution must be taken to prevent buildup of carbon dioxide.

During the 1985 recovery mission of the Salyut 7 space station, cosmonauts Dzhanibekov and Savinikh had to work in a module that lacked power and was thus both cold and unventilated. They reported headaches, lethargy, and sluggishness associated with the hypercarbia produced by their own exhala-

tions. Providing active ventilation as soon as possible through portable fans and resumption of powered systems mitigated this effect.

Forced ventilation is thus a vital component of any atmospheric system for space travel. Without adequate blending of the cabin atmosphere, heterogeneous microenvironments can occur, containing undesirable levels of carbon dioxide, particulates, water, or heat. Air movement also enables surveillance of the environment for toxins (such as smoke), permits extraction of undesirable components (such as carbon dioxide and trace contaminants), and allows control of both heat and moisture content of the atmosphere.

There is a great deal of ongoing research into onboard oxygen generating systems (OBOGSs), including molecular sieves, electrolysis, various forms of air separation, membrane technologies, carbon dioxide reduction (e.g., a Sabatier reactor), chemical absorption, and biological systems. However, only a few of these have actually been used in space up to this point.

Molecular sieves, which were first used during the Skylab missions, are also used in some military aircraft. Ambient air is forced over a zeolite crystal molecular sieve that separates the oxygen; the gas is then further concentrated and released back into the cabin atmosphere as needed.

Russian life support systems since the Mir space station have generated onboard oxygen through an Elektron oxygen system. In this system, stored water or even urine is used to produce oxygen via electrolysis ($2H_2O$ + electrical current $\rightarrow 2H_2 + O_2$). On the ISS, electrical current is provided from the station's solar panels. The hydrogen is currently vented into space, though eventually it may be combined with carbon dioxide to create additional oxygen or water in the (planned) Russian Sabatier reactor. In this reactor, hydrogen from the Elektron oxygen system is mixed with carbon dioxide from the carbon dioxide scrubbing system at relatively high temperatures (900° F to 1200° F [482° to 649° C]) to create first water, then oxygen.

Another OBOGS used since Mir is the solid fuel oxygen generator (SFOG) system, which employs a chemical reaction (heating lithium perchlorate) to create oxygen, the same technology used in the emergency oxygen mask systems of commercial aircraft. Each 2.2-kg perchlorate "candle" produces 600 L of oxygen, a supply sufficient for one person for one day.

For long-duration exploration missions, however, biological systems are likely to be more heavily employed. In 1995, a closed bioregenerative system was successfully tested, using 11 square meters of wheat in the chamber to provide oxygen for and consume carbon dioxide from a single person for 15 days. However, current plant-based systems are larger, more labor-intensive, and (as any gardener knows) less reliable than the chemical-mechanical systems. Given the criticality of the atmospheric system, the current level of uncertainty associated with biological systems is unacceptable, and additional research is needed.

Spacecraft life support systems must monitor several atmospheric parameters, including ambient pressure, partial pressure of each gas (including carbon dioxide), ventilation, temperature, and humidity.[113] Whereas the oxygen monitoring system maintains the partial pressure of oxygen relatively close to that experienced on earth, spacecraft carbon dioxide monitoring systems deal with levels significantly higher than what is seen terrestrially; 7.6 mm Hg of carbon dioxide is the upper limit of normal for both ISS and Shuttle life support (33 times Earth-normal). Current technology does not offer a method to reduce these levels further, and to date, no evidence exists that these elevated levels have long- or short-term effects on space traveler health.

Figure 97-24. Astronaut Jack Lousma inserts lithium hydroxide (LiOH) canisters into the air revitalization system servicing panel on the Shuttle middeck. (From NASA Image Exchange [NIX] at http://nix.larc.nasa.gov/info;jsessionid=1akxvdx2wwap4?id=S82-28921&orgid=8.)

Contaminants fall under two general categories: particulate and gaseous. The latter includes both biological (such as those created during food preparation or produced by the crew) and chemical (as can be seen from off-gassing of cabin materials, cleaning supplies, or certain scientific experiments). Prevention is preferred to removal, however, and spacecraft designers create their plans accordingly. For example, judicious selection of spacecraft cabin materials can minimize sources of off-gassing toxins. Carbon dioxide is by far the most important contaminant to be removed from the environment.

However, not all contaminants can be avoided. Submarine and chamber studies showed as early as the 1970s that in a contained environment, even trace contaminants can build up to hazardous levels. Prior to the Skylab program, U.S. spacecraft had neither monitored nor analyzed trace contaminants (with the exception of carbon monoxide). From Skylab on, however, numerous methods have been used to monitor the cabin environment, including gas chromatography, mass spectrometry, ultraviolet and infrared spectroscopy, and sensors for individual contaminants (e.g., carbon monoxide or hydrogen cyanide). Exploration-class missions must consider additional potential contaminants such as Martian soil or lunar dust.

During emergencies (such as a fire), additional hazards, from cyanide gas to molten metal particles, can be introduced into the atmosphere. Removal of many of these substances can be achieved through use of filters (Fig. 97-24). However, there are some contaminants that current filters cannot remove (e.g., toxic byproducts of the Halon used as a fire suppressant on the Shuttle).

The life support system of a spacecraft must protect its occupants not only from the intense cold outside, but also from buildup of heat within. There are multiple sources of heat within the spacecraft, each of which can affect thermal control: electronics, lighting, solar heating, and metabolic heat (as produced by exercise). Equipment produces high thermal loads,

and, in most cases, hyperthermia is more of a danger to space travelers than is hypothermia. The literature clearly documents decrements in task performance as crew comfort declines. Long-duration Soviet/Russian spaceflights suggest that ideal temperatures for a working environment are 22° to 24° C (72° to 75° F). When temperatures drop below 19° C (66° F), with a relative humidity around 70%, the crews complain of cold. An unusual example of contending with cold occurred on the damaged Apollo 13 spacecraft, which required strict conservation of energy on the return to Earth. The uncomfortable crew were very glad to get home (Fig. 97-25).

Humidity is a key component in physical comfort aboard the spacecraft and is integrally linked with temperature control. Although it is less noticeable at comfortable temperatures, as either extreme is approached, humidity levels become important. To maintain crew comfort, humidity must fall to promote evaporative cooling during increases in temperature, while higher humidity levels reduce evaporation and facilitate heat retention at cooler temperatures. The cabin environment is usually maintained at about 60% relative humidity (corresponding to about 0.2 psi [about 1.4 kPa] of water vapor pressure).

Circulation of cabin air is the mainstay of temperature and humidity control. As air is recirculated, moisture can be removed from it either by a desiccant (as is usually the case in spacesuits) or through condensation of the vapor and removal of water from the gaseous air. This latter method is used more often in spacecraft, since it allows reuse of the water thus produced. In general terms, condensation occurs when cabin air is cooled on heat exchangers, and the resultant water is then removed by separators (either via capillary action or hydrophobic-hydrophilic surfaces). A description of the life support system for the ISS is shown in Figure 97-26.

Odors in the cabin atmosphere are generally removed using charcoal filters. As with other contaminants, prevention is pre-

Figure 97-25. The *Apollo 13* crew, shortly after landing. (From http://www.nasm.si.edu/collections/imagery/apollo/AS13/images/AS13_return.jpg.)

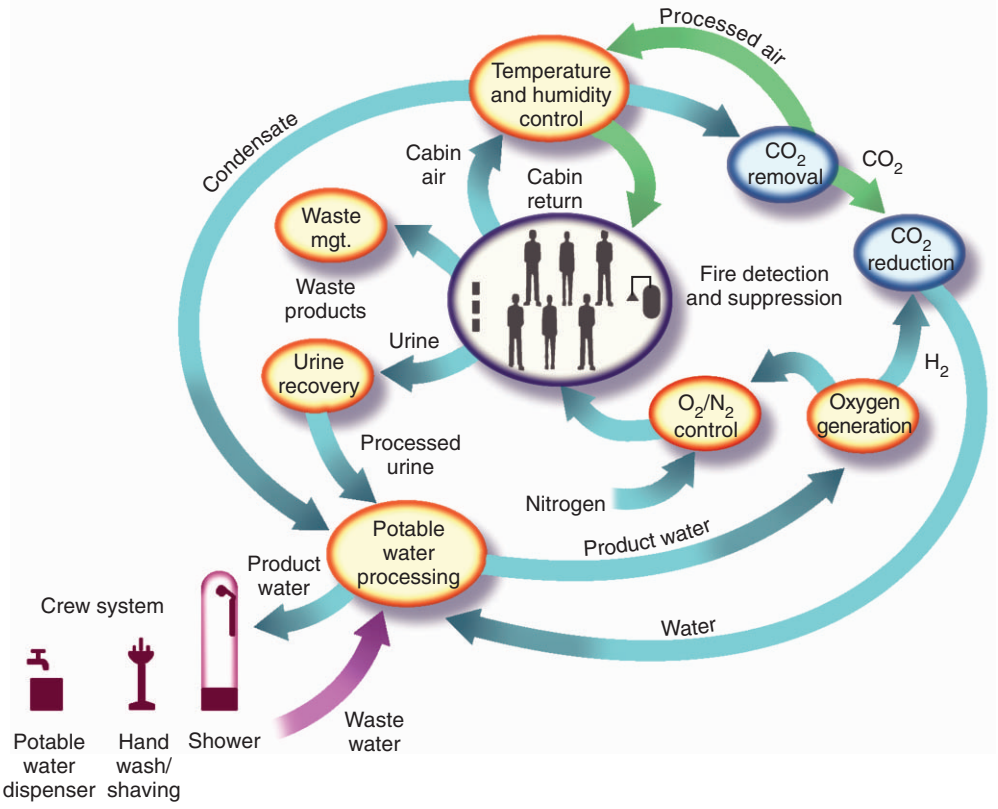

Figure 97-26. Description of the life support system for the ISS. (From http://mix.msfc.nasa.gov/IMAGES/MEDIUM/0102167.jpg.)

ferred to removal, prompting the need for an effective waste control system. As occurs lamentably often in history, previous lessons were forgotten when the ISS was designed and launched; experiences in trash handling gained aboard the Mir and Skylab space stations were not utilized. For example, there was no flight rule to prevent stowage of trash near food. The problem of waste accumulation, handling, and disposal quickly became so acute that NASA established a team tasked solely with developing an ISS Trash Plan.

Generally speaking, waste collection is designed to be as simple as possible for the crew. In some cases, crewmembers merely place the refuse in a container and stow it. In other cases, the waste may be automatically collected and stored, as is the case for the toilet appliance. In all cases, however, limited stowage space remains an issue that drives waste disposal policy. For waste material that the crew should not handle for reasons of health or safety, the appropriate systems usually collect and package the waste automatically. Virtually all forms of waste disposal currently make use of Earth, either by returning trash to the ground for final disposal or by utilizing atmospheric reentry to destroy trash in the Progress rockets. Unfortunately, these options are not ideal for long-duration exploration missions, when Earth will not be available. In these situations, waste will need to be reused or destroyed in more efficient ways, so as not to take up valuable storage space.

Biological solid wastes, such as those from food, generally contain 40% to 90% moisture and soluble organic compounds.[69] As a result, these wastes cannot be stored for extended periods, because they decompose (leading to growth of undesirable anaerobic microorganisms), produce noxious gases (including N_2O, NH_3, H_2S), and create foul odors.

In addition, wastes may be in mixed form (such as semisolid), and they may be handled by more than one system, as when water condensed from ambient air by the atmosphere-management system is sent to the water-processing system for final disposition. Life support designers must also take into account the need to conserve resources wherever possible. Particularly on long-duration and exploration missions, wastes must be recycled or reused, which imposes requirements on how wastes are categorized and handled. For example, liquid waste from food preparation might be able to be reused for hydroponics projects and should therefore be captured and stowed separately from liquids contaminated by toxic chemicals, as might result from certain payload experiments. Composting, the process of accelerating decomposition in an aerobic environment, is also being explored as an option for safely handling biological wastes.

For exploration-class missions, another category of waste must be considered: contaminants from the external environment, such as Moon dust. The habitats used by crew on lunar and Martian missions will need to have systems, such as forced air blowers, sticky strips, or other devices, to remove any such pollutants from an astronaut's suit or tools and to dispose of them in a safe manner.

In addition, there is a desire not to contaminate external environments with terrestrial compounds. For example, studies into the possibility of life (past or present) on Mars could be confounded if terrestrial bacteria were carried from a habitat module into the larger Martian environment. Accordingly, the waste collection and disposal system will likely be used by astronauts as they leave the base, as well as when they reenter it, creating (potentially) twice as much waste material of two very different kinds, one endogenous to the habitat and the other exogenous.

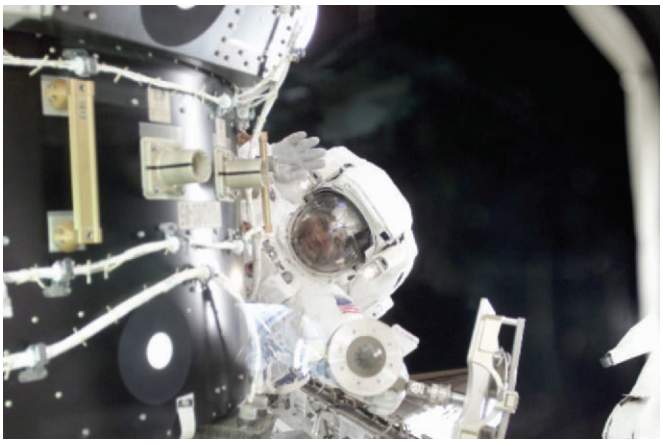

Figure 97-27. During an EVA on STS-98, astronaut Bob Curbeam was exposed to ammonia. (From http://spaceflight.nasa.gov/gallery/images/shuttle/sts-98/lores/s98e5190.jpg.)

Even missions to orbit risk exposure to outside contaminants, as was seen during STS-98, when an EVA crewmember became covered in ammonia crystals during connection of the U.S. Lab module to the rest of the ISS[50] (Fig. 97-27).

Medical waste falls into a special category, as it can be more hazardous to the crew than are other wastes. To date, there have been very few cases of illness (other than rare cases of SMS) in which crewmembers have generated large amounts of medical waste. Eventually, however, someone will become seriously ill or injured and produce a large amount of medical waste in the form of vomit, sputum, blood, diarrhea, or any other potentially contaminated body tissue or fluid. Microgravity significantly magnifies the problems associated with medical waste and caregiver protection.

There is also the question of what to do with medical samples. The current ISS blood analysis device, the iStat Portable Clinical Blood Analyzer,[66] only uses a few drops of blood, which are wicked into a self-contained cartridge. However, if longer-duration missions have expanded laboratory facilities, additional attention to the question of discarded samples will be needed.

Another question (that has largely been avoided to date) is what to do in the event of a death on orbit. Leaving aside the questions of how death would be ascertained and declared, what effects such an event would have on the other crewmembers, the impact on the mission, and other such matters, there still exists the issue of what to do with the remains. While returning the remains to Earth might be feasible, albeit unpleasant, on the Shuttle, the tight quarters of the Soyuz make it much more difficult to transport a body. And what about exploration missions? Even if a crewmember's death were to force abandonment of the mission, the time required to return to Earth might be too long to permit transport of an unpreserved cadaver. At present, there is no procedure to handle a death on orbit.

Handling of human urine and feces also poses health hazards. If the waste collection system is compromised, poor hygiene

could lead to disease outbreaks among the crew. Most current toilet systems, both Russian and American, work the same way: as wastes exit the body, they are drawn away by a stream of air and captured by the toilet.

The Shuttle toilet is known as the WCS (waste collection system) and is located in a closet-like space immediately aft of the side hatch. The compartment door is used as the ingress platform when astronauts crawl into the orbiter before launch (the vertical position of the orbiter on the launch pad makes the rear wall the floor). When it is used during flight, its door is opened and two curtains, attached to the top and the side of the door, are deployed to provide privacy. The WCS is actually a multifunctional system used to collect and process all biological wastes. The main function of the system is to collect and store fecal wastes and to process urine and transfer it to the waste water tank.[71,137] It also processes condensate and waste water from other Shuttle systems.

Crews also use urine collection devices during EVAs. Male astronauts wear a pouch-like device similar to a condom catheter, and women wear "disposable absorption and containment trunks," a pair of multilayered shorts that contain an absorptive powder.[1,3,71] Each device has a storage capacity of roughly a liter of fluid. Crew also wear urine collection garments (like adult diapers) underneath their launch and entry suits, because the Shuttle toilet is unavailable for use during those phases of flight (and even if it were available, the crew cannot easily doff the ACES suits).

To handle feces, the toilet has a cylindrical container in which a plastic bag is placed before use. Crewmembers use foot restraints and bars positioned over the thighs to hold themselves on the toilet seat, ensuring a good seal with the contoured, soft seat. The good seal is necessary to ensure that the toilet's airflow will be able to draw the waste away.[76,137] The hole for solid wastes is only 10 cm (4 inches) in diameter, and crewmembers actually practice positioning themselves on a toilet trainer before flight. A camera placed within the toilet trainer, directly beneath the hole for solid waste, allows them to see if they are positioning themselves correctly. A video showing proper use of the Shuttle toilet can be found at http://edspace.nasa.gov/text/livespace/gottago.html.

Feces enter the commode through the seat opening and are drawn in by air flowing through holes under the seat. This downrushing airflow (850 L/min) substitutes for gravity in collecting and keeping the waste material inside the commode.[76,130] The wastes are then broken up by rotating vanes and deposited along the walls in a thin layer. A hydrophobic liner inside the commode prevents free liquid and bacteria from leaving the collector. The plastic bag is then sealed, and a plunger attached to a lever forces it to the bottom of the cylinder. A new bag is then placed in the toilet for the next astronaut. When the cylinder is filled, it is replaced by a new cylinder. Figure 97-28 shows the design.

On the ISS, the toilet is in the Russian-built Service Module. This looks more like an Earthbound toilet that uses metal containers for storage. Prior to use, the containers are stored like empty buckets; however, once full they present considerable stowage and hygiene issues.

When other plant and animal life exist in the spacecraft, there are additional wastes produced, both in terms of quantity and types, with associated new microbial flora as well. This further increases the hazards to crew health in the event of improper waste management.

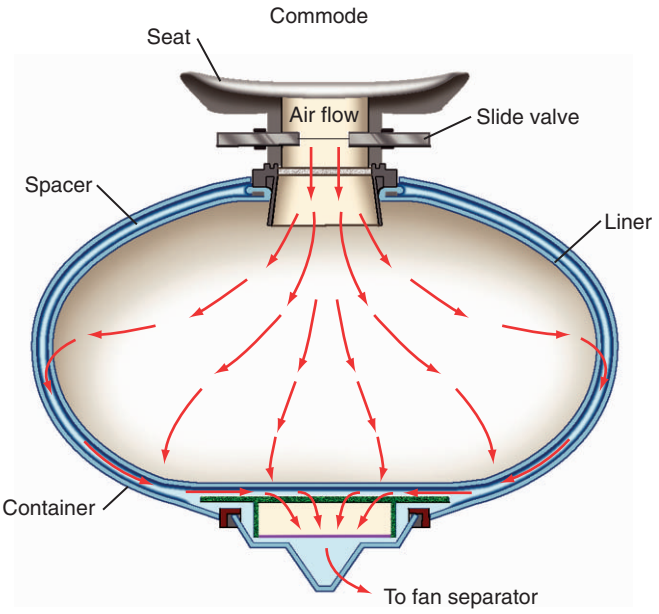

Figure 97-28. Shuttle toilet schematic. (From http://lsda.jsc.nasa.gov/kids/L&W/hygiene.htm.)

Figure 97-29. Mercury program boots, helmet, and gloves. (From http://web.mit.edu/16.00/www/aec/spacesuit.html.)

What to Wear?

In the early days of the space program, astronauts wore pressure suits during virtually the entire (short) mission. The Mercury "silver foil" spacesuit was a custom-fitted, modified version of the Goodrich U.S. Navy Mark IV high-altitude jet aircraft pressure suit. (Figure 97-29 shows Mercury program boots, helmet, and gloves.) By contrast, current spacecraft have a "shirtsleeve" cabin environment; in other words, no special gear or equipment is needed to live comfortably in the environment.[117,136] However, protective suits are worn during launch and re-entry phases of flight. Although the cabin environment at these times remains unchanged from the "shirtsleeve" parameters, crew wear protective suits because of the dangers inherent in these periods of flight. The suits differ somewhat between

the Shuttle and Soyuz spacecraft, but both are designed to protect the crew in the event of a problem during take-off or landing.

Another unique item of apparel is the Russian "penguin suit."[94] Designed as a countermeasure for muscle atrophy associated with prolonged exposure to microgravity, the penguin suit is a snug-fitting, full-length, long-sleeved jumpsuit that zips vertically to the crotch, with horizontal zip pockets on either side of the chest and just below the waist. The suit is made of a synthetic fabric with elastic inserts at the collar, waist, wrists, and ankles and along the vertical sides of the suit. The inside of the suit contains a system of elastic straps and buckles that can be used to adjust fit and tension of the suit. When astronauts and cosmonauts tighten tension on these bungee-like bands, the suit can exert resistance on the astronaut, opposing his or her movements and simulating some of the effects of gravity (see Figure 97-7).

A second Russian countermeasure suit makes use of lower body negative pressure and redistribution of blood volume to combat postflight orthostatic hypotension.[44] This suit was employed on Soviet and Russian space stations, but is not in use on the ISS. Future "special purpose" garments might include clothing into which physiologic sensors have been incorporated, allowing noninvasive monitoring. Examples include the VivoMetrics LifeShirt system (http://www.vivometrics.com/site/about_faq.html).

The clothing that is worn during an EVA is quite different from that worn by astronauts inside the spacecraft (known as intravehicular activities [IVA]). The American EMU (Extravehicular Mobility Unit) or Russian Orlan is like a tiny spaceship, one that is only large enough for a single inhabitant. Just like the Shuttle or ISS, it has its own life support and communication systems, provides a pressurized environment, and protects its wearer from the vacuum of space.

While they are in the EMU, crewmembers wear a liquid cooling and ventilation garment (LCG). The LCG contains a network of vinyl tubing that circulates cool water and air in order to maintain core body temperature at normal levels.[1,3] In conjunction with a thermal undergarment, it keeps an astronaut relatively comfortable while he or she is outside the vehicle and exposed to wildly varying temperatures (about −100° C to +120° C [about −150° F to +250° F], depending on whether he or she is in sunlight or behind Earth in shadow). The LCG that is worn under the launch and entry ACES suit only circulates water for cooling purposes. The garment that is worn under the EMU also circulates air in order to scrub out the CO_2 (Fig. 97-30) (http://www.nasa.gov/audience/forkids/home/CS_Astronaut_Fashion_Show.html).

It is important to point out that the current EMU is designed for use in orbit only. The spacesuits used by Apollo astronauts who walked on the moon were different, in part because of the different technology in those days, but also because the suits were subject to different strains, from moon dust to lunar gravity. Crews on planetary exploration missions in the future will need new spacesuits, some of which are currently being designed and tested (Fig. 97-31).

Personal Hygiene

With water a potentially mission-limiting consumable, personal hygiene in space is different from that on Earth. Water pressure on the ISS, for example, is about half that in a U.S. household. However, for morale purposes as well as basic hygiene concerns,

Figure 97-30. Shuttle crewmembers doff their EMUs after a spacewalk, showing the LCGs worn underneath. The bottom half of the EMU is visible on the crewmember at left. (From NASA Human Space Flight at http://spaceflight.nasa.gov/gallery.)

washing, shaving, and toothbrushing must be available for the crews, particularly on long-duration missions. Although both Mir and Skylab had showers on board (Fig. 97-32), none of the current spacecraft has them; instead, sponge baths, using wet washcloths, are the norm. This reduces the quantity of water required from 50 L for the average terrestrial shower to 4 L for the average ISS "bath."[59] Handwashing in space uses only about 10% of the water used for handwashing on Earth.[69,81]

Because perspiration doesn't roll off bodies the way it does on Earth, when astronauts sweat, droplets stick to skin and form thicker and thicker layers. Ventilation helps evaporate some sweat during exercise or on EVAs, but in the absence of an onboard shower, astronauts clean their bodies with wet wipes that are premoistened with soap and water. Astronauts on Mir and Skylab had showers, but these were difficult to use, required a great deal of time to set up and operate, and allowed water to "escape." In addition, the astronauts still had to smear a layer of water onto their bodies, so it wasn't that different from using washcloths.

A rinseless body bath solution is stored in standard drink pouches. Crewmembers add warm water to the pouches before use (there is no truly hot water available on orbit), then perform a modified sponge bath with washcloths. Waste water from bathing is either captured and sent to the wastewater tank or blotted up by towels, which can then be allowed to air dry in the cabin. Because of difficulties associated with hair washing, as well as the problems posed by stray hairs floating about the cabin, many crewmembers, particularly those on long-duration missions, prefer to keep their hair short. Nail clippings can also pose a problem. They can be inhalation hazards as well as having the potential to cause corneal abrasions.

Astronauts wash their hair with a rinseless shampoo originally designed for bedridden hospital patients. Crewmembers first wet their hair with water, then apply the rinseless shampoo (Fig. 97-33). The hair is scrubbed carefully to avoid flinging water drops throughout the cabin, then dried with a towel. Blow drying is not an option on orbit—crewmembers must wait for their hair to air dry. Generally the cabin humidity is held around 60%, which allows relatively rapid drying, although it

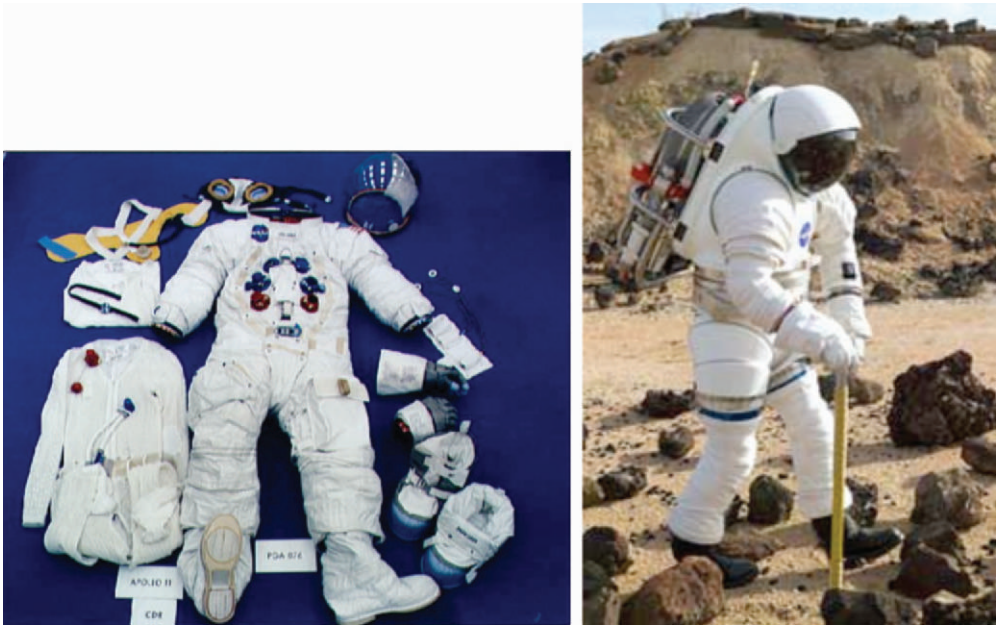

Figure 97-31. At left, Neil Armstrong's *Apollo* spacesuit. At right, a new model spacesuit for use on Mars is tested in Arizona. (Left image from http://www.fi.edu/pieces/hiley/images/jigsaws/Apollo%20Spacesuit.jpg. Right image from http://www.nasa.gov/vision/space/preparingtravel/desertrats.html.)

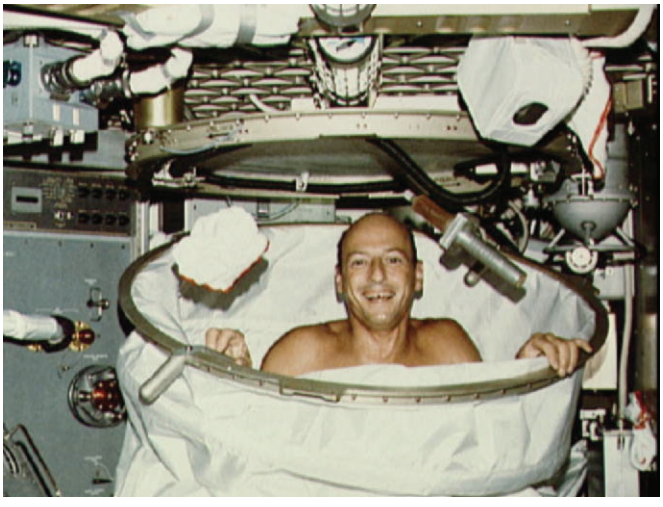

Figure 97-32. Astronaut Pete Conrad makes use of the Skylab shower. (From http://aerospacescholars.jsc.nasa.gov/HAS/cirr/em/11/11.cfm.)

does put additional strain on the humidity control system. A video of Pam Melroy washing her hair in space is available at http://spaceflight.nasa.gov/living/spacehygiene/hygiene_1.html.

For dental hygiene, astronauts use commercial toothpaste and toothbrushes. In weightlessness, saliva becomes more concentrated, which can lead to more tartar formation on the teeth. To compensate, many astronauts chew gum. The major difference between brushing teeth on Earth and in space is that astronauts must either swallow their toothpaste or spit it into a tissue.

Most male astronauts prefer to shave rather than allow their beards to grow. Generally, electric razors are preferred over safety razors and shaving cream, simply for ease of use when running water is not available.

Premenopausal female crewmembers often choose to use pharmacotherapy to suppress menstruation on orbit. For short-duration Shuttle crewmembers, it can be as simple as juggling the placebo week of oral contraceptives, delaying their periods until the mission is over. For ISS missions, however, female astronauts often turn to longer-acting medications, such as Depo-Provera, in order to avoid menses altogether. The alternative, using pads or tampons, creates an additional burden, namely dealing with contaminated waste. In addition, on long-duration missions, there exists a risk of running out of such supplies.

"Marching on Their Stomachs"

Food is a key part of crew morale and performance. The U.S. Navy has a great deal of experience showing how lack of variety in a menu or poorly prepared food can negatively impact a mission. Indeed, in addition to decrements in job performance, increased interpersonal conflicts have also been reported when palatable foods are in short supply.

As a result, there are concerted efforts to offer space travelers a variety of nutritionally balanced foods with menus individually tailored to each. Current U.S. menus include a variety of food, such as nuts, peanut butter, chicken, beef, seafood, tortillas (preferred to bread with its crumbs), candy, rice, and brownies. When international astronauts fly on the Shuttle, they often bring foods from their own countries. The current ISS food system is half U.S. and half Russian, with plans to add contributions from other International Partners as their astronauts join the ISS crew. Typical ISS foods currently include jellied perch, grits with butter, honey cake, beef with barbecue sauce, Russian cheese, cinnamon rolls, lasagna, apple-black currant juice with pulp, granola bars, stewed cabbage, lemonade, barley kasha, and chicken noodle soup.

Figure 97-33. Astronauts Bob Curbeam, Sandra Magnus, and Charlie Precourt wash their hair on the Shuttle with the rinseless shampoo. (From NASA Human Space Flight Gallery at http://spaceflight.nasa.gov/gallery.)

Unfortunately, despite these efforts, both the Russian and American space programs have consistently documented weight loss in crews during spaceflight. During the Mir program, up to 15% body mass losses were seen over the course of a 3- to 4-month mission. Approximately 1% of the loss can be attributed to water loss, due to the cephalad fluid shifts and redistribution associated with microgravity; the rest was due to bone, muscle, and adipose tissue losses. Loss of lean body mass is particularly concerning because it is associated with increased risk of skeletal fractures and thus compounds the microgravity-related effects on bone.

When adipose and muscle tissue losses are seen, particularly in the setting of lower caloric intake, inadequate nutrition is the default diagnosis. The stressful, isolated environment of spacecraft, early space motion sickness, limited menu choices, and altered taste sensation have been some of the factors frequently quoted to explain crews' weight loss in space. Microgravity-associated muscular atrophy (particularly in leg muscles, where there are significant decreases in muscle mass and performance) and bone loss (especially in weight-bearing bones) have also been implicated.

There is currently no refrigerator for food on the ISS or Shuttle, and lack of fresh produce and reliance on processed foods are likely contributing factors. Many of the physiologic adaptations to weightlessness may affect (or be affected by) nutritional intake. Although it is unlikely that an adequate diet will prevent all of these changes, it is distinctly possible that consuming too few nutrients can worsen the adaptations or at least delay an astronaut's postflight recovery.

Protein balance during spaceflight is particularly problematic. Decreased body proteins are associated with impaired performance, decreased immune function (which is also a documented effect of space flight), and clinical depression. Both caloric and protein intakes are traditionally lower in space; however, loss of body mass has occurred even in the presence of adequate nutritional intake. This suggests that protein balance may be altered significantly in the microgravity environment. Studies have shown increased whole body protein turnover during space flight, but the etiology and relevance of this finding remain unclear.

One major problem with consumption of processed foods on orbit is that they tend to be high in sodium. One study on the Shuttle documented dietary sodium intakes above 4000 mg per day.[68] Terrestrial research has demonstrated that excessive sodium increases bone turnover and urinary calcium excretion, which are already abnormal in space due to the microgravity environment. There is thus concern that the processed foods may contribute to (or worsen) bone demineralization (and subsequent increased renal stone risk) during flight. Although these findings are particularly relevant for ISS and exploration-class missions (because clinically significant deficits in bone mass are not visible until after several weeks of microgravity), they may have implications even for Shuttle flights, because biochemical changes have been found to begin within hours of weightlessness.

Systems for water storage in microgravity environments are significantly more complex than terrestrial systems are. Normally, gravity ensures that water stays in one place, separates from air, and moves down an incline. None of these things occurs in microgravity, necessitating enclosed containers, pressurized lines, and other methods for air–fluid separation. In microgravity, it is much easier to force water out of flexible-walled containers, because they can be squeezed or rolled, much like toothpaste tubes on Earth.[141] Unfortunately, these containers tend to have shorter lifespans than their rigid counterparts have. Some systems make use of both; in the Soyuz, for example, a rigid-walled tank holds a flexible bladder containing the water, and mouth suction is used to draw water out as required.

For many years, the color, smell, and taste of water in space have left much to be desired. Early U.S. spacecraft (*Mercury* and *Gemini*) used water from municipal water supplies. Tang, the drink mix forever linked to the early American space program, was actually created to mask the metallic taste of the Apollo spacecraft's water, although additional minerals and trace nutrients were also added. The Shuttle's use of iodine as a bactericidal agent led not only to complaints about the taste and color of the water, but also to elevated thyroid-stimulating hormone levels. An iodine removal system has since been added to the galley water supply to remove iodine from the water just prior to consumption. The ISS uses a silver-based system, but because the ISS does not provide cold water (as the Shuttle does), plain water is infrequently drunk. Rather, drink mixes (e.g., tea, coffee) or soups (bouillon, broths) are widely used.

Water quality can be compromised through biological contamination ("brown sludgy water"), chemical contamination

Figure 97-34. Astronaut Don Pettit with ISS water containers. (From NASA Human Space Flight at http://spaceflight.nasa.gov/gallery.)

("green glowy water"), or both. On short-duration missions, preflight verification of water quality is considered sufficient, but on longer missions, in-flight testing is also required, as contaminants could enter the storage containers during the course of the mission. Water pH, conductivity, and total organic carbon content are the parameters currently studied to ensure acceptable quality, although for exploration-class missions, the ability to perform in-flight spectrographic analysis will likely be needed.

Water is routinely delivered to the ISS via the U.S. Shuttle (often in 90-lb, duffle-bag-like water containers) and Russian "Progress" rockets (in large tanks), but doing so is expensive and creates dependency on ground supplies, rather than developing ISS autonomy[141] (Fig. 97-34). It was in part this dependence on Shuttle-delivered water that forced a decrease in ISS crew size following the grounding of the Shuttle fleet in February 2003, after the *Columbia* disaster. In the absence of regular visits from the Shuttle to replenish water supplies, a three-person crew could not be sustained aboard the ISS.

American engineers are currently developing a water recycling system that will collect water from fuel cells, urine, oral hygiene, and handwashing, as well as humidity from the air. The system is even designed to reclaim water from experimental animals that may one day live aboard the ISS. Scientists estimate that approximately 72 rats would equal one human in terms of water reclamation from respiration and urination.[38,48,57]

"Are We There Yet? I'm Bored!": Personal Recreation

For years, military leaders have been aware of the importance of providing adequate rest and recreation time to their subordinates, lest team performance drastically decrease. In many wilderness expeditions, the surroundings and the activity involved in getting to the wilderness areas provide all the recreation one can desire. In space, however, although the views can be breathtaking (Fig. 97-35), the sheer duration of the missions can lead to boredom.

Recreation takes many forms in space, depending on spacecraft geography, mission duration, and crew composition. On *Apollo 14,* for example, astronauts played some golf during

Figure 97-35. Astronaut Susan Helms enjoys the view from the ISS. (From NASA Human Space Flight at http://spaceflight.nasa.gov/gallery.)

lunar excursions, but that would not work well inside a habitat module. In Skylab, by contrast, where the three-man crews had a larger inhabitable volume than any other platform before or since, they could engage in acrobatics and tumbling.

On the ISS, where space is more limited and daily exercise is required, more sedentary pursuits are often enjoyed. Computers enable crews to enjoy music, games, books, or movies on a single device. Another popular source of entertainment is the shortwave radio. Many space station crewmembers have chatted with other radio enthusiasts during sojourns in space.

Bandwidth constrains communications to friends and family, but this has improved over the years. Astronauts on the ISS and Shuttle now enjoy a video teleconference with their families every week, and they also have e-mail and phone service, albeit somewhat intermittently.

Medical System Design

When designing a medical system, the first step is to determine the target population's needs. Design depends on preexisting medical needs, environment, skill and training of caregivers, and the ailments that are likely to occur. A thorough understanding of the activities in which the group will be engaged, as well as the likely outcomes, is critical for this.

Additionally, a significant component in system design is the level of risk the group is willing to tolerate. Under what circumstance(s) is a death or serious injury considered possible, and is this acceptable? If death were always wholly unacceptable, combat missions and many extreme sports could not exist. The potential for these outcomes to occur must be balanced against the costs (e.g., financial, training time, needed equipment) associated with trying to mitigate (or eradicate) their risk. As risk tolerance decreases, safer systems must be designed,

which usually increases cost. Similarly, if risk tolerance is relatively high, more danger within the system can be tolerated, and medical systems may be less comprehensive. The aerospace environment is a high-risk arena, but at the same time, public tolerance for risk is, generally speaking, low.

In addition, unless strong leaders demand a well–thought-out and integrated system, it is easy for well-intentioned individuals to insert into the system specific items of particular interest to them. The result can be an ungainly, poorly coordinated collection of isolated components, rather than a seamless system. Particularly in the aerospace environment, where upmass, storage space, resources, and training time are in short supply, lack of a well-planned medical system can be devastating.

The ISS may be used as an example to consider the requirements and challenges associated with designing a medical system. The ISS has a small, highly screened but diverse crew complement. There is no experienced clinician on board, and contact with the ground is only available roughly half of the time. It is an international program, which necessitates multilateral consensus on medical issues, and funding is controlled by nonclinicians who often (and rightly) consider medical issues as "off nominal" and not part of the primary mission objectives. Some guidance is provided by the System Specification for the ISS (NASA SSP 41000AG), Section 3.2.1.1.1.14e, which states that "the on-orbit Space Station shall provide preventive, diagnostic, and therapeutic medical equipment to monitor, treat, and maintain the health of a crew of six for the following criteria: provision of ambulatory care for up to 135 days, provision of emergency medical care for up to 72 hours."

This gives some indication of what the program directors originally expected from the medical system, but additional insight is needed. What is "ambulatory care"? What constitutes "emergency medical care"? What is supposed to happen after the 72 hour mark? Is it expected that a casualty will be evacuated to the ground by then? If so, will some sort of medically equipped evacuation vehicle be constantly available? Under what grounds should the determination of a "not survivable" injury or illness be made? What happens if an astronaut dies? Should the system include resources to deal with a cadaver? Will the mission be terminated in that event? In general, how much emphasis should be placed on diagnostics and therapeutics, compared with preventive measures? Should the bulk of the medical system be devoted to keeping crewmembers healthy (e.g., exercise equipment, environmental monitors) or to preserving life on those rare occasions when they are ill (drugs, imaging, hyperbaric chamber, evacuation capabilities)? Do we even know what illnesses and injuries we are likely to see, and how to recognize them? Given the physiologic changes exhibited in microgravity, it is reasonable to assume that signs and symptoms may vary. Can hypovolemia be recognized? Is jugular venous distention normal or abnormal? How long can a microgravity-adapted body preserve homeostatic function in the face of an insult before succumbing to shock? Should medical practices be adapted to accommodate these physiologic changes? If so, how?

What of the other operational constraints? Microgravity itself requires adaptation of many procedures, ranging from how caregiver and patient should position themselves, to the additional time needed for deployment of resources. The radiation environment affects system design, because some pharmaceuticals appear to break down more rapidly in space. Lastly, as is often the case in other wilderness venues, staffing must be considered a serious limitation. In addition to severe constraints on the number and type of experienced clinicians available, the number of astronauts on any given mission is often sufficiently small to create problems providing even unskilled care. How can tasks be best divided when a team member is incapacitated? What priority should be given to medical duties compared to primary mission objectives? What is the likely emotional impact of providing "buddy care" for another crewmember, particularly for prolonged periods? How much will fatigue impair the ability to achieve other mission goals?

Even if some of these answers are clear for an ISS mission in LEO, it is unlikely that the same answers will apply to a Mars mission, where such things as "medical evacuation" may be impossible due to orbital dynamics.

For the ISS medical systems, initial planning decisions favored a "scoop and run" concept, in which a crew return vehicle with medical resources would be permanently available and casualties could be evacuated speedily to a tertiary care center on Earth (Fig. 97-36). This allowed minimal on-orbit resources and required only limited medical training for crewmembers. This trade of in situ capabilities for rapid evacuation seemed appropriate, saving as it did up/down mass, stowage volume, and training time. Unfortunately, a change in the mission profile subsequently occurred, caused by changes in program funding, cancellation of the crew return vehicle (CRV) program, extension of mission duration, and decrease in crew size. As a result, neither in situ medical capabilities nor prompt emergency medical evacuation are currently available to ISS crews, creating a mismatch between required and actual system capabilities.[18a]

Predicting Likely Illnesses and Injuries

In the years ahead, commercial space ventures, as well as proposed missions to the Moon and Mars, may make space travel available to ever-increasing numbers of people. The new millennium witnessed the advent of "space tourism" with the voyage of Dennis Tito to the ISS and the successful flight of the world's first privately funded and built spacecraft (*SpaceShipOne* in July 2004). Heretofore, space travelers had been a highly trained and carefully screened group of people, who could almost be considered "pathologically healthy" when compared to the general public. However, it is doubtful that such rigid standards will be maintained in the future, and so increasing accessibility may lead to less fit people flying into space.

Selecting Limited Inventory of Clinical Resources (Benefit vs. Burden)

What do we know of illnesses during spaceflight and what can we expect as we increase the number of human-hours in space? NASA data on the first 89 Shuttle flights from 1981 to 1998 demonstrated that 98% of astronauts reported a medical event or symptom other than SMS during their mission.[21]

Like any other human endeavor into an extreme environment, the medical system for astronauts strives to minimize medical events among the crew and to maximize the ability to treat any problems during the mission with the most efficient use of resources. This cost-versus-benefit design is grounded in an analysis of known medical problems in space and analog environments. The health care system must be founded in evidence-based science, yet be flexible and improvisational. Its design must promote long-term health and optimize the performance of crewmembers. The essential strategy to achieve these goals is through primary prevention.

A

B

C

Figure 97-36. **A** to **C,** three views of the prototype NASA Crew Return Vehicle ("X-38"); **B,** A flight test over the Mojave Desert; **C,** Landing in the Mojave. (From NASA Human Space Flight at http://spaceflight.nasa.gov/gallery.)

The health care system design relies heavily on ground-based support led by the flight surgeon. This lead physician is primarily responsible for the health and well-being of the crew, and has the authority to dictate care and, in extreme cases, to terminate the mission. He or she plays an integral role in the health monitoring and training of the crew over the 2 or more years preparing for the mission. During the mission, the flight surgeon can direct the CMO in giving appropriate care to other crewmembers. The CMO is usually not a health care practitioner and receives limited training in medical care prior to the flight. One of the challenges of a long-term mission to Mars is this reliance on ground-based support. During such an extended mission, there will be delayed communication with Earth (up to a 40-minute delay, depending on orbital positions), so a more independent system of health care delivery (likely involving a physician crewmember) will be required.

System Design

Particularly in the case of medical facilities, good design features can make a critical difference. Unfortunately, none of the aerospace medical systems to date has shown particularly good architectural integration. On both the Shuttle and ISS, the galley and toilet are geographically close to each other. In the event of an illness, hygiene could be compromised and food consumption areas contaminated. In the Shuttle's cramped environs, there is no dedicated location where a sick crewmember can be placed to avoid contamination of the rest of the crew—or merely to decrease what is often (particularly for nonclinicians) the provocative stimulus of someone vomiting nearby. These close quarters can pose hazards to both general health and performance.

The ISS, with its larger internal volume, does have a dedicated location for casualties, complete with "bed" (a foldout, electrically isolated crew medical restraint system). However, the ISS medical system is based in the U.S. Lab module, at the other end of the station from the toilet facility. This suggests that if someone is seriously ill, they may have to choose between the two—having access either to medical supplies (including medical oxygen) or to a toilet. Not only will this increase the amount of care other crewmembers will have to provide to a casualty, but it also creates additional challenges for managing medical wastes.

The flip side of this debate is the undesirability of sequestering highly valuable in-flight space for exclusively medical purposes. Medical issues are not among the primary mission objectives, so many people feel that the space currently assigned to medical tasks and storage could be better utilized by payloads or other systems. It can be difficult to strike the proper balance between contingency-based systems (such as clinical assets or fire suppression systems) and those that are used during nominal operations (such as those for propulsion or food preparation).

The needs of LEO missions differ from those of exploration missions, but the overarching principles remain the same. Should space be dedicated to certain functions or remain flexible? Will there be a designated user with specialized training and knowledge (as is the case in terrestrial hospitals, where only certain personnel know how to operate the CT scanner, for example), or will the system be sufficiently user-friendly that any member of the team will be able to use it even without prior familiarization? Costs for equipment meeting the latter conditions likely will be significantly higher, because there are few commercially available, off-the-shelf devices that meet those criteria. On the other hand, if only a single team member has the necessary skills, what will the team do if that person is incapacitated and cannot provide or guide care?

Even if a trained caregiver is available, decision-support software and a robust informatics infrastructure can provide a great deal of assistance. In the event of a mass casualty—such as a widespread epidemic of food poisoning or multiple cases of hypoxia—even minor time savings can be important.

Medical systems must accommodate the number of patients they can be expected to treat. Will there be two crewmembers, as on some ISS missions, or 200, as might be expected in a permanently manned lunar base? In the latter case, a large, dedicated space with associated supplies and equipment might be needed, but such a resource would be a waste for the former crew complement. When evacuation and terrestrial-based care are impractical, as on a Mars mission, it may make more sense to invest in resources that will enhance wellness, and philosophically embrace a lower threshold for "nonsurvivable" conditions.

Currently, there is a professional, highly trained, and extremely motivated astronaut corps. However, the current astronauts are, in many cases, less fit than the first Mercury Project test pilots, and it is likely that as more people venture into space, be they tourists or maintenance workers on a moon base (Fig. 97-37), fitness levels will decrease further. Accordingly, the medical system must be prepared to handle a wider variety of preexisting conditions. This is similar to trends seen terrestrially, as a wider range of people embark on "adventure tourism" and as more contractor personnel are used in military operations overseas.

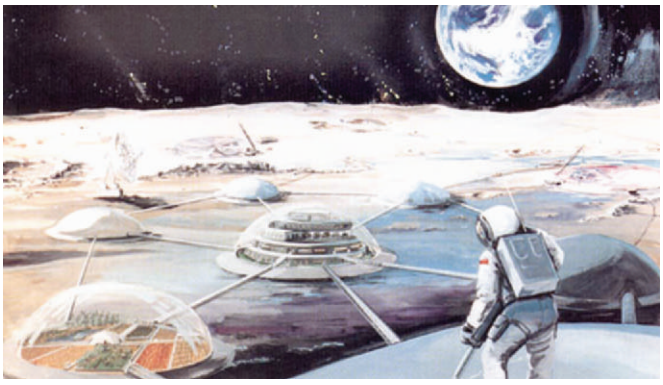

Figure 97-37. Artist's rendition of a moon base. (From NP-119 Science in Orbit: The Shuttle & Spacelab Experience, 1981–1986, at http://history.nasa.gov/NP-119/ch2.htm.)

Keeping Them Healthy: Accomplishing the Mission

In-Flight Exercise Countermeasures Programs

As early as the 1960s, Busby identified "periodic physical exercise" as a means for "maintaining an optimum level of physical 'fitness' during space missions." In the early days of the space program, vehicle dimensions did not permit much physical exercise to occur. Since the days of Skylab, in-flight exercise has been used as a way not only to have fun (as numerous crew photos attest), but also to monitor physical condition and to prepare for various tasks, such as an EVA.

One of the systems most affected by prolonged exposure to microgravity is the musculoskeletal, because the lack of a gravitational field causes muscle atrophy, bone demineralization, diminished exercise tolerance, loss of strength, increased risk of kidney stones, osteoporosis, and increased fracture risk. Even during a mission to LEO, musculoskeletal changes can prove dangerous, such as excessive fatigue leading to premature end of an EVA. Exploration-class missions could be seriously compromised if crewmembers become so weakened by their time in microgravity that they are unable to carry out physically challenging tasks upon arrival. An expert panel voiced concern that "an abrupt return to gravity [whether back on Earth or at the destination of an exploration mission] imposes high workloads on weakened muscles and [causes] post-space travel pathologies: muscle fatigue, weakness, incoordination, and delayed onset muscle soreness. . . . [A]daptation to the lower workload in microgravity may render muscle tissue more prone to structural failure when it is reloaded."[21]

One way to avoid such outcomes is through regular in-flight physical exercise, because the majority of muscular alterations during space flight are believed to be caused by gravitational off-loading. As such, the changes should be responsive to exercise-based countermeasures. Some of the standard terrestrial exercises require adaptation for use in the microgravity environment. Weightlifting is meaningless in the microgravity environment—there is no "weight" to "lift." Instead, strength training occurs through resistive devices, ranging from very simple (e.g., bungee cords, which were used as far back as the Gemini program) to highly complex equipment, such as the ISS RED (Resistive Exercise Device). For aerobic training, crewmembers have tested everything from rowing devices to exercise bicycles.

Cycle ergometers (Fig. 97-38) have proved popular, not only because bicycling is a familiar terrestrial activity, but also because with a simple substitution of hand grips for pedals, the same device can be used for upper limb training as well as lower limb. This is particularly useful for EVA crewmembers, as the majority of the work done during spacewalks is performed by the upper body.

Figure 97-38. Astronaut Frank Culbertson on the Shuttle middeck works out on the bicycle ergometer. (From http://spaceflight.nasa.gov/gallery/images/shuttle/sts-105/hires/s105e5004.jpg.)

Figure 97-40. Astronaut Yuri Gidzenko working on the ISS interim RED (Resistive Exercise Device). (From http://spaceflight.nasa.gov/gallery/index.html.)

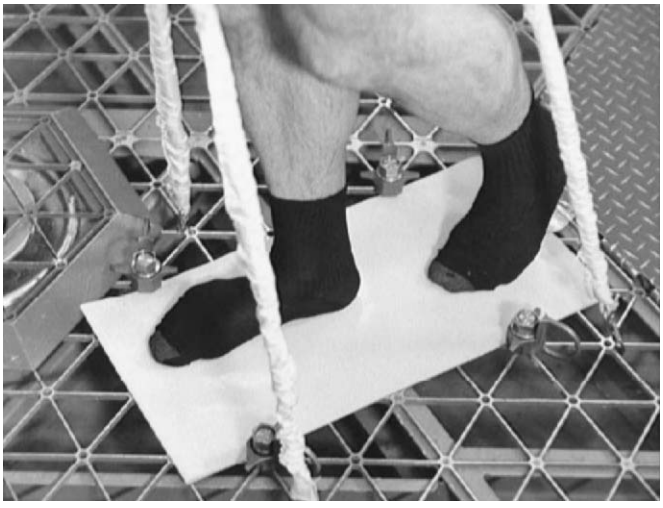

Figure 97-39. The treadmill developed for Skylab 4 consisted of a Teflon-coated aluminum plate bolted to the "floor," a restraint harness, and bungee cords. (From http://images.jsc.nasa.gov/luceneweb/caption.jsp.)

A rowing machine (a modified treadmill) was flown occasionally on the Shuttle, but did not prove as popular as other equipment, such as the ergometer and standard treadmill.

Various types of treadmill devices have been flown over the years, from unpowered ones with a Teflon-like surface to allow easy sliding (Fig. 97-39), to powered ones such as the current ISS model. In all cases, a restraint harness is required to hold the astronaut down against the treadmill surface.

The goals of the physical exercise countermeasures program include maintaining lean muscle mass and strength (thereby enhancing EVA performance and reducing risk of injury), preserving aerobic capacity, maintaining bone mineral content and structure (to reduce risk of injury during and after the mission, including the risk of long-term osteoporosis), and lowering excretion of urinary calcium (in order to reduce risk of renal stones).

The value of strength training versus aerobic conditioning for astronaut health, performance, and postflight recuperation has been much debated, but there is little solid evidence to determine which (if either) is the best countermeasure. Similarly, there are few data to identify the preflight training regimen that will best prepare astronauts for long-duration space flight and their eventual return to earth's gravitational field. As a result, both strength and aerobic exercises are currently performed by long-duration crewmembers before and during their sojourn on the ISS. Usually 30 minutes of each 90-minute training session are devoted to aerobic conditioning, with the remaining 60 minutes devoted to resistive exercise.[133] The RED allows numerous muscle groups to be exercised, including the back, quadriceps, and arms (Fig. 97-40).

Cycle ergometry use has been associated with improvements in cardiovascular deconditioning, but it does little to impair skeletal muscle atrophy. Exercise on the treadmill (Fig. 97-41), by contrast, may be able to improve cardiovascular parameters and prevent a common problem of foot-drop positioning with associated extensor range shortening and minimize skeletal deconditioning through its associated axial load and heel strike. When used in conjunction with visual stimulation (e.g., watching a DVD while running on the treadmill), it may also enhance neurovestibular readaptation. However, additional controlled studies are required before any particular form of exercise can be clearly labeled "best."

Generally, heart rate and equipment settings are used to track efficacy of the exercise program. This information is downlinked to the flight surgeon and athletic trainers with whom the crewmember had worked to prepare for the mission, and in-flight exercise "prescriptions" are then modified as appropriate. This is a highly staff-intensive method of establishing proper exercise regimens; in the future, with Moon or Mars bases and increasing numbers of personnel, it is likely that some less intensively monitored system will need to be developed.

To date, the vast majority of our space flight experience has been with relatively short missions in a microgravity environment, but the next decades may well see a return to the Moon

Figure 97-41. Astronaut Andy Thomas uses a treadmill aboard the Russian Mir Station. (From http://spaceflight.nasa.gov/gallery/images/shuttle/sts-91/hires/91375011.jpg.)

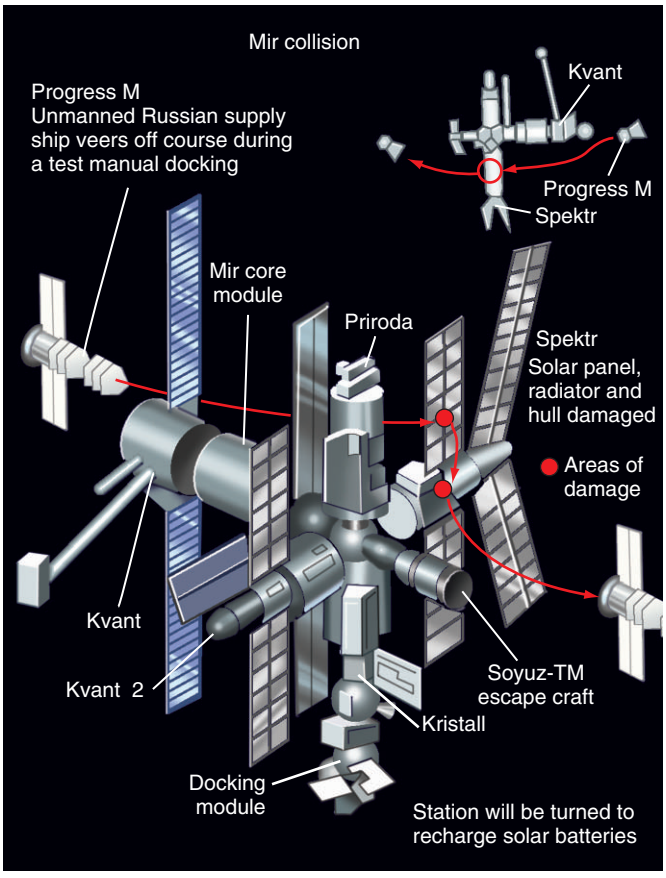

Figure 97-42. A graphic explains the damage caused in the 1997 collision between the Mir station and a supply ship. Crew fatigue was cited as a causative factor in the collision. (From http://www.cnn.com/TECH/9707/mir/repair/mir.collision.lg.jpg.)

and even a journey to Mars. These missions present new questions, as for the first time in over 30 years, space travelers will be exposed to gravitational forces (approximately ⅓ G on Mars, ⅙ G on the Moon), rather than microgravity. Although the impact of partial gravity on physiologic alterations remains unclear, it is only prudent to assume the worst and plan for the same level of exercise as that required to counteract the effects of microgravity.

Crew Medical Training (Initial, Sustainment, and "Just in Time")

Crew medical officer training has varied from basic first aid techniques to endotracheal intubation to dental extraction procedures. In part, the extent of training has been based on mission duration; the longer a crew is in space, the greater the chance that people will fall ill or be injured. However, allocated training time also reflects the level of risk acceptance and the financial and time-based constraints.

Historically, both ground-based flight surgeons and physician-astronauts have been drawn from all medical specialties. For exploration-class missions, however, it has been argued that certain skill sets should be identified prospectively and appropriate training provided. Along with a physician-astronaut, exploration crews are likely to require some kind of medical assistant. Whether this should be someone with paramedic-level training, nursing skills, or simply basic familiarization with medical equipment remains to be determined.

It may be that astronauts on exploration missions should all receive a higher level of medical training to enhance cross-training and minimize the likelihood of losing key skills in the event of an emergency. In addition, medical system design could feature preferential use of devices that are simpler to use and require less training, rather than devices that necessitate sophisticated skills. For example, direct laryngoscopy was the method traditionally taught to CMOs for use in space, but instruction in the intubating laryngeal mask airways was recently added to the ISS medical system; intubating laryngeal mask airways require less training for successful placement, and the training lasts significantly longer than does training for direct laryngoscopy.

Psychological Support

On long-duration missions, psychological support becomes critical to mission success. This takes many forms, from support of the family that has been left behind to avoidance of overwork and fatigue (Fig. 97-42).

It is well established that complex task performance decreases after a certain period of persistent work, and that cumulative fatigue and boredom play causative roles in the deterioration. Although drugs such as dextroamphetamine sulfate (Dexedrine) and caffeine can temporarily improve performance, the effect rapidly wanes, often to a level below that originally noted. By contrast, a period of restful sleep restores subjects to their original level of performance—demonstrating the value of healthy sleep/wake cycles.

Figure 97-43. A sleep station in the ISS lab module. (From NASA Human Space Flight at http://spaceflight.nasa.gov/gallery.)

As a result, the international space medical community has developed rules governing crew rest. NASA flight surgeons attempt to prevent fatigued crews from performing dangerous tasks. Although lack of normal circadian cues in space makes it more challenging to determine when crews are at their best (or worst), it is clear that crews require a few days to adjust to a new sleep schedule before they function at peak performance (Fig. 97-43).

Postflight Rehabilitation

After the crew has returned to Earth, the flight surgeon performs standard physical examinations on landing day and 3 days later. Flight surgeons meet the crewmembers at landing, whether in a Shuttle or Soyuz, and accompany them to a crew rehabilitation site, usually at JSC or Russia's Star City. After a few days of minimal physical demands, the rehabilitation process begins in earnest and is considered the crewmembers' top priority.

The goal is to return the astronaut to preflight condition (and thus to flying status) as quickly as possible. Following short-duration Shuttle flights, this may happen within a few days or weeks, but recovery from long-duration missions may take months or longer. Major milestones along the rehabilitation pathway include return to the family home, unlimited driving privileges, return to terrestrial flight status, return to short-duration space flight status, and return to long-duration space flight status.

Particularly after long-duration space flight, rehabilitation is generally guided by the same athletic trainers and flight surgeon who supervised the crewmember's in-flight exercise program. Continuity of care helps to improve the exercise regimen's efficacy as well as the interpersonal dynamic between the crewmember and his or her health care team.

The "continuum of preventive, therapeutic, and rehabilitative care on the ground, during space travel, and upon the return from space travel" described by the Institute of Medicine as required by astronauts, particularly those returning from long-duration missions, has worrying implications for exploration programs. If ISS astronauts, after flights of several months, require the close attention and rehabilitation currently provided to them, how will any Mars-bound astronauts be able to function upon reaching their destination?

Perhaps travelers to the Moon's surface will be less affected, since their trip is so much shorter, but if a moon base is established with mission assignments of months or even years, what in situ countermeasures might be needed by its personnel? On a base of any size, support personnel (e.g., cleaners, cooks, mechanics) will be required. What will happen to their long-term health if they are less fit preflight than are current astronauts or lack discipline for in-flight countermeasures?

Potential Long-term Sequelae

Despite the amount of attention paid to physiologic changes after space flight, one of the body systems most affected by missions to the ISS cannot benefit from rehabilitation. The hearing of several crewmembers has been impaired by the high levels of ambient noise on the ISS. As a result, hearing conservation programs have been developed and implemented, while work continues on mitigation strategies. In-flight audiometric testing is performed, and personal protective equipment is available to crewmembers. Prevention is the most effective strategy; in cases where a hearing decrement is noted, the flight surgeon in the postflight period can only perform serial assessments and hope that the loss proves temporary.

Many questions exist about whether astronauts experience higher incidence rates of radiation-related cancers, cataracts, or (due to the Shuttle's iodinated water system) thyroid problems. So far, the only difference between the astronaut and control group in terms of morbidity and mortality is a higher number of trauma-related deaths among the former.[21] Because the astronaut population is arguably a less risk-averse group than the general population, this finding is perhaps unsurprising.

Meeting the Crisis: When Bad Things Happen

As the numbers of hours that humans spend in space increases, so does the likelihood of a major incapacitating medical event that will require resuscitation, chronic care, or both. The body's response to trauma has been well documented on Earth, but there are few data on wound healing and post-traumatic inflammatory responses in a microgravity setting. In fact, there is still surprisingly little research or planning underway on how to provide long-term care in space—as might be required following serious injury or illness during an exploration-class mission.[18a] Instead, most efforts to improve "crew health and safety" to date have focused on crash survival, equipment, and training.

Crash Survival

Crash survival for space flight depends greatly on the phase of flight (launch, in-flight, landing). To date, the U.S. program has suffered the loss of two orbiters, one on launch (*Challenger,* January 1986), and one on landing (*Columbia,* February 2003). In neither case was the crash survivable, but both caused re-examination of the crew escape systems and crashworthiness of the Shuttle fleet. The Soviet/Russian program also suffered

several losses and near-tragedies and adapted their vehicles and/or procedures as a result.

In both of the Shuttle disasters, the crew compartment survived the initial event, but the astronauts died from blunt force trauma. In the case of *Challenger,* the crew was killed when the crew compartment impacted the ocean[124]; with *Columbia,* deaths occurred when the crew compartment finally tore apart as the Shuttle disintegrated around it and the astronauts were flung out into the air stream.[36] The crew compartment is deliberately constructed to be the most robust part of the orbiter, but in both the *Challenger* and *Columbia* disasters, the additional protection was of no value. There was no way for either crew to effect an escape despite the fact that the cabin was not immediately compromised. Unlike many terrestrial aircraft, there are no ejection seats in the current Shuttle; weight and volume constraints precluded their inclusion once the crew size increased beyond two. Instead, other escape systems are used by space travelers, some of which have been modified as a result of lessons learned in the two disasters.

Crew Safety Equipment

During launch and landing, crewmembers wear several pieces of equipment designed to improve their ability to survive a crash. Equipment includes a special pressure suit, parachute, rescue equipment, and survival equipment. In addition, the orbiter itself is equipped with several devices to improve crew survival, including a side hatch jettisoning system, egress slide, escape window, escape pole for mid-air bailouts, and "sky genie" rappelling devices.

The suit worn by astronauts in the U.S. program has changed over the years. Early in the program, no pressure suit was used, merely a (baby blue) jumpsuit. After the loss of *Challenger,* STS mission 51-L, on 28 January 1986,[139] the decision was made to have the crew wear pressure suits during launch and re-entry to protect them in the event of a loss of cabin pressure at altitudes up to 30 km (18.5 miles) and to insulate them from cold air and water in the event of a bailout. A suit was developed to provide combined anti-G protection, emergency pressurization, anti-exposure protection, high-altitude escape, and sea survival for shuttle orbiter crews. These partial pressure (twin-walled bladder type) suits were used with a specially integrated parachute, emergency oxygen system, and survival kit pack, which were worn on the back in combination with the suit.[111] Each LES garment with helmet weighed approximately 11 kg (approximately 24 lb), excluding parachute/survival pack.

The current bright orange suit, affectionately dubbed the "pumpkin suit," is donned with the help of a cadre of suit technicians in the Suit Room of the Operations and Checkout (O&C) Building at Kennedy Space Center before the crew head to the launch pad. Following successful insertion into orbit, the crew remove these suits and stow them until just before they take their seats for reentry. Donning and removing these suits in the small, microgravity environment of the Shuttle middeck is a tricky task, and crewmembers must take turns and assist each other. Gloves and helmets are usually the final items to be donned because crewmembers often find them hot or uncomfortable.

If the helmet or gloves are not worn, however, the protection offered by the suit is radically compromised. The suit functions as a "hard" pressure suit at cabin altitudes above 60,000 ft (18,288 m), when bailout is not yet possible. It can serve as an unpressurized suit at altitudes below 40,000 ft (12,192 m), where a bailout could occur.[111] If properly worn, the suit can protect the crew from loss of cabin pressure or cabin oxygen to 100,000-ft altitude (30,480 m), protect against pooling of blood in the lower extremities, and protect against cold temperatures both of the upper atmosphere and of the ocean water in the event of a bailout.[10] The suit delivers oxygen at suitable pressure and quantity (under 100,000-ft altitude) and protects the wearer from a toxic environment, including smoke, combustion products, and flames.

The suit controller is located on the right chest and regulates internal suit pressure through a valve that maintains pressure in the suit bladder and thus on the body. The valve opens to allow exhaled air to flow through the bladder and controller, but closes if the ambient cabin pressure is equivalent to that above an altitude of 35,000 to 40,000 ft. When the valve is closed, the crewmember's respirations inflate the bladder until the internal suit pressure and cabin pressure are 3.5 psi (24.1 kPa). The suit oxygen regulator is the means by which pressurized oxygen enters the helmet from stores in the orbiter. Two exhalation valves between the helmet cavity and suit bladder allow exhaled breaths to enter the bladder and exit through the suit controller. A relief valve on the right hip opens if the suit pressure reaches 5.5 psi (37.9 kPa) and remains open until the pressure has fallen to 3.5 psi (24.1 kPa).

The detachable anti-G suit that is worn only during landing has both abdominal and partial leg bladders and is controlled by a device at the upper left leg. The suit's function is to apply pressure to the body to counteract G-forces and prevent pooling of blood in the lower extremities, which would compromise cerebral perfusion. Although the orbiter does not perform any high-G maneuvers as terrestrial fighter aircraft do, the crew's microgravity-adapted physiology puts them at very high risk for orthostatic hypotension. The suit has proved helpful in preventing presyncope during landing.

The main purpose of the suit's helmet is protection of the head during bailout. It also provides a conduit between the communications carrier assembly (CCA) and headset interface unit (HIU). It has a rotating faceplate with pressure visor and rotating sunshield. An antisuffocation valve allows ambient air to enter if the visor is closed and no oxygen is available. Unfortunately, it can also let in water if the crewmember is submerged. The valve is designed to open when the pressure inside the helmet is 0.54 psi (3.7 kPa) less than outside pressure.

In addition to the pumpkin suit, the crewmember also wears a parachute harness and personal parachute assembly (PPA). The harness is the link between the astronaut and parachute, and it supports the crewmember's weight through nylon shoulder, chest, and leg straps. It also holds several pieces of survival and rescue gear, including emergency oxygen supply, life preserver unit (LPU), carabiner with Velcro cover, and emergency drinking water supply (Fig. 97-44). The harness attaches to the PPA at four points. The PPA contains the parachute and risers, a SARSAT (search and rescue satellite-aided tracking) radio beacon, and a single-person life raft. During nominal operations, it serves as a seat back cushion.

The PPA has a D-ring for attachment to the Shuttle's bail-out pole, the parachute deployment system, and a ripcord to serve as a backup system for chute deployment. There is also a drogue chute release knob, a backup method to deploy the main chute once the drogue has deployed. The pilot chute has an 18-inch-diameter (45.7-cm-diameter) canopy and serves to pull out the 4.5-ft-diameter (1.37-m-diameter) drogue chute. Once the

Figure 97-44. This picture of water survival training shows not only the parachute harness, but also the leg pockets where additional survival gear is stowed. (From NASA Image Exchange [NIX] at http://nix.nasa.gov/.)

Figure 97-45. Life preserver unit (LPU). Staying afloat during water rescue training. (From NASA Image Exchange [NIX] at http://nix.nasa.gov/.)

drogue chute has stabilized the crewmember, it is safe for the 26-ft-diameter (7.92-m-diameter) main chute to open. This is a modified circular military canopy with four antioscillation windows and a pyrotechnic reefing system to minimize the opening shock to the astronaut. The seawater-activated release system (SEAWARS) is attached to each parachute riser. When it is immersed in seawater, the SEAWARS releases the riser, separating the crewmember from the chute. There is a manual backup system.

Survival gear is stowed in several places. In the left suit leg pocket is a flare kit containing seven cartridges, each of 4.5 seconds duration and visible for up to 50 miles (80 km). The pocket is also stocked with a knife, exposure mittens, two 12-hour chem-lights, and one strobe light good for 6 to 9 hours of continual use or 18 hours of intermittent use. Smoke/flare signals that produce 16 seconds of orange smoke or 20 seconds of red light are also included. A survival radio is located in the right suit leg pocket. It can both transmit and receive; at 90% receive/10% transmission, its battery will last 24 hours. The radio can also send out a continuous beacon and is equipped with an earphone and spare antenna. Also in the pocket are motion sickness tablets (scopolamine–dextroamphetamine [Dexedrine]) and a signal mirror.

Attached to the harness is the LPU (Fig. 97-45). Horseshoe-shaped, it can keep an unconscious person's head out of the water and inflates automatically from carbon dioxide bottles upon immersion in water. It can be inflated manually if necessary.

An emergency oxygen system containing two bottles provides an additional 10 minutes of oxygen at moderate levels of exertion. The harness holds a 2-L (~0.5-gallon) supply of drinking water, plus a 2-ft (61-cm) tether line with carabiner, which can be used by rescuers to hoist the crewmember into a vehicle.

The PPA contains a single-person life raft. It is composed of eight rows of inflatable tubes, of which three inflate immediately upon immersion via the automatic firing of a carbon dioxide canister. The crewmember is then expected to fire a second CO_2 cartridge to inflate the remaining five rows. The raft can be inflated by mouth if necessary.

The raft has two spray shields with Velcro closures, a bailing cup and pump (Fig. 97-46), and reflective tape on all sides. It is connected to the PPA by a 12-ft (3.66-m) Kevlar tether. The SARSAT radio is in a pocket on the spray shield; its antenna loops around the shield, and it is automatically activated upon deployment of the parachute. The radio transmits on several frequencies: 121.5 (international distress), 243.0 (military distress), and 406.0 (satellite search and rescue). The raft has a sea anchor with a dye marker tethered to the exterior of the raft, and an extra dye packet.

Orbiter Equipment

The orbiter has a side hatch that is normally used for egress after landing. It also comes equipped with an escape slide, similar to those seen on commercial aircraft, for rapid exit in an emergency. If the side hatch is unavailable, window 8, located on the roof of the crew cabin when the orbiter rests on its landing gear, can serve as an emergency escape hatch. If the crew exit via the window, they can use "Sky Genie" descent-control devices to rappel off the sides of the orbiter and reach the ground safely. For an in-flight bailout, the most hazardous escape condition, the orbiter has an escape pole to prevent the crew from tumbling against the wing and rear of the orbiter after they jump from the crew compartment via the side hatch.

Figure 97-46. Bailing out the life raft during training. (From NASA Image Exchange [NIX] at http://nix.nasa.gov/.)

The side hatch is the primary means of entering and exiting the Shuttle.[111] In the event of an emergency where the hatch needs to be opened by the crew, for example in the case of a mid-air bailout, the crew would first pull a vent valve T-handle located behind the waste management compartment. This opens a 15-square-inch (97-square-cm) hole between the crew compartment and payload bay, depressurizing the cabin in a controlled manner. Once the cabin and ambient pressure have equilibrated, the hatch jettison T-handle is pulled. This blows the hatch by activating four linear-shaped pyrotechnic charges, two per hinge, which sever the hatch hinges. Simultaneously, two expanding tube charges sever the 70 bolts that hold the hatch adapter ring to the Shuttle, and 3 thruster packs push the hatch away at about 45 ft/sec (about 13.7 m/sec).

The escape slide, also known as the emergency egress slide system (EESS), can be used whether the hatch is opened in a normal fashion or jettisoned. It is activated by the crew, inflated automatically via an argon bottle, and remains usable for at least 6 minutes.

The window escape system is intended for use when the side hatch is unavailable. First the crew must jettison the escape panel via a pyrotechnic system, then attach to a Sky Genie, climb onto the MS2 seat and then out window 8, and rappel down the Shuttle's starboard side. The pyrotechnics are mechanical and require no power to activate. The handle is located on the top of flight deck panel C2, although a second, external control panel is located on the starboard side of the orbiter so that rescue personnel can also open window 8, if necessary. When triggered, the system ejects the window's outer thermal pane and frame outward and upward; 0.3 seconds later, the window's two inner panes hinge down and aft, coming to rest on the aft flight deck panel. A capture device prevents the inner panes from opening too forcefully and holds the panel open. If the system fails, a prybar is available to the crew as a backup.

The Sky Genie system is used to lower crewmembers from the top of the orbiter to the ground quickly but safely. It consists of a descent device, 50 ft (15.2 m) of nylon rope, rope bag, crewmember tether and hand loop, swivel snap shackle, and emergency release tab (Fig. 97-47). It can be used from either window 8 or the side hatch if the EESS is unavailable.

Figure 97-47. Astronaut Pamela Melroy practicing with the Sky Genie. (From NASA Image Exchange [NIX] at http://nix.nasa.gov/.)

In the event that neither the side hatch nor window 8 is available, rescue personnel have to gain access by cutting into the crew compartment at a specially designed section of the hull. This takes at least 45 minutes and is a method of last resort.

The bailout escape pole is designed to guide crewmembers to a trajectory that will clear the Shuttle's left wing. It is a 275-lb (125-kg) curved steel and aluminum pole with a spring-activated, telescoping deployment and eight lanyards available for the crew. It can also be activated manually. The aluminum housing attaches to the middeck ceiling and side wall by the side hatch. When the Shuttle reaches orbit successfully, the pole is removed and stowed.

In the event of a midair bailout, the crewmember uncovers the D-ring on his PPA right riser, leans forward into the hatch area, attaches the D-ring to the snap hook on the outermost lanyard, and falls forward out the open hatch. The pole is designed to carry the crewmember a safe distance away from the orbiter.

Crew Operations

Under normal conditions, the crew don their ACES suits in the O&C building, then go to the launch pad. At the 195-ft (59.4-m) level of the Fixed Service Structure (FSS), the platform alongside the Shuttle, they are helped into their parachute harness and CCA. They leave the "White Room" on the Orbiter Access Arm (OAA) and enter the Shuttle middeck through the side hatch. The order for ingress into the orbiter is by seating assignment, starting with the flight deck personnel (commander [CDR], pilot [PLT], mission specialists 1 and 2 [MS1, MS2]) and followed by the middeck personnel (MS3, MS4, MS5).

The "closeout crew" help the astronauts into their seats, where the parachutes are already positioned. At this time, the

orbiter is in an upright position in preparation for launch, so the crew lie on their backs on top of their parachutes. The support staff help the crew to strap in, and the CDR and PLT adjust their headsets and controls. The support team assists with communication and suit pressurization checks, then they leave, closing the Shuttle hatch behind them.

Approximately 1 hour after a normal launch and successful achievement of orbit, the mission specialists leave their seats, disconnect their suits from the orbiter systems, configure the flight and middecks for orbital operations, and lock the side hatch. Once the CDR and PLT have finished their piloting duties (at about 90 minutes after launch), they too leave their seats. All crewmembers then remove and stow their ACES suits.

On landing day, the crew don their suits just before assuming their seats for reentry. After landing, crewmembers unstrap immediately after wheel stop and wait for the support staff to open the side hatch and enter the Shuttle. The convoy crew, accompanied by the flight surgeon (who is usually among the first ground crew personnel to enter the Shuttle), help the astronauts to exit the vehicle and remove their suits.

There are eight emergency egress modes for getting the crew out of the Shuttle.[10] The mode type depends on the phase of flight during which the emergency occurs, the nature of the emergency, and whether support staff or rescue personnel are available to help the astronauts. In modes 1, 5, and 8, the crew must exit the orbiter without assistance. In 2, 3, 4, 6, and 7, ground personnel are present.

In all of the modes, certain basic assumptions are made.[10] First, the crew is expected to egress unaided as much as possible, even if rescue staff are present. Next, it is assumed that the crew will help each other—this may be particularly important if some of the crew are returning from long-duration missions, as when a Shuttle is ferrying home an ISS crew. The people who are authorized to declare a contingency and order an emergency egress are the crew commander, flight director, NASA test director (NTD), and convoy commander.

Mode 1 is an egress at the pad during the prelaunch period. In this mode, the closeout crew have already left the launch platform, the side hatch is closed, and the situation is too critical or unsafe to dispatch rescue teams. The crew must therefore leave the orbiter unaided.

When an egress is called for by either the CDR or NTD, the OAA must be repositioned. (The OAA is retracted from the Shuttle 7 minutes before launch, but it can be put back in place in about 30 seconds.) The crew unstrap themselves and leave their seats, then open the side hatch and crawl out headfirst to the 195-ft (59.4-m) level of the FSS. The orbiter is in its vertical launch position, which means that everything in the Shuttle is "on its side," making rapid movements difficult and creating numerous tripping hazards. They proceed in a buddy system to emergency slidewire baskets that are located in recessed areas on the west side of the FSS (Fig. 97-48).

Each of the seven baskets can hold up to three suited people and has fire-resistant material around it to protect occupants. The baskets are suspended from ¾-inch (1.9-cm) stainless steel cables by two trolleys, one of which has an anti-rollback device. Once the crewmember and his or her buddy are in the basket, they use a guillotine-like device to sever the Kevlar rope that holds the basket in place. They then slide, in a matter of seconds, to a ground-level landing zone 1168 ft away from the pad. The baskets are stopped by nets, and the south wall of the basket is released by pulling a D-ring.

Figure 97-48. Astronauts practice entering the slidewire baskets. (From NASA Image Exchange [NIX] at http://nix.nasa.gov/.)

The crew then leave the basket and hurry to shelter within a concrete bunker. Up to this point, the crew commander has been in charge; at the bunker, however, the NTD is in command. Depending on the precise nature of the emergency, they may leave the bunker in an armored personnel carrier.

In mode 2, another pad egress in the prelaunch period, the seven-person closeout crew is still present, and the side hatch is still open, but (as in mode 1) the situation is too hazardous to dispatch additional rescue personnel. When an egress of this type is ordered, the closeout crew assists the astronauts out of the Shuttle and proceed into the baskets and to the bunker with them.

In mode 3, another pad egress in the prelaunch period, the closeout crew has left, and the side hatch is closed. However, in this case, a seven-person rescue team can be sent to the 195-ft (59.4-m) level of the FSS to help the astronauts. The crew still tries to get out of the Shuttle as best they can on their own. If they cannot, then the rescue personnel open the side hatch and help them out. The astronauts and rescuers then leave the FSS, either by the slidewire baskets or the elevator, at the discretion of the rescue team leader.

In mode 4, the closeout crew is still present, the side hatch is open, and a rescue team can be sent in. The rescue personnel then help both closeout crew and astronauts to leave the Shuttle, and all leave the area together.

A mode 5 scenario is an egress postlanding, with the astronauts unassisted by ground personnel. The crew must leave the Shuttle through the side hatch (opened or jettisoned) or window 8. A "hatch jettison mode 5" is used if there is imminent danger to the crew, such as fire or collapsed landing gear. A "hatch on mode 5" is appropriate when an egress is not emergent but should be expedited or if the landing has occurred at a site without convoy crew support. Crew will descend to the ground using the EESS (preferred) (Fig. 97-49) or Sky Genie. If the side hatch cannot be used, then the commander will declare a "window 8–mode 5" egress. If the Shuttle communications systems are not available after a mode 5 emergency, the crew can contact search and rescue forces using either survival radios or light signals.

Figure 97-49. Practicing for a Mode 5 egress with the EESS. (From NASA Image Exchange [NIX] at http://nix.nasa.gov/.)

In a mode 6 egress, the Shuttle has landed on or near the runway, and a prepositioned convoy crew is available to assist the astronauts. In a mode 7 scenario, the Shuttle has landed in a remote area, and the crew are assisted by rescue personnel who arrive in helicopters.

A mode 8 emergency is an in-flight bailout. It can be over land or water, during launch or reentry.[111] The astronauts must leave the orbiter *during controlled, gliding flight at or below 30,000 ft (9100 m)*. This is an important distinction, because this criterion was not met in either the *Challenger* or *Columbia* disasters. Therefore, in-flight bailout would not have been possible (even if all the other requisite factors had been present).

During a mode 8 emergency, the commander calls for a bailout at 50,000 ft (15,200 m), and he or she then places the Shuttle in a minimum sink rate attitude and engages the autopilot while the crew ready their suits. At 40,000 ft (12,200 m), the CDR orders MS3 to vent the cabin via the pyro vent. MS3 then monitors the cabin pressure via an altimeter while the rest of the crew ready their D-rings and release their seat restraints. At 30,000 ft (9100 m), CDR orders MS3 to jettison the side hatch via the T-handle. The crew disconnect themselves from Shuttle communication and oxygen systems. At 25,000 ft (7600 m), MS3 leaves his or her seat and deploys the escape pole. If, however, deconditioned crewmembers are on board, MS3 will help them while MS2 deploys the pole. The crew leave their seats and move to the side hatch. The crew then bail out, with automatic opening of their parachutes. The main canopies should open around 14,000 ft (around 4300 m).

Rescue of the crew following a mode 8 egress could take at least 8 hours, depending on location and advance warning. The first rescuers are likely to be a C-130 Hercules aircraft with two teams of 3 pararescue jumpers (PJs) and a motorized rescue boat. The C-130 will drop two 20-person life rafts in which the astronauts and rescuers can await the helicopters.

In a case where a mode 8 bailout is ordered shortly after launch (also known as a contingency abort), the orbiter would be destroyed as soon as the crew had evacuated in order to avoid danger to cities along the East Coast.

In addition to the eight modes mentioned earlier, there are also aborted launch scenarios. In a *return to launch site* (RTLS) maneuver, the orbiter performs what *Popular Mechanics* magazine calls a "terrifying somersault in the sky."[34] In this scenario, a major problem that would prevent the Shuttle from safely reaching orbit (for example, an engine failure, cabin pressure loss, fuel leak, or compromised cooling system) occurs within seconds of launch. An RTLS can be ordered within the first 4 minutes after liftoff and will return the Shuttle to Kennedy Space Center within the next 30 minutes. To execute an RTLS, the commander must, while flying at Mach 6.5, flip the Shuttle and its external fuel tank 180 degrees and glide back to a landing at Kennedy Space Center. Astronaut John Young is quoted in *Popular Mechanics* as saying, "We have never done a powered pitch-down separation at Mach 6.7, very rapidly. Theoretically it will work, but practically will it work? . . . I'd like to see us do away with RTLS."[34]

Adding to the challenge of flipping the Shuttle around at that speed is the fact that the Shuttle would likely have a full payload bay during the maneuver, since it would occur shortly after launch. If the orbiter (which weighs about 500 tons [about 454,000 kg]) is carrying a heavy payload, such as the ISS lab module (30,000 lb [13,600 kg]) or the Chandra observatory (13,000 lb [5900 kg]), then its weight will be even greater and its center of gravity significantly different from what the commander is used to maneuvering during nominal landings. Depending on how large and massive the payload is, and how well secured, it is possible that it could even break free of its restraints during landing and slide forward, perhaps damaging (or crushing) the crew compartment. And, of course, the external fuel tank would be half full during the "flip."

The maneuver must be delayed until after the solid rocket boosters (SRBs) have burned out and separated from the Shuttle at 2 minutes after launch (Fig. 97-50). Within 30 seconds of SRB separation, and while the main engines are still firing, the commander must initiate the RTLS. At first the Shuttle would continue to fly away from Florida, at a steep pitch in order to burn off fuel in the external tank. At an altitude of about 40 miles (about 64 km) and a speed of nearly Mach 7, the commander would perform a "pitch around," in which the Shuttle's nose would be brought to point toward Florida, with the orbiter flying tail-first and with the engines firing. As it flies through its own 5000° F (2760° C) exhaust, the Shuttle would slow.

As the Shuttle lost speed and altitude, the external tank would be rapidly emptied of fuel. When the external tank was down to below 2% of its capacity, the Shuttle could separate from it without its bouncing against the orbiter. The tank would fall into the ocean while the Shuttle (now much lighter) would gain speed. Eventually, without additional fuel, the engines would shut down and the Shuttle, now gliding in a steep dive, would approach Florida. As it neared the Kennedy Space Center, the commander would pull out of the dive and enter the normal glide slope for a routine landing.

Unfortunately, this maneuver has never been tested. John Young refused to perform it during the first Shuttle flight, likening it to "Russian roulette."[34] Astronauts practice it in simulation, but as John Young says, "The first person that does one will be able to tell you whether it works or not."[34] Fellow Shuttle commander Ken Cockrell agrees: "The first crew to do it—if it should ever happen, and hopefully it won't—would be along for quite a ride."[34]

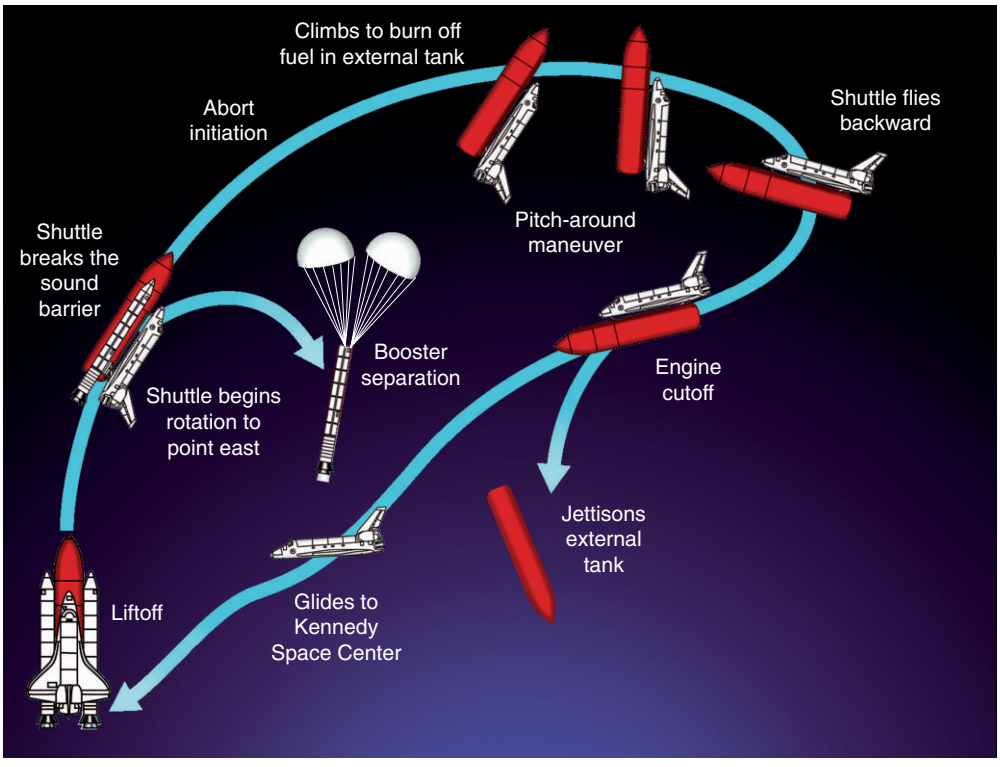

Figure 97-50. The RTLS maneuver cannot begin until after the solid-fuel booster rockets burn out and separate. The RTLS is divided into the steps shown in this diagram: It begins with the shuttle being put into a steeper climb to burn off its load of liquid fuel. The pitch-around maneuver turns the orbiter and fuel tank around. Thrust from the main engines gradually cancels the shuttle's forward motion from Mach 7 to 0. Main engines are then shut down. The external tank is jettisoned, and the shuttle enters a steep dive toward the landing site. The pilot pulls the shuttle out of the dive. The shuttle enters a glide slope for landing at the Kennedy Space Center. (From http://popularmechanics.com/science/space/2000/12/Astronauts_in_Danger/print.phtml.)

Transoceanic abort landing (TAL) sites are used in the event a failure develops too late for an RTLS to be used. Between 2½ and 8½ minutes after launch, a TAL can be ordered, and the Shuttle would then follow a trajectory across the Atlantic Ocean to a landing strip at one of four potential landing sites: Ben Guerir Air Base in Morocco, Banjul International Airport in Gambia, Zaragoza Air Base in Spain, or Morón Air Base in Spain.[67] Each of these sites has been equipped with appropriate rescue devices for use with the Shuttle and is staffed with NASA personnel, Department of Defense (DOD) personnel, or both during all launches and any contingency landings. In a TAL site scenario, the Shuttle would land within 45 minutes after liftoff.[89] The particular choice of TAL site depends on the orbiter's payload and planned orbital insertion inclination. (Shuttles may launch with inclinations between 28.5 and 57 degrees. The lower inclination launches allow for heavier payloads.)

Although less risky than an RTLS, TAL landings remain challenging—as described earlier, landing a Shuttle immediately after liftoff, with a full payload bay, can be significantly different than the normal postmission landings.[89] On a brighter note, the astronauts will not have suffered any microgravity-related deconditioning and will be in peak physical shape to perform any physical activities after landing, such as making an emergency egress (e.g., mode 5).

The SRBs and external tank will separate before landing, but the Shuttle will have to be flown at an altitude of about 350,000 ft (about 107 km) until after main engine cut-off (MECO) and external tank separation. The Shuttle will also have to perform a roll to maneuver to a heads-up position, and it will dump extra fuel to decrease weight and restore the center of gravity to a more usual location.

At 6 minutes before TAL landing, its speed will be Mach 2.5 and its altitude about 82,000 ft (about 25 km). One minute later, it will have decreased its speed to Mach 1. A minute after that, the commander will take over piloting control from the on-board computers and will execute a large turn to align the Shuttle with the center of the runway; this maneuver is called *intercepting the heading alignment circle* (HAC) or "turning the HAC." The maneuver decreases the speed still further and allows the Shuttle to enter its final approach at 13,000 ft (3960 m). At 1800 ft (550 m) altitude and 7500 ft (2290 m) from the end of the runway, the commander performs a preflare to change the glide slope from 19 degrees to 1.5 degrees. Speed at touchdown is ideally about 300 knots per hour (about 550 km/hr).

If all has proceeded according to plan, a routine powerdown of the orbiter, requiring 30 minutes, would take place before the crew exit the vehicle—unless an unsafe condition requires an emergency egress—and, if all goes well, the crew are expected to board a C-130 for home within 3 hours of landing at the TAL site.

In an *abort once around* (AOA) or *abort to orbit* (ATO), a malfunction will have rendered the engines incapable of carrying the Shuttle to its planned orbital altitude. As a result, in these abort situations, the Shuttle will fly to a lower altitude, orbit earth once, and then land. This orbit buys time for the ground control teams to study the problem and allows the Shuttle to land at a primary landing site (PLS) such as Edwards Air Force Base.

Figure 97-51. The CSA-CP (see text). (From Johnson Space Center Environmental Toxicology at http://www1.jsc.nasa.gov/toxicology/.)

Hazards: What They Are and How to Control Them

Providing a single type of hazard-control system for the spacecraft cabin is impractical, as it would need to cover the wide variety of chemicals required for years of basic research on multiple space platforms. Instead, every experiment is required to provide its own system of hazard control and to track its chemicals (and byproducts) at all times (a "cradle-to-grave" approach). Review by the Safety Office determines what containment level each rack-mounted experiment facility must have, and it provides explicit instructions for the crew on proper cleanup procedures in the event of an accidental release.

Prevention is always the most effective method of hazard control, though operational and mission objectives sometimes require dangerous substances to be flown. Safety review and engineering are also routinely used to minimize crew exposures. For example, a safety review might suggest that a less hazardous substance be substituted or that engineering controls be put in place to minimize the risk (such as the redesign of the Apollo thruster nozzle/pressure valve placement). A variety of methods, such as Drager tubes to sense the presence of specific chemicals, carbon monoxide detectors, and the compound specific analyzer-combustion products (CSA-CP) device (Fig. 97-51), are all used to alert the crew when protective measures must be taken.

When these preventive measures fail, however, more active measures must be employed, including use of oxygen masks (as were used on Mir or Apollo-Soyuz) or decontamination procedures (such as during the STS-98 event).

Fire Hazards

The closed environment of a spacecraft, like a submarine or airplane, is a deadly place for a fire. The confined space, limited atmosphere, and severely constrained evacuation routes all contribute to a fire's lethality. As a result, preventive measures are considered the first and most important line of defense. However, despite the best attempts, fires have still occurred, including a "serious electrical fire" on Salyut 1 (June 27, 1971) and another on the Mir space station in February 1997, believed to have been caused by a crack in the SFOG casing. Jerry Linenger, the U.S. astronaut on the Mir at the time of the fire, described it as a "very large flame bursting out of the canister [with] smoke billowing out. . . . [Molten] metal was flying out and splattering on the opposite bulkhead."[78] The dense black smoke not only posed a choking hazard but also obscured the environment, making it easy for the crew to become disoriented and causing difficulty in finding things, including the escape route. As another Mir crewmember remarked, "[In space,] you can't escape the smoke. You can't just open a window to ventilate the room."[113]

Oxygen-enriched environments pose a fire hazard, particularly when ambient pressures are at or above sea level (14.7 psi) (101.4 kPa). Following the *Apollo 1* tragedy, NASA established strict rules governing the oxygen concentration of the cabin environment in order to minimize this risk.

The pyrolytic byproducts of a fire can be more lethal than the thermal effects, particularly for a smoldering blaze in a confined space. Following a fire, exponential increases in atmospheric contaminants occur. Among the contaminants detected after the *Salyut 1* and Mir space station fires were smoke, particulate matter, benzene, carbon monoxide, hydrogen cyanide, hydrogen sulfide, and carbon dioxide. In addition to atmospheric contamination, there was also significant deposition on cabin surfaces, which required additional cleaning. Following the 1997 Mir fire, after removing their portable oxygen masks, crewmembers wore surgical masks during the cleanup and for several days thereafter to protect themselves from the particulate matter and "grime" in the atmosphere.[78]

Another combustion event aboard Mir, in February 1998, was much less spectacular in terms of fire and smoke—paper filters within a low temperature catalytic oxidizer ignited—yet it caused high levels of carbon monoxide (one of the monitors read 400 ppm) in the cabin environment for several days, and the crew reported symptoms ranging from headaches to nausea.[113] In many ways, a smoldering fire of this sort poses a more insidious hazard to the crew, because it can produce large quantities of carbon monoxide, which (being colorless and odorless) can go unnoticed until serious exposure has occurred.

The ventilation system is a critical component of the fire detection apparatus. Normal circulation of the air on board not only ensures homogeneous gaseous composition and temperature but also enables early detection of particulate matter, including smoke. In the event of a fire, the ventilation system can also spread the smoke throughout the spacecraft. Therefore, fans are shut off when a fire is detected in order to minimize the spread, although (as was demonstrated on the Mir) this tactic may be only partially successful.

Fire detection and suppression on board spacecraft must make use not only of crewmembers' senses but also of automated systems, particularly in unoccupied areas such as avionics bays. Detectors that sense carbon monoxide as well as smoke are necessary, and independent suppression systems

must be located in areas of the spacecraft that the crew cannot reach.

Fire extinguishers may make use of water, Halon, or carbon dioxide to extinguish a blaze in space. NASA prefers to avoid water because it can be ineffective in electrical fires. The Shuttle uses a Halon 1301 agent that is highly effective and not overtly toxic in its original form. However, when combusted, it produces cardiotoxic byproducts that are difficult for the life support system to remove from the atmosphere. Although carbon dioxide–based extinguishers require a higher concentration than Halon extinguishers do to suppress a fire, the atmospheric control systems can much more easily remove carbon dioxide from the cabin, and no additional hazards are created.

Russian fire extinguishers generally use water in foam or spray form. This avoids the problem of added toxins, but can be messy to clean up. It is also possible to extinguish a fire by denying it oxygen, either by smothering it with wet towels or by sealing off and venting the involved module. This latter option depends on the severity and extent of the fire, as well as availability of a safe haven for the crew; subsequent recovery of the decompressed module may be difficult.

During the 1997 Mir fire, crewmember Sasha Lazutkin initially attempted to smother the flame with a wet towel, but it was instantly consumed by the rapidly growing conflagration.[78,113] [A smaller fire in 1994 that began with an identical oxygen generator had been successfully smothered by cosmonaut Valery Polyakov, using an extra uniform.] Commander Valery Korzun then used a water-containing extinguisher, which created steam that added to the already dense smoke, further obscuring the atmosphere. The fire was finally extinguished only after Korzun directed the contents of three fire extinguishers into and over it.[78]

Electrical Hazards

The effects of an electrical shock can range from an unpleasant tingle to cardiac arrest and death. On the Shuttle, most risks of electrocution have been "engineered out," but spacecraft construction, whether of the ISS or, eventually, of lunar and Martian habitats, poses greater risks. Whenever space travelers are called on to build, rather than inhabit, there is unavoidable exposure to wiring and other hazards, making injuries more likely.

For nonconstruction activities, internal vehicle systems and payloads are carefully screened by numerous groups, most notably the Safety, Reliability, and Quality Assurance Office, to ensure that electrical hazards are noted and avoided wherever possible.

Risk can be mitigated by careful preplanning and extensive practice of any procedures in which electrical hazards exist. In some cases, this attention may enable alternative methods to be used, thus eliminating or minimizing the risk. If the hazard cannot be avoided, proper technique and attention to safety may provide additional safeguards. As tertiary prevention strategies, the medical system should be designed with these hazards in mind, so that proper care may be provided in a worst case scenario. Currently, on the ISS, a defibrillator is part of the Health Maintenance System and could be used in the event of an electrically induced cardiac arrest. However, in the absence of a crew return vehicle that can safely evacuate a severely compromised casualty, doubts remain about the ability of the current ISS medical system to provide the necessary continuing care.

Chemical Hazards

As described above, there are several potential sources for chemical contamination of the spacecraft environment. Propellants (such as hydrazine) are notoriously dangerous; coolants (such as ammonia or Freon) can leak into the atmosphere. Cleaning materials may also contain hazardous chemicals. Over time, volatile organic compounds (from polymer materials) can leach into the cabin environment, or toxic chemicals can be abruptly created, as in a fire.

In addition, the chemicals that are associated with the payloads and scientific research usually performed in spacecraft can pose hazards. Our understanding of the chemicals present in the spacecraft environment is only as good as the systems and sensors available to detect those chemicals. One problem following the 1997 Mir fire, for example, was that there was no onboard means to analyze air samples, and by the time samples were returned to earth for analysis, additional breakdown of the substances could have occurred. As a result, a great deal remains unknown about the actual hazards experienced in flight. However, there have been several chemical exposures during past missions that provide an opportunity for study.

Case Study

In 1975, Apollo-Soyuz astronauts Slayton, Stafford, and Brand were exposed to toxic fumes during their descent. At approximately 24,000 ft, an inadvertent firing of the capsule's jets allowed nitrogen tetroxide (which served as an oxidizer for the capsule's thrusters) to be sucked from a thruster outlet into the spacecraft through a nearby cabin pressure relief valve. The crew described it as "the cabin seemed to flood with a noxious gas . . . It irritated the skin . . . and the eyes . . . We started coughing." Nitrogen tetroxide is highly toxic; it can be lethal in sufficient quantities and can lead to both short-term effects (pneumonitis) and long-term effects (heightened vulnerability to respiratory illnesses). The crew was forced to break out the oxygen masks, but in the interim before Stafford could retrieve and distribute the masks, Brand (who was sitting closest to the valve) blacked out. Stafford reported, "I knew that I had a toxic hypoxia and I started to grunt-breathe to make sure I got pressure in my lungs to keep my head clear. I looked over at Vance [Brand] and he was just hanging in his straps. He was unconscious." With an oxygen mask in place, Brand regained consciousness within a minute, but all three subsequently developed pneumonitis, for which they were eventually hospitalized, but from which they fully recovered.[124]

More recently, on STS-98 in 2001, an EVA crewmember was covered in ammonia from coolant lines during connection of the U.S. lab module to the ISS.[50] Although the spacewalker was unaffected, the ammonia crystals posed a contamination hazard to the cabin environment when he reentered the Shuttle. His EVA partner assisted him in brushing off as much of the ammonia as they could during the spacewalk, and he then stood in the sunlight so that more could sublimate off the EMU. Following return to the Shuttle airlock, several procedures to safeguard the intravehicular crew were taken, including donning of

oxygen masks by those inside the vehicle, wiping down the contaminated EMU with wet towels, and cycling the airlock. Eventually, after the EMU had been cleaned and the entire cabin air volume had been circulated and scrubbed, the astronauts were allowed to remove their oxygen masks and test the air. No ammonia odor was detectable, and the hazard control procedures were deemed successful; however, this demonstrates that unexpected chemical hazards may be encountered even during normal operations.

Mechanical Hazards

The mechanical hazards on board spacecraft are significantly more limited than those faced on earth. Falls, motor vehicle–related accidents, and other high-energy–related sources of injuries are largely absent. With the exception of collisions between spacecraft or with micrometeorites and orbital debris, the energy in most microgravity-related injuries is relatively low. A space traveler can push off from a bulkhead with a fair amount of speed and thus could inflict or suffer an injury if an unintentional collision happened soon thereafter, but it would require a willful disregard of safe practices.

As a result, most mechanical hazards in space do not reach life-threatening levels. Pinch or crush forces exist and pose hazards, particularly to hands or digits. Torque forces could cause injuries to joints, especially during EVAs when footholds are routinely used. Sharp edges or recoiling bungee cords can also pose hazards. The latter caused a problem for one astronaut, who narrowly escaped a significant injury to the eye when a bungee cord snapped unexpectedly and struck him in the face.

All payloads and systems are carefully examined at several phases along the design and flight certification pathway to identify any potential hazards to the crew. Wherever possible, designs are adapted to eradicate the hazard or workarounds are suggested. In addition, safety procedures are developed for any unavoidable hazards, such as when the Expedition One ISS and STS-98 Shuttle crews were instructed to don eye protection to guard against any floating debris or metal shavings prior to entering the newly attached U.S. Lab module for the first time.

Although a significant issue in terrestrial aviation, vibrations are not a significant source of health concern in microgravity. An aircraft's propulsion system and atmospheric turbulence are the primary sources of vibration experienced by terrestrial aircrews, but they are not present in current spacecraft, which (following launch) rely more on inertial forces rather than powered flight, and (by definition) are unaffected by atmospheric conditions. Vibrations, outside of the launch or landing phases (where they are associated with the stresses of flight), are usually associated with either equipment or crew activity. Certain payloads can cause vibrations and may require special scheduling to prevent their inappropriately influencing other experiments. Although vibration can have significant effects on certain payloads, it is not at a level to create difficulties for manned operations. Noise, on the other hand, is a major concern in space, just as it is in terrestrial flight.

On future spacecraft, wall coverings may help to reduce the acoustic burden within the metal vehicle, and could provide noise muffling as well as insulation. Care must be taken, however, to avoid microbial growth or a potential fire hazard.

Personal protective equipment in the form of earplugs can be used to counteract loud ambient noises. However, many crewmembers find them uncomfortable or a nuisance to wear and as a result do not wear them as often as they should. This can lead to problems not only for space travelers' long-term hearing, but also for their ability to hear caution and warning alarms during the course of the mission.

Challenger and *Columbia* Disasters

The causes of the two worst disasters in American space flight go far beyond the mechanical failures that led to the orbiters' demise. Although the scope of these tragedies' origins is vast, the essential lessons in wilderness expedition planning and execution remain the same.

A successful undertaking in the wilderness—be it up mountains, over bodies of water, or into outer space—should occur within the limits of the equipment and support capabilities and maintain a steadfast adherence to safety guidelines. Although the specifics of each Shuttle disaster were unique and unprecedented, we can look to similar well-known enterprises in the past—Shackleton's Antarctic Expedition[73] or the 1996 climb on Mt. Everest described in Krakauer's *Into Thin Air*[70]—to understand that discordance between reality and expectation can ultimately lead to malfunction with dire consequences.

Much of our understanding of the roots of these events comes from the *Columbia* Accident Investigation Board's (CAIB's) extensive research into the origins of both tragedies. Beyond mechanical failure, it was the culture at NASA that, in the opinion of the CAIB, led to the Shuttle disasters.

Problems beyond Mechanical Failure

When the Shuttle was conceived at the end of the Apollo lunar landings in the early 1970s, it was initially intended as a reliable, reusable orbiter that would service an international space station—possibly multiple stations—that would act as a platform for bases on or orbiting the Moon, and eventually to a manned Martian landing. However, having won the "space race" to the Moon and consumed with domestic strife and the Vietnam War, the Nixon administration deemed the spending on space exploration politically unsustainable. The NASA budget was drastically cut, and with it went any hope for a space station for a Shuttle to service.

NASA's response was to develop a new justification for the Shuttle program, based on the need for access to LEO for scientific, commercial, and military purposes. By marketing the Shuttle in such a light, NASA, the CAIB wrote, "cast itself in a role that its nascent technology could not support."[36] Partly because of funding concerns and European competition with the commercially viable Ariane rocket, the Shuttle was prematurely declared "operational" in 1982, claiming that it had surpassed its testing missions and was well on its way to providing "routine and frequent" access to space.[36] The CAIB termed this image "illusory" and argued that it led to unsupportable expectations and pressures on the space agency.[36] In response, NASA increased an already tight launch schedule while simultaneously struggling to diagnose and treat the multitudinous problems associated with the Shuttle's first-generation technology. The result was unintentional oversights and tolerance of errors based on the fallacious argument that because such problems had not proved troublesome on previous flights, they must not be true hazards. In other words, deviance from established safety standards was not only tolerated, but it became the norm, and systems needed to be proved *un*safe, rather than safe.

On January 28, 1986, an O-ring on a solid rocket booster failed, causing the loss of the *Challenger* and its seven crewmembers shortly after lift-off.[139] President Reagan appointed the

Rogers Commission to investigate the tragedy. In addition to identifying the immediate mechanical cause of the disaster, the board also found that the push to render the Shuttle operational had exceeded the vehicle's capabilities, and the Commission faulted NASA for overlooking safety practices.

The board specifically cited both NASA and Morton-Thiokol, the contractor for the solid rocket booster, for failing to address known design abnormalities during the testing of the boosters, instead writing off such anomalies as "acceptable flight risks."[36] The Commission called for an independent NASA office that would oversee safety and quality assurance and report directly to the NASA Administrator. In short, noted the Rogers Commission, the NASA managers "may have forgotten—partly because of past success, partly because of their own well-nurtured image of the program—that the Shuttle was still in a research and development phase."

After the *Challenger* disaster there was a push for Shuttle upgrades, the addition of the *Endeavor* to the fleet (replacing *Challenger*), and a cutback of launch schedules to a more realistic timetable. In a further diminution of the Shuttle mandate, the President and the Defense Department respectively announced in 1986 that Shuttles would no longer carry commercial or military satellites.

As the Shuttles resumed flight in the late 1980s, the Soviet Union was collapsing, thus ending the Cold War between the U.S. and Russia. The end of the Cold War further lessened the political impetus for a space program, and Congress responded with reduced funding for NASA. "Rather than adjust its ambitions to this new state of affairs," concluded the CAIB, "NASA continued to push an ambitious agenda of space science and exploration, including a costly Space Station Program. . . . The search for cost reductions led top NASA leaders over the [1990s] to downsize the Shuttle workforce, outsource various Shuttle Program responsibilities—including safety oversight."[36] Two other pressures added stress to an already stretched Shuttle system. In the 1990s the Russian Space Agency was on the brink of collapse. The Clinton Administration made it a national security priority to partner with the agency and infuse it with cash, lest Russian scientists and technology fall into the hands of hostile regimes. At least $650 million of NASA's human space flight budget was used to purchase Russian hardware and services related to U.S.-Russian space cooperation. The second pressure was an ambiguity within the space agency regarding the future of the Shuttle. Some felt that the financial resources of NASA should go to the ISS and that the Shuttle's 30-year-old design was becoming obsolete and should be replaced. This had the effect of precluding any major efforts to upgrade the Shuttle fleet. Yet without a serious attempt to replace it, the wear and tear on the aging fleet continued well into the new decade.

The CAIB's assessment of the NASA culture describes a mindset that never evolved beyond the ebullient "perfect place" of the 1960s when the agency was leaner and less bureaucratic in nature and where the impetus was to build better systems to venture further into outer space. The Report noted "the Apollo-era organizational culture came to be in tension with the more bureaucratic space agency of the 1970s, whose focus turned from designing new spacecraft at any expense to repetitively flying a reusable vehicle on an ever-tightening budget." With this came a strong resistance to internal or external criticism, noted the CAIB, which persisted through the *Challenger* crisis, and "by the eve of the *Columbia* accident, institutional practices that were in effect at the time of the *Challenger* accident—such as inadequate concern over deviations from expected performance, a silent safety program, and schedule pressure—had returned to NASA."[36]

The break-up of the *Columbia* on February 1, 2003, was due to a break in the thermal protection of the leading edge of the left wing, damaged by a piece of insulating foam that struck the wing during take-off.

In managing a technologically complex endeavor with minimal tolerance for error, the CAIB compared the Shuttles to the Navy's management of nuclear warships, and offered the following key elements for success:

- Concise and timely communication of problems using redundant paths
- Insistence on airing minority opinions
- Formal written reports based on independent peer-reviewed recommendations from prime contractors
- Facing facts objectively and with attention to detail
- Ability to manage change and deal with obsolescence of [equipment] over [its] lifetime

The CAIB concluded by calling for a recalibration of expectation, by both NASA and the American public, about the Space Shuttle and the space program.[36] By understanding that space flight is far from "routine," we can diminish the stresses on the Shuttle system and the risk of disasters in the future.

Issues of Survivability

In learning the lessons from the past failures and planning future missions, survivability can only come from a survivable event, meaning that disaster contingencies should seek to maximize systems that are likely to result in a safe return for the crew. Although both Shuttle tragedies were unsurvivable events, we now know that crewmembers did not immediately die on the first mechanical failure. Such knowledge may alter future emergency systems and how the critical elements of survival are addressed. In the case of *Columbia,* these would have been surviving break-up, surviving impact, and surviving until recovery.[20]

These same lessons should apply to medical contingencies. Of all the astronauts who have perished during American and Russian space flight, none died due to a true medical emergency. In the spirit of wilderness medicine themes, mission planners and flight surgeons must promote creative solutions for diagnosis and therapy. At the same time, however, expectations must be in line with reality, and all parties need to accept that major traumas, peritonitis, or intracranial pathology, which are all treatable in a hospital on Earth, may not be survivable on a space mission.

Up to now, nearly all of the tragedies of space flight have been catastrophic, with the deaths of all crewmembers, loss of the vehicle, and complete failure of the mission. The incapacitation or death of an individual has not yet occurred, but it is likely that as mission durations lengthen, increasingly difficult decisions will need to be made. Balancing the care of one crewmember against the risk of exhausting the entire system's resources will never be an easy task, nor will be helping nonclinicians through the death of a colleague and dealing with the "survivor guilt" that may occur. Clear, prospective discussions of these issues will be necessary to allow rational medical system design and to set protocols for the future of medicine in space.

Research for the Future

Just as long-duration ISS missions differ from Shuttle missions, exploration missions differ even more. Future exploration goals

require additional preparation and research before we can be ready to move beyond LEO on a permanent basis. Planning for a lunar colony or Mars base requires additional knowledge of the dangers and best practices facing humans in these aerospace environments.

Analog Environments

There are numerous analogs for space flight exposure, ranging from parabolic flight to underwater operations, from polar expeditions to a bedrest laboratory. Each type mimics a different aspect of the space flight environment and each therefore has applicability for different studies. Microgravity itself (i.e., free fall) can be experienced through parabolic flight. The NASA KC-135A Reduced Gravity Research Program makes use of a four-engine turbojet aircraft to offer alternating intervals of zero gravity (~25 seconds) followed by 1.8G (~20 seconds) per parabola, 40 to 60 parabolas per flight. Other gravitational forces (including lunar and Martian gravity) can also be simulated, although the interval time course remains generally similar.

For research into muscle atrophy and off-loading of gravitational forces, rat models make use of tail suspension methods, whereas human subjects undergo prolonged bedrest. Head-down tilt (usually 4 degrees) can be added to the bedrest, or "dry immersion" methods can be used to create cephalad or intrathoracic fluid shifts similar to those seen on orbit. Research into fluid and electrolyte balances, musculoskeletal changes, and endocrinological changes have all been studied in this way.

To investigate the effects of prolonged isolation and small group dynamics, numerous analog environments have been used. Examples of the latter include polar station and submarine crews, the much publicized Biosphere in Arizona, NASA Extreme Environment Mission Operations (NEEMO) projects in the Aquarius underwater laboratory, and the NASA-Haughton-Mars project on Devon Island. Astronauts in their spacesuits can practice EVA tasks in a space analog environment at the NASA-Johnson Space Center Neutral Buoyancy Lab (NBL).

Valuable conclusions can be inferred from looking at health data from these analog environments. These populations have prolonged absences from proximity to health care, and have limited resources. The Australian National Antarctic Research Expeditions (ANARE) Register noted 5103 illnesses and 3910 injuries over the 9 years from 1988 to 1997.[82] Although most of these individual medical events posed little risk to the crew and mission, there were several of significant severity that needed emergency intervention.

Each of these analogs has benefits and limitations. No single one is suitable for all microgravity research, and investigators must carefully select the model most appropriate to them. Both water immersion and bedrest studies have proved ineffective at predicting changes to cardiovascular parameters in space, the periodic microgravity of the KC-135 is quite a different stress than the sustained microgravity of space flight, and motion sickness aboard the KC is not predictive of in-flight SMS. Submarines and polar stations typically have much larger crews than those seen on space stations (to date), and even the NBL cannot entirely emulate the extravehicular environment for spacewalkers, because materials often behave differently under water than they do in a vacuum. At the same time, much can be learned from working in these analogs, so long as the differences and confounding factors are clearly and prospectively identified.

Artificial Gravity

The countermeasure most avidly sought as the "solution" to many of the most concerning microgravity-associated physiologic effects is artificial gravity (AG). This could be accomplished through rotation, either of the entire spacecraft or an onboard centrifuge. It is hypothesized that, through the use of AG, muscle atrophy, bone demineralization, neurovestibular and neurosensory alterations, and many other physiologically deconditioning effects could be avoided, thus facilitating crewmember return to a gravitational field, whether on Earth, the Moon, or Mars. If space travelers could use an in-flight centrifuge (or other form of AG device) to achieve a dual-adapted state, where they are equally comfortable in microgravity or full gravity, they could potentially avoid reentry disorientation and postlanding postural instability. However, use of a centrifuge could also create vestibular problems through the Coriolis effect, leading to significant motion sickness.

A major consideration of AG is the trade-off between the radius and the rotation rate required to achieve a desired gravity level. Depending on the radius of the rotation, Coriolis and other nausea-inducing forces may persist, and gravitational forces could vary over the length of a human body. The physiologic effects of having different parts of the body at different gravitational gradients are unknown.

Even if such provocative stimuli could be avoided, there is currently little information as to what an "adequate" gravity level might be; is full gravity needed or would partial gravity be sufficient? In addition, does the AG stimulus need to be continuous, or will intermittent application (as with an onboard centrifuge) prevent the physiologic adaptations? If the latter, will the repeated shifting between microgravity and AG cause neurovestibular difficulties? How much time would be required in the centrifuge? Will it vary between crewmembers? How will the "AG prescription" be determined? One study flew a short-arm centrifuge on the Shuttle (STS-90) and found that astronauts who were subjected to 20 minutes of 0.5G to 1G along the longitudinal axis of their bodies on alternate days during the 16 day mission experienced reduced postflight cardiovascular deconditioning. This is encouraging, but not conclusive, evidence for the value of AG.

While it might seem simpler to subject the entire vehicle to a steady gravitational field, there are significant barriers to the design of a rotating spacecraft. Financial, operational, and maneuverability challenges are enormous, and it is unlikely that they will be overcome in the near future.

Exploration-Class Missions

It is with a hope for renewed visits to the Moon and a manned mission to Mars that we strive to understand the effects of space on our bodies. Much of the plan rests on future advances in medicine and engineering. To that end, the National Space Biomedical Research Institute was formed in 1997. This consortium of 12 academic institutions, partnered with research centers, investigates areas of health problems related to long-duration space travel and prolonged exposure to microgravity.[99] It has focused on several areas of potential health concerns for the Mars astronaut, including bone loss, radiation effects, technology development, and psychological effects of prolonged space travel. In addition to astronaut health, engineering feats

Figure 97-52. An artist's rendition of providing care at an extra-terrestrial base. (From NASA Human Space Flight Gallery at http://spaceflight.nasa.gov/gallery./)

Figure 97-53. Putting our world in perspective. (From http://grcimagenet.grc.nasa.gov/GRCDigitalImages/1995/1995_02393L.jpg.)

remain to be tackled. Greater experience with long-term extra-terrestrial habitation will need to be gained prior to a manned mission to Mars. A lunar outpost could serve as a testing ground for new technologies for a Martian base, as well as a place to gain further experience with planetary habitats, launch and landing vehicles, and surface exploration. For the eventual mission to Mars, Weaver and Duke[142] outlined a Mars exploration strategy for NASA with the following goals:

1. Support a crew of 5 to 7 people on the surface for up to 600 days to provide for extensive planetary exploration.
2. Minimize the amount of time the crew is continuously exposed to the interplanetary space environment.
3. Establish an operational system that emphasizes redundancy and multiple uses for a single system. This will not only reduce costs, but allow for greater maintainability.
4. Maintain flexibility throughout the mission and plan for multiple pathways toward desired objectives.
5. Emphasize a risk-mitigation strategy among technical, programmatic, mission, and safety goals.

These investigators further note the importance of accepting the inherent risk of such a long voyage and that "overcoming the temptation to look back to earth to resolve each contingency situation may be the most challenging obstacle to be overcome in embarking on the human exploration of Mars"[142] (Fig. 97-52).

For a manned mission to Mars to succeed, humans must remain the system's most valuable asset and not become its weakest link.[21] During the 4- to 6-month transit to Mars, mental and physical health maintenance will be geared not only toward combating the effects of the long trip but also toward maintaining a focus on the surface mission ahead. Upon reaching the surface, the crew can use both indigenous resources and caches of supplies from earlier unmanned launches from Earth to augment the supplies that their vessel has brought, thus increasing their mission's viability. For initial landings, the use of soil for radiation shielding may alleviate launch burdens from Earth. As successive missions journey to Mars, the possibility of creating water and oxygen and growing crops may have both tangible and psychological benefits for explorers.

The number of crewmembers on such a mission will undoubtedly affect design. Crewmembers will likely be specialists in a number of key areas, with cross training in the event of incapacitation. Among other things, the crew will need expertise in mechanical engineering, electrical engineering, geoscience, life science, and medicine/mental health.[142] Mission parameters would likely require each crewmember to master several diverse support tasks, and currently most NASA sources suggest an optimal crew size of six to eight. It is likely that there will be an emphasis placed on robotic support to alleviate the work burden on crewmembers and to minimize crew risk in performing otherwise dangerous tasks. Robotic devices could also increase the span and scope of discovery, given the limits of manned surface exploration.

As these missions seek a long-term presence first on the Moon and then on Mars, a brief discussion of colonization is worth mentioning. In the ensuing decades, we may choose to establish enough of an infrastructure to support a permanent human presence. Manned missions could progress from the current format of the ISS, where crews stay for set time periods and return home when relieved by another crew, toward larger overlapping missions, perhaps buoyed by mining endeavors or other sustainable activities. Long-term occupants (over years) will likely promote a change from the military model currently in practice to a more colony-based civilian model, including family structures. Throughout this evolution, there will be issues of social organization and governmental policy to be faced. A colony-empire dynamic may result as elements of self-sufficiency develop (via mining or sustainable agriculture). The us-versus-them phenomenon, already seen in some crews' feelings toward mission control, will probably emerge in some form on a planetary level; healthy ways of managing this conflict will need to be developed (Fig. 97-53).

The references for this chapter can be found on the accompanying DVD-ROM.

Appendix: Drug Storage and Stability

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BACKGROUND

Stability data on drug products are generally derived from studies done under controlled and artificial environmental conditions. However, drug stability in the setting of real-world variable climate conditions is more difficult to study. Only limited research in this area has been published.

This appendix reviews the data currently available on drugs likely to be carried in field and expedition medical kits. The list is extensive, and for smaller expeditions only a fraction of the more critical medications will be required. However, it is also useful for medical professionals who are setting up field hospitals and clinics in suboptimal climate conditions.

Most medications, as indicated in the following sections, have strict temperature ranges at which they should be maintained to assure their potency. Other factors, including light, humidity, and packaging, also influence the shelf life and sterility of a drug. Strong packaging is critical; if a parenteral drug (packaged in a syringe for ready use) is frozen, the drug may be fully potent, but its sterility may have been lost due to hairline cracks in the plastic caused by freezing. Polyvinyl (plastic) containers often used to package drugs may affect product stability. Glass containers, although considered the most inert of storage vessels, may leach alkali and associated decomposition products into the product, thereby initiating chemical reactions that alter drug potency. The inclusion of preservatives in the drug formulation and the actual processes used by the manufacturer may also make products more or less resilient in the face of prolonged storage. Thus, for most of the drug products listed, stability and sterility cannot be guaranteed if products are stored under conditions deviating from those recommended by the manufacturer. The following guide should be supplemented with drug packaging information provided by the manufacturer.

EXPIRATION DATES

Drug manufacturers usually set shelf life by calculations of drug potency under ideal conditions. Shelf life is the time during which the potency is expected to be greater than or equal to 90% of the drug's initial potency at manufacture. Shelf life assumes that the drug is maintained at optimal storage conditions. Extremes of temperature, high humidity, loss of integrity of original packaging, and exposure to light can all potentially shorten this period. In addition, some drugs may actually have decreased bioavailability after exposure to non–temperate climate conditions. This is because the rate of dissolution is often the rate-limiting step in the absorption of many medications taken by mouth, and further complicates a drug's pharmacodynamics. High heat and humidity conditions can affect the rate of product dissolution.

STORAGE OPTIONS

Unfortunately, the wilderness setting leaves few options for the storage of drugs under ideal environmental conditions. Vehicular storage, while a convenient option in many cases, can expose drugs to temperature variations much greater than ambient conditions unless efforts are actively undertaken to control the temperature swings. This is illustrated in a 2003 study by Helm and colleagues.[5] This 2003 pre-hospital medical study quantified the temperature stresses placed on medications in real mission conditions, including rescue helicopter, ambulance, and transport vehicles during 2 months of summer and 2 months of winter. This was performed in a moderate climatic zone (southern Germany). Recorded temperatures varied from –13.2°C to +50.6°C (8.2°F to 125°F). The recommended maximum storage temperature (+25°C [77°F]) was exceeded in all rescue vehicles (33% to 45% of total exposure time), whereas the recommended minimum storage temperature (0°C [32°F]) only fell short 19% of total exposure time. The daily maximum temperature ranged from 19°C (66°F) in winter to 32.9°C (91°F) in summer. These results show that even in a moderate climatic zone, drugs used in pre-hospital vehicles are significantly influenced by temperature stress; furthermore, these results recommend the use of temperature-controlled drug boxes.

Vehicle-powered cooling devices are a viable option in this setting, assuming the vehicle battery is kept charged. Styrofoam or plastic cooler boxes with cooling packs are an adequate storage modality as well but can be somewhat cumbersome on a trekking expedition. Many small coolers are available, however, so medications that require cooling or darkness can be separated out and stored in a manageable small unit.

Portable refrigeration units may be required for long-term storage of products that require strict constant refrigeration, especially if ice is not going to be readily available. Many of these units require electricity and are equipped with enough insulation to prevent their contents from becoming too warm in the event of a power failure for up to several hours if unopened. A few products also exist that can also run on

natural gas or kerosene, thereby ameliorating the problem of unreliable electricity supply. Systems with passive cooling, such as ice packs, tend to be lighter, whereas those with active cooling systems can be much heavier (over 50 kg [110 lbs]).

An important consideration in choosing these devices and other, simpler ice coolers is the thickness of their foam insulation and the durability of the surrounding encasement, often constructed of metal or plastic. The amount of snow or ice used for cooling varies depending on the ideal temperature storage range for each drug. A thermometer should be kept inside the box near the medications and monitored regularly.

Portable generators can weigh as little as 12 kg (26.5 lbs), but careful consideration of the power needs for the expedition should be made prior to purchase, as the weight to be carried can increase quickly with increased power requirements. Additional weight is also incurred for the generator's power source. Power types include batteries, liquid fuel, and solar panels. Solar has the added advantage of being lightweight and renewable. There are several solar products that are ideal for portable heating and cooling equipment and for recharging car batteries.

Many medications suggest storage at "room temperature" of 15° to 30°C (59° to 86°F). For these, standard refrigeration is not appropriate. Electric medication storage boxes are available and for long expeditions are a reasonable solution. A more inexpensive lightweight solution for short-term medication storage problems is to use chemical heat packs or instant cold packs (both widely commercially available) to heat or cool the ambient temperature inside a cooler. Confirm appropriate temperature range with a thermometer. Chemical heat packs can produce up to 150°F (70°C) of heat and last for several hours, so do not place them directly on the medication. Otherwise, the medication may "cook"; also avoid direct ice application to avoid inadvertent freezing. Routinely re-check the medication thermometer.

Whatever storage container is chosen should be waterproof, and, if possible, air-tight. Consider adding a desiccant to decrease damage from humidity. Many medications need to be protected from light, so if the box is transparent, place it in a dark bag. Place loose tablets in a bottle or blister pack to avoid breakage. If product packaging is discarded to save space, retain the product information leaflet and dosing information in a plastic bag.

There is no formal consensus on the long-term effect of humidity and other climate changes on many medications. Anticipate that any gel-encased medication, such as a suppository, may melt in high humidity and/or warmth, and become challenging to use even if it is still medically active.

After longer trips, consider replacing medications with a fresh stock.

"REAL WORLD" DRUG STABILITY RESEARCH

Most medication stability data is obtained from lab tests in very controlled artificial environments. The data in this appendix are primarily from these sources. However, there exists a small body of literature addressing "real world" drug storage and usage. Some of the best research that more closely mimics field use in wilderness settings comes from the pre-hospital emergency medical services (EMS) literature.

Particular attention has been focused on medications commonly stocked in pre-hospital EMS vehicles. Understandably, these are the medications most likely to undergo degradation as a result of prolonged or intermittent exposure to more extreme climate conditions. See the Helm study discussed earlier for quantified temperature variations in EMS transport vehicles.[5]

Valenzuela and colleagues[8] performed a study that monitored 23 pre-hospital medications, using gas chromatography and mass spectrometry, for degradation over the course of a typical summer in Tucson, Arizona. The medications studied were:

Aminophylline
Atropine
Bretylium tosylate
Calcium chloride
Dexamethasone
Dextrose
Diazepam
Diphenhydramine
Dopamine hydrochloride
Epinephrine
Furosemide
Isoetharine
Isoproterenol
Lidocaine
Lopressor (metoprolol)
Morphine sulfate
Naloxone
Nifedipine
Nitroglycerin tablets
Phenobarbital
Sodium bicarbonate
Thiamine
Verapamil

After being exposed to temperatures ranging from 28°C to 39°C (82.4°F to 102.2°F) in paramedic storage containers, only two of the studied medications were found to have significant chemical changes. Most notably, isoproterenol lost 11% of the parent compound over the course of the 4 weeks. Its degradation products may be similarly active, but this cannot be assumed. Volumes administered in the field may therefore contain lower than the expected dose. Epinephrine was also affected. The buffer used to maintain the epinephrine solution's pH was degraded, leading to a decrease in the pH of the solution. However, there was no increase in degradation products of the actual epinephrine component. Nifedipine capsules melted in this study, but the medication itself was found to be stable to heat over the course and conditions of the study period.

A later experiment by Church and colleagues[3] found that epinephrine is significantly degraded by heat even before if exhibits the characteristic dark brown color change normally associated with its degradation by oxidation.

Gottwald and colleagues[4] studied pre-hospital stability of diazepam and lorazepam. Injectable solutions were stored for up to 210 days in clear glass syringes in three conditions: refrigerated at 4°C to 10°C (39.2°F to 50°F), ambient temperature of 15°C to 30°C (59°F to 86°F), and oven-heated at 37°C (98°F). High-performance liquid chromatography was performed at 30-day intervals. After 210 days, the reduction in diazepam concentration was 7% refrigerated, 15% at ambient temperature, and 25% heated. The reduction in lorazepam

concentration was 0% refrigerated, 10% at ambient temperature, and 75% heated. Diazepam retained 90% of its original concentration for only 30 days of on-ambulance storage, whereas lorazepam retained 90% of its original concentration for 150 days. The decrease in lorazepam concentration correlated with an increase in the maximum ambient temperature in San Francisco. These results suggest that diazepam and lorazepam can be stored on ambulances. When ambient storage temperatures are 30° C (86° F) or less, the authors of this study suggest that ambulances carrying these medications should be restocked every 30 to 60 days. When drug storage temperatures exceed 30° C (86° F), more frequent stocking or refrigeration is required.

Johansen and colleagues[6] reviewed the effects of extreme temperatures on the chemical structure of four advanced cardiac life support (ACLS) drugs: atropine sulfate, lidocaine hydrochloride, epinephrine, and naloxone hydrochloride. The temperatures used were –20° C, +70° C (–4° F, +158° F), and a combination of the two; these were compared with room temperature controls. These temperature extremes occurred over a period of 48 hours, with four 4-hour-long temperature spikes/troughs. This extreme temperature variation did not seem to affect the chemical structures of these medications. However, the actual bioactivity of the drugs was not tested.

Carrasco and colleagues[7] recently reviewed the physical, chemical, and sterility characteristics of previously prepared saline infusions stored in ambulances. They hoped to determine how long they remained in optimum condition. A total of 8028 cultures were performed. Colonization was present in only 1.7% of cultures; only two had more than 5 colony-forming units (CFUs) per mL. This study suggested that there was no difference between "recently prepared" saline infusion solutions and those "previously prepared" and exposed for 24, 48, or 72 hours. This assists with practical real-world constraints of preparation.

Bartfield and colleagues[1] investigated the shelf life of buffered lidocaine. It was prepared 7 days, 1 day, and just before use, and compared to a control solution. They found that there was no significant difference in the extent or duration of anesthesia between the different preparation times.

Brown and colleagues[2] recently wrote a review of the last 15 years of pre-hospital literature on this topic. They noted that the evidence is clear that EMS medication storage is not consistent with the United States Pharmacopoeia (USP) definition of controlled room temperature. However, the actual impact on medication stability and potency remains unclear and needs more study to determine the extent of the problem and to develop and validate appropriate solutions.

DRUG STABILITY INFORMATION

The drug stability data on the following drugs and other medical products are nearly always based on studies done in controlled environmental conditions. It is often difficult to ascertain whether a drug product can be safely used after storage under conditions other than those specified by the manufacturer.

As mentioned previously, the question of stability of drug products pertains not only to the drug itself but also to the container in which it is packaged. If, for example, a parenteral drug packaged in a syringe for ready use is frozen, the drug itself may be fully potent, but the sterility of the product may be lost due to hairline cracks from freezing of the syringe.

Thus, for most of the drug products listed, stability and sterility cannot be guaranteed if stored under conditions other than those recommended by the manufacturers.

Slight variations in packaging and formulation between brands may influence the drug's stability. The following guide should be supplemented with drug packaging information provided by the manufacturer.

If the medication is annotated with an asterisk (*), please see the section on "Real World" Drug Stability Research for additional related information. Please note, however, that any deviation from the manufacturer's recommendations is the choice of the treating medical professional and is neither condoned nor approved by the authors of this chapter or text.

Medications are listed by their generic names. Where trade names are given, no endorsement of a particular product is implied unless explicitly stated.

By pharmaceutical convention, "room temperature" is defined as between 15° C and 30° C (59° F and 86° F) and "controlled room temperature" is defined as between 20° C and 25° C (68° C and 77° F).

Availability in the United States is subject to Food and Drug Administration (FDA) and Drug Enforcement Agency (DEA) regulations and annotated as OTC (over-the-counter), Rx (prescription required), DEA Schedule (S II to S IV), or NA (not available in the United States).

Acetaminophen Tablets, Elixir, and Suppositories (OTC)

Store at room temperature and definitely below 40° C (104° F). Avoid freezing, as stability after freezing is unknown. Not known to be light sensitive. Keep suppositories refrigerated below 27° C (81° C).

Acetaminophen with Codeine Tablets and Elixir (S III)

Store at room temperature and definitely below 40° C (104° F). Do not refrigerate or freeze. Protect from light and moisture.

Acetaminophen with Hydrocodone Tablets (S III)

Store at room temperature and definitely below 40° C (104° F). Do not refrigerate or freeze. Protect from light and moisture.

Acetazolamide Tablets, Sustained-Release Capsules, Injection, and Oral Formulation (Rx)

Store tablets at room temperature; store capsules at controlled room temperature.

Oral formulation: a studied extemporaneous oral formulation of acetazolamide 25 mg/mL in a 1:1 mixture of Ora-sweet, Ora-plus, and in cherry syrup (diluted 1:4 with simple syrup) was stable at 94% of initial concentration for up to 60 days. Temperatures tested were 5° C and 25° C (41° F and 77° F), and the solutions were protected from light.

Store powder for injection at room temperature. Manufacturer labeling states that the reconstituted drug is stable for 12 hours at room temperature and for 3 days if refrigerated (36° F to 46° F). Other research, however, has demonstrated 90% potency for 1 week at room-temperature conditions, 4 weeks when refrigerated, and 8 weeks when frozen (admixed

with 50 mL D5W, 100 mL sodium chloride injection, 50 mL Lactated Ringer's Injection, or 45 mL Lactated Ringer's Injection with 5 mL sodium bicarbonate 5%). Do not freeze if combined with Lactated Ringer's with sodium bicarbonate, as the solution becomes turbid. Do not administer if discoloration, cloudiness, or particulate matter is observed.

Acetic Acid Solution (OTC)

Store in airtight containers at controlled room temperature. Protect from light.

Albuterol Tablets, Syrup, and Inhaled Formulations (Rx)

Tablets and syrup: store between 2°C and 25°C (36°F and 77°F). Protect tablets from excessive moisture. Refrigeration of syrup can improve taste.

Aerosol inhalers: store at room temperature. Do not expose to excessive temperatures (49°C, 120°F) for more than 1 to 2 days due to explosive danger of the chlorofluorocarbons (CFCs). Do not puncture or incinerate aerosol containers.

Inhalation solution and capsules for inhalation: store between 2°C and 30°C (36°F and 86°F). Inhalation solution is light yellow to clear; discard if discolored.

Aloe Gel, Ointment, Laxatives (OTC)

Topical: *Aloe vera* has been used for burns, wounds, and as an antipruritic. Pharmacologically it is composed of substances capable of producing topical anesthesia, bactericidal activity, and increased local microcirculation, but actual therapeutic capability is not clear. Lotions are for external use only. Product stability in extremes of temperature is not well studied.

Laxatives: aloe acts as a stimulant laxative if taken internally due to anthraquinone glycosides. Aloe laxatives (e.g., Nature's Remedy) should be stored at room temperature in airtight containers. Avoid humidity and temperatures over 38°C (100°F).

Aluminum Acetate Otic and Topical Preparations (OTC)

Otic solution: a clear colorless liquid comprised of acetic acid 2% in aqueous aluminum acetate solution. Store below 30°C (86°F) but protect from freezing.

Topical solution: stable virtually indefinitely, but partially used irrigation solutions have high potential contamination rates. Label extemporaneously prepared solutions with a 7-day expiration date, which is consistent with the shelf life recommended by the manufacturer.

Amiodarone Tablets and Vials (Rx)

Store tablets and vials for intravenous (IV) administration at room temperature. Protect both from light during storage, but IV form need not be protected from light during infusion itself. Note that dosing studies were performed using polyvinyl chloride (PVC) tubing. The use of other plastic tubing may affect dosing, as amiodarone IV has been found to leach out plasticizers. This effect is exacerbated by higher drug concentrations and lower flow rates than recommended by the manufacturer.

Antacids (OTC)

Avoid freezing aluminum hydroxide or milk of magnesia products. If frozen, many antacids separate into water and gel layers upon thawing. Freezing is not known to affect the therapeutic value of the product, but anecdotally it affects taste and prevents reformation of the emulsion, even with shaking.

Aspirin Tablets, Oral Solution, and Suppositories (OTC)

Tablets: aspirin is stable in dry air but gradually hydrolyzes to salicylate and acetate and gives off a vinegar odor. Most manufacturers recommend storage at room temperature.

Oral solution: an extemporaneous oral solution can be made from a commercial buffered effervescent tablet (Alka Seltzer) with 90 mL of water.

Suppositories: store between 2°C and 15°C (36°F and 59°F).

Atenolol Tablets (Rx)

Store at controlled room temperature, 20°C to 25°C (68°F to 77°F). Dispense in well-closed, light-resistant containers.

Atropine Injection (Rx)*

Store at room temperature and protect from light. Atropine sulfate 1 mg/mL solutions packaged in Tubex (0.5 mL and 1 mL) have been shown to be stable for 3 months. Atropine methylnitrate 10 mg/mL solutions are stable for 6 months when stored in dark bottles at room temperature. Inspect solution prior to administration for the presence of particulate matter, cloudiness, or discoloration and discard if present.

Azithromycin Tablets, Capsules, Suspension, and Injection (Rx)

Tablets and capsules: store below 30°C (86°F).

Suspension: single dose packets should be refrigerated if possible (5°C to 30°C, 41°F to 86°F) and, if reconstituted with 60 mL of water, should be used immediately. Discard multiple dose suspension after 10 days.

Injection: store under 30°C (86°F). After preparation, it is stable for 24 hours at room temperature and for 7 days if refrigerated.

Bacitracin Topical (OTC) and Injection (Rx)

Topical: store at room temperature. If in ointment form, it is stable but rapidly inactivated in water. Calamine, benzocaine, and zinc oxide have been combined with bacitracin without affecting its stability. If in aqueous solution, it must be refrigerated and is stable for only 1 week due to oxidation.

Injection: store sterile powder for injection between 2°C and 15°C (36°F and 59°F) and protect from light. Solution is stable for 1 week after reconstitution if refrigerated.

Bismuth Subsalicylate Tablets and Suspension (OTC)

Store at room temperature and avoid heat greater than 40°C (104°F). Suspension should not be frozen.

Bretylium Tosylate (Rx)*

Store at room temperature. Protect from freezing.

Bupivacaine Injection (Rx)

Bupivacaine is a relatively stable drug, but excessive heat or cold decreases its shelf life. Store between 15°C and 40°C (59°F

and 104°F). If frozen, may be used after thawing provided the container is completely intact and the solution remains clear. Bupivacaine with epinephrine exposed to light and/or temperatures over 40°C (104°F) for a long period should not be used due to loss of epinephrine effect.

Butorphanol Tartrate Injection and Nasal Solution (S IV)

Store below 30°C (86°F). Protect from light and do not freeze. Inspect parenteral drug for particulate matter or discoloration prior to use and discard if present.

Calcium Chloride Injection (Rx)*

10% solution: drug pH is altered significantly if frozen or exposed to temperatures over 40°C (104°F). The product should be considered unusable if either occurs.

Ceftriaxone Injection (Rx)

Store powder for injection at or below 25°C (77°F). Protect from light. The color of solution ranges from light yellow to amber, depending on concentration, length of storage, and diluent. Once mixed, intramuscular (IM) and IV solutions remain >90% potent for up to 10 days if refrigerated at 4°C (39°F). Potency is affected by diluent, concentration, and temperature. IM and IV preparations may maintain >90% stability for as little as 1 or 3 days, respectively, at room temperature. If reconstituted with 5% dextrose or 0.9% sodium chloride solution and then frozen at –20°C (–4°F), preparations have been stable for 26 weeks in PVC or polyolefin containers. Thaw at room temperature before using. Unused thawed solutions should be discarded. Do not refreeze. Ceftriaxone may be incompatible with other antimicrobials; do not mix.

Cephalexin Capsules, Tablets, and Oral Suspension (Rx)

Capsules and tablets: store at room temperature.

Suspension: stable after reconstitution for up to 14 days if refrigerated, ideally between 2°C and 8°C (36°F and 46°F). Keep tightly closed. Shake well before using.

Charcoal, Activated (OTC)

Sealed aqueous suspensions may be stored for at least 1 year in tightly sealed container. For longest shelf life, keep in well-sealed metal or glass containers.

Ciprofloxacin Capsules, Tablets, Suspension, and Injection (Rx)

Oral: store tablets at room temperature, and store capsules and suspension diluent below 25°C (77°F), but do not freeze. Discard unused suspension after 14 days. Protect from light.

Injection: store flexible containers between 5°C and 25°C (41°F and 77°F) and IV infusion vials between 5°C and 30°C (41°F and 86°F). Avoid temperatures over 40°C (104°F) and protect from light. When injection is diluted for injection, it is stable for 2 weeks at either room temperature below 30°C (86°F) or when refrigerated between 2°C and 8°C (36°F and 46°F). Do not freeze.

Cyclopentolate Hydrochloride Ophthalmic Solution (Rx)

Store between 5°C and 25°C (41°F and 77°F). Keep in tightly capped airtight container.

DEET-Containing (N,N-Diethyl-Meta-Toluamide, Diethyltoluamide) Insect Repellents (OTC)

Store at room temperature and in airtight containers. Avoid exposures of propellant cans to temperatures of 49°C to 54°C (120°F to 130°F) due to explosion danger. Do not store or use near fire or open flame. Avoid contact with plastic or rayon. DEET may be toxic internally, so store away from other consumables.

Dermabond (2-Octyl Cyanoacrylate) Topical Skin Adhesive (Rx)

Store below 30°C (86°F), protect from moisture, direct heat, and incidental crushing. The product should be used immediately after crushing the glass ampule, as it will not flow freely after a few minutes.

Dexamethasone Injection (Rx)*

Injection: a clear, colorless to light yellow solution that is sensitive to light and extremes of temperature. Do not store at high temperature for long periods. It maintains full potency for 6 months at 40°C (104°F) and up to 3 months at 50°C (122°F). Do not use after freezing and protect from light except while injecting.

Dextroamphetamine Tablets, Elixir, and Capsules (S II)

Tablets: store in well-sealed containers at room temperature.

Elixir and extended-release capsules: store in tight, light-resistant containers, below 40°C (104°F), preferably at room temperature. Do not freeze elixir.

Dextrose Oral (OTC) and Injection (Rx)*

Oral: store in well-airtight containers.

Injection: store 50% parenteral dextrose below 25°C (77°F). Do not freeze or expose to extreme heat. Do not use if cloudy. Discard unused portions.

Diazepam Tablets, Oral Solution, Suppositories, and Injection (S IV)*

Tablets and elixir: store at controlled room temperature. Protect from moisture and avoid freezing.

Oral solution: store at controlled room temperature. Protect from moisture and avoid freezing. Available as both regular dose and concentrated solution requiring calibrated dropper for accurate dosing. Do not prepare and store doses for future use.

Suppository: store at controlled room temperature and protect from moisture.

Injection: store at controlled room temperature. If frozen, diazepam tends to flocculate and precipitate. Rewarm with warm water and, if no precipitate is visible, the product may be used. Do not inject if solution is cloudy, contains particulate matter, or is darker than slightly yellow.

Digoxin Injection (Rx)

Digoxin has been shown to be stable at room temperature for 3 months in Tubex cartridges. Protect from light and store between 15°C and 25°C (59°F and 77°F). Use diluted injection immediately. This product is compatible with most IV infusion fluids.

Diltiazem Capsules, Oral Solution, and Injection (Rx)

Capsules: store at room temperature. Protect from excess humidity.

Oral solution: 12 mg/mL solution prepared in Ora-Sweet, Ora-Sweet SF, Ora-Plus, or in a 1:4 mixture of cherry syrup and simple syrup was more than 92% potent for up to 60 days at either 5°C or 25°C (41°F or 77°F). In another study, 1 mg/mL solution prepared from dextrose, fructose, mannitol, and sorbitol (but NOT lactose) remained potent for a minimum of 50 days at 25°C (77°F).

IV solution: should preferably be stored between 2°C and 8°C (36°F and 46°F), without freezing, but may be stored at room temperature for up to 1 month, then discarded.

Diphenhydramine Tablets, Elixir (OTC), and Injection (Rx)*

Store at room temperature if possible in airtight containers. Tablet, elixir, and injection (10 mg/mL) forms are stable after freezing, but injection containers should be checked for cracks or leakage. Protect injection form from light.

Domeboro Astringent and Otic Solutions (OTC)

Astringent solution: do not cover compress or wet dressing with plastic; allow to breathe.

Otic solution: a clear colorless liquid. Store below 30°C (86°F). Avoid freezing.

Dopamine Hydrochloride Injection (Rx)*

Store at room temperature and protect from light. Brief exposure to temperatures of 40°C (104°F) is tolerated, but excessive heat should be avoided. Do not freeze. Do not use if there is yellow, pink, purple, or brown discoloration of the solution, as this indicates decomposition. The diluted form is stable for injection for at least 24 hours after dilution.

Doxycycline Capsules, Tablets, Syrup, Suspension, and Injection (Rx)

Oral formulations: store capsules, tablets, syrup, and suspension below 30°C (86°F), preferably at room temperature. Store delayed-release capsules between 15°C and 25°C (59°F and 77°F). Suspension is stable for 2 weeks after reconstitution if stored at room temperature.

Injection: frozen, reconstituted 10 mg/mL solution for injection in sterile water is stable for 8 weeks when stored at –20°C (–4°F). After thawing, excess heating is not recommended. Do not refreeze. Protect from light. The infusion should be completed within 6 to 48 hours (brand and diluent dependent) if not refrigerated.

Epinephrine Injection (Salts and Solutions) (Rx)*

Store at controlled room temperature and protect from light and air. Avoid freezing.

Heat above 40°C (104°F) and exposure to light may inactivate the product. Oxidation causes a color change to pink, then brown. Do not use if discolored, cloudy, or contains precipitate.

Erythromycin Tablets, Suspensions, Topical, and Injection (Rx)

Oral formulations: erythromycin estolate and erythromycin ethylsuccinate (EES 200 and 400) liquid suspensions maintain their potency for 14 days at room temperature. Refrigeration maintains optimal taste. Tablets should not be crushed.

Topical: stable for 2 years when stored at room temperature. Gel and pledgets should be stored at room temperature while ointment should be stored under 27°C (81°F). An extemporaneous topical solution made from tablets with hydroalcoholic vehicle as a 2% solution was stable for 60 days at 25°C (77°F). A 2.7% preparation in E-Solve was stable for 4 months when refrigerated between 4°C and 8°C (39°F and 46°F).

Injection: stable at room temperature in dry form. Reconstituted IV solution should be used within 8 hours after preparation or within 24 hours if refrigerated. Drug stability may be maintained up to 2 weeks if refrigerated but carries significant risk of contamination. Solution may be frozen for 30 days and is stable if used within 8 hours of thawing under refrigeration conditions. Do not refreeze thawed solution.

Estazolam Tablets (S IV)

Store below 30°C (86°F). Stable for 3 years after date of manufacture.

Fluocinolone Acetonide Ointment and Shampoo (Rx)

Ointment: store in a tight container below 40°C (104°F), preferably at room temperature. Avoid freezing.

Shampoo: stable for 3 months after extemporaneous formulation.

Furazolidone Tablets and Liquid (NA)

Store in light-resistant container. Exposure to strong light may cause darkening. Do not expose to excessive heat. May crush tablets and administer in spoonful of corn syrup.

Furosemide Oral Formulations and Injection (Rx)*

Oral, tablets and solutions: store in tightly closed light-resistant container at room temperature. Discard oral solution bottles 60 days after opening. Do not use if discolored.

Injection: protect containers from light. When packaged in Tubex cartridges (2 mL of 10 mg/mL) potency was retained for 3 months at room temperature. If exposed to refrigeration, solution may be used barring evidence of cracking, leaking, or other damage to glass containers. All intact ampules or syringes, when returned to room temperature, should be vigorously shaken to redissolve any constituents that may have crystallized out of solution; stability should not be affected. Do not use if discolored.

Glucagon Injection (Rx)

In powder form, glucagon should be refrigerated but is stable at room temperature for several weeks. When diluent is added to powder, the resulting solution may be refrigerated for up to

3 months. Thawing will not affect activity. Cloudy or thick diluent should not be used. Do not use if stored at temperatures greater than 35°C (95°F) for an extended period.

Hydrocortisone Tablets, Suspension, Topical Cream, and Injection (Rx)

Oral preparations: store below 40°C (104°F), at room temperature. Protect suspension from light and freezing.

Topical: do not refrigerate.

Injection: do not freeze. Only use reconstituted solutions if clear and discard after 3 days.

Hydromorphone Tablets, Elixir, Suppositories, and Injection (S II)

Tablets and elixir: store at room temperature and protect from light.

Suppositories: keep refrigerated.

Injection: store at room temperature. Avoid refrigeration/freezing as precipitation or crystallization can occur, but product may be redissolved at room temperature without affecting stability. A slight yellowish discoloration does not affect potency. Polypropylene infusion syringes containing hydromorphone hydrochloride in normal saline were stable for 30 days when stored at 30°C (86°F).

Ibuprofen Tablets (OTC)

Store at room temperature and protect from light.

Intravenous Solutions (D5W, D5NS, Etc.)*

Effects of freezing these solutions are unknown. Incomplete resolubility, especially with electrolyte-containing solutions, seems a real danger.

Isoproterenol Hydrochloride (Rx)*

Store at room temperature 15°C to 30°C (59°F to 86°F). Protect from light. Do not use if solution is pinkish to brownish in color.

Ketoconazole Shampoo and Tablets (Rx)

Shampoo: store at or below 25°C (77°F). Protect from light.

Oral tablets: store at room temperature. Protect from moisture.

Lacriserts (Hydroxypropyl Methylcellulose) Topical Ocular Solution (Rx)

Store drops at room temperature in airtight containers. Protect from freezing.

Levofloxacin Tablets and Injection (Rx)

Tablets: store at room temperature and protect from light.

Injection: store concentrate at room temperature and protect from light. Reconstituted form can be stored up to 3 days if kept below 25°C (77°F), 14 days if refrigerated between 2°C and 8°C (36°F and 46°F), and up to 6 months if frozen. Thaw at room temperature or under refrigeration conditions but do not thaw by immersion in water or by microwave. A mild yellow/greenish color is normal and not indicative of contamination or breakdown. However, do not use if particulate matter or other discoloration is noted.

Lidocaine Injection (Rx)*

Lidocaine is a relatively stable drug, but excessive heat or cold decreases its shelf life. Store between 15°C and 40°C (59°F and 104°F). If frozen, it may be used after thawing provided the container is completely intact and the solution remains clear. Lidocaine with epinephrine exposed to light and/or temperatures over 40°C (104°F) for a long period should not be used due to loss of epinephrine effect.

Lidocaine/Epinephrine/Tetracaine (LET) Topical (Rx)

Anesthetic solution stored in amber glass (light-shielding) containers is stable for 4 weeks at approximately 18°C (65°F) and for 26 weeks if refrigerated (4°C, 39°F).

Lindane (Gamma-Hexachlorocyclohexane) Lotions and Shampoo (Rx)

The lotion and shampoo become thick when frozen, but do not lose effect. The effect of high temperatures is unknown. Protect from light. Spray should not be exposed to temperatures below –2°C (28°F) or above 54°C (129°F).

Loperamide Hydrochloride Capsules (OTC)

Store at room temperature. Do not mix oral solution with other solvents.

Lorazepam Tablets, Oral Solution, and Injection (S IV)*

Tablets: store at room temperature and protect from moisture.

Oral solution: refrigerate between 2°C and 8°C (36°F and 46°F). Protect from light.

Injection: refrigerate between 2°C and 8°C (36°F and 46°F). Protect from light. Do not use if cloudy, discolored, or if particulate matter is present.

Mannitol Injection (Rx)

Injection form (5% to 25% solution): store at controlled room temperature. Solutions of 15% or higher may crystallize at temperatures below 25°C (77°F) but will resolubilize when warmed to 70°C to 80°C (158°F to 176°F) in a water bath. A reheated solution must be allowed to cool to body temperature prior to administration. Microwaves had been used to reheat mannitol, but extreme caution is indicated as explosions of ampules have been reported. Sterility cannot be guaranteed if frozen.

Meperidine Hydrochloride Oral Solution and Injection (S II)

Oral solution: store at room temperature. Protect from moisture, light, and freezing.

Injection: store at room temperature. Protect from moisture, light, and freezing. If frozen, injection form may be used after thawing if the solution shows no signs of precipitation or cloudiness and if the ampules or vials show no signs of cracking or leaking.

Metoprolol Oral Preparations and Injection (Rx)*

Store at 25°C (77°F). Excursions permitted to 15°C to 30°C (59°F to 86°F). Do not freeze. Protect from moisture. Store in a tight, light-resistant container.

Midazolam Oral Solution and Injection (S IV)

Oral solution: an oral solution of 2.5 mg/mL, made from injectable midazolam and a flavored, dye-free syrup, maintained >90% of the original concentration throughout a 56-day trial

period at temperatures of 7°C, 20°C, and 40°C (45°F, 68°F, and 104°F).

Injection: store in syringes or glass at room temperature and protect from light. Parenteral midazolam of 2 mg/mL (hydrochloride salt) in 0.9% sodium chloride injection was stable in polypropylene infusion-pump syringes at both 5°C and 30°C (41°F and 86°F) for 10 days. A 3 mg/mL concentration maintained at least 90% stability for 1 week when stored in disposable polypropylene syringes in 20°C, 32°C, and 60°C (68°F, 90°F, and 140°F). Midazolam 40 mg/L in 0.9% sodium chloride was stable when protected from light at 21°C (70°F) over 24 hours, with no loss in glass or laminate containers and less than 2% loss in PVC bags.

Modafinil Tablets (S IV)

Store at controlled room temperature. Not known to be light sensitive.

Morphine Sulfate Injection (S II)*

Store at room temperature in airtight containers. Protect from light. When exposed to air, morphine sulfate loses its water and concentration may change, producing higher than expected dosing. Avoid freezing. If freezing occurs, it does not affect potency but may form precipitate. Solution should be colorless to pale yellow. Do not inject if cloudy, discolored, or contains precipitate. May use if precipitate resolubilizes with shaking. Most studies of morphine stability have been limited to 30 days, but one study noted stability after 14 weeks of frozen storage. However, intentional freezing cannot be recommended.

Moxifloxacin Tablets and Injection (Rx)

Tablets: store at room temperature and protect from high humidity.

Injection: store premixed flexible containers at room temperature and protect from light. Do not refrigerate, as IV solution tends to precipitate. Do not use if particulate matter or discoloration is noted.

Nalbuphine Hydrochloride Injection (Rx)

Store at room temperature and protect from light. Do not use if discolored or contains precipitate.

Naloxone Hydrochloride Injection (Rx)*

Store at room temperature and protect from light. Dilute solution prior to use and if not used, discard any solution 24 hours after dilution. Do not save partial doses for future use. Do not administer if discoloration, cloudiness, or precipitate are observed. Protect from freezing and do not use if frozen.

Neosporin Ointment (OTC)

Store at controlled room temperature.

Nifedipine Capsules, Tablets, and Injection (Rx)*

Capsules: store between 15°C and 25°C (59°F and 77°F) and protect from light.

Tablets: store at room temperature and protect from light.

Injection: store below 25°C (77°F). Ready-to-use infusion retains potency for only 1 hour in daylight and 6 hours in artificial light. Keep in light-sealed container until absolutely ready to use.

Nitroglycerin Sublingual Tablets, Spray, Topical, and Injection (Rx)*

Oral preparations: keep sublingual tablets in original glass container, tightly capped. Discard cotton once removed. Store at room temperature. Protect from moisture, heat, cold, and humidity. Discard unused sublingual tablets after 6 months. Translingual spray has guaranteed potency for 4 years from the date of manufacture when stored below 50°C (122°F).

Topical ointment: tube should be kept tightly closed and stored at room temperature.

Injection: nitroglycerin solutions (diluted in 5% dextrose or 0.9% sodium chloride solutions in glass containers) are stable for up to 2 days at room temperature and up to 7 days when refrigerated. Extemporaneous solutions of nitroglycerin in concentrations ranging from 0.035 to 1 mg/mL were stable in glass for up to 70 days at room temperature and for 6 months when refrigerated. Brief exposure up to 40°C (104°F) does not adversely affect the potency or stability of the solution. Do not freeze.

Norfloxacin Tablets and Ophthalmic Solution (Rx)

Tablets: store at room temperature and definitely below 40°C (104°F). Store in tightly closed containers.

Ophthalmic solution: store at room temperature and protect from light.

Ofloxacin Tablets, Otic Solution, and Injection (Rx)

Tablets: store below 30°C (86°F) in a tightly-closed container.

Otic solution: store at room temperature in airtight container.

Injection: store single-use vials and premixed bottles at room temperature. Store premixed mini-bags at or below 25°C (77°F). Avoid light, freezing, and excessive heat. Brief exposure to 40°C (104°F) has not been shown to damage product.

Penicillin G Procaine Injection (Rx)

Should be refrigerated but is stable at room temperatures for 6 months. Higher temperatures increase hydrolysis and decrease potency. May be frozen and retain potency. Solutions prepared for injection are stable are room temperature for at least 1 day and up to 1 week if refrigerated.

Penicillin GK Injection (Rx)

Stable in powder form for 2 to 3 years if stored at no greater than 30°C (86°F). Higher temperatures cause decreased potency. Solutions prepared for injection are stable are room temperature for at least 1 day and up to 1 week if refrigerated.

Phenobarbital Injection (S IV)*

Store at room temperature and protect from light. Not stable in aqueous solutions. Inspect for precipitate and discoloration before use and do not administer if present.

Phenylephrine Nasal (OTC), Ophthalmic Solution (Rx), and Injection (Rx)

Do not use nasal, ophthalmic, or parenteral solutions if brown in color or if there is precipitation. Store parenteral solution at room temperature and definitely below 40°C (104°F). Protect all forms from light and freezing.

Phenytoin Tablets and Injection (Rx)

Oral preparations: store at room temperature. Protect from moisture and light.

Injection: store at room temperature. Solution is stable if there is no precipitation or haziness. A precipitate may form when refrigerated or frozen but will dissolve when warmed to room temperature. A faint yellow color has no effect on potency. More stable in normal saline than in 5% dextrose. Infuse within 2 hours of mixing the solution, then discard.

Polysporin Ointment (Rx)
Store between 15°C and 25°C (59°F and 77°F).

Potassium Permanganate Astringent Solution (OTC)
Diluted form is used to clean wounds, ulcers, abscesses, and dermatoses. Use 0.1% solution in water to be diluted 1 in 10 before use to provide a 0.01% solution. Undiluted potassium permanganate is composed of dark purple or almost black crystals or granular powder and is soluble in cold and boiling water. It is incompatible with iodides, reducing agents, and most organic substances and may be explosive if it contacts organic or other oxidizable substances.

Povidone Iodine Solution (OTC)
Do not heat as iodine concentration may decrease due to interaction with dissolved oxygen or may increase due to water evaporation. Store between 37°C and 42°C (99°F and 108°F).

Prednisone Tablets, Oral Solution, and Suspension (Rx)
Store at room temperature and definitely below 40°C (104°F). Protect from light using airtight container. A suspension prepared using 50 mg of prednisone powder, 100 mg of sodium benzoate, and a sufficient quantity of simple syrup to bring the volume to 100 mL was stable for 12 weeks at room temperature. Shake well prior to use.

Prochlorperazine Injection, Solution, Tablets, and Capsules (Rx)
Store at room temperature in airtight, light-resistant containers. Avoid freezing. Discard discolored solution.

Promethazine Injection, Tablets, Solution, and Suppositories (Rx)
Store oral and parenteral products at room temperature. Refrigerate suppositories between 2°C and 8°C (36°F and 46°F). Protect all forms from light and avoid freezing.

Pseudoephedrine and Pseudoephedrine/Triprolidine Tablets and Capsules (OTC)
Pseudoephedrine tablets and capsules: store between 15°C and 25°C (59°F and 77°F) in a dry place and protect from light. Should not be crushed.

Triprolidine tablets and capsules: store at room temperature and protect from light.

Sildenafil Tablets (Rx)
Store at room temperature.

Simethicone Tablets, Capsules, and Drops (OTC)
Store at room temperature in airtight container. Protect from light and freezing.

Sodium Bicarbonate Injection (Rx)*
Store 50 mL injection ampules at room temperature. Brief exposure to 40°C (104°F) will not compromise efficacy. Do not freeze and do not use if product has been frozen.

Sodium Sulfacetamide Ophthalmic Solution and Ointment (Rx)
Ophthalmic solution: store in a light-resistant container in a cool place, between 8°C and 15°C (46°F and 59°F). Discard if solution darkens to brown.

Ophthalmic ointment: store at room temperature and do not freeze.

Temazepam Capsules (S IV)
Store at room temperature in airtight container. Protect from light.

Tetanus Toxoid (Rx)
Injection: Toxoid should be refrigerated; however, it remains stable for months when stored at room temperature. Do not freeze and do not use if product has been frozen.

Tetracaine Hydrochloride Ophthalmic Solution (Rx)
Protect ampules from light. Refrigerate between 2°C and 8°C (36°F and 46°F) to prevent crystallization and oxidation. Stable for several days at room temperature. If then returned to cool storage, the stability is as labeled by the manufacturer.

Tetracycline Tablets, Topical Solution, and Injection (Rx)
Outdated products may cause renal tubular disease.

Tablets: store at room temperature. Protect from moisture or light (product will darken).

Topical: solution prepared with diluent from the manufacturer is stable for 2 months at room temperature.

Injection: reconstituted solutions are stable at room temperature for 12 hours (6 to 12 hours in 5% dextrose in water).

Tolnaftate Topical Antifungal (OTC)
Store between 2°C and 30°C (36°F and 86°F). Do not freeze. May solidify at low temperatures but liquefies easily when warmed; its potency is not affected. Aerosol products are under pressure. Do not puncture or place in proximity to heat/flame.

Triazolam Tablets (S IV)
Store at room temperature and protect from light.

Trimethoprim/Sulfamethoxazole Tablets, Suspensions, and Injection (Rx)
Oral preparations: store tablets and suspensions in a dry place, either between 15°C and 25°C (59°F and 77°F) or at room temperature, depending on the formulation.

Injection: should be used within 4 or 6 hours (depending on dilution in D5W). Injection (16 mg/80 mg per mL) has been shown to maintain greater than 90% stability for over 60 hours when stored in polypropylene syringes. Discard if cloudy or if precipitate is present. Use within 24 hours if using multiple-use vial for injection. Protect from light. Do not refrigerate or freeze.

Verapamil Hydrochloride Tablets and Injection (Rx)*

Store at controlled room temperature 15° C to 30° C (59° F to 86° F). Protect from light.

Store extended-release tablets at controlled room temperature 20° C to 25° C (68° F to 77° F).

Dispense oral forms in tight, light-resistant container. Protect from moisture.

Discard any unused amount of parenteral solution.

Zinc Salts (OTC)

Zinc oxide (e.g., calamine) is practically insoluble in alcohol and water but dissolves in dilute mineral acids. Store zinc acetate in airtight containers and zinc chloride and sulfate in airtight nonmetallic containers. Zinc sulfate ophthalmic solutions should be stored below 40° C (104° F) in tightly closed or airtight containers. Avoid freezing.

Zolpidem Tablets (S IV)

Store at controlled room temperature in airtight container.

The references for this appendix can be found on the accompanying DVD-ROM.

SUGGESTED READINGS

American Hospital Formulary Service: Drug Information 2004. Bethesda, MD, 2004. Product labeling.

Ette EI: Conscience, the law, and donation of expired drugs. Ann Pharmocother 38:1310-1330, 2004.

Goodyer L: Travel medicine (10) medical tips for travelers. Pharmaceutical J 267:10, 2001.

Micromedex Healthcare Series. Englewood, CO, MICROMEDEX, Inc. (edition expires 2005).

Parfitt K (ed): Martindale: The Complete Drug Reference. London, Pharmaceutical Press (electronic version: Englewood, CO, MICROMEDEX, Inc. Edition expired 2005.)

Physicians' Desk Reference. Montvale, NJ, Medical Economics, 2004.

Physicians' Desk Reference (electronic version). Englewood, CO, MICROMEDEX, Inc. (edition expired 2005).

Physicians' Desk Reference for Nonprescription Drugs and Dietary Supplements. Montvale, NJ, Medical Economics, 2004.

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